Effects of Posture on Exercise Performance

Measurement by Systolic Time Intervals

By DAVID H. SPODICK, M.D., AND VERONICA M. QUARRY-PICOTT, M.S.

SUMMARY

Because posture significantly influences cardiac performance, the effects of moderate supine and upright ergometer exercise were compared on the basis of proportional (+37%) rate increments over resting control. Supine exercise produced significant decreases in left ventricular ejection time (LVET), pre-ejection period (PEP), and isovolumic contraction time (IVCT). Ejection time index (ETI) and "corrected ejection time" (LVETc) did not change significantly. Upright exercise produced greater decreases in PEP and LVET, but despite the rate increase there was no change in LVET, which resulted in sharp increases in ETI and LVETc. The discordant directional effects on LVET and its rate-correcting indices between the two postures were consistent with hemodynamic studies demonstrating lack of stroke volume change during supine exercise and increased stroke volume over control during light to moderate upright exercise. Concordant effects on PEP and IVCT are consistent with the isotropic effect of exercise in both postures with an additional Frank-Starling effect postulated during upright exercise.

Additional Indexing Words:
Noninvasive  Supine exercise  Upright exercise

NONINVASIVE polygraphic methods are increasingly applied in physiologic and diagnostic studies of cardiac performance in response to exercise loads.1-5 Study protocols vary widely among investigators because of different forms of exercise (step-tests, treadmills, bicycle ergometers, leg raising, isometrics), different duration of work, different workloads, progressive loading vs staged or single loads, work-load vs rate-"-paced" testing, and exercise in the supine vs sitting or standing posture.

Despite its intrinsic artificiality, supine exercise has proved useful and is widely utilized because this is the common position for both cardiac catheterization and most noninvasive procedures. Moreover, following upright exercise subjects have been placed supine for recording,4 5 although it has been shown that accurate recording while erect is feasible.1

Posture can profoundly influence cardiocirculatory performance at rest and stroke volume changes from rest to exercise6-7 particularly at lighter levels,8-10 so that one cannot compare the results of different exercise challenges unless this variable is accounted for. Indeed, before differing postural effects were fully appreciated, such a fundamental consideration as the role of stroke volume in the exercise response was the subject of some controversy among physiologists.6, 8, 9

Because it has proved technically possible to make noninvasive polygraphic recordings in almost any posture,1, 11, 12 we decided to compare comparable exercise stresses on the bicycle ergometer while supine and upright (sitting). As the index stress we chose a 50-watt load for supine ergometry because postural differences are greatest during milder exercise.6-10 Moreover, mild supine exercise has proved to be most practical for diagnostic work.7, 10, 13 Because of expected postural differences in resting control heart rates, and because, for both steady state and nonsteady state exercise, heart rate changes reflect most sensitively the overall cardiac load,10, 14-17 the two exercise postures were compared on the basis of proportionate rate increments. Thus, the increase over control heart rate achieved while supine was matched by pedaling to the same proportionate increment while upright.

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POSTURE AND SYSTOLIC TIME INTERVALS

Material and Methods

We studied ten active, healthy nonathletically trained, male volunteers, aged 22 to 34, in each of two postures. None was remarkably obese, none was receiving medications for even minor complaints and none smoked before the studies, each of which was performed between 8:30 and 10:00 a.m. with the subject in the postabsorptive state. Subjects pedaled a Collins bicycle ergometer on two different mornings. One effort involved pedaling supine against a load of 50 W for 3 min, after which recordings were made. The other involved pedaling the same ergometer in the upright position to a heart rate (HR) increment over control HR equal to that achieved at the fixed load at which level recordings were made. Graphic data from each subject were recorded from body surface sensors, including: ECG-limb lead and precordial (sternal) electrodes; PCG-Sanborn #21050 A/B microphone adhesively sealed and belted at the mesoapex; carotid pulse—via Sanborn #21051D pulse wave pickup applied over the right neck. Data were extracted separately and independently from recordings in supine and sitting postures.

Rate Corrections

Because there is no accepted exercise-rate regression for left ventricular ejection time, this was corrected for rate in two ways—by the previously reported slope factor to yield the ejection time index (ETI = LVET +1.2 HR) and by the square root of cycle length to yield “corrected ejection time” (LVETc). PEP and IVCT are not corrected for rate because these are not significantly affected by rate per se.2 4 18-21

Definitions

Cycle Length

The R-R interval of the ECG, expressed in milliseconds.

q

Initiation of the QRS complex in lead II, whether a Q wave or the beginning of the R upstroke. In practice, because of occasional baseline artifacts, a large number of complexes were inspected and the q-to-R peak (or R upstroke to R peak) time ascertained, so that curves could be timed from the precisely registered R peak; the q-to-R peak was then added in the calculations; q was thus the zero point for all measurements in each cycle.

Im

Timing of the first high-frequency (“mitral”) oscillation of the first heart sound.

CARu

Timing of the onset of the rapid portion of the carotid (CAR) upstroke.

II

Timing of the first high-frequency (aortic) component of the second heart sound.

CARfn

Timing of the carotid incisura.

Calculations

Using standard calculations for systolic time intervals1 we used the above measurements to determine the following: Electromechanical Systole (EMS); Pulse Transmission Time (PTT); Pre-ejection Period (PEP) including isovolumic contraction time (IVCT) and q-Im interval; Left Ventricular Ejection Time (LVET); Ejection Time Index (ETI) as LVET +1.2 HR; “Corrected Ejection Time” (LVETc) as LVET/√R-R, Pre-Ejection Period divided by Left Ventricular Ejection Time (PEP/LVET).

Results

The results are tabulated in tables 1 (supine exercise) and 2 (sitting exercise), and in figures 1 and 2 and table 3 which summarize and compare both kinds of exercise.

Table 1

<table>
<thead>
<tr>
<th>Systolic Time Intervals with Supine Exercise</th>
<th>Control</th>
<th>Exercise</th>
<th>Change</th>
<th>%</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>Mean</td>
<td>Mean</td>
<td>%</td>
<td></td>
</tr>
<tr>
<td>HR (b/min)</td>
<td>63.3</td>
<td>86.7</td>
<td>+23.4</td>
<td>+37.0</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>LVET (msec)</td>
<td>292.0</td>
<td>255.4</td>
<td>-36.6</td>
<td>-12.5</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>ETI</td>
<td>367.9</td>
<td>359.4</td>
<td>-8.5</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>LVETc</td>
<td>298.6</td>
<td>306.4</td>
<td>-7.8</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>PTT (msec)</td>
<td>35.6</td>
<td>36.6</td>
<td>+1.0</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>PEP (msec)</td>
<td>108.7</td>
<td>78.4</td>
<td>-30.3</td>
<td>-28.2</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>q-Im (msec)</td>
<td>67.3</td>
<td>59.8</td>
<td>-7.5</td>
<td>-10.5</td>
<td>&lt;0.02</td>
</tr>
<tr>
<td>IVCT (msec)</td>
<td>41.4</td>
<td>18.6</td>
<td>-22.8</td>
<td>-55.1</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>PEP/LVET</td>
<td>0.372</td>
<td>0.307</td>
<td>-0.065</td>
<td>-17.5</td>
<td>&lt;0.01</td>
</tr>
</tbody>
</table>

Abbreviations: HR = heart rate; LVET = left ventricular ejection time; ETI = ejection time index; LVETc = corrected ejection time; PTT = pulse transmission time; PEP = pre-ejection period; q-Im = interval between initiation of QRS complex and first high frequency oscillation of first heart sound; IVCT = isovolumic contraction time.
Table 2

Systolic Time Intervals and Upright Exercise

<table>
<thead>
<tr>
<th></th>
<th>Control Mean</th>
<th>Control SD</th>
<th>Exercise Mean</th>
<th>Exercise SD</th>
<th>Change Mean</th>
<th>Change %</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR (b/min)</td>
<td>82.4</td>
<td>10.30</td>
<td>112.3</td>
<td>17.93</td>
<td>+29.9</td>
<td>+36.2</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>LVET (msec)</td>
<td>225.2</td>
<td>13.27</td>
<td>228.6</td>
<td>18.83</td>
<td>+3.4</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>ETI</td>
<td>324.0</td>
<td>9.66</td>
<td>363.4</td>
<td>10.11</td>
<td>+39.4</td>
<td>+12.2</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>LVETc</td>
<td>262.6</td>
<td>11.45</td>
<td>309.7</td>
<td>15.18</td>
<td>+47.1</td>
<td>+17.9</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>PTT (msec)</td>
<td>37.0</td>
<td>8.88</td>
<td>37.2</td>
<td>6.55</td>
<td>+0.2</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>PEP (msec)</td>
<td>127.0</td>
<td>17.72</td>
<td>79.6</td>
<td>11.19</td>
<td>-47.4</td>
<td>-37.3</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>q-Im (msec)</td>
<td>48.0</td>
<td>16.55</td>
<td>49.2</td>
<td>11.67</td>
<td>+1.2</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>IVCT (msec)</td>
<td>79.6</td>
<td>17.88</td>
<td>30.4</td>
<td>8.10</td>
<td>-49.2</td>
<td>-61.8</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>PEP/LVET</td>
<td>0.563</td>
<td>0.073</td>
<td>0.349</td>
<td>0.044</td>
<td>-0.214</td>
<td>-38.0</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Abbreviations: See table 1.

Supine Protocol

Significant (P < 0.001 to P < 0.02) changes included heart rate (HR), which increased 23.4 b/min (+37.0%), and decreases in LVET (-36.6 msec; 12.5%), PEP (-30.3 msec; 28%), q-Im (-7.5 msec; 10.5%), IVCT (-22.8 msec; 55.1%) and PEP/LVET (-0.065; 17.5%). Ejection time index and corrected ejection time failed to change significantly. PTT did not change.

Upright Protocol

The mean exercise heart rate (112.3 b/min) represented an increase of 36.2% (P < 0.001) over resting control (82.4 b/min) which matched the proportionate increase during supine exercise. There were significant (P < 0.001) increases in ETI (+39.4; 12.2%) and LVETc (+47.1; 17.9%) while LVET, PTT and q-Im did not change, and sharp, significant decreases occurred in PEP (-47.4 msec; 37.3%), IVCT (-49.2 msec; 61.8%) and PEP/LVET (-0.214; 38.8%).

Supine vs Upright Exercise

Resting control data varied predictably according to posture,6, 8, 9, 12, 15 (tables 1 and 2). As compared with resting supine control, resting upright values were higher for HR, PEP, IVCT and PEP/LVET, and lower for LVET, ETI, and LVETc.

Comparison of exercise results is summarized in table 3 and figures 1 and 2.

Discussion

Supine Exercise

The mild increment in HR was consistent with the mild exercise level.8, 9, 13–17 It was accompanied by a parallel fall in LVET. The principal determinants of LVET are HR and stroke volume (SV).

![Supine vs Upright Exercise](http://circ.ahajournals.org/)

![Supine vs Sitting Exercise](http://circ.ahajournals.org/)

Supine vs upright (sitting) ergometer exercise at proportional rate increments. HR = heart rate; LVET = left ventricular ejection time; ETI = ejection time index; LVETc = corrected ejection time. Dots = means; bars = standard deviation.

Supine vs upright (sitting) exercise. PTT = pulse transmission time; PEP = pre-ejection period; IVCT = isovolumic contraction time; q-Im = timing of first heart sound. Dots = means; bars = standard deviation.
Removal of the influence of heart rate from the LVET by two different methods—i.e., by the rate-regression slope factor (yielding the ETI) and by dividing LVET by the square root of cycle length (LVETc)—resulted in virtually perfect stability of each of these indices, indicating no significant change in stroke volume. This was not surprising since lack of change in SV is, in fact, typical of submaximal supine exercise. The marked reduction of PEP was due virtually entirely to a fall in isovolumic contraction time. This is consistent with increased velocity of shortening of contractile elements owing to the inotropic effect of exercise, which is independent of rate changes (which, in any case, do not by themselves affect PEP and IVCT to any significant degree as demonstrated by atrial pacing). Decrease in the PEP/LVET relationship during supine exercise was a function of the relatively more marked fall in the numerator, consistent with the improved inotropic state. The very small (7.5 msec) but statistically significant fall in q-Im suggests that, at least during supine exercise, increased velocity of contraction may begin to advance in midlateral valve closure (which occurs 15-25 msec before Im is generated).

**Upright Exercise**

The mean heart rate increase (approximately 30 b/min) was pretargeted to match the proportionate mean increment during the supine exercise phase. Despite this rise, LVET did not change (table 2 and fig. 1). The consequent sharp rise in ETI and LVETc therefore indicate increased stroke volume. Indeed, increased stroke volume over resting control is quite typical of upright exercise at this level. (An apparent lack of change in LVETc in the study of Pouget et al. may be explained by the fact that their subjects were actually supine for recording data). While q-Im failed to change significantly, the marked fall in PEP (owing to decreased IVCT) reflects the inotropic influence of exercise. Since LVET was stable, the sharp fall in PEP/LVET was also entirely due to this change in PEP.

**Upright vs Supine Exercise**

The essential differences between the supine and upright exercise at 37% HR increments over resting control HR were in the behavior of LVET and its rate-corrected indices. This behavior was to be expected from physiologic studies consistently showing no change in stroke volume during supine exertion and increased stroke volume during upright exertion. Because changes in PEP and IVCT can reflect both inotropic effects and the Frank-Starling relationship, both of these factors may have influenced their shortening during upright exercise, since the increased SV would necessarily require increased venous return per stroke, and hence, increased end-diastolic ventricular volume over resting control. By contrast, this would be unlikely during the essentially fixed SV of supine exercise (in which posture the resting ventricles already operate at fairly maximum volume). Therefore, sharply reduced PEP and IVCT during supine exercise may represent mainly, if not entirely, positive inotropic change in the absence of a significant Frank-Starling effect. By contrast, the change in IVCT during upright exercise probably represents a summation of inotropic with Frank-Starling influences.

**References**

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