EFFECT OF PHYSICAL TRAINING ON THE OXYGEN TRANSPORT SYSTEM OF SIX YOUNG WOMEN

by

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ABSTRACT

Six female university students participated in a program of physical training consisting of 3 sessions per week for 12 weeks. Bi-weekly determinations were made of circulatory parameters and respiratory gas exchange parameters during submaximal and maximal exercise conditions. Pulmonary function was measured at rest.

Maximal oxygen uptake increased 12.8% (2.44 to 2.73 l/min) due mainly to a significantly increased ($p<.05$) cardiac output during maximal exercise. Arterio-venous oxygen difference increased only marginally as a result of training, the change not being significant. Post-exercise blood lactate accumulation increased significantly during training and post-exercise pH values correspondingly declined significantly during training. A significant decline in the maximal heart rate during exhaustive exercise occurred. No consistent changes occurred in the oxygen carrying capacity of the blood. For standard submaximal exercise, both heart rate and carbon dioxide production significantly declined. The accompanying increase in stroke volume was not significant. Cardiac output for standard submaximal exercise declined in the latter stages of training accompanied by an increase in arterio-venous oxygen difference.

However, these changes were not significant. A small but significant decrease in breathing frequency was the only change noted in pulmonary function at rest.

Physiological adaptations to training are analyzed by a comparison of the time course of changes during training of the parameters measured. Recommendations are made for the design of future experiments concerning the response of female subjects to physical training.

To my wife Judy, for her patience and support.

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CHAPTER I

INTRODUCTION

Muscular exercise may be sustained for prolonged periods of time only through a sufficient supply of oxygen to the mitochondria of the cells for the production of energy. Oxygen uptake is highly related to the degree of work being performed. Therefore man's ability to do work from the energy output of aerobic processes is limited by the maximal capacity of the oxygen transport system, or maximal oxygen uptake ($\dot{V}O_2$ max). With any attempt to perform work beyond this aerobic work capacity, the accumulation of fatigue by-products from anaerobic metabolism must shortly force the curtailment of the work. Besides being a measure of the capacity for energy output from aerobic metabolism, $\dot{V}O_2$ max serves as an integrative measure of the functional capacity of the cardiorespiratory system (Astrand, 1968).

Physiological mechanisms adapt not only to acute stresses but chronic ones also. Since Robinson and Harmon (1941) demonstrated increases in aerobic power due to chronic physical exercise, numerous investigations were undertaken to explore the potential adaptation capacity of the oxygen transport system. These studies, summarized by Ekblom (1969), indicate that gains in $\dot{V}O_2$ max of approximately 15 to 20%

of initial pre-training values may be expected from programs of 2 to 6 months duration. Increased performance capacity of the athlete and the enhanced work capacity of the sedentary individual have been commonly attributed to such findings. In addition, it has been suggested that they provide evidence for the role of exercise in increasing protection against degenerative diseases and rehabilitating patients with coronary heart disease.

While such speculation may be correct, it is dangerous to make such interpretations strictly on the basis of oxygen uptake adaptation. Oxygen transport is a function of the complex interplay of the mechanisms of circulation, respiration and metabolism. Measurement of oxygen uptake provides only an integration of the individual adaptation of the various factors (Shephard, 1967). Recently, studies have focused upon the effects of training on the hemodynamics of the circulation and on the component factors of respiration together with metabolic adaptations at the cellular level.

Cross-sectional studies (athletes compared to non-athletes or sedentary people) have provided interesting and valuable comparative data between the trained and untrained states. However, it is difficult to differentiate between genetic and environmental influences on the observed differences. Longitudinal investigations (testing sedentary people before, during and after a period of training) while less economical in terms of time and re-

sources, would appear to yield more consistent and controlled data.

Longitudinal studies on the adaptations of the circulation concomitant with increased aerobic power have shown similar trends. Increased $\dot{V}O_2$ max is accomplished by an increase in maximal cardiac output ($\dot{Q}$ max), together with an increased arterio-venous oxygen difference (A-V O_2 difference). The increased $\dot{Q}$ max is due entirely to an increased stroke volume (Rowell, 1962; Ekblom et al., 1968; Saltin et al., 1968; Hartley et al., 1969). Studies on circulatory adaptations to standard submaximal work with training have not produced such predictable results. Many investigators have shown a decrease in cardiac output for a standard submaximal oxygen uptake (Tabakin, Hanson and Levy, 1965; Andrew, Guzman and Becklake, 1966; Frick and Katila, 1968; Ekblom et al., 1968; Douglas and Becklake, 1968; Klassen, Andrew and Becklake, 1970). There is disagreement, however, as to the mechanism mediating this change. Moreover, other workers have found no change in cardiac output at a standard submaximal oxygen uptake (Freedman et al., 1955; Frick, Konttinen and Sarajas, 1963; Saltin et al., 1968; Ekblom, 1969; Ekblom, 1970).

Ideally, in any evaluation of adaptive mechanisms such as those mentioned above, repeated tests should be taken serially throughout the training program to establish a time course for changes. Such data would provide for a more

sensitive analysis of the mechanisms through comparison of the trends of various parameters. Some of the conflicting data and analysis of the above work may be due to the design of the studies. All of the longitudinal studies on circulatory adaptation to training involve only pre and post training testing. Owing to technical difficulties and subject discomfort associated with the dye-dilution technique for determination of cardiac output from indwelling venous and arterial catheters, collection of data serially throughout a short-term training program is not practical. Thus subtle interaction between parameters which would be useful in elucidating any adaptive mechanisms may not be readily apparent from single tests of pre and post-training.

In summary, males seem to respond to physical training by increased aerobic power due to an increased $\dot{Q}$ max and an increased A-V O_2 difference. At a standard submaximal oxygen uptake, it would appear that $\dot{Q}$ decreases, the mechanism for which is not clear. Moreover, conflicting data from workers who have found no such decrease in $\dot{Q}$ continues to cloud the issue. However, there remains a paucity of data on the effects of physical training on the oxygen transport system of females. From the results of the few cross-sectional studies comparing female athletes to female students (Astrand, 1960; Hermansen and Andersen, 1965; Ekblom and Hermansen, 1968), it is assumed that females respond in a similar fashion as do males to training. This remains to

be substantiated. Also well known are the differences between the sexes in their physiological responses to exercise and aerobic capacity (Astrand, 1952; Astrand, 1964; Hermansen and Andersen, 1965; McNab, Conger and Taylor, 1969).

Little beyond the scope of the above quoted work has been attempted with regard to female subjects. One previous study has evaluated the increase in aerobic power due to seasonal training in women athletes (Sinning and Adrian, 1968), with only marginal improvements noted. More comprehensive data, including circulatory and metabolic measures, is needed. Attention has been focused recently upon the importance of cardiovascular fitness due to popular publications such as K.H. Cooper's Aerobics. With the knowledge that females aerobic capacity either reaches a plateau or declines past puberty, probably due to a decline in activity level (Astrand, 1968), research on the training potential and beneficial physiological adaptations to training in college age females is necessary to provide positive scientific reinforcement for females who wish to embark on a training program. In addition, the growing field of women's athletics needs similar positive feedback on the training potential of females.

The present work investigated the circulatory and metabolic adaptations accompanying increases in aerobic power due to training in 6 females of college age. The purposes of the study were to:

1. identify the mechanisms by which $\dot{V}O_2$ max is increased due to training in the 6 college-aged women studied;
2. help clarify the mechanisms involved in the circulatory adaptations to a standard sub-maximal work task.
3. establish a time course for the circulatory and metabolic adaptations to training by repeated tests taken serially throughout the training program;
4. to determine any quantitative differences in the adaptation to training of college women as observed in this study and the extensive published results on males of the same age.

CHAPTER II

REVIEW OF LITERATURE

Determinants of the Effectiveness of Training Programs

Robinson and Harmon (1941) and Knehr, Dill and Neufeld (1942) were the first investigators to demonstrate an increased maximal oxygen uptake due to physical training. Many studies have observed the same phenomenon since and reported gains between 5 and 43% of initial pre-training values. Several factors seem to account for this wide variance. Ekblom (1969) recently demonstrated that the percentage gain in aerobic power seemed to have a high negative relationship to initial fitness status. Pollock, Cureton and Gruninger (1969) found that training 4 times per week resulted in significantly greater gains than training twice per week. Wilmore, Royce and Girandola (1970) showed that continuous runs lasting 24 minutes each, 3 times per week resulted in greater gains than 12 minute runs. It seems therefore that the effectiveness of training programs depends on the frequency, intensity and duration of the training sessions. Recently, Shephard (1968) and Gledhill (1969) have demonstrated that intensity of training seems to be the most important factor.

Physiological Factors Limiting Aerobic Power

Aerobic power can be limited by the structural dimensions of the cardiorespiratory system (Holmgren, 1967). Musshoff, Reindell and Klepzig (1959) have demonstrated that maximal stroke volume is highly related to heart volume. The ability to demonstrate a high maximal stroke volume has limitations imposed by the sheer physical size of the heart volume. A similar consideration applies in the case of the high correlation of maximal stroke volume to blood volume. A large blood volume may more easily "fill" the capacitance vessels, aid venous return and create a larger pulmonary capillary blood volume. Sjostrand (1956) and Holmgren et al. (1960) hypothesized that a larger pulmonary capillary blood volume reflects a higher potential energy within the system which may help in the maintenance of a large stroke volume with a short effective filling time at high heart rates. It is generally felt that lung dimensions are not a limiting factor in the development of aerobic power (Holmgren, 1967).

Whether aerobic power may be enhanced by increases in the structural dimensions of the cardiorespiratory system is debatable. Astrand et al. (1963) and Ekblom (1969) both produced increases in heart volumes of adolescents due to training beyond that expected from normal growth. However, studies on mature subjects show little or no change in heart volume with training except in cases

where training is extended over a period of years (Ekblom, 1969, Hartley et al., 1969; Ekblom, 1970). Only the study of Saltin et al. (1968) gave evidence of increased heart volume due to short-term training. This modest increase (11%), however, followed a period of bed rest previous to training, an experimental condition not present in the other investigations.

Structural dimensions of the lungs are usually not altered past adolescence. Although Wilmore, Royce and Girandola (1970) found an increase in vital capacity and a decrease in residual volume with training, the findings of most studies show no increases (Saltin et al., 1968; Sinning and Adrian, 1968). However, an increased blood volume is a well-known response to short-term training (Holmgren et al., 1960; Oscai, Williams and Hertig, 1968).

Limitations on individual functional capacities ($\dot{Q}$ max, SV max, maximal heart rate, $\dot{V}_E$ max) may also be expected to limit maximal aerobic power and work capacity. Many studies have focused on identifying the probable limiting factor in the oxygen transport system during maximal exercise. Margaria et al. (1965) studied the kinetics of oxygen uptake in supermaximal tasks and found that the rate of increase of oxygen uptake had a half-reaction time of 30 seconds regardless of the intensity of the task performed. He concluded that limitations on energy production resulted first from the supply of oxygen-rich blood to the tissues and not

primarily from the ability to utilize oxygen at the cellular level. Therefore, maximal cardiac output was hypothesized as the limiting factor by this group.

Biochemical studies have indicated that limitations on energy production do not primarily result from the ability to utilize oxygen at the cellular level. Only when nicotinamide adenine dinucleotide (NAD) is 90% reduced at intercellular P_{O_2} levels of 0.1 mm mercury is oxidative phosphorylation too slow to maintain necessary ATP levels (Chance, Schoener and Schnidler, 1964). However, the energy-producing systems *in vivo* are probably influenced by the by-products of cellular metabolism. Oxidative phosphorylation may be limited in its functional capacity by accumulation of metabolites in the cell. Karlsson and Saltin (1970) have recently demonstrated the relationship of adenosine triphosphate (ATP) and creatine phosphate (CP) to lactate levels within the muscle cell during various types of exhaustive exercise. In this study although muscle ATP and CP levels fell quickly from pre-exercise levels with onset of exercise, no further decline of ATP and CP was noted up to the point of exhaustion, regardless of the intensity and duration of the exercise task. Muscle lactate concentration, however, rose steadily throughout all exercise tasks to maximal levels at exhaustion. This study indicates that muscle lactate accumulation may limit exercise performance more than depleted levels of high energy phosphate compounds in the cell.

Cerettelli et al. (1964) and Ouellet, Poh and Becklake (1969) determined that oxygen supply to the working muscles did not increase beyond the "levelling off" point of cardiac output during severe exercise. Further increases in oxygen uptake provided only for the high energy cost of hyperventilation. These studies although not conclusive, provide empirical support for the theories postulated by Mitchell, Sproule and Chapman (1958) and Shephard (1967), indicating that maximal cardiac output is usually the limiting factor in achieving high levels of aerobic power. Maximal heart rate is relatively fixed for each individual and declines with increasing age. Therefore, over a wide age range, the ability to attain a high cardiac output during severe exercise rests mainly upon the ability of the heart to achieve a high stroke volume.

Maximal pulmonary ventilation during severe exercise is less than that achieved during maximal voluntary ventilation (Shephard, 1967). Therefore, the functional capacity of the lungs in normal people would not seem to limit aerobic power. This was substantiated by the findings of Ouellet, Poh and Becklake (1969). Only in pathological states where the functional capacity of the lungs is severely limited (e.g. chronic bronchitis, emphysema) will the pulmonary ventilation become a limiting factor (Holmgren, 1967).

Effect of Training on Oxygen Transport System

The previously mentioned studies concerned with identifying the determinants limiting aerobic power have provided insight into the physiological characteristics of the trained state. The findings from cross-sectional studies comparing athletes and non-athletes have substantiated these observations. It is well known that athletes and physically active people have higher levels of aerobic power than sedentary people (Buskirk and Taylor, 1957; Hermansen and Andersen, 1965; McDonough, Kusumi and Bruce, 1970). Saltin and Astrand (1967) reported a mean maximal oxygen uptake for 15 champion athletes of 5.75 l/min compared to a range of 3.20 to 4.11 l/min reported for inactive or semi-active young males (Astrand, 1956; Hermansen and Andersen, 1965).

Ekblom and Hermansen (1968) recently determined that the high oxygen uptake value typical of the athlete was due to a high cardiac output. They reported mean values for maximal oxygen uptake and maximal cardiac output of 5.57 l/min and 36.0 l/min respectively. This is in contrast to the semi-active students studied by Astrand et al. (1964) who attained values for the same parameters of only 4.05 l/min and 24.1 l/min. Since the maximal heart rates of the two groups were almost identical, the difference in maximal cardiac output was due entirely to the athletes having a higher stroke volume (189 ml) than the non-athletes (134 ml). Bevegard, Holmgren and Jonsson (1963) also found higher

stroke volumes for athletes compared to sedentary people.

Athletes are capable of higher maximal pulmonary ventilations, higher systolic and mean blood pressures at maximal work and a larger A-V O_2 difference (Bevegard, Holmgren and Jonsson, 1963; Saltin and Astrand, 1967). When oxygen content of mixed venous blood is used as a comparative measure rather than A-V O_2 difference, the athletes exhibit a much lower value in maximal exercise than non-athletes (Ekblom, 1969). In this way, differences in oxygen-carrying capacity of the blood between the two groups are negated. Physical work capacity is greater in athletes. Members of this group are characterized by having significantly larger heart volumes (Musshoff, Reindell and Klepzig, 1959; Ekblom and Hermansen, 1968) and larger total blood volumes (Bevegard, Holmgren and Jonsson, 1963).

It is difficult to apportion the degree of influence that training or genetic endowment has had in developing the superior characteristics of athletes. Obviously, large volumes of important organs aid in the performance capacity of athletes. However, Bevegard, Holmgren and Jonsson (1963) also found in their series of athlete investigations that maximal stroke volume in relation to heart volume was higher than expected from regression equations derived on normals. This indicates more efficient functional adaptation of the circulation in athletes than in normal subjects with similar circulatory dimensions.

Longitudinal training studies have succeeded in elucidating some of the physiological adaptations to training. Some insight has been gained into the relative importance of environmental influences (training) and heredity on achieving high levels of aerobic power. Rowell (1962) found that increased aerobic power from an intensive training program was due to an increased cardiac output and a widened A-V O_2 difference. Similarly, Ekblom et al. (1968) found an increased maximal oxygen uptake of 16.2%, caused by an increased maximal cardiac output of 8% and an increased A-V O_2 difference of 0.5 ml/100 ml. The latter was due to a lower oxygen content in mixed venous blood. The increased cardiac output was due solely to an increased stroke volume of 15 ml, since maximal heart rate remained the same. In an other study done on young men, Saltin et al. (1968), intensive training followed a period of bed rest. This produced an increased maximal oxygen uptake of 33%. This finding was due in equal extent to an increased cardiac output and an increased A-V O_2 difference.

Two other studies of a similar nature have been done on middle-aged males. Hanson et al. (1968) demonstrated an increase of 18% in aerobic power but circulatory evaluations post training failed to extend the subjects to their maximum and no increments in cardiac output or A-V O_2 difference were noted. Hartley et al. (1969) found an increase in

aerobic power of 14% accomplished solely by an increased cardiac output.

The reasons for increased stroke volume due to training evidenced in these studies remains unclear. Only in the study by Saltin et al. (1968) did heart volume increase with training. However, even in this case, the increase in heart size was not enough to account for the increase in stroke volume. Saltin et al. (1968) imply that an enlarged heart volume seems to be the mechanism for the greater stroke volume induced by training. However, Ekblom (1969) hypothesizes that an increased myocardial contractile power occurs, resulting in the heart functioning with less systolic residual blood volume.

The increased A-V O_2 difference found from training is thought to be a result of more effective distribution of oxygen to the working muscles. (Ekblom, 1969; Hartley et al., 1969). The low A-V O_2 differences noted by Grimby, Nilsson and Saltin (1966) in middle-aged athletes together with the lack of increase in A-V O_2 difference for the middle-aged subjects of Hartley et al. (1969) reflects the limitations of aging on performance capacity and trainability. The lower oxygen carrying capacity of the blood in older men results in a lower potential for oxygen extraction at the tissues. There is, in addition, experimental evidence for an increased diffusing distance from the capillaries to working cells in aged skeletal muscle (Grimby, Nilsson and Saltin, 1966).

Other physiological variables during maximal exercise are changed with training. Maximal pulmonary ventilation increases co-incidently with maximal oxygen uptake, and maximal lactate levels increase (Astrand, 1956). Maximal lactate levels are thought to reflect man's physical fitness, as athletes have been shown to tolerate very high lactate levels in comparison to untrained individuals (Knehr, Dill and Neufeld, 1942). A rise in the maximal attainable lactate level with training is consistent with this theory and probably reflect the increased capacity for anaerobic metabolism during severe exercise.

Effects of training may be observed during submaximal as well as maximal exercise. Cross-sectional studies comparing the response of athletes and non-athletes to standard submaximal exercise indicate the greater relative efficiency of the trained state. Athletes commonly exhibit lower heart rates and pulmonary ventilations for a given oxygen uptake, reflecting higher levels of circulatory and respiratory efficiency (Musshoff, Reindell and Klepzig, 1959; Bevegard, Holmgren and Jonsson, 1963; Cobb and Johnson, 1963; Hanson and Tabakin, 1965). However, the literature generally supports the view that athletes do not have a lower oxygen uptake for a given submaximal work rate unless there is a gross difference in ventilation between athletes and normal people. In this case, the increased work of breathing for the normal person may contribute disproportionately to the

oxygen uptake. In addition, athletes exhibit a lower respiratory exchange ratio (R) for a given work rate due to a lower carbon dioxide elimination (Hanson and Tabakin, 1965). Lower levels of blood lactic acid in athletes for a given submaximal work rate have been noted by many investigators (Dill, Talbott and Edwards, 1930; Holmgren and Strom, 1959; Cobb and Johnson, 1963). The lower R probably results from lower lactate levels in the athlete since addition of non-volatile acid to the blood causes excretion via the lungs of more CO_2 than is formed metabolically ("excess CO_2"), thereby increasing R (Issekutz and Rodahl, 1961).

Cross-sectional studies on cardiac output response to standard submaximal work demonstrate more variable results. Musshoff, Reindell and Klepzig (1959) reported no differences between athletes and non-athletes although, athletes have a higher stroke volume and typically, a lower heart rate, than non-athletes. Cobb and Johnson (1963) found similar results, but Hanson and Tabakin (1965) found a consistently lower cardiac output for athletes. This was accomplished by a higher stroke volume and a much lower heart rate. However, Bevegard, Holmgren and Johnson (1963) found a higher cardiac output for standard submaximal work in athletes and a higher stroke volume.

Such conflicting data probably reflects the difficulties inherent in cross-sectional studies, as has been previously discussed. Longitudinal studies of the circulatory response

to standard submaximal work seem to indicate that training results in a decreased cardiac output for a given oxygen uptake, and therefore a higher arterio-venous oxygen difference. Tabakin, Hanson and Levy (1965) found a decreased cardiac output due to training at two mild, submaximal work loads. Andrew, Guzman and Becklake (1966) demonstrated a significant reduction in cardiac output at a given oxygen uptake in 4 college athletes and 4 non-athletic college students. These findings have been substantiated for patients with coronary heart disease (Varnauskas et al. 1966; Frick and Katila, 1968; Clausen, Larsen and Trap-Jensen, 1969), for athletes (Douglas and Becklake, 1968; Klassen, Andrew and Becklake, 1970) and for young, sedentary males (Ekblom et al., 1968). Typically, the reduction in cardiac output was accompanied by a marked drop in heart rate while stroke volume either slightly increased or did not change.

The mechanisms responsible for the reduction in cardiac output for standard submaximal exercise and hence the widened arterio-venous oxygen difference have been widely discussed. Varnauskas et al. (1966), Andrew, Guzman and Becklake (1966) and Klassen, Andrew and Becklake (1970) have postulated a more effective distribution of blood to the working muscles. It is known that redistribution of blood flow occurs during exercise, decreasing flow to the splanchnic and renal beds (Wade, 1962). Bevegard and Shephard (1966) have shown that during moderate leg exercise, forearm blood

flow remains at a resting level but is decreased markedly during severe exercise. Therefore, flow to inactive muscles is also reduced during exercise due to reflex sympathetic vasoconstriction from adrenergic vasomotor fibers.

An enhancement of these mechanisms, together with a hypothesized increased vascularization of muscle with training (Petren, Sjostrand and Sylven, 1936; Tomanek, 1970) would result in a more effective oxygen supply to working muscles.

Andrew, Guzman and Becklake (1966) suggested that the well-known decrease in ventilation for standard submaximal work with training may result in a reduction in blood flow to the external muscles of respiration. Reduction in blood flow may occur in areas such as the skin, where flow is regulated by factors other than oxygen requirements. Recently, the findings of Klassen, Andrew and Becklake (1970) have provided support for the blood redistribution theory. They found that forearm blood flow in standard exercise in paddlers remained the same before and after a period of training. However, cardiac output for a standard submaximal task (hand crank) was reduced after training, indicating a decrease in flow to non-active parts. The lack of effective blood redistribution during exercise is readily apparent in the elevated cardiac outputs seen in very unfit subjects with "vasoregulatory asthenia" (Holmgren et al., 1957).

The other mechanism commonly thought to cause both the decreased lactate levels with training and the reduction

in cardiac output is enhancement of cellular enzyme systems. Holloszy (1967) found a doubled rate of pyruvate oxidation in skeletal muscle mitochondria of trained rats. During moderate exercise such a mechanism, if effected in man as a consequence of training, may result in greater oxygen extraction and an inhibition of glycolysis. These findings are supported by Gollnick and King (1969) and Kraus, Kirsten and Wolff (1969) who found an increase in the number and size of mitochondria of skeletal muscle in trained rats. Barnard, Edgerton and Peters (1970) also demonstrated an increased capacity for oxidative metabolism in skeletal muscle of the guinea pig. These investigators found also that, trained skeletal muscle contains a greater number of 'red' or aerobic fibers. This latter finding has been reported also by Van Linge (1962).

Varnauskas et al. (1970) have recently found that muscle mitochondrial enzymatic activity in man increased by 44% as a consequence of training, lending support to the above-quoted animal studies.

A new hypothesis explaining the decrease in cardiac output has recently been proposed by Clausen (1969). He suggests that physical training does not result in more effective redistribution of blood to working muscles. Rowell, Blackman and Bruce (1964) found that the decrease in splanchnic flow was less pronounced in trained than in untrained persons during exercise at the same oxygen uptake.

Castenfors (1967) reported identical findings for renal flow. Visceral vasoconstriction during exercise is mediated by a centrally controlled sympathetic nervous activity. After maximal oxygen uptake is increased by training, the relative strain of a given oxygen uptake would be less. Therefore, a lesser degree of sympathetic activity and vasoconstriction will occur, and blood flow to the viscera and non-exercising limbs will increase. After training, the blood flow to non-exercising tissues may deviate less from the resting level at a given submaximal work load.

A reduction in cardiac output during sub-maximal work may be caused, therefore, by a decrease in flow to the exercising muscles. Such a conclusion is supported by studies showing that muscle blood flow is reduced about 20-30% when measured at identical submaximal work loads before and after training (Clausen, Larsen and Trap-Jensen, 1969; Varnauskas et al., 1970). In addition, Grimby, Haggendal and Saltin (1967) found that trained subjects had a lower muscle blood flow than untrained subjects for the same work load. The increased oxygen extraction which must occur from this reduced flow is probably due to the cellular biochemical changes discussed above. Clausen (1969) suggests that,

> Exercise hyperemia in muscle is thought to be caused by local release of metabolically linked vasodilator compounds (Haddy and Scott, 1968). The improved oxidative enzymatic capacity may keep these metabolites at lower concentrations and thus limit the blood flow increase.

As attractive as this hypothesis appears, conflicting data continues to obscure the issue. Other longitudinal studies have not observed a decrease in cardiac output with training (Freedman et al., 1955; Frick, Konttinen and Sarajas, 1963; Saltin et al., 1968; Hartley et al., 1969; Ekblom, 1970). These studies have demonstrated the classical decline in heart rate and increase in stroke volume for standard submaximal work. However, two of three sedentary subjects in the study by Saltin et al. (1968) exhibited a marked decline in cardiac output when the bed rest influence is not considered.

Investigations on Female Subjects

Studies on the effects of training have been concerned almost exclusively with males. Very few studies have dealt with the effects of training on the oxygen transport system of females, and few cross-sectional studies exist. Astrand (1952) reported a mean maximal oxygen uptake of 2.9 l/min or 48 ml/kg/min for active young female physical education students. This may be compared to a mean of 2.8 l/min for young girl swimmers (Astrand et al., 1963). Other studies on Scandinavian girl athletes have reported mean values of

55 ml/kg/min (Hermansen and Andersen, 1965) and 62 ml/kg/min (Saltin and Astrand, 1967). Sprynarova and Parizkova (1969) found a mean maximal oxygen uptake of 45 ml/kg/min in girl swimmers. These observations are in contrast to the low values typical of sedentary females. Untrained females in Scandinavia generally have a higher aerobic capacity than North American girls. Studies have reported mean values of 40 ml/kg/min (Astrand, 1960), 41 ml/kg/min (Astrand et al., 1964) and 38 ml/kg/min (Hermansen and Andersen, 1965).

For North American girls, Michael and Horvath (1965) found a mean $\dot{V}O_2$ max of 30 ml/kg/min in 30 female college students. Moody, Kollias and Buskirk (1969) reported a mean value of 37 ml/kg/min for college females while McNab, Conger and Taylor (1969) found a value of 39 ml/kg/min for 24 college women. Horvath and Michael (1970) substantiated their earlier observations more recently finding a mean value of 32 ml/kg/min for college girls.

It is obvious that the active girl athlete possesses a higher aerobic power than her sedentary counterpart. Therefore training probably creates physiological adaptations in the female in a manner similar to males. Astrand et al. (1963) related aerobic power and structural dimensions of girl swimmers to normal girls of that age range. They hypothesized that the greater than expected heart volume and aerobic power typical of girl swimmers were due to their regular training. Sinning and Adrian (1968) found an

increase in $\dot{V}O_2$ max from 34.4 to 38.9 ml/kg/min in 7 female basketball players due to seasonal training. Stovel, Bailey and Cumming (1970) found only marginal improvements in aerobic power from a 6 week home exercise program. Two other studies on females have suggested cardiorespiratory improvement in females with training by indirect testing procedures (Sloan, 1961; Applegate and Stull, 1969).

No investigations have been concerned with the circulatory adaptations to training in females. The only circulatory data on females during exercise up to maximal levels is that of Astrand et al. (1964).

Differences between the sexes in aerobic capacity are quite pronounced. Astrand (1952) reported males to have a $\dot{V}O_2$ max 29% higher than females (4.11 l/min to 2.90 l/min). In a later investigation, this difference was greater, 4.05 l/min to 2.60 l/min (Astrand et al., 1964). McNab, Conger and Taylor (1969) found a difference of 69% or 3.92 l/min compared to 2.32 l/min.

Many reasons are suggested in the literature to explain these differences. Females have a lower proportion of lean body mass to total body weight (Rauh and Shumsky, 1968). However, even when corrected for body weight and lean body weight, the difference is still highly significant (McNab, Conger and Taylor, 1969). Astrand et al. (1964) has shown that women have higher cardiac outputs than men for a given submaximal work load or oxygen consumption due

to their lower hemoglobin concentration and thus lower oxygen carrying capacity of the blood. A female's stroke volume is limited by her smaller heart volume (Astrand, 1964). To achieve a given cardiac output, the female responds with a much higher heart rate than the male, to compensate for her lower stroke volume. At maximal exercise, the limitations of stroke volume and oxygen-carrying capacity of the blood in the female cannot be compensated by a further rise in heart rate, and a lower oxygen transport capacity results. .

CHAPTER III

METHODOLOGY

Subjects

The subjects were 6 college girls who had not engaged in any form of regular training in the year previous to this investigation. Subjects A.K., C.M. and L.R. had participated in high school track competition. However, training for this competition was irregular and not concerned with cardiorespiratory endurance. Subject C.T. was formerly an intercollegiate basketball player, but had not trained regularly for 12 months prior to the onset of the training program. The subjects' anthropometric data are shown in Table I. Based on the recommendations of Andrew, Guzman and Becklake (1966) and Ekblom *et al.* (1968) the subjects served as their own controls to negate group differences.

Training

The training program was modified from that of Ekblom (1969) and Kasch and Boyer (1969). The subjects trained for 12 weeks consecutively, with 3 training sessions per week. The first 10 or 15 minutes of each session were devoted to warm-up exercises, mainly of the static stretching variety,

preparing major muscle groups and joints for more strenuous activity. The second phase of the exercise session was interval or continuous cross-country running for cardio-respiratory stress.

The interval training consisted of repeat runs of 3 to 9 minutes duration. The appropriate intensity of the exercise

TABLE I

ANTHROPOMETRIC DATA AND AGE OF THE SUBJECTS

SUBJECT	AGE	HEIGHT (cm)	WEIGHT (kg)
A.K.	25	160.9	49.3
C.L.	19	164.4	63.3
C.M.	18	162.6	52.4
L.R.	18	162.6	48.7
C.S.	19	170.9	63.9
C.T.	22	160.0	61.2

stress was determined for each individual. Periodic heart rate monitoring by palpation of the carotid pulse for 10 second intervals was employed, beginning 5 seconds after exercise ceased. The intensity of the interval runs were designed to elicit heart rates between 160 and 190 beats per minute. Karlsson, Astrand and Ekblom (1967) have shown that $\dot{V}O_2$ max is achieved or nearly achieved at a running speed

less than that necessary for high lactate accumulation. They suggest that the speed of running should not be intense enough to create undue fatigue and limit the volume of training attempted by the individual. This concept was used in the interval training sessions to attain repeated stress on the oxygen transport system. The number of runs during an interval session ranged from 3 at the beginning of training to 5 at the end of training. The rest intervals ranged from 3 minutes at the onset of training to 1 minute at the end of training.

When the subjects were able to tolerate it, continuous cross-country running was alternated with interval training. These runs lasted at least 18 minutes and elicited minimum heart rates of 150 beats per minute throughout the run. Maximal heart rates were often achieved running up hills or immediately upon completion of a run.

The training program is shown in detail in Table II. By the end of training, 4 of the 6 subjects were running 3 miles in 25 minutes or less. Toward the end of the training program, interval training was done more frequently, since heavy snow falls made continuous cross-country running impractical. Intense interval training was done on the snow-covered track, producing heart rate responses between 186 and 204 beats per minute for each work interval.

One field performance test was done on day 62 of training, a mile run for time. Subject A.K. was unable to

TABLE II

PROGRESSIVE TRAINING SCHEDULE, COMPLETED THROUGHOUT THE TRAINING PROGRAM BY 6 SUBJECTS

DATE	TYPE OF TRAINING	DURATION OF RUNS (min)	DISTANCE (miles)	DATE	TYPE OF TRAINING	DURATION OF RUNS (min)	DISTANCE
Sept 14	interval (3)	3	1.0	Oct 23	continuous	20	2.2
Sept 16	interval (4)	3	1.25	Oct 26	interval (5)	4	2.25
Sept 18	interval (2)	3	0.5	Oct 28	continuous	20	2.2
Sept 21	interval (3)	4	1.25	Oct 30	interval (5)	4	2.6
Sept 23	interval (4)	4	1.5	Nov 2	continuous	21	2.25
Sept 25	interval (4)	4	2.0	Nov 4	interval (4)	5	2.4
Sept 28	interval (3)	6	2.0	Nov 6	continuous	22	2.4
Sept 30	interval (3)	6	2.0	Nov 9	continuous	24	2.6
Oct 2	interval (3)	6	2.2	Nov 13	interval (5)	5	2.9
Oct 5	interval (3)	6	2.2	Nov 16	mile run	time trial	1.0
Oct 7	interval (3)	6	2.2	Nov 18	continuous	24	2.75
Oct 9	interval (2)	9	2.2	Nov 20	continuous	24	2.8
Oct 14	interval (2)	9	2.2	Nov 23	interval (4)	3	1.75
Oct 16	continuous	18	2.2	Nov 25	continuous	25	3.0
Oct 19	interval (2)	9	1.75	Nov 27	interval (3)	4	1.5*
Oct 21	interval (4)	4	2.0	Nov 30	interval (3)	4	1.5*
				Dec 2	interval (3)	4	1.5*

Number in parentheses after "interval" is the number of runs in the session.
* These training sessions done on snow-covered track.

perform the time trail due to illness. The results of this test are presented in Table III.

TABLE III

RESULTS OF THE MILE RUN TIME TRIAL
DONE ON DAY 62 OF TRAINING

SUBJECT	TIME
C.L.	8:25
C.M.	7:45
L.R.	7:50
C.S.	7:35
C.T.	8:00

Experimental Procedure

Seven days before the experimental testing began, the subjects were given a submaximal and a maximal exercise task on a Monarch bicycle ergometer (Stockholm, Sweden) to familiarize them with the experimental procedure. Pre-training baseline data consisted of two complete tests on each subject, taken 48 hours apart. Thereafter, the complete test was repeated for each subject every 2 weeks. At the end of training, the terminal data consisted of 2 tests on each subject completed within 7 days of termination of training. Due to scheduling difficulties, 5 of the 6 subjects had to perform their two tests post-training with only

24 hours rest between tests.

The subjects had only a light non-fat meal 3 hours previous to being tested. Following a 4 minute warm-up period (50 rpm, no load), a submaximal work rate was undertaken at 50 rpm for 5 minutes at a level corresponding to 50% of their maximal oxygen uptake, estimated from the preliminary test. Subjects C.L. and L.R. worked at 300 kpm/min, while the other 4 subjects worked at 450 kpm/min. In each subsequent test throughout the training program, this submaximal work rate remained constant for each subject, so that cardiorespiratory adaptations to a standard work rate could be observed. After a five minute rest, a maximal exercise test began. The first minute was performed with no load (70 rpm). In the second minute, the work rate corresponded to 50% $\dot{V}O_2$ max (70 rpm), and in the third minute was 80% of $\dot{V}O_2$ max (80 rpm). At the end of the third minute the maximal task was begun (80 rpm). According to the recommendations of Astrand and Saltin (1961), this work rate was selected to exhaust the subject in 3 minutes. The test was terminated when the subject could not maintain the pedalling rate. The pedalling rate during the final maximal work rate was raised to 90 rpm at test 5 (day 59 of training) to prevent limitation of work performance by local muscular fatigue.

The high pedalling rates were used for the maximal ride to ensure a maximal stress on the cardiorespiratory system. Banister and Jackson (1967) have shown that pedalling

rates from 80 to 120 rpm elicit higher oxygen uptakes for a given work rate due to a greater muscular inefficiency than lower pedalling rates (e.g. 50 rpm). They suggested that maximal tasks should be done at high pedalling rates, with a minimum exercise duration of 5 minutes.

During both submaximal and exhaustive exercise tests, heart rates were recorded for each minute of exercise. Oxygen uptake and carbon dioxide output were determined for the last 2 minutes of exercise and cardiac output was measured at the termination of both exercise sessions. From these directly determined measures the respiratory exchange ratio ($\dot{V}CO_2/\dot{V}O_2$), stroke volume ($\dot{Q}$/heart rate) and arterio-venous oxygen difference ($\dot{V}O_2/\dot{Q}$) were calculated.

Blood was drawn from the ear lobe pre-warmed by a heating pad at rest, and directly following maximal exercise for determinations of hemoglobin concentration, hematocrit, and pH. Blood was drawn from the median cubital vein 10 to 13 minutes post-maximal exercise for determination of lactic acid concentration.

Methods

Body weight was measured with a balance-beam scale before the exercise tests.

At rest before exercise, pulmonary function variables (minute ventilation, tidal volume, breathing frequency, vital

capacity and forced expiratory volume in 1.0 second) were measured by a Collins 13.5 liter respirometer (Boston, Mass.).

During exercise the subject breathed through a Collins Triple J valve (Boston, Mass.) from which expired gas composition was directly analyzed for CO_2 with a Godart NV Capnograph and for O_2 by a Westinghouse Model 211 Pulmonary Function Oxygen Monitor (Pittsburgh, Pa.). The connection between the breathing valve and the gas analyzers was $\frac{1}{4}$ in I.D. rubber tubing to the Capnograph. Direct connection to the oxygen analyzer was by $\frac{1}{8}$ in I.D. heated teflon tubing embedded in the Capnograph tubing distal to the outlet of the Collins valve. Sampling rate to the Capnograph was 2 l/min and to the oxygen analyzer 150 ml/min. Analysis was made breath by breath and recorded on 10 in paper with full scale deflection response time 0.5 seconds (Phillips Model PM 8100). Both analyzers were calibrated at two points on a linear scale with known gas concentrations analyzed by the micro-Scholander technique.

Ventilation was measured by a Parkinson-Cowan high precision low resistance gas meter connected to the Collins valve by a non-kinkable hose (I.D. $1\frac{1}{2}$ in).

Based on the work of Rahn and Fenn (1957) the mid-point between inspired and end-tidal gas concentrations might be assumed to be the mean expired gas concentration. However, subsequent investigations indicated that except

during resting states this was not the case. A comparison of 180 aliquot minute samples of expired gas withdrawn in oiled syringes from a mixing chamber inserted in the expired gas line, with concurrent breath by breath analysis indicated that the mean expired gas concentration for both carbon dioxide and oxygen was 70.9% (SD±1.0) of the end-tidal concentrations. This value was obtained over the range of ventilation rates and breathing frequencies observed in the submaximal tests of the present study. On the basis of the above findings, aliquot samples of mixed expired air were used for determination of oxygen uptake and carbon dioxide output beginning with test 3 during training.

Breath by breath analysis was continued concurrently with mixed expired gas analysis for the remainder of the training program to derive further comparisons at submaximal and maximal levels of ventilations and breathing frequencies. With submaximal ventilations, mixed expired gas concentration for O_2 was 69.9% (SD±4.3) and 69.2% (SD±4.0) for CO_2 of the end-tidal concentrations. With high ventilations and breathing frequencies during maximal exercise however, the mixed expired gas concentration for O_2 was found to be 73.5% (SD±5.0), and 86.9% (SD±6.3) for CO_2 of end-tidal concentrations. These values were used in the computation of carbon dioxide output and oxygen uptake for the initial 2 pre-training tests for each subject.

Technical difficulties with the oxygen analyzer before the test of day 15 of training made determination of oxygen uptake impossible in this instance. The rest of the measures were recorded as usual. Before the final post-training tests, the fuel cell of the oxygen analyzer was damaged. It was replaced by a fuel cell which appeared to be more sensitive to pressure of gas flow. The introduction of syringe samples of mixed expired air into the O_2 analyzer were thus in error, compared to the previous analyses. As previously mentioned, breath by breath gas analysis was done concurrently with mixed expired gas analysis (syringe samples) from test 3 to the post-training tests. For submaximal ventilations and breathing frequencies, mixed expired gas concentration for O_2 was 69.9% (SD±4.3) of end-tidal concentrations from breath by breath analysis. With high ventilations and breathing frequencies during maximal exercise, this value was found to be 73.5% (SD±5.0) for oxygen. These values were used for computation of oxygen concentration in expired air for the two post-training tests for each subject. Carbon dioxide concentration was routinely determined by analysis of mixed expired air from aliquot syringe samples.

Thus, computations of $\dot{V}O_2$ for tests 3, 4, 5 and 6 during training were done from analysis of aliquot syringe samples of mixed expired gas taken from a mixing chamber inserted in the expired gas line. The relationships between mixed expired gas concentrations for O_2 and CO_2 determined

during tests 3, 4, 5 and 6 were used to compute oxygen uptake and carbon dioxide production from breath by breath gas analysis in the pre-training and post-training tests.

Data from pre-training and post-training tests seemed consistent with the data from the tests where mixed expired air was used for analysis. Moreover, this data seems comparable with published values in the literature for similar work rates in sedentary individuals.

Cardiac output was determined by the CO_2 rebreathing method as described by Defares, Wise and Duyff (1961) and modified by Jones et al. (1967), Jernerus, Lundin and Thompson (1963) and Magel and Andersen (1967). Since no indwelling catheters are needed with this method, it was feasible to make determinations of cardiac output bi-weekly throughout the training program. The principle of this technique is that if the difference in CO_2 content between mixed venous and arterial blood is known, as well as carbon dioxide output, cardiac output may be calculated according to a modified Fick principle:

$$\dot{Q} = \frac{\dot{V}CO_2}{C\bar{v}_{CO_2} - Ca_{CO_2}}$$

where $\dot{Q}$ = cardiac output (l/min)

$\dot{V}CO_2$ = carbon dioxide output (l/min)

$C\bar{v}_{CO_2}$ = mixed venous carbon dioxide content (vol%)

Ca_{CO_2} = arterial blood carbon dioxide content (vol%)

If the P_{CO_2} of mixed venous and arterial blood is determined, the difference in CO_2 content may be calculated by using the appropriate CO_2 dissociation curves.

Mixed venous P_{CO_2} was determined by rebreathing high concentrations of CO_2 from a 5 liter bag and continuously analyzing the CO_2 concentration in the closed lung-bag system. On attainment of a steady CO_2 concentration in the lung-bag system for at least 2 consecutive breaths, it may be assumed that this concentration is equal to the P_{CO_2} of incoming mixed venous blood, with which it has been equilibrating.

Arterial blood P_{CO_2} was determined from the concentration of CO_2 in end-tidal respired air. Since this is a representative sample of alveolar air, it is theoretically the same as arterial blood P_{CO_2} (Magel and Andersen (1967). End-tidal concentrations for the breaths immediately preceding rebreathing were averaged to determine Pa_{CO_2}. A constant value of 3 mm Hg was added to correct underestimation of arterial blood P_{CO_2} from end-tidal Pa_{CO_2} (Magel and Anderson, 1967).

Recirculation of CO_2 enriched blood may prevent attainment of true equilibrium during rebreathing (Jernerus, Lundin and Thompson, 1963). However, Jones et al. (1967) showed that even at maximal exercise, recirculation did not occur within 10 seconds after onset of rebreathing to a degree which would affect the results. To establish equilib-

rium more rapidly, Jones et al. (1967) used a high concentration of 15% CO_2 in 85% O_2 for rebreathing. This procedure was used in the present study.

The mixed venous P_{CO_2} was used in conjunction with the carbon dioxide dissociation curve for blood with a complete oxygen content to determine the actual CO_2 content since this blood was fully saturated from rebreathing a gas with a high oxygen concentration (85%). Ca_{CO_2} was likewise determined from the Pa_{CO_2} value.

The subjects maintained the work rate after expired gas analysis was completed until rebreathing was done and equilibrium established. During maximal exercise, rebreathing sometimes resulted in a phenomenon where the concentration of CO_2 in an exhalation exceeded that of the previous inhalation from the bag. Equilibrium is not attained in this case. Jones et al. (1967) also noted this phenomenon and developed a formula to compute the true equilibrium point from the pattern of "phase reversal". The computation procedure of Jones et al. (1967) was used in the present study when necessary. "Phase reversal" never occurred during submaximal exercise.

Studies by Muisan, Sorbini and Solinas (1968) and Furguson et al. (1968) have demonstrated that the CO_2 rebreathing technique for cardiac output determination is both reproducible and well correlated with results obtained from other methods, especially during exercise.

Heart rates were monitored by radio telemetry (Parks Electronics, Beaverton, Oregon) and recorded on a Hewlett Packard Model 1500 A ECG.

Hemoglobin concentration was determined by the Hb-Meter No. 1010C (American Optical, Buffalo, N.Y.) on blood drawn from the ear lobe. Hematocrit was determined in an Adams Readocrit micro-hematocrit centrifuge on blood drawn from the ear lobe.

The pH was measured by means of the Astrup-micro-equipment AME-1 (Radiometer, Copenhagen) on blood drawn from the ear lobe. The apparatus was calibrated with precision buffer at two points in the pH range (6.840 and 7.381) to 0.001 pH units.

Lactate concentrations were determined from a 5 ml blood sample taken from the median cubital vein 10 to 13 minutes post-maximal exercise. The blood sample was de-proteinized and serum was separated by centrifugation. Serum lactate concentrations were obtained using the Boehringer enzymatic method (Bergmeyer, 1962).

Treatment of the Data

Subject C.M. was ill for test 5 and subject L.R. was ill for test 6 during training. Ten days of bed rest associated with the illness of subject L.R. had an adverse effect on her fitness. Therefore, the data from her post-

training tests was not included in the computations and statistical analysis. The last 3 tests during the training program therefore had data from 5 of the 6 subjects.

A within subjects, two-way analysis of variance was performed on the data to determine significant changes due to training. The F ratio was considered significant if it had a probability of less than 5% ($p<.05$). Significant differences between means were determined by the Newman-Keuls method. Data from previous tests was substituted for missing data where this occurred. For maximal exercise parameters (i.e. $\dot{V}O_2$ max), the highest value attained prior to the missed observation was used. For submaximal exercise parameters, the mean value of previous tests was substituted. In this way, the estimated values would be least likely to influence the analysis of determining significant changes due to training. The substituted values would, if anything, be detrimental to the chance of obtaining a significant F ratio.

CHAPTER IV

RESULTS

The variations of selected cardiorespiratory parameters with time for each subject and the group means are presented in Figures 1 through 15. For those measures demanding a maximal effort, the highest of the two pre-training and two post-training values were used in the graphs and computations (e.g. $\dot{V}O_2$ max, $\dot{Q}$ max). For determinations taken during submaximal exercise and at rest where motivation and maximal performance were not essential, the mean of the two pre-training and two post-training values were used. Group means of each test for all variables during training are presented in Tables IV through VII. Results of the Newman-Keuls test for significance between means for parameters with significant F ratios are shown in Table VIII. Individual raw data for all variables determined during the training program are presented in APPENDIX 1.

Respiratory Gas Exchange, Lactate and pH in Maximal Exercise (Table IV, Figures 1 to 4)

Repeat determinations of maximal oxygen uptake in pre-training tests demonstrated a reliability within 0.10 l/min or 2.0 ml/kg/min. For post-training tests, 4 of the

TABLE IV

RESPIRATORY GAS EXCHANGE, LACTIC ACID AND pH IN MAXIMAL EXERCISE. GROUP MEAN AND STANDARD ERROR SHOWN FOR EACH TEST DURING TRAINING

Test	$\dot{V}O_2Max$ l/min	$\dot{V}O_2Max$ ml/kg/min	$\dot{V}CO_2$ l/min	R	$\dot{V}E$ l/min BTPS	La mg%	pH pre	pH post
Initial n = 6	2.44 ±0.09	43.8 ±2.5	2.79 ±0.10	1.14 ±.03	100.5 ±5.3	42.8 ±7.8	7.397 ±.007	7.197 ±.023
2 n = 6	†-	†-	2.64 ±0.13	†-	102.4 ±4.2	51.8 ±9.9	7.411 ±.006	7.220 ±.023
3 n = 6	2.46 ±0.12	44.0 ±2.4	2.64 ±0.11	1.08 ±.04	106.1 ±4.5	46.3 ±12.0	7.380 ±.018	7.167 ±.025
4 n = 6	2.46 ±0.15	44.0 ±2.7	2.46 ±0.13	1.00 ±.03	102.1 ±5.4	35.7 ±8.5	7.405 ±.008	7.195 ±.018
5 n = 5	2.61 ±0.11	46.0 ±2.6	2.73 ±0.04	1.05 ±.04	107.0 ±2.8	78.6 ±6.1	7.390 ±.005	7.159 ±.019
6 n = 5	2.63 ±0.18	46.0 ±3.5	2.75 ±0.14	1.05 ±.02	112.3 ±4.5	61.0 ±10.9	7.391 ±.008	7.168 ±.030
Final n = 5	2.73 ±0.14	47.8 ±1.9	2.75 ±0.12	1.01 ±.03	109.6 ±2.5	81.6 ±10.3	7.378 ±.007	7.150 ±.025
F	*4.84	*4.71			*2.65	*5.84	*5.63	*4.69

*significant α=.05 F ratio must exceed 2.42 to be significant.

†technical difficulties prevented determination of this parameter subject L.R.

Test 5, n=5 due to illness of subject C.M. Test 6, n=5 due to illness of subject L.R. Final Test, n=5 as data for subject L.R. not included in calculations due to adverse effect of bed rest on fitness.

6 subjects had repeat determinations within that range.

Maximal oxygen uptake did not increase over pre-training values until test 5 of training (Figure 1). Thereafter, it increased until training ended, the change at this point being significant ($p<.05$). The increase over pre-training values by the end of training was 12.8% (2.44 to 2.73 l/min). Three of the subjects (AK, LR, CS) attained $\dot{V}O_2$ max values over 50 ml/kg/min, and subject LR achieved 54.0 ml/kg/min immediately prior to her illness and bed rest. Subject CL had the most widely variable results during the training program. Data from test 4 and test 6 for subject CL (APPENDIX 1) suggests that she was not exhausted on these occasions.

Carbon dioxide production decreased significantly ($p<.05$) from the beginning of training to test 4 (2.79 to 2.46 l/min). It increased thereafter approximately to pre-training values (2.46 to 2.75 l/min). The respiratory exchange ratio decreased (1.14 to 1.01) throughout training coincident with the changes in $\dot{V}O_2$ max and $\dot{V}CO_2$.

Pulmonary ventilation during maximal exercise (Figure 2) significantly increased ($p<.05$) from pre-training values to those of test 6 (100.5 to 112.3 l/min BTPS). The time course of the increase was similar to that of maximal oxygen uptake, with the greatest increases occurring after test 5 (day 59 of training).

Figure 1 Maximal oxygen uptake with days of training. Each subjects results, the group mean and the standard error (shaded area) are illustrated.

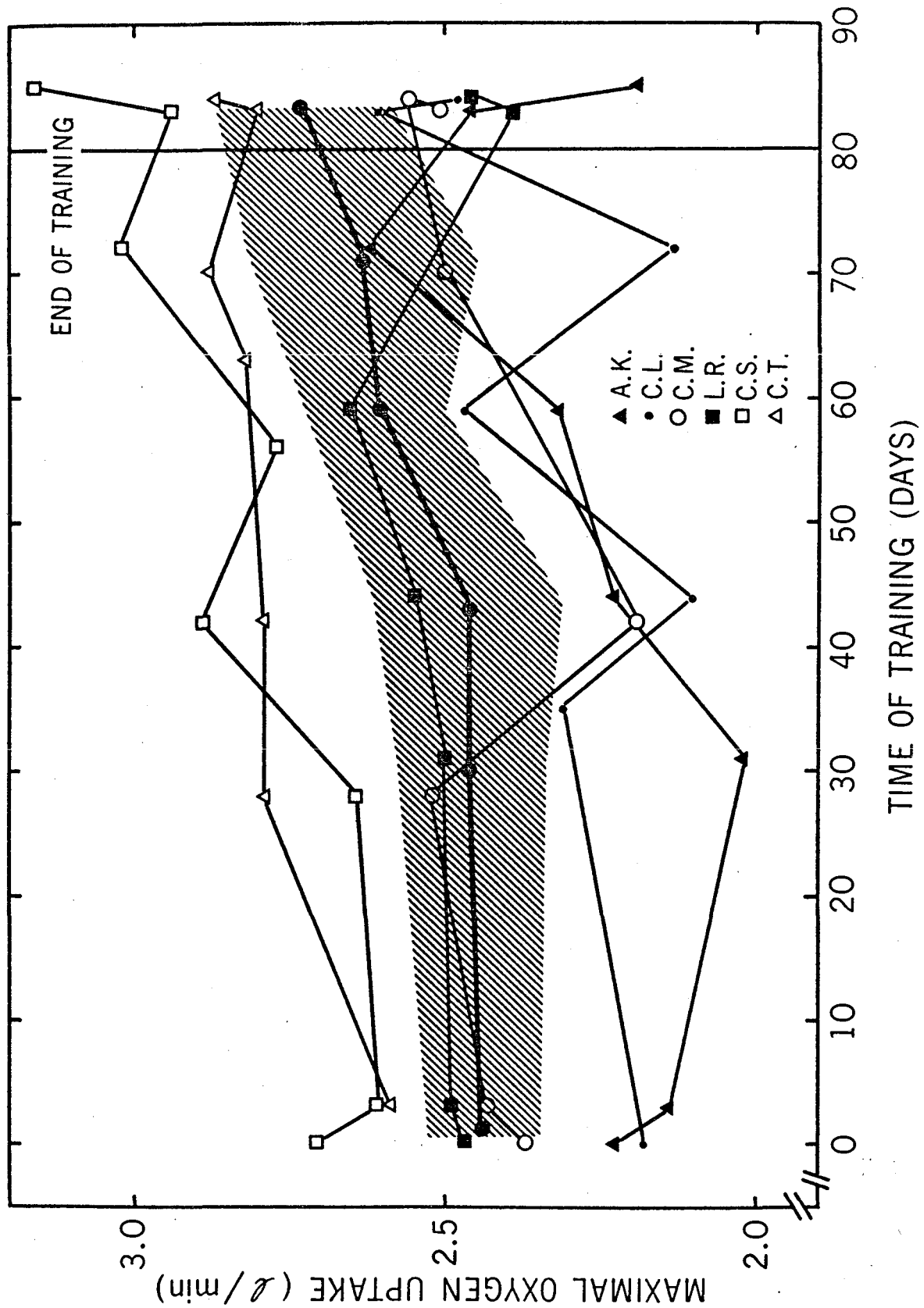

END OF TRAINING
A.K.
C.L.
C.M.
L.R.
C.S.
C.T.
MAXIMAL OXYGEN UPTAKE (ℓ/min)
3.0
2.5
2.0
0
10
20
30
40
50
60
70
80
90
TIME OF TRAINING (DAYS)

Figure 2 Pulmonary ventilation in maximal exercise with days of training. Each subject's data, the group mean and the standard error (shaded area) are illustrated.

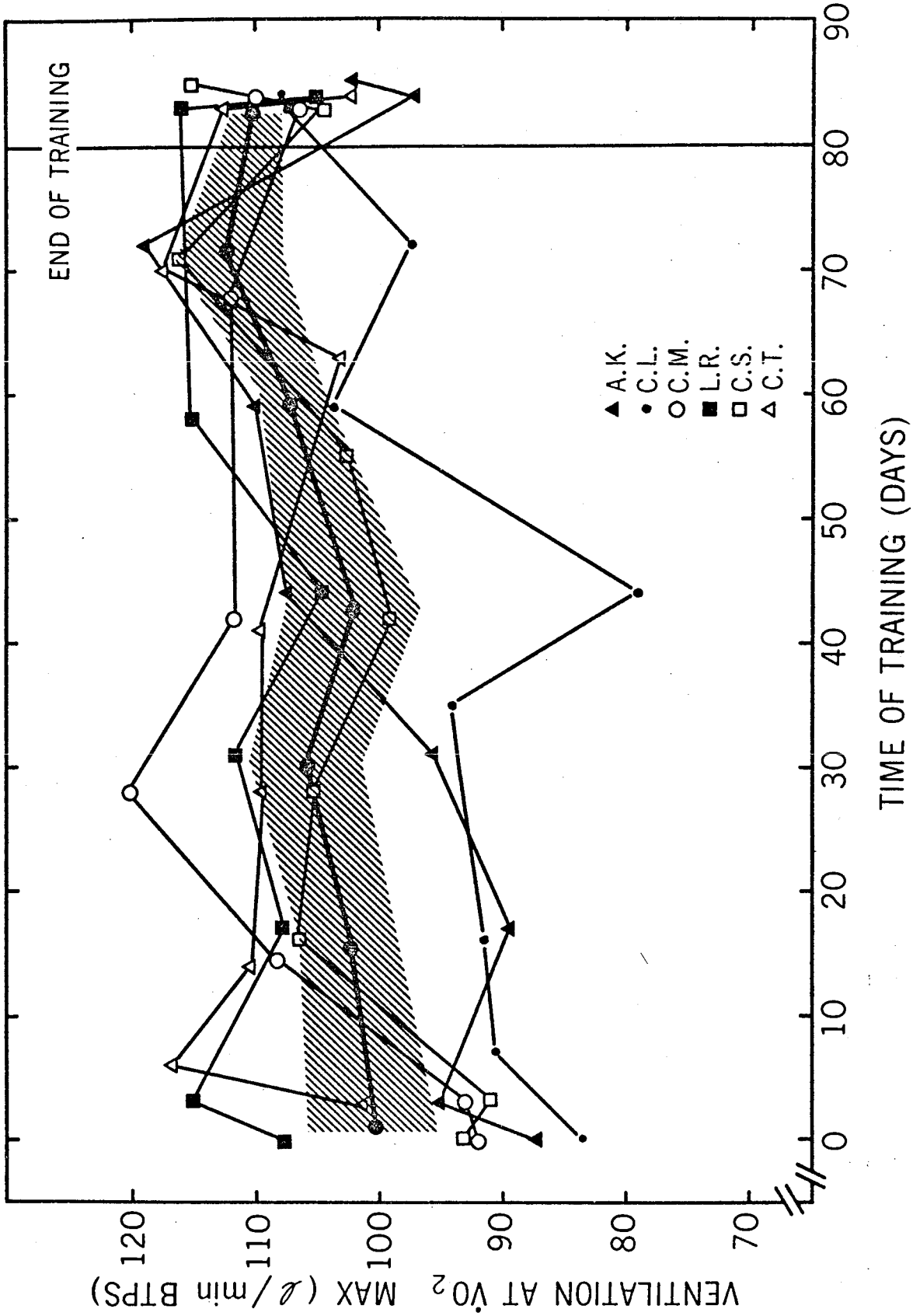

END OF TRAINING
A.K.
C.L.
C.M.
L.R.
C.S.
C.T.
VENTILATION AT $\dot{V}O_2$ MAX (ℓ/min BTPS)
120
110
100
90
80
70
0
10
20
30
40
50
60
70
80
90
TIME OF TRAINING (DAYS)

Figure 3 Post-exercise blood lactate accumulation with days of training. Each subject's data, the group mean and the standard error (shaded area) are illustrated.

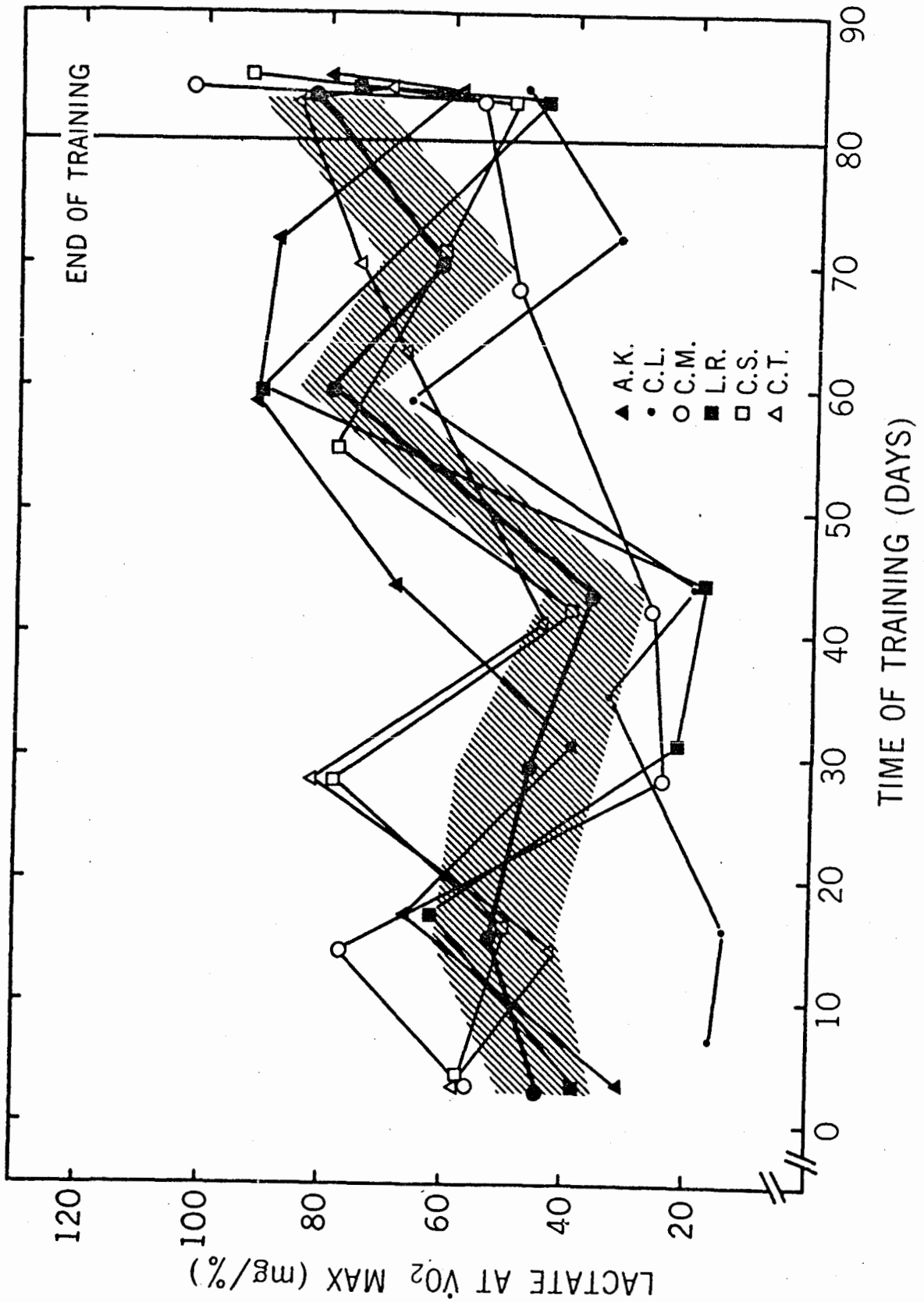
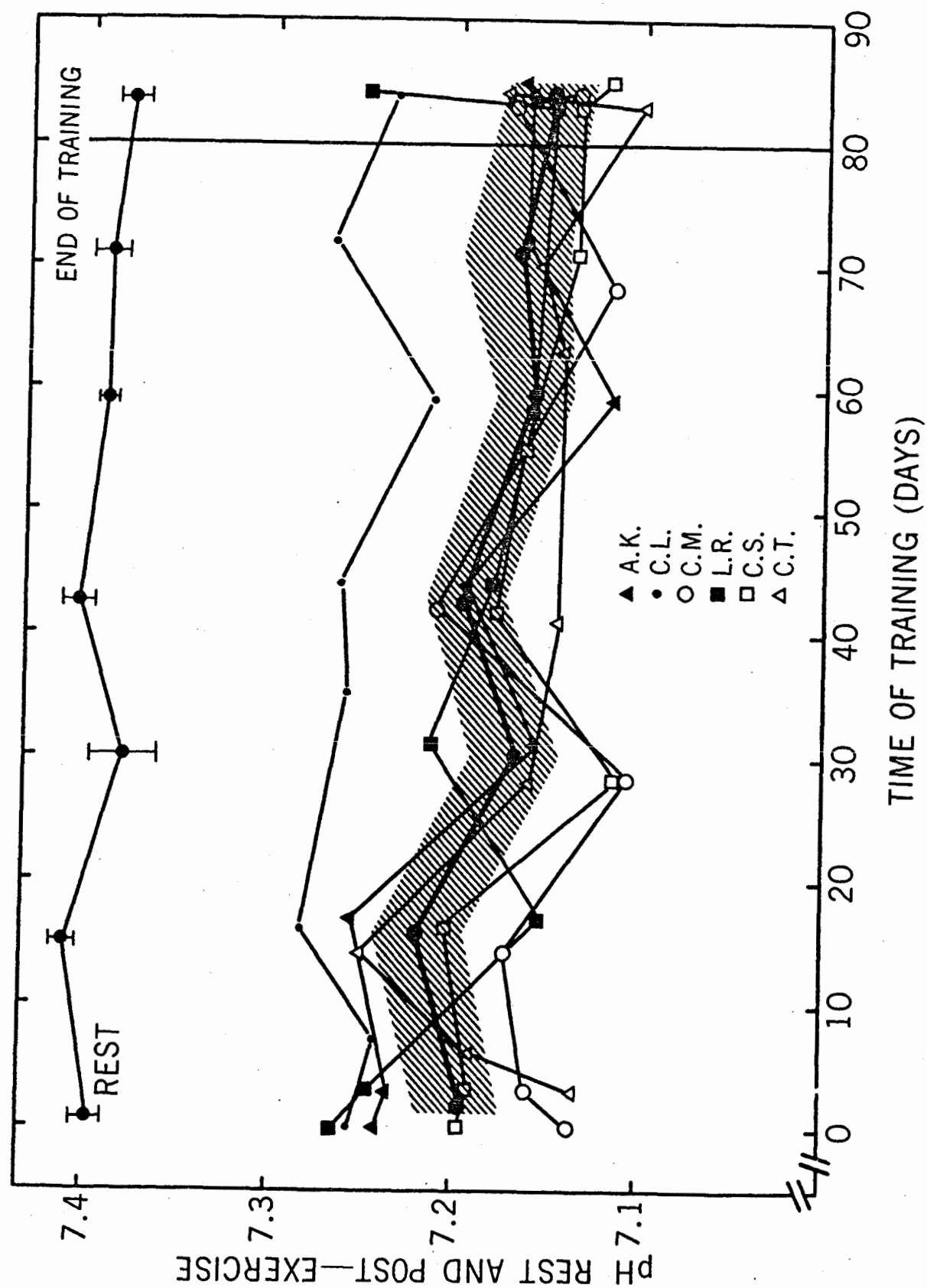

END OF TRAINING
▲ A.K.
• C.L.
○ C.M.
■ L.R.
□ C.S.
△ C.T.
LACTATE AT $\dot{V}O_2$ MAX (mg/%)
120
100
80
60
40
20
0
10
20
30
40
50
60
70
80
90
TIME OF TRAINING (DAYS)

Figure 4 Pre and post-exercise pH. Group mean and standard error only are illustrated for pre-exercise pH. Each subject's results, group mean and standard error (shaded area) are illustrated for post-exercise pH.


END OF TRAINING
REST
▲ A.K.
● C.L.
○ C.M.
■ L.R.
□ C.S.
△ C.T.
7.4
7.3
7.2
7.1
pH REST AND POST—EXERCISE
0
10
20
30
40
50
60
70
80
90
TIME OF TRAINING (DAYS)

The increase in post-exercise lactate accumulation (Figure 3) was significant ($p<.05$) from pre-training values by test 5 (day 59 of training). No increases in post-exercise lactate occurred during the first 40 days of training. Post-exercise pH values (Figure 4) reflected lactate accumulation. A significant decrease ($p<.05$) from test 2 (day 14 of training) occurred until the end of the training, illustrating an increased tolerance to acidemia resulting from severe exercise (7.220 to 7.150). Pre-exercise pH values exhibited no real trend during training. However, values for test 2 and test 4 during training were significantly higher ($p<.05$) than the values for test 3 and post-training values.

Circulatory Responses to Maximal Exercise (Table V, Figures 5 to 9)

Repeat determinations of cardiac output during maximal exercise for pre-training tests were within 1.0 l/min of each other. Reliability of post-training determinations was less precise, with repeat values within 15% of each other.

A slight, non-significant decline was noted for maximal cardiac output (Figure 5) from pre-training values to test 4 (15.3 to 14.7 l/min). Thereafter, cardiac output during maximal exercise increased steadily and significantly to the end of training ($p<.05$) (14.7 to 16.3 l/min), coincident with the increase in aerobic power. The mean increase from

TABLE V

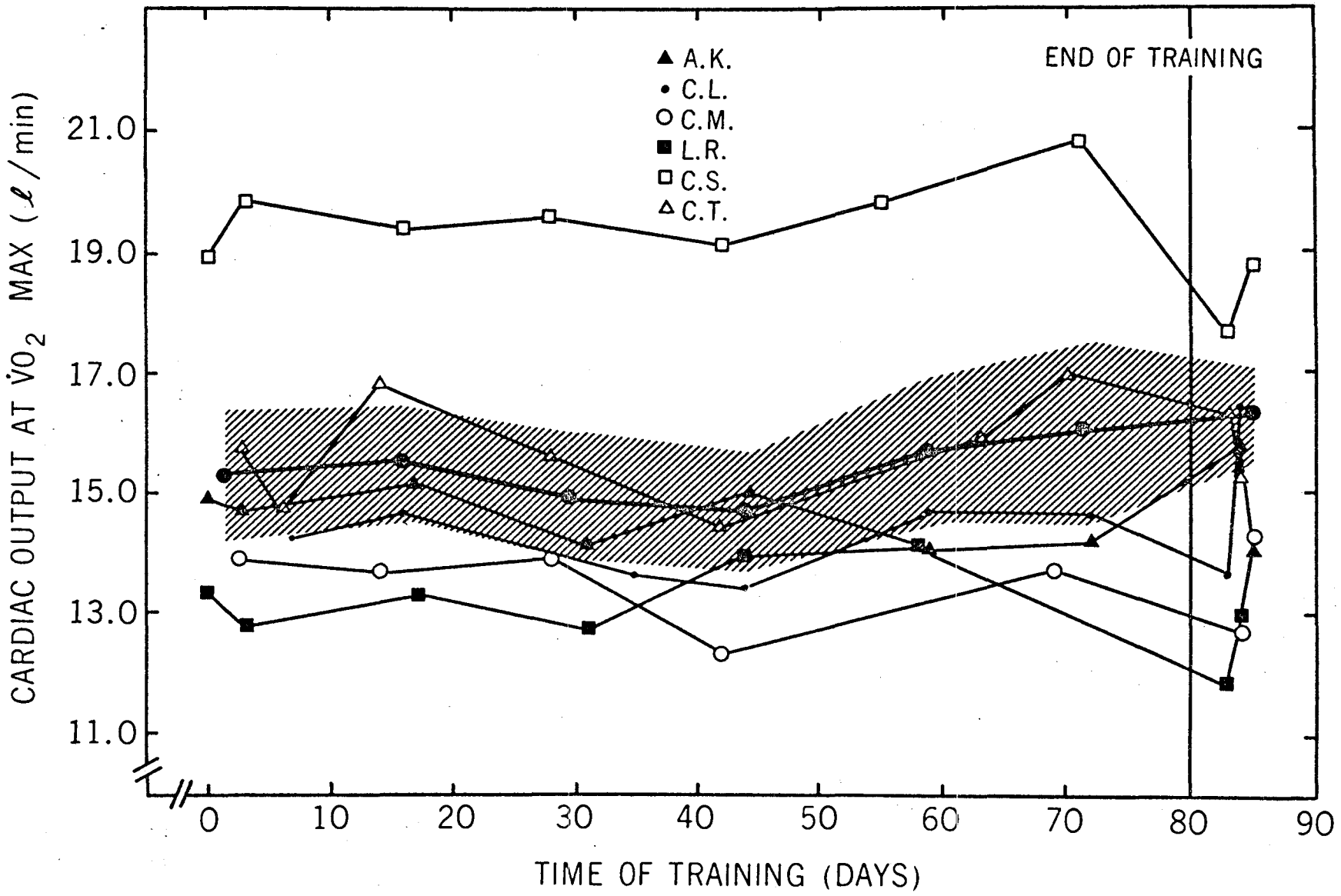
MAXIMAL OXYGEN UPTAKE AND CIRCULATORY RESPONSES IN MAXIMAL EXERCISE. GROUP MEANS AND STANDARD ERROR SHOWN FOR EACH TEST DURING TRAINING

Test	$\dot{V}O_2Max$ l/min	$\dot{Q}$ l/min	HR beats/min	SV ml	A-V O_2 diff. vol%	Hb-Pre g/100ml	Hb-Post g/100ml	Hct-Pre %	Hct-Post %
Initial n = 6	2.44 ±0.09	15.3 ±1.1	193 ±3.8	81 ±6	16.5 ±1.0	13.8 ±0.2	14.6 ±0.2	42.6 ±1.0	47.1 ±1.1
2 n = 6	† -	15.5 ±1.0	191 ±3.4	82 ±6	†-	12.6 ±0.2	14.8 ±0.4	40.7 ±1.2	46.5 ±0.5
3 n = 6	2.46 ±0.12	15.0 ±1.1	188 ±3.1	80 ±6	16.7 ±1.1	13.5 ±0.4	14.7 ±0.2	43.4 ±0.6	48.4 ±1.7
4 n = 6	2.46 ±0.15	14.7 ±1.0	185 ±3.3	80 ±5	16.8 ±0.8	13.2 ±0.3	14.4 ±0.3	43.3 ±1.9	46.8 ±1.3
5 n = 5	2.61 ±0.11	15.7 ±1.2	189 ±3.0	83 ±8	16.8 ±0.9	13.9 ±0.3	14.8 ±0.4	43.0 ±1.1	47.9 ±1.2
6 n = 5	2.63 ±0.18	16.0 ±1.5	185 ±3.3	87 ±8	16.6 ±1.0	13.5 ±0.3	14.4 ±0.2	41.9 ±1.0	44.9 ±2.0
Final n = 5	2.73 ±0.14	16.3 ±0.8	190 ±3.2	88 ±5	17.7 ±0.7	13.6 ±0.3	14.8 ±0.3	42.0 ±0.3	47.3 ±0.8
F	*4.84	*2.53	*6.67	2.03	1.29	*5.34	1.00	1.01	0.91

*significant α=.05 F ratio must exceed 2.42 to be significant.

†technical difficulties prevented determination of this parameter.

Figure 5 Cardiac output during maximal exercise with days of training. Each subjects results, the group mean and the standard error (shaded area) are illustrated.


▲ A.K.
• C.L.
○ C.M.
■ L.R.
□ C.S.
△ C.T.
END OF TRAINING
CARDIAC OUTPUT AT $\dot{V}O_2$ MAX (ℓ/min)
21.0
19.0
17.0
15.0
13.0
11.0
0
10
20
30
40
50
60
70
80
90
TIME OF TRAINING (DAYS)

pre-training values was 6.9% when each subject's highest value during the training program was compared to pre-training values. Subject C.S. attained a cardiac output of 20.8 l/min in test 6 (day 70 of training), the highest value recorded for this study.

Maximal heart rate declined significantly ($p<.05$) from pre-training values throughout training. By test 2 the decrease was already significant and it continued to decline until test 6 (193 to 185 beats/min). Although the post-training mean maximal heart rate was higher than test 6 (190 beats/min), it was still significantly lower ($p<.05$) than pre-training values.

The increased stroke volume during maximal exercise (Figure 6) occurring after test 4 (81 to 88 ml) was not significant. Stroke volume did not change during the first half of the training program and increased throughout the last half in a manner similar to the time course of change of aerobic power and cardiac output during maximal exercise. The total increase from pre-training values was 8.4% when each subject's highest value during the training program was compared to pre-training values.

The increase in arterio-venous oxygen difference (Figure 7) during maximal exercise was not significant. No changes occurred until post-training tests, when A-V O_2 difference increased from 16.6 to 17.7 vol%.

Figure 6 Stroke volume during maximal exercise with days of training. Each subject's data, the group mean and the standard error (shaded area) are illustrated.

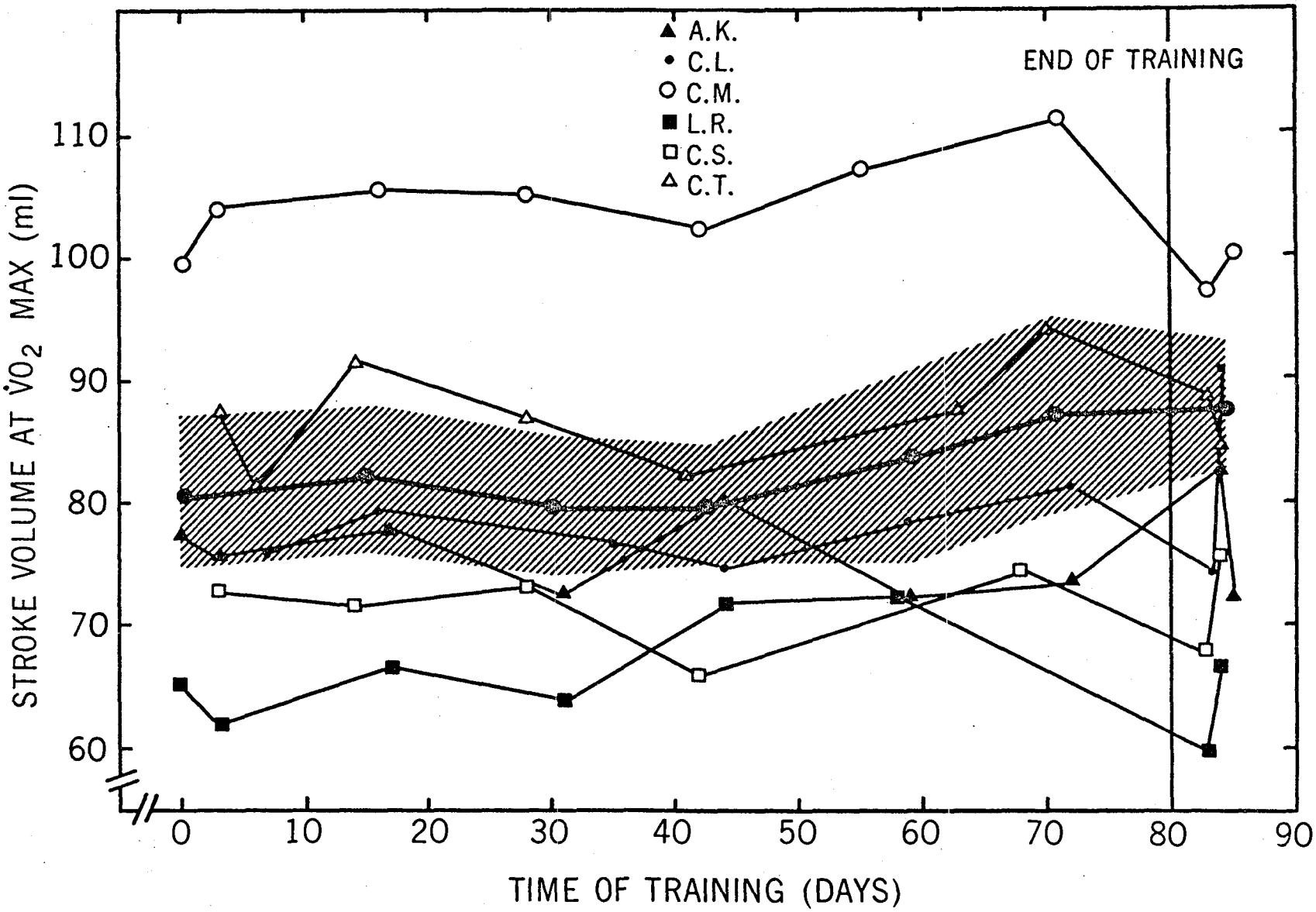

A.K.
C.L.
C.M.
L.R.
C.S.
C.T.
END OF TRAINING
STROKE VOLUME AT $\dot{V}O_2$ MAX (ml)
110
100
90
80
70
60
0
10
20
30
40
50
60
70
80
90
TIME OF TRAINING (DAYS)

Figure 7 Arterio-venous oxygen difference during maximal exercise with days of training. Each subject's data, the group mean and the standard error (shaded area) are illustrated.

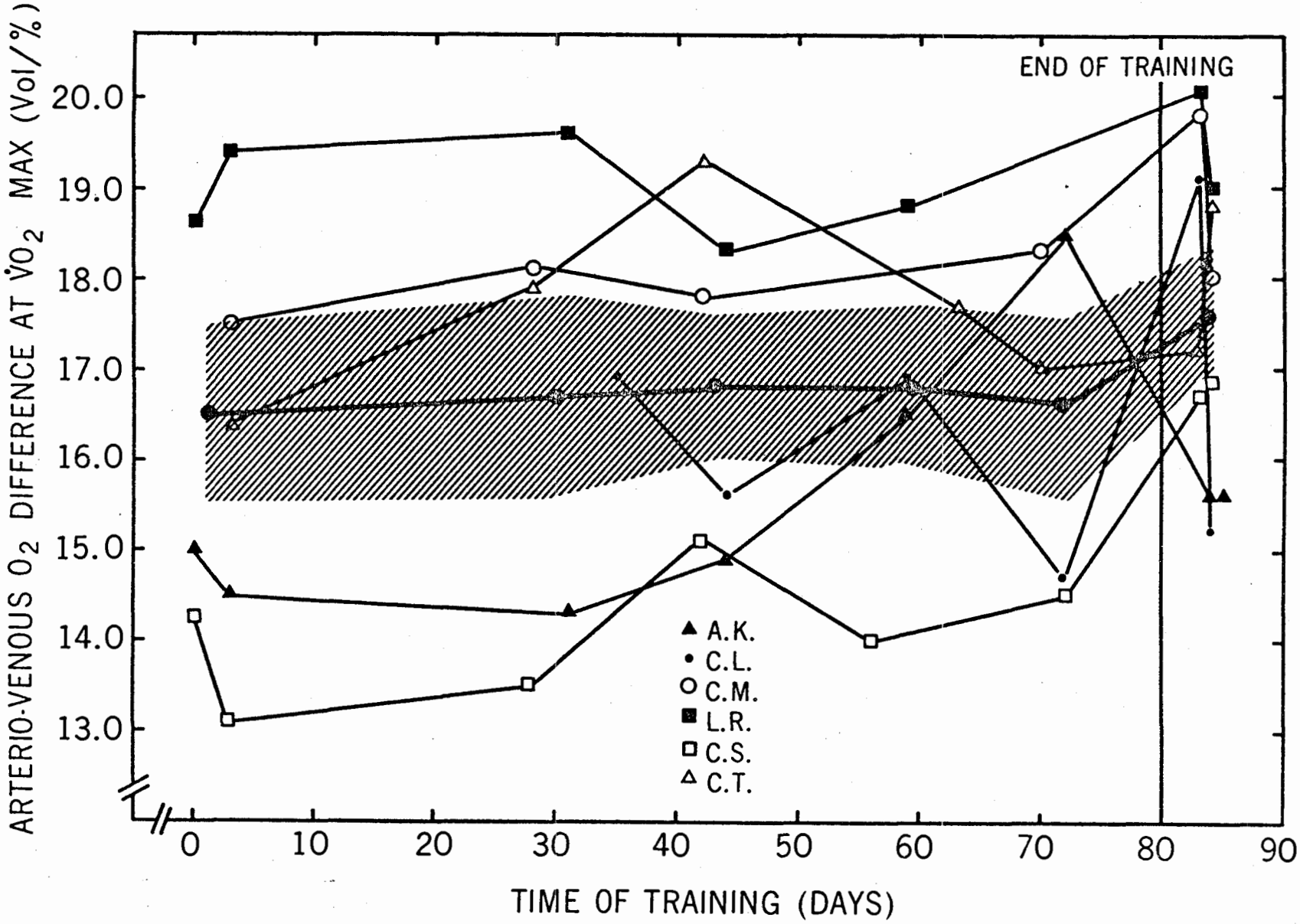
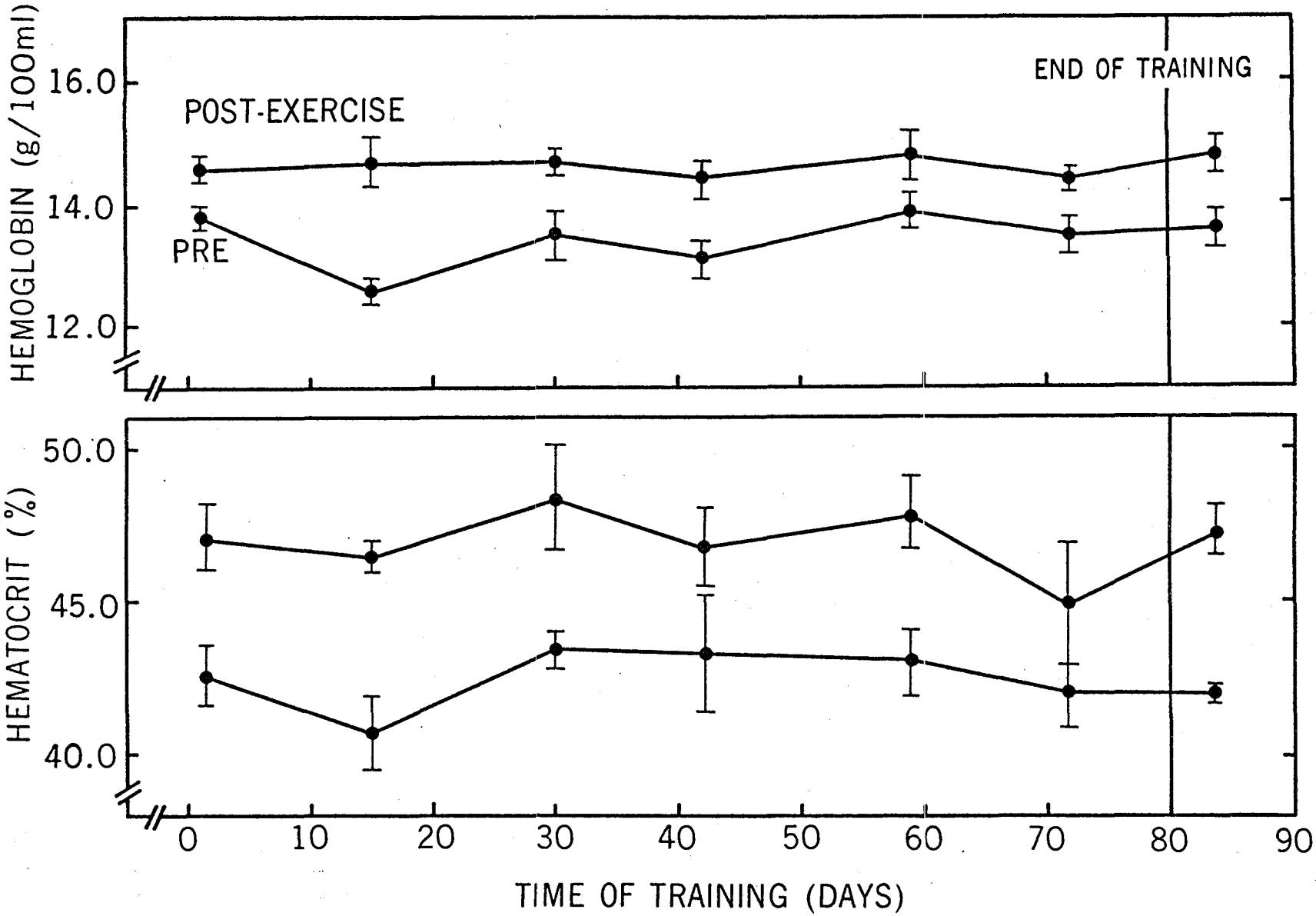

END OF TRAINING
ARTERIO-VENOUS O_2 DIFFERENCE AT $\dot{V}O_2$ MAX (Vol/%)
20.0
19.0
18.0
17.0
16.0
15.0
14.0
13.0
0
10
20
30
40
50
60
70
80
90
TIME OF TRAINING (DAYS)
A.K.
C.L.
C.M.
L.R.
C.S.
C.T.

Figure 8 Pre and post-exercise hemoglobin concentration and pre and post-exercise hematocrit with days of training. Group means and standard errors are illustrated.


HEMOGLOBIN (g/100ml)
16.0
14.0
12.0
POST-EXERCISE
PRE
END OF TRAINING
HEMATOCRIT (%)
50.0
45.0
40.0
0
10
20
30
40
50
60
70
80
90
TIME OF TRAINING (DAYS)

The oxygen carrying capacity of the blood was generally unaffected by training, as noted in the time course of hemoglobin concentration and hematocrit with days of training (Figure 8). However, pre-exercise hemoglobin concentration was significantly lower ($p<.05$) in test 2 (12.6 g/100ml) than any other test during training. Pre-exercise hematocrit was also lower in test 2 (day 14 of training) but not significantly so. Post-exercise values of hemoglobin and hematocrit were 5 to 10% higher than their pre-exercise values throughout the training program.

Cardiorespiratory Responses to Standard Submaximal Exercise (Table VI, Figures 9 to 15)

Figures 9, 10 and 12 for oxygen uptake, carbon dioxide production and cardiac output show the percentage change of subsequent tests from the mean of the two pretraining tests for each subject. Two subjects, CL and LR, were working at 300 kpm/min. Absolute values of exygen uptake, carbon dioxide production and cardiac output are directly related to the work rate. Subject CL and subject LR had markedly lower values for these variables, therefore, than the subjects performing a higher work load. In this respect pooling the absolute data from all 6 subjects to derive group means for tests throughout training for

TABLE VI

CARDIORESPIRATORY VARIABLES FOR STANDARD SUBMAXIMAL EXERCISE. GROUP MEAN AND STANDARD ERROR SHOWN FOR EACH TEST DURING TRAINING

Test	$\dot{V}O_2$ l/min	$\dot{V}CO_2$ l/min	R	$\dot{V}E$ l/min	$\dot{Q}$ l/min	HR beats/min	SV ml	A-V O_2 diff vol %
Initial n = 6	1.29 ±.06	1.06 ±.05	0.80 ±.02	37.2 ±0.6	9.4 ±.7	150 ±4.0	63 ±3.4	13.7 ±0.4
2 n = 6	†-	1.02 ±.05	†-	38.0 ±1.1	9.2 ±.7	140 ±7.4	66 ±5.9	†- †-
3 n = 6	1.21 ±.05	1.02 ±.06	0.84 ±.03	35.9 ±2.4	9.4 ±.8	137 ±6.3	69 ±6.7	13.0 ±1.0
4 n = 6	1.30 ±.07	0.95 ±.06	0.73 ±.02	35.2 ±1.1	9.0 ±.6	134 ±7.2	68 ±5.4	14.6 ±0.7
5 n = 5	1.15 ±.09	0.91 ±.07	0.80 ±.01	33.8 ±1.7	9.0 ±.7	129 ±6.1	70 ±6.5	12.8 ±0.6
6 n = 5	1.27 ±.08	1.01 ±.10	0.79 ±.03	36.7 ±1.7	9.4 ±1.1	133 ±9.0	71 ±5.0	13.8 ±0.7
Final n = 5	1.30 ±.06	1.00 ±.07	0.75 ±.02	35.9 ±1.5	8.7 ±0.4	132 ±6.1	65 ±3.4	15.3 ±0.7
F	1.69	*3.90		1.24	1.77	*5.07	1.01	2.26

*significant α=.05 F ratio must exceed 2.42 to be significant.

†technical difficulties prevented determination of this parameter.

illustration purposes may not be as precise as using the mean percentage change from original pre-training values. The submaximal work rate was designated before training began in order to impose approximately the same relative stress on each subject in terms of heart rate response or percentage of maximal oxygen uptake. Therefore, using mean absolute data of other variables such as heart rate, stroke volume, ventilation and arterio-venous oxygen difference does in fact seem justified.

Oxygen uptake exhibited no significant change over the duration of the training program (Figure 9). The time course of submaximal oxygen uptake with training suggests a slight decrease (i.e. increased mechanical efficiency). Carbon dioxide production (Figure 10), however, significantly decreased ($p<.05$) by the end of the training period (1.06 to 1.00 l/min). The gradual decrease in submaximal $\dot{V}CO_2$ became significant ($p<.05$) by test 4 during training. The respiratory exchange ratio exhibited no clear trend, although a decrease occurred by the end of training (0.80 to 0.75).

Pulmonary ventilation for standard submaximal exercise (Figure 11) decreased slightly but not significantly (37.2 to 35.9 l/min BTPS).

Cardiac output during submaximal exercise (Figure 12) decreased by the end of training (10.5%), although the change was not significant. Slight variations occurred throughout

Figure 9 Oxygen uptake for standard submaximal exercise with days of training. Each subject's changes, the group mean and the standard error (shaded area) are illustrated.

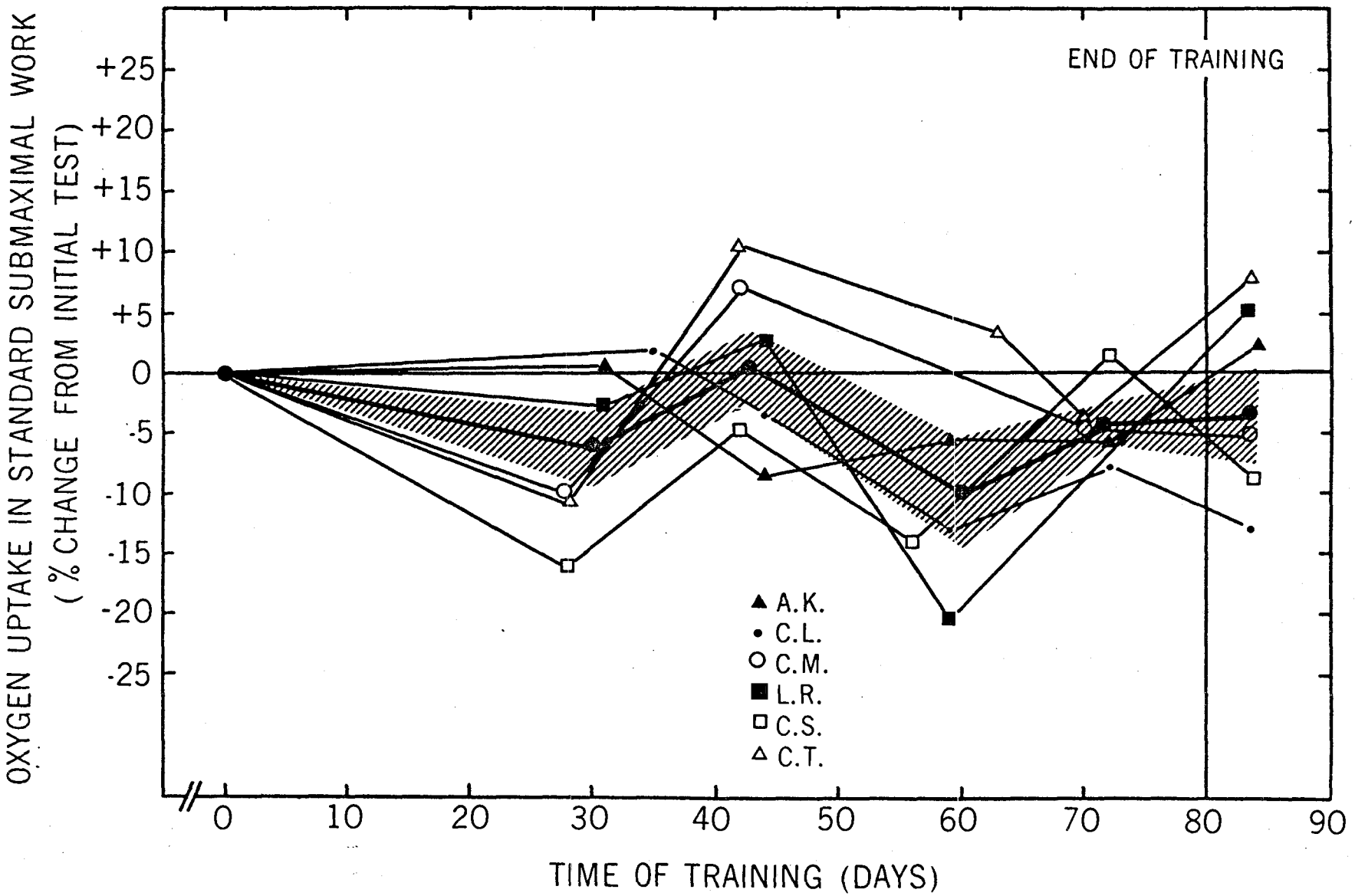

OXYGEN UPTAKE IN STANDARD SUBMAXIMAL WORK
(% CHANGE FROM INITIAL TEST)
+25
+20
+15
+10
+5
0
-5
-10
-15
-20
-25
END OF TRAINING
A.K.
C.L.
C.M.
L.R.
C.S.
C.T.
0
10
20
30
40
50
60
70
80
90
TIME OF TRAINING (DAYS)

Figure 10 Carbon dioxide production for standard sub-maximal exercise with days of training. Each subject's changes, the group mean and the standard error (shaded area) are illustrated.

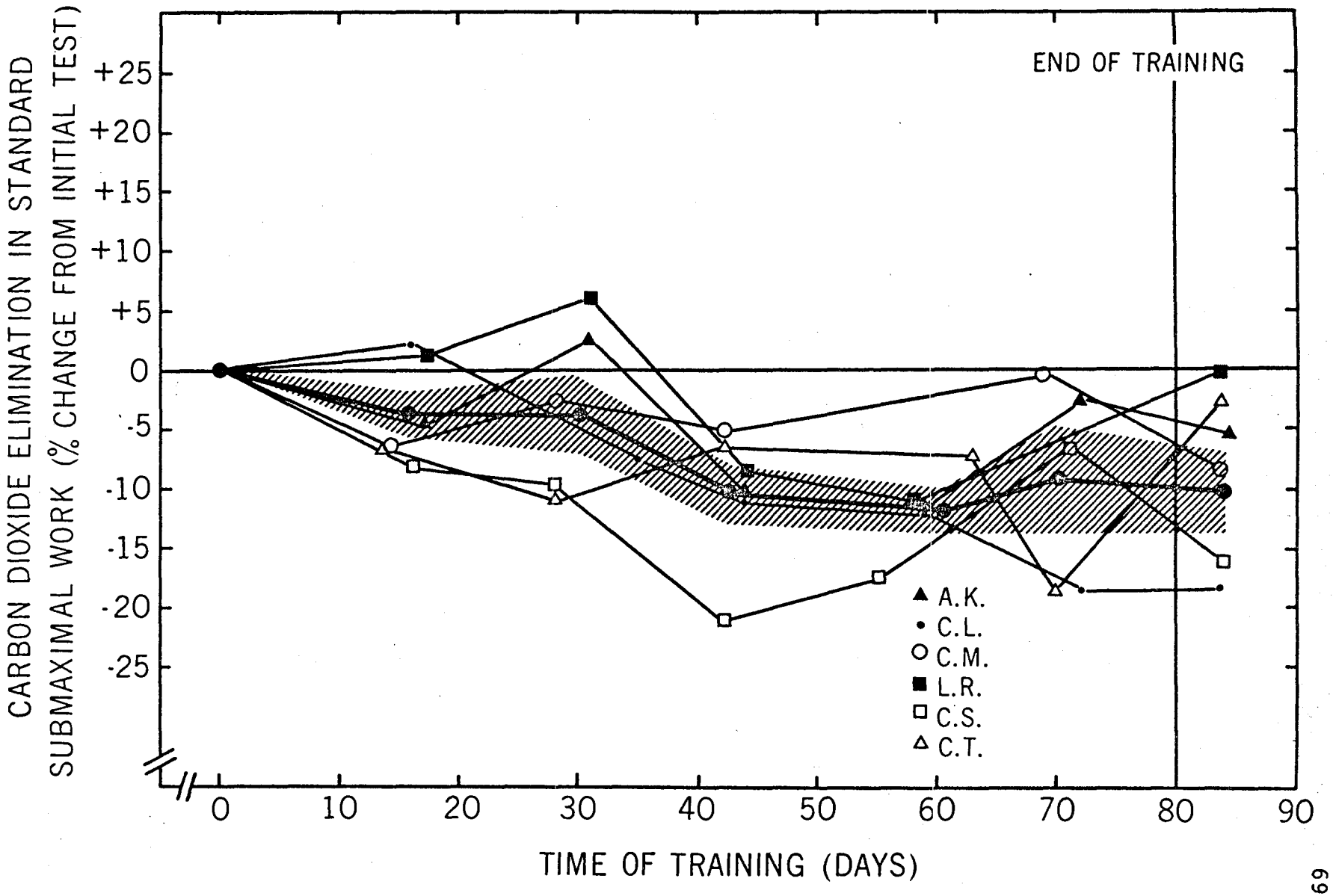

CARBON DIOXIDE ELIMINATION IN STANDARD SUBMAXIMAL WORK (% CHANGE FROM INITIAL TEST)
+25
+20
+15
+10
+5
0
-5
-10
-15
-20
-25
END OF TRAINING
A.K.
C.L.
C.M.
L.R.
C.S.
C.T.
0
10
20
30
40
50
60
70
80
90
TIME OF TRAINING (DAYS)

Figure 11 Pulmonary ventilation for standard submaximal exercise with days of training. Each subject's data, the group mean and the standard error (shaded area) are illustrated.

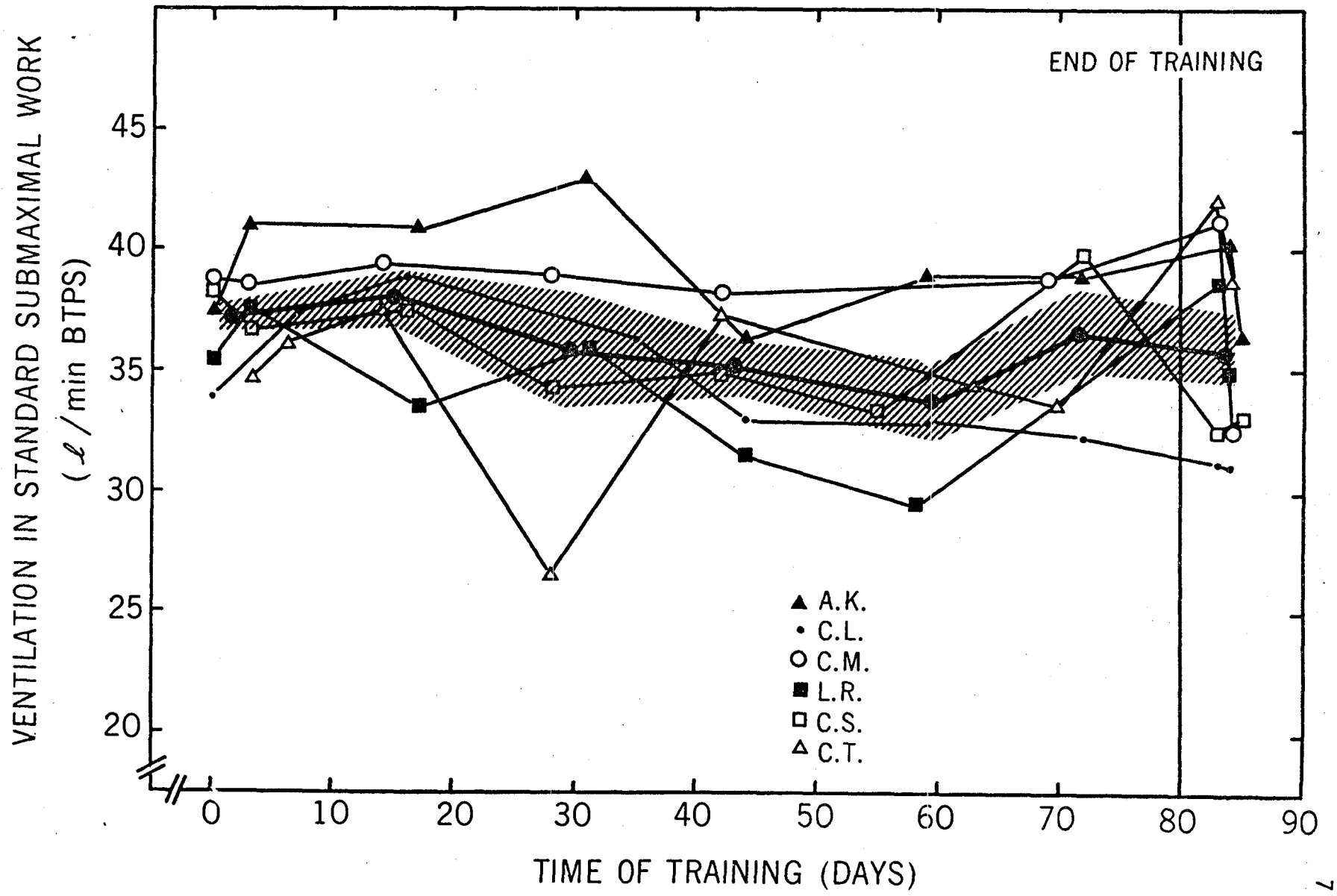
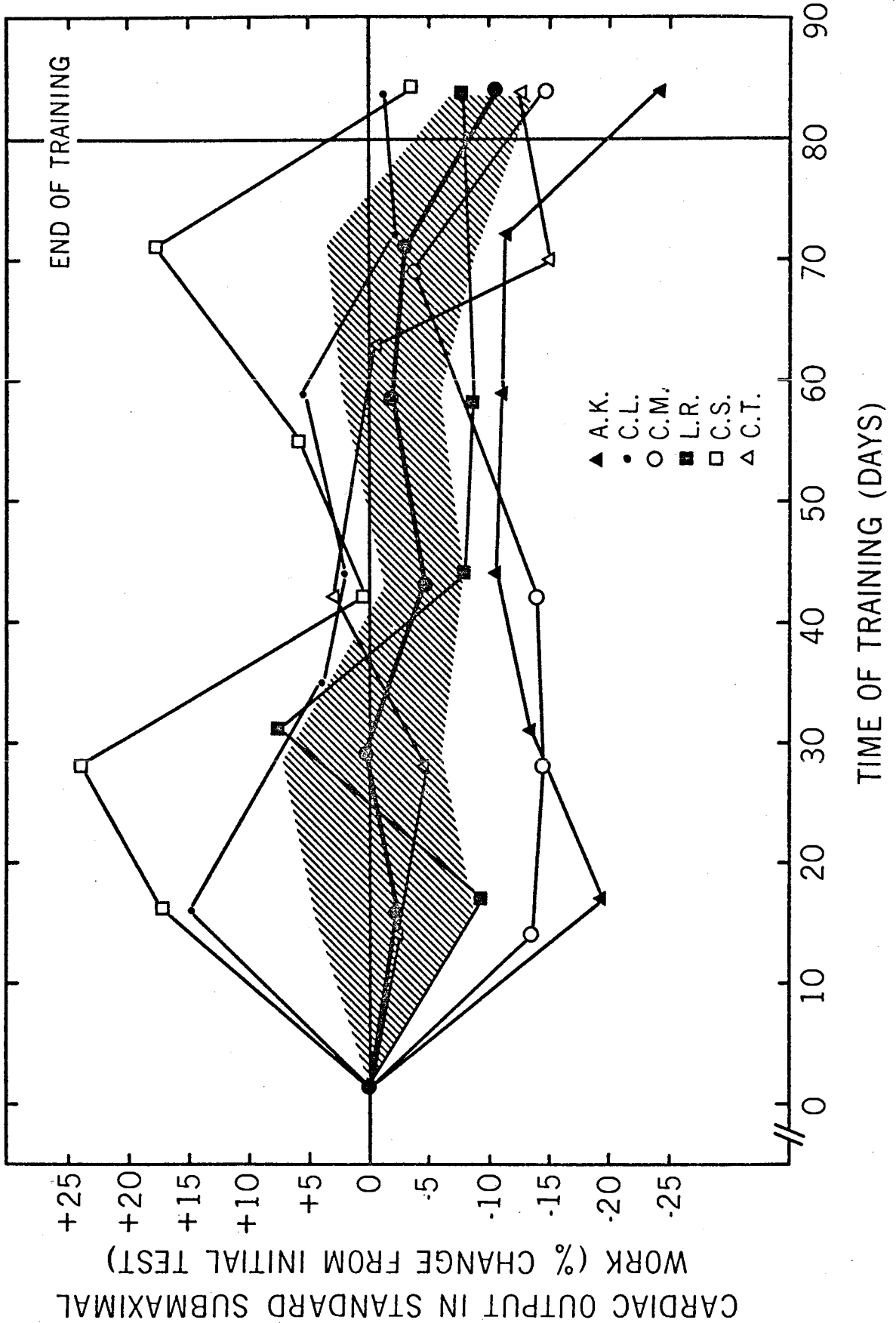

END OF TRAINING
VENTILATION IN STANDARD SUBMAXIMAL WORK
(ℓ/min BTPS)
45
40
35
30
25
20
0
10
20
30
40
50
60
70
80
90
TIME OF TRAINING (DAYS)
A.K.
C.L.
C.M.
L.R.
C.S.
C.T.

Figure 12 Cardiac output for standard submaximal exercise with days of training. Each subject's changes, the group mean and the standard error (shaded area) are illustrated.


END OF TRAINING
A.K.
C.L.
C.M.
L.R.
C.S.
C.T.
+25
+20
+15
+10
+5
0
-5
-10
-15
-20
-25
0
10
20
30
40
50
60
70
80
90
TIME OF TRAINING (DAYS)
CARDIAC OUTPUT IN STANDARD SUBMAXIMAL WORK (% CHANGE FROM INITIAL TEST)

training until post-training tests, when a marked decline was noted. Heart rate for standard submaximal exercise (Figure 13) declined steadily from the onset of training until test 6 (150 to 129 beats/min). The decline was significant ($p<.05$), with the significance first occurring at test 2 during training.

The observed increase in stroke volume for standard submaximal exercise (figure 14) up to test 6 (63 to 71 ml) was not statistically significant. Post-training tests exhibited a drop in submaximal stroke volume (65 ml).

Arterio-venous oxygen difference (Figure 15) did not change significantly for submaximal exercise throughout training since oxygen uptake and cardiac output varied little. With the decline of cardiac output in the post-training tests, however, A-V O_2 difference increased markedly (13.8 to 15.3 vol %).

Training resulted in a gradually declining heart rate coincident with an increasing stroke volume. Cardiac output, oxygen uptake and hence arterio-venous oxygen difference remained virtually constant.

The drop in cardiac output in the post-training test with an unchanged oxygen uptake was accompanied by an increased A-V O_2 difference.

The relationship between oxygen uptake and cardiac output in their response to submaximal and maximal exercise before and after training is illustrated in Figure 16.

Figure 13 Heart rate for standard submaximal exercise with days of training. Each subject's data, the group mean and the standard error (shaded area) are illustrated.

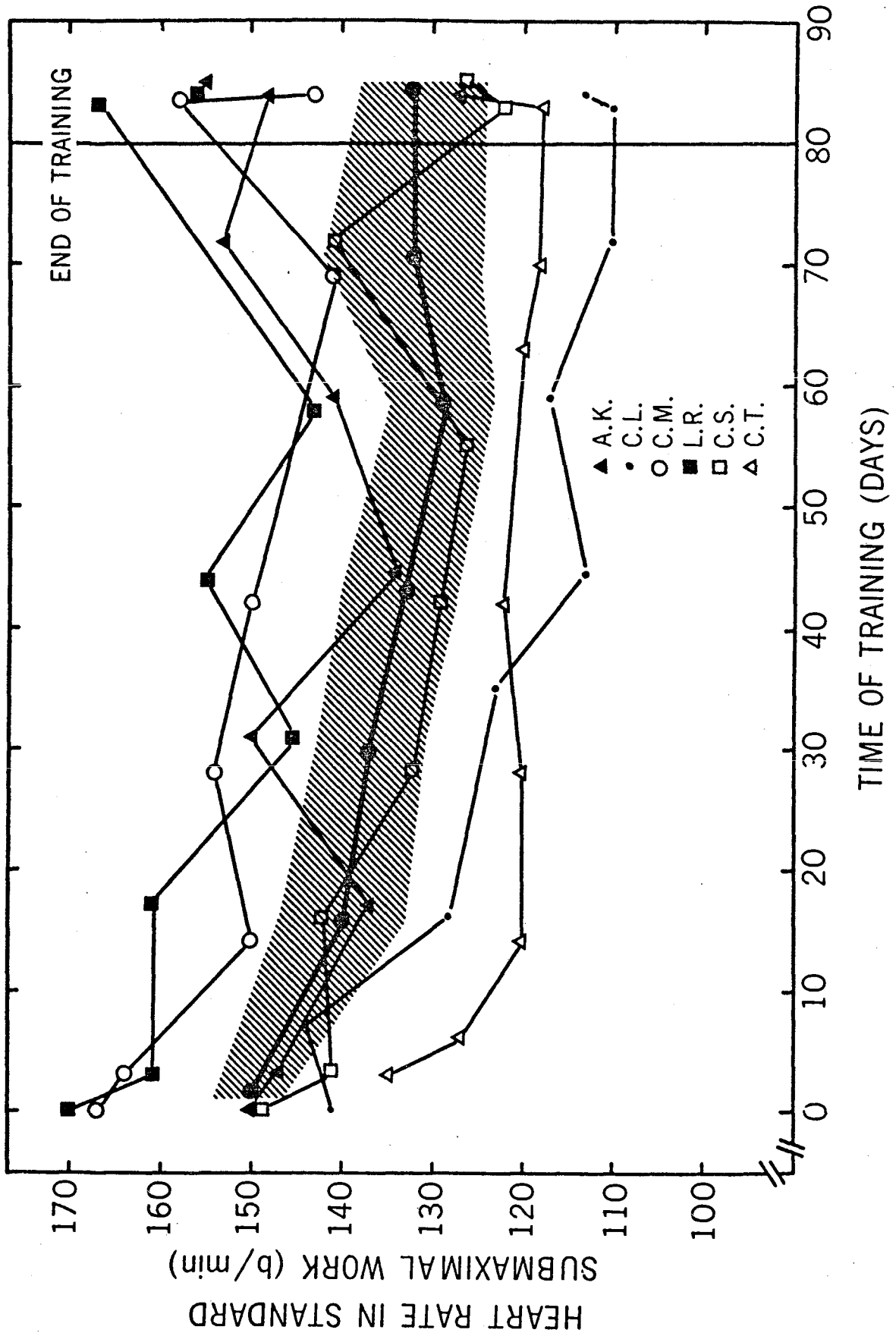

END OF TRAINING
A.K.
C.L.
C.M.
L.R.
C.S.
C.T.
HEART RATE IN STANDARD SUBMAXIMAL WORK (b/min)
TIME OF TRAINING (DAYS)
170
160
150
140
130
120
110
100
0
10
20
30
40
50
60
70
80
90

Figure 14 Stroke volume for standard submaximal exercise with days of training. Each subject's data, the group mean and the standard error (shaded area) are illustrated.

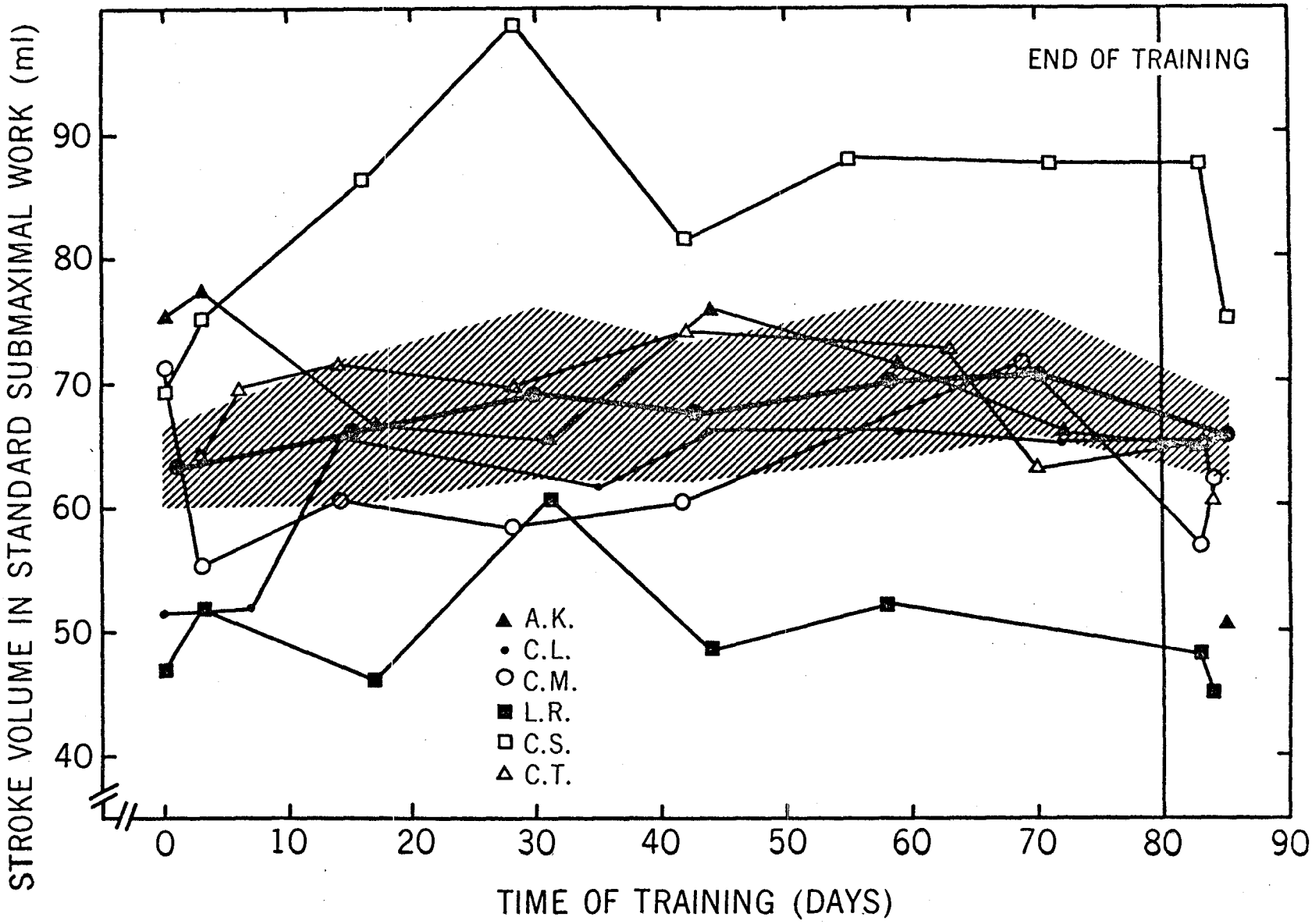

END OF TRAINING
STROKE VOLUME IN STANDARD SUBMAXIMAL WORK (ml)
TIME OF TRAINING (DAYS)
A.K.
C.L.
C.M.
L.R.
C.S.
C.T.
40
50
60
70
80
90
0
10
20
30
40
50
60
70
80
90

Figure 15 Arterio-venous oxygen difference for standard submaximal exerxise with days of training. Each subject's data, the group mean and the standard error (shaded area) are illustrated.

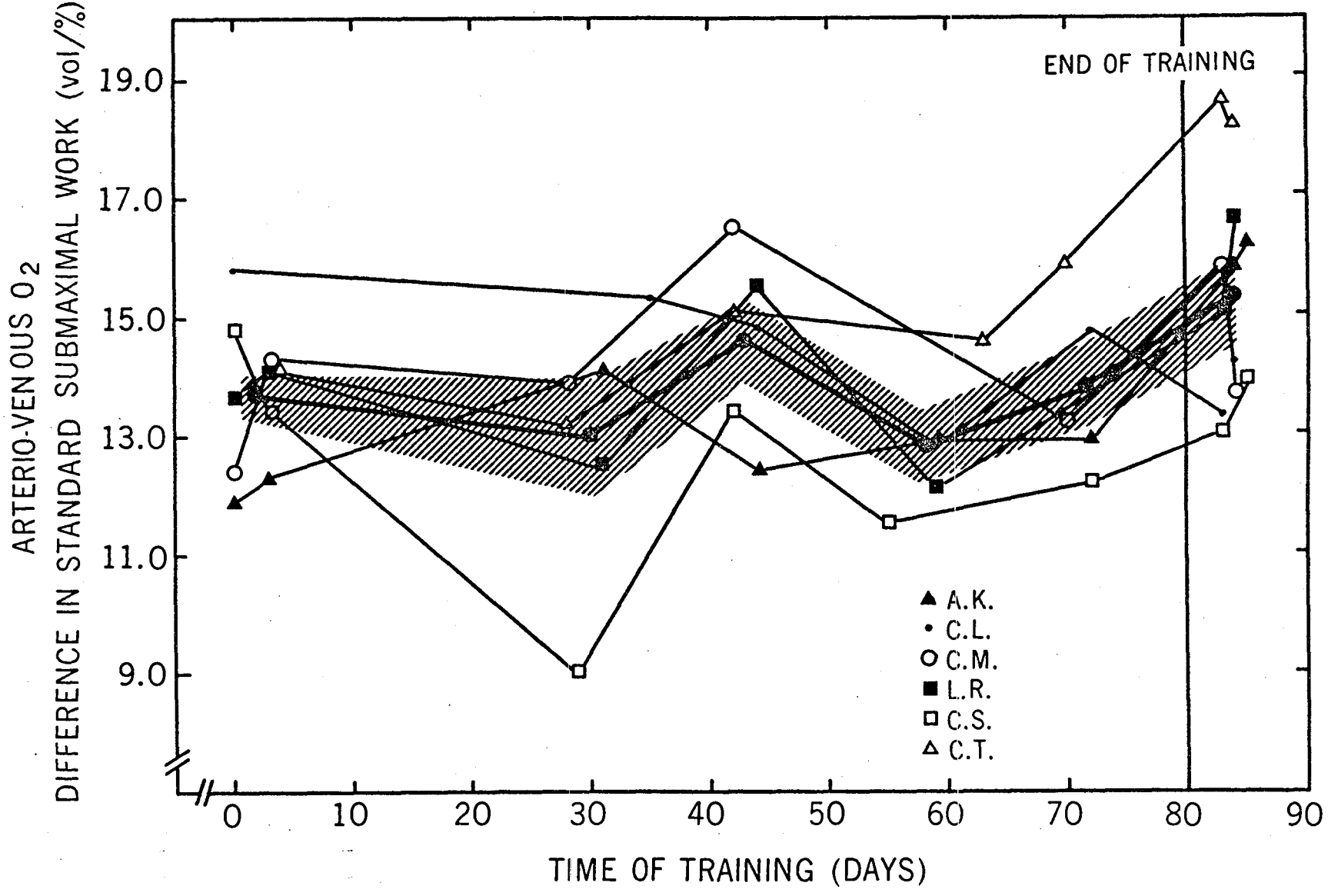
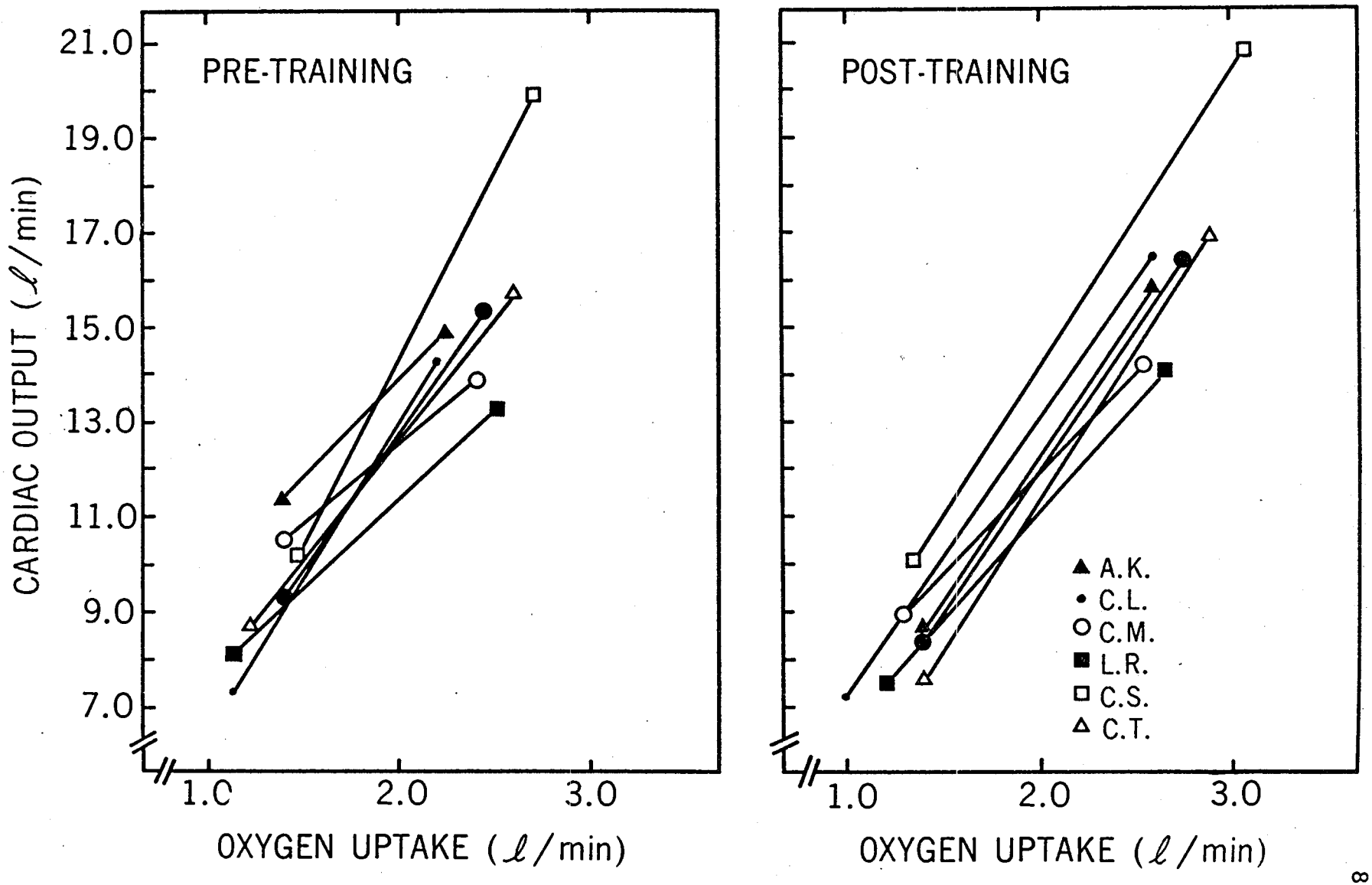

ARTERIO-VENOUS O_2 DIFFERENCE IN STANDARD SUBMAXIMAL WORK (vol/%)
19.0
17.0
15.0
13.0
11.0
9.0
END OF TRAINING
A.K.
C.L.
C.M.
L.R.
C.S.
C.T.
0
10
20
30
40
50
60
70
80
90
TIME OF TRAINING (DAYS)

Figure 16 The relationship of oxygen uptake and cardiac output in their response to exercise of increasing severity before and after training. Lines for each subject have been drawn by joining points for submaximal and maximal exercise. The group mean response line is illustrated by the heavy, dark line.


PRE-TRAINING
POST-TRAINING
CARDIAC OUTPUT (ℓ/min)
21.0
19.0
17.0
15.0
13.0
11.0
9.0
7.0
1.0
2.0
3.0
OXYGEN UPTAKE (ℓ/min)
A.K.
C.L.
C.M.
L.R.
C.S.
C.T.

Group mean response lines are shown as well as those for individual subjects. It can be seen that training resulted in a higher maximal oxygen uptake accompanied by a higher cardiac output. Standard submaximal exercise elicited an identical oxygen uptake after training with a reduced cardiac output.

Pulmonary Function Variables at Rest (Table VII)

No significant changes occurred in minute ventilation, tidal volume and forced expiratory volume in 1 second. Vital capacity remained relatively constant (3.56 to 3.72 l BTPS) except for test 2 (3.401 BTPS) which was found to be significantly different from the mean values if all other tests ($p<.05$). Breathing frequency at rest declined from 16.5 breaths/min pre-training to 13.5 breaths/min in test 6, a significant decrease ($p<.05$).

TABLE VII

PULMONARY FUNCTION AT REST. GROUP MEAN AND STANDARD ERROR SHOWN FOR EACH TEST DURING TRAINING

Test	MV l/min BTPS	f breaths/min	TV ml BTPS	VC l BTPS	$FEV_{1.0}$ % of VC
Initial n = 6	10.0 ±1.0	16.3 ±0.9	632.1 ±73.5	3.61 ±.22	87.2 ±4.4
2 n = 6	10.6 ±2.0	15.8 ±1.2	697.7 ±159.8	3.40 ±.19	93.6 ±1.3
3 n = 6	9.5 ±1.0	14.8 ±1.1	658.8 ±85.0	3.60 ±.23	87.6 ±1.2
4 n = 6	9.5 ±1.5	15.3 ±1.0	631.2 ±115.6	3.56 ±.27	92.7 ±1.8
5 n = 5	10.8 ±1.8	13.8 ±1.4	843.2 ±243.6	3.85 ±.22	90.0 ±1.6
6 n = 5	10.0 ±1.7	13.0 ±1.3	815.6 ±212.4	3.66 ±.28	91.1 ±1.9
Final n = 5	10.2 ±1.1	14.8 ±0.8	739.3 ±115.8	3.72 ±.21	94.5 ±1.7
F	0.40	*2.89	1.10	*3.60	1.89

*significant α=.05 F ratio must exceed 2.42 to be significant.

TABLE VIII

LOCATION OF SIGNIFICANT DIFFERENCES BETWEEN MEANS DETERMINED BY NEWMAN-KEULS METHOD. TESTS ARRANGED IN ASCENDING ORDER OF MAGNITUDE OF MEANS. UNDERLINING JOINING TEST NUMBERS INDICATES NO SIGNIFICANT DIFFERENCE BETWEEN MEANS

Parameter	Test Number
A. Maximal Exercise	
$\dot{V}O_2$	1 3 4 5 6 7
$\dot{V}CO_2$	4 3 2 5 6 7 1
$\dot{V}E$	1 4 2 3 5 7 6
La	4 1 3 2 6 5 7
pH-pre	7 3 5 6 1 4 2
pH-post	7 5 3 6 4 1 2
$\dot{Q}$	4 3 1 2 5 6 7
HR	4 6 3 5 7 2 1
Hb-pre	2 4 3 6 7 1 5
B. Submaximal Exercise	
$\dot{V}CO_2$	5 4 7 6 3 2 1
HR	5 7 6 4 3 2 1
C. Pulmonary Function	
f	6 5 7 3 4 2 1
VC	2 4 3 1 6 7 5

See Tables IV, V, VI and VII for means and standard errors.

CHAPTER V

DISCUSSION

Cardiorespiratory Parameters - Pre-Training

As previously defined, maximal oxygen uptake ($\dot{V}O_2$ max) serves as a measure of the functional capacity of the cardiorespiratory system. When expressed in milliliters of oxygen consumed per kilogram of body weight per minute (ml/kg/min), it is a measure of the energy from aerobic processes made available for metabolic and physical work. It has been commonly accepted as a criterion of physical fitness, and its value has ranged from 20 to 25 ml/kg/min for sedentary males and females (Astrand, 1960; Siegel, Blomquist and Mitchell, 1970) to 85 ml/kg/min for a champion athlete (Saltin and Astrand, 1967).

Previous investigations of untrained Scandinavian females have reported values of $\dot{V}O_2$ max ranging from 38 to 48 ml/kg/min (Astrand, 1952; Astrand, 1960; Astrand et al., 1964; Hermansen and Andersen, 1965). Studies on North American college-age females generally have reported lower values, ranging from 30 to 39 ml/kg/min. The mean initial $\dot{V}O_2$ max of 44 ml/kg/min (2.44l/min) for six college girls in the present study is therefore fairly high by North American standards. Astrand's (1952) finding of 48 ml/kg/min for

active university physical education students was the only study to exceed 41 ml/kg/min in Scandinavia. Since only subject C.L. (34.5 ml/kg/min) had a $\dot{V}O_2$ max below 42 ml/kg/min, it would appear that the subjects of this study did not belong in a "sedentary" category of fitness. Former participation in track in high school of three subjects (A.K., C.M. and L.R.) and intercollegiate basketball by subject C.T. would tend to support this conclusion.

The subjects of the present study also demonstrated much higher levels of pulmonary ventilation in maximal work than reported elsewhere. The mean value of the six subjects was 100.5 l/min (BTPS). Horvath and Michael's data (1970) showed a mean $\dot{V}E$ max of 60 l/min (BTPS) and Moody, Kollias and Buskirk (1969) found a $\dot{V}E$ max of 63 l/min (BTPS) in their subjects. The female students studied by Hermansen and Andersen (1965) had a mean $\dot{V}E$ max of 71.0 l/min (BTPS). The ventilations found in the present study compare favourably to those of female athletes studied by Hermansen and Andersen (1965) and Sprynarova and Parizkova (1969). The extremely low pulmonary ventilations and $\dot{V}O_2$ max values reported by other investigators may be due to inappropriate testing procedures. Other studies have found a lower $\dot{V}O_2$ max during bicycle compared with treadmill exercise (Glassford et al., 1965; Hermansen and Saltin, 1969; McNab, Conger and Taylor 1969; Hermansen, Ekblom and Saltin, 1970). These workers have suggested that local muscular fatigue

experienced during maximal bicycling limits work performance before the cardiorespiratory system becomes maximally engaged. Hermansen, Ekblom and Saltin (1970) found that mean arterial blood pressure was 10% higher during maximal bicycling compared to maximal uphill running. Since the lower $\dot{V}O_2$ max during bicycling was due to a lower cardiac output, they hypothesized that greater peripheral resistance during bicycle exercise may have placed a limitation upon $\dot{Q}$ max. This is substantiated by the finding by Hoes et al. (1968) that extremely high intramural pressures may occur in the muscle groups involved during maximal bicycle exercise, especially at low pedalling rates.

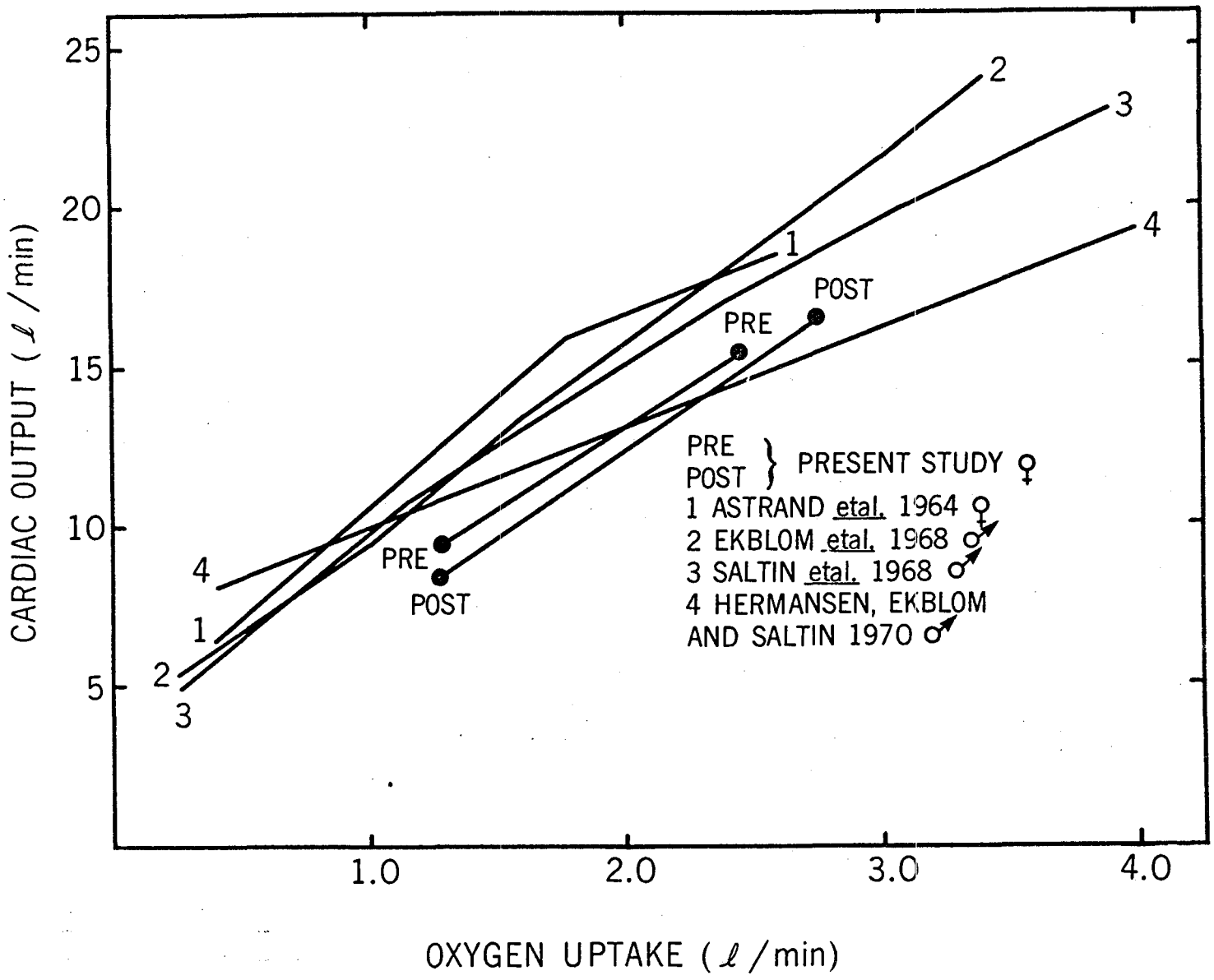
In accord with these findings, Banister and Jackson (1967) found that for given work outputs, the mechanical efficiency of the work decreased as pedalling rates increased especially in excess of 80 revolutions per minute (rpm). Therefore, higher values of $\dot{V}O_2$ max and $\dot{V}E$ max may be obtained by rapid pedalling rates. The studies reporting low values for $\dot{V}O_2$max and $\dot{V}E$ max used the standard method of controlling the subject's pedalling rate at 50 rpm, gradually increasing the resistance to pedalling until the subject could no longer maintain the rate. Maximal bicycle exercise at low pedalling rates therefore may limit work performance in female subjects for reasons other than cardiorespiratory factors, such as 1) impedance of local blood flow due to extremely high

intramural pressures, and 2) limitations of muscle mass and strength in females. The higher values of $\dot{V}O_2$ max and $\dot{V}E$ max found in the present study may, in part, be a result of eliciting a "true" maximal cardiorespiratory stress from pedalling rates ranging from 80 to 90 rpm.

During submaximal work, the oxygen uptake of 5 of the 6 subjects was slightly above the normal range (Astrand, 1960; Hermansen and Anderson, 1965). This lower mechanical efficiency may have been due to a lack of familiarity with the bicycle ergometer and the apprehension associated with initial testing. In addition, pulmonary ventilation for 5 of the 6 subjects was also slightly higher than the normal range.

Cardiac output increased with increasing oxygen uptake as has been observed previously (Figure 17). However, during both submaximal and maximal work, the arterio-venous oxygen difference was greater in the present study than others have found for female subjects (Astrand et al., 1964). There are several factors which may explain this discrepancy. The female subjects of Astrand et al. (1964) had lower hemoglobin concentrations at all levels of exercise than the subjects of this study, the former being 13.6 g/100 ml and the latter 14.6 g/100 ml at maximal exercise. Assuming an identical percentage saturation of arterial blood in the two groups and that 1 grm of hemoglobin can combine with 1.34 ml of oxygen, the subjects of the present study would have an

Figure 17 The relationship of oxygen uptake and cardiac output in their response to exercise of increasing severity. Group mean results of the present study, pre and post-training, are compared with results from similar studies on males and females.


CARDIAC OUTPUT (ℓ /min)
25
20
15
10
5
1.0
2.0
3.0
4.0
OXYGEN UPTAKE (ℓ /min)
1
2
3
4
PRE
POST
PRE
POST
PRE
POST
PRESENT STUDY ♀
1 ASTRAND etal. 1964 ♀
2 EKBLOM etal. 1968 ♂
3 SALTIN etal. 1968 ♂
4 HERMANSEN, EKBLOM
AND SALTIN 1970 ♂

oxygen content of arterial blood 1.34 ml/100 ml higher than the other group. Further, assuming an identical oxygen content in mixed venous blood in maximal exertion in the two groups, the arterio-venous oxygen difference would be 1.34 ml/100 higher in the females of this study. Whereas Astrand et al. (1964) found an arterio-venous oxygen difference of 14.3 ml/100 ml, the present study had a mean of 16.5 ml/100 ml, a difference not totally accounted for by discrepancies in hemoglobin concentration. However, Hartley and Saltin (1969) found that maximal exercise which exhausted subjects in 5 to 8 minutes produced higher cardiac outputs than the "supermaximal efforts" as practiced in this study, even though identical oxygen uptakes were achieved. The "supermaximal effort" would, therefore, elicit a higher arterio-venous oxygen difference than the longer duration exercise-test used by Astrand et al. (1964).

The calculated oxygen content of mixed venous blood in this study was 3.6 ml/100 ml. Values as low as this have been reported on non-athletes by Chapman, Fisher and Sproule (1960) on 26 normal males and Hermansen, Ekblom and Saltin (1970) on 4 normal males. However, usually only trained subjects and athletes are capable of reducing the oxygen content of mixed venous blood to this level (Ekblom, 1969). Therefore, the CO_2 rebreathing procedure may have slightly underestimated cardiac output at both submaximal and maximal work. This would result from overestimation of

$P\bar{v}_{CO_2}$, underestimation of Pa_{CO_2}, or both. Since Pa_{CO_2} was corrected upward of adding 3 mm Hg according to the recommendations of Magel and Andersen (1967), overestimation of $P\bar{v}_{CO_2}$ may have occurred. However, since the reliability of duplicate measures was good, the slight constant underestimation of cardiac output should not have invalidated any serial changes observed due to training.

Heart rates at maximal exercise were very similar to values found in other studies of female subjects (Astrand, 1960; Astrand et al., 1964). At submaximal loads, all 6 subjects had heart rates higher than the normal range when related to the work rate undertaken (Astrand, 1960). However, when oxygen uptake is related to heart rate, the values are within the normal range (Hermansen and Andersen, 1965).

Stroke volume for submaximal work (50% of $\dot{V}O_2$ max) was 78.4% of stroke volume at $\dot{V}O_2$ max. Studies on untrained males have shown that over 90% of maximal stroke volume is attained when oxygen uptake reaches 40% of its maximum (Musshoff and Reindell, 1956; Bevegard, Holmgren and Jonsson, 1960; Wang, Marshall and Shepherd, 1960; Astrand et al., 1964). However, other studies have demonstrated responses similar to those found in this study, on untrained males (Chapman, Fisher and Sproule, 1960; Hermansen, Ekblom and Saltin, 1970) and trained athletes (Ekblom and Hermansen, 1968). Stroke volume during maximal work (81 ml) was of the same magnitude reported for females by Musshoff and Reindell

(1956) but smaller than reported for semi-active females (100 ml) by Astrand et al. (1964). Only subject C.S. ($\dot{Q}$ max 19.1 l/min, stroke volume 105 ml) attained values as high as those reported by Astrand et al. (1964).

Hemoblobin concentration and hematocrit at rest were almost identical to other series on females as reviewed by Astrand (1956). Post-exercise values exhibited the well-known hemoconcentration effect of exercise, reflected by increase in both hemoglobin concentration and hematocrit (Figure 8). A hemoconcentration of between 10 and 15% is regarded as normal from rest to maximal exercise (Ekblom and Hermansen, 1968) whereas the present study exhibited a modest 5.8% increase. Delanne, Barnes and Brouha (1959) postulated a rise in muscular osmotic pressure resulting from exercise from an observed rise in plasma osmolality. This was due to increases in Na^{+}, K^{+}, Ca^{++}, phosphate, lactate and proteinate. These findings support the view that liberation of metabolites during muscular activity results in an increase in the number of osmotically active molecules and hence osmotic pressure in the muscle cells. As a result, fluid and ions are transferred from the plasma to the cells in an attempt to maintain an equilibrium in osmotic pressure.

The above-mentioned hypothesis was substantiated by Novak and Johnson (1970) who found an increase in total osmotic pressure of plasma in rabbits after acute exercise.

They suggested that non-volatile lactic acid accumulating within the cell may be the substance involved in causing the increase in osmotic pressure. Hemoconcentration is probably not caused by increased protein content of the blood during exercise, as Rottini et al. (1970) found no significant changes in blood protein values during exhaustive exercise. Environmental conditions or water loss do not affect the shifts of fluid from the plasma to the cells (Delanne, Barnes and Brouha, 1960).

Arterialized blood pH values at rest agreed closely with other investigators using the same method (Bouhuys et al., 1966). Post-exercise pH was similar in the present study (7.197 $\pm$ .023) to values reported by Bouhuys et al. (1966) on 50 males (7.190 $\pm$ .040).

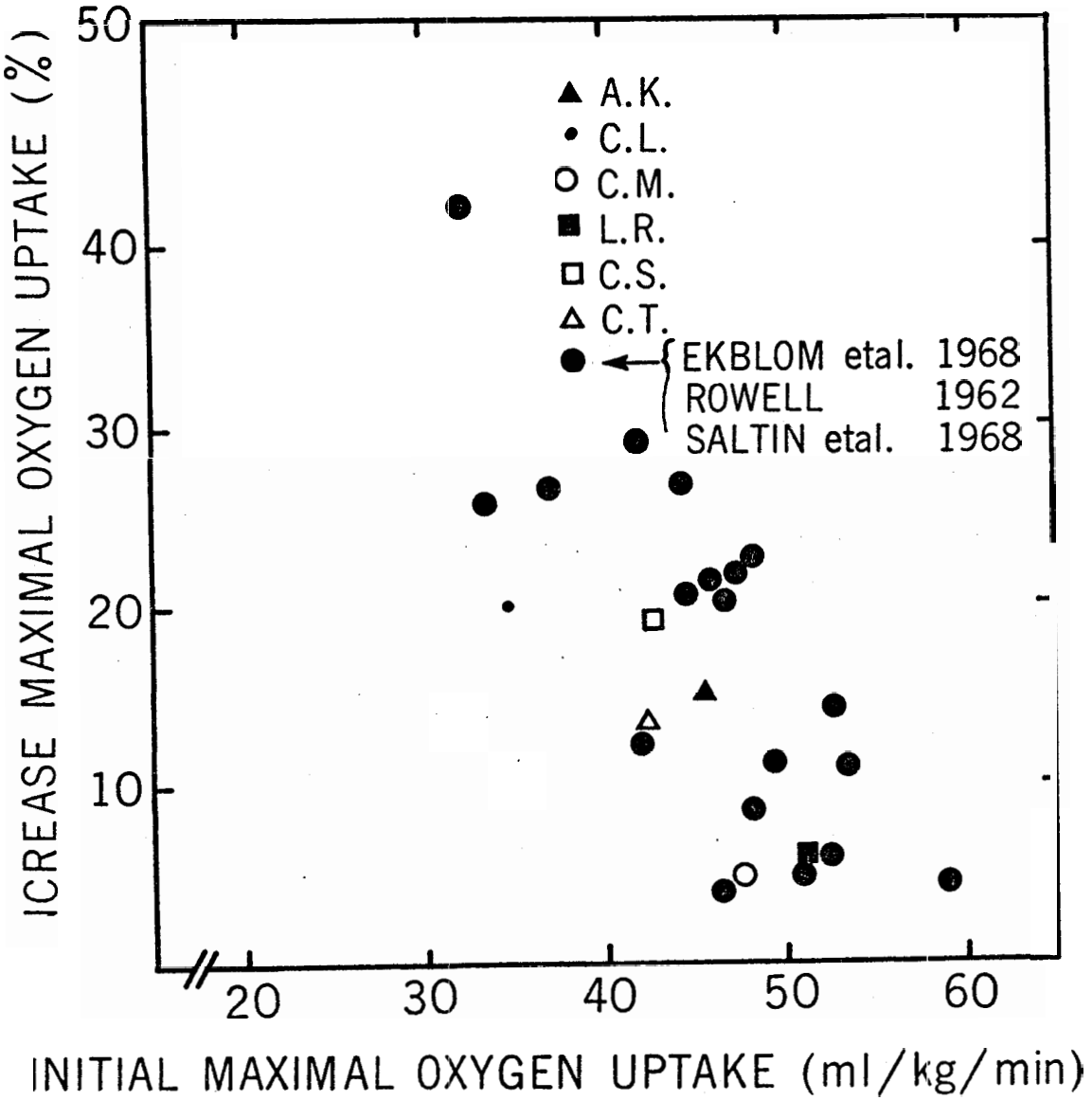
Post-exercise lactate values were modest for the subjects of the present study in comparison to those reported in the literature for maximal exercise (Knehr, Dill and Neufeld, 1942; Saltin et al., 1969). This indicates a limitation of tolerance for anaerobic metabolism during exhaustive exercise, or a constant timing error in blood sampling. Pulmonary function variables, VC and $FEV_{1.0}$, were similar in value to those reported by Astrand (1960) for young women. Minute ventilation at rest, tidal volume and respiratory rate were also within the normal range (Comroe, 1965).

Cardiorespiratory Parameters - Effects of Training Maximal Exercise

Training produced significant ($p<.05$) gains in maximal oxygen uptake ranging from 2.44 l/min to 2.73 l/min or, 12.8% of the original value. These gains are slightly lower than those reported by Rowell (1962) and Ekblom et al. (1968), but higher than Knehr, Dill and Neufeld (1942) and Ekblom (1970). The degree of improvement in $\dot{V}O_2$ max has been shown to be related to initial fitness status. As illustrated in Figure 18, the female subjects of the present study made moderate gains near the lower limit of 'normal' or 'to be expected' improvement in aerobic power due to short-term training. However, it is obvious from the time course of $\dot{V}O_2$ max with weeks of training (Figure 1) that rapid improvements were being made at the time training was terminated, since no "plateau" effect had yet occurred as was noted in other studies. Extension of the training program would probably have resulted in further significant gains in $\dot{V}O_2$ max. Further, illness and bed rest for subject L.R. and a bad cold during the intensive training stage for subject C.M. limited their training gains.

The present study confirms the findings of Rowell (1962), Ekblom et al., (1968) and Saltin et al. (1968) that increases in $\dot{V}O_2$ max were caused by an increased $\dot{Q}$ max and a widened A-V O_2 difference. The significant ($p<.05$) increase in $\dot{Q}$ max (6.9%) was due entirely to a greater stroke volume

Figure 18 The per cent increase in maximal oxygen uptake after short-term physical training in relation to the pre-training maximal oxygen uptake. The subjects of the present study are compared to those of previous studies.


ICREASE MAXIMAL OXYGEN UPTAKE (%)
50
40
30
20
10
A.K.
C.L.
C.M.
L.R.
C.S.
C.T.
EKBLOM etal. 1968
ROWELL 1962
SALTIN etal. 1968
20
30
40
50
60
INITIAL MAXIMAL OXYGEN UPTAKE (ml/kg/min)

(8.4%) in maximal exercise. The slight increase in the A-V O_2 difference during maximal exercise in the initial stages of training (days 0 to 30) did not serve to augment $\dot{V}O_2$ max, as $\dot{Q}$ max decreased slightly. Aerobic power began to increase only when stroke volume, and consequently cardiac output began to rise in maximal exercise. No appreciable increase in ventilation during maximal exercise occurred until $\dot{V}O_2$ max began to increase. The increases in aerobic power were closely paralleled by rising post-exercise lactates and declining post-exercise pH values. Thus it seems apparent that in addition to enhanced work capacity due to increased aerobic power, tolerance for increased acidemia was also developed.

From a comparison of the training program (Table II) and these results, it is apparent that interval training during the initial half of the program was not sufficient to prompt training gains. An increase in aerobic power and the capacity for anaerobic metabolism occurred only after continuous, cross-country running began (day 38).

The mechanisms producing increased maximal stroke volume are not clear. Heart volume has been shown to be highly related to maximal stroke volume (Musshoff, Reindell and Klepzig, 1959; Astrand et al., 1964). Most of the literature indicates that no increase in heart volume occurs in mature subjects from short-term training (Ekblom, 1969; Hartley et al., 1969; Ekblom, 1970). Therefore it is unlikely that the increased maximal stroke volume found in the present study is due to an enlarged heart volume.

Functionally, stroke volume may be enhanced by increased ventricular filling (Frank-Starling mechanism) or an increase in myocardial contractility. Venous return may be aided by an increased blood volume, a factor known to be associated with training (Holmgren et al., 1960; Oscai, Williams and Hertig, 1968). However, Robinson et al. (1966) recently demonstrated that an acute increase in blood volume and central venous pressure does not increase cardiac output in maximal exercise. Thus myocardial contractility may be the factor limiting stroke volume. Ekblom (1969) has suggested that an increased stroke volume without an observed change in heart volume may indicate that the heart functions with less systolic residual blood volume due to an increased myocardial contractility. The data of Saltin et al. (1968) seems to support this conclusion in part. Bed rest resulted in decreased stroke volume during upright as well as supine exercise. In the latter case impairment of venous return should not be a factor. It was hypothesized that the decrement in stroke volume and hence maximal oxygen uptake was due to "an unidentified myocardial effect." An enhancement of this same mechanism with training would seem logical. As Bevegard and Shephard (1967) state,

> Properly, recent studies have been directed to an analysis of the events at a cellular level, including the relation of initial fiber length to development of tension, the relation of contractile elements to elastic components, excitation-contraction coupling, and protein contraction and metabolic pathways for the

> production of utilizable energy. When the several components of the overall contractile process are considered, it is evident that there must be many different mechanisms by which the development of force by myocardial contraction can be altered.

It is interesting to note that subject L.R. exhibited a decrease in maximal oxygen uptake after a 10 day period of bed rest due to illness (day 68 to 78). Her lower aerobic capacity was due entirely to a reduced maximal cardiac output resulting from a lowered stroke volume, a finding co-incident with that of Saltin et al. (1968) in similar circumstances.

In the latter stages of the training, aerobic power was further augmented by a rise in A-V O_2 difference (Figure 7). More interval, anaerobic-type training was being followed in this phase (Table 2). Increased A-V O_2 difference has been thought to be a result of better distribution of arterial blood flow to working muscles (Saltin et al., 1968; Ekblom, 1969), and increased oxygen extraction at the tissues. The latter is supported by findings of enhanced capacity of oxidative metabolism in skeletal muscle of rats due to training (Holloszy, 1967; Kraus et al., 1969; Barnard, Edgerton and Peters, 1970). The proliferation of mitochondria in skeletal muscle due to training (Gollnick and King, 1969) probably occurs in previously 'white' or anaerobic fibers, changing them to aerobic or 'red' fibers (Van Linge 1962; Barnard, Edgerton

and Peter, 1970). It has been demonstrated that females have a low activity level during their adolescent years (Cumming and Cumming, 1963; Astrand, 1968). Since "the composition (e.g. 'red' vs. 'white' fibers) seems to depend on the average stimulus to which its motor-pool is subjected" (Clausen, 1969), girls may possess a considerable potential for cellular change resulting from the stimulus of training.

However, the available data seems to indicate that a training stress must indeed be considerable in order to induce changes at the cellular level. In the present study only very slight increases in A-V O_2 difference occurred until the latter and most intensive portion of the training program (Table 2). Interval training, demanding a high degree of anaerobic metabolism from the subjects, was extensively practiced during this period. It is possible that this type of training is necessary to induce cellular changes. However, these adaptations may not be immediate regardless of the intensity of the training stress and may occur only after gains in aerobic power have already been made. Hartley et al. (1969) found no increase A-V O_2 difference in middle-aged men who increased their maximal oxygen uptake by 14%, which suggests no cellular adaptations. In addition, Barnard, Edgerton and Peters (1970) found no cellular changes in skeletal muscle of guinea pigs after 9 weeks of intensive training, even though their mean running time to

exhaustion was more than six times longer than the mean of the controls. A further 9 weeks of training, however, produced cellular adaptations.

The oxygen carrying capacity of the blood in the present study was not altered with training, a finding supported by many others (Ekblom et al., 1968; Saltin et al., 1968; Ekblom, 1969). The decrease in hemoglobin concentration and hematocrit at rest in the first two weeks of training (Figure 8) was the only exception, the change for hemoglobin being significant ($p<.05$). Similar observations have been made by others, who have hypothesized an increased fragility of the erythrocyte due to hypoxia and increases in plasma free fatty acids from secretions of adrenaline during exercise (Yoshimura, 1965). Hemoglobin in red blood cells is postulated to be utilized to produce muscle protein and young red cells. Yoshimura (1965) indicated that,

> Since this regeneration (of red blood cells) is accelerated during exercise, acceleration of the red blood cell destruction in muscular exercise may be regarded as an adaptive reaction to promote the growth or hypertrophy of muscles and the regeneration of the young red cells.

Despite the lowered hemoglobin concentration at rest on day 14 (Figure 8), it is interesting to note that a greater hemoconcentration occurred during exercise to maintain the same oxygen carrying capacity of the arterial blood. The mechanism of this apparent adaptive hemoconcen-

tration is not clear. However, the higher lactate level noted on day 14 (Figure 3), possibly due to the lowered oxygen content of the blood, may have caused a greater than normal increase in the total osmotic pressure of the blood plasma. This would result in a proportionately greater fluid shift to the cells.

The apparent increase in A-V O_2 difference during maximal exercise occurred in the latter stages of the training program without any changes in the oxygen carrying capacity of the blood. This suggests that even though the oxygen carrying capacity of blood of females is lower than that of males, females are able to reduce the oxygen tension in mixed venous blood in a similar manner as do males and also achieve a greater A-V O_2 difference.

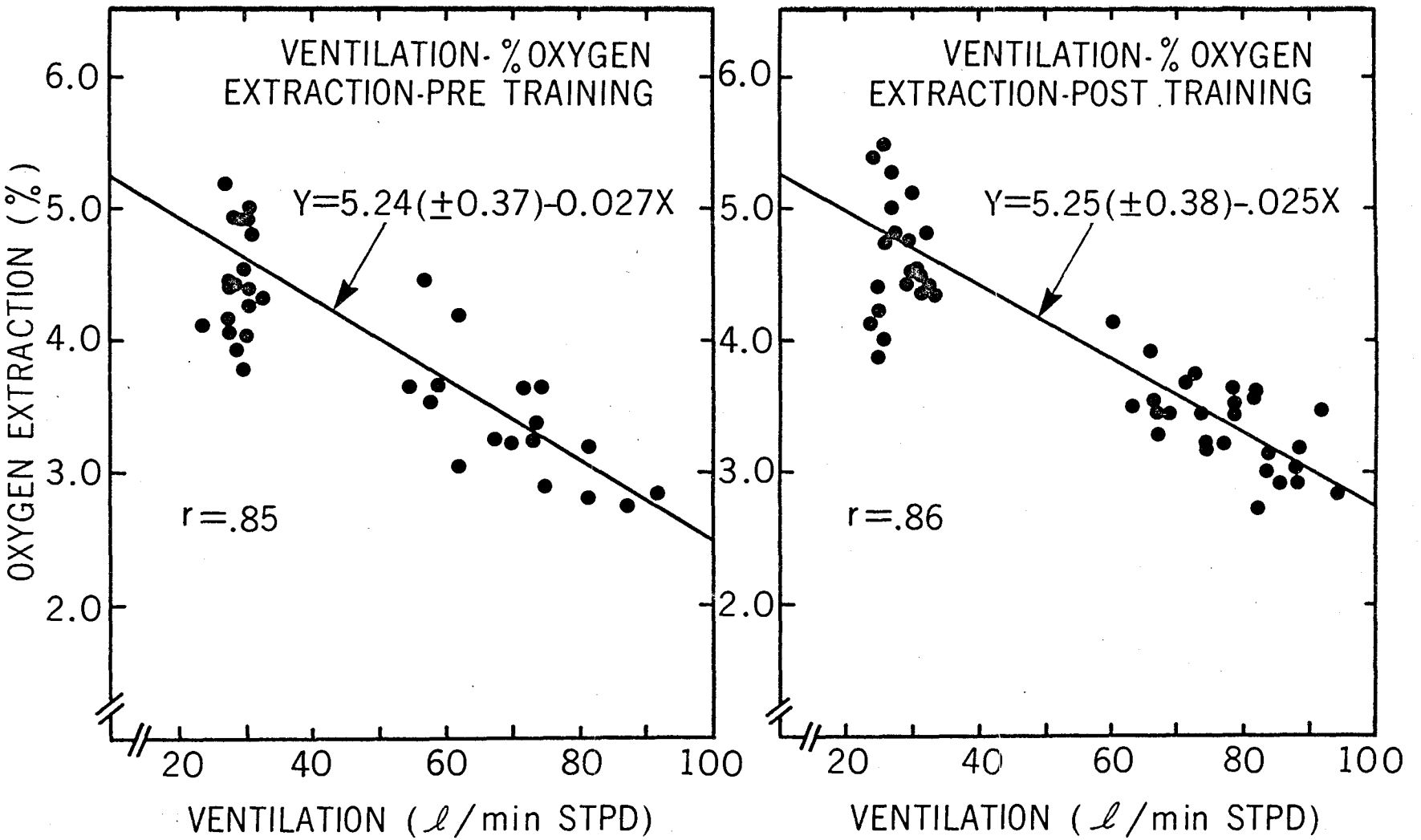
There was a small but significant decrease in maximal heart rate resulting from training. Rowell (1962) and Ekblom et al. (1968) found similar results. However, Astrand (1956) found no difference in maximal heart rates of trained and untrained people and Saltin et al. (1968) did not observe any decrease in maximal heart rate of the 5 subjects of their study. It remains to be determined whether such a decrease is due to 1) lower myocardial catecholamine concentration from reduced sympathetic drive during maximal exercise (Lester et al., 1968) or 2) an increased heart volume from training (Ekblom, 1969).

The finding of increased lactate levels after maximal exercise with training has been observed by many workers (Knehr, Dill and Neufeld, 1942; Williams et al.,

1967; Saltin et al., 1969). Maximal blood lactate is assumed to be a rough index of physical fitness as the ability to attain high values declines with age (Robinson, 1938) and athletes are able to attain much higher values than non-athletes (Robinson and Harmon, 1941). This may be due to biochemical adaptations allowing for greater lactate accumulation from anaerobic metabolism or result simply from a greater ability to tolerate pain. Comparison of the post-exercise lactate and pH curves with those for $\dot{V}O_2$ max and A-V O_2 difference suggests that the increased capacity for anaerobic metabolism is related to gains in aerobic power more than adaptations at the cellular level. It seems, however, that the increased work capacity during maximal exercise achieved by training resulted both from enhanced levels of aerobic and anaerobic power.

To understand the process of increased aerobic power resulting from training it is interesting to examine the relationship of pulmonary ventilation to the percentage of oxygen extracted from it. As illustrated in Figure 19, for equivalent ventilations before and after training the percentage of oxygen extracted from air in the lungs is greater after training. Differences in extraction between the trained and untrained state, although small at low ventilations, becomes progressively greater as pulmonary ventilation increases to maximum.

Figure 19 Regression of % oxygen extraction on ventilation (STPD) pre and post-training


VENTILATION-% OXYGEN EXTRACTION-PRE TRAINING
Y=5.24(±0.37)-0.027X
r=.85
VENTILATION-% OXYGEN EXTRACTION-POST TRAINING
Y=5.25(±0.38)-.025X
r=.86
OXYGEN EXTRACTION (%)
6.0
5.0
4.0
3.0
2.0
20
40
60
80
100
VENTILATION (ℓ/min STPD)

Similar findings have been reported by Banister (1969) from hypoxic training in one athlete. The physiological partition coefficient of oxygen between air and blood is about 1.3 in the lungs (Hatch and Cook, 1955). Therefore, optimum oxygen transfer might be anticipated when alveolar ventilation ($\dot{V}A$) is equal to 1.3 $\dot{Q}$. At rest $\dot{V}A = 0.85\ \dot{Q}$ and the efficiency of lung perfusion is not optimal. In severe exercise $\dot{V}A$ may rise to at least 80 l/min while $\dot{Q}$ rarely exceeds 30 l/min (in males) so that $\dot{V}A \geq 2\dot{Q}$. Therefore, Shephard (1967) concludes,

> A priori, 'alveolar' dead space should thus consist primarily of a perfusion component at rest and primarily of a ventilatory component during severe exercise, with maximum efficiency of oxygen transfer at intermediate respiratory minute volumes.

Improvement in the mean $\dot{V}A/\dot{Q}$ ratio towards 1.3 during severe exercise would account for the improved O_2 extraction for equivalent pulmonary ventialtions found in this study. Pulmonary ventilation is known to decline for standard work loads with training, especially at work loads approaching maximum (Andrew Guzman and Becklake, 1966; Douglas and Becklake, 1968; Ekblom et al., 1968). With a reduction in ventilation during comparatively hard muscular exercise, the mean $\dot{V}A/\dot{Q}$ ratio would decline to a more efficient value, thereby improving the efficiency of oxygen transfer across the pulmonary membrane. The higher maximal cardiac output

after training allows for a higher useful upper limit of ventilation in maximal exercise, before severe hyperventilation occurs. Saltin et al. (1968) have demonstrated a more uniform distribution of blood flow with respect to ventilation resulting from training together with a smaller alveolar-arterial O_2 pressure gradient.

Submaximal Exercise

The slightly increased mechanical efficiency (Figure 9) noted during training in this study has been observed also by others (Robinson and Harmon, 1941; Knehr, Dill and Neufeld, 1942; Ekblom, 1969). The significant ($p<.05$) progressive decline in heart rate for standard submaximal work is a well known adaptation to training (Astrand, 1956). Although it was not significant, Figure 14 and Figure 13 illustrate that stroke volume increased co-incidently with the decline in heart rate, as found in other investigations (Rowell, 1962; Ekblom et al., 1968; Saltin et al., 1968; Hartley et al., 1969). However, unlike the latter studies, the increased mean submaximal exercise-stroke volume in the present study was not associated with an increased mean maximal stroke volume and aerobic power. Submaximal exercise-stroke volume, which was 78% of maximal exercise-stroke volume before training began, increased until it was 87% of maximal after 30 days of training. During this period, maximal oxygen uptake and maximal stroke volume did not

change to any degree. As discussed earlier, maximal stroke volume seems to depend mostly on the contractile power of the heart itself. If maximal exercise-stroke volume did not increase in the first half of the training program, it suggests that myocardial contractility was not enhanced also. Therefore, the mechanism producing a decline in heart rate co-incidently with an increased stroke volume for submaximal exercise would seem to be something other than an increased contractile power of the myocardium.

In response to exercise, cardiac output is elevated by an increase in both heart rate and stroke volume. The latter may occur through an increased Frank-Starling mechanism and an increased myocardial contractility. In the normal condition, however, the Frank-Starling mechanism is obscured by the influence of the autonomic nervous system (Starling, 1920). It has been found that varying the ventricular rate by artificial pacemakers results in stroke volume adjustments to maintain the same cardiac output (Ross, Linhart and Braunwald, 1965). During exercise, if the heart rate at a given work load is increased through artificial pacemakers (Bevegard, 1962) or atrial flutter (Astrand et al., 1963), stroke volume is correspondingly decreased so that cardiac output remains unchanged. These studies seem to indicate that changes in stroke volume are secondary to changes in heart rate.

The high heart rates for a given oxygen uptake characteristic of the untrained individual may be due, in part, to greater sympathetic activity (Holmgren, 1967). In this case, stroke volume may be prevented from attaining maximal levels during moderate exercise by limitation of the Frank-Starling mechanism. However, during severe exercise, even the tachycardia and sympathetic stimulation of the myocardium that take place are not sufficient to prevent an increase in ventricular end-diastolic size and maximal stroke volume is achieved (Braunwald, Ross and Sonnenblick, 1967). The above workers state that

> the normal cardiac response to exercise involves the integrated effects on the myocardium of simple tachycardia, sympathetic stimulation and the operation of the Frank-Starling mechanism. During submaximal levels of exertion, cardiac output can rise even when one or two of these influences are blocked. However, during maximal levels of muscular exercise, the ventricular myocardium requires all three influences to sustain a level of activity sufficient to satisfy the greatly augmented oxygen requirements of the exercising skeletal muscles.

In the present study, the decline in heart rate during submaximal exercise in the initial 40 days of training probably results from 1) the slight increased mechanical efficiency, and 2) a reduced sympathetic and/or increased parasympathetic effect on the myocardium. Robinson et al. (1966) and Holmgren (1967) have hypothesized that increased vagal tone may occur from training to produce the relative bradycardia noted in the trained state. A lowering of the

heart rate in this way may allow for more effective ventricular filling and an increased stroke volume from an increased Frank-Starling mechanism.

The data suggests that levels of physical training inadequate to increase maximal aerobic power nevertheless may produce beneficial adaptations of the circulatory system during submaximal exercise due to some unexplained training effect on the autonomic nervous system.

Although the changes in cardiac output and A-V O_2 difference during submaximal work were not statistically significant, it is interesting to compare the time course throughout the period of training of these measures (Figures 12 and 15) to the hypothesis of Clausen (1969), that training results in blood flow increases to inactive parts and decreases to the active tissues. Until the latter portion of the training program, the slight decrease in cardiac output generally parallels the decline in oxygen uptake. At this point, cardiac output declines markedly due to a rise in A-V O_2 difference. Many workers have suggested that a more effective redistribution of arterial blood to the working muscules occurs with training due to a greater sympathetic vasoconstriction in non-exercising parts (Varnauskas *et al.*, 1966; Andrew Guzman and Becklake, 1966; Klassen, Andrew and Backlake, 1970). In the present study the increased A-V O_2 difference in submaximal exercise closely paralleled the rise in A-V O_2 difference during maximal exercise, in-

dicating that identical mechanisms induce the changes. It seems unlikely that sympathetic activity would increase even transiently during submaximal and maximal exercise as a result of training. Sympathetic activity is generally felt to decrease for a given work load with training. More likely, the increased A-V O_2 difference in submaximal exercise is due to an increased ability for oxidative metabolism in the muscle cells, as previously discussed for maximal work.

Clausen (1969) hypothesized a reduction of blood flow to the working muscles with a slight increase in blood flow to non-active parts to explain the decrease in cardiac output. The reduction of blood flow to active muscles is dependent on the previously discussed cellular changes occurring from training. Exercise hyperemia in muscle is thought to be caused by local release of metabolically linked vasodilator compounds (Haddy and Scott, 1968). The improved capacity for oxidative metabolism may keep these metabolites at lower concentrations and thus limit the blood flow increase (Clausen, 1969). Similar results have been found by Varnauskas et al. (1970).

The findings of the present study may be explained by the hypothesis of Clausen (1969). During the period when no increase in aerobic power was made, submaximal cardiac output for a given oxygen uptake remained relatively constant. When aerobic power began to rise, without an increased A-V O_2 difference (an indicator of cellular

adaptation), submaximal cardiac output exhibited no change. During this phase, blood flow to non-active parts may have increased but since no cellular changes were suggested, muscle blood flow would remain constant. The final period of training exhibited a further increase in aerobic power and an increased A-V O_2 difference, suggesting cellular adaptations. During this period, the submaximal cardiac output declined markedly, probably due to reduced blood flow to the active muscles from a lesser degree of exercise hyperemia.

That the degree of metabolic acidosis decreased during submaximal exercise is indicated by the steady decrease in respiratory exchange ratio resulting from a significant ($p<.05$) decline in carbon dioxide elimination in excess of that expected from the slight drop in oxygen uptake (Figures 9 and 10). It has been shown that the addition of lactate and pyruvate to the blood during exercise causes excretion via the lungs of more CO_2 than is formed metabolically ("excess CO_2"), and the respiratory exchange ratio R increases (Issekutz and Rodahl, 1961; Naimark, Wasserman and McIlroy, 1964). The decrease of R shown at the submaximal level is once again an indication of an enhanced capacity of cellular enzyme systems. Increased capacity for oxidative metabolism would result in a slowing of glycolysis and an inhibition of lactate production (Hollozy, 1967; Gollnick and King, 1969).

The small non-significant decrease in pulmonary ventilation may be mediated through the effect of declining

acidemia during submaximal work on the chemoreceptors of the carotid sinus and aortic arch. Lower CO_2 content in the blood resulting from this reduced level of acidemia may also lessen the central ventilatory drive. However, since oxygen extraction in the lungs is at maximum efficiency during intermediate work-loads (Shephard, 1967), submaximal exercise pulmonary ventilation would not be expected to decrease very much with training (Figure 11). The slight decline may also reflect the slight increased mechanical efficiency experienced and serve to maintain the already efficient $\dot{V}A/\dot{Q}$ ratio.

CHAPTER VI

CONCLUSIONS

Six women of college age participated in a program of physical training consisting of 3 sessions per week for 12 weeks. Repeated tests were taken bi-weekly in maximal and submaximal exercise conditions, for parameters such as oxygen uptake, carbon dioxide production, cardiac output, heart rate, hemoglobin concentration, hematocrit, lactate and pH. Pulmonary function was measured at rest. Derived parameters such as stroke volume, arterio-venous oxygen difference and respiratory exchange ratio were calculated. The relatively small group investigated does not allow generalization of the results to larger populations of women or men. However, on the basis of the results of this study, some definite conclusions seem warranted.

The six young women studied exhibited increases in aerobic and anaerobic power to a similar degree as young men respond to physical training. The mechanisms operating to achieve the gains in aerobic power found for these females were also similar to those found for male subjects. Increased maximal oxygen uptake (12.8%) was mainly achieved by a significantly increased cardiac output ($p<.05$) during maximal exercise, seemingly entirely due to an increased

stroke volume, and a slightly increased arterio-venous oxygen difference.

The training program was carefully graduated and controlled to prevent pain and soreness in the musculo-skeletal system. This type of training failed to produce increments in aerobic power and associated parameters in its initial stages. In the six young women studied, continuous cross-country running at a minimum of 80% of $\dot{V}O_2$ max for 18 minutes appeared necessary to prompt increases in maximal oxygen uptake, which occurred through an increased cardiac output only. The small increase in arterio-venous oxygen difference did not occur until the latter portion of the training program and coincided with the more intensive anaerobic-type interval training. It is interesting to speculate that adaptations which cause the increased arterio-venous oxygen difference may not occur at the same rate as the increased functional capacity of the heart (increased cardiac output) during training. Alternatively, it may be that anaerobic-type interval training is necessary to prompt increases in arterio-venous oxygen difference.

The increase of post-exercise lactate and the decline of post-exercise pH with training paralleled the trend for increased maximal oxygen uptake. This suggests that tolerance for anaerobic metabolism is related to gross fitness increments and this is consistent with the claim of many authors that lactate levels resulting from maximal

exercise is an indicator of physical fitness. Physical work capacity increased with training and was associated with increases in aerobic and anaerobic power.

Training resulted in a more efficient oxygen extraction in the lungs, especially during heavy exercise. This may be due to a lowered and more effective mean ventilation-perfusion ratio in the lungs.

Training resulted in a significant decrease ($p<.05$) in carbon dioxide production and heart rate response to standard submaximal exercise. Associated with the decrease in heart rate was a moderate increase in stroke volume. These adaptations occurred during the initial stages of training when no gains were noted for maximal oxygen uptake and maximal stroke volume. The data suggests that beneficial effects of training on the hemodynamic response to standard submaximal exercise may occur in absence of increases in maximal oxygen uptake and maximal stroke volume. As discussed previously, a training effect upon the autonomic nervous system, reducing sympathetic or increasing the parasympathetic effect on the myocardium may mediate the reduction in heart rate. Further studies are necessary to investigate these responses to training.

There was an indication that cardiac output decreased for standard submaximal exercise in the latter portion of the training program. However, the reduction was not statistically significant. Since oxygen uptake remained constant,

an increment in arterio-venous oxygen difference for sub-maximal exercise accompanied the decrease in cardiac output. The increase in arterio-venous oxygen difference for sub-maximal exercise occurred at the same stage of the training program as an increased A-V O_2 difference in maximal exercise. This may suggest a common mechanism inducing these changes.

The present study represents a successful achievement in training and making substantial changes in the fitness of college women. The girls who participated in this study were tremendously enthusiastic and conscientious in their training and laboratory evaluations. They exhibited no hesitancy in extending themselves consistently to exhaustion during repeated maximal exercise tests.

Further studies of the effects of training on large groups of females are needed to confirm or modify the findings of the present investigation. Quantitative differences between men and women in their response to training may be determined by repeated laboratory evaluations during a training program which imposes the same relative physiological stress on both the male and female groups. In that way, such findings as a lack of increase in maximal oxygen uptake in the first stages of training may be definitely determined to be either a true sex difference or a result of inadequate stress of the training program.

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APPENDIX 1

Individual Raw Data for all Parameters and Tests During the Training Program

Maximal Oxygen Uptake (l/min)									Maximal Oxygen Uptake (ml/kg/min)							
Test	A.K.	C.L.	C.M.	L.R.	C.S.	C.T.	$\bar{X}$	S.E.	A.K.	C.L.	C.M.	L.R.	C.S.	C.T.	$\bar{X}$	S.E.
Initial	2.23	2.18	2.37	2.47	2.71	2.59	2.44	0.09	45.3	34.5	46.5	50.7	42.4	42.3	43.8	2.51
Initial	2.14	†-	2.43	2.49	2.61	†-			43.5	†-	47.6	50.9	40.8	†-		
2	†-	†-	†-	†-	†-	†-	-	-	†-	†-	†-	†-	†-	†-	-	-
3	2.02	2.31	2.52	2.50	2.64	2.79	2.46	0.12	40.4	36.6	48.6	50.9	41.4	46.3	44.0	2.43
4	2.23	2.10	2.19	2.55	2.89	2.79	2.46	0.15	44.6	35.5	42.2	51.7	45.7	46.3	44.0	2.69
5	2.32	2.47	*-	2.65	2.77	2.82	2.61	0.11	45.8	39.7	*-	54.0	44.0	46.3	46.0	2.60
6	2.62	2.13	2.50	*-	3.02	2.88	2.63	0.18	52.1	33.9	48.7	*-	47.4	48.0	46.0	3.51
Final	2.46	2.61	2.51	2.39	2.94	2.80	2.73	0.14	49.4	41.4	48.9	48.7	46.2	46.7	47.8	1.86
Final	2.19	2.48	2.56	2.46	3.16	2.87			44.0	39.3	49.9	50.1	50.5	47.8		

Final tests for subject LR not included in calculations due to adverse effect of bed rest on fitness.

†technical difficulties prevented determination of this parameter.

*illness prevented testing n=5 for tests 5, 6, and Final.

Carbon Dioxide Production at $\dot{V}O_2$ Max (l/min)									Respiratory Exchange Ratio at $\dot{V}O_2$ Max							
Test	A.K.	C.L.	C.M.	L.R.	C.S.	C.T.	$\bar{X}$	S.E.	A.K.	C.L.	C.M.	L.R.	C.S.	C.T.	$\bar{X}$	S.E.
Initial	2.52	2.33	2.60	2.37	2.79	2.90	2.79	0.10	1.13	1.07	1.10	0.96	1.03	1.12	1.14	.03
Initial	2.63	2.62	2.74	2.60	3.20	2.93			1.23	†-	1.13	1.05	1.23	†-		
2	2.52	2.49	2.37	2.43	3.06	2.98	2.64	0.13	†-	†-	†-	†-	†-	†-		
3	2.49	2.33	2.61	2.55	2.98	2.86	2.64	0.11	1.22	1.01	1.04	1.02	1.13	1.03	1.08	.04
4	2.52	2.00	2.35	2.40	2.83	2.63	2.46	0.13	1.13	0.95	1.07	0.94	0.98	0.94	1.00	.03
5	2.74	2.62	*-	2.80	2.80	2.71	2.73	0.04	1.18	1.06	*-	1.06	1.01	0.96	1.05	.04
6	2.78	2.38	2.53	*-	3.02	3.03	2.75	0.14	1.06	1.12	1.01	*-	1.00	1.05	1.05	.02
Final	2.48	2.58	2.46	2.58	2.91	2.80	2.75	0.12	1.01	0.99	0.98	1.08	0.99	1.00	1.04	.03
Final	2.52	2.58	2.66	2.41	3.13	2.68			1.15	1.04	1.04	0.98	0.99	0.94		

Final tests for subject LR not included in calculations due to adverse effect of bed rest on fitness.

†technical difficulties prevented determination of this parameter.

*illness prevented testing n=5 for tests 5, 6, and Final.

	Cardiac Output at $\dot{V}O_2$ Max (l/min)								Heart Rate at $\dot{V}O_2$ Max (beats/min)							
Test	A.K.	C.L.	C.M.	L.R.	C.S.	C.T.	$\bar{X}$	S.E.	A.K.	C.L.	C.M.	L.R.	C.S.	C.T.	$\bar{X}$	S.E.
Initial	14.9	†-	†-	13.3	19.0	15.8	15.3	1.1	193	187	195	204	191	180	193	3.8
Initial	14.8	14.2	13.9	12.8	19.9	15.0			195	188	191	207	191	184		
2	15.2	14.7	13.7	13.3	19.5	16.8	15.5	1.0	195	185	191	200	185	†-	191	3.4
3	14.2	13.6	13.9	12.8	19.6	15.6	15.0	1.1	195	178	191	197	187	180	188	3.1
4	15.0	13.4	12.3	14.0	19.1	14.5	14.7	1.0	187	180	187	195	187	176	185	3.3
5	14.1	14.6	*-	14.1	19.9	15.9	15.7	1.2	195	187	*-	195	184	182	189	3.0
6	14.2	14.6	13.7	*-	20.8	16.9	16.0	1.5	193	180	184	*-	187	180	185	3.3
Final	15.8	13.7	12.7	11.9	17.6	16.3	16.3	0.8	191	184	187	200	182	184	190	3.2
Final	14.1	16.4	14.2	13.0	18.7	15.2			195	180	189	195	187	180		

Final tests for subject LR not included in calculations due to adverse effect of bed rest on fitness.

†technical difficulties prevented determination of this parameter.

*illness prevented testing n=5 for tests 5, 6, and Final.

	Stroke Volume at $\dot{V}O_2$ Max (ml)								A-V O_2 Diff. at $\dot{V}O_2$ Max (vol/%)							
Test	A.K.	C.L.	C.M.	L.R.	C.S.	C.T.	$\bar{X}$	S.E.	A.K.	C.L.	C.M.	L.R.	C.S.	C.T.	$\bar{X}$	S.E.
Initial	77	†-	†-	65	99	88	81	6	15.0	†-	†-	18.6	14.3	16.4	16.5	1.0
Initial	76	76	73	62	105	81			14.5	†-	17.5	19.4	13.1	†-		
2	78	79	72	67	105	92	82	6	†-	†-	†-	†-	†-	†-	-	-
3	73	77	73	64	105	87	80	6	14.3	16.9	18.1	19.6	13.5	17.9	16.7	1.1
4	80	75	66	72	102	82	80	5	14.9	15.6	17.8	18.3	15.1	19.3	16.8	0.8
5	72	78	*-	72	107	88	83	8	16.5	16.9	*-	18.8	14.0	17.7	16.8	0.9
6	74	81	74	*-	111	94	87	8	18.5	14.7	18.3	*-	14.5	17.0	16.6	1.0
Final	74	74	68	60	97	89	88	5	15.6	19.1	19.8	20.1	16.7	17.2	17.7	0.7
Final	83	91	75	67	100	85			15.6	15.2	18.0	19.0	16.9	18.8		

Final tests for subject LR not included in calculations due to adverse effect of bed rest on fitness.

†technical difficulties prevented determination of this parameter.

*illness prevented testing n=5 for tests 5, 6, and Final.

Ventilation at $\dot{V}O_2$ Max (l/min BTPS)									Lactate Post-Exercise (mg%)							
Test	A.K.	C.L.	C.M.	L.R.	C.S.	C.T.	$\bar{X}$	S.E.	A.K.	C.L.	C.M.	L.R.	C.S.	C.T.	$\bar{X}$	S.E.
Initial	87.2	83.4	92.0	107.8	93.0	101.4	100.5	5.3	†-	†-	†-	†-	†-	†-	-	-
Initial	95.1	90.4	93.0	115.3	91.1	116.4			31	16	57	38	57	58	42.8	7.8
2	89.3	91.4	108.3	107.7	106.8	110.6	102.4	4.2	66	14	77	62	50	42	51.8	9.9
3	95.7	94.0	120.2	111.6	105.2	109.7	106.1	4.5	39	33	24	22	78	82	46.3	12.0
4	107.8	79.1	111.8	104.7	99.3	109.7	102.1	5.4	68	19	26	18	39	44	35.7	8.5
5	110.1	103.7	*-	115.3	102.6	103.1	107.0	2.8	91	66	*-	91	78	67	78.6	6.1
6	119.0	97.2	111.9	*-	116.1	117.4	112.3	4.5	88	32	49	*-	61	75	61.0	10.9
Final	97.1	107.2	106.4	115.9	104.7	112.6	109.6	2.5	59	†-	55	44	50	85	81.6	10.3
Final	102.0	107.7	110.5	104.8	115.0	102.1			80	48	102	76	93	70		

Final tests for subject LR not included in calculations due to adverse effect of bed rest on fitness.

†technical difficulties prevented determination of this parameter.

*illness prevented testing n=5 for tests 5, 6, and Final.

Hemoglobin Pre-Exercise (g/100 ml)									Hemoglobin Post-Exercise (g/100 ml)							
Test	A.K.	C.L.	C.M.	L.R.	C.S.	C.T.	$\bar{X}$	S.E.	A.K.	C.L.	C.M.	L.R.	C.S.	C.T.	$\bar{X}$	S.E.
Initial	14.2	14.3	14.7	14.8	13.5	12.8	13.8	0.2	14.5	14.5	†-	14.8	14.5	14.6	14.6	0.2
Initial	†-	13.8	13.2	†-	12.8	14.2			14.3	14.5	†-	16.2	14.0	14.5		
2	12.0	13.0	13.2	13.0	12.0	12.5	12.6	0.2	14.6	14.5	14.5	16.5	14.1	14.5	14.8	0.4
3	13.3	14.2	14.5	13.6	12.2	13.6	13.5	0.4	14.8	14.3	†-	14.7	14.5	15.0	14.7	0.2
4	12.6	13.5	13.2	14.2	12.0	13.5	13.2	0.3	14.5	14.0	14.5	†-	14.0	15.2	14.4	0.3
5	13.5	14.2	*-	14.8	13.5	13.5	13.9	0.3	14.5	†-	*-	16.0	14.4	14.3	14.8	0.4
6	13.1	13.6	13.0	*-	13.3	14.5	13.5	0.3	14.1	14.0	14.9	*-	14.0	14.8	14.4	0.2
Final	13.2	12.5	14.3	14.0	12.7	13.5	13.6	0.3	15.2	†-	14.7	15.0	13.3	14.5	14.8	0.3
Final	14.5	14.0	14.8	14.0	12.5	14.2			15.0	15.8	15.8	†-	13.6	15.2		

Final tests for subject LR not included in calculations due to adverse effect of bed rest on fitness.

†technical difficulties prevented determination of this parameter.

*illness prevented testing n=5 for tests 5, 6, and Final.

Hematocrit Pre-Exercise (%)									Hematocrit Post-Exercise (%)							
Test	A.K.	C.L.	C.M.	L.R.	C.S.	C.T.	$\bar{X}$	S.E.	A.K.	C.L.	C.M.	L.R.	C.S.	C.T.	$\bar{X}$	S.E.
Initial	†-	38.3	†-	†-	44.1	41.3	42.6	1.0	†-	45.9	†-	†-	51.6	50.0	47.1	1.1
Initial	40.9	42.4	42.5	48.3	41.6	44.3			45.1	45.8	42.7	47.1	45.0	50.8		
2	†-	41.7	43.5	41.7	39.0	37.8	40.7	1.2	45.8	45.2	47.5	47.5	45.2	47.5	46.5	0.5
3	41.5	43.5	44.3	41.8	44.8	44.4	43.4	0.6	†-	45.2	†-	50.0	46.8	51.6	48.4	1.7
4	39.4	51.0	42.5	42.6	39.8	44.2	43.3	1.9	42.6	51.3	45.9	45.6	46.9	48.5	46.8	1.3
5	39.7	45.2	*-	44.4	43.1	42.7	43.0	1.1	45.5	†-	*-	49.2	50.0	46.7	47.9	1.2
6	40.0	40.3	41.0	*-	43.4	44.6	41.9	1.0	43.8	40.0	43.1	*-	47.5	50.0	44.9	2.0
Final	†-	41.3	41.5	44.0	41.1	43.3	42.0	0.3	†-	45.3	45.4	45.9	44.4	50.4	47.3	0.8
	41.3	42.7	41.5	44.4	42.2	43.1			51.4	47.4	46.8	43.4	46.9	47.7		

Final tests for subject LR not included in calculations due to adverse effect of bed rest on fitness.

†technical difficulties prevented determination of this parameter.

*illness prevented testing n=5 for tests 5, 6, and Final.

	pH Pre-Exercise								pH Post-Exercise							
Test	A.K.	C.L.	C.M.	L.R.	C.S.	C.T.	$\bar{X}$	S.E.	A.K.	C.L.	C.M.	L.R.	C.S.	C.T.	$\bar{X}$	S.E.
Initial	7.401	7.410	7.400	7.412	7.383	7.404	7.397	0.007	7.240	7.254	7.135	7.262	7.195	7.133	7.197	.023
Initial	7.392	7.410	7.403	7.427	7.373	7.344			7.235	7.241	7.159	7.244	7.192	7.190		
2	7.414	7.415	7.411	7.431	7.393	7.400	7.411	0.006	7.256	7.282	7.171	7.153	7.206	7.250	7.220	.023
3	†-	7.333	7.415	7.413	7.359	7.380	7.380	0.018	7.157	7.257	7.107	7.212	7.112	7.159	7.167	.025
4	7.419	7.418	7.395	7.398	7.422	7.378	7.405	0.008	7.195	7.262	7.211	7.179	7.177	7.145	7.195	.018
5	7.396	7.395	*-	7.401	7.385	7.375	7.390	0.005	7.114	7.215	*-	7.160	7.163	7.143	7.159	.019
6	7.379	7.406	7.371	*-	7.406	7.394	7.391	0.008	7.163	7.269	7.115	*-	7.135	7.156	7.168	.030
Final	7.349	7.390	7.413	7.408	7.366	7.371	7.378	0.007	7.162	†-	7.171	7.151	7.135	7.101	7.150	.025
Final	7.405	7.366	7.365	7.390	7.379	7.373			7.167	7.235	7.135	7.250	7.118	7.175		

Final tests for subject LR not included in calculations due to adverse effect of bed rest on fitness.

†technical difficulties prevented determination of this parameter.

*illness prevented testing n=5 for tests 5, 6, and Final.

$\dot{V}O_2$ for Submaximal Work (l/min)									$\dot{V}CO_2$ for Submaximal Work (l/min)							
Test	A.K.	C.L.	C.M.	L.R.	C.S.	C.T.	$\bar{X}$	S.E.	A.K.	C.L.	C.M.	L.R.	C.S.	C.T.	$\bar{X}$	S.E.
Initial	1.35	1.14	1.48	1.09	1.53	1.23	1.29	.06	1.10	0.83	1.26	0.79	1.25	1.06	1.06	.05
Initial	1.40	†-	1.30	1.17	1.42	†-			1.13	1.01	1.07	0.85	1.22	1.12		
2	†-	†-	†-	†-	†-	†-			1.07	0.94	1.09	0.83	1.14	1.02	1.02	.05
3	1.39	1.16	1.25	1.10	1.24	1.10	1.21	.05	1.15	0.85	1.14	0.87	1.12	0.97	1.02	.06
4	1.26	1.10	1.49	1.16	1.41	1.36	1.30	.07	1.00	0.82	1.11	0.75	0.98	1.02	0.95	.06
5	1.30	0.99	*-	0.90	1.27	1.27	1.15	.09	0.99	0.81	*-	0.73	1.02	1.01	0.91	.07
6	1.30	1.05	1.33	*-	1.50	1.18	1.27	.08	1.09	0.75	1.17	*-	1.16	0.89	1.01	.10
Final	1.54	0.94	1.42	1.21	1.39	1.43	1.30	.06	1.15	0.73	1.16	0.85	1.09	1.09	1.00	.07
Final	1.29	1.04	1.22	1.17	1.32	1.39			0.97	0.77	0.98	0.79	0.98	1.03		

Final tests for subject LR not included in calculations due to adverse effect of bed rest on fitness.

†technical difficulties prevented determination of this parameter.

*illness prevented testing n=5 for tests 5, 6, and Final.

R for Submaximal Work									$\dot{Q}$ for Submaximal Work (l/min)							
Test	A.K.	C.L.	C.M.	L.R.	C.S.	C.T.	$\bar{X}$	S.E.	A.K.	C.L.	C.M.	L.R.	C.S.	C.T.	$\bar{X}$	S.E.
Initial	0.82	0.73	0.86	0.73	0.82	0.86	0.80	.02	11.3	7.2	11.9	8.0	10.3	8.7	9.4	0.7
Initial	0.81	†-	0.82	0.72	0.86	†-			11.4	7.5	9.1	8.3	10.6	8.8		
2	†-	†-	†-	†-	†-	†-			9.1	8.4	9.1	7.4	12.3	8.6	9.2	0.7
3	0.83	0.73	0.91	0.79	0.90	0.88	0.84	.03	9.8	7.6	9.0	8.8	13.0	8.4	9.4	0.8
4	0.79	0.74	0.72	0.65	0.70	0.75	0.73	.02	10.2	7.5	9.0	7.5	10.5	9.0	9.0	0.6
5	0.76	0.82	*-	0.81	0.80	0.80	0.80	.01	10.1	7.7	*-	7.5	11.1	8.7	9.0	0.7
6	0.84	0.71	0.88	*-	0.77	0.76	0.79	.03	10.1	7.1	10.1	*-	12.3	7.4	9.4	1.1
Final	0.75	0.78	0.82	0.70	0.78	0.76	0.75	.02	9.6	7.1	9.0	8.0	10.7	7.7	8.7	0.4
Final	0.75	0.74	0.80	0.67	0.74	0.74			7.7	7.3	8.9	7.1	9.5	7.6		

Final tests for subject LR not included in calculations due to adverse effect of bed rest on fitness.

†technical difficulties prevented determination of this parameter.

*illness prevented testing n=5 for tests 5, 6, and Final.

Heart Rate for Submaximal Work (beats/min)									Stroke Volume for Submaximal Work (ml)							
Test	A.K.	C.L.	C.M.	L.R.	C.S.	C.T.	$\bar{X}$	S.E.	A.K.	C.L.	C.M.	L.R.	C.S.	C.T.	$\bar{X}$	S.E.
Initial	150	141	167	170	149	135	150	4.0	76	51	71	47	69	64	63	3.4
Initial	147	144	164	161	141	127			78	52	55	52	75	69		
2	137	128	150	161	142	120	140	7.4	67	66	61	46	86	71	66	5.9
3	150	123	154	145	132	120	137	6.3	66	62	58	61	99	70	69	6.7
4	134	113	150	155	129	122	134	7.2	76	66	60	48	82	74	68	5.4
5	141	117	*-	143	126	120	129	6.1	72	66	*-	52	88	73	70	6.5
6	153	110	141	*-	141	118	133	9.0	66	65	72	*-	89	63	71	5.0
Final	148	110	158	167	122	118	132	6.1	65	64	57	48	88	65	65	3.4
Final	155	113	143	156	126	127			51	65	62	45	76	60		

Final tests for subject LR not included in calculations due to adverse effect of bed rest on fitness.

†technical difficulties prevented determination of this parameter.

*illness prevented testing n=5 for tests 5, 6, and Final.

A-V O_2 Difference-Submaximal Work (vol%)									Ventilation-Submaximal Work (l/min BTPS)							
Test	A.K.	C.L.	C.M.	L.R.	C.S.	C.T.	$\bar{X}$	S.E.	A.K.	C.L.	C.M.	L.R.	C.S.	C.T.	$\bar{X}$	S.E.
Initial	11.9	15.8	12.4	13.7	14.8	14.1	13.7	0.4	37.4	33.9	38.8	35.6	38.2	34.7	37.2	0.6
Initial	12.3	†-	14.3	14.0	13.4	†-			41.0	37.3	38.6	37.6	36.7	36.0		
2	†-	†-	†-	†-	†-	†-	-	-	40.8	38.9	39.4	33.5	37.6	37.6	38.0	1.1
3	14.1	15.3	13.9	12.5	9.0	13.2	13.0	1.0	43.0	36.2	39.0	36.0	34.3	26.7	35.9	2.4
4	12.4	14.8	16.5	15.5	13.4	15.1	14.6	0.7	36.3	33.1	38.2	31.5	34.9	37.3	35.2	1.1
5	12.9	12.8	*-	12.1	11.5	14.6	12.8	0.6	39.0	32.9	*-	29.6	33.2	34.4	33.8	1.7
6	12.9	14.7	13.2	*-	12.2	15.9	13.8	0.7	38.9	32.3	38.9	*-	39.9	33.6	36.7	1.7
Final	15.8	13.3	15.8	15.1	13.0	18.6	15.3	0.7	40.2	31.1	41.2	38.6	32.7	42.1	35.9	1.5
Final	16.2	14.2	13.7	16.6	13.9	18.2			36.4	31.0	32.2	34.9	33.1	38.7		

Final tests for subject LR not included in calculations due to adverse effect of bed rest on fitness.

†technical difficulties prevented determination of this parameter.

*illness prevented testing n=5 for tests 5, 6, and Final.

Minute Ventilation at Rest (l/min BTPS)									Breathing Frequency (breaths/min)							
Test	A.K.	C.L.	C.M.	L.R.	C.S.	C.T.	$\bar{X}$	S.E.	A.K.	C.L.	C.M.	L.R.	C.S.	C.T.	$\bar{X}$	S.E
Initial	9.94	10.75	6.14	7.74	8.60	13.44	10.0	1.0	14	17	17	20	14	16	16.3	0.9
Initial	6.89	17.28	7.31	8.70	9.18	14.04			19	16	16	18	14	14		
2	10.77	19.49	7.48	8.64	7.83	9.41	10.60	2.01	18	14	18	19	13	13	15.8	1.2
3	12.42	11.14	6.50	9.96	8.73	8.20	9.49	0.95	16	12	18	17	13	13	14.8	1.1
4	7.53	15.70	6.83	10.77	7.00	9.02	9.48	1.51	15	14	17	19	14	13	15.3	1.0
5	10.02	17.09	*-	9.21	8.15	9.27	10.75	1.80	16	10	*-	17	13	13	13.8	1.4
6	9.78	15.58	6.75	*-	8.73	9.06	9.98	1.66	17	10	13	*-	13	12	13.0	1.3
Final	9.00	15.03	6.27	6.78	9.51	15.49	10.2	1.10	16	10	15	20	15	16	14.8	0.8
Final	8.46	15.82	7.36	7.67	7.65	13.39			16	11	14	16	12	16		

Final tests for subject LR not included in calculations due to adverse effect of bed rest on fitness.

†technical difficulties prevented determination of this parameter.

*illness prevented testing n=5 for tests 5, 6, and Final.

Tidal Volume at Rest (ml BTPS)									Vital Capacity (l BTPS)							
Test	A.K.	C.L.	C.M.	L.R.	C.S.	C.T.	$\bar{X}$	S.E.	A.K.	C.L.	C.M.	L.R.	C.S.	C.T.	$\bar{X}$	S.E.
Initial	710	632	361	387	614	840	632.1	73.5	3.83	3.23	2.71	3.42	4.24	3.81	3.61	.22
Initial	362	1080	457	483	656	1003			3.72	3.59	2.79	2.80	4.15	3.72		
2	598	1392	415	455	602	724	697.7	159.8	3.40	3.25	2.88	3.09	4.04	3.76	3.40	.19
3	776	928	361	586	671	631	658.8	85.0	3.82	3.30	2.84	3.53	4.41	3.67	3.60	.23
4	502	1122	402	567	500	694	631.2	115.6	3.76	3.48	2.60	3.38	4.48	3.63	3.56	.27
5	626	1708	*-	542	627	713	843.2	243.6	3.92	3.62	*-	3.45	4.59	3.67	3.85	.22
6	575	1558	519	*-	671	755	815.6	212.4	3.74	3.52	2.88	*-	4.45	3.69	3.66	.28
Final	563	1503	418	339	634	968	739.3	115.8	3.82	3.65	3.08	3.45	4.52	3.72	3.72	.21
Final	528	1438	526	480	638	837			3.66	3.24	2.99	3.53	4.46	3.74		

Final tests for subject LR not included in calculations due to adverse effect of bed rest on fitness.

†technical difficulties prevented determination of this parameter.

*illness prevented testing n=5 for tests 5, 6, and Final.

Forced Expiratory Volume in 1 sec (% of VC)								
Test	A.K.	C.L.	C.M.	L.R.	C.S.	C.T.	$\bar{X}$	S.E.
Initial	82.6	†-	†-	87.5	92.4	75.1	87.2	4.4
Initial	76.7	89.8	69.8	97.7	88.0	90.7		
2	90.5	94.4	92.6	93.0	97.3	†-	93.6	1.3
3	†-	87.5	88.5	83.5	89.1	89.3	87.6	1.2
4	†-	94.0	96.9	94.3	88.0	90.1	92.7	1.8
5	87.3	92.8	*-	93.8	86.3	89.9	90.0	1.6
6	89.0	96.9	93.2	*-	87.3	89.3	91.1	1.9
Final	90.0	92.2	100.0	93.1	85.1	90.1	94.5	1.7
Final	98.5	92.6	†-	92.5	92.6	90.1		

Final tests for subject LR not included in calculations due to adverse effect of bed rest on fitness.

†technical difficulties prevented determination of this parameter.

*illness prevented testing n=5 for tests 5, 6, and Final.