Effects of posture on cardiovascular responses to lower body positive pressure at rest and during dynamic exercise

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Nishiyasu, Takeshi, Kei Nagashima, Ethan R. Nadel, and Gary W. Mack. Effects of posture on cardiovascular responses to lower body positive pressure at rest and during dynamic exercise. J. Appl. Physiol. 85(1): 160–167, 1998.—We tested the hypothesis that cardiovascular responses to lower body positive pressure (LBPP) would be dependent on the posture of the subject and also on the background condition (rest or exercise). We measured heart rate (HR), mean arterial blood pressure (MAP), and cardiac stroke volume in eight subjects at rest and during cycle ergometer exercise (76 ± 3 W) with and without LBPP (25, 50, and 75 mmHg) in the supine and upright positions. At rest, the increase in MAP was proportional to the increase in LBPP and was greater in the supine (6 ± 2, 15 ± 3, and 26 ± 3 mmHg) than in the upright (2 ± 3, 9 ± 3, and 17 ± 3 mmHg) position. During dynamic exercise, the increases in MAP evoked by 25, 50, and 75 mmHg LBPP were greater in the supine (15 ± 2, 28 ± 3, and 40 ± 3 mmHg) than in the upright (7 ± 3, 12 ± 3, and 25 ± 3 mmHg) position. We conclude that the systemic pressure response to LBPP is clearly dependent on the body position, with the larger pressure responses being associated with the supine position both at rest and during dynamic leg exercise.

lower body positive pressure; upright; exercise; mean arterial blood pressure; muscle mechanoreflex; muscle metaboreflex

THE APPLICATION OF POSITIVE pressure to the lower half of the body increases tissue pressure and reduces the transmural pressure gradient across the vasculature, leading to increases in both central venous pressure and mean arterial blood pressure (MAP) (2, 4, 14, 23). At rest, the increase in MAP during the application of lower body positive pressure (LBPP) is the result of increased cardiac output (CO) and vascular resistance, induced either by the direct mechanical effects of increased tissue pressure on circulatory hemodynamics (4, 21) or by activation of the muscle mechanoreflex (13, 24, 26). During exercise, in addition to the mechanisms mentioned above, application of LBPP reduces muscle blood flow and induces an accumulation of metabolic by-products; these are thought to activate muscle metaboreceptors and so induce a reflex-activated rise in MAP (1, 15, 16, 21). The contribution made by each of these factors (mechanical, muscle mechanoreflex, muscle metaboreflex) to the pressor response associated with the application of LBPP at rest and during exercise is unknown. Furthermore, it is not known whether the cardiovascular responses to LBPP are affected by the posture (supine vs. upright) of the subject.

A pooling of blood occurs in the lower body in the upright posture compared with the supine posture (7), and this is associated with an increase in hydrostatic pressure. It would thus be expected that the cardiovascular responses to LBPP would differ depending on body posture. Limited data are available on the cardiovascular responses to increased tissue pressure in subjects wearing antigravity suits in the seated and upright positions (6, 8, 11, 12, 22). The purpose of this study was to test the hypothesis that the pressor response evoked by application of LBPP is dependent on body posture and on the background condition (i.e., rest or dynamic exercise). A greater knowledge of the physiological response to LBPP in the two positions, both at rest and during dynamic exercise, should be a useful addition to our understanding of any situation in which there is an increase in the external pressure acting on the lower body. These situations would include accidental or experimental immersion in water, the use of antigravity suits in patients with profound autonomic failure, and the use of LBPP as an experimental model for the ischemic conditions experienced by patients with arterial obliterative disease (25). For this study, we used a newly developed LBPP device that contains an integral bicycle ergometer and that can be tilted from the horizontal to the vertical position. Furthermore, to enable a fuller investigation of the effects of LBPP in a given posture, we used a range of LBPP levels (25–75 mmHg).

METHODS

We studied eight healthy volunteers (7 men and 1 woman) with a mean age of 28 ± 2 yr, body weight of 66.5 ± 2.7 kg, and height of 174.0 ± 2.0 cm. The subjects were nonsmokers, and none was taking any medication. The female subject’s experiment was conducted during the first 10 days of her menstrual cycle. All subjects came to the laboratory for orientation before the experiment and were familiarized with all measurement devices. Each subject gave informed written consent before participating in the study. The experimental protocol was approved by the Yale University School of Medicine Human Investigation Committee.

Procedures. Subjects were instructed to drink 1 liter of water the night before the experiment and to refrain from beverages containing caffeine or alcohol. They were allowed to drink (no coffee or tea) on the morning of the experiment and to eat a light breakfast. On arriving at the laboratory, the subjects drank 15 ml/kg tap water and sat quietly for 1.5 h at 26°C before beginning the experiment.

Each subject then entered the environmental chamber, which was maintained at 27°C, and assumed the supine position with the lower torso, up to the iliac crest, enclosed in the LBPP box. A seal was established at the waist at the level of the iliac crest by means of flexible neoprene shorts. The LBPP box was designed to allow both supine and upright exercise on a Collins electronically braked cycle ergometer placed within the box. The cycle ergometer was located on an adjustable slide to accommodate variations in leg length, and the box was constructed in such a way that during each cycle of the pedals the fully extended leg was never more than 20°
above or below the horizontal plane of the hip (Fig. 1). A saddle and a hip belt were provided to limit movement of the body during the application of LBPP and tilt.

After an initial 30 min of supine rest, LBPP was applied in a graded manner (25, 50, and 75 mmHg) to the subject while in the supine position, with the subject's feet secured to the pedals of the ergometer. Each level of LBPP was maintained for 3 min, with no interval between the different levels. At the end of 3 min at 75 mmHg LBPP, the pressure was returned to zero. After 15 min of recovery, the LBPP box was tilted to 90° so that the subject was then in the upright position. Five minutes later, graded LBPP was applied again. The LBPP box was then tilted back to the supine position, and the subject was allowed 30 min of recovery. At the end of this recovery period, a mouthpiece for gas measurements and a noseclip were fitted and 5 min of resting data were recorded. The subject then performed 13 min of continuous cycle ergometer exercise at an intensity of 76 ± 3 W. Each subject exercised in both the supine and upright positions, the order being determined by a balanced crossover design. The exercise intensity was selected on the basis of the power required to elicit a heart rate (HR) of 100 beats/min, as determined during the performance of a graded submaximal supine exercise protocol on a different day. Graded LBPP (25, 50, and 75 mmHg; each for 3 min) was applied beginning 4 min after the onset of exercise, with the exercise and the period of LBPP ending at the same time. Between the two exercise periods (in different postures), the subject was allowed 30 min of recovery in the supine position. Respiratory parameters, thoracic impedance (Z0), and cardiac stroke volume (SV) were measured every 30 s. HR and arterial blood pressure were measured once per minute throughout the experimental protocol.

SV was measured by using a Minnesota impedance plethysmograph (12). A tetrapolar electrode system was used with two pairs of Mylar-tape ring electrodes employed, one around the neck and one around the trunk. A phonocardiograph microphone and a set of electrocardiograph electrodes were placed on the chest. Signals were processed by computer with software provided by Bio-Technical Instruments. Ensemble averaging was used to filter out respiratory and skeletal muscle artifacts, with the averaging time set at 25 s. CO was calculated from SV and HR. Systolic and diastolic arterial blood pressures were measured noninvasively from the upper left arm with a Colin blood pressure monitor (model STBP-780B, Colin, Aichi, Japan). Mean arterial pressure (MAP) was calculated from MAP = DAP + (SAP – DAP)/3, where DAP and SAP are systolic and diastolic arterial blood pressure, respectively. During exercise, oxygen consumption, minute ventilation (Ve), and respiratory exchange ratio (RER) were measured by using a Senser Medics metabolic cart.

Statistical analysis. Data are presented as means ± SE for eight subjects. Statistical analysis was performed by a repeated-measures analysis of variance followed by a protected least significant difference post hoc test. A P value of < 0.05 was considered statistically significant.

RESULTS

The influence of body posture on cardiovascular function in subjects at rest is shown in Table 1. CO, SV, and total vascular conductance (TVC) were all higher in the supine posture than in the upright posture. HR and Z0 were lower in the supine than in the upright posture, whereas MAP values were similar regardless of posture.

Table 1. Physiological variables in subjects in the supine and upright postures at rest

<table>
<thead>
<tr>
<th>Variable</th>
<th>Supine</th>
<th>Upright</th>
</tr>
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<tbody>
<tr>
<td>Heart rate, beats/min</td>
<td>59.7 ± 2.1</td>
<td>74.0 ± 3.1*</td>
</tr>
<tr>
<td>Mean arterial blood pressure, mmHg</td>
<td>86 ± 2</td>
<td>89 ± 3</td>
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<tr>
<td>Cardiac output, l/min</td>
<td>7.6 ± 0.7</td>
<td>6.7 ± 0.6*</td>
</tr>
<tr>
<td>Stroke volume, ml</td>
<td>133.7 ± 16.6</td>
<td>90.9 ± 9.0*</td>
</tr>
<tr>
<td>Total vascular conductance, units</td>
<td>89.3 ± 9.1</td>
<td>75.9 ± 7.6*</td>
</tr>
<tr>
<td>Thoracic impedance, Ω</td>
<td>23.9 ± 0.9</td>
<td>24.9 ± 1.0*</td>
</tr>
</tbody>
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Values are means ± SE; n = 8 subjects. *P < 0.05.
Effects of LBPP at rest. In subjects at rest, LBPP (25, 50, and 75 mmHg) produced a graded increase in MAP, the increase being greater in the supine (6 ± 2, 15 ± 3, and 26 ± 3 mmHg) than in the upright (2 ± 3, 9 ± 3, and 17 ± 3 mmHg) posture (Fig. 2A). HR was slightly increased at 50 and 75 mmHg LBPP in the supine position, but it decreased during LBPP in the upright posture (Fig. 2B). During LBPP, SV was unchanged from the initial value in the supine posture, but it was increased in the upright posture (Fig. 2C). CO was increased during LBPP in both postures (Fig. 3A), but at 50 and 75 mmHg there was no difference in CO between the two postures. In the supine posture, TVC showed a decrease at 75 mmHg LBPP, whereas it increased at 25 and 50 mmHg LBPP in the upright posture (Fig. 3B). During LBPP, $Z_0$ was unchanged in

Fig. 2. Effect of posture on changes in mean arterial pressure (MAP; A), heart rate (HR; B), and stroke volume (SV; C) induced by 3 levels of LBPP at rest. Values recorded are shown separately for supine and upright postures. *Significantly different from resting condition, $P < 0.05$. †Significant difference between supine and upright postures, $P < 0.05$.

Effects of LBPP during exercise. During exercise, the graded increase in MAP that occurred with LBPP of 25, 50, and 75 mmHg was greater in the supine posture (13 ± 2, 28 ± 3, and 40 ± 3 mmHg, respectively) than in the upright posture (7 ± 3, 13 ± 3, and 25 ± 3 mmHg, respectively) (Fig. 4A). The increase in MAP at a given level of LBPP was larger during exercise than at rest.
with subjects in either posture. HR was increased during exercise with LBPP in the supine posture, but it was unchanged in the upright posture (Fig. 4B). In the supine posture, SV was unchanged during exercise at LBPP of 25 and 50 mmHg and slightly decreased at 75 mmHg LBPP. In contrast, it was increased at all levels of LBPP when exercise was performed in the upright posture (Fig. 4C). CO was increased during LBPP regardless of posture (Fig. 4A). In the supine posture, TVC was decreased during exercise at LBPP of 50 and 75 mmHg. However, in the upright posture it was slightly increased at 50 mmHg but decreased at 75 mmHg (Fig. 5B). During exercise, oxygen consumption was unaffected by posture or LBPP level (Fig. 6B). However, during supine exercise, $\dot{V} E$ and RER were elevated at LBPP of 50 and 75 mmHg (Fig. 6, A and B).

**DISCUSSION**

On application of LBPP, several cardiovascular responses can be expected to occur, and the nature and magnitude of these responses will be dependent on the pressure level used and on the background conditions (i.e., rest or dynamic exercise). MAP has been reported to be increased by 3–6 mmHg at 20–30 mmHg LBPP and by 4–15 mmHg at 40–50 mmHg LBPP (2, 4, 5, 23). In the present study, the increases in MAP recorded at 25 and 50 mmHg LBPP in the supine posture at rest were very similar to those previously reported (at the corresponding level of LBPP). In our study in the supine posture, MAP was increased by 26 mmHg at 75 mmHg LBPP, a pressure for which no previous data are available from studies using this technique. In most of the previous studies in which a LBPP technique was used with the aid of an LBPP box, the experiments were conducted in subjects in the supine posture (2, 4, 14, 21, 23). To the best of our knowledge, this study is the first to use an LBPP box with a subject in the upright posture. Our results clearly show that the increase in MAP during the application of LBPP is greater in the supine than in the upright posture both at rest and during dynamic exercise.

Cardiovascular responses at rest. At rest, LBPP (25, 50, and 75 mmHg) produced a graded increase in MAP, the increase being greater in the supine (6±2, 15±3, and 26±3 mmHg, respectively) than in the upright (2±3, 9±3, and 17±3 mmHg, respectively) posture. Because an increase in CO occurred at all levels of LBPP in each posture and because the CO changes were no different between the two postures, the augmented MAP responses in the supine posture would seem to be due to the different TVC responses. Several possible mechanisms might explain the different responses associated with the application of LBPP in the supine or upright posture at rest; these are discussed below.

Several studies (10, 17, 24, 27) have suggested that a rise in intramuscular pressure can itself reflexly increase arterial pressure. Williamson et al. (27) demonstrated that an LBPP of 90 mmHg caused by antigravity suit inflation caused an increase in MAP of 9 mmHg when cephalad translocation of fluid was prevented by upper thigh cuffs. Because the increase in MAP was diminished by the administration of epidural anesthesia, the pressure response to external compression of the legs was presumed to be due to a reflex mechanism. In the present study, we applied LBPP at levels up to 75 mmHg; thus this muscle mechanoreflex could have contributed to the observed increase in MAP during LBPP applied to subjects while in both postures. HR was slightly increased in the supine posture during LBPP, despite a marked increase in MAP, suggesting that the muscle mechanoreflex enhanced sympathetic activity and overrode the baroreflex bradycardia (23). However, the muscle mechanoreflex should have been activated to the same extent whether the subject was in the supine or the upright posture. Thus the differences noted between the two postures in the MAP response to
LBPP would have to be due to differences in mechanisms other than the mechanoreflex.

During LBPP at rest, SV was not changed while subjects were in the supine posture, although it was markedly increased while they were in the upright posture. Although Bevegard et al. (2) showed no increase in SV at 40 mmHg LBPP, Eiken et al. (4) and Shi et al. (23) reported that SV was increased by 8.9–20 ml at LBPP of 40–50 mmHg at rest. Because, in the present study, the resting value for SV was recorded while the subject’s feet were on the pedals of the cycle ergometer, the SV would have already been higher than that recorded with the subject in the usual supine position. This may have been responsible for the lack of a further increase in SV during LBPP in the supine position. In the upright posture, the higher hydrostatic pressure in the lower body causes a pooling of ~600 ml of blood in that part of the body (7). This leads to several compensating reactions (e.g., an increase in HR and a decrease in TVC), which tend to restore the blood pressure and the blood volume in the central part of the body, mainly via high- and low-pressure baroreflexes. The increase in SV seen in the upright posture during LBPP suggests that LBPP caused a marked translocation of blood from the lower to the upper part of the body. This notion is supported by the decrease in Z₀, suggesting an increase in central blood volume, which was seen during LBPP in the upright posture. This restoration of the central blood volume could load the high- and low-pressure baroreflexes. The decrease in HR in the upright posture during LBPP could be due to a loading of the high blood pressure baroreceptors. The loading effects of these baroreceptors might explain, in part, the reduced MAP response in the upright posture. However, because MAP was not changed by the initial posture change itself, in which a large blood translocation occurs, the effects of such a blood translocation itself on MAP response is presumably small. Thus this could not have been the main factor responsible for the augmented MAP response seen here in the supine posture.

Reneman et al. (19) demonstrated that changes in pressure around a limb were well transmitted from skin to muscle. It is thus assumed that each 25-mmHg
change in LBPP caused a change of ~25 mmHg in lower body transmural pressure. If the total blood flow in the lower body (lower limbs, lower abdomen, and buttocks), when enclosed in a LBPP box, is assumed to be given by tissue weight (25 kg) (muscle + other tissues) \times \text{the average flow per weight (5 ml·100 g}^{-1}\text{·min}^{-1}) (20), we get a value of 1.25 l/min. The vascular conductance of the lower body in the supine posture at rest would be 14.7 units, assuming a perfusion pressure in the lower body of 85 mmHg [MAP - CVP (0 mmHg)] at 0 mmHg LBPP, where CVP is central venous pressure. However, perfusion pressure would be only 35 mmHg at 50 mmHg LBPP in the supine posture at rest. Thus, provided the flow obeys Bernouilli’s law, the conductance in the lower body at 50 mmHg LBPP would be only ~40% of the value at 0 mmHg LBPP, and MAP would be increased by ~8 mmHg. On the other hand, when the level of LBPP exceeds the transmural pressure, the transmural pressure in the veins in the lower body would become somewhat negative. Although flow in collapsible tubes (e.g., veins) obeys the basic laws of liquid dynamics, transmural pressures near or below zero markedly reduce the cross-sectional area of the tube, and this markedly increases the viscous resistance to flow (9). Because the transmural pressure in the veins in the lower body will be markedly lower with the body in the supine posture than in the upright posture, the critical value of LBPP needed for the veins’ transmural pressure to become negative would be less in the supine posture. Thus a given value of LBPP would cause a greater increase in resistance to flow in the lower body when applied in the supine posture (and thus evoke a greater increase in MAP). We postulate that this mechanical effect on vascular resistance is mainly responsible for the marked decrease in TVC in the supine posture, and consequently for the greater increase in MAP seen in the supine posture during LBPP.

Cardiovascular responses during dynamic exercise. During dynamic exercise, the increase in MAP was markedly less in the upright posture than in the supine posture (by 6, 15, and 14 mmHg at 25, 50, and 75 mmHg LBPP, respectively). Furthermore, the increases in MAP during LBPP were slightly greater during exercise than at rest in the upright position and markedly greater in the supine posture. During exercise in the supine posture, TVC showed a graded decrease with increases in LBPP, whereas in the upright posture it was increased at 50 mmHg LBPP and decreased at 75 mmHg LBPP. The increase in CO seen during exercise with LBPP was either no different or greater in the upright than in the supine posture; thus the augmented MAP response seen in the supine posture must have been due to the different TVC responses (as also concluded for the experiments at rest).

Most of the factors (mechanical effects, muscle mechanoreflex, baroreflexes) influencing MAP during LBPP at rest would be expected to operate in a similar manner during exercise. Thus, during dynamic exercise, the augmentation of the MAP response to LBPP seen in the supine posture would be partly explained by the above factors. However, the fact that the difference between the two postures in terms of MAP responses to 50 and 75 mmHg LBPP was greater during exercise than at rest would be due to the other mechanisms associated with dynamic exercise.

Sundberg and Kaijser (25) found that, in the supine posture, LBPP reduced blood flow in the working muscle during leg exercise. Furthermore, it has been shown that during dynamic exercise, the changes in blood flow in the lower extremities produced by application of LBPP (up to 60 mmHg) reduced the O2 saturation in the femoral venous blood and increased lactate release (20, 25). Thus it is postulated that the application of 50 and 75 mmHg LBPP to subjects in the supine posture could reduce blood flow in the leg muscle,
with a consequent release of lactate in the muscle. The marked increase in Ve and RER at 50 and 75 mmHg LBPP during exercise in the supine posture would support this notion if we assume, as we think is likely, that the muscle metaboreflex was activated in the supine position during dynamic exercise (4, 21). The decrease in TVC seen at 50 and 75 mmHg LBPP in the supine posture could be partly due to the vasoconstriction induced as part of the muscle metaboreflex. On the other hand, Ve and RER did not change when LBPP was applied to subjects during exercise in the upright posture. Probably, the critical reduction in blood flow needed to initiate the muscle metaboreflex did not occur during exercise when it was performed in the upright posture. This notion is consistent with our conclusion that the decrease in TVC was due to the mechanical effects of LBPP was less marked in the supine posture than in the supine posture. Thus we conclude that in dynamic exercise in the supine position the cardiovascular response is augmented mainly by the addition of the muscle metaboreflex. This is shown particularly by the greater increase in MAP at 50 and 75 mmHg LBPP in the supine posture.

Limitations. We used the noninvasive impedance method to evaluate cardiac SV. It is known that the impedance cardiography method allows satisfactory estimates of relative values for SV at rest and during exercise at up to moderate levels but not always of the absolute values (3, 12). We calculated CO and TVC by using this method and used these values (absolute values) to estimate the mechanical effects of LBPP on the cardiovascular system as discussed above. We also used the data for limb blood flow and venous pressure obtained from previous studies. These factors have to be acknowledged as limitations of the methods discussed above.

We have discussed the possible mechanisms involved in mediating the increased blood pressure response to LBPP associated with the supine posture by discussing the actions of individual reflexes. However, it is to be expected that there will be some degree of interaction between the various reflexes. For example, it has been reported that there is interaction between the effects of the metaboreflex and the mechanoreflex on muscle sympathetic nervous activity (18). Unfortunately, as yet the physiological nature of the interactions between such reflexes is not well understood, and further investigations are needed.

In conclusion, the arterial pressure response to LBPP is dependent on posture, with the larger pressure response being associated with the supine position both at rest and during dynamic leg exercise. The blood pressure response to LBPP is greater during dynamic leg exercise than at rest, especially in the supine posture. It is suggested that this augmented MAP response to LBPP in the supine posture is mainly due to 1) the greater mechanical effects of LBPP on vascular resistance at rest and 2) both these larger mechanical effects and the presence of a greater muscle metaboreflex during dynamic leg exercise.

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