

Night and postexercise cardiac autonomic control in functional overreaching

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Abstract: The purpose of this study was to evaluate the effect of a 2-week overload period immediately followed by a 1-week taper period on the autonomic control of heart rate during the night or after exercise cessation. Eleven male endurance athletes increased their usual training volume by 100% for 2 weeks (overload) and decreased it by 50% for 1 week (taper). A maximal graded exercise test and a constant-speed test at 85% of peak treadmill speed, both followed by a 10-min passive recovery period, were performed at baseline and after each period. Heart rate variability was also measured during a 4-h period in the night or during estimated slow-wave sleep. All participants were considered to be overreached based on performance and physiological and psychological criteria. We found a decrease in cardiac parasympathetic control during slow-wave sleep (HFnu = $61.3\% \pm 11.7\%$ vs $50.0\% \pm 10.1\%$, $p < 0.05$) but not during the 4-h period, as well as a faster heart rate recovery following the maximal graded exercise test ($\tau = 61.8 \pm 14.5$ s vs 54.7 ± 9.0 s, $p < 0.05$) but not after the constant-speed test, after the overload period. There was a return to baseline for both measures after the taper period. Other indices of cardiac autonomic control were not altered by the overload period. Care should be taken in selecting the most sensitive heart rate measures in the follow-up of athletes, because cardiac autonomic control is not affected uniformly by overload training.

Key words: overreaching, overtraining, heart rate variability, heart rate recovery, parasympathetic reactivation.

Résumé : Cette étude se propose d'évaluer l'effet de deux semaines de surcharge immédiatement suivies d'une semaine d'affûtage sur le contrôle autonome du cœur mesuré durant la nuit ou après la fin de l'exercice. Onze athlètes d'endurance augmentent de 100 % durant deux semaines leur volume d'entraînement habituel (surcharge) et le diminuent de 50 % durant une semaine (affûtage). Avant et après chacune des périodes, les sujets participent à un test d'effort progressif jusqu'au maximum et à un test d'effort constant réalisé à 85 % de la vitesse maximale aérobie sur tapis roulant; les deux tests sont suivis de 10 min de récupération passive. On mesure aussi la variabilité de la fréquence cardiaque durant 4 h au cours de la nuit ou durant la période estimée de sommeil lent. À la lumière de critères physiologiques, psychologiques et de performance, les sujets sont considérés comme « surmenés ». Les résultats révèlent une diminution du contrôle parasympathique du cœur au cours du sommeil lent (HFnu = $61,3 \pm 11,7$ vs $50,0 \pm 10,1$ %, $p < 0,05$) sauf durant les 4 h de la nuit, une récupération plus rapide de la fréquence cardiaque après le test d'effort progressif ($\tau = 61,8 \pm 14,5$ vs $54,7 \pm 9,0$ s, $p < 0,05$), mais pas après le test à vitesse constante suivant la période de surcharge. Les deux mesures reprennent leur valeur de base après la période d'affûtage. Tous les autres indices du contrôle autonome du cœur ne sont pas affectés par la période de surcharge. On devrait sélectionner judicieusement les mesures les plus sensibles du rythme cardiaque pour le suivi de l'athlète, car le contrôle autonome du cœur n'est pas affecté uniformément par la surcharge à l'entraînement. [Traduit par la Rédaction]

Mots-clés : surcharge, surentraînement, variabilité de la fréquence cardiaque, récupération de la fréquence cardiaque, réactivation parasympathique.

Introduction

Functional overreaching (FOR) refers to an accumulation of training and (or) nontraining stress resulting in a transitory decrease in performance capacity (Meeusen et al. 2006). Athletes may recover quickly if the training load is adjusted to their level of fatigue and may eventually benefit from a supercompensation effect (Bosquet et al. 2007). In contrast, if they maintain the same high-volume or high-intensity training regimen, they may also experience a more severe state of overreaching, called nonfunctional overreaching (NFOR). In this case, the time required to fully recover is much longer (several weeks to several months), thus compromising their competitive season (Meeusen et al. 2006). Considering the fine line between these 2 states, early detection is

the cornerstone of any monitoring strategy. Unfortunately, the cause of FOR–NFOR is still poorly understood, and, to date, there is no pathognomonic marker that has been unanimously accepted by physicians and sport scientists to diagnose the disorder.

Heart rate is probably one of the most accessible physiological measures in sports medicine and is widely used in the monitoring of training load and performance capacity (Achten and Jeukendrup 2003). According to the model of Rosenblueth and Simeone (1934), heart rate and its modulation are determined primarily by the inotropic and chronotropic effects of the autonomic nervous system (ANS) on the myocardium and the sinus node. This is why heart rate variability (HRV) and postexercise heart rate recovery (HRR) are considered noninvasive measures of cardiac autonomic control (Buchheit et al. 2007). HRV is thought to reflect the interaction

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between the sympathetic and parasympathetic branches of the ANS (Task Force 1996), whereas HRR is thought to reflect the reactivation of the parasympathetic activity at the sinus node level and, to a lesser extent, cardiac sympathetic withdrawal (Savin et al. 1982; Kannankeril and Goldberger 2002). The scientific and clinical literature suggests that FOR and NFOR are concomitant with a dysfunction of the hypothalamus that may affect both neuronal and hormonal cardiac autonomic control (Israel 1958; Barron et al. 1985; Lehmann et al. 1998; Armstrong and VanHeest 2002), thus making HRV and HRR relevant monitoring tools.

Narrative reviews on this topic have underscored the lack of consistency of overload-induced alterations in HRV (Meeusen et al. 2006; Aubert et al. 2003; Achten and Jeukendrup 2003; Halson and Jeukendrup 2004). In their systematic review with meta-analysis, Bosquet et al. (2008) reported a moderate alteration in cardiac autonomic balance (i.e., the balance between parasympathetic and sympathetic control of the myocardium) after a short-term overload period (i.e., ≤ 2 weeks), whereas they found no modifications after overload periods longer than 2 weeks. Although HRV may therefore be considered a potential marker of short-term fatigue (possibly FOR), the moderate reliability of this measure (Sandercock 2007) outlines the need for a highly standardized protocol to detect such an effect. Because nighttime is generally free of events known to affect cardiac autonomic control, such as noise, light, or psychological stressors, Buchheit et al. (2004) assessed HRV by analyzing a stationary segment taken during a slow-wave sleep (SWS) episode. This novel sleep approach is interesting because it measures HRV in more standard conditions, thus making the interpretation of changes more relevant. This method has provided very interesting results in the monitoring of cardiac autonomic adaptation to normal training (Buchheit et al. 2004) but has never been used in the context of FOR–NFOR.

Postexercise HRR has long been recognized as an objective sign of fatigue by clinicians (Brown et al. 1983; Kereszty 1971). It is therefore not surprising to find it on the list of potential markers proposed by numerous narrative reviews (Fry et al. 1991; Hooper and MacKinnon 1995; Lehmann et al. 1993, 1997). Despite HRR's importance, a recent meta-analysis (Bosquet et al. 2008) and a review (Daanen et al. 2012) have not been able to support its validity as a possible marker of FOR–NFOR because of a lack of experimental data.

As such, the primary purpose of this study was to assess the effect of a 2-week overload period designed to induce a state of overreaching on cardiac autonomic control during the night or after exercise cessation. A secondary purpose was to compare the sensitivity of 2 different methods of assessing night HRV. Given the available literature, we hypothesized that a 2-week overload period would result in a cardiac sympathetic dominance during the night. The lack of published data did not enable us to propose a clearly stated hypothesis for the effect of overload on cardiac autonomic control after the cessation of exercise.

Materials and methods

Participants

Eleven male endurance athletes competing at a provincial-standard level participated in this study. We focused on males because cardiac autonomic control has been shown to be affected by menstrual cycle (Bai et al. 2009). Their mean \pm SD age, stature, and body mass were 29.5 ± 9.3 years, 177.0 ± 6.2 cm, and 71.6 ± 7.5 kg. All participants were nonsmokers. The protocol was reviewed and approved by the Research Ethics Board in Health Sciences of the University of Montreal (Canada), and the study was conducted in accordance with recognized ethical standards and national and international laws.

Experimental design

Following a thorough briefing, all participants signed a written statement of informed consent. Subsequently, they completed 3 experimental sessions, including a Profile of Mood State (POMS) questionnaire, a RESTQ-sport questionnaire, and a maximal continuous graded exercise test (session 1); a recording of R–R intervals during 1 night (session 2); and a constant-speed test (session 3). All sessions were separated by at least 48 h and were performed within a 7-day period, before and after a 2-week overload period consisting of a 100% increase in the baseline training volume (i.e., the training volume that was used by participants at the time of the study) and after a 1-week taper period consisting of a 50% decrease in the baseline training volume. To avoid any residual fatigue induced by recent workout, participants were asked to refrain from strenuous exercise the day before each session. Also, participants were asked to abstain from alcohol and caffeine-containing foods and beverages 24 h before the test, to avoid any influence on the autonomic control of the myocardium.

Profile of Mood States

The POMS (Mac Nair et al. 1971) is a 65-item Likert-format questionnaire that provides measures of 6 specific mood states: vigor, depression, fatigue, anger, anxiety, and confusion. These factors can also be combined to create composite measures of mood or fitness. The mood-state index was obtained by adding the 5 negative factors together and subtracting the positive factor of vigor. The energy index represented the difference between the scores of vigor and fatigue (Kentta et al. 2006).

RESTQ-sport questionnaire

The RESTQ-sport (Kellmann and Kallus 2001) is a 76-item Likert-format questionnaire that consists of 19 scales, of which 7 scales assess general stress, 5 scales assess general recovery, 3 scales assess sport-specific stress, and 4 scales assess sport-specific recovery. Each scale consists of 4 questions.

Maximal continuous graded exercise test

This test was performed on a motorized treadmill (Quinton, Bothell, Wash., USA), which was calibrated at 8 and 16 km·h⁻¹ (gradient = 0) before each session with an "in-house" system using an optical sensor connected to an acquisition card. The initial speed was set at 12 km·h⁻¹ and was increased by 1 km·h⁻¹ every 2 min until exhaustion. Less than 5 s after exercise cessation, participants sat on a chair for a 10-min passive recovery period and were asked to match their breathing frequency to an auditory metronome set at 0.2 Hz (12 breaths·min⁻¹) from the fifth to the 10th minute of the period. The grade was set at zero throughout the test. The speed of the last completed stage was considered to be the peak treadmill speed (PTS, in km·h⁻¹). Perceived exertion was assessed at the end of the test with the 10-point Borg scale (Borg 1982). Oxygen uptake ($\dot{V}O_2$) was determined continuously on a 15-s basis using an automated cardiopulmonary exercise system (Moxus, AEI Technologies, Naperville, Ill., USA). Gas analyzers (S3A and CD3A, AEI Technologies) were calibrated before each test using a gas mixture of known concentration (15% O₂ and 5% CO₂) and ambient air. Their accuracy was $\pm 0.003\%$ for oxygen and $\pm 0.02\%$ for carbon dioxide (data provided by the manufacturer). The turbine was calibrated before each test using a motorized syringe (Vacu-Med, Ventura, Calif., USA) with an accuracy of $\pm 1\%$ (Huszczuk et al. 1990). The tidal volume was set at 3 L and the stroke rate at 40 cycles per minute. The highest $\dot{V}O_2$ over a 15-s period during the test was considered to be peak oxygen uptake ($\dot{V}O_{2\text{ peak}}$, in mL·kg⁻¹·min⁻¹). Heart rate was measured continuously, beat by beat, using a heart rate monitor with a sampling frequency of 1000 Hz (S810, Polar Electro Oy, Kempele, Finland) and an accuracy of 0.3% during exercise (Kingsley et al. 2005). The highest heart rate (mean of 5 s) during the test was considered to be peak heart rate (HR_{peak}, in beats·min⁻¹).

Constant-speed exercise test

This test was performed on the same motorized treadmill as the maximal continuous graded exercise test. The instruction given to the participants was to maintain the required speed (85% of PTS) to the point of volitional exhaustion. The test was preceded by a standardized warm-up consisting of a 10-min run at a self-determined speed; a set of three 10-s repetitions at the speed of the test, interspersed by 1 min of passive recovery, to accustom themselves to the running speed; and a period of 5 min of passive recovery. The test began with the participant's feet astride the moving belt and hands holding the handrail. Time was measured to the nearest second from the moment the participant released the handrail (usually less than 3 s) until he grasped it again to signal exhaustion. Less than 5 s after exercise cessation, participants sat on a chair and followed the same procedure as that presented for the maximal continuous graded exercise test. R-R intervals were measured with the S810 (Polar Electro Oy). Perceived exertion was assessed at the end of the test with the 10-point Borg scale (Borg 1982). To increase the reliability of this test (Currell and Jeukendrup 2008), no verbal encouragement was given throughout the test, and participants were not informed about elapsed time.

Night cardiac autonomic control

R-R intervals were measured with the S810 (Polar Electro Oy). Its accuracy at rest is 0.4% (Gamelin et al. 2006). R-R intervals were edited and inspected visually so that ectopic beats could be replaced by interpolated data from adjacent normal-to-normal (N-N) intervals. In this study, all R-R series were free of ectopic beats. Two methods were compared to assess night HRV. During the first, night time was considered to be the 4-h period of sleep starting 30 min after reported bedtime (Myllymaki et al. 2011; Hynynen et al. 2010). The complete 4-h R-R series was retained for HRV analysis. During a second method, the first 10-min stationary segment in the first SWS episode lasting at least 15 min was retained for HRV analysis (Al Haddad et al. 2009; Brandenberger et al. 2005; Buchheit et al. 2004). The presence of an SWS episode was estimated according to the method of Brandenberger et al. (2005): (i) a lowest SD of N-N intervals (SDNN) when compared with other periods of the tachogram, or (ii) a round Poincaré plot that is characterized by almost equivalent SD of the instantaneous beat-to-beat variability data (SD1) and SD of the continuous long-term variability data (SD2). Mean HR, SDNN, and the root-mean-square difference of successive N-N intervals were calculated from the segment retained for HRV analysis. The same segment was resampled at 2 Hz and was detrended for subsequent analyses in the frequency domain. As recommended by the Task Force (1996), spectral analysis was performed with a Fast Fourier Transform to quantify the power spectral density of the low-frequency (LF; 0.04 to 0.15 Hz) and the high-frequency (HF; 0.16 to 0.40 Hz) bands. Additional calculations included LF+HF, LF and HF expressed in normalized units (i.e., in a percentage of LF+HF), and the LF/HF ratio. An analysis of the Poincaré plots was performed, and the following parameters were calculated as described by the Task Force. (1996): SD1 and SD2.

Postexercise cardiac autonomic control

R-R intervals were measured continuously during exercise and the 10-min passive recovery period using the same heart rate monitor (S810, Polar Electro Oy). R-R series were edited and inspected visually so that ectopic beats could be replaced by interpolated data from adjacent N-N intervals. In this study, all R-R series were free of ectopic beats. Several indices were used to characterize postexercise HRR during the 10-min passive recovery period: $\Delta 60$ (in beats·min⁻¹), T30 (in seconds), and the parameters of a monoexponential function. $\Delta 60$ was the absolute difference between the heart rate immediately at the end of the exercise (mean of 5 s) and after 60 s of passive recovery (mean of 5 s) (Cole et al. 1999). T30 was the negative reciprocal of the slope of the regres-

sion line between the natural logarithm of the heart rate and the elapsed time from the 10th to the 40th second after exercise (Imai et al. 1994; Buchheit et al. 2007). The overall kinetics of the heart rate during the 10-min transition from exercise to rest was described by the following monoexponential function:

$$[1] \quad \text{HR}(t) = (a_0 + a_1) \times e^{-t/\tau}$$

where a_0 is the asymptotic value of the heart rate (in beats·min⁻¹), a_1 is the decrement below the heart rate value at the end of the exercise for $t = \infty$ (in beats·min⁻¹), and τ is the time constant (i.e., the time needed to reach 63% of the gain, in seconds) (Perini et al. 1989; Dupuy et al. 2012). HRV was assessed in the time and frequency domains from the fifth to the tenth minute of the passive recovery period to warrant a stationary signal according to the recommendations made by the Task Force (1996).

Data analysis

Criteria for overreaching

A participant was considered as overreached when he fulfilled all the following criteria after the overload period: a decrease in physical performance evidenced by a decrease in PTS during the maximal continuous graded test or a decrease in time to exhaustion during the constant-speed test (Urhausen and Kindermann 2002); a decrease in HR_{peak} during the maximal continuous graded test (Bosquet et al. 2008); and psychological disturbances evidenced by a change in the energy index of the POMS (Kentta et al. 2006) and the recovery and stress scales of the RESTQ-sport (Kellmann 2010). Changes in these criteria after the taper period were used to assess the severity of overreaching. Short-term overreaching was characterized by a return to baseline after the taper period and could be assimilated to FOR; long-term overreaching was characterized by maintenance of observed alterations after the taper period and could be assimilated to NFOR (Meeusen et al. 2006).

Statistical analyses

Standard statistical methods were used for the calculation of means and SDs. Normal Gaussian distribution of the data was verified by the Shapiro–Wilk test. A 1-way within-group analysis of variance (ANOVA) was performed to test the null hypothesis that dependent variables were not affected by the overload or the taper periods. The compound symmetry, or sphericity, was checked by the Mauchly test. When the assumption of sphericity was not met, the significance of F ratios was adjusted according to the Greenhouse–Geisser procedure when the epsilon correction factor was <0.75 , or according to the Huynh–Feldt procedure when the epsilon correction factor was >0.75 (Vincent 2005). Multiple comparisons were made with the Newman–Keuls post hoc test. A paired Student t test was performed to test the null hypothesis that SDNN, SD1, and SD2 were similar between the 2 methods of night cardiac autonomic control assessment. The magnitude of differences was assessed by the Hedges' g (g), calculated as follows:

$$[2] \quad g = J \times d$$

where J is a correction factor calculated according to eq. [3] and d is Cohen's d , calculated according to eq. [4]:

$$[3] \quad J = 1 - \frac{3}{4df - 1}$$

where df represents the degrees of freedom ($df = n - 1$ in the case of dependent groups),

$$[4] \quad d = \frac{M_1 - M_2}{S_{\text{within}}}$$

where M_1 and M_2 are the means of the first and the second trials and S_{within} is the SD within groups, calculated as follows:

$$[5] \quad S_{\text{within}} = \frac{S_{\text{diff}}}{\sqrt{2(1-r)}}$$

where S_{diff} is the SD of differences between pairs and r is the correlation between pairs. Hedge's g was preferred to Cohen's d because the latter, in small samples, tends to overestimate the absolute value of the parameter (Hedge 1981). The scale proposed by Cohen (1988) was used for interpretation. The magnitude of the difference was considered either small ($0.2 < g \leq 0.5$), moderate ($0.5 < g \leq 0.8$), or large ($g > 0.8$). Statistical significance was set at $p < 0.05$ for all analyses.

Results

Physiological response

Mean responses during the maximal continuous graded exercise test are presented in Table 1. PTS and $\dot{V}O_{2\text{peak}}$ were not altered by the overload period. We found a moderate decrease in maximal heart rate ($p = 0.003$, $g = -0.72$), with a return to baseline after the taper period. Perceived exertion was not affected by the period.

We found a moderate decrease in time to exhaustion after the overload period (29.8 ± 9.3 min vs 22.1 ± 10.4 min, $p = 0.02$, $g = -0.71$), followed by a return to baseline after the taper period (31.0 ± 11.5 min, $p = 0.006$, $g = 0.75$). Perceived exertion was not affected by the period (8.6 ± 0.8 , 8.7 ± 0.7 , and 8.5 ± 1.1 after baseline, overload, and taper, respectively).

Psychological response

Mean results for the POMS are presented in Table 2. We found a large and systematic increase in the fatigue subscale after the overload period (41 ± 7 vs 51 ± 8 , $p = 0.0006$, $g = 1.33$), as well as a large and systematic decrease in the vigor subscale (61 ± 5 vs 51 ± 10 , $p = 0.001$, $g = -0.96$) and the energy index (121 ± 11 vs 100 ± 17 , $p = 0.0003$, $g = -1.29$). All these measures returned to baseline after the taper period.

Mean results for the RESTQ-sport are presented in Fig. 1. We found a small increase in the general stress subscale (1.2 ± 0.9 vs 1.7 ± 1.1 , $p = 0.00001$, $g = 0.34$) after the overload period, as well as a large increase in the sport-specific stress subscale (1.2 ± 1.3 vs 2.2 ± 1.5 , $p < 0.00001$, $g = 0.93$), a large decrease in the general recovery subscale (3.7 ± 1.1 vs 2.8 ± 1.1 , $p < 0.00001$, $g = -0.82$), and a large decrease in the sport-specific recovery subscale (4.1 ± 1.2 vs 2.8 ± 1.2 , $p < 0.00001$, $g = -1.60$). With the exception of the general stress subscale, all these measures returned to baseline after the taper period.

Night cardiac autonomic control

Mean time and frequency domain analyses during the 4-h night period and during the first 10-min SWS episode are presented in Table 3. SDNN and SD2 were systematically lower when they were measured during the SWS episode when compared with the 4-h period ($p < 0.05$, $1.19 < g < 1.91$). We also found a large difference between SD1 and SD2 when they were measured during the 4-h period ($p < 0.05$, $g = 1.43$), whereas there was no difference when they were measured during the SWS episode, thus confirming the presence of a round Poincaré plot. All together, these results fulfill the criteria proposed by Brandenberger et al. (2005) to ascertain that the 10-min segment was taken during a SWS episode.

HRV measures were not altered by the overload period when they were computed from the 4-h segment. In contrast, we found a moderate increase in LF/HF ($p = 0.01$, $g = 0.74$) and a large decrease in HFnu ($p = 0.01$, $g = -0.95$) after the overload period when HRV was measured during the first 10-min SWS episode, which were followed by a return to baseline after the taper period (Fig. 2).

Table 1. Acute response to the maximal continuous graded exercise test.

| Measure | Baseline | Overload | Taper |
|---|----------|--------------------|----------|
| $\dot{V}O_{2\text{peak}}$ (mL·min ⁻¹ ·kg ⁻¹) | 58.9±4.3 | 57.4±3.6 | 59.0±3.4 |
| PTS (km·h ⁻¹) | 17.2±1.3 | 17.0±1.3 | 17.3±1.3 |
| HR _{peak} (beats·min ⁻¹) | 186±9 | 179±8 ^a | 184±10 |
| RPE | 8.5±0.8 | 9.0±1.0 | 8.8±1.1 |

Note: Data presented as means ± SD. $\dot{V}O_{2\text{peak}}$, peak oxygen uptake; PTS, peak treadmill speed; HR_{peak}, peak heart rate; RPE, rating of perceived exertion.

^aDifferent from other values ($p < 0.05$).

Table 2. Subscales and composite scores of the POMS.

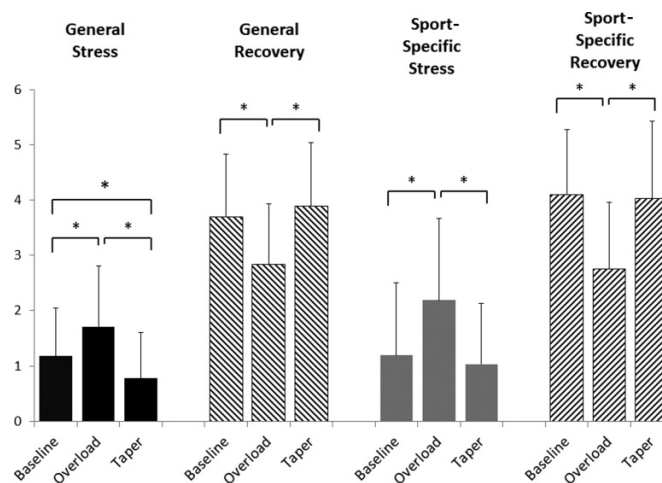
| | Baseline | Overload | Taper |
|--------------|----------|---------------------|---------------------|
| Vigor | 61±5 | 51±10 ^a | 61±7 ^b |
| Depression | 45±7 | 44±10 | 43±8 |
| Fatigue | 41±7 | 51±8 ^a | 39±7 ^b |
| Anger | 44±5 | 45±7 | 44±10 |
| Anxiety | 42±3 | 46±5 | 44±10 |
| Confusion | 45±5 | 47±8 | 44±7 |
| Global score | 110±21 | 124±26 | 107±32 |
| Energy index | 121±11 | 100±17 ^a | 122±14 ^b |

Note: Data presented as means ± SD. POMS, Profile of Mood State.

^aDifferent with baseline value, $p < 0.05$.

^bDifferent with overload value, $p < 0.05$.

Fig. 1. Composite scores of the RESTQ-sport at baseline, after overload, and after taper. *, Different, $p < 0.05$.



Postexercise cardiac autonomic control

Mean HRR and HRV measures during the postexercise passive recovery period are presented in Table 4 for both tests. We found no effect of the overload and taper periods on T30, $\Delta 60$, a_1 , and time or frequency domain indices of HRV, whatever the test. In contrast, we observed a small decrease in τ (61.8 ± 14.5 s vs 54.7 ± 9.8 s, $p = 0.036$, $g = -0.48$), as well as a large decrease in a_0 (100.5 ± 8.8 beats·min⁻¹ vs 93.1 ± 7.0 beats·min⁻¹, $p = 0.01$, $g = -0.82$) after the overload period during the recovery from the maximal graded exercise test, which was followed by a return to baseline after the taper period. It is worth noting that the decrease in a_0 was highly correlated to the decrease in HR_{peak} after the overload period ($r = 0.83$, $p < 0.05$). We also found a large decrease in a_0 during the recovery from the constant-speed test after the overload period (107.5 ± 4.8 beats·min⁻¹ vs 98.7 ± 8.6 beats·min⁻¹, $p = 0.02$, $g = -1.11$), which was followed by a return to baseline after the taper period. Surprisingly, we observed a moderate increase of τ after the taper

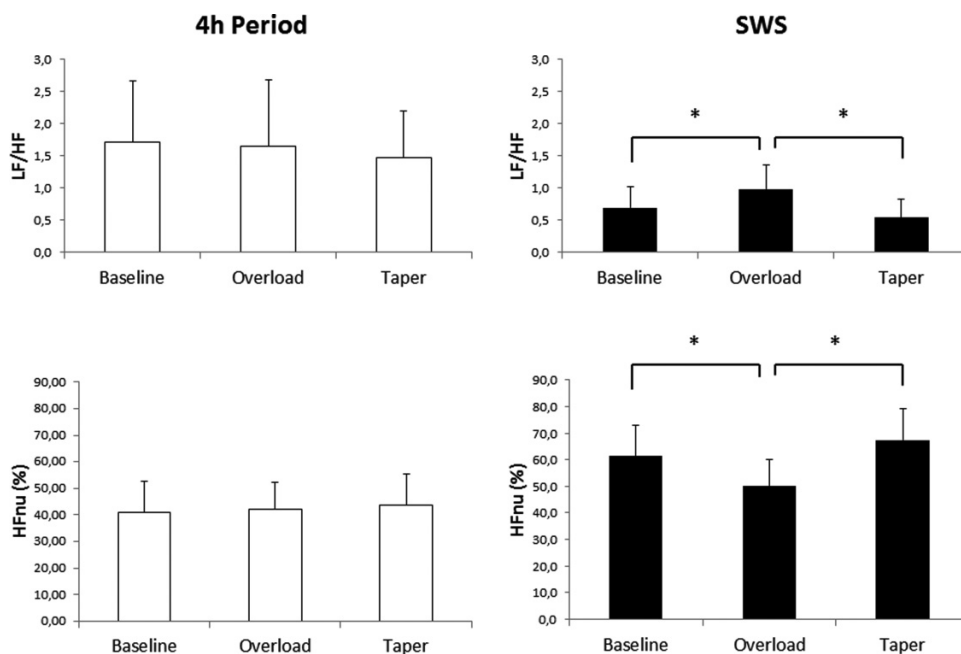
Table 3. Night heart rate variability in the time and frequency domains for the 2 conditions of measurement.

| | 4-h period | | | Estimated slow-wave sleep | | |
|-------------------------------|-------------------------|-------------------------|-------------------------|---------------------------|------------------------|------------------------|
| | Baseline | Overload | Taper | Baseline | Overload | Taper |
| Time domain | | | | | | |
| HR (beats·min ⁻¹) | 53.8±5.4 | 55.7±7.0 | 53.6±6.1 | 53.2±6.7 | 55.1±8.3 | 52.8±8.0 |
| RR (ms) | 1138±112 | 1078±210 | 1148±127 | 1144±136 | 1111±154 | 1161±169 |
| SDNN (ms) | 67.7±20.4 | 73.1±22.4 | 72.9±12.8 | 46.3±16.4 ^a | 46.4±19.5 ^a | 43.2±13.6 ^a |
| RMSSD (ms) | 76.1±30.0 | 81.5±32.7 | 84.6±19.5 | 57.1±30.5 | 57.5±28.7 | 58.3±23.5 |
| Frequency domain | | | | | | |
| HF (ms ²) | 1834±1221 | 3102±3916 | 2161±923 | 1176±934 | 1133±956 | 1105±696 |
| LF (ms ²) | 2453±1137 | 3151±1787 | 2633±722 | 580±292 | 912±652 | 468±258 |
| LF+HF (ms ²) | 4288±2400 | 6254±5526 | 4795±1405 | 1756±1175 | 2045±1482 | 1573±899 |
| Poincaré plot | | | | | | |
| SD1 (ms) | 54.4±21.3 | 58.2±23.3 | 60.4±13.8 | 42.6±18.6 | 40.9±20.4 | 41.4±16.7 |
| SD2 (ms) | 155.6±45.5 ^b | 169.5±42.4 ^b | 173.6±50.8 ^b | 62.8±20.0 ^a | 62.8±23.0 ^a | 55.9±14.8 ^a |

Note: Data presented as means ± SD. HR, heart rate; RR, R-R intervals; SDNN, SD of R-R intervals; RMSSD, root mean square difference of successive normal R-R intervals; HF, high-frequency bands; LF, low-frequency bands; SD1, SD 1 of Poincaré plot; SD2, SD 2 of Poincaré plot.

^aDifferent from the corresponding data in the “4-h period” analysis ($p < 0.05$).

^bDifferent from SD1 data in the same period ($p < 0.05$).

Fig. 2. Cardiac autonomic control during the night for 2 methods of heart rate variability analysis at baseline, after overload, and after taper. SWS, slow-wave sleep; LF/HF, low frequency/high frequency ratio; *, Different, $p < 0.05$.

period when compared with the overload period (64.3 ± 19.2 s vs 77.3 ± 21.4 s, $p = 0.03$, $g = 0.59$), whereas there was no difference between baseline and overload (68.3 ± 20.5 s vs 64.3 ± 19.2 s, ns , $g = -0.18$).

Discussion

The primary purpose of this study was to assess the effect of a 2-week overload period designed to induce a state of overreaching on cardiac autonomic control during the night or after exercise cessation. A secondary purpose was to compare the sensitivity of 2 different methods of assessing night HRV. Our main findings were (i) a decrease in cardiac parasympathetic control during SWS after the overload period, whereas it remained unchanged when measured during the 4-h night segment; (ii) a faster HRR following the maximal graded exercise test after the overload period, whereas it remained unchanged following the submaximal con-

stant intensity test; and (iii) a return to baseline of all alterations after the 1-week taper period. These findings underscore the relevance of measuring HRV and HRR in the monitoring of FOR-NFOR, but also indicate that care should be taken to select the most sensitive measures, because cardiac autonomic control is not affected uniformly by overload training.

Diagnosis of overreaching

A prerequisite to compare physical performances before and after an intervention such as the overload or taper periods is to make sure that all performances are indeed maximal. We did not find any effect of period on the rating of perceived exertion, whatever the exercise test, thus suggesting that this criterion was fulfilled in our study. The absence of alteration in $\dot{V}O_{2\text{ peak}}$ and PTS (Table 1) after the overload period is consistent with the literature, because unchanged values are not unusual in overreached ath-

Table 4. Autonomic indices during the recovery from the maximal continuous graded exercise and from the constant-speed test.

| | After maximal exercise | | | After constant speed test | | |
|---|------------------------|-----------------------|-----------------------|---------------------------|-----------------------|------------------------|
| | Baseline | Overload | Taper | Baseline | Overload | Taper |
| HRR responses | | | | | | |
| T30 (s) | 181.6±57.2 | 154.0±46.1 | 173.2±42.3 | 178.7±61.7 | 148.7±28.2 | 162.0±32.8 |
| Δ60 (beats·min ⁻¹) | 56.0±11.7 | 56.9±10.3 | 52.9±8.1 | 48.0±12.0 | 53.4±11.4 | 48.2±5.5 |
| τ (s) | 61.8±14.5 | 54.7±9.8 ^a | 65.0±8.9 ^b | 68.3±20.5 | 64.3±19.2 | 77.3±21.4 ^b |
| a ₀ (beats·min ⁻¹) | 100.5±8.8 | 93.1±7.0 ^a | 95.9±10.6 | 107.5±4.8 | 98.7±8.6 ^a | 104.0±7.5 |
| a ₁ (beats·min ⁻¹) | 93.7±9.1 | 94.4±8.9 | 95.6±6.1 | 71.9±11.9 | 78.1±12.4 | 70.0±70.6 |
| HRV in time domain | | | | | | |
| RR (ms) | 601±53 | 650±47 ^a | 610±40 | 531±110 | 597±44 ^a | 583±40 |
| SDNN (ms) | 10.5±3.7 | 16.2±9.8 | 12.0±5.1 | 9.8±3.9 | 12.7±5.3 | 10.1±3.0 |
| RMSSD (ms) | 6.9±3.1 | 10.4±8.5 | 7.9±4.6 | 5.7±2.5 | 7.7±3.4 | 5.9±2.0 |
| HRV in frequency domain | | | | | | |
| HF (ms ²) | 35.4±26.4 | 69.8±78.4 | 75.9±129.0 | 39.3±40.8 | 52.2±38 | 34.5±29.1 |
| LF (ms ²) | 81.3±106.8 | 181.5±223.3 | 70.0±48.9 | 67.6±81.4 | 111.7±174.2 | 54.9±46.4 |
| LF±HF (ms ²) | 116.6±110.5 | 251.4±289.5 | 145.9±162.5 | 106.9±119.7 | 163.9±201.4 | 89.5±64.7 |
| HFnu (%) | 43.6±13.2 | 38.0±15.1 | 40.2±17.1 | 39.5±19.2 | 39.5±17.0 | 37.1±18.5 |
| LF/HF ratio | 1.5±0.6 | 1.9±0.9 | 1.9±1.0 | 1.9±0.9 | 2.0±1.4 | 2.4±1.9 |

Note: Data presented as means ± SD. HRR, heart rate recovery; T30, negative reciprocal of the slope of the regression line between the natural logarithm of heart rate and elapsed time from the 10th to the 40th second after exercise; Δ60, absolute difference between heart rate immediately at the end of exercise and after 60 s of passive recovery; τ, time constant; a₀, asymptotic value of heart rate; a₁, decrement below the heart rate value at the end of exercise; HRV, heart rate variability; RR, R-R intervals; SDNN, SD of R-R intervals; RMSSD, root mean square difference of successive normal R-R intervals; HF, high-frequency bands; LF, low-frequency bands; HFnu, high frequency expressed in normalized units.

^aDifferent from baseline, *p* < 0.05.

^bDifferent from overload, *p* < 0.05.

letes (Bosquet et al. 2001; Fry et al. 1992; Urhausen et al. 1998). Considering that aerobic endurance is more sensitive to overload-induced fatigue than maximal aerobic power (Urhausen and Kindermann 2002), a constant-speed test at 85% of PTS was implemented to complete the assessment of physical performance. The moderate decrease we observed after the overload period is in accordance with previous reports (Dupuy et al. 2010; Bosquet et al. 2001; Urhausen et al. 1998) and confirms the recommendation by Urhausen and Kindermann (2002) that time to exhaustion at a constant-speed test is a very sensitive measure for detecting a decrease in performance capacity. All the participants in our study fulfilled this first criterion. Despite the absence of alteration in $\dot{V}O_{2\text{ peak}}$, we found a moderate decrease in HR_{peak} (Table 1). This observation is typical of overreaching, because the meta-analytic study by Bosquet et al. (2008) showed a small to moderate decrease in HR_{peak} in overreached athletes, whatever the duration of the overload period (i.e., ≤2 weeks or <2 weeks). All the participants in our study fulfilled this second criterion. Regarding the psychological sphere, we found a modified profile in the POMS (Table 2) and the RESTQ-sport (Fig. 1) after the overload period. This psychological impairment has been described consistently in the literature (Morgan et al. 1987; Dupuy et al. 2010) and confirms the usefulness of these questionnaires in the monitoring of overreaching. As already reported by Kentta et al. (2006) and later confirmed by Dupuy et al. (2010), a large decrease in the energy index computed from the vigor and fatigue subscales of the POMS was found. The decrement in the “fatigue”, “being in shape”, and “lack of energy” subscales of the RESTQ-sport are in agreement with previous observations in overreached athletes and after an intensified period of training (Nederhof et al. 2008; Kellmann 2010). In the end, all the participants in our study met 3 criteria after the overload period and were classified as overreached. The return to baseline of these criteria after the taper period led us to consider that it was a short-term overreaching that could be compared in terms of severity to the previously described FOR (Meeusen et al. 2006).

Night cardiac autonomic control

Considering that nighttime is generally free of events known to affect cardiac autonomic control, such as noise or light, we chose to assess HRV during sleep. Two methods were compared: the classical one, which consists of analyzing a 4-h period that begins 30 min after reported bedtime (Mylymaki et al. 2011; Hynynen et al. 2010), and a second one that consists of analyzing a 10-min period taken during the first SWS episode of the night lasting at least 15 min (Brandenberger et al. 2005). This second approach is interesting because it measures HRV in more standard conditions, thus making the interpretation of changes easier. Moreover, the predominance of cardiac parasympathetic activity during SWS (Buchheit et al. 2004) facilitates the detection of differences in vagal-related indices or cardiac autonomic balance when compared with complete nighttime.

We found a large decrease in cardiac parasympathetic control (HFnu) and a moderate increase in cardiac autonomic balance (LF/HF) during SWS after the overload period, followed by a return to baseline after the taper period (Fig. 2). The LF/HF ratio, at best, provides a relative balance between the innervations of the parasympathetic nervous system and the sympathetic nervous system on the sinoatrial node. In this sense, our results show no evidence to support the statement regarding a sympathetic dominance. In the meantime, there was no alteration of the same indices when they were computed from the 4-h segment. This difference in sensitivity between the 2 methods underlines the fact that combining all sleep periods into a single segment introduces some noise that prevents the detection of small or moderate HRV changes.

Results obtained during SWS are in accordance with several experimental studies that reported an overload-induced decrease in cardiac parasympathetic control and (or) an increase in cardiac sympathetic control (Iellamo et al. 2002; Pichot et al. 2000, 2002; Portier et al. 2001; Hynynen et al. 2006; Baumert et al. 2006). In contrast, others did not observe this overload effect (Bosquet et al.

2003; Hedelin et al. 2000; Uusitalo et al. 1998). Although several methodological differences among studies may have contributed to this discrepancy, such as the time at which the heart rate was measured, the main explanation appears to be the duration of the overload period per se. In their meta-analysis, Bosquet et al. (2008) used an arbitrary limit of 2 weeks to distinguish between short- and long-term interventions. Interestingly, cardiac autonomic alterations were restricted to interventions lasting ~2 weeks, because they found no modification of the cardiac autonomic balance or the total variability when the duration of the overload period was longer (up to 9 weeks). Although the exact physiological meaning of overload-induced cardiac autonomic changes is difficult to establish, HRV measured during SWS appears to be a valid sign of FOR-NFOR in endurance athletes.

Postexercise cardiac autonomic control

Postexercise HRR is thought to reflect the reactivation of parasympathetic activity at the sinus node level and, to a lesser extent, cardiac sympathetic withdrawal (Savin et al. 1982; Kannankeril and Goldberger 2002). In this respect and similar to SWS, it should be noted that HRR after exercise is particularly sensitive to changes in vagal-related indices such as τ . We found a faster HRR (as evidenced by a small decrease in τ) after the overload period when heart rate was measured after the maximal graded exercise test, but not when it was measured after the submaximal constant intensity test, because τ remained unchanged (Table 4). This difference in sensitivity between the tests may be explained by several factors. Because performance in the constant-intensity test was considered a diagnostic measure, it was not possible to standardize the duration of this protocol. As a consequence, the duration of the constant-intensity test varied greatly between periods, whereas it remained the same for the maximal graded exercise test, because PTS did not change throughout the study. On the other hand, the signal-to-noise ratio is an important determinant of the confidence of HRR indices, and more particularly of the parameter estimates from curve fitting (τ , a_0 , and a_1). Because the heart rate reaches higher values during the maximal graded exercise test (thus increasing the signal), and because the physiological intensity reached at the end of this test is better controlled (thus decreasing the noise), the signal-to-noise ratio is much higher than that of the constant-intensity test. Finally, and to some extent contradictory to the previous comment, we cannot exclude the fact that parasympathetic reactivation would have been greater if exercise intensity had been lower. In fact, data by Buchheit et al. (2007) underscored the need to control for chemoreflex activation when we aim at evaluating parasympathetic reactivation. It is therefore possible that a constant-duration test at a lower exercise intensity would have been more appropriate. It was not possible to schedule such a test in our study. However, an interesting follow-up to this work would be to compare the sensitivity of HRR indices to overreaching when measured after the cessation of a constant-duration test of moderate intensity (i.e., lower than the ventilatory threshold) and after the cessation of a maximal graded exercise test.

A faster HRR has already been reported in the literature, either in overreached athletes (Urhausen et al. 1998; Lamberts et al. 2010a) or after an intensified training period (Lamberts et al. 2009; Lamberts et al. 2010b). In this study we found a correlation between the decrease in HR_{peak} and the decrease in τ after the overload period. It is therefore conceivable that these 2 responses share some common physiological mechanisms. According to Zavorsky (2000), the decrease in HR_{peak} may be explained by an increased stroke volume consecutive to a plasma volume expansion, an altered humoral or neuronal cardiac autonomic control, and a decreased β -adrenergic receptor density and (or) sensitivity. The report by Uusitalo et al. (1998) does not support the hypothesis of an overload-induced plasma volume expansion, because it did not find any significant difference in endurance-trained females

who increased their training load by 100% for 6 to 9 weeks. Although the rapid decline in HR after exercise cessation is largely mediated by cardiac parasympathetic reactivation, the influence of the sympathetic branch of the ANS should not be disregarded. Pierpont et al. (2000) suggested that the interaction between sympathetic and parasympathetic activity at the heart level after exercise cessation was partially determined by the level of cardiac sympathetic control during exercise. The higher this level, the higher the importance of sympathetic withdrawal in the kinetics of HRR. We did not measure the sympathetic drive to the heart in this study. However, the results of Hooper et al. (1993), who reported a decrease in the concentration of catecholamines after a maximal intensity exercise that was concomitant to a decrease in HR_{peak} , suggest this activity could be decreased in our participants, because we also found a decrease in HR_{peak} . If true, this lowered cardiac sympathetic control could explain, at least partly, the faster HRR that was observed in our participants. As suggested by Halson et al. (2002), one of the most probable explanations is a down-regulation of β -adrenergic receptors, because Fry et al. (2006) reported a reduction in their number and sensitivity in 8 overreached strength athletes. This hypothesis of a desensitization of β -adrenergic receptors is clearly validated by the observations made by Lehmann et al. (1998), who reported a decreased concentration of both free fatty acids and glucose during an exercise of submaximal intensity, whereas they found an increased concentration of noradrenalin.

Limitations and practical considerations

The main result of this study (i.e., a decrease in cardiac parasympathetic control during SWS after the overload period, whereas it remained unchanged when measured during the 4-h night segment) relies on the assumption that the first segment retained for HRV analysis was taken during a true SWS episode. To determine the correct identification of a sleep stage, the use of a polysomnograph is a unique approach. Brandenberger et al. (2005) proposed an alternative approach using several HRV criteria that provided fairly good results when compared with sleep stages identification by a polysomnograph. In the current study, we followed these recommendations rigorously and provided some statistics to ensure the fulfillment of all criteria.

From a practical point of view, considering HRV and (or) HRR as valid markers of FOR is an oversimplification that may lead to an incorrect interpretation of the data. The results of the current study clearly show that cardiac autonomic control is altered by a 2-week overload period. However, it also underscores that there is only a limited set of measurement conditions and HRV or HRR measures that provide the reliability and sensitivity required to detect such an effect. The SWS approach by Brandenberger et al. (2005) should be preferred to the classical 4-h period to assess night HRV, and the signal should be analyzed in the frequency rather than the temporal domain. Additionally, postexercise HRR should be assessed after the cessation of an exercise of maximal intensity, and the signal should be fitted to a monoexponential model to obtain the time constant of heart rate decay. Additional data are required to provide a definitive conclusion on the validity of HRR after the cessation of an exercise of submaximal intensity, because the duration of the test was not controlled in this study. It should be kept in mind that although these measures presented a greater sensitivity to overload-induced cardiac autonomic changes in our study, HRV and HRR are generally considered to be only moderately reliable (Al Haddad et al. 2011; Dupuy et al. 2012). Care should therefore be taken to ensure highly standardized measurement conditions, including the control of training load the day before the test and the control of all other stimuli known to affect cardiac sympathetic control, such as beverages containing caffeine or alcohol, temperature, or luminosity. An important issue that arises from our study is the usefulness of HRV and HRR measures when a simple questionnaire like the POMS provides

both rapid and valid measures and is more accessible. Although they are very interesting in the monitoring of FOR–NFOR, psychological questionnaires like the POMS also have some limitations. In fact, Goss (1994) clearly showed that the effect of an overload period on subscores of the POMS was highly dependent on hardness in a group of competitive swimmers. This result emphasizes the fact that there is no pathognomonic marker of FOR–NFOR, and any alteration detected by a psychological questionnaire should be backed up by other possible signs, including HRV and HRR, before diagnosing the disorder. As a final word regarding the implementation of HR measures in the follow-up of athletes, it has to be recognized that night recordings are perceived as a constraint by many athletes. Therefore, HRR is probably more suited than HRV if the measures are scheduled on a very regular basis.

Conclusion

The purpose of this study was to evaluate the effect of a 2-week overload period immediately followed by a 1-week taper period on the autonomic control of heart rate during the night or after exercise cessation. A secondary purpose was to compare 2 methods of assessing night HRV. We found an alteration in cardiac autonomic control during SWS after the overload period, whereas there was no difference when the tachogram was analyzed after the 4-h night period. We also found a faster HRR after the maximal graded exercise test, but not after the constant-speed test. All the measures altered by the overload period returned to baseline after the taper period. Altogether, these results underscore the relevance of monitoring cardiac autonomic control in the follow-up of athletes, but also that care should be taken in selecting the most sensitive measures, because cardiac autonomic control is not affected uniformly by overload training.

References

- Achten, J., and Jeukendrup, A.E. 2003. Heart rate monitoring: Applications and limitations. *Sports Med.* 33(7): 517–538. doi:10.2165/00007256-200333070-00004. PMID:12762827.
- Al Haddad, H., Laursen, P.B., Ahmaidi, S., and Buchheit, M. 2009. Nocturnal Heart Rate Variability Following Supramaximal Intermittent Exercise. *Int. J. Sports Physiol. Perform.* 4(4): 435–447. PMID:20029095.
- Al Haddad, H., Laursen, P.B., Chollet, D., Ahmaidi, S., and Buchheit, M. 2011. Reliability of Resting and Postexercise Heart Rate Measures. *Int. J. Sports Med.* doi:10.1055/s-0031-1275356. PMID:21574126.
- Armstrong, L.E., and VanHeest, J.L. 2002. The unknown mechanism of the overtraining syndrome: Clues from depression and psychoneuroimmunology. *Sports Med.* 32(3): 185–209. doi:10.2165/00007256-200232030-00003. PMID:11839081.
- Aubert, A., Seps, B., and Beckers, F. 2003. Heart Rate Variability in Athletes. *Sports Med.* 33(12): 889–919. doi:10.2165/00007256-200333120-00003. PMID:12974657.
- Bai, X., Li, J., Zhou, L., and Li, X. 2009. Influence of the menstrual cycle on nonlinear properties of heart rate variability in young women. *Am. J. Physiol. Heart Circ. Physiol.* 297(2): H765–H774. doi:10.1152/ajpheart.01283.2008. PMID:19465541.
- Barron, J., Noakes, T., Levy, W., Smith, C., and Millar, R. 1985. Hypothalamic dysfunction in overtrained athletes. *J. Clin. Endocrinol. Metab.* 60: 803–806. doi:10.1210/jcem-60-4-803. PMID:2982908.
- Baumert, M., Brechtel, L., Lock, J., Hermsdorf, M., Wolff, R., Baier, V., and Voss, A. 2006. Heart rate variability, blood pressure variability, and baroreflex sensitivity in overtrained athletes. *Clin. J. Sport Med.* 16(5): 412–417. doi:10.1097/01.jsm.0000244610.34594.07. PMID:17016118.
- Borg, G.A.V. 1982. Psychological bases of perceived. *Med. Sci. Sports Exerc.* 14(5): 377–381. doi:10.1249/00005768-198205000-00012. PMID:7154893.
- Bosquet, L., Leger, L., and Legros, P. 2001. Blood lactate response to overtraining in male endurance athletes. *Eur. J. Appl. Physiol.* 84(1–2): 107–114. doi:10.1007/s004210000343. PMID:11394238.
- Bosquet, L., Papelier, Y., Leger, L., and Legros, P. 2003. Night heart rate variability during overtraining in male endurance athletes. *J. Sports Med. Phys. Fitness.* 43(4): 506–512. PMID:14767413.
- Bosquet, L., Montpetit, J., Arvisais, D., and Mujika, I. 2007. Effects of tapering on performance: A meta-analysis. *Med. Sci. Sports Exerc.* 39(8): 1358–1365. doi:10.1249/mss.0b013e31806010e0. PMID:17762369.
- Bosquet, L., Merkari, S., Arvisais, D., and Aubert, A.E. 2008. Is heart rate a convenient tool to monitor over-reaching? A systematic review of the literature. *Br. J. Sports Med.* 42(9): 709–714. doi:10.1136/bjism.2007.042200. PMID:18308872.
- Brandenberger, G., Buchheit, M., Ehrhart, J., Simon, C., and Piquard, F. 2005. Is slow wave sleep an appropriate recording condition for heart rate variability analysis? *Auton. Neurosci.* 121(1–2): 81–86. doi:10.1016/j.autneu.2005.06.002. PMID:16005265.
- Brown, R.L., Frederick, E.C., Falsetti, H.L., Burke, E.R., and Ryan, A.J. 1983. Overtraining of athletes. A round table. *Phys. Sportsmed.* 11(6): 92.
- Buchheit, M., Simon, C., Piquard, F., Ehrhart, J., and Brandenberger, G. 2004. Effects of increased training load on vagal-related indexes of heart rate variability: a novel sleep approach. *Am. J. Physiol. Heart Circ. Physiol.* 287(6): H2813–H2818. doi:10.1152/ajpheart.00490.2004. PMID:15308479.
- Buchheit, M., Papelier, Y., Laursen, P.B., and Ahmaidi, S. 2007. Noninvasive assessment of cardiac parasympathetic function: postexercise heart rate recovery or heart rate variability? *Am. J. Physiol. Heart Circ. Physiol.* 293(1): H8–H10. doi:10.1152/ajpheart.00335.2007. PMID:17384128.
- Cole, C.R., Blackstone, E.H., Pashkow, F.J., Snader, C.E., and Lauer, M.S. 1999. Heart-rate recovery immediately after exercise as a predictor of mortality. *N. Engl. J. Med.* 341(18): 1351–1357. doi:10.1056/NEJM199910283411804. PMID:10536127.
- Currell, K., and Jeukendrup, A. 2008. Validity, reliability and sensitivity of measures of sporting performance. *Sports Med.* 38(4): 297–316. doi:3843 [pii].
- Daanen, H.A.M., Lamberts, R.P., Kallen, V.L., Jin, A., and Van Meeteren, N. 2012. A Systematic Review on Heart Rate Recovery to Monitor Changes in Training Status in Athletes. *Int. J. Sports Physiol. Perform.* In press. PMID:22357753.
- Dupuy, O., Renaud, M., Bherer, L., and Bosquet, L. 2010. Effect of functional overreaching on executive functions. *Int. J. Sports Med.* 31(9): 617–623. doi:10.1055/s-0030-1255029. PMID:20544582.
- Dupuy, O., Mekary, S., Berryman, N., Bherer, L., Audiffren, M., and Bosquet, L. 2012. Reliability of heart rate measures used to assess post-exercise parasympathetic reactivation. *Clin. Physiol. Funct. Imaging.* In press. doi:10.1111/j.1475-097X.2012.01125.x. PMID:22681607.
- Fry, R., Morton, A., and Keast, D. 1991. Overtraining in athletes. An update. *Sports Med.* 12(1): 32–65. PMID:1925188.
- Fry, R.W., Morton, A.R., Garcia-Webb, P., Crawford, G.P.M., and Keast, D. 1992. Biological responses to overload training in endurance sports. *Eur. J. Appl. Physiol. Occup. Physiol.* 64(4): 335–344. doi:10.1007/BF00636221. PMID:1592059.
- Goss, J.D. 1994. Hardiness and mood disturbances in swimmers while overtraining. *J. Sport Exerc. Psychol.* 16: 135–149.
- Halson, S.L., and Jeukendrup, A.E. 2004. Does overtraining exist? An analysis of overreaching and overtraining research. *Sports Med.* 34(14): 967–981. doi:10.2165/00007256-200434140-00003. PMID:15571428.
- Hedelin, R., Kentta, G., Wiklund, U., Bjerle, P., and Henriksson-Larsen, K. 2000. Short-term overtraining: effects on performance, circulatory responses, and heart rate variability. *Med. Sci. Sports Exerc.* 32(8): 1480–1484. doi:10.1097/00005768-200008000-00017. PMID:10949015.
- Hedge, L. 1981. Distribution theory for Glass's estimator of effect size and related estimators. *Journal of Educational Statistics.* 6: 107–128.
- Hooper, S.L., and MacKinnon, L.T. 1995. Monitoring overtraining in athletes. *Recommendations. Sports Med.* 20(5): 321–327. PMID:8571005.
- Huszczuk, A., Whipp, B.J., and Wasserman, K. 1990. A respiratory gas-exchange simulator for routine calibration in metabolic studies. *Eur. Respir. J.* 3(4): 465–468. PMID:2114308.
- Hynynen, E., Uusitalo, A., Konttinen, N., and Rusko, H. 2006. Heart rate variability during night sleep and after awakening in overtrained athletes. *Med. Sci. Sports Exerc.* 38(2): 313–317. doi:10.1249/01.mss.0000184631.27641.b5. PMID:16531900.
- Hynynen, E., Vesterinen, V., Rusko, H., and Nummela, A. 2010. Effects of Moderate and Heavy Endurance Exercise on Nocturnal HRV. *Int. J. Sports Med.* 31(6): 428–432. doi:10.1055/s-0030-1249625. PMID:20419617.
- Iellamo, F., Legramante, J.M., Pigozzi, F., Spataro, A., Norbiato, G., Lucini, D., and Pagani, M. 2002. Conversion from vagal to sympathetic predominance with strenuous training in high-performance world class athletes. *Circulation.* 105(23): 2719–2724. doi:10.1161/01.cir.0000018124.01299.ae. PMID:12057984.
- Imai, K., Sato, H., Hori, M., Kusuoka, H., Ozaki, H., Yokoyama, H., et al. 1994. Vagally mediated heart rate recovery after exercise is accelerated in athletes but blunted in patients with chronic heart failure. *J. Am. Coll. Cardiol.* 24(6): 1529–1535. doi:10.1016/0735-1097(94)90150-3. PMID:7930286.
- Israel, S. 1958. Die Erscheinungsformen des Übertrainings. *Sportmedizin.* 9: 207–209.
- Kannankeril, P.J., and Goldberger, J.J. 2002. Parasympathetic effects on cardiac electrophysiology during exercise and recovery. *Am. J. Physiol. Heart Circ. Physiol.* 282(6): H2091–H2098. doi:10.1152/ajpheart.00825.2001. PMID:12003816.
- Kellmann, M. 2010. Preventing overtraining in athletes in high-intensity sports and stress/recovery monitoring. *Scand. J. Med. Sci. Sports.* 20(Suppl. 2): 95–102. doi:10.1111/j.1600-0838.2010.01192.x. PMID:20840567.
- Kellmann, M., and Kallus, K. 2001. Recovery Stress Questionnaire for Athletes: User Manual. Champaign, IL: Human Kinetics.
- Kentta, G., Hassmen, P., and Raglin, J.S. 2006. Mood state monitoring of training and recovery in elite kayakers. *Eur. J. Sport Sci.* 6(4): 245–253. doi:10.1080/17461390601012652.
- Kereszty, A. 1971. Overtraining. In *Encyclopedia of sport sciences and medicine.* Edited by L. Larsson. MacMillan, New York, N.Y., USA. pp. 218–222.
- Lehmann, M., Foster, C., and Keul, J. 1993. Overtraining in endurance athletes: a

- brief review. *Med. Sci. Sports Exerc.* **25**: 855–862. doi:10.1249/00005768-199307000-00015. PMID:8350709.
- Lehmann, M.J., Lormes, W., Opitz-Gress, A., Steinacker, J.M., Netzer, N., Foster, C., and Gastmann, U. 1997. Training and overtraining: an overview and experimental results in endurance sports. *J. Sports Med. Phys. Fitness*, **37**(1): 7–17. PMID:9190120.
- Lehmann, M., Foster, C., Dickhuth, H., and Gastmann, U. 1998. Autonomic imbalance hypothesis and overtraining syndrome. *Med. Sci. Sports Exerc.* **30**(7): 1140–1145. doi:10.1097/00005768-199807000-00019. PMID:9662686.
- Mac Nair, D., Lorr, M., and Drippelman, L. 1971. Profile of mood States manual. San Diego: Educational and industrial testing service.
- Meeusen, R., Duclos, M., Gleeson, M., Rietjens, G., Steinacker, J., and Urhausen, A. 2006. Prevention, diagnosis and treatment of the Overtraining Syndrome - ECSS Position Statement "Task Force". *Eur. J. Sport Sci.* **6**(1): 1–14. doi:10.1080/17461390600617717.
- Morgan, W.P., Brown, D.R., Raglin, J.S., O'Connor, P.J., and Ellickson, K.A. 1987. Psychological monitoring of overtraining and staleness. *Br. J. Sports Med.* **21**(3): 107–114. doi:10.1136/bjism.21.3.107. PMID:3676635.
- Myllymaki, T., Rusko, H., Syvaaja, H., Juuti, T., Kinnunen, M.L., and Kyrolainen, H. 2011. Effects of exercise intensity and duration on nocturnal heart rate variability and sleep quality. *Eur. J. Appl. Physiol.* doi:10.1007/s00421-011-2034-9. PMID:21667290.
- Nederhof, E., Zwerver, J., Brink, M., Meeusen, R., and Lemmink, K. 2008. Different diagnostic tools in nonfunctional overreaching. *Int. J. Sports Med.* **29**(7): 590–597. doi:10.1055/s-2007-989264. PMID:18050061.
- Perini, R., Orizio, C., Comandè, A., Castellano, M., Beschi, M., and Veicsteinas, A. 1989. Plasma norepinephrine and heart-rate dynamics during recovery from submaximal exercise in man. *Eur. J. Appl. Physiol. Occup. Physiol.* **58**(8): 879–883. doi:10.1007/BF02332222. PMID:2767070.
- Pichot, V., Roche, F., Gaspoz, J., Enjolras, F., Antoniadis, A., Minini, P., et al. 2000. Relation between heart-rate variability and training load in middle-distance runners. *Med. Sci. Sports Exerc.* **32**: 1729–1736. doi:10.1097/00005768-200010000-00011. PMID:11039645.
- Pichot, V., Busso, T., Roche, F., Garet, M., Costes, F., Duverney, D., et al. 2002. Autonomic adaptation to intensive and overload periods: a laboratory study. *Med. Sci. Sports Exerc.* **34**: 1660–1666. doi:10.1097/00005768-200210000-00019. PMID:12370569.
- Portier, H., Louisy, F., and Laude, D. 2001. Intense endurance training on heart rate and blood pressure variability in runners. *Med. Sci. Sports Exerc.* **33**: 1120–1125. PMID:11445759.
- Sandercock, G. 2007. Normative values, reliability and sample size estimates in heart rate variability. *Clin. Sci.* **113**(3–4): 129–130. doi:10.1042/cs20070137. PMID:17451377.
- Savin, W.M., Davidson, D.M., and Haskell, W.L. 1982. Autonomic contribution to heart-rate recovery from exercise in humans. *J. Appl. Physiol.* **53** (6): 1572–1575. PMID:7153152.
- Urhausen, A., and Kindermann, W. 2002. Diagnosis of Overtraining. What Tools Do We Have? *Sports Med.* **32**(2): 95–102. PMID:11817995.
- Urhausen, A., Gabriel, H., and Weiler, B. 1998. Ergometric and psychological findings during overtraining: a long-term follow-up study in endurance athletes. *Int. J. Sports Med.* **19**(2): 114–120. doi:10.1055/s-2007-971892. PMID:9562220.
- Uusitalo, A.L., Uusitalo, A.J., and Rusko, H.K. 1998. 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.* **19**(8): 532–540. doi:10.1055/s-2007-971956. PMID:9877144.
- Vincent, W.J. 2005. *Statistics in kinesiology*. Human Kinetics, Champaign, Ill., USA.