

# Effects of whole body heating on dynamic baroreflex regulation of heart rate in humans

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**Crandall, C. G., R. Zhang, and B. D. Levine.** Effects of whole body heating on dynamic baroreflex regulation of heart rate in humans. *Am J Physiol Heart Circ Physiol* 279: H2486–H2492, 2000.—The purpose of this project was to identify whether dynamic baroreflex regulation of heart rate (HR) is altered during whole body heating. In 14 subjects, dynamic baroreflex regulation of HR was assessed using transfer function analysis. In normothermic and heat-stressed conditions, each subject breathed at a fixed rate (0.25 Hz) while beat-by-beat HR and systolic blood pressure (SBP) were obtained. Whole body heating significantly increased sublingual temperature, HR, and forearm skin blood flow. Spectral analysis of HR and SBP revealed that the heat stress significantly reduced HR and SBP variability within the high-frequency range (0.2–0.3 Hz), reduced SBP variability within the low-frequency range (0.03–0.15 Hz), and increased the ratio of low- to high-frequency HR variability (all  $P < 0.01$ ). Transfer function gain analysis showed that the heat stress reduced dynamic baroreflex regulation of HR within the high-frequency range (from  $1.04 \pm 0.06$  to  $0.54 \pm 0.6$  beats·min<sup>-1</sup>·mmHg<sup>-1</sup>;  $P < 0.001$ ) without significantly affecting the gain in the low-frequency range ( $P = 0.63$ ). These data suggest that whole body heating reduced high-frequency dynamic baroreflex regulation of HR associated with spontaneous changes in blood pressure. Reduced vagal baroreflex regulation of HR may contribute to reduced orthostatic tolerance known to occur in humans during heat stress.

baroreceptor; orthostatic intolerance; transfer function analysis; spectral analysis

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HUMANS BECOME MORE SUSCEPTIBLE to syncope during orthostasis or gravitational (+Gz) acceleration in hyperthermic conditions compared with neutral or cool environments (2, 10, 13). This occurrence is at least partially due to a redistribution of blood from the central circulation to the skin. Baroreceptors are vital in the regulation of blood pressure. Relatively little is known about the effects of elevated internal temperature on baroreflex function in humans. The central components governing thermoregulation are located in the hypothalamus (25), and electrical stimulation of the hypothalamus modifies the baroreceptor reflex (7,

21). Thus it seems feasible that whole body heating may modify baroreflex control of blood pressure in humans, and this modification could contribute to impaired orthostatic tolerance in this environment.

Relatively few studies have been performed to investigate the effects of heat stress on baroreflex function. Stauss et al. (24) showed that the gain of baroreflex control of heart rate (HR) was significantly elevated in mature hyperthermic rats compared with normothermic conditions. Gorman and Proppe (8) assessed baroreflex control of HR in normothermic and hyperthermic baboons by monitoring the change in HR to increases and decreases in blood pressure induced by occlusion of the thoracic descending aorta and inferior vena cava, respectively. They found that in hyperthermia, baroreflex control of HR was elevated during the hypertensive challenge, reduced during hypotensive challenge, and not changed when the entire baroreflex curve was analyzed. Thus whether whole body heating alters baroreflex control of HR likely depends on the animal model, the technique used to assess baroreflex function, and the direction of change in blood pressure.

Little is known regarding the effects of heat stress on baroreflex function in humans. Crandall (4) recently revealed that the maximal gain of carotid baroreflex control of HR was not significantly altered in heat-stressed humans, whereas the maximal gain of carotid baroreflex control of blood pressure was significantly reduced when the subjects were heated. In addition to carotid baroreceptors, baroreflex control of blood pressure is also regulated by aortic and cardiopulmonary baroreceptors. Thus although assessment of carotid baroreflex function during whole body heating is informative, it does not provide information regarding the effects of heat stress on integrated baroreflex control.

Integrated baroreflex function can be assessed by transfer function analysis between blood pressure and HR spectral variability (22). In essence, this method analyzes the dynamic relationship between spontaneous changes in blood pressure with corresponding changes in HR. Such a technique permits the assessment of integrated baroreflex function without the in-

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herent challenges of drugs or mechanical devices to change blood pressure. Given the scarcity of information pertaining to the effects of whole body heating on integrated baroreflex control in humans, the primary purpose of this project was to use transfer function analysis during both fixed and random breathing to test the hypothesis that heat stress alters integrated baroreflex control of HR.

## METHODS

**Subjects.** Fourteen subjects (10 men and 4 women) participated in this study. The subjects' average age was  $31 \pm 2$  yr, and all were of normal height ( $174 \pm 4$  cm), weight ( $72 \pm 5$  kg), and health. A written informed consent from each subject was obtained before participation in this institutionally approved study.

**Instrumentation.** Each subject was instrumented for measurement of sublingual temperature with a thermistor placed under the tongue and for measurement of mean skin temperature from the electrical average of six thermocouples attached to the skin. Arterial blood pressure was continuously recorded, noninvasively, from a finger (Finapres). Verification of Finapres-obtained blood pressure was confirmed in both normothermia and during heat stress via auscultation of the Korotkoff sounds during manual cuff deflation. Beat-by-beat HR was obtained from the electrocardiogram signal interfaced with a cardi tachometer (CWE, Ardmore, PA). Forearm skin blood flow was indexed by laser-Doppler flowmetry (Perimed, North Royalton, OH). Respiratory excursions were monitored from a piezoelectric respiration transducer (Pneumotrace, Morro Bay, CA). The subject was dressed in a tube-lined suit that permitted the control of skin temperature by changing the temperature of the water perfusing the suit.

**Experimental protocol.** After instrumentation, each subject underwent a familiarization period in which they practiced breathing at a rate of 0.25 Hz (fixed breathing). Seven of these subjects also practiced breathing by following a randomized signal (frequency between 0.05 and 0.5 Hz with a mean frequency of 0.25 Hz). Fixed-frequency breathing was used to concentrate the spectral input around the respiratory rate and thus increase the spectral power within this region. In contrast, random-frequency breathing was used to broaden the spectral input. Both breathing protocols were accomplished through the use of a computer program, which prompted the subject when to inhale and exhale.

After this familiarization period, the subject rested in the supine position for a minimum of 20 min, at which time baseline hemodynamic and temperature variables were obtained. The subject then performed the 6-min breathing protocol(s). For the seven subjects who performed both fixed and random breathing protocols, the breathing method first assigned was randomized. On the completion of these procedures, skin temperature was elevated by perfusing the tube-lined suit with warm water ( $46^\circ\text{C}$ ). Once sublingual temperature was elevated  $\sim 0.7\text{--}1^\circ\text{C}$ , baseline hemodynamic and temperature data were once again obtained. Each subject then repeated the breathing protocol(s), with the order once again randomly assigned for the seven subjects who performed both protocols. Skin and sublingual temperatures were then returned to preheat stress levels by perfusing the suit with cold water.

**Data collection and transfer function estimation.** All data were acquired and stored digitally on a laboratory computer. Blood pressure and temperature data were sampled at 200 Hz while beat-by-beat HR was determined from a cardiota-

chometer at a sampling rate of 1,000 Hz. Beat-by-beat systolic blood pressure (SBP) was identified via a peak detection algorithm (Biopac Systems, Santa Barbara, CA). After data collection, we linearly interpolated and resampled the beat-by-beat SBP and HR data at 4 Hz for spectral and transfer function estimation. The time series of SBP and HR were first detrended with third-order polynomial fitting and then subdivided into 512-point segments with 50% overlap. This process resulted in five segments of data over the period of 6 min of data collection. Fast Fourier transforms were implemented with each Hanning-windowed data segment and then averaged to calculate autospectrum, cross-spectrum, coherence, and transfer functions. The spectral resolution for these estimates is  $\sim 0.0078$  Hz.

Low-frequency power of HR and SBP in the range of 0.03–0.15 Hz and high-frequency power in the range of 0.20–0.30 Hz were calculated from the integration of the autospectra. The low- and high-frequency transfer function gain and coherence function indexes were estimated as mean values in the same frequency ranges. The assumption of linearity and reliability of transfer function estimation was evaluated by the coherence function between changes in SBP and HR.

**Statistics.** Paired *t*-tests (2-tailed) were performed to compare differences in responses between normothermic and whole body heating trials for both random and fixed breathing protocols. Data are expressed as means  $\pm$  SE. The level of significance was set at  $P = 0.05$ .

## RESULTS

**Temperature and hemodynamic variables.** Hemodynamic and temperature data during normothermic and heat-stressed conditions are listed in Table 1. Increasing skin temperature from  $34.2 \pm 0.1$  to  $38.5 \pm 0.2^\circ\text{C}$  via the water-perfused suit led to significant increases in oral temperature, HR, and forearm skin blood flow (all  $P < 0.001$ ). Neither SBP nor mean blood pressures were significantly affected by the heat stress; however, diastolic blood pressure tended to be reduced during whole body heating ( $P = 0.06$ ).

**Fixed-frequency breathing protocol.** Spectral power in the low-frequency range (Fig. 1) was significantly attenuated by the heat stress for SBP (from  $11.5 \pm 2.1$  to  $4.5 \pm 0.8$   $\text{mmHg}^2$ ;  $P = 0.001$ ), whereas a tendency for such a reduction was identified for HR [from  $4.9 \pm 0.8$  to  $3.2 \pm 0.3$  ( $\text{beats/min})^2$ ;  $P = 0.059$ ]. The spectral power in the high-frequency range for HR [from  $8.4 \pm 2.3$  to  $1.4 \pm 0.8$  ( $\text{beats/min})^2$ ;  $P = 0.006$ ] and SBP [from  $3.7 \pm 0.6$  to  $2.3 \pm 0.5$   $\text{mmHg}^2$ ;  $P = 0.004$ ] were both

Table 1. *Temperature and hemodynamic responses to whole body heating*

	Pre-Heat Stress	Heat Stress	<i>P</i> Value
Skin temperature, $^\circ\text{C}$	$34.2 \pm 0.1$	$38.5 \pm 0.2$	<0.001
Sublingual temperature, $^\circ\text{C}$	$36.2 \pm 0.1$	$37.1 \pm 0.1$	<0.001
Systolic blood pressure, mmHg	$123 \pm 5$	$124 \pm 5$	0.68
Diastolic blood pressure, mmHg	$68 \pm 4$	$62 \pm 4$	0.06
Mean blood pressure, mmHg	$86 \pm 4$	$83 \pm 4$	0.14
Heart rate, beats/min	$58 \pm 2$	$84 \pm 5$	<0.001
Skin blood flow, perfusion units	$9 \pm 1$	$53 \pm 5$	<0.001

Values are means  $\pm$  SE.

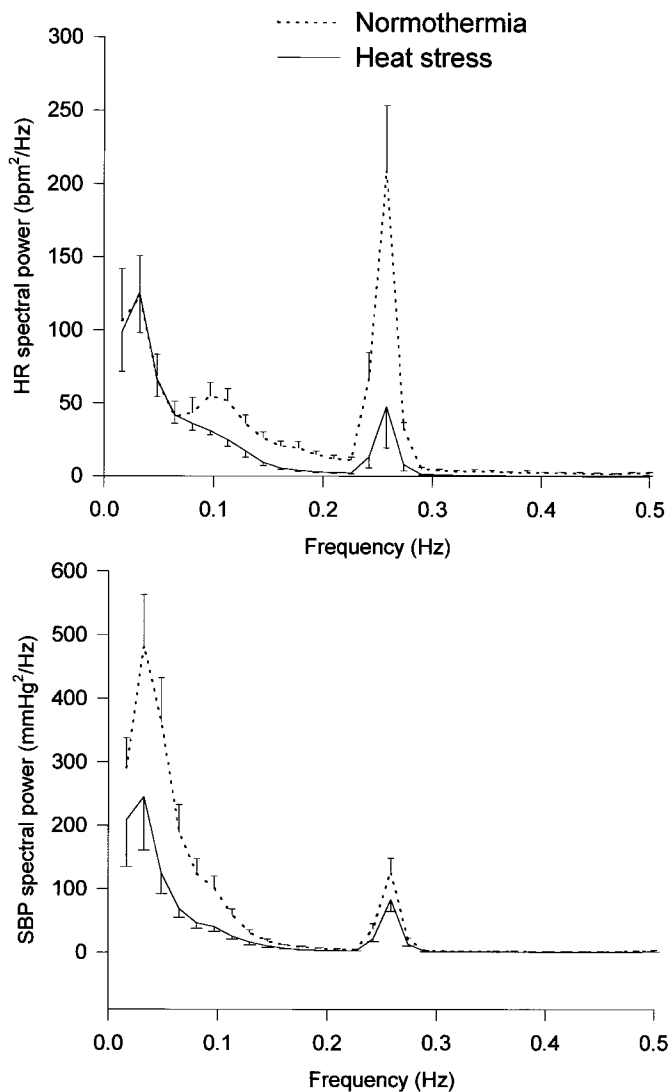


Fig. 1. Heart rate (HR; *top*) and systolic blood pressure (SBP; *bottom*) spectral power in normothermia and during whole body heating. Whole body heating significantly reduced HR variability within the high-frequency range (0.2–0.3 Hz). A reduction in HR variability within this range is indicative of reduced cardiac vagal modulation of HR. Moreover, the ratio of the spectral power between the low-frequency (0.03–0.15 Hz) relative to the high-frequency ranges was significantly elevated by the heat stress, suggesting the heat stress increased cardiac sympathetic modulation of HR. Finally, whole body heating significantly reduced the spectral power of SBP in both the low- and high-frequency regions. bpm, Beats/min.

significantly reduced by the heat stress. A reduction in HR spectral power in the high-frequency range is indicative of reduced vagal modulation of HR (1, 17, 20). Thus it is likely that the heat stress decreased cardiac vagal activity. Moreover, the ratio of spectral power of HR between the low-frequency range relative to the high-frequency range is thought to be greatly influenced by the balance between sympathetic and vagal modulation of HR (17), although this conclusion has been challenged (6). Calculation of this variable revealed that whole body heating tipped this balance toward increased sympathetic modulation of HR (from

$0.9 \pm 0.1$  to  $7.6 \pm 1.9$ ;  $P = 0.003$ ). However, the substantial increase in this ratio was attributed to a pronounced decrease in HR spectral power within the high-frequency range without an increase in spectral power within the low-frequency range. Finally, spectral power of the low- and high-frequency fluctuation in respiratory excursions was not significantly affected by the heat stress ( $P > 0.05$  for both), thereby suggesting that the heat stress did not affect respiratory variability.

The coherence and transfer function gain values for the fixed breathing protocol are listed in Table 2 and depicted in Fig. 2. Coherence between fluctuations in SBP and HR was not significantly affected by the heat stress (all  $P > 0.05$ ). The transfer function gain between the fluctuations of these variables within the high-frequency range was significantly attenuated by the heat stress (from  $1.04 \pm 0.06$  to  $0.54 \pm 0.06$  beats·min<sup>-1</sup>·mmHg<sup>-1</sup>;  $P < 0.001$ ) but was unaffected in the low-frequency range (from  $0.60 \pm 0.07$  to  $0.64 \pm 0.06$  beats·min<sup>-1</sup>·mmHg<sup>-1</sup>;  $P = 0.63$ ).

*Random-frequency breathing protocol.* Seven of fourteen subjects also performed random breathing in both thermal conditions. Coherence and transfer function gain values for this protocol are depicted in Table 3 and Fig. 2. Whole body heating tended to reduce coherence between the fluctuations in SBP and HR in the high-frequency range ( $P = 0.08$ ) but not in the low-frequency range ( $P = 0.49$ ). Similar to that observed during fixed breathing, the heat stress significantly reduced the transfer function gain in the high-frequency range (from  $1.06 \pm 0.13$  to  $0.67 \pm 0.14$  beats·min<sup>-1</sup>·mmHg<sup>-1</sup>;  $P = 0.04$ ) but not in the low-frequency range (from  $0.78 \pm 0.07$  to  $0.84 \pm 0.17$  beats·min<sup>-1</sup>·mmHg<sup>-1</sup>;  $P = 0.62$ ).

## DISCUSSION

The primary finding from this study revealed that whole body heating significantly reduced the transfer function gain between fluctuations in SBP and corresponding baroreflex-mediated fluctuations in HR in the high-frequency range (0.20–0.30 Hz) without significantly altering the relationship between the fluctuations in these variables in the low-frequency range (0.03–0.15 Hz). Reduced transfer function gain within the high-frequency range strongly suggests the heat

Table 2. *Transfer function gain and coherence values for the fixed breathing protocol in both thermal conditions*

	Pre-Heat Stress	Heat Stress	<i>P</i> Value
Transfer function gain			
LF, beats·min <sup>-1</sup> ·mmHg <sup>-1</sup>	$0.60 \pm 0.07$	$0.64 \pm 0.06$	0.63
HF, beats·min <sup>-1</sup> ·mmHg <sup>-1</sup>	$1.04 \pm 0.06$	$0.54 \pm 0.06$	<0.001
Coherence value			
LF	$0.46 \pm 0.04$	$0.43 \pm 0.04$	0.43
HF	$0.59 \pm 0.03$	$0.53 \pm 0.02$	0.08

Values are means  $\pm$  SE;  $n = 14$  subjects. LF, low-frequency range (0.03–0.15 Hz); HF, high-frequency range (0.20–0.30 Hz).

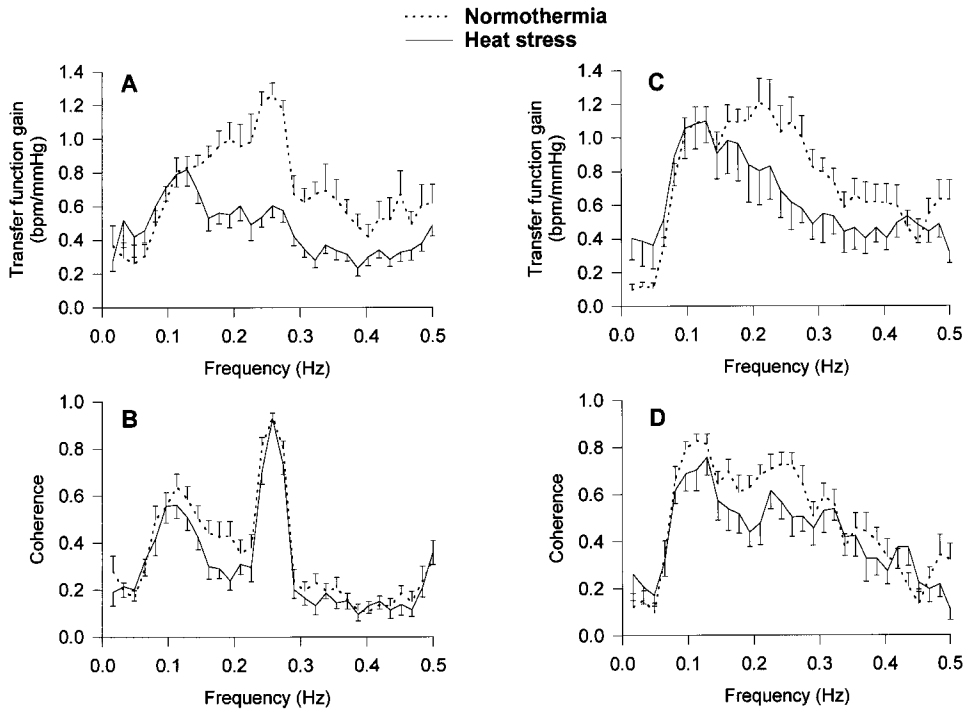


Fig. 2. Transfer function gain and coherence values during the fixed breathing (A and B;  $n = 14$  subjects) and random breathing (C and D;  $n = 7$  subjects) protocols. Whole body heating significantly reduced the transfer function gain (A and C) between variations in SBP and HR within the high-frequency range (0.2–0.3 Hz) for both breathing protocols without significantly affecting the transfer function gain between these variables in the low-frequency range (0.03–0.15 Hz). Coherence values (B and D) were not significantly affected by the heat stress regardless of the breathing protocol.

stress reduced the gain of vagal modulation of HR. These data shed new light on the effects of whole body heating on dynamic baroreflex regulation of HR by demonstrating that this perturbation has the capability of altering the control of HR. In addition, the present findings reveal that decreases in cardiac vagal activity and increases in cardiac sympathetic activity likely contribute to the elevation in HR observed during the heat stress, which confirms previous findings observed in hyperthermic baboons (9).

Exposure to hyperthermic environments reduces orthostatic tolerance (2, 10, 13). A possible mechanism resulting in this phenomenon is heat stress-induced reduction in baroreflex sensitivity. Previous studies investigating the effects of whole body heating on baroreflex function have produced mixed results. For example, Stauss et al. (24) revealed that the spontaneous baroreflex gain (sequencing technique) was significantly elevated in mature rats in hyperthermic conditions compared with normothermic conditions. In

contrast, the gain of the overall HR response to large swings in blood pressure via graded vena cava and aorta occlusions was not significantly affected in heat-stressed baboons (8). In humans, Crandall (4) and others (27) recently reported that the gain of the carotid-cardiac baroreflex was not significantly affected by whole body heating, whereas Crandall (4) found that the gain of the carotid-vasomotor baroreflex was significantly attenuated in this environment. This is in contrast to the present investigation, which suggests that integrated baroreflex control of HR within the high-frequency range is attenuated by whole body heating. Taken together, evaluation of baroreflex function during a heat stress is likely influenced by the technique used to assess baroreflex sensitivity, the baroreceptor population(s) perturbed, and/or the species studied.

The primary difference between the present investigation and those previously cited is that in the present investigation we analyzed dynamic baroreflex function in humans by assessing the relationship between spontaneous fluctuations in SBP with corresponding fluctuations in HR within fixed frequency ranges. This transfer function analysis has been developed to emphasize dynamic baroreflex control of HR as a frequency-dependent phenomenon (3, 22, 23) and allows the assessment of baroreflex gain without the use of pharmacological agents or mechanical devices. Therefore, this method of baroreflex assessment is less affected by the technique used to assess baroreflex gain. However, the method used in the present investigation identifies the gain only within the region of the sigmoidal baroreflex curve where blood pressure is spontaneously fluctuating

Table 3. Transfer function gain and coherence values for the random breathing protocol in both thermal conditions

	Pre-Heat Stress	Heat Stress	P Value
Transfer function gain			
LF, $\text{beats} \cdot \text{min}^{-1} \cdot \text{mmHg}^{-1}$	$0.78 \pm 0.07$	$0.84 \pm 0.13$	0.62
HF, $\text{beats} \cdot \text{min}^{-1} \cdot \text{mmHg}^{-1}$	$1.06 \pm 0.13$	$0.67 \pm 0.14$	0.04
Coherence value			
LF	$0.59 \pm 0.03$	$0.55 \pm 0.05$	0.49
HF	$0.67 \pm 0.04$	$0.52 \pm 0.07$	0.08

Values are means  $\pm$  SE;  $n = 7$  subjects. LF range was 0.03–0.15 Hz, and HF range was 0.20–0.30 Hz.

around an operating point. Thus the entire baroreflex curve is not assessed by this method. Without assessing the entire baroreflex curve, it remains unknown whether a change in baroreflex gain was caused by a shift in operating point to a less steep location on an unchanged baroreflex curve or whether the sigmoidal baroreflex curve itself has become flatter (i.e., less steep linear portion of the curve). Nevertheless, this technique permits the estimation of dynamic baroreflex regulation of HR caused by spontaneous changes in blood pressure within defined frequency ranges. From this analysis, the present data suggest that whole body heating reduces baroreflex buffering capacity in the high-frequency range (0.20–0.30 Hz) without altering the capacity to buffer blood pressure fluctuations within the low-frequency range (0.03–0.15 Hz).

In the present investigation, transfer function gain analysis was performed while the subjects breathed at a frequency of 0.25 Hz and, in seven subjects, during randomized breathing. Each breathing method has different strengths as it pertains to spectral and transfer function analyses of HR and SBP. The fixed breathing protocol concentrates the spectral input within the region surrounding respiration (i.e., 0.25 Hz), whereas the random breathing protocol broadens the spectral input. The fact that the results were the same regardless of the breathing protocol further supports the present interpretation of the data. That is, the heat stress reduced the transfer function gain within the high-frequency range without altering the transfer function gain in the low-frequency range. In contrast, the coherence between the fluctuations in SBP and HR was different depending on the breathing technique. It is generally accepted that the coherence function should be at least 0.50 to confidently assess baroreflex gain via transfer function analysis (22, 23). Within the low-frequency range, mean coherence values for both the normothermic and heat stress stages during the fixed breathing protocol were less than this value (0.46 and 0.43, respectively). Thus the confidence of the assessment of the transfer function gain within this frequency range could be questioned. However, the mean coherence values within the low-frequency range for the random breathing protocol were  $>0.50$ , and the transfer function gain analysis revealed that the heat stress did not significantly alter baroreflex control of HR within this range. Thus, despite the low coherence values for the data obtained during the fixed breathing protocol in the low-frequency range, we are confident that the heat stress did not significantly affect baroreflex control of HR during spontaneous fluctuation in blood pressure within this range. In contrast, coherence values for the fluctuations in SBP and HR within the high-frequency range were  $>0.50$  regardless of the breathing technique employed.

The mechanism(s) causing a reduction in the transfer function gain between fluctuations in SBP and HR within the high-frequency range remains unknown. One possibility is central inhibition of the baroreflex secondary to whole body heating. The primary neural

structures governing thermoregulation are located in the hypothalamus (25), and electrical stimulation of the hypothalamus modifies the baroreceptor reflex (7, 21). Thus it is feasible that whole body heating alters vagal baroreflex control of HR through hypothalamic modification of the baroreflex.

Another possible explanation for reduced high-frequency transfer function gain in heat pertains to the effects of the heat stress on cardiac vagal activity. Spectral power of HR within the high-frequency range, under very specific conditions, has been shown to be representative of cardiac vagal activity (1, 17, 20). In the present experiment, whole body heating decreased this index. Thus it is likely that the heat stress reduced vagal modulation of HR, and this contributed to the observed elevation in mean HR. If, during the heat stress, cardiac vagal activity is reduced to a point that it is close to being fully withdrawn (i.e., close to threshold), the magnitude of the increase in HR via vagal mechanisms may be reduced during the heat stress due to reduced reserve to further decrease cardiac vagal activity. Such a response may lead to the observed reduction in the transfer function gain between fluctuations in SBP and HR.

Whole body heating decreased spectral variability of SBP within the low-frequency range by  $\sim 60\%$  for both the fixed and random breathing protocols. Changes in spectral variability of SBP within this range are thought to reflect sympathetic modulation of vasomotor tone (12, 14, 17, 18). Thus, according to this index, sympathetic control of vasomotor tone decreased due to the heat stress. However, Crandall et al. (5) and others (16) recently identified that postganglionic sympathetic nerve activity to muscle increases substantially during heat stress in humans. Thus an indirect indicator of sympathetic modulation of the vascular suggests that whole body heating reduces sympathetic activity, whereas a direct indicator of sympathetic activity suggests the opposite. One possible explanation for this apparent discrepancy is the effects of increasing vascular capacitance within the skin on overall fluctuations in SBP. During a heat stress, skin blood flow increases substantially due in part to increases in cutaneous vascular conductance and relaxation of cutaneous veins. This response effectively increases vascular capacitance within the cutaneous circulation. Given this response, it is possible that a reduction in low-frequency fluctuations in SBP during the heat stress could occur in the face of increased sympathetic activity if the aforementioned changes in the cutaneous vasculature effectively buffered the magnitude of the fluctuations in SBP. Alternatively, it may be that there is simply less oscillations in sympathetic nerve activity during a heat stress despite a greater overall activity (5, 12) and/or that the heat stress may reduce vascular responsiveness to adrenergic neurotransmitters, as observed in hyperthermic rats (11, 15).

*Potential limitations of the interpretation of the results.* The assessment of baroreflex function in the present analysis reflects a closed-loop relationship between blood pressure and HR with the basic premise

that oscillations in blood pressure lead to baroreflex mediated oscillations in HR. However, Taylor and Eckberg (26) showed that if the R-R interval was fixed via cardiac pacing in humans in the supine position, oscillations in arterial blood pressure were reduced. They concluded that respiratory sinus arrhythmia can contribute to arterial pressure fluctuations. In the present study, we cannot exclude the possibility that oscillations in HR contributed to oscillations in arterial blood pressure in either thermal condition. However, on the basis of the observations that transfer function gain correlates significantly with other measures of baroreflex function, including the vasoactive drug method (19, 22), we consider this technique to be a useful and valid method of assessing dynamic baroreflex function, emphasizing the frequency dependence of HR and arterial blood pressure.

As previously mentioned, individuals are more prone to orthostatic intolerance or fainting during +Gz acceleration when exposed to a hyperthermic environment (2, 10, 13). It may be that reduced baroreflex responsiveness is at least partially responsible for this phenomenon. The present data suggest that baroreflex control of HR within the high-frequency range is significantly reduced by whole body heating. However, because variations in blood pressure during fixed and random breathing are relatively small compared with decreases in blood pressure during an orthostatic event, it remains unclear whether whole body heating reduces baroreflex responsiveness in humans sufficient to compromise one's ability to withstand orthostatic stress. Given this limitation, we cannot conclusively state that the reduction in orthostatic tolerance observed during a heat stress is due to reduced baroreflex gain, as indicated by a reduced transfer function gain in the high-frequency range. Nevertheless, we can state that whole body heating reduced an index of baroreflex function, and this may be indicative of the effects of heat stress on overall baroreflex regulation of blood pressure in humans.

In conclusion, whole body heating significantly reduced the transfer function gain between spontaneous variations in SBP and corresponding variations in HR in the high-frequency range without significantly affecting the transfer function gain of these variables in the low-frequency range. Reduced vagal baroreflex regulation of HR during exposure to hyperthermic environments in humans may contribute to reduced orthostatic tolerance known to occur in this environment.

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