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**James, David V ORCID: 0000-0002-0805-7453, Munson, Steven C., Maldonado-Martin, Sara and De Ste Croix, Mark B ORCID: 0000-0001-9911-4355 (2012) Heart Rate Variability : Effect of Exercise Intensity on Postexercise Response. Research Quarterly for Exercise and Sport, 83 (4). pp. 533-539. doi:10.1080/02701367.2012.10599142**

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**James, D. V., Munson, S. C., Maldonado-Martin, S., & De Ste Croix, M. B. (2012). Heart rate variability: effect of exercise intensity on postexercise response.** *Research quarterly for exercise and sport, 83*(4), 533-539.

1 **ABSTRACT**

2 Heart rate variability (HRV) is widely considered as a non-invasive method of

3 evaluating autonomic influence on the cardiac rhythm.  Following exercise,

4 through HRV assessment, acute manipulations of autonomic influence have

5 been demonstrated, but the influence of exercise intensity remains unclear.

6 **Objective:** The aim of the present study was to investigate the influence of

7 exercise of two different intensities on HRV response following exercise.

8 **Design:** Sixteen runners completed two distance-matched conditions; moderate

9 (90% of the speed at gas exchange threshold, MOD) and severe (1 km.h$^{-1}$

10 below the speed at maximal oxygen uptake, SEV) intensity interval training

11 sessions (6 x 800m runs with 3 min recovery intervals) to study post exercise

12 HRV.  At one hour prior to (-1 h) and at +1 h, +24 h, +48 h and +72 h following

13 each exercise session participants sat quietly for 20 min whilst breathing at 0.20

14 Hz. Resting HRV data was collected over the final 5 min of each 20 min period,

15 followed by measurement of arterial blood pressure. **Results:** Time domain

16 indices and high frequency component showed a significant decrease ($p<0.001$)

17 between -1 h and +1 h in SEV and low frequency component in normalised

18 units significantly increased ($p<0.01$).  Systolic blood pressure significantly

19 decreased ($p = 0.001$) for SEV at +1 h compared with -1 h. No changes were

20 demonstrated at +24 h, +48 h, +72 h for any outcome or any condition

21 compared with -1 h. **Conclusions:** These findings suggest that severe intensity

22 exercise is required to elicit a change in HRV outcomes post exercise, resulting

23 in a reduction in parasympathetic influence on the heart at +1 h, with values

24 returning to baseline by +24 h.

25

26 **Key words:**

27 Autonomic nervous system; parasympathetic; sympathetic.

28

29

1  **Introduction**

2

3  Heart rate variability (HRV) has been established as a useful non-invasive

4  method of evaluating autonomic influence on the cardiac rhythm.[1] It is widely

5  accepted that regular exercise training induces adaptations in HRV (i.e., the

6  oscillation in the interval between consecutive heartbeats) outcomes with a shift

7  of autonomic balance towards higher parasympathetic activity, consistent with

8  improved cardiac health.[1]

9

10  Less attention has been paid to the HRV response following a single bout of

11  exercise, but there is a growing body of evidence based on studies of trained

12  athletes, [2-4] trained students, [5] moderately trained, [6,7] detrained [8] and untrained

13  active participants. [3,9-12] Findings indicate that single bout of exercise may

14  result in an increase in sympathetic influence at one hour post exercise

15  cessation [2,3,5,7,9,10,12] and that by 24 h post exercise sympathetic influence

16  returns to baseline levels. [2,7,12] Such findings are of interest, since an elevated

17  sympathetic influence has been associated with increased risk of cardiac events

18  and compromised health. [13-15]

19

20  Following an initial suppression of parasympathetic influence at one hour post

21  exercise cessation, elevated parasympathetic influence has been observed at

22  48 h following prolonged exercise. [4] In contrast, in one study, moderate

23  intensity exercise has been associated with elevations in parasympathetic

24  influence within an hour after exercise cessation. [11] This is an interesting

25  finding, since such changes are known to have a cardio-protective effect. [16]

1    Even though different studies have examined HRV responses at different

2    intensities, [7,10,12,17] to the best of our knowledge only one study by Mourot et al

3    has directly explored the impact of exercise intensity over 48 h following

4    exercise cessation. [7]  In this study, both the moderate and high intensity

5    exercise resulted in reduced parasympathetic influence at one hour following

6    exercise cessation, and there appeared to be no notable differences between

7    exercise intensity condition in the time course of the response up to 48 h post

8    exercise.  However, the study by Mourot et al [7] included a potential confounding

9    variable, since participants undertook continuous exercise for the moderate

10   intensity condition and interval exercise for the severe intensity condition.  It is

11   therefore difficult to be certain whether the findings were a result of the exercise

12   intensity or exercise type (*i.e.*, continuous versus interval). To our knowledge no

13   previous studies have examined the effect of exercise intensity, using a fixed

14   exercise type, on HRV response.

15

16   Therefore, the aim of the present study was to investigate the influence of

17   exercise of two differing intensities (moderate and severe) on the HRV

18   response post-exercise.  Both exercise conditions were applied as interval

19   exercise, and the distance covered was consistent between conditions.  We

20   were interested in the nature of the response over 72 h following cessation of

21   exercise, and the contrast in the response following the differing exercise

22   intensities.

23

24

25   **Methods**

1

2   *Participants*

3

4   Sixteen (14 male; 2 female) experienced (8.8 ± 5.3 years of training) runners

5   involved in regular endurance training sessions (3.7 ± 1.1 session.wk$^{-1}$)

6   volunteered to participate (Table 1).  All participants completed a health

7   screening procedure and were fully informed of the nature of the study.

8   Participants then provided written consent to participate.  All procedures were

9   approved by the University Research Ethics Committee.  Participants had no

10  history of diabetes, hypertension or cardiovascular disease, no symptoms of

11  underlying disease, and received no medication during the course of the study

12  for any diagnosed condition.

13

14  *Study Design*

15

16  A moderate (MOD) and severe (SEV) intensity condition were administered in a

17  counterbalanced order within a fully repeated measures design.  Both

18  conditions consisted of six 800 m bouts of running with 3 min recovery intervals.

19  The MOD was completed at 90% of the velocity at gas exchange threshold (v-

20  GET).  The SEV was completed at a velocity corresponding to 1 km·h$^{-1}$ below

21  the velocity at maximal oxygen uptake (v-VO$_{2max}$).  A minimum of 72 h

22  separated each condition and participants undertook no exercise for 72 h prior

23  to the first test within each condition.

24

1    One hour prior to (-1 h) and at various intervals following (+1 h, +24 h, +48 h,

2    +72 h) each interval exercise session, participants sat quietly for 25 min whilst

3    HRV and blood pressure were recorded.  Participants were instructed not to

4    consume alcohol or caffeine for 24 h prior to each assessment, and not to

5    consume food or fluid other than water *"ad libitum"* in the 4 h prior to each

6    assessment.  In addition to performing no exercise for 72 h prior to the first

7    assessment, participants were instructed to perform no exercise between

8    subsequent assessments.  All tests were conducted at the same time of day for

9    each participant in a comfortable temperature controlled (temperature 19 ± 2.0

10   ºC; humidity 34 ± 6 %; barometric pressure 766.2 ± 7.7 mmHg) exercise

11   physiology laboratory.  Prior to exposure to the first condition, participants

12   attended the laboratory for a familiarization visit.  In addition to familiarizing

13   participants with the procedures for the determination of HRV and blood

14   pressure outcomes, participants' descriptive anthropometric and physiological

15   characteristics were assessed, including the determination v-GET and v-VO$_{2max}$.

16

17   *HRV Outcome Measures*

18

19   According to the Task Force of the European Society of Cardiology [1] there are a

20   variety of indices that are used to assess HRV, which can be divided into two

21   major categories: time domain indices and frequency domain indices.  In the

22   present investigation the included time domain indices are the mean NN interval

23   (MNN) (*i.e.*, normal-to-normal intervals between adjacent QRS complexes), the

24   standard deviation of the NN intervals (SDNN), square root of the mean sum of

25   squares of the differences between adjacent NN intervals (rMSSD), and

1    proportion of pairs of adjacent NN intervals differing by more than 50 ms

2    (pNN50).  The included frequency domain indices are: high frequency (HF), HF

3    in normalized units ($HF_{nu}$), low frequency (LF), LF in normalized units ($LF_{nu}$),

4    very low frequency (VLF), total power (TP) and LF:HF ratio.

5

6    *Procedures*

7

8    During the HRV assessment points at -1 h, +1 h, +24 h, +48 h and +72 h,

9    participants sat quietly for 20 min and controlled their breathing frequency.

10   Frequency was set at 0.20 Hz (12 breath.min$^{-1}$), [18] with each breath comprising

11   2 s of inspiration and 3 s of expiration. [5]  Breathing frequency was paced using

12   a computer-based metronome providing both audio and visual cues.

13   Participants were instructed to maintain a normal depth of breathing throughout.

14   Participants wore a chest strap consisting of two electrodes and a transmitter

15   (T61, Polar Electro Oy, Kempele, Finland).  The NN interval data were

16   transmitted to a watch receiver (Polar S810i series) by coded short-range

17   telemetry and then to a computer via an infrared interface (Polar Electro Oy,

18   Kempele, Finland) for storage.  NN interval data were collected over the final 5

19   min of the 20 min period and stored for subsequent analysis.  The data were

20   presented graphically and visually inspected to identify any spurious beats.  No

21   spurious beats were identified, and this was confirmed with the error detection

22   algorithm in the Precision Performance analysis programme (Polar Electro Oy,

23   Kempele, Finland) which filters the data using median and moving average

24   based methods in order to identify artefacts in the signal.  During a further 5 min

25   period, participants continued to sit quietly whilst three repeat measurements of

1    arterial blood pressure were made using a clinical grade automated

2    sphygmomanometry (DynaPulse DP-200M, Pulse Metric Inc., San Diego,

3    U.S.A) according to the approach of Forjaz et al. [19] with two minutes between

4    each measurement.  Collected data were transmitted via the communication

5    cable to a computer for storage and analysis (DynaPulse software version 3.8).

6

7    The progressive exercise test and the two interval exercise sessions were

8    conducted on a motorised treadmill (Ergo ELG 70, Woodway, Weil am Rhein,

9    Germany) and for participant comfort laboratory air was circulated using a floor

10    mounted fan.  Prior to the progressive exercise test participants undertook a five

11    minute warm-up at a self selected speed below v-GET and completed their

12    personal stretching routine.  In accordance with the recommendations of

13    Buchfuhrer et al. [20] starting speed was selected so that exhaustion was

14    observed at $10 \pm 2$ min.  Participants initiated running when the treadmill had

15    reached the pre-determined start speed of 8 $km \cdot h^{-1}$.  Following two minutes at

16    the first speed, treadmill speed increased 1.2 $km \cdot h^{-1} \cdot min^{-1}$ (0.16 $km \cdot h^{-1}$ every 8

17    s) at a 0% gradient.  The progressive exercise test was used to determine

18    $VO_{2max}$, v-$VO_{2max}$, GET, v-GET and $HR_{max}$.  Throughout the test HR was

19    measured using a two-electrode chest strap and data was transmitted by coded

20    short-range telemetry to a watch receiver (S810i series, Polar Electro Oy,

21    Kempele, Finland) for storage.  During the progressive test, expirate was

22    collected continuously and analysed using a conventional Douglas bag

23    technique. [21]  The procedure for determination of $VO_{2max}$, GET and the

24    associated velocities has been described previously. [21]

25

1   *Data Analysis*

2

3   Prior to analysis, the NN interval time-series were interpolated at a rate of 4 Hz

4   and detrended using the smoothness priors approach.  NN interval data were

5   analysed using HRV Analysis Software (version 1.1 for Windows, Biomedical

6   Signal Analysis Group, University of Kuopio, Finland). [22]  A continuous five

7   minute data segment was selected for analysis consistent with previous studies

8   [5,11,23] since spectral approaches are conventionally performed on stationary

9   records of 200-500 consecutive heart beats. [24]  NN intervals were at a steady

10   level which is a requirement for the application of spectral analysis. [25]  Power

11   spectrum analysis was undertaken using the autoregressive method.

12   Autoregressive coefficients were estimated using the forward-backward-linear-

13   least-squares-algorithm with a fixed model order of 18.  Frequency bands were

14   selected conventionally [1]: VLF 0.00-0.04 Hz; LF 0.04-0.16 Hz and HF 0.16-0.40

15   Hz.  TP and LF:HF ratio was also computed.  The LF and HF power

16   components were also expressed using normalized units (nu) providing a

17   relative value for each power component. [1]  The normalization procedure

18   requires dividing either HF or LF component ($ms^2$) by total spectral power (TP)

19   minus the VLF component ($ms^2$) [1] which results in a dimensionless ratio. [5]

20   Normalization is suggested to result in a reduction of the effect total power

21   changes would have on LF and HF components. [1,26]  HRV outcomes in the

22   frequency domain are not normally distributed [27] so, consistent with the

23   approach of Bernardi et al. [2] and James et al., [5] these data were transformed

24   using the natural logarithmic function prior to statistical analysis.  The

25   logarithmic transformation provides data that meets the assumptions required

1 for parametric statistical analysis. [11] Consistent with James et al., [5] all data are

2 presented as mean (68% confidence interval) as it is not possible to 'back-

3 transform' a log transformed standard deviation into the original measurement

4 unit. The presentation of data as mean (68% confidence interval) is consistent

5 with the normal convention of presenting data as mean (one standard

6 deviation).

7

8 Interactions between condition (exercise intensity; SEV or MOD) and time (-1 h,

9 +1 h, +24 h, +48 h, +72 h) were explored using 2 x 5 fully repeated measures

10 ANOVA. Whether or not a significant interaction was present, we were also

11 interested in main effects for time, since we were interested in the response

12 following each condition. One-way repeated measures ANOVA were

13 conducted for each condition to examine effects over time. When significant

14 main effects were observed, post-hoc t-tests were conducted to locate the

15 differences. Differences were considered significant when $p < 0.05$.

16

17 **Results**

18

19 Participant characteristics are presented in Table 1. The heart rate responses

20 to the moderate and severe interval exercise bouts are shown in Figure 1. The

21 mean (68% confidence interval) heart rate during the moderate exercise

22 demonstrated a slight increase from 131 (123-139) to 139 (128-150) b·min$^{-1}$

23 across the six 800 m bouts (i.e., 68-72 %$HR_{max}$). Heart rate during the severe

24 exercise increased significantly ($p < 0.01$) from 165 (156-174) to 178 (167-189)

25 b.min$^{-1}$ across the six bouts (i.e., 86-93% $HR_{max}$).

1

2 Data for the time domain and frequency domain HRV parameters are presented

3 in Table 2 and 3, respectively.

4

5 **Time domain outcomes.** Interactions between condition and time were

6 revealed for SDNN, rMSSD and pNN50, but not for MNN.  Post-hoc analysis

7 revealed lower values for SEV than MOD at +1 h ($p < 0.01$) for all time domain

8 outcomes (MNN, 11%; SDNN 31%; rMSSD 12%; pNN50 55%).  Main effects

9 for time were revealed for SEV for all time domain outcomes ($p < 0.001$) but not

10 for MOD.  Compared to the values at -1 h, values at +1 h were significantly ($p <$

11 $0.001$) decreased for SEV condition (MNN, 20%; SDNN, 39%; rMSSD, 54%;

12 and pNN50, 68%) but values returned to baseline levels by +24 h (Table 2).

13

14 **Frequency domain outcomes**. A significant interaction ($p = 0.001$) between

15 condition and time was observed for TP, with the value at +1 h significantly

16 lower (50%, $p = 0.003$) for SEV (404ms²) than MOD (810ms²).

17

18 A main effect for time for SEV was revealed for TP ($p < 0.001$), $HF_{ms}{}^{2}$ ($p <$

19 $0.001$), $LF_{nu}$ ($p = 0.003$) and $HF_{nu}$ ($p = 0.021$), with post-hoc analysis revealing a

20 significant decrease for TP (64%, $p < 0.001$), $HF_{ms}{}^{2}$ (78%, $p < 0.001$) and $HF_{nu}$

21 (47%, $p = 0.008$) respectively, and a significant increase for $LF_{nu}$ (42%, $p =$

22 $0.007$) between -1 h and +1 h, but all of these values had returned to baseline

23 by +24 h.  No condition by time interaction was revealed (Table 3).

24

1  No interaction between condition and time, and no main effects for time were

2  revealed for the LF: HF ratio (Table 3).

3

4  **Blood pressure outcomes**. Although no interaction was revealed between

5  condition and time for either systolic or diastolic blood pressures, a main effect

6  for time was revealed for systolic blood pressure for SEV exercise, with post

7  hoc analysis indicating a significant decrease (7%, $p = 0.001$) at +1h compared

8  with -1h but values had returned to baseline by +24 h (Table 4).

9

10

11  **Discussion**

12

13  Although an increasing number of studies have investigated the HRV response

14  following a single bout of exercise, the majority of these studies have included

15  either one exercise intensity, [5,8,9,11] or those that have included different

16  intensities [7,10,12,17] have not assessed HRV after long term recovery (i.e., >24h).

17  This growing body of evidence is further compounded, for comparative

18  purposes, by the diverse range of exercise bouts and the methodological

19  differences between the studies.  Therefore, to our knowledge, the present

20  study is the first to explore the influence of exercise intensity alone on the post-

21  exercise HRV response in a controlled design.

22

23  The findings of the present study suggest that severe intensity exercise reduces

24  indicators of parasympathetic influence on the heart (MNN, SDNN, rMSSD,

25  pNN50, HF, $HF_{nu}$) and increases an indicator of sympathetic influence on the

1    heart ($LF_{nu}$) at +1 h following the exercise bout.  Interestingly, these changes

2    were observed in the presence of a reduction in systolic blood pressure.  All

3    changes were reversed by +24 h, and values remained constant at +24h, +48 h

4    and + 72 h at the baseline (i.e., -1 h) level.

5

6    An increase in sympathetic influence on the heart between 30 min and one-hour

7    post exercise cessation has been noted in a number of studies. [2,3,7,9]  A

8    decrease in parasympathetic influence on the heart between 30 min and one-

9    hour post exercise cessation has also been noted in a number of studies with a

10   decrease in the HF component of the total power spectrum consistently

11   observed. [2,3,5,7,9]  Reductions in other indicators of parasympathetic influence

12   have also been observed, including $HF_{nu}$ [3,9] and SDNN. [2,5]

13

14   It is possible that the elevation in sympathetic influence and accompanying

15   reduction in parasympathetic influence on the heart at one-hour post severe

16   intensity exercise cessation in the present study was a compensatory response

17   to a reduced systolic blood pressure.  A reduced systolic blood pressure at one

18   hour following exercise cessation has been reported previously in normotensive

19   participants. [28]  It is interesting that, even in the presence of an increased

20   sympathetic influence and an associated decrease in MNN in the present study,

21   blood pressure was not restored to baseline values.  We cannot be sure that the

22   increased heart rate (i.e., reduced MNN) provided an increased cardiac output,

23   so the shortfall in systolic blood pressure at one hour is either a result of

24   reduced stroke volume (and therefore cardiac output) or reduced vascular

25   resistance.  A reduced stroke volume at rest has been reported following a

1    single bout of exercise, [29] although typically such reductions have been

2    demonstrated following very prolonged exercise. [29]  Reduced arterial

3    resistance, as indicated by increased arterial diameter, has also been reported

4    at one-hour post exercise. [30]

5

6    In many of the previous studies in this area, it is difficult to determine the exact

7    exercise intensity from the information provided, not least due to the lack of data

8    regarding the physiological response to the exercise bout.  Perhaps this is not

9    surprising, given that these studies were not primarily designed to compare the

10   responses following exercise of differing intensity.  Mourot et al [7] compared

11   continuous and interval exercise where the work done was fixed at 9.4 kJ.kg$^{-1}$.

12   In the present study, interval exercise was conducted at 1 km.h$^{-1}$ below the

13   velocity at $VO_{2max}$ (i.e., severe intensity) and 90% of the velocity at GET (i.e.,

14   moderate intensity), respectively.  It was important to ensure that the exercise

15   that took place was genuinely of a moderate and severe intensity respectively,

16   thereby ensuring significantly different exercise intensities in the two conditions.

17   The physiological responses to exercise at these two intensities confirmed the

18   differing nature of the physiological challenge, where 86-93 $\%HR_{max}$ was

19   attained in the severe intensity exercise condition as opposed to 68-72 $\%HR_{max}$

20   in the moderate condition.  It was considered particularly important to ensure

21   that the moderate intensity condition was completed at a speed below that

22   attained at gas exchange threshold.

23

24   The findings of the present study have implications for practice.  In particular, it

25   appears that severe intensity exercise may acutely alter the autonomic

1   influence on the heart post-exercise towards a greater sympathetic influence.

2   This suggests that for the duration of this changed autonomic influence, it is

3   likely that the exercise participant is at an increased risk of cardiac events such

4   as arrhythmias.  Although it is well recognised that the acute risks of conducting

5   high intensity exercise are far outweighed by the chronic benefits of regular

6   moderate exercise in the healthy population, [31] a cautious approach might be

7   necessary in higher risk populations particularly when exercise intensity

8   exceeds a moderate intensity.  It is also important to note that in the present

9   study, a moderate intensity exercise bout had no influence on post-exercise

10  HRV at +1 h compared with baseline values.  It would therefore appear that

11  moderate intensity exercise (86-93 $\%HR_{max}$) may present less of an acute risk.

12

13  In summary, the findings of the present study demonstrate a clear intensity

14  effect on the post-exercise heart rate variability response, where severe

15  intensity exercise results in a reduced parasympathetic influence on heart

16  rhythm at one hour after exercise cessation.  In contrast, moderate intensity

17  exercise results in no change in either sympathetic or parasympathetic

18  influence on the heart.  Future studies should explore the time course of the

19  heart rate variability response following severe intensity exercise, particularly

20  between one hour and 24 h following exercise cessation.

21

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3

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