Anatomical basis of minimally invasive epicardial ablation of atrial fibrillation

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Summary

Minimally invasive atrial fibrillation surgery (MIAFS) has become a well established and increasingly used option for managing patients with stand-alone arrhythmia. Pulmonary veins (PVs) isolation continues to be the cornerstone of ablation strategies. Indeed, in most cases, atrial fibrillation (AF) is triggered in or near the PVs. Nevertheless, ectopic beats initiating AF may occasionally arise from non-PV foci. The knowledge of the anatomy and underlying morphology of PVs and non-PV foci is essential for cardiac surgeons treating AF patients with epicardial minimally invasive procedures. The anatomical structures relevant to the pathogenesis and the epicardial treatment of AF include the PVs, the pericardial space, the pericardial sinuses, the phrenic nerve, the left atrium, the retro-atrial and caval ganglionated plexuses, the ligament of Marshall, the caval veins and the left atrial appendage. In this review, we briefly describe the basic anatomy of these structures and discuss their specific correlations for cardiac surgeons interested in performing MIAFS.

Keywords: Atrial fibrillation • Ablation • Anatomy

INTRODUCTION

With the introduction of different energy sources [1] and the development of new tools and advanced techniques, the treatment of atrial fibrillation (AF) has moved towards minimally invasive procedures as an alternative to catheter ablation [2] and the original ‘cut and sew’ technique [3]. Minimally invasive atrial fibrillation surgery (MIAFS) is performed on the beating heart and offers many benefits including less postoperative pain, faster recovery, reduced risk of complications and shorter hospital stay and can be offered as a stand-alone procedure in patients who do not need open heart surgery for another diagnosis [4].

However, performing MIAFS requires an expertise and a skill set that are significantly different from that required to perform traditional surgery, and a complete knowledge of the anatomy and underlying morphology of the heart structures relevant to the pathogenesis and epicardial treatment of AF is mandatory to carry out MIAFS.

Here, we present a review that summarizes anatomical features of particular relevance for cardiac surgeons who want to perform MIAFS. Supplementary Videos are provided online.

THE PERICARDIAL SPACE AND THE PHRENIC NERVE

The pericardial cavity is a potential space between the parietal and visceral layers of the serous pericardium. It is continuous with the epicardium and reflects around the roots of the great vessels and onto the visceral surface of the fibrous pericardium, which is continuous with the adventitia of the great vessels superiorly and related posteriorly to the bronchi, esophagus, descending thoracic aorta and mediastinal surface of each lung [5].

The phrenic nerve (PN) originates mainly from the fourth cervical nerve, but also receives contributions from the fifth and third cervical nerves. The PN travels inferiorly in the neck on the surface of the anterior scalene muscle entering the superior mediastinum behind the subclavian vein and passing under the internal thoracic artery. On the right, it passes lateral to the right brachiocephalic vein, the superior vena cava (SVC), then the right atrium (RA) and the inferior vena cava (IVC). It reaches the under surface of the diaphragm by passing through the vena caval foramen in the central tendon. The left PN passes through the superior mediastinum lateral to the left subclavian artery and the aortic arch. It crosses the arch lateral to the superior intercostal vein and in front of the vagus nerve and then runs laterally down the pericardium over the left ventricle towards the apex of
the heart. It descends over the antero-lateral angle of the pericardial base to reach the diaphragm at a muscular or tendinous area.

When PNs enter the middle mediastinum, they pass anteriorly to the root of the lung across the pericardium. From a thoracoscopic surgical view, the right PN runs much closer to the hilum while, on the left side, it runs midway between the hilum and the anterior chest wall (Fig. 1A and B). The pericardium is accessed through left and right thoracoscopy: it is grasped and pulled away from the heart and opened longitudinally from the SVC to the IVC with an endoscopic coagulation hook 2 cm anterior to the right PN (Fig. 1C) and posterior to the left PN on the left side (Fig. 1D).

**THE PERICARDIAL SINUSES**

The oblique sinus (OS) is a recess located behind the left atrium (LA), which is formed by the reflection of the serous pericardium around the large veins (pulmonary veins [PVs] and caval veins).

The OS communicates with the main pericardial space inferiorly. To the left, the fold of Marshall may be seen, as well as the left-sided PVs located lateral to the fold of Marshall in the superior half of the OS, with the inferior PV in closer proximity to its lateral limit. To the right, the sinus is related to regional-specific epicardial fat-ganglionated plexuses (GP) and the right PVs. Immediately behind the OS, the oesophagus and the plexal branches of the vagus nerve may be accessed [6].

The OS is entered by dissection of the pericardial reflection of the IVC (Fig. 2A and B). From within the OS, the posterior wall of the LA with its retro-atrial fat housing the retro-atrial GP can be accessed anteriorly.

The transverse sinus (TS) is located superiorly to the heart between the arterial mesocardium, which envelopes the ascending aorta and pulmonary trunk anteriorly, and the venous mesocardium, which covers the SVC, LA and PVs posteriorly and inferiorly.

This anatomical space is a passage from the left side to the right side of the pericardial cavity, which lies behind the great arteries [7]. Superiorly within the roof of the TS lies the right pulmonary artery as it passes to the right beneath the aortic arch. Inferiorly, the floor of the TS is formed by the roof of the LA,
specifically the transverse fibers that constitute Bachmann’s bundle. The posterior wall of the ascending aorta at the level of the superior margin of the aortic cusps or sinotubular junction lies anteriorly (Fig. 2C). To the left and in front lies the main pulmonary artery with the SVC and its small continuing recess called the aorto-caval sinus, to the right [8, 9].

To access the TS, the pericardial reflection of the SVC is dissected and an opening is created by gently rubbing the fat in the triangle created by the right pulmonary artery, the SVC and the RA (Fig. 2D).

THE PULMONARY VEINS

The PVs enter the posterior part of the LA with the left veins located more superior than the right veins. Anatomical studies and also studies using magnetic resonance and computed tomography imaging have reported significant variability in dimensions, shape and branching patterns of the PVs. Typical PV anatomy with four distinct ostia is present in approximately 20–60% of subjects. A frequent anatomical variation is the presence of a short or long common left trunk, observed in up to 75–80% of patients (Fig. 3). Additional abnormalities of the PV anatomy include the presence of a right middle PV, two right middle PVs or a right middle and a right ‘upper’ PV (an anomalous vein distinct from the right superior PV [RSPV]) [10–13].

The left superior PV (LSPV) lies immediately posterior to the left atrial appendage (LAA). Although the ostium of this vein is typically slightly posterior to that of the lower left vein, the main branch of the LSPV courses anteriorly to the plane of the coronary sinus (CS), and thus the anterior wall of this vein is apposed for variable length with the posterior wall of the proximal LAA. The left inferior PV (LIPV) may drain with the LSPV into a common antrum. When the veins are distinct, the proximal portion may form a carina-like structure or the inferior wall of the LSPV and superior wall of the LIPV remain apposed for a short distance (3–5 mm). The LIPV is also posterior to the posteroinferior portion of the proximal LAA. The vein/ligament of Marshall (LOM) separates the left-sided veins from the posterior wall of the LAA ostium. The RSPV courses immediately posterior to the RA and the junction of the SVC with the RA as it enters the LA from the right lung. The azygous vein, as well as a branch...
of the right pulmonary artery, is superior and posterior to the
vein. The RSPV vein rarely drains as the common antrum, where
the right inferior vein and the vein draining the right middle
lobe typically enter in the right upper vein approximately 1 cm
from the ostium. The right inferior PV is the only vein, where
the corresponding pulmonary arterial branch courses anterior to the
vein, thus separating it from other myocardial structures [14].

The transition from the atrial endocardium to the venous
endothelial layer is basically smooth. However, irregular atrial
sleeves of cardiomyocytes extend over the venoatrial junction
into the PVs walls. The longest sleeves are more frequently
encountered at the superior PVs, and Ho et al. [14, 15] have
described an average length of 5.5 mm at the inferior PVs and
10 mm at the superior PVs, although the maximum length can
achieve 25 mm. The sleeves are thicker at the venoatrial junction
and usually composed of circularly oriented fibres in the trans-
mural extension. However, there are also fascicles of longitudinal
and oblique fibres inter-digitated between the circular bundles
that can extend from the subepicardium to the subendocardium
[14]. This sudden change in the orientation of fibres can generate
non-uniform anisotropy and be a substrate for micro-re-entry, as
well as allow the propagation of impulse from a focal source
[16]. Perez-Lugones et al. [17] demonstrated the presence of
node-like cells in the PVs of 4 patients with a history of AF,
which was not found in the other 6 patients without AF. By elec-
tron microscopy, they identified pale cells with ultrastructural
features similar to sinus node and conduction system cells. More
recently, interstitial Cajal cells and Periodic acid-Schiff-positive
cells that might have a pacemaker function have been described
in human PVs, particularly in patients with AF [18, 19]. In contrast
to patients in sinus rhythm, these cells were separated by inflam-
matory infiltration and fibrosis [20].

This structure is extremely significant because electrical activity
has been demonstrated in venous sleeves although, thus far, it
has not been possible to affirm that these cells mediate the
automaticity seen at the PVs.

The video-assisted thoracoscopic surgical technique allows the
electrical isolation of the PVs bilaterally, and bipolar radiofre-
quency clamps are more frequently employed for the PVs epi-
cardial isolation due to the transmurality of lesions achieved on
the beating heart using this energy source [21].

The right PVs are accessed first. A blunt dissection is per-
formed between the RSPV and the right pulmonary artery,
lateral to the SVC, to gently separate these structures.

Before starting the ablation, the fat pad in the PVs antrum is
bluntly dissected to provide a better placement of the clamp
and to enhance a higher penetration energy.

A special articulating lighted dissector has been developed for
this procedure, which can be placed under the inferior PV and
emerge above the superior PV but below the pulmonary artery
and lateral to the SVC. Once the dissector is around the right
PVs, a red rubber catheter, already attached to the dissector, is
advanced, pulling the catheter behind the veins. The lighted
dissector is then removed, and the red rubber catheter is used to
guide the lower jaw of the clamp behind the left atrial cuff adja-
cent to the right PVs. The rubber catheter is then removed, and
correct positioning of the clamp on the atrium and not on the
PVs is verified by means of direct inspection of the device after
closing the jaws of the clamp. The technique is repeated on the
left side (Fig. 4A) with the addition of a division of the LOM. The
endpoint of PVs isolation is entrance and exit blocks. Entrance
block is defined as the absence of atrial potentials inside the
area of the lesions. Exit block is defined as failure to capture the
remaining LA when pacing from the posterior LA [21].

**LEFT ATRIUM**

The LA is located posteriorly and superiorly to the RA. The pos-
terior surface of the LA between the PVs forms the anterior wall
of the OS of the pericardium. The fibrous pericardium separates
this surface from the oesophagus. The transverse pericardial
sinus lies anterior to the LA, and in front of the sinus is the root
of the aorta. The anterior wall accommodates the aortic root in
a slight curvature that continues from the RA to the LAA; poster-
ioinferiorly, it is connected to the CS [16]. The LA is divided intoﬁve components: the septum, the appendage, the vestibule and
the venous component. From the left view, the flap valve of the
foramen ovale represents the atrial septum. The vestibular com-
ponent is the area surrounding the mitral valve (MV). Posteriorly,
overlies the left atrioventricular (AV) groove where the CS ends

![Figure 3: Schematic drawing of the most common patterns of PV in AF: (A) normal pattern; (B) short common left trunk; (C) long common left trunk; (D) right middle PV; (E) two right middle PVs; (F) right middle PV and right upper PV.](image-url)
at the great cardiac vein [10]. The venous component receives the PVs and is the major part of the LA.

The general myoarchitecture of the LA is highly complex and varies from heart to heart. However, some components are fairly constant: (i) The Bachmann’s bundle; (ii) myofibers originating from the fossa ovalis; (iii) the septopulmonary bundle; (iv) the septoatrial bundle.

The prevalent interatrial conduction pathway for propagation of the sinus impulse to the anterior left atrial wall is through the Bachmann’s bundle, which is also known as the interauricular band [22]. Indeed, it is the most prominent muscular interatrial bridge in the majority of hearts [22]. It is superficially located and composed of nearly parallel alignments of myocardial strands that blend into the musculature of the atrial walls. It branches in its rightward and leftward extensions to embrace the atrial appendages. After passing around the neck of the LAA, the arms rejoin to continue into the musculature of the lateral and posteroinferior atrial walls [16]. The only place, where the Bachmann’s bundle appears as quite a distinct bundle, separated by fatty tissues from the atrial wall, is at the anterior intertribal groove. The myocardial architecture with parallel fibres on Bachmann’s bundle and the fibres around the PVs and on the atrial septum create distinct patterns of electrical activation in the LA during sinus rhythm, with the posterior wall often often the last site to be activated. Although no specialized conduction tissue exists at this site, because of the fibre orientation and proximity posteriorly with the sinus node in the RA, this structure represents the important conducting mechanism from one atrium to the other.

The myofibers originating from the anterior rim of the fossa ovalis are deeper and more inferior to the Bachmann’s bundle and join it to reach the LA lateral wall and septal raphe after encircling the LAA.

The septopulmonary bundle is an array of longitudinally and obliquely oriented cardiomyocytes arising from the antero-superior septal raphe and passing deep to the Bachmann’s bundle before surfacing on the atrial roof; it fans out and runs in the region of the PV insertions and posteriorly fuses with the circumferential myofibers coming from the LA lateral wall.

The septoatrial bundle lies more deep than the septopulmonary bundle, arises from the anterior septal raphe, obliquely combines with the myofibers of the anterior vestibule and myofibers from the septopulmonary bundle at the atrial roof and continues up the LA posterior wall after passing trough the region of the PV orifices.

The mitral isthmus refers to the atrial myocardium between the MV annulus and the left-sided PVs (Fig. 4B). Anatomically, since this isthmus extends into the LIPV, the width of the isthmus will depend on the extent of the myocardial sleeves associated with this vein. The wall of the isthmus ranges from 2 to 8 mm in myocardial thickness. During MIAFS, ablation of the isthmus is performed in the case of persistence of AF starting from the ablation line on the antrum of the LIPV using a unidirectional bipolar pen (Fig. 3). In case of hybrid procedures (which combine a thoracoscopic epicardial ablation with a percutaneous trans-septal procedure in one step), this line can be completed by the electrophysiologist endocardially from the mitral annulus towards the CS [4]. The endpoint of the ablation of the mitral isthmus is the bidirectional block.

Different left atrial lesions may be performed during MIAFS (Fig. 5). They include a roof line connecting the superior PVs...
and an inferior line connecting the inferior PVs [24,25]. These lesions are made with a liner pen or a multidirectional bipolar pen and complete the PVs isolation to create a ‘box lesion’. Other lines that can be carried out endocardially are: a connecting line between the superior line (of the box) and the left fibrous trigone [24,26,27], a connecting line from the superior PV and the base of LAA [26, 28–31] and a line from the right inferior PV to the CS [32].

**RETRO-ATRIAL GANGLIONATED PLEXUSES**

Electrophysiological studies have found that local autonomic ganglia clustered in the epicardial fat pads play a critical role in the initiation and maintenance of AF [33,34]. The retro-atrial ganglia are composed of multiple separate plexuses (GP) distributed over the posterior surfaces of both the right and the left atria. These plexuses innervate PV myocardial sleeves and the adjacent atrial muscle and consist of anatomically closely related ganglia with extensive neural interconnections. The major GP include the superior right atrial, superior left atrial, posterior right atrial, posteromedial right atrial, posteromedial left atrial, interatrial and posterolateral left atrial GP (Figs 6 and 7A and B) [35]. The distribution of ganglia may also be considered in light of their proximity to the sinoatrial and AV nodes and divided into para-senoatrial (SA) nodal and para-AV nodal ganglia [36].

One of the difficulties in studying the significance of these ganglia in electrophysiological processes has been the complex interactions between them. Numerous studies have shown that there is extensive unidirectional and bidirectional feedback between ganglia [37, 38]. However, there is clinical evidence that ablation of the main GP on the atria increases the success of standard PV isolation by catheter ablation for AF [20]. Nevertheless, there is no way to directly visualize the ganglia during catheter ablation, and surrogate markers currently used during endocardial mapping (such as complex fractionated electrograms or induced parasympathetic responses during high-frequency stimulation) are of unclear specificity for the presence of ganglia [39, 40] and it is uncertain with what degree of success the ganglia are actually being ablated. In contrast, pericardial approaches to the autonomic ganglia may permit both more direct access and stimulation. The ganglia are localized using high-frequency stimulation and ablation and are approached in their primary location in the pericardial space [26]. The endpoint for GP ablation is the elimination of a vagal response to high-frequency stimulation. If, after the ablation of PVs and creation of a ‘box lesion’, the vagal response is still present, the ablation can be completed employing a linear pen.

However, the long-term efficacy of ganglion ablation has been questioned [41]. A canine study using radiofrequency ablation reported that AF inducibility was eliminated immediately after GP ablation, but this denervation effect was reversed within 4 weeks after the ablation [42], and more recently, it has been confirmed that, after surgical ganglion ablation, there is evidence of reinnervations at 4 weeks [43].

**CAVAL VEINS**

The SVC is about 7 cm long with an average SCV diameter of 20–22 mm. It commences at the low border of the first right costal cartilage with the confluence of the two brachiocephalic veins. The SVC passes vertically downwards behind the right border of the sternum and, piercing the pericardium, enters the upper border of the RA at the lower border of the third right costal cartilage. Behind the sternal angle, opposite the second right costal cartilage, it meets the azygos vein arches forwards over the root of the right lung. Developmentally, the SVC is formed as the persisting right anterior cardinal vein, but below the entrance of the azygos vein, and represents the persisting right common cardinal vein of the embryo.

The IVC commences opposite the L5 vertebra at a slightly lower level than the bifurcation of the aorta, by the confluence of the right and left common iliac veins behind the right common iliac artery. It runs on the right of the aorta, upwards beyond the aortic opening of the diaphragm, from which level it lies on the right crus behind the bare area of the liver, and extends to the central tendon of the diaphragm, which it pierces on a level with the body of T8 vertebra. IVC drains into the RA about after about 22–25 cm (4–5 cm from the diaphragm muscle). The average ICV diameter is variable at its different levels, being larger in its intrathoracic portion (about 30–32 mm) [44]. Unlike the SCV, the ICV ostium is secured by the vestigial Eustachian valve (sometimes, as a fenestrated Chiari network), a triangular fibromuscular flap that extends from the lateral part of the orifice and reaches the border between the CS and the oval fossa [45].

AF is frequently triggered from arrhythmogenic foci originating from the SCV [46]. Indeed, 37% of the non-PV triggers were from the SVC [47], whereas the IVC is less frequently responsible for the initiation and maintenance of AF [48].

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**Figure 6:** Schematic drawing of retro-atrial GP. 1: superior right atrial in the posterior superior surface of the RA adjacent to the junction of the SVC and the right; 2: superior left atrial in the posterior surface of the LA between the PVs; 3: posterior right atrial in the posterior surface of the RA adjacent to the interatrial groove; 4: posteromedial left atrial in the posterior medial surface of the LA; 5: posterolateral left atrial in the posterior lateral surface of the LA based on the atrial side of the AV groove, 6: interatrial: fusion of the posterior right and posteromedial left atrial GP, extending interiorly into the interatrial septum.
However, atrial myocardial sleeves were found in both caval veins [49]. In general, the most frequent pattern was a continuous transition between atrial myocardium and venous cardiac muscle. Overall, muscle sleeves were located on the outer side of venous adventitia in all cases. Myocardial fibers in these sleeves were organized predominantly circularly to the long axis of the vein, were frequently discontinuous and no specialized conduction cells were observed. Atrio-caval junction evaluation revealed a predominantly continuous pattern in the SCV in few subjects, whereas discontinuity was present in the IVC, and, finally, no major differences in the characteristics of myocardial sleeves were found between patients with and without AF [49]. Interestingly, atrial myocardial sleeves may occasionally reach the ostium, or the distal portion, of the azygous vein [13].

Circumferential caval lesions and linear SVC-to-IVC lesions represent possible strategies during MIAFS. SVC circumferential and SVC-to-IVC lesions may be indicated in patients with persistent and long-standing persistent AF who have large right atrial volume [25]. An IVC circumferential isolation is performed in patients with a small portion of intrapericardial IVC to be sure that the SVC–IVC line would stop at an area of no conduction [25]. The isolation of the SVC and the IVC is confirmed by the testing of the conduction block across the ablation lines [50]. The completeness of the SVC-to-IVC line could not be tested during standard MIAFS because of the technical difficulty of proving this without using multipolar electrophysiology (EP) catheters and EP analysing equipment, whereas it could be easily tested during hybrid procedures [4].

CAVAL GANGLIONATED PLEXUSES

Some ganglia are present beyond the SVC/atrial junction. Most ganglia around the SVC are located near the junction of the RA and inferior to the SA node (Fig. 7C). In turn, IVC-related ganglia are located along the medial surface of the IVC, proximal to the para-AV nodal ganglia and along the inferior surface of the IVC proximal to the CS (Fig. 7D). For example, those located in the vicinity of the SVC/atrial junction, when transected, may interrupt autonomic innervations to the sinoatrial node but not to the AV node, which is more affected by ganglia located in the vicinity of the IVC and LA.
**LIGAMENT OF MARSHALL**

The LOM, or oblique vein of the LA, is a remnant of the left SVC located on the epicardium between the LAA and the left PVs. The ligament is connected by the fetal remnant of the duct of Couvier to the highest left intercostal vein, and it descends along the postero-lateral wall of the LA and drains into the CS [5]. In the majority of cases, this vessel is obstructed and transformed into a fibrous chord, is a source of paroxysmal AF [51] and may activate at fast rates during persistent AF. It contains muscle bundles (Marshall bundles) that directly connect to the atrial myocardium and CS muscle sleeves. These muscle structures can serve as a source of triggers and drivers for AF and may form the substrates of re-entry. In some patients, these muscle bundles take part in accessory pathway conduction in patients with pre-excitation syndrome. Furthermore, the remnant of the vein of Marshall is covered over by fatty tissues containing abundant autonomic nerve bundle and ganglia [52]. The ridge between the LAA and LSPV and LOM may not be easily ablated by the endocardial approach [53, 54], whereas LOM is accessible by the epicardial approach and can be localized using high-frequency stimulation and easily eliminated during minimally invasive procedures [30].

**LEFT ATRIAL APPENDAGE**

The left atrial appendage is a long and narrow finger-like structure with multiple crenellations with a variable number of lobes. Its endocardial aspect is lined with muscle bundles of varied thicknesses like to the pectinate muscle of the RA, but they are arranged in a whorl-like fashion instead of an array since there is no equivalent of crista terminals in the LA [16]. It is widely believed that formation and embolism of LAA thrombi are responsible for the increased risk of stroke in AF patients [55]. Indeed, it has been reported that 90% of patients with non-rheumatic AF thrombi were located in the LAA, and this structure is of extreme importance for stroke prophylaxis [56]. Furthermore, LAA is also source of local atrial tachycardia after the ablation of long-standing persistent AF [57]. In addition, extra-PV atrial foci after PV isolation originate from the appendage, and the junctional area between the LAA and LA body is important in the AF process, acting as a source of activity spreading to the rest of the atrium [58, 59]. Compared with subjects in sinus rhythm, patients with AF have an LAA with a volume three times larger [60]. Furthermore, in these patients, the endocardial surface is smoother and associated with more extensive fibroelastosis. These features may contribute to LAA dysfunction and thrombus formation [60]. Excision or exclusion of the LAA is recommended in American College of Cardiology/ American Heart Association (ACC/AHA) guidelines [61] and is currently performed during the surgical ablation of AF. Therefore, there has been a great interest in the development and assessment of endocardial and epicardial procedures for exclusion of the LAA [62]. Many of MIAFS approaches now use a stapler to excise the appendage or, in some instances, endocardial suture exclusion [25, 27, 63]. Nevertheless, LAA excision/ligation may be difficult during minimally invasive surgery, and there has been growing concern regarding the safety of this manoeuvre. This has led to the development of new surgical tools, making this potentially dangerous manoeuvre easier [64–66].

**CONCLUSIONS**

The surgical treatment of AF has undergone dramatic changes over the last decade. New technologies have allowed the creation of transmural lesions on a beating heart through alternative, less-invasive incisions, and MIAFS is being largely used for the management of the patient with stand-alone AF. These procedures, however, require intimate knowledge of the regional anatomy of the LA, PVs, autonomic ganglia and thoracic veins to approach the epicardial surface of the heart. In this review, we have described the basic anatomy of these structures and their relationship to one another and to nearby structures, in relation to MIAFS.

**SUPPLEMENTARY MATERIAL**

Supplementary material (Videos 1 and 2) is available at EJCTS online.

Video 1: Right thoracotomy, opening of the pericardium, access to pericardial sinuses, ablation of right pulmonary veins and creation of right atrial lesions.

Video 2: Left thoracoscopy. Opening of the left pericardium, isolation and ablation of left pulmonary veins.

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