CARDIOVASCULAR RESPONSES TO SUSTAINED ISOMETRIC WORK IN A HOT ENVIRONMENT

by

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ABSTRACT

The purpose of this study was to investigate the changes in cardiovascular dynamics as depicted by systolic time intervals, blood pressure and heart rate during a 50% MVC and 100% MVC isometric contraction of the forearm in a control and heated environment.

Fourteen normal male volunteers aged 20 to 31 were used as subjects. Simultaneous recordings of the phonocardiogram, electrocardiogram, carotid pulse wave and blood pressure were conducted for each subject at rest and during exercise in a seated position. Subjects were tested in room temperature and in a sauna where the skin temperature was raised to 40°C - 41°C. Testing took place on two separate days with one day of rest in between. Half of the subjects experienced the heated conditions first, while the other half was tested in room conditions first.

The results from two of the subjects were discarded because of poor quality reproduction of the time interval recordings. For each recording only the three clearest cycles closest to the termination of the contraction period were used for statistical analysis. The data were treated with a two-way ANOVA for each dependent variable. In some cases a post-hoc analysis (Newman-Keuls method) was used to determine specific differences between workload or environment effects.

The fourteen dependent variables studied were divided into the following groups:

a) Systole related variables

left ventricular ejection time (LVET)

mechanical systole (MS)

total systole (TS)

ejection time index (ETI)

b) Diastole related variables

cycle time (CT)

diastole (DIAS)

c) Sympathoadrenergic Activity (Contractility)

pre-ejection period (PEP)

isovolumetric contraction period (ICP)

PEP/LVET (ratio)

d) Afterload

systolic blood pressure (BPs)

diastolic blood pressure (BPd)

e) Electromechanical Lag (EML)

f) Heart Rate (HR)

g) Myocardial Oxygen Consumption (Index)

triple product (TRIP)

CONCLUSIONS

1. The oxygen consumption of the myocardium as depicted by the triple product significantly increased during submaximal and maximal isometric handgrip contraction. This increase was evident at room temperature and during body heating.
2. There was no significant change in the myocardial oxygen consumption as depicted by TRIP at rest or during isometric forearm contraction

between the control and heated environments. This suggests that the heat stress did not significantly increase the myocardial oxygen requirements.

3. In a state of rest, increasing the skin temperature to between 40°C - 41°C did not significantly alter either BPs or BPd when compared to a resting state at room temperature. However, BPs and BPd were substantially lower during isometric work in the heat than during isometric work at room temperature.
4. BPs and BPd significantly increased during 50% MVC and 100% MVC static contractions of the forearm. This increase was demonstrated in both environmental conditions.
5. All variables depicting changes in left ventricular systole (LVET; MS; TS) and ventricular diastole (diastole and CT) were found to become significantly reduced with submaximal and maximal static contractions of the forearm. These changes were evident in both environments.
6. A strong inverse correlation was found between HR and LVET, CT and diastole. HR significantly increased from rest to 100% MVC in both environmental conditions. Consequently, it is suggested that alterations in LVET, CT and diastole are largely determined by the rate of myocardial contraction.
7. The ejection time index significantly increased in both environmental conditions with a 50% MVC and 100% MVC static contraction of the forearm.

8. The electromechanical lag showed a general tendency to decrease during an isometric handgrip contraction. However, subsequent post-hoc analysis (Newman-Keuls) demonstrated that EML did not significantly decrease during a submaximal or maximal isometric contraction of the forearm. It is suggested that care be taken to choose a proper statistical procedure for analysis of EML.
9. The contractility of the heart as depicted by changes in ICP, PEP and PEP/LVET increases in response to a pressure load produced by static exertion but is not significantly altered by an augmented volume load associated with heat stress.
10. HR, LVET, MS, TS, PEP, ICP, PEP/LVET and EML changed in an additive fashion from rest to 100% MVC during subjection to a volume load and pressure load simultaneously.

In contrast alterations in BPs, BPd, ETI, CT, diastole, and TRIP displayed interactive characteristics during the same test conditions.

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CHAPTER I

STATEMENT OF THE PROBLEM

I. INTRODUCTION

Recently, substantial effort has been incorporated into the investigation of the diseased heart, and as an offshoot of this work there has been much concern about the use of isometrics as a useful stressor to the heart for purposes of dynamic analysis. In contrast, there has been substantially less work performed on the changes in cardiac dynamics of a normal heart with bouts of isometric exercise. Since isometrics is reported by some to be a beneficial method of exercising the body and improving muscular strength, the popularity of practising isometric work has increased markedly. Such interest in isometric or static exercising has stimulated the curiosity of researchers, the majority of which seem to be hesitant to prescribe the use of static exercises for purposes of keeping fit. It would therefore be of interest to investigate the dynamic alterations in the cardiac cycle of a normal heart during sustained isometric contraction.

The heart may be stressed in a variety of ways, one of which has already been mentioned and another being the exposure of the body to higher than normal temperatures. Excessive heat and humidity are especially a burden to cardiac function and may induce fatal consequences for the diseased heart. The responsibility for transporting large quantities of heat from the central areas of the body to its surface, where it is mainly lost, lies primarily with the cardiovascular system. The cardiac

pump works with increasing power and speed of contraction in an effort to bring adequate quantities of blood to the skin surface. As a result the heart rate (HR), cardiac output (CO) and cardiac work done per unit time increase markedly. Imposing such a volume load stress on the heart undoubtedly causes the humoral and neural mechanisms of the body to react in favor of improving the cardiac dynamics in order to adequately meet the physiological demands of temperature regulation.

In addition to heat stress, the CV system is also responsive to changes in arterial pressure, whereupon a pressure load on the myocardial fibers during systole alters the contractile state of the fibers. Both systolic and diastolic pressure can be augmented temporarily during bouts of isometric contraction. In fact, during maximal voluntary contractions (MVC), the mean blood pressure has been shown to exceed resting values of hypertensive diseased individuals (33, 40). Since it is known that hypertensive states will cause alterations in cardiac dynamics (19, 23, 33) it would seem appropriate to suggest that an acute hypertensive state within a normal individual may also produce changes in cardiac dynamics.

Investigations into the effects of isometric exercise on the cardiovascular system can be divided into those which concern themselves with changes in left ventricular systolic time intervals and others which deal mainly with changes in hemodynamics. Lind and McNicol (38, 39) have conducted numerous studies concerning the effects of isometric exercise on the central circulatory responses. However the majority of these investigations were conducted in a neutral environment and little is known about cardiovascular adaptations to simultaneous stresses of heat

and static work in man. Rowell (69) reviews some classical experiments in cardiovascular adjustments to both dynamic exercise and thermal stress, but again studies concerning themselves with simultaneous induction of thermal and isometric stresses are rare. A substantial amount of information is known about hemodynamic adjustments of the cardiovascular system with isometric exercise and thermal stress separately, but no one has yet shown how these two quite different conditions will interact with each other in producing a coupled adjustment of cardiovascular function. Specifically it is of interest to this investigator to examine alterations in cardiac dynamics as depicted by the systolic time intervals during a variety of conditions involving combinations of thermal and isometric work stress. This approach is an attempt not only to study functional changes in cardiac function under conditions which have never been utilized before, but also to substantiate the findings of cardiovascular adjustments to separate stress conditions of excessive heat and static work.

Systolic intervals have been shown to be good indicators of ventricular function and have also been found to correlate significantly with invasive methods for the study of cardiac dynamics (48). The majority of studies which have employed systolic time intervals for functional analyses of the left ventricle during bouts of isometric work have used heart disease or hypertensive patients as their subjects. The intent of course was to identify ventricular functional abnormalities resulting from excessive production of arterial pressure during isometric exercise. This non-invasive method of cardiac investigation has proven to be a useful

clinical tool and has become popular in view of the fact that the body is not subject to internal testing.

A consideration of the above stated findings supports the idea that if both a pressure load induced by sustained isometric handgrip and volume load induced by heat stress were to burden the heart simultaneously, significant interactions in cardiac responses would result. It is therefore possible to investigate the extent of these cardiovascular (CV) interactions imposed by heat and sustained work by observing the changes in systolic time intervals, heart rate, and blood pressure. The intent of this study is to show left ventricular cardiac adjustments to thermal stress, isometric exercise, which produces an arterial pressure load, and simultaneous exposure of thermal and isometric stress. It is hoped that a conclusion may be arrived at concerning the differences between myocardial dynamics and oxygen consumption during isometric stress in a neutral environment and isometric stress accompanied by greater than normal skin temperature. Such conclusions would answer the query of whether the heart is supporting a significantly greater functional stress during a simultaneous volume and pressure load as opposed to only a pressure load.

II. PURPOSE

The purpose of this study is to investigate the changes in CV dynamics as depicted by systolic time intervals, BP, and HR during sustained submaximal and maximal isometric work in a hot and neutral environment.

III. SUBPROBLEMS

1. To compare changes in STI at rest between a neutral environment and a hot environment.
2. To compare changes in STI between rest, 50% MVC, and 100% MVC in two separate environments (heat and neutral).
3. To investigate how BP and HR change during the above stated interventions in an attempt to support previous studies.
4. To show any significant differences in cardiac dynamics as depicted by changes in STI, HR, and BP during the above stated environmental conditions between a submaximal static contraction and a maximal static contraction.
5. To show whether the CV changes induced by static contractions are additive or merely interactive with those elicited by thermal stress upon the body.

IV. ASSUMPTIONS AND LIMITATIONS

1. Invasive measurements concerned with changes in LVEDP were not taken. This limits the extent to which conclusions can be made from alterations in systolic time intervals during a static contraction. However the literature clearly states that the LVEDP in a normal intact heart does not significantly change during an increased pressure load.
2. It was assumed that all subjects produced a maximum effort when asked to grip the hand dynamometer for an initial MVC reading.

3. When using systolic time intervals the electromechanical lag (EML) is measured as the interval Q-S_1. This interval very closely approximates the period from the onset of electrical stimulation of the left ventricle to the beginning of ventricular contraction. However the Q-S_1 interval is slightly longer than the true EML since the initiation of the first heart sound (S_1) is associated with the closing of the atrioventricular (AV) valves rather than the actual commencement of myocardial contraction. In order to investigate the true changes in EML an apexcardiogram should have been used. However it was not available for this study.

4. The most appropriate method of statistically analyzing the data would have been to use a multivariate analysis of variance (MANOVA). However the number of subjects required in order to use this method were far too great. The statistical analysis was therefore confined to utilizing the standard analysis of variance (ANOVA).

V. HYPOTHESES

1. Oxygen consumption of the myocardium as reflected by the index (HR x BPs x LVET) significantly increases from rest to 100% MVC in both a heated and neutral environment.

2. Oxygen consumption of the myocardium as reflected by TRIP during a resting state is significantly greater in the hot environment than in the neutral.

3. Oxygen consumption of the myocardium as reflected by TRIP is significantly greater when the body is subjected simultaneously to heat and static muscular work than when the body is subjected to static muscular contraction alone.

4. The level of BPs and BPd is not significantly altered during rest, 50% MVC, and 100% MVC from a neutral to a heated environment.

5. BPs and BPd significantly increase from resting values during a 50% MVC and 100% MVC isometric contraction.

6. Changes in BPs, BPd, HR, variables representing ventricular systole (LVET, MS, TS and ETI), variables representing diastole (diastole and CT), variables representing the contractile state of the myocardium (ICP, PEP, and PEP/LVET), and EML change in an interactive fashion from rest to 100% MVC during simultaneous stress of heat and static work. Specifically the slopes between each workload condition are significantly different in the two environments resulting in a significant interaction for all variables.

7. All variables depicting changes in left ventricular systole (LVET; MS; TS) decrease from resting values during a 50% MVC and 100% MVC isometric contraction.

8. ETI significantly increases from a resting value during a 50% MVC and 100% MVC isometric contraction.

9. Diastole and CT significantly decrease while HR increases from resting values during a 50% MVC and a 100% MVC isometric contraction.

10. EML significantly decreases while HR increases from resting values during a 50% MVC and a 100% MVC isometric contraction.

11. The contractile state of the myocardium as depicted by changes in ICP, PEP, and PEP/LVET increases with isometric exercise.

VI. DEFINITIONS

1. Inotropic State of Myocardium

Inotropes are agents which change the contractility of the heart. Negative inotropes weaken the strength of contraction while positive inotropes augment the power of contraction. An increase in the inotropic state of the heart means that the myocardium is affected by positive inotropes such as digitalis and catecholamines which strengthen the contraction of the heart.

2. Pressure Load

A pressure load upon the CV system and the heart pump in particular is due to an increase in arterial pressure.

3. Volume Load

A volume load is associated with an increased CO and HR. It simply means that the heart has to pump more blood per unit time than normal.

4. Contractile State of Myocardium

The contractile state of the myocardium may be associated with its inotropic state since positive inotropes augment myocardial contractility. By definition contractility is the power of contraction where "Power" is equal to load x velocity of fiber shortening:

$$P = L \times V$$

Therefore an increase in contractility may be due to either an increased load or strength of contraction and/or an increased speed of contraction.

5. MVC

A maximal voluntary contraction is a 100% isometric squeeze on the hand dynamometer.

6. Systolic Time Intervals

The STI are time components of a single cardiac cycle. It is customary to divide the cardiac cycle into the following phases:

a) Cycle Time (CT) - time for one complete beat of the heart; measured as the interval S_2-S_2.

b) Diastole (D) - relaxation phase of the cycle time; time from end of ejection to onset of excitation measured as S_2-Q.

c) Electromechanical Lag (EML) - time from onset of electrical activity to the onset of mechanical activity; measured as Q-S_1.

d) Isovolumetric Contraction Period (ICP) - time from onset of contraction to onset of ejection; measured as S_1-S_2 minus C_1-C_2.

e) Left Ventricular Ejection Period (LVET) - time from the onset to the end of ejection; measured by C_1-C_2.

f) Mechanical Systole (MS) - time from the onset of contraction to end of ejection; measured by S_1-S_2.

g) Total Systole (TS) - from onset of excitation to end of ejection; measured as Q-S_2.

h) Pre-ejection Period (PEP) - from onset of excitation to onset of ejection; measured by EML + ICP.

i) PEP/LVET - ratio of the amount of time spent in preparation for ejection to the amount of time actually spent in ejecting blood from the left ventricle.

7. Triple Product (TRIP)

An index of myocardial oxygen consumption derived from the product of BP (systolic), heart rate, and LVET.

$$TRIP = BPs \times HR \times LVET$$

8. Ejection Time Index (ETI)

The ETI is LVET corrected for the effects of HR.

$$ETI = LVET + 1.7\ HR$$

9. Vmax

Maximum velocity of myofibril contraction at zero load.

10. dp/dt

The first derivative of ventricular pressure denoting the change in pressure with time.

11. Contractile Element Velocity (VCE)

Speed of contraction of the myofibril being derived at specified pressure loads.

12. Pressure Rate Product (PRP)

A second index of myocardial oxygen consumption derived as:

$$PRP = BPs \times HR$$

VII. SIGNIFICANCE OF THE STUDY

This study will lead to a better understanding of the changes in cardiac dynamics involved in a hot environment during both rest and sustained static work. In addition it will demonstrate how the heart reacts to loads of volume and pressure simultaneously. This may shed some light upon the understanding of cardiac fiber functional limitations.

CHAPTER II

REVIEW OF THE LITERATURE

I. HISTORICAL INTRODUCTION

The first comprehensive major study of the consecutive phases of the cardiac cycle was done by Wiggers (86, 87) in 1921. He utilized pressure curves from the cardiac chambers and arteries of dogs to record the changes in the ventricular cycles. In 1923 Katz and Feil (31) modified the procedures used by Wiggers. They recorded the systolic time intervals from simultaneous tracings of heart sounds, the subclavian arterial pulse and the electrocardiogram (Lead II). This contemporary procedure allowed for the investigation of cardiac function in the intact heart of humans without entering the body. It therefore became advantageous to use such non-invasive methods of investigation which allowed researchers such as Lombarde and Cope (43) in 1927 to show distinct differences in the systolic intervals between the sexes.

Further work in this area was limited in the years to come due to the renaissance of investigation into the quantitative measures of the isolated heart preparation. However, with advancement of technology the interest in systolic time intervals has been revived. Within the last two decades a multitude of studies have been completed most of which focus their efforts on the dynamic changes of the diseased heart. With the recent concern about heart disease the information available from investigations of cardiac intervals has been found to be useful in the diagnosis of myocardial malfunction.

II. CARDIAC INTERVALS

The changes in systolic intervals are dependent on a variety of physiological parameters some of which have been identified and others which are unknown. Franks et al. (18) combined data from several studies to determine the orthogonal (independent) factors of the left ventricular time components. Four factors accounted for virtually all of the variance of the eight intervals studied. Factor I represents primarily left ventricular systole, with high loadings for LVET, MS, and TS. Factor II represents ventricular diastole with high loadings for diastole and CT. Factor III represents the level of ventricular sympathoadrenergic activity which is closely associated with the contractile state of the myocardial tissue. This factor has high loadings for ICP and PEP. Factor IV is specifically associated with the electrical activity of the left ventricle represented by the time from the onset of excitation to the onset of contraction as measured by EML.

A. LEFT VENTRICULAR SYSTOLE

The most studied fraction of ventricular systole is LVET which has been reported by a number of authors to vary inversely with HR and directly with CT and SV (15, 36). Braunwald et al. (5) studied the independent factors influencing the duration of LVET in isolated heart preparations. They showed that an increase in SV alone lengthens LVET and an increase in HR alone at any given stroke volume shortens the duration of ejection per beat but prolongs the duration of ejection per minute. When mean aortic pressure was elevated at any given stroke volume and heart rate, no

significant change in LVET was evident until the mean aortic pressure reached 175-200 mm Hg. at which time a lengthening of LVET occurred. Administration of sympathomimetic amines, at any given SV, caused LVET to shorten. Similar results were obtained by Wallace et al. (80) when studying the effects of altering separately, SV, mean BP (BPm) and HR on the duration of LVET and ICP in denervated dog heart preparations. Augmenting stroke volume was found to prolong LVET and shorten ICP and had an insignificant effect on TS. Elevating BPm to 140-160 mm Hg. shortened LVET, prolonged ICP and had little effect on TS. These results are in opposition to Braunwald's findings which suggest that increasing BPm below 175 mm Hg. causes little change in LVET. Wallace also showed that increasing heart rate alone or administering digitalis and norepinephrine shortened all phases of systole including LVET and TS.

These findings clearly demonstrate that the influence of hemodynamic variables on the duration of certain phases of ventricular systole interact with each other such that it is not possible to change one variable without altering others. However a systematic analysis of the effects of changing selected hemodynamic variables on the duration of each phase of systole may help to decipher the changes in systolic time intervals of the intact heart. We should always keep in mind that by means of intrinsic mechanisms the myocardial tissue exhibits a remarkable capability to adjust the duration of each phase of systole to cope with the changing hemodynamic conditions.

Lindquist et al. (42) studied the effect of a 50% MVC isometric contraction of the forearm for one and one half minutes in 21 normal subjects and showed that LVET decreased significantly with the contraction.

However when LVET was corrected for HR using a regression equation there was a smaller insignificant decrease in LVET. Frank and Haberen (15) also showed that during a static contraction of the forearm lasting four minutes at 30% MVC the LVET shortened significantly. When LVET was corrected for HR using Weissler's (82) regression equation there was no significant change in ΔLVET. Quarry and Spodick (65) produced similar results in normal males during an IHG contraction in a sitting position. It was found that at 50% MVC LVET was reduced significantly during the first thirty seconds of contraction but insignificantly during the last thirty seconds of a one minute contraction. The greatest decrease in LVET was shown to be during a thirty second contraction at 100% MVC.

It seems as if the changes in LVET associated with a static contraction are probably more closely related to changes in HR rather than changes in BPm both of which are elevated during static work. This suggests that the proper inducing factor in the reduction of LVET during static muscular contraction is the development of tachycardia caused by neural and humoral stimulation of the cardiac tissue.

EJECTION TIME INDEX (ETI)

It is well known that in normal individuals the LVET varies inversely with heart rate. For this reason some researchers attempted to assess other determinants of the duration of LVET by diluting the effect of HR upon the LVET. An index of the relationship between HR and LVET was subsequently derived and was termed the ejection time index (ETI). Weissler et al. (81) showed that the ETI was significantly correlated with cardiac

output in normal subjects and in patients with congestive heart failure. It was found that a fall in cardiac output was reflected in a decrease in the ETI and concluded that the ETI offers a useful semiquantitative measure of the level of the cardiac output in normal individuals and in patients with congestive heart failure.

Quarry and Spodick (65) found that the ETI significantly increased ($p < 0.01$) at 30% MVC from control through four minutes of an isometric hand grip. The ETI was found to increase most during a 50% MVC of the forearm for one minute. However only a small insignificant increase in ETI was found during a 100% MVC of the forearm for thirty seconds. The authors state that although efforts were made to avoid Valsalva maneuvers throughout IHG, some subjects may have performed this maneuver during 100% MVC contraction. Since the Valsalva maneuver has been shown to increase HR but decrease LVET and ETI (16) they suggest that the combined effects of this strain pattern could account for the lower ETI at 100% MVC.

B. VENTRICULAR DIASTOLE

Changes in diastole per se have not been paid much attention in the literature for the simple reason that alterations in diastole or CT are very closely related to changes in HR. Therefore results pertaining to diastole are used in support of other findings within the cardiac cycle and do not provide additional or novel information about cardiac dynamics.

In one of the few studies which has actually presented data for CT and diastole Coutts (8) showed that both variables shortened significantly with an isometric hand grip of 75% MVC. The importance of diastole seems

obvious since it includes time for ventricular rest, filling, and coronary circulation. The shorter the period of time in which the myocardium has to accomplish these tasks, as occurs during tachycardia, the shorter diastole and CT will be.

C. SYMPATHOADRENERGIC ACTIVITY

There is common agreement within the recent literature which acknowledges the fact that PEP, ICP and possibly PEP/LVET are good indicators of myocardial contractility and the sympathoadrenergic tone of the myocardium (1, 17, 54, 83). However there is no conclusive evidence to suggest that any one of these variables is a better indicator of the contractile state of the heart than another. In most cases all three variables are used simultaneously for purposes of investigation.

Lindquist et al. (42) found that PEP and ICP decreased significantly by the end of a bout of dynamic exercise but did not change during a 50% MVC for one and one half minutes. PEP/LVET did not change with a static work bout but decreased significantly during ergometry work. In addition attempts were undertaken to determine whether there were any significant relationships between the STI and HR, BPs and BPd. No significant regressions were found between ICT and HR or any of the STI and blood pressure during isometric exercise.

Ahmed et al. (3) showed that an increase in the inotropic stimulus to the heart either in response to dynamic exercise or isoproteronol infusions would cause an increase in the contractile element velocity (VCE) of the myocardium but reduce the PEP/LVET ratio.

Garrard et al. (19) found a high inverse correlation between PEP/LVET and Ejection Fraction (EF) r = -.90 (EF = SV/LVEDV). These authors suggest that a decrease in EF measures diminished contractile power of the heart. Therefore an increase in PEP/LVET may be due to poor myocardial contractility.

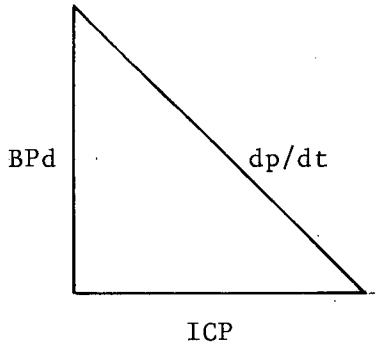
Grossman et al. (21) showed that in normals a 50% MVC for three minutes caused a decrease in PEP/LVET. However in heart disease patients with poor cardiac reserves the ratio did not significantly decrease. In normals the decrease in PEP/LVET was paralleled by an increase in stroke work without change in LVEDP, while in the patients LVEDP rose significantly and stroke work did not, reflecting a wide variation in individual responses. In addition, both Vmax and left ventricular dp/dt showed significant increases during IHG in normals. In patients increases in Vmax and dp/dt were also evident, although the resting and peak values were of smaller magnitude than in the normal group. These results indicate that the heart reacts to an afterload by contracting with more power without changes in LVEDP (preload). This change is associated with no increase in the PEP/LVET ratio. However in the diseased heart an increase in afterload if severe enough may cause changes in preload as depicted by increases in LVEDP. In such cases where the diseased myocardial tissue is not able to cope with the afterload the PEP/LVET will rise.

Martin et al. (48) studied six patients with normal coronary angiograms using both STI and catheter invasive measurements during a variety of interventions one of which was a 100% MVC isometric contraction of the forearm. They showed that STI correlated well with invasive measurements

but most importantly the data demonstrated an inverse relationship between PEP and ICP to dp/dt. Metzger et al. (54) found exactly the same relationship with anesthetized dogs. In this study an attempt was made to find the relation of true isovolumetric contraction time, as measured by invasive techniques, with two external indexes of ventricular performance, namely ICP and PEP. Excellent linear correlations were found between absolute values of true isovolumetric contraction time and both ICP and PEP. As was mentioned above the inverse relationship between true isovolumetric contraction time and dp/dt was also accurately reflected by the PEP and ICP. The authors suggest that ICP and PEP reflect reliable changes in true isovolumetric contraction time.

In view of the fact that ICP and dp/dt are associated with alteration in afterload (BPd) and preload (LVEDP) Metzger illustrates the relationship between these variables by the use of a right triangle. He states that at constant LVEDP and constant dp/dt, ICP is determined by changes in afterload. Conversely, at a constant LVEDP and BPd, ICP is inversely related to the maximal dp/dt. This concept is illustrated in Figure 1.

Figure 1.



BPd: ICP: dp/dt

In summary it is concluded that when changes in BPd are considered, with only small alterations in LVEDP, PEP and ICP give useful information about changes in ventricular performance including an estimation of changes in left ventricular dp/dt and contractility.

The right triangle relationship is supported by the findings of Matsura and Goodyer (51) who studied the effects of a pressure load on left ventricular systolic time intervals in 22 open-chested anesthetized dogs. Under normal control conditions and with infusions of isoproterenol an increase in aortic pressure reduced ICP but increased LVEDP. However propranolol infusions, which act to decrease the contractile power of the myofibrils, caused ICP to be prolonged and LVET to decrease as the afterload was increased. These results suggest that an afterload on a healthy heart will induce an increase in the power of contraction of the heart which is associated with increased dp/dt with little change or a slight reduction in ICP.

Quarry and Spodick (65) conducted an investigation comparing STI during a variety of static work loads. They showed that ICP and PEP would significantly decrease during the first thirty seconds succeeded by a rapid reascent to slightly below control levels during the last thirty seconds of a one minute 50% MVC contraction. However both ICP and PEP decreased significantly at 100% MVC. The PEP/LVET showed no change during the 50% MVC and a slight insignificant decrease during a thirty second 100% MVC.

Stefadouros et al. (75) used STI to show changes in ventricular performance in normal males during a 50% MVC for three minutes. These authors found that during the static contraction left ventricular dimensions, EF, PEP/LVET and shortening velocity did not significantly change. These

results were explained by an increase in the inotropic state of the heart which was elevated to increase the contractile state of the heart in response to the augmented afterload caused by the IHG. In addition these authors also found a strong negative correlation between the PEP/LVET ratio and EF at rest ($r = -.879$) and during static work ($r = -.775$) which is in agreement with the findings of Garrard.

Talley et al. (78) evaluated the PEP as an estimate of myocardial contractility by both invasive and non-invasive measurements of STI in dogs under a variety of experimental conditions. No effect of HR on the PEP was noted when LVEDP, BPd, and contractile indexes were held constant. Increases in aortic diastolic pressure alone caused significant but small increases in PEP. Increases in LVEDP were shown to significantly alter the PEP in an inverse fashion. There was, however, a significant correlation between the PEP and the internal indexes of contractility (dp/dt and maximal dp/dt) over a wide range of inotropic stimuli in experiments in which LVEDP was shown to vary in a random manner. According to the authors these studies indicate that PEP may be used as an index of myocardial state in situations in which LVEDP does not vary systematically under the conditions being studied.

In summary it seems as if these sympathoadrenergic associated intervals are affected by not only the inotropic state of the myocardium but also by changes in LVEDP (preload) and BPd (afterload). In a normal heart, catheterization studies have shown that despite considerable increase in LV stroke work during IHG exercise, no change in LVEDP is evident (21, 25, 58, 75). Therefore it requires little thought to conceive that when the

afterload to the heart is increased the heart tissue must contract with more vigor in order to expel the same amount of blood against a greater pressure. For this reason the change in ICP tells us something about the amount of time which the left ventricle spends in a phase of isometric contraction, whereby the length of the fibers does not change, during which time the inner ventricular tension rises. This rate of tension development (dp/dt) depends on the power of contraction or simply the contractile state of the myocardium (74). If ICP increases with an augmented afterload, as is produced with IHG exercise, this suggests that the contractility of the heart is not enough to produce a large enough dp/dt against the pressure load such that it spends longer time in increasing the intraventricular pressure to opening of the aortic valve.

D. ELECTRICAL ACTIVITY WITHIN THE LEFT VENTRICLE

Following the spread of electrical activity, mechanical activity starts in the heart after a variable interval which is referred to as EML. There is very little information available in the literature pertaining to alterations of the EML during bouts of sustained isometric exercise. A possible reason for this absence in the literature could be that EML is most frequently coupled with ICP to show changes in PEP. However Franks et al. (18) strongly suggest that EML and ICP should be analyzed separately because they represent quite different aspects of the cardiac cycle.

Quarry and Spodick (65) used the Q-Im interval which is very similar to the EML and showed that Q-Im decreased significantly with a one minute 50% MVC contraction and a thirty second 100% MVC contraction. Coutts (8)

however found that EML was not significantly altered during a ten second 75% MVC in the supine position. Perhaps ten seconds of contraction was not enough time for the body to produce complete physiological adaptation to the muscular stress such that alterations in the electrical activity of the left ventricle were insignificant or completely absent. Kumar and Spodick (36) in an excellent review of the mechanical events of the left ventricle also establish the fact that measurements of EML are rare. The only studies which they cite are concerned with changes in the Q-Im interval in pathophysiological states.

III. MYOCARDIAL OXYGEN CONSUMPTION

The most successful non-invasive method of measuring myocardial oxygen consumption has been to use the product of LVET, BPs and HR. Katz and Feinberg (32) reviewed the relations between cardiac work and its oxygen consumption and concluded that an increase in HR and/or the buildup and maintenance of tension in the ventricles causes the heart to consume most of its available oxygen (O_2) supply.

Kivowitz et al. (35) had 22 CHD patients perform an IHG exercise at 25% MVC for five minutes. They found an increase in coronary blood flow and myocardial oxygen consumption of 45% from resting values. This indicates that an IHG exercise is severe enough to tax the work output of the heart. However no evidence of myocardial lactate production was present during the bout of static work which suggests that the coronary blood flow increase during the IHG exercise was substantial to supply the myocardium with the needed oxygen.

Similar findings were reported by Nakhjavan et al. (59) who studied lactate metabolism of the heart during IHG exercise and pace produced tachycardia. Tension time index (BPs x LVET) and the triple product were used to measure myocardial O_2 consumption. The results showed that both measurements were greater during IHG at 30% MVC for five minutes than right atrial pacing. However the largest consumption of oxygen by the heart occurred during simultaneous atrial pacing and IHG exercise. These authors also found that myocardial lactate production was absent during IHG exercise but was increased during right atrial pacing.

Lindquist et al. (42) also used the triple product to assess myocardial oxygen consumption. These researchers compared the results of dynamic and static work in 22 normal subjects. They showed a larger triple product during an IHG exercise of 30% MVC for one and one half minutes than during dynamic work.

These results suggest that IHG exercise will increase the oxygen consumption of the myocardium but that this requirement is met by an increased coronary flow which is substantial to supply the needed oxygen to the working tissue.

IV. CIRCULATORY CHANGES WITH STATIC WORK

The circulatory adjustments to sustained static work have been well documented in the literature. The most marked changes associated with an IHG contraction of the forearm are acute increases in BPd, BPs and HR (38, 39, 49, 61). Both the blood pressure elevation and HR elevation are proportional to the relative force and duration of the contraction.

Mitchell and Wildenthal (56) note that neither the absolute tension developed nor the size of the muscle group is the determining factor in altering circulatory variables during isometric work. Lind et al. (40) have shown that BP rises to a similar extent with a 20% MVC of the forearm, a 20% MVC of the thigh muscles and a 20% MVC of individual fingers, despite manifold differences in absolute tension developed and in the energy requirements of the respective efforts.

According to Lind (37) the amount of blood flow restriction caused by isometric muscular contraction is the main factor which controls the length of time a contraction can be maintained. Lind et al. (40) have shown that blood flow through the arm is not completely occluded until the tension exceeds 70% MVC. For this reason a 50% MVC can be maintained for a maximum time of approximately two minutes while a 100% MVC can be maintained for about thirty seconds. A one minute contraction at 50% MVC would therefore induce a state of 50% absolute fatigue of the forearm while a 100% MVC for thirty seconds would produce complete fatigue of the forearm.

Another important variable which increases during an isometric contraction is the cardiac output. Lind (41) examined subjects in the supine position while they performed hand grip contraction at 10% MVC, 20% MVC and 50% MVC. It was confirmed that sustained hand grip contractions caused an increase in cardiac output which was a major factor in producing the increased mean blood pressure. It was also found that in the younger subjects (mean age 26) there was no change in systemic vascular resistance (SVR) while in one older subject aged 37 there was significant increase in

the SVR with hand grip exercise. In addition the cardiac stroke volume showed no systemic change at 10% MVC or 20% MVC but a consistent and clear reduction in all cases at a tension of 50% MVC, when cardio-acceleration was pronounced. This suggests that in the younger subjects the major cause of increased BP during isometric work was the augmentation of CO due directly to the cardio-acceleration since increases in SV and SVR have been shown to be absent. Mitchell and Wildenthal (56) however conclude that the increased CO present with static work results primarily from tachycardia but may also be caused in part by an increased stroke volume in some individuals.

When isometric exercise is superimposed upon dynamic exercise, the cardiovascular effects tend to be additive. Nutter et al. (61) describes that when sustained hand grip contraction is performed by subjects walking on a treadmill with an oxygen consumption of 1.1 L/minute, the increase in BP is the same as that produced by an equivalent hand grip alone despite the marked lowering of systemic vascular resistance produced by the dynamic exercise. However, during an extreme combination of walking at an oxygen consumption of 2.2 L/minute and 50% MVC the pressor response is less than that produced during less strenuous walking. It seems that the more vigorous the dynamic phase of the exercise the more extreme will be the muscular vasodilation. In such a condition the pressor response to the isometric exercise will be less pronounced.

V. CV CHANGES IN A HOT ENVIRONMENT

This subject has very recently been extensively reviewed by Rowell (69) who himself has conducted a number of classic studies. It is well known that heat exchange between the body and the environment is determined by the amount of heat brought to or from the skin by the vascular tubing. The overall cardiovascular response to direct whole body heating is a rise in CO in proportion to the rise in HR. The SV increases very little (4%-5%) from normal values and the BPm similarly falls slightly. The marked rise in total limb blood flow measured plethysmographically during indirect heating has been shown to be confined to the skin rather than the muscle mass. Rowell concludes that muscle blood flow in the leg or forearm, which is not directly heated, decreases or remains constant with whole body heating.

Two separate studies were conducted by Johnson et al. (29) and Heistad et al. (24) who researched the interaction between thermal cutaneous vasodilator and pressoreceptor reflexes in the forearm. Johnson used seven normal subjects aged 22-30 years to determine whether skin will respond to increased neurogenic vasoconstrictor activity during direct heating. All testing was performed in a supine resting condition. The results showed that sympathetic vasoconstriction is unable to overcome the high levels of vasodilation accompanying moderate levels of body heating. Heistad concluded similarly when he found that in the forearm the vasoconstriction response to lower body negative pressure was less during body heating. Since it is known that isometric exercise will

increase the amount of catecholamines in the circulation (60) it may be argued that a static contraction of the forearm would increase the pressor response of the skin. However in a hot environment although the skin retains some ability to vasoconstrict, this reflex mechanism may not override the heat-induced vasodilation.

CHAPTER III

METHODS AND PROCEDURES

I. SUBJECTS

Fourteen male subjects from the University of British Columbia aged 20 to 31 (mean age = 25.4) volunteered to be subjects in this experiment. Prior to being accepted into the study each individual was asked about their health records and if they were undertaking or had recently completed any kind of physical training programme. Only those people who had no previous health problems and who were not training or recently completed a training programme within the last six months were accepted into the study.

The subjects were all physical education students and maintained a similar activity level throughout the day. Their height and weight were recorded and compared to standard tables. None of the subjects were found to be grossly overweight or obese.

II. EXPERIMENTAL PERIOD

Each subject was tested on two separate days with one day of rest between the two testing days. The testing procedure was exactly the same in both a hot environment and neutral (room) environment. A resting measure of all variables was taken to ensure that the equipment was responsive and that the readings were clear enough for analysis. This initial period of adjustment not only permitted the access of the best possible readings for that subject but also gave the subject a chance to get acquainted with the equipment and testing procedures.

After this session the subjects were put through the entire testing procedure. Between each work load the subject was given five minutes to recover after which time his pulse was taken to ensure that the heart rate had dropped back to a resting level.

When testing in the heat initial subjection to heat was gradual, lasting from ten to fifteen minutes, until all of the equipment had been adjusted for the subject and clear recordings were attained. During this time, sauna temperature and subsequently the skin temperature rose gradually. Data collection did not begin until the skin temperature had reached 40°C at which time the thermostat for the sauna was dropped in order to maintain the skin temperature between 40°C - 41°C throughout the testing period.

III. PRE-EXPERIMENTAL INSTRUCTIONS

Subjects were asked to maintain their normal activity and rest patterns throughout their three experimental days. Testing was not done in the post absorptive state; however subjects were asked not to eat at least one hour before coming into the laboratory. All of the subjects except two did not eat for at least two hours before being tested. The two subjects who ate between one and two hours before the testing had eaten a very light lunch because they had not eaten any breakfast.

During that initial period when the subjects were getting acquainted with the testing procedures specific instructions were given to each subject:

a) Try to relax the entire body

b) Keep breathing normally and try to eliminate any sudden jerking of the musculature

c) When contracting the forearm isolate the contraction such that no other muscle in your body is facilitating the work of the forearm.

IV. PROCEDURES

The complete testing scheme for each subject is illustrated below in Figure 2. Each volunteer was subjected to three levels of contraction in two different environments totalling six different conditions.

Figure 2.

	Neutral	Heat
Rest	N_1	H_1
50% MVC	N_2	H_2
100% MVC	N_3	H_3

Testing Scheme

The subject was seated in a back reclining position of 110°. The left arm lay on a padded arm rest which was inclined slightly towards the floor in order to facilitate the taking of blood pressure. The static contractions were performed by the right forearm using a hand dynamometer which was electrically connected to an oscilloscope and gave a continuous visible recording to the subject and experimenter of the degree of contraction. Prior to commencement of any testing the maximal voluntary contraction (MVC) for each subject was identified from which both maximal (100% MVC) and submaximal (50% MVC) degrees of contraction were calculated and converted into units on the oscilloscope. The subjects were asked to contract the forearm at 100% MVC for one half minute and at 50% MVC for one minute.

All subjects were tested in a neutral environment and a heated environment. The neutral setting as existed in the physiotherapy room at the University of British Columbia did vary somewhat in both temperature and humidity from day to day. In the physiotherapy room, the temperature was seen to fluctuate between 24°C and 26°C throughout the two-week experiment while the humidity fluctuated between 51% and 58% saturation.

The heated environment was created in the sauna which was also situated in the physiotherapy room at the University of British Columbia. The humidity in the sauna was not regulated but was left to vary in accordance with the natural humidity in the air on any one day. Such a procedure was used because it was found during a pilot investigation that increasing the humidity to 100% saturation caused the subject to

perspire profusely which caused poor contact of the equipment to the skin and discomfort to the subject. In view of the fact that the guiding factor for commencement of data collection was the skin temperature it was decided not to increase the humidity in the sauna but rather induce a gradual change in the skin temperature by regulating the air temperature of the sauna only.

During each of the six experimental conditions a number of simultaneous tests were taken on each subject.

Skin Temperature

Skin temperature readings were taken continuously throughout the testing session in the sauna. A thermister was attached to the skin surface on the right side of the body two inches beneath the ribcage. The thermister was checked and calibrated every morning and afternoon before testing of subjects in the sauna.

Blood Pressure

Blood pressure was taken with a cuff sphygmomanometer whose input was electrically transferred to one channel in the Sanborn recorder. This system gave a beat to beat graphic recording from which both systolic and diastolic pressures could be read. The sphygmomanometer was calibrated for each experimental condition before each of the work bouts. The pressure recording was taken during the last five seconds of contraction for each work load.

Heart Sounds

A phonocardiogram (PCG) was used to detect the first and second heart sounds. The microphone head of the PCG apparatus was situated on the skin

surface within the fourth or fifth intercostal space, adjacent to the sternum on the left side of the body, depending on which position gave the stronger signal. The PCG was connected to a second channel of the Sanborn recorder.

Carotid Pulse Wave

Carotid pulse waves were registered from a pressure sensitive instrument strapped to the skin on the top of the carotid artery on the left side of the neck. Recordings were found to be strongest when the head was tilted to the right allowing the carotid artery on the left side of the neck to be uplifted closer to the skin surface. The subject was asked not to swallow during the five second period in which the simultaneous recordings were taken. The beat to beat recordings were also electrically transferred to one of the four active channels on the Sanborn recorder which produced graph recordings of the pulse waves.

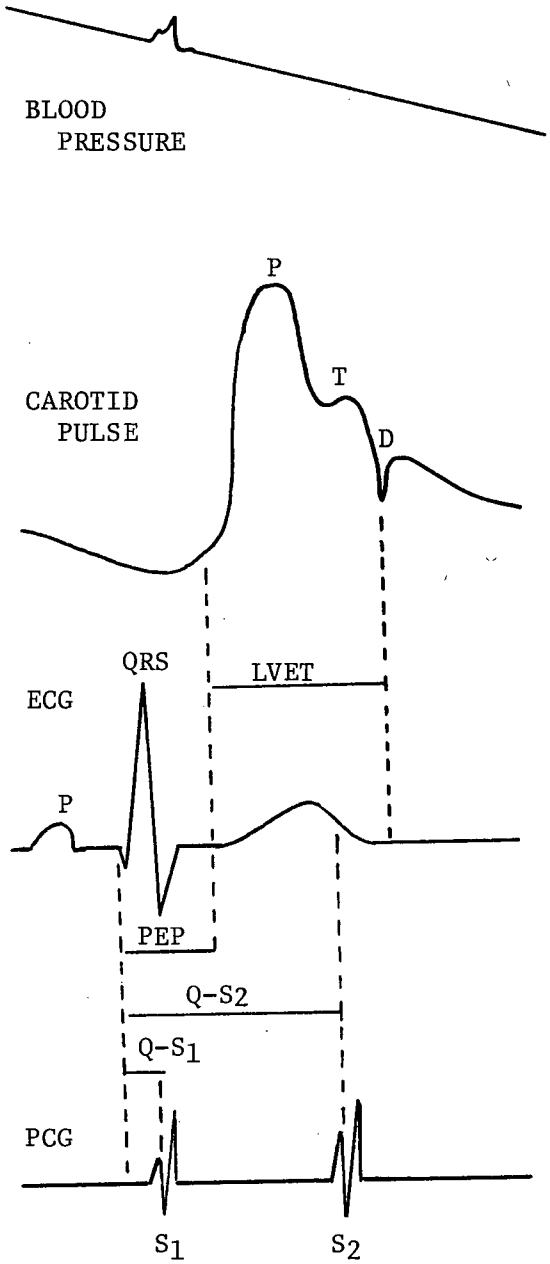
Electrocardiogram

A CM_5 lead was used for the electrocardiogram (ECG) which was taken simultaneously on the fourth channel of the Sanborn recorder along with BP, PCG, and CPW. Precautions were taken to eliminate the Valsalva manoeuver by explaining the importance of maintaining normal breathing during contraction of the forearm to each subject.

An analysis of the simultaneous recordings of ECG, PCG, and CPW permitted the calculation of the systolic time intervals in milliseconds. A graphic representation of the systolic time intervals is shown in Figure 3. A recording of these intervals was taken only during the last five seconds

Figure 3.

Systolic Time Intervals



Systolic Time Intervals measured from simultaneous recordings of electrocardiogram, phonocardiogram, and carotid pulse wave. Also blood pressure recording electrically transmitted from cuff sphygmomanometer. LVET, left ventricular ejection time; $Q\text{-}S_1$, preisovolumetric contraction period; PEP, preejection period.

of each contraction period. From these only the three strongest and clearest cycle tracings closest to the end of the contraction period were used for analysis.

All subjects were tested under six different conditions. One half of the subjects were tested first in the sauna and secondly in the neutral environment while the other half did the reverse. The order by which the work load was administered was randomly assigned by picking the assorted loads from a hat. The work loads were administered in the same order in both environmental conditions for each subject.

From the initial fourteen recordings only twelve of these which gave the sharpest tracings were used in the computer analysis. The two poorest tracings were discarded prior to computer analysis of the raw data. This guaranteed that the data were of good quality and decreased the chances of faulting the experiment by production of poor quality tracings in a few subjects.

V. STATISTICAL CONCERNS

As was mentioned above only the last five seconds of each test condition were recorded on the Sanborn. This period was ample to assure that the effects of the test condition were maximally represented on the recording and also allowed enough time to record the arterial pressure. Of this five-second tracing only the last three clearest cardiac cycles were used in the computer analysis. From these three cycles a mean was computed for six systolic intervals, BPs and BPd. These means were

then transformed by the computer resulting in fourteen dependent variables. The names of the dependent and independent variables are as follows:

Independent variables

A. Environment (2 levels)

A_1 Heat

A_2 Neutral

B. Workload (3 levels)

B_1 Rest

B_2 50% MVC

B_3 100% MVC

The fourteen dependent variables can be broken down into separate categories:

a) Blood Pressure

BPs and BPd

b) Heart Rate

c) Ventricular Systole

LVET, TS, MS, and ETI

d) Ventricular Diastole

Diastole and CT

e) Sympathoadrenergic activity

ICP, PEP, and PEP/LVET

f) Electromechanical Lag (EML)

g) Myocardial Oxygen Consumption

TRIP

The test design is illustrated below in Figure 4. Test Design.

		Heat	Neutral
Rest	V_1	$\overline{X}_1$	$\overline{X}_1$
	V_2	$\overline{X}_2$	$\overline{X}_2$
	V_3	$\overline{X}_3$	$\overline{X}_3$
	V_4	$\overline{X}_4$	$\overline{X}_4$
	V_5	$\overline{X}_5$	$\overline{X}_5$
	V_6	$\overline{X}_6$	$\overline{X}_6$
	V_7	$\overline{X}_7$	$\overline{X}_7$
	V_8	$\overline{X}_8$	$\overline{X}_8$
	V_9	$\overline{X}_9$	$\overline{X}_9$
	V_{10}	$\overline{X}_{10}$	$\overline{X}_{10}$
	V_{11}	$\overline{X}_{11}$	$\overline{X}_{11}$
	V_{12}	$\overline{X}_{12}$	$\overline{X}_{12}$
	V_{13}	$\overline{X}_{13}$	$\overline{X}_{13}$
	V_{14}	$\overline{X}_{14}$	$\overline{X}_{14}$
50% MVC	V_1	$\overline{X}_1$	$\overline{X}_1$
	V_2	$\overline{X}_2$	$\overline{X}_2$
	V_3	$\overline{X}_3$	$\overline{X}_3$
	V_4	$\overline{X}_4$	$\overline{X}_4$
	V_5	$\overline{X}_5$	$\overline{X}_5$
	V_6	$\overline{X}_6$	$\overline{X}_6$
	V_7	$\overline{X}_7$	$\overline{X}_7$
	V_8	$\overline{X}_8$	$\overline{X}_8$
	V_9	$\overline{X}_9$	$\overline{X}_9$
	V_{10}	$\overline{X}_{10}$	$\overline{X}_{10}$
	V_{11}	$\overline{X}_{11}$	$\overline{X}_{11}$
	V_{12}	$\overline{X}_{12}$	$\overline{X}_{12}$
	V_{13}	$\overline{X}_{13}$	$\overline{X}_{13}$
	V_{14}	$\overline{X}_{14}$	$\overline{X}_{14}$
100% MVC	V_1	$\overline{X}_1$	$\overline{X}_1$
	V_2	$\overline{X}_2$	$\overline{X}_2$
	V_3	$\overline{X}_3$	$\overline{X}_3$
	V_4	$\overline{X}_4$	$\overline{X}_4$
	V_5	$\overline{X}_5$	$\overline{X}_5$
	V_6	$\overline{X}_6$	$\overline{X}_6$
	V_7	$\overline{X}_7$	$\overline{X}_7$
	V_8	$\overline{X}_8$	$\overline{X}_8$
	V_9	$\overline{X}_9$	$\overline{X}_9$
	V_{10}	$\overline{X}_{10}$	$\overline{X}_{10}$
	V_{11}	$\overline{X}_{11}$	$\overline{X}_{11}$
	V_{12}	$\overline{X}_{12}$	$\overline{X}_{12}$
	V_{13}	$\overline{X}_{13}$	$\overline{X}_{13}$
	V_{14}	$\overline{X}_{14}$	$\overline{X}_{14}$

Figure 4.

The experimental testing for this study consists of a 2x3 factorial design with repeated measures on both variables as illustrated in Figure 5.

	Heat			Neutral		
	R	50	100	R	50	100
S_1						
S_2						
S_3						
S_4						
S_5						
S_6						
S_7						
S_8						
S_9						
S_{10}						
S_{11}						
S_{12}						

Figure 5.

2 x 3 Factorial Design

The data were treated with a two-way ANOVA for each dependent variable. Also correlations among all dependent variables were calculated.

CHAPTER IV

RESULTS AND DISCUSSION

I. RESULTS

The means and standard deviations of all dependent variables for each of the six conditions are given in Table 1.

TABLE 1

	Neutral			Heat		
	Rest	50% MVC	100% MVC	Rest	50% MVC	100% MVC
BPs	112 ± 9.4	139 ± 13.5	144 ± 14.0	106 ± 10.8	124 ± 15.4	132 ± 16.6
BPd	77 ± 4.2	106 ± 9.7	110 ± 12.0	72 ± 6.5	89 ± 13.9	96 ± 13.1
CT	961 ±134	769 ±157	663 ±103	778 ±104	684 ± 99	593 ± 78
EML	83 ± 10.4	78 ± 12.5	77 ± 10.0	82 ± 14.2	77 ± 10.5	75 ± 12.5
LVET	258 ± 18.2	251 ± 18.6	241 ± 16.7	239 ± 16.3	231 ± 15.5	220 ± 16.8
MS	303 ± 20.2	302 ± 16.8	281 ± 11.7	281 ± 18.2	275 ± 10.1	257 ± 16.0
TS	386 ± 21.1	379 ± 22.1	358 ± 16.7	364 ± 12.7	350 ± 16.1	333 ± 20.1
DIAST	574 ±120.5	394 ±136.9	305 ± 89.2	413 ± 96.2	335 ± 85.5	258 ± 60.2
ICP	44.9 ± 17.0	51.1 ± 9.0	39.8 ± 9.8	43.6 ± 12.9	42.5 ± 11.8	37.8 ± 14.5
PEP	128.3 ± 16.5	128.8 ± 10.4	116.8 ± 14.8	125.3 ± 8.9	119.4 ± 10.1	112.2 ± 11.2
PEP/LVET	.50 ± .085	.52 ± .057	.49 ± .084	.53 ± .067	.52 ± .065	.51 ± .066
HR	63.5 ± 9.1	80.5 ± 13.4	92.3 ± 12.8	78.5 ± 10.4	89.3 ± 12.8	102.7 ± 12.1
TRIP	1825 ±267	2813 ±570	3205 ±617	1964 ±165	2551 ±410	2969 ±476
ETI	366 ± 11.6	387 ± 19.0	398 ± 21.3	372 ± 10.3	383 ± 12.3	395 ± 11.5

Statistical Results

The results of the ANOVA for each variable are grouped according to the cardiovascular function they depict. Testing was conducted for a significant difference between the heat and neutral (control) environments averaged over the three workloads (rest to 100% MVC). The second test investigated the differences, averaged over environments, among the three workloads. The final test included the investigation of any interaction between workloads and environments on each of the dependent variables.

The sources for the ANOVA tables are termed as follows:

E - Environment Effect: Heat vs. Neutral
C - Contraction (Workload) Effect: Rest vs. 50% MVC vs. 100% MVC
C_1 - Workload (Linear change from Rest to 100% MVC)
C_2 - Workload (Quadratic change from Rest to 100% MVC)
EC - Interaction Effect: Environment x Workload

Note: Insertion of an asterisk (*) within the ANOVA table signifies statistical significance at $p < .05$ for that particular source.

Correlation coefficients are included in the results. These data were used to support or elucidate on the findings. For this reason only those coefficients which were of statistical significance (critical value of the correlation coefficient at the .01 level of significance being .658 for df = 10) were included in the results. However, a complete correlation matrix for each of the six test conditions is presented in Appendix B.

In some cases a post-hoc analysis (Newman-Keuls) was conducted. The prime concern was to show if significant differences existed from rest to 100% MVC within each environmental condition or between the environments for each specific workload.

Blood Pressure

Both BPs and BPd (Figures 6 and 7) reacted to isometric work at room temperature and in a heated environment in a very similar fashion. The ANOVA data (Tables 2 and 3) indicate that both systolic and diastolic pressures rise significantly from rest (R) to 100% MVC under the averaged influence of a control and heated environment. However the presence of a significant interaction for BPs ($p < .05$) and BPd ($p < .05$) does not allow one to concluded that blood pressure increases from rest to 100% MVC in a similar manner for both a neutral and heated environment. Consequently a post-hoc analysis of these comparisons was carried out using the Newman-Keuls method for multiple comparisons. This procedure gives the opportunity to compare the mean differences from R to 100% MVC within each environment. The results for both BPs and BPd are presented in Table 4.

Both BPs and BPd were significantly greater overall from R to 100% MVC in the control environment than in the heated environment (Table 2 and Table 3). In order to investigate exactly where this significance between the environments existed a Newman-Keuls analysis was again conducted. Comparisons were made between means of the two environments for each separate workload. The results are presented in Table 5.

Calculation of simple correlation coefficients for BPs and BPd showed significance only for BPs with TRIP (Table 6).

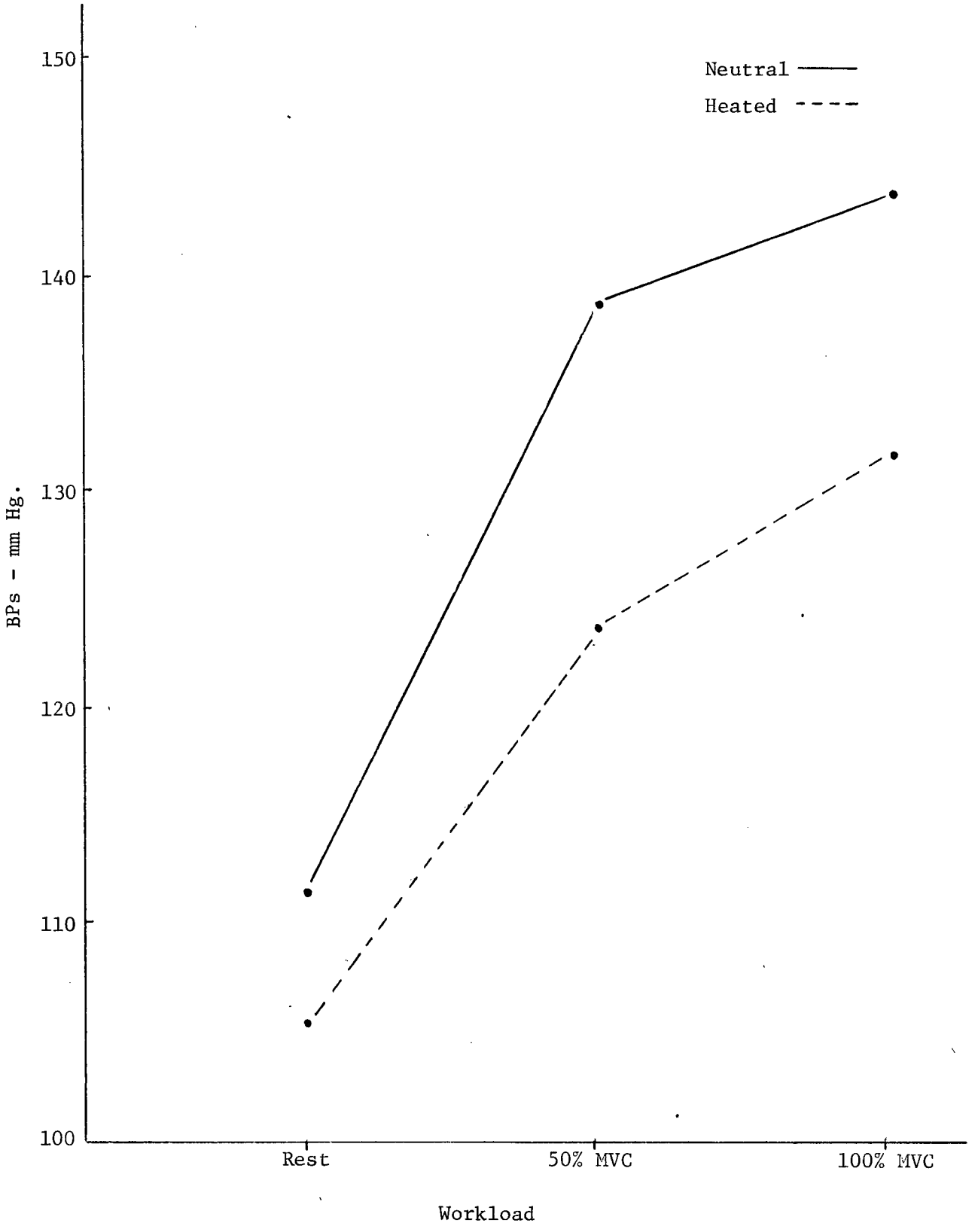


Figure 6. Systolic Blood Pressure

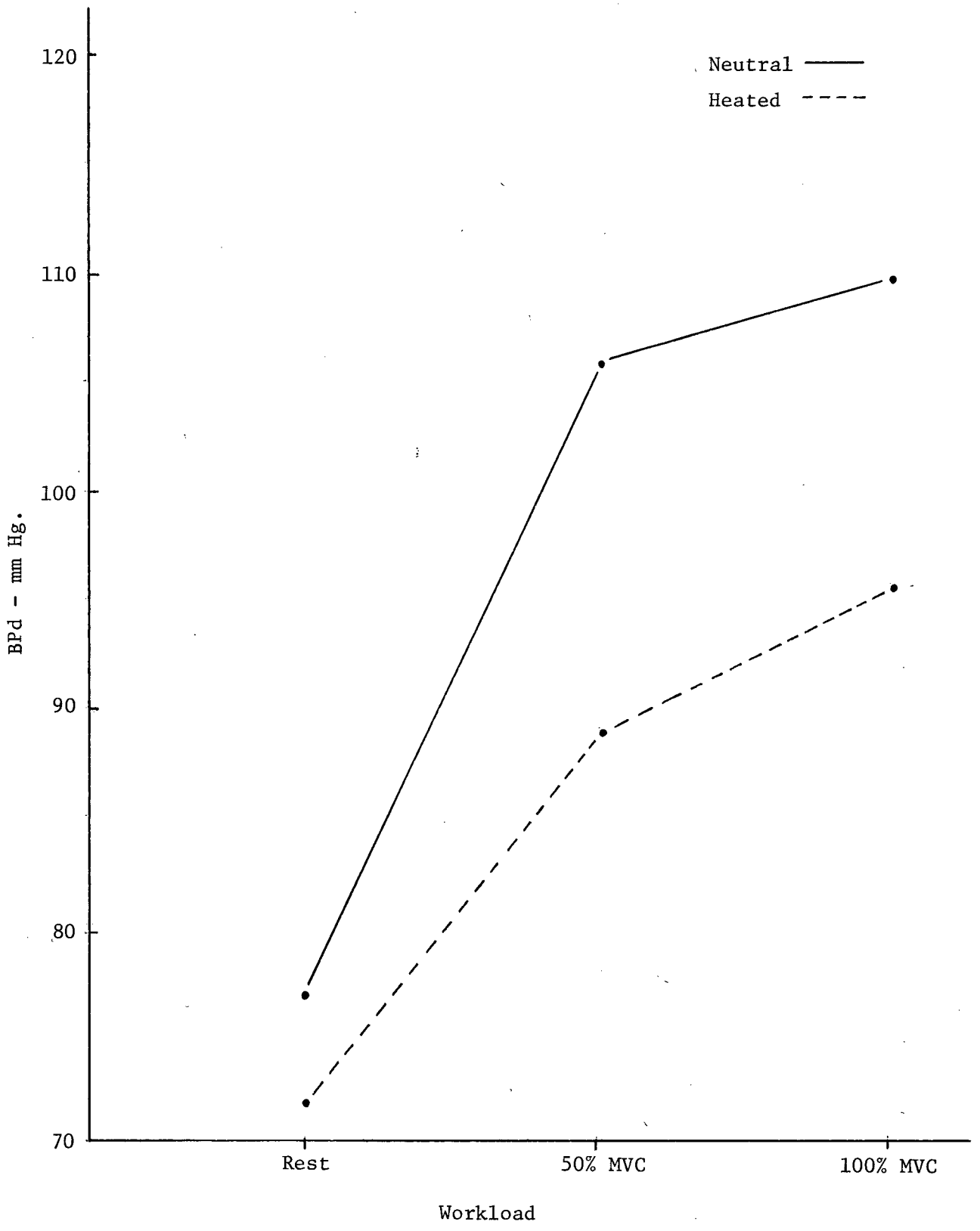


Figure 7. Diastolic Blood Pressure

TABLE 2

SUMMARY OF ANOVA

Blood Pressure (Systolic)

Source	df	Mean Square	F	P
E *	1	2080.1	25.6	<.001
Error	11	81.4		
C *	2	5502.9	53.5	<.001
Error	22	102.9		
C (1) *	1	9832.7	62.5	<.001
C (2) *	1	1173.1	24.2	<.001
EC *	2	136.8	3.8	0.038
Error	22	36.1		

TABLE 3

SUMMARY OF ANOVA

Blood Pressure (Diastolic)

Source	df	Mean Square	F	P
E *	1	2652.3	23.4	0.001
Error	11	113.1		
C *	2	5653.1	60.8	<.001
Error	22	93.1		
C (1) *	1	10034.1	85.1	<.001
C (2) *	1	1272.1	18.6	0.001
EC *	2	243.6	5.6	0.011
Error	22	43.2		

TABLE 4

NEWMAN-KEULS ANALYSIS (WORKLOAD EFFECT)

BPs and BPd

Comparison		R vs. 100% MVC		R vs. 50% MVC		50% MVC vs. 100% MVC	
Environment		N	H	N	H	N	H
BPs	Q. 95	3.55	3.55	2.94	2.94	2.94	2.94
	cal Q	10.92	8.88	9.21	6.14	1.70	2.73
	Result	p<.05	p<.05	p<.05	p<.05	N.S.	N.S.
BPd	Q. 95	3.55	3.55	2.94	2.94	2.94	2.94
	cal Q	12.05	8.78	10.61	6.18	1.43	2.58
	Result	p<.05	p<.05	p<.05	p<.05	N.S.	N.S.

From these results it can be concluded that both BPs and BPd increase from R to 100% MVC in both the control and heated environment.

TABLE 5

NEWMAN-KEULS ANALYSIS (ENVIRONMENT EFFECT)

BPs and BPd

Comparison		H vs. N Rest	H vs. N 50% MVC	H vs. N 100% MVC
BPs	Q. 95	3.11	3.11	3.11
	cal Q	2.11	5.77	4.50
	Result	N.S.	$p<.05$	$p<.05$
BPd	Q. 95	3.11	3.11	3.11
	cal Q	1.63	5.61	3.29
	Result	N.S.	$p<.05$	$p<.05$

These results show that both BPd and BPs are not significantly different at rest between the control and heated environment. However both BPd and BPs are significantly lower in the heated environment from the control condition at 50% MVC and 100% MVC.

TABLE 6

CORRELATION COEFFICIENTS

BPs

	Neutral			Heat		
	R	50% MVC	100% MVC	R	50% MVC	100% MVC
TRIP	.79	.84	.80	.62	.82	.90

These results support the strong direct association between BPs and myocardial oxygen consumption. Note however that at rest in the heat the correlation is not quite significant at the .01 level. This result is justifiable since in this condition HR plays a greater role than does BPs in the consumption of the available oxygen to the myocardium.

Heart Rate

Heart rate increased significantly from rest to 100% MVC in a linear fashion with both environments (Table 7). There were also significant differences between hot and neutral settings when averaged over the work-loads. Such conclusions can be presented from results of the ANOVA only, since there was no significant interaction. This means that any changes in HR from rest to 100% MVC were uniform in both environmental conditions. That is to say that the tachycardia induced by isometric work is additive to the tachycardia induced by thermoregulatory adjustments.

Heart rate was significantly correlated to a number of the systolic intervals as illustrated in Table 8.

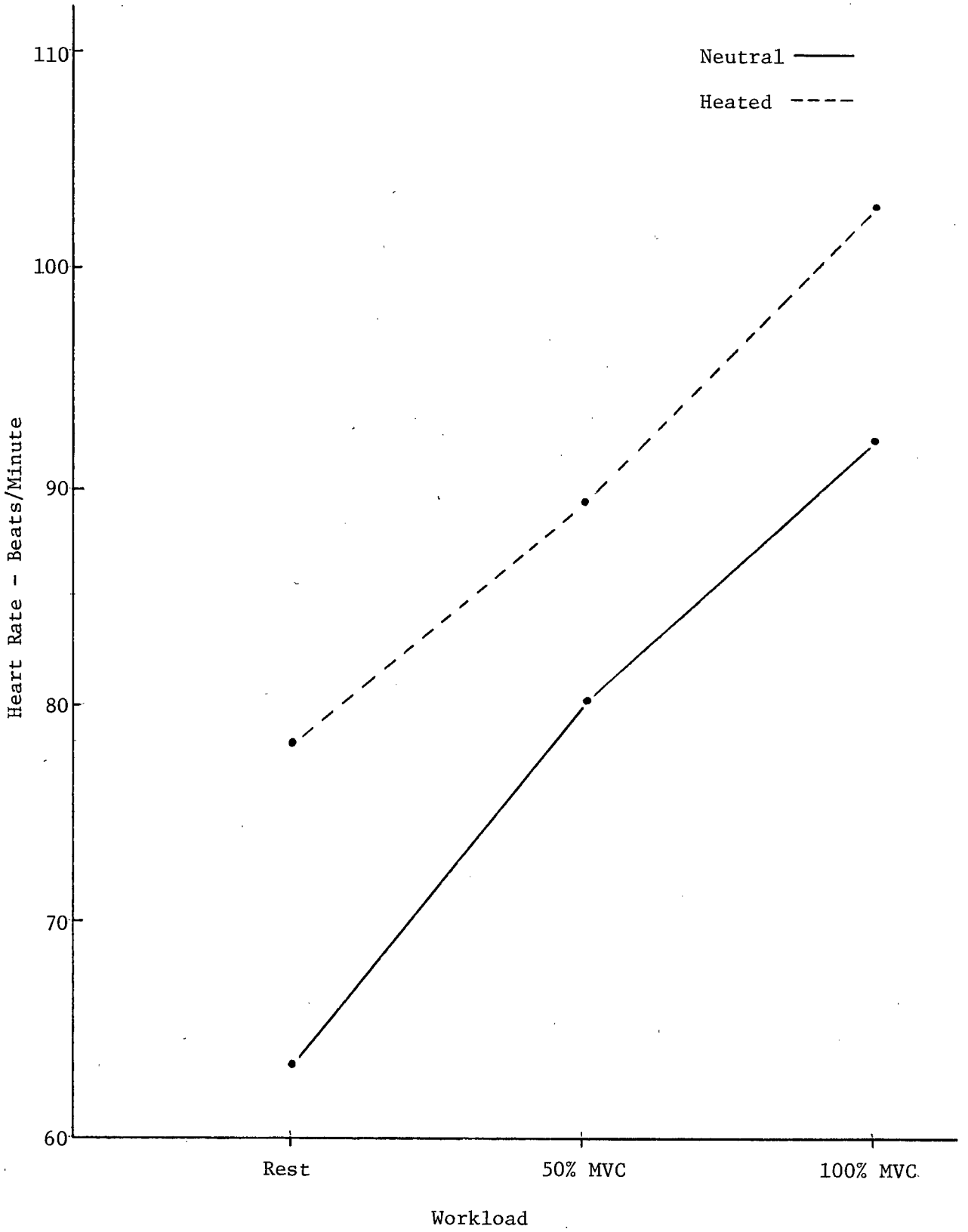


Figure 8. Heart Rate

TABLE 7

SUMMARY OF ANOVA

Heart Rate

Source	df	Mean Square	F	P
E *	1	2323.3	23.5	0.001
Error	11	98.8		
C *	2	4216.6	89.4	<.001
Error	22	47.2		
C (1) *	1	8426.9	125.6	<.001
C (2)	1	6.2	0.23	0.641
EC	2	63.3	2.2	0.131
Error	22	28.4		

TABLE 8

CORRELATION COEFFICIENTS

Heart Rate

Variable	Neutral			Heat		
	R	50% MVC	100% MVC	R	50% MVC	100% MVC
CT	-.99	-.99	-.99	-.99	-.99	-.99
LVET	-.77	-.60	-.43	-.82	-.84	-.83
TS	-.67	-.83	-.84	-.73	-.80	-.90
DIAST	-.98	-.98	-.98	-.99	-.97	-.96

These results show that HR is inversely correlated to the systolic and diastolic representatives of the cardiac cycle. However LVET does not correlate with HR as highly at 50% MVC and 100% MVC in the neutral environment as it does under the other test conditions. Why LVET should exhibit a weaker linear relationship with HR during isometric work in a control environment than in a heated environment is not known. It is possible that some other physiological factors, other than HR, which have not been revealed in this study, play a greater role in determining LVET during isometric stress in a neutral environment than in a heated environment. This would cause the total effect of HR upon LVET to become masked such that the correlation between them would appear to be diluted.

Ventricular Systole (LVET; MS; TS)

LVET, MS, and TS are the three variables used to identify alterations in systole. All three intervals changed in approximately the same manner. LVET decreased linearly from rest to 100% MVC as has been described in earlier studies (15, 42, 83). MS and TS also decreased from rest to 100% MVC with slightly different slopes than LVET. This irregularity can only be due to the changes in ICP which are incorporated into the MS and TS times. In all three cases however the results show a significant difference between environmental conditions and between workloads within each separate environment (Figures 9-11). This is supported by the presence of a non-significant interaction effect for all three variables (Tables 9-11) which signifies that the rate of decrease in LVET, MS and TS from rest to 100% MVC was very similar in both environments.

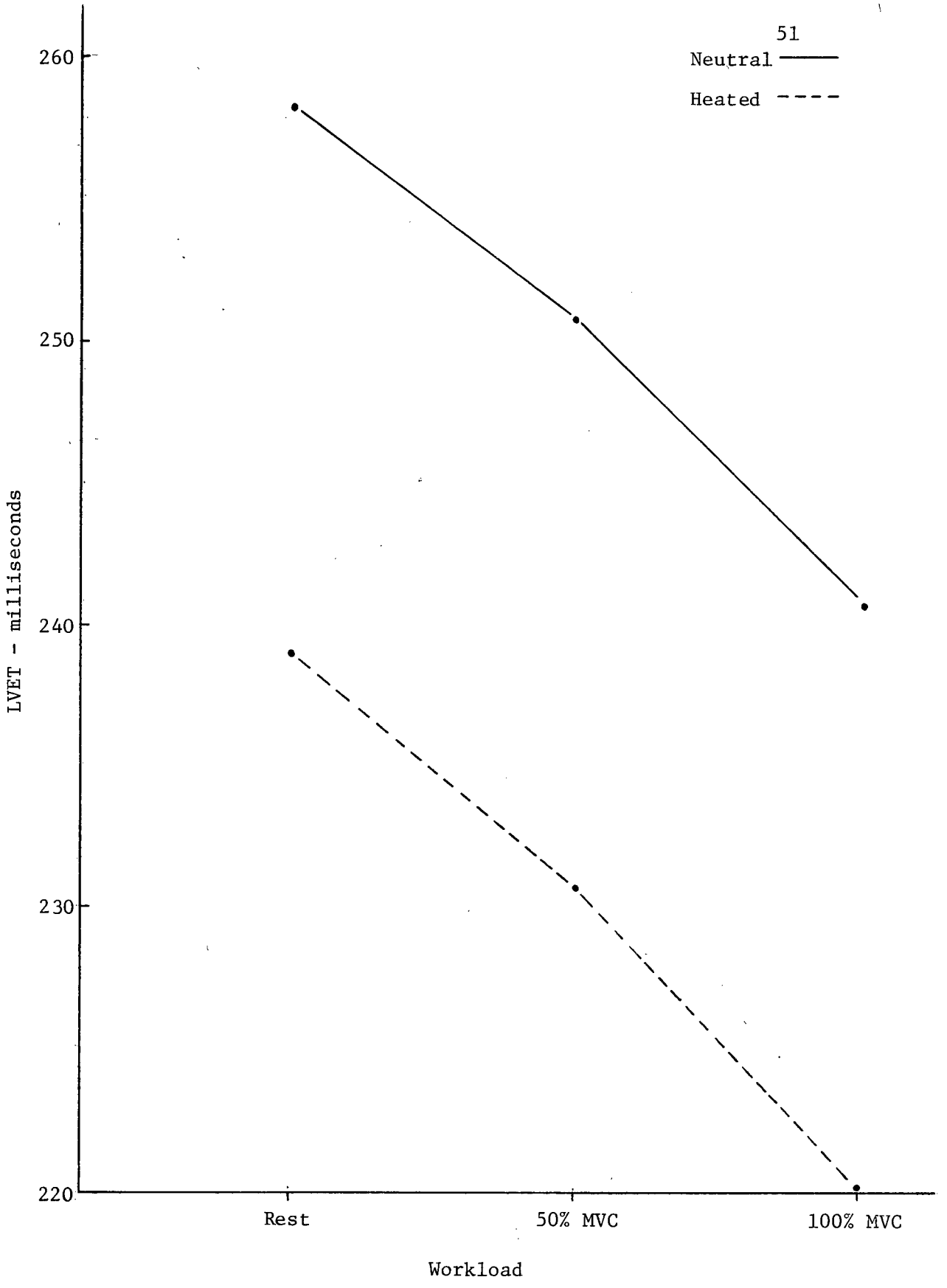


Figure 9. Left Ventricular Ejection Time

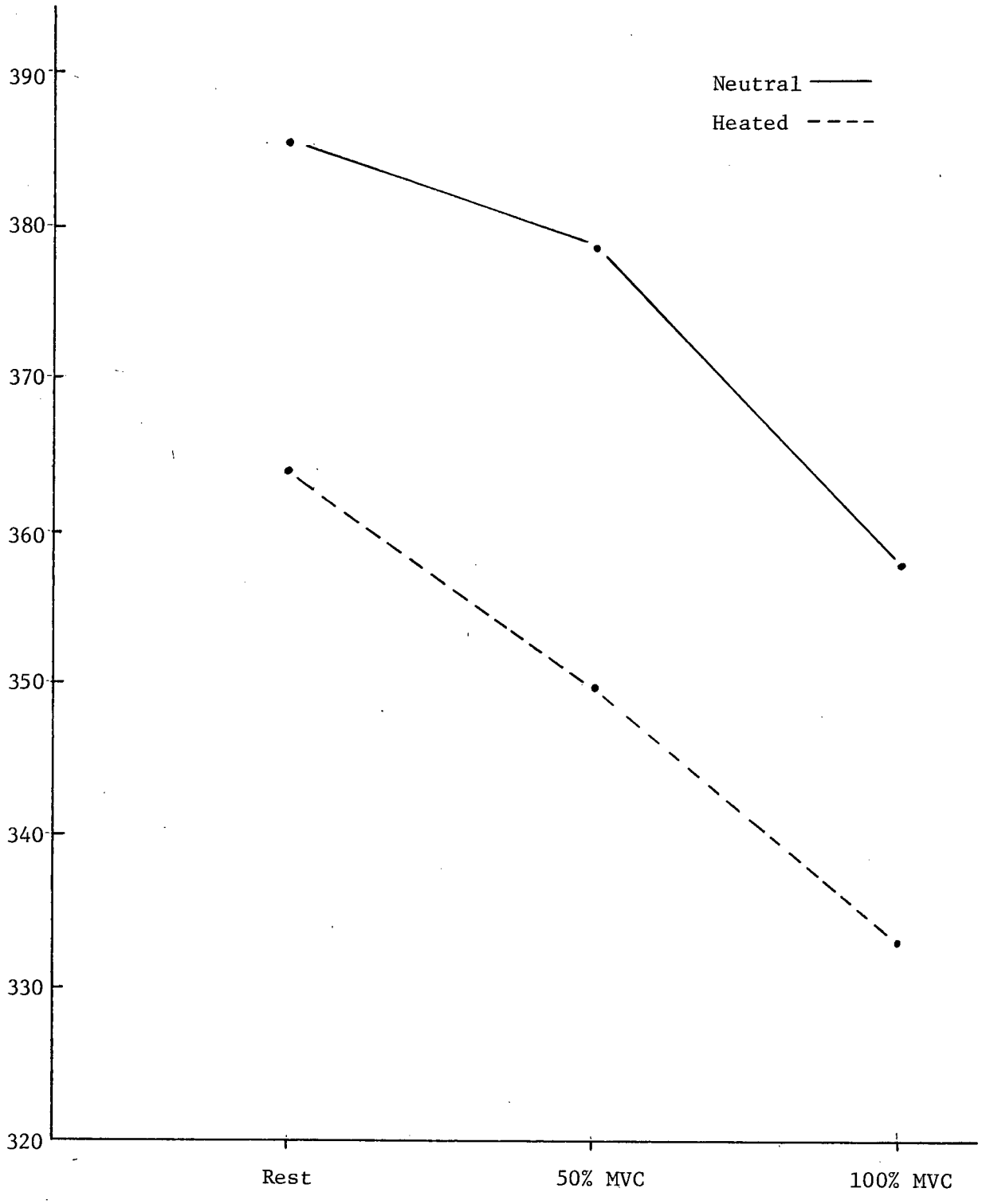


Figure 10. Total Systole

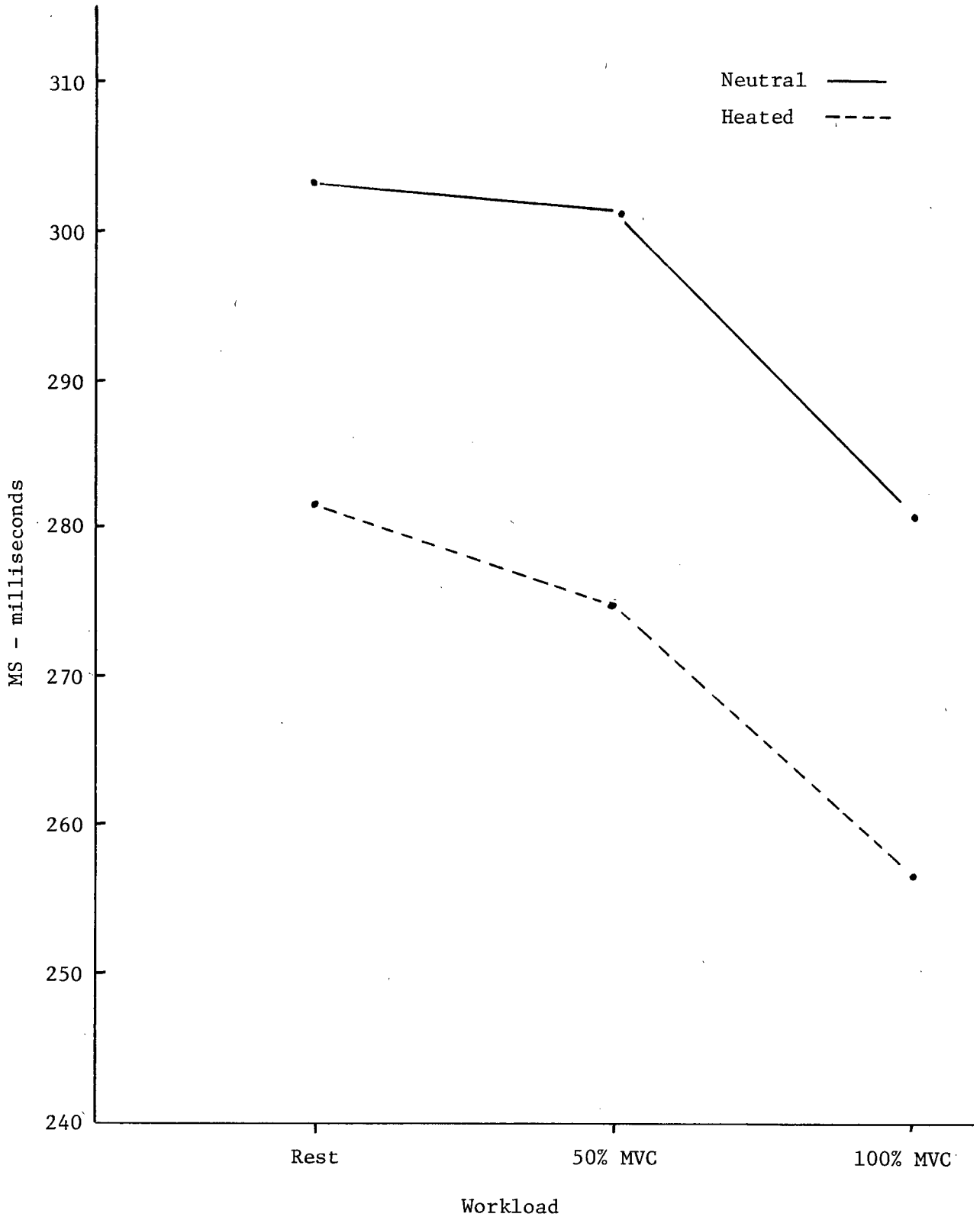


Figure 11. Mechanical Systole

TABLE 9

SUMMARY OF ANOVA

Left Ventricular Ejection Time

Source	df	Mean Square	F	P
E *	1	7219.9	17.8	0.001
Error	11	405.6		
C *	2	1804.5	18.0	<.001
Error	22	100.0		
C (1) *	1	3588.0	26.5	<.001
C (2)	1	21.0	0.32	0.580
EC	2	5.7	0.08	0.922
Error	22	70.0		

TABLE 10

SUMMARY OF ANOVA

Mechanical Systole

Source	df	Mean Square	F	P
E *	1	10512.5	21.1	.001
Error	11	498.4		
C *	2	3587.5	45.4	<.001
Error	22	79.1		
C (1) *	1	6188.0	65.0	<.001
C (2) *	1	987.0	15.7	0.002
EC	2	43.8	0.37	0.694
Error	22	117.8		

TABLE 11

SUMMARY OF ANOVA

Total Systole

Source	df	Mean Square	F	P
E *	1	11806.7	30.7	<.001
Error	11	384.2		
C *	2	5484.5	34.7	<.001
Error	22	158.2		
C (1) *	1	10620.7	61.2	<.001
C (2)	1	348.4	2.4	0.147
EC	2	71.9	0.56	0.578
Error	22	127.9		

TABLE 12

SUMMARY OF ANOVA

Ejection Time Index

Source	df	Mean Square	F	P
E	1	10.9	0.03	0.858
Error	11	324.4		
C *	2	4680.1	42.9	<.001
Error	22	109.1		
C (1) *	1	9268.4	66.0	<.001
C (2)	1	91.8	1.8	0.301
EC *	2	198.2	4.6	0.022
Error	22	43.4		

Ejection Time Index

The LVET was corrected for effects of HR using the ejection time index as described by Whitsett and Naughton (84) and Martin et al. (49). This correction eliminates HR as a significant variable influencing the duration of ventricular ejection. It is a relative index for which an increase would mean that without the associated effects of HR on LVET the time which the ventricle spends in ejection of blood is greater. However the actual time spent in ejection may be significantly decreased as a result of tachycardia.

Figure 12 shows how ETI increases linearly from rest to 100% MVC in both environments. However in view of the significant interaction (Table 12) conclusions about the increase in ETI from rest to 100% MVC within each environment could only be made after a post-hoc analysis. The results of the Newman-Keuls multiple comparison for ETI is presented in Table 13.

TABLE 13

NEWMAN-KEULS ANALYSIS

ETI

	Rest vs. 100% MVC		Rest vs. 50% MVC		50% MVC vs. 100% MVC	
	Neutral	Heat	Neutral	Heat	Neutral	Heat
Q. 95	3.55	3.55	2.94	2.94	2.94	2.94
cal Q	10.79	7.64	7.14	3.65	3.65	3.99
Result	$p<.05$	$p<.05$	$p<.05$	$p<.05$	$p<.05$	$p<.05$

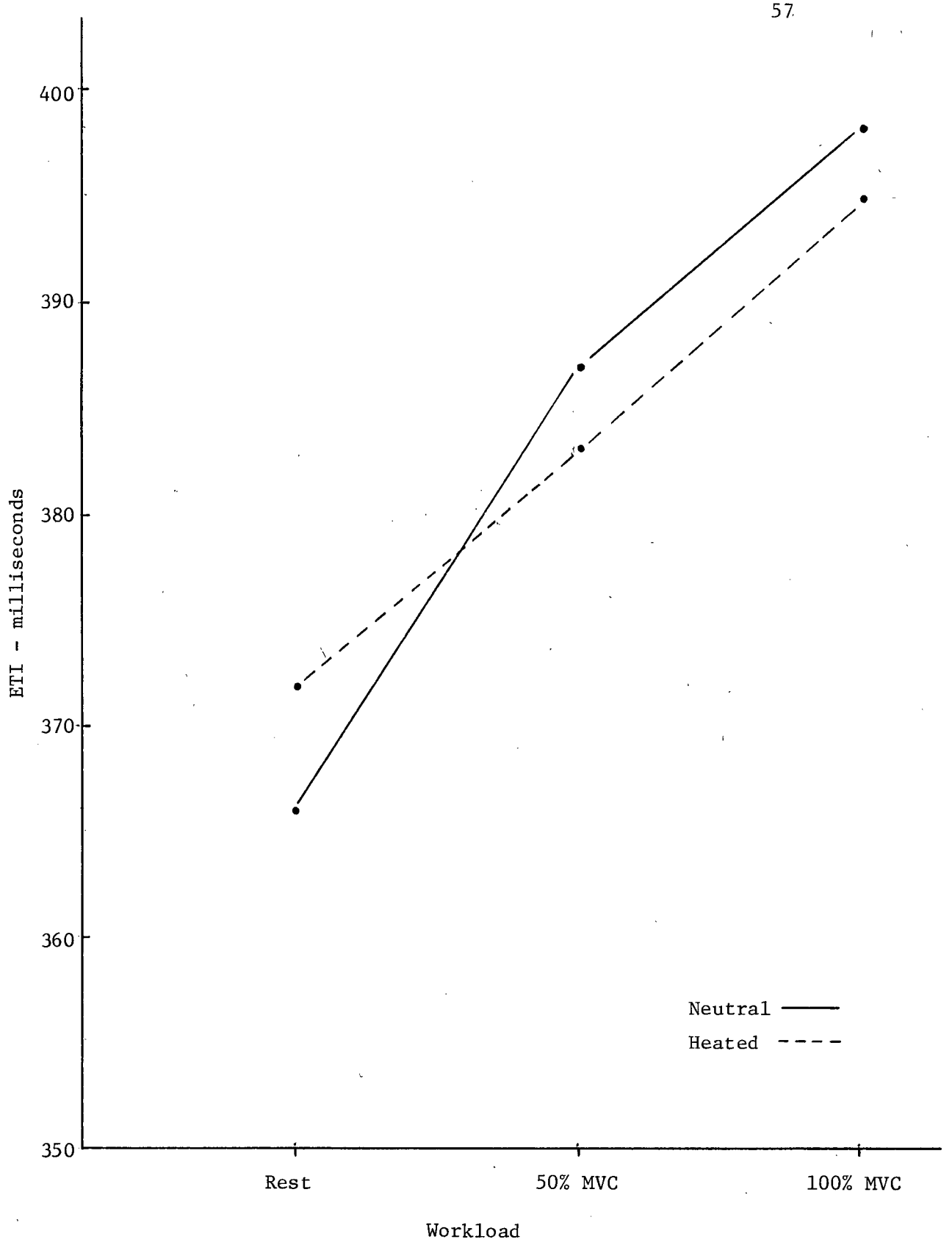


Figure 12. Ejection Time Index

The foregoing results conclusively demonstrate that ETI increases significantly from rest to 100% MVC in both a neutral and heated environment. This implies that disregarding the effects of tachycardia on the actual ventricular ejection time, an isometric contraction of the forearm causes a relative increase in the amount of time spent in left ventricular ejection.

There was no significant difference in the ETI between the two environments as shown by a non-significant environmental (E) effect. A post-hoc analysis was done to investigate if there was a significantly greater ETI at rest in the heated environment as opposed to a control environment. The results for the Newman-Keuls analysis demonstrated a calculated Q of 1.13 which was below the critical Q value of 3.11 needed for significance at the .05 level.

The significant interaction effect (Table 12) for ETI indicates that without the physiological association of HR, the rate of change in LVET from conditions of rest to isometric stress is different for each of the two environments. The interactive nature in the slopes of the two lines for ETI (Figure 12) is probably due to the difference in ETI at rest. This difference, although not statistically significant, was enough to produce a significant EC effect.

Diastole and CT

Both diastole and CT decreased linearly from rest to 100% MVC as illustrated in Figure 13 and Figure 14. However in view of a significant EC (Tables 14 and 15) for both variables a post-hoc analysis was carried out to show specifically where the significant changes occurred. The results of the Newman-Keuls comparison for both diastole and CT are presented in Table 16.

TABLE 16

NEWMAN-KEULS ANALYSIS

Diastole and CT (Workload Effect)

		Rest vs Neutral	100% MVC Heat	Rest vs. Neutral	50% MVC Heat	50% MVC vs. Neutral	100% MVC Heat
	Q. 95	3.55	3.55	2.94	2.94	2.94	2.94
Diastole	cal Q	16.42	9.44	5.43	4.69	12.05	4.76
	Result	$p<.05$	$p<.05$	$p<.05$	$p<.05$	$p<.05$	$p<.05$
	Q. 95	3.55	3.55	2.94	2.94	2.94	2.94
CT	cal Q	16.46	10.22	5.86	5.08	10.60	5.17
	Result	$p<.05$	$p<.05$	$p<.05$	$p<.05$	$p<.05$	$p<.05$

The above results confirm that both diastole and CT decrease significantly from rest to 100% MVC in both environmental conditions.

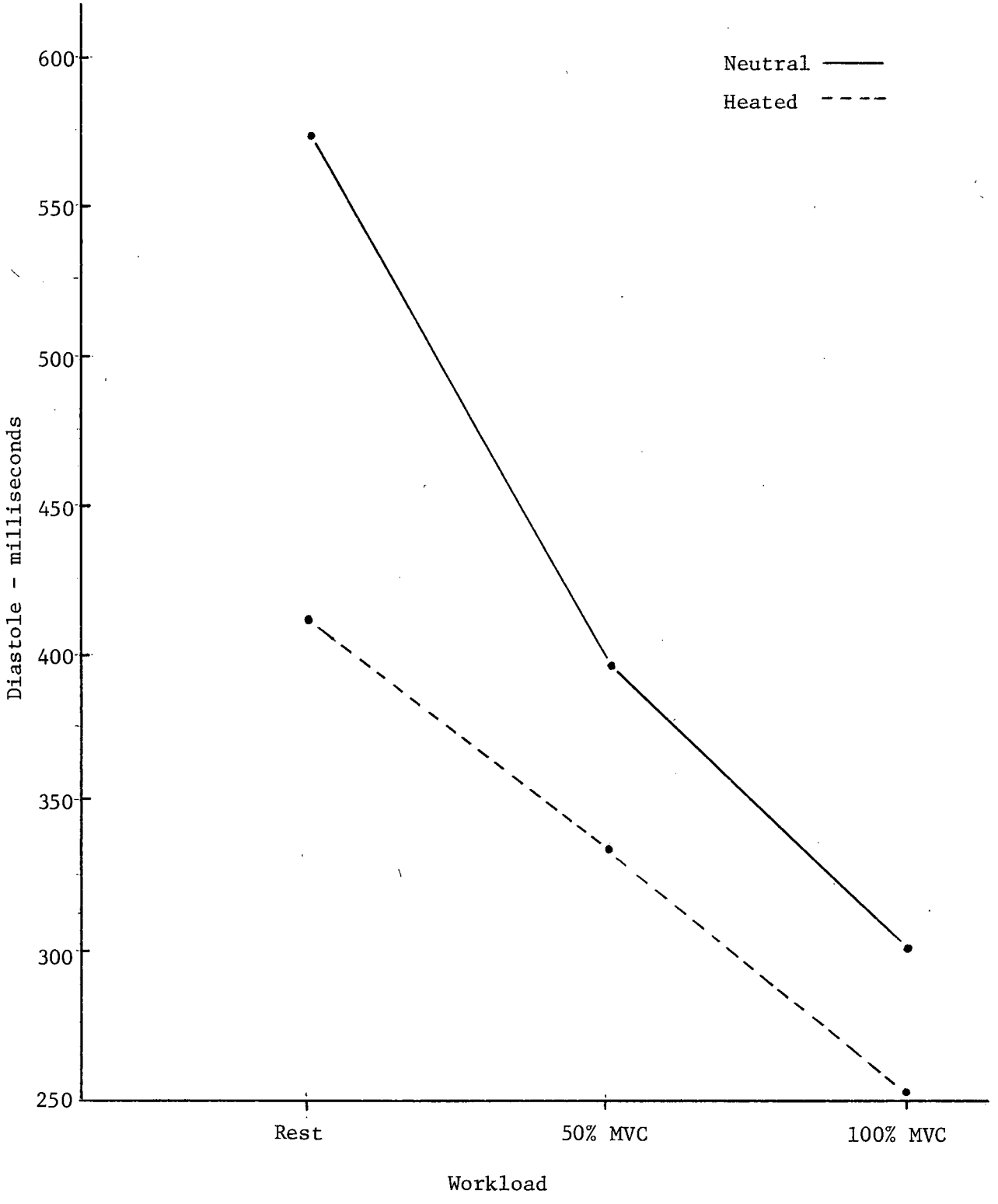


Figure 13. Diastole

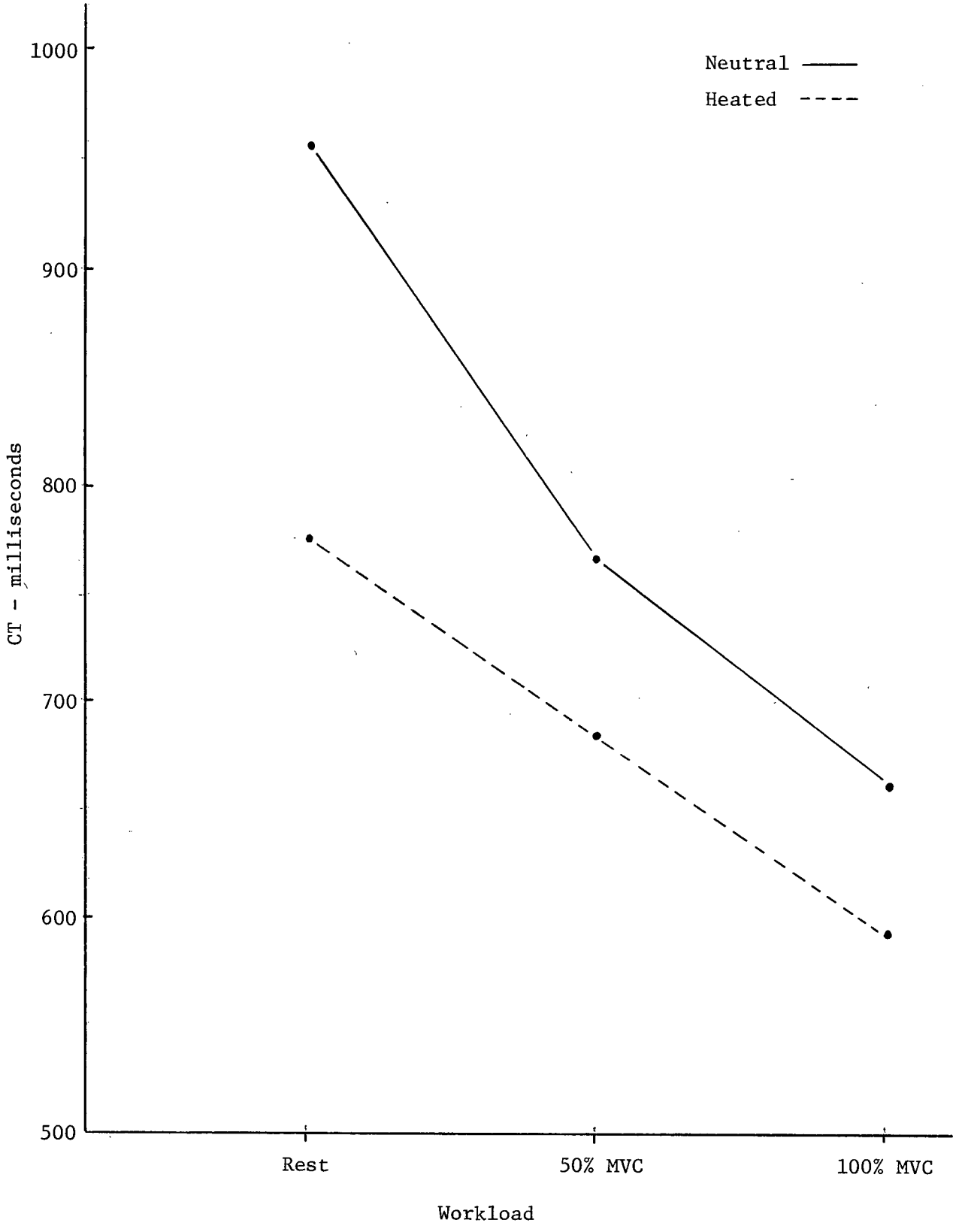


Figure 14. Cycle Time

TABLE 14

SUMMARY OF ANOVA

Diastole

Source	df	Mean Square	F	P
E *	1	141778.0	18.8	0.001
Error	11	7537.3		
C *	2	273456.3	84.5	<.001
Error	22	3235.8		
C (1) *	1	538479.1	153.1	<.001
C (2)	1	8433.4	2.9	0.119
EC *	2	23499.0	10.3	0.001
Error	22	2275.9		

TABLE 15

SUMMARY OF ANOVA

Cycle Time

Source	df	Mean Square	F	P
E *	1	227812.8	22.7	0.001
Error	11	10036.8		
C *	2	354486.3	90.4	<.001
Error	22	3919.6		
C (1) *	1	701316.4	156.3	<.001
C (2)	1	7656.3	2.3	0.159
EC *	2	22568.6	7.6	0.003
Error	22	2972.0		

Diastole and CT were also significantly less in the heated environment than in the control environment as shown by a significant E effect in Table 14 and Table 15. A Newman-Keuls analysis was again conducted to discover at what workloads the environmental difference for both diastole and CT was most significant. The results are presented in Table 17.

TABLE 17

NEWMAN-KEULS ANALYSIS

Diastole and CT (Environmental Effect)

		H vs. N Rest	H vs. N 50% MVC	H vs N 100% MVC
Diastole	Q. 95	3.11	3.11	3.11
	cal Q	6.41	2.35	2.02
	Result	$p<.05$	N.S.	N.S.
CT	Q. 95	3.11	3.11	3.11
	cal Q	6.32	2.92	2.42
	Result	$p<.05$	N.S.	N.S.

In contrast to the overall significant difference demonstrated by the ANOVA data between environments, the post-hoc analysis shows that only at rest was there a difference in diastole and CT between the heated and control environment.

The correlative data express extremely strong association between CT, diastole and HR (Table 8). In fact HR has a stronger correlation with CT and diastole than it does with LVET. In addition CT is found to be significantly correlated to a number of other variables (Table 18).

TABLE 18

CORRELATION COEFFICIENTS

CT

		EML	LVET	TS	DIAS	TRIP
	Rest	.18	.73	.65	.99	-.78
Neutral	50% MVC	.85	.57	.77	.99	-.87
	100% MVC	.74	.45	.85	.99	-.89
	Rest	-.13	.84	.75	.99	-.41
Heat	50% MVC	.64	.82	.79	.99	-.65
	100% MVC	.55	.82	.87	.98	-.75

The high correlation between CT and diastole support the findings of Franks et al. (18) who demonstrated that diastole and CT are measures of a similar dynamic function in the cardiac cycle. The significant correlations between CT and TRIP and CT and systole (LVET and TS) are probably a direct result of production of tachycardia which reduces CT and affects both systole and myocardial oxygen consumption. Of surprising interest however, is the significant correlation between EML and CT with 50% MVC and 100% MVC

especially in the neutral environment. An explanation for this finding could be that in stress conditions when the inotropic state of the myocardium is increasing causing alterations in myocardial contractility and rate of contraction, the electrical activity of the heart is altered to maintain a level of excitability needed to cope with the change in cardiac dynamics.

Sympathoadrenergic Activity

The three variables which most closely represent the contractile state of the myocardium which in turn is dependent upon the sympathetic and adrenergic sources of stimulation to the heart tissue are ICP, PEP, and PEP/LVET.

Table 19 and Table 20 demonstrate a significant workload (C) effect signifying that there was an overall change in both ICP and PEP from rest to 100% MVC. ICP increases or remains constant from rest to 50% MVC but decreases in both environments from 50% MVC to 100% MVC (Figure 14). PEP increases slightly from rest to 50% MVC in the control environment but decreases between the same workloads in the heat and between 50% MVC and 100% MVC in both environments. Since there was no significant interaction effect for either ICP or PEP further post-hoc analysis was not needed. Therefore it is concluded that both ICP and PEP change in a similar fashion during isometric work in control conditions and in a heated environment.

There was no significant difference throughout the workloads between the two environments for both ICP and PEP. The absence of any EC effect demonstrated that the changes in these two variables produced by a

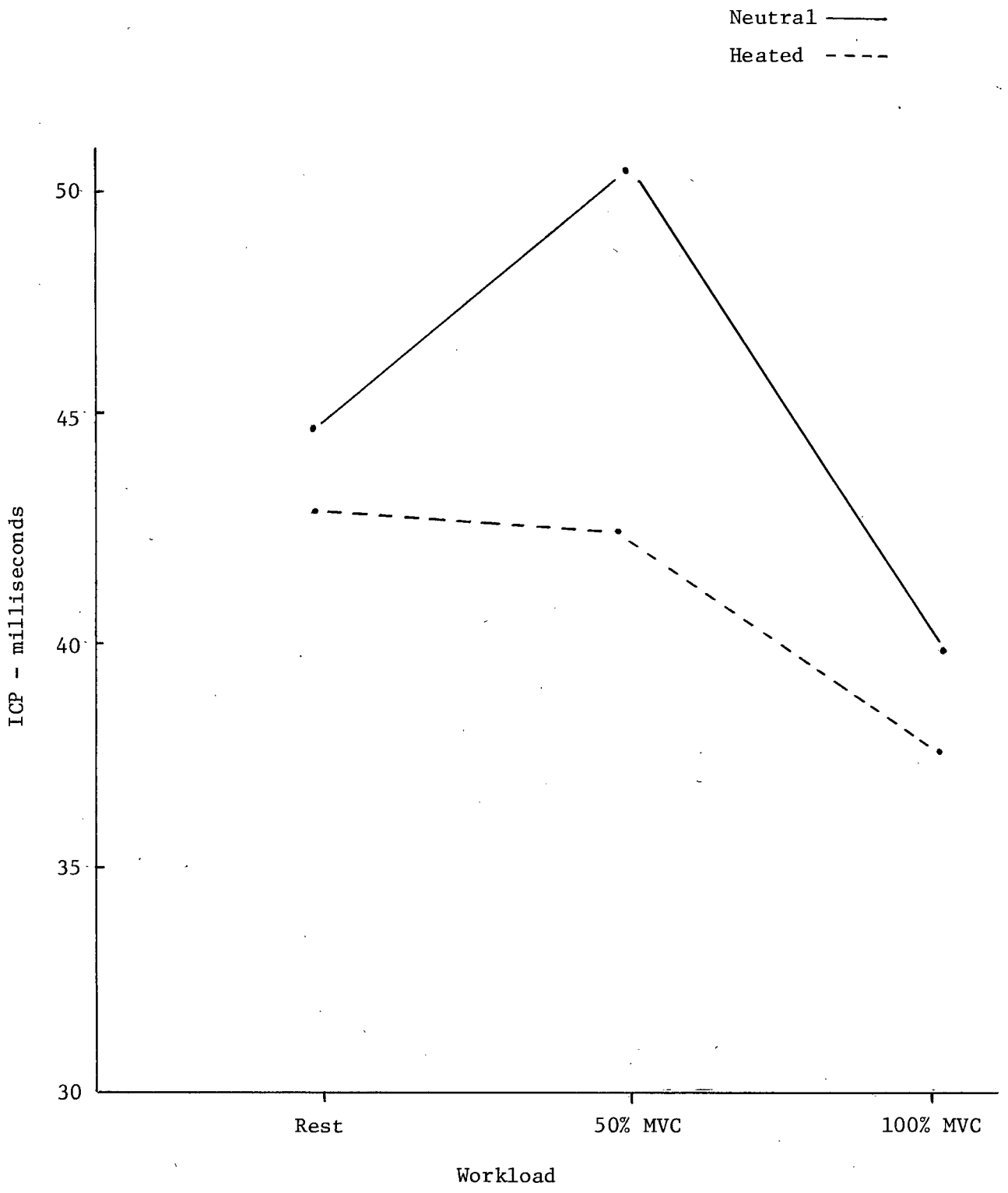


Figure 15. Isovolumetric Contraction Period

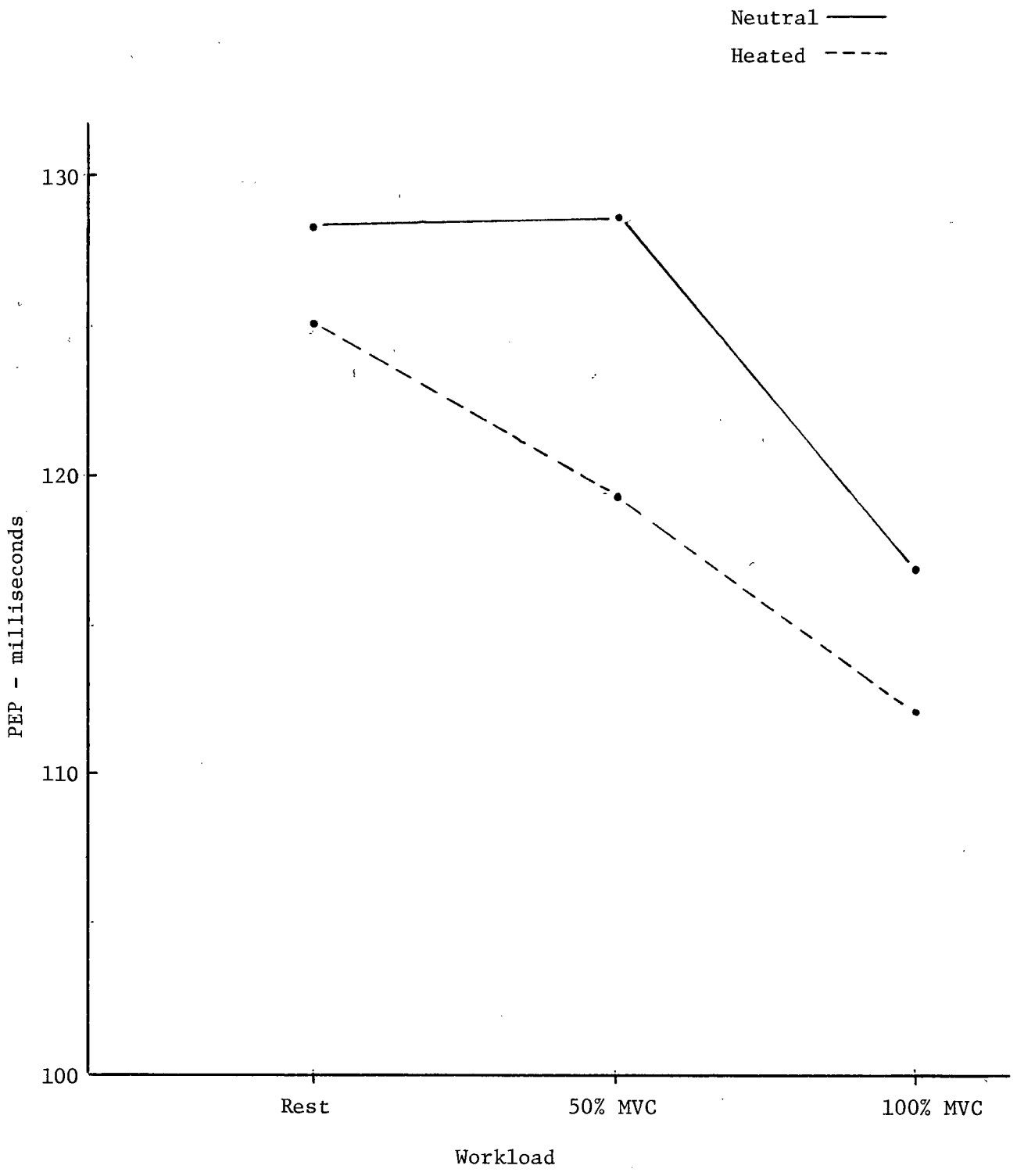


Figure 16. Pre-ejection Period

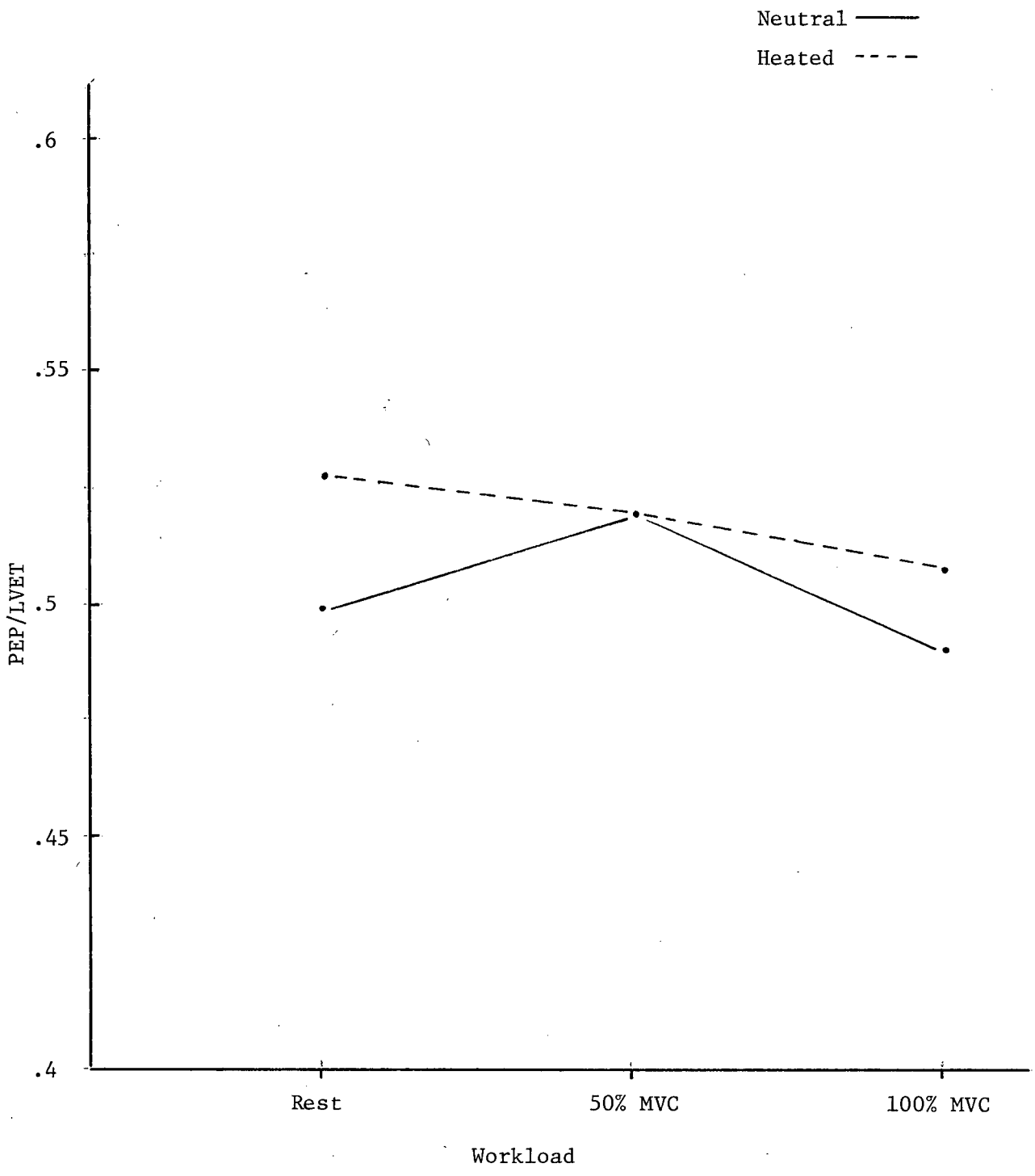


Figure 17.

Pre-ejection Period: Left Ventricular Ejection Time (Ratio)

TABLE 19

SUMMARY OF ANOVA

Isovolumetric Contraction Period

Source	df	Mean Square	F	P
E	1	284.0	1.2	0.294
Error	11	234.1		
C *	2	405.5	6.1	0.008
Error	22	66.1		
C (1)	1	363.0	3.7	0.081
C (2) *	1	448.0	13.4	0.004
EC	2	96.3	1.0	0.379
Error	22	94.9		

TABLE 20

SUMMARY OF ANOVA

Pre-Ejection Period

Source	df	Mean Square	F	P
E	1	578.0	2.8	0.124
Error	11	208.7		
C *	2	1005.7	9.4	0.001
Error	22	107.4		
C (1) *	1	1813.0	11.7	0.006
C (2)	1	198.3	3.3	0.095
EC	2	67.0	1.1	0.347
Error	22	60.4		

TABLE 21

SUMMARY OF ANOVA

PEP/LVET

Source	df	Mean Square	F	P
E	1	0.00637	0.83	0.382
Error	11	0.00769		
C	2	0.00239	0.85	0.439
Error	22	0.00280		
C (1)	1	0.00272	0.62	0.447
C (2)	1	0.00206	1.66	0.223
EC	2	0.00102	0.72	0.497
Error	22	0.00141		

simultaneous subjection to heat and muscular stress was additive in nature. However a visual note of the alterations in ICP from rest to 50% MVC in the control environments lends one to think that a significant environmental effect may in fact be present here. To investigate this point a post-hoc analysis was conducted for ICP between the two environmental conditions at 50% MVC. The results of the Newman-Keuls comparison indicated a non-significant calculated Q as presented in Table 22.

TABLE 22

NEWMAN-KEULS ANALYSIS

ICP (Heat vs. Neutral)

	Q.95	cal Q	Result
50% MVC	3.11	1.95	N.S.

The above findings clearly support the ANOVA which showed a non-significant environmental effect.

The third variable in this group, PEP/LVET, demonstrated a non-significant workload (C) and environment (E) effect (Table 21). Such results have been reported earlier for this variable (42, 75). Non-significant changes in this ratio during isometric stress suggests that the myocardium was in a state of increased contractility in response to a greater pressure load.

TABLE 23

SUMMARY OF ANOVA

Electromechanical Lag

Source	df	Mean Square	F	P
E	1	56.9	0.73	0.411
Error	11	78.0		
C *	2	290.0	4.47	0.024
Error	22	64.9		
C (1) *	1	533.3	9.9	0.009
C (2)	1	46.7	0.6	0.449
EC	2	3.76	0.07	0.936
Error	22	57.18		

Electromechanical Lag

EML decreases linearly from rest to 100% MVC when averaged over environments (Figure 18). This is supported by a significant workload (C) effect in the analysis of variance (Table 23). The absence of an interaction for this variable further complements the overall decrease in EML. However in order to specify exactly where the significant changes in EML had occurred subsequent analysis was conducted by the Newman-Keuls method. The results are presented in Table 24.

In contrast to the results of the ANOVA the post-hoc data indicate that EML did not significantly decrease from rest to 50% MVC, 50% MVC to 100% MVC, or from rest to 100% MVC in either of the environmental conditions.

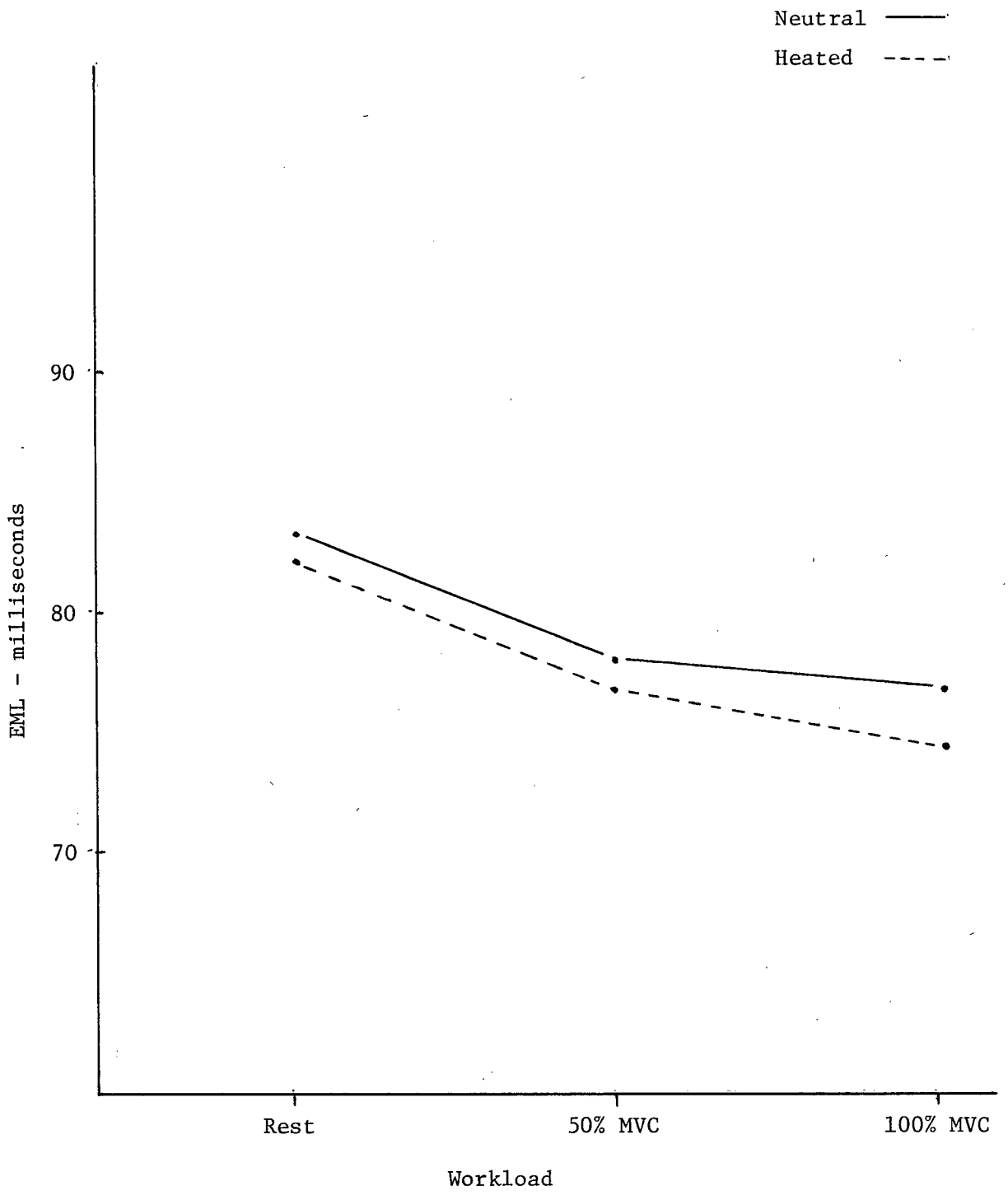


Figure 18. Electromechanical Lag

TABLE 24

NEWMAN-KEULS ANALYSIS

EML

	Rest vs. 100% MVC		Rest vs. 50% MVC		50% MVC vs. 100% MVC	
	Neutral	Heat	Neutral	Heat	Neutral	Heat
Q. 95	3.55	3.55	2.94	2.94	2.94	2.94
cal Q	2.71	3.27	0.43	1.98	2.28	1.29
Result	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.

The reason for this discrepancy is that the Newman-Keuls method is a much more conservative test than the ANOVA. It can therefore be concluded that EML has a tendency to decrease from rest to 100% MVC but that upon further analysis by a more rigorous post-hoc method no significant change in EML can be found in either environment.

A final note is that EML was not significantly altered from a neutral to a heated environment throughout the workloads. This is supported by a non-significant E effect (Table 23).

EML was significantly correlated with a number of other variables. This data is presented in Table 25.

TABLE 25

CORRELATION COEFFICIENTS

EML

	Neutral			Heat		
	Rest	50% MVC	100% MVC	Rest	50% MVC	100% MVC
Diastole	.17	.86	.71	-.14	.61	.52
CT	.18	.85	.74	-.13	.64	.55
HR	-.13	-.82	-.76	.13	-.62	-.52

TABLE 26

SUMMARY OF ANOVA

Triple Product

Source	df	Mean Square	F	P
E	1	257762.0	2.7	0.129
Error	11	95456.6		
C *	2	8820472.0	77.6	<.001
Error	22	113627.0		
C (1) *	1	17057584.0	104.4	<.001
C (2) *	1	583375.4	9.1	0.012
EC *	2	303208.3	7.2	0.004
Error	22	41906.1		

TRIP

Analysis of variance results showed an overall increase in TRIP from rest to 100% MVC (Table 26). It was thought beneficial to the study to inspect the specific changes in this variable between workloads for each environmental condition. The results of the post-hoc analysis are presented in Table 27.

TABLE 27

NEWMAN-KEULS ANALYSIS

TRIP (Workload Effect)

	Rest vs. 100% MVC		Rest vs. 50% MVC		50% MVC vs. 100% MVC	
	Neutral	Heat	Neutral	Heat	Neutral	Heat
Q. 95	3.55	3.55	2.94	2.94	2.94	2.94
cal Q	14.19	10.31	10.15	6.02	4.03	4.29
Result	$p<.05$	$p<.05$	$p<.05$	$p<.05$	$p<.05$	$p<.05$

The above results confirm those of the ANOVA. TRIP significantly increases from rest to 100% MVC in both environmental conditions.

There was no significant E effect for TRIP signifying that the changes induced by heat during each workload were not statistically significant when compared to the control environment. However, in view of the significant EC displayed in Table 26 a post-hoc analysis was again conducted to test for significant environmentally induced alterations in TRIP at each workload. The Newman-Keuls method was again administered, the results of which are presented in Table 28.

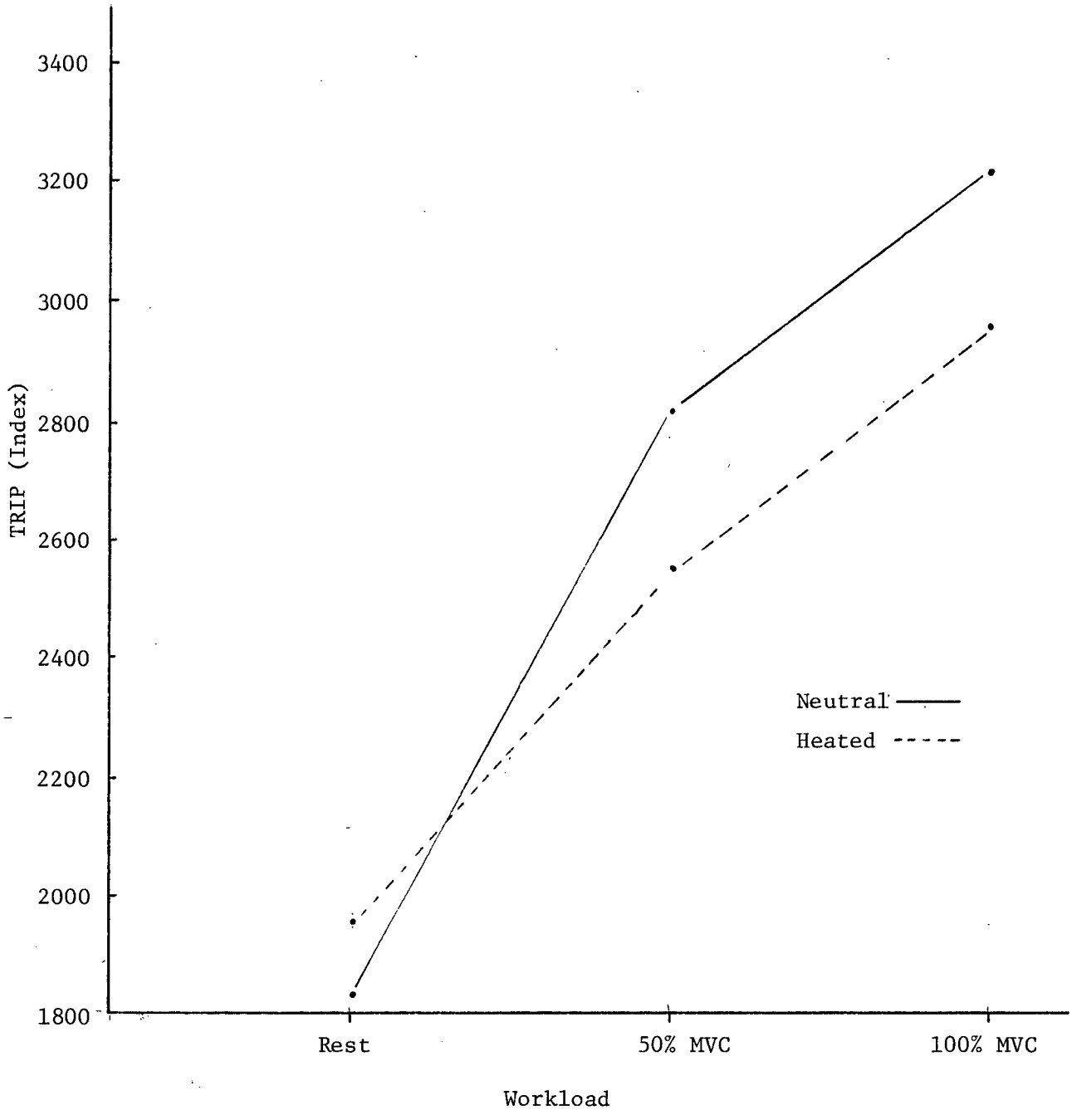


Figure 19. Triple Product

TABLE 28

NEWMAN-KEULS ANALYSIS

TRIP (Heat vs. Neutral)

	Q.95	cal Q	Result
Rest	3.11	1.57	N.S.
50% MVC	3.11	2.94	N.S.
100% MVC	3.11	2.66	N.S.

The above data confirm that indeed there is no significant change in TRIP for any of the workload conditions between the two environments. Therefore it seems that the heat stress did not significantly increase the myocardial oxygen requirements.

II. DISCUSSION

Analysis of the results stimulates a variety of conclusions about the cardiovascular dynamics associated with heat stress and static exertion. Some of the findings are novel and have created incentive for further research while others seem to support results of earlier investigations.

Blood Pressure

As has been demonstrated by many earlier investigations (26, 33, 37, 45) both BPd and BPs significantly increase during submaximal and maximal bouts of static muscular contraction. This response is not only present in a control environment but also when the skin temperature is elevated to

40°C - 41°C. However the presence of a significant interaction effect for BPs and BPd ($p < .05$) suggests that body heating causes the changes in blood pressure during each workload condition to be different from those pressures elicited in a control environment.

Analysis of variance shows that there is an overall significant decrease in both BPs and BPd throughout the workloads from a control to a heated environment. Further post-hoc testing demonstrated that BPs and BPd in a resting state did not significantly differ from a control to a heated condition which is in support of Hypothesis 4 and earlier research by Rowell (69). Of novel interest was the finding that BPs and BPd were significantly reduced during submaximal and maximal isometric work in the heat. This result does not support Hypothesis 4 but does encourage discussion about the dominant physiological adjustment during simultaneous subjection of the human body to isometric muscular contraction and thermal stress. It seems that although the pressor response to submaximal and maximal static exertion is functional during body heating, its dominance as seen during isometric work in a neutral environment, is not as powerful when thermoregulatory processes are simultaneously being activated. Such a difference is undoubtedly due to the vasodilation produced at the skin surface in response to increasing body temperature.

This result is supported by the findings of Heistad et al. (24) who used seventeen healthy men and women, 18-25 years of age, to investigate a possible central interaction of thermal and baroreceptors. They found that modification of the baroreceptor reflex by thermal stimuli may occur at a central level. Specifically interaction may occur between efferent

messages from the central component of the baroreceptor reflex, the medulla, and the central component of the thermal reflex, namely the hypothalamus. Johnson et al. (29) used similar methods to determine whether skin will respond to increased neurogenic vasoconstrictor activity during heating. The results showed that during heating, skin retains the ability to vasoconstrict but that this vasoconstriction cannot override heat-induced vasodilation.

Heart Rate

As expected HR increased linearly with submaximal and maximal static exertion in both environmental settings. It is a well documented fact that HR rises with isometric exercise (36, 37, 38) and during whole body heating (6, 9).

HR was significantly greater for all workloads during body heating. The rate of increase in HR from rest to 100% MVC was very similar for both environments, as supported by a non-significant EC effect. This suggests that the tachycardia induced by thermoregulatory processes is maintained throughout all workloads and that the rise in HR induced by isometric work is additive to the rise in HR due to thermoregulatory adjustments.

Ventricular Systole

The changes observed in LVET, TS and MS were very similar to findings reported by other workers (42, 65, 82). All three variables decreased significantly ($p < .001$) from rest to 100% MVC in both environmental conditions. These results support Hypothesis 7.

LVET, TS and MS were also significantly less ($p < .001$) in the heat than in the control environment for all workloads. This decrease was additive to that induced by isometric exertion which implies that physiological adjustment to thermal and static stress stimulates alterations in a common factor which directly affects systole. That factor is probably HR since changes in LVET and to a lesser extent MS and TS are strongly associated to changes in the rate of myocardial contraction (81, 82). The overall correlation coefficient (average of the six testing conditions) between HR and LVET in this study was .71 which supports the strong relationship between these two variables.

It seems therefore that changes in systole are dependent upon the rate of myocardial contraction. Increase in HR can be elicited by both thermoregulation and isometric muscular contraction. During such conditions decreases in systole are paralleled by simultaneous increases in HR.

Ejection Time Index

LVET was "corrected" for HR to allow for the assessment of other factors which may possibly alter the ejection period. If no factors other than HR had affected LVET, all curves for ETI would be essentially flat, which was not the case in this study. ETI increased significantly ($p < .001$) from rest to 100% MVC in both environments. Quarry and Spodick (65) reported similar results during 30% MVC and 50% MVC in sitting position. Martin et al.(48) also found the ETI to increase significantly during a 30% MVC for three minutes. These authors suggest that the augmented ETI during IHG may be indirectly caused by increases in afterload or directly due to increases in stroke volume (SV).

The effects of independent increases in systemic pressure and stroke volume on ejection time have been studied but results have not been consistent. Wallace et al. (80), using a right heart bypass preparation in dogs, found that elevating BPm to 140-160 mm Hg. shortened ejection time. However, augmenting SV separately prolonged LVET. Braunwald et al. (5) similarly found that an increase in SV alone would prolong the ejection period but in contrast to Wallace's findings, augmenting BPm to 150 mm Hg. did not significantly alter ejection time. These results suggest that without the influence of HR ejection time may be prolonged by increases in SV. However, an increase in SV is associated with a possible increase in LVEDP (preload) and since it has been reported that in normals an IHG contraction produces little change in preload (14, 25, 75) and SV (13, 37) it is unlikely that the augmented ETI displayed in this study is directly due to an increase in stroke volume.

The effect which an augmented afterload may have on the ETI is not clear. It is suggested by this investigator that an increase in the contractile state of the myocardium induced by an increased afterload, as experienced during static exertion, may be reflected in an increased ETI. Changes in PEP, ICP and PEP/LVET during submaximal and maximal isometric work suggest that the contractile state of the myocardium was augmented in both environmental conditions. Similarly, increases in ETI from rest to 100% MVC were not statistically different between the control and heated environment. Therefore both the ETI and the contractile state of the myocardium were not affected by the heat. A major contributor to the increase in the ETI during the IHG contractions may have been the greater sympathetic and adrenergic stimulation of the heart.

Although it is unlikely that alterations in SV were associated with the increases found for the ETI the results of this investigation cannot confirm this. It is also possible that the ETI is determined by changes in other physiological variables which were not detected in this study.

Diastole

The data representing diastole related functions of the cardiac cycle have been presented here for the purpose of supporting and comparing changes in other variables. As expected both diastole and CT were significantly reduced with IHG in both environments. These changes closely paralleled those for HR.

The correlative data express extremely high association between CT, diastole and HR. In fact HR has higher correlations for diastole and CT than it does for LVET. The only puzzling difference between changes in diastole and CT with those of HR is that HR displays a non-significant EC effect while diastole and CT do not. That is to say that while the effects of heat stress and static exertion seem to be additive with changes in HR they are interactive with changes in diastole and CT. This discrepancy is further supported by the post-hoc analysis which shows that both diastole and CT are not significantly different between the environments at 50% MVC and 100% MVC. Such was not the case for HR.

The cause of this inconsistency can be explained by the relationship between CT and HR. The transformation from cardiac periods into heart rates is recognized as being non-linear; that is, a constant increase in the R-R interval, at different cycle lengths, does not result in a constant linear decrease in HR. This characteristic has been explored by Khachaturian et al. (34) in newborn infants and Jennings et al. (27) in healthy adults. Their results suggest that transformation of CT into HR introduces errors, which are reflected in the mean, variance and degree of skewness of the basic data.

Sympathoadrenergic Activity

The three variables which most closely represent the contractile state of the myocardium, which in turn is dependent upon sympathetic and adrenergic sources of stimulation to the heart tissue are ICP, PEP and PEP/LVET.

The overall tendency for both ICP and PEP was to decrease from rest to 100% MVC. However, in one condition, 50% MVC in a control environment, ICP showed a non-significant increase from rest. This abrupt change was incorporated into the PEP such that the PEP showed a plateau effect from rest to 50% MVC. Quarry and Spodick (65) found that ICP and PEP decreased during the first thirty seconds of a 50% MVC contraction but then returned to slightly below control levels at one minute. It is conceivable that since the data collection for this investigation took place during the final stage of the IHG, initial decreases in ICP and PEP were not detected.

The results of ANOVA and Newman-Keuls for environmental effect demonstrate that there was no change in ICP and PEP between the control and heated conditions. This lack of change was also present for ICP at 50% MVC. It is suggested that the contractile state of the myocardium is not significantly altered by thermoregulatory processes at rest and during static work of the forearm.

The overall tendency for ICP and PEP to decrease, especially from 50% MVC to 100% MVC, suggests that the contractility of the heart increased. A decrease or little change in ICP and PEP during IHG has been previously recorded (42, 49, 65). Martin et al. (48, 49), Metzger et al. (54) and Talley et al. (78) all found a significant inverse relation between PEP or ICP

to the rate of tension developed in the left ventricle (dp/dt). It is suggested that during a state of increased afterload (BPd) an insignificant change or decrease in ICP or PEP signifies increased myocardial contractility. When the left ventricle is confronted with a greater system pressure load, the myocardial tissue must isometrically contract with increased power in order to substantially augment the internal pressure of the ventricle before the aortic valve will open. The rise in dp/dt against a large afterload can only be achieved if the contractile state of the myocardium is increased.

No change or a decrease in PEP/LVET has been associated with augmented myocardial contractility (19, 21). In this study it was found that PEP/LVET does not significantly change during IHG. There was also no change in PEP/LVET between the two environments.

The above results suggest that the contractile state of the myocardium was augmented during bouts of isometric contraction of the forearm which is in support of Hypothesis 11. However the contractile state of the heart was not significantly affected by heat in any of the workload conditions.

Electromechanical Lag

EML is indicative of the amount of time which the left ventricle spends in electrical preparation to commence the isometric phase of systole. The initial ANOVA results indicate a linear decrease in EML from rest to 100% MVC which support the findings of Quarry and Spodick (65). Subsequent post-hoc analysis revealed that EML did not significantly change during IHG exercise in either of the environmental conditions. The reason for this

inconsistency is due to difference in the statistical methods used for analysis. It is suggested that EML has a tendency to become reduced with static exertion but that upon further analysis by a rigorous post-hoc method, no significant change in EML can be found between each of the six experimental conditions.

The absence of a significant environmental effect shows that the increased tachycardia induced by thermoregulation did not affect the electrical functions in the left ventricle. However Table 25 indicates that EML was significantly ($p < .05$) correlated to HR in an inverse fashion and to CT directly. This implies that alterations in EML were related to changes in the rate of myocardial contraction. It may be that the tachycardia induced by IHG was substantial enough to promote this relationship between rate of contraction and CT and that a further increase in HR due to heat stress did not add further weight to the relationship.

Myocardial Oxygen Consumption

Myocardial oxygen consumption as reflected by the index TRIP increased with submaximal and maximal IHG contractions in both environmental conditions. This increase in myocardial oxygen consumption supports Hypothesis 1 and is in agreement with earlier findings (35, 49, 59).

Both the ANOVA and post-hoc analysis confirm that myocardial oxygen consumption as depicted by TRIP did not significantly change throughout all workloads from a control to a heated environment. Therefore it seems that heat stress did not affect the myocardial oxygen consumption in addition to those changes elicited by a pressure load during IHG. In fact during submaximal and maximal isometric work TRIP was insignificantly less in the heated condition. This suggests that an afterload determines the extent of myocardial oxygen consumption to a greater degree than does HR during a simultaneous subjection of thermal and isometric stress.

The Interaction (EC) Effect

One of the major concerns in this study was to investigate how the physiological adjustments to isometric work and thermoregulatory processes would interact to produce the final alterations in BP, TRIP, HR and the STI. It was hypothesized that all variables would react to these two quite dissimilar adjustments in an interactive rather than additive manner. However, the results showed that the variables were not affected in a similar fashion. Some displayed a significant interaction while others did not which is not in support of Hypothesis 6.

The fourteen variables can be grouped as follows:

Interactive	Non-Interactive
BPs; BPd	HR
ETI	LVET; MS; TS
CT; diastole	PEP; ICP; PEP/LVET
TRIP	EML

These results simply suggest that in the case of a significant interaction effect the complete change in a variable elicited by isometric stress was partially masked by further change in that variable elicited by thermoregulation. With a non-interactive effect of course the opposite is true. That is, the changes elicited in any variable by isometric and heat stress were additive and represented in the total result.

SUMMARY

Increases in BPs and BPd during isometric work support Hypothesis 5. The pressor response from rest to 100% MVC was present in both environmental conditions. However the magnitude of the pressor response was less during both submaximal and maximal IHG contraction in the heat. Hypothesis 4 was partially supported by a non-significant difference in BPs and BPd at rest, between the two environmental conditions. During simultaneous subjection of the human body to static muscular exertion and heat stress an interaction occurs between the medullary control of blood pressure and the thermoregulatory adjustments directed from the hypothalamus, the final result being that increase in blood pressure during IHG contraction in the heat is diminished by the thermoregulatory increase in cutaneous vasodilation.

The contractility of the myocardium was augmented during submaximal and maximal static exertion in support of Hypothesis 11. The increased afterload induced by the IHG contractions is associated with greater sympathetic stimulation of the heart which causes the myocardial tissue to contract with more power. Such an alteration is needed if the left ventricle is to eject the same amount of blood into the system against an increased arterial pressure. However the contractile state of the myocardium was not significantly affected by the increased volume load produced by tachycardia during thermoregulation in the heat. This suggests that during a bout of isometric exercise in a heated environment, where the skin temperature may rise to 40°-41°C, the volume load imposed onto the heart by thermoregulatory processes plays a minor role in augmenting the contractile state of the myocardium. The major determinant of increased myocardial contractility seems to be the afterload elicited by the pressor response to isometric exercise.

Hypothesis 1 is supported by a significant increase in TRIP from rest to 100% MVC in both environmental conditions. Hypotheses 2 and 3 were not supported as TRIP was found not to change significantly throughout all workloads from a control to a heated environment. It is well known that HR is determined primarily by the balance between the inhibitory effects on the pacemaker of the vagus nerves and the excitatory effects of the release of norepinephrine by the sympathetic nerves. At rest, heat induced tachycardia caused a non-significant increase in TRIP when compared to the control condition. In contrast TRIP was non-significantly less during isometric work in the heat when compared to the same workloads

in a control temperature even though heart rates were significantly greater during static work in the heat. The reason for this reverse change was that BPs was considerably greater throughout all workloads in the neutral environment. Consequently the increase in TRIP produced by tachycardia was masked by the lower BPs in the heat. This suggests that the myocardial oxygen requirements during a bout of static exercise in a heated environment are largely determined by the afterload imposed upon the ventricles rather than the volume load produced by thermoregulatory processes.

Not all of the variables were altered during isometric work in the heat in an interactive manner as was proposed in Hypothesis 6. Of the systolic time intervals only CT and diastole demonstrated interactive characteristics. Of the non systolic variables only HR demonstrated additive characteristics. These results suggest that the physiological adjustments to isometric stress and body heating induce opposite changes in some variables and similar changes in others.

Changes in systole (LVET; TS; MS), diastole (diastole; CT), ETI and EML were mainly incorporated into this study to supply comparative data. The results show that Hypotheses 7-10 are clearly supported. Both diastolic and systolic related intervals significantly decreased as the HR increased with isometric work and body heating. The EML decreased in association with a rise in the inotropic state of the myocardium. LVET "corrected" for HR showed a steady increase with submaximal and maximal static work. The factor related to the rise in ETI was hypothesized to be either an augmented stroke volume or increased myocardial contractility. However, the available data could not confirm this theory.

CHAPTER V

SUMMARY AND CONCLUSIONS

The purpose of this study was to investigate the changes in cardiovascular dynamics as depicted by systolic time intervals, blood pressure, and heart rate during submaximal and maximal isometric work in a hot environment. A major concern was to determine whether these dynamic changes were additive or interactive in nature when the cardiovascular system was subjected to an augmented pressure load and volume load simultaneously. A minor concern was to support the findings of previous studies dealing with the alterations in systolic time intervals, HR and BP during sustained exertion of the forearm.

Fourteen male volunteers aged 20 to 31 without any previous history of cardiovascular ailments were used as subjects. Simultaneous recordings of the phonocardiogram, electrocardiogram, carotid pulse wave and blood pressure were conducted for each subject in a seated position during rest and static handgrip contractions of 50% MVC and 100% MVC. The subjects were tested in room temperature and in a sauna where the skin temperature was raised to 40°C - 41°C. The workloads were randomly rotated for each volunteer and the same rotational order was used in both environments. Testing took place on two separate days with one day of rest in between. Half of the subjects experienced the heated conditions first and room temperature conditions on the last day of testing. The reverse procedure was used for the remaining subjects. This balanced any day's effect in the results.

The data from two of the subjects were discarded because of poor quality reproduction of the time interval recordings. This improved the overall quality of the data. Of the twelve recordings used only the three clearest cycles closest to the termination of the contraction period were used for statistical analysis.

The experimental testing consisted of a 2 x 3 factorial design with repeated measures on both variables. The data were treated with a two-way ANOVA for each dependent variable. The statistical outputs included means, standard deviations, an ANOVA table for each dependent variable and correlation coefficients. In some cases a post-hoc analysis (Newman-Keuls method) was used to determine specific differences between workload or environment effects.

The fourteen dependent variables studied were divided into the following groups:

a) Systole related variables

left ventricular ejection time (LVET)

mechanical systole (MS)

total systole (TS)

ejection time index (ETI)

b) Diastole related variables

cycle time (CT)

diastole (DIAS)

c) Sympathoadrenergic Activity (Contractility)

pre-ejection period (PEP)

isovolumetric contraction period (ICP)

PEP/LVET (ratio)

d) Afterload

systolic blood pressure (BPs)

diastolic blood pressure (BPd)

e) Electromechanical Lag (EML)

f) Heart Rate (HR)

g) Myocardial Oxygen Consumption (Index)

triple product (TRIP)

CONCLUSIONS

1. The oxygen consumption of the myocardium as depicted by the triple product significantly increased during submaximal and maximal isometric handgrip contraction. This increase was evident at room temperature and during body heating.
2. There was no significant change in the myocardial oxygen consumption as depicted by TRIP at rest or during isometric forearm contraction between the control and heated environments. This suggests that the heat stress did not significantly increase the myocardial oxygen requirements.
3. In a state of rest, increasing the skin temperature to between 40°C - 41°C did not significantly alter either BPs or BPd when compared to a resting state at room temperature. However, BPs and BPd were substantially lower during isometric work in the heat than during isometric work at room temperature.

4. BPs and BPd significantly increased during 50% MVC and 100% MVC static contractions of the forearm. This increase was demonstrated in both environmental conditions.
5. All variables depicting changes in left ventricular systole (LVET; MS; TS) and ventricular diastole (diastole and CT) were found to become significantly reduced with submaximal and maximal static contractions of the forearm. These changes were evident in both environments.
6. A strong inverse correlation was found between HR and LVET, CT and diastole. HR significantly increased from rest to 100% MVC in both environmental conditions. Consequently, it is suggested that alterations in LVET, CT and diastole are largely determined by the rate of myocardial contraction.
7. The ejection time index significantly increased in both environmental conditions with a 50% MVC and 100% MVC static contraction of the forearm.
8. The electromechanical lag showed a general tendency to decrease during an isometric handgrip contraction. However, subsequent post-hoc analysis (Newman-Keuls) demonstrated that EML did not significantly decrease during a submaximal or maximal isometric contraction of the forearm. It is suggested that care be taken to choose a proper statistical procedure for analysis of EML.
9. The contractility of the heart as depicted by changes in ICP, PEP and PEP/LVET increases in response to a pressure load produced by static exertion but is not significantly altered by an augmented volume load associated with heat stress.

10. HR, LVET, MS, TS, PEP, ICP, PEP/LVET and EML changed in an additive fashion from rest to 100% MVC during subjection to a volume load and pressure load simultaneously.

In contrast alterations in BPs, BPd, ETI, CT, diastole, and TRIP displayed interactive characteristics during the same test conditions.

REFERENCES

1. Agress, C.M., and Wegner, S. "The determinants of left ventricular isovolumetric contraction time," Japanese Heart Journal, 9: 169-179, 1968.

2. _____. "Determination of stroke volume from left ventricular isovolumetric contraction and ejection times, " Japanese Heart Journal, 9: 339-348, 1968.

3. Ahmed, S.S., Levinson, G.E., Schwartz, C.J., and Ettinger, P.O. "Comparison of systolic time intervals with direct measures of myocardial contractility in man," Clinical Research, 18: 294, 1970.

4. Badeer, H.S. "Contractile tension in the myocardium," American Heart Journal, 66: 432-434, 1963.

5. Braunwald, E., Sarnoff, S.J., and Stainsby, W.N. "Determinants of duration and mean rate of ventricular ejection," Circulation Research, 6: 319-325, 1958.

6. Burch, G.E., and Giles, T.D. "The burden of a hot and humid environment on the heart," Modern Concepts of Cardiovascular Disease, 39: 115-120, 1970.

7. Cerretelli, P., Veicsteinas, A., Fumagalli, M., and Dell'orto, L. "Energetics of isometric exercise in man," Journal of Applied Physiology, 41: 136-141, 1976.

8. Coutts, K.D. "Cardiac intervals during static muscular contraction with varied respiratory maneuvers," Application of Science and Medicine to Sport, C.C. Thomas Publisher, 78-80, 1975.

9. Damato, A.N., Lau, S.H., Stein, E., Haft, J.I., Kosowsky, B., and Cohen, S.I. "Cardiovascular responses to acute thermal stress (hot dry environment) in unacclimatized normal subjects," American Heart Journal, 76: 769-774, 1968.

10. Detry, J.R., Brengelmann, G.L., Rowell, L.B., and Wyss, C. "Skin and muscle components of forearm blood flow in directly heated resting man," Journal of Applied Physiology, 32: 506-511, 1972.

11. Dowell, R.T., Cutilletta, A.F., Rudnik, M.A., and Sodt, P.C. "Heart functional responses to pressure overload in exercised and sedentary rats," American Journal of Physiology, 230: 199-204, 1976.

12. Downey, J.M. "Myocardial contractile force as a function of coronary blood flow," American Journal of Physiology, 230: 1-6, 1976.

13. Fisher, M.L., Nutter, D.O., Jacobs, W., Schlant, R.C. "Haemodynamic responses to isometric exercise (handgrip) in patients with heart disease," British Heart Journal, 35: 422-432, 1973.

14. Flessas, A.P., Connelly, G.P., Hunda, S., Tilney, C.R., Kloster, C.K., Rimmer, R.H., Keefe, J.F., Klein, M.D., and Ryan, T.J. "Effects of isometric exercise on the end-diastolic pressure, volumes, and function of the left ventricle in man," Circulation, 53: 839-846, 1976.

15. Frank, M.N., and Haberern, N. "The effects of hand grip and exercise on systolic time intervals in human subjects," The American Journal of Medical Sciences, 261: 219-222, 1971.

16. Flessas, A.P., Kumar, S., and Spodick, D.H. "Effects of Valsalva maneuver on the cardiac systemic intervals:Beat-to-beat versus timed analysis," American Heart Journal, 80: 522-531, 1970.

17. Frank, M.N., and Kinlaw, W.B. "Indirect measurement of isovolumetric contraction time and tension period in normal subjects," The American Journal of Cardiology, 10: 800-806, 1962.

18. Franks, B.D., Wiley, J.F., and Cureton Jr., T.K. "Orthogonal factors and norms for time components of the left ventricle," Medicine and Science in Sports, 1: 171-176, 1969.

19. Garrard Jr., C.L., Weissler, A.M., and Dodge, H.T. "The relationship of alterations in systolic time intervals to ejection fraction in patients with cardiac disease," Circulation, 42: 455-462, 1970.

20. Gorlin, R., Knowles, J.H., and Clifford, F.S. "The Valsalva maneuver as a test of cardiac function - pathologic physiology and clinical significance," American Journal of Medicine, 22: 197-212, 1957.

21. Grossman, W., McLaurin, L.P., Saltz, S.B., Paraskos, J.A., Dalen, J.E., and Dexter, L. "Change in the inotropic state of the left ventricle during isometric exercise," British Heart Journal, 35: 697-704, 1973.

22. Harris, W.S., Schoenfeld, C.D., and Weissler, A.M. "Effects of adrenergic receptor activation and blockage on the systolic pre-ejection period, heart rate, and arterial pressure in man," The Journal of Clinical Investigation, 46: 1704-1714, 1967.

23. Heikkila, J., Luomanmäki, K., and Pyörälä, K. "Serial observations on left ventricular dysfunctions in acute myocardial infarction - II systolic time intervals in power failure," Circulation, 44: 343-354, 1971.

24. Heistad, D.D., Arboud, F.M., Mark, A.L., and Schmid, P.G. "Interaction of thermal and baroreceptor reflexes in man," Journal of Applied Physiology, 35: 581-586, 1973.

25. Helfant, R.H., De Villa, M.A., Meister, S.G. "Effects of sustained handgrip exercise on left ventricular performance," Circulation, 44: 982-993, 1971.

26. Hume, L., Irving, J.B., Kitchin, A.H., and Reuben, S.R. "Effects of sustained isometric handgrip on praecordial accelerocardiogram in normal subjects and in patients with heart disease," British Heart Journal, 37: 873-881, 1975.

27. Jennings, J.R., Stringfellow, J.C., and Graham, M. "A comparison of the statistical distributions of beat-by-beat heart rate and heart period," Psychophysiology, 11: 207-210, 1974.

28. Jezek, V. "Clinical value of the polygraphic tracing in the study of the sequence of events during contraction," Cardiologia, 43: 298-316, 1963.

29. Johnson, J.M., Neiderberger, M., Rowell, L.B., Eisman, M.M., and Brengelmann, G.L. "Competition between cutaneous vasodilator and vasoconstrictor reflexes in man," Journal of Applied Physiology, 25: 798-803, 1973.

30. Jones, W.B., and Foster, G.L. "Determinants of duration of left ventricular ejection in normal young men," Journal of Applied Physiology, 19: 279-283, 1964.

31. Katz, L.N., and Feil, H.S. "Clinical observations on the dynamics of ventricular systole," Archives of Internal Medicine, 32: 672-692, 1923.

32. Katz, L.N., and Feinberg, H. "The relation of cardiac effort to myocardial oxygen consumption and coronary flow," Circulation Research, 6: 656-669, 1958.

33. Kerber, R.E., Miller, R.A., and Najjar, S.M. "Myocardial ischemic effects of isometric, dynamic, and combined exercise in coronary artery disease," Chest, 4: 388-394, 1975.

34. Khachaturian, Z.S., and Kerr, J. "A methodological note: comparison between period and rate data in studies of cardiac function," Psychophysiology, 9: 539-545, 1972.

35. Kivowitz, C., Parmley, W.W., Donoso, R., Marcus, H., Ganz, W., and Swan, H.J.C. "Effects of isometric exercise on cardiac performance - the grip test," Circulation, 44: 994-1002, 1971.

36. Kumar, S., and Spodick, D.H. "Study of the mechanical events of the left ventricle by atraumatic techniques: comparison of methods of measurement and their significance," American Heart Journal, 80: 402-413, 1970.

37. Lind, A.R. "Cardiovascular responses to static exercise (Isometrics, Anyone?)," Circulation, 41: 173-176, 1970.

38. Lind, A.R., and McNicol, G.W. "Local and central circulatory responses to sustained contractions and the effect of free or restricted arterial inflow on post-exercise hyperaemia," Journal of Physiology, 192: 575-593, 1967.

39. _____. "Circulatory responses to sustained handgrip contractions performed during other exercise, both rhythmic and static," Journal of Physiology, 192: 595-607, 1967.

40. Lind, A.R., McNicol, G.W., and Donald, K.W. "Circulatory adjustments to sustained (static) muscular activity," Physical Activity in Health and Disease, edited by K.Evang, and K.L. Andersen. Oslo, Universitetsforlager. 38-63, 1966.

41. Lind, A.R., Taylor, S.H., Humphreys, P.W., Kennelly, B.M., and Donald, K.W. "The circulatory effects of sustained voluntary muscle contraction," Clinical Science, 27: 229-244, 1964.

42. Lindquist, V.A.Y., Spangler, R.D., and Blount, S.G. "A comparison between the effects of dynamic and isometric exercise as evaluated by the systolic time intervals in normal man," American Heart Journal, 85: 227-236, 1973.

43. Lombard, W.P., and Cope, O.M. "Sex-differences in heart action," American Journal of Physiology, 83: 37-41, 1927.

44. Ludbrook, P., Karliner, J.S., Orourke, D.A. "Effects of submaximal isometric handgrip on left ventricular size and wall motion," The American Journal of Cardiology, 33: 30-36, 1974.

45. Lynch, W.C., Schuri, U., and D'Anna, J. "Effects of isometric muscle tension on vasomotor activity and heart rate," Psychophysiology, 13: 222-230, 1976.

46. MacGregor, D.C., Covell, J.W., Mahler, F., Dilley, R.B., and Ross, J. "Relations between afterload, stroke volume, and descending limb of Starling's curve," American Journal of Physiology, 227: 884-890, 1974.

47. Margolis, C. "The significance of ejection period/tension period as a factor in the assessment of cardiac function and as a possible diagnostic tool for the uncovering of silent coronary heart disease - a study of III cases," Diseases of the Chest, 46: 706-713, 1964.

48. Martin, C.E., Shaver, J.A., Thompson, M.E., Reddy, P.S., and Leonard, J.J. "Direct correlation of external systolic time intervals with interval indices of left ventricular function in man," Circulation, 44: 419-431, 1971.

49. _____. "Autonomic mechanisms in hemodynamic responses to isometric exercise," The Journal of Clinical Investigation, 54: 104-115, 1974.

50. Mason, D.T. "Usefulness and limitations of the rates of rise of intraventricular pressure (dp/dt) in the evaluation of myocardial contractility in man," The American Journal of Cardiology, 23: 516-527, 1969.

51. Matsura, T., and Goodyer, V.N. "Effects of a pressure load on left ventricular systole time intervals," American Journal of Physiology, 224: 80-85, 1973.

52. McDonald, I.G. and Hobson, E.R. "A comparison of the relative value of non-invasive techniques - echocardiography, systolic time intervals, and apexcardiography - in the diagnosis of primary myocardial disease," American Heart Journal, 88: 454-462, 1974.

53. Meerson, F.Z., and Kapelko, V.I. "The significance of the interrelationship between the intensity of the contractile state and the velocity of relaxation in adapting cardiac muscle to function at high work loads," Journal of Molecular and Cellular Cardiology, 7: 793-806, 1975.

54. Metzger, C.C., Chough, C.B., Kroetz, F.W., and Leonard, J.J. "True isovolumic contraction time - Its correlation with two external indexes of ventricular performance," The American Journal of Cardiology, 25: 434-442, 1970.

55. Mitchell, J.H. Wallace, A.G., and Skinner, N.S. "Relation between end-diastolic pressure and mean rate of ejection of left ventricle," American Journal of Physiology, 211: 83-86, 1966.

56. Mitchell, J.H., and Wildenthal, K. "Static (isometric) exercise and the heart: Physiological and clinical considerations," Annual Review of Medicine, 25: 369-381, 1974.

57. Moreyra, A.E., Lardani, H., Lardani, M.V., and Cingolani, H.E. "The estimation of contractile element velocity during the isovolumic period: alterations by cardiac output and contractile state," Archives Internationales de Physiologie et de Biochimie, 82: 881-894, 1975.

58. Mullins, C.B., Leshin, S.J., Mierzwiak, D.S., Matthews, O.A., and Blomquist, G. "Sustained forearm contraction (hand-grip) as a stress test for evaluation of left ventricular function," Clinical Research, 18: 322, 1970.

59. Nakhjavan, F.K., Natarajan, G., Smith, A.M., Dratch, M., and Goldberg, H. "Myocardial lactate metabolism during isometric handgrip test: comparison with pacing tachycardia," British Heart Journal, 37: 79-84, 1975.

60. Nielsen, B. "Thermoregulation during static work with the legs," Acta Physiologica Scandanavia, 95: 457-462, 1975.

61. Nutter, D.O., Schlant, R.C., and Hurst, J.W. "Isometric exercise and the cardiovascular system," Modern Concepts of Cardiovascular Disease, 41: 11-15, 1972.

62. Pandolf, K.B., Gonzalez, R.R., and Pharo, G. "Physiological strain during light exercise in hot-humid environments," Aerospace Medicine, 45: 359-365, 1974.

63. Payne, R.M., Horwitz, L.D., and Mullins, C.B. "Comparison of isometric exercise and angiotension infusion as stress test for evaluation of left ventricular function," The American Journal of Cardiology, 31: 428-433, 1973.

64. Pettigrew, R.T., Galt, J.M., Ludgate, C.M., Horn, D.B., and Smith, A.N. "Circulatory and biochemical effects of whole body hyperthermia," British Journal of Surgery, 61: 727-730, 1974.

65. Quarry, V.M., and Spodick, D.H. "Cardiac responses to isometric exercise: comparative effects with different postures and levels of exertion," Circulation, 49: 905-920, 1974.

66. Raab, W., De Paula e Silva, P., and Starcheska, Y.K. "Adrenergic and cholinergic influences on the dynamic cycle of the normal human heart," Cardiologia, 33: 350-364, 1958.

67. Reeves, T.J., Lloyd, L.H., Jones, W.B., Coghlan, C., Prieto, G., and Carroll, J. "The hemodynamic determinants of the rate of change in pressure in the left ventricle during isometric contraction," American Heart Journal, 60: 745-761, 1960.

68. Robinson, B.F. "Relation of heart rate and systolic blood pressure to the onset of pain in angina pectoris," Circulation, 35: 1073-1083, 1967.

69. Rowell, L.B. "Human cardiovascular adjustments to exercise and thermal stress," Physiological Reviews, 54: 75-159, 1974.

70. Saltz, S., Grossman, W., Paraskos, J., Segal, H., Dalen, J., and Dexter, L. "Left ventricular function: response to sustained isometric hand grip," Circulation volume 43 and 44, Supplement II - 34, 1971.

71. Sambhi, M.P. "The isometric period of contraction as a determinant of cardiac performance and digitalis action," The American Journal of Cardiology, 6: 1042-1048, 1960.

72. Shaver, J.A., Kroetz, F.W., Leonard, J.J., and Paley, H.W. "The effects of steady-state increases in systemic arterial pressure on the duration of left ventricular ejection time," The Journal of Clinical Investigation, 47: 217-230, 1968.

73. Simaan, J. and Fawaz, G. "Myocardial oxygen consumption in isovolumic hearts with varying cardiac outputs," Cardiovascular Research, 8: 762-768, 1974.

74. Sonnenblick, E.H., Parmley, W.W., and Urschell, C.W. "The contractile state of the heart as expressed by force-velocity relations," The American Journal of Cardiology, 23: 488-503, 1969.

75. Stefadouros, M.A., Grossman, W., Shahawy, M.E., Witham, A.C. "The effect of isometric exercise on the left ventricular volume in normal man," Circulation, 44: 1185-1189, 1974.

76. _____. "Noninvasive study of effect of isometric exercise on left ventricular performance in normal man," British Heart Journal, 36: 988-995, 1974.

77. Sutton, R., Hood, MB/W.P., and Craig, E. "Comparison of noninvasive and invasive measurements of left ventricular performance," The American Journal of Cardiology, 25: 132, 1970.

78. Talley, R.C., Meyer, J.F., and McNay, J.L. "Evaluation of the pre-ejection period as an estimate of myocardial contractility in dogs," The American Journal of Cardiology, 27: 384-391, 1971.

79. Veicsteinas, A., Dell'orto, L., Fumagalli, M., and Cerretelli, P. "Cardiovascular and metabolic readjustment to isometric contraction in man," Archivo di Fisiologia, 70: 96-98, 1973.

80. Wallace, A.G., Mitchell, J.H., Skinner, N.S., and Sarnoff, S.J. "Duration of the phases of left ventricular systole," Circulation Research, 12: 611-619, 1963.

81. Weissler, A.M., Harris, L.C., and White, G.D. "Left ventricular ejection time index in man," Journal of Applied Physiology, 18: 919-923, 1963.

82. Weissler, A.M., Harris, L.C., and Schoenfeld, C.D. "Systolic time intervals in heart failure in man," Circulation, 37: 149-159, 1968.

83. _____. "Bedside technics for the evaluation of ventricular function in man," The American Journal of Cardiology, 23: 577-583, 1969.

84. Whitsett, T.L., and Naughton, J. "The effects of exercise on systolic time intervals in sedentary and active individuals and rehabilitated patients with heart disease," The American Journal of Cardiology, 27: 352-358, 1971.

85. Wiley, J.F. "Effects of body position and exercise on left ventricular intervals," Medicine and Science in Sports, 2: 132-136, 1970.

86. Wiggers, C.J. "Studies on the consecutive phases of the cardiac cycle. I the duration of the consecutive phases of the cardiac cycle and the criteria for their precise determination," American Journal of Physiology, 56: 415-438, 1922.

87. _____. "Studies of the consecutive phases of the cardiac cycle. II the laws governing the durations of ventricular systole and diastole," American Journal of Physiology, 56: 439-458, 1922.

88. Zacharoulis, A.A., Evans, T.R., Ziady, G.M., Coltart, D.J., and Shillingford, J.P. "Measurement of stroke volume from pulmonary artery pressure record in man," British Heart Journal, 37: 20-25, 1975.

APPENDICES

APPENDIX A

Raw Scores - Subjects Tested in Neutral Environment First

Subjects	Condition	Variables													
		BPs	BPd	CT	EML	ET	MS	TS	DIA	PEP	ICP	PEP/LVET	HR	TRIP	ETI
S_1	N_R	115.	70.	840.	83.	253.	297.	380.	463.	127.	43.	0.501	71.	2081.0	374.8
	N_{50}	155.	105.	720.	70.	270.	320.	400.	373.	130.	60.	0.481	83.	3487.5	411.7
	N_{100}	155.	95.	650.	70.	250.	280.	350.	300.	100.	30.	0.400	92.	3576.9	406.9
	H_R	110.	60.	710.	90.	213.	253.	343.	357.	130.	40.	0.611	85.	1983.1	357.0
	H_{50}	140.	100.	730.	83.	223.	273.	347.	370.	123.	40.	0.553	82.	2569.9	363.1
	H_{100}	145.	110.	600.	73.	210.	253.	330.	263.	120.	47.	0.571	100.	3045.0	380.0
S_2	N_R	110.	80.	1013.	70.	240.	310.	380.	620.	140.	70.	0.583	59.	1563.2	340.7
	N_{50}	145.	115.	813.	87.	230.	293.	380.	433.	150.	63.	0.654	74.	2460.2	355.4
	N_{100}	160.	130.	607.	70.	213.	270.	340.	267.	127.	57.	0.595	99.	3375.8	381.5
	H_R	110.	65.	803.	70.	237.	287.	360.	450.	123.	53.	0.522	75.	1944.4	363.6
	H_{50}	140.	97.	660.	73.	217.	273.	350.	323.	133.	60.	0.616	91.	2757.6	371.2
	H_{100}	155.	110.	550.	67.	210.	257.	327.	227.	117.	50.	0.556	109.	3550.9	395.5
S_3	N_R	115.	80.	817.	100.	237.	297.	400.	420.	163.	63.	0.691	73.	1999.6	361.6
	N_{50}	150.	110.	627.	77.	237.	283.	360.	267.	123.	47.	0.522	96.	3398.9	399.4
	N_{100}	155.	127.	590.	77.	243.	277.	353.	237.	110.	33.	0.452	102.	3835.6	416.2
	H_R	120.	80.	800.	97.	240.	270.	370.	433.	130.	33.	0.542	75.	2160.0	367.5
	H_{50}	145.	85.	637.	83.	240.	267.	350.	307.	110.	27.	0.458	94.	3279.6	400.2
	H_{100}	145.	90.	517.	80.	217.	227.	307.	213.	90.	10.	0.418	116.	3648.4	414.1
S_4	N_R	135.	75.	910.	87.	260.	270.	350.	547.	90.	3.	0.346	66.	2314.3	372.1
	N_{50}	150.	110.	647.	70.	240.	283.	350.	310.	110.	40.	0.458	93.	3340.2	397.7
	N_{100}	150.	115.	583.	70.	240.	270.	340.	237.	100.	30.	0.417	103.	3702.9	414.9
	H_R	125.	75.	883.	63.	253.	310.	373.	510.	120.	57.	0.474	68.	2150.9	368.8
	H_{50}	140.	110.	773.	90.	247.	277.	367.	407.	120.	30.	0.487	78.	2679.3	378.6
	H_{100}	150.	110.	560.	57.	217.	257.	317.	247.	100.	43.	0.462	107.	3482.1	398.8
S_5	N_R	100.	75.	1167.	97.	270.	303.	400.	763.	130.	33.	0.482	51.	1388.6	357.4
	N_{50}	125.	105.	1067.	100.	277.	313.	413.	650.	137.	37.	0.495	56.	1945.3	372.3
	N_{100}	135.	105.	783.	98.	237.	285.	382.	397.	147.	50.	0.620	77.	2447.2	366.9
	H_R	92.	70.	970.	83.	270.	304.	387.	583.	117.	34.	0.432	62.	1536.5	375.2
	H_{50}	100.	80.	837.	77.	253.	293.	370.	470.	117.	40.	0.461	72.	1816.7	375.2
	H_{100}	115.	90.	753.	80.	243.	270.	353.	397.	110.	30.	0.453	80.	2228.8	378.7
S_6	N_R	105.	80.	903.	67.	263.	320.	387.	517.	123.	57.	0.469	66.	1836.5	376.2
	N_{50}	130.	97.	663.	57.	240.	303.	360.	397.	120.	63.	0.500	90.	2822.1	393.8
	N_{100}	150.	106.	543.	60.	250.	290.	350.	213.	100.	40.	0.400	110.	4141.1	437.7
	H_R	90.	65.	667.	60.	237.	283.	347.	310.	110.	50.	0.465	90.	1917.0	389.7
	H_{50}	120.	60.	657.	57.	233.	280.	330.	323.	97.	40.	0.415	91.	2558.4	388.7
	H_{100}	125.	75.	553.	60.	230.	270.	327.	220.	97.	37.	0.420	108.	3117.5	414.3

Raw Scores - Subjects Tested in Heated Environment First

Subjects	Condition	Variables													
		BPs	BPd	CT	EML	ET	MS	TS	DIA	PEP	ICP	PEP/LVET	HR	TRIP	ETI
S_7	N_R	100.	78.	1113.	83.	280.	327.	410.	707.	130.	47.	0.464	54.	1509.0	371.6
	N_{50}	130.	115.	720.	77.	247.	300.	377.	347.	130.	53.	0.527	83.	2672.2	388.3
	N_{100}	140.	115.	627.	80.	227.	273.	357.	270.	130.	50.	0.574	96.	3038.3	389.4
	H_R	100.	78.	653.	110.	230.	250.	360.	293.	130.	20.	0.565	92.	2112.2	386.1
	H_{50}	106.	92.	543.	70.	210.	260.	330.	217.	120.	50.	0.574	110.	2458.2	397.7
	H_{100}	98.	80.	537.	87.	213.	240.	323.	210.	110.	23.	0.516	112.	2337.4	403.4
S_8	N_R	105.	80.	970.	70.	277.	327.	400.	570.	123.	53.	0.446	62.	1796.9	381.8
	N_{50}	125.	96.	723.	70.	270.	320.	393.	330.	123.	53.	0.457	83.	2799.5	411.0
	N_{100}	120.	102.	660.	73.	263.	300.	373.	280.	110.	37.	0.418	91.	2872.7	417.9
	H_R	102.	82.	697.	73.	230.	287.	360.	337.	130.	57.	0.565	86.	2020.5	376.4
	H_{50}	120.	90.	603.	70.	223.	280.	353.	247.	130.	60.	0.585	99.	2665.2	392.4
	H_{100}	130.	96.	527.	67.	193.	243.	307.	220.	113.	47.	0.587	114.	2863.3	387.0
S_9	N_R	115.	75.	897.	83.	253.	287.	373.	523.	120.	37.	0.474	67.	1949.4	367.1
	N_{50}	160.	125.	687.	77.	243.	293.	367.	323.	123.	47.	0.507	87.	3401.9	391.9
	N_{100}	156.	125.	610.	77.	237.	270.	343.	260.	107.	30.	0.452	98.	3631.5	403.9
	H_R	105.	72.	793.	83.	253.	293.	380.	413.	127.	43.	0.501	76.	2011.8	381.9
	H_{50}	135.	95.	667.	70.	237.	280.	350.	313.	113.	43.	0.479	90.	2875.5	389.7
	H_{100}	136.	110.	617.	70.	227.	280.	350.	257.	123.	53.	0.545	97.	2999.4	392.1
S_{10}	N_R	110.	75.	733.	80.	220.	267.	347.	390.	127.	47.	0.576	82.	1980.0	359.1
	N_{50}	140.	108.	660.	77.	220.	277.	350.	310.	130.	53.	0.593	91.	2800.0	374.5
	N_{100}	145.	108.	610	80.	217.	270.	347.	263.	130.	50.	0.600	98.	3090.2	383.9
	H_R	96.	70.	647.	80.	213.	277.	357.	290.	143.	63.	0.673	93.	1900.2	371.1
	H_{50}	125.	102.	543.	70.	203.	260.	323.	220.	120.	50.	0.591	110.	2806.7	391.1
	H_{100}	134.	105.	537.	70.	200.	253.	323.	217.	123.	53.	0.619	112.	2996.3	390.1
S_{11}	N_R	118.	84.	1047.	90.	277.	320.	410.	640.	133.	43.	0.482	57.	1871.5	374.1
	N_{50}	145.	100.	803.	73.	270.	330.	400.	400.	130.	57.	0.481	75.	2924.1	397.0
	N_{100}	140.	100.	830.	80.	267.	303.	387.	450.	120.	40.	0.451	72.	2698.8	389.6
	H_R	110.	75.	873.	80.	247.	287.	370.	503.	123.	43.	0.501	69.	1864.1	363.5
	H_{50}	114.	88.	730.	87.	240.	287.	373.	353.	133.	47.	0.556	82.	2248.8	379.7
	H_{100}	135.	98.	630.	80.	237.	280.	360.	270.	123.	43.	0.522	95.	3042.9	398.6
S_{12}	N_R	114.	70.	1120.	90.	263.	307.	397.	727.	133.	43.	0.507	54.	1608.2	354.4
	N_{50}	118.	90.	1097.	100.	263.	307.	403.	690.	140.	40.	0.534	55.	1700.1	356.3
	N_{100}	116.	96.	860.	90.	253.	283.	373.	487.	120.	30.	0.476	70.	2050.2	371.9
	H_R	115.	70.	840	90.	240.	270.	360.	480.	120.	30.	0.500	71.	1971.4	361.4
	H_{50}	108.	70.	833.	93.	243.	267.	360.	473.	117.	23.	0.480	72.	1892.2	365.7
	H_{100}	114.	80.	730.	103.	247.	257.	367.	360.	120.	17.	0.487	82.	2311.2	386.4

APPENDIX B

Correlation Coefficients - At Rest in a Neutral Environment

	BPs	BPd	CT	EML	LVET	MS	TS	DIAS	PEP	ICP	PEP/LVET	HR	TRIP	ETI
BPs	1.000													
BPd	-0.154	1.000												
CT	-0.352	0.054	1.000											
EML	0.245	-0.213	0.179	1.000										
LVET	-0.204	0.196	0.731	0.004	1.000									
MS	-0.592	0.477	0.597	-0.281	0.695	1.000								
TS	-0.520	0.378	0.652	0.218	0.659	0.869	1.000							
DIAS	-0.320	-0.011	0.994	0.171	0.703	0.526	0.572	1.000						
PEP	-0.440	0.273	0.023	0.281	-0.265	0.340	0.551	-0.049	1.000					
ICP	-0.585	0.410	-0.085	-0.352	-0.257	0.510	0.403	-0.150	0.799	1.000				
PEP/LVET	-0.249	0.150	-0.314	0.224	-0.649	-0.041	0.143	-0.360	0.904	0.740	1.000			
HR	0.305	-0.110	-0.989	-0.127	-0.774	-0.644	-0.671	-0.976	0.001	0.076	0.352	1.000		
TRIP	0.794	-0.099	-0.779	0.489	-0.348	-0.447	-0.614	-0.765	-0.399	-0.380	-0.148	0.731	1.000	
ETI	0.081	0.168	-0.185	-0.080	0.528	0.247	0.146	-0.214	-0.395	-0.356	0.530	0.128	0.138	1.000

Correlation Coefficients - At 50% MVC in a Neutral Environment

	BPs	BPd	CT	EML	LVET	MS	TS	DIAS	PEP	ICP	PEP LVET	HR	TRIP	ETI
BPs	1.000													
BPd	0.681	1.000												
CT	-0.603	-0.428	1.000											
EML	-0.369	-0.039	0.851	1.000										
LVET	-0.335	-0.493	0.569	0.244	1.000									
MS	-0.294	-0.525	0.423	0.013	0.893	1.000								
TS	-0.406	-0.435	0.776	0.526	0.887	0.844	1.000							
DIAS	-0.569	-0.406	0.994	0.862	0.535	0.370	0.743	1.000						
PEP	-0.285	-0.057	0.650	0.699	0.107	0.201	0.554	0.642	1.000					
ICP	0.208	0.011	-0.447	-0.584	-0.227	0.198	-0.107	-0.470	0.171	1.000				
PEP/LVET	0.018	0.301	0.089	0.362	-0.627	-0.472	-0.197	0.105	0.706	0.310	1.000			
HR	0.581	0.401	-0.988	-0.818	-0.604	-0.506	-0.830	-0.976	-0.702	0.343	-0.106	1.000		
TRIP	0.837	0.470	-0.870	0.065	-0.271	-0.164	-0.529	-0.853	-0.663	-0.337	-0.315	0.857	1.000	
ETI	0.376	0.003	-0.637	-0.768	-0.564	0.344	-0.529	-0.562	-0.126	0.086	-0.751	0.616	0.582	1.000

Correlation Coefficients - At 100% MVC in a Neutral Environment

	BPs	BPd	CT	EML	LVET	MS	TS	DIAS	PEP	ICP	PEP/LVET	HR	TRIP	ETI
BPs	1.000													
BPd	0.631	1.000												
CT	-0.673	-0.582	1.000											
EML	-0.525	-0.211	0.744	1.000										
LVET	-0.474	-0.621	0.446	-0.036	1.000									
MS	-0.545	-0.623	0.500	0.039	0.838	1.000								
TS	-0.732	-0.605	0.846	0.632	0.611	0.790	1.000							
DIAS	-0.631	-0.579	0.993	0.711	0.418	0.461	0.798	1.000						
PEP	-0.290	0.022	0.454	0.765	-0.440	-0.057	0.442	0.431	1.000					
ICP	0.089	0.245	-0.057	0.162	-0.634	-0.124	0.041	-0.061	0.760	1.000				
PEP/LVET	-0.023	0.264	0.150	0.567	-0.743	-0.379	0.075	0.145	0.927	0.845	1.000			
HR	0.665	0.581	-0.994	-0.759	-0.426	-0.488	-0.845	-0.981	-0.476	0.039	-0.176	1.000		
TRIP	0.807	0.499	-0.891	0.165	-0.178	-0.278	-0.764	-0.867	-0.663	0.364	-0.422	0.940	1.000	
ETI	0.310	0.094	-0.666	-0.768	0.362	0.251	-0.385	-0.673	-0.848	-0.378	-0.780	0.689	0.581	1.000

Correlation Coefficients - At Rest in a Heated Environment

	BPs	BPd	CT	EML	LVET	MS	TS	DIAS	PEP	ICP	PEP/LVET	HR	TRIP	ETI
BPs	1.000													
BPd	0.220	1.000												
CT	0.366	0.052	1.000											
EML	0.073	0.257	-0.127	1.000										
LVET	0.074	0.218	0.840	-0.186	1.000									
MS	0.033	0.118	0.630	-0.718	0.721	1.000								
TS	0.234	0.441	0.758	0.053	0.854	0.651	1.000							
DIAS	0.396	0.021	0.997	-0.138	0.809	0.606	0.713	1.000						
PEP	0.053	0.246	-0.474	0.445	-0.636	-0.415	-0.142	-0.481	1.000					
ICP	-0.045	-0.123	-0.170	-0.813	-0.215	0.516	-0.160	-0.161	-0.160	1.000				
PEP/LVET	-0.041	-0.019	-0.706	0.334	-0.897	-0.606	-0.539	-0.693	0.907	0.226	1.000			
HR	-0.444	-0.063	-0.993	0.129	-0.821	-0.601	-0.734	-0.994	0.472	0.166	0.701	1.000		
TRIP	0.618	0.377	-0.412	0.159	-0.374	0.239	-0.291	-0.402	0.291	-0.272	0.311	0.336	1.000	
ETI	-0.637	0.236	-0.370	-0.089	0.181	0.077	0.095	-0.422	-0.207	-0.025	-0.227	0.412	-0.109	1.000

Correlation Coefficients - At 50% MVC in a Heated Environment

	BPs	BPd	CT	EML	LVET	MS	TS	DIAS	PEP	ICP	PEP/LVET	HR	TRIP	ETI
BPs	1.000													
BPd	0.517	1.000												
CT	-0.184	-0.233	1.000											
EML	0.090	0.241	0.639	1.000										
LVET	-0.105	-0.333	0.827	0.487	1.000									
MS	-0.205	-0.216	0.564	0.005	0.664	1.000								
TS	-0.090	0.042	0.791	0.699	0.792	0.676	1.000							
DIAS	-0.174	-0.279	0.991	0.614	0.812	-0.498	0.728	1.000						
PEP	0.008	0.566	-0.007	0.363	-0.260	0.064	0.383	-0.084	1.000					
ICP	-0.070	0.283	-0.579	-0.577	-0.656	0.066	-0.284	-0.627	0.549	1.000				
PEP/LVET	0.066	0.590	-0.473	-0.028	-0.745	-0.333	-0.184	-0.516	0.838	0.754	1.000			
HR	0.094	0.236	-0.990	-0.619	-0.843	-0.608	-0.805	-0.976	0.010	0.563	0.487	1.000		
TRIP	0.822	0.413	-0.648	-0.322	-0.364	0.245	-0.426	-0.633	-0.122	0.211	0.124	0.574	1.000	
ETI	0.015	-0.023	-0.741	-0.515	-0.261	-0.184	-0.465	-0.735	-0.325	0.167	-0.073	0.739	0.545	1.000

Correlation Coefficients - At 100% MVC in a Heated Environment

	BPs	BPd	CT	EML	LVET	MS	TS	DIAS	PEP	ICP	PEP/LVET	HR	TRIP	ETI
BPs	1.000													
BPd	0.766	1.000												
CT	-0.367	-0.179	1.000											
EML	-0.593	-0.487	0.554	1.000										
LVET	-0.348	-0.422	0.815	0.495	1.000									
MS	0.002	0.163	0.532	-0.155	0.572	1.000								
TS	-0.324	-0.127	0.870	0.554	0.831	0.723	1.000							
DIAS	-0.336	-0.174	0.981	0.522	0.755	0.416	0.763	1.000						
PEP	-0.060	0.404	0.352	0.261	0.011	0.451	0.565	0.249	1.000					
ICP	0.465	0.731	-0.213	-0.666	-0.429	0.476	-0.051	-0.263	0.546	1.000				
PEP/LVET	0.153	0.566	-0.210	-0.094	-0.606	0.005	-0.062	-0.250	0.787	0.695	1.000			
HR	0.324	0.130	-0.994	-0.518	-0.827	-0.600	-0.903	-0.958	-0.394	0.149	0.187	1.000		
TRIP	0,895	0.511	-0.643	-0.386	-0.402	0.009	-0.535	-0.620	-0.358	0.103	-0.055	0.607	1.000	
ETI	0.075	-0.379	-0.582	-0.175	-0.015	-0.129	-0.397	-0.607	-0.686	-0.471	-0.550	0.574	0.375	1.000