Compression leggings modestly affect cardiovascular but not cerebrovascular responses to heat and orthostatic stress in young and older adults

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Received: 7 July 2010 / Accepted: 10 April 2011 / Published online: 28 April 2011
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Abstract We tested the hypothesis that wearing commercially available compression leggings would attenuate postural reductions in mean arterial blood pressure (MAP) and cerebral perfusion during heat stress, particularly in older adults. Six older (70 years $\pm$ 4) and six younger (29 years $\pm$ 4) males were heated (esophageal temperature raised 0.5°C) in a water-perfused suit whilst wearing compression or control leggings (>1 week apart, randomized order). Blood flow velocity in the middle cerebral artery (MCAv), blood pressure (photoplethysmography), total peripheral resistance (TPR; Model-Flow) and the partial pressure of end-tidal carbon dioxide were measured continuously before and during 3-min standing in each thermal state. When supine,

compression leggings did not change any cardiorespiratory variables in either age group or thermal condition ($P>0.05$). Upon standing, wearing compression leggings delayed ($\sim 15\%$; $P=0.044$) the maximal drop (nadir) in MAP irrespective of age or thermal condition. During the last minute of standing, wearing compression leggings in normothermia increased TPR (+16%) in older participants but dropped TPR (-8%) in younger participants ($P=0.004$ compression $\times$ age group). When standing and heated, wearing compression leggings lowered TPR in older and younger participants ($\sim 43\%$; $P<0.01$) without changing MAP or MCAv ($P>0.05$). In older adults, when standing, compression leggings maintained MAP by elevating TPR. In contrast, under combined heat and orthostatic stress, wearing compression leggings dropped TPR in both older and younger adults, though MAP and MCAv were maintained.

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Keywords Compression · Ageing · Cardiovascular ·
Cerebral blood flow

Introduction

Fainting, or syncope, is characterized by a transient loss of consciousness due to cerebral hypoperfusion. The initial period of standing represents a critical period for cerebral perfusion as the speed and magnitude of the fall in arterial blood pressure (BP), particularly systolic BP (Romero-Ortuno et al. 2010), may exceed baroreflex adjustment of vascular resis-

tance, resulting in cerebral hypoperfusion and syncope (Wieling et al. 2007; Thomas et al. 2009). Conversely, syncope during sustained orthostasis is reportedly dependant on the neural and vasomotor reserve available for vasoconstriction (Fu et al. 2004). Indeed, vasovagal (reflex) syncope is the most prevalent identified cause of fainting and is generated by systemic arterial hypotension, resulting from reflex vasodilation, bradycardia or both (Brignole et al. 2004).

Epidemiological studies show that the prevalence of syncope in the general population has a bimodal age distribution, teenagers and the elderly reporting a higher incidence (Vaddadi et al. 2007). In older populations, this is likely the result of age-related changes to cardiovascular and cerebrovascular systems including lower cerebral blood flow (Ainslie et al. 2008), a diminished cardiac output ($\dot{Q}$), attenuations in autonomic outflow (Lakatta 1993b) and a general widening and hardening of arterioles and venules (Lakatta 1993a). The majority of age-related research to date has focused on sustained orthostasis. Furthermore, the few studies investigating age and initial orthostatic stress have produced equivocal findings; some have shown age-related differences upon standing (Lucas et al. 2008; Shi et al. 2008) and others have not (Wieling et al. 1992). Thus, even though initial orthostatic hypotension can be well tolerated in healthy younger adults (Thomas et al. 2010), it is less clear whether the same applies for healthy older adults.

‘Classical’ vasovagal syncope can be mediated by either emotional or orthostatic stress (Brignole et al. 2004), with a warm environment reported as a common trigger (Ganzeboom et al. 2003). During heat and orthostatic stress there is a significant increase in venous pooling. This displacement of blood away from the heart creates a regulatory problem, decreasing central blood volume, ventricular filling pressure, stroke volume and subsequently mean arterial blood pressure (MAP) (Crandall et al. 2008). Mean arterial pressure can be maintained by increasing heart rate (HR) and myocardial contractility (and thus, $\dot{Q}$), increasing peripheral vascular resistance, and limiting venous pooling (Wilson et al. 2007; Crandall et al. 2008). However, sustaining a higher HR under prolonged stress increases the risk of cardiac events, particularly in the elderly (Donaldson et al. 2003). Hence, increasing peripheral vascular resistance and limiting venous pooling seemingly

offers a desirable means of maintaining MAP during hypotensive stress.

Wearing compression garments has been shown to reduce peripheral blood pooling and increase MAP following pneumatic thigh cuff deflation (Williamson et al. 1994; Sparrow et al. 1995). Furthermore, Denq et al. (1997) found that compression (at +40 mmHg) of the lower abdomen, thighs and calves attenuated tilt-induced reductions in BP. Such compression reduces venous pooling by reducing the overall venous cross-sectional area of the lower limb (thereby increasing the linear velocity of venous outflow), reducing venous distension, and by enhancing the emptying of valvular cusps (Agu et al. 1999). Upon standing, a reduction in systemic vascular resistance inhibits venous return (Wieling et al. 1996). Lower body muscle tensing has been shown to increase total peripheral resistance (TPR) and attenuate the acute transient drop in MAP, even during a short period of standing (1 min) (Krediet et al. 2007). Consequently, external compression could reduce heat- and orthostatically induced venous pooling and aid the maintenance of MAP and cerebral blood flow when standing. The purpose of this study was to determine whether cardiovascular and cerebrovascular strain incurred by passive heat and orthostatic stress might be alleviated—particularly in older adults—via commercially available, graduated compression, full length leggings. It was hypothesized that postural reductions in MAP and cerebral perfusion would be attenuated when wearing compression leggings, particularly in older adults relative to younger adults.

Methods

The current study was conducted in conjunction with a previously reported study of age-related effects of heat and orthostatic stress (Lucas et al. 2008). The effect of compression leggings was examined using two trials (i.e. placebo (Control) vs. compression (COMP)) in a pseudo-random order, each separated by at least 7 days, with the effect of heat stress examined within each trial (i.e. normothermic vs. passive heat). Thus, the current study focuses on compression-related changes associated with standing in older and younger adults with and without heat stress, whereas absolute, control values are published in Lucas et al. (2008).

Participants

Six healthy older (70 years (SD, 4)) and six younger (29 years (4)) males participated in this mixed design, cross-over study (Table 1; Lucas et al. 2008)). In COMP trials, commercially available, graduated compression, full length leggings covering the calf, thigh and lower abdomen (Skins[®], Brandex Ltd, Riverwood, NSW, Australia) were worn on the lower limbs, sized according to manufacturer's specifications. In Control trials, a placebo garment (i.e. similar in appearance but comprised of only 9% c.f., 24% elastic fibres) was worn. Thus, an attempt was made to blind participants to compression status. No participant had a history of cardiovascular, cerebrovascular or respiratory disease. None were taking cardiovascular medication, and all were non-smokers. Written informed consent was obtained before participation in this study, which was approved by the University of Otago Human Ethics Committee, and complied with the Declaration of Helsinki.

Experimental protocol

Participants arrived at the laboratory having abstained from exercise and alcohol for 24 h, and having not consumed a heavy meal or caffeinated items for 4 h. On arrival, participants voided their bladder, for estimation of hydration from urine specific gravity (Atago Hand Refractometer; Astra Zeneca Pty Ltd,

Tokyo, Japan). Participants were then instrumented while supine for at least 30 min, the final 6 min of which were recorded for steady-state baseline. Participants then stood up rapidly (<5 s) and remained in the free-standing position for 3 min. All participants returned to supine for 6 min and repeated the supine-to-stand protocol to provide duplicate samples for reproducibility (no difference was found between stand one and two for any dependant measure response; $P>0.05$). Standing was terminated and participants lay supine with legs elevated if pre-syncope symptoms presented (reported feelings of dizziness/nausea and systolic blood pressure of ≤ 60 mmHg). This protocol was completed at normal body temperature and at elevated body temperature (esophageal temperature (T_{es}) raised 0.5°C) in both experimental trials. Normothermia was assumed by maintaining dry bulb temperature at 22.0°C (SD, 2.7) during the experimental sessions, with the participant wearing a long-sleeved and legged, two-pieced, tube-lined perfusion suit. Passive heating was then achieved by circulating warm water (48°C (0.8)) through the suit until T_{es} increased to 0.5°C . This rise in T_{es} was facilitated and then maintained by wrapping participants in a reflective foil blanket and periodically resuming circulation of warm water through the suit. Sweat loss across the session was estimated from semi-nude body mass loss (± 20 g, Wedderburn DI-10, Teraoka model, Singapore).

Measurements

Esophageal temperature was measured using a sterile, disposable thermistor (Mallinckrodt 400 general purpose, Mallinckrodt Medical Inc. St Louis, USA; Factory calibration= $\pm 0.1^{\circ}\text{C}$). Skin temperature was measured using insulated surface thermistors (Skin Thermistor EUS-U-V5-V2, Grant Instruments, Cambridge, England) at six sites, with mean skin temperature (T_{sk}) subsequently calculated from area weightings (Hardy and DuBois 1937). Blood flow velocity in the right middle cerebral artery (MCAv) was measured using a 2-MHz pulsed Doppler ultrasound system (DWL Doppler, Sterling VA, USA) via search techniques described elsewhere (Aaslid et al. 1982). The Doppler probe was maintained in position, at a fixed angle, using a commercial headpiece. Beat-to-beat arterial BP was measured using finger photoplethysmography (Finometer, TNO TPD Biomedical Instrumentation, The Netherlands). This method reliably assesses dynamic changes in beat-to-beat BP

Table 1 Esophageal temperature (T_{es}) and mean skin temperature (T_{sk}) in older and younger participants during normothermic and passive heat stress ($+0.5^{\circ}\text{C}$)

	Normothermic		$+0.5^{\circ}\text{C}$	
	Younger	Older	Younger	Older
Control ($^{\circ}\text{C}$)				
T_{es}	36.4 (0.2)	36.4 (0.4)	36.9 (0.1)*	36.9 (0.4)*
T_{sk}	32.8 (0.9)	32.9 (1.1)	36.7 (0.7)*	35.5 (1.0)* **
COMP ($^{\circ}\text{C}$)				
T_{es}	36.4 (0.2)	36.5 (0.3)	36.9 (0.3)*	37.0 (0.4)*
T_{sk}	32.6 (1.1)	33.5 (1.7)	36.7 (1.2)*	35.3 (0.4)* **

Values are mean and SD

Control wearing placebo leggings, COMP wearing compression leggings

* $P<0.05$ between thermal conditions; ** $P<0.05$ between young and older adults

that correlate well with intra-arterial recordings in both older and younger adults (Imholz et al. 1998). Heart rate and stroke volume (SV) were estimated by applying the ModelFlow method (Beatscope 1.0 software; TNO TPD; Biomedical Instruments: The Netherlands) onto the pressure waveform. This method provides a reliable estimate of changes in SV during exercise (Matsukawa et al. 2004) as well as during prolonged tilt testing (Harms et al. 1999). Cardiac output was determined from the product of HR and SV. TPR and cerebral vascular resistance (CVR) were calculated from the ratio of MAP to $\dot{Q}$ and the ratio of MAP to MCAv, respectively. The partial pressure of end-tidal carbon dioxide (P_{ETCO_2}) was sampled from a mask and measured using a gas analyzer (model CD-3A CO₂ analyzer, AEI Technologies, Pittsburgh, PA). All data were acquired continuously at 200 Hz using an analogue-to-digital converter (Powerlab/16SP ML795; AD Instruments, Colorado Springs, CO, USA) interfaced with a computer, and stored for subsequent analysis using commercially available software (Chart version 5.02, AD Instruments).

Data analysis

Supine data were averaged from 2 min preceding a stand. Initial-stand analysis consisted of three data points: (1) baseline supine, taken from the interval 30 to 5 s before initiating each stand, and; (2) nadir, the maximal drop in MCAv and cardiovascular variables (MAP, systolic blood pressure (SBP), diastolic blood pressure (DBP) and HR) from supine baseline during initial stand period; and (3) maximal recovery, maximum MCAv and cardiovascular values obtained in ≤ 30 s following nadir (Wieling and Van Lieshout 1997). Steady-state orthostasis analysis used data averaged across the final minute (from 2 to 3 min) of standing, regarded as a stabilized stage of orthostasis (Wieling and Van Lieshout 1997).

Statistical analysis

Since there were no order effects for the two stands within the orthostatic challenge at each thermal state, responses were averaged for statistical analysis. All data were analysed using the SPSS statistics software (version 15.0, SPSS Inc, Chicago, IL, USA). A mixed model, repeated measures analysis consisting of one

between (age group) and three within factors (garment, heat and posture) was used to test for effects of compression leggings, age, thermal state and posture on each dependant variable. Two-tailed tests were used to isolate differences following significant ANOVA interaction outcomes ($\alpha=0.05$). Data are expressed as mean (SD).

Results

All participants completed the normothermic and passive heat conditions of both placebo (Control) and compression leggings (COMP) trials. Hydration status and loss of body mass was not different ($P>0.05$) between Control and COMP trials or between older and younger participants. Passive heating elevated T_{es} by 0.5°C (0.1) in both the Control and COMP trials, regardless of age ($P<0.001$; Table 1). During the COMP trial T_{sk} was not different ($P=0.854$) from Control T_{sk} , however, older participants' mean skin temperature was lower ($P=0.012$) than the younger participants with passive heating in both Control and COMP (Table 1).

Supine rest

When supine, compression leggings did not change cerebrovascular (MCAv and CVR), respiratory (P_{ETCO_2}) or cardiovascular (MAP, $\dot{Q}$, HR, SV and TPR) variables when normothermic or heat stressed ($P>0.05$; Table 3).

Initial stages of standing

In the initial seconds following standing, when wearing compression leggings, postural reductions in MCAv and cardiovascular variables (MAP, SBP, DBP and HR) were not different ($P>0.05$) from Control nadir or maximal recovery results in both thermal conditions (Fig. 1). The time taken for MCAv to reach nadir and maximal recovery was not different ($P>0.05$) between Control and COMP trials during normothermia and heat stress (Table 2). Conversely, MAP took 1.4 s (14%) longer ($P=0.044$) to reach nadir when wearing compression leggings in older and younger participants during normothermia, and 1.5 s (15%) longer when heated (Table 2).

Fig. 1 Changes at nadir (maximum drop) and recovery (maximal value attained in ≤ 30 s following nadir) following initial stand in middle cerebral artery blood flow velocity (*MCAv*), mean arterial pressure (*MAP*) and systolic blood pressure (*SBP*) when wearing compression leggings (*COMP*), relative to wearing placebo (*Control*) leggings, in older and younger participants during normothermic (*NT*) and heated (*HT*) conditions. $\dagger P < 0.05$ between thermal conditions; $\ddagger P < 0.05$ between young and older adults. Data are expressed as means $\pm$ SD

Table 2 Time course of changes in *MCAv* and *MAP*, when wearing compression leggings in normothermic and passive heat (+0.5°C) conditions

Condition	Time (s)	<i>MCAv</i>		<i>MAP</i>	
		Younger	Older	Younger	Older
Normothermic	Nadir	6.3 (1.8)	8.4 (2.2)	9.0 (0.5)*	13.1 (3.2)*. **
	Max recovery	13.9 (1.8)	15.5 (5.8)	18.0 (2.5)**	26.4 (7.1)**
Passive heat	Nadir	6.5 (1.8)	5.9 (3.7)	9.2 (0.8)*	13.4 (3.8)*. **
	Max recovery	15.3 (2.9)	16.9 (5.0)	20.4 (4.1)**	22.0 (3.0)**

Values are mean and SD

MCAv mean cerebral blood flow velocity, *MAP* mean arterial pressure

* $P < 0.05$ between compression leggings and placebo leggings; ** $P < 0.05$ between young and older adults

Table 3 Cerebrovascular and cardiovascular measures while supine or during steady-state orthostasis in normothermic and passive heat (+0.5°C) conditions, when wearing compression leggings

Dependant measures	Younger normothermic		Older normothermic		Younger passive heat		Older passive heat	
	Supine	Orthostasis	Supine	Orthostasis	Supine	Orthostasis	Supine	Orthostasis
Cerebrovascular								
MCAv (cm s^{-1})	53 (6)	47 (7)**	25 (7)****	20 (8)**	56 (9)	42 (16)**	24 (8)****	19 (10)**
CVR ($\text{mmHg cm}^{-1}\text{s}$)	1.4 (0.2)	1.7 (0.7)**	3.8 (1.4)****	5.0 (2.0)**	1.2 (0.2)	1.7 (0.6)**	3.7 (1.3)****	4.7 (3.1)**
Cardiovascular								
MAP (mmHg)	75 (6)	75 (12)	84 (3)****	85 (13)****	68 (3)***	63 (14)***	78 (6)****	72 (7)****
HR (b min^{-1})	57 (15)	72 (16)**	57 (9)	65 (8)**	71 (14)***	91 (17)*	61 (11)****	75 (12)****
SV (mL)	108 (25)	94 (19)**	121 (19)	94 (19)**	105 (17)	74 (22)**	110 (16)	88 (17)**
$\dot{Q}$ (L min^{-1})	6.2 (2.8)	6.8 (2.5)**	6.9 (1.3)	6.2 (1.5)**	7.5 (2.5)***	6.7 (2.6)**	6.5 (0.9)****	6.6 (1.4)**
TPR ($\text{mmHg L}^{-1}\text{min}$)	13.2 (3.6)	12.0 (4.2)*	12.5 (2.1)	14.4 (3.9)*	9.6 (2.2)***	5.6 (2.2)*	12.0 (1.5)****	7.8 (5.0)*
Respiratory								
$P_{\text{ET}}\text{CO}_2$ (mmHg)	39.3 (6.2)	36.6 (5.9)**	36.2 (4.0)	31.4 (7.3)**	38.7 (4.9)	34.1 (4.4)**	35.0 (7.6)	31.6 (7.9)**

Values are mean and SD

Supine data averaged from 2 min preceding stand, Orthostasis 2–3 min of stand, MCAv mean cerebral blood flow velocity, CVR cerebral vascular resistance, MAP mean arterial pressure, HR heart rate, SV stroke volume, $\dot{Q}$ cardiac output, TPR total peripheral resistance, $P_{\text{ET}}\text{CO}_2$ end-tidal partial pressure of carbon dioxide

* $P<0.05$ between compression leggings and placebo leggings; ** $P<0.05$ from supine; *** $P<0.05$ between thermal conditions; **** $P<0.05$ between young and older adults

Fig. 2 Compression-related changes at steady-state orthostasis, when wearing compression leggings relative to wearing placebo (*Control*) leggings, in older and younger participants during normothermic (*NT*) and heated (*HT*) conditions. Delta (Δ), difference between compression and control trials; **a** middle cerebral artery blood flow velocity (*MCAv*); **b** cardiac output ($\dot{Q}$); **c** total peripheral pressure (*TPR*); **d** mean arterial pressure (*MAP*); **e** heart rate (*HR*). § $P<0.05$ between compression leggings and placebo leggings; each *square* represents an older individual response; each *circle* represents a younger individual response

Steady-state orthostasis

During normothermic stand (2–3 min), relative to Control, wearing compression leggings increased TPR $1.9 \text{ mmHg L}^{-1}.\text{min}$ (16%) in the older participants but dropped TPR $1.2 \text{ mmHg L}^{-1}.\text{min}$ (8%) in the younger participants ($P=0.004$ compression $\times$ age group; Table 3 and Fig. 2). Compression leggings did not change ($P>0.05$) any cerebrovascular (MCAv and CVR), respiratory (P_{ETCO_2}) or other cardiovascular (MAP, $\dot{Q}$, HR, SV) variables in either age group.

During heat stress, wearing compression leggings (vs. control) was associated with a larger stand-induced drop in TPR of $\sim 4.9 \text{ mmHg L}^{-1}.\text{min}$ in both older and younger participants (39% and 47% respectively; $P<0.001$; Table 3). Cardiac output and SV were elevated more in the older participants

during the COMP versus Control trials (older vs. younger participants, respectively: $\dot{Q}$, $+0.3 \text{ L min}^{-1}$ vs. -0.4 L min^{-1} , $P=0.019$; SV, $+6 \text{ ml}$ vs. -1 ml , $P=0.056$; Table 3). Whereas, the younger participants had a compression-related drop in HR when standing (HR, 0 b min^{-1} vs. -3 b min^{-1} , $P=0.030$). However, for both the older and younger participants the compression did not change any cerebrovascular (MCAv and CVR) or respiratory (P_{ETCO_2}) variables ($P>0.05$; Table 3).

Discussion

The novel findings from this study are that: (1) wearing commercially available compression leggings delayed the maximal drop (nadir) in MAP upon

standing, regardless of age or thermal condition; (2) the wearing of compression leggings did not attenuate postural reductions in MAP or MCAv upon standing, in either age group or thermal state; and (3) under combined heat and orthostatic stress, the wearing of compression leggings was associated with lower TPR in older and younger adults, albeit not to the detriment (or benefit) of MAP and MCAv preservation. The garment used in the present study represents a commercially available graduated compression stocking, easily accessible to the general public (as opposed to hospital-grade compression stockings). Thus, such leggings were specifically chosen as they were considered more practical and relevant for ambulatory use.

Initial stand

Regardless of thermal condition, wearing compression leggings did not modify MCAv or arterial blood pressure responses to the orthostatic stress of standing upright in either the older or younger participants. The initial fall in arterial pressure, as a result of an active change in posture, is due to a combination of: (1) the acute decompression of arterial vessels in the legs, causing an instantaneous mechanical decrease in vascular resistance (Krediet et al. 2007); (2) a venous emptying-mediated increases in the local arteriovenous pressure gradient (Tschakovsky and Sheriff 2004); (3) rapid, locally mediated vasodilation effects (Corcondilas et al. 1964); and (4) a cardiopulmonary receptor-mediated systemic sympathetic withdrawal in response to sudden increases in right atrial pressure (Wieling et al. 1996). As such, it appears that the level of compression caused by the leg garments worn in the current study was not enough to counter the changes in blood pressure, vascular resistance and MCAv associated with the initial phase of standing. The recovery of SBP within 30 s of standing is an important determinant of orthostatic intolerance, irrespective of age or sex (Romero-Ortuno et al. 2010). However, orthostatic intolerance is ultimately the result of cerebral hypoperfusion, and MAP has been proven to be a critical factor in the maintenance of cerebral blood flow (Lucas et al. 2010). The current paper showed no compression effect on SBP or MAP recovery in either group. Thus, the wearing of commercially available compression leggings did not aid the recovery of MCAv following standing.

However, the time taken for MAP to reach nadir was delayed for both age groups in both thermal

conditions (normothermic and passive heat) when compression leggings were worn (Table 2). Although speculative, this finding indicates that the compression trousers exerted a passive physical resistance, which slowed venous pooling in the lower limbs upon standing. Despite this garment effect, wearing compression leggings did not attenuate the acute transient fall in MAP or SBP associated with standing. Furthermore, the time course and magnitude of change in MCAv remained unaffected by the wearing of compression leggings. These findings indicate that compression leggings did not attenuate or exacerbate postural reductions in arterial blood pressure and cerebral perfusion upon standing, regardless of age or thermal condition.

Steady-state orthostasis

Wearing compression leggings during more sustained orthostatic stress (final minute of standing in normothermia) had a contrasting effect on TPR in older and younger participants. For older participants, with standing, TPR was elevated from heated supine and control values whereas in the younger participants TPR was lowered. Given that the wearing of compression trousers reduced TPR in the younger participants but had no effect on HR or MAP, it is possible that the level of compression in the current study was high enough to cause a passive, mechanical reduction in lower limb blood volume, while being low enough to circumvent a centrally mediated vasoconstrictor (pressor) response or a locally mediated myogenic vasodilatory response. A pressor response has been observed when external pressures ranged from 40 to 200 mmHg (Williamson et al. 1994; Bell and White 2005). In those studies, it was postulated that compression of the lower limbs elevates intramuscular pressure, activating mechanosensitive receptors and thus elevating MAP (Williamson et al. 1994; Bell and White 2005). Conversely, Mayrovitz and Larsen (1997) observed an increase in leg pulsatile blood flow with external compression of 40 mmHg, ascribing this to myogenically mediated arteriolar dilatation due to lower transmural pressure. However, ageing is associated with an attenuated muscle mechanosensitivity (Sugiyama et al. 1996) and progressive endothelial vasomotor dysfunction (Ferrari et al. 2003). We therefore speculate that these age-related changes may have impeded any compression effect in the older

participants, the result being the differential TPR response observed between young and older adults during normothermia.

It is well established that the hyperthermic-induced drop in CVP, MAP and peripheral vascular resistance are further reduced during orthostasis (Wilson et al. 2002, 2007). In the current study, this hyperthermic-induced drop in MAP was evident, even when compression leggings were worn. In fact, TPR was almost halved for both age groups during heated stand when compression trousers, as compared to Control trousers, were worn. Therefore, it appears that an orthostatic and heat-induced increase in lower limb vascular conductance enabled a larger passive compression effect in both the older and younger participants. Interestingly, this was associated with a lower HR (-3 b min^{-1}) in young adults but an increased SV ($P=0.056$) in the older adults, with unchanged MAP and $\dot{Q}$. We speculate that mild external compression reduced venous pooling, such that the increased venous return caused a reflex-mediated reduction in pressor activity (i.e. withdrawal of vascular constriction more so than chronotropic or inotropic drive). This reflex effect would presumably be mediated by an increased preload, which would maintain MAP with a lesser degree of arteriolar constriction. Alternate HR and SV responses between the two age groups also seem consistent with age-related changes in cardiovascular control under heat and orthostatic stress (Minson et al. 1998, 1999).

Notably, other studies have shown that compression of the lower limbs increase (Denq et al. 1997) or does not change (Platts et al. 2009) peripheral resistance during orthostatic stress. Conversely, the current paper showed a drop in peripheral resistance. A greater level of compression (+30 to +77 mmHg) in these studies (Denq et al. 1997; Platts et al. 2009) may underlie these contrary findings. However, it was a specific purpose of the current paper to use commercially available compression leggings, accessible to the general public. Other methodological differences such as the calculation of TPR (derived from ModelFlow estimates vs. echocardiography (Platts et al. 2009) and bioimpedance (Denq et al. 1997)), study design (repeated measures study vs. non-repeat (Platts et al. 2009)) and the type of orthostatic stress (free standing vs. tilt (Denq et al. 1997; Platts et al. 2009)) could also contribute to differences in reported peripheral resistance.

Limitations

Statistical errors might underlie both null and positive findings, particularly null findings from small sample sizes. Thus, appropriate individual data are presented throughout to highlight the clear directionality of our results. Another limitation is estimation of changes in TPR and $\dot{Q}$ using the ModelFlow method (Bogert and van Lieshout 2005). The ModelFlow method assumes a population average of aortic area depending on sex, age, height and mass. Several studies have validated the ModelFlow method compared with the standard invasive techniques (thermodilution and pulse dye densitometry) and indicate that this method provides a reliable estimate of changes in cardiac output in healthy exercising humans (Matsukawa et al. 2004), patients with septic shock (Jellema et al. 1999) or during prolonged tilt testing (Harms et al. 1999). However, the validity of absolute ModelFlow estimates are uncertain during initial changes in posture or with ageing (Remmen et al. 2002). Therefore, the emphases of these data are on comparative changes because ModelFlow-derived estimates of SV and $\dot{Q}$ track group-averaged changes reliably (Leonetti et al. 2004). It should be noted that, as TPR is estimated by the ModelFlow method, it is not possible to completely differentiate the venous from the arterial contribution, and thus effects on preload versus afterload. We acknowledge that a greater level of leg compression and/or its longitudinal pressure gradient may have affected a larger response in dependant measures. With heating, T_{sk} was 1.3°C higher ($P<0.05$) in younger participants than in older participants. This was a direct result of maintaining equivalent increases in esophageal temperature in both age groups during the heated protocol. It should be noted that sustaining an equivalent esophageal temperature and mean T_{sk} between younger and older participants is inherently difficult. For example, a previous study (Shiraki et al. 1987) found maintaining mean T_{sk} between older and younger males resulted in older participants having a significant higher esophageal temperature. Thus, whilst acknowledging the possibility that small differences in T_{sk} may influence MAP (Wilson et al. 2002), we preferentially maintained esophageal temperature above mean skin temperature. Older participants in the current study were healthy, active individuals not on cardiovascular medication. Consequently, a normally ageing popula-

tion, may not be represented by the relatively disease free nature of this older cohort. While it was ensured that there were no major muscle contractions at rest or during the stand, it is acknowledged that some leg muscles are required to maintain a standing posture and that it is not possible (or physiologically relevant) to remove this action during a stand.

In conclusion, wearing commercially available compression leggings seemingly caused a passive physical resistance which, upon standing, delayed the maximal drop (nadir) in MAP, irrespective of age or thermal strain. Under combined heat and orthostatic stress, wearing compression leggings dropped TPR in both older and younger adults, though MAP and MCAv were maintained. Thus, commercially available compression trousers appear to passively reduce venous pooling in the lower limbs under conditions where peripheral venous capacitance is increased (such as heat and orthostatic stress) but do not attenuate reductions in MAP or MCAv.

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