CHAPTER 1

Anatomy of structures relevant to atrial fibrillation ablation

Siew Y. Ho¹, Cristina Basso², José A. Cabrera³, Andrea Corrado⁴, Jeronimo Farré⁵, Josef Kautzner⁶, Roberto De Ponti⁷

¹Cardiac Morphology Department, Royal Brompton Hospital, Imperial College, London, UK
²Cardiovascular Pathology Department, University of Padua Medical School, Padua, Italy
³Arrhythmia Unit, Cardiology Department, Quirón Hospital, Universidad Europea de Madrid, Madrid, Spain
⁴Cardiology Department, Dell’Angelo Hospital, Venice-Mestre, Italy
⁵Cardiology Department, Jiménez Díaz-Capio Foundation, Madrid, Spain
⁶Cardiology Department, Institute for Clinical and Experimental Medicine, Prague, Czech Republic
⁷Cardiology Department, Circolo Hospital and Macchi Foundation, Varese, Italy

Introduction

Over the last few years, PVs have represented the cornerstone for catheter ablation of AF. Therefore, research has focused on their anatomy, histology, and peculiar electrophysiologic features. The data gathered from these studies have provided new insights in their morphologies and electrical function with a parallel improvement in patient care. However, as the ablation treatment of AF increases, the electrophysiologists’ interest has moved also to other structures that are directly or indirectly involved in the AF ablation procedures. These structures may be of interest for the access to the LA (atrial septum/fossa ovalis), for their role as sources of atrial ectopic activities (SVC, LAA/ligament of Marshall), for their implications in the ablation strategy (mitral isthmus) or in interatrial conduction (accessory interatrial connection pathways), for their role in the pathophysiology of AF (GP), and for their possible involvement in severe complications (PNs and esophagus).

In this chapter, after describing the morphology of the LA and PVs, we focus on the above-mentioned anatomical structures, which have become of interest for the electrophysiologist in the perspective of AF ablation procedures.
2 Atrial Fibrillation Ablation, 2011 Update

**Left atrium**

The LA has a venous component that receives the PVs, a finger-like atrial appendage, and shares the septum with the RA. The major part of the atrium, including the septal component, is relatively smooth-walled, whereas the appendage is rough with pectinate muscles (Figure 1.1). The smoothest parts are

![Figure 1.1](image)

**Figure 1.1** (a) The endocast viewed from the posterior aspect shows the proximity of the right PVs (RS and RI) to the atrial septum. Note also the RPA immediately above the roof of the LA. (b) The endocast viewed from the left shows the rough-walled LAA and its relationship to the LS. The CS passes inferior to the inferior wall of the LA. (c) to (e) are variations of PV arrangement from CT angio: (c) separate PVs on left side, (d) short common trunk on left side (the most common pattern), (e) long common trunk on left side (about 15%), and (f) supranumerary PVs on right side (about 20–25%).
the superior and posterior walls that make up the pulmonary venous component and the vestibule. Seemingly uniform, the walls are composed of one to three or more overlapping layers of differently aligned myocardial fibers, with marked regional variations in thickness [1] (Figure 1.2). The superior wall, or dome, is the thickest part of LA (3.5–6.5 mm), whereas the anterior wall just behind the proximal ascending aorta is usually the thinnest (1.5–4.8 mm) [2]. Also the posterior wall, especially between the superior PVs, is thin, approximately 2.5 mm in thickness. Normal LA end-systolic dimensions as measured
on cross-sectional echocardiography in the four-chamber view demonstrate
the major axis to range from 4.1 to 6.1 cm (mean 5.1 cm) and from 2.3 to
3.5 cm/m² when indexed to body surface area. The minor axis ranges from
2.8 to 4.3 cm (mean 3.5 cm) and from 1.6 to 2.4 cm/m² when indexed.

**Pulmonary veins**

The presence of myocardial muscle extensions (“sleeves”) covering the out-
side of PVs in mammals and in humans has been recognized for many years
and are regarded as part of the mechanism regulating PV flow [3] (Figure 1.2).
PVs are commonly identified as the source of rapid electrical activity trigger-
ing AF. This combines with the histological observation of P cells, transitional
cells, and Purkinje cells in the myocardial sleeves of human PVs [4]. Interest-
ingly, computerized high-density mapping demonstrated the possibility of
proximal PV foci, triggering AF in humans [5]. Over the past several years,
these anatomic and functional observations have conditioned a progressive
change of the ablation strategy for PV electrical disconnection from the struc-
tural details of the distal PV branches to the anatomy of the venoatrial junc-
tion and from a segmental to a circumferential approach. Although PVI in the
proximal venoatrial junction may be more challenging to achieve consistently
due to its increased thickness as compared to less proximal areas, this strategy
is expected not only to reduce the incidence of postablation PV stenosis but
also to increase procedural efficacy.

Anatomic studies and studies using CT and MRI have reported the presence
of significant anatomic variants of dimensions, shape, and branching of the
PVs [6–10] (Figure 1.1). Typical anatomy with four distinct PV ostia is present
in approximately 20–60% of subjects, while a very frequent anatomic variant
is the presence of a short or long common left trunk, observed in up to 75–80%
of the cases. The presence of supernumerary PVs, mainly right middle PVs or
right upper PVs with a distinct os from the RSPV, is reported in 14–25% of the
cases [11–13]. Intensive use of preprocedural 3D imaging with CT or MR scan
in multiple centers has resulted in multiple reports of rare anatomic variants of
PVs, such as the common os or trunk of the inferior PVs [14] and the posterior
accessory PV [15]. The presence of one, two, or three PV variants in the same
patient has been observed in 34%, 12%, and 2% of the cases, respectively [12].

There is general agreement that, albeit with a marked degree of interindi-
vidual variability, myocardial muscle fibers extend from the LA into all the
PVs at a length of 1–3 cm; muscular sleeve is thickest at the proximal end of
the veins (1–1.5 mm) and it then gradually tapers distally (Figure 1.2). Usu-
ally the sleeve is thickest at the inferior wall of the superior PVs and at the
superior wall of the inferior PVs, although significant variations can be ob-
served in individual cases. Frequently, muscular fibers are found circumfer-
entially around the entire LA–PV junction but the muscular architecture is
complex, with frequent segmental disconnections and abrupt changes in fiber
orientation that may act as anatomical substrates for local reentry. Recently,
an anatomical study [16] has highlighted some peculiar anatomical features of the interpulmonary isthmus, relevant for PVI by catheter ablation. This anatomic structure, which separates the ipsilateral PVs (the so-called carina), is the place where crossing fibers connecting the ipsilateral PVs are found, in a region where the myocardial PV sleeves may be in some cases as thick as 3.2 mm with intervenous muscular connections located epicardially, at a distance of 2.5 ± 0.5 mm for the left-sided PVs (Figure 1.2). In addition to the interpulmonary carinas, there is another notable “ridge”—the posterolateral ridge of the LA—that separates the orifice of the LAA from the orifices of the left PVs (see Section “Left atrial ridge and ligament of Marshall”).

**Atrial septum/fossa ovalis**

Most of all, the anatomy of the atrial septum is of interest for the electrophysiologist for a safe transseptal catheterization. It is important to understand that the atrial septum does not correspond to the entire septal wall of the RA, as visualized by fluoroscopy. Instead, it is restricted to the fossa ovalis valve and the adjacent margin of its raised muscular rim (limbus) when seen from the right atrial aspect [17] (Figure 1.3). At particular risk of procedural complication is the anterior region of the limbus fossa ovalis, which is in close anatomical relationship with the aortic mound and is seen as a protuberance into the right atrial cavity. Puncture in this area is likely to allow the needle to enter the transverse pericardial sinus resulting in a high risk of aortic perforation.

The fossa ovalis may be either circular or oval, with approximately an average vertical diameter of 19 mm and an average horizontal diameter of 10 mm.

![Figure 1.3](https://example.com/figure1.3.png)

**Figure 1.3** (a) This view of the RA displays the septal aspect en face. The limbus of the fossa ovalis surrounds a redundant and aneurismal-looking valve of the fossa (*). The blue arrows points to the slit-like PFO. (b) The atrial chambers cut in longitudinal section shows the infolding at the limbus (star) compared to the thin valve of the fossa (arrow).
Table 1.1 Congenital or acquired diseases potentially affecting the location of the fossa ovalis.

<table>
<thead>
<tr>
<th>Disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kyphoscoliosis</td>
</tr>
<tr>
<td>Straight back syndrome</td>
</tr>
<tr>
<td>Pectus excavatum</td>
</tr>
<tr>
<td>Obesity</td>
</tr>
<tr>
<td>Persistence of LS vena cava</td>
</tr>
<tr>
<td>Pericardial agenesia</td>
</tr>
<tr>
<td>Dextrocardia</td>
</tr>
<tr>
<td>Aortic or MV disease</td>
</tr>
<tr>
<td>Marked dilatation of the ascending aorta</td>
</tr>
<tr>
<td>Marked left ventricular hypertrophy</td>
</tr>
</tbody>
</table>

[17], while its area varies from 1.5 to 3.4 cm² in adults [18–20]. The thin fossa is approximately 1–3 mm thick in normal hearts and has a bilaminar arrangement of myocytes with variable amounts of fibrous tissue [21]. Therefore, this is the target area where the TSP is expected to be easier. In the general patient population, the resistance of the fossa ovalis to puncture by the transseptal needle is not clearly predicted by its thickness, assessed by preprocedure transesophageal echocardiography [22], nor by other clinical variables [23]. In patients undergoing multiple transseptal catheterization procedures, the fossa ovalis may become resistant to repeated punctures, possibly for a fibrotic reaction in the healing process after the first puncture. The location of the fossa ovalis varies from case to case. Table 1.1 lists abnormalities of the thorax or of the cardiovascular system that may result in displacement of the fossa ovalis.

Since the limbus is an infolding of the right atrial wall with epicardial fat in between (Figure 1.3), it can become quite thick especially in its superior, posterior, and inferior margins. Indeed, in some patients, the epicardial fat may increase the thickness to 1–2 cm in the normal heart. TSP through the limbus is less likely to be satisfactory since the tissue thickness can hinder needle penetration or maneuverability of the transseptal sheath after puncture. Furthermore, septal thickness of >2 cm on noninvasive imaging is increasingly reported as indicative of lipomatous hypertrophy, with incidence up to 8% on echocardiography. On cross-sectional imaging, the septum appears like a “dumb-bell” [24] encroaching upon access to the thin fossa, which is not affected by fat deposition, especially in cases with small fossa area.

Aneurysmal fossa or so-called septal aneurysm has an incidence of 0.2–1.9% in echocardiographic reports. It is detected as a saccular excursion of >1 cm of the fossa membrane away from the plane of the atrial septum. Approximately a third is associated with a PFO. Often, the fossa membrane is thinner, devoid of muscle cells and mainly composed of connective tissue. PFO existing with or without aneurismal fossa is common, occurring in 10–35% of the population. It represents a lack of adhesion of the antero-cephalad border of the membrane to the limbus (Figure 1.3). If this portal is to be used for septal crossing, it is important to note its size and distance to the antero-cephalad wall of the LA to prevent accidental exit from the heart and to ensure adequate maneuverability for reaching the target areas in the LA.
Anatomy of structures relevant to atrial fibrillation ablation

Superior vena cava

There is a great bulk of evidence that AF is triggered mainly from ectopic foci originating from the PVs and that PVI is a key step in catheter ablation of this arrhythmia [25]. However, ectopic beats initiating AF may occasionally arise from non-PV foci, such as the SVC, left atrial posterior free wall, ligament of Marshall, crista terminalis, and/or CS. On the basis of previous studies, the SVC houses the majority of non-PV foci [26–28].

Anatomically, atrial myocardium extends into the SVC (Figure 1.4) much like what occurs at the CS and around the PV [29,30] (Figure 1.2). Such myocardial extensions into both caval veins were found in the majority of human beings (76% of cases), both with and without a history of AF. Their average length in the SVC reached 13.7 ± 13.9 mm (maximum, up to 47 mm) and in the inferior vena cava, 14.6 ± 16.7 mm (maximum, up to 61 mm). The thickness of atrial myocardium extending into the CVs was 1.2 ± 1.0 mm (maximum, 4 mm) for the SVC and 1.2 ± 0.9 mm for the inferior vena cava (maximum, 3 mm).

Several groups studied incremental value of SVC isolation in addition to PVI in patients with paroxysmal or persistent AF [31–32]. The Cleveland Clinic group revealed SVC triggers in 12% of 190 patients, and isolation of the SVC prevented recurrences of AF [31]. In Mestre [32], a total of 320 consecutive patients who had been referred to for a first attempt of AF ablation

![Figure 1.4](image-url) An example of photomicrograph of a myocardial sleeve extending around the SVC. The sleeve is located next to the adventitia of the caval vein. Its fibers are mainly circular and peripherally intermingled with fat cells. Longitudinally oriented fibers (arrows) are partially fibrotic.
were randomized into two groups—PVI only and PVI with SVC isolation. SVC isolation was performed on 134 of the 160 patients (84%), and could not be accomplished in the remaining 26 patients because of PN proximity or due to the lack of local potentials. Comparison of the outcome data between the two groups, after a follow-up of 12 months, revealed a significant difference in total procedural success solely with patients manifesting paroxysmal AF (56/73 [77%] Group I vs. 55/61 [90%] Group II; \( p = .04; \) OR 2.78). On the other hand, a Chinese group [33] studied 106 cases (58 males, average age 66.0 + 8.8 years) with paroxysmal AF who were allocated randomly to two groups: PVI only \( (n = 54) \) and PVI with SVC1 \( (n = 52) \). No difference in outcome was revealed during a mean follow-up of 4 + 2 months. Therefore, the evidence that SVC isolation in addition to PVI reduces the recurrence of AF is not overwhelming.

For the above reasons, the indications for SVC isolation should be decided upon carefully because SVC isolation may cause some complications such as PN and/or sinus node injury. According to a study by Higuchi et al. [34], SVC sleeve length \( \geq 30 \) mm and maximum amplitude of SVC potential \( \geq 1.0 \) mV strongly predicted an SVC focus of AF (100% sensitivity, 94% specificity).

Technically, SVC isolation is most frequently performed using the CMC above the junction between the RA and SVC [31,35–37]. ICE proved a useful strategy to perform ablation at the level of the lower border of the RPA in order to avoid ablation close the sinus node. Pacing from ablation catheter with a high output is used to minimize PN injury. The rate of reconduction seems to be lower than after PVI [37].

**Left atrial appendage**

In a review of published papers, it was reported that in approximately 90% of patients with nonrheumatic AF thrombi were located in the LAA, making this finger-like extension of the LA of great strategic importance for stroke prophylaxis [38]. It is also a source of focal atrial tachycardia after ablation of long-lasting persistent AF [39]. Recent investigations have demonstrated extra-PV atrial foci after PVI originating from the appendage and that the junctional area between the left appendage [40] and the LA body is important in the AF process acting as a source of activity spreading to the rest of the atrium [41]. Although generally long and narrow in appearance, the external aspect of the finger shows multiple crenellations giving wide variations in number and arrangement of lobes (Figure 1.2a). Internally, the endocardial aspect is lined with muscle bundles of varied thicknesses akin to the pectinate muscles of the RA, but they are arranged in whorl-like fashion instead of in an array since there is no equivalent of a crista terminalis in the LA. In between the muscle bundles, the wall is paper-thin. The appendage communicates with the atrial chamber through an oval-shaped os. In some hearts, the atrial wall around the os can also be thin [42] (Figure 1.5). A study of postmortem and explanted hearts revealed the atrial appendage from patients with AF to
Anatomy of structures relevant to atrial fibrillation ablation

Figure 1.5 (a) This 3D rendering of a CT scan shows the endocardial surface of the LA with a pronounced “ridge” (stars) bordering the anterior margin of the left pulmonary venous orifices. (b) The left atrial ridge (stars) displayed in similar fashion as the image shown in (a). Transillumination reveals the thin areas of the walls, especially in the LAA. (c) This cut through the LSPV, the LAA, and the muscular ridge (star) shows its rounded profile and enclosing epicardial fatty tissues. (d) This cut through the LIPV shows a flatter ridge (star).

have three times the volume of those in sinus rhythm [43]. Furthermore, the endocardial surface was smoother and associated with more extensive endocardial fibroelastosis in those with AF. These features could contribute to appendage dysfunction and predisposition to thrombus formation [43].

Left atrial ridge and ligament of Marshall

A certain degree of variability is observed in the posterolateral ridge that is integral to the left PVI line (Figure 1.5). On the endocardial surface, it appears like a ridge but is actually an infolding of the left atrial wall separating the left PVs from the atrial appendage. On the epicardial aspect of the infolding runs, the remnant of the vein of Marshall covered over by fatty tissues containing abundant autonomic nerve bundles and ganglia [44] (Figure 1.5). In approximately 66% of hearts, the fold also contains a branch from the
circumflex artery that supplies the left lateral wall that, in a smaller proportion, continues to supply the sinus node [45,46].

The muscular wall of the ridge contains extensions of the leftward branches of Bachmann’s bundle. Measurements made on 32 cadaver heart specimens showed the ridge to be narrower at its superior border with the LSPV orifice compared to its inferior border with the inferior PV orifice (range 2.2–6.3 mm vs. 6.2–12.3 mm). Moreover, the endocardial aspect of this ridge may be flat, round, or pointed in profile. The first shape may be more favorable for catheter stability when positioned in this area for ablation, whereas the second and third could be very unfavorable. Overall, the ridge was <5 mm wide in 75% of hearts, suggesting that achieving catheter stability for adequate contact can be challenging in most cases [9,45].

Mitral isthmus

Linear ablation between the inferior border of the orifice of the LIPV and the mitral annulus is carried out in AF ablation to prevent recurrences. This line, dubbed the mitral isthmus, crosses the atrial vestibule, which comprises the inferior left atrial wall measuring 2–5 cm long (Figure 1.6). On its epicardial aspect runs the GCV as the vessel approaches the CS. The wall of the isthmus ranges from 2 to 8 mm in myocardial thickness. Its endocardial surface may contain pits and troughs where the atrial wall becomes exceptionally thin [47].

Interatrial conduction pathways alternative to Bachmann’s bundle

In normal hearts, the sinus impulse is quickly propagated to the anterior wall of the LA over the Bachmann’s bundle, which functionally represents the prevalent interatrial conduction pathway [48]. When a conduction delay or block occurs over the Bachmann’s bundle or when an atrial arrhythmia or a paced rhythm from a site different from the high RA are present, accessory interatrial connections may become predominant and play a major role in the right-to-left or left-to-right atrial propagation. Moreover, delayed conduction over the Bachmann’s bundle associated with interatrial conduction occurring over alternative interatrial pathways is peculiarly observed in patients with AF [49]. These accessory interatrial connections might be multiple in the same patient, show an epicardial course in direct contact with epicardial fat, and be accompanied by numerous neural fibers and ganglia [19]. They cross the interatrial groove posteriorly, at the level of the antrum of the superior and inferior PVs, or inferiorly, at the level of the inferomedial LA and CS, which exhibits myocardial sleeves surrounding its proximal tract [2,19,48] (Figure 1.6 (b1)). Finally, also fascicles located at the level of the fossa ovalis or at the level of the CS os may serve as accessory interatrial pathways [19]. Ablation in the medial region of the LA, along the septum and/or the proximal CS,
causing disruption of accessory interatrial connections and tissue debulking [50], may considerably alter the interatrial propagation during postablation organized atrial arrhythmias with unexpected modification of the surface P-wave morphology. This may occur even during typical isthmus-dependent AFL and leads to misdiagnosis of this arrhythmia [51].

**Ganglionated plexi**

The intrinsic cardiac nervous system influences cardiac rate, atrial and ventricular refractoriness, coronary blood flow, valve function, and atrial natriuretic peptide secretion, and it appears involved in many human heart...
disorders [52–54]. In particular, in paroxysmal AF although most investigations have focused on specific histological and electrophysiological properties of PVs, there are studies suggesting that some of the rapid PV firings can be induced and eliminated by stimulation and interruption of the intrinsic cardiac autonomic nervous system [55]. There is also clinical evidence that ablation of the main GP on the atria increases the success of the standard PVI by catheter ablation for AF. In other words, the active role of PVs in AF results from the high density of adrenergic and cholinergic nerves around PVs. The areas most suitable for autonomic nervous system modification procedures are located in the immediate vicinity of the PV-left atrial junction.

The topography and structure of the human epicardiac neural plexus has been carefully investigated by Pauza et al. [53,54]. It consists of a system of seven GPs that are epicardiac extensions of mediastinal nerves entering the heart through discrete sites of the so-called heart hilum. They are mostly concentrated at the fat pads and proceed separately into regions of innervation by seven pathways, on the courses of which epicardiac ganglia, as wide ganglionated fields, are located. From the arterial part of the heart hilum (i.e., around the ascending aorta and pulmonary trunk) nerves extend predominantly into the ventricles, while from the venous part of the heart hilum (i.e., around PVs and venae cavae) (Figures 1.7 and 1.8) intrinsic nerves go to both the atria and ventricles.

In general, the human RA is innervated by two subplexuses, the LA by three, the right ventricle by one, and the left ventricle by three subplexuses. The subplexuses have been named according to their topography and/or area in which subplexal post-GNs were extended: left coronary and right coronary (between the aorta and pulmonary trunk); ventral right atrial (at the superior interatrial sulcus and nonregularly on the ventral surface of the root of SVC); ventral left atrial (between the superior interatrial sulcus and left atrial nerve fold); left dorsal (at the left atrial nerve fold); middle dorsal (between the right and LSPVs and, nonregularly, between the both right PVs and inferior vena cava), and dorsal right atrial (between the SVC and RSPV). The structural organization of ganglia and nerves within subplexuses varies considerably from heart to heart and in relation to age.

The highest density of epicardiac ganglia was identified near the heart hilum, especially on the dorsal and dorsolateral surfaces of the LA, where up to 50% of all cardiac ganglia were located. In the study by Pauza et al. [54], the number of epicardiac ganglia identified for the human hearts ranged from 706 to 1560. The human heart contains on average 836 ± 76 epicardiac ganglia. The number of neurons identified for any epicardiac ganglion was significantly fewer in aged human compared with infants. By estimating the number of neurons within epicardiac ganglia and relating this to the number of ganglia in the human epicardium, it was calculated that approximately 43,000 intrinsic neurons might be present in the epicardiac neural plexus in adult hearts.
Figure 1.7 Roof of the human LA between the orifices of the PVs. (a) Panoramic view of the left atrial wall, consisting of epicardiac fat pad, myocardium, and thin endocardium. Trichrome azan stain. (b) Close-up of the boxed area in (a): at higher magnification, epicardiac ganglia are visible. (c) Normal epicardiac ganglion. Hematoxylin–eosin stain.
Figure 1.8 Histology of the left atrial wall close to the LIPV ostium. (a) A nerve is visible close to the LIPVs ostium (trichrome azan). (b) Close-up of the nerve fibers.

Phrenic nerves

The PNs and their accompanying pericardiophrenic artery and vein descend bilaterally adherent to the surface of the fibrous pericardium. The course of the right PN is in close proximity to the superior caval vein and the superior right PV. It is particularly close to the PV with a mean minimal distance of $2.1 \pm 0.4$ mm in a study made on cadavers. The same study found that the distance was $<2$ mm in a third of the cadavers, suggesting that it could be at risk of damage during right PV isolation [56]. The left PN takes an anterior (18%), lateral (59%), or posteroinferior (23%) course on the fibrous pericardium.
overlying the left heart. The lateral course passes over the tip of the LAA, while the posteroinferior course passes over the roof of the appendage os [57].

**Esophagus**

The close relationship of the esophagus to the LA exposes it to risk of inadvertent damage when ablative lesions are made in the posterior and inferior walls (Figure 1.9). It descends in a variable course, in midline or more toward the right or the left PVs and it has peristaltic movements. It is separated from the epicardial surface of the LA by the fibrous pericardium and a plane of fibrofatty tissues that contains the arterial supply to the esophagus as well as the vagus neural plexus. The distance between the endocardial surface of the LA and the esophageal wall was <5 mm in 40% of the specimens in a study performed on cadavers [58]. Taking into account the thickness of the left atrial wall, deep and large lesions can inflict injury to the esophageal arteries and vagal nerves even though the esophageal wall itself is not directly affected.

Measurements of the thickness of the posterior left atrial wall at levels corresponding to the midline in between the superior PVs, between the inferior PVs, and centrally showed the thinnest wall at the superior location [59]. Moreover, the wall at the inferior and central locations was significantly thinner in hearts from patients with AF compared to without AF (2.5 ± 1.3 mm vs. 2.9 ± 1.3 mm and 2.2 ± 1 mm vs. 2.6 ± 1 mm, respectively) [59].

Figure 1.9 This sagittal section through the LA of a cadaver shows the proximity of the esophagus to the posterior wall of the LA. Note the nonuniform thickness of the LA wall, particularly at the level of the superior PV. (Picture courtesy of Professor Damian Sanchez-Quintana, University of Extremadura, Badajoz, Spain.)
**Conclusions**

Until the substrates for AF are fully clarified, atrial ablation procedures remain anatomically orientated. For improvements in efficacy and safety of the procedures, a comprehensive understanding of cardiac anatomy and the relationship of cardiac structures to neighboring structures is the first step. This chapter has reviewed the LA with particular emphasis on the atrial septum and highlighted cardiac and adjacent structures relevant to AF ablation.

**References**

Anatomy of structures relevant to atrial fibrillation ablation


25. Calkins H, Brugada J, Packer DL, Cappato R, Chen SA, Criqns HJ, Damiano RJ Jr, Davies DW, Haines DE, Haissaguerre M, Isaka Y, Jackman W, Jais P, Kottkamp H, Kuck KH, Lindsay BD, Marchlinski FE, McCarthy PM, Mont JL, Morady F, Nademanee K, Natale A, Pappone C, Prystowsky E, Raviele A, Ruskin JN, Shemian RY. Heart Rhythm Society; European Heart Rhythm Association; European Cardiac Arrhythmia Society; American College of Cardiology; American Heart Association; Society of Thoracic Surgeons. HRS/EHRA/ECAS expert consensus statement on catheter and surgical ablation of atrial fibrillation: Recommendations for personnel, policy, procedures and follow-up. A report of the Heart Rhythm Society (HRS) Task Force on Catheter and Surgical Ablation of Atrial Fibrillation developed in partnership with the European Heart Rhythm Association (EHRA) and the European Cardiac Arrhythmia Society (ECAS); in collaboration with the American College of Cardiology (ACC), American Heart Association (AHA), and the Society of Thoracic Surgeons (STS). Endorsed and approved by the governing bodies of the American College of Cardiology, the American Heart Association, the European Cardiac Arrhythmia Society, the European Heart Rhythm Association, the Society of Thoracic Surgeons, and the Heart Rhythm Society. Europace 2007; 9: 335–379.


43. Shirani J, Alaeddin J. Structural remodelling of the left atrial appendage in patients with chronic non-valvular atrial fibrillation: implications for thrombus formation, systemic