Exercise Evaluation of Upper- Versus Lower-Extremity Blood Pressure Gradients in Pediatric and Young-Adult Participants

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The aim of this study was to provide a normal reference for arm–leg blood pressure gradients in normal pediatric and young-adult patients before and after exercise. We assessed 216 normal participants by physical or echocardiographic exam, maximally tested using the James Cycle Protocol, with arm and leg blood pressures taken pre- and postexercise. Arm–leg gradients significantly increased from –5 mmHg at rest to 4, 2, and 1 mmHg 1, 3, and 4 min postexercise ($p < .05$). There was a small, statistically significant increase in arm–leg blood pressure with exercise, which is probably clinically insignificant. These data serve as a normal reference.

The arm–leg systolic blood pressure (SBP) gradient, measured at rest and postexercise, is a variable with clinical utility in assessing the severity of certain congenital heart lesions, most commonly coarctation of the aorta. Corrections for this anomaly include surgical correction by resection and end to end anastomosis, subclavian flap repair, balloon dilation or stent placement by interventional cardiac catheterization into the stenotic area. Arm–leg blood pressures have been used to assess the adequacy of the coarctation repair and to follow possible restenosis. When patients with surgically repaired coarctation undergo exercise testing, a significant arm–leg blood pressure gradient is found immediately postexercise when compared with rest (1,4,9,13). This is thought to reflect residual obstruction, either fixed or dynamic, and is used to follow the development of restenosis. Although exercise gradients are thought to be a significant finding in coarctation patients, it is not known whether similar gradients occur in normal patients. Thus, this study sought to establish normal standards for arm–leg gradients after exercise.

Methods

Two hundred sixteen participants (118 males and 98 females) ages 6–31 years were referred to our lab for clinical evaluation of the following symptoms or find-
ings: exercise intolerance, chest pain, shortness of breath, palpitations, ventricular preexcitation, or a prolonged resting ventricular repolarization (QT) interval. All participants were cleared of cardiac and vascular abnormalities by physical or echocardiographic exam. The participants were tested using a cycle ergometer (Lode Corival 400, Gronigen, The Netherlands) and the James Protocol (7). Heart rates and 12 lead electrocardiograms were recorded at rest; during each minute of exercise; immediately postexercise; and 1, 3, 5, 10, and 15 min postexercise (GE Case 8000 or GE Case 16, Milwaukee, WI). Blood pressures were measured in the right arm while supine at rest during upright exercise and while supine 1, 3, and 5 min postexercise using the auscultation method and a manual sphygmomanometer. Cuff sizes were chosen based on a bladder length and width of 80% and 40% of the arm circumference, respectively. The stethoscope was placed over the antecubital fossa of the arm (10,11). SBP was determined by the onset of the first Korotkoff sound (12). A second technician measured the blood pressure in the right thigh, concomitantly with the right arm blood pressure measurement, with the patient supine at rest and 1, 3, and 5 min postexercise using the same technique. Cuff-size selection was the same for the leg blood pressure determination, with the stethoscope placed over the popliteal fossa behind the knee (12). The two technicians taking the blood pressures were blinded to the measurements obtained by the other in 204/216 tests (94%). The two technicians were randomly assigned to measure leg pressure or arm pressure for each test. Oxygen consumption and carbon dioxide production were measured at rest and during each workload using a metabolic cart (Parvomedics Model TrueMax 2400, Sandy, UT). Perceived exertion was obtained during each exercise stage using the Borg scale (2). Participants were exercised until they were exhausted.

The exercise tests were judged to be maximal if two of the following criteria were met: (a) respiratory quotient (carbon dioxide production/oxygen consumption) was greater than 1.1, (b) maximal heart rate was greater than or equal to 85% of the age predicted maximal heart rate (6), or (c) maximal perceived exertion was greater than or equal to 18. No test was terminated prematurely.

Statistical Analyses

Variables were expressed as mean ± standard deviation (SD). These data were analyzed using a paired t test. A p value of < .05 was used for significance.

Results

Participants had a mean age of 15.2 ± 4.8 years, height of 1.61 ± 0.16 m, weight of 57.4 ± 16.7 kg, and a body surface area (BSA) of 1.59 ± 0.30 m². Results from the exercise tests were consistent with a maximal effort. The mean maximal VO₂ was 37 ± 7 ml · kg⁻¹ · min⁻¹, mean maximal RQ was 1.13 ± 0.07, and mean maximal heart rate was 192 ± 10 bpm. SBP measured in the right arm was 115 ± 11 mmHg at rest, 159 ± 19 mmHg 1 min postexercise, 138 ± 15 mmHg 3 min postexercise, and 122 ± 11 mmHg 5 min postexercise. Although there was a significant increase in SBP during exercise, it was in the normal range for exercise (14). SBP measured in the right thigh was 121 ± 11 mmHg at rest, 155 ± 17 mmHg 1 min postexercise, 135 ± 13 mmHg 3 min postexercise, and 122 ± 11 mmHg 5 min postexercise (Table 1).
The arm–leg blood pressure gradient significantly increased from a resting value of $-5 \pm 7$ mmHg to $4 \pm 8$ mmHg 1 min postexercise, $2 \pm 7$ mmHg 3 min postexercise, and $1 \pm 7$ mmHg 5 min postexercise ($p < .05$; Figure 1).

### Table 1  Exercise Data for the Total Group ($N = 216$)

<table>
<thead>
<tr>
<th></th>
<th>HR (bpm)</th>
<th>Arm SBP (mmHg)</th>
<th>Leg SBP (mmHg)</th>
<th>Gradient (mmHg)</th>
<th>Gradient range (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preexercise</td>
<td>73 ± 13</td>
<td>115 ± 11</td>
<td>121 ± 11</td>
<td>$-5 \pm 7$</td>
<td>$-34$ to 10</td>
</tr>
<tr>
<td>Max exercise</td>
<td>192 ± 10</td>
<td>176 ± 22</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 min postexercise</td>
<td>135 ± 15</td>
<td>159 ± 19</td>
<td>155 ± 17</td>
<td>$4 \pm 8$</td>
<td>$-22$ to 26</td>
</tr>
<tr>
<td>3 min postexercise</td>
<td>108 ± 13</td>
<td>138 ± 15</td>
<td>135 ± 13</td>
<td>$2 \pm 7$</td>
<td>$-30$ to 22</td>
</tr>
<tr>
<td>5 min postexercise</td>
<td>102 ± 11</td>
<td>122 ± 11</td>
<td>122 ± 11</td>
<td>$1 \pm 7$</td>
<td>$-26$ to 18</td>
</tr>
</tbody>
</table>

*Note. SBP = systolic blood pressure.*

**Figure 1** — Mean blood pressure gradients with standard deviations.

The arm–leg blood pressure gradient significantly increased from a resting value of $-5 \pm 7$ mmHg to $4 \pm 8$ mmHg 1 min postexercise, $2 \pm 7$ mmHg 3 min postexercise, and $1 \pm 7$ mmHg 5 min postexercise ($p < .05$; Figure 1).

## Discussion

Previous investigators have documented systemic hypertension and arm–leg gradients at rest in patients with coarctation of the aorta (5,8,13). Patients with coarctation of the aorta frequently have follow-up exercise testing. The exercise test closely monitors resting and exercise blood pressures and pre- and postexercise arm–leg gradients. The arm–leg gradient can be used as a noninvasive evaluation of residual stenosis (5). Sigurdardottir et al. (13) showed that SBP was significantly higher in patients with coarctation of the aorta than in the controls during exercise.
In addition, the arm–leg blood pressure gradient after exercise was significantly higher in coarctation patients with a significant increase over the preexercise values (13). They suggested two possible mechanisms for the gradient increase. One mechanism suggests a significant elevation of SBP in the arm and a diminished SBP in the leg when compared with controls. The diminished blood pressure in the leg is likely a result of obstruction of blood flow to the lower extremity through a residual stenosis in a nonpulsatile aortic segment. The second proposed mechanism postulates that patients with coarctation do not have normal vasodilation with exercise. Hypertension proximal to the obstruction can lead to faulty autoregulation, even though vasodilation is normal distal to the obstruction. They further postulated that exercise-induced hypertension correlates with a positive arm–leg blood pressure gradient, which, in turn, is associated with restenosis (13).

In a review of the literature on resting and exercise arm–leg gradients in patients with normal cardiac anatomy, three articles were identified that used normal controls for comparison to coarctation patients. In a study published by Markham et al. (9), 41 normal patients were used for comparison against 25 patients with coarctation of the aorta. These normal controls demonstrated no significant arm–leg gradient after exercise. The patients with coarctation of the aorta developed a significant gradient postexercise despite successful surgical intervention and the absence of a gradient at rest (9). Engvall et. al. stated, however, that a significant arm–leg gradient developed in normal participants after maximal but not submaximal exercise (3). Fripp et. al. also noted that previous investigators have found both positive and negative gradients in control patients (4).

It is generally accepted that SBP in the leg is higher than the arm, secondary to the standing wave effect (11). The participants in our study developed a statistically significant increase in arm–leg blood pressure gradient from preexercise to 1, 3, and 5 min postexercise. Considering the low absolute magnitude of the gradient and the lack of a hypertensive response in these participants, the gradients are probably of no clinical significance.

These data provide a normal reference for the evaluation of arm–leg blood pressure gradients before and after exercise in participants with normal cardiac anatomy and should prove helpful to future investigators evaluating blood pressure gradients in young participants during exercise.

References