Sports Med (2013) 43:773–781
DOI 10.1007/s40279-013-0071-8

CURRENT OPINION

Training Adaptation and Heart Rate Variability in Elite Endurance Athletes: Opening the Door to Effective Monitoring

Daniel J. Plews · Paul B. Laursen · Jamie Stanley ·
Andrew E. Kilding · Martin Buchheit

Abstract The measurement of heart rate variability (HRV) is often considered a convenient non-invasive assessment tool for monitoring individual adaptation to training. Decreases and increases in vagal-derived indices of HRV have been suggested to indicate negative and positive adaptations, respectively, to endurance training regimens. However, much of the research in this area has involved recreational and well-trained athletes, with the small number of studies conducted in elite athletes revealing equivocal outcomes. For example, in elite athletes, studies have revealed both increases and decreases in HRV to be associated with negative adaptation. Additionally, signs of positive adaptation, such as increases in cardiorespiratory fitness, have been observed with atypical concomitant decreases in HRV. As such, practical ways by which HRV can be used to monitor training status in elites are yet to be established. This article addresses the current literature that has assessed changes in HRV in response to training loads and the likely positive and negative adaptations shown. We reveal limitations with respect to how the measurement of HRV has been interpreted to assess positive and negative adaptation to endurance training regimens and subsequent physical performance. We offer solutions to some of the methodological issues associated with using HRV as a day-to-day monitoring tool. These include the use of appropriate averaging techniques, and the use of specific HRV indices to overcome the issue of HRV saturation in elite athletes (i.e., reductions in HRV despite decreases in resting heart rate). Finally, we provide examples in Olympic and World Champion athletes showing how these indices can be practically applied to assess training status and readiness to perform in the period leading up to a pinnacle event. The paper reveals how longitudinal HRV monitoring in elites is required to understand their unique individual HRV fingerprint. For the first time, we demonstrate how increases and decreases in HRV relate to changes in fitness and freshness, respectively, in elite athletes.

1 Introduction

One of the more promising methods to monitor individual adaptation to training involves the regular monitoring of cardiac autonomic nervous system (ANS) status, through the measurement of resting or post-exercise heart rate variability (HRV) [3–5]. Indeed, non-functional overreaching (NFOR) and/or negative adaptation to training is thought to be generally associated with reductions in vagal-

---

Electronic supplementary material The online version of this article (doi:10.1007/s40279-013-0071-8) contains supplementary material, which is available to authorized users.

D. J. Plews (✉) · P. B. Laursen
High Performance Sport New Zealand, AUT Millennium,
17 Antares Place, Mairangi Bay, 0632 Auckland, New Zealand
e-mail: daniel.plews@hpsnz.org.nz

D. J. Plews · P. B. Laursen · A. E. Kilding
Sports Performance Research Institute New Zealand (SPRINZ),
Auckland University of Technology, Auckland, New Zealand

J. Stanley
Centre of Excellence for Applied Sport Science, Queensland
Academy of Sport, Brisbane, Australia

J. Stanley
School of Human Movement Studies, The University of
Queensland, Brisbane, Australia

M. Buchheit
Physiology Unit, Football Performance and Science Department,
ASPIRE, Academy of Sports Excellence, Doha, Qatar

related indices of HRV [6–8], whereas increases in fitness [9–11] and exercise performance [4, 12, 13] are thought to be more associated with increases in vagal-related indices of HRV. While findings from studies involving recreational and well trained athletes suggest that HRV may be a valuable tool for assessing individual adaptation to endurance training, data obtained from elite athletes and athletes with a longer training history remains equivocal [14–18].

The purpose of this article is to present a brief summary of the studies where HRV has been investigated in response to adaptation and changes in training load. In doing so, we highlight the methodological issues inherent in its use and interpretation to date. We advance our current opinion of how HRV should best be monitored and assessed with examples from elite endurance athletes. All references to HRV throughout this manuscript refer to vagal-related indices of HRV [19]. All HRV data presented herein were recorded upon waking and measured as the last 5 min of the 6 min supine rest period (for more details on the methodology, including calculation of the "smallest worthwhile change", please refer to [1]).

2 HRV in Response to Different Training Loads

The influence of intensified and reduced training loads on HRV has been thoroughly studied (see electronic supplementary material [ESM], Table S1). In moderately trained subjects, moderate training loads increase aerobic fitness, as well as HRV [15, 20–22]. However, when training loads approach higher levels (100 % of an individual's maximal training load), HRV indices are reduced, [15, 21, 23] and are thought to rebound after periods of reduced training (i.e., taper) [13, 22, 23]. For example, after 3 weeks of overload training in swimmers and distance runners, HRV was reduced by 22 % [13] and 38 % [23], respectively. Following 2 weeks of reduced training (69 % reduction in training load compared with overload), HRV rebounded and increased by 7 % in swimmers [13], and after 1 week (40 % reduction in training load compared with overload) increased by 38 % in distance runners [23]. As with moderately trained athletes, elites and athletes with extensive training histories also show increases and decreases in HRV after moderate and high training loads, respectively [15, 18, 21]). Conversely, however, HRV can remain depressed in the lead up to competition (e.g. tapering), despite achievement of an optimal performance [15, 18]. In the case of these athletes, the reduction in HRV prior to competition possibly reflects the HRV response to consecutive days of high intensity training (with a reduction in training volume in the case of a taper) [24, 25] and/or HRV saturation at low heart rate levels [26] (see Sects. 4.1 and 4.2, respectively). With just one study examining the HRV responses of elites leading into competition, [18] the optimal HRV response to training overload and pre-competition tapers (in elites) is yet to be fully understood.

2.1 HRV and Positive Adaptation to Training

The changes in HRV in response to endurance training have been extensively studied (see ESM, Table S2). In sedentary and recreationally trained individuals, endurance training for 2 [9], 6 [27, 28], and 9 [4] weeks has been shown to induce parallel increases in aerobic fitness and HRV. For example, Buchheit et al. [4] showed that improvements in maximal aerobic running speed and 10 km run time had moderate ($r = 0.52$; confidence intervals [CI] 0.08 to 0.79) and large ($r = -0.73$; CI -0.89 to -0.41) correlations with increases in resting HRV, respectively. While this is the typical response shown in sedentary and recreationally trained individuals following a period of endurance training [9, 20, 22, 27, 28], the response in athletes with extensive training histories (e.g., elite athletes) can be markedly different. In these athletes, the HRV response to training is variable, with longitudinal studies showing no change in fitness (i.e., maximal oxygen uptake [$\dot{V}O_{2\max}$]) despite an increase in HRV [17], and other studies showing decreases in HRV despite increases in fitness [18]. As such, there is generally a bell-shaped relationship between vagally-related HRV and fitness.

2.2 HRV and Negative Adaptation to Training

Overtraining (OT) is a verb used to describe the process of undergoing intensified training to induce possible overreaching. Overreaching refers to a short-term stress-regeneration imbalance that includes negative outcomes such as increased fatigue and reductions in performance [29]. While overreaching is typically believed to be an important component of the elite athlete training cycle, prolonged overreaching can push an athlete into a state of NFOR, which is associated with reductions in performance ability that do not resume for several weeks or months [30]. To date, however, studies that have examined changes in HRV with NFOR/OT have revealed equivocal findings, with increases [31], decreases [6, 32] and no changes [31, 33, 34] in HRV reported (ESM, Table S3). In a case study of an elite cross-country skier that became OT, Hedelin et al. [31] showed reduced competition performance and lowered profile of mood states, along with substantially increased HRV. Conversely, Uusitalo et al. [32] showed that OT was associated with decreased HRV in endurance athletes undergoing heavy training over a 6- to 9-week period. Hedelin et al. [35] also reported unchanged HRV in elite canoeists, despite decreased run time to fatigue and reduced $\dot{V}O_{2\max}$. However, the inconsistent findings shown between HRV and OT/NFOR to date are likely due to the methodological approach adopted (see Sect. 3) and difficulty with discriminating between the different stages of the OT process (e.g. overtraining, overreaching, NFOR and OT syndrome) [30, 36]. This is particularly evident in studies that have purposely induced overtraining [33–35, 37], which unlikely reflects real-life training conditions [30, 36]. Finally, the possibility that two types of OT may occur in athletes (parasympathetic vs. sympathetic; [38, 39]) may further contribute to the equivocal research findings shown.

2.3 Literature Summary

It has been suggested that increases and decreases in HRV are associated with positive [4, 9–13] and negative [6, 7, 32] adaptation to endurance training regimens, respectively. However, the bell-shaped relationship typically apparent between both HRV and training load [15, 18, 21, 22], and HRV and fitness [14, 18, 40], in elites and athletes with extensive training histories, makes it difficult to practically use HRV to maximize training in these populations.

3 Methodological Consideration with the Assessment of HRV

Indices of HRV display a naturally high day-to-day variation [41]. We have recently suggested that both environmental factors influencing measurement 'noise' and acute changes in homeostasis may contribute to discrepancies in interpretation when a single data point is used for analysis. When HRV is used to assess changes in both negative [1] and positive adaptation [42], both weekly [1, 42] and 7-day rolling [1] averages have been shown to provide better methodological validity compared with values taken on a single day. For example, when HRV data points were averaged over 1 week, a meaningful representation of training status was apparent in a NFOR elite athlete (e.g., worthwhile reductions in weekly-averaged HRV were observed only during the period of NFOR) [1]. Comparatively, when single day values were used for analysis, the HRV data were misleading (i.e., worthwhile reductions in HRV indicative of NFOR occurred when the athlete was training and performing effectively). Conversely, when percentage changes in 10-km running performance were correlated with percentage changes in HRV, very large relationships were observed when HRV values were averaged over 1 week ($r = -0.76$; CI -0.92 to -0.36) but not when using single-day values ($r = -0.17$; CI -0.66 to 0.42) [42]. This suggests that averaged morning resting HRV data provide a more consistent representation of actual changes in an athlete's autonomic balance with training compared with a single isolated value. Most recently, morning resting HRV was shown to deviate little, irrespective of the prior day/s training, when positive adaptions to training occurred in well trained individuals [43].

Another methodological issue apparent within the literature is the variety of HRV indices that have been used to assess autonomic balance [19]. It has been shown that time domain indices of HRV have a lower typical error of measurement (when expressed as a coefficient variation [CV]) than other spectral indices of HRV (e.g., the natural logarithm of the square root of the mean sum of the squared differences between *R*–*R* intervals [Ln rMSSD], CV = 12.3 %; normalized high frequency power [HFnu], CV = 52.0 %) [41]. We suggest that practitioners and researchers using HRV measurements to evaluate training adaptations choose just one vagally-derived HRV variable for assessment. We prefer Ln rMSSD, as it is the most practically applicable HRV index for a number of reasons. First, Ln rMSSD is not significantly influenced by breathing frequency, unlike other spectral indices of HRV, and is therefore more suited to ambulatory measures [44]. Second, Ln rMSSD can capture levels of parasympathetic activity over a short time frame, which is more convenient for athletes who have limited time to acquire a reading [45]. Last, Ln rMSSD values can be easily calculated in MS Excel using *R*–*R* intervals [46]. In our opinion, therefore, the equivocal findings apparent throughout the HRV literature are likely due to the large day-to-day variation in HRV and the variety of HRV indices used for analysis that are more prone to errors.

4 The Relevance of HRV in Elite Athletes

Training programmes for elite athletes typically consist of periods of high training loads with limited periods of rest and recovery [47, 48]. Such athletes are always pushing the boundary between functional and NFOR in an attempt to gain the greatest possible fitness level. Despite this, published data in elite athletes are rare, with most HRV research to date involving recreational/well trained subjects [9, 15, 20, 22, 27, 28]. Due to genetics and training history [49], elite athletes may respond differently to training stresses and subsequent recovery [50]. In the following sections, we describe some of the different HRV profiles of elite athletes we have observed, and how these fluctuations may be reflective of training adaptation and the ability to perform at peak levels.

4.1 HRV Profiles in Elite Athletes

A common misconception made by sports practitioners using HRV to assess ANS status is that there is a direct linear relationship between vagal-related indices of HRV and the parasympathetic influence on heart rate (HR). In reality, however, the relationship is quadratic [51, 52] (see example in Fig. 1). This means that at both low (high HR) and high (low HR) levels of vagal tone, vagal-related HRV indices are reduced. For instance, while well trained athletes generally present both a low resting HR and increased HRV indices, a reduced HRV has also been observed in many athletes with a low resting HR [53]. This reduction of HRV at low HR is related to the fact that vagal-related HRV indexes more reflect the magnitude of modulation in parasympathetic outflow as opposed to an overall parasympathetic tone per se [54]. The underlying mechanism is likely the saturation of acetylcholine receptors at the myocyte level: a heightened vagal tone may give rise to sustained parasympathetic control of the sinus node, which may eliminate respiratory heart modulation and reduce HRV [55]. This is an important consideration for practitioners using HRV to assess training status in elites, who typically have a low resting HR, undergo high training loads and are therefore prone to saturation [26, 56]. For example, during different phases/loads of training, reductions in HRV can occur, 'theoretically' indicating ANS stress/NFOR [8, 57]. However, this trend should only be interpreted in light of the respective changes in resting HR, to assess whether this decrease can be the result of the saturation phenomenon or not. This can be achieved by using the Ln rMSSD to *R*–*R* interval ratio [1], which simultaneously considers changes in both vagal tone (*R*–*R* interval) and vagal modulation (HRV) [5].

Figure 2 shows two athletes competing at the same international rowing world cup event (Lucerne FISA World Cup 2012), both with suppressed HRV values before the race. However, in the case of Athlete A who performed optimally (second place in his event, 0.12 % behind the winner), the reduction in Ln rMSSD (falling below the smallest worthwhile change [SWC]; see [1, 2]) in the lead up to the race was a result of HRV saturation (as demonstrated by the Ln rMSSD to *R*–*R* interval ratio falling below the SWC) and unlikely fatigue [57]. However, Athlete B (performing poorly; fifth place in her event; 1.92 % behind the leader despite being a 2011 world championship medalist) incurred reductions in Ln rMSSD and increases in the Ln rMSSD to *R*–*R* interval ratio, suggesting both a loss in vagal tone and modulation. This was likely due to poor adaptation to her training load (NFOR) and sympathetic over-activity.

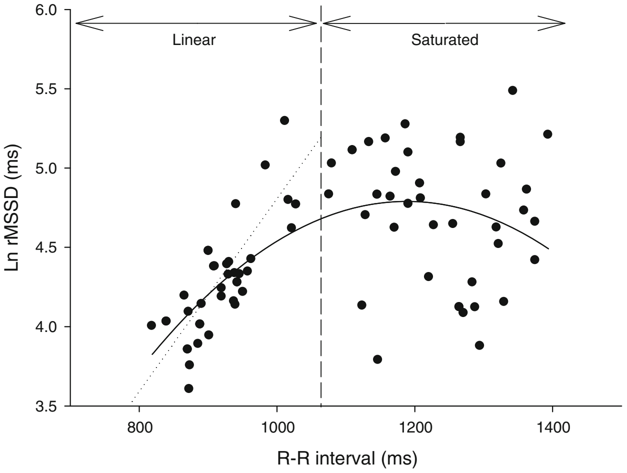

Fig. 1 Example of the relationship between the *R*–*R* interval and the natural logarithm of the square root of the mean sum of the squared differences between *R*–*R* intervals (*Ln rMSSD*) in a subject with increasing bradycardia. Here, a saturation of heart rate variability is seen with long *R*–*R* intervals. Note how at shorter *R*–*R* intervals there is a linear relationship between Ln rMSSD (*dotted line*), which becomes disassociated as the duration of the *R*–*R* interval increases, indicating heart rate variability saturation

Recently, we have also shown changes in the relationship between Ln rMSSD and the *R*–*R* interval during effective training and NFOR in an elite female triathlete [1]. In this instance, the athlete was saturated when training effectively and became linear as NFOR manifested. In our opinion, however, it is unlikely that either occurrence predicts NFOR; instead each individual has their own unique cardiac autonomic status and HRV relationship [52], which is likely related to situational and genetic factors. Figure 3 reveals the unique morning resting Ln rMSSD to *R*–*R* interval ratio profile of four Olympic and World champions in the 62-day lead up to winning their 2011/2012 event. All athletes show distinctly different profiles, irrespective of the fact that all athletes executed gold-medal winning performances.

In summary, reductions in HRV have been associated with fatigue and/or NFOR in recreationally trained and well trained subjects [7, 8, 32, 57]. However, conclusions from past literature reporting isolated HRV values should be viewed with caution [1, 42]. We suggest the use of both the Ln rMSSD (weekly average) and Ln rMSSD to *R*–*R* interval ratio to correctly interpret fatigue, or a 'readiness to perform' in elite athletes (e.g., worthwhile reductions in Ln rMSSD with concomitant increases in the Ln rMSSD: *R*–*R* interval ratio are more indicative of fatigue, with decreases in both indicating readiness to perform [Fig. 2]). Furthermore, the optimal relationship between HRV and *R*–*R* interval for training and performance alone is likely to be individual (Fig. 3; i.e., correlated, non-correlated or saturated [53]). This implies that longitudinal monitoring and an understanding of a particular athlete's response to training and competition (i.e., recognizing each athlete's

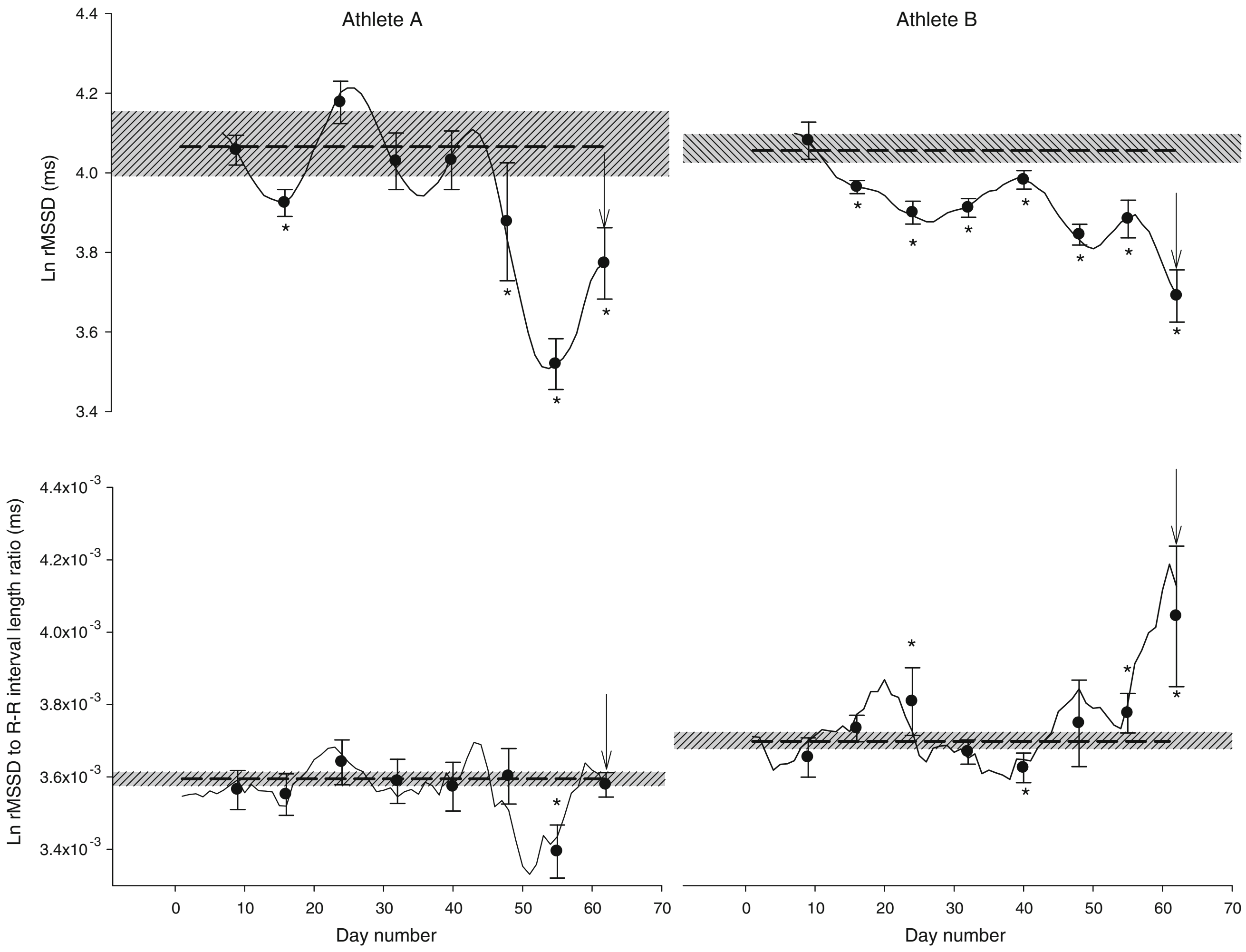

Fig. 2 Changes in the natural logarithm of the square root of the mean sum of the squared differences between *R–R* intervals (*Ln rMSSD*) and the Ln rMSSD to *R–R* interval ratio with 90 % confidence intervals (CI) for Athlete A (performing well) and Athlete B (performing poorly; see text) over a 62-day period in the build-up to a key rowing world cup event. *Black circular symbols* indicate the weekly average values for both Ln rMSSD and Ln rMSSD to *R–R* interval ratio, respectively; while the *black line* represents the 7-day rolling average. The *arrows* indicate the day of the final (medal) race. The *grey shaded area* indicates the individual smallest worthwhile change (*SWC*) in both values (see methods in Ref. [1]); the *black dashed line* represents the zero line of the SWC to indicate clear/unclear changes when the 90 % CI overlaps [2]. * indicates a 'clear' change in both weekly averaged values above the SWC in the weeks prior to the race

optimal Ln rMSSD to *R–R* interval fingerprint) is needed before this relationship can be useful enough to assist with training prescription.

4.2 Changes in HRV and Performance in Elites

As mentioned previously (Sect. 2), studies have shown that during intense training periods, vagal indices of HRV decrease acutely, and rebound beyond their pre-training level during subsequent recovery or lighter training periods [12, 13, 18, 23, 58]. The rebound of HRV has been shown to be associated with improved performance in recreationally trained and well trained athletes [12, 13]. However, as mentioned in Sect. 2.2, the bell-shaped relationship between fitness and HRV sometimes apparent in both elites and athletes with extensive training histories means that making this assessment is more challenging. Figure 4 shows Ln rMSSD values in three elite rowers during their preparation for the 2011 World Rowing Championships and 2012 London Olympic Games. Each athlete won their event. Over this 62-day period, training was at a high intensity (D.J. Plews, personal observations), with training volumes reaching 17 h 21 min ± 3 h 51 min/week (2011) and 16 h 44 min ± 5 h 05 min/week (2012). In these athletes, HRV generally increased in the week(s) before each event (going above the SWC; 4 out of 6 points being 'clear' 1–3 weeks prior to the event), before the values decreased to slightly lower levels (generally within the SWC) before the race.

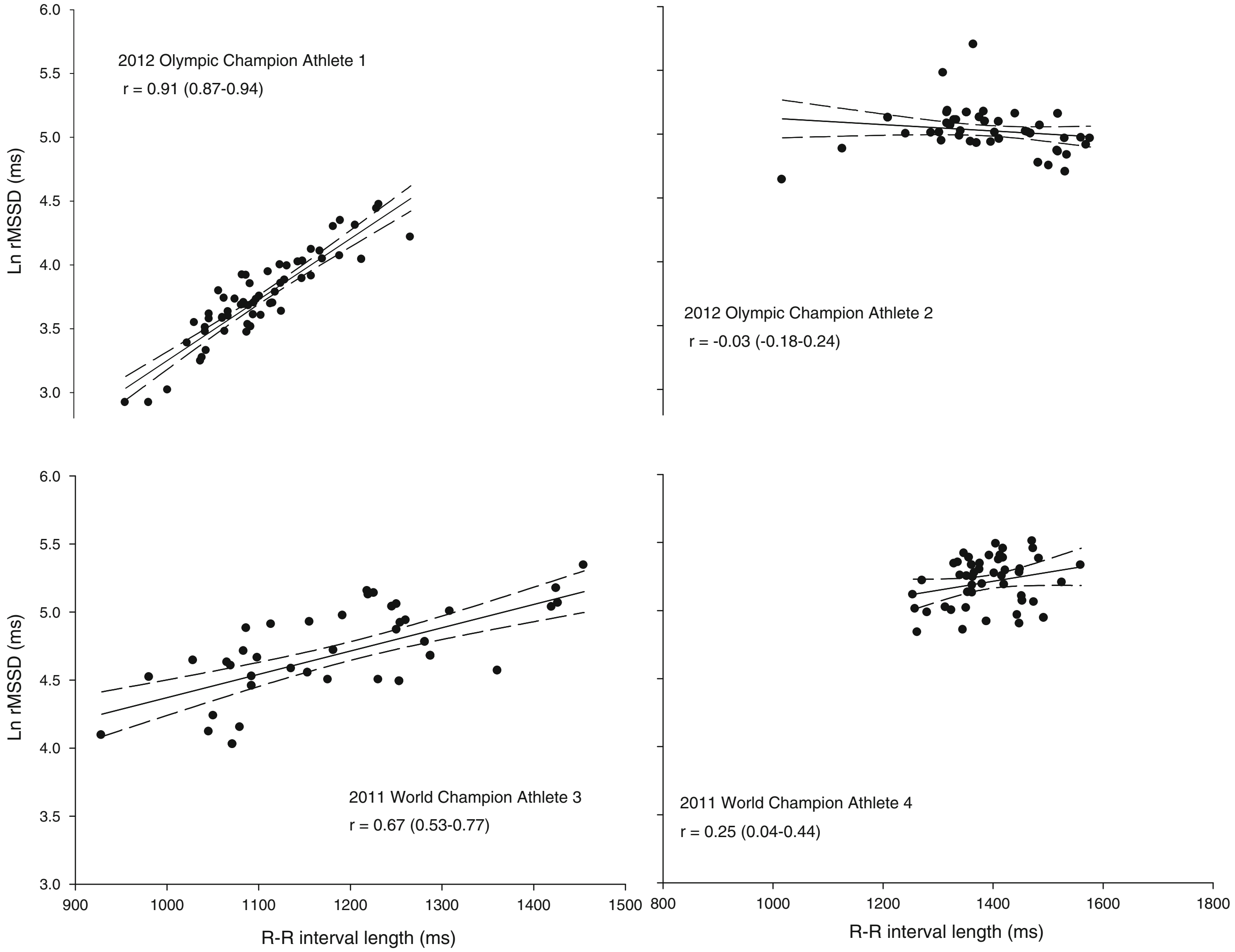

Fig. 3 Correlation and 90 % confidence intervals (CI) between the natural logarithm of the square root of the mean sum of the squared differences between *R–R* intervals (*Ln rMSSD*) and *R–R* interval length in two 2012 Olympic Champion rowers and two 2011 World Champion rowers taken every morning upon waking in their 62-day build-up to each pinnacle event. Correlation coefficients were almost perfect ($r = 0.91$; CI 0.87; 0.94) and trivial ($r = -0.03$; CI -0.18; 0.24) for Olympic Champion rowers 1 and 2, respectively. Comparatively these values were large ($r = 0.67$; CI 0.53; 0.77) and small ($r = 0.25$; CI 0.04; 044) for World Champion rowers 3 and 4

As such, it appears that for elite athletes, increases in HRV in the weeks before their event, during their highest training loads (Fig. 4), are likely associated with a positive performance outcome. This may indicate an athlete is ‘coping’ with the applied training load and is making positive adaptations. Conversely, Iellamo et al. [59] reported small, non-significant decreases in HRV profiles in Olympic rowers during strenuous training, which is likely due to the very long history of intensive training and small (undetectable) changes in fitness. It is possible, however, that the use of ‘individual’ SWCs may permit a better representation of meaningful changes in HRV in elites for the purpose of monitoring and assessing adaptation.

The fact that HRV values declined as competition approached is in agreement with other studies, in that lower levels of HRV prior to competition tend to be associated with superior performances [15, 18]. As such, it is clear that in these athletes, who are the best in the world at their event, a high HRV does not necessarily imply superior fitness [12, 13, 23, 58] and/or performance [12, 13]. The reason why HRV reduces to lower values prior to competition, from both a physiological and performance perspective, is unknown. However, as mentioned, a lower HRV does not necessarily imply fatigue (i.e., saturation; Sect. 4.1), and is therefore unlikely to ‘rebound’ in elites when training load is reduced and freshness increased. Furthermore, tapers leading into competition typically consist of reductions in training volume with the maintenance of intensity [60]. The reduction in training volume might elicit lowered blood plasma volume, and in turn, HRV [61, 62]. However, the maintenance of high intensity exercise during the taper should, in theory, attenuate HRV reductions [24, 25, 63]. Another possible explanation for the reduced HRV observed around the time of competition

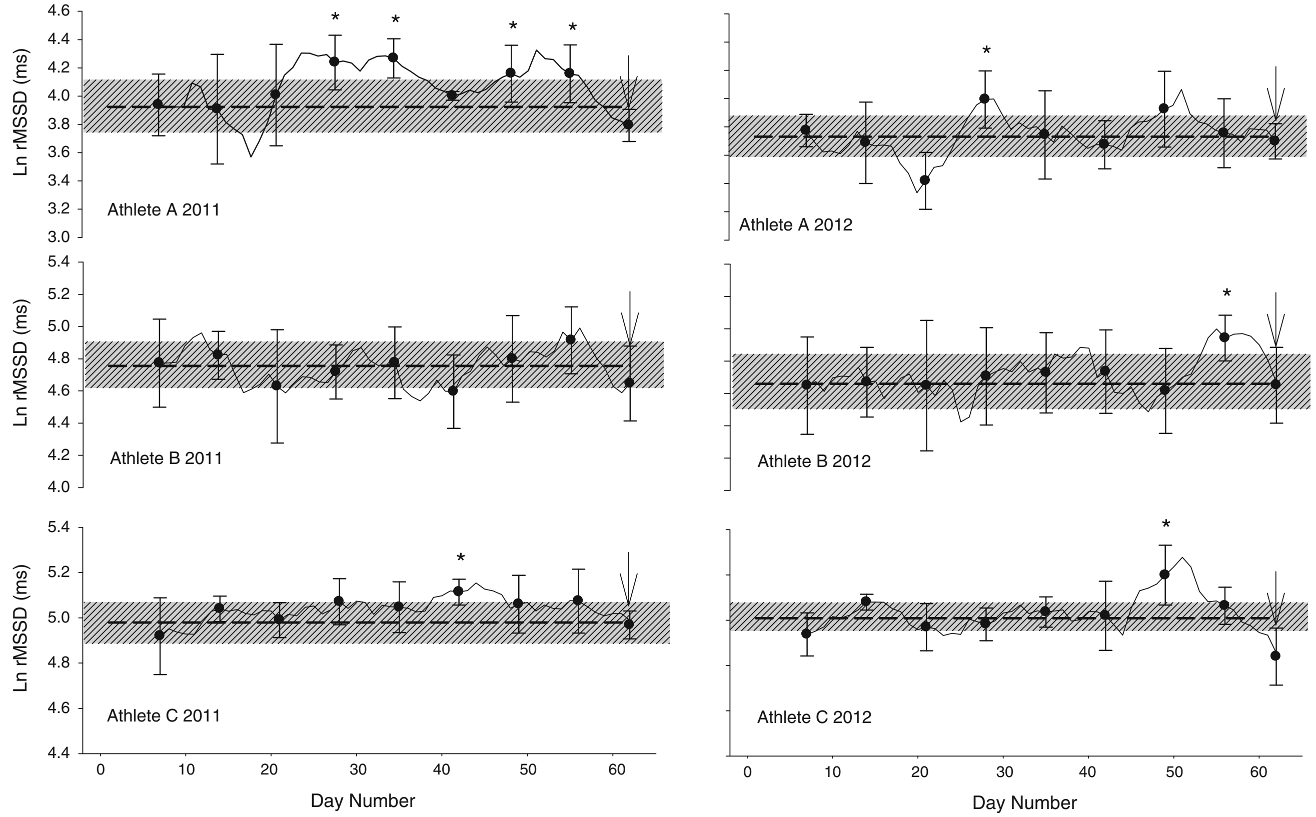

Fig. 4 The morning resting weekly averaged values of the natural logarithm of the square root of the mean sum of the squared differences between *R*–*R* intervals (*Ln rMSSD*) with 90 % confidence intervals (CI) over a 62-day period leading up to the 2011 World Rowing Championships and 2012 London Olympic Games in three elite rowers. All athletes won their events and the performance was perceived to be optimal. The *black circles* indicate the weekly averaged Ln rMSSD value, while the *black line* represents the 7-day rolling average. The *arrows* indicate the day of the final (medal) race. The *grey shaded area* indicates the individual smallest worthwhile change (*SWC*) in Ln rMSSD values (see methods in reference [1]). The *black dashed line* represents the zero line of the SWC to indicate clear/unclear changes when the 90 % CI overlaps [2]. * indicates a 'clear' change in weekly averaged Ln rMSSD values above the SWC in the week/weeks prior to the race

in elites may be due to pre-competition stress. However, changes in parasympathetic activity have not been shown to be associated with pre-competition anxiety [64], and the HRV values reported here (Fig. 4) have all been averaged over 7-day periods to reduce noise. From a performance perspective, the higher background of parasympathetic activity that is associated with intensified training loads [26] may compromise cardio-acceleration during exercise, thereby limiting oxygen delivery and performance [65]. Additionally, increases in sympathetic activity have been linked to improvement in peripheral adaptations such as faster time to peak torque [66]. Therefore, it is reasonable to assume that the reduced background of parasympathetic activity/increases in sympathetic activity [15, 18] that occurs during the taper may reflect increased 'freshness' [67], and readiness to perform. However, more research is needed to establish why HRV changes in this manner during the lead up to competition in elites, and what magnitude of change may predict 'detrimental' or 'optimal' performance.

5 Conclusion

The measurements of vagal-related indices of HRV remain promising tools for the monitoring of training status in endurance sports. However, it is clear that HRV responses are individual and dependent on fitness level and training history. As such, and although the data presented in this paper focused on elite athletes, the HRV response in any athlete with a long history of training will likely be similar to that reported here (moderately trained or elite). Accordingly, it is important to be aware of the different responses of these variables and the athlete being monitored. In this current opinion, we suggest that longitudinal monitoring is required to understand each athlete's optimal HRV to *R*–*R* interval fingerprint (i.e., Fig. 3). The possible indices of HRV that are practically useful for monitoring training status in elite athletes include weekly and 7-day rolling averaged Ln rMSSD, and the Ln rMSSD to *R*–*R* interval ratio, using the individual SWC to represent a meaningful change in fatigue and/or fitness [1]. Further, we encourage practitioners to use just one

HRV index for analysis; research suggests Ln rMSSD provides the most reliable and practically applicable measure for day-to-day monitoring. In the case of elite athletes, increasing HRV values (as competition approaches) may be a sign of positive adaptation and/or coping with training load, while reductions in HRV in the week/days before pinnacle events may represent increasing freshness and readiness to perform. Further research is needed to confirm this initial finding and gain a clearer understanding of how changes in HRV relate to training intensity distribution [25], as well as to describe further the HRV trends for elite athletes leading into major competition.

Acknowledgments No sources of funding were used to assist in the preparation of this article. The authors have no conflicts of interest that are directly relevant to the content of this article.

References

1. Plews DJ, Laursen PB, Kilding AE, Buchheit M. Heart rate variability in elite triathletes, is variation in variability the key to effective training? A case comparison. Eur J Appl Physiol. 2012;112:3729–41.
2. Hopkins WG, Marshall SW, Batterham AM, Hanin J. Progressive statistics for studies in sports medicine and exercise science. Med Sci Sports Exerc. 2009;41(1):3–13.
3. Hautala AJ, Kiviniemi AM, Tulppo MP. Individual responses to aerobic exercise: the role of the autonomic nervous system. Neurosci Biobehav Rev. 2009;33(2):107–15.
4. Buchheit M, Simpson MB. Al Haddad H, Bourdon PC, Mendez-Villanueva A. Monitoring changes in physical performance with heart rate measures in young soccer players. Eur J Appl Physiol. 2011;112:711–23.
5. Buchheit M, Papelier Y, Laursen PB, Ahmaidi S. Noninvasive assessment of cardiac parasympathetic function: postexercise heart rate recovery or heart rate variability? Am J Physiol Heart Circ Physiol. 2007;293(1):H8–10.
6. Hynynen E, Uusitalo A, Konttinen N, Rusko H. Heart rate variability during night sleep and after awakening in overtrained athletes. Med Sci Sports Exerc. 2006;38(2):313–7.
7. Hynynen E, Uusitalo A, Konttinen N, Rusko H. Cardiac autonomic responses to standing up and cognitive task in overtrained athletes. Int J Sports Med. 2008;29(7):552–8.
8. Bosquet L, Merkari S, Arvisais D, Aubert AE. Is heart rate a convenient tool to monitor over-reaching? A systematic review of the literature. Br J Sports Med. 2008;42(9):709–14.
9. Lee CM, Wood RH, Welsch MA. Influence of short-term endurance exercise training on heart rate variability. Med Sci Sports Exerc. 2003;35(6):961–9.
10. Mourot L, Bouhaddi M, Tordi N, Rouillon JD, Regnard J. Short- and long-term effects of a single bout of exercise on heart rate variability: comparison between constant and interval training exercises. Eur J Appl Physiol. 2004;92(4–5):508–17.
11. Vesterinen V, Hakkinen K, Hynynen E, Mikkola J, Hokka L, Nummela A. Heart rate variability in prediction of individual adaptation to endurance training in recreational endurance runners. Scand J Med Sci Sports. 2013;23:171–80.
12. Atlaoui D, Pichot V, Lacoste L, Barale F, Lacour JR, Chatard JC. Heart rate variability, training variation and performance in elite swimmers. Int J Sports Med. 2007;28(5):394–400.
13. Garet M, Tournaire N, Roche F, Laurent R, Lacour JR, Barthelemy JC, et al. Individual Interdependence between nocturnal ANS activity and performance in swimmers. Med Sci Sports Exerc. 2004;36(12):2112–8.
14. Buchheit M, Al Haddad H, Mendez-Villanueva A, Quod MJ, Bourdon PC. Effect of maturation on hemodynamic and autonomic control recovery following maximal running exercise in highly trained young soccer players. Front Physiol. 2011;2:69.
15. Manzi V, Castagna C, Padua E, Lombardo M, D'Ottavio S, Massaro M, et al. Dose–response relationship of autonomic nervous system responses to individualized training impulse in marathon runners. Am J Physiol Heart Circ Physiol. 2009; 296(6):H1733–40.
16. Pagani M, Lucini D. Can autonomic monitoring predict results in distance runners? Am J Physiol Heart Circ Physiol. 2009;296(6): H1721–2.
17. Portier H, Louisy F, Laude D, Berthelot M, Guezennec CY. Intense endurance training on heart rate and blood pressure variability in runners. Med Sci Sports Exerc. 2001;33(7):1120–5.
18. Iellamo F, Legramante JM, Pigozzi F, Spataro A, Norbiato G, Lucini D, et al. Conversion from vagal to sympathetic predominance with strenuous training in high-performance world class athletes. Circulation. 2002;105(23):2719–24.
19. 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; 17:354–81
20. Buchheit M, Chivot A, Parouty J, Mercier D, Al Haddad H, Laursen PB, et al. Monitoring endurance running performance using cardiac parasympathetic function. Eur J Appl Physiol. 2010;108(6):1153–67.
21. Iwasaki K, Zhang R, Zuckerman JH, Levine BD. Dose–response relationship of the cardiovascular adaptation to endurance training in healthy adults: how much training for what benefit? J Appl Physiol. 2003;95(4):1575–83.
22. Pichot V, Busso T, Roche F, Garet M, Costes F, Duverney D, et al. Autonomic adaptations to intensive and overload training periods: a laboratory study. Med Sci Sports Exerc. 2002;34(10): 1660–6.
23. Pichot V, Roche F, Gaspoz JM, Enjolras F, Antoniadis A, Minini P, et al. Relation between heart rate variability and training load in middle-distance runners. Med Sci Sports Exerc. 2000;32(10): 1729–36.
24. Kaikkonen P, Rusko H, Martinmaki K. Post-exercise heart rate variability of endurance athletes after different high-intensity exercise interventions. Scand J Med Sci Sports. 2008;18(4): 511–9.
25. Seiler S, Haugen O, Kuffel E. Autonomic recovery after exercise in trained athletes: intensity and duration effects. Med Sci Sports Exerc. 2007;39(8):1366–73.
26. Buchheit M, Simon C, Piquard F, Ehrhart J, Brandenberger G. 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(6):H2813–8.
27. Mourot L, Bouhaddi M, Perrey S, Rouillon JD, Regnard J. Quantitative Poincare plot analysis of heart rate variability: effect of endurance training. Eur J Appl Physiol. 2004;91(1):79–87.
28. Yamamoto K, Miyachi M, Saitoh T, Yoshioka A, Onodera S. Effects of endurance training on resting and post-exercise cardiac autonomic control. Med Sci Sports Exerc. 2001;33(9):1496–502.
29. Meeusen R, Duclos M, Gleeson M, Steinacker J, Rietjens G, Urhausen A. Prevention, diagnosis and treatment of overtraining syndrome. Eur J Sport Sci. 2006;6:1–14.
30. Meeusen R, Duclos M, Foster C, Fry A, Gleeson M, Nieman D, et al. Prevention, diagnosis, and treatment of the overtraining syndrome: joint consensus statement of the European College of

Sport Science and the American College of Sports Medicine. Med Sci Sports Exerc. 2013;45(1):186–205.

31. Hedelin R, Wiklund U, Bjerle P, Henriksson-Larsen K. Cardiac autonomic imbalance in an overtrained athlete. Med Sci Sports Exerc. 2000;32(9):1531–3.
32. Uusitalo ALT, 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(1):45–53.
33. Uusitalo AL, Uusitalo AJ, Rusko HK. Endurance training, overtraining and baroreflex sensitivity in female athletes. Clin Physiol. 1998;18(6):510–20.
34. Bosquet L, Papelier Y, Leger L, Legros P. Night heart rate variability during overtraining in male endurance athletes. J Sports Med Phys Fitness. 2003;43(4):506–12.
35. Hedelin R, Kentta G, Wiklund U, Bjerle P, Henriksson-Larsen K. Short-term overtraining: effects on performance, circulatory responses, and heart rate variability. Med Sci Sports Exerc. 2000;32(8):1480–4.
36. Halson SL, Jeukendrup AE. Does overtraining exist? An analysis of overreaching and overtraining research. Sports Med. 2004;34(14): 967–81.
37. Baumert M, Brechtel L, Lock J, Hermsdorf M, Wolff R, Baier V, et al. Heart rate variability, blood pressure variability, and baroreflex sensitivity in overtrained athletes. Clin J Sport Med. 2006;16(5):412–7.
38. Kuipers H. Training and overtraining: an introduction. Med Sci Sports Exerc. 1998;30(7):1137–9.
39. Kuipers H, Keizer HA. Overtraining in elite athletes: review and directions for the future. Sports Med. 1988;6(2):79–92.
40. Bosquet L, Gamelin FX, Berthoin S. Is aerobic endurance a determinant of cardiac autonomic regulation? Eur J Appl Physiol. 2007;100(3):363–9.
41. Al Haddad H, Laursen PB, Chollet D, Ahmaidi S, Buchheit M. Reliability of resting and postexercise heart rate measures. Int J Sports Med. 2011;32(8):598–605.
42. Plews DJ, Laursen PB, Kilding AE, Buchheit M. Evaluating training adaptation with heart rate measures: a methodological comparison. Int J Sports Physiol Perform. Epub 8 Mar 2013.
43. Stanley J, Peake JM, Buchheit M. Consecutive days of cold water immersion: effects on cycling performance and heart rate variability. Eur J Appl Physiol. 2013;113:371–84.
44. Penttila J, Helminen A, Jartti T, Kuusela T, Huikuri HV, Tulppo MP, et al. Time domain, geometrical and frequency domain analysis of cardiac vagal outflow: effects of various respiratory patterns. Clin Physiol. 2001;21(3):365–76.
45. Hamilton RM, McKechnie PS, Macfarlane PW. Can cardiac vagal tone be estimated from the 10-second ECG? Int J Cardiol. 2004;95(1):109–15.
46. Aubert AE, Seps B, Beckers F. Heart rate variability in athletes. Sports Med. 2003;33(12):889–919.
47. Laursen PB. Training for intense exercise performance: high-intensity or high-volume training? Scand J Med Sci Sports. 2010;20(Suppl. 2):1–10.
48. Fiskerstrand A, Seiler KS. Training and performance characteristics among Norwegian international rowers 1970–2001. Scand J Med Sci Sports. 2004;14(5):303–10.
49. Tucker R, Collins M. What makes champions? A review of the relative contribution of genes and training to sporting success. Br J Sports Med. 2012;46(8):555–61.
50. Barnett A. Using recovery modalities between training sessions in elite athletes: does it help? Sports Med. 2006;36(9):781–96.
51. Goldberger JJ, Ahmed MW, Parker MA, Kadish AH. Dissociation of heart rate variability from parasympathetic tone. Am J Physiol Heart Circ Physiol. 1994;266:H2152–7.
52. Goldberger JJ, Challapalli S, Tung R, Parker MA, Kadish AH. Relationship of heart rate variability to parasympathetic effect. Circulation. 2001;103(15):1977–83.
53. Kiviniemi AM, Hautala AJ, Seppanen T, Makikallio TH, Huikuri HV, Tulppo MP. Saturation of high- frequency oscillations of *R*–*R* intervals in healthy subjects and patients after acute myocardial infarction during ambulatory conditions. Am J Physiol Heart Circ Physiol. 2004;287(5):H1921–7.
54. Hedman AE, Hartikainen JE, Tahvanainen KU, Hakumaki MO. The high frequency component of heart rate variability reflects cardiac parasympathetic modulation rather than parasympathetic 'tone'. Acta Physiol Scand. 1995;155(3):267–73.
55. Malik M, Camm AJ, Amaral LA, Goldberger AL, Ivanov P, Stanley HE, et al. Components of heart rate variability: what they really mean and what we really measure. Am J Cardiol. 1993; 72:821–2.
56. Sacknoff D, Gleim G, Stachenfeld N, Glace B, Coplan N. Suppression of high-frequency power spectrum of heart rate variability in well-trained endurance athletes. Circulation. 1992; 86:I-658.
57. Borresen J, Lambert MI. Autonomic control of heart rate during and after exercise: measurements and implications for monitoring training status. Sports Med. 2008;38(8):633–46.
58. Hautala A, Tulppo MP, Makikallio TH, Laukkanen R, Nissila S, Huikuri HV. Changes in cardiac autonomic regulation after prolonged maximal exercise. Clin Physiol. 2001;21(2):238–45.
59. Iellamo F, Pigozzi F, Spataro A, Di Salvo V, Fagnani F, Roselli A, et al. Autonomic and psychological adaptations in Olympic rowers. J Sports Med Phys Fitness. 2006;46:598–604.
60. Mujika I, Goya A, Padilla S, Grijalba A, Gorostiaga E, Ibanez J. Physiological responses to a 6-d taper in middle-distance runners: influence of training intensity and volume. Med Sci Sports Exerc. 2000;32(2):511–7.
61. Buchheit M, Laursen PB, Al Haddad H, Ahmaidi S. Exercise-induced plasma volume expansion and post exercise parasympathetic reactivation. Eur J Appl Physiol. 2009;105(3):471–81.
62. Convertino VA. Blood volume: its adaptation to endurance training. Med Sci Sports Exerc. 1991;23(12):1338–48.
63. Kaikkonen P, Nummela A, Rusko H. Heart rate variability dynamics during early recovery after different endurance exercises. Eur J Appl Physiol. 2007;102(1):79–86.
64. Iellamo F, Pigozzi F, Parisi A, Di Salvo V, Vago T, Norbiato G, et al. The stress of competition dissociates neural and cortisol homeostasis in elite athletes. J Sports Med Phys Fitness. 2003;43(4):539–45.
65. Parouty J, Al Haddad H, Quod M, Lepretre PM, Ahmaidi S, Buchheit M. Effect of cold water immersion on 100-m sprint performance in well-trained swimmers. Eur J Appl Physiol. 2010;109:483–90.
66. Hedelin R, Bjerle P, Henriksson-Larsen K. Heart rate variability in athletes: relationship with central and peripheral performance. Med Sci Sports Exerc. 2001;33(8):1394–8.
67. Bannister EW, editor. Modeling elite athletic performance. Champaign: Human Kinetics; 1991.