was measured from regional strain curves for each segment, as the time from the beginning of the Q-wave to the time-to-peak ϵ(sys) (TPS), mechanical dyssynchrony was estimated as TPS-SD. From 12-lead surface ECG, QT interval, QT dispersion, and its corrected values were measured. According to QT(c) (QT(c) ≥440 or <440 ms), patients were categorized into two groups: long QT and normal QT HCM. In the long QT group, QT(cd) and CT in all left ventricular (LV) segments were significantly prolonged; ϵ(sys) and SR(sys) were markedly attenuated compared with the other two groups (P<0.001). LV dyssynchrony was significantly greater (P<0.001) and PSS was more frequent in long QT HCM compared with the other two groups (P<0.001). TPS-SD was correlated positively with QT(c) (r=0.38, P<0.01) and QT(cd) (r=0.45, P<0.001). QT(c) ≥440 ms identified LV mechanical dyssynchrony with 70% sensitivity, 100% specificity, and positive predictive value. QT interval prolongation on surface ECG shows significant association with mechanical dyssynchrony and LV dysfunction in HCM. This may add pathophysiological insight into understanding ECG changes in such myocardial disease

P858
Aortic biomechanics in hypertrophic cardiomyopathy

M.F. Elnoamany[1], H.M. Badran[1], G.M. Soltan[1], N. Faheem[1], M. Yacoub[2].
[1]Menoufiya Faculty of Medicine, Shebeen El-Koom, Egypt; [2]Imperial College, London, United Kingdom

Background: Ventricular-vascular coupling is an important phenomenon in many cardiovascular diseases. The association between aortic mechanical dysfunction and left ventricular (LV) dysfunction is well characterized in many disease entities, but no data are available on how these changes are related in hypertrophic cardiomyopathy (HCM).
Aim of the work: This study examined whether HCM alone is associated with an impaired aortic mechanical function in patients without cardiovascular risk factors and the relation of these changes, if any, to LV deformation and cardiac phenotype.
Methods: 141 patients with HCM were recruited and compared to 66 age- and sex-matched healthy subjects as control group. Pulse pressure, aortic strain, stiffness and distensibility were calculated from the aortic diameters measured by M-mode echocardiography and blood pressure obtained by sphygmomanometer. Aortic wall systolic and diastolic velocities were measured using pulsed wave Doppler tissue imaging (DTI). Cardiac assessment included geometric parameters and myocardial deformation (strain and strain rate) and mechanical dyssynchrony.
Results: The pulsatile change in the aortic diameter, distensibility and aortic wall systolic velocity (AWS') were significantly decreased and aortic stiffness index was increased in HCM compared to control (P<0.001) In HCM AWS' was inversely correlated to age (r=-0.32, P<0.0001), MWT (r=-0.22, P<0.008), LVMI (r=-0.20, P<0.02), E/Ea (r=-0.16, P<0.03) LVOT gradient (r=-0.19, P<0.02) and severity of mitral regurg (r=-0.18, P<0.03) but not to the concealed LV deformation abnormalities or mechanical dyssynchrony. On multivariate analysis, the key determinant of aortic stiffness was LV mass index and LVOT obstruction while the role LV dysfunction in aortic stiffness is not evident in this population.
Conclusion: HCM is associated with abnormal aortic mechanical properties. The severity of cardiac phenotype, not LV deformation, is interrelated to aortic stiffness in patients with HCM. The increased aortic stiffness seems to be promising module that can be added as clinical risk parameter in HCM.

P859
The relationship between agatston calcium score and global longitudinal strain in patients suspected of stable angina pectoris

K.E. Frimodt-Moeller, F.J. Olsen, M.C. Hoejbjerg Lassen, K. Grundtvig Skaarup, J. Bech, J. Skov Jensen, T. Biering-Soerensen. *Gentofte University Hospital, Department of Cardiology, Copenhagen, Denmark*

Background: Cardiac computed tomography (CT) scan is often performed to evaluate coronary artery status in patients with stable angina pectoris (SAP). Echocardiography may assist the selection of patients in need of cardiac CT.
Purpose: To investigate the relationship between layer-specific global longitudinal strain (GLS) by speckle tracking echocardiography and Agatston calcium score.
Methods: In a clinical registry study of 592 patients suspected of SAP (mean age of 59 years, 43.6% male, mean body mass index (BMI) of 26, 15.5% with diabetes, 26.3% with hypertension) where everyone underwent both an echocardiogram and cardiac CT, GLS was measured using two-dimensional speckle-tracking echocardiography on the three apical views. The association between GLS and calcium score was examined with a restricted cubic spline model. Multivariable logistic regression was performed to relate echocardiographic measures to high calcium score (>400) and adjust for traditional clinical risk factors.
Results: GLS decreased incrementally with increasing tertile of calcium score (1st tertile: -19.6%; 2nd tertile: -19.2%; 3rd tertile: -18.4%, p for trend 0.0012) (figure 1). Of the 592 patients, 147 (24%) were classified as having a high calcium score. Mean GLS was -19.4% ± 0.15 in patients with low calcium score and -17.4% ± 0.5 in patients with high calcium score, p<0.001. GLS remained a significant independent predictor of high calcium score after adjustment for clinical risk factors being age, gender, hypertension, hypercholesterolemia, smoking, diabetes, BMI, family history of cardiovascular disease and heart rate (OR 1.09 [1.02; 1.16], p=0.01).

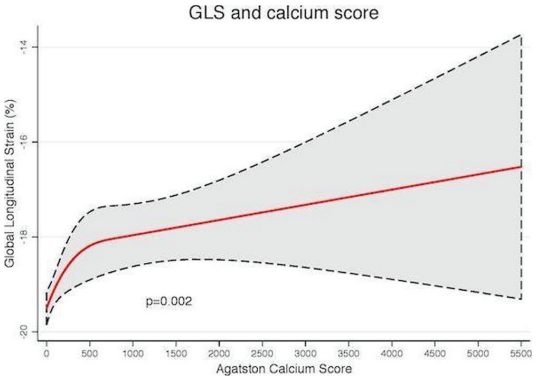

Figure 1

Conclusion: In patients suspected of having SAP, GLS becomes incrementally more impaired with increasing Agatston calcium score and remains an independent predictor of high calcium score. Performing an echocardiogram may aid in the selection of those with high calcium score and thereby assist in identifying those in need of invasive coronary angiography.

LEFT VENTRICULAR MECHANICS

P860
Left ventricular layer torsion in heart failure with preserved versus reduced ejection fraction assessed by one-beat real-time 3-dimensional speckle tracking echocardiography with high volume rate

S. Minatoguchi[1], T. Yoshizane[2], R. Tanaka[3], T. Watanabe[1], K. Ono[2], M. Saeki[2], M. Nagaya[2], H. Yagasaki[2], T. Kojima[2], M. Iwama[2], T. Noda[2], H. Ohashi[3], S. Watanabe[2], S. Minatoguchi[1], M. Kawasaki[1]. *[1]Gifu University Graduate School of Medicine, Department of Cardiology, Gifu, Japan; [2]Gifu Prefectural General Medical Center, Department of Cardiology, Gifu, Japan; [3]Murakami Memorial Hospital, Department of Cardiology, Gifu, Japan*

Background: Left ventricular (LV) remodeling caused by pressure overload may lead to heart failure (HF). LV is composed of 3 myocardial layers and torsion caused by inner and outer oblique muscle contraction plays an important role in squeezing blood out of the heart and ejection fraction (EF). LV outer muscle plays a predominant role in torsion. However, the difference in LV torsion between HF with preserved EF (HFpEF) and reduced EF <50% (HFrEF) and was not elucidated and measurement of LV torsion by echocardiography has been methodologically challenging.
Purpose: The aim is to examine the impact of LV deformational parameters in myocardial layers and LV size on LVEF in HF using real-time one-beat 3-dimensional speckle tracking echocardiography with high volume rates (3D-STE) and elucidate the difference in torsion between HFrEF and HFpEF.
Methods: LV strain and strain rate during systole (SR) in 3 directions (longitudinal, radial and circumferential), and twist and torsion at 3 layers (endocardium, mid-wall and epicardium) were assessed in 160 subjects {58 controls (67±11 years) and 40 HFpEF (77±12 years) and 62 HFrEF (68±16 years)} by 3D-STE. LV twist was defined as a difference between apical and basal rotation and torsion was defined as twist divided by long axis length for every instant in time. Significant valvular disease, prior cardiac surgery, coronary artery disease, irregular rhythm and Diabetes Mellitus were excluded.
Results: LV endo-diastolic dimension increased in HFrEF (control: 45±4, HFpEF: 45±5, HFrEF: 56±7*mm, *p<0.05 vs control). LVEF in HFrEF was associated with LV dilation, decreased strain and SR at all 3 layers and in all 3 directions, and decreased torsion (LVEF; control; 67±6, HFpEF: 64±9, HFrEF: 38±9*%, longitudinal strain and SR-S at endocardium: -19±3, -15±6*, -11±4*, and -1.5±0.6, -1.4±0.7, -1.2±0.4* s^{-1}, longitudinal strain and SR at epicardium: -11±3, -8±3*, -6±2* and -0.5±0.2, -0.4±-0.3, -0.3±-0.2* s^{-1}, torsion at endocardium: 2.5±0.4, 2.2±0.5, 2.0±0.4*, torsion at epicardium: 0.50±0.24, 0.53±0.30, 0.35±0.15*, global torsion: 1.4±0.2, 1.3±0.3, 1.1±0.2*°/cm). Twist at all 3 layers in HFrEF was not reduced compared to control (epicardium; 2.9±1.6, 3.8±2.5, 3.1±1.3°). Longitudinal and radial strain at all layers reduced in HFpEF (radial strain at endocardium; 36±13, 30±9*, 20±7*), but circumferential strain at epicardium in HFpEF were comparable to controls (-10±4, -9±3, -6±3*) associated with preserved torsion at epicardium.
Conclusions: LV torsion at epicardium was preserved in HFpEF, whereas that in HFrEF was decreased accompanied with LV dilation and reduced LV strain and SR in all directions. Torsion may represent a compensatory mechanism to maintain LVEF, suggesting that LV dilation and deterioration of torsion caused by insult of outer oblique muscle may lead to HFrEF.