Left ventricular response in healthy young men during heavy-intensity weight-lifting exercise

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Lentini, Anthony C., Robert S. McKelvie, Neil McCartney, Charles W. Tomlinson, and J. Duncan MacDougall. Left ventricular response in healthy young men during heavy-intensity weight-lifting exercise. J. Appl. Physiol. 75(6): 2703–2710, 1993.—We examined cardiac volumes (using echocardiography), intra-arterial blood pressure (BP), and intrathoracic pressure (ITP) in healthy males performing leg press exercise to failure at 95% of their maximum dynamic strength. Compared with preexercise, during the lifting phase of exercise, end-diastolic volume (EDV; 147 ± 8 to 103 ± 7 ml) and end-systolic volume (ESV; 54 ± 5 to 27 ± 4 ml) decreased (P < 0.05); heart rate (82 ± 6 to 143 ± 5 beate/min), systolic BP (160 ± 6 to 270 ± 21 Torr), diastolic BP (91 ± 2 to 180 ± 18 Torr), ITP (0.8 ± 0.8 to 57.8 ± 24 Torr), and peak systolic BP/ESV (3.0 ± 0.3 to 11.0 ± 1.5 Torr/ml) increased (P < 0.05); and stroke volume decreased (94 ± 3 to 77 ± 4 ml; P < 0.05). Full knee extension was associated with most values returning to preexercise levels except for ESV (38 ± 7 ml), heart rate (130 ± 9 beats/min), and ITP (−12.5 ± 2.1 Torr). During the lowering phase, significant decreases in EDV to 105 ± 14 ml and ESV to 27 ± 7 ml were observed with increases in systolic BP to 207 ± 23 Torr, diastolic BP to 116 ± 8 Torr, and SBP/ESV to 10.0 ± 2.5 Torr/ml. Stroke volume decreased to 78 ± 9 ml (P > 0.05). Thus rapid changes in cardiac volumes, contractility, and pressure occur during weight lifting that are related to different phases of the lift.

echocardiography; left ventricular transmural pressure; left ventricular afterload

RESISTANCE (WEIGHT-LIFTING) exercises are an integral part of the training regimen for many athletes and are recommended as part of a comprehensive fitness program for healthy adults (1). This form of exercise has been shown to increase muscle mass and strength (9, 26), to produce a decrease in body fat (9, 11, 15), and in some cases to increase aerobic capacity (7, 9, 26). Therefore, weight lifting is considered to be a beneficial form of exercise for athletes, healthy individuals of all ages (9, 11, 15), and, more recently, patients with coronary artery disease (16, 21, 22).

Although a number of investigations have examined the cardiovascular responses to isometric or static exercise (2, 13, 27, 28), muscle contraction during weight lifting consists of both a static and a dynamic component and thus can be expected to produce a different hemodynamic response. Most studies of the hemodynamics of weight lifting have involved healthy young adults and athletes, with measurements performed after exercise (7, 9, 26), whereas only a few have examined the response during resistance exercise in healthy individuals (20, 23) or patients with coronary artery disease (14, 22, 29). These latter studies have noted a phasic blood pressure (BP) response with an increase in pulse pressure during exercise that differs from that observed during static exercise (20, 23).

In a study where intra-arterial BP was measured in experienced weight lifters performing near-maximal leg press exercise to fatigue (20), significant increases in systolic, diastolic, and pulse pressures (up to 480/360 Torr) were observed. Under resting and dynamic exercise conditions it is generally accepted that the magnitude of the stroke volume (SV) relates to the magnitude of the pulse pressure. The extent to which this applies during weight lifting is not known, but if it does, the mechanisms by which the heart is able to maintain or augment SV in a situation where both peripheral resistance is increased and cardiac filling is decreased are particularly intriguing. In addition, we have noted that the performance of a brief Valsalva maneuver is an integral and unavoidable component of maximal or near-maximal contractions. The increased intrathoracic pressure that results from this maneuver exaggerates the increase in BP and would be expected to compromise cardiac filling (19).

It has been shown that echocardiography can be used to noninvasively assess cardiac function during exercise (12, 27). In a recent report where echocardiography was used to assess changes in cardiac function immediately after completion of moderate-resistance exercise [60% of one repetition maximum (1 RM)], the results indicated an increase in SV and contractility (5). However, these data must be interpreted cautiously because the measurements were made immediately after exercise, and it is known that the systemic arterial BP decreases rapidly when lifting is terminated (29). Moreover, in this study the arterial BP was not measured, thus making it difficult to determine to what extent the changes were due to alterations in preload, afterload, or contractility.

To our knowledge there have not been any previous studies that have examined the cardiac volumes during weight lifting throughout the complete period of the lift. Furthermore, it would appear that the results of the cardiovascular responses during isometric exercise cannot be extrapolated to weight lifting. The present study using
experienced weight lifters as subjects was undertaken to evaluate the cardiovascular responses to very-heavy-resistance exercise by simultaneously recording arterial BP, intrathoracic pressure (ITP), and cardiac volumes during exercise.

METHODS

Subjects. Five healthy males aged 23 \pm 1.5 (SD) yr, height 181 \pm 8.4 cm, and weight 92 \pm 21 kg were recruited for the study. They were all experienced weight lifters who had been training continuously for 5.5 \pm 0.9 yr and had previously been determined to be echogenic during weight lifting. The purpose, procedures, and risks were explained, and written consent was freely obtained from the subjects. The study was approved by the Hamilton General Hospital’s ethics committee.

Exercise protocol. Subjects’ 1 RM values for double-leg press were determined on a Global Gym apparatus (Downsview, Ontario, Canada) before the study day. Subjects were stabilized in the apparatus during lifting by using their left hand to hold the support handle on the seat and with an adjustable Velcro strap positioned securely around their waist and attached to the seat. The right arm was allowed to rest freely by their side as not to interfere with the recording of brachial intra-arterial BP from this arm. A 1 RM was determined by having the subjects perform single repetitions with progressively heavier weights, resting 2–3 min between attempts. The heaviest weight that subjects could lift once through a complete range of movement was considered to be their 1 RM.

On the day of the study, the intra-arterial and intraesophageal catheters were positioned and subjects were secured in the seat of the weight-lifting apparatus. The exercise protocol consisted of two sets of a double-leg press exercise to failure at 95% of their 1 RM. The subjects were encouraged to avoid deep inhalations while performing the Valsalva maneuver during the leg press exercise. The subjects were all able to complete at least 10 repetitions at this intensity.

Echocardiographic measurements and analysis. A Hewlett-Packard Sonos 1000 Ultrasound Imaging System (model 77030A, Hewlett-Packard, Andover, MA) with a handheld 2.5-MHz transducer was employed in this study. Two-dimensional echocardiographic images were recorded on a 0.5-in. VHS videocassette for later playback and analysis on a Sony VHS 1000 videotape recorder. A short-axis view of the left ventricle at the mitral valve level was recorded from the left parasternal position in the third or fourth intercostal space during one of the exercise sets. The apical four-chamber view was recorded from the left apical position in the eight or ninth intercostal space during the other exercise set. The recording of the echocardiographic views during each exercise set was randomized to control for order effects. Timing of the images was synchronized with the BP and intraesophageal pressure by an event marker on both recorders. Preexercise echocardiographic measurements were taken immediately before the onset of the exercise set. The exercise measurements were made over either of the last two repetitions with images obtained during lifting (concentric), point of greatest knee extension (lockout), and lowering (eccentric) phases of exercise.

Analysis was carried out after the study by trackball on monitor by using the accompanying Sonos 1000 on-board computer. End-diastolic images were determined by matching the onset of the QRS complex on the electrocardiograph with the corresponding image, and end-systolic images were those that coincided with the frame having the minimum dimension near the end of the T wave. The end-diastolic volumes (EDV) and end-systolic volumes (ESV) were computed by using a variation of the Bullet formula. This model assumes the left ventricle has a half-ellipsoid half-cylindrical geometry. Calculation of cardiac volumes required obtaining dimensions from the short-axis and apical four-chamber echocardiographic views during two different exercise sets. The systolic blood pressures (SBP) measured during recording of the short-axis and four-chamber views were matched, so echocardiographic dimensions could be obtained to calculate cardiac volumes. Matching of the pressures was accepted when there was no greater than a 5-Torr difference between the two SBP measurements associated with comparable diastolic and intrathoracic pressures.

Measurements were calibrated for each frame against known values.

Direct BP measurement. After infiltration of the subject’s skin with 0.5 ml of 2% Xylocaine (Astra Pharmaceuticals), a 20-gauge Angiocath catheter (Parke-Davis, Sandy, UT) was placed percutaneously into the right brachial artery. The catheter was connected to a disposable Transpac pressure transducer (model 42589–01, Sorenson Research, Salt Lake City, UT) for the direct measurement of intra-arterial BP. The arterial BP signal was transmitted to a strain gauge excitation amplifier (model 11–4163–01, Gould Instruments, Cleveland, OH) and then relayed to a strip chart recorder (6-channel oscillographic chart recorder; Gould Brush 260 model) for instantaneous recording and to an FM tape recorder (Hewlett-Packard 3964A) for later analysis.

This system had a linear response from 0 to 500 Torr and was calibrated against a mercury manometer at 0 and 200 Torr before each subject was studied. The reported SBP and diastolic blood pressures (DBP) were the average of the two matched pressures.

ITP measurement. ITP was measured as intraesophageal pressure by a Gaeltech catheter-tip pressure transducer (model 16 CT/S, Medical Instruments, Hackensack, NJ) inserted through the nasal passage into the esophagus to the midsternal level. The signal was transmitted through an amplifier (model 4163–01, Gould) and then recorded onto an adjacent channel of the strip chart recorder used for BPs and onto FM tape. Before each subject, the intraesophageal pressure manometer was calibrated against a mercury manometer at 0 and 180 Torr. The reported intrathoracic pressures were the average of the measurements that corresponded to the matched SBPs.

Derived variables. SV was determined by calculating the difference between EDV and ESV. The percent ejection fraction was defined as (SV/EDV) \times 100. Peak SBP/ESV ratio was determined by dividing the peak SBP by the ESV (10, 25). Left ventricular systolic trans-
mural pressure (TMP) \( (6) \) was expressed as the SBP (Torr) minus the ITP. Although brachial arterial pressure was used in this calculation, we realize that corresponding left ventricular pressure would have been somewhat lower. Mean arterial pressure was calculated as being equivalent to diastolic pressure plus one-third of the pulse pressure. Cardiac output was considered to be the product of SV and heart rate. Total peripheral resistance was calculated by dividing the mean arterial pressure by cardiac output.

**Statistical analysis.** Comparisons of measures at rest, lifting, lockout, and lowering were made by using a one-way repeated measures analysis of variance. When significant \( F \) values were obtained, a Newman-Keuls test was used to identify the location of specific differences. Linear regression was performed by the least-squares method. Statistical significance was accepted at \( P < 0.05 \). All data are presented as means \( \pm \) SE unless otherwise indicated.

**RESULTS**

Typical intra-arterial and esophageal pressure traces for a subject performing leg presses to failure are presented in Fig. 1. In this example, during the latter repetitions, BPs reached \( \sim 390/290 \) Torr (Fig. 1) with intrathoracic pressures as high as 185 Torr (Fig. 1).

**Arterial blood pressure and total peripheral resistance.** Compared with preexercise, there was a significant increase in both SBP (160 \( \pm \) 6 to 270 \( \pm \) 21 Torr) and DBP (91 \( \pm \) 1.7 to 183 \( \pm \) 18 Torr) during the lifting phase (Fig. 2). These BPs decreased significantly at the lockout phase of the lift (SBP 176 \( \pm \) 15 Torr; DBP 107 \( \pm \) 9.5 Torr) and then increased significantly again during the lowering phase (SBP 207 \( \pm \) 22.8 Torr; DBP 116 \( \pm \) 8.3 Torr). SBP values during the lifting phase were higher than those reached during the lowering phase, but this was not statistically significant (Fig. 2).

Compared with preexercise, there was a significant increase in mean arterial pressure (114 \( \pm \) 3 to 212 \( \pm \) 19 Torr) during the lifting phase (Table 1). The mean arterial pressure during the lockout (130 \( \pm \) 11 Torr) and lowering (146 \( \pm \) 12 Torr) phases tended to be greater than preexercise, but this was not statistically significant (Table 1).

In contrast, compared with preexercise, pulse pressure (65 \( \pm \) 7 Torr) was significantly higher during both the lifting (87 \( \pm \) 8 Torr) and lowering (91 \( \pm \) 18 Torr) phases of exercise. There was no significant difference in pulse pressure between preexercise and the lockout (69 \( \pm \) 10 Torr) phases.

Compared with preexercise, there was an increase \( (P < 0.05) \) in total peripheral resistance (15 \( \pm \) 1 to 19 \( \pm \) 1 Torr \( \cdot \) \( 1^{-1} \) \( \cdot \) min) during the lifting phase (Table 1). The total peripheral resistance during the lockout (10 \( \pm \) 1 Torr \( \cdot \) \( 1^{-1} \) \( \cdot \) min) phase was significantly less than preexercise (Table 1). Total peripheral resistance during the lowering phase (15 \( \pm \) 1 Torr \( \cdot \) \( 1^{-1} \) \( \cdot \) min) was not signifi-
FIG. 2. Systolic and diastolic blood pressure responses to double-leg press exercise at different phases (preexercise, lifting, lockout, and lowering) of exercise. Data are means ± SE. ■, systolic blood pressure; □, diastolic blood pressure. *P < 0.05 compared with preexercise; † P < 0.05 compared with lockout; # P < 0.05 compared with lowering.

significantly different from preexercise (Table 1). The change (exercise – preexercise pulse pressure) in pulse pressure tended to track total peripheral resistance, but the correlation was not significant (Fig. 3A).

**ITP and TMP.** Compared with preexercise (0.8 ± 0.8 Torr), ITP increased during the lifting (58 ± 25 Torr) and lowering (15.5 ± 9.6 Torr) phases, but these changes were not statistically significant (Fig. 4). TMP during lockout (−12.5 ± 2.1 Torr) was significantly lower compared with preexercise (Fig. 4). TMP increased significantly compared with rest during the lifting phase of the exercise (155 ± 5 to 206 ± 18 Torr) but not during lockout or lowering (Fig. 4). The change (exercise – preexercise pulse pressure) in pulse pressure also tended to track ITP, but the correlation was not significant (Fig. 3B).

**Heart rate (HR) response.** HR during lifting increased significantly above preexercise values (82 ± 6 to 143 ± 5 beats/min; Fig. 5). HR during the lockout (130 ± 9 beats/min) and lowering (131 ± 9 beats/min) phases were significantly greater than preexercise but similar to the lifting phase.

**Cardiac volumes.** Compared with preexercise there was a significant decrease in EDV (147 ± 8 to 103 ± 7 ml) during the lifting phase (Fig. 6). EDV increased during the lockout phase to 141 ± 13 ml, which was not significantly different from the value obtained at preexercise (Fig. 6). A significant decrease in EDV to 105 ± 14 ml was observed during the lowering phase (Fig. 6).

Compared with preexercise there was a statistically nonsignificant decrease in ESV from 54 ± 5 to 27 ± 4 ml during the lifting phase (Fig. 6). ESV increased during the lockout phase to 38 ± 7 ml but was still significantly different from the preexercise value. A decrease in ESV was observed during the lowering phase of the exercise, and this was found to be significantly lower than the preexercise and lockout values (Fig. 6).

Compared with preexercise there was a statistically nonsignificant decrease in the SV from 94 ± 3 to 77 ± 4

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**TABLE 1. Mean arterial pressure, cardiac output, and total peripheral resistance responses during the lifting, lockout, and lowering phases of single-leg press exercise.**

<table>
<thead>
<tr>
<th></th>
<th>Preexercise</th>
<th>Lifting</th>
<th>Lockout</th>
<th>Lowering</th>
</tr>
</thead>
<tbody>
<tr>
<td>MAP, Torr</td>
<td>114±3</td>
<td>212±19*††</td>
<td>130±11</td>
<td>146±12</td>
</tr>
<tr>
<td>Cardiac output, l/min</td>
<td>7.8±0.5</td>
<td>11.0±0.6*</td>
<td>13.1±0.6*</td>
<td>10.0±0.5*††</td>
</tr>
<tr>
<td>TPR, Torr·l·min</td>
<td>15±1</td>
<td>19±1*††</td>
<td>10±1*</td>
<td>10±1†</td>
</tr>
</tbody>
</table>

Values are means ± SE. MAP, mean arterial pressure; TPR, total peripheral resistance. * P < 0.05 compared with preexercise; † P < 0.05 compared with lockout; †† P < 0.05 compared with lowering.
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FIG. 4. Mean left ventricular transmural (TMP) and intrathoracic pressure (ITP) responses to double-leg press exercise at different phases (preexercise, lifting, lockout, and lowering) of exercise. Data are means ± SE. ○, Mean left ventricular transmural pressure; ●, mean intrathoracic pressure. * P < 0.05 compared with preexercise; † P < 0.05 compared with lockout.

ml during the lifting phase (Fig. 6). The SV increased to 108 ± 9 ml during the lockout phase and was not significantly different from the preexercise value (Fig. 6). During the lowering phase of the exercise SV decreased to 78 ± 9 ml, which was significantly different from that observed during the lockout phase (Fig. 6).

The average preexercise cardiac output was 7.8 ± 0.5 l/min and increased significantly during the lifting phase to 11.0 ± 0.6 l/min (Table 1). A further increase (P < 0.05) in cardiac output to 13.1 ± 0.6 l/min was observed during the lockout phase (Table 1). Compared with the lockout phase, cardiac output decreased significantly during lowering to 10.0 ± 0.5 l/min but was found to be similar to the lifting phase and greater (P < 0.05) than the preexercise period (Table 1).

FIG. 5. Heart rate responses to double-leg press exercise at different phases (preexercise, lifting, lockout, and lowering) of exercise. Data are means ± SE. * P < 0.05 compared with preexercise.

FIG. 6. Left ventricular volume responses to double-leg press exercise at different phases (preexercise, lifting, lockout, and lowering) of exercise. Data shown are means ± SE. ●, End-diastolic volume; ○, stroke volume; □, end-systolic volume. * P < 0.05 compared with preexercise; † P < 0.05 compared with lockout.

Indexes of left ventricular myocardial contractility. Compared with preexercise there was a significant increase in the peak SBP/ESV ratio from 3.0 ± 0.3 to 11.0 ± 1.5 Torr/ml during the lifting phase of exercise (Fig. 7A). The ratio declined to 5.2 ± 1.0 Torr/ml during the lockout phase. During the lowering phase this ratio increased to 9.7 ± 2.5 Torr/ml and was significantly greater than the preexercise value (Fig. 7A).

Compared with preexercise, ejection fraction increased significantly from 64 ± 1.5 to 75 ± 2.6% during the lifting phase (Fig. 7B). It remained elevated throughout the lockout and lowering phases and was significantly different compared with preexercise but not significantly different from that during lifting (Fig. 7B).

DISCUSSION

This study examined changes in intra-arterial BP, ITP, and cardiac volumes at three points during double-leg press exercise. The measurements were made during the concentric, lockout, and eccentric phases of weight-lifting exercise.

MacDougall et al. (20) have described weight-lifting exercise as a series of near-maximal static contractions to overcome the inertia of the weight interspersed by forceful dynamic concentric and eccentric contractions of the muscle group. Therefore, weight lifting can be defined as a static-dynamic form of exercise. The changes in cardiac volumes, BP, and contractility observed in this study were consistent with this description. Previous studies have examined the cardiovascular responses to both static and dynamic exercise (2, 13, 27). In this study, during the lifting phase, the hemodynamic response was similar to that described for static exercise. There was a significant increase in SBP and DBP (Fig. 2) and in HR (Fig. 5) and a decline in cardiac volumes, including SV (Fig. 6). During the lockout phase there was a significant decline in BP (Fig. 2) and HR (Fig. 5) and an increase in cardiac volumes to control values (Fig. 6). These changes are more consistent with dynamic exercise. During the
lowering phase, cardiac volumes tended to decrease (Fig. 6), whereas HR (Fig. 5) and BP (Fig. 2) tended to increase, although not to the same extent observed during lifting. These changes again resembled those found during static exercise.

Cardiac volumes were measured in this study by using a biplane echocardiographic technique that required combining views from two different sets of exercise. We do not consider this to be a serious limitation, however, because the echocardiographic image from each set was selected only if the corresponding SBPs were within 5 Torr. Also the echocardiographic dimensions used to determine the volumes showed comparable relative directional changes during exercise. Although under these conditions such a technique might be expected to affect absolute units of measurement, it does not affect conclusions based on the directional changes of these data.

**SV response during weight lifting.** The large increases in SBP and DBP and pulse pressure in the present study are similar to those in a previous study of weight lifters performing the same type of exercise (20). In that study it was suggested that the increased pulse pressure might represent an increased SV and that this might be the result of increased venous return caused by the muscle pumping action with weight lifting. A recently published study that used echocardiography also suggests that SV increases during weight lifting, but this conclusion was based on postexercise measurements (5). Miles et al. (23) used impedance cardiography to examine changes in SV during leg extension exercise and reported a decrease in SV during the concentric and eccentric phases of muscle contractions. They also found a significant increase in total peripheral resistance. The results of the present study are consistent with those of Miles et al., in that during the concentric and eccentric phases SV tended to decrease, and extends their findings by demonstrating a return to the preexercise value during the lockout phase. Therefore, the observed increase in pulse pressure is not related to an increase in SV. Another possibility is that the increase in pulse pressure might be the result of the increased total peripheral resistance that we found during the lifting phase (Table 1) and as has been documented by others (23). However, in the present study no significant relationship was found between the change in pulse pressure during exercise and the total peripheral resistance (Fig. 3A). Furthermore, no significant relationship was found between the change in pulse pressure during exercise and the ITP (Fig. 3B).

**Comparison of weight lifting to isometric exercise.** The results of this study can be directly compared with those from a recent study by Sullivan et al. (27), who also used echocardiography to examine left ventricular function in isometric exercise. In their study subjects performed sustained maximal upright isometric exercise using nearly total body muscle contraction. Isometric exercise was associated with an initial decrease in SV and ejection fraction, with a subsequent increase later during the exercise. There was also an increase in mean arterial pressure, HR, left ventricular EDV, and ESV. There was no change observed in the peak SBP/ESV relationship throughout exercise. The initial decrease in ejection fraction that they reported is consistent with previous studies that examined the cardiac response to isometric dead lift using radionuclide angiography (28). In contrast, the present study, in which the subjects performed double-leg press weight lifting to failure at 95% of 1 RM, found changes in left ventricular volumes with each phase of the lift. There was also an increase in pulse pressure during the lift, with changes in the peak SBP/ESV relationship that tended to vary with the phase of the lift. These results indicate rapid changes in cardiac volume and contractility that relate to different phases of the lift. The increase in myocardial contractility, documented in the present study, is consistent with a previous report of an increase in contractility measured by systolic time intervals during leg extension weightlifting exercise (29). Therefore, it would appear that the response to weight lifting is quite different from that observed during isometric exercise, and the results from isometric studies cannot be extrapolated to weight-lifting exercise.

**Role of central motor command regulating the hemodynamic response during weight lifting.** Previous studies have suggested that the amount of central motor com-
mand (effort) is responsible for the increase in BP and HR during exercise (8, 19, 24). On the basis of this, one can hypothesize that the magnitude of the cardiovascular response would increase as the amount of effort required to lift a weight is increased. It is well known that the amount of force that a muscle can generate during a maximum concentric contraction (lifting phase) is less than that which can be developed during maximum eccentric contraction (lowering phase) (17). Thus, while lifting a given absolute load, our subjects would be exerting a greater effort during lifting than lowering and, of course, a greater effort during both lifting and lowering compared with lockout.

Our data support this concept because the changes in BP were related to the phase of the lift, with the greatest BPs observed during the concentric phase when effort would have been greatest. The peak SBP/ESV relationship, used to assess left ventricular contractility (10, 25), also changed in concert with the level of effort, suggesting that contractility was influenced by the central motor command. The changes in cardiac volumes throughout the lift may have been due to changes in central motor command, but the associated role of alterations in venous impedance and the influence of the skeletal muscle pump function cannot be assessed from the results of this study. Muscle chemoreflexes were probably not responsible for these changes because they were too rapid, occurring over the period of one lift, and muscle mechanoreceptors were probably not significantly involved because the force produced by the muscle remained constant (8).

Role of ITP in modulating left ventricular afterload. Left ventricular afterload may be defined as the tension, force, or stress acting on the fibers in the ventricular wall after the onset of shortening (3). The law of Laplace states that tension is directly proportional to TMP and the radius. In most conditions, the arterial pressure represents TMP because ITP is usually quite low. Therefore, arterial pressure can be considered to be the main determinant of afterload (3). However, this is not always the case, and large changes in ITP can influence afterload because this variable affects left ventricular TMP. In this study the large increase in SBP (Fig. 2) was associated with a large increase in ITP (Fig. 4). This resulted in a lower left ventricular TMP than would be predicted based only on the arterial BP measurement. These results are similar to a previous study that found large increases in SBP and ITP during this type of lifting exercise (20). The results from this study and the previous study by MacDougall et al. (19) might explain why left ventricular hypertrophy has not been consistently found in weight-trained athletes despite repeated exposures to very high SBPs during training sessions (4, 18, 30). It is commonly recommended that the Valsalva maneuver not be performed during weight-lifting exercise. However, the results of this study would suggest that increasing ITP by performing a brief Valsalva maneuver during heavy weight lifting is probably advisable to mitigate the effect that large increases in SBP have on left ventricular TMP.

Summary. The major significance of this study is that it is the first to document the cardiac volumes and arterial BP and ITP responses to leg press weight-lifting exercise throughout the complete cycle of one lift. Rapid changes in these variables are observed throughout the course of a single lift and are apparently related to the varying amount of effort required during each phase of the lift. The large increases in pulse pressure observed during this type of exercise do not indicate an increase in SV. Performance of a brief Valsalva maneuver may be beneficial in offsetting the increase in SBP that occurs during lifting, thus limiting the increase in myocardial TMP and preventing the development of left ventricular hypertrophy.

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