

TR 89-13v

APNEIC BRADYCARDIA: TERRESTRIAL AND
AQUATIC RESPONSES IN MAN UNDER
WORKING CONDITIONS

BY

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MASTER'S THESIS

Submitted in fulfilment of the requirements
for the degree Master of Science

Department of Human Movement Studies
Rhodes University, 1988

Grahamstown, South Africa

ABSTRACT

The focus of this research was the reappraisal in physiological and psychophysical terms of current equivocal theories regarding the onset, course and termination of apneic bradycardia.

Sixteen healthy male subjects participated in four separate testing sessions. Maximal oxygen consumption ($\dot{V}O_2$ max) was measured on land and underwater using an identical direct, continuous progressive cycle ergometer test. On each of two other occasions subjects exercised in either environment at 50, 70 and 90% of the appropriate $\dot{V}O_2$ max, during which time heart rate was continuously recorded. An initial apneic bout at each exercise intensity was followed by performance of the same workload without apnea for an equivalent period of time. Ratings of perceived exertion (RPE) were also monitored.

While apneic bradycardia occurred at each exercise intensity studied underwater, it was apparent only at 50% $\dot{V}O_2$ max on land. With the exception of between 50 and 90% $\dot{V}O_2$ max on land, the mean apneic heart rates did not differ with varying exercise intensity ($p < 0.05$); nor did the lowest heart rate recorded, although this was lower underwater than on land. Apart from 70% $\dot{V}O_2$ max on land, apneic heart rates were lower than the equivalent values measured during exercise without apnea. Land and underwater heart rates during apneic and non-apneic conditions did not differ until 90% $\dot{V}O_2$ max. The effects of increasing exercise intensity upon the onset of bradycardia were evident in that it occurred earlier at 50% $\dot{V}O_2$ max underwater than at the heavier workloads, and only at 50% $\dot{V}O_2$ max on land.

The mean breath-hold duration did not differ between the land and underwater environments, nor was it affected by increasing exercise intensity. The order in which breath-holds was performed did not alter the length of apnea.

Land and underwater RPE did not differ and increased with increasing exercise intensity in both environments. During apneic exercise RPE was greater than the equivalent exercise without apnea.

Twelve of the original 16 subjects were divided equally into two groups on the basis of vital capacity expressed relative to body surface area. Vital capacity was measured during the first laboratory session. Neither the mean heart rate response to apneic exercise at 50% $\dot{V}O_2$ max in both environments, nor the lowest heart rates recorded differed between groups, prompting the conclusion that lung volume did not affect apneic bradycardia. Despite a longer breath-hold duration for Group A (large relative lung volume) than Group B (small relative lung volume), the onset point of bradycardia was the same for either group when expressed relative to total breath-hold duration.

ACKNOWLEDGEMENTS

In completing this study I owe my sincere gratitude to several people:

Heartfelt thanks go to my supervisor, Pat Scott, not only for her consistent guidance and encouragement over the duration of this study, but for her friendship and constant belief in my ability. It meant much to me, Pat, and I shall always appreciate it.

My warmest appreciation is extended to Professor Jack Charteris, for the support and guidance shown me both as a student and as a lecturer in his department. Thank you, Prof, for your assistance with this thesis.

I must express my gratitude to my wonderful husband, Pete, whose steadfast support in all I do was invaluable during the completion of this study.

My sincere thanks and appreciation must go to each of my subjects. A more enthusiastic and reliable group I could not have hoped for. In addition, I wish to thank those who assisted so willingly with the data collection.

I am indebted to Les Moir and Caroline Smith for their help with the laborious task of typing this script.

Finally, I wish to acknowledge the Human Sciences Research Council, whose provision of a grant made this study possible.

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CHAPTER 1

INTRODUCTION

Bradycardia, defined simply as a slowed heart rate (Guyton, 1986), is evident in man under varying conditions. Any circulatory reflex that stimulates the vagus nerve may cause a reduction in heart rate (Guyton, 1986). A slowing of the heart beat following training in athletes, for example, has been attributed to increased parasympathetic neural contribution (increased vagal tone) and decreased sympathetic activity (McArdle et al., 1986). Individuals with the carotid sinus syndrome demonstrate extreme bradycardia in response to mild pressure on the neck. In such cases an arteriosclerotic process in the carotid sinus region of the carotid artery causes excessive sensitivity of the pressure receptors located in the arterial wall. Pressure on the neck elicits a strong pressoreceptor reflex, causing intense vagal stimulation of the heart (Schriber, 1966; Guyton, 1986). Similar results are provoked by eyeball pressure (Julian, 1973). In such instances strong emotional excitation of the parasympathetic nerves to the heart and the vasodilator nerves to the skeletal muscle result in a slowing of the heart and a reduction in arterial pressure.

A similar reduction in heart rate has been observed in response to apnea, or breath-hold, immersion in water, and apnea during immersion (Magel et al., 1982; Hayward et al., 1984). While the underlying mechanisms behind such a response have been the topic of some controversy, it is generally accepted that several factors combine to bring about the development of bradycardia under these circumstances

(Song et al., 1969; Magel et al., 1982). Central to a multiple factor hypothesis is that the cessation of respiratory movement per se initiates bradycardia by means of interactions between the respiratory centre and the cardiac autonomic centres in the central nervous system. This is then modified by a variety of neural, mechanical and chemical factors (Song et al., 1969; Speck and Bruce, 1978; Lin, 1982). It has been suggested that the trigeminal nerve serves as the afferent pathway for some of these sensing systems (Andersen, 1963).

According to Gross et al. (1976), a major role in the initiation and potentiation of bradycardia in man during breath-holding is played by the carotid chemoreceptors. Gross and co-workers reported that patients who had undergone bilateral carotid body resection for the treatment of bronchial asthma experienced a progressive tachycardia during apnea, in contrast to a control group who demonstrated the typical bradycardia of normal subjects. These authors concluded that in normal apneic man, an antagonistic interaction exists between the carotid bodies, which mediate vagal bradycardia when stimulated, and other chemoreceptors such as the aortic bodies or central nervous system chemoreceptors which, when stimulated, can induce tachycardia. Thus the resulting breath-holding heart rate in normal man represents the domination of the carotid bodies over other tachycardia-producing reflexes (Gross et al., 1976).

Several other authors, however, have concluded that bradycardia, and other physiological adaptations such as peripheral vasoconstriction which are collectively known as the "diving reflex", occur partly due to apnea and partly due to nasal and facial receptor reflexes responding to

cold water (Brick, 1966; Hong et al., 1967; Strømme et al., 1970; Bergman et al., 1972). According to Paulev (1968), bradycardia in response to apnea, face immersion and apnea during face immersion can be related to two independent components. The first of these, termed an "apneic bradycardia", relates changes in heart rate to inspired lung volume during apnea, while the second, a "cold bradycardia", is seen as varying in magnitude with the water temperature.

The existence of an inverse relationship between heart rate and inspired lung volume was observed by Angelone and Coulter (1965). These authors suggested that stretch receptors located in the lungs or thoracic wall initiated a reflex responsible for this phenomenon. In addition, a proportional relationship between inspired lung volume and the extent of bradycardia has been attributed to the influence of changes in intrathoracic pressure upon the cardiovascular system (Song et al., 1969; Magel et al., 1982). Such theories emphasise the importance of the role of venous return which is greatly influenced by intrathoracic pressure. Since intrathoracic pressure becomes more negative in proportion to the depth of inspiration, venous return is facilitated, thus enhancing stimulation of the sino-aortic baroreceptors, resulting in a slowing of the heart rate. Magel et al. (1982) demonstrated that breath-holding with a maximal inspiration (about 90% vital capacity) elicited a greater degree of bradycardia than apnea following a normal inspiration. In contrast, Kawakami et al. (1967) found that there was no difference in the heart rate response during apnea when subjects inspired maximally or held their breath at the end of an expiration. The view that the influence of intrathoracic pressure upon heart rate is of primary importance has been strengthened by reports that apneic

bradycardia can be converted to apneic tachycardia by elevating the intrapulmonary pressure during Valsalva (Song et al., 1969; Magel et al., 1982). Craig (1963) observed that different Valsalva pressures elicited tachycardia which was proportional to the increased intrathoracic pressure.

It would appear, however, that a systematic study involving a comparison of the heart rate responses of subjects with different absolute and relative lung volumes would yield useful information with regard to the effects of lung volume upon apneic bradycardia. Since the amount of air inspired into the lung appears to affect the heart rate by stimulating or depressing certain pressure receptors, it is possible that individuals with large lungs (absolutely and relative to body surface area) may demonstrate a different response to those with small lungs. Candler (1986) reported that female subjects with large lung volumes (expressed relative to body surface area) had significantly lower heart rates than those with small relative lung volumes. This was true during apnea at rest on land and underwater, and when swimming and working underwater. A limitation of this study, however, was that the intensity of the exercise was not controlled. One of the aims of the present research was to further elucidate the effects of relative lung volumes upon bradycardia by means of stricter control measures than have been evident in past research.

Several factors are considered to influence the bradycardial response during apnea. In summarising the bradycardia-modifying effects of various factors, Lin (1982) stated that, in general, cold water, young age, large lung volume, low PO_2 , low intrathoracic pressure, and high

metabolic rate (also high immediate pre-breath-hold heart rate) potentiate apnea-induced bradycardia. Research investigating such influences has, however, yielded equivocal results. A number of authors, for example, have found bradycardia to be temperature-related, with greater degrees of bradycardia occurring in cold than in warm water (Hong et al., 1967; Kawakami et al., 1967; Paulev, 1968; Moore et al., 1972; Magel et al., 1982). However, Hayward et al. (1984), after measuring diving bradycardia in 160 humans submerged in water temperatures from 0 to 35°C at 5°C intervals, concluded that temperature modulation of the dive response was very weak or nonexistent. Hayward and co-workers observed no dependency of bradycardial heart rate on water temperature over the whole 35°C range. It was suggested that most of the earlier studies had involved facial immersion only, incomplete ranges of water temperatures and often a low number of subjects.

Previous studies in this area have tended to demonstrate a lack of convention with respect to the evaluation of bradycardia in response to apnea, immersion and apnea during immersion. A major criticism of much of the research to date has been that many of the cardiopulmonary changes during breath-hold dives are generalised from laboratory-conducted studies, often incorporating facial immersion only (Lin, 1984). As Craig (1963) pointed out, diving for any animal is a compound stress; for humans during apneic diving in a natural setting elevated sympathoadrenal activity may lead to exaggerated cardio-vascular responses (Lin, 1984). Authors who have discovered the existence of bradycardia during apnea have found conflicting results when looking at the point at which bradycardia commences, its course and termination. While some authors have reported an immediate and rapid

drop in heart rate (Bove et al., 1968; Bergmann et al., 1972; Lin, 1982), others have found that bradycardia only developed 5 to 10s after the face or body was submerged (Stromme et al., 1970), and others still have observed an increase in heart rate immediately after subjects dived, followed by the development of bradycardia (Scholander et al., 1962). According to Lin (1982), studies which have reported that there is a slow development of bradycardia in man refer not to the onset, but to the slow attainment of the maximal bradycardial response. Thus, much of the controversy surrounding this area may be due not only to the large variety of research protocols employed in the past, but in part to a lack of uniformity in the interpretation of the results obtained. Magel et al. (1982) pointed out that very often the influence of high heart rates measured immediately prior to breath-holding has been ignored, giving rise to misleading measures of substantial bradycardia in response to apnea or apneic submergence.

The large variety of experimental designs used, the results obtained and the explanations given for these results, tend to indicate that much has still to be clarified. An attempt to clarify some of the existing discrepancies in the literature necessitated the rigorous control of those factors considered as being influential to the bradycardial response in man under various terrestrial and aquatic conditions. Central to the present study, therefore, was an attempt to standardise such factors as the depth of immersion, posture, the water temperature, the sex, fitness and underwater experience of the subjects, the intensity at which work was performed, both on land and underwater, and the ventilatory manoeuvres used prior to breath-holding. Only in this

way could meaningful statements concerning the phenomenon of apneic bradycardia be made.

Finally, it would appear that in the past researchers have largely omitted any investigation of subjective reactions to apnea and apneic diving. Many neuromuscular and physiological parameters have been suggested as contributing to effort perception during exercise. According to Lin et al. (1974), psychological factors constitute the major determinants of the conventional breath-hold breaking point and may contribute to the wide range of breath-hold times reported in the literature. Since apnea has been cited as a primary causative factor in the development of bradycardia, the present study sought to achieve an integration between physiological and psychological concomitants to apneic bradycardia.

STATEMENT OF THE PROBLEM

The focus of the present study was an attempt to reappraise current equivocal theories regarding the onset, course and termination of apneic bradycardia in physiological and psychophysical terms. The study aimed at establishing representative heart rate responses to apnea during work, both on land and underwater.

As individual differences in breath-hold duration may occur (Hayward et al., 1984) heart rates were examined at various percentages of total breath-hold time. This made possible the investigation of the onset, course and termination of the response in terms of both absolute time and as a percentage of total breath-hold time. Breath-hold duration itself was examined in order to establish whether changes in workload in either environment would affect the length of apnea.

Since it has been suggested in the past that a relationship exists between physiological parameters such as heart rate and ratings of perceived exertion (RPE; Borg, 1970), the research set out to investigate the effect that apneic responses had upon the subject's perception of effort as measured by Borg's RPE scale.

In addition, an attempt was made to determine the effects of relative lung volumes, or more specifically, relative vital capacities, upon the parameters measured. In other words, under apneic conditions would subjects with large relative vital capacities exhibit different responses (in heart rate, breath-hold duration and RPE) to subjects with smaller relative capacities?

In this study the terms "apnea" and "breath-holding" were used synonymously. The term "bradycardia" has been defined variously as simply a slowed heart rate (Julian, 1983; Guyton, 1986; McArdle *et al.*, 1986), a relatively slow heart action (Webster's, 1986) or a heart rate less than 60 b.min^{-1} (Neufeld *et al.*, 1978; Arnold, 1985). For the purpose of this research, in which the effect of heart rates immediately prior to apnea were of importance, bradycardia was defined as a reduction in heart rate below the immediate pre-apneic level. Thus maximum bradycardia was determined as the minimum rate recorded during a given apneic condition that was below the pre-apneic level.

RESEARCH HYPOTHESES

The hypotheses generated in the present study were such as to enable a broad examination of terrestrial and aquatic physiological and psychological responses to apneic work of varying intensities.

Hypothesis 1

There is no change in either the terrestrial or aquatic heart rate response under apneic and normal respiratory conditions, during differing work intensities expressed relative to $\dot{V}O_2$ max.

This hypothesis incorporated an examination of the heart rate both in absolute terms and relative to 10% increments of breath-hold duration with regard to the onset, course and termination of the bradycardial response.

Stated statistically, the null-hypothesis was:

$$H_0 : \mu H_{ap} = \mu H_{wap}$$

Where μH is the mean heart rate response for subjects working at different intensities, either with apnea (ap) or without apnea (wap), under terrestrial and aquatic conditions.

The alternative hypothesis was:

$$H_a : \mu H_{ap} \neq \mu H_{wap}$$

Hypothesis 2

There is no difference in breath-hold duration for subjects working at differing intensities under terrestrial and aquatic conditions.

Stated statistically, the null hypothesis was:

$$H_0 : \mu D_{50} = \mu D_{70} = \mu D_{90}$$

Where μD is the mean breath-hold duration for subjects, and 50, 70 and 90 represent work intensities (%) on land and underwater, expressed relative to $\dot{V}O_2$ max.

The alternative hypothesis was:

$$H_a : \mu D_{50} \neq \mu D_{70} \neq \mu D_{90}$$

Hypothesis 3

There is no difference in the terrestrial or aquatic rating of perceived exertion (RPE) under apneic and normal respiratory conditions during work at differing intensities.

Stated statistically, the null hypothesis was:

$$H_o : \mu R_{50} = \mu R_{70} = \mu R_{90}$$

Where μR represents the mean ratings of perceived exertion obtained during terrestrial and aquatic work intensities (%) expressed relative to $\dot{V}O_2$ max.

The alternative hypothesis was:

$$H_a : \mu R_{50} \neq \mu R_{70} \neq \mu R_{90}$$

Hypothesis 4

To explore the effects of relative lung size it was hypothesised that the terrestrial and aquatic apneic heart rate responses of subjects with large relative lung volumes do not differ from those of subjects with small relative lung volumes at a given work intensity.

Stated statistically, the null hypothesis was:

$$H_0 : \mu_{(large)} = \mu_{(small)}$$

Where μ was the population mean heart rate under terrestrial and aquatic conditions at a given work intensity.

The alternative hypothesis was:

$$H_a : \mu_{(large)} \neq \mu_{(small)}$$

DELIMITATIONS

Sixteen healthy male subjects, mean age 20.9 years, participated in the study. Since it has been suggested that the apneic heart rate response while submerged in water may be modified by one's experience in these conditions (Hong et al., 1970; Oldridge et al., 1978), the study sample was restricted to subjects with a minimum of six month's experience of underwater apneic activity, such as underwater hockey or spearfishing.

All subjects underwent a direct measurement of maximal oxygen consumption using a cycle ergometer both on land and underwater, and took part in two apneic test conditions. Heart rate, breath-hold duration and ratings of perceived exertion were measured during maximal breath-holds while working on a cycle ergometer at 50, 70 and 90 percent of $\dot{V}O_2$ max in the two environments.

On the basis of lung volume analyses the subjects were then divided into two groups: those with large vital capacities relative to body surface area (Group A) and those with small relative vital capacities (Group B). In order to ensure that these groups did have significantly different lung volumes, with no overlap in the standard deviations, the number of subjects included in the subsequent analyses was reduced to twelve. In

this way, the specific effect of relative lung volume upon apneic responses was examined.

LIMITATIONS

While every effort was made to ensure rigorous control of the research in general, and in particular those factors considered as being influential to the heart rate response to apnea, the following limitations must be noted:

1. Heart rate is extremely sensitive to a variety of factors, not all of which could be controlled prior to testing. Subjects were requested to refrain from energetic physical activity, eating or smoking for at least one hour prior to each testing session, but apart from voluntary compliance with this request, external influences such as these were not controlled.
2. The exact effects of water upon the resistance load on the ergometer and the additional resistance offered to pedalling whilst underwater were not calculated, thus power output underwater could not be precisely determined. Work intensity during apneic conditions was, however, calculated relative to the "situation limited" functional $\dot{V}O_2$ max attained in each environment.
3. During the apneic testing session, subjects performed an apneic followed by a non-apneic exercise bout at three different intensities (50, 70 and 90% $\dot{V}O_2$ max). Although there was no way to evaluate the cumulative effects of the six bouts, the influence of one upon the other was minimised by random presentation. A variable recovery period ensured the return of heart rate to the initial pre-exercise

level and allowed for the removal of lactic acid following each short exercise bout.

4. Testing times were chosen as those convenient to the individual. Although this was between 9.30am and 3.30pm, with subjects in general maintaining the same time of day for each session, the effects of diurnal variation were not deliberately controlled for.

CHAPTER 2

REVIEW OF THE LITERATURE

INTRODUCTION

The bradycardia which accompanies apnea and apneic immersion in water is said to be a component of what has been described as an evolutionarily old reflex (Asmussen and Kristiansson, 1968): the mammalian dive reflex. Together with a reduction in heart rate, this oxygen-conserving process incorporates peripheral vasoconstriction, redistribution of blood flow, depressed metabolism and a decreased respiratory exchange ratio (Kawakami et al., 1967; Lin, 1982; Schilling et al., 1984). While such responses are more profound in aquatic mammals and avian species, the dive reflex is said to exist in all vertebrates examined so far, differing only in intensity and tempo (Angell-James and de Burgh Daly, 1972; Lin, 1982). Whether the bradycardial response to apnea in man constitutes part of a general oxygen conserving mechanism is, however, a matter of debate.

Wolf (1978) described the dive reflex as a "phylogenetically ancient and virtually universal oxygen conserving biological adaptation", an adaptation which is often seen as applying to man, despite considerable attenuation of the physiological adjustments to apnea compared to other species (Scholander et al., 1962; Irving, 1963; Kawakami et al., 1967; Wolf, 1978; Arnold, 1985). Support for the contention that this is a phylogenetically very old physiological mechanism is seen to lie in the fact that a similar cardiac response occurs when fish are made hypoxic in their natural environment, are taken out of water, or make routine

excursions out of water, as in the case of flying fish (Scholander et al., 1962; Angell-James and de Burgh Daly, 1972). Further support for this view is drawn from the process of human birth. Evidence exists to suggest that in the case of an asphyxiated fetus, fetal bradycardia may be followed by an acute rise in lactic acid immediately after birth, a process which appears similar to the acute rise in blood lactate as soon as recovery breathing starts in diving mammals such as the seal (Scholander et al., 1962). This prompted Scholander and associates to suggest that fetal bradycardia in man represents a general defence mechanism against asphyxia which is fully developed at the time of birth.

The similarities between man and many species of aquatic mammals in terms of physiological and morphological features have indeed been propounded within the framework of an evolutionary theory: that of the Aquatic Man (Hardy, 1960). This hypothesis attempted to provide an explanation for that blank period in human evolution now falling between Ramapithecus, dated around 9 million years ago, and Australopithecus afarensis, from about 4 million years ago. The aquatic theory envisages that during this period one hominid group embarked on a distinct path of evolution by adaptating to an aquatic environment (Morgan, 1982). When ecological situations favoured this at a later date, they returned to their earlier terrestrial lifestyle, but retained certain aquatic adaptations, of which the dive reflex was one (Morgan, 1982; Morris, 1982).

While the concepts upon which the Aquatic Theory is structured are dubious, the postulate that apneic bradycardia in man and the diving

bradycardia in animals are produced by a similar mechanism relating to a general oxygen conserving reflex, has been supported. Doubt has been expressed, however, as to whether man does possess the highly controlled cardio-vascular response evident in diving mammals (Paulev and Hansen, 1972; Magel et al., 1982). This response in diving mammals and birds involves strong and selective peripheral vasoconstriction, such that mean arterial pressure is relatively well maintained, despite a drastic drop in cardiac output. Since stroke volume is either unchanged, or drops only slightly, the reduction in cardiac output is approximately proportional to the accompanying fall in heart rate (Angell-James and de Burgh Daly, 1972; Lin, 1982). Vasoconstriction is so intense that blood flow nearly ceases in skin, skeletal muscle and the kidney. In this way limited supplies of oxygen are conserved and preferentially channelled to the heart and brain, both sensitive to hypoxia (Angell-James and de Burgh Daly, 1972; Lin, 1982).

It is argued that the only well demonstrated response to apnea which man shares with other diving species is bradycardia (Craig, 1963). Cardiac output in man, for example, has been seen to decrease (Kawakami et al., 1967), to increase (Paulev and Weltergvist, 1968) or to remain unchanged during apnea (Hong et al., 1971; Lin et al., 1979). Further suggestions have arisen that peripheral vasoconstriction may not be causally related to the development of bradycardia (Song et al., 1969) and that already known physiological mechanisms may be accountable for the reduction in heart rate during apnea, rather than an oxygen-conserving reflex as such (Craig and Medd, 1968). Even where researchers uphold the view that the integrative circulatory responses to apnea in man are qualitatively similar to those seen in diving

species (Scholander et al., 1962; Angell-James and de Burgh Daly, 1972; Lin, 1982; Schilling et al., 1984), the existence of such a response may not be synonymous with oxygen conservation (Lin, 1982). In contrast to the prompt and rapid haemodynamic adjustments manifest during apnea in diving animals, the response occurs to a much lesser degree in man (Lin, 1982; Schilling et al., 1984), who shows an ineffective conservation of oxygen (Craig and Medd, 1968; Lin, 1982). Although evidence suggests that reduced peripheral blood flow may accompany apnea, and particularly apneic immersion, such a reduction is insufficient to reduce oxygen transfer. The transfer of oxygen from the blood to the tissues depends on the chemical environment of the blood and tissue (Lin, 1982). It has been demonstrated that the pulmonary exchange of oxygen during cycle ergometry is the same with or without apnea (Craig and Medd, 1968). Oxygen bound to haemoglobin is rapidly released to the tissue during apnea; this occurs in spite of a reduced blood flow, since the chemical environment during apnea favours unloading of oxygen from haemoglobin (Lin, 1982). The ability of the organism to conserve oxygen in the face of apnea therefore depends on the complete cessation of blood flow to certain tissue beds (Lin, 1982).

Despite equivocal opinions regarding the existence, extent and modification of apneic bradycardia, researchers are in general agreement that an unusual circulatory state does exist during breath-holding, particularly when accompanied by immersion in water. Whether this constitutes part of a phylogenetically ancient reflex, or whether it can be attributed to neural influences or mechanical changes in the cardiovascular system brought about by respiratory arrest per se remains unclear.

THE INCIDENCE OF APNEIC BRADYCARDIA IN MAN

Investigations of the apneic heart rate in man and the explanations provided for the observed responses are many and varied. While exceptions to the rule do exist, it would appear that a bradycardial response to apnea at rest is reasonably well established, with a more pronounced reduction in heart rate occurring during apneic facial or whole body immersion than during breath-holding on land. Where a reduced heart rate at rest is clearly established, however, the literature is extremely equivocal on those factors said to modify the response (see Table I). Equally as contentious have been reports of apneic heart rate responses to exercise in both terrestrial and aquatic environments.

Breath-holding on land by resting man has repeatedly been observed to result in bradycardia (Irving, 1963; Craig, 1963; Hong et al., 1967; Paulev, 1968; Strømme et al., 1970; Bergman et al., 1972; Paulev and Hensen, 1972; Speck and Bruce, 1978; Oldridge et al., 1978; Gooden et al., 1978; Magel et al., 1982; Scott & Candler, in press). The degree to which the bradycardia is evident, however, appears to differ, with a pronounced response observed by some (Bergman et al., 1972; Oldridge et al., 1978; Magel et al., 1982; Arnold, 1985) and only a mild reduction recorded by others (Hong et al., 1967; Paulev, 1968; Strømme et al., 1970; Gooden et al., 1978). The lowest human heart rate associated with apnea would appear to be 5.6 b.min^{-1} , that of a 41 year old male during facial immersion in ice water ($0-2^{\circ}\text{C}$) (Arnold, 1985). Bove et al. (1968) observed that although apnea while resting on land resulted in bradycardia in adults, the same procedure failed to elicit a reduction in heart rate in children (8-14 yrs). Although bradycardia has been

Table I : Examples of the controversy regarding apneic bradycardia and factors modifying the response.

Source	Land Bradycardia	Immersion Bradycardia	Remarks
Craig (1963)	-	✓	Underwater diving, irrespective of age and underwater experience independent of depth.
Irving (1963)	✓	✓	At rest and during exercise; Greater bradycardia in experienced swimmers.
Harding <u>et al</u> (1965)	x	✓	At rest; immersion to xiphisternal joint.
Angelone & Coulter (1965)	✓	-	At rest; related to inspired lung volume.
Kawakami <u>et al</u> (1967)	x	✓	At rest; facial immersion; related to water temperature.
Craig & Medd (1968)	x	x	Apnea during cycle ergometry.
Song <u>et al</u> (1969)	✓	✓	At rest; related to inspired lung volume, intrapleural pressure and level of body submersion.
Strømme <u>et al</u> (1970)	✓	✓	At rest and during exercise; no relation to metabolic intensity or physical fitness.
Oldridge <u>et al</u> (1978)	✓	✓	At rest and during exercise; related to underwater experience.
Wolf (1978)	-	✓	Facial immersion. Related to water temperature inspired lung volume and psychological state.
Magel <u>et al</u> (1982)	✓	✓	At rest. Related to water temperature and inspired lung volume.
Hayward <u>et al</u> (1984)	-	✓	Sudden whole-body immersion. No relation to water temperature.
Sterba & Lundgren (1985)		✓x	At rest. Only in water temperatures below 32°C.
Gemma & Wells (1987)	-	✓	During synchronised swimming.

demonstrated at rest in the seated, prone and supine positions (Paulev, 1968; Bove et al., 1968; Paulev and Hensen, 1972; Magel et al., 1982), Kawakami et al. (1967) observed no significant changes in heart rate during apnea whilst seated at rest on land. A similar report was made by Raper et al. (1967). In contrast, reports of tachycardia in response to apnea at rest on land have been made (Harding et al., 1965; Campbell et al., 1969). This was likened in one instance to the heart rate pattern which accompanies the Valsalva manoeuvre, where the effect of a fall in blood pressure on the arterial baroreceptors is to initiate a reflex increase in heart rate (Harding et al., 1965).

Harding and associates found, however, that the tachycardial response to holding the breath in air was converted to a bradycardia on immersion in water. This reversal of the response occurred when the water was at the level of the xiphisternal joint. The contention that resting apnea during immersion in water, particularly of the face, with or without whole body immersion, results in more pronounced bradycardia than during breath-holding in air is widely supported (Olsen et al., 1962; Song et al., 1969; Strømme et al., 1970; Bergman et al., 1972; Speck and Bruce, 1978; Gooden et al., 1978; Magel et al., 1982). The tachycardia normally evident on land during apnea with Valsalva following a normal inspiration was not observed when the procedure incorporated facial immersion in 24 °C water (Magel et al., 1982). In addition, when Valsalva was performed following maximal inspiration, facial immersion produced significant bradycardia.

In an examination of the relative importance of wetting the face compared with simple breath-holding or applying a cool facial stimulus

other than water during apnea, wetting of the face was considered to be an important factor in eliciting the most pronounced bradycardial response (Gooden et al., 1978), although the reason for this finding remained to be elucidated. Further evidence to support the relative importance of facial immersion has been cited in terms of a combined test manoeuvre, where, after a period of 40s of apnea in air, immersion of the face resulted in a further decrease in heart rate (Strømme et al., 1970). Reversal of the procedure led to a prompt increase in heart rate as the face was removed from the water, despite the continuance of apnea.

Other researchers, however, observed that bradycardia during apnea in air was similar to that obtained with facial immersion (Brick, 1966; Paulev, 1968; Folinsbee, 1974; Oldridge et al., 1978). The drop in resting heart rate which accompanied breath-holding was described as being triggered by apnea per se, with "secondary, and comparatively minor, augmentation with facial immersion" (Oldridge et al., 1978). An important factor in this regard was that this was seen to hold true, provided the water temperature was greater than 25°C. Essentially the same heart rate response - bradycardia - was found during apnea in air and with facial immersion in 25°C and 35°C water (Paulev, 1968), while immersion in cold water (less than 22°C) accentuated this drop in heart rate. More recently, whole body immersion in cool (32°C) water was seen to elicit a marked reduction in heart rate (Sterba and Lundgren, 1985). The apparent facilitation of the response which accompanied immersion in water of varying temperatures led to the conclusion that bradycardia in response to apnea and apneic facial immersion was related to two independent components: an apneic bradycardia and a facial immersion,

or cold bradycardia varying in magnitude with the water temperature (Craig, 1963; Brick, 1966; Paulev, 1968; Magel et al., 1982).

Several authors report that the bradycardia which accompanies apnea at rest persists in man during exercise (Scholander et al., 1962; Irving, 1963; Hong et al., 1967; Strømme et al., 1970; Bergman et al., 1972; Oldridge et al., 1978; Finley et al., 1979; Scott and Candler, in press) and may even surpass that observed during rest. Many of these studies, however, revealed a remarkable lack of rigour with regard to the quantification of exercise intensity either on land or underwater, together with a disregard for the standardisation of the exercise modalities used, often when comparing responses to both environments (see Table II).

An early study stated, for example, that the bradycardia on immersion persisted in man in spite of "vigorous" underwater swimming (Irving, 1963). As Asmussen and Kristionsson (1968) later pointed out, however, if the recorded pre-apneic pulse rates were considered as an indication of intensity, it is unlikely that the exercise would be termed "vigorous". A number of studies report the existence of a bradycardia during underwater dives accompanied by "kicking". Trochus shell divers off Thursday Island exhibited a heart rate reduction which was commonly one-half the pre-dive value during active dives to depths of 3-4 fathoms (approximately 5.5 - 7.3m) (Scholander et al., 1962); this was considered to be somewhat more pronounced than the bradycardia which accompanied "quiet" apneic submersion. This observation differed from that of Hong et al. (1967) who pointed out, on the basis of their studies of five Korean Ama, that the relative reductions in heart rate

Table II: Examples of the controversial findings regarding bradycardia during apneic exercise.

REFERENCE	BRADYCARDIA	REMARKS
Scholander <u>et al.</u> (1962)	✓	Active and passive (weight belt) dives (N=31)
Craig (1963)	delayed	Active and passive dives (N=13)
Irving (1963)	✓	Slow and vigorous underwater swimming
Sasamoto (1965)	✓ passive	Active and passive dives (N=?)
	✗ active	
Asmusson and Kristiansson (1968)	✓	Cycle ergometry, facial immersion only. (N=6). Intensity relative to pre-apneic HR of 100, 120 and 150-160 b.min ⁻¹ .
Craig and Medd (1968)	✗	Underwater leg and cycling exercise. Absolute intensity (N=10)
Strømme <u>et al.</u> (1970)	✓	Surface swimming. Intensity relative to $\dot{V}O_2$ (N=40)
Bergman <u>et al.</u> (1972)	✓	Cycling exercise (land) at 300 and 600 kpm.min ⁻¹ . Handgrip at 25% MVC (N=10)
Paulev and Hansen (1972)	✗	Under water cycling exercise at 450 and 750 kpm.min ⁻¹ (N=7).
Oldridge <u>et al.</u> (1978)	✓	Cycle ergometry, facial immersion only (N=24)
Finley <u>et al.</u> (1979)	✓	Cycle ergometry, facial immersion only (N=9). 60% maximum work capacity.
Gemma and Wells (1987)	delayed	Synchronised swimming (N=10)
Lin (1988)	✗	Literature review
Scott and Candler (in press)	✓	Underwater swimming and brick carrying (N=13)

during a surface swim were similar to those seen during breath-holding in water at rest, while bradycardia occurred to a lesser extent during active dives to a depth of 5m. An earlier study of the Japanese Ama reported that in the Cachido, who dives to the sea bottom by her own effort, vagal inhibition was insufficiently strong to overcome an initial increase in heart rate during this manoeuvre, although bradycardia did develop over the duration of the dive (Sasamoto, 1965). In contrast, the Funado, who descends to the sea bottom with the aid of a 15Kg mass, demonstrated an immediate bradycardia, with no such transient increase. This concurs with the findings of Craig (1963), that consistent bradycardia accompanied dives made with the use of a weight belt to depths of 24m. He stated, however, that the onset of the response was slightly delayed, and attributed this to the effort required to descend, due to insufficient mass being used to accomplish a rapid descent. In an additional experiment tachycardia was evident with movement of the subject's flippers up and down at a moderate rate and force for 35s during an apneic dive following hyperventilation using 100% oxygen. This author concluded that vagal inhibition was not strong enough to prevent an increase in heart rate associated with exercise under such conditions. A contrary report was made by Sasamoto (1965) using non-divers totally submerged in water, where kicking the lower extremities failed to prevent a decrease in heart rate, although the development of the response was more gradual than at rest. More recently, studies of apneic heart rates during the performance of synchronised swimming figures (Gemma and Wells, 1987; Takamoto, 1987) and during underwater swimming and brick carrying (Scott and Candler, in press) have tended to substantiate earlier suggestions that while a

significant decrease is evident over time, there is either a transient increase in heart rate or a delay in the onset of a bradycardial response during such manoeuvres. It has been suggested that while apparently contradictory reports of an immediate onset of bradycardia upon apnea have been made, unlike those mentioned above, such studies generally involved subjects performing constant workloads in standardised positions (Gemma and Wells, 1987).

The contention that bradycardia persists in man during exercise has been disputed. Lin (1988) states that increased metabolic demands during apneic diving caused, for example, by exercise, lead to a rise in heart rate. In order to elucidate the effects of exercise per se upon the apneic heart rate response, researchers have attempted to examine the effects on the heart rate of breath-holding during known working conditions, both on land and underwater. The observations of bradycardia during exercise made by earlier authors (Scholander et al., 1962; Craig, 1963; Irving, 1963) have been criticised as being based on brief periods, so that a steady state was never reached, and as having no measure of intensity (Asmussen and Kristiansson, 1968). Asmussen and Kristiansson described a variable response to facial immersion during exercise of varying intensities, with some subjects having marked bradycardia and others having little or no change in heart rate. Where bradycardia was evident, the degree of heart rate reduction was smaller during heavy exercise than during light or moderate exercise, a factor which was related to the inability of subjects to hold their breath long enough at heavy work loads for bradycardia to develop fully. Similar variations in the apneic heart rate response to exercise were reported by Paulev and Hansen (1972), where steady state exercise elicited a

small fall in heart rate only in the fittest subjects, and heavier workloads resulted in tachycardia both on land and underwater. In each of these studies, however, the intensity of exercise was fixed in absolute terms. The relative intensity experienced by each individual would therefore differ according to the physical condition of each subject. Under similar experimental conditions to those used by Asmussen and Kristiansson (1968), later studies reported a significantly greater fall in heart rate during apneic facial immersion accompanying exercise than at rest (Bergman et al., 1972; Oldridge et al., 1978). The exercise intensities were set by Bergman and co-workers at 300 and 600 kpm.min⁻¹, classified by them as mild and moderate respectively. In each case decreases in heart rate accompanying apneic "wet dives" were significantly greater than those during apneic "dry dives", performed by lowering the face into an empty basin.

The intensity at which subjects worked during apneic facial immersion was determined relative to the individual by Finley et al. (1979). Apneic facial immersion during exercise at 60% of the individual's maximal work capacity resulted in a 48% reduction in heart rate. Like Asmussen and Kristiansson, Bergman and co-workers and Oldridge and associates, however, Finley et al. studied the apneic heart rate response to facial immersion during cycle ergometry. In each instance the subject remained out of the water; a basin of water was held so that he need only bend his neck to submerge his face. Although this has been termed a "simulated dive" (Asmussen and Kristiansson, 1968; Bergman et al., 1972; Finley et al., 1979), such a procedure obviously has limited application with regard to apneic diving performed in situ, where the hydrostatic pressure, thermal and psychological effects of whole body

immersion cannot be discounted. Furthermore, the method used by these researchers does not account for the possible reflex slowing of the heart which may be caused by pressure on the carotid sinus when the neck is flexed.

The effects of whole body immersion during apneic exercise of known intensity have been studied, both in absolute (Craig and Medd, 1968), and relative terms (Strømme et al., 1970). Craig and Medd, whose subjects exercised by using the lower extremities to lift weights a known distance by the use of a pulley system, observed that exercise led to tachycardia, the effects of which were decreased by about half as a result of apnea either on land or underwater. Since the exercise modality was identical in both environments, the comparison between the two conditions was valid in this case. Bradycardia was, however, observed in response to apneic swimming at a depth of approximately 3m (Strømme et al., 1970). The intensity of the activity was controlled relative to each individual by ensuring that breath-hold manoeuvres took place only once a steady state of swimming had been accomplished (stabilised heart rate). The pre-apneic steady state heart rates of approximately 120 b.min^{-1} would indicate that a moderate exercise intensity was attained.

The influence of isometric exercise on the apneic heart rate response is reportedly qualitatively and quantitatively different from the influence of dynamic exercise (Bergman et al., 1972). Heart rates were measured during a sustained isometric handgrip at 25% of maximum voluntary contraction (MVC), during both "wet" and "dry" dives, as described earlier. Bradycardia was less severe than during resting dives. It was

concluded that the combination of isometric exercise and apnea yielded a simple sum of the responses that would be expected to each alone. In contrast, the apparently paradoxical combination of stimuli toward tachycardia (exercise) on the one hand, and bradycardia (apnea) on the other may result in a marked augmentation of the bradycardial response during dynamic exercise. Bergman and associates suggest that cardiac sympathetic responses may be more readily called into play during isometric exercise than during mild dynamic exercise.

Two possible explanations for the increasing decrement with increasing heart rates during dynamic exercise have been proposed. The first involves an interaction between the vagus and sympathetic nerves whereby sympathetic tone may be directly reduced by an increase in vagal tone (Finley et al., 1979). Acetylcholine released by vagal traffic can result in a reduction of sympathetic activity (Levy, 1971), with the result that high background heart rates, seen for example during exercise, may produce a greater overall drop in heart rate. The second explanation lies in the modulation of vagal discharge by information available from afferents from many intracardiac and vascular receptors. This theory proposes, then, that heart rate itself could modulate the apneic heart rate by affecting inhibition of vagal discharge to allow greater vagal activity during apnea when background heart rates are greater (Finley et al., 1979).

It is suggested that many of the existing discrepancies with regard to apneic bradycardia, either at rest or during exercise, in terrestrial or aquatic conditions, are due in part to the large variety of research protocols employed, and in part to a lack of uniformity in the

interpretation of the obtained results. Lin (1982) points out, for example, that computation of the heart rate change during apnea depends on how the pre-dive value is derived. Criticism levelled at previous studies has included the fact that often the influence of a high control heart rate measured just prior to breath-holding has been ignored, resulting in reports of substantial bradycardia (Paulev and Hansen, 1972; Magel et al., 1982). A high percentage reduction in heart rate is likely only if the peak heart rate at the onset of breath-hold (inspiration) is taken as the pre-dive value; otherwise much smaller reductions are obtained (Lin, 1982). Lin cites an example where a relative bradycardia of 19% to 51% could be produced from the results of a single experiment, depending on the values taken for the pre-breath-hold heart rate. The determination of pre-apneic values is complicated by the heart rate change occurring in phase with respiration; thus it is imperative that computational procedures are made clear (Lin, 1982).

A further contributory factor to the controversy which accompanies this field of research is the adoption of two different approaches: that of the field physiologist working under natural conditions, and that of the laboratory worker attempting to examine the influence of a single factor under controlled experimental conditions (Paulev and Hansen, 1972). Often results are generalised from one situation to another: cardio-pulmonary changes during breath-hold dives, for example, deduced from studies conducted in the laboratory (Lin, 1984). In such an artificial setting the psychological and environmental stressors of actual diving, or whole body immersion are absent (de Mojà et al., 1987). Thus in many instances, relating to both approaches, cognisance

is not taken of the entire network of causation. The result is a lack of control of many of those factors said to modify the apneic heart rate response. A large number of studies, for example, use both male and female subjects, often apparently without the researchers having established whether significant differences existed between apneic heart rate responses of the sexes. Similarly, in a random sample of 20 reported studies, the overall range in age of the subjects was from 6 to 67 years, with the range within studies varying between 5 and 56 years. Since so many factors are believed to modify the heart rate response, the implications of which will be discussed later, it is essential that as many as possible of these be controlled if meaningful comparisons are to be made from one condition to another.

THE ONSET AND COURSE OF APNEIC BRADYCARDIA

In those studies where the existence of bradycardia during apnea is clearly established, there arise two further issues of contention: the temporal onset of the response, and the time course required for the maximal development of the response.

The occurrence of an anticipatory bradycardia at times prior to breath-holding has been reported (Wolf, 1978; Gemma and Wells, 1987). In each case, anticipatory reductions in heart rate were measured just prior to facial immersion. This led Wolf to suggest that the whole process may be initiated from the forebrain, while Gemma and Wells attributed their observation to a conditioned response in anticipation of immersion. They cite as evidence for their theory the conditioned bradycardia which can be produced in seals, alligators and other diving animals in response to sound or visual stimuli. In contradiction to

these reports, tachycardia has been measured in humans immediately prior to breath-holding, both with and without facial immersion (Hong et al., 1970; Strømme et al., 1970; Bergman et al., 1972; Folinsbee, 1974; Magel et al., 1982; Hayward et al., 1984). This was described as being largely emotionally induced, but was attributed in part to a deeper inspiration before apnea (Bergman et al., 1972).

Much of the literature would contend that the onset of bradycardia is immediate upon apnea (Irving, 1963; Kawakami et al., 1967; Bove et al., 1968; Strømme et al., 1970; Paulev and Hansen, 1972; Landsberg, 1975; Lin, 1982 and 1988; Hayward et al., 1984). Sudden whole body immersion into water also appears to produce an immediate bradycardia (Hayward et al., 1984) whilst swimming (Strømme et al., 1970), diving (Irving, 1963; Landsberg, 1975), and facial immersion only (Kawakami et al., 1967; Bove et al., 1968) have produced a similar result, prompting the conclusion that the changes in heart rate were so rapid they could only be reflexly initiated (Irving, 1963; Kawakami et al., 1967). According to Lin (1982), the onset of bradycardia occurs within a few heartbeats following respiratory arrest. It is his opinion that where reports have indicated the slow development or establishment of a bradycardial response in humans, they refer not to the onset, but to the slow attainment of the maximal bradycardial response.

The contention that bradycardia develops gradually during apnea, usually with a latency of 5-10s, is common to many studies (Craig, 1963; Asmussen and Kristiansson, 1968; Hong et al., 1970; Arnold, 1985; Gemma and Wells, 1987; Scott and Candler, in press). During dives to successively greater depths, ending at 24m, no variation in the degree

of bradycardia was observed, but the onset of the response was delayed slightly with each deeper dive (Craig, 1963). In some studies a transitory tachycardia was observed, particularly in relation to apneic exertion of some form, followed by a progressive decrease in heart rate over the duration of the breath-hold (Scholander et al., 1962; Gemma and Wells, 1987; Scott and Candler, in press). This has been explained as being the consequence of a possible Valsalva effect resulting from the effort of submergence (Scott and Candler, in press), since the Valsalva effect during apnea is known to override the bradycardial response (Magel, et al., 1982). The measurement of the heart rates during the performance of synchronised swimming figures (Gemma & Wells, 1987) revealed that, in general, heart rates increased immediately after facial immersion for each "figure" and only declined three to six body positions (movements) later. The decline in heart rate then continued throughout the duration of the breath-hold. A continued decline in heart rate over 120s of facial immersion was also reported by Magel et al. (1982).

A progressively declining heart rate over the duration of the entire breath-hold is not commonly reported. Rather, the majority of researchers propose that once the maximal bradycardial response is attained (ie. minimum heart rate), a plateau in heart rate is evident until termination of apnea. In contrast to both of the above, one study reported that having reached its lowest level within 30-40s, the apneic heart rate tended to increase (Hong et al., 1970). Difficulties also arise in determining that point at which the maximal response is attained. The reported time-course required for the development of a maximal response varies from within the first 10s (Bove et al., 1968;

Paulev and Hansen, 1972; Gooden et al., 1978), between 15-20s (Sasamoto, 1965; Bergman et al., 1972; Landsberg, 1975), 20-30s (Folinsbee, 1974; Hayward et al., 1984; Arnold, 1985; Scott and Candler, in press), and even between 40 and 60s (Scholander et al., 1962; Strømme et al., 1970).

Much of the dispute concerning the attainment of a maximal response may be due to the tendency for researchers to consider absolute values only; in other words, that the lowest heart rate occurs within a specific time following the onset of apnea. The magnitude of the heart rate reduction in humans depends on a variety of factors (Lin, 1982), the presence or absence of which may influence the temporal development of the response. Attempts have been made to relate both the degree of bradycardia and the length of time required for its attainment to the breath holding ability of the individual. Since individual differences in breath-hold duration do occur (Hayward et al., 1984), and since the effects of prolonged duration upon the bradycardial response are as yet unclear, it is possible that differences in this regard may account for some of the existing discrepancy. The tendency of some authors to set a pre-determined breath-hold duration may produce misleading results in terms of the attainment of a maximal response. Paulev and Hansen (1972), for example, found low heart rate levels to be reached within the first 6-8s of their 15s apneic periods. This would preclude the possibility of further reductions in heart rate had the apneic period been longer. Scott and Candler (in press) in contrast, reported that the bradycardial effect became more pronounced after the first 20% of breath-hold duration and levelled off during the final 20% of apnea, thus accounting for individual differences in the duration of apnea.

Attempts to establish a relationship between the degree of bradycardia and breath-hold duration have yielded equivocal results. Asmussen and Kristiansson (1978) emphasised that the final level of bradycardia achieved during moderate and severe dynamic exercise appeared to be limited by the need to breathe: the degree of pulse slowing was less during heavy exercise, when breath-hold time was shortened, than during light or moderate work. Gemma and Wells (1987) found the degree of bradycardia increased significantly the longer the breath was held during synchronized swimming, whilst others have referred to individual variability in the degree of bradycardia depending on breath-holding ability (Landsberg, 1975; Oldridge et al., 1978). Strømme et al. (1970), whilst stating that the breath-hold duration during exercise was too short to allow a conclusion as to the relation between the degree of bradycardia and breath-hold time, observed that the slowing of the heart appeared to reach a constant level after 30s. These authors stated that only within certain variable time limits could the degree of bradycardia be said to depend on the length of the breath-holding time. Others have denied the existence of a relationship between breath-hold duration and the extent of bradycardia. An examination of the effect of "diving" bradycardia upon breath-holding time revealed that even where breath-hold duration was shortened by as much as 55% in cold (20°C) compared to cool (32°C) water, the reductions in heart rate were similar (28 and 26% respectively) (Sterba and Lundgren, 1985). These observations corroborate others, where a similar lack of correlation between breath-hold time and the extent of heart rate was noted (Olsen et al., 1962; Wolf, 1978; Hayward et al., 1984).

Termination of apnea has led to a variety of effects on the heart rate. Recovery from resting and diving apnea has been described as an immediate increase to pre-apneic levels (Scholander et al., 1962; Hong et al., 1967; Gooden et al., 1978), although it has been reported that despite an immediate increase in heart rate, pre-apneic levels may not in fact be reached within 20s (Magel et al., 1982).

According to Paulev (1968), the level to which heart rates rise depends on the transmural, thoracic pressures during apnea, with higher pressures resulting in lower post-apneic heart rates. Asmussen and Kristiansson (1968) found that the return to pre-apneic values after facial immersion was delayed, so that the lowest heart rates could be found once breathing had been resumed.

It can be seen, therefore, that there exists a tremendous amount of disagreement in relation not only to the onset and course of apneic bradycardia, but to its very existence, either at rest or during exercise. Factors contributing to this can be summarised briefly as including the large variety of research protocols employed, a lack of uniformity in the interpretation of the obtained results and a disregard for the control of many of those factors said to modify the bradycardial response in man.

CONTROL OF THE HEART - CAUSES OF BRADYCARDIA

The heart is inherently rhythmic. If left to this rhythmicity, it would beat steadily between 20 and 80 b.min^{-1} (McArdle et al., 1986). However, extrinsic controls of cardiac function facilitate rapid changes in the heart rate in order to meet the demands of changing conditions and maintain homeostasis.

Intrinsic regulation is initiated by specialised neuromuscular tissue of the heart. The heart beat is generated by the sinoatrial (SA) node, situated in the posterior wall of the right atrium. Each time a rhythmic impulse is generated in any single fibre of the SA node, or pacemaker, it spreads immediately into the surrounding atrial muscle. Impulses from the SA to the atrioventricular (AV) node are preferentially conducted by internodal tracts consisting apparently of a mixture of fibres similar to Purkinje fibres and ordinary muscle fibres (Jacob et al., 1982). The AV node is located beneath the endocardium of the right atrium at the base of the interatrial septum. Here the impulse is delayed slightly to provide sufficient time for the atria to contract and force blood into the ventricles (McArdle et al., 1986). The fibres which leave the AV node constitute the Purkinje system which includes the left and right branches of the AV bundle (Bundle of His) and the peripheral Purkinje network. These fibres transmit the impulse rapidly through the ventricles. Each ventricular cell is stimulated within about 0.06s from the passage of the impulse into the ventricles, resulting in a unified and simultaneous contraction of the entire musculature of both ventricles (McArdle et al., 1986). The electrical activity about the heart creates an electrical field throughout the

body, including the skin (McArdle et al., 1986). A graphic record of the heart's activity can be obtained by means of an electrocardiograph (ECG) which records changes in electrical potential during the heart beat.

Extrinsic influences are superimposed on the inherent rhythmicity and conductivity of the myocardium (McArdle et al., 1986). These originate in two cardiac centres located in the medulla: The cardioinhibitor centre and the cardioaccelerator centre. The cardioinhibitor centre acts through the parasympathetic division of the autonomic nervous system (vagus nerve); the cardioaccelerator centre acts through the sympathetic division. The changes in the activity of the heart are apparently brought about by reciprocal variations in the activity of these centres (Jacob et al., 1982).

Anatomically, the efferent pathway of the autonomic system is unique. Whereas a skeletal muscle is innervated by a neuron with its cell body in the central nervous system and its axon extending without interruption to the muscle, smooth muscle, the heart and glands are innervated by a two-neuron chain (Jacob et al., 1982). This comprises a preganglionic neuron with its cell body in the central nervous system and axon extending to a ganglion outside the central nervous system and a postganglionic neuron (Jacob et al., 1982). The cell bodies of the sympathetic postganglionic neurons are always located in ganglia of the sympathetic chain or in various other discrete ganglia in the abdomen or thorax, rather than in the excited organ itself (Guyton, 1986). Sympathetic nerves which function as augmenters of the activity of the heart consist of postganglionic neurons arising from cell bodies in

paravertebral ganglia (Jacob et al., 1982). These nerves supply mainly the thoracic viscera and the major part of the head and neck regions.

The parasympathetic system, like the sympathetic, has both pre- and postganglionic neurons. Except in the case of a few parasympathetic cranial nerves, however, the preganglionic fibres pass uninterrupted to postganglionic neurons located in the wall of the organ that is to be excited by parasympathetic impulses (Guyton, 1986). Probably 75% or more of all parasympathetic nerve fibres are in the vagus nerve, passing to the entire thoracic and abdominal regions of the body (Guyton, 1986). However, the vagus nerve is not entirely parasympathetic in function, since it carries some voluntary skeletal nerve fibres to the laryngeal and parhyngeal muscles and some afferent nerve fibres from the pressoreceptors of the arteries and from stretch receptors of the lungs to the medulla (Guyton, 1986). The vagus nerve supplies parasympathetic nerves to the heart, pancreas and smooth muscles of the lungs and digestive tract (Jacob et al., 1982)

The heart is well supplied with both sympathetic and parasympathetic nerves. These nerves affect cardiac function in two ways: by changing the heart rate and by changing the strength of contraction of the heart (Guyton, 1986). The atria are supplied by both sympathetic and parasympathetic nerves, the ventricles largely by sympathetic nerves. There is a basic functional difference between the respective postganglionic neurons of the parasympathetic and sympathetic systems. Increased stimulation of the sympathetic nerves increases the release of norepinephrine by the nerve endings and increases the impulse rate of the SA node (Jacob et al., 1982). The vagus nerve, on the other hand,

exerts a strong, continuous, restraining action on the heart (Jacob et al., 1982). Increased stimulation of the parasympathetic nerves decreases the impulse rate at the SA node due to an increase in the release of acetylcholine by nerve endings (Jacob et al., 1982). In addition, there is a reduction in the excitability of the AV junction fibres between the atrial musculature and AV node, thereby slowing transmission of the cardiac impulse into the ventricles (Guyton, 1986). Intense vagal stimulation can stop the heart for several seconds (Jacob et al., 1982).

The importance of parasympathetic efferents in the bradycardial response to apnea and apneic diving or facial immersion is well established (Angell-James and de Burgh Daly, 1972; Gross et al., 1976; Finley et al., 1979; Lin, 1982). In animals, either vagal section or atropine administration has abolished the bradycardial response (Finley et al., 1979; Lin, 1982). Similar results have been observed in man following atropine administration, indicating a predominant parasympathetic tone (Heistad et al., 1968; Astrand and Rodahl, 1977; Finley et al., 1979). Finley et al. (1979) reported that the elimination of the bradycardial response to facial immersion by atropine occurred both at rest and during exercise.

The effects of sympathetic influences upon the heart rate response to apnea and apneic diving are less clear. According to Finley et al. (1979), sympathetic influences during apneic facial immersion are reflected in a number of concurrent phenomena: namely peripheral vasoconstriction and diastolic and systolic hypertension. These authors studied the effects of sympathetic blockade upon the heart rate response

to facial immersion. Similar decrements in heart rate to those seen without blockade occurred under both β -blockade with propranolol and α -blockade with phentolamine. While reliable diastolic blood pressure measurements were not obtained, systolic blood pressure was unaffected by facial immersion under α -blockade. It was concluded that while sympathetic stimulation may occur during apneic facial immersion, neither α - nor β -sympathetic stimulation are essential to the production of bradycardia.

Earlier, Song et al. (1969) had suggested that peripheral vasoconstriction was not causally related to the development of bradycardia. This followed observations of identical decreases in forearm blood flow during apneic facial and apneic feet submersion in cold water, although the bradycardial response was only evident in the former condition. A previous study by Heistad et al. (1968) indicated that while arterial blood pressure increased during face submersion, the bradycardial response did not parallel this rise in blood pressure. Furthermore, these authors reported that immersion of the face following the administration of atropine did not significantly alter the magnitude of the reduction in finger or forearm blood flow or the rise in arterial blood pressure observed before atropine. However, the bradycardial response evident before atropine did not occur.

Such findings tend to question the validity of a mechanical theory which places emphasis on the role of systemic arterial blood pressure in initiating the bradycardial response via stimulation of the baroreceptors. The systemic arterial mechanoreceptors are located in the walls of a number of large arteries, especially the aortic arch and

carotid sinuses (Jacob et al., 1982). Mechanical deformation of the walls of the vessels acts as the normal stimulus of these receptors; they are thus stretch receptors, and pressure as such is not an adequate stimulus (Astrand and Rodahl, 1977). However, an increase in the intravascular pressure does expand the vessel wall, thereby increasing the rate of impulse transmission from the receptors. The baroreceptor impulses inhibit the vasomotor centre of the medulla and excite the vagal centre (Guyton, 1986). Hence, the principal response to increased arterial blood pressure is vasodilation throughout the peripheral circulatory system, together with decreased cardiac rate and strength of contraction (Jacob et al., 1982; Guyton, 1986).

The concept of increases in arterial blood pressure playing a primary role in the initiation of bradycardia by means of enhanced stimulation of the sino-aortic baroreceptors during apnea has been well subscribed to (Paulev, 1968; Speck and Bruce, 1978; Magel et al., 1982). Speck and Bruce (1978), for example, stated that an inverse relationship existed between heart rate and mean arterial pressure. It has been suggested that the physiological mechanisms accountable for the development of bradycardia are related to changes in intrathoracic pressure and their effects upon venous return and diastolic filling (Paulev, 1968; Magel et al., 1982). According to Song et al. (1969), provided that no Valsalva manoeuvre is performed, intrathoracic pressure becomes more negative in proportion to the depth of inspiration, thus facilitating venous return to the right heart (Jacob et al., 1982). In accordance with Starling's Law of the Heart, this should increase the stroke volume, and consequently systemic arterial pressure, thus adding a further stimulus to the baroreceptors (Craig, 1963; Magel et al.,

1982). This is questioned by Folinsbee (1974), who suggests that there is an elevation of the intrathoracic pressure during breath-holding, which causes a decrease in venous return and an accumulation of blood in the peripheral veins. Craig (1963) stated that when the breath is held following a maximal inspiration there is an increase in intrathoracic pressure as the inspiratory muscles relax, at least to the extent of the relaxation pressure of the lungs and thorax. Nevertheless, venous return from the abdomen continues to be enhanced by the pressure differential between the abdominal and thoracic cavities (Agostini and Rahn, 1960).

From a mechanical point of view, water immersion induces more pronounced cardiovascular adjustments. When the body is immersed in water in an upright position, a graded hydrostatic pressure is exerted over the whole of the chest, least at the highest point and greatest at the deepest (Jarrett, 1965; Lin, 1982). The transdiaphragmic pressure increases due to a greater compression of the abdomen as compared to the chest (Song et al., 1969; Lin, 1982), resulting in a blood shift into the thoracic cavity. Increases in central blood volume of approximately 0.7l have been reported during head out water immersion (Löllgen et al., 1980). In accordance with the above, Song et al. (1969) stated that the extent of apneic bradycardia obtained with the body immersed in different levels of water was proportional to the level of body submersion. These authors suggested that the importance of pressure differentials in relation to venous return was clearly indicated. Contradictory findings were, however, reported by Craig (1963) who observed that the degree of bradycardia evident upon immersion in water was not related specifically to depth. Although Craig observed a slight

delay in the onset of bradycardia the deeper the subject dived, he attributed this to the effort required by the subject to descend.

Paulev (1968) reported that following maximal inspiration during whole body immersion negative intrathoracic pressure (defined as the pressure in the pleural space minus the atmospheric pressure), elicited bradycardia when the breath was held. In contrast, positive intrathoracic pressures (+10 to +20 mmHg) induced tachycardia. It was proposed that under such conditions impaired venous return led to a fall in systemic arterial pressure and a subsequent lowering of the stimulus to the baroreceptors (Paulev, 1968). Paulev observed a decrease in the resting mean arterial pressure of 35 to 47% during Valsalva and concluded that this constituted a sufficient stimulus to the baroreceptors to explain the observed changes in heart rate. This viewpoint was supported by Magel et al. (1982), who stated that the bradycardia or tachycardia produced during apnea either in air or during face immersion was related to the breath-hold condition. These authors observed that bradycardia was greater during apnea following maximal inspiration than following normal inspiration. However, tachycardia accompanying apnea was noted when subjects performed a Valsalva manoeuvre after both normal and maximal inspiration while seated on land. When subjects performed breath-holds with face immersion, bradycardia resulted in spite of Valsalva manoeuvres, with a greater response obtained in cold water (5°C) than in warm water (24°C). Magel and co-workers pointed out, however, that the extent of bradycardia during face immersion was significantly less when intrapulmonary pressure was elevated during Valsalva than when apnea was performed under identical conditions with an open glottis. These findings were

attributed in part to a decreased stimulus to the sino-aortic baroreceptors as a result of diminished venous return and lower systemic arterial blood pressure.

As noted earlier, however, several authors have suggested that hypertension and peripheral vasoconstriction, the result of sympathetic stimulation, are not causally related to the production of bradycardia during apnea, since this response has been shown to occur without changes in arterial blood pressure, or peripheral vasoconstriction (Song et al., 1969; Gooden, 1972; Finley et al., 1979). While the systemic arterial baroreceptors may not be responsible for the initiation of bradycardia, it is likely that this response is enhanced by the increased stimulation of the baroreceptors. According to Lin (1982), hypertension is a consistent finding in man during breath-holding, and the magnitude of the increase in mean arterial blood pressure is proportional to breath-hold time. In contrast to Magel et al., (1982) and Paulev (1968), who attributed a rise in arterial blood pressure to increased stroke volume, Lin stated that changes in cardiac output and stroke volume during apnea were insignificant from pre-breath-hold values, indicating that the enhanced preload of the heart (bradycardia, high central venous pressure and volume) was opposed by peripheral vasoconstriction.

Several authors have pointed out that it is unlikely that any one factor is solely responsible for evoking a bradycardial response to apnea, immersion and apnea during immersion (Song et al., 1969; Angell-James and de Burgh Daly, 1972; Gross et al., 1976; Speck and Bruce, 1978; Magel et al., 1982; Lin, 1982). Rather, it is a complex integration by

the central nervous system of the inputs from several receptor groups that is responsible for control of the heart and circulation (Angell-James and de Burgh Daly, 1972; Gross et al., 1976). Investigations into the causes of apneic bradycardia may have been compounded by the fact that in many situations the effector response to a given change of a single input has been studied under controlled experimental conditions. As Angell-James and de Burgh Daly (1972) point out, the results may be quite different when accompanied by simultaneous changes in other cardiovascular or respiratory inputs. Thus they suggest that although the role of the baroreceptors may be only of secondary importance in initiating bradycardia in terrestrial vertebrates, the possibility exists that a resetting of the baroreceptor reflex occurs upon immersion in water:

By this is meant that the curve representing the normal relationship between mean arterial pressure and heart rate is shifted in such a way that at the same blood pressure the heart rate is lower, compared with the pre-dive value (Angell-James and de Burgh Daly, 1972, pp 333).

In addition, Guyton (1986) states that since some signals that pass down the vagus nerves to the heart can bypass the vasomotor centre, the relationships that exist between constriction, pressure and heart rate are not invariable.

In contrast to those authors who describe the initiation of apneic bradycardia in terms of the mechanical and haemodynamic properties of the cardiovascular system, others propose a neural theory by means of explanation (Andersen, 1963; Angell-James and de Burgh Daly, 1972). While it is accepted that the vagus nerve constitutes the efferent pathway of the bradycardial response, it is suggested that afferent

impulses responsible for the initiation of the response are mediated by the trigeminal nerve (Andersen, 1963; Strømme et al., 1970; Angell-James and de Burgh Daly, 1972). Following studies using decerebrated ducks, Andersen (1963) concluded that the cardiovascular and respiratory responses to immersion were medullary reflexes and as such did not require cortical or thalamic integration of sensory information. He pointed out, however, that basic reflexes may be influenced by activity in higher centres. Furthermore, Andersen observed that in decerebrated ducks in which all trigeminal branches to the region of the beak had been severed, no circulatory or respiratory adjustments could be detected. It was suggested by this author that circulatory modifications were elicited by water per se, acting upon receptors located in the beak of the duck. He was, however, unable to provide information concerning the exact nature of such receptors. Andersen's observations were substantiated by Angell-James and de Burgh Daly (1972), who reported that mechanical, electrical and chemical stimulation of the nose led to bradycardia in the dog, cat, rabbit and in man. It was suggested that receptors in the nasal mucus membrane were responsible for the stimulation of the trigeminal nerve reflexes leading to bradycardia. Similar, although somewhat reduced cardiovascular reflexes from the nose were evident even when apnea was prevented by maintaining pulmonary ventilation constant with a pump.

According to Paulev (1968), however, a trigeminal reflex with unknown receptors could not be held responsible for the bradycardia seen to occur as a result of apneic facial immersion in man. Rather, he drew a clear distinction between a human "face immersion bradycardia", which he suggested was temperature dependent, and the "trigeminal bradycardia"

observed in ducks. Paulev was of the opinion that such bradycardia as was evident in man was attributable to the combined influences of two factors: that bradycardia produced by the apnea per se, and that related to the temperature of the water in contact with the face.

Although Angell-James and de Burgh Daly (1972) caution against inferring from one species to another, several other authors confirm the role of facial receptors stimulating trigeminal reflexes as being of primary importance to the initiation of apneic bradycardia in man (Kawakami et al., 1967; Strømme et al., 1970; Speck and Bruce, 1978; Caputa and Cabanac, 1979). While only speculating about the exact mechanism involved, Kawakami et al. (1967) and Strømme et al. (1970) suggested that the rapidity with which the cardiovascular responses occurred implied that the changes were due to a reflex. Strømme et al., too, proposed that peripheral receptors in the face and nose may be the site of the afferent signal, since the removal of a face mask produced a sudden decrease in heart rate both during apnea at rest and during apneic underwater swimming. Strømme et al. (1970) were of the opinion that the sensation of wetness on the face was important in eliciting a bradycardial response.

Suggestions have been made, however, that the stimulus for bradycardia may be face cooling, rather than water contact as such (Kawakami et al., 1967; Le Blanc et al., 1976 and 1978; Speck and Bruce, 1978; Caputa and Cabanac, 1979). Le Blanc et al. (1976) reported that fanning the face with cold air was sufficient to produce bradycardia, while Kawakami et al. (1967) considered cold receptors in the face to be the main triggering factor in the bradycardial response to immersion. This was

substantiated by Speck and Bruce (1978), who reported that apneic bradycardia was less when facial contact with water was prevented by the use of a facemask, than when water was allowed to touch the face. Research by Harding et al. (1965), however, demonstrated that although tachycardia was apparent upon breath-holding in air, this response was reversed with the body immersed in water to the level of the neck, and as such facial immersion was not an essential element.

The influence of facial cooling and water contact on the face is thought by many to be a minor modifying factor. The essential element in the initiation of apneic bradycardia in man is considered by several authors to be the cessation of respiratory movement per se (Harding et al., 1965; Hong et al., 1967; Strømme et al., 1970; Oldridge et al., 1978; Lin, 1982).

In their research involving dogs, Angell-James and de Burgh Daly (1972) demonstrated that the bradycardia which developed during apneic asphyxia was contingent upon there being no increase in the movement of the lungs. Supporters of this explanation of the initiation of bradycardia generally recognise that while respiratory arrest may be vital to the production of bradycardia in man, the process is then subjected to modification by various neural, mechanical and chemical factors (Lin, 1982). This forms the basis of a proposed multiple-factor hypothesis (Song et al., 1969). Thus, Speck and Bruce (1978) state that when apnea is accompanied by facial cooling the two factors act synergistically to produce a significant decrease in heart rate. Similarly, others point out that immersion in water reinforced the bradycardia developing during breath-holding (Hong et al., 1967; Paulev, 1968; Strømme et al., 1970;

Oldridge et al., 1978; Magel et al., 1982). As pointed out earlier, some attribute the enhanced bradycardia evident with immersion in water to the effects of water contact with facial receptors, whilst others adopt a mechanical explanation, choosing to describe the magnitude of the bradycardia produced in terms of varying intrathoracic pressures, and their resultant effects upon the cardiovascular system. Craig (1963) pointed out that changes in heart rate may not be due to the act of breath-holding per se, but rather to associated changes in intrathoracic pressures.

The respiratory manoeuvre of breath-holding may be executed with various intrathoracic pressures depending upon the degree of lung inflation at the time of respiratory arrest. Several authors contend that bradycardia is greatest when the breath is held following a maximal inspiration (Angelone and Coulter, 1965; Kawakami et al., 1967; Song et al., 1969; Magel et al., 1982). Possible reasons put forward to explain this include the likelihood of decreased intrathoracic pressure and increased transdiaphragmatic pressure occurring with increased inspired lung volume and subsequently influencing the cardiovascular system, together with the possible existence of stretch receptors in the lungs or thoracic wall which initiate a reflex slowing of the heart (Angelone and Coulter, 1965; Song et al., 1969; Magel et al., 1982). In relation to such stretch receptors, Angelone and Coulter (1965) proposed that lung volume may play a role independent of the intrathoracic pressure. Their research demonstrated that a typical cardiac arrhythmia was associated with various lung volume changes. This was seen to occur with or without a thoracic circumference change,

leading the authors to conclude that thoracic circumference was related to changes in heart rate only in so far as it reliably indicated lung volume.

A further proposed explanation for the effects of apnea per se upon the heart rate has been the possible irradiation of impulses between the respiratory and cardiac 'centres' in the brain (Angell-James and de Burgh Daly, 1972). According to Guyton (1986), for example, almost any factor that increases the activity of the vasomotor centre has at least a moderate effect on increasing respiration. Angell-James and de Burgh Daly (1972) point out that impulses arising from the respiratory centres during inspiration cause inhibition of the vagal centre and hence tachycardia. It is possible, however, that removal of this effect during apnea, may in certain circumstances and in conjunction with other modifying factors be strong enough to cause a degree of bradycardia. Furthermore, in contrast to those authors mentioned earlier, Angell-James and de Burgh Daly (1972) state that inflation of the lungs causes tachycardia by means of a reduction in vagal tone. They observed in dogs that if the activity of such a pulmonary vagal reflex was reduced, for example, by apnea in the expiratory position, the bradycardial response was enhanced. Similar findings were reported in man by Wolf (1978), who noted that bradycardia upon facial immersion was most pronounced when the breath was held after exhalation. Such results do, however, appear to contradict the majority of researchers in this area, who claim that inflation of the lungs prior to apnea serves to enhance the bradycardia by means of those mechanisms described earlier.

The process of apneic bradycardia is thought to be further influenced by the effects of the arterial chemoreceptors situated in the carotid and aortic bodies (Kawakami et al., 1967; Strømme et al., 1970; Angell-James and de Burgh Daly, 1972; Gross et al., 1976; Lin et al., 1983). Some controversy exists, however, as to the separate cardiac effects of the carotid and aortic bodies. Gross et al. (1976) point out that while these chemoreceptors exert similar pressor influences on vasomotor function, their cardiac effects are usually opposite; carotid body stimulation in dogs has been seen to produce bradycardia, and aortic body stimulation tachycardia. This pattern of heart rate responses was not consistent with the results of Angell-James and de Burgh Daly (1972), who reported a bradycardial response to selective stimulation of both sets of chemoreceptors. Gross et al. (1976) observed that in human subjects who had undergone bilateral carotid body resection, the typical heart rate response to breath-holding was a marked tachycardia. Earlier studies had suggested that although asphyxia may not be a major factor in eliciting the bradycardia seen during apneic facial immersion, the hypoxia and hypercapnia arising during breath-holding served to intensify and maintain the response (Kawakami et al., 1967; Strømme et al., 1970).

It can be seen, therefore, that in normal apneic man the typical heart rate response is the result of a complex integration of factors (see Figure 1) many of which enhance the effectiveness of others. Furthermore, the strength of the bradycardial response is indicated in that the cardiovascular responses concomitant with apnea or apneic facial immersion would appear to occur in the face of often antagonistic influences. Thus, Gross et al. (1976) conclude that the characteristic

heart rate response to breath-holding in man represents the domination of influences such as the carotid bodies, which mediate vagal bradycardia, over other tachycardia-producing reflexes. An example of such domination may occur in the case of the Bainbridge Reflex. This constitutes a homeostatic reflex mechanism that causes acceleration of the heart beat following an increase in blood pressure in the venae cava and right atrium (Webster's, 1986). Stretch of the right atrial wall may increase the heart rate by as much as 10 to 30% (Guyton, 1986). Since an increase in blood pressure in these areas is likely to occur as a consequence of the shift in blood volume that occurs with apnea and immersion in water, it is possible that this reflex is "overridden" by factors such as those mentioned earlier. A further example of the cardiac reflex associated with apnea and apneic immersion exists in relation to the arterial chemoreceptors. Such receptors normally play an important role in the control of respiration. During water immersion, however, their known stimulant action upon the respiratory centres appears to be partially or wholly blocked, whilst their contribution to the production of the cardiovascular responses would appear to dominate (Angell-James and de Burgh Daly, 1972).

MODIFYING FACTORS

Temperature

The effects of environmental temperature upon apneic bradycardia are believed to be of considerable importance both to the rate and extent of heart rate slowing. They are particularly relevant to water immersion where the specific heat is 1 000 times, and thermoconductivity 25 times

greater than those of air; as a result, heat loss occurs faster in water than in air of the same temperature (Hong, 1984).

A number of researchers contend that apneic bradycardia is temperature-dependent. Indeed, in explaining the initiation of such a response, several authors have proposed that it comprises two independent components: an "apneic bradycardia", varying in magnitude with the lung volume and intrathoracic pressure; and a "cold bradycardia", the size of which depends upon environmental temperatures (Paulev, 1968; Speck and Bruce, 1978; Magel et al., 1982). Cold temperatures, particularly with regard to facial immersion, are said to potentiate the reduction in heart rate (Olsen et al., 1962; Kawakami et al., 1967; Hong et al., 1967; Paulev, 1968; Song et al., 1969; Speck and Bruce, 1978; Magel et al., 1982; Hong, 1984; Lin, 1982 and 1988).

However, the range within which, or a critical temperature at which the reduction in heart rate is augmented, have not been identified. Apneic bradycardia is a multifactorial phenomenon, and the tremendous diversity in protocols used may have hindered efforts to do so. Rather than set a specific temperature range, earlier researchers proposed that the bradycardial response to apnea was augmented by immersion in water 10°C below body temperature (Olsen et al., 1962). Kawakami et al. (1967) later suggested that temperature effects upon bradycardia were mediated via facial cold receptors which showed a most prominent discharge at around 25°C . In terms of thermoreceptor excitation, temperatures of between $15\text{--}30^{\circ}\text{C}$ would result in cold-receptor stimulation, whereas temperatures as low as 0°C would evoke pain- rather than cold-receptor stimulation. The most efficient range in producing bradycardia with

non-painful stimuli was described as being between 15-25°C (Strømme et al., 1970), or 10-25°C (Caputa and Cabanac, 1979). However, experiments which have reported that the bradycardia of face submersion was greatly potentiated by lower temperatures (5°C) indicate that further work in this area is necessary (Hong et al., 1969; Hong et al., 1970; Moore et al., 1972; Magel et al., 1982).

The application of ice to the face during apnea has produced further contradictory results. Whereas decreasing water temperatures during apneic facial immersion caused an increasingly profound bradycardia, heart rate responses when the subject's face was positioned on a plastic bag containing ice did not correspond with this trend (Kawakami et al., 1967; Speck and Bruce, 1978). This may be explained by the fact that ice represents a noxious stimulus, exciting nociceptors rather than cold receptors (Speck and Bruce, 1978). However, Arnold (1985) reported that facial immersion in ice water (0-2°C) frequently produced asymptomatic heart rates of less than 12 b.min⁻¹. A suggestion made by Kawakami et al. (1967) may help clarify this apparent contradiction. While acknowledging the role of pain receptors at very low temperatures, these authors stated that the plastic in which the ice was contained in their study was not a good heat conductor. In contrast, facial cold receptors may have been effectively stimulated following direct contact with ice water in Arnold's study.

The greater bradycardial response generally observed during facial immersion compared to breath-holding in air may thus be ascribed in part to the enhanced cooling effect produced by the contact of the water with the face. The importance of the sensation of wetness on the face in

eliciting a pronounced bradycardia (Strømme et al., 1970) may lie in the rapid conduction of heat from the face afforded by the water, resulting in an effective stimulation of facial cold receptors (Kawakami et al., 1967; Paulev and Hansen, 1972; Moore et al., 1972).

Craig (1971) points out that responses to immersion in water and exposure to cold air are qualitatively the same, but they are much more rapid and the course of events is greatly shortened during immersion. The majority of studies dealing with temperature effects upon bradycardia indicate that facial cooling in particular is important. In an examination of local cooling effects using the cold hand test (5°C water for 2 min) and the cold face test (0°C, with 66 Km.h⁻¹ winds for 2 min) an increase in heart rate was observed in the former condition and a decrease in the latter, though subjects did not breath-hold (Le Blanc et al., 1978). Similarly, Song et al. (1969) reported that apneic bradycardia was greatly potentiated by facial immersion in water of 25°C and 5°C, whereas this potentiation was not seen during feet immersion. This has been explained in terms of the sensitivity of the facial area supplied by the trigeminal nerve to rapid changes in temperature (Song et al., 1969; Paulev and Hansen, 1972).

The rest of the body, in contrast, requires a longer period of time to make the same temperature adaptation (Paulev and Hansen, 1972). Breath-holding with only the face immersed has produced decrements in heart rate equal to those caused by apnea with the body immersed up to the neck in warmer water (15°C during facial immersion, compared to 22°C) (Paulev and Hansen, 1972), and with the whole body immersion in water of identical temperatures (Moore et al., 1972). In apparent

contradiction to the above, Folinsbee (1974) reported that heart rates during breath-holding with facial immersion did not differ significantly from heart rates during breath-hold with the face out of the water. In this instance, however, subjects remained in 31°C and 35°C water for two hours, during which time breath-holds were made. Thus it is possible that the effects of whole body cooling brought about by prolonged exposure to cold were of importance. Thermoneutral temperatures, those at which the body maintains thermal equilibrium, are approximately 25-30°C for an unclothed man in air and 33-35°C in water (Hong, 1984). Thermal drain therefore continues as long as the subject remains in water at lower temperatures. Although body temperatures were not reported by Folinsbee, he observed no significant difference in apneic heart rates at the different water temperatures, leading him to conclude that whole body cooling had no effect upon apneic bradycardia.

The relationship between the degree of bradycardia elicited and the thermal state of the subject is as yet unclear. While the mammalian dive reflex, of which bradycardia is a component, has been implicated in the survival of individuals following near-drowning incidents in cold water (below 20°C) (Sarnaik and Vohra, 1986), it has been suggested that the bradycardia which accompanies extreme hypothermia may be quite different from that during facial immersion (Kawakami et al., 1967; Hayward et al., 1984). The slowing of the heart during apneic bradycardia is accompanied by prolongation of the diastolic phase, together with the disappearance or flattening of P-waves, whereas bradycardia during hyperthermia is characterised by prolongation of the time required for systolic ejection, without P-wave appearance (Kawakami et al., 1967). Furthermore, the bradycardia evident during apneic

diving in cold water has been seen to disappear even while body temperature remains relatively low (approximately 35°C) (Sasamoto, 1965). In contrast, Caputa and Cabanac (1979) have suggested that bradycardia during facial immersion, albeit without apnea, may be related to the thermal state of the subject. Hyperthermic subjects (eardrum temperatures of 38.2°C) demonstrated characteristic bradycardia in response to facial immersion in 15°C water, while hyperthermic subjects (eardrum temperatures of 36°C), on the contrary, demonstrated progressive tachycardia. The opposite effects of face cooling were explained in terms of selective brain cooling. According to Caputa and Cabanac, in hypothermic subjects, the blood flows from brain to face; the only influence of face cooling originating in skin nervous inputs and producing progressive tachycardia comparable to hand cooling. Hypothermia produces a change in blood flow, from face to brain, so that the cavernous sinus and the brain are cooled upon facial immersion in cold water, resulting in a reduction in heart rate. They conclude that this may be the reason for the major bradycardial effect obtained during exercise underwater, and for much of the individual variability in heart rate responses to face cooling.

As discussed earlier, attempts to compare results with regard to the effects of temperature per se upon apneic bradycardia are confounded not only by individual variability, but by the diverse methodologies employed, as well as a lack of control of additional modifying factors. Thus results which contradict the generally stated theory that apneic bradycardia is temperature-dependent do exist. Craig and Medd (1968) for example, found no change in heart rate during apneic exercise with the whole body submerged in water of 27 and 30°C . The strongest

challenge to the theory of temperature dependency was presented by Hayward et al. (1984), who studied apneic heart rates in 160 subjects submerged in water temperatures from 0 to 35°C at 5°C intervals. They concluded that temperature modulation of the dive response was either very weak or non-existent. Their contrary conclusion was attributed to the fact that previous studies had tended to focus upon facial immersion only, had used narrow ranges of water temperatures, and relatively small sample sizes.

Physical Condition and Underwater Experience

It is well established that regular physical endurance activity will produce a resting bradycardia in man (McArdle et al., 1986). Since vagal impulses have been proposed to account for both this and the apneic bradycardia, physically well-conditioned subjects may be expected to exhibit a more pronounced depression of the heart rate during apnea and apneic immersion. The results, however, have been equivocal.

Bove et al. (1968), for example, discovered that, together with an increase in exercise tolerance as evaluated by the Harvard step test, adults showed a significant increase in apneic bradycardia following a ten week training programme. Although Bove and associates failed to provide an adequate explanation for the apparent effects of physical conditioning upon apneic bradycardia, they suggested that diving bradycardia could be used to determine which individuals were in need of physical training. Observation of extreme bradycardia in physically active subjects led Arnold (1985) to postulate that the cardiac vagus nerves were modified by the chronic effect of aerobic, endurance exercise. It is possible, however, that a training-induced bradycardia

in physically conditioned subjects will be manifest in lowered pre-apneic, as well as apneic heart rates. Thus the relative reduction in heart rate may be similar for trained and untrained subjects (Whayne and Killip, 1967). Attempts to relate the degree of bradycardia more specifically to $\dot{V}O_2$ max have failed to reveal a relationship between the two variables (Hong et al., 1970; Strømme et al., 1970; Oldridge et al., 1978; Strømme and Ingjer, 1978; Arnold, 1985). Rather, the effects of regular underwater training per se are considered to be of importance. Although some early studies observed no difference in the responses of those either accustomed or unaccustomed to underwater activity (Craig, 1965; Harding et al., 1965), there is general agreement that the greatest response is seen in competent underwater divers (Scholander et al., 1962; Irving, 1963; Hong et al., 1967; Hong et al., 1970; Oldridge et al., 1978). Swimming skill is not believed to effect the response (Oldridge et al., 1978). The apparent relationship between experience in underwater apneic activity and the extent of bradycardia has been ascribed to a greater breath-holding capacity due to changes in lung volumes and a tolerance to increased carbon dioxide tensions brought about by regular underwater training (Oldridge et al., 1978). Citing the low heart rates characteristically seen in professional skin divers (Scholander et al., 1962; Sasamoto, 1965; Hong et al., 1967; Hong et al., 1970; Shiraki et al., 1985), Asmussen and Kristiansson (1968) propose the existence of a conditioned reflex in such people, as an irradiated inhibition of the circulatory functions during the progressive respiratory inhibition of apnea.

Sex

Sex-based differences in non-apneic heart rate do exist due to fundamental physiological dissimilarities, such as the size of the heart and amount of haemoglobin, between the sexes (Guyton, 1986; McArdle et al., 1986). Common to the majority of the literature, however, is a tendency to pool data for the two sexes, with an apparent lack of regard for the possible effect of such differences upon heart rate responses to breath-holding. Although it has been reported that no significant differences exist (Hayward et al., 1984), this assumption may be responsible for much of the inter-subject variability which is evident throughout the literature. Despite considerable acclimation to apneic diving, the Korean Ama (females) exhibited less bradycardia than other similarly experienced male subjects (Hong et al., 1967). Apart from the suggestion that sexual or ethnic factors may be involved, no further explanation for this difference was given. Further sex-based differences have been reported during recovery from the bradycardia which accompanied cold wind blown on the face. Female heart rates remained significantly lower than initial levels, an effect not observed in male subjects (Le Blanc et al., 1978). Although this study did not incorporate apnea, it serves as an additional indication that further consideration of sex-related factors is necessary.

Age

Few studies have investigated the specific effects of age upon heart rate during breath-holding. The majority of those that have, support the view that young age potentiates apnea-induced bradycardia (Craig, 1965; Hong et al., 1970; Gooden et al., 1978; Lin, 1982; Pearson and

Bruce, 1984). A moderate correlation ($r = 0.63$) has been demonstrated between increasing age and bradycardia and vasoconstriction (Pearson and Bruce, 1984). The strongest responses were observed in young children, with a progressive moderation with increasing age. Gooden (1972) postulated that in the human fetus such cardiovascular adjustments constitute a protective mechanism against asphyxia during birth, and for this reason young infants may retain a particularly sensitive "diving reflex".

In contrast, Arnold (1985) noted that age did not appear to effect the degree of apneic bradycardia over a range from 16 to 54 years. Such findings were apparently supported by observations made by Craig (1963). Although comparisons by age were not made, the bradycardial heart rate of children (10 - 15 yr) and adults (overall age not reported) were of similar magnitude. A comparison of the heart rates of children (8 - 14 yr) and adults (18 - 40 yr) by Bove et al. (1968), however, revealed somewhat different responses. Where adults exhibited a reduction in heart rate during breath-holding in air and underwater, the response only occurred underwater in children.

Despite the apparent contradiction within the literature with regard to the effects of age, these have been overlooked by the majority of researchers to date. As discussed earlier, a random sample of 20 studies revealed that the range within studies varied from 3 to 56 years; of these only three mentioned the specific effects of age upon apneic bradycardia.

Lung Volume

The influence of varying lung volumes have been implicated in the findings of several authors, with the greater number contending that large lung volumes potentiate apneic bradycardia (Irving, 1963; Angelone and Coulter, 1965; Paulev, 1968; Song et al., 1969; Paulev and Hansen, 1972; Gross et al., 1976; Magel et al., 1982; Scott and Candler, in press).

The mechanical theory has been used to explain the mechanism responsible for this effect, since facilitation of venous return by increasingly negative intrathoracic pressure in proportion to the depth of inspiration results in an increase in stroke volume. The subsequent enhanced stimulation of the sinoaortic baroreceptors produces a reduction in heart rate (Paulev, 1968; Song et al., 1969; Paulev and Hansen, 1972; Magel et al., 1982; Scott and Candler, in press). According to Folinsbee (1974), part of the explanation may lie in a more pronounced vasoconstriction which may occur during breath-holding with a full inspiration. In addition, it has been suggested that there is an inverse relationship between heart rate and lung volume that may exist independently of intrathoracic pressure (Angelone and Coulter, 1965; Song et al., 1969; Magel et al., 1982). This has been ascribed to the existence of stretch receptors in the lungs or thoracic wall which initiate a reflex slowing of the heart.

There are, however, observations which are not consistent with either of these theories. In addition to reports of a marked bradycardia with complete absence of a change in blood pressure (Heistad et al., 1968; Finley et al., 1979), several authors have demonstrated that lung volume

did not influence the heart rate during breath-holding (Craig, 1963 and 1965; Kawakami et al., 1967; Asmussen and Kristiansson, 1968; Hong et al., 1970; Folinsbee, 1974).

Others yet have reported that where the degree of lung inflation did make a difference, bradycardia was most pronounced when the breath was held after exhalation (Openshaw and Woodroof, 1978; Wolf, 1978). The proposed explanations once again incorporated the effects of stretch receptors in the lungs, in addition to the slower onset of asphyxia at high lung volumes (Openshaw and Woodroof, 1978). The conclusions of Openshaw and Woodroof were drawn despite their observation that in response to apneic facial immersion, the pulse interval was the same whether the breath was held after inspiration or expiration. The fact that the increase in pulse interval after inspiration was the same both with and without facial immersion, but only occurred with expiration following facial immersion, led these authors to state that the bradycardial effect of facial immersion was therefore not apparent after inspiration.

The role of lung volume per se may be secondary to changes in intrathoracic pressure related to the breath-hold condition. Both bradycardia and tachycardia have been elicited at a given lung volume, depending on the level of the intrapleural pressure (Paulev, 1968; Song et al., 1969; Magel et al., 1982). Magel and associates, for example, noted that apneic bradycardia was converted to apneic tachycardia by elevating the intrapleural pressure during Valsalva. The interaction between lung volume and intrathoracic pressure, and their effect upon apneic heart rates does require further clarification. Song et al.

(1969) observed that at a given intrapleural pressure apneic heart rates were lower the larger the lung volume, whereas Craig (1965) found heart rates to be independent of lung volume for a specific intrathoracic pressure. Furthermore, it has been suggested that a range exists where changes in intrathoracic pressure have no effect upon apneic heart rates. According to Kawakami et al. (1967), this occurred between -5 and +16 cm H₂O. Craig (1965) reported that for a given lung volume bradycardia occurred at intrapleural pressures of between 0-2 cm H₂O, whereas higher pressures increased heart rate. Once again, however, when comparing numerous studies, recognition must be paid to the effects of diverse protocols upon conclusions drawn. Of particular importance here are the effects of varying body positions upon the haemodynamics of the cardiovascular system and the influence of increased central blood volume upon intrathoracic pressure.

The mechanical theory, implicated by several authors as the mechanism responsible for the effects of varying lung volumes upon apneic heart rate, rests upon the premise that intrathoracic pressure is indeed reduced as lung volume increases. It has been demonstrated, however, that breath holding, even in the absence of a Valsalva manoeuvre, may lead to increased intrathoracic pressure with increasing volume (Jarrett, 1965; Folinsbee, 1974; Lin et al., 1974; Ferrigno et al., 1986). This rise in pressure has been attributed to a high chest wall recoil at volumes above the chest wall resting volume of 55% of vital capacity in an upright dry condition and 15% of vital capacity in head-out immersion (Ferrigno et al., 1986). Thus it may be argued that breath-holding with large lung volumes in fact impairs venous return, leading to a reduction in stroke volume. Breath-holding after maximal

inspiration has been shown by sonography to cause an enlargement of the inferior vena cava, which, it is suggested, reflects impediment of blood flow into the chest (Ferrigno et al., 1986). Similarly, a decrease in venous return during breath-holding was implied following observations of increased limb volume as determined by leg circumference (Folinsbee, 1974). The change in limb volume was greater at lung volumes approaching vital capacity than at smaller volumes. There is evidence that breath-holding at small lung volumes (15% of vital capacity) may fail to increase venous return to the heart (Agostini and Rahn, 1960; Ferrigno et al., 1986). Juxtathoracic venous collapse is believed to account for such an occurrence, in the face of sufficiently low intrathoracic pressures. These data do not, therefore, support the theory that apneic bradycardia is a result of an increased venous return, and indicate that much remains to be clarified.

The bulk of the evidence suggests, however, that while an explanation for the effects of varying lung volumes upon apneic heart rates may not lie solely in haemodynamic changes depending on the breath-hold condition, their influences cannot be entirely discounted. In particular, the unusual circulatory state which exists during immersion in water (Lin, 1984) may help explain the potentiation of the bradycardial response which reportedly occurs with immersion compared to on land (Olsen et al., 1962; Song et al., 1969; Strømme et al., 1970; Bergman et al., 1972; Speck and Bruce, 1978; Gooden et al., 1978; Magel et al., 1982). Immersion in water produces a graded hydrostatic pressure which is exerted over the whole of the of the chest, resulting in a shift of the pressure-volume curve of the lungs and thorax (Jarrett, 1965; Craig and Dvorak, 1975). Despite an increase in

intrathoracic pressure, the effect of hydrostatic pressure during upright immersion is to create a negative pressure differential between the abdominal and thoracic cavities (Lin, 1988). A cephalad shift in blood volume amounting to approximately 700ml occurs (Burki, 1976; Lin, 1988). Thus during apneic immersion venous pressure is raised sufficiently to overcome an increase in intrathoracic pressure (Craig, 1965; Folinsbee, 1974; Lin, 1988.)

Chestward pooling of the blood is a contributory factor to the reduction in vital capacity evident upon immersion in water (Miles, 1966; Craig and Dvorak, 1975; Burki, 1976; Buono, 1983; Lin, 1988). Chestward displacement of the diaphragm (Lange et al., 1974; Lin, 1988) and the opposition to inspiratory effort caused by hydrostatic pressure (Jarrett, 1965; Miles, 1966; Burki, 1976; Buono, 1983) are additional related factors. The relative importance of these factors as a cause for the decrease in vital capacity during immersion is still under debate. Estimates of the percentage of the reduction due to blood redistribution have varied between 33% (Agostini et al., 1966) and 100% (Dahlback, 1975).

Conversely, the adaptive processes resulting from the continual exposure to breath-holding in habitual divers is manifest as an increase in static lung volumes (Carey et al., 1956; Song et al., 1965; Schaefer, 1965; Crosbie et al., 1979; Shiraki et al., 1985). Compared to control groups, significantly greater vital capacities have been reported for the Ama of Korea (Song et al., 1963), male Tsushima breath-hold divers (Shiraki et al., 1985), submarine crews training for escape procedures (Carey et al., 1956; Schaefer, 1965), and commercial

divers (Crosbie et al., 1979). The reason for this change has been the subject of some disagreement. It has been attributed on the one hand to a corresponding greater inspiratory capacity (Song et al., 1963), as a result of regular subjection to a state of negative pressure breathing while at the surface, and on the other hand, to a greater expiratory reserve volume (Shiraki et al., 1985), although this research group was unable to explain the reason for this adaptation.

Large lung volumes have been indirectly associated with the potentiation of the bradycardial response evident in regular participants of underwater apneic activities (Oldridge et al., 1978). In this instance a greater degree of bradycardia observed in synchronised swimmers compared to speed and recreational swimmers was ascribed to their longer breath-holding ability consequent to changes in lung volume. Scott and Candler (in press) however, indicated that lung volume was not a major criterion of breath-hold duration. In contrast to the majority of studies which have investigated the effect of held or inspired lung volume upon heart rates, these authors considered the relationship between apneic bradycardia and vital capacity, expressed relative to body surface area. Lower heart rates were noted in those with larger lung volumes, prompting the conclusion that relative vital capacity was a determinant of the magnitude of bradycardia during apnea.

Depth and Hydrostatic Pressure

Unless carried out to extremes, breath-hold diving involves only moderate pressure, which nonetheless elicits pronounced physiological changes (Lin, 1988). Significant alterations of function can also result from small local differences in pressure (Lanphier and Camporesi,

1982). Furthermore, it is essential to recognise that while gravity-induced responses in the cardiovascular system are balanced by equal external pressure during immersion, gravity-related forces within the confines of the thorax are not cancelled (Lanphier and Camporesi, 1982). Thus a consideration of hydrostatic pressure is inseparable from any investigation of the effects of immersion upon apneic bradycardia. A major criticism to be levelled at a large part of the research to date is that in many instances it is the face only that makes contact with the water, whereupon the results are applied to breath-hold diving in general.

The findings of different investigators with regard to the effects of whole body immersion at varying depths frequently appear to be in conflict. Some authors have observed apneic bradycardia to be independent of depth (Craig, 1963; Moore et al., 1972; Takamoto et al., 1987). Although a bradycardia was evident to a depth of 27m, Craig found that the response did not occur during simulated conditions in a hyperbaric chamber. Lanphier and Camporesi (1982) point out, however, that no form of "pressure breathing" on land can fully simulate immersion. For example, elevation of the rib cage caused by the buoyant force of the air-filled lungs, together with the cancellation of the weight of the arms and shoulder girdle normally supported by the thoracic wall, produce altered and difficult to determine respiratory pressures during immersion (Lanphier and Camporesi, 1982).

Other researchers have reported that no significant difference was evident between whole body immersion and facial immersion alone (Strømme et al., 1970; Moore et al., 1972), whereas Asmussen and Kristiansson

(1968) stated that facial immersion bradycardia was an "inconstant and fleeting" response, with a more persistent response evident during whole body immersion.

The importance of immersion of the head during whole body immersion, as opposed to head-out immersion only, is equally equivocal. An early study suggested that graded immersion in water of various depths did effect the heart rate response (Harding et al., 1965). The tachycardia which accompanied breath-holding on land progressively diminished with increasing depth, until conversion to bradycardia occurred with the water approximately at the level of the xiphisternal joint. An increased volume of blood in the thorax as a result of a graded hydrostatic effect over the lower part of the body was considered to account for this observation. In a similar study, various levels of immersion had no effect upon the heart rate response, as long as the nose was out of the water (Song et al., 1969). A greater bradycardial response seen with the head submerged was attributed to a neural reflex initiated by temperature-sensitive elements in the face. Where immersion of the face after torso immersion had no effect on the apneic bradycardia (Folinsbee, 1974), it was suggested that adaptation of facial cold receptors occurred during torso immersion. Thus immersion of the head is seen as contributing to the bradycardial response only in so far as facial cold receptors are stimulated. With the exception of pressure-related changes within the thoracic cavity, gravity effects on body fluids are nullified during submersion, so that the depth of submerged parts relative to the rest of the body is of no importance (Lanphier and Camporesi, 1982).

Findings which imply an effect on apneic heart rates related to changes in depth (Scholander et al., 1962; Hong et al., 1967) are contradictory in nature and fail to explain the possible causes. Scholander and co-workers found the degree of bradycardia during active dives at 3-4 fathoms (approximately 5.5 - 7.3m) to be greater than surface breath-holding, whereas Hong et al. found the opposite. Bradycardia was less during an active dive to 5m than during either breath-holding in water or an apneic surface swim.

Many of the variations described above may be explicable in terms of factors associated with the modification of apneic heart rates. Scholander et al. (1962) recorded much higher pre-apneic heart rates for their active dives than for surface breath-holding, the implications of which have been discussed previously. Hong and associates, on the other hand, recorded similar heart rates prior to both the surface swim and the 5m dive. The effects of temperature must also be considered: the apparently contradictory reports of Song and associates and Folinsbee become somewhat clearer when the 6°C difference in water temperatures - 25°C and 31°C respectively - are noted. Once again, therefore, it must be concluded that since the final apneic heart rate is determined by a number of variables, the alteration of one or a combination of these may produce quite different results.

Position

Much of the variation evident in reported comparisons of diverse breath-holding conditions both between and within studies may be due to differences in the position of the body. The role of body position in influencing heart rate, a factor unaccounted for in many studies, may be

of particular relevance to comparisons between land and underwater environments, since gravity-related changes in the cardiovascular system that occur on land may not apply to the latter condition. Similarly, although blood and body fluid distribution underwater are similar for any position (Lanphier and Camporesi, 1982), pressures within the thoracic cavity as affected by movement of the abdomen and diaphragm (Craig and Cummings, 1958; Agostini and Rahn, 1960), may in themselves influence central blood volume, and therefore heart rate responses. This may be of particular relevance where physical activity is incorporated, as in the case of early studies where direct comparisons were made between active and passive (assisted) dives (Scholander et al., 1962; Craig, 1963; Sasamoto, 1965), and where different changes in body position were likely during the course of a breath-hold.

Apneic bradycardia has been reported over a range of body positions. A large number of studies have focussed upon the prone (Olsen et al., 1962; Bove et al., 1968; Paulev, 1968; Strømme et al., 1970; Openshaw and Woodroof, 1978; Speck and Bruce, 1978) and seated (Kawakami et al., 1967; Craig and Medd, 1968; Gross et al., 1976; Gooden et al., 1978; Arnold, 1985; Sterba and Lundgren, 1985) positions under varying conditions. Magel et al. (1982) observed that while the pattern of heart rate response to apneic facial immersion was similar at rest in the prone and seated positions, the magnitude of the bradycardia was greater in the former. Other investigators have found apneic bradycardia while the body was supine (Paulev, 1968; Paulev and Hansen, 1972), upright (Harding et al., 1965; Moore et al., 1972; Sterba and Lundgren, 1985), or moved throughout a consequent range of

attitudes during synchronised swimming (Gemma and Wells, 1987; Takamoto et al., 1987).

Studies which have specifically examined the effects of altered position upon apneic heart rates have produced contrary results. Although an early study revealed no difference between the degree of heart rate slowing in the seated and standing positions (Olsen et al., 1962), Craig (1963) postulated that if venous return was a critical factor as an indirect determinant of heart rate during apnea, then position change should alter the response. Whereas Song et al. (1969) stated that apneic bradycardia on land was independent of posture for the sitting, supine, prone and head-down positions studied, Craig (1963) reported that on land, the tachycardia seen during apnea in the sitting position changed to bradycardia when the subject was tilted head-down in the supine position. This was attributed to increased venous pressure in the latter condition. A study of heart volume in relation to body posture indicated that a significant increase in heart volume occurred in the supine compared to standing position as a result of gravity-related changes in central blood volume (Lange et al., 1974). This was accompanied by changes in the configuration of the heart and in the position of the diaphragm, factors which may indirectly influence heart rate. Differences in venous pressure as influenced by posture effects upon central blood volume have been used to explain the greater bradycardia evident in the prone compared to seated position (Craig, 1965; Magel et al., 1982). It was suggested that a more prompt response by the venous system in overcoming increased intrapleural pressure occurred in the prone position (Craig, 1965). Furthermore,

Craig suggested that this position on land was comparable to the standing position during water immersion.

A horizontal position on land may produce effects comparable to those of immersion in any position. Where blood and body fluid distribution underwater is detectably different from that of dry recumbency, the difference must usually be attributed to respiratory gas pressure effects in the thorax (Lanphier and Camporesi, 1982). These authors point out that increased pressure during immersion is transmitted equally over the immersed parts, thus negating the importance of position or depth of immersed parts relative to the rest of the body. However, while recognising the effect of immersion is to alter the influence of gravity upon the body, Lundgren and Pasche (1984) point out that this change is itself influenced by body posture underwater. This is largely the result of changes in the direction of the graded hydrostatic pressure exerted over the air-filled, and compressible, lungs. According to Miles (1966), the pressure gradient in water is so much steeper than in air that immersion of the upright chest is sufficient to elicit respiratory and haemodynamic changes.

Breath-holding

The termination of apnea can be described as a response to "the development of a net ventilatory response too strong to be further resisted by voluntary effort" (Mithoefer, 1965). The course of breath-holding is characterised by distinctive phases, as described by Lin (1982):

1. Voluntary inhibition of respiratory muscular activity with a stable intrathoracic pressure which is slightly above the ambient.
2. Involuntary inspiratory muscular activity with the glottis closed.
3. Voluntary opening of the airway.

That point at which the glottis opens, ending breath-hold, is termed the "conventional breaking point", the "maximum breaking point", or simply the "breaking point" (Lin, 1982). It has been concluded that rigid concepts of chemical and mechanical thresholds are inadequate for interpreting the conventional break point (Godfrey and Campbell, 1968). Rather, psychological factors are recognised as the major factors determining the cessation of apnea (Lin et al., 1974; Lin, 1982; Hentsch and Ulmer, 1984; Hickey and Lundgren, 1984). Since prolongation of breath-hold duration is influenced by one's ability to overcome discomfort due to hypoxia, hypercapnia, acidosis and unpleasant respiratory sensations (Lin, 1982), there is considerable variation in the duration of apnea both within and between individuals (Lin et al., 1974; Lin, 1982; Hayward et al., 1984).

In contrast, the point at which voluntary inhibition of the ventilatory muscles is no longer tenable, and involuntary ventilatory activity (IVA) is observed, occur within a narrow range of arterial partial pressures of carbon dioxide (PCO_2) (Lin et al., 1982). The onset of IVA, termed the "physiological break point" is believed to relieve the urge to resume breathing (Lin et al., 1974). This phase is characterised by cyclic sub-atmospheric pressure in the thoracic cavity, such that mean intrathoracic pressure is continuously lowered during the latter phase of breath-holding (Lin et al., 1974). Since this process is beneficial

to venous return, and therefore cardiac output (Lin et al., 1974), it may have important implications for the heart rate response during the latter portion of a breath-hold, particularly if considered in conjunction with a progressive elevation in mean arterial pressure as breath-hold duration increases (Lin, 1982).

Among the chemical factors controlling respiration, the PCO_2 appears to be the strongest, with comparatively little stimulus being derived from the partial pressure of oxygen (PO_2) (Craig, 1961; Hickey and Lundgren, 1984). Although normal lung ventilation is regulated to keep alveolar PO_2 at about 100mmHg, and PCO_2 at approximately 40mmHg, breath-hold may force the latter to exceed 50mmHg (Hickey & Lundgren, 1984; McArdle et al., 1986). Whereas alveolar PO_2 decreases progressively over the duration of breath-hold (Craig, 1961; Hong et al., 1970; Lin, 1988) the carbon dioxide transfer from the blood into the lung is greatest during the first 20s of breath-hold, after which it proceeds at a much slower rate (Lin et al., 1974). As the PCO_2 in the lungs and blood become similar, alveolar PCO_2 may plateau (Hong et al., 1970; Lin, 1988) and may even pass from the lungs back into the blood (Craig, 1961; Hickey and Lundgren, 1984; Lin, 1988). This is brought about by the carbon dioxide concentrating effect of lung shrinkage due to the continuous transfer of oxygen into the blood, so that the PCO_2 of the lung eventually exceeds that of the blood. Hyperventilation therefore plays an important role in prolonging breath-hold times: body stores of carbon dioxide will be decreased as a function of time and alveolar ventilation (Hickey and Lundgren, 1984). Since haemoglobin is almost fully saturated during normal breathing, oxygen stores are not increased by hyperventilation, but for a given lung volume the duration of apnea

can be increased before critical PCO_2 levels are reached (Astrand and Rodahl, 1977; Hickey and Lundgren, 1984; McArdle et al., 1986).

Oxygen availability is an important aspect of breath-holding capacity. Breath-hold duration has been shown to be correlated to the reciprocal of $\dot{V}O_2$ (Craig and Cummings, 1958; Lin et al., 1974; Lin, 1982). The rate at which PO_2 decreases is much more rapid during exercise than at rest (Craig, 1961), consequently breath-hold times may be shorter (Craig and Cummings, 1958). However, the effect of exercise on breath-hold time may be more complex than an inverse relation to exercise intensity (Craig and Cummings, 1958) or $\dot{V}O_2$ (Lin, 1982). When exercise and apnea begin simultaneously, or when breathing is suspended during exercise, the result may be an improved tolerance to increased PCO_2 (Craig, 1961). During physical activity the PCO_2 at the breaking point of the breath-hold may be as high as 75mmHg, and the PO_2 may drop to 90mmHg (Astrand & Rodahl, 1977). Craig reported that subjectively the conventional breaking point was qualitatively the same during exercise and rest, but its development was different. While subjects were able to resist the urge to breathe for some time at rest, the conventional break point occurred very suddenly during exercise. Furthermore, distractions due to physical movement and conscious effort in performing a physical task may prolong apnea (Lin, 1982).

In addition to PCO_2 , pH and PO_2 , lung volume is an important element in orchestrating the onset of the breaking point (Hickey and Lundgren, 1984). At a given $\dot{V}O_2$, breath-holding time is proportional to the initial lung volume (Lin, 1982; Hickey and Lundgren, 1984). As apnea progresses the disproportionate removal of oxygen from the lungs

compared to carbon dioxide addition results in lung volume shrinkage, which may enhance respiratory drive via the Hering-Breuer deflation reflex (Lin, 1982; Hickey and Lundgren, 1984).

Furthermore, shrinkage in lung volume over the duration of a breath-hold diminishes tolerance to hypercapnia and hypoxia (Lin, 1982); large initial lung volume, in addition to movement of the respiratory muscles, may increase the tolerance for elevated PCO_2 (Mithoefer, 1965; Hickey and Lundgren, 1984). According to Lin (1982), no straight forward prediction explains the relationship between lung volume and breath-hold duration, particularly during underwater diving where the effects of elevated PO_2 due to compression of the thoracic cavity are opposed by the increased shrinkage of lung volume due to compression, and the increased oxygen demand due to muscular activity. While it is evident that inspiratory excitation increases with breath-holding time, it is as yet unclear as to whether shrinking lung volume or the cessation of breathing movement constitutes the source of respiratory distress (Lin, 1982). Interruptions of breath-holding with a single breath without improvement of alveolar gas compositions have been shown to delay breaking point even though large fluctuations in intrathoracic pressure persisted (Godfrey and Campbell, 1958; Lin, 1982). Similarly, an examination of the trainability of underwater breath-hold time revealed that significant increases in duration were accompanied by prologations of both the period without respiratory movements, and that period between the onset of IVA and the conventional breaking point (Hentsch and Ulmer, 1984).

A greater breath-holding capacity observed in submarine escape instructors in response to regular underwater training has been ascribed to increases in lung volumes, which in turn resulted in an improved tolerance to elevated carbon dioxide and lowered oxygen tensions (Schaefer, 1965). A high tolerance to carbon dioxide, as developed by the divers, appeared to be associated with a general damping of the autonomic nervous system (Schaefer, 1965). A relationship between the adaptive changes described above, breath-hold duration and apneic heart rate responses has been implied, following observations of a greater degree of bradycardia, together with a greater breath-holding capacity in synchronised swimmers compared to recreational and speed swimmers (Oldridge et al., 1978). However the quantitative effects of hypoxia and hypercapnia require further elucidation. It has been argued that the rapidity with which bradycardia appears during apnea indicates that chemoreceptor stimulation must play a minor role in producing bradycardia (Sasamoto, 1965; Kawakami et al., 1967; Lin et al., 1983).

Lin et al. (1983) reiterate that cessation of ventilatory activity per se is essential in the development of the response, and cite as support for this view the tachycardial response in humans while breathing hypoxic gas mixtures either with or without concomitant hypercapnia. During apnea, however, a profound contribution to the bradycardial response may be made by arterial chemoreceptor stimulation (Kawakami et al., 1967; Strømme et al., 1970; Lin et al., 1983). The exact nature of that contribution remains unclear. Kawakami and co-workers suggest that the maintenance of bradycardia is facilitated by hypercapnia, whereas others have dismissed PCO_2 levels as being of importance (Moore et al., 1972), or have found hypercapnia to attenuate bradycardia (Lin

et al., 1983). Analysing the contribution of apneic, hypoxic and hypercapnic effects, Lin and associates (1983) established that almost 60% of the total heart rate response during breath-holding could be attributed to hypoxia. Hyperoxia, in contrast, is reported to attenuate apneic bradycardia in humans (Moore et al., 1972).

In light of the confusion in the literature, it is clear, therefore, that the contribution of chemoreceptor stimulation to apneic bradycardia, together with the relationships between lung volume, breath-holding capacity and altered chemoreceptor sensitivity as these influence the extent of bradycardia, remain to be examined further.

Psychological State

If an integrative approach is to be adopted, the psychological component of man's make-up cannot be ignored. Evidence suggests that supra-medullary regions of the brain may influence basic cardiovascular control mechanisms (Ross and Steptoe, 1980). Studies in India of three Yogi who claimed to be able to slow their pulse by mental concentration revealed that no palpable radial pulse was evident during extreme Valsalva manoeuvre, despite a persistent rhythm displayed by electrocardiogram (Wenger et al., 1961). Observations of an anticipatory bradycardia at times prior to apnea (Wolf, 1978; Gemma and Wells, 1987) led Wolf to speculate that forebrain circuits were involved in the control of the "dive reflex". Gemma and Wells, along similar lines to previous authors (Asmussen and Kristiansson, 1968; Paulev and Hansen, 1972), ascribed this phenomenon to a conditioned response, which, it has been suggested, may be important when interpreting inconsistent results obtained from extensively repeated apnea and

immersion manoeuvres which are preceded by a distinct external stimulus (Paulev and Hansen, 1972). Differences in the incidence of heart rate reduction from one trial to another may, therefore, be the result of changes in physiological or psychological factors, or both.

Evidence exists to suggest that fear is associated with an accentuation of heart slowing (Craig, 1963; Wolf, 1978). Craig reported that of 13 subjects, the two exhibiting the most pronounced bradycardia were poor swimmers who were uncomfortable and nervous in an underwater environment. Such reports have, however, been criticised on the basis that the authors did not impose specific tasks or conditions, but relied on observation of natural variations in the emotional state of participants (Ross and Steptoe, 1980).

Following observations by Wolf (1978) that preoccupation, created by focussing the subject's attention on matters other than the experiment at hand, either attenuated or prevented bradycardia altogether, Ross and Steptoe (1980) studied the effects on apneic bradycardia of mental arithmetic compared to listening to prose. It had been suggested that cardiovascular adjustments in conditions of passive sensory intake may differ from those occurring during problem solving (Obrist, 1976). In contrast to an earlier study where subjects faced with an arithmetical problem all responded with tachycardia (Paulev, 1968), Ross and Steptoe found that although bradycardia was evident in air and water, it was much attenuated by mental arithmetic in both environments. External distraction by listening to prose, on the other hand, had no effect on cardiac response. In this instance mental work was said to have modified the neural reflex at a central level, either through

inhibiting vagal outflow, or by provoking antagonistic β adrenergic activation.

The bulk of the evidence suggests, therefore, that even where apparently identical testing conditions are maintained from one occasion to another, the heart rate response to apnea is susceptible to complex central modulation. It is possible that psychological influences on the heart rate are mediated by parasympathetic fibres, as well as through sympathetic responses (Obrist, 1976). In view of the tremendous intra- and inter-individual variability observed within some studies (Craig and Medd, 1968; Asmussen and Kristiansson, 1968; Wolf, 1978; Arnold, 1985), where it may be assumed that testing conditions were identical from one subject to the next, it may be concluded that psychological factors, together with methodological dissimilarities between studies, account for the lack of consistency in the manifestation of apneic bradycardia evident throughout the literature.

Conclusions

Conflicting evidence exists for almost all of the factors said to modify the apneic heart rate response in man. Much of the controversy may be due not only to the large variety of research protocols employed in the past, but in part to a lack of uniformity in the interpretation of the results obtained. In addition, part of the reason may lie in the tendency of many investigators to isolate particular elements considered to be of importance to the response, often without an apparent awareness of possible additional influential elements. While a comprehensive understanding of the phenomenon of apneic bradycardia necessitates the systematic examination of individual variables, cognisance must be taken

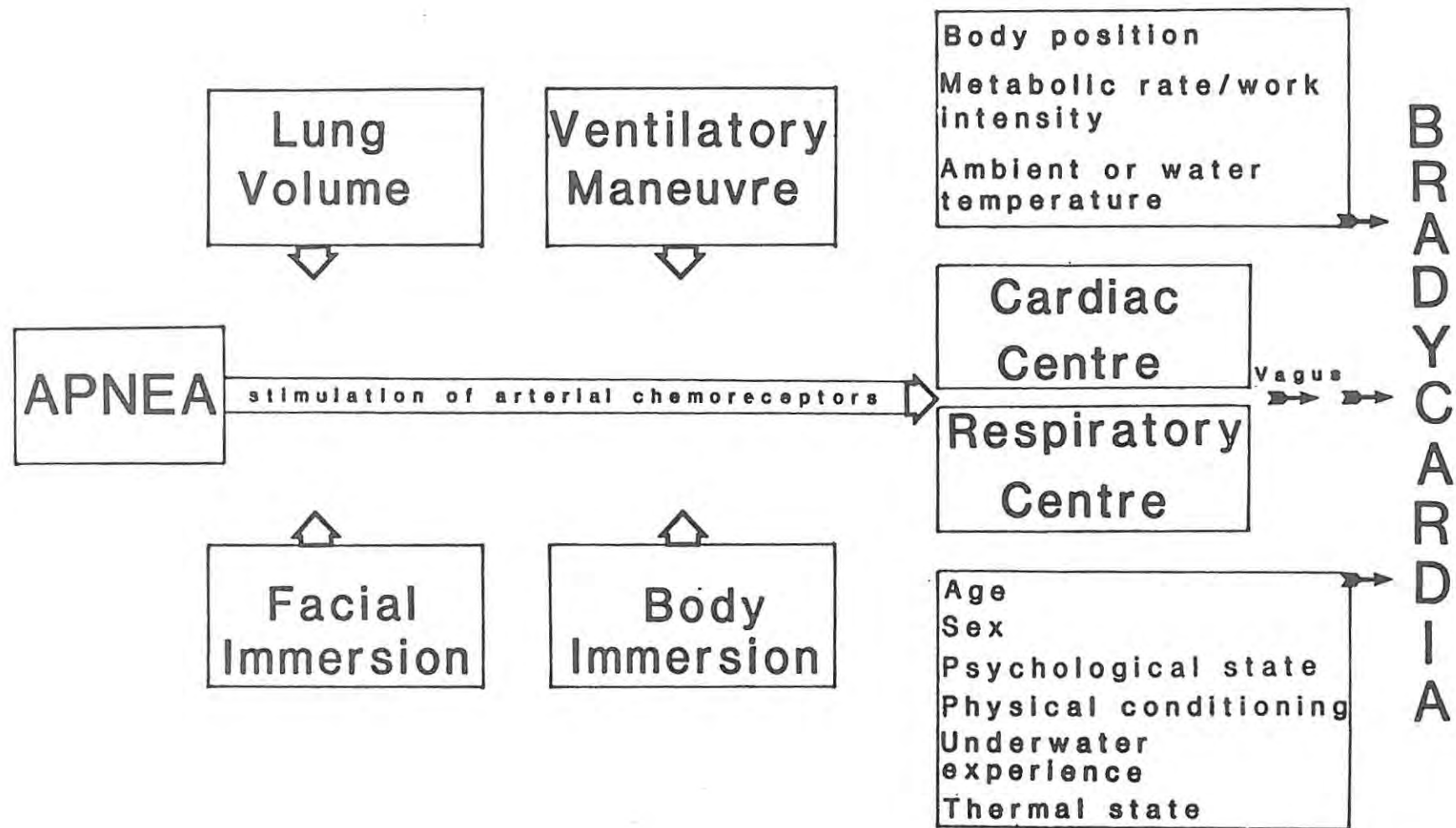


Figure 1: Schematic representation of those factors considered to influence to the bradycardial response (Adapted from Song et al., 1969).

of the entire network of causation if an integrative, holistic approach is to be adopted (see Figure 1).

MAXIMAL EXERCISE UNDERWATER

It is known that immersion in water produces prominent changes in cardiorespiratory functions. According to Greenleaf (1984), overall immersion responses in the upright position are the result of two major alterations in this condition: firstly, the increased mechanical pressure on connective tissue, which is proportional to depth, and secondly, the compression force on the abdomen, which forces the diaphragm upward and compresses cardiorespiratory structures in the thorax. Combination of these factors results in cephalad redistribution of the circulating blood, to the extent that central blood volume increases by approximately 700ml (Burki, 1976; Løllgen et al., 1980; Lanphier and Camporesi, 1982; Greenleaf, 1984; Lin, 1988). The increase in heart volume has been estimated at approximately 100ml (Lange et al., 1974), which may in turn exert additional pressure on respiratory structures (Greenleaf, 1984). Central vascular engorgement therefore has direct implications for both the cardiovascular and respiratory systems.

Reports of the precise nature of the modifications evident during immersion, however, frequently appear to be in conflict. For example, cardiac output had been described to decrease (Nadel, 1984), to remain unchanged (McArdle et al., 1976), or to increase (Denison et al., 1972; Løllgen et al., 1980; Lin 1984 and 1988). Conflicting results also exist with regard to the effects of immersion upon heart rate (Denison et al., 1972; McArdle et al., 1976; Dressendorfer et al., 1976; Coetsee

and Thiart, 1978; Lin, 1984 and 1988; Greenleaf, 1984), pulmonary ventilation (Magel and Faulkner, 1967; Cooper et al., 1976; McArdle et al., 1976; Dressendorfer et al., 1976; Coetsee and Thiart, 1978; Greenleaf, 1984), pulmonary compliance (Burki, 1976; Löllgen et al., 1980; Buono, 1983) and diffusing capacity (Löllgen et al., 1980; Lundgren and Pasche, 1984).

The effects of immersion upon maximal oxygen consumption are equally contradictory. In general, the $\dot{V}O_2$ max obtained during swimming tends to be lower than that obtained on a treadmill or cycle ergometer (Astrand and Rodahl, 1977; McArdle et al., 1986), although highly trained elite swimmers may attain similar values swimming or running (Magel and Faulkner, 1967; Holmér, 1978). Oxygen uptake during maximal fin swimming at the surface of the water and a depth of 3m was observed to be significantly lower than that obtained during treadmill running (Coetsee and Thiart, 1978). Compared to similar exercise on land, $\dot{V}O_2$ max was reportedly unchanged during cycle ergometry with the subject immersed to the level of the neck (Dressendorfer et al., 1976). In contrast, the $\dot{V}O_2$ of subjects performing graded leg exercise at loads from "light" to "forced maximal" was higher underwater than on land for all workloads, although $\dot{V}O_2$ max per se was not measured (Moore et al., 1970). However, dissimilarities in the experimental designs used, and at times a lack of uniformity between the land and underwater testing methods, tend to limit interpretation of the obtained results.

Denison et al. (1972) suggest that while altered cardiorespiratory responses to exertion will occur as a result of exposure of the body to new hydrostatic, viscous, inertial and thermal conditions, the effects

of those changes will vary with posture, workload, type of limb movement, mean intrathoracic pressure and water temperature. It can be concluded that since many of the variables measured are determined by a combination of processes, some of which counteract others, quite different results are thus possible in different subjects, or under somewhat different conditions (Lanphier and Camporesi, 1982). Hence, in a review of the available data on cardiac output during immersion in thermoneutral water (35°C), Greenleaf (1984) points out that where it was essentially unchanged in the supine position, compared with supine values in air, it increased by 25 to 35% in sitting subjects and by 63% in standing immersed subjects. Similarly, cardiac output was unchanged with sitting exercise, but tended to be 10% higher with supine exercise (Denison et al., 1972).

Although it has been stated that the immersion effect on the circulation diminishes as the water temperature decreases, (Lundgren and Pasche, 1984), the bulk of the evidence suggests that modifications in cardiorespiratory functions during immersion are likely to be most evident in upright (or sitting) subjects exercising maximally in cold water (Denison et al., 1972; Greenleaf, 1984). At heavier loads, increased resistance to motion of the chest wall and altered conditions for dissipation of body heat are likely to further influence responses (Denison et al., 1972). While much research has focussed upon immersion effects in 35°C water, generally accepted as representing the thermoneutral temperature for this environment (Craig, 1961; Hong, 1984; Lin, 1988), Lin (1984 and 1988) has pointed out that in a natural setting subjects engage in immersion and muscular activity in water below this temperature. Thus water temperature, as it affects

cardiorespiratory responses, is a critical consideration, particularly in relation to maximal effort.

As noted by Hong (1984), exercise increases heat production and thus should reduce the rate at which body temperature falls during immersion in water. However, body temperature may fall even when energy production is elevated above basal levels by 10 to 15 times, if the water temperature is sufficiently low and the insulation is inadequate (Nadel, 1984; Lin, 1988). For subjects exercising continuously at a load that doubled $\dot{V}O_2$, thermoneutral temperature dropped to 32°C (Craig, 1971). Continuous work that tripled $\dot{V}O_2$ lowered this value still further to 26°C. It should be remembered that increased muscle blood flow during activity results in considerably lower insulative capacity of muscle than during rest in water; hence the individual thermal comfort zone is closely correlated to subcutaneous fat thickness (Nadel, 1984; Hong, 1984). Although discomfort was typically experienced upon immersion and during light work at cooler temperatures (18 to 23°C) (Dressendorfer et al., 1976; McArdle et al., 1976), for maximal work subjects preferred exercising in cool (25°C) water (Dressendorfer et al., 1976). In warmer water (33 to 35°C) thermal sensations were reversed, with the result that subjects became heat stressed during maximal work. McArdle and associates (1976) observed fatter subjects to sweat at the face during heavy work in 33 and 35°C water.

At the same time, it is possible that swimmers do not achieve the same maximal aerobic power in colder water that is possible in warmer water (Nadel, 1984). Nadel reported that at temperatures of 18 and 26°C subjects were only able to reach 85% and 92% of their maximal aerobic

power in 33°C water, indicating that the extent of cold stress is an important determinant of the $\dot{V}O_2$ max. Dressendorfer and co-workers, on the other hand, noted that cycling exercise during head-out immersion yielded $\dot{V}O_2$ max values that were unchanged in 25, 30 and 35°C water, although this was accompanied by significantly lower heart rates in 25 and 30°C water.

Lower heart rates have been found during submaximal exercise in cool water compared to warmer water (McArdle et al., 1976), or the same exercise in air (Moore et al., 1970). While Dressendorfer and co-workers assumed that $\dot{V}O_2$ max remained unaltered despite differences in cardiac output, brought about reductions in heart rate in conjunction with a maintained stroke volume, McArdle et al. (1976), using the carbon dioxide rebreathing method, found the cardiac output-oxygen consumption relationship to be similar during work in air and water at 18, 25 and 33°C. The prominent adjustment to the decrease in heart rate in cooler water, therefore, was an increase in the stroke volume, which was most pronounced in 18°C water. Lin (1988) has suggested that altered pre-load is responsible for the increased stroke volume during immersion.

Dressendorfer et al. (1976), based on the results of their comparison of subjects cycling in air and during head-out immersion, concluded that the reduction in $\dot{V}O_2$ max typically reported for swimming in comparison to running or cycling, should be related to inherent differences in the activities studied, rather than the effects of water immersion per se. It is apparent, however, that in addition to prominent circulatory adjustments, which become more complex in cold water, the effects of

immersion upon the mechanics of respiration may contribute to a lowered $\dot{V}O_2$ max in this environment. In addition to the effects of raised central blood volume upon static and dynamic lung volumes, there is an increased load on the respiratory muscles, such that resting respiratory work during head-out immersion may be elevated by about 60% over that at rest in air (Lanphier and Camporesi, 1982; Lin, 1988). A lower pulmonary ventilation has been reported (Magel and Faulkner, 1967; Dressendorfer et al., 1976; Coetsee and Thiart, 1978; Holmér, 1978), although contrasting findings do exist where graded leg exercise on land and underwater at a depth of 1.22m yielded similar minute volumes (Moore et al., 1970). It has been proposed, however, that the opposing effects of hydrostatic pressure and increased intrathoracic blood volume upon gas exchange may result in sufficient oxygenation of arterial blood underwater. Furthermore, the interaction of a lower minute ventilation and a higher oxygen extraction may result in $\dot{V}O_2$ max values underwater that are equivalent to those obtained on land (Magel and Faulkner, 1967; Dressendorfer et al., 1976; Holmér, 1978, Lin, 1988).

It is clear, therefore, that despite some debate concerning the precise nature of the changes elicited by immersion, pronounced cardiorespiratory adjustments do occur. It is likely that different results on central haemodynamics and respiratory function are confounded by methodological factors such as water temperature or body position.

RATINGS OF PERCEIVED EXERTION (RPE)

Many neuromuscular and physiological parameters have been proposed as contributing to effort perception during exercise. It is evident that the perception of exertion is complex in nature, and rather than being

the outcome of a single primary cue, represents the synthesis of a variety of factors during different types of physical work (Pandolf, 1978). Since Borg's original concept focussing on the association of perceptual responses and heart rate as an index of exercise strain, more recent research has centred upon a two-factor model, as proposed by Ekblom and Goldberg (1971), in an attempt to assess the relative contributions of local versus central factors. An overall rating of perceived exertion involves integrating signals from the peripheral working muscles and joints, from the central circulatory and pulmonary functions and from the central nervous system (Birk and Birk, 1987). Gamberale (1985) points out that exertional ratings represent a subjective, distal reaction; the extent to which this distal reaction reflects the reaction within the individual organism is mediated by various influences depending upon the situation. As an estimation of effort expenditure, the 15-point RPE Scale (Borg, 1970) is known to be influenced by factors such as age, sex, physical training, mode of exercise, and intensity and duration of the activity (Sidney and Shephard, 1977; Pandolf, 1978; Robertson, 1982; Carton and Rhodes, 1985a and 1985b). An investigation of subjective reactions to apnea and apneic diving represents a unique challenge, in that many of the observed relationships between RPE and physiological parameters such as heart rate or ventilation would appear to be altered during breath-holding.

The RPE scale was designed to follow the heart rate response to increasing exercise intensity (Carton and Rhodes, 1985a). These authors state, however, that a causal relationship between these variables has never been implied; rather, they are indirectly related

through their common dependence upon physical strain. Clearly the postulated relationship between heart rate and RPE can be disturbed when exercise is performed under irregular conditions. A comparison can possibly be drawn between apneic heart rate responses and the manipulation of heart rate through the use of parasympathetic and sympathetic blocking agents (Davies and Sargeant, 1979). The RPE was unaltered at a given percentage of $\dot{V}O_2$ max, despite changes in heart rate.

Varied results have also been reported with regard to the relationship between RPE and $\dot{V}O_2$. However, a substantial number of investigators have suggested that factors such as pulmonary ventilation ($\dot{V}_e$) and respiratory resistance, which are more likely to be consciously monitored by the subject, may serve as prominent cues for rated perceived exertion (Pandolf, 1978; Mihevic, 1981; Robertson, 1982; Carton and Rhodes, 1985a). Although the influence of $\dot{V}_e$ is not applicable to the apneic condition, the perception of respiratory resistance appears to be related to sensations within the chest wall (Carton and Rhodes, 1985a), a factor which may be of relevance to the RPE during breath-holding. Sensory receptors located in the chest wall, lungs and airways respond to changes in lung volume and lung pressure, and respiratory tension (Robertson, 1982). In a recent study of apneic heart rates, Scott and Candler (in press) suggested that cardiopulmonary factors were of primary importance in determining RPE during breath-holding, particularly underwater. Studies of the effects of hypoxia, hyperoxia and hypercapnia upon RPE indicate that the metabolic intensity achieved during an exercise task may be of importance in determining the strength of cues relating to breathing

discomfort (Robertson, 1982). The point at which these sensory signals begin to be consciously monitored may be approximately 70% of $\dot{V}O_2$ max (Robertson, 1982). It must be noted, however, that such findings relate specifically to the relationship between $\dot{V}e$ and RPE as influenced by altered breathing mixtures; the extent to which hypoxia and hypercapnia influence RPE during breath-holding is speculative.

Clearly the perception of effort cannot be adequately explained in terms of physiological effort alone (Carton and Rhodes, 1985a). Morgan (1973) calculated that physiological variables were able to account for only 67% of the total variance in RPE: personality factors of a psychometric nature may further mediate perceived exertion. Personality variables such as extroversion and introversion exert a significant influence upon perceptual experience, while neurotics and depressives may lack the ability to accurately rate perceived exertion (Morgan, 1973). There is strong indication that perception is an active process (Rejeski, 1985). Factors such as personality, social learning and past experience thus form emotionally rooted differences that can alter the interpretation of sensory cues, such that similar peripheral physiological changes in two individuals do not necessarily result in identical percepts (Rejeski, 1985). This may be of particular importance to the underwater testing environment, where it has been shown that psychological stress, especially in less experienced divers, may significantly influence motor performance and perception (de Mojà et al., 1987; Griffiths et al., 1987).

CHAPTER 3

EXPERIMENTAL METHODS AND PROCEDURES

PILOT TESTING PHASE

Heart Rate Response to Exercise of Varying Intensities

The aim of this study was to examine the apneic heart rate response to various levels of exercise in an attempt to determine the relative intensities at which the subject would exercise during the main data collection on land and underwater.

Ten subjects participated in this phase of testing on land, and five of these were used to obtain apneic heart rate responses underwater. As an initial step the heart rate response to apneic exercise on land was examined, at 10% increments, from 20-100% of the maximal power output achieved on a cycle ergometer, as determined by multiplying the number of pedal revolutions by the resistive load (McArdle et al., 1986)

Power (W) = load (kg) x pedal revolutions (rpm)

Since the rate of pedalling on land remained fixed at 60 rpm throughout the test, it can be seen that X% of the maximum power output corresponded to X% of the maximum resistance load achieved. A starting point of 20% was necessary since in many cases 10% of the maximum resistance attained was less than 1kg, the lowest resistance load possible on the cycle ergometer.

Problems arose with regard to the underwater condition. Although subjects were requested to pedal at a fixed rate (60 rpm) in time to an

acoustical signal relayed via an underwater speaker, it soon became evident that they were unable to maintain the prescribed rate.

While the problem of the rate of pedalling was resolved (see pilot testing results) it meant that for the purposes of this phase of the pilot study an accurate calculation of power output underwater was not possible. This was confounded by the effects of the water upon the resistance load, and the additional resistance offered by the water while pedalling. Although it was recognised that this would play an important role in the calculation of power output, it remained a constant factor underwater. The intensities of exercise for this condition were therefore determined as a function of the maximum resistance load (20-100%) achieved during the underwater test. In this way the examination of heart rate responses to varying intensities of exercise remained standard on land and underwater. The order in which the various intensities of exercise were assigned to each subject was randomly selected.

Heart rate responses were recorded for both breath-hold and non-breath-hold conditions over identical periods of time for each intensity of exercise studied. These were obtained during one session each on land and underwater. The subject testing position, instructions delivered prior to each session and method of data collection are described under Session B: Land. Between each exercise bout the subjects were permitted sufficient time to recover fully and were instructed to continue with the next bout only when they felt ready. Heart rate was continuously monitored and only if it was within 10 beats of the initial 30s resting value (converted to beats per minute) was the

subject allowed to continue. In addition, subjects were requested to report the onset of any feeling of local or general fatigue, together with any sensations such as dizziness or light-headedness, in which case the data collection would be halted and continued at a later date. This was not found to be necessary either on land or underwater.

Heart rate analysis involved the digitization of R-R intervals for conversion into beats per minute (See Data Reduction.) The mean heart rate over the exercise duration (apnea and without apnea) was then used for statistical purposes. Statistical evaluation involved a series of two-way ANOVAs with repeated measures on both factors. Thus comparisons were made between heart rate responses to the various intensities of exercise with and without apnea, on land and underwater. Analysis in this way revealed, however, that apneic and non-apneic heart rates combined could result in a similar mean heart rate across the range of exercise intensities. For example:

	% P.O.	20	30	40	etc
Underwater:	Apnea	90	80	60	
	Without apnea	110	120	140	
	Mean:	100	100	100	

For this reason a one-way ANOVA with repeated measures was used to identify the existence of significant differences at different exercise intensities for each of the conditions. Where the existence of significant differences was indicated the data were analysed using a post-hoc Sheffé test to locate the position of those differences.

Pedal Rate Underwater

During the underwater test subjects were requested to pedal at a fixed rate, in time to an acoustical signal relayed via an underwater speaker. This was intended to match the rate of pedalling on land (60 rpm). As reported earlier, subjects were unable to maintain the prescribed rate, and reported experiencing difficulty in being able to hear the metronome signal relayed underwater. However, when pedal revolutions were recorded on a manual counter at one minute intervals for the duration of the underwater test it was found that a remarkably steady rate of pedalling was maintained, but at a lower rate than the prescribed 60 rpm. In order to investigate the subjects' ability to replicate this same pedal rate at a later stage, a related Student's t-test was applied to the recorded pedal revolutions on a test/retest basis following the readministration of an underwater $\dot{V}O_2$ max test to three subjects.

Maximal Oxygen Uptake on Land and Underwater

The purpose of this study was to evaluate maximal oxygen uptake ($\dot{V}O_2$ max) obtained using a direct continuous cycle ergometer test both on land and underwater. Test/retest reliability of the protocol was established for both conditions and an attempt was made to determine the degree to which data from the two different testing conditions were comparable. Finally, the feasibility of the administration of the test was examined in terms of $\dot{V}O_2$ max values, time to completion and subjective feelings of the subjects.

Ten healthy male subjects underwent a land test of $\dot{V}O_2$ max. Only eight however, were able to participate in the underwater tests, five of whom had specific experience in underwater activities such as spearfishing or

underwater hockey. While the remaining three were not experienced in underwater activities, they were competent swimmers and confident in a water environment.

The first phase involved the determination of $\dot{V}O_2$ max on land. Each subject reported for the laboratory tests dressed in shorts, t-shirt and running shoes at a time convenient to him. Details of the continuous cycle protocol used are described under Session A: Land. To evaluate the reproducibility of the $\dot{V}O_2$ max values obtained using this protocol, the test was readministered to three of the subjects within one week of the initial test. Although only three subjects were involved in the test/retest procedures, additional subjects were required to establish group one independent variable regressions ($\dot{V}O_2$ against time) (Ferguson, 1981) in either environment.

Following this, eight subjects participated in an underwater test of $\dot{V}O_2$ max, using exactly the same protocol as on land. Subjects wore only shorts and training shoes during underwater testing. Modifications to the cycle ergometer necessary to conduct the underwater test were carried out. (See Session C: Underwater). The test was readministered to three of the subjects along the same principles as on land.

Statistical evaluation of the results involved related Student's t-tests to establish reliability on a test/retest basis and to evaluate the land $\dot{V}O_2$ max against that obtained underwater.

Reduction of Heart Rate Data

Validation of the Hewlett-Packard ECG system was not deemed necessary, since this had been adequately performed in an earlier study (Candler 1986).

A related t-test was used to determine the validity of the digitization of R-R intervals (as described under Heart Rate Analysis) against manual measurement as methods of obtaining heart rate values. Following this, the R-R intervals of three ECG traces were digitized on a test/retest basis and a related t-test was used to evaluate the reliability of this method of data reduction.

PILOT TESTING RESULTS

Pedal Rate Underwater

Although subjects were unable to maintain the prescribed pedal rate of 60 rpm underwater, it was observed during pilot testing that a steady rate was nevertheless achieved. It should be noted at this point that the tester remained in the water for the duration of the test and was thus able to indicate to the subject whenever he began to alter his rate of pedalling. The mean pedal rates for three subjects evaluated on a test/retest basis over the duration of an underwater $\dot{V}O_2$ max test were 44.62 and 44.34 rpm. These were not significantly different at the 0.05 level. Thus it appeared that subjects were able to reproduce their chosen pedal rate.

Although the slightly fluctuating pedalling frequency underwater was not of consequence for the calculation of oxygen consumption (See Appendix

C), it was, however, important to consider the effect that this had upon the concept of progressively increasing work intensities over the duration of the $\dot{V}O_2$ max test. Since the effects of water upon the resistance load on the cycle ergometer, together with the additional resistance offered by the water to pedalling were, considered to be common factors influencing the power output of each subject, it was possible to examine the effects of fluctuating pedalling frequency along similar principles to those used on land. That is:

$$\text{Power output (W)} = \text{load (kg)} \times \text{pedal revolutions (rpm)}$$

When the pedal revolutions recorded during the readministration of the $\dot{V}O_2$ max test above were examined (Figure 2), it was observed that despite a drop in pedal rate over time, the increase in resistance load every two minutes did represent a progressively increasing workload. Thus, for the purposes of this study, the problem of pedal rate was satisfactorily resolved in the sense that, unless a drastic drop in pedal rate occurred, the final resistance load achieved by the subject underwater did represent the maximum work intensity attainable by that subject under those conditions. A comparison between land and underwater was therefore possible since heart rate responses could be examined in terms of exercise intensities relative to the maximum workload attained for a given set of conditions.

Heart Rate Response to Varying Work Intensities

Statistical analysis involving two-factor ANOVAs with repeated measures on both factors (Ferguson, 1981) revealed that significant differences did exist ($p < 0.05$) between the apneic and non-apneic condition for both the land and underwater tests. Further significant differences

existed in the apneic and non-apneic heart rate responses when those obtained on land were compared to those obtained underwater. In no case, however, was there a significant interaction between all factors; Thus the trends in heart rate response to apneic and non-apneic exercise, on land and underwater, were qualitatively the same. Figure 3a reveals that the expected relationship between heart rate and power output without apnea was reflected under apneic conditions. A similar result was evident underwater (Figure 3b).

The presence of significant differences in the heart rate response to varying intensities of exercise was also indicated for each condition. With regard to the underwater heart rate responses (apnea vs without apnea), however, separation of apneic and non-apneic conditions, on land and underwater, indicated the presence of significant differences in the mean heart rate response to varying intensities of exercise. On land in the apneic state the mean heart rates for 20 to 60% of the maximum power output were significantly lower than for 80 to 100%. Furthermore, mean heart rates at 20 and 30% were lower than at 70% of maximum power output, and the mean heart rate at 70% was significantly lower than at 100%. For the same condition underwater, significant differences existed between 20% and 80 to 100% of maximum power output. In the non-apneic state on land, the mean heart rate responses at 20 and 30% were significantly lower than at 50 to 100%; The heart rate at 40% was significantly lower than at 70 to 100% and the heart rates at 50 and 60% were significantly lower than at 90 to 100%. Underwater, the only significant differences existed between mean heart rates at 20% and 90 to 100%, and between 30% and 90% of maximal power output. In other

words, both on land and underwater, significant differences in heart rate responses occurred primarily between light and heavy workloads.

Determination of Relative Intensities of Work

Owing to the difficulties, discussed earlier, inherent in precisely determining power output underwater, it was decided not to use maximum power output as the criterion by which relative exercise intensities were determined. Rather, the exercise intensities to be used were calculated as percentages of the $\dot{V}O_2$ max values obtained on land and underwater. This was deemed to be more appropriate since exact values of $\dot{V}O_2$ could be calculated, and thus a 'situation-limited' functional maximum underwater could be obtained (See Pilot Test Results). Furthermore, since on land a direct relationship between power output and $\dot{V}O_2$ prior to the plateau reached at maximal levels is established (Astrand and Rodahl, 1977; McArdle et al., 1986), and since the pilot study revealed that a plateau in $\dot{V}O_2$ was not demonstrated in this case due to the nature of the test protocol used, the results of the heart rate analyses at varying intensities of power output on land could be applied. In order to standardise land and underwater conditions examined, and since no significant differences could be located between relative exercise intensities underwater, the exercise intensities chosen relative to $\dot{V}O_2$ max on land were also applied to the underwater condition.

In order to establish the resistance load on the cycle ergometer for a given percentage of $\dot{V}O_2$ max, the regression was used. Thus, in calculating $\dot{V}O_2$ from time, it was possible to arrive at an equation whereby the exact time from commencement of the test could be calculated

for a given percentage of $\dot{V}O_2$ max and the resistance load at that time determined.

When group regressions were calculated for pilot study land and underwater conditions, the correlation coefficient was highest for a one independent variable linear regression (Ferguson, 1981) in each case:

$$\begin{aligned}\text{Land: } \dot{V}O_2 &= 13.8 + 2.7 \text{ Time} \\ (r^2 &= 0.90)\end{aligned}$$

$$\begin{aligned}\text{Underwater: } \dot{V}O_2 &= 22.5 + 1.6 \text{ Time} \\ (r^2 &= 0.90)\end{aligned}$$

In relation to underwater $\dot{V}O_2$ max testing and the difficulties experienced therein (See Pilot Test Results), an examination of the validity of using only the last three gas collections in order to calculate a regression was made using data collected during the pilot study. For three subjects, whose data were considered representative of the pilot group as a whole, regressions calculated in this manner were evaluated against those calculated using all gas collections from the commencement of the test. An independent t-test revealed that no significant differences existed between the $\dot{V}O_2$ values calculated using both equations. Furthermore, the correlation coefficient for the two sets of values was 1.0 (thus predictive index = 1.0). It was therefore concluded that the use of the last three gas collections in determining a regression for the underwater condition was valid.

The test protocol used prescribed an increase in the resistance load every two minutes: this corresponded to a 1 kg (approximately 60 W) increase for the first two bouts (ie. until the sixth minute) followed by

0.5 kg (approximately 30 W) for every additional two minutes. However, calculation of each individual's resistance load for the percentages of $\dot{V}O_2$ max chosen revealed that where the overall test duration had been comparatively short for a few subjects, particularly underwater, the situation may have arisen where the same resistance load was prescribed for different percentages of $\dot{V}O_2$ max. For example, where 70% $\dot{V}O_2$ max was attained at 8 min 30s and 80% $\dot{V}O_2$ max at 9 min 45s, the resistance load in terms of the protocol used would be 4 kg in both cases (since 8 to 10 min = 4 kg load). For this reason it was decided that where the time for X% $\dot{V}O_2$ max fell before the mid-point of a two-minute bout, the load prescribed for that bout would be used; where it fell after the mid-point, half of the subsequently added weight would be added to the prescribed load for that bout. Thus:

<u>Time (min)</u>	<u>Load (kg)</u>	
0-1	1	
1-2	1.5	Half of next load

2-3	2	
-	-	
6-7	4	
7-8	4.25	Half of next load

8-9	4.5	

Choice of Relative Work Intensities

An examination of heart rate responses to exercise of varying intensities indicated that significant differences existed in the heart

rate responses to low and high exercise intensities on land. However, calculation of regression equations for the underwater condition revealed a generally elevated "resting" $\dot{V}O_2$. This may have been the result of a combination of some, or all of the following factors. The effects of low water temperature on metabolic rate were likely to have been of importance, especially where this occurred in conjunction with a low body mass (Hong, 1984). The effects of anxiety and anticipation upon resting physiologic state cannot be discounted, since these would serve to increase energy consumption prior to data collection. Similarly, the act of getting into the water and being secured to the ergometer prior to commencing the test would result in an elevation of so-called "resting" physiological values. Taking the above into consideration the choice of low relative workloads for data collection was decided against. Results of the pilot study indicated that there was a numeric difference in the heart rate response to exercise at 70% and 90% of the maximum resistance load achieved, both on land and underwater (see Figures 4 and 5). Intensities such as these would produce pronounced tachycardia under normal ventilatory conditions on land, and thus facilitate the investigation of the suppressive effects of apnea upon heart rate. It was decided, therefore, that for the purposes of data collection, where the exercise intensity would be determined more accurately as a percentage of $\dot{V}O_2$ max, heart rate responses to exercise at 50%, 70% and 90% of $\dot{V}O_2$ max would be studied. This represented "moderate" to "very heavy" ranges of workload.

Reduction of Heart Rate Data

No significant differences were revealed between digitization and manual measurement of traces obtained, nor were any significant differences identified following redigitization of three traces. This method of data collection was therefore accepted as valid and reliable.

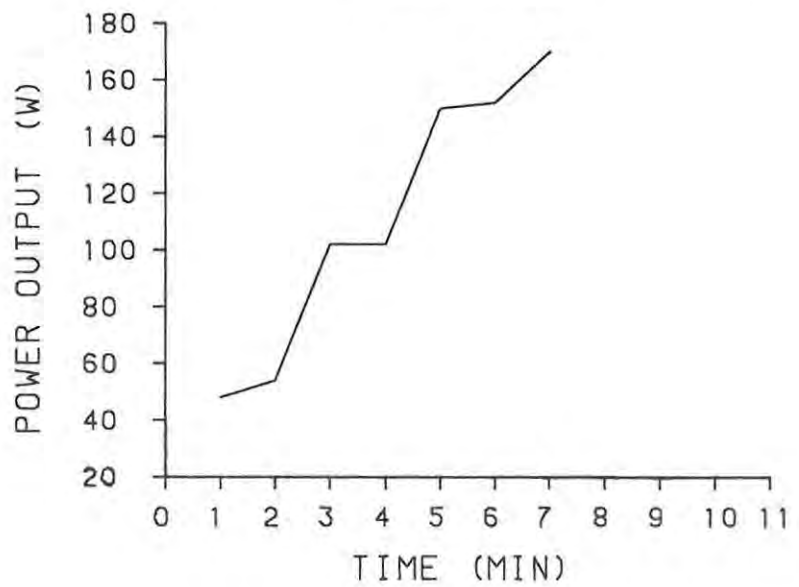
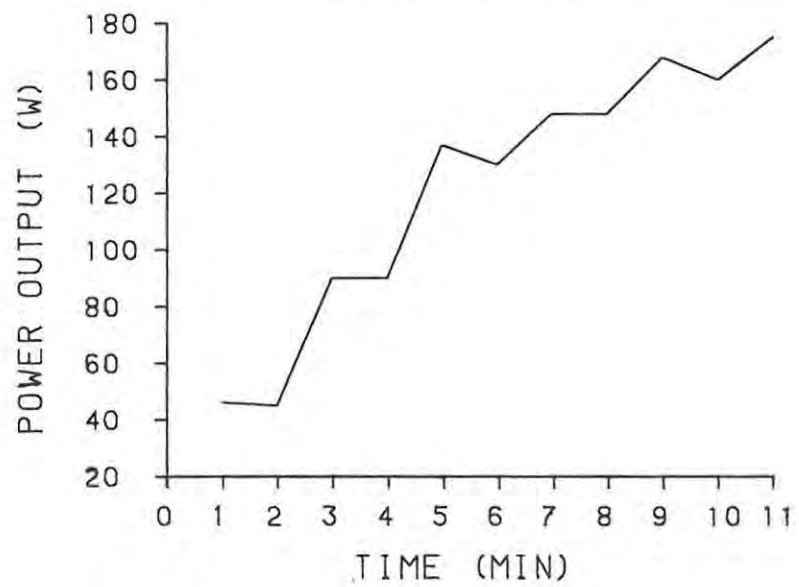
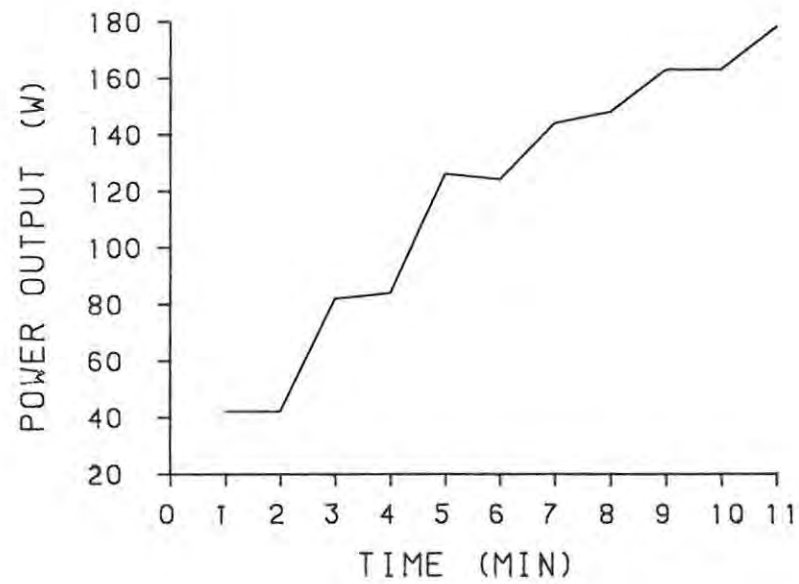


Figure 2: Progressively increasing power output recorded for three pilot subjects over the duration of an underwater $\dot{V}O_2$ max test.

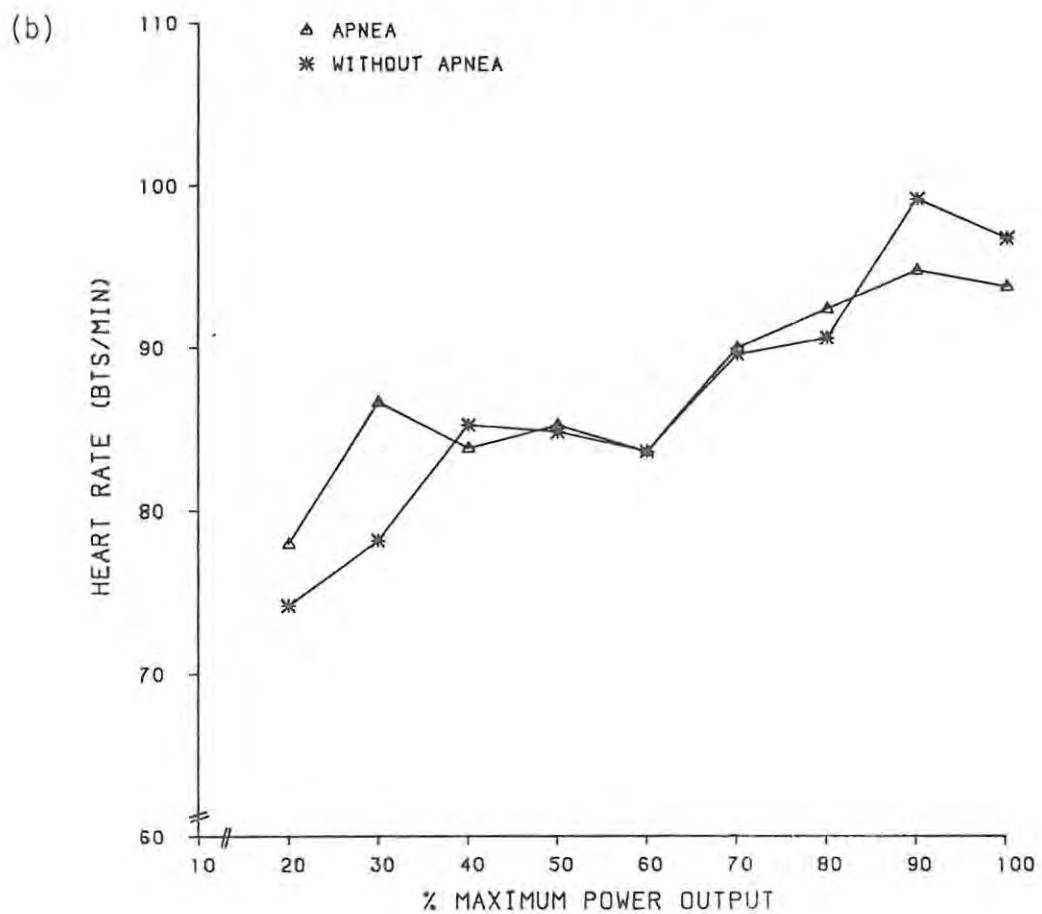
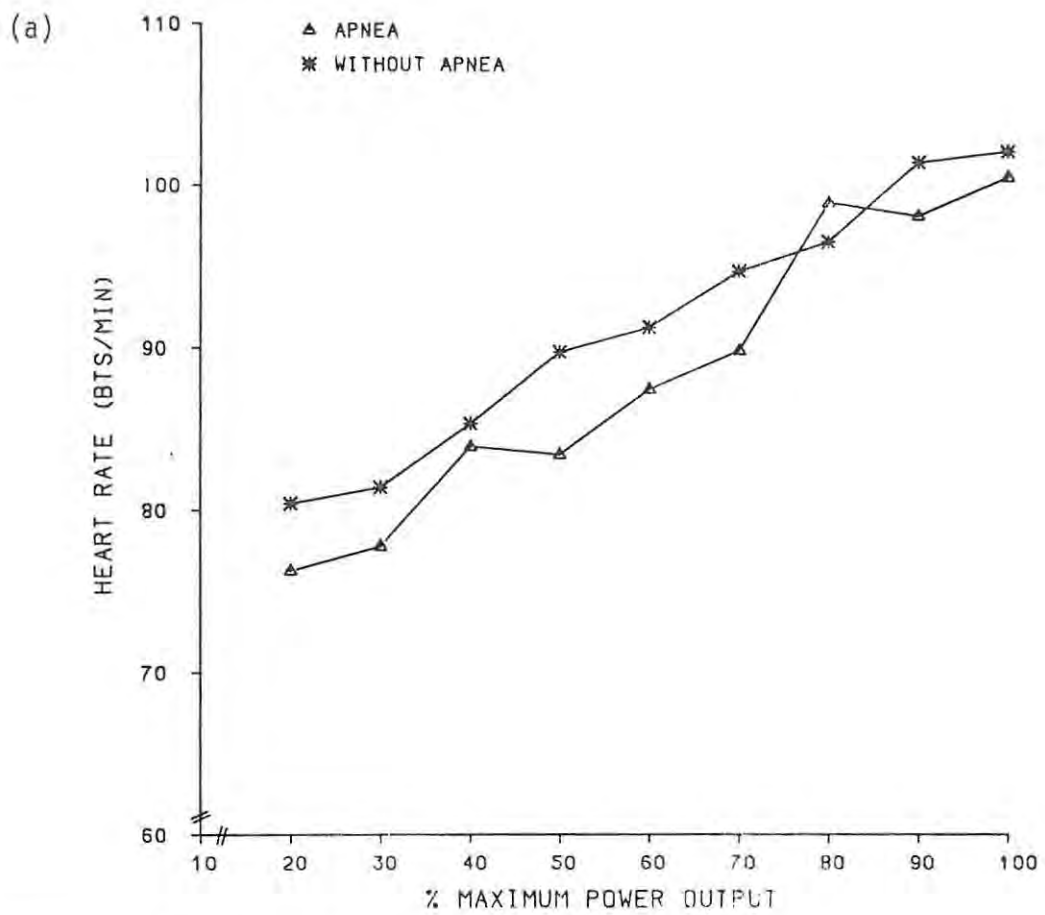


Figure 3: Apneic and non-apneic heart rate responses to exercise on land (a) and underwater (b) at 10% increments of maximal power output.

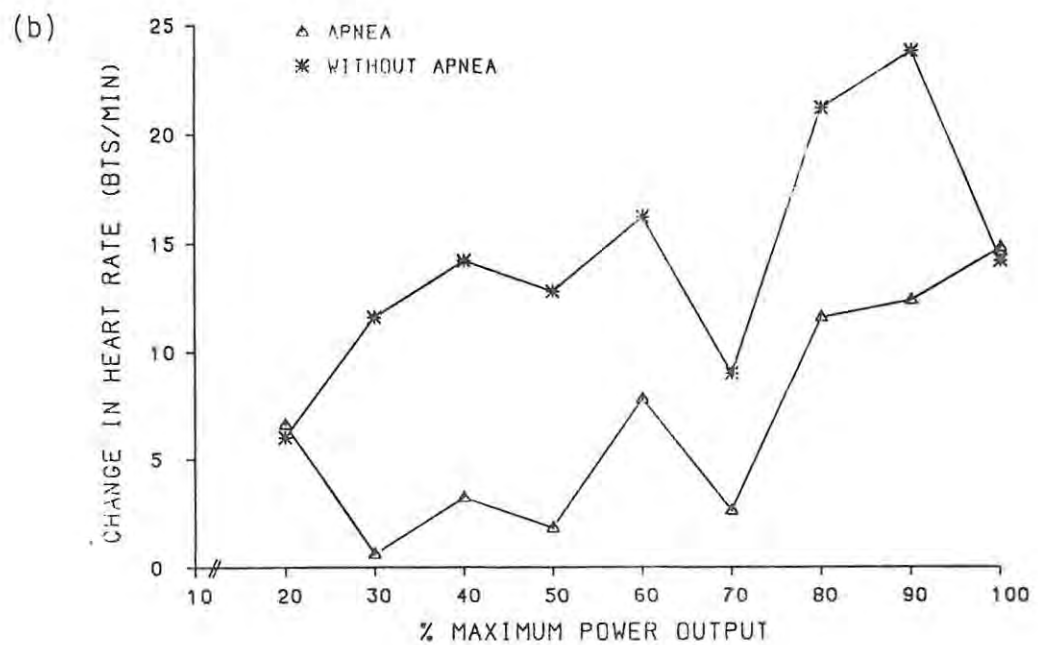
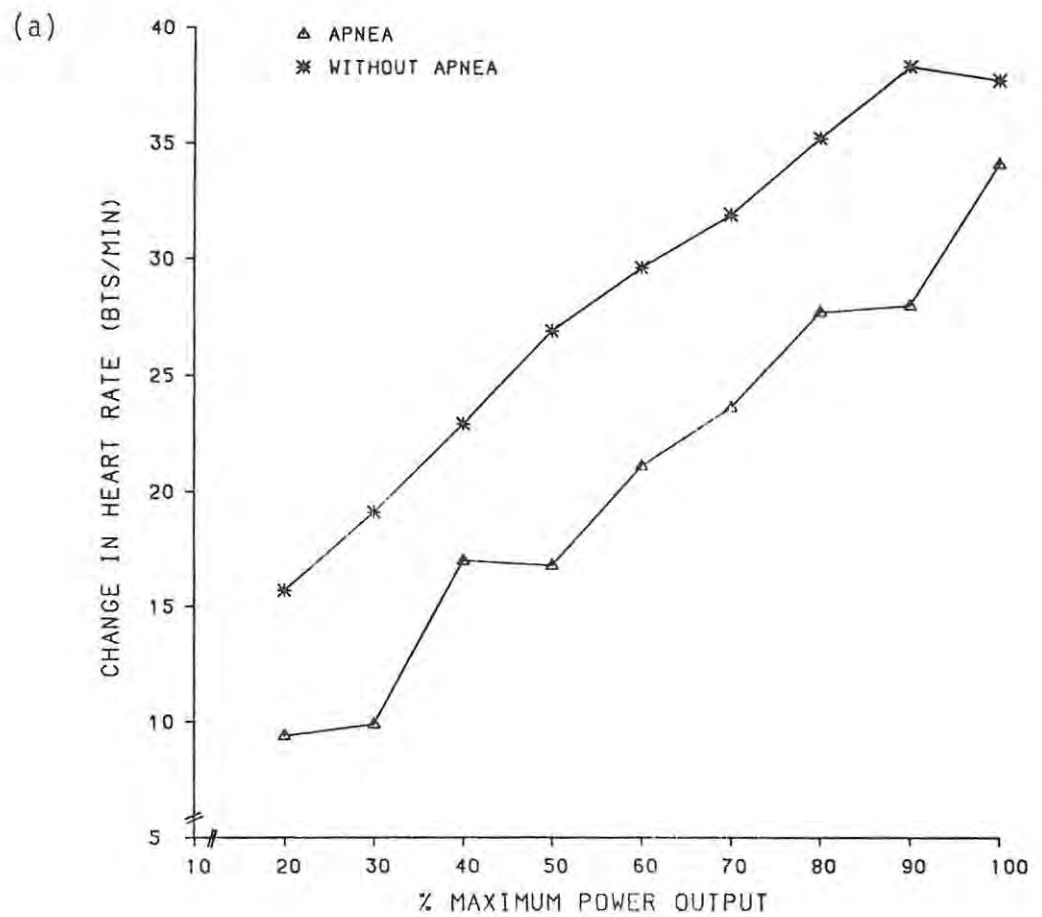


Figure 4: The change in heart rate on land (a) and underwater (b) from immediate pre-exercise levels to the mean over the exercise duration at 10% increments of maximal power output.

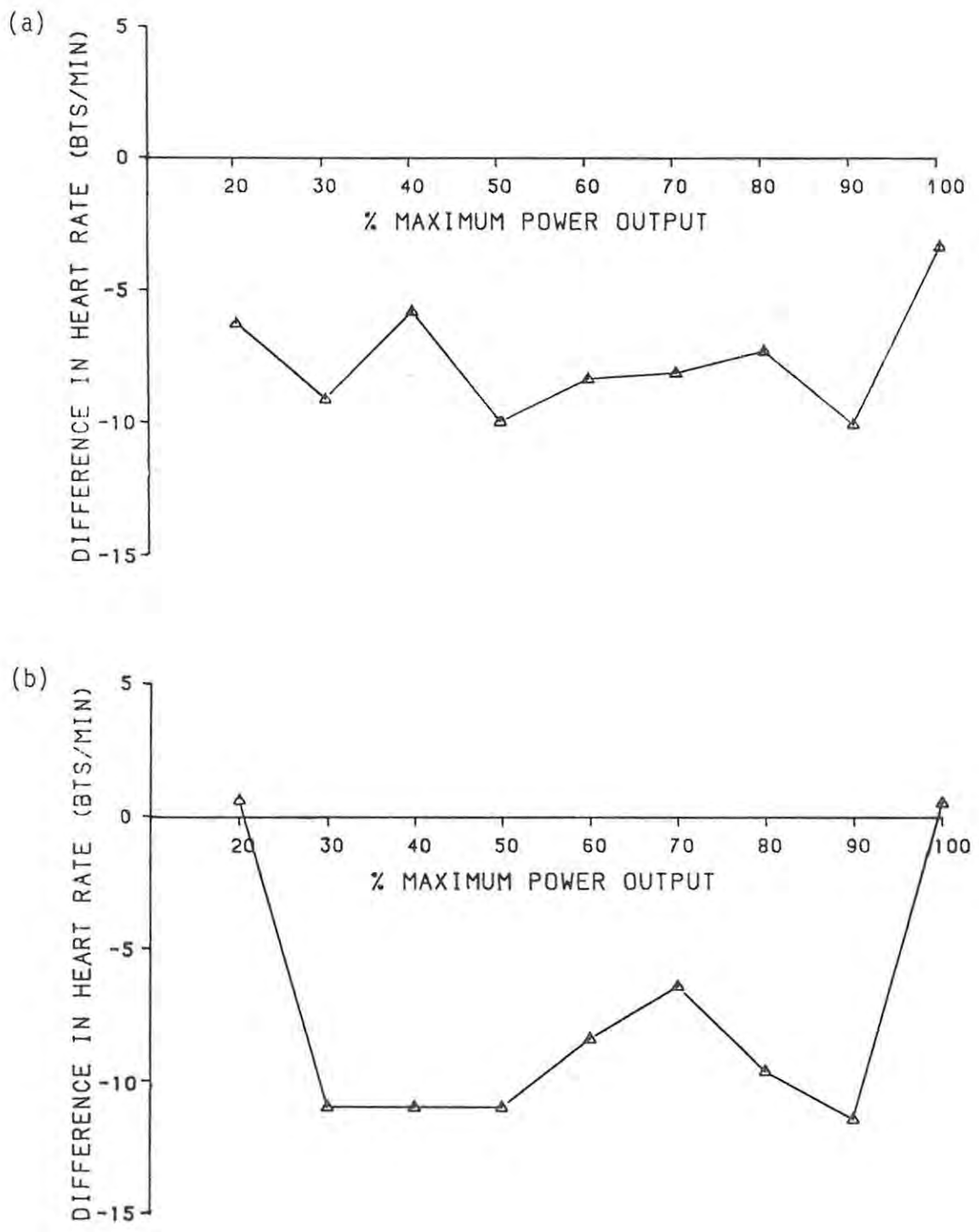


Figure 5: The difference between the non-apneic and apneic mean heart rates on land (a) and underwater (b) at 10% increments of maximal power output.

Maximal Oxygen Consumption on Land and Underwater

Ten subjects underwent a land test of $\dot{V}O_2$ max, while eight participated in the underwater test. For both conditions, the test was readministered to three subjects within one week of the initial test.

A common complaint of subjects was a feeling of local discomfort and fatigue in the thigh muscles at heavy work levels. This applied to both testing conditions, but was more pronounced in the underwater test. Although a discontinuous protocol may have helped to reduce the effects of local fatigue, the administration of such a test would have been impractical given the underwater conditions. Prolonged exposure to cold (18°C) water has reduced $\dot{V}O_2$ max in lean swimmers (Nadel et al., 1974) and although the water temperature was higher in the present study it is possible that the additional exposure necessitated by a discontinuous protocol may have had deleterious effects upon the final results.

While it was recognised that local fatigue constituted a possible limitation to the subject's ability to continue cycling at heavy workloads (McArdle et al., 1973, Astrand and Rodahl, 1977) the $\dot{V}O_2$ max values obtained for pilot subjects on land compared favourably with those previously reported for bicycle tests (McArdle et al., 1973 and 1986, Dressendorfer et al., 1976). Underwater values were, on average, 27% lower than those recorded on land, a difference which was statistically significant at the $p0.05$ level. Related t-tests revealed that there was no significant difference in the $\dot{V}O_2$ max values measured on two separate occasions either on land or underwater.

The pilot study served to highlight a practical problem with respect to underwater data collection. Since this was performed in situ, that is, in a swimming pool, some distance from the laboratory, difficulties

were experienced in the storage and transportation of gas bags. While every attempt was made to avoid direct exposure of the bags to sunlight, hot environmental conditions were experienced; Collection of gas bags from the onset of the test would have prolonged the exposure of the bags to such conditions, particularly as transportation was similarly extended. For this reason the validity of using only the last three gas bags collected was examined. The results of the subsequent analyses are reported under Determination of Relative Work Intensities.

It was concluded that for the purposes of the present study the test protocol was valid and reliable. Both on land and underwater a maximum $\dot{V}O_2$ was attainable that represented the maximum possible for the particular set of conditions. In this sense the functional maximum obtained constituted a valid measure against which relative work intensities could be determined, since testing conditions would be unaltered during measurement of apneic heart rate responses.

SUBJECT SELECTION

16 healthy males volunteered as subjects for this study. As regular underwater training has been seen to affect the apneic heart rate response (Hong et al., 1970; Oldridge et al., 1978), selection was based on their degree of experience in underwater activities such as spearfishing and underwater hockey. Subjects were required to have participated on a regular basis in either or both of the activities mentioned above for at least six months, and to be involved in regular underwater training (at least twice weekly) at the time of testing. These individuals were contacted by personal communication. Subjects

were requested to sign an informed consent (see Appendix A) prior to their involvement in the research.

In order to examine the effect of lung volumes per se, subjects were divided into two groups on the basis of vital capacity expressed relative to body surface area. Group A comprised those with large relative vital capacities, Group B those with small relative vital capacities. To ensure that the groups did have significantly different lung volumes, with no overlap in the standard deviations, twelve of the initial group of sixteen subjects were included in this part of the research.

ASSISTANTS

The same assistants aided during pilot testing and each of the testing sessions for all subjects. They were experienced in scientific research and familiar with the exact protocols and equipment being used. Both the author and one of the assistants were trained in the field of life saving and basic cardiopulmonary resuscitation.

EXPERIMENTAL PROTOCOL

Subjects were requested to participate in four separate testing sessions, the times for which were chosen as those convenient for the individual. This was, however, restricted to between 9.30 am and 3.30 pm, with subjects generally choosing the same time of day for each session. This six hour testing period avoided those times of day suggested as having a significant effect upon physiological parameters such as heart rate (Faria and Drummond, 1982). Although these authors reported a tendency for resting heart rate to rise over the afternoon, this increase was not significant until early evening. In contrast to

Shephard (1984), who reported higher $\dot{V}O_2$ max values in the early morning and evening, Faria and Drummond (1982) found no significant difference in $\dot{V}O_2$ max over a 24 hour period. Testing times of 9.30 am were possible with regard to underwater testing conditions since the swimming pool was heated, thus ensuring a more or less constant water temperature throughout the day.

Details of the measurements to be made were set out in the informed consent sheet read by the subject before his agreeing to participate in the study, and were explained verbally immediately prior to each session. Subjects were requested to refrain from energetic physical activity, eating or smoking for at least one hour before each test (Astrand and Rodahl, 1977). A break of at least two days was maintained between the $\dot{V}O_2$ max tests and at least 24 hours between apneic testing sessions.

Barometric pressure, ambient temperature, water temperature where relevant, and relative humidity were recorded before each session.

EXPERIMENTAL PHASE

Session A: Land

Duration approximately 45 min.

1. General information : This comprised name, age and years of underwater experience.
2. Stature: This was measured with a portable Holtain stadiometer while the subject stood erect and barefoot, with heels, buttocks, upper back and rear of the head in contact with the vertical section of the

stadiometer, according to stipulations of Ross and Marfell-Jones (1982). The measurement was recorded in centimetres, to the nearest 0.1 cm (tolerance limit = 0.3 cm)

3. Mass: This was measured with a Seca beam balance scale and recorded in kilograms to the nearest 0.1 kg (tolerance limit = 0.2 kg) Body mass was measured again following the test of maximal oxygen consumption.

4. Lung volume measurement

A Godart Expirograph (EP 16000) was used for the analysis of lung volumes. The expirograph comprised a light-weight 9l capacity, water-sealed spirometer. A blower with a pre-set flow of 60 l.min^{-1} , minimised airway resistance to about 2 mm water pressure. The spirometer bell was attached, via a counterweight, to a recorder pen. Displacement of the spirometer bell thus resulted in movement of the pen, which was recorded on the kymograph.

The subject sat comfortably in front of the expirograph breathing room air through a rubber mouthpiece. A nose clip was used to ensure that breathing was through the mouth only. Once the subject appeared to be relaxed and breathing normally, the recorder was turned on to a speed of 60 mm.min^{-1} and a two-way valve was turned so that the subject breathed into the apparatus. After three or four normal breaths a verbal command was given at the end of a normal expiration, and the subject inhaled as quickly and as deeply as possible. At the same time the paper speed was immediately increased to 1200 mm.min^{-1} . The breath was then held for about 4s in order to produce a plateau on the spirogram (Figure 7) whereupon a second verbal command was given and the subject exhaled as quickly and as deeply as possible. The recorder was stopped and the

subject came off the mouthpiece. If the spirogram resembled the illustration in Figure 7 the test was not repeated. Since an earlier study by Candler (1986) using the same equipment and technique had revealed that volumes did not differ significantly over three consecutive trials, it was felt that one test was sufficient if an acceptable trace was obtained.

5. Maximal oxygen consumption

The protocol used was a direct, continuous, progressive cycle ergometer test. During the test the subject breathed through a Hans-Rudolf 2700 pulmonary valve. Nose clips were used both on land and underwater to ensure that breathing was through the mouth only. Atmospheric air was inhaled via a 75cm length of 3cm diameter Collins ridged tubing. Expired air from the valve was directed through a 100cm section of the same type of tubing to a two-way stopcock, thus allowing for the continuous collection of expired air in one of two 200l capacity meteorological balloons. Identical lengths of tubing were used on land and underwater. On land subjects were allocated sufficient time to habituate to the pulmonary valve and noseclip before beginning the test. They then remained on the mouthpiece for the duration of the test.

Peddalling was begun to the beat of a metronome at a rate of 60 rpm (Astrand and Rodahl, 1977) against a starting resistance of 1kg (approximately 60 W). The duration of each work load was two minutes, with an increase in the resistance load of 1kg at the end of the second and fourth minutes. Thereafter the load was increased by 0.5kg (approximately 30 W) at the end of each bout, until exhaustion was

reached (Table III). Expired air was continuously collected over each two minute period.

The subject was permitted to stop pedalling anywhere within the two minute period. He was, however, asked to indicate at the beginning of an exercise bout if he felt that he might be unable to complete the full two minutes, since it was necessary to record the time over which expired gas was collected for the final exercise bout.

Subjects were instructed that upon completion of the test they should pedal backwards until the load on the cycle ergometer could be removed and they could continue pedalling forwards slowly. This provided for an active recovery in order to prevent the effects of venous pooling.

Table III Two-minute incremented resistance loads (kg) and corresponding power outputs (W) for the continuous cycle ergometer test of $\dot{V}O_2$ max.

Time(mins.)	Load(kg)	Power output(W)
0-2	1	60
2-4	2	120
4-6	3	180
6-8	3.5	210
8-10	4	240
-etc-	4.5	270

The fractions of expired oxygen (FeO_2) and carbon dioxide (FeCO_2) within the collected gas were measured using an Ametek S-3A1 Oxygen analyser with N-22 oxygen sensor, and a Godart Capnograph Mark III. Gas mixtures of known concentration were used to calibrate the analysers before testing. The volume of expired air was measured using a Singer gasmeter (model DTM - 115) previously calibrated by the negative pressure water removal method (Goslin et al., 1984).

The equations forming the computer package used to calculate $\dot{V}\text{O}_2$ max and related variables, together with the computer programme listing are provided in Appendices C and D. An Apple IIe microcomputer was used.

6. Determination of $\dot{V}\text{O}_2$ max.

Termination of the test both in air and underwater was subjectively determined by the subject. Subjects were, however, encouraged either verbally, or in the case of the underwater test, with the use of hand gestures, to achieve their highest level in each test.

Maximum $\dot{V}\text{O}_2$ was chosen as the highest level of oxygen uptake achieved during the test (McArdle et al., 1973; Katch et al., 1982) It has been proposed that a reliable means of evaluating whether the subject's maximal effort has been attained in such a test is the appearance of a plateau in oxygen consumption, represented by changes of less than 50ml (Morehouse, 1972) or 2 ml.kg^{-1} (McArdle et al., 1986) despite a further increase in workload. Pilot testing for the present study, however, indicated that such a plateau was unlikely to be revealed, due to the nature of the two-minute gas collection period. The establishment of the $\dot{V}\text{O}_2$ max protocol is discussed under pilot testing results.

Furthermore, cognisance was taken of the effects of local fatigue and the unique set of conditions posed by the underwater environment upon the $\dot{V}O_2$ values obtained. While the effects of water upon the resistance load on the cycle ergometer, together with the additional resistance offered by the water to pedalling were not investigated per se, they were nevertheless recognised as common factors influencing the power output and consequently the $\dot{V}O_2$ of each subject. In view of the pilot test results the $\dot{V}O_2$ values obtained were accepted as constituting the functional maximum attainable for both the land and underwater environments. Since testing conditions would be unaltered during apneic testing sessions, heart rate responses could thus be examined in terms of exercise intensities relative to the maximal $\dot{V}O_2$ attained under the described conditions.

Session B: Land

Duration approximately 30 min.

1. Heart rate monitoring

Throughout the study heart rate was monitored by means of Hewlett Packard electrocardiographic (ECG) equipment. This consisted of a monitor (model 78660 A/B), a telemetry transmitter (model 78100 A), a telemetry receiver (model 7810 A), and Narco-Scientific self-adhering surface electrodes. The frequency range of the telemetry transmitter was 450-470MHz.

Electrodes were attached to the subject's chest in a modified lead II ECG configuration. The sites of attachment comprised the right and left sterno-clavicular joints, and the V5 position (mid-axillary line). If necessary, chest hair was shaved at the attachment sites and the skin prepared by rubbing with abrasive tissue, before being cleaned with ethanol. The electrodes were prepared with Hewlett Packard Redux creme.

Care was taken to avoid the pectoral muscles as far as possible when placing the electrodes.

2. Subject preparation

Following the adjustment of the equipment to suit the individual, the subject assumed a relaxed, seated position on a Quinton cycle ergometer. Under all test conditions the subject breathed through a Hans-Rudolf 2700 pulmonary valve prior to and after breath-holding. This was deemed necessary in order to standardise the procedure on land and underwater. Nose clips were used both on land and underwater to ensure that breathing was through the mouth only. Atmospheric air was inhaled via a 75cm length of 3cm diameter Collins ridged tubing. Expired air from the valve exited into the atmosphere by means of a 100cm section of the same type of tubing. Identical lengths of tubing were used both on land and underwater.

Since subjects remained on the mouthpiece for the duration of the test apnea was achieved with the mouth open, against a closed glottis. It has been suggested that the magnitude of the inspired lung volume prior to apnea may affect the heart rate response (Angelone and Coulter, 1965; Magel et al., 1982). The measurement of vital capacity in the present research involved a maximal inspiration followed by a maximal expiration. In an attempt to standardise the inspired lung volume and enable an investigation of the effects of relative vital capacities upon the heart rate response to be made, subjects were instructed to hold the breath following a maximal inspiration. This was preceded by normal breathing activity. Hyperventilation prior to apnea was not permitted. This represented in the first instance a concern for the subject's safety, as the normal respiratory drive is compromised by hyperventilation prior to apnea, and loss of consciousness due to oxygen deprivation may occur with little or no warning (Craig, 1961, Lin 1982).

Furthermore, it has been suggested (Lin et al., 1974) that the involuntary ventilatory activities (IVA) that signal the physiological breath-hold breaking point generate periodic pressure changes in the thoracic cavity which are beneficial to venous return and cardiac output. The initiation of such diaphragmatic contractions occurs within a narrow range of partial pressures of alveolar carbon dioxide (PACO_2). Hyperventilation, by reducing the rate of carbon dioxide build-up, thus prolongs that period prior to the commencement of IVA, and in doing so, may influence the heart rate response.

Further instructions to the subjects stressed the need for the glottis to remain patent during apnea, thereby eliminating the Valsalva manoeuvre. Elevated intrathoracic pressure is known to attenuate or abolish bradycardia (Lin, 1982), and may produce tachycardia (Magel et al., 1982). Subjects were practiced in the technique of performing a breath-hold without a Valsalva manoeuvre.

3. Apnea at work on land

Following the attachment of electrodes as described above, the subject assumed his position on the cycle ergometer and went on the mouthpiece. A 30s resting heart rate was then obtained using the ECG. Heart rate was timed by stopwatch over a duration noted on the ECG trace by using the recorder's event timer. While the heart rate was continuously monitored on the ECG screen, a trace was only recorded over the relevant periods.

The resistance loads against which the subject pedalled were equivalent to those which would elicit oxygen uptakes that were 50, 70, and 90% of his $\dot{V}\text{O}_2$ max for that environment. This was based upon pilot test

results. Work intensities were thus classified as "moderate" (50% $\dot{V}O_2$ max), "heavy" (70% $\dot{V}O_2$ max) and very "heavy" (90% $\dot{V}O_2$ max), each of which would be expected to induce tachycardia under normal respiratory conditions on land. The presentation of the different intensities was randomised by drawing cards a box, and a single blind technique was used to minimise extraneous biasing influences.

While the heart rate was continuously recorded in order to provide an immediate pre-exercise value, the subject was instructed to commence the apneic bout when he was ready. He then inhaled maximally and the breath was held according to the prescribed method. The point at which the subject began and stopped breath-holding was determined by watching the movement of the diaphragm in the pulmonary valve. Breath-hold duration and the ECG trace were recorded as described above. Immediately the subject commenced apnea he began pedalling at a fixed rate of 60 rpm and continued to do so until breath-holding ceased. The recovery heart rate was recorded over 30s.

After each exercise bout the subject remained seated on the cycle ergometer. The following bout did not begin until the heart rate was within $10 \text{ b} \cdot \text{min}^{-1}$ of the initial resting heart rate (taken over 30s and converted into beats per minute) and the subject was ready to continue. The mean heart rate value which appeared on the ECG screen and at the top of the resting 30s trace was taken as a convenient baseline measure against which to evaluate recovery heart rates. As in the pilot study, it was stressed that the subject must report any feelings of discomfort to the tester.

4. Ratings of perceived exertion (RPE)

The Borg scale of Ratings of Perceived Exertion (RPE) (Appendix A) was explained to the subject at the beginning of the testing session. Immediately upon cessation of apnea, the subject gave an overall rating of perceived exertion pertaining to how he felt during work of varying intensities on land under apneic conditions. An overall rather than differentiated rating was considered to provide an adequate indication of the general perception of effort under apneic conditions.

5. Work on land without apnea

Once the subject had fully recovered following the recording of heart rate during apnea at work on land the entire procedure was repeated at the same work intensity, but without apnea. In this instance, however, the subject was instructed to cease pedalling once he had reached the total time over which the breath had previously been held. It was hoped in this way to provide a heart rate standard for work on land with normal breathing activity, against which apneic heart rate responses at the same percentage of $\dot{V}O_2$ max and over the same period of time could be compared.

As soon as the subject stopped pedalling he once again gave an overall rating of perceived exertion.

Session C: Underwater

Duration approximately 45 min.

1. Mass

This was measured using a Krups portable scale prior to immersion in water for the $\dot{V}O_2$ max test. Mass was recorded in kilograms to the nearest 0.1 kg.

2. Habituation

It was felt that since all subjects were confident during whole body immersion in water and were familiar with the use of a snorkel in underwater activities, a short habituation period immediately prior to the first underwater testing session would be sufficient.

Furthermore, pilot testing revealed that for subjects who were competent swimmers but not particularly experienced in an underwater environment, there was no significant difference in the $\dot{V}O_2$ max obtained upon readministration of the test, despite the habituation to the testing condition which can be assumed to have occurred during the first session. Subjects were informed that if initially they were not comfortable with the testing conditions they were to remain submerged on the cycle ergometer until such time as they felt completely at ease. They would then return for the actual data collection at a later stage. Personnel trained in lifesaving and basic cardiopulmonary resuscitation were on hand at all times.

During actual testing no difficulties were experienced in subjects habituating to the underwater conditions in the first session. The mean immersion time prior to the first underwater test ($\dot{V}O_2$ max) was 6 min 2s, with a range from 4 min to 10 min 10s. Prior to the apneic underwater session subjects were once again allowed to remain submerged using the breathing apparatus until each felt confident to continue with the test.

3. Use of the cycle ergometer underwater

The underwater testing protocol was intended to replicate that of on land as closely as possible. A Quinton cycle ergometer was submerged in the shallow end of a swimming pool. The range in the sitting heights of the subjects (89 - 95.5cm) was such that it was possible to maintain a reasonably uniform depth of immersion throughout the whole group ($\bar{x}$ = 172.5cm $\pm$ 1.2; range = 170-174cm), and at the same time enabled each individual to remain in a seated position with the head just out of the water prior to each test. At the commencement of the test subjects then lowered the head below the surface of the water, producing a slight forward lean over the pedals. This working position was standard to the underwater and land conditions (Figure 6).

In order to counteract the positive bouyancy of the body underwater the use of toe clips on the pedals and a restraining belt around the subject's waist was deemed necessary. This relieved the subject of the effort required to simply maintain a seated position underwater. The divers belt worn by the subject incorporated a quick-release buckle, allowing him to pull it off at will. Adjustable straps attached to the front and rear of the belt secured the subject firmly to the ergometer seat without compromising the 'quick-release' nature of the system. The fastening of the toe clips was sufficient to hold the feet on the pedals, but loose enough to ensure that the subject could remove his feet from them should he wish to. At least two safety personnel were on hand during the underwater sessions to assist subjects at all times. In order to minimise the amount of time the subject spent in the water prior to habituation and the commencement of the test, as many as possible of the adjustments necessary for the individual were performed

on land before the ergometer was submerged. Forward motion of the ergometer caused by the movement of the subject was prevented by tying the rear to a rope which was secured at the end of the swimming pool by means of an iron peg.

A Hans-Rudolf 2700 pulmonary valve was used by the subject to breathe underwater (Figure 6). Atmospheric air was inspired by means of a 75cm length of 3cm diameter Collins ridged tubing attached to the inspiratory part of the valve. The tubing was held out of the water by means of a pole suspended over the subject from the pool-side. The expiratory part of the valve was connected to a second tube, 100cm in length, suspended on the support structure at the side of the swimming pool. This in turn was attached to a two-way stop-cock which could be turned to one of two meteorological balloons to allow for the continuous collection of expired air. Meteorological balloons were numbered and returned to the laboratory for analysis. For apneic testing conditions, where it was not necessary to collect expired air, the tube was not connected to the two-way stop-cock. Subjects still remained on the mouthpiece to enable them to breathe prior to and after apnea. Weights were attached to the Collins tubing and Hans-Rudolf valve in order to achieve neutral bouyancy underwater and thus overcome the tendency of the air-filled system to float (Figure 6).

4. Resistance to breathing

The maintenance of pulmonary ventilation during exercise underwater where breathing is performed through tubing exposed to atmospheric air, as in this study, is complicated by the increased load on the inspiratory muscles. Within the lungs the pressure is the same as at

(a)



(b)



Figure 6: Frontal (a) and side (b) views of the subject testing position underwater.

the water surface, that is, atmospheric pressure, whereas the outside of the thorax is subjected to atmospheric pressure plus the pressure of the column of water above the subject (Astrand and Rodahl, 1977). The maximum pressure the inspiratory muscles can overcome is quoted as varying from just above 70mmHg (Astrand and Rodahl, 1977), to 140mmHg (McArdle et al., 1986). According to several authors, however, provided the problem of additional dead space is solved by a system of valves, a depth of approximately 1.0m (approximately 76mmHg) would be the maximum that could be tolerated (Astrand and Rodahl, 1977; Lanphier and Camporesi, 1982; Lundgren and Pasche, 1984; McArdle et al., 1986).

In the present study the depth of the inferior angle of the twelfth rib was measured as an indication of the maximum possible depth of submergence of the lung with the subject seated on the cycle ergometer. This ranged from 42-50cm ($\bar{x}$ = 45.7cm) below the surface of the water. On the basis that fresh water exerts a force of one atmosphere (760mmHg) for every 10.33m below the water's surface (Astrand and Rodahl, 1977; Lundgren and Pasche, 1984; McArdle et al., 1986), the inspiratory resistance required to overcome external resistance at these depths was calculated as follows:

Depth of lung at deepest point (d) = 42 cm

$$d = 0.42 \text{ m} = 0.042 \text{ atm} = 31.9 \text{ mmHg}$$

Where d = 50 cm:

$$d = 0.5 \text{ m} = 0.05 \text{ atm} = 38 \text{ mmHg}$$

Thus the additional pressure required to expand thoracic dimensions at these depths ranged from 31.9 to 38mmHg, with a mean for the group of

34.7mmHg. If the lowest figure cited in the literature (70mmHg) is accepted as representing the highest pressure the inspiratory muscles can overcome, it can be seen that, even at a depth of 50cm, the additional force required to overcome external resistance constituted 54.3% of the maximum inspiratory pressure as a "worst case". The maintenance of pulmonary ventilation in this study was, therefore, deemed feasible.

5. Maximal oxygen consumption underwater

This protocol was identical to that used on land (see above) with the exception that subjects were unable to maintain the prescribed pedal rate of 60 rpm. Pilot testing revealed, however that subjects were able to maintain a remarkably steady rate of pedalling. Pedal revolutions were recorded on a manual counter at one-minute intervals for the duration of the test . (For detailed discussion see Pilot Testing Results). The tester remained in the water for the duration of the test and was able to indicate to the subject whenever he began to alter his rate of pedalling. Changes in the resistance load on the ergometer were carried out at a signal from the time-keeper at the pool-side.

Before he entered the water, the subject was instructed in the hand gestures used by the tester underwater. A "thumbs-up" sign from the tester was used to enquire if all was going well; the same sign from the subject was used as an affirmative response, while a negative response was indicated by pointing the thumb downwards. Towards the end of each two minute-bout the tester asked if the subject was prepared to attempt the next load by pointing to the weight in question. The subject was instructed that as he neared exhaustion he must purposefully communicate

this to the tester by the negative gesture described. The tester's hand was then raised above the surface of the water and lowered immediately the subject ceased pedalling. At this point the timer and gas collection was stopped.

The subject could thus perform these gestures and still retain a one-handed grip on the handlebars.

Session D: Underwater

Duration approximately 45 min.

1. The recording of heart rate underwater

Since the Hewlett-Packard ECG equipment was designed specifically for use on land, it was necessary, as in previous studies (Scholander et al., 1962; Harding et al., 1965; Strømme et al., 1970) to modify the available equipment. The method used in this study followed that developed by Candler (1986).

Each of the components of the Hewlett-Packard ECG equipment was retained, with slight modification to some. The Narco-Scientific self-adhering electrodes were once again attached to the subject in a modified lead II configuration. Each electrode, however, was covered with a layer of adhesive water-proof plaster and then with another layer of highly adhesive medical plaster. The electrode wires were lengthened to approximately three metres and connected to a telemetry transmitter placed inside a sealed plastic bag taped to the side of the swimming pool.

2. Apnea at work underwater

The protocol was indentical to that used during apnea at work on land, with the workload maintained at 50, 70 and 90% $\dot{V}O_2$ max. With the head just above the surface of the water, the subject went on the mouthpiece. A 30s resting heart rate was then obtained in the manner described earlier. The recording of heart rate and breath-hold duration was indentical to that during previous sessions in the laboratory, with the exception that the beginning and end of apnea were signalled by the subject himself. The subject was instructed that following a maximal inspiration he was to indicate the beginning of apnea by immediately lowering his head below the surface of the water, thus assuming the same position as during the $\dot{V}O_2$ max test. Cessation of apnea was signalled by the subject immediately raising his head above the surface of the water.

Ratings of perceived exertion were obtained following each exercise bout, pertaining to the subject's overall feelings of exertion during apneic work underwater. The use of a waterproof rating scale enabled the subject, while underwater, to provide these ratings immediately after pedalling ceased.

3. Work underwater without apnea

Once again, the protocol followed that used on land. Following the recording of apneic recovery heart rates the subject remained seated on the cycle ergometer. Heart rate was continuously monitored and only if it was within $10 \text{ b} \cdot \text{min}^{-1}$ of the initial 30s resting value (converted to beats per minute) was the subject allowed to continue. As in the laboratory, it was stressed that any feelings of discomfort must be

reported to the tester. Once the subject felt ready to continue, the entire procedure was repeated without apnea for each of the relative work intensities. Overall ratings of perceived exertion were again obtained.

DATA REDUCTION AND ANALYSIS

Lung Function Analysis

Figure 7 represents a typical spirogram, wherein each vertical division equals 50ml and each horizontal division 1mm. Static volumes (STPD) were calculated in litres, to the nearest millilitre, by multiplying all vertical divisions by 50ml.

All lung volumes were converted to STPD. In order to cancel out the effects of body size, specifically upon vital capacity (McArdle et al., 1986), all volumes were expressed relative to body surface area according, to the formula of DuBois and DuBois (1916):

$$B.S.A = \frac{(71.84 \times M^{0.425} \times S^{0.725})}{10\ 000}$$

Where M = mass (kg); S = stature (cm)

The computer programme used to calculate lung volumes relative to body surface area is provided in Appendix D.

Determination of Subject Groups

To explore the effects of relative lung size the initial group of 16 subjects was divided into two groups on the basis of large (Group A) or small (Group B) relative vital capacities. An independent Student's t-test was then applied to the data in order to establish whether a

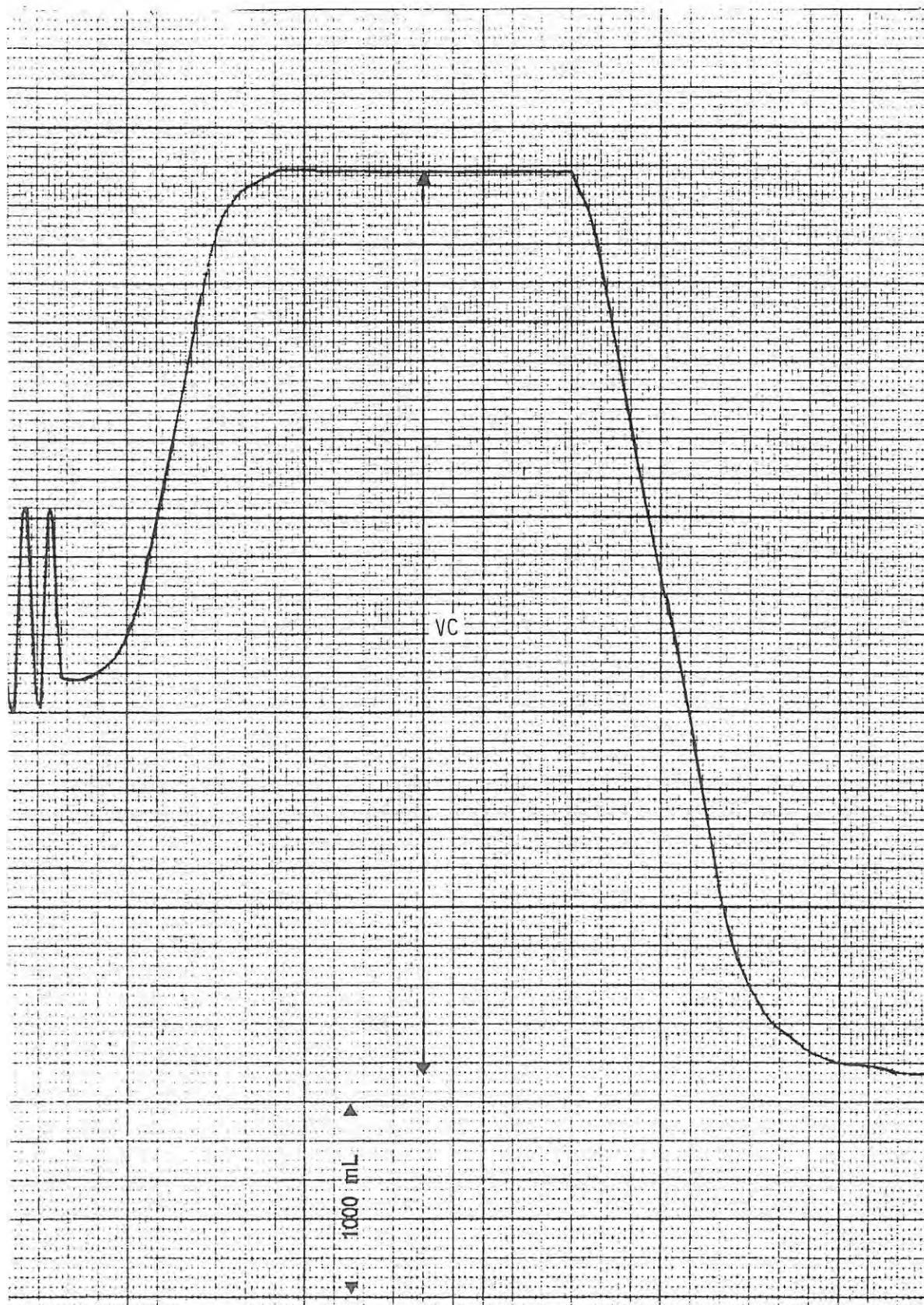


Figure 7: A typical spirogram.
Where: VC = vital capacity

significant difference did exist in the vital capacities of the two groups, without an overlap in the standard deviations of the groups. On the basis of this criterion it was necessary to reduce the sample size to 12, with six subjects in each group.

Heart Rate Analysis

The range of breath-hold duration observed in the present study (Land: 14 - 50s; Underwater: 17 - 55s) suggested that there existed great variation in absolute time for individual conventional breath-hold breaking points. This presented difficulties in establishing representative absolute time intervals for the measurement of heart rate. The examination of heart rate every 10s, for example, may have failed to illustrate important responses in the shorter breath-holds. Since it would not have been practical to examine each heart beat over the entire breath-hold, it was decided that for the purposes of statistical analyses heart rates would be measured over 10% increments, to 100% of total breath-hold duration for each subject in each condition. The examination of heart rate responses to apnea, therefore, was relative to each individuals breath-holding capacity.

Heart rate analysis was performed using a computer software programme (developed in the Department of Human Movement Studies at Rhodes University) which enabled R-R intervals to be digitized rather than measured manually (See Appendix D). A pilot study established the programme as being both valid and reliable. The R-R interval (mm) was divided into 1500 (paper speed = $25 \text{ mm.s}^{-1} \times 60\text{s}$) to give a heart rate in beats per minute. In addition to a beat-by-beat analysis of heart rate the programme provided mean heart rates over a given period (for

example, breath hold duration) and a mean of that entire period. Thus while the mean of each 10% of total breath-hold duration was used for statistical purposes it was possible to record the minimum heart rate during apnea for a given condition.

The pre-apneic heart rate was taken as the mean value from the 30s recording immediately prior to breath-holding, while recovery heart rate constituted the mean of the 30 seconds immediately after breath holding. In each case, however, beat-by-beat values were available.

Based on the definition of bradycardia as a reduction of heart rate below the pre-apneic level, the onset of bradycardia was defined as that point at which the heart rate slowed to a rate below the pre-apneic level; termination of bradycardia was that point at which the heart rate rose above the pre-apneic level.

Statistical Analyses

For all statistical procedures a level of probability of 0.05 was selected in order to reduce the probability of committing a Type I error, without unduly increasing the probability of a Type II error.

Hypothesis 1.

1. In order to examine the heart rate response to work at each intensity (50, 70, 90% $\dot{V}O_{2max}$) a two-way ANOVA with repeated measures on both factors (Ferguson, 1981) was applied to each of the four conditions: Land: Apnea and without apnea; Underwater: Apnea and without apnea. Factor One was relative exercise intensity, while Factor Two was percentage of exercise (breath-hold) duration.

In these and subsequent analyses of variance, where significant differences were indicated, a post hoc analysis in the form of a Sheffé test (Ferguson, 1981) was used to establish the exact location of those differences.

2. In order to identify more specifically the changes in heart rate response to each work intensity for each of the four conditions, a series of 12 one-way ANOVAs with repeated measures (Ferguson, 1981) was applied. In this way significant changes in heart rate over the exercise duration could be established.

3. Following this, a further three two-way ANOVAs with repeated measures on both factors were used to compare:

i) Heart rate values representing the maximum bradycardia during apnea on land and underwater (Factor One) at each of the relative exercise intensities (Factor Two).

ii) The difference in heart rate between apneic and non-apneic conditions at 90-100% of exercise duration for land and underwater environments (Factor One) at each of the relative exercise intensities (Factor Two).

iii) The difference between the pre-apneic control heart rate and the equivalent control value prior to exercise without breath-holding for land and underwater environments (Factor One) at each of the relative exercise intensities (Factor Two).

4. In order to establish whether apneic heart rates over 10% increments of total exercise duration were indeed significantly different from

corresponding non-apneic heart rates over the same time period, six two-way ANOVAs with repeated measures on both factors were applied. Land and underwater responses were separated for each of the relative intensities (50, 70, 90% $\dot{V}O_2$ max) in order to compare apneic and non-apneic heart rates (Factor One) over 10% increments of exercise duration (Factor Two). For reasons explained under pilot testing, this would not have yielded the information required under step two of this section, thus separate analyses were necessary.

This procedure was then repeated, separating apneic and non-apneic responses, in order to compare the land and underwater heart rates (Factor One) over 10% increment of exercise duration (Factor Two) for each of the relative exercise intensities.

5. The related Student's t-test (Ferguson, 1981) was used to compare 30s resting heart rates recorded at both apneic sessions and converted into beats per minute, with the heart rates measured immediately prior to each apneic bout of exercise at the three relative exercise intensities.

Hypothesis 2

1. A two-way ANOVA with repeated measures on both factors was done to examine the effect of land and underwater environments (Factor One) upon breath-hold duration for each of the three relative exercise intensities (Factor Two).

2. A second two-way ANOVA with repeated measures on both factor was used to investigate whether the order in which breath-holds were

performed (Factor Two) had any effect upon breath hold duration, in either environment (Factor One).

Hypothesis 3

Four two-way ANOVAs with repeated measures on both factors were applied to ratings of perceived exertion (RPE) in the following manner:

i) A comparison of apneic and non-apneic responses on land (Factor One) at each of the three relative exercise intensities (Factor Two).

ii) A comparison of underwater responses in the same way.

iii) A comparison of land and underwater responses during apnea (Factor One) at each of the three relative exercise intensities (Factor Two)

iv) A comparison of non-apneic responses in the same way.

Hypothesis 4

1. An independent Student's t-test (Ferguson, 1981) was used to establish significant differences between groups A and B on the basis of relative vital capacities. The same t-test was used to further compare the groups in terms of age, underwater experience, mass, $\dot{V}O_2$ max (land and underwater), breath-hold duration and pre-apneic heart rates and was applied to the environmental conditions (temperatures, relative humidity and barometric pressure) experienced by both groups during apneic testing sessions.

2. Following identification of the exercise intensity at which the most pronounced bradycardia occurred during apnea for the entire group,

(Hypothesis 1; Step 1) Separate one-way ANOVAs with repeated measures were applied to the two groups' apneic heart rate data for that percentage of $\dot{V}O_2$ max (50%), at 10% increments of exercise duration.

3. Two two-way ANOVAs with repeated measures on both factors were done to compare the general responses of the two groups in both environments. Thus Factor One was lung group, and Factor Two, percentage of exercise duration.

4. A two-way ANOVA with repeated measures on both factors was used to examine the lowest heart rates obtained (maximal bradycardia) for each group (Factor One), on land and underwater (Factor Two).

CHAPTER 4

RESULTS AND DISCUSSION

SUBJECT CHARACTERISTICS

In this study 16 healthy male subjects participated in four separate testing sessions. The physical characteristics of the subjects appear in Table IV.

In order to explore the relationship between apneic heart rates and lung volume, 12 of these subjects were then divided equally into two groups on the basis of vital capacity expressed relative to body surface area. All lung volumes were converted to STPD. A summary of the data relevant to each group appears in Table V

It is clear that in absolute terms, the vital capacities of Group B fell at the upper limit of, and those of Group A above the 4 to 5l cited as "average for young males" by McArdle et al. (1986). The values for both groups compared favourably with those reported elsewhere for similarly aged males (Craig and Dvorak, 1975; Buono, 1983; Shiraki et al., 1985). While the relative vital capacities of Groups A and B were significantly different, no significant differences existed for age, underwater experience, mass, or $\dot{V}O_2$ max either on land or underwater.

ENVIRONMENTAL CONDITIONS

The mean environmental conditions that the subjects were exposed to during the four separate testing sessions are presented in Table VI. Whereas relatively stable ambient conditions were evident in the laboratory, greater fluctuation occurred in the outdoor environment. Of importance in this regard, however, was the small difference in water

Table IV: Summary of physical characteristics and physiological data obtained for 16 male subjects experienced in underwater apneic activities.

	$\bar{X}$	S.D	C.V
Age (yrs)	20.9	3.2	15.3
Underwater experience (yrs)	2.8	2.0	71.4
Mass (kg)	75.1	7.5	10.0
Stature (cm)	179.6	4.7	2.6
Body surface area (m^2)	1.94	0.1	5.15
Sitting height (cm)	91.8	1.8	2.0
Vital capacity - absolute (l)	5.3	0.5	9.4
Vital capacity - relative (l/m^2)	2.8	0.3	10.7
Land $\dot{V}O_2$ max (ml/kg/min)	47.7	5.8	12.2
Underwater $\dot{V}O_2$ max (ml/kg/min)	40.0	5.4	13.5

Table V: Summary of physical characteristics and physiological data of subjects with large relative lung volumes (Group A) compared to those with small relative lung volumes (Group B).

		Age (yrs)	Underwater Experience (yrs)	Mass (kg)	Body Surface area (m ²)	Pre-apneic Heart rate (b/min)	Vital Capacity (l)	Vital Capacity (l/m ²)	$\dot{V}O_{2\text{ max}}$ Land (ml/kg/min)	$\dot{V}O_{2\text{ max}}$ Underwater (ml/kg/min)
Group	$\bar{X}$	21.8	3.7	72.9	1.9	89.9	5.74	3.0	49.7	43.1
A	S.D	2.8	2.8	10.6	0.14	7.6	0.4	0.1	6.3	5.7
	C.V	12.8	75.7	14.5	7.4	8.5	7.0	3.3	12.7	13.2
Group	$\bar{X}$	21	2.5	78.9	1.9	83	4.9	2.5	46.4	38.9
B	S.D	4.6	1.8	4.7	0.1	14.7	0.4	0.2	4.3	4.4
	C.V	21.9	72	18.6	7.4	17.7	8.2	8	9.3	11.3

Table VI: Environmental conditions experienced by subjects during data collection.
(Mean and Standard deviation).

	Session A (land)	Session B (land)	Session C (UW)	Session D (UW)
Temperature (Dry; °C)	22.0 ± 1.6	23.1 ± 1.7	25.7 ± 4.9	30.3 ± 3.2
Temperature (Wet; °C)	20.0 ± 3.4	19.8 ± 1.4	21.6 ± 3.4	22.5 ± 1.9
Water temperature (°C)			24.4 ± 1.0	26.1 ± 0.8
Relative humidity (%)	78.4 ± 4.7	73.7 ± 3.9	67.8 ± 15.3	48.9 ± 8.9
Barometric pressure (mm g)	716.3 ± 2.3	716.0 ± 2.2	714.3 ± 3.2	713.6 ± 1.6

temperature (1.7°C) between the two underwater conditions. A mean temperature of 24.4°C for the underwater $\dot{V}\text{O}_2$ max (Session C) was only slightly lower than the 25°C preferred by subjects performing maximally during head-out immersion (Dressendorfer *et al.*, 1976). The small variation in water temperature ($26.1^{\circ}\text{C} \pm 0.8$) during the apneic condition can be attributed to a heating system in the swimming pool which ensured a more-or-less stable temperature from one individual to the next.

RESULTS

Maximal Oxygen Consumption on Land and Underwater

Cycling while totally submerged in water resulted in a significant decrease in $\dot{V}\text{O}_2$ max from values obtained on land (Figure 8). The ranges in absolute $\dot{V}\text{O}_2$ max for the land and underwater conditions were $2.8 - 5.3 \text{ l}\cdot\text{min}^{-1}$ and $2.3 - 4.7 \text{ l}\cdot\text{min}^{-1}$ respectively. The mean value obtained underwater was $0.62 \text{ l}\cdot\text{min}^{-1}$ lower, a decrease of 17.4%. A summary of the data appears in Table VII.

The trend revealed for ventilation reflected that of $\dot{V}\text{O}_2$ max, where a decrease in the mean minute volume of $23.6 \text{ l}\cdot\text{min}^{-1}$ (24.5%) in the underwater condition was identified as significant (Figure 9). Similarly, complete immersion in water caused a significant decrease of 9% in the ventilatory equivalent for oxygen (V.E. for O_2), although the ventilatory equivalent for carbon dioxide (V.E. for CO_2) was not significantly altered (Figure 10). There was a small but significant change in the respiratory exchange ratio (R) during immersion. Cardiac output ($\dot{Q}$) was significantly reduced (13.7%) underwater.

Based on the oxygen consumption data obtained, individual one independent variable regression equations were calculated for each subject in both environments. This was done in order that the exact time from the commencement of the test could be calculated for a given percentage of $\dot{V}O_2$ max, and subsequently the resistance load at that intensity determined. The linear regressions calculated on the basis of group data, together with the relevant correlation coefficients, are presented below:

$$\text{Land: } \dot{V}O_2 = 13.91 + 2.73 \text{ Time} \\ r^2 = 0.90$$

$$\text{Underwater: } \dot{V}O_2 = 21.0 + 1.24 \text{ Time} \\ r^2 = 0.72$$

Heart Rate

The heart rate values in all figures in this thesis are representative of the preceeding 10% interval (i.e 10% breath-hold duration represents 0-10% etc).

An examination of the general heart rate responses to varying relative exercise intensities (50, 70, 90% $\dot{V}O_2$ max) under apneic and non-apneic conditions was separated into land and underwater conditions. In the non-apneic condition significant increases in the mean heart rate response were identified between 50 and 90% and 70 and 90% $\dot{V}O_2$ max on land, and between 50 and 90% $\dot{V}O_2$ max underwater. When the heart rate responses at each intensity were combined there were significant increases in heart rates examined at 10% increments of total exercise duration both on land and underwater (Figure 11). In other words, as

duration of exercise increased, heart rate increased. In both environments, however, there were significant interactions between factors; thus the heart rate trends across 10% increments of exercise duration were qualitatively different for the different intensities of exercise. In terms of apneic responses, a significant difference in heart rate occurred between 50 and 90% $\dot{V}O_2$ max on land, but not between any of the exercise intensities underwater. Thus the significant increase in mean heart rate between the relative intensities that occurred in both environments for the non-apneic conditions, was completely absent underwater and occurred only between moderate and very heavy intensities in the apneic condition on land. Apneic heart rates at 10% increments of total exercise duration were markedly different from non-apneic responses. Apnea on land resulted in an initial significant rise, followed by a drop in heart rate, such that no significant difference existed between values for 0 - 10% and 90 - 100% of breath-hold duration (Figure 11a). Underwater heart rates were significantly higher between 20 and 40% of breath-hold duration than during the initial 20%. This was followed by a reduction until heart rates from 80 - 100% of breath-hold duration were significantly lower than at 0 - 10% (Figure 11b). Once again, however, a significant interaction existed between factors in both environments.

More specific study of heart rate responses involved separating apneic and non-apneic responses, on land and underwater for each of the three relative intensities (Table VIII). On land, apnea during exercise at 50% $\dot{V}O_2$ max (Figure 12a) resulted in a significant decrease in heart rate in the last 10% of breath-hold duration. This value was significantly lower than all values from 20 - 80% of breath-hold

duration. In contrast, exercise at the same intensity without apnea resulted in significant increases in heart rate until 80% of exercise duration whereupon the rises that did occur were no longer significant. At 70% $\dot{V}O_2$ max (Figure 13a) there was an insignificant rise in heart rate between 30 and 70% of breath-hold duration, followed by a decrease which became significantly lower than these values at 90 to 100% of breath-hold duration. Once again, exercise without apnea produced significant rises in heart rate until 80% of total duration. At 90% $\dot{V}O_2$ max on land (Figure 14a) the heart rate for 40 - 50% of breath-hold duration was significantly higher than for 0 - 10%, but thereafter declined until the value for 90 - 100% of breath-hold duration was significantly lower than values from 30% to 70% of breath-hold duration. Rises in heart rate during non-apneic exercise were significant until 80% of exercise duration.

In the underwater environment, apneic exercise at 50% $\dot{V}O_2$ max (Figure 12b) produced consistent decrements in heart rate from 30% of breath-hold duration until the termination of breath holding. Values over the last 30% of breath-hold duration were significantly lower than heart rates between 10% and 40% of breath-hold duration. The mean heart rate at 90 - 100% of breath-hold duration, which represented a 30% decrease compared to the pre-apneic value, was significantly lower than all values from 10% to 60% of breath-hold duration. In the non-apneic condition, heart rate rose, to the extent that values for the final 30% of exercise duration were significantly higher than those during the first 30%. No significant changes in heart rate occurred after 40% of exercise duration.

At 70% $\dot{V}O_2$ max (Figure 13b) apnea elicited significant heart rate reductions in the final 20% breath hold duration. The value for 90 - 100% of breath-hold duration was significantly lower than all previous values, with the exception of the mean heart rate for 80 - 90% of breath-hold duration. Exercise without apnea at 70% $\dot{V}O_2$ max produced significant rises in heart rate until 60 - 70% of breath-hold duration, whereafter no significant changes occurred.

At 90% $\dot{V}O_2$ max (Figure 14b) apneic exercise in this environment produced an insignificant rise in heart rate over the first 40% of breath-hold duration, with the result that significant differences existed between these values and those from 70% to 100% of breath-hold duration. Exercise of this intensity without apnea produced significant rises in heart rate until 70% of breath-hold duration, whereafter no significant changes occurred.

A comparison of apneic and non-apneic conditions revealed that with the exception of exercise at 70% $\dot{V}O_2$ max on land (see Figure 13a), where no significant difference existed, apneic heart rate responses were significantly lower than non-apneic responses at all relative exercise intensities, in both environments. Furthermore, a significant interaction existed between factors in each case; thus heart rate responses to apneic exercise were qualitatively as well as quantitatively different from those without apnea.

When land and underwater responses were compared for apneic and non-apneic conditions, at each of the relative exercise intensities, significant differences between the heart rates in the different environments existed only at 90% $\dot{V}O_2$ max (see Figure 14). This was true

for apneic and non-apneic conditions. At this intensity heart rates underwater were significantly lower than for the same exercise on land. Once again, however, the interaction between factors was significant for all conditions.

Maximal bradycardial responses (ie: the minimum heart rate recorded) were significantly lower underwater than on land. Although the bradycardia was more pronounced the lower the exercise intensity, the differences between intensities were not statistically significant (Figure 15). The interaction between factors was not significant, indicating that although heart rates were lower underwater, the responses were qualitatively the same in both environments.

A comparison of the difference in heart rate between apneic and non-apneic conditions at 90 - 100% of total exercise duration provided an indication of the extent to which the suppression in heart rate occurred in either environment. It was evident from Figures 12 to 14 that the most marked difference in heart rate occurred at this time. However the difference in heart rate between the conditions was not significant in the land and underwater environments; nor were the values significantly different at the various relative exercise intensities (Figure 15). No significant difference existed for the interaction between factors.

Analysis revealed that immediate pre-apneic heart rates were significantly higher than a 30s resting value recorded in each condition and converted into beats per minute. The only exception to this was the heart rate immediately prior to apneic exercise at 70% $\dot{V}O_2$ max on land. However the difference between the pre-apneic heart rate and the

equivalent control value prior to exercise without breath-holding was not significant on land or underwater, or between the exercise intensities, nor was there a significant interaction between factors.

Breath - Hold Duration

Although there was a tendency towards longer breath-holds underwater (Figure 16; Table IX) they were not significantly different from those on land, nor were there significant differences between exercise intensities, despite an overall trend towards shorter breath-holds, the heavier the workload. No significant difference in the interaction between factors indicated that the trends in breath-hold duration were qualitatively the same throughout. Furthermore, the order in which breath-holds were performed did not significantly effect the breath-hold duration in both environments.

Ratings of Perceived Exertion

Ratings of perceived exertion (RPE) were significantly higher in both testing environments for the apneic than for non-apneic conditions (Table IX), although the interaction between factors was not significant. When apneic and non-apneic conditions were separated, analysis revealed that land RPE were not significantly different from underwater ratings. In the non-apneic state, RPE increased significantly with increasing exercise intensity, whereas during apnea the increase between 70 and 90% $\dot{V}O_2$ max was not statistically significant (Figure 17).

Effect of Lung Volume

Following determination of groups A (large) and B (small) on the basis of relative vital capacity as described earlier, a comparison was made

of the apneic heart rate responses of the two groups over 10% increments of breath-hold duration. Heart rate values used were those obtained during apneic exercise at 50% $\dot{V}O_2$ max on land and underwater. This was identified as the relative intensity at which the most pronounced bradycardia occurred in both environments, for the original group of 16 subjects. Independent t-tests revealed that physical characteristics, environmental conditions and pre-apneic heart rates on land and underwater, which may have affected apneic heart rate responses, particularly underwater, were not significantly different from one group to the next. Breath-hold duration, however, was significantly longer for Group A. Nevertheless, there were no significant differences between the groups in their mean heart rate response to the apneic condition, nor was there a significant interaction between factors.

The heart rate response of each group was then studied separately in each environment, at 10% increments of breath-hold duration. On land (Figure 18a) the basic trend was an initial slight increase in heart rate, followed by a decrease until the termination of breath-holding. Thus for Group A the value for 90 - 100% of breath-hold duration was significantly lower than values from 20 - 50% of breath-hold duration. While the same trend was apparent for Group B, changes in heart rate that did occur were not statistically significant. Underwater (Figure 18b) the mean heart rate for Group A at 90 - 100% of breath-hold duration was significantly lower than the value for 20-30%, whereas Group B experienced decreases over the final 30% of breath-hold duration that resulted in significantly lower heart rates than for the first 50% of breath-hold duration.

Finally, when maximum bradycardia was examined, the mean heart rate for Group B was 14.6% lower than that for Group A, a difference which was not identified as significant. Furthermore, no significant differences

Table VII: Experimental results from maximal exercise on land and underwater (mean and standard deviation).

	Land	Underwater
Immersion time prior to test (min)		5.9 $\pm$ 1.87
Depth of immersion (cm)		172.5 $\pm$ 1.12
Depth of inferior angle of 12th rib (cm)		45.6 $\pm$ 2.71
$\dot{V}O_2$ max (l/min)	3.6 $\pm$ 0.65	2.9 $\pm$ 5.7
$\dot{V}O_2$ max (ml/kg/min)	47.7 $\pm$ 5.94	40.0 $\pm$ 5.56
$\dot{V}e$ (STPD) (l/min)	96.2 $\pm$ 20.4	72.6 $\pm$ 16.1
V.E. for O_2 (l/100ml/blood)	2.7 $\pm$ 0.22	2.4 $\pm$ 0.25
V.E. for CO_2 (l/100ml/blood)	2.4 $\pm$ 0.2	2.4 $\pm$ 0.24
R	1.12 $\pm$ 0.01	1.0 $\pm$ 0.06
$\dot{Q}$ (l/min)	23.7 $\pm$ 3.73	20.4 $\pm$ 3.3

Table VIII: Mean heart rate responses over ten percent increments of exercise duration for apneic and non-apneic conditions during exercise of differing intensities expressed relative to $\dot{V}O_2$ max.

% $\dot{V}O_2$ max and condition	10% increments of exercise duration (b/min)											
	Rest (b/min)	Pre-apnea (b/min)	0 - 10	10 - 20	20 - 30	30 - 40	40 - 50	50 - 60	60 - 70	70 - 80	80 - 90	90-100
LAND	70.8											
50 - AP	± 10.5	77.4 $\pm$ 13.3	82.8	93.3	94.2	96.3	97	95.2	93.4	91.4	82	71.8
50 - WAP		67.7 $\pm$ 14.7	69.2	83.5	90.0	93.9	97.8	102.2	103.1	107.1	110.8	113.7
70 - AP		74.5 $\pm$ 14.6	85.4	92.4	96.1	101.8	101.7	103.2	103.9	99.2	92.1	78.8
70 - WAP		65.7 $\pm$ 15.4	68.7	81.6	90.1	96.9	101.3	104.9	108.7	112.8	115.9	118.5
90 - AP		80.4 $\pm$ 14	87.6	91.7	102.0	108.7	111.2	110.3	110.6	106.9	97.8	84.2
90 - WAP		68.2 $\pm$ 10	70.7	85.8	96.5	102.6	109.5	114.3	117.3	121.7	125.8	128.2
UNDER-WATER	76.4											
50 - AP	± 9.0	88.7 $\pm$ 11.9	94.5	102.3	104.7	102.6	93.6	86.6	83.7	79.9	71.8	62.1
50 - WAP		77.8 $\pm$ 13.8	80.2	88.2	91.7	94.6	93.1	95.1	96.6	98.5	100.6	102.1
70 - AP		88.9 $\pm$ 10.4	93.4	96.5	101.2	99.5	93.7	91.4	88.8	87.2	75.1	63.9
70 - WAP		75.3 $\pm$ 12.5	76.0	85.4	89.4	94.4	96.4	98.4	100.3	102.5	104.5	106.6
90 - AP		85.1 $\pm$ 11.8	89.6	93.5	103.3	108.2	99.6	96.9	92.8	83.7	75.8	65.3
90 - WAP		176.7 $\pm$ 12.2	80.9	86.1	95.2	98.6	101.3	102.7	105.7	107.9	109.2	112.3

Table IX: Mean values for breath-hold duration and ratings of perceived exertion (RPE) on land and underwater for exercise of differing intensities expressed relative to $\dot{V}O_2$ max.

		LAND			UNDERWATER		
		50	70	90	50	70	90
Breath-hold duration (s)	$\bar{X}$	32.97	31.39	30.85	35.27	36.05	35.16
	S.D	7.8	8.0	7.8	8.5	8.4	8.8
Non-apneic exercise duration (s)	$\bar{X}$	32.97	31.69	30.73	35.62	35.97	35.13
	S.D	8.0	7.6	7.7	8.6	8.4	8.9
RPE (apneic)	$\bar{X}$	12.5	13.3	14.1	12.3	13.8	14.4
	S.D	1.5	1.6	1.9	1.8	2.0	1.5
RPE (non-apneic)	$\bar{X}$	10.4	11.5	12.5	9.9	11.3	12
	S.D	1.3	1.8	1.4	2.0	1.7	1.7

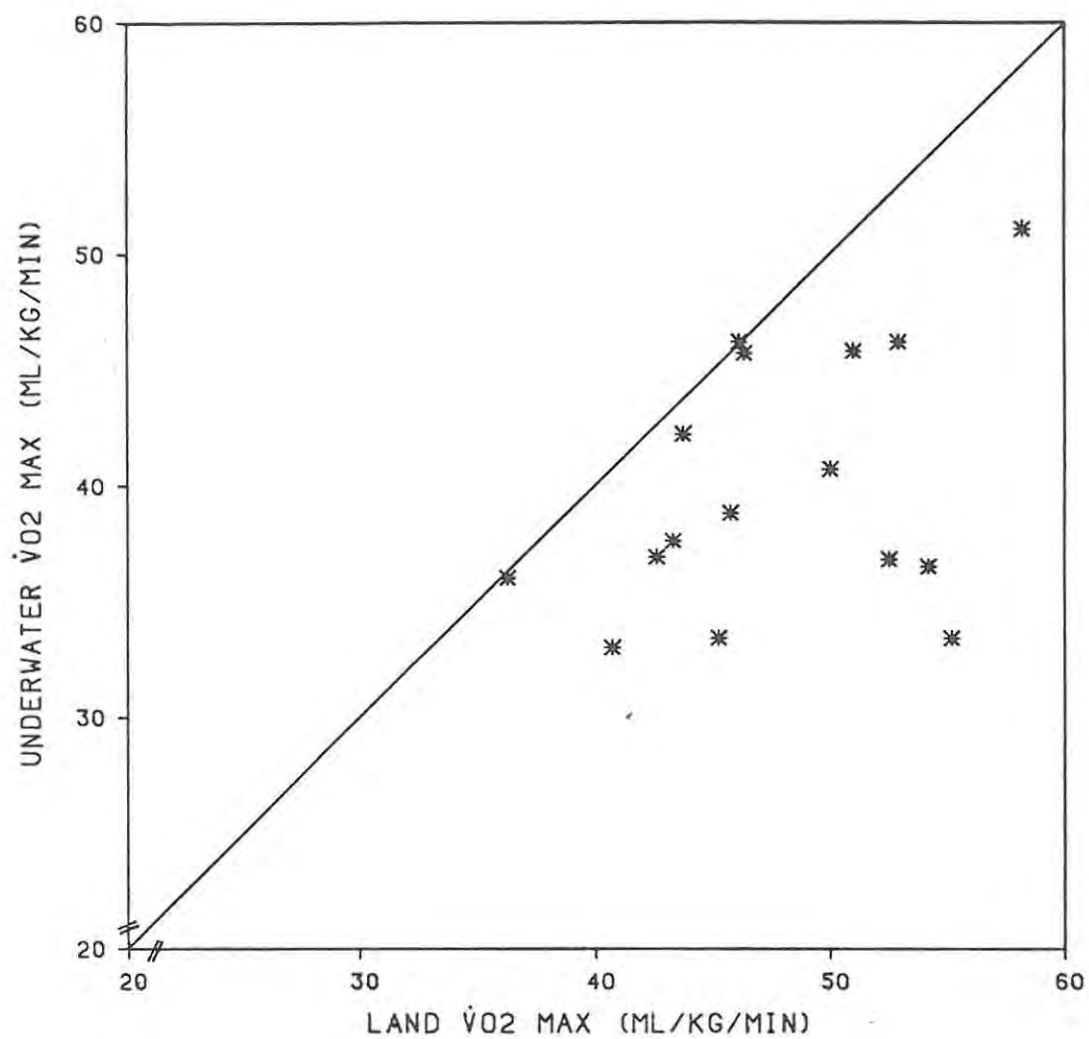


Figure 8: The relationship between $\dot{V}O_2$ max during cycling to exhaustion on land and underwater.

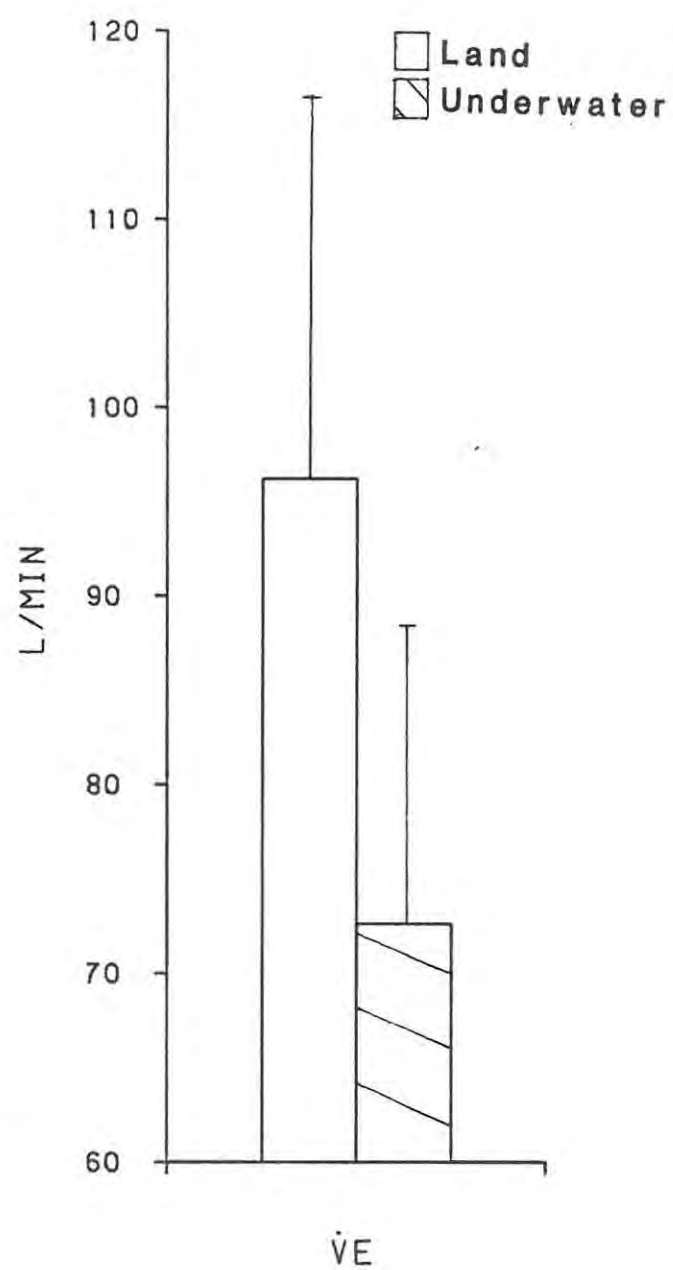


Figure 9: Minute volume ($\dot{V}_E$) during cycling to exhaustion on land and underwater.

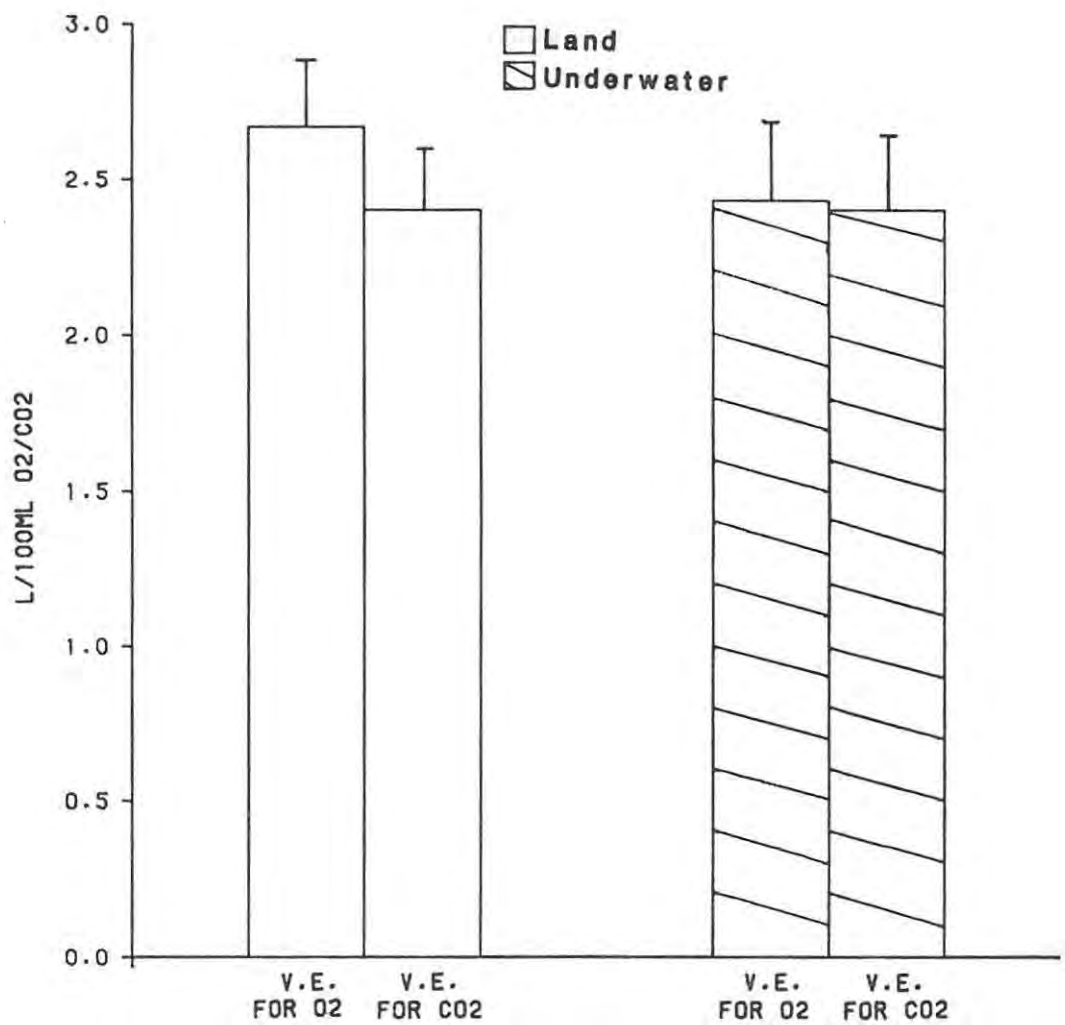


Figure 10: Ventilatory equivalent (V.E.) for oxygen and carbon dioxide during cycling to exhaustion on land and underwater.

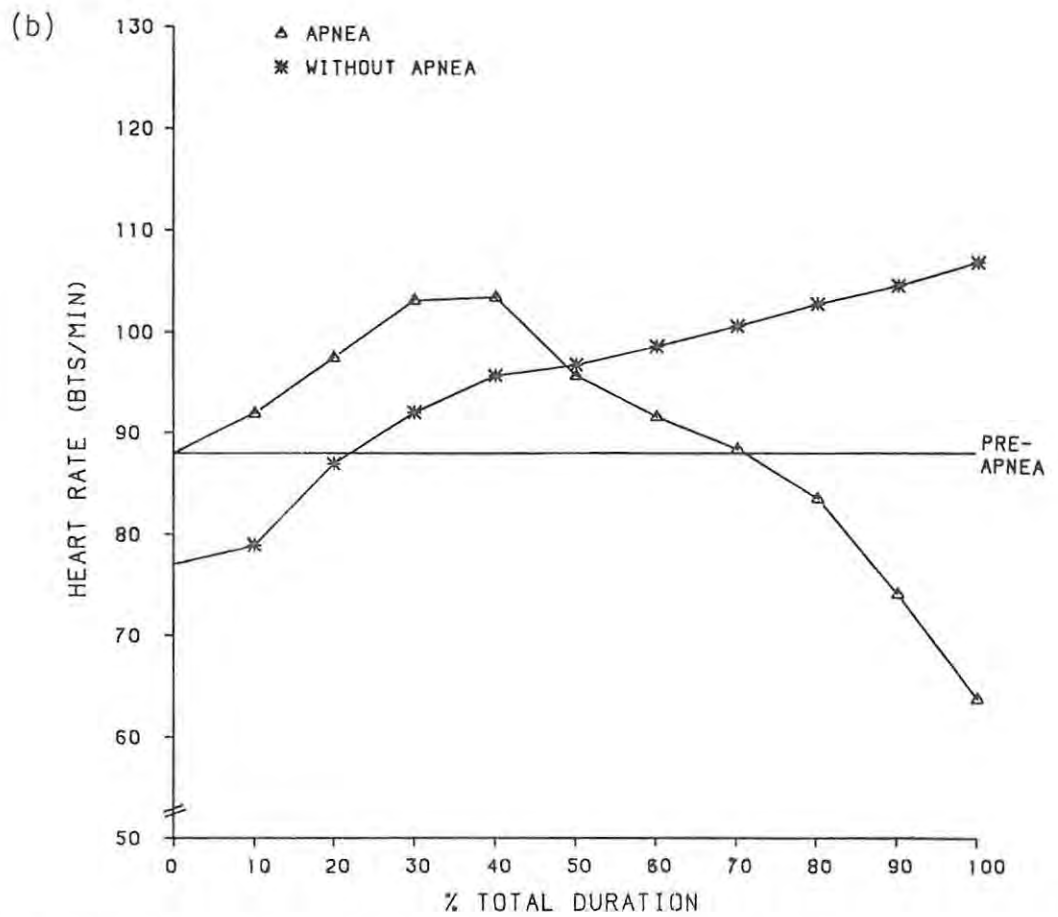
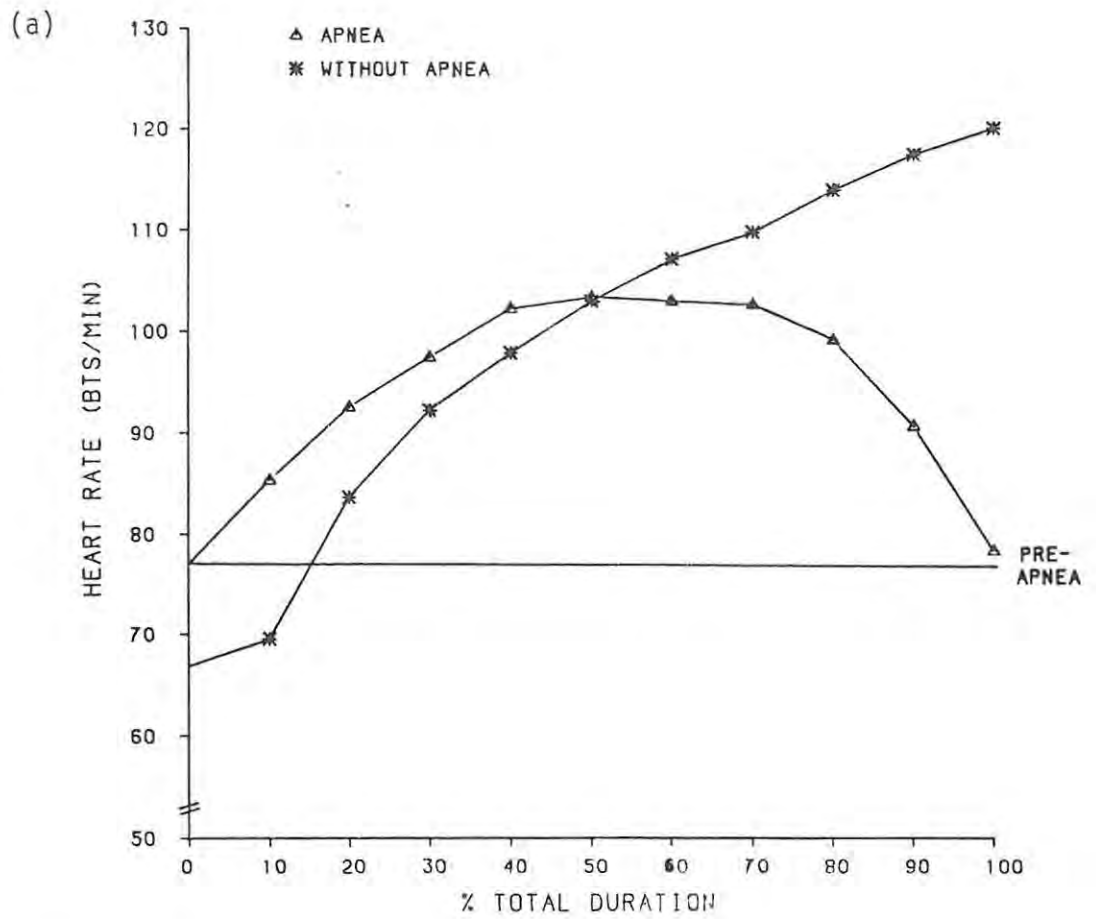


Figure 11: General heart rate responses on land (a) and underwater (b) to apneic and non-apneic exercise of varying exercise intensities.

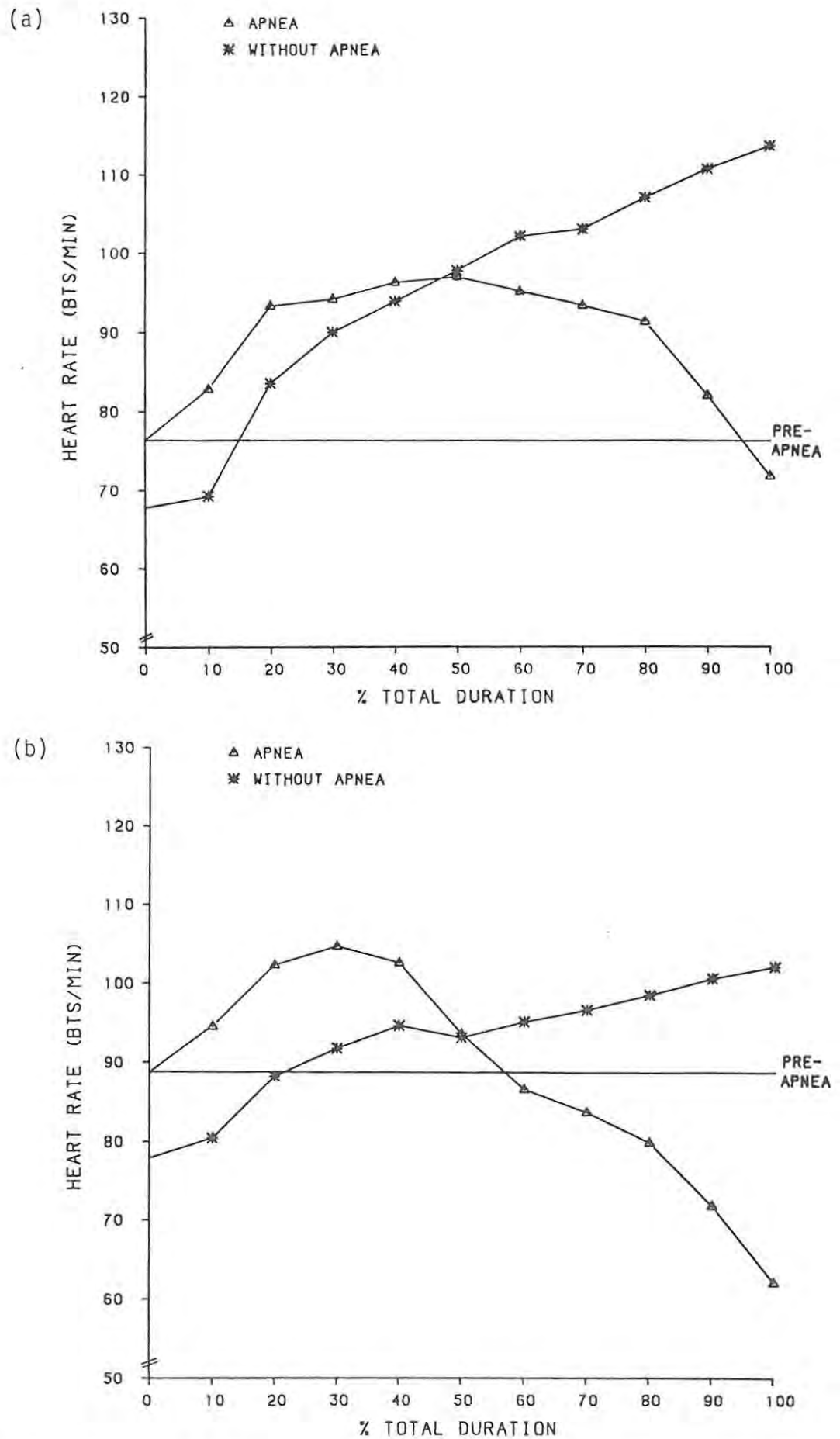


Figure 12: Heart rate responses on land (a) and underwater (b) to apneic and non-apneic exercise at 50% $\dot{V}O_2$ max.

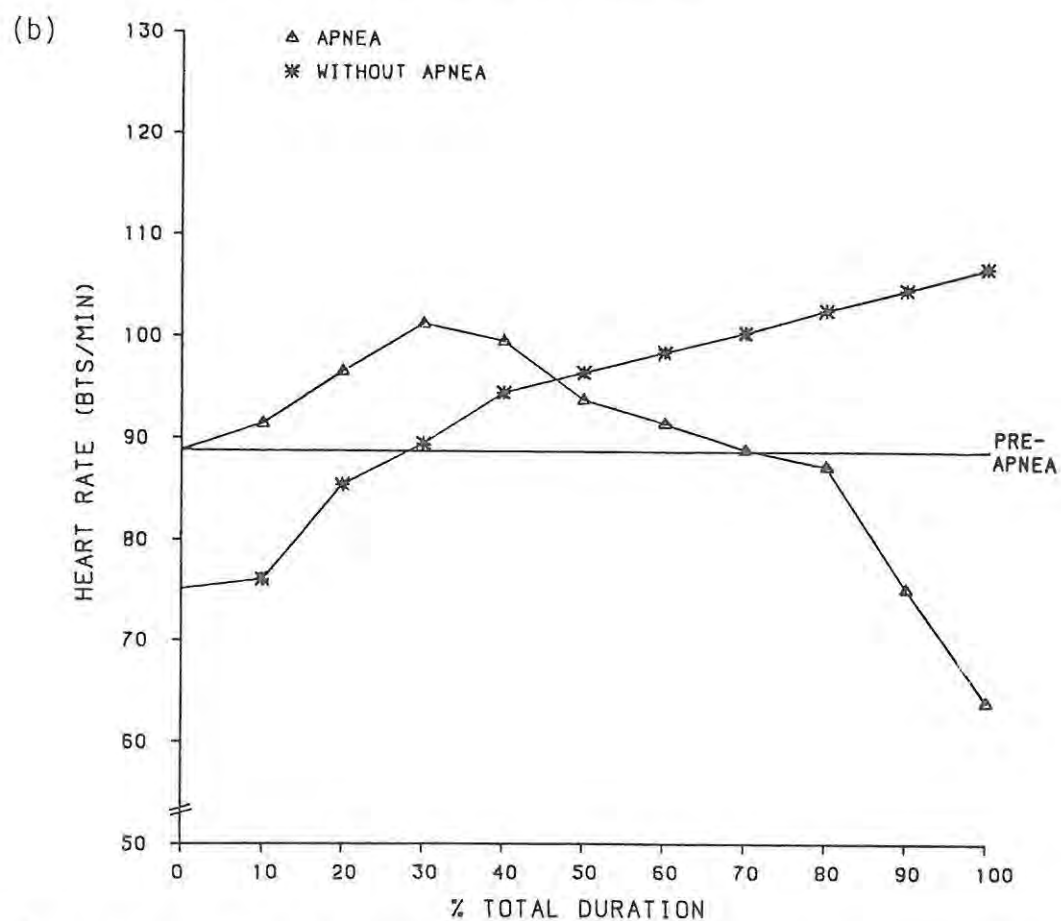
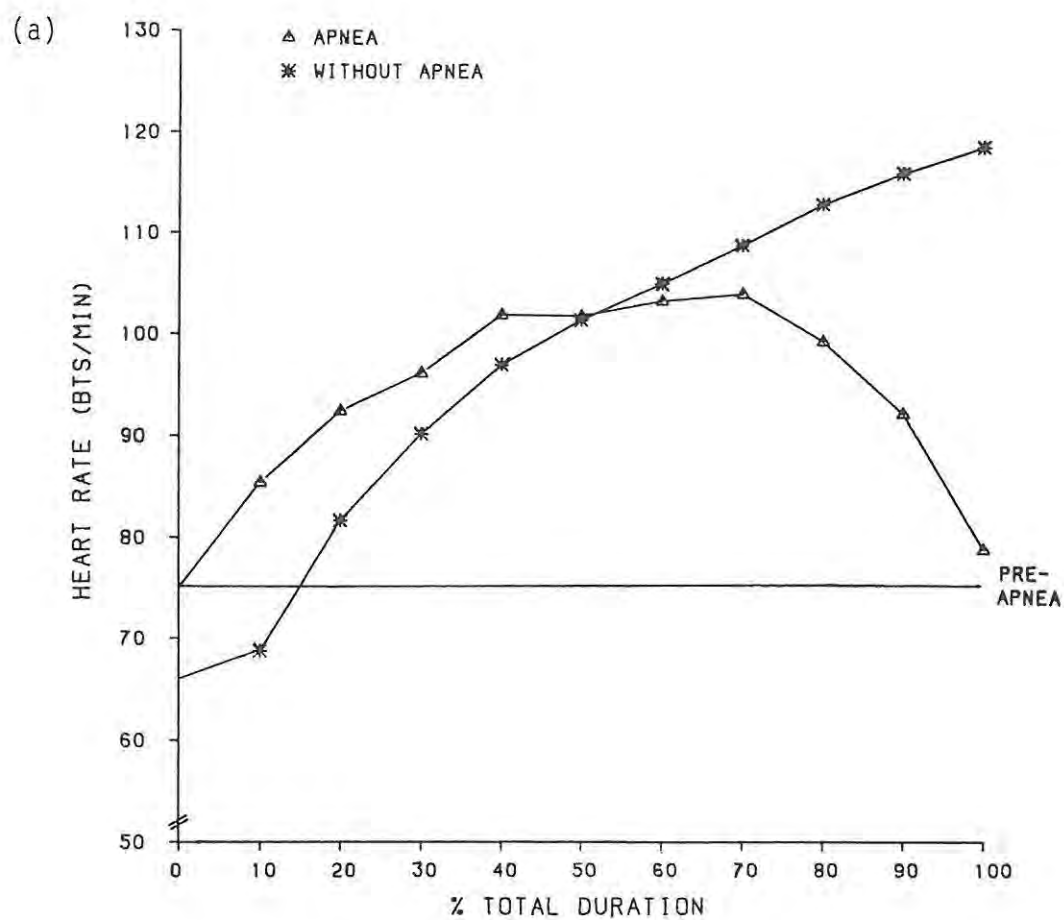


Figure 13: Heart rate responses on land (a) and underwater (b) to apneic and non-apneic exercise at 70% $\dot{V}O_2$ max.

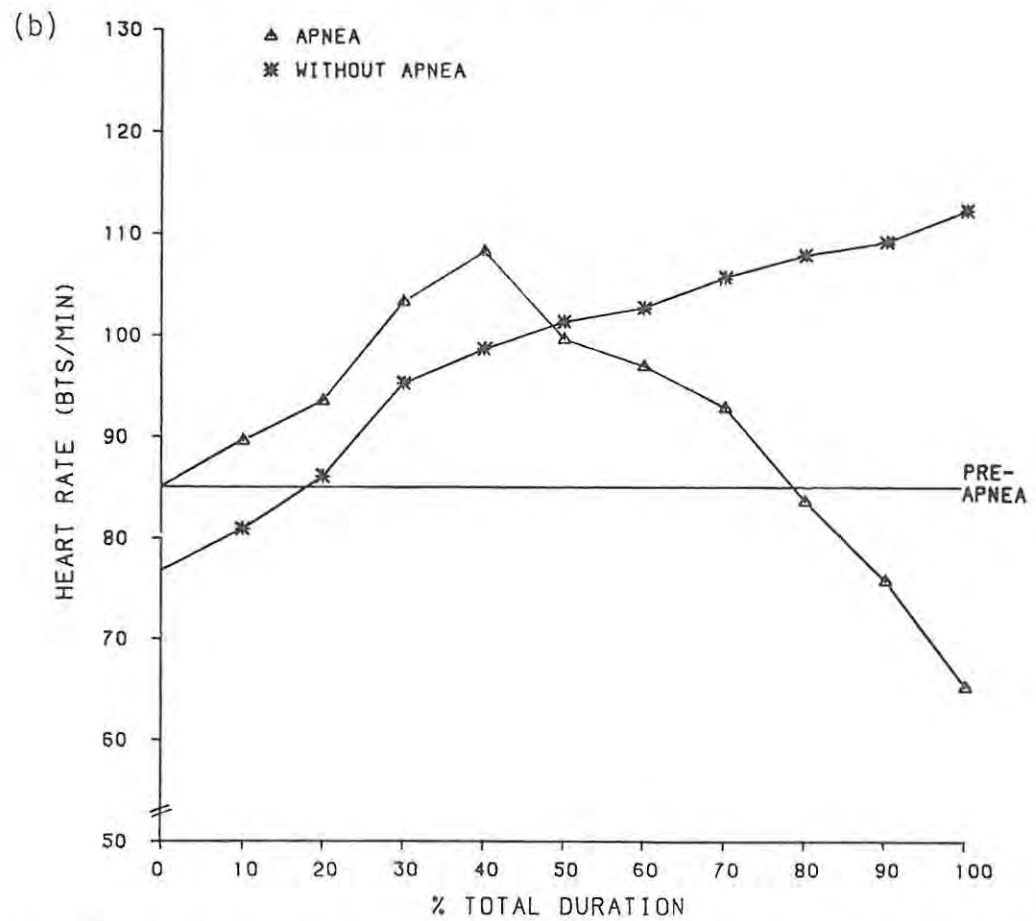
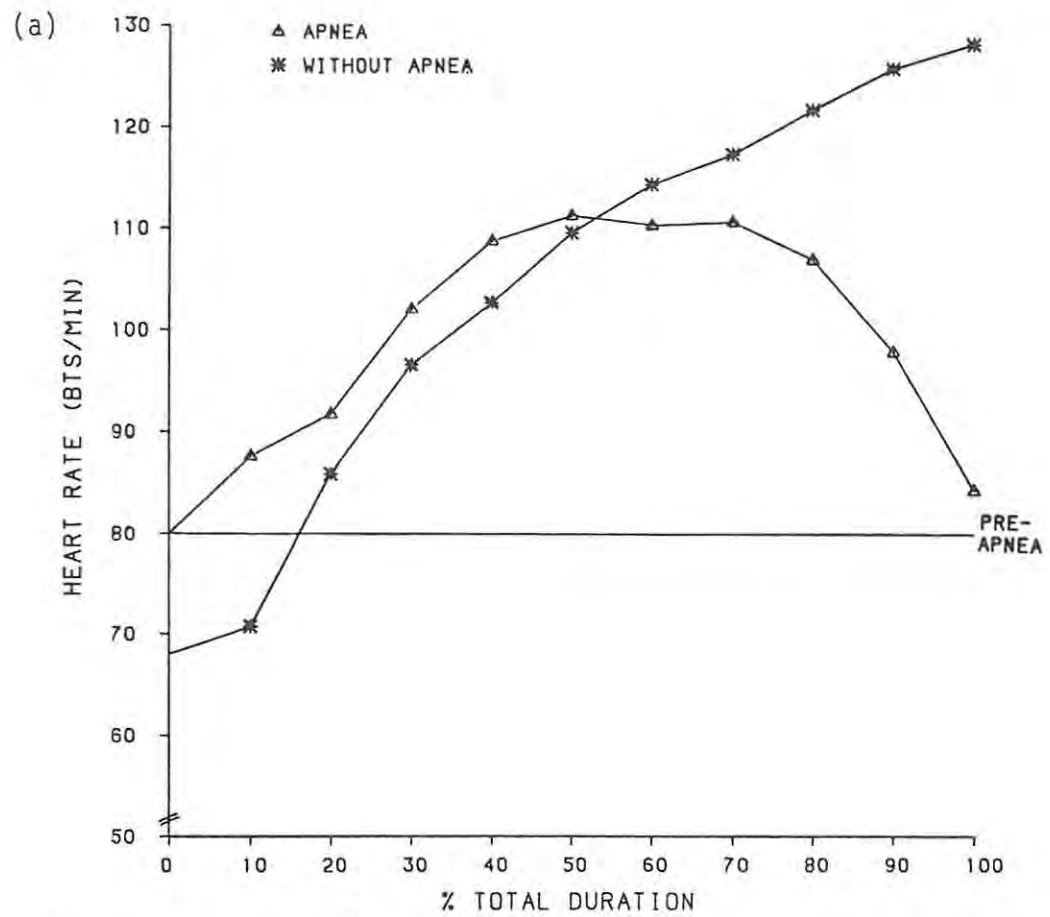


Figure 14: Heart rate responses on land (a) and underwater (b) to apneic and non-apneic exercise at 90% $\dot{V}O_2$ max.

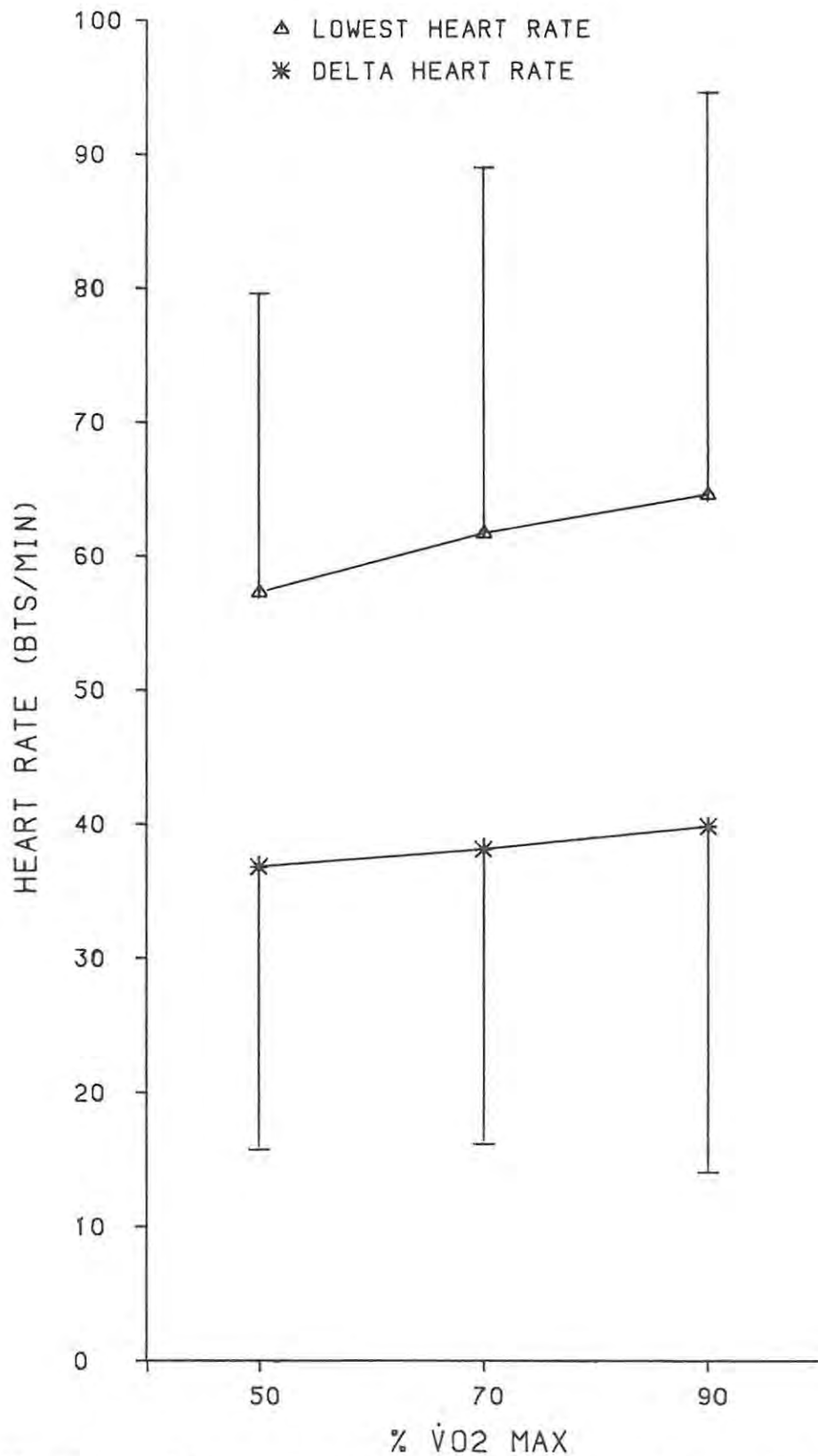


Figure 15: The lowest heart rate recorded and the difference in heart rate between apneic and non-apneic conditions at 90 to 100% of exercise duration (delta heart rate) for exercise at 50, 70 and 90% $\dot{V}O_2$ max (land and underwater responses combined).

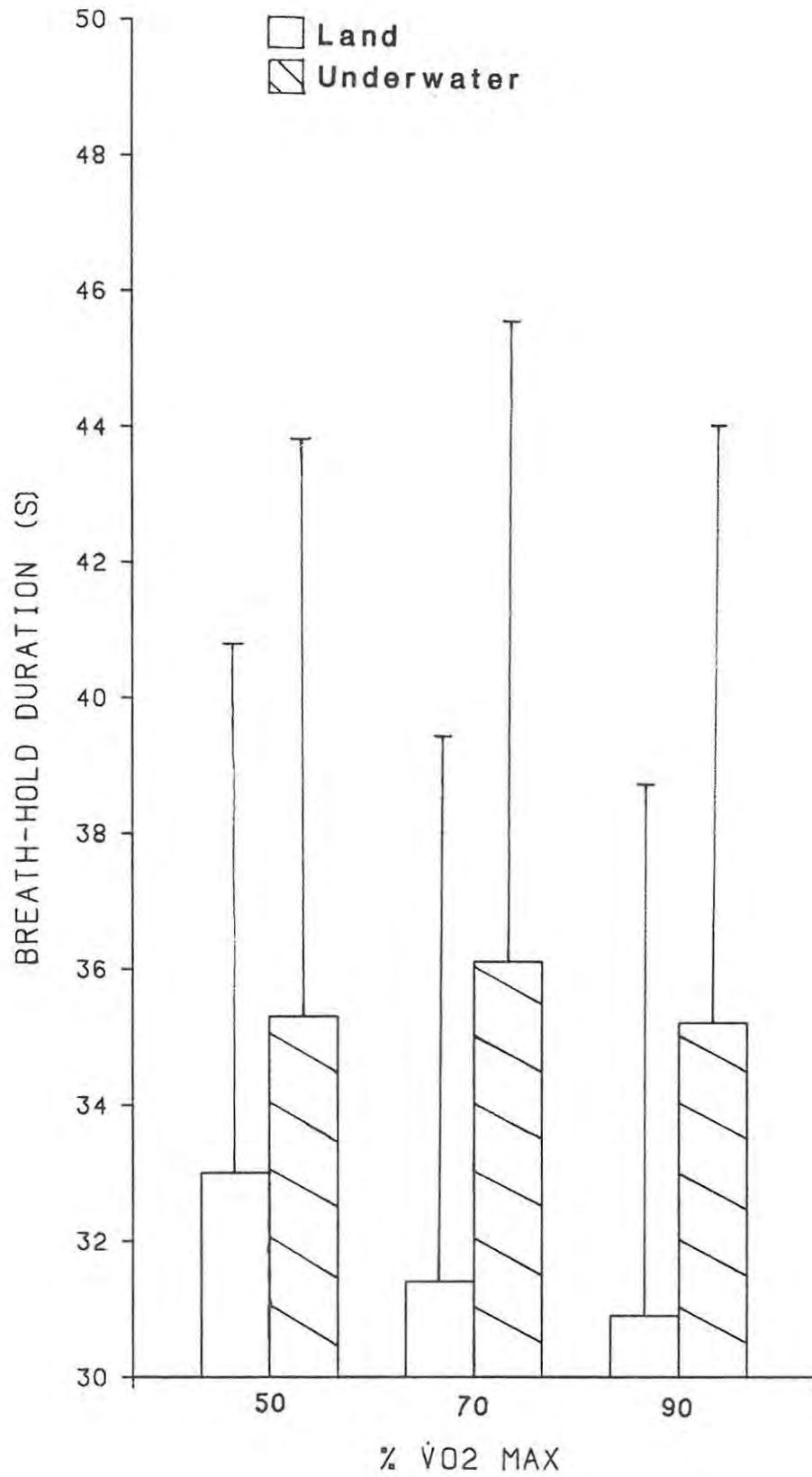


Figure 16: Mean breath-hold duration on land and underwater for exercise at 50, 70 and 90% $\dot{V}O_2$ max.

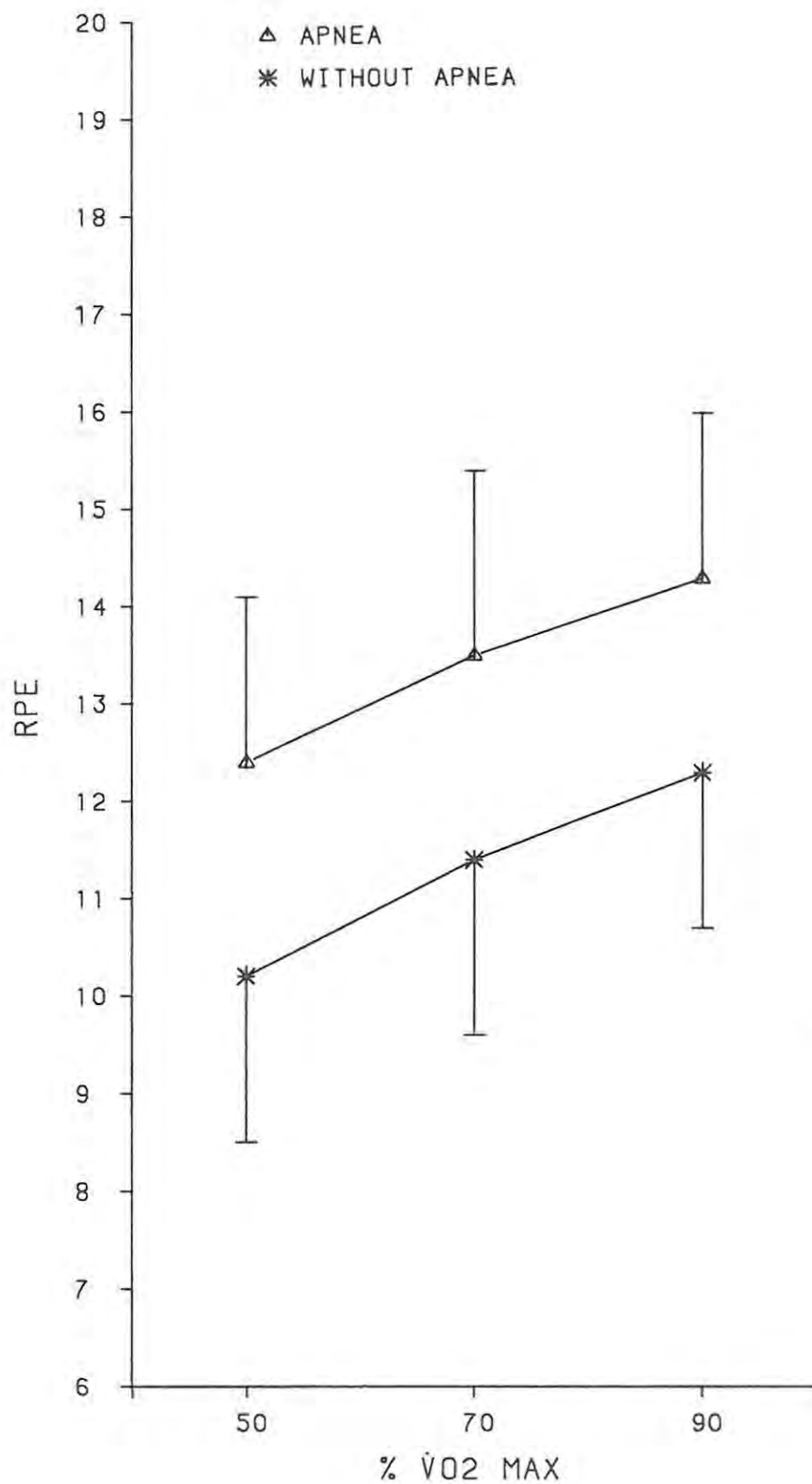


Figure 17: Ratings of perceived exertion (RPE) for apneic and non-apneic exercise at 50, 70 and 90% $\dot{V}O_2$ max (land and underwater responses combined).

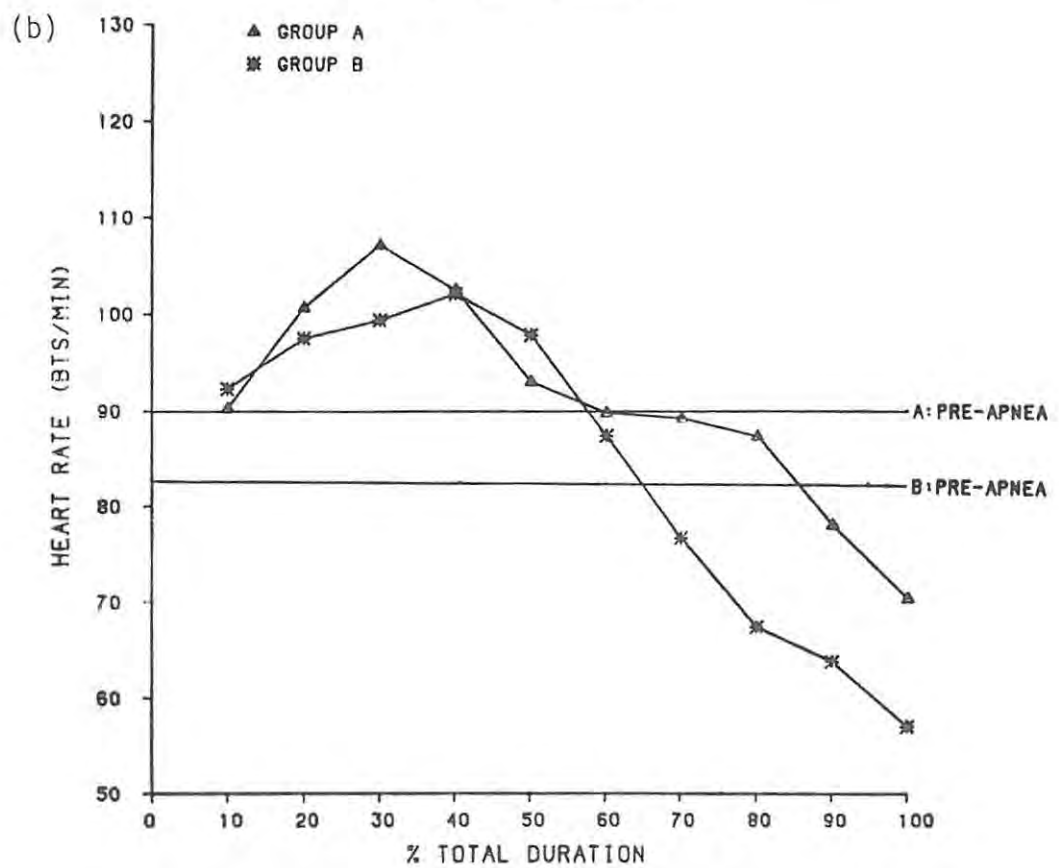
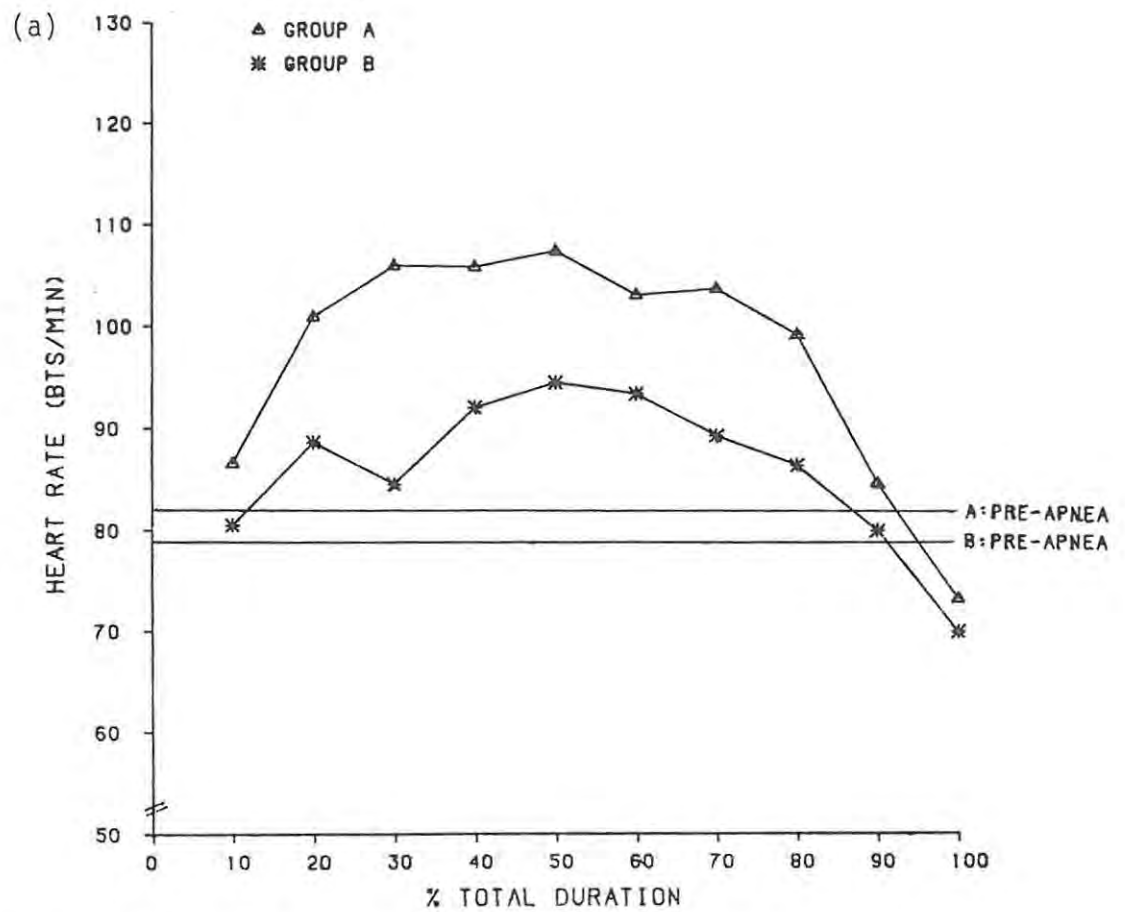


Figure 18: Apneic heart rate responses to exercise at 50% $\dot{V}O_2$ max on land (a) and underwater (b): subjects with large (Group A) and small (Group B) relative vital capacities compared.

existed between land and underwater values; nor was the interaction between factors significant.

DISCUSSION

Maximal Oxygen Consumption on Land and Underwater

A comparison of the same cycling exercise performed on land and underwater revealed that $\dot{V}O_2$ max was significantly reduced in the underwater environment. This is in contrast to the findings of Dressendorfer et al. (1976), who observed no significant change in the $\dot{V}O_2$ max of subjects cycling on land and during head-out water immersion, prompting them to conclude that the reduction in $\dot{V}O_2$ max typically reported for swimming in comparison to running or cycling (Astrand and Saltin, 1961; Holmér, 1978; Coetsee and Thiart, 1978) should be related to inherent differences in the activities studied, rather than the effects of water immersion per se. The present findings, however, indicate that the unusual cardiorespiratory conditions which exist during **total body** immersion should be taken into consideration in any comparison of performance in underwater and air environments. A reduction in $\dot{V}O_2$ max values with maximal fin swimming (SCUBA) at a depth of 3m versus at the surface has been reported (Coetsee and Thiart, 1978). Although the 6.5% decrease at 3m was not statistically significant in their case, it must be noted that a very small sample (N = 5) was used. The $\dot{V}O_2$ max values obtained in the present study for subjects cycling on land compare favourably with those reported elsewhere (McArdle et al., 1973; Dressendorfer et al., 1976), but are 23% lower than those reported by Dressendorfer and associates for cycling during head-out immersion in water of similar temperature (25°C).

The lower pulmonary ventilation ($\dot{V}_e$) during total body immersion concurs with previous observations made for whole body and head-out immersion (Magel and Faulkner, 1967; Dressendorfer et al., 1976; Holmér, 1978; Coetsee and Thiart, 1978), although contrasting findings have been reported where no significant change in minute volume occurred during graded leg exercise in air and underwater at a depth of approximately 1.22 m (Moore et al., 1970). Extensive investigations have, however, established that water immersion results in changes to the mechanics of lung function in man. These are attributed to the overall effects of increased hydrostatic pressure and intrathoracic blood volume that accompany water immersion (Burki, 1976; Holmer, 1978; Löllgen et al., 1980; Lin, 1984 and 1988).

In the present study $\dot{V}O_2$ max underwater was significantly reduced. This was accompanied by a significant reduction in the V.E. for O_2 . It is suggested, however, that the adverse effects of total body immersion upon the mechanics of respiration, manifest in a 24.5% decrease in $\dot{V}_e$, offset possible advantages to respiratory gas exchange brought about by increased pressure. Löllgen et al. (1980) point out that disturbances in cardiopulmonary function during head-out water immersion can only partly be compensated by opposing mechanisms, and the net results are ventilatory and ventilation perfusion ratio inequalities.

The V.E. for CO_2 was not significantly altered by the underwater environment. This can be attributed to a decrease in both minute volume and carbon dioxide production. That expired carbon dioxide was less for maximal exercise during total body immersion is substantiated by the significantly reduced respiratory exchange ratio (R) for this

condition. A lower respiratory exchange ratio during maximal swimming compared to running has been observed (Löllgen et al., 1980). The reduction reported for swimming was explained in terms of a lower alveolar ventilation, resulting in a higher alveolar PCO_2 .

The reduction in $\dot{V}O_2$ max observed underwater may be further explained by the significant decrease in cardiac output that occurred during total body immersion. Previous investigators have reported equivocal results with regard to the immersion effects on cardiac output, with some observing a decrease (Holmér, 1978), an increase (Löllgen et al., 1980), or an unchanged cardiac output upon water immersion (Rennie et al., 1971). Many of the apparent contradictions may be attributed to complex variations in the responses of cardiac output, heart rate and, to a lesser degree, stroke volume as determined by dissimilarities between studies in such factors as body position, type of activity or water temperature.

Effect of Apnea upon Heart Rate

The focus of this research was the reappraisal of current theories regarding the onset, course and termination of apneic bradycardia. In the light of an abundance of equivocal reports concerning this phenomenon, a central factor was the establishment of an operational definition of apneic bradycardia, against which results could be evaluated. Lin (1982) has pointed out that lack of uniformity in the interpretation of results may in many instances account for apparent discrepancies within the literature. Since the present study examined apneic heart rates during work classified as moderate to very heavy (50, 70, 90% $\dot{V}O_2$ max), a clinical definition of bradycardia as constituting a

heart rate below 50 or 60 $\text{b}\cdot\text{min}^{-1}$ (Neufeld et al., 1978; Arnold, 1985) was inappropriate. Gross et al. (1976) concluded that the characteristic heart rate response to breath-holding in man represented the domination of influences mediating vagal bradycardia over other tachycardia-producing reflexes. If this statement is considered, particularly with responses to exercise in mind, it is clear that where apneic heart rate responses are concerned, a distinction must be made between the phase in which tachycardia-producing reflexes dominate, and the phase in which vagal inhibition overrides such reflexes. Thus, in keeping with a more general description of bradycardia as a "relatively slow heart action" (Webster's, 1986), an operational definition was derived, whereby apneic bradycardia was defined as **a reduction in heart rate below the immediate pre-apneic level.** In other words, the onset of bradycardia marked that point in which tachycardial effects relative to a control heart rate immediately prior to apnea were overridden, with a resultant decrease in heart rate relative to that control value.

The present study revealed that in terms of this definition, apneic work elicited an overriding bradycardia underwater (Figure 11b), but failed to do so on land (Figure 11a). Rather, the collective response on land was what could be termed a suppressed tachycardia; despite a reduction in heart rate after an initial rise, the mean heart rate did not fall below the pre-apneic control level. The apparently enhanced slowing of the pulse by immersion during apnea is in agreement with many previous studies (Irving, 1963; Kawakami et al., 1967; Bove et al., 1968; Song et al., 1969; Strømme et al., 1970; Bergman et al., 1972), but not all (Craig, 1963; Brick, 1966; Craig and Medd, 1968; Asmussen and Kristiansson, 1968). These observations, however, highlight the

dangers which accompany a disregard for the quantification of exercise intensity in either environment. Within the general ambit of heart rate responses to apneic exercise, a significant interaction between factors was identified on land and underwater, indicating that the heart rate trends across 10% increments of breath-hold duration were indeed qualitatively different for the different intensities of exercise studied. Moreover, while the mean responses underwater were not quantitatively different at the different intensities, a significant increase in mean heart rate was established between 50% and 90% $\dot{V}O_2$ max on land.

Separate analyses of apneic and non-apneic responses, on land and underwater, for each of the three relative exercise intensities, permitted the establishment not only of the existence of bradycardia, but also the temporal development of this response. Figure 12b reveals that apneic bradycardia was in fact apparent at 50% $\dot{V}O_2$ max on land. Although the inhibitory effects of apnea upon the heart rate, manifest in a consistent decrease in values, were evident over the last 50% of the breath-hold, the decrease in heart rate below the pre-apneic level occurred within the last 10% of breath-hold duration. This, when translated into absolute terms, established the onset of bradycardia as occurring between 29.7 and 32.9s. A similar slowing of the heart rate, which commenced at 70% of breath-hold duration, was apparent at 70% and 90% $\dot{V}O_2$ max (Figures 13a and 14a). It would appear, however, that in neither case was vagal inhibition sufficient to override the increase in heart rate associated with heavy and very heavy exercise on land to the extent that bradycardia, in terms of the definition outlined previously, would occur.

In contrast, bradycardia was evident at all exercise intensities studied in the underwater condition. The onset of bradycardia, when expressed relative to total breath-hold duration, occurred between 50 and 60% at moderate workloads ($50\% \dot{V}O_2 \text{ max}$) and between 70 and 80% at heavy ($70\% \dot{V}O_2 \text{ max}$) and very heavy ($90\% \dot{V}O_2 \text{ max}$) workloads; this converted to absolute times of between 17.6 and 21.2s; 25.2 and 28.8s; and 24.6 and 28.1s respectively. In other words, at the same exercise intensity ($50\% \dot{V}O_2 \text{ max}$), the onset of bradycardia by the present definition occurred markedly earlier during immersion than on land, both in absolute and relative terms. The onset of the response was delayed similarly at 70% and 90% $\dot{V}O_2 \text{ max}$ compared to the lighter intensity in the same environment.

Once again, however, the inhibitory effect of increased vagal tone, described as a suppressed tachycardia, was evident prior to the onset of bradycardia as such. This commenced at 30% of breath-hold duration at moderate ($50\% \dot{V}O_2 \text{ max}$) and heavy ($70\% \dot{V}O_2 \text{ max}$) workloads, but was slightly delayed at the very heavy workload ($90\% \dot{V}O_2 \text{ max}$), occurring at 40% of breath-hold duration. That vagal inhibition was insufficiently strong to overcome an initial increase in heart rate concurs with some previous studies (Scholander et al., 1962; Gemma and Wells, 1987; Scott and Candler, in press). Gemma and Wells reported that the more difficult a movement pattern executed during synchronised swimming, the more prolonged was the increase in heart rate and the delay in bradycardia, but like Scholander and co-workers, offered no suggestion as to the underlying causes. Tentative proposals by Scott and Candler have included the influence of the Valsalva manoeuvre in increasing the heart rate while subjects lowered themselves into the water, and that

the mechanisms causing apneic bradycardia may only be stimulated after several seconds. Although body position in the present study was standard throughout the duration of apnea, the role of Valsalva during the initial stages of the breath-hold cannot be entirely discounted. It is possible that the effort required to overcome the initial resistance to pedalling, particularly underwater, resulted in a Valsalva manoeuvre, since a forced expiration against a closed glottis has the effect of stabilising the abdominal and thoracic cavities (McArdle et al., 1986). Subjects were, however, practised in the technique of performing a breath-hold without a Valsalva manoeuvre, and instructions to them stressed the need for the glottis to remain patent during apnea. Had Valsalva been performed for the duration of the breath-hold, it is likely that the acceleratory effects of this manoeuvre upon heart rate would have been evident over a greater proportion of the breath-hold (Magel et al., 1982). In light of the controversy regarding the causes of apneic bradycardia, the notion that such mechanisms may only be stimulated after several seconds remains speculative at present. Nevertheless, the progressive development of hypoxia and hypercapnia (Kawakami et al., 1967; Stromme et al., 1970; Angell-James and de Burgh Daly, 1972; Gross et al., 1976; Lin et al., 1983), together with a consistent increase in mean arterial blood pressure (Harding et al., 1965; Lin, 1982) over the duration of the breath-hold, have been recognised as contributory factors in the potentiation of apneic bradycardia. Furthermore, if one considers the variety of factors said to modify the response, it appears likely that a delayed inhibitory effect may occur to a greater or lesser extent, depending on the combination of factors relevant to a given condition. It has been

suggested, for example, that the bradycardial response is accentuated by facial cooling (Kawakami et al., 1967; Le Blanc et al., 1976; Speck and Bruce, 1978; Caputa and Cabanac, 1979), a factor which in this study may help explain the prolonged increase in heart rate during apnea on land compared to underwater. Whereas changes in the temperature to which the face was exposed did not occur between immediate pre-apneic and apneic conditions on land, this was the case underwater. In this environment subjects lowered their faces into the water only at the commencement of apnea. The mean water temperature of 26.1°C ($\pm 0.8^{\circ}\text{C}$) during the apneic condition fell within the range ($15\text{--}30^{\circ}\text{C}$) required to produce cold-receptor excitation (Kawakami et al., 1967). Finally, if the suggestion by Gross et al. (1976), that apneic heart rates represent the domination of vagal influences over tachycardia-producing reflexes is borne in mind, it seems feasible that during exercise at levels which would result in pronounced tachycardia during normal ventilatory conditions on land, an extended delay in that point at which vagal influences predominate may occur.

In the present study the effects of increasing exercise intensity upon the onset of apneic bradycardia were evident, in that bradycardia occurred only at $50\% \dot{V}\text{O}_2$ max on land, and not at all at the higher intensities, and the onset of the response underwater was evident earlier in the breath-hold at $50\% \dot{V}\text{O}_2$ max than at 70 and $90\% \dot{V}\text{O}_2$ max. If the responses in general were considered, the absolute time within which bradycardia commenced ranged between 17.6 and 32.9s. In other words, the bradycardia which Lin (1982) described as occurring within two or three cardiac cycles following respiratory arrest, was not apparent until at least half-way through the total breath-hold duration.

Although Lin's observation concerned primarily resting responses, similar reports exist of an immediate bradycardia during apneic swimming (Strømme et al., 1970) and diving (Irving, 1963; Landsberg, 1975). In each instance the present definition of bradycardia as a drop below a pre-apneic level, was applicable. Closer examination of the results of two of these studies (Strømme et al., 1970; Landsberg, 1975) revealed, however, that this point was not evident until 10s into the breath-hold, an absolute time which in the present study would convert to approximately 28% of the mean breath-hold duration at each exercise intensity underwater. Part of the difficulty in resolving the contention attendant upon the onset of apneic bradycardia is thus largely a problem of semantics. In other words, could the onset of the response be termed "immediate", despite an apparent latency of 10s?

Figures 12 to 14 revealed that once the point was reached where vagal inhibition was sufficient to overcome the initial tachycardia, heart rates declined progressively over the duration of the entire breath-hold. Although this concurs with some previous studies (Magel et al., 1982; Gemma and Wells, 1987), a more common observation is the existence of a plateau once the maximal bradycardial response has been attained. In contrast, the lowest heart rates observed during apnea in the present research were consistently within the final 10% of breath-hold duration; thus termination of bradycardia was synonymous with cessation of apnea. Since the overall range in breath-hold duration on land and underwater was 14 to 55s, these data do not support past suggestions that bradycardia levels-out after approximately 30s (Hong et al., 1967 and 1970; Bergman et al., 1972; Lin et al., 1983; Candler, 1986). Indeed, with the exception of eight of the total 96

breath-holds examined in the present study, the lowest heart rate always occurred within the final three cardiac cycles of the breath-hold. The recording of pneumographic tension during apneic exercise has demonstrated that the chest may expand slightly just before the conventional breakpoint (Craig and Cummings, 1958). It is likely that this constituted part of the involuntary ventilatory activities later described by Lin et al. (1974), and may be of particular relevance to the occurrence of the lowest heart rate prior to the release of the breath, since it would have implications for the effect of mechanical factors upon the heart rate. In addition to the potentiation of the response by lowered intrathoracic pressure and its role upon venous return, stimulation of stretch receptors may be enhanced. On the other hand, reports of increased intrathoracic pressure with increasing chest wall recoil as lung volume increases (Ferrigno et al., 1986), together with attenuated bradycardia in the face of artificially induced expansion of the chest (Aserinsky and DeBias, 1961; Angell-James and de Burgh Daly, 1972) would tend to negate such propositions.

Despite differences in the point at which the onset of bradycardia occurred in the submerged condition, no significant differences existed between the mean heart rates at each exercise intensity. Thus it would appear that despite variable latency in the initiation of the response, depending on how greatly homeostasis was disturbed, the degree to which the heart rate dropped was similar. On land, the mean heart rate at 90% $\dot{V}O_2$ max was significantly higher than at 50%. Nevertheless, when the difference between apneic and non-apneic heart rates at 90 to 100% of exercise duration was compared across conditions, there were no significant differences either between the environments, or the

intensities (Figure 15), suggesting that the extent to which the suppression in heart rate occurred was similar for the three exercise intensities studied. The differences between pre-apneic heart rates and the equivalent controls prior to exercise without breath-holding were not significant either. Furthermore, investigation of the lowest heart rates recorded revealed that despite a trend towards higher heart rates the higher the exercise intensity (Figure 15), the overall extent of the heart rate reduction was not significantly altered between the workloads.

Researchers in the past have reported contradictory results with regard to the effects of exercise intensity upon apneic heart rates. Asmussen and Kristiansson (1968), for example, using cycling exercise with facial immersion only, observed that the heart rate decreased less during heavy than light work. Paulev and Hansen (1972), on the other hand, found that during exercise at varying workloads, apnea elicited a small fall in heart rate, defined by these authors as bradycardia, only in the fittest subjects. In each of these studies, however, workloads were determined in absolute terms. This factor, together with the diversity of protocols used in the past, tends to limit comparisons with the present results. Few studies (Strømme et al., 1970; Oldridge et al., 1978; Finley et al., 1979) appear to have examined the effects of exercise upon apneic heart rates using intensities determined relative to individual capacities. Where this has been the case, comparisons of the responses within the broad spectrum of relative intensities have not been performed.

Based on their observation that only the fittest subjects experienced a fall in heart rate during apneic exercise, Paulev and Hansen concluded that the change in heart rate for each individual was dependent upon the ratio between the absolute work load and the aerobic working capacity of the subject. In other words, the lower this ratio, the greater the reduction in heart rate. Within the range of relative intensities examined in the present study, this was not found to be the case; nor is this view consistent with reports that apnea during facial immersion on land produced a greater drop in heart rate relative to control values during "moderately severe" exercise than during "mild" exercise (Bergman et al., 1972). In contrast to the remarks of Paulev and Hansen, Finley and associates (1979) have suggested that high background heart rates, seen for example, during exercise, may indeed lead to greater vagal activity during apnea (see page 28), and result in greater overall reductions in heart rate. Assuming this to be so, the reduction in heart rate observed during exercise might be expected to be greater, the higher the relative intensity. This was not, in fact, the case in the present study, if, along similar lines to Bergman et al. (1972), the reduction in heart rate which occurred during apnea was compared to the pre-apneic level. Using this method, relative decrements of 30%, 28% and 23% were calculated for the underwater condition at 50, 70 and 90% $\dot{V}O_2$ max respectively. On land the only drop in heart rate below the pre-apneic level, in this case 7%, occurred at 50% $\dot{V}O_2$ max. However, where apneic and non-apneic heart rates at 90 to 100% of breath-hold duration were compared (see Figures 12 to 14), the trend described above was apparent, although the overall differences in heart rate between the different intensities were not significant. The inclusion of suitable

controls in the experiment thus indicated that the theory outlined earlier on the basis of Finley and co-workers' observations, was tenable. At the same time, however, since the increased overall reduction in heart rate was not significant between 50, 70 and 90% $\dot{V}O_2$ max, and since the work of Bergman et al. (1972) inferred that the effect of increased relative exercise intensity would be apparent when markedly different intensities were compared, ("mild" as opposed to "moderately severe"), it is clear that further research is necessary if apneic heart rate responses to the entire continuum of relative exercise intensities are to be fully understood.

Central to the present research was an attempt to standardise as many as possible of those factors said to modify the heart rate response to apnea. In so doing the examination of two primary components was possible: namely apnea and immersion in water.

The comparison of apneic and non-apneic heart rates at the same exercise intensity in both environments appears rarely to have been performed in the past. The inclusion of such controls lead Craig and Medd (1968) to conclude that there was no difference in the heart rate response to exercise with and without breath-holding. In contrast, the same procedure in the present study revealed that apneic heart rates were significantly lower in all conditions, except 70% $\dot{V}O_2$ max on land. Furthermore, the heart rate responses to either condition were qualitatively as well as quantitatively different. Obviously without apnea the sequence of physiological events known collectively as the "dive reflex" would not be possible; whether the cessation of respiratory movement per se is essential to the initiation of the

response (Harding et al., 1965; Hong et al., 1967; Strømme et al., 1970; Olridge et al., 1978; Lin, 1982), does, however, remain largely speculative. Various authors have pointed out that the process initiated by apnea is subjected to modification by various neural, mechanical and chemical factors (Song et al., 1969; Paulev and Hansen, 1972; Lin, 1982). The lack of significant difference seen between the two sets of heart rates at 70% $\dot{V}O_2$ max on land may be explainable in the following terms: Vagal inhibition may have been less effective in the face of the increased workload compared to 50% $\dot{V}O_2$ max, yet the intensity may have been such that over the 31s exercise period the elevation in heart rate was not sufficiently high to result in a significant difference compared to apneic heart rates. The heart rate for 90 to 100% of the exercise period without breath-holding was only 4 $\text{b}\cdot\text{min}^{-1}$ higher than the corresponding value at 50% $\dot{V}O_2$ max. Indeed when the mean heart rate response without apnea for the different intensities on land were compared, no significant difference existed between 50 and 70% $\dot{V}O_2$ max, although Figure 13a indicates that suppression of the apneic heart rates was somewhat less at the higher intensity. In contrast, the non-apneic heart rates at 90% $\dot{V}O_2$ max were significantly higher than at the lower intensities. This elevation in non-apneic heart rates compared to the lower intensities could account for the significant difference between these and apneic heart rates, even in the face of less or delayed vagal inhibition. In addition, the lack of significant difference between apneic and non-apneic heart rates at 70% $\dot{V}O_2$ max on land may have been influenced by the fact that of all the apneic conditions, this was the only case where the immediate pre-apneic heart rate was not significantly higher than the 30s resting

value (converted into beats per minute). In other words, the effects of elevated background heart rates in producing a greater reduction in heart rate, as described by Finley et al. (1979), may not have been apparent.

It is clear therefore, that generalisations regarding apneic bradycardia should be made with reservation. If a multi-factor hypothesis is considered, it is clear that intra- and inter-subject variation is likely, depending on the extent to which modifying factors are present. In the present research, for example, certain factors inherent in the underwater condition, such as hydrostatic pressure, wetness of the skin and enhanced cooling, were inapplicable on land.

Immersion in water, even without apnea, produces a variety of physiological changes (Lin, 1984 and 1988; Greenleaf, 1984). Studies dealing with the effects of immersion upon non-apneic heart rates during exercise are confounded by the variety of activities studied, with the result that conflicting reports do exist. Figures 12 to 14 indicate that the effect of immersion in the present study was to suppress non-apneic heart rates compared to the same exercise intensities on land. Analyses revealed, however, that while this was so, the difference in the mean heart rate between the land and underwater environments was not statistically significant until 90% $\dot{V}O_2$ max, even though the terrestrial and aquatic responses were qualitatively different at all intensities. Studies which have compared the same exercise on land and underwater reported that heart rates underwater were significantly lower for maximal (Dressendorfer et al., 1976) and submaximal (Moore et al., 1970; McArdle et al., 1976) workloads,

although Denison et al. (1972) stated that immersion was associated with a 10% increase in pulse rate at all levels of exercise. Astrand and Rodahl (1977), in a comparison of swimming and running, noted that while maximal heart rates were significantly lower during swimming, submaximal values were of the same magnitude and increased with increasing workload in approximately the same way as during running. Using the same exercise in either environment, a similar trend was found by Moore et al. (1970), who reported that at workloads above light in 16°C water there was a tendency toward lower heart rate per unit work. According to Lin (1988), a lowered heart rate during immersion can be attributed to the elevation of arterial pressure caused by the increased central blood volume, which tends to act against an increase in heart rate elicited by the Bainbridge reflex. It is established that aortic (arterial) pressures increase linearly with the increase in $\dot{V}O_2$ as exercise intensity increases (Astrand and Rodahl, 1977). During exercise on land vasodilation in the working muscles reduces the peripheral resistance to blood flow which accompanies a rise in cardiac output. At water temperatures below 35°C, however, the peripheral vasoconstriction which occurs (Lin, 1984) is likely to produce higher arterial pressures than on land, with a subsequently enhanced baroreceptor-induced drop in heart rate, particularly as exercise intensity increases. Since the increase in central blood volume experienced during immersion would be uniform throughout the exercise intensities, it may be the events described above which resulted in significantly lower heart rates underwater at 90% $\dot{V}O_2$ max compared to lower intensities.

Non-apneic responses to immersion in water thus represent a complex interplay of several opposing factors, all of which were applicable to the apneic state. The inclusion of non-apneic controls in the present study therefore provided a means of determining more accurately the separate effects of immersion and apnea upon the heart rate. The overall heart rate trends which existed for the non-apneic state were reflected during apnea. In other words, although the responses in either environment were qualitatively different, and there was generally a lower mean heart rate underwater than on land, this difference was not statistically significant until 90% $\dot{V}O_2$ max. The factors described above in terms of increased arterial pressures can be applied to the enhanced slowing of the heart rate during apnea at higher intensities underwater compared to on land. The validity of a mechanical theory which places emphasis on the role of systemic arterial baroreceptors in initiating bradycardia has been queried (Song et al., 1969; Gooden et al., 1972; Finley et al., 1979), particularly since bradycardia has been observed in the absence of peripheral vasoconstriction and changes in mean arterial pressure. While these may not be causative factors in producing bradycardia, enhanced stimulation of the baroreceptors is likely to play an important role in the accentuation of the heart rate slowing observed underwater, particularly at high exercise intensities.

According to Song et al. (1969), the potentiation of apneic bradycardia during immersion is attributable to greater stimulation of facial cold receptors. While this explanation alone would not account for the significant difference between heart rates seen at one exercise intensity and not others, its effects upon heart rate responses underwater cannot be discounted. Since the water temperature in the

present research (26.1°C) was just outside the range ($10\text{--}25^{\circ}\text{C}$) described as most efficient in producing bradycardia (Strømme et al., 1970; Caputa and Cabanac, 1979), its effects could not be separated from those of immersion per se. Together with those factors discussed earlier, enhanced cooling in the underwater environment must, therefore, be considered in relation to heart rate responses with and without breath-holding. That the lowest heart rates recorded during apnea were significantly lower underwater than on land must be seen in this context: Heart rate responses in general, although only significant at $90\% \dot{V}\text{O}_2 \text{ max}$, were lower in the underwater environment. Furthermore, in light of the fact that no significant difference existed between the environmental conditions when the difference in heart rate at 90 to 100% with and without breath-holding was examined, it would appear that although in absolute terms the heart rate dropped to a lower level underwater, the relative decrease (ie, apnea compared to without apnea at 90 to 100% duration) was the same in either environment. This was substantiated by the fact that the differences between pre-apneic heart rates and the equivalent controls prior to exercise without breath-holding (Figures 12 to 14) were not significantly altered in the different environments.

Some studies in the past (Strømme et al., 1970; Speck and Bruce, 1978) have failed to take cognisance of changes in heart rate immediately prior to apnea and have used as a control a resting value obtained some time before the performance of apneic manoeuvres. The heart rate is extremely responsive to a variety of factors. Thus, unless an immediate pre-apneic level is recorded for comparison, misleading

reports, probably of less of a reduction in heart rate than might otherwise be the case, are possible.

It has been argued, however, that failure to recognise the influence of high control heart rates prior to breath-holding has given rise to substantial reports of bradycardia in response to breath-holding or facial immersion (Magel et al., 1982). In the present study subjects rested between apneic manoeuvres until the heart rate returned to within 10 beats of the reference resting value. Nevertheless, in both environments an anticipatory increase in heart rate similar to that observed by other authors (Strømme et al., 1970; Hong et al., 1970; Bergman et al., 1972; Folinsbee, 1974; Magel et al., 1982; Hayward et al., 1984) was consistently detected as subjects were told to prepare for a test manoeuvre. Higher pre-apneic control levels underwater were attributed to a greater general state of excitation and anticipation than was the case on land. After reviewing the literature, Lin (1988) reported that pre-dive heart rates in the open sea tended to be higher than the resting value in air. In light of the fact that the heart rate is so volatile, high heart rates immediately prior to breath-holding cannot be discounted in favour of a resting value recorded some time earlier, or a control heart rate established over a prolonged time period. Magel and associates (1982), for example, used as a control the mean heart rate recorded over a two minute adjustment period before each manoeuvre. A mean heart rate over 120s would, however, mask the heart rate response as subjects prepared for a breath-hold. In the present study once the heart rate was within 10 beats of the reference resting value the subject was told to commence the breath-hold when he was ready. In many cases the anticipatory

elevation in heart rate was evident for the remainder of the pre-apneic period. Although the use of a mean heart rate over 30s would also mask beat-by-beat responses, it was felt that this would provide a more accurate indication of the heart rate prior to breath-holding than a mean taken over a longer time period.

Breath-Hold Duration

It is generally understood that there is considerable variation in the duration of apnea both within and between individuals, with psychological factors having been recognised as the major determinants of the conventional breaking point (Lin et al., 1974; Lin, 1982; Hickey and Lundgren, 1984; Hentsch and Ulmer, 1984). Such variation was evident in the present data, where the breath-hold duration on land ranged from 14 to 50s ($\bar{x} = 31.7 \pm 8.0s$) and underwater from 17 to 55s ($\bar{x} = 34.7 \pm 10.3s$). This, together with the fact that where breath-hold duration during exercise has been reported, the intensity of the exercise is often unquantified, and that additional factors such as lung volume or water temperature influence duration, renders comparison with previous data of little value. Similar durations to those underwater in the present study, for example, have been reported during synchronised swimming (Gemma and Wells, 1987) and diving (Shiraki et al., 1985), where the work intensities may have been very different.

The bulk of the evidence suggests that apneic duration at rest exceeds that during exercise, with oxygen availability cited as the major underlying factor. Breath-hold duration has been shown to be correlated with the reciprocal of $\dot{V}O_2$ (Craig and Cummings, 1958; Lin et al., 1974; Lin, 1982). If this is so, the expected outcome of

increasing work intensity as in the present research, would be a decrease in breath-hold duration. While analysis established that the mean breath-hold durations on land and underwater were not significantly different, Figure 16 reveals that a decrease in duration did occur with increasing exercise intensity on land, but not underwater, with the result that overall, there was no significant difference in the duration of apnea between the conditions. The effect of exercise upon breath-holding time may, however, be more complex than an inverse relation to exercise intensity (Craig and Cummings, 1958) or $\dot{V}O_2$ (Lin, 1982). Candler (1986), in a comparison of breath-holds underwater while resting, reported that while each was significantly lower than a resting breath-hold on land, the underwater apneic conditions were all of similar durations. Shortening of the resting breath-hold during immersion was attributed to the influence of feelings of "psychological discomfort" experienced by the subjects while resting underwater. It is possible that similar psychological factors were at play during cycling exercise, particularly underwater. Previous authors have stated that distractions due to physical movement and conscious effort in performing a physical task may defer breaking point. In considering reports of adolescent competitive swimmers, predominantly breast-strokers, who have lost consciousness during swim meets, Hickey and Lundgren (1984) have proposed that these young swimmers neglected to breathe because of excitement and intense concentration and succumbed to hypoxia. It is possible, therefore, that the performance of an unusual activity (ie. cycling) in the apneic state, particularly in the underwater environment, may have had a distracting effect, with the result that overall, no change in the duration of apnea occurred between

the conditions. Interestingly, a number of the present subjects commented that during exercise on land they were more aware of factors such as thermal and respiratory discomfort than underwater, which tended to impair performance.

Since the performance of apneic exercise at 50, 70 and 90% $\dot{V}O_2$ max was randomised, further statistical analysis examined the effect that order of performance had upon the total duration of apnea. Investigations of the effects of successive breath-holds upon duration at rest have established the short-term trainability of breath-holding time (Hentsch and Ulmer, 1984). Increases of 160% were observed by these authors over five successive breath-holds underwater with three-minute intervals. The present results did not, however, demonstrate a short-term training effect, since the duration of apnea was not significantly influenced by the order of performance. According to Hentsch and Ulmer, the prolongation of apnea at rest over successive tests could be explained by two processes: (1) an increase in the time span without respiratory movements, caused probably by unconscious hyperventilation, and (2) an increase in the time span with respiratory movements through psychological adaptation. They suggest that hyperventilation may have been due to the production of lactate during breath-holding but, of more importance to the prolongation of apnea with successive trials, rapid repetition may have resulted in the metabolically produced ventilatory increase being transformed into an unconscious hyperventilation prior to the subsequent breath-hold. In the present study, however, subjects were allowed sufficient time between tests to recover fully. The next condition was conducted only

when the heart rate was within 10 beats of the initial resting value and the subject felt ready to continue. Since deliberate hyperventilation before breath-holding was proscribed, an elevated ventilation rate prior to apnea would have been noted. Thus a decrease in PCO_2 below the normal level prior to apnea was unlikely. It is possible that psychological adaptation, described by Hentsch and Ulmer as resulting in an improved ability to endure "the unpleasant feeling of the need to breathe", may occur similarly during exercise and at rest. The results indicate, however, that if this did occur, it was ineffectual in prolonging apnea; indeed, the ability to overcome discomfort due to hypoxia, hypercapnia and acidosis may be compromised more during exercise than at rest. Clearly the trainability of breath-holding time during exercise requires further clarification.

Several past researchers, when investigating the effects of apnea upon heart rates, have used predetermined breath-holding times, such as 15s (Paulev and Hansen, 1972), 30s (Magel et al., 1982; Arnold, 1985), 45s (Harding et al., 1965), or 60s (Asmussen and Kristiansson, 1968). The results of the present research suggest that particularly where relatively short periods are chosen, a set breath-hold duration may not be optimal in producing the lowest heart rate possible for a given apneic period. Figure 11 indicates that with overall mean breath-hold durations of 31.7s (land) and 34.7s (underwater), the heart rate continued to fall for the duration of apnea. Indeed, the lowest heart rates in either environment, for each of the exercise intensities were consistently recorded just prior to the cessation of apnea. Based on the results from relatively few subjects, Asmussen and Kristiansson (1968) found that the heart rate decreased less during heavy work than

during light work, prompting them to conclude that the degree of bradycardia was limited by breath-hold duration at heavier workloads, while Strømme et al. (1970) stated that breath-hold duration during exercise may be too short to allow a conclusion as to the relation between it and the degree of bradycardia. In the present research, breath-hold duration was not found to be significantly altered with varying exercise intensities. Nor, however, was there a significant difference between either the lowest heart rates, or the difference between apneic and non-apneic heart rates at 90 to 100% of duration. While the duration of apnea may be related to heart rate in the sense that had the breath been held for 50% of the maximal apnea recorded, higher heart rates than at 100% would have been measured, a clear-cut relationship between maximal breath-holding capacity and the degree of bradycardia was not possible.

Ratings of Perceived Exertion (RPE)

This study examined the perception of effort during cycling work at 50, 70 and 90% $\dot{V}O_2$ max, on land and underwater. It was established that effort perception was not significantly altered from one environment to the next. This was useful in confirming that subjects were indeed experienced and comfortable in the underwater environment. It has been shown that psychological stress during immersion, especially in less experienced divers, may significantly influence motor performance and perception (de Mojà et al., 1987; Griffiths et al., 1987). These results indicated that subjects perceived the tasks as equally demanding in either environment.

Ratings of perceived exertion for apneic exercise were significantly higher than for the equivalent exercise in the non-apneic state (Figure 17). Investigations of subjective reactions to apneic exercise represent a unique challenge, since many of the observed relationships between RPE and psychological parameters such as heart rate or ventilation would appear to be altered during breath-holding. The RPE scale was originally designed to follow the heart rate response to increased exercise intensity (Carton and Rhodes, 1985a). This relationship was reflected in the non-apneic state (Figure 17), where RPE increased significantly with increasing exercise intensity. Similar results, however, existed for the apneic state: RPE increased significantly with increasing exercise intensity, with the exception of between 70 and 90% $\dot{V}O_2$ max (Figure 17). In light of the slowing of the pulse which occurred in each condition, the conventional relationships between exercise intensity, heart rate and RPE are no longer tenable. Several factors other than heart rate are, however, involved in the perception of effort, the relative contributions of which may vary according to the nature of the task. For example, it has been noted that RPE increases according to a positively accelerating function which closely approximates minute ventilation ($\dot{V}_e$) and blood lactate response curves to metabolic demand (Carton and Rhodes, 1985a). The influence of these factors will differ between the apneic and non-apneic state. Obviously, $\dot{V}_e$ is not applicable to the apneic state, while the impact of blood lactate concentrations upon RPE may be increased as a progressively hypoxic and hypercapnic state is incurred during apnea, although the mean breath-hold durations on land and underwater (31.7 and 34.7s respectively) would imply that the anaerobic energy yield was

pronounced during both apneic and non-apneic exercise of the equivalent length of time. According to Carton and Rhodes, a substantial role in effort perception may be played by kinesthetic or proprioceptive feedback. While it could be expected that the degree of strain experienced in the active musculature would not differ significantly between the apneic and non-apneic conditions, the role of sensory receptors located in the chest wall, lungs and airways which respond to changes in lung volume, lung pressure and respiratory tension (Robertson, 1982) may be of substantial importance during apnea. In addition, the onset of involuntary respiratory movements during breath-holding is accompanied by what is described as "discomfort and an unpleasant sensation to breathe" (Lin, 1982; Hentsch and Ulmer, 1984), or "air hunger" (Lin, 1988). Clearly these sensations are consciously monitored by the subject and as such may serve as additional prominent cues for rated perceived exertion.

The ratings of perceived exertion obtained from the present subjects at 50, 70 and 90% $\dot{V}O_2$ max were (to the nearest whole number) 10, 11 and 12 respectively for exercise without apnea, and 12, 14 and 14 respectively for exercise with apnea. On the rating scale (see Appendix A) a value of 10 falls between classifications of "very light" and "fairly light", with 14 between "somewhat hard" and "hard". Based on data modified from several sources, Robertson (1982) presented RPE over a range of relative oxygen uptakes during cycling exercise. Values for 50% $\dot{V}O_2$ max varied between 9 and 11; for 70% $\dot{V}O_2$ max between 13 and 14; and for 90% $\dot{V}O_2$ max between 17 and 19. In light of the relative intensities at which the subjects in the present study worked, the ratings were thus

lower than might have been expected, especially at the heavier workloads.

If one considers the heart rate response to exercise without apnea in either environment, maximal values of 114, 118 and 128 b.min⁻¹ were recorded on land, and of 102, 107 and 112 b.min⁻¹ underwater at 50, 70 and 90% $\dot{V}O_2$ max respectively. In terms of the traditional classification of workload on the basis of heart rate response, each of the tasks on land, and the most strenuous workload underwater would be classified as "heavy", while the lighter workloads underwater would both be termed "moderate" (Astrand and Rodahl, 1977). It is important to note, however, that traditional assessment of the severity of the workloads along these lines is typically based upon prolonged physical activity. De Vries (1980) points out that during heavy exercise the heart rate increases until exhaustion is reached. Even at light to moderate workloads a plateau in heart rate may be seen only after 30 to 60s. The duration of the present tasks ($\bar{x}$ = 31.7s on land and 34.7s underwater) may therefore render classification of the workload in terms of heart rate inaccurate.

The duration of the tasks, controlled by breath-hold duration, may have had a similar influence upon RPE. The majority of the research which has examined the relationship between RPE and exercise duration appears to have focussed upon prolonged activity, where, it has been suggested, sustained elevations in RPE may be due to increases in ventilation over time. According to Robertson (1982), local factors are assumed to provide the primary sensory signals for the perception of effort, with central factors potentiating local signals in proportion to the aerobic

metabolic demand. However, central factors begin their potentiating input approximately 30 to 180s after the initiation of exercise, the time period corresponding to that required for cardiovascular and ventilatory adaptation. Thus central factors may have played a subordinate role in determining the overall RPE obtained in the present study. In addition, it is possible that local signals over a mean duration of 31.7 to 34.7s were insufficiently high to elevate RPE values to similar levels as those obtained at similar work intensities, but over a longer period of time, where both local and central cues would contribute to higher overall RPE values. Finally, the influence of factors such as personality and social learning upon the values obtained cannot be discounted. In general, the present subjects were subjectively perceived as very competitive and anxious to perform well in the given tasks. It is possible that lower than expected ratings of perceived exertion could be explained in part as a psychological suppression in values.

Effect of Lung Volume

Statistical comparison of Group A (large relative lung volume) and Group B (small relative lung volume) established that factors known to modify the heart rate response to apnea, such as age, underwater experience, $\dot{V}O_2$ max, environmental conditions to which they were exposed or pre-apneic heart rates, were uniform between the groups.

Comparison of the heart rate responses to apneic exercise at 50% $\dot{V}O_2$ max revealed that despite a significantly longer breath-hold duration for Group A, neither the mean apneic heart rate nor the lowest heart rate recorded was significantly different between the groups. Furthermore,

the heart rate responses of the two groups over 10% increments of breath-hold duration were qualitatively the same. Although not significantly different to those of Group A, there was a tendency towards lower heart rates in Group B. Figure 18 demonstrates that compared to pre-apneic levels, the reduction in heart rate was 0.7% greater for Group B than Group A on land, and 9.6% greater underwater.

With respect to the relationships between lung volume, breath-hold duration and bradycardia, these results appear contrary to those of several previous authors. Scott and Candler (in press), using a similar method to differentiate between groups as in the present study, reported that while lung volume was not a major criterion of breath-hold duration, it did appear to be a determinant of the magnitude of the bradycardial adaptation to apnea. While these authors found significantly lower heart rates for females with large lung volumes they did not, however, report stringent control of factors such as underwater experience, physical conditioning or environmental conditions between the groups. In direct contrast, the present results indicate that while lung volume did not significantly affect the apneic heart rate response, it did appear to influence breath-hold duration. Large lung volume, together with conscious movement of the respiratory muscles, may be important in increasing the tolerance for hypercapnia and hypoxia (Hickey and Lungren, 1984). Since in the present study vital capacity was expressed relative to body surface area, the effects of body size in influencing lung volume were cancelled out. Comparatively larger lung volumes for Group A would have been likely to lead to increased lung oxygen stores, greater carbon dioxide dilution and the retardation of the deflation reflex (Hickey and Lungren, 1984)

compared to Group B. Where Scott and Candler discounted the role of breath-hold duration and instead attributed lower heart rates in a group with larger lung volumes to increased venous return and enhanced stimulation of the baroreceptors, others have directly attributed greater bradycardia evident in experienced divers to their longer breath-holding ability (Hong et al., 1970; Oldridge et al., 1978). A greater breath-holding capacity noted in experienced divers has been ascribed to changes in lung volumes as well as a tolerance to increased carbon dioxide tensions (Schaefer, 1965). The results of the present study, in terms of both the overall findings and those within the lung size groups, suggest that neither the duration of apnea, nor lung size had a significant effect upon the heart rate. Clearly, further rigorous research, where factors said to modify the apneic heart rate are strictly controlled, is necessary if the relationships between lung volume, breath-hold duration and apneic heart rates are to be elucidated.

The trends in heart rate response to apneic exercise described for the overall group were evident within each of the lung size groups. In other words, the commencement of exercise resulted in a transient increase in heart rate until the inhibitory effects of apnea became apparent in a consistent decrease in heart rate over the remainder of the breath-hold (Figure 18). In both groups the onset of bradycardia was delayed on land compared to underwater; although this would contribute towards lower maximal bradycardial values in the latter environment, the difference between the lowest heart rates in either environment was not statistically significant.

Despite a longer mean breath-hold duration for one group compared to the other, the commencement of bradycardia in both environments, when expressed relative to total breath-hold duration (Figure 18), occurred at identical points for groups A and B. The mean durations of apnea on land and underwater were, for Group A, 34.7 ± 5.9 s and 40.5 ± 6.7 s respectively, and for Group B, 30.3 ± 7.0 s and 28.6 ± 3.9 s respectively. On land, the decrease in heart rate below the pre-apneic level occurred between 90 and 100% of the apneic period. In absolute terms, this corresponded to between 31.3 and 34.7s for Group A, and between 27.3 and 30.3s for Group B. Thus in absolute terms, the onset of bradycardia occurred much faster for those with small relative vital capacities, than for those with comparatively larger lung volumes. The same trend applied to the underwater condition, where the onset of bradycardia was between 60 and 70% of total breath-hold duration for each group. Once again, therefore, the onset of bradycardia as such was faster in absolute terms for Group B (between 17.2 and 20s) than Group A (between 24.3 and 28.3s). Considered in conjunction with the mean heart rate response of the groups in both environments, together with the lowest heart rates recorded, it would appear, however, that the marked difference in absolute times for the onset of bradycardia between the two groups did not significantly influence the degree to which the response occurred over the duration of apnea. The statement made earlier with regard to the extent of the relationship between breath-hold duration and bradycardia is thus reiterated. The data from both lung size groups suggest that provided the maximum duration of apnea is elicited, and modifying factors are controlled as rigorously as

possible between the samples, the absolute duration of apnea may not significantly affect the extent of the bradycardial response.

Furthermore, it would appear that the onset of bradycardia is more closely related to varying proportions of total duration depending on the conditions, than to absolute time. This may have important implications with regard to previously inferred relationships between variables such as underwater apneic experience, lung volumes, breath-hold duration and bradycardia. The present data indicate that where modifying factors such as underwater experience remained uniform between the lung size groups, neither lung size, nor breath-hold duration significantly affected apneic bradycardia. The onset of bradycardia, however, occurred within identical relative proportions of apneic duration. The temporal development of the response in relative terms thus warrants further investigation. Lin et al. (1983), for example, have reported that hypoxia contributes substantially to the development and maintenance of breath-hold bradycardia, while hypercapnia attenuates the same response. It is possible, therefore, that a progressively hypoxic state during apnea may be of particular importance to the development and onset of apneic bradycardia when expressed relative to total breath-hold duration. In other words, are critical levels of PO_2 more important to the onset of bradycardia than previously assumed, and if so, do they occur within specific proportions of the apneic period?

CHAPTER 5

SUMMARY, CONCLUSIONS AND RECOMMENDATIONS

Several investigators have studied the heart-rate response to apnea, immersion in water and apnea during immersion, with largely conflicting results. Previous studies have, however, tended to demonstrate a lack of convention with respect to the evaluation of bradycardia under such conditions. Adopting an holistic approach, the present research examined the phenomenon of apneic bradycardia in physiological and psychophysical terms, during work on land and underwater.

The study aimed at establishing representative heart rate responses to apneic work in an attempt to reappraise current equivocal theories regarding the onset, course and termination of the bradycardial response. Breath-hold duration itself was examined in order to facilitate the investigation of apneic bradycardia in terms of both absolute time and as a percentage of total breath-hold time. Furthermore, subjective reactions to apnea were studied in an attempt to achieve an integration between physiological and psychological concomitants of apneic bradycardia. A final aim of the present research was to further elucidate the effects of relative lung volumes upon apneic heart-rate responses by means of stricter control measures than have been evident in past research.

HYPOTHESES

It was hypothesised that:

1. There is no change in terrestrial or aquatic heart-rate responses to quantified exercise, with and without apnea.
2. Breath-hold duration is not altered at differing work intensities under terrestrial and aquatic conditions.
3. There is no difference in terrestrial and aquatic RPE at quantified workloads, with and without apnea.
4. The terrestrial and aquatic apneic heart-rate responses of subjects with large and those with small relative lung volumes do not differ.

SUMMARY OF PROCEDURES

In the past, few studies have recognised the necessity for the performance of the same quantified exercise on land and underwater in order to enable accurate comparisons of physiological and psychological responses to apnea between the environments. Typically, cardiopulmonary changes during breath-hold diving are generalised from laboratory-conducted studies, often incorporating facial immersion only. An initial phase of this study thus involved modification of the cycle ergometer used to study heart rate responses to apneic exercise on land, for use during immersion in water. While it was recognised that the effects of the additional resistance offered by the water confounded the precise determination of power output underwater, the use of the same cycle ergometer as in the land test allowed for the quantification of workload relative to each individual's maximal capacity in that environment.

A pilot testing protocol was then designed to examine the heart rate response, in both environments, to apneic exercise at 10% increments from 20 to 100% of the maximal power output achieved on the cycle ergometer during a continuous, progressive incremental test of maximal oxygen consumption ($\dot{V}O_2$ max). This was conducted in order to determine the relative intensities at which subjects would exercise during the main data collection phase on land and underwater. Ten male subjects participated in this phase of testing on land, with five of these repeating the procedure underwater. In one test period heart-rate responses were recorded on an electrocardiograph (ECG) for both breath-hold and non-breath-hold conditions, for each intensity of exercise studied. Based on these results, exercise intensities to be studied during the actual experiment were established, to represent "moderate" to "very heavy" ranges of workload. The use of maximum power output as the criterion by which relative exercise intensities were determined was, however, decided against, since the same power output may have been accomplished by different individuals at different oxygen consumptions. Thus, using the above results as a guideline, responses to 50, 70 and 90% of the "situation-limited" functional $\dot{V}O_2$ max attained in each environment were studied.

Further pilot testing assessed the reliability of procedures used to measure $\dot{V}O_2$ max underwater. Ten male subjects underwent a land test of $\dot{V}O_2$ max, while eight participated in the underwater test. For both conditions, the test was readministered to three subjects within one week of the initial test. The $\dot{V}O_2$ max tests, in both environments, involved pedalling against a starting resistance of 60W (1kg) at 60rpm. The duration of each bout was two minutes, with an increase in the

workload of 60W at the end of the second and fourth minutes. Thereafter the load was increased by 30W (0.5kg) at the end of each bout until exhaustion was reached.

The test/retest reproducibility of this method was apparent from the statistical analyses performed on the pilot study data. Group regression equations calculated using land and underwater pilot study $\dot{V}O_2$ data established the suitability of a one independent variable regression ($\dot{V}O_2$ against time) in determining the exact time from the commencement of the test for a given percentage of $\dot{V}O_2$ max, and subsequently the resistance load at that time. In the face of practical difficulties experienced during the underwater data collection sessions performed in situ, the validity of using only the last three gas bags collected in order to calculate individual regression equations was established.

Sixteen healthy young males, none with less than one year's experience in underwater apneic activities such as spearfishing or underwater hockey, volunteered to participate in this research. Data collection involved four separate testing sessions. At the first, in the laboratory, general information, including stature and mass, was obtained, and vital capacity measured. The subject then performed a $\dot{V}O_2$ max test using the protocol outlined above. On the second occasion in the laboratory he was asked to exercise on the cycle ergometer at 50, 70 and 90% $\dot{V}O_2$ max. The order of presentation of these conditions was randomly determined. However, each apneic bout at a given intensity was followed by performance of the same workload for the equivalent length of time, but without apnea. At the beginning of the session the resting

heart rate was recorded over 30s on an ECG. After each exercise period the subject was asked to rest quietly until the heart rate had dropped to within $10\text{b}\cdot\text{min}^{-1}$ of this initial rate. During the performance of each condition heart rate was continuously monitored to provide pre-apneic, apneic and recovery heart rates, together with their non-apneic equivalents. Ratings of perceived exertion (RPE) were collected after every condition.

Since all subjects were experienced in underwater activities, a short habituation period immediately prior to the first underwater testing session was considered sufficient to familiarise subjects with the underwater testing environment. It was determined that despite the additional pressure required to expand the thoracic cavity underwater, the maintenance of adequate pulmonary ventilation was possible. The first session underwater involved the performance of a $\dot{V}O_2$ max test, using the identical protocol to that used on land. On the final occasion, heart rate and RPE responses to apneic and non-apneic work were once again monitored in exactly the same manner as on land.

The results were analysed using the related Student's t-test to establish reliability of pilot study data collection and to compare pre-apneic heart rates to the 30s resting value for all subjects. The independent Student's t-test was used to compare the parameters relevant to Group A (large relative lung volumes) to those of Group B (small relative lung volumes). One-factor repeated measures analyses of variance were applied to heart rate data over 10% increments of the total exercise duration for apneic and non-apneic land and underwater conditions, and two-factor repeated measures analyses of variance were

applied to assess the effects of land and underwater conditions at the relative intensities studied upon heart rate, breath-hold duration and RPE. The 0.05 level of significance was chosen.

SUMMARY OF RESULTS

1. Cycling while totally submerged in water resulted in a significant decrease in $\dot{V}O_2$ max from values obtained on land. Despite the confounding effects of water upon the resistance load on the cycle ergometer, together with the additional resistance offered by the water to pedalling, the $\dot{V}O_2$ max obtained underwater represented a "situation-limited" functional maximum. Heart rate responses could thus be examined in terms of exercise intensities relative to the $\dot{V}O_2$ max attained for those unique conditions.
2. In light of the confusion apparent in past research, an operational definition of apneic bradycardia was established, as a reduction in heart rate below the immediate pre-apneic level. In terms of this definition, apneic exercise in general elicited a definite bradycardia underwater, but failed to do so on land. Rather, the collective response on land was what could be termed a "suppressed tachycardia"; despite a reduction in heart rate after an initial rise, the mean heart rate did not drop below the pre-apneic control level.
3. Separate analyses of apneic and non-apneic responses, on land and underwater, for each of the three relative exercise intensities studied, revealed, however, that apneic bradycardia was in fact apparent at 50% $\dot{V}O_2$ max on land, although not at the higher

workloads. Even where bradycardia did occur, vagal inhibition was insufficiently strong to overcome an initial increase in heart rate; thus the onset-point occurred after a latency of some seconds. The effects of increasing exercise intensity upon the onset of apneic bradycardia were evident, in that the onset was apparent earlier in the breath-hold at 50% $\dot{V}O_2$ max underwater than at 70 and 90% $\dot{V}O_2$ max, and only occurred at the moderate workload on land. In contrast to past observations of a plateau once the maximum bradycardial response was attained, the present research found heart rates declined progressively over the duration of the entire breath-hold. Termination of bradycardia was synonymous with cessation of apnea.

4. Despite differences in the point at which the onset of bradycardia occurred in the submerged condition, no significant differences existed between the mean heart rates at each exercise intensity. On land the mean heart rate at 90% $\dot{V}O_2$ max was significantly higher than at 50%. However, when the difference between apneic and non-apneic heart rates at 90-100% of exercise duration was compared across conditions, there were no significant differences, suggesting that the extent to which the suppression in heart rate occurred was similar for the three exercise intensities studied. The differences between pre-apneic heart rates and the equivalent controls prior to exercise without breath-holding were not significant either. Furthermore, investigations of the lowest heart rates recorded revealed that despite a trend towards higher heart rates at higher exercise intensities, the overall extent of the bradycardia was not significantly altered between the workloads.

5. The comparison of apneic and non-apneic heart rates at the same exercise intensity in either environment revealed that apneic heart rates were significantly lower in all conditions, except 70% $\dot{V}O_2$ max on land. Heart rate responses to either condition were qualitatively as well as quantitatively different.
6. The effect of immersion was to suppress non-apneic heart rates compared to the same exercise intensities on land. However, despite qualitative differences between terrestrial and aquatic responses at all intensities, the difference in mean heart rate between the environments was not statistically significant until 90% $\dot{V}O_2$ max. This overall trend in mean responses was reflected during apnea. The lowest heart rates recorded during apnea were significantly lower underwater than on land; however no significant difference existed between the environmental conditions when the difference in heart rate at 90-100% with and without breath-holding was examined. Thus it would appear that although in absolute terms the heart rate dropped to a lower level underwater, the relative decrease was the same in either environment.
7. Breath-hold duration was not significantly altered by either environment or exercise intensity, nor was a short-term training effect over successive breath-holds, as described by previous authors, evident, since the order in which randomised conditions were presented had no effect upon the duration of apnea. While a clear-cut conclusion regarding the relationship between maximal breath-holding capacity and the degree of bradycardia was not possible, the results suggested that the use of a set breath-hold

duration, as practised by several previous authors, may not be optimal in producing the lowest heart rate possible for a given apneic period.

8. Ratings of perceived exertion were not significantly altered from one environment to the next, but were significantly higher for apneic exercise than for the equivalent exercise in the non-apneic state. Furthermore, RPE increased significantly with increasing exercise intensity in both apneic and non-apneic conditions.
9. Statistical comparison of Groups A (large relative lung volume) and B (small relative lung volume) established that factors known to modify the heart rate response to apnea were uniform between the groups. Comparison of the heart rate responses to apneic exercise at 50% $\dot{V}O_2$ max revealed that despite a significantly longer breath-hold duration for Group A, neither the mean apneic heart rate, nor the lowest heart rate recorded was significantly different between the groups. Furthermore, the heart rate responses of the two groups over 10% increments of breath-hold duration were qualitatively the same. In absolute terms, the onset of bradycardia occurred earlier for those with small relative vital capacities than for those with comparatively larger lung volumes. When expressed relative to total breath-hold duration, however, the commencement of bradycardia in both environments occurred at identical points for Groups A and B.

CONCLUSIONS

This study confirmed that despite an elevation in metabolic rate during the performance of exercise, the heart rate was suppressed during apnea, to the extent that bradycardia occurred at workloads classified as

moderate to very heavy underwater, and at the moderate workload on land. The inclusion of suitable controls, in the form of heart rates recorded immediately prior to exercise and non-apneic heart rate responses to equivalent workloads, together with the quantification of the workload in either environment, provided for a more thorough and complete investigation of apneic bradycardia than has apparently been the case.

In recognition of the importance of an integrative, holistic approach to the subject, related variables such as breath-hold duration, RPE or lung volume were studied within the context of the strict control of as many as possible of those factors said to modify the heart rate response to apnea. Although clear-cut conclusions regarding the relationship between maximal breath-holding capacity and the degree of bradycardia were not drawn, it appeared that breath-hold duration did not significantly effect the apneic heart rate response. Nor was the apneic heart rate response altered by differences in vital capacity, although these did appear to influence the duration of apnea. Although the underlying mechanisms remain speculative at present, this study further established that the subjective perception of effort during exercise on land and underwater was significantly higher for the apneic compared to non-apneic state, even among experienced underwater exponents.

HYPOTHESES

Based on these results, tentative acceptance or rejection of the null hypotheses ($p < 0.05$) was as follows:

1. The null hypothesis was in this case rejected in favour of the alternative hypothesis:

There is a significant change in terrestrial and aquatic heart rate response under apneic and normal respiratory conditions, during differing work intensities expressed relative to $\dot{V}O_2$ max.

2. The null hypothesis was in this case tentatively accepted:

There is no difference in breath-hold duration for subjects working at differing intensities in terrestrial and aquatic conditions.

3. The null hypothesis was in this case rejected in favour of the alternative hypothesis:

There is a significant difference in the terrestrial and aquatic rating of perceived exertion under apneic and normal respiratory conditions, during work at differing intensities.

4. The null hypothesis was in this case tentatively retained:

The terrestrial and aquatic apneic heart rate responses of subjects with large relative lung volumes do not differ from those of subjects with small relative lung volumes at varying work intensities.

RECOMMENDATIONS

It is imperative that future studies examine apneic bradycardia within the framework of an entire network of causation. Thus, along similar

lines to the present study, attempts must be made to standardise as many as possible of the factors considered as being influential to apneic heart rates. While still allowing for the systematic examination of individual variables, this may result in a greater degree of uniformity in the interpretation of the obtained results. A lack of such uniformity with regard to both the acquisition and interpretation of the relevant data has hindered comparisons between studies in the past.

Few previous studies appear to have examined the effects of exercise upon apneic heart rates using intensities determined relative to individual capabilities. Since the present study examined heart rate responses to exercise at 50, 70, and 90% $\dot{V}O_2$ max, further research expanding this range of workloads is necessary if apneic heart rate responses to the entire continuum of exercise intensities are to be fully understood.

The relationship between apneic heart rates and breath-hold duration requires further clarification. The results of the present study indicated, that although marked differences in absolute time to the onset of bradycardia existed between groups, the onset-point was identical when expressed relative to total breath-hold duration. Explication of the exact nature of this relationship using larger numbers of subjects and wider ranges of exercise intensity would be useful. Using more sophisticated technology than was possible in the present study, future researchers would do well to examine the effects that PO_2 and PCO_2 levels might have upon the onset of bradycardia when expressed relative to total breath-hold duration.

The trainability of breath-hold duration itself warrants further attention. While the short-term trainability of breath-hold duration at rest has been established (Hentsch and Ulmer, 1984) the present results indicated that no such effect was apparent during exercise. Once again, however, a wider range of exercise intensities, together with a greater number of successive breath-holds at the same intensity, should be studied before conclusive statements can be made.

Finally, the subjective perception of effort in the apneic state, both at rest and during exercise on land and underwater, poses a unique challenge. The underlying mechanisms behind the elevated ratings of perceived exertion for the apneic compared to non-apneic state in the present study remain to be further elucidated. Further study is also required to clarify the relative contributions of central and local cues in the apneic state.

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APPENDIX A

Pre-Test Information to Subjects

Informed Consent

RPE Instruction

RPE Scale

RHODES UNIVERSITY
DEPARTMENT OF HUMAN MOVEMENT
STUDIES

THESIS: Apneic bradycardia: Terrestrial and aquatic responses in man under working conditions.

GENERAL: The purpose of this study is to clarify some of the discrepancies that exist in the literature with regard to heart rate responses to breath-holding, or apnea. Some have reported a slowing of the heart rate, or bradycardia, while others have observed an increase in heart rate, or tachycardia. However, a wide variety of research protocols have been used, many of which have failed to take account of a number of modifying factors, thereby largely invalidating a comparison of the obtained results. This study will aim at establishing representative heart rate responses to apnea at rest and during work, both on land and underwater. In addition, an attempt will be made to determine the effects of relative lung volumes upon the heart rate response, and the effect that apneic responses in general have upon the subjective perception of effort, as measured by Borg's RPE scale.

PROCEDURE: Your participation in this study is entirely voluntary and you are reminded that you may withdraw at any stage prior to or during the research.

Should you volunteer your services as a subject, you will be required to participate in the following testing sessions. You are requested to wear clothing that will facilitate collection of the data (eg: shorts and t-shirt, or swimming costume). Furthermore, you are asked to refrain from energetic physical activity, eating, drinking any substance containing caffeine, or smoking for at least one hour prior to

each test.

SESSION A: Duration approximately 45 minutes.

1. General information, such as age and years of experience in underwater apneic activities, will be obtained.

2. Stature and mass will be measured.

3. Lung volume measurement. You will be asked to breathe into a mouthpiece for a short period of time in order to measure vital capacity (lung volume) on a Godart Expirograph.

4. Maximal oxygen consumption ($\dot{V}O_2$ max).

The protocol used will be a direct, continuous cycle ergometer test. You will begin pedalling at a given rate against a starting resistance of 1kg (60W). This load will be increased after every two minutes by 1kg until the end of the sixth minute and thereafter by 0.5kg until exhaustion is reached. You will breathe into a mouthpiece for the duration of the test.

SESSION B: Duration approximately 30 minutes.

1. Apnea at work on land. Electrodes will be attached to your chest and heart rate monitored while you are in a relaxed, seated position on the cycle ergometer. Note that it may be necessary to shave a small patch of skin in order to attach each electrode.

You will then perform maximal breath-holds while working at given percentages of $\dot{V}O_2$ max on the cycle. Heart rate will be monitored on the ECG. Immediately afterwards ratings of perceived exertion (RPE) will be asked for. You will be instructed as to how to respond to the RPE scale.

Following each breath-holding bout you will repeat the procedure at the same exercise intensity, but without breath-holding.

SESSION C : Duration approximately 45 minutes.

1. Habituation. You will be asked to relax in a seated position on the cycle ergometer while it is underwater. The purpose of this is to enable you to become familiar with the underwater environment and breathing apparatus. The mouthpiece used will be identical to that used on land.

2. Maximal oxygen consumption ($\dot{V}O_2$ max) underwater. The protocol used will be identical to that used on land.

SESSION D: Duration approximately 30 minutes.

1. Apnea at work underwater. The testing procedure will be identical to that of session B, with the exception that it will be performed underwater. Additional water-proof tape will be used to ensure that the electrodes do not come loose in the water.

RISKS.

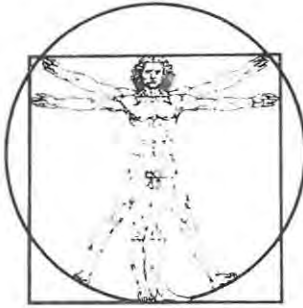
$\dot{V}O_2$ max test. The risks you may encounter during this test are mainly those encountered during light to heavy exercise - dizziness and fatigue, for example.

Underwater conditions. Each test will be performed on a cycle ergometer submerged at a depth of approximately 1.75m in the shallow end of the swimming pool. In order to counteract the positive bouyancy of the body underwater you will use toe clips on the pedals and will be asked to secure yourself to the ergometer by means of straps attached to a diver's belt. This belt incorporates a "quick-release" buckle so that you can literally pull it off, should you need to. The depth of the water will ensure that you will be able to sit comfortably with your head out of the water prior to the test. Should you need to come off the mouthpiece at any stage you need only raise your head.

At least two safety personnel will be on hand both on land and underwater to assist you at all times.

BENEFITS.

On completion of the study you will be provided with information relating to your lung volumes, $\dot{V}O_2$ max, and heart rate responses to each of the test conditions. The general results of the study will also be made available to you. In addition, you will be exposed to new and different equipment and methods of data collection in an area that is likely to be of interest to you. The findings of the study will contribute to man's understanding of human performance, particularly in an underwater environment, and may be of practical importance in both sporting and ergonomic areas.



HUMAN MOVEMENT STUDIES

SUBJECT CONSENT FORM

I, _____ having
been fully informed of the nature of the research entitled:

APNEIC BRADYCARDIA: TERRESTRIAL AND AQUATIC RESPONSES
IN MAN UNDER WORKING CONDITIONS.

do hereby give my consent to act as a subject in the abovenamed research.

I am fully aware of the procedures involved as well as the potential risks and benefits attendant to my participation as explained to me verbally and in writing. In agreeing to participate in this research, I waive any legal recourse against the researchers or Rhodes University, from any and all claims resulting from personal injuries sustained. This waiver shall be binding upon my heirs and personal representatives. I realize that it is necessary for me promptly to report to the researcher any signs or symptoms indicating any abnormality or distress.

I am aware that I may withdraw my consent and may withdraw from participation in the research at any time. I am aware that my anonymity will be protected at all times, and agree that the information collected may be used and published for statistical or scientific purposes.

I have read the information sheet accompanying this form and understand it. Any questions which may have occurred to me have been answered to my satisfaction.

Subject (or legal representative):

(PRINT NAME)

(SIGNED)

(DATE)

Person administering Informed Consent:

(PRINT NAME)

(SIGNED)

(DATE)

Witness:

(PRINT NAME)

(SIGNED)

(DATE)

Project Supervisor:

(PRINT NAME)

(SIGNED)

(DATE)

The following instruction was read to each subject prior to the apneic sessions:

"We want you to estimate how hard you feel the work is: that is we want you to rate the degree of perceived exertion you feel. By perceived exertion we mean the total amount of exertion and physical fatigue, combining all sensations and feelings of physical stress, effort and fatigue. Don't concern yourself with any one factor such as leg discomfort or shortness of breath, but try to concentrate on your total inner feeling of exertion. Try to estimate as honestly and objectively as possible. Don't underestimate the degree of exertion you feel, but don't overestimate it either. Just try to estimate as accurately as possible. When you are asked to rate your work, you should do so by giving the numerical value on the scale in front of you which indicates your evaluation of your perceived exertion at that moment. A rating of 6 corresponds with feelings of exertion while sitting quietly on the ergometer. A rating of 20 reflects maximal exertion."

(Adapted from Mihevic, 1983)

RPE SCALE

6

7 **VERY, VERY LIGHT**

8

9 **VERY LIGHT**

10

11 **FAIRLY LIGHT**

12

13 **SOMEWHAT HARD**

14

15 **HARD**

16

17 **VERY HARD**

18

19 **VERY, VERY HARD**

20

APPENDIX B

Physical characteristics and physiological
data of 16 subjects

Table X: Physical characteristics and physiological data obtained for 16 male subjects experienced in underwater apneic activities

Subject	Age (yrs)	Underwater experience (yrs)	Mass (kg)	stature (cm)	Sitting height (cm)	BSA (m ²)	Vital capacity (absolute) (l)	vital capacity (rel) (l/m ²)	Land $\dot{V}O_2$ max (ml/kg/min)	Underwater $\dot{V}O_2$ max (ml/kg/min)
1	30	1	86.9	181	2.08	95.25	5.47	2.63	40.7	33
2	18	4	74.4	185.8	1.98	94.7	5.17	2.61	50	40.7
3	25	9	78.6	175.3	1.94	91.8	5.67	2.92	54.2	36.5
4	18	1	69.3	181.5	1.89	93.2	5.13	2.72	55.2	33.4
5	19	3	78.2	182.9	2.00	91.6	4.28	2.15	46.1	46.2
6	18	4	62.3	170.2	1.72	90.7	5.15	2.93	52.9	46.2
7	19	3	91.1	185.8	2.16	91	6.26	2.91	58.2	51.1
8	19	1	76	184.8	1.99	91.9	5.23	2.63	43.3	37.6
9	23	3	73.3	181.8	1.94	93.7	6.06	3.13	46.3	45.7
10	20	4	75.8	181.1	1.96	92	5.48	2.8	55.1	45.8
11	18	1	81.7	176.1	1.98	89.2	4.71	2.37	52.5	36.8
12	22	2	64.4	181.3	1.83	90.5	5.36	2.93	43.7	42.2
13	24	1	64.5	177.34	1.80	90.6	5.82	3.23	42.6	36.9
14	20	1.5	76.9	181.8	1.98	93.5	5.68	2.87	36.3	36
15	19	2	71.2	172	1.84	89	4.94	2.69	45.2	33.4
16	22	5	76.2	174	1.91	89.5	4.64	2.43	45.7	38.8
$\bar{X}$	20.88	2.84	75.1	179.55	1.94	91.76	5.32	2.75	47.74	40.02
S.D	3.22	2.04	7.54	4.67	0.10	1.83	0.51	0.27	5.76	5.38
CV	15.42	71.83	10.04	2.6	5.15	1.99	9.59	9.82	12.07	13.44

APPENDIX C

Equations for the calculation of
 $\dot{V}O_2$ max and related variables

Computational procedures

1. Expired volume ($\dot{V}_E$) (ATPS):

$$\dot{V}_E \text{ (ATPS) (l)} = (V_2 - V_1) \times 0.9876$$

where: V_1 = initial volumeter reading (l)
 V_2 = final volumeter reading (l)
0.9876 = volumeter correction factor.

2. Minute volume ($\dot{V}_E$) (ATPS):

$$\dot{V}_E \text{ (ATPS) (l.min}^{-1}\text{)} = \frac{\dot{V}_E \text{ (ATPS) } \times 60}{\text{collection time (s)}}$$

3. Partial pressure of water vapour (P_{H_2O}):

$$P_{H_2O} \text{ (mmHg)} = 2.718 \left(2.303 \left[8.10765 - \frac{1750.286}{235 + t} \right] \right)$$

where: t = gas temperature ($^{\circ}\text{C}$)

4. Correction factor to reduce ambient conditions to standard temperature and pressure, dry (STPD)

$$\text{STPD factor} = \frac{273}{(t + 273)} \times \frac{(P_B - P_{H_2O})}{760}$$

where: t = ambient temperature ($^{\circ}\text{C}$)
 P_B = corrected barometric pressure (mmHg)
 P_{H_2O} = water vapour pressure (mmHg)

5. Power output (P.O):

$$P.O \text{ (W)} = \text{Resistance load (kg)} \times \text{pedal revolutions (rpm)}$$

6. Minute volume ($\dot{V}_E$) (STPD):

$$\dot{V}_E \text{ (STPD) (l.min}^{-1}\text{)} = \dot{V}_E \text{ (ATPS) (l.min}^{-1}\text{)} \times \text{STPD factor}$$

7. Oxygen consumption ($\dot{V}O_2$):

$$\dot{V}O_2 \text{ (l.min}^{-1}\text{)} = \dot{V}_e \text{ (STPD) (l.min}^{-1}\text{)} \times (0.265 FEN_2 - FE O_2)$$

where: 0.265 = Fraction of inspired oxygen FIO_2
 Fraction of inspired nitrogen FIN_2

$$FEN_2 = \text{Fraction of expired nitrogen:} \\ 1 - (FE O_2 - FECO_2)$$

$FE O_2$ = Fraction of expired oxygen

$FECO_2$ = Fraction of expired carbon dioxide

8. Oxygen consumption per kilogram of body mass ($\dot{V}O_2$):

$$\dot{V}O_2 \text{ (ml.kg}^{-1}\text{.min}^{-1}\text{)} = \frac{\dot{V}O_2 \text{ (l.min}^{-1}\text{)} \times 1000}{\text{Body mass (kg)}}$$

9. Carbon dioxide production ($\dot{V}CO_2$):

$$\dot{V}CO_2 \text{ (l.min}^{-1}\text{)} = \dot{V}_e \text{ (STPD) (l.min}^{-1}\text{)} \times FECO_2$$

10. Respiratory exchange ration (R):

$$R = \frac{\dot{V}CO_2}{\dot{V}O_2}$$

11. Ventilatory equivalent for oxygen (V.E. for O_2):

$$\text{V.E. for } O_2 \text{ (l.100ml } O_2) = \frac{\dot{V}_e \text{ (STPD) (l.min}^{-1}\text{)}}{\dot{V}O_2 \text{ (l.min}^{-1}\text{)} \times 10}$$

12. Ventilatory equivalent for carbon dioxide (V.E. for CO_2):

$$\text{V.E. for } CO_2 \text{ (l.100ml } CO_2) = \frac{\dot{V}_e \text{ (STPD) (l.min}^{-1}\text{)}}{\dot{V}CO_2 \text{ (l.min}^{-1}\text{)} \times 10}$$

13. Cardiac Output (Q):

$$\dot{Q} \text{ (l.min}^{-1}\text{)} = K + (5.2 \times \dot{V}O_2 \text{ (ml.kg}^{-1}\text{.min}^{-1}\text{)} \times \text{Body mass (kg)}) \\ 1000$$

where: k = a constant: 66 if the subject is male.
 75 if the subject is female.
 (Faulkner et al. , 1977)

APPENDIX D

Data Reduction and Analysis Software:

$\dot{V}O_2$ max and related variables

Vital capacity relative to body surface area

Digitization of ECG traces.

```

90 HOME
100 REM THIS PROGRAM CALCULATES A
110 REM NUMBER OF CARDIO-RESPIRATORY
120 REM PARAMETERS BASED ON KEYBOARD
130 REM INPUT OF SUBJECT DATA
135 GOSUB 4500
140 PRINT "          RHODES UNIVERSITY"
150 PRINT : PRINT "    DEPARTMENT OF PHYSICAL EDUCATION"
160 PRINT : PRINT "    WORK PHYSIOLOGY CALCULATION PACKAGE"
165 FOR ZZ = 1 TO 2000
166 NEXT ZZ
170 HOME
180 PRINT "YOU WILL BE ASKED A NUMBER OF QUESTIONS."
190 PRINT : PRINT "PLEASE RESPOND IN THE CORRECT UNITS."
200 PRINT : PRINT "IF YOU HAVE MISSING DATA....."
210 PRINT : PRINT "ENTER A '0' IN IT'S PLACE."
215 PRINT : PRINT
220 PRINT TAB( 13); FLASH : PRINT "* SPECIAL NOTE *": NORMAL
230 PRINT : PRINT "TERMINATE EACH ENTRY BY PRESSING-----"
240 PRINT : PRINT "          RETURN"
245 PRINT : PRINT : PRINT "USE THE FULL STOP AS A DECIMAL PLACE--"
246 PRINT : PRINT "----NOT THE COMMA/"
247 FOR AA = 1 TO 10000: NEXT AA: HOME
250 REM INPUT OF SUBJECT AND AMBIENT DATA
251 PRINT : PRINT "REMEMBER TO TURN ON THE PRINTER.."
252 PRINT "(ON LEFT) AND PRESS 'SEL'"
255 PRINT : PRINT
260 PRINT : PRINT "INPUT THE FOLLOWING INFORMATION:" : GOSUB 4500
270 PRINT : INPUT "SUBJECT'S NAME? "; A$
280 PRINT : INPUT "PRE-TEST BODY MASS (KG)? "; BM
290 PRINT : INPUT "MALE OR FEMALE? (M/F): "; SEX$
300 PRINT : INPUT "BICYCLE ERGOMETER? (Y/N): "; C$
310 IF C$ = "Y" GOTO 400
320 PRINT : INPUT "TREADMILL? (Y/N): "; J$
330 IF J$ = "Y" GOTO 400
340 PRINT : INPUT "LIFTING TASK? (Y/N): "; E$
350 IF E$ = "Y" GOTO 400
360 PRINT : INPUT "STEPPING ERGOMETER? (Y/N): "; G$
370 IF G$ = "Y" GOTO 400
380 PRINT : INPUT "CLIMBING TASK? (Y/N): "; F$
390 IF F$ = "Y" GOTO 400
400 PRINT : INPUT "BAROMETRIC PRESSURE? (MM HG): "; PB
410 PRINT : INPUT "AMBIENT TEMPERATURE? (DEG C): "; T
420 HOME
430 PRINT "NOW YOU WILL BE ASKED TO INPUT RAW DATA."
440 PRINT : PRINT "SAMPLE DESIGNATION AND"
442 PRINT : INPUT "ELAPSED TIME? "; B$
450 IF C$ = "Y" GOTO 3000
460 IF J$ = "Y" GOTO 3100
470 IF E$ = "Y" GOTO 3200
480 IF F$ = "Y" GOTO 3300
490 IF G$ = "Y" GOTO 3400
495 PRINT : FLASH : PRINT "REMEMBER": NORMAL : PRINT " - ENTER '0' FOR M
MISSING DATA"
500 PRINT : INPUT "HEART RATE? (L/MIN): "; HR
510 PRINT : INPUT "SKIN TEMPERATURE? (DEG C): "; SK
520 PRINT : INPUT "CORE TEMPERATURE? (DEG C): "; CT
530 PRINT : INPUT "SYSTOLIC BLOOD PRESSURE? (MM HG): "; SP
540 PRINT : INPUT "DIASTOLIC BLOOD PRESSURE? (MM HG): "; DP
550 PRINT : INPUT "BREATHING FREQUENCY? (BR/MIN): "; F
560 PRINT : INPUT "FRACTION EXPIRED O2? (FEO2): "; FO
570 PRINT : INPUT "FRACTION EXPIRED CO2? (FECO2): "; FC
580 PRINT : INPUT "FINAL VOLUMETER READING? (L): "; FV
590 PRINT : INPUT "INITIAL VOLUMETER READING? (L): "; IV
600 PRINT : INPUT "GAS COLLECTION TIME? (S): "; GT
610 PRINT : INPUT "POST-TEST BODY MASS? (KG): "; PM
620 GOSUB 2000
630 HOME
640 FLASH
650 PRINT "CALCULATIONS IN PROGRESS - PLEASE WAIT"
660 FOR Z = 1 TO 2000
670 NEXT Z
680 NORMAL
690 HOME
700 REM PRINT OUT OF COMPUTED DATA
705 PRINT D$;"PR#1"

```

```

710 GOSUB 4000
730 IF FV = 0 GOTO 790
731 IF IV = 0 GOTO 790
740 PRINT " VE(ATPS) = "; FN R1(VA); " L"
750 IF GV = 0 GOTO 790
751 PRINT " VE(ATPS) = "; FN R1(VM); " L/MIN"
770 PRINT " PH2O = "; FN R1(PH2O); " MM HG"
780 PRINT " STPD FACTOR = "; FN R2(STPD)
790 IF P = 0 GOTO 800
795 PRINT " POWER OUTPUT = "; FN R2(P); " W"
800 REM GOSUB 4100
805 REM GOSUB 4000
807 IF GT = 0 GOTO 930
810 PRINT " VE(STPD) = "; FN R1(VESTPD); " L/MIN"
820 IF FO = 0 GOTO 930
821 IF FC = 0 GOTO 930
830 PRINT " VO2 = "; FN R2(VO2); " L/MIN"
840 IF BH = 0 GOTO 860
850 PRINT " VO2 = "; FN R1(O2BM); " ML/KG/MIN"
860 PRINT " VCO2 = "; FN R2(VCO2); " L/MIN"
870 PRINT " R = "; FN R2(R)
880 PRINT " V.E. FOR O2 = "; FN R2(EO); " L/100ML O2"
890 PRINT " V.E. FOR CO2 = "; FN R2(EC); " L/100ML CO2"
900 PRINT "CARDIAC OUTPUT = "; FN R1(GLM); " L/MIN"
910 IF HR = 0 GOTO 950
920 PRINT " STROKE VOLUME = "; FN R2(SV); " ML/BT"
930 IF SK = 0 GOTO 950
931 IF CT = 0 GOTO 950
940 PRINT "MEAN BODY TEMP = "; FN R1(MBT); " DEG C"
950 IF F = 0 GOTO 960
952 IF FO = 0 GOTO 964
955 PRINT " TIDAL VOLUME = "; FN R2(VBART); " L/BR"
960 IF HR = 0 GOTO 964
961 IF FO = 0 GOTO 964
962 PRINT " OXYGEN PULSE = "; FN R1(OP); " ML/BT"
964 IF SP = 0 GOTO 975
965 IF DP = 0 GOTO 975
970 PRINT " MEAN ART PRES = "; FN R2(MAP); " MM HG"
972 PRINT "PULSE PRESSURE = "; FN R2(PP); " MM HG"
975 PRINT D$;"PR#0"
977 REM GOSUB 4100
980 PRINT : PRINT "ARE THERE MORE SAMPLES FOR THIS SUBJECT"
985 INPUT "THAT YOU WANT TO ANALYSE? (Y/N): "; H$
990 IF H$ = "Y" GOTO 420
1000 HOME
1010 PRINT : PRINT "INPUT THE FOLLOWING DATA IN ORDER"
1020 PRINT "TO CALCULATE SUMMARY DATA:"
1022 IF PH > 0 GOTO 1030
1025 PRINT : INPUT "POST-TEST BODY MASS? (KG): "; PH
1030 PRINT : INPUT "REST MEAN BODY TEMP? (DEG C): "; RM
1040 PRINT : INPUT "FINAL EXERCISE MEAN BODY TEMP? (DEG C): "; EM
1050 DBHC = (3.47 * PH - RM) - (3.47 * BM * RM)
1060 DH = (BM - PH) * 2430
1070 HP = DBHC + DH
1075 PRINT D$;"PR#1"
1080 PRINT : PRINT "DELTA BODY HEAT CONTENT = "; FN R2(DBHC); " KJ"
1090 PRINT "DELTA HEAT = "; FN R2(DH); " KJ"
1100 PRINT "HEAT PRODUCED = "; FN R2(HP); " KJ"
1110 PRINT D$;"PR#0"
1115 REM GOSUB 4100
1120 PRINT : INPUT "DID YOU COLLECT RECOVERY GAS? (Y/N): "; I$
1125 HOME
1130 IF I$ = "Y" GOTO 1150
1140 IF I$ = "N" GOTO 1295
1150 PRINT : PRINT "TO CALCULATE OXYGEN DEBT AND"
1160 PRINT "EFFICIENCY - INPUT THE FOLLOWING:"
1170 RT = 0
1171 RO = 0
1180 GOSUB 4200
1190 PRINT : INPUT "TOTAL EXERCISE DURATION (MIN): "; TE
1200 ODEBT = RO - (0.3 * RT)
1210 MRVO2 = ODEBT / TE
1220 GOSUB 2300
1230 NEFF = NU / (((EXVO2 - 0.3) + MRVO2) * 21)
1240 PRINT D$;"PR#1"
1250 HOME

```

```

1285 PRINT : PRINT "OXYGEN DEBT = "; FN R2(ODEBT); " L"
1290 PRINT : PRINT "FOR EXERCISE MINUTE "; K$
1295 PRINT "THE NET EFFICIENCY IS "; FN R1(NEFF); " %"
1300 PRINT D$;"PR#0"
1305 GOTO 1300
1310 GOSUB 2600
1315 REM GOSUB 4100
1320 PRINT : PRINT "ANY MORE EFFICIENCY CALCULATIONS FOR"
1325 INPUT "THIS SUBJECT? (Y/N): "; L$
1330 IF L$ = "Y" GOTO 1220
1335 PRINT : PRINT "DO YOU WISH TO COMPUTE EFFICIENCIES FOR"
1340 INPUT "ANOTHER SUBJECT? (Y/N): "; M$
1345 IF M$ = "Y" GOTO 1120
1350 PRINT : PRINT "DO YOU WISH TO RESTART THE PROGRAM"
1355 PRINT "TO REPEAT ALL THE CALCULATIONS"
1360 INPUT "FOR ANOTHER SUBJECT? (Y/N): "; N$
1365 IF N$ = "Y" THEN CLEAR : HOME : GOTO 260
1370 HOME
1375 PRINT "REMEMBER TO REMOVE THE DISK BEFORE"
1380 PRINT "YOU TURN OFF THE POWER TO THE COMPUTER,"
1385 PRINT "THE TV MONITOR AND THE PRINTER."
1390 PRINT : PRINT "THANK YOU - GANKIE"
1395 END
1400 IF FV = 0 GOTO 2270
1405 IF IV = 0 GOTO 2270
1410 VA = FV - IV
1415 IF GT = 0 GOTO 2270
1420 VM = (VA * 60) / GT
1425 IF PB = 0 GOTO 2270
1430 IF T = 0 GOTO 2270
1435 RB = 1750.286 / (235 + T)
1440 PH20 = 2.718 ^ (2.303 * (9.10765 - 58))
1445 STPD = (273 / (T + 273)) * ((PB - PH20) / 760)
1450 VESTPD = VM * STPD
1455 IF FO = 0 GOTO 2270
1460 IF FC = 0 GOTO 2270
1465 N2 = 1 - (FO + FC)
1470 V02 = VESTPD * ((0.265 * N2) - FO)
1475 IF BM = 0 GOTO 2130
1480 Q2BM = (V02 * 1000) / BM
1485 VCO2 = VESTPD * FC
1490 R = VCO2 / V02
1495 EQ = VESTPD / (V02 * 10)
1500 EC = VESTPD / (VCO2 * 10)
1505 IF SEX$ = "M" GOTO 2180
1510 IF SEX$ = "F" GOTO 2210
1515 BM = 66 + (5.2 * 02BM)
1520 QLM = (QM * BM) / 1000
1525 GOTO 2230
1530 FQ = 75 + (5.2 * 02BM)
1535 QLM = (FQ * BM) / 1000
1540 IF HR = 0 GOTO 2250
1545 SV = (QLM * 1000) / HR
1550 OP = (V02 * 1000) / HR
1555 IF F = 0 GOTO 2270
1560 VBART = VESTPD / F
1565 IF SK = 0 GOTO 2290
1570 IF CT = 0 GOTO 2290
1575 ST = (SK / 3) + (2 * (CT / 3))
1580 IF SP = 0 GOTO 2330
1585 IF DP = 0 GOTO 2330
1590 PAP = (SP + (3 * DP)) / 4
1595 AP = SP - DP
1600 RETURN
1605 IF C$ = "Y" THEN C = 0.1
1610 IF J$ = "Y" THEN C = 0.16
1615 IF E$ = "Y" THEN C = 0.1
1620 IF F$ = "Y" THEN C = 0.16
1625 IF G$ = "Y" THEN C = 0.16
1630 NJ = 6 * (PD + (C * PD))
1635 RETURN
1640 PRINT : PRINT "TO CALCULATE EFFICIENCY - INPUT THE"
1645 PRINT "FOLLOWING INFORMATION: "
1650 GOSUB 2800
1655 NEFF = NJ / ((EXV02 - 0.3) * 25)
1660 PRINT D$;"PR#1"
1665 HOME

```

```

2450 PRINT : PRINT "FOR EXERCISE MINUTE ";K$
2460 PRINT "THE NET EFFICIENCY IS " : FN R1(NTEFF); " %"
2470 PRINT D1;"PR$)"
2480 RETURN
2490 PRINT : PRINT "WHICH EXERCISE MINUTE DO YOU WISH TO"
2500 INPUT "CALCULATE EFFICIENCY FOR? ";K$
2510 PRINT : INPUT "VO2 FOR THAT EX MINUTE? (L/MIN): ";EXVO2
2520 PRINT : INPUT "POWER OUTPUT - SAME MINUTE? (W): ";PO
2530 GOSUB 2500
2540 RETURN
2550 PRINT : INPUT "AIR RESISTANCE LETTING? (N): ";RE
2560 PRINT : INPUT "PEDAL RATE? (RPM): ";RP
2570 P = RE * RP
2580 GOTO 495
2590 PRINT : INPUT "TREADMILL VELOCITY? (M/SEC): ";TV
2600 PRINT : INPUT "TREADMILL PERCENT GRADE? (%): ";G
2610 P = 0.1 * BM * G * TV
2620 GOTO 495
2630 PRINT : INPUT "MASS LIFTED? (KG): ";ML
2640 PRINT : INPUT "VERTICAL DISTANCE MOVED THROUGH? (M): ";VD
2650 PRINT : INPUT "NUMBER OF REPETITIONS? ";NR
2660 PRINT : INPUT "EXERCISE DURATION? (S): ";ED
2670 P = (ML * 10) * ((VD * NR) / ED)
2680 GOTO 495
2690 PRINT : INPUT "VERTICAL DISTANCE MOVED? (M): ";VT
2700 PRINT : INPUT "EXERCISE DURATION? (S): ";DU
2710 P = (BM * 10) * (VT / DU)
2720 GOTO 495
2730 PRINT : INPUT "STEP HEIGHT? (M): ";SH
2740 PRINT : INPUT "STEP RATE? (STEPS/MIN): ";SR
2750 P = BM * ((SH * SR) / 6)
2760 GOTO 495
2770 PRINT "          SUBJECT: ";A$
2780 PRINT : PRINT "TEST CONDITION: ";B$
2790 PRINT
2800 RETURN
2810 FLASH : PRINT : PRINT "COPY THE ABOVE DATA BEFORE PROCEEDING": NORMAL
2820 PRINT
2830 PRINT "PRESS ANY KEY TO CONTINUE..."; GET W$
2840 HOME
2850 RETURN
2860 PRINT : PRINT "NUMBER OF RECOVERY GAS"
2870 PRINT : INPUT "      SAMPLES COLLECTED? ";K
2880 N = 1
2890 PRINT : PRINT "DURATION OF SAMPLE ";N;"? (MIN): ";: INPUT "":J
2900 PRINT : PRINT "VO2 FOR SAMPLE ";N;"? (L/MIN): ";: INPUT "":L
2910 RT = RT + J
2920 W = L * J
2930 RO = RO + W
2940 N = N + 1
2950 IF N = (K + 1) GOTO 4300
2960 GOTO 4220
2970 RETURN
2980 W$ = " "
2990 DEF FN R1(X) = INT (X * 10 + 0.5) / 10
3000 DEF FN R2(X) = INT (X * 100 + 0.5) / 100
3010 DEF FN R3(X) = INT (X + 0.5)
3020 D$ = CHR$(4)
3030 RETURN

```

```

10 REM *****
20 REM *** STATIC AND DYNAMIC LUNG VOLUMES ***
30 REM *****
40 POKE 33,40: POKE 34,0: HOME :D$ = CHR$(4):G$ = CHR$(7)
50 PRINT " STATIC AND DYNAMIC LUNG VOLUMES"
60 PRINT " *****"
70 PRINT : PRINT "PLEASE ENTER THE FOLLOWING INFORMATION:-"
75 POKE 34,5: HOME
80 PRINT : INPUT "SUBJECT NAME: ";NM$
90 PRINT : INPUT " AGE(YRS): ";AGE
100 PRINT : INPUT " BODY MASS(KG): ";M
110 PRINT : INPUT " STATURE(CM): ";L
120 VTAB 23: INPUT "ANY MISTAKES(Y/N)? ";AN$
130 IF AN$ < > "Y" AND AN$ < > "N" THEN PRINT G$;: GOTO 120
140 IF AN$ = "Y" THEN HOME : GOTO 70
150 HOME : PRINT : INPUT " BAROMETRIC PRESSURE(mmHG): ";BP
160 PRINT : INPUT "WATER VAPOUR PRESSURE(mmHG): ";WP
170 PRINT : INPUT " AMBIENT TEMP.(deg C): ";T
180 VTAB 23: INPUT "ANY MISTAKES(Y/N)? ";AN$
190 IF AN$ < > "Y" AND AN$ < > "N" THEN PRINT G$;: GOTO 180
200 IF AN$ = "Y" GOTO 150
210 DEF FN AB(X) = INT (X * ((BP - WP) / (BP - 47)) * (310 / (273 + T)
) * 1000 + 0.5) / 1000
220 HOME : PRINT : PRINT "ENTER NO. OF VERTICAL DISPLACEMENTS OF TIDAL
VOLUME TRACE ON THE GRAPH PAPER.": PRINT : INPUT "NO. OF VERT DISPLA
CEMENTS: ";VD
225 TV = (VD * 50) / 1000: PRINT : PRINT "TIDAL VOLUME = "; FN AB(TV);" L
ITRES"
230 PRINT : INPUT "NO. OF VERTICAL DISPLACEMENTS ABOVE TOP OF TV TRACE:
";ABOVE
240 PRINT : INPUT "NO. OF VERTICAL DISPLACEMENTS BELOW BOTTOM OF TV
TRACE ";BELOW
250 PRINT : INPUT "VOL. EXPIRED IN 1 SEC (l): ";EV
260 VTAB 23: INPUT "ANY MISTAKES(Y/N)? ";AN$
270 IF AN$ < > "Y" AND AN$ < > "N" THEN PRINT G$;: GOTO 260
280 IF AN$ = "Y" GOTO 220
290 BSA = (71.84 * ((M ^ 0.425) * (L ^ 0.725))) / 10000
310 IRV = (ABOVE * 50) / 1000
320 ERV = (BELOW * 50) / 1000
330 IC = TV + IRV
340 VC = IC + ERV
350 FEV1 = EV / VC
360 FI = (FEV1 / VC) * 100
365 RLS = FN AB(VC) / BSA
370 DEF FN R3(X) = INT (X * 1000 + 0.5) / 1000
380 PRINT D$;"PR#1"
390 PRINT CHR$(9);"SON"
400 PRINT TAB(20);"STATIC AND DYNAMIC LUNG VOLUMES"
410 PRINT TAB(20);"=====
420 PRINT : PRINT
430 PRINT "SUBJECT NAME: ";NM$
440 PRINT " AGE(YRS): ";AGE
450 PRINT " BODY MASS(KG): ";M
460 PRINT " STATURE(CM): ";L
470 PRINT : PRINT
480 PRINT " BAROMETRIC PRESSURE(mmHG): ";BP
490 PRINT "WATER VAPOUR PRESSURE(mmHG): ";WP
500 PRINT "AMBIENT TEMP(deg C): ";T
510 PRINT : PRINT
520 PRINT "LUNG VOLUMES ABSOLUTE(l) RELATIVE(l/sq. m.
)"
530 PRINT : PRINT "TIDAL VOLUME";: POKE 36,33: PRINT FN AB(TV);: POKE 3
6,46: PRINT FN R3((FN AB(TV)) / BSA)
540 PRINT : PRINT "INSPIRATORY RESERVE VOLUME";: POKE 36,33: PRINT FN A
B(IRV);: POKE 36,46: PRINT FN R3((FN AB(IRV)) / BSA)
550 PRINT : PRINT "EXPIRATORY RESERVE VOLUME";: POKE 36,33: PRINT FN AB
(ERV);: POKE 36,46: PRINT FN R3((FN AB(ERV)) / BSA)
560 PRINT : PRINT "INSPIRATORY CAPACITY";: POKE 36,33: PRINT FN AB(IC);
: POKE 36,46: PRINT FN R3((FN AB(IC)) / BSA)
570 PRINT : PRINT "VITAL CAPACITY";: POKE 36,33: PRINT FN AB(VC);: POKE
36,46: PRINT FN R3((FN AB(VC)) / BSA)
580 PRINT : PRINT
590 PRINT "FORCED EXPIRATORY VOLUME: ";FEV1
600 PRINT : PRINT "FORCED EXPIRATORY VOLUME INDEX: ";FI
610 PRINT : PRINT "RELATIVE LUNG SIZE(l/sq. m.): "; FN R3(RLS)
620 PRINT D$;"PR#2"
630 POKE 34,3: HOME
640 PRINT : INPUT "DO YOU WISH TO RE-RUN THE PROGRAM(Y/N)? ";AN$
650 IF AN$ < > "Y" AND AN$ < > "N" GOTO 640
660 IF AN$ = "Y" THEN CLEAR :X = FRE(10): GOTO 10
670 POKE 34,0: HOME : PRINT "THE END": END

```



```

10 REM *****
15 REM *** DIGITIZE INTERVALS ***
20 REM *****
25 LOMEM: 25392
30 DEF FN R1(X) = INT (X * 10 + 0.5) / 10
35 DIM D(500),SD(500)
40 D$ = CHR$(4):B$ = CHR$(7):I$ = CHR$(9):R$ = CHR$(13):E$ = CHR$(
    (27)
45 PRINT D$;"CLOSE GRAPHICS TABLET SOFTWARE"
50 PRINT D$;"OPEN TAB.INFORMATION,D1"
55 PRINT D$;"READ TAB.INFORMATION"
60 INPUT SL: INPUT XL: INPUT YL: INPUT XH: INPUT YH
65 PRINT D$;"CLOSE TAB.INFORMATION"
70 XA = XH - XL:YA = YH - YL
75 LT = INT ((XA + YA) / 2)
80 PI = INT (LT / 11)
85 XM = XL:YM = YL + PI
90 PRINT D$;"PR#0": PRINT D$;"IN#0": HOME
95 POKE 33,40: POKE 34,0: HOME
100 HTAB 10: INVERSE: PRINT "DIGITIZE INTERVALS": NORMAL: POKE 34,3: HOME

105 SF = 1500:MF$ = "D":UN$ = "BEATS/MIN"
107 PRINT "ENTER A TITLE FOR THE PRINTOUT": INPUT "TITLE: ";TL$: HOME
110 INPUT "DO YOU WANT HEART RATE ANALYSIS?(Y/N)":HR$: IF HR$ < > "Y" AND
    HR$ < > "N" THEN PRINT G$: GOTO 110
115 HOME: IF HR$ = "Y" GOTO 155
120 PRINT: PRINT "YOU MAY NOW ENTER A SCALE FACTOR TO": PRINT "CONVERT
    THE DISTANCE BETWEEN DIGITIZED": PRINT "POINTS FROM MM TO SOME OTHER
    UNIT. YOU": PRINT "MAY ALSO ENTER THE NAME OF THAT UNIT."
125 PRINT: PRINT "IF YOU DO NOT WISH TO SCALE THE DATA, SIMPLY PRESS
    RETURN WHEN ASKED FOR THE SCALE FACTOR."
130 PRINT: INPUT "ENTER SCALE FACTOR: ";Z$: IF Z$ = "" THEN SF = 0: HOME
    : GOTO 155
135 SF = VAL (Z$)
140 PRINT: INPUT "ENTER NAME OF SCALED UNIT: ";UN$
145 VTAB 18: PRINT "IS THE SCALE FACTOR TO BE MULTIPLIED": INPUT "OR DIV
    IDED BY THE INTERVAL? (M/D) ";MF$: IF MF$ < > "M" AND MF$ < > "D" THEN
    PRINT "": GOTO 145
150 HOME
155 VTAB 21: PRINT "PRESS RETURN IF THERE IS MISSING DATA."
160 VTAB 4: PRINT "PLEASE ENTER THE FOLLOWING INFORMATION:--"
165 INPUT "      SUBJECT NAME: ";SN$
170 INPUT "      SUBJECT AGE: ";AG$
175 INPUT "      SUBJECT SEX: ";SX$
180 INPUT "      DATE: ";DT$
185 INPUT "      TIME: ";TH$
190 INPUT "      CONDITION: ";CN$
195 INPUT "      TEMP (DEG C): ";TE$
200 INPUT "      REL HUMIDITY (%): ";RH$
205 INPUT "BAROMETRIC PRES(MMHG): ";BP$
210 INPUT "VO2 MAX (ML/KG.MIN): ";VM$
215 GOSUB 1015
220 IF C$ = E$ GOTO 150
225 POKE 34,3: HOME: IF HR$ = "N" GOTO 275
230 VTAB 2: HTAB (40 - LEN (SN$)) / 2: PRINT SN$
235 HOME: PRINT "PLEASE ENTER THE FOLLOWING INFORMATION:--"
240 INPUT "  % VO2 MAX: ";PV$
245 INPUT "      LOAD: ";LD$
250 INPUT "  CONDITION: ";CU$
255 INPUT "3-H TIME(C): ";T3$: d = VAL (T3)
260 GOSUB 1015
265 IF C$ = E$ GOTO 225
270 POKE 34,3: HOME
275 VTAB 12: HTAB 15: PRINT "TURN PRINTER ON": GOSUB 1255
280 GOSUB 1315: REM - PRINT HEADER INFO
285 POKE 34,3: HOME
290 PRINT "HOW MANY SECTIONS DO YOU WISH TO": INPUT "DIGITIZE(1-10) ";NS
    : IF NS < 1 OR NS > 10 THEN PRINT G$: GOTO 290
295 PRINT
300 FOR I = 1 TO NS
305 PRINT "NAME SECTION ";I: INPUT " ";NM$(I)
310 IF LEN (NM$(I)) > 20 THEN NM$(I) = LEFT$ (NM$(I),20)
315 NEXT

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```

320 GOSUB 1015
325 IF C$ = E$ GOTO 285
330 HOME : PRINT "ENTER THE NO. OF THE SECTION YOU WISH": INPUT "TO DIGI
      TIZE: ";ND: IF ND < 1 OR ND > NS THEN PRINT G$: GOTO 330
332 NM$ = NM$(ND)
335 VTAB 2: HTAB (40 - LEN (NM$(ND))) / 2: PRINT NM$(ND)
340 GOSUB 1040
345 POKE 34,3: HOME
350 VTAB 5: PRINT "OPTIONS:-"
355 VTAB 7: PRINT "1. ANALYSE ANOTHER SECTION"
360 VTAB 8: PRINT "2. PERFORM SUB-ANALYSIS ON THIS SECTION"
365 VTAB 9: PRINT "3. FINISH WITH THIS ANALYSIS"
370 VTAB 11: INPUT "ENTER OPTION (1-3) ";C: IF C < 1 OR C > 3 THEN PRINT
      ""; GOTO 370
375 IF C = 1 THEN GOSUB 2000: GOTO 330
377 IF C = 3 GOTO 480
380 HOME : VTAB 5: PRINT "SUB-ANALYSIS OPTIONS:-"
385 VTAB 7: PRINT "1. SINGLE SUB-SECTION (EG 25%-50%)"
390 VTAB 8: PRINT "2. PERCENTAGE INCREMENTS (EG 10%)"
395 VTAB 10: INPUT "ENTER OPTION (1-2) ";C: IF C < 1 OR C > 2 THEN PRINT
      ""; GOTO 395
400 IF C = 2 GOTO 435
405 VTAB 12: INPUT "IDENTIFY INTERVAL - FROM: ";LL
410 INPUT "                - TO: ";UL
415 IF UL < LL THEN PRINT ""; GOTO 405
420 NM$ = STR$(LL) + "-" + STR$(UL) + "%"
425 GOSUB 1630: REM - SUB-ANALYSIS
430 GOTO 345
435 VTAB 12: INPUT "ENTER PERCENTAGE INCREMENTS: ";CR
440 HOME : VTAB 12: HTAB 15: PRINT "BUSY - PLEASE WAIT"
445 II = INT (100 / CR)
450 FOR I = 1 TO II
455 LL = (I - 1) * CR:UL = I * CR
460 NM$ = STR$(LL) + "-" + STR$(UL) + "%"
465 GOSUB 1630: REM - SUB-ANALYSIS
470 NEXT
475 GOTO 345
480 POKE 34,3: HOME
485 VTAB 5: PRINT "OPTIONS:-"
490 VTAB 7: PRINT "1. RE-RUN PROGRAM WITH SAME SUBJECT"
495 VTAB 8: PRINT "2. RE-RUN PROGRAM WITH NEW SUBJECT"
500 VTAB 9: PRINT "3. QUIT"
505 VTAB 11: PRINT "ENTER OPTION (1-3) I1:"
510 VTAB 11: HTAB 21: GET C$
515 IF C$ = R$ THEN C$ = "1"
520 VTAB 11: HTAB 21: PRINT C$
525 C = VAL (C$)
530 IF C < 1 OR C > 3 THEN PRINT G$: GOTO 505
535 ON C GOTO 340,345,550
540 GOTO 225
545 GOTO R5
550 HOME : VTAB 12: HTAB 15: PRINT "THE END": END
1000 REM *****
1005 REM *** ROUTINES ***
1010 REM *****
1015 REM
1020 REM *** ACCEPT OR CANCEL INPUT
1025 REM
1030 PRINT G$
1035 POKE 34,21: HOME : INVERSE : PRINT "PRESS ESC TO CANCEL": PRINT "PR
      ESS SPACE TO ACCEPT"; GET C$: NORMAL : RETURN
1040 REM
1045 REM - DIGITIZE & PRINT SET OF INTERVALS
1050 REM
1055 POKE 34,3: HOME
1060 VTAB 10: HTAB 10: PRINT "DIGITIZE INTERVALS": POKE 34,12: HOME
1065 GOSUB 1115: REM - DIGITIZE HEART BEATS
1070 IF SF = 0 GOTO 1100
1075 FOR J = 1 TO KNT
1080 IF MF$ = "M" THEN SD(J) = FN R1(SF * D(J)): GOTO 1090
1085 SD(J) = FN R1(1500 / D(J))
1090 NEXT
1095 LL = 1:UL = KNT
1100 GOSUB 1440: REM - PRINT DIGITIZED DATA
1105 D = TD:NI = KNT

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```

1110 RETURN
1115 REM
1120 REM *** DIGITIZE DATA
1125 REM
1130 KNT = 0
1135 PRINT G$; GOSUB 1210; GOSUB 1275; HOME
1140 IF FLAG < > 1 THEN HOME : FOR K = 1 TO 5: PRINT G$;; NEXT : HTAB
15: PRINT "ERROR": PRINT " PLEASE RE-DIGITIZE THIS POINT": GOTO 113
5
1145 D1 = X
1150 KNT = KNT + 1
1155 PRINT G$; GOSUB 1210; GOSUB 1275
1160 IF FLAG = 1 GOTO 1185
1165 IF FLAG = 2 GOTO 1180
1170 IF FLAG = 3 GOTO 1200
1175 HOME : FOR K = 1 TO 5: PRINT G$;; NEXT : HTAB 15: PRINT "ERROR": PRINT
" PLEASE RE-DIGITIZE THIS INTERVAL": KNT = KNT - 1: GOTO 1135
1180 HOME : FOR K = 1 TO 5: PRINT G$;; NEXT : PRINT " RE-POSITIO
N THE PAPER &": PRINT " RE-DIGITIZE THE LAST POINT": GOTO 1135

1185 D2 = X
1190 D(KNT) = (D2 - D1) / 10: D1 = D2
1195 GOTO 1150
1200 RETURN
1205 REM
1210 REM *SUBROUTINE TO READ TABLET*
1215 REM
1220 XCF = 0: YOFF = 0: SCAL = 2
1225 PRINT D$; "PR#"; SL: PRINT "T1,X" : XOFF; ",Y" : YOFF; ",S" : SCAL; ",R"
1230 PRINT D$; "IN#"; SL
1235 INPUT " " : X,Y,Z
1240 IF Z < > 2 GOTO 1235
1245 PRINT D$; "PR#0": PRINT D$; "IN#0"
1250 RETURN
1255 REM
1260 REM *** CONTINUE
1265 REM
1270 PRINT G$; VTAB 23: INVERSE : PRINT "PRESS SPACE TO CONTINUE"; GET
C$: NORMAL : PRINT : HOME : RETURN
1275 REM
1280 REM *** WHAT AREA OF TABLET?
1285 REM
1290 FLAG = 0
1295 IF X > XM AND X < XH AND Y > YM AND Y < YH THEN FLAG = 1: GOTO 1310
1300 IF X > XL AND X < XL + (PI / 2) AND Y > YL AND Y < YL + (PI / 2) THEN
KNT = KNT - 1: FLAG = 2: GOTO 1310
1305 IF X > XH - (PI / 2) AND X < XH AND Y > YL AND Y < YL + (PI / 2) THEN
KNT = KNT - 1: FLAG = 3
1310 RETURN
1315 REM
1320 REM *** PRINT HEADER INFO
1325 REM
1330 PRINT D$; "PR#1"
1335 PRINT I$; "SON"
1340 POKE 36,20: PRINT TL$
1350 PRINT : PRINT
1355 PRINT " SUBJECT NAME: " : SN$
1360 PRINT " SUBJECT AGE: " : AG$
1365 PRINT " SUBJECT SEX: " : SX$
1370 PRINT " DATE: " : DT$
1375 PRINT " TIME: " : TM$
1380 PRINT " CONDITION: " : CN$
1385 PRINT " TEMP (DEG C): " : TE$
1390 PRINT " REL HUMIDITY (%): " : RH$
1395 PRINT "BAROMTERIC PRES(MMHG): " : SP$
1400 PRINT " VO2 MAX (ML/KG.MIN): " : VM$
1405 IF HR$ = "N" GOTO 1430
1405 PRINT
1410 PRINT " % VO2 MAX: " : PV$
1415 PRINT " LOAD: " : LD$
1420 PRINT " CONDITION: " : CD$
1425 PRINT "B-H TIME(S): " : TS$
1430 PRINT D$; "PR#0"

```

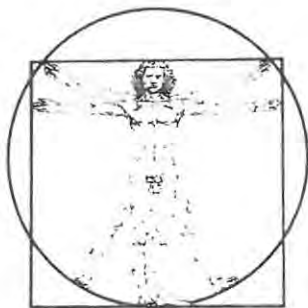
```

1435 RETURN
1440 REM
1445 REM *** PRINT DIGITIZED DATA
1450 REM
1455 PRINT D$;"PR#1"
1460 PRINT I$;"SON"
1465 PRINT : PRINT NM$: PRINT
1470 PRINT " MM";
1475 IF SF < > 0 THEN POKE 36,22: PRINT UN$
1480 TD = 0:TS = 0:MD = 0:MS = 0:KNT = UL - LL + 1
1485 FOR J = LL TO UL
1490 TD = TD + D(J)
1495 Z = D(J): GOSUB 1585
1500 POKE 36,5 - PTR: PRINT Z$;
1505 IF SF = 0 THEN PRINT : GOTO 1525
1510 TS = TS + SD(J)
1515 Z = SD(J): GOSUB 1585
1520 POKE 36,25 - PTR: PRINT Z$
1525 NEXT
1530 MD = TD / KNT: IF SF < > 0 THEN MS = TS / KNT
1535 PRINT
1540 PRINT "TOTAL DIST(MM) = ";TD;: IF SF < > 0 THEN POKE 36,30: PRINT
    "TOTAL ";UN$;" = ";TS;
1545 PRINT : PRINT
1550 PRINT "NO OF INTERVALS = ";KNT;: POKE 36,30: PRINT "NO OF INTERVALS
    = ";KNT;
1555 PRINT : PRINT
1560 PRINT " AVE DIST (MM) = "; FN R1(MD);: IF SF < > 0 THEN POKE 36
    ,30: PRINT "AVE ";UN$;" = "; FN R1(MS);
1565 PRINT : PRINT
1570 IF BF = 1 THEN PRINT " CUM. TIME (S) = "; FN R1(CBH)
1575 PRINT D$;"PR#0"
1580 RETURN
1585 REM *** ROUTINE TO LINE UP DEC PT IN TABLES OF NUMERIC OUTPUT
1590 Z$ = STR$ (Z)
1595 ZL = LEN (Z$)
1600 PTR = 0
1605 FOR ZI = 1 TO ZL
1610 IF MID$ (Z$,ZI,1) = "." THEN PTR = ZI - 1: GOTO 1625
1615 NEXT
1620 IF PTR = 0 THEN PTR = ZL
1625 RETURN
1630 REM
1635 REM *** SUB-ANALYSIS
1640 REM
1645 LX = LL * D / 100:UX = UL * D / 100:TT = 0
1650 FOR ZI = 1 TO NI
1655 TT = TT + D(ZI)
1660 IF TT < LX GOTO 1665
1661 IF TT - LX < = D(ZI) / 2 THEN LL = ZI + 1:TT = TT + D(ZI + 1): GOTO
    1670
1662 LL = ZI: GOTO 1670
1665 NEXT
1670 FOR ZI = LL + 1 TO NI
1675 TT = TT + D(ZI)
1680 IF TT < UX GOTO 1685
1681 IF TT - UX < = D(ZI) / 2 THEN UL = ZI: GOTO 1690
1682 UL = ZI - 1: GOTO 1690
1685 NEXT
1690 GOSUB 1440: REM - PRINT
1695 RETURN
1700 REM
1705 REM *** CHOOSE SECTION
1710 REM
1715 HOME
1720 FOR I = 1 TO NS
1725 PRINT I;" ";NM$(I)
1730 NEXT
1735 POKE 34,20
1745 RETURN

```

APPENDIX E

Subject feedback



HUMAN MOVEMENT STUDIES

August, 1988

Dear

At last, the long-awaited results of your participation in my study. I have presented the data in tabular form. Unfortunately for ethical reasons names could not be included, but direct comparison of your results with those of your fellow divers may be of interest to you. If you have any queries at all, or wish to see the results in more detail, don't hesitate to contact me.

Finally, I must thank each of you for the enthusiastic and goodnatured way in which you took part in this research. Obviously without you it would not have been possible, and I am extremely grateful.

Many thanks, and all the best for the future



WHAT THE DATA MEANS

$\dot{V}O_2$ max

Maximal aerobic power ($\dot{V}O_2$ max) can be defined as the maximum amount of oxygen which can be consumed per unit of time. It is an indication of the functional capacity of the cardiovascular and respiratory systems. Since it represents the maximal rate at which energy can be released in the presence of oxygen, it may serve as an indication of one's potential for endurance-type activities. Bearing in mind that other energy pathways are also used during activity, it is obviously not a measure or predictor of performance as such. This study confirmed that total immersion in water produces significant reductions in circulatory and respiratory responses to maximal exercise. In addition to decreased $\dot{V}O_2$ max, reductions were also observed in cardiac output (the amount of blood pumped out of the heart every minute), and pulmonary ventilation (the amount of air breathed in and out every minute). This may have important implications for the evaluation of the energy cost of tasks performed underwater.

Average $\dot{V}O_2$ max values obtained on a cycle ergometer are approximately 50ml.kg.min on land, and 48ml.kg.min during head-out, rather than total immersion.

Vital Capacity

This represents the total volume of air that can be voluntarily moved in one breath, from full inspiration to maximum expiration. Since vital capacity is influenced by body size (ie. large people tend to have large lung volumes), this value was expressed relative to body surface area in this study. Average absolute values are approximately 4-5 litres in healthy young men. Habitual divers generally develop larger than average vital capacities, which, it is thought, is the result of constant exposure to increased pressure on the chest.

Heart rate data

It has been suggested that the drop in heart rate that has been observed with breath holding constitutes part of what is termed the "dive reflex". This is particularly evident in diving mammals and birds and, in addition to a decrease in heart rate, incorporates constriction of the peripheral blood vessels and reduced cardiac output. While this is said to represent an oxygen-conserving mechanism in animals, the existence, extent and role of such a response in man is the subject of some debate. The degree to which the heart slows (bradycardia) may be affected by factors such as water temperature, underwater experience, fitness, lung volume and psychological state.

The response of this group as a whole to breath holding was a reduction in heart rate which occurred at all exercise intensities studied. The degree of bradycardia was greatest during exercise at 50% $\dot{V}O_2$ max, and the heart rate dropped to a lower level underwater than on land. Your lowest heart rate recorded, together with the corresponding pre-breath hold value so that you can see how far it dropped, are presented in the table. The "(l)" or "(w)" refers to whether this occurred on land or underwater.

Table 1: Feedback data following participation in the study:
Apneic bradycardia: terrestrial and aquatic
responses in man under working conditions.

Subject	Mass (kg)	Stature (cm)	Sitting height (cm)	Vital capacity (L-ab)	Vital capacity (L-rel)	Land $\dot{V}O_2$ max (ml.kg ⁻¹ . min ⁻¹)	Water $\dot{V}O_2$ max (ml.kg ⁻¹ . min ⁻¹)	$\bar{X}$ Breath hold duration (sec) (land)	$\bar{X}$ Breath hold duration (sec) (water)	Lowest heart rate (b.min ⁻¹)	Pre- BH heart rate (b.min ⁻¹)	% $\dot{V}O_2$ max
1	86.9	181	95.25	5.47	2.63	40.7	33	37.04	33.4	50.7	93.7	70(w)
2	74.4	185.75	94.65	5.17	2.61	50	40.7	28.9	34.8	34.9	90.4	90(w)
3	78.6	175.3	91.75	5.67	2.92	54.2	36.5	32.5	38.78	28	67.7	90(l)
4	69.3	181.45	93.15	5.13	2.72	55.2	33.4	35.3	38.2	41.4	77	70(l)
5	78.15	182.85	91.55	4.28	2.15	46.1	46.2	33.1	28.5	37.7	70.9	50(w)
6	65.25	170.2	90.65	5.15	2.93	52.9	46.2	30.4	40.2	27.4	83.5	70(w)
7	91.1	185.75	91	6.26	2.9	58.2	51.1	36.8	38.4	36.6	81.2	90(l)
8	76	184.8	91.9	5.23	2.63	43.3	37.6	18.7	27.3	53.4	75.1	90(w)
9	73.3	181.75	93.65	6.1	3.13	46.3	45.7	42	40.7	105.6	101	50(w)
10	75.8	181.05	92	5.48	2.8	51	45.8	44.9	46.1	26	77.2	90(l)
11	81.7	176.1	89.15	4.7	2.37	52.5	36.8	22.3	21.2	41.2	80.4	50(w)
12	64.4	181.25	90.5	5.36	2.93	43.7	42.2	28.3	51.9	31.4	83.6	70(w)
13	64.5	177.35	90.55	5.82	3.23	42.6	36.9	31.4	31.2	78.1	95.5	50(w)
14	76.9	181.75	93.45	5.68	2.87	36.3	36	40	41.3	35.5	95.4	50(w)
15	71.2	171.95	89	4.94	2.69	45.2	33.4	16.9	19.43	57.3	79.1	90(w)
16	76.2	174	89.45	4.64	2.43	45.7	38.8	29.4	36.1	31.3	83.6	70(w)
$\bar{X}$	75.23	179.27	91.73	5.32	2.75	47.74	40.02	31.72	35.47			
S.D.	7.26	4.41	1.83	0.51	0.27	5.76	5.38	7.59	8.27			

ADDITIONAL COMMENTS:

BH = Breath-hold
L-ab = litres, absolute
L-rel = litres, relative
to body surface area