

Heart rate variability assessment of the effect of physical training on autonomic cardiac control

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Running Title

Physical training and HRV

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Abstract

Background:

The effect of exercise interventions on autonomic nervous system (ANS) control of the heart by heart rate variability (HRV) is often investigated in just one position. It was hypothesized that results of exercise-induced changes on ANS are dependent on body position and that it is possible to distinguish between exercise induced changes in vagal and sympathetic influence by taking measurements in different body positions.

Methods:

183 (male=100, female=83) healthy volunteers, between 18 and 22 years, participated in a prospective twelve week medium to high intensity exercise intervention study with a self-control design. The influence of the exercise intervention was investigated on supine, rising and standing as well as on the orthostatic response. Time domain, frequency domain and non-linear (Poincaré) HRV analysis were performed.

Results:

The exercise intervention lead to a significant increase ($P<0.05$) in vagal influence during supine, rising and standing. Sympathetic control in the supine position was decreased and increased during rising and standing. In the initial orthostatic response to rising from the supine position, the exercise intervention lead to increased ($P<0.05$) vagal withdrawal as well as increased sympathetic control. The orthostatic response measured as the difference between standing and supine indicated only an exercise induced increase in sympathetic control.

Conclusions:

Exercise-induced changes in sympathetic and parasympathetic ANS control differ, depending on posture and period of measurement. Exercise induced changes in parasympathetic and sympathetic outflow, respectively, can be extracted from measurements from supine, through the orthostatic response, to standing, thereby detecting changes in ANS that are otherwise obscured.

Key Words: exercise, autonomic nervous system, heart rate variability, posture.

Regular physical activity has many health benefits such as the prevention of, or decreasing in, the incidence of coronary heart disease, positive changes in cardiovascular functioning and beneficial metabolic, psychological and neurovegetative effects.¹⁻³ Exercise based clinical interventions are widely recommended to reduce morbidity and all-cause mortality.^{4,5} The protective influence of exercise on the heart to counter damaging cardiac events is believed to be the result of adjusted influences by the autonomic nervous system (ANS) on, for example, heart rate (HR) and heart rate variability (HRV). However, certain questions remain as to the effects of exercise on ANS control of the heart.⁶⁻⁸

It is generally known that the initial aerobic training-induced effects on indices of HRV were heterogenic and controversial.⁹⁻¹¹ According to Hautala et al.¹ reasons for this heterogeneity in reports may lay in age, small sample size, the duration and also the intensity of the intervention. However, there is a consistent body of evidence that exercise increases resting vagal cardiac control, in healthy as well as patient groups.^{1,12-15} Although it is theorized that posture change and an orthostatic challenge may highlight ANS changes better than the resting supine position,^{16,17} and that reduced ANS responsiveness to an excitatory stimulus is seen as the most common feature of patho-physiological states,¹⁸ exercise induced changes in HRV during standing and in response to an orthostatic stressor is less known.

This study investigated the influence of a standardised, intensive physical training programme, in a controlled environment, on ANS cardiac control by means of HRV quantification. The exercise induced changes in overall HRV were measured in the supine, rising and standing positions as well as the adjustments in orthostatic response. Analytical techniques used were time domain, frequency domain and non-linear (Poincaré) analysis.

It was hypothesized that results of exercise induced changes on ANS are dependent on the body position and should be assessed not only in the resting position but also during standing and during an orthostatic stressor. It was also hypothesized that it is possible to better distinguish between exercise induced changes in vagal and sympathetic influence by taking measurements in different body positions.

METHODS

Study type and study population

This was a prospective twelve week exercise intervention study with a self-control design. The study protocol was submitted and approved by the University Ethics Committee. All participants gave written informed consent before commencement of the intervention. The volunteers were between 18 and 22 years of age, consisted of 100 males and 83 females, and were of predominantly African ethnicity (African = 171; Mixed = 5; Caucasian = 5; Indian = 2). Mass and body mass index remained relatively constant over the study period as can be seen in Table 1. None of the participants were professional athletes or high level sport participants. Exclusion criteria included refusal to freely give written informed consent; history of cardiovascular, hepatic, respiratory or renal impairment, as well as pulmonary, metabolic, and orthopaedic disease requiring medical attention; lung/ respiratory tract infection in the previous two weeks; medication that could influence cardiovascular control and psychological disorders.

All participants followed a strictly enforced, Basic Training Programme. They were subjected to the same standardised 24 hour routine (exercise, diet and sleep) for the duration of the twelve week exercise intervention. The aim of the physical training program was to develop basic fitness components such as cardio respiratory endurance and muscular endurance. The calculated average basal metabolic rate (BMR) for participants, taking weight, age account, was 6371 kJ/day. This, in addition to the energy expenditure of the training and exercise activities, resulted in a

calculated average energy consumption of 8485 kJ/day, which can be classified as a medium to high intensity exercise program.¹⁹

Data sampling and HRV quantification

Participants were instructed not to exercise or drink any alcohol or caffeine the 24 hours before measurements. They were allowed to eat a low protein breakfast (cereal with milk) on the morning of testing. POLAR RS800 heart rate monitors were used to obtain RR interval data sets (tachograms) from participants at the start (pre-intervention) and at the end (post-intervention) of the twelve week exercise period. After a 2 min stabilisation period in the supine position, ten minute tachograms were obtained for supine and 10 minute tachograms during standing upright, leaning with their backs against a wall, feet 30cm apart and 30cm from the wall.

Data sets were exported and artefacts in RR interval data were removed with standard Polar software programmes with a low filter power and a minimum beat protection zone of six beats per minute. The RR interval sets were analysed using HRV Analysis Software 1.1 for windows developed by the Biomedical Signal Analysis Group, Department of Applied Physics, University of Kuopio, Finland. Smoothness priors for trend and Model Eye programme settings were used for detrending with an Alpha value of 500. The autoregressive model order value was 16 and the interpolation rate was 4 Hz. Standard time domain, frequency domain and non-linear (Poincaré analysis) techniques were implemented.^{20,21}

The Poincaré analysis method was included due to its applicability to non-stationary data sets.²² With this method SD1 and SD2, were determined. SD1 is an indicator of the standard deviation of the immediate, or short term, RR variability due to parasympathetic efferent (vagal) influence on the sino-atrial node. SD2 is an indicator of the standard deviation of the long-term or slow variability of the heart rate representing global variation.²¹ Recommended time domain HRV indicators such as SDNN, RMSSD and pNN50 were determined and reported with RR interval and heart rate. Spectral components analysed with frequency domain analysis included high frequency (HF), 0.15 – 0.40 Hz, low frequency (LF), 0.04 – 0.15 Hz, and the LF/HF ratio. The indicators LF/HF, LFnu and HFnu were used as indicators of autonomic balance or relative power distribution between the sympathetic and parasympathetic branches of the ANS.²⁰ LFnu (normalised units) represent the relative power of the LF component in proportion to the total power minus the VLF component, i.e., LF / (total power-VLF). The HFnu (normalised units) represent the relative power of the HF component in proportion to the total power minus the VLF component, i.e., HF / (total power -VLF), while LF/HF is used to assess the fractional distribution of power.²⁰

A minimum tachogram length of 1 minute is essential to assess the high frequency (HF) components, and at least 2 minutes for the low frequency LF components during HRV analysis.²⁰ In the current study the non-stationary period during rising were analysed separately. One tachogram in supine position (directly before rising), one tachogram during rising (0 to 180s), one tachogram during standing (180 sec to 360s standing) and one tachogram during continued standing (360s to 540s standing) were used for HRV quantification.

The orthostatic response was quantified by the percentage difference (% Δ) between the HRV indicator values obtained during the first stabilised standing period (180-360s) and that obtained during the supine position (% Δ HRV indicator value = [standing – supine]/ supine x100). In addition, the percentage change was also calculated between the non-stationary rising-to-standing period HRV values (0-180s) and supine, as well as between the second stationary standing period (360-540s) and supine.

Statistical analysis

The T-test is based on the assumption of normality, hence it is necessary to confirm that this assumption is met. In this study the chi-square goodness-of-fit test was used due to the relatively large sample size (rather than a test such as Kolmogorov-Smirnov which is usually used for smaller samples). The Chi-Square test was applied to all data sets (HR, RR, SDNN, RMSSD, pNN50, SD1, SD2, LF Power (ms²), HF Power (ms²), HF Power (nu.), LF Power (nu.) and LF/HF to determine which indicator values were non-normally distributed. From these, RMSSD, pNN50, SD2, LF Power (ms²), HF Power (ms²) showed P-values < 0.05 providing statistical evidence of significant differences from the normal distribution. This violates the assumption of normality of the T-Test. In such cases, two options are available; transformation of the data (using ln or square root) to obtain a more symmetrical distribution; or the use of non-parametric tests. As interpretation of transformed variables may be complicated, it was decided to use the Wilcoxon signed rank test (95% confidence level) to assess exercise intervention induced changes in the non-normal distributed data sets and the Matched T-Test for the rest. These tests were also used to determine if there was a difference in the % change (%Δ) in HRV indicator values in response to an orthostatic stressor as measured before and after the training period.

Results

The anthropometric characteristics of the group are shown in Table 1. As can be seen the mass, and therefore the BMI, remained relatively constant over the period.

In Table 2 the HRV indicator values and standard deviations for the supine, rising and standing periods are depicted including the level of significance in differences found between pre-and post-intervention values. All HRV indicators, except the standing ANS balance indicators, showed significant exercise induced changes. All vagal and mixed origin HRV indicators (sympathetic and vagal) showed significant increased variability, while the supine ANS balance indicators, LF/HF and LFnu, showed significant decreases.

The percentage exercise induced changes (pre-intervention vs. post-intervention) in the supine position are shown in Figure 1. All indicators were significantly different (P<0.05) after the exercise intervention. It illustrates how variability in vagal and mixed origin indicators increased, while the ANS balance indicators LF/HF and LFnu decreased.

Table 1. Anthropometric characteristics of the study group: Mean and standard deviation

Characteristic	Males Pre-Intervention	Males Post-intervention	Females Pre-Intervention	Females Post-intervention
Height (cm)	171.36 (SD=5.86)	171.36 (SD=5.86)	159.26 (SD=5.49)	159.26 (SD=5.49)
Mass (kg)	61.78 (SD=6.89)	63.18 (SD=6.61)	60.22 (SD=8.99)	60.04 (SD=7.48)
Body Mass Index (kg.m ²)	21.43 (SD=2.16)	22.42 (SD=2.47)	23.40 (SD=3.04)	22.52 (SD=2.34)

SD= Standard Deviation

Table 2. Comparison of average HRV indicator values as determined before and after the exercise intervention for the Supine, Rising and Standing periods. The significance of difference (Pre Δ vs. Post Δ) was determined by the Matched t-test and Wilcoxon signed-rank test depending on distribution of data.

Indicator	Pre (SD)	Post (SD)	P-value	Pre (SD)	Post (SD)	P-value
	Supine			Rising (0-180s)		
HR(bpm)	72.58 (10.94)	61.38 (9.96)	<0.0001	89.28 (12.49)	80.12 (12.14)	<0.0001
RR(ms)	0.85 (0.13)	1.01 (0.16)	<0.0001	0.70 (0.11)	0.78 (0.13)	<0.0001
SDNN(ms)	0.05 (0.02)	0.07 (0.03)	<0.0001	0.05 (0.02)	0.06 (0.02)	<0.0001
RMSSD(ms)	57.35 (33.36)	83.95 (44.72)	<0.0001	33.2 (20.73)	47.1 (26.26)	<0.0001
pNN50(%)	34.55 (21.83)	58.45 (22.03)	<0.0001	9.4 (13.96)	14.9 (17.10)	0.0003
SD1(ms)	44.72 (23.74)	64.61 (31.58)	<0.0001	24 (14.79)	34.2 (18.70)	<0.0001
SD2(ms)	72.8 (36.95)	86.1 (47.15)	0.0020	108 (49.0)	130.6 (54.31)	<0.0001
LF(ms ²)	243 (396.9)	329.5 (873.2)	0.017	356 (373.8)	472.5 (501.4)	0.0001
HF(ms ²)	288.5 (391.3)	525.5 (729.8)	<0.0001	89 (172.9)	161 (225.3)	<0.0001
LF/HF	0.96 (3.13)	0.64 (10.13)	0.044	3.82 (17.50)	3.46 (12.34)	0.93
LFnu	46.45 (19.99)	38.2 (19.16)	0.0022	76.2 (19.03)	73.55 (20.86)	0.47
HFnu	50.1 (19.28)	58.95 (20.38)	0.0071	19.6 (18.60)	22.15 (17.23)	0.96
	Standing (180-360s)			Standing (360-540s)		
HR(bpm)	91.83 (11.69)	81.95 (12.40)	<0.0001	93.10 (12.31)	82.46 (13.62)	<0.0001
RR(ms)	0.67 (0.10)	0.75 (0.12)	<0.0001	0.66 (0.10)	0.75 (0.12)	<0.0001
SDNN(ms)	0.03 (0.01)	0.041 (0.02)	<0.0001	0.03 (0.02)	0.05 (0.02)	<0.0001
RMSSD(ms)	22.2 (15.49)	32 (29.23)	<0.0001	19.65 (16.53)	31.15 (23.44)	<0.0001
pNN50(%)	2.6 (12.57)	8.95 (17.16)	<0.0001	1.95 (12.27)	9.85 (17.30)	<0.0001
SD1(ms)	18.46 (11.03)	27.28 (18.45)	<0.0001	17.43 (11.43)	26.95 (16.71)	<0.0001
SD2(ms)	52.9 (25.55)	76.55 (34.98)	<0.0001	49.85 (25.61)	75.65 (34.48)	<0.0001
LF(ms ²)	155 (254.8)	285.5 (401.1)	<0.0001	143.5 (227.0)	344.5 (510.3)	<0.0001
HF(ms ²)	35 (105.9)	77.5 (210.6)	<0.0001	32.5 (91.5)	70.0 (187.0)	0.0002
LF/HF	4.91 (24.07)	4.86 (29.70)	0.94	4.47 (20.67)	4.44 (29.49)	0.92
LFnu	80.3 (19.50)	79.75 (18.73)	0.67	80.50 (18.38)	80.00 (17.05)	0.34
HFnu	16.4 (18.15)	16.7 (17.06)	0.52	17.75 (16.66)	17.55 (16.73)	0.48

HR=heart rate; bpm=beats per minute; RR= RR interval; HF=high-frequency components; LF=low-frequency components; pNN50= percentage of intervals differing by >50 ms from preceding interval; RMSSD=root mean square of successive differences in RR intervals; SDNN=standard deviation of RR interval; SD1=standard deviation of short term variability; SD2=standard deviation of the long-term variability s: seconds; SD=Standard Deviation

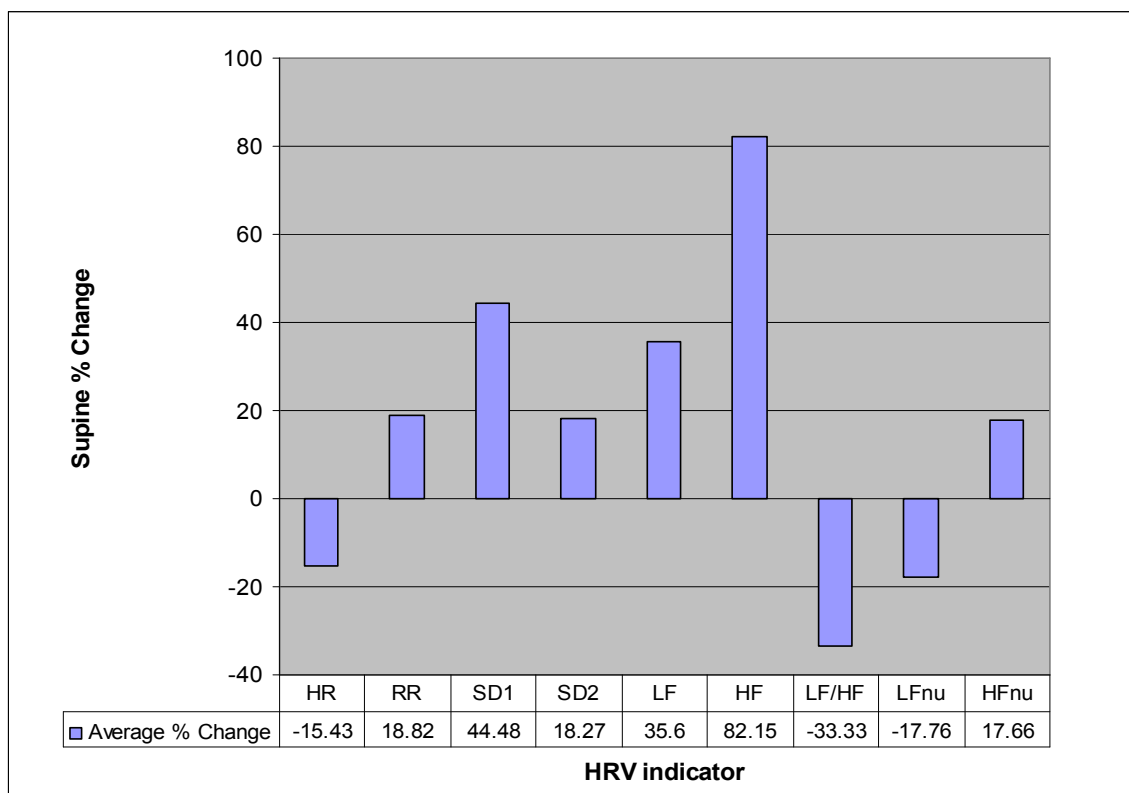


Figure 1. Percentage exercise induced changes on supine HRV indicators: [(Post exercise – pre-exercise)/pre-exercise] x 100

Table 3 shows the exercise induced changes (Δ) in orthostatic response when the orthostatic response was calculated as a) the difference between indicator values obtained during rising (0-180s) and supine, b) the difference between values of the first period of stabilisation in the standing position (180-360s) and supine and c) the difference between values obtain during the second period of standing (360-540s) and supine.

Indicators of vagal influence (RMSSD, pNN50, SD1, HFms²), showed a significant exercise induced decrease when the orthostatic response was calculated from the values during rising, i.e., [(0-180s) – supine]/supine x100. However, when the response was calculated from either the first standing period i.e., [(180-360s) – supine]/supine x100 or, the second standing period, i.e., [(360-540s) – supine]/supine x100, no significant exercise induced changes was visible. In contrast, indicators of mixed origin (SD2, LFms²) did not show significant exercise induced effects when the 0-180s period was used in the calculation, but showed significant increases when the 180-360s and 360-540s periods were used.

Discussion

Initially there were conflicting reports on the effects of exercise on the autonomic nervous system, but it is now generally accepted, at least for the supine position, that exercise can increase the vagal influence on the heart and thus the RR interval. However, from the positive, but relatively weak, association between the increase in RR interval and the increase in vagal activity,¹² it is clear that the increase in the vagal regulatory input to the heart cannot be seen as the only contributor to the exercise-induced lowering of heart rate. In contrast to the now accepted fact that physical training can lead to an increase in the parasympathetic control of the heart, the effect on the sympathetic nervous system has not unequivocally been proved by HRV analysis.

Table 3. The exercise induced changes (Δ) in orthostatic response determined during a) rising: (0-180s) rising HRV-supine HRV), b) (180-360s standing HRV-supine HRV) and c) (360-540s standing-supine). The significance of difference (Pre Δ vs. Post Δ) was determined by the Matched t-test and Wilcoxon signed-rank test depending on distribution of data.

Indicator	Δ orthostatic response: % Change during rising = (0-180s rising-supine)			Δ orthostatic response: % Change during standing = (180-360s standing-supine)			Δ orthostatic response: % Change during continued standing = (360- 540s standing-supine)		
	Pre	Post	P-value	Pre	Post	P-value	Pre	Post	P-value
Δ HR(bpm)	21.77	33.12	0.0001	26.32	36.75	0.0000	27.94	37.66	0.0001
Δ RR(ms)	-16.18	-22.86	0.0001	-20.07	-25.49	0.0000	-21.09	-25.72	0.0001
Δ SDNN(ms)	2.53	-8.06	0.035	-26.46	-26.80	0.8582	-29.36	-25.93	0.5780
Δ RMSSD(ms)	-36.32	-46.79	0.0004	-57.05	-60.04	0.2334	-60.72	-60.79	0.5749
Δ pNN50(%)	-58.73	-70.41	0.0068	-88.18	-85.00	0.5311	-92.36	-80.82	0.0595
Δ SD1(ms)	-26.92	-39.82	0.0001	-49.86	-53.73	0.3194	-53.37	-54.00	0.4751
Δ SD2(ms)	55.15	46.53	0.8758	-28.02	10.76	0.0297	-30.77	-13.27	0.0234
Δ LF(ms ²)	67.12	54.52	0.8513	-23.66	-3.26	0.1178	-36.69	0.00	0.0395
Δ HF(ms ²)	-63.10	-72.79	0.0398	-84.08	-87.41	0.2686	-85.95	-85.98	0.4053
Δ LF/HF	269.49	480.00	0.0032	340	567.56	0.1304	331.73	535.09	0.0591
Δ LFnu	48.93	85.96	0.0232	157	131.57	0.0003	46.55	101.27	0.0004
Δ HFnu	-53.15	-63.88	0.0091	-53.4	-51.53	0.1085	-62.28	-70.24	0.1144

HR=heart rate; bpm=beats per minute; RR= RR interval; HF=high-frequency components; LF=low-frequency components; pNN50=percentage of intervals differing by >50 ms from preceding interval; RMSSD=root mean square of successive differences in RR intervals; SDNN=standard deviation of RR interval; SD1=standard deviation of short term variability; SD2=standard deviation of the long-term variability s: seconds; SD=Standard Deviation

As autonomic regulation of the heart is of paramount importance, not only in the supine position, but perhaps even more so during standing and in response to standing up from the supine position, it speaks for itself that the influence of exercise programs would perhaps be better assessed by measuring it in more than one position and in response to an orthostatic challenge. Although it is assumed that RR intervals sampled in the supine position is more reliable than during tilt or standing,¹² Dietricha et al.²² reported satisfactory reproducibility of these short-term, non-invasive measurements in the supine, as well as in the standing position.

The present study investigated the effect of a 12 week standardised exercise intervention in a controlled environment on a healthy young-adult, predominantly African, population. It investigated the influence of the intervention on the supine, the rising, standing HRV, as well as the orthostatic response. Recordings were analysed by time domain, frequency domain and Poincarè analyses. It was hypothesized that the influence of exercise on the vagal and sympathetic cardiac control, respectively, can be better assessed and understood by measurements in different positions.

The influence of an exercise intervention on heart rate and RR interval in the supine and standing positions as well as during an orthostatic stressor

In the present study the exercise intervention led to a decreased HR and an increased RR interval in the supine, rising and standing positions (Table 2). HR was decreased by on average 15% in the supine position and 11% in the standing position, while the length of the RR intervals increased by 18% and 12%, respectively. HR was significantly lower ($p < 0.0001$) with RR and SDNN (standard deviation of RR interval) significantly higher ($p < 0.0001$) during all four post-intervention tachogram periods (Table 2). The decrease in supine HR and increase in RR intervals are in line with previous publications, as reported in a 2005 meta-analysis of the effect of exercise on HRV in healthy participants¹² as well as that of a more recent review¹⁴ on improvements in HRV with exercise therapy. Several authors referred to the lowering of heart rate by exercise intervention as exercise-induced bradycardia.^{10,12,23,24} Textbook bradycardia is characterized by a heart rate below 60 beats per minute while normal resting rate is considered to be between 60 to 100 beats per minute.²⁵ Thus, although significant decreases in HR occurred in the present study, the twelve week, medium to high intensity intervention, did not result in bradycardia as the average supine heart rate of the participants were still above 60 beats per minute.

In addition to the lowering effect of the exercise intervention on the supine and on the standing heart rate, an effect was also seen on the heart rate during the orthostatic response. During rising from the supine position to the standing position the healthy heart will show an increase in rate. In the present study heart rate increased by 21.77% upon rising before the intervention, and by 33.12% post-intervention.

Before the exercise intervention a 16.18% decrease was found in the length of the RR-intervals upon rising (0-180s), with a post-intervention reduction of 22.86% (Table 3). Thus a 7% lower increase in the length of the RR-interval upon rising after the 12 week exercise intervention than before the intervention. This is in agreement with Gilder et al.⁶ who, in a cross-sectional study, showed a 6% higher decrease in RR-interval length in a low volume exercise group than in a high volume exercise group. It is said that these exercise induced changes measured in HR and RR interval during rising and standing, indicates increased responsiveness in the vagal reaction and sympathetic vasoconstrictor outflow upon stimulation of the baroreceptors.²⁷

SDNN is generally seen as an indicator of global variability. It is of interest, that both in this study and that of Gilder et al.²⁶ SDNN over the period of rising, was 11% higher after the exercise intervention than before. In view of the relationship between HRV and health, this exercise induced increase in HRV, during the period generally marked by vagal withdrawal, once again illustrates the beneficial effect of exercise interventions on health.

Exercise induced changes in the parasympathetic autonomic (vagal) cardiac control in the supine and standing position analysed by time domain, frequency domain and Poincarè analyses

Results of this study (Table 2) indicated that the average of all post-intervention indicators of pure parasympathetic (vagal) induced heart rate variation, as measured by RMSSD, pNN50, HFms² and SD1, were significantly higher ($p < 0.0001$ to $p = 0.0030$) than pre-intervention. This exercise-induced effect, as in the case for heart rate, was found for all 4 periods measured, i.e., in the supine position, during rising, as well as two standing periods.

A number of past studies reported conflicting results on the effect of exercise on the resting heart rate variability.^{28,29} Factors such as differences in study populations, exercise regimes and different analytical techniques (time domain, frequency domain

and non-linear analysis), could have contributed to the differences.^{1,30} Nevertheless, at present the majority of cross sectional,^{1,31,32,33,34,35} as well as longitudinal studies,^{1,10,36,37} are in agreement that exercise can increase the vagal cardiac control. Unfortunately, the influence of exercise induced changes measured with short term HRV, are with some exceptions,³⁸ mostly reported only for the supine position.^{12,14}

Our results are thus in agreement with the current view on the effect of exercise on supine vagal control. In addition, it showed that exercise will also increase the average vagal influence during rising and standing. The results of the three HRV techniques were, although not in the magnitude of change, similar in the direction of change.

The influence of an exercise intervention on the sympathetic autonomic HR control in the supine, rising and standing position analysed by time domain, frequency domain and Poincaré analyses

Although it is often assumed that exercise can lower the sympathetic outflow to the heart, the HRV assessment of the sympathetic nervous system's response to exercise remains problematic. This is due to the fact that both sympathetic and parasympathetic influences are present in the LF heart rate oscillations.³⁹

The effect of exercise on sympathetic activity has also been assessed by measurement of muscle sympathetic nervous system activity (MSNA). However, these results also vary from increased, to decreased, to unchanged sympathetic activity.^{40,41}

Results from the current study (Table 2) showed, not only significant increased variation in parasympathetic HRV indicators, but also in indicators of mixed origin (sympathetic activity + vagal activity), such as: SDNN, SD2 and LF(ms²), over all four time periods. However, as shown in Figure 1, the exercise-induced increases in the average values of the mixed indicators (SD2:18.27%; LFms²:35%) were, for the supine position, consistently lower than the exercise induced vagal increases (SD1:44%; HFms²:82%). This did not apply to the rising and standing positions. The observation that the pure vagal influence increased more than the increase in the combination of the two branches is significant as it points towards an exercise-induced decrease in the supine sympathetic influence. It is, however, not possible to state this empirically without examining the effects on the autonomic balance.

Results from the supine recordings on autonomic balance (Table 2) showed that the exercise intervention induced a significant shift towards increased parasympathetic influence, as seen in the pre- to post-intervention increase in HFnu (P=0.0071) and decreases in LF/HF (P=0.044) and LFnu (P=0.0022). The autonomic balance indicators for rising and standing did not show any exercise-induced changes. The statistical significant supine values, especially LFnu, supported the notion of an exercise-induced decrease in the sympathetic influence in the supine position.

These findings of an exercise-induced increase in vagal and decrease in sympathetic activity during rest are, although in contrast to a number of other studies,^{23,26,42} in line with the conclusions in a review by Carter et al.¹⁰ who reported endurance training to increase resting/supine HRV and parasympathetic activity while decreasing sympathetic activity.

When autonomic balance was taken into consideration, conclusions different from that of the supine was reached for the effects of the exercise intervention on the rising and standing position. In this study, in agreement with Gilder et al. (2008),²⁶ no significant changes were found in the autonomic balance indicators during either the rising or the standing periods. The non-significance of the rising and standing exercise-induced changes in the LF/HF, LFnu en HFnu were thus probably due to an equivalent exercise-induced increase in average sympathetic outflow during rising and standing.

The findings of the present study of an increased parasympathetic and decreased sympathetic control in the supine position, are in line with the beneficial effects of a physical training program on the heart and with the lowering of resting heart rate. As the weak association between the effect of exercise on the heart rate and that on the vagal influence suggests that other factors may play a role in the lowering of supine heart rate through exercise interventions, this exercise-induced reduction in the sympathetic outflow could very well make a considerable contribution.¹² In addition, an exercise-induced increase in sympathetic control during rising and standing would be in agreement with the normal homeostatic mechanisms involved in blood pressure regulation upon rising from the supine position, and with the beneficial effects of exercise to individuals prone to syncope.⁴³

Influence of the exercise intervention on the orthostatic response measured during 3 different tachogram periods

HRV quantification of the response to rising from the supine to the standing position can give valuable insight into exercise induced ANS changes. Reduced ANS responsiveness to this type of excitatory stimulus is seen as the most common feature of patho physiological states.¹⁸ It is said that postural changes, such as standing up, elicit sympathetic stimulation which, if attenuated, may be a marker of early sympathetic impairment.⁴⁴ However, it may also be an indication of exercise induced changes in the ANS. It is important to take cognisance of the fact that the orthostatic response can detect effects not visible in the supine position and that it can be a useful clinical tool to measure autonomic responsiveness, both in clinical medicine⁴⁵ and in exercise physiology.²⁶

Uniformity in the assessment of the ANS to an orthostatic response is problematic and periods and lengths of recording differ. The orthostatic response is generally seen as the difference between values obtained during the supine period and that obtained in response to the orthostatic stressor.^{18,26,43} Complicating factors include uncertainties about the exact tachogram starting point during or after standing-up, the length of recording, which is critical due to the activation and normalisation of homeostatic mechanisms,⁴⁶ and the importance of stationarity during HRV measurements.²⁰ The interpretation of when to record the values in response to the stressor differ. While the initial non-stationary period upon rising from the supine to the standing position is discarded by some authors,^{47,48} others include this period.^{43,46} Even the length of this initial period, whether included or discarded, vary from 30 seconds,⁴⁸ to two minutes,⁴⁷ to 5 minutes,⁴⁶ to 6 minutes.²³

In the present study, the vagal orthostatic response by the ANS was quantified by determining the % difference between supine vagal indicator values and that of rising/standing values at 0-180s, 180-360s and 360-540s, respectively. The difference between the pre-intervention and the post-intervention responses from supine to rising and standing are seen in Table 3. When the period during rising (standing 0-180s) was used for the calculation of the orthostatic response, highly significant exercise induced increases ($P < 0.0001$ to $P = 0.0398$) in vagal withdrawal (RMSSD, pNN50, SD1, HFms²) were found from pre- to post-exercise intervention. The exercise intervention did not change the orthostatic response as reflected by the indicators of mixed origin (SD2: $P = 0.8758$; LFms²: $P = 0.8513$). It can thus be inferred that, for the indicators of mixed origin to stay the same, in the presence of a significantly larger vagal withdrawal, the sympathetic response must have increased during post-exercise rising. This was confirmed by the significant changes ($P < 0.05$) in the values of the indicators of autonomic balance (LF/HF, LFnu and HFnu) in favour of increased sympathetic outflow, and the overall decrease in SDNN. These results are in agreement with the study by La Rovere et al. (1992)⁵⁰ who reported, after 4 weeks exercise intervention, a significant higher resting-to-tilt increase in the LF component of HRV with a significant resting-to-tilt decrease in the HF component. The initial ANS

orthostatic response to rising (0-180s), was thus significantly enhanced by the 12 week exercise intervention, both in terms of the vagal and sympathetic response.

The influence of the exercise intervention on the orthostatic response as calculated from the HRV values of the 180 to 360s period of standing minus the supine values was subsequently investigated. We have in a previous publication showed that HRV indicators already stabilized for the standing position during this period (180 to 360s after rising).⁴⁶ No significant exercise-induced changes in pure vagal HR control (RMSSD, pNN50, SD1 and HFms² were found). However, in the face of no exercise-induced change in vagal indicators, the increase in the SD2 indicator of non-linear rhythms and LFnu, showed a pre-post exercise induced increase in the sympathetic response.

In summary

The results of this study are in agreement with the concept of a lowering of heart rate, an increase in resting vagal control of the heart and a general increase in HRV by exercise. The results further confirmed the assumption that a decrease in sympathetic control contributes to exercise-induced lowering of the resting heart rate. In addition, it was shown that both vagal and sympathetic control increased during rising without redistribution of spectral frequency components. In contrast to the post-exercise increase in supine, rising and standing vagal activity, sympathetic activity, while lower at rest, was increased, not only during the period of rising, but also during the standing period. This, in the face of the post-exercise increase in vagal activity during standing, could be an expression of blood pressure maintenance in the standing position. When the effect of the exercise intervention on the orthostatic response was judged by using the values obtained over the non-stationary period (from supine-through-rising- to-standing) a significant stronger response was seen, both in terms of vagal withdrawal and sympathetic activity. However, when the influence of the exercise intervention on the orthostatic response was assessed as the difference between the stationary standing period and supine, the exercise-improved orthostatic response was indicated as a predominantly sympathetic increase. It is thus clear that different results will be obtained on the influence of exercise, depending on the time of measurement relative to body position.

Conclusions

Results on the measurement of the influence of exercise on ANS functioning are dependent on the body position and assessments should be done, not only in the resting position, but also during standing and during an orthostatic stressor. It is possible to better distinguish between exercise-induced changes in vagal and sympathetic influence by taking measurements in different body positions and during orthostatic stress. The same should be done when testing patients with cardiac pathology.

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