Diet and Diabetes

ZACHARY T. BLOOMGARDEN, MD

This is the second of a series of articles on presentations at the American Diabetes Association Annual Meeting, Orlando, Florida, 4–8 June 2004, addressing issues related to diet and current approaches of surgical treatment of obesity.

Effects of specific macronutrients

John Miles (Rochester, MN) discussed effects of meal ingestion on protein metabolism, acute effects of dietary protein and amino acids on glucose metabolism, and effects of protein in persons with diabetes, noting that insulin decreases endogenous proteolysis during a mixed meal. In a study of protein ingestion in persons with diabetes, plasma amino acids and, subsequently, urea levels increased. Usually, increasing protein ingestion is associated with protein oxidation, which is important as there is no physiologic mechanism for accumulation of protein stores, while conversely we adapt to decreasing dietary protein by decreasing amino acid oxidation. The effect of protein on glucose metabolism can be studied by measurement of arterial-venous balance, with evidence that amino acids displace glucose as metabolic fuels. Assessing independent effects of protein and of insulin on systemic and regional glucose metabolism, Miles showed evidence that insulin decreases circulating amino acid levels, and that with addition of amino acid infusion, there is decreased insulin-mediated glucose uptake and decreased insulin-mediated suppression of hepatic glucose output. Thus, amino acids cause a form of insulin resistance. Following acute protein ingestion in normal persons, however, there is little change in glucose homeostasis, presumably reflecting increases in insulin and in glucagon levels. As far as chronic effects of isocaloric high- or low-protein diets administered for 5 weeks in persons with diabetes, there is evidence of a decrease in glucose and triglyceride levels with a high-protein diet, as well as a trend to weight loss. The energy cost of fat ingestion and storage is low, and that for carbohydrate is modest, but there is a 25–30% thermic effect of protein ingestion, potentially contributing to decreasing calorie effect.

Daniel Bessesen (Denver, CO) discussed the relationship between dietary carbohydrate and insulin resistance, using data from animal studies, epidemiologic studies, and interventional studies. Studies analyzing development of diabetes may offer the most important information. There is evidence that carbohydrate intake has increased since the 1960s, particularly that of refined carbohydrate, and even more particularly of high-fructose corn syrup, showing temporal association with increasing prevalence of obesity and diabetes. One cannot conclude that the high fructose corn syrup is causative, as decreasing caloric expenditure has also occurred during this period. The difficulty of changing this trend is that palatability is a priority for consumers. Bessensen reviewed evidence of an association of fructose with insulin resistance, noting that fructose does not directly increase blood glucose levels but causes hepatic and later peripheral insulin resistance in rodent models, independent of weight gain, although this has not been demonstrated in humans. Ingested glucose shows ~30% hepatic extraction, while 50–70% of ingested fructose is extracted, leading to higher net carbohydrate delivery to the liver, which may be considered a hepatic "nutrient stress."

Starches exist in the branched form of amylopectin, which is rapidly digested and absorbed, and the relatively unbranched chains of glucose molecules of amylose, which are more slowly digested and absorbed, and Bessesen contrasted the related concepts of high-fiber foods versus "resistant starch," containing greater amounts of amylose. Increased fiber intake has been associated with reduction in development of diabetes among men (1) and women (2) and with greater insulin sensitivity, but one also must consider the glycemic impact of a starch based on its rapidity of digestion. This has led to the concept of the glycemic index of foods, and to the related measure of glycemic load of a food, obtained when the glycemic index is multiplied by the quantity of carbohydrate, with evidence that this is related to development of diabetes. Bessesen commented, however, that fat and fructose, as well as sugar alcohols, have a low glycemic index, so that this measure alone is not sufficient to distinguish foods contributing to diabetes from those with protective effects. Proponents of the measure note that it can be used effectively in modifying glycemia. In a meta-analysis of randomized controlled trials of low–glycemic index diets, with 14 studies of 356 persons with diabetes, HbA_{1c} decreased by 0.4% with the intervention, suggesting value in diabetes management (3). There is, however, no evidence that such diets result in improvement of insulin sensitivity, while a several-day period of high-carbohydrate diet actually improves glucose tolerance. Furthermore, lifestyle interventions lowering dietary fat (and hence increasing dietary carbohydrate) have been associated with reduction in development of diabetes, although one can argue that other components of the intervention may have played a role. Low-carbohydrate diets do produce weight loss, but there is limited data as to the effects of these diets on insulin action. In a 1-year analysis of obese persons with diabetes following a low-carbohydrate diet, glucose levels decreased, with trends to decrease in HbA_{1c} and insulin levels (4), which Bessensen interpreted to mean that "energy restriction trumps composition of the diet." If possible, he suggested a fat restricted diet, recognizing that some persons may more readily reduce energy intake with carbohydrate restriction, in which case this probably becomes the more desirable approach.

In a report at the meeting, Boden et al. (abstract 321) studied 10 obese type 2 diabetic patients strictly following the "Atkins Diet," with 21 g carbohydrate, 164 g fat, and 151 g protein, daily, compared with their customary diet of 309 g carbohydrate, 154 g fat, and 137 g protein, showing that over 2 weeks the daily calorie count decreased from 3,111 to 2,181 with attendant weight decrease from 114.8 to 112.4 kg. Urinary and plasma ketones increased, while there

was no change in total energy expenditure or body water. There was improved insulin sensitivity and glycemic control and reduced plasma cholesterol and triglyceride levels. Costacou et al. (abstract 187) estimated insulin sensitivity in 280 men and 268 women with type 1 diabetes, showing interesting evidence that insulin resistance was associated with lower dietary protein intake.

Jennifer Lovejoy (Kenmore, WA) discussed relationships between dietary fat and insulin resistance. Low-fat diets are beneficial in leading to weight loss. Astrup, in a meta-analysis of 14 randomized trials, showed that low-fat high-carbohydrate diets decreased daily energy intake by >1,100 kcal/day, leading to a 3.2-kg mean weight loss, with greater weight loss in persons who were heavier at baseline (5). In a meta-analysis of 37 National Cholesterol Education Program Step I and Step II dietary interventions, Yu-Poth showed that for every 1% decrease in dietary calories as total fat, there was a 0.3-kg decrease in body weight (6). Fat intake is also associated with the development of diabetes, with the seven-country study showing that both saturated and monounsaturated fat predict diabetes. In the Health Professionals' Follow-Up Study, higher fat intake, adjusted for age and activity, predicted diabetes, although adjustment for obesity eliminated the association, suggesting that weight gain is the mediator of the adverse effect of dietary fat. The EPIC-Norfolk (European Prospective Investigation into Cancer in Norfolk) study showed higher dietary fat to predict HbA_{1c} levels in normal populations (7). In the Insulin Resistance Atherosclerosis Study (IRAS), higher fat intake was associated with lower insulin sensitivity measured using IVGTT (8). Lovejoy's studies with IVGTT measurement of insulin sensitivity confirmed that low-fat high-carbohydrate diet was associated with greater insulin sensitivity, with similar results reported in euglycemic clamp studies. Insulin levels tend to be lower on high carbohydrate than on high fat diets after a several day period, so extrapolation from the lower insulin levels seen after single high fat meals appears not to be valid. In a study presented at the meeting shedding light on the complex relationship between dietary fat and lipids, Robitaille et al. (abstract 229) reported that there was no overall correlation between dietary fat and plasma apoprotein B, but that those persons with a A94 allele of the T94A polymorphism in the gene encoding liver fatty acid–binding protein were protected against a fat-related increase in apoprotein B, while persons homozygous for the T94 allele had almost a tripling of prevalence of apoprotein B above mean when consuming >30% of calories from fat (or >10% of calories from saturated fat), suggesting a potential diet-gene interaction.

Different fatty acids have varying effects on insulin resistance. In animal studies, muscle insulin sensitivity decreases with exposure to a variety of fatty acids, with ω-3 fatty acids having the least effect and monounsaturated fatty acids also having relatively favorable effects. Saturated fats appear to be associated with the greatest degree of worsening of insulin sensitivity. In her studies in lean adults, the monounsaturated fat oleic was compared with the saturated fat palmitic acid and with a *trans* fatty acid, showing no difference in insulin sensitivity, although there was a trend for heavier subjects to have adverse effect of saturated fats. In the Nurses Health Study, *trans* fatty acid intake correlated with diabetes risk, and this has been reported in a number of studies. In Lovejoy's studies of single meals enriched in *cis* or *trans* oleic acid, there was evidence of insulin resistance with the latter fat. A number of studies have analyzed effects of ω-3 fatty acids, with some inconsistency, suggesting that fish oils higher in DHA (docosahexaenoic acid), such as menhaden or salmon, have more beneficial effects on insulin sensitivity and greater glycemic effect among persons with diabetes than do fish oils high in eicosapentaenoic acid, such as tuna and mackerel. In a study of a 1.8-g DHA supplement daily in 11 overweight adults, however, Lovejoy found no evidence of consistent benefit, although in another study of 72 persons given a high-fat breakfast enriched in *trans*, ω-3, or ω-6 fatty acids, the ω-3 fatty acid supplement was associated with lesser increase in insulin and glucose levels, suggesting improvement in insulin sensitivity. In a report at the meeting, Skurnick-Minot et al. (abstract 189) randomized 14 women with type 2 diabetes to daily ingestion of 3 g fish oil containing 1.8 g of ω-3 polyunsaturated fatty acids or to placebo. Over two months, dual-energy X-ray absorptiometry–measured fat mass and abdominal fat decreased without change in body weight or energy intake, with a trend to increased adiponectin levels but no change in glycemia, lipids, or insulin sensitivity.

Cathy Nonas (New York, NY) argued against attempting to translate these studies of potential benefit of diets with specific macronutrient composition to clinical practice, noting the difficulty that individuals encounter in applying these concepts to actual diets, and emphasizing that achieving desirable weight is the most important goal of dietary intervention. The best approaches to weight loss, she stated, are caloric restriction and exercise, citing the Diabetes Prevention Program findings (9), with her view being that macronutrient composition is considerably less important than behavioral approaches such as portion control. In a study attempting to compare the effect of caloric restriction with that of weight loss, comparing an initial very low (400-calorie) diet followed by 1,000 calories in weight maintenance with a 1,000-calorie diet to achieve 11% weight loss, the former led to greater reduction in blood glucose, but with glucose levels increasing somewhat during weight maintenance, suggesting that there is particular benefit of caloric restriction and that the more strict the caloric restriction the greater the fall in glucose levels (and hence the need for persons with diabetes to reduce their hypoglycemic treatment) (10). "It doesn't seem to matter," Nonas summarized, "whether it's higher fat, whether it's lower 'carb,' whether it's lower protein, they all seem to have a very good effect where there's weight loss." Particularly with long-term studies "all diets look the same," with the goal being to assist patients to achieve a 5–7% body weight loss.

The glycemic index

At another symposium specifically addressing the glycemic index, Karen Teff (Philadelphia, PA) discussed the physiology of the glycemic index, which was originally proposed by Jenkins in 1981 as an easier approach than carbohydrate counting (11). In the treatment of persons with diabetes, with a goal to limit glycemic excursion, the effects of varying foods on glycemia indeed appears to be a reasonable measurement. Subsequent work in the field has suggested that in nondiabetic persons, foods with a high glycemic index may also mediate disease develop-

ment, perhaps because of consequent hyperinsulinemia that may mediate insulin resistance and changes in free fatty acids and triglycerides.

The glycemic index is defined as the incremental area above baseline from 0 to 120 min of a 50-g portion of a food, as compared with a standard food, typically white bread. Note that with a glucose-containing liquid, blood glucose initially increases to a greater degree, with subsequent lesser increases. "It's not always easy to predict" which foods will have high and which low glycemic index, with Teff giving as an example the higher glycemic index of potatoes than pasta. Another problem with the measure is that with a mixed nutrient meal containing 55% carbohydrate, 25% fat, and 20% protein, at 120 min the plasma glucose will not have returned to baseline, so the measure may underestimate total glycemic excursion. Liquids are absorbed quickly, resulting in high peak glucose responses, sometimes with a subsequent dip below baseline glucose levels, whereas solid foods require digestion, leading to blunting of the plasma glucose response. Different macronutrients are emptied at different rates, with gastric emptying of fat the slowest, so that adding fat to carbohydrate decreases gastric emptying. Protein stimulates insulin secretion, potentially further adding to the complexity of predicting the effect of a given set of foods. Part of these effects are due to differences in the actions of different macronutrients on gut hormone responses, leading to variation in both gastric emptying and insulin secretion. Persons with diabetes may be relatively more sensitive to amino acid stimulation of insulin secretion, another potential complicating factor. Teff pointed out that insulin is not part of the calculation of the glycemic index and that high–glycemic index foods may cause adverse effects in persons without diabetes because of increases in insulin secretion, perhaps leading to a subsequent decrease in glucose levels promoting counterregulatory responses, increase in food intake, and free fatty acid release–stimulated sympathetic activation, all potentially contributing to insulin resistance. Although it has not been demonstrated that the <10-mg/dl dip in glucose seen with typical mixed meals stimulates hypoglycemia, meals containing greater amounts of high–glycemic index foods, particularly in liquid form, may lead to greater falls in glucose levels.

Addressing the effects of high–glycemic index foods on food intake and weight gain, Teff noted that the evidence for hyperinsulinemia promoting food intake includes animal studies, with acute insulin administration promoting hypoglycemia and hence food intake, and prolonged elevation of basal insulin to nonphysiologic levels also increasing food intake. Furthermore, there is extensive clinical evidence that exogenous insulin promotes weight gain. In the central nervous system, however, insulin actually decreases food intake, acting with leptin to decrease food intake via effects on brain neurochemical responses. In the periphery, insulin directly promotes adipocyte leptin secretion. Thus, the hypothesis that high–glycemic index foods have adverse consequence because of an insulin stimulatory effect has not been proven. Furthermore, in Pima Indian studies, insulin release following meals and following oral glucose, as well as the acute (10-min) insulin response to intravenous glucose, are inversely related to food intake. Studies of diets enriched with high–glycemic index foods have not actually been shown to induce insulin resistance, although the effects of previous meals and studies of appropriate at-risk populations may not have been carried out. Teff addressed the question of why epidemiologic studies appear to show this relationship and pointed out that consumption of soda, sweetened with high-fructose corn syrup, has increased over the past 30 years. Comparing beverages sweetened with either glucose or fructose, the glycemic index is higher with glucose, with consequent hyperinsulinemia, but high-fructose meals result in more marked elevation in triglyceride levels.

Angela Liese (Columbia, SC) discussed methodological issues in the study of glycemic index, further stressing the need to "dispel the notion of simplicity" of this measure. The effect of a food of given glycemic index has three components, the glycemic index of the food, the number of servings ingested per day, and the quantity of carbohydrates ingested per serving, factors that can be summed as the glycemic load. In the epidemiologic setting, both glycemic index and glycemic load have been used in attempts to assess the effects of carbohydrates on diabetes and its complications. Food frequency questionnaires are used to assess dietary intake in large populations, measures that have been validated to show moderate correlation with actual carbohydrate intake, although it is important to realize that in many studies foods of rather differing glycemic index may be grouped together and that glycemic index databases are not well standardized. Reference to the "international tables of glycemic index" of Foster-Powell, Holt, and Brand-Miller (12), however, shows that there is moderate variability between published glycemic index values for a number of foods, further adding to variability (as is the case with most foods, which Brand-Miller noted after the talk). Food frequency questionnaires usually cannot address factors such as food processing, cooking, ripeness, storage, food variety, and effects of mixed meals, and this further decreases the ability to assess actual dietary intake. Furthermore, food frequency questionnaires have not been validated for glycemic index, which might have an effect on the accuracy of the measurement.

Liese noted that it is equally important to question how the glycemic index and glycemic load fit into the hierarchy of dietary patterns, food groups, and nutrients, referring to the concept of "food synergy," i.e., that foods and food patterns act synergistically to influence the risk of chronic diseases. An example given in the development of this concept is whole-grain consumption. The benefit of whole-grain foods is related to phytochemicals that are located in the fiber matrix, in addition to the fiber itself, as well as to consumption of a diet otherwise high in plant foods (13). Is glycemic index, Liese asked, a marker of lifestyle or an etiologic agent? If the latter, calories, physical activity, smoking, exercise and other associated factors should be eliminated from analysis, but if consumption of low–glycemic index foods is important as part of an overall lifestyle, then eliminating the associated factors would be counterproductive. The question can be rephrased as: Which correlates of glycemic index are confounders, and which are effect modifiers? Liese referred to her IRAS investigations, which revealed that the glycemic index is associated with ethnicity, age, sex, education, physical activity, and cigarette smoking. From a conceptual standpoint, there is no need to adjust the glycemic index for caloric intake, but caloric intake is strongly associated with

glycemic load, and different approaches to adjusting for caloric intake have different impacts on interpretation of the data.

Reviewing correlations of glycemic index, glycemic load, and IRAS nutritional data, the glycemic index does in fact show a modest association with calories, while there is the expected strong association of glycemic load with calories and carbohydrates and an inverse association of glycemic index with fiber. Interestingly, there was a strong correlation of glycemic index and glycemic load with fat, with Liese suggesting that this is due to typical dietary patterns, with high–glycemic index foods often eaten in conjunction with high-fat foods. Rather than conceptualizing the glycemic index as being like a nutrient, Liese concluded, it may be better to view it as a marker of the dietary pattern of different populations having different risks of developing diabetes, cardiovascular disease, and other conditions.

David Ludwig (Boston, MA) discussed the relationship between the glycemic index and obesity, noting that since the 1970s average caloric intake has increased by 150–300 calories/day, with little increase in fat intake but with a 65-g/day increase in carbohydrate intake, exclusively in the form of refined carbohydrates and concentrated sugars. Mixed meals with identical macronutrients show that hunger is suppressed to a greater extent by low–glycemic index foods, both in acute studies of single meals and in studies carried out over longer times. Blood glucose levels are, he stated, higher after high–glycemic index meals, and there is consistent subsequent mild hypoglycemia, with glucose levels falling ~10 mg/dl below baseline and free fatty acids suppressed to a greater extent, reflecting high insulin and suppressed glucagon levels. There may be biological significance of these changes, as epinephrine levels increase with high–glycemic index meals. After high–glycemic index meals, ad libitum food intake is greater than after low–and medium–glycemic index meals, with additional factors such as palatability potentially also having an effect on this finding. Addressing the chronic effects of high–glycemic index foods, Ludwig noted that increased hunger and hyperinsulinemia may occur, causing weight gain. Although insulin administration to the central nervous system has been shown to decrease food intake, peripheral insulin may have differing effects, and in the setting of insulin resistance, there also may be resistance to the central insulin effect of reducing food intake in the setting of hyperinsulinemia. Ludwig illustrated this point with the EURODIAB study of persons with type 1 diabetes, which suggests an independent association of glycemic index with waist circumference (14). Ludwig reviewed several intervention studies in which low–glycemic index diets reduced body fat and weight in persons with and without diabetes. In his initial studies of a low–glycemic index diet, BMI decreased 1.5 kg/m^2, while showing no change with a low-fat diet. Subsequent studies from his group have continued to show this effect, with dietary fat not associated with weight loss but a decrease in glycemic index appearing to be an important predictor in multivariate analysis. He noted that this may reflect confounding by other dietary factors. Animal studies of high –versus low–glycemic index diets suggest greater energy efficiency of the former, suggesting that lower intake of high–glycemic index foods is required to maintain weight. Visceral fat levels also increase in animal studies using a high–glycemic index diet, suggesting an independent adverse effect. Thus, low–glycemic index diets may prevent weight gain, decrease postprandial hyperglycemia, and decrease a late postprandial free fatty acid surge seen following ingestion of high–glycemic index foods, with all of these factors potentially decreasing the development of diabetes. Jenkins showed evidence of worsening islet histology from the animal studies, further suggesting potential diabetogenic effects of high–glycemic index foods. When asked whether low-carbohydrate Atkins-type diets function in a sense similarly to low–glycemic index diets, Ludwig replied that "it's not just carbohydrate reduction, although there may be synergistic mechanisms," suggesting that "the low–glycemic index diet is the perfect integrator" of the Atkins diet and the traditional diet.

David Grotto (Evanston, IL) discussed approaches to the current epidemic of obesity and suggested that it is important to convey a "healthy eating message" that includes the concept of low glycemic index. He pointed out that the food guide pyramid, which had been created a number of years ago by the U.S. Department of Agriculture to encourage eating patterns, is being revised to include concepts of nutrient density and may be relevant to the different factors that affect glycemic response to meals (see http://www.health.gov/dietaryguidelines/dga2005/report for the 2005 Dietary Guidelines Advisory Committee Report). Fiber content, particularly that of viscous fiber, amylose-to-amylopectin ratio, acidity, ripeness, duration of storage and of cooking, and other nutrient content of meals will have effects on blood glucose response to foods. He reviewed the large number of dietary choices that can be used to reduce glycemic index, including whole grains, fresh fruits and vegetables, beans, legumes, and nuts, and pointed out that it is not necessary to exclusively eat low–glycemic index foods to obtain benefit from this approach.

Bariatric surgery

At a symposium addressing psychological and dietary aspects of bariatric surgery, which is now attracting widespread usage in the U.S., Christian Lemmon (Augusta, GA) discussed patient selection criteria based on psychological evaluation to address factors determining patients' readiness for surgery and their postoperative outcomes. Surgery is currently recommended for patients with morbid obesity (BMI >40 kg/m^2) and those with lesser degrees of obesity (BMI 35–40 kg/m^2) but with serious underlying medical problems, although Lemmon noted that persons >50 years of age tend to have worse outcome. He reviewed the "stages of change" model of preparation, precontemplation, and contemplation; most patients are ready for surgery in the latter stage of psychological preparedness. Diets that patients have already followed and current food intake are reviewed, particularly assessing maladaptive eating behaviors, including ingestion of high-calorie soft drinks and binge eating and whether the subject engages in purging. Patients are assessed for depression, anxiety, poor awareness of sensitivity to stimuli originating inside of the body, and other general psychological dysfunction. Patients who attempt to outmaneuver the gastric banding or bypass procedures often have such underlying psychological conditions and may require follow-up surgery because of these self-manipulations. Some 20% of bariatric surgery candidates fulfill criteria for bulimia nervosa and, as such, are not good candidates for the pro-

cedure. "A lot of these patients," Lemmon concluded, "come in looking for a quick fix, and they're not interested in looking at all these other factors that contribute to this." Management of depression, for example, is an important factor to be addressed before the surgical procedure.

Nutritional management in the pre- and postoperative period were reviewed by Judy Dowd (Springfield, MA). Preoperative nutrition interventions are important to prepare patients for "the big changes to come." Before the surgery, it is necessary for the patient to be medically and psychologically stable and for the patient to "take responsibility for their vast number of calories," a process aided by interactions with other patients. Reiterating Lemmon's comments of the underlying psychopathology of many persons with severe obesity, she noted that one "does not want to perform surgery on someone who does not mind vomiting." Her group also insists that patients discontinue cigarette use 2 months before surgery and assesses patients for sleep apnea and initiates treatment for this before surgery in an effort to optimize the patient's medical readiness for surgery. It is necessary for patients to have documented failure to lose weight with medically supervised diets during the 1–2 years before performing the surgery. The 6- to 12-month preoperative period allows adequate time for patients to participate in support group meetings. Postoperative weight loss results from reduced ingested food volume capacity of the pouch created with the Roux-en-Y gastric bypass, vertical gastric bypass, vertical gastric banding, and laparoscopic banding procedures. The pouch increases modestly in size over the first postoperative year, but even by this time it takes ~1 h for a patient to consume just one sandwich, creating an effective barrier to overeating behaviors in persons with preoperative binging. Furthermore, there may be hormonal changes caused by the procedure, resulting in decreased appetite, with some evidence of a fall in ghrelin levels following bariatric surgery.

During the initial 2 weeks after surgery, patients need to restrict intake to liquid or puréed foods. Malabsorption of calories from the bypassed small intestine may be associated with malabsorption of iron, calcium, and B-vitamins, so that vitamin and mineral supplements are recommended (usually best taken as chewable tablets). More than 60% of the weight loss caused by the surgery is seen during the first postoperative year; therefore, compliance with a number of dietary strategies is required. Meal timing, assessing volume, and pacing are crucial, making slow ingestion of small meals every 3–4 h and adequate fluid intake important. Anticipation of the feeling of fullness while eating is an important component of the postoperative learning period to avoid vomiting. Consumption of high-fiber foods also contributes to optimal outcome of the surgery and helps to prevent postoperative constipation. Patients who have had bariatric surgery are instructed to regularly consume only sugar-free beverages or diluted fruit juices. Assuring adequate protein intake, of at least 60–80 g daily, is important, particularly in the postoperative period, with dairy and soy proteins useful for persons who have difficulty ingesting meat after the procedure. Muscle loss may accompany rapid weight loss, further increasing the importance of protein ingestion. Avoidance of high-sugar foods, particularly liquids and concentrated sweets, helps to prevent postoperative dumping syndrome, which may include vomiting, diarrhea, and symptoms of hypotension. Weight regain after surgery can be prevented by regular exercise, at least 30 min daily, ideally including weight training, with her group recommending that if an individual cannot participate in exercise, then they should not be considered good candidates for surgery. Participation in a postoperative support group is important to address the behavioral changes required, with Dowd noting that "structure will help [patients] ground behaviors." The support group helps patients to learn to deal with the loss of enjoyment of rapid food ingestion.

Psychological and educational issues were further discussed by John Guare (Indianapolis, IN), who stressed the need for patients to make lifelong changes and to cope with those changes. Patients need to understand that surgery is "just a tool" that allows the patient to accomplish the behavioral changes leading to weight loss. He recommended that patients have a variety of educational materials and referred the audience to the Internet site Obesityhealth.com as a resource, but noted that it is possible to "overwhelm" patients with too much information, stressing the need to formally assess whether patients actually understand all the information provided. "Just because things have been explained clearly does not mean that patients have actually heard what was said," he noted, recalling a patient whose main question after a long discussion was "How long will I have to wait to eat fudge?" Patients often have unrealistic expectations for benefits of bariatric surgery on body weight and on medical and even socioeconomic problems.

Guare discussed approaches to performing surgery in patients with psychiatric illnesses. Binge eating is reduced by the surgery, because of the development of fullness, vomiting, and dumping syndrome symptoms, so that binge eating disorder patients may experience great initial weight loss, although these patients are at risk for excessive subsequent weight regain. Depression also tends to improve after weight loss and therefore should not be regarded as a contraindication unless it is severe enough that it must be initially addressed. Anxiety also tends to improve. Personality disorders, in contrast, which consist of pathological trends in personality structure, accompanied by little anxiety and no personal sense of distress, tend not to change following weight loss.

Social interactions and relationships tend to improve following the surgery, although Guare noted "if you do not have a healthy marriage it is likely to deteriorate." Body image is an important preoperative concern and improves significantly with weight loss, although not all patients are happy with their postoperative body image, some feeling that they do not recognize themselves after weight loss. He reviewed additional psychological issues which may be encountered. Occasional persons purposefully gain weight to meet the BMI criteria for surgery, which may exacerbate health problems and will be likely to require particularly great reduction in food intake after the procedure, worsening the patient's difficulty. Some patients report having gained weight initially as a tactic to avoid sexual abuse, and there may be major feelings of vulnerability and issues of sexuality following weight loss. Anorexia nervosa–like behaviors may be seen following weight loss, with fear of weight gain. Patients experience grieving for the loss of food, and have difficulty managing cravings for favorite "comfort foods," so that it is necessary to "normalize the experience of cravings" and teach cognitive

and behavioral skills to prevent relapse and to engage the patient in the process of long-term weight control. Finally, Guare pointed out that health care professionals themselves need to make certain that they are not biased against obese persons.

Bruce Wolfe (Carmichael, CA) discussed the outcomes of obesity surgery and the assessment of which procedures are effective. The Roux-en-Y gastric bypass can either be performed with open surgery or with a laparoscopic approach, creating a 15- to 20-ml gastric pouch and diverting bile and pancreatic secretions to the jejunum. A modification is the "duodenal switch" with more distal diversion of bile and pancreatic secretions to delay the rate of nutrient absorption and possibly reduce dumping syndrome. Finally, the laparoscopic banding procedure, which places an adjustable band at the proximal stomach, is increasingly being used to reduce gastric capacity. Wolfe noted that the mechanism of action of weight loss with these operations is "complex and not understood fully," with malabsorption and changes in learned behaviors both appearing to be components and with neural and endocrine signals affecting satiety also appearing to be involved. The response to surgery is related to the amount of weight lost. In a study of long-term response to surgery, there is rapid weight loss through 12 months, with reduction in the weight of weight loss through 18 months and subsequent modest weight regain. A Canadian study showed that 67% of excess weight is lost and maintained with bariatric surgery, with a mortality rate of 0.7% in the bariatric surgery cohort compared with 6.17% in a matched group of obese persons who did not have surgery (15), and although Wolfe recognized that such an analysis is intrinsically flawed, he stated that a randomized prospective trial would not be possible. He noted that 15–20% of persons undergoing bariatric surgery have diabetes and that an additional 10–20% have some abnormality of glucose tolerance. Wolfe reviewed a number of studies of persons with diabetes, with long-term follow-up of 479 patients following surgery, 101 of whom had type 2 diabetes and 62 impaired glucose tolerance, showing only 22 having abnormal glucose tolerance at 10-year follow-up (16). Persons with longer duration of diabetes and receiving higher insulin doses are least likely to show resolution of diabetes, although improvement is often seen. The resolution of diabetes has been reported to be seen in approximately two-thirds of patients with laparoscopic banding, suggesting the lesser weight loss with this procedure to lead to somewhat less glycemic improvement (17). Weight loss leads to resolution of diabetes in 80–85% of patients undergoing Roux-en-Y gastric bypass, so Wolfe suggested that early bariatric surgery intervention should be considered in view of the finding that the frequency of resolution decreases with increasing duration of diabetes, although he noted that diabetes is a risk factor for adverse surgical outcome.

Studies to more clearly delineate the risks versus the benefits of bariatric surgery in persons with diabetes of longer duration, particularly those over age 50 and those with complications, appear not yet to have been performed, perhaps limiting our ability to recommend this approach for many patients.

References

1. Fung TT, Hu FB, Pereira MA, Liu S, Stampfer MJ, Colditz GA, Willett WC: Whole-grain intake and the risk of type 2 diabetes: a prospective study in men. *Am J Clin Nutr* 76:535–540, 2002
2. Schulze MB, Liu S, Rimm EB, Manson JE, Willett WC, Hu FB: Glycemic index, glycemic load, and dietary fiber intake and incidence of type 2 diabetes in younger and middle-aged women. *Am J Clin Nutr* 80:348–356, 2004
3. Brand-Miller J, Hayne S, Petocz P, Colagiuri S: Low-glycemic index diets in the management of diabetes: a meta-analysis of randomized controlled trials. *Diabetes Care* 26:2261–2267, 2003
4. Stern L, Iqbal N, Seshadri P, Chicano KL, Daily DA, McGrory J, Williams M, Gracely EJ, Samaha FF: The effects of low-carbohydrate versus conventional weight loss diets in severely obese adults: one-year follow-up of a randomized trial. *Ann Intern Med* 140:778–785, 2004
5. Astrup A, Grunwald GK, Melanson EL, Saris WH, Hill JO: The role of low-fat diets in body weight control: a meta-analysis of ad libitum dietary intervention studies. *Int J Obes Relat Metab Disord* 24: 1545–1552, 2000
6. Yu-Poth S, Zhao G, Etherton T, Naglak M, Jonnalagadda S, Kris-Etherton PM: Effects of the National Cholesterol Education Program's Step I and Step II dietary intervention programs on cardiovascular disease risk factors: a meta-analysis. *Am J Clin Nutr* 69:632–646, 1999
7. Harding AH, Sargeant LA, Welch A, Oakes S, Luben RN, Bingham S, Day NE, Khaw KT, Wareham NJ: Fat consumption and HbA_{1c} levels: the EPIC-Norfolk study. *Diabetes Care* 24:1911–1916, 2001
8. Mayer-Davis EJ, Monaco JH, Hoen HM, Carmichael S, Vitolins MZ, Rewers MJ, Haffner SM, Ayad MF, Bergman RN, Karter AJ: Dietary fat and insulin sensitivity in a triethnic population: the role of obesity: the Insulin Resistance Atherosclerosis Study (IRAS). *Am J Clin Nutr* 65: 79–87, 1997
9. Knowler WC, Barrett-Connor E, Fowler SE, Hamman RF, Lachin JM, Walker EA, Nathan DM, Diabetes Prevention Program Research Group: Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. *N Engl J Med* 346:393–403, 2002
10. Wing RR, Marcus MD, Salata R, Epstein LH, Miaskiewicz S, Blair EH: Effects of a very-low-calorie diet on long-term glycemic control in obese type 2 diabetic subjects. *Arch Intern Med* 151:1334–1340, 1991
11. Jenkins DJ, Wolever TM, Taylor RH, Barker H, Fielden H, Baldwin JM, Bowling AC, Newman HC, Jenkins AL, Goff DV: Glycemic index of foods: a physiological basis for carbohydrate exchange. *Am J Clin Nutr* 34:362–366, 1981
12. Foster-Powell K, Holt SH, Brand-Miller JC: International table of glycemic index and glycemic load values: 2002. *Am J Clin Nutr* 76:5–56, 2002
13. Jacobs DR Jr, Steffen LM: Nutrients, foods, and dietary patterns as exposures in research: a framework for food synergy. *Am J Clin Nutr* 78:508S–513S, 2003
14. Toeller M, Buyken AE, Heitkamp G, Cathelineau G, Ferriss B, Michel G, EURODIAB IDDM Complications Study Group: Nutrient intakes as predictors of body weight in European people with type 1 diabetes. *Int J Obes Relat Metab Disord* 25:1815–1822, 2001
15. Christou NV, Sampalis JS, Liberman M, Look D, Auger S, McLean AP, MacLean LD: Surgery decreases long-term mortality, morbidity, and health care use in morbidly obese patients. *Ann Surg* 240:416–424, 2004
16. Pories WJ, MacDonald KG Jr, Morgan EJ, Sinha MK, Dohm GL, Swanson MS, Barakat HA, Khazanie PG, Leggett-Frazier N, Long SD: Surgical treatment of obesity and its effect on diabetes: 10-y follow-up. *Am J Clin Nutr* 55:582S–585S, 1992
17. Pontiroli AE, Pizzocri P, Librenti MC, Vedani P, Marchi M, Cucchi E, Orena C, Paganelli M, Giacomelli M, Ferla G, Folli F: Laparoscopic adjustable gastric banding for the treatment of morbid (grade 3) obesity and its metabolic complications: a three-year study. *J Clin Endocrinol Metab* 87:3555–3561, 2002