When humans assume the upright posture (orthostasis), a significant volume of blood is displaced from the central circulation into the veins of the legs [1, 2]. The rapid reduction in central blood volume results in a fall in central venous pressure, stroke volume and cardiac output [1–3]. In an attempt to maintain arterial pressure (AP), the arterial baroreceptors, (located in the carotid sinus and aortic arch), reduce their firing rates, resulting in a reflex increase in heart rate (HR) and total peripheral resistance [4, 5]. Despite the falling cardiac output, AP is either maintained or elevated, due largely to the increase in sympathetically mediated vasoconstriction [6]. However, while some humans may tolerate the reduced cardiac output, others are unable to compensate for the falling atrial and ventricular filling pressures, resulting in orthostatic intolerance [2, 3].

Normotensive orthostatic stress evokes significant increases in muscle sympathetic nerve activity and norepinephrine (NE) concentrations and non-significant rises in epinephrine (EPI) concentrations [7–10]. However, in young individuals, orthostatic intolerance is associated with a sudden cessation of sympathetic outflow and the inhibition of vasoconstrictor impulses.

Abstract: In young individuals, orthostatic intolerance is associated with marked increases in plasma epinephrine (EPI) concentrations and attenuated rises in plasma norepinephrine (NE) concentrations. This study investigated the cardiovascular, EPI and NE responses of healthy elderly males during orthostatic stress. Twelve men (68±1 yr) with a recent history of orthostatic hypotension and who exhibited orthostatic intolerance (HYPO) during 90° head-up tilt (HUT) were compared with 12 men (69±1 yr) without a history of orthostatic hypotension and who remained normotensive (NORMO) throughout 90° HUT. Beat-by-beat recordings of heart rate (HR), mean (MAP), systolic (SBP), diastolic (DBP), and pulse (PP) pressures were made throughout 90° HUT. Blood samples obtained during supine rest and 90° HUT were analyzed for changes in EPI and NE concentrations, hematocrit, hemoglobin and plasma volume. Compared to supine rest, orthostatic intolerance was characterized by significant reductions (p<0.0001) in MAP, SBP, DBP, and PP. The HR, MAP, SBP, DBP, and PP at the termination of 90° HUT was significantly lower (p<0.0001) for HYPO than NORMO. The 90° HUT position resulted in significant increases (p<0.01) in NE for both HYPO and NORMO, with the rise in NE significantly lower (p<0.05) in HYPO. There were no differences between groups regarding EPI concentrations at the termination of 90° HUT. These results suggest that the magnitude of arterial pressure (AP) reduction does not influence the EPI response during orthostasis in healthy elderly men. However, marked reductions in AP, leading to orthostatic intolerance, are associated with inadequate increases in NE in these individuals. [Japanese Journal of Physiology, 50, 59–66, 2000]
The finding of diminished sympathetic activity during orthostatic intolerance in young individuals has also been confirmed by others [8, 9]. Ziegler et al. [8] found that during standing in young subjects, plasma NE concentrations were significantly decreased when mean arterial pressure (MAP) fell from 84 to 53 mmHg. In young men and women undergoing head-up tilt (HUT), marked reductions in MAP, from 94 to 50 mmHg, were associated with attenuated NE concentrations [9]. Additionally, previous investigators [9–13] have reported significant increases in plasma EPI concentrations during orthostatic intolerance in young individuals. During orthostatic intolerance in young men and women, marked reductions of 37 and 44 mmHg in MAP have been associated with a two- to eight-fold increase in plasma EPI concentrations [9, 11–13]. While it is unknown if the significant increases in EPI concentrations are the cause or the result of hypotension, it has been suggested that EPI may contribute to orthostatic intolerance through its ability to cause peripheral vasodilation [3, 11, 13, 14] and stimulate cardiac vagal fibres [3, 15, 16].

Elderly individuals are reported to be highly susceptible to orthostatic intolerance [2, 17, 18]. However, while marked reductions in AP resulting in orthostatic intolerance are associated with characteristic changes in EPI and NE in young individuals [8–13], the sympathoadrenal responses of elderly individuals during orthostatic intolerance have been inadequately addressed. When elderly individuals experience moderate reductions in MAP, from 100 to 87 mmHg, during orthostatic stress, the increase in NE concentration is not significantly different from normotensive control subjects [19]. Furthermore, moderate reductions (13 to 25 mmHg) in MAP during HUT have failed to significantly alter plasma EPI concentrations in elderly subjects [19–21]. These results may suggest that the magnitude of AP reduction is important in determining the EPI and NE response to the upright posture. The present investigation examined the EPI and NE responses of healthy elderly men, with and without susceptibility to orthostatic intolerance. We hypothesized that, in healthy elderly men, absolute and relative reductions in MAP that were similar to that previously reported in young individuals would be associated with significant increases in EPI concentrations and attenuated rises in NE concentrations.

METHODS

Subjects. Twenty-four healthy men aged between 65 and 75 yr participated in this study. Twelve men with a history of orthostatic hypotension (HYPO) were compared to twelve age-matched control subjects (NORMO). All HYPO subjects had exhibited intolerance to the upright posture on at least one occasion within the 6 months prior to the experiment, and failed to maintain a MAP above 50 mmHg during a pre-screening 15-min head-up tilt and standing test. None of the NORMO subjects had a history of orthostatic hypotension. Additionally, all NORMO subjects completed the 15-min pre-screening standing and head-up tilt test without any signs or symptoms of orthostatic hypotension. Subjects were non-smokers with normal pulmonary function and normal resting twelve-lead electrocardiogram and echocardiogram. Each subject had a resting AP of less than 160/90 mmHg. In addition, each subject completed a comprehensive medical evaluation and incremental exercise test to volitional exhaustion without any clinically significant findings. Subjects were not prescribed or taking any medication that was likely to interfere with cardiovascular or sympathoadrenal responses. Subjects received a clear explanation of the study, including the risks and benefits of participation, and written consent was obtained. The Griffith University Ethics Committee approved all experimental procedures.

Experimental procedures. During a preliminary visit to the laboratory, each subject was familiarized with all procedures. Familiarization sessions were identical for all subjects. Standard anthropometry (height, body mass and skinfold thickness at four sites: triceps, biceps, subscapular, suprailiac) and pulmonary function tests were also conducted during the familiarization sessions.

Blood volume estimation. Total blood volume (BV) was estimated for each subject using the following prediction equation [22]:

$$\text{Estimated BV (l)} = 0.414 \times \text{Height}^3 + 0.0328 \times \text{Weight}^{0.03},$$

where Height and Weight are the subjects’ height (m) and body mass (kg), respectively. When compared to the Evans blue (T-1824) dye dilution technique, the prediction of BV from body mass and cube of height has been shown to provide a valid estimate of total BV [23].

Peak oxygen consumption. Following a successful medical examination, subjects underwent a medically supervised incremental exercise test to volitional exhaustion on an electronically braked cycle ergometer (Lode Excalibur, Groningen, Netherlands). The incremental exercise test began with a 3 min warm up at 15 W, after which power was incremented by 5 W every 20-s until volitional exhaustion. Peak
oxygen consumption ($\bar{V}O_2$ peak) was determined for each subject using open-circuit spirometry. The pneumotach (Hans Rudolph 3830, Kansas City, MO, USA) and oxygen and carbon dioxide analyzers (Exerstress OX21, CO21, Sydney, Australia) were calibrated prior to and following each test using volumes and gases of known concentrations. Oxygen consumption ($\bar{V}O_2$), carbon dioxide production ($\bar{V}CO_2$), pulmonary minute ventilation ($\bar{V}E$, BTPS) and respiratory exchange ratio (RER) were recorded at 20-s intervals throughout the incremental exercise test. Peak exercise values were determined as the highest values obtained during the incremental exercise test.

90° head-up tilt protocol. A custom-made padded tilt-table was used to investigate the cardiovascular and sympathoadrenal responses during orthostasis. The tilt-table rotated around a central axis that could be adjusted to 90° within 2 s. Each subject was familiarized with the operation of the tilt-table on at least one occasion prior to the 90° HUT test. Approximately 1 week after being familiarized with the tilt-table, the 90° HUT test was administered to all HYPO subjects. Identical procedures were then performed on all NORMO subjects. All tests were conducted in a temperature-controlled environment (22–24°C DB). Subjects were encouraged to void their bladder before commencement of the test. The subjects lay supine and electrocardiographic (ECG) electrodes were applied in the CM5 position. Beat-by-beat measurements of heart rate (HR) were obtained from an electrocardiograph (Lohmeier M607, Munchen, Germany) while continuous systolic (SBP), diastolic (DBP), and mean arterial pressure (MAP) were obtained non-invasively via a Finapres (Ohmeda 2300, Louisville, CO USA), which was applied to the subject’s left hand. The Finapres device was maintained by supportive strapping at heart level throughout the entire experimental procedure. Three 10-cm wide adjustable straps secured the subjects to the table. The straps were firmly, but comfortably applied around the chest, thigh, and lower leg of the participants. The amplified signals from the Finapres and electrocardiograph were collected at 100 Hz using a Biopac system (Model MP 100) and Acqknowledge 2.0 Software Package.

A 21-gauge venous catheter was inserted into a right antecubital vein under sterile conditions for multiple blood sampling. Subjects were requested to remain relaxed, maintain normal respiration, and avoid all muscle contraction. The lights in the room were dimmed and subjects were instructed to close their eyes in an attempt to promote relaxation. Following insertion of the venous catheter, subjects rested in the supine position for 20 min. A 10-ml blood sample was collected at the completion of the 20-min rest period. To minimize anticipatory changes in HR or AP, each subject was randomly assigned an additional resting period ranging from 2–10 min prior to 90° HUT. At the completion of the additional randomized 2–10 min rest period, subjects were tilted smoothly to the 90° HUT position within 2 s. Beat-by-beat AP and HR were recorded continuously throughout the entire 90° HUT experiment. A cushioned footplate supported the subject during 90° HUT. No conversation was made between the subject and investigators throughout the tilt-table experiment. When compared to direct intraarterial pressure, it has been demonstrated that Finapres provides a valid measurement of arterial pressure during prolonged orthostatic stress [24]. Furthermore, the intraclass correlation coefficients for test-retest reliability for HR and MAP responses during prolonged 90° HUT were 0.90 and 0.97, respectively [25].

Subjects were monitored for signs and symptoms of presyncope. Presyncope was defined as the interval between the onset of sustained hypotension (≥5 mmHg in MAP) during 90° HUT and orthostatic intolerance [26]. The 90° HUT test was terminated if MAP fell below 50 mmHg or by subject request. The fall in MAP was usually associated with symptoms of nausea, clammy skin, profuse sweating, and pallor of the skin. If presyncopal symptoms developed or MAP fell below 50 mmHg, blood samples were drawn and the subject was immediately returned to the supine position. For all NORMO 90° HUT tests, blood samples were obtained at times similar to those obtained for HYPO. Blood samples were analyzed for plasma EPI and NE using high-performance liquid chromatography (HPLC) coupled with electrochemical detection. Using this procedure, the intra and inter-assay coefficients of variation for EPI and NE were 2.8 and 3.8%, and 2.0 and 4.0%, respectively. Hematocrit (Ht) and hemoglobin (Hb) were determined using a Coulter Counter (Coulter Electronics, Inc., Model T660, Hialeah, FL, USA). The intraclass correlation coefficients for test-retest reliability for the determination of Ht and Hb were 0.97 and 0.97, respectively [27]. Plasma volume (PV) changes were calculated according to the equations described by Greenleaf et al. [28]. Values for EPI and NE were corrected for changes in PV using the following equation:

$$\text{NE}_{\text{corr}} \text{ or } \text{EPI}_{\text{corr}} = [100\% + \Delta \text{PV} (\%)] \times \text{NE}_{\text{meas}} \text{ or } \text{EPI}_{\text{meas}}$$

where subscripts meas represents the measured values for NE and EPI uncorrected for changes in PV, and

corr represents the measured values corrected for changes in PV.

AP and HR were recorded and averaged using procedures recently described by Jardine et al. [26]. Values were obtained before 90° HUT (−10, −5 min); baseline (0 min); early 90° HUT (1 min); and throughout 90° HUT (5, 10, 15 min) or until orthostatic intolerance. Presyncopal recordings were taken from the onset of presyncope and 1 min before orthostatic intolerance [26].

Statistics. Data were analyzed using SAS software integrated with a Unix system (SAS Version 6.12, SAS Institute Inc., NC 27513, USA). Supine resting and 90° HUT responses for HR, MAP, SBP, DBP, and PP were compared using repeated measures ANOVA. Differences in physical characteristics and changes in EPI, NE, Ht, Hb and PV were compared between groups using independent samples and paired-samples t-tests, respectively. The level of significance was set at $p<0.05$ and all data are reported as mean±SE.

RESULTS

Table 1. Physical characteristics, spirometry and maximum exercise test results of participating subjects.

<table>
<thead>
<tr>
<th></th>
<th>Hypotensive</th>
<th>Normotensive</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>68±1</td>
<td>69±1</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>173±1</td>
<td>173±1</td>
</tr>
<tr>
<td>Body mass (kg)</td>
<td>81±4</td>
<td>77±2</td>
</tr>
<tr>
<td>Sum of four skinfolds (mm)</td>
<td>48±3</td>
<td>48±2</td>
</tr>
<tr>
<td>Estimated blood volume ($l$)</td>
<td>4.75±0.2</td>
<td>4.65±0.1</td>
</tr>
<tr>
<td>FEV$_1$ ($l$, BTPS)</td>
<td>3.2±0.1</td>
<td>3.0±0.2</td>
</tr>
<tr>
<td>FVC ($l$, BTPS)</td>
<td>3.9±0.1</td>
<td>3.7±0.2</td>
</tr>
<tr>
<td>FEV$_1$/FVC (%)</td>
<td>81±1</td>
<td>81±1</td>
</tr>
<tr>
<td>$\dot{V}O_2$ peak ($l \cdot min^{-1}$)</td>
<td>1.98±0.1</td>
<td>1.89±0.1</td>
</tr>
<tr>
<td>$\dot{V}O_2$ peak (ml kg$^{-1}$ min$^{-1}$)</td>
<td>24.1±0.9</td>
<td>24.7±1.0</td>
</tr>
<tr>
<td>HR peak (beats min$^{-1}$)</td>
<td>145±7</td>
<td>158±5</td>
</tr>
<tr>
<td>$\dot{V}E$ peak ($l \cdot min^{-1}$, BTPS)</td>
<td>79.2±3.9</td>
<td>85.0±7.7</td>
</tr>
<tr>
<td>Power peak (W)</td>
<td>162±5</td>
<td>162±5</td>
</tr>
</tbody>
</table>

Hypotensive group: $n=12$; normotensive group: $n=12$. Values are mean±SE.

The physical characteristics and results of the incremental exercise test to exhaustion are presented in Table 1. No significant differences ($p>0.05$) were found between HYPO and NORMO groups for any physical characteristics or incremental exercise test results. The estimated blood volume was similar ($p>0.05$) between the groups.

Fig. 1. Heart rate and arterial pressure (mean, systolic, diastolic, pulse) during supine rest and 90° head-up tilt for men aged 65 to 75 yr. NORMO, normotensive group ($n=12$); HYPO, hypotensive group ($n=12$). Values are mean±SE and time intervals are described in text. et, early tilt; ps1, onset of presyncope; ps2, 1 min before orthostatic intolerance; oi, orthostatic intolerance; HR, heart rate; MAP, mean arterial pressure; SBP, systolic blood pressure; DBP, diastolic blood pressure; PP, pulse pressure.
Orthostatic Intolerance in Elderly Men

Table 2. Absolute and relative changes in plasma norepinephrine (NE) and epinephrine (EPI) concentrations, hematocrit (Ht), hemoglobin (Hb), and plasma volume (PV) during supine rest and 90° head-up tilt (HUT) for men aged 65 to 75 yr.

<table>
<thead>
<tr>
<th></th>
<th>Hypotensive</th>
<th>Normotensive</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Supine</td>
<td>HUT</td>
</tr>
<tr>
<td>NE&lt;sub&gt;meas&lt;/sub&gt; (pg ml&lt;sup&gt;-1&lt;/sup&gt;)</td>
<td>197±24</td>
<td>297±38*</td>
</tr>
<tr>
<td>NE&lt;sub&gt;corr&lt;/sub&gt; (pg ml&lt;sup&gt;-1&lt;/sup&gt;)</td>
<td>—</td>
<td>267±32*</td>
</tr>
<tr>
<td>EPI&lt;sub&gt;meas&lt;/sub&gt; (pg ml&lt;sup&gt;-1&lt;/sup&gt;)</td>
<td>28±5</td>
<td>42±7</td>
</tr>
<tr>
<td>EPI&lt;sub&gt;corr&lt;/sub&gt; (pg ml&lt;sup&gt;-1&lt;/sup&gt;)</td>
<td>—</td>
<td>38±7</td>
</tr>
<tr>
<td>Ht (%)</td>
<td>42.9±0.4</td>
<td>45.4±0.6*</td>
</tr>
<tr>
<td>Hb (g dl&lt;sup&gt;-1&lt;/sup&gt;)</td>
<td>13.7±0.2</td>
<td>14.8±0.2*</td>
</tr>
<tr>
<td>PV (%)</td>
<td>—</td>
<td>—</td>
</tr>
</tbody>
</table>

Hypotensive group: n=12; normotensive group: n=12. Blood samples obtained after 3–14 (8±1) min for both hypotensive and normotensive groups. Values are mean±SE. *Significantly different from supine rest (p<0.05). † Hypotensive group significantly different from normotensive group (p<0.05). meas: measured values uncorrected for changes in plasma volume; corr: measured values corrected for changes in plasma volume.

Cardiovascular responses

The AP and HR responses for HYPO and NORMO groups during 90° HUT are presented in Fig. 1. All subjects with a history of orthostatic hypotension exhibited orthostatic intolerance during 90° HUT, while all NORMO subjects completed the 90° HUT test without any signs or symptoms of orthostatic hypotension. Marked reductions in AP (decreased 50 mmHg) occurred in the HYPO group after an average of 8±1 (range: 3–14) min. During supine rest, no significant differences (p>0.05) were found between the HYPO and NORMO groups for HR, MAP, SBP, DBP and PP. Multivariate repeated measures analysis revealed significant differences in HR over time (p<0.0001) with significant group × time interactions during 90° HUT (p<0.01). As expected, significant differences (p<0.0001) were observed between the HYPO and NORMO groups for MAP, SBP, DBP, and PP responses during 90° HUT. Significant time (p<0.0001) and group × time (p<0.0001) interactions were observed for MAP, SBP, DBP, and PP responses during 90° HUT. During 90° HUT, the NORMO group maintained an AP at values similar to supine rest. However, HR (p<0.05), MAP, SBP, DBP, and PP for the HYPO group were significantly (p<0.0001) lower than measurements for the NORMO group at the termination of 90° HUT.

Plasma norepinephrine and epinephrine concentrations

The absolute and relative changes in plasma NE and EPI concentrations at the termination of 90° HUT are shown in Table 2. Blood samples were obtained after 8±1 (range: 3–14) min for both HYPO and NORMO groups. No significant differences (p>0.05) existed between the groups for supine resting concentrations of plasma NE or EPI concentrations. Plasma NE concentrations were significantly increased (p<0.01) at the termination of 90° HUT for both HYPO and NORMO groups. However, the relative change in plasma NE concentrations at the termination of 90° HUT was significantly lower (p<0.05) for the HYPO group as compared to the NORMO group. Plasma EPI concentrations at the termination of 90° HUT were not significantly different (p>0.05) from supine rest for both HYPO and NORMO groups. No differences (p>0.05) existed for changes in plasma EPI concentrations between groups.

Hematocrit, hemoglobin, and plasma volume changes

Absolute and relative changes in Ht, Hb, and PV at the termination of 90° HUT are presented in Table 2. No significant differences (p>0.05) were found between the HYPO and NORMO groups for supine resting Ht and Hb. Compared to supine rest, significant increases (p<0.001) were found in Ht and Hb during 90° HUT for both HYPO and NORMO groups. No significant differences (p>0.05) were found between the HYPO and NORMO groups for ΔHt and ΔHb. Accordingly, the reduction in PV at the termination of 90° HUT was similar (p>0.05) for the HYPO and NORMO groups.

DISCUSSION

The present study investigated the EPI and NE responses of healthy elderly men with and without susceptibility to orthostatic intolerance. The results of this study suggest that the magnitude of AP reduction...
does not influence the EPI response during orthostasis in healthy elderly men. However, marked reductions in AP, leading to orthostatic intolerance, are associated with inadequate increases in NE in these individuals.

Previous investigators [9–13] have reported significant increases in EPI during orthostatic intolerance in young individuals. Indeed, plasma EPI concentrations at the termination of orthostatic stress have been reported to be two- to eight-fold greater in young subjects experiencing symptomatic hypotension [9–13]. Due to its ability to cause peripheral vasodilation and stimulate cardiac vagal fibres, EPI has been implicated as a possible mechanism responsible for orthostatic intolerance [3, 11, 13–16]. The present study found no significant differences in plasma EPI concentrations during HYPO and NORMO 90° HUT in a homogeneous group of healthy elderly men. During standing in young individuals, Robinson and Johnson [11] reported a five-fold increase in EPI when MAP fell from 90 to 58 mmHg. Similar increases in EPI have been reported during HUT when MAP fell from 94 to 50 mmHg [9]. Conversely, during HUT in the elderly, moderate reductions in MAP from 100 to 87 mmHg [19], 99 to 74 mmHg [20], and 88 to 72 mmHg [21] did not result in significant increases in EPI concentrations. These results may suggest that moderate reductions in MAP do not elicit significant increases in EPI, and during orthostasis, a significant EPI response is dependent on an absolute and relative reduction in AP. However, the present study found no significant change in EPI concentrations with a symptomatic reduction in MAP from 81±3 to 43±2 mmHg in healthy elderly men. The results of the present study suggest that significant increases in EPI concentrations are not associated with orthostatic intolerance in healthy elderly men.

The removal of sympathetic tone has been suggested as a possible cause of orthostatic intolerance [7–9, 26]. In the present study, the lower NE concentrations in the HYPO group, coupled with marked differences in HR and AP, confirms that inadequate sympathetic outflow may be responsible for orthostatic intolerance in healthy elderly men. It is well documented that the arterial baroreceptors provide the critical regulation of AP during orthostatic stress [4], and that the unloading of arterial baroreceptors results in reflex increases in sympathetic outflow and decreases in parasympathetic activity [29]. Significant reductions in AP and PP, coupled with increases in HR have been suggested as evidence of arterial baroreceptor activation [30]. Therefore, it is likely that the previously documented increases in HR and NE that accompanied moderate reductions in AP during orthostasis in the elderly [19–21] reflected an appropriate baroreflex response. However, in the present study, the finding of a decrease in HR, despite a significantly lower AP, PP, and NE, suggests that baroreflex regulation of sympathetic outflow and AP is diminished during orthostatic intolerance.

It has been suggested that susceptibility to fainting is increased with lower blood volume [31, 32] and that the magnitude of central hypovolemia determines the cardiovascular responses to orthostatic stress [2, 21]. Unlike results in younger individuals [31], in the present study, blood volume (as estimated from height$^3$ and body mass) was also not significantly different between the HYPO and NORMO groups. Furthermore, the percentage change in plasma volume during 90° HUT was also not significantly different between groups. These findings are in agreement with Tarazi et al. [33], who found similar reductions in plasma volume during HUT in hypertensive fainters and non-fainters. In the present study, the differences in cardiovascular responses between the groups during 90° HUT, concomitant with similar estimated blood volume and plasma volume reductions, suggest that orthostatic tolerance in healthy elderly men is mediated through factors in addition to or other than circulating blood volume [2]. While considered a valid method for estimating total blood volume [22, 23], the use of height and body mass to predict blood volume should be utilized with caution as vascular volumes may also be influenced by aerobic capacity and lean body mass [34]. However, the effects of changes in aerobic capacity on blood volume are equivocal, with reports of elevated or unchanged [34–36] vascular volumes in the endurance-trained state. Furthermore, in elderly men, exercise-induced hypervolemia may occur without significant changes in $V_{O2 \text{peak}}$ [37]. Therefore, despite having similar aerobic capacities and lean body mass (as estimated from the sum of four skinfolds), differences in the measured blood volumes of HYPO and NORMO subjects cannot be discounted. Nonetheless, while acknowledging the potential factors that may influence vascular volumes, the similar $V_{O2 \text{peak}}$, height, body mass, and lean body mass of the HYPO and NORMO subjects suggest that total blood volume did not differ between the groups.

It is well documented that HUT evokes increases in the electromyographic activity of the soleus [38], gastrocnemius [5], and tibialis anterior [38] muscles. In the present study, a cushioned footplate supported the subjects during 90° HUT. While all subjects were given identical instructions to relax antigravity muscles, the possibility of differences in muscle tension
impacting on cardiovascular responses must be considered. It is possible that the ability to maintain AP in the NORMO group was accomplished through increased tension in the antigravity musculature, thereby producing accentuated compression of the vascular tree, and allowing greater venous return to the heart [2, 3, 38, 39].

In summary, the results of this study suggest that the magnitude of AP reduction does not influence the EPI response during orthostasis in healthy elderly men. However, marked reductions in AP, leading to orthostatic intolerance, are associated with inadequate increases in NE in these individuals.

The authors would like to thank the volunteer subjects for their support of this project. The authors would also like to express their appreciation to the Princess Alexandra Hospital Brisbane for their assistance with the plasma catecholamine assay. This research was funded by the National Health and Medical Research Council of Australia and Griffith University.

REFERENCES

Physiol 47: 1031–1038, 1979