ever, if the doctor has prescribed dieting and it has been unsuccessful, the child or the family may feel that they have disappointed the doctor before and may not return when they need help.

REFERENCES

A SUMMARY OF SOME CLINICAL ASPECTS OF OBESITY

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As a point of departure for this summary I wish to present an outline of mechanisms for the development of obesity. With minor changes, it is one recently proposed by Van Itallie1 and is based on the concept that there exist multiple etiologic factors—genetic, traumatic and environmental—in the development of obesity.2

The fact that there are two main categories—1) regulatory, i.e., with no primary metabolic abnormality, and 2) metabolic—does not imply that patients with obesity obey some law higher than that of the conservation of energy. Studies by Newburgh and his co-workers3 established this for all patients beyond argument from wishful nonbelievers. Mayer2 has stressed, however, that Newburgh’s work should not serve as a rigid, at times puritanical, obstacle to consideration of the problems of individual patients, a point of view inherent in Newburgh’s listing of different “proximate causes to which some persons respond (in part) by overeating.”4

Patients with a regulatory disorder may develop obesity as a result of increased caloric intake or decreased caloric output or frequently both. Under increased caloric intake, we have two categories, “organic” and “functional.” We have put quotes about both headings because this common form of categorization implies a sharp division in our thinking and sometimes interferes with our therapeutic approach to patients; most of us still feel more comfortable with a patient whose complaints are organic rather than so-called functional. We think of nephrosis and galactosemia as organic diseases but, in the present state of our knowledge, have to fall back on calling the increased glomerular permeability of the former and the enzymatic defect of the latter, lesions in chemical anatomy. Perhaps advances in electronmicroscopy5 and histochemistry6 will ultimately make use of this term more literal.

Injury of the hypothalamus in experimental animals as described by Brobeck7 and disease, such as encephalitis or brain tumors, may lead to hyperphagia and obesity.8 Wilder9 considered psychologic overeating a functional disorder of the hypothalamus, attributed by Mayer to a diminished arteriovenous difference of glucose in the hypothalamus. The details of this theory and the objections to its acceptance are summarized in a monograph of the New York Academy of Sciences.10

ADDRESS: Monument Street and Rutland Avenue, Baltimore 5, Maryland.
TABLE I

Types of Obesity According to Mechanism*

<table>
<thead>
<tr>
<th>I. Regulatory Obesity (No primary metabolic abnormality)</th>
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<tbody>
<tr>
<td>A. Increased caloric intake</td>
<td></td>
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<tr>
<td>1. “Organic”: central nervous system disease of hypothalamus</td>
<td></td>
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<tr>
<td>2. “Functional”: psychologic</td>
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<tr>
<td>Neurotic overeating</td>
<td></td>
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<tr>
<td>Non-neurotic overeating (cultural pattern)</td>
<td></td>
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<tr>
<td>B. Decreased caloric output</td>
<td></td>
</tr>
<tr>
<td>1. “Organic”: forced immobilization of convalescence</td>
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</tr>
<tr>
<td>2. “Functional”: awkwardness in sports—adolescent day dreaming</td>
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<tr>
<th>II. Metabolic Obesity</th>
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<tr>
<td>A. Neurologic: e.g., lipodystrophy (rare); adiposa dolorosa (rare)</td>
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<tr>
<td>B. Hormonal: hyperadrenocorticism (rare); adrenal carcinoma hypothyroidism (rare)</td>
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<tr>
<td>C. Enzymatic: genetic obesity in mice, obese-hyperglycemic syndrome</td>
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</tbody>
</table>

* Modified from Van Itallie and Mayer.

Some clinical circumstances under which one sees neurotic overeating have been described by Lourie in this symposium, and in numerous publications by Bruch and others. Here, indeed, is a psychosomatic complaint but, as Browne has said: “The psychosomatic fence is a bright, white-painted picket fence on a green lawn which has no fence on it, and the medical people constantly fall over the fence on one side and the psychiatrists fall off it on the other side, and there isn’t any fence there.” A frontal attack on the eating habits of an obviously disturbed, obese child is as meaningful therapeutically as prescribing tepid sponges for the fever of a child with meningitis.

Non-neurotic overeating is in this outline equated with cultural dietary patterns. The economic causes are obvious, be they overconsumption of readily available rich foods as in Denmark, or of bread and potatoes or other starches where family income is low. Bruch in describing the family frame of obesity has pointed out the special problems of immigrant families, and Weymuller has summed up another category under the heading: “The cooking is too good.” We would like to stress a cultural pattern now emerging for young infants. The ingredients are a permissive doctor, a compulsive mother (less frequently vice versa) a ready-made formula in which neither fat nor carbohydrate content can be manipulated, and the early introduction of easily available solids into the diet of a young infant whose intelligence and ultimate development are judged by his ability to swallow rather than to choke, and to gain weight rapidly. It is true that most obese babies do not remain obese, but it is also true that many obese infants become obese children, and many obese children become obese adults, particularly if there is a family history of obesity or if the patient is unable to follow prescribed treatment because of internal or environmental factors.

Hill has already alluded to the decreased caloric output of convalescence—it is seen after rheumatic fever or poliomyelitis or fractures. Awkwardness in sports leads to preference for a sedentary existence and if the patient is exposed to competitive athletic pressures in school, anxiety may lead
to further withdrawal and sometimes increased intake of food as well. An approach by the physician to the proper school authorities, to a youth club director, or to the father may obtain personalized athletic instruction of the patient, usually a boy, sufficient to overcome at least part of his awkwardness. This is why we have inserted arrows indicating a reversible reaction in the outline.

Adolescent day dreaming needs no special comment except to note that recent studies by Johnson suggest the importance of the diminished physical activities, particularly during the school year, of adolescents. A visit to the soda counter of a drug store near a high school will supply a vivid picture of caloric intake during group mooning.

So much, then, for the regulatory types of obesity, the subdivisions of which, alone or in combination, include the overwhelming majority of our patients. There are, however, certain additional considerations.

Most physicians are aware that some patients show a segmental obesity, more marked in either the lower or upper segments of the body. Striking examples are to be seen in Rony's book. We had a child in the pediatric metabolism clinic of the New York Hospital some years ago whose excess fat was confined to the lower half of her body, and whose mother showed the same distribution of obesity, suggesting a genetically conditioned local anomaly of fatty tissue metabolism. True, these subjects obey the law of conservation of energy, and yet there must be a difference in the storage or mobilization of fat by the upper and lower halves of the body. Its most extreme form is illustrated in Rony's book by the patient who developed lipoatrophy of the upper segment, most marked in the face, beginning at 10 years of age, with obesity in the lower segment beginning at 15 years of age. Because local fat deposition or atrophy can be produced by stimulation or inhibition of the autonomic nervous system, a neurologic component may well contribute to the pathogenesis of more localized disorders.

Although endocrine disorders rarely are responsible for obesity, there are occasional patients whose marked obesity stems from an adrenal tumor. Recently, Furth has reported production of obesity in mice by a transplantable pituitary adrenocorticotropic tumor.

Hypothyroidism may also occasionally account for obesity both in children and adults. In a series of more than 400 children whom we observed at the New York Hospital, there were four, less than 1%, with hypothyroidism as indicated by the response of the low basal metabolic rate and obesity to small doses of thyroid.

This brings us then to a consideration of a form of obesity specifically called enzymatic. This occurs in a hereditary form in a strain of mice that show hyperglycemia and an increase in lipogenesis from radioactive acetate when compared with that in mice with obesity due to injections of gold-thioglucose. These conditions have been studied extensively by Mayer and his co-workers. Although no syndromes have been described in human beings which resemble this enzymatic obesity, it is an example of another form of metabolic obesity.

In summary, there has been presented in this symposium, a description of genetic, physiologic and psychobologic aspects of obesity. From the clinical descriptions, it is apparent that usually the parent brings the patient to the physician when the symptom, obesity, has been present for some time. It is hardly likely that we can help in a hurry; neither the initial visit nor revisits can be handled in the short time or with the personal satisfactions that accompany so many other pediatric experiences. But if we do take time to recognize the multiple factors which may in the future contribute, or have already contributed, to the development of obesity, we are in a position to aid, either prophylactically or therapeutically, in better physical and emotional development. This includes the following:
An inculcation of attitudes of moderation in young mothers toward intake of food and gain in weight in early infancy, and of an understanding that bigger is not necessarily better.

Early recognition of beginning of excessive gains in weight which can still be modified by relatively simple manipulations of environment and attitudes.

The devotion of sufficient time to history taking to obtain a clear picture of the variety of factors that may contribute to obesity in any given patient as a guide to therapy.

Where it is desirable or necessary, it is the responsibility of the physician to arrange psychiatric referral. This responsibility also includes knowledge about the particular psychiatrist’s methods, to help insure the patient’s follow-up of this referral. Referral to the psychiatrist cannot come as a sloughing of responsibility by the pediatrician.

Finally, where psychiatric referral is not desirable, or desirable but not feasible, the pediatrician must give directly to the child, usually an adolescent, and to the parents sufficient support, both active and passive, in meeting the more general problems of adolescence, in the hope that this will help with the specific problem of obesity.

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