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Cardiovascular Responses in High, Moderate and Low Aerobic Capacity Males During Static and Dynamic Work.

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**CARDIOVASCULAR RESPONSES IN HIGH, MODERATE AND LOW
AEROBIC CAPACITY MALES DURING STATIC AND DYNAMIC WORK**

The Louisiana State University and Agricultural and Mechanical Col. **Ph.D. 1982**

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**CARDIOVASCULAR RESPONSES IN HIGH, MODERATE AND LOW AEROBIC
CAPACITY MALES DURING STATIC AND DYNAMIC WORK**

A Dissertation

**Submitted to the Graduate Faculty of the
Louisiana State University and
Agricultural and Mechanical College
in partial fulfillment of the
requirements for the degree of
Doctor of Philosophy**

in

**The School of Health, Physical Education,
Recreation and Dance**

**by
John Francis Harper
B.A., University of South Carolina, 1973
M.A.T., University of South Carolina, 1975
August 1982**

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ABSTRACT

Eighteen male subjects participated in an investigation to examine the effects of static, dynamic, and combination work (static and dynamic) on the variables heart rate, systolic and diastolic blood pressure, pressure rate product, and maximal oxygen consumption ($\dot{V}O_2$ max). The subjects, who were all free from any cardiovascular problems, were divided into 3 groups based on their maximum oxygen consumption levels. The high (Group 1), moderate (Group 2), and low (Group 3) aerobic capacity groups had mean $\dot{V}O_2$ max levels of 64, 55, and 47 ml.kg⁻¹.min⁻¹, respectively. Each subject performed a static test (holding a weighted brief case), a dynamic test (walking on a treadmill) and a combination test (holding a weighted brief case while walking on a treadmill). Workloads for all subjects were relative to their $\dot{V}O_2$ max level and to their maximum voluntary contraction. In addition to the static test, dynamic test, and the combination test, which were counter-balanced to prevent any ordering effect, the subjects also performed two maximal treadmill tests in order to observe whether static contractions during maximal work would increase $\dot{V}O_2$ max. The first was a standard dynamic treadmill test while the second followed the same protocol with the addition of a static component during the last 2 minutes.

Statistical analysis (MANOVA, ANOVA, Duncan's Multiple Range Test) revealed that aerobic fitness level did not affect the heart rate, blood pressure, or pressure-rate product response to the three types of work examined. Furthermore, $\dot{V}O_2$ max was not increased by the addition of static work during a maximal test. Heart rate and pressure rate product were significantly higher ($p < .05$) during combination

work than during dynamic work, and during dynamic work they were significantly higher than during static work. Diastolic blood pressures were significantly different across all three modes of work. Ordered from high to low, they were static work, combination work, and dynamic work. Systolic blood pressure was significantly higher for combination work, but measures taken during static and dynamic work were not significantly different.

CHAPTER 1

INTRODUCTION

Cardiovascular Responses to Static Work

Although the cardiovascular changes that occur while performing dynamic or static exercise have been studied extensively, relatively little attention has been concentrated on the hemodynamic effects of combined dynamic and static work. There has been consistency in the literature regarding the cardiovascular responses during static and dynamic work. However, there has been disagreement about the cardiovascular changes occurring during combination work (dynamic plus static).

Lindhard (1920) was the first to report on the cardiovascular and respiratory responses to static work. He noted that static contractions occluded the blood flow to the muscles. Since then, many researchers have investigated the effects of static contractions on blood pressure (BP), heart rate (HR), cardiac output, blood flow, peripheral vascular resistance, and ventilation.

Systolic and diastolic BP have been noted to increase during hand-grip contractions (Humphrey & Lind, 1963; Lind & McNicol, 1965; Lind, Taylor, Humphreys, Kennelly, & Donald, 1964), and the degree of BP elevation is influenced by the percentage of the maximum voluntary contraction (MVC) and not by the absolute force involved (Lind & McNicol, 1967a). In addition, when two different muscle groups contract simultaneously at identical percentages of their MVC, the BP response is the same as if only one of the muscle groups was contracting.

If two different muscles contract at different percentages of their respective MVC, then the pressure response is determined by the muscle contracting at the higher relative percentage of its MVC (Lind & McNicol, 1967a, 1968).

Heart rate increases can be seen in less than 1 second after the initiation of an isometric contraction (Freyschuss, 1970), but quickly reach a steady state level if the contraction is sustained below 15% of MVC (Lind & McNicol, 1967c). At sustained tensions above 15% of MVC HR, BP, and cardiac output all continue to rise in an approximately linear fashion (Humphreys & Lind, 1963). However, it is uncommon to find HR values above $120 \text{ beats} \cdot \text{min}^{-1}$ at the point when subjects can no longer maintain a specified percentage of their MVC (Lind, 1970).

Cardiovascular Responses to Dynamic Work

The cardiovascular responses to dynamic work show many differences from those described for static effort. The key to the differences lies in the fact that generally during dynamic work larger muscle masses are involved and the blood flow to the muscle is not reduced to the extent that it is during static work. If large muscle masses are involved, as in running, HR increases to meet the demands for both oxygen and heat transport. Vasodilation in the contracting muscles, followed by cutaneous vasodilation, results in a decrease in total peripheral resistance (Åstrand & Rodahl, 1977; MacDonald, Sapru, Taylor, & Donald, 1966). The fall of peripheral vascular resistance allows the active muscle to be perfused with blood with only a small rise in BP during mild rhythmical contractions (Lind & McNicol, 1967c).

Cardiovascular Responses to Combined Static and Dynamic Work

Although there is some disagreement regarding the cardiovascular effects from combination work, the majority of evidence suggests that combined work has a higher physiological cost than the sum of equivalent levels of dynamic and static work done separately (Jackson, Reeves, Sheffield, & Burdeshaw, 1973; Lind & McNicol, 1965; Sanchez, Monod, & Chabaud, 1979). This augmentation could place persons who have coronary heart disease in danger.

Angina normally follows a rise in the work of the myocardium to a level which is essentially fixed in each patient. The level at which angina begins is consistently related to the product of HR and systolic BP (Robinson, 1967). Any static exertion, which often occurs suddenly in everyday life, can place a person close to dangerous circulatory overload with little warning. "Airport Angina", which is a severe and unexpected angina that some persons experience while walking at a normal rate through an airport while carrying a briefcase or suitcase, represents this situation very clearly (Jackson et al., 1973).

Responses to Aerobic Training

Training by aerobic activities is known to decrease arterial BP and HR and to increase myocardial efficiency, efficiency of peripheral blood distribution and return, and red blood cell mass, all of which can increase maximal levels of oxygen consumption and therefore increase aerobic performance (Clarke, 1972). A question which needs to be addressed is whether or not aerobic training can reduce the cardiovascular stresses imposed upon the body during combination work (static and dynamic combined). One method of answering this is by comparing

low, moderate, and highly aerobically trained groups under three different conditions: (1) static work; (2) dynamic work; and (3) a combination of static and dynamic work. A highly aerobically trained group would be expected to respond with lower arterial BP, HR, and pressure-rate product (PRP) than the lower aerobic capacity groups. If such a consequence of this prediction should be substantiated, there would be additional support for aerobic training of both normal persons and those with heart disease.

Review of Literature

Cardiovascular Responses to Static Work

Static (isometric) contractions do not constitute work according to physical laws, since no external movement occurs. However, the muscle fibers do change in length, which requires the use of energy and can be very fatiguing if the blood flow to the muscle is not adequate to renew the substrate utilized in the muscle contraction. It is common to describe static work in terms of the product of developed tension multiplied by the time of contraction (Åstrand & Rodahl, 1977). Although purely static work is an unusual occurrence in daily activities, cardiovascular responses have been extensively documented in the literature.

Lindhard (1920) discovered that during the recovery period following a strong contraction in which the subject had hung by the hands from a beam with flexed arms (pull-up position), a greater increase in oxygen uptake and cardiac output occurred than that observed during the actual contraction. He concluded from this recovery data that during the period of contraction the muscles were occluded by mechanical

compression. Since then, many groups of researchers have investigated this phenomenon. Asmussen and Hansen (1938) noted an immediate increase in oxygen uptake, stroke volume (SV), HR, and BP when they had subjects push with both legs against a dynamometer. During recovery, oxygen uptake and cardiac output increased slightly and then dropped toward resting values, while BP and HR dropped immediately.

Heart Rate

By far the most extensively reported cardiovascular responses to static work have been on HR and BP. Alam and Smirk (1938) inflated a sphygmomanometer cuff placed around one ankle to a pressure of 200 mm Hg, which arrested the blood flow through the foot. The foot was then exercised by alternate dorsi and plantar flexion of the toes. In a series of over 30 experiments it was observed that, when exercise was performed by the calf muscles while the blood flow was occluded, the HR increased and remained elevated an average of $11 \text{ beats} \cdot \text{min}^{-1}$ after cessation of exercise if occlusion was maintained. Upon reestablishing circulation the HR increased slightly before returning to normal values in 1 to 2 min. This temporary acceleration of the pulse accompanied by hyperpnea was believed to be caused by the action of the metabolites which were released from the legs into the general circulation. Alam and Smirk (1938) also performed a similar experiment without occlusion of the blood flow. This experiment indicated that the maintenance of a high pulse rate following exercise is dependent upon the arrest of the circulation. When the blood flow to the legs was occluded, but no exercise was performed, little change in pulse rate was observed for the first 10 min of occlusion. These results indicated that metabolites

formed during muscular exercise and retained in the muscles by the arrest of the circulation caused the pulse-accelerating reflex.

Knox (1951) attempted to separate the effects of an exercise containing both static and dynamic components on HR. Subjects initially pulled on springs with both arms and held this stretch for 20 sec. Subsequently, the subjects moved their arms in the same way, but with no tension in the springs. Heart rate in the dynamic experiment subtracted from the HRs in the dynamic plus static experiment indicated that the static effort caused a HR increase of about $10 \text{ beats} \cdot \text{min}^{-1}$ after 20 sec. Knox concluded that both static and dynamic exercise affect HR and that the effects are additive. Tuttle and Horvath (1957) substantiated the effects of static work on HR by having their subjects squeeze a hand dynamometer at maximum effort for one minute. The HR increased from 66 to 93 $\text{beats} \cdot \text{min}^{-1}$ within 15 sec and showed only a moderate increase to 97 $\text{beats} \cdot \text{min}^{-1}$ at the end of 45 sec of work.

In an attempt to determine the cause of increased HR during static exercise, MacDonald et al. (1966) compared HR responses to sustained handgrip contractions at 30% MVC under two conditions: (1) normal contraction and (2) contraction after 10 mg of propranolol had been injected intravenously. The HR response during the handgrip contraction following administration of propranolol was found to be significantly increased over that during the contraction without propranolol when expressed as a percentage of the resting value. It was felt that a part of the HR response to the contraction was adrenergic in nature, although the rapid increase in HR at the initiation of a contraction suggested that primarily neural rather than humoral mechanisms were involved.

Freyschuss (1970) also used 10 mg of propranolol in a similar experiment but found essentially no difference in the magnitude of the HR increase when compared to contractions without propranolol present. The deviation between the findings of MacDonald et al. (1966) and Freyschuss (1970) might be explained by the fact that the HR was measured at different time intervals from the start of the contraction, 7 sec and 60 sec, respectively. Freyschuss (1970) and MacDonald et al. (1966) agreed that propranolol did modify, but not abolish, the HR response to static work by blocking the beta-adrenergic receptors, and that at least part of the HR response during exercise was possibly a result of vagal inhibition. Petro, Hollander, and Bouman (1970) concurred and further suggested that the muscle spindles might be involved in the reflex decrease of vagal dominance.

Lind and McNicol (1967a, 1968) reported that, when two or more muscle groups contracted at the same relative tension, the HR response was the same whether they contracted together or separately. This finding was substantiated in 1970 by Freyschuss. Furthermore, if two or more muscle groups contract simultaneously at different relative tensions, the HR response was determined by the muscle group contracting at the higher relative tension.

Additional studies of the HR response to isometric contractions have been reported by Bruce, Lind, Franklin, Muir, MacDonald, McNicol, and Donald (1968) who examined the effects of fatigue on HR during exhaustive exercise. A modest increase in HR of $23 \text{ beats} \cdot \text{min}^{-1}$ was observed at the instant the subject could no longer hold a contraction at 30% MVC. The HR fell abruptly after the contraction ended. These

results were in agreement with earlier work by Lind and McNicol (1967c) and point to the importance of neural controlling factors.

Blood Pressure

The literature is replete with the effects of isometric contractions on BP. As early as 1877, Gaskell described the relationship between muscular activity and blood flow to active muscles. Gaskell described two opposing features: (1) a local vasodilation of the local vessels which he deduced to be caused by a substance released from the muscles, and (2) a throttling effect (mechanical constriction) of the blood vessels by the compression of the contracting muscle fibers. Lindhard (1920) also reported that occlusion of blood flow in the muscles was caused by mechanical compression of the blood vessels during muscular contractions.

However, it was not until 1937 that the mechanism responsible for the increased BP during isometric contractions was exclusively investigated. Alam and Smirk (1937) observed a BP raising reflex arising from muscular exercise of a limb when the circulation through its working muscles was arrested by mechanical pressure. They concluded that the rise in BP was caused by the passage of nerve impulses out from the exercised muscle and that these nerve impulses were caused by the accumulation of metabolites in the muscle. When these metabolites were maintained in the muscle by local arrest of the circulation, the BP reflex was maintained. Blood pressure was found to increase before many subjects reported experiencing pain, and BP fell at once when circulation through the limb was re-established, indicating that adrenaline was not the BP raising stimulus (Alam & Smirk, 1937; Humphrey & Lind, 1963).

Thompson (1954) reported that isometric work caused a significant rise in diastolic pressure and suggested that the cause was reflexive in nature. Similar results were reported by Tuttle and Horvath (1957) who had subjects squeeze a hand dynamometer at maximum effort for 1 min. Systolic and diastolic BP rose from a resting value of 120/82 to 161/109 mm Hg at the end of work. Research by Whipp and Davis (1970) substantiated this rise in diastolic pressure.

Lind et al. (1964) measured cardiac output, forearm blood flow, aortic BP, and HR in subjects performing handgrip contractions at 10, 30, and 50% MVC while in the supine position. The sustained handgrip contractions caused an abrupt rise in BP which returned to normal within 1 min after contraction had ended. This BP rise was shown to be due to a rise in cardiac output which, in turn, was dependent on an increase in HR. The systemic vascular resistance showed little or no change, and the SV did not increase at any time during the contractions and in fact decreased in a number of instances at the higher levels of exertion. Compared to the response to dynamic exercise, there was a disproportionate rise in cardiac output in relation to the increase of oxygen uptake. The fact that the muscle receiving an inadequate blood supply can invoke powerful central circulatory reflexes designed to increase its perfusion pressure and circulation, and the fact that this reflex occurs in the presence of complete circulatory occlusion, indicates an afferent neural pathway.

These results were in agreement with research by Staunton, Taylor and Donald (1964) who postulated that active contraction of muscle cells under naturally or artificially occurring ischemic conditions caused stimulation of local receptors responsible for initiating and

maintaining a pressor reflex. This pressor reflex operates such that perfusion pressure progressively increases as blood flow becomes inadequate for metabolic requirements or for the removal of metabolites. This increase in perfusion pressure is potentiated by external vascular occlusion to the working muscle.

Blood Flow

Although vasodilation occurs in a muscle during a contraction, blood flow is often hindered by the mechanical compression of the vessels by the contracting muscles. Barcroft and Millen (1939) reported that blood flow through the calf muscles during sustained contractions was cut off at a tension of less than 20% of MVC. Later evidence strongly suggested that the blood flow was not cut off by mechanical compression in the legs at a tension of 20% MVC, but by "nipping" of the artery between shortening of the soleus and gastrocnemius muscles (Humphreys & Lind, 1963).

Humphreys and Lind (1963) conducted experiments to estimate the tension at which intramuscular pressure overcame the perfusion pressure of blood through the contracting muscle, and to assess the functional significance of the blood flow through the muscle during the contractions. Using a Whitney (1953) strain-gauge plethysmograph, Humphreys and Lind were able to show that blood flow through the forearm increased during handgrip contractions sustained at tensions up to at least 70% MVC. At tensions above 70% MVC intramuscular pressure occluded the blood supply, and the rate of fatigue (the point at which a specified percentage of MVC could no longer be held) was inversely related to the increase in muscle tension.

Humphreys and Lind (1963) determined that the increased blood flow went only to active muscles by measuring the temperature in both active and inactive muscles during contractions. Metabolism was eliminated as the main cause of the increase in muscle temperature by applying arterial occlusion to the contracting arm, resulting in only a small temperature increase. They concluded that during static work the increased blood flow goes directly to the active muscle and that there was no increase in blood flow to the inactive muscle, although there was an increase in perfusion pressure. Because there was an increase in BP with no increase in blood flow to the inactive muscles, this indicated that there was vasoconstriction in the inactive muscles.

Fatigue

Fatigue, or the inability to maintain a contraction at a specified percentage of MVC, was investigated by Lind and McNicol (1967b) who had subjects perform sustained handgrip contractions from 5 to 20% MVC. Tensions maintained above 15% MVC were found to eventually cause a decrement in performance, while contractions held at tensions below 15% MVC did not. Lind et al. (1964) had earlier reported that muscle contractions at a tension of 10% MVC appeared to be indefatigable (one subject maintained this tension for more than 1 hour without fatigue). This led to the conclusion that at tensions below 15% MVC the blood supply to the muscles was adequate for the metabolic needs. Also, in the normal physiological state, the blood perfusion of the muscles during and after contractions is related to the metabolic requirements of the muscle. Any post-exercise hyperemia following sustained contractions bears a close relationship to the metabolism of the active muscle.

Endurance

Muscle endurance is affected by blood flow (Barcroft & Millen, 1939; Lind & McNicol, 1967b; Start & Holmes, 1963), motivation (Johnson & Nelson, 1967; Simonson, 1971), and temperature (Clarke, Hellon, & Lind, 1958; Lind, 1959). Bruce et al. (1968) conducted a series of experiments to establish the reproducibility of responses to handgrip contractions over a 10-day period. Analysis of the 10-day series showed that the most reproducible measurement, having a coefficient of variation of 4.5%, was the MVC performed at the start of each experiment. The average duration of any of 3 successive contractions sustained at a tension of 30% MVC had a coefficient of variation of about 20% for the initial 2 days and fell to 7% for the last 2 days of testing. Both HR and BP values remained the same at the point of fatigue during all contractions.

Kroll (1968) reported that low-level strength groups fatigued more slowly than high- and middle-level strength groups. A 20-lb difference in maximum strength existed between each of the three groups. These results suggested that the impediment of blood flow, which is the main contributor to fatigue in static work, might depend upon the absolute rather than the relative muscle tension. It appears from similar results reported by Mundale (1970) that relative isometric grip endurance is inversely related to the absolute isometric grip strength and the contraction time. Mundale concluded that "weaker subjects can maintain a higher proportion of their maximal strength during prolonged activity than those with superior strength."

Recovery of the ability to exert maximum tension after fatigue in isometric contractions performed at one-third of MVC was 90% complete

in 7 min and 100% complete after 20 min (Lind, 1959). However, recovery to maintain maximum duration for a sustained contraction at one-third maximum tension was only 70 to 75% complete after 40 min, and full recovery took several hours (Lind & McNicol, 1967a).

Stephens and Taylor (1972) reported that fatigue of maintained MVC occurs in two phases. In the first, lasting for around 1 min, force falls to about 50% MVC, and in the second phase force falls at a relatively slower rate than in phase one. They interpreted their results as indicating that fatigue arises initially from neuromuscular junction fatigue, but later contractile element fatigue becomes progressively more important. Because the blood supply is cut off during the first stage, arterial occlusion would make no difference in the progress of fatigue. However, during the second stage as force falls, blood flow returns to the muscle; and fatigue progresses little further unless occlusion is maintained by external mechanical compression.

Recovery from static work is much faster than from dynamic work. Little, if any, oxygen debt is incurred (Lind et al., 1964; Tuttle & Horvath, 1957); and BP and HR usually have returned to resting levels within 1 minute after cessation of exercise. Simonson (1971) discusses many other causes of fatigue during static work in addition to those mentioned above.

Papers published in 1967 (Donald, Lind, McNicol, Humphreys, Taylor & Staunton; Lind & McNicol c) and in 1968 (Lind & McNicol) present excellent reviews of the cardiovascular responses to static contractions. The degree of BP elevation was reported to be influenced by (1) the percentage of the MVC of the muscle isometrically contracting and not of the absolute mass involved; (2) if two different muscle

groups contracted simultaneously at identical percentages of their MVC, the BP response was the same as if only one of the muscle groups was contracting; and (3) if two different muscles contract at different percentages of their respective MVC, then the pressure response is determined by the muscle contracting at the higher relative percentage of its MVC.

Percentage of MVC as a determinant has recently been questioned by Buck, Amundsen, and Nielsen (1980); Freund, Hobbs, and Rowell (1978); and Saltin, Mitchell, Schibye and Payne (1978). Buck et al. (1980) evaluated systolic BP responses during isometric contractions of large and small muscle groups and reported that systolic BP increased more rapidly and reached higher absolute levels during fatiguing handgrip contractions at 40% MVC compared to similar contractions of index finger adductor muscles. Lind and McNicol (1967a) had suggested that a lower limit of muscle mass may exist which is required to elicit a maximal pressor response to a sustained muscle contraction. This could explain the differences between the results of the two studies.

McCloskey and Streatfeild (1975) substantiated the importance of percentage MVC by examining the pressor responses to handgrip and finger exercise. However, Buck et al. (1980) disagreed with McCloskey and Streatfeild's findings, maintaining that there was no significant difference between the muscle mass of the index finger adductor muscle and the flexors of the little finger. Buck et al. (1980) also believed that additional muscle recruitment had occurred in the studies by Lind and McNicol (1967a) and by McCloskey and Streatfeild (1975) which may have affected their results. This was based upon the fact that, during a sustained isometric handgrip contraction, a progressive

spread of excitation to synergistic muscles occurs as fatigue ensues (Ramos, Mundale, Awad, Witsoe, Cole, Olson & Kottke, 1973). The amount of recruitment and the rise in BP were related to the degree of intensity and duration of the contraction.

Cardiovascular Responses to Dynamic Work

Dynamic work is certainly the most common type of work. Whenever an external mass is moved some distance, dynamic work can be quantified as a product of the developed force multiplied by the vertical displacement (Åstrand & Rodahl, 1977). Dynamic work can be observed often in day-to-day activities such as walking and running. Adjustments made by the cardiovascular system to dynamic work have been extensively reported, so will not be documented in detail here. A cursory review of the effects of dynamic work on BP, peripheral resistance, blood flow distribution, HR, and SV will be given.

Blood Pressure

Blood pressure during dynamic work has been reported to respond very differently from that during static work (Bruce et al., 1968; Lind, et al., 1964; Lind & McNicol, 1967c; Tuttle & Horvath, 1957). Normally, the systolic pressure is increased during exertion, and the amount of increase is dependent upon the severity of exertion. The diastolic pressure remains unchanged or might decrease or increase only slightly, while the mean pressure increases in proportion to the increases in systolic and diastolic pressures.

Arterial BP is influenced by (1) cardiac output, (2) peripheral resistance, (3) elasticity of the arteries, (4) viscosity of the blood, and (5) blood volume (Åstrand & Rodahl, 1977). During dynamic exercise,

cardiac output and peripheral resistance are the main determinants of the BP response. With increasing levels of dynamic work, the cardiac output is increased in response to an increase in HR.

It is important to use a standard procedure in the measurement of BP, because the method by which BP is determined and the location of measurement are important considerations in obtaining reliable and valid measurements. Systolic pressure normally increases, and diastolic pressure decreases as the point of measurement moves from the aorta to more peripheral arteries at rest (McDonald, 1960) and during upright exercise (Rowell, Brengelmann, Blackmon, Bruce & Murray, 1968).

Kuhlemeier (1972) reported a correlation of $r = .96$ between invasively measured aortic and auscultatory measured brachial mean blood pressures, indicating that an exercise test which estimates central pressures from peripheral pressures is feasible.

Peripheral Resistance and Blood Flow Distribution

The normal response to dynamic exercise is a dilation of blood vessels in the active muscles (and later in the skin to reduce body heat) and a constriction of vessels in the splanchnic, renal, and hepatic beds. As a result of these changes, systemic vascular resistance may fall as much as 50% of the level found at rest (Lind & McNicol, 1967a). This fall in resistance obscures the significant visceral vasoconstriction, thereby maintaining mean arterial pressure (Rowell et al., 1968).

Blood flow is increased to the active muscles as dilation of their vessels occurs in proportion to the metabolic demand and is shunted away from the visceral area where it is not vitally needed. In general,

blood flow to the non-working muscles and internal organs (except the heart) decreases, and that to the working muscles increases in proportion to the workload, while cerebral blood flow remains constant throughout exercise.

Heart Rate, Stroke Volume, and Cardiac Output

It is well established that HR increases linearly with increasing workload (Åstrand & Rodahl, 1977). Normally, the HR increase is relatively large at the start of exercise and then nears steady state within 30 to 60 sec if the work is light or moderate (deVries, 1980). However, at higher workloads a steady state may not be achieved; and HR may reach maximal levels shortly before exhaustion occurs. Normal limits have been determined for maximal HR levels for each year of age, and there is a gradual decline in maximal HR with increasing age (Ellestad, Allen, Wan, Kemp, 1969). Maximal HRs are around 200 beats·min⁻¹ in healthy young men, but a standard deviation of ± 10 beats·min⁻¹ exists for maximal HR during exercise.

Stroke volume also increases during upright dynamic exercise and reaches a maximal value at a workload with an oxygen uptake of about 40% of the maximum and a HR of about 110 - 120 beats·min⁻¹ (Åstrand, Cuddy, Saltin, & Stenberg, 1964). Further, in healthy young subjects SV did not decrease during maximal exercise at HRs of or above 200 beats·min⁻¹, indicating that diastolic filling of the heart was not reduced at these rates.

The increase in HR and SV can elevate cardiac output to six times the resting level (Åstrand et al., 1964; Åstrand & Rodahl, 1977). At exercise levels up to 40 to 60% of a person's maximum capacity, increases

in both HR and SV are responsible for the increase in cardiac output. When exercise levels are higher, the increase results entirely from the continued increase in HR.

Cardiovascular Responses to Combined Static and Dynamic Work

Rarely are everyday activities purely static or dynamic in nature, but rather a combination of the two. Unfortunately, the research in this area of combination work has received relatively little attention.

In 1956, Hellebrandt, Houtz, Hockman, and Partridge had subjects perform static contractions with one hand while simultaneously contracting rhythmically with the other. Dynamic exercise was found to always augment the duration of the static contraction. Dynamic work was also increased, but to a lesser degree. Also, when rhythmic contractions were continued until near fatigue, and then the contralateral hand began doing static work, a reversal in the fatigue curve was observed to occur.

Lind and McNicol (1967a) evaluated the cardiovascular responses to sustained handgrips at three tensions (20, 30, and 50% MVC) while subjects walked on a treadmill at three different levels of O_2 uptake (1.1, 1.7, and $2.8 \text{ l}\cdot\text{min}^{-1}$). The increments in HR and BP at 20 and 30% MVC were similar at all levels of O_2 uptake. However, under the most extreme combination of static and dynamic exercise that Lind and McNicol examined (50% MVC during treadmill walking at an O_2 cost of $2.8 \text{ l}\cdot\text{min}^{-1}$), the BP and HR response was less than during the milder walking rates. They concluded that the systemic resistance at the fastest rate of walking was so reduced by the demands of the dynamic exercise that the combined effect of increasing cardiac output plus the

additional vasoconstriction were not sufficient to produce the normal pressor response to 50% MVC.

Despite this, the raised perfusion pressure due to the static contraction caused an increase in the volume of blood flowing through these muscles engaged in isometric contraction and presumably also through these muscles performing rhythmic exercise. Finally, the mechanical resistance to flow caused by the isometrically contracting muscles could increase the volume of blood available to the general circulation for rhythmic contractions (Lind & McNicol, 1967a). Although the hemodynamics are quite different in rhythmic and isometric work, the cardiovascular responses to sustained handgrip contractions while walking on the treadmill were typical in both pattern and magnitude to those obtained when the handgrip was done singly. This is in contrast with Sanchez et al. (1979) and Jackson et al. (1973) who reported that combined work had a higher physiological cost (HR and O_2 uptake) than the simple sum of the dynamic plus the static work.

Lind and McNicol (1968) and Petrofsky and Lind (1978) evaluated the cardiovascular responses to carrying and lifting tasks. In an experiment in which teams of two men walked at 2 mph on the level carrying a stretcher, on one occasion by hand and on the other occasion by a shoulder harness, extreme differences in HR and BP were noted. When the stretcher was carried by hand, the HR and mean BP rose steadily with final values at 3 min being 145 beats·min⁻¹ and 143 mm Hg, respectively. In contrast, the corresponding values at 3 min when the stretcher was supported in a shoulder harness were 124 beats·min⁻¹ and 96 mm Hg, respectively. The subjects were fatigued after 3 min of hand carriage, whereas after 15 min of carriage by the shoulder straps, they were not.

These results not only confirmed earlier research by Lind and McNicol (1967a, 1968) which indicated that it is the proportion of the maximum tension that a muscle exerts rather than the size of the muscle mass which induces these cardiovascular responses, but it also illustrated the magnitude of cardiovascular stress which can be imposed on a person by combination work if not done as efficiently as possible. The fact that combined static and rhythmic effort increases HR disproportionately in comparison with the increase in maximum oxygen consumption ($\dot{V}O_2$ max) has been confirmed often (Lind & McNicol, 1967a). Minute ventilation has also been shown to increase directly with rhythmic exercise, but disproportionately to the metabolic need when static effort was added, producing an inefficient, energy-consuming hyperventilation (Wiley & Lind, 1975).

Cardiovascular Adaptations to Aerobic Training

The cardiovascular system has been the center of investigation for many years, but only in the last two decades has the functioning of the cardiovascular system during exercise been intensively examined. Numerous researchers (Clausen, 1976; Ekblom, 1969; Frick, Konttinen, & Sarajas, 1963; Saltin, Blomqvist & Mitchell, 1968; Siegel, Blomqvist, & Mitchell, 1970) have conducted longitudinal and cross-sectional studies to evaluate heart rate (HR), blood pressure (BP), stroke volume (SV), cardiac output ($\dot{Q}$), pressure rate product (PRP) and maximal oxygen consumption ($\dot{V}O_2$ max) before and after an aerobic training program.

Cardiovascular adaptations to aerobic training are dependent on numerous factors such as the level of fitness at the onset of training, genetic endowments, age, health status, previous training (especially

regarding length of time and age it occurred), and the actual structure and length of the training program (Froelicher, 1973). Specific considerations must also be given to the type, frequency, duration, and intensity of the aerobic exercise if optimum cardiovascular gains are to be seen (Froelicher, 1973; Siegel et al., 1970).

It is well known that a reduction in resting HR occurs fairly predictably in response to a period of aerobic training and that trained athletes have lower HRs at rest when compared to normal sedentary people (Clausen, 1976; Saltin et al., 1968). The mechanism responsible for this sinus bradycardia at rest is attributed to a greater dominance of the parasympathetic system (increase in vagal stimulation) and a reduction in the sympathetic drive. Although other factors such as cardiac hypertrophy, increased blood volume and reduced stimulation from peripheral receptors may also carry some of the responsibility for the sinus bradycardia, they have not been substantiated by conclusive evidence (Clausen, 1976).

In addition to a lower HR at rest, aerobically trained persons, when compared to untrained persons, also have a decrease in HR during submaximal exercise (Boyer, 1972; Buskirk, 1973; Clausen, 1976; Frick, 1968; Froelicher, 1973; Wilmore & Norton, 1975). This reduction in HR is believed to be the result of a larger SV which is attributed to myocardial hypertrophy or to an increase in blood volume. Froelicher (1973) reviewed 10 training studies (6 dealing with normal men and the remaining 4 with men having cardiovascular problems). The HR was reduced in all of the trainees, both at rest and during submaximal exercise, and in the studies in which SV was measured it was increased in every case during maximal and submaximal exercise.

Cardiac output ($HR \times SV$) has been studied extensively in subjects at rest and while performing submaximal and maximal exercise. In the majority of training studies reviewed by Froelicher (1973), cardiac output was reduced at rest following physical training, unchanged at submaximal work, and increased during maximal exercise. Frick et al. (1963) reported a 10% increase in the resting cardiac output following a 2-month training period. This increase was attributed to a larger stroke volume even though the resting heart rates had been reduced. Although there is still some controversy in the literature regarding cardiac output changes following training programs, the majority of literature is in support of higher cardiac values for trained persons performing maximal work (Wilmore & Norton, 1975).

The effects of training on systolic and diastolic blood pressure have been well documented in the literature. Boyer and Kasch (1970) evaluated the effects on BP of a 6-month walk/jog training program meeting twice weekly in 2 groups of hypertensive and nonhypertensive (normal) men. Reductions in systolic and diastolic pressures of 13 mm Hg and 12 mm Hg, respectively, were noted in the hypertensive group, while the diastolic pressure in the normal group dropped 6 mm Hg with no change in the systolic. The authors commented that the drop in blood pressure was probably due to the vasodilating effect of endurance exercise.

Montoye, Metzner, Keller, Johnson, & Epstein (1972) also reported that more active men had significantly lower systolic and diastolic BP as evidenced from the Tecumseh Project in which 1700 men were studied. Frick et al. (1963) also reported decreases in their subjects' resting

systolic, diastolic, and mean pressures following a 2 month training program. Similar findings were presented by Buskirk (1973).

These decreases in BP appear to result from the decreased HR response and to a peripheral vasodilation and blood redistribution during submaximal exercise. Although the greatest changes in BP occur in those persons who at the onset of an exercise program have an abnormally elevated pressure, the evidence does not discount the benefits of reduced pressures for normals involved in aerobic training.

The trained heart works at a lower rate and ejects a larger volume than the untrained heart. This increase in stroke volume requires for a given workload a smaller amount of oxygen than an untrained heart. Therefore, the trained heart is more efficient and can perform with less stress at relative workloads compared to the untrained heart. Myocardial work, myocardial oxygen demand, can be assessed by the pressure-rate product (PRP) ($\text{systolic blood pressure} \times \text{heart rate}/100$). As work increases, HR and systolic BP normally increase, and therefore the myocardial oxygen demand increases. Trained persons have a reduced PRP at a fixed workload when compared to the untrained (Buskirk, 1973), and therefore they have an improved power function and efficiency of the myocardium (Boyer, 1972; Frick, 1968; Frick et al., 1963).

In addition to these cardiovascular adaptations to aerobic training, the literature is replete with studies reporting increases in $\dot{V}O_2$ max following training (Clausen, 1976). Oxygen consumption can be calculated as the product of cardiac output ($\dot{Q}$) and the arteriovenous oxygen difference ($a-\dot{V}O_2\text{diff}$) (Clausen, 1976). The $a-\dot{V}O_2\text{diff}$ represents the difference in concentration of oxygen in the arterial and venous blood. An increase in the $a-v\dot{O}_2\text{diff}$ indicates an increase in oxygen utilization by the

tissues. As the $\dot{Q}$ increases in proportion to the workload, the supply of blood to the active tissues is increased. Trained persons have been shown to have an increased $a-\bar{v}O_2\text{diff}$ and a higher $\dot{Q}$ at maximal work levels (Buskirk, 1973; Wilmore & Norton, 1975). This greater oxygen extraction from the increased $\dot{Q}$ is responsible for the enhanced aerobic capacity in the trained individual.

In conclusion, the results of a physical conditioning program of sufficient duration, intensity, and frequency will induce the physiological changes discussed. These changes enable a person to more efficiently respond to demanding work, therefore placing less stress on the cardiovascular system.

Statement of the Problem

The purpose of this study was to investigate the effect of aerobic capacity on cardiovascular responses (HR, BP, PRP) elicited by static, rhythmic, and a combination of static and rhythmic work (combination work). Additional experiments were carried out to evaluate the effect of an isometric handgrip contraction performed during maximal dynamic work on $\dot{V}O_2\text{ max}$.

Research Hypotheses

It was hypothesized that subjects having a high $\dot{V}O_2\text{ max}$ would exhibit a lower BP, HR, and PRP response when compared to subjects with a moderate or low $\dot{V}O_2\text{ max}$ during static, rhythmic, and combination work. It was also hypothesized that an increase in $\dot{V}O_2\text{ max}$ would occur when an isometric handgrip contraction was superimposed on maximal dynamic work.

Assumptions

1. All subjects exerted maximum effort for determination of their MVC.
2. Psychological or emotional influences on cardiovascular responses were randomly distributed.

Limitations of the Study

1. Subjects were instructed not to eat or drink anything other than water for at least 2 hours before each test. Strenuous physical activity was also a suggested constraint for at least 6 hours before a test. There was no assurance that subjects adhered to these recommendations.
2. Psychological factors other than those perceived from exertion may have influenced each subject's cardiovascular responses.
3. Generalization of the results to persons with cardiovascular heart disease was not possible since subjects in this study were assumed to be free from cardiovascular heart disease.

Significance of the Study

Cardiovascular responses during isometric (static) and isotonic (rhythmic) work are of interest to researchers, medical practitioners, coaches, and physical educators. Of particular concern are the effects of isometric exercise on BP, HR, cardiac output, left ventricular performance, and myocardial oxygen consumption (Haissly, Messin, Degre, Vandermoten, Demaret, & Denolin, 1974; Helfant, DeVilla, & Meister, 1971; Kivowitz, Parmley, Donoso, Marcus, Ganz, & Swan, 1971; Quarry & Spodick, 1974; Strong, Miller, Striplin, & Salehbhai, 1978).

The pressure rate product has been established as a correlate of myocardial oxygen consumption (Robinson, 1967), and angina pectoris is known to reoccur in patients at a predictable PRP level during graded exercise testing (Wahren & Bygdeman, 1971). Due to the augmentation of cardiovascular responses by static work when done concomitantly with rhythmic work, persons often unknowingly place themselves in dangerous situations. This is especially true of persons who have coronary heart disease. Angina is normally a result of a rise in the work of the myocardium to a level which is essentially fixed in each patient.

Wahren and Bygdeman (1971) reported that in coronary heart disease patients HR, PRP, peak systolic, systolic mean, diastolic, and mean arterial pressures all increased more steeply during arm exercise than during leg exercise even though a smaller workload was performed by the arms. These increases were associated with the use of a smaller muscle mass and with the static component of work performed by the muscles of the back and hip that help to stabilize the body. Static exertion, which often occurs suddenly in everyday life, can place a person close to dangerous circulatory overload with little warning. A good example is "airport angina" which is a severe and unexpected angina which some persons experience while walking at a normal rate through an airport while carrying a briefcase or suitcase (Jackson et al., 1973).

Aerobic-type activities are known to improve the functional capabilities of the cardiovascular system. A person with a high aerobic capacity, compared to a person with a low capacity, works at a lower relative workload in everyday dynamic activities. An important question is whether or not aerobic training can reduce the cardiovascular

responses imposed upon the body during combination work. An aerobically trained person would be expected to respond to any standard exercise with lower arterial BP, HR, and PRP than a person with a low aerobic capacity.

One of the limiting factors in aerobic activities is the ability to deliver oxygen to the working muscles. Because isometric work causes a dramatic rise in BP and therefore increases perfusion pressure and blood flow to the exercising muscles, an isometric hand contraction might possibly enhance $\dot{V}O_2$ max and thus benefit athletic performance.

The consequences of this study might be (1) additional support for aerobic training of both normal persons and those with heart disease in order to reduce the cardiovascular responses to combination work and (2) support for the use of isometric contractions during dynamic work in an effort to increase $\dot{V}O_2$ max and thereby increase performance.

CHAPTER 2

METHODS

Introduction

Different cardiovascular responses are elicited by static and dynamic work. However, the cardiovascular responses from combination work are not easily separated. In order to evaluate the effects of aerobic fitness on cardiovascular responses to combination work, three groups having high, moderate, and low levels of aerobic capacity were compared under three conditions in a counter-balanced design: (1) static work; (2) dynamic work; and (3) combination work. An additional experiment was used to examine the effect on $\dot{V}O_2$ max of adding a static contraction during maximal rhythmic work.

Subjects

Subjects for this study were 18 young, healthy males who were selected on the basis of $\dot{V}O_2$ max (Table 1). The design of the study dictated that a prime criterion for acceptance as a subject was that $\dot{V}O_2$ max ($\text{ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$) should be below 51 for a low fit group, between 51 and 60 for a moderately fit group, and above 60 for a high fitness group.

Prior to the selection of the subjects, those having any medical or physical limitations which would prevent them from engaging in exhaustive exercise were excluded. The exclusion was based on results obtained from a questionnaire which the potential subjects completed at an orientation session. As far as could be determined, all subjects were non-smokers and had no physical or mental impairment which would

Table 1
Descriptive Data of Subjects

Group	Age (yrs)	Height (cm)	Weight (kg)	$\dot{V}O_2$ max (ml · kg ⁻¹ · min ⁻¹)	MVC (kg)
Low Fit (n = 5)	24 ±5	179 ±3	86 ±18	47 ±5	58 ±7
Mod Fit (n = 5)	22 ±2	179 ±5	74 ±7	55 ±1	57 ±10
High Fit (n = 8)	23 ±5	178 ±8	70 ±12	64 ±3	56 ±11

have affected performance. The orientation session allowed the subjects to become familiar with all of the equipment and with the testing procedures. A written summary of the purpose of the study and the procedures to be used in the research were given to each subject, and all gave written consent prior to being tested.

Testing Procedures

Preliminary Tests

Prior to performing the three tests, the subjects came to the laboratory for an orientation session during which time they underwent a simulation of all testing procedures. Height and weight were measured by standard procedures with the subjects wearing only shorts. Right handgrip MVC was determined at this time by having the subjects perform three maximal voluntary contractions with durations of less than 2 sec and at intervals of 1 min. The mean of these was accepted as the MVC. A Lafayette grip dynamometer (Model 78010) was used in this procedure.

During the latter portion of the orientation, Hewlett-Packard electrodes (No. 14389A) were applied to CM5 lead sites following standard skin preparation procedures, and the subjects underwent the standard or superstandard treadmill test protocol of the International Committee for the Standardization of Physical Fitness Tests (ICSPFT) (Larson, 1974, pp. 476-478). The ICSPFT standard protocol was used on those volunteers who subjectively rated themselves as being only moderately aerobically conditioned. The standard protocol involved walking at 3.0 mph at 2.5% grade for 2 min and increasing the grade every 2 min until the subjects reached 85% of their age-estimated maximum HR ($220 - \text{age}$). Those subjects who rated themselves as highly

aerobically conditioned were tested using the super-standard protocol. This involved walking at 3.75 mph at 4% grade and increasing the grade 2% each min until the subjects reached 85% of their age-estimated maximal HR. The $\dot{V}O_2$ max for all subjects was estimated by extrapolating the subject's HR to the predicted maximal level and estimating the maximum workload in metabolic equivalents (METS) which could be obtained at this maximum HR. For these and all other tests, HR was calculated from R-R intervals across four ECG complexes, recorded on a Hewlett-Packard 1511-B electrocardiograph.

Maximal Treadmill Test

To determine actual $\dot{V}O_2$ max, the subjects performed the following test based upon their estimated $\dot{V}O_2$ max from the ICSPFT treadmill test. The subject warmed-up for 5 min by walking at a speed and grade calculated to require 30% of $\dot{V}O_2$ max followed by a speed and grade for 2 min estimated to require 80% of $\dot{V}O_2$ max. Thereafter, continued 2% increases in grade were made each minute until exhaustion was reached.

Measurements of $\dot{V}O_2$ and HR were made every 15 sec following the warm-up and continued until the test was terminated. Blood pressure was measured by standard auscultatory techniques during the last 30 sec of each min following the warm-up until the test was terminated. The same technician made all BP measurements. A standard open circuit method was used to measure $\dot{V}O_2$ and the criterion for achievement of maximum was observation of a plateau in $\dot{V}O_2$ accompanying an increase in workload.

Subsequent to the maximal test, a rotation-group technique was utilized in the assignment of test conditions to counterbalance any ordering

effect. A minimum of 2 days elapsed between each of the following: orientation session, $\dot{V}O_2$ max test, and experimental test conditions. All experimental test conditions were performed on the same day with a 15 min recovery period between each condition.

Static Test

Each subject stood on the treadmill belt with his right hand resting on the right side railing. The hand rested so that the railing passed between the first and second metacarpals. The subject then grasped the handle of a briefcase containing weight equivalent to 20% of the subject's MVC which was determined on the orientation day. The weight of the handle rested midway between the head and base of the proximal phalanges 2, 3, and 4. The subject held the contraction for 5 min, with BP being measured in the left arm during the last 30 sec of each min by the standard auscultatory method. The same technician made all BP measurements on all of the subjects. Heart rate was calculated from ECG recordings which were taken every 15 sec.

Dynamic Test

Subjects walked for 5 min on the treadmill at a speed and grade estimated to elicit 30% of their measured $\dot{V}O_2$ max. ECG traces were recorded each 15 sec and BP was measured in the left arm during the last 30 sec of each min by standard auscultatory methods.

Combination Test

Subjects walked for 5 min on the treadmill at a speed and grade estimated to elicit 30% of their measured $\dot{V}O_2$ max while simultaneously

replicating procedures used in the static test. Heart rate and BP were recorded as in the static and dynamic tests.

Maximal Test Plus Isometric Contraction

Immediately following the last of the three treatments, the treadmill speed and grade were set to a level which demanded 80% of the subject's $\dot{V}O_2$ max. Following 2 min at this speed and grade, the subject was instructed to maximally contract and sustain his grip on a rubber ball. Simultaneously, the treadmill grade was increased as in the previous maximal treadmill test until the subject could no longer continue. The $\dot{V}O_2$ max obtained from this test was compared to that from the previous maximal treadmill test to evaluate the effect of the isometric contraction. The method of measuring $\dot{V}O_2$ max, the times during which data were taken, and the criterion for achievement of $\dot{V}O_2$ max were exactly as in the previous maximal test.

Statistical Analysis

A 3(group) X 3(trial) MANOVA with repeated measures on trials was used to determine whether differences existed among three conditions (static, dynamic, combination work) and at three levels (high, moderate, and low aerobic capacity). The three dependent variables used in the MANOVA were HR, systolic and diastolic BP. Following MANOVA, examination of univariates were conducted. Duncan's Multiple Range test were performed on all significant univariates.

Because PRP is a product of SBP and HR a separate 3(group) X 3(trial) ANOVA was performed to avoid multicollinearity. When significant differences ($p < .05$) were found, Duncan's Multiple Range test was performed to isolate the differences.

A 3(level) X 2(condition) factorial analysis of variance was used to determine whether differences existed among three fitness levels (high, moderate, and low aerobic capacity) under two conditions (dynamic and combination work). Maximum oxygen uptake ($\text{ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$) was the dependent variable examined in this analysis.

CHAPTER 3

RESULTS AND DISCUSSION

Results from the Wilks' Lamda MANOVA indicated no significant group differences ($F_{2,15} = 1.45$) but significant trial differences ($F_{2,30} = 34.04$). Follow up ANOVA's were conducted on the dependent variables HR, systolic and diastolic BP for the trial differences, and when significant results were found Duncan's Multiple Range tests were performed to isolate the differences between means (Tables A3 - A8).

Heart Rate

Significant differences ($p < .01$) were found in HR across trials for all groups (Figure 1). Significant differences ($p < .05$) in HR were found between all modes of exercise. Static work was the least demanding, dynamic was intermediate in stress, and combination work was the most rigorous.

The significant differences that were found across trials were not unexpected, being in general agreement with the literature (Astrand & Rodahl, 1977; Donald et al., 1967; Freyschuss, 1970; Kilbom & Persson, 1981; Lind & McNicol, 1967a). The acceleration of HR at the onset of static exercise is a result of vagal withdrawal and to a function of the percentage of MVC involved (Shepherd, Blomqvist, Lind, Mitchell, & Saltin, 1981). Besides vagal withdrawal, this contraction also caused increased sympathetic activity, the combination increasing HR to the appropriate level within seconds. Increased HR and the squeezing-out of venous blood from the muscles at the beginning of the static exercise

Figure 1. Means and standard errors for HR (group x trial)

increase venous return to the heart and are responsible for the rapid increase in SV and $\dot{Q}$.

The subjects in this study were performing at 20% MVC, so the HR response was slight. During dynamic work, the HR typically increases linearly with increasing workload and may reach maximal levels shortly before exhaustion occurs (Åstrand & Rodahl, 1977). The subjects in this study were working at only 30% $\dot{V}O_2$ max, so only small increases were observed in HR response. The metabolic demand of the large muscle mass used in walking at 30% $\dot{V}O_2$ max would clearly be much greater than that required to hold a static contraction of a small muscle group at 20% MVC. This, rather than differences in vagal withdrawal and/or sympathetic stimulation, probably constitutes the primary cause of observed differences, although the other factors mentioned may contribute.

Heart rates for combination work were significantly higher than for either static or dynamic work alone. This is in agreement with Jackson et al. (1973) and Sanchez et al. (1979). However, Kilbom & Persson (1981) reported that at combination work above 95% $\dot{V}O_2$ max the HR response was unaffected by the static component to a near-maximal sympathetic activation. At levels less than 95% $\dot{V}O_2$ max combination work does produce an additive stress on the cardiovascular system, which was evidenced in the present study. That the stress is additive does not mean that HR response to combination work could be predicted by adding increments above resting level from the other two bouts. There are complex interactions among neural and metabolic control mechanisms for combination work which were not investigated in this study, and constitute a challenge for further research.

No significant differences in HR occurred among the 3 groups, indicating that aerobic fitness probably does not affect the degree to which a person responds to the three experimental treatments (Figure 1). It has been clearly established that HR responses to absolute levels of work are inversely related to fitness level (Clausen, 1976), but apparently in exercise relative to individual capacities, responses are unrelated to aerobic fitness. Use of absolute loads would certainly elicit significant group differences in dynamic work, but responses to static and combined exercise would be of interest. Another point that should be examined is the degree to which similar results would be obtained if higher relative loads were used.

Blood Pressure

Systolic blood pressure was significantly ($p \leq .05$) higher for all groups during combination work (Figure 2). This elevation is a result of an increased cardiac output that must be moved into the arterial tree against a significantly greater peripheral resistance. Resistance to blood flow occurs locally when static contractions cause a mechanical hindrance to flow by higher intramuscular pressures (Asmussen, 1981). In addition, a general vasoconstriction occurs in inactive tissues during dynamic work (Lind & McNicol, 1967a; Perez-Gonzalez, 1981). These factors interact to necessitate a higher driving force, the perfusion pressure.

No significant differences were found between the SBP responses to static and dynamic work (Figure 2). The light static contractions (20% MVC) caused only slight increases in HR ($12 \text{ beats} \cdot \text{min}^{-1}$) and $\dot{Q}$, and required only mild perfusion pressures to overcome intramuscular

Figure 2. Means and standard errors for systolic and diastolic blood pressure (group x trial).

tensions. Similarly, the dynamic work was mild ($30\% \dot{V}O_2 \text{ max}$) which did not increase HR substantially ($34 \text{ beats} \cdot \text{min}^{-1}$) over rest. Evidently, the lower HR with the greater peripheral resistance was equivalent to the slightly higher HR and lower resistance in terms of demands for perfusion pressure.

No significant group differences ($p < .05$) in SBP responses were evident, indicating that aerobic fitness level does not alter the SBP response to the three types of work examined. This is in contrast to literature reporting that SBP is lowered at rest and during exercise following aerobic training (Frick et al., 1963; Montoye et al., 1972). One explanation for this difference might be that the use of relative rather than absolute loading did not significantly stress the subjects' pressure regulating mechanisms to cause differences at these low work levels. It would be of interest to observe the affect of absolute loads on SBP under these three experimental conditions.

The diastolic blood pressure for all groups was most elevated during static work (Figure 2), in agreement with the literature (Tuttle & Horvath, 1957; Whipp & Davis, 1970). Static work increases DBP as a result of the increases in HR and $\dot{Q}$ which provide a greater perfusion pressure. This compensates for the increased peripheral resistance caused by compression of blood vessels (Lind et al., 1964). Diastolic blood pressure was significantly lowered ($p < .05$) during dynamic work compared to static work (Figure 2). This is characteristic of this type of exercise and reflects the generalized vasodilation occurring in the active tissue along with removal of the mechanical obstruction caused by high intramuscular pressures in static work.

During combination work the DBP was significantly increased above that found during dynamic work, but was still significantly lower than that measured during static work. Active tissue vasodilation from the dynamic component of combination work may account for this reduced response. During dynamic work, the main determinants of the BP response are $\dot{Q}$ and peripheral resistance. Normally, SBP and mean arterial pressure increase when $\dot{Q}$ increases in response to a rise in HR. During this response, vasodilation in the active muscles and vasoconstriction in the inactive regions occur, controlling changes in DBP and SBP. However, during combination work the normal balance between vasoconstriction and vasodilation is upset by the static component. Staunton et al. (1964) postulated that the diastolic pressure reflex operated such that perfusion pressure progressively increased as blood flow became inadequate for metabolic requirements or for the removal of metabolites. Also, with static contractions, hindrance to blood flow occurs by the increased peripheral resistance and DBP increases.

No significant group differences ($p < .05$) were found, suggesting that aerobic fitness does not affect DBP response to static, dynamic, or combination work, at least when the work bouts are set as percentages of maximum capacities. The question remains as to the responses to absolute loading at various levels of work.

Pressure Rate Product

Pressure rate product ($SBP \times HR/100$) results for static, dynamic, and combination work were all significantly different ($p < .05$) (Figure 3). The PRP for all groups increased sharply from static to dynamic work and from dynamic to combination work. Because PRP is directly

Figure 3. Means and standard errors of maximum pressure rate products (group x trial).

related to oxygen consumption by the heart, this indicated that the myocardial oxygen demands were greatest during combination work and least during static work.

These findings are in agreement with those of Haissly et al. (1974), Jackson et al. (1973), Lind & McNicol (1967a), and Robinson (1967) who reported that combination work had a higher physiological cost (HR and O_2 uptake) than either static or dynamic work alone. The cardiovascular system responds with an increase in SBP and HR which in turn results in a significantly increased PRP.

With significantly higher SBP and HR in combination work, it was inevitable that the higher PRP for that type of work should occur, PRP being computed by $SBP \times HR + 100$. Also, because SBP for static and dynamic work was similar, PRP for these conditions reflected significant HR differences. The importance of using PRP lies in its being a much more valid predictor than HR or SBP alone of myocardial demand and insufficiency of oxygen. The large and significant differences reflect a critical point, that, with respect to this indicator of myocardial need for oxygen, combination work triggers significant and meaningful increments in PRP above baselines set by either static work or dynamic work alone. It is emphasized that this is true for light work combinations; the occurrence of "airport angina" under more severe conditions is understandable. In addition, myocardial supply and demand are also a function of relative fitness; what might be an acceptable PRP and oxygen demand for the fit heart might be deadly to one who suffers from some pathological condition.

Unexpectedly, there were no significant differences between groups, suggesting that aerobic capacity does not affect the PRP response to

work in standardized work relative to individual maxima. This is contrary to the findings of Boyer (1972), Buskirk (1973), Frick (1968), and Frick et al. (1963) who reported that PRP was lower in more active persons and could be lowered by aerobic training. The obvious explanation for this discrepancy is found in consideration of the differences in responses to absolute and relative loads.

$\dot{V}O_2$ Max

Significant differences in $\dot{V}O_2$ max appeared between groups simply as a condition of subject assignment and experimental design. There were no significant differences ($p < .05$) in $\dot{V}O_2$ max between the first test (dynamic work only) and the second test (dynamic plus static work) (Table 2). The fact that these means would be significantly different if considered at an alpha of .20 may justify further investigation on the effects of static contractions on $\dot{V}O_2$ max. Certainly, both practical and theoretical questions arise in examining the relationship.

It would appear that when static and dynamic work are combined, an increase in $\dot{V}O_2$ max should occur due to the increased blood flow to the active muscles caused by the higher perfusion pressure elicited by the static component. However, the results in this study did not support this theory. The type and intensity employed in conjunction with dynamic work did not significantly affect $\dot{V}O_2$ max.

Kilbom & Persson (1981) evaluated the responses to combined static handgrip at 20% MVC and dynamic leg exercise at levels of 25%, 45%, and 95% of $\dot{V}O_2$ max. Their results indicated that, as maximal oxygen uptake was approached, the HR and BP response to handgrip contractions was progressively less marked. At 95% of $\dot{V}O_2$ max, the systolic BP response

Table 2
Means and Standard Deviations for
 $\dot{V}O_2$ max (ml·kg⁻¹·min⁻¹)

Work Type	High $\dot{V}O_2$ max	Mod $\dot{V}O_2$ max	Low $\dot{V}O_2$ max
Dynamic	64 ±3	55 ±1	47 ±5
Dynamic + static	65 ±3	56 ±2	47 ±7

to handgrip was abolished probably as a result of a competitive use of the same pressor response mechanism by the dynamic work, and because the vasoregulatory mechanisms in the leg muscles counterbalanced the rise in BP.

This explanation could well be the reason that there was no significant increase in $\dot{V}O_2$ max in this study. Because the subjects did not begin their static contraction until they were at over 90% $\dot{V}O_2$ max, the potential additive BP response from the static work was abolished, and the increase in perfusion pressure which should have enhanced oxygen delivery to the muscles was not present. Possibly, had the subjects performed the static contraction at lower percentages of $\dot{V}O_2$ max, significant differences might have been observed. However, it was the purpose of this study to evaluate the effects of static contractions during maximal work to determine whether any increases in $\dot{V}O_2$ max would occur which might have implications for athletic performance.

Summary and Conclusions

The results indicate that aerobic fitness level does not affect the HR, SBP, DBP, or PRP response to work at 20% MVC, dynamic work at 30% $\dot{V}O_2$ max, or to a combination of the static and dynamic work. Furthermore, $\dot{V}O_2$ max was not increased by the addition of static work during a maximal dynamic test. However, static, dynamic, and combination work did have significant effects on HR, SBP, DBP, and PRP. Heart rate and PRP were highest during combination work and lowest during static work. Systolic and diastolic blood pressure were highest during combination and static work, respectively, while DBP was lowest during dynamic work. No differences in SBP were observed between static and dynamic work.

Although the results did not support the hypotheses that high $\dot{V}O_2$ max subjects will have reduced HR, SBP, DBP, and PRP than moderate or low $\dot{V}O_2$ max subjects, they did support the literature regarding the effects of static, dynamic, and combination work on HR, SBP, DBP, and PRP. Static work was shown to elicit the highest increases in DBP and combination work caused the highest increases in SBP, HR, and PRP. These results are important, especially to persons having cardiovascular problems such as hypertension or angina. A knowledge of the effects of static and combination work on the cardiovascular system would enable these persons to limit their activities to levels which do not result in dangerous cardiovascular changes.

This study has presented a number of areas in which additional research should be pursued. A question remains as to the responses to absolute loading at various levels of work. The results suggest that, at absolute loads, differences in HR, SBP, DBP, and PRP might appear in subjects having different $\dot{V}O_2$ max levels. Furthermore, the differences in $\dot{V}O_2$ max levels between dynamic work and dynamic plus static work justify further investigation using static contractions at lower percentages of $\dot{V}O_2$ max. The possibility still remains that a static contraction might benefit performance by increasing $\dot{V}O_2$ max if the static contraction is performed at lower levels of $\dot{V}O_2$ max than were used in this study.

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APPENDIX A

Table A1
Dependent Variables by Subject
Static and Dynamic Work

Subject	Group	Trial	HR	SBP	DBP	PRP
1	1	1	84	121	95	102
2	1	1	78	121	88	94
3	1	1	68	134	97	91
4	1	1	75	133	89	100
5	1	1	76	139	89	99
6	1	1	73	147	91	107
7	1	1	94	128	75	121
8	1	1	96	122	71	117
9	2	1	90	126	95	112
10	2	1	104	151	112	158
11	2	1	82	147	95	121
12	2	1	94	139	102	131
13	2	1	69	115	81	80
14	3	1	64	139	89	89
15	3	1	92	140	101	129
16	3	1	88	110	81	95
17	3	1	83	125	87	103
18	3	1	71	135	101	97
1	1	2	105	137	60	144
2	1	2	91	145	67	132
3	1	2	88	133	64	117
4	1	2	86	123	61	106
5	1	2	105	153	65	160
6	1	2	68	131	62	89
7	1	2	106	121	55	127
8	1	2	130	135	60	176
9	2	2	113	128	65	145
10	2	2	136	141	71	192
11	2	2	105	131	64	137
12	2	2	109	149	71	162
13	2	2	95	123	60	116
14	3	2	82	141	64	116
15	3	2	97	121	73	117
16	3	2	95	101	64	100
17	3	2	131	149	65	195
18	3	2	98	127	83	124

Group 1 = High $\dot{V}O_2$ max
Group 2 = Mod $\dot{V}O_2$ max
Group 3 = Low $\dot{V}O_2$ max

Trial 1 = Static
Trial 2 = Dynamic

Table A2
Dependent Variables by Subject
Combination Work

Subject	Group	Trial	HR	SBP	DBP	PRP
1	1	3	100	135	77	134
2	1	3	89	144	67	129
3	1	3	91	125	75	114
4	1	3	107	152	59	163
5	1	3	105	156	78	164
6	1	3	85	155	87	131
7	1	3	111	149	61	162
8	1	3	124	139	69	172
9	2	3	114	147	71	167
10	2	3	150	164	81	245
11	2	3	110	146	86	160
12	2	3	113	156	73	176
13	2	3	97	116	65	113
14	3	3	86	152	78	131
15	3	3	104	141	83	147
16	3	3	115	117	65	134
17	3	3	138	153	75	212
18	3	3	103	134	95	138

Group 1 = High $\dot{V}O_2$ max
 Group 2 = Mod $\dot{V}O_2$ max
 Group 3 = Low $\dot{V}O_2$ max

Trial 3 = Combination

Table A3
Means and Standard Deviations
By Groups and Trials

Group	Variable	Static		Dynamic		Combination	
		X	SD	X	SD	X	SD
<hr/> High $\dot{V}O_2$ max <hr/>							
	HR beats·min ⁻¹	81	10	97	18	101	13
	SBP mm Hg	131	9	135	11	144	11
	DBP mm Hg	87	9	62	4	72	9
	PRP	104	11	132	28	146	21
<hr/> Moderate $\dot{V}O_2$ max <hr/>							
	HR beats·min ⁻¹	88	13	112	15	117	20
	SBP mm Hg	136	15	134	11	146	18
	DBP mm Hg	97	11	66	5	75	8
	PRP	120	29	151	29	172	48
<hr/> Low $\dot{V}O_2$ max <hr/>							
	HR beats·min ⁻¹	80	12	101	18	109	19
	SBP mm Hg	130	13	128	19	139	15
	DBP mm Hg	92	9	70	8	79	11
	PRP	102	16	130	37	152	34

Table A4
Analysis of Variance
for Heart Rate

Source	df	MS	F
Group	2	703.430	1.17
Error A	15	602.894	
Trial	2	3286.772	55.81**
Group*Trial	4	45.457	.77
Error B	30	58.889	
Total	53		

** $p \leq .01$

Duncan's Multiple Range Test ($p \leq .05$)

No significant Group differences.

Static significantly different from Dynamic
Dynamic significantly different from Combination
Combination significantly different from Static

TABLE A5
Analysis of Variance
for Systolic Blood Pressure

Source	df	MS	F
Group	2	158.422	.44
Error A	15	362.794	
Trial	2	696.285	8.94**
Group*Trial	4	19.839	.25
Error B	30	77.897	
Total	53		

** $p < .01$

Duncan's Multiple Range Test ($p < .05$)

No significant Group differences.

Static significantly different from Combination
Dynamic significantly different from Combination
Static not significantly different from Dynamic

Table A6
Analysis of Variance
for Diastolic Blood Pressure

Source	df	MS	F
Group	2	277.921	1.72
Error A	15	161.358	
Trial	2	2954.356	100.54**
Group*Trial	4	34.353	1.17
Error B	30	29.384	
Total	53		

** $p \leq .01$

Duncan's Multiple Range Test ($p \leq .05$)

No significant Group difference.

Static significantly different from Dynamic
Dynamic significantly different from Combination
Combination significantly different from Static

Table A7
Analysis of Variance
for Pressure Rate Product

Source	df	MS	F
Group	2	2175.563	1.19
Error A	15	1824.495	
Trial	2	9989.843	33.14**
Group*Trial	4	50.621	.17
Error B	30	301.435	
Total	53		

** $p \leq .01$

Duncan's Multiple Range Test ($p \leq .05$)

No significant Group difference.

Static significantly different from Dynamic
Dynamic significantly different from Combination
Combination significantly different from Static

Table A8
Analysis of Variance
for $\dot{V}O_2$ Max

Source	df	MS	F
Group	2	960.159	37.19**
Error A	15	25.820	
Time	1	6.953	2.05
Group*Time	2	.882	.26
Error B	15	3.395	
Total	35		

** $p \leq .01$

Duncan's Multiple Range Test ($p \leq .05$)

Group 1 significantly different from Group 2
Group 2 significantly different from Group 3
Group 3 significantly different from Group 1

$\dot{V}O_2$ max (dynamic) not significantly different
from $\dot{V}O_2$ max (dynamic + static)

Table A9
Wilks' Lambda MANOVA

Source	df	Wilks' Lambda	F
Group	2	.562	1.45
Subject (Group)	15		
Trial	2	.046	34.04***
Group*Trial	4	.686	.95
Error	30		
Total	53		

*** $p \leq .001$

VITA

John Francis Harper was born on September 27, 1951 in Florence, South Carolina. He was graduated from Frankfurt American High School in Frankfurt, Germany in 1969. He attended the University of South Carolina, Columbia, South Carolina, from 1969 to 1973 where he was graduated with a Bachelor of Arts Degree in Health and Physical Education.

Following graduation from the University of South Carolina, Dr. Harper accepted a graduate assistantship at the University of South Carolina, Columbia, South Carolina, and began working toward a Master of Arts in Teaching Degree. Dr. Harper accepted a position as a Teaching Associate at the University of South Carolina, Columbia, South Carolina in 1974 which delayed completion of his Master's Degree until 1975. During his three and one-half years as a Teaching Associate, Dr. Harper was lab director, ECG technician, instructor, and researcher.

Dr. Harper enrolled in the Doctor of Philosophy Degree program offered by the School of Health, Physical Education, Recreation, and Dance at Louisiana State University in 1978. Prior to completion of this degree, Dr. Harper accepted a teaching position as Assistant Professor in exercise physiology in the Department of Health, Physical and Recreation Education at West Texas State University, Canyon, Texas. The Doctor of Philosophy Degree was awarded in the major area of Exercise Physiology the Summer semester of 1982.

EXAMINATION AND THESIS REPORT

Candidate: John Francis Harper

Major Field: HPERD (Exercise Physiology)

Title of Thesis: Cardiovascular Responses in High, Moderate and Low Aerobic Capacity Males During Static and Dynamic Work

Approved:

Major Professor and Chairman

Dean of the Graduate School

EXAMINING COMMITTEE:

Date of Examination:

July 19, 1982