Stroke volume decline during prolonged exercise is influenced by the increase in heart rate

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Fritzsche, Ricardo G., Thomas W. Switzer, Bradley J. Hodgkinson, and Edward F. Coyle. Stroke volume decline during prolonged exercise is influenced by the increase in heart rate. J. Appl. Physiol. 86(3): 799–805, 1999.—This study determined whether the decline in stroke volume (SV) during prolonged exercise is related to an increase in heart rate (HR) and/or an increase in cutaneous blood flow (CBF). Seven active men cycled for 60 min at ∼57% peak O2 uptake in a neutral environment (i.e., 27°C, 40% relative humidity). They received a placebo control (CON) or a small oral dose (i.e., 7 mg) of a β1-adrenoceptor blocker atenolol (BB) at the onset of exercise. At 15 min, HR and SV were similar during CON and BB. From 15 to 55 min during CON, a 13% decline in SV was associated with an 11% increase in HR and not with an increase in CBF. CBF increased mainly from 5 to 15 min and remained stable from 20 to 60 min of exercise in both treatments. However, from 15 to 55 min during BB, when the increase in HR was prevented by atenolol, the decline in SV was also prevented, despite a normal CBF response (i.e., similar to CON). Cardiac output was similar in both treatments and stable throughout the exercise bouts. We conclude that during prolonged exercise in a neutral environment the decline in SV is related to the increase in HR and is not affected by CBF.

blood pressure; blood volume; body temperature regulation; cardiovascular regulation; exertion; forearm venous volume

Methods

Subjects. Seven healthy and active men (21–37 yr of age) provided written informed consent to participate in this 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 (VO2peak), and maximal HR (means ± SE) were as follows: 1.78 ± 0.03 m, 75.5 ± 2.1 kg, 3.90 ± 0.13 l/min, and 184 ± 3 beats/min, respectively. VO2peak and maximal HR were determined during a continuous, incremental cycle-ergometer protocol. The subjects also completed one familiarization trial during which they practiced the cardiac output (CO) rebreathing technique used in the study.

Protocol and experimental design. Subjects cycled for 60 min at a constant work rate that elicited ∼57% of VO2peak in a neutral environment (27°C and 18°C dry and wet bulb temperature, respectively; no fan). Within 2 min before the exercise bout, they ingested 1) 0.1 mg/kg body mass of the β1 (cardioselective)-adrenoceptor blocker atenolol (i.e., BB) or 2) a placebo control (i.e., CON). BB and CON were ingested with 10 ml/kg body mass of a 6% carbohydrate-electrolyte solution to prevent confounding effects of dehydration on HR and SV.

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To obtain the desired atenolol dose, 100-mg tablets (Tenormin) were converted to a fine powder and mixed, and the target amount was weighed on a precision scale. Inasmuch as all desired variables could not be measured during a single exercise bout, identical duplicate exercise bouts (i.e., bouts A and B) were performed for each experimental treatment. Experimental measurements during bout A included O₂ uptake (VO₂), CO₂ production (VCO₂), CO, HR, systolic and diastolic blood pressures (SBP and DBP, respectively), Hb and hematocrit, esophageal and rectal temperatures, skin temperatures, laser-Doppler blood flow, and rating of perceived exertion, body mass, and maximal HR. Bout B of each experimental treatment was used to record FBF and forearm venous volume. HR and rectal temperature were also recorded during bout B to ensure that cardiovascular and thermoregulatory responses were similar during bouts A and B.

Pharmacodynamic studies described the atenolol effects on exercise-induced tachycardia 2 h after oral atenolol doses ranging from 12.5 to 400 mg (3) and the time course of decline in resting, standing HR after 100 and 200 mg of oral atenolol administration (10). However, to our knowledge, this is the first study to use partial β-blockade to maintain a stable HR. The present dose and timing of atenolol administration (0.1 mg/kg immediately before exercise) were selected by trial and error during preliminary experiments. The goal of these experiments was to find the atenolol dose and timing of ingestion that prevented the increase in HR that usually occurs during prolonged exercise (i.e., after ~15 min), but not the increase in HR elicited by the initial responses to exercise (i.e., from 0 to ~15 min of exercise). To prevent the confounding effects of dehydration on cardiovascular responses, the treatments were ingested with ~750 ml of fluid.

All exercise bouts were performed ≥3 days apart, and their order was counterbalanced. For each exercise bout the subjects reported to the laboratory at the same time of the day and ≥3 h postprandially. Exercise intensity and environmental conditions were selected to allow comparisons with previous studies (6, 7, 19) often cited (32) in the cardiovascular drift literature.

Experimental procedures. On arrival, subjects dressed in shorts and cycling shoes. Before bout A of each experimental treatment, they inserted their esophageal and rectal probes, voided their bladder, and recorded their nude body mass. An antecubital vein was catheterized for blood sampling, and subjects entered the environmental chamber and sat quietly on the cycle ergometer (Leger) for 15 min before a resting blood sample was obtained. Then they received their experimental treatment and started the 60-min exercise bout. To compare maximal HR with and without β₁-adrenoceptor blockade, at 60 min, six of seven subjects exercised to fatigue (~2–3 min) at 120% of the work rate previously estimated to elicit VO₂peak.

Respiratory and cardiovascular measurements. VO₂ and VCO₂ were measured using open-circuit spirometry. Briefly, subjects breathed through a two-way sliding valve (Hans Rudolph) attached to a one-way Daniel valve, connected in turn to a dry gas volume meter (model CD4, Parkinson-Cowan) and to a mixing chamber. Expired air was continuously sampled from the mixing chamber and analyzed for O₂ (model S-3A/I, Ametek) and CO₂ (model CD-3A, Ametek) concentrations. Both gas analyzers and the dry gas meter were interfaced to a laboratory computer. The gas analyzers were calibrated every 20 min by using gases of known concentrations. CO was measured with the CO₂-rebreathing technique outlined by Collier (4). End-tidal and equilibrium CO₂ concentrations were recorded from continuous (i.e., breath-by-breath) recordings made at the mouthpiece-valve connection by another CO₂ analyzer (model CD-3A, Ametek) interfaced to the same laboratory computer. CO is reported as the average of three measurements collected during the 3- to 10-, 12- to 19-, 32- to 39-, and 52- to 59-min periods. HR was recorded continuously and averaged every 15 s (Polar Vantage XL Heartwatch). The HR recorded during the 1-min period before CO₂ rebreathing was used for the calculation of SV. Also during this period, SBP and DBP were measured by auscultation on the right arm with use of microphones under a blood pressure cuff (model STBP-680, Colin). Mean arterial pressure (MAP) was calculated as MAP = (SBP + 2DBP)/3. Systemic vascular resistance (SVR) was calculated during each determination of CO as SVR = MAP/CO.

Blood volume. During bout A of each experimental treatment, blood samples (totaling ~18 ml/treatment) were withdrawn immediately before exercise and at 3, 6, 9, 12, 15, 18, 35, and 55 min. Hb concentration was analyzed in triplicate with the cyanmethemoglobin technique. Hematocrit was measured in triplicate after microcentrifugation and corrected for trapped plasma and venous sampling (14). The changes in blood volume (percent change from rest) were calculated from the changes in Hb and hematocrit (5).

CBF and forearm venous volume. CBF was measured continuously during bout A by laser-Doppler flowmetry (model 21, ALF). The skin probe was placed on the ventral side of the left forearm. Laser-Doppler CBF is reported as a percentage of the resting value recorded immediately before treatment ingestion. Exercise time to the onset of cutaneous vasodilation (t₁), exercise time to attenuation of the rate of cutaneous vasodilation (t₂), and exercise time to a stable CBF (t₃) were determined from the CBF-time graph by an experienced researcher who was blinded to the subject and experimental condition being analyzed.

FBF was measured by venous occlusion plethysmography according to the procedures outlined by Whitney (41). Briefly, the occlusion cuff was inflated to 55 mmHg and blood flow to the hand was restricted. FBF was measured as the average of 8–10 values obtained at rest and every 5 min during exercise. FBF values were used as an index of forearm CBF (19). The procedures outlined by Wenger and Roberts (40) were used to measure forearm venous volume at rest (15 min after sitting on the ergometer in the environmental chamber) and once every 5 min during exercise. Briefly, after FBF recordings, blood flow to the hand remained restricted, and the occlusion cuff was inflated to ~30 mmHg and maintained until a stable forearm circumference was recorded. Then the occlusion cuff was rapidly deflated, and when forearm circumference was stable again, it was recorded. The difference between these two forearm circumferences was extrapolated to calculate differences in forearm volume (41).

Body temperatures, sweat volume, and rating of perceived exertion. Esophageal temperature was recorded using a thermistor (model 491A, Yellow Springs Instrument) inserted through the nasal passage and swallowed to a depth of one-fourth of the subject’s standing height (22), and mean skin temperature was recorded every 5 min from skin thermistors (model 409A, Yellow Springs Instrument) attached to plastic holders and placed at six skin sites (16). Rectal temperature was recorded at rest and at the end of all exercise bouts using a thermistor (model 401, Yellow Springs Instrument) inserted 12 cm past the anal sphincter. Body mass was measured with a platform scale (model FW 150 KA, Acme), and whole body sweat volume was calculated from body mass changes (28). Rating of perceived exertion (2) was recorded at 20, 40, and 60 min during exercise.

Statistics. Data were analyzed with a two-way (treatment-by-time) multivariate ANOVA for repeated measures. Accor-
ing to the original statistical analysis plan, cardiovascular and thermoregulatory data collected at 3–10, 10–20, 30–40, and 50–60 min were averaged to 5, 15, 35, and 55 min, respectively. After a significant F-test, the significance of pairwise comparisons was determined with Tukey's post hoc tests. The onset of β-blockade was estimated by comparing HRs between BB and CON every 5 min with use of paired t-tests (to improve the likelihood of detecting a significant difference). To assess the association between the decline in SV from 15 to 55 min of exercise during CON and potentially related variables, stepwise forward linear regression analysis was performed on three time points (15, 35, and 55 min). For regression analysis, all variables were transformed to standardized z scores to remove the variation between subjects. Standardized z scores were calculated for each subject and each variable by using the 15-, 35-, and 55-min time points. The level of statistical significance on all tests was set at \( P < 0.05 \).

**RESULTS**

Respiratory and cardiovascular variables. Subjects performed the experiment at a work rate (144 ± 5 W) that elicited 57% \( \dot{V}O_2 \)peak. \( \dot{V}O_2 \) and \( \dot{V}CO_2 \) were similar in CON and BB (Table 1). CO was maintained throughout exercise (\( \dot{V}O_2 \)peak and \( \dot{V}CO_2 \)) were similar in BB and CON (Table 1). Total sweat volume was similar during CON and BB (936 ± 85 and 939 ± 102 ml, respectively). FBF and CBF, FBF and laser-Doppler CBF were similar in CON and BB throughout exercise (Fig. 1). FBF and CBF did not increase significantly (\( P = NS \)) during the 15–35 min period; however, in two subjects, FBF and CBF continued to increase until ~25 min. From 35 to 55 min, FBF and CBF were clearly stable in all subjects (Fig. 1). Forearm venous volume was similar in BB and CON at rest (2.22 ± 0.21 and 2.37 ± 0.16, respectively) and throughout the 60-min exercise period (Table 1).

Variables that changed over time during the first 20 min of exercise (before a significant β-blockade effect). From 5 to 15 min of exercise, SV and blood volume declined and HR increased during BB and CON (\( P < 0.05 \); Fig. 1). Esophageal temperature as well as both measures of CBF (i.e., FBF and CBF) increased rapidly from 5 to 15 min during BB and CON (\( P < 0.05 \); Fig. 1). The \( t_1, t_2, \) and \( t_3 \) were similar in BB and CON: 5.04 ± 0.51 and 6.64 ± 0.43 min (\( t_1 \)), 12.14 ± 0.72 and 12.57 ± 1.06 min (\( t_2 \)), and 18.29 ± 2.40 and 16.86 ± 3.22 min (\( t_3 \)), respectively. Therefore, the main increase in CBF occurred from ~5 to ~15 min. Forearm venous volume declined significantly below resting values during the first 5 min of exercise (\( P < 0.05 \)) but remained relatively stable thereafter.

**RESULTS**

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### Table 1. Exercise responses during placebo control and β1-adrenoceptor blockade

<table>
<thead>
<tr>
<th>Variable</th>
<th>5 min</th>
<th>15 min</th>
<th>35 min</th>
<th>55 min</th>
</tr>
</thead>
<tbody>
<tr>
<td>( \dot{V}O_2 ), l/min</td>
<td>CON 2.17 ± 0.04</td>
<td>BB 2.14 ± 0.05</td>
<td>2.32 ± 0.05*</td>
<td>2.32 ± 0.05*</td>
</tr>
<tr>
<td>( \dot{V}CO_2 ), l/min</td>
<td>CON 2.12 ± 0.05</td>
<td>BB 2.05 ± 0.05</td>
<td>2.15 ± 0.06</td>
<td>2.15 ± 0.06</td>
</tr>
<tr>
<td>CO, l/min</td>
<td>CON 1.66 ± 0.05</td>
<td>BB 1.64 ± 0.05</td>
<td>1.63 ± 0.05</td>
<td>15.9 ± 0.4</td>
</tr>
<tr>
<td>SBP, mmHg</td>
<td>CON 166 ± 2</td>
<td>BB 169 ± 2</td>
<td>167 ± 2</td>
<td>163 ± 2*</td>
</tr>
<tr>
<td>DBP, mmHg</td>
<td>CON 66.6 ± 1</td>
<td>BB 66.7 ± 1</td>
<td>66 ± 1</td>
<td>162 ± 2</td>
</tr>
<tr>
<td>MAP, mmHg</td>
<td>CON 99.8 ± 1</td>
<td>BB 101 ± 0.7</td>
<td>99.9 ± 0.9</td>
<td>194 ± 1.4*</td>
</tr>
<tr>
<td>SVR, dy·cm·s⁻⁵</td>
<td>CON 487 ± 12</td>
<td>BB 500 ± 16</td>
<td>480 ± 14</td>
<td>477 ± 16</td>
</tr>
<tr>
<td>( T_{es} ), °C</td>
<td>CON 31.4 ± 0.3</td>
<td>BB 31.3 ± 0.3</td>
<td>31.9 ± 0.9</td>
<td>31.9 ± 0.9</td>
</tr>
<tr>
<td>FVV, ml/100 ml</td>
<td>CON 1.26 ± 0.14</td>
<td>BB 1.37 ± 0.15</td>
<td>1.15 ± 0.10</td>
<td>0.92 ± 0.12</td>
</tr>
</tbody>
</table>

Values are means ± SE of 7 subjects for \( \dot{V}O_2 \) uptake (\( \dot{V}O_2 \)), CO2 production (\( \dot{V}CO_2 \)), cardiac output (CO), systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP), systemic vascular resistance (SVR), mean skin temperature (\( T_{es} \)), and forearm venous volume (FVV) during 60 min of exercise at ~57% of peak \( \dot{V}O_2 \). CON, placebo control; BB, \( \beta_1 \)-adrenoceptor blockade.

* Different from 15 min, BB, and CON pooled (\( P < 0.05 \)).
sion analysis tested the strength of the association between SV as a dependent variable and HR, CO, CBF, FBF, forearm venous volume, MAP, esophageal temperature, mean skin temperature, and blood volume as independent variables. HR was entered in the first step [total $R^2 (R^2_1) = 0.908$], CO in the second step ($R^2_2 = 0.96$), and MAP in the third step ($R^2_3 = 0.976$), with no further variables reaching statistical significance. $R^2_2$ illustrates the fraction of variance in SV explained by HR (1st step), HR and CO (2nd step), and HR, CO, and MAP (3rd step).] A legitimate criticism of this regression analysis is that part of the association of HR and CO with SV occurs because HR, CO, and SV are not independently measured (i.e., SV is calculated from CO and HR). Therefore, a second regression analysis on SV was performed after HR and CO were excluded. When HR and CO were excluded, esophageal temperature was the only variable significantly associated with SV ($R^2_1 = 0.861$). Esophageal temperature was not significantly associated with SV in the first regression analy-
PROLONGED-EXERCISE STROKE VOLUME DEPENDS ON HEART RATE

DISCUSSION

The main finding of this study is that the decline in SV that occurs after 15 min of exercise is related to an increase in HR and is temporally unrelated to an increase in CBF. The observations that support this finding are twofold: 1) during BB, prevention of the increase in HR after 15 min of exercise prevented the decline in SV, despite a normal CBF; and 2) during CON, a stable CBF after 15–20 min of exercise did not prevent the decline in SV.

In the present experiment, during the 15–55 min of CON, SV declined 13%, HR increased 11%, and final core temperature was 37.8°C. Similar cardiovascular and thermoregulatory responses were previously reported during prolonged moderate-intensity exercise in a neutral environment (6–8, 19). Furthermore, in the present and other studies (19, 25, 26), mean CBF was stable after 30 min of exercise, although individual CBF responses may deviate from this pattern (19). Therefore, the cardiovascular and thermoregulatory responses observed in the present study appear similar to responses (6–8, 19) referenced by Rowell (32) to describe cardiovascular drift.

In the present study, partial β1-adrenoceptor blockade was used successfully to maintain a stable HR after 15 min of exercise. During prolonged exercise, chronic atenolol administration in typical clinical doses (i.e., 100 mg/day) lowers HR, blood pressure, and CO (12) and increases perceived exertion (21). However, because of the small dose of atenolol used in this study (i.e., 7.5% of a usual dose), perceived exertion, CO, and blood pressure were not different between BB and CON. Additionally, the lowering of submaximal and maximal HR induced by atenolol was relatively small in the present compared with other studies (12, 21) and was not noticed until after 15 min of exercise. It is not surprising that a small dose of atenolol did not affect the cutaneous circulation in this experiment, inasmuch as no significant effect was observed even with large doses (12).

This study shows that when the increase in HR during 15–55 min of exercise was prevented by BB, SV failed to decline. A logical explanation for this observation is that under the present experimental conditions the increase in HR is largely responsible for the decline in SV during prolonged exercise. It seems unlikely that an unknown mechanism related to atenolol ingestion prevented the decline in SV during BB. All measured variables that could have been causally related to the decline in SV (i.e., blood volume, laser-Doppler CBF, FBF, forearm venous volume, esophageal temperature, skin temperature) were similar during BB and CON. Therefore, the lack of a decline in SV during BB suggests that an increase in HR elicits the decline in SV normally observed during prolonged exercise (i.e., during CON).

Increases in HR, by reducing diastolic filling time (39), have the potential to decrease end-diastolic volume and SV, provided that end-diastolic volume is not maximal (e.g., during moderate upright exercise) (27). Indeed, experiments using heart pacing at rest and during exercise (1, 30, 38) indicate that increases in HR produce reductions in SV. Therefore, it seems that increases in HR have the potential to elicit the decline in SV during prolonged exercise observed in CON and other studies (6–8).

CBF did not appear to be related to the decline in SV during the 15- to 55-min period in the present study. First, data from CON and BB and from other studies (19, 25, 26) indicate that CBF does not increase after 20–30 min of moderate-intensity exercise in a neutral environment. Additionally, CBF was not significantly associated (stepwise regression analysis) with the decline in SV during CON. Also, during BB a normal cutaneous circulatory response (i.e., similar to CON) did not elicit a decline in SV. It could be argued that the cutaneous circulatory response was not normal during BB and that, despite similar CBFls, BB somehow elicited a reduction in cutaneous venous compliance and volume that prevented the decline in SV. However, our knowledge, β1-adrenoceptor blockers do not have any effect on cutaneous veins. Forearm venous volume provides an estimate of cutaneous venous tone and venous compliance during exercise (11). Because forearm venous volume was not different between BB and CON at any time point, it appears that atenolol did not influence cutaneous veins. Further evidence against venous pooling as a mechanism for cardiovascular drift in the present study is the observation that the decline in SV induced by prolonged exercise is not prevented during supine exercise (7), a condition that should minimize venous pooling. Therefore, the available evidence suggests that the decline in SV after 20–30 min of moderate-intensity exercise in a neutral environment (i.e., cardiovascular drift) is not normally elicited by an increase in CBF or venous volume.

The lack of relationship between CBF and SV during prolonged moderate-intensity exercise (in a neutral environment) does not argue against the fact that several treatments that do indeed manipulate venous pooling and/or the cutaneous circulation can modify SV. It is known that the hydrostatic effects of upright posture cause large increases in the volume of blood contained in the veins below the heart (32). By reducing venous volume below the heart and thus increasing venous return, supine exercise (27) and upright exercise with leg bandages (15) increase SV compared with regular upright exercise values. However, the decline in SV observed during supine exercise (7) or exercise with leg bandages (15) does not appear to be different from the decline in SV observed during prolonged regular upright exercise (i.e., cardiovascular drift). During exercise at skin temperatures that are high enough to abolish cutaneous venous tone (i.e., ~38°C) (33), the cutaneous circulation also appears to have powerful effects on SV that can be reversed by cooling the skin (34). Therefore, venous pooling can certainly lower SV. However, to our knowledge, there is no evidence to indicate that progressive venous pooling...
occurs during prolonged moderate-intensity exercise in the present environmental conditions (i.e., skin temperatures of 31–32°C).

In the present study, CBF was not associated with the decline in SV during prolonged exercise. However, it should be recognized that the decline in SV observed during the first 15 min of exercise could be elicited by increases in CBF, as well as by increases in HR and/or declines in blood volume. It should also be recognized that the present findings might not apply to other exercise intensities, environmental conditions, or subject populations. Lower exercise intensities might have prevented a decline in SV (4, 37), whereas higher intensities would be expected to increase the magnitude of the decline in SV (8, 24). Likewise, warmer (24) and cooler (37) environmental conditions might also have modified the magnitude of the decline in SV observed during CON.

One way to explore the potential mechanism for the increase in HR during prolonged exercise is to compare the exercise model of this study with other models in which cardiovascular drift is prevented. The increase in HR during prolonged exercise was prevented when the exercise intensity or environmental stress is lower (37) or when trained, euhydrated, heat-acclimated subjects familiar with the exercise mode are used (13). In these studies, when the increase in HR during prolonged exercise was prevented, the increase in perceived exertion (37) and core temperature (13, 37) was also prevented. In contrast, in the present study, perceived exertion and core temperature increased in parallel with HR. The increase in perceived exertion during prolonged exercise observed in the present study may be related to an increase in the effort needed to recruit the same or a higher number of motor units. Iasmuch as it occurs during static exercise (36), a progressive increase in motor unit recruitment could elicit the progressive increase in HR observed in this study (via central command and/or muscle feedback). Although it seems unlikely that the 0.3°C increase in core temperature observed in this study could, by itself, elicit a 15 beat/min increase in HR, increases in core temperature, through direct effects on the intrinsic HR (20), activation of muscle thermoreflexes (35), and/or increases in whole body sympathetic activity (9), could also contribute to the increase in HR during prolonged exercise. Additional studies would need to separate the contribution of endurance training, heat acclimation, and familiarization with the exercise mode to the attenuation of the increase in HR during prolonged exercise and to separate the influence of perceived exertion and core temperature on the increase in HR.

In summary, during CON the decline in SV during the 15- to 55-min exercise period was not associated with an increase in CBF, which was stable during this period. Most importantly, when partial β1-adrenoceptor blockade prevented the normal increase in HR, the decline in SV during the 15- to 55-min exercise period was also prevented, whereas CBF was unaffected. We conclude that the decline in SV during prolonged moderate-intensity exercise in a neutral environment (i.e., cardiovascular drift) depends on the increase in HR and is not related to changes in the cutaneous circulation.

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