

Peak Atrial Longitudinal Strain (PALS): Better Call it Stretch?

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Abstract

Left atrial (LA) strain is gaining more and more relevance in medical literature, with many applications in different clinical settings. The term “strain”, meaning deformation, is applied to the contraction phase of the left ventricle (LV), due to its myocardial shortening along the longitudinal axis, to the LA relaxation phase, correlated to its distensibility and elastic compliance in receiving blood from the pulmonary veins, and even to LA contraction consequent to the electric activation of LA myocardium. This manuscript describes the main anatomical and physiological characteristic of the left atrium and discusses the use of the term strain from terminological and conceptual points of view.

Keywords: Left atrium; strain; stretch

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Introduction

The left atrium (LA) is an important cardiac chamber with a dual function, conduit and pump. The foundation of the LA function is its anatomical and myocardial fibre orientation.[1]

In essence, the myocardial fibre orientation almost mirrors the one of the left ventricle (LV). The pectinate fibres occupy the basal region of the LA, similar to the circumferential fibres at the base of the LV, control the modest systolic function of the basal region.[2] On the other hand, the predominant muscle layer is the longitudinal fibres which controls the longitudinal shortening of the cavity. These fibres originate at the back of the LA and insert around the circumference of the mitral annulus, thus pulling the base of the atrium towards its back during atrial systole, after the P wave, and as they relax they bring the annulus back to its resting position at end-diastole and the onset of the Q wave of the electrocardiogram (ECG). This phase is followed by LV systole during which its longitudinal subendocardial fibres contract to shorten the cavity across its long axis by pulling the mitral annulus, the site of their insertion, toward the LV apex.

During LV systole, the LA cavity is stretched longitudinally while its pressure drops and hence its filling from the pulmonary veins. For the LA, this phase is referred to as ‘reservoir’ at the end of which the intracavitary pressure increases and as it exceeds that of the LV the mitral valve opens and the onset of LV filling starts with fast acceleration. The deceleration of LV early diastolic filling is dictated by the diastolic pressure difference when LV pressure rises. LV diastasis follows early diastole before the start of atrial systole (Figure 1).

LA structure measurements in the form of longitudinal and transverse diameters, cavity area and volume are well established. LA function is historically assessed in the form of area change and volume fall during atrial systole, i.e. late diastole. Also, three-dimensional assessment of LA volume changes during different phases of the cardiac cycle have been recently invoked, with promising potential. While these measurements reflect overall LA cavity function, they do not assess intrinsic myocardial properties. Myocardial velocities have been used to overcome this limitation at different sites of basal LA segments,

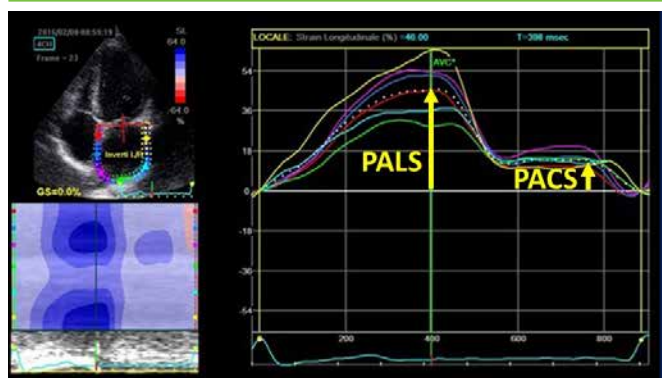


Figure 1. Diagram showing left atrial physiology during cardiac cycle.

lateral, septal, anterior and posterior. Those measurements have been found to reflect the old differentiated M-mode velocities.[3] While these velocities reflect the longitudinal LA segments, using the time of their peak played an important role in studying and assessing LA synchronicity.[4]

Recently, studying myocardial intrinsic function has become possible using speckle tracking 2D echocardiography technology, from which myocardial segmental and global strain and its derived strain rate can be evaluated.[5] Despite the technology was originally developed to assess LV deformation, it has been applied for studying LA deformation too with great success.[6] LA myocardial strain during atrial systole can now be accurately measured and is referred to as peak atrial contraction strain (PACS), with its respective strain rate. Changes happening in LA myocardium during LV systole have been referred to as peak longitudinal strain (PALS) (Figure 2).

In addition, LA myocardium with its elastic component acts as a hemodynamic mediator between LV and the pulmonary circulation. It has to ensure optimum filling of the LV while protecting the pulmonary capillary bed from possible pressure and volume overloads. In order to achieve that, particularly during its reservoir phase (assessed by PALS) three important factors contribute to LA function; its myocardial ultrastructure (ratio between elastic fibres and fibrous tissue), LV filling pressures and severity of volume overload. Thus, LA function reservoir function cannot be seen as purely passive as a consequence of mitral plane movement towards the apex. This is further evidenced by the strong relationship found between PALS and the invasively estimated LV filling pressures, over and above other standard echo parameters.[7] Likewise, LA compliance which negatively correlates with severity of mitral regurgitation.[8]

According to the principles of physics, the term 'strain' describes the deformation of the tissues based on the relative displacement of the speckles, as a result of a force applied to the tissue. However, the direction of the deformation must be specified. Applying the LA physiology described above shows that changes happening to LA myocardial fibres in two opposite directions, i.e. active shortening in late diastole and stretching during LV systole, can make it ambiguous for both to be synonymously called 'strain'. Does LA 'strain' then reflect a deformation caused by external or internal forces? PACS involves electric

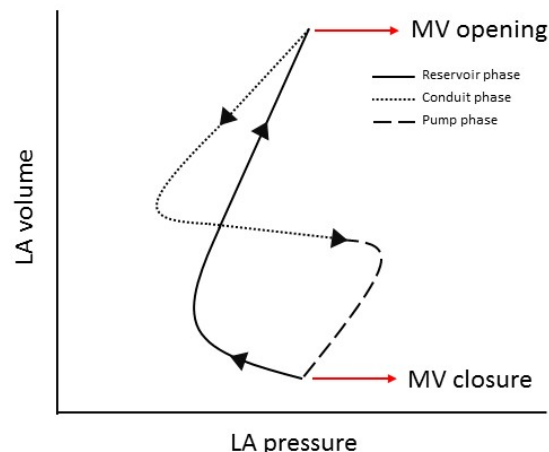


Figure 2. Evaluation of peak atrial longitudinal strain (PALS), at the end of left atrial reservoir phase and peak atrial contraction strain (PACS), the smaller peak before the atrial contraction. The dashed curve represents the averaged left atrial longitudinal strain value.

activation of LA myocardium, development of action potential, and ionic movements in and out of the myocytes; none of these electric-chemical-mechanical interactions occur during PALS. Furthermore, elastic properties of the LA do not require electric activation but are able to store potential energy that is converted to kinetic energy successively used in the early filling phase, a component that is lost in the presence of LA fibrosis. Thus, the basis of LA deformation during the two phases of diastole are quite different.

In view of the above it seems clear that PALS can not reflect absolute LA myocardial active function, but is a reflection of complex atrio-ventricular interaction in early diastole. If the echocardiography community elect to continue using 'Strain' as a reflection of myocardial contraction, as in the case of left and right ventricles, the scientific accuracy would be improved by replacing PALS by 'peak atrial longitudinal stretch', similar to LV longitudinal stretch during atrial systole i.e. late diastole.

Declarations of Interest

The authors declare no conflict of interest

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