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TITLE

CARDIOVASCULAR EFFECTS OF WARYING G-SUIT PRESSURE AND COVERAGE DURING +1 Gz POSITIVE PRESSURE BREATHING

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ORIGINAL RESEARCH

Cardiovascular Effects of Varying G-Suit Pressure and Coverage During +1 Gz Positive Pressure Breathing

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With the continued evolution of anti-G suits, used to counter the cardiovascular dysfunction arising from +1 Gz hypoxia protection positive pressure breathing (PPB), it was hypothesized that full-coverage anti-G-suits would offer equal protection while using lower inflation pressures than the traditional 4:1 ratio. Nine experienced subjects were exposed to 2 min of 70 mm Hg PPB while wearing either the COMBAT EDGE (CE) and Tactical Life Support System (TLSS) garments with the G-suit inflated to 4 × breathing pressure, and the Advanced Tactical Anti-G-Suit (ATAGS) at 4, 3, 2, and 1 × the breathing pressure. All subjects were measured with impedance cardiography (IC), and six were measured simultaneously with both IC and the Cardioscint® nuclear probe. IC-estimated stroke volume, relative left ventricular (LV) end-diastolic volume, LV ejection fraction, and peak filling rate were depressed most in the CE and ATAGS 1 conditions ($p < 0.001$). Heart rate and mean arterial blood pressure changes were highest and lowest, respectively, using the CE and ATAGS 1 garments ($p < 0.001$). There were no differences in these variables between the TLSS and ATAGS 2–4 conditions. Thus, protection against the PPB-induced fall in LV preload and cardiovascular function may still be adequately afforded by lower G-suit inflation pressures when using full-coverage anti-G suits during PPB intended for high altitude-protection.

DISTURBANCES in cardiovascular function when performing +1 Gz (emergency high altitude get-me-down) positive pressure breathing (PPB) in hypobaric chambers have been previously well documented. Elevated intrathoracic pressure leads to increased right atrial transmural pressure and profound blood pooling in dependent venous capacitance beds, resulting in compromised left ventricular (LV) stroke volume, according to the Starling Law (3,9,16).

Recently, we used both standard radionuclide blood-pool imaging angiography, and a light-weight miniaturized nuclear probe (NP) device (Cardioscint™, Oakfield Instruments Inc., Oxon, England) to study LV function in more detail during PPB (17). For subjects wearing extended-coverage G-suits, there were only small decrements in both end-diastolic and end-systolic volumes during PPB, with only minor reductions in left ventricular ejection fraction. This contrasted to the large reductions in these measures when using standard-configuration suits during PPB. In addition, when using the extended coverage garments, there was a lesser reliance upon the atrial contribution to late LV diastolic filling. These results supported previous work comparing estimated indexes of stroke volume, using impedance cardiography (1,16).

In this and other studies demonstrating the superior protection available with increased coverage G-suits, the traditional G-suit pressurization schedule had been set at between 3.2 and 4 times the breathing pressure (13,15,16; Ackles K. Personal communication). This concept originated from work conducted in the 1970's and 1980's using standard coverage (CSU-13B/P-type) G-suits. It was established that pressurizing the G-suit to multiples of the breathing pressure markedly improved cardiovascular function during PPB (1–3,20). The use of the 3.2:1 and 4:1 ratios has continued to the present day.

However, during recent DCIEM +1 Gz PPB experiments using an extended (TLSS) and a near-full-coverage prototype from the USAF (Advanced Technology Anti-G-Suit; ATAGS), subjects frequently reported significant abdominal and foot pain (Fraser, WD. Personal communication). In addition, PPB bradycardia has been frequently observed in some subjects, with significant but transient post-PPB hypotension episodes. These responses suggest that G-suit inflation might be inappropriately high when using the newer near-full coverage G-suits. For use in altitude PPB applications, the traditional 4:1 ratio may need to be re-evaluated, since these were based upon the older standard coverage CSU-13 G-suit designs.

The purpose of this study, therefore, was to test the effect of reducing (from 4 to, 3, 2, and 1× breathing pressure) G-suit pressure during +1 Gz ground-level PPB using varying-coverage G-suits. In this manner, we hoped to determine the lowest tolerable pressurization

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level which best protects aircrew against the above cardiovascular effects of PPB, while still maintaining an optimal level of aircrew comfort and mobility. This study was carried out using simultaneous impedance cardiography for estimates of stroke volume changes, and the Cardioscint™ NP, for measurement of LV performance and diastolic function.

METHODS

Nine (two female, seven male) medically-screened experienced subjects from the DCIEM civilian and military staff volunteered to participate. Six of these subjects completed the experiments with both simultaneous NP and impedance cardiography (IC), and three additional subjects performed the experiments instrumented with only IC. Prior to participation, subjects signed a consent form outlining the study protocol, which had been previously approved by the DCIEM Human Ethics Committee, and met all guidelines for work with human radioisotope labeling studies. Each of the subject's 6 PPB exposures were completed in 1 d (~5 h total time) to avoid the potential day-to-day variations in PPB tolerance, garment fitting, and to avoid multiple radioisotope dosing.

Preliminary Nuclear Cardiology Procedures

Subjects were first in-vivo labeled with 925 MBq Technetium^{99m} for standard gamma-camera resting multigated blood pool ventriculography studies prior to the use of the Cardioscint™. Three separate 5-min resting supine acquisitions at three angles were then performed. Left ventricular wall motion and global left ventricular ejection fraction were determined off-line using standard techniques (14,22) to confirm that all subjects were within normal limits of LV anatomy and function.

Afterwards, a lead disk, the same diameter as the Cardioscint™ detector, was placed over the LV with the subject in the seated position. The disk was positioned with the aid of a continuous persistence image of the heart so as to create a shadow over the entire LV, thus defining its anatomical landmark on the chest. This landmark was then drawn on the chest with a felt pen for later positioning of the Cardioscint™ probe. Subjects were allowed some light drinks and food after the procedure.

Physiological Measurements and Data Acquisition

The subjects were then instrumented for IC using a Minnesota Impedance Cardiograph (Model 304B; Instruments for Medicine Inc., Grenwich, CT) as described elsewhere (13,15,16,19). A standard CM_5 ECG electrode configuration was used to record ECG and measure heart rate, and to continuously display the ECG waveform (Tektronix Inc., Model 408, Beaverton, OR). Blood pressure was displayed, and non-invasively measured using a Finapres 2300 monitor (Ohmeda Inc., Englewood, CO). The finger cuff was held at heart-level by a sling. IC (dZ/dt; $\Omega \cdot s^{-1}$ and Zo; Ω), Finapres and ECG (for heart rate [HR]; bpm) waveforms were analyzed using customized data display and analysis software, as described elsewhere (13,15,16).

Estimated stroke volume (SV_e; $ml \cdot beat^{-1}$) was calculated using Kubicek's equation (19). Estimated cardiac output ($\dot{Q}e$ $L \cdot min^{-1}$) was calculated as $SV_e \times HR/1000$. Finapres BP waveforms were used to calculate mean arterial blood pressure (MAP; mm Hg), calculated as DBP + 0.33 [SBP-DPB]. Only those waveforms during the normal involuntary 5–8 s end-inspiratory apneic periods which occur during PPB were selected for the analysis, due to the marked and variable effect of respiration on blood pressure during PPB. Estimated total peripheral resistance (TPR_e; $dyne \cdot s^{-1} \cdot cm^5$) was calculated as $MAP/\dot{Q}e \times 80$.

Cardioscint™ NP Measurements

The Cardioscint™ NP system has been previously described (5,6,17). It has been validated in our laboratory for left ventricular ejection fraction (LVEF) against standard blood pool imaging techniques with reasonable accuracy (r = 0.71, SE = 0.21) (24). Others have also compared the Cardioscint's™ LVEF (r = 0.94, SE = 2.1) and peak diastolic filling measures against blood pool imaging techniques with equal or better results (5).

After the above preparation, but prior to donning the PPB Garments, the NP was positioned over the premarked chest landmark, coinciding with the center of the LV. Precise placement of the detector final caudal/distal tilt positioning, and determination of baseline background counts were verified using the Cardiocint™ positioning software/algorithm, as described in detail elsewhere (6,17). A foam donut fitted over the probe ensured that jerkin inflation did not affect comfort or probe placement. The jerkin was then carefully donned over the instrumentation, and the subject was seated in the altitude chamber. The NP was not removed between the six PPB runs to ensure its reliable positioning.

NP data acquisition for each of the six preceding control and PPB sessions were not initiated until stable resting-level LV function and heart rate were attained. During rest and PPB, NP data was automatically acquired in continuous 10-s averaged gated intervals on the IBM™ PS1 computer and software interfaced to the Cardioscint™. The automatic background mode (=74% of end-diastolic counts) was used for these studies. Immediately after PPB commenced, the automatic background level was reset to account for the potentially large and rapid changes in pulmonary blood volume with the onset of PPB. The time of this adjustment was recorded in the Cardioscint™ file. Resetting of automatic background count rate took between 10 and 20 s. Consequently, only NP data between 20 and 120 s were analyzed. The determination of peak filling rate is unaffected by background count settings, and was thus analyzed from 0–120 s of PPB.

The following data were automatically and continuously calculated in 10-s epochs: relative end-diastolic volume (EDVr; background-corrected LV counts $\cdot$ 10 ms^{-1}), relative end-systolic volume (ESVr; background-corrected LV counts $\cdot$ 10 ms^{-1}), stroke counts (SC, LV counts $\cdot$ $beat^{-1}$); left ventricular ejection fraction (LVEF, %; [EDVr—ESVr]/[EDVr—background counts] $\times$ 100); peak filling rate (pfr; $+EDV \cdot s^{-1}$).

Experimental Procedures

After instrumentation, subjects underwent six individual 2-min PPB exposures at 70 mm Hg. The six PPB

conditions performed were: a) the COMBAT EDGE (CE) ensemble (standard CSU 13 G-suit and thoracic jerkin) using a 4:1 G-suit to breathing/jerkin pressure ratio; b) Tactical Life Support System (TLSS) ensemble (with 40% more bladder coverage than the CE G-suit) pressurized to 4:1; c) ATAGS ensemble consisting of a near full-coverage G-suit with inflatable foot bladders at 4:1; d) ATAGS at 3:1; e) ATAGS at 2:1; and f) ATAGS at 1:1. The same thoracic jerkin was used in the CE and ATAGS ensembles, and was similar in shape and coverage to the integrated jerkin in the TLSS garment. Subjects wore a standard flight suit beneath the CE and ATAGS garments. The one-piece TLSS garment was worn over undergarments only, with the addition of a light T-shirt for the two female subjects.

Each set of control PPB exposure was preceded by a minimum of 2 min of quiet rest, and was not commenced until LV function and heart rate had normalized to pretesting levels (as monitored in real-time using heart rate and left ventricular ejection fraction measures from the Cardioscint™). These six PPB levels were performed in two counterbalanced segments to avoid lengthy and experimentally confounding donning and doffing procedures. The CE and the TLSS runs were grouped together and counterbalanced to occur either before or after the ATAGS runs. Within the four ATAGS conditions, the four G-suit/breathing pressure ratio conditions were randomly assigned. Thus, subjects only changed between the CE and TLSS garments and remained in the ATAGS garments, eliminating garment fitting variations within the ATAGS conditions.

Ground-level (normoxic room air) PPB was conducted in the DCIEM altitude chamber using the "through the wall" technique described in detail elsewhere (16); a pressure differential of 70 mm Hg (±1 mm Hg) is created between the ground and altitude chamber by ascending to ~2500 ft. When a solonoid valve is opened, a flow of pressurized room air is allowed to pass across a hole in the chamber wall, and into the oronasal mask and jerkin. The control rest, PPB and post-PPB rest were performed after the chamber was ascended. During the four ATAGS PPB conditions, the chamber was maintained at the designated chamber altitude. G-suit pressure was supplied by a separate G-valve (Fairchild Model 10, Winston-Salem, NC), and was manually operated by an inside tender for delivery of the 1:1 (70 mm Hg), 2:1 (140 mm Hg), 3:1 (210 mm Hg), and 4:1 (280 mm Hg) during the ATAGS conditions, using a calibrated Validyne™ (Northridge, CA) digital pressure monitor (accuracy of G-suit pressurization is ±2 mm Hg). The chamber temperature was maintained at 22°C.

All PPB experiments were supervised by a qualified flight surgeon. PPB could be terminated voluntarily by the subject by activation of push-button signals, or by the flight surgeon if symptoms of pre-syncope or hypotension (>20% decrease in blood pressure during PPB) occurred. Subjects were continuously monitored by video camera, and communicated with the experimenters by microphone, and during PPB by push-button signals as described elsewhere (15,16).

Statistical Analysis

HR, SVe, Zo, and MAP data were averaged over five 10-s epochs, occurring at 0–10 s, 20–30 s, 50–60 s, 80–90 s, and 110–120 s of PPB. These means were subtracted from the mean of the first 0–90 s of the preceding 2-min control period, and were analyzed using a two-within (garment/G-suit pressure condition and time) and zero-between factors repeated-measures analysis of variance model running on commercially available software (SuperAnova, Adobe Concepts Inc., Berkeley, CA). Analysis was also performed on the absolute SVe, $\dot{Q}_e$ and TPR_e values for the mean of 0–90s of the control period, vs. the mean of 120 s of PPB using a zero-between and two-within (garment/G-suit pressure and condition [control vs. mean PPB]).

The change in the NP LV function parameters were measured by subtracting the averaged values during PPB (starting from the point where resetting of background counts was completed to the end of PPB) from the mean of the first 0–90 s of the preceding 2-min control period. The differences of these means were analyzed using a one-within (garment) and zero-between subjects factors analysis of variance model using commercially available software (SuperAnova, Adobe Concepts Inc., Berkeley, CA). Peak filling rate data for mean control, initial (0–10 s of PPB), and the mean of the remaining 20–120 s of PPB were analyzed using a two-within (garment/G-suit pressure condition and time) and zero-between factors repeated-measures analysis of variance model.

For all analyses, differences between means were assessed using pre-planned orthogonal contrasts, with the hypothesis made that (1) CE and ATAGS 1 = TLSS and ATAGS 2–4; (2) ATAGS 2 = TLSS, ATAGS 3–4. To compare the association between group mean difference data for NP, MAP, and IC parameters, Pearson product correlation coefficients were also analyzed. The significance

TABLE Ia. MEAN DIFFERENCES IN HEART RATE AND MEAN ARTERIAL PRESSURE (MAP) DURING 120 s OF PPB FROM THE MEAN OF THE CONTROL PERIOD COMPARING THE CE, TLSS, AND ATAGS GARMENTS.

	PPB Garment/G-suit Pressure Ratio (mm Hg)					
Variable	CE 4 (280)	TLSS 4 (280)	ATAGS 1 (70)	ATAGS 2 (140)	ATAGS 3 (210)	ATAGS 4 (280)
ΔHeart Rate (bpm)	18‡	−1*	9	0*	−7*†	−9*†
	2.6	5.5	3.3	5.6	5.0	5.5
ΔMAP (mmHg)	52.5	64.3*	45.6	56.8*	62.3*	63.9*
	2.3	2.5	2.1	2.4	1.9	1.9

* Significantly different from CE 4 and ATAGS 1; † significantly lower HR vs. ATAGS 1, CE 4; ‡ significantly different from TLSS 4 and ATAGS 1-4. Data are mean ± SEM.

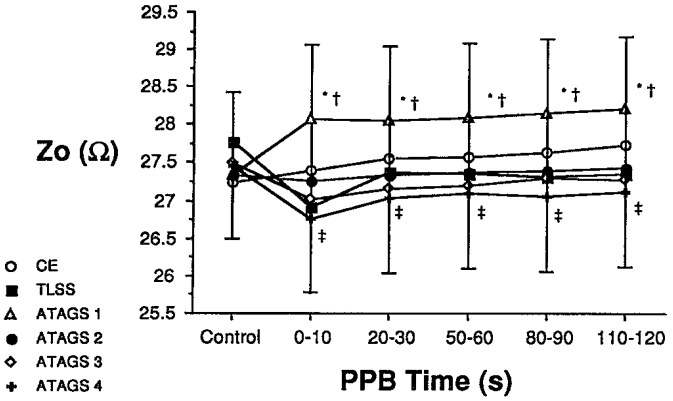

Fig. 1. Base thoracic impedance (Zo) obtained from the impedance cardiograph at control, and at five intervals during 70 mm Hg PPB for the six garment/pressure conditions. Elevated Zo indicates decreased thoracic blood volume; decreased Zo indicates increased thoracic blood volume. * significantly greater than control; † significantly different from CE 4, TLSS 4, ATAGS 2, 3 4; ‡ significantly different from control for ATAGS 3 and 4 conditions. Data are expressed as Mean ± SEM.

level adopted for all main and interaction effects was $p < 0.05$.

RESULTS

Subjects completed all G-suit PPB conditions without pre-syncopal symptoms. An average of 5 min rest was required after the CE and ATAGS 1 runs before baseline (using LVEF and HR criteria) was achieved. This duration was reduced to the minimum 2 min after the TLSS and ATAGS 2–4 runs. The changes in heart rate (ΔHR), blood pressure (ΔMAP) are presented in **Table Ia.** HR was significantly elevated using the CE and ATAGS 1 garments ($p < 0.001$). There were significant reductions in HR during PPB for the ATAGS 3 and ATAGS 4 conditions, which were significantly different from the CE 4 and ATAGS 1 conditions ($p < 0.05$). There were no differences in HR response for ATAGS 2–4 vs. TLSS 4.

There was a main effect of time on MAP, collapsed across all garment conditions ($p < 0.0001$). MAP was lowest during the first 10 s, and progressively increasing during PPB, but with no differences in the response between garments over time. There was a significant garment effect ($p < 0.0001$), with a significantly lower mean MAP difference during PPB during the CE and ATAGS 1 conditions, vs. ATAGS 2–4 and TLSS conditions ($p < 0.05$).

Impedance-Cardiography

The responses of thoracic base impedance (Z_o) during the control and 10 s PPB periods are presented in **Fig. 1.** There was a significant main effect of garment ($p < 0.0001$), and a garment × PPB time interaction ($p < 0.02$). Z_o was significantly elevated above baseline conditions for the ATAGS 1 conditions, but was significantly reduced in the ATAGS 3 and 4 conditions during PPB. There were no changes for the CE 4 TLSS 4 and ATAGS 2 conditions.

Absolute values during the control and mean of 120 s of PPB for SV_e, $\dot{Q}_e$, and TPR_e are presented in **Table Ib.** SV_e was significantly depressed from the control period for the CE 4 and ATAGS 1 conditions only, with a significant garment × condition interaction ($p < 0.0001$). There were significant elevations in mean SV_e over the entire PPB period for the TLSS 4, ATAGS 3 and 4 conditions ($p < 0.05$). **Fig. 2** illustrates changes in SV_e over 120 s of PPB. There was a significant main time effect ($p < 0.002$), but no differences between garments across time. In the TLSS 4 and ATAGS 2, 3, and 4 conditions, SV_e's were maintained or increased above baseline levels, were not significantly different from each other. Only the CE4 and ATAGS 1 condition produced significant decreases in SV_e throughout PPB ($p < 0.0001$). There were no significant changes in $\dot{Q}_e$ during PPB with respect to the control period. TPR_e was elevated during PPB ($p < 0.0001$), but there were no differences between the garment conditions (Table Ib).

Nuclear Probe Ventriculography

Left ventricular function (NP) results are presented in Fig. 3–5. For the CE 4 and ATAGS 1 conditions, LVEF (**Fig. 3**) and EDVr (**Fig. 4**) were significantly depressed below baseline conditions during PPB ($p < 0.001$). There were only minor non-significant changes in these measures for the TLSS 4, and ATAGS 2, 3, and 4 conditions. For peak filling rate (**Fig. 5**), there were significant main and interaction effects, with significant transient elevations within the first 10 s of PPB for the TLSS 4 and ATAGS 3 and 4 conditions ($p < 0.02$). Between 20–120 s of PPB however, pfr was not significantly changed from control conditions for these garments, whereas for the CE 4 and ATAGS 4 conditions, pfr was significantly depressed ($p < 0.004$).

There was a significant main effect of garment on

TABLE Ib. MEAN VALUES FOR IMPEDANCE CARDIOGRAPHY-ESTIMATED STROKE VOLUME (SV_e), CARDIAC OUTPUT ($\dot{Q}_e$) AND TOTAL PERIPHAL RESISTANCE (TPR_e) DURING THE MEAN OF 0–90 s OF CONTROL, AND 120 s OF PPB FOR EACH GARMENT CONDITION.

	PPB Garment/G-suit Pressure Ratio (mm Hg)						
Variable	Control (0–90s)	CE 4 (280)	TLSS 4 (280)	ATAGS 1 (70)	ATAGS 2 (140)	ATAGS 3 (210)	ATAGS 4 (280)
SVe (ml)	72.5	59.8*†	80.5‡	62.2*†	79.8	83.5‡	84.2‡
	4.03	4.37	6.14	3.88	6.72	6.95	5.19
$\dot{Q}e$ ($L \cdot min^{-1}$)	5.54	5.07	5.87	5.42	5.95	5.78	5.57
	0.16	0.11	0.20	0.23	0.21	0.19	0.14
TPRe ($dyne \cdot s^{-1} \cdot cm^5$)	14.8	24.33‡	23.78‡	23.36‡	21.98‡	23.17‡	24.42‡
	0.37	0.99	2.11	1.88	1.75	1.88	1.79

* Significantly less than control; † significantly different from TLSS 4, ATAGS 2–4; ‡ significantly greater than control. Data are mean ± SEM.

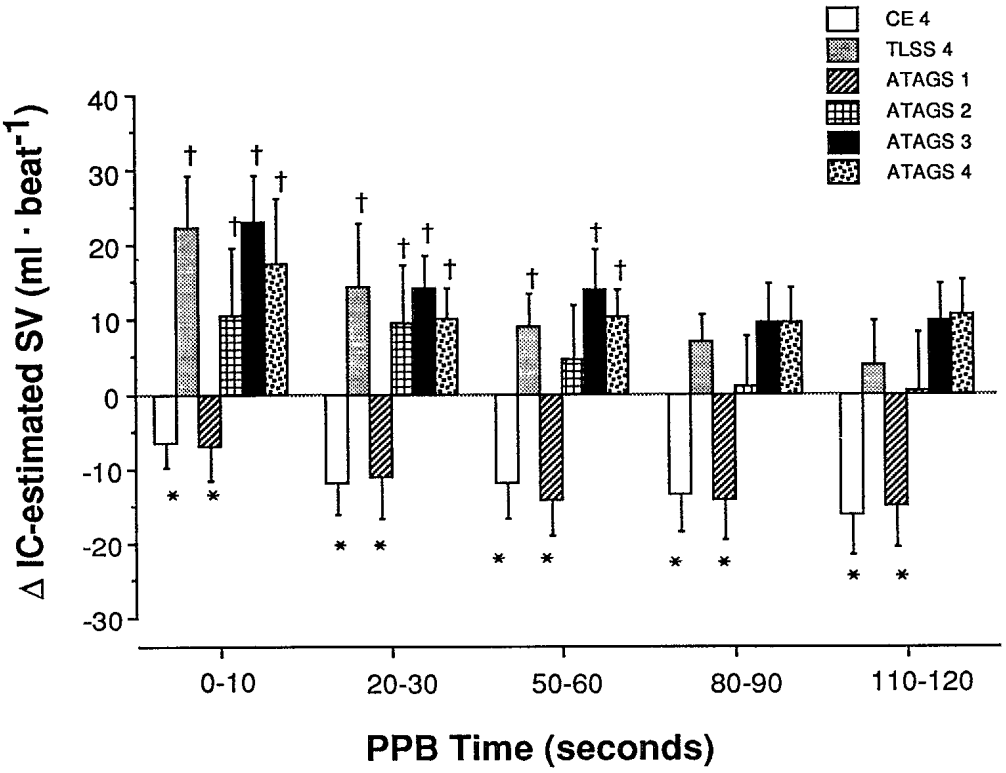

Fig. 2. Changes in impedance cardiography (IC)-estimated stroke volume (SV) at 0–10 s, 20–30 s, 50–60 s, 80–90 s, and 110–120 s of 70 mm Hg positive pressure breathing, from the mean of 0–90 s. of the control period for CE, TLSS, and ATAGS G-suit conditions (n = 9).* $P < 0.001$ vs. control, and $p < 0.05$ vs. TLSS 4, ATAGS 2, 3, 4; † $p < 0.001$ vs. control. Between 5–6 individual stable-baseline IC dz/Dt waveforms were analyzed and averaged over these individual 10-s time windows, and are expressed as Mean ± SEM.

stroke counts ($p < 0.0005$), with significant reductions during the CE 4 and ATAGS 1 conditions with respect to the control period. There was also a linear relationship between the changes in mean NP-based stroke counts and changes in IC-determined SV (SV_e) during PPB across the 6 garment conditions ($y = -7.935 + 0.302\times$ r = .98; **Fig. 6**).

DISCUSSION

The cardiocirculatory responses during PPB while using standard (i.e., CE) and extended (i.e., TLSS) garments agree with findings presented in earlier work (1,16,17). Using the CE G-suit, there were large and progressive declines in SV_e, with corresponding increases in HR at 70 mm Hg PPB. The responses with TLSS however, were opposite in direction, with no change in HR, and preservation, or even slight increases in SV_e. The increased G-suit coverage afforded by the TLSS garment also preserved LV diastolic volume and filling rate, as illustrated in this and previous work (17).

With the addition of the ATAGS G-suit in the present

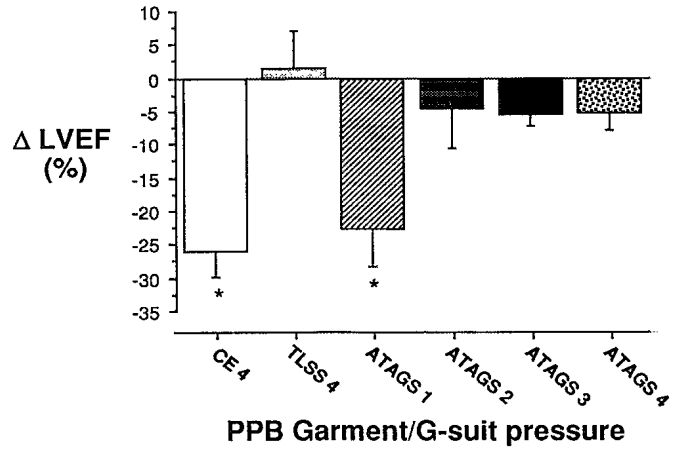

Fig. 3. Mean changes in left ventricular ejection fraction (ΔLVEF) from the Cardioscint® nuclear probe, averaged over 120 s PPB, from the mean of 0–90 s of the control period for CE 4, TLSS 4, and ATAGS 1–4 G-suit conditions (n = 6). * $p < 0.001$ vs. control, and $p < 0.05$ vs. TLSS 4, ATAGS 2, 3, 4. Data are expressed as Mean ± SEM.

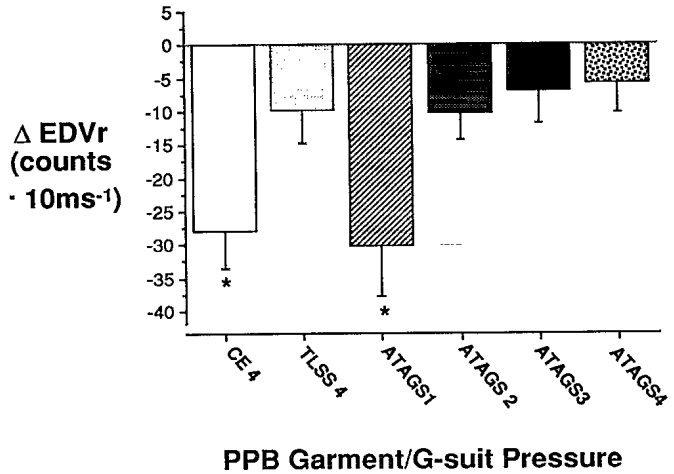

Fig. 4. Mean changes in relative left ventricular end-diastolic volume (ΔEDVr) from the Cardioscint® nuclear probe, averaged over 120 s PPB, from the mean of 0–90 s of the control period for CE 4, TLSS 4, and ATAGS 1–4 G-suit conditions (n = 6). * $p < 0.001$ vs. control, and $p < 0.05$ vs. TLSS 4, ATAGS 2, 3, 4. Data are expressed as Mean ± SEM.

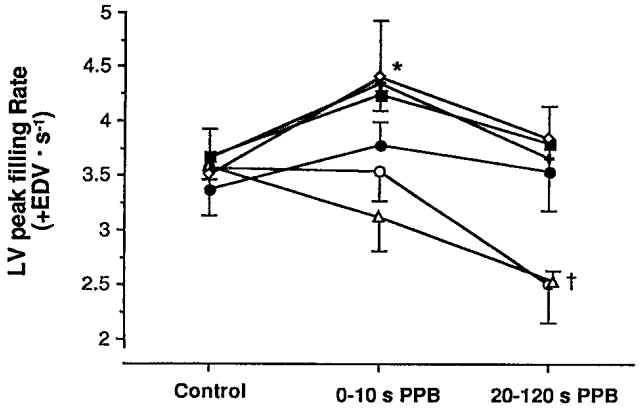

Fig. 5. Mean left ventricular peak filling rate from the Cardioscint® nuclear probe, from the average of 0–90 s of control, the first 10 s of 70 mm Hg PPB, and the average of the remainder of the PPB exposure (20–120 s) (n = 6). * Significantly elevated from the control period for TLSS 4, ATAGS 3, 4; † statistically depressed from the control condition for ATAGS 3 and 4. Data are expressed as Mean ± SEM. For legend, see Fig. 1.

study, G-suit coverage is further increased to almost the entire lower extremities by circumferential bladder coverage, and the addition of pressurized foot socks. We had predicted that this would add another incremental level

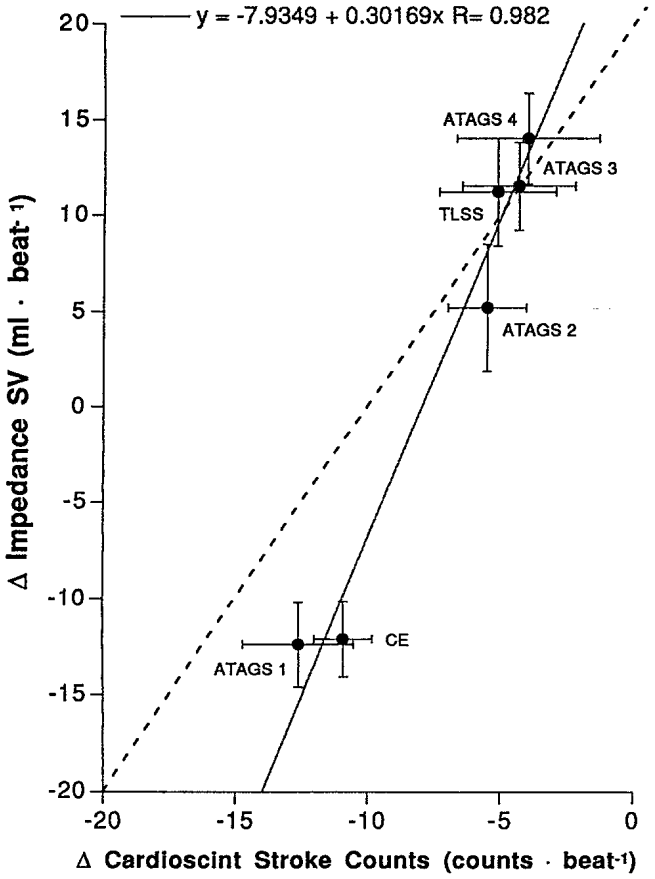

Fig. 6. Plot of mean values for impedance cardiography stroke volume (SV) vs. mean values for Cardioscint® nuclear probe stroke counts measured simultaneously in six subjects during 70 mm Hg PPB for the six garment/pressure conditions. y = −7.935 + 0.032x r = .98 ($p < 0.001$). Data are expressed as Mean ± SEM.

of central blood volume preservation during PPB when maintaining the 4:1 pressure schedule. However, as the data clearly indicate, the ATAGS suit, pressurized to the 4:1 ratio, provided no appreciable physiological benefit when compared to the TLSS G-suit at 4:1, or ATAGS pressurized at 3 or 2 times the breathing pressure.

Although all subjects completed the 2-min ATAGS 1 PPB runs without presyncopal incidents, this level of G-suit pressurization was clearly insufficient to reverse the declining cardiocirculatory function. This is evident by the significantly reduced MAP and elevated HR responses during PPB (Table I), which were similar in magnitude to the responses observed in the CE 4 condition. Further, SV_e, LVEF, EDVr and pfr were also depressed, indicating that even with near-full G-suit coverage, the use of a 1:1 G-suit pressure ratio was ineffective in preventing venous pooling-induced reduction of effective central blood and, consequently, LV volume.

With the ATAGS garment inflated to 2 times the breathing pressure however, MAP, HR, SV_e, and indices of LV diastolic volume and filling measured by the NP, improved markedly. With the ATAGS 1 condition, only the increase in MAP was significantly smaller than during the ATAGS 3 and 4 conditions (Table I), with no significant differences in SV_e, LVEF, EDVr and pfr. Lassvik, et al. (21) reported reductions in SV when the ratio was lower than 1.5:1, and that a 50% drop in G-suit pressure during +1 G_z PPB resulted in no corresponding reduction in blood pressure. In contrast, a 50% reduction in full-coverage G-suit pressure in our study (with ATAGS 2) did in fact result in a statistically significant reduction in MAP change, with the reduced hypertensive response being less than CE pressurized to 4 times the breathing pressure.

Theoretically, in order to preserve venous return, G-suit pressure must exceed the PPB-induced increase in systolic arterial pressure [which has added to it about 80%, or ~56 mm Hg, of the PPB pressure—the actually transmitted pressure to the heart and circulation (3,10,16)]. With the G-suit inflated to 1 times the breathing pressure, only 70 mm Hg of counterpressure is applied to the splanchnic area and lower extremities. Since systolic blood pressure would be 130 + 56 = 186 mm Hg, blood would still be allowed to enter the splanchnic and limb venous reservoirs. It is reasonable to assume that similar to the response in the thoracic cavity during PPB, pressure transmission to the deeper limb veins by the G-suit is also not 100% complete due to the plasticity and compliance of tissues (7,9). Presumably, the loss of pressure is compensated for by the increase in coverage, so that the response to 70 mm hg PPB using ATAGS 1 is about equal to that of the CE 4.

Accordingly, the addition of 210 (70 × 3) or 280 mm Hg (70 × 4) of G-suit pressure with full-coverage garments occludes arterial inflow to the limbs, preventing venous pooling altogether. However, there is still a rise in Z_o, and fall in SV_e, EDVr and pfr, suggesting that there may be a further loss of effective blood volume elsewhere, probably to the interstitial extravascular spaces by osmotic pressure changes and filtration (23). The fall in LVEF during PPB, significant during the CE 4 and ATAGS 1 conditions, may be due to a combination of reduced venous return due to both blood pooling and

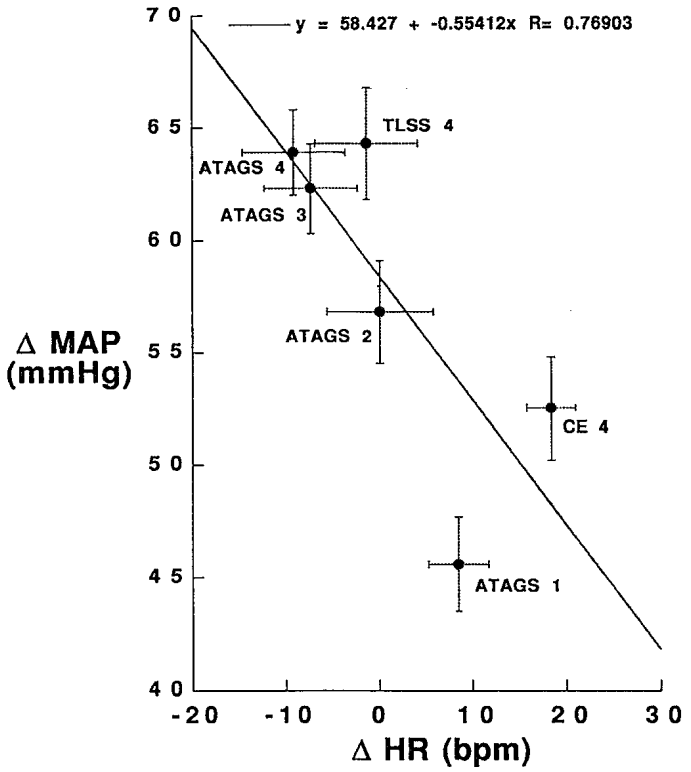

Fig. 7. Plot mean changes in mean arterial blood pressure (ΔMAP) vs. changes in heart rate (ΔHR), for all G-suit/pressure conditions during 70 mm Hg PPB (n = 6). r = 0.77, $p < 0.01$. Data are expressed as Mean ± SEM.

filtration. However, the slight and insignificant fall in LVEF with TLSS and ATAGS 2–4 would be due only to an increased afterloading of the left ventricle.

In contrast to the cardiac function and blood pressure results, there were no significant differences in estimated Q_e and TPR_e across any TLSS/ATAGS suit and pressure condition (Table Ib). Since vasomotor controls depends upon multiple inputs to the cardiovascular center from central and peripheral baroreceptors, it is probable that the TPR_e response observed in our data is a reflection of the complex interaction of competing influences. With full-coverage pressurization, as in the ATAGS 4 condition, the elevated pressures transmitted to the thoracic and carotid artery baroreceptors might elicit a vagally-mediated response, tending to provoke vasodilatation, and a net reduction in TPR_e, with a concomitant reduction in HR (8). In contrast, insufficient limb counterpressure coverage (CE 4) or pressure (ATAGS 1) fails to maintain peripheral venous return, right atrial transmural pressure, and, consequently, left ventricular stroke volume, leading to diminished MAP, which is sensed by these baroreceptors. This causes a relative baroreflex-mediated increase in sympathetic-mediated vasoconstrictor tone and, thus, TPR_e to maintain venous return, with increased HR in order to maintain cardiac output and blood pressure. These events are illustrated in **Fig. 7,** with the mean changes in MAP plotted against the mean changes in HR for each garment/pressure condition. There is a significant negative linear relationship between MAP increase and HR change, with the higher G-suit coverage/pressures registering the largest reduction in HR and increase in MAP; the lowest G-suit coverage/pressure registering increases in HR and lowest increases in MAP during PPB.

In the mid-range G-suit pressure conditions, combined with full-coverage counterpressure (TLSS 4 and ATAGS 2, 3), the vasoconstrictor response is adjusted accordingly. The net result is a relatively unchanged TPRe across the various coverage/pressure conditions.

With regard to the impedance cardiographic-estimated SV, it is noted that SV_e was actually elevated above baseline control levels for approximately half of the PPB period for the TLSS 4, and ATAGS 2–4 conditions (Fig. 2). This transient elevation in SV at the onset of PPB has been observed in prior studies using the TLSS garments (15,16). Fraser et al. (12) recently studied the responses to 20 min. of PPB using ATAGS garments, and reported reductions in HR and elevated SV for the first minute of PPB. This response agrees with our pfr data from the Cardioscint™ probe, where significant elevations in pfr were observed within the first 0–10s of PPB, followed by a decline in all garment conditions. (Fig. 5). Due to the background re-setting in the first 20 s of PPB, these elevations are not visible in the NP volumetric LVEF and EDVr data.

The effect of a transient increase in SV is magnified with the full-coverage G-suits. In the first 10–20 s of PPB, there is a net increase in LV filling arising from G-suit-induced emptying of splanchnic veins, and perhaps a transient emptying of blood from the spleen and pulmonary circulations by the elevated intrathoracic pressure. However, as venous return soon becomes disrupted by the elevated intrapulmonary pressure, coincident with a reduced right atrial transmural pressure (3), LV volumes and filling rates begin to decline after 10–20 s in the low coverage/pressure suits, and by 50–60 s in the higher pressure/coverage suits.

Using base thoracic impedance (Z_o), the measurement of relative changes in thoracic fluid content is possible. It is apparent that Z_o was significantly reduced throughout the entire PPB period with the TLSS, ATAGS 3 and 4 conditions, suggesting that thoracic congestion had occurred (Fig. 1). On the other hand, during the CE 4 and ATAGS 1 conditions, Z_o was elevated throughout PPB, indicating a relative loss of central blood volume. Although IC-measured SV declined toward the end of the PPB exposure with the ATAGS 3 and 4 conditions, the reduction in Z_o was persistent, suggesting that considerable thoracic engorgement persisted throughout the entire PPB exposure. Since NP data also does not indicate enlarged LV end-diastolic volumes and filling rates at 120 s of PPB, it is speculated that the pulmonary venous circulation is the reservoir for this extra blood volume in the ATAGS 3 and 4 conditions.

The NP analog of SV (Stroke counts) from the Cardioscint™ agrees with SV measured by impedance cardiography, with the largest declines in both SC and SV corresponding to the lowest coverage and pressure garments, and the smallest decline (or increase) in SC and SV occurring with the full coverage/high pressure garments (Fig. 6). There is some offset of this relationship, however, probably due to the differences in values produced by both methods. The Cardioscint's™ version of stroke volume is a count-based measurement, whereas imped-

ance cardiography uses thoracic impedance and blood resistivity, in addition to other presumptions, for the calculation of SV.

CONCLUSIONS

During +1 G_z (hypoxia) PPB, the use of the near-full coverage ATAGS G-suit does not afford any further significant physiological protection advantage beyond that observed with TLSS when using the traditional 4:1 G-suit pressurization ratio. In fact, G-suit overpressurization occurs, resulting in potentially negative effects, such as a slowing of HR, due possibly to stimulation of intrathoracic baroreceptors (8,11), and/or central baroreflex-mediated vagal stimulation (seen also during the transition to $-G_z$ from $+G_z$) (4). Conversely, inflating the full-coverage ATAGS G-suit to only 1 times the breathing pressure provides inadequate protection against the cardiovascular effects of PPB. We conclude that pressurizing ATAGS G-suit to multiples of 2 or 3 times the breathing pressure affords satisfactory physiological protection for this PPB application. Finally, it is important to stress that these findings apply to +1 G_z PPB uses only, and not to PPB $+G_z$ protection (PBG) applications, as recently demonstrated (18).

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