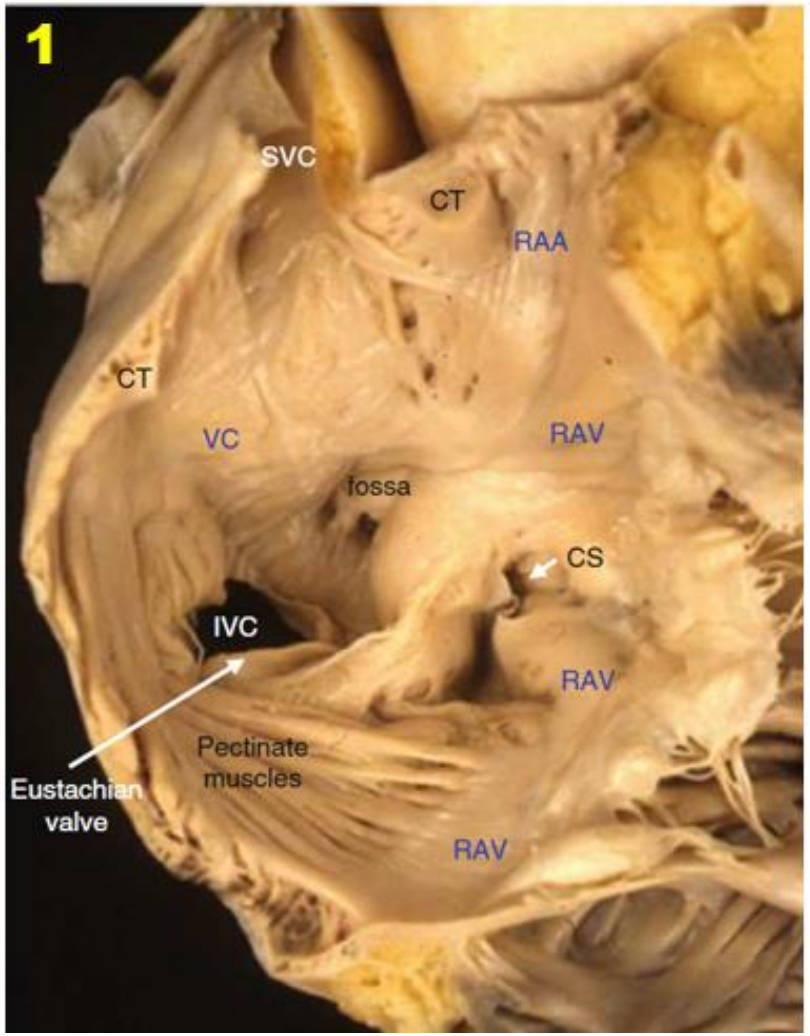
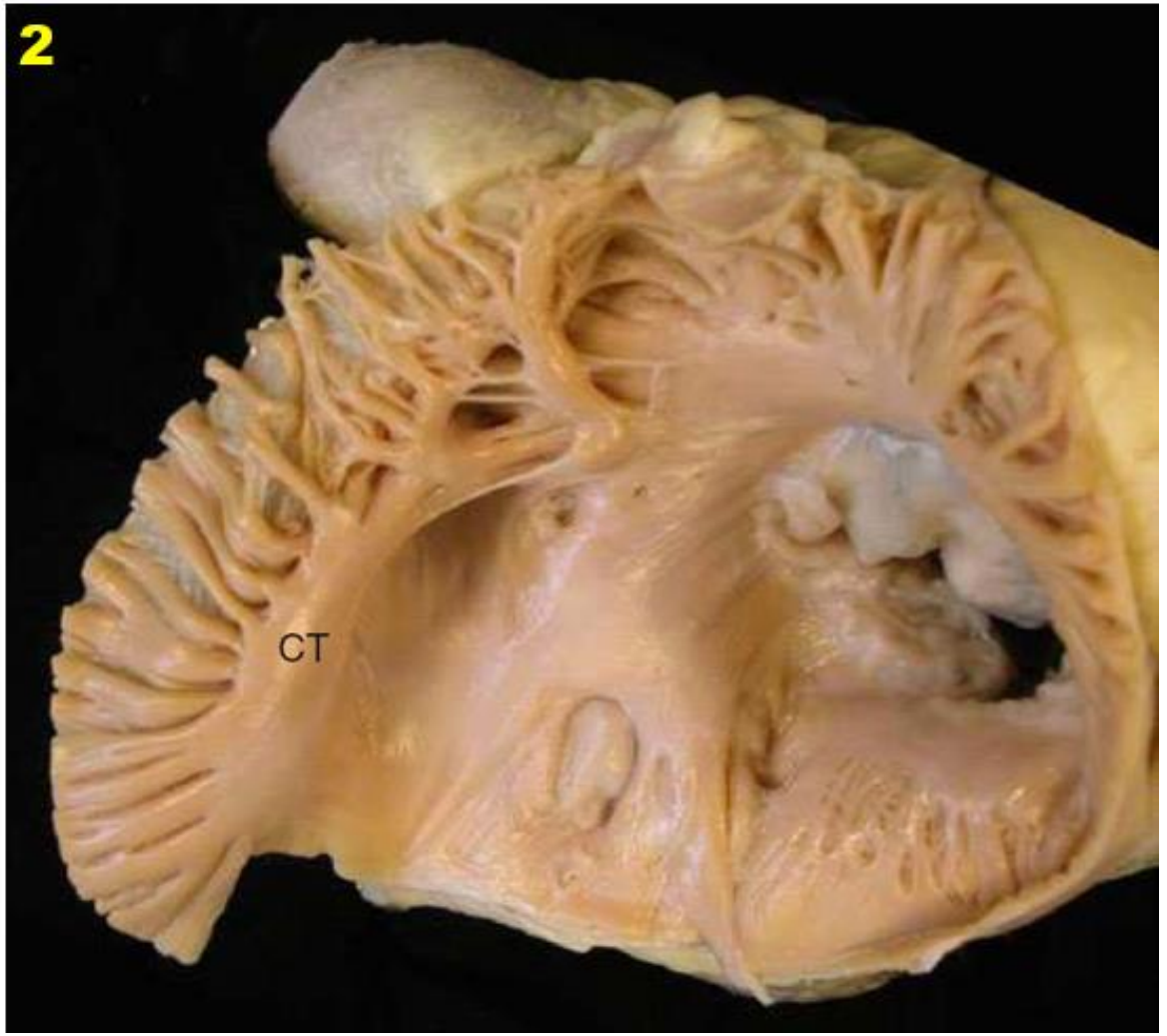
Anatomical and electrophysiological notions of supraventricular structures for clinical and electrophysiologists

The Right Atrium (RA) lies anterior, inferior and rightward from the LA and is composed of the venous component, appendage and vestibule. The anatomy of the RA is shown in **Fig. 1 and fig 2**.





Figures 1 and 2 Anatomy of the RA (**Figure 1**) and the crista terminalis (**Figure 2**). The RA is anteroinferior to the LA and is composed of the venous component *VC*), RA appendage (*RAA*) and RA vestibule (*RAV*). The superior (*SVC*) and inferior vena cava (*IVC*) are seen connected posteriorly into the RAV. The RA appendage is an anterior structure composed of pectinate muscles originating from the crista terminalis (*CT*). The coronary sinus (*CS*) is inferior and posterior. Superior and slightly posterior on the interatrial septum is the fossa ovalis. In **Figure 2** the crista terminalis is seen with pectinate muscles radiating out

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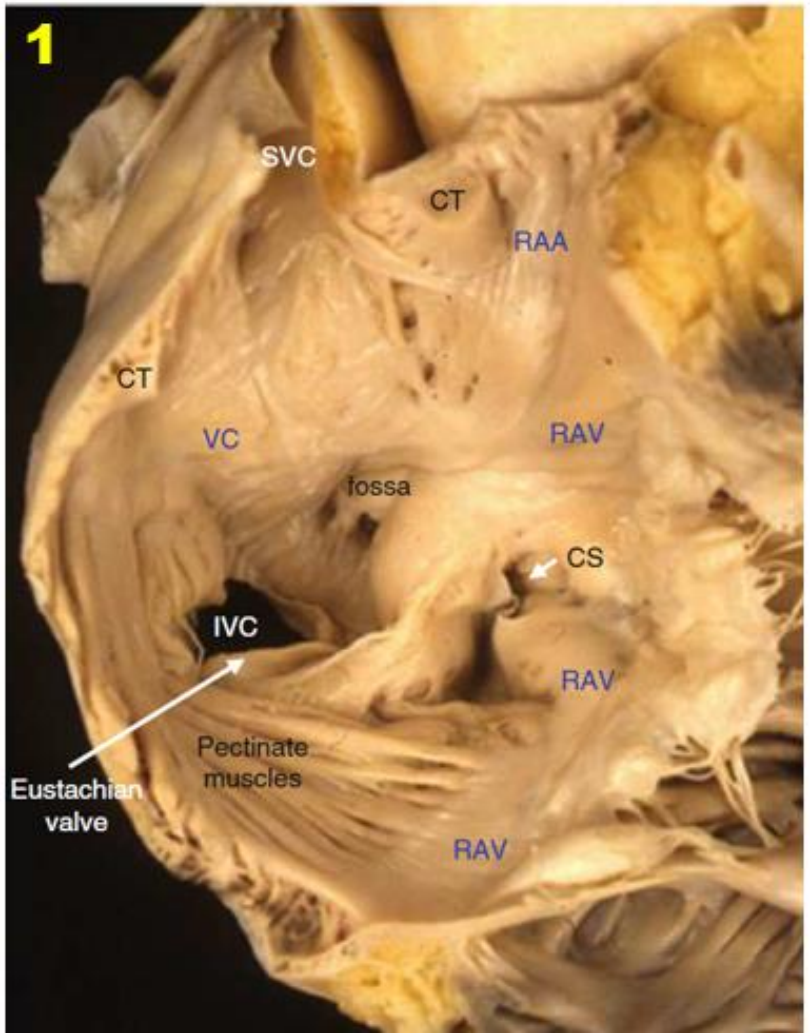
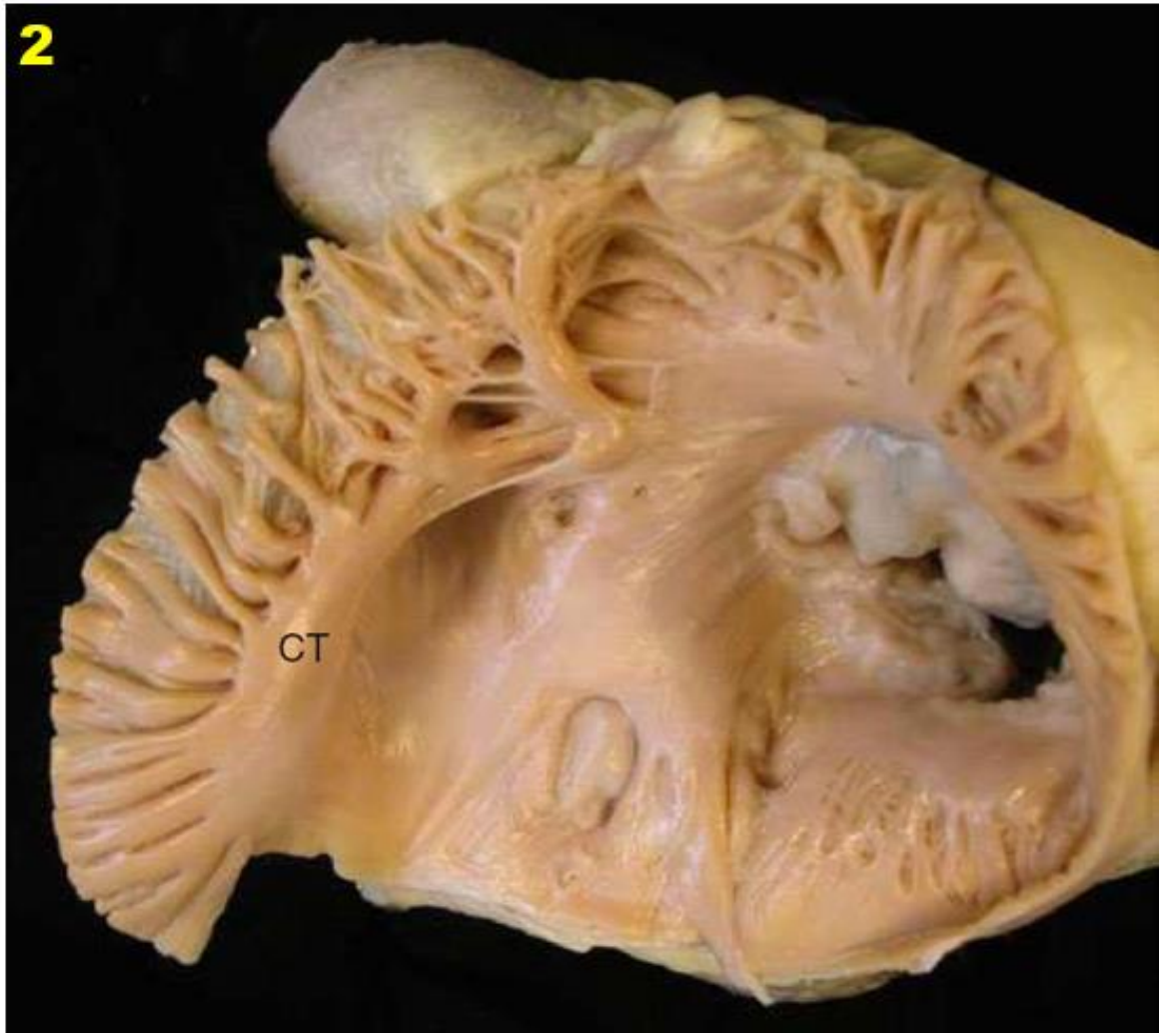




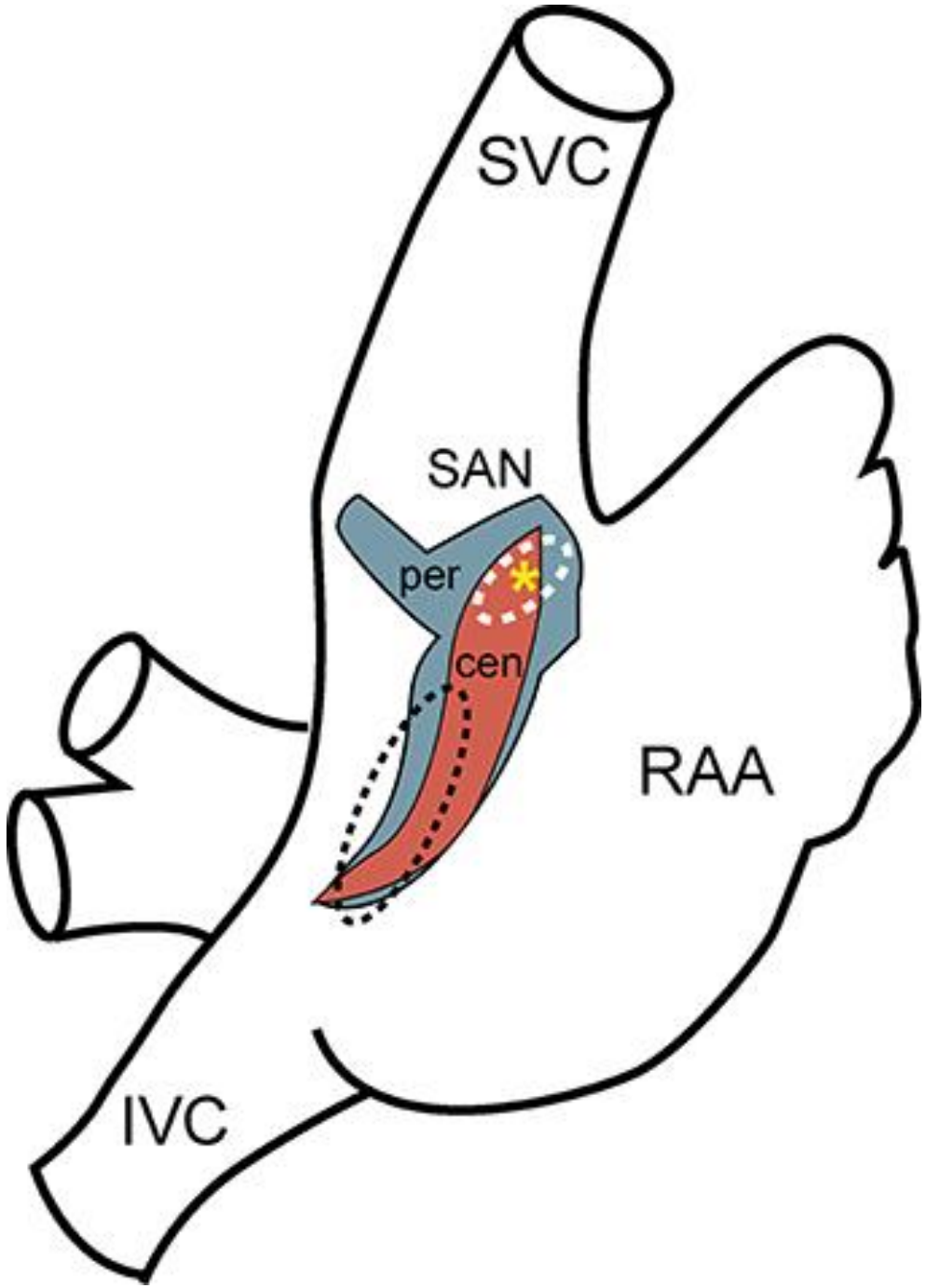
Figures 1 and 2 Anatomy of the RA (**Figure 1**) and the crista terminalis (**Figure 2**). The RA is anteroinferior to the LA and is composed of the venous component *VC*), RA appendage (*RAA*) and RA vestibule (*RAV*). The superior (*SVC*) and inferior vena cava (*IVC*) are seen connected posteriorly into the RAV. The RA appendage is an anterior structure composed of pectinate muscles originating from the crista terminalis (*CT*). The coronary sinus (*CS*) is inferior and posterior. Superior and slightly posterior on the interatrial septum is the fossa ovalis. In **Figure 2** the crista terminalis is seen with pectinate muscles radiating out

The superior and inferior vena cava drain systemic blood into the smooth-walled posterior venous component of the RA. Coronary blood flows through the CS into the RA through the CS os which is located between the **inferior vena cava (IVC)** and the **tricuspid annulus (TA).** The CS os is to a variable degree covered by the **Thebesian valve** which is a thin crescent shaped structure attached at the posterior and inferior boundary of the coronary sinus os. The degree of coverage varies but may practically occlude the CS os in up to 25 % of individuals [4]. The RA appendage is a triangular broad based structure composed of pectinate muscles originating from the **crista terminalis** and is generally where atrial pacemaker leads are positioned for stability. The **crista terminalis** is one of the most common regions responsible for focal atrial tachycardia's as well as acting as a functional electrical barrier essential for **typical atrial flutter**. It separates the pectinated appendage from the venous component (or intercaval area) of the atrial body. The latter is posterior whereas the vestibule which surrounds the atrial outlet leading to the tricuspid valve is anterior.

The Sinus Node (SN)

The SN is a collection of nodal cells within a tough matrix of connective tissue lying just below the epicardium and separated from the endocardium by a layer of atrial myocardium. It is located at the junction between the superior vena cava and the RA, at the anterolateral quadrant marked by the crista terminalis (CT) (**Figure 3**) and statistically measures 10–20 mm in length, 3 mm in width and 1 mm in depth but there is an enormous anatomical variation between individuals [5]. Cells in the SN tend to be much smaller than those in the RA measuring approximately 5–10 μm. Typical nodal cells known as P cells are located in the centre of the SN and are generally poorly organized myofilaments. There are fibroblasts and collagen fibers interspersed throughout the SN. There is a gradual transition between the SN and the RA with a disparity in conduction velocity between the cells thus preventing SN depolarization as a result of atrial depolarization [6].

SA Figure 3



Spontaneous phase IV diastolic depolarization starts at −65 mV until the activation threshold is reached at −40 mV resulting in rapid depolarization. The action potential of the SN differs from that of a Purkinje myocyte with a more gradual upslope and the absence of a plateau phase. Diastolic depolorization occurs as a result of activation of the If current. This operates in a voltage range more negative than normally occurs in the central pacemaker cells (less than −45 mV). It therefore has maximum activity during hyperpolarization and progressively increases opposing repolarization and then initiating diastolic depolarization [7]. The rate of diastolic depolarization in the SN is affected by both sympathetic adrenergic and parasympathetic muscarinic stimulation. This is predominantly affected by the I_f channel. Sympathetic adrenergic stimulation results in an increase in the gradient and duration of diastolic depolarization with minimal effects on the overall action potential duration [8]. This occurs as a result of a shift in the activation curve to more positive voltages without a change in the conductance of the If channel as a result of an increase in intracellular cAMP [9]. The reverse occurs with parasympathetic muscarinic stimulation [10]. Slow inward Ca^{2+} channels are involved in the later phase of diastolic depolarization [8] as well as the upslope in the action potential. The transient T type Ca^{2+} channel is activated at more negative voltages and therefore opens fi rst followed by the long lasting L component which opens during the

upslope of the action potential [8]. The delayed rectifier Ik channel is the predominant potassium channel in the SN and contributes to repolarization allowing the following depolarization to be initiated.

Respiratory sinus arrhythmia occurs as a result of a reduction in the PP interval with inspiration and a prolongation of the PP interval with expiration. The maximum difference between the longest PP and shortest PP interval should be < 160 ms. This phenomenon reduces with age.

Ventriculophasic sinus arrhythmia is seen in association with third degree AV block in which the PP interval surrounding a QRS complex is shorter than the PP interval not surrounding a QRS complex.

SN dysfunction encompasses sinus bradycardia, sinus pause, sinoatrial exit block, chronotropic incompetence and inappropriate sinus tachycardia.

Sinus bradycardia is a relatively common finding and in the absence of symptoms is generally of no clinical signifi cance. A sinus pause is defi ned as the absence of a P wave for greater than or equal to 2 s (although generally not considered clinically significant unless greater than or equal to 3 s while awake or 5 s while asleep). If the duration of the sinus pause is a multiple of the PP interval, then sinoatrial node exit block should be considered.

Chronotropic incompetence is defined as failure to achieve 70–80 % of maximal predicted heart rate (maximal predicted heart rate = 220-age) during peak exercise.

Inappropriate sinus tachycardia is a persistent elevation in heart rate greater than 100 bpm at rest with no obvious precipitating cause. There is an exaggerated increase in sinus rate with minimal activity and a reduction or normalization of sinus rate during sleep. The P wave morphology and axis are unchanged.

It is important to rule out all potential causes as well as other arrhythmias such as right atrial tachycardia close to the SN or SN re-entry tachycardia. Inappropriate sinus tachycardia is most likely multifactorial with a change in the overall autonomic supply to the SN which may include a reduction in the sensitivity to anticholinergic effects or an increased sensitivity to adrenergic activity [11]. Pharmacological options for inappropriate sinus tachycardia include beta adrenergic blockers, nondihydropyridine calcium channel blockers and the selective I_f channel inhibitor ivabradine. Given the selective nature of ivabradine this has been shown to have a useful role in patients with symptomatic inappropriate sinus tachycardia unresponsive to beta adrenergic blockers and calcium channel blockers [12]. Further data is awaited whether this may be considered as fi rst line treatment in this condition. Catheter ablation for SN modifi cation is an alternative strategy in select cases of inappropriate sinus tachycardia. The SN is often diffi cult

to modify from the RA endocardium as there are multiple connections with sites of early activation between the SN and the RA. Additionally the bulk of the SN is subepicardial, has a significant amount of connective tissue, is often covered by thick muscle of the CT and there is a signifi cant cooling effect from the SN artery. The usual area to target is the superomedial aspect of the CT targeting areas of local activation 15–60 msec ahead of the surface p wave looking for a reduction in the sinus rate to less than 90 bpm and a 20–25 % reduction in the maximum sinus rate with isoprenaline [13]. Although acute results are good long term maintenance is less successful [14]. High output pacing should be performed at sites being considered for ablation to avoid phrenic nerve injury. The need for a permanent pacemaker is unusual but a potential complication of this procedure.

Crista Terminalis (CT)

As shown in Fig. 2 this is a C-shaped structure which begins septally at the superior aspect becoming more anterior as it traverses the connection with the SVC and then moves posterior and inferior along the lateral wall of the RA towards the junction with the IVC [15]. The pectinate muscles which form the right atrial appendage span out from the CT. Approximately two thirds of focal atrial arrhythmias occur along this structure [16].

RA Conduction

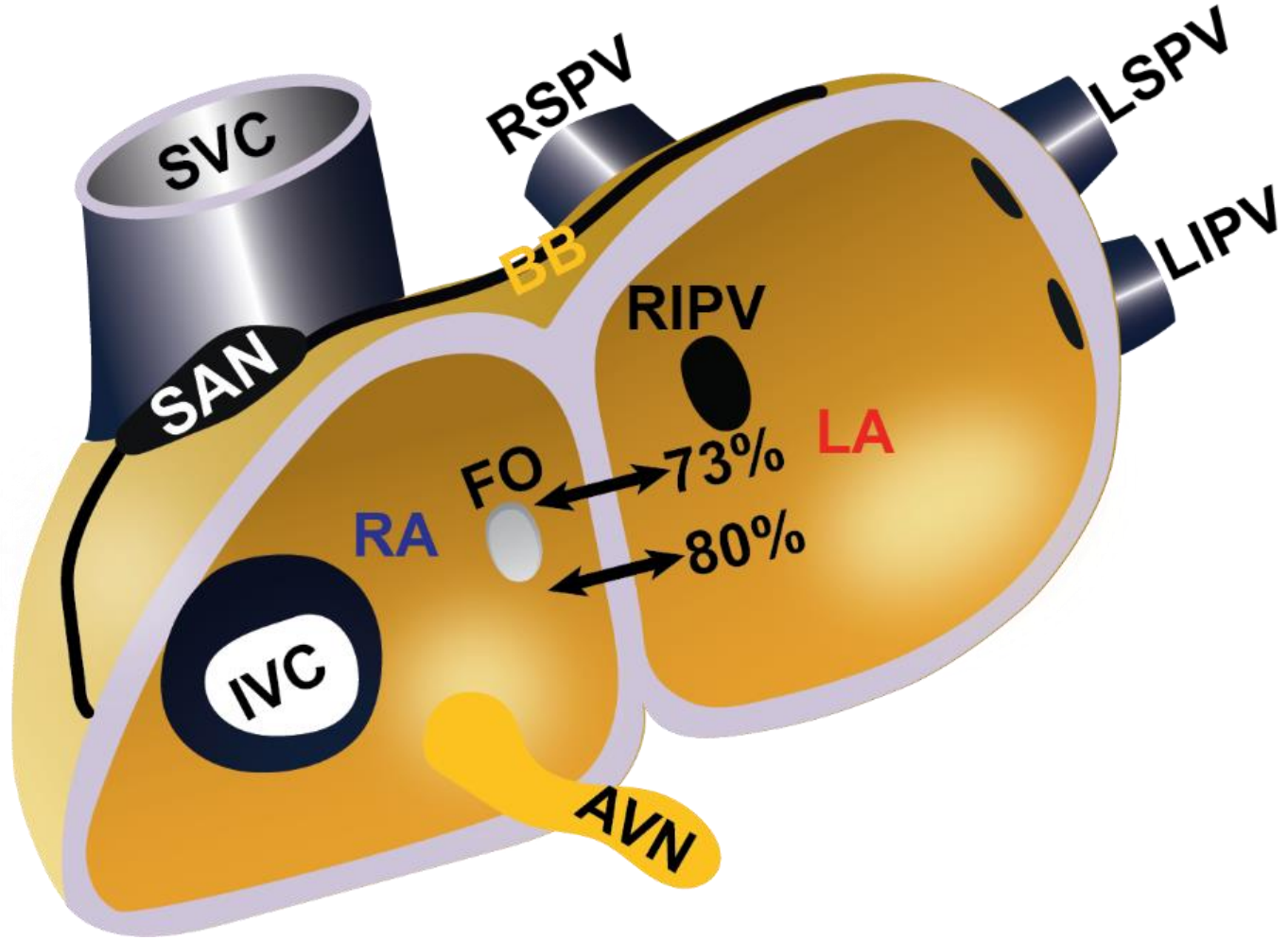
Following discharge from the SN, conduction occurs through the RA using the muscular architecture of the atrial wall that comprises muscle bundles with well aligned working myocytes that preferentially carry the sinus impulse [17]. The notion of three specific internodal tracts is controversial because histologically specialized tissue tracts akin to the insulated ventricular conduction bundles have never been demonstrated anatomically.

Muscular connections between the right and left atrial Bachmann's bundle (BB)

BB is also known as the anterior bundle is responsible for right to left atrial conduction. It is not a discrete bundle nor is it insulated with a fibrous sheath. Instead, it is a muscle bundle with well aligned myocytes, superficially located across the anterior interatrial groove. Its rightward extension reaches superiorly to the area of the sinus node and inferiorly toward the right atial vestibule. Usually it is the most prominent interatrial bundle [18]. The obvious muscular connections between the atriums, important for conduction between them, are the margins of the oval foramen. This structure normally closes at birth, when the lungs become functional, the pulmonary vascular pressure decreases and the LA pressure exceeds that of the RA. These are no more than infoldings of the walls. The floor of the oval fossa, however, although itself a

septal structure, is usually a fibro-collagenous wall in the postnatal heart, so this area does not provide electrical interatrial continuity. The most important muscular interatrial bridge is provided by the insertion of the terminal crest (crista terminalis) into the atrial roof anterior to the mouth of the SVC. When seen externally, this insertion is directly continuous with the anterior interatrial groove (**Figure 1**). The myocytes forming the atrial wall are aggregated together in parallel fashion at this point, and continue into the LA wall as Bachman's bundle (Bachmann 1916). In addition to the bundle itself, there are often robust connections through the inferior part of the anterior interatrial groove (Ho, Anderson et al. 2002), whereas further muscular bridges between the walls of the coronary sinus and LA provide for inferior and leftward communications (Chauvin, Shah et al. 2000). It is the proximity of Bachman's bundle to the terminal crest, and the site of the SAN, however, that makes this the most significant electrical interatrial connection (Anderson and Cook 2007).

Figure 4

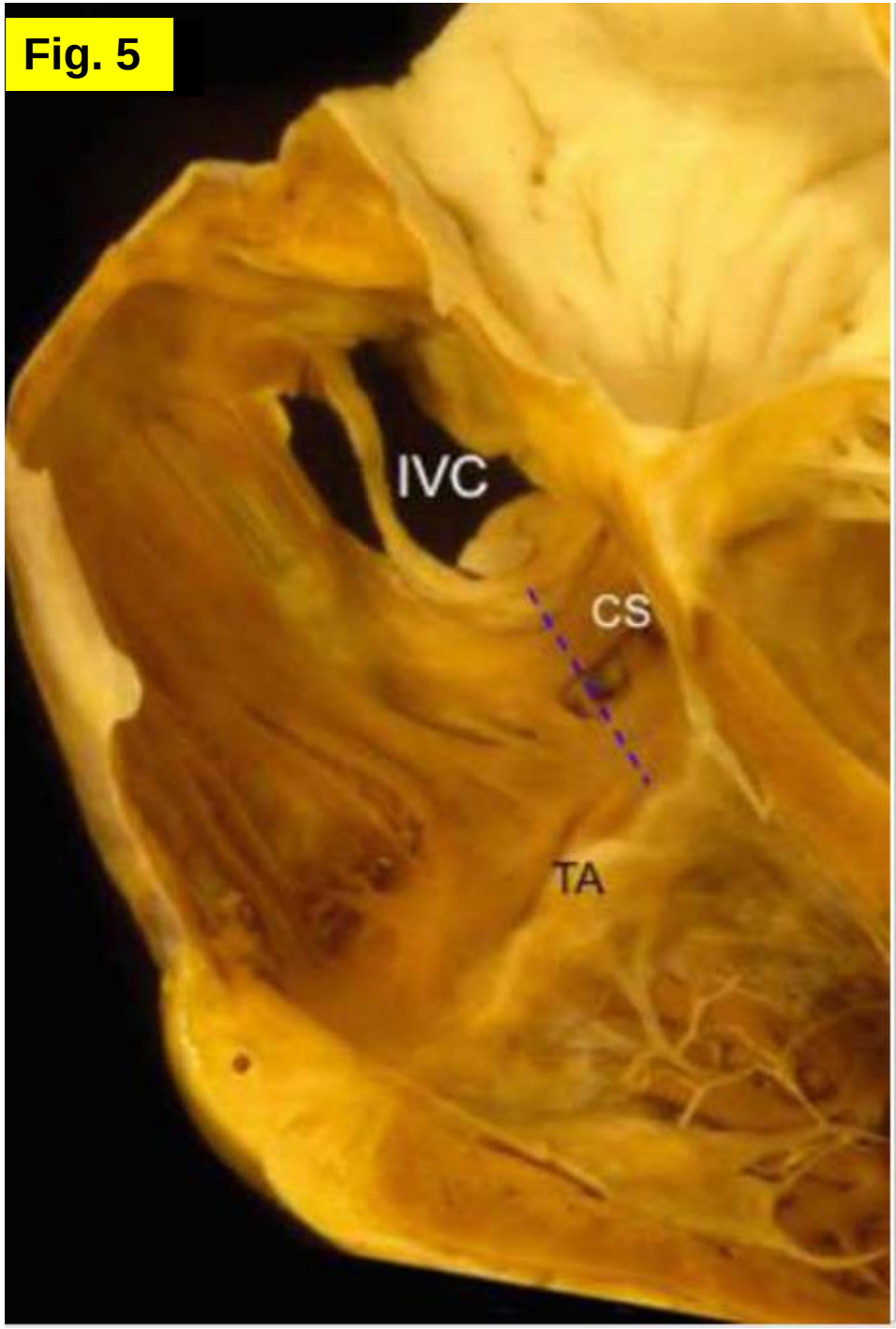


The drawing shows how Bachmann's bundle is the direct continuation of the insertion of the terminal crest into the anterior interatrial groove. It also shows the frequency of other communication through the groove as found in the study of Ho et al (Ho, Anderson et al. 2002). AVN: atrioventricular node; BB: Bachmann's bundle; FO: fossa ovalis; LA: left atrium; LIPV: left inferior pulmonary vein; LSPV: left superior pulmonary vein; RA: right atrium; RIPV: right inferior pulmonary vein; RSPV: right superior pulmonary vein; SAN: sinoatrial node.

Cavotricuspid Isthmus (CTI)

This is the region of slow conduction in typical atrial flutter bounded anteriorly by the septal component of the TA and posteriorly by the Eustachian valve (EV) and the IVC (**Fig. 5**). Conduction in this region is slow due to the criss-cross arrangement of the myocytes and distal ramifications of the crista terminalis relative to the better aligned and circumferential arrangement of myocytes in the vestibule leading to the tricuspid valve (TV) [19]. In an LAO projection the ablation catheter is generally positioned in the mid isthmus in the 6 o'clock position. In the RAO position the catheter is moved from the right to the left keeping the catheter inferiorly. The EV separates the vestibular inferior RA from the IVC. The Eustachian ridge is an elevated region of tissue between the fossa ovalis and the coronary sinus in continuation withthe insertion point of the EV. The Tendon of Todaro (TT) runs in this rim towards the AV node [18].

Fig. 5 Anatomy of the cavo-tricuspid isthmus (CTI). The CTI runs between the tricuspid annulus (TA) and the inferior vena cava (IVC). The dotted line shows a septal approach close to the coronary sinus (CS) which may be used as a line for catheter ablation in order to transect the isthmus.



The left atrium ***LA***

The LA is posterior, superior and to the left of the RA. The tip of the left atrial appendage (LAA) contributes to the left side of the cardiac silhouette in a PA image. The LA is a smoother structure with the muscular appendage confi ned to a small tube-like structure arising from the superior and left side of the chamber. As shown in **Fig. 4** the four pulmonary veins (PV's) drain into the posterior quadrants of the smooth-walled area, which is actually the most posterior region of the heart. The left sided pulmonary veins are best seen in the LAO projection and are posterior to the left atrial appendage. The right pulmonary veins are best visualized inan RAO projection. The right superior pulmonary vein is posterior to the junction between the right atrium and superior vena cava. The LA myocardial fi bers extend over variable distances into the pulmonaryveins. These connections are generally the targets for pulmonary vein isolation. The LA wall is generally a thin structure and therefore care must be taken when manipulating catheters in this region. The lateral wall is approximately 3.9 ± 0.7 mm in thickness, the posterior wall 4.1 ± 0.7 mm, anterior wall 3.3 ± 1.2 mm and the roof 4.5 ± 0.6 mm when measured on cadaver heart specimens [19].

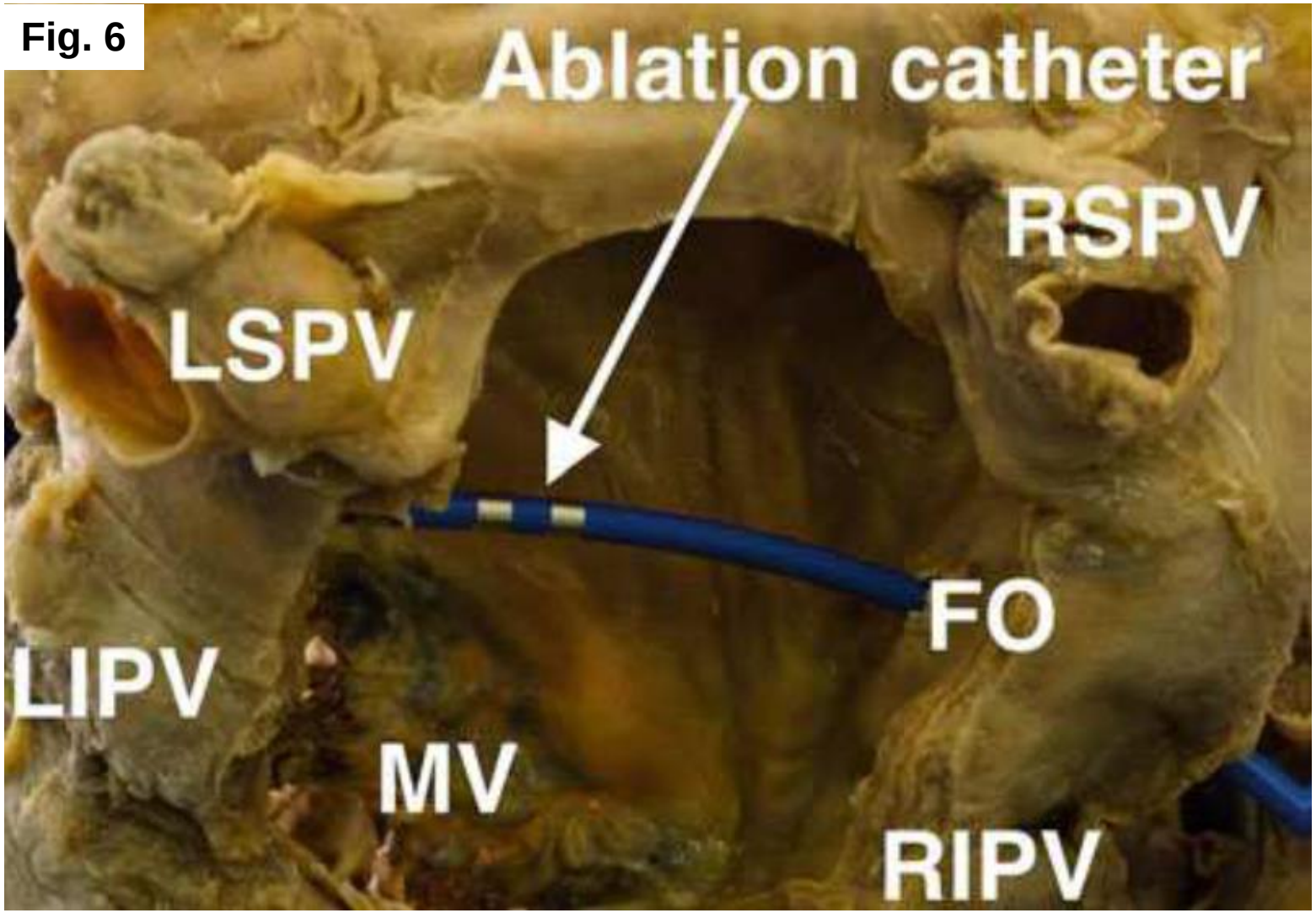


Fig. 6: Anatomy of the LA. This is seen from a posterior view. An ablation catheter is positioned via the intra-atrial septum at the location of the fossa ovalis (FO). The left superior pulmonary vein (LSPV), left inferior pulmonary vein (LIPV), right superior pulmonary vein (RSPV) and right inferior pulmonary vein (RIPV) are seen as posterior structures in the LA. The mitral annulus (MV) is seen inferior to the LIPV

The LA is anterior to the esophagus. This is of real importance in terms of posterior wall ablation. The esophagus has a variable course in relation to the LA. There is also a variability in the thickness of the fibrofatty tissue between the LA and the esophagus. In clinical practice this generally results in the application of lower power (25–30 W) and shorter duration lesion in the posterior left atrial wall in order to attempt to minimize the possibility of esophageal injury.

Anterior to the LA is the ascending aorta which is an important consideration when performing transseptal access.

Atrio-Ventricular (AV) Junction

The compact AV node is the atrial component of the specialized AV junctional area and is located between the coronary sinus os and the septal leafl et of the tricuspid valve. It therefore lies inside the **triangle of Koch** . It measures approximately 5 mm in length and is histologically quite complex. It is not insulated by connective tissue and therefore may potentially be damaged by RF application. The inferior extensions of the compact AV node also run within the triangle of Koch with the rightward extension (fast pathway) parallel to the tricuspid valve and the leftward extension (slow pathway) towards the coronary sinus. These

extensions pass either side of the AV nodal artery and also are not protected structures. The histological appearances of these extensions are the same as the compact AV node and are involved in the AVNRT circuit [20]. The boundaries of the triangle of Koch are shown superimposed on an anatomic specimen in **Fig. 7**.

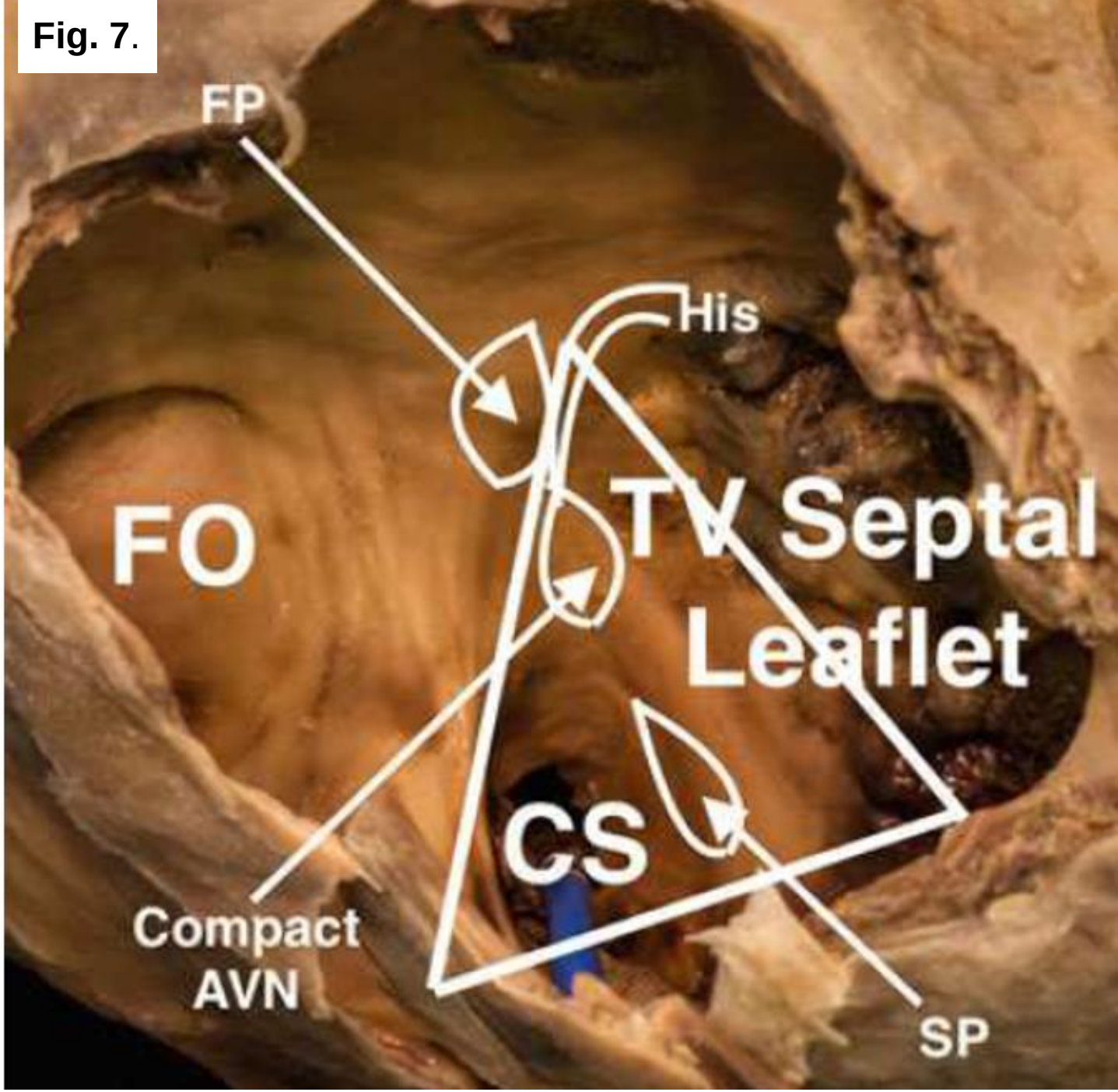
Fig. 7 

Fig. 7 Boundaries of the triangle of Koch superimposed on ananatomic specimen. This is formed by the coronary sinus (*CS*), septal leafl et of the tricuspid valve (*TV*) inferiorly and the tendon of Todaro anterosuperiorly. A catheter is positioned via the inferior vena cava (*IVC*) into the coronarysinus (*CS*). Also shown is the fossa ovalis, approximate location of the slow pathway (*SP*),compact AV node, fast pathway (*FP*) and His.

His Bundle

The His bundle is a continuation of the compact AV node and with similar specialized cells although these are more parallel aligned [21]. It is better insulated than the AV node and therefore is not as easily damaged with RF, although this is still possible. The proximal bundle runs from the distal AV node into the fibrous tissue of the central body where it is termed the penetrating portion. Following this it emerges on the ventricular side of the fibrous body, sandwiched between the membranous septum and the muscular ventricular septum, Taking an initial course usually to the left side of the septum, it then bifurcates into the right bundle (RB) and left bundle (LB) branches, still insulated by fibrous tissue sheaths. The RB tends to have a more anterior origin in the membranous septum.

The CS

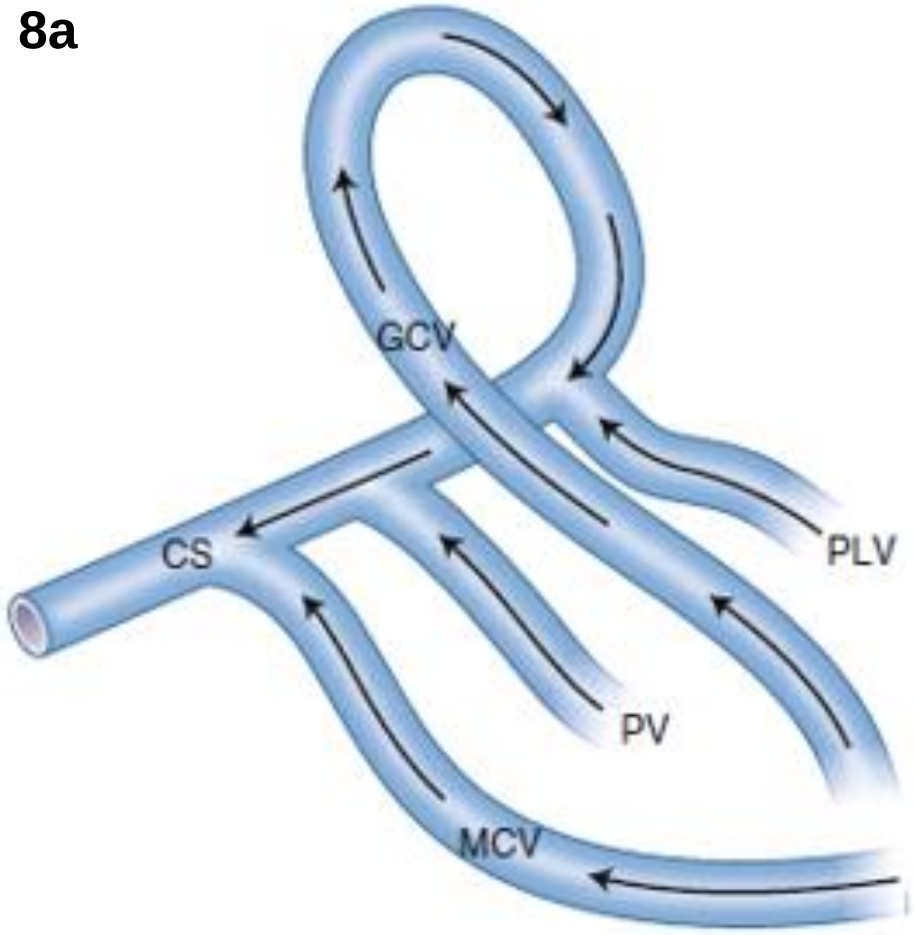
The CS is a tubular shaped structure which runs from either the Valve of Vieussens or in its absence the entrance of the vein/ligament of Marshall to the CS os where it enters the RA. It is approximately 7 cm in length [22] and 6–16 mm in diameter [23].

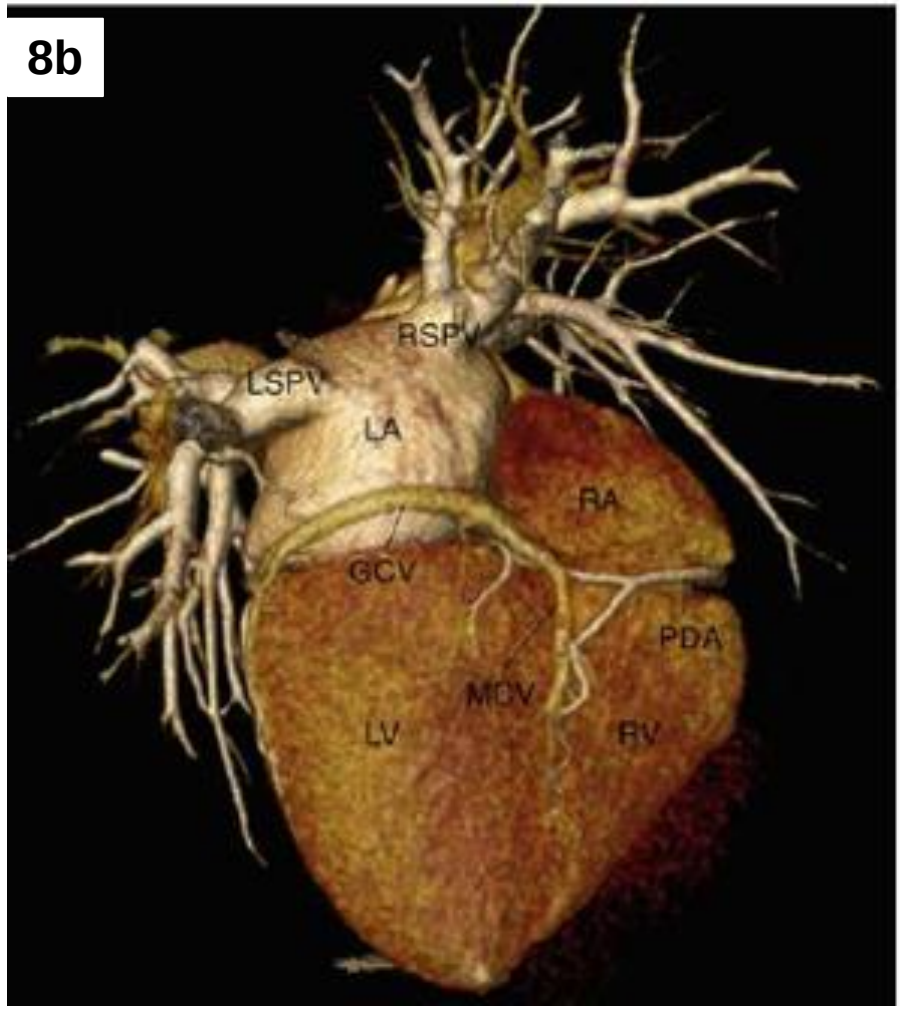
As shown in **Figs. 8a and 8b** the CS receives blood from the great cardiac vein that channels blood from its tributaries, the anterior interventricular vein, the middle cardiac vein, the left obtuse marginal vein, the right coronary vein and atrial veins of which the most well known is the vein of Marshall. The coronary sinus is generally surrounded by myocardial musculature which extends from the right and left atrial walls [24]. This musculature may extend for a further 2–11 mm along the great cardiac vein [25].

Distal to this the venous wall is not surrounded by musculature and therefore perforation through instrumentation is more likely. The Valve of Vieussens rarely causes a significant obstruction to the advancement of a catheter but rather the acute bend in the vein beyond this or an advancement into a side branch are more common causes of cannulation problems. It is therefore better to slowly withdraw and rotate the catheter rather than to try to advance further. The anterior interventricular vein courses from close to the LV apex and then continues into the great cardiac vein that into the left AV groove under the

left atrial appendage[26]. Distally the great cardiac vein receives left atrial veins including the Vein of Marshall and more proximally ventricular veins from the anterior RV and LV and the interventricular septum. The middle cardiac vein joins the CS close to the os. Occasionally it may also enter the RA directly. This vein runs along the diaphragmatic surface between the LV and RV with a close proximity to the right coronary artery and in particular the branch to the AV node. It may be used to map accessory pathways in the pyramidal space.

Figs. 8a





Figs. 8a and 8b The coronary sinus and its branches. The Fig 6a image is a diagrammatic representation of the coronary sinus (*CS*) and its main tributaries the great cardiac vein (*GCV*), middle cardiac vein (*MCV*) as well as posterior veins (*PV*) and posterolateral veins (*PLV*) seen in an LAO view. In the image 6b this the CT shows the coronary sinus running along the inferior left atrium (*LA*) as the great cardiac vein (*GCV*). The middle cardiac vein (*MCV*) runs between the left ventricle (*LV*) and the right ventricle (RV). The posterior descending artery (*PDA*) is seen running between the right atrium (*RA*) and the right ventricle (*RV* . Also seen in this image is the left superior pulmonary vein (*LSPV*) and the right superior pulmonary vein (*RSPV*)

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