

New Concepts of Myocardial Mechanics in Hypertrophic Cardiomyopathy

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Background: Abnormalities of systolic (S) and diastolic (D) function in hypertrophic cardiomyopathy (HCM) are due to myocardial hypertrophy with myofiber disarray and fibrosis and are not easily defined by standard echo Doppler studies.

Methods: We studied 30 patients with HCM and 20 normal controls using a novel automated B-mode technique, velocity vector imaging (VVI, Siemens Medical Solutions) to track the endocardial border. We measured torsion time (TT), detorsion time (DTT), radial (RV) and twist (TV) velocities and twist angle (TA) at 3 short axis planes, base, mid and apex of the LV. Longitudinal myocardial strain (LS) and systolic (LS-S), and diastolic (LS-D) strain rate were measured at 3 septal and 3 lateral wall segments.

Results: Age, BSA, Doppler mitral and pulmonary inflow velocities were similar for the 2 groups. Left atrial (LA) volume index (40.2 vs. 24.2 cc, $p=0.0005$) and LA pressure (16.0 vs. 9.4, $p=0.04$) were higher in the HCM group. Systolic TV and torsion angle were similar at the base but greater at the mid level for HCM (0.60 ± 0.62 vs -0.29 ± -0.48 $p<0.0001$) and (1.83 ± -2.05 vs -1.51 ± -2.16 $p<0.0001$). Other differences are as follows:

	HCM	Normal	P Value
Septal Thickness	1.91 \pm 0.57	1.01 \pm 0.19	<0.0001
Apical Untwist Velocity	0.83 \pm 0.57	0.45 \pm 0.30	0.004
Radial Velocity S: base	2.03 \pm 0.70	1.62 \pm 0.49	0.01
Radial Velocity D: apex	-2.24 \pm 1.27	-1.73 \pm 0.56	0.05
Torsion (msec)	35.1 \pm 11.3	40.8 \pm 8.3	0.03
Detorsion (msec)	44.17 \pm 10.16	37.55 \pm 8.42	0.001
Longitudinal Strain (avg)	-16.7 \pm 4.32	-22.2 \pm 3.9	<0.0001
Longitudinal Strain Rate S	-0.86 \pm 0.27	-1.18 \pm 0.4	0.008
Longitudinal Strain Rate E	0.81 \pm 0.4	1.25 \pm 0.5	0.004

Conclusion:

1. Systolic abnormalities in HCM include increased basal radial velocity, apically displaced twist, more rapid torsion time and decreased longitudinal strain and strain rates.
2. Diastolic abnormalities include increased apical untwist, prolonged detorsion time, decreased diastolic strain rate and increased LA pressure.
3. These abnormalities provide further insights into the pathophysiology of HCM.