ABSTRACT

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The purpose of this investigation was to define the relationship between age and several body composition parameters among sedentary (S), active (A), and active cardiac (AC) males, aged 35-72 years. The body density (BD), percent body fat (PBF), and lean body weight (LBW) of the 68 randomly selected subjects was essessed densitometrically. Mean B.D. for the S, A, and AC males was; 1.0424 gm/cc (SD ± .0143), 1.0499 gm/cc (SD ± .0102), 1.0386 gm/cc (SD ± .01499), respectively. Mean PBF (Brozek equation) for the S, A, and AC males was; 24.3% (SD ± 6.3), respectively. Mean LBW for the S, A, and AC males was; 59.46 kg. (SD ± 6.69), 62.54 kg. (SD ± 5.82), and 58.53 kg. (SD ± 5.8), respectively. An ANOVA releaved a significant difference ($P < 0.05$) between PBF in the A and AC groups. No significant relationship ($P > 0.05$) was exhibited between age and B.D., PBF, or LBW, respectively among the A, S, or AC groups or total combined group.

DENSITOMETRIC ANALYSIS OF THE BODY COMPOSITION OF ADULT MALES WHO ARE SEDENTARY, ACTIVE, OR CLINICALLY DIAGNOSED CARDIACS, AGED 35-72 YEARS

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University of Wisconsin-LaCrosse

In Partial Fulfillment
of the Requirements for the
Master of Science Degree

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Master of Science - Physical Education

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Chapter I

INTRODUCTION

During the existence of the human organism there is no point at which a static or stable state is attained, in terms of form or composition. The metabolic processes are continually in a state of change, both on the molecular level and grossly in terms of overall body composition, or the ratio of lean body mass to depot fat (Keys and Brozek, 1953). This continual process of change is brought about by a multitude of factors associated with growth and development and ageing and senescence, upon which are superimposed environmental influences. Examples of such influences are diet, physical activity, and disease. It is the relationship between these environmental factors and body composition that was the subject of this study.

In the last twenty years, several studies have looked at the relationship of age, activity levels, and sex to body composition and alterations to body composition among populations under 30 years of age (Bloom and Eidex, 1967; Lesser, Kumar, and Steels, 1963; Parizkova, 1963; Keys, et.al., 1953; Pollock, Braide, Kendrick, Miller, Janeway, and Linnenrund, 1972; Pollock, Laughride, Coleman, Linnerud, and Jackson, 1975; Greenleaf, Bernauer, Juhos, Young, Morse, and Staley, 1977). Proportionately, few studies have been completed on the body compositional variations associated with increasing age. As early as 1953, Keys, et.al., in their work on body fat in adult man, acknowledged that systematic quantitative data covering middle and older aged populations was not readily available. In

1963, Lesser and his associates and Parizkova both looked at changes in body composition with age. Their respective samples, though small (less than 20), allowed them to draw the conclusion that possible increases in body fat could not be attributed to solely age increases.

Whole body density demonstrates a steady decrease with age (Malina, 1969), indicating either a gain in total body fat or a decrease in the weight of the lean tissues. Since both lean tissues and body fat are influenced by the environmental factors of diet (Parizkova, 1963; Albanese, 1978; Munro and Young, 1978), physical activity patterns (Jokl, 1963; Pollock, Dimmick, Miller, Kendrick and Linnerud, 1975; Wilmore, Miller, and Pollock, 1974; Greenleaf, et.al., 1977; Misner, Boileau, Massey, and Mayhew, 1974), heredity (Wilmore, et.al., 1974), and disease (Weinsier, Fuchs, Key, Tribuesser, and Lancaster, 1976; Mann, 1974) it is difficult to isolate the role of the ageing process in accounting for the magnitude of these declines.

This problem, of isolation, was studied by Wilmore and his associates in 1974 with an indirect approach. Instead of analyzing the population longtitudely the authors studied three highly conditioned endurance male athletes in their eighth decade of life. This samples physiological and body compositional results were then compared with those of a normal population sample of equivalent age and with those of a group of endurance athletes of younger ages. The authors results indicated that "creeping obesity" is not a natural consequence of the ageing process and that older men could maintain a body compositional status similar to that of an adolescent and young adult athlete through vigorous physical activity.

Specifically, they determined that the body fat percentage could be lowered to the level of younger athletes with a conditioning program and with diet. This entire study was performed utilizing the densitometric approach to body composition assessment.

The small, specific, and cross-sectional population utilized by Wilmore, et.al., (1974) to draw the conclusion that "creeping obesity" was not a natural consequence of age was the source of questioning in the present study. The purpose of the present study was to re-evaluate the contentions drawn by Wilmore and his associates. The present study, densitometrically analyzed the body composition of a large, general, and cross-sectional population. Specifically, the present study analyzed the body composition of middle and older aged men who were either normal (asymptomatic for disease) and sedentary, normal and physically active, or had been clinically diagnosed as having coronary artery disease (CAD) but were physically active. The population was chosen because, it was contended that, generally, today's population of middle or older aged males fall into one of these categories and as such is representative of a less specified population.

Statement of the Problem

The avenues of inquiry which were followed in this study were determined by a three-fold problem:

1. There exists little or no information on the body composition of general populations who are middle or older aged males. The conclusions previously drawn by several authors reviewing this subjects

has been based on research populations that have been small, specific, and cross-sectional. Therefore, there was a need for a study that utilized a larger, less specific, and cross-sectional population.

2. Due to the size of the populations studied the contention that "creeping obesity" is not a function of age lacks quantification with a larger and less specific population. Furthermore, a larger population is needed to isolate the role that physical activity may have in the "control" of the contented increase in obesity with age.

3. There exists in the literature little or no support for the contention that differences in body composition and the associated gain and loss mechanisms exists between the three sub-groups, and this concept requires further quantification on a larger population.

Therefore, the densitometric quantification of the relationship of physical activity patterns, age, and body composition forms the primary purpose of inquiry in this investigation.

Need for the Study

There was a need for the analysis and description of the relationship of body composition to age among normal and physically active males, normal and sedentary males, and clinically diagnosed CAD but physically active males. Specific to this need was the use of densitometric analysis as the mode of body composition assessment due to the need for increased comparative densitometric data.

There was a need for the quantifying and validating of Wilmore and his associates (1974) contentions that "creeping obesity" or, more specifically, slow increases in body fat percentage are not natural consequences of the ageing process. This validation and quantification will result from the use of a larger and less specialized population (in contrast to the small population used in earlier studies).

Finally there was a need for the analysis of the body composition of clinically diagnosed male patients with CAD. There presently exists little or no published knowledge on the weight and fat gain and loss mechanisms, as they relate to populations with CAD.

Purpose of the Study

The purpose of the study was to analysis and describe the body composition of middle and older aged males who were either normal and active, normal and sedentary, or active and clinically diagnosed to have CAD. The body composition was determined through densitometric analysis and resulted in the evaluation of the subject's Lean Body Mass (LBM), and Percent Body Fat (PBF).

The subjects were classified into three groups: 1. normal (asymptomatic for CAD) physically active; 2. normal sedentary; and 3. physically active cardiacs. The results of all the groups were statistically analysed. The resulting data allowed for the quantifying of the relationship(s) between PBF, LBM, age, and health status, and physical activity patterns. The study analyzed the relationships between the body composition of....

- Normal Active Males and Normal Sedentary Males
- Normal Active Males and Physically Active Cardiac Males

- Physically Active Cardiac Males and Normal Sedentary Males and

analyzed the relationships between age and....

- Normal Physically Active Males
- Normal Sedentary Males
- Physically Active Cardiac Males.

Hypotheses

Tnree hypotheses will be investigated experimentally. All three hypotheses have been couched in the null form.

- The body composition of adult males, who either participate in regular physical or who are sedentary shall not differ. ($P>0.05$)
- The body compositional adjustment mechanisms associated with regular activity among normal adult men shall not differ from those mechanisms of adult men with clinically diagnosed CAD.
- Fat weight increases are not a natural consequence of the ageing process and can be controlled by physical activity, irregardless of the health status.

Assumptions

This study assumed that the subjects understood the instructions as to their intent and procedure. Furthermore, this study assumed that the subjects performed these instructions to their best of their ability.

Also, this researcher assumed that the apparatus used to assess body composition were be valid and reliable.

Delimitations

This study was delimited to the random selection of 68 middle and older aged males between the ages of 35 and 75 years. This sample consisted of 30 randomly selected active males from the Adult Fitness Unit of the LaCrosse Exercise Program, 18 randomly selected active and clinically diagnosed CAD males from the Cardiac Rehabilitation Unit of the LaCrosse Exercise Program, and 20 randomly selected sedentary males from the University of Wisconsin-LaCrosse academic faculty and the City of LaCrosse community.

This study was further delimited to a single evaluation session during which Body Density and Residual Volumes was assessed.

Limitations

Within the boundaries of this study, certain limitations were recognised:

1. The method body volume assessment was limited to the hydrostatic weighing approach.
2. The assessment of residual volume was limited to and calculated by a modified closed-circuit oxygen rebreathing method outside the hydro0 static weighing tank.
3. The subject's motivation and capacity to perform the maximal expirations required during the body volume and residual volume assessments could not be directly controlled.
4. The subject's adherance to the instruction to fast for 12 hours prior to their body composition could not be monitored or controlled.

Definition of Terms

Active Male Males who are asymptomatic for coronary heart disease and participate in regular physical activity.

Adipose Tissue A form of connective tissue consisting of fat cells lodged in areolar tissue and arranged in lobules along the course of small blood vessels (Blakiston, 1973).

Body Density The concentration of the body, measured as a mass per unit volume. Density (Kg./L.) Mass (Kg.) Volume (L.). In man, the density of the body usually from 1.000 KG./L.[1] to 1.100 Kg./L.

Body Fat The neutral lipid content of adipose tissue which is exclusive of the cellular framework (Muldowney, 1961). This neutral lipid forms the ether-extractable fraction that is assessed in densitometry.

Cardiac Males Males who have been clinically diagnosed as having coronary heart disease, either by the method of cine-angiography, the completion of a positive Graded Exercise treadmill test, or having had a myocardial infraction.

Coronary Artery Disease The biological process where atherosclerotic plaque deposits decrease the opening of the lumen in the coronary arteries.

Closed-Circuit Oxygen Rebreathing Method A method for assessing residual volume developed by Lundsgaard and Van Slyke in 1918 (Wilmore J., 1969) and modified by Wilmore in 1969.

Densitometry The science of assessing body density.

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1. In extremely obese subjects body density values of less than 1.000 Kg./L. have been experienced and reported (Keys, et.al., 1953).

Hydrostatic Weighing A method by which the volume of the body can be assessed. Hydrostatic weighing is based on the Archimedian principle that a body immersed in a fluid is acted upon by a buoyancy force, which is evidenced by a loss of weight equal to the weight of the fluid displaced (Behnke et.,al., 1974).

Lean Body Mass All the tissues of the body with the exclusion of stored fat. It is comprised of bone, muscle, and organ tissue. Lean body mass includes the functional fat which is incorporated into the structure of the cells.

Percent Body Fat The proportion of body fat to total body weight (PBF).

Regular Physical Activity Participation in physical activity or exercise which is performed with an intensity of 65% to 85% maximum predicted heart rate, for a duration of 45 minutes per session, an a frequency of three sessions per week.

Residual Volume The volume of air left in the lungs at the end of a maximal expiration.

Sedentary Males Males who do not participate in regular physical activity.

Chapter II

REVIEW OF LITERATURE

Early Studies in Body Composition Analysis

The 17th Century anatomist Ambroise Paré wrote of fat;

> The fat is of an oily substance bred of the aiery and vaporous portion of the blood........The greatest part of it lies between the fleshy pannicle and the common coat of the muscle, otherwise,it is diffused over all the body, in some places more, in some places less, yet it is always about the nervous bodies, to which it delights to cleave........The use (of fat) is to moisten the parts which can become dry by long fasting, vehement exercise or immoderate heat, and besides to give heat, or keep the parts warm (Pare, 1634).

This description of the role and structure of fat was very perceptive for its time. Pare recognized the effects exercise had on this body component, the insulatory effect it had, and the areas of the body within which deposition commonly occurs. The assessment of this bodily compartment has been studied for the past century by numerous investigators Historically, this subject has been approached with direct and indirect analysis methods. A century ago, the concept of measurement of fat (and its role) began to dominate the work of the German anatomists, and for fifty years there was much effort devoted to direct measurement, physical and then chemical, of the body and all its parts (Keys, et.al., 1953). From this period came the data which, until very recently, was all that was known about the gross composition of the human body. In 1888, Vierodt, in his "Tabellen", gave four pages of tables of the specific gravities of the anatomically seperable parts of the human body, including hair, epidermis from various regions: even such minute structures as the cornea, the vitrous body, and the lens of the eye (Keys, et.al., 1953). Unfortunately, their data was derived from only two cadavers and; as has been shown, not completely applicable to the general population (Keys, et.al., 1953). More recently, through the work of Widdowson, McCance, and Spray (1951) and Mitchell, Hamilton, Steggerda, and Bean, (1945) five cadavers were analysed and produced results which could act as calibration data for indirect analysis. The direct analysis of the gross body composition is a laborious procedure which is not commonly conducted and the results of such procedures are still subject to question on the basis of the specificity of the cadavers, in terms of their age, sex, and "normality". This specificity makes it difficult to apply the results to the general population and the only way to correct such a dilemma is to increase the number of cadaver analysis (which brings up the original applications problem of the laboriousness of the methods and the applicability of the results). Therefore, while direct anatomical and, more importantly, the chemical methods of analysis of body composition are fundamental, the study of living individuals must be accomplished indirectly.

Implicit in indirect methodologies (for the assessment of body composition) is the need to partition the body into components (or compartments). The use of total body weight (TBW) is not alone indicative of a subject's body composition, for TBW represents, in different individuals, very different mixtures of the body's basic components - bone mineral, fat, extracellular fluid, and "cells" (Keys, et.al., 1953). In the in vivo analysis of body composition it is possible to use models made up of four components (fat, water, protein, and mineral) or two components (fat and fat-free matter) (Malina, 1969). Four component models are most common among those who conduct direct cadaver analysis. The two component models are the most common among researchers involved in the indirect analysis of the body. This model is used in quantifying the percentage of body weight that is fat and that which is lean tissue. There are several indirect methods commonly used to assess these two major components of the body; densitometry, Potassium-40 counting, total body water analysis, fat-soluble indicators, and anthropometrics. These methods will now be discussed.

Determination of the density of the body can be traced back to Archimedes and his study of floating bodies. It was through Archimedes' establishment of the principles of specific gravity that present day scientists are able to effectively assess body composition indirectly. The earliest record of a specific gravity (of the body) observations have been attributed to an English librarian, John Robertson, in 1814.

This gentleman constructed a cistern large enough to hold a man and had ten "middling-sized men" plunge into it seperately. Robertson then measured the accorded rise in the water level. Knowing the specifications of the cistern and the weight of his subjects Robertson calculated a crude specific gravity for each of his subjects. His primary motivation for the experiment was the determination of the quantity of wood needed to keep a man afloat in water. His results indicated "momentous" decreases in total body weight in water and therefore only an "oar" would suffice for support in water (Spivak, 1915). A correlation between corporeal density and fatness was suspected in 1901 by Stern (Behnke, 1961). Unfortunately, this researcher lacked an accurate technique for measuring body density and therefore could not establish a well-defined relationship. The same problem, lack of an accurate methodology, plagued Spivak (1915) in his attempts to quantify the relationship of corporeal density and fatness. In 1933, Boyd performed a comprehensive analysis of all the specific gravities (irregardless of methodology reported in the literature between 1906 and 1933 (a total of 787 specific gravities). Even though these specific gravities were calculated by both underwater weighing methods (189) and water displacement methods (598) and during various phases of respiration, Boyd still concluded "obesity tends to decrease specific gravity". It was not until the early 1950's that researchers began to discern the effective differences which resulted from the use of specific gravity versus density of the body. The differences created by selection of the wrong parameter was eloquently described by Keys and Brozek in 1953. These authors determined that specific gravity was an abstract figure which only represented the ratio of the body's density to the density of water, at a sepecified temperature. In contrast, density is a dynamic figure that is the mass of the subject divided by its volume and is thus expressed, normally, in gm./cc.. Through the correct use of density and the actual calculation of the subject's lung volume (during the hydrostatic weighing procedure) an accurate methodology was established which was more sympathetic to alterations in either the fat or the lean components of the body. The principle and application of densitometry will be discussed in greater detail in chapter III. Notation should be given to the fact that prior to 1942 little or no attention was given to the effect of water temperature on body density and thus those findings reported prior to this recognition make comparisons with later determined data difficult.

Through the determination of the body's density it became possible to make more accurate determination of the body's components, specifically, the percentage of the body that was fat. In order to determine the percentage contribution it is essential to know the density of the body's fat and lean tissue component. Knowledge of the accurate density of human fat relates back to the earlier discussion on the number of in vivo direct cadaver analysis performed. It is through these analysis that the required calibration data emerges. The work of Widdowson, et.al., (1951) and Mitchell, et.al., (1945) supplied the scientific community with density figures for body fat and lean body tissue. One criticism still heard regarding these data is that the resultant data remains cadaver specific and limited in its application to the general population. The data presented by these authors allowed for the development of a "reference man" (Keys, et.al., 1953). The "reference man" served as the tool for determining PBF from total body density for 10 years. In 1963, Brozek and his coworkers reviewed the quantitative assumptions that had been prevalent in this area of study. These authors critically analysed the extrapolated cadaver data that made up the "reference man" and developed what is now referred to as the "reference body". The primary difference between the "reference man" and the "reference body" is the definition of fat. The authors determined that a different density was exhibited in fat when the fat component of the body was defined as "weight gained", "weight loss", or "static weight difference". The review by these authors was necessary because, depending upon the way in which fat was defined, different equations could be developed. These varying equations created differing translations of total body density into PBF and percentage lean body mass values.

The calculation of the volume of the body has been performed by several methods. Two such methods have been described, hydrostatic weighing and water displacement. A comparison of these two approaches was performed by Ann Ward and her co-workers in 1978. Ward et.al., found a significant difference between the mean volumes determined by underwater weighing and water displacement methods ($P<0.05$) (in terms of percent body fat the differences between the two methods was 0.7%). It should be noted that a correlation of 0.96 was determined even though the differences were significant. In 1963, Graedinger, Reinecke, Pearson, Van Huss, Wessel, and Montoye studied the differences between body volumes determined by underwater weighing and the popular (at that time) German approach of air displacement. The results of this study demonstrated a inter-correlation coefficient of 0.36 and a significant difference between the two methods. Furthermore the results indicated that both methods could distinguish between individuals differing widely in density, but not between individuals of similar density. The authors attributed the variations (in the air displacement method) to the inability to accurately obtain readings of temperature, pressure, and relative humidity. These same authors also analysed the relationship between the helium dilution technique of body volume measurement (popularised by

William Siri) and the air displacement methods. The intercorrelation coefficient between the methods was 0.19. The researchers used fourteen market weight pigs and attributed the extremely low correlation to the activity of the animals inside the chamber, which caused rapid changes in the concentration of helium.

As mentioned earlier, there are available other methods of body compositional assessment; total body water methods, potassium 40 methods, and anthropometrics (skinfolds). Several studies have reviewed the relative attributes and disadvantages of these methods (Yang, et.al., 1977; Myhre, et.al., 1966; Kryzwicki, et.al., 1974). Kryzwicki and his associates (1974) compared total body water method values with body underwater weighing approaches and with potassium 40 body composition calculations. These authors failed to reach a conclusion that clearly defined which method was the most accurate. This study demonstrated inter-correlations ranging from 0.47 to 0.72. Although significant differences in absolute magnitudes of the estimates were found, the three techniques are highly correlated (that is, relative magnitudes were similar across techniques). Kryzwicki's conclusions where found to be in contrast to those found by Myhre, et.al., (1966). Myhre's study elicited a correlation of 0.87 between K_{40} and Densitometry, but with a significant difference in the means. The potassium 40 method demonstrated an average 4% higher readings over the underwater weighing method. Equally important was the fact that the overestimation ranged from 0.4% to 22%. The differences concluded in this study were greater in older subjects; the relation of age to the magnitude of these differences was statistically significant. It was suggested that this discrepancy depended on the increase with ageing in the ratio of protein structures low in potassium, as in connective tissue, to protein structures high in potassium, as in muscle. Such replacement would lead to an under estimate of lean body mass by the scintillation counting method but would not affect the estimate obtained by densitometry.

Anthropometric equations have been developed so that a mobile and filed method of evaluation could be utilized. The equations developed are regression equations based on either body density, PBF, or lean body weight calculations. The accuracy of these equations is therefore dependent upon the calculated base parameter. As has been, discussed the relative accuracies of these base parameters are dependent upon the relative accuracies of the methods used to calculate the parameter.

It is now clear that all of these methods are estimates of what the actual body composition of a subject is. In terms of the densitometry approach the "translation" of the body's density into a relative PBF component is perhaps the "weak" point of the approach. Many researchers have evaluated the actual densities of the body's lean body component and the body's fat component. These values, as will be further discussed in the methods chapter, were developed on the basis of limited cadaver analysis. Therefore, the applicability of the values calculated to the general population are a genuine source of criticism. The reasons why the calculated component densities (from a singular assessment) may not be generally applicable to all populations and, specifically, all ages will now be discussed.

Body Composition and Age

The ageing process is characterized by a long list of functional and structural changes. One of the most notable two component model, the body can be divided into a fat component and a fat-free component (lean body mass). Initially, discussion will center on the alterations of lean body mass (LBM) with increasing age. This will be followed by an analysis of the ageing process and its effect on body fat component.

Lean Body Mass and Age

In 1972, Forbes studied the growth of LBM in man. Utilizing cross-sectional data, this author illustrated that the male exhibits a pronounced adolescent "spurt" in LBM growth, which reaches a maximum by age 20-25, or at about the age at which maximum height was attained. This finding was in agreement with that of Novak, (1963), who determined that maximal LBM was attained during the "third decade" of life. That this is a true maximum is further quantified by the documented declinein LBM during the adult years (Forbes, 1976; Forbes and Reina, 1970; Lesser, Kumar and Steele, 1963; Tzankoff and Norris, 1977).

Graphically, the relationship between LBM and age is a positively upsloping curve, up to approximately 25 years of age. The velocity of the rate of growth of LBM may be lower than reported by Forbes (1972) due to the cross-sectional data utilized. Forbes (1972), himself, expounds the hypothesis that a longtitudional analysis of a series of individuals could present a broader and lower rate of growth of LBM.

With the attainment of a maximal LBM during man's third decade of life there commences a slow but calculable decline in his component. The question has been raised as to whether this decline is due to biologic or secular origins. In attempting to answer this question several researchers completed both longtitudional and cross-sectional studies (Forbes, et.al., 1970; Forbes, 1976; Kuta, Parizkova and Dycka, 1970; Parizkova, 1963; Norris, Lundy and Shock, 1963; Myhre and Kessler, 1966).

Forbes (1976), in a longtitudional study on a small sample of four subjects, demonstrated a loss of LBM at a rate of 0.36 kilograms per year. This figure was developed by the analysis of a regression slope of this sample, over a 14 year period and a minimum of 13 assessments during this period. An important observation of this study was that some subjects gained weight while others lost weight during the period of observation, yet there was no marked alteration to the continual decrease in LBM. Therefore, there appeared no relationship between total body weight and LBM. Sims, Goldman, Gluck, Horton, Kelleher, and Rowe (1968), in a study on experimental obesity, deliberately over-fed male volunteers. This over-feeding resulted in both increases in PBF and LBM. Sims, et.al., (1968) concluded that, even though LBM increased, the increases in LBM were not statistically significant. These findings add credence to the notion that total body weight and LBM tend to be independent (in terms of LBM's decreasing capacity). Further complimenting this notion, are the results of Forbes, et.al., (1970). In this cross-sectional study, an analysis of 9000 subjects was conducted, and the conclusion/observation was made that LBM declined at a rate of 0.29 kgm. LBM. per year in men.

Further evidence of the decline has been offered by Keys, Taylor and Grande (1973). These authors studied the relationship between basal metabolism and age in adult men. During this study, the authors demonstrated a gradual reduction in the size of the man's LBM with increasing age. These authors further observed that the LBM declined gradually during thr fourth and fifth decades followed by a more rapid decline up to their seventh decade of life (and death). This observation was also noted by Tzankoff, et.al., (1977) and Shukla, Ellis, Dombrowski and Cohn, (1973).

Several investigators, previously mentioned, have reported estimates of LBM based on total body water, body density, or Potassium 40 (K_{40}) methodologies. The variation associated with such methodologies are calculable, but the conclusion is still undeniable; the decline of LBM is a biologic, not secular, fact of adult human life.

Concern can now be turned to the contributing factors to the age-related decrements in LBM. The primary factors contributing to LBM are muscle mass, skeletal mass, vital organs, and the body's fluid compartments.

Muscle Mass: Muscle accounts for approximately 25 percent of body weight in a neonate, for approximately 45 percent in a young adult, and for approximately 27 percent in people more than 70 years of age (Munro and Young, 1978). This, represents a decreased contribution of muscle mass to LBM with increases in age. Ageing is associated with changes in the amount and distribution of protein in the body. Forbes, et.al., (1970) demonstrated that whole body potassium levels (an index of protein content) decreases with age.

Data from post mortem studies have shown that a major proportion of the decrease in protein results from the breakdown of skeletal muscle (Korenchevsky, 1961). Further support for this finding comes from studies on the relationship between creatinine excreation and LBM. The urinary output of creatinine is assumed to be a measure of muscle mass (Garn, 1963; 1961). The amount of creatinine excreted is decreased in elderly subjects, and the decrease correlates with their smaller body cell mass, as determined by whole potassium levels (Malina, 1969; Tzankoff, et.al., 1977; Lesser, et.al., 1963; Norris, et.al., 1963).

The rate of whole-body protein turnover (synthesis and breakdown) falls as a young man approaches adulthood and then continues to decline slowly during adult years (Munro, et.al., 1978). Long, Haverberg and Young (1974) determined that a primary contributant to the protein loss in the body (via the notable decrease of skeletal muscle mass) is the breakdown of the actin and myosin proteins.

Skeletal Mass: In adult men, bone mineral compromises of approximately 4 to 5 percent of the total body weight (Merz, Trotter and Peterson, 1956). Trotter, Bromen, and Peterson, (1960) analyzed the bones of several Negroes and Caucasians with view of observing the possible decreases in density with age. These authors found such a decrease. They determined that the density of adult bones decreased with age at a uniform rate. Furthermore, the authors were quick to point out that a decrease in bone density (mass) is not necessarily accomplished by a parallel decrease in bone mineral (e.g. ash). Basically, the conclusion that bone density decreases with age has been accepted by the medical community, but argument still exists as to whether maintained bone mineral can diminish this age-related phenomena.

Osteoporosis is recognized as a decrease in total bone mass (therefore...density) without changes in the chemical composition (that is, normal calcium; protein ratio). This clinical entity is found, primarily, in middle and older-aged women, but is not uncommon amongst similar aged mens groups. Osteoporotic bone is a recognized contributor to decreased LBM and other anatomical changes e.g. height (Albanese, 1977).

Vital Organs Mass: As mentioned, ageing is accompanied by changes in the amount and distribution of protein in the body. Tzankoff, et.al., (1977) expressed the poinion that in "healthy men" the combined mass of vital organs are "not likely" to change "much" with age. Tzankoff and his associates have assumed that, generally, these physiological and anatomical decreases do not occur. There is evidence that vital organs do decrease their contribution to LBM (Brozel, 1952; Munro, et.al., 1978). Through the breakdown of their protein construction. For example, the liver accounts for a progressively smaller proportion of the body weight from birth onwards; 4% in a neonate, about 3% in a young adult, and approximately 2% in an elderly person (Guyton, 1976). The decreasing contribution of the body's vital organs to LBM varies from individual to individual and still requires greater research. Notably, age-related decreases in vital organ mass are contributory to decreases in LBM.

Fluid Compartment Mass: In 1963, Lesser, et.al., demonstrated several alterations in the body's cellular fluid (extra-cellular fluid) structure with age. These author's found an increase in the proportion of extra-cellular fluid in the body's lean tissues (LBM) with age. In the youthful and small (N=3) population studied a mean (ECF/LBM X 100) of 18.2% was demonstrated, in contrast to 21% in an older population (N=5). Lesser, and his associates, also demonstrated decrease in the proportion of intra-cellular fluid (ICF) and LBM with age. The ICF/LBM averaged 52.9% for the young subjects (N=3) and 49.5% for the older subjects (N=5). Based on this sample the authors concluded that these cellular fluid changes could not be solely attributed to obesity; and therefore, viewed them as a function of the ageing process. These alterations in the cellular fluid distribution further contribute to the overall decrease in LBM of the body with age.

Body Fat and Age

The effect of ageing on the fat component of the body has been extensively reviewed and researched (Parizkova, 1963; Lesser, et.al., 1963; Wilmore, et.al., 1974; Krzywicki, et.al., 1967; Parizkova, 1961; Novak, 1963; Brozek, 1952). In an extensive analysis of a large population sample, Parizkova (1961) attempted to illustrate the growth of body fat in males between the ages of 9 and 17 years. This author demonstrated a progressive increase in density which translated into an decrease in body fat percentage of total body weight. Parizkova showed a decrease in fat percentage from 21.5% at 11.5 year to 11.7% at 16.5 years. This decrease in body fat was also demonstrated by Hunt and Heald in 1963. These authors demonstrated a decline from 23.4% at 12 years to 13.1% at 17 years. Also in 1963, Heald, Hunt, Schwartz, Cook, Elliot and Vajda demonstrated a linear decline in PBF between the ages of 12 and 18 years according to the regression equation,

$$\begin{gathered}\text{PBF} = 48.22 - 2.07\ (\text{Age}) \\ \text{Standard Error of Estimate} = 5.9\%\end{gathered} \tag{1}$$

these authors showed the mean fat content of 12 year olds in 23.4% of body weight, and by 18 years of age it has declined to 10.9%.

Several recent studies have utilized populations with ages ranging from 18 to 26 years in the development of equations which predict various body compositional parameters (Pollock, Hickman, Kendrick, Jackson, Linnerund, and Dawson, 1976; Katch and McArdle, 1973; Sloan, 1967; Haisman, 1970; Durnin, and Rahaman, 1967; Wilmore and Behnke, 1968; Wilmore and Behnke, 1969). In developing these equations it was necessary to assess the density and the (PBF) of these populations by one of the accepted indirect methods previously discussed (the majority of the above mentioned studies utilized the densitometric approach to body composition assessment). In Table 1., a summary of the values of PBF reported in the literature as they relate to the population's occupation and age. The results of these nine studies demonstrate that between the age ranges of 17 years to 26 years, the PBF ranges from 10.9% to 15.3%. Therefore, by summating these and other studies on comparable samples (Nagamine and Suzuki, 1964) an overall statistical picture develops. For a population (n=821) with a mean age of 21.46 (range = 17-26), a mean of 13.5% (Mean range = 10.9% - 15.3%) was demonstrated.

In 1976, Pollock, et.al., reported the body compositions of young and middle-aged males. These authors assessed the body composition of 95 young men, with a mean age of 19.7 years (range = 18-22). As previously related, they demonstrated a mean PBF of 13.6% (S.D.= ±6.0%). The study utilized the densitometric approach to body composition assessment. The authors then assessed, densitometrically, the body composition of 84 healthy and sedentary middle-aged men with a mean age of 44.9 years (range = 40-55 years). This sample was shown to have a PBF of 24.7% (S.D.= ±5.9%). This was found to

Table 1. SUMMARY of RELATED LITERATURE ANALYSIS of PBF of YOUNG POPULATIONS

STUDY	SUBJECT	MEAN AGE	MEAN TBW	MEAN PBF *
Brozek and Keys (1951)	133 students	20.3	69.1	12.1
Pascale (1956)	88 soldiers	22.1	68.3	13.7
Sloan (1967)	50 students	22.4	70.6	10.9
Durnin and Rahaman (1967)	60 young men	22.0	68.2	13.7
Wilmore and Behnke (1968)	54 students	22.7	74.1	15.3
Wilmore and Behnke (1969)	133 students	22.0	75.6	14.6
Haisman (1970)	55 soldiers	22.6	68.7	12.5
Katch and McArdle (1973)	53 students	19.3	71.4	15.1
Pollock, et.al., (1976)	95 students	19.7	74.6	13.6

* PBF was calculated from the originally reported body densities by use of the formula of Brozek, et.al., (1963), where PBF - $(4.57/D_b - 4.142) \times 100$.

be a significant difference ($P<0.01$) in PBF. Further analysis of the raw data showed that the mean total body weights and ages of the young and middle-aged samples were significantly different ($P<0.01$). It was also noted that the lean body mass of the middle-aged group was significantly lower ($P<0.01$) than the younger aged group. The authors, therefore, noted that the significant decrease in LBM between the groups pointed to body fat accumulation as being the contributor to the increased total body mass in the middle-aged group. The authors further suggested that increases in age are associated more with fat accumulation. This observation was agreed with by the research of Brozek, et.al., (1951), Krzywicki, et.al., (1967), Malina (1969), Lesser, et.al., (1963), Brozek and Keys (1953), and Behnke and Wilmore (1974). It should be noted that only one study reviewed reported no significant differences in PBF between young, middle and older-aged males (Norris, et.al., 1963).

In the analysis of 273 clinically healthy men, divided into age groups ranging from 20 to 55 years, Brozek, et.al., (1953) demonstrated two observations: (1) within each age group the portion of body weight, accounted for body fat, increased with increasing relative weight and (2) at the same value of relative body weight, the fat content is considerably larger in older individuals. These observations led Brozek, et.al., to conclude that, for men of standard weight (for age, sex and height), the relationship between fat content (as a predicted percentage of weight) and age (X) is calculable by the equation:

$$PBF = 0.92836\ (X) - 0.006776\ (X2) - 5.55564 \qquad (2)$$

Data for several ages between 20 and 55, derived from this equation are given in Table 2.

Recently, disagreement with the "fact" that fat accumulation is a natural consequence of age has emerged (Wilmore, et.al., 1974; Pollock, Miller and Wilmore, 1974; Grimby and Saltin, 1966; Pollock, Cureton and Grenninger, 1969). These studies, criticizing this "natural" development, are based on the observations that fat accumulation can be decreased, maintained, or increased by various environmental parameters (for example, diet, physical activity, heredity) irregardless of the subject's age. The observation that increases in fat accumulation may be due to sedentary lifestyle rather than the ageing process shall be discussed and reviewed in the following section.

In summary, the LBM of the male increases linearly up to approximately the attainment of maximal height (which usually corresponds with the middle portion of the subject's third decade of life). The body's LBM decreases slightly during the fourth and fifth decades of life followed by a decline to death. Forbes (1972) and Keys, et.al., (1973) demonstrated a mean decline of 0.29 kilogram of LBM per year. The decreases in LBM have been attributed to decreases in muscle mass, bone density, vital organ mass, and alterations in the body's intra- and extra-cellular fluid components.

Body fat, as a component has been observed to increase from the neonate to immediately post-puberty (Forbes, 1952; Forbes, 1964; Lohman, Boileau and Massey, 1975). During post-puberty (mean age = 12 years) there is a linear decrease in density up to approximately 18 years. Since LBM is increasing during this period, the decrease in density is attributed to a decrease in the PBF. Previously mentioned data illustrated that the mean

Table 2. AVERAGE PBF for MEN of STANDARD WEIGHT but DIFFERING in AGE.

AGE (years)	20	25	30	35	40	45	50	55
PBF *	10.3	13.4	16.2	18.6	20.7	22.5	23.7	25.0

* Values established by use of the equation,
PBF $0.92836 \text{ (age)} - 0.006776 \text{ (age}_2\text{)} - 5.55564$.

PBF of normal healthy men, between the ages 17 to 26 years, is in the range of 10.9% to 15.3%. Also previously mentioned data demonstrated that, with developing and increasing age, the proportion of fat to total body weight increases. That is, there appears to be a trend that fat accumulation is a natural consequence of the ageing process. Arguments have been placed that say this increase in PBF is solely attributable to the linear decrease in the body's LBM component. Furthermore, recent arguments have been placed that argue that fat accumulation is a consequence of sedentary lifestyles and environmental factors rather than a biological phenomena. This hypothesis shall now be discussed.

Body Composition and Physical Activity

The relationship between body composition, age, and lifestyle (physical activity) has received a considerable amount of scrutiny over the past 15 years. The work of a number of investigators over the past 25 years has demonstrated unequivocally that exercise is an effective agent in either the control or alteration of body composition or both (Parizkova, 1963; Misner, et.al., 1974; Pollock, et.al., 1969; Pollock, et.al., 1972; Pollock, et.al., 1975; Sims, et.al., 1968; Jokl, 1963; Greenleaf, et.al., 1977). The actual mechanisms involved in precipitating these alterations appear to be considerably more complex than the simple imbalance between caloric intake and caloric expenditure.

Experimental Studies

The alterations of the relationship between the body's fat component and fat-free component via physical activity (whether it be habitual or sporadic) have been analyzed in both cross-sectional studies (Boileau, Bruskirk, Horstman, Mendez and Nicholas, 1971; Parizkova, 1963) and longtitudional studies (Brozek, 1952; Wilmore, Royce, Girandola, Katch and Katch, 1970; Pollock, et.al., 1972). The present discussion will center on these relationships and the resultant effects.

As early as 1952, Brozek suspected that the fat component of the body could be affected by an increase or decrease in physical activity. This suspicion was based on the observation that males who habitually participated in physical activity demonstrated a leaner state that an equivalent population who were sedentary. Parizkova, (1963) further analyzed the relationship. He observed that, through analysis of active and inactive groups, a higher density was always found in the active groups. This author made further observations which demonstrated more than a direct relationship. He observed that the maintenance of a greater proportion of lean body weight at the expense of fat is not an entirely constant characteristic in physically active persons. He based this observation on a longtitudional study of sportsmen. This study demonstrated that sportsmen displayed a dynamic dependency of their body composition on the intensity of physical training. Parizkova observed, when testing a group of male and female Olympic gymnastic team members, that during the team's preparatory intensive training period, body weight virtually did not change but the amount of subcutaneous and total body fat strikingly fell along with a concurrent development of their lean body weights (both shown by body density alterations and measurements). These observations were complimented by the fact that after interruption of the intensive training the athletes weight rose, attributed too an increase in fat accumulation. Parizkova concluded,

> .....that the lean body mass and depot fat are in a dynamic state of equilibrium, which relatively rapidly and significantly reflects changes in energy output and balance; during increased muscular activity the lean body mass hyperthrophies and fat disappears, and with substantial reduction of muscular activity muscular mass decreases slightly in size and fat accumulates (1963).

More recently quite a number of physical conditioning studies have noted the effects of intensity, duration, frequency, and modality of exercise on body composition (Boileau, et.al., 1972; Greenleaf, et.al., 1977; Katch and McArdle, 1977; Misner, et.al., 1974; Pollock, et.al., 1969; Pollock, et.al., 1972; Pollock, et.al., 1976; Sims, et.al., 1968; Wilmore, et.al., 1970).

Pollock has been prolific the past 10 years in studying the effects of intensity and frequency of exercise on body composition. In a 1969 study, Pollock and his associates formed two experimental and one control group with ages ranging from 28 to 39 years of age. The experimental group I exercised for two days a week and group II exercised for four days a week. Both groups exercised for 30 minutes a session for 20 weeks. Their program consisted of continuous running, jogging, and walking with increasing intensity as exercise tolerance improved. Body compositional differences were assessed anthropometrically utilizing a predetermined population-specific equation (basically, the sum of six treated skinfold measurements) (Pollock, et.al., 1969). The results (in terms of the sum of the six skinfolds) demonstrated that the control (sedentary) group increased their accumulation of fat (122mm to 135mm); the twice-a-week group remained statistically the same, and the four-times-a-week group decreased their fat stores appreciably (131mm to 108mm). The total body weight of the four-day-a-week group dropped appreciably also (79.7 kg. to 76.8 kg.). The losses in body fat stores are in agreement with the findings of Wilmore, (1974), Roby, (1962), Pollock, Miller and Kendrick, (1974), and Curetin and Phillips, (1964). These authors also studied the effects of conditioning programs, on body composition and found that the accumulated fat loss was more noticeable in experimental groups that exercised a greater number of times per week than in groups exercising a less number of times per week.

Since the 1969 study, Pollock and his associates have further studied the effects of twice-a-week and four-times-a-week training programs. In 1972, these investigators maintained two groups on the twice-a-week regimen but varied the intensity of the workouts. That is, the two groups exercised and trained at eighty percent and ninety percent of their respective maximum heart rates. Again, a 20 week training period was utilized. The results of this particular study showed that the 90% group recorded significant reductions in subcutaneous fat stores (as ascertained by the sum of the six skinfolds previously validated by Pollock) (149mm to 138mm). The alterations of the 80% group were not significant. Also, changes in total body weight were not significant in either group.

Two years later Pollock and his associates (1974) conducted a follow-up study on the four-day-a-week training schedule. Again, Pollock manipulated the parameter of intensity. In the original 1969 study, the training was performed at 90-95% of the subject's maximal heart rates; this intensity was thought to be impractical (and undesirable) and thus the experimentors wished to evaluate the effects of a lower intensity but with the same frequency, modality, and duration on an experimental group. The results indicated that this alteration (that is, training at a level of 80% of ones maximal heart rate) in intensity resulted in significant decreases in subcutaneous fat stores.

The unfortunate aspect of Pollock's studies was that they did not utilize body composition methods sympathetic to alterations in body compartments other than subcutaneous fat depots. As early as 1962, Parizkova discussed the relative increase in lean body weight with conditioning. He further discussed the state of equilibrium that occurs between fat and lean body weight and the relative increase in lean body weight with conditioning that could be the contributing factor to the PBF decreases reported. The degree of lean body weight changes (attributable to a conditioning program) have proven to be overly significant. Slight increases in lean body weight (1 to 4% increases) have been reported in several studies (Pollock, et.al., 1972; Wilmore, et.al., 1971; Pollock, et.al., 1974; Boileau, et.al., 1971; Wilmore, et.al., 1970). These slight increases in lean body weight have been attributed (by all the above mentioned authors) to muscular hypertrophy. The slight increases tend to vary according to the modality used in the conditioning program. For example, the degree of muscular hypertrophy was greater in subjects who were conditioned on a weight training regimen (Misner, et.al., 1974; Wilmore, et.al., 1974). In contrast a smaller lean body weight variation was noted in studies utilizing jogging, running, or walking as conditioning modalities (Pollock, et.al., 1974; Wilmore, et.al., 1970).

Table 3 summarizes the data collected from studies and illustrates the

Table 3.

A REVIEW of RELATED LITERATURE. AN ANALYSIS of the EFFECTS OF CONDITIONING PROGRAMS on TOTAL BODY WEIGHT, PBF, AND LEAN BODY WEIGHT; a PRE-POST-ANALYSIS

STUDY	SUBJECT NO.	MEAN AGE	MEAN TOTAL BODY WT. (kg)		MEAN PBF		MEAN LEAN BODY WT. (kg)	
			Pre	Post	Pre	Post	Pre	Post
Boileau, et.al., (1973)	14	34.7	86.3	86.6	29.1	28.5	60.75	61.4
Pollock, et.al., (1975)	26	38	84.7	83.4	21.7	19.9	66.32	66.8
Misner, et.al., (1974)	18	38.3	85.5	85.6	26.9	24.2	66.25	64.5
Pollock, et.al., (1972)	24	37.5	81.1	80.4	23.1	22.6	62.37	62.2
Wilmore, et.al., (1970)	55	38	79.6	78.6	18.9	17.8	64.21	64.4
Wilmore, et.al., (1971)	44	48.1	81.4	78.5	24.5	20.9	61.1	61.8

fact that physical activity elicits a decrease in total body weight and total body fat.

To explain the losses in total body fat one has to turn to the concept of energy (calorie) expenditure. The loss of body fat is associated with an increases expenditure of calories. It should be noted that the caloric equivalent of many activities used in the conditioning studies quoted were under that necessary to account for the fat loss demonstrated in these studies. The work of Mayer (1963) demonstrated the possibility that there was a concomitant decrease in caloric intake with exercise, which results in a caloric imbalance of the magnitude necessary to explain the fat loss. Mayer and his associates demonstrated that exercise up to one hour in duration per day tends to suppress appetite. Several researchers have investigated the area of biochemistry for answers to this fat loss mechanism. Crews, Fuge, Oscai, Holloszy and Shark, (1969) argued the possibility that exercise stimulates lipolytic activity. At this stage, definitive conclusions have not been drawn relative to the causal mechanism (s).

The muscular hypertrophy that has been argued as contributing to lean body weight increases is possibly due to an increase or elevation in the level of serum-human-growth-hormone (HGH) which has been shown to rise with exercise (Sutton, Young, Lazarus, Hickie and Maksvytis, 1968). HGH is considered a protein anabolic hormone, and in animals receiving growth hormone, the increased deposition of protein is accompanied by a loss of carcass fat (Gordon, 1970; Oscai, Babirak, Dubach, McGarr, and Spirakis, 1974). This latter finding could be related to the loss of fat which has been a consistent finding in the studies reviewed in this section.

Body Composition and Coronary Artery Disease

The relationships between coronary artery disease (CAD) and body composition have received considerable review over the past 25 years. This subject has primarily been reviewed indirectly, that is, evaluators have assessed the relationship between high blood pressure and hypertension and elevated body fat levels. Mann (1974) presented a paper reviewing the whole subject of the influence of obesity on health. This author reveiwed the commonplace fattening of our population with age and the dilemma of health scientists that comes from the conspicuous association of obesity with a number of chronic diseases that appear in middle and old age. One such chronic disease, associated with obesity, is coronary artery disease. Mann felt that the health scientists of today have yet to distinguish if obesity is a causal or associated factor in coronary artery disease.

As mentioned, CAD has been associated with overweight subjects via elevated blood pressures. Several authors have commented on the association between blood pressure and obesity of overweight subjects (Mann, 1974; Alexander, 1963; Keys, Aravanis, Blackburn, Van Bucham, Buzina, Djordjevic, Fidanza, Karvonen, Menotti, Puddu, and Taylor, 1972; Heyden, Humes, Bartel, Weinsier, Fuchs, 1971; Weinsier, Cassel, Kay, Triebwasser, and Lancaster, 1976; Kannel, Brand, Skinner, Dawbar, and McNamara, 1967). Statistically, one of the better studies being conducted on this subject has been the Framingham Study by Kannel and his co-workers. This longitudinal study indicated that high blood pressure is developed 10 times more often in subjects 20 percent or more overweight, and men who maintained their weights nearly constant over a 20 year period of adult life had only one-fifth as much high blood pressure in middle life. Even though a relation between these two factors has a correlative basis, the causal relation still defies definition. Alexander (1963) argued that obesity, even when in the extreme, did not lead "invariably" to the development of hypertension. This attitude was also adopted by Spain and his co-workers (1963) when they argued;

> "relative muscle mass rather than an increase in adipose tissue probably has the more direct association with the prevelance of coronary atherosclerotic heart disease"

Spain's point hinges on the direct relationship between these two factors, that is, elevated fat stores and CAD.

Recent studies have been reviewing overweight and obesity as secondary factors in CAD Heyden, in 1971, and his associates conducted a study which indicated a statistically significant and direct relationship between weight change (up and down scale) and both systolic and diastolic blood pressure in middle and older aged males. In contrast, these authors found no such a relationship among the same population when they were twenty years of age. One of the problems studies on this subject have had is the use of relative weight to demonstrate an association between body fat and CAD. Spain, et.al., (1963) discussed that the potential relationship between CAD and weight could be traced to particular components of the body rather than the total weight of the subject and pinpointed either body fat or LBM. The studies completed up till now, have failed to utilize body compositional assessment methods that were sympathetic to these components. One such reason has been due to the sheer volume of their populations. Ancel Keys and his co-workers (1972) developed a prediction equation based on skinfolds which they used to indicate the body fat component of their subject's. Through multi-variate analysis the authors determined a significant relationship between CAD and total body weight (TBW) and their index. This relationship was based on the premise that, as is universally agreed, the risk of developing CAD is directly and causally related to arterial pressure and arterial pressure is related to man's TBW and PBF (Keys, et.al., 1972).

A very specific study was performed by Weinsier and his associates in 1976 on body fat and its relationship to CAD, blood pressure, blood lipids and other risk factors. These researchers studied a large male population and determined the body fat content from the population's total body water, measured by the tritium dilution technic ($N = 1483$ subjects). The mean age of these subjects was 37 years (S.D.= ± 9 years) and they had a mean PBF of 20.6% (S.D.= ± 5.5%). The results of this study indicated several relationships. These authors failed to demonstrate an independant contribution of obesity to CAD when the effect of blood pressure was removed. This result, when further analysed, demonstrated that a statistically significant ($\underline{P}<0.001$) relationship existed between hypertensive (as defined by elevated systolic and diastolic blood pressures) subjects and PBF levels. These findings agreed with those of Mann (1974), Keys, et.al., (1972) and Kennel, (1967). The study further illustrated significant correlations and relationships between blood lipid levels and PBF levels. The implications of this study are that the size of the body's fat component is directly associated with two of the more accepted risk factors of CAD; hypertension (high blood pressure) and elevated blood lipid levels. It should be pointed out though that body fat levels are not the sole determinants of these factors levels but contributory. Furthermore, the direct effect that elevated PBF levels may have on CAD appears to be small or non-existent. Therefore, one can conclude that PBF is contributory to the accepted determinants of CAD, which makes it a secondary or mild contributor to CAD in some cases.

Kannel, LeBauer, Dawbar, and McNamara (1967), in a further presentation of their Framingham Study, offered several possible mechanisms, physiological and metabolic in nature, by which obesity (as defined by elevated body fat levels) could be associated, directly or indirectly, an excess of CAD in a population. They were;

(1) increased cardiac workload and blood pressure resulting from excessive weight;

(2) increase in the amount of coronary atherosclerosis resulting from higher caloric intake and increased lipid levels and blood pressure accompanying gain in weight;

(3) decrease in physical activity with obesity, which may result in deficient development of collateral circulation in subjects with atherosclerotic involvment of the coronary arteries;

(4) combinations of (1) to (3) above.

All of these factors enumerated could produce excessive occurance of myocardial infarction as well as angina pectoris and sudden death.

Notice should be now drawn to the fact that mechanisms (No. 2 and 3) are associated with physical activity. Increased physical activity produces an elevated caloric expenditure which would counter the increased caloric consumption associated with weight gains (Katch, et.al., 1977; Mayer, 1963). Secondly, physical activity would affect the collateral circulation development argued in mechanism (3). The point being, physical activity, as discussed in the previous section, is a weight controlling mechanism which can decrease the amount of body fat and therefore alleviate some of the contributory mechanisms associated with CAD risk factors and indirectly CAD itself. A review of the literature does not produce any evidence that would lead one to conclude that the weight loss and gain mechanisms of coronary artery diseased subjects would differ in any way from those of normal and healthy subjects. This lack of evidence leads one to hypothesize that these subjects would, therefore, react to physical activity in a fashion similar to normals.

CHAPTER III

METHODS

The purpose of this chapter is to assist reviewers in the evaluation and, possibly, the replication of results.

Subject Selection

All subjects selected for this study were volunteers from either the Adult Fitness Unit of the La Crosse Exercise Program (L.E.P.), the Cardiac Rehabilitation Unit of the L.E.P., the University of Wisconsin - La Crosse academic staff, or the City of La Crosse community. Sixty-eight males, aged 35 to 73 yrs. ($\bar{X}$=48.6, S.D.= ± 10 yrs) were evaluated. They had been informed of the possible dangers involved in participation and had signed an informed consent from (Appendix E).

All testing was conducted in the University of Wisconsin - La Crosse Human Performance Laboratory over a six week period. The subjects were divided into three groups according to activity and/or disease status. Thirty subjects were classified as physically active, 20 as inactive and sedentary, and 18 as active and clinically diagnosed as being cardiacs patients.

The physically active males had been participants in the L.E.P. Adult Fitness Unit for a minimum of six months. Their activity regimen consisted of running for a minimum of 45 minutes on three or more days a week (specifically, Monday, Wednesday and Friday) at an intensity of 65% to 85% of their maximum heart rate. This group's ages ranged from 35 to 62 years ($\bar{X}$=46.17, ±6.81 yrs.).

The inactive or sedentary group was enlisted from the University of

Wisconsin - La Crosse academic staff and the City of La Crosse community. These subjects participated in no regular physical activity and had ages ranging from 35 to 62 years ($\bar{X} = 43.9$, ±7.12 yrs.).

The active clinically diagnosed cardiacs were participants in the L.E.P. Cardiac Rehabilitation Unit track-advanced section. They too had been participating in regular physical activity (as defined) for a minimum of 6 months. These men were clinically diagnosed as having coronary artery disease (CAD) by the criteria of having had a positive Graded Exercise Test, having had a positive cine-angiographic demonstration of occlusive disease, or having had a documented myocardial infarction. This group had an age range of 35 to 73 years ($\bar{X} = 57.9$, ± 11.44 yrs.).

Each subject's personal physician was informed of their patients participation.

Experimental Treatment

On the day of the test, the subjects followed their normal activity pattern. No food intake had occurred for a period of 12 hours before testing so as to create a post-absorptive state in the subjects.

Upon reporting to the laboratory the subjects signed an informed consent form. A nude weight was then obtained, followed by a shower with a neutral soap. During the shower, emphasis was placed on the hydrating of the body hair and the subject's swimming costume. The subjects then reported to the densitometric tank. They were outfitted with a weighted diving belt (approximately 10 pounds) and a nose clip. The subjects then entered the tank and seated themselves on a suspended chair, with the water level being chin height. Subjects were given preliminary instructions and, if needed, a complete demonstration of the underwater technique by the investigator. Underwater weight measurements were taken too the nearest 25 grams, following a maximal expiration and submersion by the subject. Water temperature was taken after each of the nine trials performed. Upon completion of the underwater assessment the subjects re-showered and dressed.

Once dressed, the subjects reported for residual lung volume (RV) assessment. They were outfitted with a nose clip and performed an oxygen rebreathing maneuver. These maneuvers allowed the calculation of residual lung volume. The R.V. assessment was performed twice. This completed the study's assessment design.

Development of Instrumentation

Instrumentation and methodology for the determination of body weight, body volume, residual volume, body density, PBF, and lean body mass are explained in detail.

Body Weight

Subjects were weighed nude on a bean balance scale (Health-O-Meter, Continental Scale Corporation) to the nearest 0.25 pounds. The scale had been previously calibrated.

Body Volume

As will be explained in a later section, determination of the net body volume permits an estimate of the density of the whole body. Several approaches that have yielded reliable data are hydrostatic weighing, water displacement, hydrometry, air displacement, and helium gas dilution (Gnaedinger, Reineke, Pearson, Van Huss, Wessel, and Montoye, 1963). The method of hydrostatic weighing (commonly referred to as underwater weighing) has enjoyed considerable usage due to ease of methodology, reliability, validity, and it's relatively inexpensive equipment requirements (Ward, et.al., 1978; Cureton, et.al., 1975; Gnaedinger, et.al., 1963; Goldman and Buskirk, 1961; Shepard, Jones, Ishii, Keneko and Olbrecht, 1969). Therefore, due to the reasons of reliability and validity of the method (Keys, et.al., 1953(and the ease with which large samples can be assessed efficiently with such a method (Shepard, et.al., 1969), underwater weighing was utilized in this study.

The Principles

Archimedes stated that when submerged in water, an object displaces a volume of water equal to the volume of the object itself. Therefore, to ascertain the volume of the body, calculation of the volume of water displaced by the body was performed. This was accomplished by utilizing the formula:

$$\text{Volume of Water} = \frac{\text{Mass of Water}}{\text{Density of Water}} \qquad (3)$$

The density of water is calculable from the temperature of the water for there exists a nonlinear, but defineable, relationship between water temperature and water density (Weast, 1967).

The mass of water was calculable by the usage of Archimedes Principle. His principle states that a body immersed in a fluid is acted upon by a buoyancy force, made evident by a loss of weight, equal to the weight of the fluid displaced. In essence, the difference between the weight of the body in air (MA) and the weight of the body when completely submerged in water (MW) is the weight of the displaced water:

$$\text{Mass of Water} = MA - MW \qquad (4)$$

Therefore, the total external volume of the body is represented by the equation:

$$\text{External Body Volume} = \frac{MA - MW}{DW} \qquad (5)$$

Two other volumes contribute to the volume actually measured and have been subtracted from the total body volume; these are, (1) the volume of air in the lungs after a maximal expiration (R.V.) and (2) the total volume of gas bubbles in the gastrointestinal tract. The former is a relatively large space and will be discussed in detail in the following section. The latter, represents a volume which is susceptable to noticeable variation. On the basis of the findings of Bedell, Marshall, DeBois and Harris, (1956), a proposed correction of 100 milliliters is offered to allow more valid assessment of the body volume being calculated. Thus the equation becomes:

$$\text{Body Volume} = \frac{(MA - MW)}{DW} - \text{R.V.} - 100 \text{ ml.} \qquad (6)$$

The Procedure

Each subject's underwater weight was measured in a 4 x 4 x 4 ft. stainless steel hydrostatic weighing tank. After being outfitted with a lead weighted diving belt (with a dry weight of 10 lbs.) around their waist, the subjects descended into the tank by means of a ladder. They then assumed a standing position and removed the bubbles from their body and from inside their swimming garment. The subjects then seated themselves on a plastic tubular chair. The chair was suspended from a 15 kg. Chatillon cadaver which was attached to a ceiling anchorage point.

The depth to which the subjects were seated was adjusted so that the water level was just below the subject's chin. This position offered two advantages. First, the subject was in complete control of the submerging, thus eliminating fear of the procedure. A second advantage of this position was that the greater proportion of the subject's body was already immersed and the bending forward, to attain complete submersion, was done with only a small degree of motion and with only slight disturbance of the water, minimizing the oscillation that can occur on the scale.

In this seated position, the subjects were instructed to perform, as precisely as possible, the following physical and respiratory maneuvers.

The subject was outfitted with a noseclip which eliminated ventilation through the nasal passages. They were then instructed to perform a deep inspiration followed by a "long and slow" expiration. The subject was to continue this expiratory maneuver until he felt that no more air could be expelled. At this point, the subject shut his mouth and slowly bent forward, through flexion at the waist and the neck, so as to totally submerge the body. Prior to submersion, the subject was instructed to try and expel any more air that may be left in his mouth or lungs after submersion. This final respiratory maneuver provided the effect of acting as a signal to the assessor that he had attained maximal expiration. Once the subject had signalled the assessor with a final "shot" of air, or had attained a steady state underwater, the scale was read for their underwater weight. This reading or figure was measured to the nearest 25 grams.

After the underwater weight was measured, the assessor submerged his hand and tapped the subject upon the head, so as to signal the end of the measurement trial. Following each measurement trial, the water temperature was assessed and recorded in degrees Celsius (°C) by a standard thermometer with 0.1 °C graduations

The number of trials performed was nine in accord with the work of Katch (1967). This author demonstrated that there exists a learning response to the maneuvers performed. He showed that with increased practice, the subjects become more accustomed to expelling air and more consistent in the volume of air expelled, thus increasing their apparent weight underwater. The author, therefore, concluded that, with inexperienced subjects, 9 to 10 trials should be performed to obtain a stable and true maximal underwater weight.

Once all nine trials had been performed, the subject's underwater weight was selected by set criteria. Thus, (1) the subject's heaviest underwater weight was selected if it occurred or appeared more than once. Failing to meet this requirement, (2) the second heaviest underwater weight was selected if it appeared more than once. And, failing to meet the first two criteria, (3) the mean of the final three recordings was calculated and utilized.

The resulting data allowed for the calculation of the body's volume, corrected for certain internal volumes. The most prominent of these volumes is that of residual volume. The assessment of this parameter will now be discussed.

Residual Volume

As mentioned in the previous section, the volume of air in the lungs at the end of a maximal expiration was represented in the total body volume calculation. Therefore, this lung volume had to be calculated and the body volume corrected.

Residual volume cannot be directly assessed through conventional spirometric analysis. Consequently, the residual volume measurement was accomplished through an indirect analysis method. The majority of indirect methods have been classified into three major approaches: (1) the pneumatometric approach, using some form of whole body plethsmograph; (2) the closed circuit approach, where there is a dilution and eventual equilibration of an inert tracer or indicator gas such as nitrogen, helium, or hydrogen; and (3) the open - circuit approach, where nitrogen is "flushed out" of the lungs during a specified period of oxygen breathing. All of these approaches have been extensively reviewed (Christie, 1932; Lassen, Cournand and Richards, 1937; Cournand, Darling, Mansfield and Richards, 1940; Wolfe and Carlson, 1950; Motley, 1957; Wilmore, 1969) and appropriately critiqued. The pneumatometric approach has been opposed from the view of disproportionate expense and intrinsic methodological errors (this approach rarely appears in the literature). The open- and closed - circuit methods of R.V. assessment have shown to be the most commonly utilized approaches. These approaches have been shown to have intrinsic errors and have been reviewed considerably (Christie, 1932; Wilmore, 1969; Wolfe, et.al., 1950). A discussion of these errors will be discussed in a later section on analysis of error in measurement.

The various methods of nitrogen washout, helium dilution, and hydrogen rebreathing are all regarded as valid and reliable but they all have the common limitation of requiring considerable time to complete a single determination for one subject. Therefore, the method adopted in this study was a slight variation on the modified closed - circuit oxygen dilution method developed and reported by Wilmore (1969).

The variation consisted of the following. A Collins 6.0 liter respirometer was altered to minimize the effective dead space. This alteration was accomplished by removing the length of tubing connecting the breathing valve to the respirometer. The two-way breathing valve, and the attached nitrogen analysing head, was connected directly to the inlet-outlet orifice of the respirometer. This effectively reduced the effective dead space from 2.35 to 1.46 liters, as calculated by the dilution technique of Christie (1932) and substantiated by geometrical estimation. By the reduction of the effective dead space of the respirometer a faster oxygen flushing (clearance of nitrogen from the system) period was accomplished along with increased accuracy of the measurements, due to a decreased total volume of the system. Further modifications consisted of a single oxygen inlet line was placed up through the respirometer water resevoir and was attached above the water level.

Continuous analysis of inspired and expired nitrogen concentrations (in the form of percentages) were assessed on a Collins Nitrogen Analyser (Model No. 21232). The remote analysing head (an ionization chamber) of the Nitrogen (N_2) analyser was placed at the distal end of the respirometer system, between the two-way breathing valve and the subject's mouthpiece. The resolution of this system was ± 0.1% N_2 with a readout accuracy of ±0.2% N_2 and a response time of 15 milliseconds to within 90% of final value. These specifications allowed a determination of ± 10 ml. of the "true" residual volume.

Prior to the testing of each subject, the water level of the respirometer was checked and, if necessary, filled with clean water to a predetermined level so that a constant dead space was maintained. The system was then flushed with Oxygen (99.95% 02) so that the respirometer, tubing, and breathing valve was free of nitrogen. Once cleared of N_2, exactly 4.54 liters of Oxygen was introduced into the bell of the system which, when combined with the 1.46 liters of Oxygen in the dead space, gave a total effective respirometer volume of 6.000 liters of Oxygen. This volume of 6.00 liters facilitated subsequent calculations of residual volume and represented the upper limits of the normal range of vital capacity in middle and older aged males (Hurtado and Boller, 1933; Brozek, 1960; Berglund, Birath, Bjure, Grimby, Kjellmer, Sanqvist and Soderholm, 1963; Kaltreider, Fray and Van Zile Hyde, 1933; Morris, Koski and Johnson, 1971).

All assessments were conducted in the sitting position (which closely approximated the subject's position adopted during the underwater weighing assessment). The subject was informed of the intent and procedure of the assessment. A noseclip was then attached to the subject's nose and the mouthpiece was comfortably positioned in his mouth. After 5-6 normal ventilations, with the breathing valve turned to the room air, the subject was instructed to inhale deeply and then to exhale as fully as possible, and he was asked to indicate to the investigator (by a tap on the back of the investigator's hand) that point when a maximal exhilation had been attained. At this point the two-way breathing valve was pushed in to connect the subject with the respirometry system, and the nitrogen reading at the end point of this expiration was recorded and assumed to be representative of the initial alveolar nitrogen concentration (AiN_2). The subject was then instructed to inhale very slowly on the initial intake of Oxygen from the respirometer. At the end of this slow inhalation the N2 reading was recorded and assumed to represent the concentration of N2 initially in the Oxygen resevoir (that is, the impurity). Following this initial inhalation the subject then proceeded to expire and inspire at approximately two-thirds his vital capacity at a rate of one respiration every 2-3 seconds.

Rebreathing continued until an equilibrium state was attained between the lungs and the respirometer system. A visual observation of the continuously decreasing oscillations in the nitrogen percentage of the inspired and expired air allowed a fairly precise estimation of the establishment of a concentration equilibrium. When equilibrium was attained, usually after 7-10 breaths, the equilibrium range[1] was recorded. The subject was then told to again take a deep inhalation and follow this with a maximal exhalation. At the end of the maximal expiration the breathing valve was turned back to room air. The subject then removed the mouthpiece and the respirometer was emptied and the nitrogen reading during this emptying was recorded and assumed to represent the final alveolar nitrogen concentration (AfN_2). This completed the initial residual volume assessment. After two minutes of normal ventilation the whole procedure was repeated. If the residual values calculated from both assessments exceeded each other by greater than 100 ml., the procedure was repeated a third time and a mean of the three recordings was assumed to represent the subject's residual volume.

From the data obtained from this method, residual volume was calculated by the following formula, which is a modification of the formula developed by Lassen, Cournand, and Richards, (1937).

$$R.V. = \frac{VO_2\ (EN_2 - IN_2)}{(AiN_2 - AfN_2)} - D.S. \qquad (7)$$

---

[1] In the majority of cases a single equilibrium concentration was not attainable, in this regard a constant equilibrium range was assumed to represent the attainment of an equilibrium state. The single equilibrium concentration (EN_2) was calculated by establishing the mid-point between the equilibrium range recorded.

where

R.V. - Residual Volume

VO_2 - Initial Volume of Oxygen of respirometer including dead space between the breathing valve and the respirometer bell (6.0 liters total in the present study).

EN_2 - Percentage of nitrogen at which point equilibrium occurred.

IN_2 - Percentage of nitrogen initially in VO_2 (impurity).

AiN_2 - Percentage nitrogen in alveolar air initially when breathing room air.

AfN_2 - Percentage of nitrogen in alveolar air at termination of the test (this closely resembles EN_2).

D.S. - Dead space of the mouthpiece, senseing element of nitrogen analyser, and a small portion of the breathing valve (in this study, this figure was a constant 0.35 ml.).

The methods and instrumentation utilized to calculate the subject's body densities will now be discussed.

Body Density Determination

Utilizing the parameters of body weight, body volume, residual volume, and estimated gas volume in the gastro-intestinal tract, the assessor was able to calculate the density of the subject's body.

Density is defined as the concentration of matter measured as the mass per unit volume (Goldman, et.al., 1961). Therefore the equation,

$$\text{Density of Body} = \frac{\text{Mass of Body}}{\text{Volume of Body}} \qquad (8)$$

The determination of the subject's mass was accomplished by weighing the subject in air, as discussed in a previous section. The volume of the subject's body was determined by the use of the underwater weighing method previously discussed. This volume was corrected for by calculating the subject's residual volume and estimating the volume in the subject's gastro-intestinal tract.

The equation for body density (DB) was then:

$$DB = \frac{MA}{\frac{(MA - MW)}{DW} - R.V. - 100\ ml.} \qquad (9)$$

where

DB = Density of the Body

MA = Weight of the Body in Air

MW = Weight of the Body in Water

R.V. = Residual Volume

100ml = Estimated Gas Volume in the gastro-intestinal tract.
(Bedell, et.al., 1956)

The determination of the body density permitted the in vivo measurement of the body composition of that respective subject. Specifically, the determination of body density permitted the calculation of the relative percentages of the two basic components of the body; body fat and lean body tissue. A description of the mathematical calculation of these components will now be the center of discussion.

The Calculation of Relative Body Fat from Body Density

Historically, the assessors of body composition have utilized a two-component model to describe body status (Spivak, 1915; Rathbun and Pace, 1945; Body, 1933). Rathbun, et.al., (1945) which considered the body to be made up of a nonfat portion and pure fat. The fat component has long since benn viewed as a labile component that is not specifically pure. It is now viewed as an either extractable substance that is metabolically labile. The nonfat or fat-free component of the body is generally referred to as the Lean Body Mass (LBM); this should not be confused with the fat-free body (FFB). LBM is an in vivo concept which is based on the understanding that the constients of LBM remain constant in their composition throughout life

(Malina, 1969). FFB, in contrast, is an in vitro concept attainable only in the sterile laboratory environment (Malina, 1969).

Archimedes illustrated the fact that a mixture of two components, A and B, of differing densities, d_1 and d_2, the gross density, D, is determined by the proportional masses of A and B in the system.

$$D \quad \frac{A \quad B}{(A/d_1) \quad (B/d_2)} \qquad (10)$$

Therefore, with a knowledge of the gross density of the body (D_B) and an equal knowledge of the individual densities of the fat and LBM components, d_{fat} and d_{LBM} respectively, calculation of the proportional masses of the two components is possible. These calculations are performed by using the mathematical sentences of

$$PBF \quad \frac{1}{D_B} \times \frac{d_{fat} \times d_{LBM}}{(d_{LBM} - d_{fat})} - \frac{d_{fat}}{(d_{LBM} - d_{fat})} \times 100 \qquad (11)$$

(Keys, et.al., 1953)

for PBF and......

$$\%LBM \quad \frac{1}{D_B} \times \frac{d_{LBM} \times d_{fat}}{(d_{fat} - d_{LBM})} - \frac{d_{LBM}}{(d_{fat} - d_{LBM})} \times 100 \qquad (12)$$

for % LBM.

The validity of the calculation of PBF depends upon the density values of fat and LBM. These variables, as entities, have received considerable attention and debate over the past thirty years (Rathbun, et.al., 1945; Brozek, 1952; Forbes, 1952; Keys, et.al., 1953; Fidanza, Keys, Anderson, 1953; Siri, 1956; Brozek, et.al., 1963; Malina, 1969). Historically, the "reference man", derived by direct measurements of body density in a young group of men by Brozek, (1952), was the standard of reference for the densitometric determination of body fat. Brozek, calculated an average body density of 1.063 for the group and assumed that the young men had a PBF of

14%. Utilizing the findings of Fidanza, et.al., (1953) that the density of pure body fat is 0.9007 gm./cc., Keys and Brozek (1953) suggested the formula,

$$PBF = \frac{420.1}{DB} - 381.3$$

Since the value of 14% was hypothetically chosen there required an empirically determined reference body for which the fat content was known and for which the density could be calculated on the basis of its constituents. Brozek, et.al., (1963) presented the results of three reports on the direct chemical analysis of three male cadavers. The resultant information, when averaged and assessed, was used to "construct" an empirically defined "reference body" with a density of 1.064 gm./cc. and a 15.3% body fat. These researchers further pointed out that "obesity tissue" (which is described as a combination of extracellular fluid, fat, and "cell residue") can be defined as either weight gain, weight loss, or as a static weight difference. Depending on the definition of obesity tissue, there exist different ratios of its constituents to its whole. This definitional problem resulted in different densities for obesity tissue depending upon the definition applied. Therefore the authors felt that utilizing the definition of obesity as a relatively static weight difference it was possible to establish a formula which was applicable to the estimation of the fat content in individuals whose body weight had been free from large recent fluctuations, either up or down. They determined that

$$PBF = \frac{457}{DB} - 414.2$$

Siri (1956) disagreed with Keys, et.al., (1953 and 1963) as to the applicability of equations based on the "reference body" system. Siri felt that using a fat-free body as a reference would better reflect the consistancy comparability of tissue to populations. In contrast, Keys, et. al., (1963) argued that a fat-free body possesses some "essential" fat and the densities of the fat and lean body mass components will vary without recognition. Siri felt that this line of argument was unsubstantiated and developed his own formula using the fat-free body as the correct reference (Lean Body Mass Density = 1.1 gm./cc. and Fat Density = 0.90 gm./cc.)

$$PBF = \frac{495}{DB} - 450$$

In summary, it is worthy noting Brozek, et.al., (1963) final comments

> "It appears no universally valid formulas for densitometric estimation of the body's fat content can be offered at present".

Sources of Error in the Calculation of Relative Body Fat

Densitometric analysis, utilizing the hydrostatic weighing method, possesses intrinsic sources of error. These sources can be eliminated (to a degree) through body recognition and refinement of methods. Discussion of these sources will now be dealt with. The methods utilized in assessment were selected on the basis of their accuracy.

Density of Water

The density of water has a calculable relationship with temperature. Buskirk, (1961) reported that the water in the hydrostatic weighing tank should be maintained at a temperature comparable to the mean body temperature values, that is, 35 to 37 °C. (95-98.5 degrees F.). The density of water at this temperature is 0.994 gm./cc. Buskirk further discussed the use of a "reasonable" temperature range, more specifically, he is quoted as saying; "The water bath should not be so hot or so cold as to induce changes in mean body temperature". Furthermore, question has been raised as to the variations induced by using distilled water rather than tap water. The practical importance of such a question has been dismissed due to the minute variations that the different densities induce. The density of distilled water at 35 °C. is 0.99406 gm./cc. as compared to tap water at 35 °C., 0.99411 gm./cc. This variation alters the body density value in the 5th and, sometimes, the 4th decimal places only which is not significant.

The Mass of the Body in Air and Water

The accuracy to which these weights are taken extremely important. Several authors have suggested the calculation of both air and underwater weights to the nearest 25 grams (Wilmore, et.al., 1969; Katch, 1968; Pollock, et.al., 1975; Buskirk, 1961). In obtaining an underwater weight, with the suspension system, the accuracy should be ± 25 grams, this includes the dampening of the scale which occurs in the methodology (Buskirk, 1961). The mathematical changes in density with changes in assessed underwater weight are demonstrated in Table 4.

Residual Volume

The variation in the calculated value of body density induced by differing values of residual volume are demonstrated in Table 5. Several studies have analysed the advantages (or disadvantages) of calculating residual volume by helium or oxygen closed circuit rebreathing methods,

TABLE 4

CHANGE IN DENSITY WITH CHANGE IN WEIGHT UNDER WATER

Underwater Weight Change (Kg)	Change in Density Units (gm/cc)
0.10	0.002
0.20	0.004
0.50	0.010
0.70	0.015
1.00	0.022
1.50	0.034
2.00	0.046

When: weight in air, water density, and residual volume are held constant.

(Zavaleta, 1976).

TABLE 5

CHANGE IN DENSITY WITH CHANGE IN RESIDUAL VOLUME

Residual Volume Change (ml)	Change in Density Units (gm/cc)
25	0.0004
50	0.0008
100	0.0016
150	0.0024
200	0.0032
250	0.0040
300	0.0048

When: weight in air, weight underwater, and density of water are held constant.

or the nitrogen washout open circuit method and the application of their results to the calculation of relative body fat (Wilmore, 1969; Girandola, Wizwell, Mohler, Romero and Barnes, 1977; Wilmore, 1969; Zuti, et.al., 1973; Katch, et.al., 1967; Minh, Dolan, Linaweaver, Friedman, Konopka, and Brach, 1977; Bondi, Young, Bennet and Bradley, 1976; Prefaut, Lupi-H and Anthonisen, 1976; Motley, 1957). In 1969, Wilmore reviewed a large number of studies as to the accuracy and applicability of their results. He reported that the standard error of measurement is approximately ± 100 ml. in all methods. He raised the question as to the applicability of actual, predicted, and constant residual volume values in the assessment of relative body fat. Specifically, Wilmore demonstrated that there existed no statistically significant difference, at the 0.05 level, between the means for density, percent body fat, and lean body mass calculated using the actual residual volume and the means calculated using either the estimated or constant residual volumes. He also demonstrated a high correlation, indicating a substantial relationship between these three calculated residual volumes. The author further explained that these findings were surprising and that they only reflected generalized means, for there existed a large percentage of subjects whose values differed sufficiently enough to seriously question the practice of using either an estimated or constant residual volume value. In conclusion, the author argued that the selection of a predicted or constant residual volume value will not influence the relative values of density and percent fat, they will, however, greatly influence the absolute values of such parameters. Thus, when obsolute accuracy is required it is necessary to directly measure residual volume.

Of recent concern, is the time and place during which residual volume is to be measured. Several authors have analysed the effects of water immersion on man's residual volume. Some investigators have found decreases with immersion (Bondi, et.al., 1976; Brozek, Henschel and Keys, 1949; Jarrett, 1965) and others have found no significant changes in the residual volume with immersion (Carey, Schaeffer and Alvia, 1956; Craig and Ware, 1967; Prefaut, et.al., 1976). Recently, Girandola, et.al., (1977) studied the effects of water immersion on the lung volumes in man and their implications for body compositional analysis. Experimentally, these authors, compared the residual volume values measured in and out of the water. They demonstrated a 6.7% increase in residual volume when measured in the water. The implication of this finding to body compositional analysis demonstrated a mean decrease of 0.6% body fat if the measurement was taken in the water, in contrast to outside the water residual volume measurement. This "difference" was conceded by the authors as not being "physiologically" significant. Furthermore, this study's methodology can be criticized for the authors measured residual volume in the standing position and the density assessment and calculation in the seated position. Brozek, et.al., (1949) demonstrated significant variations in residual volumes with alterations in posture.

In conclusion, the variations demonstratedare really beyound the specified accuracies of the methods used (that is, ± 100 ml.). Wilmore (1969) agreed with Buskirk (1961) that ± 100 ml. is an acceptable standard deviation for the residual volumes assessment methodologies presently used. The present author felt that this experimental standard deviation took into consideration and covered individual biological variability for the questions as to what variation is produced by biology, and what by technique, still remains unswered.

Other Errors

In addition to the error sources thus far discussed, gas bubbles commonly adhere to the skin or are trapped in the scalp and hair of the body. It should be noted that these bubbles can usually be wiped off by the subject. Notably, the combined effect of these bubbles probably would never exceed 10 ml., even if no attempts were made to remove the dissolved air from the weighing tank. Therefore, the effect upon the calculated body density would be in the range of ± 0.00018 gm./cc.

The value of gas stored in the gastro-intestinal tract has already been discussed in the section on the principle of body volume measurement.

Statistical Treatment of Data

The reliability and reproducibility of the body composition and residual volume methods utilized in this study was assessed by the development of test-retest relaibility coefficients on all the variables on a seperate experimental sample group ($N=15$, $\bar{X}$ age = 28.2 years, ± 6.7 years) using the Pearson-Product Moment correlation.

The experimentally collected data from the samples were statistically described by calculating the means ($\bar{X}$) and standard deviations (S.D.) for all the variables investigated.

An analysis of variance (ANOVA) among group means was then conducted to determine the differences, if any, among the groups means for age, PBF, residual volume, body density, lean body weight, fat weight, and body weight. A Scheffe Post-Hoc test was then conducted, if appropriate, to locate any significant paired mean differences between the groups. The alpha level was set at 0.05 level.

CHAPTER IV

RESULTS AND DISCUSSIONS

The purpose of the present study was to densitometrically analyze, describe, and compare the body composition of middle- and older-aged sedentary, active, and clinically diagnosed cardiac (CAD) males. The density (BD), lean body weight (LBW), and percent body fat (PBF) of all the subjects were calculated for the total population and the three subgroups and compared directly to age. Specific concern was given to the trends elicited by BD/LBW, and PBF when compared to age, with special reference to the subgroup's physical activity and pathological status.

In this chapter the results and discussion have been integrated in order to prevent duplication and to provide a degree of continuity in information presentation. The first part of this chapter deals with the characterictics of the population (and its respective subgroups). The second section addresses itself to the reliability of the methods of this researcher, and the equipment used to assess the variables researched in this study. In the third portion of this chapter, all the subjects have been considered as one total group; therefore, their results are presented and discussed with such consideration. Finally, the fourth part of this chapter deals with the results of the subgroups involved in this study.

Subjects

Of the 90 subjects originally randomly selected only 68 agreed to participate in the study due to time conflicts. All the subjects were male, between the ages of 35 and 72 years ($\bar{X}$ = 48.6 years, S.D. = ± 10 years) and resided in the City of LaCrosse or the surrounding area. The total group was divided into three sub-groups; active, inactive/sedentary, and active clinically diagnosed cardiacs. The active population ($N=30$, $\bar{X}$ age = 46.2 years, S.D. = ±6.2 years) was a randomly selected sample from the LaCrosse Exercise Program's Adult Fitness Unit. The sedentary population ($N=20$, $\bar{X}$ age = 43.9 years, S.D. = ±7.1 years) was randomly selected sample from the University of Wisconsin-LaCrosse academic staff and from the City of LaCrosse community. The third sub-group, the clinically diagnosed active cardiac patients ($N = 18$, $\bar{X}$ age = 57.9 years, S.D. = 11.4 years) was a randomly selected sample from the Cardiac Rehabilitation Unit of the LaCrosse Exercise Program. The subjects were not allowed to participate in this study until the approval of their primary attending physician was given (Appendeces/B & C). Table 6 presents mean subject characteristics of the individual sub-groups and the group as a whole.

A one-way Anova was conducted on each of the characteristics of age, height, and weight, so as to ascertain any physical differences between the sub-groups (Table 7.). The sub-group's ages demonstrated significant differences ($\underline{P} < 0.01$). A Scheffe Post-Hoc analysis isolated the differences: (a) a significant difference ($\underline{P} < 0.01$) between the mean ages of the CAD sub-group and the Active sub-group, (b) significant difference ($\underline{P} < 0.01$) between the mean age of the CAD sub-group and the sedentary sub-group. Table 6 further presents the means and standard deviations of height and weight for the three sub-groups analyzed. The mean height of the CAD sub-group was 4.2 cm. shorter than the active sub-group. The mean weights of all the three sub-groups were within 0.55 kg. of each other. The CAD sub-group was marginally heavier than the sedentary sub-group and was not different from the active sub-group. These height and weight differences were not statistically significant ($\underline{P} > 0.05$).

Table 6

Characteristics of the Subjects in Their Respective Sub-Groups and as a Combined Group

	Active (N=30)	Sedentary (N=20)	Cardiac (N=18)	Combined (N=68)
Age (yrs.)	46.2 ±6.8	43.9 ±7.1	57.9 ±11.4	48.6 ±10.0
Height (cm.)	177.3 ±5.6	176.5 ±5.4	173.2 ± 5.9	176.1 ± 5.9
Weight (kg.)	79.6 ±9.4	79.09 ±10.9	79.6 ±11.5	79.5 ±10.4

Values are Means ± Standard Deviations

Table 7

Statistical Analysis
of Subgroup's Mean Ages

(A) Analysis of Variance

Grand Total = 3305*
Number of observations = 68
Total Group Mean = 48.6029

Source of Variation	Degrees of Freedom	Sum-of-Squares	Mean-Square	F - Ratio**
Treatments	2	2172.53	1086.3	15.5738
Error	65	4533.72	69.75	
Total	67	6706.25		

(B) Scheffe Post-Hoc Analysis

1. Between the Normal Active Subgroup and the Normal Sedentary Subgroup; Scheffe F - ratio = 0.8865.
2. Between the Normal Active Subgroup and the Clinically Diagnosed Cardiac Subgroup; Scheffe F - ratio = 22.19 ($P < 0.01$).**
3. Between the Normal Sedentary Subgroup and the Clinically Diagnosed Cardiac Subgroup; Scheffe F - ratio = 26.62 ($P < 0.01$).**

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*Total Summation of Subgroup's Ages.

**An F ratio value of 4.95 is needed for significance at the 1 percent level.

Reliability of Methodologies

A pilot study evaluating the reliability of the methods utilized was performed prior to the collection of data for this study. Two assessments of total body weight (TBW), residual volume (RV), and percent body fat (PBF) were performed on 14 active males and females ($\bar{X}$ age = 28.2 years, S.D. = ±6.17 yrs.) who were from the City of LaCrosse area. The assessments were conducted within 48 hrs. of each other.

Test-retest reliability correlation coefficients were calculated by means of Pearson-Product Moment correlation techniques. The test-retest correlations on the 14 subjects for TBW, RV, and PBF were 0.9989 ($\underline{P}<0.01$), 0.9269 ($\underline{P}<0.01$), and 0.9801 ($\underline{P}<0.01$) respectively. In addition, the mean PBF, TBW, and RV values were not significantly different between repeated measurements ($\underline{P}<0.01$). A summary of the statistical results of this pilot study is presented in Table 8.

Table 9 lists the means and standard deviations of the total group's (N=68) ages, residual volumes, and body densities. The total group's mean age ($\bar{X}$ age = 48.6 years, S.D. = ±10.0 years) was very comparable to the population studied by Pollock, et.al., 1976 ($\bar{X}$ age = 44.9 years, S.D. = ±4.8 years, N=84), Pollock, et.al., 1972 (N=22, $\bar{X}$ age = 39 years), Pollock, et.al., 1975 (N = 26, $\bar{X}$ age = 38 years), Lewis, et.al., 1975 (N=45, $\bar{X}$ age = 47.2 years, S.D. = ±8.6 years), Boileau, et.al., 1973 (N=21, $\bar{X}$ age = 37.7 years, S.D. = ±7.9 years), Misner, et.al., 1974 (N=24, $\bar{X}$ age = 38.3 years), Pollock, et.al., 1974 (N = 24, $\bar{X}$ age = 54.9 years), Zuti, et.al., 1973 (N=60, $\bar{X}$ age = 36.8 years, S.D. = ±7.96 years), and Brozek, et.al., 1951 (N=122, $\bar{X}$ age = 49.0 years, S.D. = ±2.8 years). The comparability of the ages of these populations

Table 8

Statistical Results of Pilot Study

Designed to Assess Reliability of Data Collection Methods

		Means	SD**	Pearsons correlation coefficient ("r")	students t-test value.
Weight (kgm.)	Test 1.	68.254	12.65	0.9989***	-1.12481****
	Test 2.	68.444	12.91		
Res Vol (l.)	Test 1.	1.1372	0.205	0.9269***	-1.13711****
	Test 2.	1.1623	0.227		
PBF* (%)	Test 1.	16.18	4.96	0.9801***	-0.93889****
	Test 2.	16.47	5.57		

*PBF calculated by Brozek, et.al., (1963) equation; PBF = 457/DB-414.2.

**Values are ± Standard Deviations.

***At the 0.05 level of significance and with 13 degrees of freedom a "r" value of 0.5139 is required for significance.

****At the 0.05 level of significance and with 14 degrees of freedom a t-test value of 2.145 is required for significance.

Table 9

Mean Body Densities and Residual Volumes

of the Sub-Groups and Combined Group

	Active (N=30)	Sedentary (N=20)	Cardiac (N=18)	Combined (N=68)
Age (yrs.)	46.1 ±6.8	43.9 ±7.1	57.9 ±11.4	48.6 ±10.0
Residual Volume (L.)	1.557 ±0.343	1.419 ±0.439	1.845 ±0.431	1.60 ±0.42
Body Density (gm/cc)	1.0499 ±0.0102	.0424 ±0.0143	1.0386 ±0.1499	1.0446 ±0.014

Values are Means ± Standard Deviations.

will become important in the discussion to follow on the relationship between age and body composition.

The residual volumes of the total group ($\bar{X}$ R.V. = 1.6 liters, S.D = ±0.42 liters) demonstrated a linear increase with age ($r = 0.782$, $P < 0.001$). This is in agreement with the findings of Kaltreider, et. al., (1933), Brozek, et.al., (1960), and Hurtado, et.al., (1933). The role R.V. play in affecting the body density (B.D.) assessment has been previously discussed in the methods chapter and described in Table 5. Since the equations utilized corrected for theR.V. of the subjects, the R.V.'s were not a determining factor of the resultant body compositional data but were regarded as contributory. Furthermore, the potential errors induced by the subjects not maximally expiring were considered insignificant because of the practice received. As such this investigator felt that no subject failed to expire maximally to their recorded R.V. value. If, as argued by Bondi, et.al., (1976), Brozek, et.al., (1949), and Jarrett, et. al., (1965) that R.V. determined underwater is less than the R.V. assessment in room air is correct it can be counter-argued that the potential errors in PBF calculation induced by such a decrease are cross-sectional and increase only the relativity of the measurement. The effect of alterations in relativity would not affect the statistics or the trends reflected by the present results. Furthermore, these potential alterations in R.V. values (and their resultant effect) have been countered by the findings of Cory, et.al., (1956), Craig, et.al., (1967), and Prefaut, et.al., (1976) who's studies demonstrated no statistical changes in R.V. in or out of the densitometry tank.

The determined total group mean body density of 1.0446 gm/cc with a standard deviation of 0.014 gm/cc compares well with the group mean body densities reported by other researchers on populations of comparable age; Myhre, et.al., 1966 ($\bar{X}$ density = 1.0466 gm/cc, S.D. = ±0.017 gm/cc), Pollock, et.al., 1976 ($\bar{X}$ density = 1.0431 gm/cc, S.D. = ±0.013 gm/cc), and Brozek, et.al., 1951 ($\bar{X}$ density = 1.0521 gm/cc, S.D. = ±0.118 gm/cc). The mean body density of this study population was less than those values reported on populations who were specifically active (a further discussion of this observation will be presented in a subsequent section).

The total group mean PBF (Table 10) was calculated from the subject's individual body density values through the "translation" equations of Brozek, et.al., (1963) and Siri (1961). This study's total population had a Brozek et.al., calculated mean PBF of 23.4% (S.D. = ±5.9%) and Siri calculated mean of 23.9% (S.D. = ±6.2%). These mean values are, again, comparable to those determined means on other similar population studies; $\bar{X}$ PBF (Brozek-equation) = 22.5% S.D. = ±6.2% of Myhre, et.al., (1966) study and $\bar{X}$ PBF (Siri equation) = 24.7%, S.D. = ±5.9% of the Pollock, et.al., (1976) study.

It is important to emphasize that the effective translation of body density into the relative values of PBF and lean body weight must be a source of discussion and contention. The accurate applicability of the derived densities of LBW and fat assumed and calculated by Siri in his equation and Brozek, et.al. in his equation must be reviewed. Such a review will be dealt with in the individual subgroup discussions

Table 10

Mean Body Fat Percentage Values

According to the Equations of Brozek, et.al.

and Siri for the Sub-Groups and Combined Group

	Percent Body Fat	
	Brozek, et.al.* (1963)	Siri** (1961)
Active (N-30)	21.1 ±4.2	21.5 ±4.5
Sedentary (N-20)	24.3 ±5.9	24.9 ±6.4
Cardiac (N-18)	25.9 ±6.3	26.6 ±6.9
Combined (N-68)	23.4 ±5.9	23 ±6.1

Values are Means ± Standard Deviations

*Brozek, et.al. (1963) formula: PBF = 457/DB - 414.2

**Siri (1961) formula: PBF = 495/DB - 450.

for it is the contention of this author that it is amongst the subgroups that the equation assumed densities of LBW and fat would vary (as a function of the subgroup population's activity and pathological states).

Table 11 is a presentation of the relationship between BD, RV, PBF, and LBW and specific age ranges. The number of subjects in each age-group varies and as such this author accepts that the greater the number the greater the credulance given to that group's mean. As previously mentioned, R.V. increased with age in an extremely regular manner. This increase may be attributed to the dimished elastic recoil of the lungs and the diminished mobility of the thoracic cage reported with increasing age by Kaltreider, et.al., (1933).

Figure 1 is the graphical plotting of PBF with age and LBW with age for the total group. The resultant correlation ($r = 0.21$) between PBF and age was not significant ($P > 0.05$) and the developed regression equation (for this relationship) was inadequate in its predictive capacity (due to the unacceptably high standard error of estimate of 5.7). Also, in Figure 1, the relationship between LBW and age elicited a non-significant correlation coefficient of -0.03. Furthermore the regression equation developed to predict LBW from age was also inadequate because of the very large standard error of estimate (6.03).

Percent Body Fat - Total Group

In Table 11, an analysis of the age-range groups illustrated some interesting points. the 46-50 year group demonstrated the greatest homogeneity in PBF, as reflected by the small standard deviation value. An ANOVA was conducted to assist in the definition of any differences that

Table 11

Summary of Total Group Results
(With Special Respect to Age)

	Age Ranges (years)							
	35-40	41-45	46-50	51-55	56-60	61-65	66-70	71-75
Number	16	17	11	8	4	7	2	3
Body Density (gm/cc)	1.0439 -0.013	1.0497 -0.013	1.0413 -0.006	1.05694 -0.0106	1.0288 -0.022	1.0374 -0.011	1.0413 -0.018	1.0410 -0.0102
Residual Volume (e)	1.26611 -0.277	1.5044 -0.297	1.5501 -0.342	1.7128 -0.439	1.8771 -0.496	1.9771 -0.280	2.1043 -0.072	2.2772 -0.426
PBF** (%)	23.63 -5.28	21.24 -5.64	24.68 -2.62	18.21 -4.25	30.15 -9.24	26.38 -4.69	24.725 -7.63	24.84 -4.31
Lean Body Weight** (kg.)	61.97 -5.95	61.23 -4.51	63.33 -7.15	58.20 -7.54	58.48 -6.07	56.76 -7.10	63.17 -5.13	55.35 -4.09

* Values are Means - Standard Deviations.

** PBF calculated by means of Brozek, et.al. (1963) PBF - $457/D_B$ - 414.2).

***Lean Body Weight calculated from the PBF* and subject's total body weight.

may have existed between the age-range groups. The ANOVA and the Scheffe Post-Hoc analysis (Table 12) demonstrated significant differences ($P<0.05$) between the mean PBF values of the 46-50 and 51-55 age-range groups and the 56-60 and 61-65 age-range groups.

The sharp decrease in the mean PBF in the 51-55 age-range group was partially attributable to the presence of an exceptional lean subject who had a measured PBF of 8.2%. This value was 11.5% lower than the mean PBF (19.6%) of that age-range group. The large variability and standard deviation of the group may be the reason that there existed a significant difference between the means of this (51-55) age-range group and the 56-60 age-range group. Furthermore, through coincidental selection, this group consisted of 6 members of the active subgroups and one member each of the CAD and sedentary subgroups. Zuti, et.al., (1973) and Boileau, et.al., (1971) demonstrated that physically active men elicited lower mean PBF values than normal sedentary or CAD populations. Due to the point that the overwhelming majority of the 51-55 age-range group were physically active an explanation of the significantly lower mean PBF value than the 56-60 age-range group becomes obvious. That is, the more active members in a sample group could be followed by a lower mean PBF value. This section of discussion will be expounded upon in a subsequent area.

Table 11 further indicated that the 66-70 year age-range group and the 71-75 year age-range group both reflected slight decreases in PBF. This observation can not be attributed to the sole observation of age-range group membership. The 66-70 year age-range group consisted of two CAD subgroup members. Also the 71-75 age-range group consisted of 3 CAD subgroup

Table 12

Statistical Analysis of Age-Range Group's PBF Values

(A) Analysis of Variance

Grand Total = 1585.63*
Number of observations = 68
Total Group Mean = 23.3181

Source of Variation	Degrees of Freedom	Sum-of-Squares	Mean-Squares	F-Ratio**
Treatments	7	423.16	106.72	2.31
Error	60	25571.42	46.2	
Total	67	2994.58		

(B) Scheffe Post-Hoc Analysis

1. Between the 46-50 age-range group and the 51-55 age-range group; Scheffe F - ratio = 3.01 ($P < 0.01$).***
2. Between the 51-55 age-range group and the 56-60 age-range group; Scheffe F - ratio = 7.24 ($P < 0.01$)***
3. Between the 56-60 age-range group and the 61-65 age-range group; Scheffe F - ratio = 5.61 ($P < 0.01$).***
4. Scheffe F - ratio values for all other age-range group comparisions were less than 2.17.

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*Total Summation of age-range group PBF values.

**An F - ratio value of 2.17 is needed for significance at the 5 percent level.

***An F - ratio value of 2.95 is needed for significance at the 1 percent level.

members. The phenomena of a declination in PBF values above a certain age was reported by both Siri (1962) and Fryer (1962). Siri observed a decline in PBF after the age of 60 years of age to a proportion similar to that of a 20-29 year population ($\bar{X}$ PBF = 22.6%). This finding was verified in the work of Fryer. The current explanation, offered by these two authors, of such a decrease is that it is due to natural selection, since subjects with very high PBF values are less likely to live as long as those found in the older limits of this study. Notation should be given to the small members (N=2 and N=3) of this study's 66-70 and 71-75 year age-range groups, and as such agreement with Siri's and Fryer's explanation might be solely coincidental.

A summary of the results of 3 major and comparable studies are available in Table 13. The results indicate that the mean PBF calculated in Kryzwicki, et.al., (1967) were very linear with age. The mean PBF values in Kryzwicki's study were all higher than those of the present study, except for the age-range groups of 56-60 years (this value was extremely comparable; 29.1% versus 30.1%). The reasons for the slightly lower values expressed in this study as compared to Kryzwicki's may lie in the populations utilized by this author. Kryzwicki's population was purely sedentary in contrast to the present study's two habitually active and one sedentary subgroup. This, the involvement of the parameter of physical activity has skewed the results of this group.

The mean values presented by Myhre's, et.al., (1966) study also reflect too, a linear relationship between age and PBF. When the present study's data is worked to reflect 10 year age-range groups (as is Myhre's

Table 13

Summary of Data Collected in Comparable Analysis

Study	Subject Number	Age Range	$\bar{X}$ PBF
Kryzwicki, et.al. (1967)	13	35-39	24.0
	25	40-44	24.4
	24	45-49	26.6
	12	51-54	28.6
	4	55-59	29.0
	5	60-64	31.2
	2	65-69	35.2
Myhre, et.al. (1966)	24	24-38	17.9
	16	40-48	22.4
	12	50-58	26.1
	8	60-68	23.4
Norris, et.al. (1963)	20	30-39	27.4
	34	40-49	29.2
	30	50-59	30.5
	25	60-69	30.4
	21	70-79	28.9

study's data is expressed) the results are very comparable (22.9% for a 40-49 year age-rage group; 24.2% for the 50-59 year age-range group; 25.3% for the 60-75 year age-range group). This study's mean PBF was only marginally higher in the 40-49 year age-range group, marginally lower in the 50-59 year age-range group, and slightly higher in the 60-75 year age-range group. Importantly, the present study as well and Kryzwicki, et.al., (1967) and Myhre, et.al., (1966) all reflected Siri (1963)'s observation of a decline in the population mean PBF in the age ranges above 60 years. This observation, as previously discussed, has been attributed to population limitations rather than physiological or anatomical reasons.

The findings of Norris and associates (1963) demonstrate further the linear increase in PBF up to the 60-69 year age range at which a slight decrease is exhibited (further adding creedance to Siri's observed phenomena). Norris's population groups were larger and totally sedentary males. Due to the nature of their classification (sedentary) a closer analysis of their results will be expounded upon and compared directly in the discussion of the sedentary subgroup's results.

Lean Body Weight - Total Group

Illustrated in Figure 1. was the plotting of LBW and age. The resulting relationship was insignificant (r = -0.03) and the regression equation statistically inadequate (due to the excessively high Standard Error of Estimate). The lack of relationship between LBW and age demonstrated in this study is in diagreement with several related studies. Norris, et.al., (1963) demonstrated linear decreases in LBW with age and

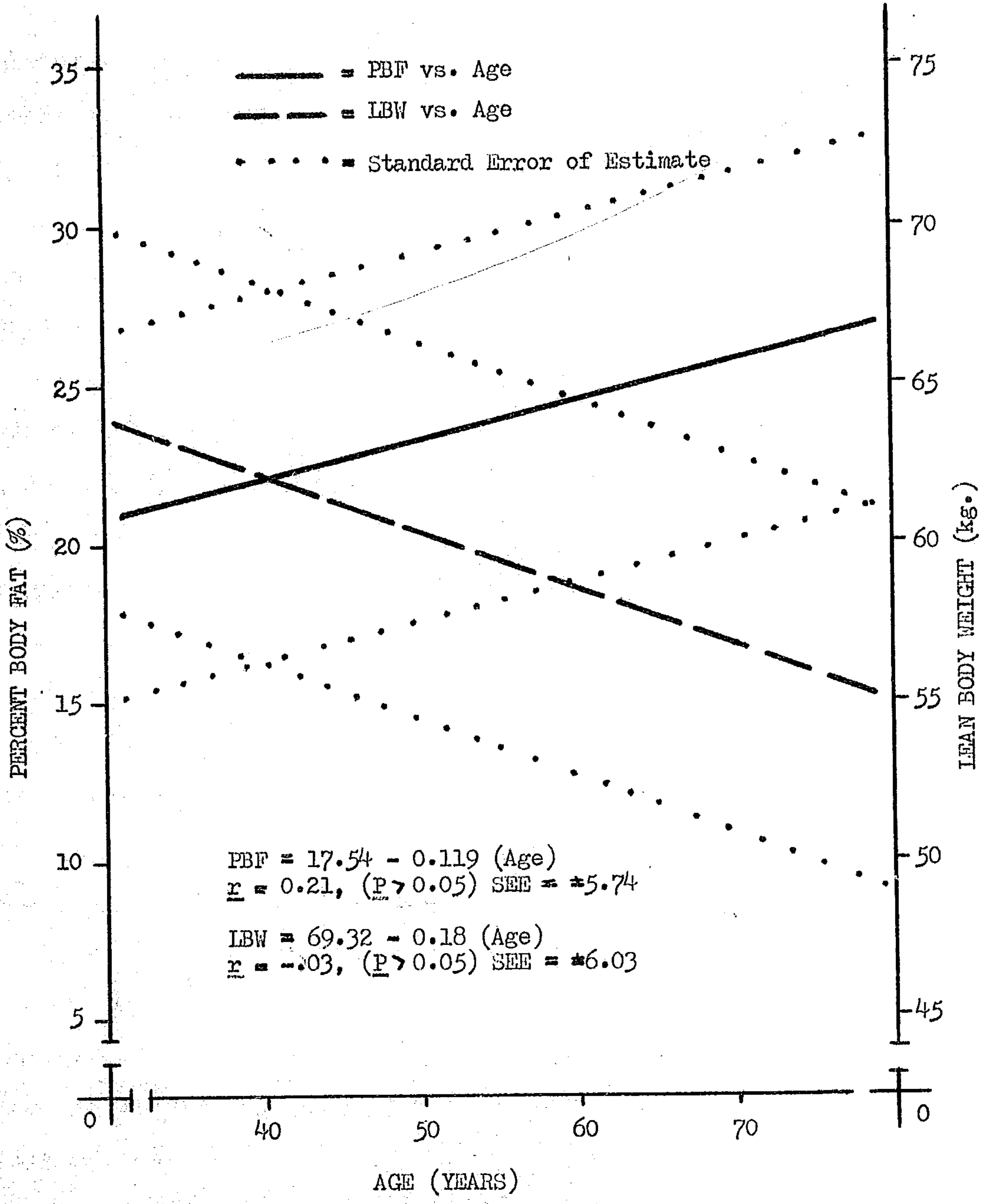

Figure 1. Percent body fat, lean body weight, and age - total group. Regression lines with standard error of estimates.

argued that the decrease was a function of vital organ, muscle mass, and bone substance decreases. Norris, utilizing the direct relationship between creatinine and muscle mass demonstrated a linear decrease in muscle mass with age ($P<0.001$). This observation was in agreement with the similar statistical analysis performed by Tzankoff, et.al., (1977) when they illustrated the direct linear relationship between creatinine excetion and muscle mass protein decomposition and LBW declines. Further analysis on this subject has been quantified by the several studies performed by Gilbert Forbes and his associates (Forbes, et.al., 1976; Forbes, et.al., 1972; Forbes, et.al., 1970), and has already been discussed in great length in the literature review of this study.

When all data was collated, the present study demonstrated that the subjects in this study had LBW that were declining at a rate of 0.18 kg. per year. This annual loss is comparable to Forbes, et.al., (1970) who reported a loss rate of 0.24 kg. per year (based on the cross-sectional analysis of 9000 subjects) and to Norris's, et.al., (1963) study which reported a LBW decline at a rate of 0.14 kg. per year.

Upon analysis fo the inter-age-group relationships in Table 11 a relative steady-state in LBW is exhibited between the 35-40, 41-45, and 46-50 year age-groups. This steady-state is followed by a slow decrease in LBW in each of the remaining age-range groups, (except for the two subjects in the 66-70 age-range group). This observation is in agreement with those of Forbes, et.al., (1970) who also observed that LBW was highest during the third decade of life (20-30 years) after which it decreased slowly for the next two decades (30 through 50 years) and then declined more rapidly.

Body Density - Total Group

The previous two parameters discussed, LBW and PBF, are both functions of the parameter of body density. The body densities of this study's population demonstrated a broad linear relationship with age. In some respects this observation is a source of confusion in terms of the fact that body density, too, did not demonstrate a linear decrease with age as did the population LBW values. The observations of this study are in contrast to those of Norris, et.al. study (1963) which demonstrated a linear decline in body density with age (though the decline was not statistically significant).

The translation of body density into the parameters of LBW and PBF have been a source of contention by this and several other authors, Brozek and Keys, in 1963, developed a translatory equation utilizing empirically determined total body values rather than the previously used hypothetical values for the density of LBW and fat. This formula, though empirically determined, required the acceptance of values for LBW density and fat density established on limited cadaver studies and analysis. These values have subsequently been debated as to their application to total population irregardless of a populations environment, activity, pathological, or dietary status. As discussed in chapter II, there exists a steady decline in LBW in normal subjects and the rate of such a decline is variable but distinct. There is a noticeable steady decline in bone density . Werdein and Kyle, in 1960, demonstrated the variability of the density of the body's LBW component due to alterations in the body's bone mineral content. Using subjects with osteoporosis and osteosclerosis, these authors were able to demonstrate that the density of the body's LBW component varied from 1.057 gm/cc in osteoporotic subjects to 1.189 gm/cc in the osteosclerotic subjects. Although quantitative descriptions of total body changes accompanying "normal" ageing are not available, in general, qualitative appraisals indicate a loss of bone mineral during the span of the adult years (Norris, et.al., 1963; Forbes, et.al., 1976; Forbes, et.al., 1972; Munro, et.al., 1978; Malina, 1969; and Albanese, 1977).

Further contributing to the variation in LBW density is the quantified decline in muscle mass due to protein break-down (Munro, et.al., 1978; Malina, 1969). This, in conjunction with a documented decline in the body's vital organ mass (Tzankoff, et.al., 1977; Brozek, et.al., 1952; Munro, et.al., 1978), point towards a parameter (LBW) that reflects enormous range and variability. Due to the evidence indicated in the studies already mentioned it would seem basically incorrect to utilize a single value for LBW (irregardless of age) in translating a subject's body density into LBW and PBF paramenters. Furthermore, considering the variability of the LBW's density with age, the utilization of a "cross-the-board" translatory equation (developed on the basis of "standardized" LBW and body fat densities) to calculate the PBF or LBW of a 12 year old boy athlete and for a 69 year old sedentary male, would result in inestimable errors in the actual values of LBW and/or PBF. It is, therefore, the contention of this author that the effective translation of a subject's body density into LBW and PBF values would be related to the age of that subject. Thus, the incorrect translation would reflect only relative relationships rather than absolute relationships.

The lack of noticeable trending amongst the body densities of this population may solely be a function of the lack of sensitivity induced by the equations utilized.

Results and Discussion - Subgroups

The purpose of this section is to present and discuss the resultant, PBF, LBW, and body density values of the individual subgroups. This was done with specific reference to the respective environmental and pathological differences between the subgroups.

Percent Body Fat - Subgroups

In Table 10 the means for PBF for each subgroup were calculated by Brozek's, et.al. and Siri's "translatory" equations. Accepting, the relative accuracy of these results, and the theoratical basis of the equations, one can ascertain differences. The PBF for the active subgroup was 21.1% (S.D. = ±4.2%). This mean was significantly lower ($P<0.05$) (Table 14) than the mean PBF of the CAD subgroup ($\bar{X}$ PBF = 26%, S.D. = ±6.4%). There existed no significant differences between the means of the active and the sedentary subgroups nor between the CAD subgroup and the sedentary subgroup. Since both the active and the CAD subgroups were active populations by definition the significant difference between them could be argued to be a function of the significant age differences ($P<0.01$) previously discussed (Table 7). Due to the elevated mean age of the CHD population the argument that the PBF means would be commensurately elevated would be in agreement with the results of several studies by Malina (1969), Lesser, et.al., (1963) Myhre, et.al., (1966). The reported increases in PBF could be possibly due to the decreasing

Table 14

Statistical Analysis of Subgroup's Mean PBF Values

(A) Analysis of Variance

Grand Total = 1585.63*
Number of observations = 68
Total Group Mean = 23.3181

Source of Variation	Degrees of Freedom	Sum-of-Squares	Mean-Squares	F-Ratio**
Treatments	2	285.39	142.69	4.918
Error	65	1885.81	29.012	
Total	67	2171.2		

(B) Scheffe Post-Hoc Analysis

1. Between the Normal Active Subgroup and the Normal Sedentary Subgroup; Scheffe F - ratio = 4.24.
2. Between the Normal Active Subgroup and the Clinically Diagnosed Cardiac Subgroup; Scheffe F - ratio = 8.86 ($P < 0.01$)***
3. Between the Normal Sedentary Subgroup and the Clinicially Diagnosed Cardiac Subgroup; Scheffe F - ratio = 0.82.

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*Total Summation of Subgroup's PBF values

**An F-Ratio value of 3.14 is needed for significance at the 5 percent level.

***An F-Ratio value of 4.95 is needed for significance at the 1 percent level.

sensitivity of the translation equations (Keys, et.al., 1953). The former conclusion, that the PBF changes are a function of the age differences, may be more applicable due to the relativity of the results.

Arguments by Weinsier, et.al., (1976) that elevated mean PBF levels in CAD populations exist do not explain satisfactorily the differences noted between the CAD subgroup and the active group. Weinsier, et.al., (1976) demonstrated significant correlations between PBF and several risk factors (systolic and diastolic blood pressure, and blood lipid levels) associated with CAD. On this basis he and his associates concluded that a distinct relationship existed between CAD and PBF (indirect rather than direct). A parallel relationship also exists between these risk factors to CAD and exercise. Exercise has been shown to decrease both elevated blood pressure levels and elevated blood lipid levels (Ekblom, Saltin, Stenberg and Wallstrom, 1968; Cassel, et.al., 1971; Amsterdam, Wilmore and DeMaria, 1978). The significant PBF differences between the active and CAD group are not explained fully by the fact that the CAD group had elevated PBF because of their disease. The significant difference in PBF values between the Active and the CAD subgroups can be attributed to two mechanisms. Primarily, a significant age difference was demonstrated between the Active and CAD subgroup (Table 7). Brozek, et.al., (1953), Forbes, et.al., (1976), Parizkowe, et.al., (1963), Norris, et.al., (1963) and Myhre, et.al., (1966) all reported significant increases in PBF with increasing age. Secondly, the CAD group performed their exercise prescription differently from the Active subgroup. Specifically, the level of intensity at which their exercise was performed was lower. This lower intensity may have been the determining factor in the PBF differences. Furthermore, a combination of the age and intensity differences could explain the PBF variences between the groups. Not enough information was elicited by the present study's methodologies to comment further on the role of the CAD subgroup's pathological state in affecting their PBF values.

In Figures 2, 3, and 4, the parameters of PBF and LBW have been graphically plotted against age for the three individual subgroups studied, active, sedentary and CAD. For each plotting, correlation coefficients and regression equations were developed. When PBF for the active, CAD and sedentary subgroups were plotted against age the correlation coefficients of 0.25, 0.21, and -0.22 were elicited. These coefficients were non-significant ($\underline{P} > 0.05$). Furthermore, all the regression equations developed to describe and predict PBF from age were statistically inadequate. Without exception, the regression equations predicting PBF from age in all three subgroups possessed unacceptably large Standard Error of Estimate values. The relationship between age and LBW for all three subgroups were also established. When plotted (Figures 2, 3 and 4) LBW and age elicit an $\underline{r} = -0.10$ for the active subgroup, $\underline{r} = -0.41$ for the CAD subgroup and an $\underline{r} = -0.28$ for the sedentary subgroup. The subsequently developed regression equations for predicting LBW from age demonstrated themselves statistically insignificant due to excessively high standard error of estimate values. The negative trending of the broad scatterplotting of LBW and age in Figures 2, 3 and 4 was at least marginally in agreement with the trends indicated in studies that will be discussed in the next section.

The marginal trend elicited, though non-significant, in figure 4 by the plotting of PBF and age was confusing, when compared to comparable relationships reported in the literature. An overview of the literature

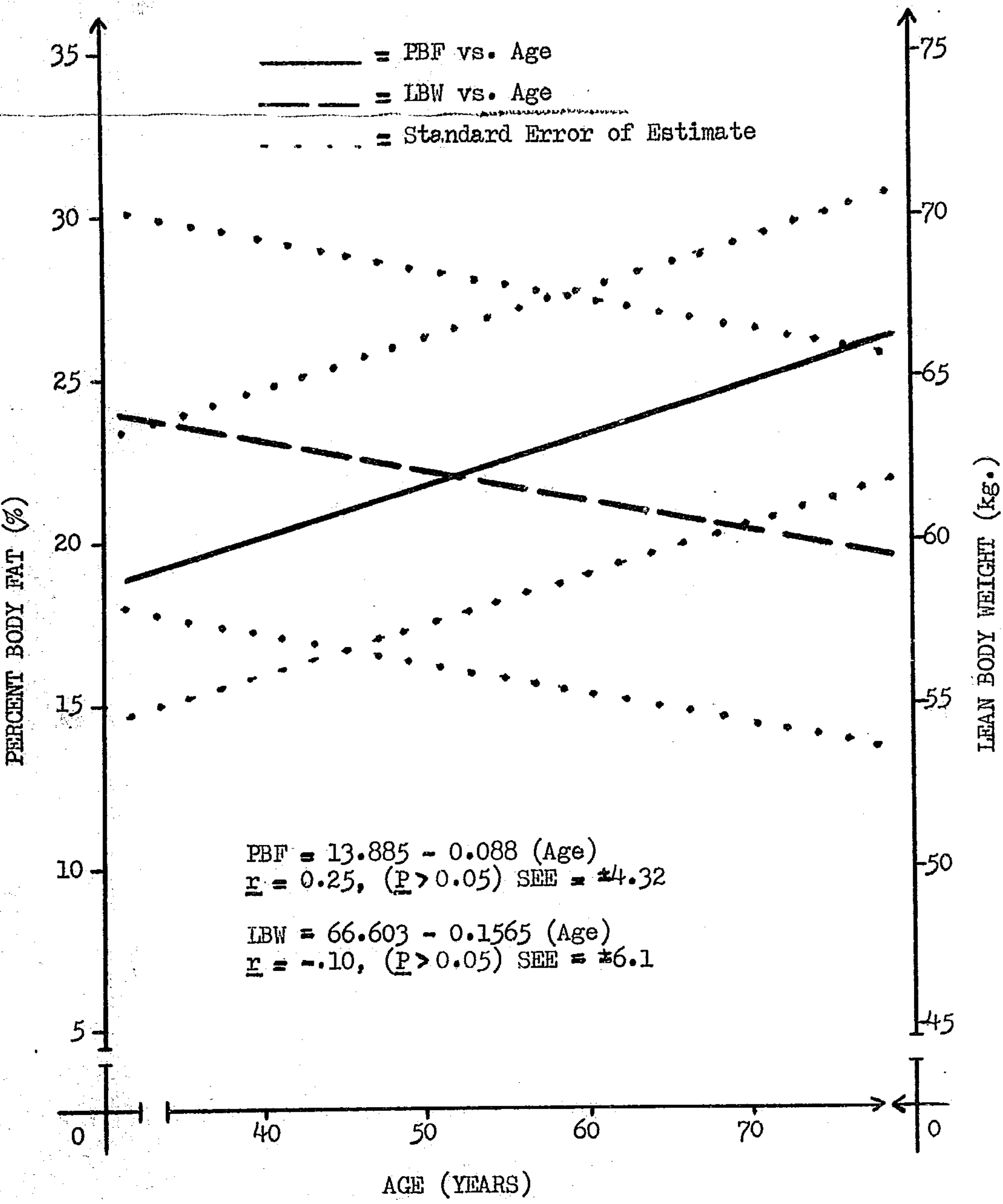

Figure 2. Percent body fat, lean body weight, and age - normal active sub-group. Regression lines with standard error of estimates.

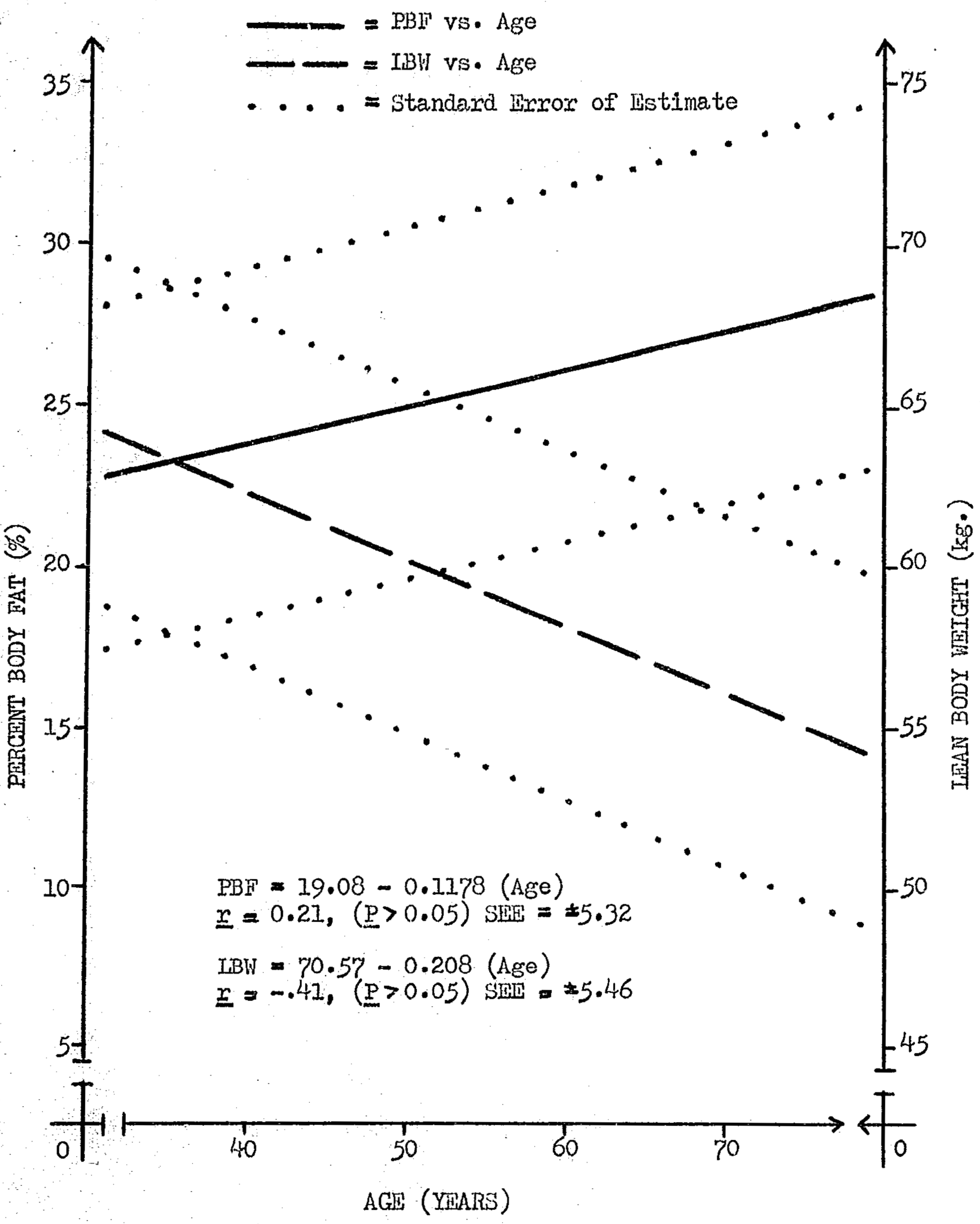

Figure 3. Percent body fat, lean body weight, and age - clinically diagnosed cardiacs. Regression lines with standard error of estimates.

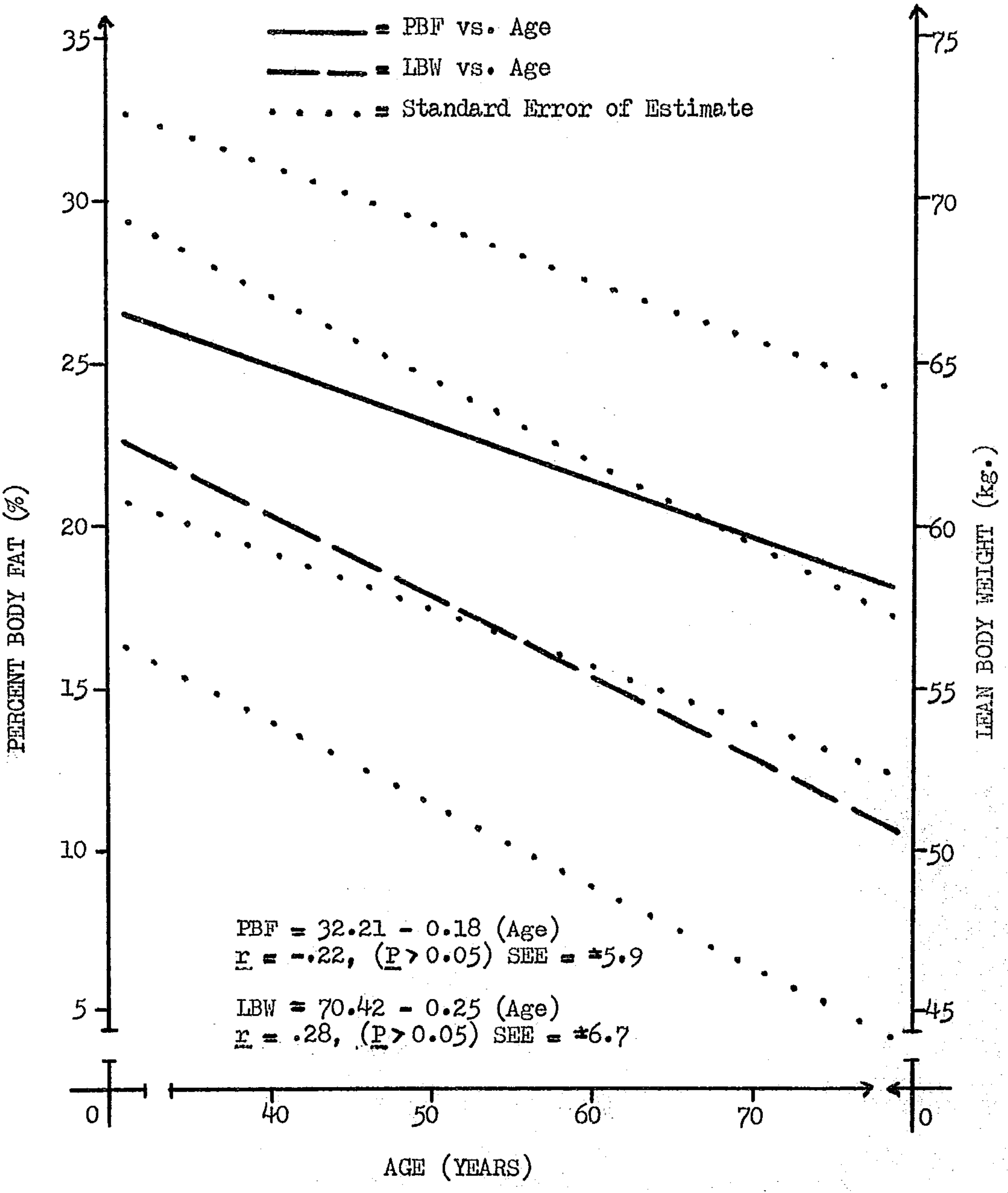

Figure 4. Percent body fat, lean body weight, and age - <u>normal sedentary group</u>. Regression lines with standard error of estimates.

has indicated that PBF either increases or is maintained with age (refer Table 2). This confusion was attributable to some exceptional results by a certain members of that subgroup. Within the sedentary subgroup was a 51 year old subject who had a PBF of 8.2% (this value was 16.7% lower than the group mean). Also the two oldest members (61 and 62 years) of the subgroup elicited noticeably lower PBF values (19.4% and 18.3% respectively) than the subgroup mean (24.9%). These two population-orientated factors effectively skewed the distribution of the population and elicited trends contrary to expectation.

The results and analysis give rise to the questioning of Wilmore's, et.al., (1969) hypothesis that "creeping obesity" was a parameter that could be controlled with physical activity. The results of this study demonstrated a non-significant relationship between PBF and age and the habitual physical activity that some subgroups were involved in did not retard the gradual onset of increased fat levels, Wilmore's conclusions were drawn on a small and specialized population. It becomes apparent that the specialization of this population may explain the conclusion the author drew. The population studied was comprised of three septeenarian distance runners. They weekly trained and ran an average of 22 miler per week. The intensity, duration, frequency and modality (running versus jogging) may explain the differences between the mean PBF reported in Wilmore's Study (16.5%) and the results for a similar age group in this study's active subgroup (X PBF for 55-65 year age-group - 22.07%) and it's CAD subgroup (24.8%). Wilmore's results are comparable to those of Pollock, et.al., (1974) study on Champion American athletes who were septegenarians.

Pollock, et.al., demonstrated a 13.6% fat in a sample of three men who ran an average of 20 miles per week. The control of "creeping obesity" may lie in the intensity and duration that one participates in activity. The notable trend in this study indicates that the physical activity participated in by this study's subjects did not retard the development of elevated fat levels if in fact physical activity can retard this potentially age-related phenomena.

Lean Body Weight - Subgroups

Table 15 presents the means and standard deviations of the three subgroup's LBWs. Utilizing Brozek's translatory equation, the calculated LBWs of the three subgroups demonstrated no statistical difference from each other ($\underline{P} > 0.05$). In figures 2, 3 and 4 the LBWs of the three subgroups were plotted against age. The resultant regression lines indicated non-significant relationships between LBW and age in all three subgroups. The correlation coefficients for the relationships, graphically presented in Figures 2, 3 and 4, were also non-significant. When LBW was plotted against age the following correlation coefficients were elicited; $\underline{r} = -0.10$ ($\underline{P} > 0.05$) for the active subgroup, $\underline{r} = -0.41$ ($\underline{P} > 0.05$) for the CAD subgroup, and $\underline{r} = -0.28$ ($\underline{P} > 0.05$) for the sedentary subgroup. The respective regression equations developed to describe the relationships between LBW and age in all three subgroups were all statistically inadequate in their predictive capacity. This was due to the unacceptably large standard error of estimate. The broad scatterplotting of LBW versus age indicated marginal decreases in LBW with age. The decline in LBW of the active population was less rapid than that observed among the CAD and the

Table 15

Mean Lean Body Weight Values According to the Equations of Brozek, et.al. and Siri for the Sub-groups and Combined Group

	Lean Body Weight (Kg.)	
	Brozek, et.al.*	Siri**
Active (n=30)	62.54 ±5.82	62.22 ±5.7
Sedentary (n=20)	59.46 ±6.69	53.91 ±6.6
Cardiacs (n=18)	58.53 ±5.8	57.86 ±5.7
Combined (n=68)	60.57 ±6.26	60.09 ±6.21

Values are Means ± Standard Deviations

*Brozek, et.al., (1963) formula: $LBW = T.W. - TW\ (4.57/D_B - 4.142)$

**Siri (1961) formula: $LBW = TW - TW\ (4.95/D_B - 4.5)$

when TW = Total body weight

sedentary subgroups. The active subgroup's LBW declined at a rate of 0.16 kg. of LBW per year. In contrast the decline amongst the CAD subgroup was 0.21 kg. of LBW per year and amongst the sedentary subgroup the decline was 0.25 kg. LBW per year. These observations are in agreement with the conclusions drawn by Forbes, et.al., (1970) who reported that the decline in LBW was retarded marginally by habitual physical activity. Forbes, et.al. argues that a synthesis of protein in the body's skeletal muscles contributes to a slight elevation of the muscle mass and thus an alteration of the LBW proportionate contribution to the body's density and also adds concern to the questioning of the equation-accepted LBW density. The modality of running or jogging have elicited similar results in comparable studies performed on middle and older aged males. Boileau, et.al., (1973) demonstrated a mean LBW amongst conditioned adult males as 60.6 kg. (S.D. = 3.4 kg.). Pollock, et.al., (1975) demonstrated a mean LBW as 66.38 kg. (S.D. = 4.7 kg.). Misner, et.al. reported mean values of 62.1 kg. (S.D. = 11.2 kg.). These values and those study's are in contrast to those reported in studies utilizing a different modality. Boileau, et.al., (1973) also studied the effects of weight training on body composition. Specifically Boileau and his associates demonstrated 4 - 5% increases in LBW when a subject actively participates in weight training. Misner, et.al., (1976) demonstrated up to 5% increases in LBW with weight training. These studies further demonstrated no significant alterations in LBW with the modality of running or jogging. On the basis of these findings it becomes apparent that part of the decline in LBW by all three sub-groups may be attributed to an age-related function as distinct from environmental, pathological, or physical activity conditions or patterns.

Body Density - Subgroups

Statistical analysis of all three subgroups demonstrated no significant relationship between age and body density. This observation was in agreement with the findings of Norris, et.al., (1963). This author demonstrated no significant regression in body density with age. This was expected because LBW and PBF, in all three subgroups, demonstrated non-significant relationships with age. Norris's (1963) findings were confusing when this author demonstrated linear (and declining) relationships between PBF and LBW (both derivatives of BD) and age. The discussion as to the applicability of standardized LBW and body fat densities being utilized in the translatory equations has been dealt with in an earlier section of this chapter. The results of that discussion seems applicable in clarifying the above mentioned discrepancy. Thus the observation of no relationship between age and body density in any of the subgroups, can be attributed to the lack of sensitivity of the translatory equations. This author feels that the relative steadiness of the subjects body density's with increasing age may be a function of the varying LBW and body fat densities. There may exist a "play-off" within the equations, that is, as the independent density of, say, a subject's bone component declines, the muscle mass density of that same subject may elevate slightly this maybe due to physical activity and thus the subject's body fat density exhibits a variable state. The exact values of LBW density and body fat density are of critical importance when studying this area of concern. There exists a need for the development of an age corrected or age sensitive translatory equation.

An area of discussion which requires further treatment was the possible effects of intensity and frequency of exercise on the retarding the onset of elevated PBF levels with age. Pollock, et.al., (1969, 1972, 1974) demonstrated that body density (and the resultant PBF) varied considerably with alterations in intensity and the frequency of exercise. These authors in 1969 demonstrated significant decreases in both body density and PBF (assuming the equations utilized were sensitive and applicable enough to demonstrate such a decrease in the population studied) when subjects trained at 65-80% maximum heart rate, 4 times per week. In contrast, Pollock and his associates other experimental group who trained 65-80% maximum heart rate and only two days a week demonstrated no significant changes in body density or PBF.

A follow-up study by Pollock and his associates in 1972 demonstrated that significant decreases in body density and PBF were elicited by experimental groups who trained at 90% their maximum heart rate and two days a week. These studies demonstrate the possibility of Wilmore's contention that "creeping obesity" could be retarded by exercise of greater intensity and/or frequency than that performed by subjects in the present study.

With the possibility that noticeable and statistically significant PBF changes are a function of the exercise quality (in terms of duration, frequency, intensity and modality) this author had to seriously look at the quality of the active and CAD subgroups exercise. The reason the findings of this study were not in total agreement with Wilmore's study may be attributed to the disparity that may exist between the quality of exercise Wilmore's population participated in and the present study's participation. The intensity and duration of the CAD and active subgroups may not be comparable to the "marathoning" activity of Wilmore's group. Even though the exercise prescription applied to the CAD and active subgroups were in agreement with the ACSM standards (American College of Sports Medicine, 1978) and Pollock's studies, the intensity and duration of actual activity by these subgroups may not have been. Specifically, the exercise prescription applied to these subgroups was self-administered and as such was not policed by this researcher. The net effect of such self-administration may result in the CAD and active subgroups being in a limbo state between a "true" or intensely active state (like Wilmore's population) and a sedentary state. This disparity in intensity of activity would explain part of the reason why the results of this study do not parrallel the findings of Wilmore. Wilmore's hypothesis that "creeping obesity" would be retarded with physical activity hinges solely on the quality and quantity of the activity performed and if the participants do not maintain these parameters then the expected retardation of PBF increases would not result. This area of study requires further quantification.

Chapter V

CONCLUSIONS

Summary

The present study examined the relationship between body composition and age amongst middle- and older-aged males. Densitometrically derived values of body density (BD), lean body weight (LBW), and percent body fat (PBF) were collected and presented on 68 male subjects aged 35 to 72 years (X age = 48.6 years). The correlation coefficient describing the relationship between LBW and age was found to be -0.03 ($P > 0.05$). The correlation coefficient for PBF versus age was 0.21 ($P > 0.05$).

The total group was divided into three subgroups, normal actives, normal sedentarys, and clinically diagnosed cardiacs (CAD). The active males (N=30, X age = 46.17 years) were participants in the Adult Fitness Unit of LaCrosse Exercise Program. The sedentary males (N=20, X age = 43.9 years) came from the City of LaCrosse area and did not participate in any regular physical activity. The CAD males (N=18, X age = 57.9 years) were participants in the Cardiac Rehabilitation Unit of the LaCrosse Exercise Program. There existed significant differences between the mean ages of the subgroups. The correlation coefficients describing the relationships between age and LBW and PBF for the active subgroup were -0.1 ($P > 0.05$) and 0.25 ($P > 0.05$) respectively. In contrast, the correlation coefficients for LBW versus age and PBF versus age among the sedentary group were -0.28 ($P > 0.05$) and -0.22 ($P > 0.05$) respectively. An analysis of the relationship between LBW and Age and PBF and age among the CAD subgroup elicited correlation coefficients of -0.413 ($P > 0.05$) and 0.21 ($P > 0.05$) respectively.

Significant differences were found to exist between the PBF of the active subgroup and the CAD subgroup. No significant difference was ascertained to exist between the mean LBWs of the three subgroups. A significant difference in the BDs of the active and CAD males did exist. LBW declined at a rate of 0.18 kg./year. This author hypothesised that the lack of significant statistical support was a function of the lack of sensitivity to age changes in LBW and body fat of the mathematical equations used to "translate" BD into the parameters of LBW and PBF. Furthermore, this author also felt that there existed a disparity in the physical activity performed by this population studied and the populations utilized in comparative studies.

Conclusions

There existed an, ANOVA verified, significant difference in the BDs among the three subgroups. This difference occurred between the active and CAD subgroups. But no difference existed between the active and the sedentary nor the sedentary and the CAD subgroups. A similar difference was found among the PBF values of the three subgroups. Specifically the differences lay between the active and the CAD subgroups.

The Scheffe Post-Hoc analysis did not isolate any significant difference between the active and the sedentary subgroups Hypothesis 1, Chapter 1, (no differences between the body compositions of the active and sedentary subgroups) may be accepted at the 0.05 level of significance.. The acceptance of this hypothesis was based on the, ANOVA verified, finding of no significant differences existing between the PBF, LBW, or BD values of active and sedentary subgroups.

There existed significant differences between the PBF and BD means of the active and CAD subgroups (F ratio = 8.86, $P < 0.05$ and F ratio = 8.74, $P < 0.05$ respectively). On the basis of this difference, Hypothesis 2 (no difference between the body composition adjustment mechanisms associated with physical activity among normal active and CAD active males) can neither be rejected or accepted. The statistical differences between the active and the CAD subgroups can not solely attributed to age differences because this study did not demonstrate any statistical relationship between age and PBF and BD values. The statistical results of PBF and BD differences between the active and CAD subgroups can only verify that a difference existed and cannot comment as to the mechanisms involved. Quantification of the mechanisms was beyond the scope of the research methodologies utilized in this study.

Hypothesis 3 (PBF increases are not a natural consequence of the ageing process and can be controlled by physical activity, irregardless of health status) cannot be conclusively accepted or rejected. Statistically, a non-significant relationship was demonstrated to exist between PBF and age. This non-significance was demonstrated in both subgroup and total group analysis. For the hypothesis to be tested fully the methodologies involved would have required larger subgroup samples and a better and larger distribution of subjects over the age range of 35 to 75 years. The smaller number of subjects present in the older age-range groups (Table 11) in the present study disallowed accurate statistical analysis.

The lack of significant statistical support in the present study has been partly attributed, by this author, to the mathematical equations used to "translate" BD into PBF and LBW values. Although not experimentally tested, the hypothesis that the equations, which assume constant LBW and body fat densities throughout life, used to "translate" BD into LBW and PBF lack sensitivity when applied to the total population (both extremely young to the extremely old) appears to be a distinct possibility. The independant LBW and body fat densities have been shown to vary with age and as such, an age-sensitive, or age-corrected "translation" equation may have elicited different statistical results and comparable trends. The concept that "creeping obesity" can be retarded with the ageing process is based on a steady amount of quality physical activity. Primary to the testing of this concept is the manipulation of the intensity and frequency of the physical activity. Concurrence with Wilmore's hypothesis was not forthcoming in the present study due to differences in the intensity (and possibly duration) with which the subjects in the present study performed their activity in comparison with those subjects of Wilmore's study.

Recommendations

Based on the conclusions drawn by the author, the following recommendations for further study are:

1. Appropriate testing of the hypothesis that increased intensity of exercise retards the onset of "creeping obesity" with age.
2. The development of an age-sensitive or age-corrected equation to be used in the "translation" of body density to the parameters of lean body weight and percent body fat.

3. The body composition of CAD males in the Cardiac Rehabilitation Unit of the LaCrosse Exercise Program should be assessed densitometrically.

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APPENDIX

APPENDIX A

Active Group

Subject Number	Age	Weight	Body Density	Residual Volume	Brozek % fat	Brozek LBW
1	51	81.07	1.0585	1.7435	17.55	66.84
2	42	77.89	1.0492	1.6079	21.35	61.26
3	43	66.8	1.0642	1.4337	15.24	57.47
4	45	80.05	1.0574	1.5031	17.98	65.66
5	46	95.69	1.0379	1.1672	26.09	70.72
6	55	80.84	1.0557	2.3355	18.69	65.73
7	39	72.9	1.0447	1.075	22.4	56.57
8	50	74.72	1.0449	1.8939	23.14	56.82
9	62	91.95	1.0290	2.091	29.92	64.44
10	46	98.3	1.0434	1.2978	23.81	74.89
11	43	75.51	1.0718	1.8241	12.18	66.31
12	50	99.32	1.0261	1.7195	31.21	68.32
13	55	69.5	1.0496	2.0735	21.18	54.78
14	54	79.71	1.0544	1.4506	19.34	64.29
15	38	76.08	1.0634	1.6567	15.56	64.24
16	48	88.32	1.0449	1.7163	23.16	67.86
17	37	76.76	1.0524	1.2591	20.06	61.36
18	35	75.51	1.0532	1.4535	19.71	60.63
19	49	88.78	1.0415	1.5256	24.58	66.96
20	46	71.77	1.0401	1.1131	25.16	53.71
21	41	87.87	1.0558	2.0244	18.63	71.5
22	53	74.38	1.0492	1.5247	21.35	58.5
23	45	77.21	1.0478	1.5926	21.96	60.25
24	53	64.4	1.0539	1.1807	19.42	51.89
25	39	93.65	1.0343	1.2208	27.64	67.76
26	42	72.34	1.0617	2.0468	16.25	60.58
27	39	72.9	1.0468	1.258	22.38	57.58
28	58	67.12	1.0562	1.8723	18.49	57.71
29	40	83.79	1.0527	1.0175	19.91	67.11
30	41	72.11	1.0551	1.6333	18.96	58.44

Sedentary Group

Subject Number	Age	Weight	Body Density	Residual Volume	Brozek % fat	Brozek LBM
1	51	48.87	1.082	2.1694	8.164	44.88
2	38	62.47	1.0425	1.036	24.17	47.37
3	42	81.41	1.0378	1.263	26.15	60.12
4	45	94.78	1.0152	1.084	35.95	60.71
5	41	91.27	1.042	1.224	24.39	69.01
6	35	80.61	1.032	1.373	28.63	57.53
7	41	78.9	1.0552	1.115	18.89	64.0
8	47	78.9	1.0455	1.233	22.9	60.83
9	46	82.88	1.0495	1.938	21.25	65.27
10	43	70.07	1.0539	1.2131	19.43	56.45
11	61	71.43	1.0251	2.295	31.63	48.84
12	39	84.13	1.0353	0.8031	27.22	61.23
13	40	77.1	1.0488	1.2071	21.51	60.52
14	43	77.21	1.041	1.0625	24.82	58.05
15	44	74.04	1.0453	1.311	22.99	57.02
16	40	81.86	1.0334	1.1685	28.04	58.91
17	45	79.02	1.0324	1.423	28.48	56.52
18	62	79.37	1.0566	2.361	18.34	64.81
19	39	88.77	1.0476	1.381	22.02	69.22
20	36	98.87	1.026	1.067	31.23	67.99

CHD Group

Subject Number	Age	Weight	Body Density	Residual Volume	Brozek % fat	Brozek LBM
1	64	68.03	1.0365	1.675	26.69	49.87
2	46	74.38	1.0375	1.346	26.29	54.83
3	50	74.20	1.0431	2.1006	23.93	56.45
4	52	73.36	1.0525	1.2246	20.02	58.67
5	67	53.81	1.0541	2.0536	19.33	59.54
6	73	75.64	1.0293	1.8443	29.81	53.08
7	71	77.1	1.0475	2.292	22.09	60.07
8	35	82.09	1.0658	1.985	14.58	70.12
9	62	86.62	1.0327	2.0511	28.32	26.09
10	43	70.52	1.0588	1.4938	17.43	58.23
11	39	96.15	1.022	1.2172	32.98	64.44
12	60	99.89	1.0075	1.2732	39.14	60.52
13	72	68.37	1.0462	2.6952	22.64	52.89
14	56	100.15	1.0167	2.487	35.31	66.08
15	68	95.58	1.0285	2.155	30.12	66.79
16	62	78.91	1.0336	1.798	27.94	56.86
17	60	72.45	1.0349	1.876	27.39	52.61
18	62	64.51	1.0481	1.634	21.83	50.43

APPENDIX B

APPENDIX B

PROCEDURE LETTER TO PARTICIPANTS

Dear La Crosse Exercise Program Participant:

Tim Kostelnik and David Lee, Graduate Students of the La Crosse Exercise Program, are conducting a research study which will compare, describe, and predict body composition (% fat, density, lean body weight) in active, sedentary, and coronary heart diseased males between the ages of 35 to 72 years. The study has been approved by the appropriate medical advisory personnel of the La Crosse Exercise Program. By random sampling procedures you have been chosen to be part of this study. Participation in the research project is entirely voluntary and will require a single visit to the Human Performance Laboratory of approximately 45 minutes duration. This time will be arranged personally with the researchers upon your agreement to participate.

If you agree to be a participant you will receive the following 3 tests:

1. Underwater weighing - to calculate the body volume and ultimately body density.
2. Residual volume assessment - to calculate the residual volume and therefore to be used in the correction of the density of the body for dead air space.
3. Several body (anthropometric) measurements - to allow the establishment of prediction equations for body composition.

It should be emphasized that the equipment and technique involved in the above assessments are the most modern available. The new pieces of equipment, installed recently in the Human Performance Lab., now allow the researchers to assess body composition with an even greater degree of accuracy than previously available.

Due to the size and scope of the study, we also require a group of sedentary males between the ages of 35 to 72 years. If you have a friend who is NOT involved in a regular program of physical fitness and who would like to have his body composition assessed, please let us know. The evaluations of these members of the sedentary group will be entirely free, as will your evaluation.

The results of the study will be fully explained to you (and your friend) in regard to physiological and health benefits. As mentioned earlier, arrangements for the session of your choice will be made by a follow-up phone call or personal contact "at the Program" by David or Tim. We hope you decide to assist us.

Yours,

Philip K. Wilson
Executive Director

APPENDIX C

APPENDIX C

PROCEDURAL LETTER TO PHYSICIANS

Dear Dr. ____________________:

Tim Kostelnik and David Lee, Graduate Students of the La Crosse Exercise Program, are conducting a research study which will compare, describe, and predict body composition (% fat, density, lean body weight) in active, sedentary, and coronary heart diseased males between the ages of 35 to 72 years. The study has been approved by the appropriate medical advisory personnel of the La Crosse Exercise Program. By random sampling procedures your patient(s), ____ has been chosen to be part of this study. Participation by your patient in the research project is entirely voluntary.

The patient will receive the following three tests:

1. Underwater weighing - to calculate the body volume and ultimately body density.
2. Residual volume assessment - to calculate the residual volume and therefore to be used in the correction of the density of the body for dead air space.
3. Several body (anthropometric) measurements - to allow the establishment of prediction equations for body composition.

The results will be fully explained to the patient (and a copy to yourself) with regard to physiological implications.

Please find attached the following:

1. A description of the methods and procedures to be used.

2. A copy of the Patient's Consent Form.

3. A Physician Consent Card for your approval (or disapproval) of the patient(s) to participate in the study.

We look forward to receiving the Physician Consent Card (#3) as soon as possible.

Yours sincerely,

Philip K. Wilson
Executive Director

APPENDIX D

APPENDIX D

PHYSICIAN'S CONSENT FORM

I _____APPROVE _____ DISAPPROVE of my patients

1. ______________________________
2. ______________________________
3. ______________________________
4. ______________________________
5. ______________________________
6. ______________________________
7. ______________________________
8. ______________________________
9. ______________________________
10. ______________________________
11. ______________________________

participating in the aforementioned research study. I understand that he will be underwater weighed, have residual lung volume assessed, and have a series of anthropometric measurements taken.

Signed ______________________________ Date ______________________________

Please return in enclosed envelope.

APPENDIX E

APPENDIX E

LACROSSE EXERCISE PROGRAM
UNIVERSITY OF WISCONSIN-LACROSSE

INFORMED CONSENT FOR BODY COMPOSITION ASSESSMENT

I desire to engage voluntarily in the Hydrostatic Weighing as a procedure of the LaCrosse Exercise Program. I understand that this research project compares and predicts body composition (% fat, density, lean body weight) in active, sedentary, and coronary heart diseased males between the ages of 35 and 72. My participation in this research project has been approved by my physician Dr. ______________________ of ______________________ clinic. If I am NOT a participant of the LaCrosse Exercise Program, I personally take the responsibility of my participation in the aforementioned research project.

As a participant in this research project, I will receive the following three tests:

1. Underwater weighing
2. Residual lung volume assessment
3. Several body (anthropometric) measurements

For the underwater weighing procedure I will be seated on a suspended weighing chair in a densiometry tank containing body temperature water. Furthermore, I will perform either a maximal inspiration or a maximal expiration followed by self immersion for 3 - 5 seconds while my underwater weight is recorded. This procedure will be repeated 8 - 10 times.

For residual lung volume assessment I will be seated in a chair outside of the densiometry tank. While breathing into a vitalometer I will perform a maximal exhalation and then begin breathing medical grade oxygen (99% 02), for 6 - 8 breaths. A nitrogen gas analyzer will assess the amount of nitrogen gas left in my lungs. Proceeding another maximal expiration I will begin to breath normal room air again.

The body measurements consist of several skinfolds, body circumferences, and body diameters. Body measurements will be assessed with a skinfold caliper, body anthropometer, and a steel measuring tape.

There exists the possibility of certain physiological changes occurring during the underwater weighing and residual lung volume assessment. These include variations in blood pressure, heart rate, and in very rare instances, "heart attack". Every effort will be made to minimize the possibility of such effects.

For my benefit, I will receive the results of my body composition assessment with a full explanation of their implications.

I have read the foregoing and I understand it. Any questions which have arised or occurred to me have been answered to my satisfaction.

SIGNED: ______________________ WITNESS: ______________________

DATE: ______________________

APPENDIX F

APPENDIX F

UNDERWATER WEIGHING DATA SHEET

NAME: ______________________________________ AGE: ____________

First Last

HEIGHT: ______________________ cm WEIGHT: __________ lbs. __________ kgm.

Immersion Tank Temperature: ______________ °C.

Density of Water: 0. ____________________

Residual Volume: ____________________ L.

Mass In Water: ____________________ kgm. (M_x)

Mass of Weighing Apparatus: ____________________ kgm. (M_y)

Mass of Water ($M_x - M_y$) ____________________ kgm. (M_W)

M_x Trials

1.
2.
3.
4.
5.
6.
7.
8.
9.
10.

Air in Gastro-Intestinal Tract = approx. 100 ml.
= 0.1 L.

$$\text{Density of Body } (D_b) = \frac{\text{Mass in Air}}{\dfrac{(M_a - M_W)}{D_W} - RV - 0.1\ \text{L.}}$$

= ____________________

APPENDIX G

APPENDIX G

RESIDUAL VOLUME DATA SHEET

NAME: ________________________________ AGE: ______________

First Last

	Trial 1.	Trial 2.
Initial Volume of O_2 (VO_2):	____________	____________
%N_2 (Impurity) (IN_2):	____________	____________
%N_2 (Alveolar) (AI_{N_2}):	____________	____________
%N_2 (Equilibrium) Range:	____________	____________
%N_2 (Equil.) (EN_2):	____________	____________
%N_2 (Final) (Af_{N_2}):	____________	____________
Dead Space (D.S.):	____________	____________

$$\text{RESIDUAL VOLUME} = \frac{VO_2\ (EN_2 - IN_2)}{(AI_{N_2} - Af_{N_2})} - \text{D.S.}$$

$$= \underline{\hspace{6cm}} \text{ L.}$$

APPENDIX H

APPENDIX H

LA CROSSE EXERCISE PROGRAM

Body Composition Study Results:

NAME: ______________________________

HEIGHT: ______________ cm. WEIGHT: ______________ kgm.

RESIDUAL VOLUME: ______________________ L.

% BODY FAT: ______________________ %

LEAN BODY WEIGHT: ______________________ kgm.

Therefore, assuming you have no pronounced alterations in your Lean Body Weight, for you to have an acceptable percent (%) body fat or 15% (or less) you will need to attain a minimum total body weight of ______________________ kgm.

Tim Kostelnik and David Lee wish to thank you for the gracious contribution of your time and interest. Please find below the Averages of each of the individual groups so that you may compare yourself with your peer group.

Active Group Average % Body Fat - 21.11%

Sedentary Group Average % Body Fat - 24.31%

Cardiac Group Average % Body Fat - 25.89%