

# Measurement of Changes in Left Ventricular Volume and Strain during Isovolumic Relaxation

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**Introduction:** Left ventricular (LV) active relaxation occurs predominantly during the isovolumic interval (IVR), and thus conventional volume provides little information regarding relaxation. However, during this period, changes have been measured in LV dimensions<sup>1,2</sup>, tissue mechanics<sup>3-5</sup>, and the energy associated with diastolic suction<sup>2,6</sup>. We hypothesize that LV volume defined by the endocardium and a plane at the base of the LV increases during IVR, with an associated change in the principal strains, which is conserved via the inward motion of the mitral leaflets toward the apex. The goals of this study are to measure LV volume changes based on muscle boundaries during IVR and to correlate these with changes in longitudinal, circumferential, and radial strain.

**Methods:** 10 healthy volunteers participated in this study. Images were acquired using a 1.5 T Siemens Sonata MRI scanner. LV volume was determined for all cardiac phases using disk summation of short axis SSFP cines. Long axis cines were used to identify the LV apex and base, allowing fractional inclusion of short axis slice volumes<sup>7,8</sup>. Typical sequence parameters were 1.5/3.0 ms TE/TR, 120×256 matrix, 65° flip angle, 248×400 mm<sup>2</sup> FOV, rate 2 parallel imaging, and 12 views per segment (VPS, 39 ms temporal resolution). LV deformation was determined using grid tagging for 5 short axis slices spanning the LV, and linear tags for long axis slice orientations. Typical sequence parameters were 2.8/4.6 ms TE/TR, 75×192 matrix, 14° flip angle, 212×400 mm<sup>2</sup> FOV, 5 VPS (23 ms temporal resolution), and reconstructed to 10 ms/cardiac phase. All slices were 8 mm thick and acquired during breath holds. Echocardiographic cines were also acquired to observe leaflet motion.

**Results:** Volume and global strain are shown over time in Figure 1. The changes in these values over IVR are listed in Table 1. During IVR, these parameters, measured at 10 ms intervals, were correlated with volume changes (Table 1) and shown for circumferential and longitudinal endocardial strain in Figure 2. LV volume was estimated from strain values by modeling the chamber as half of an ellipsoid and using endocardial circumferential and longitudinal strains to define changes in the ellipsoid's dimensions. This estimated volume is plotted against measured volume in the third panel in Figure 2. Cardiac ultrasound images confirmed leaflets descending towards the apex prior to their separation in all subjects.

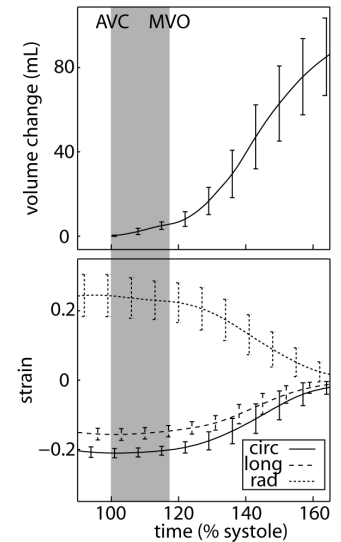


Figure 1. Average early filling time curves for volume and global principal strains.

Table 1. Changes during IVR

	Change	r
Volume (mL)	4.6 ± 1.5	
Circumferential strain (%)		
Global	0.87 ± 0.64	0.86*
Endocardial	0.98 ± 0.76	0.86*
Longitudinal strain (%)		
Global	0.93 ± 0.57	0.68*
Endocardial	0.77 ± 0.78	0.63*
Radial strain, global (%)	-1.46 ± 1.66	-0.37*

\* indicates p<0.01 correlation with volume change

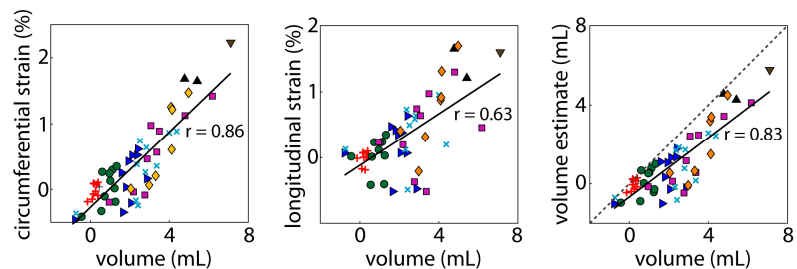


Figure 2. Changes in circumferential and longitudinal endocardial strain and LV volume estimated from endocardial strains are plotted against volume changes for 10 ms intervals starting from 5 ms after aortic valve closure until mitral valve opening. The following symbols indicate increasing time: + ● ► × ■ ◆ ▲ ▼.

**Conclusions:** While closed mitral and aortic valves ensure true isovolumic conditions during IVR, circumferential and longitudinal lengthening and radial shortening during this interval contribute to an increase in effective LV volume prior to mitral valve opening. The observed inward motion (towards the apex) of the mitral leaflets allows for conservation of volume. This leaflet motion and the conformational changes in the LV during isovolumic relaxation and likely reflect the LV pressure decline and the development of ventricular suction<sup>2,6</sup>.

**References:** 1. Ruttley MS et al. *Circulation* 1974;50(2):306-16. 2. Flewitt JA et al. *Am J Physiol Heart Circ Physiol* 2007;292(6):H2817-23. 3. Wang J et al. *Circulation* 2007;115(11):1376-83. 4. Notomi Y et al. *Am J Physiol Heart Circ Physiol* 2008;294(1):H505-13. 5. Sengupta PP et al. *JACC Cardiovasc Imaging* 2008;1(3):366-76. 6. Wang Z et al. *Am J Physiol Heart Circ Physiol*. 2005;288(4):H1641-51. 7. Kirschbaum SW et al. *Invest Radiol* 2008;43(1):1-6. 8. Thunberg P et al. *Clin Physiol Funct Imaging* 2008;28(4):222-8.