A simplified left ventricular end-diastolic mean wall thickness-to-volume ratio estimated from left ventricular mass and end-diastolic volume distinguishes physiological from pathological hypertrophy

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BACKGROUND

Cardiovascular magnetic resonance (CMR) accurately measures left ventricular (LV) end-diastolic (ED) volume (EDV) and LV mass (LVM), but LVED mean wall thickness (LVEDMWT) is not routinely measured clinically.

We sought to (1) derive and validate a simplified measure of LVEDMWT estimated from LVEDV and LVM by CMR, and (2) evaluate the ability of a thickness-to-volume ratio (TVR) to distinguish pathological from physiological hypertrophy.

METHODS

Patients underwent LV cine CMR imaging at 1.5T. LV epicardial and endocardial borders were manually delineated in all slices in a full coverage LV short-axis cine stack in end diastole and end systole. An in-house developed algorithm measured the LVED wall thickness at 24 equally circumferentially distributed positions per slice, excluding regions with thickness <2 mm, and averaged over the whole LV with weighting according to slice circumference.

Based on geometrical assumptions of the relationship mass and volume upon wall thickness, the formula LVEDMWT[mm]=a+b*LVM[g]^x*LVEDV[ml]^y was optimized iteratively compared to measured LVEDMWT. TVR was calculated as TVR[dimensionless]=LVEDMWT[mm]/LVEDV indexed to body surface area [ml/m^2]*1000.

RESULTS

A validation-derivation cohort (*n*=537) was comprised of volunteers, endurance athletes and patients with varying pathologies. In a derivation subset (*n*=269/537), the best fit formula was: estimated LVEDMWT[mm]= 0.050+1.60*LVM[g]^0.837*EDV[ml]^-0.487. In a separate validation subset (*n*=268/537), estimated LVEDMWT agreed with measured LVEDMWT (R2=0.95, $p < 0.001$, mean±SD bias 0.01±0.23 mm), Figure 1.

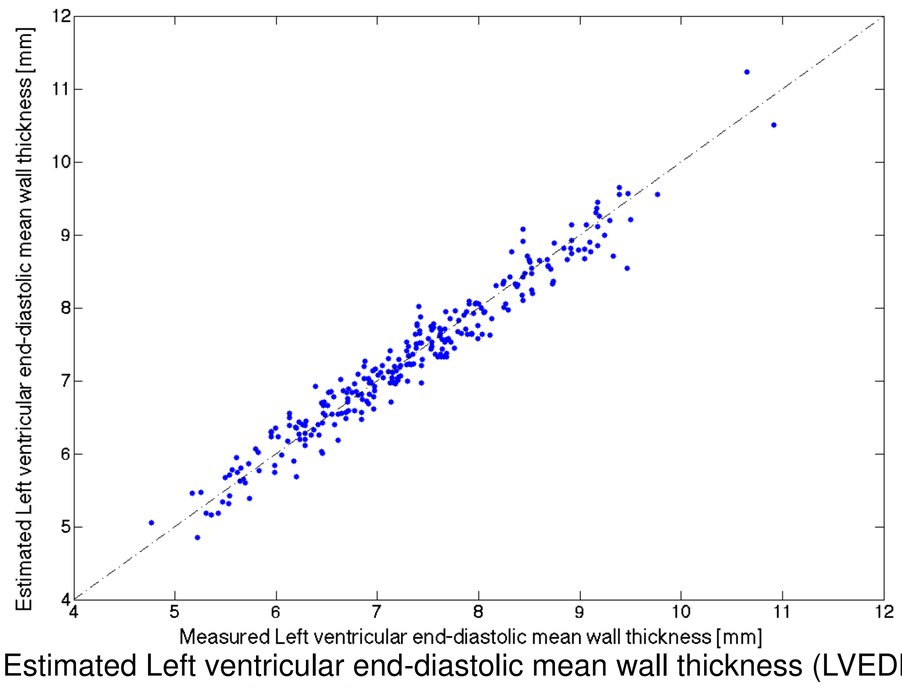

Figure 1. Estimated Left ventricular end-diastolic mean wall thickness (LVEDMWT) plotted versus measured LVEDMWT for the validation subset of the cohort (*n*=268/537), R^2=0.95, $p<0.001$, mean±SD bias 0.01±0.23 mm, identity line shown.

RESULTS (continued)

In a largely overlapping cohort enriched with healthy volunteers and hypertrophic cardiomyopathy (HCM), healthy volunteers (*n*=56) yielded 95% confidence intervals for LVEDMWT: 5.63-8.67 mm (men) and 4.47-7.42 mm (women), and for TVR: 58.4-104.4 (men) and 51.9-97.8 (women). Compared to healthy volunteers, gender-specific estimated LVEDMWT was higher in both athletes (*n*=86) and in patient groups including cardiac resynchronization therapy candidates (CRT, *n*=35), acute myocardial infarction (AMI, *n*=300), cardiac syndrome X (CSX, *n*=39), and HCM (*n*=26), $p < 0.05$ for all groups, Figure 2. In contrast, gender-specific TVR was lower in athletes (all athletes had TVR < 95) and CRT, and higher in CSX and HCM compared to normals, $p < 0.05$ for all groups. For AMI, women had higher TVR than normals (p=0.002) whereas there was no difference for men (p=0.24), Figure 3.

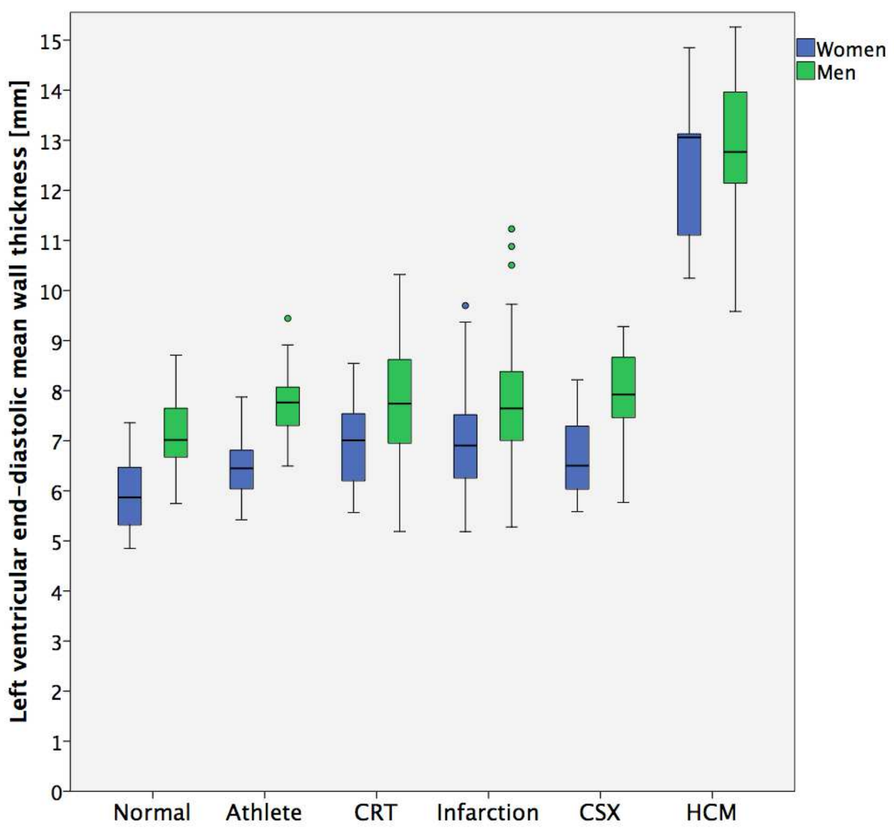

Figure 2. The distribution of estimated left ventricular end-diastolic mean wall thickness (LVEDMWT) in mm for normal healthy controls, endurance athletes, cardiac resynchronization therapy (CRT) candidates, patients with myocardial infarction, patients with cardiac syndrome X (CSX) and patients with hypertrophic cardiomyopathy (HCM). All groups had LVEDMWT greater than gender-specific normals (p<0.05).

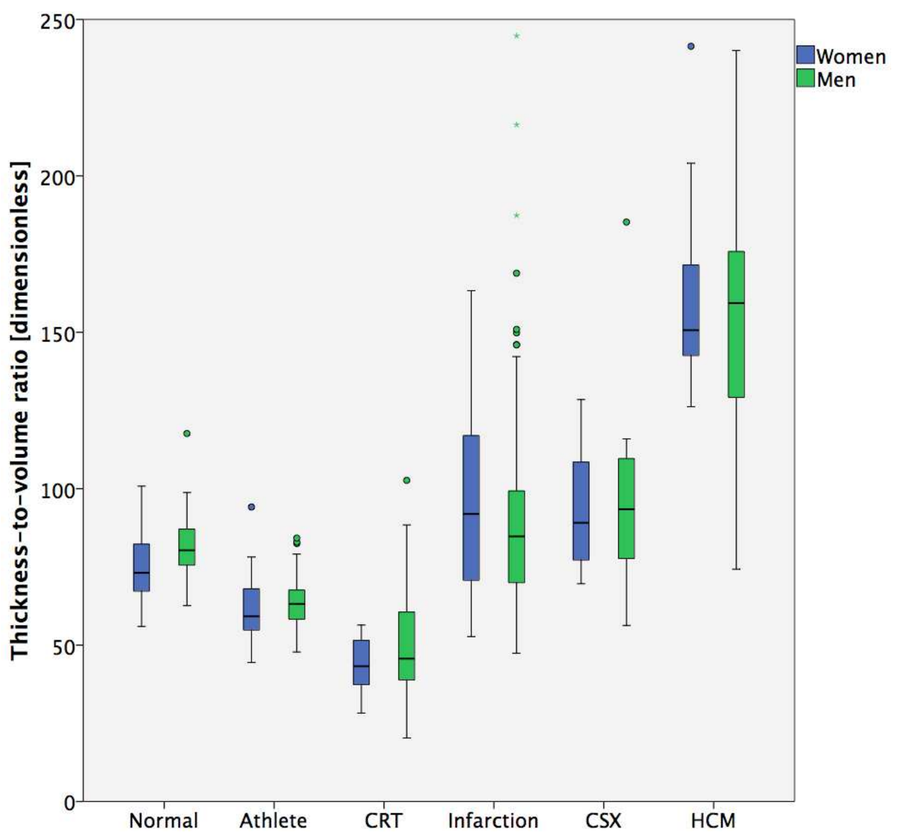

Figure 3. The distribution of the thickness-to-volume ratio (TVR, dimensionless) for normal healthy controls, endurance athletes, cardiac resynchronization therapy (CRT) candidates, patients with acute myocardial infarction, patients with cardiac syndrome X (CSX) and patients with hypertrophic cardiomyopathy (HCM). All groups had a TVR that differed from gender-specific normals (p<0.05) except males with infarction.

CONCLUSIONS

LVEDMWT can be simply estimated from LVM and LVEDV with high accuracy and precision. Estimated LVEDMWT can in turn be used to calculate TVR as a new index of relative wall thickness. The maximum TVR found in endurance athletes was lower than the upper limit for healthy normals. Therefore, increased TVR effectively rules out athlete's heart as an alternative diagnosis in patients with increased wall thickness.

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