THE PHYSIOLOGICAL MECHANISM BEHIND THE TALK TEST

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Abstract:

The Talk Test (TT) is a very simple marker of exercise intensity, which has been shown to be a useful surrogate of the ventilatory (VT) and respiratory compensation (RCT) thresholds. The purpose of this study was to evaluate a potential mechanism behind the TT. Healthy, college-aged subjects (n=20) performed a maximal and two sub-maximal cycle ergometer tests. The two submaximal tests were performed: with the Talk Test (EXP) and without speaking (the control trial – CON). Oxygen uptake (VO_2), CO_2 output (VCO_2), minute ventilation (V_E), breathing frequency (BF), end-tidal CO_2 pressure ($P_{ET}CO_2$) and TT times were recorded. VO_2, VCO_2 and V_E were reduced during the TT and increased immediately after it. BF was reduced during the TT. $P_{ET}CO_2$ values (a surrogate of P_aCO_2) were highest during the TT and lowest before the TT. The time to complete the TT increased across progressive stages. This study supports the hypothesis that talking causes CO_2 retention, which may cause ventilatory drive to increase. Since the ventilatory drive is already high above the VT, the apparent CO_2 retention associated with speech may cause talking to become uncomfortable.

Key words: exercise prescription, exercise test, metabolic thresholds, respiratory gas exchange, speech

Introduction

Over 35 years ago it became evident that 'the relative percent concept' for exercise prescription had certain deficiencies (Katch, Weltman, Sady, & Freedson, 1978; Scharhag-Rosenberger, Meyer, Gassler, Faude, & Kindermann, 2010). Further, when using the relative percent concept as a basis for exercise prescription, a maximal exercise test is required (Pescatello, 2014). In both preventative and rehabilitation populations this is inconvenient or even impossible, meaning that the most strongly evidence-based method of prescribing exercise intensity is, in a practical sense, less than ideally useable. Further, using maximal test derived variables during exercise training is not simple because it usually requires direct monitoring of HR during training sessions.

A recent consensus document has suggested that concepts based on metabolic thresholds are preferable for exercise prescription (Mezzani, et al., 2012). However, this presents the same practical limitation as the relative percent concept, in that direct measurement of metabolic thresholds is costly and technically demanding. Given the prac-

tical limitations on evaluation-based methods for exercise prescription, subjective methods of guiding exercise training intensity have become popular. In recent years the Rating of Perceived Exertion (RPE) (Borg, 1998; Eston, 2012) and the Talk Test (TT) have been studied extensively. Both RPE (Parfitt, Evans, & Eston, 2012) and the TT have been shown to be valid and reliable methods for exercise intensity prescription (Foster, et al., 2008; Woltmann, et al., 2015). The TT, which may present a surrogate of both ventilatory (VT) and respiratory compensation (RCT) thresholds (Dehart-Beverley, Foster, Porcari, Fater, & Mikat, 2000; Foster, et al., 2008; Recalde, et al., 2002), has the advantage of fitting into a threshold-based scheme of exercise prescription (Mezzani, et al., 2012).

The concept of the TT originated in 1939, when mountaineers were told not to climb at a pace faster than that at which they were able to speak comfortably (Goode, 2008). During incremental exercise the last positive (LP) stage of the TT (the last stage where the subject is definitely able to speak comfortably) is typically observed at intensities just below the VT. The equivocal (EQ)

stage (the stage when the subject is first uncertain if he/she can speak comfortably) is typically very close to the VT. The first negative (N) stage of the TT (the stage when the subject is first definitely not able to speak comfortably) typically matches the RCT (Dehart-Beverly, et al., 2000; Recalde, et al., 2002; Rodriguez-Marroyo, Villa, Garcia-Lopez, & Foster, 2013). However, other than the observation that stages of the TT correspond with metabolic thresholds, it is not clear why the TT behaves in such a convenient manner.

Breathing has multiple purposes, the most important being respiratory gas exchange. Breathing is also a necessary step in the production of sound (e.g. speech). Speech requires a decrease in ventilation (V_E), particularly because breathing frequency (BF) must be suppressed to allow speaking using continuous, fluent sentences. When speaking, the expiratory duration must be increased since speech is produced only during expiration. The need for longer expiration while speaking during exercise causes a conflict because BF normally increases during exercise, particularly at about the intensity of the ventilatory threshold (Wasserman, et al., 2011).

The physiological mechanism behind the TT is thought to center on the conflict between the need to suppress BF to allow speech and the need to increase BF because of an increased ventilatory drive above the VT (Brawner, et al., 2006). Goode (2008) suggests that epinephrine, norepinephrine and H^+ all increase beyond the 'anaerobic threshold', leading to an increase in the drive to breathe including an increase in BF. Although the increase in V_E and BF at the VT and RCT is likely driven by neutrally-based feed-forward mechanisms rather than by buffering of blood borne metabolites, the net result is that the mismatch of normal breathing mechanics during speech results in decreases in speech comfort. This change in speech comfort conceivably accounts for the practical utility of the EQ and N stages of the TT as surrogates of VT and RCT, respectively.

The suppression of ventilation, because of a decrease in BF during speech, can lead to the reduction in both VO_2 and VCO_2, secondary to a decrease in V_E. While ATP needs can be met briefly using non-VO_2 dependent sources, there is virtually no alternative way to deal with reductions in VCO_2, except for increasing arterial CO_2 partial pressure (P_aCO_2). However, an increase in P_aCO_2 typically causes a dramatic increase in the drive to breathe and the sensation of breathlessness (Wasserman, et al., 2011). Indeed, it is normal for P_aCO_2 to decrease above the intensity associated with the RCT, presumably as a strategy to control the drive to breathe in the face of metabolic acidosis. This suggests that the need to suppress total ventilation and breathing frequency to allow speech might lead to an increase in P_aCO_2 which would increase the

drive to breathe, and be interpreted as a decrease in the comfort of producing speech as a plausible mechanism behind the utilitarian value of the TT. The purpose of this study was to test the hypothesis that an increase in P_aCO_2 during speaking explains the decrease in speech comfort.

If speech causes a decrease in BF and V_E, and a decrease in VO_2 and VCO_2, we expect an increase in P_aCO_2 during speech. However, while P_aCO_2 is inconvenient to measure routinely, end-tidal CO_2 pressure ($P_{ET}CO_2$) is well accepted as a surrogate of P_aCO_2. Respiratory measurement of $P_{ET}CO_2$ during speech can provide insight into the behavior of P_aCO_2. Our hypothesis was that speaking contributes to retention of CO_2, which increases P_aCO_2 and the ventilatory drive. When the ventilatory drive becomes too high, suppression of BF for speech becomes more difficult and speaking is no longer perceived as comfortable, leading to discrimination between LP, EQ and N TT stages as surrogates of VT and RCT.

Methods

Participants

The participants in this study were 20 healthy students. Characteristics of the participants are presented in Table 1. None of the participants were active in competitive sports, although all were recreationally active. Written informed consent was obtained from the participants included in the study, based on the principles articulated in the Declaration of Helsinki. All participants filled out the Physical Activity Readiness Questionnaire (PAR-Q) to rule out contraindications for exercise. The local human subjects committee approved the protocol.

Table 1. Descriptive characteristics of the participants (mean±SD)

	Men (n=8)	Women (n=11)
Age (years)	22.0±1.9	23.2±1.2
Height (cm)	177.9±3.8	169.4±6.8
Body mass (kg)	78.9±10.6	65.5±9.5
VO_2max (ml·min^{-1})	2752±562	2447±576
VO_2max (ml·kg^{-1}·min^{-1})	35.2±7.0	37.5±7.4
VO_2 @ VT (ml/min^{-1})	1310±276	1106±268

Protocol

First, the participants performed a graded exercise test on an electronically braked cycle ergometer (Lode Excalibur, Groningen, The Netherlands) to obtain VO_2max. During this test the TT was performed. This test was used for estimating the power output (PO) at different TT stages for the subsequent two tests. After warming up for 3

min at a PO of 25W, the PO was increased by 25W every 3 min until participant's exhaustion. During the last 45 s of every stage the subject was asked to recite a standard 101-word paragraph (Dehart-Beverly, et al., 2000) from a cue card. After reciting the passage the subject was asked: "Can you speak comfortably?" The possible answers were 'yes', 'yes, but…' or 'no'. The last stage at which the participant said 'yes' was denoted as the last positive (LP) stage of the TT. The first stage where the subject gave the answer 'yes, but…' was denoted as the equivocal (EQ) stage and the first stage the participant was definitely not able to speak comfortably was denoted as the negative (N) stage. After the N stage of the TT the subject was not asked to recite the passage, but continued cycling until volitional cessation.

Tests 2 and 3 were performed in a counterbalanced order. The tests started at the PO two stages below the LP stage (LP-2) from the VO_2max test. Every 3 minutes the PO was increased by 25W until the PO of the N stage was completed. During the experimental (EXP) test, at 2:15 min into each stage the participant was asked to recite the TT, the same (101-word passage) used during the VO_2max test. During the control test (CON – test 3) subjects were asked to breathe normally.

During all tests respiratory gas exchange was recorded breath-by-breath using the Oxycon Mobile (CareFusion Corp., San Diego, CA). A facemask was used, so the subjects could speak during the experiments. The respiratory variables recorded were oxygen uptake (VO_2), carbon dioxide output (VCO_2), pulmonary ventilation (V_E), breathing frequency (BF) and end-tidal CO_2 pressure ($P_{ET}CO_2$). During the EXP trial the time required for the 101-word paragraph was measured. $P_{ET}CO_2$ was taken as a surrogate for pulmonary capillary P_aCO_2 (Wasserman, et al., 2011). Between the tests there was at least one rest day. The overall activity pattern of the subjects did not change during their participation in the study.

Data analysis

Before starting the analysis, the data was visually inspected. The patterns of BF and $P_{ET}CO_2$ in a trial with TT are shown in Figure 1. The speech passages are easy to recognize by a sharp decrease in BF and a sharp increase in the $P_{ET}CO_2$ during the TT. Because this was the case, we selected data guided by the recorded time when the TT was started and ended.

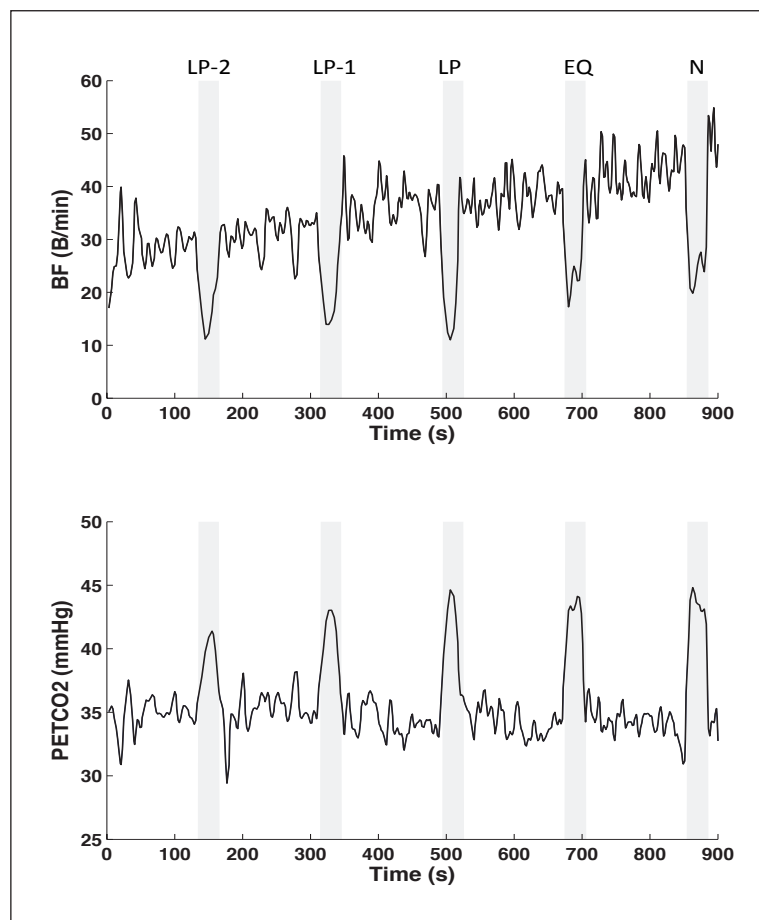All data was converted from breath-by-breath values to values per second using Matlab (Math-

Figure 1. Pattern of BF and PETCO2 during an experimental trial of a representative subject. The recitation of the standard 101-word paragraph of the TT at LP-2, LP-1, LP, EQ and N stage took place at the gray areas.
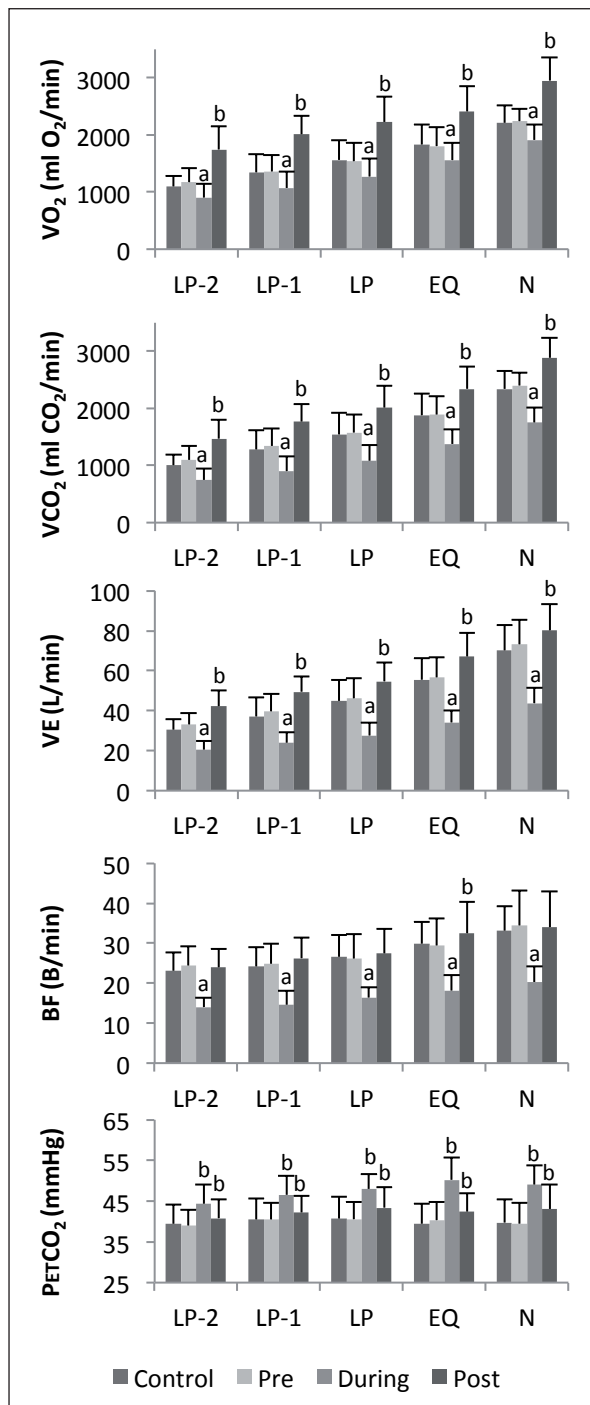
Figure 2. VO2, VCO2, VE, BF and PETCO2 at the LP-2, LP-1, LP, EQ and N stages during the control trial and at pre TT, during TT and post TT during the experimental trial. Statistical significant smaller responses compared to pre TT values are indicated by 'a' and statistical significant larger responses compared to pre TT values are indicated by 'b'.

works, Matlab R2013a). The last minute of each stage was analyzed. Average values were calculated for the last 15 seconds before the TT, the time during the TT and the first 15 seconds after the TT. Because the timing of the standard 101-word paragraph of the TT was different in each subject and every stage, the averages for the timing of the TT could not always be taken for exactly 15 seconds.

In this case the averages of the available number of seconds within that minute were taken. Also, for the CON trial the averages of the last minute in each stage were calculated.

For each variable, differences between measurements (Control, Pre, During, Post) at each stage of the TT (LP-2, LP-1, LP, EQ, N) were compared using repeated measures analysis of variance (ANOVA). When justified by the ANOVA results, the differences between specific means were identified using Tukey's *post-hoc* procedures. Data were analyzed using SPSS (ver. 22) and a significance level of $p \leq .05$ was used for all analyses.

Results

VO_2, VCO_2, V_E, BF and $P_{ET}CO_2$ values for the CON trial and pre TT, during TT and post TT during the EXP trial for the LP-2, LP-1, LP, EQ, and N stages are presented in Figure 2. The values pre TT were not significantly different from CON values. VO_2, VCO_2 and V_E were significantly decreased during speech and significantly increased immediately after the speech compared to pre TT, which is in line with the hypothesized result. BF was significantly decreased during speech, supporting the hypothesis that speaking causes a reduction in BF. This also shows that disruption of ventilation and respiratory gas exchange occurred only during the TT itself. Due to the reduced BF and V_E during speech, $P_{ET}CO_2$ of the limited breaths during speech was significantly increased. $P_{ET}CO_2$ post TT returned toward normal, but was still significantly higher than the pre TT. Together with the decrease in VCO_2 during TT, these results support the hypothesis that speaking causes retention of CO_2 and likely a higher arterial CO_2.

The TT times for the LP-2, LP-1, LP, EQ, and N stages were 25.3 s (3.6), 25.6 s (3.7), 26.4 s (3.5), 27.4 s (4.9) and 27.7 s (5.8), respectively, showing a non-significant increase in the time required to complete reciting the standard paragraph across stages.

Discussion and conclusions

The TT is a very simple and useful marker of exercise intensity, and has been shown to be closely related to the VT and RCT (Dehart-Beverly, et al., 2000; Recalde, et al., 2002; Rodriguez-Marroyo, et al., 2013), and to be both useful and user-friendly for prescribing exercise in people ranging from cardiac patients to elite athletes (Brawner, et al., 2006; Foster, et al., 2009; Petersen, Maribo, Hjortdal, & Lausten, 2014; Rodriguez-Marroyo, et al., 2013; Voelker, et al., 2001; Zanettini, et al., 2013). However, other than a known suppression of BF during speech, there has been little direct evidence of why the TT works so effectively.

The main purpose of this study was to test the hypothesis that the physiological mechanism

behind the TT was related to suppression of BF, with resulting decreases in V_E and VCO_2, leading to an increase in $P_{ET}CO_2$. The presented results are in accordance with earlier research showing a decrease in V_E and an increase in $P_{ET}CO_2$ during speech (Doust & Patrick, 1981; Meckel, Rotstein, & Inbar, 2002). The results of our study support the concept that the decrease in VCO_2 observed during speaking leads to an increase in P_aCO_2 and thus to an increase in the drive to breathe, which consequently influences speech comfort. In accordance with our expectations, it was found that VO_2, VCO_2, V_E, and BF were decreased during speech, but that $P_{ET}CO_2$ increased in a manner consistent with the idea of CO_2 retention during speech.

$P_{ET}CO_2$ of the limited number of breaths taken during speaking were increased. $P_{ET}CO_2$ values are thought to be a reasonable surrogate for P_aCO_2 values (Wasserman, et al., 2011). Our results show lower VCO_2 and higher $P_{ET}CO_2$ during speech, supporting the idea that speaking causes retention of CO_2, likely contributing to a higher arterial CO_2. This supports the concept that an increase in P_aCO_2 is plausibly responsible for the progressive loss in speech comfort. However, since there was not a progressive increase in $P_{ET}CO_2$ as exercise intensity increased, it seems reasonable to suggest that the increasing BF across stages and the slight increase in speech duration while completing the 101-word passage served to limit the magnitude of increase in $P_{ET}CO_2$ (i.e. P_aCO_2), with a loss in speech comfort as the result. In the present study, we did not observe the decrease in $P_{ET}CO_2$ that often occurs during exercise at intensities above the RCT. However, as the N stage of the TT is thought to occur at approximately the RCT, we likely stopped the test before the normal decrease in $P_{ET}CO_2$ had a chance to occur.

During speech $P_{ET}CO_2$ was shown to be significantly higher than before speaking. Since $P_{ET}CO_2$ is thought to be a surrogate measure of P_aCO_2, the data suggest that CO_2 was retained during speech. This study supports the hypothesis that talking causes CO_2 retention which causes the ventilatory drive to increase. Above the VT the ventilatory drive is already high and the increase in P_aCO_2 causes speech to become uncomfortable. This appears to be a reasonable candidate for the mechanism behind the Talk Test.

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Conflict of Interest

The authors declare that they have no conflict of interest.