

## Research Space

Journal article

**Left ventricular mechanical, cardiac autonomic and metabolic responses to a single session of high intensity interval training.**  
**Edwards, Jamie, Wiles, Jonathan D., Vadaszy, Noemi, Taylor, Katrina A. and O'Driscoll, Jamie**

This is the accepted version of:

Edwards, J.J., Wiles, J.D., Vadaszy, N. *et al.* Left ventricular mechanical, cardiac autonomic and metabolic responses to a single session of high intensity interval training. *Eur J Appl Physiol* (2021). <https://doi.org/10.1007/s00421-021-04840-z>

1 **Left ventricular mechanical, cardiac autonomic and metabolic responses to a single**  
2 **session of high intensity interval training.**

3

4 Jamie J. Edwards<sup>1</sup>., Jonathan D. Wiles<sup>1</sup>., Noemi Vadasz<sup>1,2</sup>., Katrina A. Taylor<sup>1</sup>., & Jamie M.  
5 O'Driscoll<sup>1</sup>.

6

7 <sup>1</sup>School of Psychology and Life Sciences, Canterbury Christ Church University, Kent, CT1  
8 1QU, UK

9

10 <sup>2</sup>Leicester Kidney Lifestyle Team, Department of Health Sciences, University of Leicester,  
11 UK.

12

13 Correspondence to Dr Jamie O'Driscoll, School of Psychology and Life Sciences, Canterbury  
14 Christ Church University, North Holmes Road, Canterbury, Kent, CT1 1 QU. Email:  
15 [jamie.odriscoll@canterbury.ac.uk](mailto:jamie.odriscoll@canterbury.ac.uk); Telephone: 01227782711.

16

17

18

19

20

21

22

23

24

25 **Abstract**

26 **Purpose:** High intensity interval training (HIIT) produces significant health benefits. However,  
27 the acute physiological responses to HIIT are poorly understood. Therefore, we aimed to  
28 measure the acute cardiac autonomic, haemodynamic, metabolic and left ventricular  
29 mechanical responses to a single HIIT session.

30 **Methods:** Fifty young, healthy participants completed a single HIIT session, comprising of  
31 three 30-second maximal exercise intervals on a cycle ergometer, interspersed with 2-minutes  
32 active recovery. Cardiac autonomies, haemodynamics and metabolic variables were measured  
33 pre, during and post HIIT. Conventional and speckle tracking echocardiography was used to  
34 record standard and tissue doppler measures of left ventricular (LV) structure, function and  
35 mechanics pre and post HIIT.

36 **Results:** Following a single HIIT session, there was significant post-exercise systolic  
37 hypotension ( $126\pm 13\text{mmHg}$  to  $111\pm 10\text{mmHg}$   $p<0.05$ ), parallel to a significant reduction in  
38 total peripheral resistance ( $1640\pm 365\text{dyne}\cdot\text{s}\cdot\text{cm}^5$  to  $639\pm 177\text{dyne}\cdot\text{s}\cdot\text{cm}^5$ ,  $p<0.001$ ) and  
39 significant increases in baroreceptor reflex sensitivity and baroreceptor effectiveness index  
40 ( $9.2\pm 11\text{ms}\cdot\text{mmHg}^{-1}$  to  $24.8\pm 16.7\text{ms}\cdot\text{mmHg}^{-1}$  and  $41.8\pm 28$  to  $68.8\pm 16.2$ , respectively) during  
41 recovery compared to baseline. There was also a significant increase in the low to high  
42 frequency heart rate variability ratio in recovery ( $0.7\pm 0.48$  to  $1.7\pm 1$ ,  $p<0.001$ ) and significant  
43 improvements in left ventricular global longitudinal strain ( $-18.3\pm 1.2\%$  to  $-29.2\pm 2.3\%$ ,  
44  $p<0.001$ ), and myocardial twist mechanics ( $1.27\pm 0.72^\circ\cdot\text{cm}^{-1}$  to  $1.98\pm 0.72^\circ\cdot\text{cm}^{-1}$ ,  $p=0.028$ ) post  
45 HIIT compared to baseline.

46 **Conclusion:** A single HIIT session is associated with acute improvements in autonomic  
47 modulation, haemodynamic cardiovascular control and left ventricular function, structure and

48 mechanics. The acute responses to HIIT provide crucial mechanistic information, which may  
49 have significant acute and chronic clinical implications.

50

51 **Key Words:** High intensity interval training, cardiac autonomies, metabolism, cardiac  
52 mechanics.

53

54 **Abbreviations:**

55 Baroreceptor Effectiveness Index (BEI)  
56 Baroreceptor sensitivity (BRS)  
57 Blood pressure (BP)  
58 Diastolic blood pressure (dBp)  
59 End diastolic volume (EDV)  
60 Heart Rate (HR)  
61 Heart rate variability (HRV)  
62 High Frequency (HF)  
63 High intensity interval training (HIIT)  
64 Left Ventricle (LV)  
65 Low Frequency (LF)  
66 Moderate intensity continuous training (MICT)  
67 Respiratory exchange ratio (RER)  
68 Stroke Volume (SV)  
69 Systolic blood pressure (sBP)  
70 Task Force Monitor (TFM)  
71 Total peripheral resistance (TPR)

72

73

74

75

76

77

78

79

80 **Introduction**

81 Physical inactivity is associated with the progression of numerous chronic health conditions,  
82 which increases the risk of all-cause mortality (Ekelund et al. 2016). It is well-established that  
83 achieving the current physical activity guidelines improves health outcomes (World Health  
84 Organization 2015). Despite this, physical inactivity remains detrimentally high at an estimated  
85 27.5% globally (Guthold et al. 2018) and adherence to physical activity guidelines may be as  
86 low as 5% when measured objectively (Troiano et al. 2008).

87

88 Behavioural psychology research has identified motivation and perceived lack of time as the  
89 most common barriers to physical activity, which are therefore targeted areas for behaviour  
90 change (Herazo-Beltrán et al. 2017). One proposed approach is to increase exercise efficiency  
91 through a reduction in duration while attempting to maintain similar health benefits. High-  
92 intensity interval training (HIIT) is an exercise modality, which supports this approach through  
93 its combination of practicality and efficacy. HIIT is a convenient, time-efficient form of  
94 exercise which typically involves short bouts of high intensity work separated with appropriate  
95 active recovery periods. HIIT has seen significant empirical success in improving health  
96 measures with multiple meta-analyses supporting its role in weight loss, aerobic capacity and  
97 cardiometabolic health; as well as promoting positive psychological responses, which have  
98 implications for adherence (Batacan et al. 2017; Oliveira et al. 2018; Roy et al. 2018; Cao et  
99 al. 2019).

100

101 Mechanistically, much of the reported benefits of HIIT are associated with chronic peripheral  
102 adaptations regarding mitochondrial content, capillary density, insulin sensitivity, glycaemic  
103 control, and vascular health (MacInnis and Gibala 2017). Our current understanding of any

104 myocardial adaptations associated with HIIT is based upon the work of O’Driscoll et  
105 al.,(O’Driscoll et al. 2018) who reported significant improvements in left ventricular function  
106 and mechanics, as well as a significant increase in cardiac autonomic modulation following a  
107 2-week HIIT intervention. Whilst the training effects of HIIT have been previously  
108 documented, the acute responses are not well characterised and may provide important  
109 mechanistic information for the chronic adaptations reported following HIIT.

110

111 To our knowledge, no study to date has attempted to measure the combined cardiac autonomic,  
112 continuous haemodynamic, metabolic and myocardial functional, structural and mechanical  
113 responses to HIIT. With the combination of these measurements, the aim of this study is to  
114 clearly establish the acute physiological responses to a single session of HIIT in a cohort of  
115 physically inactive adults. We hypothesize acute improvements in cardiac autonomic and  
116 haemodynamic modulation, and myocardial mechanics following HIIT.

117

118

119

120

121

122

123

124

125

126 **Methodology**

127

128 ***Ethical Approval***

129 This research was approved by the Canterbury Christ Church University Ethics Committee and  
130 conformed to the Declaration of Helsinki principles (Ref: 17/SAS/47F). All participants  
131 completed and signed informed consent before testing.

132

133 ***Participant characteristics***

134 Fifty (25 male and 25 female) young, healthy participants were recruited. All participants (age  
135  $22.87 \pm 2.58$  years; height  $171.3 \pm 9.5$  cm; weight  $73.8 \pm 14.9$  kg; BMI  $25.24 \pm 4.47$  kg/m<sup>2</sup>)  
136 had blood pressure within the normal range, were taking no medication, had no history of  
137 cardiac or metabolic disease, and with a normal clinical cardiovascular examination and 12-  
138 lead electrocardiogram. All participants were physically inactive, as defined by not meeting  
139 the current global physical activity guidelines (World Health Organization 2010).

140

141 ***Experimental procedures***

142 Participants were required to visit the laboratory on a single occasion after fasting for 8 hours  
143 and refraining from alcohol and caffeine consumption for 24-hours prior to testing. On arrival,  
144 the participants height and weight were measured using a SECA 213 stadiometer and SECA  
145 700 mechanical column scales (SECA GmbH & Co., Hamburg, Germany) respectively.  
146 Resting blood pressure (BP) was measured according to the current guidelines (Whelton et al.  
147 2018) using an automated oscillometric blood pressure monitor (Dinamap Pro 200 Critikon;  
148 GE Medical Systems, Freiburg, Germany).

149 *Cardiac autonomic and Haemodynamic assessment*

150 Cardiac autonomic and haemodynamic variables were measured using the Task Force  
151 Monitor (TFM) which is a validated non-invasive beat-to-beat monitoring system providing  
152 automatic calculations of all outputs. The TFM continuously recorded heart rate and stroke  
153 volume through a six-channel electrocardiogram and impedance cardiography respectively.  
154 The impedance cardiography functioned via an electrode strip located at the nape of the neck  
155 and two electrodes on the torso in line with the xiphoid process. With the recording of these  
156 two values (HR and SV), cardiac output was automatically calculated. Additionally, total  
157 peripheral resistance was calculated in accordance with Ohm's law. Continuous systolic,  
158 diastolic and mean blood pressure (sBP, dBP and mBP) measurements were obtained via the  
159 use of the vascular unloading technique at the proximal limb of the index or middle finger.  
160 These recordings were automatically corrected to oscillometric BP values obtained at the  
161 brachial artery of the opposite arm. With the sBP and heart rate recordings, the TFM  
162 calculated continuous rate pressure product measurements.

163

164 Through power spectral analysis and an autoregressive model, cardiac autonomic variables  
165 were obtained via assessment of the amplitude of R-R intervals and oscillating fluctuations in  
166 frequency (Akselrod et al. 1981). Using the TFM automatic QRS algorithm, high and low  
167 frequency parameters of heart rate variability were calculated and automatically expressed in  
168 both absolute ( $\text{ms}^2$ ) and normalised units (nu) (Pan and Tompkins 1985)(Li et al. 1995). As  
169 separate mechanistic measures, baroreceptor sensitivity and baroreflex effectiveness index  
170 were recorded via the sequence method which relies on the linear regression of continuous  
171 changes in sBP and the lengthening or shortening of the R-R interval (Taylor et al. 2017).



172 From all regressions, a mean slope of BRS was calculated and only sections with correlation  
173 coefficients of  $r > 0.95$  were analysed.

174 Intervention stages were used to distinguish and separate specific periods of measurement for  
175 appropriate data organisation. Using the intervention marks, cardiac autonomic and  
176 haemodynamic measurements were continuously recorded during a 5-minute pre-exercise  
177 rest period, which is presented as baseline. Recording then proceeded during the three  
178 separate 30-second exercise periods, which correspond to HIIT 1, HIIT 2 and HIIT 3, and the  
179 2-minute rest periods in between each exercise interval were also recorded. Finally, a 5-  
180 minute recovery period was recorded immediately post-exercise with the participant in a  
181 supine position.

182

### 183 *Metabolic measures*

184 Gas exchange measures were acquired using the Oxycon Pro (Jaeger, Wurzburg, Germany)  
185 online gas analyser. Prior to testing, calibration of the gas cylinder was performed to  
186 appropriate concentrations (15% O<sub>2</sub>; 5% CO<sub>2</sub>). Additionally, flow was calibrated using a 3-L  
187 syringe (Cosmed, Rome, Italy). Participants were appropriately fitted with a Hans Rudolph  
188 mask, with an attached pneumotach flowmeter for measurement. Continuous recording of  
189 breath-by-breath gas analysis data was achieved throughout each intervention period.

190

### 191 *Conventional echocardiographic image acquisition*

192 Transthoracic echocardiography was performed pre and immediately post HIIT, following  
193 methodology previously detailed (O'Driscoll et al. 2018). All images were acquired using a  
194 Vivid-q ultrasound system (GE Healthcare, Milwaukee, Wisconsin) with a 1.5-3.6 MHz

195 phased array transducer (M4S-RS Matrix cardiac ultrasound probe). All participants were  
196 measured in the left lateral decubitus position by one consistent sonographer. Cardiac  
197 measurements were recorded in accordance with the current guidelines (Lang et al. 2015) and  
198 stored for offline analysis using commercial software with the results averaged (EchoPAC,  
199 V.113.0.x, GE Healthcare). Images were captured in the parasternal short and long-axis and  
200 apical 2-, 3-, and 4-chamber views. Interventricular septal and posterior wall thickness,  
201 fractional shortening and left ventricle (LV) internal dimensions were measured, and relative  
202 wall thickness was calculated as  $(2 \text{ LV posterior wall thickness})/\text{LV internal diameter}$ . LV  
203 ejection fraction was determined via the modified biplane Simpson's rule. Pulsed-wave  
204 Doppler measures were acquired to assess transmitral early (E) and late (A) diastolic-filling  
205 velocities from the apical 4-chamber view, with the sample volume placed at the tips of the  
206 mitral valve. Isovolumic relaxation time was measured from the start of aortic valve closure  
207 to mitral valve opening. Tissue Doppler imaging was captured at the lateral and septal mitral  
208 annulus to assess peak longitudinal ( $S'$ ), peak early diastolic ( $E'$ ), and peak late diastolic ( $A'$ )  
209 velocities, with values averaged. LV filling pressure was estimated from the mitral  $E/E=$   
210 ratios (Ommen et al. 2000). Total peripheral resistance was calculated through Ohm's law.  
211 Stroke volume was derived from LV end diastolic and LV end systolic volumes, with cardiac  
212 output achieved as the product of heart rate and stroke volume.

213

### 214 ***Myocardial Mechanics***

215 Speckle-tracking imaging was utilised pre and post HIIT to achieve the LV global  
216 longitudinal and time-derivative strain rate from the apical 2-, 3-, and 4-chamber views. The  
217 average value of peak systolic longitudinal strain and peak systolic strain rate from all three  
218 views was calculated as global strain and strain rate. Peak global strain rate during early and

219 late diastole and their ratio as indices of diastolic function was calculated as proposed in  
220 previous work (Wang et al. 2007). The parasternal short axis view from the LV base, level  
221 with the mitral valve (mitral valve leaflets on view) and apex (circular LV cavity with no  
222 papillary muscle visible) was used to acquire the LV radial and circumferential strain and  
223 strain rate, and LV rotation and rotational velocity; again as previously applied (Leitman et  
224 al. 2004; Notomi et al. 2005; van Dalen et al. 2008; Weiner et al. 2010). For effective  
225 speckle-tracking analysis, the highest quality images were used for tracing the endocardium  
226 and a full-thickness myocardial region of interest was selected. All images were reviewed to  
227 validate quality and those that did not achieve the required optimisation and standardization  
228 were excluded. Images were optimized for scan depth and sector width to obtain high frame  
229 rates (>60 Hz) and kept constant throughout each examination. The endocardial trace line  
230 and/or region-of-interest width was readjusted to ensure an adequate tracking score. Raw  
231 frame-by-frame rotation and rotation-rate data was normalized to the percentage duration of  
232 systole and diastole using cubic-spline interpolation to allow for between and within subjects  
233 comparison as basal and apical rotation are not acquired from the same cardiac cycle  
234 (GraphPad Prism 6 Software, La Jolla, CA) (Stembridge et al. 2014). LV twist and untwist  
235 parameters were acquired via subtraction of the basal data from the apical data at each time  
236 point, with LV torsion defined as LV twist per unit length and calculated by dividing the total  
237 twist by LV diastolic length (Stembridge et al. 2014). The sonographer's reproducibility of  
238 speckle-tracking indices has been reported in previous work (O'Driscoll et al. 2017, 2018).

239

#### 240 ***Exercise protocol***

241 The HIIT exercise protocol consisted of a single Wingate session, characterised by three 30-  
242 second periods of maximal intensity cycling. Using a WATT bike pro (Nottingham, England),

243 the exercise periods were loaded with 7.5% of the participants body mass and separated with  
244 2-minutes of unloaded active recovery. Consistent and enthusiastic verbal encouragement was  
245 given during the exercise periods for intensity maintenance. Each participant performed a 2-  
246 minute warm up with no active recovery post-exercise. Cardiac autonomic, haemodynamic and  
247 metabolic parameters were recorded continuously for 5-mins at baseline, during the 3-HIIT  
248 intervals and 5-minutes immediately post HIIT for the recovery period in the supine position.  
249 Cardiac imaging was performed at baseline and immediately following HIIT in the recovery  
250 period.

251

### 252 *Statistical analysis*

253 All continuous variables are presented as mean  $\pm$  standard deviation. Data analysis was  
254 performed using statistical package for social sciences (SPSS 26 release version for Windows;  
255 SPSS Inc., Chicago, IL). A one-way repeated measures ANOVA was performed with a  
256 Bonferroni post-hoc test to identify statistically significant differences. Correlation analyses  
257 was performed to ascertain any associations between BRS and BEI with LF and HF HRV  
258 parameters. Data was reported as statistically significant when  $p < 0.05$ .

259

260

261

262

263

264

265 **Results**

266 All fifty participants successfully completed the single HIIT session with no adverse events  
267 reported.

268

269 *Haemodynamics*

270 Figure 1 presents the haemodynamic responses throughout each stage of the HIIT session.

271 There was a significant increase in sBP from baseline ( $126\pm 13$  mmHg) compared to HIIT 1

272 ( $152\pm 38$ mmHg,  $p<0.001$ ), HIIT 2 ( $154\pm 19$ mmHg,  $p<0.001$ ) and HIIT 3 ( $152\pm 35$ mmHg,

273  $p<0.001$ ), with a significant decrease in recovery post HIIT ( $111\pm 10$ mmHg,  $p<0.001$ ), which

274 was significantly lower than baseline ( $p<0.05$ ). mBP significantly increased from baseline

275 ( $88\pm 8$ mmHg) to HIIT 1 ( $111\pm 36$ mmHg,  $p<0.001$ ), HIIT 2 ( $109\pm 24$ mmHg,  $p<0.05$ ) and HIIT

276 3 ( $108\pm 34$ mmHg,  $p<0.05$ ), and significantly decreased in recovery post HIIT ( $76\pm 8$  mmHg).

277 dBP significantly increased from baseline ( $69\pm 8$ mmHg) to HIIT 1 ( $93\pm 35$ mmHg,  $p<0.001$ ),

278 HIIT 2 ( $89\pm 24.8$ mmHg,  $p<0.05$ ) and HIIT 3 ( $92\pm 30$ mmHg,  $p<0.001$ ), and significantly

279 decreased post exercise in recovery post HIIT ( $59\pm 9$ mmHg,  $p<0.001$ ).

280

281 Heart rate significantly increased from baseline ( $69\pm 10$ b·min<sup>-1</sup>) to HIIT 1 ( $148\pm 17$ b·min<sup>-1</sup>,

282  $p<0.001$ ), HIIT 2 ( $157\pm 16$ b·min<sup>-1</sup>,  $p<0.001$ ), HIIT 3 ( $160\pm 18$ b·min<sup>-1</sup>,  $p<0.001$ ) and

283 significantly decreased in recovery post HIIT ( $100\pm 12$ b·min<sup>-1</sup>,  $p<0.001$ ) when compared to

284 HIIT 3, but remained significantly elevated post HIIT when compared to baseline ( $p<0.001$ ).

285 Stroke volume significantly increased from baseline ( $65.7\pm 11.1$ ml) to HIIT 1 ( $97.6\pm 24.4$ ml,

286  $p<0.001$ ), HIIT 2 ( $102.2\pm 25.8$ ml,  $p<0.001$ ), HIIT 3 ( $102.2\pm 23.3$ ml,  $p<0.001$ ) and recovery post

287 HIIT ( $103.8\pm 32.2$ ml,  $p<0.001$ ). As a result of these responses, cardiac output significant

288 increase from baseline ( $4.49\pm 0.98\text{L}\cdot\text{min}^{-1}$ ) to HIIT 1 ( $14.29\pm 3.52\text{L}\cdot\text{min}^{-1}$ ,  $p<0.001$ ), HIIT 2  
289 ( $15.86\pm 3.48\text{L}\cdot\text{min}^{-1}$ ,  $p<0.001$ ), HIIT 3 ( $16.18\pm 3.57\text{L}\cdot\text{min}^{-1}$ ,  $p<0.001$ ) followed by a significant  
290 decrease post exercise in recovery ( $10.28\pm 3.17\text{L}\cdot\text{min}^{-1}$ ,  $p<0.001$ ) when compared to HIIT 3,  
291 but remained significantly elevated post HIIT when compared to baseline ( $p<0.001$ ).

292

293 Rate pressure product significantly increased from baseline ( $8642\pm 1414$ ) to HIIT 1  
294 ( $22541\pm 6308$ ,  $p<0.001$ ), HIIT 2 ( $24202\pm 4142$ ,  $p<0.001$ ) and HIIT 3 ( $23983\pm 6225$ ,  $p<0.001$ ),  
295 with a significant decrease in recovery post HIIT ( $11054\pm 1798$ ,  $p<0.001$ ). Total peripheral  
296 resistance significantly decreased from baseline ( $1640\pm 365\text{dyne}\cdot\text{s}\cdot\text{cm}^5$ ) to HIIT 1  
297 ( $638\pm 231\text{dyne}\cdot\text{s}\cdot\text{cm}^5$ ,  $p<0.001$ ), HIIT 2 ( $576\pm 158\text{dyne}\cdot\text{s}\cdot\text{cm}^5$ ,  $p<0.001$ ), HIIT 3  
298 ( $586\pm 213\text{dyne}\cdot\text{s}\cdot\text{cm}^5$ ,  $p<0.001$ ) and in recovery post HIIT ( $639\pm 177\text{dyne}\cdot\text{s}\cdot\text{cm}^5$ ,  $p<0.001$ ).

299

### 300 *Cardiac autonomic and metabolic parameters*

301 As presented in Figure 2A, there was a significant decrease in HRV expressed as R-R power  
302 spectral density from baseline ( $3101.7\pm 3571.6\text{m}^2$ ) to HIIT 1 ( $927.2\pm 934.6\text{m}^2$ ,  $p<0.001$ ), HIIT  
303 2 ( $565\pm 1194.9\text{m}^2$ ,  $p<0.001$ ), HIIT 3 ( $381.6\pm 521.7\text{m}^2$ ,  $p<0.001$ ) and in recovery post HIIT  
304 ( $578.1\pm 1317.9\text{m}^2$ ,  $p<0.001$ ). Figure 2B shows a significant decrease in low frequency  
305 (normalized units) from baseline ( $47.7\pm 15.5\%$ ) compared to HIIT 1 ( $38\pm 13.7$ ,  $p<0.05$ ), HIIT  
306 2 ( $35.5\pm 11.3$ ,  $p<0.001$ ) and HIIT 3 ( $32.3\pm 11.5\%$ ,  $p<0.001$ ), with a paradoxical significant  
307 increase in recovery post HIIT ( $62.3\pm 15.5\%$ ), which was significantly greater than baseline  
308 and HIIT 3 (both  $p<0.001$ ). Accordingly, high frequency (normalized units) significantly  
309 increased from baseline ( $52.3\pm 15.5\%$ ) to HIIT 1 ( $62.2\pm 13.2\%$ ,  $p<0.05$ ), HIIT 2 ( $64.5\pm 11.3\%$ ,  
310  $p<0.001$ ) and HIIT 3 ( $67.7\pm 11.5\%$ ,  $p<0.001$ ), with a significant decrease in recovery post HIIT  
311 ( $37.7\pm 15.5\%$ ), which was significantly lower than baseline and HIIT 3 (both  $p<0.001$ ). As a

312 result of these inverse changes, there was no significant change in low frequency/high  
313 frequency (LF/HF) ratio from baseline ( $1\pm 0.59$ ) to HIIT 1 ( $0.9\pm 0.43$ ) and HIIT 2 ( $0.85\pm 0.45$ ),  
314 with a significant decrease from baseline to HIIT 3 ( $0.7\pm 0.48$ ,  $p<0.05$ ). However, there was a  
315 significant increase in recovery post HIIT, which was significantly greater than baseline ( $1.7\pm 1$ ,  
316  $p<0.001$ ) (Figure 2C). The absolute frequency domain responses are shown in Table 1.

317

318 As shown in Figure 2D, there was no significant change in BRS from baseline  
319 ( $9.2\pm 11\text{ms}\cdot\text{mmHg}^{-1}$ ) compared to HIIT 1 ( $7.1\pm 7.4\text{ms}\cdot\text{mmHg}^{-1}$ ), HIIT 2 ( $9\pm 11.3\text{ms}\cdot\text{mmHg}^{-1}$ )  
320 and HIIT 3 ( $6.7\pm 9.3\text{ms}\cdot\text{mmHg}^{-1}$ ). However, there was a significant increase in recovery post  
321 HIIT ( $24.8\pm 16.7\text{ms}\cdot\text{mmHg}^{-1}$ ) from HIIT 3, which was significantly greater than baseline (both  
322  $p<0.001$ ). Figure 2D also shows no significant difference in BEI from baseline ( $41.8\pm 28$ ) to  
323 HIIT 1 ( $41\pm 22.2$ ), but a significant decrease from baseline to HIIT 2 ( $24.3\pm 23.5$ ,  $p<0.05$ ) and  
324 HIIT 3 ( $16.2\pm 17.3$ ,  $p<0.001$ ); followed by a significant increase post exercise in recovery  
325 ( $68.8\pm 16.2$ ) from HIIT 3, which was also significantly greater than baseline (both  $p<0.001$ ).

326

327 Correlation analyses demonstrated a significant association between BRS and LF ( $r = 0.7$ ;  
328  $p<0.001$ ) and BRS and HF ( $r = 0.66$ ;  $p<0.001$ ), during HIIT 1; BRS and LF ( $r = 0.86$ ;  $p<0.001$ )  
329 and BRS and HF ( $r = 0.93$ ;  $p<0.001$ ) during HIIT 2, and BEI and LF ( $r = 0.5$ ;  $p=0.004$ ) and  
330 BEI and HF ( $r = 0.59$ ;  $p=0.001$ ) during HIIT 3. In recovery, there was a significant correlation  
331 between the LF/HF ratio and BRS ( $r = 0.4$ ;  $p=0.014$ ).

332

333 As illustrated in Table 1, aerobic capacity ( $V\dot{V}O_2$ ), carbon dioxide production ( $V\dot{V}CO_2$ ) and  
334 breathing frequency ( $L\cdot\text{min}^{-1}$ ) significantly increased from baseline compared to all 3 HIIT

335 stages and recovery post HIIT (all  $p < 0.05$ ). Minute ventilation ( $V_{\square E}$ ) and a-vO<sub>2</sub> difference  
336 ( $\text{mLO}_2 \cdot 100\text{mL}^{-1}$ ) both significantly increased from baseline compared to the 3 HIIT stages (all  
337  $p < 0.001$ ), with a significant decrease from HIIT 3 to recovery post HIIT ( $p < 0.001$ ). Respiratory  
338 exchange ratio (RER) significantly increased from baseline compared to HIIT 1 ( $p < 0.001$ ),  
339 HIIT 2 stages ( $p < 0.001$ ) and recovery ( $p < 0.05$ ), but there was no significant difference between  
340 HIIT 3 and recovery post HIIT ( $p < 0.001$ ).

341

### 342 *Cardiac structure and function*

343 Baseline and post HIIT echocardiographic structural, functional and LV tissue doppler  
344 parameters are presented in Table 2. There was a significant decrease in LV internal diameter  
345 systole ( $p = 0.002$ ) and left ventricular end-diastolic posterior wall thickness ( $p = 0.037$ ).  
346 Separately, there were significant decreases in both Peak E/A ratio ( $p < 0.001$ ), isovolumetric  
347 relaxation time ( $p = 0.032$ ), and a significant increase in Peak A velocity ( $p = 0.001$ ). There were  
348 also several significant changes in global LV systolic function, with significant decreases in  
349 LV end-diastolic volume ( $p = 0.033$ ), LV end-systolic volume ( $p = 0.004$ ), and significant  
350 increases in LV ejection fraction ( $p = 0.002$ ), fractional shortening ( $p = 0.006$ ) and lateral and  
351 septal peak S' (both  $p = 0.001$ ). There were no significant changes in estimated LV filling  
352 pressures from pre to post HIIT.

353

### 354 *Left ventricular mechanics*

355 Pre and Post HIIT myocardial mechanics are displayed in Table 3. Peak global longitudinal  
356 strain ( $p < 0.001$ ), strain rate ( $p = 0.001$ ) and global longitudinal strain rate in early diastole  
357 ( $p = 0.004$ ) significantly increased in recovery immediately following HIIT. There was a



358 significant increase in basal systolic ( $p=0.001$ ) and diastolic ( $p=0.001$ ) rotational velocity, and  
359 significant decreases in basal radial strain ( $p=0.009$ ) and strain rate ( $p<0.001$ ), but no  
360 significant change in basal rotation, circumferential strain or strain rate. Apical rotation  
361 ( $p=0.025$ ) and apical systolic ( $p<0.001$ ) and diastolic ( $p=0.016$ ) rotational velocity all  
362 significantly increased, as well as significant increases in apical circumferential strain  
363 ( $p=0.003$ ) and strain rate ( $p<0.001$ ), but no significant change in apical radial strain or strain  
364 rate. These mechanical changes produced significant increases in all LV twist parameters,  
365 including LV twist ( $p=0.034$ ), systolic twist velocity ( $p=0.001$ ), untwist velocity ( $p=0.001$ ) and  
366 LV torsion ( $p=0.028$ ).

367

368

369

370

371

372

373

374

375

376

377

378

379

## 380 **Discussion**

381

382 As the first study to investigate the combined physiological responses to a single HIIT  
383 session, we found significant improvements in cardiac autonomic modulation and  
384 haemodynamic regulation, as well as improvements in LV systolic and diastolic function and  
385 cardiac mechanics. As illustrated in Figure 3, the physiological responses following HIIT  
386 occur through a complex interplay of numerous mechanistic pathways, some of which are not  
387 conclusively understood.

388

## 389 **Cardiac autonomies**

390 This is the first study to investigate the acute cardiac autonomic, haemodynamic, metabolic  
391 and myocardial responses to a single HIIT session. HIIT induced a significant step wise  
392 reduction in HRV and associated absolute low and high frequency domains. A greater  
393 proportion of the HRV frequency remained in the HF domain, which is supported by the HFnu  
394 response and significant reduction in LF/HF ratio. During recovery post HIIT, all absolute  
395 HRV parameters remained significantly depressed compared to baseline; however, there was  
396 a significant increase in the proportion of HRV within the LF domain, represented by LFnu,  
397 which is supported by the significant increase in LF/HF ratio and indicates a relative  
398 sympathetic predominance in recovery. These responses are similar to those reported following  
399 aerobic exercise (Kaikkonen et al. 2008); however, they are opposite to those previously  
400 reported following isometric exercise (Taylor et al. 2017). Compared to baseline, our results  
401 demonstrate a decline in BRS and significant reduction in BEI during HIIT. This suggests  
402 active resetting of the baroreceptors, which is associated with increasing HR and BP, and is  
403 similar to responses reported during other forms of exercise (Hartwich et al. 2011). However,

404 of mechanistic importance, BRS and BEI significantly increased in recovery immediately post  
405 HIIT, which was significantly greater than baseline. The 2.7- and 1.7-fold increase in BRS and  
406 BEI, respectively, is similar to that reported following alternative short duration exercise  
407 (Taylor et al. 2017), which may be associated with the BP responses seen in the recovery period  
408 following HIIT. However, these results are in contrast to responses following both aerobic and  
409 dynamic resistance training, which commonly produce a post-exercise reduction in  
410 baroreceptor reflex modulation (Somers et al. 1985; Niemelä et al. 2008).

411

412 The cardiac autonomic results are of interest, since the improved BRS and BEI and increased  
413 LF and LF/HF ratio immediately post-HIIT is contradictory, compared to previous research.  
414 Cote et al., (2015) reported similar results with a significant increase in LF/HF post HIIT, but  
415 reported a significant decrease in BRS. Despite methodological differences, such as timing of  
416 post exercise measures (30-mins vs immediately post HIIT), the mechanistic underpinning of  
417 this post-exercise sympathetic dominance accompanied by an increase in baroreflex  
418 functioning is unclear and certainly requires future research. Although is not always the case,  
419 the withdrawal of sympathetic autonomic activity may often occur following such maximal  
420 exercise, which in combination with venous pooling, can result in reduced cerebral blood flow  
421 and consequently induce vasovagal post-exercise syncope. Since our HRV results indicate the  
422 contrary, one mechanistic hypothesis is a sympathetic response induced as a direct preventative  
423 mechanism of this common syncope; as supported through previous work identifying increases  
424 in LF/HF and normalised LF power during orthostasis, especially in young cohorts  
425 homogenous to the present study (Kawaguchi et al.; Sato et al. 2007). Conversely, perhaps such  
426 a response is not a result of complex neural-physiological mechanistic interactions, but rather  
427 reflects methodological complications with the application of HRV indices. Specifically,  
428 research from Goldstein et al., (Goldstein et al. 2011) suggested that the LF parameter of HRV

429 provides an index of baroreflex function rather than sympathetic tone based on various lines of  
430 evidence (Goldstein et al. 2011). As an example, LF power has often been shown not to  
431 increase during exercise (as exhibited in our findings), despite evident increases in cardiac and  
432 extracardiac sympathetic outflows (Warren et al. 1997; Goldstein et al. 2011). Furthermore,  
433 patients following bilateral thoracic sympathectomies have normal baroreflex function and LF  
434 power, despite partial cardiac sympathetic denervation (Moak et al. 2005). Since this  
435 hypothesis appears to align well with our findings, perhaps the HRV results are actually  
436 representing the changes in baroreflex function as opposed to sympathetic tone. Our correlation  
437 analysis supports this concept.

438

### 439 **Haemodynamics**

440 Compared to baseline, HIIT induced a significant increase in sBP, mBP and dBP, which  
441 remained relatively stable over each interval. During post exercise recovery, there was a  
442 significant decrease in sBP, which was significantly lower than baseline. This is similar to  
443 previously reported acute evidence (Cote et al. 2015), while generally aligning with the training  
444 effects typically observed (O'Driscoll et al. 2018). Since cardiac output remained elevated  
445 post-HIIT, this reduction can be directly attributed to changes in peripheral vascular resistance,  
446 as supported by the significant reductions in TPR, which remained in the recovery period. HIIT  
447 has been linked to the promotion of greater sheer stress-induced nitric oxide bioavailability  
448 through an increased flow mediated dilation response compared to lower intensity modalities  
449 (Ramírez-Vélez et al. 2019). This increase in endothelial derived-nitric oxide may act on  
450 vascular smooth muscle cells to induce vasodilation through increasing cyclic guanosine  
451 monophosphate production via the activation of soluble guanylate cyclase; thus explaining the  
452 reduced TPR and hypotension (MacInnis and Gibala 2017). In addition, the arterial baroreflex

453 is a fundamental regulator of short and long-term BP with compelling evidence for its role in  
454 post exercise hypotension.

455

## 456 **Myocardial responses**

457 Our results show significant acute cardiac responses to HIIT with improved LV function and  
458 cardiac mechanics. Specifically, we found significant improvements in peak global LV  
459 longitudinal strain and strain rate, which were not observed following a 2-week HIIT  
460 intervention (O'Driscoll et al. 2018). Global longitudinal strain and strain rate, have been  
461 proposed as strong indicators of measuring myocardial function; thus, the results from the  
462 present study may provide important clinical implications (Karlsen et al. 2019). Additionally,  
463 we found significant reductions in LV end-diastolic posterior wall thickness and end-systolic  
464 internal diameter. These parameters independently provide implications regarding structural  
465 health and clinical outcomes; and thus, although these changes are not always observed in  
466 chronic interventions, these acute responses may be of clinical importance (Quiñones et al.  
467 2000; O'Driscoll et al. 2018).

468

469 A single HIIT session elicited significant improvements in LV twist, systolic twist velocity,  
470 untwist velocity and torsion. In addition to providing prognostic implications, increased LV  
471 twist enhances potential energy during the ejection phase with recoil of this systolic  
472 deformation and release of elastic energy contributing to pressure decay, enhancing LV  
473 diastolic suction and thus filling (Sengupta et al. 2008; O'Driscoll et al. 2017). Despite this  
474 increase in diastolic function, LV end-diastolic volume (EDV) decreased post HIIT, potentially  
475 as a consequence of the sustained elevation in heart rate and a pooling-induced decrease in  
476 venous return. This post HIIT reduction in EDV combined with the increased stroke volume

477 resulted in a greater ejection fraction. It may be postulated that increases in stroke volume and  
478 ejection fraction post HIIT are attributed to the LV mechanical and functional improvements,  
479 as supported through the enhancements of contractility parameters such as end-systolic internal  
480 diameter and fractional shortening. These observed LV mechanical changes may be explained  
481 via the same mechanistic pathway responsible for decreased peripheral vascular resistance,  
482 which induced post HIIT systolic hypotension, resulting in a decreased afterload and thus  
483 improved LV systolic function. This mechanistic explanation is supported through the  
484 significant increases in systolic tissue doppler parameters and the non-significant decreases in  
485 LV filling pressures post HIIT; as well as being endorsed in the chronic HIIT literature  
486 (O'Driscoll et al. 2018).

487

#### 488 **Metabolic responses**

489 Interest in HIIT interventions has been predominantly based upon its ability to produce  
490 significant improvements in aerobic capacity, comparable to that observed following  
491 traditional moderate-intensity continuous training (MICT), despite being an anaerobic  
492 modality in nature (Milanović et al. 2015; MacInnis and Gibala 2017). While the acute results  
493 of the present study support this anaerobic predominance, there also appears to be some aerobic  
494 contribution to HIIT, particularly in the final interval, with a respiratory exchange ratio (RER)  
495 below the threshold of 1, predominantly facilitated by an increase in oxygen uptake. This  
496 transfer in primary energy metabolism towards the later stages of the HIIT session highlights  
497 the potential to manipulate acute programme variables (such as exercise bout duration) of this  
498 modality to favour either aerobic or anaerobic metabolic pathways and may be an important  
499 mechanism for improvements in aerobic capacity (MacInnis and Gibala 2017). This response  
500 however, may reflect anaerobic endurance and/or fatigue.

501 **Limitations**

502 Our study investigated healthy and young participants and therefore may have limited  
503 application to ageing and clinical populations, suggesting the need for future research using  
504 participants from specific demographics. The primary limitation of this study lies within the  
505 application of HRV measurement in this setting. Indeed, the short duration of recording and  
506 changes in respiration induced via acute maximal exercise may affect HRV recordings and is  
507 a limitation regarding interpretation. However, given the novelty of this study, we considered  
508 cardiac autonomic measurements integral to provide a comprehensive non-invasive assessment  
509 of the combined physiological responses to HIIT. Further, these results should be interpreted  
510 in the context of the short-duration HIIT protocol employed, and thus the relative applicability  
511 of these findings to differing HIIT protocols of longer durations is unknown. Finally, cycle  
512 wattage was not recorded during HIIT and as such, we are unable to report on power output at  
513 each stage of HIIT.

514

515 **Conclusion**

516 A single HIIT session is associated with significant improvements in cardiac autonomic  
517 modulation and haemodynamic regulation, as well as improvements in LV systolic and  
518 diastolic function, mechanics and cardiac remodelling. In general, the acute responses detailed  
519 support the established chronic adaptations following a programme of HIIT, which may have  
520 independent clinical implications.

521

522

523

524 **Declarations**

525

526 **Funding:** None

527

528 **Acknowledgments:** We thank the participants who volunteered to participate in the study.

529 We also thank Cameron Stevens for his assistance in constructing the central figure.

530

531 **Conflict of Interest:** There are no conflicts of interest.

532

533 **Ethics Approval:** This research was approved by the Canterbury Christ Church University

534 Ethics Committee and conformed to the Declaration of Helsinki principles (Ref:

535 17/SAS/47F).

536

537 **Data Availability:** The sharing of data in an open-access repository was not included in our

538 participants consent. Thus, in accordance with standard ethical practice, data may only be

539 available on request from the corresponding author.

540

541

542

543

544

545

546



547 **References**

- 548 Akselrod S, Gordon D, Ubel FA, et al (1981) Power spectrum analysis of heart rate  
549 fluctuation: A quantitative probe of beat-to-beat cardiovascular control. *Science* (80- )  
550 213:220–222. <https://doi.org/10.1126/science.6166045>
- 551 Batacan RB, Duncan MJ, Dalbo VJ, et al (2017) Effects of high-intensity interval training on  
552 cardiometabolic health: A systematic review and meta-analysis of intervention studies.  
553 *Br. J. Sports Med.* 51:494–503
- 554 Cao M, Quan M, Zhuang J (2019) Effect of high-intensity interval training versus moderate-  
555 intensity continuous training on cardiorespiratory fitness in children and adolescents: A  
556 meta-analysis. *Int. J. Environ. Res. Public Health* 16
- 557 Cote AT, Bredin SSD, Phillips AA, et al (2015) Greater autonomic modulation during post-  
558 exercise hypotension following high-intensity interval exercise in endurance-trained  
559 men and women. *Eur J Appl Physiol* 115:81–89. [https://doi.org/10.1007/s00421-014-](https://doi.org/10.1007/s00421-014-2996-5)  
560 [2996-5](https://doi.org/10.1007/s00421-014-2996-5)
- 561 Ekelund U, Steene-Johannessen J, Brown WJ, et al (2016) Does physical activity attenuate,  
562 or even eliminate, the detrimental association of sitting time with mortality? A  
563 harmonised meta-analysis of data from more than 1 million men and women. *Lancet*  
564 388:1302–1310. [https://doi.org/10.1016/S0140-6736\(16\)30370-1](https://doi.org/10.1016/S0140-6736(16)30370-1)
- 565 Goldstein DS, Benth O, Park MY, Sharabi Y (2011) Low-frequency power of heart rate  
566 variability is not a measure of cardiac sympathetic tone but may be a measure of  
567 modulation of cardiac autonomic outflows by baroreflexes. *Exp. Physiol.* 96:1255–1261
- 568 Guthold R, Stevens GA, Riley LM, Bull FC (2018) Worldwide trends in insufficient physical  
569 activity from 2001 to 2016: a pooled analysis of 358 population-based surveys with 1·9

570 million participants. *Lancet Glob Heal* 6:e1077–e1086. <https://doi.org/10.1016/S2214->  
571 109X(18)30357-7

572 Hartwich D, Dear WE, Waterfall JL, Fisher JP (2011) Effect of muscle metaboreflex  
573 activation on spontaneous cardiac baroreflex sensitivity during exercise in humans. *J*  
574 *Physiol* 589:6157–6171. <https://doi.org/10.1113/jphysiol.2011.219964>

575 Herazo-Beltrán Y, Pinillos Y, Vidarte J, et al (2017) Predictors of perceived barriers to  
576 physical activity in the general adult population: a cross-sectional study. *Brazilian J*  
577 *Phys Ther* 21:44–50. <https://doi.org/10.1016/j.bjpt.2016.04.003>

578 Kaikkonen P, Rusko H, Martinmäki K (2008) Post-exercise heart rate variability of  
579 endurance athletes after different high-intensity exercise interventions. *Scand J Med Sci*  
580 *Sport* 18:511–519. <https://doi.org/10.1111/j.1600-0838.2007.00728.x>

581 Karlsen S, Dahlslett T, Grenne B, et al (2019) Global longitudinal strain is a more  
582 reproducible measure of left ventricular function than ejection fraction regardless of  
583 echocardiographic training. *Cardiovasc Ultrasound* 17:18.  
584 <https://doi.org/10.1186/s12947-019-0168-9>

585 Kawaguchi T, Uyama O, ... MK-TJ of, 2001 undefined Orthostatic hypotension in elderly  
586 persons during passive standing: a comparison with young persons. *academic.oup.com*

587 Lang RM, Badano LP, Victor MA, et al (2015) Recommendations for cardiac chamber  
588 quantification by echocardiography in adults: An update from the American Society of  
589 Echocardiography and the European Association of Cardiovascular Imaging. *J Am Soc*  
590 *Echocardiogr* 28:1-39.e14. <https://doi.org/10.1016/j.echo.2014.10.003>

591 Leitman M, Lysyansky P, Sidenko S, et al (2004) Two-dimensional strain-A novel software  
592 for real-time quantitative echocardiographic assessment of myocardial function. *J Am*

593 Soc Echocardiogr 17:1021–1029. <https://doi.org/10.1016/j.echo.2004.06.019>

594 Li C, Zheng C, Tai C (1995) Detection of ECG Characteristic Points Using Wavelet  
595 Transforms. *IEEE Trans Biomed Eng* 42:21–28. <https://doi.org/10.1109/10.362922>

596 MacInnis MJ, Gibala MJ (2017) Physiological adaptations to interval training and the role of  
597 exercise intensity. *J. Physiol.* 595:2915–2930

598 Milanović Z, Sporiš G, Weston M (2015) Effectiveness of High-Intensity Interval Training  
599 (HIT) and Continuous Endurance Training for VO<sub>2</sub>max Improvements: A Systematic  
600 Review and Meta-Analysis of Controlled Trials. *Sport. Med.* 45:1469–1481

601 Moak JP, Eldadah B, Holmes C, et al (2005) Partial cardiac sympathetic denervation after  
602 bilateral thoracic sympathectomy in humans. *Heart Rhythm* 2:602–609.  
603 <https://doi.org/10.1016/j.hrthm.2005.03.003>

604 Niemelä TH, Kiviniemi AM, Hautala AJ, et al (2008) Recovery pattern of baroreflex  
605 sensitivity after exercise. *Med Sci Sports Exerc* 40:864–870.  
606 <https://doi.org/10.1249/MSS.0b013e3181666f08>

607 Notomi Y, Lysyansky P, Setser RM, et al (2005) Measurement of ventricular torsion by two-  
608 dimensional ultrasound speckle tracking imaging. *J Am Coll Cardiol* 45:2034–2041.  
609 <https://doi.org/10.1016/j.jacc.2005.02.082>

610 O’Driscoll JM, Taylor KA, Wiles JD, et al (2017) Acute cardiac functional and mechanical  
611 responses to isometric exercise in prehypertensive males. *Physiol Rep* 5:.  
612 <https://doi.org/10.14814/phy2.13236>

613 O’Driscoll JM, Wright SM, Taylor KA, et al (2018) Cardiac autonomic and left ventricular  
614 mechanics following high intensity interval training: A randomized crossover controlled  
615 study. *J Appl Physiol* 125:1030–1040. <https://doi.org/10.1152/jappphysiol.00056.2018>

616 Oliveira BRR, Santos TM, Kilpatrick M, et al (2018) Affective and enjoyment responses in  
617 high intensity interval training and continuous training: A systematic review and meta-  
618 analysis. PLoS One 13:e0197124. <https://doi.org/10.1371/journal.pone.0197124>

619 Ommen SR, Nishimura RA, Appleton CP, et al (2000) Clinical Utility of Doppler  
620 Echocardiography and Tissue Doppler Imaging in the Estimation of Left Ventricular  
621 Filling Pressures. *Circulation* 102:1788–1794.  
622 <https://doi.org/10.1161/01.CIR.102.15.1788>

623 Pan J, Tompkins WJ (1985) A Real-Time QRS Detection Algorithm. *IEEE Trans Biomed*  
624 *Eng BME-32:230–236*. <https://doi.org/10.1109/TBME.1985.325532>

625 Quiñones MA, Greenberg BH, Kopelen HA, et al (2000) Echocardiographic predictors of  
626 clinical outcome in patients with left ventricular dysfunction enrolled in the SOLVD  
627 registry and trials: Significance of left ventricular hypertrophy. *J Am Coll Cardiol*  
628 35:1237–1244. [https://doi.org/10.1016/S0735-1097\(00\)00511-8](https://doi.org/10.1016/S0735-1097(00)00511-8)

629 Ramírez-Vélez R, Hernández-Quiñones PA, Tordecilla-Sanders A, et al (2019) Effectiveness  
630 of HIIT compared to moderate continuous training in improving vascular parameters in  
631 inactive adults. *Lipids Health Dis* 18:42. <https://doi.org/10.1186/s12944-019-0981-z>

632 Roy M, Williams SM, Brown RC, et al (2018) High-Intensity Interval Training in the Real  
633 World: Outcomes from a 12-Month Intervention in Overweight Adults. *Med Sci Sports*  
634 *Exerc* 50:1818–1826. <https://doi.org/10.1249/MSS.0000000000001642>

635 Sato Y, Ichihashi K, Kikuchi Y, et al (2007) Autonomic function in adolescents with  
636 orthostatic dysregulation measured by heart rate variability. *Hypertens Res* 30:601–605.  
637 <https://doi.org/10.1291/hypres.30.601>

638 Sengupta PP, Tajik AJ, Chandrasekaran K, Khandheria BK (2008) Twist Mechanics of the

639 Left Ventricle. Principles and Application. *JACC Cardiovasc. Imaging* 1:366–376

640 Somers VK, Conway J, LeWinter M, Sleight P (1985) The role of baroreflex sensitivity in  
641 post-exercise hypotension. *J Hypertens* 3:S129-30

642 Stembridge M, Ainslie PN, Hughes MG, et al (2014) Ventricular structure, function, and  
643 mechanics at high altitude: Chronic remodeling in Sherpa vs. short-term lowlander  
644 adaptation. *J Appl Physiol* 117:334–343.  
645 <https://doi.org/10.1152/jappphysiol.00233.2014>

646 Taylor KA, Wiles JD, Coleman DD, et al (2017) Continuous cardiac autonomic and  
647 hemodynamic responses to isometric exercise. *Med Sci Sports Exerc* 49:1511–1519.  
648 <https://doi.org/10.1249/MSS.0000000000001271>

649 Troiano RP, Berrigan D, Dodd KW, et al (2008) Physical activity in the United States  
650 measured by accelerometer. *Med Sci Sports Exerc* 40:181–188.  
651 <https://doi.org/10.1249/mss.0b013e31815a51b3>

652 van Dalen BM, Vletter WB, Soliman OII, et al (2008) Importance of Transducer Position in  
653 the Assessment of Apical Rotation by Speckle Tracking Echocardiography. *J Am Soc*  
654 *Echocardiogr* 21:895–898. <https://doi.org/10.1016/j.echo.2008.02.001>

655 Wang J, Khoury DS, Thohan V, et al (2007) Global diastolic strain rate for the assessment of  
656 left ventricular relaxation and filling pressures. *Circulation* 115:1376–1383.  
657 <https://doi.org/10.1161/CIRCULATIONAHA.106.662882>

658 Warren JH, Jaffe RS, Wraa CE, Stebbins CL (1997) Effect of autonomic blockade on power  
659 spectrum of heart rate variability during exercise. *Am J Physiol - Regul Integr Comp*  
660 *Physiol* 273:. <https://doi.org/10.1152/ajpregu.1997.273.2.r495>

661 Weiner RB, Hutter AM, Wang F, et al (2010) The impact of endurance exercise training on

662 left ventricular torsion. *JACC Cardiovasc Imaging* 3:1001–1009.

663 <https://doi.org/10.1016/j.jcmg.2010.08.003>

664 Whelton PK, Carey RM, Aronow WS, et al (2018) 2017

665 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA Guideline for

666 the Prevention, Detection, Evaluation, and Management of High Blood Pressure in

667 Adults: Executive Summary: A Report of the American College of

668 Cardiology/American Heart Association Task F. In: *Journal of the American Society of*

669 Hypertension. Elsevier, pp 579.e1-579.e73

670 World Health Organization (2015) *Physical Activity: Global recommendations on physical*

671 *activity for health Consequences of physical inactivity*. WHO Reg Off Eur.

672 <https://doi.org/ISBN 978 92 4 159 997 9>

673 World Health Organization (2010) *Global recommendations on physical activity for health*

674

675

676

677

678

679

680

681

682

683

684

685

686 **Figure legends**

687

688 Figure 1: Hemodynamic responses to high intensity interval training. Values are presented as  
689 mean±SEM. A) systolic, mean and diastolic blood pressure responses. B) heart rate and rate  
690 pressure product responses. C) total peripheral resistance response. D) stroke volume and  
691 cardiac output responses. \*p<0.05, \*\*p<0.001 between baseline and all stages. §§p<0.001  
692 between HIIT 3 and recovery.

693

694 Figure 2: Autonomic responses to high intensity interval training. Values are presented as  
695 mean±SEM. A) R-R power spectral density (heart rate variability) response. B) R-R  
696 normalized units low-frequency and high-frequency responses. C) R-R LF:HF ratio response.  
697 D) baroreceptor reflex sensitivity and baroreceptor effectiveness index response \*p<0.05,  
698 \*\*p<0.001 between baseline and all stages. §§p<0.001 between HIIT 3 and recovery.

699

700 Figure 3: Central illustration of the acute mechanistic responses to HIIT.