J Appl Physiol 99: 1728–1735, 2005.
First published July 7, 2005; doi:10.1152/japplphysiol.00487.2005.

Hemodynamic and autonomic changes induced by Ironman: prediction of competition time by blood pressure variability

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Submitted 28 April 2005; accepted in final form 7 July 2005

Gratze, Gerfried, Richard Rudnicki, Wolfgang Urban, Harald Mayer, Alois Schlögl, and Falko Skrabal. Hemodynamic and autonomic changes induced by Ironman: prediction of competition time by blood pressure variability. *J Appl Physiol* 99: 1728–1735, 2005. First published July 7, 2005; doi:10.1152/japplphysiol.00487.2005.—We hypothesized that the extreme endurance exercise of an Ironman competition would lead to long-standing hemodynamic and autonomic changes. We investigated also the possibility of predicting competition performance from baseline hemodynamic and autonomic parameters. We have investigated 27 male athletes before competition, 1 h after, and then for the following week after the competition. The Task Force monitor was used to measure beat-to-beat hemodynamic and autonomic parameters during supine rest and active standing. Heart rate ($P < 0.001$) was increased, and stroke index ($P = 0.011$), systolic blood pressure ($P = 0.004$), diastolic blood pressure ($P < 0.001$), total peripheral resistance index ($P < 0.001$), and baroreceptor reflex sensitivity ($P < 0.001$) were decreased after the competition. The 0.05- to 0.17-Hz band of heart rate and blood pressure variability was increased ($P < 0.001$ and $P < 0.001$, respectively), the 0.17- to 0.40-Hz band of heart rate interval variability was decreased after the competition ($P < 0.001$). All parameters returned to baseline values 3 days after the competition. After the competition, the autonomic response to orthostasis was significantly impaired. The 0.05- to 0.17-Hz band of diastolic blood pressure variability before competition and weekly net exercise training, but not the other hemodynamic and autonomic parameters, were related to competition time in multivariate regression analysis (multiple $r = 0.70$, $P < 0.001$). The marked hemodynamic and autonomic changes after an ultraendurance race, which are compatible with myocardial depression in the face of sympathetic activation and reduction of afterload, return to baseline after only 1–3 days. Because the 0.05- to 0.17-Hz band of diastolic blood pressure variability contributes to the prediction of competition time, the analysis of blood pressure variability in the frequency domain deserves further study for the prediction of endurance capacity.

hemodynamics; autonomic nervous system; exercise

THE IRONMAN TRIATHLON, WHICH consists of 3.9 km of swimming, 180.2 km of cycling, and 42.2 km of running is considered as an extreme endurance load in our sedentary society, even for very well-trained athletes. Some of the hormonal and cardiac changes induced by ultradistance triathlon have been studied (7, 17). It has been reported that serum levels of catecholamines and cortisol are elevated for several hours after the triathlon (30, 32), and free testosterone concentration is significantly reduced for several days after the competition (37). It also has been reported that left ventricular function is impaired immediately after an Ironman (5). Despite the broad interest in the physiology of endurance sports, detailed hemodynamic and autonomic nervous system functions have not been studied in athletes competing for the Ironman. We hypothesized that this extreme endurance exercise would lead to long-standing hemodynamic impairment and sympathetic activation. We were also interested to see whether it is possible to predict competition performance from baseline hemodynamic and autonomic parameters. We studied hemodynamic and autonomic parameters before the competition and were prepared to study them for as long afterwards as it would take for full recovery.

METHODS

We have studied 27 healthy male athletes. All athletes participated in the Ironman in Klagenfurt, Austria. Subsequently, 2 of the 27 subjects were excluded from the analysis: one because of permanent atrial fibrillation, the other because of frequent ventricular extrasystoles on the day before the event, which precluded an analysis of the autonomic nervous system, but not a successful participation of both athletes in the competition. Clinical details of the 25 subjects are given in Table 1.

The subjects were studied on the day before (day−1), 1 h (hour+1), 1 day (day+1), and 3 and 7 days (day+3 and day+7) after the competition. The follow-up was finished at that time point, because all hemodynamic and autonomic parameters had returned to baseline levels. Two subjects were not available after the competition (hour+1), but their results were included at all other time points. All measurements were performed during 10 min of supine bed rest and subsequently during 6 min of active standing. During the first hour after the competition, subjects rested for recovery. They were allowed to drink nonalcoholic fluids ad libitum. Body weight was measured at day−1 and hour+1 by using a calibrated electronic scale (seca, Vogel and Halke, Hamburg, Germany). All subjects were weighed with emptied bladder in standardized clothing. Instrumentation was performed immediately after the subjects lay down, but data collection for the supine measurements was started only 10 min later after a steady state had been reached.

The Task Force Monitor[1] was used to monitor beat-to-beat heart rate (HR) by ECG, beat-to-beat stroke index (SI) by an improved method of impedance cardiography (12), and beat-to-beat blood pressure by the vascular unloading technique (8), which was corrected automatically to the oscillometric blood pressure measured on the

[1] Task Force Monitor, www.cnsystems.at.

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Table 1. *Clinical data at baseline, weight change, and performance time of subjects investigated*

Characteristic	Mean±SE	Range
Age, yr	36.7±1.53	25–51
Height, m	1.8±0.98	1.68–1.90
Weight, kg	73.4±1.36	57–85
BMI, kg/m^2	22.6±0.33	19.6–25.8
WNET, h	11.3±0.60	6–17.5
ΔWeight, kg	−1.95±0.09	−3.1 to −1.1
CT, min	659.3±13.75	506–807

BMI, body mass index; WNET, weekly net exercise training; ΔWeight, weight change between day before competition and 1 h after competition; CT, competition time.

contralateral arm. Total peripheral resistance index (TPRI) was calculated according to Ohm's law:

$$\text{TPRI} = \text{mean arterial blood pressure/cardiac index}$$

The Task Force Monitor allows intervention marks to be set to define periods for automated statistical analysis. Intervention marks were set at the beginning and the end of supine bed rest and 1 min after the beginning and at the end of active standing (see Fig. 1). Mean and standard deviation (SD) of the measured parameters were computed automatically for these defined steady-state periods. The Task Force Monitor automatically provides the 0.05- to 0.17-Hz [low-frequency (LF) band] and 0.17- to 0.40-Hz band [high-frequency (HF) band] of HR interval variability in normalized units (RRLFnu and RRHFnu, respectively, where RR is R-wave to R-wave) and the 0.05- to 0.17-Hz band of systolic (SBP) and diastolic blood pressure (DBP) variability in absolute values (SBPLF and DBPLF, respectively) and normalized units (SBPLFnu and DBPLFnu, respectively), using power spectral analysis, applying an autoregressive methodology (8, 36). Keeping in mind the limitations of spectral analysis in quantifying autonomic nervous system tone by the power spectral densities of these bands (18), they are referred to as autonomic (parasympathetic = RRHFnu and sympathetic = RRLFnu, SBPLF, DBPLF, SBPLFnu, DBPLFnu) modulation of the sinoatrial node and vasomotion (20, 24, 36). Additionally, baroreceptor reflex sensitivity (BRS) was automatically assessed by using the sequence technique (25).

The study was approved by the local ethics committee, and all subjects gave informed consent. All functions of the Task Force monitor (8) and of its predecessor (12) have been assessed previously, and the instrument has already been used successfully in a number of clinical studies (1, 13, 27, 34).

Statistical analysis. Data are presented as means ± SE. Because the variables showed no deviation from normal distribution, untransformed data were used in the statistical tests. Group differences were compared by the paired Student's *t*-test and by repeated-measures ANOVA, as appropriate. All reported *P* values are two-sided. The Bonferroni procedure was applied to adjust the *P* values for the fact that multiple comparisons were made. Adjusted *P* values of <0.05 were considered to indicate statistical significance. Multiple linear regression models with competition time as the dependent variable were used. In multiple regression, a stepwise backward elimination procedure was used. In the backward procedure, variables with *F* value < 3.9 were removed from the model. All analyses were performed with the use of the Systat 8.0 statistical software.

RESULTS

Figure 1*A* shows the original tracings of hemodynamic and autonomic recordings in a representative subject at day−1 (*left*) and hour+1 (*right*). Figure 1*B* shows the three-dimensional power spectra of HR and blood pressure variability in the same subject. Clinical data at baseline, the weight change between day−1 and hour+1, and performance time of the subjects are shown in Table 1. Table 2 and Fig. 2 show the results of all hemodynamic and autonomic parameters during supine rest at all time points. The time course of hemodynamic and autonomic parameters was assessed by repeated ANOVA (Table 2). As can be seen, HR and cardiac index were significantly increased and SI decreased at hour+1 compared with day−1 and returned to basal values at day+1. SBP and DBP were lower at hour+1 and day+1 and returned to basal values at day+3. TPRI was significantly reduced at hour+1 compared with day−1 and returned to basal values at day+1. BRS was significantly lower at hour+1 and day+1 compared with day−1 and returned to basal values at day+3. RRLFnu, SBPLFnu, and DBPLFnu were increased at hour+1 and day+1 and returned to basal values at day+3. DBPLF was increased at hour+1 and returned to basal values at day+1. RRHFnu was decreased at hour+1 and day+1 and returned to basal values at day+3. (For the respective significance levels, see Table 2.)

Figure 3 shows the percentage changes of hemodynamic and autonomic parameters from supine rest to active standing at the different time points of the study. As can be seen, there was a significant increase in SBP, TPRI, SBPLFnu, DBPLFnu, and RRLFnu, and decrease in RRHFnu after active standing at all time points, except 1 h after the competition. At that time point, the subjects were unable to raise their SBP, TPRI, and sympathetic modulation of vasomotor tone, and they showed a paradoxical increase in parasympathetic modulation of the sinus node after active orthostasis. Seven of the 23 subjects fainted in response to active orthostasis. There was no significant difference in weight change (Δ) between fainters and nonfainters from day−1 to hour+1 (Δbody weight: −1.9 ± 0.07 vs. −2.0 ± 0.13 kg, mean ± SE, not significant). All of the fainters showed significantly higher vagal activation in response to active orthostasis 1 h after the race compared with those subjects who did not faint ($\chi^2 = 5.406$, $P = 0.021$, 1 degree of freedom).

In multivariate linear regression models, age, weekly net exercise time (WNET), HR, RRLFnu, RRHFnu, SBPLFnu, DBPLFnu, SBPLF, and DBPLF were used as independent variables and competition time as the dependent variable. In a number of different models, it was always WNET and DBPLFnu on day−1 that contributed independently and significantly to the prediction of competition time. In contrast, age, HR, RRLFnu, RRHFnu, SBPLFnu, SBPLF, and DBPLF were all excluded from the regression models during the stepwise backward elimination procedure (Table 3).

DISCUSSION

The Ironman is one of those ultraendurance competitions requiring a high aerobic capacity, with a mean maximum O_2 uptake during treadmill running between 52 and 72 $ml \cdot kg^{-1} \cdot min^{-1}$ (23). To date, neither the short-term hemodynamic and autonomic responses to the competition nor the ensuing recovery has been studied in detail. As can be seen from Fig. 1*A*, artifact-free recording of the measured parameters is feasible. During supine rest and orthostasis up to HRs of up to 160 beats/min, no limitations of the recording or applied algorithms have been observed (13). The highest recorded HR in the present study, however, was only 124 beats/min.

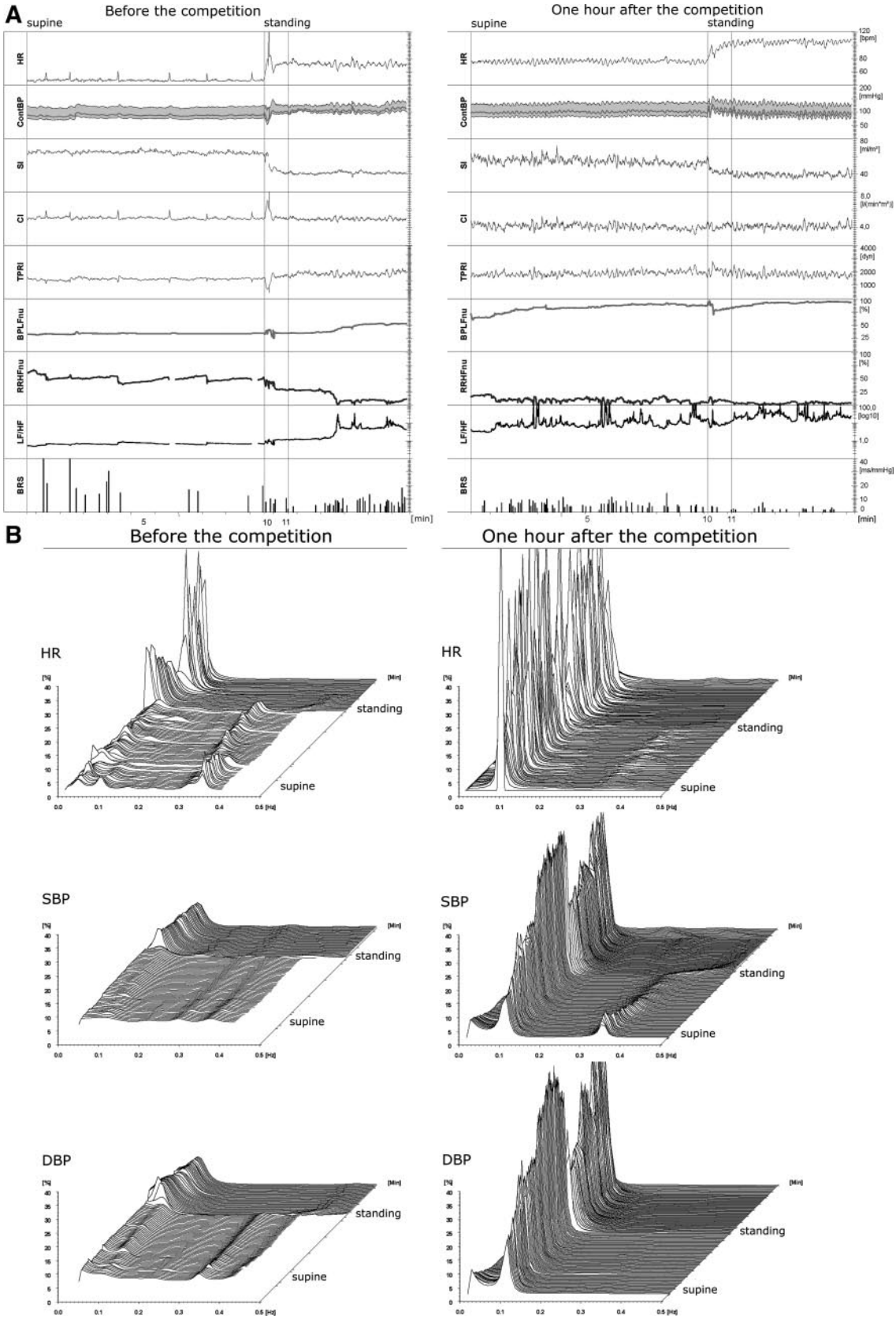

Fig. 1. *A*: original tracings of hemodynamic and autonomic recordings during supine rest (*left*) and after active standing (*right*). *B*: spectral analysis of heart rate (HR) and systolic (SBP) and diastolic blood pressure (DBP) variability during supine rest (*left*) and after active standing (*right*). ContBP: mean blood pressure, SBP, and DBP; SI, stroke index; CI, cardiac index; TPRI, total peripheral resistance index; BPLFnu, low-frequency (LF) blood pressure in normalized units; RRHFnu, R-wave to R-wave high frequency (HF) in normalized units; LF/HF, BPLFnu/RRHFnu; BRS, baroreceptor reflex sensitivity.

Table 2. *Hemodynamic and autonomic parameters during supine rest*

Characteristic	Day−1	Hour+1	Day+1	Day+3	Day+7	*P*
HR, beats/min	60.9±1.55	77.5±1.83*	63.7±1.65§	57.2±1.29	59.1±1.74	<0.001
SI, ml/m^2	50.8±2.12	8.0±2.47§	49.6±2.01§	51.2±2.08	53.7±1.85	0.011
CI, l · min^{-1} · m^{-2}	3.0±0.11	3.7±0.19*	3.1±0.13§	2.9±0.14	3.2±0.13	<0.001
SBP, mmHg	125.6±2.33	118.8±2.29†	119.7±1.84‡	121.9±1.94	125.2±2.62	0.018
DBP, mmHg	80.4±1.59	76.5±1.58§	73.9±1.41*	77.9±1.33	78.9±1.82	0.035
TPRI, dyn · s · m^{-2} · cm^{-5}	2,476±113.0	2,010±104.3*	2,283±112.6§	2,550±142.9	2,409±156.9	0.001
BRS, ms/mmHg	18.9±1.66	6.3±0.69*	16.1±1.37‡	23.4±1.45	19.1±1.51	<0.001
DBPLFnu, %	50.4±2.42	76.7±2.72*	67.9±2.49*	49.7±2.86	48.5±3.84	<0.001
SBPLFnu, %	43.9±2.21	68.7±3.51*	56.9±2.66*	44.6±2.58	42.8±2.88	<0.001
RRHFnu, %	43.6±2.53	15.3±2.35*	33.1±3.03*	46.6±3.49	45.6±3.88	<0.001
DBPLF, mmHg^2	4.2±0.66	15.2±4.06‡	6.7±1.61§	3.8±0.51	7.6±2.08	0.002
SBPLF, mmHg^2	7.5±2.16	16.2±3.12§	10.8±1.86§	10.5±1.62	8.3±1.59	0.065
RRLFnu, %	56.4±2.53	83.7±2.37*	66.9±3.03†	53.5±3.49	54.4±3.88	<0.001

Values are presented as means ± SE. HR, heart rate; SI, stroke index; CI, cardiac index; SBP, systolic blood pressure; DBP, diastolic blood pressure; TPRI, total peripheral resistance index; BRS, baroreceptor reflex sensitivity; DBPLFnu, DBP low frequency in normalized units; SBPLFnu, SBP low frequency in normalized units; RRHFnu, R-wave to R-wave high frequency in normalized units; DBPLF, DBP low frequency in absolute values; SBPLF, SBP low frequency in absolute values; RRLFnu, R-wave to R-wave low frequency in normalized units. The *P* values have been derived from a repeated-measures ANOVA model and denote a significant difference between all time points. *$P < 0.001$, †$P < 0.01$, and ‡$P < 0.05$ denote differences compared with day−1 (paired Student's *t*-test with Bonferroni correction); §not significant.

One hour after the competition, we observed the expected increase in HR (16), and a significant fall in SI, SBP, DBP, BRS, and TPRI, which slowly recovered to reach baseline levels already after only between 1 and 3 days after the competition (Fig. 2, *A* and *C*, and Table 2). The results shown in Fig. 2*B* and Table 2 indicate increased sympathetic modulation of vasomotor tone and sinus node and decreased parasympathetic modulation of sinus node. It is interesting to note that SBP, DBP, and TPRI decreased, despite the increase of sympathetic modulation of vasomotor tone. This, however, can be explained by metabolic vasodilation after strenuous exercise (4). One, therefore, can safely assume that blood pressure fall after exercise would even be greater than observed, would it not be partly compensated by the increased sympathetically mediated vasomotion (29).

BRS was markedly depressed after the race, as would be expected from the raised RRLFnu (26). Because autonomic parameters and BRS reached baseline levels at day+3, a remarkable recovery from an ultraendurance race in well-trained athletes occurs within merely 3 days.

The observed decrease in SI is probably related to the myocardial depression, observed after an Ironman (5), which may be due to an impaired myocardial shortening as a consequence of ischemia or metabolic abnormalities (6). The decrease in SI is probably not the consequence of the higher HR and hence reduced filling time, as adrenaline causes both a positive chronotropic and positive inotropic effect (19). Another example of the hemodynamic effects of endogenous adrenaline secretion is given in Fig. 4. This figure shows a beat-to-beat recording of hemodynamics in a normal subject during insulin-induced hypoglycemia. Using the same recording technique as in the present paper, we were able to show a simultaneous increase of HR and SI caused by the reactively released endogenous adrenaline. The fall in SI was also probably not a consequence of a reduced plasma volume after the competition. Although body weight was lower by 1.95 ± 0.09 kg at hour+1 compared with day−1, we can assume an increase of plasma volume of ~11–15% (9, 35). The mechanism of raised plasma volume, despite the decrease in body weight, is at least partly the result of a water shift from intracellular to the extracellular space due to oxidation of fat and glycogen stores and release of water stored with muscle and liver glycogen (9, 31, 33). The markedly raised adrenaline (32) and the reduced afterload (see Fig. 2*A*) after an Ironman should, therefore, also cause an increase in SI, which was not observed. Consequently, we can safely assume a depressed myocardial function after the competition.

All impedance methods used for SI determination are better suited for assessing relative changes in SI than absolute values (40). It is possible and even likely that the absolute values of SI, as reported in the present paper, underestimate the true SI in these very well-trained athletes.

As can be seen from Fig. 1*B*, the 0.05- to 0.17-Hz peak bandwidth of SBP, DBP, and HR variability becomes significantly smaller at hour+1 compared with the bandwidth at day−1. This is caused by an impressively rhythmic oscillation of HR and blood pressure after the competition (see *top* two traces, Fig. 1*A*, *right*). We are not aware that this phenomenon has been described before and can only speculate about its cause. Perhaps the very prominent sympathetic activation observed after a near-maximal effort synchronizes the different cerebral nuclei responsible for sympathetic activation at a very small frequency range.

The rise of TPRI usually seen after active standing is markedly impaired immediately after the race (Fig. 3). This might be the consequence of the inability to further stimulate an already maximally stimulated sympathetically mediated vasomotor response (Fig. 2*B* and Fig. 3). In addition, we found that the physiological decrease in parasympathetic tone after passive tilt is replaced by a paradoxical increase of vagal tone (Fig. 3) 1 h after the competition but not on the following days. We found that 7 of 23 subjects fainted in response to active standing. All of the fainters showed significantly higher vagal activation in response to active orthostasis 1 h after the race compared with those subjects who did not faint. Plasma volume is known to be very important for maintaining orthostatic tolerance (15). Given the known increase of plasma volume after an Ironman competition (9, 35), we have no reason to assume that a decrease in plasma volume was responsible for our observation. There was also no difference in weight

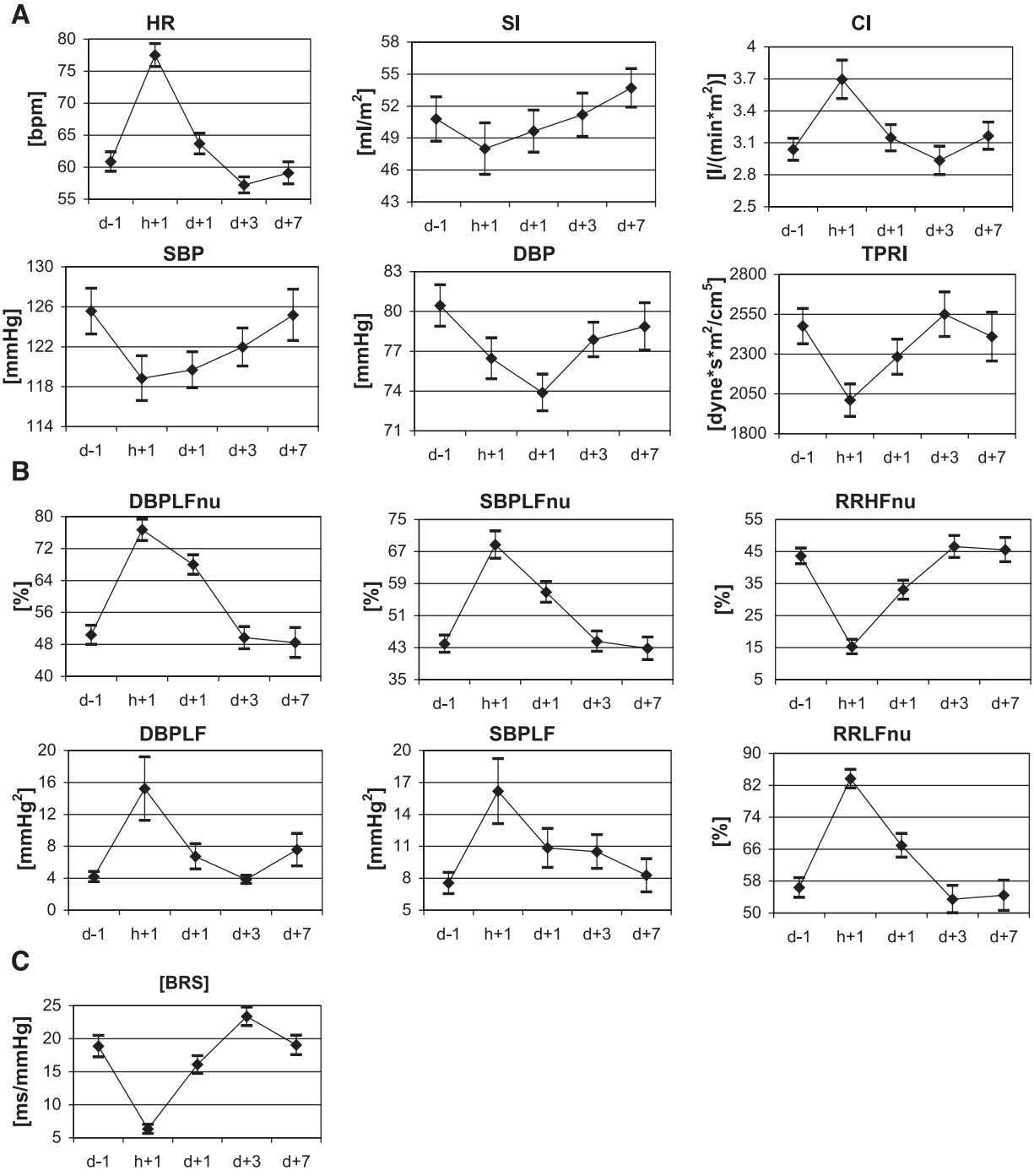

Fig. 2. Hemodynamic parameters (*A*), autonomic parameters (*B*), and BRS (*C*) during supine rest at all time points of the study. Values are means ± SE. For significance levels, refer to Table 2. DBPLFnu, DBP LF in normalized units; SBPLFnu, SBP LF in normalized units; DBPLF, DBP LF in absolute values; SBPLF, SBP LF in absolute values; RRLFnu, R-wave to R-wave LF in normalized units; d-1, day before competition; h+1, 1 h after competition; d+1, d+3, and d+7: 1, 3, and 7 days after competition.

changes after the competition between fainters and nonfainters. Fainting after ultraendurance performance is characterized by markedly raised sympathetic tone at supine rest (Fig. 2*B*) and paradoxical vagal activation during orthostasis (Fig. 3). Thus it mimics the low orthostatic tolerance observed in postural tachycardia syndrome, which is also associated with an increased sympathetic drive to the heart (3). In vasovagal syncope occurring independent of physical exertion, a raised sympathetic tone has also been described (21). Unfortunately, the equipment used does not provide cross-spectral analysis of R-R interval and of blood pressure, which could have helped to identify the subjects prone to syncope already in the supine state (14).

In the past, no significant relationship between overall competition finish time and maximum O_2 uptake in a small group of Ironman triathletes was found (22). We attempted to predict

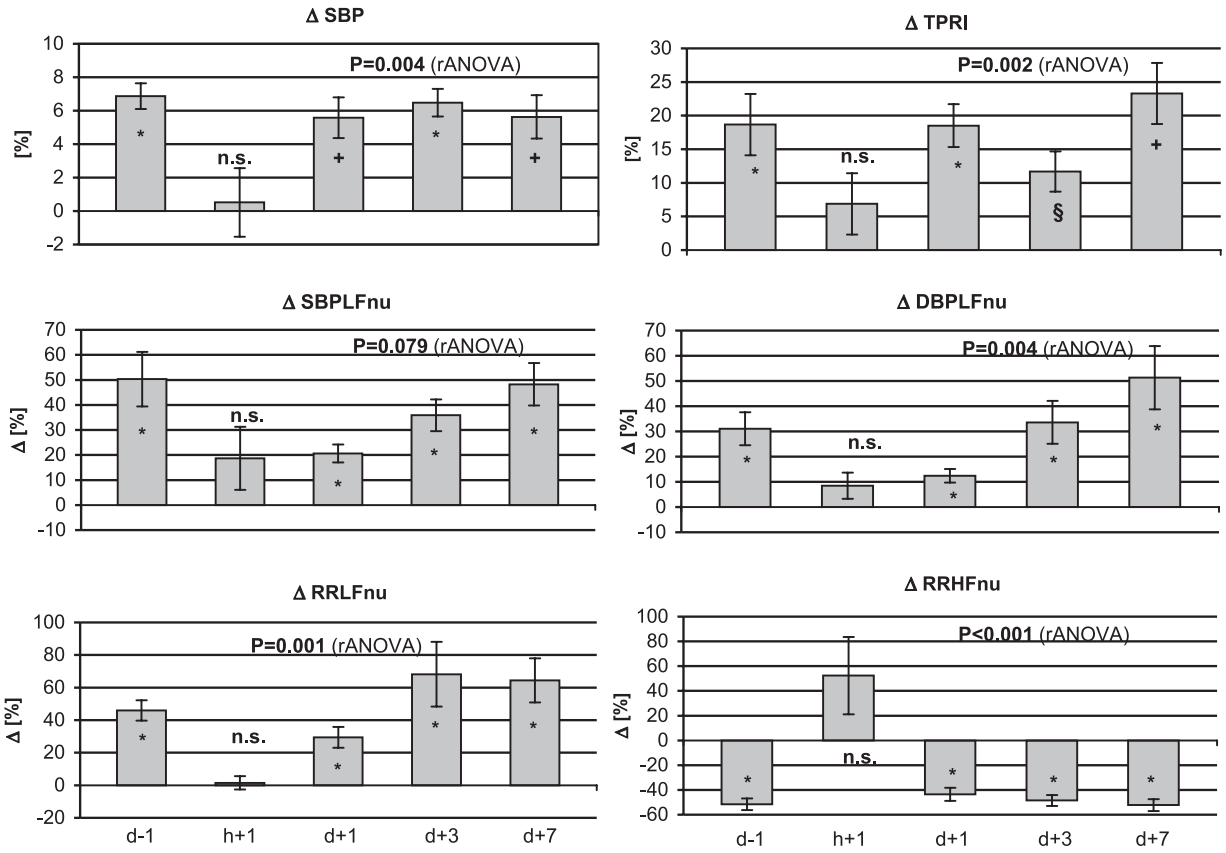

Fig. 3. Percent changes (Δ) of hemodynamic and autonomic parameters from supine rest to active standing. Values are means ± SE. There is a significant difference among the time points, as described by repeated-measures ANOVA (rANOVA). *$P < 0.001$, $^{+}P = 0.002$, §$P = 0.046$, n.s., not significant. Differences are shown of the values measured during active standing and in supine position (paired Student's *t*-test with Bonferroni correction).

competition time not only from WNET but also from baseline hemodynamic and autonomic parameters. In the multivariate regression analysis, which included age, WNET, HR, and baseline autonomic parameters in a number of different models, only WNET and DBPLFnu contributed significantly to the prediction of competition time. A high WNET and a low DBPLFnu before the competition were related to a short competition time (Table 3). Despite the small sample size (which is accounted for by the statistical tests used), this multivariate analysis gave a highly significant result. In contrast, RRLFnu, which also has been used to quantify sympathetic tone (2), was not related to competition time in the multivariate regression analysis (Table 3). The reason for this is probably that the LF band of the HR interval reflects not only sympathetic tone, but also to some extent parasympathetic tone (11, 39). Thus it appears that the analysis of beat-to-beat blood pressure in the frequency domain could become a valuable additional tool for optimizing the training of athletes.

Table 3. *Multivariate linear regression analysis with competition time as the dependent variable*

Model	Variable	Coefficient	*F*	*P*
1	Intercept	709		
	WNET	−13.6	15.494	0.001
	DBPLFnu day−1	2.06	5.701	0.026
	Age	0.25	1.424	0.246
	HR day−1	0.17	0.664	0.431
	RRHFnu day−1	0.18	0.662	0.425
2	Intercept	814		
	WNET	−13.7	13.009	0.001
	Age	0.33	2.631	0.119
	HR day−1	0.28	1.908	0.181
	RRHFnu day−1	0.02	0.005	0.943
	SBPLFnu day−1	0.31	2.357	0.139
3	Intercept	814		
	WNET	−13.7	13.009	0.001
	Age	0.33	2.631	0.119
	HR day−1	0.28	1.908	0.181
	RRHFnu day−1	0.02	0.005	0.943
	RRLFnu day−1	−0.02	0.005	0.943

The squared multiple is $r = 0.493$ for *model 1*, $r = 0.361$ for *model 2*, and $r = 0.361$ for *model 3*.

In conclusion, an ultraendurance performance like an Ironman leads to acute changes in baseline hemodynamics and autonomic regulation characterized by a decrease in SI, blood pressure, TPRI, BRS, and vagal modulation of the sinus node, and an increase in HR, cardiac index, and of sympathetically mediated vasomotor tone. Furthermore, there is a paradoxical response of the autonomic nervous system in response to active standing characterized by paradoxic activation of HF band of HR variability and a blunted increase in sympathetically mediated vasomotor tone. This makes these athletes very vulnerable to orthostatic challenges after the competition. The vasovagal syncope induced by sympathetic activation from other causes, such as observed in postural tachycardia syndrome (3, 10), may represent a similar example of the same basic mech-

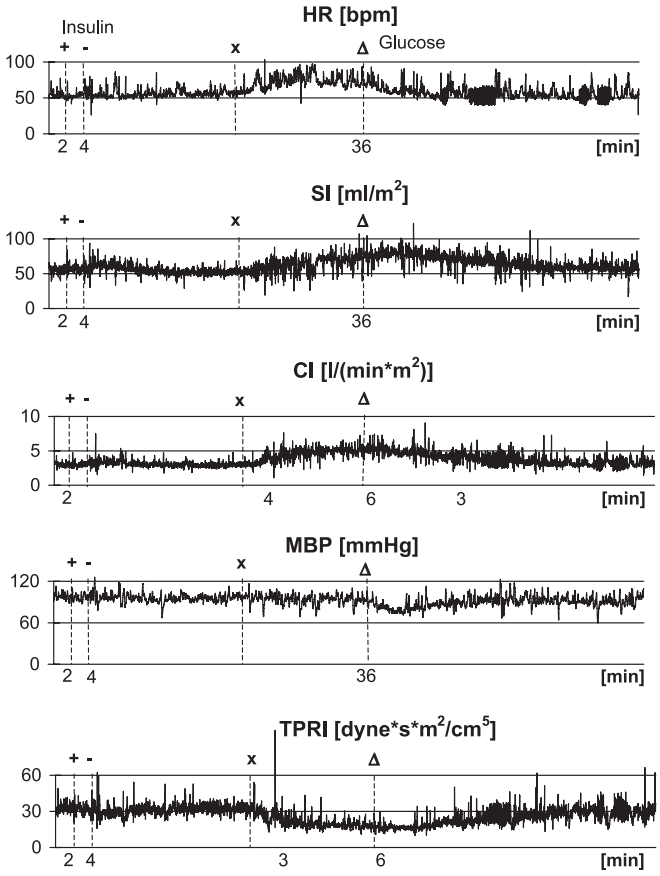

Fig. 4. Hemodynamic changes during insulin-induced hypoglycemia. +, Beginning of insulin injection; -, end of insulin injection; x, beginning of insulin-induced hypoglycemia; Δ, beginning of glucose injection; MBP, mean blood pressure.

anisms. Surprisingly, the high WNET necessary to participate in such a competition enables the athletes to completely and rapidly recover within 3 days after the event. A low sympathetically mediated stimulation of vasomotor tone, despite a high WNET, is significantly related to a fast competition time in the multivariate regression analysis. This signals that there could be a narrow range of an appropriate level of training for any given training state characterized by a low sympathetic drive. This extends the observation that overtraining usually leads to an increased sympathetic drive in the majority of athletes (28) well into the physiological range. Our investigation supports the concept of autonomic control of training and overtraining. Maintaining a low sympathetic tone, despite an increasing training load, may be the ideal way to optimize competition performance. Monitoring of beat-to-beat blood pressure might become an additional tool for optimizing the training of athletes.

The rapid recovery after what we consider now to be an extreme endurance exercise may throw some light on the true capabilities of the human body acquired during human evolution. For the largest part, namely for the 5 million years of the hunter-gatherer age, mankind had to perform daily a regimen of 10 up to 30 km of walking and running for survival (38). This would correspond to up to 4-h daily exercise time and a WNET of 28 h. Our "well-trained" athletes achieved only between 20 and 60% of this duration of training, which demonstrates the change of perspective that occurred with the beginning of the industrial age.

ACKNOWLEDGMENTS

We thank J. Batzel for valuable help with the manuscript and P. Kotanko for advice with the statistical analysis.

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