Managing Peri-Mitral Flutter

Michael Lim W[1] and Peter Kistler[1]

[1]The Alfred

November 22, 2022

Abstract

The exponential rise in the incidence of peri-mitral flutter has paralleled the increasing use of more extensive atrial substrate ablation for atrial fibrillation (AF). Given the relative paucity of randomised evidence to support its role in AF management, mitral isthmus ablation should largely be reserved for patients with peri-mitral flutter. Catheter ablation for peri-mitral flutter is challenging due to complex anatomic relationships. The aim of this report is to review the anatomic considerations and approaches to catheter ablation for peri-mitral flutter.

Managing Peri-Mitral Flutter

Michael W Lim[1,2,3,5] MBBS and Peter M Kistler MBBS, PhD[1,2,4,5]

Affiliations

The Alfred Hospital, Melbourne, Australia[1]; Baker Heart & Diabetes Institute, Melbourne, Australia[2]; The Royal Melbourne Hospital, Melbourne, Australia[3]; Monash University[4] and The University of Melbourne, Melbourne, Australia[5]

Corresponding Author

Professor Peter Kistler

Heart Centre

The Alfred Hospital

Commercial Road, Melbourne, Victoria, Australia 3004

Tel: +61 3 8532 1111, Fax: +61 3 8532 1100,

Email: peter.kistler@baker.edu.au

Abstract

The exponential rise in the incidence of peri-mitral flutter has paralleled the increasing use of more extensive atrial substrate ablation for atrial fibrillation (AF). Given the relative paucity of randomised evidence to support its role in AF management, mitral isthmus ablation should largely be reserved for patients with peri-mitral flutter. Catheter ablation for peri-mitral flutter is challenging due to complex anatomic relationships. The aim of this report is to review the anatomic considerations and approaches to catheter ablation for peri-mitral flutter.

Key Words

Peri-mitral flutter, mitral isthmus, catheter ablation

Background

Peri-mitral flutter is a common cause of recurrent tachyarrhythmia following atrial fibrillation (AF) ablation, accounting for up to 60% of post-ablation atrial tachycardias (AT)[1-5]. As with other atypical atrial flutters, its pathogenesis is dependent on the presence of abnormal electrical substrate resulting in areas of slow conduction. Accordingly, while occasionally seen de novo in the ablation-naïve, the exponential rise in the incidence of peri-mitral flutter has paralleled the rapid increase in AF ablations being performed worldwide as well as techniques incorporating substrate modification beyond pulmonary vein isolation (PVI).[6,7]The incidence of left atrial flutter steadily increases from approximately 5% with PVI alone, to about 25% with the addition of linear lesions and ablation of complex electrograms, to up to 50% with the extensive stepwise approach to persistent AF ablation.[4,8-11] In the context of frequent recurrence post cardioversion and generally inadequate control with anti-arrhythmic drugs, cardiac electrophysiologists are increasingly called upon to perform catheter ablation for post-procedural peri-mitral flutter. While successful in eliminating recurrent tachyarrhythmia in the majority of patients, achieving bidirectional block with catheter ablation is challenging due to complex anatomic relationships. The importance of attaining this endpoint has been previously highlighted by the significant incidence of recurrent atrial flutters when the mitral isthmus line is incomplete.[12] Prior studies report the majority of re-entrant left ATs post AF ablation traverse prior ablation lines.[13] The aim of this report is to review the anatomic considerations and approaches to catheter ablation for peri-mitral flutter.

Ablation Options and Anatomical Challenges

The mitral isthmus traditionally refers to the left atrial region between the mitral valve annulus and the anterior aspect of the left inferior pulmonary vein (LIPV). However, there are, in fact, multiple isthmuses to which ablative therapy may be applied to interrupt the flutter circuit as it circumnavigates the mitral annulus, each associated with different anatomical advantages and challenges. These ablation lines are defined as follows (see Figure 1):

- The lateral mitral isthmus line connects the LIPV and the mitral valve annulus at approximately the 4 o'clock position, lateral to the left atrial appendage (LAA).
- The anterior mitral line connects the superior aspect of the mitral annulus and the:
- left superior pulmonary vein (LSPV) septal to the LAA ('anterolateral line')
- roof line ('true anterior line')
- right superior pulmonary vein (RSPV; 'anteromedial line')
- The superolateral mitral line connects the posterior base of the LAA orifice adjacent to the LSPV and the mitral annulus.

Lateral Mitral Isthmus Line

The lateral mitral isthmus is a common target to treat peri-mitral flutter owing to its relatively short length, averaging 34.6mm but ranging from 17-51mm, and ability to clearly define bidirectional block using the coronary sinus (CS).[14] However, bidirectional block can be difficult to achieve with endocardial ablation only, owing to several anatomic factors.

Firstly, autopsy studies report a wide variation of myocardial thickness in this region – on average 3mm at the level of the LIPV (range 1.4-7.7mm), 2.8mm at the mid-isthmus (range 1.2-4.4mm) and 1.2mm at the mitral annulus (range 0-3.2mm).[14] The thickness of the mitral isthmus has been shown to determine the acute success of ablation in this region.[15] The operator is typically unaware of individual variability in tissue thickness and generally adopts fixed output ablation along the isthmus. In recent times, high power short duration ablation has been adopted. However, when deeper lesions are required, lower power for longer durations may be preferable.[16]

Secondly, the CS lies on the epicardial surface adjacent to the endocardial aspect of the mitral isthmus as it traverses the inferior left atrial wall approximately 1cm above the mitral valve annulus and becomes the great cardiac vein beyond the Valve of Vieussens and/or the bundle of Marshall.[14] The CS limits the achievement of bidirectional block across the mitral isthmus in two main ways. The CS has a myocardial sleeve of variable thickness (ranging from 0.3 to 2.5mm) which extends a variable distance from the CS

ostium (mean 40±8mm).[17] This cuff of muscle can act as an epicardial bridge, bypassing the endocardial mitral isthmus at the site adjacent to endocardial ablation. In addition, the CS blood pool can act as a 'heat sink' that reduces conductive heating of the subepicardium and limits lesion transmurality.[18] This idea is supported by previous studies demonstrating a reduced need for epicardial CS ablation when CS balloon occlusion is performed prior to endocardial ablation and that a larger CS diameter is associated with increased ablation time and the need for epicardial CS ablation to achieve bidirectional block.[19,20]

Finally, the bundle of Marshall, a fibromuscular sleeve that surrounds the vein of Marshall (VOM) as it traverses epicardially along the ridge between the LAA and left pulmonary veins (PVs), may have variable connections to the CS musculature and the left atrium (LA), thereby providing another source of epicardial connection which can prevent mitral isthmus block.[21] As a result of these anatomic challenges, between 48 and 97% of patients have been reported to require epicardial ablation within the CS in addition to endocardial ablation to achieve mitral isthmus block.[22-24] More recently, the convenient anatomic location of the VOM on the epicardial aspect of the mitral isthmus has been used to advantage. Valderrabano et al. demonstrated extensive lateral mitral isthmus ablation with the use of ethanol infusion into the VOM (VOM ETOH).[25,26]Following VOM ETOH, minimal ablation is then required at the mitral annular end of the isthmus to achieve bidirectional block.

While the left circumflex artery is separated from the CS by adipose tissue, this protective cushion is variable and in some patients, it lies in close proximity to the CS and may be susceptible to injury during ablation.[14] In a single-centre series, the incidence of symptomatic circumflex artery occlusion post mitral isthmus ablation was rare at 1 in 499 patients.[27] However, in a separate study, the incidence of asymptomatic coronary artery injury with mitral isthmus ablation was much higher at 28%.[28] Fortunately, all coronary stenoses resolved with intracoronary nitrates suggesting the injury was thermally-mediated vasospasm without permanent injury.[28] Ablation within the CS, proximity of the left circumflex artery to the CS and a small distal left circumflex artery diameter were risk factors for injury in this study.[28] While pulsed field ablation has been reported to reduce the risk of injury to non-myocardial tissue, left circumflex artery occlusion due to vasospasm has been reported due to PFA of the endocardial mitral isthmus line.[29]

Anterior Mitral Lines

The anterior mitral line may extend from the superior aspect of the mitral valve to the RSPV, a roof line or more laterally to the LSPV. The major advantage of this approach is that it avoids the epicardial connections of the CS muscle coat and bundle of Marshall. However, this approach is also associated with several anatomic challenges. The anterior mitral line is significantly longer than the lateral mitral isthmus line.[30] Furthermore, thick epicardial muscle bundles within Bachmann's bundle that extend anteriorly and invaginate the base of the LAA can make transmural ablation difficult. As opposed to the lateral line there is no ready access to the epicardial aspect of the anterior line. Finally, anterior mitral line ablation results in greater activation delay of the LAA compared with lateral mitral isthmus lines with some case reports describing transient ischaemic attacks and strokes in patients post anterior linear ablation despite appropriate oral anticoagulation.[30,31] If performed in conjunction with a lateral mitral isthmus line or in the presence of posterolateral scar, the LAA may become electrically isolated with a consequent increased risk of thromboembolic events.[32] Regarding the choice between an anterolateral or anteromedial line, the anterolateral line courses across the region of maximal myocardial thickness [14,33] Conversely, imaging studies have noted that ridges and diverticuli are found most frequently along the anteromedial ablation line making adequate contact and contiguous transmural ablation along this line difficult to achieve.[33] While the sinus node artery branch of the left circumflex artery may be found in close proximity to both the anteromedial and anterolateral lines, it is most commonly found nearer to the anteromedial line.[33] Generally the course of the anterior line is directed through the shortest route from the superior mitral valve transecting the region of low voltage towards a PV or roof line (see Figure 2).

Superolateral Mitral Line

This ablation line is less commonly used and extends from the posterior base of the LAA orifice adjacent

to the LSPV to the mitral annulus and has been associated with high rates of successful transmural block without the need for CS ablation, likely due to the CS myocardial sleeve terminating more medially.[34] Additionally, it may also interrupt epicardial connections within the ligament of Marshall.[34] However, the thin atrial myocardium over the superolateral aspect of the LAA increases the risk of cardiac tamponade.[34]

Our Approach

Diagnosis and Mapping

In the setting of either previous ablation lines or atrial scar, the utility of the 12-lead ECG in the diagnosis of peri-mitral flutter is limited. If the patient arrives in the laboratory in sinus rhythm, we attempt to induce atrial flutter with burst atrial pacing and programmed extrastimuli from the CS catheter. Induction of flutter can be challenging and is often preceded by short periods of AF. The CS activation pattern is usually the first clue to localise the AT. Proximal to distal CS activation is expected with counter-clockwise peri-mitral flutter but also seen with cavotricuspid isthmus-dependent flutter, right ATs and tachycardias that originate from the right PVs or interatrial septum. Distal to proximal activation occurs with clockwise peri-mitral re-entry or tachycardias originating locally from the lateral LA. Entrainment is the most important diagnostic manoeuvre and is more rapid and accurate than any form of 3-dimensional (3D) mapping. A post-pacing interval minus tachycardia cycle length of less than 20 milliseconds from the proximal, mid and distal CS electrodes is consistent with peri-mitral flutter and may be confirmed with left atrial mapping (see Figure 3). If not initially performed under general anaesthetic, this is initiated prior to transseptal access. We would then proceed with double transseptal, transesophageal echocardiogram (TEE) guided access using SL1 sheaths, one of which is subsequently exchanged for a steerable Agilis sheath. Thereafter, intravenous heparin is administered to target an activated clotting time of [?]350 seconds. A high-density multipolar mapping catheter is used to map the LA. Entrainment from the LA roof as well as points on the anterior and posterior walls is needed to exclude the possibility of dual or multi-loop tachycardias. High density 3D electroanatomic mapping is then performed to confirm activation patterns but predominantly to construct a voltage map to guide the ablation strategy.

Ablation

Ablation is performed using a 3.5mm irrigated force-sensing ablation catheter via a steerable Agilis sheath to ensure catheter stability and adequate tissue contact with 'point-by-point' ablation guided by 3D electroanatomic mapping. High frequency low volume ventilation is used to facilitate catheter stability. Ablation should not be performed in AF but rather during flutter or atrial pacing. Our decision regarding the location of our ablation line is determined by the voltage map with the lateral mitral isthmus line generally preferred in the setting of normal atrial voltages. An anterior mitral isthmus line is pursued only in the presence of extensive regions of anterior low voltage. A meta-analysis of peri-mitral flutter ablation studies showed no difference in rates of acute bidirectional mitral isthmus block or ablation time when comparing anterior and lateral mitral isthmus ablation lines but anterior lines were associated with a higher percentage of patients maintaining sinus rhythm during follow-up.[30]

When performing a lateral mitral isthmus line, ablation commences at a 4 o'clock position on the lateral mitral valve annulus and point by point ablation is completed pursuing the shortest line to the anterior aspect of the LIPV. If the left PVs have not already been isolated then wide antral circumferential ablation is completed first, prior to the lateral mitral line. Given the aim of obtaining deep, transmural lesions, we use 30-35W rather than higher power settings. A contact force of >10g is maintained at all times, targeting an ablation index (AI) or lesion size index (LSI) of 600 or 6, respectively. More often than not, mitral isthmus block is not achieved at the first pass and ablation is extended a variable distance superiorly along the perceived endocardial course of the ligament of Marshall to the junction of the left PVs. The line is then remapped and if there are no obvious 'gaps', we proceed to ablate within the CS. When ablating within the CS, the catheter is deflected towards the endocardial line such that atrial electrograms are seen on the distal electrode of the electrode catheter and the impedance closely monitored. Ablation lesions are delivered at 25W, watching closely for rises in impedance. We generally do not utilise CS balloon occlusion

or displacement. If mitral isthmus conduction is still present, we return to the LA and map predominantly just superior to the endocardial line and deploy an additional line. We then map, ideally using a high density multipolar mapping catheter, during CS distal pacing to determine the earliest breakthrough. In difficult cases, the ablation may be in close vicinity to the base of the LAA, corresponding to the endocardial aspect of the ligament of Marshall. When available, if mitral isthmus block is not achieved with endocardial ablation and ablation within the CS, we consider the use of VOM alcohol ablation. The approach to the lateral mitral isthmus ablation is summarized in Figure 4.

If opting to perform an anterior mitral isthmus line instead, we utilise 40W of power, aiming for a contact force of >10g and targeting an AI or LSI of 400 or 4-5, respectively. Shorter duration lesions are typically delivered at sites of low tissue voltage and longer duration lesions in the endocardial region adjacent to Bachmann's bundle. Typically with the assistance of a steerable sheath, ablation would begin at the superior mitral annulus with counter-clockwise rotation releasing the curve to ascend superiorly to the RSPV, LSPV or roof line, depending on the location and extent of the low voltage anteriorly.

Confirmation of Bidirectional Block

Lateral mitral isthmus block is suspected in the presence of widely split double potentials of fixed separation recorded along the length of the ablation line during pacing from the distal CS electrode. Differential pacing is then performed with the ablation or multipolar catheter placed in the LAA. In the presence of lateral mitral isthmus block, pacing from the LAA should result in the atrial electrogram being recorded earlier on the His electrode than the CS proximal electrode followed by counter-clockwise activation around the mitral annulus with proximal to distal CS activation (see Figure 5). The activation time should be identical when pacing in the reverse direction from the CS distal electrode to the catheter in the LAA. An activation map may then be completed immediately superior to the line to confirm latest activation immediately adjacent to the line. Finally, the stimulus-to-electrogram interval in the catheter placed in the LAA should be longer with pacing from the distal CS electrode than with pacing from a more proximal CS electrode.

To confirm *anterior mitral line block* , differential pacing manoeuvres may be performed. However, distinguishing conduction delay from complete block can be more challenging due to the length of the line. Widely split local double potentials of a fixed separation recorded by the ablation catheter would be expected along the length of the ablation line during pacing from a multipolar mapping catheter placed lateral to the line, typically in the LAA. Testing is then performed with the ablation catheter placed septal to the line. Pacing from the ablation catheter should result in a long activation time to the LAA. This activation time should be identical when pacing in the reverse direction from the LAA and recording from the ablation catheter septal to the line. When pacing from the LAA, there should be clockwise activation around the mitral valve annulus with distal to proximal CS activation. Conversely, when pacing from the ablation catheter, there should be counter-clockwise activation around the mitral valve annulus with proximal to distal CS activation. During pacing from the ablation catheter septal to the line, an activation map can be performed using the multipolar catheter to confirm lateral to septal activation with the latest electrograms recorded immediately adjacent to the line. It is very important to pace from two sites septal to the long anterior line, typically higher up adjacent to the right superior PV and lower down nearer to the mitral annulus. Conduction block may be falsely assumed if pacing a considerable distance from the site of breakthrough in the line.

Conclusions

Peri-mitral flutter is increasingly common in the presence of more extensive atrial substrate ablation for AF. Accordingly, mitral isthmus ablation should not be considered a first-line strategy for AF given the proarrhythmic nature of incomplete or non-transmural ablation lines. As newer tools and techniques such as VOM alcohol ablation and electroporation become more established, the role of empiric mitral isthmus ablation may evolve. Given the relative paucity of randomised evidence, mitral isthmus ablation should largely be reserved for patients with peri-mitral flutter.

References

1. Vlachos K, Denis A, Takigawa M, et al. The role of Marshall bundle epicardial connections in atrial tachycardias after atrial fibrillation ablation. Heart Rhythm 2019;16:1341–1347.
2. Takigawa M, Derval N, Martin CA, et al. Mechanism of recurrence of atrial tachycardia: comparison between first versus redo procedures in a high-resolution mapping system. Circ Arrhythm Electrophysiol 2020;13:e007273.
3. Deisenhofer I, Estner H, Zrenner B, et al. Left atrial tachycardia after circumferential pulmonary vein ablation for atrial fibrillation: incidence, electrophysiological characteristics, and results of radiofrequency ablation. Europace 2006;8:573–582.
4. Haissaguerre M, Hocini M, Sanders P, et al. Catheter ablation of long-lasting persistent atrial fibrillation: clinical outcome and mechanisms of subsequent arrhythmias. J Cardiovasc Electrophysiol 2005;16:1138–1147.
5. Chugh A, Oral H, Lemola K, et al. Prevalence, mechanisms, and clinical significance of macroreentrant atrial tachycardia during and following left atrial ablation for atrial fibrillation. Heart Rhythm 2005;2:464–471.
6. Jais P, Shah DC, Haissaguerre M, Hocini M, Peng JT, Takahashi A, Garrigue S, Le Metayer P, Clementy J. Mapping and ablation of left atrial flutters. Circulation. 2000 Jun 27;101(25):2928-34.
7. Calkins H, Hindricks G, Cappato R, et al. HRS/EHRA/ECAS/APHRS/SOLAECE expert consensus statement on catheter and surgical ablation of atrial fibrillation. Europace. 2017;2018(20):e1-e160.
8. Rostock T, Drewitz I, Steven D, et al. Characterization, mapping, and catheter ablation of recurrent atrial tachycardias after stepwise ablation of long-lasting persistent atrial fibrillation. Circ Arrhythm Electrophysiol 2010;3:160–9.
9. Wasmer K, Monnig G, Bittner A, et al. Incidence, characteristics, and outcome of left atrial tachycardias after circumferential antral ablation of atrial fibrillation. Heart Rhythm 2012;9:1660–6.
10. Hermida A, Kubala M, Traulle S, et al. Prevalence and predictive factors of left atrial tachycardia occurring after second-generation cryoballoon ablation of atrial fibrillation. J Cardiovasc Electrophysiol 2018;29:46–54.
11. Matsuo S, Lim KT, Haissaguerre M. Ablation of chronic atrial fibrillation. Heart Rhythm. 2007 Nov;4(11):1461-3.
12. Fassini G, Riva S, Chiodelli R, Trevisi N, Berti M, Carbucicchio C, et al. Left mitral isthmus ablation associated with PV isolation: long-term results of a prospective randomized study. J Cardiovasc Electrophysiol. 2005;16(11):1150–6.
13. Chae S, Oral H, Good E, et al. Atrial tachycardia after circumferential pulmonary vein ablation of atrial fibrillation: mechanistic insights, results of catheter ablation, and risk factors for recurrence. J Am Coll Cardiol. 2007;50:1781-1787.
14. Becker AE. Left atrial isthmus: anatomic aspects relevant for linear catheter ablation procedures in humans. J Cardiovasc Electrophysiol. 2004;15:809-12.
15. Latcu DG, Squara F, Massaad Y, Bun SS, Saoudi N, Marchlinski FE. Electroanatomic characteristics of the mitral isthmus associated with successful mitral isthmus ablation. Europace. 2016 Feb;18(2):274-80.
16. Leshem E, Zilberman I, Tschabrunn CM, Barkagan M, Contreras-Valdes FM, Govari A, et al. High-power and short-duration ablation for pulmonary vein isolation: biophysical characterization. JACC: Clin Electrophysiol. 2018;4(4):467–79.
17. Chauvin M, Shah DC, Haissaguerre M, Marcellin L, Brechenmacher C. The anatomic basis of connections between the coronary sinus musculature and the left atrium in humans. Circulation. 2000;101(6):647–52.
18. Wittkampf FH, van Oosterhout MF, Loh P, Derksen R, Vonken EJ, Slootweg PJ, et al. Where to draw the mitral isthmus line in catheter ablation of atrial fibrillation: histological analysis. Eur Heart J. 2005;26(7):689–95.
19. Wong KC, Jones M, Qureshi N, Sadarmin PP, De Bono J, Rajappan K, et al. Balloon occlusion of the distal coronary sinus facilitates mitral isthmus ablation. Heart Rhythm. 2011;8(6):833–9.
20. Wong KC, Jones M, Sadarmin PP, De Bono J, Qureshi N, Rajappan K, et al. Larger coronary

sinus diameter predicts the need for epicardial delivery during mitral isthmus ablation. Europace. 2011;13(4):555–61.
21. Kim DT, Lai AC, Hwang C, Fan LT, Karagueuzian HS, Chen PS, et al. The ligament of Marshall: a structural analysis in human hearts with implications for atrial arrhythmias. J Am Coll Cardiol. 2000;36(4):1324–7.
22. Matsuo S, Wright M, Knecht S, Nault I, Lellouche N, Lim KT, et al. Peri-mitral atrial flutter in patients with atrial fibrillation ablation. Heart Rhythm. 2010;7(1):2–8.
23. Weerasooriya R, Jais P, Wright M, Matsuo S, Knecht S, Nault I, et al. Catheter ablation of atrial tachycardia following atrial fibrillation ablation. J Cardiovasc Electrophysiol. 2009;20(7):833–8.
24. Anousheh R, Sawhney NS, Panutich M, Tate C, Chen WC, Feld GK. Effect of mitral isthmus block on development of atrial tachycardia following ablation for atrial fibrillation. Pacing Clin Electrophysiol. 2010;33(4):460–8.
25. Baez-Escudero JL, Morales PF, Dave AS, Sasaridis CM, KimYH, Okishige K, et al. Ethanol infusion in the vein of Marshall facilitates mitral isthmus ablation. Heart Rhythm. 2012;9(8):1207–15.
26. Briceno DF, Valderrabano M. Recurrent perimitral flutter due to vein of Marshall epicardial connections bypassing the mitral isthmus: response to ethanol infusion. Circ Arrhythm Electrophysiol. 2014;7(5):988–9.
27. Takahashi Y, Jais P, Hocini M, Sanders P, Rotter M, Rostock T, et al. Acute occlusion of the left circumflex coronary artery during mitral isthmus linear ablation. J Cardiovasc Electrophysiol. 2005;16(10):1104–7.
28. Wong KC, Lim C, Sadarmin PP, Jones M, Qureshi N, De Bono J, et al. High incidence of acute subclinical circumflex artery 'injury' following mitral isthmus ablation. Eur Heart J. 2011;32(15):1881–90.
29. Gunawardene MA, Schaeffer BN, Jularic M, Eickholt C, Maurer T, Akbulak RO, Flindt M, Anwar O, Hartmann J, Willems S. Coronary Spasm During Pulsed Field Ablation of the Mitral Isthmus Line. JACC Clin Electrophysiol. 2021 Dec;7(12):1618-1620.
30. Aldass O, Lupercio F, Lin AY, et al. Ablation of mitral annular flutter ablation utilizing a left atrial anterior line versus a lateral mitral isthmus line: a systematic review and metaanalysis. J Interv Card Electrophysiol. 2022;63(1):87–95.
31. Feld GK. Alternative Approaches for Ablation of the Mitral Isthmus for Treatment of Atrial Fibrillation. JACC Clin Electrophysiol. 2022 Mar;8(3):377-379.
32. Di Biase L, Mohanty S, Trivedi C, et al. Stroke risk in patients with atrial fibrillation undergoing electrical isolation of the left atrial appendage. J Am Coll Cardiol. 2019;74:1019–1028.
33. Cho Y, Lee W, Park EA, Oh IY, Choi EK, Seo JW, et al. The anatomical characteristics of three different endocardial lines in the left atrium: evaluation by computed tomography prior to mitral isthmus block attempt. Europace. 2012;14(8):1104–11.
34. Maurer T, Metzner A, Ho SY, et al. Catheter ablation of the superolateral mitral isthmus line: a novel approach to reduce the need for epicardial ablation. Circ Arrhythm Electrophysiol. 2017;10.

Figures

Figure 1: Ablation Line Options for the Management of Peri-Mitral Flutter

Ablation line options schematically demonstrated on reconstructed computed tomography images of the left atrium

Left: Antero-posterior view. AM, anteromedial line; TA, true anterior line; AL, anterolateral line

Right: Left lateral view. SL, superolateral line; L, lateral line

Hosted file

`image1.emf` available at https://authorea.com/users/541097/articles/600450-managing-peri-mitral-flutter

Figure 2

Hosted file

`image2.emf` available at https://authorea.com/users/541097/articles/600450-managing-peri-mitral-flutter

Figure 3

Hosted file

`image3.emf` available at https://authorea.com/users/541097/articles/600450-managing-peri-mitral-flutter

Figure 4: Approach to Lateral Mitral Isthmus Ablation

LAA, left atrial appendage; LIPV, left inferior pulmonary vein; RIPV, right inferior pulmonary vein; MA, mitral annulus; LOM, ligament of Marshall; LSPV, left superior pulmonary vein; CS, coronary sinus; LA, left atrium; DCR, direct current cardioversion; SR, sinus rhythm

Hosted file

`image4.emf` available at https://authorea.com/users/541097/articles/600450-managing-peri-mitral-flutter

Figure 5: Electrograms During Lateral Mitral Isthmus Ablation

Left: During left atrial appendage (LAA) pacing post endocardial lateral mitral isthmus ablation, coronary sinus (CS) activation is distal to proximal suggesting persistent conduction across the lateral mitral isthmus

Middle: During LAA pacing post additional ablation along the perceived endocardial course of the ligament of Marshall to the junction of the left pulmonary veins, the stimulus-to-electrogram interval on the CS electrodes has increased but the CS activation pattern appears fused with CS 5-6, CS 3-4 and CS distal activated almost simultaneously, suggesting slowed but persistent conduction across the lateral mitral isthmus

Right: During LAA pacing post additional ablation within the CS, the stimulus-to-electrogram interval on the CS electrodes increases further and the CS activation pattern is now clearly proximal to distal consistent with block across the lateral mitral isthmus

Hosted file

`image5.emf` available at https://authorea.com/users/541097/articles/600450-managing-peri-mitral-flutter

Hosted file

`Figures.pptx` available at https://authorea.com/users/541097/articles/600450-managing-peri-mitral-flutter