CHAPTER 1

Anatomy of the left atrium and pulmonary veins

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Introduction

Over the past five years, the technique of catheter ablation of atrial fibrillation (AF) using a "pulmonary vein" (PV) approach has emerged from being a highly experimental procedure to the most common ablation performed in many electrophysiology laboratories throughout the world [1–4]. It is now well established that the PVs and posterior left atrium (LA) play a critical role in the initiation and maintenance of AF. It is for this reason that electrical isolation of the PVs forms the cornerstone for catheter ablation of AF. The PVs are also of great importance as PV stenosis can result from inadvertent delivery of radiofrequency (RF) energy within a PV [5–7]. Increasing evidence suggests that the risk of PV stenosis may be minimized and the success maximized by delivery of RF energy to the ostial portion of the PV [5–10]. Understanding PV anatomy is also important in the development of balloon-based ablation technologies [10–13].

Embryologic considerations

The location of the precursors of the conduction system is defined, during embryological development of the heart, by the looping process of the heart tube [14]. Specialized conduction tissue that is derived from the heart tube and is destined to have pacemaker activity has been shown to be located within the myocardial sleeves of the PVs [15,16]. Perez-Lugones demonstrated the presence of P cells, transitional cells, and Purkinje cells in the human PVs. The presence of these tissues provides an explanation for the observation that electrical activity within the PVs is commonly observed after electrical disconnection of the PVs musculature from the atrium [17]. These observations also help provide an understanding for why the PVs are commonly identified as the location of rapidly electrical activity which triggers the development of AF [1–3].

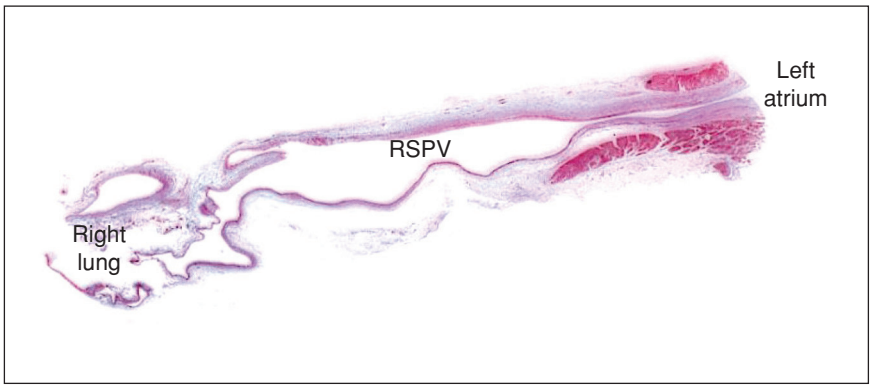

Figure 1.1 This histologic section through the length of the right superior pulmonary vein (RSPV) shows the myocardium from the left atrium continuing along the outer aspect of the venous wall to form the myocardial sleeve.

PV muscular sleeves: pathologic and histologic characteristics

The presence of myocardial muscle extensions ("sleeves") covering the outside of PVs in mammals and in humans has been recognized for many years and regarded as part of the mechanism regulating PV flow [18]. There is general agreement that, albeit with a marked degree of interindividual 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 1.1). Usually 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 observed in individual cases.

Over the past several years a progressive change of the ablation protocols for AF has shifted interest from the structural details of the PVs to the analysis of the arrangement of the muscle fibers of the venoatrial junction [19]. Frequently, muscular fibers are found circumferentially 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. Consequently, the elimination of these areas of atrial myocardium with catheter ablation may play an important role in the effectiveness of AF ablation procedures.

In addition, there are abundant adrenergic and cholinergic nerves in the ganglionated plexi in the vicinity [20]. Preferential location of these structures includes the left superior PV at the junction with the atrial roof, the posteroinferior junction of the inferior PVs, and the anterior border of the right superior PV.

Electrophysiologic characteristics of the PV muscular sleeves

A recent study using optical mapping techniques examined the electrophysiologic properties of 45 PVs from 33 dogs and found action potential duration to be longer in the endocardium of these PVs as compared with the epicardium [21]. There was marked slowing of conduction in the proximal portion of the PV as compared with the adjacent LA. With rapid atrial pacing, 2:1 conduction block into the veins was observed. These findings led the authors to propose that AF resulted from a focal trigger arising from within the PVs and was maintained as a rapid reentrant circuit within the PV. A somewhat different approach was used by Hocini et al., who examined the electrophysiologic characteristics of the PVs blood perfused heart preparation and obtained intracellular and extracellular recordings [22]. These authors identified zones of conduction delay in all PVs. Fractionated signals were also found in areas of slow conduction. Zones of slow conduction were correlated to sudden changes in fiber orientation observed on histology. These changes could facilitate reentry. In another interesting study, Kalifa et al. examined the impact of increasing atrial pressure on the PV activation [23]. They reported that as left atrial pressure was increased above 10 cm H_2O, the LA–PV junction became the source of dominant rotors. These observations help to explain the clinical link between AF and increased atrial pressure.

Normal size and anatomy of the LA

The LA has a venous component that receives the PVs, a fingerlike atrial appendage, and shares the septum with the right atrium. The major part of the atrium, including the septal component, is relatively smooth-walled whereas the appendage is rough with pectinate muscles (Figure 1.2). The smoothest parts are 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 [24]. The superior wall, or dome, is the thickest part of LA (3.5–6.5 mm), whereas the anterior wall just behind the aorta is usually the thinnest (1.5–4.8 mm) [25]. Also the posterior wall, especially between the superior PVs, is thin (Figure 1.3) and ablating this area may increase the risk of atrioesophageal fistula. 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/m^2 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/m^2 when indexed.

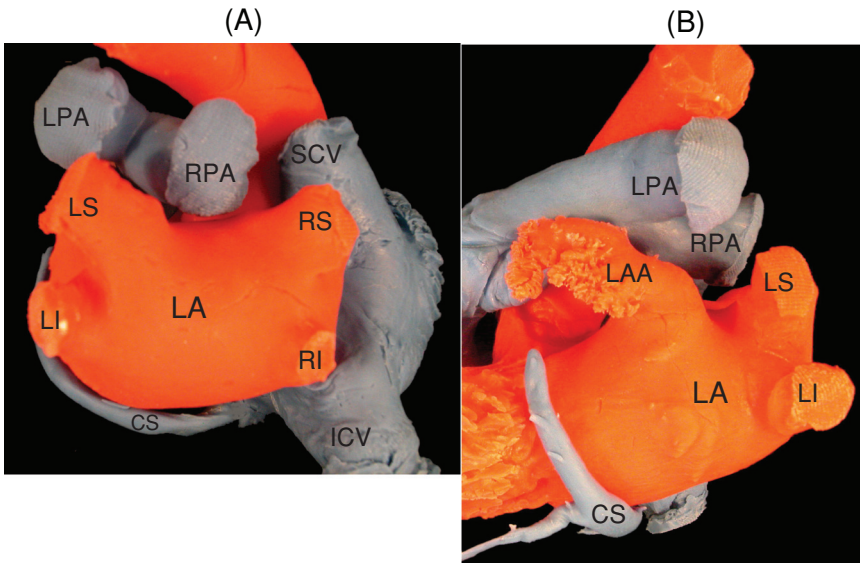

Figure 1.2 (A) The endocast viewed from the posterior aspect shows the proximity of the right pulmonary veins (RS and RI) to the atrial septum. Note also the right pulmonary artery (RPA) immediately above the roof of the left atrium (LA). (B) The endocast viewed from the left shows the rough-walled left atrial appendage (LAA) and its relationship to the left superior (LS) pulmonary vein. The coronary sinus (CS) passes inferior to the inferior wall of the left atrium. ICV, inferior caval vein; LI, left inferior pulmonary vein; LPA, left pulmonary artery; SCV, superior caval vein.

Normal size and anatomy of the PVs

The transition between LA and PVs is smooth without pronounced ridges. The venoatrial junction is least distinct when the entrance of the vein is funnel-like. Anatomic studies and also studies using magnetic resonance (MR) and computed tomography (CT) imaging have reported significant variability in dimensions, shape, and branching patterns of the PVs [26–30].

Typical PV anatomy with four distinct PV ostia is present in approximately 20–60% of subjects (Figure 1.4) [26,30]. A frequent anatomical variation is the presence of a short or long common left trunk observed in up to 75–80% of patients with MR or CT imaging (Figures 1.4–1.6). Additional abnormalities of 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) (Figure 1.4).

The orifices of the left PVs are located more superiorly than that of right PVs. The right and the left superior PVs project forward and upward, while the right and left inferior PVs project backward and downward. The right superior PV lies just behind the superior vena cava or right atrium and the left PVs are positioned between the LA appendage and descending aorta. The orifice of

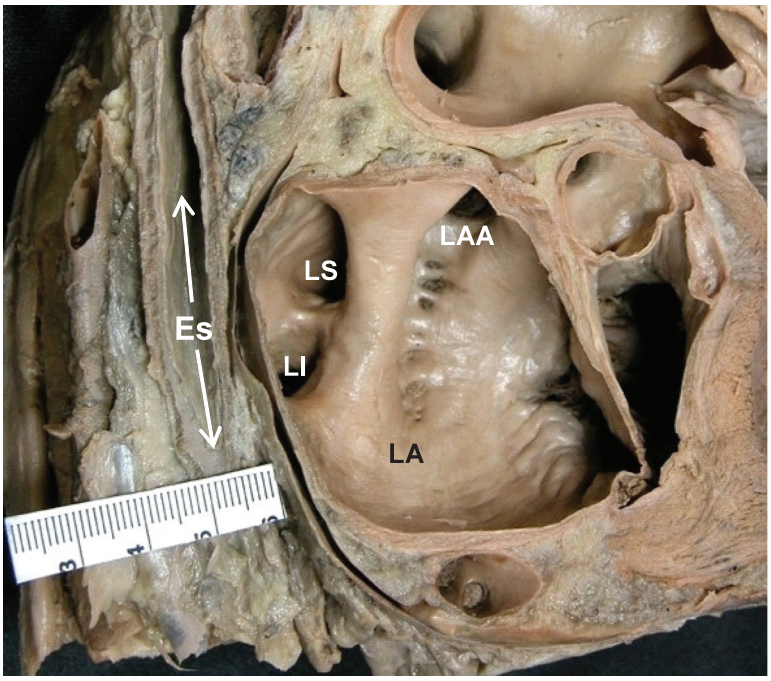

Figure 1.3 This sagittal section through the left atrium of a cadaver shows the proximity of the esophagus (Es) to the posterior wall of the left atrium (LA). The wall is particularly thin at the level of the superior pulmonary veins. Note the "ridge" between the left superior pulmonary vein (LS) and the left atrial appendage (LAA). LI, left inferior pulmonary vein. (Courtesy of Professor Damian Sanchez-Quintana, University of Extremadura, Badajoz, Spain.)

the LA appendage lies in close proximity to the ostia of the left superior and left inferior PV (Figure 1.2) and is separated by them by a fold in the atrial wall that appears like a ridge in the endocardial surface [26] (Figure 1.4). The width of this ridge is 3.8 ± 1.1 and 5.8 ± 2.0 mm at its narrowest point at the level of left superior PV and left inferior PV, respectively. The ridge is narrower than 5 mm in the majority of patients, thus determining the inability to obtain stable catheter position in this region [29].

In heart specimens, the distance between the orifices of the right PVs ranges from 3 to 14 mm (mean 7.3 ± 2.7 mm) and that between the orifices of the left PVs from 2 to 16 mm (mean 7.5 ± 2.8 mm). Anatomic studies and clinical series have shown that the PV orifices are oblong rather than round in shape with a funnel-shaped morphology at the venoatrial junction (Figure 1.5) in both AF patients and in controls with the anterior–posterior diameter less than superior–inferior diameter. Thus, identifying anatomic limits of the PV ostium is not usually easy. Discounting the common vestibule, the anatomic diameter of the PV orifices at the venoatrial junction ranges from 8 to 21 mm (12.5 ± 3 mm).

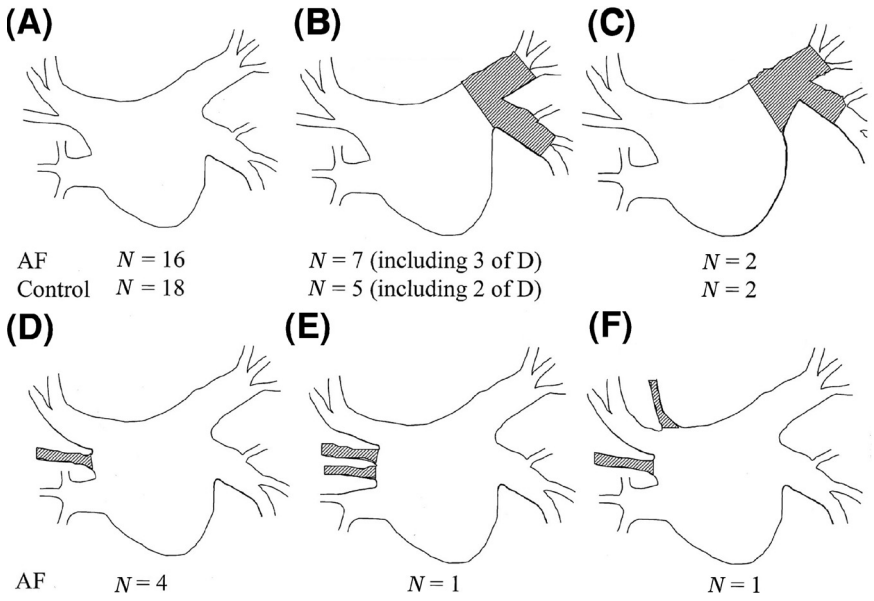

Figure 1.4 Branching pattern of pulmonary vein (PV) anatomy in atrial fibrillation (AF) patients and controls (from Kato et al., 2003). Shaded portions indicate different parts from typical anatomy: (A) typical branching pattern; (B) short common left trunk; (C) long common left trunk; (D) right middle PV; (E) two right middle PVs; (F) right middle PV and right "upper" PV.

Impact of AF on LA and PVs size

During the past decade, extensive animal-based and clinical research has demonstrated that AF results in electrical and structural remodeling of the atrium [31–36]. The results of these studies, taken as a whole, suggest that AF can be viewed as a rate-related atrial cardiomyopathy. It is now well recognized that AF, if persistent enough, generates molecular, cellular, and architectural

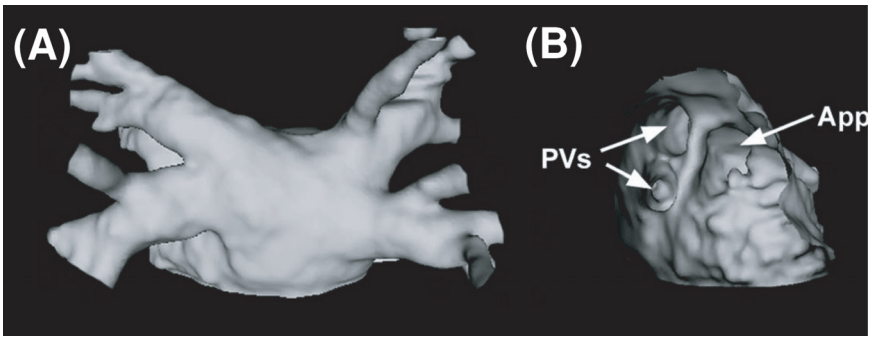

Figure 1.5 Magnetic resonance angiography of the left atrium and pulmonary veins depicted: (A) in posteroanterior view; (B) as virtual endoscopic image of the left-sided pulmonary veins (PVs) and appendage (App). Note a common antrum of the left-sided PVs and its oval appearance.

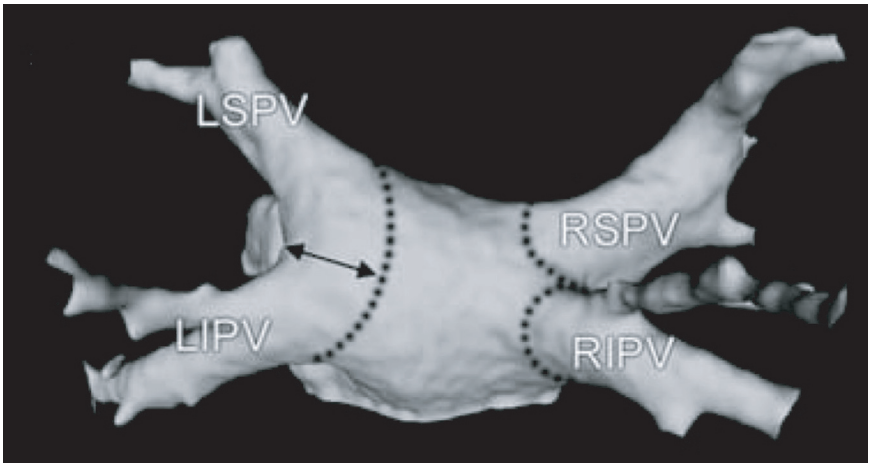

Figure 1.6 Typically 3-D reconstruction of left atrium and pulmonary veins (PVs) obtained from magnetic resonance angiography. Dotted lines mark boundaries of ostia of the PVs. 3-D magnetic resonance and computed tomography imaging have shown the presence of a left common ostium in up to 75–80% of patients. This finding has been confirmed with intracardiac echocardiography. LIPV, left inferior pulmonary vein; RIPV, right inferior pulmonary vein; LSPV, left superior pulmonary vein; RSPV, right superior pulmonary vein. (Reproduced from Reference [30], with permission.)

alterations in the atrial myocardium, resulting in electrophysiological, then structural, changes. Other modifiers that impact atrial size include the presence or absence of pressure overload resulting from systolic or diastolic left ventricular dysfunction or valvular regurgitation.

In a study, Ho et al. compared the dimensions of the LA among patients undergoing AF ablation and controls [25]. The longitudinal, anterior–posterior, and transverse diameter of the LA was 64.2 ± 7.8, 33.1 ± 6.3, and 56.1 ± 6.3 mm, respectively, in AF patients and 54.9 ± 5.5, 28.1 ± 3.5, and 46.5 ± 5.6 mm, respectively, in controls ($p < 0.001$).

In another study, Kato et al. compared the size of PV orifices and found no difference in AF patients whereas the left inferior PV was smaller than the right inferior PV in controls [26].

Overall the PV size was larger in AF patients as compared with controls ($p < 0.05$).

Summary and conclusion

In conclusion, it is clear that those who are involved in the field of catheter ablation of AF, and particularly those performing the procedures, should have a clear understanding of the anatomy of the LA and PVs. Not only is it important to have an understanding of the location and orientation of the PVs, but it is essential to recognize that the anatomy of the PV varies considerably between patients. Of even greater importance is the awareness of variant PV anatomy. The presence of an anomalous PV arising from the roof of the atrium would

likely impact the lesion set delivered for ablation. Because of these issues, we recommend that MR or CT imaging be performed prior to AF ablation procedures.

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