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True hypothyroidism, obesity and metabolic insufficiency" indication for thyroid therapy"

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TRUE HYPOTHYROIDISM, OBESITY AND "METABOLIC INSUFFICIENCY" INDICATION FOR THYROID THERAPY?

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FORWORD

There has long been a tendency to lay the blame for obesity on "glandular trouble." Treating obesity with thyroid extract has been attempted time and again, rarely with satisfactory results. In general it may be said that physicians are aware of the fallacy of this treatment, and recognize that success is quite often temporary, and that the body restandardizes its physiologic mechanisms to the original status.

In recent years a new metabolic state has appeared in the literature, that of "metabolic insufficiency" or "hypometabolism." This syndrome, as championed by Dr. Paul Starr of California, is a distinct and separate entity from hypothyroidism. Since obesity has been observed as a common symptom, of this syndrome, the question of cause and effect again arises. Among the etiologic factors of obesity might the previously unrecognized syndrome of "metabolic insufficiency" be a substantial and prominent factor?

It is the purpose of this paper to attempt to answer this question, and in so doing I will discuss in turn hypothyroidism, metabolic insufficiency and obesity. Furthermore, I will attempt to show the relationship, if any be present, and to evaluate the use of thyroid drugs in the treatment of obesity.

MECHANISM OF THYROID GLAND

The first part of this paper is a review of the thyroid gland's mechanism of action and physiology as found in any standard text book. (9, 28)

The Thyroid Gland produces iodine containing amino acid compounds called thyroxin and triiodothyronine. The relative amounts of each of these in normal and abnormal states has not been determined. Collectively these two compounds may be referred to as the circulating "thyroid hormone", found in the plasma as thyroxin bound to plasma protein. Thyroid hormone acts by stimulating cellular oxidative processes throughout the body. An increased amount of the circulating hormone leads to increased oxidative processes and symptoms of hypermetabolism, while a decreased amount leads to decreased oxidative processes and hypometabolic symptoms.

The activity of the thyroid gland in its production of thyroxin is controlled by thyroid stinulating hormone, secreted by the pituitary gland, which in turn is controlled by the circulating level of thyroid hormone. A decreased amount of circulating thyroid hormone stimulates the pituitary gland to secrete greater amounts of thyroid stimulating hormone which in turn increases activity of the thyroid

gland and raises the level of circulating thyroid hormone. A high level of circulating hormone fails to initiate this cycle, with the end result of decreased production and maintenance of homeostosis.

Iodine is absorbed from the gut in ionic form. This iodide ion is picked up or trapped from the circulating blood by the thyroid gland by some unknown mechanism. The mechanism is enhanced by thyroid stimulating hormone or by low stores of hormonal iodide within the thyroid gland, while it is blocked by thyocyanate or perchlorate. Next the iodide ion is oxidized to iodine, again by an unknown mechanism - possibly by means of the cytochrome - cytochrome oxidase or peroxidase enzyme systems. This step may be blocked by thiourea and mercaptoimidazole compounds, and sulfonamides. The third step is the combination of tyrosine and iodine to form first monoidotyrosine and then diiodotyrosine. Two molecules of diiodotyrosine unite in another oxidative reaction to form thyroxin. 3, 5, 3' triiodothyronine is also formed; either by deiodination of a diiodthyronine molecule prior to coupling, or by deiodination of thyroxin by the peripheral tissues of the body. The product of the third step previously mentioned, thyroxin, is stored as thyroglobulin in follicles of the thyroid gland. Proteolytic enzymes hydrolyze this into

active polypeptides and release them into the circulation.

Symptoms of hypothyroidism are manifest when the circulating level of thyroid hormone is decreased. A marked deficiency causes a syndrome referred to as myxedema. Congenital deficiency is known as cretinism or infantile myxedema. There are varying degrees of this deficiency - a lesser degree being simply a hypometabolic state or simple hypothyroidism.

Cretinism and infantile myxedema are characterized by mental retardation, dwarfism, stocky build, a broad flat nose and wide set eyes, thick lips, spade-like stubby hands and retarded bone age (as evidenced by xray). Infants have the characteristic face, hoarse cry, large tongue, pot belly, and umbilical hernia.

Adult myxedema is characterized by a dull expression, puffy eyelids, alopecia of the outer third of the eyebrows, creamy pallor of the skin over the face, dry and rough skin elsewhere, doughy subcutaneous tissue, coarse brittle and dry hair, swollen tongue, halting and slurred speech, slow activity both mental and physical, anemia, constipation, increased sensitivity to cold, and muscular aches and pains.

Mild hypothyroidism may be responsible for vague symptoms of fatigue, emotional instability, multiple aches and pains, menstrual disturbances, obesity, and anemia.

Arnold Jackson (13) feels that hypothyroidism is the most frequent cause of daily headaches of persons living in the middle west. He also points out other common symptoms such as lack of pep, chilling easily, forgettfulness, dry skin and falling hair.

Diagnosis of hypothyroidism is based on clinical signs and symptoms as well as various labratory tests. It must be realized that deficiency may arise at several points; namely the pituitary gland, thyroid gland, insufficient iodide intake or absorption, or faulty utilization by peripheral tissues (7, 22, 31, 33).

The actual technique of the various studies will not be considered here. The basal metabolic rate is only a measure of metabolic activity and is increased to higher levels by emotional stress and tensions, and physical, autonomic or mental activity. The basal metabolic rate rarely errs on the side of being low, but rather being increased due to the above poorly controllable factors. Thus a normal basal metabolic rate may mask an actual hypothyroidism. (25) The protein bound iodine is one of the most accurate tests since it measures the circulating level of thyroid hormone and alleviates these means of error. (13) However,as will be discussed later under the heading of "metabolic insufficiency", the protein bound iodine may be

normal while peripheral utilization is the defect. (7,22,31,33)
Actual values diagnostic of hypothyroidism are :

1. Basal metabolic rate from minus 20 to minus 40.
2. Serum Cholesterol 300 to 700 mg%
3. Protein bound iodine $<$2 ug% (normal 3.0 to 8.5 ug%)
4. I ^{131} uptake - little or none

Pituitary insufficiency is differentiated by a normal serum cholesterol, and a rise to normal protein bound iodine and uptake of I ^{131} following treatment with thyroid stimulating hormone for two days.

Treatment of hypothyroidism is begun gradually with care to avoid sudden release of stored thyroid hormone. A sudden increase in metabolism in some patients causes toxic psychologic and cardiovascular symptoms. Again, the details of this technique are not important here. The total amount of dessicated thyroid substance or thyroxin seldom need exceed one or two grains daily. Some cases refractory to this dose and drug are further discussed under the heading of "metabolic insufficiency", where they do respond to other treatment.

METABOLIC INSUFFICIENCY

Metabolic insufficiency is a somewhat vague term which is not clearly defined or acceptable to some endocrinologists.

However, various physicians have used this phrase and defined it in terms of signs, symptoms and labratory studies. They have gone to the extent of treating these cases and have recorded some excellent results.

Dr. Joseph H. Morton of New York (18) feels that the syndrome of metabolic insufficiency (hypometabolic state) is perhaps one of the most common metabolic disorders seen in everyday practice. He points out the various reasons why it so often goes unrecognized or misdiagnosed. In the first place its recognition as a clinical entity is rather new; secondly it is characterized by vague and non specific complaints; and thirdly it can not be detected by thyroid function tests.

Dr. Morton defines the syndrome as follows:

1. Signs and symptoms of deficient metabolism
2. A low normal basal metabolic rate (-15 to -30%)
3. Normal thyroid function as indicated by protein bound iodine, radioactive I^{131} uptake, etc.

He rules out abnormalities of the adrenals, pituitary, and gonads as well as considering anorexia nervosa and malnuitrition before he assumes the diagnosis of "metabolic insufficiency". The difficulty of diagnosis of this syndrome is easily recognized here in view of the commonly observed falsely high basal metabolic rates which indicate a normal

result when in fact the true rate is low. Nothing further is gained from the "normal" protein bound iodine and iodine uptake. This means that diagnosis may depend on knowing ones patient and carefully evaluating his clinical signs and symptoms.

Dr. Morton studied 80 patients with hypometabolic symptoms, sterility, gynecologic problems, and obesity, the predominent symptom being chronic fatigue and the predominent sign, obesity. In these, the basal metabolic rate ranged from plus 6% to -40%. 55 had been previously treated with no excellent results and only 7 good results. The explanation of this comes from several recent investigators, who have found that thyroxin must be de-iodinated peripherally to triiodothyronine to be actively utilized. Because of some failure in this mechanism thyroxin therapy has been ineffective. Dr. Morton used sodium liothyronine (3,5, 3 - L - triiodothyronine) in doses ranging from 10 to 100 meq. daily. His results in two weeks were startling by comparison with the status of these patients prior to therapy as well as to those having previous thyroxin therapy. Of the 80 patients, 36 showed excellent results of complete or marked remission within two weeks; 35 showed good results with marked but incomplete response; and 6 had fair results of partial remission of symptoms;

while only 4 had no change. The most universal relief was from the symptom of fatigue. Also 22 of 38 obese patients lost an average of 10 pounds, and all 5 primarily obese patients lost up to 55 pounds in a 9 month period. These results were still maintained over 9 months later.

Dr. C. Robert Tittle of Albington, Pennsylvania, (31) conducted a similar study on only 8 subjects. His diagnostic criteria, treatment and results were the same as Dr. J. H. Morton's. With one to two months of treatment 6 of the 8 patients showed symptomatic and metabolic response. Fatigue, muscle aching, joint stiffness, constipation, sensitivity to cold, obesity and facial puffiness were among the symptoms alleviated or improved. Dr. Tittle points out that these patients had naturally not responded to thyroxin or thyroid extract when previously treated, since there was no deficiency of this substance as evidenced by normal results in thyroid function studies. Reiss and Haigh (22) suggest that in these patients the defect is due to insensitivity of the body tissues to thyroid hormone. Winkler and others (33) suggest a rapid destruction or inactivation of thyroid hormone in such patients, while Freedberg and his co-workers (7) suggested a defect in peripheral transformation of thyroxin to a more active form or impermeability of the tissues to thyroxin. At any rate L- triiodothyronine bypasses the defect and is

effective in the patients where thyroid extract has failed.(31)

Dr. Elmore M. Fields of Hempstead, New York (5) conducted a study with a total of 40 cases of"metabolic insufficiency", and 60 cases of hypothyroidism ; all of which he treated with sodium liothyronine (L- triidothyronine). All of his 100 patients ranged in age from 1 to 18 years. His diagnostic criteria for metabloic insufficiency was similar to that ofthe previous studies except here, obesity was not stressed as a common symptom. All 100 patients had been unsuccessfully or incompletely treated with thyroid extract and thyroglobulin prior to this study. Again, the results were startling: 57% showed excellent results when more than 90% of their symptoms subsided; 37% showed good results when 70 - 90% of their symptoms subsided. Only 5% demonstrated poor results with 50 - 70% subsiding symptoms; and 1 had less than 50% subsidence of symptoms. These last 6 and 4 others were treated with increased doses causing temporary toxic symptoms in 5 and further improvement in the other 5.

All of the investigators whose works were reviewed for this paper found a much lower incidence of toxicity and side reactions as well as less severity of those reactions.(5, 7, 18, 22, 31, 33)

OBESITY

Obesity is a metabolic disorder charaterized by the accumulation of excess adipose tissue. (29)

It is a complicated syndrome rather than a single disease entity involving not one but a complex or contributing factors; emotional, genetic, hypothalmic, and endocrine. (12) Obesity has been variously defined in terms of arbitrary excesses of weight beyond the average norm for height and stature or build. A person may be considered obese by definition when his weight exceeds 20% of his "ideal weight". (2)

It has been universally accepted that the basic cause of obesity is an intake of calories in excess of metabolic demands. (21) Obviously the rates of intake to output may be varied at either side. Basal metabolic demands and voluntary energy expenditure tend to decrease in old age with eating habits being maintained. The result is imbalance of intake and output with resultant gain in weight. However, many other factors are involved in imbalance of intake and output.

Genetic and enviromental factors have been observed in obesity. Mayer (16, 17) refers to studies in the United States which show that less than 10% of the children of normal weight parents are obese while 50% are obese if one parent is, and 80% are obese when both parents are. Because this might

be the effect of environment rather than inherited genes, the studies have been conducted with identical twins in separate environments.

The results have shown clearly that there is a definite hereditary factor and that eating habits have not been the main factor.

Another study substantiating these findings was conducted by Dr. Childes (4) who also found it necessary to work with identical twins in order to eliminate the environmental factors. In his series of 1000 obese patients 73% had one or both parents who were also obese. He refers to papers by Rony (23), Angel (1), and Gurney (11), showing similar results to his and also those of Mayer mentioned above.

Thorpe (30) leans more toward the environmental side of the picture. He first excludes emotional problems as the main factor on the basis that it is unreasonable to think such a large portion of the population is so badly maladjusted. He also excludes from his discussion of etiology the metabolic disorders and other rare specific diseases causing obesity, leaving "the common or simple type obesity". He proposes "the reasonable concept that the American people are the best-fed people in the world and have at their command the most food, in its most acceptable forms. We have the highest level of economy in the world and the most engrained habits of self

satisfaction of any people. This habit of satisfying our tastes by overindulgence of good food is, I believe, the most rational explanation for the great number of patients we must treat for excess weight." He further substantiates this proposal by reference to Pennington (19) who points out that Eskimos were never obese when following their accustomed diet of lean and fat meat but that obesity rapidly appeared among them when concentrated carbohydrate foods were introduced.

For the past 20 years psychiatrists have been accumulating well documented data of emotional problems in obese patients - usually only on selected individual cases. However, Bruch (3) has systemically observed large numbers of unselected obese children as well as a great many adults. She devides obese patients into two groups:

1. The developmental type obesity is due to an intrinsic defect of growth and development with existing emotional problems being secondary or independent.
2. The reactive group which consists of persons who are inactive and overeat as an expression or reaction to life's stresses. It is in some of the patients of this group that the overeating adaptation to life's stresses is so vital that it stands between health and serious mental illness. In referring to this classification, Hamburger (12) points out that obviously those of the last mentioned group should not be treated, as the effects would be more

damaging than the ordinary complications of obesity - hypertension, cardiac strain, diabetes, hernia and menstrual irregularities. Just how commonly emotional maladjustment is the etiologic factor in obesity remains to be determined. Precise evaluation is difficult; however, there has been found a high correlation of emotional stability in persons successfully reducing by standard diets and therapy, and conversely a poor response in those patients with clear cut emotional problems.

Endocrine obesity has been discussed in part under the section of this paper discussing hypothyroidism. In that instance diagnosis is clear cut by protein bound iodine and radioactive iodine uptake. It is practically universally stressed that metabolic disorders are quite rare as the etiologic factor in obesity. (29) The extension of this statement to include metabolic insufficiency has not yet been proposed or documented.

Dr. Elmore M. Fields of New York (6) states that , "patients who present a true endocrine obesity are primarily hypopituitary in function and, secondarily, hypothyroid, with a distrubance in salt, water and electrolyte metabolism. In addition to their caloric deficit they have a marked retention of salt and water, which primarily accounts for their obesity."

The mechanism of pan-hypopituitary dysfunction is further supported by Dr. Hilton Salhanick of the University of Nebraska.(24)

Treatment of obesity simmers down to a careful consideration

of the cause. If it be endocrine in nature as discussed by Fields, his recommended program is cytomel (triiodothyronine) or whole calf pituitary extract, and a diuretic such as Diamox. (6).

If there be a hypothalamic lesion or if a genetic predispostion exists there is probably little to be done. Emotional causes may be treated by psychiatric care in some instances but by no means should all emotional problems be so dealt with. Often the problems may be cleared up, alleviating further treatment. In others, where overeating is the patients' adaptation to stresses which cannot be alleviated the cure is worse than the disease and no therapy is recommended. (12)

In the great majority of "simple obesity" as discussed by Thorpe (30) the treatment has been standard diets, consisting of restricted calories with a high protein content and vitamin-mineral supplement. There are many variations, some quite radical with or without special exercise. Strong, (27) found that a 3 hour daily walk greatly increased emergy output without increasing the appetite once it became established as daily routine rather than sporadic. This was usually accomplished within one week.

Thorpe (30) criticises a starvation diet on the basis that it universally results in failure for two reasons;

1. It wastes all body tissues rather than just adipose tissue,

and has side effects of weakness, decreased energy, lethargy and constipation.

2. It doesn't correct the original bad eating habits in which large amounts of concentrated carbohydrates were ingested. Chronic undernourishment and malnuitrition result. He also criticises the usual low calorie diet on the basis of radio-active trace studies which show that the carbohydrate present readily replaces fat stores mobilized for energy (10) and that this same carbohydrate suppresses the fat-mobilizing action of the pituitary gland while at the same time increases the fat-depositing activity of insulin (19), (20). He further points out that standard low calorie diets supply insufficient bulk with concomitant continual hunger pangs and failure of re-education in a diet that will maintain the new weight.

Dr. Thorpe has had excellent success with a high protein, high fat diet in a rather exact ratio of 3:1 with elimination of virtually all carbohydrate. In this regime caloric intake was unrestricted as long as the protein: fat ratio was maintained 3:1 and no carbohydrate was added. Black coffee, clear tea, water and salt were allowed in unlimited amounts. The unlimited quantity of this diet alleviated all hunger complaints and side reactions of dieting. Most people on this diet have stabilized at about 6 ounces of lean meat and 2 ounces of fat, 3 times a day. Equivalent to 2100 calories/day.

The method of cooking and preparation is immaterial. Weight loss will occur at a rate of 12-14 pounds per month. Within about 4 weeks the patient complains of monotony, and fruits and vegetables containing 3% to 5% carbohydrate may be added for the sake of variety. This diet may be maintained for a long period of time with a weight loss of 6-7 pounds per month. Good eating habits are established and the new weight level can be easily maintained.

DISCUSSION

The physiologic mechanism of the thyroid gland and its circulatory hormone was discussed earlier in this paper. If an obese person (or any person for that matter), whose thyroid gland was normal were to be treated with thyroid extract or thyroxin; the circulatory blood level would be raised. By its natural physiologic mechanism this would decrease production of thyroid stimulating hormone, and in turn decrease production of natural thyroxin produced by the thyroid gland. The time lag in establishing this homeostosis results in a temporary increase of the body's metabolic rate, with subsequent weight loss. However, once the process is again balanced the metabolic rate returns to its original status, and so does the patient's weight. By increasing the dose of extraneous thyroxin

the viscious cycle may be perpetuated, in a stepwise fashion, way beyond normal levels of circulating thyroid hormone. To fall into this pattern of therapy would show extremely poor judgement on the part of the physician. As discussed early in this paper there are several accurate and practical studies to prove or disprove the diagnosis of hypothyroidism. If an accurate diagnosis is made, there is no question that thyroid therapy should be instituted. However, Elmore Fields (6) has expressed an opinion based on his clinical experience that a true endocrine obesity is more often on the basis of pan-hypofunction of the pituitary, and because of this, he institutes other therapeutic drugs as well as the thyroid drugs. Actually, as mentioned above, it is almost universally accepted that hypothyroidism is rarely the basic cause of obesity. (29)

While both Paul Starr (26) and Arnold Jackson (14) feel from their experience that hypothyroidism is a common disorder, neither feels that obesity is a significantly consistent finding in hypothyroidism. And both make direct statements that hypothyroidism is rarely the primary etiologic factor in obesity. The argument is further substantiated by C. Robert Tittle, (32) who points out that obesity is not as common a manifestation of hypothyroidism as is commonly believed. So the evidence and opinions of reliable authorities are quite unanimous that hypothyroidism is rarely the etologic factor of obesity.

In discussing the metabolic insufficiency syndrome one must immediately take issue with the means of diagnosis. Falsely high basal metabolic rates are quite common, and well understood on the basis of emotional and anxiety processes, thought processes, and muscle contraction. Paul Starr (26) was able to vary his own basal metabolic rate 26% without his technician being aware of any technical differences in the two tests run within one minute of each other. Because of this a low basal metabolic rate is frequently masked. On the other hand, in contradiction to statements that a low basal metabolic rate is significant, there are several causes for low values other than decreased utilization of the thyroid hormone. Each of these states must be ruled out before a low reading is accepted as completely significant. These include myxedema, cachetic states, psychic disorders, kidney disease, and adrenal deficiency. (15)

It seems fitting to discuss in turn each paper referred to in my presentation of metabolic insufficiency. Each author presented facts related to the multiple signs and symptoms of metabolic insufficiency. It was not their intention in any case to single out the one sign of obesity. However, this singling out of obesity does fit my purpose, and by personal correspondence with each author, I have attempted to fairly evaluate the relationship of obesity to this new syndrome.

It should be recognized to begin with that the metabolic insufficiency syndrome is a different entity from hypothyroid ism in that the gland activity and production of circulating thyroxin are normal. Yet, the clinical result is equivalent on the basis of target or end-organ failure. Further, the new syndrome is more vague and sub-clinical.

Because of the similarity of the two diseases, one is inclined to impulsively declare that the relationship to obesity is the same in both instances. I have tried to approach this problem more deliberately, giving every reasonable benefit to the possibility of this endocrine syndrome being a substantial factor in obesity.

Dr. Joseph Morton's study of 80 patients included only 5 who were primarily obese patients. All five lost significant amounts of weight up to 55 pounds and maintained this at least over the 9 months they were followed. The success of his therapeutic program of tri-iodothyronine can not be denied. The importance here is the fact that only 4% of the patients in his study were primarily obese. If we were to assume that Paul Starr is correct in stating that 5% of the total adult population is afflicted with the syndrome, (26) and we were to correlate that 4% of these patients were primarily obese and would respond to treatment, our calculations would place 0.2% of the total adult population in this category. And

this 0.2% would respond to the therapy of triiodothyronine. The figure would be higher considering only obese persons instead of the entire population, however, the percentage would still be of small significance. (less than 1%).

On the other hand, if we included the 38 patients who manifest mild obesity as one of several symptoms, our calculations would bring us to 2.1% of the total population or perhaps 5 - 10% of the obese population. Here the incidence takes on some significance, however, the therapeutic results are less encouraging. Only 60% show good results, and at that show no more than an average loss of 10 pounds. Thus a trend toward normal weight has been established, but the problem remains. I can not accept that these figures represent primary etiology in obesity to any significant degree. However, of incidental interest is the high degree of success with triiodothyronine in this series where some 70% had previously been treated with thyroid extract without good results.

Dr. C. Robert Tittle's study can not bear significant weight because it included only 8 patients. Even disregarding this, the results as applied specifically to obesity were not particularly encouraging. Only one of the four obese patients improved in this regard. By personal correspondence, Dr. Tittle indicated that not only was metabolic insufficiency a rare entity but that obesity was not a common symptom of either this

syndrome or hypothyroidism. (32)

Dr. Elmore Fields used a significant number of patients in his series which included 60 cases of hypothyroidism and 40 cases of metabolic insufficiency. In this study the patients ranged from one to eighteen years of age. It is highly significant that obesity was not a common symptom in any of these 100 children, but rather they were for the most part thin and underweight in both the metabolic insufficiency and hypothyroid groups. In fact, poor development was part of the diagnostic criteria in Field's study. (5) This point was strongly emphasized in personal correspondence from Dr. Fields. (6) in which he states : "As an etiologic factor in obesity, the syndrome of metabolic insufficiency is non-existent." He does express in his letter the opinion that in his practice of Pediatric Endocrinology, the true endocrine obese children are primarily hypopituitary in function. Since his study which is discussed earlier in this paper included 16 hypopituitary children who also showed retarded growth and no mentioned prominence of obesity; one must assume either the rarity of obesity in even this endocrine disorder, or that obese children were not included in the study on the basis of diagnostic criteria. There is little doubt that the former is true. It should probably be mentioned that Dr. Morton, in his slightly larger group of 80 adult patients with metabolic insufficiency, showed that obesity to some degree was his most common finding. This finding was

present in 43 of the 80 (54%). As already mentioned the 5 primarily obese patients were significantly helped by treatment, while the 38 who exhibited obesity as one of several symptoms were not significantly improved in this respect.

In correlating the material of these two workers, I feel that obesity, if present in these patients, is only an incidental finding. Drawing that conclusion dispels the conflict between these two fine studies. Both studies showed a high degree of response to triiodothyronine even when previous thyroxin therapy had failed to give good results. Since few of the obese patients showed a significant weight loss, which is not consistent with improvement of other symptoms, I think it is fair to assume that in these patients the obesity was only incidental even though common.

To add one more note to the same point, Drs. Freedberg and Kurland (8) expressed their opinion that metabolic insufficiency is not a common etiologic factor in obesity. In short, all workers referred to in this paper firmly agreed on this point, and made direct statements to that effect by personal correspondence.

Obesity is clearly an involved problem of multiple etiology. Reliable workers in the various fields provide many explanations for the cause of obesity, all of which employ sound principles and appear well justified. The

endocrinologists have quite fairly minimized the incidence of endocrine obesity. The psychiatrists present sound reasoning and good evidence of emotional states as a cause of overeating with subsequent obesity. Many studies have substantiated this, yet is is unreasonable to accept this as the only cause of obesity, for certainly not all or even a majority of obese patients can have such severe emotional problems as this. Bruch, herself, classified two types of obesity - one presenting obesity as the result of overeating and the other presenting emotional problems as the cause of overeating. Neither can one argue with the figures showing environmental and genetic factors involved in obesity. When 80% of the offspring of obese parents are also obese there appears to be a definite environmental component present with a suggestion of possible heredity. The studies with identical twins in separate foster homes bear out definite genetic components of obesity.

In the final analysis, I think Thorpe's views are most accurate and applicable. Obesity is caused by an excess of food in relation to the energy used, and this is the result of eating habits. Eating is pleasurable, and large quantities of rich foods are easily acquired with the present day incomes of the great majority of families.

If we accept as complete truth the observation of Anthro-

pologist and Arctic Explorer Dr. Vilhjalmur Stefanson (as reported by Pennington (19),) that none of the Eskimos were ever fat when following their accustomed diet of lean and fat meat, but that obesity occured among them with great rapidity when concentrated carbohydrate foods were introduced into their diets; we can then conclude that this change in diet is the primary cause of obesity. It is interesting that apparently none of them were obese prior to this. This would suggest that such factors as endrocrine imbalance, emotional states and heredity all play a relatively unimportant role in obesity and that perhaps evidence in this direction is on a coincidental basis.

Naturally to make such an uncompromising conclusion on the basis of one man's observations is hasty and unscientific. Nevertheless, many workers in this field believe this to be the true state of affairs, and I too am convinced that this is so. A high protein and fat diet provides considerably more bulk and satisfaction of hunger than concentrated carbohydrates which contain many more calories.

The trial of the lean meat and fat diet in more studies could well establish a new outlook on the treatment of obesity. One obvious drawback is the expense of such a diet as far as people of the lower class are concerned. The reasoning behind the success of this diet as presented by Thorpe is not entirely

sound. He maintains a different route of utilization of carbohydrates, proteins and fats; when actually all are broken down to the same two-carbon chains and metabolized from there. The mechanism by which pyruvic acid blocks mobilization of fatty stores is a true fact. But it must be recognized that any source of calories, not just carbohydrates, follows this same metabolic path. The pyruvic acid is formed from the products of protein and fat as well. Thus, the mechanism is one of limiting caloric intake, and the use of the high protein-fat diet is s sound method of attaining this end. One hundred gram portions of roast beef, beef liver, fudge and sweet chocolate provide respectively 224, 136, 411 and 503 calories. (34) If obese people can be re-trained in their eating habits to the lower caloric, hunger satisfying type of diet, they will naturally loose weight and maintain the loss once established.

CONCLUSIONS

1. Endocrine obesity is rare. Neither hypothyroidism nor "Metabolic Insufficiency" are significant as etiologic factors in obesity.

2. The primary cause of obesity is an over indulgence of food, specifically, concentrated carbohydrates.

3. The best therapeutic regime for obesity is establishment of proper eating habits associated with moderate daily exercise.

4. A diet consisting of lean meat and fat in the ratio of 3:1 in unlimited quantites, and no carbohydrates, appears to have good results. This diet coupled with moderate daily excercise, pending further studies in the future, may well become the treatment of choice in obesity.

5. Thyroid drugs are only indicated in states of endocrine imbalance as proved by protein bound iodine and other specialized tests. They have no use in the treatment of obesity as such.

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