Left Ventricular Wall Stress During Leg-Press Exercise Performed With a Brief Valsalva Maneuver*

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Study objectives: To assess the effects of leg-press (LP) exercise performed with a brief (2 to 3 s) Valsalva maneuver on left ventricular (LV) systolic function and LV wall stress in five healthy men (mean ± SD age, 27.6 ± 2.9 years).

Methods and measurements: Subjects performed submaximal (80% one repetition maximum [1RM], 337.9 ± 109.1 kg; 95% 1RM, 400.6 ± 129.8 kg) and maximal LP exercise (420 ± 118.6 kg) during which central arterial pressure, intrathoracic pressure, and two-dimensional echocardiographic analysis of LV systolic function and LV wall stress were measured.

Results: Compared with baseline, LP exercise resulted in an increase in intrathoracic pressure (baseline, 1.7 ± 2.9 mm Hg; 80% 1RM, 111.7 ± 20.2 mm Hg; 95% 1RM, 112.2 ± 21.1 mm Hg; 100% 1RM, 111.0 ± 21.3 mm Hg; p < 0.05) and LV end-systolic pressure (baseline, 120.0 ± 13.2 mm Hg; 80% 1RM, 255.3 ± 15.3 mm Hg; 95% 1RM, 255.3 ± 12.2 mm Hg; 100% 1RM, 242.8 ± 16.5 mm Hg; p < 0.05) with no changes in LV end-systolic transmural pressure (baseline, 118.3 ± 12.6 mm Hg; 80% 1RM, 140.0 ± 6.1 mm Hg; 95% 1RM, 143.1 ± 16.1 mm Hg; 100% 1RM, 131.8 ± 29.7 mm Hg; p > 0.05), LV end-systolic wall stress (baseline, 91.7 ± 20.2 kilodyne/cm²; 80% 1RM, 78.0 ± 24.4 kilodyne/cm²; 95% 1RM, 81.4 ± 25.3 kilodyne/cm²; 100% 1RM, 85.9 ± 20.1 kilodyne/cm²; p > 0.05), or LV fractional area change (baseline, 0.48 ± 0.03; 80% 1RM, 0.52 ± 0.11; 95% 1RM, 0.53 ± 0.06; 100% 1RM, 0.52 ± 0.05; p > 0.05).

Conclusion: LP exercise performed with a brief Valsalva maneuver is not associated with an alteration in LV wall stress or LV systolic function in healthy young men.

Key words: left ventricular systolic function; leg-press exercise; Valsalva maneuver; wall stress

Abbreviations: LP = leg press; LV = left ventricular; LVESTMP = LV end-systolic transmural pressure; 1RM = one repetition maximum

Submaximal or maximal resistance exercise is associated with a transient abrupt elevation in arterial pressure (ie, > 300 mm Hg).1,2 A widely held belief in sport cardiology is that the heightened pressure load and concomitant left ventricular (LV) wall stress associated with resistance exercise may be a potent stimulus in altering LV morphology.3 However, we have found that short- (< 5 years) or long-term (> 18 years) resistance training was not associated with alterations in LV dimensions or mass in elite male and female resistance-trained athletes.4,5 The reason for this observation is not clear. Although it is possible that the actual resistance-mediated pressure load on the LV may be of insufficient magnitude or too brief in duration to alter LV morphology,4 other mechanisms may be acting to attenuate the LV response.

LV wall stress is directly related to LV transmural pressure (ie, intravascular pressure minus extravascular pressure) and LV geometry (ie, end-systolic cavity to myocardial area ratio). Alterations in either variable may affect LV wall stress. Data from the study of Galanti et al6 suggest that the LV alters its shape such that the increased pressure load during isometric exercise (without a Valsalva maneuver) is dissipated over a larger wall thickness, resulting in no alteration in LV wall stress. Lentini et al7 found that the increased intravascular pressure during resistance exercise was secondary to the elevated in-
trathoracic pressure associated with performing a Valsalva maneuver; however, the pressure to which the heart was actually exposed (ie, LV transmural pressure) was lower than that predicted by the intravascular pressure alone. These observations suggest that acute heart-lung interactions may occur during resistance exercise to reduce LV wall stress. However, the acute effects of resistance exercise performed with a brief (2 to 3 s) Valsalva maneuver would not be associated with an increase in LV end-systolic transmural pressure (LVSTMP) or LV wall stress.

**MATERIALS AND METHODS**

The subjects for this study were five healthy men (mean ± SD age, 27.6 ± 2.9 years; height, 175.5 ± 7.3 cm; weight, 79.2 ± 6.4 kg). Four subjects were experienced resistance-trained athletes (three power lifters and one rower), whereas the remaining subject performed recreational resistance training. Ethical approval for this study was obtained from the University of Alberta Faculty of Medicine ethics committee for human experimentation, and informed consent was obtained before study participation.

**Maximal Muscular Strength**

Maximal muscular strength was assessed as a voluntary one-repetition maximum (1RM) using the bilateral inclined LP exercise. After a suitable warm-up, the subjects performed a light set of 8 to 10 repetitions. This was followed by a second set of six to eight repetitions with a heavier weight (50 to 60% 1RM). The subjects then completed a subsequent two repetition sets of increasing load until only one repetition could be performed despite attempting a second repetition. This testing protocol usually required a total of five to seven sets. The LP exercise was standardized so that all subjects lowered the weight to a knee joint angle of 90° before initiating the concentric phase of the lift. Three to five minutes of rest was allowed between sets, and verbal encouragement was consistently provided. All subjects returned to the research laboratory for a second session, during which time they performed submaximal (80% and 95% 1RM) and maximal LP exercise with hemodynamic, intrathoracic, and transesophageal echocardiographic monitoring (Fig 1). Each set of LP exercise was performed with a brief (2 to 3 s) Valsalva maneuver.

**Central Arterial Pressure Monitoring**

Central arterial pressure was measured with a 5F pressure-tip catheter (Millar Instruments; Houston, TX). After infiltrating the skin with lidocaine, the left brachial artery was cannulated with a 6F vascular sheath using a modified Seldinger technique. The catheter was then positioned in the descending aorta at the level of the LV, and the signal was amplified (Electronics for Medicine; Torrance, CA) and stored digitally on a four-channel recorder (EP LAB; Quinton; Bothell, WA). The pressure transducer was calibrated at baseline and before and after every LP set.

**Intrathoracic Pressure Monitoring**

Esophageal pressure (a surrogate for intrathoracic pressure) was measured with a pressure-tip catheter (Millar Instruments) that was inserted orally and positioned in the mid-esophagus. The catheter was attached to a control unit (TCB 500; Millar Instruments), and the signal was amplified (Electronics for Medicine) and stored digitally on a four-channel recorder (EP LAB; Quinton). The catheter was calibrated at baseline and before and after every exercise set.

**LV Imaging**

LV imaging was performed with a commercially available ultrasound instrument (Sonos 2500; Hewlett Packard; Avondale, PA) with a 5-MHz transesophageal transducer. Before oral intubation, the gag reflex was abolished with topical lidocaine. The transesophageal probe was then inserted through the mouth and positioned into the stomach (Fig 2). LV two-dimensional images were obtained from the transgastric short-axis view at the level of the mid-papillary muscles and were averaged over three cardiac cycles to obtain the following measures: end-systolic cavity area (the smallest endocardial area), end-diastolic cavity area; end-systolic total cavity area (the total area enclosed by the epicardium), and end-systolic myocardial area (equal to the end-systolic total area minus the end-systolic cavity area).

**Calculations**

LV cavity areas, central arterial pressure, and intrathoracic pressure measurements were obtained at rest (baseline) and during the last repetition of the 80% and 95% 1RM sets as well as during the maximal LP set and were used to calculate the following:

1. LVSTMP, where LVSTMP (mm Hg) equals LV end-systolic pressure minus intrathoracic pressure. (In the absence of aortic stenosis or LV outflow tract obstruction, diastolic central arterial pressure has been shown to approximate LV end-systolic pressure. Because none of our subjects had
aortic stenosis or LV outflow obstructions, dicrotic central arterial pressure was used as a surrogate for LV end-systolic pressure.)

2. LV stroke area, equal to the end-diastolic cavity area minus the end-systolic cavity area.10

3. LV systolic function was measured as fractional area change, equal to the stroke area divided by the end-diastolic cavity area.10

4. LV contractility, equal to LVESTMP divided by the end-systolic cavity area.

5. LV end-systolic meridional wall stress, equal to 

\[ 1.33 \times \text{LVESTMP} \times \text{end-systolic cavity area divided by end-systolic myocardial area}. \]

**Statistical Analysis**

Comparison of all measured variables between baseline and LP exercise was performed with a repeated measures analysis of variance using Statistica (Statsoft; Oklahoma City, OK) software. If a significant F ratio was found, then a Scheffé multiple comparison test was performed. The α level was set a priori at \( p < 0.05 \).

**RESULTS**

The brachial arterial catheterization and esophageal oral intubations were not associated with any complications. In addition, all subjects tolerated the exercise protocol without any side effects. Because of technical difficulties, intrathoracic pressure was not obtained during the 95% and 100% sets in one subject. In addition, complete data were not obtained in another subject, who was excluded from the analysis.

**Maximal Bilateral LP 1RM**

The subjects’ maximal bilateral LP 1RM was equivalent to 420 ± 118.6 kg. During the pressure measurement exercise session, the subjects lifted 337.9 ± 109.1 kg for 9.3 ± 2.2 repetitions (80% 1 RM) and 400.6 ± 129.8 kg for 4 ± 0 repetitions (95% 1 RM) as well as the above 1 RM set.

**Acute Hemodynamic Responses During LP Exercise**

Compared with baseline, submaximal and maximal LP exercise performed with a brief (2 to 3 s) Valsalva maneuver was associated with a significant increase in peak heart rate, systolic pressure, diastolic pressure, and intrathoracic pressure (Table 1). LVESTMP was not significantly altered during LP exercise compared with baseline and was significantly lower than the simultaneous LV end-systolic pressure during exercise (Table 1).

**Effects of LP Exercise on LV Cavity Areas, Systolic Function, and End-Systolic Wall Stress**

LV end-diastolic and end-systolic cavity areas were significantly smaller during submaximal and maximal LP exercise compared with baseline (Table 1). However, no significant change was found for end-systolic myocardial area, end-systolic cavity to myocardial area ratio, LV contractility, stroke area, fractional area change, or LV end-systolic wall stress (Table 1).

**DISCUSSION**

The major finding of this study was that LP exercise performed with a brief (2 to 3 s) Valsalva maneuver was not associated with alterations in LV systolic function or LV end-systolic wall stress.

**Acute Hemodynamic Responses During LP Exercise**

The finding that LP exercise was associated with extreme elevations in heart rate, systolic pressure, diastolic pressure, and intrathoracic pressure is similar to that previously reported for younger individuals.1,2,7,11 The mechanism responsible for our hypertensive BP response was secondary to the
increased intrathoracic pressure associated with performing a brief (2 to 3 s) Valsalva maneuver. Table 1 shows that LV end-systolic pressure was higher during LP exercise compared with baseline measures; however, LVESTMP was not elevated above baseline values. This finding is consistent with earlier studies that demonstrated that positive swings in intrathoracic pressure (i.e., Valsalva maneuver) were transmitted directly to the arterial vasculature as an increase in systolic pressure; however, the pressure to which the heart was actually exposed was not elevated above resting values. Therefore, the heart, unlike the peripheral arteries, is protected by the heightened intrathoracic pressure associated with a brief Valsalva maneuver performed alone or when incorporated during LP exercise and should not be discouraged during this form of exercise.

### Effects of LP Exercise on LV Systolic Function

The effect of heavy resistance exercise performed with a brief (2 to 3 s) Valsalva maneuver on LV systolic function has not been well studied. Lentini et al., using transthoracic echocardiography, found that LP exercise was associated with a decline in end-diastolic volume, end-systolic volume, and stroke volume. Despite the attenuated cardiac volume response, LV contractility was higher during LP exercise, resulting in a concomitant elevation in LV systolic function. Our finding that LP exercise was associated with a decrease in end-diastolic cavity area, end-systolic cavity area, and stroke area is consistent with the findings of Lentini et al. The mechanism responsible for the reduced stroke area is secondary to the increased resistance to venous return associated with performing a brief Valsalva maneuver. For example, positive swings in intrathoracic pressure elevate the downstream pressure for venous return, resulting in a decline in LV preload. Despite the attenuated preload, an acute increase in LV contractility and concomitant decline in end-systolic cavity area resulted in no alteration in LV systolic function. The finding that LV systolic function was not altered during LP exercise performed with a brief (2 to 3 s) Valsalva maneuver is consistent with that previously reported. Together, these findings suggest that heavy LP exercise performed with a brief (2 to 3 s) Valsalva maneuver will not likely result in an acute deterioration in LV systolic function in healthy young men.

### Effects of LP Exercise on LV End-Systolic Wall Stress

The finding that LP resistance exercise performed with a brief (2 to 3 s) Valsalva maneuver was not associated with an alteration in LV end-systolic wall stress (Table 1) is consistent with an earlier study that reported that athletes with physiologic LV hypertrophy had reduced resting LV end-systolic wall stress. Colan et al. assessed resting LV end-systolic wall stress in power lifters and age-matched sedentary individuals. Despite the athletes’ higher end-systolic pressure, their calculated LV end-systolic wall stress was 31% lower than the sedentary controls. The attenuated end-systolic wall stress was secondary to an

### Table 1—Short-term Hemodynamic and LV Responses During LP Exercise Performed With a Brief (2 to 3 s) Valsalva Maneuver

<table>
<thead>
<tr>
<th>Variable</th>
<th>Baseline</th>
<th>80% 1RM</th>
<th>95% 1RM</th>
<th>100% 1RM</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR, beats/min</td>
<td>96.0 ± 8.1</td>
<td>166 ± 23.6</td>
<td>150.3 ± 22.7</td>
<td>135 ± 6.4</td>
</tr>
<tr>
<td>PSP, mm Hg</td>
<td>146.5 ± 15.2</td>
<td>297.0 ± 10.7</td>
<td>307.3 ± 20.6</td>
<td>290.2 ± 24.0</td>
</tr>
<tr>
<td>PDP, mm Hg</td>
<td>92.1 ± 9.1</td>
<td>226.0 ± 11.5</td>
<td>236.0 ± 16.3</td>
<td>220.6 ± 16.5</td>
</tr>
<tr>
<td>LVESTMP, mm Hg</td>
<td>120.0 ± 13.2</td>
<td>251.0 ± 15.3</td>
<td>255.3 ± 12.2</td>
<td>242.8 ± 16.5</td>
</tr>
<tr>
<td>LVESP, mm Hg</td>
<td>115.3 ± 12.6</td>
<td>140.0 ± 6.1</td>
<td>143.1 ± 16.1</td>
<td>131.8 ± 29.7</td>
</tr>
<tr>
<td>ITP, mm Hg</td>
<td>1.7 ± 2.9</td>
<td>111.7 ± 20.2</td>
<td>112.2 ± 21.1</td>
<td>111.0 ± 21.3</td>
</tr>
<tr>
<td>EDCA, cm²</td>
<td>22.2 ± 1.4</td>
<td>16.7 ± 4.6</td>
<td>16.5 ± 5.2</td>
<td>17.3 ± 4.3</td>
</tr>
<tr>
<td>ESTA, cm²</td>
<td>32.2 ± 2.7</td>
<td>27.1 ± 3.6</td>
<td>27.7 ± 2.9</td>
<td>26.8 ± 4.4</td>
</tr>
<tr>
<td>ESCA, cm²</td>
<td>11.6 ± 1.2</td>
<td>7.8 ± 1.4</td>
<td>7.6 ± 2.2</td>
<td>8.3 ± 2.1</td>
</tr>
<tr>
<td>ESMA, cm²</td>
<td>20.6 ± 1.9</td>
<td>19.3 ± 3.1</td>
<td>20.1 ± 1.9</td>
<td>18.5 ± 2.8</td>
</tr>
<tr>
<td>ESCA/ESMA</td>
<td>0.57 ± 0.06</td>
<td>0.41 ± 0.09</td>
<td>0.38 ± 0.12</td>
<td>0.45 ± 0.08</td>
</tr>
<tr>
<td>LVSA, cm²</td>
<td>10.6 ± 0.8</td>
<td>8.9 ± 4.0</td>
<td>8.9 ± 3.4</td>
<td>9.0 ± 2.5</td>
</tr>
<tr>
<td>LV contractility, mm Hg/cm²</td>
<td>10.2 ± 1.7</td>
<td>17.7 ± 3.8</td>
<td>18.0 ± 5.4</td>
<td>15.3 ± 5.8</td>
</tr>
<tr>
<td>FAC</td>
<td>0.48 ± 0.03</td>
<td>0.52 ± 0.11</td>
<td>0.53 ± 0.06</td>
<td>0.52 ± 0.05</td>
</tr>
<tr>
<td>WS, kilodyne/cm²</td>
<td>91.7 ± 20.2</td>
<td>78.0 ± 24.4</td>
<td>81.4 ± 25.3</td>
<td>85.9 ± 20.1</td>
</tr>
</tbody>
</table>

*Values are mean ± SD. EDCA = end-diastolic cavity area; ESCA = end-systolic cavity area; ESMA = end-systolic myocardial area; ESTA = end-systolic total area; FAC = fractional area change; HR = heart rate; ITP = intrathoracic pressure; LVESP = left ventricular end-systolic pressure; LVSA = left ventricular stroke area; PDP = peak diastolic pressure; PSP = peak systolic pressure; WS = wall stress.  
†p < 0.05 vs baseline.  
‡p < 0.05 vs LVESTMP performed at the same exercise intensity.
increased LV wall thickness, which decreased the systolic load per fiber. Galanti et al. confirmed and extended these findings. In their study, they assessed the acute LV end-systolic wall stress response during submaximal (30% maximal voluntary contraction) isometric handgrip exercise (without a Valsalva maneuver) in athletes and sedentary individuals. Compared with rest, isometric exercise was associated with a 25% increase in systolic pressure in both groups. However, LV end-systolic wall stress was not altered in the athletes during isometric exercise (compared with rest), whereas it increased by 28% in the sedentary subjects. The attenuated end-systolic wall stress was secondary to an acute increase in LV wall thickness accompanied by a decline in cavity dimension (equivalent to a decrease in the end-systolic cavity area to myocardial area ratio), which effectively counteracted the increase in systolic pressure.

The first of two possible mechanisms responsible for our finding of no change in LV end-systolic wall stress during LP exercise was that this form of exercise was not associated with a significant alteration in LVESTMP. Therefore, the pressure to which the heart was actually exposed during this form of exercise was similar to that measured at baseline. Second and more important, acute alterations in LV geometry (ie, reduced end-systolic cavity area to myocardial area ratio) occurred during LP exercise that prevented an increase in LV end-systolic wall stress. Therefore, it appears that transient alterations in LV geometry (ie, increase in myocardial area) occur during submaximal isometric handgrip exercise (without a Valsalva maneuver) or during very heavy LP exercise performed with a brief (2 to 3 s) Valsalva maneuver that minimize LV end-systolic wall stress.

A limitation of the present study was that esophageal pressure was measured as a surrogate for intrathoracic pressure. However, Kingma et al. have shown that esophageal pressure underestimates pericardial pressure (the “true” surrounding pressure of the heart) during positive swings in intrathoracic pressure. Therefore, the actual LVESTMP and concomitant LV end-systolic wall stress during LP exercise performed with a brief (2 to 3 s) Valsalva maneuver may have been lower than our reported values.

In summary, LP exercise performed with a brief (2 to 3 s) Valsalva maneuver is associated with short-term heart-lung interactions that prevent an alteration in LV systolic function or LV end-systolic wall stress.

References

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