Left atrium phasic volume assessment: Its application in stroke patients

Stroke is one of the most common causes of morbidity and mortality. The survivors of stroke have a risk of recurrence that leads to long-term disability and death. Embolism with a cardiac origin, however, is difficult to document, but accounts for approximately 20% of ischemic stroke. The emboli are most likely to originate either from atria, ventricles, or cardiac valves. The left atrial appendage (LAA) as an embryological remnant functioning as a reservoir is the prevalent site of thrombosis in patients with atrial fibrillation (AF) or rheumatic mitral valve disease. The specific LAA morphology with or without organized atrial systole is independently associated with an increased risk of stroke. Beyond morphology, LAA without organized atrial systole is independently associated with mitral valve disease. The specific LAA morphology with or without organized atrial systole is independently associated with mitral valve disease. The left atrium (LA) attached by LAA is most likely to originate either from atria, ventricles, or cardiac valves. The left atrial appendage (LAA) as an embryological remnant functioning as a reservoir is the prevalent site of thrombosis in patients with atrial fibrillation (AF) or rheumatic mitral valve disease. The specific LAA morphology with or without organized atrial systole is independently associated with an increased risk of stroke.

Despite the well-established methodology of measuring LAA structures and functions, the assessment depends mostly on invasive transesophageal echocardiography. Complete structural and functional evaluations of the LAA are inherently prone to substantial variability during data acquisition, particularly pulsed-wave Doppler interrogation, because of anatomical challenges. The left atrium (LA) attached by LAA is easily evaluated by transthoracic echocardiography. The morphological changes of LA were a significant predictor of AF recurrence after transcatheter ablation. Regardless of the presence of AF or valvular abnormalities, LA enlargement is independently associated with cardiovascular events, particularly ischemic stroke, across various populations and genders.

The LA modulates left ventricle (LV) filling by serving as a reservoir or a conduit delivering blood from pulmonary veins to LV as well as an active contractile chamber in late diastole and a suction source in early systole. Growing evidence suggests an association between LA enlargement and ventricular, arterial, and arrhythmic disorders. In addition, LA dysfunction occurs in patients with patent foramen ovale, which is associated with cryptogenic stroke. An enlarged LA may be a marker of the complexity of other cardiovascular conditions that could also increase stroke risk.

Conventionally, echocardiographic assessment measuring maximum anteroposterior dimension on M-mode or 2D echocardiography in the parasternal long axis used extensively in clinical practice is an inaccurate presentation of the true LA size or volume; in particular, enlargement occurs asymmetrically in the medial—lateral or superior—inferior axes. The American Society of Echocardiography, in conjunction with the European Association of Echocardiography, recommended either an ellipsoid model or the Simpson’s method for LA volume measurement. LA volume evaluated by recently developed three dimensional (3D) echocardiography has more favorable agreement in test—retest, intra—observer, and interobserver variations than other echocardiographic techniques. The major disadvantage of the 2D or 3D echocardiographic methodology is that procedures are time consuming and thus not practical in clinical practice. Moreover, the size of LA varies during the cardiac cycle. Total LA volume is a composite of phasic volume changes reflecting LA dynamic functions. The LA passive emptying volume represents early diastolic filling; the LA conduit volume is the amount of blood transited from pulmonary veins to LA whereas mitral valve is open; the LA active emptying volume represents blood actively propelled into LV.

Velocity vector imaging (VVI) is a novel image quantitative analysis technique using a speckle tracking echocardiographic method that assesses details of regional and global myocardial functions. VVI involves complex signal processing but requires only a single frame tracing of endocardial borders for an easy assessment of the time—volume information. VVI provided a highly correlated evaluation of LA volume with a reduction in time of measurement as compared with the conventional 2D methodology. Furthermore, VVI assesses LA phasic function beyond maximum and minimum LA volume and LA function.

In this issue of the Journal of the Chinese Medical Association, Dr Chen and his colleagues prospectively enrolled healthy controls and patients with various types of stroke in sinus rhythm. The phasic LA volumes were assessed by VVI and were compared across 20 controls and 87 stroke patients. The LA function assessed by pulmonary venous flow was of no significant difference among healthy controls and stroke patients of all types. However, LV diastolic function was impaired in stroke patients. In spite of similar maximum LA volume, minimum LA volume was elevated in the stroke patients. Compared to controls, patients with stroke had impaired LA reservoir function as well as depressed passive emptying function whereas the active emptying function had greater contribution in LV filling. The same observation...
occurred in stroke patients as the elevated mitral A indicated an increased LA contractile function in the setting of LV diastolic dysfunction. LA phasic functions were not different in the subtypes of stroke. The authors concluded that their findings suggest that functional changes of LA may occur without significant structural changes.

There are several points that need to be addressed. First, LA volume assessed by VVI has not been compared to the gold standard indexed by cardiac magnetic resonance imaging or computed tomography. Previous analyses indicate that the VVI method tends to overestimate maximum LA volume and to underestimate minimum LA volume. Second, VVI analyses were performed utilizing the 4-chamber view. However, the conventional 2D echocardiography using the biplane analysis is more accurate for LA volume assessment. In addition, the LA volumes presented in this study were not indexed to the measure of body size, which are preferred by the guideline. Third, the contribution of LA phasic function to LV filling is dependent on diastolic properties. Besides reservoir and contractile functions, LA conduit function accounts for 35% of LV filling in the normal heart and decreases in abnormal LV relaxation. The conduit function was not evaluated in the current study. Finally, in spite of atrial phasic function assessed by volume changes, the current information indicates that maximum and minimum LA volume occurring prior to mitral valve opening and closure is associated with the development of adverse cardiovascular outcomes.

References

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