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Review article

A highly-detailed anatomical study of left atrial auricle as revealed by in-vivo computed tomography

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ABSTRACT

The left atrial auricle (LAA) is the main source of intracardiac thrombi, which contribute significantly to the total number of stroke cases. It is also considered a major site of origin for atrial fibrillation in patients undergoing ablation procedures. The LAA is known to have a high degree of morphological variability, with shape and structure identified as important contributors to thrombus formation. A detailed understanding of LAA form, dimension, and function is crucial for radiologists, cardiologists, and cardiac surgeons.

This review describes the normal anatomy of the LAA as visualized through multiple imaging techniques such as computed tomography (CT), magnetic resonance imaging (MRI), and echocardiography. Special emphasis is devoted to a discussion on how the morphological characteristics of the LAA are closely related to the likelihood of developing LAA thrombi, including insights into LAA embryology.

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Abbreviations and acronyms

LAA	left atrial auricle
RAA	right atrial auricle
CT	Computed Tomography
AF	atrial fibrillation
CCTA	Cardiac Computed Tomography Angiography
ANP	atrial natriuretic peptide
TEE	transesophageal echocardiography
TTE	transthoracic echocardiography
LAAO	Left atrial auricle occlusion

1. Introduction

{Citation}The clinical significance of atrial auricleshas been historically underappreciated, with their role being primarily associated with arrhythmias and the risk of thromboembolism. However, recent research has highlighted the LAA as an important contractile reservoir in volume overload, as well as a key site for the synthesis and secretion of atrial natriuretic factors. This reevaluation of the LAA highlights the need for further investigation into atrial auricles anatomical and physiological characteristics. Cardiac Computed Tomography Angiography (CCTA) provides a highly accurate non-invasive morphological assessment of atrial auricles, and can accurately describe its remarkable morphological heterogeneity [[1–3]]. Dual Energy Computed Tomographic (DECT) imaging has evolved like an imaging technique for improve the image contrast of vascular structures and for reduce contrast material and even beam-hardening artifacts thanks to its unique post-processing opportunities [[4,5]]. A comprehensive knowledge of the morphology of the auricles, and in particular of the LAA, is very important to identify potential structural factors that could complicate procedures inserting exclusion devices in cases of high risk for as well as limitations in the use of anticoagulants. Such knowledge can help reducing the risk of iatrogenic damage and misplacement of exclusion devices during invasive procedures. Detailed analysis of LAA based on high-resolution imaging studies is increasingly given the recent development of percutaneous auricle closure devices for the stroke prevention. In addition to CT, also MRI shown an important role for a precise characterization of LAA (geometry, size, orifice diameter, volume and function) [[6,7]] and for the identification of LAA thrombus using early gadolinium enhancement sequences characterized by the highest diagnostic accuracy [[8]].

In this paper we present an accurate morphological and morpho-functional description and evaluation of the LAA, including details on shape, morphological classifications, LAA ostium structure and conformation, isthmus morphology and spatial relationships, emphasizing how such characteristics correlate with the probability of developing LAA thrombi.

We have also reviewed the occurrence of embryological vestiges that can potentially influence LAA occlusion procedures or its hemodynamic and endocrine functions. Finally, we have also shortly reviewed some aspects of the morphology of the often neglected right atrial auricle (RAA).

2. The left atrial auricle

2.1. Morphology

The LAA is the most common location for thrombus formation in atrial fibrillation (AF) and atrial disease [[9–12]]. It is also considered a trigger for AF in 27% of patients referred to cardiac ablation procedures [[9]]. Most of the prothrombotic susceptibility of the LAA relies on its morphology [[13–17]]. A deep knowledge of LAA anatomy is essential for cardiologists, radiologists, and cardiac surgeons to adequately differentiate the presence of thrombi from anatomical structures such as hypertrophied pectinated muscles, to insert occlusion devices into anatomically appropriate sites and to avoid iatrogenic injury during invasive procedures [[10]]. The LAA is a finger-shaped extension protruding from the lateral or inferolateral wall of the left atrium [[13–17]] (Fig. 1). It represents the embryonal remnant of the left atrium [[14,15]]. The LAA is much smaller than the contralateral RAA, with a variable length (16–51 mm) that decreases with advancing age [[9]]. In 80% of cases, the LAA has a multilobular shape [[9]].

2.2. Morphological classification

The LAA body features several lobes and folds [[18]]. The most frequent LAA shape is with 2 lobes (54%), followed by 3 lobes (23%), 1 lobe (20%), and 4 lobes (3%) [[19]]. The number of lobes has been positively correlated with the risk of auricle thrombosis [[20]]. The highest number of lobes is near the left atrio-ventricular sulcus and the left ventricular surface [[19]]. The LAA has been morphologically classified into four types:

1. “chicken wing”, exhibiting a prominent proximal or central fold of the main lobe (Fig. 2A–D);
2. “cactus”, characterized by a dominant central lobe, with secondary lobes branching off superiorly and inferiorly (Fig. 3A–D);
3. “windsock”, featuring a dominant lobe filling the entire extension of the auricle (Fig. 4A–C);

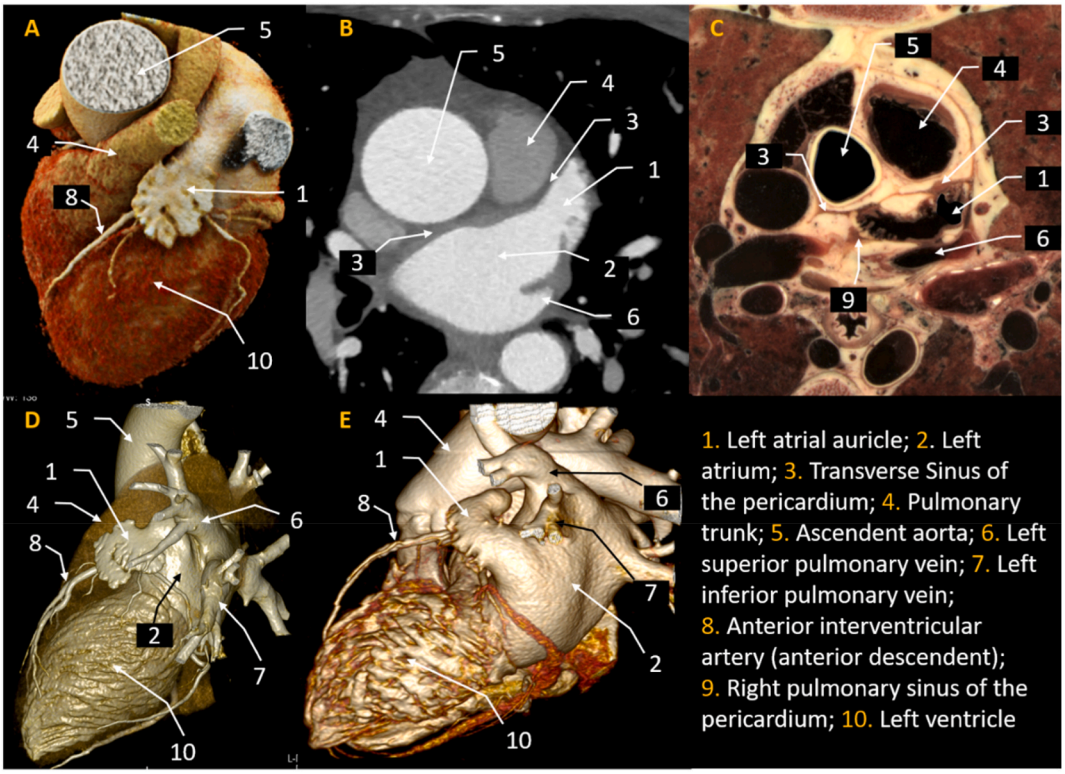

Fig. 1. Cinematic rendering (A), Axial reconstruction (B) and Volume Rendering cardiac-CT images (D–E), highlighting LAA main features and its anatomical landmarks. Thoracic axial section through LAA obtained from Visible Human Server (C; courtesy of Prof. R.D. Hersch. Ecole Polytechnique Federale de Lousanne (EPFL). Switzerland. Visible Human Web Server. http://visiblehuman.epfl.ch).

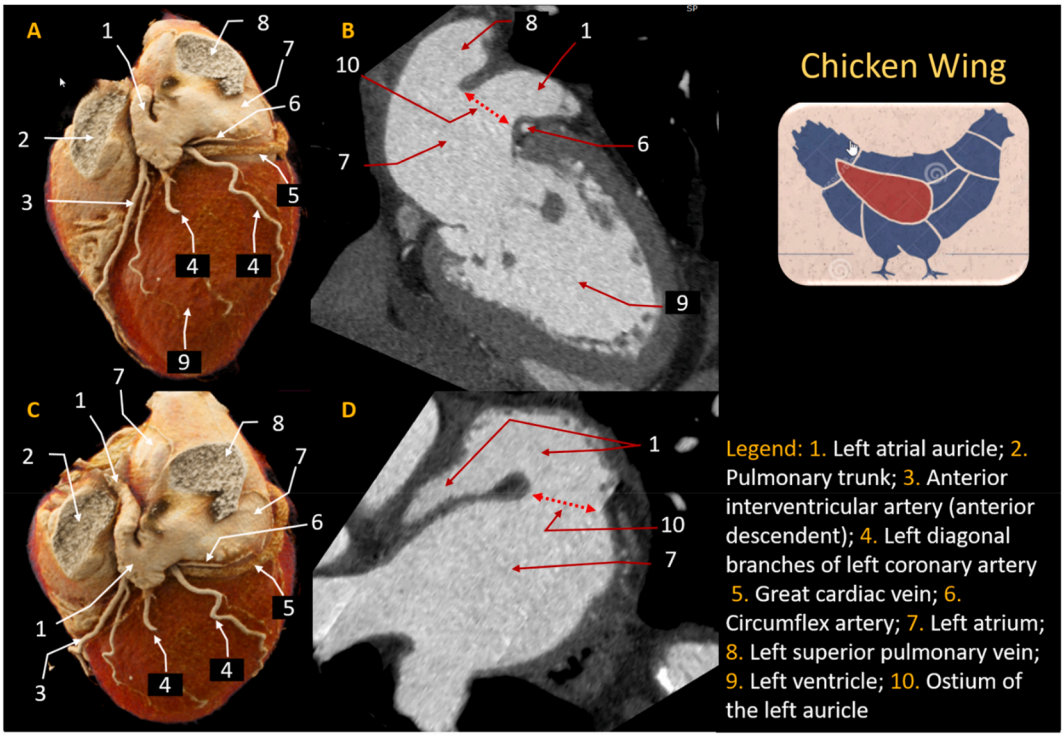

Fig. 2. (A–D). Cinematic rendering (A,C) and multiplanar (B,D) CT reconstruction images of "chicken wing" LAA morphology.

4. "cauliflower", characterized by a small length, complex internal structures, and multiple tiny lobes [[21,22]] (Fig. 5A–C).

According to such imaging criteria, the most frequent type is "chicken wing" (48%), followed by "cactus" (30%), "windsock" (19%), and "cauliflower" (3%) [[22,23]], while in the autoptic population, "cauliflower" is the most common morphology (41%), followed by "chicken wing" (37%), "cactus" (12%) and "windsock" (10%) [[9]]. These discrepancies can be explained by the difficulty of identifying the exact morphology of the LAA according to objective criteria and/or by the alterations that the atrial and auricular myocardium undergoes in the state of *rigor mortis*.

Morphology, shape, volume, length and width of the LAA clearly correlate with the probability of developing LAA thrombi [[13,15,20,24,25]]. LAAs with "chicken wing" morphology have a lower risk of thrombosis than other types, and among these others, "cauliflower" is the most hazardous [[15,26]].

2.3. Morpho-functional correlations

The endocardial surface of the LAA is characterized by several thin-branched pectinated muscles, with a thickness greater than 1 mm, which make the auricle very contractile [[26–30]] (Fig. 6A–I). Sometimes, one or more of these muscles may be so evident that they may resemble an intracavitary thrombus or mass [[13]]. There is a correlation between the kind of the muscular trabeculae and the macroscopic morphology of the LAA: a single thin trabecula is present in "chicken wing" LAA, more evident trabeculae are present in "cactus" LAA (Fig. 3C), and extended and prominent trabeculae in "cauliflower" shape. Muscular trabeculae are more commonly located in the mitral isthmus (34.5%) rather than in the LAA isthmus (located between the LAA ostium and the mitral annulus – see below) (4.5%) [[31]]. A high grade of anastomoses among branched pectinated muscles is a robust and independent thromboembolic risk factor, along with a small LAA orifice and a short LAA [[31]]. The LAA may present diverticula and small recesses of 0.5–10.3 mm, variably distanced from the LAA ostium (1.4–21 mm) [[32]]. Different LAA morphologies, and above all, the "chicken wing" morphology, may influence and complicate LAA exclusion procedures [[33]]. Congenital absence of the LAA is very rare, and only a few cases have been reported so far [[10]] (Fig. 7A–D). In the autoptic study by Ernst et al., the LAA volume ranged from 770 mm^3 to 20.000 mm^3 [[24]]. The LAA volume positively correlates with the thromboembolic risk [[24]].

2.4. The LAA ostium

Due to its tubular shape, the LAA creates a narrow ostium with the left atrium, which contains an internal smooth prominent ridge along the superior and posterior margin of the left atrium [29]. The left atrial ridge is a structure located in the left atrium between the LAA ostium and the left-sided ostia of the pulmonary veins [[29]]. It is also known as the left "warfarin" or "coumadin" ridge, since it is commonly misdiagnosed as a thrombus. The LAA ostium is morphologically variable, but is most commonly elliptic, with its long axis oblique to the mitral annulus [[32]]. Other morphologies include the oval, rectangular, triangular, and teardrop shapes [[34]]. The

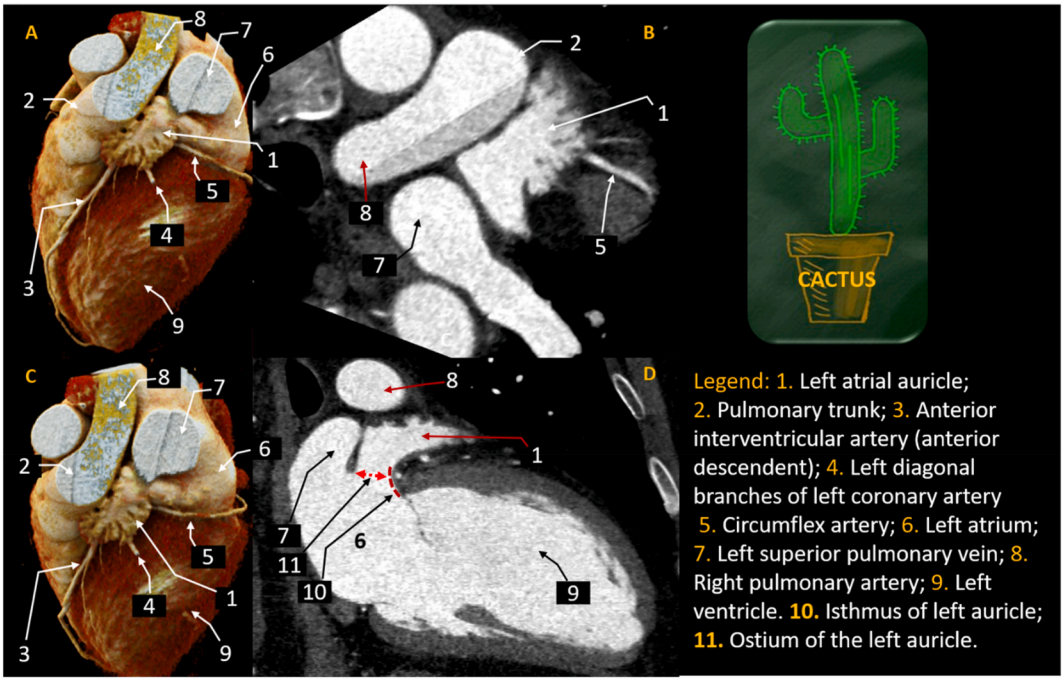

Fig. 3. (A–D). Cinematic rendering (A,C) and multiplanar (B,D) CT reconstruction images of "cactus" LAA morphology.

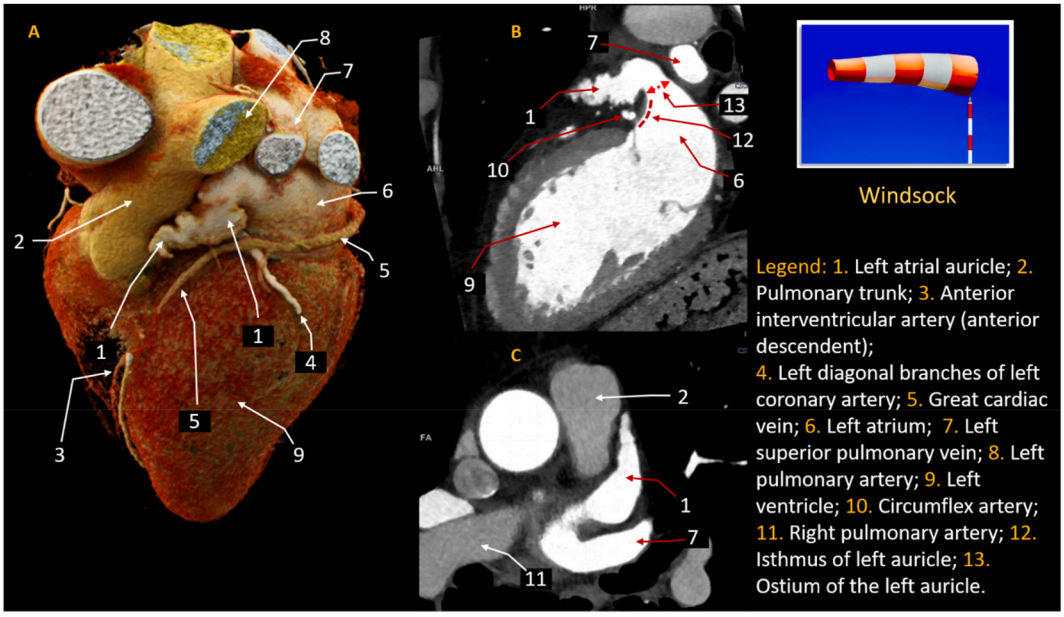

Fig. 4. (A–C). Cinematic rendering (A) and multiplanar (B,C) CT reconstruction images of "windsock" LAA morphology.

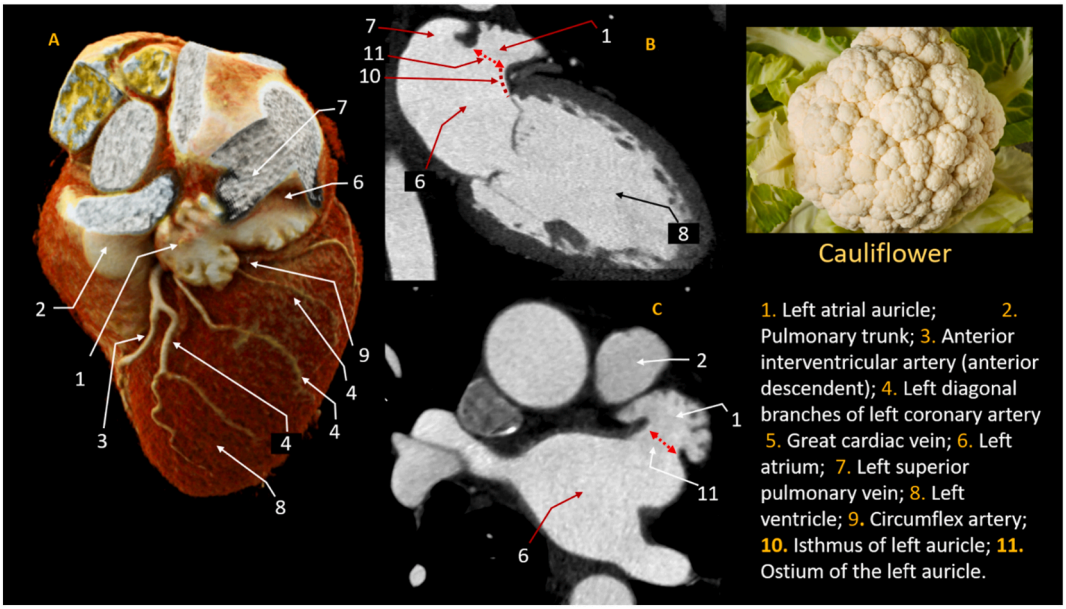

Fig. 5. (A–C). Cinematic rendering (A) and multiplanar (B,C) CT reconstruction images of "cauliflower" LAA morphology.

average greatest diameter is 17 ± 4 mm in case of oval LAA ostium [35][[32]]. Average LAA ostium dimensions slightly range during the cardiac cycle in normal sinus rhythm (±1–2 mm), whereas complete absence of changes has been observed in case of atrial fibrillation.

2.5. The LAA isthmus

The LAA isthmus is located between the LAA ostium and the mitral annulus, and has been recently reported as a new potential ablation line in AF ablation procedures. Holda et al. have hypothesized that in some cases the LAA isthmus may replace the mitral isthmus in ablation procedures or that the two isthmi may be jointly ablated to garner better results. The LAA isthmus is significantly shorter than mitral isthmus (average length difference: 15 ± 8 mm; range 0.1–54 mm). Further, the LAA isthmus line is smooth in >95% of cases, while pouches and diverticula are present in less than 5%. It is also quite close to the circumflex coronary artery, with an average distance of 11.3 ± 5.2 mm [[35]].

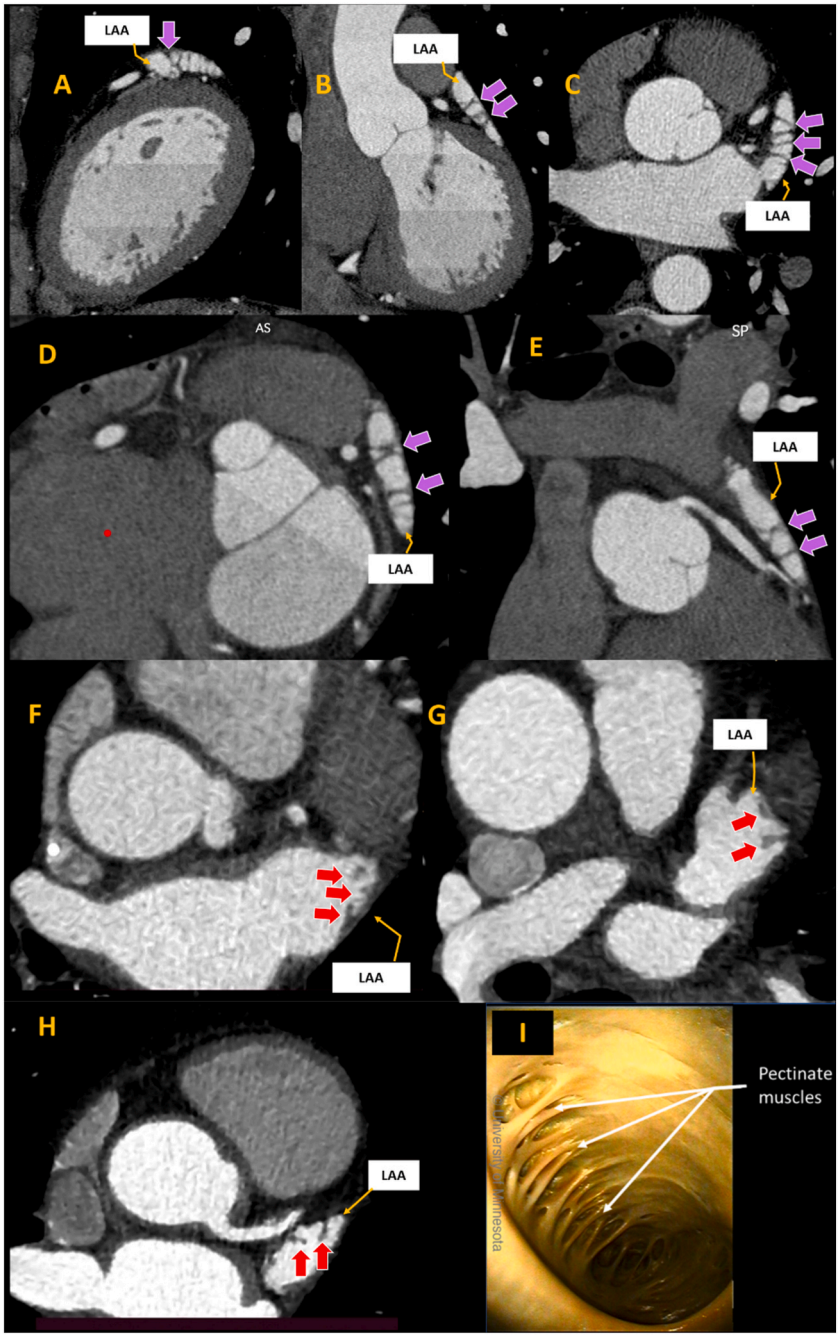

Fig. 6. Muscular trabeculations in LAA. Trabeculations are indicated by violet arrows in multiplanar CT reconstruction A-E. Pectinate muscles in LAA are indicated by red arrows in multiplanar CT reconstruction F–H. Fig. 6I is an *ex-vivo* endoscopic image wich presents pectinate muscles of LAA. Fig. 6I is a courtesy of Prof. Iaizzo, Visible Heart Laboratory Atlas of Human Cardiac Anatomy. Minnesota University,http://www.vhlab.umn.edu/atlas. (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.)

2.6. *LAA relationships (Figs. 1–5)*

The LAA closest anatomical relationship is with the pericardial sac. The visceral pericardial leaflet covers the entire auricle and is the LAA epicardium. Between the LAA visceral and parietal pericardium, a pericardial space, called left pulmonary sinus, is found, covering the LAA mainly above its superior ridge. Such pericardial sinus is in continuity with the right pericardial sinus located beneath the right pulmonary artery, the inferior aortic sinus, and the transverse pericardial sinus. The LAA is generally directed anterolaterally and runs alongside the left convex side of the pulmonary trunk (Fig. 1A), just above the pulmonary valve. In some cases,

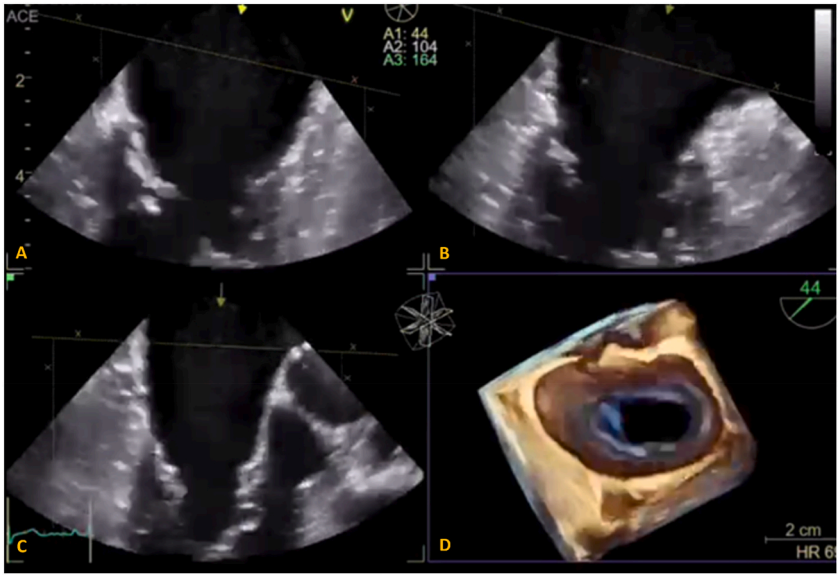

Fig. 7. Multiple transesophageal ultrasound left atrium B-Mode (A,B,C) and 3D (D) views obtained at mid-esophageal level show a rare case of congenital absence of the LAA.

the LAA is related even with the posterior wall of the aortopulmonary pedicle. The anterior wall of the LAA contributes to forming the rear wall of the transverse pericardial sinus (Fig. 1A), along with the anterior wall of the left atrium. The LAA body often extends above the left atrioventricular sulcus and hides the course of the great cardiac vein (Fig. 4A), the first tract of the circumflex artery (Fig. 2A–D), and the left main coronary artery. Knowledge of these arterial and venous anatomical relationships is essential in endocardial or epicardial LAA exclusion procedures.

2.7. Cardiac Computed Tomography Angiography protocol

All individuals underwent ECG-triggered CCTA performed by using a 128-slice scanner (Somatom Definition AS+, Siemens Healthineers, Forchheim, Germany). Each patient lied in supine position, feet-first, with raised arms on the scanner table. All scans were acquired in a cranio-caudal direction with a z-axis coverage from the tracheal carina to the apex of the heart with retrospective ECG-triggering. All patients received oral β-blocker prior the use of the contrast medium (Iomeprol 400 mgI/ml, 80 cc, 5.0 ml/s): intravenous (IV) contrast medium was injected with a mechanical injector into an antecubital vein via wide gauge IV cannula (18 G) in order to obtain an iodine delivery rate (IDR-gI/s) of 2.0 gI/s. A pre-monitoring phase was achieved, by using the bolus tracking technique; a region of interest (ROI) was positioned in the ascending aorta with a preferred Hounsfield Unit (HU) threshold. When the chosen threshold was reached, the acquisition started automatically. Scanning parameters were as follows: gantry rotation time 0.33 s and matrix size 512 × 512 pixels. All images were reconstructed at 60–80% of the R–R interval with a 0.6 mm slice thickness, both with filtered back projection (FBP) kernel (B46) and IR (Sinogram Affirmed Iterative Reconstruction - SAFIRE; I46) kernel. For image analysis and 3D-cinematic rendering technique, a dedicated post-processing and evaluation software (Syngo.via, MultiModality Workplace, version VE61A, Siemens Healthineers, Erlangen, Germany) was used.

2.8. Non invasive evaluation of LAA

To identify morphology and auricle's anatomy and to rule out LAA thrombi, TEE is the most frequently utilized diagnostic method. It is also the most common technique for intraprocedural imaging and choosing the device size for auricle closure. TTE represents an exam of fast and practical execution although not optimal in patients with esophageal pathology and unsuccessful TEE probe insertion [[28]].

For the evaluation of LAA morphology and size, the development of 3D ultrasound TTE is essential. It enables a more precise characterization of the LAA's dimensions and morphology, and 3D imaging is more useful in determining the structure (calcifications) and mobility of the thrombus itself than 2D imaging. For more, echocardiography 2D and 3D with contrast agents are helpful in situations where LAA pictures are not as clear as they should be. Contrast is used to reduce various artifacts and typically shows full opacification of the LAA or filling faults in its body [[28]].

When better spatial resolution is required for exclusion of thrombus, CCTA play an important role; the multi-planar reconstruction of axial acquisitions allows fully visualize the auricle and morphology and it is unavoidable for pre- and post-procedural evaluation of the LAA. In fact, it allows to establish, based on characteristics of LLA, the type of device to be used, its correct sizing and provides information about different transeptal puncture position [[2]].

2.9. Embryological considerations of relevance for LAA occlusion procedures

LAA is the only cardiac structure of the left atrium that derives from the primitive atrium [[36,37]]. All the other atrial structures derive from the fusion of the left pulmonary ostia [[36,37]]. The atrial auriclesbegin their development in the fifth week of gestation. At this point in time, the entire embryo is only some 4 mm long and the heart is essentially a primitive and looped tube. From then onwards, in only one week time, and by a process of 'ballooning', the left and right atria (as well as the ventricles) first become recognizable as parietal bulges on the heart tube (Fig. 8A), which then expand to embrace the outflow tract (Fig. 8B)[[36,37]].

During their initial development and throughout the fetal period, the atrial auriclesexhibit a relatively larger size proportionally compared to their size in adults (Fig. 8C–D)[[36]]. The variations in shape of the LAA that are described in the adult heart, such as chicken-leg and cauliflower types, are not obvious at this point. In most cases of the adult life, vestige of the left superior vena cava persists as an indentation of the myocardial surface between the LAA and the left superior pulmonary vein [[38]]. The Marshall vein/ligament is located inside such indentation, which has an average width of 11 ± 4 mm, and can be used as an anatomical landmark in endocardial and epicardial LAA occlusion procedures [[39]]. However, when the indentation is too narrow, iatrogenic injury the left superior pulmonary vein during LAA occlusion procedures may occur [[39]].

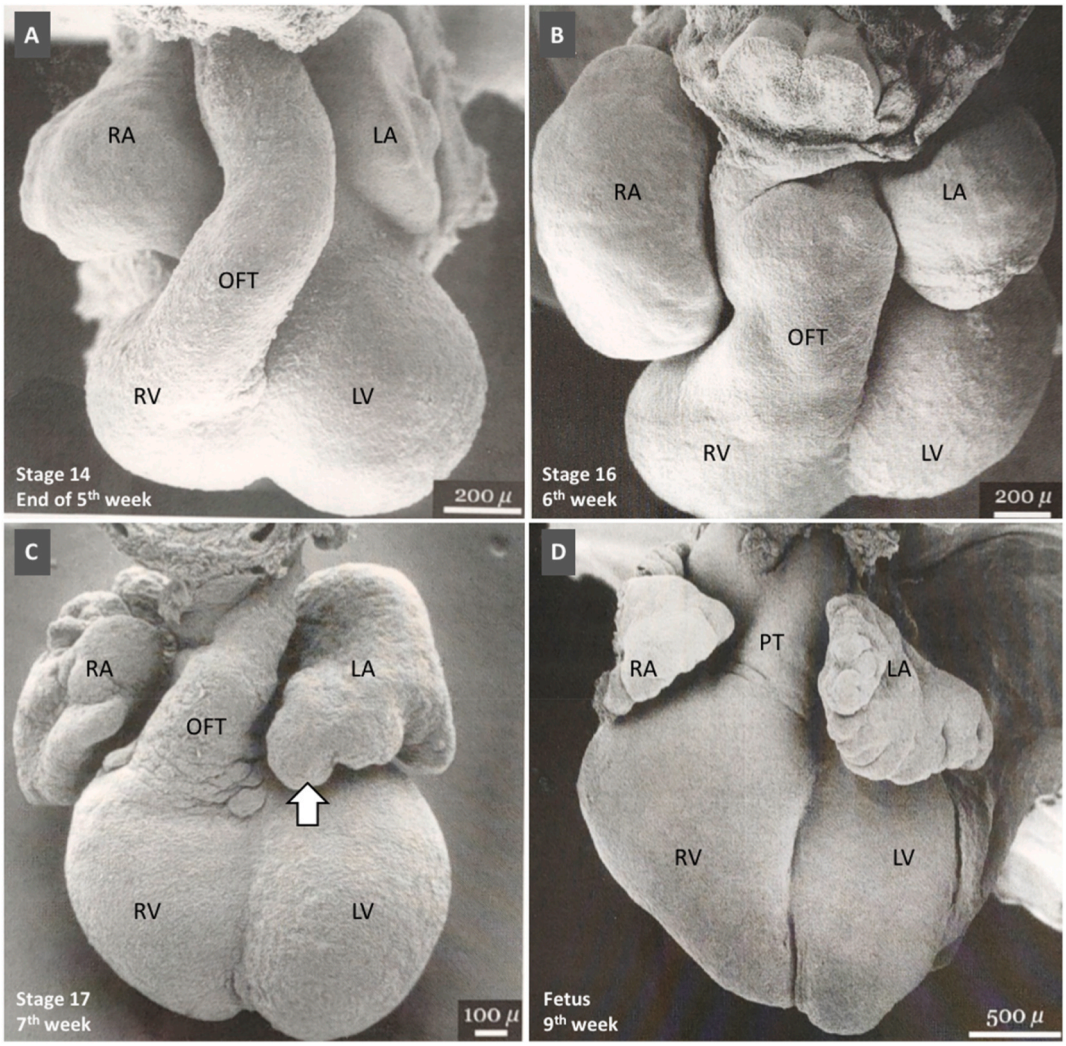

Fig. 8. Gestational development of the atrial auricles. **A**. Early chamber formation in the end of the fifth gestational week studied by Scanning **Electron** Microscopy. **B**. In the sixth week, the chambers are pronounced. **C**. In the seventh week, an adult-like LAA can be seen (white arrow) but the resemblance is transient only. **D**. In early fetal development, the left atrial appendage is proportionally large. All images are adapted from Oostra et al., 2007 [39] with permission. "Stage" refers to the Carnegie staging (1–23) of human embryos. LA, left atrium; LV, left ventricle; OFT, outflow tract; PT, pulmonary trunk; RA, right atrium; RV, right ventricle.

2.10. LLA functions

2.10.1. Mechanical and hemodynamic functions

The LAA can be considered a left ventricular systolic contractile reservoir; a proto-diastolic pulmonary veins-to-left ventricle blood outflow tract; an end-diastolic actively-contracting chamber increasing left ventricular filling; and a proto-systolic blood-suction chamber [[30,40]].

In 40–70% of patients in sinus rhythm evaluated withTEE, blood flows in and out of the LAA in a four-phase cycle [[41]]. Following the beginning of the proto-diastolic *trans*-mitral flow, the cycle starts with a first outflow phase from the LAA, followed by a short-lived inflow phase towards the auricle [[41]]. The first phase is tightly linked to the onset of the ventricular diastole, suggesting a causal correlation between the left ventricular relaxation and the early LAA emptying [[41]]. The LAA contraction generates a novel phase of auricular outflow throughout the atrial systole, followed by a final phase of inflow, plausibly triggered by auricular elasticity. This sequence is constant and independent from the heart rate [[41]]. When the LAA contracts physiologically with these phases, and the blood flow is normal, there is minimal risk of intra-auricular thrombi formation [[13]].

It has been proposed that systolic left ventricular distention within the pericardial cavity might aid auricular emptying by compressing the auricular infero-medial wall between the relatively stiff pericardium and the left ventricle free wall. Besides, ventricular filling produces an intracavitary suction effect, affecting atrial and auricular emptying and filling. Additionally, auricular emptying and filling are negatively correlated with the heart rate, with considerable implications for optimal heart rate control in atrial fibrillation-related thromboembolism.

Several studies have shown that the LAA has an intrinsic contractile activity, enabled by a rich apparatus of pectinate muscles, that explains the four-phases cycle of auricular emptying and filling, particularly at lower heart rates [[41]]. Furthermore, echocardiography and computed tomography imaging show active LAA contraction. In humans, LAA clamping in cardiac surgery can trigger an increase in LA pressure and dimensions and *trans*-mitral and pulmonary diastolic flow velocity [[40]]. Because of its distensibility, the LAA can improve its hemodynamic function by modulating the LA pressure-volume ratio during high pressure or volume overload states [[42]].

2.10.2. Endocrine functions

The LAA plays a significant endocrine function, as it is responsible for the synthesis and secretion of 30% of the atrial natriuretic peptide (ANP) produced in response to wall-stretching stimuli [[43]]. Animal studies have shown that the LAA stretching causes an increase in heart rate, urine formation, and sodium excretion [[44,45]], suggesting a neuroendocrine feedback driven by the secretion of ANP from stretched left auricular wall cardiomyocytes [[41]]. Hence, surgical removal of the LAA in animals may determine a reduction in pulmonary vein flow, an increase in *trans*-mitral diastolic flow [46] and a 50% drop in cardiac output [[46]]. In practice,

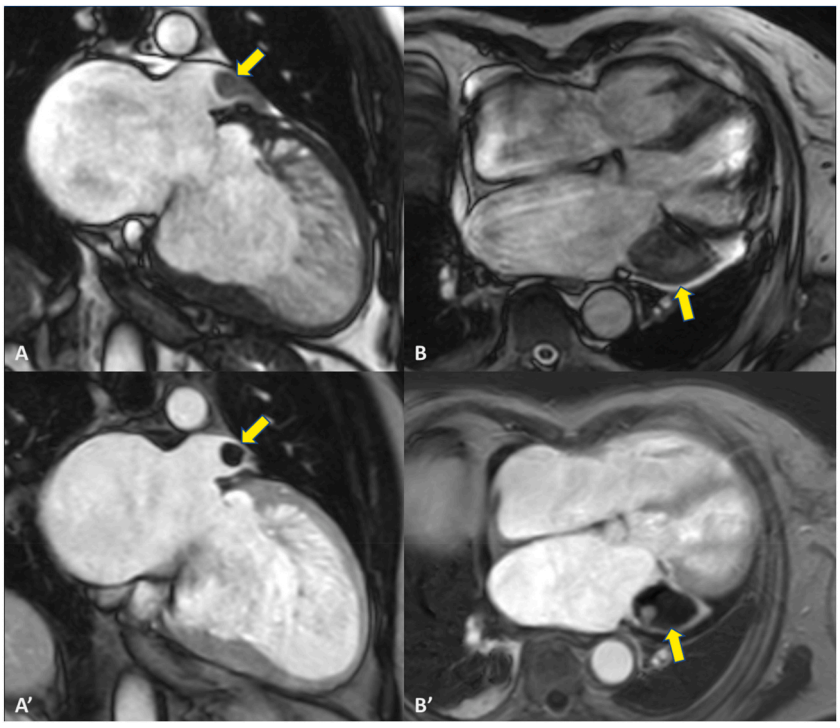

Fig. 9. Cardiac magnetic resonance images of LAA thrombi in a 50-year old man in sinus rhythm with previous catheter ablation of atrial fibrillation (vertical long axis view, A and A′) and in a 84-year old women with permanent atrial fibrillation (four chamber view, B and B′).

however, possibly due to a counterbalancing effect of the contralateral auricle, LAA closing procedures do not cause adverse effects or significant changes in human cardiac physiology [[47]].

In addition to its natriuretic function, the LAA is likely involved in regulating water distribution and homeostasis: animals with an injured LAA lose the ability to adjust their water intake through the sense of thirst when dehydrated or hypovolemic [[48]].

2.11. Pathophysiological and surgical implications of LAA anatomy

2.11.1. Thromboembolism in atrial fibrillation

Echocardiography-recorded AF episodes may highlight, in such conditions, decreased auricular contractility, decreased stretching, and a significant reduction of intra-auricular blood inflow velocity [[49]]. In AF, the standard biphasic model of auricular outflow is profoundly altered; occasionally it can deteriorate into a "sawtooth" pattern, up to the loss of any hemodynamic modelling of auricular emptying [[50]]. These factors favor blood stagnation in the highly trabeculated LAA cavity, frequently leading to thrombus formation [[49]]. Loss of efficient auricular contraction in AF leads to increased filling pressures, causing auricle dilatation and eventually blood stagnation around the pectinate and trabecular muscles, resulting in further auricular stretching and hypertrophy of pectinate muscles [[51]]. Indeed, TEE findings of reduced auricular contractility and contraction velocities are very strong predictors of cardioembolic stroke [[52]].

Patients with AF also have elevated hypercoagulability and pro-thrombotic markers, such as platelet factor 4, β-thromboglobulin, prothrombin fragment 1 + 2, and D-dimer [[51]], possibly only in part related to blood stasis. These markers are correlates of increased blood thrombogenicity. Besides, chronic AF elicits fibrosis and inflammation, which may cause endocardial microscopic remodeling, in turn also favoring the formation of thrombi [[51]], particularly in the LAA (Fig. 9A and B,A′,B′). Hence, 90% of thrombi in "non-valvular" AF (and 15–38% of those in non-AF-associated cardiac patients) originate in the LAA [[53]]. Thrombus formation occurs relatively quickly in AF: indeed, LAA thrombi have been found in up to 14% of patients with recently diagnosed (<3 days) AF [[54]]. Cardioembolic stroke has a remarkably high incidence in patients with AF (on average 5 fold higher than in patients without AF, although with a high variability depending on associated risk factors), and it is one of the most adverse outcomes to prevent in the course of interventional procedures aiming at LAA exclusion or closure (Fig. 10A–D) [55].

2.11.2. Non-atrial fibrillation thromboembolism

Patients with pronounced left ventricular disfunction and elevated left ventricular end-diastolic pressure are also at greater risk for LAA thrombi formation, even in the absence of AF [[13]]. As an important inference, the likelihood of thrombus formation in the LAA

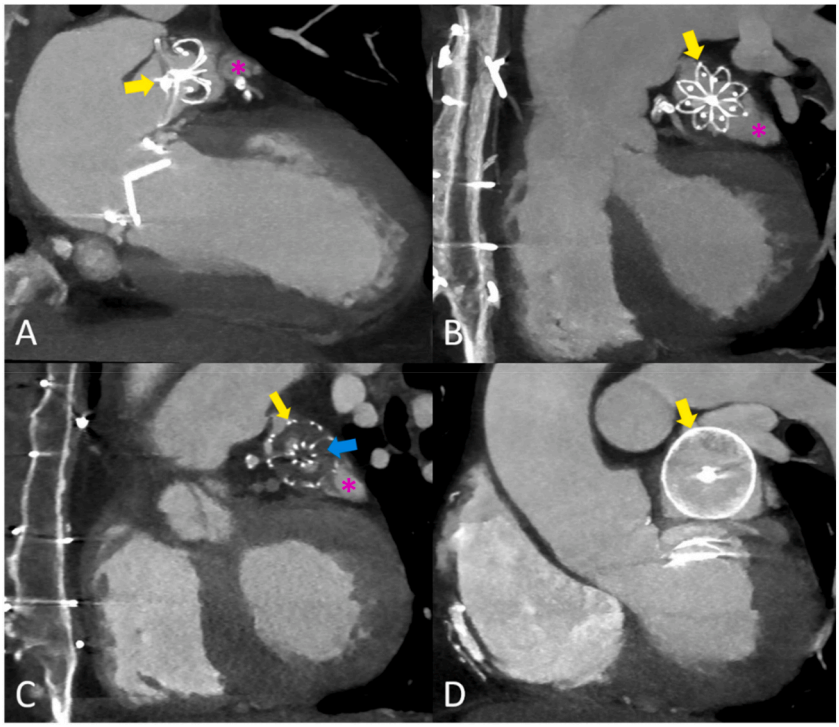

Fig. 10. (A–D): A case of LAA occlusion for stroke prophylaxis in a patient with non-valvular atrial fibrillation and high risk of cardioembolic stroke. Multiplanar CT reconstruction Images show a LAA occluding device (yellow arrows) appearing only partially thrombosed (blue arrow). Pink asterisks show a partially still open LAA. (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.)

appears to be related to worsened LAA contractile function and higher ventricular filling pressures, regardless of the underlying causes [[13]].

2.11.3. Arrhythmias

LAA can be a focal site of atrial tachycardia or a re-entry circuit site in 2–27% of patients with post-ablation relapsing AF [[23]]. Currently, there is no agreement on the best therapeutic strategy for this set of auricular arrhythmias, as present data are based on observational studies with short-term follow-ups [[15]]. Auricular electrical insulation might be a valid option, since few patients show AF relapses after 1-year [[15]]. Indeed, auricular ostium insulation with a circular catheter appears to be associated with a lower risk of relapses than site-specific ablation. Besides, LAA closure (Fig. 9A,B,A′,B') may result in electrical insulation, due to local necrosis, turning up to be a valid option both for treating atrial fibrillation and for preventing strokes [[15]]. LAA might also trigger arrhythmias in young adults without structural cardiac disease: about 3% of secondary abnormal-automaticity arrhythmias indeed originate from the LLA. Both the left auricular wall and the adjacent left atrium wall can be targeted by ablative procedures [[25]].

2.12. Left atrial auricle occlusion

Left atrial auricle occlusion (LAAO) is a procedure that is performed to reduce the risk of stroke in patients with non-valvular atrial fibrillation and contraindication to long-term anticoagulation therapy.

During LAAO, a catheter is inserted through a vein in the groin and guided to the heart and a small device, such as a plug or a clip, is then inserted into the LAA to permanently seal it off from the rest of the heart. This prevents blood clots from forming in the LAA and thus reduces the risk of stroke. LAAO is considered a minimally invasive alternative to other stroke prevention methods such as long-term anticoagulation therapy or surgical removal of the LAA.

Percutaneous Left Atrial Auricle Transcatheter Occlusion, the Watchman and Watchman FLX and the Amplatzer Amulet are the most common percutaneous devices developed for LAAO.

All devices have in common the same mechanism of anchoring to the auricle (self-expandable systems) and differ in material structure and size.

The first one (Percutaneous Left Atrial Auricle Transcatheter Occlusion) comprises of a self-expanding cage with a polytetrafluoroethylene-coated occlusive membrane on its atrial surface [[56]].

Watchman and Watchman FLX devices consists of a self-expanding titanium and nickel structure covered with a polyethylene terephthalate membrane and fixation tines that attach to the LAA orifice on the atrial surface. To guarantee device stability and adequate compression on the LAA wall, the chosen device should be bigger than the diameter of the ostium in the LAA. The device's width and length are almost the same.

The Amplatzer AMULET device consists of a proximal disc that covers the ostium and a self-expanding, sharp metal mesh that creates a distal lobe that fits into the LAA body. The device can be used in situations when the LAA anatomy is shorter because its length is less than its diameter..

3. The right atrial auricle

3.1. Outer shape (Fig. 11A–E)

The RAA is a large isosceles triangle-shaped, hollow muscular evagination with irregular margins [[57]]. It is divided into:

- a base ("ridge"), along the lateral side of the terminal sulcus;
- a convex lateral wall, slightly corrugated by thin and mostly parallel ridges as a reflex of the inner presence of pectinate muscles;
- a concave medial (deep) wall, suited to the convexity of the first tract of the ascending aorta;
- an apex, usually tied to the lateral wall of the ascending aorta.

The anterior-inferior margin of the RAA runs alongside the chondro-sternal sector of the right atrioventricular groove, and partially or entirely covers the corresponding tract of the right coronary artery. Sometimes such a margin may be included in fat tissue. Instead, the superior margin is downwardly oblique, and surrounds the convexity of the first tract of the ascending aorta. The RAA is widely communicating with the right atrium. It shows an average height (anterior-posterior distance) of 21.6 ± 3.4 mm (*range* 16–29 mm) and a base of 26.6 ± 4.3 mm (*range* 20–36 mm), according to Veinot et al. and Manolis et al. findings [[57]]. Typically, the RAA increases in size in patients affected by AF. Transesophageal echocardiographic studies have demonstrated that the RAA average superficial area is 4.7 ± 1.6 cm^2 in patients with sinus rhythm, whereas in becomes higher (5.6 ± 1.9 cm^2) in AF [[57]].

3.2. Inner structure

The RAA has an endocardial wall, well highlighted in endoscopic images, such as those of the "Atlas of Human Cardiac Anatomy" [[58]], characterized by a medial border represented by the crista terminalis and a pectinated anterior-lateral wall distinguished by the adjacent smooth endocardial surface called *sinus intercavarum*. The inferior border reaches the tricuspid vestibule. The RAA inner surface includes the:

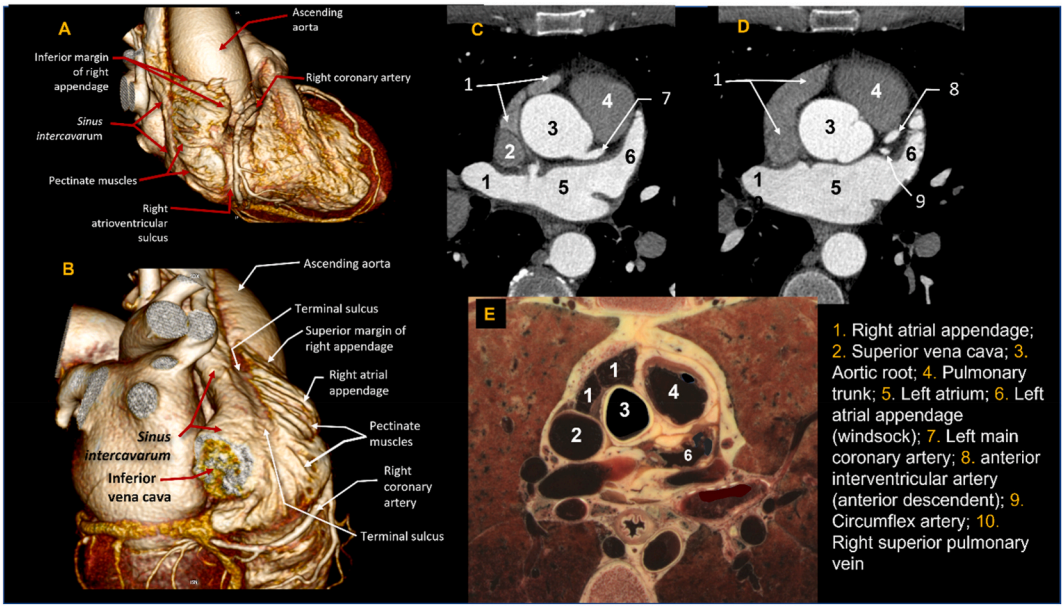

Fig. 11. (A–E). Volume rendering (A,B) and Axial reconstruction (C,D) CT images highlighting right and LAA main features and its anatomical landmarks. Axial section through human thorax showing LAA and RAA (E; courtesy of Prof. R.D. Hersch. École Polytechnique Federale de Lousanne (EPFL). Switzerland. Visible Human Web Server. http://visiblehuman.epfl.ch).

1. Crista terminalis
2. Taenia sagittalis (sagittal bundle)
3. Pectinate muscles

The sagittal bundle is a thick muscular cord that originates from the proximal tract of the crista terminalis and sagitally reaches the anterior border of the RAA, between the medial third and the lateral two thirds. Few studies have investigated the detailed anatomy of the RAA [[58]]. According to Loukas et al., the taenia sagittalis has been reported in 85% of cases (45/300), while in the studies by Siddiqui et al. and Ueda et al. it was present in 80% (30/150) and 89.7% of cases (35/39), respectively. The RAA has been described as a single thick muscular cord in some subjects, ranging from 55% to 88.6%, whereas it has been depicted as a double muscular cord in 20–25% [59]. The average thickness of the taenia sagittalis is 0.4 mm (*range*, 0.2–0.6 mm) and its length ranges from 4 to 15 mm, with an average of 12 mm [59]. According to Sanchez-Quintana et al., pectinate muscles are particularly numerous and create an anastomosed net at the tip of the RAA, with a characteristic "coral shape" [[59]]. In 80% of cases, pectinate muscles are widely intertwined due to several thin irregular decurrent trabeculae, while parallel muscular trabeculae associated with interposed lacunae are observed in 20% of cases [[59]]. Microscopic analyses have demonstrated that cardiomyocytes run parallel to the crista terminalis and the sagittal bundle [[60]]. Ueda et al. have hypothesized that the sagittal bundle may resemble a ring structure around the extremity of the RAA, suggesting a potential reentry pattern of the electrical impulse originating from the sinus node [[59]]. The preferential conduction pathway in the RAA may be cranio-caudal and postero-anterior, due to the macroscopic and microscopic position of the crista terminalis and the sagittal bundle [[59]]. Although the preferential trigger site of left-sided cardiac thrombi is the LAA in patients with AF, the RAA can also contribute to thrombus formation and pulmonary or even systemic emboli in such conditions, although more rarely (in the latter cases due to paradoxical migration across patent foramen ovale.

4. Conclusions

In the last decade, understanding of the structure and function of the LAA, as well as the RAA, has significantly advanced due to the growing utilization of transcatheter or surgical LAA exclusion or closure, and recognition of the LAA as a trigger site for recurrent AF post-catheter ablation. An accurate knowledge of LAA morphology, with the use of CT, cardiac MRI and TEE is important for the accurate planning of exclusion/closure interventions and for selected ablation procedures.

Author contribution statement

All authors listed have significantly contributed to the development and the writing of this article.

Data availability statement

No data was used for the research described in the article.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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