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Heart Rate Variability in Elite Synchronized Swimmers

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Purpose: To determine whether heart rate (HR) variability (HRV) was correlated with other training load and training tolerance markers for monitoring the effect of a training session on elite synchronized swimmers. **Methods:** The authors recorded the resting HRVs of 12-elite swimmers (mean age = 21.5 [3.5] y) over 1 week with a cadence of 48 hours prior to the 2015 World Swimming Championships. The authors continuously monitored heart rate and obtained salivary cortisol (SC) samples before and after the last training session of the week. The authors measured capillary blood lactate (La_{peak}) 2, 4, and 8 minutes after the last training session and monitored recovery HRV. The authors assessed rating of perceived exertion over the entire session, and the authors tested the association between La_{peak} , SC, and rating of perceived exertion and relative changes ($\Delta\%$) in the natural logarithm of the root mean square successive difference of intervals (LnRMSSD). The authors also calculated the smallest worthwhile change of the averaged pre and post LnRMSSD measurements. **Results:** There were periods of pronounced bradycardia (60.5 [16.7] bpm) during training exercises corresponding to apneic exercise. The magnitude-based inferences showed nonclinically meaningful changes of LnRMSSD. La_{peak} (6.8 [2.7]) related positively with $\Delta\%$ LnRMSSD and $\Delta\%$ SC. **Conclusions:** There was no change in LnRMSSD and La_{peak} , $\Delta\%$ SC, and rating of perceived exertion indicated reduced sympathetic activation and positive adaptation to the stress imposed by the session. Isolated HRV assessment may reveal a controversial interpretation of the autonomic nervous system status or the training tolerance in elite synchronized swimming athletes that are influenced by training response.

Keywords: aquatics, apnea, autonomic nervous system, synchronized swimming, monitoring load

Synchronized swimming (SS) is an Olympic sport involving routines that are technically and physically very demanding and require intense whole-body work that is performed during apneic episodes^{1,2} interspersed by short breathing intervals.² During the voluntary apnea periods, which constitute up to 50% of the routine time,³ oxygen is mobilized from finite stores in the lungs, blood, and other tissues and the cardiovascular diving response restricts blood flow to selected regions and reduces heart rate (HR) and cardiac output.³ Elite SS athletes perform 2 training sessions (TS) per day, and the total training load is roughly 40 hours per week.^{2,4} With only a few hours of recovery between a TS and few days without training, physical recovery is hampered.⁴ Therefore, monitoring the recovery of SS athletes is important for determining whether an athlete is adapting to the training program and minimizing his or her risk of nonfunctional overreaching, injury, and illness.⁴

In the last decade, studies have attempted to address the physiological responses of SS athletes during different types of training.^{5,6} The variables that have been most frequently investigated include blood lactate (La) concentration,^{3,5} HR,^{1,2,7} rating of perceived exertion (RPE),¹ oxygen consumption,⁶ and salivary cortisol (SC).⁸ However, it is difficult to make any generalizations about these results given the differences in the tests conducted and their methodologies.

Heart rate variability (HRV) describes the variation in beat-by-beat intervals in different situations.⁹ Several studies have used HRV to monitor individual recovery and training adaptations to better understand the status of the autonomic nervous system, stress, and recovery balance in elite athletes.^{4,10,11} The natural logarithm of the root mean square successive difference of intervals (LnRMSSD) is the most recommended applicable variable of the time domain of HRV indices to assess training tolerance and/or performance level.^{10,12}

A measurement of HRV at rest or after exercise has been suggested to indicate both positive and negative adaptations to training.^{10,12} The increases in root mean square successive difference that elite athletes exhibit in the weeks before their main event are likely associated with positive adaptation or coping with training load, and reductions in root mean square successive difference during tapering may represent increasing readiness to perform.¹¹ However, it is unlikely that HRV will increase in elite athletes when the load is reduced because taper leading into competition typically consists of reductions in training volume with the maintenance of intensity. Therefore, these changes in training load will attenuate HRV reductions.^{11,13}

There is a lack of research pertaining to why elite athletes exhibit different behavior of HRV than recreational or amateurs prior to competition and how HRV correlates with other training markers. Therefore, the aim of the present study was to determine whether monitoring LnRMSSD contributes information about the physiological stress imposed by TS and how LnRMSSD correlates with other training load markers (La and RPE) and other training tolerance markers such as SC during tapering. We hypothesized that LnRMSSD would increase during the recovery period and be positively correlated with other physiological markers such as La, RPE, and SC. This trend, if verified, would suggest that SS athletes were coping with the applied training load before a competition.

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Methods

Subjects

A total of 12 elite female SS athletes, members of the senior Spanish national SS team, volunteered for this study. The mean age of the cohort was 21.5 (3.5) years. The mean height and body mass of the group were 170.1 (5.8) cm and 56.3 (5.7) kg, respectively. The average body mass index of the group was 19.3 (1.0) kg/m². The study was approved by the local research ethics committees and conducted in accordance with the Declaration of Helsinki. The swimmers were provided with oral and written information about the study procedures before giving their informed consent, and parental permission was obtained for subjects under 18 years of age.

Design

The study was an observational research conducted over the course of 2 weeks 1 month prior to the 2015 World Swimming Championships.

Experimental Overview

We monitored the swimmers' baseline and recovery HRV in their training facilities 3 times per week across 2 weeks for consistency¹² and during the same menstrual phase to avoid the influence of hormonal disturbances.⁴ We divided the team into 2 groups according to the menstrual phase, and each group was assessed during the same week. We assessed baseline HRV in a supine position for a 10-minute period. The baseline measurements were interspersed by 48 hours, and the averaged HRV during that week was considered to be the baseline HRV value (Rest). Training load markers (La and RPE) and training tolerance markers (SC and HRV during recovery [Rec]) were assessed on the last TS of that week. All of the measurements and the TS were performed in the team's training facilities in a 50-m indoor pool with a water temperature of 25°C to 26°C.

The session consisted of a standardized warm-up followed by roughly 4 hours of specific training exercises. The exercises were organized by parts of the technical team routine (TT). There was no rest period during the session, and the swimmers only exited the water to practice the first part of the TT routine. The TS concluded with the execution of a complete TT (3 min and 3 s).

Baseline SC (SC_{Rest}) and HRV assessments were obtained 30 minutes before the TS after the athletes had taken a nap in the same testing room at the side of the pool. This procedure ensured that stressor events that may have been overcome by a morning waking measure were avoided.¹⁰ We continuously monitored HR before,

during, and after the TS. About 2, 4, and 8 minutes after the execution of the TT, we took capillary La samples from the swimmers' earlobes. Just after taking the La samples, the swimmers were asked to walk to a quiet room next to the pool. Fifteen minutes after the TT, the SC samples (SC_{post}) were obtained, and we started the HRV assessment. The Rec HRV assessments were performed from 15 to 35 recovery minutes. The averaged recovery HRV measured during the last 2 sessions of the assessed week was used in our analyses. We inquired about the entire TS RPE when the participants completed the Rec assessment (~35 min after TT; Figure 1).

We measured HR using waterproof Suunto Memory Belt HR monitors (Suunto Oy, Vantaa, Finland) that record HR beat by beat. These devices were placed on each swimmer's chest and removed after the Rec assessment. To minimize potential instrumentation bias, swimmers wore their HR monitors for the TSs that occurred 1 week prior to the last testing session.^{2,7} We assessed HR from rate-to-rate intervals and transferred the measurements to the Suunto Movescount App (version 2.4.5; Suunto Oy), which interpolated the data every 2 seconds.

We assessed recovery HRV after the TS and during the last 20 minutes of the recovery period. We excluded the first 5 minutes from the analysis due to the modulation mechanism of the autonomic transition from sympathetic to parasympathetic activation.¹² The measurements were averaged every 5 minutes: HRV during the recovery period 20 to 25 minutes (Rec₂₀₋₂₅); HRV during the recovery period 25 to 30 minutes (Rec₂₅₋₃₀); and HRV during the recovery period 30 to 35 minutes (Rec₃₀₋₃₅). We analyzed only LnRMSSD because it is the most practically applicable and reliable of the spectral indices.¹¹ We used Kubios software (version 2.0; Kubios Oy, Finland) for the HRV analysis.

We obtained capillary blood samples from each swimmer's earlobe using a portable device (Lactate Pro LT-1710; Arkray, Inc, Kyoto, Japan); the highest value of La concentration (La_{peak}) was considered in the analyses.

Endocrine responses to training were measured via SC (in nanograms per milliliters). Two saliva samples (4 mL) were collected using Salivettes (Sarstedt Ltd, Nümbrecht, Germany). For the SC sampling, all of the swimmers were instructed to avoid the use of dental floss and toothpaste and not to ingest a major meal for 1 hour prior to the TS and to avoid alcohol and caffeine intake for 24 hours prior to the TS. Changes in SC expressed as a percentage difference between the pre- and post-TS values ($\Delta\%$ SC) were considered in the analysis. The samples were centrifuged at 3000 rpm for 10 minutes and stored at -20°C until analysis. We determined SC levels using an automated electrochemiluminescence immunoassay (Modular E; Roche Diagnostics GmbH, Mannheim, Germany); the analytical sensitivity of the assay was 0.054 µg/dL, and the interassay coefficient of variation was 4.5%.

Figure 1 — Testing protocol. SC_{Rest} indicates baseline salivary cortisol; HRV_{Rest}, baseline heart rate variability; TT, technical routine; HR, heart rate; La, capillary blood lactate concentration; SC_{Post}, salivary cortisol after the training session; Rec₂₀₋₂₅, HRV during the recovery period 20 to 25 minutes; Rec₂₅₋₃₀, HRV during the recovery period 25 to 30 minutes; Rec₃₀₋₃₅, HRV during the recovery period 30 to 35 minutes; RPE, rating of perceived exertion.

We used the CR-10 Borg scale to determine RPE for the entire TS.¹ A colored, verbally anchored scale was shown to the swimmers after the HRV assessment. All of the athletes received instruction about the scale interpretation, and they were assessed repeatedly during at least 3 TSs to account for learning effects and to improve the consistency of the measurements.^{1,7} The session RPE was calculated by multiplying the duration of the TS by the RPE.¹

Statistical Analysis

The data are presented as mean and (SD). To satisfy the normality assumptions, LnRMSSD was calculated. The differences between the Rest LnRMSSD and the Rec_{20–25}, Rec_{25–30}, and Rec_{30–35} outcomes were analyzed using 1-way analysis of variance for repeated measures with Bonferroni post hoc comparisons and statistical significance set at $P < .05$. The data were also analyzed using effect size Cohen's d and interpreted as follows: <0.2 , trivial; 0.2 to 0.6 , small; 0.6 to 1.2 , moderate; >1.2 , large.¹²

To analyze the variation of within-swimmers recovery, we calculated the smallest worthwhile change in LnRMSSD, using the between-swimmers standard deviation (SD) of the averaged baseline pretraining variable (eg, 0.5 multiplied by the between-swimmer SD of the mean LnRMSSD values baseline pre-TS). It has been suggested that a change of **more or less than** of an athletes' is worthwhile, and should, therefore, be deemed of interest.¹⁴ This was calculated to assess substantial changes in LnRMSSD **posttraining** using an approach based on magnitudes of change. Quantitative chances of changes in LnRMSSD values **posttraining** also evaluated qualitatively as follows: 25% to 75%, possibly; 75% to 95%, likely; 95% to 99%, very likely; $>99\%$ almost certain. If the chance of higher or lower differences was $>5\%$, then the true difference was assessed as unclear.¹⁴

We used a paired t test to assess the differences between the mean values of SC_{Rest} and SC_{post}. To isolate the effect of the TS on the LnRMSSD, this analysis was performed on the difference between Rest and the mean of 3 Rec periods (Rec_{20–25}, Rec_{25–30}, and Rec_{30–35}) represented as relative changes (ie, $\Delta\%$ LnRMSSD).

Pearson's correlation was used to compare the relation between $\Delta\%$ LnRMSSD, La_{peak}, $\Delta\%$ SC, and RPE. We adopted a level of statistical significance of $P < .05$.

Results

Table 1 lists the individual and group HR values before, during, and after both the TS and the TT. Complete data sets were obtained for only 8 from 12 swimmers for HR during TS; the missing values were due to failed or poor-quality HR recordings. Figure 2 shows an example of an HR response profile, where the athlete's HR quickly increased to high levels of tachycardia interspersed with periods of intense bradycardia during the exercise bouts performed in apnea.

Analysis of variance revealed no significant differences in LnRMSSD as a function of time (LnRMSSD [$F_{3,9} = 1.97$; $P = .18$]). The effect size between baseline LnRMSSD and each recovery period ranged from trivial (0.17) to small (-0.40) (Table 2). Most of the individual smallest worthwhile change of the LnRMSSD was from possibly negative to possibly trivial (Figure 3).

The average RPE from the last TS of the week was 6.3 (1.8); the data ranged from 4 to 9 a.u. The average session RPE from the same TS was 1520 (426) a.u. and ranged from 1008 to 2268 a.u. The average La_{peak} was 6.8 (2.7) mmol/L. On average, SC_{Rest} was 0.2 (0.1) and SC_{post} was 0.3 (0.2); there were no significant differences between these measurements ($T = -0.98$, $P = .52$). We noted a moderate effect size for the 2 measurements: $d = -0.45$ (-1.14 [0.28]).

The La_{peak} was correlated positively with $\Delta\%$ SC ($r = .89$, $P = .001$), $\Delta\%$ LnRMSSD ($r = .61$, $P = .04$), and no other correlations were found.

Discussion

This study is the first in which HR was continuously monitored during an entire TS in elite SS athletes previous to the main event of the season. Previous studies monitored only a single routine.^{1,2,7}

Table 1 Heart Rate Parameters Before (Pre) and After (Post) the Entire TS As Well As During Performance, Both During the T and the TT

Subject	Pre		Performance										Post		
	HR _{pre}		HR _{peak}		HR _{mean}		HR _{mean} ^a		HR _{min}		HR _{range}		HR _{post3}		
	T	TT	T	TT	T	TT	T	TT	T	TT	T	TT	HR _{post1}	TS	HR _{post5}
1	64	118	130	144	90	138	62.5%	95.8%	65	130	65	14	145	91	86
2	68	131	164	147	113	144	69%	87.6%	70	140	94	7	157	88	84
3	45	80	158	163	89	148	54.5%	90.9%	34	142	124	21	139	76	72
4	43	76	127	152	84	144	55.2%	94.5%	34	137	93	15	143	90	81
5	73	119	164	162	114	148	69.2%	90.2%	70	139	94	23	150	95	92
6	81	109	133	147	109	144	74.4%	97.8%	76	141	57	6	156	99	97
7	73	119	138	144	108	138	75.2%	96%	67	134	71	10	144	87	83
8	68	129	162	162	111	150	68.7%	92.5%	68	138	94	24	142	83	79
Mean	64.4	110	147	152.6	102.3	144.2	66.1%	93.2%	60.5	137.6	86.5	15	147	88.6	84.3
(SD)	(13.5)	(21.0)	(16.4)	(8.4)	(12.4)	(4.4)	(8%)	(3.5%)	(16.7)	(4)	(21.3)	(7.1)	(6.6)	(7.1)	(7.7)

Q5 Abbreviations: HR_{peak}, HR_{mean}, HR_{min}, and HR_{range}: heart rate peak, mean, minimum, and range values during performance; HR_{post1}, HR_{post3}, and HR_{post5}: heart rate after first, third, and fifth minutes during recovery; HR_{pre}: last minute before starting the exercise; TS, training session; TT, technical team routine.

^aExpressed as % of HR_{peak}. The data are grouped per subject.

Figure 2 — Heart rate profile during a training session within the competitive period including the technical team routine on an Olympic and world medalist. Line depicts smoothed 6-second averaged values for clarity. HR_{peak} indicates heart rate peak during the routine; HR_{range} , heart rate difference between the minimum heart rate and the maximum value during the routine; HR_{min} , minimum heart rate during the routine; HR_{mean} , the average heart rate during the routine.

Moreover, the data are ecologically valid and obtained in very high-caliber athletes who have won Olympic and World Champ medals.

The pattern of HR response was characterized by the athletes attaining their peak HR during the execution of the competitive TT routine; there were interspersed periods of marked bradycardia in the middle of the TS corresponding to exercises in apnea.

Postexercise LnRMSSD was characterized by unchanged autonomic nervous system activity as the SS athletes had trivial smallest worthwhile change and only 3 SS athletes presented possibly negative and likely positive changes (Figure 3). The RPE scores and the La_{peak} could indicate that the stress imposed by both the TS and

the TT was moderate. Nevertheless, La_{peak} was similar in value to the data reported after competition for elite SS athletes.^{3,7} Moreover, the lack of differences between pre-SC and post-SC are also related with the decreased sympathetic tone during the recovery.

A positive relation was found between La_{peak} and $\Delta\%$ LnRMSSD, and $\Delta\%$ SC, which are likely associated with the intense exercise bouts performed in apnea. We suggested that the unsubstantial changes in LnRMSSD after TS (Figure 2) can lead to a misinterpretation of the “true” change in ANS is due to the effect of the diving response because the tapering sessions of elite SS athletes include up to 50% of the routine and some of the exercise time is spent in apnea.³

Table 2 Cardiac Autonomic Responses and Its ESs Between the Baseline Conditions (Rest) and the 3 Recovery Periods (Rec_{20–25}, Rec_{25–30}, and Rec_{30–35})

	Rest	Rec _{20–25}	Rec _{25–30}	Rec _{30–35}	Rest vs Rec _{20–25} , ES (90% CI); rating	Rest vs Rec _{25–30} , ES (90% CI); rating	Rest vs Rec _{30–35} , ES (90% CI); rating
LnRMSSD, ms	3.54 (0.65)	3.65 (0.69)	3.29 (0.53)	3.30 (0.55)	0.17 (–0.51 to 0.83); trivial	–0.40 (–1.07 to 0.29); small	–0.40 (–1.06 to 0.30); small

Abbreviations: CI, confidence interval, ES, effect size; HRV, heart rate variability; LnRMSSD, natural logarithm of the root mean square successive difference of intervals. Rec_{20–25}, HRV during the recovery period 20 to 25 minutes; Rec_{25–30}, HRV during the recovery period 25 to 30 minutes; Rec_{30–35}, HRV during the recovery period 30 to 35 minutes. Note: Values are presented as mean (SD). Significant differences ($P < .05$). For ES interpretation: <0.2 , trivial; >0.2 to 0.6 , small; >0.6 to 1.2 , moderate; and >1.2 , large.

Figure 3 — Magnitude-based inferences for negative (sympathetic autonomic nervous system predominance) or positive (parasympathetic autonomic nervous system predominance). The data show the individual substantial changes in LnRMSSD as (mean [post-pre] $\pm$ 90% confidence limits). The smallest worthwhile change was set at 0.5 of the baseline averaged measurements SD. LnRMSSD indicates natural logarithm of the root mean square successive difference of intervals.

A remarkably high preactivation HR was observed for all swimmers before the TT was performed (Table 1). As suggested previously,² this variation in HR before starting the TT routine may be explained by previous body work (ie, 4 h of training) with a short recovery (~5 min) before the performance, the sympathetic activation and parasympathetic withdrawal necessary to ensure anticipatory metabolic and cardiovascular responses to a physical effort,¹⁵ and the mental stress and anxiety associated with competitive performance proximity.¹⁶

In humans, the physiological response to immersion is called the diving response,^{17,18} which is triggered by chilling of the face and holding of the breath.^{17,18} This reflex is characterized by a reduction in HR or bradycardia, a decrease in cardiac output, a redistribution of blood flow due to peripheral vasoconstriction and an increase in mean arterial blood pressure.¹⁸ The diving response is initiated when the head of the human is submerged.¹⁸ In elite SS athletes this response is powerful enough to override the HR response to exercise during apnea.² Although apnea and facial immersion increase the parasympathetic tone and cause bradycardia, exercise increases sympathetic stimulation of the heart and increases HR.^{17,19} However, during the apneic bouts, both inputs compete with one another for control of HR,¹⁹ and the O₂ conservation diving response finally prevails until the swimmer is able to breathe again.² In our investigation, swimmers attained a minimum HR of 60.5 (16.7) beats per minute (average) (Table 1), representing a 42% HR reduction compared with their mean HR value. These findings are virtually identical to those found during the execution of competitive performances in elite SS athletes.^{1,2,7}

On the other hand, La_{peak} was positively correlated with $\Delta\%$ LnRMSSD (Table 3). This may be explained by the effect of the diving response mentioned above. Although there is a progressive accumulation of lactate due to the peripheral vasoconstriction and the activation of the glycolytic metabolism in the exercising muscles,^{2,3} the increased vagal activity that causes bradycardia and that is mediated by the parasympathetic efferent pathway should have increased the LnRMSSD 20 minutes after TS. Nevertheless, the LnRMSSD did not change for most of the swimmers. These results confirm, for elite SS athletes, what has been previously described in patients with bradycardia²⁰ and scuba divers.^{17,21}

The pronounced bradycardias reported in our swimmers might at least partly explain the trivial increased parasympathetic tone

Table 3 The Relationship Between the Training Load Markers (RPE and La_{peak}) and the Training Tolerance Markers ($\Delta\%$ Cortisol and $\Delta\%$ LnRMSSD)

	La _{peak} , mmol/L	$\Delta\%$ Cortisol	$\Delta\%$ LnRMSSD
RPE, a.u.	0.23	0.23	-0.46
La _{peak} , mmol/L		0.89*	0.61*
$\Delta\%$ Cortisol			0.41

Abbreviations: La_{peak}, capillary peak blood lactate concentration during recovery; RPE, rating of perceived exertion; $\Delta\%$ Cortisol, salivary cortisol changes expressed in percentage when comparing pre and post values; $\Delta\%$ LnRMSSD, relative changes of the natural logarithm of the square root of the mean sum of the squared differences between RR.

*Significance ($P < .05$).

in Rec_{20–25}. Contrarily, LnRMSSD did not significantly increase during Rec_{20–25} and the qualitative inferences for positive and negative changes of LnRMSSD were only “possibly” (Table 2 and Figure 3). Six from 12 SS athletes showed “possibly” trivial changes, 4 of them showed “possibly” negative changes and only 2 had “likely” positive changes. This result is contradictory to the findings of previous studies conducted with dryland athletes, where parasympathetic modulation was reflected by a significant decreased HRV 30 minutes after running exercises.²² We suggest that the bradycardic response as a result of the apneic work,¹⁸ triggered by an increased vagal tone with concurrent inhibition of the sympathetic outflow,¹⁹ would have not only prevailed during the performance but also influenced the periodic fluctuations in HR during the recovery period.²¹

Another important factor that may have influenced the unchanged LnRMSSD was the effect of the training practice: a lower HR during recovery is a specific adaption in trained SS athletes.²³ The increased LnRMSSD at low HR is linked to the fact that vagal-related HRV indices more strongly reflect the magnitude of modulation in parasympathetic outflow rather than an overall parasympathetic tone per se. 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.^{11,24} Therefore, it appears that the decreased HR_{rec} of our swimmers (Table 1) might explain the similar LnRMSSD values obtained across the entire recovery period (Table 2).

One might perhaps believe that the TL was applied could not have caused significant stress on the autonomic cardiac modulation during the competitive period,²⁵ as typically occurs during the tapering period.^{11,26} However, the observed moderate values of La_{peak} similar to those found in real competition (6.8 [2.7] vs 7.1 [2.4] mmol/L, respectively)² and the low Δ%SC would indicate that our swimmers were coping well with the training stimulus during the tapering phase. As it is advised for SS athletes, the measurement of La after performances should be interpreted with caution.³ The athletes’ mostly short breathing pauses would reduce the accumulation of lactate, likely by increasing oxygen stores and aerobic metabolism and by allowing some lactate metabolism during performances.³ Training practice in SS appears to produce such adaptations, which improves effectiveness⁵ and might explain the improvements in work economy with a reduction in lactate production during the competitive period,² and a reduction of 6.25% after a 5-week.⁵

The lack of differences between pretraining and posttraining SC values observed in the present study may be related to the athletes’ adaptation to the workload.²⁷ Several studies of aquatic sports have reported marked reductions in SC levels during the tapering phase.^{8,27,28} Based on the fact that SC concentration is subject to psychological stress,²⁹ the physical stress induced by the previous intensive training may have been replaced during recovery by the psychological stress induced by the oncoming national championships.²⁷ Another possible explanation for the lack of differences in SC may be directly related to the decreased sympathetic tone during the recovery. The reduced sympathetic adrenal response to exercise, typically related to higher levels of fatigue in this kind of swimmer,⁴ could have been attenuated by the effect of the vagal nervous system as a result of the apneic work. In this vein, Schaal et al⁴ reported similar results in 10 elite synchronized swimmers 11 weeks prior to their qualifying for the 2012 Olympic Games. Furthermore, Bonifazi et al²⁸ noted a decrease in the SC levels of 18 elite swimmers competing in a 50-m sprint and a

middle-distance race (the most similar disciplines to SS) before the main competitions of the season. In our investigation, we noted a positive association between La_{peak} and Δ%SC (Table 3). We speculate that the decrease in SC represents an expression of the training adaptation that leads to an enhancement in anaerobic metabolism.²⁸

Certain issues and limitations regarding the design, methodology, and overall validity of this study need to be considered. To preserve the ecological and external validity of the design, there were restrictions imposed by the coaches in terms of testing so as not to disturb the athletes and their preparations for the World Championship. It was accordingly not possible to ensure quiet stationary conditions to assemble more than 2 weeks of HRV measurements.^{11,12} Moreover, we had to divide the team into 2 groups according to the menstrual phase, which made it difficult to obtain more than 3 baseline measurements of HRV during the same week. In addition, to better understand the autonomic nervous system balance in apneic sports, the duration of immersions (during performances) and the respiratory rate (during the recovery) should be recorded.

Practical Applications

Our findings highlight that HRV during recovery may be influenced by the execution of apneas. We encourage SS coaches to apply individual longitudinal monitoring of HRV together with time of immersion to more clearly understand how trends in HRV relate to training distribution and autonomic balance. We recommend combining HRV assessments with other physiological markers to determine whether demands from TS stimuli are in accordance with official competitive outputs.

Conclusions

Monitoring the effect of the training load and training stress from 15 to 35 minutes after the TS revealed no changes in LnRMSSD among elite SS athletes. The interpretation of LnRMSSD in elite SS athletes is compromised by the exercise bouts performed in apnea and the high level of expertise and the training background of these individuals. Additional markers are necessary to better understand autonomic nervous system status and monitor the effect of training in apneic sports disciplines.

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Queries

- Q1.** Please check if “Ramon Llull University” is correct.
- Q2.** Please ensure author information is listed correctly here and within the byline.
- Q3.** Please provide city name for “Kubios Oy.”
- Q4.** Please check the edits made to the abbreviation of “Rec_{30–35}” in Figure 1 caption.
- Q5.** Please check if the edits to Table 1 footnote are correct.
- Q6.** Please check if “h:min:seg” should be “h:min:s” in Figure 2 (ie, “seg” refers second).
- Q7.** Please spell out "ANS" at first occurrence.
- Q8.** Please spell out "RR" at first occurrence.
- Q9.** Please check if the edits made to sentence "We suggest that the bradycardic . . ." are correct.
- Q10.** Please check if the edits to the sentence “As it is advised for . . .” retain the intended meaning.
- Q11.** Originally, Refs. 17 and 23 were identical. Hence the duplicate has been removed from the reference list and the subsequent references have been renumbered both in text and in reference list. Please verify.
- Q12.** Year has been updated in Ref. 20. Please check.