Left ventricular function during arm exercise: influence of leg cycling and lower body positive pressure

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Goodman JM, Freeman MR, Goodman LS. Left ventricular function during arm exercise: influence of leg cycling and lower body positive pressure. J Appl Physiol 102: 904–912, 2007.—The purpose of this study was to characterize left ventricular (LV) diastolic filling and systolic performance during graded arm exercise and to examine the effects of lower body positive pressure (LBPP) or concomitant leg exercise as means to enhance LV preload in aerobically trained individuals. Subjects were eight men with a mean age (± SE) of 26.8 ± 1.2 yr. Peak exercise testing was first performed for both legs [maximal oxygen uptake (V˙O2) = 4.21 ± 0.19 l/min] and arms (2.56 ± 0.16 l/min). On a separate occasion, LV filling and ejection parameters were acquired using non-imaging scintigraphy using in vivo red blood cell labeling with technetium 99m first during leg exercise performed in succession for 2 min at increasing grades to peak effort. Graded arm exercise (at 30, 60, 80, and 100% peak V˙O2) was performed during three randomly assigned conditions: control (no intervention), with concurrent leg cycling (at a constant 15% leg maximal V˙O2) or with 60 mmHg of LBPP using an Anti G suit. Peak leg exercise LV ejection fraction was higher than arm exercise (60.9 ± 1.7% vs. 55.9 ± 2.7%; P < 0.05) as was peak LV end-diastolic volume was reported as % of resting value (110.3 ± 4.4% vs. 97 ± 3.7%; P < 0.05) and peak filling rate (end-diastolic volume/s; 6.4 ± 0.28% vs. 5.2 ± 0.25%). Concomitant use of either low-intensity leg exercise or LBPP during arm exercise failed to significantly increase LV filling or ejection parameters. These observations suggest that perturbations in preload fail to overcome the inherent hemodynamic conditions present during arm exercise that attenuate LV performance.

Arm exercise; ventricle; end-diastolic volume; afterload; lower body positive pressure

The cardiovascular responses to dynamic leg exercise have been well characterized using a wide range of techniques; however, data describing left ventricular (LV) hemodynamic function during arm exercise, particularly perturbations that influence LV filling and ejection characteristics, have been limited by technical aspects of cardiac imaging and temporal resolution. Steady-state measures of stroke volume and cardiac output during arm exercise have been well characterized mostly by indirect Fick methods (1, 8, 12, 17, 23, 24, 34). At a given submaximal oxygen consumption (V˙O2) arm cranking results in a higher heart rate and blood pressure but a lower stroke volume (SV) at a similar relative intensity [% maximal V˙O2 (V˙O2 max)] (6, 28, 36, 42). Maximal arm cranking exercise elicits a lower peak V˙O2, cardiac output (Q), stroke volume (SV), and heart rate (HR) compared with leg exercise (6, 12, 28, 35, 36, 42). The lower SV and Q values have been attributed to blood pooling secondary to the orthostatic challenge of inactive lower limbs (35). In addition, the reflex sympathetic vasoconstriction in the inactive leg muscles and gut (3, 10, 39) elicits a greater total peripheral resistance and arterial blood pressure during arm exercise, presumably increasing afterload and limiting LV ejection, although this has not been adequately addressed in the literature. New information would help to elucidate the limits to LV function and the role of LV filling during arm exercise. In addition, such data may contribute to interventions for clinical conditions, including orthostatic hypotension.

A limited muscle pump from the inactive legs may contribute to diminished venous return and LV preload, thereby reducing SV (6, 28, 39, 42). Our group (26) and others (23, 24) have observed a modest “autotranslocation” of the stasis venous pool during arm exercise using lower body positive pressure (LBPP) in paraplegic subjects; however, the data describing these perturbations on LV loading conditions and the LV filling and ejection characteristics during arm exercise remain poorly understood (20, 27, 43). Traditional methods to examine LV are limited by temporal constraints or by respiratory maneuvers that may alter LV filling characteristics. Although echocardiography has previously been used to reliably describe LV volumes and indexes of systolic and diastolic function using LBPP (37), it remains problematic to obtain images during arm ergometry, and measures obtained immediately following exercise are often dubious validity due to rapidly changing LV loading conditions. Others have recently utilized invasive measures to establish the effect of LBPP during dynamic leg exercise (41); however, data describing LV hemodynamics during upper extremity dynamic exercise are lacking. The use of a nuclear technique that affords rapid temporal resolution (in seconds) in LV function may help in understanding the LV response to progressive arm exercise, and the effects of various perturbations in loading conditions. Accordingly, our objectives were first to characterize, with high temporal resolution, LV diastolic filling characteristics and systolic performance during graded arm exercise and second to examine the LV response to perturbations of cardiac filling by use of LBPP and concomitant leg cycling in healthy individuals. We hypothesized that maneuvers aimed at increasing venous return, including light leg exercise and LBPP, would improve LV filling and output (end-diastolic filling and SV) during arm exercise.

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METHODS

Subjects and Experimental Design

Eight well-trained male subjects ranging in age between 23 and 28 yr of age volunteered for the experiment. Six of these subjects were recreationally competitive triathletes. Subjects had a mean age (±SE) of 26.8 ± 1.2 yr, with a mean body mass of 73.8 ± 2.3 kg and a mean height of 1.79 ± 0.3 m. All subjects were informed of the experimental procedures and were required to sign an informed consent form before participating. The experimental protocol had been previously approved by the University and Hospital Human Ethics Committees and was in accordance with the Helsinki Declaration pertaining to use of human experimental subjects and informed consent.

The study was conducted in two phases. Phase I took place in an exercise laboratory for separate determinations of arm and leg maximal aerobic power and determination of work rates to be used in the phase II sessions. Phase II was conducted in a nuclear cardiology research laboratory, where radionuclide labeling and cardiac nuclear imaging were conducted.

Preliminary Arm and Leg Exercise Testing

Subjects underwent a maximal arm ergometry and leg cycle ergometry test for measurement of maximal VO₂ (VO₂max) and the establishment of a target heart rate and work rate subsequent testing sessions. Subjects arrived at the laboratory at least 3 h after consuming a meal and were asked to refrain from caffeine or alcohol ingestion and any exercise within 24 h of the tests. Subjects first completed the arm crank test using a calibrated Monarch Rehab Trainer (Varberg, Sweden) arm ergometer. After 2 min of loadless cranking at 60 rpm, the work rate was increased by 6 W every 2 min until either exhaustion or a plateau of VO₂ with increasing work rates was demonstrated in combination with a respiratory exchange ratio (RER) of >1.10. Subjects used these pedaling rates during subsequent exercise testing procedures. Minute ventilation and expired gases for determination of peak VO₂ (VO₂peak) were collected with an automated metabolic cart (Morgan, Chatham, Kent, UK). A two-point calibration with known gas concentrations and nitrogen was performed before each test.

After 120 min of rest, the subjects completed a maximal leg cycling test on a standard Monarch cycle ergometer. After 2 min of loadless pedaling at 60 rpm, the work rate was increased by 25 W every 2 min until similar end points described for graded arm exercise were obtained. Following a recovery period, subjects were habituated to simultaneous arm exercise and leg cycling, the latter set to 15% of the previously determined leg VO₂peak work rate. This allowed subjects to become accustomed to simultaneous upper and lower body activity to be used subsequently.

These tests established the work rates and corresponding HR to be used in subsequent exercise test protocols, which were set to elicit 30%, 60%, 80%, and 100% of maximum work rate (W) for both leg cycling and arm cranking.

Arm and Leg Exercise Nuclear Probe Studies

Within 7 days of baseline exercise testing, subjects reported to the Nuclear Cardiology Laboratory for the second phase of the experiment. This session was 3 h in duration. Subjects arrived in the morning, having consumed no food, alcohol, or caffeine in the previous 3 h and having refrained from arm exercise or heavy cycling within 24 h of the tests.

LV non-imaging scintography was performed using the VEST, which was as previously described by our group (19) and others (33, 46) and is well validated in the clinical and nuclear cardiology literature. The system consists of a NaI probe that is strapped to the subject’s chest using a special harness. The position of the probe over the LV is usually verified by concurrent gated LV nuclear techniques.

The VEST system uses beat-to-beat count data to produce non-imaged time-activity curves, and LV function data are available for up to 4 h after radiolabeling of red blood cells according to the natural half-life of the radiolabeling agent. These are then manipulated by custom software to assign LV filling and ejection measures either on a single cardiac cycle or averaged over seconds. After standard in vivo red blood cell labeling with 650 MBq technetium 99m, the VEST and ECG electrodes (CMs configuration) were then placed on the subject to allow for gating of the HR signal. HR was also monitored by a wireless heart monitor (Polar, Kempe, Finland). The VEST (Capintec, Ramsey, NJ) is a single-crystal NaI detector that measures the absolute count activity over the LV every 10 ms, generating discrete beat-to-beat time-activity curves. These curves are processed to derive relative end-diastolic (EDV) and end-systolic volumes (ESV), in addition to LV ejection fraction (EF) corrected for background activity. ECG and time-activity curves for the entire study are stored on magnetic tape, and offline analysis using the customized VEST software is performed to calculate 30-s trend plots of HR (beats/min), LV EF [LVEF; % as [(end-diastolic counts - end-systolic counts)/ end-diastolic counts], background-corrected relative EDV (EDVr) and ESV (ESVr; % resting counts/10 ms), peak filling rate (PFR; EDV/s), peak ejection rate (PjER; EDV/s), and relative Q (Qt; EDV/min). Our laboratory (47) has shown good correlations of LVEF between the VEST and multi-gated nuclear ventriculography (MUGA) during multiple stages of upright bicycle ergometer exercise in patients with a wide range of resting LV EFs (r = 0.94; SE = 0.04).

Before exercise studies, the VEST nuclear probe was positioned with the aid of a large field of view gamma camera over the LV in the seated position. The probe was stabilized with the manufacturer’s custom chest harness. The position of the probe over the LV was confirmed by gamma camera imaging before and after each arm crank condition to assess probe/harness system displacement during exercise, ensuring the probe’s field of view remained over the LV during arm cranking.

LBBP and control measures. Resting control measures were obtained before exercise testing in the seated position with and without LBBP by using a custom-fitted military CSU-13B/P five-bladder cut-away anti G-suit. The abdominal bladder was positioned to ensure that the splanchic area was correctly covered, while avoiding pressurization of the rib cage. The G-suit was pressurized to 60 mmHg, which was manually controlled using a needle valve. The G-suit pressure was displayed on a digital manometer (Valdyne, Northbridge, CA) with a resolution of 1 mmHg. The first 2-min acquisition consisted of the subject seated quietly wearing the VEST probe while wearing the uninflated G-suit. After a 5-min rest, data were acquired with the G-suit inflated. Systolic (SBP) and diastolic blood pressures (DBP) were measured twice by an investigator using a standard cuff/stethoscope, averaged for each condition. Mean arterial blood pressure (MAP) was subsequently calculated as 0.33(SBP − DBP) + DBP.

Leg and Arm Ergometry Protocols

Leg exercise. Following resting control measures, the subjects were positioned on an electronically braked cycle ergometer (Quinton, Seattle, WA), and data were acquired while subjects performed graded leg exercise at work rates equal to 30, 60, 80, and 100% of the previously determined maximal leg exercise work rate, each lasting 2 min in duration at a pedal cadence set to 60 rpm.

Arm exercise. Subjects rested for 120 min and were allowed to consume water before performing the arm cranking tests. The subjects were then seated on the cycle ergometer with a back rest to support the subject. A customized adjustable table holding the arm crank ergometer was then positioned in front of the subject, above the flywheel of the cycle ergometer. The hand cranks were adjusted so that the elbows were slightly flexed at full extension, and the crank axle was level with the gleno-humeral joint (12). In total, three randomly assigned arm crank test conditions were performed, beginning at 30% of...
maximal work rate for 2 min without any intervention and then progressed continuously through 60, 80, and 100% of the previously established maximal arm work rate for 2 min each at a cadence of 60 rpm, similar to the protocol of Toner et al. (42). Between each condition, subjects remained seated in a comfortable chair for at least 30 min, while wearing the VEST, continuing the next test sequence when the HR recovered to within 10 beats of its resting value. The three test conditions were as follows: arm-only condition (AO): subjects performed graded arm crank ergometry with legs remaining stationary; arm and leg condition (AL): the leg-cycle ergometer load was individually set to 15% of the subject’s previously determined maximum cycling work rate to ensure minimal resistance and smooth cycling without an appreciable metabolic cost, held constant throughout the entire arm exercise test; G-suit inflation condition (AG): graded arm exercise was performed with the anti-G-suit inflated to 60 mmHg at the onset of the 60% exercise level and for the duration of the test.

### Data Manipulation and Statistical Analysis

Nuclear count data were replayed from the ambulatory data logger directly into the IBM RT computer for storage and analysis. The VEST software produced trend plots of the ECG and nuclear data in 30-s averaged increments for the entire session (seated rest, seated G-suit inflation, leg cycle test, and three arm tests). Five-second data averaged from beat-to-beat LV time-activity curves were rendered for each 2-min exercise stage, and the final 30 s from each exercise stage was averaged. The 30-s averaged data was then transferred as ASCII files to a statistical software package for analysis (SuperAnova, Abacus, Berkely, CA).

Resting blood pressures, LVEF, LV filling rates, and relative LV volumes during quiet seated rest with and without G-suit inflation were compared using paired t-tests. LV data from maximal arm and leg ergometry tests were compared using paired t-tests. For comparison of LV function data across the AO, AL, and AG arm crank conditions at 60, 80, and 100% relative intensities were compared using a two-way repeated-measures ANOVA. For all analyses, differences were considered significant at the $P < 0.05$ level of probability.

### RESULTS

All eight subjects completed the study, with LV count data for all subjects successfully acquired during arm and leg exercise trials. A typical time-compressed plot of one subject’s entire session, showing changes in HR, LVEF, EDVr, and ESVr, is reproduced in Fig. 1.

#### LV Function During Arm and Leg Exercise

Changes in LV SV, LVEF, and relative volumes (EDVr and ESVr) during graded arm exercise are presented in Fig. 2. There were small gains observed for EDVr at 80% $\dot{V}O_2$ peak ($P < 0.05$) with parallel but proportionally smaller and insignificant changes in both ESVr and LV SV. Peak arm and leg
Cardiorespiratory end point data are compared in Table 1. Cardiorespiratory end points were significantly lower during peak arm vs. peak leg exercise (39% lower V\textsuperscript{\textcircled{\textregistered}}O\textsubscript{2} peak, 11% lower peak HR, 8% lower peak LVEF, and 12% lower peak LVEDV\textsubscript{r}; \textit{P} < 0.01). Mean Q\textsubscript{r} at each stage of exercise was significantly greater during leg vs. arm ergometry (\textit{P} < 0.01), as shown in Fig. 3. The mean PFR at each stage of exercise (Fig. 4) was significantly greater during leg vs. arm ergometry (\textit{P} < 0.01). In contrast, there were no increases in PFR beyond 30% of peak exercise levels during arm exercise.

**Control Seated Rest and G-suit Inflation**

During seated G-suit inflation (Table 2), MAP was significantly increased compared with the control seated condition (\textit{P} < 0.05). LVEF was significantly decreased during G-suit inflation with respect to control conditions (\textit{P} < 0.01), without a change in LVEDV\textsubscript{r}. Peak LV filling rate was significantly reduced during G-suit inflation vs. control sitting (\textit{P} < 0.01). HR was insignificantly lower during seated G-suit inflation.

**Arm Exercise and Manipulations**

The LV response to each exercise condition (AL, AO, AG) is presented in Fig. 5, A–C. HR was marginally but insignificantly higher during the AL condition compared with AO. Conversely, HR was lower in the AG condition only compared with the AL condition at intensities between 30 and 80% V\textsuperscript{\textcircled{\textregistered}}O\textsubscript{2} peak (\textit{P} < 0.05), but not different than the AO condition throughout exercise (Fig. 5A). The addition of the legs (AL) and G-suit inflation (AG) had an insignificant effect of raising EDV\textsubscript{r} with a diminishing effect throughout higher work rates (\textit{P} < 0.07) (Fig. 5B). There was a significant increase in LVEF from rest to the 30% exercise level for all three arm crank conditions (\textit{P} < 0.05); however, there were no differences in LVEF across the three arm cranking conditions at any exercise intensity (Fig. 5C).

**DISCUSSION**

This is the first report to describe serial changes in LV diastolic and systolic function during graded arm exercise and the influence of altered LV loading conditions on these variables under high temporal resolution. We demonstrated that arm exercise is associated with a lower LVEF and reduced LV diastolic filling rates despite correspondingly lower HRs compared with leg exercise. In addition, concomitant use of either low-intensity leg exercise or LBPP had minimal effects on LV filling and no effects on LV ejection characteristics during submaximal arm exercise.

**Arms vs. Leg Exercise: Comparison of LV Response**

During both arm and leg ergometry, the LVEF rose significantly from rest and was accompanied by an increase in LVEDV\textsubscript{r}. However, peak LVEF and LVEDV\textsubscript{r} were signifi-

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**Table 1. Resting and peak oxygen consumption and left ventricular data during arm and leg exercise**

<table>
<thead>
<tr>
<th></th>
<th>HR, beats/min</th>
<th>(V_{O2}\textsuperscript{\textcircled{\textregistered}}\text{peak}, \text{1/min})</th>
<th>LVEF, %</th>
<th>PER, EDV/s</th>
<th>LVEDV\textsubscript{r}, % cts/10 ms</th>
<th>Qr, EDV/min</th>
<th>PFR, EDV/s</th>
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<tbody>
<tr>
<td><strong>Arm exercise</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
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<tr>
<td>Rest</td>
<td>61 ± 5</td>
<td>49.0 ± 1.6</td>
<td>2.56 ± 0.19</td>
<td>91.0 ± 2.2</td>
<td>8.6 ± 1.8</td>
<td>28.2 ± 1.8</td>
<td>2.2 ± 0.20</td>
</tr>
<tr>
<td>Peak</td>
<td>152 ± 5</td>
<td>59.5 ± 2.7</td>
<td>5.095 ± 0.37</td>
<td>97.3 ± 3.7</td>
<td>115.2 ± 5.1</td>
<td>86.6 ± 4.2</td>
<td>5.2 ± 0.25</td>
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<tr>
<td><strong>Leg exercise</strong></td>
<td></td>
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<td></td>
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<td></td>
<td></td>
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</tr>
<tr>
<td>Rest</td>
<td>63 ± 6</td>
<td>48.2 ± 2.3</td>
<td>2.49 ± 0.25</td>
<td>98.5 ± 7.5</td>
<td>31.1 ± 3.8</td>
<td>31.1 ± 3.8</td>
<td>2.2 ± 0.22</td>
</tr>
<tr>
<td>Peak</td>
<td>169 ± 4*</td>
<td>60.9 ± 1.7</td>
<td>5.82 ± 0.61*</td>
<td>110.3 ± 4.4*</td>
<td>115.2 ± 5.1*</td>
<td>115.2 ± 5.1*</td>
<td>6.4 ± 0.28*</td>
</tr>
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Data are means ± SE. \(V_{O2}\textsuperscript{\textcircled{\textregistered}}\text{peak}, \text{peak oxygen consumption}; HR, heart rate; LVEF, left ventricular ejection fraction; LVEDV\textsubscript{r}, relative left ventricular end-diastolic volume (measured in % baseline counts per 10 ms); Qr, relative cardiac output; PFR, peak filling rate; PER, peak ejection rate. *\textit{P} < 0.01 leg vs. arm.
cantly greater during leg exercise (Table 1), as was the peak filling rate and cardiac output (Figs. 3 and 4). Notable was the constant LVEF during arm exercise of any intensity (Fig. 2). A comparison of cardiac responses at a similar absolute work rate for leg and arm exercise was not performed directly. However, our data suggest that \( \dot{V}O_2 \) peak during arm exercise was \( \sim 60\% \) of what was achieved with leg exercise. When comparing the relative cardiac outputs (Fig. 3) and peak filling rates (Fig. 4) during arm exercise at 100% of \( \dot{V}O_2 \) peak to that obtained at 60% \( \dot{V}O_2 \) peak for leg exercise, relative cardiac output and peak filling rate for arm exercise remains 10 and 6% lower, respectively.

The Frank-Starling mechanism is a key determinant of LV function during leg exercise (5, 18, 20, 27, 43, 48) and orthostatic challenges alone can limit ventricular filling pressures and reduce SV (4). Despite recruitment of neurohumoral mechanisms to augment hemodynamics during arm exercise (i.e., peripheral vasoconstriction and augmented arterial pressure), LV filling remained less than leg exercise (2, 7, 8, 28). The attenuated LVEF response during arm exercise may also be related to limited systolic function. We observed an increase in LVESVr during progressive arm cranking compared with leg exercise (25); however, in that study, subjects were older and were not rigorously screened for coronary disease, whereas the likelihood of disease in our subjects was extremely low. This hemodynamic response cannot be explained by changes in LV filling alone, since EDVr and ESVr both decline at this point. We could not measure arterial blood pressure during arm cranking in this study, but previous studies have shown higher SBPs during arm exercise at comparable HRs (7, 8, 12). It remains possible that postexercise reduction in afterload or increased LV contractility contributed to this response.

**Effects LBPP on LV Filling at Rest**

The anti-G suit used to create LBPP has been shown to successfully counteract dependent blood pooling during positive pressure breathing at +1 G\( _x \) (19), defending head-level arterial blood pressure (9). Although its use in the high +G\( _x \) environment is associated with an increase in total peripheral resistance and a minimal influence on venous return (38, 45), prior reports indicate that G-suit inflation can correct clinical orthostatic hypotension and reduce tachycardia (11), likely due to increased venous return. Notwithstanding, we observed that G-suit inflation at rest resulted in a 7% increase in MAP, no change in LVEDVr, and a reduction in HR and LVEF. Stickland et al. (41) reported that LBPP elicited increases in resting right atrial pressure and pulmonary artery wedge pressure, whereas other investigators have demonstrated that LBPP during leg exercise mediated an increase in MAP, attributed to pressure-sensitive mechanoreceptors (14). The fall in LVEF at rest in the present study could be attributed to a rise in increased MAP secondary to a modest rise in sympathetic outflow known to occur after inflation or activation of muscle mechano-reflexes independent of central cardiovascular control (44). Although Seaworth et al. (37) found that G-suit inflation at +1 G\( _x \) caused an increase in LV end-diastolic
diameter and SV (using echocardiography) when subjects moved from a supine to an upright position, they used pressures in excess of 100 mmHg, and the postural change may have rapidly unloaded the LV, augmenting the G-suit effect. In other studies where G-suits were inflated for long durations with subjects resting passively with head-up tilt to 70°, significant increases in LVEDV and SV were also reported, but without changes in LVEF and $Q\dot{}$ (15).

**LBPP and LV Function During Arm Exercise**

The normal response to acute arm exercise in able-bodied individuals includes a sympathetic-mediated vasoconstriction in inactive muscles and the gut, a reduced venous return, and attenuated cardiac output response compared with leg exercise. (3, 7, 8, 28). We proposed that augmentation of venous return during arm exercise by either light leg pedaling or LBPP would improve LV filling and output, and although small increases were seen at lower exercise intensities (particularly 30% $\dot{V}O_2\text{peak}$) the main effect failed to reach statistical significance ($P = 0.07$). The present findings are similar to an earlier report by our group (26) and by Hopman et al. (23), where in both cases no change was observed in SV or cardiac output. In another study by the same group (23), G-suit inflation failed to improve maximal arm $V_2$ or maximal exercise capacity. In contrast, Ng et al. (32) were able to show significant improvements in submaximal SV during inflation of medical anti-shock trousers in normal subjects and is the most comparable study to ours. The garment was inflated to 50 mmHg in the standing position during arm cranking and reported a decreased HR and a 12% increase in SV ($CO_2$ rebreathe), resulting in a significantly increased $Q$ at rest and at 70% of arm $V_2\text{max}$. In efforts to characterize pulmonary circulatory dynamics, an improved version of the anti-G-suit was used to develop LBPP during dynamic leg exercise in a recent study and reported significantly elevated cardiac filling pressures but not in CO or SV (41). Similarly, Gallagher et al. (14), using both cuff inflation and pneumatic-based LBPP, found no improvements in CO during progressive leg ergometry. The difference in findings may be due to body position, since standing may pose a greater orthostatic challenge, enabling a larger effect to be observed (37).

**Impact of Leg Exercise on LV Function During Arm Ergometry**

Some investigators have suggested that a reduced SV observed in normal individuals during arm exercise might be reversed by activating the muscle pump in the inactive legs (13, 16, 17, 42). Functional nervous stimulation (FNS) of the
legs can increase SV and Q˙ by 15–30% in able-bodied subjects at rest (28, 29), likely acting through this mechanism. In the present study, our subjects simultaneously exercised the legs on a cycle ergometer at low work rates to activate the muscle pump. Muscle pump activity appears to be determined by both the contraction frequency (21) and the interaction of the respiratory muscle pump (30). The work rate (15% of maximal leg work rate) was chosen to minimize the metabolic cardiovascular demand yet provide significant muscle pump activity, since low-level muscle pump activity is known to increase venous return, providing that adequate blood flow to the muscle group in question is maintained (40). This level of leg exercise was also conducted at an intensity that did not distract the subject from attaining the necessary cadence during arm ergometry, especially with increasing work rates. Although a small rise in HR during this condition occurred, it is likely that the increase in HR was mediated through activation of the ergo-reflex rather than the metabolic cost of the activity (31) or an adequate increase in lower limb blood flow. Nevertheless, unlike the data of Toner et al. (42), it appears that this maneuver contributed to only modest changes in LV filling and output during arm exercise.

Limitations

We could not account for all factors that might influence LV function, particularly activating the venous muscle pump during the AO condition. Subjects might have involuntarily stabilized their legs, although these maneuvers were monitored closely and were not observed. It was also impractical to vary the pressure of the G-suit, and it is possible that higher (supra-systolic) pressures might be required to increase LV preload during arm exercise. We minimized the LBPP pressure to limit the potential of increasing LV afterload and inducing a baroreflex-mediated lowering of HR. Furthermore, the level of LBPP was constant, despite increasing work rates during arm exercise. Greater precision would have been offered if we scaled the degree of LBPP and/or leg cycling to account for the greater metabolic rates during incremental testing, and although technically challenging, this should be explored in subsequent investigations. We could not measure blood pressure during arm ergometry, and postexercise measures would be problematic, especially during LBPP due to local pressure changes in the arms during arm cranking, and validity problems associated with intermittent and/or immediate postexercise measures were avoided (22). Although we have determined that VEST-measured LV function during arm cranking is both feasible and reproducible, we could not validate VEST results during arm ergometry against “gold-standard” techniques (first-pass radionuclide angiography, contrast angiography, etc.) because of the interference created by arm movement. Finally, the limited sample size and power (0.68) may have contributed to our failure to detect significant changes in EDV during the interventions (main effect P = 0.07) across low to moderate workrates with the interventions used (leg exercise and LBPP) during exercise.

In conclusion, LV function is reduced during dynamic arm exercise compared with leg exercise. Our data indicate that intrinsic loading conditions imposed by arm exercise restrict the diastolic filling rate and EDV. In addition, LV systolic performance is also attenuated, likely due to an elevated LV afterload unique to arm exercise. The use of LBPP or low-intensity leg cycling was not successful in significantly aug-

![Fig. 5. Means (±SE) data during exercise for heart rate (A), relative end-diastolic volume (EDV; B), and ejection fraction (C) for each arm cranking condition at the work rates representing 30%, 60%, 80%, and 100% of previously determined arm VO₂ peak. AO, arm crank; AG, arm cranking with anti-G suit inflated to 60 mmHg; AL, arm cranking with simultaneous leg cycling at 15% of the leg VO₂ peak. *P < 0.05, AG vs. AL only.](http://jap.physiology.org/)
menting LV hemodynamic function during dynamic arm exercise, and these strategies aimed at augmenting venous return had little effect on LV filling or on systolic function. These observations suggest that perturbations aimed at enhancing preload fail to overcome the hemodynamic disadvantage of reduced filling and increased LV afterload, both of which limit LV performance during arm exercise.

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