The Unintended Consequences of Tension in the Abdominal and Lumbar Musculature on Swimmers' Ventilatory and Metabolic Indices

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Abstract

Swimming performance is governed by the ability of the body to consume oxygen for energy production, and for most events increase the resistance on the propulsive surfaces, and decrease resistance on the non-propulsive surfaces of the body. It has been proposed that the abdominal and erector spinae muscles contract during swimming to decrease form resistance or drag on the swimmers' body to increase speed. We hypothesized that contracting these muscle groups would negatively impact pulmonary functions and increase oxygen consumption. Significantly lower pulmonary functions and higher resting oxygen consumption were observed while the abdominal and erector spinae muscles were contracted. We propose that contracting the abdominal and erector spinae muscles could cause decrements in crawl stroke swimming performance.

Key Words: Swimming, Ventilation, V02, Propulsion, Resistance

INTRODUCTION

Pulmonary function does not appear to place a limit on maximal exercise in most physical activities. However, in competitive swimming, the posture and movements required to properly execute different strokes often impedes the ability of the body to ventilate the lungs and consume oxygen while swimming the butterfly, breast stroke and crawl stroke where breathing is restricted to the movement pattern of the arms and legs and the swimmers' faces in the water.

In competitive swimming, particularly during the crawl stroke, the position of the head in the water and the sequence within the stroke where the swimmer can breathe places some restrictions on pulmonary ventilation. In spite of these limitations, pulmonary ventilation has not been shown to limit the performances of competitive crawl stroke swimmers (8). The pulmonary functions of swimmers have been studied with regularity because some researchers consider the water pressure against the rib cage a challenge to ventilation that results as an adaptation to exercise not shown in other sports. Clanton and co-workers (2) compared the effects of 12 weeks of inspiratory muscle training, regular aerobic training and 12 weeks of swim training. Significant improvements in the swim training group were noted for vital

capacity $p < 0.01$, functional residual capacity $p < 0.0001$, and total lung capacity $p < 0.0025$ when compared to the regular aerobic training group.

Competitive swimmers can have greater total lung capacities than other athletes as shown by Cordain et al., (3) and greater force expiratory volume Courteix co- workers (5). Armour and co-workers (1) compared pulmonary functions of age-matched swimmers and runners. Swimmers had significantly greater total lung capacity, vital capacity, maximal inspiratory capacity and forced vital capacity than runners when the data were normalized for age and body size. Therefore, swimmers' breathing mechanics and lung volumes should not necessarily limit their performances. These findings suggest that swimmers are not limited directly by ventilatory factors and because of either genetic and/or developmental reasons, appear to have better respiratory and pulmonary functions than athletes participating in other sports.

In terms of bioenergetics, swimming performance is determined by the swimmer's ability to expend energy from both aerobic and non-aerobic sources depending on the events in which they compete. Biomechanically, the swimmer must increase resistance on the propulsive surfaces of the body like the hands, legs and perhaps the forearms to produce force. Conversely, they must also decrease resistance on the non-propulsive surfaces of the body like the torso, head and hips described by Counsilman & Counsilman. (4). Skinner (11) suggests that the posture of the body should be optimized while swimming the crawl stroke to decrease form drag and increase speed. This change in posture involves contracting the abdominal muscles to flatten the abdomen and erector spinae muscles to flatten the lumbar portion of the spinal column which collectively produce a more streamlined body. This proposed change in posture could interfere with the coordination of movement between the abdominal muscles and the diaphragm, and work in direct opposition to the accessory respiratory muscles limiting the swimmer's ability to ventilate air and consume oxygen. No experimental data in the literature was found that substantiates that contracting these muscle groups decreases resistance on a swimmer. We hypothesize that these muscular contractions limit the swimmers' ventilatory capacities and increases oxygen consumption during swimming.

METHODS

Prior to initiating a logistically complex study of swimmers in the water, a land-based study was undertaken to determine if contracted muscles would negatively affect pulmonary functions measured in a controlled laboratory setting. Pulmonary functions and resting oxygen consumption were measured under control and experimental conditions. The non-contracted or control conditions (NC) involved measurements made with subjects in a standard sitting position. The experimental or contracted condition (CC) involved participants sitting upright in a chair with their abdominal and erector spinae muscles tensed. This condition was documented by holding a measuring tape around the abdominal area and the participants were instructed to maintain a constant circumference. The participants maintained an upright vertical position of the upper body that was documented by using a meter stick placed next to the trunk. The lumbar spine of the participants was flattened against

the back of the chair as close as possible according to the recommendations of Skinner (2007) and the USA Swimming Sport Science Department. It was hypothesized that if vital capacity (VC), maximal voluntary ventilation (MVV), forced vital capacity (FVC1) and resting oxygen consumption (RVO2) and resting carbon dioxide production (VC02) were altered with the subject's abdominal and erector spinae muscles contracted in a seated posture, then maximal exercise could be impeded by the decreased ventilatory capacity and increased oxygen consumption and place limits on the performance of the competitive crawl stroke swimmer.

Thirteen participants involved in swimming activities (8 males, 5 females) from 22-60 years of age volunteered for evaluation of their VC, MVV, FVC1, RVO2 and VC02 under the two differing postural conditions NC and CC. Each participant was measured on a Spirometrics Flowmate III Spirometer for pulmonary functions and a metabolic cart for RVO2 and VC02. There were 3 minutes of rest between counter-balanced trials for all measurements. All participants' pulmonary functions were expressed relative to their age, body weight and height and resting RVO2 relative to body weight.

A one-way analysis of variance (ANOVA) was used to compare NC and CC conditions. We used the Bonferonni correction factor of 0.05/3 which required a p value of less than 0.0167 for the correlated variables of VC, MVV, and FVC1. The variables of RVO2 and VC02 were not significantly correlated and a 0.05 level of significance was used to test these hypotheses. We sought to account for the percent of variance in the dependent variables related to the variance in the independent variable by using the Omega Squared statistic (13).

RESULTS

There were significant differences between evaluations for VC, MVV, FVC1, and RVO2 between EC and CC. An Omega Squared computation showed the amounts of variance in the dependent variables that were accounted for by the variance in the independent variable or CC. Results are reported mean plus or minus standard error.

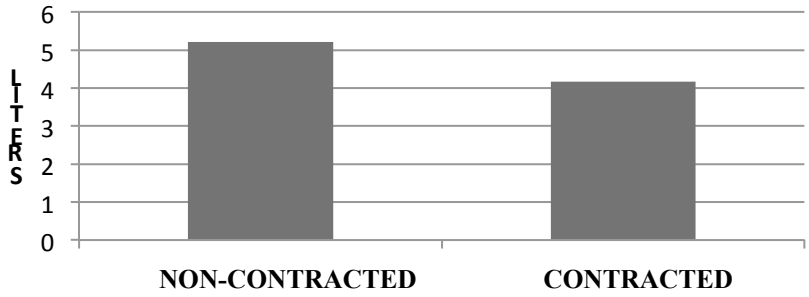

Figure 1. Vital capacity.

VC measured under NC 5.21 + .27 liters and was 111% of the predicted volume. The CC was 4.17 + .26 liters which was 89% of the predicted volume. This difference was significant ($F = 6.77$, $p < .01$). For VC the Omega Squared was 0.63 indicating that 62% of the variance was accounted for by the CC.

MVV was 127 + 12.2 l·min-1 NC conditions and was 109% of the predicted volume while CC conditions elicited a volume of 87.6 + 9.5 l·min-1 which was 89% of the predicted volume. This difference was significant (F = 6.44, p<.01). The Omega Squared was .62 indicating that 62% of the variance was accounted for by the CC. FVC was 5.0 + .27 l for NC which was 109% of the predicted volume while CC elicited a volume of 4.1 + .24 l which was 86.6% of the predicted volume. This difference was significant (F = 7.96, p<.01). The Omega Squared value was .70 indicating that 70% of the variance was accounted for by the CC.

RVO2 for NC was .39 + .01 l x m-1 x kg-1 and .52 + .03 l x m-1 x kg-1for CC. This difference was significant (F = 14.75, p<.01). The Omega Squared was .84 indicating that 84% of the variance was accounted for by the CC. With regard to carbon dioxide production, there was no significant difference inVC02 between NC and CC (p >.10)

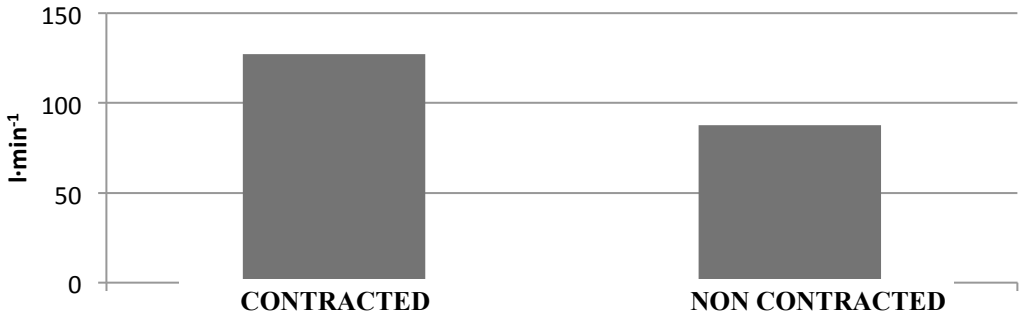

Figure 2. Maximum voluntary ventilation.

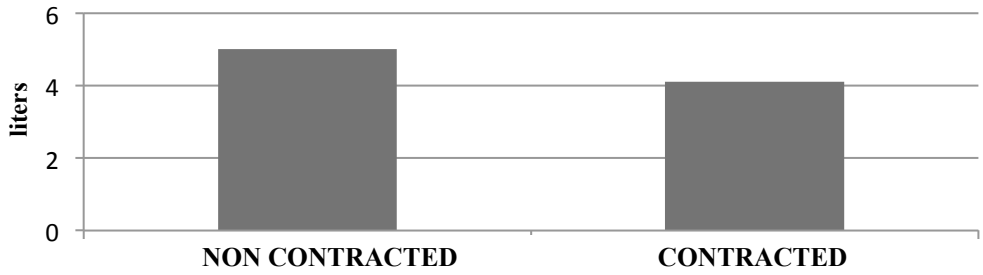

Figure 3. Forced vital capacity.

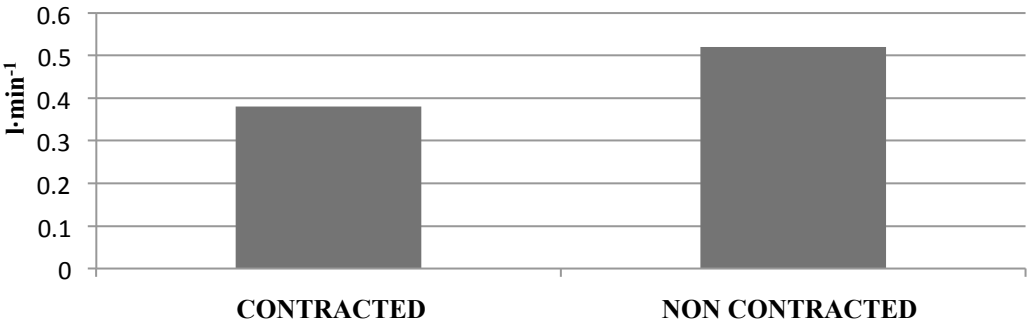

Figure 4. Resting oxygen consumption.

DISCUSSION

These results demonstrate that contracting muscles to streamline abdominal posture had negative effects on pulmonary functions and caused an increase in energy cost to maintain the posture while seated and not exercising in laboratory conditions. The statistical tests indicate that these differences would occur 99 times out of 100 if the experiment were repeated. The Omega Squared for the VC, MVV and FEV1 yielded a 62% to 70% chance that the change in the variance in these dependent measures was caused by the CC. However with the V02 measurements 84 percent of the change found in the variance in the dependent measures was accounted for by the experimental procedure. The CC produced pulmonary functions at less than predicted values while the NC produced pulmonary functions at greater than predicted values. This indicated that these muscular contractions could be detrimental to performance of crawl stroke swimming.

Karine and coworkers (9) found that swimmers' ventilation kinetics were dependent on the coordination between the diaphragm and the abdominal muscles. Swimmers had better coordination between the ribs motion and the thoracoabdominal volumes compared to other athletes. This indicated that swimmers have a more coordinated action between the abdominal muscles and diaphragm. These authors suggested that swimming practice leads to the formation of optimized breathing patterns in competitive swimmers that partially contributed to the larger lung volumes observed in swimmers. These findings provide evidence that the abdominal and erector spinae muscles should not be intentionally contracted during crawl stroke swimming.

We suggest that this posture increases the work of breathing given an increase in oxygen consumption when the abdominal and erector spinae muscles were contracted statically. Harms and co workers (7) found that as the work of breathing was artificially increased during exercise that leg muscle blood flow via vasoconstriction was reduced as well as oxygen consumption being decreased by 10% compared to control values. Gomez, and co workers (6) found that when respiratory muscles became fatigued after vigorous exercise of the abdominal muscles that there was a significant increase in fatigue of the respiratory muscles. We propose the contraction of the abdominal and erector spinae muscles during exercise causes a disruption of the coordination of respiratory muscles described by Karine and co workers (9)

and increases the work of breathing causing the decrements in blood flow described by Harms and others (7).

Toshimasa (14) found that the ability of the swimmers to roll their body around its longitudinal axis during front-crawl occurred as a result of forces developed by the center of buoyancy and that the buoyant force was the primary source of generating body roll. We speculate that restricting the ability of the swimmers' to fully ventilate their lungs would decrease the swimmers buoyancy and negatively impact body roll during crawl stroke swimming.

CONCLUSIONS

Contracting the abdominal and erector spinae musculature has not been shown to elicit a decrease in form resistance during swimming. In addition, muscle contractions proposed by Skinner (11) caused decrements in ventilatory function and increases in oxygen consumption even while the participants were seated and at rest. Harms and co workers (7) have shown that as the metabolic cost of breathing increased during exercise decrements in oxygen consumption occurred in the working muscles due to vasoconstriction. These muscle contractions used during the CC caused an increase in oxygen consumption and this oxygen could have only been consumed by the ventilatory muscles. Contraction of the ventilatory muscles could impair coordination between the diaphragm and abdominal muscles placing a further limit on pulmonary ventilation. We recommend not using the tactic of contracting abdominal muscles and erector spinae muscles to reduce form resistance on the body during crawl stroke swimming.

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