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Obesity in childhood

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- OBESITY IN CHILDHOOD -

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The Literature is crowded with articles on obesity. Few of these authors have made actual studies on the subject. Textbooks are deficient or entirely lacking chapters dealing with fat children. It was because of this condition that the author of this thesis chose the subject of "Obesity in Childhood."

Credit is due Dr. John L. Gedgoud for his assistance and very helpful advice.

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The undernourished child has so usurped the attention and worries of the modern mother that his counterpart, the overweight child, usually is ignored unless, perchance, he is held up as an object of envy to that perennial pediatric problem, the child who won't eat. The average physician gives little thought to obesity. If he gives any advice at all to the parents of a fat child, it usually consists of certain admonitions in a very professional manner, and, perhaps, a list of foods or even a diet list. However, obesity is of extreme importance to the internist and of tremendous interest to the laity.

HISTORY:

The term "obesity" is derived from the Latin word, obesus, meaning that has eaten itself fat, stout, plump--which derivation suggests that for those who first introduced it in the seventeenth century the word designated a condition actually dependent on eating too much.

William Wadd (48) a surgeon in London in 1816, was of the same opinion as to the cause of obesity or corpulence as he called it. He was very much concerned with this disease and edited a small book on the condition. The following paragraphs are a few excerpts from his book, some of which can still be applied to our present day, and others which are only of interest.

"If the increase of wealth and the refinement of modern times, have tended to banish plague and pestilence from our cities, they have probably introduced the whole

train of nervous disorders, and increased the frequency of corpulence."

Wadd (48) goes on to help clarify the ideas as to location and etiology.

"The quantity and quality of fat varies according to the age, and the parts in which it is deposited. It is firmer and higher coloured in old persons, than in young ones. It is also more condensed and solid in parts liable to compression, than in the omentum, or about the heart, stomach and intestines. In children the fat is distributed over the surface of the body, but as we grow older, it diminishes on the surface in proportion as it becomes deeper seated."

"Boerhoane and Vanswieten were of opinion that fat is deposited from the blood by its slower circulation in the extreme vessels. Malpighi and other anatomists have thought that there was a glandular apparatus superadded to the cellular membrane, to assist in the formation of fat. But this, though consistent with the general system of the economy, has never been discovered."

"When the accumulation of fat becomes excessive, it is not only burthensome, but becomes a disease, disposes to other diseases, and to sudden death."

One of the most interesting parts of Wadd's book is the section on treatment in which he refers to various men's types of therapy.

"Various medicines that have, at different times,

been recommended as specifics:

(a) Coelius Aurelianus--mode of cure into two parts:

1- Taking food that has little nutrition in it.

2- Observing certain rules of exercise.

(b) Borelli advises chewing tobacco.

(c) Few things have been more generally administered in the cure of corpulence, than acids of various kinds. The emaciating properties of acid liquors, particularly vinegar are very well known.

(d) Dr. Flemyng strongly recommended soap because of its diuretic properties. He perscribed every night at bed time, one quarter ounce of common home-made castile soap, dissolved in a quarter pint of soft water.

(e) Author of "Zoonomia"--eating of much salt, or salted meat, is more efficacious than soap, as it increases perspiration, and produces thirst, by which, if the patient can bear it, the absorption of his fat will be greatly increased, as in fever.

(f) Dr. Fothergill--"A strict vegetable diet reduces exuberant fat more certainly than any other means I know."

"These I believe, are the principal articles that have been resorted to in the treatment of this disease; and the person who depends solely on the benefit to be derived from the use of any of them, will find himself

grievously disappointed.

"How can a magic box of pills,

"Syrup, or vegetable juice,

"Eradicate at once those ills,

"Which years of luxury produce?"

Not only is the medical literature a source of material on obesity but the classics. No paper on obesity would be complete without mentioning Dicken's (14) character "the fat boy." His descriptions are typical of that type often known as endogenous.

"-----and on the box sat a fat and red-faced boy, in a state of somnolence, whom no speculative observer could have regarded for an instant without setting down as the official dispenser of the contents of the before mentioned hamper, when the proper time for their consumption should arrive.

"---the fat boy waddled to the same perch and fell fast asleep instantly. ---the leaden eyes, which twinkled behind his mountainous cheeks, leered horribly upon the food as he unpacked it from the basket. --The train of nods which the fat boy gave by way of assent, communicated a blanc-mange like motion of his fat cheeks."

In the field of art, a good example of infantile obesity is shown in the famous painting "The Virgin, The Christ Child and John," by Lorenzi di Crede (1453-about 1535), the Tuscan painter, fellow student of Leonardi da Vinci under Andreas Verocchio. The increased number of folds in

the skin due to the fat underneath are particularly well done. (37)

IMPORTANCE OR SIGNIFICANCE OF OBESITY:

Obesity is relatively common in childhood and early adolesence--it has been and always will be, but there is considerable uncertainty as to its significance.

Should obesity in children be ignored? Is the reduction in weight of the obese child worth all the effort?

It is not a proven fact whether all the degenerative changes that accompany adiposity in the adult occur in childhood--but it has been shown that obese children are poorer surgical risks, that they are more prone to develop pneumonia and pulmonary complications, and that diabetes is a potentiality to be considered. Faulty posture, usually a lumbar lordosis, and various orthopedic abnormalities of the feet are aggravated or initiated by obesity. And one of the most important--fat children are the victims of continuous teasing, which in some is apt to initiate a feeling of inferiority resulting in serious behavior problems. Surely, reduction in weight by the proper treatment, for the child's mental well being, would more than justify its consideration.

In a pediatric practice many children are encountered whose chief complaint is that of being considerably overweight. Often this condition of the child is accepted by the parents as a sign of good health and digestion, and indeed the child may appear so robust and cheerful that any

criticism of his weight is resented.

On the other hand, some children are brought to their physician because obesity is the cause of a definite handicap, mental or physical. They may be taunted by their companions until they become acutely sensitive of their appearance, or they are unable to participate in games, and so become less active and still heavier. The overweight girl may realize that she is not as attractive as her more slender companions. These effects may in time lead to definite personality changes or behavior problems which may be detrimental to the whole future of the individual. Fortunately, such an eventuality is not the rule, for many obese children will assume a more normal configuration as adolescence advances, and many are cheerful in spite of their weight.

Obesity, therefore, is a disease entity and necessitates further consideration. What constitutes obesity? Which child can be called obese, and which one cannot?

DIAGNOSIS OF OBESITY:

Bruch (5) defines obesity as a nutritional state in which the storage of fat exceeds the amount that is commonly considered an expression of adequate alimentation. Talbot (44) defines it in a slightly different way, as an accumulation in the body of an amount of subcutaneous fat which is excessive in relation to the amount of muscle. Based upon this suposition, Talbot states that persons of normal weight who show such a disproportion should be considered obese, also that the person who is overweight but

has superior development of the muscular system should not be considered obese. It was on this basis that he carried out his studies on the creatinine coefficient studies as a measurement of obesity which will be discussed later.

It is difficult to determine what actually constitutes obesity in a child. So much emphasis is placed on the various height-weight tables that individual differences in body build and in constitutional make-up, often are over looked. It is stated generally that a child is obese when the excess surpasses 20 per cent. While this holds for the majority, one must keep in mind the occasional young "husky" of splendid build and muscular development, who may exceed that limit over the average, and yet not be obese. Just as there is no general rule that will sharply distinguish undernutrition and normal nutrition, so there is the same lack of definite standards which separate overnutrition or obesity from normal nutrition.

For all practical purposes, perhaps the keen observer can gauge obesity more accurately by clinical inspection, taking into consideration the distribution of fat and general physique, than by the use of height-weight-age tables.

The old height-weight-age tables are very inadequate because they are based on averages and fail to recognize individual differences. Pryor (32) recently revised her width-weight tables which would appear to be a much more accurate measurement of nutrition. This new width-length index is the figure for the biiliac diameter divided by

that for the standing height and multiplied by 1,000. It is a numerical expression of the relative width or body build of a subject. Careful checking has shown the width-length index to be consistent and to have high validity as a measure of body build. A child found to be 8% broader than the average for his age-sex group at the age of 10 years was found to have remained approximately 8% broader than average when he had attained the age of 14 years. This type of a standard table would surely be more accurate than the old height-weight-age tables.

An accurate quantitative measurement of obesity can be determined by the creatinine coefficient. Talbot (44) as previously mentioned, has done a great amount of work along this line, and it is his conclusions, to which are referred in this paper concerning the creatinine coefficient. The excretion of creatinine is a product of muscular catabolism. The amount excreted is surprisingly uniform from day to day, provided the collection of urine is accurate. Differences between persons are dependent largely in variations in the muscle mass. The proportion of the muscle in the body may be derived from the ratio of the amount of creatinine excreted in the urine to the body weight. This ratio, $\frac{\text{milligrams of creatinine / 24 hours}}{\text{Kg. of body weight}}$ was named the creatinine coefficient by Shaffer(40). Under most circumstances a low creatinine coefficient is an index of obesity. Age is known to be related to the output of creatinine. In infants and children the coefficient is

lower than it is in adults. According to Hunter (22), it is around 2.7 at birth. It rises to from 5.5 to 8.0 within the first few months; in the third year it is from 11 to 13; in the ninth, between 13 and 19. Although there is a rise in the coefficient from birth to puberty, the periods of greatest rise are found during the first four years and during the adolescent period, from the twelfth to the sixteenth year. The periods of rapid growth in the musculature are found during the same age periods.

In Talbot's (44) studies a group of 37 children were chosen from the wards and the outpatient department of the Children's hospital in Boston. An estimate was made of the proportion of subcutaneous fat to underlying muscle by inspection and palpation of the abdominal wall and extremities, with the muscles partially contracted. They were grouped accordingly, and creatinine of 24 hour urine specimens was determined by Folin's method.

The average creatinine coefficients of obese, normal, and lean children were found to be 14.0, 20.4, and 30.7 respectively. The weight of the muscles, as calculated from the output of creatinine of these groups, constitutes, respecitively, 25, 37, and 55% of the body weight. A small group of children with marked emaciation had an average creatinine coefficient of 9.0, and their muscles were calculated to yield 16% of the body weight.

The inconvenience associated with the determination of the creatinine coefficient precludes its general use as

an index of obesity. However, the creatinine coefficient, combined with clinical appraisal, is an accurate index of obesity.

In conclusion, is it not true, that the diagnosis of obesity is not difficult if the physician has two good eyes and two good hands; but it is the etiological factor or factors which have produced the obesity, which are more important, and proper treatment.

ATTEMPTS AT CLASSIFICATION OF OBESITY:

The majority of authors of papers dealing with obesity in children begin their discussion with a classification of obesity. The most common classification, being that of exogenous (nonglandular) or endogenous (glandular); and than in perhaps the next paragraph, they conclude that in the majority of cases these types are not clearly defined.

Lindsay (28) believes all cases of obesity are the result of a peculiar metabolism, which in the last analysis is probably governed by glandular action.

Schultz (38) goes into a much more detailed classification. He divides obesity into four main types--(a) true hypothyroid obesity, (b) hypothalamic obesity, (c) comparatively rare forms of frontal lobe obesity, and (d) the simple exogenous, environmental obesity. According to his characteristic findings for each type (of which he has many), it would be very difficult to label each child who presents obese characteristics, as to whether he belongs to group a,b, c, or d, and if possible to do so, the treatment for any

obese child regardless of type is all the same. The reader is asked to refer to the discussion of treatment which is discussed in this thesis, where this concept is further clarified.

Concepts of this kind which classify obesity as exogenous or endogenous, and et cetera, presuppose that the energy requirements and oxidation processes of an obese child are different from those of a normal person. In "exogenous" obesity the food intake or energy expenditure or both are supposed to be markedly different from those of a normal person; whereas in "endogenous" obesity the actual economy of the metabolic process is supposedly disturbed. The apparent simplicity of this classification made it popular for a whole generation, and it continues to be used in spite of increasing and well founded evidence against its correctness.

Is a classification necessary? Is it not more important to determine the cause of a child's obesity, and treat the cause rather than to try and put a label on every case presented? The end results prove to be much better.

ETIOLOGY OF OBESITY: (CHARACTERISTIC FINDINGS)

Proceeding with the discussion from this point, what then are causes or percipitating factors of obesity in childhood?

Age--The age of onset of obesity varies. In Ellis and Tallerman's study (15) of 50 obese children it was found:

13	developed	obesity	in infancy
7	"	"	5-7 years of age
16	"	"	7-10 years of age
11	"	"	10-12 years of age
3	"	"	12-14 years of age

In eleven instances in the last two groups, the obesity was found to coincide with, or immediately precede (within six months,) the appearance of the first evidences of puberty.

Sex--In Ellis and Tallerman's study (15) the sexes were distributed equally. The same is true of other investigators; in some the females predominate. In Bruch's (5) observations of 142 obese children ranging from 2-13 years of age, 75 were females and 67 males.

Race--Race has been considered a factor because it was thought that the diet used in the average Jewish household of hospital class might be found to be especially inducive of obesity. Ellis (15) found 48% Jewish, 52% non-Jewish; Bruch (5) found 40% to be Jewish. Various nationalities were represented.

Family History--Heredity is often considered a very definite factor. Ellis (15) found that there was one or more members of the immediate family affected in 30 out of 50 cases; the mother in 13 cases.

Nixon (31) in his studies at the Childrens Hospital in Los Angeles found 75% of the obese childr en had a definite family history of obesity.

As to the patient's position in the family Bruch (5) found in 142 obese children--35% were only children, 35%

were the youngest in the family, 22.5% were the oldest child, frequently in a two child family in which the second child was more than five years younger, so that the patient had been in the position of an only child during a period of life when many important attitudes are formed.

Intelligence--Obese children are normal or above the average in intelligence. This is true with few exceptions, as adiposity occurring in the Laurence-Moon-Biedl syndrome which is discussed later.

Infection--The role of infection in the causation of obesity is apt to be assumed on insufficient evidence, especially when the coincident factors such as good food, rest, and prolonged convalescence are taken into account. Obesity of sudden onset, sometimes accompanied by other symptoms such as polyuria and polydipsia, has however, been recorded following specific fevers, chorea, and etc., sufficiently often to suggest the possibility of a definite causual relationship in some instances. It has not been proven however, and is merely mentioned as a possibility.

Growth Quoting Hilde Bruch (4), "Obese children differ only quantitatively in their physical makeup from the numerous possible patterns due to individual variations in growth. The rhythm of their growth is disturbed; it is not abnormal in a qualitative way."

She studied and observed more than 200 obese children (3). In all of them obesity developed before puberty. The weight ranged from 25 to 150% over normal standards.

Obesity was the outstanding physical finding, and no patient exhibited signs of an intracranial lesion. Following are her results and findings:

1. The heights of obese children fall more or less above the normal height, indicating an acceleration in statural growth. Not only height but many other body measurements are unusually large in obese children.

2. Children with the most advanced growth during preadolescence have a shorter growth period.

3. Two thirds of the cases indicated an advanced skeletal development, sometimes by several years--as shown by roentgenograms of the hands and wrists.

4. Puberty and menarche appear early in obese girls.

5. Nothing in this study justifies the assumption that obesity in childhood is in any way associated with hypogenitalism or delayed puberty.

6. Tall, rapidly growing children tend to have early puberty and stop growing at a younger age. The growth pattern of obese children represents an exaggeration of this normal trend.

Metabolic Speed-- The basal metabolic rate, that is, the measure of the level of energy expenditure necessary to maintain basal metabolic processes, is the resultant of multidinous, and increasing biochemical reactions; on these biochemical reactions depend the integrity of the body and the normal function of its cells. This so-called metabolic speed is innately controlled by the vegetative nervous system.

Any constitutional aberration in basal metabolic behavior obviously provokes a disturbance in fundamental body phenomena.

There are three well known methods which serve in the estimation of metabolic speed: namely, the assessment of skeletal development, the determination of the basal metabolic rate and the measurement of the total cholesterol of the serum.

Assessment of carpal development:--Skeletal development and inferentially, general bodily maturation may be measured roentgenologically. For such an examination, the carpal centers are the most accessible; assessment of their development by persons of unequal experience has given consistent results. "Assessment of the hand shows the smallest standard deviation, and is therefore, the skeletal area which gives the most generally satisfactory results." (47)

The clinician in applying the measure of skeletal development to determine the bodily maturation has preferred to assess carpal development. For all patients with delay in skeletal development a low metabolic rate was found. As a consequence, clinicians and roentgenologists have syllogistically assumed that delay in maturation, a retarded bone age, is a measure of the hypothyroid state and of the metabolic rate. Shelton (39) stated: "Bone age is a reliable index of metabolic speed in childhood." Skeletal development and metabolic speed therefore, are assumed to be parallel and commensurate. The logic and validity of

such a conclusion is questioned.

Determinations of basal metabolic rate:--The clinical measure of the metabolic rate is imperfect. Since a true basal state can never be accurately attained, the consumption of oxygen can be determined only under approximately standard conditions of rest and fasting. From such data, the basal metabolic rate is best calculated in terms of calories per square meter of body surface.

Various adventitious factors and the momentary mental and physical state of the subject may disturb the conditions under which such a test is given and may render the result inaccurate. Thus standard conditions are difficult to ascertain. This is especially true when working with children, however Talbot maintains basal metabolic rates can be determined for children over two years of age.

Measurement of total cholesterol:--"In most, if not all persons in health, the amount of cholesterol in the serum appears to be maintained at a constitutional level which is characteristic for each individual, and from which large deviations do not ordinarily occur." (23) The constancy of such a level is not grossly altered by the intake of food or by seasonal influences. The usually accepted average level of total colesterol of the serum in children, between the neonatal period and puberty, ranges between 100 and 164 mg. per hundred cubic centimeters of blood. (30) Many investigators have demonstrated a qualitative, or inverse, relation between the level of the total cholesterol and the basal metabolic rate. (24) When, however, the basal

metabolic rate is altered by non constitutional factors, such as drugs, the level of total cholesterol remains unchanged. (46)

With the three available methods of studying metabolic speed, and in view of questionable values of each Tobias and Stockford (46) sought to determine the most efficient procedure. 216 subjects were selected at random, for whom two or more of these measures had been made were used. Basal metabolic rate tests were conducted under identical conditions (overnight fasting, omission of all matinal food and fluids and a rest of one hour preceeding the test, temperature and humidity constant.) Body surface standards of Du Bois were used. The total cholesterol was measured during the same fasting state by the modified Bloor method. The subjects age range was representive of that encountered in the average pediatric practice. Neither age nor sex significantly influenced the data obtained. According to these men, when basal metabolic rate, the degree of carpal development and the level of total cholesterol are obtained for children with hypometabolism, neither age nor sex needs to be considered. Following are their results and conclusions: (46)

Carpal development:--For the patients with delay in carpal development the average basal metabolic rate was - 17.8 % and the average level of total cholesterol was 199.9 mg per hundred cubic centimeters of blood. The generally accepted corollary, that all patients with slow metabolic speed have a delay in bone development, is not valid.

Retarded bone age does not indicate a low basal metabolic rate. This fact is shown statistically by the coefficient of correlation, - 0.23 $\pm$ 0.016, between the basal metabolic rate the per centage of delay in carpal development. The extent or percentage of delay in carpal development, therefore, is not commensurate with the lowness of the basal metabolic rate or with the highness of the total level of cholesterol. When, therefore, carpal development is the sole measure used to determine metabolic speed, a low metabolic rate and a high level of total cholesterol will not be recognized in 64% of the patients. Only 36% of those patients with a significantly low basal metabolic rate and a significantly high level of total cholesterol showed a retardation in bone age.

Level of total cholesterol:--The level of total cholesterol is constant; it is relatively unaffected by the food intake or by seasonal or climatic variances; the procedure for determining it is simple and economical. These men (46) found that the level of total cholesterol is the most valid of all measures of slow metabolic speed. When the level of total cholesterol is known the approximate basal metabolic rate can be inferred. The reverse however is not true.

In summarizing the measure of bone age determines an approximate maturational level; as such, an indigenous low level of maintenance metabolic activity is present. The measures of the total cholesterol and the basal metabolic

rate serve to determine metabolic speed. The bone age, on the one hand, and the measures of total cholesterol and basal metabolic rate, on the other, fundamentally, therefore, subserve different functions, even though they may be remotely related.

With the preceding discussion of ways of measuring the metabolic speed, it then becomes important to know the basal metabolic rate and serum cholesterol of obese children.

Determinations of basal metabolism are widely used in the clinical study of obese patients, apparently in the expectation of arriving at some understanding of the underlying disturbance. Since hypofunction of the thyroid is associated with a depression of the basal metabolism, the conclusion is frequently drawn that a low basal metabolic rate in an obese person is indicative of hypothyroidism.

In dealing with growing children, who are constantly changing subjects, the interpretation of metabolic tests is even more difficult. The extensive work of Talbot and his associates (45) has shown that surface area standards, which give accurate information for the adult, are not satisfactory for children; they have based one set of standards on weight. On the whole, the problem of heat production in normal growing children has been adequately handled. No satisfactory method has been found, however, of interpreting the findings for children of abnormal bodily dimensions, such as obese children.

de Bruin (13) reported his observations on 13 boys

and 22 girls, 2 3/4 to 13 years old and 20 to 57% overweight. He proposed the use of a new standard for obese children and suggested "the standard for weight for the actual height increased by one third of the excess weight over the standard weight." This correction was arrived at empirically; it is offered as an expression of the metabolic activity, of the excess fat tissue, which, though less active than normal tissue, nevertheless cannot be considered completely inactive. No attempt was made to interpret the findings in terms of pathologic deviation or dysfunction of the endocrine glands.

What then is the clinical usefulness of estimations of basal metabolic rates of obese children? Does any connection exist between obesity and hypofunction of the thyroid gland? Is the serum cholesterol of value in differentiating between obesity due to hypothyroidism and obesity in which there is no evidence of thyroid disease? Hilde Bruch (2) attempted to answer these questions in one of her studies on obesity, and following are her conclusions:

47 boys and 49 girls, 2 to 14 years of age, and 20 to 108 % overweight were studied. The boys were on the average 43.9 % overweight and the girls 46.2 %. The degree of obesity is expressed as the percentage difference between the observed weight and the expected weight for height and age according to the Baldwin-Wood tables.

BMR of 72 obese boys and girls: (2)

Rates calculated in %	Talbot Height standard	Talbot Weight stand.	DuBois Surface area standard
above + 30----------	9 (12.5)	----------	----------
+ 10 to + 30----------	36 (50.)	6 (8.4)	1 (1.4)
0 to + 10----------	22 (30.6)	20 (28.)	6 (8.3)
0 to - 10----------	4 (5.5)	29 (40.2)	23 (32)

-10 to -30----------	1 (1.4)	16 (22)	40 (55.6)
below - 30----------		1 (1.4)	2 (2.7)

(The figures in parentheses indicate the percentage of the total number of cases and add up to 100 in each column.)

The averages for these three calculations are: for boys, -12.9 % on the basis of surface area, -5.8 % on the basis of body weight and + 15.9 % on the basis of standing height,; and for girls, -11.8 %, -3.94 % and + 15.3 %, respectively.

Determinations of serum cholesterol were done (2) for 43 boys and 46 girls. The level of serum cholesterol scattered widely, varying from 108 mg. to 301.3 mg. per hundred cubic centimeters. The mean value showed little difference between the sexes; it was 205.3 for boys and 195 for girls. The mean for the whole group of 89 children was 200.3 mg. per hundred cubic centimeters, with a standard deviation of ± 37.9. No relation could be established between the degree of obesity and the level of serum cholesterol. Further attempts to correlate the cholesterol level with other clinical findings, such as distribution of fat tissue, height development, bone age, sexual maturation, race and duration of obesity were equally unsuccessfu. No relation could be detected between the cholesterol level and either of the basal metabolic rates. This finding makes it unlikely that hypothyroidism is of importance, if active at all, in the pathogenesis of obesity.

Summary and conclusions: (2)

1- The total basal metabolism of obese children is

higher than that of normal children of comparable height and age.

2- Calculation of the basal metabolic rate according to different standards furnishes widely diverging results, none of which can be considered the correct one.

3- The mean value for the total serum cholesterol is 200.3 mg. per hundred cubic centimeters of serum, with a standard deviation of ± 37.9. The ratio of combined to free cholesterol is 2.60 ± 0.24.

4- No relation between the serum cholesterol concentration and the basal metabolic rate was revealed in this study.

5- Thyroid medication and its interruption fails to influence the serum cholesterol concentration and the rate of loss of weight.

Quoting Dr. Bruch (2), "The figures observed and the correlations calculated lead to the conclusion that determinations of the basal metabolism as ordinarily carried out and as conventionally reported are an untrustworthy guide when the clinician is seeking to estimate the part played by the thyroid in the pathogenesis of obesity. The basal metabolism of obese children should remain the object of clinical research until the many unknown components which enter into its computation are better recognized and understood. That no clinically useful information can be obtained from it, must be drawn with regard to the value of single determinations of serum cholesterol."

ETIOLOGY:

The etiology of obesity in children is largely theoretical with few exceptions. An attempt has been made to discuss this phase of the subject under two main divisions--obesity as a nutritional problem; and obesity as an endocrine problem. A third minor type is merely mentioned, and that is obesity associated with mental deficiency.

Obesity as a nutritional problem:

It has been pointed out and argued by many that in obese patients the energy expended is less than the caloric intake, such laziness and gluttony resulting in a positive energy balance. This however does not explain why the obese child actually consumes more food than is required to maintain his normal weight, or why his energy output is usually decreased.

Gordon (19) stated that in his experience overeating per se is an infrequent cause of obesity in children. Hess and Kunstadter (21) expressed themselves in a similar way, saying that obesity due to hyperalimentation is the exception rather than the rule. In both papers, however, dietary restriction was mentioned in the first place as necessary for effective treatment.

Ellis and Talleman (15) placed more emphasis on overeating. Obesity in the majority of their 50 cases was described as primarily exogenous in type, the patients showing a tendency to overeat, particularly with regard to carbohydrates.

Bruch (5) investigated this problem more thoroughly and following are her observations and conclusions. She observed 142 obese children, 2-13 years of age. The degree of obesity was recorded in percentage of weight excess as compared with the Baldwin-Wood tables. The children were grouped according to the severity of the obesity--moderately obese (20-39% overweight), severely obese (40-69% overweight), and extremely obese (70-110% overweight). An attempt was made to study the usual food intake of these children. The tendency to minimize the amount of food intake was encountered frequently, both in the children themselves and their parents.

A consideration of the weight curve of obese patients is important for a correct appraisal of the role of the food intake. The evaluation of this factor is complicated in children, since one is dealing with growing persons in whom a certain gain in size is normal. In Bruch's study (5) a gain of weight was arbitarily judged to be abnormal when it was more than 10 pounds a year in younger children. Most obese adults, after reaching a certain weight which is characteristic for them, keep their weight as constant as normal persons. Since their basal energy requirements are somewhat higher than normal, their food intake will be high, though not conspicuously so. During the period, however, then the condition develops and the body accumulates progressive amounts of fat, the energy inflow must be definitely above normal. In childhood, this developmental phase of the condition is the rule.

The food intake is decidely greater than normal during periods of excessive weight increase. It is not necessarily different from that of normal children at times when the weight is stationary or is increasing at a normal rate, although the patient continues to be overweight. (5)

The composition of the diet showed in a great number of cases (5) an astonishing uniformity. Nearly all children overeat on starchy food. Bread, cake, ice cream, candy and soups ranged first in all diet lists. On the whole, milk did not count among the favorite foods. In a great number of cases the dislike for milk was given as one reason for the addition of cake and other sweets to the diet. Not one child in the whole group had learned to eat a mixed, well balanced diet. Vegetables and salads led in the series of foods which were disliked. Fruits, with the exception of bananas and orange juice, came next. The attitude toward meat varied: Some children liked large amounts; others were extremely fussy. None of the children were allergic to eggs, but few had received them regularly in their diet. Most children soon complained about the monotomy of an egg for breakfast every day, and refused to take it.

Feeding habits--In Bruch's study (5) it was felt in the beginning that the main reason for the preponderance of starchy food was economic, since carbohydrates are the cheapest provider of energy. In a number of cases this opinion found support in the fact that the onset of obesity had coincided with a change in the economic status of the

family. The resistance shown against any change in the diet, however, was not on an economic basis. In cases in which the financial status was poor the family was advised not only about a well balanced diet but about economical ways of shopping, so that the new diet could be provided at even lower cost than the previous one with its large amount of cakes and sweets. In such cases it appeared obvious that the one-sided composition of the diet had developed not from financial considerations alone but rather from the inability or unwillingness of the patient (or his parents) to give up previously acquired habits and to add new experiences. The pronounced likes and dislikes for certain foods which are observed so regularly in these children may be described as representing an early, infantile phase of taste development,; economic considerations were only a contributing factor.

The same lack of training and adjustment to social requirements is reflected in the tendency to eat between meals. Not one of these children ate at mealtimes only; in a few instances there was no adherence to regular mealtimes. These children were used to receiving or taking food whenever they wanted it. In many cases the caloric value of the food eaten between meals exceeded that of food taken at mealtimes.

In conclusion, the food intake and increase in weight are correlated. This statement of fact does not, however, clarify the underlying cause of the condition. The discovery

of a high food intake merely represents another aspect of the same clinical problem; it is only one of the mechanical factors by which the body accumulates the large deposits of adipose tissue. For an understanding of the increased desire for food or the tendency of the body to grow beyond normal dimensions, a discussion of the mechanism by which the body regulates its food intake and weight is necessary.

Hunger and appetite--The bodily sensations which lead to the intake of food are hunger and appetite. Though often confused, they are essentially different--in physiologic basis, in localization and in psychic elements. Cannon (10) and Carlson (11) studied extensively the physiologic basis of the sensation of hunger.

Hunger is felt as a dull ache or gnawing pain referred to the lower sternal region and the epigastrium. It is caused by periodic contractions of the empty stomach, which do not begin until after the stomach has been empty for some time. Appetite is related to previous sensations of taste and smell of food; it has important psychic elements in its composition. Hunger is a primitive elemental sensation which imperiously demands relief; appetite implies wishing, longing or yearning for something especially desirable. The person influenced by an appetite is tempted; he seeks satisfaction and wishes to renew previous pleasures. Hunger may be abolished by the first few mouthfuls of a meal, while the appetite still calls. Thirst is similar to hunger as a powerful, persistent stimulus, which demands

relief. The appetite for drink closely resembles, the appetite for food with all its psychic implication.

Hunger contractions of the stomach occur when the sugar content of the circulating blood falls about 25%. Appetite is stimulated by the memory, smell or sight of agreeable food. At present no evidence is available indicating that any specific bodily change underlies the sensation of appetite. The initial secretion of gastric juice is stimulated by appetite. Although the two sensations thus may exist separately, they nevertheless have the same function of leading to the intake of food, and they usually appear together. The cooperation of hunger and appetite is probably the reason for their being so frequently confused.

Cooperating with hunger and thirst is the sensation of having had enough. It protects the organism against being overstocked with food and water. The feeling of satiety is little understood; it involves the element of contrast between the uncomfortable tension of hunger and the sensation of fullness, together with the lingering memories of the taste and smell of food. The sensation of satiety does not function adequately in obese subjects.

Growth and food intake--It is a truism to say that the intake of food is determined by the needs of the individual. These needs may have a physiologic or a psychologic origin. Under pathologic conditions, such as diabetes mellitus or exophthalmic goiter, the metabolic disturbance

results in an increased urge for food. The rapidly growing young organism requires more energy per unit of body weight than the fully grown. If the rate of growth and development exceeds the average, the need for food rises correspondingly. Abundant ingestion of food results in accumulation of fat deposits only when the intake is in excess of the metabolic and structural needs of the body.

The rate of growth of obese children exceeds that of normal children. The food intake, however, is far in excess of the demands of the organism for growth, and the surplus is stored as fat deposits.

Cortical Innervation and Morbid Hunger--Disturbances in the cortical innervation may cause morbid hunger, or bulimia. The older literature recorded several cases of hunger of this type. Mill (29) reported in 1887 the case of a 16 year old boy with a tumor of the second frontal gyrus and wrote: "He was ravenously hungry during his illness and bolted his food at all times." More recently Frazier (17) mentioned this symptom in a series of cases of tumor involving the frontal lobe.

In other cases that have been reported, morbid hunger developed after injury to the head. Fulton and his co-workers (18) recorded the increase of the hunger sensation in monkeys in which experimental lesions of the brain had been produced. Lesions of the prefrontal area were associated with ravenous hunger, which sometimes involved ingestion of two or three times the normal amount

of food. Despite this circumstance the animals lost weight.

It is probable that the ravenous hunger of some idiots and other persons mentally defective is due to irritation or destruction of the intestinal representation in the cortex or of tracts originating there.

The problem of increased desire for food in mentally normal obese children is different, however. Although many children have ravenous appetites which are uncontrollable under the conditions of their homes, decreased ingestion of food is not associated with symptoms comparable to the irresistable urgency of morbid hunger. The discomfort of the obese patient on a reducing regimen is rarely due to a true hunger sensation; it is the dissatisfaction of a person accustomed to having his desires and wishes fulfilled who suddenly meets denial. Within a few days, most obese patients adjust to the lower intake. Apparently the disturbance in appetite in the obese can be influenced from without, and the organism can satisfy its metabolic needs by drawing on its stores of fat.

Hypothalamic Regulation of Food Intake--The regulation of food intake by the central nervous system is not limited to the cerebral cortex. Another central representation has been discovered in the vegetative nuclei of the hypothalamus, the tuber cinereum and the walls of the third ventricle. Experimental lesions in the region of these vegetative centers can be associated with adiposity.

Keller and Noble (26) noted the great greediness and hypothalamic polyphagia of the animals in which adiposity developed after hypothalamic or infundibular lesions. In one dog it was observed an enhanced appetite for both milk and for solid food which continued until a new level for body weight was reached by the deposition of fat, after which the food intake returned to normal. This observation is in good agreement with the clinical course of obesity, which in many cases has been found to be a self-limited disorder.

The comparative neglect with which appetite and food intake have been treated as factors in the development of adiposity is in distinct contrast to the attention which thirst and fluid intake have received as symptoms of diabetes insipidus. Obesity and diabetes insipidus have many parallel features, and the may occur together as manifestations of lesions at the base of the brain. An outstanding difference in the mechanism of these two disorders arises from the circumstance that in diabetes insipidus fluid is excreted in excessively large amounts through the kidneys and is therefore lost to the body; hence the urge for more fluid is a conspicuous symptom. In obesity the superfluous intake is retained in the fat deposits and can be utilized again for the energy metabolism if need arises. The symptoms of hunger and appetite in obese persons are therefore less pronounced, and the whole process is reversible. Fat storage, however, is not an entirely passive process

determined only by the supply.

Neural Regulation of Fat Storage--Recent experimental studies on neural regulation of fat deposits in connective tissue carry convincing evidence that fat storage and mobilization are regulated through neural influences. These experiments were conducted by resecting the nerves of the fat bodies of mice and rabbits on one side, leaving the other side intact. Within 3 to 5 weeks an enormous amount of fat accumulated on the denervated side. The fat (nerveless) body continued to store fat even during periods of starvation, until nearly all other deposits were exhausted.

Since the hypothalamus initiates nerve impulses which are discharged through the autonomic nerves, one may surmise from these observations that the deposition of fat is regulated from the midbrain. Adiposity following lesions in the region of the hypothalamus may be considered as a sequel to the altered tonus in the innervation of the adipose tissue. Further study in this direction may eventually lead to an understanding of the various localizations of the fat deposits.

It is not inconceivable that the increased desire of some obese patients for food is secondary to the change in the fat metabolism. Specifically, this mechanism may play a role in all those clinical conditions in which adiposity develops suddenly in the course of a disease involving the hypothalamus region.

The incidence of pathological lesions in the hypothalamic region associated with adiposity, however, is certainly small. (e.g. neoplasms, or post encephalitis and chorea.) The similarity between obesity of known central origin and simple obesity makes it likely that analagous mechanisms are involved in the production of the condition. The polyphagia may be produced either by abnormally intense stimulation of normal "appetite centers" or by stimuli of normal intensity acting on hyperirritable "appetite centers."

Psychologic Aspects--

Emotional Immaturity and Overprotection--Lack of social maturation is an outstanding feature in the development of all obese children and manifests itself in many other ways. Many children are not capable of dressing themselves or of any other self care at an advanced age. For such children, deprived of normal outlets and satisfying contacts, the activity and pleasure of eating gain inordinate importance, and such statements of mothers as "His heart goes out for food," or "Eating is all he thinks of and lives for" describe the situation. Yet this strong attachment to food is not something that has suddenly befallen the child; it can always be traced to experiences in early life.

The maternal attitude of "overprotection" is often times present. One factor which may help to explain this in some patients is to be found in the unusual family position of obese children.

Influence of Upsetting Experiences--Not all children are obese from infancy. Frequently the increased desire for food became manifest only after some upsetting experience. Some patients develop an obesity because of a fear of some operation as appendectomy or tonsillectomy, and the whole proceedure is often used as a threat by parents. In a large number of cases the fear of sexual maldevelopment has similar effects. It may be a playmate who gives the enlightening information to the child, in the majority of cases a physician, a school nurse or some other person in authority has warned the parents about the poor prognosis for spontaneous development. Generally several weeks or months of unhappiness and conflict have followed the information. The patients, already overweight, have feared that something essential in their physical condition is wrong or is being neglected, and they resort to eating, their usual way of seeking comfort. This ensuing rapid increase in weight is sometimes considered additional evidence that the glandular system is disturbed. In other cases eat may be used as a form of sublimating aggressive tendencies.

Origin of Mother's Attitude toward Obesity--In Bruch's study (5) the following are some of the factors which helped to shape the parents attitude. Many of them were immigrants who had experienced poverty and cruel hunger during their own childhood. Giving food in abundance to their children represented to them something more than appeasement of a

bodily need. It also meant the safety of a peaceful country, the fulfilment of longings of their own which had been denied to them when they were younger. To such mothers it is inconceivable that they should train their children to restrict food intake. The special emotional significance of food to Jewish mothers may be a factor. However, regardless of nationality, to all of the mothers food stood for more than its caloric value; it represented health and security, and more often, love and affection.

Prenatal attitude of Mothers--The need to display their love so obviously or bid for their children's affection by bribing them with food arose in some of these mothers from feelings of guilt toward a child who had been unwanted or from self accusation of having neglected another child who had died. In more than half of the cases in which inquiry was made into the antepartum attitude of the mother toward the child, the information was given that the pregnancy had not been wanted.

In all cases food represented an important tie between mother and child. To many of these mothers the offering of food represented the only way of expressing their devotion, and the child received what he wanted and what his need for satisfaction and security endowed with high value.

The most recent psychologic studies on obese children is that by Bronstein and his associates (52). The purpose of their study was to determine whether the milieu of these children was of a characteristic type which predisposed them

to such a habit. Other purposes were to determine objectively the masculinity or femininity of these obese children, to study the educational level of the group through the use of achievement tests and to obtain some objective measure of the personality of the children. 35 children (24 males and 11 females) were studied. The tests used were the revised Stanford-Binet intelligence scale, form L, the new Stanford reading and arithmetic achievement tests, the pinter "aspects of personality" and the Terman-Miles attitude-interest analysis scale, form B.

From comprehensive physical studies of these 35 children, no ascertainable endocrinologic evidence was found as a basis for their obesity.

The mean intelligence of this group was above the mean of the population as a whole. There seemed to be a tendency for the children to fall into two classes, superior and retarded, almost 50% were above average and 25% below.

As far as can be determined from achievement tests, these children showed little or no difference from children of similar capacity.

Of the 24 boys, only 2 showed a tendency toward femininity. Thus so far as could be determined by the Terman-Miles test, this group showed no greater tendency toward femininity than might be expected of any comparable group. This is contrary to the view held by many.

The "aspects of personality" test indicated that the subjects were no different from other children as far as

ascendancy--submission was concerned, but that they had a tendency toward extroversion and instability.

There was a definite tendency for the children to be sensitive about their obesity.

A large majority of the children were reported to be interested in sedentary play activities, such as going to the movies, listening to the radio and reading.

There was no known history of obesity in the majority of the parents or siblings of these children.

Finally, Bronstein (52) emphasized that in the absence of any physical defects the adiposity of these children was probably contributed to by their sedentary habits, their abnormal appetites, parental attitudes and other environmental factors.

All the facts relating to food intake presented in this thesis cannot be considered as the cause of obesity; they are, however, contributing factors which have been found with such regularity in the history of obese children that they deserve to be looked on as more than accidental occurences and may be considered as having a specific significance. That identical or similar constellations are found in families whose children do not become obese but show other signs of maladjustment does not contradict this conclusion. Life situations and emotional experiences of this kind provoke increased desire for food only in certain type of persons and result in obesity only when such a person has a special tendency to store fat in larger

amounts than others and does not increase the energy expenditure correspondingly. It is possible that experiences in early life condition in a genetically predisposed person to such a way of reaction. (5)

Julius Bauer (8) was of somewhat the same opinion. His conclusions as to the cause of obesity are:

1. The frequent combination of overweight and overgrowth in children can not be explained by an excess in food intake.

2. It is due to an individual constitutional factor: the combination of abnormal genes both for obesity and overgrowth. (gigantism)

3. The time of manifestation of these abnormal genes may vary considerably. In exceptional cases the manifestation takes place in fetal life. These cases may be called macrosomia adiposa congenita according to Christiansen (12). In all other varieties with a later manifestation we have to deal with essentially the same pathological condition as far as its nature, etiology and pathogenesis are concerned. The difference is merely that such a macrosomia adiposa is not congenita. Abnormalities of habits, temperament or metabolism are the result of the abnormal anlage rather than the cause of obesity. This may or may not be associated with overgrowth.

Bauer's (8) ideas and theories are interesting to say the least, however, a little difficult to prove scientifically.

Obesity as an endocrine problem:--Until recently most of the literature considered hypothyroidism or hypopituitarism as causes of obesity in children. However, the findings of intensive growth and early maturation are not consistent with the characteristic findings in hypothyroidism and hypopituitarism; nor with the results of treating obesity with these hormones--they are consistent with observations of the growth-promoting effect of abundant nutrition.

This fact is best shown by Wilkins and his associates (51) in their studies on the characteristic findings in hypothyroidism in childhood.

In adults, many of the familiar physical signs of hypothyroidism depend on a lowered rate of metabolism and alterations in the circulation. In childhood, thyroid deficiency gives rise to additional changes due to its influence on growth and development. These changes may be grouped as physical and functional changes. Considering physical changes:

I. Skeletal changes--

1. Stunted growth--Hypothyroidism is one of the most important causes of dwarfism.

2. Skeletal proportions--The ratio of the skeletal segments remains that of a younger child, corresponding to the height age instead of the chronological age.

3. Naso-Orbital configuration--The characteristic facies so frequently seen in the child with hypothyroidism

is due largely to the peculiar naso-orbital configuration. The bridge of the nose is flat and broad, causing the eyes to appear widely spaced. It seems that the hypothyroid facies is caused, at least partly, by the persistence of infantile characteristics, just as the proportions between the upper and lower skeletal segments remain infantile.

4. Retardation of osseous development--It has been known for many years that hypothyroidism always causes delay in the appearance of ossification in the cartilagenous centers. However, endochondral ossification is at times definitely delayed in conditions which are not hypothyroid in origin, im such cases thyroid medication has no effect in the rate of osseous development.

5. Defective dental development--Development of the teeth is always retarded, and the delay is usually proportional to that in the endochondral ossification. In addition, the teeth which erupt during the period of thyroid deficiency are defective in structure and undergo early caries.

6. Epiphysial dysgenesis--This is probably the most specific of all the anatomic changes which are found in hypothyroidism. It is due to a disorder of the cartilages of the epiphyses and round bones leading to irregularities in their subsequent ossification. If thyroid deficiency exists during the period in which ossification normally occurs, the appearance of the deposition of calcium is considerably delayed. When calcification finally occurs,

it appears as multiple, small, irregular foci scattered over a considerable area of the cartilage. These grow larger and coalesce to form a single irregular center. According to the stage of the process, the roentgenogram may show multiple small centers of ossification or a single center which may appear either stippled, porous, fluffy or fragmented.

7. Retardation and defect in development of the brain--When thyroid deficiency occurs in the early years of life it causes delay in the development of the brain. If the condition remains untreated permanent damage may result. If hypothyroidism does not occur until the later years of childhood, there may be no defect in the development of the brain, and the I.Q. may be entirely normal, although the patient may be mentally sluggish and slow in response.

If hypothyroidism exists, one should find not only anatomic changes, but also definite physiologic evidences of diminished thyroid function.

II. Functional changes--

1. Mental sluggishness and physical inactivity are shown to more or less degree, by all patients with hypothyroidism. The torpor and slow mental reactions are distinct from retarded or defective development of the brain, and show definite response to therapy.

2. Diminution in the cardiac output and circulatory rate are constantly present. Evidences of a decreased circulation are a characteristic pale, grayish

color of the cheeks and lips and a circulatory mottling of the skin.

3. Laboratory findings--(a) Basal metabolic rate--The measurement of the BMR in young children or mentally subnormal children is unreliable, because of the difficulty in obtaining cooperation and establish basal conditions. However, when the Talbot height standards are used, children with hypothyroidism show definitely low metabolic rates, while obese children who do not have hypothyroidism have normal or even elevated rates. The BMR should not be used exclusively as a basis of diagnosis of hypothyroidism.

(b) Serum cholesterol and creatine excretion--The serum cholesterol in the majority of children with hypothyroidism is between 250 and 600 mg./100 cc. Normal children have 100 to 300 mg./100 cc. A cholesterol value below 300 or 325 mg./100 cc. does not exclude thyroid deficiency, but a concentration above this is suggestive of this diagnosis, if other causes of hypercholesteremia, such as diabetes, nephrosis or hepatic disease, are not present. There is a tendency for the creatine excretion to be low in the child with hypothyroidism. There are, however, great differences in the excretion of creatin in normal children, varying from 0.5 to 8 mg. per kilogram of body weight daily. It is therefore impossible to differentiate the untreated child with hypothyroidism from the normal child on the basis of the excretion of creatine.

(c) The serum phosphatase of children with

untreated hypothyroidism tends to be abnormally low, and it is restored to normal by thyroid therapy. (43) The normal values for serum phosphatase for children 2-10 years old is approximately 4.5 to 12 units. In hypothyroidism it is less than 4.5 units.

By comparison of these findings and those of obese children, discussed previously, it will be readily seen there is no resemblance.

The other type of glandular disturbance in obesity which is so frequently misdiagnosed is that of pituitary dystrophia adiposogenitalis of Frohlick's type. If correctly applied the diagnosis involves failure to mature sexually. Frohlick's syndrome is fairly common. It is characterized by adiposogenital dystrophy with excessive fatty deposits around the abdomen, hips, thighs, breasts, upper parts of the arms and back of the neck. In the male it produces a characteristic feminine appearance. The penis and testes show hyopdevelopment, and the pubic hair is feminine in distribution. As a rule the fingers are long and tapering. The patients are usually of normal intelligence, although they may undergo psychic changes secondary to their appearance and sexual development.

Etiology:--(20) In Frohlick's original case the condition was described as being the result of a pituitary tumor. Now it is conceded that the syndrome can occur with tumor, hemorrhage, infection or cyst within the sella turcica, or can result from fracture of the skull,

hydrocephalus, cyst or tumor located outside the sella turcica but able to produce pressure on the region surrounding or adjacent (the hypothalamus) to the pituitary gland. It has also been proved that the syndrome can appear in the absence of any demonstrable pathologic process within or about the sella turcica or in the hypothalamus. The present status of medical knowledge indicates beyond reasonable doubt that the seat of the lesion causing the obesity is in the hypothalamus and that the seat of the lesion causing the hypogenitalism is in the hypophysis.

May it be concluded however, that the ordinary obese child does not exhibit any of the dramatic symptoms of these exceptional tumor cases, except for the existence of adiposity. (3)

Frohlick's syndrome does exist, but it is not as common as it is so frequently diagnosed to be. This is particularly true of fat boys who present two particular characteristics, one of which is real and the other frequently only apparent. The first of these characteristics is the eunuchoid or female distribution of fatty tissue over the body surface. The second characteristic of fat boys, which is usually only apparent, is the small size of their genital organs. This, even more than their obesity is the reason which brings them to the physician. The physician, in his turn, frequently makes the statement of partial or incomplete descent of the testicles, and, on the basis of the association of the eunuchoid type of obesity

with apparent genital hypoplasia, he makes a diagnosis of primary hypogonadism (eunuchoidism) or pituitary dystrophia adiposogenitalis of Frohlich's type. Then, on the basis of the diagnosis, he advises glandular therapy, or sometimes more radical treatment, such as roentgen ray therapy or even surgical procedures.

Bauer (9) believes that in most of these cases the diagnosis is incorrect, and the treatment ill advised for three reasons: (a) The type of fat deposition which has been described is normal in the prepuberal years for individuals of both sexes, (b) the hypoplasia of the genital organs is usually apparent and not reat, and (c) the diagnosis of undescended testicles is made without adequate examination.

Rony (35) studied 50 cases of obesity in childhood and adolescence for the possible role of endocrine disturbance in the etiology of obesity. The study included history, clinical examination, measurements pertaining to growth, sexual and mental development, basal metabolism, sugar tolerance, and xray pictures of the sella turcica.

Disturbances of the pituitary and sex glands, moderately low basal metabolism without hypothyroidism, abnormal sugar tolerance, and mental deficiencies are frequent findings in juvenile obesity. Only six of the fifty cases studied showed no abnormal findings in at least one of these fields. However, the anomalies found represent deviations from the normal in both directions inasmuch as

in some cases hypofunction and in others hyperfunction of the same gland was found. This along with other observations strongly suggests that there is a relationship between endocrine anomalies and obesity, but it is not of etiological nature. (35)

Rony (35) states that the etiology of obesity, juvenile or adult, "exogenous" or endogenous is apparently uniform; it is a disturbance of the mechanism that regulates the body fat content, and is probably hypothalamic in origin.

Thus one is forced to agree with Wilder, as quoted by Nixon (31), "When, all is said on the score of the endocrine glands, it leaves one with the impression that their role in the production of obesity has been astonishingly overestimated." Wilder was of the opinion that "the theory of abnormal central irritability is more probable than any of the explanations of obesity based on postulations of endocrine disturbance or abnormal economy of energy."

Obesity associated with mental deficiency, (Laurence-Moon-Biedl syndrome.)

This is not a common syndrome. Reilly and Lisser (33) were able to collect 77 cases from the literature and since their article was published about 12 cases have been added. The classic syndrome consists of adiposogenital dystrophy, mental retardation, retinal degeneration (frequently pigmentary), polydactylism and familial occurrence. Skeletal defects of various types are frequently noted, as are various other anomalies, such as atresia ani.

Etiology--At the present time the etiology of the syndrome rests on the theory that it is the result of a genetic mutation in which at least three genetic factors are involved. Hecker and Warren (20) believe that the mental defect is the factor of primary importance and that the anatomic manifestations are merely secondary phenomena. They conclude that the Laurence-Moon-Biedl syndrome is the result of primary amentia, which in turn results from defective germ plasm.

TREATMENT OF OBESITY IN CHILDHOOD:

With increasing age obese children suffer more and more from their conspicuous and ungainly appearance, and they are under a severe social and psychologic handicap. A reduction of the excessive weight is indicated.

Treatment by dietary restriction:--Regardless of the various authors views as to the cause of obesity, they all include a restriction of the caloric intake in their recommended therapy.

There is general agreement that the success of a reducing regimen depends largely on the cooperation of the patient. Unwillingness to break a habit and expectation that some drug will make the accumulation of fat disappear are the most frequently mentioned reasons why some patients cooperate and others do not.

Circumstances are different in private practice and under conditions of a dispensary. The patient who consults a private physician with the intention of losing weight will,

as a rule, cooperate better than a patient who attends a clinic for some disability, is sent from one department to another and finally receives instruction for a low caloric diet because overweight is considered to be the cause of his complaint.

Obesity is a condition that per se rarely induces a patient to apply for treatment. In young children striking degrees of obesity escape the attention of the parents, as long as the child eats large quantities of food, the parents are not worried about the progressive increase in weight.

The evaluation of the results of treating children with obesity meets with a number of difficulties. Since the children are growing in stature and undergo rapid change in their biologic maturation, a report of the loss in weight or its percentage in relation to height is not entirely representative. The previous course of the weight development has to be considered also. For a young child who has been gaining excessively during the preceding years, constancy of weight or normal rate of gain during the following years presents a satisfactory result. In older children whose weight is above that of fully grown normal adults, actual loss in weight is desirable. The same holds true for extremely obese children to whom the excessive weight is a handicap in physical activities and social relations. That a rigid adherence to a low caloric diet produces a loss in weight is a well established fact. The

efficacy of any treatment of obesity can be appraised only the permanence of the result. The real problem of treatment lies in the unwillingness or inability of the children to adhere to the prescribed diet. It is the task of the physician to gain the cooperation of the patients and their parents. Since food intake is not an accidental isolated factor but plays an essential role not only for the physical needs but even more for the emotional demands of the patient, change in the dietary regimen should be accompanied or preceded by adjustment of other difficulties.

The reduction of the diet should not be so drastic as to impose on the child demands which are not within his power to follow.

The cooperation of the family should be gained or the possibility for it assessed before any dietary advice is given.

Since most of the obese children have been overprotected and have not learned to take responsibilities, the dietary instructions should be given as an inducement for the patient to prove that he can do something himself without supervision.

In many cases of serious maladjustment, cooperation regarding diet cannot be expected before the underlying disturbance has been relieved. In no case should one restrict the diet without offering to help the child find some other outlet and satisfaction.

Diet:--In general the diet is the all important

factor in the treatment of obesity in children. It should be sufficiently low in calories to insure a negative energy balance, Enough protein should be prescribed to assure a nitrogen balance, to protect the body proteins, and to allow for normal growth and development. The diet should include an ample supply of the vitamins as well as mineral salts, especially calcium and iron. Foods which allay hunger and give the greatest satisfaction are most desirable. It has been pointed out for instance, that meat sticks to the ribs the longest of the protein foods, and that hard boiled eggs have a higher satiety value than soft boiled eggs and potatoes are preferable to bread for the same reason.

Some doctors give only general rules for the regulation of the diet, which are usually sufficient, at least in beginning treatment. Lindsay (28) found the following to be satisfactory:

Do not eat:--fried food, sausages, pork, salt fish, cake, candy, jam, syrup, ice cream, pastry.

Eat small quantities of bread, potato and butter.

Eat largely of:--green vegetables, salads and fresh fruits, lean meat, cheese and fish. Take 30 ounces of milk daily, but no cream. Buttermilk or skimmed milk is sometimes preferable. Take only one small glass (6 ounces) of water before or during the meal. No table salt should be added at meals. Additional fat soluble vitamins should be given.

Concerning the question of water intake there seems

to be a controversy. Nixon (31) quotes Friese and Jahr as having proved conclusively that fat children show no greater tendency to retain water than do normal children and that the restriction of the fluid intake is of no importance in the treatment of obesity.

Kerley (25) gave his private patients a definite diet to follow. Following is his method of treatment:

Vitamin A,B,and D in generous amounts supplemented the feeding plan to compensate for the reduced vitamin A and D in the diet.

	CHO	P	F
Fruits--All cooked and raw fruits except banana, ½ cup or 4 oz.	14.4	0.7	
Fruit Juices--tomato, orange, or grapefruit, 1 cup or 8 oz.	18.		
Eggs--Boiled, poached or scrambled without fat, 1.		7.7	6.9
Cheese--Any variety, 2 oz.	1.	13.3	18.1
Meats--Poultry, lamb, beef, liver, or fish, 3-4 oz.		27.	5.4
Vegetables--Spinach, lettuce, celery tomatoes, string beans, cabbage, brussel sprouts, chard, cauliflower, broccoli, onion, asparagus, ½ cup at each of 2 meals.	8.	2.	
Salads--Any of above vegetables or fruits, served with lemon and mineral oil dressing, ½ cup at each of 2 meals.	8.	2.	
Deserts--Fruit, gelatin, sherbets, skim milk puddings, ½ cup at one meal.	30.	3.6	0.4
Breads--Preferably whole wheat or rye, ½ slice at each meal; Ry-krisp.	21	4.5	1.8
Beverages--Skim milk, not over 1 pint daily; cocoa made with skim milk; saccharine to be used as sugar, ¼ gr. tablet equalling 1 tsp. sugar.	25	17.4	1.
Total calories 1115.8	125.4	78.2	33.6
Approximately 1000 to 1200 caloric diet.	125	75	35

Sample menu:

Breakfast--1 cup orange juice
1 egg
1 glass skim milk
½ slice whole wheat bread

Dinner or lunch--3-4 oz. lean meat or fish
½ cup cooked spinach
½ cup oranges and grapefruit
Salad on leaf of lettuce
Mineral oil mayonnaise
½ slice bread
1 glass skim milk
Lemon sherbet

Lunch or dinner--cottage cheese on lettuce 2 oz.
Asparagus salad ½ cup
½ cup string beans
½ slice bread
1 glass skim milk
½ cup frosted raspberries.

Kerley (25) states that "In the application of the diet plan, reasonable latitude is permitted particularly in the older patients. This will be found necessary in order to secure continued cooperation over considerable periods constituting a re-education of the eating habits. At all ages one or two apples a day may be eaten at any time. Occasionally additional portions of meat, fish, or eggs may be given for the sake of variety. A portion of cooked cereal may be added to the breakfast of the older children if desired." In his study of 103 cases of nutritional obesity weight increase was not only retarded, but weight loss was made by each child.

Energy expenditure:--

As an adjuvant of dieto-therapy, exercise undoubtedly is of great importance. Increased activity should be encouraged, beginning with gentle exercises and increasing

in proportion to the child's tolerance. Swimming and bicycling are perhaps two of the most suitable forms of exercise for these children whose weight definitely hampers their movement. Some of these patients may be indolent and lazy, and may require a certain amount of urging to keep at their exercises. They may be discouraged by their lack of skill and success in games, and by the derision of their playmates.

Sharr (41) states that during the onset of puberty, games of endurance should be avoided, as well as sports which stress the competitive element, for they may expose the ungainly youngster to ridicule and hence to a reluctance to participate in athletics. Mass drills are good, and organized games have advantages, apart from the exercise, in terms of social behavior. Best of all, he believes, where the expense can be afforded, are individual exercises under expert supervision.

Medicinal treatment:--

Thyroid and pituitary preparations--The concept that obesity is the result of an endocrine disturbance has resulted in the idea that glandular medication is necessary for its treatment. The administration of endocrine products constitutes, for the majority of practicing physicians, the treatment of choice and often the only therapy in obesity of childhood.

Endocrine treatment of obesity refers in most cases to products of the thyroid and pituitary glands, although preparations of practically all endocrine glands have been

described as helpful, without their becoming so popular. Oral administration of thyroid gland deserves the merit of priority. It was introduced into therapy in 1894 by York-Davies in London and Leichtenstern and Wendelstadt in Cologne (7). These early reports emphasize the need for simultaneous dietary restriction and reserve thyroid medication for exceptional cases. The reported successes stimulated enthusiastic expectations for an effortless treatment of obesity. Only six years later, van Noorden as quoted by Burch (7), expressed his disappointment over the failure of thyroid therapy, saying that "today (in 1900) one would turn one's back on it." Despite this critical attitude of von Noorden's, thyroid has remained the most widely used medication for obesity.

Usage of thyroid is being defended on two grounds, as specific replacement therapy or as a calorigenic drug. The concept that obesity is the result of thyroid underactivity is no longer tenable in view of the convincing evidence that no metabolic disturbance exists which would justify this diagnosis. The opinion prevails, however, that obesity in childhood is per se an indication of endocrine disturbance. As late as 1937, Gordon (19) stated that the underlying cause of obesity in childhood was mainly endocrine.

Again, there is a great difference of opinion as to the effacacy of the endocrines. Nixon (31) says "The medicinal treatment seems of little importance even

though the use of various endocrine preparations has been all too common. Every obese child can be made to lose weight on diet and exercise alone, provided there is the proper cooperation between the child, his parents and the doctor. Occasionally, the addition of desiccated thyroid by mouth is advantageous as an adjunct to diet and exercise in the child who, on the usual regime, has established a level and is unable to further reduce his weight without the catabolic effect of the thyroid. The drug, however, should not be used indiscriminately in every case of obesity."

Bram (1) states that second to diet in importance is medication, and the most valuable ingredient in the materia medica is desicated thyroid. The brand of thyroid is important--American firms is four times the strength of European firms. It is best to adhere to a given brand of thyroid throughout the course of the patient's treatment. Generally speaking it is safest to begin with minimal doses of 1/40 to 1/20 grain, gradually increasing during a period of a few weeks to one or two grains administered two or three times a day. He believes that therapeutic hyperthyroidism may be employed with advantage to expedite recovery in those patients that are refractory to the small mode of attention; but this treatment can be administered only with the patient under a proper check-up regime. In cases presenting clear-cut evidences of anterior pituitary deficiency, thyroid medication may be supplemented by injections of anterior pituitary substance.

Lindsay (28) expresses himself by saying "Perhaps the most widely used remedy for reducing weight is thyroid extract, irrespective of whether there are signs of hypothyroidism or not. Thyroid does increase the rate of cell oxidation and so burns up fat. It also tends to with draw water from the system. Usually it has very little effect in raising the BMR in cases which are not frankly myxoedematous. Nevertheless, small doses under controlled conditions can be given, certainly without harm and often with benefit, especially at puberty. Pituitary preparations have their advocates, but the accredited results are not always convincing. Given by mouth, they have little effect, while the trouble and expense of giving them hypodermically is only justified under very exceptional circumstances."

Bauer (9) remarks that as to treatment, it must be emphasized again that the use of pituitary or testicular preparations is unwarranted unless there is a marked delay in the onset of puberty. In the majority of cases normal puberal evolution of the genital organs, as well as of the whole organism, will occur at the proper time, or slightly later, without the use of any therapy. Such glandular preparations are also without definite value in the reduction of the body weight. The use of thyroid preparations is rarely indicated.

Bruch (7) presented two interesting cases in a recent article, to show that obesity may develop in children receiving the very glandular products which are commonly

considered to be of specific value in the treatment of obesity. The cases are summarized:

Case # 1:--6 year old boy in whom obesity had developed during long continued treatment with gonadotropic substance for an undescended testicle. The patient had shown mild manifestations of allergy during infancy. He reacted to the injections with severe general symptoms such as irritability, sleepiness, and nausea. The treatment and his reactions to it, stimulated overprotective measures and overfeeding by the mother and provoked anxiety symptoms in the patient. The immediate mechanism of the obesity in this patient can be explained on the basis of overfeeding and inactivity. The concurrence of glandular treatment and development of obesity is not only a chronologic coincidence, but also a revelation of a casual interrelationship.

Case # 2:--The case of a 10½ year old girl in whom obesity developed after the age of 6 years. A marked increase in weight occured after an enlargement of the thyroid had been noted and the possibility of endocrine disorder had been suspected. Although continued clinical observation excluded symptoms of hypothyroidism, the family was unable to accept later reassurance on this point. The concept of a "lazy gland" had become deeply ingrained in the minds of the patient and her parents. The obesity had resulted from marked overfeeding and restriction of physical activities. These precautions and overprotective

measures had been provoked by the constant and exaggerated fear that asthmatic attacks, which had cleared long ago, might recur.

The reader is privileged to draw his own conclusions with regard to the endocrine therapy of obesity.

Benzedrine sulfate (amphetamine) in the treatment of obese children and adolescents:--More recently the search has been directed toward a drug which would depress the inordinate appetite of the obese and would thus insure a lowering of the caloric intake. During the past few years a number of authors have reported on the favorable effect of benzedrine sulfate, which is supposed to act by decreasing the appetite and increasing physical activity. Kunstadter (27) reports a study in which benzedrine sulfate was administered to thirty obese children between 2½ and 16 years of age. These children were selected after they failed to lose weight on a prescribed reducing diet. Many patients, in addition to a reducing diet, had received thyroid extract. The average weekly weight loss of twenty-six patients who received continuous treatment for over two weeks was 0.831 pounds per week. The greatest weight loss occurred during the first two weeks of treatment. There was little or no variation in the basal metabolic rate while under benzedrine treatment. The effect of benzedrine upon pulse rate and blood pressure was variable. A slight fall in systolic blood pressure occurred in nine patients, a slight elevation in four, and no significant change in four of the

twenty-five patients on whom determinations were taken regularly. Two patients developed a marked bradycardia. Forty percent of the patients experienced unpleasant reactions at the onset of the treatment. However withdrawal of the drug was necessary in only four instances because of severe reactions. The optimal effective dose of benzedrine for obese children apparently is between 10 and 30 mg. daily. Weight loss was primarily due to loss of appetite with subsequent decrease of food intake. Improvement in the state of mind, with increase of will power, was probably a contributory factor. Withdrawal of the drug or decrease in the dose usually resulted in a return of appetite and an increase in weight. Tolerance to the drug developed frequently, requiring gradual increase in the dose. Kunstadter concludes that although benzedrine sulfate is frequently an effective adjunct in the management of obesity in children, it is not a panacea. Social and psychologic adjustments, including improvement of the food habits, should be attempted during the course of treatment so that, following withdrawal of the drug, excessive food intake with regain of weight loss will not occur.

In view of previous reports of the use of benzedrine Bruch and Waters (6) conducted a study to determine the effect of a uniform dosage of 10 mg. of benzedrine sulfate on the weight of twenty obese children and adolescents. In order to evaluate the influence of suggestion and of regular clinic attendance, tablets resembling benzedrine sulfate but containing no drug were given in alternate

periods of four weeks. The loss of weight during the periods of placebo was slightly lower than during benzedrine administration. The difference was not of satistical significance. The best loss in weight occurred during the first few weeks of treatment. This cannot be explained as drug action, but is an unspecific effect depending on the patient's enthusiasm for a new therapy. The best loss of weight was observed in a control group of children who showed good cooperation and regular clinic attendance without receiving medication. In evaluating the effect of any medicine on the appetite and weight of obese patients, the influence of other factors, such as suggestion and better cooperation, has to be differentiated from the true pharmacologic effect. Although this study did not show a significant influence of 10 mg. benzedrine sulfate upon the weight loss, the findings encourage further observations of the effect of larger dosage.

BIBLIOGRAPHY

1. Bram, Israel, The fat youngster, Arch. Ped. 55: 381-387. June 1938
2. Bruch, Hilde, Obesity in childhood--II Basal metabolism and serum cholesterol of obese children, Am. J. Dis. Child. 58: 1001-1022. Nov. 1939
3. Bruch, Hilde, Obesity in relation to puberty, J. Pediat. 19: 365. Sept. 1941
4. Bruch, Hilde, Physical growth and development of obese children, Am. J. Dis. Child. 58: 457-484. Sept. 1939
5. Bruch, Hilde, Physiologic and psychologic aspects of the food intake of obese children, Am. J. Dis. Child. 59: 739-781. April 1940
6. Bruch, Hilde; Waters, Irene, Benzedrine sulfate (amphetamine) in the treatment of obese children and adolescents, J. Pediat. 20: 54-64. Jan. 1942
7. Bruch, Hilde, Obesity in childhood and endocrine treatment, J. Pediat. 18: 36-56. Jan. 1941
8. Bauer, Julius, Some conclusions from observations on obese children, Arch. Pediat. 57: 631-639. Oct. 1940
9. Bauer, Julius, Common diagnostic and therapeutic errors in the management of fat boys, M. Rec. 151; 89-92. Feb. 1940
10. Cannon, W. B., Bodily changes in pain, hunger, fear, and rage, ed. 2, New York, D. Appleton and Co., 1929
11. Carlson, A. J., The control of hunger in health and disease, Chicago, University of Chicago Press, 1916
12. Christiansen, T., Macrosomia adiposa congenita. New dysendocrine syndrome of familial occurrence, Endocrinology, 13: 149. March-April 1929
13. de Bruin, M., Basal metabolic rate in children with abnormal bodily dimensions, Am. J. Dis. Child. 57: 29 Jan. 1939
14. Dickens, Charles, Pickwick Papers, Bigelow, Brown and Co. Inc., New York
15. Ellis, R. W. B.; Talleman, K. H., Obesity in childhoood, Lancet 2: 615, 1934
16. Faber, H.; Roberts, D., Serum proteins and lipoids in eczema of infants and children, J. Pediat. 3: 78-83, July 1933.
17. Frazier, C. H., Tumor involving the frontal lobe alone: A symptomatic survey of one hundred and five verified cases, Arch. Neurol. and Psychiat. 35: 525. March 1936
18. Fulton, J. F.; Jacobsen, C. F.; Kennard, M. A., A note concerning the relation of the frontal lobes to posture and forced grasping in monkeys, J. Michigan M. Soc. 33: 175, 1934
19. Gordon, M. B., Endocrine obesity in children; clinical and laboratory studies and results of treatment, J. Pediat. 10: 204, 1937
20. Hecker, A. O.; Warren, V. C., Adiposity among mentally deficient males, Am. J. Dis. Child. 54: 1257-1271, Dec. 1937
21. Hess, J. H.; Kunstadter, R. H., Diagnosis and treatment of obesity in children, M. Clin. North America 22: 161, 1938

BIBLIOGRAPHY

22. Hunter, A., Creatine and creatinine, monograph on biochemistry, New York, Longmans, Green and Co. Ltd., 1928
23. Hurxthal, L.; Hunt, H., Clinical relationships of blood cholesterol with summary of our present knowledge of cholesterol metabolism, Ann. Int. Med. 9: 717-727, Dec. 1935
24. Hurxthal, L., Blood cholesterol and hypometabolism, Arch. Int. Med. 53: 824-831, June 1934.
25. Kerley, C. G., Nutritional obesity in children in private practice, J. Pediat. 19: 241, Aug. 1941
26. Keller, C. D.; Noble, W., Adiposity with normal sex functions following extirpation of the post. lobe of the hypophysis in the dog, Am. J. Physiol. 113: 79, 1935; Further observations on enhanced appetite with resultant adiposity following removal of the post. lobe of the hypophysis, ibid, 116: 90, 1936
27. Kunstadter, R. H., Benzedrine sulfate in obesity, J. Pediat. 17: 490-500, 1940
28. Lindsay, L. M., The overweight child, Canad. M. A. Jr. 44: 504-506, May 1941
29. Mill, C. K., Two cases of brain tumor, J. Nerv. and Mental Diseases 14: 707, 1887
30. Molitch, M.; Poliakoff, S., Cholesterol metabolism in children, Arch. Pediat. 53: 613-616, Sept. 1936
31. Nixon, N. K., Obesity in children, J. Pediat. 4: 295-306, March 1934
32. Pryor, H. B., Width-Weight tables, Am. J. Dis. Child.
33. Reilly, W. A.; Lisser, H., Laurence-Moon-Biedl syndrome, Endocrinology 16: 337-357, 1932
34. Roberts, L. J.; Blair, R.; Lenning, B.; Scott, M., Effect of a milk supplement on the physical status of institutional children, Am. J. Dis. Child. 56: 287, Aug. 1938
35. Rony, H. R., Juvenile obesity, Endocrinology 16: 601-610, Nov.-Dec. 1932
36. Rothbart, H., Basal metabolism in children of normal and subnormal intelligence, with blood cholesterol and creatinine values, Am. J. Dis. Child. 49: 672-688, March 1935
37. Ruhrah, John, Pediatrics in art--Infantile obesity, Am. J. Dis. Child. 45: 371, Feb. 1933
38. Schultz, F. W., What to do about the fat child at puberty, J. Pediat. 19: 375, Sept. 1941
39. Shelton, E. K., Osseous development as index of metabolic speed, with special reference to mentally subnormal and emotionally unstable children, Endocrinology 17: 667-676, Nov.-Dec. 1933
40. Shaffer, P. A., The excretion of Kreatinin and Kreatin in health and disease, Am. J. Physiol. 23: 1, 1908
41. Sharr, Endocrine problems in adolescence, J. Pediat. 19: 329, Sept. 1941
42. Talbot, N. F.,Basal metabolism standards for children, Am. J. Dis. Child. 55: 455 March 1938

BIBLIOGRAPHY

43. Talbot, N. B., Serum phosphatase as an aid in the diagnosis of creatinism and juvenile hypothyroidism, Am. J. Dis. Child. 62: 273-278, Aug. 1941
44. Talbot, N. B., Measurement of obesity by the creatinine coefficient, Am. J. Dis. Child. 55: 42-50, Jan. 1938
45. Talbot, N. F.; Wilson, E. B.; Worchester, J., Basal metabolism of girls. Physiologic background and application of standards, Am. J. Dis. Child. 53: 273, Jan. 1937
46. Tobias, M., Measure of metabolic speed in children, Am. J. Dis. Child. 61: 675-686, April 1941
47. Todd, T. W., Atlas of Skeletal Maturation, St. Louis, C. V. Mosby Co., 1937
48. Wadd, William, Cursory Remarks on Obesity, 1816
49. Ward, K. M., Study of blood cholesterol in childhood, Arch. Dis. Childhood 6: 329-342, Dec. 1931
50. Webster, Standard Metabolism of adolescence, J. Pediat. 19: 347, Sept. 1941
51. Wilkins, Lawson; Fleischmann, Walter, Diagnosis of hypothyroidism in childhood, J.A.M.A. 116: 2459, May 31, 1941
52. Bronstein, I.P.; Wexler, S.; Brown, A.W.; Halpern, L.J., Obesity in childhood--psychologic studies, Am. J. Dis. Child. 63: 238-251, Feb. 1942