Endurance Exercise Improves Heart Rate Complexity in the Presence of Vagal Withdrawal in Young Adults

Steven E Perkins¹, Herbert F Jelinek¹, Berverlie de Jong¹, David J Cornforth², Mika P Tarvainen^{3,4}, Hayder A. Al-Aubaidy⁵

¹Charles Sturt University, Albury, NSW, Australia

²Applied Informatics Research Group, Faculty of Science and IT, The University of Newcastle,

Callaghan, NSW, Australia

³University of Eastern Finland, Kuopio, Finland

⁴Kuopio University Hospital, Kuopio, Finland

⁵University of Tasmania, Hobart, Tasmania, Australia

Abstract

Sudden cardiac death (SCD) has been reported during and following physical activity. SCD may be due to vagal withdrawal and/or sympathetic dominance associated with the exercise occurring at any time during, immediately following, or up to several days after exercise. Heart rate variability (HRV) describes the influence of the autonomic nervous system on heart rate. We assessed the immediate post-exercise influence of endurance training on HRV in young adults following the morning exercise bout and also on the same day in the afternoon. Linear domain parameter root mean square of successive RR interval differences (RMSSD) showed vagal withdrawal when analysed both immediately after the AM session and also when pre exercise HRV was compared to post exercise HRV during the early evening (median average change 6.6%) suggesting a possible increased risk of adverse cardiac events. However multiscale Rényi entropy indicated either no change immediately following the exercise for all scaling factors or an increase in HRV complexity of the heart rate for the afternoon recordings. Despite decreased vagal influence, endurance training may be protective for some individuals that retain a higher heart rate complexity in the presence of vagal withdrawal.

1. Introduction

Heart rate variability (HRV) analysis measures the autonomic modulation of the heart rate. Time domain analysis for HRV is based on mean and standard deviation calculations over a series of RR intervals usually totalling from 5 minutes to 24 hours [1]. Time domain analysis includes the measure of root mean sum of squared differences (RMSSD), which provides information about the parasympathetic modulation of the heart rate. Frequency domain analysis involves the division of the heart rate signal into discrete frequencies,

which provide measures of sympathovagal balance and parasympathetic modulation of the heart rate. However, analyses within time and frequency domain are sensitive to ectopic beats and other noise in the heart rate signal and hence are limited in the information they can provide about the HRV.

Nonlinear HRV analyses including entropy and fractal based analyses quantify the complexity of the autonomic modulation of the heart rate and are less sensitive to noise in the heart rate signal [2, 3]. These additional methods of HRV analysis can therefore provide complementary information to time and frequency domain analyses about the overall HRV following exercise.

Endurance exercise has various long term health benefits for all ages in both healthy and diseased populations including increased quality of life and decreased mortality [4]. In terms of cardiovascular health endurance exercise decreases resting heart rate and is accompanied by a decrease in resting sympathetic activity and increased parasympathetic activity within 12-weeks of training [5]. However, during and immediately following exercise heart rate is acutely increased, parasympathetic influence is decreased and sympathetic increased [6]. This time frame is also associated with an increased risk of adverse cardiac events including sudden cardiac death (SCD) [7]. Immediately following exercise HRV complexity has been shown to increase as measured by sample entropy (SampEn) and detrended fluctuation analysis (DFA) [8].

1.1. Multiscale Rényi entropy

Applications of multiscale measures including Rényi entropy and applied to physiologic time series has been discussed in [9]. Rényi entropy H is a generalization of the Shannon entropy:

$$H_{\alpha}(X) = \frac{1}{1-\alpha} \log_2\left(\sum_{i=1}^n p_i^{\alpha}\right)$$

where α is an exponent which determines the order. In

terms of deriving a measure from a recording of R-R intervals, X is the vector of R-R intervals and p_i is the probability of each sub-sequence of X. This can be estimated by its similarity with all other sequences of the same length p_i [10]. Each sub-sequence is regarded as a point in a π -dimensional space, and its probability is estimated using a Gaussian kernel centered on each such point [11]. Then p_i is given by this density function:

$$p_i = \sum_{j=0}^n exp\left(\frac{-dist_{ij}^2}{2\sigma^2}\right)$$

where σ is a parameter controlling the width of the density function and *dist()* is a distance measure:

$$dist_{ij} = \sum_{k=0}^{\pi} (x_{i+k} - x_{j+k})^2$$

Here, x_{i+k} is one R-R sample out of sequence of length π , the pattern length over which comparison occurs. Rényi entropy may be calculated for a range of α providing a spectrum of measures.

1.2. Moments

Moments are measures of distribution, in this work the distribution of R-R intervals. The familiar arithmetic mean and variance can be informally viewed as moments of order 1 and 2 respectively, where order refers to the exponent used in calculating these values. Higher order moments can be defined as:

$$m_k = E[(X - \mu)^k]$$

where E[x] is the expectation of X, and μ is the arithmetic mean of the variable X.

2. Methods

This study was completed as part of a larger study titled "Recovery of the autonomic modulation of the heart following a single session of exercise; a comparison between high intensity interval exercise and endurance exercise." This study was approved by the Human Research Ethics Committee (Charles Sturt University, Albury, Australia), protocol number: 2014/161.

2.1. Participants

Participants were recruited through flyers, personal contacts and from student lectures. Inclusion criteria were as follows: age: 18-30 years, within a healthy weight range (18-30 kgm⁻²), normotensive (blood pressure: 90/60 - 140/90 mmHg), not taking medications that affect the heart, no diagnosis of an acute or chronic health condition and not having trained more than 2 times per week in the last 3 months. Prior to participation participants were

screened with the Exercise and Sports Science Australia adult Pre-Exercise Screening Tool (2011) stages 1 and 2. All experimental procedures were explained and participants provided written informed consent. Nine students volunteered to participate, all met the inclusion criteria, and were safe to participate in moderate intensity endurance exercise.

2.2. Endurance exercise

The endurance exercise intervention consisted of a single moderate intensity exercise bout of 45 minutes duration on an air-braked ergometer cycle (Wattbike Ltd, Nottingham, UK). The exercise for each participant was always completed between 0800 and 1000 hours to minimize potential circadian variation. Moderate intensity was defined as 55-70% maximum heart rate with maximum heart rate calculated as equal to 208 - 0.7*(age) [12]. 62.5% of maximum heart rate was calculated for each participant and participants were asked maintain this heart rate for the 45 minutes.

2.3. **RR** interval samples

All samples were obtained using a heart rate monitor (RS800CX; Polar Electro Ltd.) with chest strap set to record RR intervals to an accuracy of 1 ms. Two hour ambulatory samples were obtained prior to and immediately following the exercise intervention in the morning. RR intervals were also recorded in the afternoon between 1800 and 2000 hours, which coincides with an increased period for heart attacks [13]. Two hour samples were then obtained for the three days following the exercise between 0800 and 1000 hours. Participants were required to abstain from alcohol and caffeine for the 12 hours prior to the intervention and for the four days post intervention, and from exercise 48 hours prior to the intervention and for 4 days following the intervention. Data were extracted from the heart rate watch and to computer for analysis.

2.4. RR interval analysis

Prior to analysis all two-hour samples were preprocessed to remove all artefacts and ectopic beats. Frequencies below the LF band (below 0.04 Hz) were filtered out from the RR time series by using smoothness priors detrending [14]. Furthermore, the RR interval time series were interpolated using 4 Hz cubic spline interpolation to have evenly sampled data for spectral analysis. Time and frequency feature were calculated for the two-hour samples using Kubios HRV software [15] and Rényi entropy with purpose written software in our laboratory respectively.

2.5. Statistical analysis

A nonparametric Mann-Whitney test was applied for assessing differences between pre and post exercise recordings. Significance was set at p < 0.05.

3. **Results**

Standard time and frequency HRV features did not show any significant differences between pre and posttraining for the morning recordings. A trend (p < 0.08) was however observed for DAF α 2 and Sample Entropy. Complexity measured by Rényi entropy increased immediately following exercise in the morning and nearly reached significance for settings of $\pi = 8$ and $\alpha = -2$ with p = 0.052. Our application of moments indicated a significant difference for Moment 10 (p=0.046) with higher even moments (14 to 18) also trending to significance (p < 0.08).

Afternoon recordings in contrast to the morning HRV results showed significant changes only with moment statistics. RMSSD was still lower following the exercise but again did not reach significance (Figure 1).

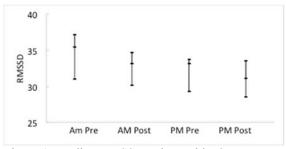


Figure 1. Median RMSSD values with IQR

Significant differences were obtained only for uneven moments from moment 7 to moment 19 with p values below 0.05. The best discrimination was observed with moment 7 at a p value of 0.006.

When comparing longer time periods post exercise a significant change was observed between post 24 to post 48 hours and post 48 to post 72 hours (Figure 1). Here significant differences were detected between HRV at 24 and 48 hours post exercise for some values of Rényi entropy. Significant differences (p = 0.024, 0.022, 0.042) were obtained between 24 and 48 hours for Rényi Entropy using $\pi = 8$ ($\alpha = -5, -4, -3$). These significant results were not seen for any other measures derived from HRV. Notice also the trend suggested by Figure 2, where complexity indicated by Rényi entropy is increased 72 hours after exercise. The figure is but one example, and similar features are apparent in results for other settings of the Rényi parameters.

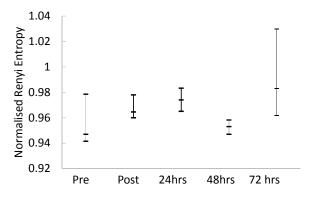


Figure 2. Normalized Rényi Entropy values from immediately following exercise and between 0800 and 1000 AM for the following days for different phases of the endurance trial obtained using sequence length $\pi = 8$ and alpha $\alpha = -4$.

4. Discussion

The important links between exercise, SCD and the autonomic control of the heart have prompted much research on the how cardiac autonomic modulation is affected post exercise. However, the majority of these investigations have used linear methods of HRV analysis to assess this. Although nonlinear HRV analysis is a prognostic indicator of many chronic health conditions, only a few studies have investigated the nonlinear HRV variation following endurance exercise. In the present study we examined post exercise nonlinear HRV using multiscale Rényi entropy.

The results show changes in HRV complexity immediately following morning endurance exercise that remained into the late afternoon. These changes occurred despite a minimal decrease detected in the linear HRV measure of RMSSD. Changes in some specific measures of Rényi entropy and moment statistics were detected at 72 hours following exercise.

The findings of increased Rényi entropy and moment statistics calculated within the day of the exercise are consistent with previous published studies that used other nonlinear HRV variables showing that the complexity of the heart rate signal remains deviated from baseline levels [16]. The increase in the complexity of the heart rate signal may potentially reflect the cardio protective effect of endurance exercise.

Contrarily, complexity as measured via DFA α 2 and SampEn decreased, potentially indicating a loss of longrange correlation and overall complexity. The contrast in these two findings on the complexity is unable to be explained by the data collected in this study however Rényi entropy may be more sensitive to heart rate variability.

It remains unclear as to how long range cardiac autonomic modulation is affected following moderate

intensity endurance exercise. We found various sequence lengths of Rénvi entropy to be significantly varied from their baseline up to 72 hours post exercise when measured in the morning. However, the majority of previous studies investigating recovery of the autonomic control of the heart following moderate intensity endurance exercise have taken samples within few hours immediately following exercise. Furthermore, those that have investigated longer timelines of 24 to 72 hours have confined their HRV analyses to linear methods. Following moderate intensity endurance training it was concluded that full parasympathetic recovery occurs within 24 hours post exercise [17]. One study observed decreased mean RR intervals at 24 and 48 hours post endurance exercise in an upright position despite no difference when measured with the participants in supine position or differences in any other measure of parasympathetic activity [18].

The deviations measured with Rényi entropy beyond 24 hours post exercise from baseline measures potentially indicate that although parasympathetic autonomic activity has recovered the heart rate signal may remain influenced by moderate intensity endurance exercise. Due to the potentially higher sensitivity of Rényi entropy these measures may provide additional information on the autonomic modulation of the heart rate following exercise.

Acknowledgements

The authors wish to acknowledge Roche Australia for providing blood glucose measurements strips.

References

- Task Force. Heart rate variability: standards of measurement, physiological interpretation and clinical use. Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology. Circulation. 1996 Mar 1;93(5):1043-65.
- [2] Tulppo MP, Kiviniemi AM, Hautala AJet al. Physiological background of loss of fractal heart rate dynamics. Circulation. 2005;112:314-9.
- [3] Heffernan KS, Fahs CA, Shinsako KKet al. Heart rate recovery and heart rate complexity following resistance exercise training and detraining in young men. Am J Physiol - Heart Circ Physiol. 2007 November 2007;293(5):H3180-H6.
- [4] Garber CE, Blissmer B, Deschenes MRet al. American College of Sports Medicine position stand. Quantity and quality of exercise for developing and maintaining cardiorespiratory, musculoskeletal, and neuromotor fitness in apparently healthy adults: guidance for prescribing exercise. Med Sci Sports Exerc. 2011 Jul;43(7):1334-59.

- [5] Melanson EL and Freedson PS. The effect of endurance training on resting heart rate variability in sedentary adult males. Eur J Appl Physiol. 2001 Sep;85(5):442-9.
- [6] Borresen J and Lambert MI. Autonomic control of heart rate during and after exercise : measurements and implications for monitoring training status. Sports medicine (Auckland, N.Z.). 2008;38(8):633-46.
- [7] Dangardt FJ, McKenna WJ, Luscher TFet al. Exercise: friend or foe? Nat Rev Cardiol. 2013 Sep;10(9):495-507.
- [8] Millar PJ, Rakobowchuk M, McCartney Net al. Heart rate variability and nonlinear analysis of heart rate dynamics following single and multiple Wingate bouts. Applied Physiology, Nutrition, and Metabolism. 2009;34(5):875-83.
- [9] Cornforth D, Tarvainen M and Jelinek HF. How to Calculate Rényi Entropy from Heart Rate Variability, and Why it Matters for Detecting Cardiac Autonomic Neuropathy. Frontiers in Bioengineering and Biotechnology. 2014 2014-September-9;2.
- [10] Jelinek HF, Tarvainen MP and Cornforth DJ. Rényi entropy in the identification of cardiac autonomic neuropathy in diabetes. Computing in Cardiology. 2012;39:909-11.
- [11] Bayes T. An essay towards solving a problem in the doctrine of chances. Philos Trans R Soc Lond B Biol Sci.1763(53):370-418.
- [12] Tanaka H, Monahan KD and Seals DR. Age-predicted maximal heart rate revisited. J Am Coll Cardiol. 2001 Jan;37(1):153-6.
- [13] Messa JBL, Leiza JRG, Garcia MDAet al. Cardiovascular Risk Factors in the Circadian Rhythm of Acute Myocardial Infarction. Rev Esp Cardiol. 2004;57(9):850-8.
- [14] Tarvainen MP, Ranta-Aho PO and Karjalainen PA. An advanced detrending method with application to HRV analysis. IEEE transactions on bio-medical engineering. 2002 Feb;49(2):172-5.
- [15] Nemeroff CB and Musselman DLAHJs-. Are platelets the link between depression and ischemic heart disease? . American Heart Journal. 2000;140(suppl):57-62.
- [16] Armstrong RG, Kenny GP, Green Get al. Diurnal variation in heart rate variability before and after maximal exercise testing. Chronobiology International. 2011;28(4):344.
- [17] Stanley J, Peake JM and Buchheit M. Cardiac parasympathetic reactivation following exercise: implications for training prescription. Sports Medicine. 2013 Dec;43(12):1259-77.
- [18] Mourot L, Bouhaddi M, Tordi Net al. Short- and long-term effects of a single bout of exercise on heart rate variability: comparison between constant and interval training exercises. European Journal of Applied Physiology. 2004;92(4-5):508-17.

Address for correspondence.

Herbert Jelinek School of Community Health Thurgoona Drive Albury NSW 2460 Australia hjelinek@csu.edu.au