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EDITOR
GEORGE L. JORDAN, JR.

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Editor's Preface

THIS VOLUME of *Advances in Surgery* contains eight chapters concerning a variety of topics. The topics, as in the past, were chosen carefully by the editors to reflect areas of current interest or controversy. They reflect, therefore, a diversity of subjects. Only two, "Surgery for Bleeding Esophageal Varices" and "The Treatment of Ascites," are somewhat related, as both problems are, for the most part, complications of cirrhosis of the liver.

The first article entitled "Metabolic Surgery for Obesity" by Drs. DenBesten and Kuchenbecker is a concise and critical review of the place of operative treatment of obesity. The results of treatment by intestinal bypass are reviewed and different types of bypasses are evaluated. This information is contrasted to the experience with gastric operations for obesity, and leads one to conclude that currently there is a strong move towards gastric operations rather than bowel operations for weight control. The techniques of gastric operations are still in a state of flux; the current status of these techniques is described.

Endoscopic equipment now is being used increasingly for therapy as well as diagnostic purposes. In the last volume of *Advances in Surgery* a report by Wolff described the treatment of over 7,000 polyps of the colon by colonoscopy, removing both polypoid and sessile polyps in all portions of the colon. An article this year entitled "Endoscopic Sphincterotomy for Diseases of the Biliary Tree" by Drs. Geenen and LoGuidice describes one of the newer techniques of treating the disease of the biliary tract by endoscopic means. The technique of endoscopic transduodenal division of the sphincter of Oddi and removal of intraductal stones has been used extensively in Germany and is receiving increasing popularity in this country. The results that can be obtained by this procedure, as well as the risk and complications, are described. This is a procedure with which everyone should be familiar at this time.

The necessity for wearing a permanent appliance has been a disadvantage of the surgical treatment of ulcerative colitis and other lesions requiring total removal or total diversion of intestinal contents from the colon, since the first ileostomy was created. There has been a continuing search for techniques that would avoid the necessity of a permanent appliance. In an article entitled "The Quest for Continence in the Surgical Treatment of Ulcerative Colitis," Professor J. C. Goligher explores in detail the multiple surgical techniques that have been utilized, including the Kock pouch and ileorectal anastomoses in selected cases, as well as other techniques. He draws extensively upon the literature and his own personal experience in a profusely illustrated article.

The two major surgical complications of cirrhosis – bleeding esophageal varices and ascites – are described in separate manuscripts. Dr. George Johnson presents a review of the various surgical techniques that have been utilized for the treatment of esophageal varices, providing comparative data and a concise statement of the current status of these operations. Dr. LeVeen provides an up-to-date description of the technical details of the shunt procedure for ascites with a description of the possible complications and methods to avoid them in the utilization of this procedure.

With the exception of those surgeons interested in transplantation, immunology, in general, has not been a subject of great interest to surgeons. At the present time, however, immunologic aspects of surgical disease in relation to nutrition, infection and the ability to withstand anesthesia and operative intervention are becoming increasingly important, and great strides are being made in the understanding of altered immunologic competence and practical applications of these observations. An extensive review of this problem is presented in the article entitled "Surgical Immunology: Identification of Immune Elements and Opportunities for Intervention" by Dr. Barry D. Kahan. He provides a review of the recognized immune mechanisms and the current techniques for testing for immune competence. He points out the clinical surgical problems that are now recognized to be related. This article will plow new ground for many surgeons who read it. It will provide the basis for understanding of many articles that undoubtedly will be published in the surgical literature in the years ahead. There is no question but that ad-

vances in understanding of immunology as applied to surgery have a potential of opening whole new realms of surgical intervention, and improving results that can be obtained with current procedures commonly performed.

Dr. H. F. Seigler's article is a discussion of the subject of melanoma that points out the current concepts in evaluation of the disease, newer methods of classification and their relations to decisions concerning surgical management. Immunotherapy is being evaluated as one of the possible techniques to control this lesion. There has been a progressive change in the approach to this lesion as new information has been accumulated over the past several years and this manuscript provides a good and comprehensive statement of the current approach to this disease.

The last article entitled "The Acute Abdomen" is written by the editor and diverges somewhat from most articles on this subject. Rather than reviewing the classic history and physical and routine examinations that have been described for many years, emphasis is placed upon the evaluation of those patients in whom initial examination failed to provide an adequate diagnosis. A discussion of the newer techniques including peritoneal lavage, ultrasound, CAT scan and laparoscopy, specifically in the evaluation of the acute abdomen, are described with an attempt to put these in some perspective. Current advances and controversies in the evaluation and management of most of the common causes of the acute abdomen are then reviewed. In addition to lesions classically described, such as appendicitis, trauma is considered as one of the causes of the acute abdomen in view of the changes in approach to patients with abdominal wounds.

The editor wishes to express his appreciation to the authors who have contributed to this volume, as well as to the staff of Year Book Medical Publishers, for their valuable contributions in the preparation of this publication.

GEORGE L. JORDAN, JR., M.D.
EDITOR

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Metabolic Surgery for Obesity

LARRY DENBESTEN, M.D., AND STEPHEN KUCHENBECKER, M.D.

Department of Surgery, University of California, Los Angeles, and the Surgical Service, Veterans Administration Center, Sepulveda, California

OBESITY is a widespread symptom of an imbalance in an individual's body energy economy. In this modern era of abundantly available calories and a reduction in the physical activity of many Americans there is a widespread breakdown in the regulatory mechanism that maintains body weight at the most healthful level. The cause of this excess intake and storage of energy is multifactorial and incompletely understood.

Despite the lack of a clear understanding as to the cause of obesity, surgery has become increasingly utilized as a therapeutic modality in morbid obesity. Future usage of surgery as treatment of morbid obesity requires ongoing critical evaluation of the success and risks of each type of procedure to maximize the therapeutic benefit for each patient.[1]

Definitions of Obesity

Definitions of obesity must distinguish those individuals who are significantly overweight from those with life-threatening, or morbid, obesity. *Obesity* is that condition in which an inordinate excess of adipose tissue has been accumulated, i.e., more than 20% above the ideal body weight in relation to age, sex, height

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and skeletal frame.[2] *Morbid obesity* has been defined as 100 lbs overweight or 200% or more of desirable weight as defined by the Metropolitan Life Insurance Company.[1] The scope of the problem of obesity in the United States can be seen by the fact that severely obese males (30% over desirable weight) and females (50% over desirable weight) between 20 and 74 years of age total an estimated 2.8 and 4.5 million, respectively.[1] The impact of this excess adipose tissue on the health and quality of life of the obese and their inability to lose weight has spawned a great volume of scientific investigation and treatment modalities.

Classification of Types of Obesity

Three systems for the classification of obesity are widely accepted. Each of these classification systems attempts to focus on the etiology of the caloric excess resulting in obesity, but with a special emphasis on a specific aspect of the etiology. The first classification divides obese subjects on the basis of the source of excess calories, namely, *endogenous* and *exogenous*. The second focuses on mechanisms by which excess calories cause obesity, namely, *hyperplastic* (an increased number of adipocytes) and *hypertrophic* obesity (increased fat loading of existing adipocytes). The third classification attempts to catalogue the *psychologic, biologic* and *pharmacologic* etiologic aspects of obesity, any or all of which may play a role in the obesity of a specific patient.

In the *first system of classification* obesity is classified as endogenous when secondary to an endocrinopathy and exogenous when resulting from intake of superfluous calories.[3] The *second classification* of obesity is descriptive and is based on the number and size of adipocytes.[4-9] The former denotes excess triglyceride storage in existing cells (hypertrophic obesity) and the latter an increase in number of adipocytes (hyperplastic obesity). These features usually coexist in both lifelong mild obesity and life-threatening morbid obesity. This anatomic distinction assumes genetic factors in the etiology of the obesity of certain patients. Thus, juvenile-onset (before the age of 20) but not adult-onset obesity is characterized by hyperplasia, an increased total number of fat cells, which begins early in life and is irreversible. Hyperplastic obesity, often accompanied by hy-

pertrophy of the adipocyte population, often is a more severe form of obesity characteristically unresponsive to treatment and associated with a high rate of weight gain following any weight loss.[10, 11] Pure hypertrophic adult-onset obesity is a milder disorder, and is more often successfully controlled by dietary restrictions.[11-13] The *third classification* of obesity represents an effort to provide the proper focus for rational future therapeutic efforts when more effective control of the various complex etiologies may be possible. Etiologic categories include (1) hypothalamic obesity,[14] (2) endocrine obesity, (3) physical inactivity, (4) dietary obesity, (5) genetic obesity, (6) drug-induced obesity,[6] (7) nervous or psychogenic obesity and (8) social and behavioral obesity.[10, 11, 14-16] Most types of obesity probably have a multifactorial basis.

Our current level of understanding does not allow precise identification and treatment of the various etiologies. *Hypothalamic obesity* is very rare and seldom of severe magnitude (over 250 pounds). It usually is characterized by hypertrophy of adipocytes secondary to increased food intake rather than hyperplasia. The most common pathologic lesion is the craniopharyngioma, although other intracranial neoplasms have been implicated. All are associated with other commonly seen manifestations of intracranial disease.[15] The chief interest in this unusual etiology of obesity stems from the central role that the ventral hypothalamus plays in the coordination of energy balance.[14, 15, 17-20] The lateral regions of the ventral hypothalamus (VLH) control the initiation of food-seeking behavior and feeding whereas the medial region (VMH) is considered the satiety center and normally has a dominant inhibitory influence over the hunger center (VLH).[12, 14] Fluctuating nutrient status and long-term energy balance are factors that modulate the interaction of these control centers to stabilize body weight at a set point.[13] This balance seems central to the question of obesity that is the end product of inappropriate control manifested by excessive intake, whether this excessive intake is due to primary errors in the controlling mechanism, the hypothalamus, or inaccurate input and/or feedback to these centers.[11, 12, 14, 21] *Endocrine obesity* comprises those hormone derangements that result in excess accumulation of fat, such as Cushing's syndrome[22, 23] with its characteristic truncal obesity, hypertension and glucose intolerance, the hyperinsulinism of insulino-

mas and adult-onset diabetes[24] and the Stein-Leventhal syndrome[25] with its characteristic adrenal and ovarian hyperfunction and hyperphagia leading to weight gain. The magnitude of obesity in this class is mild and reversible with correction of the underlying endocrine abnormality. *Decreased physical activity* is a major factor in human obesity[26, 27] and is likely to become more prominent as our technology provides more energy-saving appliances, tools and recreational pursuits that revolve around the vicarious involvement in activity while expending little energy. *Dietary factors* contributing to obesity are related to speed of eating, number of meals per day and quantity of fat in the diet. Data from animal studies[21, 28-30] indicate that ingestion of a set number of calories in one or two meals and eating rapidly increases body fat content when compared to eating the same amount slowly as frequent meals. Furthermore, increasing dietary fat and thereby caloric density leads to weight gain in rats (hypertrophic obesity) when compared to animals receiving the same total number of calories but with a lesser caloric density. *Genetic forms* of obesity probably include an inheritable predisposition to hyperplastic obesity[11, 31-33] as well as several rare syndromes. These include Laurence-Moon-Bardet-Biedl,[34] Alström,[35] hyperostosis frontalis interna[36] and Prader-Willi,[37] in which obesity is one of several significant features. *Drug-induced obesity* is related to administration of phenothiazines, estrogens and cyproheptadine.[6] *Nervous or psychogenic obesity* is predicated on the feeling by the patient that eating removes noxious emotional states and thereby is reinforced.[38] Social and behavioral influences on obesity are the result of positive social and behavioral reinforcement for eating. For example, evidence has accumulated that the experience of eating is more pleasant for an obese individual than one of normal weight,[38-40] especially when the energy control mechanisms sense that the set-point weight is higher than the individual's current weight.[41-43]

Physiology of Obesity

Obesity is associated with a number of endocrine and metabolic changes that seem to be the result of positive energy balance and are reversible by a return to normal body weight[12, 13, 44, 45] (Table 1).

TABLE 1.—Some Endocrine and Metabolic Features of Obesity*

Plasma glucose	Increased; basal and postprandial
Glucose tolerance	Impaired
Plasma insulin	Increased; basal and postprandial; increased response to common secretagogues; normal or increased rate of removal from the circulation
Plasma proinsulin	Increased; normal in relation to hyperinsulinemia
Insulin sensitivity	Reduced; mainly associated with insulin resistance
Plasma adrenal glucocorticoids	Normal or slightly increased; increased rate of turnover
Plasma growth hormone	Reduced response to common secretagogues
Plasma glucagon	Normal or slightly increased
Plasma free fatty acids	Increased in fed state; increased rate of turnover; impaired response in fasting
Plasma triglycerides	Increased; increased rate of turnover in VDRL
Plasma cholesterol	Increased; increased rate of turnover
Plasma glycerol	Increased; increased rate of turnover
Plasma amino acids	Increased arginine, leucine, tyrosine, phenylalanine, valine

*From Bailey.[12]

Increased basal and stimulated levels of circulating insulin are found in obesity.[46-50] These increased insulin levels have a linear correlation with the mass of excess adipose tissue.[51] Increased levels are secondary to increased production of insulin by the beta cells of the pancreatic islets.[52] The hyperinsulinism of obesity may be the result of an imbalance in the hypothalamic regulator of the pancreas, which acts via the vagus nerves. Elevated levels of glucose, amino acids and fatty acids are also present and are potent stimulants to insulin secretion.[53] In addition, overfeeding induces secretion of insulinogenic hormones by the gastrointestinal tract.[54-56] The fact that increased insulin and glucose levels coexist indicates a fault in the tissues' responsiveness to insulin, i.e., insulin resistance.[57] Hyperlipidemia and hypercholesterolemia are also seen in obesity, as is hypertrophy of the adipocyte population. These metabolic abnormalities as well as the hyperinsulinism, insulin resistance and decreased glucose tolerance of obesity usually are reversed by weight loss.[13]

Risks of Obesity

Death rates for the morbidly obese are 11 times greater than for the nonobese.[1] The risks of obesity include: *diabetes mellitus, cardiovascular disorders* (sudden death predisposition, hypertension, atherosclerosis, ischemic heart disease, cerebrovascular accidents, varicose veins), *respiratory disorders* (alveolar hypoventilation), *renal disorders* (nephritis), *osteoarthritic conditions, gastrointestinal disorders* (appendicitis), *hepatic and biliary disorders, psychologic disturbances, reproductive problems* (menstrual disturbances, complications of pregnancy), *hiatal hernia* and *complications of surgery and anesthesia.*[1, 2, 50, 58, 59, 76] Weight loss is the most effective therapy for reducing the risks of morbid disease.[50, 77, 78] Risk-benefit relationships for the moderately obese are less well known.[60, 79] Benefits associated with weight loss include frequent amelioration of hypertension, decreased risk of gallstone formation, reversal of cardiorespiratory impairment, reduction of hypertriglyceridemia, improvement and sometimes disappearance of maturity-onset diabetes, greater agility and mobility and, frequently, a striking psychosocial rehabilitation.[1, 64, 80-82] In addition to the risks of illness, a general discrimination in social and economic opportunity plagues the morbidly obese; this includes opportunities for employment, education, insurance and medical care.[1, 50] This discrimination is reversible with weight loss.

Nonsurgical Treatment

Therapy for obesity must be chosen on the basis of the patient's degree and type of obesity, motivation and the relative success of various available modalities. Increases in physical exercise and decreases in caloric intake form the basis of most dietary treatments of obesity.[11, 21] Paradoxically, increasing activity from very low levels actually may decrease food intake or restore an appropriate and more favorable relationship between exercise and intake.[83] For increasing amounts of exercise, food intake increases in a direct relationship until a certain point is reached, at which time the advantages of exercise are magnified.[11, 12] Unfortunately, this level of exercise is beyond the ability of most seriously obese individuals. Restriction of calories with special restriction of carbohydrates and adjustment of the

eating pattern to frequent smaller meals rather than fewer larger meals are also advantageous, especially for mild to moderate adult-onset (hypertrophic) obesity. The limiting factor in aggressive nonsurgical treatment of obesity is that only one-third to two-thirds of morbidly obese persons who seek such treatment will remain on these regimens for sufficient periods to lose a significant proportion of their excess weight. Of those who are successful, only about 10–20% are able to maintain the loss for more than a few years.[1, 84, 85] This high recidivism rate possibly reflects the persistence of an elevated body weight set point and is the chief reason for abandoning dietary therapies.

Drugs, lay groups emphasizing counseling and positive and negative reinforcement, hypnosis, voluntary incarceration programs, aversion therapy and jaw wiring all have their advocates. Although these therapies are associated with significant weight loss in some, average weight loss is poor, and all are hampered by a high recidivism rate,[84, 86] which makes them inappropriate as a sole therapy modality for the morbidly obese.

Behavior modification treatment of obesity as initially described by Ferster[87] and applied by Stuart[88] has been associated with moderate loss of weight and maintenance of that weight loss at 1 year.[89, 90] It is apparent that behavior modification deserves a place in the multidisciplinary approach to obesity and may have a special place in the program for total rehabilitation of patients following gastric bypass or gastroplasty.

However, behavior modification techniques alone have not yet been successful in inducing an adequate degree of weight loss or in maintaining weight loss in the majority of patients who are morbidly obese.[91, 92]

Surgical Treatment

Surgical treatment of morbid obesity was born out of the despair surrounding repeated failures of other available treatments and recognition of the substantial risks of morbid obesity that could be ameliorated by weight loss.

Intestinal Bypass

The first experimental animal work on jejunoileostomy was reported 25 years ago by Kremen, who studied its effects on ni-