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Left atrial volume index as a clinical marker for atrial fibrillation and predictor of cardiovascular outcomes

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Editorial

The prevalence of AF increases with aging and with the severity of the heart disease reaching up to 40% in advanced cases. In patients with heart failure, AF is an independent predictor of morbidity and mortality increasing the risk of death and hospitalization. It is well known that the presence of atrial enlargement in patients with organic heart disease increases the chances to develop AF.[1–5] Left atrial (LA) enlargement measured by echocardiography is considered to be a useful tool in the evaluation of cardiovascular outcomes. Guidelines from the American Society of Echocardiography provide clarification as to which of the multiple methods to estimate LA size should be used in clinical practice. It has been demonstrated that LA volume and LA volume index provide a more accurate measure of LA size than conventional M-mode LA dimension.[6,7] The LA mechanical functionality can be described to be as a reservoir, a conduit, and a contractile function.[8] During ventricular systole and isovolumic relaxation, the LA functions as a reservoir that receives blood from pulmonary veins. Then, during the early phase of ventricular diastole, the LA operates as a conduit for transfer of blood into the left ventricle (LV) after mitral valve opening via a pressure gradient. In addition, the atrial kick or the contractile function of the LA serves to increase the LV stroke volume by approximately 20-30%.[9] When the diastolic function is normal, the relative contribution of the reservoir function of the LA to the LV filling is 40%, the conduit 35%, and the contractile function 25%.[10] The relative contribution of LA phasic function to LV filling is dependent upon the LV diastolic properties in a way that this LA booster pump function becomes more dominant in the setting of LV dysfunction.[11,12] When the LV relaxation is altered, the relative contribution of LA reservoir and contractile function increases and the conduit function decreases. However, as LV filling pressure progressively increases with advancing diastolic dysfunction, the LA serves predominantly as a conduit.[10]

Several alterations are associated with LA remodeling and dilatation. The increase in prevalence of AF in older persons has been reported to be associated with degeneration of the atrial muscle in pathological studies. It was demonstrated that there is clear evidence in the human atrial muscle of age-related electrical uncoupling of the side-to-side connections between bundles, related to the proliferation of extensive collagenous tissue septa in intracellular spaces.[13,14] These age-induced changes include a reduction in the number of myocardial cells within the sinus node, a generalized loss of atrial myocardial fibers, as well as an increase in fibrosis which leads to an apparent loss of myocardial fiber continuity.[15–17] The LA remodeling and dilatation process will also occur in response to pressure and volume overload. LA enlargement due to pressure overload is usually secondary to increased LA afterload. High blood pressure increases LV end-diastolic pressure and induce LV diastolic dysfunction, which subsequently increases the LA pressure and causes stress on LA walls. The LA pressure overload induces pathophysiological changes, which causes structural and functional remodeling. These changes alter the electrophysiological properties of the atrial myocardium increasing atrial vulnerability and the predisposition to develop episodes of AF.[18–22] LA dilation is attributed to impairment of the diastolic blood flow from the LA to the LV due to the increased LV stiffness. It has been suggested that LA dilatation can also occur in response to pressure overload resulting from fibrosis and calcification of the LA, a condition known as stiff LA syndrome.[23,24] This entity causes a reduction of LA compliance, a marked increase in LA and pulmonary pressures, and right heart failure. Chronic volume overload associated with conditions with high output states can also contribute to generalized chamber enlargement.[25,26] Two-dimensional and tissue Doppler imaging at different phases of the cardiac cycle have been utilized in LA volume measurement and various LA functions. Interesting prospective results and outcomes from large population-based studies have established a relationship between M-mode antero-posterior LA diameter and the risk of developing AF.[27,28] For example, in the Framingham study, a 5-mm incremental increase in antero-posterior LA diameter was associated with a 39% increased risk for subsequent development of AF.[27] In the Cardiovascular Health Study, subjects in sinus rhythm with an antero-posterior LA diameter greater than 5cm had approximately fourfold the risk of developing AF in the follow-up period.[28] LA volume index has been shown to predict AF in patients with cardiomyopathy, and also in first-diagnosed nonvalvular AF.[29–32] These studies have shown that LA volume index represents a superior measure over LA diameter for predicting cardiovascular outcomes and provided prognostic information that was incremental to clinical risk factors. Tenekecioglu E et al.,[33] demonstrated in their investigation that the left atrium in the hypertensive group with AF was characterized by further enlargement when compared to the hypertensive group without AF.[33] While LA booster pump

function was increased in hypertensive patients when compared to normotensive subjects, it was impaired in hypertensive subjects with AF as compared with hypertensive patients without AF.[33] These are interesting data since arterial hypertension causes an increase in LV wall stress that generates myocardial hypertrophy. Increased LV wall thickness elevates LV diastolic filling pressure inducing a fibro-degenerative process within the myocardium of the left chambers. This fibrosis constitutes a favorable substrate for reentrant arrhythmias. The occurrence of AF in hypertensive individuals may be associated with impairment of atrial contractility.

Patients with an enlarged LA and altered atrial myocardium tend to have increased load and wall stress with atrial myocardial impairment causing contractile dysfunction and a milieu for electrical conduction abnormalities within the atrial myocardium. LA volume index and function is a useful tool for monitoring cardiovascular risk and outcomes and for guiding medical therapy. Continuously evolving technology will enhance its utility and may prove to have an interesting impact in global health care.

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Conflicts of interest

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