Proc. Nutr. Aust. (1990) 15

CLINICAL APPLICATIONS OF BODY COMPOSITION MEASUREMENTS

BOYD J.G. STRAUSS

Summary

Body composition changes can be both markers of biological development and indicators of disease processes which are common in Australian society. Methods of measuring and following these changes have been developed with varying levels of portability, simplicity, precision and costs. These techniques are available in a number of Australian hospitals, and are providing a further method of assessing nutritional status and response to treatment.

I. WHAT CAN BE MEASURED, AND WHY SHOULD IT BE MEASURED ?

The measurement of components of body composition (BC), and following the changes over time as a result of normal changes, disease, training or therapy has become more widely discussed over recent years (Forbes 1987). Ideally, we ought to be able to follow changes in the composition of the several major organ systems of the body, and correlate these with physiological and pathological changes. To date, this is available only on a relatively crude basis. It is also true that a newer understanding of whole body physiology has emerged by coming to some understanding of the changes which can take place over the whole body. This has required the development of a number of models of body composition compartments, with an associated attempt to correlate these compartments with meaningful function.

II. THE TWO COMPARTMENT MODEL OF BODY COMPOSITION

The two compartment model of body composition defines a fat compartment and a lean compartment, and is the oldest of the models (Bruce et al. 1980). Essentially, it is based on the separation of water-miscible and lipid containing components of the body. This is a simplistic model in that all cell membranes contain lipid, and such a separation is probably artificial. There is some confusion about the terms which are used within this model.

Fat mass (FM) is defined as the mass of stored triglyceride, phospholipid and circulating fatty acids in the body, and is thus truly water-free.

The fat-free mass (FFM) is the remainder of the body weight after subtraction of the fat mass.

Both of these compartments are physiological concepts. The adipose tissue (AT) compartment consists of the mass of stored triglyceride associated with adipocytes, its supporting connective and vascular tissue, and the surrounding extracellular water.

The lean body mass (LBM) consists of the nonadipose tissue compartment of the body, including the skeleton and extracellular connective tissue, and includes the lipid component of the cell membranes included in this mass. The terms lean body mass and fat-free mass, and adipose tissue and fat mass are often used interchangeably, but is can be readily seen that there is a small but significant degree of overlap.

III. THE FOUR COMPARTMENT MODEL OF BODY COMPOSITION

The four compartment model involves measuring three subdivisions of the FFM, in addition to assessing the FM (Heymsfield et al. 1990). Total body water (TBW) is the volume of water in which the LBM exists. It has both an intracellular (ICW) and an extracellular (ECW) component.

Total body protein (TBP) is the mass protein found both extra- and intracellularly.

Total body calcium (TBC) effectively measures the mineral component of the skeleton

Monash University Department of Medicine and Body Composition Laboratory, Monash Medical Centre, Prince Henry's Hospital, St Kilda Rd, Melbourne, Victoria 3004

IV. NORMAL VARIATIONS IN BODY COMPOSITION

An understanding of the risk or the actual changes of body composition in disease requires knowledge of normal variation.
The various components of body composition, including height, weight, LBM and AT are under genetic influences (Bouchard et al. 1985). Age and sex are important determinants of the components of body composition, and relative body composition is changed throughout life (Strauss et al. 1987). Pregnancy is accompanied by body composition changes in the mother, with an increase in ECF and ICF and AT and AT distribution, whilst the composition of the foetus varies throughout development (Hytten and Chamberlain 1980). The onset of puberty is accompanied by a spurt increase in AT, especially in girls (Tanner and Whitehouse 1975). During adult years, AT varies much more than LBM and accounts for much of the variability in body weight. In late adulthood, there is a decline in LBM (Forbes 1987). Height is an important determinant of LBM and TBC, as is ethnicity, in an manner independent of height. TBC is higher in American blacks than in caucasians, whilst asians have a smaller LBM than caucasians (Mazess and Mather 1975).

V. HOW CAN THE COMPONENTS OF BODY COMPOSITION BE MEASURED. WHAT LEVEL OF SOPHISTICATION IS REQUIRED?

All clinical methods of body composition are approximate, since the absolute assessment of chemical analysis is unavailable, and, in any case, has been attempted on very few occasions (Widdowson and Dickerson 1964).

The methods of body composition analysis which are of clinical use vary in their availability, portability, precision, simplicity and cost.

Anthropometry (A) is the most widely used form of assessment, and is used directly to measure growth and fat distribution, and, by comparison with other methods, to derive FM and FFM. It is a method well suited to epidemiological studies as well as bedside assessment in the very sick individual.
The components able to be measured include height, weight, circumferences and skinfold thicknesses. Some of these parameters can be measured very precisely, to less than 1 %, whilst other components are subject to some intra- and inter-observer variation (Durnin and Womersley 1974).

The impedance of the body to a weak alternating current (800 μA, 50 Hz) is proportional to TBW. Recent development of bioelectrical impedance (BEI) has confirmed its value in the study of selected healthy populations and individuals, with a small error, when TBW shifts occur as a result of disease or therapy, the results are less reproducible (Lukaski et al. 1986). Most of the other techniques which are available tend to be located within hospital or laboratory research areas, and are hence more suited to use in individuals. Whilst the equipment is generally expensive to purchase, maintain and operate, more of the components of BC can be assessed, with very small disposable costs per test, at all acceptable level of invasibility.

Under-water densitometry has been a 'gold standard' measurement of the two compartment model for many years. It is not a practical instrument for measuring BC in sick or elderly individuals; there is an underlying assumption that the density of the FFM or LBM is uniform, and comparable between individuals, which is not true (Garrow et al. 1979).

Deuterium oxide dilution is beginning to replace tritiated water dilution as a relatively noninvasive 'gold standard' for measuring TBW; salivary assessment will make it acceptable to small children (Mendez et al. 1971). The technique may be useful in field studies where sample collection may take place, with later laboratory analysis.

Dual energy x-ray absorptiometry (DEXA) is a recent addition to the BC analysis field. Originally developed to quantify bone mass and bone density, this rapid technique, which involves a very low (0.02 mSv) x-ray exposure, is able to quantify regional and whole body AT, LBM and LBM. It has a very small error for measuring bone mass, whilst the error in measuring regional, especially abdominal, and whole body AT and LBM is beginning to be documented (Mazess et al. 1990a).

Neutron activation analysis is a tool which is potentially able to provide a simultaneous measurement of the whole body content of a range of elements; in practice, only nitrogen

measurements, to assess TBP, have been developed, because of the radiation exposure required (Cohn et al. 1980).

A number of other imaging tools enable body composition measurements; they include CT scanning, (Kvist et al. 1986) with the possibility of further defining fat distribution, and magnetic resonance imaging, (Seidell et at. 1990) with a potential for measuring intracellular elemental content; in practice these modalities are often fully utilised in performing scans for other disease processes.

VI. HEALTH RISKS AND DISEASES ASSOCIATED WITH DISORDERS OF BODY COMPOSITION

Body composition changes throughout life, and may be a good marker of the biological stage of development. The skeletal mass, for example, continues to grow into the third decade of life, and, in women, undergoes some involution peri-menopause (Genant et al. 1982).

Whilst convincing markers of the ageing process remain to be established, in the elderly person there is a shift of the TBW pool with a relative increase in the ECW, even in the apparent absence of cardiac, renal or hepatic disease, all of which may increase the ECW. This may be the result of a cell membrane process (Borkan and Norris 1977).

The health risks associated with the increased AT and LBM of obesity have been appreciated for many years. These include an increase in all-cause mortality, cardiovascular disease processes such as hypertension and coronary artery disease, endocrine disturbances such as non-insulin dependent diabetes mellitus, hirsutism and infertility, gallstone formation, osteo-arthritis, breast and endometrial malignancy, and a variety of psycho-social disturbances (Bray 1987).

Recently, it has been shown that many of these risks are increased further when fat is distributed abdominally rather than around the buttocks, although it is still unclear whether this is a function of increased mesenteric fat, abdominal subcutaneous fat or both. Differing metabolic profiles of adipocytes from these sites have been demonstrated (Larsson et al. 1984).

The response to reducing energy intake to a level below that of expenditure results initially, in a release and excretion of the ICW water which is bound with glycogen stores, which are not assessed in any of the body composition models. Eventually, both AT and LBM are reduced, although the site from which AT is principally lost cannot be predicted; on occasions it is possible to lose relatively more LBM than AT, giving rise to a form of protein/energy malnutrition. In general, thin people in negative energy balance lose a relatively greater amount of LBM per unit of weight loss than do obese people.

In protein-energy malnutrition, with or without a reduction in AT, there is loss of TBP and distortions in the TBW, resulting in immunosuppression, with a significantly increased risk of infection, poor wound healing, and increased mortality. This condition is commonly found in major hospitals, in association with anorexia (Mazess et al. 1990b), chronic inflammatory bowel disease, short bowel syndrome (Andersson et al. 1986), chronic obstructive pulmonary disease (Prijatmoko et al. 1990) and alcohol abuse (Bainbridge et al. 1990). Usually, other nutrient deficiencies are also seen.

Osteoporosis, in which there is a reduction of TBC, carries a major risk for bone fracture in postmenopausal women and very elderly men (Genant et al. 1982).

Apart from defining these clinical processes, BC measurements enable progress to be followed over time. The components of weight gain or loss in patients wit burns, on TPN or following gastric reduction surgery are of clinical importance.

VII. WHAT IS AVAILABLE NOW IN AUSTRALIA ?

A number of clinical centres have now developed major interests in measuring body composition in Australia, IVNAA for nitrogen is available at the Monash Medical Centre in Melbourne as part of a body composition laboratory, and is also available at Royal North Shore Hospital in Sydney, with plans for a paediatric development as well in that city. It is also available in Auckland New Zealand. Total body potassium measurements are available at the Monash Medical Centre and in Brisbane. The increasing availability of DEXA in several centres in Melbourne, Sydney and elsewhere gives a wider availability to this new dimension to BC measurements.

REFERENCES

ANDERSSON, H., BOSAEUS, I., BRUMMER, R. J. L. et al. (1986). Digest. Dis. 4: 193.
BAINBRIDGE, R., STRAUSS, B. J. G.,TAI,E. et al. (1990). In 'Body Composition in Chronic Obstructive Airways Disease'. (proc. AUSPEN 1990).
BORKAN, G. A. and NORRIS, A. H. (1977). Hum. Biol. 49: 495.
BOUCHARD, C., SAVARD, R., DESPRES, J. P., et al. (1985). Hum. Biol. 57: 61.
BRAY, G. A. (1987). Ann. N. Y. Acad Sci. 249: 14.
BRUCE, A.,ANDERSON, M., ARVIDSSON, B. and ISAKSSON, B. (1980). Scand. J. Clin. Lab. Invest. 40: 461.
COHN, S. H., VARTSKY, D., YAMASURA, S., et al. (1980). Am. J. Physiol. 239: E524.
DURNIN, J. V. G. SA. and WOMERSLEY J. (1974). Brit. J. Nutr. 32: 77.
FORBES, G. B. (1987). In 'Human Body Composition: Growth, Ageing, Nutrition and Activity'. (Springer-Verlag, New York).
FORBES, G. B. (1987). Nutr. Rev. 45: 225.
GARROW, J. S., STALLEY, S., DIETHELM, R., et al. (1979). Brit. J. Nutr. 42: 173.
GENANT, H. K., CANN,C. E. and FAULL, D. D. (1982) In 'Non-invasive Bone Measurements : Methodological problems.' eds J. Daqueker and C.C. Johnson (IRL press : Oxford).
HEYMSFIELD, S. B., LICHTMAN, S., BAUMGARTNER, R. N. et al. (1990). Am. J. Clin. Nutr. 52: 52.
HYTTEN, F. E. and CHAMBERLAIN G. (Eds.) (1980). In 'Clinical Physiology in Obstetrics'. (Blackwell Scientific Publications : London).
KVIST, H., SJOSTROM, L., and TYLEN, U. (1986). Int. J. Obesity. 10: 53.
LARSSON, B., SVARDSUDD, K., WEILIN, L. et al. (1984). Brit. Med. J. 288: 1401.
LUKASKI, H. C., BOLONCHUK, W. W., HALL, C. B.,et al. (1986). J. App. Physiol. 60(4): 1327.
MAZESS, R. B. and MATHER W. (1975). Am. J. Clin. Nutr. 27:916.
MAZESS, R. B., BARDEN, H. S., BISEK, J. P. et al. (1990a). Am. J. Clin. Nutr. 51: 1106.
MAZESS, R. B.,BARDEN, H. S. and OHLRICH, E. S. (1990b). Am. J. Clin. Nutr. 52: 438.
MENDEZ, J., PROKOP, E., et al. (1971). J. App. Physiol. 31: 957.
PRIJATMOKO, D., LAMBERT, J.R., STRAUSS, B. J. G. et al. (1990). In 'Body Composition in Alcoholic Cirrhosis : a Clinical Study'. (Proc. AUSPEN 1990).
SEIDELL, J. C., BAKKER, C.J. G., and VAN DER KOOY, K. (1990). Am. J. Clin. Nutr. 51: 953.
STRAUSS, B. J. G. BENGTSSON, C. and ISAKSSON B. (1987). Int. J. Obesity 11 (supp 2): 76.
TANNER, J. M. and WHITEHOUSE, R. H. (1975). Arch. Dis. Child. 50: 142.
WIDDOWSON, E. M. and DICKERSON, J. W. T. (1964). In 'Mineral Metabolism' Vol. 2 part A. pp 2. eds. C. L. Comar and F.Bronner (Academic Press : New York/London).