Short-axis epicardial volume change is a measure of cardiac left ventricular short-axis function, which is independent of myocardial wall thickness

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Ugander M, Carlsson M, Arheden H. Short-axis epicardial volume change is a measure of cardiac left ventricular short-axis function, which is independent of myocardial wall thickness. Am J Physiol Heart Circ Physiol 298: H530–H535, 2010. First published November 20, 2009; doi:10.1152/ajpheart.00153.2009.—Fractional shortening (FS) by echocardiography is considered to represent the short-axis contribution to the stroke volume (SV), also called short-axis function. However, FS is mathematically coupled to the amount of myocardium, since it rearranges during atrioventricular plane displacement (AVPD). The SV is the sum of the volumes generated by 1) reduction in outer volume of the heart, and 2) inner AVPD. The long-axis contribution to the SV is generated by AVPD, and thus the short-axis contribution is the remaining outer volume change of the heart, which should be unrelated to myocardial wall thickness. We hypothesized that both endocardial and midwall shortening indexed to SV are dependent on myocardial wall thickness, whereas epicardial volume change (EVC) indexed to SV is not. Twelve healthy volunteers (normals), 12 athletes, and 12 patients with dilated cardiomyopathy (ejection fraction <30%) underwent cine cardiac magnetic resonance imaging. Left ventricular long-axis function was measured as the portion of the SV, in milliliters, generated by AVPD. EVC was defined as SV minus long-axis function. Endocardial and midwall shortening were measured in a midventricular short-axis slice. Endocardial shortening/SV and midwall shortening/SV both varied in relation to end-diastolic myocardial wall thickness ($R^2=0.16$, $P=0.008$ and $R^2=0.14$, $P=0.012$, respectively), whereas EVC/SV did not ($R^2=0.00$, $P=0.37$). FS is dependent on myocardial wall thickness, whereas EVC is not and therefore represents true short-axis function. This is not surprising considering that FS is mainly caused by rearrangement of myocardium secondary to long-axis function. FS is therefore not synonymous with short-axis function.

cardiac short-axis function; echocardiography; fractional shortening; left ventricle

THE END PRODUCT OF CARDIAC pumping is the volume of blood ejected during one cardiac cycle, the stroke volume. The stroke volume can be seen as the sum of two parts, namely the volumes of blood generated by long- and short-axis function. We have previously shown that 1) long-axis function corresponds to the volume of blood generated by atrioventricular plane displacement (AVPD), 2) short-axis function corresponds to the volume of blood generated by the epicardial volume change (EVC) of the heart, and 3) the sum of these two volumes equals the stroke volume (5, 6). Using these definitions, the normal heart has been shown to have a left ventricular stroke volume, which is generated by 60% long-axis function and 40% short-axis function, whereas the right ventricular stroke volume is generated by 80% long-axis function and 20% short-axis function (5).

In echocardiography, long-axis function has been quantified in long-axis images as the movement of the atrioventricular plane during systole, also called AVPD (9). By analogy, short-axis function has been quantified as fractional shortening (FS) and measured as the decrease in endocardial diameter in the short-axis plane (10, 15). Moreover, endocardial FS has been found to be influenced by myocardial hypertrophy, and thus midwall FS has been proposed as a measure of short-axis function, which is less influenced by myocardial hypertrophy (2, 7). The measures involved in calculating both endocardial and midwall shortening and FS as well as the EVC are illustrated in Fig. 1. Importantly, absolute shortening or FS employing measurement of the endocardial or midmural inner diameter of the left ventricle are currently used to assess short-axis function during both systole (13, 17, 20) and diastole (14) and to compare the relationship between long- and short-axis function (1, 3, 4, 16, 19).

However, both endocardial and midmural FS are measures that are mathematically coupled to long-axis function through rearrangement of myocardium, which occurs when the atrioventricular plane moves toward the apex during systole. Thus both endocardial and midmural FS appear to be measures of short-axis function that also are influenced by the amount of myocardium (Fig. 2). Yet, short-axis function can be measured as EVC, which, theoretically, is not influenced by the amount of myocardium.

We hypothesized that both endocardial and midwall shortening are dependent on myocardial wall thickness, whereas the short-axis EVC is not. The current study sought to address this hypothesis by measuring both left ventricular endocardial and midmural shortening as well as EVC and analyzing how these measures relate to myocardial wall thickness measured by cardiovascular magnetic resonance (CMR) imaging. Furthermore, we sought to assess the relationship between long- and short-axis function by CMR.

METHODS

Study population. The study was approved by the local committee on human research ethics. Healthy volunteers (normals) and athletes provided written informed consent, and the local ethics committee provided a waiver of written informed consent for retrospective inclusion of patients. We studied 12 healthy volunteers, 12 Swedish national elite triathletes, and 12 patients with dilated cardiomyopathy (ejection fraction <30%). We have studied this population previously (6).

CMR imaging. All subjects underwent CMR imaging in the supine position. Imaging was undertaken with a 1.5T scanner (Intera; Philips, Best, The Netherlands) using a five-element cardiac synergy coil and a cine steady-state free precession sequence as previously described (6). Cine image acquisition included a contiguous short-axis stack and also the two-chamber, four-chamber, and left ventricular outflow tract...

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long-axis views. In short, spatial resolution was typically $1.4 \times 1.4 \times 8$ mm, and temporal resolution was 30 ms per image.

**Image analysis.** Left ventricular stroke volume was determined by manual planimetry in short-axis slices (11). AVPD was measured as the mean of six measurements, namely, one measurement at each of the two atrioventricular valve plane positions per image in the two-chamber, four-chamber, and left ventricular outflow tract long-axis views, as previously described (6). Left ventricular long-axis function (LAF) was measured as the portion of the stroke volume generated by AVPD using the following formula:

$$\text{LAF} = \frac{\text{AVPD}}{\text{Aepid} (1)}$$

where Aepid is the mean of the largest end-diastolic epicardial area of the left ventricle encompassed by the AVPD. Extensive details and volumetric validation of this method have been previously described (6). Figure 3 summarizes the method for measuring long-axis function. EVC was defined as stroke volume minus long-axis function.

![Fig. 1. Endocardial shortening, midwall shortening, and epicardial volume change (EVC). Schematic illustration of a short-axis view through the left ventricle showing measures involved in calculating endocardial and midwall shortening and fractional shortening and EVC. The dotted line denotes the epicardial border in end diastole. **Top** shows the measures for calculation of endocardial shortening. Dendo$_d$ denotes the endocardial diameter of the left ventricle in the short-axis view in end diastole. Dendo$_s$ is the corresponding measure in end systole. Endocardial shortening (millimeters) was calculated as Dendo$_s$ - Dendo$_d$, and endocardial fractional shortening (percent) was calculated as (Dendo$_s$ - Dendo$_d$)/Dendo$_s$. **Bottom** shows the measures for calculation of midwall shortening. Dmwd denotes the midwall diameter of the left ventricle in the short-axis view in end diastole. Dmw$_s$ is the corresponding measure in end systole. Midwall shortening (millimeters) was calculated as Dmwd$_s$ - Dmwd$_d$, and midwall fractional shortening (percent) was calculated as (Dmwd$_s$ - Dmwd$_d$)/Dmwd$_d$. **Adapted from de Simone et al. (7).**](image1)

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![Fig. 2. Schematic illustration of the effects of wall thickness on fractional shortening. The illustration depicts the long-axis (**top**) and short-axis (**middle**) of a theoretical model of a left ventricle with no myocardium and only short-axis EVC (A) and 3 models (B–D) with no short-axis EVC in the setting of no myocardium and only long-axis function (B), normal myocardium (C), and hypertrophied myocardium with increased wall thickness (D). Solid lines represent the borders of the left ventricle in end diastole, and dashed lines in end systole. The thin, dotted line represents the intersection of the long- and short-axis views. All models have the same stroke volume. In model A, the stroke volume is generated by only short-axis EVC but no long-axis function. In models B–D, the stroke volume is generated by only long-axis function by way of the atrioventricular plane displacement (AVPD) but not short-axis function. In models C and D, an unchanged volume of myocardium is rearranged between end diastole and end systole. The addition of myocardium to the model in C and D increases the ejection fraction. Note how fractional shortening is influenced by the wall thickness despite the absence of any short-axis function in models C and D. The short-axis function in the form of an EVC is illustrated in Fig. 4. **Adapted from Carlsson et al. (6).**](image2)

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![Fig. 3. Schematic illustration of how the long-axis contribution to the stroke volume was measured. **Left:** the epicardial contour of a schematic left ventricle with only long-axis pumping and no short-axis pumping. The broken lines indicate the position of the atrioventricular (AV) plane in end systole. The long-axis contribution to the stroke volume, as gray, extends from the AV plane toward the apex. **Middle:** the gray region is unchanged in size and indicates the outer diameter (d) multiplied by the AVPD. **Right:** myocardium is added to the model. Myocardium reduces the inner contour of the ventricle, but the volume of the myocardium is constant throughout the cardiac cycle. The myocardium is rearranged as it pulls the AV plane toward the apex. This illustrates how long-axis function contributes to fractional shortening in the short-axis plane (dotted line) in the absence of any short-axis function. **Adapted from Carlsson et al. (6).** The short-axis function in the form of an EVC is illustrated in Fig. 4.](image3)

Fig. 3. Schematic illustration of how the long-axis contribution to the stroke volume was measured. **Left:** the epicardial contour of a schematic left ventricle with only long-axis pumping and no short-axis pumping. The broken lines indicate the position of the atrioventricular (AV) plane in end systole. The long-axis contribution to the stroke volume, as gray, extends from the AV plane toward the apex. **Middle:** the gray region is unchanged in size and indicates the outer diameter (d) multiplied by the AVPD. **Right:** myocardium is added to the model. Myocardium reduces the inner contour of the ventricle, but the volume of the myocardium is constant throughout the cardiac cycle. The myocardium is rearranged as it pulls the AV plane toward the apex. This illustrates how long-axis function contributes to fractional shortening in the short-axis plane (dotted line) in the absence of any short-axis function. **Adapted from Carlsson et al. (6).** The short-axis function in the form of an EVC is illustrated in Fig. 4.
Endocardial and midwall shortening (millimeters) and FS (percent) were calculated from measurements in a single midventricular short-axis slice as described in Fig. 1. The endocardial diameter (Dendo) of the left ventricle was calculated as two times the endocardial radius. The endocardial radius (Rendo) and epicardial radius (Repi) were calculated according to the following formula:

\[
Rendo = \frac{Dendo}{\pi}^{1/2} \quad (2)
\]

\[
Repi = \frac{A_{epi}}{\pi}^{1/2} \quad (3)
\]

where Aendo and Aepi are the endocardial and epicardial areas of a midventricular short-axis slice, respectively, and \( \pi \) is 3.14. Endocardial diameter was measured in both end diastole and end systole. Midwall diameter (Dmw) was calculated according to:

\[
Dmw = \frac{1}{2} (WT) + Dendo + \frac{1}{2} (WT) \quad (4)
\]

where WT is wall thickness, and Dendo is the endocardial diameter. Wall thickness was determined as the difference between epicardial and endocardial radii as determined by Eqs. 2 and 3. Midwall diameter was measured in both end diastole and end systole.

**Statistical methods.** Statistical analysis was performed with the software SPSS (version 16). Data are presented as means ± SE. Differences between subject groups were tested with the Mann-Whitney test. Univariate regression was performed using Pearson correlation coefficient and expressed as its square (\( R^2 \)). Differences between linear and logarithmic regression were tested by comparing the mean squared residuals using a paired \( t \)-test. \( P < 0.05 \) was considered statistically significant.

**RESULTS**

Subject characteristics including measures of left ventricular function are displayed in Table 1. Endocardial and midwall shortening were influenced by wall thickness (\( P < 0.05 \) for both), whereas EVC was not (Table 2). The dependence of endocardial shortening on wall thickness is illustrated in Fig. 4, which shows representative long- and short-axis magnetic resonance (MR) images of two subjects. The subject with markedly different end-diastolic wall thickness has a greater endocardial shortening.

Table 1. Subject characteristics

<table>
<thead>
<tr>
<th>Measure</th>
<th>Normals</th>
<th>Athletes</th>
<th>Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subjects, ( n )</td>
<td>12</td>
<td>12</td>
<td>12</td>
</tr>
<tr>
<td>Age, yr</td>
<td>24±1</td>
<td>35±1†</td>
<td>54±2†</td>
</tr>
<tr>
<td>Females, ( n ) (%)</td>
<td>5 (42)</td>
<td>4 (33)</td>
<td>4 (33)</td>
</tr>
<tr>
<td>BSA, m²</td>
<td>1.94±0.03</td>
<td>1.90±0.03</td>
<td>2.03±0.04</td>
</tr>
<tr>
<td>Heart rate, beats/min</td>
<td>63±2</td>
<td>55±1</td>
<td>77±2*</td>
</tr>
<tr>
<td>LVEDV, ml</td>
<td>185±10</td>
<td>218±10*</td>
<td>333±27†</td>
</tr>
<tr>
<td>LVESV, ml</td>
<td>69±5</td>
<td>78±7</td>
<td>261±24‡</td>
</tr>
<tr>
<td>LVEF, %</td>
<td>63±1</td>
<td>65±2</td>
<td>22±2†</td>
</tr>
<tr>
<td>Endocardial shortening, mm</td>
<td>11.1±0.4</td>
<td>11.8±0.4</td>
<td>8.1±0.4‡</td>
</tr>
<tr>
<td>Endocardial fractional shortening, %</td>
<td>68±2</td>
<td>68±3</td>
<td>58±3‡</td>
</tr>
<tr>
<td>Endocardial shortening, mm/ml SV</td>
<td>0.098±0.004</td>
<td>0.085±0.003*</td>
<td>0.122±0.011*</td>
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<tr>
<td>Midwall shortening, mm</td>
<td>3.9±0.2</td>
<td>4.2±0.1</td>
<td>1.3±0.2‡</td>
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<tr>
<td>Midwall fractional shortening, %</td>
<td>21±1</td>
<td>21±1</td>
<td>16±1‡</td>
</tr>
<tr>
<td>Midwall shortening, mm/ml SV</td>
<td>0.034±0.002</td>
<td>0.031±0.001</td>
<td>0.019±0.003‡</td>
</tr>
<tr>
<td>Short-axis EVC, ml</td>
<td>47±4</td>
<td>60±3*</td>
<td>24±3‡</td>
</tr>
<tr>
<td>Short-axis EVC, %SV</td>
<td>40±2</td>
<td>43±2</td>
<td>33±2</td>
</tr>
<tr>
<td>Long-axis function, ml</td>
<td>69±4</td>
<td>80±4</td>
<td>48±2‡</td>
</tr>
<tr>
<td>Long-axis function, %SV</td>
<td>60±2</td>
<td>57±2</td>
<td>67±2</td>
</tr>
</tbody>
</table>

Values are means ± SE. BSA, body surface area; LV, left ventricle; EDV, end diastolic volume; ESV, end systolic volume; EF, ejection fraction; SV, stroke volume; EVC, epicardial volume change. *, †, and ‡ denote \( P < 0.05 \), \( P < 0.01 \), and \( P < 0.001 \) compared with healthy volunteers (normals), respectively.

**DISCUSSION**

The main finding of the current study is that both endocardial and midwall shortening are affected by wall thickness, whereas EVC is not. This implies that FS is not solely a measure of short-axis function and should not be referred to as such.

It is not known why cardiac physiologists and cardiologists have focused their attention on the endocardium when trying to quantify short-axis function. It may be that the tradition of measuring left ventricular volumes with endocardial delineations in cine cardiac tomographic images has contributed to this endocardial focus. The left ventricular stroke volume is typically measured by delineating the endocardial surface of the left ventricle in end diastole and end systole. However, the volume of left ventricular myocardium is nearly constant during the cardiac cycle (6). Thus the stroke volume is the same when measured by delineating the endocardial surface as when measured by delineating the epicardial surface of the left ventricle in end diastole and end systole, respectively (6).

This study has presented a theoretical model and empirical evidence showing that endocardial or midwall shortening is by definition influenced by myocardial wall thickness. It is not surprising that both endocardial and midmural FS are related to myocardial wall thickness, since they are mathematically coupled. An appendix based on the work of Riordan and Kovacs (14) is provided that gives the details of this mathematical relationship.

The current work has been based on a previously presented and validated theoretical framework for measurement of the short- and long-axis function as mutually exclusive and complementary volumetric components of the stroke volume (5, 6). Absolute (millimeters) or relative (percent) FS employing measurement of the endocardial or midmural inner diameter of the left ventricle have been used to assess short-axis function during both systole (13, 17, 20) and diastole (14). Notably, the current study measured the volume of long- and short-axis contribution to the stroke volume between end diastole and end systole. By analogy, the same methods could be applied to each time frame throughout both systole and diastole. Such future studies might be of value to provide further insight into the relative contributions of long- and short-axis function during the E-wave and A-wave of diastolic filling.
We have shown that the EVC can be visualized as a small change in short-axis epicardial area. This finding confirms that of a previous study that called this phenomenon the “crescent effect” (18). That study performed two-dimensional measurements of this space, and the current study built on those findings to quantify the entire volume of the short-axis contribution to the stroke volume.

Furthermore, we found that the relationship between short-axis function (EVC) and long-axis function is linear. FS has been used in studies of the relationship between long- and short-axis function by echocardiography (3, 16) with conflicting results with regard to the linearity of this relationship. Previous studies have showed a linear relation between AVPD and endocardial FS (1, 4, 19). By comparison, Ballo et al. (3) showed a nonlinear relation between AVPD and midwall FS and claimed that there is a nonlinear relationship between circumferential (short-axis) and longitudinal (long-axis) function. However, the current study has shown that the relationship between EVC and long-axis function is linear over a wide range of values for short- and long-axis function.

Our study showed a trend toward a greater long-axis contribution to the stroke volume in patients compared with controls. This may seem counterintuitive considering that a number of previous studies have shown that the AVPD is reduced in the diseased heart (1, 16, 19). The patients in our study did indeed have a reduced AVPD (6). However, the contribution to the stroke volume made by long-axis function is the product of AVPD and epicardial short-axis. Our patients had larger epicardial short-axis areas due to left ventricular dilatation, thereby contributing to a larger total long-axis function. This finding illustrates that the relative contri-

![Fig. 6. A schematic diagram illustrating a simplified cylinder as the left ventricle. Inner radius (ri), outer radius (ro), and height (h) of the cylinder are shown in end diastole (ed; solid lines) and end systole (es; dotted lines).](image-url)
bution to the stroke volume by long-axis function may be preserved or even increase despite a reduction in AVPD.

The findings in the current study may be motivation to reassess the appropriateness of using endocardial or midwall FS when specifically assessing left ventricular short-axis function in future studies. Midwall FS has been successfully used as a measure for diagnostic quantification of left ventricular function in left ventricular hypertrophy (7). The current study does not question the use of FS as a quantitative measure of ventricular function as such, it merely clarifies that FS is not synonymous with short-axis function. Since FS is dependent on myocardial wall thickness and thus the apparent thickening that occurs as a result of long-axis function, it might be more appropriate to discuss FS in terms of ventricular function without a specification of its relation to a particular short or long axis or merely endocardial or midwall shortening.

A study using echocardiography measured left ventricular long-axis function and found that it comprised 82% of the stroke volume (8). Our (6) previous study using CMR found that ~60% of the left ventricular stroke volume was generated by long-axis function. CMR is generally considered to be the most accurate modality for volumetric quantification of left ventricular volumes owing to its ability to image the full left ventricle in three dimensions without restrictions on image angulation (12). It may be that the discrepancies between CMR and echocardiography are due to difficulties with using two-dimensional echocardiography to accurately acquire short-axis images perpendicular to the long axis of the left ventricle. Two-dimensional echocardiography will remain the everyday tool for assessment of left ventricle function.

Limitations. Clinical echocardiography is undertaken with a higher temporal resolution compared with clinical MR imaging. However, although high temporal resolution is important when quantifying velocities, the temporal resolution of MR for quantifying volumes as in the current study is more than sufficient given that temporal resolution was at most 30 ms. Taken together, it may be that MR is more accurate for quantifying true long- and short-axis function.

FS is dependent on myocardial wall thickness, whereas EVC represents true short-axis function and is not dependent on myocardial wall thickness. This is not surprising considering that FS is mainly caused by rearrangement of myocardium myocardial wall thickness. This is not surprising considering that FS is not synonymous with short-axis function. Since FS is dependent on myocardial wall thickness and thus the apparent thickening that occurs as a result of long-axis function, it might be more appropriate to discuss FS in terms of ventricular function without a specification of its relation to a particular short or long axis or merely endocardial or midwall shortening.

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Wall thickness is thereby dependent on both the outer, epicardial, and the inner, endocardial, dimensions of the ventricle. Also note that the amount of myocardium is unchanged throughout the cardiac cycle. Thus the myocardium is thicker in end systole compared with end diastole.

The EVC is calculated as follows:

$$EVC = (\pi \cdot r_{oes}^2 \cdot h_{oes}) - (\pi \cdot r_{val}^2 \cdot h_{val})$$

Note that the inner radius of the cylinder, ri, and thereby the wall thickness, is not involved in this calculation.

By comparison, the endocardial FS, FS_{endo}, is calculated as follows:

$$FS_{endo} = \left\{ \frac{(2 \cdot r_{oes} - 2 \cdot r_{val})}{(2 \cdot r_{val})} \right\}$$

Also, the midwall FS, FS_{mw}, is calculated as follows:

$$FS_{mw} = \left\{ \frac{(2 \cdot r_{oes} + 1/2 \cdot (r_{oes} - r_{val})) - (2 \cdot r_{oes} + 1/2 \cdot (r_{oes} - r_{val}))}{(2 \cdot r_{oes} + 1/2 \cdot (r_{oes} - r_{val}))} \right\}$$

Note that calculations of both endocardial and midwall FS include the inner radius, an endocardial measurement, and are thereby mathematically coupled to wall thickness.

In summary, these formulae show that both endocardial and midwall FS are functions of the myocardial wall thickness, whereas EVC is independent of wall thickness.

**GRANTS**

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**DISCLOSURES**

No conflicts of interest are declared by the author(s).

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