Editorial

Left Atrium Reservoir Strain and Cardiovascular Hemodynamics: the Hidden Truths Reveal Far More Than What is Readily Apparent

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"If it is any point requiring reflection,
we shall examine it to better purpose in the dark."
— Edgar Allan Poe, The Purloined Letter (1844)

Over the past 15 years, Speckle-tracking Echocardiography has evolved into a robust technique for assessing both global and regional myocardial function. Among its expanding applications, Left Atrial (LA) strain, particularly in the reservoir phase, has emerged as a clinically relevant parameter. A reduced Left Atrial Strain reservoir (LASr) has been independently associated with elevated Left Ventricular (LV) filling pressures, recurrence of atrial fibrillation, heart failure hospitalizations, and overall cardiovascular mortality.[1,2] Reflecting this growing body of evidence, LASr has been formally incorporated into clinical practice in the recently published "*Recommendations for the Evaluation of Left Ventricular Diastolic Function by Echocardiography and for Heart Failure With Preserved Ejection Fraction Diagnosis: An Update From the American Society of Echocardiography.*"[3]

Despite this, the complexity of LA mechanics has largely followed an oversimplified approach: it is often treated as a surrogate marker of filling pressures, evaluated in isolation and interpreted using fixed cut-off values, without integrating the hemodynamic and mechanical context in which the atrium operates. While such an approach has facilitated its clinical adoption, it may obscure important physiological nuances and lead to misinterpretation in complex scenarios.

The LA wall is histologically similar to the pulmonary veins, with a mean thickness ranging from 2.3 to 4.4 mm, which can be less than 30% of the LV wall thickness.[4] This makes it highly sensitive to the mechanical influence of adjacent structures that operate at a much higher force scale. Therefore, from a physiological standpoint, LA reservoir strain is a composite biomarker, shaped by at least three major components:[5,6]

1. Early atrial relaxation, shown by the S1-wave of pulmonary venous flow, which indicates the initial suction phase of the reservoir function. This is best appreciated when LA volume changes are analyzed alongside LA pressure waveforms, and occasionally appears as a subtle notch at the onset of the LASr curve.
2. Longitudinal systolic function of the ventricle, responsible for the apical displacement of the Atrioventricular (AV) plane during systole. This component stretches the LA wall and directly contributes to reservoir strain amplitude.
3. Left atrial pressure, particularly the V-wave, which modulates atrial wall tension and LA compliance.

These three forces interact dynamically and define the shape and magnitude of the LASr curve. This concept has been supported by physiological studies showing that acute changes in afterload, such as those induced by isometric handgrip, can significantly impact LA reservoir strain.[7] In this setting, increased LV pressure results in altered LASr patterns, even in the absence of changes in intrinsic atrial function — highlighting the load dependency of this metric (Figure 1).

Such physiological interplay may yield practical clinical insights. Consider patients with non-severe mitral regurgitation undergoing Cardiac Resynchronization Therapy (CRT). Improvement in mitral regurgitation often reduces LA preload[8] and subsequently decreases LASr, despite favorable LV remodeling and decreased LV end-systolic volume. Without hemodynamic contextualization, this apparent paradox might be misinterpreted as functional worsening, when in fact it reflects normalization of loading conditions.

A second, more complex scenario involves patients with paradoxical low-flow, low-gradient aortic stenosis, a subgroup that often overlaps with HFpEF physiology. In such cases, low-dose nitroprusside infusion has been explored as a hemodynamic tool to assess contractile reserve and guide treatment. Simultaneous assessment of Pulmonary Capillary Wedge Pressure (PAWP) and Left Atrial (LA) strain during vasodilation can provide further insights: a reduction in PAWP accompanied by an increase in LASr, in response to reduced LA pressure, may indicate a load-sensitive phenotype with increased afterload-mediated systolic and diastolic dysfunction, which supports prioritizing guideline-directed medical therapy for HFpEF and provides caution and concerns towards more invasive treatment as Transcatheter Aortic Valve Implantation (TAVI), that could potentially lead to further increase in pulsatile vascular load and thus worsening abnormal ventricular-vascular coupling.[9] While these strategies remain exploratory, they reflect a growing recognition that dynamic assessment of atrial mechanics can enrich the diagnostic and therapeutic process.

Another fact that we need to consider is the interplay that exists between the different phases of Left Atrial physiology. In

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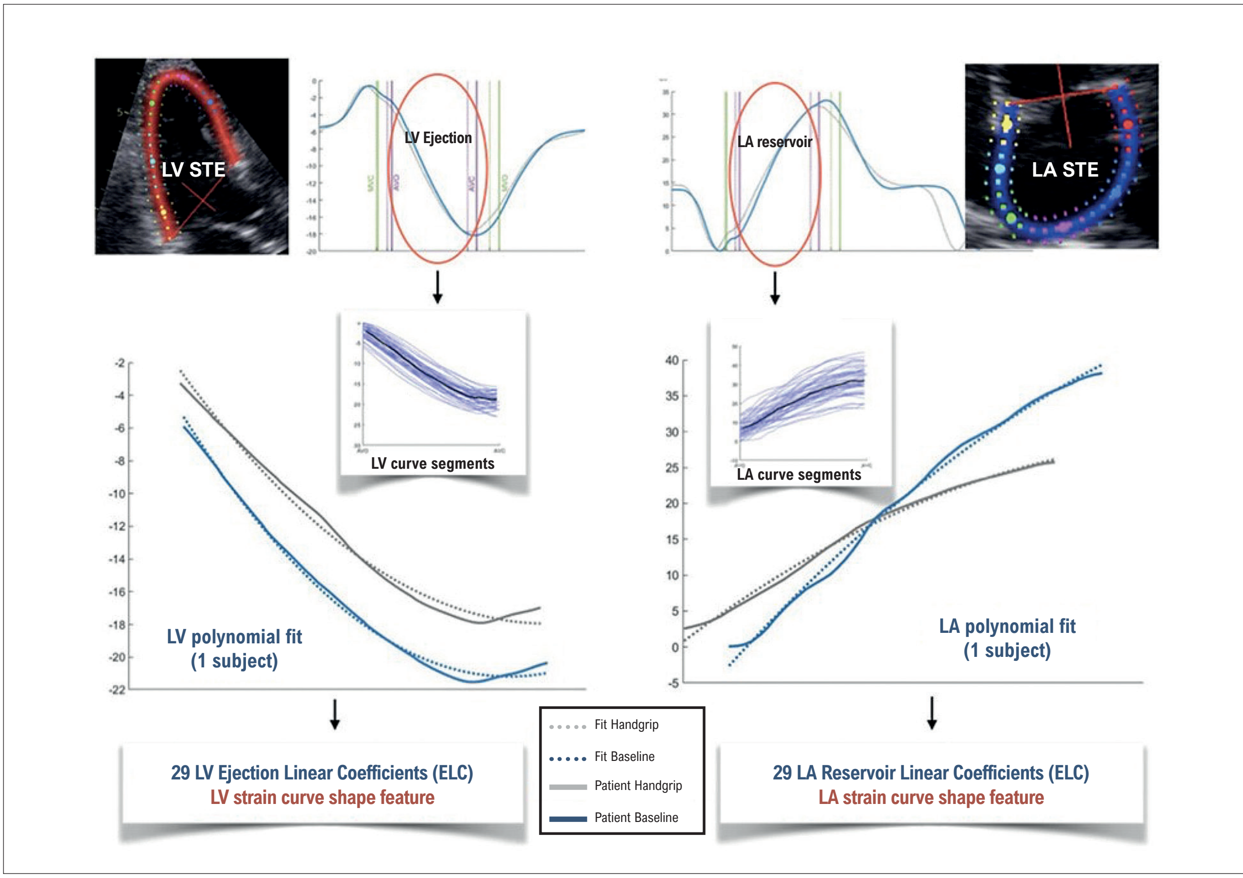

Figure 1 – *Speckle-tracking Echocardiography (STE) was used to extract strain curve segments from the left Ventricle (LV) during the ejection phase and from the Left Atrium (LA) during the reservoir phase. Polynomial regression was applied to each segment, generating 29 shape coefficients per phase: LV Ejection Linear Coefficients (ELC) and LA Reservoir Linear Coefficients (RLC). Curves from a representative subject (in blue) are displayed alongside population-level polynomial fits for baseline and isometric handgrip conditions (dotted lines). This approach was used to quantitatively assess ventricular and atrial myocardial mechanics beyond peak strain values. The acute increase in afterload induced by the handgrip maneuver mobilizes the contractile reserve of the LV and, in ventricles with limited reserve, reduces the velocity and magnitude of contraction. This imbalance may result in afterload-mediated LV systolic dysfunction and modify the slope of the LA reservoir strain curve, thereby altering its overall shape. These changes are quantitatively captured by shifts in the polynomial coefficients,[6] providing a quantitative marker of atrioventricular coupling and functional reserve under stress.*

some circumstances, a decrease in LA pressure could not affect the reservoir phase. This is probably because this phase also reflects the rigidity of the Left Atrium, which can be impacted by the chronicity of the disease. In these cases, the active contractile function of the left atrium may more accurately indicate improvements in hemodynamics.[10]

In conclusion, LASr should be interpreted not merely as a static marker of atrial function, but rather as a dynamic signal, modulated by ventricular mechanics and loading conditions. It is not so much what is revealed by absolute LASr values, but what is concealed within its shape, context, and variation under stress, that demands investigation. Future studies should aim to refine its application through integrated multiparametric approaches, incorporating both strain and hemodynamic markers. Such strategies may enhance the diagnostic precision of echocardiography in complex conditions like HFpEF, valvular disease, and atrial cardiomyopathy — where understanding the heart as a system, rather than as isolated chambers, is critical.

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