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Four weeks of controlled frequency breathing training reduces respiratory muscle fatigue in elite college swimmers.

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FOUR WEEKS OF CONTROLLED FREQUENCY BREATHING TRAINING
REDUCES RESPIRATORY MUSCLE FATIGUE IN ELITE COLLEGE SWIMMERS

By

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B.S., University of Louisville, 2013

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ABSTRACT

FOUR WEEKS OF CONTROLLED FREQUENCY BREATHING TRAINING
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Alex R. Burtch

May 9, 2015

Controlled frequency breathing (CFB) is a common swim training modality that involves holding one's breath for ~12 strokes before taking another breath. We sought to examine the effects of CFB training on reducing respiratory muscle fatigue (RMF). Elite swimmers (n = 25) were divided into either the CFB or a group that breathed regularly, every ~3rd stroke. The training intervention included 16 sessions of 12x50-m repetitions with either breathing pattern. RMF was defined as the drop in maximal inspiratory mouth-pressure (MIP) between rest and 46 seconds after a 200 yard free-style swimming race (~114 seconds). Pooled results demonstrated a reduction in MIP after the race at baseline (-11%, p <0.01). After ~4 weeks of training, only the CFB group prevented a decline in MIP values pre to post race. However, swimming performance did not improve. In conclusion, RMF was prevented only in CFB swimmers, with no improvement in swimming performance.

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INTRODUCTION

Unlike terrestrial sports, swimming puts a unique stress on respiratory system. First, submersion of the thorax up to the neck in water creates a hydrostatic force which counteracts the natural expansion of the chest during inspiration [1]. This results in a decrease in vital capacity and residual volume decrease by 9% and 16% respectively [1]. Such submersion can be seen in three of the four strokes in swimming: butterfly, breaststroke and freestyle (front crawl). Second, the submersion of the face in water enforces hypoventilation and rhythmic breathing timed within the stroke mechanics. Each of these aspects has led to increase development of total lung capacity as well as the vital capacity in children who participate in rigorous swim training at pre-pubescent ages [2, 3]. Data published in the early 1990's has shown collegiate swimmers to exhibit larger static lung volumes such as inspiratory capacity, vital capacity, residual volume and total lung capacity against both college runners and non-athletic populations [4].

A common training mechanism by swim coaches today incorporates elongated hypoventilation, commonly termed 'hypoxic swimming'. This theory uses controlled frequency breathing (CFB) versus swimming with a stroke matched (SM) breathing pattern. Therefore, this training increases the time between breaths from every two to three strokes in SM swimming to more than double that amount for CFB.

However, 'hypoxic swimming' may not be the most accurate term for coaches to describe CFB training. Woorons *et al.* [5] recently illustrated that holding the breath close to total lung capacity in swimming is hypercapnic, not hypoxic. Nevertheless, hypoxia can be induced via voluntary hypoventilation at a very low lung volume (close to residual volume), that is exhaling completely before initiating the breath hold [5]. But holding one's breath at residual volume is rarely done in swimming.

Studies have examined the effects of hypoxia and hypercapnia on diaphragmatic fatigue in humans. Both hypoxia and hypercapnia have been shown to significantly increase respiratory muscle fatigue after exercise thus validating the theory of CFB training [6, 7]. While hypercapnia may not induce as much respiratory muscle fatigue as hypoxia, that style is a more practical method for training with respect to comfort of the athletes [6]. Lavin *et al.* [8] examined the effects of CFB in respiratory muscle fatigue, diffusing capacity and running economy in novice swimmers. They found that after four weeks of CFB swim training, novice swimmers were able to improve their maximum expiratory pressure (MEP; $11\% \pm 15$, $p < 0.05$) which, along with maximum inspiratory pressure (MIP), can be used as a marker of increased respiratory muscle strength. The CFB group, however, demonstrated a significant improvement in a 150 yard time trial (-13.2 ± 8.5sec, $p < 0.01$) as a test of performance post training. Does this same training schematic promote the same benefits in elite-level swimmers with more than a decade of swimming experience?

Purpose of the Study

The present study will seek to replicate training procedures from Lavin and colleagues to determine if controlled frequency breath-holding can reduce respiratory

muscle fatigue in elite level swimmers. The information produced within this study may lead to an updated understanding of CFB training and validation as a mechanism of improvement in swim training for today's athletes. Not only will this affect the scope of swim training in collegiate swimmers, but provide scientific evidence of an improvement in respiratory muscle strength from CFB training to be encouraged at all levels of swimming and perhaps adapted into other terrestrial sport training.

Research Questions & Hypotheses

1. Does a four-week training program of controlled frequency breath-holding improve respiratory muscle strength in elite National Collegiate Athletic Association (NCAA) swimmers? Based upon the results seen by Lavin *et al.*, we expect that maximal expiratory pressure will improve by ~10 (SD ±16) cmH$_2$O, with no significant changes in maximum inspiratory pressure.

2. Does a four-week training program of controlled frequency breath-holding decrease respiratory muscle fatigue in elite NCAA swimmers? We believe that RMF will be eliminated in the CFB group by demonstrating no reduction in MIPS or MEPS post-race, after the four week swimming intervention. The effect size is estimated to be moderate (~0.6).

3. Do the suspected benefits associated with a four week training program of controlled frequency breath-holding improve swimming performance in elite NCAA swimmers? We believe the performance measure of a 200 yard freestyle swimming time to be improved (reduced) by ~3 (4) seconds.

Definition of Terms

For the purpose of this study, the following terms and their associated abbreviations are defined.

1. Controlled frequency breathing (CFB) – a training style in which the subject limits his or her breathing beyond the normal scope of swimming (breathing every 8 to 12 strokes vs. every 3 to 4 strokes).

2. Stroke-matched breathing (SM) – the subject breathes in a rhythmic pattern, every two to four strokes.

3. Maximal inspiratory pressure (MIP) – the maximal amount of pressure generated at the mouth during inspiration

4. Maximal expiratory pressure (MEP) – the maximal amount of pressure generated at the mouth during expiration

5. Transdiaphragmatic pressure (Pdi) – the difference between esophageal and gastric pressures measured via bilateral phrenic nerve stimulation recordings.

6. VO_{2max} – the maximal amount of oxygen the body can consume per unit time.

7. Percentage VO_{2max} – expresses intensities that are relevant to a given subject's maximal capacity.

8. PaO_2 – represents the arterial partial pressure of oxygen in the blood (normal values for PaO_2 are expected between 90-110 mmHg).

9. $PaCO_2$ – represents the arterial pressure of carbon dioxide in the blood(normal values for $PaCO_2$ are expected between 35-45 mmHg)

10. Short course swimming – a pool where each length, or lap, is 25 yards in length.

11. Long course swimming – a standard Olympic size pool where each length is 50 meters.

12. Mean and standard deviation were denoted by 'mean (SD)' while range and the 95% confidence interval are given in brackets where notated. Standard deviation is the average deviation of the data upon the mean, a measure of dispersion or variation. A confidence interval defines a mean range of values for which the true value of a measure most likely exists 95% of the time.

13. Rate of perceived exertion (RPE) is a self-reported level of effort based upon the original Borg scale [9] from six to twenty; six being very light exercise and twenty as maximal effort.

Limitations

Our expected limitations include population specificity and timing of the study. This study examined the benefits of CFB training in elite swimmers and therefore results are population specific within the realm of swimming. This study is occurred during the offseason and beginning weeks of preseason in the athletes' training cycle. This most likely had potential effect on 200-yard performance times within the [de]conditioning of the athletes. Incorporating a control group within the study should help offset any training improvements for the performance parameters.

Delimitations

Delimitations can include the choice to not include a novice swimming control group. Lavin *et al.* [8], already studied triathletes as a novice swimming group and therefore the results of that study exist as a novice group by our standards. Literature

reviewed for the purpose of intervention prescription where hypoxia was employed was not included. We encouraged athletes to hold their breath at a high pulmonary volume to induce hypercapnia rather hypoxia which occurs at a low pulmonary volume [5].

Assumptions

We assume that the subjects accurately reported their number or breaths taken per 50 meter interval, and rate of perceived exertion was also accurately reported to the investigators. Anonymity through the study was maintained to encourage honestly from all participants. It is assumed that each athlete gave maximal efforts on all three time trial efforts and all subsequent MIP and MEP testing sessions.

LITERATURE REVIEW

Assessment of Respiratory Muscle Strength

In 2002, the American Thoracic Society and the European Respiratory Society jointly published a statement addressing the variations in testing respiratory muscle strength. In that statement, two of the ATS/ERS's four considerations in testing are applicable of this study: first, "pressures at a given point are usually measured as a difference from barometric pressure" [10]. Second, "pressures measured at a point are taken to be representative of the pressure in that space. Differences in pressure at different locations in normal subjects can arise from two causes: gravity and shear stress. Gravity causes vertical pressure gradients related to the density of the contents of the space. In the thorax this gradient is 0.2 cm $H_2O \cdot cm^{-1}$ height and is related to lung density. In the abdomen, this gradient is nearly 1 cm $H_2O \cdot cm^{-1}$ height. Pressure fluctuations are usually little affected by gravitational gradients. Deformation of shape-stable organs can cause local variations in pressure, such as those that occur when the diaphragm displaces the liver during a large forceful diaphragmatic contraction" [10].

Maximal inspiratory and expiratory pressure measurements represent static pressure generated at the mouth. A single MIP effort primarily reflects the strength of the diaphragm whereas a MEP effort involves supporting respiratory musculature

and the abdominal wall. Both, however are assumed to represent the strength of the respiratory muscles along with passive elastic recoil during an expiratory effort. The passive elastic recoil pressure of the respiratory system can influence MIP by as much as -30 cmH_2O at residual volume and +40 cmH_2O at total lung capacity during expiratory efforts. However, for clinical settings, MIP and MEP typically do not subtract the recoil factor. While the relationship between diaphragm, supporting musculature and the chest wall is complex, these procedures are generally regarded as a global representation of respiratory muscle strength [10]. The device of measurement for this study was a portable, handheld device (MicroRPM™, Carefusion, Yorba Linda, CA) with an electronic display of pressure. Such devices have been shown to be both reliable and valid methods of data collection against laboratory standard pressure transducers [11].

Literature Review Operational Definitions

The online database MEDLINE via the U.S. National Library of Medicine (PubMed) was searched for studies that assessed respiratory muscle fatigue using a mechanism of training. Six studies were found because they met the following criteria: 1) used a pretest-posttest research design with or without randomization 2) recruited a swimming specific population 3) incorporated a control group for comparison. Table 1 includes the results from studies included.

Training Methodologies

The Clanton and colleagues were among the first to evaluate inspiratory muscle training with external pressure devices. Their portable unit created an isotonic environment in which the inspiratory load was between 50-60% of each subjects' initial

MIP measurement [12]. Training sessions were terminated if a subject failed to inspire through the device without creating sufficient flow rates. Mickleborough and Shei [13] used a RT2 trainer, a pressure manometer, with a test of incremental respiratory endurance regimen. This test requires subjects to exhale to residual volume before inhaling maximally. The computer then draws the pressure curve as sustained maximal inspiratory pressure and the area under said curve set the 80% sustained maximal inspiratory pressure training intensity [14]. Subjects 'trained' via breathing into the RT2 trainer for six sets of six resisted inspirations with decreasing work to rest ratios [13].

Kilding *et al.* incorporated a training style like others previously mentioned however volume differed drastically. Experimental subjects performed 30 inspirations against 50% MIP twice daily for six weeks (84 sessions) using a POWERbreathe pressure threshold device [15]. The Wells study [16] was the only to incorporate the PowerLung trainer which incorporates subject's three second inspiratory and expiratory duty cycles. This allows the device to manage flow rates along with the increasing percentage MIP training protocol. The final study, Lavin [8], utilized the controlled frequency breathing swim training which has been discussed previously and is to be used in this study. The results of the literature review are displayed in Table 1.

Table 1: Literature Review

Description of Training Protocols					
First Author Year	Type of Training	Group Characteristics	Group Definition	Study Characteristics	Study Results
Clanton, T.L. 1986	Bourden-type Pressure Gauge Inspiratory Muscle Training	24 Female Age: 19 ± 1	Collegiate Elite 8 Experimental; 8 Sham; 8 Non-swimmers	10 Weeks 3 sessions per week	Increase in MIP*, no difference between groups
Kilding, A.E. 2009	POWERbreathe Inspiratory Muscle Training	10 M; 6 F Age: 19 ± 2	Competitive Club 8 Experimental; 8 Sham-training	6 Weeks Exp: Twice per day; Sham: once daily	Exp: +9 ± 4% MIP 100m Swim: -2 ± 1% 200m Swim -2 ± 1%
Lavin, K.M. 2015	Controlled Frequency Breath Hold Swim Training	9 M; 9 F Age: 25 ± 6	Novice Swimmers 8 Control; 10 Experimental	4 Weeks 3 sessions per week	MIP did not increase Exp: +11 ± 15% MEP* Exp: -13 ± 9s 150-yard swim*
Mickleborough, T.D. 2008	RT2 Trainer: TIRE Inspiratory Muscle Training	15 M; 15 F Age: 18 ± 2	Collegiate Elite 10 Experimental; 10 Sham; 10 Control	12 Weeks 3 sessions per week	Increase in MIP and MEP*, no difference between groups
Shei, R.J. 2014	RT2 Trainer: TIRE Inspiratory Muscle Training	12 M; 12 F Age: 20 ± 3	Competitive Club 8 Experimental; 8 Sham; 8 Control	12 Weeks 3 sessions per week	Exp: +91% MIP*, no difference in Sham or Con
Wells, G.D. 2005	PowerLung Inspiratory and Expiratory Muscle Training	14 M; 20 F Age: 16 ± 1	Club Elite 17 Experimental; 17 Sham	12 Weeks ≈10 sessions per week	Female Exp: +14 ± 12% MIP*, no difference in Male Exp or Sham

TIRE - Test of Incremental Respiratory Muscle Endurance; * - denotes statistical difference at p < 0.05; M - male; F - female; MIP - maximal inspiratory pressure; MEP - maximal expiratory pressure; s - seconds; y - yards; Exp - Expiremental Group; Sham - Sham or Placebo training group; Con - Control Group

Results Interpreted

Increases in MIP were somewhat varied across the swimming population. Comparing the results of Clanton and Wells, it appears that women have a higher affinity to MIP strength improvements than males. However, training incorporating the test of incremental respiratory endurance produced increases across both sexes for elite and competitive populations. It is reasonable to assume that a training volume difference between Kilding and the other studies examined likely contributed to the both sex's increases. These results allude to the potential for strength development at any level of competitiveness however with careful consideration to population and training regimen.

To date, Lavin and colleagues were the only researchers to incorporate an intervention that modified breathing patterns in swimming to evaluate change in respiratory muscle strength. While these results found no improvement in MIP, MEP and swimming performance improved in the experimental group [8]. Kilding *et al.* [15] also found improvements in performance at the 100-meter (-1.7%) and at the 200-meter swim (-1.5%) distances. Therefore it is reasonable to expect an improvement at a 150 yard swim distance as done in the Lavin study. Due to the novice ability of the swimmers, however, a ~13% improvement in swim time was found in the experimental group versus a ~8% improvement in the control. This results in a net ~5% performance difference that can be attributed to the controlled frequency breath holding; marginally larger than the ~2% improvement for the 100-meter and 200-meter distances observed in the Kilding study [15]. It is clear that respiratory muscle strength can be improved by a variety of different methods however it is hardest to induce training effects in elite athletes.

Therefore the present study will incorporate elite level athletes and CFB in order to broaden the scope of potential evidence-based performance improvements.

Evidence of Respiratory Muscle Fatigue

Bilateral phrenic nerve stimulation incorporates either electrical or magnetic stimulation at the cervical spine level which induces contraction of the diaphragm so that fatigue of the respiratory muscles can be objectively measured. This method removes any influence of central fatigue given the external stimulation. Balloon catheters are inserted into the patients' respiratory tract at both the gastric and esophageal levels which then measure pressure deviations from atmospheric pressure. The difference between measured gastric and esophageal pressures results in the transdiaphragmatic pressure (Pdi) which specifically represents the pressure development across the diaphragm, and therefore, inspiratory muscle strength [10]. Respiratory muscle fatigue has been confirmed to exist after both voluntary hyperpnea and breathing through an occluded device across as range of stimulation frequencies free of exercise [17, 18]. Therefore this method has also been applied to gauge the effects of exercise on the diaphragm. Two studies have taken a closer look into diaphragmatic fatigue, via Pdi, post-exercise and its' ability to recover.

Babcock *et al.* [18] used a low frequency bilateral phrenic nerve stimulation technique after a 'highly fit' and 'fit' group participated in high intensity (88-92% VO_{2max}) exercise. Each group exercised for ~15 minutes experiencing a 23% decrease in Pdi indicating similar diaphragmatic fatigue. Furthermore, the time for recovery for each group was 60 minutes. Another study has summarized that heavy exercise for 8-10 minutes at >85% VO_{2max} can stimulate diaphragm fatigue between 15-30% due to an

increased ventilatory demand. [19]. Both studies suggest the notion that although higher fit subjects exercise with higher a ventilatory demand, they exhibit the same amount of fatigue as compared to less fit individuals working at a lower absolute aerobic capacity [18, 19]. This supports the notion that rib cage and abdominal muscles are recruited during increased ventilation in highly fit subjects which encourages the possibility of training adaptations [18, 19].

Respiratory Muscle Fatigue in Performance

During hyperpnea, the relative cost of breathing increases exponentially when moving from moderate exercise to heavy and maximal exercise levels [20]. While at moderate exercise, the cost of the respiratory system accounts for 3-6% total VO_{2max}, heavy exercise accounts for a ~10% demand and maximal exercise accounts for anywhere between 13-15% [20]. The average percentage of total VO_2 devoted to this increased hyperventilation at maximal workloads averaged ~39% [20]. With respect to performance, loading and unloading respiratory musculature has resulted in decreased and increased time to exhaustion, respectively. Using a feedback-controlled proportional-assist ventilator to unload and mesh screens to load the respiratory muscles, subjects' performance was affected by ~14% in each direction when exercising at 90% VO_{2max} [21]. Both loading and unloading significantly differed from separate control trials ($p < 0.05$). At iso-time, five minutes, absolute VO_2 was reduced during respiratory unloading therefore confirming that a percentage decrease in total VO_2 during unloading would be attributable to the decreased demand from respiratory muscles [20, 21].

The link between peripheral muscle fatigue and the increasing demand of respiratory muscles during high intensity exercise has been examined in multiple studies.

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In examining the peak twitch force of the quadriceps after exercise, a significant improvement of force production is evident when the respiratory muscles are unloaded [22]. Quadriceps fatigue was reduced by almost one-third that of control; a ~28% reduction in normal breathing versus ~20% in unloaded breathing conditions [22]. In contrast, loading the respiratory muscles resulted in increased fatigue of the quadriceps from ~12% in normal breathing to ~20% in loaded conditions ($p < 0.01$) [22]. It is believed that loading the respiratory muscles leads to locomotor deoxygenation and therefore increased fatigue levels [23].

As the VO_2 requirements of the respiratory muscles increase, more oxygen-rich blood must be directed towards the working muscles and therefore compromising the peripheral muscles' ability to perform work [20, 24]. In addition, at rest and during exercise, expiratory flow limitations can decrease stroke volume therefore increasing heart rate in order to maintain cardiac output demands [25]. As the heart rate approaches maximum, the ability to compensate for stroke volume may be lost which can reduce overall cardiac output and therefore decreases performance during maximal exercise. Such flow limitations have been examined in swimmers from exercise-induced bronchoconstriction [26]. Air pollutants within enclosed pools can create a reduction in broncodilation spurring the aforementioned chain of action [27].

Controlled Frequency Breathing

Swim coaches have postulated that constant exercise intensity with reduced frequency breathing can increase oxygen extraction and have used this as a training modality. Swimming already uses a form of hypoventilation during the natural rhythm of the stroke. However, this modality further restricts breathing anywhere between two to

five times below the normal breathing frequency. When the breathing frequency is cut in half, for example breathing every two strokes to every four, the amount of measured inspiratory muscle fatigue nearly doubles, 11% to 21% respectively [28]. In submaximal swim intensities, P_aO_2 and P_aCO_2 remain unchanged yet CFB training exhibits decreased P_aO_2 and increased P_aCO_2 [29]. Since exercise intensity did not change between CFB and normal swimming, the postulation of increased O_2 extraction is confirmed [29, 30]. Hypercapnia remains the primary avenue for fatigue resistance with respect to controlled frequency breathing for this study. Lavin's proposed mechanism of action, or improvement, is included below; this helps to illustrate what previous research studies suggest.

Figure 1: Lavin's Proposed Mechanism of Action
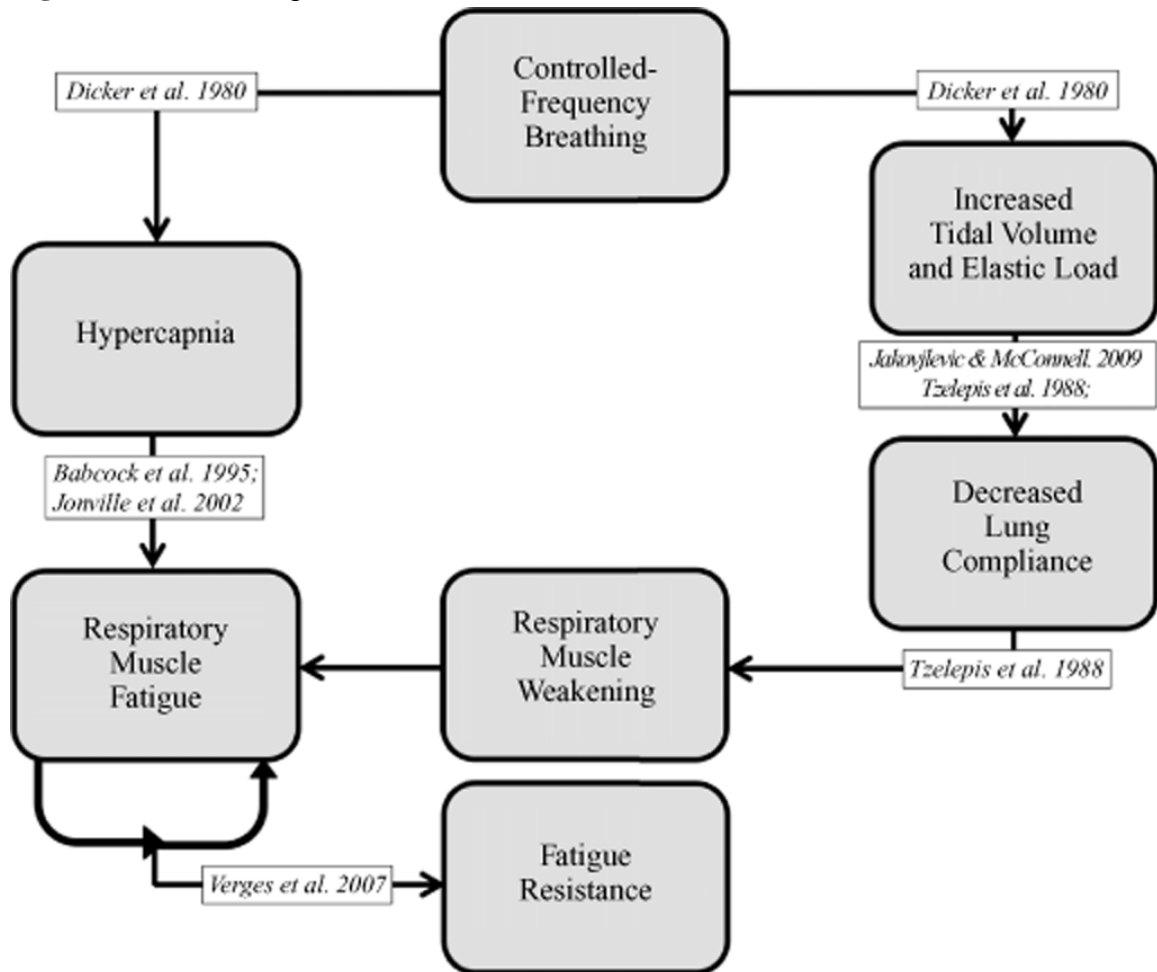
Obtained from Lavin, *et al*. [8].

METHODS

This study was conducted at the University of Louisville. The subjects were athletes on the University's competitive swim team; Institutional Review Board approval was granted from the University, approval #14.0103. Every subject was provided with an informed consent document explaining their responsibilities and risks by participating in this study. There was a familiarization period of two weeks within which all subjects participated in swimming test procedures. Members of this team were considered as elite level athletes having competed on a team that was 11th at the NCAA Division I Championships for the men and 15th for the women in 2014. These rankings place each respective program within the top 10% of Division I eligible programs.

Subjects were eligible for this study if they have competed for the University during the 2013-2014 swim season. No time standard requirements were set for entry into the study, i.e. USA Swimming national standards. Fourteen men and eleven women were recruited.

Settings

All testing and the intervention occurred on campus at the University of Louisville's Ralph Wright Natatorium. During all swimming sessions, pool water temperature was closely monitored and kept between 78-80° F per competitive swimming guidelines set by USA Swimming [31], the national governing body for

swimming. Air temperature was closely maintained to match pool temperature, 78-80°F. For the static MIP and MEP measurements recorded before and after time trial events, subjects were offered a towel to dry off and seated in a chair next to the on-deck computer.

Testing

This study consisted of basic anthropometric data collection, time trial testing, and the swimming intervention. Each testing measure was repeated both before and after the training intervention. This was done in order to facilitate more accurate baseline data collection. After the four week training intervention, post-training data was collected using the same methods.

During MIP and MEP testing, subjects were seated and asked to hold the portable device (MicroRPM™, Carefusion, Yorba Linda, CA) fitted with a flanged mouthpiece. The flanged mouthpiece was selected to decrease leaks around the mouth as subjects perform their efforts. The device was connected to a computer and quality control was evaluated by the investigators using pulmonary management software (PUMA™, Carefusion, Yorba Linda, CA). Subjects performed three maximal efforts for MIP testing, followed by three maximal efforts for MEP testing. When performing MIP testing, subjects were asked to exhale to residual volume before placing their lips on the flanged mouthpiece and inspiring maximally. They were verbally encouraged to ensure maximal efforts, each lasting over 1.5 seconds in duration. The average of the two closest measurements with less than 10% variation produced by the MicroRPM™ device was recorded as a subject's maximal measure. When performing MEP testing, subjects were asked to inspire to total lung capacity before placing their lips on the flanged mouthpiece

and expiring maximally. In accordance with ATS guidelines [10], at least 30 seconds rest was maintained throughout each test to allow for recovery. No noseclips were used during MIP and MEP testing. Sniff tests exist as an alternate method of testing. While the sniff maneuver may generate more Pdi (in cmH$_2$O) than static procedures, the field based research being conducted in this study favors the ease of MIP and MEP testing.

After the warm up, subjects were pre-tested to simulate any deviation from baseline during a competition prior to racing. The athletes then performed a maximal 200 yard short course, freestyle effort using starting blocks; flip turns were allowed and subjects were allowed to select their own breathing frequency. Time was monitored on a S141 300 Lap Memory Stopwatch (Seiko, Tokyo, Japan) and all results from this session were both manually recorded and digitally stored as back-ups. Three MIP and MEP efforts were performed immediately after the time-trail and at ten minutes post-effort in order to evaluate the decrease in pressures and also the recovery rate of the respiratory muscles.

During the familiarization period, age (y), anthropometric data such as height (m) and weight (kg), and body composition were recorded. Body composition was measured via hydrostatically weighing in the Exercise Physiology Lab at the University of Louisville. The Siri and Brozek [32, 33] equations were used for hydrostatically weighing; body composition was recorded as the average of the two equations.

Training Intervention

Each session lasted approximately thirty-five minutes; each subject underwent a standardized 1000 meter warm up of easy, mixed swimming. The training intervention

occurred at the Ralph Wright Natatorium on site for the University of Louisville, set up for long course swimming. The training intervention is described in Table 2.

Table 2: Intervention Description

Training Progression	Group Instructions

Week 1:
     12x50m Front Crawl
     @ 1:00 per 50m
 	
 Weeks 2, 3:
     12x50m Front Crawl
     @ :55 per 50m
 	
 Week 4:
     12x50m Front Crawl
     @ :50 per 50m
	Experimental:
	
	   Limit breathing to 2-3 breaths per 50m
	   24-30 breaths per workout
	
	 Control:
	
	   Breath every 2-3 strokes per 50m
	   105-120 breaths per workout

Only breaths taken while swimming were considered countable breaths during data collection. The controlled frequency breathing group was encouraged to limit their breathing to two breaths per lap resulting in about 24 breaths per workout. The control group was asked to breathe on a stroke-matched basis, breathing every 2-3 strokes accumulating to 10-12 breaths per lap. At the end of each workout, each subject reported their number of breaths taken during the workout along with RPE. Training sessions were supervised by at least one member of the University of Louisville swimming coaching staff.

Research Design

The research design implemented for this study was a pre/post-test design with control group. This was a quasi-experimental design in which a convenient sample of elite college swimmers was used.

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Statistical Analysis

To compare groups at baseline for standard physical and anthropometric characteristics, independent *t*-tests were performed. For these variables that were not normally distributed, a Mann-Whitney U test was used to compare groups. To compare swimming times and resting MIP and MEP between the familiarization session and the baseline session, paired *t*-tests were used. If any of the paired variables were not normally distributed (as determined by a Shapiro-Wilk's test), a Wilcoxon Signed Rank test was used instead.

A repeated measures analysis of variances was used to assess mean changes in MIP and MEP values at four different time-points during the baseline session. These time-points were: after the warm-up but before the race, ~45 s, ~80 s, and ~110 second post-race. Another repeated measures ANOVA was run to establish potential effects between sex and respiratory muscle fatigue development. To determine how much MIP values decreased from pre to post race, and when the MIP values recovered back to normal pre-race values a Bonferroni correction was performed *post hoc*.

To examine changes in the drop in MIP values between pre and post training, a 2x2 repeated measures analysis of variance was used. The independent variable was the training program [Experimental Group = CFB training group; Control group = stroke matched (SM) group] and the number of measurements per variable (two measurements per variable: baseline and post-testing). As such, subjects were nested within group and crossed with time, such that the Lee notation looks like: $S_{10} \cdot (G_2) \cdot T_2$.

Indices of responsiveness to CFB training to respiratory muscle fatigue was calculated according to previous methods [34, 35]. Effect size (ES) was defined as the mean change of the variable between baseline and post-training divided by the SD of the variable at baseline. An effect size of 0.0 to 0.2 was considered trivial, 0.2 to 0.5 was small, 0.5 to 0.8 was moderate, and 0.8 and above was strong. The standardized response mean (SRM) was calculated as the average change divided by the SD of the change. The *t*-statistic was defined as the mean change of the variable between base-line and post training divided by the standard error (which is the SD divided by the square root of the sample size).

For the dependent variables that were not altered between familiarization and baseline sessions (i.e. swimming performance), the day-to-day coefficient of variation was calculated as the mean of the two trials divided by the standard deviation of the two trials x 100. Measurement error (otherwise known as the typical error or the within subject standard deviation) was calculated as the square root of the within-subjects error variance (i.e., the within-subject standard deviation) derived from a repeated measures ANOVA. Reproducibility was defined as $2.77 \times$ the measurement error [36]. That is, the difference between two measurements obtained on different days for the same subject is expected to be less than 2.77 times the within-subject standard deviation for 95% of pairs of observations [36]. Since the calculation of reproducibility may be considered too stringent, the smallest meaningful change was reported as half of the reproducibility [37].

Multiple linear regression analyses (stepwise model) was used to determine which combination of sex, MIP values, MEP values, and respiratory muscle fatigue (MIP pre-race – MIP 46 s post-race) best predicted 200 yard free-style swimming time.

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Sample size calculation was estimated from the change in the drop in MIP and MEP values between pre and post-intervention. Using online statistical software (G*Power Version 3.1.7, Universität Kiel, Germany), the following was calculated for the difference between two dependent means (matched pairs, t-test family): statistical power $(1 - \beta)$ was set at 0.8 (80%), type I error rate at 5% ($\alpha = 0.05$), and a moderate effect size = ~0.60. A total of 24 subjects was estimated and 25 subjects were recruited.

The data was analyzed by a statistical software package (SPSS Version 21.0, IBM SPSS Statistics Inc., Chicago, IL). Statistical significance was declared when $p < 0.05$ unless otherwise noted.

Data Management and Storage

Data was manually recorded on specially formatted collection sheets which were stored in a locked file cabinet at the laboratory of the faculty advisor. This lab was locked at all times with access granted only to the faculty advisor and student researchers. Electronic backup of information was provided by the investigators on excel spreadsheets on password protected computers.

RESULTS

Twenty-five subjects were recruited for participation during this study, eleven women and fourteen men. Subjects were randomly placed into either control (n=12) or experimental (n=13) groups. During the study, one subject was unable to continue due to illness while another did not meet the minimum session requirements. Three additional subjects were lost due to travel at the time of follow-up testing. Therefore, twenty subjects were retained through the end of the study. Nine of these were experimental group (five men and five women) and eleven in control (seven men and four women). All subjects completed both familiarization and baseline data collection. The subjects' baseline anthropometric data at baseline is described in Table 3. All data was normally distributed except for age. There were no differences between any of the participant characteristics.

Table 3: Participant characteristics at baseline

Variables	Control, SM (n = 12)	Experimental, CFB (n = 13)	p -value	Combined Mean (n = 25)
Age (yrs)	19 (1) [18 to 22]	20 (1) [19 to 22]	0.13	20 (1) [18 to 22]
Weight (kg)	78.3 (10.3) [63.0 to 93.9]	76.8 (10.5) [56.8 to 89.8]	0.71	77.6 (10.2) [56.8 to 93.9]
Height (cm)	176 (8) [162 to 189]	178 (11) [156 to 191]	0.64	177 (9) [156 to 191]
BMI (kg/m²)	23.4 (1.4) [21.4 to 25.9]	22.8 (1.8) [20.2 to 26.5]	0.33	23.1 (1.6) [20.2 to 26.5]
Body fat percentage	17 (6) [9 to 26]	15 (3) [9.8 to 22.3]	0.51	16 (5) [9 to 26]
Wing span (cm)	183 (11) [165 to 199]	184 (13) [158 to 199]	0.88	183 (12) [158 to 199]
Wing span divided by height (%)	104 (2) [98 to 106]	103 (2) [100 to 108]	0.53	104 (2) [98 to 108]

Mean (SD), [range]

Intervention Data

Baseline testing and follow-up both occurred within one week of the intervention period, respectively. There were 38 (8) days between baseline testing and follow-up. Each subject completed at least the minimum of twelve training sessions with a group average at 14 (2) sessions. The number of breaths taken during the intervention period was not normally distributed so a Mann-Whitney U test was run to assess statistical

differences. RPE was normally distributed. There was an overall difference between groups for both RPE and the number of breaths taken in total per workout (Table 4).

Table 4: Intervention Data

Group		Weekly Interval Progression				Average	p-value
		1:00	:55	:55	:50		
Experimental	Total Breaths	24 (2)	24 (2)	25 (1)	27 (6)	25 (3)	<0.001
	RPE	14 (1)	15 (1)	15 (1)	17 (1)	15 (1)	
Control	Total Breaths	113 (13)	111 (9)	111 (6)	114 (9)	112 (9)	<0.001
	RPE	10 (1)	11 (1)	10 (1)	12 (2)	11 (1)	

Mean (SD)

There was strong correlation ($r = 0.95$, $p < 0.001$) between the two swim trial times. The week to week variability in 200-yard freestyle times between familiarization and baseline sessions was 1.4%. The measurement error 1.5 seconds, the subject reproducibility was 4.3 seconds, and the smallest measureable change was 2.1 seconds. There was no statistical difference in swim time between familiarization and baseline testing ($p = 0.247$). However, MIP values improved by 7 (13) cmH_2O [95% CI, +2 to +13 cmH_2O, $p = 0.01$] between familiarization and baseline, which is roughly a ~6% gain. MEP improved by 13 (20) cmH_2O [95% CI, +6 to +21 cmH_2O, $p < 0.01$] between familiarization and baseline for a ~10% gain. Information regarding the subjects' MIP and MEP measurements against normative values are reported in Table 5 [38].

Table 5: Baseline MIP measurements against normative values

	Control (n=13)		Experimental (n=12)		Combined (n=25)	
	Observed	% Predicted	Observed	% Predicted	Observed	% Predicted
MIP (cmH$_2$O)	137 (28) [91 to 185]	139 (34)* [76 to 204]	125 (31) [91 to 171]	126 (36)* [69 to 202]	131 (30) [83 to 185]	133 (36)* [69 to 204]
MEP (cmH$_2$O)	142 (31) [88 to 184]	113 (34) [79 to 191]	156 (20) [115 to 184]	121 (26)* [86 to 174]	149 (27) [88 to 184]	117 (31)* [79 to 191]

Mean (SD) [range]; * - denotes statistical significance from predictive values ($p < 0.05$)

Respiratory Muscle Strength

Both groups were collapsed into a single evaluation given that there were no differences between groups at both time points. MIP values (at rest, pre-race) did not improve between pre and post-intervention ($p = 0.629$). MEP was consistent between time points with no increase in strength ($p = 0.968$) (Figure 2).

Figure 2: Respiratory Muscle Strength At Rest
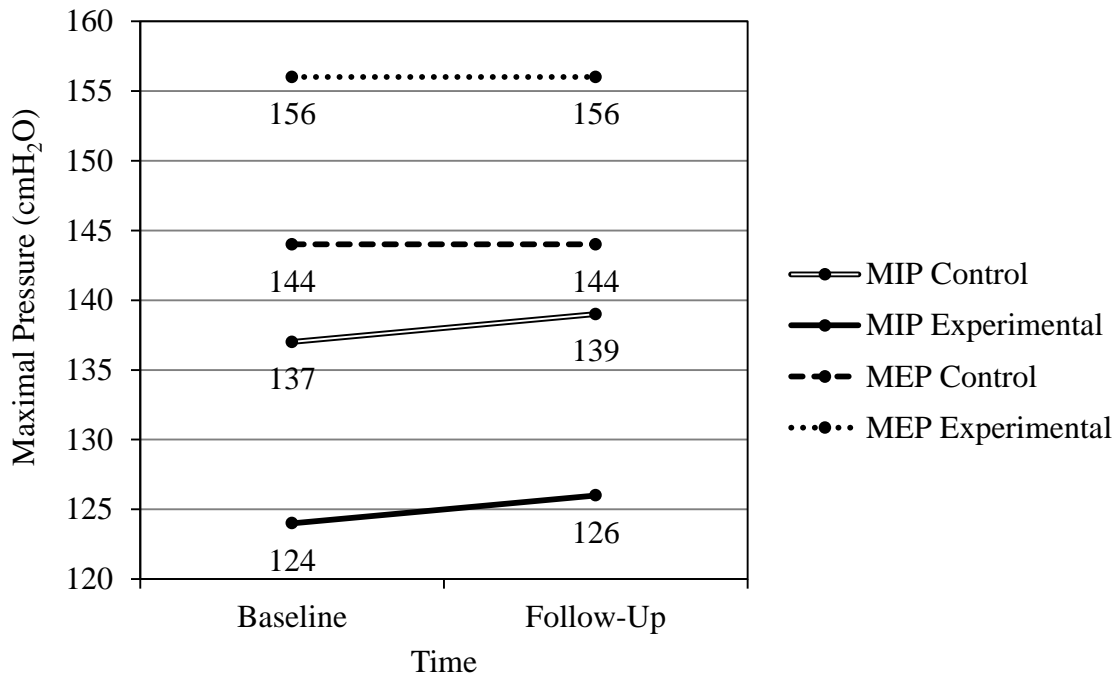
Despite no improvements, a multiple regression analyses predicting swim time was established using baseline data (n = 25). Sex, maximal MIP and MEP were entered into a stepwise regression prediction. Both sex and maximal MIP were selected as variables contributing to 200-yard swim time while MEP was not considered. This regression equation has a standard error of 3.6 seconds and F-ratio of 32.9. The formula is follows where female equals '0' and '1' for male for the sex consideration:

200 yard swim time (seconds) = 127.2 – (10.7)·(Sex) – (0.05)·(MIP in cmH$_2$O)

**(F-ratio = 32.9, SEE = 3.6sec, Adjusted r^2 = 0.73, p < 0.001)**

Respiratory Muscle Fatigue

During the baseline session, MIP values were tracked as different measurement time-points. About 30% of the variance in MIP scores was explained for by time (partial η^2 = 0.31). There were no differences between either groups or sex ($p = 0.878$, $p = 0.670$

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respectively) during the baseline session, nor were there any interaction effects. Thus, for the purpose of only the baseline session, both groups and sexes were combined into a single representation below (Figure 3). There was a -15 (14) cmH_2O decrease in MIP at ~46 sec post-race [95% CI, -20 to -9 cmH_2O, $p < 0.001$]. The decrease was similar between males and females [males = -16 (17), females = -14 (11) cmH_2O, $p = 0.732$]

Figure 3: MIP Fatigue at Baseline
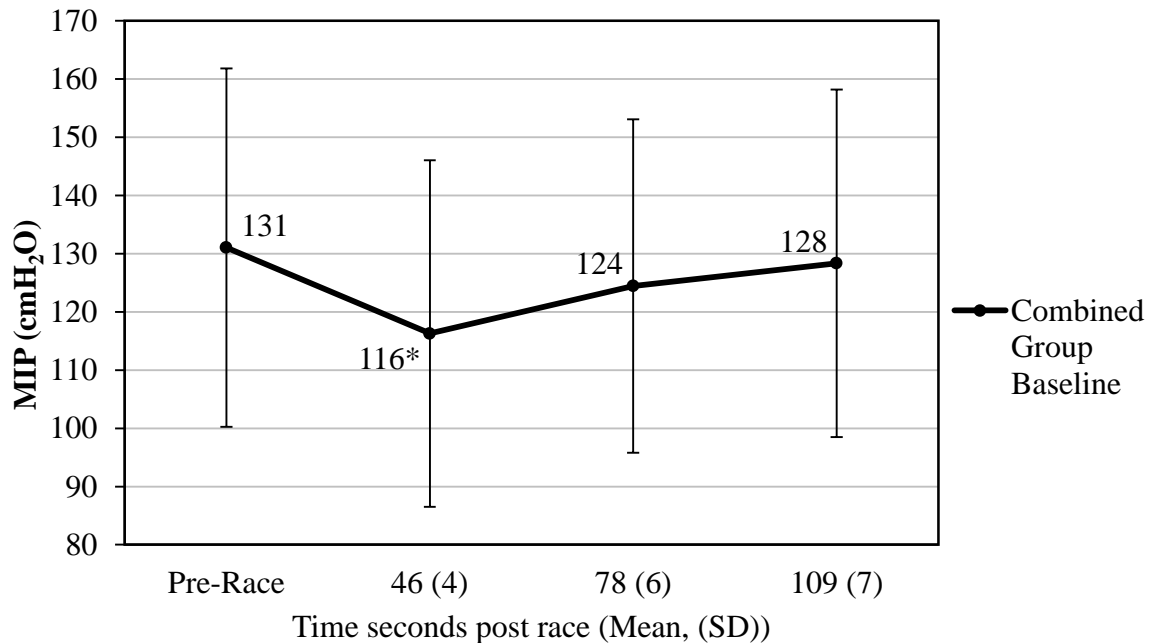
Post-intervention, fatigue was diminished in the CFB group ($p = 0.046$) while fatigue was still present in the control group (Figure 4). In reference to sex, neither group saw a difference between drop in MIP post-training (EXP: males = +2 (13), females = -8 (13) cmH_2O, $p = 0.320$; CON: males = -5 (14), females = -24 (13) cmH_2O, $p = 0.056$). The specific difference between groups and initial MIP measures is illustrated below.

Figure 4: Change in MIP
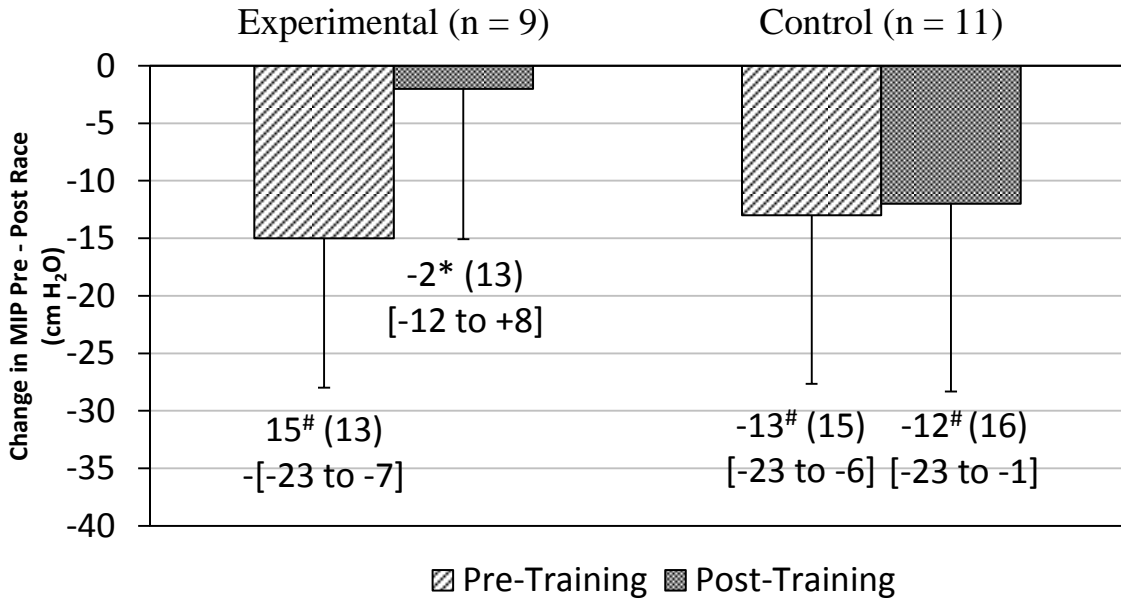
(Mean (SD) [95% Confidence Interval]; * - denotes difference from pre-race, $p < 0.05$; # - denotes no difference)

The MEP tests were always performed after all the MIP tests. Thus, MEP values did not show any changes at any time-point pre or post intervention in either group represented by Figure 5 below. Given the rate of recovery, all MIP and MEP measurements taken at 8-12 minutes post time trial showed no statistical difference from pre-exercise. These values are represented in Table 6 along with significance values compared against the resting state for that day.

Figure 5: MEP Values Pre and Post Time Trial at Baseline
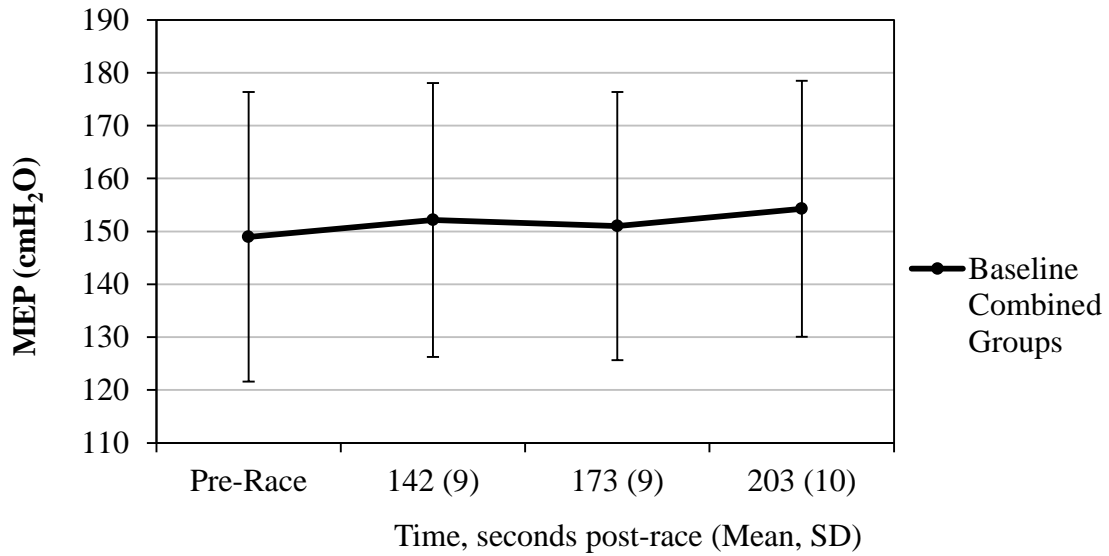
Table 6: MIP and MEP Values at 8-12 Minutes Post-Exercise

Baseline, Combined Groups (n=25)			Follow-Up Experimental (n=9)			Follow-Up Control (n=11)		
Time (sec)	MIP cmH_2O	*p-value*	MIP cmH_2O	*p-value*	MIP cmH_2O	*p-value*		
Rest	131 (31)	-	126 (37)	-	139 (26)	-		
541	131 (33)	0.880	133 (39)	0.112	134 (30)	0.076		
579	131 (27)	0.848	128 (40)	0.714	132 (39)	0.243		
608	129 (32)	0.380	124 (36)	0.675	138 (32)	0.817		
Time (sec)	MEP cmH_2O	*p-value*	MEP cmH_2O	*p-value*	MEP cmH_2O	*p-value*		
Rest	149 (27)	-	156 (20)	-	144 (27)	-		
642	150 (32)	0.892	150 (29)	0.376	146 (27)	0.656		
674	151 (29)	0.674	151 (31)	0.368	142 (28)	0.662		
705	153 (29)	0.430	155 (27)	0.829	143 (26)	0.896		

Mean (SD); Time - Time in seconds post time trial effort; MIP - maximal inspiratory pressure; MEP - maximal expiratory pressure. The specific p-value compared against rest

DISCUSSION

The purpose of this study was to answer three research questions. First we wished to assess whether a four-week training program of controlled frequency breath-holding improve respiratory muscle strength in elite NCAA swimmers. There was no change in MIP values as we hypothesized however the suspected increase of ~10 cmH_2O in MEP was not observed. Next, we wished to assess whether this training program improved swimming performance in elite NCAA swimmers. While the smallest measureable change was two seconds, we did not see any improvement in swimming performance as a whole in either group. Finally, we assessed whether this CFB training reduced respiratory muscle fatigue? In the CFB group, high frequency inspiratory muscle fatigue was reduced.

Respiratory Muscle Strength

With regards to increasing the maximum strength of the respiratory muscles, the population may have contributed the largest determining factor. Each group demonstrated MIP and MEP values that were 26% and 21% beyond predicted values respectively. A ceiling effect could have limited the improvement capabilities of our group. Clanton *et al.* [12] observed that competitive swim training can increase the strength of the respiratory muscles so it is conceivable that elite swimming populations already function at a high

level. However, respiratory muscle training that increases MIP values may be applicable to swimmers who fail to achieve predicted values.

Developing respiratory muscle strength has a statistically significant effect on improving swimming performance as predicted by our regression equation. This analysis determined that for 10 cmH_2O improvement in maximal static MIP values, 200 yard swimming performance improves by 0.5 sec. Given the range of percent predicted MIP values that we saw (69 to 202% predicted), it's reasonable to assume that a number of swimmers can improve their performance by improving respiratory muscle strength. Since 2001, the difference in medal times for the 200-yard freestyle at the NCAA championships has been tight. For men, the average difference between first and second is ~0.6 seconds and between second and third, only ~0.4 seconds. For women, the differences have been ~0.6 seconds and ~0.5 seconds respectively. Therefore, at the elite level, an improvement of 0.5 seconds can make a large difference.

Respiratory Muscle Fatigue

The relative cost of breathing discussed in females is 13.8% compared to 9.4% in males [39]. Separate studies suggest that women have a higher resistance to fatigue than their male counterparts; at 10 minutes following a maximal effort cycling test, the male diaphragm fatigued ~31% versus ~21% of females [40]. Due to the increased cost of breathing during exercise, it can be hypothesized that over time, females develop greater resistance to fatigue. In the present study however, there were no significant sex differences in the decrease in MIP values at either the baseline or follow-up time points.

Traditionally, respiratory muscle fatigue has been documented with either extended periods of endurance exercise or high intensity bouts lasting eight to ten

minutes [19]. Our data, however, suggests that respiratory muscle fatigue can occur in swimming after maximal or supramaximal bouts of work lasting under two minutes. Documented instances of fatigue have been measured between 15-30% of pre-exercise values, yet our swimmers exhibited an 11% drop [18, 19]. Previous research suggests that increased respiratory muscle VO_2 requirements increases peripheral muscle fatigue therefore jeopardizing a 200-yard swim performance [19, 22]. It can be reasoned that similar distances with different strokes (for example, the 200-yard breaststroke) may incur the same fatigue. Increasing the fatigue resistance of the muscles should alleviate the relative oxygen demand of ventilation [20].

Since the MIP values were taken at three separate points after the swim time trial, we were able to gauge the rate of recovery for these swimmers. Maximal strength developments are typically measure at excess of 50 Hz while maximum duration of volitional force develop is measured around 20 Hz [41]. Here has been the traditional divide between high frequency muscle fatigue and low frequency muscle fatigue; a difference between peak power and maximum contraction duration [41]. Multiple studies have cited that a low frequency phrenic stimulation assessment of respiratory muscle fatigue takes up to and beyond 60 minutes to completely recover [18, 19]. With the rate of fatigue recovery under one minute, this type of power reduction is typical of high-frequency muscle fatigue. Many elite swim programs utilize one or two practices per week with multiple max efforts, termed 'VO$_2$' workouts. Repeated exposure to this practice style could have led to high adaptability of the diaphragm and supporting musculature.

After four weeks of training, the experimental group was able to prevent high frequency respiratory muscle fatigue. With a relatively short exposure period (four weeks) to the training stimulus, it is encouraging to see such benefits. Elite or experienced swimmers have become numb to the traditional 'hypoxic' training method; coaches should elevate the intensity to incur beneficial adaptations. Following the study protocol established here, limiting the swimmers' breaths to three or less per 50m with multiple 'sets' per week can decrease respiratory muscle fatigue.

Why was there a reduction in respiratory muscle fatigue in the CFB group? We can only speculate. One proposed mechanism of action can be derived from a study evaluating the mechanics of breath holding. In trained apnea divers, the actions of the respiratory muscles during extended breath holds can be divided into two phases: an *easy-going* phase and a *struggle* phase [42]. During a single breath hold that lasts about 209 sec, 55% (115 sec) was spent during the easy-going phase and congruently, 45% (94 sec) in the struggle phase. During the struggle phase of breath-holding, there is a progressive pressure development against the glottis creating higher elastic loading, resulting in increased muscular recruitment of both inspiratory (diaphragm and rib cage muscles) and expiratory muscles (abdominal wall). Cross and colleagues [42] demonstrate that during the final 40% of the struggle phase (the last ~38 sec), recruitment of the inspiratory rib cage musculature is preferred over the diaphragm to resist diaphragmatic fatigue. During controlled frequency breathing in our group of swimmers, we can speculate that they spent significantly more time in the *struggle* phase of each breath hold, as demonstrated by the elevation in perceived exertion over the training period (Table 4). The increase exposure to the *struggle* phase may have aided the CFB

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group to selectively recruit rib cage musculature over the diaphragm in order to preserve normal diaphragm function, therefore resisting fatigue.

Highly fit (VO_{2max} = 69 ml/kg/min) and fit subjects (VO_{2max} = 50 ml/kg/min) both experience diaphragmatic fatigue after 15 minutes of exercise at 88-92% of relative VO_{2max} to exhaustion [18]. During the first nine minutes of exercise, the diaphragm of highly fit subjects produced ~30% more force compared to the fit group in order to meet the higher ventilation requirements. Yet over the final six minutes of exercise, there was no difference in diaphragm force output between groups, suggesting that the same rib-cage and abdominal muscle recruitment may have occurred as seen in the divers [42]. During controlled frequency breathing, both tidal volume and $PaCO_2$ rise as there is a positive, linear relationship between the change in tidal volume and the change in $PaCO_2$ [29, 43]. An increased tidal volume is primarily accommodated by rib cage displacement, therefore increased recruitment of those rib cage muscles [43].

A second, proposed mechanism for why there was reduced respiratory muscle fatigue from CFB training is via the increased CO_2 build-up in the blood. It has been shown that hypercapnia from restricted breathing induces arterial blood acidosis which impairs diaphragmatic strength [6]. Thus, over a period of training, exposure to increased acidosis could improve fatigue resistance. In fact, Verges and colleagues [44] revealed that subjects who developed more than 10% diaphragmatic fatigue, reduced both blood lactate and the amount of diaphragmatic fatigue after four to five weeks of respiratory muscle training. Thus, while speculative, it is reasonable to assume that CFB training: (A) can induce accessory respiratory muscle recruitment, sparing diaphragmatic work,

(B) exposes the swimmer to increased acidosis induced by hypercapnia, eventually resulting in adaptations to prevent/reduce high frequency inspiratory muscle fatigue.

Performance Implications

The 1.4% week-to-week variability in 200 yard, short course, freestyle swimming performance is low and is expected with athletes at this caliber. This variability can be used to describe in-season time trials. With most sports that require a taper, end-season performance is desired to be markedly better than in-season benchmarks. Our training protocol assessed respiratory muscle fatigue over four weeks with no change in performance attributed to the decreased respiratory muscle fatigue. An intervention lasting much longer and possibly crossing over into end-season tapered performances may show a link between improved fatigue and improved performance.

Study Limitations

One of the largest limitations of this study is qualifying respiratory muscle fatigue by a simple field test static MIP and MEP measurements. This is a gross oversimplification of respiratory muscle fatigue. This test is more associated with high neuronal firing frequency and thus may be a poor indicator of low frequency fatigue [10]. Thus, if these swimmers had long lasting low-frequency fatigue, we were not able to assess it. Furthermore, these tests are highly effort dependent and the reduction could represent any one or a combination of factors such as lack of motivation, central fatigue, or peripheral high frequency fatigue [10]. However, these swimmers are used to providing a maximal volitional effort and we believe that lack of motivation was not a factor here. Furthermore, the ATS/ERS guidelines suggest that MIPS and MEPS are

widely used for specific tests of respiratory muscle strength, and can be used to detect high frequency neuronal respiratory muscle fatigue [10]. Thus, we feel that as a field test measurement, the drop in MIPS accurately demonstrates high-frequency inspiratory muscle fatigue.

The ATS/ERS guidelines suggest 30 seconds rest between maximal volitional efforts of either MIP or MEP [10]. Generating inspiratory pressure requires contraction of the diaphragm which is principally associated with respiratory muscle strength and fatigue. While both expiratory and inspiratory muscle fatigue occur, the latter is a clear representation of the diaphragm and therefore, respiratory specific [45, 46]. Therefore the priority was placed on MIP over MEP; recovery was observed before the final MIP measurement rendering post-race MEP measures obsolete. One limitation of these measurements is that they disallow discrimination between weaknesses of different respiratory muscles. However the ease and simplicity of these measures provide worthwhile selections.

Another limitation was that this study took place during the swimmer's summer pre-season where their ability to perform the 200-yard free time trial at 'lifetime best' was not possible. However, since no improvements were seen, and with high correlation between separate days and efforts, a maximal effort lasting 90-120 seconds provided requisite stimulus for fatigue changes to be observed.

Conclusions

In conclusion, this study demonstrated these important findings:

1. Restricted breath-holding swimming training reduces high frequency inspiratory muscle fatigue in elite college swimmers. However, this did not result in any improvement in performance.

2. Regression analyses demonstrated that 200-yard swimming performance can be improved by 0.5 seconds for every 10 cmH_2O improvement in maximal static MIP scores.

3. The smallest measureable change in 200 yard freestyle swimming performance in top elite college swimmers is 2.1 seconds. Any change that is less than 2.1 seconds represents week-to-week biological variability.

4. With regards to volitional tests of respiratory muscle strength, a familiarization session must be performed prior to baseline testing if MIP and MEP values are to be assessed.

Future Research Directions

With regards to swimming, performance at the end of the year is largely believed to be a representative body of work put in by the athlete and coaching/support staff. Therefore given the nature of competitive sports, it is impractical to assign a single cohort of swimmers within the team to a rigorous controlled frequency breath holding program with known benefits. Instead, an entire team could adopt the training protocol over the course of an entire season wherein their performance can be measured against other elite teams using swimming time databases. A study focused on concurrent inspiratory

muscle training to develop respiratory muscle strength can further describe the relationship between strength, fatigue and performance. If an observable increase in strength can be observed using methods described elsewhere [15], would this help or harm the reduction in fatigue associated with controlled frequency breathing?

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