Atrioventricular plane displacement is the major contributor to left ventricular pumping in healthy adults, athletes, and patients with dilated cardiomyopathy

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Atrioventricular plane displacement is the major contributor to left ventricular pumping in healthy adults, athletes, and patients with dilated cardiomyopathy. Am J Physiol Heart Circ Physiol 292: H1452–H1459, 2007. First published November 10, 2006; doi:10.1152/ajpheart.01148.2006.—Previous studies using echocardiography in healthy subjects have reported conflicting data regarding the percentage of the stroke volume (SV) of the left ventricle (LV) resulting from longitudinal and radial function, respectively. Therefore, the aim was to quantify the percentage of SV explained by longitudinal atrioventricular plane displacement (AVPD) in controls, athletes, and patients with decreased LV function due to dilated cardiomyopathy (DCM). Twelve healthy subjects, 12 elite triathletes, and 12 patients with DCM and ejection fraction below 30% were examined by cine magnetic resonance imaging. AVPD and SV were measured in long- and short-axis images, respectively. The percentage of the SV explained by longitudinal function (SVAVPD%) was calculated as the mean epicardial area of the largest short-axis slices in end diastole multiplied by the AVPD and divided by the SV. SV was higher in athletes [140 ± 4 ml (mean ± SE), P = 0.009] and lower in patients [72 ± 7 ml, P < 0.001] when compared with controls [116 ± 6 ml]. AVPD was similar in athletes [17 ± 1 mm, P = 0.45] and lower in patients [7 ± 1 mm, P < 0.001] when compared with controls [16 ± 0 mm]. SVAVPD% was similar both in athletes (57 ± 2%, P = 0.51) and in patients (67 ± 4%, P = 0.24) when compared with controls (60 ± 2%). In conclusion, longitudinal AVPD is the primary contributor to LV pumping and accounts for ~60% of the SV. Although AVPD is less than half in patients with DCM when compared with controls and athletes, the contribution of AVPD to LV function is maintained, which can be explained by the larger short-axis area in DCM.

Left ventricle; stroke volume; cardiac pumping; magnetic resonance imaging

Cardiac pumping is the result of the contraction of myocardial fibers organized in different orientations and different layers (15, 35), resulting in both longitudinal and radial shortening of the ventricles (27). Longitudinal shortening through atrioventricular plane displacement (AVPD) can be observed as the movement of the base of the ventricles toward the apex in systole (16, 21, 25, 29). Previous studies using echocardiography in healthy subjects have reported conflicting data regarding the percentage of the left ventricular (LV) stroke volume (SV) resulting from AVPD (SVAVPD%), although this could be explained by differences in methodology and definitions (4, 9, 43). Furthermore, the percentage of SV explained by AVPD may differ between controls, athletes, and patients with decreased cardiac function, and this could be of importance in understanding differences in the pumping physiology between these groups.

It has been proposed that the portion of the SV which is generated by AVPD (SVAVPD) can be derived by multiplying the AVPD by the epicardial LV area located 2–3 cm apical to the base of the heart (25). This method has been employed using echocardiography in a study of healthy volunteers (9). However, the method does not take into account the variation in diameter of the LV within the range of the AVPD, and it has not been validated by independent measurements. Magnetic resonance imaging gives the opportunity to measure both endocardial and epicardial volumes with great accuracy and precision and to image the whole heart in any plane. This makes it possible to validate the derived method against direct volumetry of the SV generated by AVPD.

Therefore, the purposes of this study were 1) to measure the percentage of the SV explained by longitudinal function measured as AVPD in healthy subjects, athletes, and patients with severely decreased LV function due to dilated cardiomyopathy (DCM) using MRI, 2) to revise the method for calculation of the longitudinal component of the SV (SVAVPD), 3) to validate the revised method against direct volumetry of the SV generated by AVPD, and, finally, 4) to anatomically localize where the SV is generated.

Materials and Methods

Study population. The study was approved by the local ethics committee. Written informed consent was either obtained (controls, athletes, and 9 patients) or waived by the ethics committee (3 patients). Twelve healthy controls (mean age 24 yr, 5 women), 12 elite triathletes (mean age 35 yr, 4 women), and 12 patients with DCM and ejection fraction below 50% (mean age 54 yr, 4 women) were examined with MRI in the supine position. The controls had normal blood pressure (<140/90 mmHg), a normal ECG, no medical history of any cardiac condition, and no cardiovascular medication. The triathletes were selected from the Swedish national elite of triathletes and exercised 12 ± 3 h/wk (mean ± SD). The subjects in the patient group were selected from patients with idiopathic DCM referred to a routine clinical MRI. DCM was determined by medical records, MRI findings, and the exclusion of significant coronary disease by myocardial perfusion imaging and/or coronary angiography.

MRI. A 1.5 T MRI scanner (Philips Intera, Philips, Best, The Netherlands) with a cardiac synergy coil was used. Cine images in the short-axis plane and three long-axis planes and the two-chamber, four-chamber, and LV outflow tract views were obtained in all subjects during end-expiratory apnea.

Sequences and imaging parameters. A steady-state free precession sequence with retrospective ECG triggering was used to achieve 30

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time phases per heart cycle, giving a temporal resolution of typically 30 ms, repetition time 2.8 ms, echo time 1.4 ms, flip angle 60°, and spatial resolution of $1.4 \times 1.4 \times 8$ mm. Parallel imaging with a sensitivity encoding factor of 2 was used in the short-axis images. Fifty phases per heart cycle were acquired in the long-axis planes in the control group. In nine control subjects, a radial stack of 18 long-axis slices of the LV was obtained at 10° intervals (Fig. 1) (8).

**Image analysis.** All images were evaluated using freely available software (Segment 1.466; available at http://segment.heiberg.se) (18).

**SV.** The SV was calculated from manual delineations of short-axis images by subtracting the systolic from the diastolic ventricular volume as previously described (30). This can be expressed as the following formula:

$$SV = \text{Endo}_{\text{ED}} - \text{Endo}_{\text{ES}}$$

where Endo$_{\text{ED}}$ and Endo$_{\text{ES}}$ are the endocardial contours of the LV in end diastole and end systole, respectively. Moreover, it is known that the epicardial volume of the LV (Epi$_{\text{ED}}$ or Epi$_{\text{ES}}$) is equal to the endocardial volume plus the myocardial volume according to the following formulas:

$$\text{Epi}_{\text{ED}} = \text{Endo}_{\text{ED}} + \text{myocardial volume}_{\text{ED}}$$

$$\text{Epi}_{\text{ES}} = \text{Endo}_{\text{ES}} + \text{myocardial volume}_{\text{ES}}$$

Furthermore, it is known that the myocardial volume is constant over the cardiac cycle (17, 36, 40), and thus

$$\text{myocardial volume}_{\text{ES}} = \text{myocardial volume}_{\text{ED}}$$

Hence Eqs. 1–4 can be combined and rewritten as the following:

$$SV = \text{Epi}_{\text{ED}} - \text{Epi}_{\text{ES}} = \text{Endo}_{\text{ED}} - \text{Endo}_{\text{ES}}$$

Consequently, the SV was calculated using both the epicardial and endocardial borders of the LV. The radius of each short-axis slice was calculated as the square root of the ratio between the area and π ($3.14$). The difference in radius from end diastole to end systole was calculated, and the mean of the apical and midventricular two-thirds of the LV was obtained for each subject.

**Derived method for SV$_{\text{AVPD}}$.** The AVPD can be seen as a piston-like movement of the AV plane in the base-apex direction within the LV. The volume explained by the AVPD is the volume at the base of the LV between the position of the AV plane in end diastole and end systole (Fig. 2, A–D). The SV generated by the AVPD (SV$_{\text{AVPD}}$) was calculated as the LV short-axis area multiplied by the AVPD. The basal part of the ventricle including the AV plane is not flat but rather is shaped like a dome. This is both illustrated schematically and seen in vivo in Fig. 2. This dome shape has consequences for the derived method to calculate the part of the SV generated by the longitudinal AVPD. The area multiplied by the AVPD cannot be the short-axis area at the mitral annulus (d$_2$) but rather must be the largest epicardial short-axis area of the LV (d$_1$). It is important to note that the area used to calculate the longitudinal contribution to the SV should be the epicardial area as explained in Fig. 2, C and D. The epicardial area was taken from the short-axis slice or slices that encompassed the range of the AVPD. Thus, for patients, the area of the single largest short-axis slice was used because the thickness of one short-axis slice and the mean AVPD were both 6 mm. In controls and athletes, the mean AVPD was 16 mm, and, therefore, the mean area of the largest two slices was used for these groups. The resulting volume was divided by the SV to calculate the percentage of SV explained by longitudinal function for the LV (SV$_{\text{AVPD}}$). Furthermore, the diameter at the mitral annulus (d$_2$) and the epicardial short-axis at the midventricular level (d$_1$) were measured in the four-chamber magnetic resonance (MR) images of the control subjects to compare the diameter used for SV$_{\text{AVPD}}$ (d$_1$) and mitral annular excursion volume (d$_2$) (4).

**Validation of SV$_{\text{AVPD}}$ by the volumetric method.** In nine control subjects, the volume contribution of the AVPD to the SV was evaluated in a stack of radial long-axis slices by direct planimetry (Fig. 1). For each long-axis slice in a radial stack, the epicardial border of the LV in end diastole was outlined. Next, the position of the AV plane in end systole was identified. The outline of the epicardial border of the LV in end diastole was then translated along the long axis of the LV toward the apex so that the basal part of the outline had a position corresponding to the position of the epicardial border of the LV in end systole. The AV plane was moved to the position of the atrioventricular (AV) plane in end systole. The volume illustrated by diagonal lines between the contours represents the stroke volume (SV) generated by the AV plane displacement (AVPD). The white arrow indicates concomitant radial shortening causing a further decrease in area of the basal part of the LV during systole.

**Fig. 1.** Radial projections of the left ventricle (LV). A: a short-axis magnetic resonance (MR) image of the LV in end diastole. White lines indicate how the radial stack of 18 long-axis slices was acquired at 10° intervals. The dashed white line shows the position of the long-axis images in B and C. The solid white line in B shows the contour of the epicardium in end diastole. The contour is copied to the end-systolic image (C). The curved dotted line is the contour of the basal part of the LV in end diastole moved to the position of the atrioventricular (AV) plane in end systole. The volume illustrated by diagonal lines between the contours represents the stroke volume (SV) generated by the AV plane displacement (AVPD). The white arrow indicates concomitant radial shortening causing a further decrease in area of the basal part of the LV during systole.
AVPD is the major contributor to LV pumping

The AVPD (vertical arrows) of the LV can be viewed as a piston-like movement of the basal part of the ventricle. Valves are omitted from the illustration for the sake of simplicity. Broken lines indicate the position of the AV plane in end systole. The volume of blood ejected from the heart by the AVPD is the volume basal to the position of the AV plane in end systole, shown in gray. $d_1$ denotes the largest diameter of the LV defined as the greatest epicardial area in a short-axis plane; $d_2$ denotes the diameter at the position of the mitral valve. Gray regions indicate the diameter ($d_1$) multiplied by the AVPD. This region is the same size as the gray region in $A$ (see $D$). $C$: myocardium is added to the model, thereby reducing the inner contour (endocardium) of the ventricle. Myocardium in end diastole is indicated by solid lines and in end systole by dashed lines. The volume of the myocardium is constant throughout the cardiac cycle. The myocardium is rearranged as it pulls the AV plane toward the apex and therefore appears thickened. The portion of the SV that is generated by the AVPD ($SV_{AVPD}$) is again indicated in gray and is identical to the gray regions in $A$ and $B$. This illustrates that the area at $d_1$ (the largest short-axis diameter) will be used when calculating the contribution of AVPD to SV. The SV caused by the AVPD would be underestimated if the area of either the mitral annulus at $d_2$ or the endocardial area at $d_1$ would be used. $D$: the gray region in $A$ and $C$ is divided by a dotted line and broken apart. When they are added together, the gray region in $B$ is generated. This illustrates why the largest epicardial area of the ventricle should be multiplied by the AVPD in the derived method for calculating $SV_{AVPD}$. $E$: a schematic short-axis view in end diastole (solid line) and end systole (broken line) at the level of the thin dotted line in $C$. The horizontal dashed lines indicate the position of the nonmoving epicardium in end diastole and end systole. Note that in this model there is no radial squeezing motion. However, the endocardium will still move inward during systole (compare $F$) as the result of the longitudinal AVPD, and this gives the false impression of a squeezing motion when viewing the endocardium in a short-axis plane. $F$: the left ventricle in four-chamber long-axis MR images ($1$ and $2$) and corresponding short-axis images at the levels indicated by thin dotted lines ($3$–$6$). The LV, right ventricle (RV), left atrium (LA), and right atrium (RA) can be seen. The solid white line is the contour of the epicardium in end diastole. The end-diastolic epicardial contour is copied to end systole. The curved dotted line in $1$ and $2$ is the basal contour of the epicardium in end diastole moved to the position of the AV plane in end systole. The area between these contours (arrows) corresponds to the gray regions in $A$–$D$. The piston-like movement of a smaller basal part of the ventricle into a larger midventricular part as seen in $1$ and $2$ explains most of the apparent epicardial movement inward during systole seen in $3$ and $4$. In contrast, the epicardial area from the level of $5$ and $6$ becomes smaller as the position approaches the apex. Thus longitudinal AVPD will not move a smaller area into a larger area in the apical and midventricular parts of the ventricle, meaning that the epicardial inward movement at these levels rather reflects true radial function.

AV plane in end systole. The volume difference between the two borders in the base of the end-diastolic contour was calculated for all radial slices.

AVPD. The AVPD was measured in long-axis images (Fig. 3). The basal location of the muscular insertion of the ventricle to the AV plane was manually identified in each of the three long-axis images along a line parallel to the long axis of the LV. This resulted in six locations for measuring the AV plane position in the LV. The maximum AVPD was calculated from end diastole to end systole, and the average of the six locations was calculated.

Anatomical location of the contribution to the SV. The difference in epicardial volume between end diastole and end systole for each short-axis slice was calculated for all subjects. The relative contribution of each short-axis slice position to the SV was calculated by dividing the difference in epicardial volume by the total SV for that subject. Furthermore, the contribution to SV for each individual short-axis slice was compared with the epicardial volume of that slice in end diastole.

Apex position. In nine control subjects, the most apical part of the ventricle was determined in end diastole and end systole in all radial slices, and the mean distance of the apical motion was measured. In all other subjects, the position of the LV apex in end diastole and end systole was determined in the three long-axis slices, and the distance between the positions was measured.
higher in patients compared with controls. Athletes had slightly higher end-diastolic volumes compared with controls. Typical MR images from a control, athlete, and patient are shown in Fig. 4.

Validation of $SV_{AVPD}$. The $SV_{AVPD}$ determined by the volumetric method did not differ from the $SV_{AVPD}$ from the derived method in the nine control subjects ($70 \pm 14$ vs. $68 \pm 11$, $P = 0.67$). The regression line comparing the volumetric and derived method ($r = 0.82$, $P = 0.007$) was described by the following equation: derived = ($1.1 \times$ volumetric) − 3.0. The difference between the volumetric and the derived method was 2 ± 8 ml.

$SV$. The $SV$ was higher in athletes ($140 \pm 4$ ml, range 115–157, $P = 0.009$) and lower in patients ($72 \pm 7$ ml, range 43–121, $P < 0.001$) when compared with controls ($116 \pm 6$ ml, range 77–152) (Fig. 5). The $SV$ indexed to body surface area was also higher in athletes ($74 \pm 2$ ml/m$^2$, range 65–83, $P < 0.001$), and lower in patients ($36 \pm 3$ ml/m$^2$, range 21–61, $P < 0.001$) when compared with controls ($60 \pm 2$ ml/m$^2$, range 48–73). There was a linear relation between $SV$ determined by epicardial delineation ($SV_{epi}$) and endocardial delineation ($SV_{endo}$) $(r = 0.99$, $P < 0.001$) according to the equation: $SV_{endo} = (1.0 \times SV_{epi}) − 1.4$. Also, the difference between $SV_{epi}$ and $SV_{endo}$ was $0.8 \pm 5.4$ ml.

$AVPD$. $AVPD$ for controls was $16 \pm 0$ mm (range 14–19). $AVPD$ was similar in athletes ($17 \pm 1$ mm, range 14–20, $P = 0.45$) and lower in patients ($7 \pm 1$ mm, range 5–11, $P < 0.001$) (Fig. 5).

Derived method $SV_{AVPD}$. $SV_{AVPD}$ for controls was $60 \pm 2\%$ (range 51–69). $SV_{AVPD}$ did not differ for athletes ($57 \pm 2\%$, range 41–66, $P = 0.51$) or for patients ($67 \pm 4\%$, range 49–88, $P = 0.24$) (Fig. 5).

Short-axis area. The short-axis area used for calculating the $SV_{AVPD}$ for controls was $42 \pm 2$ cm$^2$ (range 30–53). The

### Table 1. Subject characteristics

<table>
<thead>
<tr>
<th></th>
<th>Controls</th>
<th>Athletes</th>
<th>Patients</th>
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<tr>
<td>$n$, $%$</td>
<td>12</td>
<td>12</td>
<td>12</td>
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<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>
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<tr>
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<td>1.90±0.03</td>
<td>2.03±0.04</td>
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<td>Sinus rhythm, $n$ (%)</td>
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<td>12 (100)</td>
<td>9 (67)</td>
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<td>Atrial fibrillation, $n$ (%)</td>
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<td>0 (100)</td>
<td>3 (33)</td>
</tr>
<tr>
<td>Heart rate, beats/min</td>
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<td>55±1</td>
<td>77±2‡</td>
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<tr>
<td>LVEDV, ml</td>
<td>185±10</td>
<td>218±10*</td>
<td>333±27†</td>
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<td>LVESV, ml</td>
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<td>78±7</td>
<td>261±24†</td>
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<td>LVEF, %</td>
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<td>65±2</td>
<td>22±2‡</td>
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<td>LBBB, $n$ (%)</td>
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<td>Medication, $n$ (%)</td>
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<tr>
<td>ACEI</td>
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<td>Unknown</td>
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Values for continuous variables are means ± SE. BSA, body surface area; LVEDV, left ventricular end-diastolic volume; LVESV, left ventricular end-systolic volume; LVEF, left ventricular ejection fraction; LBBB, left bundle branch block; ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin II receptor blocker; NYHA class, symptoms of heart failure according to the New York Heart Association classification. DCM, dilated cardiomyopathy. *$P < 0.05$, †$P < 0.001$ denote significant differences from controls for continuous variables.
athletes’ areas were similar (48 ± 2 cm², range 38–58, P = 0.08), but the patients’ areas were larger (67 ± 4 cm², range 45–83, P < 0.001). In controls, the epicardium moved 3.4 ± 0.2 mm (range 1.8–4.5) toward the center of the LV from end diastole to end systole in the apical and midventricular two-thirds of the LV. The epicardial radial inward motion for athletes did not differ (3.7 ± 0.2 mm, range 2.6–5.0, P = 0.51) when compared with controls, but the epicardial motion of the patients was lower (1.0 ± 0.2 mm, range 0.3–2.3, P < 0.001). For controls, the epicardial short-axis diameter at the midventricular level (d₁, 70 ± 2 mm, range 62–81 mm) was nearly double the diameter of the mitral annulus (d₂, 36 ± 2, range 28–48 mm).

Anatomical location of contribution to SV. The relative contribution to SV was plotted for each short-axis slice position of the LV for each group (Fig. 6). The greatest portion of the SV was generated in slice positions at the base of the LV. Only a weak correlation was found between the relative contribution to SV of each short-axis slice and the relative size of that slice (r = 0.35, P < 0.001). Regardless, the largest difference in epicardial volume and thus contribution to SV was found at the base of the ventricles in all groups (Fig. 6). The location of the largest part of the SV at the base of the ventricle can be seen in the superimposed contours of the long-axis images in Fig. 4.

Apical motion. The apical motion between end diastole and end systole was 1.9 ± 0.5 (range −0.1 to 5.1), 1.8 ± 0.5 (range −0.8 to 5.3), and 0.1 ± 0.2 mm (range −1.4 to 1.4) for controls, athletes, and patients, respectively. Athletes and controls were similar (P = 0.93), but patients had less apical movement than did controls (P = 0.004). Negative apical movement, i.e., apical dyskinesia, was seen in seven patients (see example in Fig. 4) but only in one healthy subject.

DISCUSSION

The major finding of this study was that ~60% of the SV is generated by the longitudinal AVPD and this does not differ in athletes or in patients with dilated ventricles. The largest portion of the SV can be found at the base of the ventricles. Furthermore, the previously proposed method for calculating the SV generated by AVPD (25) has been revised and validated. The revised method showed good agreement to direct planimetry of this volume.
Longitudinal function. AVPD has been recognized as an important contributor to LV pumping (13, 16, 19, 22, 25) and used as a measure of global LV function (1, 2, 37), but the magnitude of the longitudinal contribution to SV has been unclear (4, 5, 9, 43). The current study showing that 60% of the SV is derived from AVPD ($SV_{AVPD}$) differs from earlier studies using echocardiography, suggesting the $SV_{AVPD}$ to be as high as 82% (9) and from the volume generated by the mitral annular movement, which has been reported to be 19% (4). The discrepancy compared with the first study may be explained by known methodological limitations with two-dimensional echocardiography, for example, an underestimation of measured volumes as discussed by the authors of that study (9). MRI has the advantage of accurate and precise volume measurements due to three-dimensional coverage of the whole LV. Furthermore, MRI offers excellent soft tissue contrast, which gives the ability to delineate the epicardium and endocardium, thereby making MRI particularly well suited for these types of studies. In the current study, short-axis images were positioned parallel to the AV plane and thus perpendicular to the AVPD, giving optimal accuracy in measurement of the short-axis area. Furthermore, the current study has taken into account the variation in diameter of the LV and used the mean of the short-axis areas encompassed by the range of the AVPD. This revised method showed good agreement when compared with direct measurement of the $SV_{AVPD}$. The second study using three-dimensional transesophageal echocardiography determined the part of the SV explained by mitral annular excursion to be 19% (4). However, as shown by the current study, the mitral annular excursion volume does not represent the entire portion of the SV generated by AVPD. This can be explained by the finding that the diameter of the mitral annulus was one-half of the diameter of the midventricular part of the ventricle. This has been commented by those authors in a recent reply (5) to a letter to the editor (43) in American Journal of Physiology-Heart and Circulatory Physiology. In Fig. 2C, the mitral annular excursion volume would be represented by the area at $d_2$ multiplied by AVPD, whereas the entire $SV_{AVPD}$ measured in the present study is defined as the area at $d_1$ multiplied by AVPD. AVPD was greater in the present study (16 mm) compared with the study by Carlhall and coworkers (10 mm), and this can be explained by several factors. First, the subjects in Carlhall’s study were older (56 ± 11 yr), and AVPD is known to decrease with age (42). Second, six of the subjects of Carlhall’s study were imaged during general anesthesia, and this could affect AVPD as commented by Carlhall and coworkers. Last, the AVPD was measured at the mitral annulus in Carlhall’s study and at the tip of the muscular wall in our study. Notably, the AVPD of the present study is in accordance with recently published normal values of AVPD by MRI (26).

Radial function. Since 60% of the SV is generated by longitudinal AVPD, the remaining 40% of the SV must be the result of radial shortening. The endocardial movement toward the center of the lumen has previously been described as radial thickening due to the contraction of circular myocardial fibers (31, 35) but has been shown to be the result of more complex myocardial mechanics (3, 12, 17, 28, 36). The current study has shown that either epicardial or endocardial contours can be used interchangeably for the calculation of LV stroke volume. This is in line with earlier findings of a constant volume of the LV myocardium despite shortening and thickening during myocardial contraction (17, 36, 40). Thus the current results show that the inward motion of the endocardium is primarily the result of longitudinal shortening and redistribution of myocardium, as has been suggested previously (25, 40) (Fig. 2). Furthermore, the epicardial area at the mitral annulus in end diastole is smaller than the midventricular area because of the dome shape of the basal part of the LV. The dome shape will contribute to an apparent inward epicardial motion at the base of the LV during AVPD. Isolated radial function can be identified in the radial inward movement of the epicardium in the midventricular and apical two-thirds of the LV. This movement was found to be 3–4 mm in controls and athletes and ~1 mm in patients. The movement in controls is slightly larger but in agreement with the findings in previous studies in humans (~2 mm) (9, 25) and dogs (~1.5 mm) (36).

The reported contribution of longitudinal function to the inward motion of the endocardium has implications for the measurement of radial contribution to LV function. The reduction of endocardial diameter during systole, also called fractional shortening (11), has been used as a measure of radial function (31). Fractional shortening, however, is influenced by longitudinal function, as shown in the current study, and thus not solely a product of radial function. Therefore, it may be motivated to reevaluate the role of fractional shortening as a measure of radial function. The inward motion of the epicardium described in the present study corresponds in part to the “crescent effect” of the LV described by Kovacs and coworker.
 ers (32, 44). The crescent effect is the radial pericardial displacement of the LV most often seen at the posterolateral wall. However, as seen in Figs. 2 and 4, we found an inward epicardial movement circumferentially in the short-axis plane. Thus the crescent effect in part corresponds to the radial function of the LV.

Methodological aspects of using epicardial areas. The rationale for using epicardial areas when determining longitudinal function can be explained by considering the LV as the tube of a telescope that can shorten and lengthen along its long axis. The volume of the telescope decreases when the tube shortens, and this would be the SV of the tube. To calculate the decrease in volume, two measures need to be known: 1) the decreased length of the tube (the AVPD of the LV) and 2) the outer cross-sectional area of the tube (the short-axis epicardial area).

The outer area of the tube would be used because the decrease in volume is not affected by what is inside the tube. The volume decrease is only affected by what has disappeared from where the tube was before it was shortened lengthwise. Two tubes with the same outer area but one with a thick wall and the other with a thin wall (different thickness of the myocardium in the LV) will decrease the same volume given the same outer area and same long-axis shortening. Also, the wall of the tube is noncompressible, i.e., it retains its volume when shortening the tube. This analogy is true for the myocardium; the volume will be the same during the cardiac cycle because of its noncompressible nature.

Apical movement. The present study found that the contribution to the SV by apical motion is negligible (Fig. 6) and that the apex remains essentially stationary during systole. Earlier studies have also reported the apex to be relatively stationary (3, 14, 20, 34, 38). We (6) and others (44) have previously reported a limited shortening (0.9 ± 0.5% and 0.03 ± 1.0%, respectively) of the entire heart in the apex-base direction during systole and a limited movement (2.3 ± 0.2 mm) of the center of volume of the heart during the cardiac cycle (7). Taken together, these findings imply that the apical movement can be no more than a few millimeters, and this is confirmed by the present study. In contrast, a study using epicardially implanted radiopaque markers in sheep reported that apical motion constitutes 22% of the longitudinal shortening of the ventricle (33). However, the same study found that isolated AVPD correlated better to ventricular stroke work than AVPD combined with apex movement. This implies that apical epicardial motion is not a large contributor to the SV. Furthermore, it is possible that the increased apical movement found in the sheep study may be an effect of the surgical preparation with pericardiotomy. Moreover, the epicardial contour of the apex moves very little; however, this does not imply that the apical myocardium does not contribute to LV pumping. The mechanics of the myocardium as such were not studied in the present study.

Similarity of SVAVPD% between the groups. Controls, athletes, and patients had similar SVAVPD%. This can be explained by the nonsignificant trend toward larger short-axis areas in athletes and significantly larger short-axis areas in patients compared with controls. This is further discussed in Fig. 6.

Anatomical location of SV. The largest part of the SV is generated at the basal area of the ventricles in all groups (Fig. 6). In patients, the most basal short-axis image was particularly prominent, although the difference when compared with the healthy groups was not significant. This is explained by a large basal area and lower AVPD, typically 7 mm. With 8-mm slice thickness, the position of the base of the ventricle in patients will shift one short-axis slice position during systole, thus generating a large amount of the SV. Furthermore, the SV is decreased in the patient group, which gives a larger percentage of the contribution of the most basal slice to the SV compared with the healthy groups. In controls and athletes, the position of the base of the ventricle will shift two short-axis slice positions during systole (AVPD of 16 mm and slice thickness of 8 mm). The area is smaller in the most basal slice compared with the slice below it. This explains why the second slice generates the most SV in the healthy groups.

The fact that the SV is primarily generated in the basal part of the LV has implications for cardiac imaging. The basal location of a large part of the SV underscores the importance of imaging the entire LV, including the base. Furthermore, great care must be taken to account for the AVPD when measuring the SV by planimetry.

Further studies. Heart rate and level of physical exertion may affect SVAVPD, and studies undertaken in situations with increased cardiac output would be of value. Also, it would be of interest to investigate patients with coronary artery disease, LV hypertrophy, and mitral valve disease since the relationship between longitudinal and radial function in these patients has been reported to differ from healthy subjects (24).

Limitations. AVPD is known to decrease with age (42), and in the present study, patients were older and had higher heart rates than controls. Thus part of the difference in AVPD may be related to these disparities. Three of the patients had atrial fibrillation, which has been reported to decrease the AVPD (10). The patients with atrial fibrillation, however, had a SVAVPD% and AVPD that were similar to those of the other patients, suggesting that these may not be a major factor in DCM. The contraction of the myocardial tissue as such is more complex than solely longitudinal and radial shortening. The coupling between longitudinal, helical, and circumferential myocardial fibers (3, 41) and the torsion of the myocardium during contraction (23, 35, 39) have not been studied. However, the aim of the study was not to investigate the mechanics of myocardial fibers as such. Furthermore, this study did not seek to investigate differences in efficiency of the contraction caused by the differences in ventricular geometry between the groups. However, future studies are merited to elucidate such differences.

In conclusion, the present study has demonstrated that the largest part of the SV is generated at the base of the ventricles and that longitudinal AVPD is the primary contributor to LV pumping, accounting for ~60% of the LV stroke volume. Although AVPD is less than one-half in patients with DCM compared with controls and athletes, the contribution of AVPD to LV function is maintained. This can be explained by the larger short-axis area in patients with DCM.

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