Validity of auscultatory and Penaz blood pressure measurements during profound heat stress alone and with an orthostatic challenge

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Ganio MS, Brothers RM, Lucas RAI, Hastings JL, Crandall CG. Validity of auscultatory and Penaz blood pressure measurements during profound heat stress alone and with an orthostatic challenge. Am J Physiol Regul Integr Comp Physiol 301: R1510–R1516, 2011. First published August 10, 2011; doi:10.1152/ajpregu.00247.2011.— Despite frequent reporting of blood pressure (BP) during profound passive heat stress, both with and without a hypotensive challenge, the method by which BP is measured often varies between laboratories. It is unknown whether auscultatory and finger BP measures accurately reflect intra-arterial BP during dynamic changes in cardiac output and peripheral resistance associated with the aforementioned conditions. The purpose of this investigation was to test the hypothesis that auscultatory BP measured at the brachial artery, and finger BP measured by the Penaz method, are valid measures of intra-arterial BP during a passive heat stress and a heat-stressed orthostatic challenge, via lower body negative pressure (LBNP). Absolute (specific aim 1) and the change in (specific aim 2) systolic (SBP), diastolic (DBP), and mean BPs (MBP) were compared at normothermia, after a core temperature increase of 1.47 ± 0.09°C, and during subsequent LBNP. Heat stress did not change auscultatory SBP (6 ± 11 mmHg; P = 0.16), but Penaz SBP (−22 ± 16 mmHg; P < 0.001) and intra-arterial SBP (−11 ± 13 mmHg P = 0.017) decreased. In contrast, DBP and MBP did not differ between methods throughout heat stress. Compared with BP before LBNP, the magnitude of the reduction in BP with all three methods was similar throughout LBNP (P > 0.05). In conclusion, auscultatory SBP and Penaz SBP failed to track the decrease in intra-arterial SBP that occurred during the profound heat stress, while decreases in arterial BP during an orthostatic challenge are comparable between methodologies.

lower body negative pressure; passive heat stress; arterial blood pressure

METHODS

Subjects. Seven men and three women (n = 10) participated in this study; subjects’ mean ± SD age, mass, and height were 42 ± 11 yr, 77.7 ± 10.9 kg, and 175 ± 7 cm, respectively. Subjects were nonsmokers, not taking medications, and were free of any known cardiovascular, metabolic, or neurological diseases. Subjects refrained from alcohol and exercise 24 h and caffeine 12 h before the study. Subjects began testing with a urine-specific gravity of <1.028. Written, informed consent was obtained from all subjects before participating in this study. Study procedures were approved by the Institutional Review Boards of the University of Texas Southwestern Medical Center and Texas Health Presbyterian Hospital Dallas.

Instrumentation and measurements. Each subject was dressed in a water-perfused, tube-lined suit (Med-Eng, Ottawa, Canada) that covered the entire body, except the head, face, hands, feet, and one forearm. Each subject was then placed into an LBNP chamber, sealed at the iliac crest, while in the supine position. The suit permitted the manipulation of skin and thus core temperature by changing the temperature of the water perfusing the suit. Core temperature was measured from an inguinal pill telemetry system (HQ, Palmetto, FL). The pill was ingested immediately on arrival at the laboratory, which was ~1.5 h before the onset of data collection. Mean skin temperature was measured in a manner similar to other studies (6, 8, 10).
BP measurements. BP INTRA was measured with a 20-gauge catheter inserted into the radial artery of the nondominant arm using sterile techniques after application of local anesthesia (~2 ml of lidocaine). The cannula was connected to a pressure transducer (Maxim Medical, Athens, TX) that was positioned 5 cm below the sternal notch. Calibration of the transducer was verified after the removal of each catheter by attaching a manometer to the pressure transducer and confirming calibration at 0 mmHg and at least two pressures encompassing the range exhibited by the subject.

Arterial BP was also measured by K-sound gated auscultation of the brachial artery (BP AUSC) via electrophysiology manometry (Tango™, SunTech, Raleigh, NC) integrated with the electrocardiogram (10). An appropriate sized cuff was placed directly on the skin, underneath the water-perfused suit, and the deflation rate was set to “auto” to accommodate changes in heart rate that occur with the various perturbations. Device calibration was verified before use. Due to equipment malfunction of the Tango™ during data collection for one subject, manual BP AUSC, which correlates well with automated BP AUSC (9, 21), was obtained by a trained nurse for that individual.

Beat-by-beat artery BP was measured with an appropriate sized finger cuff attached to the hand of the same arm with the arterial catheter (BP PENAZ; Finometer Pro, FMS, Amsterdam, the Netherlands). The pulse waveform was “reconstructed” by the Finometer device to estimate brachial artery BP (11). The automated physiological calibration (i.e., servo) remained on throughout testing. Calibration periods were excluded, and the 1-s delay in the analog output of the Finometer device was accounted for before data analysis.

To avoid interruptions in beat-by-beat BP INTRA and BP PENAZ, and because BP does not differ between arms in healthy individuals (7), BP AUSC was measured on the arm opposite to BP INTRA and BP PENAZ measures.

Experimental protocol. After instrumentation, subjects were supine for at least 10 min before normothermic measures, during which 34°C water was perfused. Subjects were then exposed to a passive heat stress for at least 10 min before normothermic measures. The initiation and completion of each BP AUSC were noted, and the 1-s delay in the analog output of the Finometer device was accounted for before data analysis.

To avoid interruptions in beat-by-beat BP INTRA and BP PENAZ, and because BP does not differ between arms in healthy individuals (7), BP AUSC was measured on the arm opposite to BP INTRA and BP PENAZ measures.

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Data analysis. To examine absolute BP AUSC and BP PENAZ validity (specific aim 1), values obtained from these methods were compared with BP INTRA. To examine the ability of BP AUSC and BP PENAZ to track changes in BP (specific aim 2), the change in BP (i.e., delta) from normothermia to hyperthermia was compared. Likewise, changes in BP were compared with BP PENAZ measures after core temperature increase (i.e., measures at a 1.5°C core temperature increase) to measures during LBNP were compared.


dependent of the perturbation (i.e., normothermia, heat stress, or LBNP), differences in BP INTRA vs. BP AUSC and BP PENAZ were quantified with mean bias and limits of agreement using Bland-Altman plots (3). The difference between these BP measures, with a 95% probability, will lie within the respective limits of agreement of these plots. Limits of agreement were calculated by multiplying the SD of the mean difference between paired BP methods by 2 SDs (2).

Heart rate and skin and core temperatures were sampled at a minimum of 50 Hz via a data-acquisition system (Biopac System, Santa Barbara, CA). Data were analyzed at baseline normothermia, after a 1.5°C core temperature increase, at moderate LBNP, at severe LBNP (i.e., corresponding to the last BP AUSC), and at presyncope (for only BP INTRA and BP PENAZ methods). A one-way repeated-measures ANOVA was used to examine differences in skin and core temperature responses between the aforementioned perturbations. Additionally, a one-way ANOVA was used to compare absolute BP PENAZ to BP PENAZ and to BP PENAZ at each time point (specific aim 1). A two-way (BP method × time) repeated-measures ANOVA was used to compare changes in BP INTRA to changes in BP AUSC and BP PENAZ over time (specific aim 2). Auscultatory MBP (MBP AUSC) was calculated as ½ pulse pressure + diastolic pressure. Bonferroni post hoc tests comparing BP PENAZ to BP AUSC and BP PENAZ to BP PENAZ were conducted when a significant main effect or interaction was identified from the ANOVAs. Data were analyzed using SigmaStat 3.11 (Chicago, IL) with significance set at P < 0.05 and reported as means ± SD.

Table 1. Thermal and hemodynamic data while normothermic, after an ~1.5°C core temperature increase (heat stress) and at moderate, severe, and presyncope LBNP

<table>
<thead>
<tr>
<th></th>
<th>Normothermic</th>
<th>Heat Stress</th>
<th>Moderate LBNP</th>
<th>Severe LBNP</th>
<th>Presyncope</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean skin temperature, °C</td>
<td>34.7 ± 0.5</td>
<td>39.2 ± 0.8*</td>
<td>38.8 ± 1.1*</td>
<td>39.0 ± 1.2*</td>
<td>39.0 ± 1.2*</td>
</tr>
<tr>
<td>Core temperature, °C</td>
<td>37.04 ± 0.42</td>
<td>38.51 ± 0.43*</td>
<td>38.55 ± 0.53*</td>
<td>38.73 ± 0.56*</td>
<td>38.75 ± 0.57*</td>
</tr>
<tr>
<td>Heart rate, beats/min</td>
<td>62 ± 9</td>
<td>107 ± 17*</td>
<td>117 ± 15*</td>
<td>134 ± 19†</td>
<td>137 ± 13†</td>
</tr>
</tbody>
</table>

Values are means ± SD; n = 10 for normothermic, heat stress, and moderate lower body negative pressure (LBNP); n = 6 for severe LBNP and presyncope conditions. *Significantly different from normothermic (P < 0.05). †Significantly different from heat stress (P < 0.05).
RESULTS

Core and mean skin temperatures increased with heating (both $P < 0.001$; Table 1), but there were no further increases in either of these variables during LBNP. Heart rate increased during heat stress and moderate LBNP relative to normothermia, increasing further at severe LBNP and presyncope relative to heat stress alone (all $P < 0.001$; Table 1).

For the entire protocol, the mean difference between $\text{BP}_{\text{AUSC}}$ and $\text{BP}_{\text{INTRA}} \pm$ the limits of agreement (i.e., $2 \text{ SD}$) was $6 \pm 25 \text{ mmHg}$ for SBP, $3 \pm 19 \text{ mmHg}$ for DBP, and $4 \pm 9 \text{ mmHg}$ for MBP (Fig. 1, A, C, and E). The mean difference between $\text{BP}_{\text{PENAZ}}$ and $\text{BP}_{\text{INTRA}} \pm$ the limits of agreement was $-10 \pm 22 \text{ mmHg}$ for SBP, $0 \pm 16 \text{ mmHg}$ for DBP, and $-2 \pm 16 \text{ mmHg}$ for MBP (Fig. 1, B, D, and F).

Specific aim 1: is absolute BP measured with $\text{BP}_{\text{AUSC}}$ and $\text{BP}_{\text{PENAZ}}$ during heat-stress with and without an orthostatic challenge valid?

Normothermic Penaz SBP ($\text{SBP}_{\text{PENAZ}}$), DBP ($\text{DBP}_{\text{PENAZ}}$), and MBP ($\text{MBP}_{\text{PENAZ}}$) and $\text{BP}_{\text{AUSC}}$ were not different from $\text{BP}_{\text{INTRA}}$ ($P > 0.05$); see Table 2. During heat stress, auscultatory SBP ($\text{SBP}_{\text{AUSC}}$) was greater than ($P = 0.042$) and $\text{SBP}_{\text{PENAZ}}$ was less than ($P = 0.008$) intra-arterial SBP ($\text{SBP}_{\text{INTRA}}$). There were no differences in

![Fig. 1. Bland-Altman plot comparing systolic (SBP; A and B), diastolic (DBP; C and D), and mean arterial pressures (MBP; E and F) in intra-arterial blood pressure ($\text{BP}_{\text{INTRA}}$) to auscultatory ($\text{BP}_{\text{AUSC}}$; A, C, E) and Penaz BPs ($\text{BP}_{\text{PENAZ}}$; B, D, F). Bold horizontal line indicates mean difference between methods; dashed horizontal lines are the limits of agreement (i.e., $\pm 2 \text{ SD}$ of the mean difference). ◆ BP during normothermia ($n = 10$); ● BP during heat stress [before lower body negative pressure (LBNP); $n = 10$]; △ BP during moderate LBNP ($n = 10$); □ BP during severe LBNP ($n = 6$); ■ BP at presyncope ($n = 6$).](http://ajpregu.physiology.org/ Downloaded from)
DBP or MBP between methods \((P > 0.05)\) while heat stressed.

\(\text{SBP}_{\text{AUSC}}\) was greater than \(\text{SBP}_{\text{INTRA}}\) at moderate \((P = 0.008)\) and severe \((P = 0.035)\) LBNP, while \(\text{SBP}_{\text{PENAZ}}\) was less than \(\text{SBP}_{\text{INTRA}}\) at moderate \((P < 0.001)\) LBNP. \(\text{SBP}_{\text{PENAZ}}\) and \(\text{SBP}_{\text{INTRA}}\) did not differ at presyncope \((P = 0.123)\). Auscultatory DBP \((\text{DBP}_{\text{AUSC}})\), \(\text{MBP}_{\text{AUSC}}\), and \(\text{BP}_{\text{PENAZ}}\) did not differ from \(\text{BP}_{\text{INTRA}}\) during any point during LBNP \((P > 0.05)\); see Table 2).

**Specific aim 2:** do \(\text{BP}_{\text{AUSC}}\) and \(\text{BP}_{\text{PENAZ}}\) track the change in \(\text{BP}_{\text{INTRA}}\) that occurs with heat stress and a subsequent orthostatic challenge? The magnitude of the decrease in SBP from normothermia to heat stress was greater for \(\text{SBP}_{\text{PENAZ}}\) than for \(\text{SBP}_{\text{INTRA}}\) \((P = 0.002)\) (Fig. 2). Because \(\text{SBP}_{\text{INTRA}}\) decreased with heat stress and \(\text{SBP}_{\text{AUSC}}\) did not \((P = 0.156)\), the change in SBP with heat stress differed between these methods \((P < 0.001)\). The magnitude of the decrease in SBP and MBP with heat stress was similar between measurement methods \((P > 0.05)\) (Fig. 2).

The magnitude of the reduction in SBP, DBP, and MBP throughout LBNP was not different between BP measurement methodologies \((P < 0.05\) for all; Fig. 3).

### DISCUSSION

The purpose of this study was to examine differences in arterial BP responses between three methods during a profound passive heat stress alone and in combination with a supine hypotensive challenge via LBNP. The main findings from this study are as follows: 1) during heat stress, absolute \(\text{SBP}_{\text{AUSC}}\) is higher and \(\text{SBP}_{\text{PENAZ}}\) is lower than \(\text{SBP}_{\text{INTRA}}\) and the changes in \(\text{SBP}_{\text{AUSC}}\) and \(\text{SBP}_{\text{PENAZ}}\) from normothermia to heat stress are different from \(\text{SBP}_{\text{INTRA}}\) (Table 2 and Fig. 2); 2) there were no differences in MBP or DBP between methodologies when heat stressed; 3) during subsequent LBNP, the reductions in systolic, diastolic, and mean \(\text{BP}_{\text{AUSC}}\) and \(\text{BP}_{\text{PENAZ}}\) validly tracked the reduction in \(\text{BP}_{\text{INTRA}}\) (Fig. 3); and 4) there is a large degree of variability in \(\text{BP}_{\text{AUSC}}\) and \(\text{BP}_{\text{PENAZ}}\) readings compared with \(\text{BP}_{\text{INTRA}}\) (Fig. 1).

\(\text{SBP}\) did not differ between BP methods in normothermic conditions, but hyperthermic \(\text{SBP}_{\text{AUSC}}\) and \(\text{SBP}_{\text{PENAZ}}\) failed to track the decrease in \(\text{BP}_{\text{INTRA}}\) that occurred. This resulted in absolute \(\text{SBP}_{\text{AUSC}}\) overestimating and \(\text{SBP}_{\text{PENAZ}}\) underestimating actual \(\text{SBP}_{\text{INTRA}}\). The source of this error is not known. Although speculative, one possibility is that the large increase in limb blood flow during the heat stress \((5)\) may affect the identification of the Korotkoff sounds by the microphone for the \(\text{BP}_{\text{AUSC}}\) technique, resulting in earlier identification of the initial Korotkoff sound. With the Penaz approach, it is possible that large increases in skin blood flow and finger volume, which occurs with whole body heating, alters the capacity of that approach to validly measure \(\text{SBP}\). Despite errors in \(\text{SBP}_{\text{AUSC}}\) and \(\text{SBP}_{\text{PENAZ}}\) measures during heat stress, slight differences in DBPs, although not significant, resulted in no overall differences in MBPs between methodologies. Regardless, the MBP data should be considered within the context of the very large degree of variability between these measures by the heat stress (see discussion below).

The invalid measurement of absolute \(\text{SBP}_{\text{AUSC}}\) and \(\text{SBP}_{\text{PENAZ}}\) during heat stress persisted throughout the hypotensive challenge (Table 2). This appeared to be independent of sex (data not shown), but a formal analysis is not warranted due to the small number of women tested \((n = 3)\). It is important to emphasize that the magnitude of the reduction in SBP during LBNP was similar between methodologies (specific aim 2, Fig. 3). This latter observation suggests that differences in absolute

### Table 2. Blood pressure measurements while normothermic, after an ~1.5°C core temperature increase (heat stress) and at moderate, severe, and presyncope LBNP

<table>
<thead>
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<tbody>
<tr>
<td><strong>Systolic blood pressure</strong></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Intra-arterial</td>
<td>126 ± 12</td>
<td>115 ± 10</td>
<td>100 ± 16</td>
<td>79 ± 14</td>
<td>69 ± 18</td>
</tr>
<tr>
<td>Auscultatory</td>
<td>121 ± 12</td>
<td>127 ± 13*</td>
<td>110 ± 14*</td>
<td>89 ± 9*</td>
<td>n/a</td>
</tr>
<tr>
<td>Penaz</td>
<td>121 ± 13</td>
<td>99 ± 15*</td>
<td>86 ± 15*</td>
<td>72 ± 14</td>
<td>64 ± 17</td>
</tr>
<tr>
<td><strong>Diastolic blood pressure</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intra-arterial</td>
<td>68 ± 5</td>
<td>62 ± 4</td>
<td>57 ± 7</td>
<td>49 ± 8</td>
<td>44 ± 10</td>
</tr>
<tr>
<td>Auscultatory</td>
<td>76 ± 8</td>
<td>65 ± 11</td>
<td>61 ± 11</td>
<td>48 ± 10</td>
<td>n/a</td>
</tr>
<tr>
<td>Penaz</td>
<td>68 ± 8</td>
<td>60 ± 7</td>
<td>55 ± 6</td>
<td>51 ± 7</td>
<td>47 ± 8</td>
</tr>
<tr>
<td><strong>Mean blood pressure</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intra-arterial</td>
<td>88 ± 7</td>
<td>79 ± 6</td>
<td>71 ± 10</td>
<td>59 ± 10</td>
<td>52 ± 12</td>
</tr>
<tr>
<td>Auscultatory</td>
<td>90 ± 8</td>
<td>85 ± 9</td>
<td>77 ± 11</td>
<td>62 ± 9</td>
<td>n/a</td>
</tr>
<tr>
<td>Penaz</td>
<td>88 ± 10</td>
<td>76 ± 9</td>
<td>67 ± 9</td>
<td>59 ± 9</td>
<td>53 ± 13</td>
</tr>
</tbody>
</table>

Values are means ± SD; \(n = 10\) for normothermic, heat stress, and moderate LBNP; \(n = 6\) for severe LBNP and presyncope conditions. n/a, The time required to obtain auscultatory blood pressure prevented a presyncope measurement. *Significantly different than intra-arterial blood pressure at the corresponding time point \((P < 0.05)\).
SBP\textsubscript{PENAZ}, SBP\textsubscript{AUSC}, and SBP\textsubscript{INTRA} during a heat-stressed hypotensive challenge (Table 2) are due to the effect of heat stress alone and are not due to the hypotensive challenge per se. This is reinforced by data showing that SBP\textsubscript{PENAZ} appropriately measures SBP during a hypotensive challenge when individuals are normothermic (13, 24).

Particularly noticeable is the large degree of variability in the difference between the two noninvasive measures of BP and BP\textsubscript{INTRA} (Fig. 1). This level of variability has been previously reported in a variety of other testing situations (12–14, 17, 27, 33). Standards for the American Association for the Advancement of Medical Instrumentation criteria requires a mean difference of <5 mmHg, along with a standard deviation < 8 mmHg between measurement methods (1). According to these criteria, both auscultation and Penaz measurements of absolute BP are unacceptable during heat stress and a simulated orthostatic challenge (Fig. 1), as the standard deviation of these techniques were > 8 mmHg. This unacceptable degree of variability is consistent with the standards of the British Hypertension Society, in which at least 50% of the measurements between compared devices must have a difference ≤ 5 mmHg (23). Only 14% of SBPAUSC, 24% of DBPAUSC, and 26% of MBPAUSC measures throughout the protocol differed by ≤ 5 mmHg from the corresponding BP\textsubscript{INTRA}. Similarly 26% of SBPPENAZ, 36% of DBPPENAZ, and 36% of MBPPENAZ measures throughout the protocol differed ≤ 5 mmHg from the corresponding BP\textsubscript{INTRA}. Given this large degree of variability, the lack of mean differences between methodologies for DBP and MBP is misleading.

The Bland-Altman plots also reveal that the variability in device validity is independent of BP. As BP decreased with heat stress and the subsequent orthostatic challenge, the difference in BP\textsubscript{INTRA} and BP\textsubscript{AUSC} or BP\textsubscript{PENAZ} remained highly variable. Also, it is important to point out that the variability occurred not only between subjects, but also within a given subject. For example, the difference between MBPAUSC and intra-arterial MBP in one subject was −4 mmHg at rest, 11 mmHg when heat stressed, 1 mmHg during moderate LBNP, and −9 mmHg during severe LBNP. This emphasizes the point that applying a correction factor to either an individual or a group would not be appropriate. This large degree of variability should be considered when obtaining BP noninvasively.

Criteria for test termination during LBNP or upright tilt often includes a consistent SBP of <70–90 mmHg (6, 8, 18, 20, 31, 35, 36), as well as a 15- to 20-mmHg decrease in SBP (8, 15). Therefore, the validity of both absolute BPs and the changes in BP are important to appropriately identify presyncope and thus test termination during LBNP or similar orthostatic challenge. The present data suggest that, if using absolute SBP as the sole termination criteria, the length of an orthostatic tolerance test in heat-stressed individuals may vary, depending on the BP measurement method (Table 2). However, if the termination criteria are based on the magnitude of the decrease in SBP, the present data suggest that decreases in SBP\textsubscript{PENAZ} appropriately reflect decreases in SBP\textsubscript{INTRA} (Fig. 3).

Invasive pressures were obtained at the radial artery, whereas noninvasive pressures were either measured at the brachial artery, for BP\textsubscript{AUSC}, or reconstruction to the brachial artery from the finger, for the BP\textsubscript{PENAZ}. Due to pulse-wave amplification, SBP may be slightly greater in the radial vs. brachial artery (22), while amplification is more pronounced when the reference vessel is the aorta (19). It is important to point out that, regardless of measurement site, MBP is not affected by pulse-wave amplification (22). While subjects were normothermic, there was no evidence of pulse-wave amplification (Table 2). However, pulse-wave amplification is affected by a variety of factors, such as increased heart rate and vasodilation (4, 34). It is, therefore, possible that some of the observed differences in SBP while heat stressed were due to increases in heart rate and vasodilation affecting pulse-wave amplification. To investigate if the observed differences in SBP were due to differences between radial and brachial artery pressures profiles, BP\textsubscript{INTRA} waveforms were reconstructed to reflect brachial artery waveforms with the same technology used to reconstruct Finometer waveforms (BeatScope version 1.1, FinaPres Medical Systems, Arnhem, the Netherlands). Reconstructed SBP\textsubscript{INTRA} was 8 ± 3 mmHg lower (P < 0.001).

Fig. 3. Decreases in SBP, DBP, and MBP from BP\textsubscript{INTRA}, BP\textsubscript{AUSC}, and BP\textsubscript{PENAZ} while heat stressed due to moderate LBNP (n = 10), severe LBNP (n = 6), and presyncope LBNP (n = 6). Values are means ± SD. *Significant change from previous condition independent of measurement method (P < 0.05).
than the original SBP_{INTRA}, and this decrease was independent of perturbation (i.e., normothermia, heat stress, and LBNP; \( P = 0.096 \)). Consequently, the absolute values of reconstructed SBP_{INTRA} and SBP_{PENAZ} were similar during heat stress and at moderate LBNP (\( P = 0.35 \) and 0.22, respectively). Despite the effects of reconstructing SBP_{INTRA} on the interpretation of the findings, we chose to use non-reconstructed data for the primary analysis of BP_{INTRA}. Justification for this selection is that most laboratories that have the capacity to measure BP_{INTRA} will not have the capability to reconstruct these radial pressures. In those laboratories, decisions regarding the effects of a perturbation, such as LBNP tolerance, will be based on the non-reconstructed pressure readings. Independent of this, the large variability in MAP illustrated in Fig. 1 would remain after accounting for pulse-wave amplification, given similar MBPs across the arterial tree.

Limitations. Throughout the study, subjects were severely passively heated and in a supine position. The magnitude of increase in core temperature was chosen to be consistent with numerous studies using similar levels of heating \((6, 8, 18, 35, 36)\). During profound heat stress, such as the one employed, skin and limb blood flow responses are influenced by body position (supine vs. upright) and activity level (rest vs. exercise) \((16)\). Given this, coupled with the possibility that the inaccuracies of SBP_{AUSC} and SBP_{PENAZ} during passive heat stress were due to changes in skin and limb blood flow during heat stress, the results may be different if heat stress occurred while subjects were upright or during dynamic exercise or at different body positions. Therefore, the present findings are specific to the employed level of passive heat stress and LBNP while subjects are supine and are not necessarily representative of differing experimental conditions.

Conclusions. With the exception of SBP, both BP_{PENAZ} and BP_{AUSC} track absolute BP_{INTRA} across all perturbations. However, there is a large degree of variability between both BP_{INTRA} and BP_{PENAZ}, as well as BP_{INTRA} and BP_{AUSC}. This large degree of variability should be considered when noninvasively measuring BP during heat stress alone and in combination with an orthostatic challenge. Finally, the relative decreases in BP_{INTRA} during an orthostatic challenge while subjects are heat stressed are appropriately tracked with BP_{AUSC} and BP_{PENAZ}.

ACKNOWLEDGMENTS

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GRANTS

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DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the author(s).

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