Non-Weight Bearing Water Exercise: Changes in Cardiorespiratory Function in Elderly Men and Women

by

Darrell James Jessop
B.Sc., University of British Columbia, 1977

A THESIS SUBMITTED IN PARTIAL FULFILLMENT OF
THE REQUIREMENTS FOR THE DEGREE OF
MASTER OF PHYSICAL EDUCATION
in
THE FACULTY OF GRADUATE STUDIES
School of Physical Education and Recreation

We accept this thesis as conforming to the required standards

THE UNIVERSITY OF BRITISH COLUMBIA
October, 1988

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Department of **Physical Education/Recreation**

The University of British Columbia
Vancouver, Canada

Date **October 13, 1988**
ABSTRACT

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The purpose of this study was to evaluate the impact of a 5 week program of aquatic exercise on selected cardiorespiratory parameters in the elderly participant. Fifteen men and women (mean age 68.5 years, range 61-75 years) were recruited voluntarily from regional adult day-care and community centre facilities. Participants underwent a series of physiological tests before the program started and 5 weeks later at the end of the program. Measurements included height, weight, spirometry measurements (FVC, FEV\textsuperscript{1.0}, V\textsubscript{Emax}), resting blood pressure, resting heart rate, exercise heart rate and VO\textsubscript{2max} as determined by a continuous treadmill test (modified after Jones and Campbell, 1982).

Following the 5 week aquatic exercise program, the experimental group (n = 8) showed a significant decrease in resting systolic blood pressure (SBPR) (EXPTL:131.5<CTRL:133.4 mmHg) and resting heart rate (HR\textsubscript{REST}) (EXPTL:71.0<CTRL:76.6 bts·min\textsuperscript{-1}) in comparison to the control group (n = 7) which exhibited no change. In addition, the experimental group yielded a significant increase in forced expiratory volume (FEV\textsubscript{1.0}) (EXPTL:2.4>CTRL:2.2 l·sec\textsuperscript{-1}) and maximal oxygen uptake (VO\textsubscript{2max}) (EXPTL:25.8>CTRL:23.5 ml·kg\textsuperscript{-1}·min\textsuperscript{-1}) in comparison to the control group.

The findings in this study indicate that the exercise capacity of the elderly participant can increase with aquatic exercise: supervised aquatic exercise at or above the recommended intensity of exercise performed three times weekly can produce significant changes in the physical work capacity of the elderly.
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ACKNOWLEDGEMENT

This work is dedicated to Karen, whose unwavering enthusiasm and encouragement spurred me on and to my parents and family who always believed in me.

I would also like to thank all those who assisted me in the completion of this work: Dr. Jack Taunton (committee chairman), committee members Dr. Richard Ham, Dr. E. Rhodes and Dr. P. Grantham. Additional thanks also to Dr. Gary Sinclair and Dr. R. Schutz.

Thank you everyone for giving direction to my neuroticism.
The aging process has been defined as a gradual decline in the ability of the individual to adapt to changes in the environment (Barry, 1986). As the population ages, health care professionals will be pressed to meet the special needs of the elderly. A program of regular exercise may aid in meeting these goals.

North Americans generally engage in less physical activity as they age. According to the 1981 Canada Fitness Survey, approximately 25% of those between the ages of 20 and 24 engage in sport or other forms of vigorous activity at least 3 hours per week. With individuals between the ages of 65 and 69, only 6.5% are seen to engage in regular activities (Hogan, 1986). Less than 9% of Canadians over the age of 55 participate in any deliberate exercise. It is particularly the institutionalized elderly that are the least active (Molloy and Watson, 1987). Currently, only 24% of Canadians can be considered adequately active for maintaining or improving cardiovascular fitness (Hogan, 1986).

An understanding is needed concerning the relationship between the physiological decline in aging individuals and the effects of exercise and regular activity, both in the presence and absence of disease. Information concerning appropriate activity levels and prescription guidelines for them are presently inadequate (Holm and Kirchhoff, 1984). Exercise that is initiated in adulthood and continues throughout the lifetime into old age is different from exercise that commences in old age after years of inactivity, an acute illness (such as a cardiovascular event) or chronic debilitation (arthritis). Physiological changes that are seen to occur with aging may be difficult to separate from those resulting from disease (Holm and Kirchhoff,
1984), but increasing evidence suggests that much of the disability seen in the elderly is a result of disuse or hypokinetic disease (Hogan, 1986).

The literature addresses aging and exercise from many varied perspectives. In recent years, there have been reports of significant improvements in physiologic function and performance in the elderly who have undergone extensive physical training. Evidence is accumulating that regular, long-term exercise slows the effects of the aging process in several body systems and helps in the prevention of cardiovascular disease (Kent, 1982). Exercise may prevent or reverse the effects of premature aging and disease, but it does not appear to affect the "natural" rate of aging, as measured by the maximum life span (Kent, 1982). There are few conclusive studies examining the effects of regular activity on longevity in humans (Molloy and Watson, 1987).

In addition to the effect of exercise in the reduction of mortality from cardiovascular disease, exercise has been shown to lessen the age-related increases in serum lipid and insulin levels and reduce the mineral loss in bone (Molloy and Watson, 1987). The inevitable physiological changes that are seen to accompany aging include increased blood pressure levels, a decreased cardiac reserve (primarily secondary to a decrease in maximal heart rate), and a decrease in vital capacity and chest wall compliance, resulting in a reduced maximum voluntary ventilation. These changes limit the elderly individual's capacity to maintain high levels of aerobic fitness (Barry, 1986). Aging also instigates an overall decrease in muscular strength, although this may be predominantly related to changes in individual lifestyles (i.e. decreased levels of activity) (Piscopo, 1985). Osteoporosis is a serious problem in the elderly and its sequelae (i.e. vertebral compression and hip fractures) are major causes of mortality (Barry, 1986). Aging causes a decrease in bone density of approximately 30% to 50% at the
rate of approximately 0.4% per year, beginning at about age 50 (Hogan, 1986). Men do not experience difficulties as a result of the cumulative effects of osteoporosis until approximately the eighth decade of life (Barry, 1986). In women, bone loss may begin at age 30-35 years and continues at a more rapid rate than that observed in man. The rate of bone loss in woman may progress from approximately 1% per year to 2% - 3% per year at post-menopause (Mazess; 1979, Smith, 1982). Individuals that are immobilized, bedridden (unweighted) or who have experienced a loss of muscular function may experience an even greater rate of bone loss, as high as 1% per week (Barry, 1986). The effects of regular exercise on the structural integrity of bone have been demonstrated in numerous studies. It has been shown that the bone mineral content in the cortex is thicker in individuals who exercise regularly when compared to those who do not (Dalen and Olsson, 1974; Jones et al., 1977; Montoye et al., 1980).

From the fifth decade of life, there is a 3% - 5% loss of muscle tissue per decade with the greatest loss seen in the musculature of the legs and trunk (Barry, 1986). This loss of muscle tissue is enhanced by the resulting disuse from a sedentary lifestyle. Decreased flexibility is usually encountered with the loss of musculature; age-related degenerative changes in the elastin of connective tissue and the effects of osteoporosis and arthritis in addition to the actual shortening of the musculo-tendinous unit all contribute to a decrease in overall flexibility. Exercise programs have yielded significant improvements in joint flexibility and range of motion (Munns, 1981).

A slowing in reaction time is seen with increasing age, but this also appears to be related to disuse from inactivity. Decreased peripheral nerve function in elderly individuals is substantiated by the increased incidence of absent deep tendon reflexes (Hogan, 1986). In addition to the overall
reduction in sensory function, a slowing of mentation and impairment of motor responses are noted. Coordination and balance may decline, contributing to poor motivation and inability to perform everyday tasks (Barry, 1986). Regular exercise has been shown to slow the deterioration in memory, cognitive and psychomotor function as well as proving to have beneficial effects on depression, anxiety and stress (Molloy and Watson, 1987).

Circulatory diseases (heart disease, stroke and hypertension), digestive diseases and mental disorders head the list of current health care expenditures. Health expenses continue to rise and with the increase in the number of middle-aged and older adults, health professionals forecast higher costs in the years ahead (Piscopo, 1985). The prospect of continued escalation in health care spending makes it a major issue for both the public and private sectors of our society.

Our health care system is primarily designed to assist individuals after they have become ill or functionally incapacitated. Longevity is desirable, but the individual's enjoyment of life, sense of well-being and contributions to society outweigh any quantitative dimension of aging. Medical technology has expanded the aging population and will continue to do so. Many specific diseases are now curable and the effects of many others can now be controlled but we have only recently begun to recognize the value of improving the aged population's health and well-being through preventative and health promotional measures. It is clear that a continuing emphasis on the development of acute care modalities must be balanced with an increased emphasis on factors which will improve the quality of life for the elderly individual and improve their ability to maintain independence.

The maintenance of health and preventative medicine have emerged as important aspects of health care. The beneficial effects of exercise for the elderly have been documented and it is believed that the elderly should be
encouraged to engage in regular physical exercise. With the increasing number of such programs, it has become necessary to evaluate them in terms of their ability to positively change the level of fitness in the participant.

The factors preventing the elderly from participating in a structured exercise program are varied, numerous and not unlike the reasons cited by the younger population for not implementing regular exercise in their daily activities (Piscopo, 1985). Factors unique to the elderly population that may be seen as a contraindication to an exercise program include osteoporosis, cardiopulmonary impairment by pathology, neurological deficit (Alzheimer's, stroke), metabolic disorders and decreased mobility. Some areas of concern with exercise and its prescription in the elderly has been the motivation to exercise, the risk of injury and the requirement for proper medical assessment before participation in an exercise program (Hogan, 1986).

In supervised exercise programs, musculoskeletal injury serious enough to prohibit participation has been seen to occur in up to 50% of participants. In 20% of the participants, the injury will be severe enough to warrant permanent cessation of the activity (Piscopo, 1985). Due to the high prevalence of disease states in the elderly, prior medical assessment is essential before prescribing any program of exercise. The detection of ischaemic heart disease should be the focus of this assessment (Hogan, 1986). Underlying disease states should not be considered as a contraindication to exercise but as an indication of the requirement to modify the exercise program to suit the individual.

Aquatic or in-pool exercise programs have recently been cited as an effective means to maintain existing levels of fitness in injured runners (Koszuta, 1986). Water programs provide a means by which the major musculature can be exercised; the fluid medium provides an accommodating type of resistance and the buoyant forces decrease the impact forces imparted to
the participant. Conventional "walk-jog" programs may not be practical or even possible for the elderly individual who exhibits decreased mobility or gait problems as a result of orthopedic intervention or chronic arthritic conditions. Clinically and for the purpose of providing these individuals with a means by which they can increase their level of fitness in a safe and enjoyable manner, investigation into the potential of aquatic exercise programs is both warranted and necessary.

Water exercise has been shown to provide its benefits to strength, flexibility and cardiovascular conditioning independently of the participants' skill level and it reduces the likelihood of injury from overuse syndromes and heat-stress problems (Koszuta, 1986). Water exercise utilizes the water's resistance to achieve or maintain fitness: vigorous movements for a prescribed length of time account for the cardiovascular and respiratory benefits. The "aerobic" water exercise sessions are similar to the land-based classes in that choreographed movements are performed to music while an instructor leads from the pool deck or in the water. Classes usually last for one hour and are held in the shallow end of the pool. As the water is only chest deep, the participants need not be swimmers. Participants are encouraged to follow the instructor and they can vary the intensity or pace of their own workout. In addition, water acts as an equalizing medium as it provides patients who have arthritis or back problems an opportunity to engage in a supervised exercise program which may not be possible for them on dry land. In addition to the potential cardiovascular benefits, arthritics may benefit from this exercise as well particularly when range-of-motion exercises are included (Koszuta, 1986).

There have been few noteworthy studies concerning water exercise and the elderly. It will be the purpose of this study to investigate the impact of a 5 week program of aquatic exercise on selected cardiorespiratory parameters
in the elderly participant. The subjects will be culled from, and representative of, adult day-care and community centre facilities in the Lower Mainland. These people are, for the most part, unfit volunteers who all exhibit some degree of social impairment and isolation.
Numerous studies have attempted to describe the relationship between exercise and health (Belloc and Breslow, 1972) and researchers have shown interest in how the elderly feel about health and physical activity. One study characterized the beliefs that the elderly held concerning exercise and physical activity in general (Sidney and Shephard, 1976). Some of the attitudes included (1) they perceived the requirement for physical activity decreases with age; (2) exercise is dangerous; (3) light, sporadic exercise is beneficial to health; and (4) their own personal physical abilities were limited. The author's contention was that such attitudes may be the consequence of prevalent social attitudes which decree that an individual should "slow down" at retirement.

With aging, the older individual is faced with adapting to a variety of developmental changes (Riffle, 1982). These include an altered state of physical health, a possible increase in dependency level, dealing with the loss of a spouse and family members (siblings) and friends through death, and a possible change in living situation. Aging also tends to predispose people to less physical activity, presenting a situation that has numerous potentially negative effects upon both psychological and physiological functioning. Clinically, this could constitute a health threat (Riffle, 1982).

The elderly's perception of exertion appears to be implicated with the inclination for exercise. Studies (Sidney and Shephard, 1977; Sidney and Shephard, 1977b) have shown that when the Borg Psychophysical Scale was administered to volunteers (60-70 years of age) during a pre-retirement exercise program, the perception of physical exertion was 2-3 units higher than that perceived by younger adults (age 30-50) in previous studies. This
difference was noted at all heart rates. Physical training was not seen to significantly change this perceived exertion at any work load even though the resulting heart rates were substantially lower (Sidney and Shephard, 1977). It was also reported that men and women (the sample contained subjects 60-80 years of age) tended to overestimate time devoted to exercise, as continuously monitored heart rates did not coincide with the described activity (Sidney and Shephard, 1977). A substantial amount of time was actually devoted to activities which resulted in heart rates below 120 beats per minute.

The issues of whether poor health or a fear of exertion are important variables in the elderly’s attitudes concerning exercise have not been addressed. The reason(s) for tendency to engage in fewer physical activities with increasing age is/are not clear.

**Epidemiology**

These studies are of importance to the field of aging as they suggest a beneficial requirement for physical activity and a need to encourage it as part of a normal, healthy lifestyle. Originally, interest in physical fitness by the public was outlined by Hedley (1939). This study and subsequent studies indicated that the incidence of cardiovascular events, particularly myocardial infarction and sudden death, were lower in subjects routinely involved in physically active and demanding occupations when compared with individuals engaged in sedentary occupations. The historical conclusion is that physical inactivity is not as potent a risk factor as smoking, hypertension and/or hypercholesterolemia.

Morris et al. (1958, 1973) similarly reported rates of cardiac mortality in occupationally active subjects even though it was determined that "occupational selection" may have influenced the outcome. Regardless, Morris’ observations were substantiated at postmortem examination by the
appearance of differences in the type of lesions in the myocardium when comparing active and sedentary people. He concluded that even isolated "weekend" bouts of increased physical activity produced benefits not conferred to totally sedentary populations.

Active populations may exhibit higher incidences of varied symptoms of coronary heart disease even when the overall incidence of infarction and sudden death are lower (Naughton, 1982). Therefore, it is apparent that physical activity is not solely preventative of the development of coronary obstruction.

Kannel and Sorlie (1979) (the Framingham Study) typified physical inactivity as a risk factor in the development of ischaemic heart disease in men of all ages. To this end, The Framingham investigation provided the first emphatic health statement concerning the need to promote regular physical activity.

Paffenbarger's (1978) longitudinal study allowed controlled observations for the major coronary risk factors. The findings suggest that there is a direct relationship between occupational or leisure-time activity and the incidence of cardiac events.

Physiological Observations

Various population groups have been investigated. Studies have examined physically active subjects and determined the effects of physical conditioning on previously sedentary subjects. Regardless of the intent and design of the study, the findings have been consistent: physically active subjects are capable of a greater work capacity, have lower resting heart rates, have lower heart rates and systolic blood pressures in submaximal exercise than sedentary subjects (Naughton, 1982). Regular physical activity also promotes increased lean body mass (i.e. increased muscle mass with a
corresponding decrease in percent body fat.), improved glucose tolerance and improved insulin tolerance.

The influence of age on physical fitness in healthy subjects has been investigated by Naughton (1982), Robinson (1938) and Dill (1963). The findings have been consistent in that the level of physical fitness decreases with age in both men and women. At all ages, women exhibited lower levels of fitness than men. These studies were cross-sectional and biased by subject selection, particularly in the young subjects. Taylor and Montoye (1974) attempted to correct this by studying fitness levels reported in healthy population in relation to their activity patterns (vigorous, moderate or sedentary activity). Their findings indicated that differences in fitness based on activity levels existed across all ages, but that regardless of group assignment, the level of physical fitness decreased with advancing age. Alexander (1974) examined the decrease of fitness with age based on some of the reported observations. He found a decrease in physical fitness ranging from 0.93 to 1.04 ml oxygen per kg body weight per min with each successive year (when determined in relation to body weight). When the decrease was analyzed in relation to habitual physical activity status, the decline with advancing age was steeper for the sedentary than for the physically active subjects. Naughton and Nagle (1965), studying a healthy population of middle-aged men, did not find the usual high level of fitness in the younger subjects but a decrease with men in the population group studied. They found that the fitness levels were nearly identical across the four decades. This indicates that many young subjects in the general population may not develop a high level of fitness and that for the nonactive population the more normal level fitness is 32 ml oxygen per kg body weight per min or about 8 to 9 mets ( 1 met = approx. 3.5 ml oxygen per kg body weight per min.), instead of the significantly higher levels reported by other investigators. Eighteen
subjects were re-evaluated following conditioning and it was found that fitness levels had increased significantly regardless of age. Therefore, healthy individuals of all ages have a capacity for conditioning and an expected range of change is obtained. Cupelli et al. (1984) demonstrated that regular training provided a decreased resting heart rate (as an expression of decreased sympathetic stimulation of the sinoatrial node) and a greater efficiency while those who trained irregularly showed a decreased physical work capacity with higher systolic blood pressure values at rest. The authors also state that training induces a decrease in myocardial oxygen consumption. Their conclusions are that elderly subjects, particularly those with a higher basic blood pressure, should be encouraged to participate in aerobic activities.

Investigators have studied the effects of physical activity on serum lipids, particularly cholesterol, triglycerides and high-density lipoprotein cholesterol (HDL-C) (La Rosa et al., 1981). Physical activity itself has not been found to influence serum cholesterol, as weight loss corresponding to the activity program may cause the decline in cholesterol. As percent body fat is reduced (and lean body mass increased), triglyceride levels are seen to decrease. La Rosa (1981) concluded that HDL-C is not affected significantly by physical activity programs. It is suggested that prior studies that have shown a significant relationship between HDL-C and physical activity exhibit bias in subject selection. In contrast, Wood et al. (1977) had shown earlier that the average HDL level was 33% higher in men runners than in sedentary men and 25% higher in women runners than sedentary women. Leon et al. (1977) showed a significant increase in HDL levels among obese sedentary men on a walking program without dietary changes. Hartung et al. (1980) summarily described a progressive increase in HDL levels directly related to the quantity of exercise.
I CARDIOVASCULAR CHANGES WITH AGING - AN OVERVIEW

A gradual decline in all body systems is presumed with aging (Holm and Kirchhoff, 1984). Although exercise does help to retard cardiovascular decline with advancing age, it cannot prevent it. Studies have documented the diminution with age in the beneficial effects of exercise on the cardiovascular system (Montoye, 1982).

Blood pressure increases with age, with systolic blood pressure increasing to a greater extent than diastolic pressure. A decreased cardiac output response to exercise may be due to the intrinsic inability of the aging myocardium to respond to an additional load placed upon the ventricle by decrements in the vascular network. Holm and Kirchhoff (1984) state that the decrease in exercise stroke volume is primarily due to an increase in afterload and is not related to changes in preload ability and myocardial contractility. It is felt that the combination of decreases is maximal heart rate and exercise stroke volume reduces cardiac output (Yerg et al., 1985).

Yin et al., (1981) in an animal study, demonstrated a 20% increase in impedance and a 28% decrease in peripheral resistance with no increase in stroke volume in old animals (dogs) during graded treadmill exercise. In contrast, the younger animals showed no increase in impedance, a progressive decrease in peripheral resistance and a progressive increase in stroke volume at similar exercise levels. The marked difference in the older animals cardiovascular response to exercise resulted in a reduced cardiac output, heart rate, maximal exercise capacity and oxygen consumption.

Wenger (1981) outlined four features that ultimately limit the elderly individual's capacity to work: 1) a decrease in maximal oxygen consumption ($V_{O2max}$); 2) a decrease in maximal heart rate, 3) a decrease in the exercise stroke volume, and 4) a resultant decrease in cardiac output. A decline in $V_{O2max}$ has been demonstrated in many studies, both cross-sectionally (Hossack
and Bruce, 1982; Strandell, 1963) and longitudinally (Astrand et al., 1973; Robinson et al., 1975). This decline in $VO_{2\text{max}}$ is linear throughout the adult years (McArdle et al., 1981) and is estimated to amount to a total loss of 30-40% in the 65 year old compared to the young adult (Shephard, 1978).

From age 20 years to 45 years, there is little age difference seen in $VO_2$ during incremental graded treadmill walking (Montoye, 1982). Beyond age 45 years, the increase in $VO_2$ is slight, although statistically significant and represents a small decrease in walking efficiency. Hanson et al. (1968) and Adams et al (1969) reported no age differences in $VO_2$ among adults walking at submaximal work loads. On a cycle ergometer, the oxygen uptake during submaximal exercise was not related to age in adults (Julius et al., 1967). This is also true for ergometry (Durnin and Mikulicic, 1956) although Norris et al. (1955) reported a higher oxygen requirement for older subjects in arm work. Along with a demonstrated decline in maximal heart rate, it can be seen that the heart loses its efficiency as a pumping device with age (Whitbourne, 1985). Both central and peripheral age-related changes are responsible for the reported age losses in maximum heart rate and oxygen consumption.

A reduction in the left ventricular ejection volume is seen with age (Whitbourne, 1985). This potentiates a decreased stroke volume and results in a decreased cardiac output (Hossack and Bruce, 1982; Julius et al., 1967; Port et al., 1980; Robinson et al., 1975). Maximum oxygen consumption ($VO_{2\text{max}}$) is therefore reduced, as the resulting decrease in blood flow compromises arterial flow and subsequent tissue oxygen extraction. Ventricular efficiency is reduced due to increased wall thickness and an overall increase in ventricular mass (Gerstenblith, 1980; Lakatta, 1979). The effect of these anatomical changes is seen with an increase in the time required to complete the left ventricular cardiac cycle (Granath et al.,
1970; McArdle et al., 1981; Port et al, 1980). A reduction in myocardial contractility is observed (Whitbourne, 1985). Electrocardiographic changes and/or abnormalities may be noted during exercise; S-T segment depression being the most common (Bengsston et al., 1978; Montoye, 1975). Therefore, it is apparent that a temporary, exercise-induced ischaemia may occur and the involved musculature may be inadequately supplied with oxygen.

Several investigations support the hypothesis that peripheral factors are responsible for a decline in cardiac function. One suggestion is that the peripheral vascular system increases systemic resistance to blood flow, this being due to an increased rigidity of the arterial walls. The result is a decreased ability to accommodate systolic flow surges and an overall increase in blood flow during exercise (Brooks and Fahey, 1984; Shephard and Sidney, 1978). This mechanism could account for the higher resting systolic and diastolic blood pressures seen in older age populations (Sato et al., 1981) and during submaximal (Bengsston et al., 1978) and maximal exercise (Bengsston et al., 1978).

A reduced overall muscle mass may also account for a decreased $V_{O_2}^{\text{max}}$ in the elderly. Less oxygen is extracted by the muscles as there are fewer skeletal muscles requiring oxygen during exercise (Brooks and Fahey, 1984; McArdle et al., 1981).

With advancing age, alterations occur in the cardiovascular system. What proportion of these changes is due to the process of aging alone and what is due to environmental influences is unknown.

A. **Maximal Exercise**

1. **Oxygen consumption ($V_{O_2}^{\text{max}}$)**

The maximal capacity of the cardiovascular system to deliver oxygen to the working muscles has been shown to decrease with age. This has been found
in longitudinal (Dawson and Hellebrandt, 1945) and in cross-sectional studies (Astrand, 1960; Robinson, 1938); estimates of this reduction range from 21 to 30% over a 30 to 40 year range. Conversely, VO$_{2\text{max}}$ has been increased 10% after 2 months (Mazzarella et al., 1966) and 17% after 6 months (Robinson, 1938) of endurance training. Naughton (1982), Robinson (1938) and Dill (1963) examined the influence of age on physical fitness (as measured by VO$_{2\text{max}}$) and it was seen to decrease with advancing age in both men and women. Women were also seen to exhibit a lower level of fitness than men at all ages. Montoye (1974) found that differences in fitness based on activity levels existed across all ages and that the levels of physical fitness decreased with advancing age. Alexander (1974) found a decrease in VO$_{2\text{max}}$ with age ranging from 0.93 to 1.04 ml of oxygen per kg of body weight per minute with each advancing year (when determined in relation to body weight). Naughton and Nagle (1965), examining a healthy population of middle-aged men, found that the younger subjects did not necessarily exhibit a higher level of fitness than their older counterparts. A decrease in VO$_{2\text{max}}$ with age was not seen; fitness levels were nearly identical across the four representative decades. This indicated that many young subjects in the general population may not develop a high level of fitness and that for the nonactive population the more normal level of fitness is 32 ml of oxygen per body weight per min, or about 8-9 mets (1 met = approximately 3.5 ml oxygen per kg body weight per min), instead of the significantly higher levels reported by other investigators.

The rate of decline in VO$_{2\text{max}}$ seems to be greater in one time elite athletes who have become sedentary in comparison with their untrained counterparts who have remained sedentary and untrained (Robinson, 1964). Such is not the case with the athlete who remains active into old age; Clarence DeMar had a VO$_{2\text{max}}$ of 60 ml kg$^{-1}$ min$^{-1}$ at 49 years of age only
somewhat below a mean value of 17-20 year old males of 52.0 ml kg\(^{-1}\) min\(^{-1}\) (Dill, 1965). In a series of athletes ages 42 to 68 years, (Grimby and Saltin, 1966), \(V_{O_2\text{max}}\) was 30% higher than in non-athletes of comparable age at all ages.

\(V_{O_2\text{max}}\) per unit of lean body mass (LBM) shows a similar decline. Fischer et al. (1965) noted that although \(V_{O_2\text{max}}\) (in ml kg\(^{-1}\) min\(^{-1}\) LBM) declines with age, active subjects in their seventh decade had significantly higher values than their sedentary counterparts.

2. Heart Rate

Maximal heart rate (HR\(_{\text{max}}\)) is seen to decrease with age in both men and women (Astrand, 1960; Robinson 1938; Astrand, 1973; Julius, 1967). For a given increase in oxygen consumption over basal levels, heart rate remains constant or declines with age (Cotes, 1974; Granath, 1964). The effect of training has been found to be negligible in some cases whereas other investigators have found a decrease in heart rate. Mazzarella et al. (1966) reported little or no change following a 2 month exercise program. Skinner et al. (1964) reported the same findings following a 6 month program (endurance training with middle-aged men). Older athletes have shown a decreased HR\(_{\text{max}}\) which is not unlike that found in untrained men of the same age (Astrand, 1956). Seals et al. (1984) found that heart rate after training was reduced at the same absolute work rates and unchanged at the same relative work rates. Although the heart rate of the older subject physiologically decreases, the maximal heart rate and the heart rate at rest following training provide indications of vagotony (Granath et al., 1970; Zoneraich and Rhee, 1977).

With age, there are noted changes in the effect of exercise on systolic blood pressure and heart rate. With increasing age, systolic blood pressure tends to be higher at a given intensity of exercise (Bevegard, 1967; Julius,
1967). Maximum heart rate falls (Astrand, 1973; Julius, 1967), but at a submaximal exercise level, heart rate is greater in the older subject (Astrand, 1973). For a given increase in oxygen consumption over basal levels, heart rate remains constant or declines with age (Cotes, 1974; Granath, 1964). Rowlands et al. (1984) using a sample of 33 subjects (including hypertensives), found that during dynamic (walking) exercise there was a significant increase in blood pressure and heart rate.

3. Stroke Volume

There appears to be a slight decrease in maximal stroke volume ($SV_{\text{max}}$) with increased age (Astrand, 1967). Granath et al. (1964) found a diminished SV in very old, untrained men. An increased SV has been found in some middle-aged and older athletes (Grimby and Saltin, 1966). A decrease in SV has been reported to occur in older individuals at near maximal work rates (Port et al., 1980).

4. Cardiac Output

Since maximal HR decreases and SV is reduced to a commensurate degree with aging, maximal cardiac output decreases also (Granath et al., 1960). A decrease in CO has been reported by a series of studies in which SV and/or HR are directly or indirectly measured (Brandfonbrener et al., 1955; Hossack and Bruce, 1982; Julius et al., 1967; Port et al., 1980; Robinson et al., 1975).

Grimby and Saltin (1966) found little difference between the maximal CO of young and old athletes, but both had higher values than did untrained men of the same age.

B. Submaximal Exercise

1. Heart Rate

Although the maximal HR is seen to decrease with age, there is little difference in the HR at a given submaximal workload (Asmussen and Mathiasen,
1962; Robinson, 1938; Strandell, 1964). At a submaximal exercise level, heart rate is greater in the older subject (Astrand, 1973). However, with training, HR is seen to be decreased at a given level of submaximal work (Barry et al., 1966; Skinner et al., 1964). An elevated HR while performing a standard work task is found in sedentary individuals compared to athletes or physically active men (Taylor et al., 1963).

It takes longer for the circulatory (and respiratory) systems of the older person to adapt to a workload (Robinson, 1938). More time is required for their HR to return to the resting level following exercise (Astrand, 1967). Following a six-month endurance training program, significant reductions in the recovery HR of middle-aged men were seen (Skinner et al., 1964).

2. Systolic Blood Pressure

The systolic BP at fixed submaximal workloads has been shown to be higher in older persons (Norris et al., 1953; Robinson, 1938). Endurance training has produced significant reductions in the systolic BP of previously sedentary middle-aged subjects (Skinner et al., 1964) and elderly subjects (Barry et al., 1966).

C. Aging of the Cardiovascular System: Effects of Exercise - A Summary

The major dependent variables of interest in training studies are aerobic capacity (maximum oxygen consumption) and heart rate under conditions of maximum exercise (Whitbourne, 1985). Various investigations have examined the same parameters under submaximal exercise conditions. Submaximal studies have progressed from the premise that a more efficient cardiovascular system will require less effort (as indicated by a lower heart rate and increased stroke volume and cardiac output) to perform successfully at a given level of oxygen consumption.
1. Effects of Physical Training on $VO_{2\text{max}}$

Naughton and Nagle (1965) found that fitness levels (i.e. $VO_{2\text{max}}$) increase significantly regardless of age with training. It appears that healthy individuals of all ages have a capacity for training and increased fitness and an expected rate of change is obtained. Regularly performed endurance exercise increases $VO_{2\text{max}}$ in both the young (Astrand, 1964; Hickson et al., 1981; Seals et al., 1983) and middle-aged (Hanson et al., 1968; Pollock et al., 1971). Endurance training could, therefore, reverse the age-related decline in aerobic capacity if older individuals undergo an adaptive increase in $VO_{2\text{max}}$ (Seals et al., 1984). Previous investigations have indicated little or no change in $VO_{2\text{max}}$ in response to training in individuals over 60 years of age (Niinimaa and Shephard, 1978; Pollock, 1973), yet increases in $VO_{2\text{max}}$ varying from 0.0 to 38.0 percent as a result of training have been reported in the elderly (Barry et al., 1966; Berestad, 1965) Thomas et al., 1985, examining 88 elderly men (mean age 63 years), found an average increase of 12 percent in $VO_{2\text{max}}$.

Few studies have investigated the training-induced adaptations in the determination of $VO_{2\text{max}}$ (i.e., maximal stroke volume, maximal heart rate and maximal arteriovenous oxygen difference) in middle-aged and older individuals (Seals, 1984). Hartley et al. (1969) reported that a 14 percent increase in $VO_{2\text{MAX}}$ was due to an increased maximal stroke volume and cardiac output in the elderly, whereas the 15 percent increase in $VO_{2\text{max}}$ after training observed in the studies concerning younger men was obtained as a result of increases in maximal cardiac output and maximal arteriovenous oxygen difference. They attributed the lack of marked change in $VO_{2\text{max}}$ subsequent to training in middle-aged and older subjects to their ability to elicit significant increases in maximal arteriovenous oxygen difference. Seals et al. (1984), in contrast to Hartley et al. (1969), suggest that the increase
in the VO\textsubscript{2max} of their subjects appears to have been primarily due to adaptations in the skeletal muscle resulting in an improved ability to extract oxygen, as reflected in a higher maximal arteriovenous oxygen difference. The adaptations of skeletal muscle to endurance exercise includes both an increase in mitochondria and an increase in capillary density, both of which could account for the increase in oxygen extraction (Holloszy et al., 1977; Saltin et al., 1977).

Kiessling et al., (1974), however, state that mitochondrial volume is unaffected. It appears that increased peripheral circulation is potentiated by changes in the arterial blood flow.

2. Intensity and Duration of Exercise Training

Conflicting conclusions about the effect of training intensity in the elderly have been reported. Badenhop et al. (1983) reported that both high or low intensity training produced the same increase in VO\textsubscript{2max}, while others (Seals et al., 1984; Sidney and Shephard, 1978) found that high intensity training resulted in much larger gains than were seen with a low intensity program. Seals et al. (1984) also determined that in older men and women, six months of high intensity training (75-80% of maximum age-adjusted heart rate) produced larger gains in the directly measured VO\textsubscript{2max} than lower intensity training of the same duration. Sidney and Shephard (1978) reported that walking or jogging at a high intensity (heart rates of 140 to 150 beats per minute) produced larger gains than at low intensity (heart rate of 120 to 130 beats per minute).

Previous studies on older subjects have usually involved only mild to moderate intensity exercise for relatively brief periods (6-12 weeks) (Berestad, 1965; Niimaa and Shephard, 1978). Failure to previously observe significant improvements in VO\textsubscript{2max} in older subjects may have been the result of an inadequate training stimulus (Seals et al., 1984). Emes (1979) found
that a light program of exercise consisting of three 45-minute sessions per week for 12 weeks resulted in noted differences in pre-post measures of systolic and diastolic blood pressures, weight and resting rate. Seals et al. (1984) determined that 12 months of endurance exercise training can elicit increases in VO$_{2\text{max}}$ in healthy older men and women of equal of greater magnitude than have been previously reported for younger (Astrand, 1964; Pollock et al., 1971; Seals et al., 1983) and middle-aged (Hanson et al., 1968; Pollock et al., 1971) populations. Their findings indicate that a moderate increase in daily physical activity of a six month period can result in a small but significant increase in VO$_{2\text{max}}$. The 25-30 percent increase in directly measured VO$_{2\text{max}}$ after 12 months of the same program exceeds the responses previously reported for subjects 60 years of age and over (Berestad, 1965; Suominen et al., 1977). This suggests that the training stimuli in earlier studies were of insufficient intensity and/or duration to elicit the adaptations resulting in an increased VO$_{2\text{max}}$.

Thomas et al. (1985), examining 88 elderly men (mean age 63 years), found an average increase of 12 percent in VO$_{2\text{max}}$ following 12 months of training. The investigators stated that the best predictor of what an elderly subject's VO$_{2\text{max}}$ would be after one year of training is the initial VO$_{2\text{max}}$. DeVries (1971) examined the relation of change in VO$_{2\text{max}}$ estimated from a submaximal exercise test with training intensity and initial fitness. Multiple regression analysis indicated that 41 percent of the variance in the change score could be explained by the initial predicted VO$_{2\text{max}}$ and the percentage of heart rate range at which the subject trained.

3. Long-Term Training Effects

Prior athletic training does not preclude an individual to the age related decrements in aerobic capacity (Robinson et al., 1973). It appears that continued exercise throughout the middle adult years prevents age losses
in aerobic capacity and perhaps even reverses them (Whitbourne, 1985). Significant increases in $VO^{\text{max}}$ in the 13 years between testing in one study by Robinson et al. (1975) were seen only in those subjects who continued to exercise regularly throughout the duration of the study. Subjects who trained and then stopped showed the same rate of loss of aerobic power as the others in the longitudinal sample. Endurance athletes, such as orienteers, skiers and other long-distance athletes have been shown to have much larger aerobic capacities than their sedentary counterparts, even those that are considerably younger (Suominen et al., 1980; Cumming, 1967; Gollnick et al., 1972).

The literature supports the contention that champion or master's athletes who continue to compete have higher levels of $VO^{\text{max}}$ than sedentary adults of the same age and younger (Heath et al., 1981; Kavanagh and Shephard, 1978). Further studies on older adults who have remained active throughout their middle years have provided additional support for the advantage provided by continued exercise in that their subjects have the functional capacity of sedentary individuals who are 10 to 20 years younger (Plowman et al., 1979; Wright et al., 1982).

Seals et al. (1984) found that heart rate after training was reduced at the same absolute work rates and unchanged at the same relative work rates. Equivalent or larger reductions in heart rate at the same absolute work rates occurred in response to low intensity training compared with high intensity training. Low intensity training was six months of unsupervised walking for 20-30 minutes at a heart rate of 120 beats/min at least three times per week. High intensity training was six months of supervised endurance exercise, progressing from walking to cycling ergometer or graded treadmill as their fitness improved. The choice of training modality depended upon the subject's orthopedic status. The duration and the intensity of the exercise
progressed from 30 minutes at 75 percent of the maximal heart rate to 45 minutes at 85 percent of the maximal heart rate (156 ± 6 beats/min). Although the heart rate of the older subject physiologically decreases, the maximal heart rate and the heart rate at rest following training provide indications of vagotony (Granath et al., 1970; Zoneraich and Rhee, 1977).

In summary, the research indicates that there is a significant potential benefit in aerobic exercise as it increases the aging cardiovascular system's ability to transport blood to meet tissue requirements. Continued and ongoing involvement in endurance activities appear to compensate for the age-related functional losses normally seen. The highly trained older person possesses a left ventricle that is capable of a high level of contractility thereby ensuring a large stroke volume and cardiac output, particularly during exhausting work (de Vries, 1980).

4. Short-Term Training Effects

The negative effect of aging on the maximum oxygen consumption can be offset by aerobic exercise training. The normal loss of $V_{O_{2}\text{max}}$ over the adult age span is estimated to be 1% per year (Brandfonbrener et al., 1955), or 40% between 25 and 65 years of age, but this loss can be reduced by a maximum 50% in any 2 to 3 month training study in which the participants train 3 hours per week at 60% or more of their aerobic capacity (Hodgson and Buskirk, 1977). Most of the research in which this effect is seen involves subjects not older than 65 years of age (Whitbourne, 1985). When the intensity of the exercise is sufficiently strenuous, men and women in their 70s have demonstrated an increase in $V_{O_{2}\text{max}}$ (de Vries, 1980; Whitbourne, 1985).

Studies have generally not shown a favorable training effect on maximum heart rate in middle-aged and older adults (Hartley et al., 1969), but a decreased heart rate during submaximal exercise has been seen (Blumenthal et
Cardiac output at submaximal work levels remains constant in older adults despite training (Rost et al., 1979). Maximum cardiac output is increased by training in middle-aged and older adults (Skinner, 1970). As the maximum heart rate is not altered by training, an increased cardiac output indicates an improved functioning of the left ventricle during maximal work, thereby ensuring a greater stroke volume.

Submaximal exercise in the elderly produces a maximal stroke volume similar to that attained during moderate submaximal exercise in young subjects (Astrand et al., 1964; Hartley et al., 1969; Rodeheffer et al., 1984). A decrease in stroke volume has been reported to occur in older individuals at near maximal work rates (Port et al., 1980).

In general, the adaptions observed during submaximal exercise after training are qualitatively similar to those reported previously in younger populations (Astrand and Rodahl, 1977; Ekblom et al., 1968; Hartley et al., 1969).

II RESPIRATORY CHANGES WITH AGING - AN OVERVIEW

The literature contends that with age there is a reduction in the amount of oxygen that is taken up by the tissues during exercise. The structure(s) of the respiratory system are rendered less efficient in the older person, resulting in an overall decreased efficiency of gaseous exchange in the lungs. The aging respiratory system adds to the limitations of the aging cardiovascular and muscular systems (Whitbourne, 1985).

A. Gaseous Exchange and Ventilation

The functional measure representing the efficiency of diffusion across the alveolar capillary interface is the difference between the arterial oxygen pressure compared to the alveolar oxygen pressure. Studies have
documented the drop in arterial oxygen pressure and/or the increase in the alveolar-arterial oxygen difference with age (Marshall and Wycke, 1972; Melmgaard, 1966). The explanation for this reduced oxygen transport may be an increased resistance to oxygen diffusion across alveolar and capillary membranes (Anderson and Shephard, 1969; Mauderly, 1978).

Robinson (1938) reported that the respiratory exchange ratio during moderate work on a treadmill increased from ages 8 to 63 years. Julius et al. (1967) confirmed this in subjects aged 18 to 68 years. Wessel et al. (1968) reported a slight increase in respiratory exchange ratio from ages 20 to 59 years and then a decrease in the 60 to 69 year age group. These differences were not statistically significant and the exercise values were thought to be low. Rulli and Menotti (1969) also reported no statistically significant relationship between respiratory exchange ratio and age.

Wessel et al. (1968) and Rulli and Menotti (1969) reported no relationship between age and ventilation ($V_E$) during submaximal exercise. Robinson et al. (1973) and Patrick et al. (1983) showed an increase in $V_E$ with age as a result of an increased tidal volume whereas Harris and Thomson (1958) attributed the increase in $V_E$ to higher respiratory rates in older subjects.

B. **Dynamic and Static Ventilatory Volumes**

Lung function may be quantified by the efficiency by which the lungs and their associated structures can move air volume. Measurements of this efficiency may describe the components of the total lung capacity to hold air or describe the volumes of air that can be moved during a period of time. Lower values during submaximal work indicate a greater respiratory efficiency, in terms of supplying oxygen, with a minimum of energy cost. High levels of function on these measures indicate that the lungs can support the oxygen requirements for the working muscles. The functional measurements
of the lung based on lung volumes are useful for describing age differences in the pulmonary response to exercise (Whitbourne, 1985).

With age, there appears to be a reduction in vital capacity accompanied by an increase in residual volume (Asmussen et al., 1975). This process begins approximately at age 40 (Brady et al., 1974) and results in a total of 40% loss of vital capacity between the ages of 20 and 70 (Lynne-Davies, 1977). Age changes in compliance are largely responsible for an increased closing volume, and it has been suggested that the same process would account for the age-related increase in the residual volume (and accompanying decrease in vital capacity) (Whitbourne, 1985).

At submaximal work loads, a low ventilatory rate is desirable. The literature is confusing as to the age effects on ventilatory rate. Some investigators show no change with age (Denolin et al., 1970), a decrease (de Vries and Adams, 1972) or an increase (Robinson et al., 1975). Older individuals are less able to maintain high rates of ventilation at maximal levels of exercise (Saltin and Grimby, 1968). A decreased chest wall compliance with age is a contributing factor (Baldwin et al., 1948). Since age has not been shown to affect tidal volume under conditions of maximal exercise, it has been suggested that the decreased maximum frequency of breathing with age is responsible for the demonstrated age loss in maximum ventilatory rate (Montoye, 1982).

Forced expiratory volume (1 sec.) is an index and measure of flow rate. The flow rate is reduced with age as it is dependent upon the elastic qualities of the lungs (Whitbourne, 1985). There appears to be a decrease in flow rate (as expressed by forced expiratory volume) with age (Kannel and Hubert, 1982; Shephard and Sidney, 1978).

Ventilatory efficiency (rate of ventilation/oxygen uptake) also reflects the efficiency of the oxygen transport mechanisms of the body (de Vries,
1980). At submaximal levels, the lungs of older adults are less effective in oxygen transport, this being indicated by a higher ventilatory efficiency index (Robinson et al., 1975). Concurrently, the maximum level of ventilatory efficiency is reduced with age, suggesting that the older individual is less able to provide adequate levels of oxygen at maximal levels of work, as ventilation is compromised.

The responsivity of the respiratory response to hypoxia is reduced with age (Petersen et al., 1981). Structural and/or functional changes in the respiratory system or the neuromuscular control over ventilation may be responsible. It has been suggested that a decreased neural output to the respiratory muscles under conditions of hypoxia is the major reason for this age effect (Petersen et al., 1981). There is, however, evidence to show increased sympathetic activity in the older individual. It is possible, then, that the decreased response to hypoxia with age is a function of the inability of the lungs and chest wall to respond to sympathetic stimulation (Whitbourne, 1985).

The respiratory control of breathing in response to pressure variations in carbon dioxide has not been seen to change with age (Rubin et al., 1982).

C. Airway Closure and Compliance

There appears to be an increasing non-uniformity in the distribution of air in the lungs with age, resulting in a reduced oxygen concentration in the arterial blood. This non-uniformity of air distribution results in a discrepancy between ventilatory and perfusion rates (Edelman et al., 1968). Such a discrepancy is also prevalent in young adults, but it appears that in this age group, the efficiency of gaseous exchange is not compromised. The lung of older individuals yields a greater total area of inequality between ventilation and perfusion. The result is a decreased oxygenation of the blood leaving the lungs (West, 1977). At all ages, the total quality of
blood leaving the lungs contains a large volume of blood from the lower lung. In the older person, this blood is not well-oxygenated. This is due to a reduced ventilation in the lower aspect of the lung due to the decreased elasticity of the tissue. A decreased elasticity promotes premature airway closure in expiration, thereby allowing unexpired air to be trapped inside. Gravitational forces also cause a discrepancy in the recoil activity in the upper and lower lung (the pressure over the lung surface is more positive, by approximately 7.5 cm H₂O in comparison to the lower lung). As the lung tissue loses elasticity, it is the bottom regions that are more affected. The result is that the alveoli in the lower aspect of the aging person's lung are likely to be underventilated, relative to the pulmonary capillary blood flow (Begin et al., 1975; Bode et al., 1976). A lower elastic recoil with age is indicated by less pressure across the lung surface at a given lung volume. In addition, lung compliance (the volume increase for each pressure increase) is seen to be greater in older adults. An increased compliance with age is indicative of the decreased resistance to lung expansion in inspiration (Whitbourne, 1985).

Alterations in alveolar structure are seen with age. Elastic and collagenous integrity are compromised (Brandstetter and Kazenni, 1983; Lynne-Davies, 1977) and alveolar ducts and alveoli increase in size (Reid, 1967). This results in a decreased total number of alveoli and functional surface area.

Aging also corresponds with an increase in the rigidity of the chest wall structures (Shephard and Sidney, 1978). This, along with the decreased elasticity of the lung, further compromises the ability of the lung to compress in expiration and expand during inspiration, and increases the amount of work required by the respiratory musculature in the ventilation cycle (Brooks and Fahey, 1984).
D. **Pulmonary Hemodynamics**

The normal range of pulmonary arterial pressures gradually increases with age. It has been determined that the mean resting arterial pulmonary pressure increases 0.8 mm Hg per decade after the age of 40. Diastolic arterial pulmonary pressure increases 0.7 mm Hg per decade while systolic arterial pulmonary pressure is not seen to change significantly (Perrault et al., 1969).

Tartfulier et al., (1972) found that during supine exercise, the observed relationship between pulmonary artery pressures (systolic, diastolic and mean) and cardiac output were linear and were seen to increase with age. It was seen that the pulmonary capillary wedge pressure increased with age and is responsible for the age-related increase in pulmonary artery pressures during supine exercise. The pressure gradient across the pulmonary vascular bed (PAP-PCW) remains constant, indicating that pulmonary vascular resistance exhibits little increase with age (Palevsky, 1986).

E. **Effects of Exercise Training on the Respiratory System**

The only muscular tissue in the respiratory system that can be strengthened by training are the muscles that control ventilation (inspiration and expiration). The cardiovascular system, by contrast, has a greater potential for the reversal of age effects due to the trainability of the myocardium (Whitbourne, 1985).

The long term effects of chronic inactivity have not been investigated to test the possibility that decreased levels of participation in exercise contribute to age-related decrements in respiratory function (Whitbourne, 1985).

Athletes and former athletes show less of a decline in vital capacity with age than sedentary adults (Plowman et al., 1979; Robinson et al., 1973). This finding has not always been consistent with all studies (Astrand, 1973).
Short-term studies have not shown a positive change in vital capacity with aerobic training (Niinimaa and Shephard, 1978; Pollock et al., 1976), although an increase in vital capacity at maximum levels of exertion was seen in an 8-week training program in which both young and middle-aged men and women participated (Heikkenen, 1978). It is, therefore, not clear whether exercise can enhance vital capacity or whether it is other variables such as lifestyle that may be more important. Whitbourne (1985) states that exercise may have the greatest effect on vital capacity on those no older than 50-60 years of age.

Ventilatory rate (tidal volume x respiratory frequency), known to decline with age, is modified by both long and short-term exercise programs. However, this seems to be true only for ventilatory rates measured at maximum (not submaximal) levels of exertion. Training, then, increases the potential maximum rate of ventilation (Astrand, 1973; de Vries, 1970). This may be as a result of an increased maximum breathing frequency (Saltin et al., 1969) or an increased tidal volume (de Vries, 1970; Robinson et al., 1973).

Ventilatory flow is seen to increase in middle-aged and older adults with training (Whitbourne, 1985). It has been established that the total volume of air inspired and expired during a maximum ventilatory effort is greater in athletes and former athletes than their sedentary counterparts at comparable ages (Saltin and Grimby, 1968). The amount of air forcibly expired during a maximum respiratory attempt was found to be higher in athletes and former athletes. Improvements with training have not been shown (Pollock et al., 1976).

Since the maximum rate of ventilation and ventilatory flow can be increased by exercise training, one may expect the index of ventilatory efficiency (ventilatory rates relative to oxygen consumption) to increase also. Training has, however, no effect on the ventilatory efficiency index.
at either maximal or submaximal levels of exercise (Shephard and Sidney, 1978; Stanford, 1974).

Exercise training enables the middle-aged and older adult to increase the rate of breathing to levels that meet the demands presented by maximal levels of work. Training has, however, no effect on lung structure and volume: tidal volume, vital capacity and ventilatory rate (measurements dependent upon lung volume), do not show positive change(s) with training (Whitbourne, 1985).

III THE PHYSIOLOGICAL RESPONSE TO AQUATIC EXERCISE

There is an increasing amount of literature available that describes various water exercise programs. Many have been evaluated as to their worth in providing adequate components of fitness. These components include potential cardiovascular benefits, flexibility, strength, body composition, inclusion of warm-up and cool-downs and safety precautions (Koszuta, 1986). Based on the adequacy that these criteria are met, 11 of the 15 programs evaluated were deemed satisfactory. Whether the various books, videos and innovations are beneficial in terms of satisfying the above criteria may be inconsequential; it is felt by some that any exercise aid or program that will not cause injury may be helpful as a means of increasing the motivation to exercise (Koszuta, 1986).

Attempts have been made to determine the difference in metabolic demands between exercise in water and on land. Costill (1971) found that subjects in water could perform 66.7% to 71.4% of the total amount of work performed on land. The oxygen uptake during submaximal work was found to be significantly higher in the water exercise conditions. When comparing the caloric cost of submaximal water work in water to that of exercising on land, water exercise was found to increase the energy requirements by 33% to 42% for any given
work level. In response to the increased caloric demand in water exercise, heart rates were also higher at all levels of submaximal exercise. Water resistance appears to be responsible for a mean increase of approximately 34% in the energy requirements during submaximal exercise.

Vickery et al. (1983) presented the first physiological response data concerning water "aerobic" exercise ("aqua dynamics"). They proceeded from the premise that it was not known whether an exercise program consisting primarily of aquatic calisthenics offered sufficient intensity to elicit acute training effects. The heart rate and energy expenditure of 3 college women were determined during three "Aqua Dynamics" workouts, ranging from 20 minutes to 60 minutes in duration. Each workout consisted of a series of water exercises performed consecutively without stopping, followed by lap swimming. The exercise included warm-up stretching, twisting, simulated crawl swimming, running in place, kicking and treading water. Fluctuations in measured heart rate during the course of the workout suggest that individual exercise varied in intensity. Kicking movements and bobbing movements produced the highest heart rates and lap swimming produced the highest heart rate and oxygen uptake. All three workouts were found to be of moderate intensity, producing average heart rates of 132 to 152 beats/minute (or 70% to 77% of the maximal age-adjusted heart rate), average oxygen uptakes of 1.2 to 1.3 liters/minute (or 51% to 57% of VO$_{2\text{max}}$), and average rates of energy expenditure of 5.9 to 65 kcals/minute. It was found that the difference between the three workouts was total calorie expenditure and duration of exercise and not the intensity of work. The authors concluded that although some exercises in the workout produced a lower than required intensity of work, the overall response to this type of exercise suggests that regular and sufficient participation would probably improve physical
work capacity, particularly in those individuals with relatively low physical work capacities.

Koszuta (1986) describes a study in which six aerobically fit male subjects participated in an aquatic exercise program. The three routines (at 21, 36 and 46 minutes in duration) were similar to the conventional "aerobic" classes on land. Heart rates and mean oxygen uptakes were maintained at the lower end of the recommended ranges for fit athletes (American College of Sports Medicine, 1978). The investigators concluded that individuals with low to average aerobic capacity would likely receive cardiovascular and weight control benefits from such a program.

It appears then, that despite an inadequacy in the number of investigations examining this type of exercise, there is a general consensus that water exercise can be regarded as a viable and effective form of exercise and it may be particularly suited for those individuals with low levels of fitness.
METHODS AND PROCEDURES

Fifteen subjects (8 females and 7 males, mean age 68.5 years, range 61-75 years) were recruited voluntarily from regional community adult day-care facilities. All subjects were fully informed of the risks and potential discomfort associated with the testing procedures and participation in the program before giving their consent. All subjects were required to undergo medical screening and clearance by their physicians. The subjects were required to demonstrate an absence of:

1. recent illness requiring hospitalization,
2. chronic (debilitating) arthritis or related joint disorders,
3. uncontrolled diabetes,
4. uncontrolled hypertension,
5. symptomatic cardiovascular disease,
6. renal/hepatic disease,
7. neurological disease (pronounced decline in motor abilities and/or mentition).

(see Appendix F for contraindications for exercise participation and testing). All subjects completed forms of informed consent prior to entry into the program (see Appendices G and H).

Participants in the study underwent a series of physiological tests before the program started and 5 weeks later at the end of the program. The results of these physiological measurements and physical parameters at the start of the program are given in Table II. Measurements included height, weight, a 5-lead exercise electrocardiogram, spirometry measurements (forced vital capacity (FVC), forced expiratory volume in 1 second (FEV\textsuperscript{1.0}), maximum ventilation ($V_{E_{max}}$)), resting blood pressure (systolic/diastolic), resting heart rate, exercise heart rate and $V_{O_{2max}}$ as determined by a continuous
treadmill test (modified after Jones and Campbell, 1982). The protocol consisted of a 3 minute warm-up walk at 2.0 mph (0% grade). Treadmill speed was then increased to 2.5 mph with 2% increases in elevation every 2 minutes. The reliability of $V_{O2\max}$ determinations in elderly subjects has been described by Thomas et al. (1985) with the correlation coefficients between a first stage I (Jones and Campbell) test and a second stage I test one year later being 0.90.

The criteria for stopping the exercise test was modified from that suggested by Cunningham and Rechnitzer (1974). All subjects were encouraged to continue the test until they i) attained their maximal heart rate and/or, ii) fatigue (breathlessness, claudication) ensued.

Subjects were acquainted with all tests and treadmill protocol during initial orientation sessions at the J.M. Buchanan Research and Fitness Center at the University of British Columbia. Testing was done on successive visits to the facility. Blood pressures were obtained by auscultation. Resting heart rates were obtained by a radial pulse and exercise (testing) heart rates were continuously monitored by direct ECG. Forced expiratory volume in 1 second ($FEV_{1.0}$) and forced vital capacity (FVC) were recorded as the highest of three trials (after Thomas et al., 1985). Expired gases were continuously collected and analyzed by a Beckman Metabolic Measurement Cart (BMMC) interfaced with a Hewlett-Packard 3052A Data Acquisition System for 15 second determinations of respiratory gas exchange variables. The accuracy of this means of data collection has been deemed highly satisfactory by Montoye et al. (1970). The analysis of expired gases for each subject was preceded by the calibration of the CO$_2$ and O$_2$ analyzers with a calibration gas. Maximum oxygen consumption, maximum ventilation and maximum respiratory exchange ratios were determined by averaging the highest 2 consecutive fifteen second values.
Exercise Program

Subjects were randomly assigned to either control (n = 7) or experimental (n = 8) groups. Females were assigned to groups first and then males to ensure equal representation of sex in each group. The exercise sessions for the experimental group were held on three (3) mornings per week, on non-consecutive days for a 5 week period at the UBC Aquatic Center. A total of seventeen (17) sessions were attended by the experimental group.

Each exercise session began with 25 minutes of strength and flexibility movements on dry land which included torso, arm and shoulder exercises while standing and mat work where each subject performed additional stretching movements for the musculature in the back and legs, ending with a cool-down period. The subjects then entered the pool and proceeded to a shoulder or chest depth of water. The pool work began with a warm-up for the arms and torso, proceeding to active running/jogging movements which involved the entire body and was followed by a slower paced cool-down with stretch and strength movements incorporating the sides of the pool (isometrics and stretching). All pool work was performed to selected pre-recorded music in an attempt to dictate a cadence for the participants. Total pool time for each session was 30 minutes, with the entire session lasting 55 minutes. Pool temperatures ranged from (and were controlled at) 28.3°C (83°F) to 30°C (86°F).

The intensity of exercise was recorded as a radial pulse obtained during each exercise session (recorded as number of beats in 15 seconds, multiplied by 4 to give beats per minute). Two heart rate determinations were obtained from each subject per session.

Those subjects assigned to the control group proceeded with their normal daily activities during the course of the study.
EXPERIMENTAL DESIGN AND DATA ANALYSIS

Data analysis was of a repeated measures design; one grouping factor (treatment and control) and one within factor (time). Analysis of variance was performed using the program BMDP2V (BMDP: Statistical Software Inc., California; revised 1982).

The model for this repeated measures design is detailed in Appendix D.

Analysis of variance was performed on each dependent variable to determine group, time and group x time effects. All hypothesis testing was controlled at a predetermined alpha level of 0.01 (\(\alpha = 0.01\)). For each variable, the hypothesis was that the (T1-T2) change in the experimental group would be different from the (T1-T2) change in the control group, with the null hypothesis being \(\mu_{\text{GROUP1}} = \mu_{\text{GROUP2}}\) at \(\alpha = 0.01\). For all variables, when \(P > 0.01\), the null hypothesis was accepted, when \(P < 0.01\), the null hypothesis was rejected.

The dependent variables are:

1. SBPR: systolic blood pressure
2. \(\text{VE}_{\text{MAX}}\): maximum ventilation
3. \(\text{FEV}_{1.0}\): forced expiratory volume (in 1 second)
4. FVC: forced vital capacity
5. HRO: exercise heart rate at 0% treadmill grade
6. HR2: exercise heart rate at 2% treadmill grade
7. HR4: exercise heart rate at 4% treadmill grade
8. HR6: exercise heart rate at 6% treadmill grade
9. HR8: exercise heart rate at 8% treadmill grade
10. HR10: exercise heart rate at 10% treadmill grade
11. HR12: exercise heart rate at 12% treadmill grade
12. $V_{O_{2}}^{MAX}$: maximum oxygen uptake
13. $H_{R_{\text{MAX}}}$: maximum heart rate
14. $H_{R_{\text{REST}}}$: resting heart rate
RESULTS

The physical characteristics and metabolic parameters of the subjects are summarized in Tables I and II. ANOVA tables are reproduced in Appendix E. A summary of variable analysis is given in Table III.

Analysis of variance yielded main (Time) effects for $HR_{\text{REST}}$ ($P = 0.001$) and $FEV^{1.0}$ ($P = 0.002$) demonstrating that, when averaged over groups, a significant change from T(1) to T(2) is seen in these variables. Significant interactions between Group and Time were seen with, $FEV^{1.0}$ ($P = 0.001$), $HR_{\text{REST}}$ ($P = 0.003$) and $V_{\text{O}_2\text{MAX}}$ ($P = 0.007$), demonstrating that the change in these variables from T(1) to T(2) are not the same for both groups (experimental and control). No significant main (Group) effects were noted at T(1). Therefore, the two groups did not show any significant differences at T(1) for physical or metabolic characteristics.
<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean ± SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>AGE (yrs)</td>
<td>68.5 ± 4.68</td>
</tr>
<tr>
<td>WEIGHT (kg)</td>
<td>77.57 ± 18.70</td>
</tr>
<tr>
<td>HEIGHT (cms)</td>
<td>166.65 ± 7.33</td>
</tr>
<tr>
<td>SYSTOLIC BP (REST) (mmHg)</td>
<td>136.00 ± 10.57</td>
</tr>
<tr>
<td>VE\textsubscript{MAX} (1\cdot min\textsuperscript{-1})</td>
<td>35.57 ± 10.32</td>
</tr>
<tr>
<td>FEV\textsubscript{1.0} (l\cdot sec\textsuperscript{-1})</td>
<td>2.20 ± 0.52</td>
</tr>
<tr>
<td>FVC (l\cdot sec\textsuperscript{-1})</td>
<td>3.49 ± 0.96</td>
</tr>
<tr>
<td>HR\textsubscript{MAX} (bts\cdot min\textsuperscript{-1})</td>
<td>143.80 ± 13.87</td>
</tr>
<tr>
<td>HR\textsubscript{REST} (bts\cdot min\textsuperscript{-1})</td>
<td>76.40 ± 5.97</td>
</tr>
<tr>
<td>HR 0% (bts\cdot min\textsuperscript{-1})</td>
<td>119.40 ± 13.88</td>
</tr>
<tr>
<td>HR 2% (bts\cdot min\textsuperscript{-1})</td>
<td>124.13 ± 13.79</td>
</tr>
<tr>
<td>HR 4% (bts\cdot min\textsuperscript{-1})</td>
<td>131.33 ± 13.51</td>
</tr>
<tr>
<td>HR 6% (bts\cdot min\textsuperscript{-1})</td>
<td>137.00 ± 12.74</td>
</tr>
<tr>
<td>HR 8% (bts\cdot min\textsuperscript{-1})</td>
<td>137.77 ± 11.89*  (13)</td>
</tr>
<tr>
<td>HR 10% (bts\cdot min\textsuperscript{-1})</td>
<td>141.14 ± 16.69*  (7)</td>
</tr>
<tr>
<td>HR 12% (bts\cdot min\textsuperscript{-1})</td>
<td>167.00 ± 0.00*  (1)</td>
</tr>
<tr>
<td>VO\textsubscript{2MAX} (ml\cdot kg\textsuperscript{-1}\cdot min\textsuperscript{-1})</td>
<td>24.44 ± 1.99</td>
</tr>
</tbody>
</table>

*all values are means ± SD
*all values n = 15 except where * (number in brackets is n)
## TABLE II

Mean and Standard Deviations of Physical and Metabolic Characteristics

<table>
<thead>
<tr>
<th>Variable</th>
<th>Control</th>
<th>Experimental</th>
</tr>
</thead>
<tbody>
<tr>
<td>AGE (yrs)</td>
<td>67.71 ± 5.38</td>
<td>69.12 ± 4.26</td>
</tr>
<tr>
<td>WEIGHT (kg)</td>
<td>75.41 ± 18.76</td>
<td>79.46 ± 19.72</td>
</tr>
<tr>
<td>HEIGHT (cms)</td>
<td>167.10 ± 7.82</td>
<td>116.25 ± 7.38</td>
</tr>
<tr>
<td>SYSTOLIC BP (REST) (mmHg)</td>
<td>132.43 ± 9.34</td>
<td>139.12 ± 11.17</td>
</tr>
<tr>
<td>VE(_\text{MAX}) (l\cdot\text{min}^{-1})</td>
<td>34.70 ± 11.64</td>
<td>36.32 ± 9.77</td>
</tr>
<tr>
<td>FEV(_1.0) (l\cdot\text{sec}^{-1})</td>
<td>2.31 ± 0.68</td>
<td>2.11 ± 0.37</td>
</tr>
<tr>
<td>FVC (l\cdot\text{sec}^{-1})</td>
<td>3.35 ± 0.82</td>
<td>3.61 ± 1.10</td>
</tr>
<tr>
<td>HR(_\text{MAX}) (bts\cdot\text{min}^{-1})</td>
<td>141.43 ± 17.93</td>
<td>145.87 ± 9.92</td>
</tr>
<tr>
<td>HR(_\text{REST}) (bts\cdot\text{min}^{-1})</td>
<td>77.00 ± 2.77</td>
<td>75.78 ± 8.01</td>
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<tr>
<td>HR 0% (bts\cdot\text{min}^{-1})</td>
<td>116.57 ± 11.12</td>
<td>121.87 ± 16.25</td>
</tr>
<tr>
<td>HR 2% (bts\cdot\text{min}^{-1})</td>
<td>119.42 ± 12.16</td>
<td>128.25 ± 14.57</td>
</tr>
<tr>
<td>HR 4% (bts\cdot\text{min}^{-1})</td>
<td>127.14 ± 12.06</td>
<td>135.00 ± 14.40</td>
</tr>
<tr>
<td>HR 6% (bts\cdot\text{min}^{-1})</td>
<td>134.43 ± 13.26</td>
<td>139.25 ± 12.70</td>
</tr>
<tr>
<td>HR 8% (bts\cdot\text{min}^{-1})</td>
<td>138.14 ± 16.12</td>
<td>137.33 ± 5.24* (6)</td>
</tr>
<tr>
<td>HR 10% (bts\cdot\text{min}^{-1})</td>
<td>142.50 ± 22.55* (4)</td>
<td>139.33 ± 8.02* (3)</td>
</tr>
<tr>
<td>HR 12% (bts\cdot\text{min}^{-1})</td>
<td>167.00 ± 0.00* (1)</td>
<td></td>
</tr>
<tr>
<td>VO(_2\text{MAX}) (ml\cdot kg^{-1}\cdot \text{min}^{-1})</td>
<td>23.84 ± 1.99</td>
<td>24.98 ± 1.97</td>
</tr>
</tbody>
</table>

No. in each group: 7, 8

All values are means ± SD

All values n = 15 except where * (number in brackets is n)
TABLE III
Summary of Variable Analysis

<table>
<thead>
<tr>
<th>Variable</th>
<th>EXPERIMENTAL</th>
<th></th>
<th>CONTROL</th>
<th></th>
<th>TIME</th>
<th>GROUPxTIME</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>T(1)</td>
<td>T(2)</td>
<td>T(1)</td>
<td>T(2)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>AGE (YRS.)</td>
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<td>69.1</td>
<td>67.7</td>
<td>67.7</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>WEIGHT (Kg)</td>
<td>79.4</td>
<td>79.4</td>
<td>75.4</td>
<td>75.4</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>HEIGHT (cm)</td>
<td>116.2</td>
<td>116.2</td>
<td>167.1</td>
<td>167.1</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>SBPR (mmHG)</td>
<td>139.1</td>
<td>131.5</td>
<td>132.4</td>
<td>133.4</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>$V_{E\text{MAX}}$ (l*min$^{-1}$)</td>
<td>36.3</td>
<td>36.9</td>
<td>34.7</td>
<td>35.0</td>
<td>NS</td>
<td>NS</td>
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<td>$FEV_{1.0}$ (l*sec$^{-1}$)</td>
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<td>2.4</td>
<td>2.3</td>
<td>2.2</td>
<td>0.002</td>
<td>0.001</td>
</tr>
<tr>
<td>FVC (l*sec$^{-1}$)</td>
<td>3.6</td>
<td>3.9</td>
<td>3.3</td>
<td>3.3</td>
<td>NS</td>
<td>NS</td>
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<tr>
<td>$HR_{\text{MAX}}$ (bts*min$^{-1}$)</td>
<td>145.8</td>
<td>144.6</td>
<td>141.4</td>
<td>141.5</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>$HR_{\text{REST}}$ (bts*min$^{-1}$)</td>
<td>75.8</td>
<td>71.0</td>
<td>77.0</td>
<td>76.6</td>
<td>0.001</td>
<td>0.003</td>
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<tr>
<td>$HR_{0%}$ (bts*min$^{-1}$)</td>
<td>121.8</td>
<td>116.2</td>
<td>116.5</td>
<td>115.8</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>$HR_{2%}$ (bts*min$^{-1}$)</td>
<td>128.2</td>
<td>123.3</td>
<td>119.9</td>
<td>118.1</td>
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<td>NS</td>
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<tr>
<td>$HR_{4%}$ (bts*min$^{-1}$)</td>
<td>135.0</td>
<td>128.5</td>
<td>127.1</td>
<td>127.0</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>$HR_{6%}$ (bts*min$^{-1}$)</td>
<td>139.2</td>
<td>132.3</td>
<td>134.4</td>
<td>133.8</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>$HR_{8%}$ (bts*min$^{-1}$)</td>
<td>137.3</td>
<td>132.8</td>
<td>138.1</td>
<td>138.1</td>
<td>NS</td>
<td>NS (13)</td>
</tr>
<tr>
<td>$HR_{10%}$ (bts*min$^{-1}$)</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>(7)</td>
</tr>
<tr>
<td>$HR_{12%}$ (bts*min$^{-1}$)</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>(1)</td>
</tr>
<tr>
<td>$VO_{2\text{MAX}}$ (ml*kg$^{-1}$*min$^{-1}$)</td>
<td>24.9</td>
<td>25.8</td>
<td>23.8</td>
<td>23.5</td>
<td>NS</td>
<td>0.007</td>
</tr>
</tbody>
</table>

all values n = 15 except where * (number in brackets is n)
significance at $P < 0.01$
NS denotes non-significance
(-) denotes insufficient data
### TABLE IV
Comparison of Selected Parameters

<table>
<thead>
<tr>
<th>VARIABLE</th>
<th>Thomas et al., 1985</th>
<th>Seals et al., 1984</th>
<th>Larson et al., 1984</th>
<th>Jessop 1988</th>
</tr>
</thead>
<tbody>
<tr>
<td>AGE (YRS.)</td>
<td>62.6 ± 3.1</td>
<td>63.3 ± 3.0</td>
<td>67.0 ± 0.00</td>
<td>68.5 ± 4.68</td>
</tr>
<tr>
<td>WEIGHT (kg)</td>
<td>79.8 ± 1.1</td>
<td>____</td>
<td>77.1 ± 10.7</td>
<td>77.57 ± 18.70</td>
</tr>
<tr>
<td>$V_O^{2\text{MAX}}$ (ml·kg$^{-1}$·min$^{-1}$)</td>
<td>29.6 ± 0.6</td>
<td>25.4 ± 4.6</td>
<td>____</td>
<td>24.44 ± 1.99</td>
</tr>
<tr>
<td>$V_{E\text{MAX}}$ (l·min$^{-1}$)</td>
<td>83.4 ± 2.2</td>
<td>67.2 ± 16.4</td>
<td>____</td>
<td>35.57 ± 10.32</td>
</tr>
<tr>
<td>$H_R^{\text{MAX}}$ (bts·min$^{-1}$)</td>
<td>155.3 ± 2.4</td>
<td>174 ± 10</td>
<td>____</td>
<td>143.80 ± 13.87</td>
</tr>
<tr>
<td>FEV$^{1.0}$ (l·sec$^{-1}$)</td>
<td>2.86 ± 0.05</td>
<td>____</td>
<td>2.80 ± 0.6</td>
<td>2.20 ± 0.52</td>
</tr>
<tr>
<td>n =</td>
<td>100</td>
<td>24</td>
<td>644</td>
<td>15</td>
</tr>
</tbody>
</table>

all values are means ± SD

(-) denotes data not available
<table>
<thead>
<tr>
<th>Parameter</th>
<th>EXPTL (T2)</th>
<th>CTRL (T2)</th>
<th>Supported Status</th>
</tr>
</thead>
<tbody>
<tr>
<td>VO₂MAX (ml·kg⁻¹·min⁻¹)</td>
<td>VO₂MAX EXPTL (T2) &gt; VO₂MAX CTRL (T2)</td>
<td>Supported</td>
<td></td>
</tr>
<tr>
<td>BP SYSTOLIC (RESTING)</td>
<td>BPSR EXPTL (T2) &lt; BPSR CTRL (T2)</td>
<td>Not Supported</td>
<td></td>
</tr>
<tr>
<td>VE MAX (l·min⁻¹)</td>
<td>VE MAX EXPTL (T2) &gt; VE MAX CTRL (T2)</td>
<td>Not Supported</td>
<td></td>
</tr>
<tr>
<td>FEV₁.₀ (l·sec⁻¹)</td>
<td>FEV₁.₀ EXPTL (T2) &gt; FEV₁.₀ CTRL (T2)</td>
<td>Supported</td>
<td></td>
</tr>
<tr>
<td>FVC (l·sec⁻¹)</td>
<td>FVC EXPTL (T2) &gt; FVC CTRL (T2)</td>
<td>Not Supported</td>
<td></td>
</tr>
<tr>
<td>HR REST (bts·min⁻¹)</td>
<td>HR REST EXPTL (T2) &lt; HR REST CTRL (T2)</td>
<td>Supported</td>
<td></td>
</tr>
</tbody>
</table>

T1: baseline measurement  
T2: measurements taken at end of study  
EXPTL: experimental (GROUP 1)  
CTRL: control (GROUP 2)  

Relationships supported on basis of significance $P < 0.01$
DISCUSSION

Tables I and II summarize the variables for the subjects in the study. The subjects were found to represent a homogeneous sample as indicated by the relatively small standard deviations for most of the variables. When some select physical and metabolic parameters are compared with values obtained by previous investigators (Table IV), it can be seen that there are some parameters, particularly body weight, in which a large standard deviation is found. Standard deviations for variables such as weight, age, VO$_{2}\text{max}$, maximum heart rate and FEV$_{1.0}$ are comparable to variability found in other investigations (i.e. Seals et al., 1984; Thomas et al., 1985). Differences are noted in VE$_{\text{max}}$; a lower mean value obtained in this study may be due to the older mean age of the subjects. Generally, in comparison to other similar training studies involving elderly subjects, the subject in this study appears to be both older and exhibits a greater variation in body weight.

I CARDIOVASCULAR PARAMETERS

A. Resting Systolic Blood Pressure

The experimental group did not show a significant decline in resting systolic blood pressure (Table III). This is not in accordance with the findings by Skinner et al., (1964) who found significant reductions in the systolic blood pressure of previously sedentary middle-aged subjects and by Barry et al., (1966) who found similar reductions in elderly subjects. Emes' (1979) 12-week study also demonstrated a reduction in resting systolic blood pressure.

It is known that the body adjusts arterial pressure by altering heart rate, stroke volume and peripheral vascular resistance. The major factor
that seems to be altered in the aging process is the increased rigidity of the arterial walls, resulting in an overall increased systemic resistance to blood flow (Sebban et al., 1981). This is seen by the higher resting systolic and diastolic blood pressures seen in the elderly (Sato et al., 1981). It is unlikely that training could reverse the gradual age-related increase in the elastic constant of the arterial walls and yet reductions in resting systolic pressures are seen. Both systolic and diastolic blood pressures can be lowered significantly by a regular program of exercise (McCardle et al., 1981). This has been seen with normotensive (Kasch and Boyer, 1969) and hypertensive (Boyer and Kasch, 1970; Choquette and Ferguson, 1973) subjects at rest. The Tecumseh Michigan study (Scheuer and Tipton, 1977) showed that regardless of age, active men had significantly lower systolic and diastolic pressures than less active men.

For mean arterial pressure to decrease, both central and peripheral mechanisms that control cardiac output and total peripheral resistance must be considered. Training has been reported to be associated with increased, unchanged or reduced resting cardiac output but the decrease in cardiac output has not been associated with a fall in blood pressure (Scheuer and Tipton, 1977). Total peripheral resistance after training was seen to increase in some studies (Mitchell and Wildenthal, 1974) and decrease in others when compared to control values, although an increase in cardiac output obtained during maximal exercise with trained muscle groups generally has been related to a reduced peripheral resistance and an increased systemic vascular conductance (Clausen, 1977).

Since the literature generally agrees with the finding of a decreased resting systolic blood pressure with training, this current study was inconclusive as it may have been too short in duration to facilitate such a decline in blood pressure (Robinson, 1938).
B. Resting Heart Rate

Resting heart rate was seen to decrease significantly over the 5-week study ($P = 0.003$) in the experimental group whereas the control group did not exhibit a change (Table III). This 6.43% decline in resting heart rate agrees with Cupelli et al. (1984), Emes (1979), Granath et al. (1970) and Zoneraich and Rhee (1977).

It has been suggested that training caused an increased centrogenic vagal cholinergic drive combined with a sympathoinhibitory mechanism, resulting in a decreased resting heart rate (Astrand, 1977). In younger populations, the literature states that training creates an imbalance between the tonic activity of the sympathetic (accelerator) and parasympathetic (depressor) neurons in favor of a greater vagal dominance. It is believed that this is mediated by increased parasympathetic activity and a decreased sympathetic discharge. It has also been suggested that the intrinsic firing rate of the sino-atrial (SA) node is decreased by training. Myocardial cholinesterase concentrations are not affected by training (Scheuer and Tipton, 1977); these findings lend credence to the theory that training increases parasympathetic activity. Although animal studies have indicated increased levels of atrial choline acetyltransferase activity and more myocardial acetylcholine per gram of tissue with chronic exercise (Williams et al., 1984; Wyatt et al., 1978), the literature holds that parasympathetic activity to the myocardium increases and resting sympathetic activity decreases with training.

C. Maximal Heart Rate

Neither experimental or control groups showed any significant change in maximal heart rate over the course of the study (Table III). Training effects have been described as being negligible in some investigations and have been seen to cause a decline in maximal heart rate in others.
Mazzarella et al. (1966) reported no significant change following a 2 month exercise program and Skinner et al. (1964) reported similar findings as a result of a 6 month program with middle-aged men. Granath et al. (1970) and Zoneraich and Rhee (1977) described indications of a decreased maximal heart rate following training. It is possible that the duration of this study was too short to produce significant changes in maximal heart rate (i.e. 5 weeks vs. 6-12 mos. in other investigations) or the age of the subjects was too advanced to produce such a change, regardless of the duration of the study. The literature has not shown any changes in maximum heart rate in older subjects (65 years of age and older), and the majority of previous investigations implicate an age-related decline in maximum heart rate as being unalterable by exercise.

Early papers describing younger subjects state that training caused an increased stroke volume at a given submaximal VO_2 and a proportional decrease in heart rate. It became common knowledge that the decreased heart rate was secondary to the increase in stoke volume although later studies indicated that an increased stroke volume is not essential to a decreased heart rate (Clausen, 1977). The potential importance of peripheral cardiovascular adjustments was suggested.

In contrast to studies involving elderly subjects, training studies describing younger subjects have indicated a decrease in maximal heart rate, regardless of the mode of exercise (Schaible and Scheuer, 1985). Autonomic control, endogenous catecholamines, CNS adaptations or an increased stroke volume have been suggested as causative factors. Training may alter the peripheral or central components of the baroreceptor reflex, thus affecting the peripheral vascular resistance and heart rate. Also, training probably increases the contractility of the myocardium. Increased contractile forces may be met with intracardial or baroreceptive reflexes which cause a decline
in heart rate, but no experimental evidence supports this. There is little evidence to account for the possibility of no change in the maximal heart rate following chronic exercise, but some studies suggest that in subjects who are 60 years of age and older, the changes of cardiac output, stroke volume and left ventricular ejection fraction are considerably less than in younger subjects following acute bouts of exercise (Kuikka and Lansimies, 1982). Port et al., (1980) suggest that the left ventricular response to exercise is changed in the seventh decade of life and by the eigth decade there is practically no increase in ejection fraction. It is possible, then, that chronic exercise may not result in an enhanced stroke volume in the elderly subject as the myocardium cannot respond to the effects of training. This could explain why maximal heart rate does not change in the elderly subject and may re-establish the relationship between stroke volume and heart rate, particularly in the elderly.

D. Heart Rate at Changing Treadmill Elevations

During the treadmill test, heart rate was continuously monitored as the elevation was increased. The experimental and control groups did not show significant decreases in exercise heart rate at treadmill elevations corresponding to 0, 2, 4, 6, 8, 10 and 12 percent grades (Table III). It has been shown with training that heart rate decreases at a given submaximal workload (Barry et al., 1966; Skinner et al., 1963), but the results of this current study appear to be inconclusive as appropriate declines in heart rate were not seen at any of the workloads.

Whether a decline in exercise heart rate occurs by central or peripheral adaptations, the degree of change seems to be associated with an increase in VO$_{2\text{max}}$. Since a significant increase in the VO$_{2\text{max}}$ of the experimental group was seen in this study, one would expect a decline in the exercise heart rate at all workloads as a result of central and/or peripheral
adaptations. It is possible that peripheral adaptations may account for the change in \( VO_{2\text{max}} \); training has been shown to affect the arteriovenous (a-v) oxygen difference. Longitudinal studies with younger subjects show an increase in the (a-v)\( O_2 \) difference at maximal exercise and this difference increases with training (Scheuer and Tipton, 1977). Factors that can cause the increase in (a-v)\( O_2 \) difference include: changes in the function and number of mitochondria, increased oxidative (aerobic) enzymes, changes in fiber type recruitment and increased capillary density. The working muscle can increase its ability to extract oxygen by: (1) a decrease in the affinity of hemoglobin for oxygen at low \( P_{O_2} \) tensions, (2) increased capillary density, (3) elevated myoglobin concentrations, or (4) changes in the number and/or function of mitochondria in active cells (Astrand, 1977). These local muscular adaptations could explain the increased \( VO_{2\text{max}} \) in the absence of unchanged exercise heart rates. It remains to be seen whether a longer study would elicit decreases in the exercise heart rates.

E. Maximum Oxygen Uptake

The subjects in this study exhibited baseline \( VO_{2\text{max}} \) values that were comparable to other investigators (Table IV). \( VO_{2\text{max}} \) showed a strong significant increase in the experimental group (\( P = 0.007 \)) whereas the control group showed no change (Table III). A 3.4% increase was seen in \( VO_{2\text{max}} \) over 5 weeks of training. Mazzarella et al. (1966) demonstrated a 10% increase in \( VO_{2\text{max}} \) after 2 months of endurance training and Robinson (1938) showed a 17% increase in \( VO_{2\text{max}} \) after 6 months. It is possible that an aquatic program of greater duration (i.e. 6-12 months) could produce even greater increases in \( VO_{2\text{max}} \). During the exercise sessions, the mean heart rates obtained (117 ± 3.0 beats per min.) represented 77.5 % of the age-adjusted maximum heart rate. Since heart rate was not continuously monitored during the exercise sessions, it is assumed that the heart rates
did not drop significantly below this level throughout the 30 minutes of active pool activities. When the experimental group's actual mean baseline maximal heart rate is considered (145.8 ± 9.9), the mean exercise heart rates actually represented 80% of the mean maximum heart rate.

There have been few studies to investigate the adaptations in the determination of VO$_{2\text{max}}$ in older individuals (Seals, 1984). Hartley et al. (1969) reported that a 14 percent increase in VO$_{2\text{max}}$ following training in elderly subjects was due primarily to an increase in maximal stroke volume and resulting cardiac output. These same investigators, reporting on a 15 percent increase in VO$_{2\text{max}}$ following training in younger men, stated that an increased maximal (a-v)O$_2$ difference and resulting increased cardiac output were responsible for the enhanced VO$_{2\text{max}}$. Hartley et al. (1969) believed, then, that elderly subjects could not elicit significant increases in their maximal (a-v)O$_2$ difference. Seals et al. (1984) suggested that the increased VO$_{2\text{max}}$ in their elderly subjects was in fact due to adaptations in muscle, resulting in an improved ability to extract oxygen, as reflected in a higher maximal (a-v)O$_2$ difference. Clearly, this is little agreement as to the mechanism(s) responsible for, or contributing to, increases in VO$_{2\text{max}}$ in the elderly subject. Since neither experimental or control group exhibited weight loss over the course of this study, it is possible that the experimental group increased its muscle mass and decreased its body fat, thereby increasing the total lean body mass. This relative increase in muscle mass could account for the increase in VO$_{2\text{max}}$ (Brooks and Fahey, 1984; McArdle et al., 1981).
II  RESPIRATORY PARAMETERS

Both the experimental and control groups showed no change in FVC (Table III). This agrees with the findings of Niinimaa and Shephard (1978) and Pollock et al. (1976) who also could not demonstrate positive change in vital capacity in a short-term aerobic program. Heikkenen (1978), however, did show an increase in the vital capacity of young and middle-aged men and women during an 8 week program. At this point in time, it is not known how decreased levels of activity contribute to the known age-related decline in respiratory function. Presumably, exercise primarily affects the muscles of ventilation, so increases in the strength of these and accessory muscles will augment measures of flow rate. It is doubtful that this form or any form of exercise will do much to significantly alter pulmonary hemodynamics (ventilation: perfusion inequalities) and it is realized that much of the benefit from exercise will be in the way of improved respiratory mechanics.

Saltin and Grimby (1968) demonstrated that the total volume of air inspired and expired during a maximum ventilatory effort is greater in athletes and former athletes than their sedentary counterparts at similar ages. Pollock et al. (1976) showed that the amount of air forcibly expired during a maximum attempt was found to be higher in athletes and former athletes, and that training effects had not been shown. Similarly, Thomas et al. (1985) could not demonstrate changes in FEV$^{1.0}$. The subjects in the experimental group in this present study in contrast to Thomas's study demonstrated a significant increase in FEV$^{1.0}$ ($P = 0.001$). Again, since FEV$^{1.0}$ is dependent upon the elastic components of the lung parenchyma (Whitbourne, 1985), any increase in this measurement must be due to the augmentation of the respiratory musculature. Ventilatory flow has been seen to increase in middle-aged and older adults with training (Whitbourne, 1985). It is possible that the strength components of the program increased the
strength of the involved musculature thereby facilitating an increase in 
FEV\textsuperscript{1,0}, although specific tests to detect changes in muscular strength were 
not incorporated in this present study.

Maximum ventilation has been seen to increase with training (Astrand, 
1973; de Vries, 1970) This may be as a result of an increased breathing 
frequency (Saltin et al., 1969) or an increased tidal volume (de Vries, 1970; 
Robinson et al., 1973). Neither the control or experimental group were seen 
to demonstrate any change in $VE_{\text{max}}$ over the 5 week period. Thomas et al. 
(1985), however showed a 15% increase in $VE_{\text{max}}$ over a 12 month period. One 
reason for this discrepancy may be due to the duration of the study; this 
study being too short in duration to produce any changes in $VE_{\text{max}}$. Another 
possibility is that the low average baseline $VE_{\text{max}}$ (for combined control and 
experimental groups) (Table I) is a reflection of the condition of the 
subjects. It had been noted previously that subjects in this experiment 
differed from comparably aged subjects in other studies with respect to the 
great variation seen in body weight. Additional weight, in the form of fat, 
can hinder chest wall mobility and compliance and may be a contributing 
factor. Since age has not been shown to affect tidal volume under conditions 
of maximal exercise, it is possible that the resulting decreased maximum 
frequency of respiration due to a decreased chest wall compliance may augment 
the known age-related loss in maximum ventilatory rate.

Another potential factor in the low ventilation values obtained in this 
study could be the general decline of the respiratory response to hypoxia 
with age (Petersen et al., 1981). An increased sympathetic activity has been 
shown to exist in the older individual, yet the lungs and chest wall do 
not/cannot respond to this increase in sympathetic stimulation. This factor 
may exert more of an effect on ventilation in older subjects.
In any event, the baseline values for $VE_{\text{max}}$ obtained in this investigation (mean $35.57 \pm 10.32 \text{ l.min}^{-1}$) are lower than the values reported by Thomas et al. (1985) or Seals et al. (1984). This study reported $VE_{\text{max}}$ values that were only 42.6% of those reported by Thomas et al. and 52.9% of those reported by Seals et al. (Table IV). The question that remains at this time is whether the older age of the subjects in this study (mean 68.5 ± 4.7 yrs.) accounts for some of the discrepancy in $V_E$ values seen in Thomas et al. (mean 62.6 ± 3.1 yrs.) or Seals et al. (mean 63.3 ± 3.0 yrs.).

None of the subjects demonstrated a history of chronic obstructive pulmonary disease. The ratio $\text{FEV}^{1.0}/\text{FVC}$ (providing a fraction of the total expired volume delivered in 1 second) can give an indication of pulmonary function (West, 1985). An $\text{FEV}^{1.0}/\text{FVC}$ of 80% is considered to denote normal pulmonary function. Less than 80% is considered as being indicative of obstructive changes and values greater than 80% is considered as representing restrictive (fibrotic) lung disease. The subjects in this study yielded a mean $\text{FEV}^{1.0}/\text{FVC}$ of 63% which, by definition, denotes them as being obstructive (exhibiting increased airway resistance and limited expiration due to alveolar collapse). Indeed, in the elderly, a general decrease in lung elasticity promotes premature airway closure in expiration. The result is a trapping of air within the lung and a slight hyperinflation. Brandstetter and Kazenni (1983), Lynne-Davies (1977) and Reid (1967) all describe changes in alveolar structure with age which cause a decrease in the alveolar surface area. These age-related changes are continuous with an obstructive process.

The results of the hypothesis testing are given in Table V.

It is apparent that the majority of exercise studies concerning exercise and the elderly involve activities other than aquatic, in-pool exercise
programs. It is difficult to meaningfully compare data when the modes of exercise are different. Some meaningful comparisons can be made in general terms as some studies have been done to show some acute effects of aquatic exercise. Vickery et al. (1983) demonstrated that 30 minute sessions of "Aqua Dynamics" produced exercise heart rates of 145 ± 3.0 beats per minute in 21-year old college women. This heart rate represented 77% of the maximum heart rate of these subjects. A similar level of intensity was attained by the considerably older subjects in this study. Johannessen et al. (1986) examined ten women (mean age 54.7 ± 1.0 yrs.) during moderate-intensity exercise programs five days a week for ten weeks. Actual aerobic activity comprised 20 to 35 minutes of each session and different modes of aerobic exercise were used each week throughout the ten weeks (brisk walking/slow jogging, ergometry, swimming and water calisthenics and aerobic dancing). A 20% increase in \( VO_{2\text{max}} \) was found after the ten weeks. Whether the subjects in this study could have attained similar increases in \( VO_{2\text{max}} \) after a similar period of time or with daily exercise is open to contention.

One aspect of aquatic exercise that has not been addressed in this or related studies is the effect of water on the metabolism and cardiovascular adjustment. Craig and Dvorak (1969) reported heart rates averaging 10 beats per minute lower in 23°C water than in warmer water or in air at similar levels of \( VO_2 \). These same investigators also reported observations of bradycardia during water immersion at both exercise and rest. Holmer and Bergh (1974) found that at similar swimming speeds, \( VO_2 \) was significantly higher in colder water compared to warmer water. In addition, the heart rate was significantly lower in colder water at the same \( VO_2 \). McArdle et al. (1976) state that with immersion in cold water a smaller fraction of the cardiac output is shunted to the skin for heat dissipation. Increased peripheral vasoconstriction and hydrostatic pressure on the body surface
causes an increase in both central volume and venous return, therefore resulting in a larger stroke volume. It is possible that a decreased heart rate observed in cold water would be balanced by the increase in stroke volume. Therefore, the cardiac output would be the same at similar levels of VO$_2$ in water of different temperatures. McArdle et al. (1976), using successive increments of discontinuous work with arm-leg ergometry found that during work in 25 and 18°C water, the VO$_2$ averaged 9.0\% (150 ml) and 25.3\% (400 ml) higher, respectively, than values observed in 33°C water. VO$_2$ averaged 250-700 ml higher in cold water compared to air and 33°C water at a mean heart rate. At similar levels of VO$_2$, stroke volume was significantly greater in 25 and 18°C water than in air or 33°C water. It is apparent, then, that a reduction in heart rate during work in cold water is compensated for by a proportionate increase in stroke volume. The investigators concluded that cardiac output is maintained at similar levels of energy expenditure in air, 18, 25 and 30°C water. Pool temperatures recorded during this study ranged from 28.3°C (83°F) to 30°C (86°F). Therefore, recorded exercise heart rates were likely true indications of the intensity of exercise and were not inflated values due to cold water temperatures and there was likely no discrepancies between the heart rates and VO$_2$ during exercise.

Energy expenditure is another area that requires exploration in this type of exercise. Vickery et al. (1983) reported average energy expenditures of 5.9 to 6.5 Kcals per minute during "Aqua Dynamics" sessions. This energy expenditure is greater than the quantity of energy expended in a brisk level walk and is similar to the rate of energy expenditure for slow jogging at a 12 minute mile pace and cycling at approximately 10 mph for college-aged women at this bodyweight. Costill (1971), using leg ergometry in water ranging in temperature from 24.6 to 25.0°C discovered that when comparing the
caloric requirements of submaximal work in the water to those of exercising on land, water exercise was found to increase the energy requirements by 33 to 42 percent for any given work level. In response to the increased energy requirements at each of the exercise levels, the subjects' heart rates were also higher in the water than on land. More study is required in the area of energy expenditure and aquatic exercise.

The treadmill was chosen as a testing device for this study because of the familiarity of the subjects with walking (as opposed to cycling) and because many previous investigations have also utilized treadmill testing for older subjects (Drinkwater et al., 1975; McDonough et al., 1970; Pollock et al., 1974; Sidney and Shephard, 1977). The use of the treadmill data also facilitated the comparison of data. It was subsequently found, however, that some subjects had difficulties with the treadmill, often adopting exaggerated or unnatural gaits during the testing. This was due in some cases to a perceived fear of the treadmill or due to genuine gait problems. Some subjects, due to obesity, had difficulty attaining a "steady-state"; they appeared to be continually adjusting stride length and/or frequency. Some subjects, clearly not used to walking any distance, also had difficulty adjusting to the treadmill. It is difficult to ascertain how much of a bearing these problems had on the test results. Decisions concerning the choice of testing apparatus should consider the conditions often seen in the elderly: obesity, osteoarthritis and rheumatoid arthritis in addition to any other gait problems.
CONCLUSIONS

It was the purpose of this study to determine the efficacy of aquatic exercise for the elderly participant. Water exercise provides its benefits independently of participants' skill levels and it reduces the likelihood of injury from overuse syndromes and heat related problems (Koszuta, 1986). This study has indicated that a supervised and controlled aquatic exercise program is capable of providing a positive source of aerobic conditioning for the elderly as can be evidenced by the outcome of hypothesis testing. This mode of exercise may be prescribed by the primary care physician for the aging individual knowing that the criteria for aerobic benefits can be met. Research, using other modes of exercise, indicates that the minimum intensity required for the development of physical work capacity in unconditioned adults is approximately 50 to 60% of maximum oxygen uptake or approximately 70% of the maximum heart rate. The heart rate responses in this study were well above this minimum value and, like Vickery et al. (1983), suggest that aquatic exercise has sufficient intensity to positively change the physical work capacity of elderly participants who are typically poorly conditioned and/or have low levels of physical work capacity.

Another important concern in the prescription of exercise is compliance. The investigator found initial recruitment of subjects difficult, no doubt due to the perception that many elderly individuals maintain concerning activity (particularly when gait problems are apparent). Compliance for this study was 100%; full turn out for every session. Participants generally found these exercise sessions beneficial and rewarding, reporting that they "felt better" as a result of participation. The "fun factor" should not be discounted; although participants were continually urged to work as hard as...
they could with each session, it is the impression of this investigator that participants did not regard this as exercise as such.

The elderly individual also tends to show marked decreases in social interaction and it is felt that this mode of exercise will do much to improve this situation through the non-threatening nature of the exercise itself and the encouraging compliance. Psychological and social interaction factors are difficult to measure and were beyond the scope of this study, but they should be regarded as being no less important than the cardiorespiratory considerations.

In a general scope, the ongoing debate concerning duration and intensity in aerobic types of exercise must be re-evaluated; this study indicated that supervised aquatic exercise at or above the recommended intensity of exercise performed three times weekly can show significant changes in physical work capacity in the elderly. The elderly participants need not resign themselves to daily exercise for extended periods of time in order for measurable benefits to occur; although continued participation should be encouraged, it is important for the elderly and their physicians to recognize the resilience and adaptive capacity of the elderly individual so they themselves can be the source of encouragement for participation and prescription.
RECOMMENDATIONS

It is the sincere hope that this study will serve to stimulate continued interest in the field of exercise and the elderly. Specifically, there are some areas that merit further investigation:

1. There are other components of this type of exercise that should be evaluated, such as changes in range of motion, muscular strength, body composition and psychological/social factors.

2. Additional studies assessing changes in cardiac output as a result of this type of exercise (i.e. CO₂ rebreathing).

3. Additional investigations concerning aquatic exercise should be undertaken to evaluate the findings in this study and to build a data base upon which meaningful comparisons can be made between studies of this type.

4. Longer term (duration) studies are recommended to determine if a longer exercise protocol will produce better results.

5. Continued efforts are required to determine which specific components of the exercise program provide the most cardiorespiratory benefit.

6. A larger scale study (more subjects) is necessary to determine whether the results of this study have larger, general implications for the elderly population.

7. When dealing with an elderly group of subjects, one must carefully decide on the testing apparatus to be used: a decision that must take gait, bodyweight and motivational factors among others into account.
**APENDIX A**

**Time Effect: Comparison of Means at Baseline (T1) and 5 Weeks Later (T2)**

*(Experimental Group, n = 8)*

<table>
<thead>
<tr>
<th>Variable</th>
<th>T(1)</th>
<th>T(2)</th>
</tr>
</thead>
<tbody>
<tr>
<td>SBPR (mmHg)</td>
<td>139.12 ± 11.17</td>
<td>131.50 ± 7.44</td>
</tr>
<tr>
<td>VE&lt;sub&gt;MAX&lt;/sub&gt; (l·min&lt;sup&gt;-1&lt;/sup&gt;)</td>
<td>36.32 ± 9.77</td>
<td>36.91 ± 1.54</td>
</tr>
<tr>
<td>FEV&lt;sub&gt;1.0&lt;/sub&gt; (l·sec&lt;sup&gt;-1&lt;/sup&gt;)</td>
<td>2.11 ± 0.37</td>
<td>2.42 ± 0.18</td>
</tr>
<tr>
<td>FVC (l·sec&lt;sup&gt;-1&lt;/sup&gt;)</td>
<td>3.61 ± 1.10</td>
<td>3.94 ± 0.37</td>
</tr>
<tr>
<td>HR&lt;sub&gt;MAX&lt;/sub&gt; (bts·min&lt;sup&gt;-1&lt;/sup&gt;)</td>
<td>145.87 ± 9.92</td>
<td>144.62 ± 2.19</td>
</tr>
<tr>
<td>HR&lt;sub&gt;REST&lt;/sub&gt; (bts·min&lt;sup&gt;-1&lt;/sup&gt;)</td>
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<td>71.00 ± 2.75</td>
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<td>HR 0% (bts·min&lt;sup&gt;-1&lt;/sup&gt;)</td>
<td>121.87 ± 16.25</td>
<td>116.25 ± 16.19</td>
</tr>
<tr>
<td>HR 2% (bts·min&lt;sup&gt;-1&lt;/sup&gt;)</td>
<td>128.25 ± 14.57</td>
<td>123.37 ± 10.83</td>
</tr>
<tr>
<td>HR 4% (bts·min&lt;sup&gt;-1&lt;/sup&gt;)</td>
<td>135.00 ± 14.40</td>
<td>128.50 ± 8.73</td>
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<tr>
<td>HR 6% (bts·min&lt;sup&gt;-1&lt;/sup&gt;)</td>
<td>139.25 ± 12.70</td>
<td>132.37 ± 7.77</td>
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<tr>
<td>HR 8% (bts·min&lt;sup&gt;-1&lt;/sup&gt;)</td>
<td>137.33 ± 5.24</td>
<td>132.83 ± 2.74</td>
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<tr>
<td>VO&lt;sub&gt;2MAX&lt;/sub&gt; (ml·kg&lt;sup&gt;-1&lt;/sup&gt;·min&lt;sup&gt;-1&lt;/sup&gt;)</td>
<td>24.98 ± 1.97</td>
<td>25.83 ± 0.213</td>
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</tbody>
</table>

All values are means ± SD
APPENDIX B

Time Effect: Comparison of Means at Baseline (T1) and 5 Weeks Later (T2)
(Control Group, n = 7)

<table>
<thead>
<tr>
<th>Variable</th>
<th>T(1)</th>
<th>T(2)</th>
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<tbody>
<tr>
<td>SBPR (mmHg)</td>
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<td>133.43 ± 2.31</td>
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<tr>
<td>V_{E_{MAX}} (l·min^{-1})</td>
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<td>FEV_{1.0} (l·sec^{-1})</td>
<td>2.31 ± 0.68</td>
<td>2.29 ± 0.09</td>
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<td>FVC (l·sec^{-1})</td>
<td>3.35 ± 0.82</td>
<td>3.35 ± 0.09</td>
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<td>HR_{MAX} (bts·min^{-1})</td>
<td>141.43 ± 17.93</td>
<td>141.57 ± 1.57</td>
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<tr>
<td>HR_{REST} (bts·min^{-1})</td>
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<td>115.86 ± 4.23</td>
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<td>HR 2% (bts·min^{-1})</td>
<td>119.42 ± 12.16</td>
<td>118.14 ± 11.18</td>
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<td>HR 4% (bts·min^{-1})</td>
<td>127.14 ± 12.06</td>
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<td>HR 6% (bts·min^{-1})</td>
<td>134.43 ± 13.26</td>
<td>133.86 ± 2.51</td>
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<tr>
<td>HR 8% (bts·min^{-1})</td>
<td>138.14 ± 16.12</td>
<td>138.14 ± 3.32</td>
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<tr>
<td>VO_{2_{MAX}} (ml·kg^{-1}·min^{-1})</td>
<td>23.84 ± 1.99</td>
<td>23.52 ± 0.82</td>
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</table>

all values are means ± SD
### APPENDIX C

#### Means of Experimental and Control Groups at Baseline (T1) and 5 Weeks Later (T2)

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<tr>
<td>SBPR (mmHg)</td>
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<td>FEV₁₀</td>
<td>2.11 ± 0.37</td>
<td>2.31 ± 0.68</td>
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<tr>
<td></td>
<td>(l·sec⁻¹)</td>
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<tr>
<td>FVC</td>
<td>3.61 ± 1.10</td>
<td>3.35 ± 0.82</td>
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<td></td>
<td>(l·sec⁻¹)</td>
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<tr>
<td>HR 6%</td>
<td>139.25 ± 12.70</td>
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<td></td>
<td>(bts·min⁻¹)</td>
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<tr>
<td>HR 8%</td>
<td>137.33 ± 5.24</td>
<td>138.14 ± 16.12</td>
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<tr>
<td></td>
<td>(bts·min⁻¹)</td>
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<tr>
<td>VO₂MAX</td>
<td>24.98 ± 1.97</td>
<td>23.84 ± 1.99</td>
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<tr>
<td></td>
<td>(ml·kg⁻¹·min⁻¹)</td>
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<tr>
<td>HR REST</td>
<td>75.87 ± 8.01</td>
<td>77.00 ± 2.77</td>
</tr>
<tr>
<td></td>
<td>(bts·min⁻¹)</td>
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</tbody>
</table>

*All values are means ± SD*

*Underlined P values are significant (P < 0.05)*
Where:

\[ Y_{ijk} = \mu + \alpha_1 + \pi_k(i) + \beta_j + (\alpha \beta)_{ij} + (\beta \pi)_{jk(i)} \]

- \( \alpha_1 \) represents the grouping factor (i.e.: treatment and control)
- \( \pi_k(i) \) represents subjects within the grouping factor (used to obtain the error term for the grouping factor)
- \( \beta_j \) represents the within factor (i.e. the time effect)
- \( (\alpha \beta)_{ij} \) represents the interaction between the grouping factor and the within factor
- \( (\beta \pi)_{jk(i)} \) represents the interaction of the within factor and the subjects within the grouping factor (used to obtain the error term for the within factor and for the interaction of the grouping factor and within factor).

Program is BMDP2V (BMDP: Statistical Software Inc., California; USA, Revised 1982).
## Resting Systolic Blood Pressure

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<tr>
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<th>Ms</th>
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<th>Prob</th>
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### Maximum Ventilation

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<td>Time (Pre/Post)</td>
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Forced Expiratory Volume (in 1 second)

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underlined P values are significant (P < 0.01)
### Forced Vital Capacity

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Underlined P values are significant (P < 0.01)
(Exercise) Heart Rate at 0% Grade

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**Exercise** Heart Rate at 6% Grade

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(Exercise) Heart Rate at 8% Grade

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all values n = 13
(Exercise) Heart Rate at 10% Grade

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all values n = 8
**Maximum Oxygen Uptake**

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Underlined P value is significant (P < 0.05)
APPENDIX F

Contraindications for Exercise and Exercise Testing (Out-of-Hospital Setting)

A. Contraindications
1. Acute myocardial infarction
2. Unstable or at-rest angina pectoris
3. Dangerous arrhythmias (ventricular tachycardia or any rhythm significantly compromising cardiac function)
4. History suggesting excessive medication effects (digitalis, diuretics, psychotropic agents)
5. Manifest circulatory insufficiency (congestive heart failure)
6. Severe aortic stenosis
7. Severe left ventricular outflow tract obstructive disease (IHSS)
8. Suspected or known dissecting aneurysm
9. Active or suspected myocarditis or cardiomyopathy (within the past year)
10. Thrombophlebitis—known or suspected
11. Recent embolism, systemic or pulmonary
12. Recent or active infectious episodes (including upper respiratory infections)
13. High dose of phenothiazine agents

B. Relative Contraindications
1. Uncontrolled or high-rate supraventricular arrhythmias
2. Repetitive or frequent ventricular ectopic activity
3. Untreated severe systemic or pulmonary hypertension
4. Ventricular aneurysm
5. Moderate aortic stenosis
6. Severe myocardial obstructive syndromes (subvalvular, muscular, or membranous obstructions)
7. Marked cardiac enlargement
8. Uncontrolled metabolic disease (diabetes, thyrotoxicosis, myxedema)
9. Toxemia

C. Condition Requiring Consideration and/or Precautions
1. Conduction disturbances
   a. Complete atrioventricular block
   b. Left bundle branch block
   c. Wolff-Parkinson-White anomaly or syndrome
   d. Lown-Ganong-Levine syndrome
   e. Bifascicular block (with or without 1st block)
2. Controlled arrhythmias
3. Fixed-rate pacemaker
4. Mitral valve prolapse (click-murmur) syndrome

In the practice of medicine, the benefits of evaluation often exceed the risks for patients with these relative contraindications.
5. Angina pectoris and other manifestations of coronary insufficiency
6. Certain medications
   a. Digitalis, diuretics, psychotropic drugs
   b. Beta-blocking and drugs of related action
7. Electrolyte disturbance
8. Clinically severe hypertension (diastolic above 110, grade III retinopathy)
9. Cyanotic heart disease
10. Intermittent or fixed right-to-left shunt
11. Severe anemia (hemoglobin below 10 gm/d.)
12. Marked obesity (20% above optimal body weight)
13. Renal, hepatic, and other metabolic insufficiency
14. Overt psychoneurotic disturbances requiring therapy
15. Neuromuscular, musculoskeletal, orthopedic, or arthritic disorders which would prevent activity
16. Moderate to severe pulmonary disease
17. Intermittent claudication
18. Diabetes

After American College of Sports Medicine, Guidelines for Graded Testing and Exercise
Non-Weight Bearing Water Exercise: Changes in Cardiopulmonary Function in Elderly Men and Women

J.E. Taunton, M.D., E.C. Rhodes, Ph.D., R.J. Ham, M.D., P.R. Grantham, M.D., D.J. Jessop, B.Sc.

The purpose of this investigation is to examine the cardiovascular and pulmonary responses to non-weight bearing in-pool exercise (training) in the elderly. The projected aims of the study are to determine:

1) the efficacy of a representative, established program of exercise for the elderly,

2) the adaptations of the elderly cardiovascular system following 1 month of exercise,

3) the adaptations in pulmonary function in the elderly following 1 month of exercise,

4) whether such a program of exercise could be clinically applicable and prescriptive.

You will perform a graded exercise test on a bicycle ergometer and/or a motor-driven treadmill. The purpose of this test is to examine the response of your heart and lungs to exercise. The test consists of running, or riding the ergometer at one or more levels of difficulty. Your electrocardiogram will be monitored throughout the exercise and recovery periods. It is expected that you will complete this exercise test with no complications. Because of the very uncommon, unpredictable response of some individuals to exercise, unforeseen difficulties may arise which would necessitate treatment. Complications have been few during exercise tests and these usually clear quickly with little or no treatment. You are asked to report any unusual symptoms during the test. We may stop the test at any time because of signs of fatigue or you may stop when you wish to because of personal feelings of fatigue or discomfort. Every effort will be made to conduct the test in such a way as to minimize discomfort and risk. However, there exists the possibility of potential risks such as; abnormal blood pressure, fainting, disorders of heart beat, and very rare instances of heart attack.

You will also perform a test of lung capacity.

The exercise program will be performed three (3) times per week for a maximum of 1 month. You are reminded to take all necessary precautions when in the pool, particularly when entering and leaving the pool and surrounding area.

In signing this consent form you state that you have read and understand the description of the tests, the experiment and their complications. You enter
the battery of tests and the experiment willingly and may withdraw at any time.

Additionally, your identity and test results will be kept in confidence and will become the property of the above investigators.

Consent

I have read the above comments and understand the explanation, and I wish to proceed with the test. I hereby acknowledge the receipt of a copy of this consent form.

__________________________
subject (signature)

__________________________
witness (signature)

__________________________
date
REFERENCES


