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Review

Morphology and pathophysiology of target anatomical sites for ablation procedures in patients with atrial fibrillation. Part I: Atrial structures (atrial myocardium and coronary sinus)

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ABSTRACT

Experimental and clinical evidence suggests that the natural history of atrial fibrillation is characterised by increased structural remodelling, which may play a pivotal role in maintaining the arrhythmia and clinically favours progression from paroxysmal to persistent atrial fibrillation. In this setting, anti-arrhythmic therapy gradually becomes inefficient, and this limitation has led to the introduction of new non-pharmacological interventions such as surgical or catheter ablation. At the same time, interest in the functional morphology and electrophysiological properties of the atria and their related anatomical structures has greatly increased. This article is the first of a two-part review whose main purpose is to describe the anatomical and functional details of some of the principal anatomical locations that are commonly targeted by ablative procedures to treat this supraventricular arrhythmia. In particular, this manuscript has dealt with the atrial structures (atrial myocardium and coronary sinus). General information on ablation procedures has also been provided.

1. Introduction

1.1. Background and aims

Atrial fibrillation (AF) behaves as a progressive disease in which the arrhythmia itself may induce further structural changes and a worsening in the underlying diseases, thus creating a vicious circle ("AF begets AF") that does nothing but make the myocardial architecture distortion worse, and very often leads to paroxysmal AF becoming persistent or permanent [1,2]. Structural remodelling only seems to be reversible during the first phases of the arrhythmic disorder, but its extent is crucial because it may reach a threshold beyond which sinus rhythm can no longer be restored [1].

The inadequate long-term efficacy of anti-arrhythmic therapy and the proportion of patients who discontinue treatment because of side effects have been the major reasons for the development of non-pharmacological interventions in the treatment of AF. The aim of atrial ablation is to create lines of irreversible myocardial necrosis (with subsequent scarring) around the atrial tissue involved in sustaining the arrhythmia. This is in order to disconnect the players involved in the initiation and maintenance of AF, i.e. triggers (pulmonary vein, PV, and non-PV foci, see also Part II of this review) in the presence of an underlying structural–functional substrate favouring the perpetuation of the arrhythmia [3,4].

The electrophysiological remodelling of AF is manifested by those atrial myocyte electrical changes that take place during the first few hours of sustained atrial tachycardia in such a way to promote both the occurrence and maintenance of the arrhythmia. Structural remodelling is a subsequent and slower process that encompasses all of the morphological changes affecting the atrial myocardial architecture and ultrastructure (mainly the interstitial fibrosis), and seems to play a crucial role as an important contributor to the AF substrate in the initiation and perpetuation of the arrhythmia [1,5].

Ablation with isolation of the pulmonary veins (PVs) is very often successful in curing many patients with paroxysmal AF but its success is limited in some of these and in most patients with persistent/permanent AF, probably because of atrial remodelling beyond the PVs. Non-PV triggers of AF can be identified in a significant number of patients referred for catheter ablation [6,7]. The most frequent of these are the posterior

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wall of the left atrium (LA), the superior vena cava (SVC), the coronary sinus (CS), the ligament of Marshall, and the region adjacent to the AV valve annuli; furthermore, the atrial ganglionated plexi may also play a significant role in the pathogenesis of AF [3].

This article is the first of a two-part review that aims to describe the anatomical and functional characteristics of atria with some specific references regarding the main anatomical locations targeted by ablative procedures to treat AF. This first part will deal with structures related to atrial chambers (atria and CS), while the second one will consider non-myocardial atrial-related locations (i.e. PVs, ganglionated plexi, caval veins, ligament of Marshall). General information about site-specific ablation procedures will also be provided.

A complete PubMed search was used to identify full-text, English-language articles published between 1960 and 2012, from which we selected study papers, recently published review articles, editorials from peer-reviewed journals, and book chapters.

2. Atrial chambers

Unlike that of the ventricular chambers, the atrial conduction system is only located within the sino-atrial node (SAN) and atrioventricular node (AVN). Consequently, the cardiac impulse generated in the SAN is conducted to the atria and the AVN through the non-specific working atrial myocardium, making it vital that atrial muscle architecture is preserved [8]. What follows are some generalities concerning the gross anatomy and main muscle bundles of the atria.

2.1. Left atrium anatomy

When observed from its chamber, the left atrial appendage (LAA) is smaller than that of the RA, finger-like, and its junction towards the main atrial chamber is not delimited by any crest or groove (Fig. 1A). However, its endocardial aspect is lined by pectinate muscles that are usually much less pronounced than those of the RAA [9,10].

The outlets of the four PVs end posteriorly at the LA posterior wall (Fig. 1B and C). However, this region is subject to fairly frequent anatomical variations, particularly the number of veins (e.g. early branching of the right inferior PV, supernumerary PVs, right top PV, and the common ostium of the homolateral PVs) [11,12]. As in the case of the SVC, although the border between the atrial myocardium and vein is basically smooth, irregular sleeves of atrial cardiomyocytes (up to 25 mm long) extend over the veno-atrial junction and into the vein walls. Interestingly, these sleeves have intrinsic electrical activity [13].

The smooth mitral valve vestibule merges with the smooth wall of the LA body, and represents part of the "mitral isthmus" (MI), which is located between the left inferior PV and the mitral valve annulus. Moreover, the posterior LA vestibule overlaps the wall of the CS [14,15]. Even though this postero-inferior area of the lateral LA wall cannot be considered a true anatomic entity, it is now officially known by electrophysiologists as the "LA isthmus" or MI [16]. In a recent autopsy study, Becker investigated the size of the MI [17]. The average distance between the left inferior PV and mitral valve was 34.6 mm (range 17–51 mm). Average atrial thickness at the level of the left inferior PV was 3.0 mm but varied widely (range 1.4–7.7 mm); at the level midway between the PV and the mitral annulus, it was 2.8 mm (range 1.2–4.4 mm) and at the site of the mitral valve annulus, it was 1.2 mm (range 0–3.2 mm). Given this variability in atrial myocardial MI thickness, the specific anatomical characteristics of the MI region can also be investigated before an ablation procedure by means of multislice computed tomography [18].

2.2. Right atrium anatomy

In comparison with the LA, the RA has a more complex architecture and the roughly triangular-shaped RA appendage anterolaterally dominates its internal and external aspect. When observed from its chamber, the boundary between the smooth endocardial surface of the venous component and the ridged wall of the right atrial appendage (RAA) is marked by the largest muscle bundle of the right atrium (RA), the horseshoe-shaped "terminal crest" (Fig. 2A) that runs from the left-hand side of the SVC ostium, descends to the right of the inferior vena cava (IVC), and ends in the cavo-tricuspid isthmus (between the IVC and the tricuspid valve) by way of ramifying fibres [19,20]. In particular, the cavo-tricuspid isthmus forms a quadrilateral area bounded by the Eustachian valve and the Thebesian valve (see below), the tricuspid valve, and a line connecting the IVC and the tricuspid valve [21]. A series of thick bundles (the anterior pectinate muscles) stem laterally from the origin of the terminal crest, extend around the tricuspidal orifice, and join the interatrial septum in the sub-Thebesian sinus area (see below). In the terminal crest distal portion, the pectinate muscles are more widely spaced and fan out towards the tricuspidal vestibule [19].

The right-hand part of the RA is represented by its intercaval area (the venous component) (Fig. 2A). The orifice of the SVC has no valves but, occasionally, its distal portion has irregular sleeves of atrial myocardium extending into the venous adventitia [22]. The SAN is usually located within the terminal crest at its antero-lateral junction with the SVC [15]. Unlike the SVC, the IVC ostium is delimited by the essentially vestigial Eustachian valve — a triangular fibrous (or fibro-muscular) flap that extends from the lateral part of the orifice and reaches the border between the CS and the oval fossa [8,10].

The CS, whose ostium is guarded by a usually crescent-shaped and incomplete valve (the Thebesian valve) is located inferiorly, adjacent to the right side of the IVC orifice (Fig. 2B) [15].

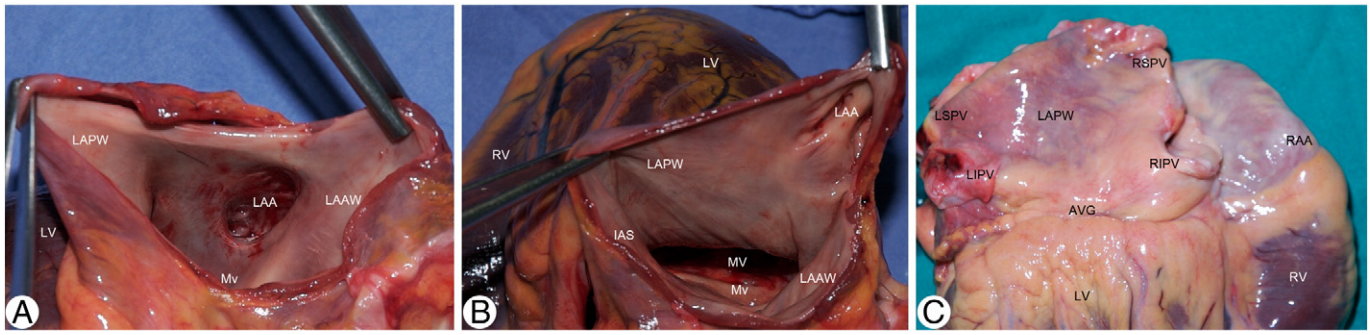

Fig. 1. Left atrium (human heart). A) Ostium of the left atrial appendage. B) Superior view of the left atrial chamber. C) External posterior view of the left atrium. Abbreviations. AVG: atrio-ventricular groove; IAS: interatrial septum; LAA: left atrial appendage; LAAW: left atrial anterior wall; LAPW: left atrial posterior wall; LIPV: left inferior pulmonary vein; LSPV: left superior pulmonary vein; LV: left ventricle; MV: mitral valve; Mv: mitral vestibulum; RAA: right atrial appendage; RIPV: right inferior pulmonary vein; RSPV: right superior pulmonary vein; RV: right ventricle.