Myocardial Contraction Fraction: A Volumetric Index of Myocardial Shortening by Freehand Three-Dimensional Echocardiography

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OBJECTIVES This study sought to evaluate myocardial contraction fraction (MCF) as an index of myocardial shortening by comparison to conventional shortening indices in patients with hypertensive hypertrophy, athletes with physiologic hypertrophy and sedentary normal adult subjects.

BACKGROUND A significant percentage of patients with hypertensive hypertrophy have “normal” or “preserved” left ventricular (LV) systolic function by conventional echocardiographic measures whereas their systolic function is depressed when measured by the two-dimensional echocardiographic mid-wall shortening fraction (MWSF). A three-dimensional echocardiographic measure of myocardial shortening analogous to MWSF has been lacking. We describe a volumetric measure of myocardial shortening, the MCF, as the ratio of stroke volume (SV) to myocardial volume (MV), and hypothesize that it may be useful to compare myocardial performance in patients with different degrees and types of hypertrophy.

METHODS We compared the MCF using freehand three-dimensional echocardiographic reconstruction of the LV to conventional measures of LV function (ejection fraction [EF], endocardial shortening fraction [SF] and MWSF) in subjects with pathologic hypertensive hypertrophy, heart failure symptoms and preserved EF (n = 17), athletes with physiologic hypertrophy (n = 41) and normal sedentary adults (n = 80).

RESULTS The EF was in the normal range for all three groups. The MCF was lower in hypertensive hypertrophy compared with normal subjects (0.33 ± 0.05 vs. 0.44 ± 0.07, p < 0.01). The endocardial SF did not distinguish athletes from normal subjects and the MWSF did not distinguish hypertensive from physiologic hypertrophy. The MCF may be useful in assessing differences in myocardial performance in patients with similar degrees of hypertrophy.

CONCLUSIONS The MCF, a volumetric measure of myocardial shortening, demonstrates that myocardial shortening is decreased in hypertensive hypertrophy and increased in physiologic hypertrophy. The MCF may be useful in assessing differences in myocardial performance in patients with similar degrees of hypertrophy.

Assessment of ventricular function in patients with concentric hypertrophy may be problematic. Many patients with hypertrophy and heart failure (HF) symptoms may have “normal” or “preserved” left ventricular (LV) systolic function by conventional measures (1). Endocardial measures of ventricular function (shortening fraction [SF] or ejection fraction [EF]) may demonstrate normal or enhanced function (2,3) while experimental (4–8) and clinical studies using a mid-wall measure of ventricular function (mid-wall shortening fraction [MWSF]) have demonstrated depressed myocardial performance (9–13). Although three-dimensional echocardiography yields highly accurate volumes and EF (14–16), a three-dimensional measure of myocardial shortening analogous to the two-dimensional MWSF has not been described. To address this, we describe a three-dimensional, volumetric measure of myocardial shortening, the myocardial contraction fraction (MCF), and compare it with two-dimensional echocardiographic measures of ventricular performance, the endocardial SF and the MWSF, as well as the three-dimensional EF in normal subjects, patients with hypertensive hypertrophy and HF and subjects with physiologic hypertrophy.

Stroke volume (SV) is a measure of ventricular performance integrating the influence of all factors affecting the ventricle: preload, afterload, contractility and geometry. When it is assessed relative to end-diastolic pressure, fiber length or volume, or a related parameter, ventricular function is described (17). As a measure of myocardial shortening, SV is most appropriately assessed relative to the myocardium, and specifically to myocardial volume (MV), because it is the myocardium that shortens. Thus, the MCF, defined as the ratio of SV to MV (MCF = SV/MV), is a measure of ventricular function. In this ratio, SV is a measure of the amount by which the myocardium contracts (i.e., shortens) during systole relative to the total MV, although the myocardium itself has not undergone a reduction in volume (18). During systole the myocardium shortens and thickens, reducing its contained volume by the amount of the SV. Therefore, the SV is a measure of the amount of shortening and thickening that has occurred, and its ratio to MV is an index of the fractional shortening of the myocardium in volumetric terms.
Because this ratio is independent of ventricular geometry, we hypothesize that it may be a useful measure of myocardial performance analogous to the two-dimensional MWSF and may be better able to delineate differences in ventricular function in patients with different degrees and types of hypertrophy than conventional measures. To test this hypothesis, we compared the MCF obtained by freehand three-dimensional echocardiographic reconstruction of the LV with conventional two-dimensional echocardiographic measures of LV function in: 1) subjects with hypertensive hypertrophy, HF symptoms and preserved EF; 2) sedentary normal young to elderly adult male and female subjects; and 3) athletes with physiologic hypertrophy.

METHODS

Study subjects. All subjects were examined according to institutional review board-approved protocols and gave informed consent. The two groups of subjects with hypertrophy were: 1) healthy young adult endurance athletes (n = 41); and 2) patients with hypertension, left ventricular hypertrophy, previous symptoms of HF and normal or “preserved” EFs (n = 18). Endurance athletes were recruited from a pool of subjects undergoing self-directed programs in physical training. The athletes had a high level of physical condition (mean maximal oxygen consumption [Vo_{2max}] ± SD was 50.6 ± 11.9 ml/kg/min) and no history or clinical evidence of cardiac disease. The patients with hypertensive hypertrophy HF were recruited from patients being seen for clinical symptoms. The hypertensive patients had increased LV wall thickness with preserved EF and clinical evidence of HF, defined by a reduction in functional capacity with either fatigue or shortness of breath with exertion. All these patients met criteria for diastolic HF as defined by the European Society of Cardiology (19). The mean Vo_{2max} for these patients with hypertensive hypertrophy was 19.3 ± 3.5 ml/kg/min. The third group included normal sedentary young to elderly adult male and female subjects without evidence of cardiovascular disease by history, physical examination or echocardiography (n = 80).

Echocardiography. A standard two-dimensional echocardiographic examination was performed on each subject before the three-dimensional echo data acquisition. The same echocardiograph was used for both examinations. The ventricular minor axis dimension and posterior wall thickness were measured on the parasternal long axis view at a position 1 cm below the tips of the mitral leaflets on three independent beats. The measurements were averaged and used to calculate endocardial SF and MWSF. The modified ellipsoid two-shell model taking into account relative epicardial migration of the mid-wall circumferential fibers with systole was used to determine h ′, the thickness of the inner myocardial shell at end-systole (20). Endocardial SF was calculated using the formula:

\[ SF = (D - S)/D \]

Mid-wall shortening fraction was then calculated using the formula:

\[ MWSF = ((D + 2h) - (S + 2h'))/(D + 2h) \]

where D = end-diastolic chamber dimension, h = end-diastolic posterior wall thickness/2, S = end-systolic chamber dimension and h ′ = calculated thickness of the inner myocardial shell at end-systole.

The equipment and procedures of freehand three-dimensional echocardiography have been previously described in detail (21–24). Briefly, the equipment consists of a conventional real-time echocardiograph, a three-dimensional acoustic spatial locator, a personal computer and custom software. Images acquired from the real-time scanner are digitized and saved in the computer along with their corresponding spatial coordinates. For quantitative ventriculography a series of 11 short axis video loops of the ventricle are acquired. These loops include end-diastole and end-systole, and span the ventricle from the inferior surface of the aortic valve to the epicardial apex about one centimeter apart. Off-line the endocardial and epicardial boundaries of the images are traced in systole and diastole. From these traced boundaries, four ventricular surfaces—endocardial and epicardial at end-diastole and end-systole—are reconstructed and their volume and surface area computed. One complete data set was acquired for each subject and analyzed immediately after acquisition. These data provide ventricular chamber volume, MV, SV and the ratios derived from these, EF and MCF. The analysis of each ventricular reconstruction was performed by a single, highly skilled cardiac sonographer (L. E-K. C.) who was blinded during the course of acquisition and analysis of the data to the concept of the MCF and the project to validate it.

Statistics. The differences between the groups for each parameter, EF, endocardial SF, MCF and MWSF, were compared using analysis of variance with Dunnett’s test to determine significant differences from normal. Results are expressed as mean ± SD. A p value < 0.05 was considered statistically significant. SAS for Windows (Version 8.0, SAS Institute Inc., Cary, North Carolina) was used for all analyses.
The primary finding of this study is that the MCF, a three-dimensional volumetric measure of myocardial shortening, most clearly distinguishes the three groups whereas conventional shortening parameters—EF, endocardial SF and MWSF—do not. The MCF is increased in subjects with physiologic hypertrophy (endurance athletes), reflecting a relatively greater increase in SV than MV, and the MCF is decreased in hypertensive hypertrophy, reflecting the converse. The endocardial SF is increased in concentric hypertrophy, due to geometric changes in the ventricle, but does not differentiate physiologic hypertrophy from sedentary normal subjects. The MWSF is not significantly different among the three groups. The lack of statistically significant difference may be due to either variability of the two-dimensional measurement, small sample size, or the superimposition of clinical HF in the group with hypertensive hypertrophy. Thus, the MCF measures myocardial shortening in a manner qualitatively similar to the MWSF and appears to be more useful comparing and differentiating physiologic hypertrophy from hypertensive hypertrophy than conventional measures. Its utility for more general application in other groups of patients requires further study.

**Influence of chamber volume.** The MCF incorporates only SV in the numerator and MV in the denominator, thus removing the geometric influence of chamber volume and wall thickness from the shortening expression. Each of the conventional indices of ventricular function—EF, SF and MWSF—inorporates the end-diastolic chamber dimension or volume in its calculation. In the case of the MWSF, the dimensions used are the sum of the ventricular dimension plus one-half the wall dimension. As a consequence, assessment of shortening by these parameters is fully or partially influenced by volume. By eliminating chamber volume from the shortening assessment, the MCF expresses the shortening relationship only in terms of that which shortens, the myocardium.

**Myocardial shortening abnormality.** A decrease in the MCF indicates abnormal myocardial shortening induced either by hypertrophy or by intrinsic myocardial disease that reduces SV. Geometric changes in hypertensive hypertrophy have been shown to mask a generalized myocardial shortening abnormality not apparent when endocardial measures of chamber function are employed (25). The MWSF studies have identified reduced function in some but not all hypertrophied subjects (11). The MWSF represents the contraction of the middle, circumferential layer of myocardial fibers at the base of the ventricle. It is not representative of all myocardial contraction because there is significant heterogeneity of myocardial fiber shortening not only from epicardium to endocardium, but also from apex to base. The MCF confirms the findings of studies using spatially modulated magnetic resonance imaging (MRI) that show a generalized myocardial shortening abnormality with hypertrophy (26). The latter studies show depressed

### Table 1. Clinical Characteristics of Study Groups

<table>
<thead>
<tr>
<th></th>
<th>Sedentary Normals (n = 80)</th>
<th>Adult Athletes (n = 41)</th>
<th>Hypertensive Hypertrophy (n = 18)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>47 ± 21</td>
<td>31 ± 4</td>
<td>59 ± 12</td>
</tr>
<tr>
<td>Gender (male/female)</td>
<td>37/43</td>
<td>30/11</td>
<td>9/8</td>
</tr>
<tr>
<td>Systolic blood pressure (mm Hg)</td>
<td>132 ± 12</td>
<td>113 ± 8</td>
<td>161 ± 19</td>
</tr>
<tr>
<td>Diastolic blood pressure (mm Hg)</td>
<td>78 ± 8</td>
<td>73 ± 8</td>
<td>93 ± 16</td>
</tr>
<tr>
<td>Heart rate</td>
<td>66 ± 10</td>
<td>61 ± 10</td>
<td>69 ± 7</td>
</tr>
</tbody>
</table>

Results expressed as mean ± SD where applicable.

### DISCUSSION

The primary finding of this study is that the MCF, a three-dimensional volumetric measure of myocardial shortening, most clearly distinguishes the three groups whereas conventional shortening parameters—EF, endocardial SF and MWSF—do not. The MCF is increased in subjects with physiologic hypertrophy (endurance athletes), reflecting a relatively greater increase in SV than MV, and the MCF is decreased in hypertensive hypertrophy, reflecting the converse. The endocardial SF is increased in concentric hypertrophy, due to geometric changes in the ventricle, but does not differentiate physiologic hypertrophy from sedentary normal subjects. The MWSF is not significantly different among the three groups. The lack of statistically significant difference may be due to either variability of the two-dimensional measurement, small sample size, or the superimposition of clinical HF in the group with hypertensive hypertrophy. Thus, the MCF measures myocardial shortening in a manner qualitatively similar to the MWSF and appears to be more useful comparing and differentiating physiologic hypertrophy from hypertensive hypertrophy than conventional measures. Its utility for more general application in other groups of patients requires further study.

### Table 2. Echocardiographic Characteristics of Study Groups

<table>
<thead>
<tr>
<th></th>
<th>Sedentary Normals (Mean ± SD)</th>
<th>Adult Athletes (Mean ± SD)</th>
<th>Hypertensive Hypertrophy (Mean ± SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVDd (cm)</td>
<td>4.8 ± 0.6</td>
<td>5.0 ± 0.5</td>
<td>5.0 ± 0.7</td>
</tr>
<tr>
<td>LVDs (cm)</td>
<td>3.2 ± 0.5</td>
<td>3.4 ± 0.5</td>
<td>3.1 ± 0.6</td>
</tr>
<tr>
<td>PWT (cm)</td>
<td>1.0 ± 0.1</td>
<td>0.97 ± 0.12</td>
<td>1.42 ± 0.3</td>
</tr>
<tr>
<td>RWT</td>
<td>0.42 ± 0.07</td>
<td>0.39 ± 0.05</td>
<td>0.58 ± 0.15</td>
</tr>
<tr>
<td>3D EDV (ml)</td>
<td>91.6 ± 20.9</td>
<td>144.3 ± 26.2</td>
<td>130.7 ± 40.9</td>
</tr>
<tr>
<td>3D ESV (ml)</td>
<td>38.8 ± 9.9</td>
<td>65.1 ± 12.7</td>
<td>62.2 ± 22.4</td>
</tr>
<tr>
<td>3D MV (ml)</td>
<td>52.8 ± 12.1</td>
<td>79.5 ± 14.6</td>
<td>68.1 ± 20.3</td>
</tr>
<tr>
<td>3D LVMI (g/m²)</td>
<td>120.8 ± 28.5</td>
<td>159.9 ± 23.1</td>
<td>208.1 ± 64.1</td>
</tr>
</tbody>
</table>

LVDd (LVDs) = left ventricular end-diastolic (end-systolic) endocardial dimension; PWT = posterior wall thickness; RWT = two-dimensional echo relative wall thickness = 2 x posterior wall thickness/LVDd; 3D EDV = three-dimensional echo end-diastolic volume; 3D ESV = three-dimensional echo end-systolic volume; 3D LVMI = three-dimensional echo left ventricular mass in gramsindexed to height in meters raised to the power 2.4; 3D MV = three-dimensional echo myocardial volume; 3D SV = three-dimensional echo stroke volume.
longitudinal as well as circumferential myocardial shortening in hypertensive hypertrophy (27).

**Distinguishing physiologic from pathologic hypertrophy.** The differentiation of physiologic from pathologic hypertrophy has important clinical implications (28) and is particularly difficult when distinguishing athletes from subjects with mild forms of hypertrophic cardiomyopathy (29,30). Previously proposed criteria include resting and/or post-exercise LV diastolic dysfunction (31), a ventricular wall thickness >16 mm (32), and more recently, tissue Doppler assessment of diastolic function (33) and metabolic exercise stress testing (34,35). In our study, the MCF, along with peak VO₂, differentiated subjects with hypertensive hypertrophy and HF from endurance athletes with hypertrophy. The athletes in the present study were endurance athletes, and our experience with this group may not apply to athletes who participate primarily in strength training. Further study of the MCF in subjects with hypertrophic cardiomyopathy is needed.

**Advantages.** The MCF has a clear, simple, easily understood definition. As a dimensionless index, analogous to the EF, it easily permits comparison of myocardial shortening between subjects. As an additional attribute, it may be easily calculated by any three-dimensional tomographic technique, including MRI, computed tomography and three-dimensional echocardiography. However, the MCF requires accurate measurement of SV and MV, and its use may be restricted to three-dimensional techniques. Estimates of SV and MV obtained by two-dimensional techniques and Doppler echocardiography probably are neither sufficiently accurate nor reproducible to be comparable to three-dimensional results.

Three-dimensional tomographic reconstruction of the ventricle, whether by echocardiography, MRI or computed tomography, is the most accurate and reproducible method for obtaining chamber and MV at the present time (16). We believe this accuracy and reproducibility are essential for calculation of a useful MCF. Three-dimensional echocardiography and MRI have been shown to yield equivalent results that are superior to m-mode and two-dimensional echocardiographic techniques (36–39). The superior accuracy of three-dimensional tomographic reconstruction is based on several characteristics. First, three-dimensional methods avoid use of geometric assumptions by measuring an additional spatial dimension. Second, in freehand three-dimensional echocardiography, visual guidance of image plane location decreases errors of image plane position.

Third, three-dimensional methods decrease sampling errors by increasing about fivefold the number of images used to represent the ventricle (40).

**Study limitations.** The results of this study are limited to the groups of subjects studied. This study does not encompass the full range of ventricular diseases encountered clinically. For example, the MCF may not accurately reflect myocardial function where shortening (SV) is affected by valve disease. In the presence of regional disease, such as coronary artery disease, the MCF, as a global parameter, will reflect net myocardial shortening.

**Conclusions.** The MCF is a useful measure for assessing myocardial shortening because it is independent of chamber size whereas conventional measures are not. As a volumetric measure of myocardial shortening, it parallels the two-dimensional MWSF, confirming that myocardial shortening is decreased in hypertensive hypertrophy and increased in physiologic hypertrophy. The MCF may be useful in assessing differences in myocardial function in patients with similar degrees of hypertrophy.

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**Table 3. Comparison of Ventricular and Myocardial Function Indices**

<table>
<thead>
<tr>
<th></th>
<th>EF (Mean ± SD)</th>
<th>SF (Mean ± SD)</th>
<th>MCF (Mean ± SD)</th>
<th>MWSF (Mean ± SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sedentary normals</td>
<td>0.58 ± 0.04</td>
<td>0.32 ± 0.07</td>
<td>0.44 ± 0.07</td>
<td>0.20 ± 0.05</td>
</tr>
<tr>
<td>Adult athletes</td>
<td>0.55 ± 0.03*</td>
<td>0.33 ± 0.08†</td>
<td>0.50 ± 0.05*</td>
<td>0.21 ± 0.05†</td>
</tr>
<tr>
<td>Hypertensive hypertrophy</td>
<td>0.53 ± 0.04*</td>
<td>0.38 ± 0.08*</td>
<td>0.33 ± 0.04*</td>
<td>0.21 ± 0.05†</td>
</tr>
</tbody>
</table>

*p < 0.05 compared with normals; †p = NS compared with normals.

**EF = ejection fraction; MCF = myocardial contraction fraction; MWSF = mid-wall shortening fraction; SF = endocardial shortening fraction.**

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**REFERENCES**


