Endurance training, overtraining and baroreflex sensitivity in female athletes

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Summary

We examined heavy training-induced changes in baroreflex sensitivity, plasma volume and resting heart rate and blood pressure variability in female endurance athletes. Nine athletes (experimental training group, ETG) increased intense training (70-90% VO₂max) volume by 130% and low-intensity training (<70% VO₂max) volume by 100% during 6-9 weeks, whereas the corresponding increases in six control athletes (CG) were 5% and 10% respectively. Maximal oxygen uptake (VO₂max) in the ETG and CG did not change, but in five ETG athletes VO₂max decreased from 53.0 ± 2.2 (mean ± SEM) (46.8-59.2) ml kg⁻¹ min⁻¹ to 50.2 ± 2.3 (43.8-56.6) ml kg⁻¹ min⁻¹ (P<0.01), indicating overtraining. Baroreflex sensitivity (BRS) measured using the phenylephrine technique and blood pressure variability (BPV) did not change, but the low-frequency power of the R-R interval variability increased in the ETG (P<0.05). The relative change in plasma volume was 7% in the ETG and 3% in the CG. The changes in BRS did not correlate with the changes in plasma volume, heart rate variability and BPV. We conclude that heavy endurance training and overtraining did not change baroreflex sensitivity or BPV but significantly increased the low-frequency power of the R-R interval variability during supine rest in female athletes as a marker of increased cardiac sympathetic modulation.

Introduction

Endurance training has been previously shown to increase vagal activity in relation to sympathetic activity (Ekblom et al., 1973), which can be detected by increased heart rate variability (Seals & Chase, 1989). Overtraining, resulting from heavy endurance training without adequate recovery, is also supposed to induce changes in autonomic function (Israel, 1976; Lehmann et al., 1993). Overtraining is a general term that indicates that an individual has been stressed by training and extraneous stressors to the extent that he/she cannot perform at an optimum level after an appropriate regeneration period (Fry et al., 1991). Over-reaching is short-term overtraining in which the restoration of performance capacity may take from several days to a few weeks. The overtraining state results from long-term overtraining in which the restoration of performance capacity may take several weeks and/or months (Kreider et al., 1998).

Baroreflex function is vagally mediated and essential in the regulation of blood pressure and fast heart rate changes (Reid, 1988). Baroreflex sensitivity (BRS) has been reported to increase (Somers et al., 1991; McDonald et al., 1993), decrease (Bedror & Tipton, 1987; DiCarlo et al., 1988; Gwirtz et al., 1990, Mack...
et al., 1991; Chen et al., 1995; Hyek et al., 1993) or be unchanged (Seals & Chase, 1989; McDonald et al., 1993; Sheldahl et al., 1994) with endurance training in athletes, in sedentary subjects and in animals and to be higher (Barney et al., 1988), lower (Smith et al., 1988; Shi et al., 1993a) or similar (Levine et al., 1991; Williamson & Raven, 1994) in athletes compared with sedentary subjects. Only a few studies have investigated the effects of training on BRS in female subjects (e.g. Hudson et al., 1987; Somers et al., 1991) and none in overtrained athletes. Hypothetically, the changes in BRS with endurance training could result from increased plasma volume (Mack et al., 1991, 1993), increased vagal tone (Barney et al., 1988; O'Leary & Seamans, 1993), increased vascular and ventricular compliance, cardiac hypertrophy and decreased baroreceptor density (Shi et al., 1993a).

Our purpose was to study whether endurance training and overtraining induce changes in baroreflex sensitivity, plasma volume and resting heart rate, and blood pressure variability in female endurance athletes.

Subjects and methods

Subjects

Fifteen healthy female endurance athletes (five runners, four cross-country skiers, three triathletes, three orienteers) with no smoking history were informed of the nature and aim of the experiment. They gave their written consent to participate in the study. They were familiarized with the experimental procedures in one preliminary measurement session 2 weeks before the actual experiment and were divided into two groups: (1) experimental training group (ETG, n = 9) and (2) control group (CG, n = 6). The purpose of the heavy experimental training period was to overtrain the ETG athletes. The criteria for overtraining were (1) decreased maximal oxygen uptake by at least 2 ml kg⁻¹ min⁻¹, (2) decreased treadmill performance, (3) unwillingness to train and a feeling of inability to continue training in combination with certain overtraining signs and symptoms, e.g. mood disturbances (decreased positive feelings: energetic, helpful, calm, vigorous, relaxed, confident; and increased negative feelings: irritable, depressed, moody, fatigued, anxious, confused, excited, desperate, unable to concentrate), sleeping problems, menstrual irregularities, bad appetite, trembling hands, sweating or other psychosomatic symptoms, and (4) no illness, injury or other factors explaining the performance decrement (Kuipers & Keizer, 1988; Fry et al., 1991; Hooper & McKinnon, 1995). Five ETG athletes were diagnosed as being overtrained and they formed a subgroup of overtrained athletes (OA subgroup). The athletes did not take any medication including oral contraceptives during the experimental period. All athletes had trained regularly for at least 1 year before the experiment. The physical characteristics of the athletes are presented in Table 1. The study protocol was reviewed and approved by the ethics committee of the University of Jyväskylä.

Training

During the entire training period, the ETG trained 7 days a week. Their training was closely supervised

Table 1 Physical characteristics of the athletes.

<table>
<thead>
<tr>
<th></th>
<th>n</th>
<th>Age (years)</th>
<th>Weight (kg)</th>
<th>Height (cm)</th>
<th>Fat (%)</th>
<th>Training (years)</th>
</tr>
</thead>
<tbody>
<tr>
<td>ETG</td>
<td>9</td>
<td>23 ± 12</td>
<td>62.7 ± 6.2</td>
<td>171.0 ± 4.9</td>
<td>19.9 ± 3.2</td>
<td>7.9 ± 5.3</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(19.0–27.3)</td>
<td>(50.6–70.5)</td>
<td>(164.0–177.0)</td>
<td>(16.4–24.3)</td>
<td>(1–16)</td>
</tr>
<tr>
<td>C</td>
<td>6</td>
<td>23 ± 7</td>
<td>67.5 ± 5.2</td>
<td>171.9 ± 4.9</td>
<td>20.4 ± 4.6</td>
<td>92 ± 3.7</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(20.7–27.5)</td>
<td>(58.9–73.9)</td>
<td>(163.7–178.0)</td>
<td>(160–26.8)</td>
<td>(3–13)</td>
</tr>
<tr>
<td>OA</td>
<td>5</td>
<td>25 ± 1</td>
<td>66.7 ± 3.7</td>
<td>173.6 ± 3.0</td>
<td>20.5 ± 2.6</td>
<td>7.6 ± 5.2</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(22.4–27.3)</td>
<td>(60.5–70.5)</td>
<td>(169.0–177.0)</td>
<td>(17.9–24.3)</td>
<td>(1–12)</td>
</tr>
</tbody>
</table>

*Calculated from skinfolds according to Durnin & Womersley (1974).
†Training years before entering the experiment.
Results are expressed as means ± SD (range).
ETG, experimental training group; CG, control group; OA, mean values of the five overtrained athletes, subgroup of the ETG.

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by the research staff. The intensity of training was determined individually based on the lactate threshold ($T_{\text{lac}}$). The training consisted of intense training (IT, intensity $\geq T_{\text{lac}}$ and 70–90% VO$_{2\text{max}}$), which included interval running [(5–12) km with a 2-min recovery in between] and continuous fast running (5–12 km) and low-intensity training (LIT, intensity $< T_{\text{lac}}$ and $<70\%$ VO$_{2\text{max}}$), which was mainly long-term (50 min to 3 h) running but also consisted of cycling, cross-country skiing and swimming. The frequency and volume of IT was increased by one exercise session each week, starting with one intensive exercise session and a treadmill test during the first week. The volume of LIT was planned to increase by 7–10% per week. The CG was allowed to train according to their own training schedule. Training of all athletes was light (LIT for no more than 1 h) for 2 days before the measurements. All training sessions were controlled by heart rate monitors (Polar Electro Sport Tester, Finland) and subjective feelings were recorded daily.

Venous blood samples, haemoglobin and haematocrit analyses and calculation of plasma volume changes

The athletes entered a quiet laboratory with constant temperature (20–23°C) and dimmed lights at 08.00, 09.00 or 10.00 h, 1 h after a light standard (300 kcal) breakfast. They were not allowed to drink coffee, tea, chocolate and cola drinks that morning or the previous evening or alcohol during the previous 48 h. Athletes rested on the examination table in the supine position for 20 min, after which a venous blood sample was taken from the antecubital vein. Haemoglobin and haematocrit were analysed immediately using a Sysmex microcell counter F-800 (Toa Medical Electronics, Japan). Relative changes in plasma volume were calculated by using the formula of Strauss (Greenfield et al., 1963; McDonald et al., 1993):

$$\%\Delta PV = \frac{100[Hb_B/Hb_A - (1 - Hct_B^{10^{-2}})/(1 - Hct_A^{10^{-2}})]}{100}$$

where $PV$ is plasma volume, $Hb$ is haemoglobin, $Hct$ is haematocrit, subscript B is before and subscript A is after the experimental training period.

Data acquisition and analysis of heart rate and blood pressure variability

Ten minutes after the blood sample the cardiovascular autonomic function measurement was started. Three standard ECG electrodes were attached to the chest and connected to the ECG transducer (M9407, Medikro Oy, Finland). Continuous arterial blood pressure signal was registered with an Ohmeda 2300 NIBP Monitor (Ohmeda, USA). A finger cuff of appropriate size was wrapped around the second phalanx of either the left or the right middle finger. During the study, this finger was carefully kept at heart level. A controlled, computer-generated signal was used to pace breathing at the rate of 0.20 Hz (2.5 s for both inspiration and expiration).

The analogue outputs of the ECG transducer and the Finapres device were connected to the analogue interface (M9401 Transducer Interface, Medikro Oy, Finland) of an IBM PC/AT compatible microcomputer. The microcomputer was equipped with a software package (CAFTS, Medikro Oy, Finland, Tahvanainen et al., 1992) for evaluating heart rate and blood pressure variabilities in time and frequency domains. Both the ECG and the blood pressure signal were analogue-to-digital converted (200 Hz, 12 bits) and saved on the hard disk for subsequent off-line analysis. The R–R intervals (RRIs) were detected using a temporal resolution of better than 2 ms. The RRI detection was followed by evaluation of systolic (SAP) and diastolic (DAP) arterial pressure in each RRI. The following variables were calculated in the time domain from the 5-min supine rest measurement: mean ± 1 SD of RRI, HR, SAP and DAP and
the root mean square of successive RRI (RRI RMSSD). The regions of interest were selected by excluding any ectopic beats and by visual judgement of stationarity.

The same time series of RRI, SAP and DAP were subjected to power spectral density analysis in the frequency domain. Modified covariance autoregressive modelling with a fixed model order of 18 was used for spectral analysis. Total powers of RRI, SAP and DAP variability (RRI TP, SAP TP and DAP TP) were generated after linear detrending of the signals. The powers of RRI, SAP and DAP variability in the three frequency bands [very low-frequency band = 0.00 - 0.07 Hz (VLFP), low frequency band = 0.07 - 0.15 Hz (LFP) and high-frequency band = 0.15 - 0.40 Hz (HFP)] were calculated by integration. RRI HFP and RRI LFP were also analysed as normalized units (HFPnu = HFP/HFP + LFP and LFPnu = LFP/HFP + LFP) and expressed as a percentage. Sympathovagal balance was estimated by dividing the RRI, SAP and DAP power in the LF band by that in the HF band and expressed as a percentage (LFPIHFP).

Measurement of 

Phenylephrine test and baroreflex sensitivity

In the morning of the second day (the instructions were the same as on the previous day), a catheter was inserted into the antecubital vein of the athletes after they had assumed a supine position. The athletes rested for 20 min, after which three consecutive phenylephrine tests (Smyth et al., 1963) of 5-min duration were performed by injecting i.v. 150 μg of phenylephrine hydrochloride (Laboratories Winthrop, Belgium) by bolus technique. After the phenylephrine bolus, the catheter was immediately flushed with 5 ml of saline (Natrosteril 0.9%, Medi-polar, Orion Oy, Finland). Heart rate and blood pressure and their variabilities were measured and analysed using the same devices and methods as on the first day.

The interbeat intervals were regressed against increasing SAP measured at the respective preceding beat on a beat-to-beat basis. The slope of the linear portion of the relationship for each individual was taken as baroreflex sensitivity. Only statistically significant phenylephrine tests were used in the analysis.

ventilation (VE), \( \text{O}_2 \) uptake and \( \text{CO}_2 \) production were measured during the exercise test at 20-s intervals using a Sensormedics 2900es gas analyser (Sensormedics, USA) connected on-line to a computer and calibrated before and after each test. \( \text{VO}_{2\text{max}} \) was calculated as the mean of the three highest consecutive 20-s determinations. Maximal treadmill performance was calculated as the oxygen demand of exercise (\( \text{VO}_{2\text{max, demand}} \)) during the last minute before exhaustion based on the velocity and the inclination of the treadmill at maximal work rate (Balke & Ware, 1959).

The lactate threshold was defined as the starting point of blood lactate increase over the initial steady level (1-2 mmol l\(^{-1}\)) observed at the lowest exercise intensities and was confirmed by the first non-linearity in the VE/VO\(_2\) ratio corresponding to the 'anaerobic threshold' (Aunola & Rusko, 1986; Wasserman et al., 1973).
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Statistics
The distribution of each variable was calculated. All variables were normally distributed, with the exception of RRI and SAP LFP/HFP. However, because of the small number of subjects non-parametric tests (Friedman two-way ANOVA, Wilcoxon matched-pairs signed-rank test, Mann–Whitney U-Wilcoxon rank sum W-test, Spearman correlation coefficients) were used to analyse the data (SPSS release 6.1).

The results in the text are expressed as means ±SEM [95% confidence intervals (CI)]. A P-value of 0.05 was used as a critical level of significance.

Results
Training
The reasons for interrupting the heavy training were feelings of psychological and physical exhaustion in seven athletes, leg injury in one athlete and fever in another. Total training volume of the ETG increased by 80% (P < 0.05), the volume of LIT by 98% (P < 0.01) and the volume of IT by 130% (P < 0.01) from the first training week to the END. Their strength training volume decreased by 54%. The corresponding increases in the CG were 6%, 5%, 10% and 21%. Total and LIT training volumes were higher on the first (P < 0.05) and the second (P < 0.01) training week in the ETG than in the CG. Strength training volume was higher (P < 0.05) on the second, fifth, sixth and the last training week in the ETG than in the CG. The training of the OA subgroup did not differ from the training of the other ETG athletes.

Relative changes in plasma volume
The individual changes are shown in Fig. 1a. There were no significant group differences in plasma volume changes during the training period.

Baroreflex sensitivity
There were no significant changes in baroreflex sensitivity (Fig. 1b), phenylephrine-induced changes in heart rate (ETG −34 b.p.m. and −26 b.p.m.; CG −25 b.p.m. and −28 b.p.m. at baseline and at the END respectively) or systolic arterial blood pressure (ETG 16 mmHg and 17 mmHg; CG 19 mmHg and 17 mmHg at baseline and at the END respectively). The ETG had higher BRS (24.3 ± 11.2 ms mmHg⁻¹, P < 0.01) and a greater HR response (P < 0.05) to the phenylephrine bolus at baseline than the CG.

Resting supine heart rate and blood pressure variability
RRI LFP in the ETG increased during the training period (P < 0.05, Table 2). Blood pressure variability did not change in either of the groups or in the OA subgroup (Table 3). The ETG had lower RRI SD (P < 0.05), RRI TP (P < 0.05) and RRI LFP (P < 0.01) values than the CG after 4 weeks of training. There were great interindividual differences in the HRV and BPV variables, even between overtrained athletes.

Treadmill test
Maximal oxygen demand (VO₂max demand, P = 0.05) and maximal heart rate (HRmax, P = 0.01) decreased in the ETG during the training period. Their VO₂max demand was 52 ± 2 (48–56) ml kg⁻¹ min⁻¹ at baseline, 53 ± 1 (50–56) ml kg⁻¹ min⁻¹ after 4 weeks of training and decreased to 51 ± 1 (48–54) ml kg⁻¹ min⁻¹ at the END (P < 0.05). The respective HRmax values were 190 ± 4 (186–200) b.p.m., 192 ± 3 (179–201) b.p.m. and 189 ± 4 (180–199) b.p.m. HRmax increased (P < 0.05) to 195 ± 3 (188–203) b.p.m. after the recovery period. The body mass of the ETG decreased from 63 ± 2 kg at baseline to 62 ± 2 kg (P < 0.05) at the END.

VO₂max, VO₂max demand and HRmax decreased in the OA subgroup (P < 0.05). VO₂max decreased from 53 ± 2 (47 to 59) ml kg⁻¹ min⁻¹ at baseline to 50 ± 2 (44–57) ml kg⁻¹ min⁻¹ at the END (P < 0.05). The decreases in VO₂max demand and HRmax were from 56 ± 2 (51–60) ml kg⁻¹ min⁻¹ to 52 ± 1 (49–55) ml kg⁻¹ min⁻¹ (P < 0.05) and from 190 ± 1 b.p.m. to 186 ± 2 b.p.m. (P < 0.05) respectively. The body mass of the OA subgroup decreased from 67 ± 2 kg (62–71) kg at baseline to 65 ± 1 (62–68) kg at the END and increased back to the baseline level after the recovery (P < 0.05). Peak blood lactate did not change in any group, although it
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Correlation analyses

No significant correlations were found between the changes in different variables.

The effect of the menstrual cycle on the BRS, HRV and BPV results could not be controlled, but it may have had some influence on the results. The athletes were in different menstrual phases at baseline [eight in follicular phase, six in luteal phase and one in between (day 15)] and had no menstrual irregularities. Six athletes (five ETG and one CG) had late periods during the experimental period. BRS, HRV and BPV were not systematically higher in any of the phases.

Discussion

The main finding of our study was that there were no endurance training induced changes in the female athletes’ arterial baroreflex sensitivity and in the responses of heart rate and blood pressure to phenylephrine bolus during the experimental period. This is consistent with the previous study of Sheldahl et al. (1994), in which the phenylephrine test was also used to measure BRS. In that study the subjects were
middle-aged and aged sedentary men. There are very few longitudinal training studies concerning the baroreflex control of very fit humans (e.g. Mack et al., 1993; cardiopulmonary BRS decreased during a 10-week training period), which is obviously different from sedentary subjects. It could be assumed that because of their changed baseline values, the changes in the BRS of very fit humans are not very big, even if their training volume and intensity increased excessively. However, we found that the CG had the lower BRS and HR response to phenylephrine at baseline than the ETG. This was the case even if both groups included experienced athletes and their VO2max was similar. This suggests that the CG athletes had greater long-term training volumes than the ETG, and that more than 6-9 weeks of training is needed to induce changes in the BRS of endurance athletes. Another reason for the low BRS in the CG could be an inherited feature of having attenuated responses. The attenuated BRS in the CG resulted mainly from the attenuated HR response, which is consistent with previous findings of training-induced BRS changes in very fit athletes (Shi et al., 1993b). The attenuated HR response could be caused by decreased baroreceptor sensitivity or density and afferent activity from the arterial baroreceptors, by attenuated central integration of afferent activity and/or by attenuated ability of the sinus node to respond to efferent vagal impulses. Chen et al. (1995) have suggested that reduced central gain of the arterial baroreflex regulation of heart rate is responsible for the decreased HR response to phenylephrine injection in trained rats, without changes in the reactivity of baroreceptor afferents.

It is well justified to study baroreflex sensitivity changes induced by endurance training and over-training because endurance training has been reported to increase vagal tone and maximal oxygen uptake (Seals & Chase, 1989), which are correlates of baroreflex sensitivity (Sleight et al., 1995). We found that RRI LFP increased in the ETG during the training period as a possible marker of increased cardiac sympathetic modulation at rest due to excessive training (Akselrod et al., 1985; Pagani et al., 1986). During the first four training weeks, the HRV had a tendency to decrease in the ETG and to increase in the CG, which suggests that excessive training (including significant increases in training volume) and normal training might cause different changes in the HRV (but not yet in maximal performance on the treadmill). This could indicate that a decreased HRV may be a sign of impending fatigue.

Increased plasma volume is supposed to be a reason for decreased baroreflex sensitivity in endurance athletes (Mack et al., 1991). It did not correlate with BRS changes in this study. The relative change in plasma volume was 7% (range from 1% to +20%) in the ETG and 3% (range from 2% to +12%) in the CG. These changes are consistent with the study of Mack et al. (1993), in which blood volume increased by 8% in unfit sedentary men during 10 weeks of training and by 2% in control men. The fact that the change in plasma volume was calculated based on the haemoglobin concentration and haematocrit makes it somewhat uncertain, although this formula has been reported to give nearly identical results of plasma volume expansion as the method using 125I-labelled human serum albumin (Green et al., 1991, McDonald et al., 1993). The other hypothesized reasons for

Table 3 Blood pressure and blood pressure variability at supine rest at baseline.

<table>
<thead>
<tr>
<th>Variable</th>
<th>ETG</th>
<th>CG</th>
</tr>
</thead>
<tbody>
<tr>
<td>SAP (mmHg)</td>
<td>113 ± 4</td>
<td>108 ± 7</td>
</tr>
<tr>
<td>(105-122)</td>
<td>(91-126)</td>
<td></td>
</tr>
<tr>
<td>DAP (mmHg)</td>
<td>52 ± 2</td>
<td>52 ± 6</td>
</tr>
<tr>
<td>(47-57)</td>
<td>(37-57)</td>
<td></td>
</tr>
<tr>
<td>SAP TP (mmHg²)</td>
<td>26.41 ± 8.47</td>
<td>31.55 ± 7.21</td>
</tr>
<tr>
<td>(11.10-41.72)</td>
<td>(13.02-50.08)</td>
<td></td>
</tr>
<tr>
<td>SAP VLFP (mmHg²)</td>
<td>20.52 ± 5.15</td>
<td>26.68 ± 5.78</td>
</tr>
<tr>
<td>(8.16-32.49)</td>
<td>(4.34-30.82)</td>
<td></td>
</tr>
<tr>
<td>SAP LFP (mmHg²)</td>
<td>3.71 ± 1.42</td>
<td>2.88 ± 0.95</td>
</tr>
<tr>
<td>(0.36-7.06)</td>
<td>(0.43-5.33)</td>
<td></td>
</tr>
<tr>
<td>SAP HFP (mmHg²)</td>
<td>2.31 ± 0.42</td>
<td>2.95 ± 0.72</td>
</tr>
<tr>
<td>(1.33-3.30)</td>
<td>(1.10-4.80)</td>
<td></td>
</tr>
<tr>
<td>SAP LFP/HFP (%)</td>
<td>173 ± 62</td>
<td>90 ± 17</td>
</tr>
<tr>
<td>(27-320)</td>
<td>(42-137)</td>
<td></td>
</tr>
<tr>
<td>DAP TP (mmHg²)</td>
<td>8.10 ± 1.58</td>
<td>7.08 ± 1.16</td>
</tr>
<tr>
<td>(4.37-11.83)</td>
<td>(4.11-10.06)</td>
<td></td>
</tr>
<tr>
<td>DAP VLFP (mmHg²)</td>
<td>5.74 ± 1.32</td>
<td>5.33 ± 0.98</td>
</tr>
<tr>
<td>(2.61-8.87)</td>
<td>(3.05-8.47)</td>
<td></td>
</tr>
<tr>
<td>DAP LFP (mmHg²)</td>
<td>1.31 ± 0.45</td>
<td>1.02 ± 0.21</td>
</tr>
<tr>
<td>(0.25-2.37)</td>
<td>(0.47-1.57)</td>
<td></td>
</tr>
<tr>
<td>DAP HFP (mmHg²)</td>
<td>1.01 ± 0.28</td>
<td>0.70 ± 0.17</td>
</tr>
<tr>
<td>(0.34-1.68)</td>
<td>(0.26-1.14)</td>
<td></td>
</tr>
<tr>
<td>DAP LFP/HFP (%)</td>
<td>178 ± 61</td>
<td>188 ± 73</td>
</tr>
<tr>
<td>(33-323)</td>
<td>(1-375)</td>
<td></td>
</tr>
</tbody>
</table>

SAP, systolic arterial pressure; DAP, diastolic arterial pressure. For other abbreviations see Table 2.
Training-induced changes in BRS, including changes in vascular and ventricular compliance, baroreceptor density and cardiac hypertrophy could not be detected in this study. However, changes in cardiac size are possible because cardiac hypertrophy has been reported to take place after 6 weeks of endurance training (Shapiro & Smith, 1983).

The present study is unique because there are no previous studies on overtraining and BRS or longitudinal training studies on training-induced changes in BRS in female athletes. The subjects in the previous studies have been mainly untrained men. It has been suggested that women have lower BRS values than men (Abdel-Rahaman et al., 1994). In women the effect of menstruation should be taken into consideration. This effect could not be completely excluded.

The biggest limitation of the study was the small number of subjects, which is inevitable in controlled training studies on athletes, but can be one reason for the non-significant changes.

Another important reason for the non-significant changes was great individual variability. Different types of baroreceptor responsiveness have previously been reported in very fit subjects (Savard & Lundie, 1995). The present study concentrated on resting supine values. It could be hypothesized that changes in the regulation of haemodynamics resulting from endurance training and overtraining could be more obvious during exercise and during postural changes when several different components of regulation are involved in the adaptation to new requirements (Wieling, 1988; O'Leary & Seamans, 1993).

The difficulty with all overtraining studies, including this one, is the diagnosis of overtraining because there are no consistent criteria for the overtraining state. We determined our criteria based on previous knowledge of overtraining. The most important criteria were decrements in maximal oxygen uptake and maximal performance on the treadmill. Some ETG athletes did not meet all of the criteria because their reasons for interrupting heavy training were local overstrain (leg injury), immunological deficit (illness) and mental but not physical fatigue. This suggests that there are different ways of responding to high training loads (training-induced stress). Loss of body mass in the ETG and the OA subgroup, probably due to increased catabolism of glycogen and concomitant hypovolaemia, could have been one reason for the slight decrease in VO2max kg⁻¹ in some ETG athletes. Consequently, the present criteria underdiagnosed rather than overdiagnosed overtraining state.

Conclusion

We did not find significant changes in arterial baroreflex sensitivity during the heavy endurance training period or in the overtraining state in female athletes. The low-frequency power of R-R interval variability increased in the experimental group as an indicator of increased cardiac sympathetic modulation at rest and as a result of a significant increase in training volume and intensity over a short period of time.

Acknowledgements

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