Continuous Measurement of Left Ventricular Performance During and After Maximal Isometric Deadlift Exercise

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Background. Isometric exercise produces a reflex increase in arterial blood pressure that is proportional to the intensity and mass of muscle used during contraction. Little is known about the transient effects of heavy weight lifting on left ventricular performance. In this study, we measured continuous changes in left ventricular performance during maximal large-muscle isometric exercise using the standing deadlift position.

Methods and Results. Ten healthy young men performed serial deadlifts at 50% of maximal voluntary effort for 90 seconds and 100% of maximal effort for 30 seconds. Echocardiographic imaging (apical four-chamber view), arterial blood pressure (brachial artery catheter), and electrocardiographic monitoring were recorded throughout the deadlift and for 30 seconds of recovery. Aortic flow velocity was also monitored during a separate series of deadlifts. During 100% maximal deadlift, mean arterial pressure increased from 108±4 to 164±6 mm Hg. Left ventricular ejection fraction declined initially (from 57±2% to 49±3%) at 15 seconds into the lift and recovered (56±1%) due to significant increases in end-diastolic volume (104±11 ml to 132±16 ml) by the end of the lift. The peak systolic pressure/end-systolic volume ratio did not change during the deadlift. After cessation of the deadlift, mean arterial pressure declined precipitously (to 88±4 mm Hg) within 5 seconds and gradually returned to baseline after 30 seconds. Left ventricular performance indexes all increased significantly during the recovery phase (ejection fraction to 68±3%, peak systolic pressure/end-systolic volume ratio to 5.9±0.9). Findings were qualitatively similar for the 50% deadlift.

Conclusions. During an intense isometric deadlift, left ventricular performance declines initially but is restored by the Frank-Starling mechanism. Upon release of the deadlift, increased left ventricular performance develops in conjunction with a rapid decrease in arterial pressure. The combined effects of increased wall stress during the lift phase and enhanced contractility during the release phase probably contribute to left ventricular hypertrophy associated with repetitive weight training. (Circulation 1992;85:1406–1413)

KEY WORDS • isometric exercise • echocardiography • left ventricle

Isometric exercise produces a characteristic pressor increase in arterial blood pressure causing both systolic and diastolic pressure to increase continually during skeletal muscle contraction and to fall abruptly upon relaxation.1–3 In normal subjects, the somatic pressor response is mediated by increases in heart rate and cardiac output with little or no change in stroke volume or systemic vascular resistance.3–5 These cardiovascular responses are initiated by central (cortical) recruitment of muscle tension and subsequently facilitated by afferent chemoreflexes arising from contracted muscle tissue.6,7 This provides continual input to medullary cardiovascular control centers that is proportional to cortical motor activity and the degree of local ischemia and secondary metabolic changes produced by muscle contraction.8 Thus, the magnitude of increase in blood pressure and heart rate during isometric exercise is determined by the relative intensity of muscle contraction (percent of maximum voluntary contraction), the size or mass of muscle groups used during contraction, and the duration that contraction is maintained.9 Recent studies have reported arterial pressure values exceeding 350/250 in weight lifters performing heavy repetitive isometric lifting.10

The effect of isometric exercise and corresponding increases in systemic arterial pressure on left ventricular performance has traditionally been studied during submaximal intensity with small-muscle-group isometric exercise (e.g., isometric handgrip maintained at 30–50% maximum voluntary contraction). Most available reports have shown little or no change in ventricular performance under these conditions in normal subjects.11–13 However, little is known about left ventricular performance during sustained maximal intensity isometric exercise using combined large-muscle groups, which is the type of effort frequently performed by weight lifters, other athletes, and laborers.

We recently reported that a significant decrease in radionuclide left ventricular ejection fraction occurred
in healthy subjects during maximal isometric exercise using the standing deadlift position. This mode of isometric exercise was selected because radionuclide angiographic imaging could be performed during sustained whole-body muscle contraction. However, we were concerned that these initial results did not provide a complete description of a potentially complex pattern of change in left ventricular performance because of the inherent limitations of radionuclide angiographic imaging during short-term, non-steady-state conditions. In addition, left ventricular angiograms were not obtained during the release phase after cessation of the deadlift.

In the present study, we used Doppler echocardiography to measure beat-by-beat changes in left ventricular performance along with simultaneous intra-arterial blood pressure during the upright deadlift maneuver and during the postdeadlift recovery period. Our objectives were 1) to determine whether the decreases in left ventricular performance identified by radionuclide angiography were sustained throughout the deadlift, 2) to evaluate left ventricular performance indexes during the rapid decline in arterial pressure immediately after the release of deadlift and, 3) to compare these responses during submaximal (50%) and maximal (100%) deadlift intensities.

Methods

Subjects

Ten healthy young men (age, 27±1 year) with no history of cardiovascular disease were recruited by advertisement from the University Hospital Sports Medicine Clinic (University of Wisconsin, Madison). Before participation, all subjects signed a consent form for the protocol approved by the University of Wisconsin Clinical Science Center Human Subjects Committee. All subjects had high-quality normal standing and recumbent baseline echocardiograms and normal resting electrocardiograms. None were on medications or performance-enhancing drugs.

Isometric Deadlift

Isometric deadlifting exercise was performed on a force platform consisting of an adjustable T-bar and chain connected to a Wheatstone bridge strain-gauge system with outputs to a digital display and a Gould (model 7) three-channel chart recorder. Force platform calibration showed a linear output from 0 to 200 kg. Each subject was instructed on the proper technique of lifting, with the feet shoulders-width apart, the knees at 15–20° of flexion, the hands grasping the bar shoulders-width apart in an overhand grip with the elbows in full extension, and the fingers locked between the bar and the thighs.

A preliminary 3-second maximal deadlift was performed to establish the force transducer output level for 100% maximum voluntary effort. One half of this output was used for the 50% maximal effort deadlift. After maximal deadlift force production was determined, the subject assumed an erect posture on the force platform, and after a 4–5-minute period of recovery, baseline blood pressure, echocardiographic, and Doppler studies were recorded.

A series of sustained deadlift exercise trials was then performed. Submaximal (50%) deadlifts were maintained for 90 seconds and maximal (100%) deadlifts were held for 30 seconds. The 50% maximal deadlift sequence was performed first because initial studies indicated that subjects became excessively fatigued when the 100% deadlift series was performed first. During the deadlift, subjects were required to reach target force production within 5 seconds and instructed to maintain this level within ±5 kg for the duration of the lift phase. Subjects were continually instructed to maintain slow rhythmic breathing and to avoid the Valsalva maneuver. A two-dimensional echocardiogram of the left ventricle was obtained during the first submaximal or maximal deadlift, and Doppler sampling of aortic flow velocity was performed during the second deadlift. In some instances, a third deadlift was required for optimal data acquisition.

The recovery phase began with abrupt cessation of muscle contraction. The subject was instructed to drop the crossbar into the hands of an investigator and to continue to maintain the semisquatting deadlift position. This allowed uninterrupted echocardiographic data acquisition and resulted in a more extreme drop in arterial blood pressure. After completion of each deadlift, the subject was allowed to rest in a seated position for 4–5 minutes or until heart rate and blood pressure returned to baseline values.

Blood Pressure Measurement

Blood pressure was continuously measured through an indwelling 20-gauge brachial artery catheter coupled by a 20-cm polyethylene extension tubing to a Gould-Statham P50 pressure transducer that was maintained at right atrial level. The transducer system showed a linear static calibration response from 0 to 300 mm Hg and a sinusoidal frequency response that was flat to 25 Hz with a damping coefficient of 0.25. Arterial blood pressure was continuously recorded on the Gould recorder at a paper speed of 10 mm/sec. Systolic and diastolic pressures were calculated from the numerical average of consecutive arterial pulse pressures within a 5-second interval at selected points described below (see “Data Analysis” section). Mean arterial pressure was calculated as mean arterial pressure = diastolic + 1/3(systolic − diastolic) pressures. Total peripheral resistance was calculated as total peripheral resistance = mean arterial pressure/cardiac output (see below).

Esophageal Pressure Measurement

Intrathoracic pressure changes were monitored in three additional subjects to determine whether the breathing pattern used during deadlifting produced a Valsalva-like influence on arterial blood pressure. A balloon-tipped PE 240 polyethylene catheter was inserted into the midesophagus via the nasopharynx. The balloon was inflated to 1.5 mL and pressure was monitored with a Gould-Statham P50 transducer. Intrathoracic pressure transmission was verified by measuring esophageal pressure during voluntary Valsalva maintained at 40 mm Hg mouth pressure. In each case, the esophageal pressure was in agreement with mouth gauge pressure. Esophageal pressure was then measured during 50% maximal and 100% maximal deadlift using the protocol described previously.
Cardiac Imaging

Left ventricular imaging was continuously recorded in the apical four-chamber view using a Hewlett-Packard 77020A ultrasound imaging system. Timing of images within the deadlift exercise trial was synchronized with audio cues and a digital clock, and the entire deadlift and recovery sequence was recorded on ½-in. videocassette tape. The taped images were examined for the most representative beat (i.e., best delineation of endocardial borders and tomographic view most similar to baseline) occurring within the time interval selected for blood pressure measurement. The video image was transferred to a Dextra D-200 image analysis system, and the two-dimensional echocardiogram of the left ventricle was traced to determine end-diastolic volume (the first frame after onset of QRS complex) and end-systolic volume (the frame with minimum dimension). The volumes were calculated using a single-plane Simpson’s algorithm of 30 discs. Stroke volume was defined as the difference between left ventricular end-diastolic and end-systolic volumes, percent ejection fraction as (stroke volume/left ventricular end-diastolic volume)×100, and cardiac output as the product of stroke volume and heart rate.

The ratio of peak systolic pressure to end-systolic volume was calculated from the blood pressure and echocardiographic data for each time period.\textsuperscript{16–19}

Doppler Echocardiography

On a separate deadlift, Doppler echocardiography was used to measure central aortic flow velocity. A 1.9-MHz dedicated Pedof continuous-wave Doppler transducer was placed at the suprasternal notch and angled to measure maximal flow within the ascending aorta. Aortic velocity was recorded continuously on videocassette at a sweep speed of 100 mm/sec. Three consecutive beats were averaged at each measurement point during the deadlift and recovery sequence using the dedicated analysis package of the Hewlett-Packard system. Peak aortic flow velocity and mean aortic flow velocity acceleration were determined.

Data Analysis

Blood pressure, heart rate, and echocardiographic and Doppler aortic flow velocity measurements were analyzed at rest (baseline), at 15 seconds after the onset of the deadlift and immediately before the termination of deadlift. This occurred at 90 seconds for the 50% maximal lift and at 30 seconds for the 100% maximal lift. During recovery, data were analyzed at 5 seconds and at 30 seconds for both deadlift intensities. When measurement artifact was present within the blood pressure tracing, the most representative blood pressure values were selected and the corresponding echo and Doppler frames were analyzed within ±5 seconds of the blood pressure recordings.

Data are expressed as mean±1 SEM. Statistical analysis used repeated-measures analysis of variance for differences within and between lift intensities; \( \alpha \) was set at <0.05.

Results

The results are summarized in Table 1 and are illustrated in graphic form in Figures 1–4.

Fifty Percent Maximal Deadlift

Blood pressure and heart rate increased as expected during the deadlift. An example of recorded intraarterial blood pressure response is shown in Figure 1. During 50% maximal deadlift, mean blood pressure increased from a resting value of 107±3 mm Hg to 145±4 mm Hg (\( p<0.01 \)). Heart rate increased from a resting value of 88 beats per minute to 113±7 beats per minute (\( p<0.01 \)). With cessation of the deadlift, there was an abrupt fall in mean arterial pressure to 101±4 mm Hg within 2–5 seconds followed by a return to approximate resting values after 30 seconds of recovery. Heart rate declined only slightly after termination of the deadlift, decreasing to 105±6 beats per minute at 30 seconds (Figure 2).

Stroke volume initially decreased during the deadlift from a resting value of 60±5 ml to 51±7 ml after 15 seconds (\( p<0.01 \)) but subsequently returned to approximate resting values before the termination of the deadlift (Figure 2). Cardiac output was 5.4±0.4 l/min at rest, was unchanged after 15 seconds of deadlift, and subsequently increased to 7.6±1 l/min (\( p<0.01 \)) at the end of the deadlift. With release, stroke volume fell promptly to 52±5 ml and cardiac output decreased to 5.9±0.5 l/min. Both of these parameters remained unchanged at 30 seconds of recovery. Calculated peripheral resistance increased slightly (\( p<0.05 \)) during the initial 15 seconds of deadlift, then returned to control values at the end of deadlift. With cessation of deadlift, peripheral resistance fell rapidly (\( p<0.05 \)) and remained significantly reduced at 30 seconds (Figure 2).

Ejection fraction decreased from 58±2% to 46±3% (\( p<0.01 \)) during the initial 15 seconds of deadlift and then returned to approximate rest levels at the end of the deadlift (Figure 3). The initial decline in ejection fraction was associated with a significant increase in left ventricular end-systolic volume from a resting value of 43±5 ml to 58±6 ml at 15 seconds (\( p<0.01 \)). End-systolic volume remained constant after this initial rise. There was also a small but nonsignificant increase in left ventricular end-diastolic volume from 104±8 ml at rest to 118±7 ml at the end of the deadlift. Thus, ejection fraction was restored to resting values by the end of deadlift. At 5 seconds after cessation of the isometric deadlift, left ventricular ejection fraction rose rapidly to 67±3%. This value was maintained at 30 seconds of recovery. Both left ventricular end-systolic and end-diastolic volume declined rapidly immediately after release and then partially recovered by 30 seconds (Figure 3). The calculated ratio of peak systolic pressure/left ventricular end-systolic volume was unchanged at 15 seconds and 30 seconds of the deadlift but increased significantly at 5 seconds and 30 seconds of recovery (Figure 3).

Peak aortic flow velocity at rest was 94±5 cm/second and showed no significant change during deadlift (see Table 1). After termination of the deadlift, peak aortic flow velocity increased significantly to 133±8 cm/second and remained unchanged throughout the 30-second period of recovery. Resting mean aortic flow velocity acceleration was 1,599±229 cm·sec\(^{-2}\) and remained unchanged during 90 seconds of isometric deadlift (Table 1). With termination of the deadlift, mean aortic
flow velocity acceleration increased rapidly to 2,630±371 cm·sec⁻¹ and remained in this range for 30 seconds of recovery (p<0.01).

One Hundred Percent Maximal Deadlift
During 100% maximal deadlift, the pattern of response for all measured parameters was similar to that observed during the 50% maximal deadlift. The peak mean arterial pressure attained during 100% maximal deadlift was significantly greater than the 50% maximal deadlift at 15 seconds (146±5 mm Hg versus 130±4 mm Hg) and at 30 seconds (164±6 mm Hg versus 145±4 mm Hg), both p<0.001 versus 50% deadlift (Figure 2). The heart rate response was also significantly greater, with values of 119±5 beats per minute versus 103±4 beats per minute at 15 seconds and 134±6 beats per minute versus 113±7 beats per minute at 30 seconds (both p<0.01 versus 50% maximal deadlift) (Figure 2). After release of the deadlift, mean arterial pressure fell rapidly, reaching a significantly lower minimum value of 88±4 mm Hg at 2–5 seconds and gradually increased to 98±5 mm Hg at 30 seconds (both p<0.01 versus 50% maximal deadlift). Heart rate remained elevated after the 100% deadlift, declining to 122±6 beats per minute at 30 seconds of recovery. Calculated peripheral resistance was unchanged during 100% deadlift and also fell during the recovery period to values that were significantly less than the 50% maximal deadlift recovery period (p<0.05 for 5 and 30 seconds).

There was no initial change in stroke volume at 15 seconds followed by a significant rise to 74±9 ml at 30

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**TABLE 1. Hemodynamic, Echocardiographic, and Doppler Responses During 50% and 100% Isometric Deadlifting**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Rest 50%</th>
<th>Rest 100%</th>
<th>15 Seconds</th>
<th>Deadlift Maximal</th>
<th>5 Seconds</th>
<th>30 Seconds</th>
</tr>
</thead>
<tbody>
<tr>
<td>MAP (mm Hg)</td>
<td>107±3</td>
<td>108±4</td>
<td>130±4*</td>
<td>146±5*</td>
<td>145±4</td>
<td>164±6</td>
</tr>
<tr>
<td>Heart rate (min⁻¹)</td>
<td>88±5</td>
<td>88±5</td>
<td>103±4*</td>
<td>119±5*</td>
<td>113±7*</td>
<td>134±6*</td>
</tr>
<tr>
<td>Stroke volume (ml)</td>
<td>50±4</td>
<td>50±4</td>
<td>51±7*</td>
<td>54±6</td>
<td>64±6</td>
<td>74±9*</td>
</tr>
<tr>
<td>Cardiac output (l/min)</td>
<td>104±8</td>
<td>104±11</td>
<td>109±11</td>
<td>110±11</td>
<td>118±11</td>
<td>132±16*</td>
</tr>
<tr>
<td>LVESV (ml)</td>
<td>43±5</td>
<td>43±5</td>
<td>50±6*</td>
<td>56±7*</td>
<td>54±6</td>
<td>59±8*</td>
</tr>
<tr>
<td>SBP/LVESV</td>
<td>3.6±0.3</td>
<td>3.8±0.4</td>
<td>3.3±0.3</td>
<td>3.9±0.4</td>
<td>3.8±0.4</td>
<td>4.2±0.6</td>
</tr>
<tr>
<td>PAFV (cm·sec⁻¹)</td>
<td>94±5</td>
<td>95±5</td>
<td>90±5</td>
<td>81±3*</td>
<td>87±6</td>
<td>85±5</td>
</tr>
<tr>
<td>MAFVFA (cm·sec⁻¹)</td>
<td>1,599±229</td>
<td>1,629±223</td>
<td>1,507±174</td>
<td>1,241±73</td>
<td>1,608±313</td>
<td>1,537±174</td>
</tr>
</tbody>
</table>

MAP, mean arterial pressure; TPR, total peripheral resistance; EF, left ventricular ejection fraction; LVEDV, left ventricular end-diastolic volume; LVESV, left ventricular end-systolic volume; SBP, systolic blood pressure; PAFV, peak aortic flow velocity; MAFVFA, mean aortic flow velocity acceleration.

*Significant difference (p<.001) compared with rest. Differences between 50% vs. 100% intensities at each time period are indicated by NS (not significant) or by p<0.05.

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**FIGURE 1. Recordings show arterial blood pressure (mm Hg) and force platform tension (kg) during maximal deadlift and subsequent recovery (purposely recorded at a slower speed to illustrate the entire lift).**
FIGURE 2. Graphs show cardiovascular responses to isometric deadlift. Mean values for baseline, 15 seconds, maximum deadlift (30 seconds for 100% maximal lifting and 90 seconds for 50% maximal lifting), and 5 and 30 seconds of recovery are shown. MAP, mean arterial pressure; HR, heart rate; SV, stroke volume; TPR, total peripheral resistance. Error bars indicate 1 standard error. *Value is significantly different from rest (p<0.01).

seconds (Figure 2). Cardiac output increased progressively from 5.0±0.4 l/min at rest to 6.5±0.8 l/min at 15 seconds and to 9.5±1 l/min at 30 seconds (p<0.01). After cessation of the deadlift, stroke volume fell promptly in a pattern similar to the 50% maximal deadlift. Cardiac output also decreased rapidly but remained above resting levels after 30 seconds of recovery (Table 1).

Left ventricular ejection fraction, left ventricular end-systolic volume, end-diastolic volume, peak aortic flow velocity, mean aortic flow velocity acceleration, and the ratio of peak systolic pressure/left ventricular end-systolic volume all showed nearly identical values and response patterns compared with the 50% maximal deadlift (Figure 3 and Table 1). There was no statistical difference in any of these parameters during the 100% deadlift at 15 seconds and 30 seconds compared with the corresponding time of measurement at 15 seconds and 90 seconds in the 50% maximal deadlift protocol.

Esophageal Pressure Responses

Intrathoracic pressure was monitored in three additional subjects during 50% and 100% maximal deadlift (Figure 4). At rest, each subject exhibited the expected pattern of respiratory fluctuations with −2 to −5 mm Hg decline during inspiration. During deadlifting, there were no significant changes in esophageal pressure while rhyth-
mic breathing was maintained. The maximal values for arterial pressure and heart rate in these three subjects was similar to the mean for the first 10 subjects.

**Subjective Responses**

All subjects tolerated the deadlift protocols without difficulty. Three subjects reported transient dizziness during the first 5–10 seconds after the release of deadlift contraction. This corresponded to the time frame of rapid fall in mean arterial pressure. No arrhythmias were observed during these studies.

**Discussion**

**Left Ventricular Response to Deadlift**

This study demonstrates a characteristic triphasic pattern of change in left ventricular performance indexes during and after sustained submaximal and maximal isometric deadlift exercise. These phases are 1) an initial transient decline in left ventricular stroke volume and ejection fraction at the onset of deadlift, 2) restoration and maintenance of left ventricular stroke volume and ejection fraction during continued deadlifting, and 3) a hyperdynamic increase in left ventricular ejection fraction, peak systolic pressure/end-systolic volume ratio, peak aortic flow velocity, and aortic flow acceleration after the release of deadlift.

We postulated that our previous studies using first-pass radionuclide ventriculography detected an initial phase of decreased left ventricular performance during isometric deadlift but failed to identify the subsequent compensatory phase or hyperdynamic phase after cessation of deadlift. In the present study (using continuous echocardiographic measurements) we confirmed that left ventricular ejection fraction decreases approximately 10% shortly after the onset of isometric deadlift. This decline in ejection fraction is attributed to an increase in left ventricular end-systolic volume in response to the abrupt rise in systemic arterial pressure and corresponding left ventricular wall stress. The initial decrease in left ventricular ejection fraction is rapidly restored by an augmentation of left ventricular end-diastolic volume. Thus, at the end of both 50% and 100% maximal deadlifts, cardiac output has increased significantly due to increased heart rate in combination with a stroke volume that either increased slightly or remained unchanged.

**Submaximal Versus Maximal Deadlift**

The magnitude of blood pressure increase during the deadlift was significantly greater at the 100% maximal intensity compared with the 50% intensity. This difference is attributed to higher heart rate and cardiac output at 100% maximal intensity because calculated peripheral vascular resistance was unchanged. These findings are consistent with previous studies that have shown that peripheral vascular resistance does not change in normal subjects who perform graded increases in the intensity of isometric contraction using the same muscle mass.

The changes in left ventricular end-systolic volumes and ejection fraction were similar for the 50% and 100% maximal intensities despite significantly higher mean arterial blood pressure values attained during the 100% deadlift intensity. The ratio of peak systolic blood pressure/end-systolic volume did not increase compared with control values for either the 50% or 100% deadlift intensity. Recent studies suggest that although this ratio appears to be relatively independent of changes in preload, it may be significantly influenced by changes in end-systolic volume and end-systolic pressure, that is, nonproportional changes in afterload. Thus, in the circumstance of the deadlift, it is not possible to derive any definite conclusions about changes in contractility from the lack of change in this ratio. However, the mean left ventricular end-diastolic volume was significantly greater than baseline at the termination of 100% maximal deadlift and showed an upward, nonsignificant trend at termination of the 50% deadlift. This indicates a primary reliance on the Frank-Starling mechanism to maintain left ventricular ejection fraction and stroke volume during heavy isometric deadlifting.

**Left Ventricular Response to Release of Deadlift**

At the cessation of deadlift, blood pressure declined precipitously within 2–5 seconds. For the 100% maximal intensity, the change in mean arterial pressure was approximately 80 mm Hg. The rapid fall in blood pressure is attributed to the sudden release of muscle tension followed by hyperemic dilation of previously compressed vascular elements within the large mass of skeletal muscle used in maintaining the deadlift effort. Muscle sympathetic nerve traffic is known to decline rapidly after the termination of isometric contraction, but this probably contributed little additional effect to the locally mediated vasodilatation within skeletal muscle.

Left ventricular ejection fraction increased rapidly in response to the reduction in systemic arterial blood pressure. Both left ventricular end-diastolic and end-
systolic volumes decreased by similar magnitudes after both the 50% and 100% maximal deadlifts. Stroke volume was maintained at near control levels and cardiac output remained increased because of continued elevation of heart rate. The decrease in end-diastolic volume was probably due to impairment of venous return associated with acute dilation of vascular capacitance vessels within skeletal muscle after cessation of isometric contraction. Subjects remained standing and motionless after the deadlift; this posture undoubtedly contributed to decreased venous return.

End-systolic volume was substantially decreased after the release of deadlift. This change is attributed to the combined effects of afterload reduction and enhanced contractility. The calculated ratio of peak systolic pressure/end-systolic volume remained significantly increased throughout the 30 seconds of recovery after 50% and 100% maximal deadlift. The maintained increase in the ratio strongly suggests increased left ventricular inotropic status. Peak aortic flow velocity and mean aortic flow velocity acceleration also showed significant increases during the entire 30 seconds of recovery after deadlift. The increase in both of these parameters probably reflects enhanced contractility. However, these parameters have also been shown to be strongly affected by changes in systolic blood pressure. Thus, the major increases in aortic flow velocity and acceleration measured after the release of deadlift are probably due to the combined effect of increased contractility and rapid fall in systolic pressure.

Comparison With Previous Studies

To our knowledge, there are no previous studies that have continuously measured left ventricular performance and hemodynamics during sustained maximal upright isometric exercise using nearly total muscle contraction. Jones et al. showed a moderate decrease in left ventricular ejection fraction in normal subjects performing 2 minutes of isometric handgrip at 50% maximal intensity. These studies were performed with the subject in the supine position, using radionuclear probe imaging of left ventricular ejection fraction at 15-second intervals. The decrease in ejection fraction appeared to be continuous, with no evidence of the Frank-Starling compensation as seen in our studies. They also did not observe a hyperdynamic phase of left ventricular performance after cessation of isometric handgrip.

Robson et al. recently reported continuous measurements of left ventricular performance using M-mode echocardiography and Doppler aortic blood flow velocity during isometric handgrip at 30% maximal intensity for 3 minutes. Under these conditions, there was no significant change in left ventricular end-systolic volume, end-diastolic volume, or ejection fraction. The reported increase in mean arterial pressure was only 18 mm Hg.

It appears that submaximal isometric exercise performed in the supine position does not elicit significant changes in left ventricular performance. This is probably explained by the modest increases in arterial blood pressure produced by small-muscle–group isometric exercise and the favorable effect of the supine position on venous return.

Potential Limitations of Measurements

The reliability and accuracy of echocardiographic measurements of ventricular volumes, stroke volumes, and ejection fraction have shown good correlation with other methods. However, echocardiographic determinations tend to underestimate ventricular volume. Thus, the reported stroke volumes and cardiac outputs in this study may be less than if measured with other methods. The underestimation by echocardiography has been found to be linear over a wide range of values similar to those reported by this study. Because the primary purpose of this study was to compare beat-by-beat changes in volume, output, and ventricular performance, echocardiographic measurements should provide reliable estimates of relative change in these parameters. Absolute volume measurements may also have been affected by the fact that the study was performed in the upright, standing position rather than supine. However, prior studies have shown that valid, reproducible measurement of volume can be obtained by echocardiography during aerobic exercise on a treadmill. In this study, the transducer and patient remained stable throughout the entire deadlift and care was taken to select cardiac cycles that had identical orientations to baseline for analysis during the deadlift. Doppler flow velocity integral calculations were not used to determine stroke volume because it was not possible to monitor the diameter of the ascending aorta during the deadlift.

The Valsalva maneuver could potentially influence hemodynamic responses observed in this study. Previous reports have documented large increases in arterial pressure and impaired venous return when the Valsalva maneuver is maintained while weight lifting. However, we found no significant change in intrathoracic pressures in three subjects who maintained a shallow cyclic breathing pattern while performing the deadlift protocol.

Physiological and Clinical Implications

Mild to moderate concentric left ventricular hypertrophy is commonly reported in weight lifters, rowers, and other athletes engaged in power-based sports. Presumably, left ventricular hypertrophy develops due to repeated acute increases in left ventricular afterload and wall stress that occurs during the pressor response to isometric exercise. Isometric deadlift produces hemodynamic responses that probably occur in several similar positions routinely used in weight training. Our results suggest that the combination of increased left ventricular wall stress during the muscle contraction phase and the compensatory hyperdynamic state that occurs during the recovery phase after weight lifting may each contribute to the development of left ventricular hypertrophy. The relative influence of these two mechanisms on the development of left ventricular hypertrophy remains to be determined.

The rapid decrease in arterial blood pressure that occurs after cessation of isometric deadlift probably explains transient dizziness experienced by some weight lifters. Cardiac output and mean arterial blood pressure appear to be protected by sustained increases in heart rate and enhanced left ventricular performance during the recovery phase after muscular relaxation.
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References
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