VENTILATORY RESPONSE TO CARBON DIOXIDE AND EXERCISE IN SWIMMERS

by

Ann Margaret Leevers

B.Sc. Simon Fraser University 1976

THESIS SUBMITTED IN PARTIAL FULFILLMENT OF

THE REQUIREMENTS FOR THE DEGREE OF

MASTER OF SCIENCE

in the Department

of

Kinesiology

© Ann Margaret Leevers April,1985

SIMON FRASER UNIVERSITY

All rights reserved. This work may not be reproduced in whole or in part, by photocopy or other means, without permission of the author.

APPROVAL

Name: Ann Margaret LEEVERS

Degree: Master of Science (Kinesiology)

Title of Thesis: Ventilatory Response to Carbon Dioxide and Exercise in Swimmers

Examining Committee:

Chairman: Dr. J. Wilkinson

Dr. J. Morrison
Senior Supervisor

Dr. T. Smith

Professor M. Savage

Dr. J. Fleetham
External Examiner
U.B.C. Health Sciences Centre

Date Approved: 17 April 1985

PARTIAL COPYRIGHT LICENSE

I hereby grant to Simon Fraser University the right to lend my thesis, project or extended essay (the title of which is shown below) to users of the Simon Fraser University Library, and to make partial or single copies only for such users or in response to a request from the library of any other university, or other educational institution, on its own behalf or for one of its users. I further agree that permission for multiple copying of this work for scholarly purposes may be granted by me or the Dean of Graduate Studies. It is understood that copying or publication of this work for financial gain shall not be allowed without my written permission.

Title of Thesis/Project/Extended Essay

Ventilatory Response to Carbon Dioxide and Exercise in Swimmers

Author:

(signature)

ANN M. LEEUERS

(name)

April 18/85

(date)

ABSTRACT

Previous studies have suggested that SCUBA divers, breath-hold divers and endurance runners exhibit a low ventilatory response to hypercapnia. They also appear to have a reduced steady-state, and possibly a lower neurogenic, ventilatory response to exercise. The extent to which competitive swimmers may differ in any one of these factors is unclear. The purpose of this study was to test the hypothesis that swimmers adapt to their activity in such a way that their ventilatory response to hypercapnia and exercise is different from non-swimmers.

Resting arterialized venous blood samples were taken from 16 untrained subjects, 16 endurance runners and 16 competitive swimmers and analyzed for pH, PCO_2, PO_2 and bicarbonate. VC, $FEV_{1.0}$ and MVV were determined by spirometry. Ventilatory response to inspired CO_2 was determined at rest by a rebreathing method. The subjects performed the Sjostrand PWC_{170} bicycle ergometer test during which $\dot{V}O_2$ and $\dot{V}I$ were determined. The neurogenic ventilatory response to exercise was estimated from the increase in ventilation observed during the first 15 seconds of exercise at each workload. The steady-state ventilatory response to exercise was measured as the difference between the steady-state ventilation from one workload to the next. Both the neurogenic and steady-state ventilatory responses to exercise were normalized with respect to the change in $\dot{V}O_2$ between loads.

No significant differences in either acid-base status or ventilatory response to CO_2 were found among the groups. The swimmers had significantly greater VC and $FEV_{1.0}$ ($P<0.05$) than both the runners and the nonathletes. The nonathletes also had a significantly lower MVV ($P<0.05$) than the other two groups. The estimated aerobic capacity, as determined by the PWC_{170} test and the slope of exercise heart rate to $\dot{V}O_2$, differed significantly between groups. The runners had the highest aerobic capacity ($P<0.05$) and the nonathletes the lowest ($P<0.05$). Both the runners and the swimmers had a lower steady-state ventilatory response to exercise than the nonathletes ($P<0.05$). The only significant difference in the neurogenic response occurred during the recovery phase of the exercise test, with the nonathletes having a greater response than the swimmers ($P<0.05$). The swimmers exhibited a unique exercise breathing pattern consisting of a high tidal volume and a low breathing frequency relative to the other two groups ($P<0.05$). Neither the lung functions or breathing pattern of the swimmers were related to aerobic capacity, suggesting a training effect specific to swimming.

ACKNOWLEDGEMENTS

I would like to express my sincere gratitude to my senior supervisor, Dr. J.B. Morrison, for his patience and support throughout the research and writing of this thesis.

Further sincere thanks are extended to my committee members, Dr. T.J. Smith and Prof. M. Savage, for their time and assistance and to N. Ison for his statistical expertise. Additional thanks go to P. Sullivan and W. Burke for providing their invaluable help during the testing, and to the 48 subjects who made it all possible.

Finally, I would like to express my gratitude to all my friends, especially Sharon Williams, for the support and practical help given to me throughout my tenure as a graduate student.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

I. Literature Review

Introduction

The responses of the human respiratory system to various stimuli have been studied extensively. The effects of hypercapnia have been of particular interest, primarily because of the importance of carbon dioxide in the control of respiration (West, 1979). Similarly, the ventilatory response to muscular exercise has been examined as a means of elucidating the mechanisms involved in the regulation and control of breathing. The possibility that the ventilatory responses elicited by these two different stimuli (CO_2 and exercise) may be related has also aroused considerable interest. There are several factors that may affect or modify the respiratory response to CO_2 and/or exercise. The purpose of this review is to describe the effects of hypercapnia and exercise on the respiratory system and to briefly examine the various factors that may be involved.

Ventilatory Response to Carbon Dioxide

Increasing alveolar carbon dioxide tension results in an increase in ventilation. When ventilation is graphed against rising alveolar PCO_2, the response is generally linear if the alveolar PO_2 is normal (100 mmHg). Decreasing the alveolar PO_2 results in a steeper slope and a higher ventilation for a given PCO_2. In addition, the initial portion of the curve is exponential (West, 1979). The result is the classic "hockey stick response curve". Figure 1.0(a) illustrates the affects of different alveolar PO_2 levels on the ventilatory response to CO_2. Figure 1.0(b) demonstrates the ventilatory response to increasing inspired CO_2 concentrations when the alveolar PO_2 is high (above 200 mmHg). CO_2 concentrations greater than approximately 13 percent appear to have little further affect on ventilation as evidenced by the levelling off of the curve. The ventilation response has a characteristic pattern consisting of initially an increase in tidal volume, which is then followed by a rise in breathing rate, if CO_2 tension continues to increase (Schaefer, 1958). The tidal volume increases gradually upon acute exposure to CO_2 and slowly returns to normal upon withdrawal (Schaefer, 1958).

The normal response to CO_2 appears to have a wide range of variability (Hirschman *et al.*, 1975; Irsigler, 1976; Sahn *et al.*, 1977; Schaefer, 1958). There is some evidence to suggest

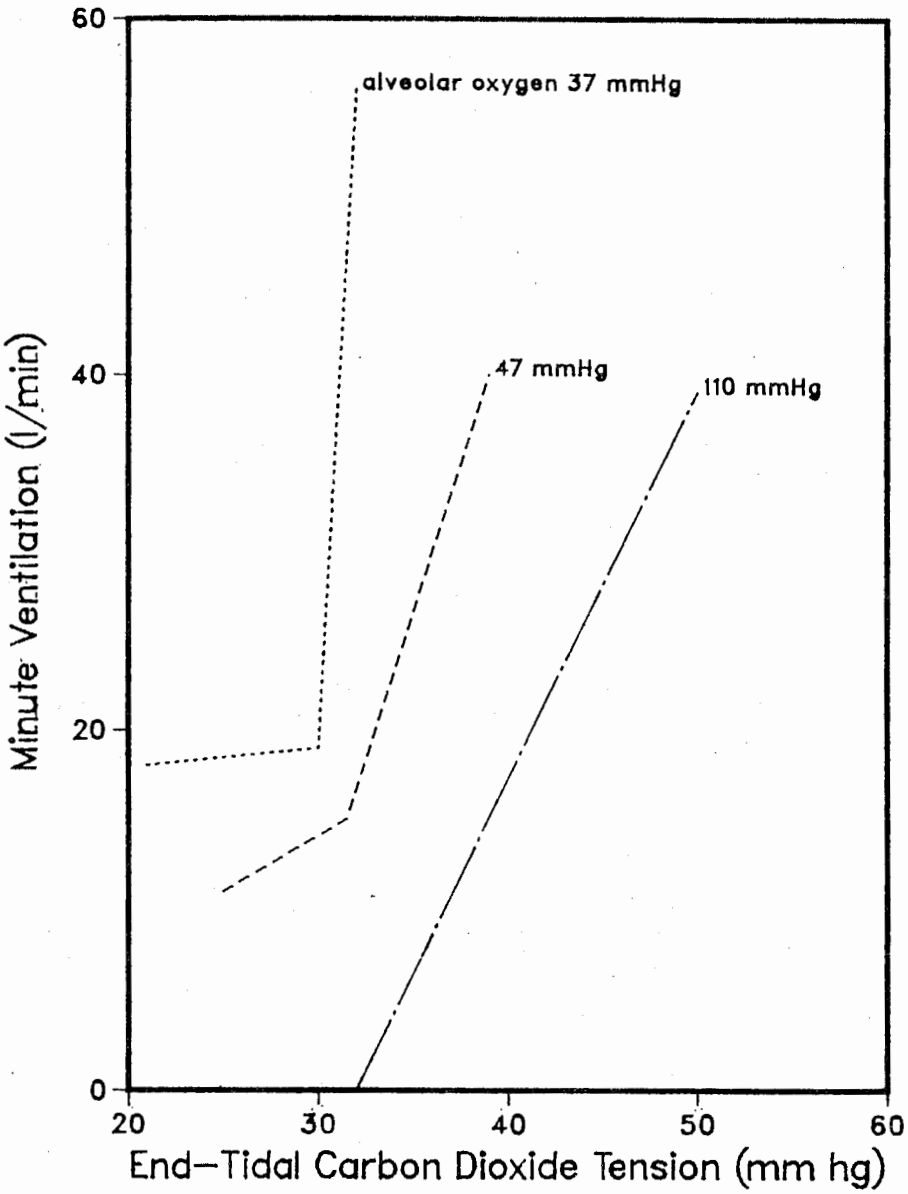


Figure 1.0(a): Ventilatory response to CO_2 at different alveolar PO_2 levels (West, 1979).

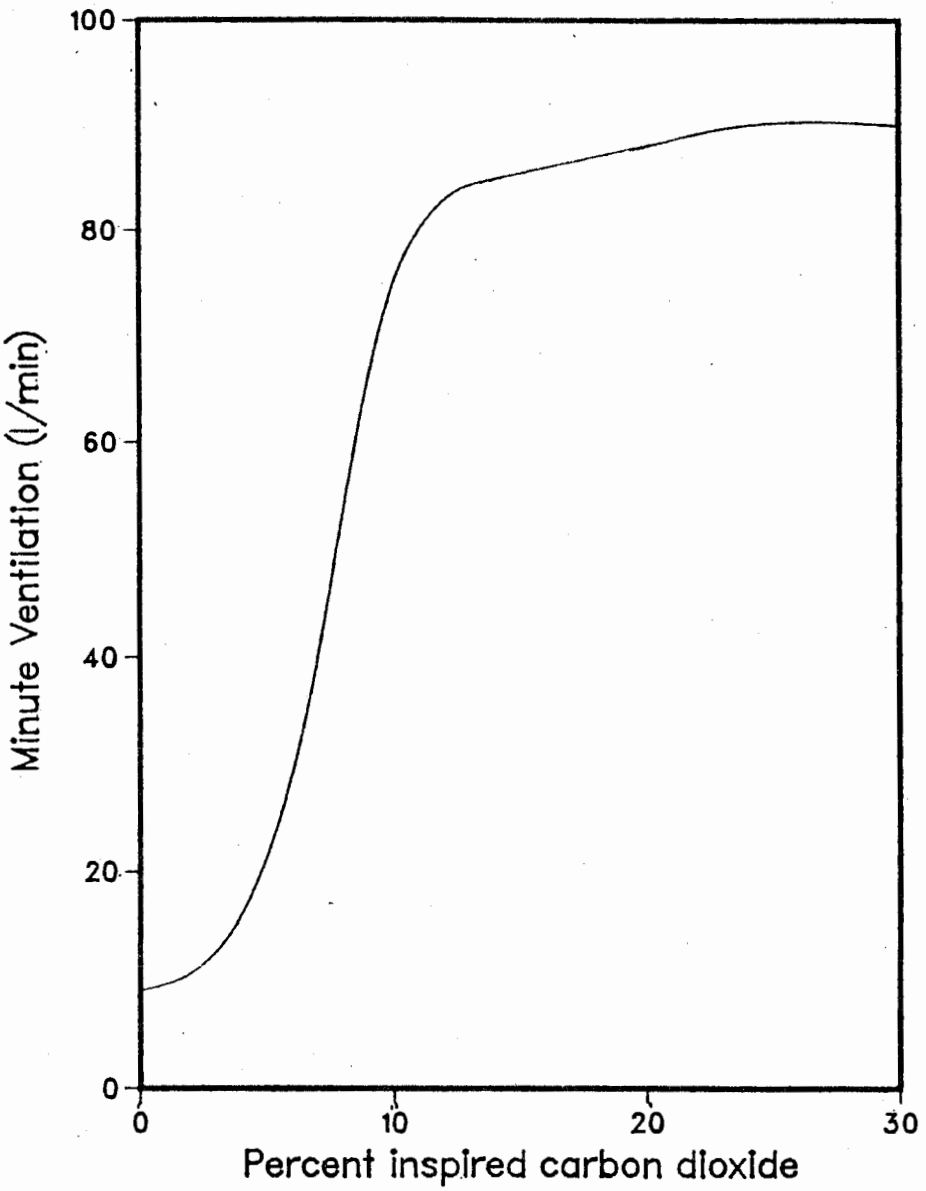


Figure 1.0(b): The ventilatory response to different concentrations of inspired CO_2 (West, 1979).

that women have a lower response to CO_2 than men (Irsigler, 1976; Patrick and Howard, 1972; Read, 1967) but this is not supported by the work of Hirschman and colleagues (1975). Haywood and Bloeke (1969) studied the respiratory responses of 20 young women to inhalation of gas mixtures with varying concentrations of CO_2. They compared their results with those of several reported experiments involving male subjects and concluded that the effects of CO_2 inhalation on women are as variable, but not dissimilar from effects on men. There does not seem to be any correlation between hypercapnic response and age (Hirschman et al., 1975; Irsigler, 1976; Patrick and Howard, 1972), except in the very elderly, in whom there appears to be a reduced response (Brischetto et al., 1980). Most investigators have not observed any correlation between CO_2 response and height or weight (Irsigler, 1976; Patrick and Howard, 1972), with the exception of Hirschman and coworkers who found a positive correlation. Some of the variance in hypercapnic response could be due to genetic factors or physical training effects.

In an attempt to determine whether the interindividual variability in hypercapnic response could be attributed to an hereditary component, Arkinstall et al. (1974) studied the ventilatory response to CO_2 of 30 sets of twins. They were unable to demonstrate any significant genetic influence on the minute ventilation response but estimated that genetic factors were responsible for 80 to 90 percent of the variability in

breathing pattern. A later study of familial influences on the hypercapnic response of five distance runners also failed to determine any hereditary basis (Scoggin et al., 1978). Conflicting evidence has been presented by Saunders et al. (1976). They selected a group of young swimmers and compared their respiratory response to CO_2 to that of their parents and siblings. They found that there was a strong relation between the CO_2 response of siblings of the same family. However, it was not clear if this correlation was due to genetic or non-genetic familial factors.

It is difficult to define what effect, if any, athletic training has on the ventilatory response to CO_2. Many investigators focused on comparing the slope of the resting ventilatory response to hypercapnia of trained athletes to that of untrained controls and have reported conflicting observations. Some studies have shown the slope to be reduced in athletes (Byrne-Quinn et al., 1971; Miyamura et al., 1976), whereas in other reports, athletes have had the same response as untrained subjects (Heigenhauser et al., 1983; Mahler et al., 1982; Scoggin et al., 1978). Martin et al. (1979) found that male distance runners had a lower response to CO_2 than nonathletes during both light and heavy exercise, but did not analyze the resting response. Since the athletes in these investigations differed in degree and type of training, it is difficult to interpret the results. As such, it is not possible to assess whether a particular response is due to physical

training or is an innate characteristic of the athlete. The specific effect of training was prospectively examined in three studies and three different conclusions were reached. Bradley et al. (1980) tested nine untrained males, before and after six to eight weeks of aerobic training, and found no change in hypercapnic respiratory response. In contrast to the above study, two other groups of researchers reported opposite conclusions. After training, Blum et al . (1979) found that the slope of the response was reduced for the five male subjects, whereas six males who underwent seven months of rowing training exhibited an increased slope (Kelley et al., 1984).

Although there are large individual variations in ventilatory hypercapnic response, certain groups of individuals have been identified as being generally less responsive to inspired CO_2. The common factor shared by these particular groups is that some degree of breath-holding or prolonged inspiratory effort, in water is required by their activities. Both breath-hold divers (Schaefer, 1955; Song et al., 1963) and SCUBA divers (Brousolle et al., 1968; Florio et al., 1979) have been shown to have a reduced response to CO_2. Underwater hockey players could also be included in the category of breath-hold divers, since their sport involves extended periods of breath-holding while swimming underwater. Unpublished data on participants in this activity indicate that the hockey players had a ventilatory response that was only 55 percent of the control level (Wallersteiner et al., 1980). SCUBA divers tend to

practise a form of apneustic breathing while diving. Several investigators have observed that SCUBA divers are less sensitive to hypercapnia than non-divers (Brousolle et al., 1968; Florio et al., 1979).

It is possible that apneustic breathing is equivalent to repeated exposure to higher than normal CO_2 tensions (Goff and Bartlett, 1957; Schaefer, 1955; Song et al., 1963). If so, divers' low hypercapnic drive may reflect a respiratory acclimatization to CO_2. Support for an acclimatization theory is provided by investigations of individuals who were exposed to chronic hypercapnia. Several studies concerned with the effects of prolonged elevated CO_2 demonstrated that, following exposure, CO_2 inhalation had a decreased effect on the subjects' ventilation (Brackett et al., 1969; Schaefer et al., 1963; van Ypersele, 1974).

Transition and Steady-State Ventilatory Response to Exercise

Ventilation increases with muscular exercise. The cause(s) of the increase in ventilation which accompanies exercise still remains a relatively unsolved problem in spite of considerable research in the area and a number of reviews aimed at evaluating the research (Dejours, 1964; Dempsey et al., 1979(b); Levine, 1978; Whipp, 1983). The amount of increase generally depends on: (1) the intensity of the exercise; (2) the type of exercise; (3) phases of the exercise; and (4) environmental conditions

(Dejours, 1964; Levine, 1978). Because of the complexity of the subject of exercise hyperpnea, this review will concentrate on the respiratory response to exercise in terms of its possible relation to the hypercapnic response.

Dejours (1963) was one of the first to describe the pattern of the ventilatory response to dynamic muscular exercise. Figure 2.0 illustrates the classic response. The specific pattern that he described, and which is now generally accepted, consists of essentially two separate phases. With onset of exercise, there is an immediate increase in ventilation followed by a plateau and then a further progressive increase to steady-state levels during moderate exercise. A similar abrupt decrease in ventilation occurs at cessation of activity, followed by a further slow decline. Exercise is designated as moderate if it is below the anaerobic threshold (Levine, 1978; Wasserman et al., 1973; Whipp, 1983). Anaerobic threshold may be defined as the point at which lactic acid accumulation in the blood accelerates due to the involvement of anaerobic metabolic processes in meeting energy requirements (Astrand and Rodahl, 1979).

The first phase of the response is the immediate increase in ventilation accompanying exercise onset. It is considered to have neural origins due to its short time span (Asmussen, 1973; Broman and Wigertz, 1970; Dejours, 1963; Paulev, 1971; Sinclair, 1978; Dempsey et al., 1979(a)). There appears to be large individual variation in the magnitude of the neural response and

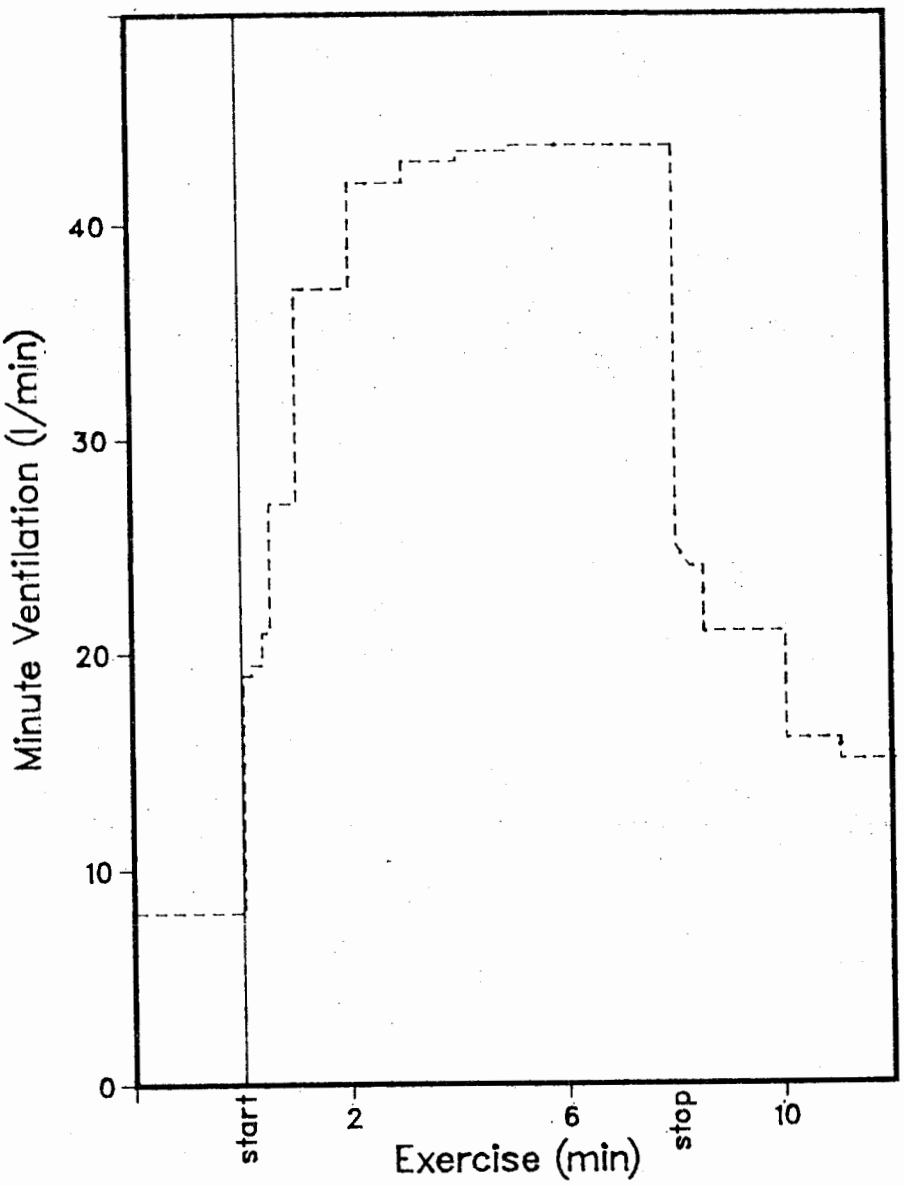


Figure 2.0: The classic ventilatory response to dynamic exercise (Dejours, 1963).

it may not be present in all subjects (Beaver and Wasserman, 1968; Broman and Wigertz, 1970; Jensen et al., 1971). Most studies concerning exercise hypernea showed immediate increases in ventilation occurring at the initiation of dynamic exercise, with the increment in ventilation lasting between 15 and 20 seconds (Asmussen, 1973; D'angelo and Torelli, 1971; Paulev, 1971). This was generally the case when work was imposed from rest. However, when the transition was between two levels of work, a more slowly developing hyperpnea was usually evident (Broman and Wigertz, 1970; Casaburi et al., 1977; Pearce and Milhorn, 1977). An exception was the investigation by Bennett and coworkers (1981), who found both a fast and slow component of ventilatory response to exercise from one workload to another. They also observed that the fast component had a time constant which was comparable to that reported by Fujihara et al. (1973(a)). The model of the ventilatory response to exercise formulated by Fujihara and coworkers (1973(b)) incorporated two components, a fast one and a slow one. The fast component had a time constant of approximately 19 seconds. This two component model is currently accepted as being the most appropriate to describe the response characteristics of exercise hyperpnea (Whipp, 1983).

The magnitude of the neural component may be dependent on exercise intensity and type. D'angelo and Torelli (1971) investigated the neural respiratory response during different types of activity. They concluded that the initial increase in

ventilation was related to the metabolic intensity of the exercise and seemed to depend on the form of activity. Later research by Asmussen (1973) also suggested that the size of the fast component was roughly related to energy expenditure. Other investigations do not corroborate these findings and show the increment in ventilation to be relatively constant irrespective of workrate (Dejours, 1963; Jensen, 1972). The psychological effects of variations in testing format, such as prewarning and the use of different starting orders, do not appear to have any measurable affect on the neural response (Jensen et al., 1971).

The second (slow) phase of exercise-induced hyperpnea is thought to be elicited by humoral pathways (Dejours, 1963; Levine, 1978; Whipp, 1983). As seems to be the case for the neurogenic response, the magnitude of steady-state ventilation depends on the intensity and type of activity. During moderate exercise, steady-state ventilation is linearly related to metabolic rate (Astrand and Rodahl, 1979; Dejours, 1964; Wasserman et al., 1967; Wasserman et al., 1973). It can be quantitatively assessed by utilizing the relationships of amount of ventilation (VE) per unit oxygen consumption (VE/VO_2), or the amount of ventilation per unit CO_2 production (VE/VCO_2) (Wasserman, 1978; Whipp, 1983). VE/VO_2 is not significantly changed from the resting state during steady-state dynamic exercise (Comroe, 1964; Wasserman, 1976). At similar levels of oxygen consumption, bicycle exercise elicited greater increases in ventilation than treadmill exercise (Koyal et al., 1976). The

steady-state ventilatory response also appears to be lower during swimming than walking or running (Holmer et al., 1974; McArdle et al., 1971). The majority of studies comparing athletes and non-athletes have found that, in general, athletes have a lower exercise ventilation per unit oxygen uptake than non-athletes (Dempsey et al., 1977; Heigenhauser et al., 1983; Martin et al., 1979).

It is debatable whether a relationship exists between the ventilatory responses to CO_2 and exercise. There is some evidence which suggests that individuals with a low hypercapnic drive also tend to have an unusual response to exercise in terms of alveolar PCO_2, ventilation, and/or breathing pattern. In a study of well-trained underwater swimmers, Goff and Bartlett (1957) found that the swimmers had a higher average end-tidal CO_2 tension, associated with a lower VE/VO_2, than non-swimmers during swims at various speeds. There was no apparent difference in resting (underwater) end-tidal CO_2 tensions between the two groups. Similar results were reported by Lally et al. (1974) in comparing SCUBA divers and endurance runners to control subjects. During treadmill exercise, both the divers and the runners exhibited a higher alveolar PCO_2 and a relative hypoventilation.

Several researchers have hypothesized that hypercapnic ventilatory drive is directly associated with ventilation during exercise. A positive correlation between ventilatory response to CO_2 at rest and exercise hyperpnea (measured as either VE/VO_2 or

$\dot{V}E/\dot{V}CO_2$) has been reported in a number of papers (Byrne-Quinn et al., 1971; Dempsey, 1976; Martin et al., 1978; Martin et al., 1979). Another study involving marathon runners versus nonrunners found a positive correlation between resting hypercapnic response and exercise $\dot{V}E/\dot{V}CO_2$ for the marathoners, but not for the nonrunners (Mahler et al., 1982). Two more recent investigations, one dealing with swimmers (Heigenhauser et al., 1983) and the other with 'normal' subjects and patients with obesity hypoventilation syndrome (Menitove et al., 1984), failed to demonstrate any connection between CO_2 response and $\dot{V}E/\dot{V}CO_2$.

Whereas steady-state ventilatory response to exercise has been compared between different groups, there is little information on the comparison of the initial (neurogenic) response. This is an apparently unique aspect of the study by Lally et al. (1974) which contrasted the ventilatory dynamics of SCUBA divers to that of endurance runners and control subjects. The exercise protocol consisted of treadmill walking up a ten percent grade at three different speeds. This entailed three separate experiments involving a transition from rest to walking. The steady-state ventilatory response was measured as the difference between the minute ventilation during the last two minutes of exercise and the resting $\dot{V}E$. The initial response was measured as the increase in inspired ventilation observed during the first 15 seconds of walking. The ventilatory responses were normalised by dividing by body weight. The

results were plotted as a function of oxygen uptake at each speed. The steady-state ventilatory response of the divers was significantly less than that of the control group at the two fastest speeds (3.2 and 4.8 km/hr) and significantly less than the runners at 3.2 km/hr. Both the divers and the runners exhibited a reduced neurogenic response relative to that of the controls at the higher workloads, but the differences were not significant. Although the authors were unable to demonstrate any significant difference in neurogenic response among the three groups, they suggest that the lower exercise ventilation of the divers was a result of both a "conditioned response phenomenon" and "a reduced chemosensitivity".

Breathing Pattern

Breathing patterns at rest and during exercise have been examined thoroughly. The general conclusion is that any particular minute ventilation is comprised of the combination of tidal volume and breathing frequency which is most efficient in terms of energy expenditure (Bouhuys, 1977; Vidruk and Dempsey, 1980; Yamashiro et al., 1975). It is not clear how the regulation of breathing pattern occurs. One popular hypothesis suggests that ventilation is regulated by a system which is governed by the central nervous system and is therefore controlled in the same manner as other types of movement. The theory specifies that there is a precise program for activating

the respiratory muscles according to the "principle of minimal effort" (Vidruk and Dempsey, 1980; Yamashiro et al., 1975). Such a program would be subject to learning and could be altered when necessary to achieve the most efficient ventilation.

Optimization of ventilatory energy expenditure depends primarily on tidal volume and, to a lesser extent, on respiratory rate. The total work of breathing is the sum of the work against elastic and flow-resistive forces (Bouhuys, 1977; West, 1979). The amount of elastic work is a function of the depth of breathing. The relationship is alinear, such that a high inspiratory volume requires a large energy expenditure to overcome the elastic recoil of the lungs and chest wall, and is very inefficient (Bouhuys, 1977; West, 1979). Therefore, even during exercise (in air), tidal volume does not usually exceed 50 percent of VC (Astrand and Rohdal, 1979; Bouhuys, 1977). The force required to overcome the elastic properties of the lungs and chest wall is independent of the rate of inflation so that progressively increasing the rate of breathing does not affect elastic work (Otis, 1964). For a given minute ventilation, minimization of flow-resistive work appears to depend to some degree on breathing frequency, but there is a wide range of variability in the optimum frequency (Bouhuys, 1977). The optimum rate may also be a function of muscle force, since the rate values corresponding to minimum work also correspond to those for minimum muscle force (Mead, 1960). When ventilation is increased, as in exercise, flow rates rise regardless of whether

breathing frequency, or tidal volume, or both are increased (Bouhuys, 1977; West, 1979). Higher flow rates require a greater amount of work against flow-resistive forces.

In terms of strictly mechanical work, it would appear that a low tidal volume and a high breathing rate would be most efficient, due to the reduction in elastic work. However, the goal of ventilation is to provide sufficient exchange of O_2 and CO_2 in the alveoli to satisfy the body's metabolic requirements. A low VT, high frequency breathing pattern cannot achieve adequate alveolar ventilation and results in inefficiency of gas exchange (Bouhuys, 1977; West, 1979). Therefore, assuming that alveolar ventilatory needs are being met, the most efficient ventilation depends on the minimization of the sum of the work involved in any combination of tidal volume and respiratory rate.

In addition to the actual mechanical work of breathing, there is an associated perceived effort. The sense of respiratory effort is related to the work required to overcome elastic and flow-resistive forces and therefore, during 'normal' breathing in air, is determined by tidal volume and breathing frequency (Jones, 1984). It follows that normal resting ventilation would result in minimization of respiratory sensation. Although ventilatory pattern appears to be governed primarily by the demand for minimal energy expenditure of the respiratory muscles, there may be other, sometimes conflicting, influences operating.

One possible influence may be hypercapnic ventilatory drive. The same groups of individuals who exhibit a reduced ventilatory response to CO_2 (breath-hold and SCUBA divers) also appear to share a similar type of breathing pattern at rest. This pattern is characterized by an high tidal volume and a low respiratory frequency (Florio et al., 1979; Schaefer et al., 1963; Wallersteiner et al., 1980). These observations are consistent with those of other investigators who concluded that frequency of breathing and the slope of the CO_2 response curve are positively correlated within a 'normal' population of subjects (Hey et al., 1966; Hirschman et al., 1975; Schaefer, 1958).

Information provided about the ventilation of divers during exercise confirms that they have a distinctive breathing pattern (Crosbie et al., 1979; Lally et al., 1974). In an attempt to determine whether this was attributable to an aerobic training effect, Lally et al. (1974) examined the exercise minute ventilation of SCUBA divers in relation to runners and nonathletes. The divers' ventilation was achieved with an high tidal volume and a low respiratory frequency relative to the patterns for the other two groups. In this study, the results of the runners were between those of the divers and the controls. However, Martin et al. (1979) found no difference in the components of tidal volume or respiratory rate at matched ventilation between runners and control subjects. The important feature of the above investigations was the lack of correlation

between aerobic capacity and exercise response in terms of breathing pattern. This would seem to suggest that the unusual ventilatory behavior of divers is not fitness related.

Pulmonary Function

Measures of pulmonary function such as lung volumes and ventilatory capacities are other factors that may be related to hypercapnic drive. A study by Irsigler (1976) employed a large number of subjects to determine the limits of the normal ventilatory response to CO_2. He concluded that there was significant correlation between the slope of the CO_2 response and vital capacity (VC) and maximum voluntary ventilation (MVV). In addition, the same groups categorized as low CO_2 responders tended to have a high VC and in some cases, a high inspiratory capacity (IC) and maximum breathing capacity (MBC). Stuart and Collings (1959) tested the lung functions of athletes and non-athletes and reported that athletes had a greater VC. The subjects in this study were from various sports and included both endurance and sprint athletes. Two other investigations concerned with the pulmonary function of endurance runners indicated that they also had greater forced vital capacities (FVC) than non-runners (Novak et al., 1968; Raven, 1977). In contrast, Mahler and colleagues (1982) found no significant difference in either FVC or forced expired volume between runners and controls. Schaefer (1958) measured lung volumes of

31 subjects and found that those with low ventilatory response to CO_2 had a larger tidal volume and VC than those subjects with a greater CO_2 sensitivity. Breath-hold divers were observed to have high VC, IC and MBC levels (Song et al., 1963) and underwater hockey players exhibited a greater VC than control subjects (Wallersteiner et al., 1980). Conflicting results have been reported for SCUBA divers. Two groups of researchers were unable to distinguish any measurable differences in lung functions between divers and non-divers (Froeb, 1960; Florio et al., 1979). Commercial divers demonstrated a greater FVC than non-divers in another investigation (Crosbie et al., 1979).

Hypercapnia and Acid-Base Status

Most CO_2 is transported in the blood as bicarbonate as a result of the hydration of CO_2 to carbonic acid and the subsequent dissociation into HCO_3^- and H^+ ions.

i.e. $CO_2 + H_2O <-----> H_2CO_3 <-----> HCO_3^- + H^+$

The dissociation of carbonic acid into HCO_3^- and H^+ is very rapid and therefore the actual amount of carbonic acid in the blood is minimal (West, 1979). Since the transport of CO_2 involves an important buffer system, it must be related to the acid-base status of the blood.

Brackett et al. (1965) defined the 'normal' acid-base response curve for acute hypercapnia. They found that the plasma bicarbonate concentration of their subjects rose in a

curvilinear fashion as PCO_2 was increased. Despite the compensation provided by the bicarbonate buffer, H^+ did not return to 'normal', but increased in direct proportion to the degree of hypercapnia; each mm Hg increase of PCO_2 resulting in a 0.76 nmole increment in the H^+ activity.

van Ypersele (1974) attempted to define an acid-base response curve for chronic hypercapnia. He reported an increase in plasma bicarbonate concentration that was incrementally greater than that observed for acute hypercapnia. Despite the increase in bicarbonate, H^+ concentration was not returned to 'normal' but rose linearly with the degree of hypercapnia. However, the increase in H^+ concentration for each mm Hg rise in PCO_2 was much less than that observed in acute hypercapnia (0.32 nmole H^+ / mm Hg PCO_2).

The results from van Ypersele's study (1974) provide support for the conclusions of Schaefer et al. (1963 and 1964) that man undergoes a respiratory acclimatization to CO_2. They reported that following chronic exposure to elevated CO_2, subjects had an increased plasma bicarbonate and a pH that had returned towards 'normal' after an initial decrease.

It may be that the respiratory acclimatization to CO_2 which is indicated by an attenuation of the ventilatory response to CO_2 is a function of the bicarbonate concentration of the blood. Evidence for this idea is provided by Turino et al. (1974). They investigated man's ventilatory response to breathing 5% CO_2 and found a good correlation between serum bicarbonate and

increments in ventilation.

Swimming

The circulatory-respiratory systems' response to swimming may be different from its response to other sports activities because of the possible effects of certain unique aspects of swimming. They are as follows: (1) swimming is performed in the horizontal position; (2) ventilation is restricted; (3) external thoracic pressure is increased due to water pressure; and (4) heat conductance of water is higher than that of air. A few researchers have attempted to compare the effects of swimming versus other activities. In two studies comparing the physiologic response of swimmers during swimming and running, several disparities were observed (Magel, 1975; McArdle et al., 1971). Both maximum ventilation and heart rate attained during swimming were less than levels attained running. In addition, for any given level of oxygen consumption, both pulmonary ventilation and heartrate were lower while swimming. Holmer (1972), in a study on oxygen uptake during swimming, observed that the maximum oxygen uptake obtained was less than that reached in uphill treadmill running. In a later investigation, Holmer et al. (1974) compared the hemodynamic and respiratory responses in swimming and running. They found that at submaximal workloads, cardiac output, stroke volume and heart rate were similar during swimming and running, but at maximal loads oxygen

uptake was 15% lower and cardiac output 10% lower in swimming. Ventilation during maximal work was also lower in swimming, and was attributed primarily to a reduced frequency of breathing.

There is evidence that swimmers tend to have a high total lung capacity and forced vital capacity (Saltin and Astrand, 1967; Shephard et al., 1974), similar to levels found in commercial divers (Crosbie et al., 1979). This might lend further support to the premise that ventilatory response is somehow altered in swimming. The fact that swimmers appear to have a greater VC than other athletes (Cunningham and Eynon, 1975; Novak et al., 1968; Shapiro et al., 1964) would suggest a specific swimming effect separate from a general training one.

It seems reasonable to hypothesize that swimmers might also exhibit a ventilatory response to CO_2 different from the norm. Data concerning the differences in ventilatory response to hypercapnia between swimmers and untrained subjects seems to be limited. Heigenhauser, et al. (1983) investigating the ventilatory responses of three groups of females, used recreational swimmers as the control group. Since all three groups of subjects were involved in swimming to some extent, it is not surprising that no difference in hypercapnic drive was found among the three groups. Saunders et al. (1976) studied the ventilatory response in 23 children from a swimming class and reported that there was no significant difference in response between the swimmers and non-swimming siblings of similar age. The young age of the subjects and the actual amount of swim

training involved makes it difficult to draw definitive conclusions from these results. A valid investigation of ventilatory response to hypercapnia in swimmers versus untrained controls suggested that swimmers had a reduced response, but the difference was not significant (Ohkuwa et al., 1980).

Summary

The human respiratory response to hypercapnia may be dependent on numerous factors including breathing pattern, plasma bicarbonate levels, and lung volumes and ventilatory capacities. In turn, acclimatization to CO_2 seems to be responsible for lowering the ventilatory response to CO_2 and possibly, to exercise. It is difficult to separate the factors actually involved in the process of acclimatization to CO_2. Acclimatization to CO_2 could be accomplished by improving pulmonary function, altering breathing pattern (increasing tidal volume and decreasing breathing frequency), and/or increasing plasma bicarbonate. It may be that reduced hypercapnic drive is not a result of any changes in the above components of the respiratory system, but reflects a decreased reactivity of the autonomic nervous system. Such an explanation was provided by Schaefer (1975) who noted that adaptation to hypercapnia appeared to produce a damping effect on the cholinergic system. It seems evident that there is a need for a greater understanding of the complexities of the respiratory response to

hypercapnia.

II. Objectives

The purposes of this study were:
(1) To test the hypothesis that swimmers undergo respiratory acclimatization to their activity to the extent that they have a lower hypercapnic ventilatory drive and/or exercise ventilatory drive, and a different breathing pattern, than nonswimmers;
(2) To test the hypothesis that ventilatory drive is modified by both general and specific (swimming) training effects. Endurance runners were included as a second control group to identify responses that could be attributed to a general training affect;
(3) To determine if swimmers have greater pulmonary functions (greater VC, $FEV_{1.0}$, MVV and $FEV_{1.0}/VC$) than nonswimmers.

In addition, there were two secondary objectives: to determine whether differences in ventilatory drive can be related to other respiratory variables; and to test the hypothesis that there is no difference in the ventilatory response to CO2 between males and females.

If there is respiratory acclimatization to swimming, it may in fact be adaptation to CO2. Adaptation to CO2 could be a result of: metabolic compensation, such as an increase in blood bicarbonate buffer; changes in pulmonary function; conditioned response; damping of the cholinergic system; or a combination of these factors. Alternatively, a change in any of the above factors, with or without a corresponding change in hypercapnic

response, could in itself reflect an acclimatization to swimming. Therefore, in this study, several respiratory variables including: pulmonary function, neurogenic ventilatory response to exercise, breathing pattern and acid-base status were examined to elucidate possible causes of any differences between swimmers and untrained subjects.

III. Methods

Research Design

(A) Subjects:

There were three experimental groups of subjects: 16 swimmers from the Simon Fraser University swim team; 16 endurance runners from the general population; and 16 non-athletes from the general population. Each group had 10 males and six females.

The following prerequisites were established for the experimental groups:

1. swimmers
    a. currently competing;
    b. training approximately 24 hours/week for four months prior to testing;
2. endurance runners
    a. currently training;
    b. not currently swimming more than once a week;
    c. matched to the swimmers as closely as possible for height and weight;
3. non-athletes

a. could be active but not involved in endurance training or regular strenuous exercise;

b. aerobic capacity (determined by a submaximal fitness test) to be distributed about the average according to norm tables (CAPHER, 1969);

c. matched as closely as possible to the swimmers for height and weight.

All subjects fulfilled the following requirements:

1. non-smokers or those who had not smoked for at least one year;
2. no currently practicing SCUBA divers;
3. free of respiratory dysfunction (determined by a medical history form);
4. between the ages of 18 and 30 years;

The subjects were asked to refrain from strenuous activity for 12 hours prior to testing and to refrain from eating for at least two hours prior to testing.

This study received approval from the University Ethics Committee in advance of any subject testing. Each of the subjects was asked to read a participant information form and to complete medical history and informed consent forms as a precondition to testing. Each subject received a payment of ten dollars following completion of testing.

(B) Experimental Protocols:

The experimental design consisted of five separate test protocols. Each subject completed the five protocols on the same day (between 9 A.M. and 6 P.M.) in the following order:

1. determination of acid-base status;
2. measurement of vital capacity (VC), forced expired volume in one second ($FEV_{1.0}$) and maximum voluntary ventilation (MVV);
3. measurement of height, weight and skinfolds;
4. determination of ventilatory response to carbon dioxide;
5. bicycle ergometer exercise test, which included the measurement of heart rate, mixed expired oxygen and carbon dioxide fractions, and breath-by-breath inspired volume.

This particular order of testing was chosen to minimize any possible effect of one test on another. The exercise test was performed last because it could have affected the acid-base status, which in turn might have affected the CO_2 response. Similarly, ventilation could have been elevated as a result of anxiety over the blood test or because of the lung function tests. Therefore, measuring height, weight and skinfolds after these two tests gave the subject an opportunity to recover and ventilation a chance to return to resting levels. The CO_2 response test was followed by a rest period (10 to 15 minutes) during which the participants breathed 100 percent oxygen for the first few minutes to ensure that they began the ergometer

test with normal resting alveolar O_2 and CO_2. Prior to each set of tests, room temperature and barometric pressure were measured. All the subjects were tested under similar environmental conditions and they all followed the same procedures immediately prior to and during the experiments.

Table 1.0 outlines the variables measured and calculated for the entire experimental protocol.

Acid-Base Status

An estimate of the acid-base status of the resting subject was made by using the BMS2 MK2 Blood microsystem (Radiometer, Copenhagen) and the Astrup equilibration technique (Siggard-Andersen *et al.*, 1960) to analyze blood samples. Arterialized venous blood was collected in heparinized glass capillary tubes (volume approximately 60 ul. and length 7.5 cm.). The procedure for blood collection involved heating the subject's ear with a hot pack for about one minute, and then pricking the earlobe with a microlancet. Three capillary tubes were filled with blood anaerobically, by placing the tube in the center of the blood droplet. Two of the blood samples were equilibrated with high (8%) and low (4%) PCO_2 gas mixtures, respectively, and then measured for corresponding pH values. The third unequilibrated sample was used for measurement of actual (in vivo) pH. The Siggard-Andersen Curve nomogram was used to determine $PaCO_2$, standard bicarbonate and base excess from the

Table 1.0: Variables Measured and Calculated for the Entire Experiment

Measured Variables	
pH (units)	actual, resting
pH (units)	equilibrated to high PCO_2
pH (units)	" " low PCO_2
VC (liters)	best of 3 trials
$FEV_{1.0}$ (liters)	" " " "
MVV (l/min)	" " " "
Height (cm)	
Weight (kg)	
Skinfold thick.(mm)	six sites
VI (liters BTPS)	every 30 sec. for 4 min.(CO_2response)
$PACO_2$ (mmHg)	" " " " " " " "
VI (liters BTPS)	16 min. of exercise test
HR (B/min)	last 10 sec. of each min. for 16 min.
FEO_2 (%)	last 10 sec. of rest,exerc. & recov.
$FECO_2$ (%)	" " " " " " " "
VT (liters BTPS)	16 min. of exercise test

Calculated Variables	
$PaCO_2$ (mmHg)	from pH values
Standard Bicarbonate	
$\dot{V}I$ (l/min BTPS)	from VI for each 30 sec.of CO_2 test
Slope	$\dot{V}I$ to $PACO_2$
Intercept	" " "
PWC_{170} (kpm/kg)	from HR and workload
$\dot{V}I$ (l/min BTPS)	for last and first 15 sec of each load
Ave. VT (liters BTPS)	for last and first 20 sec." " "
Bf (Br/min)	" " " " " " " " "
$\dot{V}O_2$ (l/min STPD)	from FEO_2, $FECO_2$ and VI
$\Delta\dot{V}I/\Delta\dot{V}O_2$ (onset)	from $\dot{V}I$ and $\dot{V}O_2$
$\Delta\dot{V}I/\Delta\dot{V}O_2$ (steadystate)	" " " "

obtained pH values (Siggard-Andersen et al., 1960).

The pH electrodes were calibrated every morning and immediately before each blood collection according to the instructions given in the BMS2 MK2 operating manual (the sensitivity of the electrode is ±0.008 pH).

Lung Function

Lung function tests were performed on a Collins 9-liter spirometer. A Collins 13.5 liter spirometer was used for three of the swimmers for a more accurate measure of their vital capacity.

Vital capacity (VC): The drum speed was set at 32 mm/minute and the subject was asked to breathe normally into the spirometer (nose clip on). An initial period of adjustment (several breaths) was given to allow the subject to relax and become familiar with the equipment. The subject was then asked to make a maximal inspiration, followed by a maximal expiration. VC was determined from the difference between the value (in ml) at the lowest point and the value (in ml) at the highest point of the breathing manoeuvre.

Forced expired volume in one second ($FEV_{1.0}$): The subject was asked to follow the same preparation as for VC, then after a maximal inspiration, to exhale as forcefully as possible. Immediately after the maximal inspiration, the drum speed was accelerated to 1920 mm/min. The volume exhaled in the first

second was the $FEV_{1.0}$. This was determined by subtracting the value (in ml) at that point in the expiration curve representing one second of expiration from the value (in ml) at the point where expiration began.

Maximum voluntary ventilation (MVV): This manoeuvre required the subject to breathe in and out as fast and as forcibly as possible for 15 seconds with the kymograph speed set at 160 mm/min. Integrated output was obtained from a mechanical integrator attached to the system. The slope of the curve was used to calculate the expired minute ventilation (l/min) for the 15 seconds.

Each measure was repeated three times and the best result recorded. The three respiratory variables were normalized using Cotes (1965) lung volume nomographs. The $FEV_{1.0}/VC$ ratio was also determined.

Anthropometric Data

The subject's height, weight and age were recorded. Skinfold thickness was measured with a Harpenden skinfold caliper (Edwards et al., 1955). Skinfolds were measured at six sites: subscapular, suprailliac, triceps, umbilical, front thigh and calf, as illustrated in Figure 3.0. The sum of the six measurements was used as an indicator of adiposity (Yuhasz, 1962).

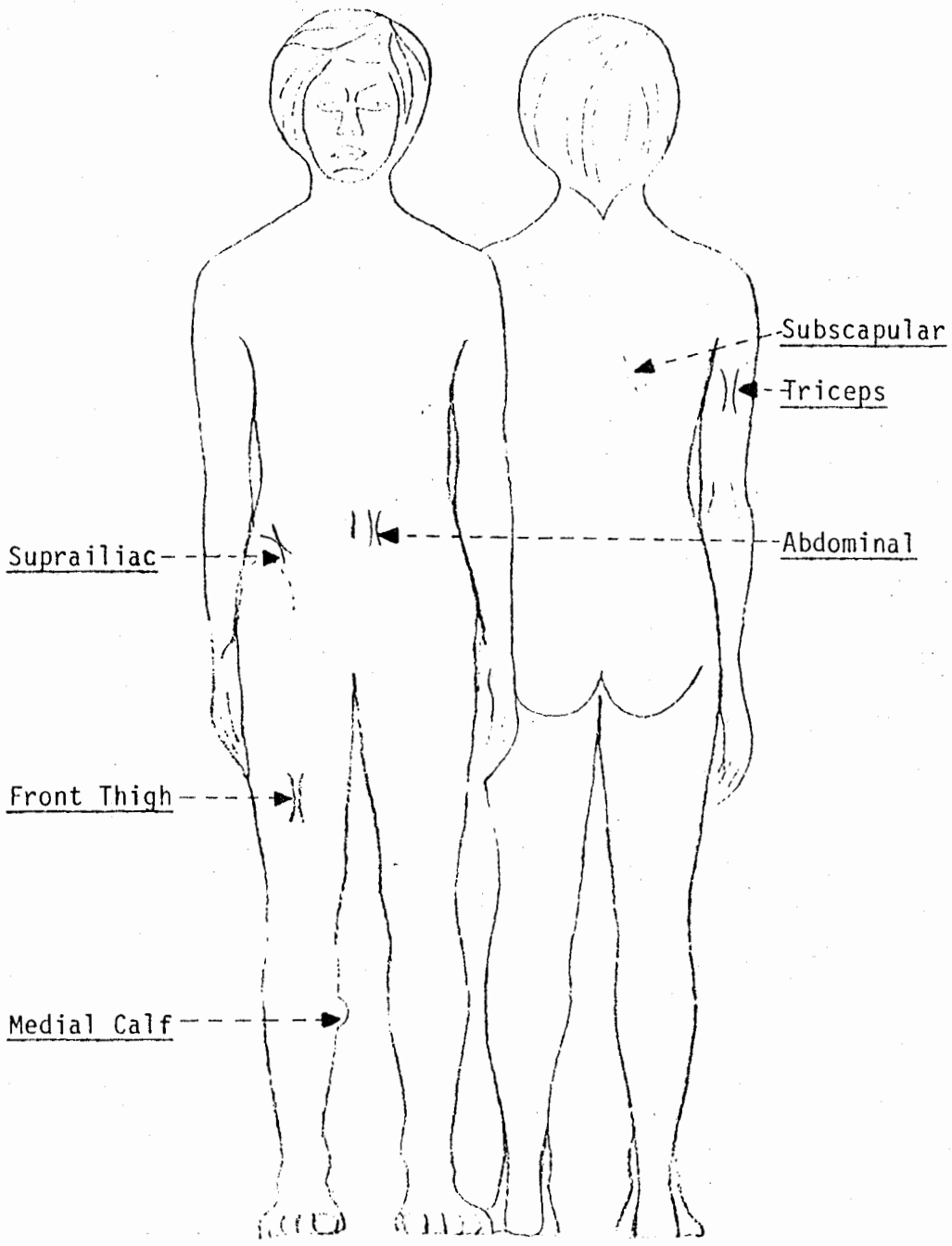


Figure 3.0: Sites for skinfold measurement.

Ventilatory Response to CO_2

Ventilatory response to carbon dioxide was determined using a rebreathing procedure, similar to that developed by Read (1967). The equipment utilized for the procedure is shown schematically in Figure 4.0. It consisted of a 10 liter rubber bag inside a rigid, plexiglass box. The bag was connected, via a flexible Collins hose and a low resistance 2-way selection valve, to a mouthpiece. The mouthpiece was connected, via the valve, to either the rebreathing bag or a balloon containing a gas mixture of 51% O_2 and the balance N_2. A Collins hose connected the plexiglass box to a Parkinson-Cowan dry gas ventilation meter via a 2-way respiratory valve. This arrangement allowed the ventilation of the subject to be recorded indirectly. Respiratory gases were sampled via a gas line inserted at the mouthpiece and connected to a rapid response oxygen analyzer (Applied Electrochemistry) and a rapid response CO_2 analyzer (Godart,Capnograph). A Hewlett-Packard pen recorder was connected to the CO_2 analyzer to allow for measurement of respiratory frequency from the oscillations of CO_2. A thermistor probe was placed at the mouthpeice to measure gas temperature.

The procedure was performed with the subject sitting in a chair. The rebreathing bag was filled to a volume of approximately 10 liters with a gas mixture of 7% CO_2 / 50% O_2 and the balance N_2. The subject breathed the O_2 / N_2 gas mixture

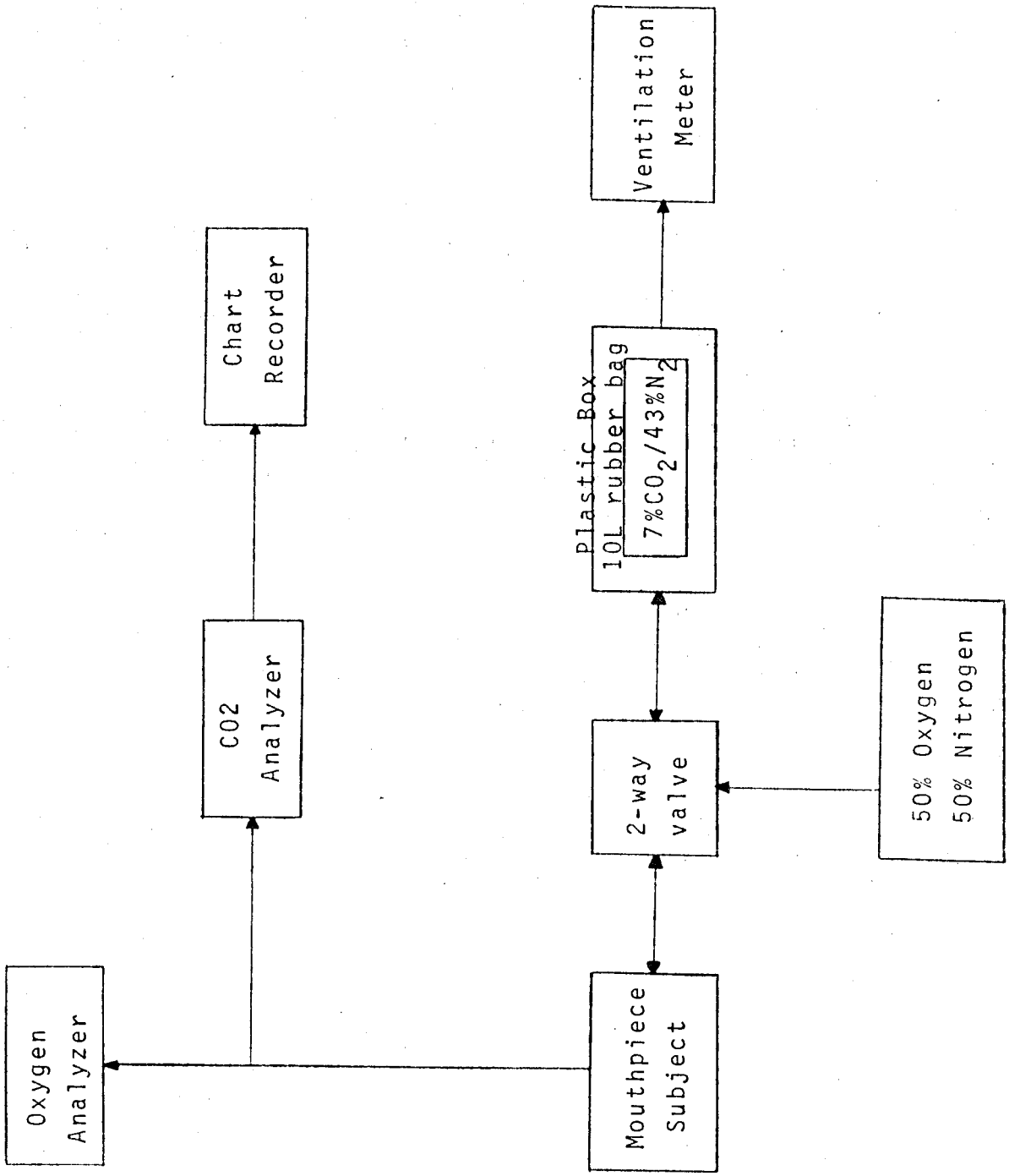


Figure 4.0: Schematic diagram of the experimental setup for the determination of the ventilatory response to CO_2.

from the balloon for three minutes and then was asked to expire maximally. Following the maximal expiration, the 2-way valve was switched to the rebreathing bag and the subject rebreathed from the bag for a maximum of four minutes. As the subject rebreathed from the bag, the CO_2 content of the bag increased. The inspired air volume was measured by volume displacement by means of the ventilation meter attached to the plexiglass box. The respiratory gases were sampled continuously at the mouthpiece and analyzed for inspired and alveolar O_2 and CO_2 fractions.

The O_2 and CO_2 analyzers and the pen recorder were calibrated daily to primary standards, gravimetric gas mixtures of known composition. Data were recorded for each 30 second interval of rebreathing.

Ventilation was plotted against alveolar CO_2 tension. The relationship between alveolar CO_2 and ventilation is represented as follows:

$$V = s(PACO_2 - B),$$

where s is the slope of the line expressed as change in ventilation / unit change in $PACO_2$ and B is the extrapolated intercept on the abscissa ($PACO_2$ axis).

Estimation of Aerobic Capacity

Aerobic capacity was determined by having the subject perform a modified Sjostrand PWC_{170} test (CAHPER, 1969) on a manually braked Monark bicycle ergometer. Following a five

minute (approximately) rest period, the subject cycled continuously at 50 RPM for 16 minutes. Pedal frequency was maintained by having the subject keep pace with a mechanical metronome. An initial load, dependent on the subject's sex, size, and fitness level, was preset on the ergometer. The fitness level as judged by the tester, was dependent on the subject's experimental group, competitive background, present activity level, and subjective evaluation. The workload was increased after four and eight minutes of cycling. The load that was set was determined by the heart rate during the final minute of the previous exercise period. A chart providing ranges of exercise heart rates at different workloads was used to establish the correct load that would keep the heart rate below 170 beats per minute (CAPHER, 1969). The last four minutes of exercise was a recovery period during which the subject pedalled at zero load. Heart rate (HR) was recorded on a Nihon Kohden electrocardiogragh (Cardiofax, ECG2101) via three surface electrodes (CM5 placement: right and left mid-axilliary, and left upper back) for the last 10 seconds of each minute. The heart rate recorded during the last minute of exercise at each workload was plotted against workload. Regression analysis was used to obtain the equivalent workload at the heart rate of 170 beats per minute. The PWC_{170} (physical work capacity at a heart rate of 170 beats/minute) was then calculated in terms of the subject's body weight.

Transition and Steady State Ventilatory Response to Exercise

Neurogenic Ventilatory Response

The neurogenic ventilatory response to exercise was also measured during the fitness test protocol by employing a procedure adapted from that of Lally et al. (1974). The experimental setup is shown in Figure 5.0. To ensure a resting state, the subject was prepared for testing and then asked to sit quietly on the bicycle for approximately five minutes prior to pedalling. Inspiratory flowrate was monitored at rest and throughout the exercise period by means of a Fleisch pneumotachograph and a Validyne differential pressure transducer (Model DP45, ±25cm H2O). The pneumotachograph was calibrated before each test. The output from the pneumotachograph was collected for calibration and for the entire exercise protocol on an Hewlett-Packard analog 12-channel tape recorder (Model #3907-06A)

Mixed expired air was analysed for O_2 and CO_2 using a rapid response O_2 analyser (Applied Electrochemistry) and a rapid response CO_2 analyser (Capnograph,Godart). The percent O_2 and CO_2 were recorded at rest and during the last ten seconds of each exercise load. A detailed outline of the test protocol is given in Table 2.0.

The respiratory gas analysers were calibrated daily and before each exercise test.

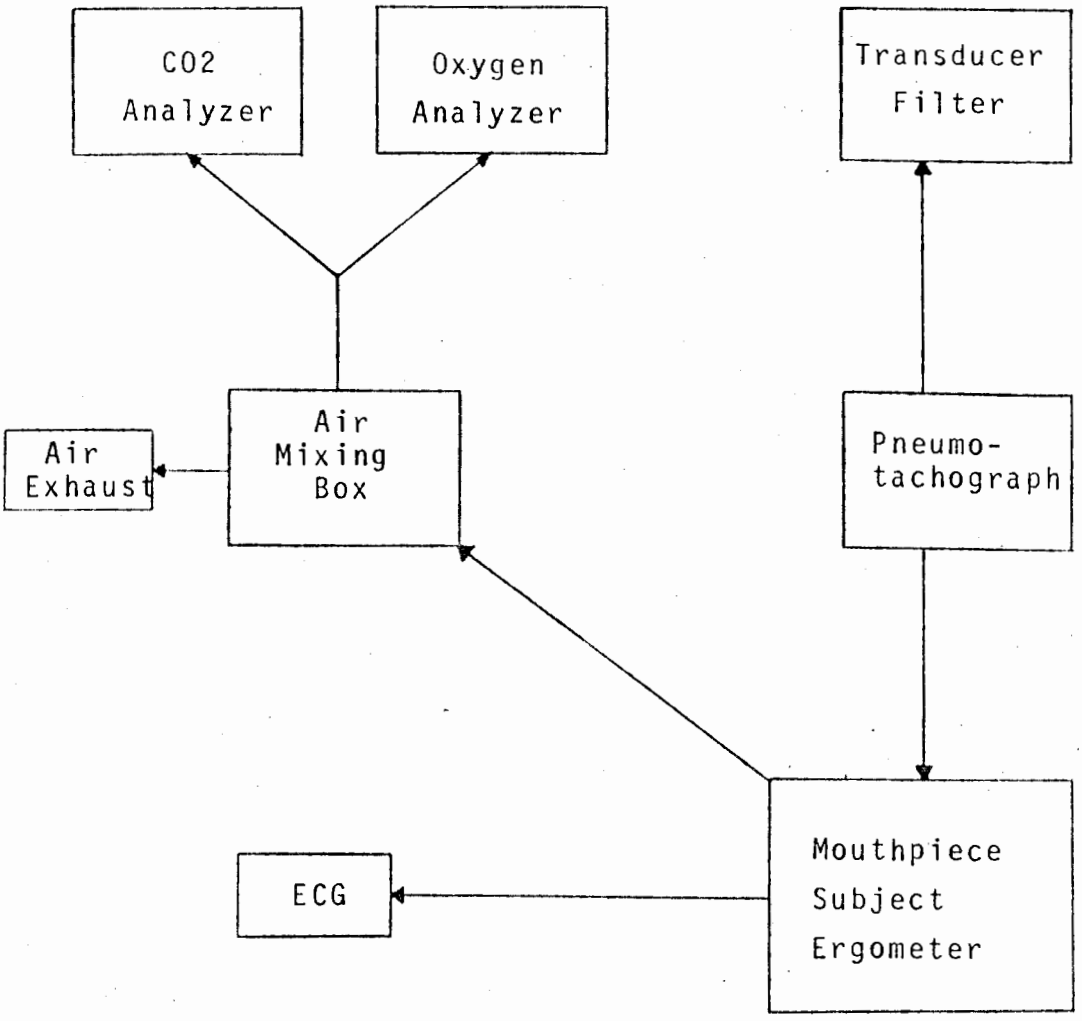


Figure 5.0: Schematic diagram of the experimental setup for the exercise response.

Table 2.0: Ergometer Test Protocol

Time (min:sec)	Load (kpm)	ECG (6sec)	$\%O_2, \%CO_2$	VI (collect 20sec)
-1:00	set			
-0:30	(no pedalling)			on
-0:10		on	record	
0:00	pedalling			on
3:30				on
3:50		on	record	
4:00	increase			on
7:30				on
7:50		on	record	
8:00	increase			on
11:30				on
11:50		on	record	
12:00	load off			on
15:30				on
15:50		on	record	
16:00	stop			

The raw data were replayed from the tape via an A-to-D converter to a LSI-1103 computer. Respiratory data digitized at 100Hz were integrated using numerical methods. The integrated data were used to provide breath-by-breath inspired tidal volume for 20 second intervals immediately before and following the change of workload.

Mean inspired minute ventilation ($\dot{V}I$) was calculated for the first 15 seconds (approximately) of each 20 second interval of inspired tidal volumes.

Average tidal volume (VT) was calculated from the sum of inspired tidal volumes divided by the number of breaths in 20 seconds.

Oxygen consumption ($\dot{V}O_2$) was determined at rest and for each exercise load by the equation:

$$\dot{V}O_2 = \dot{V}I \; [\; FIO_2 - FEO_2(FIN_2/FEN_2) \;],$$

where FIx and FEx are the fractions of inspired and expired gases.

The increase in ventilation observed during the first 15 seconds of each exercise load was utilized as an estimate of the neurogenic component of exercise ventilatory response. The neurogenic ventilatory response was calculated as the change in ventilation in the first 15 seconds following onset of a new workload from the ventilation for 15 seconds, within the last 30 seconds, of the previous exercise load. For analysis purposes, each neurogenic response was then normalised to the change in steady-state $\dot{V}O_2$ from one exercise load to the next.

Steady-State Ventilatory Response

Steady-state ventilatory response to a given step load was measured as the difference between the steady-state $\dot{V}I$ attained after four minutes of pedalling at each exercise load (the final 15 sec.) and the steady-state $\dot{V}I$ of the previous four minute work period. Again, for analysis purposes, each steady-state response was then normalised with respect to the change in $\dot{V}O_2$ between loads.

Statistical Treatment of Data

For each variable measured, the means and the standard deviations were calculated for the subjects grouped by training (swimmer,non-athlete, runner) and by training and sex (male swimmer, female runner, etc.). Significant differences among the above groups for each variable were examined by applying oneway analysis of variance tests, using the BMDP ANOVA program (Dixon,1981). A probability of 0.05 was the level used to accept a difference as significant.

A correlation matrix of the variables was constructed for all the subjects, for the subjects grouped by sex and for the subjects grouped by training.

A multiple analysis of variance was applied to the group means for all the variables to test for differences between any

of the variables (using the SPSS MANOVA program) (Nie et al., 1979).

IV. Results

Subjects

Individual subject data and environmental measures are given in Appendix A. The prerequisites outlined in the methods were met by all the subjects, with the exception of training status. Ten of the swimmers completed their training approximately two weeks prior to the testing.

Acid-Base Status

The mean values of pH, $PaCO_2$ and standard bicarbonate, measured while the subjects were at rest, are shown in Table 3. There were no significant differences in mean pH, $PaCO_2$ and standard bicarbonate among the groups.

Lung Function

The mean vital capacity (VC), forced expired volume ($FEV_{1.0}$), maximum voluntary ventilation (MVV) and $FEV_{1.0}/VC$ of the three groups of subjects are shown in Table 4. The values of VC, $FEV_{1.0}$ and MVV have been normalized with respect to height, according to the predictions of Cotes (1965), as described in

Table 3.0: Mean values of acid-base status of each group: measured at rest.

Group	N	pH (units)	$PaCO_2$ (mmHg)	Stand.Bicarb. (meq/l)
Swimmers	16	7.43 ±0.01	44.87 ±1.16	27.43 ±0.47
Nonathletes	16	7.43 ±0.01	43.75 ±1.33	27.47 ±0.53
Runners	16	7.43 ±0.01	45.35 ±1.17	28.60 ±0.83

(values are Means ±S.E.M.)

Table 4.0: Mean values of lung function of each group: data is normalized with respect to height according to Cotes (1965) and is presented as a fraction of the predicted value.

Group	VC	$FEV_{1.0}$	MVV	$FEV_{1.0}/VC$
Swimmers (n=16)	1.23±0.04*	1.10±0.02*	1.20±0.05±	0.78±0.03
Nonathletes (n=16)	1.06±0.03	0.94±0.03	1.04±0.05	0.79±0.02
Runners (n=16)	1.06±0.03	1.00±0.03	1.23±0.03±	0.82±0.02

(values are Means ±S.E.M.)

* denotes significantly greater than nonathletes and runners.
± denotes significantly greater than nonathletes only.

the methods. The absolute values are given in Appendix B. Both the VC and $FEV_{1.0}$ levels for the swimmers were found to be significantly greater than values for the nonathletes' and the runners' ($P<0.01$). The nonathletes had a significantly lower MVV than either the swimmers or the runners ($P<0.05$). There was no significant difference in $FEV_{1.0}$ / VC between groups.

Anthropometric Data

Table 5.0 shows the mean age, height, weight and sum of the skinfolds of the three groups of subjects. The attempt to match the subjects in terms of height and body weight was successful with the exception of the difference in weight between the swimmers and the runners. The swimmers had a mean body weight of 73.2 kg, which was significantly greater than the mean weight of the runners of 60.3 kg ($P<0.01$). This was principally due to the difference in weight of the males, rather than the females. There also appeared to be differences in the mean skinfolds among the three groups, but the only significant difference was a greater skinfold of the nonathletes compared to the other two groups ($P<0.05$). When separated according to sex, both the female swimmers and female nonathletes were found to have significantly greater skinfolds than the female runners ($P<0.05$ and $P<0.01$, respectively), whereas only the skinfolds of the male nonathletes were significantly greater than the runners ($P<0.05$). No significant differences in skinfolds were found

Table 5.0: Mean values of anthropometric data of each group.

Group	N	Age (years)	Weight (kg)	Height (cm)	Skinfolds (mm)
Swimmers					
(total)	16	20.1±0.3	73.2±3.3≠	176.0±3.2	74.2±7.4≠
(males)	10	20.2±0.4	81.0±2.3≠	183.7±2.7	61.9±7.2
(females)	6	20.0±0.6	60.3±4.0	163.1±3.0	94.7±11.9≠
Nonathletes					
(total)	16	20.6±0.9	67.4±2.7	172.8±2.5	92.9±9.8*
(males)	10	20.4±1.0	70.4±3.8	176.6±2.1	74.9±10.7≠
(females)	6	21.0±1.8	62.6±2.9	166.4±4.7	122.9±11.5≠
Runners					
(total)	16	23.4±1.0	60.3±1.6	171.5±2.0	43.0±2.7
(males)	10	22.9±1.2	63.0±1.5	174.7±2.0	37.9±2.1
(females)	6	24.2±2.1	55.8±2.5	166.2±3.1	51.4±4.7

(values are Means ±S.E.M.)

* denotes significantly greater than swimmers and runners
≠ denotes significantly greater than runners only.

between either the male swimmers and nonathletes or the females of those two groups.

Ventilatory Response to Carbon Dioxide

Each of the subjects exhibited a similar type of response during the rebreathing test. Their ventilation increased as the CO_2 fraction in the rebreathing bag increased. This is demonstrated in Figure 6.0 which shows the relationship of ventilation to end-tidal carbon dioxide tension ($PETCO_2$) for the three groups. The mean x ($PETCO_2$) and y ($\dot{V}I$) for each individual was used to calculate the mean x and y (center of gravity) for each group.

The mean slopes and intercepts calculated by regression of each subjects' ventilation on $PETCO_2$ are given in Table 6. The individual slopes and intercepts are shown in Appendix C and a plot of all the slopes and intercepts is given in Appendix D. There was no significant difference in either the mean slope or the mean intercept among the three groups of subjects, or between the males and females. To allow for differences in subject size, the results were normalized by dividing the slope by the subject's VC (Cameron, 1979). Again, there was no significant difference among the three groups. However, the mean value of the slope/VC for the females was significantly greater than that for the males ($P<0.05$).

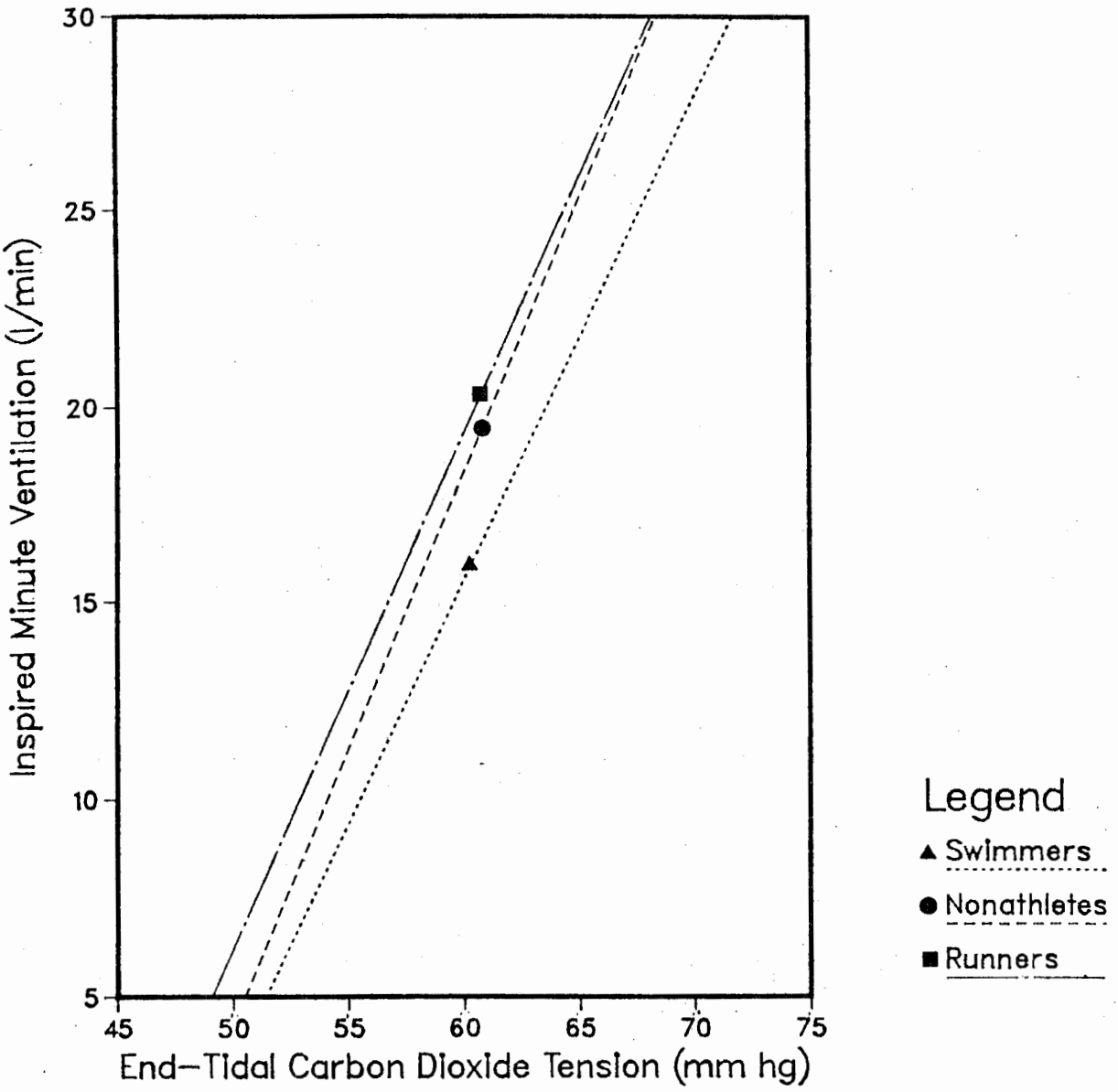


Figure 6.0: Graph of ventilation against end-tidal CO_2: based on the mean x and y for each group (the points represent the center of gravity of each regression line).

Table 6.0: Mean slope and intercept of the response of ventilation (l/min.BTPS) to $PETCO_2$.

Group	N	Slope (l/min.mmHg)	x-Intercept (mmHg PCO_2)
Swimmers			
(total)	16	1.35 ±0.17	45.5 ±1.7
(males)	10	1.46 ±0.20	47.1 ±1.4
(females)	6	1.15 ±0.33	42.8 ±3.8
Nonathletes			
(total)	16	1.54 ±0.13	45.6 ±1.3
(males)	10	1.50 ±0.14	45.4 ±1.6
(females)	6	1.59 ±0.28	45.9 ±2.6
Runners			
(total)	15	1.44 ±0.16	44.3 ±1.1
(males)	9	1.37 ±0.21	44.5 ±1.2
(females)	6	1.54 ±0.25	43.9 ±2.4

(values are Means ±S.E.M.)

Estimation of Aerobic Capacity

The mean PWC_{170} and the mean PWC_{170}/kg of the subjects are shown in Table 7. The estimated aerobic capacity as determined by the PWC_{170} test, differed significantly between groups. Both the swimmers and the runners had a significantly greater absolute PWC_{170} than the nonathletes ($P<0.01$). The swimmers mean PWC_{170} was lower by 255 kpm than that of the runners, but this difference was not significant. When the means were calculated by group and sex, the difference in PWC_{170} between the female swimmers and nonathletes disappeared. The runners had a greater work capacity per kilogram body weight (PWC_{170}/kg) than the other two groups ($P<0.01$), both when treated as a composite group and when compared by sex. The swimmers also demonstrated a greater PWC_{170}/kg than the nonathletes ($P<0.01$), but when separated according to sex, again only the male difference was significant ($P<0.01$).

Transition and Steady State Ventilatory Response to Exercise

Neurogenic Response

Table 8.0 shows the mean neurogenic ventilatory response to exercise which was calculated as described in the "Methods" and then normalized as the transient change in ventilation per unit increment in steady state $\dot{V}O_2$ (L/min.$\dot{V}$I)/(L/min.$\dot{V}O_2$) elicited by

Table 7.0: Mean PWC_{170} and PWC_{170}/kg of each group: (for definition of PWC_{170} see methods).

Group	N	PWC_{170} (kpm)	PWC_{170}/kg (kpm/kg)
Swimmers			
(total)	15	1172.3 ±95.2≠	15.75 ±0.84≠
(males)	9	1414.2 ±65.8≠	17.34 ±0.68≠
(females)	6	809.3 ±95.8	13.36 ±1.34
Nonathletes			
(total)	16	755.8 ±47.9	11.24 ±0.64
(males)	10	845.6 ±59.3	12.13 ±0.89
(females)	6	606.2 ±25.5	9.75 ±0.48
Runners			
(total)	16	1429.4 ±78.2≠	23.65 ±1.02*
(males)	10	1607.4 ±63.1≠	25.59 ±0.84*
(females)	6	1132.8 ±93.7≠	20.41 ±1.71*

(values are Means ±S.E.M.)

* denotes greater than swimmers and nonathletes
≠ denotes significantly greater than nonathletes only

the step change of workload. The only significant difference in neurogenic response found was between the swimmers and the nonathletes in the transition from load 3 to zero load (onset of recovery), wherein the nonathletes had a greater response than the swimmers ($P<0.01$).

Table 8.0: Mean neurogenic ventilatory response (onset) to exercise in the transition from rest to load 1; from load 1 to load 2: from load 2 to load 3; and the mean of 3 onset of load transients (mean); and from load 3 to zero load (recovery): ($L.min.^{-1}\dot{V}I/L.min.^{-1}\dot{V}O_2$)

Group	Onset Load1	Onset Load2	Onset Load3	Onset Mean	Onset of Recovery
Swimmers (N=13)	8.48±1.49	7.68±1.56	7.21±1.85	7.5±1.0	1.52±0.42
Nonathletes (N=16)	7.04±1.36	5.78±1.66	13.05±2.9	8.6±1.5	4.67±1.26*
Runners (N=15)	9.61±1.44	7.79±0.98	9.09±1.53	8.8±0.8	3.36±0.47

(values are Means ±S.E.M.)

* denotes significantly greater than swimmers only

Steady State Response

The steady state ventilatory response to exercise was also normalized as the steady state change in ventilation per unit increment in steady state $\dot{V}O_2$ (L/min.$\dot{V}I$)/(L/min.$\dot{V}O_2$) elicited by step change in workload. The calculated mean responses of the three groups are shown in Table 9. Significant differences in response between groups occurred following four minutes of exercise at load 3 (S.S.load3), and after the period of pedalling at zero load (S.S.recovery). The nonathletes had a much greater response to load 3 and a greater recovery response than either the swimmers' or runners' ($P<0.01$). The response of the nonathletes to load 2 also was significantly greater than that of the runners ($P<0.05$).

The relative contribution of the initial ventilatory response to the steady-state ventilation was calculated as a percentage (Table 10.0). Significant differences among the groups occurred during the first workload and the recovery phase of the exercise. Both of the other two groups' ventilation had a greater neurogenic component than that of the nonathletes during the first workload. During recovery, the opposite was true with the nonathletes having a relatively greater neurogenic contribution than the swimmers ($P<0.05$). The ratio for the runners was between that for the swimmers and the nonathletes. The neurogenic component seemed to make a larger contribution to

Table 9.0: Mean steady state ventilatory response (S.S.) to exercise at load 1, load 2, load 3 and the mean of 3 loads and recovery: ($L.min.^{-1}\dot{V}I/L.min.^{-1}\dot{V}O_2$)

Group	S.S. Load1	S.S. Load2	S.S. Load3	S.S. Mean	S.S. Recovery
Swimmers (N=13)	16.7±1.1	24.0±1.5	22.0±2.0	20.9±1.0	18.8±1.0
Nonathletes	19.6±1.4 (N=15)	30.9±1.7≠ (N=15)	43.2±4.2* (N=13)	31.2±2.4	28.7±2.4* (N=16)
Runners (N=15)	17.9±1.4	22.3±0.8	25.5±1.2	20.0±0.6	20.0±0.6

(values are Means ±S.E.M.)

* denotes significantly greater than swimmers and runners
≠ denotes significantly greater than runners only

Table 10.0: Percent contribution of the neurogenic ventilatory response to the steady-state ventilation (N/S.S.) at load 1, load 2, load 3 and recovery.

Group	N/S.S. Load1	N/S.S. Load2	N/S.S. Load3	N/S.S. Recovery
Swimmers (N=13)	51.0 ±0.1	30.4 ±0.1	37.1 ±0.1	8.3 ±0.02
Nonathletes	30.5 ±0.1* (N=15)	21.2 ±0.1 (N=15)	23.9 ±0.05 (N=13)	20.6 ±0.1≠ (N=13)
Runners (N=15)	52.3 ±0.1	35.9 ±0.06	36.1 ±0.06	16.4 ±0.02

(values are Means ±S.E.M.)

* denotes significantly less than swimmers and runners
≠ denotes significantly greater than swimmers only

the steady-state response at the lightest workload, relative to the two heavier loads. This was true for all three groups.

Regression analysis was performed on exercise ventilation ($\dot{V}I$) plotted against VT and $\dot{V}O_2$, and exercise heart rate plotted against $\dot{V}O_2$/kg, for each subject. The mean slope of each analysis was then calculated for the three groups. The mean slopes are shown in Table 11.0 and the graphs of the mean regression lines are shown in Figures 7.0, 8.0 and 9.0. The mean slope of $\dot{V}I$ to VT was significantly less for the swimmers compared to the nonathletes or the runners ($P<0.05$). This indicates that the swimmers had a unique breathing pattern consisting of an high tidal volume and a low breathing frequency. The nonathletes were found to have a greater ventilatory response to exercise in terms of ventilation per unit $\dot{V}O_2$ ($\dot{V}I/\dot{V}O_2$) ($P<0.01$). They also had an higher HR/$\dot{V}O_2$ slope than the swimmers and the runners ($P<0.01$). Finally, the swimmers' HR to $\dot{V}O_2$ slope appeared to be greater than the runners' ($P<0.05$).

Correlation of Variables

A correlation matrix of all the variables is given in Appendix E. Several of the variables were significantly correlated. These will be discussed in the appropriate sections of the "Discussion". Table 12.0 shows the correlations found between hypercapnic response, exercise response and breathing

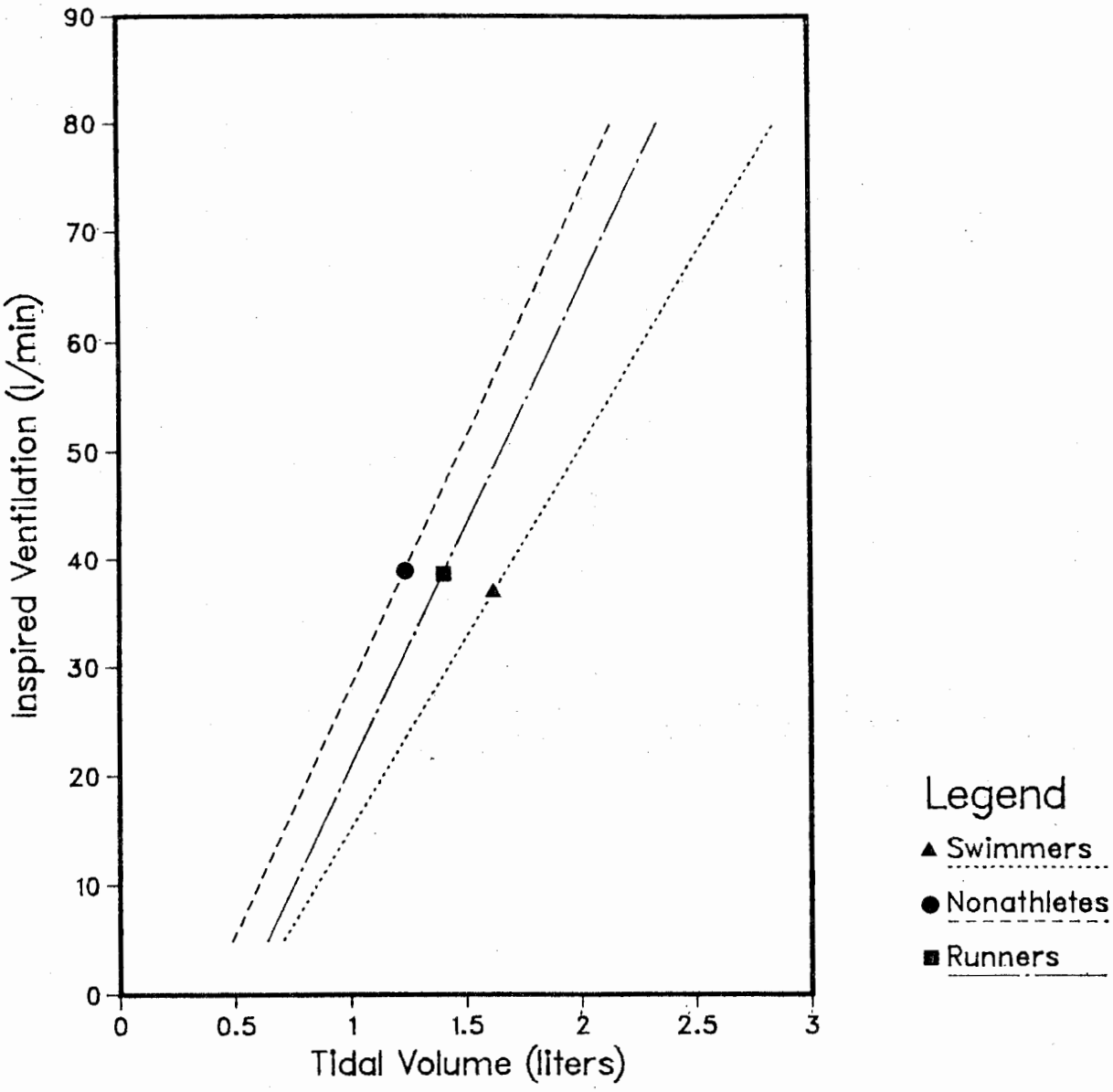


Figure 7.0: Graph of the mean regression line of exercise ventilation ($\dot{V}I$) plotted against VT (the points represent the center of gravity of each regression line).

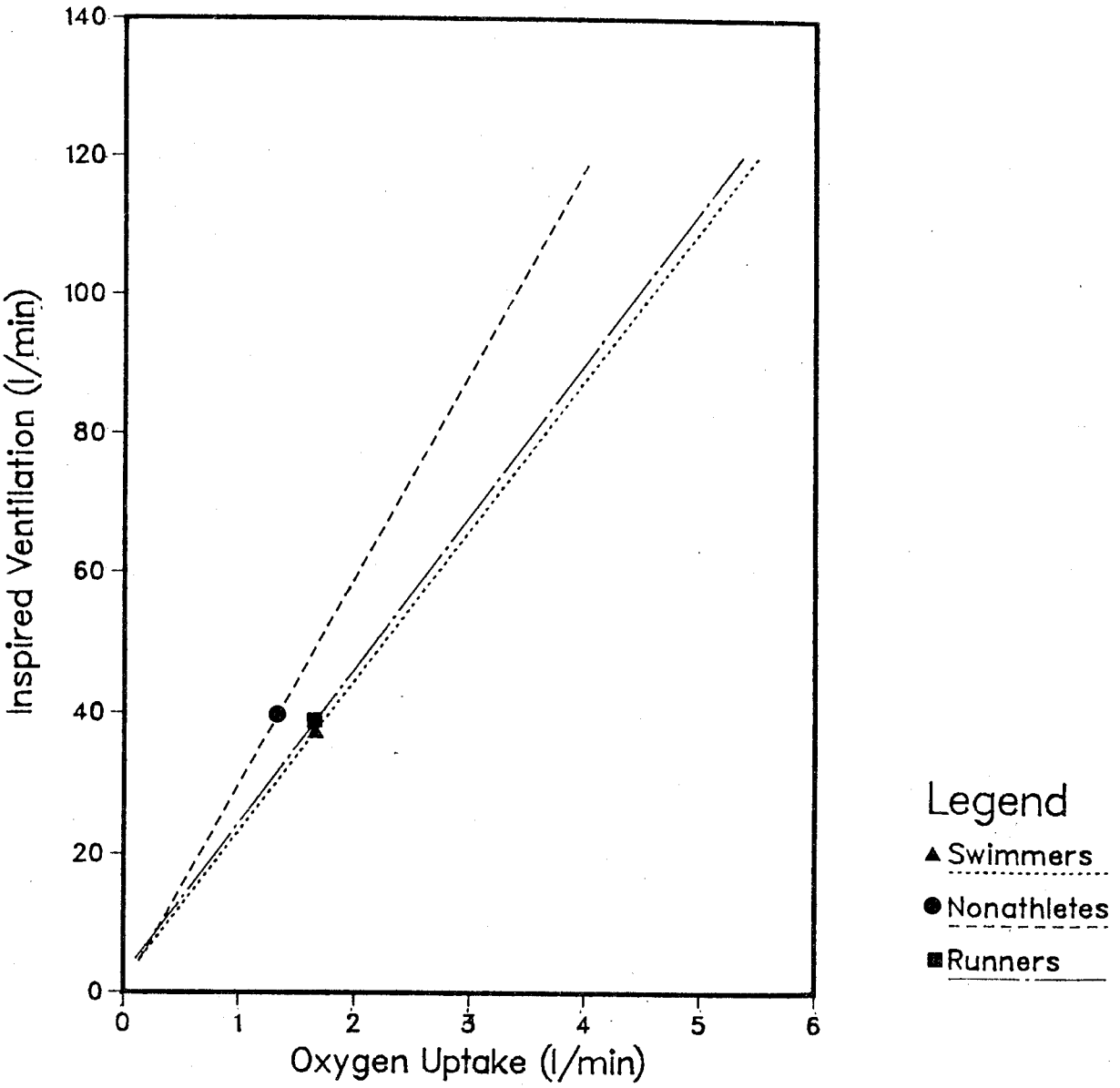


Figure 8.0: Graph of the mean regression line of exercise ventilation ($\dot{V}I$) plotted against $\dot{V}O_2$ (the points represent the center of gravity of each regression line).

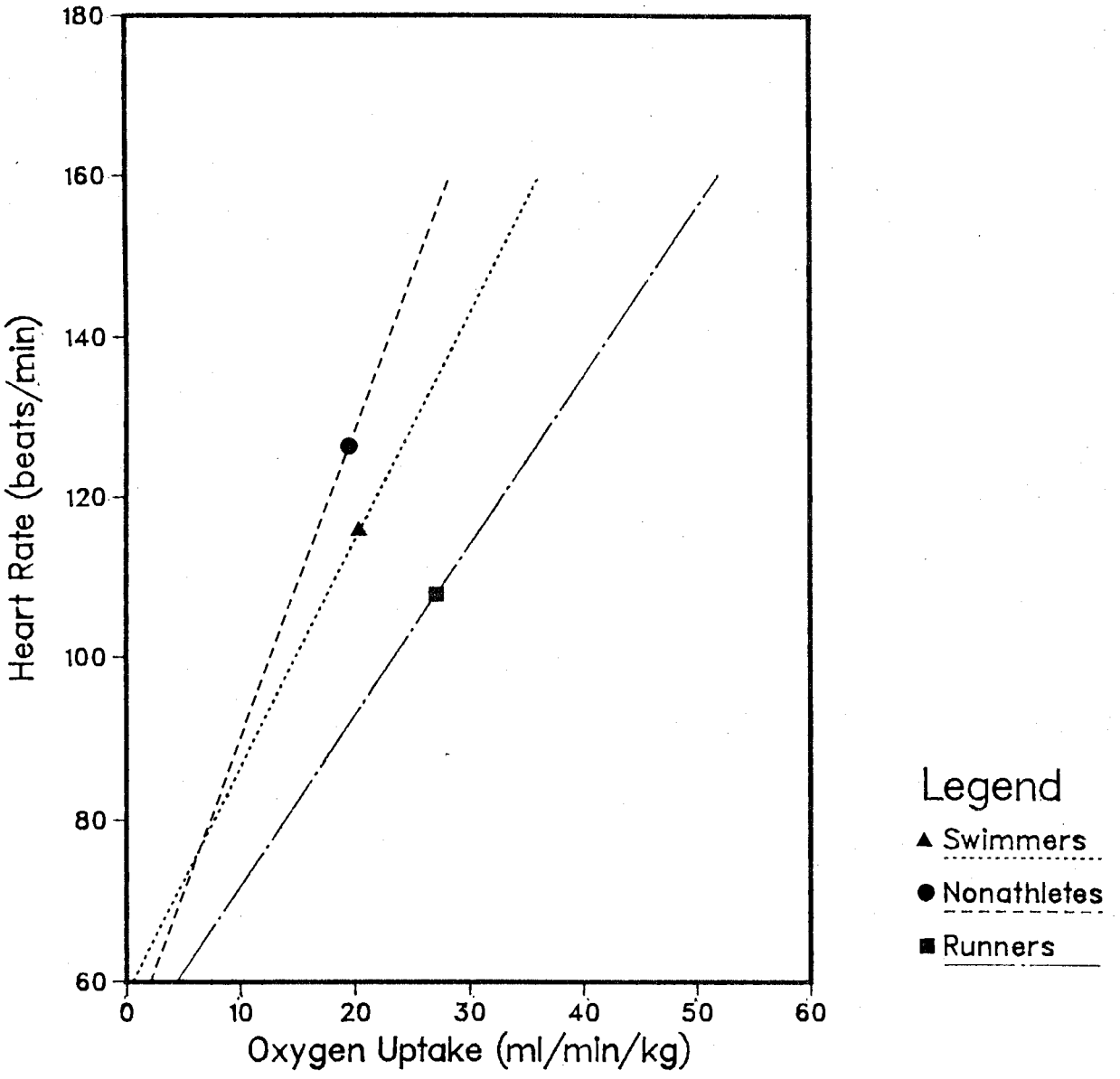


Figure 9.0: Graph of the mean regression line of exercise heart rate (HR) plotted against $\dot{V}O_2$/kg (the points represent the center of gravity of each regression line).

Table 11.0: Calculated mean slopes of exercise $\dot{V}I/VT$ and $\dot{V}I/\dot{V}O_2$; and exercise $HR/\dot{V}O_2/kg$.

Group	N	Slope $\dot{V}I/VT$	Slope $\dot{V}I/\dot{V}O_2$	Slope $HR/\dot{V}O_2/kg$
Swimmers	13	35.0 ±3.7	21.5 ±2.2	2.8 ±0.2#
Nonathletes	16	45.4 ±3.9≠	29.8 ±1.4*	3.8 ±0.2*
Runners	15	44.2 ±2.0≠	21.9 ±0.6	2.1 ±0.1

(values are Means ±S.E.M.)

* denotes significantly greater than swimmers and runners
≠ denotes significantly greater than swimmers only
# denotes significantly greater than runners only

pattern, and aerobic fitness (PWC_{170}/kg and HR/VO_2/kg) and lung function ($FEV_{1.0}$ and VC). The ventilatory response to CO_2 was significantly correlated to $FEV_{1.0}$ ($P<0.05$) and nonsignificantly correlated to aerobic fitness ($HR/\dot{V}O_2$/kg) ($P<0.1$). The steady-state ventilatory response to exercise ($VI/\dot{V}O_2$) was significantly correlated to both aerobic capacity (PWC_{170}/kg and $HR/\dot{V}O_2$/kg) and lung function ($FEV_{1.0}$ and VC) ($P<0.05$). Significant correlations were also found between breathing pattern (VI/VT) and lung function ($FEV_{1.0}$ and VC) ($P<0.05$).

Table 12.0: Correlations between hypercapnic response, exercise response and breathing pattern, and aerobic fitness (PWC_{170}/kg and $HR/\dot{V}O_2$/kg) and lung function ($FEV_{1.0}$ and VC).

CO_2 response ($VI/PETCO_2$)	Exercise response ($VI/\dot{V}O_2$)	Breathing pattern (VI/VT)
	PWC_{170}/kg	
($HR/\dot{V}O_2$/kg P<0.1)	$HR/\dot{V}O_2$/kg	
$FEV_{1.0}$	$FEV_{1.0}$	$FEV_{1.0}$
	VC	VC

V. Discussion

The primary objective of this study was to determine whether any of several respiratory variables were unique to a population of swimmers. Many researchers have examined various aspects of ventilation, but this study appears to be unprecedented in its attempt to identify and relate such a wide range of factors.

Subject Data

The swimmers and non-athletes were successfully matched for age, height and weight. The male swimmers were significantly heavier than the male runners. No relation between body weight and hypercapnic drive was observed by Patrick and Howard (1972) and Irsigler (1976), but Hirschman and colleagues (1975) did find a positive correlation. No correlation was found in this study. The skinfold measurments of the swimmers were between those of the runners and non-athletes, although they were not significantly different from the non-athletes. These results are in agreement with other data on swimmers (Cook and Brynteson, 1971; Plowman, 1975), runners (Costill, 1972; Wilmore and Brown, 1974) and non-athletes (Katch and McArdle, 1983).

The sum of six skinfold measurements was used as an indicator of adiposity (Yuhasz, 1962). Estimation of body

fatness (percent fat) from prediction equations using skinfolds has recently been shown to be unreliable (Martin, 1984) and a considerable amount of the variability in the body fat estimates depends on the prediction equation used (Lohman et al., 1984). Therefore, transformation of skinfolds to percent body fat is of little value. However, skinfold caliper data is still useful in monitoring training effects and in showing the external adipose tissue patterning (Lohman et al., 1984). In effect, the skinfold measurements could be considered to reflect the type of activities in which the subjects were involved. Since fat adds extra weight, one would expect distance runners to have low body fat (indicated by low skinfold measurements) to minimize the work of running. On the other hand, fat is bouyant in water and as a result, an individual with a greater degree of adiposity expends less energy to keep the body floating (Astrand and Rodahl, 1979). Therefore, it could be an advantage for swimmers to have more body fat than other athletes.

Acid-Base Status

The $PaCO_2$ and standard bicarbonate values reported in this study are higher than would be expected for arterial blood (Bondi and VanLiew, 1973; Forster et al., 1972). Poor sampling technique or problems inherent to the sampling method may have resulted in desaturation of the oxyhemoglobin. The error introduced by desaturation would lead to determination of an

higher $PaCO_2$ and bicarbonate than actually existed in the arterial blood (Nunn, 1977). It is also possible that the ear blood may have been venous. Since PO_2 was not measured, it is not possible to confirm the source of error. Despite the high values obtained for $PaCO_2$ and bicarbonate, the means of those parameters and the mean pH were virtually identical for the three groups. Thus, the indication is that the three groups of subjects maintained similar acid-base levels at rest. Presuming that the swimmers or the runners are subjected to increased alveolar PCO_2 during their activities, it is evidently not sufficient to cause either elevation of resting blood CO_2 tension or metabolic compensation by bicarbonate buffer. Data on trained underwater swimmers reported by Goff and Bartlett (1957) lends support to this argument. Although the underwater swimmers had elevated end-tidal PCO_2 during swimming, they were not different from the untrained control subjects at rest.

In spite of the fact that there were no measurable differences in mean acid-base values among the three groups, there appeared to be some evidence of a relationship between standard bicarbonate levels and aerobic fitness. There was found to be a trend to positive correlations between standard bicarbonate and both PWC_{170}/kg and the slope from the regression of heart rate on $\dot{V}O_2/kg$ ($P<0.1$)

Lung Function

The results from this study support the hypothesis that swimmers have different pulmonary functions than non-swimmers. Both the VC and $FEV_{1.0}$ of the swimmers were greater than those of either the runners or the non-athletes. These findings verify the conclusions of other authors (Novak, et al., 1968; Shephard, et al., 1974) regarding the lung functions of swimmers compared to untrained controls. The lack of any difference in VC and $FEV_{1.0}$ between the runners and non-athletes is not in agreement with some previous reports (Kaufman, et al.,1974; Novak, et al., 1968) but is supported by others (Mahler, et al., 1982). Confirmation of the positive effect of physical training on ventilatory capacity (Shapiro, et al.,1964) is suggested by the large MVV attained by both the swimmers and the runners. Further support is provided by the significant correlation shown between MVV and aerobic capacity.

The fact that the swimmers appear to have distinct pulmonary functions is not surprising. Swimming presents limitations to breathing that could possibly result in functional adaptations. In fact, Hong et al. (1969) ascertained that the total work of breathing increases during submersion in water by approximately 60 percent. This is due to increases in both flow-resistive and elastic forces. Hydrostatic pressure exerted on the thorax creates additional work, making inspiration and breathholding more difficult. Expiration takes

place underwater and involves a modified Valsalva manoeuvre. The water pressure at the mouth causes an increase in resistance to airflow (Faulkner, 1966; Holmer, 1974). The cumulative affects of stressing the respiratory muscles during swim training could induce strength and endurance gains in these muscles. There is some evidence of improvements in respiratory muscle power due to specific training affects (Leith and Bradley, 1976). In addition, Korean female breathhold divers (amas) had vital capacities which were much larger than predicted values. This was found to be due entirely to their greater inspiratory capacity (Song et al., 1963). The greater VC and $FEV_{1.0}$ of the swimmers in this investigation implies, respectively, greater inspiratory and expiratory muscle strength.

Response to CO_2

Although the swimmers had the lowest mean regression slope of ventilation on $PETCO_2$, the difference was not significant. Therefore, the hypothesis that swimmers would have a reduced ventilatory response to CO_2 relative to non-swimmers is not corroborated. Existing reports dealing specifically with swimmers give mean slopes of 1.43 (l/min.mmHg) (Ohkuwa et al., 1980) and 1.48 (l/min.mmHg) (Heigenhauser, et al., 1983) for subject values, very similar to the value of 1.35 (l/min.mmHg) found for the swimmers in this study. The present results confirm the findings of the other two studies that swimmers do not exhibit a ventilatory response to CO_2 significantly

different from the norm.

Since hypercapnic response may be directly related to body size (Cameron, 1979), the slope of the regression line was divided by the subject's VC to normalize the response with respect to lung volume. Although the normalized response of the swimmers was approximately 25 percent lower than that of the nonathletes, the differences among the groups were again not significant.

The hypothesis that there is no difference in absolute ventilatory response to CO_2 between men and women is substantiated by the results of this study. These results agree with the work of Hirschman and colleagues (1975) and Haywood and Bloeke (1969), but not Irsigler (1976) or Patrick and Howard (1972). However, when the slope of the regression line was divided by VC to adjust for differences in subject size (Cameron, 1979), the females demonstrated a significantly greater response than the males. This implies the opposite effect from previously reported differences between males and females, in which females had a lower absolute ventilatory response (Irsigler, 1976; Patrick and Howard, 1972). However, it is possible that the reduced response of females observed by Irsigler (1976) and Patrick and Howard (1972) were related to the smaller VC in females, rather than sex per se. Similarly, in the present study, the greater ventilatory response shown by the females, when the results were normalized with respect to VC, do not necessarily represent a true sex difference. The females in

this study were significantly less aerobically fit as a group than the males. It has been reported by Miyamura and colleagues (1976) that athletes with a high aerobic capacity have a significantly lower hypercapnic response than unfit subjects. The results of this investigation also suggest a correlation ($P<0.1$) between CO_2 response and aerobic fitness ($HR/\dot{V}O_2/kg$). It may be that the apparent disparity in the normalized ventilatory response to CO_2 is due to the differences in aerobic fitness. It is not possible to make any comparisons with, or draw any conclusions, in terms of the above two investigations (Irsigler, 1976; Patrick and Howard, 1972), since fitness and VC were not reported.

Martin and coworkers (1978) identified a positive correlation between the ventilatory response to CO_2 and the exercise ventilation of a group of endurance runners. The present results appear to follow a similar trend, since the ventilatory response to CO_2 was found to be positively correlated to the steady-state $\dot{V}I/\dot{V}O_2$ at the first and third workloads. The apparent link between exercise $\dot{V}I$ and CO_2 response may be related to aerobic fitness. This is suggested by what appears to be a trend to a positive correlation between CO_2 response and the slope of the regression of heart rate on $\dot{V}O_2$ observed in the present investigation ($P<0.1$). In addition, both ventilatory response to CO_2 and exercise $\dot{V}I$ were correlated ($P<0.05$) to expiratory power (represented by $FEV_{1.0}$). The above observations suggest that ventilatory response to both CO_2 and

exercise (steady-state) could be a function of $FEV_{1.0}$, fitness, or a combination of the two.

Aerobic Capacity

The estimation of aerobic capacity, in terms of both the absolute PWC_{170} and the PWC_{170} per kilogram body weight of the subjects, was similar to previously reported data on swimmers (Cunningham and Eynon, 1975), runners and nonathletes (CAPHER,1969). In addition, when cardiovascular fitness was determined by relating heart rate to $\dot{V}O_2$ during the bicycle exercise (Table 11.0) the results agreed with those of Astrand and Rodahl (1979). They claim that, compared to untrained individuals, trained subjects would have a lower heart rate at any given $\dot{V}O_2$. According to the present results, the aerobic fitness level of the swimmers was between that of the other two groups. However, due to training specificity, it is probable that physical work capacity measured during cycling is not representative of a swimmers' actual aerobic power. The problem exists with using a bicycle ergometer to test athletes involved in an activity as divergent from cycling as swimming. Cycling uses primarily the leg muscles, whereas swimming engages practically all the muscle groups of the body to some extent. Competitive swim training departs from general recreational swimming in that the leg kicks are deemphasized, and most of the work is done by the arms (Holmer, 1974).

The following two studies are examples illustrating how oxygen uptake is constrained by training specificity. Reybrouck et al. (1975) had competitive and recreational swimmers perform arm, leg and combined arm-leg ergometry. They observed that the ratio of peak arm / treadmill $\dot{V}O_2$ was significantly greater for the competitive swimmers. It was suggested that the higher ratio was due to the relatively greater conditioning of the arms of the competitive swimmers versus the controls. Another investigation conducted by Magel et al., (1975) found that subjects who reached similar maximal oxygen uptakes during running differed significantly when swimming with arms only. The swim-trained subjects attained a much higher maximum $\dot{V}O_2$ than the control subjects.

It is possible that the runners' work capacity was also underestimated by the bicycle ergometer exercise. When treadmill and bicycle exercise were compared, oxygen uptake in distance runners was 14 percent higher on the treadmill (Verstappen et al., 1982). Despite the above arguments, the lower aerobic capacity of the swimmers compared to the runners, implied by the results, was expected. Running involves the use of larger muscle groups than swimming and therefore imposes a greater demand on the central cardiovascular system, and results in a larger training effect in terms of oxygen transport (Astrand and Rodahl, 1979).

Transition and Steady-State Ventilatory Response to Exercise

Steady-state Ventilation

The transition (neurogenic) and steady-state ventilatory responses to the bicycle ergometry were calculated in a manner similar to that reported by Lally et al. (1974). The results for three nonathletes were eliminated for the third workload and the means recalculated and analyzed. This was done because their calculated steady-state $\dot{V}I$ appeared too high to be aerobic and therefore were not representative of a steady-state. Calculation of the $\dot{V}I$ to $\dot{V}O_2$ ratio at the third workload confirmed that these three subjects were hyperventilating. Although the results were modified by eliminating non-steady-state $\dot{V}I$, the significances from statistical analyses were not changed. The steady-state responses in the present study followed the same trend as that found by Lally et al. (1974), with the runners and the swimmers having less response than the nonathletes at all workloads and during recovery (significant at all but the first workload). The results from the regression of ventilation against $\dot{V}O_2$/kg (Table 10.0) provide confirmation that the swimmers and the runners had lower exercise ventilation per unit metabolic rate. Numerous authors have presented similar data on athletes (Byrne-Quinn et al., 1971; Goff and Bartlett, 1957; Heigenhauser et al., 1983; Martin et al., 1979). It has been suggested that the lower ventilation of athletes at any given

$\dot{V}O_2$ in moderate to heavy work is a result of training. The usual explanation is that training causes a decrease in metabolic acidosis (Dempsey et al., 1977; Jones, 1984) or an increase in efficiency in terms of oxygen utilization (Astand and Rodahl, 1979; Costill, 1972). The aerobic fitness level, as reflected by either the PWC_{170} test or the heartrate to $\dot{V}O_2$ curve, was found to be significantly correlated to $\dot{V}I/\dot{V}O_2$ (inspired ventilation per unit $\dot{V}O_2$) in the present investigation. This would seem to imply the presence of a cardiovascular training effect on exercise ventilation. However, the aerobic fitness level of the swimmers was between that of the runners and nonathletes. Therefore, it seems likely that an additional mechanism is operating in the swimmers. One explanation could be that the low exercise $\dot{V}I$ of the swimmers is at least partially due to a specific respiratory muscle training effect. This argument is consistent with the greater lung capacities of the swimmers ($FEV_{1.0}$ and VC), and the evidence that both steady-state $\dot{V}I$ and the $\dot{V}I/\dot{V}O_2$ slope were correlated to $FEV_{1.0}$.

Neurogenic Ventilatory Response

The neurogenic ventilatory response to exercise was not significantly different among the three groups at any of the workloads. This is in agreement with the results of Lally and coworkers (1974) for SCUBA divers, endurance runners and control subjects. However, the trend toward a lower neurogenic response

in athletes and SCUBA divers noted by Lally and coworkers (1974) was not evident in either the runners or swimmers of this study. The recovery phase presented an exception, with the nonathletes responding more than the swimmers ($P<0.05$). Not all the subjects demonstrated a neurogenic response; ventilation either did not increase in the first 15 seconds during transition between workloads, or actually decreased. This is not unusual as it has been shown that a neurogenic response may not always be present (Beaver and Wasserman, 1968; Broman and Wigertz, 1971; Jensen et al., 1971). Since it is not possible to have a true negative neurogenic response (it is negative as a result of the calculation method), those results which appeared as negative values were: (1) changed to zero response and (2) eliminated. The mean values for each workload were then recalculated and analyzed. Again, elimination of the negative values did not affect the statistical significance of the results.

The neurogenic response was examined as a percentage of the steady-state ventilation (Table 10.0). The general trend for all three groups was for the ratio to be greatest at the lightest workload and less by approximately the same amount at the two heavier loads. The larger neurogenic component at the first workload may be a function of leg movement, since this was the only time during the exercise test where there was a transition from rest to pedalling. The nonathletes' initial ventilation made a significantly smaller contribution to the overall response at the lightest load, compared to the other subjects.

Nonathlete percentages also appeared to be less at the higher workloads, but differences were not significant. In this sense, the initial neurogenic response of the swimmers and runners constituted a greater portion of the overall ventilatory response to exercise than did that of the nonathletes. This is the opposite effect to that inferred by Lally et al. (1974), and would suggest that the lower steady-state ventilatory response to exercise in these two groups is probably humoral, rather than neurogenic in origin. A reverse situation was found during the transition from load 3 to recovery. In this case, the nonathletes' absolute neurogenic component was greater and constituted a relatively larger portion of their steady-state ventilation, relative to the other two groups.

The workloads imposed on the subjects in this investigation were compared to those of Lally et al. (1974) by looking at $\dot{V}O_2$ at each load. It appeared that walking at the two fastest speeds on the treadmill (Lally et al., 1974) was roughly equivalent to cycling at the two lighter loads on the bicycle ergometer in terms of steady-state $\dot{V}O_2$. Although there are obvious limitations in comparing physiological results from two different types of exercise, it is noteworthy that some agreements were found. At the workloads for which the $\dot{V}O_2$ was similar, the magnitudes of the initial and steady-state $\dot{V}I$ and the ratios of neurogenic to steady-state $\dot{V}I$ were within the same ranges in both studies. For example, for the range of $\dot{V}O_2$ between 1.0 and 1.5 liters/minute (middle speed on the treadmill

and first workload on the bicycle), the initial $\dot{V}I$ varied from 7.0 to 9.6 ($L.min.^{-1}\dot{V}I/L.min.^{-1}\dot{V}O_2$) in the present investigation and from 7.4 to 9.3 ($L.min.^{-1}\dot{V}I/L.min.^{-1}\dot{V}O_2$) in the other. Similarly, the range of the relative neurogenic component was 38.2 to 56.0 versus 45.0 to 48.0 respectively.

Breathing Pattern

The steady-state ventilation of the swimmers and the runners was similar in magnitude, but the swimmers achieved it differently. The breathing pattern during exercise is indicated by the regression of $\dot{V}I$ on VT (Table 11.0). The mean slope for the swimmers was significantly less than that for the other two groups and demonstrates that they breathed with a higher VT and a lower Bf. This author was unable to find any information dealing specifically with the ventilatory pattern of swimmers compared to that of nonswimmers during treadmill or bicycle exercise. However, data have been reported on the performance of SCUBA divers (Lally et al., 1974) and runners (Martin et al., 1979) during treadmill exercise that is in agreement with the present results. That is, the divers' exercise ventilation was reduced and was characterized by a high tidal volume and a low respiratory frequency, whereas the runners' ventilatory pattern did not differ significantly from that of the controls.

It seems reasonable to hypothesize that the unique exercise breathing pattern exhibited by swimmers could have developed in

response to the restrictions imposed by swim training. Exercise in general increases respiratory effort, both in terms of mechanical work and perceived magnitude. Because the body is submerged in water, swimming creates additional demands on ventilation by increasing the work done by the respiratory muscles. As stated previously in the discussion of lung function, this is due to increases in both elastic and flow-resistive forces (Faulkner, 1966; Holmer,1974; Hong,1969). Another restriction in swimming is that breathing frequency tends to be synchronized to stroke and the duration of inspiration is shorter (Faulkner, 1966; Holmer, 1974). Competitive swimmers also often voluntarily limit their inspiration to alternate strokes. Two different observations of swimming subjects appear to suggest that resistance to airflow, especially during expiration, may be of greater importance to the increased ventilatory effort than elastic forces. Firstly, Holmer (1974) observed that the inspiratory reserve volume was utilized exclusively to increase VT in water, whereas in air, both the expiratory reserve volume and the inspiratory reserve volume were used equally. Secondly, during maximum swimming, subjects breathed at a slower rate and with a greater VT than during maximum running (Holmer, 1974; McArdle et al., 1971). This kind of pattern is similar to one adopted by people with chronic airway obstruction in whom high expiratory resistance is associated with a low breathing frequency and a marked reduction in inspiratory duration (Jones, 1984). The above observations

imply that the alterations in respiratory pattern that occur while swimming may be an attempt to minimize the work of breathing, especially during expiration.

The particular breathing pattern adopted during swimming may become a learned response through repetition, just as other types of movement patterns are learned (Magill, 1980). Therefore, even while exercising on land, swimmers could continue to exhibit an unusual ventilatory rhythm. This learned behaviour of high VT and low Bf would seem at first to oppose the concept of minimization of respiratory effort. On the other hand, the swimmers' different breathing pattern, while learned, may also be the most energy efficient as a result of their greater lung capacities. Another possibility is that the ventilatory behaviour of the swimmers is not a result of learning at all, but is entirely due to different pulmonary functions. The results from this study suggest that swimmers have greater lung capacities (VC) than nonswimmers. In addition, the positive correlation between the slope from the regression of $\dot{V}I$ on VT and VC and $FEV_{1.0}$ found in the present investigation lends further support to the above argument.

Summary

This study has examined several respiratory variables and attempted to identify those which were unique to a population of swimmers. The primary hypothesis that swimmers would have a reduced ventilatory response to CO_2 due to their training has

not been validated. However, the hypotheses that swimmers have a significantly lower ventilatory response to exercise than nonathletes, and that swimmers have a distinctive breathing pattern, consisting of a high VT and low Bf have been validated. The results also support the hypothesis that swimmers have greater lung functions than nonswimmers; in this case significantly greater VC and $FEV_{1.0}$.

A particularly interesting aspect of the present study was the relationships found among ventilatory response to CO_2 and exercise, aerobic fitness ($HR/\dot{V}O_2/kg$) and expiratory power ($FEV_{1.0}$). Exercise response was significantly correlated to both aerobic fitness (PWC_{170} and $HR/\dot{V}O_2/kg$) and expiratory power. There was also a significant correlation between hypercapnic response and expiratory power, and a trend towards a negative correlation to aerobic fitness ($HR/\dot{V}O_2/kg$). It seems apparent from these relationships that ventilatory drive is modified by both general aerobic ($HR/\dot{V}O_2$) and specific swimming ($FEV_{1.0}$) training effects.

The results suggest that swimming requires some specific ventilatory adaptations. Neither lung function or breathing pattern was related to aerobic capacity. Therefore, it would appear that the differences in lung function and breathing pattern, measured between the swimmers and nonswimmers, are due to a training effect specific to swimming.

REFERENCES

Arkinstall,W.W.,K.Nirmel,V.Klissouras and J.Milic-Emili. Genetic differences in the ventilatory response to inhaled CO_2. J.Appl.Physiol.36:6-11,1974.

Asmussen,E. Ventilation at transition from rest to exercise. Acta.Physiol.Scand.89:68-68,1973.

Astrand,P.O. and K.Rodahl. Textbook of Work Physiology. New York: McGraw Hill, 1979.

Beaver,W.L. and K.Wasserman. Transients in ventilation at start and end of exercise. J.Appl.Physiol. 25(4):390-399,1968.

Bennett,F.M.,P.Reischl,F.S.Grodins,S.M.Yamashiro and W.E. Fordyce. Dynamics of ventilatory response to exercise in humans.J.Appl.Physiol.51(1):194-203,1981.

Blum,J.,D.Kanarek,B.Callahan,N.Braslow and H.Kazemi. The effect of training on CO_2 ventilatory responsiveness in normal subjects. Am.Rev.Resp.Dis.119(Suppl.): 291,1979.

Bondi,K.R.,H.D.VanLiew. Fluxes of CO_2 in the lung gas studied by continuously recorded arterial pH. J.Appl.Physiol. 35:42-46, 1973.

Bouhuys,A. The Physiology of Breathing. New York: Grune and Stratton,Inc., 1977.

Brackett,N.,J.Cohen and W.Schwartz. Carbon dioxide titration curve in normal man. New Eng.J.Med.272:6-12,1965.

Brackett,N.C.,C.F.Wingo,O.Muren and J.T.Solano. Acid-base response to chronic hypercapnia in man. New.Eng.J.Med.280: 124-130,1969.

Bradley,B.L.,J.Mestas and K.M.Unger. The effect on respiratory drive of a prolonged physical conditioning program. Am.Rev.Resp.Dis.122:741-746,1980.

Brischetto,M.J.,R.P.Millman,D.D.Peterson,D.A.Silage and A.I.Pack. Effect of aging on ventilatory response to exercise and CO_2. J.Appl.Physiol.56(5):1143-1150,1984.

Broman,S. and O.Wigertz. Transient dynamics of ventilation and heart rate with step changes in workload from different load levels. Acta.Physiol.Scand.81:54-74,1971.

Brousolle,B.,E.Bensimon,A.Michaud and I.Lonjon. Sensibilite' ventilatoire au gaz carbonique des plongeurs sous-marin. J.Physiol.Paris. 60:410,1968.

Byrne-Quinn,E.,J.V.Weil,I.E.Sodal,G.F.Filley and R.F.Grover. Ventilatory control in the athlete. J.Appl.Physiol. 30:91-98,1971.

Cameron,I.R. The investigation of the respiratory response to hypercapnia. Bull.Eur.Physiopathol.Respir. 15(suppl.): 213-221, 1979.

C.A.P.H.E.R. The Physical Fitness Performance and Work Capacity of Canadian Adults Aged 18 to 44 Years. Ottawa: The Canadian Association for Health, Physical Education and Recreation, 1969.

Casaburi,R., B.J.Whipp, K.Wasserman, W.L.Beaver and S.N.Koyal. Ventilatory and gas exchange dynamics in response to sinusoidal work. J.Appl.Physiol. 42:300-311,1977.

Comroe,J.H. Physiology of Respiration. Chicago: Year Book Medical Publishers Inc., 1974.

Cook,B. and P.Brynteson. Effect of a season of collegiate swimming competition and training on selected responses. Research Quart.44(1):63-70,1971.

Costill,D.L. Physiology of marathon running. J.A.M.A. 221(9):1024-1029,1972.

Cotes,J.E. Lung Function-Assessment and Application in Medicine. Philadelpia: F.A.Davies Co., 1965.

Crosbie,W.A.,J.A.Reed and M.C.Clarke. Functional characteristics of the large lungs found in commercial divers. J.Appl. Physiol. 46(4):639-645,1979.

Cunningham,D.A. and R.B.Eynon. Working capacity of swimmers 10-16 years of age. Swimming Technique.12(2):39-43,1975.

D'angelo,E. and G.Torelli. Neural stimuli increasing respiration during different types of exercise. J.Appl.Physiol. 30(1):116-121,1971.

Dejours,P. The regulation of breathing during muscular exercise in man. A neurohumoral theory. In: D.J.C.Cunningham and B.B.Lloyd (Eds). The Regulation of Human Respiration. Oxford: Blackwell, 1963.

Dejours.P. Control of respiration in muscular exercise. In: W.O.Fenn and H.Rahn (Eds). Handbook of Physiology: Respiration. Washington: American Physiological Society. 1:631-648, 1964.

Dempsey,J.A. CO_2 response: Stimulus definition and limitations. Chest. 70(suppl.):114-118, 1976.

Dempsey,J.A.,N.Gledhill,W.G.Meddan,H.V.Forster,P.G.Hanson and A.D.Claremont. Pulmonary adaptation to exercise: effects of exercise type and duration, chronic hypoxia and physical training. Ann.N.Y.Acad.Sci.301:243-261,1977.

Dempsey,J.A., D.A.Pelligrino, D.Aggarwal and E.B.Olson. The brain's role in exercise hyperpnea. Med.Sci.Sports. 11(2) :213-220, 1979(a).

Dempsey,J.A., E.H.Vidruk and S.M.Mastenbrook. Pulmonary control systems in exercise. Fed.Proc. 39:1498-1505, 1979(b).

Dixon,W.J. BMDP Statistical Software. Berkley Cal: University of California Press, 1981.

Edwards,D.A.W., W.M.Hammond, M.J.R.Healey, J.M.Tanner and R.H.Whitehouse. Design and accuracy of calipers for measuring subcutaneous tissue thickness. Brit.J.Nutr.. 9:133-143, 1955.

Faulkner,J.A. Physiology of swimming. Res.Quart. 37:41-54,1966.

Florio,J.T.,J.B.Morrison and W.S,Butt. Breathing pattern and ventilatory response to carbon dioxide in divers. J.Appl. Physiol. 46:1076-1080,1979.

Forster,H.V.,J.A.Dempsey,J.Thomson,E.Vidruk and G.A.doPico. Estimation of arterial PO_2, PCO_2, pH and lactate from arterialized venous blood. J.Appl.Physiol. 32(1):134-137,1972.

Froeb,H.F. Ventilatory response of SCUBA divers to CO_2 inhalations. J.Appl.Physiol.16:8-10,1960.

Fujihara,Y.,J.R.Hildebrandt and J.Hildebrandt. Cardiorespiratory transients in exercising man. 1.Tests of superposition. J.Appl.Physiol.35(1):58-67,1973(a).

Fujihara,Y.,J.R.Hildebrandt and J.Hildebrandt. Cardiorespiratory transients in exercising man. 2.Linear models. J.Appl.Physiol.35(1):68-76,1973(b).

Goff,L.G. and R.G.Bartlett. Elevated end-tidal CO_2 in trained underwater swimmers. J.Appl.Physiol.10:203,1957.

Graham,T.,B.Wilson,M.Sample,J.Van Dijk and B.Goslin. Short and long term work responses during hypercapnia. Med.Sci.Sports. 12:106,1980.

Haywood,C. and M.E.Bloeke. Respiratory response of healthy young women to carbon dioxide inhalation. J.Appl.Physiol. 27:32-35,1969.

Heigenhauser,G.J.F.,N.B.Oldridge and N.L.Jones. The CO_2 responsiveness and ventilatory response to leg and arm exercise in female swimmers. Resp.Physiol.53:263-272,1983.

Hey,E.N.,B.B.Lloyd,D.J.C.Cunningham,M.G.M.Jukes and D.P.G. Bolton. Effects of various respiratory stimuli on the depth and frequency of breathing in man. Resp.Physiol. 1:193-205,1966.

Hirschman,C.A.,R.E.McCullough and J.V.Weil. Normal values for hypoxic and hypercapnic ventilatory drives in man. J.Appl. Physiol. 38(6):1095-1098,1975.

Holmer,I. Oxygen uptake during swimming in man. J.Appl.Physiol. 33:502-509,1972.

Holmer,I. Physiology of swimming man. Acta Physiol. Scand. (Suppl.):407, 1974.

Holmer,I.,E.M.Stein,B.Saltin,B.Ekblom and P.O.Astrand. Hemo-dynamic and respiratory responses compared in swimming and running. J.Appl.Physiol.37:49-54,1974.

Hong,S.K.,P.Cerretelli,J.C.Cruz and H.Rahn. Mechanics of respiration during submersion in water. J.Appl.Physiol. 27:535-538,1969.

Irsigler,G.B. Carbon dioxide response lines in young adults: the limits of the normal response. Am.Rev.Resp.Dis.114:529-536,1976.

Jensen,J.I. Neural ventilatory drive during arm and leg exercise. Scand.J.Clin. Lab.Invest. 29:177-184, 1972.

Jensen,J.I.,H.Vejby-Christensen and E.S.Petersen. Ventilation in man at onset of work employing different standardized starting orders. J.Appl.Physiol.13:209-220,1971.

Jones,N.L. Dyspnea in exercise. Med.Sci.Sports. 16(1) :14-19,1984.

Katch,F.and W.D.McArdle. Nutrition, Weight Control and Exercise. Philadelphia: Lea and Rebiger, 1983.

Kaufmann,D.A.,E.W.Swensen,J.Ferel and A.Lucas. Pulmonary function of marathon runners. Med.Sci.Sports.6:114-117,1974.

Kelley,M.A.,M.D.Laufe,R.P.Millman and D.D.Peterson. Ventilatory response to hypercapnia before and after athletic training. Resp.Physiol.55:393-400,1984.

Koyal,S.N.,B.J.Whipp, D.Huntsman, G.Bray and K.Wasserman. Ventilatory responses to the metabolic acidosis of treadmill

and cycle ergometry. J.Appl.Physiol. 40:864-867,1976.

Lally,D.A.,F.W.Zechman and R.A.Tracy. Ventilatory responses to exercise in divers and nondivers. Resp.Physiol. 20:117-129,1974.

Leith,D.E. and M.Bradley. Ventilatory muscle strength and endurance training. J.Appl.Physiol. 41:508-516, 1976.

Levine,S. Ventilatory response to muscular exercise. In: D.G. Davies and C.D.Baines(Eds.). Regulation of Ventilation and Gas Exchange. New York: Academic Press, pp.38-67, 1978.

Lohman,T.G., M.L.Pollock, M.H.Slaughter, L.J.Brandon and R.A.Boileau. Methodological factors and the prediction of body fat in female athletes. Med.Sci.Sports. 16(1):92-96, 1984.

Magel,J.R. Comparison of the physiologic response to varying intensities of sub-maximal work in tethered swimming and running. J.Sports Med.Phys. Fitness. 11:203-212, 1971.

Magel,J.R.,G.F.Foglia, 1.D.McArdle, B.Gutin, G.S.Pechar and F.I.Katch. Specificity of swim training on maximum oxygen uptake. J.Appl. Physiol. 38:151-155, 1975.

Magill,R.A. Motor Learning. Wm.C.Brown Co., Dubuque, Iowa, 1980.

Mahler,D.A.,E.D.Moritz and J.Loke. Ventilatory responses at rest and during exercise in marathon runners. J.Appl.Physiol. 52:388-389,1982.

Martin,A. PHd Thesis. Burnaby: Simon Fraser University, 1984.

Martin,B.J.,J.V.Weil,K.E.Sparks,R.E.McCullough and R.F.Grover. Exercise ventilation correlates postively with ventilatory chemoresponsiveness. J.Appl.Physiol.45:557-564,1978.

Martin,B.J.,K.E.Sparks,C.W.Zwillich and J.V.Weil. Low exercise ventilation in endurance athletes. Med.Sci.Sports.11(2): 181-185,1979.

McArdle,W.D., R.M.Glaser, J.R.Magel. Metabolic and cardiorespiratory response during free swimming and treadmill walking. J.Appl.Physiol. 30:733-738, 1971.

Mead,J. Control of respiratory frequency. J.Appl.Physiol. 15:325-336, 1960.

Menitove,S.M.,D.M.Rapoport,H.Epstein,B.Sorkin and R.M.Goldring. CO_2 rebreathing and exercise ventilatory responses in humans. J.Appl.Physiol.56(4):1039-1044,1984.

Miyamura,M,T.Yamashina and Y.Honda. Ventilatory responses to CO_2 rebreathing at rest and during exercise in untrained subjects and athletes. Jap.J.Physiol.26:245-254,1976.

Nie,N.H. Statistical Package for the Social Sciences. New York: McGraw-Hill Book Co., 1975.

Novak,LP.P,R.E.Hyatt and J.F.Alexander. Body composition and physiologic function of athletes. J.Amer.Med.Ass. 205:765-770,1968.

Nunn,J.F. Applied Respiratory Physiology. London: Butterworth and Co. Ltd., 1977.

Ohkuwa,T.,N.Fujitsuka,T.Utsuno and M.Miyamura. Ventilatory response to hypercapnia in sprint and long distance swimmers. Eur.J.Appl.Physiol.43(3):235-241,1980.

Otis,A.B. The work of breathing. In: W.O.Fenn and H.Rahn (Eds), Handbook of Physiology. Washington: American Physiological Society. pp.463-476, 1964.

Patrick,J.M.and A.Howard. The influence of age,sex,body size and lung size on the control and pattern of breathing during CO_2 inhalation in caucasians. Resp.Physiol.16:337-350,1972.

Paulev,P.-E. Respiratory and cardiac responses to exercise in man. J.Appl.Physiol.30:165-172,1971.

Pearce,D.H. and H.T.Milhorn. Dynamic and steadystate respiratory responses to bicycle ergometer exercise. J.Appl.Physiol. 42:959-967,1977.

Plowman,S. Physiological characteristics of female athletes. Swimming Technique.12:10-14,1975.

Raven,P.B. Pulmonary function of elite distance runners. Ann.N.Y.Acad.Sci.301:371-381,1977.

Read,D.J.C. A clinical method for assessing the ventilatory response to carbon dioxide. Aust.Ann.Med.16:20-32,1967.

Reybrouck,T., G.F.Heigenhauser and J.A.Faulkner. Limitations to maximum oxygen uptake in arm, leg and combined arm-leg ergometry. J.Appl.Physiol. 38:774-779,1975.

Sahn,S.A.,C.W.Zwillich,N.Dick,R.E.McCullough,S.Lakshminarayan and J.V.Weil. Variability of ventilatory response to hypoxia and hypercapnia. J.Appl.Physiol.43:1019,1977.

Saltin,B. and P.O.Astrand. Maximal oxygen uptake in athletes. J.Appl.Physiol.23:353-358,1967.

Saunders,N.A.,S.R.Leeder and A.S.Rebuck. Ventilatory response to carbon dioxide in young athletes: a family study. Am.Rev. Resp.Dis.113:497-502,1976.

Schaefer, K.E. The role of carbon dioxide in the physiology of human diving. In: L.G.Goff(Ed). Proceedings of Underwater Physiology Symposium. Washington,D.C.: Natl.Acad.Sci.-Natl.Res.Council, Publ. 377:131-139,1955.

Schaefer,K.E. Respiratory pattern and respiratory response to CO_2. J.Appl.Physiol.13(1):1-14,1958.

Schaefer, K.E. Carbon dioxide effects under conditions of raised environmental pressure. In: P.B.Bennett and D.H.Elliot (Eds). The Physiology and Medicine of Diving. London: Bailliere Tindal. pp.187-206,1975.

Schaefer,K.E.,B.Hastings,R.Carey and G.Nichols. Respiratory acclimatization to carbon dioxide. J.Appl.Physiol. 18:1071-1078,1963.

Schaefer,K.E.,G.Nichols and C.Carey. Acid-base balance and blood and urine electrolytes of man during acclimatization to CO_2. J.Appl.Physiol.19:48-58,1964.

Scoggin,C.H.,R.D.Doekel,M.H.Kryger,C.W.Zwillich and J.V.Weil. Familial aspects of decreased hypoxic drive in endurance athletes. J.Appl.Physiol.44:464-468,1978.

Shapiro,W.,C.E.Johnston, R.A. Dameron and J.L. Patterson. Maximum voluntary ventilatory performance and its limiting factors. J.Appl.Physiol.19:199-203,1964.

Shephard,R.J.,G.Grodin and R.Campbell. Characteristics of medium and long distance swimmers. Eur.J.Appl.Physiol. 32:99-116,1974.

Siggard-Andersen,O., K.Engel,K.Jorgensen and P.Astrup. A micro method for determination of pH,carbon dioxide tension,base excess and standard bicarbonate in capillary blood. Scand. J.Clin.Lab. Invest.12:172-176,1960.

Sinclair,J.D. Neural factors in the control of breathing during exercise. In: R.Porter (Ed). Studies in Neurophysiology . Cambridge: Cambridge University Press,1978.

Song,S.H.,D.H.Kang,B.S.Kang and S.K.Hong. Lung volumes and ventilatory responses to high CO_2 and low O_2 in the ama. J.Appl. Physiol.18(3):466-470,1963.

Stuart,D.G.and W.D.Collings. Comparison of vital capacity and maximum breathing capacity of athletes and non-athletes. J.Appl.Physiol.14(4):507-509,1959.

Turino,G.M.,R.M.Goldring and H.O.Heinemann. The extracellular bicarbonate concentration and the regulation of ventilation in chronic hypercapnia in man. In: G.Nahas and K.E.Schaefer (Eds.)., Carbon Dioxide and Metabolic Regulations. New York: Springer-Verlag. pp.273-279,1974.

van Ypersele de Strihou,C. Acid-base equilibrium in chronic hypercapnia. In:G.Nahas and K.E.Schaefer (Eds.).Carbon Dioxide and Metabolic Regulations. New York: Springer-Verlag. pp.266-279,1974.

Verstappen,F.T.J.,R.M.Huppertz and L.H.E.H.Snoeckx. Effect of training specificity on maximal treadmill and bicycle ergometer exercise. Int.J.Sports.Med.3:43-46,1982.

Vidruk,E.H. and J.A.Dempsey. Peripheral and central nervous system mechanisms controlling exercise-induced breathing patterns. In: R.S.Hutton and D.I.Miller(Eds.). Exercise and Sport Sciences Reviews. Seattle: The Franklin Institute. 8:129-147, 1980.

Wallersteiner,U.C.,J.B.Morrison,C.P.Bolter,P.Rowe and M.L.Conn. Respiratory adjustments in underwater hockey players. Unpublished paper,1980.

Wasserman,K. Testing regulation of ventilation with exercise. Chest. 70(Suppl.):173-178,1976.

Wasserman,K. Breathing during exercise. N.Eng.J.Med. 298: 780-785,1978.

Wasserman,K., A.L.VanKessel and G.G.Burton. Interaction of physiological mechanisms during exercise. J.Appl.Physiol. 22:71-85,1967.

Wasserman,K., B.J.Whipp, S.N.Koyal and W.L.Beaver. Anaerobic threshold and respiratory gas exchange during exercise. J.Appl.Physiol. 35:236-243, 1973.

West,J.B. Respiratory Physiology - The Essentials. The Williams and Wilkins Co., Baltimore, 1979.

Whipp,B.J. Ventilatory control during exercise in humans. Ann.Rev.Physiol. 45:393-413, 1983.

Whipp,B.J. and S.A.Ward. Ventilatory control dynamics during muscular exercise in man. Int.J.Sports Med. 1:146-159,1980.

Wilmore,J.H. and C.H.Brown. Physiological profiles of women distance runners. Med.Sci.Sports. 6(3):178-181,1974.

Yamashiro,S.M., J.A.Daubenspeck, T.N.Lauritsen and F.S.Grodins. Total work rate of breathing optimization in CO_2 inhalation

and exercise. J.Appl.Physiol.. 38:702-709,1975.

Yuhasz,M.S. Physical Fitness and Sports Appraisal. A Laboratory Manual. London, Ontario: University of Western Ontario, 1962.

APPENDIX A

Individual subject data and environmental measures

Swimmers individual subject data and environmental measures

Subject	Height (cm)	Weight (kg)	Age (years)	Bar.Pres. (mmHg)	Temp. (°C)
1	199.2	90.5	19	735	20.0
2	152.4	56.0	19	738	20.0
3	164.3	55.5	20	740	20.0
4	164.7	72.5	22	738	20.0
5	182.1	80.5	21	739	20.0
6	170.0	71.0	20	732	20.0
7	167.6	69.0	20	732	20.0
8	190.5	85.5	20	738	21.0
9	180.3	79.0	20	730	20.0
10	179.4	90.5	20	732	21.0
11	171.1	60.0	21	732	20.0
12	188.9	84.0	20	738	20.0
13	156.2	47.0	18	738	20.0
14	180.3	71.5	18	729	20.0
15	189.4	75.0	23	740	20.0
16	179.0	84.0	21	726	20.0

Nonathletes individual subject data and environmental measures

Subject	Height (cm)	Weight (kg)	Age (years)	Bar.Pres. (mmHg)	Temp. (°C)
1	173.9	59.5	26	731	20.0
2	167.5	65.5	25	733	22.0
3	147.3	57.5	18	738	21.0
4	181.6	69.5	21	730	20.0
5	178.0	55.5	18	740	20.0
6	175.9	71.5	22	731	20.0
7	172.7	54.0	25	735	20.0
8	187.6	77.5	18	740	20.0
9	180.8	94.5	18	740	20.0
10	170.5	66.0	18	738	21.0
11	164.8	59.5	19	740	20.0
12	181.1	74.0	18	739	20.0
13	167.2	56.0	19	736	20.0
14	182.1	81.5	18	740	20.0
15	171.4	72.5	19	732	20.0
16	161.7	74.5	28	730	20.0

Runners individual subject data and environmental measures

Subject	Height (cm)	Weight (kg)	Age (years)	Bar.Pres. (mmHg)	Temp. (°C)
1	176.5	67.5	24	739	20.0
2	165.6	56.0	19	735	20.0
3	173.9	61.0	23	738	20.0
4	166.7	57.0	28	738	20.0
5	170.0	60.0	22	738	20.0
6	153.0	49.5	29	738	21.0
7	162.6	53.5	30	736	20.0
8	171.1	60.0	30	738	20.0
9	168.9	65.0	23	739	20.0
10	174.7	60.0	26	730	20.0
11	180.3	65.0	24	730	20.0
12	166.6	50.0	19	739	20.0
13	181.5	69.5	19	739	20.0
14	171.1	57.0	18	739	20.0
15	176.6	65.5	21	740	20.0
16	184.8	68.5	19	739	20.0

APPENDIX B

Absolute lung function values

Swimmers absolute lung function values

Subject	Sex	VC (ml)	$FEV_{1.0}$ (ml)	MVV (l/min)
1	M	8461.00	5741.00	179.00
2	F	3042.00	3328.00	129.50
3	F	4232.00	3703.00	150.00
4	F	5268.00	3978.00	136.00
5	M	7108.00	4913.00	169.00
6	F	5290.00	3956.00	148.80
7	M	5422.00	4430.00	152.90
8	M	8110.00	5223.00	172.20
9	M	6854.00	5532.00	218.00
10	M	6773.00	4417.00	159.00
11	F	4606.00	4044.00	152.00
12	M	7824.00	6193.00	183.00
13	F	3284.00	2865.00	112.00
14	M	6039.00	4783.00	137.80
15	M	6502.00	5113.00	199.70
16	M	7472.00	4573.00	154.00

Nonathletes absolute lung function values

Subject	Sex	VC (ml)	$FEV_{1.0}$ (ml)	MVV (l/min)
1	M	4926.00	4750.00	133.60
2	M	4648.00	3731.00	137.70
3	F	3025.00	2499.00	92.00
4	M	5808.00	4794.00	208.00
5	M	4981.00	4331.00	155.70
6	M	5168.00	4122.00	150.00
7	F	4717.00	3042.00	73.00
8	M	6094.00	4463.00	162.50
9	M	5808.00	4728.00	115.70
10	F	3683.00	3978.00	111.00
11	F	3813.00	3218.00	106.00
12	F	4254.00	3615.00	132.20
13	M	5036.00	3449.00	146.00
14	M	4849.00	3813.00	145.00
15	M	5102.00	3846.00	140.50
16	F	4672.00	3196.00	130.90

Runners absolute lung function values

Subject	Sex	VC (ml)	$FEV_{1.0}$ (ml)	MVV (l/min)
1	M	5312.00	4397.00	168.00
2	M	4298.00	3868.00	165.30
3	M	5157.00	3747.00	147.40
4	M	4673.00	4044.00	158.40
5	M	4794.00	3857.00	149.00
6	F	3485.00	3036.00	112.50
7	F	4276.00	3504.00	130.90
8	M	4210.00	3108.00	158.40
9	F	4265.00	3593.00	139.00
10	F	4474.00	3317.00	133.60
11	M	5686.00	4111.00	147.40
12	F	3703.00	3460.00	135.00
13	M	5565.00	4673.00	159.80
14	F	4617.00	3791.00	136.40
15	M	5190.00	4353.00	159.80
16	M	6993.00	5576.00	186.00

APPENDIX C

Slope and PCO_2 intercept (x-intercept) from the regression of ventilation on end-tidal PCO_2

Swimmers slope and x-intercept from the regression of ventilation on end-tidal PCO_2

Subject	Sex	VI/PCO_2	X-Intercept
1	M	1.74	50.10
2	F	0.88	46.40
3	F	0.47	42.60
4	F	0.26	24.50
5	M	2.75	50.40
6	F	1.39	50.20
7	M	1.15	53.10
8	M	0.85	45.90
9	M	0.94	38.70
10	M	1.62	47.50
11	F	0.83	44.70
12	M	0.62	42.50
13	F	2.48	48.50
14	M	1.22	51.80
15	M	1.67	46.20
16	M	0.81	45.10

Nonathletes slope and x-intercept from the regression of ventilation on end-tidal PCO_2

Subject	Sex	VI/PCO_2	X-Intercept
1	M	0.77	46.20
2	M	1.11	41.80
3	F	0.65	36.20
4	M	2.03	52.30
5	M	0.94	39.00
6	M	1.21	36.40
7	M	1.49	50.30
8	M	2.11	51.70
9	M	1.57	48.70
10	F	2.70	52.20
11	M	1.44	42.80
12	M	1.32	43.10
13	M	1.16	46.40
14	M	1.11	45.20
15	M	1.68	46.10
16	M	1.13	50.90

Runners slope and x-intercept from the regression of of ventilation on end-tidal PCO_2

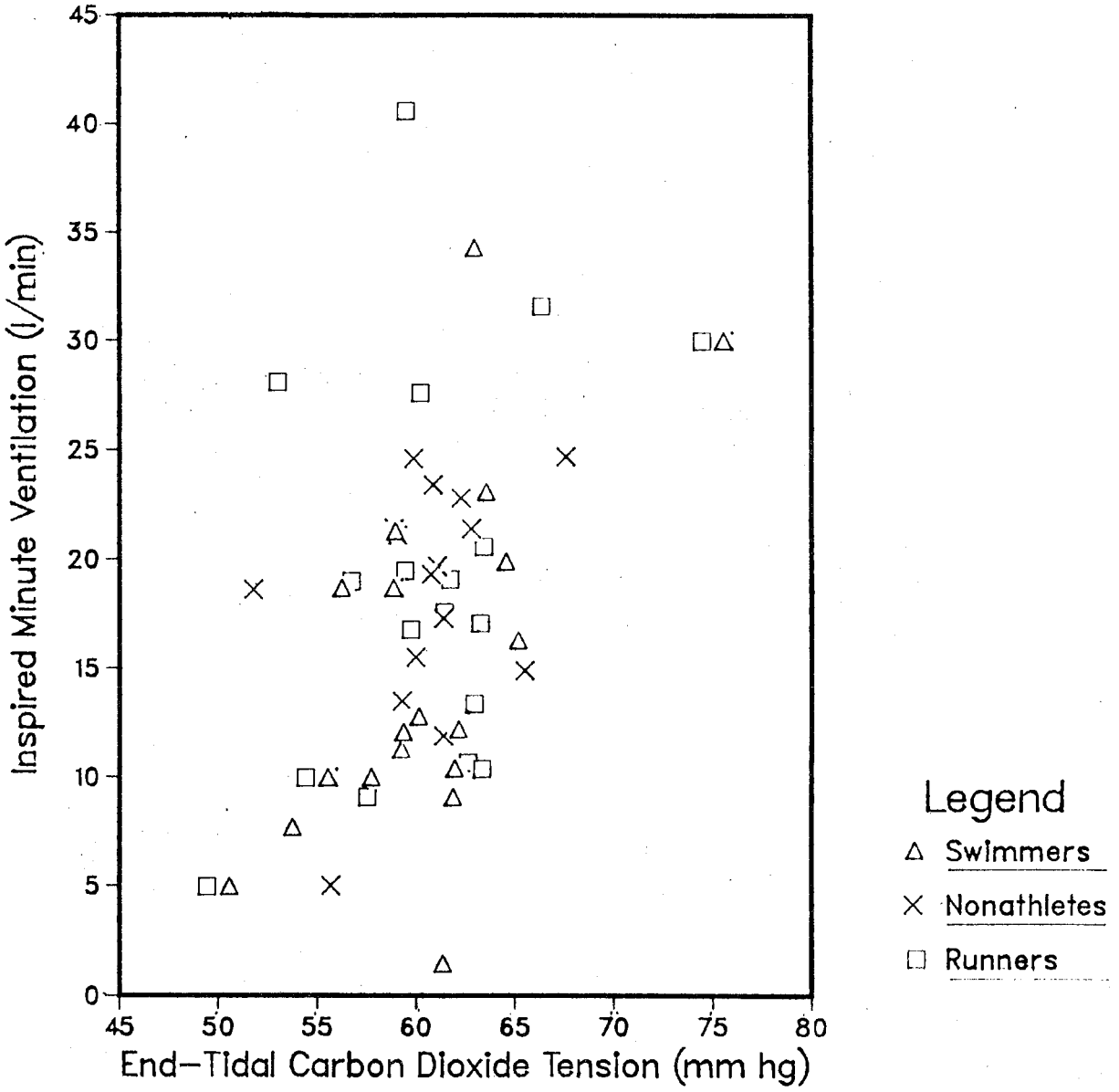
Subject	Sex	$VIPCO_2$	X-Intercept
1	M	0.53	43.40
2	M	0.80	46.10
3	M	2.38	42.30
4	M	.00	.00
5	M	1.34	47.10
6	F	1.92	38.30
7	F	.00	42.80
8	M	1.20	46.70
9	F	1.24	46.20
10	F	0.51	36.70
11	M	0.54	42.60
12	F	1.53	51.90
13	M	0.93	38.30
14	F	2.18	47.50
15	M	2.04	50.70
16	M	1.51	43.70

APPENDIX D

A plot of the slopes and intercepts ($PETCO_2$) from the regression of ventilation on end-tidal PCO_2 for all the subjects

APPENDIX D

A plot of the slopes and intercepts ($PETCO_2$) from the regression of ventilation on end-tidal PCO_2 for all the subjects


Inspired Minute Ventilation (l/min)
45
40
35
30
25
20
15
10
5
0
45
50
55
60
65
70
75
80
End-Tidal Carbon Dioxide Tension (mm hg)
Legend
Swimmers
Nonathletes
Runners

APPENDIX E

Correlation matrix of all the variables

Definition of variable names

N1 = neurogenic response at load 1

N2 = " " " " 2

N3 = " " " " 3

NR = " " " recovery

SS1 = steady-state response at load 1

SS2 = " " " " " 2

SS3 = " " " " " 3

SSR = " " " " recovery

PWC = physical work capacity heart rate of 170 b/min

PWCKG = PWC_{170} / kg

VIVT = slope of $\dot{V}I$ to tidal volume

VIVO2 = " " " " $\dot{V}O_2$

HRVO2 = " " heart rate to $\dot{V}O_2$

VIPCO2 = " " $\dot{V}I$ to end-tidal PCO_2

YINTERC = for CO_2 response ($\dot{V}I$)

XINTRCP = " " " ($PETCO_2$)

VC = vital capacity (normalized)

FEV = forced expired volume in one second (normalized)

MVV = maximum voluntary ventilation (normalized)

HT = height

WT = weight

PCO_2 = actual measured arterial PCO_2

PH = actual measured pH from blood analysis

STBICAR = standard bicarbonate from blood analysis

- - - - - - - - - - - - - - - PEARSON CORRELATION COEFFICIENTS - - - - - - - - - - - - - - -

	SEX	N1	N2	N3	NR	SS1	SS2	SS3	SSR	PWCKG	VIVT
SEX	1.0000	-.0751	.0511	.0772	-.1483	.2097	-.0757	-.0010	-.1425	-.3082	-.0092
	(48)	(44)	(44)	(44)	(44)	(44)	(44)	(44)	(44)	(47)	(44)
	P= .	P= .628	P= .742	P= .618	P= .337	P= .172	P= .625	P= .995	P= .356	P= .035	P= .953
N1	-.0751	1.0000	.1473	.0550	-.1248	.3521	-.2610	-.0312	-.1866	.1321	-.0750
	(44)	(44)	(44)	(44)	(44)	(44)	(44)	(44)	(44)	(44)	(44)
	P= .628	P= .	P= .340	P= .723	P= .419	P= .019	P= .087	P= .841	P= .225	P= .393	P= .629
N2	.0511	.1473	1.0000	-.1995	-.0887	-.4805	.2548	-.4499	-.1351	.1301	-.1382
	(44)	(44)	(44)	(44)	(44)	(44)	(44)	(44)	(44)	(44)	(44)
	P= .742	P= .340	P= .	P= .194	P= .567	P= .001	P= .095	P= .002	P= .382	P= .400	P= .371
N3	.0772	.0550	-.1995	1.0000	.0632	.4376	-.1573	.5558	.3379	-.2254	.2150
	(44)	(44)	(44)	(44)	(44)	(44)	(44)	(44)	(44)	(44)	(44)
	P= .618	P= .723	P= .194	P= .	P= .684	P= .003	P= .308	P= .000	P= .025	P= .141	P= .161
NR	-.1483	-.1248	-.0887	.0632	1.0000	.1060	-.0195	.6408	.6598	-.0701	.5737
	(44)	(44)	(44)	(44)	(44)	(44)	(44)	(44)	(44)	(44)	(44)
	P= .337	P= .419	P= .567	P= .684	P= .	P= .494	P= .900	P= .000	P= .000	P= .651	P= .000
SS1	.2097	.3521	-.4805	.4376	.1060	1.0000	-.3809	.6014	.2142	-.2451	.2052
	(44)	(44)	(44)	(44)	(44)	(44)	(44)	(44)	(44)	(44)	(44)
	P= .172	P= .019	P= .001	P= .003	P= .494	P= .	P= .011	P= .000	P= .163	P= .109	P= .181
SS2	-.0757	-.2610	.2548	-.1573	-.0195	-.3809	1.0000	-.1256	.3070	-.3391	-.2354
	(44)	(44)	(44)	(44)	(44)	(44)	(44)	(44)	(44)	(44)	(44)
	P= .625	P= .087	P= .095	P= .308	P= .900	P= .011	P= .	P= .417	P= .043	P= .024	P= .124
SS3	-.0010	-.0312	-.4499	.5558	.6408	.6014	-.1256	1.0000	.7838	-.3951	.4321
	(44)	(44)	(44)	(44)	(44)	(44)	(44)	(44)	(44)	(44)	(44)
	P= .995	P= .841	P= .002	P= .000	P= .000	P= .000	P= .417	P= .	P= .000	P= .008	P= .003
SSR	-.1425	-.1866	-.1351	.3379	.6598	.2142	.3070	.7838	1.0000	-.3223	.3450
	(44)	(44)	(44)	(44)	(44)	(44)	(44)	(44)	(44)	(44)	(44)
	P= .356	P= .225	P= .382	P= .025	P= .000	P= .163	P= .043	P= .000	P= .	P= .033	P= .022
PWCKG	-.3082	.1321	.1301	-.2254	-.0701	-.2451	-.3391	-.3951	-.3223	1.0000	.0028
	(47)	(44)	(44)	(44)	(44)	(44)	(44)	(44)	(44)	(47)	(44)
	P= .035	P= .393	P= .400	P= .141	P= .651	P= .109	P= .024	P= .008	P= .033	P= .	P= .986
VIVT	-.0092	-.0750	-.1382	.2150	.5737	.2052	-.2354	.4321	.3450	.0028	1.0000
	(44)	(44)	(44)	(44)	(44)	(44)	(44)	(44)	(44)	(44)	(44)
	P= .953	P= .629	P= .371	P= .161	P= .000	P= .181	P= .124	P= .003	P= .022	P= .986	P= .

- - - - - - - - - - - - - - PEARSON CORRELATION COEFFICIENTS - - - - - - - - - - - - - -

	SEX	N1	N2	N3	NR	SS1	SS2	SS3	SSR	PWCKG	VIVT
VIVO2	-.0071	-.0866	-.1103	.2306	.5304	.3196	.4257	.6330	.8360	-.3824	.2285
	(44)	(44)	(44)	(44)	(44)	(44)	(44)	(44)	(44)	(44)	(44)
	P= .964	P= .576	P= .476	P= .132	P= .000	P= .034	P= .004	P= .000	P= .000	P= .010	P= .136
HRVO2	.2900	-.1110	-.3328	.5565	-.0129	.4685	.1588	.5596	.3861	-.8114	.0742
	(44)	(44)	(44)	(44)	(44)	(44)	(44)	(44)	(44)	(44)	(44)
	P= .056	P= .473	P= .027	P= .000	P= .934	P= .001	P= .303	P= .000	P= .010	P= .000	P= .632
VIPCO2	-.0151	.0632	-.3166	.2349	.0303	.3349	-.2127	.3090	.1906	-.0792	.1816
	(47)	(43)	(43)	(43)	(43)	(43)	(43)	(43)	(43)	(46)	(43)
	P= .920	P= .687	P= .039	P= .129	P= .847	P= .028	P= .171	P= .044	P= .221	P= .601	P= .244
YINTERC	-.0149	-.0483	.3638	-.2147	.0044	-.3825	.2519	-.2922	-.1258	.1356	-.2360
	(46)	(42)	(42)	(42)	(42)	(42)	(42)	(42)	(42)	(45)	(42)
	P= .922	P= .761	P= .018	P= .172	P= .978	P= .012	P= .108	P= .060	P= .427	P= .375	P= .132
VC	.0462	.0159	.1787	-.0446	-.4144	-.1974	.0030	-.2973	-.2849	-.1223	-.4211
	(48)	(44)	(44)	(44)	(44)	(44)	(44)	(44)	(44)	(47)	(44)
	P= .755	P= .919	P= .246	P= .774	P= .005	P= .199	P= .985	P= .050	P= .061	P= .413	P= .004
FEV	-.0038	.0628	.3130	-.0789	-.4253	-.4319	.0923	-.4463	-.2965	.0323	-.3646
	(48)	(44)	(44)	(44)	(44)	(44)	(44)	(44)	(44)	(47)	(44)
	P= .980	P= .685	P= .038	P= .611	P= .004	P= .003	P= .551	P= .002	P= .051	P= .829	P= .015
MVV	.0730	.2430	.4289	-.1299	-.3064	-.2982	.0417	-.3859	-.1627	.2892	-.3102
	(46)	(42)	(42)	(42)	(42)	(42)	(42)	(42)	(42)	(45)	(42)
	P= .630	P= .121	P= .005	P= .412	P= .048	P= .055	P= .793	P= .012	P= .303	P= .054	P= .045
PCO2	-.2488	.0153	.0189	-.0944	-.2155	-.2948	.0522	-.1957	-.1686	.1897	-.0528
	(48)	(44)	(44)	(44)	(44)	(44)	(44)	(44)	(44)	(47)	(44)
	P= .088	P= .922	P= .903	P= .542	P= .160	P= .052	P= .737	P= .203	P= .274	P= .202	P= .734
PH	.2222	.0678	.1186	-.0206	.0783	.0649	-.0505	.0772	-.0282	.0650	-.0171
	(48)	(44)	(44)	(44)	(44)	(44)	(44)	(44)	(44)	(47)	(44)
	P= .129	P= .662	P= .443	P= .895	P= .613	P= .675	P= .745	P= .618	P= .856	P= .664	P= .912
STBICAR	-.0752	.0453	.1007	-.0796	-.1288	-.2213	.0016	-.1202	-.1717	.2685	-.0656
	(48)	(44)	(44)	(44)	(44)	(44)	(44)	(44)	(44)	(47)	(44)
	P= .611	P= .770	P= .516	P= .608	P= .405	P= .149	P= .992	P= .437	P= .265	P= .068	P= .672
PWC	-.5257	.1388	.1190	-.2641	-.1375	-.2818	-.3384	-.4195	-.3577	.8899	-.1221
	(47)	(44)	(44)	(44)	(44)	(44)	(44)	(44)	(44)	(47)	(44)
	P= .000	P= .369	P= .441	P= .083	P= .373	P= .064	P= .025	P= .005	P= .017	P= .000	P= .430

- - - - - - - - - - - - - - - PEARSON CORRELATION COEFFICIENTS - - - - - - - - - - - - - - -

	SEX	N1	N2	N3	NR	SS1	SS2	SS3	SSR	PWCKG	VIVT
XINTRCEP	-.1347	.2823	-.1958	.0920	-.2392	.2165	.0619	.0734	.0020	-.1480	-.0961
	(47)	(43)	(43)	(43)	(43)	(43)	(43)	(43)	(43)	(46)	(43)
	P= .367	P= .067	P= .208	P= .558	P= .122	P= .163	P= .693	P= .640	P= .990	P= .326	P= .540
HT	-.6164	.1503	.0337	-.0324	-.0055	-.0822	.0888	.0467	.1593	.0901	-.2865
	(48)	(44)	(44)	(44)	(44)	(44)	(44)	(44)	(44)	(47)	(44)
	P= .000	P= .330	P= .828	P= .834	P= .972	P= .596	P= .567	P= .763	P= .302	P= .547	P= .059
WT	-.5005	.0478	.0235	-.0678	-.1305	-.1094	-.0344	-.0587	-.0924	-.1707	-.2375
	(48)	(44)	(44)	(44)	(44)	(44)	(44)	(44)	(44)	(47)	(44)
	P= .000	P= .758	P= .880	P= .662	P= .398	P= .480	P= .824	P= .705	P= .551	P= .251	P= .120

- - - - - - - - - - - - - - PEARSON CORRELATION COEFFICIENTS - - - - - - - - - - - - - -

	VIVO2	HRVO2	VIPCO2	YINTERC	VC	FEV	MVV	PCO2	PH	STBICAR	PWC
VIVO2	1.0000	.3672	.1764	-.1568	-.3288	-.3334	-.0992	-.2752	.0955	-.1449	-.4867
	(44)	(44)	(43)	(42)	(44)	(44)	(42)	(44)	(44)	(44)	(44)
	P= .	P= .014	P= .258	P= .321	P= .029	P= .027	P= .532	P= .070	P= .537	P= .348	P= .001
HRVO2	.3672	1.0000	.2883	-.3327	-.0022	-.1771	-.3511	-.2119	-.0969	-.2841	-.7620
	(44)	(44)	(43)	(42)	(44)	(44)	(42)	(44)	(44)	(44)	(44)
	P= .014	P= .	P= .061	P= .031	P= .988	P= .250	P= .023	P= .167	P= .531	P= .062	P= .000
VIPCO2	.1764	.2883	1.0000	-.9833	-.1901	-.3512	-.1858	-.2191	.1808	-.0719	-.0821
	(43)	(43)	(47)	(46)	(47)	(47)	(45)	(47)	(47)	(47)	(46)
	P= .258	P= .061	P= .	P= .000	P= .200	P= .015	P= .222	P= .139	P= .224	P= .631	P= .587
YINTERC	-.1568	-.3327	-.9833	1.0000	.1918	.3840	.2639	.2158	-.1576	.0838	.1289
	(42)	(42)	(46)	(46)	(46)	(46)	(44)	(46)	(46)	(46)	(45)
	P= .321	P= .031	P= .000	P= .	P= .201	P= .008	P= .083	P= .150	P= .295	P= .580	P= .399
VC	-.3288	-.0022	-.1901	.1918	1.0000	.5873	.1351	-.0352	-.0107	-.1627	.1368
	(44)	(44)	(47)	(46)	(48)	(48)	(46)	(48)	(48)	(48)	(47)
	P= .029	P= .988	P= .200	P= .201	P= .	P= .000	P= .371	P= .812	P= .943	P= .269	P= .359
FEV	-.3334	-.1771	-.3512	.3840	.5873	1.0000	.5283	.2493	-.1968	-.0006	.1482
	(44)	(44)	(47)	(46)	(48)	(48)	(46)	(48)	(48)	(48)	(47)
	P= .027	P= .250	P= .015	P= .008	P= .000	P= .	P= .000	P= .087	P= .180	P= .997	P= .320
MVV	-.0992	-.3511	-.1858	.2639	.1351	.5283	1.0000	.0474	.0957	.1381	.1678
	(42)	(42)	(45)	(44)	(46)	(46)	(46)	(46)	(46)	(46)	(45)
	P= .532	P= .023	P= .222	P= .083	P= .371	P= .000	P= .	P= .754	P= .527	P= .360	P= .270
PCO2	-.2752	-.2119	-.2191	.2158	-.0352	.2493	.0474	1.0000	-.4770	.6335	.2199
	(44)	(44)	(47)	(46)	(48)	(48)	(46)	(48)	(48)	(48)	(47)
	P= .070	P= .167	P= .139	P= .150	P= .812	P= .087	P= .754	P= .	P= .001	P= .000	P= .137
PH	.0955	-.0969	.1808	-.1576	-.0107	-.1968	.0957	-.4770	1.0000	.3351	-.0561
	(44)	(44)	(47)	(46)	(48)	(48)	(46)	(48)	(48)	(48)	(47)
	P= .537	P= .531	P= .224	P= .295	P= .943	P= .180	P= .527	P= .001	P= .	P= .020	P= .708
STBICAR	-.1449	-.2841	-.0719	.0838	-.1627	-.0006	.1381	.6335	.3351	1.0000	.1560
	(44)	(44)	(47)	(46)	(48)	(48)	(46)	(48)	(48)	(48)	(47)
	P= .348	P= .062	P= .631	P= .580	P= .269	P= .997	P= .360	P= .000	P= .020	P= .	P= .295
PWC	-.4867	-.7620	-.0821	.1289	.1368	.1482	.1678	.2199	-.0561	.1560	1.0000
	(44)	(44)	(46)	(45)	(47)	(47)	(45)	(47)	(47)	(47)	(47)
	P= .001	P= .000	P= .587	P= .399	P= .359	P= .320	P= .270	P= .137	P= .708	P= .295	P= .

- - - - - - - - - - - - - - - PEARSON CORRELATION COEFFICIENTS - - - - - - - - - - - - - - -

	VIVO2	HRVO2	VIPCO2	YINTERC	VC	FEV	MVV	PCO2	PH	STBICAR	PWC
XINTRCEP	.0893	.2939	.5436	-.6379	-.1024	-.1876	-.1277	-.0871	.0701	-.0256	-.1104
	(43)	(43)	(47)	(46)	(47)	(47)	(45)	(47)	(47)	(47)	(46)
	P= .569	P= .056	P= .000	P= .000	P= .494	P= .206	P= .403	P= .560	P= .640	P= .864	P= .465
HT	-.0306	-.0892	.1021	-.0718	.2894	.1399	-.0953	.0905	-.1850	-.1397	.4270
	(44)	(44)	(47)	(46)	(48)	(48)	(46)	(48)	(48)	(48)	(47)
	P= .844	P= .565	P= .495	P= .635	P= .046	P= .343	P= .529	P= .541	P= .208	P= .344	P= .003
WT	-.2767	.0800	.0270	-.0370	.4844	.2167	-.2205	.0625	-.2158	-.1991	.2772
	(44)	(44)	(47)	(46)	(48)	(48)	(46)	(48)	(48)	(48)	(47)
	P= .069	P= .606	P= .857	P= .807	P= .000	P= .139	P= .141	P= .673	P= .141	P= .175	P= .059