TRICEPS SKINFOLD, MID ARM CIRCUMFERENCE AND MID ARM MUSCLE CIRCUMFERENCE IN A MENTALLY RETARDED POPULATION

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INTRODUCTION

Triceps skinfold (TSF), mid arm circumference (MAC) and mid arm muscle circumference (MAMC) are valuable tools in assessing nutritional status (Jelliffee and Jelliffee, (1969). A large proportion of the retarded population falls below the tenth percentile for height and weight when compared to normal children. Many retarded have been reported to exhibit below normal measurements for head length, leg length, biacromial and biiliac diameter, and trunk diameter after age ten (Bailit and Whelan, 1967; Pozsonyi and Lobb, 1967; Thelander and Pryor, 1966; Mosier, Grossman, and Dingman, 1965). In 1883 Tarbell compared stature and weight of normal and institutionalized retarded children and demonstrated that the retarded were lighter in weight and shorter in stature (Bailit and Whelan, 1967). Thelander and Pryor (1966) showed specific growth abnormalities for the Down's Syndrome boys and girls, including short stature due to shorter legs, lack of head growth after age three and microcephaly. Standing height and trunk diameters were reported to be normal for the first ten years of life in a cerebral palsy population but showed a definite slump after age ten which Pryor and Thelander (1967) postulated may possibly be due to a lack of an adolescent growth spurt. Twenty-five

to forty percent of a group of cultural-familial retarded were two or more standard deviations below the normal population mean for height, head length, biacromial and biiliac diameters (Bailit and Whelan, 1967).

Thus growth retardation and deviation from the norm for other growth parameters have been documented in the handicapped and retarded population. A common criteria of growth retardation is a height that falls below the fifth percentile (Fiser, Meredith and Elders, 1975; Weaver and Owen, 1977). A height quotient, i.e., a height age (fiftieth percentile) divided by a chronological age, of .8 or less is also indicative of growth retardation or growth failure (Fiser et al., 1975). However, no data on TSF, MAC and MAMC has been made available for this population. It is, therefore, essential to know how the mentally retarded population compares to the normal population.

Well established criteria are used for assessing the nutritional status of children and adolescents. The measurements of length, weight, fat-fold thickness and radiographic assessment of skeletal maturity together provide objective measurements of the quality of growth for the normal child. Mentally retarded children often exhibit reduced muscle mass and are small for the developmental age. Weight for them becomes a less meaningful reference standard and circumference then measures less muscle and

more fat (Garn and Weir, 1971).

The MAMC and TSF provide an estimate of body muscle mass and protein reserve, and body fat and caloric reserve, respectively (Burgert and Anderson, 1979). Experimental and clinical studies have shown that the decrease in muscle mass, determined either through creatinine output or limb measurements, during malnutrition exhibit a greater reduction than body weight. The reduction in muscle size occurs as a compensatory mechanism to provide amino acids for gluconeogenesis and protein synthesis in the liver (Frisancho, 1974). The child with greater muscle size would reflect a greater protein reserve than a less muscular one. Increased subcutaneous fat, as measured by TSF, would reflect a greater caloric reserve. The retarded child exhibiting decreased muscle mass and decreased subcutaneous fat would appear to have a small protein and caloric reserve.

TSF and MAMC thus can be used for nutritional assessment; however, any evaluation of nutritional status must take into account the variation in the amount of subcutaneous fat and muscle mass (Frisancho, 1974). There is a critical need for standards that are applicable to the population and that will determine acceptable (not necessarily optimum) levels of TSF and MAMC in the mentally retarded population.

This study was done to determine if growth retardation in the mentally retarded population would also affect TSF, MAC and MAMC. A significant difference in TSF, MAC and MAMC between a mentally retarded and a normal population was expected. The purpose of this study was to determine if there is a need to establish separate norms for TSF, MAC and MAMC for the mentally retarded population.

PROBLEM STATEMENT

The questions to be addressed in this study are: Is the mentally retarded population in this study growth retarded as has been documented in other studies of mentally retarded children? Does growth retardation in the mentally retarded population also result in deviation from the norm in TSF, MAC and MAMC? Which of the anthropometric measurements correlates best with the height/weight index, i.e., the ratio obtained by dividing the height (in inches) by the weight (in pounds): TSF, MAC or MAMC? If the subjects are grouped by etiology of mental retardation, is there a significant difference in the growth parameters between the various groups?

HISTORICAL PERSPECTIVE

Severe mental retardation is often associated with neurological involvement including motor, sensory and behavioral abnormalities and accompanied by brain lesions. Neurological involvement and observable pathology is less often seen in milder forms of mental retardation (Stein and Kassab, 1972). Seventy-five percent of the retarded fall into the mild retardation group (Clarke and Clarke, 1977). Many of the mentally retarded children are of short stature and exhibit abnormal growth patterns.

Factors which have been implicated in causing mental and/or physical growth retardation are: Malnutrition, intrauterine insults, environmental factors (i.e., social factor) and genetic factors. Extensive studies on malnutrition appear to show that malnutrition in early life retards cell division and ultimately decreases the number of cells in various parts of the brain and body. Winick and Noble (1966) proposed that undernutrition during the growth period in any tissue when cell division (hyperplasia) occurs will lead to a permanent reduction in the number of cells attained. On the other hand, undernutrition during the hypertrophic phase would result in a smaller cell size,

but normal cell size could be attained with adequate later nutrition.

In humans, brain cell number increases rapidly from conception until twelve to fifteen months of age. Two-thirds of the adult head circumference is achieved by twenty-four months of age (Nellhaus, 1968). Winick (1968) found a decreased DNA content suggesting a reduced number of cells in infant brains who died from marasmus during the first year of life. Graham (1967), and Stoch and Smythe (1967) concluded that growth retarded children also exhibited a decreased head circumference which could affect the size and effectiveness of the brain. Dodge, Prensky and Feigin (1975) reported that numerous examples of dramatic increases in head size following rehabilitation are documented in the North American literature. In other parts of the world, nutritional rehabilitation has also been demonstrated to accelerate head growth, although serial observations are surprisingly few (Dodge et al., 1975).

Dobbing (1970) felt that the brain was most likely to be growth retarded if malnutrition occurred at a time of growth spurt and that different degrees of growth retardation would be noticeable depending on the growth velocity at the time of undernutrition. The author also noted that growth in synaptic connections between cells and myelination

took place at the time of growth spurt, and these might also be affected by undernutrition.

Studies have shown that undernutrition during growth periods of the rat would retard growth, but nutritional therapy could rehabilitate the animal to normal body and brain weight. In general, the later during the growth period the nutritional insult occurs, the more likely catch up growth will take place (Winick and Brasel, 1980). Catch up growth has also been reported in humans. A child may grow at a rate above the expected rate for some time following undernutrition or illness during which growth had ceased or was decreased (Pipes, 1977). Children with celiac disease between the ages nine and fifteen months demonstrated complete catch up growth within three years following initiation of treatment (Barr, Shmerling and Prader, 1972). Cravioto and Robles (1965) noted catch up growth to developmental level in older Mexican children but not in those who were treated for acute nutritional disorders prior to one year of age. Eid (1971) found a group of failure-to-thrive children to be significantly growth retarded during the first year regardless of whether they had been treated or not. The author concluded that the interaction of the duration of illness and undernutrition were the cause of the growth retardation. Marks, Borns,

Steg, Stine, Stroud and Vates (1978) were able to demonstrate catch up brain growth in two children with the use of CAT scan.

It has also been suggested that malnutrition may affect the maturation of the central nervous system. Cravioto and Delicardie (1968) believed that protein deficiency could result in structural lesions of the nervous system which would lead to mental damage. Several investigators have reported encephalographic (EEG) abnormalities in a large number of malnourished children which did not become normal even if the children were rehabilitated (Nelson and Dean, 1959). Platt, Pampiglione and Stewart (1965) noted retardation in motor development, EEG abnormalities and neuromotor incoordination in pigs on protein-deficient diets. Developmental delays in motor and language abilities have been reported in malnourished children, and it has been suggested that permanent damage in language ability may result if malnutrition occurs before the age of six months (Stoch and Smythe, 1968). Monckeberg, Tisler, Toro, Gattas and Vega (1972) studied children in Chile who were diagnosed and treated for severe marasmus at one to five months of age. Follow up studies six years later showed significantly lower weight, head circumference and I.Q. when compared to normal children. In children with cystic fibrosis,

differences in I.Q. have been shown to disappear after five years of age suggesting that social factors may compensate for initial undernutrition (Lloyd-Still, Hurwitz, Wolff, and Shwachman, 1974). Latham (1974) stated that there is no real evidence to show that brain size is positively correlated with higher intelligence or superior mental functioning.

Stein, Susser, Saenger and Marolla (1972) studied the effects of the Dutch World War II famine on birth weight and rates of mental retardation in men at age nineteen years who had been born either before, during or following the famine. Although the birth weight decreased during and shortly following the famine, the study showed no significant increase in mild or severe mental retardation nor a significant decrease in intelligence. The most striking variation observed was the lower intelligence score of the manual working class when compared to nonmanual workers. This suggests that, in this case, environmental factors were more important than nutritional factors. Stein et al. pointed out that these results cannot be generalized to the effects of chronic malnutrition with its different nutritional deficiences seen in developing countries.

Recently, the effects of intrauterine growth retardation and its role on fetal growth have been studied (Rosso, 1980). Intrauterine growth retardation may have various

causes. Infection, such as rubella, herpes and syphilis have been shown to cause a decrease in placental growth. Maternal medical abnormalities, such as hypertension and renal disease, may possibly correlate with uterine growth retardation (Irving, 1979). Battaglia (1978) noted that the amount of placental tissue at a given time defines the upper limit of fetal size. Although it is possible to have a small, growth retarded fetus in a large placenta, a large fetus is not possible without a large placenta. Rosso (1980) reviewed maternal malnutrition and placental growth and found that undernourished women and women living in poverty conditions in developing countries have a lower mean placental weight than well nourished women and women from higher income groups. The author concluded that the wide difference in placental weight reflected variations in the severity of maternal malnutrition during pregnancy. Although famine conditions resulted in more severe food deprivation than chronic moderate undernutrition, Rosso felt that the higher placental weight in Dutch women affected by famine during the second and third trimester of pregnancy could be explained by a better nutritional status at the beginning of pregnancy. The author postulated that fetal growth retardation may result when maternal malnutrition leads to reduced blood volume expansion and an inadequate increase in cardiac output which in turn causes a decreased

placental blood flow. The decreased placental blood flow results in reduced placental size and reduced nutrient transfer to the fetus.

Sinclair and Coldiron (1969) pointed out that viral infection can cause a deficit in the number of cells in many organs. Boue and Boue (1969) studied embryonic cells in culture with rubella virus. The rubella virus inhibited cellular division and increased chromosomal breaks which lead to a proportion of the cells not being viable during later duplication. Naeye and Blanc (1965) studied infants with congenital rubella dying in the first weeks of life and found that all organs, including the brain, were growth retarded as a result of mitotic inhibition. Naeye (1967) also found a reduced number of normal sized cells in infants with congenital cytomegalic inclusion disease suggesting arrest of cell multiplication along with viral cytopathic activity which the author suggested may be a feature of chronic viral infections of the fetus.

Prolonged pre- and perinatal hypoxia of the animal and human fetus also appears to cause growth retardation in the offspring (Sinclair and Coldiron, 1969). Drugs taken during pregnancy or given to the infant may also retard fetal and infant growth. The administration of tetracycline has been documented to inhibit pre- and postnatal growth (Cohlan, 1963). Cigarette smoking, narcotic addiction and

and irradiation are other factors which appear to reduce birth weight in man (Sinclair and Coldiron, 1969). Meyer, Jonas and Tonascia (1976) found twelve percent of the babies whose mothers smoked twenty or more cigarettes per day were below 2500 g compared to 4.7 percent of the babies of non-smokers. Lower birth weight appeared to be due to a direct effect of the mother's smoking.

Perkins (1977) pointed out that poverty fosters overcrowding, poor hygiene, ignorance and superstition which contribute to malnutrition and disease and may result in both physical and mental retardation. The child born in the lower socioeconomic class is affected by the cumulative ill effects of poverty. Frequently his mother has been faced with lifelong poor nutrition and lack of education.

Many genetic disorders have been associated with mental and physical growth retardation. Down's Syndrome, Turner's Syndrome and Prader-Willi are examples of genetic disorders which result in both mental and physical retardation (Palmer and Ekvall, 1978).

Frequently the mentally retarded child's nutritional status is suboptimal. It is difficult to determine, however, if the growth retardation is the result of the disorder or cause which affected his mental ability, or whether postnatal nutritional status is actually compromising growth in the retarded. Factors which have been

shown to affect the nutritional status of the retarded are: Feeding problems, drug therapy, and social and psychological causes.

Feeding problems are often the result of neuromuscular abnormalities. Neuromuscular development follows in an orderly pattern. The child with central nervous system damage may exhibit delayed or abnormal responses in the coordination of sucking, swallowing and breathing. For example, the child may exhibit a bite reflex, i.e., exhibit strong clamping down on the spoon with his teeth. This will interfere with food intake and results in delays in chewing development (Palmer and Horn, 1978). Neuromuscular abnormalities may also prevent the child from feeding himself, i.e., being able to bring the spoon to the mouth. He will, therefore, depend on others to feed him. In addition gross structural anomalies, such as cleft palate, may also interfere with food intake. Malocclusion of teeth may cause difficulty in biting and chewing (Palmer and Horn, 1978).

Drug therapy in the retarded child may also affect his nutritional status. Nutritional disturbances associated with anticonvulsant drugs include decreased serum folate levels (Biale, Lewenthal and Ben-Adereth, 1976; Moore and Ball, 1972), and altered vitamin D and calcium metabolism (Peterson, Gray and Tolman, 1976; Offerman,

Pinto and Kruse, 1979). Loss of appetite has been reported with chlorpromazine and primidone (March, 1978).

The Committee Report of Nutrition of the American Academy of Pediatrics has estimated that three-quarters of all mentally defective children are born in an environment of poverty (Clarke and Clarke, 1977). Overcrowding, poor hygiene and ignorance associated with poverty (Perkins, 1977) would also affect the nutritional status of the retarded child in the same way it affects the mother. Inadequate maternal care and emotional deprivation are also factors which have been documented to affect the nutritional status and growth of the child (Patton and Gardener, 1962; Olshin, 1968).

Gouge and Ekvall (1975) reported low ascorbic acid and iron intakes, lack of a daily source of fluoride and protein intake of borderline biological quality in handicapped children. Calvert, Vivian and Calvert (1976) found low intakes in calories, calcium, iron, vitamin A and thiamin in a Down's Syndrome population. The Ten State Nutrition Survey and the HANES Study noted low iron, vitamin A and C, and riboflavin intakes in the U. S. population. Malnutrition or danger of it was noted in a considerable proportion of the low income population (Obert, 1978).

It thus appears that the mentally retarded child often

has suboptimal nutrient intake. Impaired digestion and malabsorption have been associated with undernutrition. Enzyme biosynthesis appears to be affected by malnutrition and may negatively influence metabolism and growth. Barbezat, and Hansen (1968) noted reduced output of pancreatic enzymes, especially lactase, which may lead to diarrhea and malabsorption.

It has been demonstrated that malnutrition affects the body's immune response. Suskind (1980) noted that the ability of the malnourished child to respond to an antigenic stimulus has been compromised in certain instances. The risk of infection has often enhanced malnutrition, and infection in turn exaberates the poor nutritional status. The role infection plays in jeopardizing the nutritional status is increasingly being recognized (Mata, 1979). Poor nutrition and infection have synergistic effects on the poor health status of the malnourished child.

It is thus suggested that growth in humans may be affected by pre- and postnatal nutrition, infection or trauma during pregnancy, and may be influenced by undernutrition during childhood. It is, however, extremely difficult to separate biological, nutritional, social, and educational factors and determine the extent of each as a cause of growth and mental retardation.

Regardless of the cause of mental retardation, numerous studies have documented growth retardation and abnormal growth patterns in the mentally retarded. Decreased physical size, delayed sexual development, decreased head circumference and metabolic aberrations have been reported by several investigators.

Physical growth in the retarded was studied by Tarbell in 1883. Tarbell compared stature and weight of normal and institutionalized retarded children and demonstrated that the retarded were lighter in weight and shorter in stature (Bailit and Whelan, 1967). Goddard (1912) showed that stature and weight could be positively correlated with intelligence. Flory (1936) also reported that mentally retarded boys were typically below normal in height and weight for their age. The author found delayed sexual development and concluded that the greater the degree of mental retardation, the greater the retardation in development. The author felt that the retarded grew at a slower rate but for a longer period of time and concluded growth at an absolute smaller size.

Dutton (1959) found that a "metabolic" group, who originally was separated from the remaining patients investigated because of an absence of any evidence of organic pathology but did show the common feature of depressed height, was also characterized by retarded development and

biochemical anomalies. Biochemical abnormalities were noted in steroid excretion, urinary non-protein nitrogen excretion and serum alkaline phosphatase. Retarded development included decreased height and weight, delayed skeletal development, delayed onset of puberty and development of genitals. Dutton concluded that a basic biochemical abnormality seemed to underlie the widespread disturbance of growth and maturation. In his study, a nonpathological group had no demonstrable organic symptoms and was normal in growth and physical development. Dutton concluded that these subjects appeared to be a normal variant of the lower end of the distribution curve of intelligence.

Culley, Jolly and Mertz (1963) reported that a group of patients without motor dysfunction were significantly shorter than a normal population for their age, weighed less but deviated only slightly from normal body build. Patients with motor dysfunction had a greater tendency to fall below the third percentile for weight and height. About one half of the patients with motor dysfunction also appeared to be underweight for their height. The tendency toward being underweight increased with the severity of the motor dysfunction. They concluded that severe motor problems interfered with chewing and swallowing, and thus decreased the caloric intake of these patients.

Kugel and Mohr (1963) also found an excess of retarded children who were below the seventeenth percentile for height and weight and concluded that the greater the degree of mental retardation, the greater the degree of growth failure.

Mosier, Grossman, and Dingman (1962) found a fairly consistent delay in sexual development in the lower ranges of I.Q. The males tended to mature later than the female children. Females with mongolism showed the most striking pattern in that they were more delayed in the onset of sex features.

Mosier, Grossman and Dingman (1965) also noted that the bulk of the evidence favored the conclusion that the physical growth rate in mentally defective children proceeds at a slower rate than in normal children and continues for a longer period of time. In their study, body weight, crown-heel height, symphysis-heel height, biacromial diameter and bicristal diameter had smaller dimensions than normal children in both sexes and the degree of impairment related to the degree of mental deficit. The Down's Syndrome children were most stunted, and the difference was due to shortening of the lower extremities.

Thelander and Pryor (1966) also showed specific growth abnormalities for the Down's Syndrome boys and girls which included short stature due to shorter legs, lack of head

growth after age two and one half to three years, and microcephaly. Dimensions of the face lagged in both height and width resulting in the Mongoloid retaining his "baby face". Trunk diameter and sitting heights were close to normal for each age and sex group.

Pryor and Thelander (1967) found children with multiple congenital anomalies were shorter than average. The height curves lagged progressively after age five, and there was no adolescent growth spurt. Cerebral palsy children were within normal limits until ten years of age when standing height and trunk measurements began to slump. Head measurements were smaller than average, but few of them were microcephalic. Children with minimal brain damage were within normal limits for all measurements.

Van Geldern (1962) suggested that the time of insult is critical to the differential growth and that normal growth could occur if mental deficiency took place postnatally. Pozsonyi and Lobb (1967), however, found that patients with severe postnatal infection and consequent encephalopathy were among the most stunted subjects. Pozsonyi and Lobb also reported that linear and skeletal growth of a "nonpathological" group was normal, while Down's Syndrome and disorders of metabolism resulted in the most severe growth retardation.

Twenty to forty percent of a group of familial retarded studied by Bailit and Whelan (1967) were two or more standard deviations below the normal population mean for height, head length, biacromial and biiliac diameters. The boys were relatively smaller than the girls; however, the difference was not significant. They also found that I.Q. and size were independent within the sample.

Roberts and Clayton (1969) in studying a group of dwarfed children noted three types of early weight gain: (1) within normal limits, (2) within normal limits followed by a falling off later and (3) no initial progress at all, i.e., failure to grow properly from the beginning.

Approximately one third of the subjects in Gouge and Ekvall's study (1975) were below the third percentile for height and weight.

Culley and Middleton (1969) reported that institutionalized retarded children who were ambulatory had energy requirements similar to those of normal children if height was used as a standard for estimating caloric needs. Because many of the children in their study had short stature, their caloric requirements were less than those of normal children of the same age. The authors found that motor dysfunction severe enough to cause children to be nonambulatory did result in significantly lower caloric requirements per centimeter of height. Mertz and associates,

on the other hand, reported that a group of emaciated children in an institution for the mentally handicapped had energy requirements in excess of normal children. Even though such children consumed caloric intakes recommended for their age, they remained emaciated (Pipes, 1977).

Thus it appears that a large proportion of the mentally retarded children exhibit growth retardation and growth failure. Dobbing (1970) has explained the abnormal growth by suggesting that, regardless of the cause of the insult, "the entire growth program appears to have been reset on a different and lower trajectory, an effect which long outlasts the period of restriction... the brain may therefore react nonspecifically in a 'final common path' manner to any restriction on its proper development during its growth spurt" (p. 415).

Because of the abnormal growth in the retarded population, it is difficult to evaluate the nutritional status of the retarded child. Hamill, Drizd, Johnson, Reed, Roche, and Moore (1979) supported the use of percentile growth curves (Appendices 1 and 2) by stating:

> "Anthropometry is an effective and frequently performed child health and nutrition screening procedure. The value of physical growth data depends on their accuracy and reliability and how they are recorded and interpreted... The new National Center for Health Statistics (NCHS) percentiles can be used to improve identification of potential health and nutritional problems and to facilitate the epidemiological comparison of one group with others" (p. 607).

The authors state that measurements between the twenty-fifth and seventy-fifth percentile are likely to represent normal growth. Children with values above the ninetieth and below the tenth percentile should be referred for further evaluation.

Although no single parameter can precisely assess the nutritional status, upper arm dimensions can greatly contribute to the diagnosis as well as the evaluation of nutritional management. Arm measurements are easily performed techniques requiring a minimum amount of time and equipment. The TSF provides an estimate of body fat and the MAMC an indication of body muscle mass (Jelliffee, 1966). The MAMC is calculated from the MAC and TSF.

Frisancho (1974) supported the need for updated standards for TSF and MAMC norms for assessment of nutritional status by stating:

> "Nutritional status evaluations are usually made through comparison of the estimated upper arm muscle circumference of a given population against the 'standards'. It must be noted, however, that the standard of muscle circumference of children six to fifteen years was estimated from the arm circumference of British children (reported in 1955), and the triceps skin fold of children in the United States obtained prior to World War II and reported in 1941. It is quite evident that normative data derived through this procedure are not applicable in terms of time or population. Clearly, there is a crucial need for the development of more appropriate estimates of muscle size" (p. 1052).

Frisancho used data from the Ten State Nutrition Survey to provide updated estimates of upper arm muscle size

derived from the arm circumference and triceps skinfold of the white population in the United States. The standards for TSF, MAC and MAMC developed by Frisancho are included in Appendix 2 and 3.

The procedure used in obtaining MAC and TSF measurements for the Ten State Nutrition Survey was as follows (Frisancho, 1974): (1) Upper arm circumference (millimeters) was measured to the nearest millimeter with a steel tape on the right arm while hanging relaxed. The measurement was taken midway between the tip of the acromion process (shoulder) and the olecranon process (elbow), (2) Triceps skin fold (millimeters) was measured to the nearest tenth of a millimeter with a Lange Skinfold Caliper having a pressure of 10 g/mm^2 of contact surface area. The measurement was taken on the back of the arm and midway between the point of the acromion and the olecranon process with the arm hanging relaxed, (3) Arm muscle circumference (mm) was derived by computation as follows: Arm muscle circumference (mm) = arm circumference - π (triceps skinfold). A similar procedure was used by the First Health and Nutrition Examination Survey (HANES) 1971-72 (Health Resources Administration, 1975). The HANES study specified that the skinfold calipers were applied 1 mm below thumb and forefingers, and the fingers were not released when taking the skinfold measure.

Although the Ten State Nutrition Survey reported right arm measurements only, Burgert and Anderson's study (1979) suggested that those measurements can be considered equivalent to left arm measurements. Roche (1978) recommended the use of Lange Skinfold Calipers because appropriate reference data on U. S. children using these calipers are available.

The HANES study reported weight, height and skinfold measurements of white and black children with regard to race, sex and income. The TSF measurements were larger in white children for both sexes. Higher triceps skinfold measures were noted in children from income groups above the poverty level, but subscapular skinfold measures were not consistently higher (Health Resource Administration, 1975).

Weinsier, Butterworth and Sahm (Butterworth and Weinsier, 1980) developed triceps skinfold reference graphs (adapted from data of Seltzer and Mayer, Tanner and Whitehouse, and Frisancho) which are included in Appendix 5 and an arm muscle circumference reference graph (adapted from Frisancho) which is found in Appendix 6. The triceps skinfold measures are divided into minimal, low, normal, and excessive fat reserves. The mid arm circumference was divided into low and normal muscle reserves. The authors pointed out that patients with a MAMC in the low muscle

reserve range are likely to be severely malnourished and that arm muscle depletion is possible despite values in the normal range.

Thus, while numerous studies are found in the literature documenting growth retardation in the retarded population, none of the investigations have examined TSF, MAC and MAMC in the mentally retarded population.

HYPOTHESIS

The null hypotheses were: (1) There will not be a significant difference in height, weight, TSF, MAC and MAMC between a mentally retarded population and their normal age counterparts. The minimum level for rejecting the null hypothesis in this study will be $p < 0.05$.

(2) There will not be a significant relationship between the height/weight index and (1) TSF, (2) MAC and (3) MAMC. The minimum level for rejecting the null hypothesis will be $p < 0.05$.

(3) There will not be a significant difference in height, weight, TSF, MAC and MAMC between various etiologies of mental retardation. The critical level of statistical probability used to reject the null hypothesis will be $p < 0.05$.

RESEARCH DESIGN

Subjects

Mentally retarded patients between the ages ten and eighteen institutionalized at a residential care facility (i.e., a long term care facility providing twenty-four hour residential and health care, and educational and special programs) were selected based on a review of their medical chart to determine the etiology of their retardation. For the purpose of this study, an I.Q. of seventy or less was considered mentally retarded. The subjects included in this study were those with retardation due to pre-, peri- and postnatal trauma and infection. Not included were those with retardation due to chromosomal aberration and hereditary disorders including inborn errors of metabolism. Subjects with unknown etiology of their retardation were included, however. Ninety-two (42.6 percent) of the subjects fell in this category. Excluded from the study were subjects with a height quotient of .6 or less. This eliminated subjects with extreme growth retardation. Since an I.Q. of seventy or less is a criteria for placement at the residential care facility, all subjects are functioning in the retarded range. All patients meeting the above criteria were included in this study. The subjects were grouped by age

and by sex. For example, the ten year old group included boys or girls with an age of nine years six months to ten years five months. The study included 216 subjects of which 112 were male and 104 female. Fifty-two percent of the subjects were Caucasian, thirty-nine percent were black, eight and one half percent Mexican-American and one half percent Oriental. Twenty-nine percent of the subjects were nonambulatory.

Procedure

Following selection, each subject was measured for height to the nearest one quarter inch and weighed to the nearest one quarter pound. An upright scale was used for ambulatory subjects. Nonambulatory subjects were weighed on a bedscale. The scales were balanced prior to each use. A recumbent height was obtained for the nonambulatory subjects.

Lange Skinfold Calipers were employed in this study and the following procedure (Frisancho, 1974) was used to determine the TSF: (1) the midpoint between the acromial process and the tip of the olecranon was marked on the nondominant arm, (2) the fat pad was grasped one centimeter below the marked midpoint, (3) the calipers were placed at the marked midpoint, and (4) the skinfold thickness value was read after a count of three. This measurement was repeated three times; the three scores were averaged and

the averaged mean was reported on the Anthropometric Data Form (Appendix 7). The MAC was measured with an insertion tape at the midpoint between the tip of the acromial and olecranon process (Frisancho, 1974) and was recorded. All TSF and MAC measurements were performed by the investigator to avoid errors in standardization. The MAMC was computed using the values obtained from the TSF and MAC measurements. The formula employed was (Frisancho, 1974): MAMC (mm) = MAC (mm) - $\left[3.14 \times \text{TSF (mm)}\right]$.

The height and weight measurements were compared to the fiftieth percentile of the NCHS growth curves (Hamill et al., 1979), Appendix 1 and 2. The TSF, MAC and MAMC were compared to the fiftieth percentile of each measurement of data obtained from the Ten State Nutrition Survey of 1968-70 (Frisancho, 1974), Appendix 3 and 4.

Statistical Analysis

Multiple t tests of difference between a sample mean and the population mean were used to determine whether height, weight, TSF, MAC and MAMC differed significantly from those of the normal population (fiftieth percentile) at the $p < 0.05$ level.

A Pearson's Product-Moment Correlation (r) was used to determine the relationship, if any, between the height/weight index and each of the growth parameters: TSF, MAC and MAMC.

The subjects were group by etiology of mental retardation. The height, weight, TSF, MAC and MAMC of each subject was converted to a percentage of normal. A one way analysis of variance was used to determine if a significant variation existed in the growth parameters when comparing various etiologies of mental retardation. The minimum level for rejecting the null hypothesis in this study was $p < 0.05$.

RESULTS AND DISCUSSION

Two hundred and sixteen mentally retarded subjects were selected based on etiology of their mental retardation. The subjects were grouped by age and by sex. Only four subjects were included in the eleven year old group for girls because no others were at the residential care facility.

After review of the medical chart, each subject was measured for height, weight, TSF, and MAC. With a few exceptions the four measurements were obtained at the same time. Some difficulty was encountered in measuring the severely scoliotic children (i.e., children with curvature of the spine) and those with muscle contractions of the extremities. The height was estimated on these subjects by following the midline from head to toe. The hyperactive children were difficult to weigh on an upright scale. In a few instances, where they could not be made to stand still long enough to be weighed, the children were weighed by having them sit on the bedscale. This was assumed to be a more accurate means of weighing these subjects. The TSF measurement presented a problem in a few spastic children. In these subjects, any type of stimulation could result in a muscle contraction. Muscle contracture might invalidate

the TSF measurement. The decision was made on a case by case basis if two measurements were sufficient or if three measurements were to be taken several minutes apart (to allow the muscle to relax).

One of the foremost problems with studies of growth retardation in mentally retarded is the difficulty of assigning retarded patients to meaningful and homogeneous diagnostic categories (Bailit and Whelan, 1967). This study included subjects with retardation due to pre-, peri- and postnatal trauma and infection. Also included were subjects with retardation due to unknown etiology of their retardation. Forty-two percent of the subjects fell into this category. This study did not consider social class, birthweight or height of the parents. These factors may have an effect on the size of the offspring. This study also did not consider the I.Q. of the subjects, i.e., test the hypothesis that growth retardation increases with decreasing I.Q.

Table 1 shows the mean height of the normal and retarded girls. All differences between the two groups were significant ($p < 0.05$) except for the eleven year old group. The $\underline{t}$ test was not significant because of the small sample size ($N = 4$). The height of the normal girls increased from 54.5 inches at age ten to 64.5 inches at age eighteen. The retarded girls increased in height from

TABLE 1

MEAN HEIGHT, STANDARD DEVIATION AND DIFFERENCE BETWEEN NORMAL AND RETARDED GIRLS (IN INCHES)

Age	N	Normal (a)	Retarded			
		Mean	Mean and SD	Diff.	% of Normal	sig. of t
10	11	54.5	48.9 ± 3.31	5.6	89.7	.001
11	4	57.0	50.6 ± 3.40	6.4	88.8	**
12	10	59.7	52.8 ± 4.18	6.9	88.4	.001
13	10	61.9	52.0 ± 3.52	9.9	84.0	.001
14	10	63.2	56.0 ± 4.60	7.2	88.6	.001
15	15	63.7	56.7 ± 3.11	7.0	89.0	.001
16	16	64.0	58.1 ± 2.65	5.9	90.8	.001
17	14	64.3	59.3 ± 2.93	5.0	92.2	.001
18	14	64.5	58.2 ± 2.13	6.3	90.2	.001

(a) From Hamill et al., 1979
** Sample size too small to be significant

48.9 inches at age ten to 58.2 inches at age eighteen. The height of the retarded girls at all ages fell below the fifth percentile when plotted on the NCHS growth charts (Appendix 8). The height was 89.7 percent of normal at age ten and 90.2 percent at eighteen years. The thirteen year old retarded girls deviated slightly more with a height of 84.0 percent of normal. This greater difference may possibly be the result of the normal thirteen year old girls having reached the adolescent growth spurt while the retarded have not. The height of the retarded girls paralleled the normal girls but remained at a lower level throughout the period examined.

Table 2 represents the mean height for normal and retarded boys. The difference in height between the groups was significant ($p < 0.05$) at all ages. The height of the normal group increased progressively with age from 54.2 inches at ten years of age to 69.7 inches at age eighteen. The retarded group increased in height from 47.7 inches at age ten to 62.9 inches at age eighteen. The twelve year old retarded boys measured 49.4 inches compared to the 50.8 inches noted in the eleven year old boys. No explanation can be made for this difference in height. The mean height of the retarded group at all ages fell below the fifth percentile when plotted on the NCHS growth charts (Appendix 9), and it appears to parallel the height of the normal

TABLE 2

MEAN HEIGHT, STANDARD DEVIATION AND DIFFERENCE BETWEEN NORMAL AND RETARDED BOYS (IN INCHES)

Age	N	Normal (a)	Retarded			
		Mean	Mean and SD	Diff.	% of Normal	sig. of t
10	11	54.2	47.7 ± 2.02	6.5	88.0	.001
11	10	56.5	50.8 ± 3.43	5.7	89.9	.001
12	8	59.0	49.4 ± 2.74	9.6	83.7	.001
13	10	61.7	54.4 ± 4.38	7.3	88.2	.001
14	12	64.3	56.4 ± 4.33	7.9	87.7	.001
15	15	66.6	59.4 ± 4.30	7.2	89.2	.001
16	17	68.4	61.4 ± 3.46	7.0	89.8	.001
17	17	69.4	61.8 ± 3.59	7.6	89.0	.001
18	12	69.7	62.9 ± 3.99	6.8	90.2	.001

(a) From Hamill et al. 1979

group but all values of the retarded fell lower than the normal values. The ten year old retarded boys had a height of 88.0 percent of the normal boys and the difference remained constant through eighteen years of age when the retarded were 90.2 percent of normal. The twelve year old group had a somewhat lower height and it was 83.7 percent of normal. The data suggests that the height of the retarded boys remained retarded throughout the period examined and the retarded boys did not exhibit catch up growth.

Table 3 represents the mean weight of normal and retarded girls. All differences between the two groups were significant ($p < 0.05$) with the exception of the eleven year olds. The sample size ($N = 4$) was too small to be significant. The weight of the normal ten year olds was 71.6 pounds and it increased to 124.6 pounds at age eighteen. The retarded girls were 47.7 pounds at age ten and 87.0 pounds at age eighteen. All mean weights fell below the fifth percentile when plotted on the NCHS growth charts (Appendix 10). The difference between the two groups increased from 23.9 pounds to 37.6 pounds. The weight of the retarded girls was 66.6 percent of normal at age ten and 69.8 percent at age eighteen. An exception to this trend was observed in the seventeen year old group. Although the weight of this group was below normal (80.8

TABLE 3

MEAN WEIGHT, STANDARD DEVIATION AND DIFFERENCE BETWEEN NORMAL AND RETARDED GIRLS (IN POUNDS)

Age	N	Normal (a)	Retarded			
		Mean	Mean and SD	Diff.	% of Normal	sig. of t
10	11	71.6	47.7 ± 11.10	23.9	66.6	.001
11	4	81.3	57.5 ± 16.28	23.8	70.7	**
12	10	91.4	61.8 ± 23.65	29.6	67.6	.003
13	10	101.4	64.7 ± 15.15	36.7	63.8	.001
14	10	110.6	71.0 ± 24.47	39.6	64.2	.001
15	15	118.1	80.3 ± 15.82	37.8	68.0	.001
16	16	123.0	88.9 ± 21.18	34.1	72.3	.001
17	14	124.7	100.7 ± 26.13	24.0	80.8	.004
18	14	124.6	87.0 ± 18.29	37.6	69.8	.001

(a) From Hamill et al., 1979
** Sample size too small to be significant

percent of normal) it was noted that the height was below the fifth percentile while the weight fell between the fifth and tenth percentile.

Table 4 shows the mean weight of normal and retarded boys. The difference in weight between the two groups was significant ($p < 0.05$) at all ages. The weight of the normal boys increased with age from 69.2 pounds at age ten to 151.5 pounds at eighteen years. The retarded group increased in weight from 52.0 pounds at age ten to 103.5 pounds at age eighteen. Although the weight of the retarded group increased with age, the increase was smaller than in the normal group. The difference between the two groups was 17.2 pounds for the ten year olds and 48.0 pounds for the eighteen year olds. The weight of the retarded fell at the fifth percentile at age ten when plotted on the NCHS growth charts (Appendix 11) but as age increased the weight fell progressively more below the fifth percentile. The ten year old retarded boys weighed 75.1 percent of normal. By the age eighteen the weight of the retarded had fallen to 68.3 percent of the normal eighteen year olds. The lower weight of the twelve year old boys when compared to the eleven year old retarded boys may be explained by their lower height.

Although the retarded children were shorter in stature, the data suggests that body build in both groups, boys and

TABLE 4

MEAN WEIGHT, STANDARD DEVIATION AND DIFFERENCE BETWEEN NORMAL AND RETARDED BOYS (IN POUNDS)

Age	N	Normal (a)	Retarded			
		Mean	Mean and SF	Diff.	% of Normal	sig. of t
10	11	69.2	52.0 ± 12.00	17.2	75.1	.001
11	10	77.7	59.0 ± 12.56	18.7	75.9	.004
12	8	87.5	57.3 ± 11.78	30.2	65.5	.001
13	10	98.9	74.4 ± 27.32	24.5	75.2	.02
14	12	111.7	75.6 ± 18.71	36.1	67.7	.001
15	15	124.8	89.2 ± 22.15	35.6	71.5	.001
16	17	136.6	96.2 ± 22.16	40.4	70.4	.001
17	17	145.9	95.0 ± 16.46	50.9	65.1	.001
18	12	151.5	103.5 ± 26.75	48.0	68.3	.001

(a) From Hamill et al., 1979.

girls, appears to be normal as can be determined from the fact that both height and weight were below the fifth percentile. A height above the fiftieth percentile and a weight below the fifth percentile would indicate that the subjects were underweight (Mitchell, Rynbergen, Anderson and Dibble, 1976). Although the retarded children appear to have an appropriate weight for height as a group, each subject would still have to be assessed individually to determine appropriate weight for height. It must also be kept in mind that the growth charts represent the population as it exists and not what is ideal or optimal, especially with regard to body weight (Owen, 1978). This is especially true at the upper end of the weight scale.

Table 5 shows the mean TSF for normal and retarded girls. The normal girls showed a rapid increase in TSF from ten millimeters at age eight to seventeen millimeters at age eighteen (Frisancho, 1974). The retarded girls increased from a low TSF of seven millimeters to 12.8 millimeters at age eighteen. An exception to this trend was seen in the seventeen year old retarded group which showed a TSF of 16.2 millimeters or 101.2 percent of normal. This group also had a higher weight than the sixteen and eighteen year old retarded girls. The TSF of the retarded girls also did not differ significantly ($p > 0.05$) for the eleven year old group due to the small sample size ($N = 4$)

TABLE 5

MEAN TSF, STANDARD DEVIATION AND DIFFERENCE BETWEEN NORMAL AND RETARDED GIRLS (IN MM)

Age	N	Normal (a)	Retarded			
		Mean	Mean and SF	Diff.	% of Normal	sig. of t
10	11	12	7.0 ± 1.00	5.0	58.3	.001
11	4	12	8.1 ± 2.25	3.9	67.5	**
12	10	13	7.2 ± 3.99	5.8	55.4	.001
13	10	14	11.3 ± 6.50	2.7	80.7	*
14	10	15	8.6 ± 4.65	6.4	57.3	.002
15	15	16	11.3 ± 5.68	4.7	70.6	.006
16	16	15	11.0 ± 4.56	4.0	73.3	.003
17	14	16	16.2 ± 6.41	.2	101.2	*
18	14	17	12.8 ± 6.54	4.2	75.3	.031

(a) From Frisancho, 1974
* Non significant
** Sample size too small to be significant

and the thirteen year old group. The TSF of this group was 80.7 percent of normal. The data for the TSF suggests that the rapid increase in TSF seen in the normal children at puberty is not observed in the retarded girls (Appendix 12). The lower increase in weight noted in the retarded girls appears, at least in part, to be responsible for the smaller TSF in girls.

Table 6 represents mean TSF for the normal and retarded boys. The TSF in normal boys exhibits a slow increase from eight millimeters at age eight until twelve years when the mean TSF is eleven millimeters. After this age, the TSF declines until seventeen years when a low value of eight millimeters is attained (Frisancho, 1974). The mean TSF of the retarded boys ranged from 6.5 millimeters to eleven millimeters. No clear trend was noted in the retarded boys. Significant differences ($p < 0.05$) were recorded for ten, twelve and fourteen year old boys. All other age groups did not differ significantly ($p > 0.05$). The very small TSF range in boys between ages ten and eighteen (eight to eleven millimeters) appears to account for the nonsignificant differences in TSF (Appendix 13).

Table 7 represents mean MAC for normal and retarded girls. The MAC in the normal group increased from 203 millimeters to 260 millimeters. On the other hand, an increase of 54 millimeters from 176 to 230 millimeters was

TABLE 6

MEAN TSF, STANDARD DEVIATION AND DIFFERENCE BETWEEN NORMAL AND RETARDED BOYS (IN MM)

Age	N	Normal (a) Mean	Retarded Mean and SD	Diff.	% of Normal	sig. of t
10	11	10	6.5 ± 2.30	3.5	65.0	.001
11	10	10	7.8 ± 3.23	2.2	78.0	*
12	8	11	8.7 ± 2.49	2.3	79.1	.038
13	10	10	11.0 ± 5.94	1.0	110.0	*
14	12	10	7.6 ± 3.26	2.4	76.0	.026
15	15	9	10.5 ± 6.79	1.5	116.7	*
16	17	9	9.8 ± 3.34	.8	108.9	*
17	17	8	8.4 ± 3.54	.4	105.0	*
18	12	10	9.7 ± 3.63	.3	97.0	*

(a) From Frisancho, 1974
* Non signficant

TABLE 7

MEAN MAC, STANDARD DEVIATION AND DIFFERENCE BETWEEN NORMAL AND RETARDED GIRLS (IN MM)

Age	N	Normal (a)	Retarded			
		Mean	Mean and SD	Diff.	% of Normal	sig. of t
10	11	203	176.0 ± 12.19	27.0	86.7	.001
11	4	210	192.5 ± 35.45	17.5	91.7	**
12	10	220	188.0 ± 32.49	32.0	85.5	.012
13	10	230	205.1 ± 38.35	24.9	89.2	*
14	10	240	191.3 ± 36.45	48.7	79.7	.002
15	15	245	213.5 ± 31.34	31.5	87.1	.002
16	16	249	221.8 ± 35.56	27.2	89.1	.008
17	14	250	246.5 ± 39.12	3.5	98.6	*
18	14	260	230.0 ± 30.77	30.0	88.5	.003

(a) From Frisancho, 1974
* Non significant
** Sample size too small to be significant

seen in the retarded. The MAC of the seventeen year old girls was 246.5 millimeters or 98.6 percent of normal. The t test was nonsignificant ($p > 0.05$). A nonsignificant difference ($p > 0.05$) was also seen in the eleven year old group due to the small sample size and the thirteen year old group. It appears that the retarded girls begin and end at a lower level of MAC, but the difference between normal and retarded does not increase with age (Appendix 14).

Table 8 shows the mean MAC of the normal and retarded boys. The MAC for the normal boys increased from 200 millimeters at age ten to 292 millimeters at age eighteen. The MAC of the retarded boys showed an increase of 58.5 millimeters from 181.5 millimeters at age ten to 240.0 millimeters at age eighteen. The eleven and thirteen year old retarded groups did not differ significantly ($p > 0.05$) from the normal group. All other age groups showed a significant difference ($p < 0.05$) when compared to normal. The difference between normal and retarded increased steadily from 90.8 percent at age ten to 82.2 percent of normal at age eighteen. Exceptions were seen in the eleven and thirteen year olds which showed a MAC of 93.8 and 94.3 percent of normal, respectively.

The MAC in normal boys increased by forty-six percent between the ages ten and eighteen. In comparison the increase in MAC in the retarded was thirty-two percent.

TABLE 8

MEAN MAC, STANDARD DEVIATION AND DIFFERENCE BETWEEN NORMAL AND RETARDED BOYS (IN MM)

Age	N	Normal (a)	Retarded			
		Mean	Mean and SD	Diff.	% of Normal	sig. of t
10	11	200	181.5 ± 21.90	18.5	90.8	.019
11	10	208	195.2 ± 34.79	12.8	93.8	*
12	8	216	196.5 ± 7.35	19.5	91.0	.001
13	10	230	217.0 ± 31.16	13.0	94.3	*
14	12	243	202.2 ± 15.04	40.8	83.2	.001
15	15	253	217.3 ± 37.37	35.7	85.9	.002
16	16	262	220.5 ± 29.83	41.5	84.2	.001
17	17	275	223.7 ± 17.16	51.3	81.3	.001
18	12	292	240.0 ± 24.13	52.0	82.2	.001

(a) From Frisancho, 1974
* Non significant

This suggests that the retarded boys lag behind the normal boys in MAC (Appendix 15), and the small increase in MAC seen in the retarded boys appears to correlate with the smaller increase in weight seen in the retarded.

Table 9 shows the mean MAMC for normal and retarded girls. The MAMC for the normal girls increased from 163 millimeters at age ten to 205 millimeters at age eighteen. The increase in MAMC in the retarded girls was 37.8 millimeters from 154.0 millimeters at age ten to 191.8 millimeters at age eighteen. Significant differences ($p < 0.05$) in MAMC were seen at the ages ten, fourteen and fifteen. The MAMC was nonsignificant in all other groups. The MAMC for the retarded ranged from 85.2 percent to 99.9 percent of normal. The seventeen year old group had a high MAMC (99.9 percent of normal). Only one group showed a MAMC of less than 90 percent, i.e., 85.2 percent for the fourteen year olds (Appendix 16).

The data for the retarded girls suggests that the difference (decrease) in MAC is due to the decreased TSF and not due to the MAMC. The retarded girls compared favorably to the normal population with regard to body protein reserve as measured by the upper arm circumference. Caloric reserve of the retarded girls is much lower than normal as is indicated by the below normal body weight and TSF measurement.

TABLE 9

MEAN MAMC, STANDARD DEVIATION AND DIFFERENCE BETWEEN NORMAL AND RETARDED GIRLS (IN MM)

Age	N	Normal (a)	Retarded			
		Mean	Mean and SD	Diff.	% of Normal	sig. of t
10	10	163	154.0 ± 9.54	9.0	94.5	.001
11	4	171	167.3 ± 28.80	3.7	97.8	**
12	10	179	165.8 ± 21.88	13.2	92.6	*
13	10	185	169.7 ± 21.70	15.3	91.7	*
14	10	193	164.4 ± 29.71	28.6	85.2	.014
15	15	195	178.3 ± 17.50	16.7	91.4	.002
16	16	200	187.3 ± 24.13	12.7	93.7	*
17	14	196	195.8 ± 22.82	.2	99.9	*
18	14	205	191.8 ± 26.37	13.2	93.6	*

(a) From Frisancho, 1974
* Non significant
** Sample size too small to be significant

Table 10 shows the mean MAMC of the normal and retarded boys. The MAMC for the normal boys increased from 168 millimeters to 258 millimeters. The retarded boys showed an increase in MAMC of 48.7 millimeters from 161.0 to 209.7 millimeters. The difference between normal and retarded was 7.0 millimeters at age ten and 48.3 millimeters at age eighteen. The MAMC was 95.8 percent of normal in the ten year olds and it progressively decreased to 81.3 percent of normal at age eighteen (Appendix 17). It is therefore concluded that the decreased MAC in the retarded boys is due to a below normal MAMC rather than a decreased TSF. The lower weight in the retarded when compared to normal can in part be explained by the small muscle mass. The retarded boys therefore appear to have a lower protein reserve than normal boys.

The increase in the MAMC between the ages ten and eighteen was 53.5 percent in the normal boys and 30.0 percent in the retarded. By maturity, the sexual dimorphism in the muscle area is 56 percent (Frisancho, 1974). It thus appears that the retarded boys do not exhibit the large increase in muscle mass which is seen in the normal boys at puberty. The arm muscle mass is approximately equal between the retarded boys and girls (if it is assumed that bone size is equal). Thus it appears that sexual dimorphism is less apparent (or practically nonexistent) with regard

TABLE 10

MEAN MAMC, STANDARD DEVIATION AND DIFFERENCE BETWEEN NORMAL AND RETARDED BOYS (IN MM)

Age	N	Normal (a)	Retarded			
		Mean	Mean and SD	Diff.	% of Normal	sig. of t
10	11	168	161.0 ± 18.22	7.0	95.8	*
11	10	174	170.8 ± 28.51	3.2	98.2	*
12	8	181	169.1 ± 12.58	11.9	93.4	.032
13	10	195	182.4 ± 18.57	12.6	93.5	*
14	12	211	178.3 ± 15.52	32.7	84.5	.001
15	15	220	184.3 ± 27.29	35.7	83.8	.001
16	17	229	189.6 ± 23.73	39.4	82.8	.001
17	17	245	197.2 ± 17.89	47.8	80.5	.001
18	12	258	209.7 ± 27.73	48.3	81.3	.001

(a) From Frisancho, 1974
* Non significant

to upper arm size.

Although the retarded boys have a slightly higher weight, MAC and MAMC than the retarded girls, the difference is comparatively small. Lack of physical activity due to ambulatory problems may explain the decreased muscle mass in some instances, but it would not explain the significant difference seen between the retarded and normal boys. Therefore, it is concluded that other unknown causes account for the significant decrease in the muscle mass of the retarded boys.

Figures 1 and 2 show the TSF reference graph (Butterworth and Weinsier, 1980) for the girls and boys, respectively. With the exception of the ten year old boys, all groups fell into the "normal fat reserves" category. While the TSF ranged from 58.3 to 116.8 percent of the normal children of the same age, the reference graph suggests that, as a group, the retarded children fall within the normal range of fat reserve. However, the children would have to be assessed individually to determine low, normal and high fat reserves. Quite a few retarded children not included in this study because of extreme growth retardation with TSF measures of two to four millimeters have not responded to nutritional rehabilitation.

Figure 3 shows the MAMC reference graph (Butterworth and Weinsier, 1980) for males and females. The MAMC

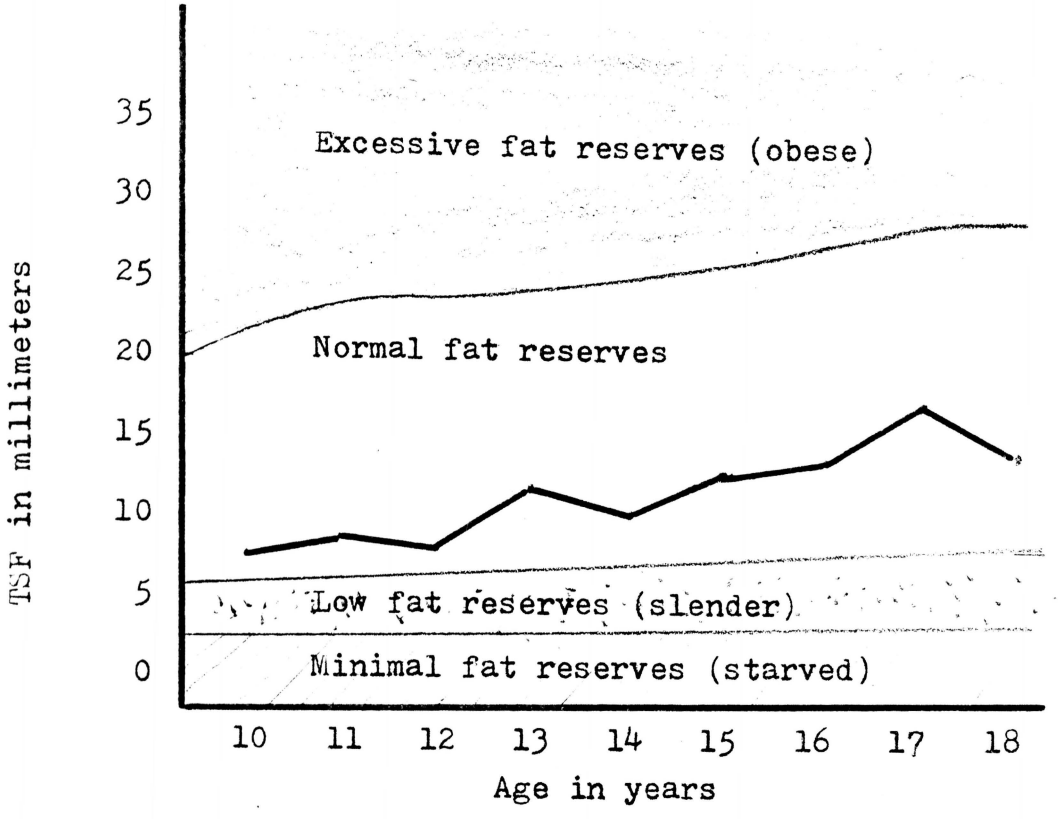

Fig. 1. Triceps Skinfold reference graph for girls with mean TSF of retarded girls between ages 10 and 18 (Butterworth and Weinsier, 1980).

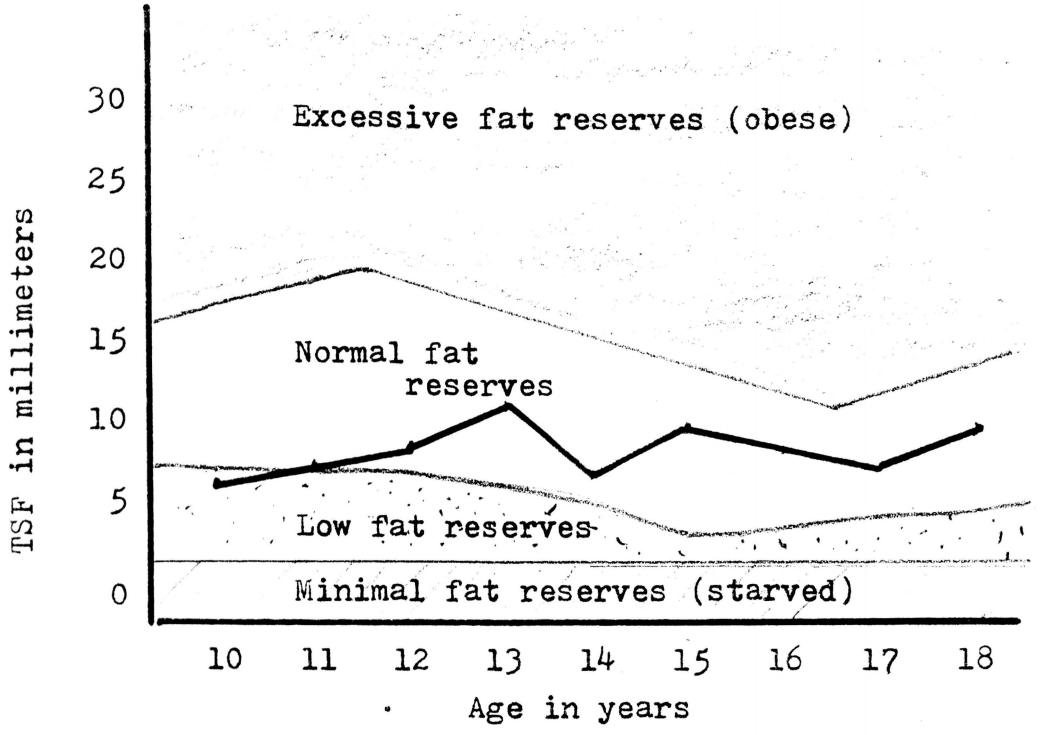

Fig. 2. Triceps skinfold reference graph for boys with mean TSF of retarded boys between ages 10 and 18 (Butterworth and Weinsier, 1980).

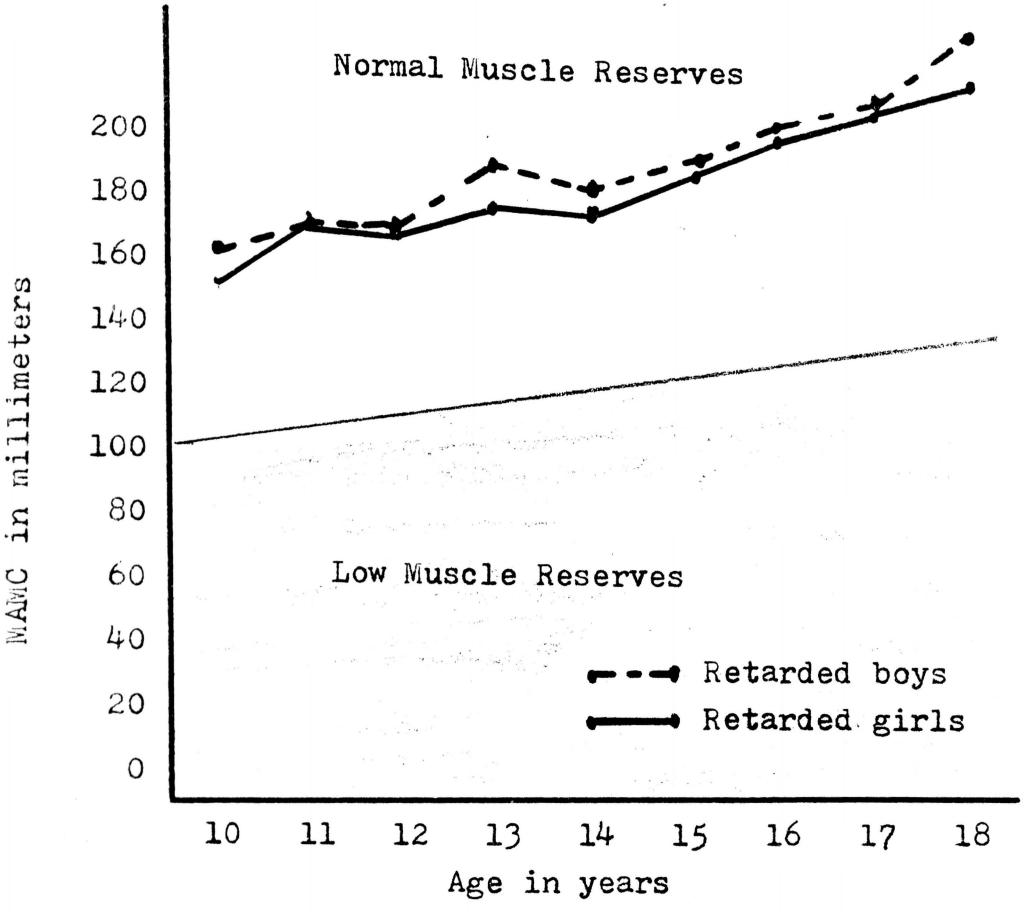

Fig. 3. MAMC reference graph with mean MAMC of retarded boys and girls between ages 10 and 18. (Butterworth and Weinsier, 1980)

measurement for the retarded ranged from 80.5 to 99.9 percent of normal. As with TSF, the retarded children, as a group, fall within the normal muscle reserve category. Again it must be emphasized that the children must be assessed individually to determine the degree of their muscle depletion. Butterworth and Weinsier (1980) pointed out that patients with low muscle reserves are likely to be severely malnourished and that arm muscle depletion is possible despite values in the normal range.

Biochemical tests are also used for nutritional assessment. Although these were not included in this study, serum albumin and creatinine levels were recorded, if available, during the chart review.

Serum albumin is a gross indicator of visceral protein status. It decreases slowly during malnutrition, but serum concentrations fall more rapidly in patients with acute metabolic stress (Rowlands and Jensen, 1979). Serum albumin less than 3.5 g/dl suggests protein malnutrition (Halpern, 1979). One hundred and twenty-five subjects had a laboratory value for serum albumin in their chart (within the past six months). Of these subjects, four had a serum albumin of less than 3.5 g/dl (one boy 2.9 g/dl, one girl 2.9 g/dl, two girls 3.4 g/dl). Thus despite the lower protein reserve, as indicated by lower MAMC, the retarded population in this study does not appear to be protein-malnourished.

Decreased muscle mass is also indicated by a decreased serum creatinine level (Halpern, 1979). The normal serum creatinine level is .6-1.2 mg/dl. Of the 125 subjects with laboratory values for serum creatinine, fifty-six (forty-five percent had a value of .5 mg/dl or less and a total of ninety-seven (seventy-eight percent) subjects had a serum creatinine of .7 mg/dl or less. The serum creatine levels in these subjects tend to support the findings of small muscle mass in the mentally retarded subjects.

Tables 11 and 12 represent the correlation coefficients of the height/weight index with TSF, MAC, and MAMC. The height/weight index is a ratio of height per unit of weight. Using the height/weight index instead of height only has the advantage of correlating both height and weight with each of the growth parameters.

High negative correlations between the height/weight index and MAC were obtained for the girls at all ages, and moderately high to high negative correlations were seen between the height/weight index and MAMC. Similar results were noted between the height/weight index and MAC and MAMC for the boys. Exceptions were seen in the twelve year old and seventeen year old boys for MAC and MAMC, and the thirteen year old boys for MAC. Poor correlations were noted in these groups. Although the correlations between the height/weight index and TSF were higher for the girls than the

TABLE 11

CORRELATION COEFFICIENT OF HEIGHT/WEIGHT INDEX WITH TSF, MAC, AND MAMC FOR GIRLS

Age	TSF	MAC	MAMC
	r	r	r
10	-.825	-.813	-.779
11	-.924**	-.985	-.988**
12	-.870	-.989	-.976
13	-.576*	-.760	-.744
14	-.493*	-.862	-.841
15	-.618	-.845	-.888
16	-.725	-.898	-.898
17	-.803	-.888	-.817
18	-.329*	-.818	-.602
Average of all age groups	-.940	-.980	-.970

* Non-significant; all other values are significant at $p \leq 0.05$ level.

** Sample size too small to be significant (N = 4).

The height/weight index is the ratio obtained by dividing the height (in inches) by the weight (in pounds).

TABLE 12

CORRELATION COEFFICIENT OF HEIGHT/WEIGHT INDEX WITH TSF, MAC, AND MAMC FOR BOYS

Age	TSF	MAC	MAMC
	r	r	r
10	-.597*	-.745	-.664
11	-.359*	-.893	-.808
12	.409*	.054*	-.217*
13	-.665	-.865	-.424*
14	-.476*	-.861	-.586
15	-.540	-.765	-.636
16	-.501	-.856	-.849
17	-.711	-.475*	-.019*
18	.147*	-.736	-.704
Average of all age groups	-.627	-.937	-.939

* Non-significant; all other values are significant at $p \leq 0.05$ level.

The height/weight index is the ratio obtained by dividing height (in inches) by the weight (in pounds).

boys, neither showed a high correlation.

These results suggest that MAC and to a slightly lesser extent MAMC are good predictors of a height/weight ratio in the retarded. The data can be interpreted that, as MAC or MAMC increases, the height/weight index will decrease. In other words, as the MAC increases, the height per unit of weight would decrease or the weight per unit of height increase.

Each anthropometric parameter was also analyzed for significance by considering etiology of mental retardation. Causes of retardation or medical diagnosis included in this analysis were: Prenatal infection and trauma; congenital rubella; birth trauma and hypoxia; postnatal infection and trauma; cerebral palsy; and others which included prematurity and microcephaly. Congenital rubella was separated from the prenatal infection and trauma group because of the large number of subjects with congenital rubella. The subjects were grouped by diagnosis. The height, weight, TSF, MAC, and MAMC of each subject was converted to a percentage of normal. A one way analysis of variance on this data was performed. However, none of the results were statistically significant ($p > 0.05$). Therefore, the null hypothesis that there will not be a significant difference in height, weight, TSF, MAC, and MAMC between various etiologies of mental retardation was not rejected. The data suggests that,

regardless of etiology, all retarded subjects in this study are equally growth retarded and show similar growth patterns.

CONCLUSIONS

A mentally retarded institutionalized population between the ages ten and eighteen was examined for differences in height, weight, TSF, MAC and MAMC. Pre- and postnatal factors which may contribute to growth retardation were discussed. Numerous studies documenting growth retardation in the mentally retarded were cited.

Significant differences ($P < 0.05$) were found in height and weight in all age groups and in most age groups in MAC in both retarded boys and girls. The difference in MAC in the retarded girls was due to the lack of increase in TSF at puberty and in the boys due to the lack of increase in MAMC. Based on these results, it was concluded that the mentally retarded girls have a lower caloric reserve and the boys have a lower protein reserve than the normal girls and boys. The MAC correlated better with the height/weight index than did either the MAMC or the TSF.

No significant differences were seen when each growth parameter was analyzed for significance by considering etiology of mental retardation. This suggests that regardless of the etiology of mental retardation, all retarded subjects in this study are equally growth retarded and show similar growth patterns.

Because of the apparent lower caloric and protein reserve found in the mentally retarded children in this study, these children are considered at a much higher nutritional risk than normal children. Acute illness, surgery and other stress situations would result in a much quicker depletion of body protein and caloric reserve and, most likely, also of other nutrients. In stress situations nutritional intervention is, therefore, considered to be more important for the mentally retarded child and should not be delayed if malnutrition is to be avoided. When height, weight, TSF, MAC and MAMC are monitored regularly, they can serve as valuable tools for the dietitan in assessing changes in nutritional status. In addition to the traditional factors considered in nutritional assessment (i.e., dietary, anthropometric, biochemical and clinical), the evaluation of the mentally retarded child should always include a feeding assessment.

Butterworth and Weinsier (1980) have set the lower limits of "normal fat reserves" (Appendix 5) at six to eight millimeters for the girls between the ages ten and eighteen and at four to six millimeters for the boys of the same age. Until guidelines for minimum acceptable TSF levels are made available, these levels of TSF might be useful indicators for the dietitian to determine the "at

risk" population among the mentally retarded children.

While obesity has been reported in the mentally retarded and is prevalent in certain syndromes, such as Prader-Willi, Down's Syndrome, certain types of cerebral palsy and spina bifida, obesity was not noted in the population in this study. Only five subjects had TSF measurements falling in the obese range according to the obesity standards for Caucasian Americans (Seltzer and Mayer, 1965). More subjects might possibly be considered to be obese if the small size was taken into consideration. It is postulated that obesity was not seen in the population in this study for the following reasons: (1) The retarded children admitted to a residential care facility at a young age are likely to have more severe neurological involvement which could interfere with food intake, (2) the nursing personnel is monitoring weight gain and excessive increases are referred to the dietitian and physician for changes in diet orders and (3) food intake in patients on calorie restricted diets is monitored outside the dining room to a certain extent to decrease excessive snacking on high calorie foods. The prevalence of obesity in the patients at the residential care facility appears to increase with increasing age, especially in females. Whether this is due to the heavier patients surviving longer (possibly because the manifestations of the disorder are less severe)

or due to the patient's eventual adaptation is not known. It has also been observed that patients who are admitted to the residential care facility in their late teens and early twenties are more likely to be overweight. This may be due to the family's lack of concern for ideal body weight.

Dietary goals for the mentally retarded should, therefore, be directed to the attainment of the highest feeding skills possible, the prevention of obesity, and the prevention of nutritional deficiencies due to poor intake, utilitzation or drug-nutrient interactions.

Results from this study indicate that more research needs to be done on TSF, MAC and MAMC using larger samples of mentally retarded subjects. Furthermore, noninstitutionalized mentally retarded children should also be studied for TSF, MAC and MAMC to determine if statistically significant differences exist between them and institutionalized retarded and normal children.

While separate norms do not appear to be justified based on the results of this study, establishing minimum levels of acceptable TSF and MAMC would assist the dietitian and physician in determining whether the nutritional status of the mentally retarded individual is adequate. Such minimum acceptable levels might be more appropriate if based on height rather than on age.

Research with regard to food consumption is also needed. Longitudinal studies of food intake of the retarded child beginning in infancy are much needed and could provide answers to the question of whether growth retardation could be decreased or prevented if food consumption was optimum throughout life.

Information with regard to interpretation of the RDA's for the mentally retarded person is also needed. Biochemical studies are needed to determine if nutrient needs differ in this population.

APPENDIX 1: NCHS HEIGHT PERCENTILES

TABLE 5
Smoothed percentiles of stature (in cm) by sex and age: data and statistics from NCHS, 2 to 18 years.

Sex and age	Smoothed[1] percentile						
	5th	10th	25th	50th	75th	90th	95th
	Stature in centimeters						
Male							
2.0 years[2]							
2.5 years	82.5	83.5	85.3	86.8	89.2	92.0	94.4
3.0 years	86.4	86.5	88.5	90.4	92.9	95.6	97.8
3.5 years	89.0	90.3	92.8	94.9	97.5	100.1	102.0
4.0 years	92.5	93.9	96.4	99.1	101.7	104.3	106.1
4.5 years	95.8	97.3	100.0	102.9	105.7	108.2	109.9
5.0 years	98.9	100.6	103.4	106.6	109.4	111.9	113.5
5.5 years	102.0	103.7	106.5	109.9	112.8	115.4	117.0
6.0 years	104.9	106.7	109.6	113.1	116.1	118.7	120.3
6.5 years	107.7	109.6	112.5	116.1	119.2	121.9	123.5
7.0 years	110.4	112.3	115.3	119.0	122.2	124.9	126.6
7.5 years	113.0	115.0	118.0	121.7	125.0	127.9	129.7
8.0 years	115.6	117.6	120.6	124.4	127.8	130.8	132.7
8.5 years	118.1	120.2	123.2	127.0	130.5	133.6	135.7
9.0 years	120.5	122.7	125.7	129.6	133.2	136.5	138.8
9.5 years	122.9	125.2	128.2	132.2	136.0	139.4	141.8
10.0 years	125.3	127.6	130.8	134.8	138.8	142.4	144.9
10.5 years	127.7	130.1	133.4	137.5	141.6	145.5	148.1
11.0 years	130.1	132.6	136.0	140.3	144.6	148.7	151.5
11.5 years	132.6	135.1	138.7	143.3	147.8	152.1	154.9
12.0 years	135.0	137.7	141.5	146.4	151.1	155.6	158.5
12.5 years	137.6	140.3	144.4	149.7	154.6	159.4	162.3
13.0 years	140.2	143.0	147.4	153.0	158.2	163.2	166.1
13.5 years	142.9	145.8	150.5	156.5	161.8	167.0	169.8
14.0 years	145.7	148.7	153.6	159.9	165.3	170.5	173.4
14.5 years	148.8	151.8	156.9	163.1	168.5	173.8	176.7
15.0 years	152.0	155.0	160.1	166.2	171.5	176.6	179.5
15.5 years	155.2	158.2	163.3	169.0	174.1	178.9	181.9
16.0 years	158.3	161.2	166.2	171.5	176.3	180.8	183.9
16.5 years	161.1	163.9	168.7	173.5	178.1	182.4	185.4
17.0 years	163.4	166.1	170.6	175.2	179.5	183.6	186.6
17.5 years	164.9	167.7	171.9	176.2	180.5	184.4	187.3
18.0 years	165.6	168.5	172.4	176.7	181.0	185.0	187.6
	165.7	168.7	172.3	176.8	181.2	185.3	187.6
Female							
2.0 years[2]							
2.5 years	81.6	82.1	84.0	86.8	89.3	92.0	93.6
3.0 years	84.6	85.3	87.3	90.0	92.5	95.0	96.6
3.5 years	88.3	89.3	91.4	94.1	96.6	99.0	100.6
4.0 years	91.7	93.0	95.2	97.9	100.5	102.8	104.5
4.5 years	95.0	96.4	98.8	101.6	104.3	106.6	108.3
5.0 years	98.1	99.7	102.2	105.0	107.9	110.2	112.0
5.5 years	101.1	102.7	105.4	108.4	111.4	113.8	115.6
6.0 years	103.9	105.6	108.4	111.6	114.8	117.4	119.2
6.5 years	106.6	108.4	111.3	114.6	118.1	120.8	122.7
7.0 years	109.2	111.0	114.1	117.6	121.3	124.2	126.1
7.5 years	111.8	113.6	116.8	120.6	124.4	127.6	129.5
8.0 years	114.4	116.2	119.5	123.5	127.5	130.9	132.9
8.5 years	116.9	118.7	122.2	126.4	130.6	134.2	136.2
9.0 years	119.5	121.3	124.9	129.3	133.6	137.4	139.6
9.5 years	122.1	123.9	127.7	132.2	136.7	140.7	142.9
10.0 years	124.8	126.6	130.6	135.2	139.8	143.9	146.2
10.5 years	127.5	129.5	133.6	138.3	142.9	147.2	149.5
11.0 years	130.4	132.5	136.7	141.5	146.1	150.4	152.8
11.5 years	133.5	135.6	140.0	144.8	149.3	153.7	156.2
12.0 years	136.8	139.0	143.5	148.2	152.6	156.9	159.5
12.5 years	139.8	142.3	147.0	151.5	155.8	160.0	162.7
13.0 years	142.7	145.4	150.1	154.6	158.8	162.9	165.6
13.5 years	145.2	148.0	152.8	157.1	161.3	165.3	168.1
14.0 years	147.2	150.0	154.7	159.0	163.2	167.3	170.0
14.5 years	148.7	151.5	155.9	160.4	164.6	168.7	171.3
15.0 years	149.7	152.5	156.8	161.2	165.6	169.8	172.2
15.5 years	150.5	153.2	157.2	161.8	166.3	170.5	172.8
16.0 years	151.1	153.6	157.5	162.1	166.7	170.9	173.1
16.5 years	151.6	154.1	157.8	162.4	166.9	171.1	173.3
17.0 years	152.2	154.6	158.2	162.7	167.1	171.2	173.4
17.5 years	152.7	155.1	158.7	163.1	167.3	171.2	173.5
18.0 years	153.2	155.6	159.1	163.4	167.5	171.1	173.5
	153.6	156.0	159.6	163.7	167.6	171.0	173.6

[1]Smoothed by cubic spline approximation, as described in the appendix.
[2]Because of a logistic problem the percentiles of stature for children under 2.5 years are not highly reliable. The age interval represented is 2.00-2.25 years.

Am. J.Clin Nutr. 1979 32:607-629.

APPENDIX 2: NCHS WEIGHT PERCENTILES

TABLE 6
Smoothed percentiles of weight (in kg) by sex and age: data and statistics from NCHS, 1.5 to 18 years.

Sex and age	Smoothed[1] percentile						
	5th	10th	25th	50th	75th	90th	95th
	Weight in kilograms						
Male							
1.5 years	9.72	10.18	10.51	11.09	12.02	12.95	14.42
2.0 years	10.49	10.96	11.55	12.34	13.36	14.38	15.50
2.5 years	11.27	11.77	12.55	13.52	14.61	15.71	16.61
3.0 years	12.05	12.58	13.52	14.62	15.78	16.95	17.77
3.5 years	12.84	13.41	14.46	15.68	16.90	18.15	18.98
4.0 years	13.64	14.24	15.39	16.69	17.99	19.32	20.27
4.5 years	14.45	15.10	16.30	17.69	19.06	20.50	21.63
5.0 years	15.27	15.96	17.22	18.67	20.14	21.70	23.09
5.5 years	16.09	16.83	18.14	19.67	21.25	22.96	24.66
6.0 years	16.93	17.72	19.07	20.69	22.40	24.31	26.34
6.5 years	17.78	18.62	20.02	21.74	23.62	25.76	28.16
7.0 years	18.64	19.53	21.00	22.85	24.94	27.36	30.12
7.5 years	19.52	20.45	22.02	24.03	26.36	29.11	32.73
8.0 years	20.40	21.39	23.09	25.30	27.91	31.06	34.51
8.5 years	21.31	22.34	24.21	26.66	29.61	33.22	36.96
9.0 years	22.25	23.33	25.40	28.13	31.46	35.57	39.58
9.5 years	23.25	24.38	26.68	29.73	33.46	38.11	42.35
10.0 years	24.33	25.52	28.07	31.44	35.61	40.80	45.27
10.5 years	25.51	26.78	29.59	33.30	37.92	43.63	48.31
11.0 years	26.80	28.17	31.25	35.30	40.38	46.57	51.47
11.5 years	28.24	29.72	33.08	37.46	43.00	49.61	54.73
12.0 years	29.85	31.46	35.09	39.78	45.77	52.73	58.09
12.5 years	31.64	33.41	37.31	42.27	48.70	55.91	61.52
13.0 years	33.64	35.60	39.74	44.95	51.79	59.12	65.02
13.5 years	35.85	38.03	42.40	47.81	55.02	62.35	68.51
14.0 years	38.22	40.64	45.21	50.77	58.31	65.57	72.13
14.5 years	40.66	43.34	48.08	53.76	61.58	68.76	75.66
15.0 years	43.11	46.06	50.92	56.71	64.72	71.91	79.12
15.5 years	45.50	48.69	53.64	59.51	67.64	74.98	82.45
16.0 years	47.74	51.16	56.16	62.10	70.26	77.97	85.62
16.5 years	49.76	53.39	58.38	64.39	72.46	80.84	88.59
17.0 years	51.50	55.28	60.22	66.31	74.17	83.58	91.31
17.5 years	52.89	56.78	61.61	67.78	75.32	86.14	93.73
18.0 years	53.97	57.89	62.61	68.88	76.04	88.41	95.76
Female							
1.5 years	9.02	9.16	9.61	10.38	10.94	11.75	12.36
2.0 years	9.95	10.32	10.96	11.80	12.73	13.58	14.15
2.5 years	10.80	11.35	12.11	13.03	14.23	15.16	15.76
3.0 years	11.61	12.26	13.11	14.10	15.50	16.54	17.22
3.5 years	12.37	13.08	14.00	15.07	16.59	17.77	18.59
4.0 years	13.11	13.84	14.80	15.96	17.56	18.93	19.91
4.5 years	13.83	14.56	15.55	16.81	18.48	20.06	21.24
5.0 years	14.55	15.26	16.29	17.66	19.39	21.23	22.62
5.5 years	15.29	15.97	17.05	18.56	20.36	22.48	24.11
6.0 years	16.05	16.72	17.86	19.52	21.44	23.89	25.75
6.5 years	16.85	17.51	18.76	20.61	22.68	25.50	27.59
7.0 years	17.71	18.39	19.78	21.84	24.16	27.39	29.68
7.5 years	18.62	19.37	20.95	23.26	25.90	29.57	32.07
8.0 years	19.62	20.45	22.26	24.84	27.88	32.04	34.71
8.5 years	20.68	21.64	23.70	26.58	30.08	34.73	37.58
9.0 years	21.82	22.92	25.27	28.46	32.44	37.60	40.64
9.5 years	23.05	24.29	26.94	30.45	34.94	40.61	43.85
10.0 years	24.36	25.76	28.71	32.55	37.53	43.70	47.17
10.5 years	25.75	27.32	30.57	34.72	40.17	46.84	50.57
11.0 years	27.24	28.97	32.49	36.95	42.84	49.96	54.00
11.5 years	28.83	30.71	34.48	39.23	45.48	53.03	57.42
12.0 years	30.52	32.53	36.52	41.53	48.07	55.99	60.81
12.5 years	32.30	34.42	38.59	43.84	50.56	58.81	64.12
13.0 years	34.14	36.35	40.65	46.10	52.91	61.45	67.30
13.5 years	35.98	38.26	42.65	48.26	55.11	63.87	70.30
14.0 years	37.76	40.11	44.54	50.28	57.09	66.04	73.08
14.5 years	39.45	41.83	46.28	52.10	58.84	67.95	75.59
15.0 years	40.99	43.38	47.82	53.68	60.32	69.54	77.78
15.5 years	42.32	44.72	49.10	54.96	61.48	70.79	79.59
16.0 years	43.41	45.78	50.09	55.89	62.29	71.68	80.99
16.5 years	44.20	46.54	50.75	56.44	62.75	72.18	81.93
17.0 years	44.74	47.04	51.14	56.69	62.91	72.38	82.46
17.5 years	45.08	47.33	51.33	56.71	62.89	72.37	82.62
18.0 years	45.26	47.47	51.39	56.62	62.78	72.25	82.47

[1]Smoothed by cubic spline approximation, as described in the appendix.

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APPENDIX 3: MAC AND TSF PERCENTILES

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TABLE 1

Percentiles for upper arm circumference and triceps skin folds for whites of the Ten-State Nutrition Survey of 1968–1970

Age group	Age Midpoint, years	No.	Arm circumference percentiles, mm					Triceps skin fold percentiles, mm				
			5th	15th	50th	85th	95th	5th	15th	50th	85th	95th
					Males							
0.0–0.4	0.3	41	113	120	134	147	153	4	5	8	12	15
0.5–1.4	1	140	128	137	152	168	175	5	7	9	13	15
1.5–2.4	2	177	141	147	157	170	180	5	7	10	13	14
2.5–3.4	3	210	144	150	161	175	182	6	7	9	12	14
3.5–4.4	4	208	143	150	165	180	190	5	6	9	12	14
4.5–5.4	5	262	146	155	169	185	199	5	6	8	12	16
5.5–6.4	6	264	151	159	172	188	198	5	6	8	11	15
6.5–7.4	7	309	154	162	176	194	212	4	6	8	11	14
7.5–8.4	8	301	161	168	185	205	233	5	6	8	12	17
8.5–9.4	9	287	165	174	190	217	262	5	6	9	14	19
9.5–10.4	10	315	170	180	200	228	255	5	6	10	16	22
10.5–11.4	11	294	177	186	208	240	276	6	7	10	17	25
11.5–12.4	12	294	184	194	216	253	291	5	7	11	19	26
12.5–13.4	13	266	186	198	230	270	297	5	6	10	18	25
13.5–14.4	14	207	198	211	243	279	321	5	6	10	17	22
14.5–15.4	15	179	202	220	253	302	320	4	6	9	19	26
15.5–16.4	16	166	217	232	262	300	335	4	5	9	20	27
16.5–17.4	17	142	230	238	275	306	326	4	5	8	14	20
17.5–24.4	21	545	250	264	292	330	354	4	5	10	18	25
24.5–34.4	30	679	260	280	310	344	366	4	6	11	21	28
34.5–44.4	40	616	259	280	312	345	371	4	6	12	22	28
					Females							
0.0–0.4	0.3	46	107	118	127	145	150	4	5	8	12	13
0.5–1.4	1	172	125	134	146	162	170	6	7	9	12	15
1.5–2.4	2	172	136	143	155	171	180	6	7	10	13	15
2.5–3.4	3	163	137	145	157	169	176	6	7	10	12	14
3.5–4.4	4	215	145	150	162	176	184	5	7	10	12	14
4.5–5.4	5	233	149	155	169	185	195	6	7	10	13	16
5.5–6.4	6	259	148	158	170	187	202	6	7	10	12	15
6.5–7.4	7	273	153	162	178	199	216	6	7	10	13	17
7.5–8.4	8	270	158	166	183	207	231	6	7	10	15	19
8.5–9.4	9	284	166	175	192	222	255	6	7	11	17	24
9.5–10.4	10	276	170	181	203	236	263	6	8	12	19	24
10.5–11.4	11	268	173	186	210	251	280	7	8	12	20	29
11.5–12.4	12	267	185	196	220	256	275	6	9	13	20	25
12.5–13.4	13	229	186	204	230	270	294	7	9	14	23	30
13.5–14.4	14	184	201	214	240	284	306	8	10	15	22	28
14.5–15.4	15	197	205	216	245	281	310	8	11	16	24	30
15.5–16.4	16	187	211	224	249	286	322	8	10	15	23	27
16.5–17.4	17	142	207	224	250	291	328	9	12	16	26	31
17.5–24.4	21	836	215	233	260	297	329	9	12	17	25	31
24.5–34.4	30	1153	230	243	275	324	361	9	12	19	29	36
34.5–44.4	40	933	232	250	286	340	374	10	14	22	32	39

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APPENDIX 4: MAMC PERCENTILES

TABLE 2
Percentiles for upper arm diameter and upper arm circumference for whites of the Ten-State Nutrition Survey of 1968–1970

Age midpoint, years[a]	Arm muscle diameter percentiles, mm					Arm muscle circumference percentiles, mm				
	5th	15th	50th	85th	95th	5th	15th	50th	85th	95th
					Males					
0.3	26	30	34	40	42	81	94	106	125	133
1	32	34	39	44	46	100	108	123	137	146
2	35	37	40	44	46	111	117	127	138	146
3	36	38	42	46	48	114	121	132	145	152
4	38	39	43	48	50	118	124	135	151	157
5	39	41	45	50	53	121	130	141	156	166
6	40	43	47	51	53	127	134	146	159	167
7	41	43	48	52	55	130	137	151	164	173
8	44	46	50	55	59	138	144	158	174	185
9	44	46	51	58	64	138	143	161	182	200
10	45	48	53	59	64	142	152	168	186	202
11	48	50	55	62	67	150	158	174	194	211
12	49	52	58	66	70	153	163	181	207	221
13	51	54	62	71	77	159	169	195	224	242
14	53	58	67	74	84	167	182	211	234	265
15	55	59	70	80	86	173	185	220	252	271
16	59	65	73	83	89	186	205	229	260	281
17	66	69	78	86	92	206	217	245	271	290
21	69	74	82	91	97	217	232	258	286	305
30	70	77	86	94	100	220	241	270	295	315
40	71	76	86	96	101	222	239	270	300	318
					Females					
0.3	27	29	33	37	40	86	92	104	115	126
1	31	32	37	41	43	97	102	117	128	135
2	34	36	40	44	46	105	112	125	140	146
3	34	37	41	44	46	108	116	128	138	143
4	36	38	42	46	48	114	120	132	146	152
5	38	40	44	48	51	119	124	138	151	160
6	38	41	45	49	53	121	129	140	155	165
7	39	42	47	52	56	123	132	146	162	175
8	41	44	48	53	59	129	138	151	168	186
9	43	45	50	56	62	136	143	157	176	193
10	44	47	52	58	62	139	147	163	182	196
11	44	48	55	62	67	140	152	171	195	209
12	48	51	57	64	68	150	161	179	200	212
13	49	53	59	66	71	155	165	185	206	225
14	53	56	61	70	74	166	175	193	221	234
15	52	55	62	70	74	163	173	195	220	232
16	54	57	64	72	83	171	178	200	227	260
17	54	56	62	71	77	171	177	196	223	241
21	54	58	65	73	80	170	183	205	229	253
30	56	60	68	78	87	177	189	213	245	272
40	57	61	69	80	89	180	192	216	250	279

[a] The age group and n are the same as in Table 1.

Am. J. Clin. Nutr. 1974. 27:1052-1058 (Frisancho).

APPENDIX 5: TRICEPS SKINFOLD REFERENCE GRAPH

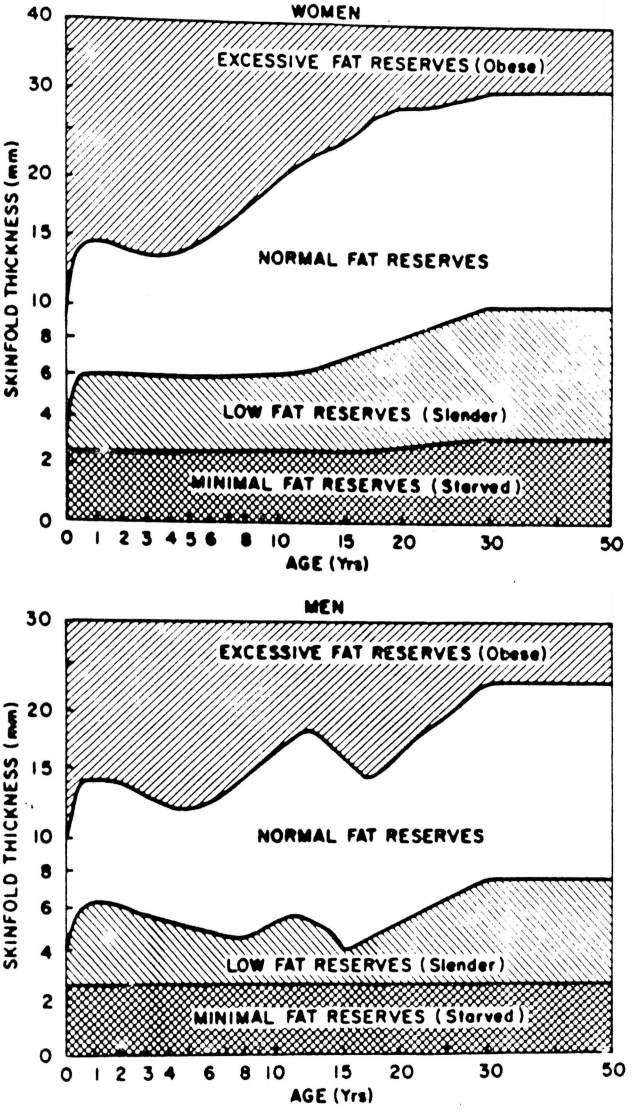


Fig. 22-3. Triceps skinfold reference graphs (all ages). (Adapted from data of Seltzer and Mayer,[9] Tanner and Whitehouse[10] and Frisancho,[11] and reprinted from Weinsier, Butterworth and Sahm: *Handbook of Clinical Nutrition*. Birmingham, University of Alabama Department of Nutrition Sciences, 1977.)

Butterworth and Weinsier, (1980)

APPENDIX 6: MAMC REFERENCE GRAPH

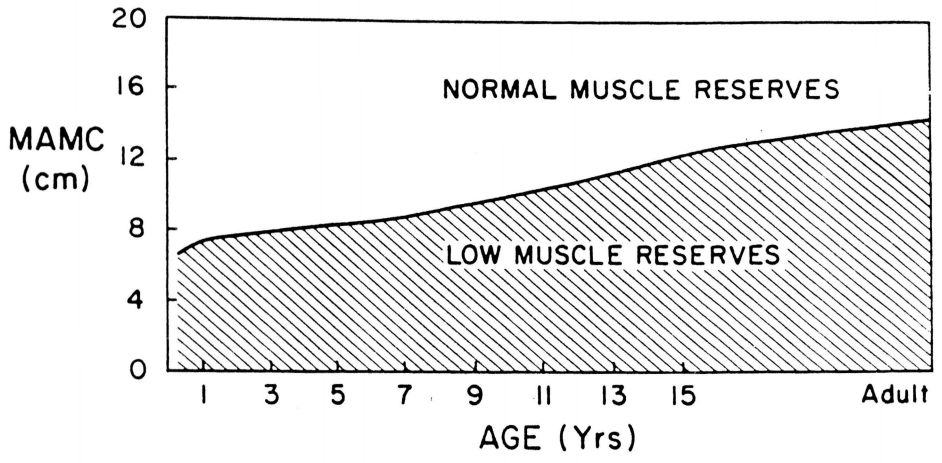


Fig. 22–4. Midarm muscle circumference (MAMC) reference graph, both sexes, all ages (MAMC = arm circum. − 3 × skinfold [cm]). Patients with values in the "low muscle reserves" area are likely to be severely malnourished. It is possible, however, to fall in the "normal" range and be depleted. (Data adapted from Frisancho[11] and reprinted from Weinsier, Butterworth and Sahm: *Handbook of Clinical Nutrition.* Birmingham, University of Alabama Department of Nutrition Sciences, 1977.)

Butterworth and Weinsier, (1980)

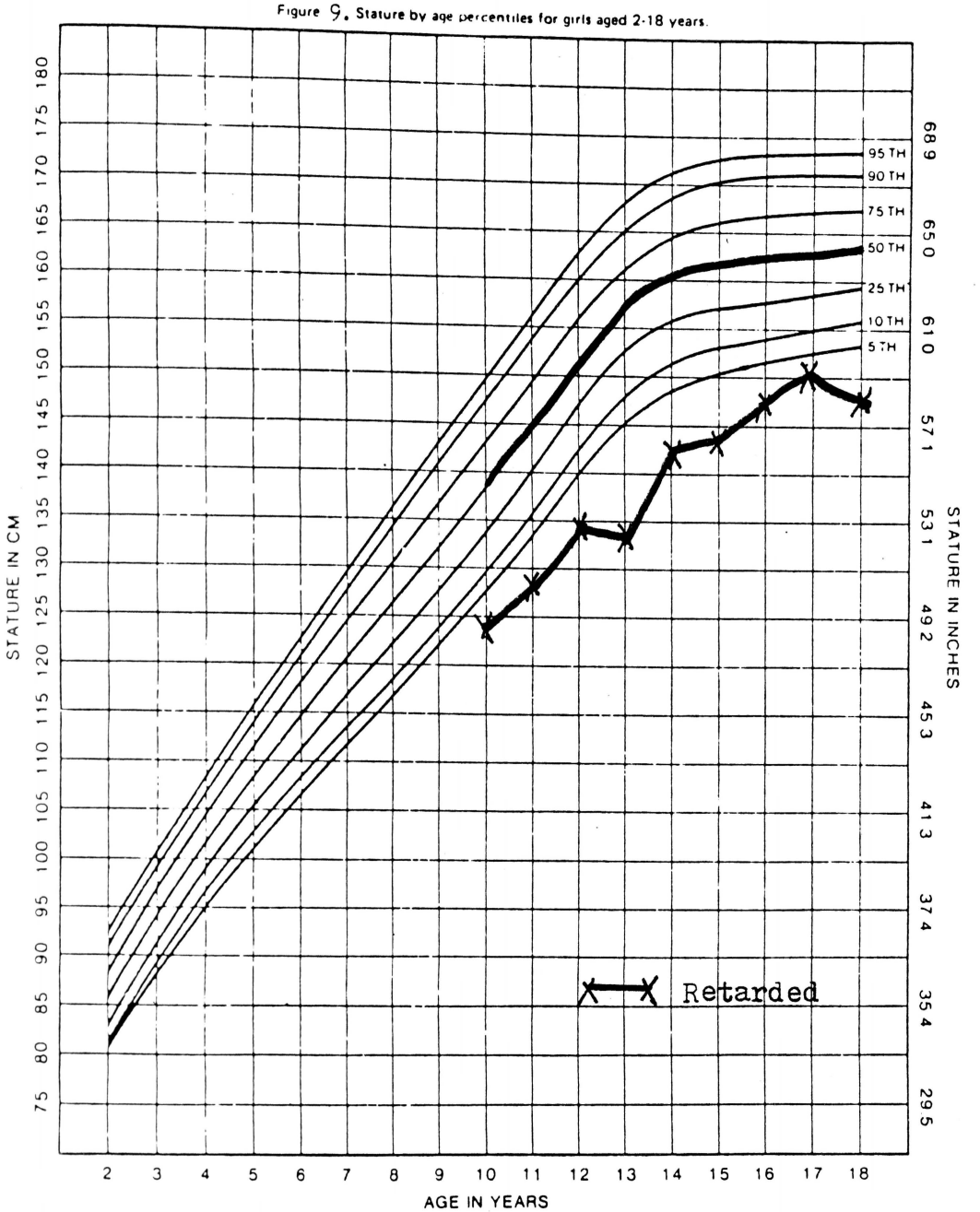
APPENDIX 7:

ANTHROPOMETRIC DATA FORM DATE________________

	SEX	HT	WT	TSF	MAC	MAMC	COMMENTS

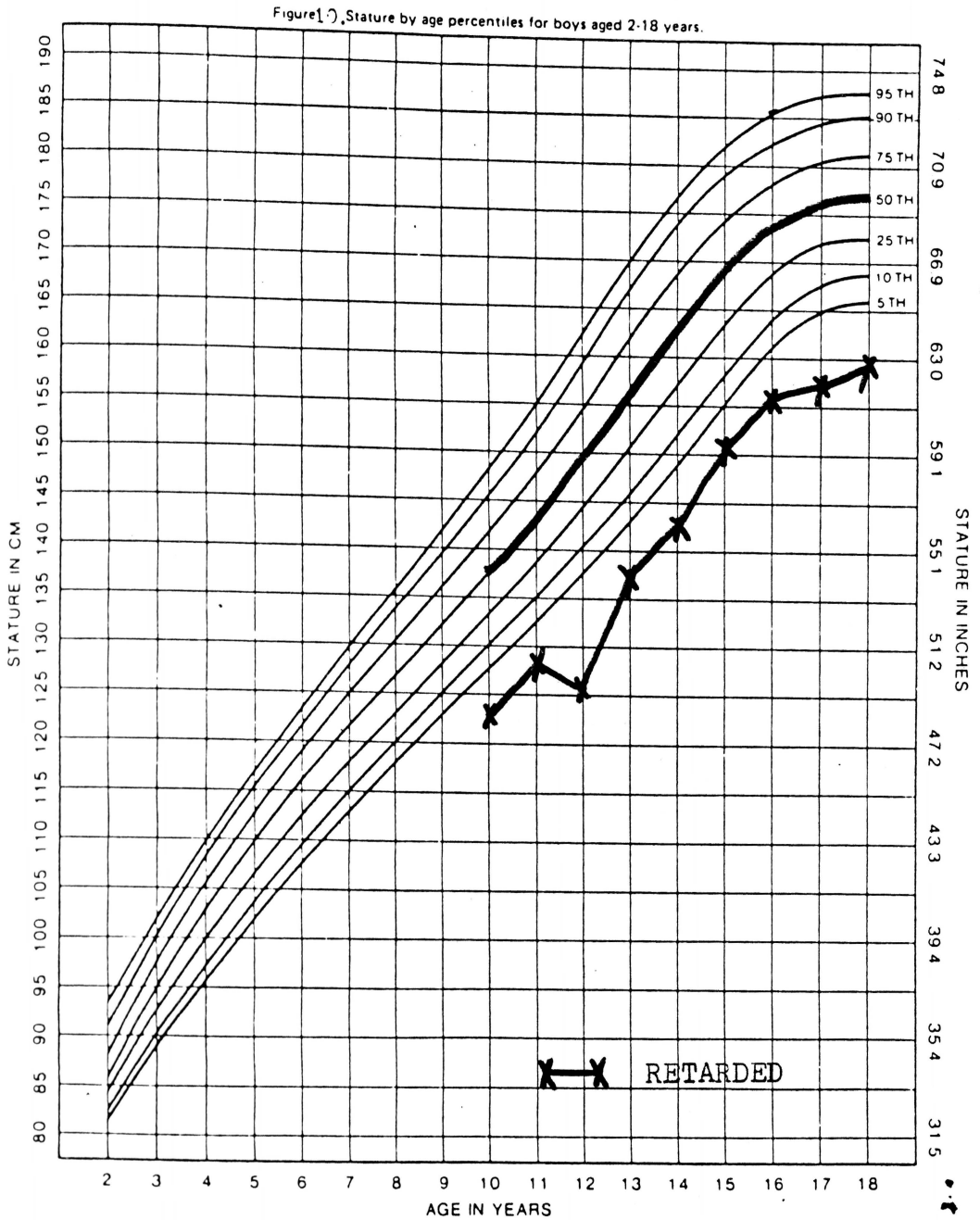
APPENDIX 8: NCHS GROWTH CHART (HEIGHT) FOR FEMALE

Figure 9. Stature by age percentiles for girls aged 2-18 years.



Hamill et al. (1979)

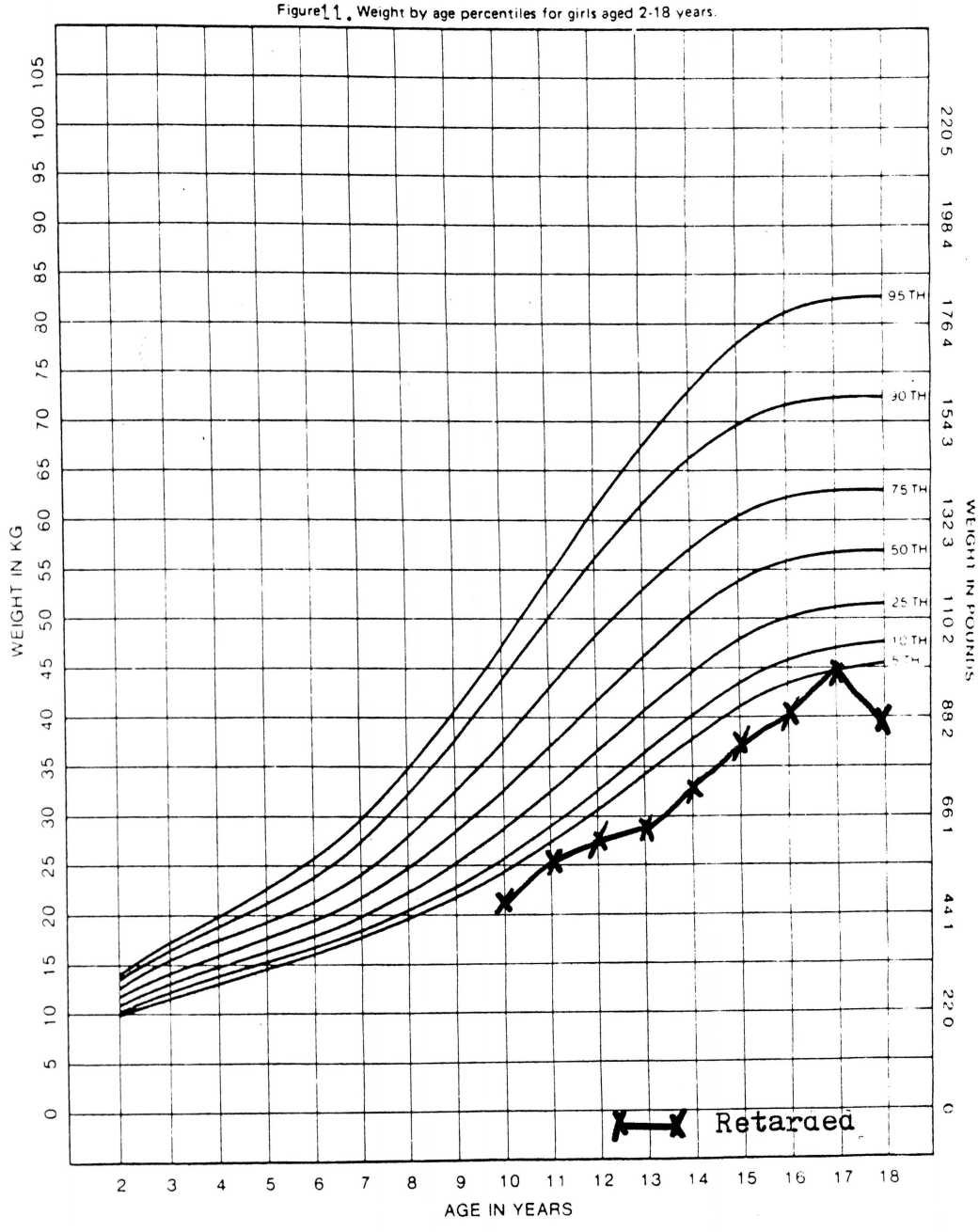
APPENDIX 9: NCHS GROWTH CHART (HEIGHT) FOR MALE



Hamill et al. (1979)

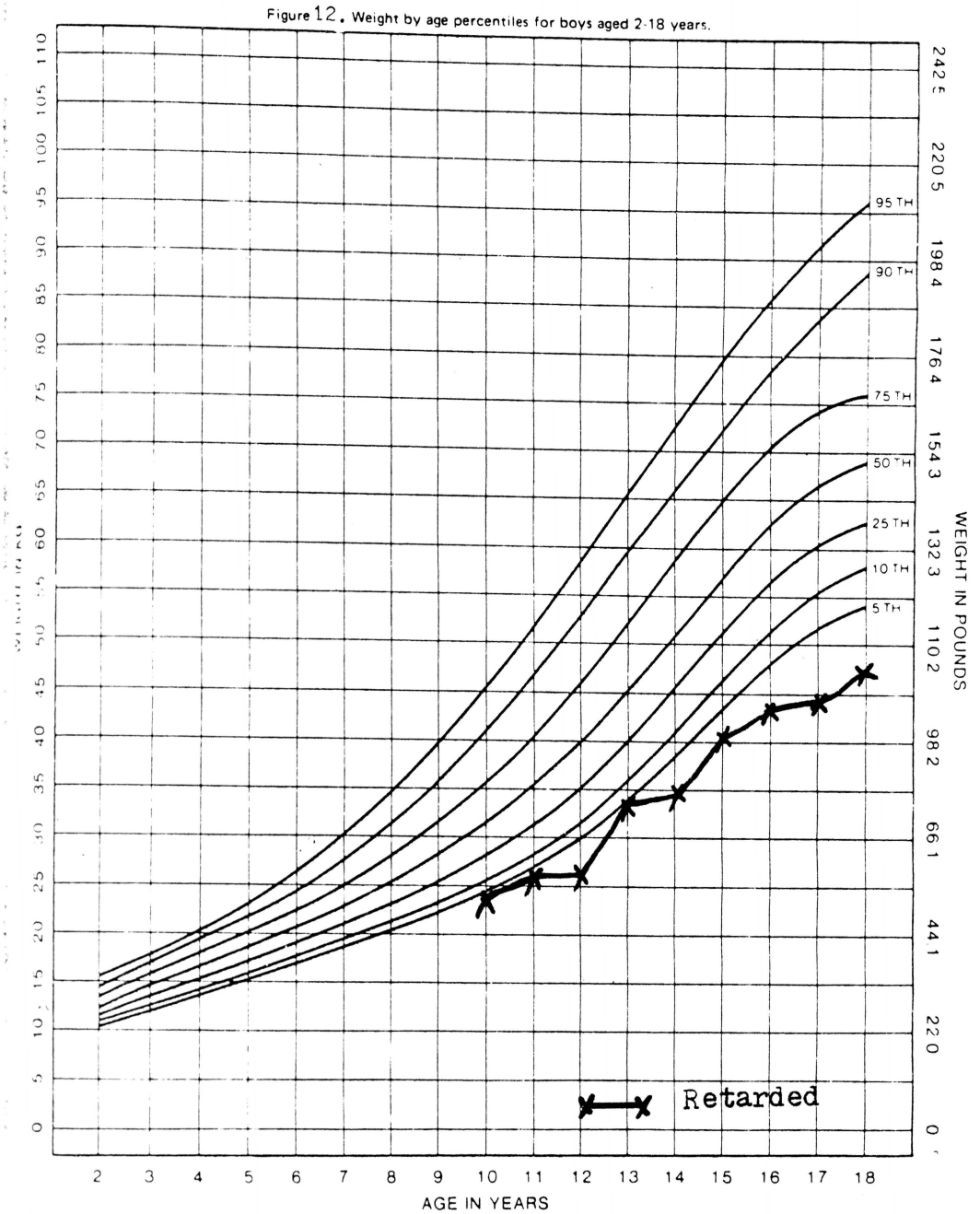
APPENDIX 10: NCHS GROWTH CHART (WEIGHT) FOR FEMALE

Figure 11. Weight by age percentiles for girls aged 2-18 years.



Hamill et al. (1979)

APPENDIX 11: NCHS GROWTH CHART (WEIGHT) FOR MALE



Hamill et al. (1979)

APPENDIX 12: TSF GRAPH FOR FEMALES

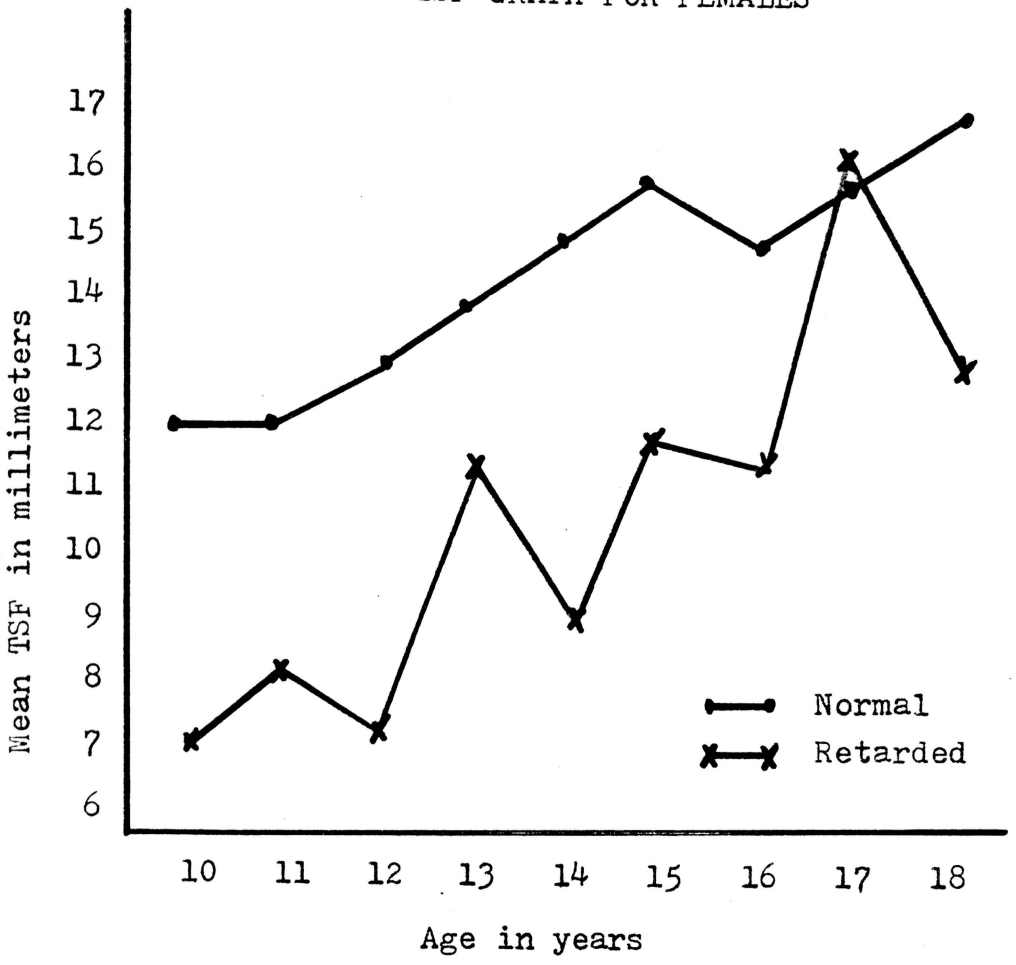


Fig. 4 Changes in mean TSF for normal and retarded girls between ages 10 and 18.

APPENDIX 13: TSF GRAPH FOR MALES

Fig. 5 Changes in mean TSF for normal and retarded boys between ages 10 and 18.

APPENDIX 14: MAC GRAPH FOR FEMALES

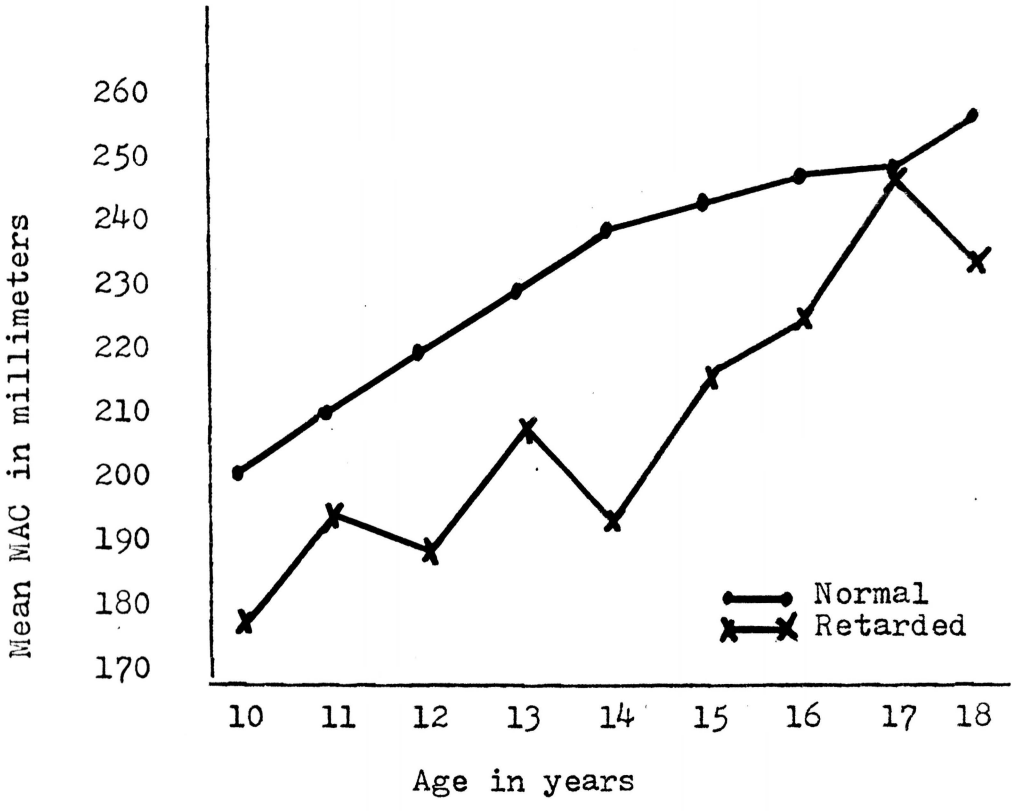


Fig. 6 Changes in mean MAC for normal and retarded girls between ages 10 and 18

APPENDIX 15: MAC GRAPH FOR MALES

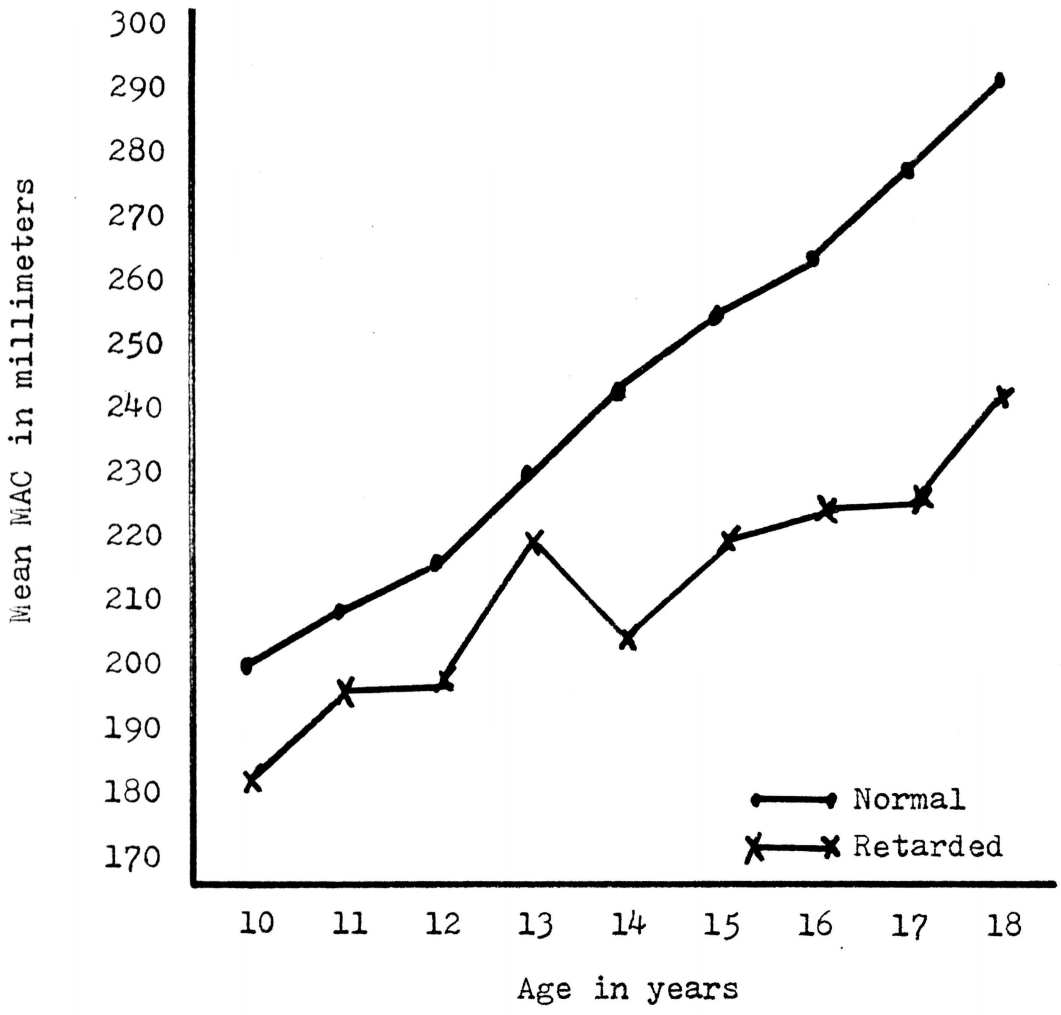


Fig. 7 Changes in mean MAC for normal and retarded boys between ages 10 and 18.

APPENDIX 16: MAMC GRAPH FOR FEMALES

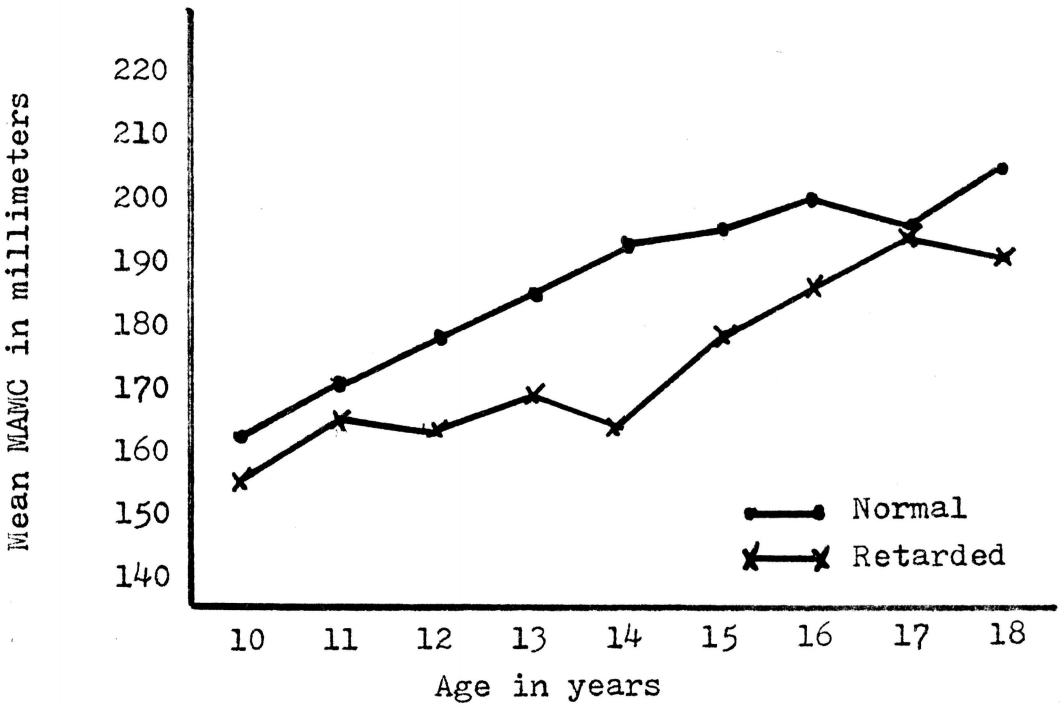


Fig. 8 Changes in mean MAMC for normal and retarded girls between ages 10 and 18.

APPENDIX 17: MAMC GRAPH FOR MALES

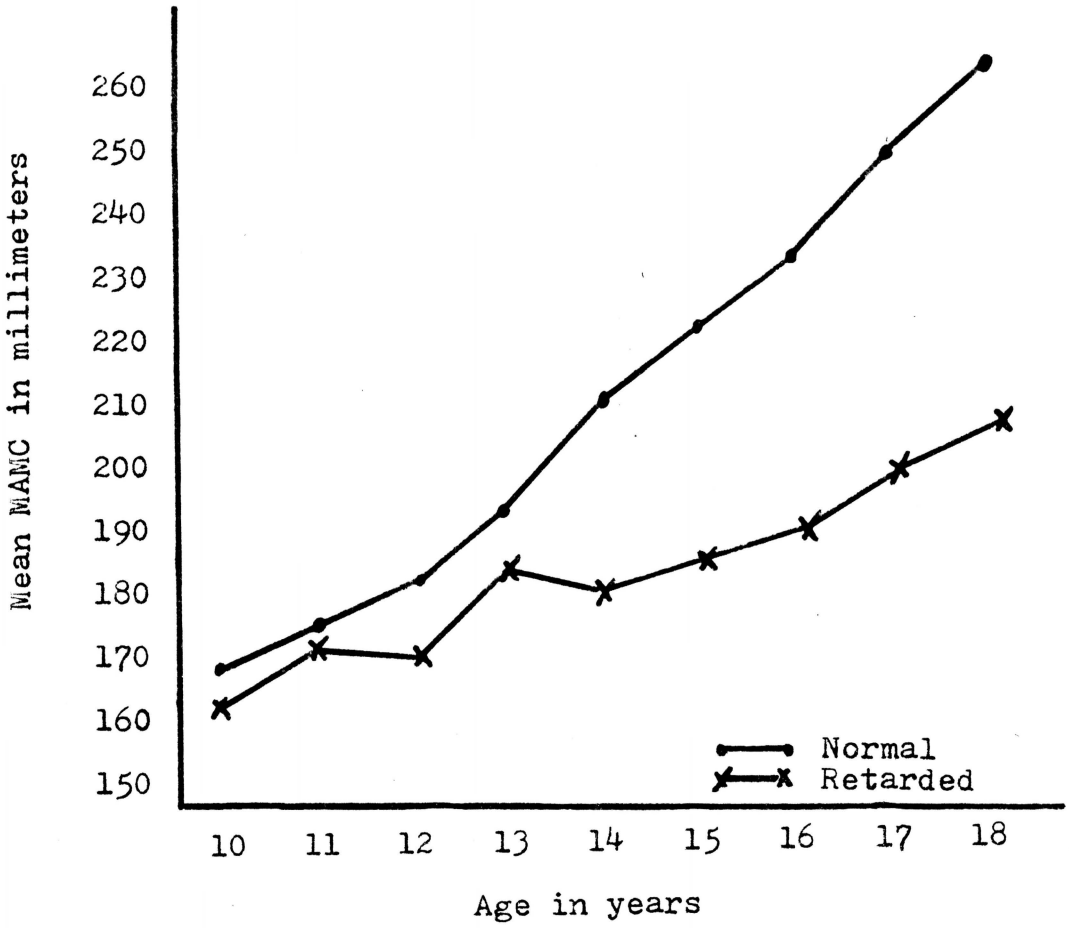


Fig.9 Changes in mean MAMC for normal and retarded boys between ages 10 and 13.

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