Interaction of hyperthermia and heart rate on stroke volume during prolonged exercise

Joel D. Trinity, Matthew D. Pahnke, Joshua F. Lee, and Edward F. Coyle

Human Performance Laboratory, Department of Kinesiology and Health Education, The University of Texas at Austin, Austin, Texas

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Address for reprint requests and other correspondence: E. F. Coyle, Dept. of Kinesiology and Health Education, The Univ. of Texas at Austin, 1 Univ. Station-D3700, Austin, TX 78712-0360 (e-mail: coyle@mail.utexas.edu).

Interaction of hyperthermia and heart rate on stroke volume during prolonged exercise. J Appl Physiol 109: 745–751, 2010. First published July 1, 2010; doi:10.1152/japplphysiol.00377.2010.—People who become hyperthermic during exercise display large increases in heart rate (HR) and reductions in stroke volume (SV). It is not clear if the reduction in SV is due primarily to hyperthermia or if it is a secondary effect of an elevation in HR during ventricular filling. In the present study, the upward drift of HR during prolonged exercise was prevented by a very small dose of the β1-adrenoreceptor blocker (atenolol; βB), thus allowing SV to be compared at a given HR during normothermia and hyperthermia. Eleven men cycled for 60 min at 57% of peak O2 uptake after receiving placebo control (PL) or a low dose (0.2 mg/kg) of βB. Hyperthermia was induced by reducing heat dissipation during exercise. Four experimental conditions were studied: normothermia-PL, normothermia-βB, hyperthermia-PL, and hyperthermia-βB. Hyperthermia increased skin and core temperature by 4.3°C and 0.8°C (P < 0.01), respectively. βB prevented HR elevation with hyperthermia: HR values were similar at minute 60 during normothermia-PL and hyperthermia-βB (155 ± 11 and 154 ± 13 beats/min, respectively, P = 0.82). However, SV was increased by 7% during the final 20 min of exercise during hyperthermia-βB compared with normothermia-PL (treatment × time interaction, P = 0.03). In conclusion, when matched for HR, mild hyperthermia increased SV during exercise. Furthermore, the reduction in SV throughout prolonged exercise under normothermic and mildly hyperthermic conditions appears to be due to the increase in HR.

Cardiovascular control; heart function; β1-adrenoreceptor blocker; cardiovascular drift

EXERCISE RAISES CORE TEMPERATURE, and it has been reported that cardiac stroke volume (SV) during exercise declines at high core temperatures (12, 14, 17, 19, 30, 31, 35, 38, 39, 42). It is not clear if the reduction in SV is due primarily to hyperthermia or if it is a secondary effect of heart rate (HR) elevation reducing ventricular filling. The interactions of hyperthermia, HR, and SV are evident by the close relationship between increased core temperature and elevations in HR (11, 32) and the finding that a lack of an increase in core temperature is associated with a stable SV (15, 17). Hyperthermia during exercise elevates HR (15, 19, 25, 35, 48), and it is possible that the reduction in SV is related to reduced ventricular filling time due to this increase in HR (1, 34, 44). The effect of hyperthermia during exercise on SV independent of the elevation in HR has not been described.

Cardiovascular drift, a phenomenon characterized by progressive reductions in SV and increases in HR, illustrates the interaction of core temperature, HR, and SV (8, 11, 24, 35). The classic concept of cardiovascular drift maintains that the reduction in SV is due to progressive increases in cutaneous blood flow (CBF) as core temperature increases (36, 37, 39). It is thought that the rise in CBF leads to an increase in skin venous volume, reducing ventricular filling pressure, end-diastolic volume, and, thus, SV (37). However, a previous report found that the pattern of cardiovascular drift is not temporally related to increases in CBF (11). More importantly, it was demonstrated that when the upward drift in HR is prevented by a small dose of a β1-adrenoreceptor blocker, the reduction in SV is also prevented (11). This implies that the characteristic reduction in SV during prolonged exercise is due to an increased HR (1, 34, 44). The aforementioned studies of cardiovascular drift were conducted at moderate temperatures, and not in a hot environment. Exercise in a hot environment produces substantially greater cardiovascular drift as well as higher rates of CBF (13, 19, 35, 37, 39, 43). It is possible that the decline in SV during prolonged exercise in the heat is due to factors other than elevated HR, including a direct effect of hyperthermia on myocardial function (21, 41). Whether controlling HR during exercise with heat stress prevents the reduction in SV has yet to be determined.

The purpose of this study was to determine if hyperthermia, independent of an increase in HR, reduces SV during exercise. To accomplish this, we used a low dose of the β1-adrenoreceptor blocker atenolol to prevent HR from drifting upward during exercise in normothermic and hyperthermic subjects. A second aim of this study was to determine if the progressive reduction in SV and increase in HR are temporally related to increases in CBF during exercise under hyperthermic conditions.

METHODS

Subjects

Eleven healthy and active men who were not heat acclimated [24 ± 5 (range 18–34) yr of age] provided written informed consent to participate in the study. The protocol, experimental design, and informed consent form were approved by the Institutional Review Board at The University of Texas at Austin. The subjects’ stature, body mass, peak O2 uptake (V02peak), and maximal HR (means ± SD) were as follows: 1.77 ± 0.06 m, 77.8 ± 12.4 kg, 3.88 ± 0.54 l/min, and 193 ± 8 beats/min, respectively.

Experimental Trials

Subjects cycled for 60 min at a constant work rate that elicited ~57% of V02peak. At 3 min before the exercise bout, subjects ingested 1) 0.2 mg/kg of the β1 (cardioselective)-adrenoreceptor blocker atenolol (βB) or 2) placebo control (PL). βB was in liquid oral suspension (2 mg/ml). One hundred milliliters of a noncaloric flavored
solution was used to mask the βB and to match the flavor with that of the PL. The appropriate dose of βB was determined on the basis of pilot testing as a lower dose (0.1 mg/kg) of βB used by Fritzsche et al. (11), was not sufficient to attenuate the increase in HR under hyperthermic conditions.

**Experimental Protocol and Design**

**Preliminary testing.** The submaximal O₂ uptake (V̇O₂) vs. work rate relationship was determined during a continuous incremental cycle ergometer (Excalibur Sport, Lode, Groningen, The Netherlands) protocol. This protocol consisted of 20 total min of cycling at four submaximal workloads at a constant pedal rate (freely chosen by subject). Work rates varied slightly between subjects but typically started at ~80 W and progressed every 5 min until ~200 W. This protocol served to 1) provide a warm-up prior to the maximum V̇O₂ (V̇O₂max) test and 2) establish the V̇O₂ vs. work rate relationship. After a 10-min rest, subjects returned to the cycle ergometer, and V̇O₂peak and maximal HR were determined during a continuous incremental cycle ergometer protocol. This protocol consisted of 2-min stages, with exhaustion occurring between 8 and 12 min. O₂ and CO₂ concentrations of inspired and expired gases were determined by a mass spectrometer (MGA 1100, Perkin-Elmer, St. Louis, MO).

**Familiarization trials.** Subjects performed two familiarization trials. The first familiarization trial was performed under normothermic conditions, while the second was performed under hyperthermic conditions. The familiarization trials mimicked the experimental trials as described below, with the exception of the antecubital vein catheterization. Sweat volume during 1 h of exercise was determined during the familiarization trials: [sweat volume (liters) = (preexercise nude body mass − postexercise nude body mass) + fluid consumed]. This information was used to determine the appropriate volume of fluid consumption during the experimental trials to maintain euhydration. On the basis of the familiarization trials, sweat volume and fluid consumption were significantly greater during hyperthermia than normothermia trials (1.65 ± 0.35 vs. 1.27 ± 0.41 liters, P < 0.01).

**Experimental Procedures**

Upon arrival to the laboratory, subjects voided their bladder, recorded their nude body mass, and inserted the rectal temperature probe. Hydration status prior to each trial was determined by measurement of urine specific gravity. Thereafter, the subjects sat quietly while an antecubital vein was catheterized for blood sampling; they then moved to the cycle ergometer, where instrumentation was completed. Hyperthermic conditions were achieved by having subjects wear a vinyl rain jacket and nylon/spandex leg coverings while two parabolic electric heaters (Heatdish, National Presto Industries, Eau Claire, WI) were directed at the subject (front and back). Normothermic conditions were achieved without fan cooling with an environmental temperature of 23°C and 35% relative humidity. Subjects wore cycling shorts, socks, and shoes during the normothermia trials. A set of Dill and Costill (7). Complete data for Hb and Hct are reported for nine subjects because of technical problems with blood sampling in two subjects.

**CBF and forearm blood flow.** CBF was measured continuously by laser-Doppler flowmetry (MoorLab, Moor Instruments) on the ventral side of the left forearm. For consistency across trials, the location of the laser-Doppler probe was marked with an indelible marker. Forearm blood flow (FBF) was measured by venous occlusion plethysmography (EC6 plethysmograph, Hokanson, Bellevue, WA) according to the procedures outlined by Whitney (45). During this measurement, a mercury-in-Silastic strain gauge was placed over the largest circumference of the left forearm. For consistency of placement within a trial as well as across trials, the location of the gauge was marked with an indelible marker. An occlusion cuff was placed at the wrist and inflated to 250 mmHg to restrict blood flow to the hand for 2 min prior to the FBF measurement. A second cuff was placed around the upper arm and rapidly inflated to 50 mmHg, which occludes venous outflow while arterial inflow continues. The increase in forearm circumference, measured by the strain gauge, was plotted with data acquisition software (NIVP3, Hokanson). During the FBF measurements, the arm was suspended just above heart level in a custom-made sling that supported the weight of the arm. FBF was measured prior to exercise and heating and during exercise at minutes 12, 35, and 55. A series of at least six successive FBF measurements were performed, with the mean value representing the FBF for a given time point.

**Body temperatures and rating of perceived exertion.** Rectal (core) temperature was recorded using a thermistor (model 401, Measure-
ment Specialties, Hampton, VA) inserted 12 cm past the anal sphincter. Skin temperature was recorded from skin thermistors (model 409A, Measurement Specialties) at six sites: chest, back, upper arm, forearm, thigh, and calf. All skin thermistors were placed on the left side of the body. Mean skin temperature was calculated on the basis of the weighted average of the six sites (20). Ratings of perceived exertion [RPE; scored at 6–20 on the Borg scale (2)] were reported at minutes 5, 10, 15, 20, 30, 40, 50, and 60.

Statistics

Data are expressed as means ± SD unless otherwise stated. A two-way (treatment × time) repeated-measures ANOVA was used to determine significant differences between means. According to our original statistics plan, changes from minute 10 to 60 (effect of time within a given treatment) were treated as planned comparisons, and least significant difference comparison was used to determine significance of changes from minute 10 to 60. The Sidak correction for multiple comparisons was used to determine significant differences between all other comparisons. If the sphericity assumption was violated, the Greenhouse-Geisser correction was employed to ensure significant differences for the main effect. If no difference was observed between PL and βB trials for a given temperature condition, the trials were combined to determine the independent effect of temperature in normothermic and hyperthermic conditions. A paired-samples t-test was used to determine significant differences between means of combined groups. Correlations of the changes in HR vs. core temperature and SV vs. CBF were completed using the Pearson product moment of correlation method. To determine the onset of βB, a paired-samples t-test was used to determine the first significant difference in HR for normothermia-PL vs. normothermia-βB and hyperthermia-PL vs. hyperthermia-βB. Significance was accepted at P < 0.05.

RESULTS

Respiratory Responses

For all trials, the 1-h bout of exercise was performed at 147 ± 18 W, which elicited ~57% of V̇O₂peak. V̇O₂ was similar between trials and increased significantly (P < 0.01) by 6% from minute 10 (range 2.08–2.19 l/min) to minute 60 (range 2.24–2.30 l/min).

Body Temperature Regulation: Core and Skin Temperatures

The technique employed to induce hyperthermia was successful at increasing core and skin temperatures during exercise (Fig. 1). The earliest significant difference for core temperature between normothermia and hyperthermia trials occurred at minute 30 (P < 0.01). During exercise, skin temperature was well maintained between 30.3°C and 31.5°C during normothermia trials and between 35.1°C and 36.0°C during hyperthermia trials. At minute 60, hyperthermia elevated core and skin temperatures by 0.8°C (38.9 ± 0.5°C vs. 38.1 ± 0.3°C, P < 0.01) and 4.3°C (35.7 ± 0.7°C vs. 31.4 ± 0.7°C, P < 0.01), respectively. There were no differences for core or skin temperature between normothermia-PL and normothermia-βB or between hyperthermia-PL and hyperthermia-βB trials. The increases in core temperature and HR from minute 10 to 60 were significantly correlated during the PL trials (r = 0.59, P < 0.01).

Cardiovascular Responses

The onset of βB occurred at minute 15 during normothermia-βB (142 ± 12 vs. 136 ± 12 beats/min, P < 0.01) and minute 10 during hyperthermia-βB (149 ± 11 vs. 144 ± 14 beats/min, P = 0.03). The HR response under each condition is presented in Fig. 2. During normothermia-PL, HR increased 11% from minute 10 to 60 (from 139 ± 13 to 155 ± 11 beats/min, P < 0.01). Hyperthermia-PL nearly doubled the increase in HR (i.e., 21% increase) during this same time period (from 149 ± 11 to 180 ± 9 beats/min, P < 0.01). Normothermia-βB prevented the normal increase in HR during minute 10 to 60 (from 136 ± 12 to 135 ± 12 beats/min, P = 0.89). HR increased 7% during hyperthermia-βB (from 144 ± 14 to 154 ± 13 beats/min, P < 0.01) from minute 10 to 60. However, after minute 15 there were no further significant increases in HR during hyperthermia-βB. HR was not different at any time point between normothermia-PL and hyperthermia-βB (Fig. 2). Therefore, the βB treatment was successful at controlling HR, despite a significant increase in core and skin temperature.

The SV response under each condition is presented in Fig. 2. SV declined from minute 10 to 60 by 9% during normothermia-PL (from 135 ± 17 to 123 ± 15 ml/beat, P < 0.01) and by 14% during hyperthermia-PL (from 128 ± 14 to 111 ± 14 ml/beat, P < 0.01; Fig. 2). During normothermia-βB and hyperthermia-βB, SV did not significantly change from minute 10 to 60. Contrary to our original hypothesis, hyperthermia increased SV at a given HR, as evidenced by the 7% higher SV during hyperthermia-βB than normothermia-PL at minutes 40,
50, and 60 (treatment × time interaction, P = 0.03; Figs. 2 and 3). Furthermore, hyperthermia resulted in a rightward shift in the SV vs. HR response, indicating that, under hyperthermic conditions, SV was increased for a given HR (Fig. 3). The hyperthermia trials were characterized by a slight but significant increase in CO (P < 0.01; Table 1). There were no significant differences in MAP between normothermia-PL and normothermia-βB or between hyperthermia-PL and hyperthermia-βB. Overall, MAP decreased from minute 10 to 60 during the normothermia and hyperthermia trials. MAP was consistently and significantly reduced during exercise in the hyperthermia trials (Table 1).

**Forearm and Cutaneous Blood Flow**

FBF and CBF were not different between normothermia-PL and normothermia-βB or between hyperthermia-PL and hyperthermia-βB. Hyperthermia increased FBF and CBF during exercise. By minute 60, hyperthermia increased FBF by 40% (P < 0.01) and CBF by 37% (P < 0.01) compared with normothermia (Fig. 4). SV was not significantly correlated to CBF when expressed in absolute terms (r = 0.08, P = 0.49) or as the change in SV vs. the change in CBF from minute 10 to 60 (r = 0.34, P = 0.14). Overall, FBF and CBF did not increase significantly after minute 15 in any condition.

**Body Mass and Blood Volume**

Hydration status prior to exercise as measured by preexercise body mass (77.9 ± 11.5, 77.7 ± 11.5, 77.8 ± 11.9, and 77.5 ± 11.5 kg during normothermia-PL, normothermia-βB, hyperthermia-PL, and hyperthermia-βB, respectively, P = 0.41) and urine specific gravity (1.013 ± 0.007, 1.019 ± 0.008, 1.013 ± 0.007, and 1.017 ± 0.008 during normothermia-PL, normothermia-βB, hyperthermia-PL, and hyperthermia-βB, respectively, P = 0.22) were not different between trials. There was no significant difference in sweat volume between normothermia-PL and normothermia-βB or between hyperthermia-PL and hyperthermia-βB. Sweat volume was higher during the hyperthermia trials than during the normothermia trials (1.81 ± 0.40 vs. 1.23 ± 0.53 liters, P < 0.01). Fluid replacement maintained body mass between 0.1 ± 0.2 and −0.2 ± 0.2 kg of preexercise body mass. BV was equally reduced during exercise compared with rest for all trials (Table 2). There were no significant differences between conditions for changes in BV.

**Perceived Exertion**

There were no significant differences in RPE between normothermia-PL and normothermia-βB or between hyperthermia-PL and hyperthermia-βB. RPE increased gradually over time in all trials, and the first indication that the hyperthermia trials were perceived as requiring greater effort occurred at minute 30 (12.9 ± 0.8 and 13.8 ± 1.0 during normothermia and hyperthermia, respectively, P < 0.01), coinciding with the first significant difference in core temperature between the hyperthermia and normothermia trials. At minute 60, RPE remained elevated in the hyperthermia trials compared with the normothermia trials (15.8 ± 1.7 vs. 14.1 ± 1.2, P < 0.01).

**DISCUSSION**

This study used a small dose of βB to successfully match HR during hyperthermic exercise (hyperthermia-βB) with the HR typically seen during normothermic exercise (normothermia-PL, Fig. 2). Our first major finding is that, with nearly
identical HR during the normothermia-PL and hyperthermia-βB conditions. SV was significantly increased by 7% with mild hyperthermia (Fig. 3). Hyperthermia (0.8°C elevation of core temperature) had no effect on reducing SV, provided HR was controlled. We also found that the progressive reduction in SV was temporally unrelated to altered CBF during exercise under hyperthermic conditions. Furthermore, during the hyperthermia-βB trial, SV was high when CBF was also elevated, indicating that elevations in skin blood flow, under the present conditions during exercise, do not necessarily reduce SV.

To our knowledge, the present study is the first to document that mild heat stress in combination with moderate-intensity exercise results in an increase in SV when HR is controlled to match normothermic levels. Evidence of maintained or elevated SV during hyperthermia has been reported during passive heat stress at rest (5, 23, 28, 29, 37, 38, 47). The mechanism responsible for this elevated SV is not entirely clear but may be related to increased cardiac function or reduced afterload, as evidenced by reductions in MAP. Hyperthermia resulted in a significant 7-mmHg reduction in MAP compared with normothermia, which agrees with previously reported reductions in MAP during heat stress at rest and during exercise (23, 37) (Table 1). Regarding the possibility of improved cardiac function, Crandall et al. (4, 6) showed that an elevation of 1°C in core temperature at rest increases ejection fraction by 8% as a result of a 24% reduction in end-systolic volume. Although ejection fraction is an imperfect index of cardiac contractility, these authors (4, 6) and others (37) hypothesized that the improvement in cardiac function may be a result of increased diastolic function resulting in larger diastolic filling for a given filling pressure (6, 46). Direct and more precise echocardiographic evidence of improved atrial and ventricular systolic function during passive heat stress has been reported during passive heat stress (3). Whether similar improvements in cardiac function accounted for the 7% increase in SV at a given HR during exercise in hyperthermia is unknown.

Table 1. CO and MAP during 1-h exercise bout for normothermia-PL, normothermia-βB, hyperthermia-PL, and hyperthermia-βB

<table>
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<th>Time, min</th>
<th>5</th>
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<tr>
<td>Normothermia-PL</td>
<td>7.0 ± 1.1</td>
<td>18.1 ± 2.2</td>
<td>18.6 ± 1.9</td>
<td>18.6 ± 2.1</td>
<td>18.9 ± 2.3</td>
<td>19.2 ± 2.2</td>
<td>18.9 ± 2.2</td>
<td>19.3 ± 2.1</td>
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<tr>
<td>Normothermia-βB</td>
<td>6.9 ± 1.1</td>
<td>18.1 ± 1.7</td>
<td>18.1 ± 1.9</td>
<td>18.2 ± 1.7</td>
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<td>18.8 ± 2.3</td>
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<td>NORMOTHERMIA</td>
<td>7.0 ± 1.1</td>
<td>18.1 ± 1.9</td>
<td>18.4 ± 1.9</td>
<td>18.4 ± 1.9</td>
<td>18.7 ± 2.1</td>
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<td>7.1 ± 0.9</td>
<td>19.0 ± 2.3</td>
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<tr>
<td>Normothermia-PL</td>
<td>93.7 ± 8.5</td>
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<td>96.0 ± 6.8</td>
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<td>NORMOTHERMIA</td>
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<td>111.2 ± 7.6</td>
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<td>Hyperthermia-PL</td>
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<td>Hyperthermia-βB</td>
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<td>HYPERThERMIA</td>
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*Significant difference between NORMOTHERMIA and HYPERThERMIA; †significantly different from minute 10 (P < 0.05).

During exercise in hot environments, SV is attenuated while HR and CBF are elevated. It has been suggested that the attenuation of SV is partly due to the increased CBF and reduced venous return (24). In the present study, during hyperthermia-βB, CBF was high, yet SV was also high. Similar to the resting hyperthermic conditions described above, high CBF, per se, does not necessarily reduce SV during exercise. However, hyperthermia will raise HR, which secondarily appears to lower SV.

It should be noted that the purpose of this study was not to understand the role of βB in cardiovascular control during prolonged exercise. βB was used as a tool to control HR. Previous investigators have elegantly described the effects of therapeutic doses of atenolol and propranolol on cardiovascular and thermoregulatory function during exercise (10, 26, 27). βB, when given in therapeutic doses (100 mg/day, nearly 7-fold higher than the present study), elicits large changes in peripheral and central hemodynamics, as evidenced by an increase in FBF, elevated rectal and skin temperature, and reductions in CO, MAP, and total peripheral resistance (10). In the present study, the small dose of βB only altered the HR and SV response but had no effect on any of these other variables.

Reductions in SV and CO have been reported during intense and/or prolonged exercise in the heat (12, 14, 15, 17–19, 30, 31, 35, 38, 39, 42). Hyperthermia elevates HR, and it is likely that this contributes to a reduction in SV. Gonzalez-Alonso et al. (15) found that hyperthermia, without dehydration, reduced SV by 11 ml/beat and increased HR by 8 beats/min. A follow-up study (18) revealed that supine exercise in hyperthermic subjects restored two-thirds of the reduction in SV and prevented one-third of the increase in HR. On the basis of these findings (15, 18) and the consistent reduction in SV, hyperthermia appeared to be a critical factor affecting SV during exercise. However, in these studies (15, 18) and others (25, 36, 39, 42, 43), hyperthermia always resulted in a concomitant elevation in
HR compared with thermoneutral or fan-cooled environments. This persistent tachycardia associated with hyperthermia confounds the ability to determine the independent effect of hyperthermia, per se, on SV. Perhaps the severity of hyperthermia prevented a reduction in SV under the conditions of the present study. Although significantly higher than normothermia by 0.8°C, the core temperature achieved during the present hyperthermia trials was, on average, 38.9°C, substantially less than that reported by Gonzalez Alonso et al. (39.3–40.2°C) (15, 19). It is possible that the elevation in SV for a given HR as was observed in the normothermia-PL condition may not occur under conditions of more severe hyperthermia.

In conclusion, when the typical increase in HR that accompanies elevations in core temperature during exercise is prevented, the classically observed reduction in SV is not observed, and, in fact, SV is increased by 7%. This implies that mild hyperthermia during exercise does not compromise heart function, per se. The progressive increase in HR that occurs during exercise under normothermic and hyperthermic conditions appears to be the primary factor responsible for the decrease in SV.

**DISCLOSURES**

No conflicts of interest, financial or otherwise, are declared by the authors.

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**Table 2. Percent change in BV from rest to exercise for normothermia-PL, normothermia-βB, hyperthermia-PL, and hyperthermia-βB**

<table>
<thead>
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<th>Time, min</th>
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<tr>
<td>Hyperthermia-βB</td>
<td>-6.4 ± 2.2</td>
</tr>
</tbody>
</table>

Values are means ± SD of 9 subjects. Percent change in blood volume (BV) from rest to exercise was measured during 60 min of exercise at −57% of peak O₂ uptake. All changes were significantly different from rest (P < 0.05).

This study is similar to our previous study in which a small dose of atenolol was administered at the start of prolonged exercise (11). The prior study was conducted during thermoneutral conditions, and thus the present study, by adding hyperthermia trials, examines the cardiovascular responses during more stressing conditions and when HR is more markedly elevated. The present findings during the two normothermic conditions agree very well with the observations of Fritzsch et al. (11).

The reason for the continuous increase in HR during the 10- to 60-min period of prolonged exercise is not entirely clear but is most likely related to the continual increase in core temperature and, possibly, to an increase in feedback from exercising muscle as fatigue progresses. Increases in core temperature can elicit increases in HR through direct effects on intrinsic HR at the sinoatrial node (25), via activation of muscle thermoreflexes (40), and/or by increasing whole body sympathetic activity (9). On the basis of the results of this study, the progressive increase in HR during normothermic and hyperthermic conditions is not temporally related to progressive increases in CBF, FBF, or skin temperature, as none of these variables changed significantly from minute 15 to 60 of exercise. Typically, during prolonged exercise, the increases in HR, perceived exertion, and core temperature are prevented by reducing the environmental stress, reducing the exercise intensity (43), or having well-trained, euhydric, heat-acclimated athletes (15, 16) perform the exercise bout. An alternate explanation for the increase in HR as put forth by Fritzsch et al. (11) is that progressive fatigue and an increase in motor unit recruitment coupled with an increase in core temperature may account for the increase in HR. The current finding of a significant relationship (r = 0.59, P < 0.01) between core temperature and HR during the hyperthermia-PL and normothermia-PL trials lends support for the role of the progressive increase in core temperature leading to part of the increase in HR.

Fig. 4. Forearm blood flow (A) and cutaneous blood flow (B) at rest and during 1 h of exercise. Values are means ± SE of 11 subjects. †Significant difference between pooled hyperthermia and pooled normothermia data (P < 0.05).
REFERENCES


