

STATE-OF-THE-ART REVIEW

Left Atrial Appendage

Embryology, Anatomy, Physiology, Arrhythmia and Therapeutic Intervention



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ABSTRACT

Known for the pathological connection to atrial fibrillation (AF), the left atrial appendage (LAA) is the most common source of thromboembolism in patients with AF and may be an arrhythmogenic source for the maintenance of AF. Potential interventions of the LAA for stroke prevention have recently been developed through better understanding its anatomy and physiology. Occlusion of the LAA is an alternative to the use of life-long anticoagulation in selected nonvalvular AF cases. The PROTECT-AF (The WATCHMAN LAA Closure Device for Embolic PROTECTION in Patients with Atrial Fibrillation) and PREVAIL (Randomized Trial of LAA Closure vs. Warfarin for Stroke/Thromboembolic Prevention in Patients with Non-valvular Atrial Fibrillation) randomized controlled trials demonstrated that LAA exclusion using the Watchman percutaneous device is not inferior to warfarin. However, the appendage is structurally complex and has considerable morphological variations among individuals, and it can be challenging to generalize the device for all patients. Continued technological developments including occlusion/ligation through epicardial, endocardial, or surgical approaches, as well as operator expertise regarding LAA anatomy, physiology, and pathophysiology, should improve interventional outcomes. Furthermore, the optimal strategy for re-entrant tachyarrhythmias arising from LAA remains unknown. Whereas an observational study suggested that LAA isolation was more effective than focal ablation, LAA isolation may be associated with significant impairments in LAA contractility, predisposing individuals to a risk of thrombosis. (J Am Coll Cardiol EP 2016;2:403-12) © 2016 by the American College of Cardiology Foundation.

The left atrial appendage (LAA) is a derivative of the atrial primordium that has anatomical and physiologic variations from the left atrium (LA), which is an extension of the embryological pulmonary vein (PV) bud. As the major source of thromboemboli in patients with atrial fibrillation (AF), our current understanding of the LAA anatomy and function has extended to the development of LAA closure devices for stroke prevention. This review focuses on the background of the LAA anatomy and physiology and provides an overview of closure techniques, in particular, the Watchman device (MedStar,

Washington, District of Columbia), which has been recently approved by the U.S. Food and Drug Administration (FDA) for clinical use. Excellent review for currently available LAA closure devices and in development has been provided elsewhere (1,2).

EMBRYOGENESIS

The LAA is the only cardiac structure in the LA derived from the primitive atrium; the rest are a part of PVs and are characterized by a smooth endocardium (Online Video 1, Online Figure 1) (3). At week 4 of gestation,

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**ABBREVIATIONS
AND ACRONYMS**

- AF** = atrial fibrillation
- CT** = computed tomography
- LAA** = left atrial appendage
- LA** = left atrium
- LOM** = ligament of Marshall
- LSPV** = left superior pulmonary vein
- LV** = left ventricle
- PA** = pulmonary artery
- PV** = pulmonary vein
- TEE** = transesophageal echocardiography

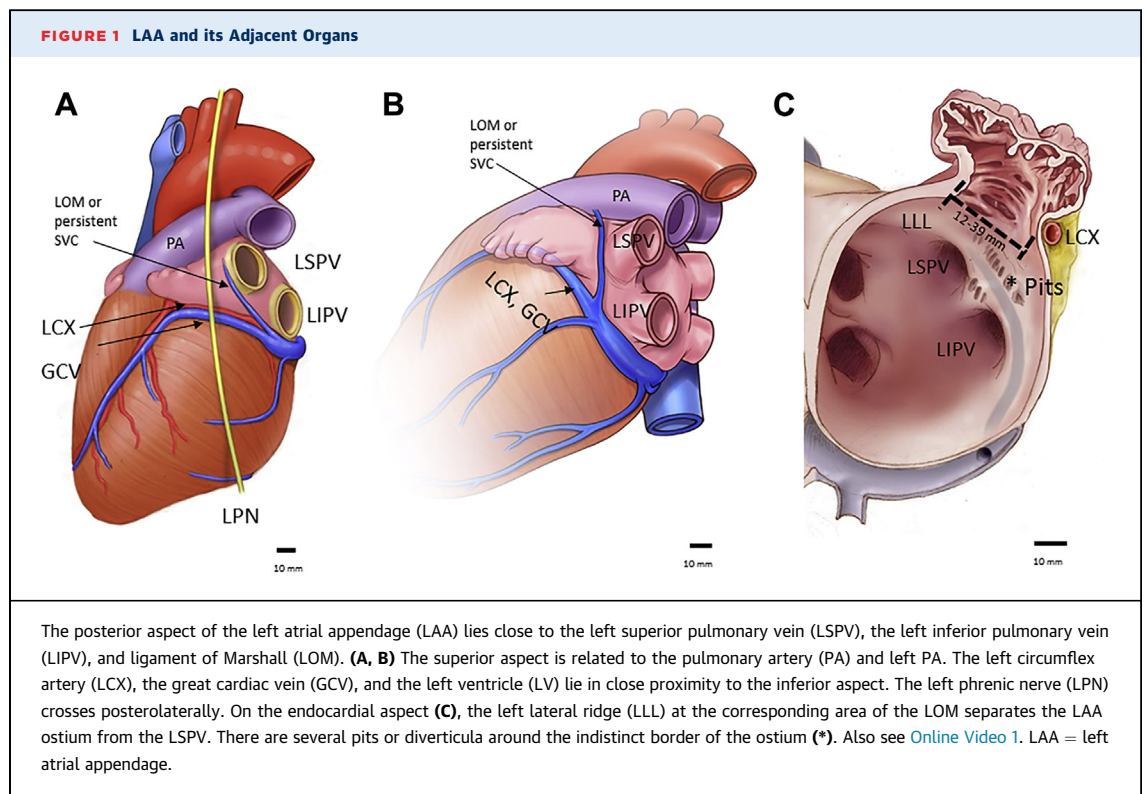
the primitive atrium undergoes a right-handed looping toward its ultimate location. The subsequent cellular protrusion phase solidifies the basal mesodermal layer and forms the trabeculae/pectinate muscles that lead to the rough endocardium characteristic of the LAA (3,4). When the lungs develop, an outgrowth of the primordial PV buds connects to the primitive atrium and completes the development of LA at approximately day 50 of the embryologic life (3).

DEVELOPMENTAL ANOMALIES. Very few developmental abnormalities of the LAA have been reported. Isomerism of the LAA is associated with fatal congenital syndromes (5). It is important to distinguish the congenital absence of the LAA from the LAA membrane, found incidentally, from complete or flush thrombotic occlusion (6). Congenital LAA aneurysms are associated with pectinate muscle dysplasia, predisposing to thrombus formation, supraventricular arrhythmias, and rupture. Therefore, aneurysmectomy is generally recommended (7). The congenital absence of the pericardium may be associated with appendage herniation that could result in difficulties in a pericardial intervention of the LAA (2).

ANATOMY

Fixed in the front part of the pericardial space, the LAA is within close proximity to several vital organs. Projecting to a various curve, the body of the LAA is anterior to the LA and parallel to the left PVs (8). Its tip points to the pulmonary artery (PA), right ventricular outflow tract, and left ventricular (LV) free wall (Figures 1 and 2) (9). The PA and left PA then course superoposteriorly and are separated by the transverse sinus. The inferior aspect and ostium are closely related to the left circumflex artery and the great cardiac vein that course along the atrioventricular groove and the mitral valve. The left phrenic nerve also courses posterolaterally. An indentation of the ligament of Marshall (LOM), or a remnant of left superior vena cava, serves as an epicardial landmark between the left lateral aspect and left superior PV (LSPV). Slightly lateral to the ligament, there is Bachmann’s bundle encircling the LAA neck (2).

Albeit heavily trabeculated endocardium, the LAA wall is remarkably thin (~1 mm) (2). Its ostium has an incomplete boundary and a prominent ridge from the LOM (also known as the left lateral ridge, or “Q-tip” sign on echocardiography), posterosuperiorly separating LAA and LSPV. The anterior and inferior aspects are indistinct from the LA.



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