Assessment of the Effects of Controlled Frequency Breathing on Lactate Levels in Swimming

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Abstract

Objective: The purpose of this study was to assess differences in performance and physiological variables after a 100-yard freestyle swim in free-breathing and controlled-frequency breathing conditions.

Methods: A randomly assigned maximal effort 100-yrd swim test was conducted twice, once with normal breathing (NB) pattern (1 breath every 2-3 strokes), and the second using controlled-frequency breathing (CFB) pattern (1 breath every 7 strokes) in 21 trained female swimmers (19.0 ± 1.1 years). Post-swim blood lactate levels, heart rate, and time to completion were measured and assessed after completion of each bout. Blood samples were taken from the earlobe at rest (prior to the bout), 0-min, 1.5-min, 3-min, and 5-min post-swim. Heart rate was taken at rest and at the same time points as blood lactate.

Results: There was no difference in blood lactate at 0-min (NB 4.34 ± 0.62 mmol/L; CFB 4.56 ± 0.34 mmol/L), 1.5-min (NB 7.24 ± 0.56 mmol/L; CFB 6.84 ± 0.63 mmol/L; CFB 6.04 ± 0.52 mmol/L), and 5-min (NB 6.10 ± 0.61 mmol/L; CFB 4.7 ± 0.51 mmol/L) post-swim. Post-exercise heart rate was significantly higher (p = 0.02) in NB trial (184.9 ± 12.0 bpm) than the CFB trial (174.8 ± 14.8 bpm). The NB 100-yard swim averaged 60.23 ± 0.53 s, and the CFB trial average 61.36 ± 0.62 s (p>0.05).

Conclusions: The results suggest that CFB has no significant immediate effect on blood lactate levels after high intensity swimming races. The significant difference in heart rate between the CFB and NB swims could be explained by the decrease in ventilation produced by the CFB swim causing the heart rate to slow even during exercise.

Keywords

Blood lactate; Hypoxia; Ventilation; Hypercapnia

Abbreviations: NB: Normal Breathing; CFB: Controlled-Frequency Breathing; VO2: Oxygen consumption; BLA: Blood Lactate; V′E: Ventilation; PAO2: Alveolar partial pressure of oxygen

Introduction

The ability to tolerate high levels of lactate during anaerobic metabolism to accommodate high intensity exercise is of extreme importance in the sport of swimming where oxygen consumption (VO2) is limited [1]. Biomechanically, the athlete is in the most hydrodynamic position when the head is submersed, making it necessary for athletes to efficiently breathe during a race, ideally as few times as possible [2]. In addition to efficient breathing an elevated VO2(max), carbon dioxide tolerance, and high blood lactate (BLA) threshold are positively correlated to performance in elite swimmers [3,4]. While research has been completed to determine the effects of controlled frequency breathing (CFB) in training, the influence such training actually has on lactate threshold is still debated, with some studies indicating an increase in BLA and others indicating no change [1,3,5-8]. It has also been demonstrated that CFB does not necessarily restrict oxygen extraction due to ventilation (V′E) adaptations. Studies have shown that although V′E decreases substantially with CFB, tidal volume increases acute VO2, which may attenuate the hypoxic effect [3,4,8].

Breath restriction and athletic performance can be a delicate balance. Swimmers can only tolerate a certain level of breath restriction before performance and technique are compromised. Potential reasons for compromised performance include hypoxic conditions and elevated blood lactate. Peyrebrune et al. [7] assessed performance effects on maximal effort swimming, comparing normal breathing (NB) with CFB. The researchers determined that due to lack of tolerance to the breath restrictions, hypoxia at the arterial level may not have even been achieved during experimentation. Also, adaptations such as increasing stroke rate or increasing tidal volume to provide more oxygen to the working muscle may counteract the intended outcome of CFB training. However, the desire to increase anaerobic performance through hypoxic training has continued to hold the interest of researchers, especially due to the contradictory research. One of the most frequent conflicting results observed is the BLA response to CFB [3,5,7-11]. As noted by West et al. [8], it is assumed that lack of available oxygen would cause a shift from aerobic respiration to glycolysis to provide for the unmet demanded ATP, and subsequently increase lactate production. The shift in BLA levels can be expected in sprint swim races, duration of 20-120 s, which depend heavily on glycolytic pathways. It was proposed that restricted breathing should further decrease the amount of oxygen available, causing the body to rely more heavily on glycolysis, thereby driving BLA levels higher. If the postulated process actually occurs, the lingering question remains of what detrimental effects elevated BLA levels may have on race performance [11].

Many have seen significant differences in BLA levels between NB and CFB swimming trials [4,8,11,12]. One study observed significantly decreased BLA levels immediately after CFB swimming [10], and others noted no significant difference [5,7]. Researchers have attempted to explain the differences (or lack thereof) in lactate with a decreased VO2 and equally demanding intensities. Heart rate uniformly tends to decrease when breath rate is limited, but was typically deemed insignificant in affecting BLA levels [3,5,8]. Elevated carbon dioxide concentrations have also been noted as a potential factor since increased carbon dioxide in the bloodstream can inhibit BLA accumulation during exercise [8-10,13]. Holmer and Gullstrand [10] suggested that breath-controlled induced hypercapnia during training may actually be the determining factor in improving swimming performance, instead of the coveted hypoxic training technique.
Furthermore, it has been proposed that CFB may delay blood lactate removal response from the muscle during exercise [3,12,14]. In a free swimming protocol comparing CFB to NB, results showed significant differences in BLA despite insignificant fluctuations in the partial pressure of oxygen pre- and post-swing [15]. However, in a study by West et al. [8] in which tethered and free swimming protocols were used on swimmers utilizing CFB patterns, it was concluded that CFB was not strenuous enough on the respiratory system to cause hypoxia and subsequently increased BLA levels. The general understanding of respiratory and metabolic physiological functions do not appear to be congruent with the observations recorded in high-intensity swimming. Therefore these inconclusive, and often conflicting, results should encourage further investigation on this front.

The present study sought to explore the effects of CFB on post-swing BLA levels within sprint swims, basing the method of experiment on those of Holmer and Gullstrand [16]. In their study, eight highly trained male swimmers completed two separate testing regimens to test BLA and respiratory differences induced by CFB. Lactate levels during CFB patterns resulted in lower BLA immediately post-swim, than the NB counterpart at both the 100-yd and 200-yd distance (p<0.001). The lack of significant change in alveolar partial pressure of oxygen (PAO2) raised doubt that hypoxia was the cause of the results. The authors readily admitted to the surprising nature of the result, without any conclusive reasoning for the lowered BLA levels. Yamamoto et al. [14] conducted a similar experiment with cyclists, in which they observed a delayed response in BLA removal from the muscle in CFB. Yamamoto monitored subjects’ BLA levels periodically up to 16 minutes post-exercise, whereas Holmer and Gullstrand measured BLA levels once immediately post-swim. The present study combined the sprint swim testing utilizing CFB and NB protocols with lengthened monitoring periods of BLA levels post-swing. The purpose of this study was to compare performance and physiological responses for an extended period of time after completing a 100-yard swim sprint test in both CFB and NB conditions.

Methods

Participants and study design

Twenty-two female experienced swimmers from an NCAA Division I varsity swimming team began the study. One subject dropped out due to injuries sustained that were unrelated to the study. All participants were required to complete university-approved documentation prior to participation. They were also asked to refrain from any exercise for at least 24 hours prior to testing sessions.

Procedures

After completing the informed consent form, the participants attended a familiarization session in which each participant completed the Cooper swim test. The Cooper swim test is a maximal distance swim, where the subject swam as far as they could in 12 minutes. The amount of yards swum allowed the subject to be placed in a predetermined fitness category based on age and gender [16].

The first testing sessions were completed two days after the familiarization session, followed by the second testing session one week later. A random cross-over design was used. The test consisted of a maximal effort 100-yard (91.44 m) freestyle swim from a competitive swimming start. During the NB trial, the subject was instructed to breathe freely at a self-selected pattern, which was 1 breath every 2-3 strokes. The CFB trial consisted of a pattern of 1 breath for every 7 strokes. The subject was instructed to maintain this controlled pattern of breathing at all times, including the wall-turns. Prior to each testing session, all subjects were asked to complete a 500-yard (457.2 m) warm-up consisting of low-level intensity swim, drill, or kick at the subject’s discretion. Heart rate (HR) was assessed with a Polar HR monitor (Polar Electro, Kempele, Finland). Lactate levels were assessed using a Lactate Pro (Arkay Inc, Minami-Ku, Japan). Lactate measurements were taken from the earlobe prior to exercise at rest, immediately after completion of the swimming bout, at 1.5 minutes, 3 minutes, and 5 minutes post-exercise completion while the swimmer rested in a seated position next to the pool. HR was also measured at the same time points as BLA post-swing. The subject’s tempo, cycle count, velocity, finish time, and splits were also calculated during testing using Race Analyzer (Parametrix Research, Portland, OR).

Statistics

A repeated measures analysis of variance (ANOVA) was used (2 groups × 5 measurements) to address difference in BLA and HR. A Tukey HSD post-hoc analysis was used for subsequent analyses. A one-way ANOVA was used to assess differences in breathing protocols for time to complete the swim, tempo, cycle count, velocity, and splits. Alpha level was set at 0.05 to determine significance. All statistical procedures were calculated using IBM SPSS Statistics 19 (Armonk, NY).

Results

Subjects swam on average 948.8 ± 56.0 yds (867.6 ± 51.2 m) during the Cooper Fitness Test, and all subjects scored in the “excellent” benchmark of swim aerobic fitness [16]. The 21 test subjects were on average 19.0 ± 1.1 years old, 61.4 ± 12.2 kg, and 163.0 ± 26.3 cm. There was no statistical difference (p>0.05) in BLA levels between the NB trials (1.07 ± 0.29 mmol/L) and the CFB trials (1.10 ± 0.44 mmol/L) prior to the 100-yard swim or at any of the time points after the swim. All post-exercise BLA data are shown in Figure 1. The largest BLA difference measured was at the 5-min marker; NB swims (6.10 ± 0.61 mmol/L) averaged 1.4 mmol/L higher than CFB swims (4.71 ± 0.51 mmol/L). However, these results were not statistically significant, p=0.089.

Table 1 shows the differences in HR and performance times for the NB and CFB swim trials. The post- HR was 10 bpm higher in

![Figure 1](http://dx.doi.org/10.4172/2324-9080.1000164)
Considering the evidence, it appears as though the hypothesis put forth by Yamamoto, suggesting that elevation of BLA is delayed in CFB due to lowered respiration rate does not apply to these data, and therefore may not be universally applied to all forms of exercise utilizing CFB.

In Holmer and Gullstrand’s study [10], the average BLA levels post swim at the 100-yd distance was NB 4.64 mmol/L and CFB 3.70 mmol/L. The average BLA levels immediately post-swim (0 minute) in this study were NB 4.34 ± 2.85 mmol/L and CFB 4.56 ± 1.54 mmol/L. Both of the current study’s values correspond with the NB BLA levels from Holmer and Gullstrand’s 10×100 m trial, which offers evidence that a similar intensity was exerted to break anaerobic threshold. However, in the previous protocol, the subject increased the number of arm pulls per breath within the 100-m swim (3.5, 7, 9 pulls per breath×25 m), while maintaining the same intensity of 85% max. The protocol given in this study maintained a consistent breathing pattern of 7 pulls per breath at a maximum effort and consisted of only 1×100-yd swim. Due to the differences in the CFB trials, it could be suggested that the CFB protocol in the previous study may have more effectively altered oxygen and carbon dioxide pressures. The current study did not measure PAO2, as in the Holmer and Gullstrand protocol, which does limit interpretation and comparison of BLA results in that regard. However, it could be hypothesized that oxygen deprivation was not achieved during the 100-yd trial of the present study, and if it had, a delay in BLA release would have been observed. This is also not verifiable since Holmer and Gullstrand did not monitor BLA levels post-exercise for an extended period of time, but deserves further attention and study.

It is equally important to note that CFB BLA levels did not rise above NB BLA levels, as would be expected if an internal hypoxic environment was achieved during the trial which would increase the production of lactate. Therefore, the results suggest that oxygen demand did not sufficiently outpace oxygen supply to cause an increased shift towards glycolytic pathways. Again, without PAO2 measurements, no definitive conclusions can be drawn. However, even in Holmer and Gullstrand’s study, which did provide such measurements, PAO2 did not decrease enough to suggest that the blood was not saturated with oxygen [10]. This calls into question whether the subjects’ environment was hypoxic, even with the significant difference in BLA levels, or if other variables confounded their results. Regardless, the present study and results appear to suggest that restricted breathing at the 100-yd distance does not affect BLA levels more than breathing normally in the water.

This observation may also have important implications for swimming biomechanics. When respiration is systematically restricted in a maximal intensity exercise, the demands on the muscle become more anaerobic and perhaps limited in performance due to build-up of lactate. Under respiratory stress, it would be reasonable to see an increase in stroke count and cycle rate to increase the frequency of breaths within the CFB swim. This would counteract hypoxia within the respiratory system by increasing PAO2. Biomechanically, these changes may also alter the distance per cycle, and subsequently velocity due to an increase in work. However, as noted in the results, no significant differences in any of the variables appeared in the data collected. Mean stroke counts between trials were nearly identical, as were the other three performance variables. This suggests that even under respiratory limitations, the subjects were able to maintain efficiency and identical stroke mechanics throughout the 100-yd swim. Also, it negates the implication that hypoxia was counteracted
as a result of biomechanical adaptation. Biomechanical efficiency is obtained through rigorous training, which all the subjects were proficient at and had a high-caliber of swim training. These athletes were also trained for high-tolerance of hypoventilation, and displayed comfort and control in an oxygen-restricted environment. Because there appeared to be no disparity in biomechanical variables between NB trials and CFB trials, it is doubtful that any of these variables would have played any significant role in affecting the BLA levels during testing.

In competitive swimming, the 100-yd distance race, especially at the elite level, is an extremely tight race. At the 2012 Olympics in London, the difference between 1st and 8th place in the finals of the women’s 100-yard freestyle was 1.1 seconds [18]. In the current study, the 100 yard freestyle swim with CFB was, on average, 1.1 seconds slower than the NB trial, with 76% of the participants swimming between 0.05 and 5.65 seconds slower in the CFB trial. The five subjects whose times improved with the CFB trial swam an average of 0.72 s faster than the NB trial. CFB patterns are beneficial biomechanically [2]; turning the head to breathe affects the aerodynamic form of the swimmer and therefore increases drag. Swimmers should consider practicing breath reduction patterns to discern the maximum strokes per breath that can be taken before performance begins to suffer. The effects of CFB on time would need to be observed and analyzed over more than two trials to accurately assess the performance and mechanical advantage.

The only significant effect the CFB pattern had on the subjects was a lower HR immediately post-swim (p=0.020); the NB trial was increased by a mean of 10.1 bpm. A slower HR in the CFB trial may be due to the need to increase transit time at the lung and at the muscle to allow for the greatest amount of gas exchange, or due to a reduction in workload from less breathing, or perhaps a parasympathetic response that accompanies breath holding. This slower HR may have prevented a hypoxic environment from developing in the CFB trial.

Conclusion

Coaches and athletes have been and continue to utilize CFB in training and competitions without fully understanding the physiological effects. Many coaches use CFB to mimic hypoxia, but research tends to refute this reasoning. There is no evidence from this study to suggest that lactate levels are altered due to the reduction of oxygen consumption at high-intensity, short-duration freestyle swimming. It is questionable whether hypoxia is even achieved in CFB, or if any CFB pattern would be extreme enough to produce hypoxia in a competitive swimmer. The interplay of hypoxia and hypocapnia is complex, and does not appear to have a firm understanding of their interplay in an oxygen-restricted activity such as swimming. This study lacked the means to measure PAO2, limiting the scope of the study, but it is clear that lactate levels were not elevated or reduced from CFB pattern over an extended period of time. Further study should be devoted to understanding the effects of CFB patterns on PAO2, alveolar partial pressure of carbon dioxide, BLA levels post-swim, VO2, stroke mechanics, and velocity.

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