Effects of supine and lateral positions on cardiac output and intracardiac pressures: an experimental study

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ABSTRACT Hemodynamic measurements in human subjects and in experimental animals are generally made in the supine position; not much attention is paid to potential beneficial or harmful effects of right or left lateral positions on cardiac output or other hemodynamic variables. To evaluate the potential influence of such positional changes on cardiac performance, we measured cardiac output and left and right ventricular pressures (with micromanometer catheters) in anesthetized experimental animals (eight dogs and nine pigs) in the supine, right lateral, and left lateral positions. Cardiac output increased from supine to left lateral (mean ± SD, 2.6 ± 0.9 to 3.1 ± 1.0 liters/min; p < .001) and from supine to right lateral positions (2.6 ± 0.9 to 3.1 ± 1.1 liters/min; p < .001). There was an associated decrease in arteriovenous oxygen saturation difference from supine to left lateral position (31 ± 8% to 24 ± 4%; p < .001) and from supine to right lateral position (32 ± 9% to 25 ± 6%; p < .001). Left ventricular systolic and end-diastolic pressures increased from supine to left lateral (128 ± 17/9 ± 2 to 147 ± 19/16 ± 4 mm Hg; both p < .001) and from supine to right lateral positions (128 ± 19/9 ± 2 to 141 ± 16/16 ± 7 mm Hg; p < .01 and p < .001, respectively). Similarly, right ventricular systolic and end-diastolic pressures also increased from supine to left lateral (30 ± 7/3 ± 2 to 38 ± 7/8 ± 2 mm Hg; both p < .001) and from supine to right lateral positions (31 ± 8/3 ± 2 to 43 ± 8/11 ± 4 mm Hg; both p < .001). Systolic and end-diastolic right ventricular pressures were significantly higher in the right lateral position than in the left (both p < .001). Heart rate did not change with positional maneuvers. Neither the sequence of positional changes nor the species of animal (dog vs pig) had any apparent influence on the results. Roentgenographic analysis of the differences in height of the right ventricle relative to the inferior vena cava suggests that changes in hydrostatic pressure may be entirely responsible for the increases in right ventricular end-diastolic pressure when animals are changed from the supine to the left or right lateral positions. We conclude that a change from supine to lateral position significantly increases intracardiac pressures and cardiac output in experimental animals. If confirmed in humans, these findings may have significant implications for the assessment of hemodynamic status of patients in intensive care unit and catheterization laboratory settings and for the treatment of patients in low-cardiac output states.

while Ueland et al.\textsuperscript{1} could not demonstrate any difference. The effect of supine and lateral positions on intracardiac pressures measured by micromanometer has not been reported to our knowledge.

The purpose of this study was to evaluate the effects of supine and left and right lateral positions on cardiac output and intracardiac pressures under controlled conditions in anesthetized animals. Subsequent studies are planned to evaluate hemodynamic changes with positional maneuvers in human subjects.

**Methods**

Eight mongrel dogs weighing 18 to 28 kg and nine young pigs weighing 29 to 37 kg were studied. The use of two species enabled assessment of whether findings were species specific. The animals were anesthetized with sodium pentobarbital intravenously (25 mg/kg) and were ventilated by a Harvard pump with a tidal volume of 15 cc/kg room air and a respiratory rate of 15/min. High-fidelity micromanometer catheters (PC-470, Millar Instruments, Houston, TX) were inserted into the left and right ventricles via the left carotid artery and the left internal jugular vein, respectively. The zero reference point of pressures was taken at mid chest with each animal supine, and high-fidelity micromanometer pressure signals were matched with pressures measured through the fluid-filled lumen of each catheter, during each supine recording. With the high-fidelity pressure signals, right and left ventricular systolic pressures were measured as the peak pressures during systole, and ventricular end-diastolic pressures were measured after the “a” wave, just before the rapid systolic rise in ventricular pressure. The pressures reported represent data averaged from at least 3 beats recorded during end-expiration. A No. 7F Swan-Ganz catheter was inserted into the pulmonary artery via the right femoral vein in each animal. Cardiac output was measured by the thermodilution method (Edwards 9520A cardiac output computer, Santa Ana, CA). The injectate was 10 ml of normal saline at a temperature of 0\degree to 1\degree C. Each cardiac output value was the mean of at least three consecutive determinations having variation of less than 10%. Oxygen saturations of systemic arterial and mixed venous blood, drawn from the left ventricle and the pulmonary artery, respectively, were measured by the reflectance oximeter method (Reflectance Oximeter, American Optical Corporation, Buffalo, NY).

**Experimental protocol.** All 17 animals were evaluated initially in the supine position. After left and right ventricular pressures had been stable for at least 30 min, cardiac output, blood oxygen saturations, and left and right ventricular systolic and end-diastolic pressures were measured. The positions of the animals were then changed from supine to left lateral (three dogs, three pigs) or from supine to right lateral (five dogs, six pigs). Lateral positions were maintained for 20 min, with continuous recordings of left and right ventricular pressures to assess time course and stability of pressure changes. Measurements of cardiac output, blood oxygen saturations, and left and right ventricular systolic and end-diastolic pressures were repeated between 10 and 20 min after change to the lateral position. The animals were then returned to the supine position, micromanometer pressures were rematched with fluid-filled pressures as necessary, and measurements were repeated between 10 and 20 min after. Positions were then changed to the alternate lateral position (left lateral in five dogs and six pigs and right lateral in three dogs and three pigs), with continuous recording of left and right ventricular pressure again to assess time course and stability of pressure changes. All measurements were repeated between 10 and 20 min after the change in position. Because of technical factors, not all measurements could be obtained in each animal.

**Additional studies.** To define the potential influence of changes in height of the inferior vena cava (venous filling reservoir) relative to the right ventricle in supine, right lateral, and left lateral positions, two additional dogs and two additional pigs were studied. Catheters were inserted into the right femoral vein and advanced into the right ventricle. Catheter position in the right ventricle was confirmed by pressure recording. Radiographs were taken in the supine, right lateral, and left lateral positions. The height of the inferior vena cava relative to the right ventricle was measured in centimeters. The measurement was corrected for magnification and was converted into equivalent millimeters of mercury by dividing the number of centimeters by the conversion factor of 1.36. Changes in height of the inferior vena cava relative to the right ventricle with alterations in position from supine to left lateral and from supine to right lateral were then computed.

To assess the possibility of position-related compression of the inferior vena cava, seven additional pigs were studied. A Swan-Ganz catheter was inserted into the right femoral vein and advanced into the right atrium, where the position of the tip was confirmed by pressure recording. With the fluid-filled catheter system, simultaneous pressures were then recorded in supine, right lateral, and left lateral positions both from the catheter tip in the right atrium and from the side-hole in the inferior vena cava.

**Table 1.** Effects of change from supine to lateral positions on hemodynamic variables (mean ± SD)

<table>
<thead>
<tr>
<th></th>
<th>n</th>
<th>Supine</th>
<th>Left lateral</th>
<th>Supine</th>
<th>Right lateral</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR (bpm)</td>
<td>17</td>
<td>117±20</td>
<td>115±18</td>
<td>116±21</td>
<td>115±19</td>
</tr>
<tr>
<td>CO (l/min)</td>
<td>16</td>
<td>2.6±0.9</td>
<td>3.1±1.0\textsuperscript{a}</td>
<td>2.6±0.9</td>
<td>3.1±1.1\textsuperscript{a}</td>
</tr>
<tr>
<td>AVO\textsubscript{SD} (%)</td>
<td>11</td>
<td>31±8</td>
<td>24±4\textsuperscript{a}</td>
<td>32±9</td>
<td>25±6\textsuperscript{a}</td>
</tr>
<tr>
<td>LVSP (mm Hg)</td>
<td>12</td>
<td>128±17</td>
<td>147±19\textsuperscript{a}</td>
<td>128±19</td>
<td>141±16\textsuperscript{a}</td>
</tr>
<tr>
<td>LVEDP (mm Hg)</td>
<td>12</td>
<td>9±2</td>
<td>16±4\textsuperscript{a}</td>
<td>9±2</td>
<td>16±7\textsuperscript{a}</td>
</tr>
<tr>
<td>RVSP (mm Hg)</td>
<td>17</td>
<td>30±7</td>
<td>38±7\textsuperscript{a}</td>
<td>31±8</td>
<td>43±8\textsuperscript{a,b}</td>
</tr>
<tr>
<td>RVEDP (mm Hg)</td>
<td>17</td>
<td>3±2</td>
<td>8±2\textsuperscript{a}</td>
<td>3±2</td>
<td>11±4\textsuperscript{a,b}</td>
</tr>
</tbody>
</table>

HR = heart rate; CO = cardiac output; AVO\textsubscript{SD} = arteriovenous oxygen saturation difference; LVSP = left ventricular systolic pressure; LVEDP = left ventricular end-diastolic pressure; RVSP = right ventricular systolic pressure; RVEDP = right ventricular end-diastolic pressure.

\textsuperscript{a}p < .01, right or left lateral vs supine.

\textsuperscript{b}p < .01, right vs left lateral.
cava located 30 cm from the catheter tip. The zero reference point of pressures was taken at mid chest in the supine position.

**Statistical analysis.** Measurements for all variables were determined to be normally distributed by the Wilkes-Shapiro test, permitting use of parametric comparison methods. Variances for each species (obtained from two-way analysis of variance) were demonstrated equivalent by F test and the data were therefore pooled. Resulting distributions were normal by the Wilkes-Shapiro test, with homogeneous variances (Levene's test). Data were then analyzed for statistical significance with an appropriate multiple-sample comparison test (two-way analysis of variance, followed by blocked Newman-Keuls test). Data were considered significant at the p < .05 level. Measurements are reported as mean ± SD.

**Results**

Hemodynamic data in supine and lateral positions are summarized in table 1. There was no significant change in any of the hemodynamic measurements between both supine positions. Heart rate, measured in all 17 animals, did not change significantly with rotation into right or left lateral positions (figure 1). Cardiac output, measured in 16 of 17 animals, increased after a change from supine to left lateral position (p < .001) and from supine to right lateral position (p < .001) (figure 2). Arteriovenous oxygen saturation difference, measured in 11 of 17 animals, decreased from supine to left lateral position (p < .001) and from supine to right lateral position (p < .001) (figure 3). In each of the 11 animals with measurements of both cardiac output and oxygen saturations, the increase in cardiac output observed with a change in position from supine to right or left lateral was associated with a corresponding decrease in arteriovenous oxygen saturation difference.

Left and right ventricular pressures, measured in 12 and 17 animals, respectively, changed within 1 min of alteration in position in each animal, reached a stable state by 1 to 5 min, and then remained stable throughout the subsequent recording period. The recorded pressures are those obtained 10 min after change of position. Left ventricular systolic pressure rose from supine to left and right lateral positions (p < .001 and p < .01, respectively) (figure 4). Left ventricular end-diastolic pressure also increased from supine to left and right lateral positions (both p < .001) (figure 5). Both right ventricular systolic and end-diastolic pressures increased from supine to left and right lateral positions (all p < .001) (figures 6 and 7). Systolic and end-diastolic right ventricular pressures were both higher in the right lateral position than in the left lateral position (both p < .001).

When percent changes from supine to left and right lateral positions were compared, they were found to be equivalent for all except three variables. The percent

![FIGURE 1](http://circ.ahajournals.org/)

**FIGURE 1.** Changes in heart rate (HR) from supine (S) to left lateral (L) and right lateral (R) positions in 17 animals. Values in dogs and pigs are shown by solid and dashed lines, respectively. Mean values are represented by the horizontal bars.

![FIGURE 2](http://circ.ahajournals.org/)

**FIGURE 2.** Changes in cardiac output (CO) from supine (S) to left lateral (L) and right lateral (R) positions in 16 animals. Values in dogs and pigs are shown by solid and dashed lines, respectively. Mean values are represented by the horizontal bars.
FIGURE 3. Changes in arteriovenous oxygen saturation difference (AVO2SD), given as differences in percent saturations, from supine (S) to left lateral (L) and right lateral (R) positions in 11 animals. Values in dogs and pigs are shown by solid and dashed lines, respectively. Mean values are represented by the horizontal bars.

FIGURE 4. Changes in left ventricular systolic pressure (LVSP) from supine (S) to left lateral (L) and right lateral (R) positions in 12 animals. Values in dogs and pigs are shown by solid and dashed lines, respectively. Mean values are represented by the horizontal bars.

FIGURE 5. Changes in left ventricular end-diastolic pressure (LVEDP) from supine (S) to left lateral (L) and right lateral (R) positions in 12 animals. Values in dogs and pigs are shown by solid and dashed lines, respectively. Mean values are represented by the horizontal bars.

FIGURE 6. Changes in right ventricular systolic pressure (RVSP) from supine (S) to left lateral (L) and right lateral (R) positions in 17 animals. Values in dogs and pigs are shown by solid and dashed lines, respectively. Mean values are represented by the horizontal bars.
increase in left ventricular systolic pressure was greater in the left lateral position (p < .05), and increases in right ventricular systolic and end-diastolic pressures were greater in the right lateral position (p < .01 and p < .05, respectively).

The results of roentgenographic analysis of catheter position are shown in figure 8. In the two dogs studied, the height of the right ventricle relative to the inferior vena cava differed by 8.4 cm with a change from supine to left lateral position, equivalent to a difference in hydrostatic pressure of 6.2 mm Hg. In the two pigs, there was an 11.3 cm difference, equivalent to an 8.3 mm Hg difference in hydrostatic pressure. From supine to right lateral position, the changes in height of the right ventricle relative to the inferior vena cava were 11.2 and 11.4 cm for dogs and pigs, respectively, equivalent to 8.2 and 8.4 mm Hg differences in hydrostatic pressure. In the seven pigs that underwent simultaneous measurements of mean right atrial and inferior vena caval pressures with fluid-filled catheters, the pressure gradients between inferior vena cava and right atrium were 3.8 ± 0.8 mm Hg (mean ± SD) in the supine position, 3.1 ± 0.6 mm Hg in the right lateral position, and 5.4 ± 0.9 mm Hg in the left lateral position (figure 9). Each of the three gradients was significantly different from the other two at the p < .001 level.

Discussion

It is well known that certain positional changes may significantly affect cardiovascular performance. Cardiac output, for example, decreases by approximately 15% to 30% when normal human subjects change from a supine position to a 70 to 75 degree head-up tilt. This decrease has been attributed to a reduction in venous return, resulting from venous pooling in the lower extremities. In normal pregnant women in the third trimester, a change from supine to lateral positions has been shown to increase cardiac output by a mean of up to 27%. Compression of the inferior vena cava by the gravid uterus in the supine position and relief of this compression upon changing to the lateral position are thought to be the physiologic basis of this

FIGURE 7. Changes in right ventricular end-diastolic pressure (RVEDP) from supine (S) to left lateral (L) and right lateral (R) positions in 17 animals. Values in dogs and pigs are shown by solid and dashed lines, respectively. Mean values are represented by the horizontal bars.

FIGURE 8. The height of the catheter tip in the right ventricle (RV) relative to the height of the catheter in the inferior vena cava (IVC) was assessed roentgenographically with animals in the supine (S), left lateral (L), and right lateral (R) positions. Measurements (in cm) have been corrected for magnification of the x-ray film. The changes in height of the right ventricle relative to the inferior vena cava with changes from supine to lateral positions were computed in centimeters and then converted into equivalent millimeters of mercury hydrostatic pressure.
FIGURE 9. Differences between mean inferior vena cava pressure (IVCP) and mean right atrial pressure (RA) in supine and lateral positions. The pressures were measured simultaneously in seven pigs by means of fluid-filled catheters.

Our experimental study in anesthetized animals yielded results different from those obtained in some previous human studies. Cardiac output, measured by the thermomilution method, rose significantly from supine to both lateral positions. Changes in arteriovenous oxygen saturation differences support the validity of the thermomilution measurements. In the studies of Ueland et al. and others in awake human beings, reflex changes may have blunted or otherwise altered the cardiac output response to lateral position, thereby preventing expression of the hemodynamic changes seen in our study.

The effects of supine and lateral positions on ventricular pressures have not, to our knowledge, been determined previously. However, several reports do describe the effects of positional changes on aortic or pulmonary arterial pressures in human beings. Newton and Eggers found no significant differences among arterial pressures recorded in the supine and left and right lateral positions, and Kennedy et al. observed no significant positional differences in pulmonary arterial systolic pressure. Again, the differences between these findings and our own may reflect reflex changes in the conscious human subject to maintain constant arterial pressure. In addition, however, in these previous studies, pressures were measured with fluid-filled catheters. When such catheters are used, the zero reference point should optimally be changed whenever a patient’s position is altered during the course of a study. Since identification of the proper zero reference point in each position may be difficult, actual changes in intravascular pressures may not be detected. To exclude the potential for such error, we used high-fidelity micromanometers that do not require that the zero reference point be changed in each position. In our experimental study, both systolic and end-diastolic left and right ventricular pressures were significantly higher in lateral positions than in the supine position.

The differences in height of the right ventricle relative to the inferior vena cava, as determined from roentgenographic studies, suggest that increases in hydrostatic pressure may be entirely responsible for the increases in right ventricular end-diastolic pressure when animals are changed from the supine to the left lateral position and from the supine to the right lateral position. The increase in hydrostatic pressure would be expected to increase right ventricular diastolic filling and stroke volume, accounting for the increases in cardiac output and left ventricular filling pressure in the lateral positions. The hydrostatic pressure differences, however, are somewhat larger than the changes...
in right ventricular end-diastolic pressure observed in the 17 animals studied with micromanometer-tipped catheters. This would raise the possibility of variable compression of the inferior vena cava, perhaps by the liver, in the three positions. Indeed, the pressure gradients recorded between the inferior vena cava and right atrium with fluid-filled catheters, which are insensitive to changes in hydrostatic pressure, support the possibility of compression and suggest that compression may be greatest in the left lateral position.

Several limitations of this study should be emphasized. First, there are no data with regard to chamber volumes. Although thermodilution cardiac output measurements and changes in arteriovenous oxygen saturation differences suggest that increases in preload are responsible for increases in left and right ventricular end-diastolic pressures in the lateral positions, other contributing factors, such as changes in ventricular compliance, variable compression of the right ventricle by the left, and alterations in right or left ventricular afterload, cannot be excluded. Second, inferior vena cava flows were not measured. Therefore the possibility of variable, position-related compression of the inferior vena cava cannot be assessed further. Third, although we can conclude that positional changes in two animal species significantly affect intracardiac pressures and cardiac output, it has not been determined that similar changes occur in other animal species. In particular, whether and under what circumstances positional changes affect cardiac performance in human beings must be clarified. If similar hemodynamic changes are confirmed for man, they may help to explain several clinical observations, including the preference of some patients with congestive heart failure or angina for certain positions (trepopnea) and the deterioration in arterial oxygen saturation that often occurs when patients with bilateral pulmonary disease assume the left lateral position.  

References
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