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RESPIRATORY CHEMOSENSITIVITY IN SYNCHRONIZED SWIMMERS AND SWIM-TRAINED WOMEN

by

John Andrew Taylor

A Thesis Submitted to the Faculty of the

DEPARTMENT OF EXERCISE AND SPORT SCIENCES

In Partial Fulfillment of the Requirements
For the Degree of

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In the Graduate College

THE UNIVERSITY OF ARIZONA

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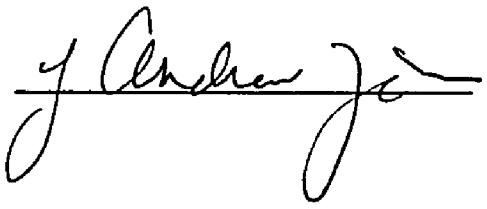
SIGNED: 

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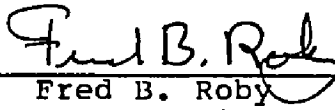

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ABSTRACT

This study investigated the role of the respiratory chemosensitivity in breath-holding in a group chronically exposed to hypercapnia. Nine synchronized swimmers and nine swim-trained control subjects performed CO2 rebreathing and repeated continous breath-holds. Each test was administered a second time with the subject immersed in water to determine the effect of increased inspiratory muscle work upon respiratory chemosensitivity. There were no significant differences in chemosensitivity between groups in the seated position. The synchronized swimmers displayed significantly longer breath-hold times and higher PCO2 values as compared to the control group in both seated and immersed positions. Chemosensitivity did not correlate with any breath-holding parameter. Immersion significantly reduced the chemosensitive slope in only the synchronized swimmers (from 1.Ø5 to Ø.67). From these data it was concluded that medullary chemosensitivity

is not related to BHT. It was postulated that the synchronized swimmers' extended breath-holding ability could be mediated by an adaptation at the inspiratory muscles.

CHAPTER 1

INTRODUCTION

The mechanisms involved in respiratory control have yet to be fully determined. No single control mechanism is involved but rather a complex system of afferent and efferent activity exists. Ventilation depends on many factors involving various receptors located in the lung tissue itself, in peripheral tissue, and centrally in the medulla, each receptor responding to a particular stimulus (81). If the receptor is exposed to a particular stimulus for abnormal lengths of time, an adaptation may result which increases the stimulus threshold needed to produce an action potential (24). Chronic hyperstimulation may result in a permanent change in the threshold level or in a transient change persisting as long as the receptor is chronically exposed to that stimulus (24).

Breath-holding induces hypercapnia which significantly lowers the pH in cerebro-spinal fluid (43). The lowering of pH stimulates the medullary

chemoreceptors which then send efferent signals to inspiratory muscles to increase ventilation (81). If these neurons are chronically stimulated, their responsiveness to changes in pH may decrease, resulting in less efferent activity to inspiratory muscles for a given partial pressure of carbon dioxide (PCO2).

The synchronized swimmer is a good example of a subject population which is chronically exposed to a stimulus and displays a reduced response to that stimulus. This is shown in their ability to breath-hold for long periods of time. This ability could be an adaptation to repeated exposure to a hypercapnic stimulus or just a genetic anomaly. Their unusual breath-holding ability may be the result of a decreased responsiveness to the chemical stimulus, or an increased ability to consciously over-ride the mechanical stimulus, or, most likely, a combination of the two.

Apneusis, or breath-holding, is a perturbation of the natural rhythm of the pulmonary system. The cessation of ventilation causes hypoxia and hypercapnia which increase efferent activity to the inspiratory muscles via medullary and peripheral chemoreceptors (81). This is coupled with the continuation of

rhythmic stimulation to the inspiratory muscles (23). During a normal ventilatory cycle the rhythmic stimulation is answered by an afferent response from stretch receptors and an afferent response in the form of muscular contraction (81). If this response is terminated by apneusis, the efferent stimulation builds in the muscle, reaching the point that ventilation can no longer be consciously overridden. This afferent feedback system is termed the mechanical stimulus for ventilation. The chemical stimuli, hypercapnia and hypoxia, increase the efferent stimulation to the inspiratory muscles. Greater chemical stimulation results in increased chemoreceptor afferent activity causing an increase in the efferent mechanical stimulus. However, during a single breath-hold, the chemical stimulus never reaches a level that will cause the termination of apneusis. This is shown in the observation that breath-holding may be continued after an exhalation and inhalation which does not change PCO2 or PO2 (27). This rebreathing manuever abolishes the mechanical stimulus while the chemical stimulus remains unaltered. If rebreathing were continued, the chemical stimuli would eventually reach the point that breath-holding would be impossible. As breath-holding

time increases, the chemical stimulus then also increases. Therefore, those with an increased breath-holding time could either have a decreased sensitivity to the chemical stimulus or be better able to override the mechanical stimulus resultant from the chemical stimulus.

The response of medullary neurons to a decreasing pH, induced by increasing PCO2, is to increase ventilation. As PCO2 increases, there is a linear increase in ventilation (65). This is attained by an increase in either ventilation rate or tidal volume (65). This ventilatory response to increasing PCO2 is termed respiratory chemosensitivity or carbon dioxide sensitivity. By this definition, a lowered chemosensitivity would mean a lower minute ventilation for the same PCO2.

In order to measure sensitivity to the carbon dioxide stimulus alone, Read (65) developed a procedure which involves rebreathing expired air to increase the PCO2 while maintaining PO2 well above the level that normally stimulates peripheral chemoreceptors (65). Investigators have used this procedure to compare the chemosensitive drive in different athletic groups; however, conflicting results (22,32,39,50,60) have been

reported. The response of Japanese pearl divers to this test has been of particular interest. They are renowned for their ability to remain underwater for extended periods of time and studies have shown that they have a decreased chemical drive during breath-hold (33) possibly related to a decreased chemosensitivity (49,74). Synchronized swimmers exhibit breath-holding ability comparable to that of the pearl divers. One might postulate that they too may demonstrate reduced chemosensitivity. Yet the only published data on synchronized swimmers (32) showed that these athletes demonstrated only a trend toward a reduced reponse when compared to control subjects.

The findings of Heigenhauser (32) do not support the data of those who have studied the responses of trained underwater divers (33,49,74) and others chronically exposed to a hypercapnic stimulus (22,39,70,71). If one group of subjects that is chronically exposed to a hypercapnic load during apneusis exhibit decreased chemosensitivity then would not a similar response be shown by synchronized swimmers who practice breath-holding manuevers on a daily basis? Synchronized-swimmers expend substantial energy during their breath-hold. Such exertion would

add to the hypoxic and hypercapnic burden, creating an even greater stimulus for ventilation. Hence one would expect to see a reduced chemosensitivity in these athletes. Clearly this area is in need of further investigation.

A possible explanation for the unique breath-holding ability of the synchronized swimmer may lie in the relationship of chemosensitivity to BHT during immersion. Immersion imposes a negative pressure load on the pulmonary system. This causes the inspiratory muscles to produce greater force for the same amount of ventilation. It has been shown, with negative pressure breathing, that the work output of the inspiratory muscles and end-tidal PCO2 are linearly related and that negative pressure loads decrease the response to CO2. Hence, immersion, by increasing work output of the inspiratory muscles with a positive pressure load, may decrease chemosensitivity resulting in greater breath-holding time (BHT), if BHT and chemosensitivity are linearly related.

Synchronized swimmers are unique in that their sport is one which involves strenuous work while under severe acid-base perturbation caused by apneusis. Their acid-base balance is taxed not only through the

production of lactic acid and carbon dioxide but also through the inability of the system to eliminate carbon dioxide via ventilation. They perform these manuevers over and over in competition and during training. With the repeated exposure to such a stimulus, an adaptation at the respiratory chemoreceptors may result in a reduced chemosensitivity, manifest in an increased breath-holding ability. However, if synchronized swimmers do exhibit a significantly lower chemosensitivity when compared to swimming controls, this apparent adaptation could be explained by a genetic predisposition toward blunted respiratory chemosensitivity. Nonetheless, synchronized swimmers provide a unique opportunity to study: 1) the effects of breath-hold training on chemosensitivity, 2) the relationship between breath-hold ability and chemosensitivity in individuals with a high breath-holding capacity, and 3) possible differences between breath-hold-trained and -untrained subjects in the effect of positive pressure breathing on BHT and chemosensitivity.

Statement of the Problem

Synchronized swimmers undergo rigorous breath-hold training during the course of a competitive season. The ventilatory response to increasing PCO2 (chemosensitivity) might be expected to be decreased in these athletes since they are chronically exposed to high levels of blood carbon dioxide. This training adaptation may also result in the ability of the synchronized swimmers to hold their breath for a longer period of time compared to non-swimming controls. The purpose of this study was to determine whether individuals who undergo intense physical activity while holding their breath under water have a lowered chemosensitivity and longer breath-holding time (BHT) as compared to control subjects. Chemosensitivity was compared to BHT to determine if chemosensitivity could be the primary determinant of breath-hold time. A secondary purpose was to determine if negative pressure from immersion in water decreased chemosensitivity.

The specific aims of the study were:

1. To determine if chemosensitivity and breath-holding time are significantly different in synchronized swimmers as compared to swim-trained controls.
2. To determine if breath-hold time is related to chemosensitivity.
3. To determine if negative pressure breathing decreases chemosensitivity.

It was hypothesized that:

1. Synchronized swimmers would exhibit significantly decreased chemosensitivity and increased BHT as compared to controls.
2. There would be an inverse relationship between BHT and chemosensitivity, stronger in the synchronized swimmers compared to controls.
3. There would be a decrease in chemosensitivity with immersion in both groups.

Significance of the study

This research could provide information on the physiological relationships between chemosensitivity and BHT. Also, the importance of the chemical stimulus to exercise ventilation and the adaptibility of the sensitive medullary areas could be shown in the marked difference in breath-holding abilities between synchronized swimmers and control subjects. If the carbon dioxide stimulus is attenuated by an adaptation, then the mechanical stimulus should become the overriding factor in breath-hold capacity.

CHAPTER 2

LITERATURE REVIEW

As early as 1905, with Haldane and Priestly's classic inquiry into the control of ventilation, pulmonary physiologists have been interested in the chemical control of respiration. Haldane and Priestly attributed the regulation of respiration solely to carbon dioxide pressure at the respiratory centers (30). Since then, physiologists have determined that there is a much more complex interaction between effectors and controllers of the respiratory system. In fact, many physiologists doubt that hypercapnic stimuli directly affect respiration and argue that if carbon dioxide were a main controller then there would have to be either a venous or lung chemoreceptor (16). There is no evidence for chemoreceptors on the venous side, but there are arguments supporting the possibility of lung receptors responsible for chemoreception (78).

Chemoreceptors

There are three recognized chemoreceptive regions responsible for the ventilatory responses to hypercapnia and hypoxia. The carotid bodies, located at the bifurcation of the carotid artery, and the aortic bodies, located in the aortic arch, are the peripheral receptors which are primarily sensitive to oxygen levels. The carotid bodies have been well characterized because of their easy accessibility, larger size, and vascular isolation, whereas the aortic bodies have only been studied by eliminating their afferents through denervation (15). The carotid bodies respond primarily to PO2 but also play a role in the physiological response to PCO2 and pH (15,61). They have been shown to respond to oscillations in PCO2 (5) and possibly account for some of the response to hypercapnia (15,45).

Most of the response to hypercapnia is thought to originate at the central chemoreceptors. Leuson (43), in 1950, observed changes in ventilation with perturbation of acid-base levels in cerebro-spinal fluid. Later, Loeshke (44) discovered the particular chemosensitive regions in the fourth ventricle of the

medulla. The medullary chemoreceptors respond to a hydrogen ion change resulting from a carbon dioxide stimulus in tissue fluid in contact with these receptors. Alveolar ventilation has been shown to be a linear function of the pH at this site (62).

The chemoreceptors in these three regions provide feedback to the respiratory controller. The peripheral chemoreceptors send their afferents through the vagus (61) and have a rather rapid response to ventilatory perturbations due to their location (45). Although they are in the same medullary region, the central chemoreceptors are anatomically separate from the medullary respiratory center and their afferent pathway has not yet been discovered (81). Even though there is an extended medullary chemoreceptor response time (45), hypercapnia does cause an immediate increase in output to the inspiratory muscles to increase ventilation (15,30,44,52,61). This same ventilatory response is evident in hypoxia. When both hypercapnia and hypoxia are present together, there is an additive effect in the ventilatory response (15,45,61). Evidence from studies on patients with carotid body resection shows that the carotids contribute only 10%-30% of the response to hypercapnia (79).

Pappenheimer (62) showed almost no contribution from the carotid bodies in animals. Also, hypercapnia causes cerebral vasodilation resulting in enhanced diffusion of CO2 across the blood-brain barrier and, because it contains much less protein, the buffering capacity of the cerebral spinal fluid is less than the blood (81); therefore, the available PCO2 stimulus is sufficient to stimulate the central receptors. Although the carotid bodies may respond more to breath-by-breath deviations in PCO2 because of their fast response time (15,61), the central chemoreceptive region is the primary controller of ventilation in chronic and extended acute bouts of hypercapnia (31).

Exercise and Chemoreception

Exercise creates a hypercapnic load on the pulmonary system and can severely decrease blood pH. Many pulmonary and exercise physiologists have been interested in the effect this has on the respiratory control system and the control of homeostasis during this perturbation. Exercise ventilation has been divided into two responses: the fast response which attributes for the immediate increase in ventilation

with the onset of exercise and the slow response which takes over as intensity increases and matches the metabolite production. The fast response is most likely neurogenic in nature. Increased ventilation has been demonstrated in animals with active and passive limb movements (1,77). There is also argument that perhaps a neurogenic drive arising from group III and IV afferents accounts for some of the slow reponse (77).

The slow response has been said to be humorally driven. Central and peripheral chemoreceptors respond to the metabolic acidosis and increase their afferent activity to the medullary respiratory center which increases efferent activity to inspiratory muscles. This theory has always been a source of contradiction because arterial blood pH, PCO2, and PO2 are regulated well within resting values during exercise (19,76). So, there must be no change in stimulus at the chemoreceptors. It is only when exercise intensity is heavy that there is any possible increase in chemoreceptor stimulus. There are some investigators who contend that there is a pulmonary chemoreceptor

responsible for the close matching of Ve and VCO2 (78,29). There is very little supporting evidence and also evidence refuting these claims (16,76).

Characterization of central chemoreceptor sensitivity by CO2 rebreathing has allowed researchers to investigate the role of the carbon dioxide stimulus during exercise and in adaptations caused by training. The methodology for determination of chemosensitivity, evidence of altered chemosensitivity during exercise and after training, and arguments both for and against the rebreathing test as a measure of medullary chemical control will be discussed in the following section.

Measurement of Chemosensitivity

In 1966, D. J. C. Read (65) developed a standardized rebreathing method for assessing the ventilatory response to carbon dioxide, enabling quantification of the chemical/humoral drive of respiration. The closed loop system allows for expired carbon dioxide to be constantly returned to the lungs and therefore the PCO2 of all body fluids progressively rises. An initial high concentration of oxygen within the system eliminates any hypoxic stimulus.

Read's initial results showed a linear increase of the carbon dioxide stimulus (VCO2) with time, resulting in increases in minute ventilation (Ve) and tidal volume. In order to determine possible influences of acute depletion of body carbon dioxide stores upon chemosensitivity, subjects hyperventilated prior to rebreathing. This resulted in a modification of the increases in ventilation, tidal volume, and end tidal PCO2 with time, but did not alter the slope of Ve/VCO2 during rebreathing. Artificial changes in carbon dioxide storage capacity and metabolic production rate were introduced by increasing the size of the rebreathing bag. A statistically significant shift in the response curve was affected but the slope, again, was unchanged.

Although the association of alveolar ventilation to change in PCO2 is dependent upon the metabolic rate, carbon dioxide storage capacity, and regional buffering capacity in the chemosensitive area (65), Read's procedure has been validated as an indirect measure of central chemosensitivity. Barcroft and Margaria (4) found that inspiratory flow rate increased linearly with PCO2. It has also been found

that, for a given carbon dioxide percentage in the inspired air, there is a constant mean inspiratory flow rate (58).

Several studies have used occlusion pressure (P0.1) to validate their use of the rebreathing method. This is the pressure developed at the mouth within the first 100 milliseconds of occluding airflow during inspiration and is an indirect measure of the neurogenic drive to breath (52). Maranetra and Pain (47) found a close association between P0.1 during rebreathing and the chemosensitive slope. Yet, P0.1 during ambient air breathing does not correlate with either hypoxic or hypercapnic ventilatory response (54).

Chronic obstructive pulmonary disease provides a patient population which can be used to validate the rebreathing test. Victims of this disease are unable to completely ventilate the lung so that there is an abnormally large residual volume and chronic hypercapnia. This chronic stimulation of the central chemoreceptors results in a decreased response to CO2 rebreathing in these individuals (47). Decreased reponsiveness to hypercapnia is also demonstrated in

another patient population characterized by a hypoventilatory central regulatory abnormality (51).

There is much inter-individual variability with the Read method. Body size has been shown to have an effect upon the response. There is a lower response in smaller individuals (54) which may also account for the lower response in women as compared to men (63). Women also have an intra-individual variability, exhibiting a decrease during the luteal phase of menstruation (72). Some investigators have demonstrated reduced chemosensitivity with increasing age (37,64), while others have shown no difference in central responsiveness between infants and adults (3).

Chemosensitivity in Athletic Populations

The responses of patient groups show that adaptations at the central chemosensitive areas can occur. Also, the response in the hypoventilatory abnormality demonstrates that the chemosensitive medullary areas play some role in controlling ventilation. It is evident from the P0.1 studies that the rebreathing manuever increases efferent activity to the inspiratory muscles and therefore does test the

sensitivity of the chemoreceptors. But, the results from studies using PØ.1 do not implicate carbon dioxide as a primary controller of respiration.

Endurance Trained Athletes

Many investigators have shown a decreased chemosensitivity in endurance trained individuals (11,39,66,72). Byrne-Quinn and colleagues (11) reported a decreased responsiveness to both hypoxic and hypercapnic stimuli in 13 athletes compared with nonathletes. The athletes' hypercapnic response was 47% of the response present in controls. Based on their results and studies which show a relationship between hypoxic and hypercapnic responsiveness (75), their conclusion was that there is decreased peripheral chemoreceptor contribution to the hypercapnic response in athletes. Schoene et al (72) showed significantly lower carbon dioxide chemosensitivity in endurance trained females. In one of the earliest investigations in this area by Rebuck and Read (66), endurance athletes had response slopes significantly lower than

average while sprinters had a tendency toward a higher ventilatory reponse to CO2 compared to untrained subjects.

These results have not been reproducible by other investigators. Scoggin (73) and Mahler (46) did not show decreased response in endurance trained runners, although the hypoxic response was decreased in Scoggin's study. Also, training studies have not had consistent results. Kelley (39) had varsity rowers detrain while a group of previously sedentary subjects trained 5 days per week for seven months. At the end of the training period, the trained subjects had increased responsiveness while the detrained subjects showed a slight insignificant decrease. Blum's (6) subjects were also previously untrained but, after training, had a decreased response. In a third study, training did not produce any change in sensitivity (8). These subjects were unfit prior to training but did not acheive a high degree of fitness after training. A high level of fitness (31) and/or a high level of performance (69) have been indicated in being necessary to demonstrate a difference in response to hypercapnia between trained and untrained individuals.

Some investigators have shown a positive correlation between resting hypercapnic ventilatory response and exercise ventilatory response, especially in athletic populations (11,46,48). Mahler et al (46) studied twenty accomplished marathon runners and compared their CO2 sensitivity to a control group. There was no difference between the two groups in chemosensitive response, but the runners' exercise ventilatory response (Ve/VCO2) did correlate with their rebreathing response. Both endurance and non-endurance athletes studied by Martin and co-workers (48) and non-athletes studied by Rebuck (66) had hypercapnic responses which correlated with Ve/VCO2 during exercise. From these results, some researchers have concluded that, at rest, carbon dioxide does not play a major role in ventilatory control, but, its increase with exercise accounts for much of the exercise hyperpnea (46,80). A decreased responsiveness might produce less dyspnea and enhance exercise tolerance (48,73).

Others have been reluctant to apply this model to exercise (19,51). Recently, Menitove (51) postulated that ventilation during exercise and during CO2 rebreathing test complimentary functions and are

not two tests of the controller of ventilatory homeostasis. His assertion was that exercise ventilation is a response to transients in metabolic rate whereas the CO2 rebreathing procedure tests the body's defense mechanism to supernormal deviations of CO2. This idea was based on the lack of correlation he found between exercise ventilation and CO2 sensitivity in a patient group. These patients displayed obesity hypoventilation syndrome characterized by a severely reduced response to carbon dioxide possibly caused by a central regulatory abnormality (51). These subjects exhibited no significant difference in exercise response from the control group as would be expected if CO2 sensitivity is a predictor of the slope of the change in ventilation over the change in VCO2 during exercise.

Swimmers and Divers

Although the results supporting exercise training as a means of reducing chemosensitivity are equivocal, decreased responsiveness has been repeatedly demonstrated in those chronically exposed to hypercapnia (47,50,55,59,70,71,74). Japanese Amas earn

their livelihood by harvesting the ocean floor. There are two types of Amas: Funado, who use weights and an assistant who aids them in ascent so that they can dive deeper and Kachido, who are unassisted and dive to much lower depths. Early studies of the Japanese diving women measured the alveolar gas changes during descent and ascent (35). Most important in affecting a central chemosensitive adaptation would be the marked increase in alveolar PCO2 during descent (35). Evidence indicates that the Funado do exhibit an adaptation and the Kachido do not (49,50,55). Only one study has shown a reduced response in Kachido (36), but the method for determining chemosensitivity was not Read's (65). Instead, Ve plotted over PCO2 during breathing of room air and of 15% CO2 was used to derive a slope. Results using Read's chemosensitivity test have indicated no difference in Kachido and controls (50,55). A possible explanation for decreased sensitivity only in the Funado is that they dive deeper and longer than the Kachido. Therefore, the Funado is exposed to a more hypercapnic and hypoxic condition.

Other divers have demonstrated a decreased sensitivity to carbon dioxide. Schaefer (70,71) investigated the hypercapnic response of Naval

submarine escape instructors. The determination of chemosensitivity involved steady-state breathing of various concentrations of carbon dioxide. A significant difference between the instructors and controls was shown. Subsequently, these results were validated by Florio (22). His study of Navy divers utilized Read's (65) rebreathing method and demonstrated a slope 33% lower than controls. Interestingly, both Schaefer (70) and Florio (22) as well as another investigator (30) found correlations between breathing pattern in divers and chemosensitivity. Those divers who had a large tidal volume and slow respiratory rate tended to have a low CO2 sensitivity. Their conclusion was that this correlation was due to a development of the inspiratory muscles from prolonged periods of negative-pressure breathing (22,70). The effect of negative pressure breathing upon the ventilatory response to hypercapnia will be discussed the section on immersion.

Swimmers represent an unique population when investigating the effect of training upon chemosensitivity. Not only may training have an effect upon their responsiveness, but their controlled breathing may also play a role in adaptation. Two

studies in particular have looked at their CO2 sensitivity. Ohkuwa (60) tested untrained subjects, long-distance swimmers, and sprint swimmers. Both groups of swimmers had a significantly lower response than the controls and there was an even lower response in the long-distance swimmers, although statistically insignificant. Saunders (69), however, found no difference in teenage swimmers.

One study in particular is pertinent to this investigation. Heigenhauser (32) compared three groups of swimmers. Synchronized, speed, and recreational swimmers were tested for chemosensitivity and exercise ventilatory response. His comparison of the ventilatory reponse to leg and arm exercise did not show any significant differences betweeen the three groups, but, most importantly, there were no differences in chemosensitivity between any group. He concluded that the synchronized and speed swimmers are analogous to the Kachido divers in that they are not subject to a great enough hypercapnic burden to cause a central adaptation.

It is still not clear whether chemosensitivity is determined by genetic factors or training or a combination of both. If reduced chemosensitivity aids

performance in particular athletic events, a predisposition to perform well may precede any adaptation. The conflicting results from training studies have not elucidated the relationship between training and chemosensitivity (6,8,39). Saunders (69) found a close relationship in CO2 sensitivity between swim-trained teenagers and their siblings. Scoggin (73) found significant correlations between hypoxic responses in siblings of both endurance-trained and untrained individuals. Conversely, Igarashi (36) presented evidence supporting the adaptability of the chemoreceptors to chronic hypercapnic stimulation. The Amas in his study had decreased their chemosensitivity after the harvesting season when they are most often exposed to hypercapnic and hypoxic stimuli.

Breath-holding

Most of the literature on chemosensitive adaptations with training presented in the previous section discusses training in its traditional sense: endurance or sprint training. The subjects in this study were synchronized swimmers: endurance swim-trained, but also trained in breath-holding.

Breath-holding is a unique perturbation of the respiratory system. Its control and effects will be reviewed in this section.

Although the chemical stimuli - carbon dioxide and oxygen - play pivotal roles in modulating ventilation, they can be overridden. The respiratory system is unique from other life-sustaining systems in that it can be brought under conscious control. Holding one's breath overrides the hypoxic and hypercapnic afferents as well as the respiratory 'rate meter' located at the medullary respiratory centers. In the first minute of a breath-hold, arterial PCO2 increases 6 to 8 mm Hg, arterial PO2 falls from about 100 to 40-50 mm Hg (15), and the efferent nervous activity in the inspiratory muscles builds up in ramp-like fashion (23,26,27).

The limiting factor in breath-hold time (BHT) is a combination of these mechanical (neural) and chemical (humoral) stimuli to breath. The breaking point is a result of the increasing chemical stimulus which, in turn increases the mechanical stimulus. For a given increase in PCO2 there is a linear increase in ventilation (58). Thus, during a breath-hold, the increasing PCO2 potentiates the efferent activity from

the medullary respiratory center while the respiratory rhythm generator continues to send efferent activity to the inspiratory muscles (23,81). The role of PO2 is more difficult to assess since the hypoxic effect on BHT is alinear (20). Hypoxia would provide a third time dependant stimulus, summated with PCO2 and the mechanical stimuli (26).

Early work done in this area by Fowler (23) showed that if, at the end of a single breath-hold, a few breaths of a gas mixture which did not change the blood gases was allowed, then further breath-holding was possible. By allowing the lung to ventilate, the mechanical stimulus was abolished. But, the second breath-hold is always shorter due to the greater chemical stimulus at the onset (23). From this he concluded that the breaking point of a breath-hold is the point at which a chemically induced ventilation urge, which corresponds to a certain magnitude of alveolar ventilation, exceeds the conscious inhibitory factors (23).

There is possibly a second mechanical factor which is independant of the chemical stimulus. Cain (12) has stated that the stretch afferents are slow adapting and the long breath-holding time could allow

them a determining role. However, Muxworthy (56) showed that the relationship between lung volume and BHT was linear. Therefore, the breath may be held for a longer time at total lung capacity than at residual volume even though there is a greater stretch stimulus. The decreased hypercapnia and hypoxia due to larger lung volumes must, therefore, over-ride the increased neural output from stretch receptors.

It has been demonstrated that the chemical stimulus does not reach its threshold in a single breath-hold (as opposed to breath-holds after rebreathing) (23,27). Godfrey and Campbell (27) postulated that the PCO2 at which breath-holding is impossible is usually about 70 to 80 mm Hg. Their experiments in which this level of hypercapnic stimulus was used resulted in BHT of one or two seconds, making it impossible to distinguish between breath-holding and rebreathing.

Psychological factors also play a role in determining BHT. Breath-holding time can be increased by informing the subject of time or by giving incorrect time, ie. 5 instead of 7 seconds etc. (40). With practice, breath-holding becomes easier and can be maintained longer. Koboyashi (40) eliminated the

hypoxic stimulus and measured the time and end-tidal CO2 of daily breath-holding trials. Breath-hold time increased with repetitive daily trials but the end-tidal CO2 did not decrease. Since the chemical stimulus did not decrease, he attributed the longer BHT to an increased will power.

Although will power cannot be discounted as a determinant of breath holding time, there are adaptations with long term breath-holding other than psychological. Physiological adaptations to breath-hold diving have not been absolutely proven, but there is evidence linking adaptive pulmonary changes to chronic breath-hold training. Breath hold divers have been shown to have increased vital capacities in comparison to others (70,71,73) and after training (13). An increased lung volume would increase BHT by decreasing the time for the chemically induced ventilation urge to exceed the conscious over-riding effort. Honda et al. (33) quantified the contribution of chemical drive to the BHT in Amas. No significant differences were found between controls and the Amas in

either BHT or PCO2 drive during breath-holding. Although it is not unequivocal, there is evidence to suggest adaptive pulmonary changes with breath-hold training.

Effect of Negative Pressure Breathing

Since synchronized swimmers perform breath-holding while immersed, the effects of immersion upon chemosensitivity and breath-holding ability may be pertinent. Immersion imposes two restrictions upon the pulmonary system. The first is an increase in central blood volume which decreases the lung capacity and the second is an inspiratory flow resistive load caused by the restriction of the chest wall (2,10,67). Both of these may affect the response of the pulmonary system to PCO2.

It has been well established that immersion decreases the vital capacity without changing residual volume. The amount of decrease in vital capacity varies between 6 and 15 percent (2,10,14,67), with individual variations most likely dependent upon blood volume and distensibility of the chest wall. There are two mechanisms by which this decrease could be

mediated. When immersed to the neck, the inspiratory muscles are opposed by a hydrostatic pressure of approximately 16 cm H_2O which may limit the inspired volume (34), and there is central vascular engorgement caused by a shift of blood into the thorax from the extremities, decreasing lung compliance (17). The relative contribution of these two factors has not been completely elucidated.

The effect of this decrease in vital capacity on chemosensitivity and breath-holding can only be conjectured since no work has been done in this specific area. If BHT is a linear function of lung volume (56), then the decreased lung volume due to immersion should decrease BHT. Chemosensitivity should be unaltered since there is no correlation between lung size and the slope of Ve/PCO2 (54). However, immersion may result in changes in chemosensitivity because of inspiratory flow resistance from hydrostatic pressure upon the rib cage.

During immersion, distension of the chest wall forces the inspiratory muscles to work at a disadvantageous length (67) while hydrostatic pressure introduces an added load to the inspiratory effort (2,10). Both of these factors would increase the work

of breathing. Milic-Emili (53) found that the work output of the inspiratory muscles and end-tidal CO2 tension were linearly related. His results led him to the conclusion that carbon dioxide levels directly control the activity of the inspiratory muscles and that the activity of the expiratory muscles is only coincidentally involved. Flenley (21) concurred with this assessment of the role of carbon dioxide in inspiratory control. If their results are indicative of the relationship between the work of breathing and CO2 levels then one would expect increased chemosensitivity with immersion.

Flenley and Milic-Emili used positive pressure breathing in their tests and immersion is analogous to negative pressure breathing. During immersion, the inspiratory muscles have to work harder for the same amount of ventilation whereas positive pressure breathing decreases the amount of work needed. With this increase in force production, afferent feedback from the contracting muscle should be increased. More afferent feedback would generally indicate increased ventilation. So, the respiratory rate-generator would decrease its output because of the higher afferent input. One example of this is emphysema, a restrictive

respiratory disease which presents its victims with a continuous negative pressure load (9). These subjects have been shown to have a decreased response to carbon dioxide which can be increased after administration of a bronchodilator (9). When the work of breathing was reduced in normal subjects with the use of helium as a carrier gas, the response to hypoxic hypercapnia was greater than when nitrogen was the carrier gas (25). Other studies using both animals and normal humans (7,68) have demonstrated reduced ventilatory response to CO2 when a resistance is added to breathing.

The conclusion is that, with immersion, the increase in the work of breathing would result in a decreased chemosensitivity. This would not apply to BHT. Since breathing is suspended, only the decreased lung volume would affect the ability to breath hold. Consequently, BHT should also decrease with immersion.

CHAPTER 3

METHODOLOGY

The subjects consisted of 9 synchronized swimmers and 9 female controls matched for height and weight. The average age of the synchronized swimmers was 19 years and the controls averaged 24 years. The difference in age was not deemed large enough to have a significant effect upon the results. The synchronized swimmers had been competing for an average of nine years and had been training continously for the previous three months. The female control subjects had been swim training for at least three months prior to testing. The rationale for using swim-trained controls was to isolate the effects of breath-hold training from those of swim-training.

All testing was done two hours post-prandial and neither caffeine nor other stimulants had been ingested two hours prior to testing. Subjects performed rebreathing and breath-holding tests on separate days at approximately the same time. No attempt was made to control for phase of menstruation.

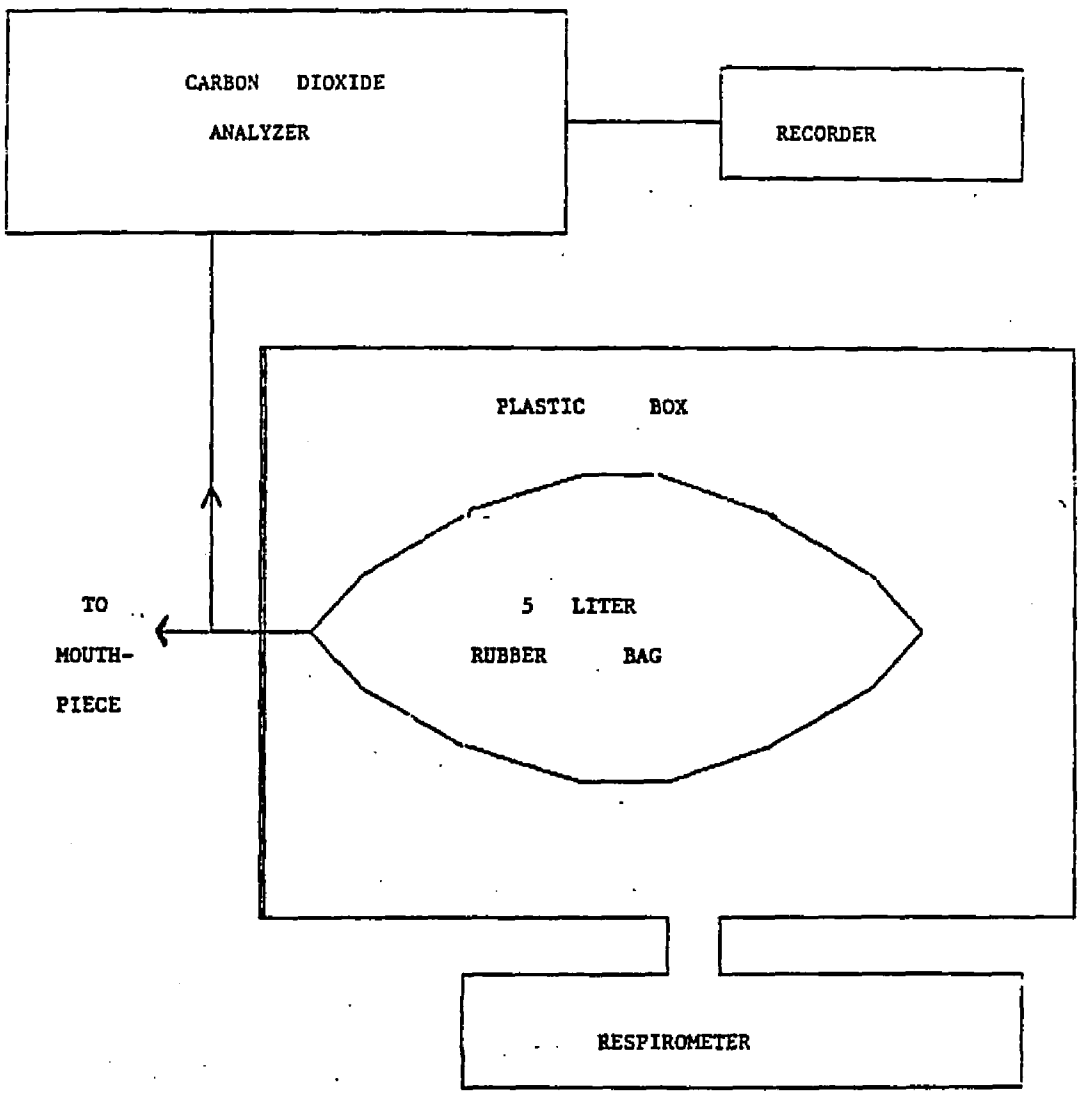
A Collins 620 DS respirometer unit was used for lung function tests. These included forced vital capacity (FVC), total lung capacity (TLC), forced expiratory volume in 1 second (FVC_1), maximal ventilatory volume (MVV), and determination of residual volume (RV).

Chemosensitivity

To determine chemosensitivity, the rebreathing method of Read (65) was used (Fig 1). A five liter rubber anesthesia bag was filled with 7% CO2 and 93% O2. End-tidal CO2 was sampled using an Applied Electrochemistry CO2 analyzer calibrated before and after each test with 16.2% O2 and 3.93% CO2. The analyzer interfaced with a Texas Instruments strip chart recorder so that a recording of CO2 levels could be obtained. The anesthesia bag was enclosed in a plexiglas box which was in turn connected to a 9 liter Collins respirometer. Exhalation displaced air within

Figure 1

Schematic of Rebreathing Apparatus



the plexiglass box which, in turn, moved the bell of the respirometer allowing ventilation volume to be measured.

The rebreathing procedure was performed a total of three times, once as an introduction to the system and procedures and once during both seated and immersed trials. Prior to performing the rebreathing manuever, the subject breathed ambient air for five minutes so that depth and rate of respiration would be normal. The subject was instructed to exhale to residual volume before rebreathing so that bag gas and blood gas equilibrated as quickly as possible. Rebreathing continued for 4 minutes. The rebreathing test was performed by each subject a second time on a different day while immersed in water. The subjects were seated in 38^{o} C water up to the clavicle so that the chest was fully immersed.

To determine the chemosensitive reponse, end-tidal PCO2 (P_ACO2) was plotted against ventilation volumes (Ve) at half minute intervals (65). The CO2 sensitivity of the individual was obtained from a statistical analysis of the relationship, $S= Ve/P_ACO2$.

Breath-holding Time

The apparatus used was similar to that used for the rebreathing method described above (65). The five liter bag was filled with 7% CO2 and 93% O2. The respirometer was not connected to the anesthesia bag system since volume measurements were not needed. End tidal CO2 was measured by an Applied Electrochemistry CO2 analyzer and recorded by a Texas Instruments strip chart recorder. Each subject performed the test three times, once as an introduction to the procedures and once during both seated and immersed trials.

The subject was seated and breathed ambient air for five minutes, then exhaled completely, and inhaled the bag gas. The first breath-hold and each successive breath hold was maintained to the breaking point. At breaking point, subjectively determined by each subject, the subject exhaled into the bag and re-inhaled to breath-hold again. This continued until the PCO2 reached such a level that breath-holding was impossible. The test was ended either when the subject determined she could breath-hold no longer or when successive breath-holds were five seconds or less

apart. Breath-holding using the above procedure was performed a second time while immersed in 38° C water up to the clavicle.

Repeated breath-holds facilitate isolation of the mechanical stimulus to breathe from the chemical stimulus. A single ventilation after a breath-hold, without a subsequent change in gas content, abolishes the mechanical stimulation to breathe without decreasing the chemical stimulus, i.e. the PCO2 in the blood. Therefore, the chemical stimulation is increased with each subsequent breath-hold. The elapsed time (BHT_i) and the PCO2 of the first breath-hold and ($PCO2_i$) were used to assess the relative sensitivity to or strength of the mechanical stimulus which is thought to determine 'true' breath-holding ability. The PCO2 reached by each subject above which further breath-holding was impossible ($PCO2_f$) and the total time of breath-holding procedure (BHT_f) were used to indicate the ability to withstand hypercapnic stress.

Statistical Analysis

Differences between seated and immersed chemosensitivity and breath-holding trials were determined with a dependant student's t-test. Differences between groups in chemosensitivity and breath-holding variables were determined with an independant students' t-test. To determine the relationship between chemosensitivity and breath-holding, linear regression analysis was used to find the relation between breath-holding variables, ($PCO2_i$, BHT_i, $PCO2_f$, and BHT_f) and chemosensitive slopes. Linear regression was used within an intervention only, so that the variables for seated breath-holding were compared with the seated chemosensitive slope only and immersed breath-holding variables were compared with the immersed chemosensitive slope only. Correlation and regression analysis between initial BHT and PCO2 and final BHT and PCO2 were also determined.

CHAPTER 4

RESULTS AND DISCUSSION

Subject characteristics are shown in table 2. There was no difference in height or weight between the two groups. The control group did have a higher mean age, but this difference was not large enough to have a significant effect on the parameters measured. Pulmonary function tests showed that there was no difference between the synchronized swimmers and the controls in forced vital capacity (FVC), forced expiratory volume in one second (FEV_1), or maximal ventilatory volume (MVV). There was a significant difference in total lung capacity (TLC) and residual volume (RV) (table 1). The synchronized swimmers had a higher TLC (5529 $\pm$ 490 ml.) than the control group (4966 $\pm$ 604) and a higher RV (1373 $\pm$ 421) than the controls (1008 $\pm$ 165); however, differences in lung volume have been shown to have no effect upon hypercapnic response slope (54).

TABLE 1

Pulmonary Characteristics of Subjects
(values are mean + S.D.)

	FVC(L)	FEV1(L)	TLC(L)	RV(L)	MVV(L)
SYN SWIM	4224	3727	5529*	1373*	137.6
(n=9)	335	246	490	421	16.5
CONTROL	3952	3552	4966	1008	125.0
(n=9)	481	422	604	313	17.9

TABLE 2

Physical Characteristics of Subjects
(values are mean + S.D.)

	AGE(yr)	HT(cm)	WT(kg)	YRS IN TRAIN
SYN SWIM	19.0	162.6	53.6	10.0
(n=9)	1.6	5.1	3.6	2.7
CONTROL	23.5	162.6	54.0	1.8
(n=9)	3.3	5.1	5.4	3.0

* $p < 0.05$ vs. control

Chemosensitivity

Comparison of the chemosensitive slopes from the synchronized swimmers and control subjects showed no difference between the two groups (Table 3). Although the mean slope for the synchronized swimmers (1.05 ± 0.18) was lower than the mean slope for the control group (1.26 ± 0.66), this difference was not statistically significant. This finding is consistent with data of Heigenhauser et al. (32) who showed no difference between speed, synchronized, and recreational swimmers. Similarly, his data showed a statistically insignificant lower sensitivity in the synchronized swimmers. However, his data demonstrated a much higher and more variable chemosensitive slope in the synchronized swimmers than was found in this study. Heigenhauser's synchronized swimmers displayed responses in the range of those found in the controls of this study. The discrepency between the synchronized swimmers' response in Heigenhauser's study and the present study is inexplicable since both groups were of comparable experience and level of competition.

TABLE 3

Chemosensitivity^ of subjects

SYNCHRO SUBJECT#	DRY CHEMO	WET CHEMO
1	1.17	.75
2	1.00	.87
3	1.40	.66
4	1.00	.84
5	.88	.50
6	.87	.94
7	1.00	.32
8	.82	.87
9	1.27	.32
$\bar{X}$	1.05	.67
S.D.	.18	.23
CONTROL SUBJECT#		
1	1.31	.50
2	.77	1.09
3	.87	.93
4	2.89	1.05
5	1.76	.98
6	.60	.61
7	1.11	1.68
8	.88	.80
9	1.17	1.43
$\bar{X}$	1.26	1.01
S.D.	.66	.35

^ described by the slope Ve/PCO2
* $p<0.025$ vs. control

The control group showed much more variability than the synchronized swimmers in their chemosensitive slope. It is postulated that this wide variability in the control subjects may have masked a difference between the two groups. Also, the homogeneity of the synchronized swimmers' response could reflect either an adaptive mechanism to breath-hold training or a selective elimination of those with high chemosensitivity.

Breath-holding Time

There were significant differences between the swimmers and control groups in all the parameters measured during breath-holding (Table 4). The initial breath-hold time was longer in the synchronized swimmers, 1.7 $\pm$0.8 minutes compared to 0.9 $\pm$0.4 minutes in the control group ($p<0.025$). Also, the PCO2 at the end of that initial breath-hold was higher than that of the controls (56.3 versus 49.2 mmHg; $p<0.025$). At the end of the series of breath-holds, the synchronized swimmers displayed greater ability to withstand the stress of hypercapnic breath-holding. The total breath-holding time (5.8 versus 3.2 min; $p<0.025$) and

the final PCO2 (79.6 versus 65.6 mmHg; $p<0.025$) were longer and higher, respectively, in the synchronized swimmers.

It had been thought that there would be a linear relationship between chemosensitivity and breath-holding time; but, there was no correlation between chemosensitivity and initial breath-hold time (BHT_i), initial PCO2 ($PCO2_i$), total breath-holding time (BHT_f), or final PCO2 ($PCO2_f$) (Table 5). It appears that the initial breath-holding time was determined not by the end tidal PCO2 but by the amount of mechanical efferent activity generated by the PCO2 stimulus and the relative ability to withstand the mechanical stimulus to breathe. If chemosensitivity were the major determinant of breath-holding ability, then a lowered chemosensitivity would have resulted in less efferent activity and, thus, longer breath-holding time. Since no correlation was found in either group or by pooling the groups, it is concluded that the greater breath-holding ability of the synchronized swimmers was not due to a central regulatory difference.

TABLE 4

Values for Breath-holding Parameters
(mean + S.D.)

INITIAL

	SEATED		IMMERSED	
	TIME (min)	PCO2 (mmHg)	TIME (min)	PCO2 (mmHg)
SYN SWIM (n=9)	1.7*	56.3*	1.7*	56.3*
	.8	3.2	.7	4.2
CONTROL (n=9)	.9	49.2	1.0	50.5
	.4	3.3	.5	3.6

FINAL

	SEATED		IMMERSED	
	TIME (s)	PCO2 (mmHg)	TIME (s)	PCO2 (mmHg)
SYN SWIM (n=9)	5.8*	79.6*	5.5*	78.2*
	.4	5.6	.9	5.6
CONTROL (n=9)	3.2	65.6	3.2	61.2
	.9	5.4	1.0	4.3

* $p < 0.025$ vs. control

TABLE 5

Correlations between Breath-holding Variables for Pooled Subjects (n=18)

	CHEMO	BHTi	PCO2i	BHTf	PCO2f
CHEMO		-.29	.01	-.28	-.13
BHTi			.57	.59x	.54
PCO2i				.68*	.85*
BHTf					.81*
PCO2f					
WET CHEMO		-.30	-.10	-.34	-.29
WET BHTi			.23	.56	.38
WET PCO2i				.27	.82*
WET BHTf					.72*
WET PCO2f					

x $p < 0.10$
* $p < 0.05$

These data would indicate that the synchronized swimmers were better able to override the increasing efferent input to the inspiratory muscles physiologically and/or psychologically. Since it was determined that there were no central regulatory differences at the medullary chemosensitive areas and if psychological 'will-power' were not the sole cause, the magnitude of the difference was most likely effected by a peripheral physiological factor. In the loop of efferent and afferent signals activated during breath-holding, the inspiratory muscles are the primary peripheral component. If there were a decrease in the amount of afferent feedback from these muscles, then there would be less impingement upon the conscious centers causing the desire to breathe. This lowered sensitivity coupled with greater conscious overriding of the afferent stimulus would result in increased breath-holding capacity. This is the most probable mechanism by which the synchronized swimmers are able to demonstrate such extended breath-holds.

Immersion

Both rebreathing and breath-hold tests were administered in the same manner during immersion. The control subjects showed no alteration in their chemosensitive response or in their ability to breath-hold while immersed to the clavicle. The synchronized swimmers, however, showed a significant reduction in their hypercapnic response slope from 1.Ø5 to Ø.67 (table 3); yet, their breath-holding times and end PCO2 values were unaltered (table 4). This provides further evidence for the dissociation of chemosensitivity and breath-holding ability.

It was hypothesized that immersion would decrease the chemosensitive slope of all subjects due to the increase of afferent input from the inspiratory muscles. Presumably, at the same PCO2, the hydrostatic pressure exerted on the inspiratory muscles resulted in an increased inhibitory afferent feedback to the respiratory centers. The output from the respiratory rate-generator would then be decreased because of the higher afferent input. Yet, only the synchronized swimmers appeared to be sensitive to the change in inspiratory muscle feedback.

Such a finding would lead to the hypothesis that the synchronized swimmers are more responsive to afferent activity from the inspiratory muscles. Breath-holding may demonstrate that the synchronized swimmers had minimized this afferent drive during hypercapnia. This observation coupled with the decrease in chemosensitive slope during immersion emphasizes the apparent importance of the afferent drive in the synchronized swimmers' response to hypercapnia. Their afferent response is decreased yet its influence on the efferent response is maximized.

It had also been hypothesized that BHT would be decreased during immersion due to a decrease in TLC. There was a significant correlation between TLC and dry BHT_i ($p<0.05$). It has been shown that immersion decreases TLC (2,10,14,67); apparently, the decrease in VC during immersion was not sufficient to have an effect upon breath-holding ability.

CHAPTER 5

SUMMARY

This study investigated the respiratory drive to inhaled CO2 at rest in synchronized swimmers. These athletes were used because of their ability to breath-hold for long periods of time. Swim-trained control subjects were used so that the effect of breath-hold training could be distinguished from swim-training. Respiratory sensitivity to CO2 (chemosensitivity) was determined by rebreathing a 7% CO2, 93% O2 mixture (Read technique, 65). The resulting minute ventilations were plotted over end-tidal PCO2 to give an indirect measure of the medullary chemosensitivity to blood PCO2. Repeated breath-holding which allows the subject to ventilate between each breath-hold without decreasing blood PCO2 was used to assess the subjects' breath-holding ability and the subjects' ability to withstand hypercapnic stress.

A secondary purpose of this study was to investigate the effect of increased inspiratory work on chemosensitivity and breath-holding. Each subject performed both rebreathing and breath-holding tests a second time while immersed in water to the clavicle. Immersion increases the force production needed for a normal tidal volume. In turn, this increase in force increases the afferent feedback from the inspiratory muscles to the respiratory centers.

No significant differences in chemosensitivity were found between the control subjects and the synchronized swimmers. Initial and final breath-hold times (BHT_i and BHT_f) were longer and initial and final PCO2 values ($PCO2_i$ and $PCO2_f$) were higher in the synchronized swimmers. There was no correlation between BHT_i, BHT_f, $PCO2_i$, $PCO2_f$ and chemosensitivity. The chemosensitive slope was reduced in the synchronized swimmers from 1.05 to 0.67 with immersion, but, was unchanged in the control group. BHT and PCO2 values were unchanged in both groups with immersion.

The data indicates that the medullary chemoreceptors do not play a direct role in determining BHT. If these receptors influenced breath-holding ability, chemosensitivity should correlate with BHT.

These data also suggest that the synchronized swimmers may have some altered response in the loop of afferents and efferents activated during both CO2 rebreathing and breath-holding. In conclusion, the primary mechanism by which the synchronized swimmers are able to perform extended breath-holds does not appear to be a reduced medullary sensitivity to blood PCO2. Further studies to elucidate the role of the sensitivity of the inspiratory muscles to efferent input and the role of their afferent output in determining efferent activity during breath-holding are needed.

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