# ABSENCE OF BAROREFLEX MODULATION OF SKIN SYMPATHETIC NERVE ACTIVITY AND SWEAT RATE DURING WHOLE-BODY HEATING IN HUMANS

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Baroreflex control of skin blood flow during heating is well documented, but the effects of baroreceptor loading/unloading on sweating are less clear. Therefore, this project tested the hypothesis that pharmacologically induced alterations in blood pressure in heated humans would lead to baroreflex mediated changes in skin sympathetic nerve activity and sweat rate. In seven subjects, and under normothermic and heat stress conditions, mean arterial blood pressure was first reduced (~10 mmHg) and then increased (~15 mmHg) via bolus infusions of 100 µg sodium nitroprusside follow by 150 µg phenylephrine. These drugs were infused through a catheter inserted into an antecubital vein. Bolus phenylephrine administration began approximately 60 seconds after the onset of nitroprusside infusion. In both normothermic and heat stress conditions the following responses were monitored: sublingual and mean skin temperatures, heart rate, beat-by-beat blood pressure (Colin), skin blood flow (laser-Doppler flowmetry), local sweat rate (capacitance hygrometry), and skin sympathetic nerve activity (microneurography from peroneal nerve). Whole-body heating increased skin and sublingual temperatures, heart rate, cutaneous blood flow, sweat rate, and skin sympathetic nerve activity, but did not change arterial blood pressure. During whole-body heating, heart rate was significantly elevated during sodium nitroprusside-induced reductions in blood pressure (74 ± 4 to 92 ± 4 bpm; $P<0.001$) and significantly reduced during phenylephrine-induced elevations in blood pressure (92 ± 4 to 68 ± 4 bpm; $P<0.001$), thereby demonstrating appropriate baroreflex function in these subjects. Skin sympathetic nerve activity was not affected by pharmacologically induced alterations in blood pressure regardless of the thermal condition. Similarly, sweat rate was not attenuated when blood pressure was reduced during whole-body heating. To eliminate the possibility that the lack baroreflex modulation of skin sympathetic nerve activity and sweat rate was due to rapid and transient changes in blood pressure, in four subjects steady state intravenous infusions of sodium nitroprusside (20 to 60 µg • $kg^{-1}$ • $min^{-1}$) were administered over a period of 8-12 minutes. Steady-state decreases in mean arterial blood pressure of ~10 mmHg caused baroreflex-mediated increases in heart rate (~20 bpm) but did not change skin sympathetic nerve activity during normothermia. Furthermore, during the heat stress steady-state reductions in mean arterial blood pressure did not significantly change skin sympathetic nerve activity or sweat rate. These results indicate that the lack of change in sweat rate and skin sympathetic nerve activity observed during bolus infusions of vasoactive drugs was not due to the short time period in which blood pressure was altered. Taken together, these data suggest that skin sympathetic nerve activity and sweat rate are not modulated by arterial baroreflexes in normothermic or moderately heated individuals.

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