Influence of posture on left ventricular long- and short-axis shortening

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Sundblad, Patrik, and Bengt Wranne. Influence of posture on left ventricular long- and short-axis shortening. Am J Physiol Heart Circ Physiol 283: H1302–H1306, 2002.—End-diastolic volume and left ventricular stroke volume are increased in the supine compared with upright position, but the contribution of long-axis (LAS) and short-axis shortening (SAS) to these changes with change in posture has not been established. We examined long- and short-axis motion and dimensions with echocardiography in 10 healthy subjects in the upright and supine position. Long-axis length at end diastole was almost identical, whereas the diastolic short-axis diameter was increased in the supine position. At end systole, there was a decreased long-axis length and increased short-axis length in the supine vs. upright position. Both LAS and SAS were enhanced in supine vs. upright positions [LAS: 9.3 ± 2.2 vs. 15.1 ± 3.1 mm (P < 0.001); SAS: 12.7 ± 3.2 vs. 16.3 ± 2.8 mm (P < 0.001)], presumably via Starling mechanisms. LAS increased more in the lateral part of the mitral annulus than in the septal part [7.7 ± 2.6 vs. 4.0 ± 2.8 mm (P < 0.006)], which implies that the more spherical form, in the supine position, induces more stretch at the lateral free wall than in the ventricular septum. These findings support the notion that Starling mechanisms affect systolic LAS.

METHODS

Subjects. Three women and seven men participated in the study. Their height, weight, age, and body surface area were (means ± SD) 177 ± 8 cm, 72 ± 8 kg, 24 ± 2 yr, and 1.9 ± 0.2 m², respectively. All were healthy and had normal electrocardiograms, echocardiograms, and blood pressures. The experimental protocol was approved by the ethics committee at Linköping University Hospital.

Protocol and measurements. Echocardiographic images were obtained with the subjects flat on their backs or standing on a tilt board. The subjects stood for at least 3 min before echocardiographic imaging. The recordings were made during breath holding after a relaxed expiration to functional residual capacity. First, parasternal short-axis views of the LV were obtained; thereafter, apical four-chamber views were obtained. All recordings were done in duplicate for each posture.

All echocardiographic information was obtained with a GE-Vingmed System V system (Vingmed A/S; Horten, Norway) and stored on an Echopac digital storage system. Data were exported to a personal computer with software (TVIv60, Vingmed A/S) capable of postprocessing the images using anatomic M-mode (19), which is a feature that allows M-mode measurements of motion in directions that do not coincide with the direction of the beam of the echocardiographic probe. The costs of publication of this article were defrayed in part by the payment of page charges. The article must therefore be hereby marked “advertisement” in accordance with 18 U.S.C. Section 1734 solely to indicate this fact.

Achieving a normal physiological left ventricular (LV) stroke volume and ejection fraction (EF) requires shortening of both longitudinal and transverse (short-axis) inner diameters of the LV. Without the longitudinal component, normal sarcomere shortening would lead to a shortening fraction of ~12% and an EF of <30% (10, 12). Longitudinal shortening also contributes to short-axis shortening because myocardial tissue volume is noncompressible and therefore constant during contraction, whereas the outer diameter is unchanged or decreased (3, 11). Thus the displacement of myocardial tissue toward the apex caused by long-axis shortening decreases LV volume also by decreasing the inner diameter. In contrast to this, the extent of long-axis shortening is not modified by changes in the short axis.
ultrasound (Fig. 1, B and C). The following measurements were done.

First, long-axis length at end diastole and end systole was measured between the epicardial apex and the septal and lateral part of the mitral annulus, respectively (Fig. 1A). The average of these two distances was then considered as the length between the apex and atrioventricular plane.

Second, short-axis diameter in diastole and systole was measured using the anatomic M-mode (Fig. 1B). The measurements were made perpendicular to the interventricular septum at the level of the tips of the mitral leaflets.

Finally, LV long-axis function was defined as the motion of the mitral annulus. The total amplitude of atrioventricular plane motion (AVPD) as well as the atrial contribution (a wave) to the AVPD were measured (Fig. 1C).

All echocardiographic recordings were done by the same experienced operator, and the various measurements were done off-line by another person. The intra- and interinvestigator reproducibility have been shown to have a coefficient of variance ranging between 2% and 5% in these kinds of measurements obtained in our laboratory (13).

Statistics. Data are expressed as means ± SD. The difference in means was evaluated with Student’s paired two-tailed t-test to test the difference between values obtained in the supine and upright positions. Bonferroni corrections for repeated measures were used when appropriate. The level of significance was set to P < 0.05.

RESULTS

LV long-axis length at end diastole was similar in the upright and supine position (Fig. 2A). AVPD during contraction was enhanced in the supine position, which led to a shorter LV long axis at end systole in the supine compared with upright position (Fig. 2A). Both the total AVPD and the atrial contribution to AVPD (a wave) were enhanced in the supine position. There was a more prominent increase in total AVPD at the lateral part of the mitral annulus compared with the septal part [7.7 ± 2.6 vs. 4.0 ± 2.8 mm (P < 0.006)]. The increases of the a wave amplitude in the supine position were similar at the lateral and septal parts of the mitral annulus.

Short-axis dimensions were larger in the supine posture both during diastole and systole (Fig. 2B). Short-axis shortening during systole was increased in the supine compared with upright position, and fractional shortening tended to be increased (Table 1).

DISCUSSION

The present study examined the effects of posture on cardiac dimensions and contraction patterns with a special focus on the interaction between long- and

Fig. 1. A–C: echocardiographic images illustrating how the measurements were conducted. Note that the anatomic M-mode allows measurements of motions that are not in line with the beam of ultrasound. A: left ventricular (LV) long-axis length was measured from the epicardial apex to the septal and lateral part of the mitral annulus. The mean of these two measurements was regarded as the LV long-axis length. B: short-axis diameter was measured in M-mode images at end diastole and end systole. C: long-axis motion was also assessed in M-mode images. Bottom, describes how the total atrioventricular plane displacement (AVPD_{tot}) and atrial contribution (a wave) were measured.
short-axis shortening. From these results, it can be concluded that the LV of the heart is more spherical at diastole in the supine posture than in the upright posture, i.e., increased short-axis diameter, with unchanged diastolic long-axis length (Fig. 3). This is probably mainly dependent on increased diastolic filling pressure in the supine position but could also partly be due to effects on the heart and diaphragm induced by a changed gravitational vector. Similar LV long-axis length in a relaxed heart in the supine and upright position seems logical. Cardiac filling pressure is ~5 mmHg higher in the supine position compared with the upright position (18). The increased cardiac filling pressure causes similar increases in atrial and ventricular pressures at end diastole. At end diastole, the transmural flow and gradient are low, and there is no additional force, in the supine compared with upright position, that would act to displace the atrioventricular plane. At end systole, however, LV long-axis length was shorter in the supine position due to enhanced systolic AVPD, whereas the short-axis diameter was slightly increased (Fig. 2). It has previously been shown that AVPD is representative of long-axis shortening because the fibrous apex does not move in relation to the ultrasound probe during the cardiac cycle (3, 17).

We suggest that increased myocardial tension in diastole enhances atrial and ventricular contraction, as indicated by an increased wave and AVPD, and that the increased contraction is mediated via Starling mechanisms. Because sympathetic tone and heart rate, which are positively correlated to contractility, are lower in the supine compared with upright position (6) (Table 1), it is unlikely that those parameters had any influence on the increased force of contraction. Enhanced AVPD during the supine posture with an increased heart size is in agreement with recent find-

![Diagram](https://example.com/diagram.png)

Fig. 3. A schematic drawing illustrating the differences in cardiac dimensions in diastole and long-axis motion between the upright (solid lines) and supine posture (dashed lines). Note that the heart is more spherical while in the supine position; this induces both atrial and ventricular stretch in the myocardium. Long-axis length during diastole is similar in the two postures. The increased AVPD and the increased atrial contribution are presumably dependent on Starling mechanisms. Because a posture change from upright to supine will probably increase tension in the free lateral wall more than in the septal wall, it follows that the increase in contraction is greater and AVPD is more pronounced on the lateral side. RV, right ventricle; LA, left atrium; RA, right atrium.

<table>
<thead>
<tr>
<th>Variables</th>
<th>Upright</th>
<th>Supine</th>
<th>P Values</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total AVPD</td>
<td>9.3 ± 2.2</td>
<td>15.1 ± 3.1</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Total a wave</td>
<td>2.1 ± 1.3</td>
<td>4.0 ± 1.0</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Lateral AVPD</td>
<td>9.5 ± 2.3</td>
<td>17.1 ± 3.0</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Lateral a wave</td>
<td>1.9 ± 1.4</td>
<td>3.9 ± 0.9</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Septal AVPD</td>
<td>9.1 ± 2.1</td>
<td>13.1 ± 3.8</td>
<td>0.002</td>
</tr>
<tr>
<td>Septal a wave</td>
<td>2.3 ± 1.3</td>
<td>4.1 ± 1.3</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>AVP-apex distance at end diastole</td>
<td>95.9 ± 9.1</td>
<td>95.9 ± 8.4</td>
<td>NS</td>
</tr>
<tr>
<td>AVP-apex distance at end systole</td>
<td>86.6 ± 7.6</td>
<td>80.7 ± 8.0</td>
<td>0.03</td>
</tr>
<tr>
<td>LVEDD</td>
<td>42.7 ± 5.8</td>
<td>49.5 ± 5.5</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>LVESD</td>
<td>30.0 ± 4.6</td>
<td>33.2 ± 4.1</td>
<td>0.007</td>
</tr>
<tr>
<td>LVEDD – LVESD</td>
<td>12.7 ± 3.2</td>
<td>16.3 ± 2.8</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>FS</td>
<td>30 ± 7</td>
<td>33 ± 4</td>
<td>0.053 (NS)</td>
</tr>
<tr>
<td>HR</td>
<td>75 ± 11</td>
<td>68 ± 12</td>
<td>0.07 (NS)</td>
</tr>
</tbody>
</table>

Values are means ± SD. All dimensions are in millimeters except for fractional shortening (FS; %) and heart rate (HR; beats/min). AVPD, atrioventricular plane displacement; LVEDD and LVESD, left ventricular (LV) end-diastolic and end-systolic diameter, respectively; NS, not significant. FS = (LVEDD – LVESD)/LVEDD.
ings showing that AVPD correlates well with cardiac dimensions and volumes in normal subjects (2).

The increased atrial contraction is probably dependent solely on the increased preload induced by the augmented filling in the supine posture. Increased ventricular stretch, on the other hand, could depend both on increased passive filling and on the amplified atrial contraction in the supine posture, which acts to elevate the AVPD and thus induce stretch of the ventricular wall. The increased passive filling, which makes the LV more spherical, mainly affects circumferential stretch, whereas increased atrial contraction induces a longitudinal stretch. Patients with atrial fibrillation have decreased AVPD compared with age-matched controls with sinus rhythm (5), which supports the notion that atrial contraction might affect the longitudinal contraction force.

AVPD in the supine posture was more amplified at the lateral part of the mitral annulus than in the septal part. This might be due to the LV lateral free wall being relatively more stretched during increased passive filling than the LV septal wall. Augmented passive filling in the supine position affects both the right ventricle and LV, which implies that primarily the free walls of the heart are expanded, whereas the intracardiac walls such as the ventricular septum are less affected. It has previously been shown that the lateral part of the mitral annulus has a significantly larger amplitude of motion than the septal part (9, 21, 22). These studies were conducted on subjects in supine or left lateral recumbent position and are thus in agreement with the present findings. In a recent study (4), it has been shown that the epicardial part of the atrioventricular plane has a larger motion amplitude than the endocardial part. This could be due to an enhanced stretch during diastole in the epicardial layer or due to the fact that the longitudinal fibers in the epicardium reach from the atrioventricular plane all the way down to the apex, contrary to endocardial fibers (8). The effect of increased atrial contraction on ventricular wall stress is presumably similar because the amplitude of the a-wave is similar in the lateral and septal parts of the mitral annulus in both postures. Wandt et al. (21) found that the a-wave shows a linear increase in amplitude with increasing age. They proposed that this finding was coupled to the progressively decreasing rate of ventricular relaxation with age (20). The present study puts forward that increased atrial stretch during diastole, induced by the impaired ventricular relaxation, might be the mechanism behind the age-dependent increase in the atrial contribution (a wave).

LV long-axis length during systole is shorter in the supine position than in the upright position, whereas the short-axis diameter is larger. The fact that long-axis length decreases more than the short-axis diameter in absolute terms during systole in the supine position does not imply that end-systolic volume is less in the supine than upright position. This is given by the fact that the changes of the radius (r) contribute to volume according to the formula \( \pi r^2 \). Following the same line of reasoning, the increase in short-axis shortening during the supine posture is the main contributor to the increase in stroke volume rather than the increase in long-axis shortening (AVPD).

However, long-axis shortening also contributes to the short-axis shortening during systole due to the relative constancy of LV myocardial volume during the contraction; more myocardial tissue is displaced down in the ventricle in the supine position (Fig. 4), whereas the outer diameter of the heart is unchanged or slightly decreased during systole (3, 11, 14). It is well known from theoretical calculations that it is impossible to accomplish a realistic EF (60% or more) using only circumferential muscle fibers capable of shortening by only ~15%. It is necessary to decrease the long axis, with the aid of oblique and longitudinal fibers, to attain a physiological EF (12). The present study emphasizes this fact by showing that the well-known increase in LV stroke volume during the supine position compared with the upright position is at least partly achieved by increased AVPD.

Limitations. Arterial pressure was not recorded in the present experiments. However, it has repeatedly been shown that mean arterial pressure at the level of the heart is unchanged in the upright compared with supine position, whereas systolic pressure decreases and diastolic pressure increases slightly (16). It is assumed that slight changes in arterial pressure will only have limited effect on LV work in young healthy subjects.

Intrathoracic pressure (ITP) is decreased and functional residual capacity is increased in the upright vs. supine position (1). A lower ITP usually enhances venous return and cardiac output; however, in the present setting, this effect is counteracted by the prominent pooling of blood away from the thoracic compartment in the upright position, and the net effect is decreased venous return and cardiac output (18). The effect of decreased ITP on cardiac afterload is minor with changes in posture. Even though mean ITP is lower during the upright position, the intrapleural pressure gradient in the lung, from apex to base, gives an almost unchanged ITP in the basal parts and lower ITP in the apical parts of the lung (1). These gradients

Fig. 4. Illustration describing the concept of how AVPD contributes to short-axis shortening under the assumption that myocardial volume and the outer contour of the heart are constant. Thus the myocardium in the basal part of the ventricle is displaced downward toward the apex when the length of the ventricle decreases during systole. This displacement of myocardial tissue, which is incompressible, acts to increase ventricular wall thickness and decreases the inner diameter if the outer diameter is unchanged or decreased during contraction.
in ITP also makes it difficult to estimate local ITP from recordings of esophageal pressure. In summary, we did not record ITP in the current experiment, but previous work suggests that the altered ITP with a change in posture would only have a limited influence on our conclusions regarding LV function in the supine and upright positions.

Short-axis shortening is measured as the decrease in the transverse inner diameter. These measurement might have been slightly exaggerated because, during systole, there is an endocardial infolding of the trabeculae that could give an impression of increased wall thickness and decreased inner diameter (7). We acknowledge this possibility, but the slight underestimation of the short axis in systole is likely to be similar in upright and supine positions and would not have any significant effect on the conclusions in this study.

Finally, through-plane motion of the heart is an inherent problem in echocardiography. However, in the short-axis view, the control of the location of the scanning plane is quite easy with the anatomic landmarks of the mitral valves and papillary muscles. A systematic difference between supine and upright positions is therefore unlikely. Through-plane motion is less of a problem in the apical view, where this phenomenon at end expirium is very negligible.

In conclusion, the present study has shown that the LV is more spherical in both systole and diastole in the supine vs. upright posture. It is proposed that the increased myocardial stretch in the supine position enhances longitudinal contraction of the left atrium and LV, which is evidenced by increased AVPD. There is a more pronounced increase of AVPD at the lateral part of the mitral annulus, probably caused by elevated filling pressure that, in a supine subject, primarily induces stretch at the free walls of the heart, whereas intracardiac walls, such as the ventricular septum, are less affected. It is concluded that increased AVPD during the supine posture contributes to the elevation of stroke volume. Emptying of the LV depends on long- and short-axis shortening, and enhanced AVPD contributes to both.

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