Transesophageal Echocardiography in Atrial Fibrillation

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KEYWORDS

• Transesophageal echocardiography • Atrial fibrillation • Stroke • Left atrial appendage

KEY POINTS

- Transesophageal echocardiography (TEE) plays an important role in atrial fibrillation (AF), mainly to detect the presence of left atrial appendage (LAA) thrombus.
- TEE has proven to be useful in direct current cardioversion guidance and is indispensable for AF ablation and LAA occlusion.
- With the increasing numbers of patients affected by AF, the use of TEE will grow and become an important screening modality for detecting LAA thrombus.
- The future direction includes broader multi-institutional use; the implementation of further strategies to risk stratify the patients; as well as the use of new oral anticoagulants and their cost-effectiveness in patients with AF undergoing direct current cardioversion, AF ablation, and LAA occlusion.

Videos of spontaneous echocardiographic contrast (SEC), sludge and thrombus accompany this article at http://www.cardiacep.theclinics.com

Atrial fibrillation (AF) is the most common pathologic supraventricular tachycardia,[1] with an overall prevalence of 0.4% to 1% in the general population, increasing with age.[2–4] The estimate of the prevalence of AF in the United States ranged from ~2.7 million to 6.1 million in 2010 and is expected to increase to between ~5.6 and 12 million in 2050.[2,5] The mean age of patients with AF is 66.8 years for men and 74.6 years for women.[6] Approximately 2% patients in AF are 60 to 69 years old and 5% are greater than or equal to 70 years old. It is more common in men than in women and in Caucasians than in African-Americans.[7–10] AF is associated with an increased long-term risk of stroke,[11] heart failure, and all-cause mortality, especially in women.[12] The age and gender-adjusted 30-day and 1-year mortality is 11% and 25% respectively.[13]

AF is characterized by chaotic contraction of the atrium resulting in loss of atrial mechanical function, which leads to impaired diastolic filling of the left ventricle and predisposes to blood stasis. The coupling of endocardial damage as well as abnormalities of coagulation, platelets and fibrinolysis fulfill Virchow's triad for thrombogenesis and is consistent with a prothrombotic or hypercoagulable state in this arrhythmia.[14] The most serious complication of AF is systemic thromboembolism. AF is associated with approximately 1 in 6 ischemic strokes.[7] Data from the

Funding Support: None.
Financial Disclosures and/or Conflict of Interest: The authors have nothing to disclose.
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Card Electrophysiol Clin 6 (2014) 43–59
http://dx.doi.org/10.1016/j.ccep.2013.11.006
1877-9182/14/$ – see front matter

Framingham study indicated that AF alone is associated with a 3-fold to 4-fold increased risk of stroke after adjustment for other stroke risk factors.[7] An ischemic stroke may occur in patients with AF either as the initial presentation and despite appropriate antithrombotic prophylaxis. When intracardiac thrombus is identified in patients with nonvalvular AF, its location is the left atrial appendage (LAA) in more than 90% of the cases.[15]

Electrical cardioversion of patients with AF to normal sinus rhythm is performed frequently to relieve symptoms, improve cardiac performance, and possibly decrease cardioembolic risk. AF ablation has emerged as an established strategy for a restoration of sinus rhythm in patients with AF. LAA occlusion offers a theoretically appealing method to reduce the incidence of stroke. This article discusses the role of transesophageal echocardiography (TEE) in patient evaluation before cardioversion, AF ablation, and LAA occlusion.

LAA

The LAA is a small, actively contracting, fingerlike blind cul-de-sac, situated on the lateral aspect of the left atrium (LA). The mouth of the LAA is located between the left ventricle and the left upper pulmonary vein (PV), extending over the atrioventricular groove and the surface of the left ventricle toward the left circumflex artery in the anterior direction (**Fig. 1**).[16] It is variable in size (ranging from 0.77 to 19.27 cm),[17] shape, and orientation (the principal axis is markedly bent or spiral). The appendage communicates with the atrial chamber through a narrow oval-shaped orifice with a mean long diameter of 17.4 ± 4 mm and a short diameter of 10.9 ± 4.2 mm measured in heart specimens.[18] It is lined with endothelium and trabeculated by pectinate muscle. The embryology of LAA is distinct from the body of the LA. The LA is formed by the absorption of the primordial PV and its branches, resulting in the smooth-walled cavity. In contrast, the trabecular LAA is the remnant of the original primordial LA, which develops during the third week of gestation.[19] The multiple small tunnels created by pectinate muscles and the narrow apex of the LAA are the two anatomic factors that lead to thrombus formation when LAA systolic function is impaired.[20] An autopsy study of 500 normal human hearts noted that 80% of the LAAs had multiple lobes and the presence of 2 lobes was most common, found in 54% of cases, followed by 3 lobes (23%), 1 lobe (20%), and 4 lobes (3%).[21]

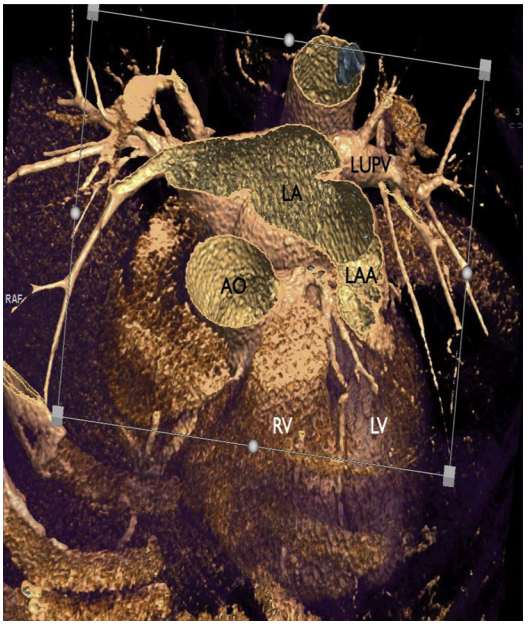

Fig. 1. Anatomic correlation of LAA and other cardiac structures. Ao, aorta; LA, left atrium; LUPV, left upper pulmonary vein; LV, left ventricle; RV, right ventricle. (*Courtesy of* Paul Schoenhagen, MD, Cleveland Clinic, Cleveland, OH.)

The LAA is best visualized in the midesophageal window starting at 0° and often with slight flexion or withdrawal of the probe to a more cranial position.[22] It is critical to image the LAA from multiple imaging planes including 0°, 45°, 90°, and 120°. The complex shape and multilobed structure of the LAA are usually only noted at an angle beyond 100° (**Fig. 2**). Often the biplane feature during TEE is used to assess the LAA in perpendicular views simultaneously and this is especially important for assessing LAA thrombus.

LAA FLOW VELOCITY PATTERN

LAA flow velocities can be assessed with TEE using pulsed wave Doppler with the sample volume placed 1 cm within the LAA. In patients with sinus rhythm, the LAA flow is quadriphasic (**Fig. 3**A, B) with a distinct pattern of contraction, as described below.[23,24]

1. During atrial systole, LAA contraction and emptying flow or late diastolic emptying velocity is seen immediately after the P wave, which is the most important wave during sinus rhythm. It is a marker of LAA contractile function; correlates with LAA ejection fraction, LA size, and pressure; and is a significant predictor of thromboembolic risk. The average LAA contraction velocity is 50 to 60 cm/s.