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Cutaneous and hemodynamic responses during hot flashes in symptomatic postmenopausal women

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Abstract

Objective—The aim of this study was to test the hypothesis that the postmenopausal hot flash is accompanied by rapid decreases in arterial blood pressure and increases in cutaneous vascular conductance (CVC), as evaluated by continuous measurements of these variables in symptomatic women.

Design—Twelve healthy, normotensive, postmenopausal women rested in a temperature-controlled laboratory (26°C) for approximately 90 minutes. The onset of a hot flash was objectively identified as a transient and pronounced elevation of sternal sweat rate (capacitance hygrometry).

Results—Twenty-three hot flashes were recorded during the experimental sessions (3.4 ± 1.4 min; range, 1.3–6.5 min). Mean arterial blood pressure decreased 13 ± 2 mm Hg during 11 hot flashes in five participants. Data from these participants, categorized as responders, were analyzed separately from data for those participants whose blood pressure did not change during their hot flashes ($n = 7$, 12 hot flashes). Heart rate (obtained from an electrocardiogram) significantly increased during the hot flashes, but there was no difference between the responder and nonresponder groups (9 ± 2 vs 10 ± 1 beats/min, respectively; $P > 0.05$). The increase in CVC was not different between groups at either the forearm ($15\% \pm 3\%$ vs $12\% \pm 3\%$ maximal CVC, $P > 0.05$) or sternum ($24\% \pm 5\%$ vs $21\% \pm 3\%$ maximal CVC, $P > 0.05$).

Conclusions—These data demonstrate that in a subset of participants, the hot flash is accompanied by a significant reduction in blood pressure, but there is no difference in CVC between these women and women with no drop in blood pressure.

Keywords

Skin blood flow; Blood pressure; Hot flash

Hot flashes are a primary symptom of female menopause that can seriously disrupt the lives of symptomatic women.¹ Approximately 70% of women experience hot flashes for the first 1 to 5 years after the onset of the menopausal transition.^{1–4} The incidence and severity of symptoms are even higher in women after surgically induced menopause and in female oncology patients.^{3,5,6} Hot flashes are typically defined as sudden subjective sensations of

heat, frequently accompanied by skin flushing and perspiration, that generally begin in the chest and radiate to the face, head, and arms.^{1,4,7,8} Symptomatic women also report a range of additional symptoms during a hot flash, such as anxiety, frustration, embarrassment, nausea, and depression.^{1,8–10} Of importance, hot flashes can negatively affect mood, concentration, sleep quality, and sexual function and result in fatigue and stress,^{1,11–14} thereby significantly reducing the quality of life and overall health of afflicted women.^{2,14–16}

Despite the clear disruption that hot flashes cause to the lives of menopausal women, physiological mechanisms associated with hot flashes are not completely understood. Previous research has reported transient increases in hand, calf, and forearm blood flow during hot flashes using plethysmographic techniques.¹⁷ The plethysmographic technique, however, provides a discontinuous measure of blood flow that does not distinguish between different circulatory beds (eg, skin vs muscle). Although increases in limb blood flow during a hot flash are likely to be directed to the skin, we are unaware of studies that have assessed this question. Similarly, the effects of the hot flash on systemic hemodynamics are relatively unknown, except for modest elevations in heart rate.^{17,18} Less clear is the effect of the hot flash on blood pressure (BP), with one study reporting no change in BP during hot flashes,¹⁷ whereas a recently published case study¹⁹ reported pronounced reductions in BP during a hot flash in one patient. Differences in the findings of these studies may be related to discontinuous BP measurements in the former study, whereas BP was continuously obtained in the latter study. The aim of this study was to test the hypothesis that the postmenopausal hot flash is accompanied by decreases in BP and increases in cutaneous vascular conductance, evaluated by continuous measurements of both variables, in symptomatic women.

METHODOLOGY

Participants

Twelve postmenopausal (eight surgically and four naturally menopausal) women were randomly selected to participate in this study. A sample size smaller than 12 participants was initially chosen after it was determined that this size would provide appropriate power to identify cutaneous vascular responses to the hot flash. However, additional participants were added, given the observation that BP responses differed between subsets of women (see “Results”). The participants’ mean anthropometric data are presented in Table 1. All participants had been amenorrheic for at least 12 months and were experiencing at least four hot flashes a day, which was verified by their completing a hot flash frequency journal for 1 week before participating in the study.¹³ Participants were healthy and free from cardiovascular and metabolic diseases and were not taking hormone therapy or any other treatments to alleviate hot flash symptoms. Participants refrained from alcohol and exercise for 24 hours and caffeine for 12 hours before the study. Institutional approved written, informed consent was obtained from all participants before they enrolled in the study. All procedures were consistent with the principles of the Declaration of Helsinki.

Experimental protocol

Experiments were performed in a temperature-controlled laboratory ($26 \pm 1^\circ\text{C}$) in the morning or early afternoon at least 2 hours postprandially. Each participant dressed in a water-perfused tube-lined suit (Med-Eng, Ottawa, ON, Canada) that covered the entire body except the head, face, hands, feet, upper sternum, and one forearm. The water-perfused suit permitted control of skin temperature by changing the temperature of the water perfusing the suit. After instrumentation, participants rested in a semirecumbent position for approximately 90 minutes while 34°C water was perfused through the suit.

Instrumentation and measurements

Heart rate was obtained from an electrocardiogram (Space-Labs, Redmond, WA) interfaced with a cardiometer (CWE, Ardmore, PA). Continuous beat-by-beat arterial BP was recorded from a digit using a Finapres device (Ohmeda, Louisville, CA). Intermittent arterial BP was also measured from the brachial artery by electrophygmomanometry (SunTech, Raleigh, NC). Core temperature was measured from an ingestible pill telemetry system (HTI Technologies, Palmetto, FL). Sweat rate was continuously recorded from the dorsal forearm and sternum not covered by the water-perfused suit using capacitance hygrometry (Viasala, Woburn, MA). This procedure is performed by perfusing 100% nitrogen at a flow rate of 150 mL/min through a ventilated capsule (surface area = 2.83 cm²) attached to the skin's surface. Absolute humidity is calculated from the relative humidity and temperature of the gas leaving the chambers. Sweat rate (mg/cm² · min⁻¹) is calculated as the product of absolute humidity (mg/m³) and gas flow (m³/min) divided by the capsule surface area (cm²). At the same locations, skin blood flow was indexed using laser Doppler flowmetry measurements of skin blood flux with integrating flow probes (Perimed, North Royalton, OH). An index of cutaneous vascular conductance (CVC) was calculated from the ratio of laser Doppler flux to mean arterial BP. Forearm and sternum CVC was expressed as a percentage of maximal cutaneous vasodilation as identified either by local heating to 42°C for 30 minutes²⁰ or by intradermal microdialysis administration of 50 mmol/L sodium nitroprusside²¹ after the completion of all procedures.

Data analysis

Data were sampled at 50 Hz with a data acquisition system (Biopac Systems, Santa Barbara, CA) and analyzed using a statistical software package (SigmaStat 3.11, Systat Software, Inc, San Jose, CA). The onset of a hot flash was objectively identified as a transient and pronounced elevation of sternal sweat rate as has been used previously.^{22,23} This method corresponds very well with increases in sternal skin conductance, an electrical index of sweating, that is also used as an objective identifier of a hot flash.²²⁻²³ An increase in sternal sweat rate of 0.001 mg/cm² · minute⁻¹ per second was used as a criterion to identify a hot flash. Because of the variance in the length of hot flashes, each hot flash was divided into eight equal segments (each segment representing 12.5% of hot flash duration). Five-second periods of data at the end of each segment and every 15 seconds over a period of 2 minutes before and after the hot flash were used in the statistical analysis. Differences in the length of hot flashes and anthropometric data of participants categorized as responder and nonresponder groups (see explanation in "Results") were assessed using *t* tests. Differences in thermoregulatory and hemodynamic responses between the responder and nonresponder groups before, during, and after the hot flash periods were evaluated using a two-way mixed model analysis of variance with main effects of time (repeated variable) and group (nonrepeated variable). All values are reported as means ± SEM. *P* values less than 0.05 were considered statistically significant.

RESULTS

Twenty-three hot flashes were recorded during the experimental sessions. The average duration of the hot flash was 3.4 ± 1.4 minutes (range, 1.3–6.5 min). Analysis of individual BP responses during the hot flashes indicated that 5 of the 12 participants exhibited decreases in BP during their hot flashes. These five individuals were categorized as BP responders, and their data were compared with those for the individuals whose BP remained stable during the hot flash (ie, nonresponders). Eleven hot flashes were recorded in the BP responder group. There was no difference in the duration of hot flashes between the BP responder and nonresponder groups (3.5 ± 0.4 vs 3.4 ± 0.5 min, *P* > 0.05). There were no statistical differences in age (49 ± 5 vs 51 ± 4 y, *P* > 0.05), resting mean arterial BP (88 ± 4 vs 94 ± 11 mm Hg, *P* > 0.05), or heart rate (67 ± 7 vs 70 ± 11 beats/min, *P* > 0.05) between the BP responder and nonresponder groups,

respectively. In addition, there were no statistical differences in height (167 ± 5 vs 161 ± 5 cm, $P > 0.05$), weight (66 ± 6 vs 80 ± 22 kg, $P > 0.05$), or body mass index (24 ± 3 vs 31 ± 7 kg/m², $P > 0.05$) between the BP responder and nonresponder groups, respectively.

In the BP responder group, the peak beat-by-beat derived BP decrease was 13 ± 2 mm Hg, which occurred at 12.5% of the duration of the hot flash, whereas BP did not change during the hot flashes of the nonresponder group ($P < 0.05$) (Fig. 1A). This decrease in BP of the responder group was confirmed by electrospychmomanometric BP recordings from the brachial artery. Aside from BP, all other variables during the hot flashes were not different between the BP responder and nonresponder groups. Thus, non-BP data from these groups were combined and are presented to identify the typical response during the hot flash.

The peak increase in heart rate during the hot flashes was 10 ± 1 beats/minute ($P < 0.001$) (Fig. 1B). Core temperature before the hot flashes was not different between the BP responder and nonresponder groups (37.38 ± 0.08 vs $37.20 \pm 0.08^\circ\text{C}$, $P > 0.05$). The changes in core temperature just before, during, and after the hot flashes are presented in Figure 2. Core temperature did not change during the 2-minute period before the hot flash or up to 87.5% of the duration of the hot flashes. Thereafter, core temperature slightly decreased throughout the 2-minute post-hot flash period ($-0.09 \pm 0.02^\circ\text{C}$, $P < 0.001$).

Consistent with our objective definition of hot flashes, sternal sweat rate increased at the onset of each hot flash, peaking at 25% of the duration of the hot flash and then returning to baseline by the end of the flash ($P < 0.001$) (Fig. 3A). Similarly, forearm sweat rate increased at the onset of the hot flash, peaking at 25% to 37.5% of the duration of the hot flash ($P < 0.001$) (Fig. 3B) and then returned to baseline by the end of the hot flash. Five of the 23 hot flashes occurred during the identification of maximal cutaneous vasodilation (3 from the responder group and 2 from the nonresponder group), during which time neurally mediated changes in CVC are masked because of the imposed stimulus to evoke maximal cutaneous vasodilation. Therefore, the CVC data from these hot flashes were excluded, and subsequently forearm and sternal CVC data from 8 and 10 hot flashes from the responder and nonresponder groups, respectively, were analyzed and presented. Relative to the non-hot flash state, forearm CVC significantly increased approximately 15 seconds before and throughout the first half (0%–50%) of the hot flash ($P < 0.001$) (Fig. 4A). The peak of the increase in forearm CVC typically occurred in concert with the initial increase in sweating and was approximately twofold above the pre-hot flash baseline. Similarly, sternal CVC significantly increased from baseline approximately 30 seconds before the hot flash ($P < 0.001$) (Fig. 4B), with the peak increase occurring near the onset of sternal sweating (ie, 0% of hot flash). Peak sternal CVC also increased approximately twofold above baseline.

DISCUSSION

The aim of the present study was to test the hypothesis that the postmenopausal hot flash is accompanied by decreases in BP and increases in CVC. To achieve this aim, thermoregulatory and hemodynamic responses were continuously monitored before, during, and after spontaneous hot flashes under normothermic conditions in symptomatic women. The primary findings of this study are that transient decreases in BP occurred, in contrast to a prior observation,¹⁷ during hot flashes in a subset of participants. Second, large transient increases in trunk and forearm cutaneous blood flow occurred before the onset of sternal sweating.

Despite the clear disruption that hot flashes cause to women's lives, the physiological mechanisms of a hot flash are not completely understood, particularly with regard to changes in skin blood flow and BP. Previous studies have reported transient increases, relative to the pre-hot flash baseline, in finger, hand, and limb blood flow during hot flashes, ranging from

30% to 1,100% above pre-hot flash baselines.^{17,24–27} However, in each of these studies, blood flow was measured using the plethysmographic technique, which does not allow the identification of the vascular bed (ie, muscle or skin) responsible for increases in limb blood flow. Moreover, this technique provides a discontinuous assessment of blood flow, so transient changes in blood flow associated with a hot flash may be missed. Although these authors speculated that the observed increases in limb blood flow during a hot flash were directed to the skin, this hypothesis had not been directly examined. In the present study, using laser Doppler flowmetry, we continuously measured skin blood flow on the sternum and forearm during multiple hot flashes. On average, hot flashes resulted in an approximately twofold increase in sternal and forearm skin blood flow, confirming earlier speculations of increases in skin blood flow during hot flashes.^{17–19,26,27} The mechanism(s) of increases in skin blood flow during a hot flash are unknown. Increases in skin blood flow of nonglabrous (ie, hairy) skin of this magnitude during a hot flash could be achieved through the withdrawal of sympathetic vasoconstrictor activity, increases in sympathetic cholinergic vasodilator activity, or a combination of both neural mechanisms and nonneural factors, as proposed by Freedman et al²⁷ and Wilkin.²⁸

Hot flashes typically have been subjectively described by symptomatic women as beginning in the chest and radiating to the face, head, and arms.^{1,4,7,8} In the present study the start of the increase in sternal and forearm skin blood flow occurred at the same time during the hot flash episodes (15–30 s before the hot flash), which is in contrast to the subjective descriptions of hot flashes reported previously. The chest is one of the strongest thermal discriminators of the body.²⁹ During a hot flash, women may therefore perceive the increase in skin blood flow at the chest before the forearm despite these increases occurring at the same time.

BP is controlled through a combination of alterations in cardiac output and total peripheral resistance (or its inverse, vascular conductance). Pronounced elevations in vascular conductance of a large circulatory bed, such as the skin during a hot flash, can lead to an acute reduction in BP if there is inadequate cardiovascular compensation. To the authors' knowledge, only two prior studies have investigated BP responses during hot flashes. In one study no change was reported in intermittently measured BP,¹⁷ whereas the other study showed a 40-mm Hg decrease in systolic BP during one hot flash from one participant when BP was continuously measured.²² Before the present study it was not clear whether differences between these findings were related to the physiology of the participants evaluated or to the possibility that the first group missed a hypotensive response because of intermittent measurement of BP. To that end, in the present study 5 of the 12 participants exhibited pronounced decreases in BP, which was measured both continuously and intermittently during their hot flashes. Given the present observations, possible explanations for differences in the findings of the previous study that showed no change in BP during a hot flash¹⁷ could be that in the prior study the sample population did not include participants we classified as BP responders and/or that a hypotensive response was missed in that study because BP was obtained intermittently, despite our observation of reductions in BP in some participants even when obtained intermittently.

The mechanism resulting in a decrease in BP during a hot flash is not clear, except that it would be caused by a reduction in cardiac output, a reduction in total vascular resistance, or a combination of both mechanisms. Measurements of cardiac output were not made in the present study, although given similar increases in heart rate between groups during the hot flashes, it would be unlikely that a reduction in cardiac output is responsible for the reduction in BP (assuming stroke volume was similar between groups). Thus, reduced total vascular resistance is the most likely cause of the reduction in BP during the hot flash. During a hot flash cutaneous vascular resistance (expressed as an increase in CVC) decreases, and this decrease in vascular resistance has the capability of reducing BP. However, the magnitude of the elevation in CVC

was not different between the BP responder and nonresponder groups, suggesting that the reduction in BP during the hot flash was not due to greater increases in CVC in the BP responder group. Thus, changes in vascular resistance of noncutaneous beds of the BP responder group are the most likely mechanism for the observed reductions in BP during a hot flash. Further studies are warranted to confirm these speculative conclusions.

It is interesting to note that despite BP decreasing approximately 10 mm Hg during the hot flashes of the BP responder group, the elevation in heart rate was similar between groups. Assuming that the hot flash does not alter baroreflex responsiveness, one would expect that a greater reduction in BP during the hot flash would be accompanied by a greater increase in heart rate. An explanation for the absence of a greater increase in heart rate during the hot flash of the BP responders, despite a lower BP, could be impaired baroreflex responsiveness of these participants, as proposed previously.²² Alternatively, it could be that the elevation of heart rate due directly to the hot flash is not additive to a baroreflex-mediated increase in heart rate associated with the hypotensive challenge. If the latter is correct, then the elevation in heart rate would not necessarily be greater in those participants whose BP decreased during the hot flash.

We recognize that the small number of participants in the BP responder and nonresponder groups is a limitation of the study. There were no statistical differences in baseline anthropometric characteristics between the BP responder and nonresponder groups, suggesting that participants in the BP responder group are not distinguished by particular anthropometric features. Given the small numbers of participants in both groups, however, it is unclear whether the lack of differences in anthropometric characteristics between these groups would still be evident in larger numbers of BP responder and nonresponder participants. Despite the small numbers of participants in the BP responder and nonresponder groups, the difference in the BP responses during the hot flashes of these two groups was clearly evident. Given the possibility of a type II error for those responses where no differences were observed, further research may be necessary to confirm the present findings as well as to examine associated mechanisms of the observed changes.

CONCLUSIONS

In summary, large transient increases in cutaneous blood flow occurred upon the onset of a hot flash, confirming earlier speculations of increases in skin blood flow during hot flashes. In addition, transient decreases in BP occurred during hot flashes in some symptomatic postmenopausal women. There were no differences between BP responder and nonresponder groups with respect to increases in heart rate, CVC, and sweating during the hot flash. The absence of a difference in the increase in CVC between groups suggests that the lower BP during a hot flash in the BP responder group was unlikely due to greater reductions in cutaneous vascular resistance. The mechanism of a decrease in BP during a hot flash in these individuals, as well as mechanisms responsible for increases in skin blood flow in both groups, remain to be determined.

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FIG. 1. Blood pressure (A) and heart rate (B) during 11 hot flashes in the responder subgroup ($n = 5$) and 12 hot flashes in the nonresponder subgroup ($n = 7$). # $P < 0.05$ vs 2 minutes before a hot flash. * $P < 0.05$, responders vs nonresponders.

FIG. 2. Core temperature during 11 hot flashes in the responder subgroup (n = 5) and 12 hot flashes in the nonresponder subgroup (n = 7). #P < 0.05 vs 2 minutes before a hot flash.

FIG. 3.

Changes in sternal (A) and forearm (B) sweat rate during 11 hot flashes in the responder subgroup ($n = 5$) and 12 hot flashes in the nonresponder subgroup ($n = 7$). # $P < 0.05$ vs 30 seconds before a hot flash. Two forearm sweat rate responses from one participant in the nonresponder group were larger than the rest of the group, causing the larger variation in this group's data.

FIG. 4. Sternal (A) and forearm (B) cutaneous vascular conductance (CVC) (percent maximum) during hot flashes in the responder ($n = 5$) and nonresponder ($n = 7$) subgroups. $\#P < 0.05$ vs 2 minutes before a hot flash.

TABLE 1

Anthropometric data for the entire participant group

Age (y)	Height (cm)	Weight (kg)	BMI (kg/m ²)	SBP (mm Hg)	DBP (mm Hg)	MABP (mm Hg)
50 ± 5	163 ± 6	74 ± 16	28 ± 6	120 ± 10	73 ± 4	92 ± 9

Data are mean ± SD; n = 12. BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; MABP, mean arterial blood pressure.