Effects of Age and Fitness on Tolerance to Lower Body Negative Pressure

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Background. The purpose of this investigation was to determine the effects of age and fitness on tolerance to maximal lower body negative pressure (LBNP).

Methods. Ten older fit (OF) [73.9 ± 2 years; 39.0 ± 2 ml·kg⁻¹·min⁻¹ (age and estimated VO₂peak, respectively)], 10 older unfit (OU) (70.9 ± 1 years; 27.1 ± 2 ml·kg⁻¹·min⁻¹), 10 young fit (YF) (22.6 ± 0.5 years; 57.1 ± 2 ml·kg⁻¹·min⁻¹), and 10 young unfit (YU) (23.1 ± 1 years; 41.1 ± 2 ml·kg⁻¹·min⁻¹) participants underwent graded LBNP of −10 mmHg every 4 minutes to either presyncope or −100 mmHg.

Results. Compared to the other groups, YF had an earlier increase in heart rate (−40 mmHg vs the last stage; YF vs OF, OU, and YU, respectively) and decline in stroke volume (−20 mmHg vs −40 mmHg; YF vs OF, OU, and YU, respectively). OU had a higher resting mean arterial pressure; this difference was maintained until the last stage. OF had an earlier decline in total peripheral conductance than the other groups (−20 mmHg vs −40 mmHg). Tolerance to maximal LBNP did not differ among the groups.

Conclusions. Despite differences in the responses to submaximal LBNP, neither age nor cardiovascular fitness affect tolerance to maximal LBNP.

The ability to maintain cerebral blood perfusion with a change in posture is known as orthostatic tolerance. A number of mechanisms enable the human body to maintain blood pressure in an upright posture. Both age and fitness level can affect these mechanisms. Orthostatic hypotension has (1) and has not (2) been reported among older persons. Similar ambiguity exists in the research with respect to endurance athletes who may (3–7) or may not (8,9) have reduced tolerance to orthostatic stress. Many of these aging studies focused on the responses to submaximal orthostatic stress rather than tolerance to maximal orthostatic stress (1,2,10–15). Furthermore, they generally had focused on either aging or fitness, not both. In other words, we are aware of only one study (2) that assessed the effects of aging on maximal orthostatic stress. However, in this study (2), Tsutsui and colleagues applied lower body negative pressure (LBNP) to only −60 mmHg, and only 13 of 37 participants became presyncope; although the participants are described as healthy, their level of fitness was not assessed. Thus, it is entirely unclear how chronic exercise training affects orthostatic tolerance in older persons.

The purpose of this investigation was to determine the effects of age and fitness level on both orthostatic tolerance and cardiovascular responses to orthostatic stress assessed with LBNP. We hypothesized that tolerance to maximal LBNP would be greater in unfit than fit participants. Furthermore, we hypothesized that tolerance to maximal LBNP would be greater in older than younger participants.

Methods

Participants
Younger and older participants free of any diagnosed cardiovascular disease and not on any cardiac medications were recruited from the collegiate and surrounding communities for this investigation. The participants were parsed into four groups based on age and fitness: 20 young participants (age < 30 years; 10 males and 10 females) and 20 older participants (age > 60 years; 10 males and 10 females) were subsequently divided into fit and unfit groups (n = 10 each; 5 men and 5 women) according to their fitness level. All participants underwent a maximal graded exercise test (Bruce protocol for younger participants and Modifed Bruce protocol for older participants) to determine their fitness status. VO₂peak estimates were obtained for all the participants using the time to termination of the stress test as the criterion. The participants were grouped into fit and unfit using age- and sex-based norms (16). All the young women were tested during their follicular phase (days 3–10 following the onset of the menstrual cycle). Participant characteristics are summarized in Table 1.

General Experimental Design
Participants reported to the laboratory on three separate occasions. The first meeting was to assess anthropometric status and determine fitness level. The second visit served to orient the participants to the testing apparatus and protocol. The third visit was for the LBNP tolerance test. Prior to data collection, all participants were verbally informed of the risks and benefits of this study and provided written informed consent. All participants refrained from any exercise and any alcohol, tobacco, or caffeine ingestion for 12 hours, and from any food intake for 3 hours before their LBNP test. The Institutional Review Board of Iowa State University approved this investigation; the results reported here are part of an ongoing investigation in our laboratory.
LBNP Testing
Each participant reported to the laboratory no sooner than 48 hours after the test to estimate VO\textsubscript{2peak} and, following instrumentation, assumed a supine posture inside the LBNP testing chamber. When inside the chamber, they straddled a padded bicycle seat with their feet well clear of the base of the chamber and were sealed at the level of the iliac crest. Following 12 minutes of rest at ambient barometric pressure, negative pressure was induced using a commercially available vacuum and quantified with a pressure transducer (PS309; Validyne, Northridge, CA). Graded LBNP was invoked with 10-mmHg increases in negative pressure every 4 minutes. The LBNP test was terminated when the participant either completed 4 minutes at −100 mmHg, at the onset of presyncope symptoms, or by participant request. Signs of impending presyncope included dizziness, nausea, profuse sweating, or a rapid change in blood pressure (defined as either a decrease in systolic blood pressure by 25 mmHg or a decrease in diastolic blood pressure by 15 mmHg within 1 minute).

Assessment of Cardiovascular Responses
Forearm blood flow (FBF) was measured using mercury-in-silastic strain gauge plethysmography (D.E. Hokanson, Bellevue, WA) with the strain gauge placed around the proximal portion of the left forearm about one-third the distance from the olecranon to the ulnar styloid. A wrist cuff was used to occlude circulation to the hand during forearm blood-flow measurements; FBF was assessed every 20 seconds. Blood pressure was measured every minute via a Dinamap (J&J Medical, Tampa, FL). Heart rate was assessed continuously using 5-lead electrocardiography. Stroke volume was determined every minute using impedance cardiography (Minnesota Impedance Cardiograph Model 304B; Surcom, Minneapolis, MN) and ensemble R-wave averaging on 55 seconds of data acquired in each 60-second period. The analog heart rate and forearm blood-flow signals were input into an on-line personal computer system using commercially available software (BIOPAC, Santa Barbara, CA). The heart rate and impedance cardiograph signals were also analyzed using commercially available software (Microtronics, Chapel Hill, NC) to determine stroke volume and calculate cardiac output.

Analysis
LBNP tolerance was quantified as the LBNP tolerance index (LTI). This index was calculated as the sum of the products of duration spent at each negative pressure and the change in pressure from the previous stage (17). The LTI is a linear function for the conditions used in the present study. Cardiac output (Q) was calculated every minute as the product of stroke volume and the concurrent heart rate. Mean arterial pressure (MAP) was determined every minute from the Dinamap. Forearm vascular conductance (FVC) was calculated as FBF/MAP, and total peripheral conductance (TPC) as Q/MAP. The anthropometric, estimated VO\textsubscript{2peak} and LTI data were compared using two-way analysis of variance (age \times fitness). Mean cardiovascular responses for minutes 2–4 of each stage of LBNP common to all participants, the last completed stage of each participant, and each of the last 2 minutes of LBNP were compared using three-way (age \times fitness \times negative pressure) repeated measures analysis of variance. Post hoc comparisons were made using the Tukey test. Statistical significance was set at .05 with data reported as means \pm standard error of the mean.

RESULTS
Table 1 summarizes the anthropometric characteristics of all participants (n = 20 women and 20 men) in the study. The groups did not differ significantly in either height or body surface area. As desired, the groups differed significantly with respect to age and fitness (p < .001). The only exception was that old fit (OF) and young unifit (YU) groups did not differ in their fitness. The unfit groups were fatter than their fit peers (p < .001). The young fit (YF) group was leaner than the other three groups (p < .001). Importantly, the groups did not differ in LTI. All participants terminated the protocol due to signs or symptoms of impending presyncope except for three participants who finished the entire protocol. One was in the YF group and two were in the older unfit (OU) group. Their data were excluded from the comparisons of the last 2 minutes of LBNP.

Resting Data
The resting heart rate of the YF group was significantly lower than that in the other groups (Table 2) (p < .001). The YF group had significantly higher resting stroke volumes than did the OU group (Table 2) (p < .030). Cardiac output did not differ between any of the groups (Table 2). The OU group had significantly higher blood pressures than did all the other groups (p < .001) (Table 2). The YF group had a significantly lower diastolic blood pressure than did the OU group and the OF group (Table 2) (p < .001). FVC was higher in the OF group than in the other groups (p < .05). TPC tended to be lower in the OU group than in the other groups (p = .054; Table 2).

Cardiovascular Responses to Submaximal LBNP
Hemodynamic responses to LBNP are illustrated in Figures 1–3. Two participants reached presyncope (one OU female and one OF male) before −40 mmHg of LBNP.
DISCUSSION
The purpose of this study was to examine the effects of age and exercise training on orthostatic tolerance using maximal LBNP. We hypothesized that tolerance to maximal LBNP would be greater in unfit than in fit participants. Furthermore, we hypothesized that tolerance to maximal LBNP would be greater in older than in younger participants. In this study, all the groups tolerated graded maximal LBNP equally well. Thus our findings do not support our hypotheses, suggesting that neither age nor fitness affect orthostatic tolerance. However, there were small, statistically significant between-group differences in these responses. The YF group responded to LBNP with a greater tachycardic response and higher initial stroke volumes. The OU group had higher resting blood pressures, which were maintained throughout the protocol. The OF group coped with a reduced central blood volume by decreasing TPC more than the other groups did. Thus, our data suggest that participants differing in age and fitness maintain blood pressure in the face of a marked

and were excluded from these analyses. There were no significant main effect differences between the groups other than those seen at rest. However, there were some LBNP by group interactions.

In all the groups, heart rate rose above rest at the last stage (Figure 1) (p < .001); in the YF group, heart rate rose above rest at −40 mmHg (Figure 1) (p < .001). Stroke volume fell below rest at −40 mmHg in all the groups except the YF group; in this group, it did so at −20 mmHg (p < .001) (Figure 1). The increases in heart rate offset the declines in stroke volume such that all the groups experienced similar decreases in the cardiac output across the protocol (Figure 2).

The OU group maintained significantly higher MAP than did the other groups with LBNP to −40 mmHg (Figure 2, Table 2) (p < .001). However, compared to the other groups, the OU group had significant decreases in MAP at the last completed stage (p < .001). The LBNP stage at which TPC decreased below rest differed between groups: −20 mmHg in the OF group, −30 mmHg in the YF group, −40 mmHg in the YU group (p < .001; Figure 3), whereas TPC in the OU group did not differ significantly from TPC at rest. The OF group showed a significant decrease in FVC below rest (−30 mmHg; p < .001), whereas FVC did not change significantly in the other groups (Figure 3).

Presyncope
Results are presented as 1 minute before the end (E-1) and the last minute of the test (E) (Figures 1–3). Three participants finished the protocol and were excluded from this analysis. The test was concluded due to presyncopal symptoms in the remainder of the participants. Heart rate was significantly higher than rest for the YU and YF groups, and stroke volume was significantly lower than rest for all groups (p < .001) (Figure 1). Cardiac output was lower than rest for all groups in the last completed stage of LBNP and at E-1 and E for the OU and OF groups (p < .001). MAP was significantly lower than rest at E-1 for only the OU group, but significantly was lower than rest at E for all the groups (p < .001) (Figure 2). There were no changes observed in FVC or TPC in the last 2 minutes of the test (Figure 3).
orthostatic challenge, but do so utilizing different cardiovascular mechanisms.

Previous studies have found conflicting results concerning the effects of exercise training on the hemodynamic responses to LBNP and LBNP tolerance. Convertino (9) found that the better the fitness status, the better the orthostatic tolerance. However, Raven and Pawelczyk (3) showed that high fit (60 ± 0.8 ml·kg⁻¹·min⁻¹) participants had lower tolerance to LBNP than did mid fit (48.9 ± 1 ml·kg⁻¹·min⁻¹) and low fit (35.7 ± 0.9 ml·kg⁻¹·min⁻¹) participants. More recently, Franke and colleagues (8) compared the tolerance of highly trained swimmers (69.5 ± 2.6 ml·kg⁻¹·min⁻¹) and runners (70.0 ± 1.6 ml·kg⁻¹·min⁻¹) and found that the tolerances of these participants were comparable to much less fit participants. In the present study, the less fit groups had tolerances to LBNP comparable to those of their considerably more fit peers, suggesting that cardiovascular fitness does not affect tolerance.

The effects of age on orthostatic tolerance remain unclear. There is an attenuated increase in heart rate among the older population as compared to younger persons in response to similar submaximal orthostatic stress (1,10,11). Recently, Tsutsui and colleagues (2) found that the LBNP-associated increase in heart rate and decrease in stroke volume were smaller in older participants than in younger participants. More recently, Franke and colleagues (8) compared the tolerance of highly trained swimmers (69.5 ± 2.6 ml·kg⁻¹·min⁻¹) and runners (70.0 ± 1.6 ml·kg⁻¹·min⁻¹) and found that the tolerances of these participants were comparable to much less fit participants. In the present study, the less fit groups had tolerances to LBNP comparable to those of their considerably more fit peers, suggesting that cardiovascular fitness does not affect tolerance.

In assessing the interactive effects of age and fitness, Fortney colleagues (12) found that highly trained (52.4 ± 1.7 ml·kg⁻¹·min⁻¹) older participants had smaller decreases in cardiac volumes and MAP and smaller increases in heart rate in response to submaximal LBNP as compared to a control group (31.0 ± 2.9 ml·kg⁻¹·min⁻¹). Our OU group had higher resting MAP and lower TPC; with LBNP, MAP declined earlier in this group at the last stage, becoming similar to that of the OF group. Neither MAP nor TPC differed significantly in the last 2 minutes of LBNP. Gabbett and colleagues (13) found that a cycling endurance-training program in healthy physically active older men elicited increases in VO₂peak without significant changes in cardiovascular responses to a 90° head-up tilt. In neither study (12,13) was orthostatic tolerance explicitly assessed. Nevertheless, the findings of the present investigation are consistent with these studies—higher levels of fitness affect the responses to submaximal LBNP in older participants without affecting orthostatic tolerance.

It has been suggested that young participants with VO₂peak above 55 (9) or 65 ml·kg⁻¹·min⁻¹ (3) are prone to reduced orthostatic tolerance. One of the reasons suggested was a reduced tachycardic response (6,9). This attenuated response was not seen in this study. The YF group was the most fit group (57 ml·kg⁻¹·min⁻¹), yet it had the most
pronounced tachycardic response to LBNP. It may be that the level of fitness necessary to attenuate the heart rate response to orthostatic stress, and in turn tolerance, is higher than the fitness level of the YF group in the present investigation.

Several limitations of the present investigation need to be recognized. First, this study was cross-sectional in nature, so we do not know if pre-existing differences in orthostatic tolerance affected our findings. Second, our participants were relatively unique in that they were apparently healthy and not on any medications that might have affected their responses to LBNP. Finally, this healthy status may have contributed to our OU group actually being fair-to-average in fitness for their age (16).

Conclusion
Neither aging nor fitness level affect tolerance to the orthostatic stress of LBNP. Although differences were observed with age and fitness in the responses to submaximal orthostatic stress, these differences did not translate to differences in tolerance.

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