TRACKING CHANGES IN THE UPPER BOUNDARY OF THE HEAVY-INTENSITY EXERCISE DOMAIN: END-TEST POWER VERSUS RESPIRATORY COMPENSATION POINT

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Abstract:

The aim of this study was to compare how respiratory compensation point (RCP) and end-test power (EP) change in response to the same four-week high intensity interval training (HIIT). The power output associated with RCP and EP before and after HIIT were recorded in 24 recreationally active participants (14 men and 10 women). RCP was determined from an incremental exercise test and EP was derived from a three-minute maximal effort test on a cycle ergometer. A significant time (pretest/posttest) × measurement (EP/RCP) interaction was found ($F_{(1,23)}=5.119$, p<.05). Results from a paired-sample *t*-test indicated that both EP ($t_{(23)}=-5.221$, p<.05) and RCP ($t_{(23)}=-3.049$, p<.05) increased significantly from pretest to posttest. Furthermore, a small effect size (d=.36, 90%CI=[.13, .58]) was calculated for the pre/posttest changes in the examined thresholds indicating greater potential improvements in EP compared to RCP. The pre/posttest change in EP (mean=21 W, 90%CI=[14, 28 W]) exceeded its standard error of estimate (14 W), while RCP did not. Correlation analysis revealed that EP correlated with RCP at both pretest (r=.813, p<.05) and posttest (r=.873, p<.05), however, delta values between the two measures were not significantly related. Both EP and RCP can be used to assess the change of aerobic capacity after HIIT, but may be reflective of different physiological adaptations. Further, EP may be preferred over RCP when assessing the effects of HIIT.

Key words: training, testing, physiology, fatigue

Introduction

Critical power (CP) is estimated from the examination of the hyperbolic relationship between certain levels of power output and the corresponding time that the power output can be sustained (Moritani, Nagata, Devries, & Muro, 1981; Poole, Ward, Gardner, & Whipp, 1988; Vanhatalo, Doust, & Burnley, 2008). A three-minute all-out cycling test (3MT) was recently developed (Burnley, Doust, & Vanhatalo, 2006; Vanhatalo, Doust, & Burnley, 2007), which yielded a steady power output towards the end of the test (end-test power; EP) that has been demonstrated to be an acceptable alternative for the original multi-trial CP test proposed (Monod & Scherrer, 1965; Moritani, et al., 1981). One of the more recent applications of CP/EP is to demarcate different exercise intensity domains (Jones, Wilkerson, DiMenna, Fulford, & Poole, 2008; McClave, LeBlanc, & Hawkins, 2011; Moritani, et al., 1981; Poole & Gaesser, 1985; Poole, et al., 1988).

Many studies have shown that EP is greater than the power outputs associated with the ventilatory threshold (VT) and gas exchange threshold (GET) (Burnley & Jones, 2007; Francis, Quinn, Amann, & LaRoche, 2010; McClave, et al., 2011; Vanhatalo, et al., 2007). The exercise intensity domain model is an exercise prescription model proposed by Whipp, Ward, and Rossiter (2005), which partitions intensity into ranges of power outputs that elicit common pulmonary oxygen uptake (VO₂) response characteristics. During exercise at the lower boundary of the heavy exercise intensity domain, VO₂ increases and lactate production exceeds the rate of lactate removal (Gaesser & Poole, 1996). The lower boundary of the severe exercise intensity domain is the highest work intensity at which blood lactate and VO₂ ultimately reach steady-state phases (Gaesser & Poole, 1996). Within the severe exercise intensity domain, VO₂ and lactate fail to stabilize, and VO₂ is driven to the maximal level (Cooper & Storer, 2001; Gaesser & Poole, 1996). Poole et al. (1988) demonstrated that exercising at CP induced a stable VO₂, while exercising at 5% above CP resulted in a different metabolic response and the achievement of maximal VO₂ (VO₂peak). Jones et al. (2008) found a significant difference in muscle metabolic response between exercises performed just below and just above CP, and suggested that intensities below CP allow for relative metabolic homeostasis and can be sustained without the development of fatigue.

Another method of distinguishing between these exercise intensities is by the assessment of respiratory compensation point (RCP) during an incremental exercise test (Beaver, Wasserman, & Whipp, 1986). The RCP is a result of metabolic acidosis, which could be distinguished by an increased minute ventilation relative to CO₂ production (Whipp, 1994). Beaver et al. (1986) reported that RCP was consistently higher than VT, while others found that the power output at RCP during incremental exercise and CP determined from the results of several exhaustive constant-power tests were not different (Dekerle, Baron, Dupont, Vanvelcenaher, & Pelayo, 2003). In agreement, Bergstrom et al. (2013) reported that both RCP and EP occurred at power outputs that were approximately 80% of VO₂peak. Recently, Keir et al. (2015) reported similar mean values for the VO₂ associated with CP and RCP, suggesting that CP and RCP may be physiologically equivalent and interchangeable. The correlation analysis results from the previous studies have been equivocal, with the r values varying from .07 to .91 (Beaver, et al., 1986; Bergstrom, et al., 2013; Broxterman, et al., 2015). As suggested by Cross and Sabapathy (2012), existing research evidence is insufficient to conclude that CP and RCP are equivalent parameters.

Previous research has demonstrated EP and CP to respond in a similar manner following interval training and to represent similar exercise intensities (Vanhatalo, et al., 2008). However, potentially as a result of different mechanisms when compared to GET and VT (Bergstrom, et al., 2013). Furthermore, Pettitt, Jamnick, and Clark (2012) have demonstrated that critical speed (the running analog of CP) was not different from the mean value of speed at GET and VO₂peak. Broxterman et al. (2015) found that the mean values of critical speed and RCP were not different, but the high degree of variability between them impeded the precise estimation of one from the other parameter. Their findings also suggest different mechanisms for each parameter. Keir et al. (2015) pointed out that VO₂peak was associated with an intrinsic measurement error between 2.5-5%, and the unit change in VO₂ was 100 times smaller than that of power output. Thus the relatively small changes in VO₂ may be interpreted as large changes in power output, which may help to explain the disagreement between CP and RCP.

Since a mechanistic link between EP or CP and RCP has not been demonstrated, their equivalence is still inconclusive, so an examination of how these two parameters respond to training is needed. No previous studies, however, have introduced a training intervention to potentially associate or dissociate the adaptations in EP and RCP. The purpose of this study was to compare how EP and RCP change in response to the same four-week high intensity interval training (HIIT) program. HIIT involves repeated bouts of high-intensity exercise intermixed with recovery. HIIT was used primarily due to the reported metabolic response improvements, including CP (Thomas, Reading, & Shephard, 1992), EP (Vanhatalo, et al., 2008), VT (Poole & Gaesser, 1985), RCP (Robinson IV, et al., 2014), and VO₂peak (Helgerud, et al., 2007).

Methods

Experimental design

Each participant visited the laboratory on two separate occasions (occurring on nonconsecutive days) before and one week after the four-week training period. Anthropometric measures were collected during the first visit. Each participant then performed an incremental exercise test on a cycle ergometer to determine peak power output (PPO), GET, and RCP. PPO achieved during this test was defined as the power output of the last two-minute stage completed by the participants, and was used to establish individual training intensities during the subsequent training program. During the second visit, a 3MT was performed to assess EP. The study was approved by the Institutional Review Board.

Participants

Thirty-one recreationally active individuals (18 men and 13 women), between the ages of 18 and 35 years, were recruited by word of mouth and flyers. There was a minimum requirement of VO₂peak for the participants (35 ml·kg⁻¹·min⁻¹ for men and 30 ml·kg⁻¹·min⁻¹ for women). Twenty-four (14 men and 10 women) participants (age: 23.29±3.16 years; body height: 173.20±10.12 cm; body mass: 74.27±13.54 kg; BMI: 24.59±2.84) completed the training protocol and their results were included in the final analysis. The rest of seven participants served as the control group, who just completed performance testing at the same time points as the other participants, and their data was used to calculate the testretest reliability. Before obtaining written informed consent, testing procedures were fully explained to each participant. The Physical Activity Readiness Questionnaire (PAR-Q) was used to exclude participants who were not able to perform physical exercise (Thomas, et al., 1992). To ensure the optimal performance of the test, we asked the participants to refrain from any strenuous physical activity for 48 hours prior to testing. In addition, we instructed participants to arrive at each testing session two hours fasted.

Estimation of VO₂peak, GET, and RCP

Participants performed an incremental test till volitional exhaustion on a cycle ergometer (Lode Excalibur Sport; Groningen, The Netherlands) to determine PPO, GET, and RCP. A heart rate monitor (Polar Electro Inc., Kempele, Finland) was used to record the participants' heart rate. We asked the participants to maintain a pedaling rate of 70-75 revolutions per minute (RPM) at an initial work rate of 75 W. The work rate increased 25 W every two minutes until the participant was unable to keep the cadence above 70 RPM for about 10 s despite verbal encouragement, as described by Bergstrom et al. (2013). During the incremental test, VO_2 , carbon dioxide production (VCO₂), ventilation $(V_{\rm F})$, and respiratory exchange ratio (RER) were monitored using a metabolic cart (TrueOne 2400[®]), Parvo Medics, Inc., Sandy, UT) and expressed as 30-second averages described by Day, Rossiter, Coats, Skasick, and Whipp (2003).

The GET was estimated from the VCO₂-versus-VO₂ relationship as described by Beaver et al. (1986), and defined as the VO₂ value corresponding to the breakpoint at which the VCO₂ value has a non-linear increase. The test-retest reliability for GET had an intraclass correlation coefficient (ICC)=.97 and standard error of estimate (SEM)=0.1 ml·kg·min⁻¹. RCP was defined as the VO₂ value corresponding to the point of departure from linearity of the V_E -versus- VCO₂ relationship, which was determined using the method described by Beaver et al. (1986). The test-retest reliability for RCP was ICC=.87 (SEM=0.2 ml·kg·min⁻¹). The power output at RCP was determined by the regression equation derived from the plot between the power output values from the incremental test and VO₂ values. The test-retest reliability for power output at RCP was ICC=.85 (SEM=11 W).

Three-minute maximal effort cycling test

Following the procedures of Vanhatalo et al. (2007), EP was determined on an electronicallybraked cycle ergometer (Lode Excalibur Sport; Groningen, The Netherlands). After a standardized warm-up, the participant completed one minute of unloaded cycling at 90 RPM prior to an all-out three-minute effort with resistance and corresponding work rate being determined by a function of pedaling rate. Participants were asked to accelerate to about 110 RPM over the last five seconds of the unloaded period. The resistance was adjusted automatically and continuously during the all-out effort using the pedaling rate dependent linear mode on the cycle ergometer that uses a linear factor described previously (Burnley & Jones, 2007). To minimize pacing and maximize RPM as long as possible, the participant was not made aware of the elapsed time and strong verbal encouragement was

provided. EP was the mean power output during the final 30 seconds of the test. The test-retest reliability for EP was ICC=.81 (SEM=14 W).

Training protocol

Participants participated in a four-week HIIT protocol with three sessions per week on a cycle ergometer (Lode Corival 400, Groningen, the Netherlands). A constant work rate, independent of the RPM, was designated for each training session. The participants were offered at least one day of rest between training sessions. The exercise training program (as shown in Figure 1) contained alternating training sessions around supra-maximal and sub-maximal work rates as a percentage of the PPO from the incremental exercise test (Robinson IV, et al., 2014). After a five-minute warm-up at a self-selected work rate, participants started an exercise protocol of five two-minute exercise bouts with one minute of complete rest between each bout. If participants were unable to complete the entire twominute exercise bout, they were asked to complete the one-minute rest period and attempt subsequent bouts. Training logs showed that the completion rate was 82.64±9.55 %.



Figure 1. The intensity schedule utilized during the 4-week high-intensity interval training program.

Statistical analyses

Data are presented as M±SD. A two-way [time (pretest, posttest) × measurement (EP, RCP)] repeated measures analysis of variance (ANOVA) with *post-hoc* testing was performed for participants in the training group. An alpha level of .05 was used to determine statistical significance for the ANOVA and paired-samples *t*-tests comparisons. Bland-Altman plots were used to assess the limits of agreement between EP and RCP and one sample *t*-tests were used to determine if the average difference between values were significantly different from zero. In order to compare the pre/posttest changes in EP and RCP, the effect size and corresponding 90% confidential interval (CI) was analyzed using a custom spreadsheet (Hopkins, 2007) and interpreted as follows: trivial (<0.2), small (0.2-0.6), moderate (0.6-1.2), and large (1.2-2.0) (Batterham & Hopkins, 2006). If the CI crossed thresholds for both substantially positive (≥ 0.2) and negative (\leq -0.2) effects, differences were considered unclear. Bland-Altman analysis was used to assess the agreement (determined by one-sample *t*-test) between the pretest and posttest values of EP and RCP, as well as the post-pre (delta) changes for each of these measures. Bivariate correlation analysis was used to assess the relationship between the pretest and posttest values of EP and RCP, as well as the post-pre (delta) changes for each of these measures. The correlation coefficient (r) of this relationship was categorized as low (.5-.7), moderate (.7-.8), or high (.9-1.0) according to Vincent and Weir (2012).

Results

Comparison of EP and RCP

There was a statistically significant time × measurement interaction ($F_{(1, 23)}=5.119$, p<.05). Results from paired-sample *t*-tests indicated that there was no difference in the overall group for EP (178±51 W) vs. RCP (180±37 W) at pretest

 $(t_{(23)}=-.406, p=.688)$ and EP (199±51 W) vs. RCP (191±38 W) at posttest ($t_{(23)}=1.648, p=.113$), however, both EP ($t_{(23)}=-5.221, p<.05$) and RCP ($t_{(23)}=-3.049, p<.05$) increased significantly from pretest to posttest. A small effect size (d=.36, 90% CI=[.13, .58]) was calculated for the pre/posttest changes in the examined thresholds indicating greater potential improvements in EP compared to RCP. Percentage change values from pretest to posttest for both EP and RCP is shown in Figure 2.

Relationship between EP and RCP

Figure 3 shows Bland-Altman plots depicting the agreement between pretest EP and RCP (upper left panel), between posttest EP and RCP (upper right panel), and between delta EP and delta RCP (lower left panel). The mean bias between pretest EP and RCP (p=.688), as well as the bias between posttest EP and RCP (p=.113) was not different from zero. The mean bias between delta EP and delta RCP (p<.01) was greater than zero. Correlation analysis (as shown in Figure 4) revealed that EP correlated with RCP in both pretest (r=.813, p<.001) and posttest (r=.873, p<.001). However, no significant relationship was found between delta (r=.154, p=.472) values for RCP and EP.



Figure 2. Percentage change values from pretest to posttest for end-test power (EP) and respiratory compensation point (RCP).



Figure 3. Bland-Altman plots between pretest EP and RCP (upper left panel), between posttest EP and RCP (upper right panel), and between delta EP and delta RCP (lower left panel).



Figure 4. Correlation analysis with 95% confidential intervals between end-test power (EP) and respiratory compensation point (RCP) for pretest values (upper left panel), posttest values (upper right panel), and delta (post – pre) changes (lower left panel) after high intensity interval training. Dashed lines represent line of identity.

Discussion and conclusions

While improvements in EP/CP and RCP following HIIT interventions have been reported in separate investigations (Broxterman, et al., 2015; Helgerud, et al., 2007; Kendall, et al., 2009; Robinson IV, et al., 2014; Smith, et al., 2009), this is the first study to investigate the potential of a HIIT intervention to associate or dissociate adaptations in EP and RCP. The major finding of this study was that both EP and the power output associated with RCP increased significantly after a four-week HIIT program, with EP improving to a greater degree than RCP. The larger change in EP from the 3MT suggests, supported by a small effect size, that it may be a more discriminative measure than RCP when assessing the effects of HIIT. Furthermore, the pre/posttest change in EP (mean=21 W, 90%CI=[14 W, 28 W]) exceeded its SEM (14W), while that in RCP (mean=11 W, 90%CI=[5 W, 17 W]) did not fully exceeded its SEM (11 W). The improvements in EP and RCP were likely due to the training intensities employed (from 80% to 120% of PPO at VO₂peak) being at or above the intensity domains associated with these measures. The HIIT-induced physiological changes are likely due to improved metabolic efficiency through more efficient skeletal muscle substrate utilization (Laursen, Shing, Peake, Coombes, & Jenkins, 2005), and enhanced respiratory control resulted from increased mitochondrial density (Jacobs, et al., 2013).

Recently, Bergstrom et al. (2013) reported no significant differences between EP (187±47 W) and the power output associated with the RCP (190±49 W). In agreement, the current study demonstrated EP and RCP were correlated before and following the HIIT program. Furthermore, several studies have suggested that EP and RCP demarcate the heavy and severe exercise intensity domains (Burnley, et al., 2006; Francis, et al., 2010; McClave, et al., 2011). Various factors associated with exercise-induced acidosis have been presented as mechanisms for EP, RCP, or both thresholds (Bergstrom, et al., 2013; Jones, et al., 2008; Meyer, Faude, Scharhag, Urhausen, & Kindermann, 2004).

However, the lack of differences between EP and RCP should not be interpreted as an equivalence because there was no correlation between the change in EP and RCP (as shown in Figure 4). The Bland-Altman plots for delta EP and delta RCP also showed poor agreement, indicating the dissociation of these two measurements after the HIIT intervention. Some researchers suggested that the discrepancy in changes of CP and RCP could be due to differential responses to the training stimuli of the participants or the difficulties in determining the hyperventilation point from the gas exchange data (Broxterman, et al., 2015). As previously stated, EP and RCP may be indicators of different physiological mechanisms. EP/CP depend upon the hyperbolic relationship between power output and time to exhaustion, thus the fatigue at or above EP/ CP is likely based on the interaction of the anaerobic capacity, VO₂max and VO₂ slow component (Burnley & Jones, 2007), and may be related to the availability of high-energy phosphate and crossbridge dysfunction resulting from the accumulation of metabolites (Jones, et al., 2008). That being said, EP/CP is involved with both accumulation and depletion processes (Skiba, Chidnok, Vanhatalo, & Jones, 2012), while RCP relies primarily on the accumulation of metabolic by-products signaled by exercise induced lactic acidosis and hyperventilation (Meyer, et al., 2004). The reported association between EP/CP and RCP may be the result of these two parameters occurring at approximately 80% VO₂max (Bergstrom, et al., 2013; Broxterman, et al., 2015). Broxterman et al. (2015) posited that the significant relationship between CP and RCP might be coincidental and the current findings support this notion. Thus, individual variations in aerobic fitness, as well as altered buffering capacity and chemoreceptor sensitivity in response to HIIT, may have resulted in the dissociation between changes in EP and RCP.

Furthermore, the different exercise protocols to determine EP vs. RCP may have also contributed to this high degree of variability in the change scores (Robinson, IV, et al., 2014). The use of a standardized incremental protocol may have resulted in inappropriate increment in work rates for some individuals, leading to the potential for error in the RCP and EP measurements. Keir et al. (2015) used both metabolic (VO_2) and performance (power output) measurements to examine the relationship between CP and RCP. Different from the current study, they found that the difference only existed in performance measurements (RCP: 262±48 W; CP: 226 ± 45 W). So it is possible that the dissociation between metabolic and performance measurements may affect the interpretation of the data. A more recent study demonstrated that differences in work rate increment during a graded exercise test affect the determination of power output associated with RCP (Leo, Sabapathy, Simmonds, & Cross, 2017). Future studies should focus on the underlying mechanisms behind the differential change of EP/CP and RCP, and if possible, examining CP with multiple constant power trials.

Some limitations should be noted for the current study. Future studies with larger sample sizes should examine the gender effect on the changes in EP and RCP after training interventions due to potentially unique metabolic adaptations. In addition, the influence of the graded exercise test protocol cannot be discounted on fatigue thresholds and the use of ramp as opposed to stage procedures may allow for a more precise estimation of the power output associated with RCP.

Even though the equivalence between CP/EP and RCP is still debatable, previous studies (Beaver, et al., 1986; Bergstrom, et al., 2013; Poole, et al., 1988) have suggested that both CP/EP and RCP could be used to distinguish exercise intensities. Separately CP/EP and RCP have been reported to improve following a short-term interval training; however, a direct comparison of the training adaptations in these fatigue thresholds had not been conducted (Robinson IV, et al., 2014; Vanhatalo, et al., 2008). This study provides information regarding how CP/EP and RCP change in response to HIIT. The estimation of CP/EP requires an additional performance test to be completed and uses data generated from a graded exercise test, which is also used to determine RCP. Thus, some investigators attempt to estimate the upper-limit of the heavy-intensity domain by measuring RCP alone; however, according to the findings of the present study, this approach may not be valid. In summary, while the current findings suggest that after a period of HIIT, both EP and RCP can be used to assess the change of aerobic power. The lack of agreement in the delta change between EP and RCP after HIIT intervention limits the interchangeable nature of these thresholds. The differential training responses between EP and RCP, with RCP being less affected than EP, potentially reflect different mechanisms of fatigue or physiological adaptations.

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