Heat acclimation increases arterial elasticity in young men

Trin Kaldur, Jaak Kals, Vahur Õöpik, Andres Burik, Priit Kampus, Maksim Zagura, Mihkel Zilmer, and Eve Unt

Abstract: The major physiological adaptations that occur during heat acclimation (HA) are well documented. However, no studies have provided compelling evidence about the effect of HA on arterial elastic properties. The aim of this study was to examine the changes in large artery elasticity (LAE) and small artery elasticity (SAE) concomitant with HA and to determine the potential relationships among changes in arterial elasticity, baseline aerobic fitness level, and improvement in endurance capacity (EC). During 10-day HA, the subjects (n = 21) exercised daily on a treadmill for 110 min at an intensity of 55%-60% of peak oxygen uptake in a climatic chamber preset to 42 °C and 18% relative humidity. EC was tested in the heat before and after HA. Arterial elasticity was assessed by diastolic pulse wave analysis (HD/Pulse Wave CR-2000) at baseline and after HA. Blood samples were drawn at baseline. After HA, there was a 17% increase in LAE (from 21.19 ± 4.72 ml-mm Hg-1 x 10 to 24.77 ± 5.91 ml-mm Hg-1 x 10, p < 0.05) and an 18% increase in SAE (from 9.32 ± 1.76 ml-mm Hg-1 x 100 to 10.98 ± 1.75 ml-mm Hg-1 x 100, p < 0.01). EC increased by 86% (from 88.62 ± 27.51 min to 161.95 ± 47.80 min, p < 0.001) as a result of HA. No significant associations were revealed between changes in arterial elasticity parameters and improvement in EC or baseline aerobic fitness level. We demonstrated, for the first time, that HA has a positive impact on the parameters of arterial elasticity. Further investigations are needed to determine the mechanisms underlying these changes and the potential relationships among arterial elasticity, aerobic fitness level, and EC.

Key words: cardiovascular risk, exercise in the heat, endurance capacity.

Introduction

The capacity for prolonged exercise is diminished in the heat relative to normothermic environments (Galloway and Maughan 1997). Exercise in the heat can pose a severe challenge to human cardiovascular control, particularly if individuals have to cope with the stress of dehydration and hyperthermia (Gonzalez-Alonso et al. 2008). A high ambient temperature increases cardiovascular strain during exercise (Gonzalez-Alonso et al. 1997) and that strain has been proposed as a possible mediator of fatigue during prolonged exercise in the heat (Cheung and Sleivert 2004).

Heat acclimation (HA) is a key cardiovascular strain mitigation strategy that has been employed widely to prepare athletes and military personnel to perform in hot environments. It is well established that the physiological adaptations that arise from repeated exposure to exercise-heat stress (e.g., adjustments in plasma and stroke volume, heart rate, cutaneous blood flow, sweating, core temperature) lead to heat adaptation within an organism, which results in improvement in heat tolerance and work performance (Houmard et al. 1990; Sawka et al. 1996).

To the best of our knowledge, the potential impact of HA on arterial elastic properties has never been investigated, and data on the effect of acute heat stress on arterial elasticity are scarce and inconsistent. Specifically, there is in vitro evidence suggesting
that direct heating of isolated iliac arteries to a very high temperature (60 °C) increases vessel elasticity (Mitchel et al. 1994). In contrast, in a small group of men and women of various ages, a core temperature increase of up to 1.5 °C above baseline achieved with passive heating by means of a water-perfused suit did not affect the average peripheral or central arterial elasticity (Garino et al. 2011). Furthermore, there is a lack of consensus regarding the effect of exercise stress on arterial elastic properties. In both younger and older adults, arterial stiffness (inverse of elasticity) is decreased for a short time through a single bout of endurance exercise (Kingwell et al. 1997; Nickel et al. 2011), and regular exercise is associated with decreased arterial stiffness (Maeda et al. 2008; Tanaka et al. 2000). In older adults with multiple cardiovascular risk factors, short-term improvements in arterial stiffness induced by aerobic training became attenuated over the long term (Madden et al. 2012). There is evidence that the response of the arteries' elasticity parameters to endurance exercise is related to the subject's maximal oxygen uptake (VO₂max) (Kampus et al. 2008). Nevertheless, increased aortic stiffness has been observed in marathon runners compared with age-matched control subjects (Vlachopoulos et al. 2010), and resistance exercise has been shown to decrease arterial elasticity (DeVan et al. 2005; Miyachi et al. 2004). No studies have provided compelling evidence regarding the effect of HA from repeated exposure to combined exercise and heat stress on arterial elasticity. Therefore, the purpose of the current investigation was to examine the changes in arterial elasticity of the large and small arteries concomitant with HA and to determine the potential relationships among changes in arterial elasticity, baseline aerobic fitness level, and improvement in endurance capacity (EC).

Materials and methods

Ethical approval

The subjects were informed of all details of the experimental procedures and the associated risks and discomfort before they provided their informed consent in writing. The study was approved by the Research Ethics Committee of the University of Tartu, and the study protocol conformed to the ethical guidelines of the 1975 Declaration of Helsinki.

Subjects

Twenty-one healthy young men between the ages of 19 and 32 years, who were recruited from the university population and the military community (military college cadets), volunteered to participate in the study. The volunteers were nonsmokers, taking no medication, and free of any symptoms of cardiovascular disease; had not been exposed to warm weather over the preceding 2 months; and did not have a history of heat illness. All subjects were characterized as physically active; they exercised habitually from 2 to 10 times per week (mean 4.4 ± 1.9 times per week). The mean duration of each training session was 1.5 ± 0.4 h and their mean habitual exercise volume was 6.6 ± 3.4 h per week. During their involvement in the study, the training load was assessed by a questionnaire, which revealed that there was no additional training volume except for the exercise related to the HA program and testing procedures.

Study design

The current investigation was part of a complex HA study (Burk et al. 2012). Experiments were conducted in Estonia (situated on the coordinates of 59° north latitude and 26° east longitude) during the winter-spring period (from December to March) to avoid natural HA. Therefore, the subjects' natural HA state should have been at the annual nadir. The highest average outdoor temperature during the study period was registered in March (−1.1 °C) and the lowest in January (−12.7 °C). The parameters of arterial elasticity and EC in the heat were measured at baseline and after the HA program (Fig. 1).

Preliminary measurements

The subjects underwent a medical examination, and baseline blood samples were drawn the morning after an overnight fast, after the baseline measurement procedures for arterial elasticity. Total cholesterol, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, triglycerides, creatinine, and glucose were determined in the local laboratory of Tartu University Hospital according to standardized protocols. Body height, using Martin’s metal anthropometer, and body mass, using an electronic scale (CH3G1501 Combics, Sartorius AG, Goettingen, Germany), were measured (±0.001 m and ±0.001 kg, respectively).

Before the actual experiment, each subject was tested for peak oxygen uptake (VO₂peak). A walking exercise test in thermoneutral conditions was performed to assess the peak exercise capacity in the heat.
HA program

The HA program employed in the current study was a modification of that used previously by Yamada et al. (2007). The subjects completed a 10-day program, in which they exercised for 10 consecutive days in a climatic chamber maintained at a hot temperature (42 °C; relative humidity 18%). According to the HA protocol, daily exposure consisted of 110 min of exercising on a treadmill (two 50-min bouts of exercise with 10 min of rest between bouts) at a workload predicted to elicit an oxygen uptake of 55% of VO2peak during the first 5 days; the workload was raised to the level of 65% of VO2peak for the second 5 days of the HA protocol. The intensity of the exercise was controlled by changing the grade of the treadmill belt (ranging from 5% to 15%), whereas the speed was kept constant at 6 km·h⁻¹. The participants wore shorts, socks, and athletic shoes. Sessions took place under the supervision of experienced personnel. Core temperature measured via a rectal probe (TX-2, Columbus Instruments, Columbus, Ohio, USA) and heart rate measured via a transmitter strap (Suunto Dual Belt, Suunto OY, Finland) were monitored continuously throughout all heat exposure to detect clinical symptoms of heat illness. Termination criteria included the following: (i) completion of the protocol, (ii) a rise in core temperature to 39.5 °C for 5 min, (iii) a rise in heart rate to 95% of maximal heart rate for 5 min, (iv) symptoms of exertional heat illness, or (v) a subject’s request to stop.

Measurements of arterial elasticity

Measurements of arterial elasticity were conducted twice (at baseline and after the HA program) in the morning after an overnight fast in thermoneutral conditions (Fig. 1). The arterial waveform was measured in the dominant arm by a cardiovascular profiling instrument (HDl/pulse Wave CR-2060, APPL. PHYSIOL. NURS. METAB. VOl. 38, 2013). The results of this test were used to establish the workload for the protocol of the EC test and for the HA program.

Measurements of EC in the heat

The EC test was conducted at baseline and after the HA program (Fig. 1). The subjects were instructed to refrain from alcohol for 24 h and from caffeine for 12 h prior to each experimental trial. To ensure that they were hydrated adequately, the subjects were instructed to consume an additional 500 ml of water the night before and the morning of each experimental trial day. The test was performed on a treadmill in a climatic chamber (Design Environmental Ltd., Gwent, South Wales, UK) in the heat (42 °C; relative humidity 18%). The intensity was adjusted individually to 60% of the subject’s personal VO2peak by employing a constant speed of 6 km·h⁻¹ and regulating the grade of the belt of the treadmill (within the range of 7%-15%). The participants performed the test until exhaustion or until indications for the termination of an EC test occurred (core temperature above 40 °C for more than 5 min; heart rate above 95% of the individual maximal heart rate for at least 5 min; or symptoms of exertional heat illness, such as nausea, headaches, and dizziness).

Data analyses

All tests were performed using the Statistical Package for the Social Sciences (SPSS) software, version 18.0. All data were checked for normal distribution using the Kolmogorov–Smirnov test. Dependent samples (paired) t tests were used to compare the values of the baseline parameters and the values of the parameters after HA. Independent-samples t tests were used to determine the differences between the groups. A Pearson and Spearman product moment coefficient of correlation was used to determine the relationships among variables. For all statistical analyses, a 0.05 level of significance was used. All data are presented as means ± SD.

Results

Subject characteristics are outlined in Table 1. All subjects were characterized by a relatively high baseline aerobic fitness level, with a mean VO2peak > 50 mL·min⁻¹·kg⁻¹; 5 subjects had a VO2peak > 60 mL·min⁻¹·kg⁻¹. Systolic blood pressure (pre-HA 120.14 ± 9.65 mm Hg and post-HA 120.38 ± 7.95 mm Hg, p > 0.05) and diastolic blood pressure (pre-HA 65.88 ± 7.52 mm Hg and post-HA 63.86 ± 7.81 mm Hg, p > 0.05) did not change over the 10-day HA program. However, significant changes in resting heart rate, from 56.36 ± 9.35 beats·min⁻¹ to 49.81 ± 8.00 beats·min⁻¹ (p < 0.001), and core temperature, from 37.2 ± 0.2 °C to 37.0 ± 0.2 °C (p < 0.01), confirmed that an acclimated state was achieved successfully in our subjects.

After the HA program, all subjects showed improvement in EC (individual variation from 10 to 128 min); the subjects’ mean EC showed a statistically significant increase of 66% (from 88.62 ± 27.51 min to 161.95 ± 47.80 min, p < 0.001). Arterial elasticity

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Mean ± SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (y)</td>
<td>24.9±3.7</td>
</tr>
<tr>
<td>Height (m)</td>
<td>1.83±0.06</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>80.39±4.9</td>
</tr>
<tr>
<td>VO2peak (mL·min⁻¹·kg⁻¹)</td>
<td>5.84±7.1</td>
</tr>
<tr>
<td>Cholesterol (mmol·L⁻¹)</td>
<td>4.4±0.52</td>
</tr>
<tr>
<td>HDL cholesterol (mmol·L⁻¹)</td>
<td>1.4±0.31</td>
</tr>
<tr>
<td>LDL cholesterol (mmol·L⁻¹)</td>
<td>3.04±0.86</td>
</tr>
<tr>
<td>Triglyceride (mmol·L⁻¹)</td>
<td>0.9±0.53</td>
</tr>
<tr>
<td>Creatinine (µmol·L⁻¹)</td>
<td>8.2±1.07</td>
</tr>
<tr>
<td>HR (beats·min⁻¹)</td>
<td>56.3±6.35</td>
</tr>
<tr>
<td>PFBP (mm Hg)</td>
<td>122.1±5.65</td>
</tr>
<tr>
<td>PDBP (mm Hg)</td>
<td>65.8±5.72</td>
</tr>
</tbody>
</table>

Note: VO2peak: peak oxygen uptake; HDL: high-density lipoprotein; LDL: low-density lipoprotein; HR: heart rate; PFBP, peripheral systolic blood pressure; PDBP, peripheral diastolic blood pressure.

Table 1. Subject characteristics.
improved after HA: LAE and SAE (Fig. 2) showed a statistically significant increase after acclimation compared with the baseline values.

Considering the large interindividual variability in EC parameters and attempting to more comprehensively elucidate potential relationships between EC and arterial elasticity, we divided our subjects into 4 groups. The first 2 groups were based on EC values after the HA program: 99–150 min (Group 1; n = 11) vs 170–300 min (Group 2; n = 10). The second 2 groups were based on improvement in EC (EC value after HA program – EC value at baseline): 10–68 min (Group 3; n = 10) vs 73–128 min (Group 4; n = 11).

There were no statistically significant differences in arterial elasticity measured at baseline and after HA, or in HA-related changes in arterial elasticity between the groups made based on EC values after the HA program and those made based on improvement in EC (Table 2). However, we demonstrated significant improvements in LAE in Group 2 and in Group 4, which were characterized by superior EC after HA and greater improvement in EC due to HA, respectively. Significant improvement in SAE values occurred only in Group 1, in which the EC after HA was modest.

Correlation analysis determining the relationships among changes in arterial elasticity, baseline aerobic fitness level, and improvement in EC is outlined in Table 3.

Discussion
To our knowledge, this is the first experimental study to quantify HA-induced changes in arterial elasticity. The lowered resting heart rate and core temperature observed after, compared with before, a 10-day HA program confirmed that HA was achieved successfully. The main new finding of the current study was that heat and exercise stress together have a beneficial effect on arterial elastic properties. The current study showed a statistically significant improvement in the subjects’ arterial elasticity parameters compared with baseline values: a 17% increase in the LAE and an 18% increase in the SAE. Our study did not reveal a significant association between the simultaneous changes in arterial elasticity and the improvement in EC.

Arterial elasticity is determined primarily by the intrinsic elastic properties of the arteries. The elements of the arterial wall that determine its elasticity are the composition of elastin and collagen and the vascular tone exerted by its smooth muscle cells. An increased pulsatile flow in the arteries associated with exercise training may evoke an acute release of nitric oxide (NO) and lead to upregulation of NO production and an increase in the production of other vasoconstricting factors (Delp and Laughlin 1997; Rubanyi et al. 1986; Spier et al. 1999), which may regulate vascular tone. Changes in arterial smooth muscle tone alter the relative loading of collagen and elastin fibers (Belz 1995) so that vascular smooth muscle relaxation transfers wall stress from the stiffer collagen fibers to the more extensible elastin fibers, thus making the arterial wall more compliant.

It has also been suggested that elevations in core temperature may partly explain postexercise improvement in arterial elasticity. Increased blood flow through the arteries associated with elevated core temperature increases shear stress, which, in turn, releases NO and other endothelium-derived factors, which may regulate vascular tone and thereby decrease arterial stiffness (Bellien et al. 2010; Kellogg et al. 2003; Kinlay et al. 2001; Sugawara et al. 2007).

It has been shown that a single 30-min bout of exercise at 65% of VO_{2max} alone, without heat stress, elevates whole-body arterial elasticity by 66%, but this elevation declines to its baseline value within 1 h after exercise (Kingwell et al. 1997). It has also been demonstrated that a short-term period of endurance training improves arterial elasticity by 5%, but the effect cannot be maintained without continuing regular physical exercise (Kakiyama et al. 2005). In vitro evidence suggests that heat stress increases arterial elasticity (Mitchel et al. 1994), but the results of a study carried out on humans do not support this suggestion (Ganio et al. 2011). In the current study, the changes in the arterial elasticity parameters were probably caused by the combined effect of exercise and heat stress. Whatever the determinants and mechanisms underlying these changes in arterial elasticity parameters in our study, the changes observed are physiologically relevant to effective acclimation, specifically to cardiac loading. It has been established that a higher exercise capacity is associated with reduced arterial stiffness (Vaitkevicius et al. 1993). LAE is an important determinant of cardiac workload. A less stiff aorta and large arteries could contribute to the increased coronary blood flow during diastole, decreased cardiac afterload, and augmented left ventricular function, and thus to the improvement in aerobic exercise capacity (Kingwell 2002). Improved arterial elasticity helps decrease cardiovascular strain and delay the onset of fatigue during exercise.

In addition to contributing to the understanding of HA benefits on arterial elasticity, our study demonstrates that in the case of HA, it is important to monitor and take into account the impact of HA on the parameters of arterial function to improve cardiac adaptation and cardiac performance. Moreover, improved arterial elasticity helps minimize the risk of cardiovascular morbidity and mortality (Mattace-Raso et al. 2006; Weber et al. 2004).

A substantial increase in EC that occurred in our subjects in the heat over a rather short HA is in agreement with the findings of several previous studies (Amorim et al. 2008; Nielsen et al. 1993; Yamada et al. 2007). As discussed in detail earlier (Burk et al. 2012), the increased EC was apparently based on the observed physiological effects of HA (lower resting core temperature and increased
plasma volume, and reduced heat storage rate during exercise). Other researchers have observed a small increase in VO_{peak} (Lorenzo et al. 2010) and a reduction in oxygen uptake at a given absolute exercise intensity (Young et al. 1985) during HA programs of a similar duration. Therefore, to some extent, both of the latter effects of HA may have contributed to the increased EC in our subjects as well.

However, our study did not reveal significant associations between the improvement in the arterial elasticity parameters and the improvement in EC. There was a significant inverse correlation only between the improvement in LAE and EC measured after HA; individuals who had the biggest improvement in SAE had the lowest EC after HA. Our study data also showed that individuals who had the biggest HA-induced improvement in SAE had the lowest baseline SAE. Ganio et al. (2011) showed similarly that changes in arterial stiffness during passive heating are predicated on baseline stiffness. In our study, the arterial elasticity parameters did not differ in the subgroups of subjects formed either on the basis of EC values registered after HA or on the basis of the extent of improvement in EC over HA. These findings suggest that HA-induced changes in arterial elasticity and EC occur simultaneously but are not directly related to each other.

We demonstrated a significant improvement in LAE in the groups in which the values of EC after HA were higher (Group 2) and in which the improvement in EC after HA was greater (Group 4). There was no relationship between baseline VO_{peak} and HA-related changes in LAE and SAE in our subjects. This finding differs from the results of our previous study (Kampus et al. 2008), which suggested that the response of the arterial elasticity parameters to physical exercise may depend on the subject's VO_{max}. However, in that particular study (Kampus et al. 2008), a very small group (n = 7) of well-trained cadets (mean VO_{max} 66.0 mL·min^{-1}·kg^{-1}, range 57.6–77.3 mL·min^{-1}·kg^{-1}) was studied during a 3-day extreme physical load in the absence of environmental heat stress. Thus, substantial differences in the subjects' characteristics, the physical load employed, and environmental factors could explain the discrepancy in the data of these 2 studies. Unfortunately, there is a lack of systematic data on the association between VO_{max} and arterial elasticity parameters. Most previous studies employed a cross-sectional or case-control design, in which the VO_{max} described a more current physical activity level and which were confounded by other factors (age, sex, overweight, and so forth). Furthermore, all the participants in the current study were healthy young males with arterial elasticity values in the normal range. This may partially explain the absence of associations between the improvement in arterial elasticity indices and baseline VO_{peak}. Clearly, further studies are needed to elucidate the independent association between VO_{peak} and arterial elasticity.

The current study has some limitations: a relatively small number of study participants divided into small groups and the relatively high and homogenous fitness level of the whole sample. However, the nature of this kind of study design presumes that participants have a high fitness level because of the high physical strain in the HA program. The findings of our study have significant implications for future research. Because the sample size was small, the results of our study should be verified in large-scale investigations. Future trials may focus on different age and sex groups, as well as on participants with different fitness levels. Preliminary studies are needed to understand the exact mechanism by which HA improves arterial elasticity. Further research is also warranted to assess whether HA benefits can be caused by exercise or heat stress alone and how long these HA benefits can be retained.

In conclusion, the current study demonstrates that HA significantly improves LAE and SAE. No significant associations were revealed among changes in arterial elasticity parameters, baseline aerobic fitness level, and improvement in EC. The preliminary findings of this study have significant clinical implications for the monitoring of cardiovascular adaptation of HA. Further investigations are needed to determine the mechanisms underlying these changes in arterial elasticity and to determine the potential relationships among arterial elasticity, aerobic fitness level, and EC.

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### Table 2. Between-group differences in arterial elasticity.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>ΔLAE (r)</th>
<th>ΔSAE (r)</th>
<th>p</th>
<th>Group 1 (n = 11)</th>
<th>Group 2 (n = 10)</th>
<th>Group 3 (n = 10)</th>
<th>Group 4 (n = 11)</th>
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</thead>
<tbody>
<tr>
<td>VO_{peak}</td>
<td>0.218</td>
<td>-0.339</td>
<td></td>
<td></td>
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<tr>
<td>ΔEC</td>
<td>0.160</td>
<td>-0.396</td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>EC at baseline</td>
<td>0.092</td>
<td>-0.362</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>EC after HA</td>
<td>0.154</td>
<td>-0.465</td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>LAE at baseline</td>
<td>-0.362</td>
<td>-0.234</td>
<td></td>
<td></td>
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<tr>
<td>SAE at baseline</td>
<td>-0.015</td>
<td>-0.756***</td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>

Note: ΔLAE, difference between elasticity value measured after HA program and value measured at baseline; ΔSAE, difference between elasticity value measured after HA and value measured at baseline; VO_{peak}, peak oxygen uptake; EC, difference between EC value measured after HA and value measured at baseline; ΔEC, difference between EC value measured after HA and value measured at baseline; ΔEC, difference between EC value measured after HA and value measured at baseline; ΔEC, difference between EC value measured after HA and value measured at baseline; EC, endurance capacity; LAE, large artery elasticity; SAE, small artery elasticity. *p < 0.05; **p < 0.001.
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References


