Anatomic Pitfalls: Be Careful!

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Objectives
At the conclusion of this lecture, the participant should be able to:
1) Understand the embryological basis of many common anatomic variants
2) Classify the anatomic pitfalls by location in the heart
3) Differentiate anatomic pitfalls from a true pathological finding

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Introduction
Normal structures or variants can be misinterpreted as pathological conditions, called anatomic pitfalls. Artifacts or errors in interpretation of images are due to the inherent properties of the ultrasound technology being used. It is important to be familiar with the common anatomic pitfalls and artifacts to facilitate recognition and avoid unnecessary interventions.

Anatomic Pitfalls Result from Embryologic Remnants
Many normal anatomic variants are remnant structures from embryological development and in utero circulation. Further, some minor abnormalities of embryological development can result in common but usually inconsequential findings on TEE.

There are three structures in the right atrium that develop from vestigial embryological structures:
1) Superiorly, the structure between the smooth atrial tissue and the trabeculated part of the right atrium is the **crista terminalis**. 2) Inferiorly, the **valve of the inferior vena cava** or the **eustachian valve** is formed, which serves to direct blood flow from the inferior vena cava across the foramen ovale in utero. 3) Inferiorly, the **valve to the coronary sinus**, the **thebesian valve** is formed.

Also within the atria, migration of the atrial septum to create separate right and left atria leads to the characteristic appearance of the **foramen ovale**, with the possibility of a **patent foramen ovale**.
In the left atrium, the smooth tissue of the pulmonary veins is incorporated into the wall of the left atrium; it displaces the trabeculated tissue into the left atrial appendage. Further, the junction of the left atrial appendage and the left superior pulmonary vein appears to be a ridge of tissue, which has been called the coumadin ridge.

A failure of cardiac vein development and reabsorption may lead to a persistent left superior vena cava, which connects to and creates a dilated coronary sinus.

Description of Anatomic Pitfalls

**Crista Terminalis:** The crista terminalis is seen at the junction of the SVC and the right atrium, forming a structure, which may appear to protrude into the right atrium. This structure is often visualized in the bicaval view, not to be mistaken for thrombus or tumor.

**Eustachian Valve:** The eustachian valve can be seen in the 4-chamber view or the bicaval view of the right atrium; it is seen in approximately 25% individuals, at the junction of the IVC and right atrium. It appears as an elongated, membranous, possibly undulating structure. Usually, it is of no physiological consequence, but can be confused with an intracardiac thrombus, cause turbulent atrial blood flow, complicate IVC cannulation or serve as a site for endocarditis formation.

**Thebesian valve:** This is a valve, which may be seen as a thin piece of tissue guarding the entrance to the coronary sinus in the 4 chamber 0-degree view (slightly pushed in to visualize the coronary sinus). It may inhibit cannulation of the coronary sinus for retrograde cardioplegia.

**Chiari Network:** The chiari network is a very mobile, filamentous, thin and possibly perforated structure commonly seen within the right atrium. It is probably a remnant of sinus venosus derived structures. The chiari network moves about the right atrium with a random movement, not necessarily related to valvular movement. It has little significance but has been associated with a patent foramen ovale, paradoxical embolus and an interatrial septal aneurysm.

**Coronary Sinus:** At a mid-esophageal 0 degree view with slight probe advancement, the coronary sinus is seen in a longitudinally axis as an echo-free space just superior to the tricuspid valve in the right atrium. Dilation of the coronary sinus to greater than 1 cm suggests a persistent left superior vena cava. The coronary sinus can also be seen in the 2-chamber 90-degree view as small echo-free circles in the AV groove on either side of the left atrium.

**Persistent Left Superior Vena Cava (LSVC):** A persistent left superior vena cava will drain into the coronary sinus along the atrioventricular groove, making the coronary sinus larger than usual. It can easily be seen between the left atrial appendage and descending aorta, where it is usually small. In this setting it may be misinterpreted as a cyst or abscess cavity. A persistent left superior vena cava can also be seen between the left upper pulmonary vein and the left atrial appendage; it should have color flow in it. An agitated saline injection into a left upper extremity vein which opacifies the coronary sinus and then the right atrium will confirm the diagnosis.
**Trabeculations:** Trabeculations are more characteristic of the right atrium and ventricle than the left atrium and ventricle. They are caused by muscle bundles on the endocardial surface. Right ventricular hypertrophy may accentuate these trabeculations. The left atrial appendage may have trabeculations. The left ventricle is characterized by a smoother endocardial surface but may have finer trabeculations than the RV.

**Pectinate Muscles:** A series of parallel ridges known as pectinate muscles course across the anterior endocardial surfaces of the left and right atria, including both appendages. Prominent pectinate muscles should not be associated with arrhythmias such as atrial fibrillation or low flow states such as mitral stenosis to distinguish them from thrombus, especially in the left atrial appendage.

**Right Atrial Appendage (RAA):** The RAA is most commonly seen in a bicaual view and may have prominent trabeculations or pectinate muscles. The RAA can appear as an echo free space anterior to the ascending aorta and near the right ventricular outflow tract at 120-degrees beam rotation.

**Left atrial appendage (LAA):** The LAA is best seen in a 2-chamber 90-degree view; it is separated from the left superior pulmonary vein by the coumadin ridge. Trabeculations and pectinate muscles must be distinguished from thrombus.

**Ligament of Marshall:** The atrial tissue separating the entrance of the left upper pulmonary vein from the left atrial appendage commonly has a globular fatty appearance, not unlike a “Q-tip”. It has been termed the "warfarin" or "coumadin ridge" as it has frequently been misinterpreted to be thrombus leading to treatment with anticoagulants.

**Normal foramen ovale:** The normal foramen ovale appears as thin slice of tissue bound by thicker ridges of tissue, one of which is seen as a “flap” of tissue. Up to 25% of foramen ovale may be probe patent. Evaluation of the foramen ovale should include color flow mapping and a bubble study with a valsalva maneuver to provoke right to left shunting if it is present. The importance of a patent foramen ovale must be evaluated in combination with other information such as a history or paradoxical emboli or the finding of unexpectedly high right-sided cardiac pressures.

**Lipomatous Hypertrophy of the Atrial Septum:** Lipomatous thickening of the interatrial septum may be quite striking, and may mimic an infiltrative process. This benign process creates a dumbbell-like appearance of the superior and inferior atrial septum, and is characterized by the lack of involvement of the fossa ovalis. The echogenic fat may also involve the right atrial wall.

**Interatrial septal aneurysm:** This condition may be idiopathic or may develop as the result of right heart dysfunction, perhaps with high right-sided pressures. The interatrial septum is enlarged and seen to be moving in and out of each atrium with the cardiac cycle. It is associated with a patent foramen ovale and perhaps paradoxical embolism as it may be prone to thrombus formation.

**Moderator band:** The most prominent of the muscle bands is moderator band, which lies in the apical third of the right ventricle. It can be confused with a tumor or thrombus. The moderator band is involved with the conduction system as Pukinjie fibers may course through it. The left ventricle does not have a moderator band.

**False tendons:** False tendons can be seen in the left ventricle and usually have little clinical significance. They are finer, more filamentous structures than a moderator band. These structures are thought to represent false chordae tendonae. Some have claimed an association with murmurs, arrhythmias and tethering of papillary muscles.
Pitfalls

The normal pericardial sac contains about 30 ml of pericardial fluid, not easily seen with TEE. However, a larger effusion can separate the myocardium from the pericardium and create echo free spaces. The clinical significance is dependent on the degree of ventricular compromise due to the accumulation of fluid or blood. The pericardial sac may also be calcified. The transverse sinus is formed from a pericardial reflection between the posterior wall of the ascending aorta, the anterior left atrium and the posterior pulmonary artery. It may be misinterpreted as a cyst or abscess cavity. It should not have color flow in it, but can have fibrinous material, fat or fluid in it. The oblique sinus is a pericardial reflection inferior to the transverse sinus, between the right and left pulmonary veins and medial to the inferior vena cava.

Pleural effusion – Effusions (most commonly in the left pleural space) can occasionally be seen lateral and posterior to the heart.

Nodulus Arantii: These are small fibrous nodules, which can be seen at the center of the free edge of each cusp of the aortic valve. The clinical significance is unknown but they may be due to repeated cusp coaptation.

Lambl’s excrescences: These are small filamentous structures, which appear off of the aortic valve leaflets, usually on the aortic side of the valve. They have questionable clinical significance, but have been thought to be associated with stroke.

Abnormals to be distinguished from Anatomic Variants
A right atrial appendage or right atrial wall clot – is not a pectinate muscle
Right atrial endocarditis - is not the crista terminalis, Eustachian valve or a thebesian valve
A left atrial appendage clot – is not the ligament of Marshall
A LV thrombus - is not a LV trabeculation
Aortic valve endocarditis - is not a Lambl’s excrescence

How to distinguish normal anatomic variants from pathological structures
Anatomic variants are seen in multiple planes and are persistent with changes in gain, compression, and depth (which distinguish them from artifacts). These features distinguish them from many artifacts. Anatomic variants usually do not impose any physiological consequence. For example, a eustachian valve which appears to be a secundum ASD would not be associated with signs of volume overload such as RAE or RVH. Further PA pressure may be normal when one would expect high PA pressures with a large, previously undetected ASD. Anatomic variants, which masquerade as endocarditis (trabeculations, pectinate muscles, Lambl’s excrescences) can be distinguished by a lack of other supportive evidence for endocarditis such as fever and positive blood cultures. However, some anatomic variants do have implications for care of the patient. For example, a persistent left superior vena cava may prohibit the use of retrograde cardioplegia. Another example is that the finding of an interatrial septal aneurysm should prompt a search for a patent foramen ovale. Some general principals apply:

1. Look for similar density of tissue as surrounding tissue. If very different - likely to be pathologic
2. Does it appear in multiple planes and views - distinguish from artifact
3. Does it appear with changes of depth and gain?
4. Look for secondary signs – smoke in LA
5. Does it cause a functional problem? - a right atrial mass causing tricuspid stenosis, know HPI
6. Distinguish right anatomy with agitated saline injections
7. Know the usual variants and artifacts