THE ROLE OF ANTHROPOMETRY IN PREDICTING RESTING ENERGY EXPENDITURE AND OUTCOME IN SURGICAL PATIENTS

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Dissertation submitted to the University of Glasgow for the degree of Master of Science

January 1989

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TABLE OF CONTENTS

ABSTRACT

Anthropometry as a method of measuring body composition has been shown to correlate significantly with other body measurement techniques. Consequently anthropometry is widely used in clinical practice to estimate body mass, subcutaneous fat stores and skeletal muscle mass.

As skeletal muscle mass has been related to basal heat production it may therefore be appropriate to use arm anthropometry as a method of predicting resting energy expenditure. This hypothesis was considered in a group of weight stable and weight losing patients with or without malignant disease. Resting energy expenditure was measured using a fixed indirect calorimeter. In the four patient groups, there was a significant correlation between arm muscle circumference and resting energy expenditure measured by indirect calorimetry. This permitted calculation of equations for these patient groups to relate energy expenditure to arm muscle circumference.

The derived regression equation was tested in a small homogeneous group of overweight patients with benign disease studied prior to elective cardiac surgery. All patients underwent anthropometric and biochemical assessment followed by indirect calorimetry. Conventional predictive formulae as well as the previously derived regression equation were used to estimate resting energy expenditure and results compared with indirect calorimetry. Predictive formulae had poor ability in predicting resting energy expenditure. However, the uncalculated measurement of total mid upper arm circumference correlated strongly with resting energy expenditure measured by indirect calorimetry. Therefore in an

overweight population where the delineation of the triceps skinfold is difficult, it may be appropriate to use mid upper arm circumference rather than arm muscle circumference in the calculation of resting energy expenditure from arm anthropometry. These results indicate that derived regression equations are affected by body morphology.

Various disease states, surgical trauma and starvation affect the body's endogenous energy stores, reflected by changes in anthropometric measurements. The effect of surgery on body composition may be different depending on the patient's preoperative nutritional status. A group of elective patients admitted for major surgery were nutritionally assessed using anthropometric and biochemical indices on admission and at discharge. Arm anthropometry was used to classify patients as either normally nourished or malnourished in order to examine the effects of surgical trauma on body composition. Nutritional status defined by anthropometry was also considered as a predictor of outcome used either as a single anthropometric parameter or a prognostic nutritional index. Anthropometry had no predictive ability in the identification of postoperative complications. However, results suggest that the metabolic response to major surgery may be affected by preoperative nutritional status.

ACKNOWLEDGEMENTS

I wish to express my sincere gratitude to the following:

Professor D.C. Carter, Regius Professor of Clinical Surgery, Royal Infirmary of Edinburgh, for his advice and support.

Mr. Robert Birrell, Senior Chief MLSO and Mr. Ian Shanley, Chief MLSO for their skilful technical assistance.

Mr. H.J.G. Burns, Senior Lecturer in the University Department of Surgery for his clinical advice.

Mr. D.T. Hansell, Senior Registrar, for allowing me to use data which has been published previously as part of his Doctor of Medicine thesis and without which this dissertation would be incomplete.

Miss Anne McKellar for her friendship and secretarial expertise.

Dr. A. Shenkin, Consultant Biochemist, Glasgow Royal Infirmary for acting as my supervisor and for his support and invaluable advice.

Mr. O. James Garden, Senior Lecturer in Surgery, Royal Infirmary of Edinburgh for acting as my supervisor and for his friendly support, guidance and encouragement.

DECLARATION OF PUBLISHED WORK

The work presented in this dissertation was performed solely by the author, except where stated.

This work has been presented in part to the European Society of Parenteral and Enteral Nutrition (Leipzig 1988) and the Parenteral and Enteral Nutrition Group (Newcastle 1986). This work has been abstracted in the following journals:

Richardson, R.A. and Hansell, D.T. The role of anthropometry in predicting resting energy expenditure. Proceedings of the Parenteral and Enteral Nutrition Group, Newcastle, March 1986.

Richardson, R.A., Taggart, D.P., McMillan, D.C. et al. Inability of conventional predictive formulae to estimate resting energy expenditure (REE) in an overweight hospital population. Clinical Nutrition 1988, 7, 21.

Richardson, R.A., Burns, H.J.G., Roger, P. et al. Nutritional consequence of elective general surgical operations. Clinical Nutrition 1988, 7, 54.

This work has been published in part in the following journal:

Hansell, D.T., Richardson, R.A., Davies, J.W.L. and Burns, H.J.G. Estimation of resting energy by anthropometry. Clinical Nutrition 1987, 7, 51-57.

LIST OF TABLES

LIST OF FIGURES

LIST OF ABBREVIATIONS

AMA = Arm muscle area

AMC = Arm muscle circumference

BCM = Body cell mass

BW = Body water

CHI = Creatinine height index

FFM = Fat free mass

Ht = Height

LBM = Lean body mass

K_e = Exchangeable potassium

MUAC = Mid upper arm circumference

Na_e = Exchangeable sodium

NCWS = Non-cancer weight stable

PNI = Prognostic Nutritional Index

REE = Resting energy expenditure

TSF = Triceps skinfold thickness

Wt = Weight

CHAPTER 1 HISTORICAL REVIEW

i. Introduction

ii. Methods of measuring body composition

- a. Body weight
- b. Total body potassium
- c. Neutron activation body composition analysis
- d. Isotope dilution - radioactive isotopes
  - stable isotopes
- e. Creatinine height index
- f. Arm muscle circumference

iii. Methods of measuring fat mass

- a. Body densitometry
- b. Ultrasonic technique
- c. Electrical conductivity
- d. Skinfold thickness

iv. Nutritional status of surgical patient

v. Prediction of outcome in surgical patients

vi. Estimation of resting energy expenditure by anthropometry

vii. Aims of dissertation

i. INTRODUCTION

The word anthropometry is derived from the Greek 'anthropo' denoting a relationship to man and 'metron' to measure. Anthropometry is the science which deals with the measurement of body size, skinfold thickness and arm muscle circumference. These anthropometric measurements correlate significantly with other methods of estimating body composition and as a consequence are widely used in the assessment of nutritional status.

Early elementary studies on body composition were carried out over a century ago in Germany (Bischoff, 1863). In 1945 the first quantitative studies on body composition became available when information was obtained from the dissection and analysis of cadavers (Mitchell et al, 1945; Widdowson et al, 1951). Variations in the fat content of these bodies were found, while the composition of the fat free tissue was fairly constant containing approximately 73% water, 20% protein and 68 mmols potassium/kg. In 1964 Alexander and colleagues, as a result of post-mortem examinations on 155 cadavers, attempted to provide formulae for the calculation of total body fat, muscle mass and weight. The aim of these early studies was to establish references for healthy adults. However, these subjects died from a variety of diseases some of which may be considered 'metabolically active' causing changes in body composition. Today, accurate methods of measuring body composition enable monitoring of changes in metabolism and include radiographic techniques, isotopic labels and whole body density. Body composition studies have allowed the simple division of the body into compartments, each accounting for a proportion of the total body weight; skeletal muscle 30%, interstitial fluid 20%, skin and skeleton 10%, and viscera and other

tissues 15%. These compartments comprise 75% of the total body weight and may collectively be referred to as the fat free mass (FFM). The remaining 25% body weight is fat, appreciable quantities of which are found in splanchnic organs where, unlike subcutaneous fat, it is not utilised during starvation. All of these compartments vary in protein content ranging from 0.5% in fat to 35% in skeletal muscle. The assessment of protein and fat stores is of particular interest when assessing nutritional status as these are the body's endogenous energy stores and may be affected by surgery, trauma, sepsis or starvation.

The simplest least expensive and quickest method of assessing FFM and fat mass is by the use of anthropometry. There are a number of inherent errors associated with the use of anthropometry but when used correctly it is a valuable and informative component of nutritional assessment. The simplest, crudest and most widely used anthropometric parameter is body weight, combining an assessment of FFM and fat mass.

ii. METHODS OF MEASURING BODY COMPOSITION

a. Body weight

The determination of nutritional state from body weight is usually undertaken by comparing actual body weight with 'standard' weight for height tables. The most commonly used tables are the Metropolitan Life Insurance tables (1983). These are derived from an American population who applied for life insurance whereas 'ideal' body weight is derived from a population with the lowest mortality at given heights. Weight for height is further sub-divided into frame

size by simple division into small, medium and large frame. Various other methods of measuring frame size have been proposed and include measurement of wrist circumference (Grant, 1980), elbow breadth (Frisancho and Flegel 1983), and bony chest breadth (Garn et al, 1983), though none are commonly used in clinical practice. The use of these tables may be inappropriate for two reasons. Firstly they are derived from a population seeking life insurance and may therefore be self selective and secondly they are based on an American population which may be geographically and ethnically biased.

Another method of using weight as a nutritional index is by using 'power' indices, where height (ht) is expressed as a power function of weight (wt). Such indices include wt/ht ratio, wt/ht^2, wt/ht^3 and wt/ht^p where p is derived from observed wt/ht data of a given population. It has been suggested by Roche et al (1981) that wt/ht^2 is the most accurate indicator of leanness or obesity as it is the least height biased. Desirable body mass indices for men and women are between 20-25. These indices may, however, inaccurately classify muscular subjects as obese as demonstrated in a study by Welham and Behnke (1942) which described a group of professional footballers as being 25% overweight. Another physiological change not accounted for in these indices is the shortening of the spinal column as part of the ageing process and this invalidates their use in an elderly population.

American and Swedish studies (Hanes II, 1979; Symreng, 1982) demonstrated that a more accurate method of estimating nutritional status from body weight was to examine a large population of men and women with a wide age distribution and divide their body weights for

age into percentiles. This enabled evaluation of nutritional status by a predicted weight for age.

The importance of weight loss among hospitalised patients was first considered by Studley (1936). This classic study linked a 20% reduction in body weight to a 33% mortality rate in a group of patients undergoing peptic ulcer surgery. This study highlighted a need to quantify weight loss among surgical patients. Estimation of weight loss may be calculated by subtracting actual weight (A) from recalled (R) or predicted weight (P) which is derived from equations or tables. Morgan et al (1980) compared the use of R-A or P-A in a group of 86 surgical patients using a predicted weight derived from the local population. Recalled weight may be subject to inaccuracies as ill patients may weigh themselves infrequently. Results of this study show that R-A or P-A can reasonably estimate weight loss, although R-A had a smaller standard error of mean and hence gives a more precise estimate of weight loss.

Body weight is the simplest nutritional parameter to record in the ambulant patient but its value may be limited in the seriously ill patient where acute fluid balance changes may influence interpretation. A technique of weighing bed bound patients was described by Hansell (1982). Four sets of bathroom scales placed under the bed leg were used to weigh both patient and bed. The patient was then hoisted above the bed by means of a variable height trolley to allow the bed weight to be measured. Estimation of the patient's body weight was obtained by subtraction.

The only nutritional parameter that is routinely performed on admission to hospital is body weight. Weight will therefore continue to play a key role in nutritional assessment despite its limitations.

As body weight is a combined estimate of FFM and fat mass, it would be of interest to quantify these components. A number of techniques are now available.

b. Total body potassium

Potassium is one element present in significant amounts within the FFM that has a predictive value of body composition. The isotope ^{40}K comprises 0.012% of all naturally occurring potassium and its gamma ray emissions may be measured by means of a whole body monitor. This latter device consists of a chamber in which the subject is isolated and gamma rays, produced by disintegration of endogenous ^{40}K, are detected by scintillators. To minimise radiation from the external environment the monitor is surrounded by a steel or lead screen. Calculation of total body potassium enables a quantitative estimation of fat and fat free mass. Although the potassium content of tissues varies, for the purpose of calculation an average value is assumed of 66 mmol/kg and 60 mmol/kg in men and women respectively (Garrow, 1982).

Whole body monitors are expensive to build and their operation requires technical expertise. Consequently they are only found in specialist centres. Irrespective of these logistical problems the use of body monitors in clinical practice is limited. Many chronic and acute clinical conditions result in reduced potassium levels which may invalidate the use of total body potassium as a means of assessing body composition in the hospitalised patient.

c. Neutron activation body composition analysis

A non-invasive method of measuring body composition was

described by Hill and co-workers in 1978. The surface tissues of the body are irradiated with a small dose of high energy neutrons, creating an isotope of nitrogen (^{13}N) which emits gamma rays for a short period of time. Following irradiation the subject is transferred to a whole body radiation counter where the induced radioactivity is counted. Calibration factors are obtained by irradiating anthropometric phantoms of various shapes and sizes containing known quantities of urea. Total body nitrogen is obtained and protein is then calculated by multiplying by 6.25 (1 g nitrogen = 6.25 g protein). Similarly the elements calcium, phosphorus and sodium may also be quantified using this technique.

Determination of fat mass is derived from four site skinfold thickness measurements (Durnin and Womersley, 1974). This allows calculation of body water by subtracting weights of protein, minerals and fat from actual body weight. Although the radiation doses are small, equivalent to a chest radiograph, a precise analysis of some elements may be obtained using a tenfold increase in radiation dose. Whilst this is an accurate method of measuring body composition, the subjection of individuals to even small amounts of radiation is questionable. This technique is also limited in terms of the specialist equipment required and the need to transfer patients to the site of the equipment.

d. Isotope dilution

Isotope dilution is an invasive method of measuring body composition and uses both radioactive and stable isotopes.

radioactive isotopes

To obtain radioactive isotopes, elements are bombarded with neutrons, some of which are useful in metabolic studies and may be used to measure FFM. Since FFM contains approximately 73% water, an estimation of body water (BW) enables measurement of the FFM. This is achieved by using a radioactive isotope of water containing tritium (^3H_2O). A tracer dose of water with the isotope label is injected and reaches equilibrium after 3-4 hours. When the equilibrium concentration is measured the volume of dilution can be calculated:

$$FFM = BW/0.73$$

Body fat is calculated as the difference between FFM and the subjects body weight.

The FFM may be subdivided into extracellular and body cell mass. The size of the body cell mass is related to total exchangeable potassium. However, the radioactive isotope of potassium ^{42}K decays rapidly and is of no use in clinical practice. Shizgal (1981) validated a technique which indirectly measures total exchangeable potassium (Ke), Ke was determined from measurement of exchangeable sodium (Na_e) and body water using radioactive isotopes. The isotopes used were tritiated water (^3H_2O) and ^{22}Na which are both stable with half lives of twelve and two and a half years respectively.

$$Ke = R \times BW - Na_e$$

$$R = \frac{Na + K \text{ (whole blood)}}{H_2O \text{ content (whole blood)}}$$

$$\text{Body cell mass} = 0.00833 \times Ke$$

Extracellular mass = FFM -BCM

Shizgal (1981) using radioactive isotopes investigated a method of monitoring body cell mass as an index of the efficacy of nutritional support. He showed a significant correlation between the state of nutrition and the ratio of the extracellular and cell mass as reflected by Na_e and Ke. In 25 controls the Na_e/Ke ratio equalled 0.98 compared with a Na_e/Ke ratio of 1.98 in 75 malnourished patients; thus an increase in the Na_e/Ke ratio is associated with malnutrition.

The use of radioactive isotopes in metabolic studies is relatively cost effective. However, the primary prohibitive factor with the use of these isotopes is that they involve irradiation of the subject which is considered to be unethical particularly during pregnancy and in children.

stable isotopes

Stable isotopes as the name implies exist with much longer half lives than their radioactive counterparts and therefore do not emit radiation. They may be used to quantify FFM and in long term dynamic studies of various metabolic processes (Rennie, 1985, 1986). Stable isotope dilution methods may be used to analyse quantitatively total body water and muscle mass, using deuterium ($^2H_2$) and ^{15}N as the tracers in water and creatine. Deuterium not only quantifies FFM as derived from total body water but it is also a component of doubly labelled water ($^2H_2{}^{18}0$). This stable isotope can be used to measure energy expenditure in free living individuals for periods of weeks to a year. This is particularly useful in clinical studies of the 'critically ill' patient (Novick et al, 1988).

As stable isotopes are not radioactive large doses may safely be given to any subject including children and pregnant women. An infinite number of compounds may be labelled using the stable isotopes of hydrogen, carbon, oxygen, nitrogen and sulphur enabling the study of many metabolic processes. Quantitative analysis of the isotope concentration is by a radiometric method which is considered to be more sensitive than scintillation counting used with radioactive isotopes. As these isotopes occur naturally it is important that samples are handled carefully to ensure they are not contaminated by background isotopes.

e. Creatinine height index

Creatine is an energy storage molecule present in muscle and some 2% is converted to creatinine daily which is then excreted unaltered in the urine. The creatinine height index is related to measurement of muscle mass which is closely related to FFM. Reference tables for predicted urinary creatinine excretion have been derived from a healthy adult population. In man, the predicted creatinine excretion is 23 mg creatinine/kg ideal body weight and for women 18 mg creatinine/kg ideal body weight (Bistrian et al, 1975). To determine creatinine height index urine is collected over 24 hours for analysis of total creatinine and the creatinine height index calculated thus:

$$\text{Creatinine Height Index (CHI)} = \frac{\text{Actual urinary creatinine}}{\text{Predicted urinary creatinine}} \times 100$$

During starvation the production of creatinine is reduced with a consequent reduction in creatinine height index. Excretion values are expressed as a percentage of standard. Blackburn and co-workers (1977) classified protein depleted patients into two groups; those

with CHI's of 60-80% were moderately depleted and those with a CHI less than 60% were classed as severely depleted.

The predicted values were derived from a small group of young adults and although creatinine excretion does decrease with age, normal values for this group have not yet been determined. Whilst this method of determining FFM appears relatively simple, Greenblatt and Ransil (1976) have demonstrated the difficulties in obtaining accurate 24 hour urine collections by showing daily fluctuations of up to 36% in the estimation of creatinine excretion.

f. Arm muscle circumference

The anthropometric measurement arm muscle circumference (AMC) is used as an index of skeletal muscle mass which accounts for 30% of the FFM. Arm muscle circumference is derived from the triceps skinfold thickness (TSF) and mid upper arm circumference (MUAC):

$$\text{AMC (cm)} = \text{MUAC (cm)} - (\pi = 3.14 \times \text{TSF})$$

The subjects' AMC is compared with 'standard' values and can be used as a simple method of detecting protein malnutrition.

Arm muscle size has been used extensively in third world field studies, to assess the nutritional status of children. Standard AMC values for children were collated by Jelliffe (1966) and derived from studies on Polish and British children. Ideally 'standards' for AMC should be obtained from a local healthy, well fed population, but logistically this is often impossible.

The use of AMC as a nutritional parameter in adults was popularised by Bistrian et al (1974). Reference values for AMC in adults (Jelliffe, 1966) were obtained from surveys of military personnel in three Mediterranean countries and a 1941 survey of

10,042 women by American garment manufacturers for the construction of dress patterns. The standard AMC for males is 25.3 cm and for females 23.2 cm. These fixed values have not been stratified for age nor have any secular trends in muscle mass towards a taller and heavier population been accounted for (Bakwin and McLaughlin, 1964). Frisancho (1974) used the American 10 State nutrition survey of 1968-1970 to derive 'norms' for upper arm muscle size. Results were based on a cross sectional sample of 12,396 subjects ranging in age from 0-44 years. As shown by 50th percentile values, their results demonstrated that AMC in children is not independent of age. The AMC in boys of 1-5 years increases by 15% and in girls by 11%, whilst between the ages 6-13 years there is a 32% increase in AMC in boys compared with 34% in girls, and from the age 14-30 years there is a 27% increase in AMC in men and only a 13% increase in women. Gender differences in AMC are present by the age of 13 years. This study also used arm muscle area (AMA) as a nutritional marker:

$$AMA_{(mm^2)} = \frac{\pi}{4}(\text{arm diameter}^2)$$

There were marked gender differences by the age of 40, there being over 56% difference in AMA between men and women.

Another population study critical of Jelliffe's reference values for AMC came from Sweden. Symreng (1982) used 1860 healthy individuals between 20-101 years to establish reference values for AMC. Results showed that AMC in women decreased during the tenth decade, whilst there was an increase from the 3rd-6th decade. In men no variation was seen during the 3rd-7th decades although decreases were shown to the tenth decade. The data obtained from this population study was then applied to a group of 112 surgical

patients, 6% of whom were found to be protein depleted. The 5th and 10th percentiles were used to define moderate and severe depletion of muscle mass. The use of percentiles in analysis of anthropometric results was used since data was positively skewed. There is no limit to the obesity of a subject, while there are limits as to how thin a person may be. This study also examined the AMC in the surgical population in terms of more practical age limits (20-79, 80-89 years) and compared this with the AMC results for each decade. With the use of decade groups there were 4% positive and 11% negative arm anthropometry assessments compared with Jelliffe's standard which gave rise to 44% false positives and 8% false negative assessments.

Arm muscle circumference is a one dimensional measurement and as such will undergo proportionally smaller changes than AMA. Arm muscle area is also used as an expression of muscle mass and the calculation is based on four approximations:

i) that the mid-upper arm is circular

ii) the triceps skinfold is twice the average fat rim diameter of the arm

iii) the mid arm muscle compartment is circular

iv) the bone included in measurement of AMA atrophies in proportion to muscle.

Heymsfield et al (1982) compared calculated AMA with AMA measured by computerized axial tomography and noted that each of these four approximations were subject to an over-estimation of between 20-25%. This group revised the equation of AMA, having identified two correctable sources of error. These were a 10-15% over-estimation in the mid arm muscle compartment and a 5-10% over-estimation due to inclusion of mid arm cross sectional bone area.

These revised equations for AMA were validated in healthy and chronically undernourished subjects. Radiographic techniques were used to check the revised equations and CHI to establish a correlation between revised AMA and body muscle mass. There was a significant correlation with CHI and AMA in males and females.

A sub-group with refractory protein energy malnutrition were reviewed to establish a lower range of corrected AMA by undertaking measurements on nine chronically malnourished patients who had died. The mass of arm muscle tissue at death was measured and this proportion of muscle was termed 'metabolically unavailable' and was allowed for in the equation for 'available' AMA. This revised equation had the effect of reducing the over-estimation of AMA to between 7-8%.

Neutron activation analysis has also been used to validate anthropometry in assessing protein stores. Collins and colleagues (1979) studied a group of normal subjects and surgical patients with varying degrees of weight loss. A regression equation for prediction of total body nitrogen in the normal subjects was calculated from anthropometric measurements including AMC and AMA. Armour-Forse and Shizgal (1980) also demonstrated a significant correlation between AMA and body cell mass derived from the use of stable isotopes in 216 obese, normal and malnourished surgical patients. The accuracy of AMC, AMA and MUAC in the assessment of protein stores was shown to be of value when studying groups of patients while, in the individual, these measurements were subject to large variance.

Arm muscle circumference is a subjective measurement and as such observer differences will be recorded. To be of any value these measurements must be reproducible. The precision of upper limb

anthropometry has been studied (Hall et al, 1980; Harris et al, 1984). Hall developed a statistical technique which allowed an estimation of measurement variance between observers. This is useful in determining real rather than apparent changes in AMC. Harris studied the reproducibility of anthropometry in normal and obese subjects examining intra and inter observer error. Variation in AMC was high, especially in the obese population when the measurement with least variation was MUAC. A malnourished group with Crohn's disease was also studied and MUAC was found to have the strongest correlation with changes in laboratory and other anthropometric markers (Harris et al, 1982).

The upper arm is a site readily accessible in the bed bound critically or chronically ill patient and is also a sensitive site for measurement of muscle mass as it is virtually unaffected by oedema. While the use of AMC is subject to several limitations, it remains a simple, quick, cheap and reproducible method of assessing protein status.

iii. METHODS OF MEASURING FAT MASS

Estimates of fat mass may be derived by densimetric methods, ultrasonic techniques, electrical conductivity and by measurement of skinfold thickness.

a. Body densitometry

Discovery of the theory of density has been credited to Archimedes, the Greek scientist, and is often referred to as the Archimedes principle. At body temperature fat has a density of 0.900 g/ml and fat free mass a density of 1.099 g/ml; therefore the

smaller a subject's body density the greater the proportion of fat. By submerging a subject in water, body density may be calculated from the volume of water displaced (Benhke et al, 1942). The proportion of fat to total body mass may be calculated using Siri's formula (1956):

Fat (% body weight) = (4950 - density - 4.500) x 100

The volume of displaced water at time of measurement will include that due to air trapped in the gut and lungs. The air in the lungs can be measured by 'three breath nitrogen dilution' (Rahn et al, 1949). This method of measuring fat mass requires a considerable degree of subject compliance and mobility and is therefore of little use in clinical practice.

b. Ultrasonic technique

This technique is based on the reflection of ultrasonic waves from the interface between subcutaneous fat and muscle to measure the thickness of fat and skin. As a means of determining fat mass, Bullen et al (1965) compared ultrasound with skinfold caliper readings from the triceps and abdomen in 100 healthy individuals. There was a strong correlation between ultrasound and skinfold measurements in both men and women. In the clinical setting it is unlikely that ultrasound facilities would be available for body compositional studies.

c. Electrical conductivity

Stainless steel wires are inserted into the skin, one wire at an arbitrary distance into subcutaneous tissue, the second is advanced until the electrical resistance shows a maximum rate of

change with movement. At this point the wire should be at the fat muscle interface. Prior to withdrawal, the wire is clamped with forceps to mark the position of skin surface and the distance between the forceps and tip of wire is measured (Booth et al, 1966).

This method of measuring subcutaneous fat thickness is invasive and unpleasant despite the use of preliminary anaesthesia and is therefore unacceptable in the assessment of hospitalised patients.

d. Skinfold thickness

A substantial proportion of the fat mass lies subcutaneously (Edwards, 1950) and anatomical experience suggests that as much as two-thirds of the fat mass may be located there. During periods of nutrient deprivation these fat stores will be drawn upon to supply energy. Skinfold thickness measurements provide a method of quantifying the amount and change in this energy reserve. A number of sites have been found appropriate (Durnin and Womersley, 1974) and include the triceps, biceps, sub-scapular and supra-iliac regions. Skinfold measurements include a double layer of subcutaneous fat, which must be picked up and measured in the prescribed manner (Tanner, 1959).

Skinfold measurements are taken using specially designed calipers. Investigations into caliper design and the accuracy of their use has been carried out by Edwards and co-workers (1955). The most reproducible results within and between observers was found with the Harpenden Skinfold Caliper (Holtain Ltd., Bryberian, Crymmych, Pembrokeshire, UK). The Harpenden caliper was adapted for use in the Harpenden Growth Study (Tanner, 1953) having originally been used in industry to measure the thickness of metal, plywood and leather. It

has been observed that an experienced individual would obtain more accurate results in skinfold measurements than an inexperienced user. Another type of skinfold caliper commonly used in the United States is the Lange Caliper (Cambridge Scientific Industries Inc., Cambridge, Maryland, USA). Both the Harpenden and Lange calipers are relatively inexpensive, but are cumbersome. Plastic skinfold calipers (McGaw Laboratories, Irvine, California, USA) were introduced in the mid seventies as a cheap, light alternative to conventional instruments. Burgert and Anderson (1979) considered the efficacy of the plastic calipers compared to the Lange calipers in the measurement of triceps skinfold (TSF) thickness. There was a significant correlation with Lange calipers, although the absolute values measured by plastic calipers were lower. However, these plastic calipers were unable to measure triceps skinfolds that exceeded 45 mm.

Factors that influence skinfold measurements include obesity, oedema and skinfold compressibility. Fletcher (1962) examined these factors in 240 patients with various diseases including obesity and anorexia nervosa. Skinfold measurements were taken in triplicate at nine sites by one observer and variation was found to be low. The use of anthropometry in estimating fat mass was validated in this study by using tritiated water to measure total body water from which fat mass may be derived. Skinfold measurements correlated with fat mass measured by isotope dilution techniques.

Some of the most important work on the estimation of fat mass from skinfold thickness has been performed by Durnin and Womersley (1974, 1977). Four site skinfold and densimetric methods were used to assess the fat mass in 209 men and 272 women. Subjects included

anorexic, obese and muscular individuals whose fat content ranged from 5-50% and 10-61% in men and women respectively. A table sub-divided for sex and age was derived from their results in which percentage body fat corresponded to the four site skinfold measurements. In this study there were no significant differences in skinfold thickness between left and right limbs or between Harpenden or Lange calipers. Further work from this group (Wormersley and Durnin, 1977) has predicted the amount of body fat in obese subjects using densitometry, skinfold measurements, weight/height relationships, percentage overweight and the independent variables weight and height. The best correlation with densitometry in assessing fat mass was found to be skinfold measurements. Although the standard error of prediction was similar, it may therefore be appropriate to use another technique to estimate fat mass to validate the accuracy of skinfold predictions.

Anthropometry was also studied by Bray et al (1978) to assess weight loss in two weight reduction programmes. Serial measurements of biceps, triceps, sub-scapular, suprailiac and knee skinfold thickness showed no correlation in these obese subjects. However, significant correlation was demonstrated between sub-scapular skinfold measurements and weight loss. Intra-observer variation was found to be largest in obese subjects compared to lean individuals. This group concluded that the use of skinfold calipers was inappropriate in assessment of fat mass in the obese individual since the skinfold is often too large to be accommodated within the caliper's jaws.

The most commonly used single skinfold measurement is the triceps skinfold (TSF). As with AMC 'standard' values have been

derived from population studies of healthy individuals (Jelliffe, 1966). Symreng (1982) noted significant increases in TSF in females from the 3rd-6th decade while in males there was no variation in TSF until the 10th decade at which time TSF fell. In this study 1860 individuals aged 20-101 years were included. A similar American population study (Frisancho, 1974) considered 12,396 subjects from infancy to 44 years of age and reported that subcutaneous fat as indicated by TSF in males is characterised by slow deposition, whilst in women it is continuous throughout life. Gender differences in TSF are apparent by the age of three. Triceps skinfold and AMC measurements were evaluated in 91 healthy subjects (Burgert and Anderson, 1979) and compared with Jelliffe's standards. There was no significant difference between measurement of either right or left arm in male or female individuals. Arm muscle circumference was found to be greater in those subjects involved in right handed sports. Results of this study showed that TSF for men, AMC and MUAC in women were higher than Jelliffe's standard values, while AMC and MUAC in men and TSF in women were found to be lower than Jelliffe's standards.

Ultrasound, skinfold measurements at infra-scapular and abdominal sites, and electrical conductivity were assessed as methods of measuring subcutaneous fat (Booth et al, 1966). Electrical conductivity and ultrasonic techniques correlated strongly with skinfold thickness in the assessment of subcutaneous fat stores.

The use of skinfold measurement in nutritional assessment provides a simple, quick, cheap, readily available, non-invasive method of estimating the body's fat reserves.

iv. NUTRITIONAL STATUS OF SURGICAL PATIENTS

The pioneering work of Studley (1936) suggested that significant preoperative weight loss might be responsible for the high mortality rate found in ulcer patients following major surgery. However, it was not until Dudrick and co-workers (1968) developed a technique for the safe administration of nutrients via a central vein that the nutritional status of hospitalised patients was examined with renewed interest.

The existence and prevalence of malnutrition in hospitals was reported by Bistrian and colleagues (1974, 1976) who examined the nutritional status of surgical and medical patients in the United States. A survey of 113 surgical patients (Bistrian et al, 1974) reported a 50% incidence of malnutrition using the indices TSF, AMC and albumin. The later and larger survey (251 patients) on medical patients (Bistrian et al, 1976) demonstrated that 44% were malnourished as measured by anthropometric and somatic protein indices. One important difference between the medical and surgical populations studied was that although the medical patients were more energy depleted they had an improved protein status when compared to their surgical counterparts. This may in part by due to the greater catabolic nature of surgical illness.

The search for an index or indices that would readily identify the malnourished patient continued. Young et al (1978) attempted to identify a sensitive nutritional marker in a group of surgical patients preoperatively, less than a week postoperatively and one week postoperatively. A battery of anthropometric and laboratory markers were used to determine patient's nutritional status and the

effect of a surgical insult. Preoperatively, patients had low pre-albumin, retinol binding protein and arm-muscle circumference while all other plasma proteins and anthropometric measurements were essentially normal. Following surgery there was a progressive decrease in anthropometry and plasma proteins. The variables pre-albumin, transferrin, percentage weight and AMC were considered the most valuable indices in the nutritional assessment of the surgical patient. However, the decrease in postoperative measurements may have been a result of either surgery or semi-starvation. Work on the validity of using biochemical and anthropometric indices in assessment of nutritional status has been carried out by Armour-Forse and Shizgal (1980) and Collins et al (1979). Neutron activation was used to assess the value of anthropometry in predicting protein status in surgical patients (Collins et al, 1979). Anthropometry was found to be a useful predictor of protein malnutrition in groups of patients, although it was insensitive to changes in body composition in the individual. The results of that study were supported by Armour-Forse and Shizgal (1980) who used isotope dilution techniques to validate the nutritional indices; weight/height, albumin, total proteins, AMA, TSF, creatinine/height index and grip strength in 216 patients. Each index significantly correlated with patient's nutritional state as assessed by body composition studies. As with the work of Collins et al (1979) the confidence limits were wide and the nutritional indices were insensitive to changes in body composition. These results suggest that this approach to nutritional assessment of the individual may be inappropriate. As previously stated nutritional indices are affected by other variables which include state of hydration, drug therapy, surgery, sepsis,

anaesthetics and consequently those indices may not reflect the patient's 'true' nutritional status.

The wasting of skeletal muscle is one effect of malnutrition; therefore muscle function has been considered as a nutritional index which may not be influenced by non-nutritional variables. Lopes et al (1982) used skeletal muscle function in malnourished patients and controls to assess the force of muscle contraction and the maximal relaxation rate following electrial stimulation of the adductor pollicis muscle. Results demonstrated that the malnourished patients had greater muscle fatigue, weaker muscle contraction and slower relaxation when compared with the control group. Muscle function improved after four weeks intravenous feeding. Another method of assessing muscle function is by using a Handgrip Dynamometer (British Indicators Ltd) which measures grip strength and comparing results with standards derived from a control group (Klidjian et al, 1980). A study by Elia et al (1984) on starved controls, partially starved patients undergoing abdominal surgery and fed surgical patients demonstrated that grip strength was not significantly altered by 4 days starvation despite a fall in levels of circulating plasma proteins falling while surgery had the effect of reducing plasma proteins and causing a temporary reduction in grip strength. This study suggests that grip strength is an insensitive marker of starvation and while there was a sustained reduction in grip strength postoperatively, this may not be due to changes in primary muscle function alone. Surgery and anaesthesia may affect grip strength. Conversely it has been suggested (Klidjian, 1980) that grip strength is the most sensitive predictor of postoperative complications, correctly predicting 48 of 55 complications in 102 surgical patients.

Despite the inherent errors associated with these methods of nutritional assessment, malnutrition is a common finding among surgical patients. It would seem a logical assumption that changes in body composition, acute or chronic, may affect postoperative outcome.

v. PREDICTION OF OUTCOME IN SURGICAL PATIENTS

The implications of malnutrition in a surgical population were considered by Mullen et al (1978), who assessed the nutritional status of 64 preoperative patients using anthropometric and biochemical indices. Ninety-seven percent had one abnormal nutritional index and 37% had three abnormal indices prior to surgery. In this study albumin, transferrin and delayed hypersensitivity were found to be good prognostic indicators of postoperative mortality and morbidity. Similarly Hill and co-workers (1977) found that patients with exacerbations of inflammatory bowel disease requiring emergency surgery had low plasma proteins when compared to a control group. Postoperatively plasma proteins and AMC were found to be even lower in patients who developed major complications. This group suggest that perioperative nutritional therapy is appropriate in patients with inflammatory bowel disease requiring urgent surgery. Conversely the early postoperative parenteral feeding of malnourished cardiac patients (Abel et al, 1976) was shown to have no effect on morbidity and mortality when compared to a group of well matched controls. However, the feeding period in this study was only 5 days and perhaps not sufficiently long for the administration of effective nutritional therapy.

The efficacy of preoperative nutritional therapy was examined

by Muller et al (1982) in a landmark paper on the use of intravenous feeding in patients with malignant gastrointestinal disease. Those patients who received preoperative intravenous feeding had an improved nutritional status as defined by plasma proteins and a significantly lower mortality and incidence of major complications when compared to the control group.

To facilitate the prediction of outcome in surgical patients there has been interest in the derivation of a Prognostic Nutritional Index (PNI) (Mullen et al, 1980; Simms et al, 1982; Garden et al, 1985) from a range of nutritional indices. A PNI should possess the ability to prospectively identify malnourished patients who are at risk of developing complications. Mullen and co-workers (1980) developed a PNI from a linear predictive model which relates the risk of postoperative mortality and morbidity in individual patients using the indices albumin, transferrin, TSF and delayed skin sensitivity. Using this PNI patients are classified as being at low (PNI <40%); intermediate (PNI 40-49%) or high risk (PNI >50%) of developing postoperative complications. When this PNI was used on 100 surgical patients (Buzby, 1980) from the intermediate and high risk groups it correctly predicted 92% of the major complications and 93% of the deaths that occurred. A modified PNI was developed by Simms et al (1982) using total iron binding capacity instead of skin sensitivity in his formula. In a group of 122 patients the 'Simms' modified PNI detected 70% of patients who developed postoperative complications. Those patients with a PNI greater than 50 were classified as being at 'risk' of developing nutritional associated complications. This PNI was compared with dynamometry in a study of 70 patients (Griffith and Clark, 1984) and PNI and dynamometry were able to identify patients

at risk of developing major complications or death. There was no correlation with either variable and minor complication rates. One criticism of this study is the definition of major complication, as a hospital stay exceeding two weeks. Hospital stay may be prolonged on social rather than medical grounds.

The PNI has been severely criticised since it is more likely to reflect severity of illness than nutritional status. The development of complications may be unrelated to nutritional status but due to the progression of disease or surgical technique. The use of PNI, single objective nutritional measurements and a Subjective Global Assessment (medical history, physical examination) were compared as predictors of outcome in 60 patients (Detsky et al, 1984), and subdivided into low and high risk groups. The subjective global assessment was found to be the most sensitive and specific predictor of complications in these patients.

The isolation of the nutritional 'component' and its effect on complication rate in surgical patients has not been completely defined. To do so we must not only consider the nutritionally related indices but also the disease state and technical assessment of the surgical procedure.

vi. ESTIMATION OF RESTING ENERGY EXPENDITURE (REE) BY ANTHROPOMETRY

Measurement of REE may be obtained by calorimetric methods. Direct calorimetry is a measurement of heat dissipated by the body and involves the isolation of the subject in a whole body calorimeter for periods greater than 24 hours. Indirect calorimetry measures the heat loss produced by the oxidative process measured by the subjects oxygen consumption and carbon dioxide production (Frayn, 1983).

Indirect calorimeters may be mobile when the subject is required to wear face mask and nose clip or static when the patient's head is enclosed in a fixed canopy. Measurements may be taken over much shorter time periods using indirect calorimetry and results extrapolated to give REE for 24 hours.

Resting energy expenditure is closely related to FFM (Miller and Blyth, 1952) and consequently predictions of REE may be derived from the anthropometric data of height, weight and body surface area which accurately predict the size and activity of the FFM. Various predictive formulae using the variables age, sex, height and weight are used to estimate REE and include those of Harris-Benedict (1919), Kleiber (1932), Robertson-Reid (1952) and Fleisch tables (1951). The Harris-Benedict and Robertson-Reid formulae were derived from indirect calorimetry measurements of REE in 239 and 2310 healthy individuals respectively. The Kleiber formula has been derived from the original Harris-Benedict data and the Fleisch tables were generated from a review of 24 studies involving REE measurements. All these studies were carried out mainly on young healthy subjects who were presumed to have normal body composition. In various disease states, alterations in body composition may occur and this may lead to inaccuracies in the prediction of REE from formulae and tables. Recent studies (Feurer et al, 1984; Roza and Shizgal, 1984) have shown that use of predictive formulae and tables have limitations in the prediction of REE in a hospitalised population. Feurer and co-workers (1984) studied 200 (100 male, 100 female) weight stable patients and 72 controls using a mobile indirect calorimeter and the Harris-Benedict and Kleiber formulae. They reported that REE as predicted by Harris-Benedict and Kleiber

formulae was greater than that measured by indirect calorimetry in both controls and patient groups. The Harris-Benedict formula over or underestimated (± 10%) REE in 20% of controls and 40% of patients. The Kleiber formula was inappropriate in 33% of controls and 46% of patients. These percentages are considerably greater than the 12% and 8% of normal individuals who fall outwith + 10% measured REE reported by Harris-Benedict (1936) and Boothby et al (1922, 1936) respectively.

Roza and Shizgal (1984) studied 33 normally nourished patients and 41 malnourished patients and found the Harris-Benedict formula unreliable in predicting REE in the malnourished group. Using multiple isotope dilution method they demonstrated a decreased body cell mass and increased extracellular mass in the malnourished group and concluded that the inaccuracy of the Harris-Benedict formula in these malnourished patients was due to altered body composition.

All these predictive formulae are based on the assumption that height and weight reflect the size of the metabolically active portion of the body which is known to reflect energy expenditure. It could therefore be postulated that the anthropometric measurement AMC could be used as a predictor of REE. Voluntary muscle is not involved in the changes in extracellular fluid volume that occurs during nutritional depletion and repletion (Starker et al, 1983). Unlike body weight which is often altered in various disease states, AMC is less prone to oedema and is a site readily accessible in the hospitalised patient. Brown and colleagues (1984) considered the use of AMC as a method of predicting whole body oxygen consumption in 42 control, 24 weight losing and 16 patients with sepsis. There was a strong correlation with AMC and oxygen consumption in the control and

weight losing groups. A correlation with AMC and REE also existed but it was not as close as that of oxygen consumption. The authors suggest that the relationship between AMC and oxygen consumption may be of value in determining the patient's degree of catabolism. There was a poor correlation of AMC with oxygen consumption in the septic group of patients.

vii. AIMS OF DISSERTATION

The aims of the studies described in this dissertation were:

(1) to examine prospectively AMC as a predictor of REE in weight stable and weight losing patients with or without malignant disease. Derived regression equations would be examined in a group of weight stable cardiac patients whose energy expenditure would be measured by indirect calorimetry.

(2) to use anthropometric and biochemical nutritional indices to determine preoperative and postoperative nutritional status. The effect of nutritional status on postoperative outcome would be considered in a group of patients undergoing major elective surgery.

CHAPTER 2 THE ESTIMATION OF RESTING ENERGY EXPENDITURE BY ANTHROPOMETRY

i. Introduction

ii. Patients and methods

iii. Results

iv. Discussion

i. INTRODUCTION

Resting energy expenditure can be measured using calorimetric techniques or by the stable isotope doubly labelled water. Direct calorimetric facilities are available only in a few research centres. The use of doubly labelled water is expensive, requires specialist equipment and expertise for analysis, and is not suitable for short-term studies. Body composition may be altered in various disease states (Shizgal, 1981) with consequent changes in energy expenditure. Indirect calorimetry using either fixed or mobile apparatus provides a method of measuring REE in hospitalised patients. However, many ill patients are unable to tolerate the nose clips or head canopies that are used in indirect calorimetric techniques.

Resting energy expenditure can also be estimated using various conventional predictive formulae (Harris-Benedict, 1919; Kleiber, 1932; Robertson Reid, 1952; Fleisch Tables, 1951) all of which were derived from indirect calorimetric studies on normal healthy subjects. When applied to a hospitalised population, these formulae have been shown to be inaccurate (Roza and Shizgal, 1984; Feurer et al, 1984). Moreover, the weighing of ill, bed-bound patients is often impractical making the use of predictive formulae to estimate energy expenditure impossible. A simpler method of estimating REE would therefore be of value in determining patients metabolic state and to facilitate the prescription of adequate nutritional support.

Anthropometry provides a simple, quick and cost effective method of assessing patient's nutritional status and as previously described (page 1) may permit an estimation of REE from the metabolically active portion of the body. The measurements TSF, MUAC and AMC were therefore be considered in the estimation of REE in

several groups of patients. Patients were classified as weight stable and weight losing with or without malignant disease as the presence of weight loss or malignancy may affect REE.

ii. PATIENTS AND METHODS

Patients were admitted to the University Department of Surgery for investigation or elective surgery. One hundred and forty two patients were included in the study (Table 2.1 : Appendix Chapter 2). Excluded from the study were those with clinical or bacteriological evidence of infection, ascites, oedema or those who had undergone surgery, radiotherapy or chemotherapy in the preceeding year.

Patients were weighed in hospital clothes without shoes on beam balance standing scales (Weylux 424, UK). Those patients who had lost 10% or more of their pre-illness weight were classified as weight losing. Whilst this is a subjective method of assessing weight change, it was considered more appropriate than the use of standard weight for height tables.

Resting energy expenditure was measured using a rigid canopy indirect calorimeter (Kinney et al, 1964; Fig. 2.1). All patients underwent a 30 minute acclimatization run followed by 40 minutes calorimetry. The rigid canopy is placed around the patient's head and sealed around the neck using a flexible collar. Room air of a known oxygen and carbon dioxide concentration is delivered to the patient at a controlled flow rate. A mixture of this room air and the patient's expired air is then extracted from the canopy at a controlled rate and dehumidified. The oxygen concentration is measured by a dual cell pragmatic oxygen analyser (Servomex Ltd., Crowborough, Sussex, UK) and a single channel infra-red carbon

TABLE 2.1 Pathological diagnoses in weight stable and weight losing cancer and controls

Diagnosis	Cancer	
	Weight stable (n = 54)	Weight losing (n = 44)
Colorectal cancer	36	20
Gastric cancer	14	15
Bronchial cancer	2	5
Oesophageal cancer	2	2
Pancreatic cancer	-	2

	Control	
	Weight stable (n = 27)	Weight losing (n = 17)
Gastric ulceration	1	1
Duodenal ulceration	3	5
Pyloric stenosis	-	2
Cholelithiasis	15	3
Diverticular disease	3	2
Benign colorectal polyp	3	2
Ulcerative colitis	1	-
Crohn's disease	-	1
Hiatus hernia	1	1

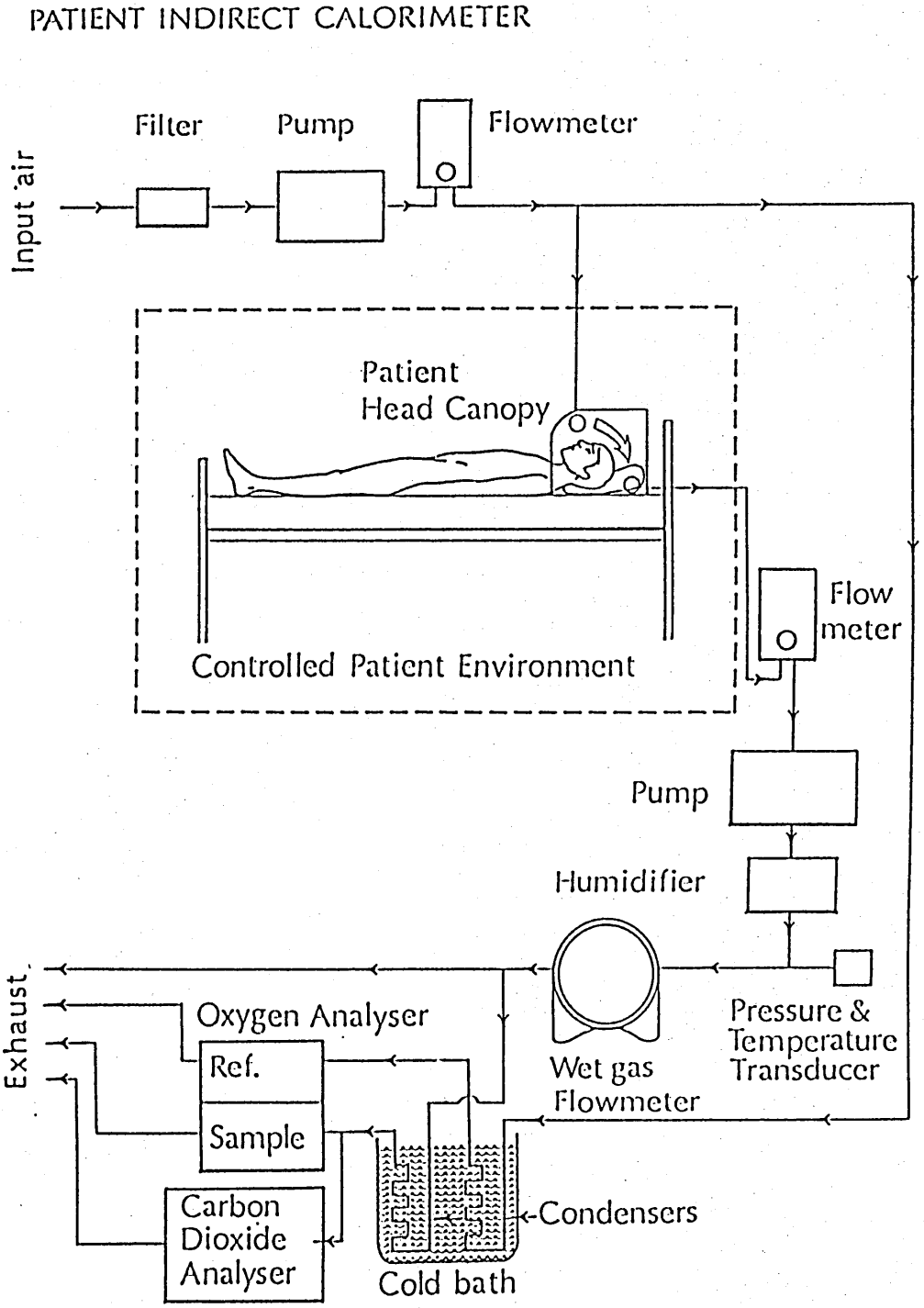


FIGURE 2.1 Schematic representation of the gas circuitry of the indirect calorimeter.

dioxide analyser (Sieger Ltd., Poole, Dorset, UK) is used to measure carbon dioxide concentration. Oxygen and carbon dioxide concentrations in room air are compared with the oxygen and carbon dioxide content of mixed air which has been corrected for barometric pressure and temperature, and from which the oxygen consumption and carbon dioxide production of the patient can then be derived.

The calorimeter is regularly calibrated using 0.8% carbon dioxide and air at a known barometric pressure. Periodically butane gas was burned in the canopy to check the accuracy and sensitivity of the calorimeter. The indirect calorimeter had an overall error in the measurement of oxygen consumption and carbon dioxide production of less than $\pm$ 5%.

On the morning of calorimetry, patients were required to remain resting in bed. For twelve hours prior to calorimetry patients received nothing orally while hydration was maintained by an infusion of 5% dextrose peripherally (960 ml/24 hr). Each study began at approximately 09.00 when patients were transferred in their hospital bed to the calorimeter, at which time the calorimetry procedure was carefully explained to each patient.

Once installed in the head canopy, measurements of oxygen consumption and carbon dioxide production were made and recorded every 30 seconds. Results were recorded and processed by the 'on line' microcomputer (Apple IIIe, California, USA).

After the acclimatisation period, the 80 estimates of oxygen consumption and carbon dioxide production recorded and collected by the microcomputer were converted to mean energy production (watts) using the abbreviated Weir formula (1949):

REE (kcal/day) = (3.9 VO_2 + 1.1 VCO_2) 1440 min/day

where kcal/day = watts x 20.65

VO_2 = oxygen consumption (l/min)

VCO_2 = carbon dioxide (l/min)

In addition FFM was measured using an isotope dilution technique to measure total body water. On the morning of calorimetry the patient was injected with tritiated saline (4 m Bq) and serum samples were obtained 3 and 4 hourly following injection. During the period of equilibrium all urine passed was collected to measure the excretion of tritium in the urine. Fat free mass was derived from total body water assuming that FFM contains 73% water (Moore, 1984).

The anthropometric measurements TSF, MUAC and AMC were performed by the same observer on the day of calorimetry. The patients non-dominant arm was used and the mid-point between the acromion and olecranon processes was marked. Triceps skinfold thickness was measured using the thumb and first finger to lift a skinfold approximately 1 cm above the mid-point mark. Using Harpenden skinfold calipers (British Indicators, UK) skinfold readings were taken 3 times and the average taken as the absolute value. Mid-upper arm circumference was measured using a flexible measuring tape at the mid-point mark. From measurements of TSF and MUAC the AMC is derived using the formula:

AMC = MUAC -(3.14 x TSF)

in which AMC, MUAC and TSF are expressed in cm.

To test whether there were any significant difference between the several groups of data an analysis of variance was used. The Mann Whitney U-test was used for pairwise comparisons in order to minimise the risk of detecting differences in data which were not

normally distributed. Linear regression analysis was performed using the method of least squares and correlation coefficients (r) determined. Linear regression equations are in the form:

$$y = a + bx$$

in which a is the intercept on the y axis and b is the gradient of the line. Any statistical significance between regression line slope was assessed using a t-test for paired data. All confidence intervals (CI) quoted are 95% CI. Significant differences occurred when the probability of these arising by random sampling error was less than 1 in 20 ($p<0.05$) and highly significant when this probability was less than 1 in 100 ($p<0.01$).

iii. RESULTS

There was a predominance of male patients in the weight stable cancer group and females predominated in the weight stable control group (Table 2.2). All groups were well matched with respect to age and height. Both weight losing groups had lost in excess of 15% of their pre-illness weight and had significantly lower mean body weights when compared with the weight stable counterparts. Fat free mass was significantly lower in the weight losing cancer group when compared with the weight stable cancer group. However, there was no significant difference in FFM between the control groups.

The weight losing groups irrespective of disease state had lower TSF, MUAC and AMC when compared with their weight stable counterparts (Table 2.3). The weight losing cancer patients had significantly lower REE and oxygen consumption (uncorrected for body size) than their weight stable counterparts. There was no significant difference in either REE or oxygen consumption

TABLE 2.2 Clinical details of weight stable and weight losing cancer patients and controls

	Cancer		Control	
	Weight stable	Weight losing	Weight stable	Weight losing
n	54	44	27	17
Male : Female	38:16	20:24	7:20	8:9
Age (years)	66±1.5	66±1.6	62±2.7	64±3.8
Height (cm)	164±1.3	161±1.3	159±1.6	162±2.6
Body weight (kg)	64.2±1.6[b]	52.2±1.8[a]	66±2.9[b]	55.4±3.2
Fat free mass (kg)	49.8±1.7	43.6±1.6[a]	48.9±2.2	45.9±2.3
Weight loss (%)	4±0.5	18±1.1[a]	1±0.5[c]	16±1.2[a]

mean ± sem

a = $p<0.01$ versus weight stable cancer patients and weight stable controls

b = $p<0.05$ versus weight losing controls

c = $p<0.05$ versus weight stable cancer patients

TABLE 2.3 Clinical details of weight stable and weight losing cancer patients and controls

		Cancer		Control	
		Weight stable	Weight losing	Weight stable	Weight losing
TSF	(mm)	13.9±0.9	10.1±0.7	19.3±1.7[a]	11.1±1.2
MUAC	(cm)	28.2±0.5	24.2±0.5	29.9±0.9	24.9±0.9
AMC	(cm)	23.8±0.4	21.1±0.4	23.8±0.5	21.4±0.8
REE	(kcal/day)	1426±25.6	1278±37.8[a]	1340±42.5	1279±58.0[a]
VO_2	(ml/min)	206.7±3.53	187.3±5.27[a]	193.9±5.68[a]	184±8.09[a]

mean ± sem a = $p<0.01$

(uncorrected for body size) between the two control groups (Table 2.3).

There was a significant correlation between REE and AMC in all male and female patients (Fig. 2.2). The correlation between REE and AMC remained significant when patients were grouped according to disease and weight status (Fig. 2.3). The slope of the regression line for the cancer weight losing group was significantly different from that of the cancer weight stable group. When patients were subdivided into cancer and control groups, the correlation between REE and AMC remained significant. However, there was no difference in the slope of the regression lines (Fig. 2.4). When all weight losing patients were compared with all weight stable patients the correlation between REE and AMC persisted, but there was a significant difference in the slope of the regression lines (Fig. 2.5).

The correlation between MUAC and REE was also found to be significant and the slopes of the regression lines similar to those found with AMC and REE. However, the correlation with MUAC was not as close as that found with AMC and REE (Fig. 2.6).

Arm muscle circumference also correlated significantly with oxygen consumption but this correlation was not as close as that between AMC and REE. The correlations between AMC and oxygen consumption were:

cancer weight stable : $r = 0.437$, 95% CI (1.6, 6.0)
$p<0.01$

cancer weight losing : $r = 0.684$, 95% CI (6.0, 11.8)
$p<0.01$

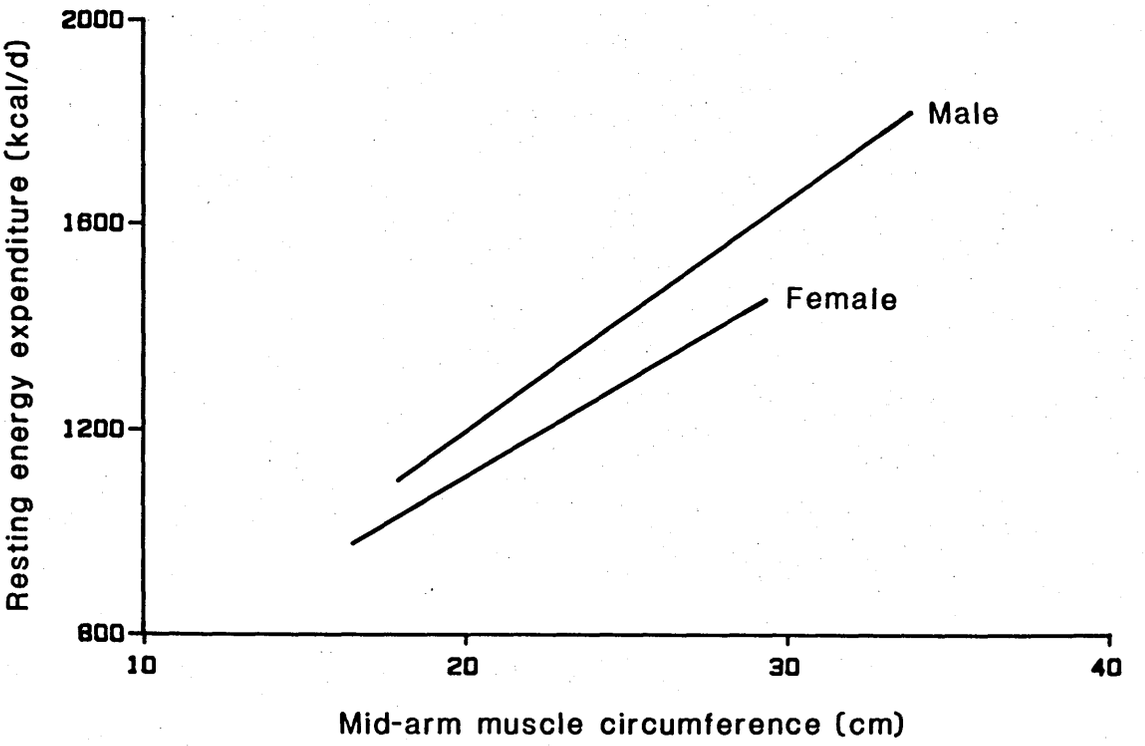


FIGURE 2.2 The relationship between resting energy expenditure and arm muscle circumference in all male and all female patients.

Male
n=73; r=0.583; p<0.01 y=393 + 45.0x
95% CI (30.0, 59.9)

Female
n=69; r=0.634; p<0.01 y=381 + 39.0x
95% CI (27.4, 50.6)

No significant differences between the slopes

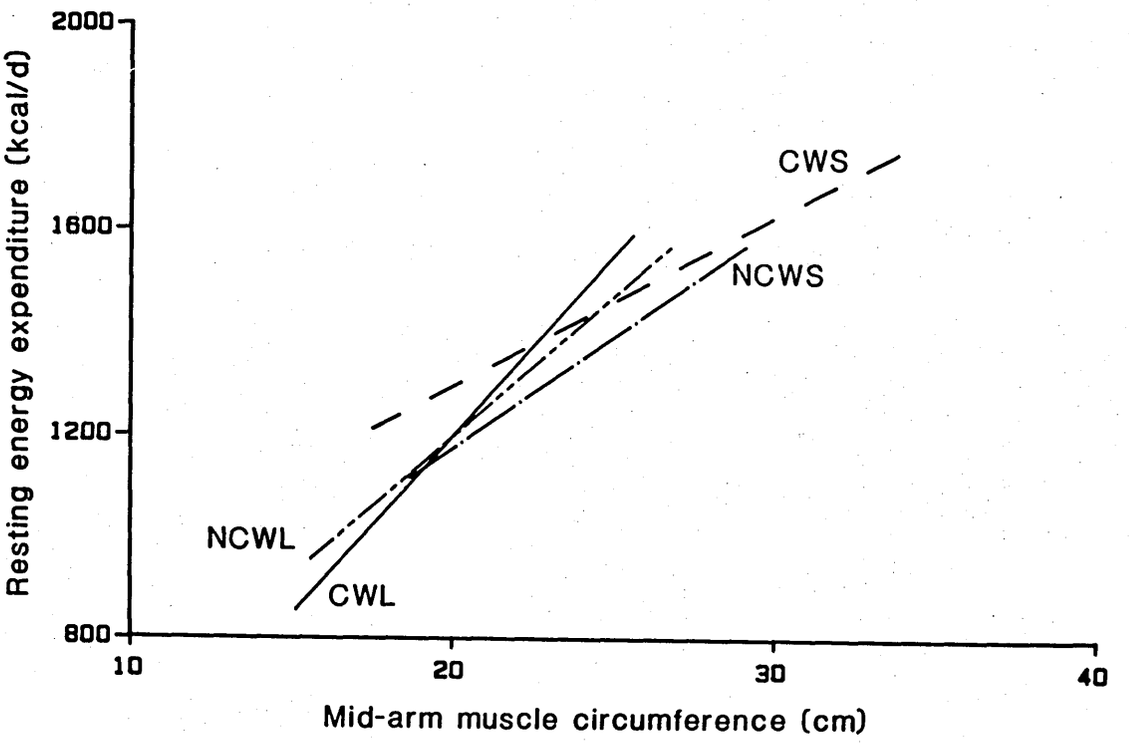


FIGURE 2.3 The relationship between resting energy expenditure and arm muscle circumference in each of the groups.

CWL (cancer weight losing)
n=44; r=0.751; p<0.01 y=213 + 70.5x
95% CI (51.4, 89.6)

CWS (cancer weight stable)
n=54; r=0.511; p<0.01 y=628 + 33.3x
95% CI (17.8, 48.8)

NCWL (control weight losing)
n=17; r=0.728; p<0.01 y=89 + 55.6x
95% CI (28.6, 82.6)

NCWS (control weight stable)
n=27; r=0.539; p<0.01 y=304 + 43.5x
95% CI (16.4, 70.6)

The CWL slope is significantly different from the CWS slope (p<0.01)

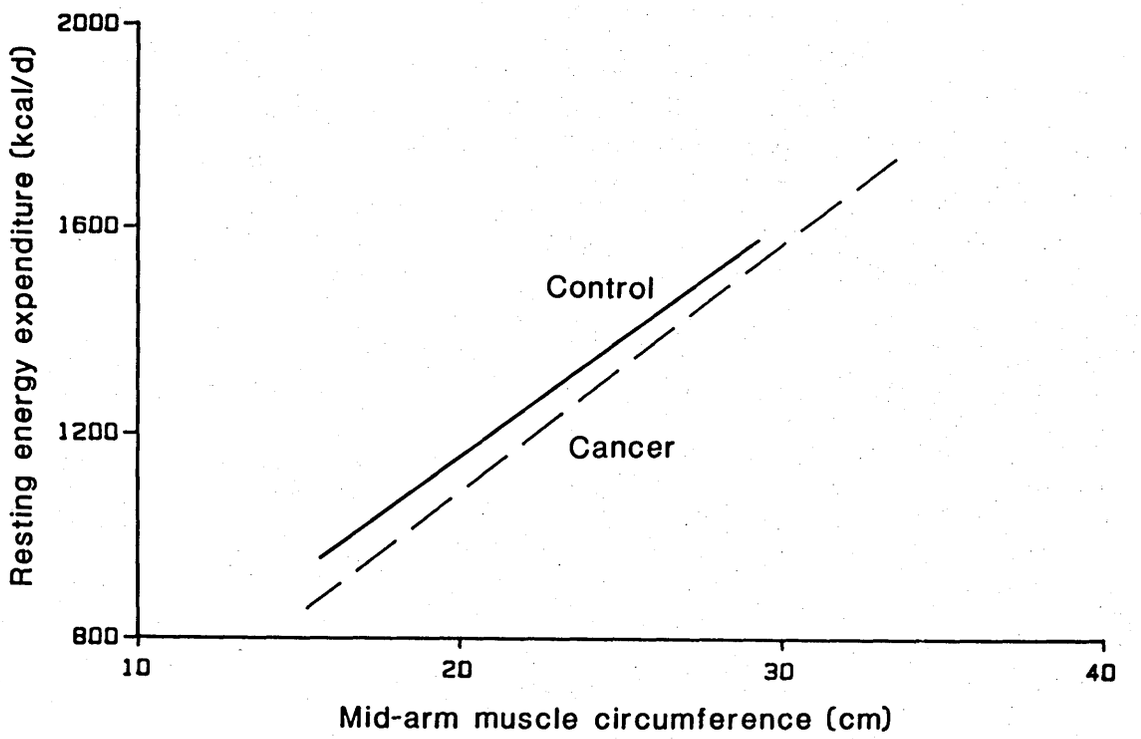


FIGURE 2.4 The relationship between resting energy expenditure and arm muscle circumference in all cancer and all controls.

Cancer
n=98; r=0.670; p<0.01 y=240 + 49.3x
95% CI (38.2, 60.4)

Control
n=44; r=0.619; p<0.01 y=277 + 45.4x
95% CI (27.6, 63.2)

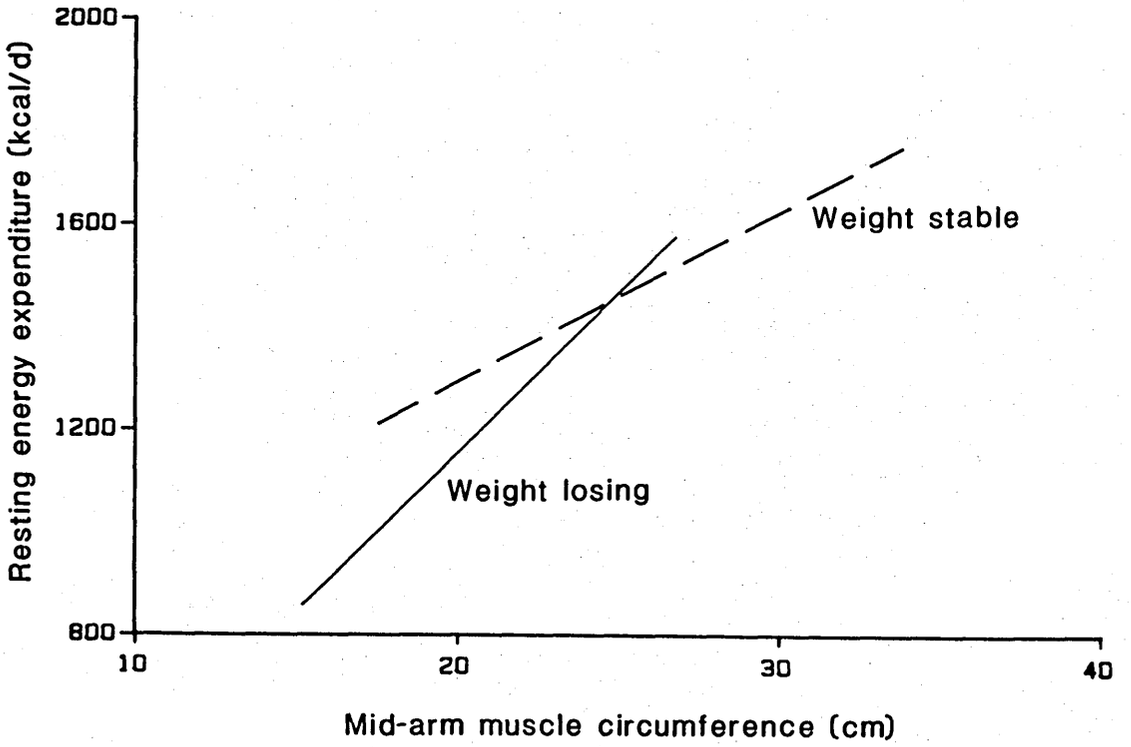


FIGURE 2.5 The relationship between resting energy expenditure and arm muscle circumference in all weight stable patients and all weight losing patients.

Weight stable
n=81; r=0.513; p<0.01 y=518 + 36.8x
95% CI (23.0, 50.6)

Weight losing
n=61; r=0.741; p<0.01 y=-108 + 65.3x
95% CI (49.9, 80.7)

The weight stable slope is significantly different from the weight losing slope (p<0.01)

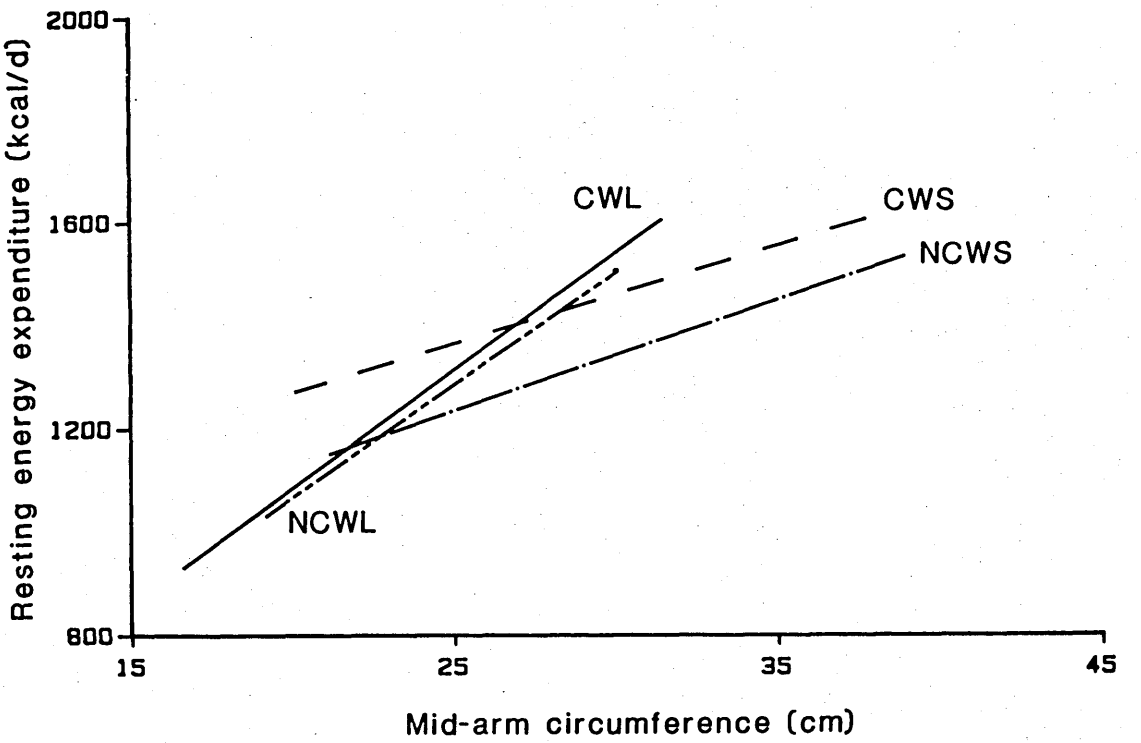


FIGURE 2.6 The relationship between resting energy expenditure and midarm circumference in each of the groups.

CWL (cancer weight losing)
n=44; r=0.659; p<0.01 y=178 + 45.4x
95% CI (29.4, 61.4)

CWS (cancer weight stable)
n=54; r=0.352; p<0.01 y=890 + 19.0x
95% CI (5.0, 33.0)

NCWL (control weight losing)
n=17; r=0.667; p<0.01 y=205 + 43.1x
95% CI (18.2, 68.0)

NCWS (control weight stable)
n=27; r=0.444; p<0.05 y=698 + 21.4x
95% CI (4.1, 38.7)

The CWL slope is significantly different from the CWS slope (p<0.05)

control weight stable: r = 0.486, 95% CI (1.4, 9.0)

$p<0.05$

control weight losing: r = 0.726, 95% CI (3.9, 11.5)

$p<0.01$

These correlations were less significant than those found with REE and AMC.

Resting energy expenditure correlated significantly with body weight and FFM for each of the four groups (Table 2.4). There was no correlation between TSF and REE or oxygen consumption in any of the groups.

iv. DISCUSSION

As skeletal muscle mass has been related to basal heat production (Krebs, 1950) it would therefore seem appropriate to consider AMC in the estimation of heat production or REE. Skeletal muscle is also unaffected by the changes in extracellular fluid volume found during nutrient deprivation (Starker et al, 1983). The arm is easily accessible in almost all patients and provides a simple, quick, non-invasive method of estimating skeletal muscle mass. Unlike other anthropometric sites, the arm is less susceptible to oedema. In an earlier study, Brown and colleagues (1984) showed that there was a stronger correlation between oxygen consumption and REE than between AMC and REE measured by indirect calorimetry. Meanwhile, Harris and co-workers (1984) demonstrated that changes in body weight correlated more with MUAC than AMC. Perhaps this may be due to the inherent errors associated with measurement of TSF, found particularly in the obese population.

An interesting observation of the present study was the

TABLE 2.4 Correlation between resting energy expenditure (REE) with body weight and fat free mass in weight stable and weight losing cancer patients and controls

	Cancer		Control	
REE related to:	Weight stable	Weight losing	Weight stable	Weight losing
Body weight	0.646 (6.9,13.5)	0.804 (15.3,19.3)	0.750 (7.2,15.0)	0.772 (8.1,19.9)
Fat free mass	0.559 (5.1,13.1)	0.845 (17.4,26.4)	0.874 (13.5,20.9)	0.887 (16.4,28.6)

correlation coefficient (95% confidence interval) $p<0.01$ for all values

different relationship demonstrated between REE and AMC in the weight stable and the weight losing group. The significantly steeper slope of the regression line found in the weight losing group suggests that at a low AMC, the REE will be less than that in the weight stable population (Fig. 2.5). This indicates a degree of adaptation of the weight losing group which would lead to a slowing down of metabolic rate with weight loss. In both the weight losing groups the correlation coefficient was greater than in the weight stable counterparts. This may be accounted for by the increased error associated with TSF measurement in a weight stable population (Bray et al, 1978).

The presence of malignant disease appeared not to affect the relationship of AMC with REE (Fig. 2.4). This finding suggests that skeletal muscle mass may not be responsible for any changes seen in REE with malignancy. The effect of tumour growth on host metabolism is controversial. Several groups report increased REE with neoplastic disease (Waterhouse et al, 1951; Fenninger and Mider, 1954; Bozzetti et al, 1980). Conversely others have suggested that cancer patients adapt their energy expenditure to a weight losing state (Lindmark et al, 1984; Hansell et al, 1986) and the presence of hepatic metastases was shown by Hansell and co-workers (1986) to have no significant effect on REE. The present study indicates that formulae to predict REE do not require to make allowance for underlying neoplastic disease.

On the other hand, in this study no patient was stressed or septic, conditions which have been shown to affect the relationship between REE and AMC. Brown et al (1984) reported that in sepsis the correlation with AMC and VO_2 became less significant which suggests

that modifications of the regression equation may be required when considering the early postoperative stressed or septic patient.

Arm muscle circumference has therefore been shown to correlate with REE and equations have been derived which provide a useful method of estimating REE in the hospitalised patient. Since the correlation coefficient varied between 0.51 and 0.74 (Fig. 2.4 and 2.5) only 26-56% of the REE can be considered to be related to the AMC. This will be partly due to the variations of measurement of AMC and REE and partly due to other aspects of clinical and nutritional conditions also having an effect. Weight status affected the relationship between REE and AMC, while the presence of malignancy did not. The techniques and equipment used in anthropometry are both simple and cost effective, and being non-invasive have a high level of patient acceptability.

CHAPTER 3 THE USE OF PREDICTIVE FORMULAE TO ESTIMATE RESTING ENERGY EXPENDITURE IN AN OVERWEIGHT HOSPITAL POPULATION

i. Introduction

ii. Patients and methods

iii. Results

iv. Discussion

i. INTRODUCTION

The heterogeneous patient population studied in Chapter 2 showed that AMC correlated significantly with REE as measured by indirect calorimetry. If validated, the clinical and research implications of these findings are considerable. A simple and readily available technique such as AMC would permit the estimation of REE in any hospital, general or specialist, and thus facilitate the provision of appropriate nutritional support.

A relatively homogeneous patient group comprising 13 weight stable males with benign disease aged 47-61 years, was used to investigate prospectively the non-cancer weight stable (NCWS) regression equation derived from the study in Chapter 2. Various other conventional predictive formulae (Harris-Benedict, 1919; Kleiber 1932; Robertson Reid, 1952; Fleisch Tables 1951) used to estimate REE were also considered in this study and compared with REE as measured by indirect calorimetry. These conventional predictive formulae were derived from indirect calorimetric studies performed on groups of normal healthy individuals who were presumed to have normal body composition. The Harris-Benedict (1919) formula was derived from indirect calorimetric measurements performed on 239 subjects,while the much larger study of Robertson-Reid (1952) examined indirect calorimetry measurements of 2310 individuals. The Kleiber formula was derived from the original work of Harris-Benedict (1919), and the Fleisch Tables (1951) were generated from a review of 24 studies involving REE measurements.

ii. PATIENTS AND METHODS

All patients were admitted to the University Department of

Cardiac Surgery, Glasgow Royal Infirmary to undergo surgery for coronary artery disease. Thirteen male patients aged 47-61 years were included in the study. Anthropometric and indirect calorimetry were performed preoperatively using the methods previously described in Chapter 2.

Resting energy expenditure was also predicted in five ways:

1. Harris-Benedict formula (kcal/day)

   Males: $66.473 + 13.752\ (wt) + 5.003\ (ht) - 6.755\ (A)$

2. Robertson Reid formula ($kcal/m^2/hr$)

   Males: $37.405365 - 0.06944\ (A)$

3. Kleiber formula (kcal/day)

   Males: $71.2\ wt^{0.75}\ (1 + 0.004\ (30-A) + 0.010\ (ht/wt^{0.33}) - 43.4)$

4. Fleisch Tables ($kcal/m^2/hr$)

5. NCWS regression equation (kcal/day)

   Males or Females: 304 + 43.5 AMC (Fig. 2.3)

where

A = age in years

Wt = body weight in kilograms

Ht = height in centimetres

AMC = arm muscle circumference in centimetres

Predictive formulae were compared with REE derived from indirect calorimetry using a Wilcoxon Signed Rank test. Linear regression analysis was performed using the method of least squares and correlation coefficients (r) were determined. Linear regression equations are in the form:

$$y = a + bx$$

where a is the intercept on the y axis and b is the gradient of the line. Any statistical significance between regression line slopes

was assessed using a t-test for paired data. All confidence intervals (CI) quoted are 95% CI. Significant differences occurred when the probability of these arising by random sampling error was less than 1 in 20 ($p<0.05$) and highly significant when this probability was less than 1 in 100 ($p<0.01$).

iii. RESULTS

Patients were studied prior to surgery and all underwent a standard coronary artery bypass procedure. Anthropometric data are shown in Table 3.1. The patients had a mean body weight of 113% of ideal (Metropolitan Life Insurance Tables, 1959), and a mean TSF and AMC of 93% and 109% of standard (Jelliffe, 1966) respectively.

Mean REE was 1282 $\pm$ 337 kcals/day when measured by indirect calorimetry and was significantly lower than REE calculated by NCWS (1503 $\pm$ 97 kcals/day; $p<0.05$); Harris-Benedict (1582 $\pm$ 195 kcals/day; $p<0.05$); Kleiber (1589 $\pm$ 180 kcals/day; $p<0.05$); Robertson-Reid (1508 $\pm$ 139 kcals/day; $p<0.05$) and Fleisch Tables (1580 $\pm$ 150 kcals/day; $p<0.05$: Table 3.2).

Resting energy expenditure measured by indirect calorimetry correlated significantly with REE calculated by the Harris-Benedict formula ($r = 0.575$, $p<0.05$; Fig. 3.1) and Kleiber formula ($r = 0.579$, $p<0.05$; Fig. 3.2) and showed poor correlation with Fleisch Tables ($r = 0.535$, NS; Fig. 3.3), Robertson Reid formula ($r = 0.490$, NS; Fig. 3.4) and the NCWS equation ($r = 0.279$, NS; Fig. 3.5).

However, the strongest correlation was found between the uncalculated arm anthropometric measurement MUAC and REE measured by indirect calorimetry ($r = 0.675$; $p<0.02$; Fig. 3.6).

TABLE 3.1 Anthropometric Data

Patient	Age	Height (cm)	Weight (kg)	% Ideal Body Weight	TSF	% TSF	AMC	% AMC
1	61	180	87.0	118	8.0	64	24.5	97
2	54	163	58.6	98	7.0	56	25.8	102
3	51	180	100.0	136	14.0	112	30.1	119
4	50	183	84.0	107	12.0	96	29.2	115
5	59	170	76.4	116	15.0	120	27.3	108
6	55	173	82.0	120	13.0	104	30.4	120
7	60	178	71.0	124	13.5	108	28.8	114
8	51	175	76.5	111	12.5	100	27.0	107
9	48	165	64.0	107	9.0	72	27.5	109
10	61	168	71.0	108	8.0	64	30.5	120
11	58	170	80.0	105	14.0	112	28.3	112
12	47	163	57.0	114	10.5	84	25.1	99
13	48	164	60.0	112	14.0	112	23.9	94
mean (± SD)	54± 5.2	171± 7.0	74.4± 12.6	113± 9.7	11.6± 2.7	93± 21.9	27.6± 2.2	109± 6.6

TABLE 3.2 Resting energy expenditure measured by indirect calorimetry and calculated using predictive formulae (kcal/day)

Patient	Indirect Calorimetry	NCWS	Harris-Benedict	Kleiber	Robertson Reid	Fleisch Tables
1	1184	1370	1751	1723	1639	1719
2	1121	1426	1323	1350	1308	1378
3	1821	1613	1997	1972	1775	1874
4	1191	1574	1799	1798	1671	1765
5	1094	1492	1569	1575	1495	1569
6	1416	1626	1688	1688	1572	1655
7	1123	1557	1528	1536	1494	1568
8	1471	1478	1649	1658	1552	1638
9	1179	1500	1448	1470	1387	1467
10	856	1631	1471	1486	1492	1500
11	2077	1535	1625	1627	1527	1604
12	1043	1396	1348	1370	1311	1388
13	1095	1344	1388	1410	1378	1421
mean (± SD)	1282± 337	1503± 97	1583± 195	1589± 180	1508± 139	1580± 150

p<0.05 comparison with indirect calorimetry (Wilcoxon-Signed Rank)

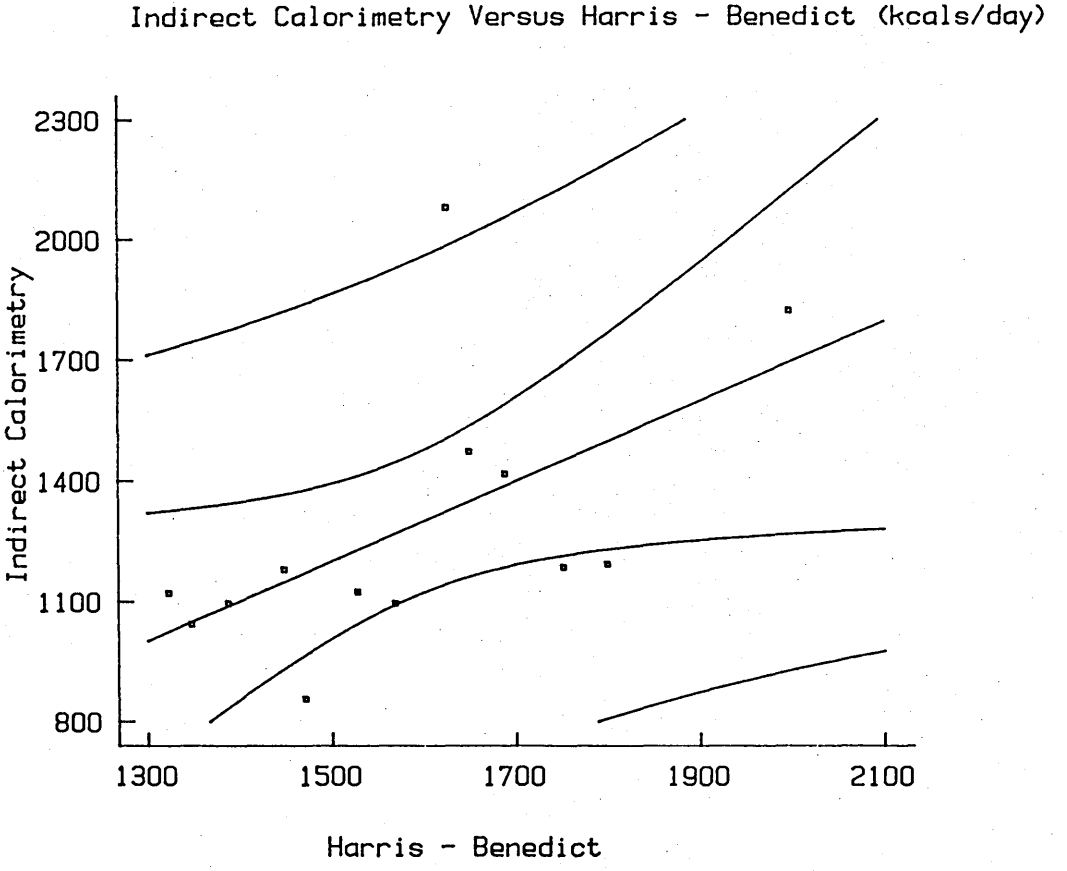


FIGURE 3.1 The relationship between resting energy expenditure measured by indirect calorimetry and the Harris-Benedict formula.

Male n = 13; r = 0.575; p<0.05 y = -288 + 1x
95% CI for slope
(0.06, 1.93)

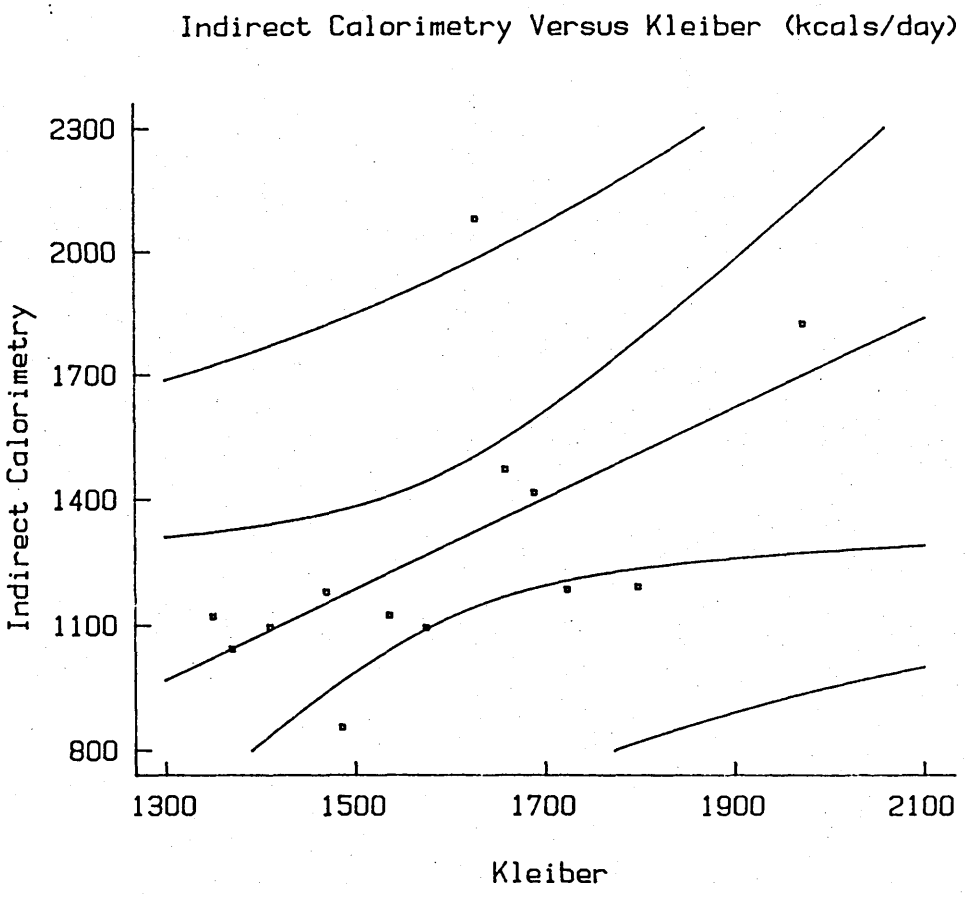


FIGURE 3.2 The relationship between resting energy expenditure measured by indirect calorimetry and the Kleiber formula.

Male n = 13; r = 0.579; p<0.05 y = -422 + 1.1x
95% CI for slope
(0.07, 2.09)

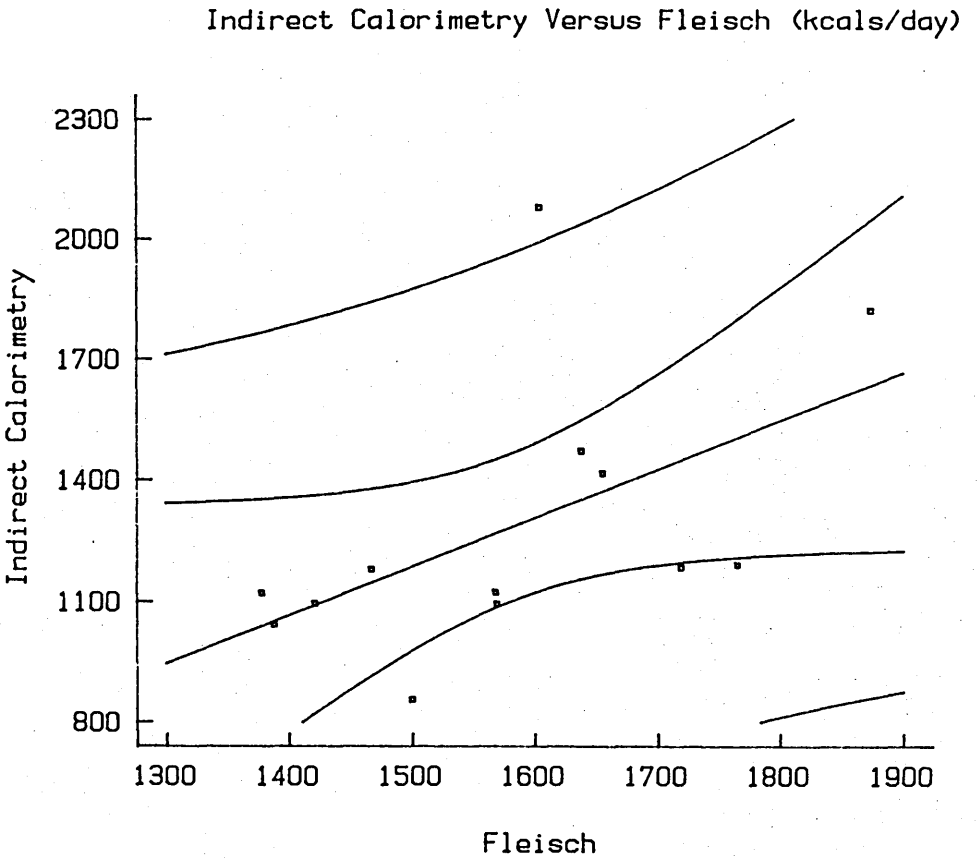


FIGURE 3.3 The relationship between resting energy expenditure measured by indirect calorimetry and Fleisch Tables.

Male n = 13; r = 0.535; NS y = -612 + 1.2x
95% CI for slope (-0.06, 2.46)

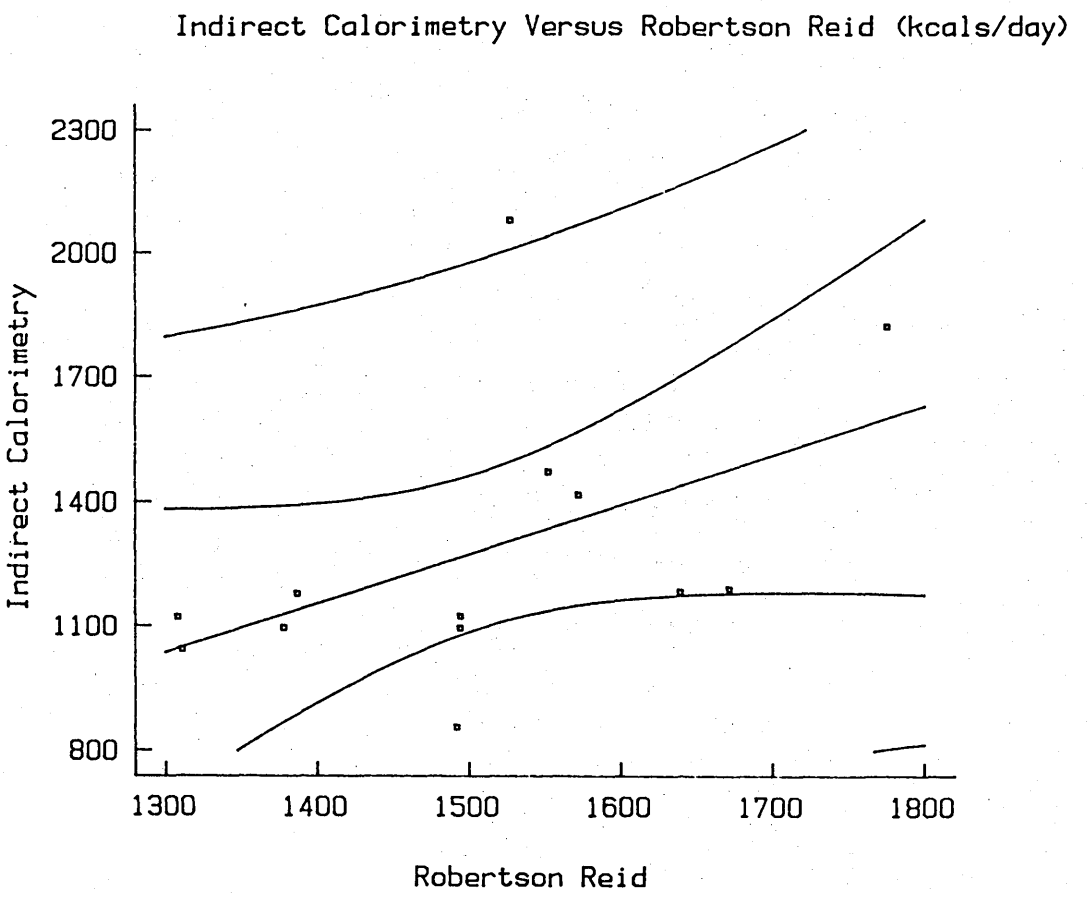


FIGURE 3.4 The relationship between resting energy expenditure measured by indirect calorimetry and Robertson-Reid formula.

Male n = 13; r = 0.490; NS y = -508 + 1.2x
95% CI for slope (-0.22, 2.59)

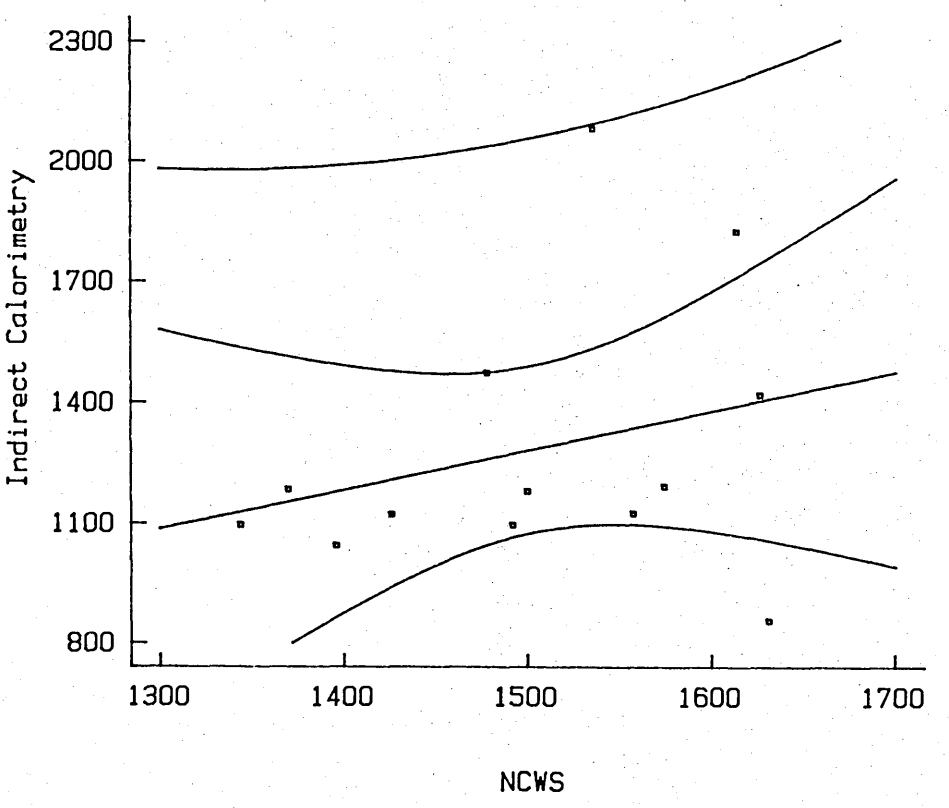


FIGURE 3.5 The relationship between resting energy expenditure measured by indirect calorimetry and the non-cancer weight stable equation.

Male n = 13; r = 0.279; NS y = -172 + 1x
95% CI for slope (-1.24, 3.18)

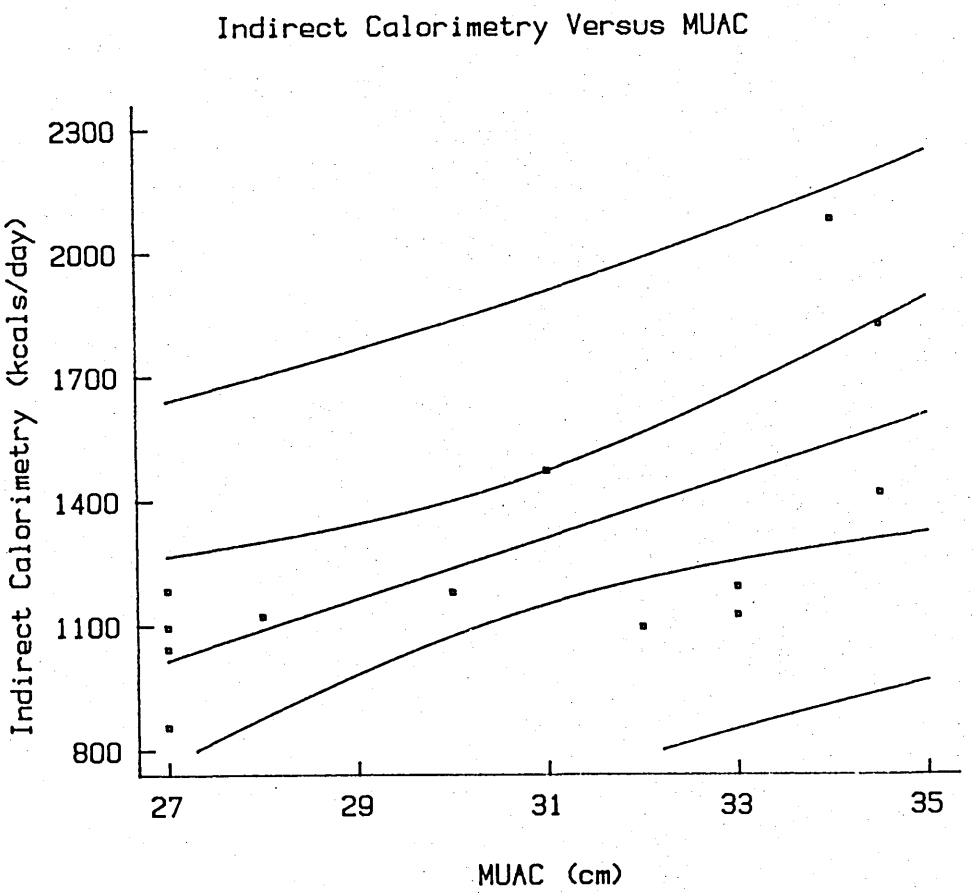


FIGURE 3.6 The relationship between resting energy expenditure measured by indirect calorimetry and mid-upper arm circumference.

Male n = 13; r = 0.675; p<0.02 y = -974 + 74x
95% CI for slope
(21, 127)

iv. DISCUSSION

In attempting to validate the regression equation for energy expenditure using the previously obtained NCWS equation (Fig. 2.6), it was important to consider a relatively homogeneous group of patients. This group was chosen to minimise the influence of variables such as sex, age or diagnosis. For example females have a slightly lower metabolic rate than males (Fleisch, 1951) and as an individual ages, metabolic rate decreases (Keys et al, 1973). The 13 patients included in the study were of ideal or above ideal body weight (Metropolitan Life Insurance Tables 1959) and one patient was obese as assessed by McLaren (1981). Whilst their body weights suggested a high degree of adiposity, this was not reflected in their TSF measurements which were normal. Similarly AMC measurements were normal as defined by Jelliffe's (1966) Standards (Table 3.1).

In Chapter 2 it was noted that AMC correlated significantly with REE measured by indirect calorimetry (Fig. 2.6). However, in the 13 patients currently under study the correlation between AMC and REE by indirect calorimetry was poor ($r = 0.279$; Fig. 3.5). This poor correlation may be explained by the use of AMC in a group of 'overweight' patients as the delineation of the triceps skinfold is technically more difficult in an obese group. In a study of obese patients Bray et al (1978) recommended that skinfold calipers should not be used in the assessment of subcutaneous fat stores, although these same workers did find a good correlation with MUAC and changes in body weight. The work of Bray and colleagues agrees with the findings of Harris et al (1985) who studied arm anthropometry in normal and obese subjects and demonstrated that measurement of MUAC was more reproducible and had a smaller variation than that of TSF or

derived AMC. In the light of these findings a comparison of MUAC with REE was made and a significant correlation was demonstrated between them ($r = 0.675$, $p<0.02$; Fig. 3.6).

The use of other predictive formulae (Harris-Benedict, 1919; Kleiber, 1932; Robertson Reid, 1952; Fleisch 1951) were also considered in the present study. These formulae which presuppose that the height and weight of an individual reflect accurately the metabolic portion of the body, and were derived from indirect calorimetric studies which were performed on normal healthy individuals. The presence of disease or change in body weight may alter body composition (Shizgal, 1981) and subsequently the use of predictive formulae may prove inappropriate in a hospitalised population. All predictive formulae overestimate significantly REE as measured by indirect calorimetry in this patient group (Table 3.2). This overestimation of REE has been reported previously by other workers. Feurer et al (1984) in a study of 200 clinically stable patients and 72 controls reported that REE predicted by the Kleiber and Harris-Benedict formulae was greater than that measured by indirect calorimetry. Daly and colleagues (1985) compared REE which had been derived from the Harris-Benedict formula and from measurements obtained by direct and indirect calorimetry in 127 healthy individuals aged 18-67 years and found that the Harris-Benedict formula overestimated REE by 10-15%. In contrast Roza and Shizgal (1984) demonstrated that the Harris-Benedict equation underestimated REE in a group of malnourished patients. Knox et al (1983) studied 200 malnourished cancer patients and determined REE by indirect calorimetry. Patients were classified as: hyper-, normo- or hypometabolic in order to compare REE measured by indirect

calorimetry and calculated by the Harris-Benedict formula. This latter formula overestimated REE in the hypometabolic group and underestimated REE in the hypermetabolic patients.

A factor which could account for these variations in predictive formulae and indirect calorimetry could be the variety of indirect calorimetric techniques employed. Robertson and Reid (1952) used a Benedict-Roth spirometer which is not portable and where the subject is required to wear a nose clip while breathing through a mouth piece. Daly et al (1985) and Feurer et al (1984) used a mobile indirect calorimeter with mouth piece and nose clip while in the present study a fixed indirect calorimeter with sealed head canopy was used. The studies of Daly et al (1985), Knox et al (1983) and Feurer et al (1984) also examined relatively heterogenous populations with wide ranging ages and pathologies. The malnourished patient may have a reduced body cell mass and increased extracellular mass (Roza and Shizgal, 1984) with unaltered body weight, and this alteration in body composition may invalidate the use of conventional predictive formulae to determine REE. Whilst the use of predictive formulae may be appropriate in some hospital population studies (Feurer, 1980) this is not so when considering REE in the individual patient.

The present study concentrated on a well defined group of patients in order to minimise the influence of the variables previously described. Although the patient group size was small the use of conventional predictive formulae was shown to be inaccurate and consistently overestimated REE (Table 3.2). Resting energy expenditure calculated by the Robertson-Reid (1952), Fleisch Tables (1951) and the NCWS formulae had no significant correlation with REE measured by indirect calorimetry (Fig. 3.3-5). However, REE

calculated using the Harris-Benedict and Kleiber formulae did correlate significantly with REE measured by indirect calorimetry; this relationship was not as strong as the correlation with MUAC. Therefore, in this overweight patient group the use of MUAC represents a more accurate method of predicting REE. The poor correlation of AMC with REE measured by indirect calorimetry may be explained by the inappropriate use of AMC in overweight individuals, where arm anthropometry is technically more difficult.

Results from Chapter 2 and 3 suggest that in the clinically stable hospitalised patient, body morphology has a greater influence on energy expenditure than disease state. This hypothesis should be examined further in larger homogeneous patient groups and tested for its application in septic and other hypercatabolic groups. Once the various predictive formulae using arm anthropometry have been validated, specific formulae may be applied to particular groups or individuals depending on their body morphology and/or disease state.

The determination of REE from arm anthropometry may also be a useful parameter in determining optimum feeding regimens, thus avoiding the adverse clinical complication of hepatic dysfunction associated with overfeeding (Grant et al, 1977) or the provision of an inappropriate energy intake to the starved or catabolic patient.

CHAPTER 4 NUTRITIONAL STATUS AND OUTCOME IN SURGICAL PATIENTS

i. Introduction

ii. Patients and methods

iii. Results

   a. Arm muscle circumference

   b. Triceps skinfold thickness

iv. Discussion

i. INTRODUCTION

The prevalence of malnutrition among surgical patients has been well documented (Bistrian et al, 1974; Hill et al, 1977) and it has been suggested that pre-existing malnutrition results in increased morbidity and mortality postoperatively (Buzby et al, 1980; Mullen et al, 1980; Roy et al, 1985). In a group of surgical patients with malignant disease, Muller et al (1982) demonstrated that a period of preoperative nutritional support reduced the incidence of major complications and mortality following major surgery. However, in this study the patients' preoperative nutritional status was not considered. Smale and co-workers (1981) examined retrospectively the effect of parenteral feeding in patients sub-divided into 'high' and 'low' risk groups as defined by a PNI (Mullen et al, 1980). The results of this uncontrolled study supported the findings of Muller's group by showing that the 'high' risk patients who received preoperative parenteral feeding had reduced morbidity and mortality when compared to patients receiving normal hospital diet.

The metabolic response to surgery and its nutritional consequence may be different depending on the preoperative nutritional status. It is, however, difficult to isolate which abnormalities are due to the effects of malnutrition, disease or to the actual surgical procedure. Objective anthropometric and biochemical measurements have been individually or collectively used in an attempt to identify the patient 'at risk' of developing nutritionally associated complications. Identification of such a group of patients would permit efficient, effective and economic targetting of nutritional support.

The aim of this study was to examine prospectively the effect of major surgery on nutritional status and outcome in a group of patients admitted for elective surgery.

ii. PATIENTS AND METHODS

Forty-eight patients were examined prospectively (see Appendix Chapter 4). All patients studied were admitted to the University Department of Surgery, Glasgow Royal Infirmary. Malignant disease was proven histologically in 24 patients and 24 patients presented with benign disease (Table 4.1). All patients underwent surgery and operative procedures are shown in Table 4.2.

Nutritional assessment by biochemical and anthropometric measurements was performed on the day of admission and at discharge. Anthropometric criteria were used to classify patients as normal or malnourished. Those with an AMC >90% were classified as normal (Group A) and those with AMC <90% as malnourished (Group B). Triceps skinfold thickness was also used to sub-divide patients: a TSF >80% being normal (Group C) or a TSF <80% malnourished (Group D). Other anthropometric measurements carried out included percentage ideal body weight (Metropolitan Life Insurance Tables, 1959), MUAC, hand grip strength and pre-illness weight loss. Patients were weighed on beam balance scales (Weylux 424, UK) in hospital clothes and without shoes. The scales were calibrated regularly using standard weights. A subjective assessment of pre-illness weight loss (PIWL) was also determined using the formula:

$$PIWL = \frac{\text{current weight}}{\text{normal weight}} \times 100$$

TABLE 4.1 Pathological Diagnoses

Benign Disease	
Chronic pancreatitis	8
Peptic ulceration	3
Gastritis	3
Benign biliary stricture	2
Irritable bowel disease	1
Intestinal obstruction	1
Colonic abscess	2
Crohn's disease	1
Ano-rectal fistula	1
Stabbing	1
Hypersplenism	1
Malignant Disease	
Oesophageal carcinoma	1
Pancreatic/cholangiocarcinoma	9
Gastric carcinoma	5
Rectal carcinoma	4
Colonic carcinoma	3
Adrenal carcinoma	1
Hepatic carcinoma	1

TABLE 4.2 Operative Procedures in 48 Patients

Laparotomy only	1
Oesophagectomy	1
Gastrectomy	5
Revisional gastric surgery	7
Pancreatico-biliary surgery	18
Splenectomy	1
Adrenalectomy and nephrectomy	1
Small bowel resection	1
Sigmoid colostomy	3
Colonic resection	2
Reversal of colostomy	1
Abdomino-perineal resection	6
Reconstruction of Hartmann's procedure	1

Arm anthropometry was performed using the method previously described in Chapter 2.

A hand grip dynamometer (Holtain Ltd., UK) was used to assess muscle function. The patient's dominant hand was used and readings taken three times, with a three minute rest period allowed between measurements. No patient suffered from rheumatoid arthritis. The average of these readings was taken as the absolute value and results expressed as a percentage of standard (Klidjian et al, 1980):

male - 40 kg

female - 27.5 kg

Anthropometric measurements were performed by the same observer throughout the study.

Biochemical assessment of nutritional status was by measurement of serum albumin and transferrin. Serum albumin was estimated on a multichannel analyser (SMAC II, Technicon, USA) by the bromocresol green dye binding method (reference range 40-52 g/l). Transferrin was measured using an immuno-turbidometric method on a centrifugal analyser (Encore, Baker, UK : reference range 2-4 g/l). A PNI modified from that of Simms et al (1982) was calculated for each patient using the formula (Garden et al, 1985):

$$PNI = 150 - 1.66\ (Alb) - 0.78\ (TSF) - 0.20\ (TFN)$$

where:

Alb = serum albumin (g/l)

TSF = triceps skinfold thickness (mm)

TFN = serum transferrin (g/l)

Patients at risk of developing postoperative complications could be predicted by their having a PNI greater than 70.

The operating surgeon recorded his assessment of the risk of a

patient developing a major complication on a linear analogue scale. The scale was 100 mm long running from left to right and from low to high risk. Immediately following the operative procedure, a second assessment of risk was made (Fig. 4.1).

The postoperative course of each patient was monitored until death as an in-patient or discharge from the hospital. Complications documented included death, clinical or bacteriological evidence of infection, fistula formation, pneumonia, wound dehiscence, myocardial infarction, respiratory failure, pulmonary embolus, cerebrovascular accident and shock.

The Mann-Whitney U-test was used for the statistical analysis on pairwise comparisons of non-parametric data between groups. The Wilcoxon Signed Rank test was used on non-parametric paired data within groups.

iii. RESULTS

Clinical, anthropometric and biochemical data on admission is shown in Table 4.3. Of the 48 patients (26 male, 22 female; mean age 54 years), 24 had benign disease and 24 malignant disease. Body weight, AMC and grip strength measurements were normal; TSF measurements were low (Fig. 4.2). Serum albumin and transferrin levels were at the lower limits of the reference range. There was a total of ten postoperative complications which included three deaths. An increase in ranking postoperatively was recorded in seven patients, five of whom developed complications.

a. Arm muscle circumference

When AMC was used to classify patients as normally nourished

TABLE 4.3 Clinical, anthropometric and biochemical data on admission

	(mean ± 1 SEM)	reference range
Number of patients	48	
Age (years)	54 ± 1.1	
Male : Female	26 : 22	
Non-Cancer : Cancer	24 : 24	
Ideal body weight (%)	95 ± 2.4	
Triceps Skinfold (%)	74 ± 3.5	
Arm muscle circumference (%)	96 ± 1.6	
Grip Strength (%)	101 ± 31.7	
Albumin (g/l)	38 ± 0.9	40-52
Transferrin (g/l)	2.6 ± 0.08	2-4
Complications	10	
(Deaths)	(3)	

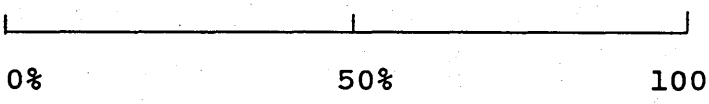
Patient outcome study - risk assessment

Name:

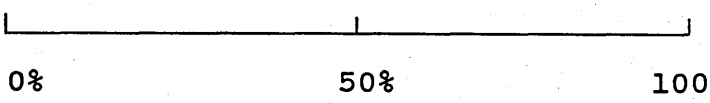
Date of Surgery:

Operation Planned:

Preoperative assessment: Place a cross on the line to indicate your preoperative assessment of the risk of complications in the patient



Postoperative assessment:



Operation performed:

Reason for change:

FIGURE 4.1 Linear analogues representing preoperative and postoperative risk of complications.

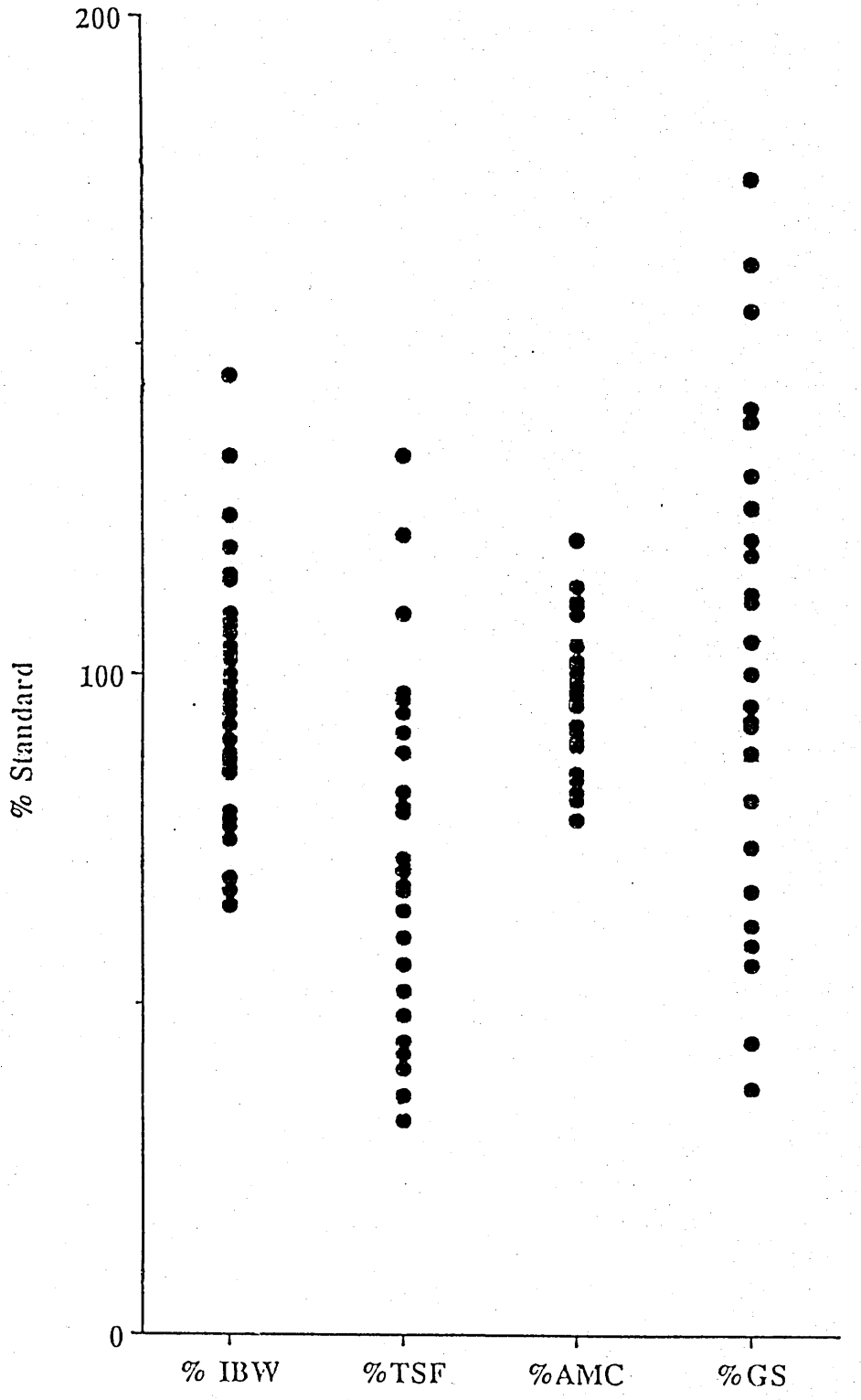


FIGURE 4.2 Anthropometric data on admission

(Group A) or malnourished (Group B), one patient in four was malnourished. Preoperative clinical, anthropometric and biochemical data are shown in Table 4.4. Both groups were well matched for age. Males predominated in Group A while in Group B there was a predominance of females. On admission the patients in Group B had significantly lower body weight, AMC, grip strength, serum albumin and serum transferrin than their normally nourished counterparts (Group A), and in addition had a significantly higher PNI than those in Group A.

The changes found within the groups from admission to discharge were as follows: Group A had significantly lower body weights (100% ± 15, 98% ± 15; $p<0.01$); TSF (70% ± 25, 64% ± 24; $p<0.02$); AMC (100% ± 8, 97% ± 8; $p<0.01$); serum albumin (40 ± 6 g/l, 37 ± 5 g/l; $p<0.001$); serum transferrin (2.7 ± 0.5 g/l, 2.2 ± 0.5 g/l; $p<0.001$) at discharge. Group B had no significant differences in biochemical or anthropometric parameters from admission to discharge (Table 4.5). Malnourished patients (Group B) had a significantly higher PNI (88 cf 76; $p<0.02$) than the normally nourished patients (Group A). Postoperative complications developed in seven patients (out of 36; 19%) in Group A and three (out of 12; 25%) in Group B.

b. Triceps skinfold thickness

When TSF was used to determine nutritional status the normally nourished patients (Group C) accounted for a third of the patient population. Clinical, anthropometric and biochemical data on admission is shown in Table 4.6. Patients were well matched for age. There was a predominance of females in Group C; conversely in Group D males predominated. Patients in Group D had significantly lower

TABLE 4.4 Clinical, anthropometric and biochemical data on admission - nutritional status determined by AMC

	Group A (± SEM)	Group B (± SEM)
Number of patients	36	12
Age (years)	52 ± 2.4	58 ± 5.4
Male : Female	23 : 13	3 : 9
Ideal body weight (%)[1]	99.9 ± 2.6	81.7 ± 3.8
Pre-illness weight loss (%)	8.9 ± 1.6	10.1 ± 2.3
Triceps skinfold thickness (%)	70 ± 4.2	60 ± 8.2
Arm muscle circumference (%)[2]	100 ± 1.3	81 ± 1.1
Grip strength (%)[3]	104 ± 4.9	80 ± 11.9
Serum albumin (g/l)[4]	40 ± 1.0	33 ± 1.2
Serum transferrin (g/l)[5]	2.7 ± 0.1	2.2 ± 0.2
Prognostic nutritional index[6]	76 ± 1.5	88 ± 2.7
Length of hospital stay	22 ± 4.3	26 ± 4.3
Complications (deaths)	7 (2)	3 (1)

Levels of statistical significance (Mann-Whitney U-test)

[1] & [2] $p<0.001$

[3] $p<0.05$

[4] $p<0.005$

[5] $p<0.02$

[6] $p<0.0$

TABLE 4.5 Changes in anthropometric and biochemical data on admission and at discharge - nutritional status determined by AMC (mean + 1SEM)

	Group A (normal)		Group B (malnourished)	
n	36		12	
	Admission	Discharge	Admission	Discharge
Body weight (%)	100±2.6[a]	98±2.8[b]	82±3.7	83±3.5
Triceps skinfold thickness (%)	70±4.2[c]	64±4.2[d]	61±8.2	66±10.1
Arm muscle circumference (%)	100±1.3[e]	97±1.5[f]	81±1.1	81±2.0
Serum albumin (g/l)	40±1.0[g]	37±0.9[h]	33±1.2	33±1.6
Serum transferrin (g/l)	2.7±0.1[i]	2.2±0.1[j]	2.2±0.2	1.9±0.2
Grip strength (kg)	104±5.0	99±5.6	81±13.0	63±10.6

Levels of statistical significance (Wilcoxon-Signed Rank)

g vs h; i vs j	$p<0.001$
a vs b; e vs f	$p<0.01$
c vs d	$p<0.05$

TABLE 4.6 Clinical, anthropometric and biochemical data on admission - nutritional status determined by TSF

	Group C (± SEM)	Group D (± SEM)
Number of patients	15	33
Age (years)	57 ± 3.4	53 ± 3.0
Male : Female	5 : 10	21 : 12
Ideal body weight (%)[1]	107 ± 3.6	90 ± 2.6
Pre-illness weight loss (%)	8.5 ± 2.5	9.9 ± 1.6
Triceps skinfold thickness (%)[1]	99 ± 4.5	54 ± 2.5
Arm muscle circumference (%)[2]	101 ± 2.7	93 ± 1.8
Grip strength (%)	104 ± 5.9	94 ± 6.9
Serum albumin (g/l)	38 ± 1.6	38 ± 0.1
Serum transferrin (g/l)	2.7 ± 0.1	2.5 ± 0.1
Prognostic nutritional intake	75 ± 2.7	80 ± 1.8
Length of hospital stay (days)	20 ± 2.2	24 ± 3.0
Complications (deaths)	3 (1)	7 (2)

Levels of statistical significance (Mann-Whitney U-test)

[1] $p<0.001$

[2] $p<0.05$

body weights, TSF and AMC when compared to their normally nourished counterparts. There were no significant differences in PNI values between groups. Within the groups, the following changes were observed: Group C had lost significant amounts of body weight (107 ± 14, 104 ± 14 kg; $p<0.02$); serum albumin (37 ± 6, 35 ± 4 g/l; $p<0.05$) and serum transferrin (2.7 ± 0.5, 2.2 ± 0.5 g/l; $p<0.01$) between admission and discharge. Patients in Group D had significantly lower serum albumin (38 ± 6, 36 ± 6 g/l; $p<0.01$) and serum transferrin (2.5 ± 0.6, 2.1 ± 0.5 g/l) at discharge (Table 4.7). Five complications and two deaths (7 out of 33; 21%) occurred in Group D compared with three complications and one death in Group C (4 out of 15; 27%).

In order to identify clearly those patients who were energy and/or protein depleted, they were divided into three groups (Table 4.8). There was no significant change in biochemical or anthropometric markers at discharge in those patients classified as both energy and protein depleted (Group G; n = 14).

iv. DISCUSSION

For many years arm anthropometry has been used in nutritional surveys in developing nations (Jelliffe, 1966). The use of arm anthropometry as a clinical indicator of nutritional status was adopted in 1974 (Bistrian et al, 1974). A sub-normal AMC measurement may identify the protein depleted or stressed patient while a low TSF measurement may identify the starved or energy depleted patient. Whilst there is some debate as to the accuracy of using TSF and AMC in estimating fat and fat free mass, arm anthropometry may contribute to the patients nutritional assessment and management. The use of

TABLE 4.7 Changes in anthropometric and biochemical data on admission and at discharge - nutritional status determined by TSF (mean + 1SEM)

	Group C (normal)		Group D (malnourished)	
n	15		33	
	Admission	Discharge	Admission	Discharge
Body weight (%)	107±3.6[a]	104±3.9[b]	89±2.6	89±2.6
Triceps skinfold thickness (%)	99±4.5	88±5.6	54±2.5	52±3.1
Arm muscle circumference (%)	101±2.8	98±2.4	93±1.8[g]	90±2.0[h]
Serum albumin (g/l)	37±1.6[c]	35±1.0[d]	38±1.1[i]	36±1.1[j]
Serum transferrin (g/l)	2.7±0.1[e]	2.2±0.1[f]	2.5±0.1[k]	2.1±0.1[l]
Grip strength (kg)	103±6.2	91±8.4	95±6.9[m]	95±6.4[n]

Levels of statistical significance (Wilcoxon-Signed Rank)

i vs j;	m vs n	$p<0.001$
e vs f;	k vs l	$p<0.01$
a vs b		$p<0.02$
c vs d;	g vs h	$p<0.05$

TABLE 4.8 Changes in anthropometric and biochemical data on admission and at discharge (mean + 1SEM)

	Group E (normal)		Group F (TSF <80%)		Group G (TSF <80%, AMC <90%)	
n	15		23		10	
	Admiss.	Disch.	Admiss.	Disch.	Admiss.	Disch.
Body weight (%)	107±3.6[a]	104±4.0[b]	94±2.9	94±3.3	79±3.8	80±3.5
Triceps skinfold thickness (%)	99±4.5	88±5.6	55±2.9	50±3.7	52±5.1	52±7.8
Arm muscle circumference (%)	102±2.7	98±2.4	98±1.5[e]	96±1.8[f]	80±1.0	79±1.8
Serum albumin (g/l)	37±1.6	35±1.1	41±1.2	37±1.2	32±0.9	32±1.8
Serum transferrin (g/l)	2.7±0.1[c]	2.3±0.1[d]	2.7±0.1[g]	2.1±0.1[h]	2.1±0.1	2.0±0.2
Grip strength (kg)	103±6.2	97±7.1	107±6.5[i]	102±5.6[j]	80±17.3	63±14.4
Complications (deaths)	3 (1)		3 (1)		4 (1)	

Levels of statistical significance (Wilcoxon-Signed Rank)

a vs b; c vs d; e vs f $p<0.02$

g vs h; i vs j $p<0.001$

AMC (<90%) or TSF (<80%) to define protein or energy malnutrition is an adaptation of the anthropometric classifications used by Bistrian et al (1974), Mullen et al (1979) and Klidjian et al (1980).

When AMC was used to determine nutritional status those patients who were classified as malnourished (Group B) on admission had lower body weights, skeletal muscle mass, serum albumin and transferrin with reduced muscle function than the normally nourished patients (Group A). Following surgery the malnourished patients were able to conserve body weight, skeletal muscle, subcutaneous fat stores and maintain levels of serum albumin and transferrin (Table 4.4) whereas this did not occur in the normally nourished group where the nutritional parameters with the exception of grip strength were significantly lower at the time of discharge. This deterioration in nutritional status may be a consequence of the metabolic response to surgery. The acceleration in skeletal muscle breakdown may be an attempt to maintain the protein content of the viscera. This translocation of amino acids from skeletal muscle to viscera is a fundamental aspect of the metabolic response to injury (Kinney, 1980). Reiss (1959) demonstrated in rats that the stress of infection increased catabolism in muscle whilst the protein content of the viscera was maintained. The degree of catabolism or muscle breakdown is reflected postoperatively by an increase in urinary nitrogen excretion, which has been shown to be most marked in healthy young males (Kinney, 1967). Interestingly males predominate in the normally nourished group, but urinary nitrogen excretions were not monitored.

The postoperative depletion of plasma proteins was first documented half a century ago by Cuthbertson and Tompsett (1935).

The explanation of the postoperative fall in plasma proteins is multifactorial, related not only to rates of synthesis and breakdown but also to changes in the volume and distribution of body fluids and proteins as a result of increased capillary membrane permeability (Carpentier et al, 1982; Raines et al, 1984). The fall in concentration of these plasma proteins occurs as early as 2-4 hours after the first skin incision (Myers et al, 1984). Despite the influence of these factors on serum albumin and transferrin levels, concentrations of serum proteins continue to be used as nutritional markers in the postoperative period. Serum proteins may remain depressed for several days or weeks postoperatively depending on the magnitude of the surgical insult or the onset of complications. Interestingly, serum albumin and transferrin levels fell only in patients with normal AMC (Group A). Those patients classified as malnourished by AMC appeared unable to mount this metabolic response to surgery where there was no significant change in anthropometry or plasma protein levels postoperatively. This may either be as a consequence of the patients severely depleted preoperative nutritional state or an effect of fluid retention which may have masked any postoperative changes.

When TSF was used to determine nutritional status a much larger proportion of patients were classified as malnourished (Group D). These patients on admission had lower body weights, subcutaneous fat and skeletal muscle stores than their normally nourished counterparts (Group C). The effect of surgery in the normally nourished patients was that they lost 13% subcutaneous fat stores and 4% body weight while malnourished patients did not lose subcutaneous fat but appeared to lose small amounts of skeletal muscle. These results

again suggest that the malnourished patients (Group D) may adapt to the weight losing state and that surgical trauma does not appear to produce any additional demand on body fat and energy stores. However, the maintenance of skeletal muscle was less efficient in these patients. The reduction in plasma protein levels found in both groups (Groups C & D) may be attributed to factors previously described.

As the simple division of patients into two groups did not clearly identify those patients who were both energy and protein depleted, patients were divided into three groups: normal (Group E: TSF >80%; AMC >90%); energy depleted (Group F: TSF <80%) and an energy and protein depleted group (Group G: TSF <80%; AMC <90%). Results suggest the energy depleted patients (Group F) may be in a state of semi-starvation, which may be a consequence of the disease process causing a reduction in dietary intake with a lowering of energy expenditure in an attempt to balance caloric intake (Keys et al, 1950). The energy and protein depleted group (Group G) appeared to have the greatest ability of conserving their limited subcutaneous fat and muscle stores as well as maintaining plasma protein levels. This may be an effect of chronic starvation or due to alterations in extracellular volume (Shizgal, 1981). These results suggest that the more profound the patients malnutrition, the greater the body's ability to adapt to a weight losing state. Thus the impact of major surgery on body composition and plasma proteins was less in those patients identified as energy and protein depleted.

However, it is important when interpreting arm anthropometry to note that the technique is affected by a number of inherent errors. For example, limb oedema will render measurements invalid, skin

compressibility varies with age and sex, and distribution of body fat varies with age, sex and ethnic group (Goodison and Dickerson, 1988). In this study arm anthropometry alone was used to determine nutritional status as other workers have been criticised for exaggerating the prevalence of hospital malnutrition by using other inappropriate nutritional indicators (McLaren, 1988).

In this study the complication rate may be considered by some to be high (20%). However, the unit is a secondary referral centre for complex pancreatico-biliary and other surgery which are known to be associated with a higher postoperative morbidity and mortality (Table 4.2). Anthropometry had no predictive ability in identifying the patient at risk of developing postoperative complications. The modified PNI (Garden et al, 1985) was also a poor predictor of postoperative complications. Although the PNI used the anthropometric parameter TSF, the PNI value is strongly influenced by the serum albumin level. This was illustrated by the fact that significantly higher PNI values were calculated in patients classified as malnourished by AMC. Rather than identifying patients 'at risk', the PNI was merely selecting those with low serum albumin levels.

Detsky et al (1984, 1987) suggested that a subjective assessment (medical history, physical examination) had a greater predictive ability than an objective nutritional assessment. However, the problem with subjective measurements is their reproducibility, within and between observers. Half the postoperative complications in this study were predicted by the surgeon in the peri-operative period. There was an increase in ranking on the linear analogue in seven patients, five of whom

developed postoperative complications. These results support the findings of Pettigrew et al (1987) who suggested that it was the operative performance itself rather than the preoperative nutritional status which was a major factor in the development of postoperative complications.

Muscle function has also been identified as an accurate predictor in identifying the 'at risk' patient (Klidjian et al, 1980), but grip strength had no predictive ability in this study. Griffith and Clark (1984) used the criteria of a hospital stay in excess of 14 days to identify the patient who suffered a postoperative complication. In the present study a hospital stay greater than 14 days was attributed to other factors as well as the development of a clinical complication.

In conclusion, anthropometric measurements were unable to predict those patients who were at risk of developing postoperative complications. Major factors that influenced outcome were the surgical procedure and technical difficulty of the operation. Therefore, when considering the role of nutrition and its effect on surgical complication rate, a subjective assessment of the surgery should also be recorded.

The impact of major surgery on body composition was affected by preoperative nutritional status, as defined by anthropometry. Malnourished patients appeared to have the ability to conserve body mass whereas better nourished patients showed a more pronounced metabolic response to surgery. Aggressive preoperative nutritional support may alter the postoperative metabolic response in these malnourished patients.

CHAPTER 5 CONCLUSIONS

The anthropometric measurements body weight, TSF, AMC and MUAC will continue to play a key role in the nutritional assessment of hospitalised patients. While use of the aforementioned measurements are associated with a number of inherent errors, in the hands of a skilled and experienced operator they permit, at minimal cost, a rapid assessment of the body's endogenous stores.

The use of standard weight for height tables have been criticised for being self selective and geographically and ethnically biased. The recent publication of new weight for height tables on population studies carried out in the United Kingdom (Knight, 1986) may permit a more appropriate guide to 'ideal' body weights in this country. However, with regard to hospitalised patients we should consider the changes in body weight rather than absolute values. Furthermore, it is also important to consider the magnitude of pre-illness weight loss related to 'usual' body weight. Despite the limitations associated with the use of body weight as a nutritional indicator, it continues to be used routinely in clinical practice.

This dissertation has reviewed a wide spectrum of accepted and often complicated methods used in the assessment of body composition, many of which are unsuitable for routine use in hospital practice. However, the use of arm anthropometry, a non-invasive procedure to measure fat and FFM, permits assessment of endogenous energy stores in the critically ill or bed bound patient. The further use of arm anthropometry as a means of estimating REE was considered in a large group of patients defined as weight stable or weight losing with or without malignant disease. There was a significant correlation between REE and AMC in each of the four patient groups, the strongest correlations being observed in the two weight losing groups.

Interestingly, the presence of malignancy did not affect the relationship of REE with AMC, which suggests that skeletal muscle is not responsible for disease specific changes in REE in these patients. However, the weight status of the patient did affect the relationship of REE with AMC; at any given AMC measurement the weight losing patients had a lower REE when compared with their weight stable counterparts. These same weight losing patients also had a stronger correlation between REE and AMC than the weight stable group, which may be accounted for by the increased precision of arm anthropometry in weight loss. Results of this study suggest that AMC may be used in hospitalised patients to provide an estimate of REE. This would enable assessment of REE in patients at any district general hospital and would facilitate the prescribing of appropriate nutritional support.

Indirect calorimetry was used to assess REE in a small group of patients who following an anthropometric assessment were found to be 'overweight'. These patients were well defined in terms of age and diagnosis, two factors known to affect body composition. A number of conventional predictive formulae used to estimate REE were also tested using this patient group; these formulae were derived from indirect calorimetry carried out in heterogeneous groups of healthy volunteers. The conventional predictive formulae and the derived non-cancer weight stable regression equation consistently overestimated REE when compared with REE measured by indirect calorimetry. There was also no significant correlation between REE derived from AMC and REE measured by indirect calorimetry. These results suggest that use of the previously derived regression equation is inappropriate in an 'overweight' population. One

interesting finding was the significant correlation between the uncalculated measurement MUAC and REE from indirect calorimetry. This may be due to difficulties in the isolation of subcutaneous fat within the jaws of the skinfold calipers, and this may be attributed to the fact that there is poor delineation of fat and muscle in obese limbs.

These studies suggest that the various regression equations used to assess resting energy expenditure are applicable to different patient groups and may be dependent on body morphology rather than disease state. Further work will need to be undertaken before arm anthropometry will be accepted as a method of estimating energy expenditure. In clinical practice the most accepted and widely used method of measuring REE is indirect calorimetry. However, the stable isotope doubly labelled water has recently become available for use in human metabolic studies. The more simple technique of estimating REE by anthropometry could be validated using doubly labelled water to calculate energy expenditure.

Anthropometry has also been demonstrated to be a useful technique in categorising patients nutritional status prior to operation. Patients showed a different metabolic response to operation depending on the preoperative nutritional status. Well nourished patients appeared to have an increased metabolic response with respect to changes in anthropometry and serum protein levels when compared with a protein and energy depleted group. At time of discharge the well nourished patients had lost significant amounts of fat and skeletal muscle mass. This finding may have important implications during the provision of preoperative nutritional support and may lead to a more substantial metabolic response among

malnourished patients. Whether increased or reduced metabolic response imparts a beneficial effect in these patients is unclear. Increased understanding of the relationship between body composition, nutritional status and the metabolic response to injury will be necessary to provide a more rational basis for the optimal timing and technique of nutritional support.

This dissertation suggests that anthropometry may provide information of theoretical and practical importance in understanding the nutritional requirements of hospitalised patients.

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APPENDIX

CHAPTER 2 - THE ESTIMATION OF RESTING ENERGY EXPENDITURE BY ANTHROPOMETRY

CLINICAL AND ANTHROPOMETRIC DATA - NON-CANCER WEIGHT STABLE GROUP

Patient No.	Sex	Age	Diagnosis	Height (cms)	Weight (kgs)	% Weight Loss
1	F	44	Gallstones	151	86.9	0
2	M	70	Gallstones	166	67.8	0
3	M	61	Gallstones	158	53.8	6
4	F	69	Gallstones	146	49.0	0
5	F	50	Hiatus hernia	162	64.1	0
6	F	71	Gallstones	154	51.2	0
7	F	55	Gastric ulcer	151	38.3	7
8	F	55	Gallstones	154	63.0	0
9	M	68	Gallstones	165	66.9	0
10	M	40	Duodenal ulcer	167	72.9	0
11	M	50	Gallstones	178	85.9	0
12	F	72	Gallstones	149	63.7	0
13	F	70	Gallstones	158	71.4	0
14	F	77	Gallstones	153	69.9	0
15	F	77	Gallstones	163	77.5	0
16	F	50	Gastric ulcer	162	71.0	0
17	F	80	Short bowel syndrome	150	43.8	0
18	F	45	Gastric ulcer	153	52.5	0
19	F	86	Diverticular disease	152	51.8	9
20	F	80	Duodenal ulcer	151	56.7	6

CLINICAL AND ANTHROPOMETRIC DATA - NON-CANCER WEIGHT STABLE GROUP (Continued)

21	M	59	Ulcerative	173	75.0	4
22	F	40	Gallstones	169	101.9	0
23	F	51	Rectal polyp	167	93.5	0
24	F	80	Rectal polyp	156	68.7	0
25	F	71	Rectal polyp	151	56.9	0
26	M	62	Gallstones	169	62.1	2
27	F	41	Gallstones	166	74.9	6

% weight loss of 0 was recorded when patient had no recent weight loss

APPENDIX

CHAPTER 2 - THE ESTIMATION OF RESTING ENERGY EXPENDITURE BY ANTHROPOMETRY

CLINICAL AND ANTHROPOMETRIC DATA - NON-CANCER WEIGHT STABLE GROUP

Patient No.	TSF (mm)	% TSF	AMC (cm)	% AMC	MUAC (cm)	REE (kcals/day)
1	34.0	61	28.3	122	39.0	1413
2	10.0	80	23.9	94	27.0	1458
3	9.0	72	22.6	89	25.5	1253
4	21.0	127	19.9	86	26.5	1158
5	14.5	88	23.0	99	27.5	1263
6	16.0	97	21.4	92	26.5	978
7	7.6	46	18.7	81	21.0	1113
8	27.0	164	25.9	112	34.5	1199
9	15.4	123	24.0	95	29.0	1528
10	12.0	96	27.1	107	31.0	1658
11	7.6	61	27.5	109	30.0	1584
12	20.5	124	23.5	101	30.0	1175
13	24.0	145	22.0	95	29.5	1299
14	19.8	120	25.1	108	31.5	1179
15	25.0	152	21.6	93	29.5	1347
16	26.0	158	23.6	102	32.0	1327
17	13.0	79	19.4	84	23.5	1084
18	15.5	94	21.8	94	26.5	1323
19	15.5	94	24.1	104	29.0	989
20	15.5	94	25.6	110	30.5	1072
21	15.5	124	25.1	99	30.0	1707

CLINICAL AND ANTHROPOMETRIC DATA - NON-CANCER WEIGHT STABLE GROUP (Continued)

22	30.5	185	29.3	126	39.0	1705
23	32.0	194	28.4	122	38.5	1570
24	34.0	206	23.9	103	34.5	1273
25	16.0	97	22.9	99	28.0	1183
26	9.0	72	21.2	84	24.0	1537
27	36.0	218	23.6	102	35.0	1666

APPENDIX

CHAPTER 2 - THE ESTIMATION OF RESTING ENERGY EXPENDITURE BY ANTHROPOMETRY

CLINICAL AND ANTHROPOMETRIC DATA - CANCER WEIGHT LOSING GROUP

Patient No.	Sex	Age	Diagnosis	Height (cms)	Weight (kgs)	% Weight Loss
1	M	61	Gastric Ca.	160	54.4	10
2	M	76	Bronchial Ca.	168	46.6	26
3	F	62	Gastric Ca.	159	39.4	18
4	F	71	Colonic Ca.	155	53.2	11
5	M	33	Bronchial Ca.	188	68.1	18
6	F	63	Gastric Ca.	154	42.6	16
7	F	62	Bronchial Ca.	154	37.0	22
8	F	73	Colonic Ca.	151	48.8	10
9	M	45	Gastric Ca.	164	41.2	28
10	M	62	Rectal Ca.	177	62.7	10
11	M	65	Colonic Ca.	163	66.7	13
12	M	71	Bronchial Ca.	156	47.0	18
13	M	62	Gastric Ca.	172	53.5	35
14	M	73	Rectal Ca.	164	49.2	21
15	M	64	Prostate Ca.	165	63.9	13
16	F	72	Rectal Ca.	158	50.9	24
17	F	54	Oesophageal Ca.	159	74.9	15
18	F	69	Bronchial Ca.	149	34.6	16
19	F	83	Rectal Ca.	147	43.8	37
20	F	54	Colonic Ca.	166	62.1	11
21	M	63	Colonic Ca.	172	60.2	14

CLINICAL AND ANTHROPOMETRIC DATA - CANCER WEIGHT LOSING GROUP
(Continued)

22	M	72	Rectal Ca.	179	77.5	19
23	F	72	Gastric Ca.	154	36.6	19
24	F	77	Rectal Ca.	155	43.5	16
25	F	66	Pancreatic Ca.	162	34.5	28
26	F	74	Colonic Ca.	158	44.6	21
27	F	60	Colonic Ca.	162	58.0	17
28	F	69	Colonic Ca.	153	51.4	13
29	F	77	Gastric Ca.	157	40.5	13
30	M	74	Colonic Ca.	162	62.2	11
31	M	70	Gastric Ca.	160	43.4	14
32	F	52	Colonic Ca.	167	54.6	13
33	F	64	Gastric Ca.	150	41.8	37
34	F	70	Rectal Ca.	150	66.2	13
35	F	60	Gastric Ca.	149	44.3	17
36	F	64	Colonic Ca.	162	43.4	12
37	M	48	Gastric Ca.	163	64.0	18
38	M	73	Rectal Ca.	170	45.3	29
39	F	81	Gastric Ca.	152	52.1	18
40	F	88	Colonic Ca.	148	50.6	12
41	M	70	Gastric Ca.	169	46.5	23
42	M	51	Gastric Ca.	166	49.1	19
43	M	77	Colonic Ca.	171	61.0	17
44	M	55	Gastric Ca.	168	51.8	28

APPENDIX

CHAPTER 2 - THE ESTIMATION OF RESTING ENERGY EXPENDITURE BY ANTHROPOMETRY

CLINICAL AND ANTHROPOMETRIC DATA - CANCER WEIGHT LOSING GROUP

Patient No.	TSF (mm)	% TSF	AMC (cm)	% AMC	MUAC (cm)	REE (kcal/day)
1	7.4	59	23.9	94	26.0	1417
2	2.8	22	18.1	72	19.0	1144
3	6.8	41	17.0	73	19.0	998
4	15.5	94	21.1	91	26.0	1234
5	11.2	90	23.0	91	26.5	1843
6	11.6	70	16.9	72	20.5	1205
7	5.7	34	16.5	71	18.0	1028
8	18.2	110	19.8	85	25.5	963
9	5.0	40	17.9	71	19.5	1109
10	5.3	42	22.8	90	24.5	1592
11	8.2	66	25.2	100	27.5	1432
12	4.2	34	19.9	79	21.0	1140
13	6.3	50	22.2	88	24.0	1335
14	5.5	44	21.8	86	23.5	1302
15	10.8	86	24.6	97	28.0	1884
16	12.4	75	20.5	88	24.5	1366
17	21.0	127	24.9	107	31.5	1473
18	6.4	39	16.8	72	19.0	1039
19	13.5	82	19.3	83	23.5	961
20	15.0	91	23.4	101	28.0	1366
21	9.0	72	22.1	87	25.0	1635

CLINICAL AND ANTHROPOMETRIC DATA - CANCER WEIGHT LOSING GROUP
(Continued)

22	7.5	60	25.6	101	28.0	1765
23	8.2	50	17.4	75	19.5	1037
24	8.5	52	20.3	88	23.0	1170
25	4.5	27	15.1	65	16.5	794
26	10.0	61	19.8	85	23.0	1121
27	15.0	91	23.3	100	28.0	1358
28	19.0	115	21.5	93	27.5	1131
29	10.0	61	20.6	89	21.0	973
30	11.0	67	23.5	93	27.0	1318
31	4.5	36	20.5	81	22.0	948
32	22.0	133	24.5	106	31.5	1502
33	11.0	67	20.0	86	23.5	1168
34	17.0	103	24.1	104	29.5	1296
35	15.0	91	21.3	92	26.0	1244
36	13.5	82	21.2	91	25.5	1269
37	11.0	88	24.5	97	28.0	1722
38	4.0	32	18.7	81	20.0	1288
39	10.5	64	22.2	88	25.5	1152
40	12.0	73	21.2	84	25.0	1136
41	4.0	32	18.7	74	20.0	1162
42	4.5	36	22.5	89	24.0	1152
43	13.0	104	22.9	91	27.0	1551
44	6.0	48	23.6	93	25.5	1300

APPENDIX

CHAPTER 2 - THE ESTIMATION OF RESTING ENERGY EXPENDITURE BY ANTHROPOMETRY

CLINICAL AND ANTHROPOMETRIC DATA - NON-CANCER WEIGHT LOSING GROUP

Patient No.	Sex	Age	Diagnosis	Height (cms)	Weight (kgs)	% Weight Loss
1	F	64	Duodenal ulcer	151	57.4	14
2	F	66	Gallstones	152	77.8	22
3	F	77	Gastric ulcer	154	38.4	25
4	M	85	Caecal polyp	170	53.1	21
5	F	57	Duodenal ulcer	151	37.7	14
6	M	77	Gallstones	167	78.3	10
7	M	32	Crohn's disease	177	62.8	10
8	F	63	Diverticular disease	168	69.2	16
9	M	46	Duodenal ulcer	165	49.2	14
10	M	73	Duodenal ulcer	179	56.8	15
11	F	80	Gallstones	153	63.0	24
12	M	60	Duodenal ulcer	159	62.9	21
13	F	65	Duodenal ulcer	153	36.8	20
14	M	56	Crohn's disease	183	59.0	12
15	F	34	Duodenal ulcer	155	42.3	11
16	M	79	Rectal polyp	169	56.5	11
17	F	76	Diverticular disease	148	40.5	18

APPENDIX

CHAPTER 2 - THE ESTIMATION OF RESTING ENERGY EXPENDITURE BY ANTHROPOMETRY

CLINICAL AND ANTHROPOMETRIC DATA - NON-CANCER WEIGHT LOSING GROUP

Patient No.	TSF (mm)	% TSF	AMC (cms)	% AMC	MUAC (cms)	REE (kcal/day)
1	20.0	121	20.2	87	26.5	1286
2	20.0	121	23.6	102	30.0	1428
3	11.0	67	15.5	67	19.0	979
4	5.6	45	19.8	78	21.5	1014
5	8.5	52	16.9	73	19.5	1074
6	14.6	117	22.9	91	27.5	1582
7	7.8	62	24.1	95	26.5	1522
8	20.0	121	23.7	102	30.0	1504
9	7.0	56	23.3	92	25.5	1280
10	7.0	56	20.8	82	23.0	1364
11	16.0	97	23.9	103	29.0	1129
12	8.5	68	26.8	102	29.5	1347
13	10.5	64	19.2	83	22.5	897
14	5.0	40	23.9	94	25.5	1690
15	9.0	55	18.1	78	21.0	1158
16	8.0	64	23.5	93	26.0	1442
17	10.0	61	17.8	77	20.9	973

APPENDIX

CHAPTER 2 - THE ESTIMATION OF RESTING ENERGY EXPENDITURE BY ANTHROPOMETRY

CLINICAL AND ANTHROPOMETRIC DATA - CANCER WEIGHT STABLE GROUP

Patient No.	Sex	Age	Diagnosis	Height (cms)	Weight (kgs)	% Weight Loss
1	M	62	Rectal Ca.	173	96.4	0
2	M	72	Oesophageal Ca.	172	74.0	3
3	M	68	Rectal Ca.	163	60.1	6
4	M	71	Colonic Ca.	162	48.6	7
5	F	70	Colonic Ca.	157	89.6	7
6	M	73	Gastric Ca.	167	63.9	6
7	M	44	Oesophageal Ca.	174	60.9	8
8	M	75	Rectal Ca.	178	76.9	0
9	M	67	Colonic Ca.	177	76.7	8
10	M	84	Colonic Ca.	169	66.6	9
11	M	62	Colonic Ca.	174	75.9	5
12	M	75	Gastric Ca.	171	55.0	4
13	M	63	Gastric Ca.	166	52.2	8
14	F	55	Gastric Ca.	159	80.1	0
15	F	72	Rectal Ca.	149	65.4	7
16	M	54	Bronchial Ca.	171	66.2	0
17	M	77	Colonic Ca.	161	72.4	0
18	F	71	Colonic Ca.	159	72.1	0
19	M	70	Bronchial Ca.	168	52.6	0
20	M	72	Colonic Ca.	167	68.2	0
21	M	79	Gastric Ca.	163	61.4	6

CLINICAL AND ANTHROPOMETRIC DATA - CANCER WEIGHT STABLE GROUP (Continued)

22	F	74	Colonic Ca.	154	56.6	0
23	F	60	Rectal Ca.	156	49.5	3
24	M	61	Rectal Ca.	161	53.7	6
25	F	83	Colonic Ca.	147	52.0	6
26	M	69	Colonic Ca.	161	58.7	0
27	F	77	Colonic Ca.	153	52.7	0
28	F	80	Gastric Ca.	136	61.5	9
29	M	70	Rectal Ca.	168	71.6	0
30	M	74	Colonic Ca.	165	66.1	0
31	F	69	Colonic Ca.	133	39.7	4
32	M	44	Rectal Ca.	172	70.9	7
33	M	37	Rectal Ca.	168	58.8	3
34	M	57	Colonic Ca.	163	78.7	5
35	M	66	Gastric Ca.	180	84.2	0
36	M	73	Gastric Ca.	176	78.5	5
37	F	68	Gastric Ca.	146	44.0	8
38	M	60	Gastric Ca.	161	76.9	5
39	M	51	Colonic Ca.	169	61.0	6
40	M	60	Colonic Ca.	166	47.9	9
41	F	64	Rectal Ca.	165	60.0	8
42	M	77	Colonic Ca.	169	63.8	8
43	F	50	Rectal Ca.	161	62.3	0
44	M	71	Rectal Ca.	160	53.7	0
45	M	68	Rectal Ca.	158	58.2	0
46	M	46	Colonic Ca.	168	56.6	10
47	M	62	Rectal Ca.	167	65.4	0

CLINICAL AND ANTHROPOMETRIC DATA - CANCER WEIGHT STABLE GROUP (Continued)

48	M	50	Gastric Ca.	173	70.1	0
49	F	69	Colonic Ca.	155	48.4	5
50	M	66	Gastric Ca.	168	79.2	6
51	F	68	Gastric Ca.	167	52.5	0
52	F	64	Colon Ca.	156	66.8	0
53	M	76	Gastric Ca.	161	77.9	0
54	M	41	Colonic Ca.	153	55.8	6

% weight loss of 0 was recorded when patient had no recent weight loss

APPENDIX

CHAPTER 2 - THE ESTIMATION OF RESTING ENERGY EXPENDITURE BY ANTHROPOMETRY

CLINICAL AND ANTHROPOMETRIC DATA - CANCER WEIGHT STABLE GROUP

Patient No.	TSF (mm)	% TSF	AMC (cm)	% AMC	MUAC	REE (kcal/day)
1	14.0	112	33.8	133	38.0	1676
2	13.5	108	25.8	102	30.0	1407
3	14.0	112	21.5	85	26.6	1314
4	5.0	40	18.3	72	20.0	1109
5	33.0	200	25.9	112	36.0	1619
6	11.5	92	23.8	94	27.5	1362
7	6.8	54	20.4	81	22.5	1269
8	9.8	78	24.6	97	27.5	1442
9	13.0	104	24.3	96	28.5	1818
10	12.0	96	25.2	100	29.0	1697
11	13.8	110	23.9	94	28.0	1483
12	4.2	34	21.9	87	23.0	1399
13	7.2	58	20.7	82	23.0	1352
14	32.0	194	26.5	114	36.5	1561
15	20.5	124	22.5	97	29.0	1238
16	13.0	104	25.1	99	29.0	1347
17	9.8	78	25.9	102	29.0	1640
18	25.0	152	22.4	96	30.0	1296
19	6.6	53	22.4	88	24.5	1510
20	10.5	84	24.2	96	27.5	1518
21	8.0	64	26.9	106	29.5	1434

CLINICAL AND ANTHROPOMETRIC DATA - CANCER WEIGHT STABLE GROUP
(Continued)

22	14.5	88	24.4	105	29.0	1156
23	19.0	115	20.0	86	26.0	1177
24	10.5	84	21.7	86	25.0	1288
25	16.0	97	22.9	99	28.0	1230
26	10.0	80	24.8	98	28.0	1580
27	20.0	121	23.7	102	30.0	1166
28	31.0	188	24.2	104	34.0	1160
29	11.5	92	23.8	94	27.5	1572
30	12.5	100	26.5	105	30.5	1222
31	28.0	170	19.7	85	28.5	1189
32	15.0	120	24.8	98	29.5	1701
33	17.0	136	24.1	95	29.5	1512
34	18.5	148	27.2	108	33.0	1761
35	13.5	108	26.2	104	30.5	1660
36	19.0	152	26.6	105	29.5	1510
37	11.0	67	22.0	95	25.5	1164
38	11.0	88	28.0	111	31.5	1518
39	8.0	64	23.5	93	26.0	1734
40	5.5	44	20.3	80	22.0	1247
41	15.5	94	24.1	104	29.0	1269
42	12.0	96	22.7	90	26.5	1450
43	19.0	115	23.5	101	29.5	1417
44	10.0	80	24.4	96	27.5	1269
45	16.0	128	21.9	87	27.0	1195
46	7.0	56	23.8	94	26.0	1458
47	13.0	104	24.4	96	28.5	1329

CLINICAL AND ANTHROPOMETRIC DATA - CANCER WEIGHT STABLE GROUP
(Continued)

48	7.0	56	27.3	108	29.5	1582
49	17.0	103	20.6	89	26.0	1205
50	17.0	136	26.6	105	32.0	1744
51	13.0	79	17.4	75	21.5	1545
52	22.5	136	21.4	92	28.5	1393
53	10.5	84	26.7	106	30.0	1325
54	4.5	36	25.1	99	26.5	1500

APPENDIX

CHAPTER 4 - NUTRITIONAL STATUS AND OUTCOME IN THE SURGICAL PATIENT

CLINICAL AND ANTHROPOMETRIC DATA ON ADMISSION

Patient No.	Sex	Age	Diagnosis	IBW (%)	TSF (%)	AMC (%)	GS (%)	Albumin (g/l)	Trans-ferrin (g/l)
1	F	67	Colonic abscess	86	133	82	74	42	3.2
2	M	68	Ca.colon	107	80	110	130	45	3.0
3	M	60	Ca.stomach	124	88	109	105	36	2.4
4	F	74	Ano-rectal	107	91	111	93	37	2.4
5	M	43	Pancreat-itis	97	96	92	125	40	2.7
6	F	48	Pancreat-itis	109	97	95	67	36	2.9
7	F	72	Ca.rectum	92	97	90	140	41	2.6
8	M	34	Ob. jaundice	109	80	111	138	38	2.7
9	F	64	Ca. pancreas	115	94	110	92	31	3.5
10	F	52	Ob. jaundice	104	82	89	92	30	2.5
11	F	45	Intestinal obstruction	106	109	109	118	43	3.4
12	F	59	Cholangio carcinoma	114	133	95	81	22	1.6
13	F	72	Cholangio carcinoma	102	121	98	92	40	2.6
14	M	61	Ca.rectum	145	88	111	105	39	2.4
15	F	38	Cholangio carcinoma	95	103	112	118	45	3.1

CLINICAL AND ANTHROPOMETRIC DATA ON ADMISSION (Continued)

16	F	39	Pancreat-itis	65	42	85	155	29	1.9
17	F	25	Crohn's disease	72	42	79	-	27	1.9
18	M	27	Stabbing	100	40	100	125	47	3.0
19	M	43	Ca.colon	99	60	97	120	44	2.6
20	M	33	Pancreat-itis	88	56	102	162	38	2.5
21	M	65	Peptic ulceration	99	52	99	100	41	2.9
22	M	51	Peptic ulceration	85	68	90	95	45	3.5
23	F	46	Pancreat-itis	85	64	81	111	30	3.0
24	F	85	Ca.colon	100	76	80	85	32	2.0
25	M	45	Gastritis	95	40	102	95	43	2.7
26	M	54	Irritable bowel dis.	103	56	104	95	40	3.3
27	F	69	Colonic abscess	90	70	96	92	51	2.8
28	M	38	Pancreat-itis	79	36	97	112	44	2.7
29	F	72	Cholangio carcinoma	67	67	91	73	31	2.6
30	F	67	Ca.rectum	119	79	101	59	44	3.3
31	F	76	Ca.pancreas	69	67	78	56	34	2.2
32	M	58	Ca.rectum	94	64	99	112	42	2.2
33	F	61	Ca.stomach	89	67	91	45	29	1.2
34	M	33	Peptic ulceration	108	60	113	62	48	2.7
35	F	85	Ca. oesophagus	98	52	81	30	30	1.5

CLINICAL AND ANTHROPOMETRIC DATA ON ADMISSION (Continued)

36	M	27	Gastritis	87	48	99	130	41	2.2
37	M	63	Ca.stomach	70	20	78	55	34	2.6
38	F	76	Ca.pancreas	78	48	82	44	33	1.8
39	M	39	Pancreat-itis	75	36	91	138	44	2.8
40	M	47	Gastritis	88	32	96	62	45	3.6
41	M	55	Ca.stomach	77	44	84	112	35	2.2
42	M	27	Hyper-splenism	133	40	120	125	46	3.4
43	M	62	Cholangio carcinoma	96	48	99	88	32	2.2
44	M	51	Pancreat-itis	106	72	96	105	44	3.1
45	F	60	Cholangio carcinoma	86	52	92	92	34	1.9
46	M	70	Ca.liver	76	60	74	-	36	1.7
47	M	36	Adrenal Ca.	100	72	95	175	38	1.7
48	M	68	Ca.stomach	90	44	92	95	39	2.5

APPENDIX

CHAPTER 4 - NUTRITIONAL STATUS AND OUTCOME IN THE SURGICAL PATIENT

CLINICAL AND ANTHROPOMETRIC DATA ON DISCHARGE

Patient No.	IBW (%)	TSF (%)	AMC (%)	GS (%)	Alb. (g/l)	Trans-ferrin (g/l)	Complic-ations	Length of Hospital stay (days)
1	88	133	89	74	38	2.1	No	22
2	104	96	100	120	37	2.2	No	31
3	115	80	112	88	39	2.5	No	15
4	-	91	92	30	36	1.9	No	15
5	-	48	91	112	41	2.5	No	62
6	100	85	93	74	37	2.8	No	35
7	90	73	96	111	33	1.6	No	13
8	109	80	100	138	38	3.1	No	37
9	114	79	101	92	29	2.9	No	11
10	98	70	90	89	29	1.7	No	22
11	99	88	112	92	33	1.5	Yes	18
12	-	-	-	-	-	-	Died	16
13	102	121	98	48	33	2.6	No	31
14	138	96	115	120	35	2.7	Yes	78
15	92	91	88	111	-	-	No	74
16	73	48	84	81	-	-	No	8
17	80	58	82	74	41	3.1	No	29
18	94	48	95	105	40	2.4	No	21
19	96	68	96	125	36	2.2	No	14
20	90	68	96	170	-	-	No	10
21	94	36	105	100	33	2.2	No	9

CLINICAL AND ANTHROPOMETRIC DATA ON DISCHARGE (Continued)

22	80	56	86	100	41	2.7	No	19
23	77	69	74	92	-	-	Yes	36
24	94	96	76	98	31	1.7	Yes	9
25	91	32	100	95	38	2.5	No	21
26	89	40	93	75	35	2.1	Yes	25
27	92	67	93	92	42	2.2	No	21
28	77	32	90	105	43	2.5	No	22
29	-	67	84	-	34	2.3	Yes	20
30	114	73	104	59	32	1.9	No	33
31	-	-	-	-	-	-	Died	19
32	-	-	-	-	42	2.3	Died	14
33	-	-	-	-	30	1.1	No	14
34	108	60	113	125	41	1.7	No	30
35	94	48	80	22	32	1.6	No	27
36	78	40	94	130	43	2.5	No	28
37	73	25	71	45	-	-	No	11
38	74	48	78	37	30	1.6	No	19
39	72	34	87	122	44	1.9	No	11
40	82	36	93	75	40	2.5	No	11
41	76	36	83	100	32	2.1	No	12
42	125	44	116	120	44	2.8	No	10
43	116	44	94	-	23	1.3	No	21
44	105	80	94	95	38	2.2	Yes	33
45	86	39	94	92	26	1.7	No	19
46	74	56	70	-	28	1.6	No	10
47	95	72	94	162	39	1.9	No	9

CLINICAL AND ANTHROPOMETRIC DATA ON DISCHARGE (Continued)

48	99	48	95	88	31	2.0	No	29