ELSEVIER

Review

Atrial structure and fibres: morphologic bases of atrial conduction

Siew Yen Ho[a],*, Robert H. Anderson[b], Damián Sánchez-Quintana[c]

[a]*Paediatrics, Faculty of Medicine, National Heart and Lung Institute, Imperial College of Science, Technology and Medicine, and Royal Brompton and Harefield NHS Trust, London, UK*
[b]*Cardiac Unit, Institute of Child Health, University College, London, UK*
[c]*Department of Anatomy, Universidad de Extremadura, Departamento de Anatomía Humana, Facultad de Medicina, Badajoz, Spain*

Abstract

The relationship between anatomy and function has long been recognised. Understanding the gross structure, and the myoarchitecture, of the atriums is fundamental to investigations into the substrates and therapy of atrial fibrillation. Based primarily on our experience with normal human hearts, this review provides, firstly, a basis of comparison of gross structures as seen in the clinical situation, and in animals commonly used in experimental studies. Secondly, we discuss the general arrangement of myocardial fibres with respect to gross topography in the normal human heart. The right atrium is dominated by an extensive array of pectinate muscles within the extensive appendage, whereas the left atrium is relatively smooth-walled, with a much smaller tubular appendage. Myoarchitecture displays parallel alignment of fibres along distinct muscle bundles, such as the terminal crest and Bachmann's bundle. Within the smooth wall of the left atrium, there is a marked transmural change in the orientation of the muscular fibres. Abrupt changes in orientation, and mixed arrangements, are common between bundles. Other than Bachmann's bundle, the muscular bridges which provide interatrial connections, and connections between the left atrium and the coronary sinus and inferior caval vein, are highly variable. Inhomogeneities both in gross structure and myoarchitecture are common in the normal heart. These should be taken into account when investigating hearts from patients known to have had a history of arrhythmias, in devising computer models, or when refining diagnostic and therapeutic strategies.
© 2002 Elsevier Science B.V. All rights reserved.

Keywords: Ablation; Arrhythmia (mechanisms); Computer modelling; Histo(patho)logy; Sinus node; Supraventr. arrhythmia; Veins

1. Introduction

Describing Harvey as the 'patron saint' of anatomists, Arthur Keith, in his Harveian Lecture [1] given in 1918, referred to the architecture of the musculature of the heart as a 'well-worn theme' of the 17th century. In his earlier treatise written in 1907 on the jugular pulse, Keith [2] had opined that each atrium and ventricle contained two sets of muscular fibres—circular and longitudinal. He suggested that the circular fibres in the right atrium were for compressing the chamber and expelling the blood, while the longitudinal fibres were antagonists of those to be found in the right ventricle. Along with his associate Flack [3], Keith [2] had explored extensively the anatomical substrates for atrial contraction and excitation. According to Keith [1], such meticulous work had been dismissed by Hunter, a century and a half after the days of Harvey, in the phrase 'Much more pains than were necessary have been taken to dissect and describe the course and arrangement of the muscular fibres of the heart'. It is not surprising, therefore, that myocardial architecture was subsequently ignored. This has continued through the last century. Thus, the architecture of atrial musculature in human, in particular, is scarcely mentioned, apart from the aforementioned studies of Keith and Flack [1,2,4], those of Papez [5] in the early part of the century, and our own works in the latter part [6,7]. Our interest had been prompted by the developments in diagnostic technology

---

*Corresponding author. Paediatrics, National Heart and Lung Institute, Imperial College of Science, Technology and Medicine, Dovehouse Street, London SW3 6LY, UK. Tel.: +44-20-7351-8752; fax: +44-20-7351-8230.

E-mail address: yen.ho@ic.ac.uk (S.Y. Ho).

Time for primary review 31 days.

that allowed atrial function to be analysed in the clinical setting. Concurrently, innovations in electrophysiologic mapping within the cardiac chambers stimulated a need for better understanding of the gross arrangement of atrial musculature to provide a morphologic basis for atrial conduction. In this review, so as to set the scene, first we describe the major components of the atriums and their variations in normal human hearts. We then make comparisons with atrial structure in animals that are commonly used in experimental studies. Finally, we describe the normal atrial myoarchitecture as seen in the human. In our opinion, interpretation of the findings from patients with atrial arrythmias, as and when these become available for study, will be impossible without a firm knowledge of normal structure and its variations.

2. Gross structures

In order properly to appreciate the clinical significance of atrial structure, it is vital that the arrangement of the cardiac components be described in attitudinally correct orientation. Thus, when viewed from the front, the cavity of the right atrium is positioned to the right and anterior, while the left atrium is situated to the left and mainly

posteriorly (Fig. 1A,B). The front of the left atrium lies just behind the transverse pericardial sinus, which is bordered anteriorly by the aortic sinuses. The posterior wall is just in front of the tracheal bifurcation. Owing to the angulation of the entrances between the superior and inferior caval veins, the posterior wall of the right atrium in humans is slightly indented. In mammals, like dogs and horses, the angulation is sharper, with a distinct fold seen inferior to the entrance of the superior caval vein. This is described as the tubercle of Lower [8]. In these mammals, the right pulmonary veins pass in the intercaval angle. In man, in contrast, the right superior pulmonary vein passes posterior to the superior caval vein, with the right inferior vein passing behind the venous sinus of the right atrium (Fig. 1B). The musculature from the right atrium extends up to 1 cm over the wall of the superior caval vein in the dog, allowing electrical activity to be recorded in the vein [9]. In man, the muscular extension is short along the superior caval vein, but hardly found in the inferior caval vein, rendering a discrete termination of conduction propagation inferiorly.

The atrial septum, best appreciated in the transverse plane, runs obliquely from the front, extending posteriorly and to the right. The pulmonary veins join the posterior part of the left atrium, with the orifices of the left
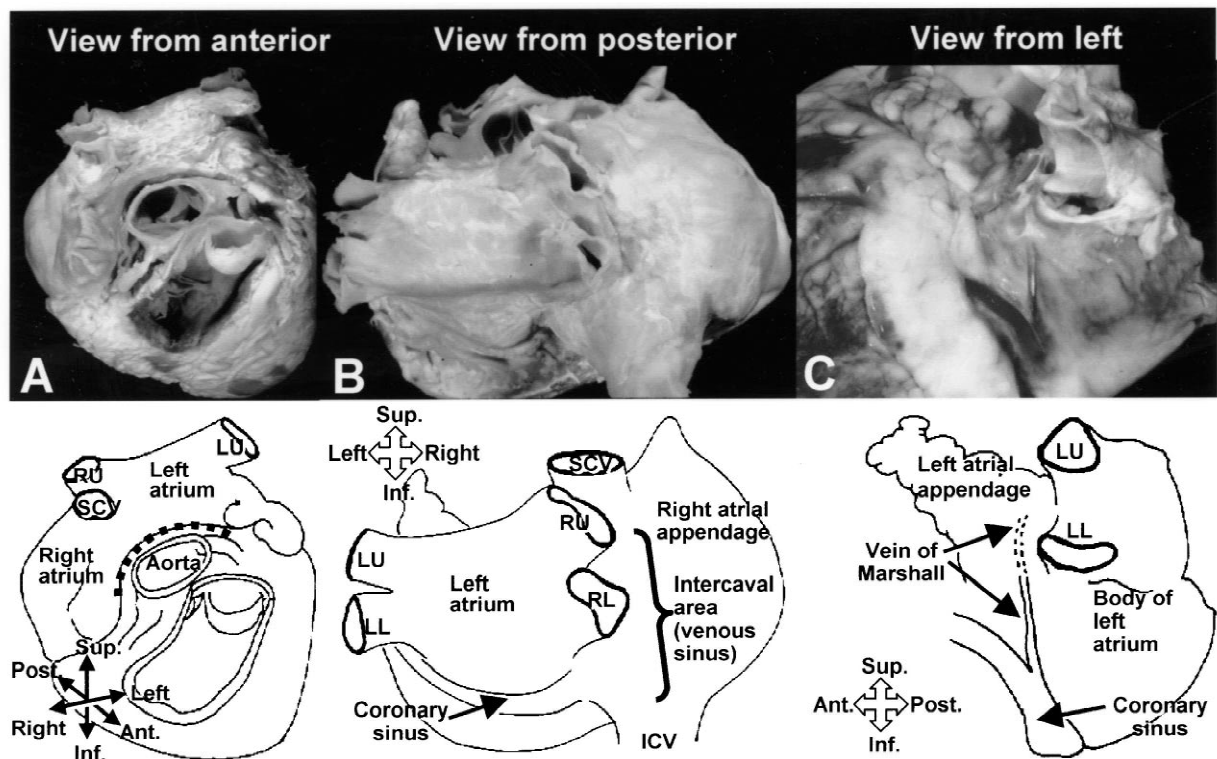

Fig. 1. Normal human hearts with corresponding diagrams. (A, B) Show the spatial relationships of the atrial chambers. The transverse pericardial sinus (dotted line) runs between the aorta and the anterior wall of the left atrium. The right upper pulmonary vein (RU) passes behind the superior caval vein (SCV). (C) Shows the finger-like left atrial appendage anterior to the course of the vein of Marshall. Parallel broken lines mark the vein obscured by epicardial fat. Ant., anterior; ICV, inferior caval vein; Inf., inferior; LL, left lower; LU, left upper pulmonary vein; Post., posterior; RL, right lower pulmonary vein; Sup., superior.

pulmonary veins being more superiorly located than those of the right pulmonary veins. In the human, it is most common to find two orifices on each side (Fig. 1B). Sometimes, two veins of one, or both, sides are united prior to their entry to the atrium. In others, an additional vein is found, more frequently on the right side. Five or six orifices are described for the canine heart, but there may be fewer orifices when some veins become confluent before entering the atrium [8]. Related posteriorly and inferiorly to the left atrium is the coronary sinus, which is the continuation of the great cardiac vein (Fig. 1C). It occupies the left atrioventricular groove. The oblique vein of Marshall, located between the left atrial appendage and the left upper and lower pulmonary veins, runs inferiorly along the inferior atrial wall to join the coronary sinus (Fig. 1C). This vein is obliterated for the most part in the majority of individuals. It remains patent as an isolated malformation, the persistent left superior caval vein draining into the coronary sinus, in 0.3% of the normal population. Much more frequently, persistent patency is associated with other congenital cardiac malformations. In some mammals, especially pigs and other ruminants, persistent patency of the oblique vein is the norm. Somewhat confusingly, the structure is called the azygos vein in veterinarian texts, since the vein also drains the return from the parietal chest wall in these animals.

2.1. The right atrium

A muscular sack, the wall of the right atrium is best considered in terms of three components — the appendage, the venous part, and the vestibule (Fig. 2A,B). There is then a fourth component, the septum, which is shared by the two atriums (Fig. 2A). From the epicardial aspect, the dominant feature is the large, triangular shaped, appendage which is located anterior and laterally (Fig. 2B). Usually, a fat-filled groove corresponding internally to the terminal crest ('crista terminalis' or tænia terminalis) can be seen along the lateral wall. This is the terminal groove, or sulcus terminalis. The sinus node is located in this groove, close to the cavoatrial junction (Fig. 2B, C). Even viewed from the outside, the extensive array of pectinate muscles so characteristic of the right atrium is evident. Arising from the terminal crest, the pectinate muscles spread throughout the entire wall of the appendage, reaching to the lateral and inferior walls of the atrium. Inside the atrium, the branching and overlapping arrangement of the pectinate muscles is clearly visible. In between the ridges
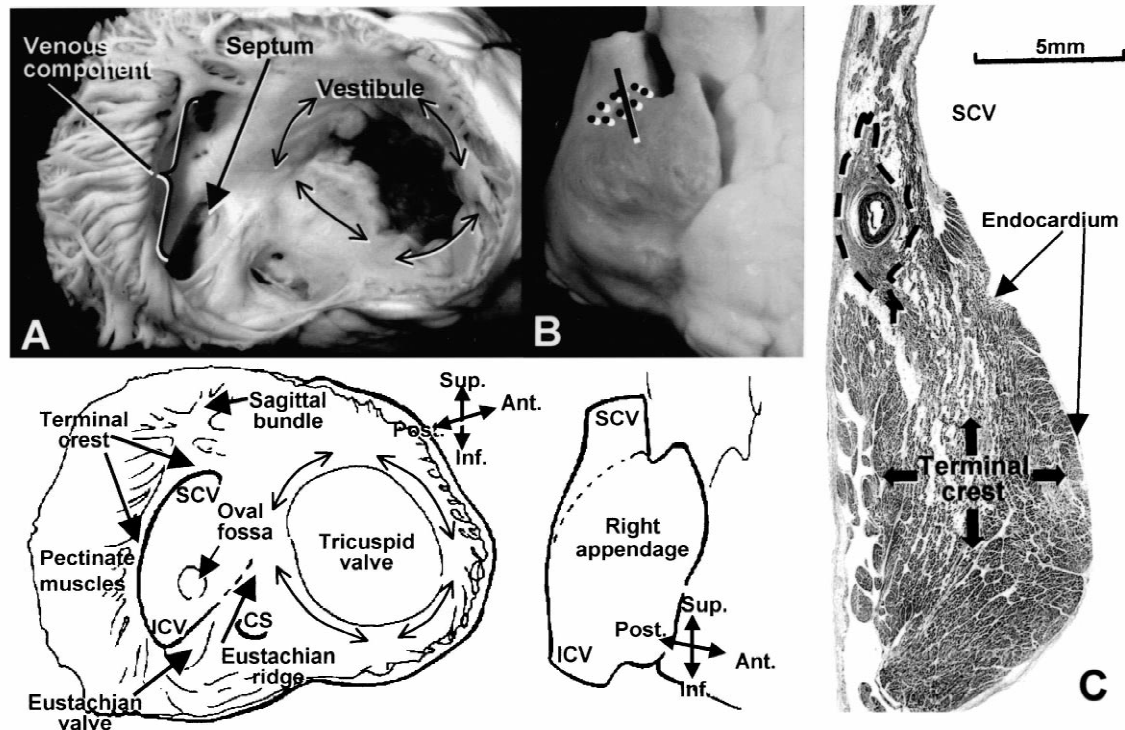

Fig. 2. (A) is a simulated right anterior oblique view of the inside of the right atrium displayed by incising the lateral atrial wall and reflecting it back. The corresponding diagram shows the atrial components. The terminal crest separates the smooth wall of the venous sinus from the rough wall of the appendage. This en face view of the septal surface gives a false impression of an extensive atrial septum. (B) shows the broad triangular-shaped appendage forming the major portion of the right atrium. The dotted oval marks the sinus node which is located in the terminal groove (indicated by broken line on the diagram). The solid line indicates the plane of the histological section shown on panel (C). Cut in cross-section, the sinus node (within broken line on C) and its arterial supply occupies the subepicardial region in relation to the musculature of the terminal crest (Masson's trichrome stain). CS, coronary sinus. Other abbreviations as in Fig. 1.

of pectinate muscles, the wall is very thin and almost parchment-like. Although extensively arranged, the pectinate muscles never reach the orifice of the tricuspid valve. Always, a smooth muscular rim, the vestibule, surrounds the valvar orifice, with the musculature inserting into the valvar leaflets (Fig. 2A). The venous component receiving the caval veins is again characterised by the smoothness of its walls. The terminal crest marks the junction of the venous and rough zones.

The eustachian valve, guarding the entrance of the inferior caval vein, is variably developed. Usually it is a triangular flap of fibrous or fibro-muscular tissue that inserts medially to the eustachian ridge, or sinus septum, which is the border between the oval fossa and the coronary sinus (Fig. 2A). In some cases, the valve is particularly large and muscular, posing an obstacle to catheters passed from the inferior caval vein to the inferior part of the right atrium. Occasionally, the valve is perforated, or even takes the form of a delicate filigreed mesh. When it is extensive and stretches across to the superior caval vein or the tubercle of Lower, it is described as a Chiari network [9]. The free border of the eustachian valve continues as a tendon that runs in the musculature of the sinus septum. It is usually described as the tendon of Todaro [10–12]. It is one of the borders of the triangle, named for Koch, that delineates the location of the atrioventricular node. In attitudinal orientation, the node is within the triangle's apex pointing superiorly (Fig. 3A, B). The concept of Koch's triangle has been disputed recently [13], despite its known value to cardiac surgeons and electrophysiologists alike [14]. These protestations are unjustified, since its boundaries are clear-cut. Thus, the anterior border is marked by the hinge of the septal leaflet of the tricuspid valve. Superiorly, the central fibrous body is the landmark for penetration of the bundle of His. The

inferior border of the triangle is the orifice of the coronary sinus, and the vestibule immediately anterior to it (Fig. 3). This part of the vestibule, forming a discrete and apparently septal isthmus, is the area often targeted for ablation of the 'slow pathway' in atrioventricular nodal re-entrant tachycardia. Contrary to its designation, however, the musculature of the isthmus is not strictly septal. It is formed by the thin atrial wall which overlies the ventricular musculature (Fig. 4A,B). The laminar arrangement is particularly well displayed in dissections into the inferior pyramidal space, including surgical procedures for avulsion of accessory atrioventricular pathways [15–17]. The so-called 'fast pathway' corresponds to the area of musculature close to the apex of the triangle of Koch [18–20].

A small crescentic flap, the Thebesian valve, usually guards the orifice of the coronary sinus. Frequently, the valve is fenestrated. An imperforate valve completely covering the orifice is very rare. The atrial wall inferior to the orifice of the coronary sinus is usually pouch-like. It is often described as the sinus of Keith, or the sub-eustachian sinus. In attitudinal orientation, the pouch is anterior to the orifice of the inferior caval vein and sub-Thebesian. It forms the posterior part of the so-called 'flutter' or 'lateral' isthmus, between the inferior caval vein and the tricuspid valve (Fig. 4A, C, D). In simulated right anterior projection, this isthmus is the 'inferior' border of the right atrium, not lateral (Fig. 3A). In contrast to the shorter and smooth septal isthmus, three morphologic zones are usually distinguishable in the inferior isthmus [21]. It receives a variable number of muscular branches of varying morphology from the terminal crest (Fig. 4C, D). The most posterior zone is often fibrous, the middle zone is trabeculated, made up of the extensions of the pectinate muscles, while the anterior zone is the smooth vestibular wall.

2.2. The atrial septum

When seen from the right atrial aspect, the septum is limited to the floor of the oval fossa, the latter being surrounded by a raised muscular rim. The extensive musculature seen antero-medially relative to the fossa is not septal, being the wall of the atrium lying immediately behind the aorta (Fig. 2A). The superior rim of the fossa is then the infolded wall between the superior caval vein and the right pulmonary veins (Fig. 5A). In about one-quarter to one-third of the normal population, there is probe patency of the oval fossa, even though the valve is large enough to overlap the rim. This is because the adhesion of the valve to the rim is incomplete. This leaves a gap, usually in the antero-superior margin, corresponding to a sickle-shaped mark in the left atrium just behind the anterior wall (Fig. 5B). Thus, the left aspect of the atrial septum lacks the crater-like feature of the right side. The valve itself is usually fibrous, being populated by relatively few myocytes. The superior and posterior margins of the oval fossa are infolded to produce the prominent muscular
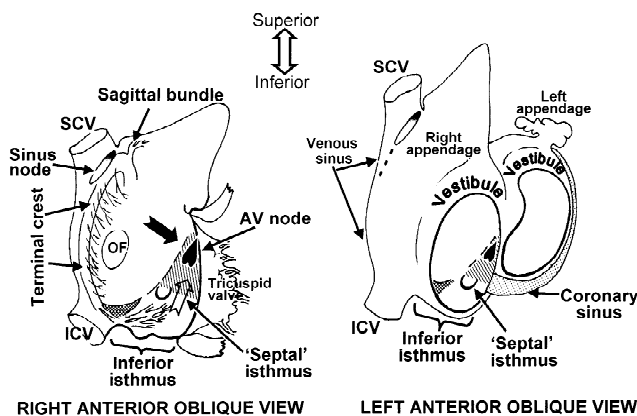

Fig. 3. Schematic representations to show the relationship of the atrial structures in standard fluoroscopic views. The triangle of Koch is marked by diagonal hatching and the eustachian valve by cross hatching. The putative fast and slow pathways are represented by filled and open notched arrows respectively. The sinus node (shaded oval) is located in the terminal groove (dotted line). AV, atrioventricular. Other abbreviations as in Fig. 1.
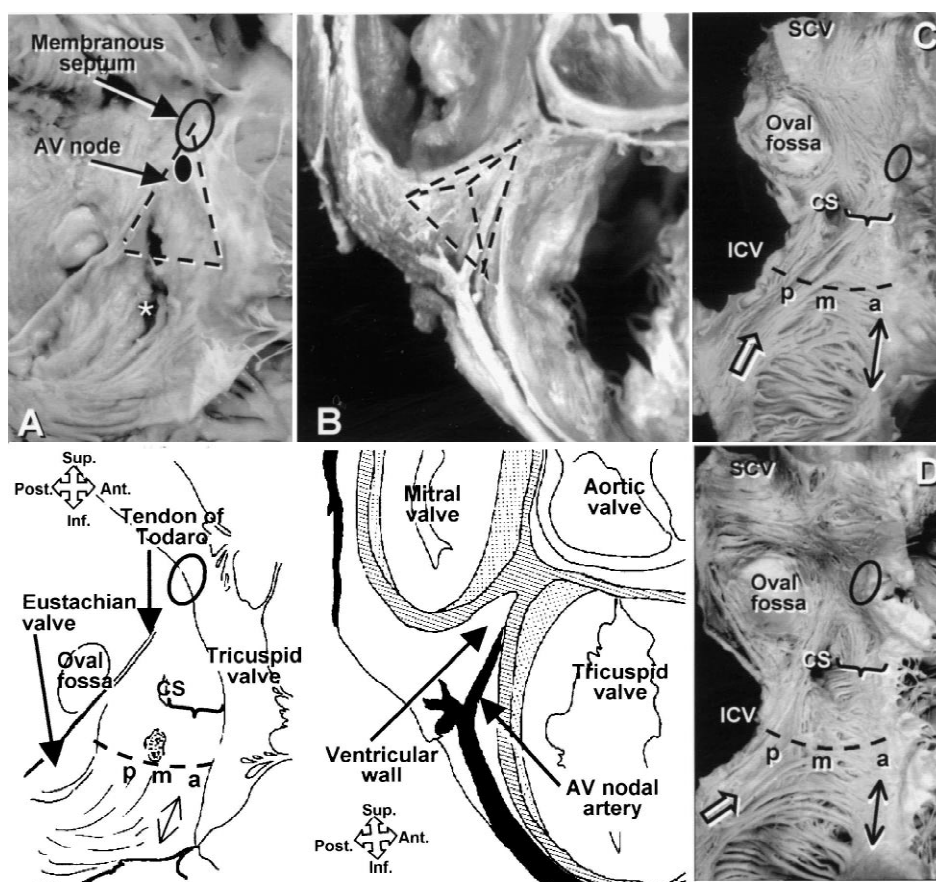
Fig. 4. (A) is a simulated right anterior oblique view showing the triangle of Koch demarcated by the tendon of Todaro posteriorly, the tricuspid valve anteriorly, the coronary sinus inferiorly and the central fibrous body (O) at the apex. The 'septal' isthmus (}) is part of the smooth vestibule (↔). The inferior isthmus (---) lies between the orifice of the inferior caval vein and the tricuspid valve. The eustachian valve guarding the inferior caval vein is large in this heart. Note the pouch (★), or sinus of Keith, inferior to the coronary sinus and the muscular rim around the oval fossa. (B) is a dissection displaying the inferior pyramidal space. The corresponding diagram shows the atrial walls (stippled) and their cut edges (hatching) overlying ventricular myocardium. (C and D), in similar orientation as A, display the internal aspects of the isthmic region with the lateral wall retracted inferiorly. The endocardium has been removed to display the myoarchitecture. (p, m and a) indicate the posterior, middle and anterior zones of the inferior isthmus. Note the parallel alignment of the fibres in the body of the terminal crest (open arrow) and the divisions distally in the approach to the inferior isthmus. C shows a major fascicle continuing into the middle zone while D shows fine multiple branches. AV, atrioventricular; CS, coronary sinus; ICV, inferior caval vein; SCV, superior caval vein.

rims in these areas. These infoldings are not septal. Access to the infoldings from the epicardium is through the interatrial groove, recognised by surgeons as Waterston's or Sondergaard's groove (Fig. 5A). Sandwiched between the fold are epicardial tissues, frequently containing the arterial supply to the sinus node. The prominent antero-inferior fold of the fossa, however, separating the flap valve from the triangle of Koch and the mouth of the coronary sinus, is muscular and constitutes an atrial septal component.

2.3. The left atrium

As with the right atrium, the left atrium possesses a venous component, a vestibule and an appendage, and shares the septum (Fig. 5B). In the left atrium, however, there is also a prominent body. The pulmonary venous component, with the venous orifices at each corner, is found posteriorly, and is directly confluent with the body (Fig. 1B, C). Surrounding the mitral orifice is the vestibular component. The atrial appendage is characteristically a small finger-like extension in human hearts (Figs. 1C and 5B). It has crenellations, or lobes [22], that are potential sites for deposition of thrombus [23]. Owing to its tubular shape, its junction with the left atrium is narrow and well-defined (Fig. 5B). In the pig, the left appendage is broader and spade-shaped, but it still has a narrow junction with the rest of the atrium. The pectinate muscles are much less extensive in the left atrium, being confined within the tubular appendage. They form a complicated network of muscular ridges lining the endocardial surface. Since the left atrium lacks a muscular bundle equivalent to the terminal crest, the division between the rough and smooth walls is at the mouth of the appendage. Occasionally, a
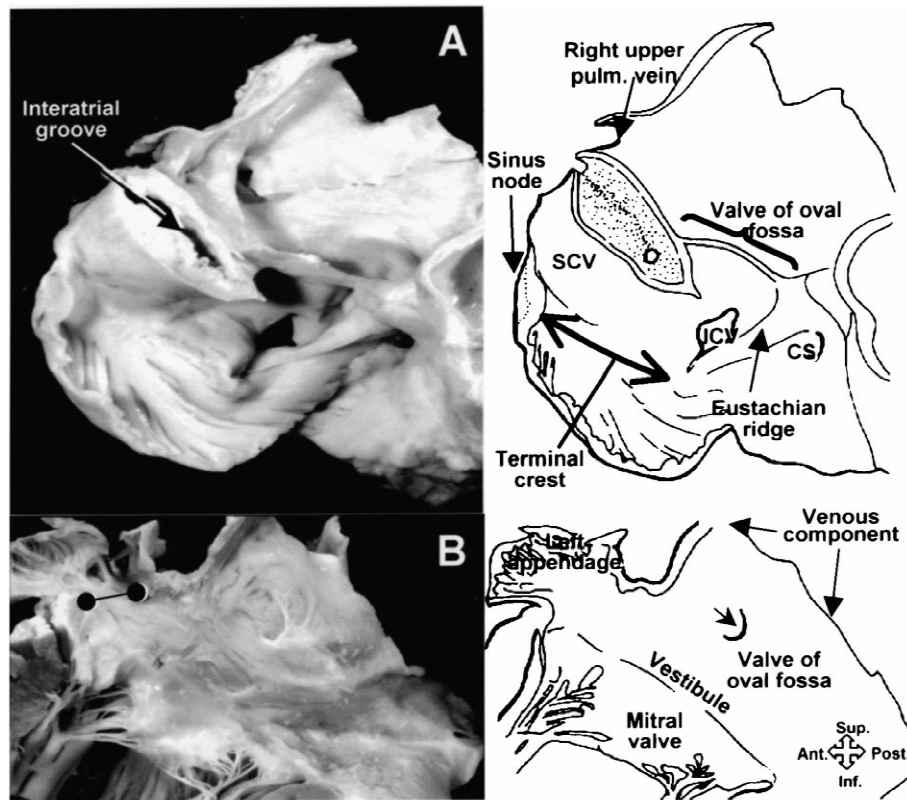
Fig. 5. (A) is a human heart sectioned in four-chamber plane to show the true septum limited to the valve of the oval fossa and its immediate environs. The interatrial groove has been dissected to demonstrate the infolding and the nodal artery (∘ on diagram). Note the subepicardial location of the sinus node. (B) is a view of the left aspect of the septum. The crescent (arrow) is the edge of the valve that often allows a probe or catheter in the right to enter the left atrium. The os to the appendage is narrow (∙-∙). CS, coronary sinus. Other abbreviations as in Fig. 1.

shelf or protrusion on the endocardial surface marks the junction between the mouth of the appendage and the orifice of the left upper pulmonary vein. Thus, the major part of the left atrium, including the septal component, is smooth-walled (Fig. 5B). The smoothest parts are the superior and posterior walls that make up the body, the pulmonary venous component, and the vestibule. The area of the anterior wall just behind the aorta is usually thin, an area noted by McAlpine [24] as vulnerable to being torn. The superior wall, or dome, is thickest, measuring 3.5 to 6.5 mm in formalin-fixed normal specimens [7].

The orifices of the right pulmonary veins are directly adjacent to the plane of the atrial septum (Fig. 5A). The transition between atrium and vein is smooth. Musculature of the atrial wall extends into the veins for various distances (Fig. 6A,B). Close to the venous insertions, the sleeves are thick, and completely surround the epicardial aspect of the veins. The distal margins of the sleeves, however, are usually irregular as the musculature fades out. Earlier studies of the venoatrial junctions have suggested a sphincteric or mechanical role for the myocardial sleeves [25,26]. Independent pulsation has long been recognised in the veins, the first observation being made by Brunton and Fayrer in 1876 [27]. Independent electrical activity has also been known for some time [28,29]. It is

only in recent years, however, that focuses of ectopic activity have been the target of ablative procedures for treatment of paroxysmal atrial fibrillation. It is perhaps no coincidence that the myocardial sleeves are longest in the superior pulmonary veins, corresponding to the highest frequency of ectopic focuses reported in several series [7,25,30,31]. Myocardial sleeves are unlikely to be innocent bystanders, although the substrate for arrhythmogenic activity remains to be identified.

2.4. Sinus node, atrioventricular node and internodal conduction

Located in subepicardial position, and identifiable histologically as a mass of specialised myocardial cells, the sinus node in the adult is approximately 3-mm-thick and 10-mm-long (Fig. 2B,C). Since the bulk of the node is subepicardial, and therefore separated from the right atrial cavity by the thickness of the terminal crest, it may be difficult to modify the sinus node by ablation from within the atrium. At the borders of the node, transitional cells interpose between typical nodal cells and ordinary myocardium. A similar arrangement exists with the atrioventricular node. The compact atrioventricular node receives inputs from histologically discrete transitional cells from the
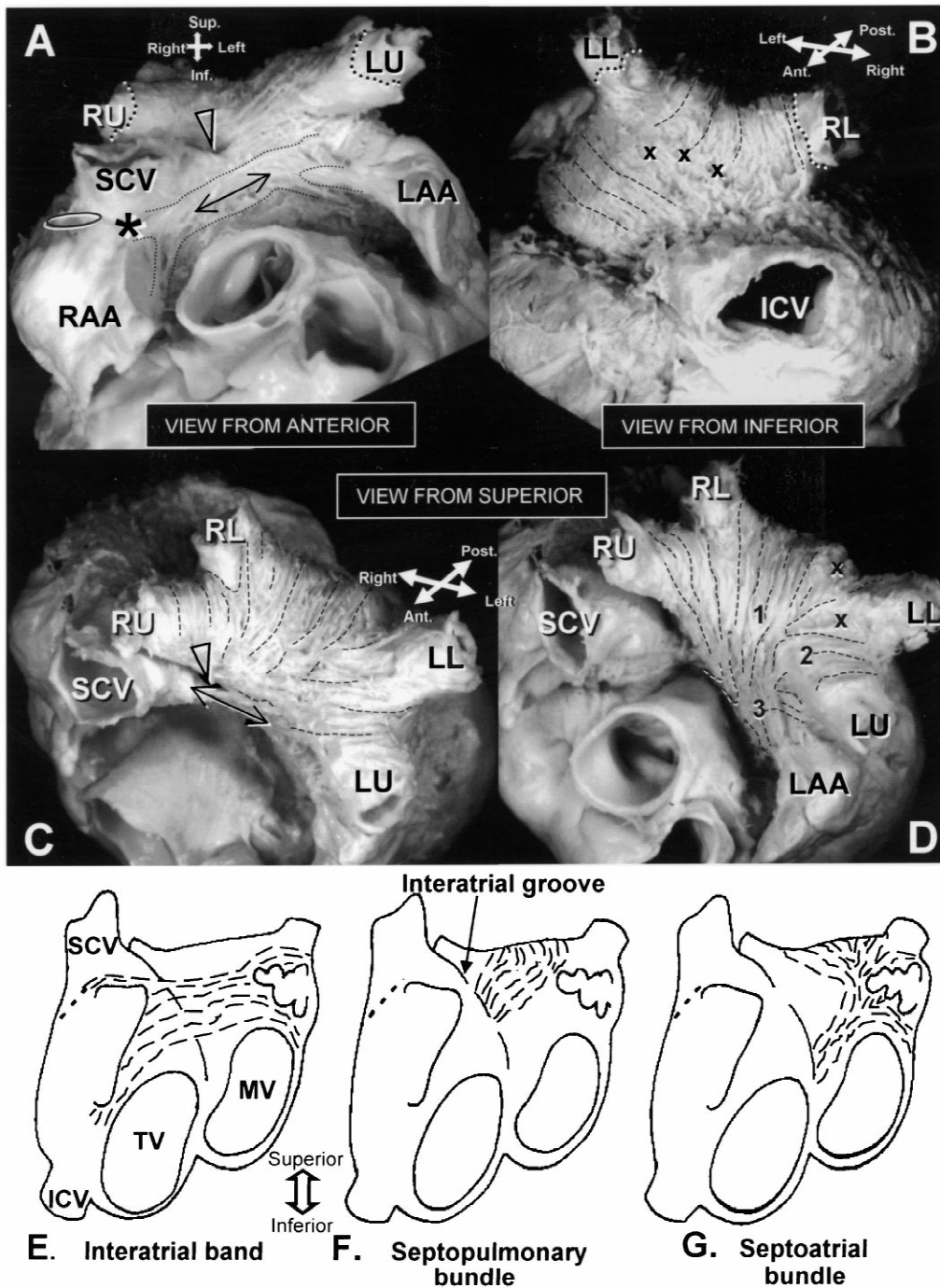
Fig. 6. Serial dissections to display the atrial myoarchitecture in a normal human heart. Broken lines highlight the major orientations. (A) shows the interatrial or Bachmann's bundle (↔ and fine dots) crossing the anterior interatrial groove (triangle) and branching toward the atrial appendages. It combines with the superficial circular fibres passing to the left lateral wall. The location of the sinus node (oval) is superimposed in the terminal groove, the epicardial landmark for the terminal crest. The asterisk denotes the area of the 'concentration point' as described by Lewis and colleagues [34]. Bold dotted lines mark the terminations of myocardial extensions over the venous walls. (B) shows the longitudinal fibres of the septopulmonary bundle in the posterior wall of the left atrium and the superficial circular fibres in the inferior wall. In this heart, a zone of mixed fibres (x) interpose between the major bundles. (C) displays the subepicardial fibres in the dome of the left atrium. The septopulmonary bundle arises from the interatrial groove (triangle) underneath Bachmann's bundle (↔), fanning out to line the pulmonary veins and to pass longitudinally over the dome. (D) is a deeper dissection showing the myoarchitecture in the subendocardium. The interatrial bundle and septopulmonary bundle have been removed to show the septoatrial bundle and its three major fascicles (1, 2, 3). The fibres run obliquely across the dome. Note the areas of mixed fibres (x) between the branches. (E, F and G) are representations of tilted left anterior oblique views showing sequentially the changes in myoarchitecture from subepicardium to subendocardium in the left atrium. Short broken line marks the location of the terminal groove. LAA, left atrial appendage. Other abbreviations as in Fig. 1.

walls of both left and right atriums, as well as from the inferior margin of the atrial septum. In contrast, there are no histologically specialised tracts which run through the atrial musculature to link the cardiac nodes. Instead, spread of the excitation wave from the sinus node is along preferential routes dictated by atrial geometry [32]. The orifices of the veins, and the oval fossa, divide the atrial myocardium into muscular areas, some connected by muscular bundles and others forming narrow zones or isthmuses (Fig. 3). Since propagation in a direction parallel to the length of the myocardial fibre will be faster than in a direction perpendicular to it [33], it is reasonable that internodal conduction is modulated by the arrangement, or myoarchitecture, of the myocardial fibres in the internodal areas.

3. Myoarchitecture

By myoarchitecture, we refer to the gross arrangement of myocardial fibres that make up the walls of the cardiac chambers [34]. 'Fibre' is used in a macroscopic sense, referring to a group of similarly orientated myocytes of sufficient bulk to be seen by the naked eye. Dissections are made on fixed specimens to reveal the fibres. 'Bundle' denotes a collection of fibres with the same general alignment. Changes in orientation through the thickness of the wall may be gradual or abrupt. Major overlapping bundles with different orientations are described as 'layers', without implying physical separation or insulation by fibrous sheaths. Following the precedence of Keith [2], we define two main orientations, using the planes of the atrioventricular junctions as our point of reference. Thus, circular fibres are more or less parallel to the atrioventricular junction, while longitudinal fibres run nearly perpendicularly. Oblique fibres are those inclined between the two major axes.

3.1. The right atrium

Internally, the most prominent muscular bundle in the right atrium is the terminal crest. It extends almost in a C-shape from the anteromedial wall of the right atrium, but its precise origin is unclear (Fig. 2A). Forming a discrete subendocardial ridge, it passes anterior to the orifice of the superior caval vein. It then sweeps rightward and laterally to descend inferiorly toward the orifice of the inferior caval vein, where it ramifies as a series of trabeculations leading into the 'flutter' isthmus (Fig. 4C, D). The close relationship of its proximal course to the entrance of the superior caval vein had caused Keith [2] initially to infer that it was responsible for closing the venous orifice, preventing reflux of blood from the right atrium. He later conceded that its contraction is never adequate completely to occlude the orifice. Nevertheless, it is a distinct bundle in most hearts, showing longitudinally aligned myocardial fibres

(Fig. 4C, D). This arrangement would seem to favour preferential conduction [33,35]. Lewis and his colleagues [36] described a 'concentration point' in their elegant investigation of the origin and propagation of the excitatory process in the canine heart published in 1914. From histological sections, they determined this focus to be close to the 'head' of the sinus node, where muscular bundles radiate 'eager to act as outgoing messengers' to the appendage, body of the atrium, the septum and the 'interauricular band'. Indeed, this junctional point is where the sagittal bundle originates to pass into the tip of the right appendage superiorly (Figs. 2A and 3A). From the same point, the apex of the terminal crest descends inferiorly, while the interatrial bundle, called the interauricular band by Lewis and his colleagues, and also commonly known as Bachmann's bundle, takes its origin from this point before passing leftward into the left atrium (Fig. 6A).

Our recent observations have revealed non-uniform arrangement of fibres in the junctional areas between the terminal crest and pectinate muscles and the adjoining intercaval musculature. The distal portions of the crest add to the variability in topography of the flutter isthmus (Fig. 4C, D) [21,37]. In some hearts, the isthmus is completely muscular, or a large trabeculation from the crest is seen to run posteriorly. In the majority of hearts, the posterior portion is membranous, while the midportion contains branching and interconnecting muscular trabeculations. The anterior portion of the isthmus in all hearts is the smooth wall of the vestibule. It is then the disarray of the trabeculations that has the potential for delaying conduction.

In the lateral wall, the pectinate muscles arise almost perpendicularly from the crest. They run in a posterior–anterior direction as seen in simulated right-anterior oblique projection (Fig. 2A), and extend to insert into the vestibule. Keith [2], followed by Flack [4], emphasised the function of the pectinate muscles in atrial contraction, pulling on the ventricle. Keith counted 15 to 18 pectinate muscles, each of 1 to 2 mm in diameter. In between these trabeculations are finer, criss-crossing, muscles, many of them originating from the pectinate muscles. Antero-superiorly, the tip of the appendage shows a less regular pattern of pectinate muscles. Some of these taking origin from the sagittal bundle (septum spurium) branch like fronds or palm-leaves [38]. Others are arranged in interlacing whorls.

On the endocardial aspect, the vestibule that anchors the pectinate muscles is variously termed the circular musculature of the auricular canal [2], the right anterior crest [5], the annular bundle [6], and the anterior vertical bundle (Figs. 2A, 3A and 4A) [39]. Toward the tricuspid orifice, it thins out to abut the insertion of the valve. On the epicardial aspect only a small part of the vestibule is visible since it extends much further towards the tricuspid valve in the subendocardium. The vestibular muscle is also

arranged circularly, but is continuous with the lower bifurcation from the right extremity of the interatrial band (Fig. 6A, B). When traced inferiorly and toward the septum, the annular bundle loses its prominence as the pectinate muscles diminish. The upper bifurcation of the interatrial bundle sends strands into the subepicardium of the atrial appendage and into the area of the terminal crest (Fig. 6A).

The rim of the oval fossa is prominent in most hearts. The myofibres are arranged circularly around the fossa. The peripheral fibres in the antero-superior rim extend toward the origin of the terminal crest. Similarly, the fibres in the anterior rim combine with fibres from the apex of Koch's triangle and the eustachian ridge [19] in this region corresponding to the 'fast pathway' in atrioventricular nodal re-entrant tachycardia. Fibres in the posterior rim blend into obliquely arranged fibres of the intercaval bundle that cover the epicardial surface of the venous sinus between the interatrial groove and the terminal groove [5,6].

3.2. The left atrium

Seemingly uniform, the smooth walls are composed of one to three, or more, overlapping layers of differently aligned myocardial fibres, with marked regional variations in thickness [5–7,40] (Fig. 6B–F). Without a terminal crest for anchorage, the pectinate muscles tend to be less regularly arranged than in the right appendage. In some hearts, they appear like whorls of fine ridges lining the lumen of the tubular appendage. Although Keith [2] described a left tænia terminalis (terminal crest), an opinion endorsed recently by Victor [38], in our opinion this ridge is no more than an exaggerated fold in the atrial wall. It is seen in only a proportion of human hearts. It corresponds to the upper bifurcation of the interatrial bundle on the epicardial aspect (see below).

Most hearts have a general pattern of arrangement of fibres in the smooth portion of the left atrium, but local variations are frequent. On the epicardial aspect, the most prominent bundle is the interatrial bundle. This buttresses and runs in parallel with the circularly arranged left atrial fibres (Fig. 6C, E). The circular fibres arise from the anterior and antero-superior margin of the atrial septum. They then sweep leftward, where they blend with the leftward extent of the interatrial bundle before bifurcating to encircle the appendage, rejoining in the lateral wall to form a broad band in the inferior wall which then enters the septal raphe (Fig. 6A, B). Deeper than the circular fibres is a layer of initially oblique fibres that arise from the antero-superior septal raphe (Fig. 6C, F). Termed the septopulmonary bundle by Papez [5], this structure sweeps superiorly to become mainly longitudinal, with branches fanning out to pass around the insertions of the pulmonary veins, continuing as the muscular sleeves surrounding the veins. On the posterior wall, the septopulmonary bundle

branches into two oblique fascicles which fuse with the superficial circular bundle. In some hearts, the septopulmonary bundle blends into an area of mixed fibres, without a dominant orientation (Fig. 6B). Deeper still, in the subendocardium, the dominant fibres in the anterior wall arise from a bundle described by Papez [5] as the septoatrial bundle. Ascending obliquely from the anterior septal raphe, this layer soon fans out. It then proceeds as a broad band which combines with the longitudinal fibres of the septopulmonary bundle toward the orifices of the right pulmonary veins (Fig. 6D, G). Another band from the fan turns laterally, combining more superficially with leftward fibres of the septopulmonary bundle toward the orifices of the left pulmonary veins. Thus, there is an abrupt change of fibres, or mixed fibres, in the subendocardium of the posterior wall (Fig. 6D). A third branch is circumferential, passing leftward to surround the mouth of the appendage, and then combining with the circular fibres of the subepicardium in the inferior wall (Fig. 6D, G).

3.3. Interatrial connections

The complexity of the atrial walls is compounded by the presence of interatrial connections at sites other than at the true septum. Muscular continuity between the atriums is frequently found as bridges in the subepicardium. Acquired changes in these bridges, that could abolish or prolong interatrial conduction, may provide a substrate for paroxysmal and chronic atrial fibrillation [41]. The most prominent interatrial bridge is Bachmann's bundle, seen anteriorly (Fig. 6A, C, E). This bundle is by far the largest of the anatomic interatrial communications, and probably accounts for the largest part of interatrial conduction. The broad muscular band crosses the anterior septal raphe. It is not, however, a discrete, cable-like, structure. Most frequently, it is a flat band, or several bundles, of muscular fibres arranged in parallel fashion that blend into the adjoining myocardium. It has bifurcating branches to both the right and left atriums that encircle the atrial appendages. Although it can be distinguished as a discrete bundle at the raphe, it blends in with adjacent musculature elsewhere (Fig. 6C). Its width and prominence varies from heart to heart. The muscular fibres in Bachmann's bundle, as in the terminal crest, are well aligned. Smaller interatrial bundles are frequently seen alongside Bachmann's bundle which cross the anterior interatrial groove [7].

Other bridges are sometimes present posteriorly, joining the left atrium to the intercaval area on the right and to the insertion of the inferior caval vein (Fig. 7A, B) and providing the potential for posterior breakthrough of sinus impulse. Keith and Flack [4] considered them part of the interatrial band that continues with the circular left atrial fibres. Papez [5] drew attention to a rightward extension of the septopulmonary bundle that joins with the intercaval bundle on the posterior wall (Fig. 7A). Inferiorly, further muscular bridges from the left atrial wall often overlie and
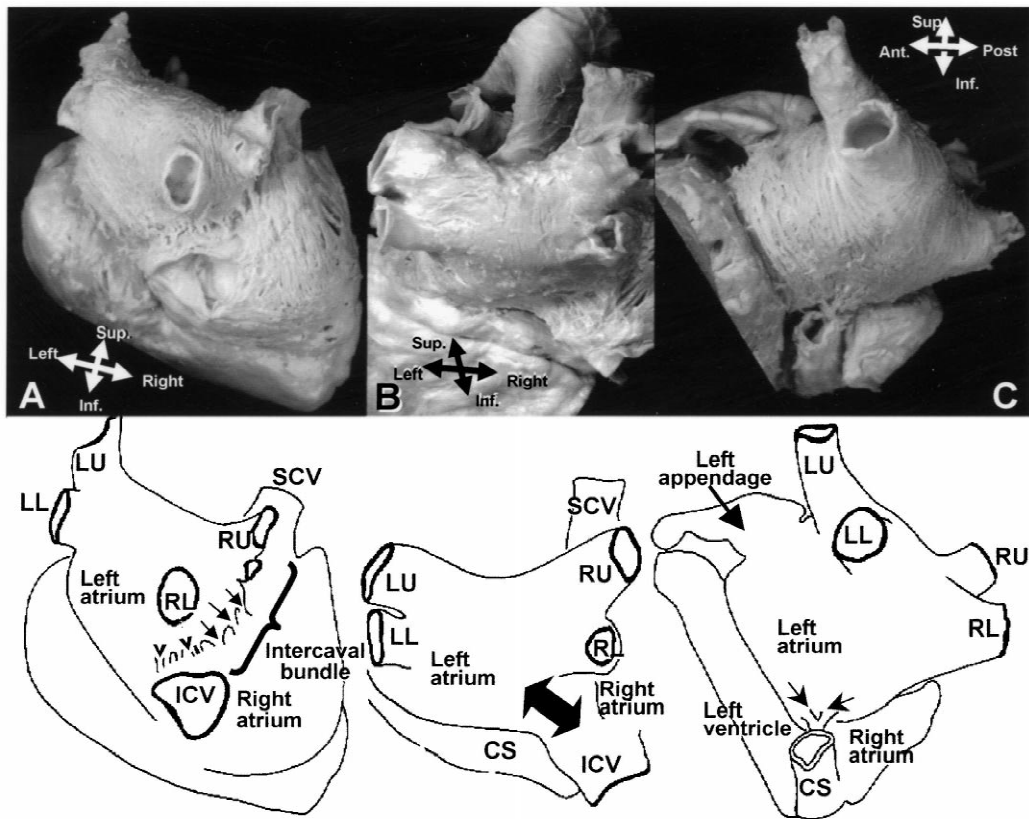
Fig. 7. Interatrial muscular bridges in the subepicardium. (A) shows thin bridges crossing the posterior interatrial groove. There are several thin bridges (arrows) between the interpulmonary bundle and the intercaval bundle. Other small bridges (v) connect the circular fibres of the inferior wall with the entrance of the inferior caval vein. (B) shows the posterior view of a heart with a broad band (double-headed arrow) in the subepicardium connecting the inferior wall of the left atrium to the inferior cavo-atrial junction in the right atrium. (C) is a view from the left showing small muscular bridges (small arrows) between the inferior wall of the left atrium and the coronary sinus (CS). Abbreviations as in Fig. 1.

run into the wall of the coronary sinus, providing further pathways of conduction between the right and left atriums (Fig. 7C) [40,42]. Fine bridges connecting the remnant of the vein of Marshall to left atrial musculature have also been demonstrated [43].

4. Conclusions

The substrates for atrial fibrillation are still unclear. Understanding of normal atrial structures and myoarchitecture will allow hypotheses to be made to generate models of abnormal conduction. Principally, the atrial wall can no longer be conceived as a homogenous monolayer of myocytes. The topography of the endocardial surface of the right atrium is strikingly irregular, dominated by extensive pectinate muscles. The studies of Spach and his colleagues [44,45] highlighted the role of structural discontinuities in wave propagation. Jalife and his colleagues [46,47] further developed this theme in addressing the effects of three-dimensional structural heterogeneity in destabilizing re-entry. With regard to the left atrium, our

studies on normal hearts show its apparently smooth wall to be a complex of overlapping muscular bundles with different orientations, blending into each other in some areas. In other areas, there are abrupt changes in orientation, suggesting discordance between subepicardial and subendocardial conduction. Local variations, and mixed arrangement of fibres, are common. If we are to understand the pathology of atrial arrhythmias, then it is crucial that knowledge of normal variations be taken into account when attempting to interpret findings from abnormal hearts, as and when these become available. For example, from Moe's 'multiple wavelet' hypothesis [48] of fibrillation, based on a model of two-dimensional atrial sheet, elucidation of the pathophysiological mechanisms of atrial fibrillation can further be advanced by consideration of the atrial structure in multi-dimensions. Account should certainly be taken of the heterogeneous transmural and transeptal myoarchitecture when developing computer models [49,50]. It is also crucial to note the marked differences in structure between the human and the mammals frequently used in the experimental laboratory. Finally, if our findings are to prove of value to the

electrophysiologist, then it goes without saying that, like ourselves, clinicians should now describe their own findings using attitudinally correct terminology [51].

Acknowledgements

References

[1] Keith A. Harveian lecture on the functional anatomy of the heart. Br Med J 1918;361–3.

[2] Keith A. An account of the structures concerned in the production of the jugular pulse. J Anat Physiol 1907;42:1–25.

[3] Keith A, Flack M. The form and nature of the muscular connections between the primary divisions of the vertebrate heart. J Anat Physiol 1907;41:172–189.

[4] Flack M. The heart. In: Hill L, editor, Further advances in physiology, London: Edwards Arnold, 1909, pp. 34–71.

[5] Papez JW. Heart musculature of the atria. Am J Anat 1920–21;27:255–277.

[6] Wang K, Ho SY, Gibson D, Anderson RH. Architecture of the atrial musculature in humans. Br Heart J 1995;73:559–565.

[7] Ho SY, Sanchez-Quintana D, Cabrera JA, Anderson RH. Anatomy of the left atrium: implications for radiofrequency ablation of atrial fibrillation. J Cardiovasc Electrophysiol 1999;10:1525–1533.

[8] Ho SY, Anderson RH, Sanchez-Quintana D. Gross structure of the atriums. More than anatomical curiosity? PACE 2002; (in press).

[9] Spach MS, Barr RC, Jewett PH. Spread of excitation from the atrium into the thoracic veins in human beings and dogs. Am J Cardiol 1972;30:844–854.

[10] Chiari H. Ueber Netzbildungen im rechten Vorhofe Herzens. Beitr Path Anat 1897;22:1–2.

[11] Todaro F. Novelle richerche sopra la struttura muscolare delle orechiette del coure umano e sopra la valvola d'Eustachio. Sperimentale 1865;16:217.

[12] Voboril ZB. Todaro's tendon in the heart. 1. Todaro's tendon in the normal human heart. Folio Morph Praha 1967;15:187–196.

[13] Ho SY, Anderson RH. How constant anatomically is the tendon of Todaro as marker for the triangle of Koch? J Cardiovasc Electrophysiol 2000;11:83–89.

[14] James TN. Todaro's tendon and the 'triangle of Koch'. Lessons from eponymous hagiolatry. J Cardiovasc Electrophysiol 1999;10:1478–1496.

[15] McGuire MA. Koch's triangle: useful concept or dangerous mistake? J Cardiovasc Electrophysiol 1999;10:1497–1500.

[16] McAlpine WA. In: Heart and coronary arteries, Berlin: Springer–Verlag, 1975, pp. 160–162.

[17] Sealy WC, Gallagher JJ. The signal approach to the septal area of the heart based on experiences with 45 patients with Kent bundles. J Thorac Cardiovasc Surg 1980;79:542–551.

[18] Sánchez-Quintana D, Ho SY, Cabrera JA, Farré J, Anderson RH. Topographic anatomy of the inferior pyramidal space: relevance to radiofrequency catheter ablation. J Cardiovasc Electrophysiol 2001;12:210–217.

[19] Janse MJ, Anderson RH, McGuire MA, Ho SY. 'AV nodal' reentry. Part 1: 'AV nodal reentry revisited'. J Cardiovasc Electrophysiol 1993;4:561–572.

[20] Sánchez-Quintana D, Davies DW, Ho SY, Oslizlok P, Anderson RH. Architecture of the atrial musculature in and around the triangle of Koch: its potential relevance to atrioventricular nodal reentry. J Cardiovasc Electrophysiol 1997;8:1396–1407.

[21] Hocini M, Loh P, Ho SY, Sánchez-Quintana D, Thibault B, deBakker JMT, Janse MJ. Anisotropic conduction in the triangle of Koch of mammalian hearts: electrophysiologic and anatomic correlations. J Am Coll Cardiol 1998;31:629–636.

[22] Cabrera JA, Sánchez-Quintana D, Ho SY et al. Angiographic anatomy of the inferior right atrial isthmus in patients with and without history of common atrial flutter. Circulation 1999;99:3017–3023.

[23] Veinot JP, Harrity PJ, Gentile F et al. Anatomy of the normal left atrial appendage: a quantitative study of age-related changes in 500 autopsy hearts: implications for echocardiographic examination. Circulation 1997;96:3112–3115.

[24] Ernst G, Stollberger C, Abzieher F et al. Morphology of the left atrial appendage. Anat Rec 1995;242:553–561.

[25] McAlpine WA. In: Heart and coronary arteries, Berlin: Springer–Verlag, 1975, pp. 58–59.

[26] Burch GE, Romney RB. Functional anatomy and 'throttle valve' action of the pulmonary veins. Am Heart J 1954;47:58–66.

[27] Nathan H, Eliakin M. The junction between the left atrium and the pulmonary veins. Circulation 1966;34:412–422.

[28] Brunton TL, Fayrer J. Note on independent pulsation of the pulmonary veins and vena cava. Proc Royal Soc Lond 1876–77;25:174–176.

[29] Zipes DP, Knope RF. Electrical properties of the thoracic veins. Am J Cardiol 1972;29:372–376.

[30] Haïssaguerre M, Jaïs P, Shah DC et al. Spontaneous initiation of atrial fibrillation by ectopic beats origination in the pulmonary veins. New Engl J Med 1998;339:659–665.

[31] Saito T, Waki K, Becker AE. Left atrial myocardial extension onto pulmonary veins in humans: anatomic observations relevant for atrial arrhythmias. J Cardiovasc Electrophysiol 2000;11:888–894.

[32] Janse MJ, Anderson RH. Specialized internodal atrial pathways — fact or fiction? Eur J Cardiol 1974;2:117–136.

[33] Roberts DE, Hersh LT, Scher AM. Influence of cardiac fibre orientation on wave front voltage, conduction velocity and tissue resistivity in the dog. Circ Res 1979;44:701–712.

[34] MacCallum JB. On the muscular architecture and growth of the ventricles of the heart. Johns Hopkins Hosp Rep 1900;9:307–335.

[35] Spach MS, Dolber PC, Heidlage JF et al. Influence of the passive anisotropic properties on directional differences in propagation following modification of sodium conductance in human atrial muscle: a model of reentry based on anisotropic discontinuous propagation. Circ Res 1988;62:811–832.

[36] Lewis T, Meakin J, White PD. The excitatory process in the dog's heart. Part I. The auricles. Philos Trans R Soc Lond B Biol Sci 1914;205:375–420.

[37] Waki K, Saito T, Becker AE. Right atrial flutter isthmus revisited: normal anatomy favours nonuniform anisotropic conduction. J Cardiovasc Electrophysiol 2000;11:90–94.

[38] Victor S, Nayak VM. Aneurysm of the left atrial appendage. Tex Heart Inst J 2001;28:111–118.

[39] McAlpine WA. In: Heart and coronary arteries, Berlin: Springer–Verlag, 1975, p. 88.

[40] Ho SY, Sánchez-Quintana D. Structure of the left atrium. Eur Heart J Suppl 2000;2(Suppl K):K4–K8.

[41] Cosio FG, Palacios J, Vidal JM et al. Electrophysiological studies in atrial fibrillation. Slow conduction of premature impulses: a possible manifestation of the background for reentry. Am J Cardiol 1983;51:122–130.

[42] Antz M, Otomo K, Arruda M et al. Electrical conduction between

the right atrium and the left atrium via the musculature of the coronary sinus. Circulation 1998;98:1790–1795.

[43] Kim DT, Lai AC, Hwang C et al. The ligament of Marshall: a structural analysis in humans hearts with implications for atrial arrhythmias. J Am Coll Cardiol 2000;36:1324–1327.

[44] Spach MS, Miller III WT, Dolber PC et al. The functional role of structural complexities in the propagation of depolarization in the atrium of the dog: cardiac conduction disturbances due to discontinuities of effective axial resistivity. Circ Res 1982;50:175–191.

[45] Spach MS, Dolber PC, Anderson PAW. Multiple regional differences in cellular properties that regulate repolarization and contraction in the right atrium of adult and newborn dogs. Circ Res 1989;65:1594–1611.

[46] Gray RA, Pertsov AM, Jalife J. Incomplete reentry and epicardial breakthrough patterns during atrial fibrillation in the sheep heart. Circulation 1996;94:2649–2661.

[47] Jalife J, Gray RA. Insights into the mechanisms of atrial fibrillation: role of the multidimensional atrial structure. In: Murgatroyd FD, Camm AJ, editors, Non-pharmacologic management of atrial fibrillation, Armonk: Futura Publishing, 1997, pp. 357–377.

[48] Moe GK, Rheinboldt WC, Abildskov JA. A computer model of atrial fibrillation. Am Heart J 1964;67:200–220.

[49] Harrild DM, Henriquez CS. A computer model of normal conduction in the human atria. Circ Res 2000;87:e25.

[50] Zemlin CW, Herzel H, Ho SY, Panfilov AV. A realistic and efficient model of excitation propagation in the human atria. In: Virag N, Blanc O, Kappenberger; L, editors, Computer simulation and experimental assessment of cardiac electrophysiology, New York: Futura Publishing, 2001, pp. 29–34.

[51] Cosio FC, Anderson RH, Kuck K, Becker A et al. Living anatomy of the atrioventricular junctions. A guide to electrophysiological mapping. A consensus statement from the Cardiac Nomenclature Group of Arrhythmias, European Society of Cardiology, and the Task Force on Cardiac Nomenclature from NASPE. Circulation 1999;100:e31–e37.