Diabetes Treatment, Part 1: Diet and Exercise

Michael J. Fowler, MD

Editor's note: *This article is the third in a 12-part series reviewing the fundamentals of diabetes care for physicians in training. Previous articles in the series can be viewed at the* Clinical Diabetes *website (http://clinical.diabetesjournals.org).*

A cornerstone of diabetes treatment is attention to lifestyle. Unhealthy lifestyles, such as lack of physical activity and excessive eating, initiate and propagate the majority of type 2 diabetes. As discussed in previous articles in this series,[1,2] the incidence and prevalence of obesity is rising quickly, both in the United States and in the rest of the world. The frequency of diabetes has risen in tandem with overweight and obesity in essentially all age-groups and ethnicities in the United States, and not by coincidence. Studies have thoroughly demonstrated strong relationships between excess weight and the risk of developing type 2 diabetes, hypertension, and hyperlipidemia. Physicians are frequently challenged with the task of motivating patients to lose weight and exercise to improve patients' diabetes control and slow or even reverse the natural course of the disease.

Lifestyle modification, although different, is an equally integral part of type 1 diabetes management. Patients with type 1 diabetes, because of their universal need for insulin, must learn to count or at least closely estimate the amount of carbohydrate they consume to help regulate their blood glucose levels and adjust their insulin doses. Failure to do so can lead to dangerous hyperglycemia or hypoglycemia.

Primary Prevention of Diabetes

It is difficult to overstate the importance of the relationship between lifestyle and the risk of developing type 2 diabetes. A recent study demonstrated that both women and men who have a BMI > 35 kg/m^2 had a 20-fold increase in their risk of developing diabetes compared to people with a BMI of 18.5–24.9 kg/m^2.[3] Furthermore, prospective studies have demonstrated that lifestyle modification in the form of diet and regular moderate exercise sharply decreases the likelihood of developing type 2 diabetes in high-risk individuals who have impaired glucose tolerance or impaired fasting glucose. The effectiveness of this intervention superseded that of metformin therapy.[4] It is crucial, therefore, to properly educate obese patients and patients with glucose intolerance or impaired fasting glucose about the significance of exercise and weight loss in preventing diabetes, especially because many patients may make the presumption that medical therapy is the more important approach.

Control of Existing Diabetes

Lifestyle interventions are not just beneficial before the development of diabetes. Several studies have clearly demonstrated the benefit of a healthful diet, regular exercise, and weight loss in individuals already diagnosed with diabetes. Substantial dietary restriction to 1,100 kcal/day has been shown to decrease fasting blood glucose of obese patients with diabetes and even in those without diabetes in as few as 4 days. This improvement was likely the result of decreased hepatic glucose output. After 28 days of calorie restriction, there was further decline in the fasting glucose levels of obese diabetic subjects, and insulin sensitivity was significantly improved. It is also noteworthy that improvement in insulin sensitivity correlated well with decrease in fasting glucose and insulin sensitivity.[5] These results occurred with an average weight loss of only 6 kg. These studies did not show an improvement in insulin secretory capacity.[5,6]

Obese people also have a high incidence of hypertension and hyperlipidemia compared to nonobese people, which may further increase their risk of microvascular and macrovascular complications of diabetes.[2] Weight loss also has been shown to decrease systolic and diastolic blood pressure[7] and LDL cholesterol and lipid levels[8] in obese diabetic patients, albeit less dramatically than it affects glucose. Ongoing trials are studying the ability of intensive lifestyle interventions to decrease the rate of cardiovascular disease events in type 2 diabetes.[9]

Dietary Considerations

Carbohydrate

People with type 1 diabetes, because they experience absolute insulin deficiency, must use insulin to control glucose excursions after meals. Since 1994, the American Diabetes Association (ADA) has recommended that, for patients with type 1 diabetes, 60–70% of total calories come from carbohydrate and monounsaturated fat. Although some studies have considered whether

a preponderance of calories from unsaturated fat or carbohydrate may be more beneficial, there is no consensus on the relative amount of each. There are demonstrated improvements, however, from adjusting the doses of prandial rapid- or short-acting insulin based on the carbohydrate content of meals for patients using basal-bolus insulin regimens involving multiple daily injections or continuous subcutaneous insulin infusion. Similarly, patients on fixed doses of rapid- or short-acting insulin should attempt to keep the amount of carbohydrate relatively constant from meal to meal.[10,11]

Recommendations for carbohydrate consumption for people with type 2 diabetes are similar to those for patients with type 1 diabetes. Carbohydrate and monounsaturated fat should comprise 60–70% of total calories. However, there is some concern that increased unsaturated fat consumption may promote weight gain in obese patients with type 2 diabetes and thereby decrease insulin sensitivity.[12] Glycemic excursions appear to be similar between starches and sucrose ("table sugar"); therefore, sucrose does not need to be eliminated from the diet.[13]

The "glycemic index" is an attempt to compare the glycemic effects of various foods to a standard, such as white bread. Although several authors have proposed its clinical usefulness in controlling postprandial hyperglycemia, prospective studies have not demonstrated a clear improvement in hemoglobin A_{1c} (A1C) in patients using low–glycemic-index diets.[12] One cross-sectional study[14] suggested a relationship between low–glycemic-index diets and low A1C levels in patients with type 1 diabetes, but it is important to note that this study did not control for patients using once-daily, twice-daily, or more intensive insulin therapy regimens to control their glucose excursions.[14] Another more recent meta-analysis of low–glycemic-index diets[15,16] did suggest a mild but significant improvement in A1C levels. Therefore, there may exist a small benefit in pursuing a low–glycemic-index diet in patients with diabetes. This benefit, however, appears to be less than the benefit of either matching insulin doses to carbohydrate consumed or controlling carbohydrate consumed when using fixed insulin doses.

Many sweeteners are available to the general public; perhaps the most common is sucrose. Studies comparing the impact of sucrose versus the impact of the same amount of starch on glycemic control have shown that their impact is essentially identical. As described above, sucrose should be adequately covered by rapid- or short-acting prandial insulin but does not need to be eliminated from the diet. Fructose may cause less postprandial hyperglycemia, but there is some evidence suggesting that it may also lead to or worsen hyperlipidemia. Therefore, the addition of fructose to the diet as a sweetening agent is not recommended by the ADA; foods that contain naturally occurring fructose, such as fruits, do not need to be avoided.[12,15]

The Federal Drug Administration (FDA) has approved several sugar alcohols for use as sweeteners. These include products such as sorbitol, a common sweetener in chewing gum. Sugar alcohols cause less hyperglycemia than naturally occurring sugars and may also decrease the risk of dental caries. They are only partially absorbed from the intestinal tract and therefore may lead to diarrhea or gastrointestinal discomfort, especially if consumed in higher amounts.[17] They provide approximately half the calories of natural sugars and should be included in carbohydrate counting at half the impact of sucrose. Despite a lower risk of cavities, they have not been shown to facilitate weight loss or improve glycemic control.[12,15]

Several nonnutritive sweeteners are also available and do not affect blood glucose levels. These include aspartame, sucralose, saccharin, neotame, and acesulfame potassium. Although at one time linked to carcinogenesis in laboratory animals at extremely high doses, saccharin is no longer considered a cancer-causing chemical by the FDA.[12] One of the most recently released sweeteners, sucralose, has been shown to have no significant effect on blood glucose levels and therefore may be omitted from carbohydrate calculations.[12,15,18] These sweeteners have not been shown to facilitate weight loss or improve glycemic control.

Patients should exercise caution whenever introducing artificial sweeteners into the diet or decreasing their carbohydrate consumption. Making these changes without adjustment in diabetes medications could cause hypoglycemia, especially in patients using insulin or insulin secretagogues.

Protein

Although the majority of clinical focus on the management of diabetes is on carbohydrate metabolism, protein metabolism in the state of diabetes is also abnormal. Patients with type 2 diabetes exhibit a more negative nitrogen balance than individuals without diabetes. Protein degradation appears to be exacerbated by hyperglycemia and improved by controlling glucose levels with insulin therapy.[19–21] These studies suggest that the protein requirements for people with type 2 diabetes may be slightly greater than those for nondiabetic individuals, but as pointed out by Franz et al.,[12] most individuals in the United States consume considerably more protein than the recommended daily allowance. Patients with type 1 diabetes can and do convert amino acids into glucose depending on the level of insulinization; therefore, protein consumption may cause hyperglycemia.[12]

Studies of patients with type 2 diabetes, however, have demonstrated that protein consumption does not increase plasma glucose concentrations and that endogenous insulin release is, in fact, stimulated by protein consumption.[22]

There may also be an association between high-protein diets and the risk

of developing diabetic nephropathy. In a cross-sectional study of patients with type 1 diabetes,[23] patients with macroalbuminuria were more likely than those with microalbuminuria or normal albumin excretion to report consuming > 20% of their calories in the form of protein. High-protein diets are not recommended.

Dietary Fat

Recommendations regarding fat in the diet of people with diabetes are similar to those for patients with coronary artery disease. This is primarily because studies have shown that the risk of myocardial infarction in diabetic patients is similar to that of nondiabetic patients who have already suffered a myocardial infarction.[24] Because saturated fats are the major dietary determinants of serum LDL cholesterol levels, people with diabetes should strive to keep saturated fat consumption to < 7% of total daily calories and to minimize consumption of trans-fatty acids. Cholesterol consumption should be < 200 mg/day.[15]

When incorporated into a controlled-calorie diet in which individuals are not losing weight, programs that emphasize either carbohydrate or monounsaturated fats both lower cholesterol, but the higher-carbohydrate diets may exacerbate hyperglycemia. In diets in which total calories were reduced to facilitate weight loss, however, the hyperglycemic effect of the high-carbohydrate diet appeared mitigated.[15] Mediterranean-style diets, which are high in polyunsaturated fats, have been associated with lower mortality in elderly Europeans, but this study was not specific to people with diabetes.[25] Diets high in fish oil may decrease the risk of cardiovascular disease and all-cause mortality.[26]

Plant sterols are plant esters that decrease intestinal absorption of both dietary and hepatobiliary cholesterol. They have been shown in prospective studies of diabetic patients to decrease LDL cholesterol. To avoid unnecessary weight gain, the ADA recommends that, if they are used in the diet to decrease cholesterol, they should replace cholesterol sources rather than simply be added.[15,27]

There has been a great deal of interest in using micronutrients such as chromium, zinc, antioxidants, and herbal supplements to improve diabetes control. Although some small studies have suggested a benefit from chromium, other studies and meta-analysis have not reached the same conclusion. Currently, there are no large convincing studies that prove benefit of micronutrients in the management of diabetes.[15]

Considerable attention and marketing has been focused on the macronutrient content of diets. A recent study suggested that a diet low in carbohydrate and high in fat and protein may yield greater weight loss than other diets in nondiabetic patients.[28] Similar diets studied in diabetic patients have also suggested that a low-carbohydrate diet may produce similar or superior weight loss than balanced diets. Changes in triglycerides may be more favorable in low-carbohydrate diets, and A1C levels may be lower in low-carbohydrate diets.[29,30] Meta-analysis of several studies, however, suggested that low-carbohydrate diets may raise LDL levels.[15,31] It is important to note that the existing studies of low-carbohydrate diets are short-term studies and that the long-term effects of such diets is unknown. This is especially concerning because of their widespread use and the association of diabetic kidney disease with diets consisting of > 20% of calories from protein. For these reasons, a low-carbohydrate diet (< 130 g of total carbohydrate per day) is not recommended by the ADA.[15]

Exercise

Patients with type 1 or type 2 diabetes have an increased risk of coronary artery disease. The ADA recommends that patients who plan to begin a moderate- to high-intensity exercise program undergo screening for cardiovascular disease if they are > 35 years of age. Patients who are > 25 years of age should also be screened if they have had type 2 diabetes for > 10 years or type 1 diabetes for > 15 years, have an additional risk factor for coronary disease, or have microvascular disease, peripheral vascular disease, or autonomic neuropathy. Decisions regarding screening of patients who plan low levels of physical activity, such as walking, are left to the discretion of the treating physician. Because some activities can lead to retinal hemorrhage or detached retina in the setting of proliferative retinopathy, patients with this condition should consult their ophthalmologist before beginning an exercise regimen.[32]

People with type 1 diabetes who begin an exercise regimen should tailor their exercise regimen to their specific condition. For instance, a patient with peripheral neuropathy must take precautions to avoid blisters and abrasions and check closely for such conditions after exercising. Patients should consider delaying exercise if their blood glucose is > 250 mg/dl and ketones are present or if their blood glucose level is > 300 mg/dl. They should monitor blood glucose before and after physical activity and be cautious about hypoglycemia, which can develop during or even several hours after exercise. They should have carbohydrate sources available and consume them as necessary to avoid hypoglycemia. Although studies have not demonstrated a clear benefit of aerobic exercise on A1C levels in type 1 diabetes, aerobic exercise is clearly beneficial in controlling other risk factors for cardiovascular disease.[32]

Physical exercise is a key component of lifestyle modification that can help individuals prevent or control type 2 diabetes. Although diet is probably more important in the initial phases of weight loss, incorporating exercise as part of a weight-loss regimen helps maintain weight loss and prevent weight regain.[33] Mild to moderate activity levels have been associated with a lower risk of developing diabetes or pre-diabetes. Men with low degrees of cardiorespiratory

fitness may have up to a 1.9-fold increased risk of developing impaired fasting glucose compared to men with high degrees of fitness.[34] Patients should understand that the amount of exercise that produces a beneficial effect on health is not large; as little as 30 minutes of moderate physical activity daily may offer protection from diabetes.[33,35] As with the lowering of A1C levels, there is a gradient of benefit with higher levels of exercise and activity. Greater levels of physical activity are associated with lower risks of developing diabetes in women compared with lesser levels of activity.[36] These studies indicate that exercise should be a mainstay of primary prevention of diabetes.

In patients with type 2 diabetes, structured regimens of physical activity for 8 weeks or longer improved A1C independent of changes in body mass. There may also be further improvement in A1C with increasing intensity of exercise.[37,38] Exercise in type 2 diabetes has not been associated with peripheral neuropathy or worsening of nonproliferative retinopathy. Physical activity may cause transient increases in urinary albumin excretion, but exercise has not been shown to increase the rate of progression of diabetic kidney disease. Resistance exercises may be incorporated into a weekly exercise regimen in the absence of contraindications.[37] As further evidence regarding the benefit of exercise, in men with diabetes, the degree of physical fitness correlated with overall mortality, and this association was independent of BMI.[33,39]

When physicians are confronted with newly presenting patients with diabetes or glucose intolerance/impaired fasting glucose, one of the most vexing questions posed is to what extent to rely on exercise, weight loss, and dietary measures to control the disease. Evidence supports the contention that controlling blood glucose through modification of diet and lifestyle should be a mainstay of diabetes therapy. With many oral and injectable pharmaceutical agents to help patients control their glucose levels, it is easy for a practitioner to overlook or forget to emphasize and reinforce the importance of lifestyle modification on the treatment of diabetes. Despite being one of the most time-consuming discussions to have with patients, this is probably the most important patient-physician discussion in regard to diabetes control and prevention of disease progression and complications.

REFERENCES

[1]Harris MI, Flegal KM, Cowie CC, Eberhardt MS, Goldstein DE, Little RR, Wiedmeyer HM, Byrd-Holt DD: Prevalence of diabetes, impaired fasting glucose, and impaired glucose tolerance in U.S. adults: the Third National Health and Nutrition Examination Survey, 1988–1994. *Diabetes Care* 21:518–524, 1998

[2]Mokdad AH, Ford ES, Bowman BA, Dietz WH, Vinicor F, Bales VS, Marks JS: Prevalence of obesity, diabetes, and obesity-related health risk factors, 2001. *JAMA* 289:76–79, 2003

[3]Field AE, Coakley EH, Must A, Spadano JL, Laird N, Dietz WH, Rimm E, Colditz GA: Impact of overweight on the risk of developing common chronic diseases during a 10-year period. *Arch Intern Med* 161:1581–1586, 2001

[4]Knowler WC, Barrett-Connor E, Fowler SE, Hamman RF, Lachin JM, Walker EA, Nathan DM: Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. *N Engl J Med* 346:393–403, 2002

[5]Markovic TP, Jenkins AB, Campbell LV, Furler SM, Kraegen EW, Chisholm DJ: The determinants of glycemic responses to diet restriction and weight loss in obesity and NIDDM. *Diabetes Care* 21:687–694, 1998

[6]Maggio CA, Pi-Sunyer FX: The prevention and treatment of obesity: application to type 2 diabetes. *Diabetes Care* 20:1744–1766, 1997

[7]Amatruda JM, Richeson JF, Welle SL, Brodows RG, Lockwood DH: The safety and efficacy of a controlled low-energy ('very-low-calorie') diet in the treatment of non-insulin-dependent diabetes and obesity. *Arch Intern Med* 148:873–877, 1988

[8]Anderson JW, Brinkman-Kaplan V, Hamilton CC, Logan JE, Collins RW, Gustafson NJ: Food-containing hypocaloric diets are as effective as liquid-supplement diets for obese individuals with NIDDM. *Diabetes Care* 17:602–604, 1994

[9]Espeland M: Reduction in weight and cardiovascular disease risk factors in individuals with type 2 diabetes: one-year results of the Look AHEAD trial. *Diabetes Care* 30:1374–1383, 2007

[10]Rabasa-Lhoret R, Garon J, Langelier H, Poisson D, Chiasson JL: Effects of meal carbohydrate content on insulin requirements in type 1 diabetic patients treated intensively with the basal-bolus (ultralente-regular) insulin regimen. *Diabetes Care* 22:667–673, 1999

[11]Hamet P, Abarca G, Lopez D, Hamet M, Bourque M, Peyronnard JM, Charron L, Larochelle P: Patient self-management of continuous subcutaneous insulin infusion. *Diabetes Care* 5:485–491, 1982

[12]Franz MJ, Bantle JP, Beebe CA, Brunzell JD, Chiasson JL, Garg A, Holzmeister LA, Hoogwerf B, Mayer-Davis E, Mooradian AD, Purnell JQ, Wheeler M: Nutrition principles and recommendations in diabetes. *Diabetes Care* 27 (Suppl. 1): S36–S46, 2004

[13]Bantle JP, Swanson JE, Thomas W, Laine DC: Metabolic effects of dietary sucrose in type II diabetic subjects. *Diabetes Care* 16:1301–1305, 1993

[14]Buyken AE, Toeller M, Heitkamp G, Karamanos B, Rottiers R, Muggeo M, Fuller JH: Glycemic index in the diet of European outpatients with type 1 diabetes: relations to glycated hemoglobin and serum lipids. *Am J Clin Nutr* 73:574–581, 2001

[15]American Diabetes Association: Nutrition recommendations and interventions for diabetes [Position Statement]. *Diabetes Care* 30 (Suppl. 1): S48–S65, 2007

[16]Brand-Miller JC, Petocz P, Colagiuri S: Meta-analysis of low-glycemic index diets in the management of diabetes: response to Franz. *Diabetes Care* 26:3363–3364; author reply 3364–3365, 2003

[17]Payne ML, Craig WJ, Williams AC: Sorbitol is a possible risk factor for diarrhea in young children. *J Am Diet Assoc* 97:532–534, 1997

[18]Mezitis NH, Maggio CA, Koch P, Quddoos A, Allison DB, Pi-Sunyer FX: Glycemic effect of a single high oral dose of the novel sweetener sucralose in patients with diabetes. *Diabetes Care* 19:1004–1005, 1996

[19]Gougeon R, Marliss EB, Jones PJ, Pencharz PB, Morais JA: Effect of exogenous insulin on protein metabolism with differing nonprotein energy intakes in type 2 diabetes mellitus. *Int J Obes Relat Metab Disord* 22:250–261, 1998

[20]Gougeon R, Styhler K, Morais JA, Jones PJ, Marliss EB: Effects of oral hypoglycemic agents and diet on protein metabolism in type 2 diabetes. *Diabetes Care* 23:1–8, 2000

[21]Gougeon R, Pencharz PB, Sigal RJ: Effect of glycemic control on the kinetics of whole-body protein metabolism in obese subjects with non-insulin-dependent diabetes mellitus during iso- and hypoenergetic feeding. *Am J Clin Nutr* 65:861–870, 1997

[22]Gannon MC, Nuttall JA, Damberg G, Gupta V, Nuttall FQ: Effect of protein ingestion on the glucose appearance rate in people with type 2 diabetes. *J Clin Endocrinol Metab* 86:1040–1047, 2001

[23]Toeller M, Buyken A, Heitkamp G, Bramswig S, Mann J, Milne R, Gries FA, Keen H: Protein intake and urinary albumin excretion rates in the EURODIAB IDDM Complications Study. *Diabetologia* 40:1219–1226, 1997

[24]Haffner SM, Lehto S, Ronnemaa T, Pyorala K, Laakso M: Mortality from coronary heart disease in subjects with type 2 diabetes and in nondiabetic subjects with and without prior myocardial infarction. *N Engl J Med* 339:229–234, 1998

[25]Trichopoulou A, Psaltopoulou T, Orfanos P, Hsieh CC, Trichopoulos D: Low-carbohydrate-high-protein diet and long-term survival in a general population cohort. *Eur J Clin Nutr* 61:575–581, 2007

[26]Wang C, Harris WS, Chung M, Lichtenstein AH, Balk EM, Kupelnick B, Jordan HS, Lau J: n-3 Fatty acids from fish or fish-oil supplements, but not alpha-linolenic acid, benefit cardiovascular disease outcomes in primary- and secondary-prevention studies: a systematic review. *Am J Clin Nutr* 84:5–17, 2006

[27]Lee YM, Haastert B, Scherbaum W, Hauner H: A phytosterol-enriched spread improves the lipid profile of subjects with type 2 diabetes mellitus: a randomized controlled trial under free-living conditions. *Eur J Nutr* 42:111–117, 2003

[28]Gardner CD, Kiazand A, Alhassan S, Kim S, Stafford RS, Balise RR, Kraemer HC, King AC: Comparison of the Atkins, Zone, Ornish, and LEARN diets for change in weight and related risk factors among overweight premenopausal women: the A TO Z Weight Loss Study: a randomized trial. *JAMA* 297:969–977, 2007

[29]Foster GD, Wyatt HR, Hill JO, McGuckin BG, Brill C, Mohammed BS, Szapary PO, Rader DJ, Edman JS, Klein S: A randomized trial of a low-carbohydrate diet for obesity. *N Engl J Med* 348:2082–2090, 2003

[30]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

[31]Nordmann AJ, Nordmann A, Briel M, Keller U, Yancy WS Jr, Brehm BJ, Bucher HC: Effects of low-carbohydrate vs low-fat diets on weight loss and cardiovascular risk factors: a meta-analysis of randomized controlled trials. *Arch Intern Med* 166:285–293, 2006

[32]Zinman B, Ruderman N, Campaigne BN, Devlin JT, Schneider SH: Physical activity/exercise and diabetes. *Diabetes Care* 27 (Suppl. 1):S58–S62, 2004

[33]Klein S, Sheard NF, Pi-Sunyer X, Daly A, Wylie-Rosett J, Kulkarni K, Clark NG: Weight management through lifestyle modification for the prevention and management of type 2 diabetes: rationale and strategies: a statement of the American Diabetes Association, the North American Association for the Study of Obesity, and the American Society for Clinical Nutrition. *Diabetes Care* 27:2067–2073, 2004

[34]Wei M, Gibbons LW, Mitchell TL, Kampert JB, Lee CD, Blair SN: The association between cardiorespiratory fitness and impaired fasting glucose and type 2 diabetes mellitus in men. *Ann Intern Med* 130:89–96, 1999

[35]Bassuk SS, Manson JE: Epidemiological evidence for the role of physical activity in reducing risk of type 2 diabetes and cardiovascular disease. *J Appl Physiol* 99:1193–1204, 2005

[36]Hu FB, Sigal RJ, Rich-Edwards JW, Colditz GA, Solomon CG, Willett WC, Speizer FE, Manson JE: Walking compared with vigorous physical activity and risk of type 2 diabetes in women: a prospective study. *JAMA* 282:1433–1439, 1999

[37]Sigal RJ, Kenny GP, Wasserman DH, Castaneda-Sceppa C, White RD: Physical activity/exercise and type 2 diabetes: a consensus statement from the American Diabetes Association. *Diabetes Care* 29:1433–1438, 2006

[38]Boule NG, Haddad E, Kenny GP, Wells GA, Sigal RJ: Effects of exercise on glycemic control and body mass in type 2 diabetes mellitus: a meta-analysis of controlled clinical trials. *JAMA* 286:1218–1227, 2001

[39]Church TS, Cheng YJ, Earnest CP, Barlow CE, Gibbons LW, Priest EL, Blair SN: Exercise capacity and body composition as predictors of mortality among men with diabetes. *Diabetes Care* 27:83–88, 2004

Michael J. Fowler, MD, is an assistant professor of medicine in the Division of Diabetes, Endocrinology, and Metabolism, Vanderbilt Eskind Diabetes Clinic, at Vanderbilt University Medical Center in Nashville, Tenn. He is an associate editor of Clinical Diabetes.