Heart Rate Variability, Blood Pressure Variability, and Baroreflex Sensitivity in Overtrained Athletes

Mathias Baumert, PhD,†‡ Lars Brechtel, MD,§ Jürgen Lock,¶ Mario Hermsdorf,§ Roland Wolff, MD,§ Vico Baier,* and Andreas Voss, PhD**

Objective: To assess the effects of abruptly intensified physical training on cardiovascular control.

Design: Retrospective longitudinal study.

Setting: Research laboratory.

Participants: Ten healthy athletes (5 men and 5 women) from track and field as well as triathlon.

Interventions: A 2-week training camp, including daily stepwise increasing cycling tests, running of 40 minutes, and additional cycling of 60 minutes.

Main Outcome Measurements: Time and frequency domain parameters of resting heart rate and blood pressure variability (HRV and BPV) and baroreflex sensitivity (BRS), before, during, and after the training camp.

Results: We found significantly reduced HRV during the training camp (mean beat-to-beat interval: 1042 [937 to 1194] ms vs. 933 [832 to 1103] ms vs. 1055 [947 to 1183] ms, $P < 0.01$; root-mean-square of beat-to-beat interval differences: 68 [52 to 95] ms vs. 52 [38 to 71] ms vs. 61 [48 to 78] ms, $P < 0.05$). Further, BRS was significantly reduced: 25.2 (20.4 to 40.4) ms/mmHg vs. 17.0 (12.9 to 25.7) ms/mmHg vs. 25.7 (18.8 to 29.1) ms/mmHg, $P < 0.05$. These effects disappeared at a large degree after 3 to 4 days of recovery.

Conclusion: Abruptly intensified physical training results in an altered autonomic cardiovascular activity towards parasympathetic inhibition and sympathetic activation that can be monitored by means of HRV and BRS analyses and might provide useful markers to avoid the overtraining syndrome.

Key Words: overtraining, autonomic cardiovascular control, heart rate variability, blood pressure variability, baroreflex sensitivity

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From the *Department of Medical Engineering, University of Applied Sciences Jena, Germany, §Institute for Sports, Humboldt University, Berlin, Germany; ¶*real*,- BERLIN-MARATHON, Berlin, Germany; †Centre for Biomedical Engineering, The University of Adelaide, Australia; and ‡School of Electrical & Electronic Engineering, The University of Adelaide, Australia.

Reprints: Mathias Baumert, The University of Adelaide, School of Electrical & Electronic Engineering, SA 5005, Australia (email: mbaumert@eleceng.adelaide.edu.au).

INTRODUCTION

Physical training has shown to influence autonomic cardiovascular control. Moderate endurance training increases parasympathetic activity and decreases sympathetic activity in the human heart at rest, which is considered a cardioprotective effect against lethal arrhythmias.[1,2] Overtraining, however, is thought to influence autonomic cardiovascular control negatively.[3,4] The overtraining syndrome or chronic fatigue, burnout, and staleness, respectively, remain a serious problem in athletes. It was estimated that 65% of national and international elite runners suffer from the overtraining syndrome at least once in their career,[5] but recreational athletes are also affected.[6,7] The overtraining syndrome is considered an imbalance between training and competition versus recovery. Budgett defined the overtraining syndrome as a condition of fatigue and underperformance,[8] often associated with frequent infections and depression that occur after hard training and competition. Overreaching, ie, short-term overtraining, resolves within 2 weeks of adequate rest and must be distinguished from long-term overtraining and can be seen as a normal part of athletic training or peaking for performance. The transition from overreaching to overtraining is blurred.[9] The overtraining syndrome has been divided into a sympathetic and a parasympathetic manifestation, and it was hypothesized that the underlying pathophysiological mechanisms include alterations of the endocrine and autonomous nervous system.[10,11] It is thought that the sympathetic form is dominant in the early stages of overtraining. In more advanced stages, the sympathetic system is inhibited, resulting in marked dominance of the parasympathetic system. This hypothesis is more or less based on the different patterns of observed symptoms,[12,13] as well as on data from parameters like increased plasma catecholamine concentrations at rest,[14] an inadequate increase during exercise and decreased nocturnal catecholamine excretions.[15] Since there is no diagnostic test available to detect overtraining, it is important to screen the athlete to exclude other causes of chronic fatigue.

This study was conducted to assess the impact of increased training in the framework of a 2-week training camp on autonomic cardiovascular control in athletes. We hypothesized that the monitoring of heart rate and blood pressure variability (HRV and BPV) and baroreflex sensitivity (BRS) is suitable to detect autonomic changes due to abrupt increases in the training amount and might consequently be used as markers for the detection of overtraining.

HRV and BPV refer to the beat-to-beat fluctuations of heart rate and blood pressure, respectively, and BRS measures

heart rate adaptations to rapid blood pressure changes. HRV, BPV, and BRS reflect regulatory processes in the cardiovascular system and therefore allow the assessment of cardiac and vascular autonomic control. Short-term blood pressure regulation is mainly accomplished by neural sympathetic-mediated and parasympathetic-mediated cardiac baroreflexes and peripheral vessel resistance, whereas long-term regulation is achieved by hormonal pathways such as the renin-angiotensin system, body temperature, and other systems.[16] BPV, BRS, and HRV have proven to be independent predictors for sudden cardiac death after acute myocardial infarction,[17] chronic heart failure,[18] or dilated cardiomyopathy.[19,20]

METHODS

Data and Preprocessing

Ten healthy experienced athletes (5 men and 5 women from track and field, middle and long distance running, as well as triathlon with competition experience for more than 2 years) participated in this study during a training camp. The results of the medical examination (medical history, physical examination, and graded cardiopulmonary exercise test including ECG) were negative. No athlete was on medication before or during the study. All subjects were fully recovered from the competition season (subjective reports during the medical examination as well as normal profile and values in the "Profile of Mood States" questionnaire).[19–20] Anthropometric data and peak oxygen uptake (measured during the below described stepwise increasing cycling until subjective exhaustion; K4b2 COSMED S.r.l., Rome, Italy) are shown in Table 1.

To assess autonomic cardiovascular control, high resolution ECG (1600 Hz) and noninvasive continuous blood pressure (Portapres M2, TNO Biomedical Instrumentation, The Netherlands[21]) were simultaneously recorded over 30 minutes in supine position under standardized resting conditions. For HRV and BPV analyses and BRS estimation, respectively, time series of beat-to-beat intervals and systolic blood pressure were extracted. Artifacts and ectopic beats were rejected and interpolated.

The investigation conforms to the principles outlined in the Declaration of Helsinki. Written informed consent of all athletes has been provided. The subjects in the study were recruited from a bigger study of heart rate variability in relation to the menstrual cycle in trained and untrained women that has been approved by an ethics committee.

TABLE 1. Anthropometric Data and Peak Oxygen Uptake of the Investigated Athletes Presented as Medians and Interquartile Ranges (IQR)

	Women		Men	
Sex	**Median**	**IQR**	**Median**	**IQR**
Age [years]	24.8	24.7–26.4	26.6	26.5–28.8
Body mass [kg]	54.8	50.4–61.8	72.0	69.0–86.8
Height [cm]	163	162–168	181	181–182
VO_2 peak ml/[kg*min]	51.1	48.9–52.2	65.9	61.4–74.6

Test Protocol

The training camp started after 1 day of traveling and acclimatization and lasted 13 days, with 1 day of rest after 6 days of training. Training, sleep, and mood state (see below) were monitored by a log and by recordings of the investigators. A typical daily training program included a stepwise increasing cycling test and additional running of 40 minutes and cycling of 80 minutes (Table 2). Parameters of training and performance capacity were the exercise amount in minutes, peak performance in watts, and maximal heart rate. The step tests were carried out in an exercise lab using 2 calibrated cycle ergometers 1 week before the training camp (M1), after 1 week of training (M2), and 3 to 4 days after the training period (M3). The step test started at 50 Watts for female and 100 Watts for male athletes. The load was increased every 3 minutes by 50 watts until subjective exhaustion. Objective criteria of exhaustion were taken from online monitoring of respiratory data (respiratory ratio >1.11 or leveling of oxygen uptake). Parallel continuous recordings of heart rate were carried out by ECG monitoring. Running and cycling endurance exercises took place in the afternoon and were carried out individually or in small groups of nearly equal performance capacity. Training amount and intensity (85 to 90 percent of the individual anaerobic threshold as described by Stegmann et al.[22]) were above the normal activity level the athletes are adapted to. After returning home, the athletes recovered for 3 to 4 days. In this period no (1 to 2 days) or maximal 30 minutes regenerative training of very low intensity was carried out (intensity at 70 percent of the individual anaerobic threshold). All of the above mentioned exercise was registered in a training log.

Supine heart rate and blood pressure recordings for assessing autonomic control took place 1 week before training (M1), after 1 week of training (M2), and 3 to 4 days after the training period (M3).

Before the measurements, the athletes completed the "Profile of Mood States" questionnaire.[23] The German version contains 35 items and it yields measures of depression, anger, vigor and fatigue.[24] The global score of mood was calculated in relationship to the use in a sports setting as described by Morgan et al.[25,26]

Heart Rate and Blood Pressure Variability Analysis

A parameter set of time and frequency domain measures was calculated according to specifications by the Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology.[27] Frequency domain

TABLE 2. Daily Sleep Duration and Training Amount Presented as Medians and Interquartile Ranges (IQR)

	Median	IQR
Sleep per day [hours]	7.5	7.0–8.0
Training amount – running [minutes]	40	40–40
Training amount – cycling [minutes]	80	80–95
Total training amount [minutes]	170	150–190

analysis was performed on linear equidistantly interpolated time series with a resolution of 500 ms applying Fast Fourier Transform and a Blackman-Harris window function. The following parameters were computed:

1. meanNN / smeanNN / dmeanNN–Mean of all normal beat-to-beat intervals / systolic / diastolic blood pressure values; in ms / mmHg
2. sdNN / bsdNN – standard deviation of all normal beat-to-beat intervals / systolic blood pressure values; in ms / mmHg
3. rmssd / brmssd – Square root of the mean of the summed squared differences between adjacent beat-to-beat intervals / systolic blood pressure values; in ms / mmHg
4. LF / bLF–Power in the low frequency band (0.04 to 0.15 Hz); in ms^2 / $mmHg^2$
5. HF / bHF–Power in the high frequency band (0.15 to 0.4 Hz); in ms^2 / $mmHg^2$
6. LFn–Normalized low frequency power LF/(LF+HF) of HRV; in normalized units

Baroreflex Sensitivity

To assess spontaneous baroreflex, we applied the "sequence method."[28] In this way, systolic blood pressure time series are scanned for sequences of at least three 3 dropping systolic blood pressure values that are followed by a sequence of successively shortening beat-to-beat intervals with a delay of 1 heart beat. Subsequently, least square linear regression functions are fitted between these baroreflex sequences, and the average slope is used as an estimate of BRS.

Statistical Analyses

For statistical analysis of physical training effects, nonparametric statistics were computed, including median, interquartile ranges, and the nonparametric Friedman test for repeated measurements, using the SPSS software package. Alterations between M1 and M2 (test 1), M2 and M3 (test 2), and M1 and M3 (test 3) were tested post hoc for significance by using the Wilcoxon test for paired data. Values $P < 0.05$ were considered statistically significant. One blood pressure recording of M1 and two blood pressure recordings of M3 had to be excluded from the statistics due to cold-finger vasoconstriction. Consequently, BPV and BRS parameters of 2 women could not be considered in the Friedman test, and BPV and BRS parameters of 1 woman could not be included in the Wilcoxon tests.

RESULTS

Performance capacity dropped slightly but significantly during the training camp ($P < 0.05$) and returned slightly above the baseline levels after the recovery period ($P < 0.01$, Table 3). Although the overall score of mood state remained stable during the training course (Table 3), the profile of the subscales showed beginning pattern of a so-called inverted "iceberg profile" as described by Morgan et al[19] with a significant decrease of the T-Scores for the subscale "vigor" ($P < 0.05$), accompanied by an inverse but not statistically significant increase of "fatigue." The profile of mood states returned largely to baseline levels (M1) after returning from the training camp, with the exception of a further decrease of the subscale "vigor" ($P < 0.05$).

Numeric results of measures from heart rate, blood pressure, and baroreflex analysis are presented in Table 4. Bar plots of significantly altered parameters are shown in Fig. 1.

Heart Rate Variability

Compared with the first measurement, mean heart rate was significantly increased during training camp measurement and normalized until the recovery phase measurement (Fig. 1A). Contrarily, overall variability of heart rate (sdNN) was not significantly affected by the increased training. Beat-to-beat fluctuations of heart rate (rmssd) were, however, significantly reduced during the training camp measurement compared with the recovery phase measurement (Fig. 1B). Spectral analysis of HRV showed no significant alterations in the HF and LF bands.

Blood Pressure Variability

Mean systolic and diastolic blood pressure were not significantly altered between all 3 measurements. Increased training had also no significant influence on time domain parameters (bsdNN and brmssd) of BPV. Spectral analysis revealed also no significant changes during training camp compared with the first measurement and the recovery phase measurement.

TABLE 3. Peak Performance, Profile of Mood States (POMS) Before (M1), During (M2) and After Training Camp (M3) Presented as Medians and Interquartile Ranges (IQR)

	M1		M2		M3	
Measurement	**Median**	**IQR**	**Median**	**IQR**	**Median**	**IQR**
Peak performance [watts]*,#,†	301	219–350	279	217–317	309	234–338
POMS-depression	45	45–47	45.5	44–47	44	44–44
POMS-anger	47.5	44.5–57.5	47.5	44.5–57.5	44.5	44.5–50.5
POMS-vigor#,†	63.5	59–65	59.5	53–61.5	54.5	51–57
POMS-fatigue	52.5	47–61.5	64	45–69	49.5	42–58
POMS global score	93	83–102	103	83–125	88	83–103

*Significant changes between M1 and M2.
#Significant differences between M2 and M3.
†Significant changes between M1 and M3.

TABLE 4. HRV, BPV, and BRS Before (M1), During (M2) and After Training Camp (M3) Presented as Medians and Interquartile Ranges (IQR)

	M1		M2		M3	
Measurement	**Median**	**IQR**	**Median**	**IQR**	**Median**	**IQR**
HRV						
meanNN [ms]*,#	1042	937–1194	933	832–1103	1055	947–1183
sdNN [ms]	91	64–96	74	60–95	83	64–99
rmssd [ms]#	68	52–95	52	38–71	61	48–78
LF [ms^2]	226	80–632	211	85–443	139	60–226
HF [ms^2]	201	42–351	108	46–292	79	53–260
LFn [n.u.]	0.53	0.46–0.71	0.70	0.44–0.73	0.50	0.40–0.67
BPV						
smeanNN [mmHg]	118	115–129	114	108–120	127	113–130
dmeanNN [mmHg]	58	54–64	58	51–65	63	56–65
BsdNN [mmHg]	7.8	7.5–8.6	8.3	6.6–10.2	8.4	5.8–10.3
Brmssd [mmHg]	3.0	2.8–3.2	3.0	2.0–3.2	2.9	2.2–3.6
bLF [$mmHg^2$]	1.14	0.58–2.71	2.46	0.92–6.31	1.11	0.52–1.70
bHF [$mmHg^2$]	0.35	0.12–0.85	0.34	0.15–0.74	0.17	0.09–0.48
Baroreflex						
BRS [ms/mmHg]*,#	25.2	20.4–40.4	17.0	12.9–25.7	25.7	18.8–29.1

*Significant changes between M1 and M2.
#Significant differences between M2 and M3.
†Significant changes between M1 and M3.

Baroreflex Sensitivity

During the training camp measurement, the spontaneous baroreflex sensitivity was reduced compared with the first measurement and the recovery phase measurement (Fig. 1C).

DISCUSSION

We investigated effects of increased physical training on autonomic cardiovascular control in athletes. By monitoring HRV, BPV, and BRS, we aimed to derive markers for assessing training effects at the level of sympatho-vagal control, which might be useful to investigate the "overtraining syndrome." Our study design was completely noninvasive; therefore, the measurements were easy to obtain. However, this indirect approach hampers insight into vagal and sympathetic tones and rather gives estimates of cardiac and vascular activity mediated by vagal and sympathetic efferents. In particular, the Portapres measures peripheral blood pressure in the finger but not the central arterial pressure. Despite these limitations, HRV, BPV, and BRS analyses have been proven useful for the assessment of cardiovascular control.[21,27] In our study, supine HRV and BRS showed significant changes during a 2-week training camp.

A decreased peak performance in a standardized lab test and alterations of mood state are typical findings after an increase of training load.[25,26,29] A long-lasting drop of the sports-specific performance, even after 2 weeks of regeneration, was recommended to be essential for diagnosis of an

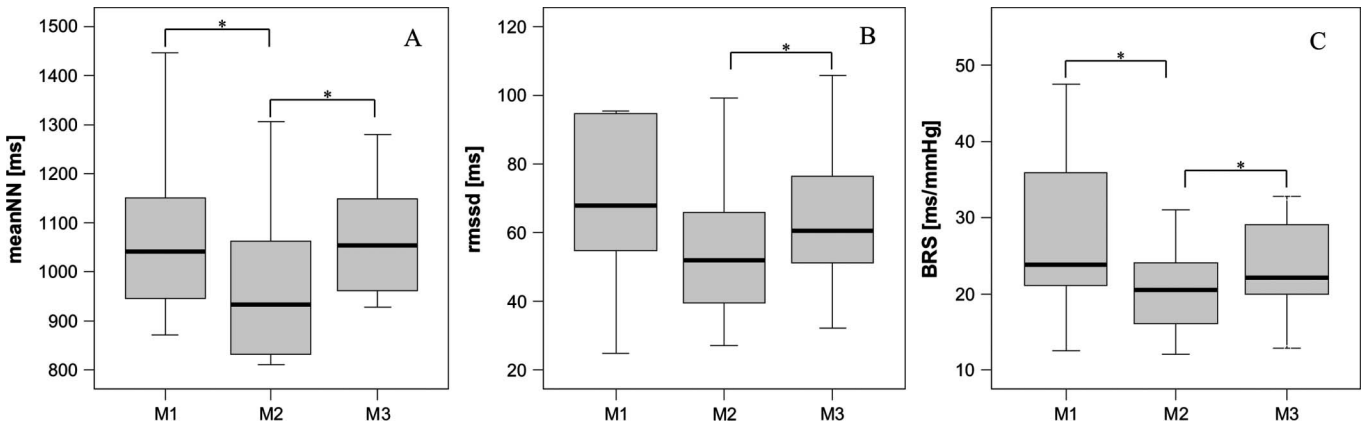

FIGURE 1. Bar plots of all heart rate and variability parameters and baroreflex sensitivity that are significantly altered in the Friedman test for repeated measurements. M1, measurement before training camp; M2, measurement during training camp; M3, measurement after recovery period. *Significant changes obtained with the Wilcoxon test for paired comparisons.

overtraining syndrome.[29] The decrease of performance and the inverse behavior of the score of mood state subscales "vigor" and "fatigue" during the training camp expressed only an insufficient regeneration of the investigated athletes in this study.[29] However, the general findings at the end (M3), a slightly enhanced performance and a nearly normal general mood state after 4 days of recovery, indicate that just a state of overreaching occurred, induced by a short-time overload followed by a minimal supercompensation.[30]

The increased heart rate (reduced meanNN) during the training camp, which recovered after 3 to 4 days of rest, clearly indicates an increased sympathetic activation due to increased training. The reduction of beat-to-beat fluctuations (rmssd), which are predominantly accomplished by vagal efferents,[27] indicates a shift of the sympatho-vagal balance toward sympathetic activity due to increased training amount. In agreement with other studies, we conclude that heavy training could increase cardiac sympathetic modulation during the supine position.[30] Interestingly, the changes in rmssd were significant only in test 2 (ie, between M2 and M3). We assume that the different individual performance capacity of the male participants before the training camp blurred the statistics, whilst the recovery phase clearly demonstrates the significant impact of the training camp. Supporting this assumption, a cross-sectional study by Mourot et al[31] found a reduced supine HRV in a group of 7 overtrained athletes compared with 8 trained athletes and untrained probands, suggesting also an increased sympathetic activity. However, a study from Bosquet et al[32] that observed overnight HRV in 9 endurance athletes during and 2 weeks after overtraining found no significant changes. A cross-sectional overtraining study of 12 athletes by Hynynen et al[33] found reduced parasympathetic heart rate control after awakening. Another study during a 6-day training camp has also not revealed significant changes in HRV.[34] Discrepancies in study results might partly arise from the different measurement conditions. For HRV analysis, quasi-stationary signal behaviour is essential.[27] Assessing the whole overnight HRV, for example, which is strongly assumed to contain nonstationary phases due to body movement and the like, might blur significant differences such as we have found under well-defined resting conditions. Also the duration and kind of the applied training amount might be different in the aforementioned studies and may result in different findings. Furthermore, high-frequency heart rate and blood pressure oscillations are caused directly and indirectly by respiration. Following different philosophies, some studies standardized this influence by forced breathing while others investigated the free-running condition. However, it has been shown that both approaches did not result in significant differences in HRV spectral indices.[35] For this study, we focused on free-running respiration, assessing overall changes in autonomic cardiovascular control, including cardiorespiratory changes.

A study of female endurance athletes has shown that progressively increased training load and overtraining did not induce significant changes in intrinsic heart rate or cardiac autonomic modulation.[36] Another study, investigating untrained, moderately trained, and heavily trained probands has found alterations in autonomic cardiac modulation only in moderately trained probands, but not in the heavily trained probands.[2] A study by Winsely et al[7] indicates that overreaching has a stronger effect on HRV in untrained women than in trained women. Thus, the abrupt change of load, such as in the training camp situation, seems to stimulate changes in autonomic control, whereas a progressively increasing load might trigger cardiovascular adaptation effects. Further, HRV depends on gender. Women have shown slightly decreased LF variability, and the parasympathetic heart rate control is thought to be stronger than in men.[37] In this study, however, we were not able to investigate gender effects due to small group sizes. Nevertheless, we assume that the training camp triggers similar autonomic responses in men as well as women, allowing the pooling of both genders in this first study, even though the basal levels might be different. Future investigations have to consider gender differences.

Systolic and diastolic blood pressure values were unaltered between all measurements. Also BPV was seemingly not affected by increased training. This is in accordance with a study of overtrained female athletes.[38] A study of elite runners during a training camp by Portier et al[39] has, however, shown that endurance training might lead to a decreased vasomotor activity.

BRS was also affected by the abrupt increase in training load, showing a significant reduction during training camp and recovery effects 3 to 4 days after training. Since the cardiac baroreflex is primarily mediated by vagal efferents,[28] this might also be interpreted as a shift in the sympatho-vagal balance toward sympathetic activation and vagal inhibition; however, an overtraining study of female athletes found no BRS changes.[38]

Our investigation indicates an increased sympathetic activity paralleled with a reduced vagal activity during periods of intensified training. Interestingly, this agrees with the assumption of an increased sympathetic activity in the first days or weeks during the development of an overtraining syndrome.[11] Further, we found that these effects are almost vanished after 3 to 4 days of recovery, suggesting that the 2-week training camp resulted just in overreaching, rather than an overtraining syndrome.

In conclusion, HRV and BRS reflect autonomic responses to increased training and therefore seem to be suitable markers for overreaching and might be useful also to study the overtraining syndrome. Limitations of our study are the small number of athletes, disallowing a gender-specific analysis and possibly reducing the statistical power of our analyses and furthermore the physical performance, varying between the male individuals. Future studies should therefore investigate the intraindividual relationships among physical performance, autonomic control, and blood-based measures of overtraining in large groups.

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REFERENCES

1. Carter JB, Banister EW, Blaber AP. Effect of endurance exercise on autonomic control of heart rate. *Sports Med.* 2003;33:33–46.
2. Buchheit M, Simon C, Piquard F, et al. Effects of increased training load on vagal-related indexes of heart rate variability: A novel sleep approach. *Am J Physiol Heart Circ Physiol.* 2004;287:H2813–2818.
3. Hedelin R, Wiklund U, Bjerle P, et al. Cardiac autonomic imbalance in an overtrained athlete. *Med Sci Sports Exerc.* 2000;32:1531–1533.
4. Iwasaki K, Zhang R, Zuckerman JH, et al. Dose-response relationship of the cardiovascular adaptation to endurance training in healthy adults: how much training for what benefit? *J Appl Physiol.* 2003;95:1575–1583.
5. Morgan WP, O'Connor PJ, Sparling PB, et al. Psychologic characterization of the female elite distance runner. *Int J Sports Med.* 1987;8:124–131.
6. Yates A, Leehey K, Slisslak CM. Running–an analogue of anorexia? *N Eng J Med.* 1983;308:251–255.
7. Winsley RJ, Battersby GL, Cockle HC. Heart rate variability assessment of overreaching in active and sedentary females. *Int J Sports Med*. 2005; 26:768–773.
8. Budgett R. Fatigue and underperformance in athletes: the overtrining syndrome. *Brit J Sports Med.* 1998;32:107–110.
9. Halson SL, Jeukendrup AE. Does overtraining exist? An analysis of overreaching and overtraining research. *Sports Med*. 2004;34:967–981.
10. Urhausen A, Gabriel H, Kindermann W. Impaired pituitary hormonal response to exhaustive exercise in overtrained endurance athletes. *Med Sci Sports Exerc*. 1998;30:407–414.
11. Lehmann M, Foster C, Dickhuth H, et al. Autonomic imbalance hypothesis and overtraining syndrome. *Med Sci Sports Exerc.* 1998;30: 1140–1145.
12. Brechtel LM, Braumann KM, Wolff R. Time course of symptoms during the development of a parasympathetic overtraining syndrome. *Med Sci Sports Exerc*. 1999;31:176.
13. Hooper S, Mackinnon L, Howard A, et al. Markers for monitoring overtraining and recovery. *Med Sci Sports Exerc*. 1995;27:106–112.
14. Lehmann M, Gastmann U, Peterson K, et al. Training-overtraining: performance, and hormone levels, after a defined increase in training volume vs intensity in experienced middle and long-distance runners. *Brit J Sports Med*. 1992;26:233–242.
15. Lehmann M, Schnee W, Scheu R, et al. Decreased noctural catecholamine excretion: parameter for an overtraining syndrome in athletes? *Int J Sports Med*. 1992;13:236–242.
16. Berntson GG, Bigger JT, Eckberg DL, et al. Heart rate variability: origins, methods, and interpretative caveats. *Psychophysiology.* 1997;34:623–648.
17. Kleiger RE, Miller JP, Bigger JT, et al. Decreased heart rate variability and its association with increased mortality after acute myocardial infarction. *Am J Cardiol*. 1987;59:256–262.
18. Tsuji H, Larson MG, Venditti FJ, et al. Impact on reduced heart rate variability on risk for cardiac events. *Circulation*. 1996;94:2850–2855.
19. Hofmann J, Grimm W, Menz V, et al. Heart rate variability and baroreflex sensitivity in idiopathic dilated cardiomyopathy. *Heart*. 2000;83:531–538.
20. Szabo BM, van Veldhuisen DJ, van der Veer N, et al. Prognostic value of heart rate variability in chronic congestive heart failure secondary to idiopathic or ischemic dilated cardiomyopathy. *Am J Cardiol*. 1997;79:978–980.
21. Imholz BP, Wieling W, van-Montfrans GA, et al. Fifteen years experience with finger arterial pressure monitoring: assessment of the technology. *Cardiovasc Res*. 1998;38:605–616.
22. Stegmann H, Kindermann W, Schnabel A. Lactate kinetics and individual anaerobic threshold. *Int J Sports Med*. 1981;2:160–165.
23 McNair DM, Lorr M, Droppelmann LF. Manual for the Profile of Mood states. Educational and Industrial Testing Service, San Diego, CA, 1971.
24. Bullinger M, Heinisch M, Ludwig M, et al. Skalen zur Erfassung des Wohlbefindes: Psychometrische Analysen zum'Profile of Mood States' (POMS) und zum 'Psychological Gerneral Well-Being Index' (PGWB). *Z Differentielle und Diganostische Psychologie*. 1990;11: 53–61.
25. Morgan WP, Brown DR, Raglin JS, et al. Psychological monitoring of overtraining and staleness. *Br J Sports Med*. 1987;21:107–114.
26. Morgan WP, Costill DL, Flynn MG, et al. Mood disturbance following increased training in swimmers. *Med Sci Sports Exerc*. 1988;20:408–414.
27. Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology. Heart rate variability. Standards of measurement, physiological interpretation, and clinical use. *Eur Heart J.* 1996;17:354–381.
28. Bertinieri G, di Rienzo M, Cavallazzi A, et al. A new approach to analysis of the arterial baroreflex. *J Hypertens Suppl*. 1985;3:79–81.
29. Kuipers H. Training and overtraining: an introduction. *Med Sci Sports Exerc*. 1998;30:1137–1139.
30. Uusitalo AL, Uusitalo AJ, Rusko HK. Heart rate and blood pressure variability during heavy training and overtraining in the female athlete. *Int J Sports Med*. 2000;21:45–53.
31. Mourot L, Bouhaddi M, Perrey S, et al. Decrease in heart rate variability with overtraining: assessment by the Poincare plot analysis. *Clin Physiol Funct Imaging*. 2004;24:10–18.
32. Bosquet L, Papelier Y, Leger L, et al. Night heart rate variability during overtraining in male endurance athletes. *J Sports Med Phys Fitness*. 2003; 43:506–512.
33. Hynynen E, Uusitalo A, Konttinen N, et al. Heart rate variability during night sleep and after awakening in overtrained athletes. *Med Sci Sports Exerc*. 2006;38:313–317.
34. Hedelin R, Kentta G, Wiklund U, et al. Short-term overtraining: effects on performance, circulatory responses, and heart rate variability. *Med Sci Sports Exerc*. 2000;32:1480–1484.
35. Patwardhan A, Evans J, Bruce E, et al. Heart rate variability during sympatho-excitatory challenges: comparison between spontaneous and metronomic breathing. *Integr Physiol Behav Sci*. 2001;36: 109–120.
36. Uusitalo AL, Uusitalo AJ, Rusko HK. Exhaustive endurance training for 6–9 weeks did not induce changes in intrinsic heart rate and cardiac autonomic modulation in female athletes. *Int J Sports Med*. 1998;19:532–540.
37. Jensen-Urstad K, Storck N, Bouvier F, et al. Heart rate variability in healthy subjects is related to age and gender. *Acta Physiol Scand*. 1997; 160:235–241.
38. Uusitalo AL, Uusitalo AJ, Rusko HK. Endurance training, overtraining and baroreflex sensitivity in female athletes. *Clin Physiol*. 1998;18:510–520.
39. Portier H, Louisy F, Laude D, et al. Intense endurance training on heart rate and blood pressure variability in runners. *Med Sci Sports Exerc*. 2001; 33:1120–1125.

