

Investigating the Left Atrial Function by Strain Echocardiography: Modern Answers to Old Questions?

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Abstract

Modern ultrasound technology allows investigating both atrial and ventricular morphofunctional abnormalities. This article deals with the rising evidence for strain echocardiography to be a promising tool for preclinical assessment of left atrial chamber impairment and disease staging in several clinical settings.

Keywords: Atrial fibrosis; Atrial function; Atrial stiffness; Echocardiography; Strain echocardiography

Editorial

Echocardiography has been proven to be a useful diagnostic tool for assessing the cardiac function in several clinical settings noninvasively. There is a rising scientific evidence indicating that modern ultrasound techniques like tissue Doppler and strain imaging are likely to overcome some limitations of standard m-mode, two-dimensional and Doppler modalities, especially when an early myocardial impairment has to be established [1,2].

Over the last three decades, the left ventricular (LV, left ventricle) systolic function has been identified by regional wall motion analysis and ejection fraction, which still remain the most largely accepted semi-quantitative markers in daily clinical practice. However, strain imaging is leading to a drastic improvement in some pathophysiological concepts and the preclinical diagnosis of LV dysfunction became easier than in the past [2,3].

In addition, evolving strain techniques are currently being tested for LA function [4,5]. In the previous issue of the Journal, Mahfouz and Colleagues [6] present an interesting study on the utility of LA stiffness in predicting clinical outcomes in 64 children with Acute Rheumatic Fever (ARF).

Though the occurrence of ARF has been dramatically reduced by improvements in health care systems, it remains a cause of morbidity and mortality in resource-limited Countries [7]. Regardless of the modern antibiotic therapies, Group-A Streptococcal infection can cause an autoimmune pancarditis (often limited to endocarditis) that may result in a chronic invalidating heart disease. Current guidelines for such patients are largely based on expert opinion, and the absence of randomized trials do not allow precise indications to their management [7,8]. Therefore, even single-group contributions are welcome in order to improve our clinical practice.

The study of Mahfouz et al. [6] deals with the prognostic impact of LA stiffness, calculated as the ratio between E/E' ratio and the peak longitudinal strain (PALS), in children with rheumatic carditis. Higher chamber volume (26.1 ± 3.5 ml/m²), reduced peak global longitudinal strain (PALS) ($20.8 \pm 4.6\%$) and increased stiffness (0.69 ± 0.19) were

demonstrated to affect patients with than without carditis. These findings likely indicate a remarkable involvement of the LA in the rheumatic disease, and a LA stiffness ≥ 0.63 was predictive of clinical events (included ARF recurrences) during the follow-up. The interest of this study is on the fact that cardiologists, beyond valve morphology, should better investigate the whole cardiac function in ARF patients.

As for the LV, LA compliance is a dynamic marker of advantageous relationship between filling pressure and volume ($\Delta P/\Delta V$) within the atrial chamber. Because the noninvasive estimation of LA stiffness has not been validated yet, this study offers further hints by confirming that both PALS and E/E' ratio can be surrogate markers of LA stiffness.

Actually, Khurram et al. [9] recently demonstrated that LA stiffness index (as the ratio between invasive pressure estimation and volume measurement at cardiac magnetic resonance imaging) increases with age and it is higher in adult patients with persistent atrial fibrillation (AF) in a setting of reiterate ablative treatment. This would confirm that atrial fibrosis is underestimated in clinical practice, especially in the elderly.

Mahfouz et al., however, indicate the risk of atrial wall impairment is also higher in ARF children. Should the acute process be eradicated, the residual LA fibrosis may represent an arrhythmic substrate on the adult age [6]. Accordingly, it is conceivable that other (and more common) inflammatory diseases in the early stages of our life may result in a subliminal risk for atrial (and/or ventricular) wall impairment, which could be detected by merging strain and magnetic resonance imaging findings, as reported in hypertrophic cardiomyopathy [10] and patients with AF [11]. High LA stiffness was also related to arrhythmic recurrences after ablation for AF [9].

Despite the lack of atrial-tailored software, strain echocardiography is being to be validated to this purpose. Three phases of the atrial cycle (Figure 1) have been clearly identified by both speckle-tracking and vector-velocity strain methods. Peak longitudinal strain seems to be the most important component of the reservoir function, and it is considered a surrogate marker of atrial wall damage in various clinical settings (hypertension, diabetes, protein deposition, interstitial fibrosis) [11-13].

Both PALS and LA contraction were found to impair (irrespective of the chamber size) in both hypertrophic cardiomyopathy and TTR-

amyloidosis, but greatly in this latter setting due to a more frequent amyloid deposition into the atrial wall [14].

More recently, Marino et al. [15] also demonstrated a linear relationship between invasive estimation of LA stiffness and longitudinal strain from both the left cardiac chambers, confirming an essential atrio-ventricular functional continuum. These authors suggest the formula $0.735+0.051*LV$ longitudinal strain as a best way to calculate LA stiffness from LV deformation.

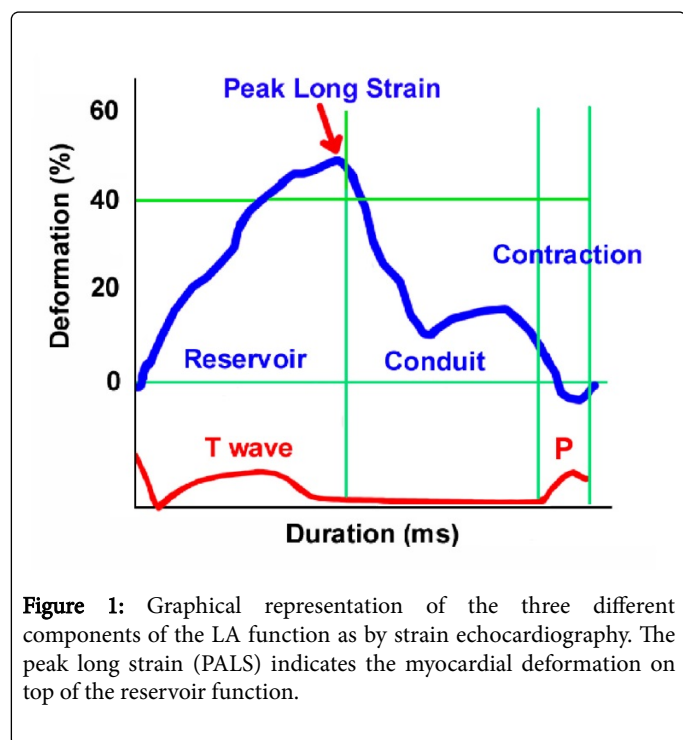


Figure 1: Graphical representation of the three different components of the LA function as by strain echocardiography. The peak long strain (PALS) indicates the myocardial deformation on top of the reservoir function.

Regrettably, concerns have risen about reproducibility of strain measurements. Significant inter-Vendor discrepancies have been shown and clinical variables as age, gender, body mass, blood pressure and probably the race, likely affect strain values [16]. For these reasons, both the American and European Echocardiography Societies recommend researchers to be careful while comparing data from different devices and strongly encourage industries for a technical standardization [17,18].

In conclusion, our understanding of LA morphology and function is rapidly evolving, and recent studies suggest such an unexpected role in arrhythmic disorders and heart failure patients. Within the affluent multimodality scenario, strain echocardiography is going to explain old pathophysiological questions from various clinical settings. Again appreciating the Authors of this study, we are confident in a forthcoming implementation (and standardization) of strain techniques in order to widely support clinicians in their efforts to disclose myocardial dysfunction as early as possible, and improve the chances for a better clinical management.

Disclosures

No conflicts of interest declared.

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