Moderate Recovery Unnecessary to Sustain High Stroke Volume during Interval Training. A Brief Report

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Abstract

It has been suggested that the time spent at a high stroke volume (SV) is important for improving maximal cardiac function. The aim of this study was to examine the effect of recovery intensity on cardiovascular parameters during a typical high-intensity interval training (HIIT) session in fourteen well-trained cyclists. Oxygen consumption (VO₂), heart rate (HR), SV, cardiac output (Qc), and oxygenation of vastus lateralis (TSI) were measured during a HIIT (3×3-min work period, 2 min of recovery) session on two occasions. VO₂, HR and Qc were largely higher during moderate-intensity (60%) compared with low-intensity (30%) $(VO_2, effect size; ES = +2.6; HR, ES = +2.8; Qc, ES = +2.2)$ and passive (HR, ES = +2.2; Qc, ES = +1.7) recovery. By contrast, there was no clear difference in SV between the three recovery conditions, with the SV during the two active recovery periods not being substantially different than during exercise (60%, ES = -0.1; 30%, ES = -0.2). To conclude, moderateintensity recovery may not be required to maintain a high SV during HIIT.

Key words: High-intensity interval training; cardiac output; cardiac function; arteriovenous oxygen difference.

Introduction

High-intensity interval training (HIIT) is recognized as one of the most effective means of improving cardiorespiratory and metabolic function in athletes (Buchheit and Laursen, 2013). A typical HIIT session involves the repetition of periods of high-intensity exercise (i.e., the intervals) interspersed with periods of lower intensity (i.e., recovery periods) (Buchheit and Laursen, 2013). Long intervals (i.e., >3 min) are typically completed close to the speed/power associated with maximal oxygen consumption (VO_{2max}), because it is believed that this is optimal stimulus for eliciting maximal cardiovascular and peripheral adaptations (Buchheit and Laursen, 2013). However, because VO_2 and cardiac output (Qc) can be dissociated during intense exercise (Lepretre et al., 2004), and attaining and maintaining an elevated stroke volume (SV) is likely important for improving maximal cardiac function (Cooper, 1997), increasing time spent at maximal Qc (Qcmax) and/or training at an intensity associated with maximal SV may also be important (Lepretre et al., 2004).

The intensity of exercise that maximizes the time at maximal SV is difficult to predict (González-Alonso, 2008; Mortensen et al., 2005;Warburton and Gledhill,

2008). The most appropriate HIIT format inducing increased time at Qcmax in well-trained and elite athletes remains to be determined. In untrained males, compared with peak exercise during a graded exercise test, 30-s allout sprints (typical of sprint interval training sessions (Buchheit and Laursen, 2013)), might allow attainment of similar Qc (effect size; ES = -0.1) and even largely higher SV (ES = +1.3) (Fontana et al., 2011). The recovery period is another key factor of HIIT with respect to cardiopulmonary responses (Buchheit and Laursen, 2013). Conjecture remains as to whether maximal SV is reached during the recovery period or during work periods-and whether this response is recovery-intensity dependent (Buchheit and Laursen, 2013; Cumming, 1972; González-Alonso, 2008; Warburton and Gledhill, 2008). Therefore, the aim of the current study was to examine the effect of recovery intensity on cardiovascular parameters during HIIT.

Methods

Subjects

Fourteen endurance-trained male cyclists participated in the study (age, 25 ± 4 years; body mass, 69.6 ± 4.9 kg; height, 1.77 ± 0.04 m; VO_{2max} , 66.6 ± 4.2 mL·kg⁻¹·min⁻¹; peak power output, 405 ± 28 W). The experimental procedure was approved by the Human Research Ethics Committee at The University of Queensland.

Experimental overview

The complete methodology has been reported elsewhere (Stanley et al., 2014). Briefly, the cyclists completed an incremental cycling test to determine the power at VO_{2max} (pVO_{2max}) and maximal Qc (Qcmax), and two cycling (Wattbike[®], Wattbike Ltd., UK) HIIT sessions (12-min warm-up, three 3-min work periods (Ex) at 90% pVO_{2max}) 5-7 days apart. The 2-min recovery periods were completed at either 30% pVO_{2max} (30%) or 60% pVO_{2max} (60%) in a randomized order with passive (PAS) recovery always following the final interval. Respiratory gas exchange (ParvoMedics TrueOne[®] 2400, Utah, USA) and HR, SV and Qc (PhysioFlow, Manatec Biomedical, France) (Charloux et al., 2000) were measured continuously. Oxygenation of vastus lateralis (tissue saturation index; TSI) was determined using near-infrared spectroscopy (PortaMon, Artinis Medical Systems BV, The Netherlands). Arteriovenous difference (a-vO₂) was calculated using the Fick equation. All data were sampled at 5-s intervals, and the average for each work period (e.g., 3 min average for Ex, and 2 min average for the recovery period) was determined during the HIIT. Unfortunately due to technical difficulties, Qcmax was only determined for 5 cyclists during the incremental test. The coefficient of variation (CV) for each variable during Ex was as follows: HR, 1.2%; SV, 5.5%; Qc, 5.8%; VO₂, 3.4%; TSI, 9.9%; and a-vO₂ diff, 2.5%.

Data analysis

All data in the text and figures are presented as mean (n = 14) or percentage (n = 5) of incremental test peak values with 90% confidence limits (CL). Standardized differences/changes in all monitored variables were calculated using the pooled between-subject standard deviations. Threshold values were >0.2 (small), >0.6 (moderate) and >1.2 (large) (Hopkins et al., 2009). Uncertainty in each effect was expressed as 90% CL and its probabilities that

the true difference was substantially greater or lower than the CV for each variable. These probabilities were used to make a qualitative mechanistic inference about the true effect (Hopkins et al., 2009). Data as a percentage of incremental test peak values were included to provide an indication of relative load and demonstrated similar trends to raw values. Statistical comparisons were not performed on data as a percentage of incremental test peak values because data was only available for 5 cyclists.

Results

The effect of recovery intensity on cardiorespiratory variables (both mean and percentage of incremental test peak values) is displayed in Figure 1. No clear differences in SV were observed between recovery conditions. There was a possible small difference in SV between PAS and Ex.

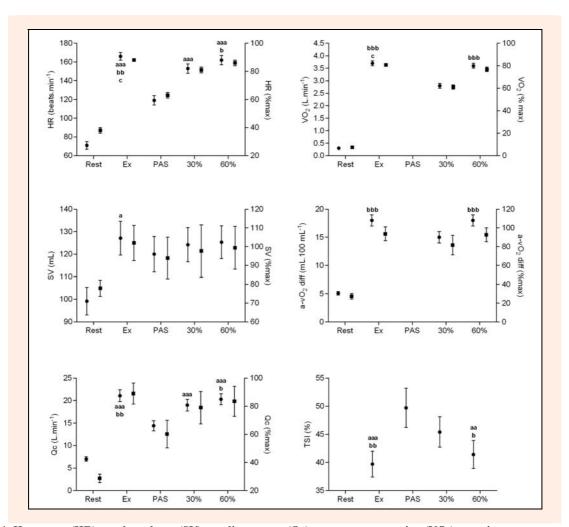


Figure 1. Heart rate (HR), stroke volume (SV), cardiac output (Qc), oxygen consumption (VO₂), arteriovenous oxygen difference (a-vO₂ diff), and tissue saturation index (TSI) measured simultaneously during the different intensities during the HIIT session: pre-exercise rest (Rest), 3-min work periods at 90% pVO_{2max} (Ex), passive recovery (PAS), low-intensity recovery (30%), moderate-intensity recovery (60%). Data are presented as mean (n = 14; circles) and percentage of incremental test peak values (n = 5; squares) with 90% confidence intervals. VO₂ values for Rest were calculated based on (5 mL·kg⁻¹) (Medbø and Tabata, 1989) and were not available for PAS. TSI was not measured during rest or the max test. Letters 'a', 'b', and 'c' indicate a substantial difference versus PAS, 30%, and 60% respectively, with the number of letters representing a small (1 letter), moderate (2 letters), and large (3 letters) standardized differences overlapped small positive and negative values, the magnitude was deemed unclear; otherwise that magnitude was deemed to be the observed magnitude (Hopkins et al., 2009). For clarity, letters have been omitted for differences between Rest and all other intensities as all were clear and large. Data as a percentage of incremental test peaks were provided to indicate the relative load and demonstrated similar trends to raw values, however, because data was only available for 5 cyclists, statistical comparisons were not performed.

Discussion

This is the first study to describe Qc and SV during a typical HIIT session performed by well-trained athletes. The main finding was that moderate-intensity recovery periods are not necessary to maintain high SV during the exercise intervals of HIIT.

In the present study, during the 3-min work periods at 90% pVO_{2max}, Qc reached ~89% and SV reached ~102% of the peak values attained during the incremental test. Due to the limited number of data sets in the present study (n = 5), caution is advised when comparing these findings. Nevertheless, the Qc values in the present study are consistent with the ~85% observed during a 4-min exercise interval at an intensity ~90% pVO_{2max} (Richard et al., 2004). However, present data contrast with that observed during a sprint interval session (30-s all-out sprints), where Oc reached ~102%, and SV ~134% of Qcmax and maximal SV respectively (Fontana et al., 2011). Nevertheless, HIIT is likely a better stimuli for cardiovascular improvements as the total time spent at or near maximal SV and VO_{2max} is likely substantially greater during a HIIT session (e.g., 15-min HIIT: 3 × 3-min at 90% pVO_{2max} interspersed with 2 min active recovery = >9 min near max) compared with a SIT session of comparable duration (e.g., 15-min SIT: 6×30 -s all-out efforts interspersed with 2 min passive recovery (Buchheit and Laursen, 2013) = ~ 3 min near max). These data illustrate that cardiovascular responses during exercise are likely HIIT protocol-dependent.

During recovery periods we observed that SV did not surpass the levels attained during exercise (Figure 1). Because the statistical analysis included all 14 cyclists, we are confident with this finding. This is contrary to the belief that SV could reach maximal levels during the recovery periods of HIIT (Buchheit and Laursen, 2013; Cumming, 1972). In a well-trained cyclist, higher SV values were consistently observed during recovery periods, not work periods, irrespective of the type of exercise (incremental exercise, long intervals, or sprints) (Buchheit and Laursen, 2013). The reason for these disparities is not clear. However, differences in training status (well-trained vs. untrained) and mode of exercise (upright vs. supine) likely influenced venous return, cardiac preload, and peripheral resistance that can influence SV.

Interestingly, we observed that despite the expected differences in peripheral metabolic demands (as inferred from the greater a-vO₂ diff and lower TSI, figure 1), the SV response was not affected by recovery intensity (Figure 1). Our data suggest that SV already reached a plateau at a low intensity (González-Alonso, 2008; Warburton and Gledhill, 2008). Because active recovery involves rhythmical contraction of exercising muscles irrespective of intensity, it is likely that the effect of the muscle pump influenced peripheral resistance and venous return, such that a similarly high SV (compared with the exercise intervals) was maintained.

Conclusion

The present study demonstrates that moderate-intensity

recovery periods are not necessary to maintain high SV during the exercise intervals HIIT. We acknowledge that the impedance method used to assess cardiovascular function in this study has not been validated during maximal exercise. Therefore, rather than compare our data in absolute terms (to previous studies), we have made comparisons with respect to difference in exercise intensity. Nevertheless, the CV for our data suggest similar reliability to that observed during sub-maximal exercise (Charloux et al., 2000). The practical implication of this finding is that reducing the intensity of the recovery period during a HIIT protocol may prolong the time to exhaustion (Dupont et al., 2003), potentially allowing completion of additional high-intensity intervals, which can, in turn, increase the time accumulated at Qcmax. Using the HIIT protocol in the current study as an example, completion of an additional two intervals would increase the time spent at Qcmax by 67%. Whether achieving a specific quantity of time at Qcmax is necessary to maximize cardiac adaptation remains unknown.

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References

- Buchheit, M. and Laursen, P.B. (2013) High-intensity interval training, solutions to the programming puzzle part i: Cardiopulmonary emphasis. *Sports Medicine* 43, 313-338.
- Charloux, A., Lonsdorfer-Wolf, E., Richard, R., Lampert, E., Oswald-Mammosser, M., Mettauer, B., Geny, B. and Lonsdorfer, J. (2000) A new impedance cardiograph device for the noninvasive evaluation of cardiac output at rest and during exercise: Comparison with the "direct" fick method. *European Journal of Applied Physiology* 82, 313-320.
- Cooper, G. (1997) Basic determinants of myocardial hypertrophy: A review of molecular mechanisms. *Annual Review of Medicine* 48, 13-23.
- Cumming, G.R. (1972) Stroke volume during recovery from supine bicycle exercise. *Journal of Applied Physiology* 32, 575-578.
- Dupont, G., Blondel, N. and Berthoin, S. (2003) Performance for short intermittent runs: Active recovery vs. Passive recovery. *European Journal of Applied Physiology* 89, 548-554.
- Fontana, P., Betschon, K., Boutellier, U. & Toigo, M. (2011). Cardiac output but not stroke volume is similar in a wingate and vo_{2peak} test in young men. *European Journal of Applied Physiology*, 111, 155-158.
- González-Alonso, J. (2008) Point:Counterpoint: Stroke volume does/does not decline during exercise at maximal effort in healthy individuals. *Journal of Applied Physiology* 104, 275-276.
- Hopkins, W.G., Marshall, S.W., Batterham, A.M. and Hanin, J. (2009) Progressive statistics for studies in sports medicine and exercise science. *Medicine and Science in Sports and Exercise* **41**, 3-13.
- Lepretre, P. M., Koralsztein, J. P. & Billat, V. L. (2004). Effect of exercise intensity on relationship between VO_{2max} and cardiac output. *Medicine and Science in Sports and Exercise* 36, 1357-1363.
- Medbø, J. and Tabata, I. (1989) Relative importance of aerobic and anaerobic energy release during short-lasting exhausting bicycle exercise. *Journal of Applied Physiology* 67, 1881-1886.
- Mortensen, S.P., Dawson, E.A., Yoshiga, C.C., Dalsgaard, M.K., Damsgaard, R., Secher, N.H. and Gonzalez-Alonso, J. (2005). Limitations to systemic and locomotor limb muscle oxygen delivery and uptake during maximal exercise in humans. *Journal of Physiology-London* 566, 273-285.
- Richard, R., Lonsdorfer-Wolf, E., Dufour, S., Doutreleau, S., Oswald-

Mammosser, M., Billat, V. and Lonsdorfer, J. (2004) Cardiac output and oxygen release during very high-intensity exercise performed until exhaustion. *European Journal of Applied Physiology* **93**, 9-18.

- Stanley, J., Peake, J., Coombes, J. and Buchheit, M. (2014) Central and peripheral adjustments during high-intensity exercise following cold water immersion. *European Journal of Applied Physiology* 114(1), 147-163.
- Warburton, D.E.R. and Gledhill, N. (2008) Counterpoint: Stroke volume does not decline during exercise at maximal effort in healthy individuals. *Journal of Applied Physiology* **104**, 276-278.

Key points

- Moderate-intensity recovery periods may not be necessary to maintain high stroke volume during the exercise intervals of HIIT.
- Stroke volume did not surpass the levels attained during the exercise intervals during the recovery periods of HIIT.
- The practical implication of these finding is that reducing the intensity of the recovery period during a HIIT protocol may prolong the time to exhaustion, potentially allowing completion of additional highintensity intervals increasing the time accumulated at maximal cardiac output.

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