Increased cardiovascular response to static contraction of larger muscle groups

DOUGLAS R. SEALs, RICHARD A. WASHBURN, PETER G. HANSON, PATRICIA L. PAINTER, AND FRANCIS J. NAGLE

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Seals, Douglas R., Richard A. Washburn, Peter G. Hanson, Patricia L. Painter, and Francis J. Nagle. Increased cardiovascular response to static contraction of larger muscle groups. J. Appl. Physiol.: Respirat. Environ. Exercise Physiol. 54(2): 434-437, 1983.—The purpose of this study was to investigate the influence of the size of the active muscle mass on the cardiovascular response to static contraction. Twelve male subjects performed one-arm handgrip (HG), two-leg extension (LE), and a “dead-lift” maneuver (DL) in a randomly assigned order for 3 min at 30% of maximal voluntary contraction. O$_2$ uptake (VO$_2$), heart rate (HR), and mean intra-arterial blood pressure (MABP) were measured at rest and, in addition to absolute tension exerted, throughout contraction. There was a direct relationship between the size of the active muscle mass and the magnitude of the increases in VO$_2$, HR, and MABP, even though all contractions were performed at the same relative intensity. Tension, VO$_2$, HR, and MABP increased progressively from HG to LE to DL. It was concluded that at the same percentage of maximal voluntary contraction, the magnitude of the cardiovascular response to isometric exercise is directly influenced by the size of the contracting muscle mass.

The increase in blood pressure and HR were more pronounced in HG than in LE (9). However, the magnitude of these changes is determined by the duration and intensity of contraction, with the latter expressed as percent of the maximal voluntary contraction (%MVC) (8). Investigations concerning the influence of the size of the actively contracting muscle mass or the absolute tension developed have produced conflicting results. Lind and McNichol (7) reported that the increase in HR and blood pressure during static contraction was independent of the size of the contracting muscle mass and the tension produced when %MVC was held constant. Other investigators (4, 11) have reported similar findings; however, two recent studies (2, 12) provide evidence for a greater cardiovascular response to static contraction when larger muscle groups are involved. It has been suggested (2) that the above discrepancies may in part be due to the rather small range of muscle masses activated during the static contractions used by these investigators. Ramos et al. (17) have shown that blood pressure increases in proportion to synergistic muscle recruitment during fatiguing static contraction. If the sizes of the actively contracting muscle masses were not markedly different, greater synergistic muscle recruitment in one type of contraction could reduce the initial mass differences. Consequently differences in the cardiovascular response to static contractions of apparently different-sized muscle masses would be minimized. The purpose of the present study was to examine the cardiovascular response to static contraction of widely varying amounts of active muscle mass.

METHODS

Twelve healthy adult male volunteers 20–30 yr of age served as subjects and provided informed consent before testing. On arriving for the experiment the subject’s resting arterial blood pressure was determined by auscultation in each arm to verify that there was no discrepancy in blood pressure between right and left arms. Maximal voluntary contraction in three different types of static contractions was then measured by using the highest value recorded during multiple trials. The three types of static contractions (Fig. 1) were as follows.

One-arm handgrip. One-arm handgrip (HG) was performed on a calibrated Stoelting hand dynamometer, adapted so that tension exerted during contraction was displayed on a Sanborn chart recorder. With the subject seated, the contracting (dominant) arm rested against the inside of the thigh in a downward position. Hand dynamometer size was individually adjusted.

Two-leg extension. Two-leg extension (LE) was performed against a padded cast iron bar on which a strain gauge had been mounted. Subjects were secured by a seat belt in a hydraulically adjustable chair with a stationary base. The angle of the knee joint was set at 70° prior to full extension to facilitate tension production in the maneuver. The strain gauge was calibrated with known weights at the same angle at which tension was generated (i.e., 70°) and was linear within the ranges studied. Tension was displayed on the Sanborn recorder. In HG and LE a constant reminder was given to relax parts of the body not involved in the contraction.

Dead lift. A “dead-lift” maneuver (DL) was performed by assuming a semisquat position, with feet parallel and back straight. The subject pulled up on a handle attached to a cast iron base by an adjustable metal chain. A strain gauge was mounted on the base and calibrated as in LE.
Tension was displayed and recorded as above. During DL subjects were asked to generate tension by both extending the legs and pulling with the arms and shoulders. In this way as much of the available muscle mass as possible was involved in contraction.

After determining the MVC tensions for each type of contraction, the subject was readied for intra-arterial catheterization. The antecubital surface was prepared with povidone iodine and alcohol, and 1% lidocaine was infiltrated over the brachial artery. A 20-gauge 1.25-in. catheter was inserted percutaneously into the brachial artery 2–3 cm distal to the antecubital fossa. The catheter was connected by extension tubing to a Statham P23D6 pressure transducer that had been calibrated with a mercury monometer and kept patent with heparinized saline.

After catheterization the subject was seated in the upright position. The pressure transducer was positioned at the level of the 4th intercostal space, and mean arterial blood pressure (MABP) was recorded via electrical damping on a Gilson polygraphy recorder. HR was determined from an electrocardiographic (ECG, CM-5) tracing by using 10 consecutive R-R intervals. Vo2 was measured with a Beckman metabolic measurement cart frequently calibrated with standard gases verified by Scholander analysis.

The subject then performed the three types of static contractions described above in a randomly assigned order at 30% MVC for 3 min. Tension, Vo2, HR, and MABP were recorded throughout exercise. Comparisons across the different conditions were made by an analysis of variance procedure for repeated measures. A Tukey post-hoc procedure was then applied where appropriate. The 0.05 level of confidence was used to determine significance.

**Results**

Mean absolute tension exerted during isometric contraction increased progressively from HG to LE to DL (all P < 0.01; Table 1). During DL mean (±SE) tension was three times greater than that developed during HG (42 ± 3 vs. 14 ± 1 kg). Mean values for Vo2 also increased progressively from rest to HG to LE to DL (all P < 0.01). Peak Vo2 attained during DL (841 ± 34 ml min⁻¹) was almost threefold greater than that elicited in response to HG (322 ± 10 ml min⁻¹).

HR increased progressively from rest throughout the 3 min of sustained contraction in all three types of exercise (Fig. 2). Mean values for ∆HR (the increase from

**Table 1. Values obtained at rest and after 3 min static contraction (30% MVC)**

<table>
<thead>
<tr>
<th></th>
<th>Tension,* kg</th>
<th>Vo2,† ml min⁻¹</th>
<th>∆Vo2,† ml min⁻¹</th>
<th>HR,‡ beats min⁻¹</th>
<th>∆HR,‡ beats min⁻¹</th>
<th>MABP,* mmHg</th>
<th>∆MABP,* mmHg</th>
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<tbody>
<tr>
<td>Rest</td>
<td>243±7</td>
<td>60±2</td>
<td>92±2</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>HG</td>
<td>14±1</td>
<td>322±10</td>
<td>79±10</td>
<td>80±4</td>
<td>20±3</td>
<td>173±3</td>
<td>25±3</td>
</tr>
<tr>
<td>LE</td>
<td>33±2</td>
<td>496±28</td>
<td>25±27</td>
<td>105±8</td>
<td>45±7</td>
<td>131±8</td>
<td>39±8</td>
</tr>
<tr>
<td>DL</td>
<td>42±3</td>
<td>841±34</td>
<td>598±39</td>
<td>116±6</td>
<td>56±5</td>
<td>143±4</td>
<td>51±3</td>
</tr>
</tbody>
</table>

Values are means ± SE. Vo2, O2 uptake; HR, heart rate; MABP, mean arterial blood pressure; HG, handgrip; LE, leg extension; DL, dead lift. † Rest < HG < LE < DL (all P < 0.01). ‡ Rest < HG (P < 0.01) < LE (P < 0.01) < DL (NS, P > 0.05).
Mean ΔHR was over twofold greater in response to LE (45 ± 7 beats·min⁻¹) compared with HG (20 ± 3 beats·min⁻¹) and almost three times greater in DL (56 ± 5 beats·min⁻¹) than in HG.

MABP also exhibited a progressive increase from rest throughout the 3 min of sustained contraction in all three types of exercise (Fig. 3). The magnitude of the increase in MABP from rest to end exercise (ΔMABP) became progressively greater from HG (25 ± 3 mmHg) to LE (39 ± 3 mmHg) to DL (51 ± 3 mmHg) (all P < 0.01), with ΔMABP during DL twice as great as that during HG.

DISCUSSION

The purpose of this study was to determine whether the blood pressure and HR responses to static contraction varied, depending on the active muscle mass involved. The three static contractions performed were chosen so that the size of the active muscle mass during each maneuver would be quite different. It has been suggested (2) that similarities in the size of the active muscle mass during various types of static contraction may have contributed to the conflicting findings from earlier studies. The markedly different mean absolute tensions and VO₂ values attained during the three types of static contractions in the present study suggest that the sizes of the active muscle masses during contraction were also markedly different.

In this study mean values for ΔHR and ΔMABP were progressively greater from HG to LE to DL. Therefore, the cardiovascular response to static contraction was greater as the size of the active muscle mass was increased. Previous investigators have reported an inverse (18), a direct (12), or no (7, 11) relationship between the size of the actively contracting muscle mass and the magnitude of the cardiovascular response to static contraction. Our results would support the conclusions of Mitchell et al. (12) who reported a direct relationship between the absolute tension generated (size of the active muscle mass) and the relative increases in HR and blood pressure during four types of static contractions.

The cardiovascular response to static exercise is regulated by both central (5) and peripheral (10) mechanisms. The central mechanism involves the irradiation of impulses from the motor cortex to the medullary cardiovascular center. It is directly related to the level of perceived exertion and is in part under voluntary control (13). This “central drive” may be the stimulus for the abrupt increase in HR and blood pressure during the onset of static contraction. However, recent evidence by Mitchell et al. (13) suggests that central drive is also involved in determining the magnitude of the cardiovascular response attained during contraction.

The peripheral mechanism consists of a reflex pathway originating in contracting muscle. Although this pathway has not been completely elucidated, it may involve the release of some metabolite (e.g., potassium) from active muscle and/or an increase in the osmolarity of the interstitial fluid (12). These changes could activate nerve endings, which in turn feedback centrally to the medullary cardiovascular center.

The relative contribution of these two mechanisms in determining the magnitude of the cardiovascular re-
Cardiovascular response to static contraction is not known. Furthermore, it is not clear how the two mechanisms regulate the cardiovascular response to static contraction of varying active muscle mass. Mitchell et al. (13) suggest that central and peripheral mechanisms combine to increase blood pressure and HR with greater mass (tension) involvement. The greater the contracting muscle size, the greater would be the number of centrally activated motor units (increased central drive). The large contracting muscle mass would also elicit greater peripheral input from skeletal muscle nerve endings.

Recent findings by Petrofsky and his associates (14-16) suggest that the fiber-type (metabolic) profile of the contracting muscle fibers may also influence the magnitude of the cardiovascular response. They demonstrated in cat skeletal muscle that the pressor response to static contraction of predomi-
nately fast-twitch muscle (gastrocnemius), whereas contraction of slow-twitch muscle (soleus) produced no increase in blood pressure. These findings may help explain the recent findings of Mitchell et al. (13) that the cardiovascular response to static contraction of varying muscle mass would also elicit greater peripheral input from skeletal muscle nerve endings.

In conclusion we examined the cardiovascular response to static contractions of markedly different active muscle mass. At the same %MVC we found a positive relationship between the magnitude of the increase in blood pressure and HR and the size of the active muscle mass.

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References