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Nutrition in Diabetes Mellitus

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Introduction

Diabetes mellitus is a common metabolic disorder.[1,2] The hallmark of diabetes is fasting and/or post-prandial hyperglycemia. Hyperglycemia results from insulin deficiency or interference with its action (insulin resistance) or both. Uncontrolled diabetes leads to widespread metabolic derangement. Sixteen million people in the United States have diabetes mellitus. The prevalence of diabetes is increasing at an alarming rate in all age groups, from children to the elderly. The disorder is progressively more common with advancing age. Fifty percent or more of the population after 80 to 90 years of age has glucose intolerance or diabetes. It is the sixth leading cause of death due to disease in the U.S. and decreases the average life expectancy up to 15 years when compared to the population without diabetes. Diabetes has an enormous social impact, mostly due to its chronic microvascular (retinopathy, nephropathy, and neuropathy) and macrovascular complications. Diabetic retinopathy is the leading cause of blindness in the U.S. In people with diabetes, age 20 to 74 years, there are 12,000 to 24,000 new cases of blindness each year. Diabetic nephropathy is responsible for a large number of the patients on renal dialysis and undergoing renal transplantation. In 1996 more than 130,000 people with diabetes underwent either dialysis or kidney transplantation. Diabetic neuropathy is present in 60 to 70% of all patients with diabetes. Besides increasing the risk for sudden death and silent myocardial infarction, diabetic neuropathy leads to impotence, which is experienced by 50% of men with diabetes. Diabetic neuropathy together with peripheral vascular disease is responsible for more than 200,000 cases of foot ulcers and 80,000 limb amputations each year. Finally, diabetes is also responsible for macrovascular complications including peripheral vascular disease, stroke, and cardiovascular disease. Cardiovascular disease is the leading cause of death in diabetes (80% of all patients with diabetes die of cardiovascular disease). Seventy five percent of the cardiovascular mortality in diabetes is from coronary heart disease and 25% is from cerebral or peripheral vascular disease. Coronary heart disease, peripheral vascular disease, and stroke account for nearly one million hospital admissions each year among patients with diabetes. In women, diabetes carries yet another burden since it may lead to problems during pregnancy — mainly congenital malformations in babies born to diabetic mothers. The rate of major

congenital malformations is 10% compared to 2% in non-diabetic women, and fetal mortality occurs in 3 to 5% of pregnancies.

Classification and Diagnostic Criteria of the Several Subtypes of Diabetes and Intermediate Syndromes

Classification of Diabetes Mellitus

Type 1 Diabetes

Type 1 diabetes is characterized by pancreatic β-cell destruction, usually leading to absolute insulin deficiency.[3,4] Its etiology is likely due to a combination of genetic and environment factors. Most type 1 diabetes is an organ-specific autoimmune disease characterized by T-cell mediated autoimmune destruction of the pancreatic β-cells. In a few cases there is no evidence of an autoimmune process, and these cases are classified as idiopathic.

Type 2 Diabetes

Type 2 diabetes is characterized by insulin resistance and an insulin secretory defect.[3,4] It is the most prevalent type of diabetes, and comprises 90% of the population with diabetes. As with type 1 diabetes, type 2 has both a genetic and an environment component. Type 2 diabetes may range from cases with predominantly insulin resistance and relative insulin deficiency to cases with a predominantly secretory defect and some degree of insulin resistance.

Other Specific Types of Diabetes

Genetic defects in β-cell function or in insulin action as well as several diseases of the exocrine pancreas, endocrinopathies, infections, drugs or chemicals, uncommon forms of immune-mediated diabetes, and other genetic syndromes that may lead to hyperglycemia are included under the definition of other specific types of diabetes. A comprehensive list is included in Table 53.1.

Gestational Diabetes (GDM)

Gestational diabetes is defined by any degree of glucose intolerance that is initially recognized during pregnancy. The type of treatment required to manage glucose intolerance in pregnancy and whether or not the glucose intolerance continues after pregnancy does not affect the diagnosis. Six or more weeks after pregnancy it is necessary to reclassify the patient and determine whether diabetes, impaired glucose tolerance, impaired fasting glucose, or normoglycemia is present. About 4% of all pregnancies are complicated by gestational diabetes, and the diagnosis is commonly made during the third trimester.

At present it is not recommended that all women be screened for gestational diabetes. Screening should, however, be performed in women if they a) are more than 25 years of age, b) are overweight, c) have family history of diabetes, or d) are Hispanic, Asian, Afro-American, or native American. The first test to be performed is a screening test using a 50 g load of glucose. If the screening test is positive, a 100 g diagnostic loading test needs to be performed.

TABLE 53.1

Etiologic Classification of Diabetes Mellitus

I. Type I diabetes* (β-cell destruction, usually leading to absolute insulin deficiency)
   A. Immune mediated
   B. Idiopathic
II. Type II diabetes* (may range from predominantly insulin resistance with relative insulin deficiency to a predominantly secretory defect)
III. Other specific types
   A. Genetic defects of β-cell function
      1. Chromosome 12, HNF-1a (MODY3)
      2. Chromosome 7, glucokinase (MODY2)
      3. Chromosome 20, HNF-4a (MODY1)
      4. Mitochondrial DNA
      5. Others
   B. Genetic defects in insulin action
      1. Type A insulin resistance
      2. Leprechaunism
      3. Rabson — Mendenhall syndrome
      4. Lipoatrophic diabetes
      5. Others
   C. Diseases of the exocrine pancreas
      1. Pancreatitis
      2. Trauma/pancreatectomy
      3. Neoplasia
      4. Cystic fibrosis
      5. Hemochromatosis
      6. Fibrocalculous pancreatopathy
      7. Others
   D. Endocrinopathies
      1. Acromegaly
      2. Cushing's syndrome
      3. Glucagonoma
      4. Pheochromocytoma
      5. Hyperthyroidism
      6. Somatostatinoma
      7. Aldosteronoma
      8. Others
   E. Drug- or chemical-induced
      1. Vacor
      2. Pentamidine
      3. Nicotinic acid
      4. Glucocorticoids
      5. Thyroid hormone
      6. Diazoxide
      7. β-adrenergic agonists
      8. Thiazides
      9. Dilantin
      10. α-interferon
      11. Others
   F. Infections
      1. Congenital rubella
      2. Cytomegalovirus
      3. Others
   G. Uncommon forms of immune-mediated diabetes
      1. "Stiff-man" syndrome
      2. Anti-insulin receptor antibodies
      3. Others
   H. Other genetic syndromes sometimes associated with diabetes
      1. Down's syndrome

TABLE 53.1 *(Continued)*

Etiologic Classification of Diabetes Mellitus

2. Klinefelter's syndrome
3. Turner's syndrome
4. Wolfram's syndrome
5. Friedreich's ataxia
6. Huntington's chorea
7. Laurence-Moon-Biedl syndrome
8. Myotonic dystrophy
9. Porphyria
10. Prader-Willi syndrome
11. Others
IV. Gestational diabetes mellitus (GDM)

* Patients with any form of diabetes may require insulin treatment at some stage of their disease. Such use of insulin does not, of itself, classify the patient.

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Impaired Glucose Tolerance and Impaired Fasting Glucose

These two syndromes are usually associated with an intermediate metabolic stage between normal glucose metabolism and diabetes. Impaired glucose tolerance is associated with levels of plasma glucose ≥140 mg/dl but <200 mg/dl after an oral load of 75 g of glucose. Patients with glucose intolerance are often hyperglycemic if challenged with an oral glucose load, but in normal conditions they may have normal or near-normal plasma glucose levels and hemoglobin A1c. Impaired fasting glucose corresponds to fasting levels of plasma glucose ≥110 mg/dl but <126 mg/dl. Neither of these two syndromes is a disease per se, but they are considered risk factors for the development of macrovascular disease and diabetes. They are considered as risk factors for macrovascular disease because both syndromes can be associated with the insulin resistance syndrome, a metabolic syndrome previously named by Reaven as syndrome X.[5] This syndrome includes visceral obesity, insulin resistance, compensatory hyperinsulinemia, hypertriglyceridemia, low HDL-cholesterol, hypertension, and the presence of dense LDL. The two syndromes can also be observed in the disease processes listed in Table 53.1. Interestingly however, a recent study by Tominaga et al.[6] examining the cumulative survival rates from cardiovascular disease in a Japanese population of 2534 individuals with normal glucose tolerance, impaired glucose tolerance, impaired fasting glucose, or diabetes showed no significant difference between the survival rates from cardiovascular disease in subjects with normal glucose tolerance and subjects with impaired fasting glucose, but a significant decrease in survival in subjects with impaired glucose tolerance and diabetes. They therefore concluded that impaired glucose tolerance was a risk factor for cardiovascular disease, but impaired fasting glucose was not.

Diagnostic Criteria

The diagnostic criteria for diabetes, for type 1, type 2, and other specific types of diabetes, have been modified recently. In the past, the criteria used was that recommended by the National Diabetes Data Group[3] or World Health Organization (WHO);[1] the revised criteria are shown in Table 53.2. For epidemiological studies determining prevalence and/or incidence of diabetes, the first criterion (fasting plasma glucose >126 mg/dl) can be used, but it will lead to slightly lower estimates of prevalence/incidence than the combined use of the fasting plasma glucose and oral glucose tolerance test. That is clearly demonstrated by the data obtained in NHANES III[2] as summarized in Table 53.3. WHO criteria were

TABLE 53.2

Criteria for the Diagnosis of Diabetes Mellitus*

1. Symptoms of diabetes plus casual plasma glucose concentration ≥200 mg/dl (11.1 mmol/l). Casual is defined as any time of day without regard to time since last meal. The classic symptoms of diabetes include polyuria, polydipsia, and unexpected weight loss.

or

2. FPG ≥126 mg/dl (7.0 mmol/l). Fasting is defined as no caloric intake for at least 8 h.

or

3. 2-h PG ≥200 mg/dl (11.1 mmol/l) during an OGTT. The test should be performed as described by WHO (2), using a glucose load containing the equivalent of 75-g anhydrous glucose dissolved in water.

* In the absence of unequivocal hyperglycemia with acute metabolic decompensation, these criteria should be confirmed by repeat testing on a different day. The third measure (OGTT) is not recommended for routine clinical use.

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TABLE 53.3

Estimated Prevalence of Diabetes in the U.S. in Individuals 40 to 74 Years of Age using Data from the NHANES III

Diabetes Diagnostic Criteria	Prevalence (%) of Diabetes by Glucose Criteria without a Medical History of Diabetes*	Total Diabetes Prevalence (%)#
Medical history of diabetes	—	7.92
WHO (2) criteria for diabetes:		
FPG ≥140 mg/dl (7.8 mmol/l) or 2-h PG ≥200 mg/dl (11.1 mmol/l)	6.34	14.26
FPG ≥126 mg/dl (7.0 mmol/l)	4.35	12.27

* Diabetes prevalence (by glucose criteria) in those without a medical history of diabetes × (100%-prevalence of diabetes by medical history).

\# First column of data plus 7.92.

Data are from K. Flegal, National Center for Health Statistics, personal communication. Reprinted with authorization from American Diabetes Association (*Diabetes Care* 23(1): 11S; 2000).

based on the fact that the prevalence of retinopathy and nephropathy would rise markedly when the level of glucose 2 h after a standardized glucose load was >200 mg/dl. The revised criteria are based on results of several studies showing that fasting plasma level >126 mg/dl, like a 2-h post glucose load of >200 mg/dl, is associated with a marked rise in the prevalence of vascular complications.[4] In other words, levels of glucose ≥126 mg/dl reflect a serious metabolic disorder associated with the development of serious chronic diabetic complications.

Impaired fasting glucose is defined by glucose levels ≥110 mg/dl and <126 mg/dl after an eight-hour fast. Impaired glucose tolerance is defined by a level of glucose ≥140 mg/dl but <200 mg/dl two hours after a 75 g oral glucose load. The impaired glucose tolerance criteria will identify more people with impaired glucose homeostasis than the criteria of impaired fasting glucose.

Criteria for Screening for Diabetes

Screening for type 1 diabetes is not recommended. Type 1 diabetes is commonly an autoimmune process characterized by a variety of auto-antibodies against intracellular or

TABLE 53.4

Major Risk Factors for Diabetes Mellitus

Family history of diabetes (i.e., parents or siblings with diabetes)
Obesity (i.e., ≥20% over desired body weight or BMI ≥27 kg/m^2)
Race/ethnicity (i.e., African-Americans, Hispanic-Americans, Native Americans, Asian-Americans, Pacific Islanders)
Age ≥45 years
Previously identified IFG or IGT
Hypertension (≥140/90 mm Hg)
HDL cholesterol level ≥35 mg/dl (0.90 mmol/l) and/or a triglyceride level ≥250 mg/dl (2.82/l)
History of GDM or delivery of babies over 9 lb

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surface protein epitopes in the β-cell. The markers that may identify patients at risk before development of the disease are many. However, the levels of the markers that would permit the diagnosis of high-risk patients are not well established. Furthermore, the methodology is not easily accessible, and there is no consensus about what to do if high levels of auto-antibodies are observed. Nowadays, there is no effective and safe treatment to prevent the development of type 1 diabetes. A number of ongoing clinical trials testing various ways to prevent the development of type 1 diabetes are being conducted, and it is possible that in the near future, screening for patients at high risk for developing type 1 diabetes will be justifiable. At present the cost effectiveness and clinical relevance of such testing is questionable.

Screening for type 2 diabetes is, however, highly recommended. Undiagnosed type 2 diabetes is very common in the U.S. Approximately 50% of patients with type 2 diabetes are undiagnosed.[7] Some epidemiological studies have shown that retinopathy will start developing seven years prior to making the diagnosis of diabetes.[8] Even more worrisome is the fact that patients with undiagnosed diabetes are at significantly higher risk of developing premature macrovascular disease.[9] The risk of developing type 2 diabetes increases with age, obesity, and lack of physical activity. Furthermore, diabetes is more common in certain racial/ethnic groups (Hispanic, Asian, African-Americans, and native Americans), in women with gestational diabetes, and in individuals with a family history of diabetes, hypertension, or dyslipidemia. The major risk factors for developing diabetes are listed in Table 53.4.

The American Diabetes Association (ADA) recommends screening individuals who have one or more of the risk factors shown in Table 53.4 at three-year intervals. Fasting plasma glucose measurement or oral glucose tolerance test are adequate to perform screening for diabetes. Fasting, as mentioned before, represents a period of at least eight hours without food or beverage other than water. When an oral glucose tolerance test is performed, a load of 75 g of anhydrous glucose is considered the standard load for adult testing. Interpretation of the results is crucial, and should be made according to the criteria shown in Table 53.5. It is important to remember that certain drugs including furosemide, glucocorticosteroids, thiazides, estrogen-containing preparations, β-blockers, and nicotinic acid may induce hyperglycemia. In community screening tests it is sometimes impossible to use a fasting plasma glucose assay; therefore, a fasting capillary whole blood glucose is performed due to its convenience and simplicity of measurement. The levels are not, however, as accurate as those measured in plasma, and they are lower. If the measurement is made in capillary whole blood, individuals with blood glucose ≥110 mg/dl should be referred to a physician for further evaluation and testing. Criteria for diagnosis of gestational diabetes mellitus (GDM) is summarized in Table 53.6.

TABLE 53.5

Criteria for the Diagnosis of Diabetes Mellitus using Glucose Tolerance Results

Normoglycemia	Impaired Glucose Metabolism	DM*
FPG < 110 mg/dl 2-h PG† < 140 mg/dl	FPG ≥ 110 mg/dl and < 126 mg/dl 2-h PG† ≥ 140 mg/dl and < 200 mg/dl	FPG ≥ 126 mg/dl 2-h PG† ≥ 200 mg/dl Symptoms of DM and random plasma glucose concentration ≥ 200 mg/dl

* A diagnosis of diabetes must be confirmed on a subsequent day by measurement of FPG, 2-h PG or random plasma glucose (if symptoms are present). The FPG test is greatly preferred because of ease of administration, acceptability to patients, and lower cost. Fasting is defined as no caloric intake for at least 8 h.

† This test requires the use of a glucose load containing 75 g anhydrous glucose dissolved in water.

* DM, diabetes mellitus; 2-h PG, 2-h postload glucose.

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TABLE 53.6

Screening and Diagnosis Scheme for Gestational Diabetes Mellitus (GDM)*

Plasma Glucose	75-g Oral Glucose Load	100-g Oral Glucose Load
Fasting	95 mg/dl	95 mg/dl
1-h	180 mg/dl	180 mg/dl
2-h	155 mg/dl	155 mg/dl
3-h	not done	140 mg/dl

* The diagnosis of GDM requires any two or more plasma glucose values obtained during the test to meet or exceed the values shown above.

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Diabetic Complications: Microvascular

Retinopathy

Diabetic retinopathy is a specific microvascular complication present in both type 1 and type 2 diabetes, strongly correlated with the duration of diabetes. After 20 years of diabetes, nearly all patients with type 1 diabetes and more than 60% of the patients with type 2 diabetes will have some degree of retinopathy.[10]

Retinopathy can be defined as damage to the retina, a cell layer in the posterior part of the eye that contains the photoreceptors necessary for vision. It can be classified as mild, nonproliferative (also called background retinopathy), or moderate to severe non-proliferative retinopathy, characterized by hard exudates and retinal blot hemorrhages. This type of retinopathy advances to a preproliferative phase when retinal ischemia becomes more severe. Proliferative retinopathy is the most advanced stage of retinopathy, and is characterized by the growth of new blood vessels on the retina and posterior surface of the vitreous. Proliferative retinopathy usually leads to loss of vision due to retinal detachment, and is the leading cause of blindness in persons 30 to 65 years of age. Vision loss may also occur in patients without proliferative retinopathy when vascular leakage (macular edema) and/or occlusion occurs in the area of the macula. Maculopathy is more common in type 2 than type 1 diabetes and is an important cause for decreased visual acuity in this group of patients.

TABLE 53.7

Screening and Followup of Patients with Diabetes for Retinopathy

	Recommended Ophthalmologic Examination*	Recommended Minimum Followup
Type 1 diabetes > 10 years of age	Within 3-5 years after onset of disease	Yearly
Type 2 diabetes	At the time of diagnosis of diabetes	Yearly or more often if retinopathy is progressing
Diabetic patients during pregnancy	Prior to conception if programmed and during the 1st trimester	As often as necessary, according to physician
Patients with macular edema, severe proliferative retinopathy	Immediately after diagnosis of the condition	As often as necessary, according to physician

* The ophthalmologic exam recommended is a dilated and comprehensive exam by an ophthalmologist or optometrist.

Screening

Screening for the presence of retinopathy depends on the rates of progression of diabetic retinopathy and the risk factors that may alter these rates. Most of the available data is based on studies on Caucasian populations, and it is not certain whether these data can be applied to the ethnic groups with the highest incidence of diabetes and complications. The guidelines for screening and followup of patients with diabetic retinopathy are summarized in Table 53.7.

Influence of Glycemic Control and Treatment of Hypertension and Dyslipidemia

Data from the Diabetes Control and Complications Trial Research Group (DCCT) clearly show a definitive and direct relationship between glycemic control and diabetic microvascular complications, including retinopathy.[11] The DCCT shows that intensive insulin therapy for type 1 diabetes reduced or prevented the progression of diabetic retinopathy by 27% when compared with conventional therapy. Similar results were observed in type 2 diabetes as shown by the U.K. Prospective Diabetes Study Group (UKPDS).[12,13] The earlier that intensive control is started in the course of diabetes, the more effective it is in preventing the development of retinopathy. Besides poor glycemic control, proteinuria is also associated with retinopathy. Hypertension is an established risk factor for proteinuria. Thus, it is important to tightly control hypertension. Finally, maculopathy consists of edema and/or lipid exudates; since the lipid exudates observed in cases of maculopathy originate from circulating blood lipids, aggressive treatment of lipid abnormalities is also important in the prevention of retinopathy/maculopathy.

The main reason that screening for diabetic retinopathy is essential is the well known efficacy of laser photocoagulation therapy in patients with proliferative retinopathy and macular edema. The surgery, as well demonstrated in the Early Treatment Diabetic Retinopathy Study and the Diabetic Retinopathy Study, is extremely efficient in preventing loss of vision, but does not have much impact in reversing visual acuity if already diminished. Since proliferative retinopathy and macular edema are quite often asymptomatic, screening is crucial.

Neuropathy

Symptomatic and potentially disabling neuropathy affects nearly 50% of diabetic patients. Neuropathy can be symmetrical or focal, and often involves the autonomic nervous

TABLE 53.8

Classification of Diabetic Neuropathy

Diabetic Polyneuropathies	Diabetic Mononeuropathies
Distal symmetrical	Peripheral
Chronic sensorimotor	Cranial
Autonomic	Radiculopathy
Proximal motor	Isolated nerve lesions
Acute sensory	

TABLE 53.9

Symptoms and Signs of Diabetic Polyneuropathy

	Symptoms	Signs
Polyneuropathy	Pain and paresthesias most common at night	Diminished sensation to touch, temperature, pain, and vibration; loss of reflexes Atrophy of intrinsic hand muscles; sensory impairment

system. The prevalence of symmetrical neuropathy is similar in type 1 and type 2 diabetes, but the focal forms of neuropathy are more common in the older type 2 diabetic patient. The classification of neuropathy is made according to the areas affected due to the relatively poor understanding of the pathogenic mechanisms of this diabetic complication. Table 53.8 includes the most commonly accepted classification of diabetic neuropathic syndromes.

The cause for mononeuropathies is unknown, but they usually have a sudden onset, which suggests a vascular component in their pathogenesis. They usually tend to resolve with time, and although they occur in diabetes they are not the typical neuropathic lesions of diabetes. Diabetic polyneuropathies are the main problem for diabetic patients, and they will be discussed in some detail. To assess diabetic neuropathy, history of clinical symptoms and physical exam, electrodiagnostic studies, quantitative sensory testing, and autonomic function testing should be performed. Table 53.9 summarizes the clinical signs and symptoms of diabetic polyneuropathy, and Table 53.10 summarizes the functional changes associated with autonomic failure. In Table 53.11, adequate diagnostic testing to assess diabetic neuropathy is summarized.

TABLE 53.10

Functional Changes Associated with Autonomic Failure

Systems Involved	Manifestations
Cardiovascular	Resting tachycardia, impaired exercise-induced cardiovascular responses, cardiac denervation, orthostatic hypotension, heat intolerance, impaired vasodilatation, impaired venoarteriolar reflex (dependent edema)
Eye	Decreased diameter of dark-adapted pupil (dark-adapted miosis)
Gastrointestinal	Esophageal enteropathy, gallbladder atony, impaired colonic motility (diarrhea, constipation), anorectal sphincter dysfunction (incontinence)
Genitourinary	Neurogenic vesical dysfunction (decreased bladder sensitivity/incontinence/retention), sexual dysfunction, (male: penile erectile failure and retrograde ejaculation; female: defective lubrication)
Sudomotor	Anhidrosis/hyperhidrosis (heat intolerance), gustatory sweating
Endocrine	Hypoglycemia-associated autonomic failure

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TABLE 53.11

Electrodiagnostic Studies, Sensory Testing, and Autonomic Function Testing for the Diagnosis of Diabetic Neuropathy

Sensory Testing	Electrodiagnosis	Autonomic Function Testing
Vibration/touch thresholds	Motor and sensory nerve conduction studies	R-R variations, orthostasis, Valsalva
Thermal thresholds		Resting heart rate
Pain thresholds	Needle electromyography of extremity and paraspinal muscles	QTc, DAPS, NPT, CMG+BST
		REPs, QSART, TST
		Solid phase gastric motility
		Clamped hypoglycemia
		Clamped insulin infusion test

Abbreviations: QTc — corrected QT interval on EKG; DAPS — dark-adapted pupil size; NPT — nocturnal penile tumescence, CMG+BST — cystometrogram+Bethanechol supersensivity test; REPs — reflex-evoked potentials; QSART — quantitative sudomotor axon reflex test; TST — thermoregulated sweat test.

TABLE 53.12

Definition of Abnormalities in Albumin Excretion

	24-h Collection	Timed Collection	Spot Collection
Normal	<30 mg/24 h	<20µg/min	<30 mg/g creatinine
Microalbuminuria* (incipient nephropathy)	30-300 mg/24 h	20-200 µg/min	30-300 mg/g creatinine
Nephropathy*	>300 mg/24 h	>200 µg/min	>300 mg/g creatinine

* Two out of three urine specimens collected within a 3 to 6 month period should be abnormal before diagnosing a patient as having incipient nephropathy or nephropathy.

Influence of Glycemic Control

Data from the DCCT and UKPDS clearly show a definitive and direct relationship between glycemic control and diabetic microvascular complications, including neuropathy.[11-13] The DCCT data showed that intensive insulin therapy when compared with conventional therapy reduced or prevented the progression of diabetic neuropathy in patients with type 1 diabetes. The UKPDS showed the same results in patients with type 2 diabetes.

Nephropathy

Diabetic nephropathy is characterized by persistent albumin excretion exceeding 300 mg/24 h, a progressive decline in the glomerular filtration rate, and increased blood pressure.[14] The earliest clinical evidence of nephropathy is the increased excretion of albumin in the urine. This phase of incipient nephropathy is designated as microalbuminuria. The levels of albumin excretion in the microalbuminuria stage of nephropathy range from 30 to 300 mg/24 h. Table 53.12 summarizes the cutoff levels for diagnostic purposes as well as the correspondent values in spot urine collections. Measurement of creatinine and albumin excretion simultaneously in the same urine specimen is necessary when a spot urine is collected, and it is also recommended in timed specimens to ensure that a proper urine collection was obtained. Interpretation of microalbuminuria needs to take into consideration factors such as hyperglycemia, level of exercise preceding the urine collection, uncontrolled hypertension, urinary tract infections, acute febrile illnesses, and heart failure, since all of these conditions may lead to increased albuminuria. Diagnosis of nephropathy needs to be based on data from three urine specimens collected within a three- to six-month period. At least two of the specimens may be concordant to allow establishment of a valid diagnosis.

About 20 to 30% of patients with type 1 or type 2 diabetes develop nephropathy. A high percentage of subjects with type 2 diabetes are found to have microalbuminuria shortly

after their initial diagnosis. Two possible reasons are that in many cases diabetes has been present for many years and not diagnosed, and microalbuminuria in type 2 diabetes is less specific of diabetic nephropathy, as shown by renal biopsy studies.

Approximately 80% of individuals with type 1 diabetes who develop sustained microalbuminuria will progress to overt nephropathy over a period of 10 to 15 years. Once overt nephropathy occurs and if there is no therapeutic intervention, 50% of these patients will progress to end-stage renal disease in 10 years, and 75% in 20 years. The progression to overt nephropathy in type 2 diabetes, without therapeutic intervention, is less than in type 1 diabetes (approximately in 20 to 40% of the cases), and only approximately 20% will progress to end-stage renal disease. A marked racial/ethnic variability exists, however, as far as progression to end-stage renal disease in type 2 diabetes. Native Americans, Mexican-Americans and African-Americans have a much higher risk of developing end-stage renal disease than the other populations with type 2 diabetes. In the U.S., diabetic nephropathy is responsible for one third of all cases of end-stage renal disease, and that is a terrible burden in the country's economy. Regardless of the fact that subjects with type 1 diabetes are more prone to progress to end-stage renal disease, half of the patients with diabetes on dialysis have type 2 diabetes due to the higher prevalence of type 2 diabetes in the population.

Two major risk factors that can be easily intervened upon are involved in the progression of nephropathy: hypertension and hyperglycemia. The standards of care for hypertension and hyperglycemia in diabetes will be discussed later in this section.

Influence of Hypertension and Glycemic Control

In type 1 diabetes, hypertension is usually caused by the underlying diabetic nephropathy, and typically is detected at the time microalbuminuria becomes apparent. In type 2 diabetes, hypertension is present at the time diabetes is diagnosed in one third of the patients. The hypertension may be related to the underlying diabetic nephropathy, may be secondary to other diseases, or may be a coexisting disease, "essential hypertension." Commonly, subjects with type 2 diabetes, before being diagnosed as having diabetes, have been found to have an insulin resistance syndrome, which basically comprises a cluster of problems including hypertension, obesity, dyslipidemia, and glucose intolerance. The presence of both systolic and diastolic hypertension contributes to the accelerated development of diabetic nephropathy, and therefore treating hypertension aggressively in patients with diabetes is an essential step that cannot be overemphasized.

Hyperglycemia has been shown in recent trials to have a major impact in the development of microvascular complications, including nephropathy. Intensive treatment of diabetes to obtain near-normal glucose and hemoglobin A1c levels has significantly reduced the risk of development of microalbuminuria and overt nephropathy.[11-13]

Influence of Protein Restriction and Treatment of Lipid Disorders

It is well known that microalbuminuria is a marker for increased cardiovascular mortality and morbidity in patients with either type 1 or type 2 diabetes. In reality, microalbuminuria is considered as an indicator to screen patients for macrovascular complications (see Macrovascular Complications). Interestingly, some preliminary evidence also shows that lowering cholesterol leads to a reduction in the level of proteinuria. More work is needed to adequately validate this observation. Protein restriction has been shown to be of great benefit in animal studies to reduce progression of renal disease, including diabetic nephropathy. However, studies in humans are less clear. Several small studies seem to indicate that patients with overt nephropathy treated with a diet containing protein at 0.7 g/kg of body weight had mild retardation in the fall of the glomerular filtration rate. A recent

study of patients with renal disease in which 3% had type 2 diabetes failed to show any benefit of protein restriction. In reality, marked decrease of protein intake in patients with end-stage renal disease on dialysis showed that the main predictive factor of mortality was low albumin due to protein-energy malnutrition.

Diabetic Complications: Macrovascular Disease

General Considerations

Macrovascular disease, which includes coronary artery disease (CAD), cerebrovascular disease, and peripheral vascular disease, is the leading cause of mortality in people with diabetes. Individuals with diabetes have at least a two- to fourfold increased risk of cardiovascular events and stroke, and an eightfold increased likelihood of peripheral vascular disease compared with age-matched subjects without diabetes. The atherosclerotic process in diabetic patients is indistinguishable from that affecting the nondiabetic population, but begins earlier and is more severe. Most deaths in the diabetic population are due to complications of CAD. Although diabetic patients have a higher prevalence of traditional CAD risk factors (i.e., hypertension, dyslipidemia, obesity) compared to people without diabetes, these risk factors account for less than half the excess mortality associated with diabetes. Thus, the diagnosis of diabetes is a major independent risk factor for the development of CAD and adverse outcomes following a myocardial event. Other abnormalities induced by diabetes such as increased levels of small, dense atherogenic LDL, oxidized or glycated LDL, increased platelet aggregation, hyperviscosity, endothelial cell dysfunction, decreased fibrinolysis, and increased clotting factors and fibrinogen are likely responsible for accelerated atherosclerosis in diabetic patients.

Current Treatment and Prevention Strategies

Current treatment of macrovascular complications includes reduction of cardiovascular risk factors (obesity, smoking, sedentary lifestyle), with special emphasis on the treatment of hypertension and dyslipidemia. Diabetic patients with existing or incipient macrovascular disease in general require multiple modifications of lifestyle and diet, as well as a polypharmaceutical approach to address the optimization of lipid level, blood pressure, and other disease risk factors. Glycemic control seems also to contribute to a reduction in macrovascular events both in type 1 and type 2 diabetes, but its impact is much less marked than impact of treatment of cardiovascular risk factors such as dyslipidemia and hypertension.

Treatment of Hypertension

Hypertension accelerates not only atherosclerosis, but also nephropathy and probably retinopathy. Thus in diabetes, it is important to treat even minimal elevations of blood pressure that in nondiabetic patients might be dismissed. The normal nocturnal fall in blood pressure may be lost in diabetic patients, leading to a more sustained hypertension throughout the day. Initially, nonpharmacologic measures such as weight loss, exercise training, and sodium restriction should be implemented (see Section 48). If blood pressure is not lower than 130/85 mmHg, drug therapy is indicated. Among the various therapeutic options, ACE inhibitors offer special advantages, mainly when there is concomitant renal disease. Calcium channel blockers and vasodilators have no adverse metabolic effects, and therefore are good alternatives.

Treatment of Dyslipidemia

Dyslipidemia is common in subjects with diabetes. The more common lipid abnormalities in diabetes are increased triglycerides and low HDL cholesterol levels. Small, cholesterol-poor, dense LDL particles are also common in patients with hypertriglyceridemia and the insulin resistance syndrome. Dense LDL is more readily oxidized and more atherogenic. Aggressive treatment of dyslipoproteinemia is crucial to prevent the development and/or progression of macrovascular complications in diabetes.[15] Recommendations by the ADA concerning goals for therapy of hyperlipidemia as well as guidelines for medical nutrition therapy (MNT), physical activity, and drug treatment have been widely promulgated. Optimal triglyceride levels in diabetes are below 150 mg/dl, as in nondiabetic patients. This level was recommended by the National Cholesterol Education Program Adult Treatment Panel III (NCEP0-ATP III) on the recently released guidelines[15a] and it is lower than the 200 mg/dl previously recommended by both the NCEP-ATP II and the ADA. HDL-cholesterol levels above 45 mg/dl are the target in diabetes. According to ADA guidelines and the NCEP-ATP III guidelines, values below 40 µg/dl for men and above 50 mg/dl are used to define the metabolic syndrome. Levels of HDL-cholesterol below 40 mg/dl instead of 35 mg/dl are considered in the new NCEP guidelines a risk factor for CHD. The AFCAPS-Tex CAPS trial was instrumental in this change since it showed clearly that CHD risk decreases with levels of HDL-cholesterol above 40 mg/dl. Weight loss, increased physical activity, and good glycemic control are important measures to lower triglycerides and increase HDL-cholesterol levels. Increased LDL-cholesterol levels were not considered important in the treatment of diabetic dyslipidemia until recently. However, in the past decade it became apparent that lowering LDL-cholesterol levels in diabetes led to a significant reduction in the risk of a major congestive heart disease (CHD) event. That was clearly shown in several clinical trials including two major secondary prevention trials: Scandinavian Simvastatin Survival Study[16] and CARE trial.[17] The results of these trials were instrumental in the establishment of the guidelines to treat dyslipidemia in diabetes recently published by the ADA, and together with the results of several studies, among them the East West Study,[17a] were behind the change in the NCEP-ATP III guidelines that now considers diabetes as a CHD equivalent. Optimal LDL-cholesterol levels for adults with diabetes are <100 mg/dl, regardless of their risk factor profile or presence or absence of established cardiovascular disease.

Indications for Cardiac Testing

Asymptomatic CAD and silent myocardial infarction (MI) are frequent in subjects with diabetes. Thus, early diagnosis of CAD in patients with diabetes is very important, and allows earlier implementation of preventive programs aimed at reducing the risk of future coronary morbidity and mortality, initiation of treatment with anti-ischemic medications in silent ischemia, and earlier identification of patients in whom revascularization is appropriate. Indications for cardiac testing are summarized in Table 53.13.

Diabetic Complications: Hypoglycemia

It is well established that glycemic control will prevent specific long-term complications of diabetes. However, in order to prevent complications intensive treatment of diabetes is necessary, and unfortunately this may lead to hypoglycemia. It is obvious that even in optimal conditions, hypoglycemia is the limiting factor in the management of patients

TABLE 53.13

Indications for Cardiac Testing in Diabetic Patients

Testing for CAD is warranted in patients with the following:

1. Typical or atypical cardiac symptoms
2. Resting EKG suggestive of ischemia or infarction
3. Peripheral or carotid occlusive arterial disease
4. Sedentary lifestyle, age ≥ 35 years, and plans to begin a vigorous exercise program
5. Two or more of the risk factors listed below (a-e) in addition to diabetes
   a) Total cholesterol ≥ 240 mg/dl, LDL cholesterol ≥ 160 mg/dl, or HDL cholesterol < 35 mg/dl
   b) Blood pressure > 140/90 mm Hg
   c) Smoking
   d) Family history of premature CAD
   e) Positive micro/macroalbuminuria test

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with type 1 diabetes. Hypoglycemia is defined as a blood glucose of ≤60 mg/dl that may occur with or without symptoms.[18] During the course of the DCCT trial and even under optimal conditions, the incidence of severe hypoglycemia was more than three times higher in patients on intensive therapy when compared with patients treated with conventional therapy. The effects of hypoglycemia cannot be ignored, since they can be devastating, particularly on the brain. The first signs of hypoglycemia are shakiness, sweating, tachycardia, hunger, irritability, and dizziness. These symptoms are followed by inability to concentrate, confusion, slurred speech, irrational behavior, blurred vision, and extreme fatigue. Finally, the symptoms of severe hypoglycemia are seizures, unresponsiveness, and loss of consciousness. Symptoms of hypoglycemia may occur at any time, and therefore patients with diabetes should always be prepared to address them.

The level of glucose that leads to symptoms of hypoglycemia may vary from person to person and also varies in the same individual under different circumstances. Hypoglycemia is a much less frequent problem for people with type 2 diabetes except in the elderly, mainly when they have associated diseases that require the use of beta blockers. Hypoglycemia usually occurs gradually, and in general is associated with warning signs, including rapid heart beat, perspiration, shakiness, anxiety, and hunger. However, warning symptoms of hypoglycemia may be absent, causing the clinical syndrome of hypoglycemia unawareness. This syndrome results from excessive insulin in the setting of absent glucagon secretory responses to falling glucose levels. These episodes, in turn, cause reduced autonomic responses and lead to further decrease of the warning symptoms of hypoglycemia. This creates a vicious cycle that can only be broken by avoiding inducing iatrogenic hypoglycemia. The most common causes of hypoglycemia include: a) skipping, delaying, or reducing the size of the meals and snacks; b) increased physical activity without adequately adjusting therapy; c) alcohol intake mainly on an empty stomach; and d) treatment with excessively high levels of insulin or other antidiabetic medications. Hypoglycemia occurs mainly when the patient is being treated with insulin or sulphonylureas. In theory, biguanidines, thiazolidinediones, and α-glucosidase inhibitors would not be expected to induce hypoglycemia, since by themselves they will not increase the level of plasma insulin. However, it is conceivable that any intervention that limits hepatic glucose production, favors glucose utilization, or both may lead to hypoglycemia, since increased hepatic glucose production and limited glucose utilization are mechanisms of defense against a drop in plasma glucose levels. In the elderly, it is not uncommon to have hypoglycemia episodes, and these may be dangerous since these subjects often live alone. A recent study examining the risk of sulphonylurea-induced hypoglycemia in elderly type

2 diabetic patients concluded that therapy with sulphonylureas is well tolerated by the elderly, and that the primary mechanism of protection against hypoglycemia is an increase in epinephrine secretion.[19] That suggests that glucagon secretion in elderly patients is diminished, and supports the concept that treatment of these patients with sulphonylureas or insulin when they are also being treated with β-blockers may be dangerous, and close followup is needed. Oral antidiabetic agents other than sulphonylureas are probably better candidates for the treatment of these patients if their hyperglycemia is relatively modest. Although hypoglycemia during treatment with these agents may also occur, it is likely to be less frequently observed and less severe.

Standards of Medical Care for Diabetic Patients

Standards of medical care for diabetic patients have been markedly influenced by the results of recent major clinical trials. Some of the trials were specifically designed to address the importance of intensive glycemic control in subjects with type 1 or type 2 diabetes (DCCT and UKPDS). Some clinical trials, although not designed to specifically address questions related to diabetic patients, had a sufficiently large number of patients with type 2 diabetes and glucose intolerance to allow drawing conclusions on the effect of lipid-lowering therapy in the development of macrovascular complications (CARE, 4S, AF-CAPS/TEX-CAPS). The data published concerning these trials as well as the technical reviews of the ADA[20] will provide evidence for the standard-of-care measures proposed by the ADA for the treatment of patients with diabetes.

Standards of diabetes care are expected to provide health care providers taking care of patients with diabetes the means to establish treatment goals, assess the quality of the diabetes treatment provided, identify areas where more self management is needed, and define situations when referral to specialists is necessary. Also, the same standards of diabetes care should allow patients with diabetes to assess the quality of medical care that they receive, understand their role in the treatment of their disease, and compare their treatment outcomes with standard goals.

General Principles

It is accepted that lowering blood glucose levels to normal or near-normal levels will reduce:

- The danger of acute decompensation due to diabetic ketoacydosis or hyperosmolar hyperglycemic nonketotic syndrome
- The symptoms of blurred vision and symptoms/signs usually accompanying diabetes (polyuria, polydipsia, weight loss with polyphagia, fatigue) as well as vaginitis or balanitis
- The development or progression of diabetic retinopathy, nephropathy, and neuropathy
- Triglycerides leading to a less atherogenic lipid profile

It is also well accepted that lowering lipid levels will result in a decrease in diabetic macrovascular complications.

Thus, proper standards of diabetes care should include:

- Appropriate frequency of self monitoring of blood glucose
- Adequate medical nutrition therapy
- Regular exercise
- Adequate regimens with insulin and/or oral glucose-lowering agents
- Instructions in the prevention and treatment of hypoglycemia
- Instructions in the prevention and treatment of acute and chronic diabetes complications
- Adequate regimens of lipid-lowering therapy
- Continuing education and reinforcement programs
- Periodic assessment of treatment goals

Specific Goals for Management of Diabetes

An overview of the steps, goals, and treatment needed to obtain optimal care of patients with diabetes is summarized in Table 53.14.

Special Considirations

Pregnancy

To reduce the risk of fetal malformations and maternal and fetal complications, pregnant diabetic women require excellent glycemic control. Followup by a multidisciplinary team including a diabetologist, internist or family physician, obstetrician, and diabetes educator is essential. Other specialists need to be called upon if necessary. Self-management skills essential for glycemic control and preparation for pregnancy include:

- Designing an appropriate meal plan, with timing of meals and snacks and an appropriate physical activity plan
- Self-monitoring blood glucose levels, choosing the time and site for insulin injections, using therapy with glucagon or carbohydrate intake for treatment of hypoglycemia, and self-adjusting insulin dosages
- Reducing stress and coping with denial

Before conception, it is essential to have a good laboratory evaluation including HbA1c, baseline assessment of renal function, thyroid function tests, and lipid profile. Other tests may need to be added according to medical history and physical exam. Conception should be deferred until the initial evaluation is completed and specific goals of therapy, including glucose control and dietary and physical activity adherence, are attained. Since the safety of oral antidiabetic agents is not well established for the fetus, patients need to be switched to insulin therapy. The goals for blood glucose are 70 to 100 mg/dl preprandial and <140 or <120 mg/dl respectively one or two hours postprandial. Hypertension, retinopathy, nephropathy, gastroparesis, and other neuropathies as well as elevated lipid levels need to be stabilized prior to conception. Pregnancy will exacerbate and accelerate acute and long-term complications of diabetes. Continuing care by a team of professionals is essential in the management of pregnant diabetic patients.

Macrovascular Disease

Recommendations listed in Table 53.14 for LDL and HDL cholesterol levels as well as for triglycerides have a goal of reducing the risk for development of coronary heart disease

TABLE 53.14

Recommended Diabetes Management Guidelines

Parameters to Assess	Frequency of Evaluation	Goal	Action Indicated If:	Recommended Treatment
Assessment of Metabolic Control				
HbA1c	Quarterly	≤7%	≥8%	Diet, exercise, oral agents, and/or insulin
Self-monitoring of blood glucose:				
Preprandial	As necessary for glycemic control	80-120mg/dL	>140 mg/dL	Stepped adjustment of medication/diet to obtain adequate glycemic control
Bedtime		100-140 mg/dL	>160mg/dL	
Technique check	Annually	Proficient	Not proficient	Referral for teaching
Hypoglycemic episodes	Each visit	No episodes	Episodes occur	Change in lifestyle, diet, and/or drug treatment
Hyperglycemic episodes/ ketonuria	Each visit	No episodes	Episodes occur	Change in lifestyle, diet, and drug treatment
Assessment Macrovascular Complications				
Blood pressure	Each visit	≤ 130/80 mm Hg	> 130/80 mm Hg	ACE Inhibitors and off other antihypertensive medications
Lipid profile:	At least yearly. Quarterly or more frequently if levels are abnormal			Stepped approach to lipid control with lipid lowering medications, diet, and exercise Low dosage aspirin for patients with established macrovascular disease or patients with several risk factors for macrovascular disease
LDL-Cholesterol		< 100 mg/dL**	>100 mg/dL	
HDL-Cholesterol		>45 mg/dL	< 45 mg/dL	
Triglycerides		<150 mg/dL	> 150 mg/dL	
EKG	Annually	Normal	Abnormal	Stress test and/or referral to cardiology
Ankle/brachial ratio	Annually	Normal	Abnormal	Peripheral vascular assessment and/or referral to vascular surgery

TABLE 53.14 *(Continued)*

Recommended Diabetes Management Guidlines

Parameters to Assess	Frequency of Evaluation	Goal	Action Indicated If:	Recommended Treatment
Peripheral pulses	Each visit	Normal	Abnormal	See above
Assessment of Microvascular Complications				
Retinopathy:				
Dilated eye exam by eye care specialist	Annually	Normal	Abnormal	Referral to ophthamology Adequate glycemic control
Nephropathy:				
Microalbumin	Annually or quarterly if abnormal	< 30 mg/24 h or < 30 mg/g of creatinine (spot urine)	≥ 30 mg/24 h or ≥ 30 mg/g of creatinine (spot urine)	Adequate glycemic control Adequate treatment with ACE inhibitors Adequate treatment of hyperlipidemia Referral to nephrology if necessary
Neuropathy:				
Peripheral sensory	Annually	Intact sensation	Abnormal	Protective and preventive education Adequate glycemic control Drug treatment for symptomatic disease
Feet exam	Each visit	No complications	Corns, calluses, ulcers, wounds, infections	Referral to podiatry and/or vascular surgery specialist Adequate control of lipid abnormalities and blood glucose Treatment of infections if present

Assessment of Other Complications				
Oral/periodontal	Each visit Dental visit and hygiene every 6 months	Healthy gums/teeth	If no routine dental visits and hygiene are being performed	Referral for dental hygiene and care Adequate glycemic control*
Other infection	Each visit	Absence of infection	If infection is present	Adequate glycemic control* Appropriate treatment of infection and referral to ID if necessary
Lifestyle Assessment				
Exercise	Each visit	20-45 minutes on most days	< 3 times weekly	Exercise counseling related to type, frequency, duration, and intensity
Smoking, tobacco use	Each visit	No use	Any use	Smoking cessation counseling
Weight	Each visit	Ideal body weight	Patient is over- or underweight	For overweight: diet adjustment for short term-weight lost of 0.2-0.5 kg/week; for long term weight loss as much as needed to attain IBW For underweight: if severe — consult NST If mild — assess the reasons for weight loss and treat accordingly
Nutrition	Each visit Annual in-depth assessment by RD	Healthy eating daily weight control Metabolic control	Poor glucose or lipid control or increased weight	Referral for nutrition counseling In-depth nutrition assessment, plan, and followup by RD
Overall diabetes self-management practices	Each visit Annual in-depth assessment and self-management update	Healthy diabetes management with metabolic control and at least annual diabetes assessment and self-management education update	Early signs of complications and early signs of poor self-management of diabetes	Referral to diabetes educator or formal diabetes education classes for assessment, plan, evaluation, and followup by CDE

Severe gum disease or any local of systemic infection is associated with higher glucose levels. Treatment of infection improves glycemic control.

TABLE 53.15

Treatment Decisions Based on LDL Cholesterol Levels in Adults

	Medical Nutrition Therapy		Drug Therapy	
	Initiation Level	LDL Goal	Initiation Level	LDL Goal
With CAD, PVD, or CVD	>100	≤100	>100	≤100
Without CAD, PVD, or CVD	>100	≤100	≥130	≤100

Data are given in mg/dl.

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and to stop progression or cause regression in patients with already established macrovascular disease. The goal for LDL cholesterol levels is 100 mg/dl for all diabetics. Lipid-lowering drug therapy is recommended to be started in patients with established vascular disease (coronary heart disease, peripheral vascular or cerebrovascular disease) if the levels of LDL-cholesterol are above 100 mg/dl. In diabetic patients without established macrovascular disease, lipid-lowering drug therapy is recommended for LDL-cholesterol of 130 mg/dl or above. The recommendations for treatment of elevated LDL-cholesterol are summarized in Table 53.15. Pharmacological therapy should be initiated after behavioral interventions are used. However, in patients with clinical CAD or very high LDL-cholesterol levels (i.e., ≥200 mg/dl), pharmacological therapy should be initiated at the same time that behavioral therapy is started. According to the NCEP-ATP III guidelines, diabetes drug treatment and behavioral modification should be performed simultaneously. The ADA guidelines recommend that diabetic subjects with clinical CAD and an LDL cholesterol level of >100 mg/dl be treated with pharmacological agents. For diabetics without preexisting CAD, the current ADA recommendations for starting pharmacological therapy are LDL-cholesterol levels ≥130 mg/dl. Recent clinical trial data strongly suggests that these goals may become more stringent soon.

A point to consider in diabetic patients is the method used to measure LDL-cholesterol. Due to the prevalence of dense LDL in these patients, the conventional method to calculate LDL-cholesterol is inappropriate and the levels determined are in general falsely low.

Increased triglyceride levels are also recognized as a target for intervention. The levels of triglycerides considered acceptable are <150 mg/dl, and the HDL-cholesterol levels >45 mg/dl. The initial therapy for hypertriglyceridemia is behavioral modification with weight loss, increased physical activity, and no alcohol consumption. In the case of severe hypertriglyceridemia (≥1,000 mg/dl according to the ADA guidelines and ≥500 mg/dl according to NCEP-ATP III guidelines), severe dietary fat restriction (15 to 20% of kcalories as fat) in addition to pharmacological therapy is necessary to reduce the risk of pancreatitis. These patients are hard to manage, and improving glycemic control rather tightly is a very effective measure for reducing triglyceride levels and should be aggressively used before the introduction of fibric acid derivatives.

Nutritional Recommendations and Principles for the Dietary Treatment of Diabetics

Goals of Nutrition Therapy

The goals of medical nutrition therapy are to optimize health, control diabetes, and prevent or delay complications of diabetes.[21] The goals are summarized in Table 53.16. MNT is

TABLE 53.16

Goals of Medical Nutrition Therapy for Diabetes

- Achieve and maintain near-normal blood glucose goals
- Achieve and/or maintain optimal blood lipid levels
- Achieve and/or maintain normal blood pressure
- Prevent, delay or treat nutrition-related complications
- Provide adequate kcalories for achievement of reasonable body weight
- Provide optimal nutrition for maximizing health and for growth, development, pregnancy, and lactation

individualized for the person with diabetes to integrate the therapy into the daily routine of living. A registered dietitian completes a nutritional assessment and develops the individualized meal plan and behavioral interventions with the person with diabetes and the family.[22] The effectiveness of the dietary interventions in helping the person with diabetes achieve the identified goals should be evaluated routinely until goals are achieved. If goals are not met, changes in the overall management plan are needed. When goals are achieved, reassessment, continuing education, and evaluation should occur at least annually, and more often with changes in lifestyle, to assure optimal control of diabetes and maintenance of health.

Nutrition Therapy for Different Types of Diabetes

Type 1 Diabetes

The person with type 1 diabetes is typically thin or within recommended weight range. Prior to diagnosis the patient may have experienced weight loss, frequent urination (polyuria), thirst and increased fluid intake (polydipsia), and hunger (polyphagia). The initial goals of MNT are to replace fluids, normalize blood glucose and lipids, and provide appropriate kcalories for healthy living. Food, insulin administration, and physical activity need to be well balanced to obtain optimal control. It is essential that the person with diabetes coordinates the eating and exercise patterns with the onset of action and the duration of the insulin. The person with newly diagnosed type 1 diabetes may be overwhelmed with changes in daily routine; thus, initially the focus is on survival skills for managing diabetes (Table 53.17) followed by teaching self-management knowledge and skills which are needed to optimally control diabetes and its complications.[23,24] Dietary changes to optimize health can be made more slowly over time. Figure 53.1 outlines the

TABLE 53.17

Survival Skills for Managing Diabetes

- To acquire an adequate knowledge of:
  - Basic food and meal guidelines (including when eating out)
  - Effect of carbohydrates on blood glucose
  - Amount of carbohydrates taken daily
  - Carbohydrate food groups, portion sizes, and label information
- To coordinate insulin administration with food intake
- To be able to perform self-monitoring of blood glucose
- To schedule exercise according to food intake and glucose control
- To acquire knowledge of how to treat hypoglycemia:
  (in general, 15 grams of carbohydrates should raise blood glucose 50-100 mg/dl in 15 minutes)
- To know that alcohol intake may cause hypoglycemia (by inhibiting gluconeogenesis in liver)
- To know why, when, and how to call the health care provider and/or dietitian
- To have an established plan for recording self-management and returning for continuing care

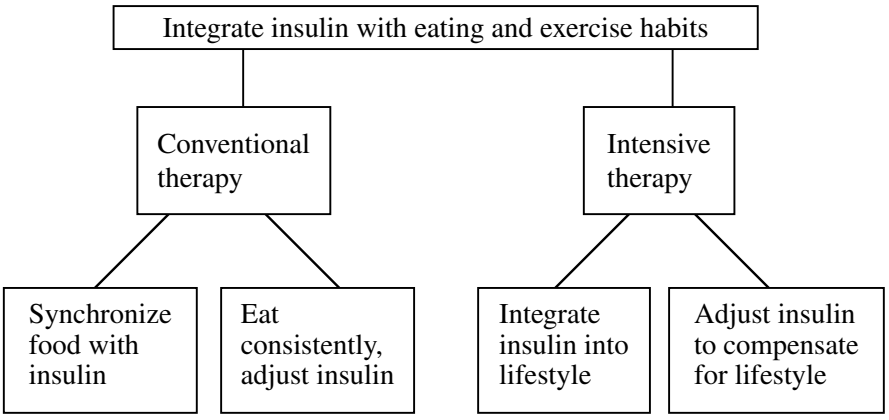

FIGURE 53.1
Nutrition therapy for type 1 diabetes. Reprinted with permission from *Maximizing the Role of Nutrition in Diabetes Management*, American Diabetes Association, Alexandria, 1994.

two approaches to nutrition therapy currently recommended by the ADA.[25] Blood glucose levels need to be monitored and insulin doses and/or food intake to control blood glucose adjusted according to recommended levels (Table 53.14).

Type 2 Diabetes

The person with newly diagnosed type 2 diabetes may have had asymptomatic type 2 diabetes for a number of years prior to diagnosis, and may present with one or more complications. Most are obese or have increased percentage of body fat distributed predominately in the abdominal region. The goals of therapy are to achieve and maintain glucose, lipid, and blood pressure within the recommended range and to achieve a moderate weight loss (5 to 9 pounds) if overweight.[26,27] Methods for attaining these goals are outlined in Figure 53.2. There is no clear answer about which goal should have first priority; however, the UKPDS researchers found that blood pressure control produced the most improved outcomes.[28] The desired outcomes of medical nutrition therapy for persons with type 2 diabetes are outlined in Tables 53.14 and 53.16.

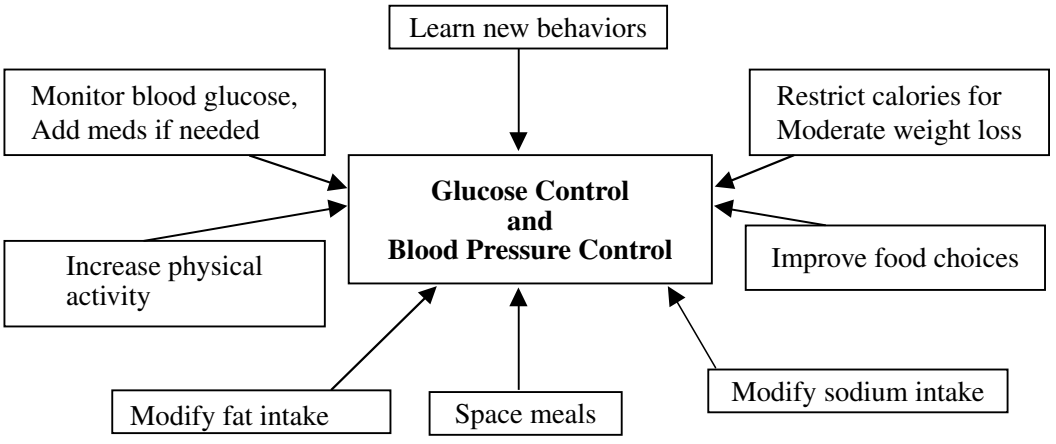

FIGURE 53.2
Nutrition therapy for type 2 diabetes. Adapted with permission from *Maximizing the Role of Nutrition in Diabetes Management*, American Diabetes Association, Alexandria, 1994.

TABLE 53.18

Goals for Medical Nutrition Therapy in GDM

- Optimal nutrition for developing fetus
- Optimal nutrition for mother
- To maintain maternal euglycemia keeping an adequate diet
- Good nutrition patterns taught to the family 'gatekeeper'
- To develop nutritional patterns that prevent or forestall recurrence of GDM and onset of type 2 diabetes mellitus

Reprinted with permission from American Diabetes Association: Thomas-Dobersen, D., et al. *Clinical Diabetes* 1: 172; 1999.

TABLE 53.19

Blood Glucose Goals for Pregnancy

	Preexisting Diabetes Mellitus	Gestational Diabetes
Fasting	70-100 mg/dl	≤105 mg/dl
Premeal	70-100 mg/dl	70-105 mg/dl
Postprandial 1 hour	≤140 mg/dl	≤120 mg/dl
Postprandial 2 hour	≤120 mg/dl	≤120 mg/dl

Adapted with permission from Hoeer, J.H., Green-Pastors, J., *Diabetes Medical Nutrition Therapy*, American Diabetes Association, Alexandria, ch. 8, 1997.

Diabetes in Pregnancy

The goal of nutrition therapy for diabetes in pregnancy is to produce a healthy baby at term and maintain optimal health for the mother. If type 1 or type 2 diabetes is diagnosed prior to pregnancy, counseling is recommended to attain optimal control of diabetes prior to conception and throughout pregnancy. The pregnant woman is defined as having gestational diabetes if first diagnosed during the present pregnancy. GDM does not exclude the possibility that diabetes may have been unrecognized prior to pregnancy; however, GDM typically occurs around the 24th week of pregnancy. Women diagnosed with GDM in a prior pregnancy have a 30 to 65% probability of developing GDM in a subsequent pregnancy. Studies also show that women with GDM have a 22 to 30% probability of developing type 2 diabetes in 7 to 10 years and a 50 to 60% risk of developing diabetes in their lifetime. Additionally, the offspring have an increased risk of obesity and GDM.[29]

The overall goals for medical nutrition therapy are shown in Table 53.18, and blood glucose goals are shown in Table 53.19. To accomplish these goals, meal patterns, nutrient composition, and caloric needs are reviewed in Table 53.20, and a typical meal/snack pattern is shown in Table 53.21.

Nutritional Recommendations

Guidelines for nutrient consumption and other nutrition components such as sweeteners and cholesterol along with technical reviews of the evidence for the guidelines have been published by the ADA,[30-34] and are summarized in Table 53.22. The energy nutrients can be converted into blood glucose, and Table 53.23 summarizes their degree of conversion. Protein and fats have minimal effect on blood glucose except in patients whose diabetes is poorly controlled, since these nutrients may lead to a rapid increase in gluconeogenesis and deteriorated glycemic control.[32] Many persons with diabetes initially think that only sugar can increase blood glucose; thus, diabetes education about the effect of nutrients (especially carbohydrates) is essential to good control.

TABLE 53.20

Summary of Intensive Nutritional Therapy for GDM

Meal Pattern

Three meals plus three or more snacks (2-3 hour intervals)
(breakfast with low carbohydrate content)
ADA exchange pattern individualized

Composition

Nutrition therapy and exercise only (no insulin)
38-45% carbohydrate complex, high fiber, >150 g minimum
(lower level can be used as long as no ketonuria is present)
20-25% protein (1.3 g/kg body weight)
30-40% fat (mono or polyunsaturated emphasized)

Energy Levels

Second trimester, 25-30 kcal/kg IBW prepregnant
Third trimester, 30-35 kcal/kg IBW prepregnant

Weight Gain

Adjust kcalorie level to achieve appropriate weight gain for prepregnancy BMI category:

	Prepregnancy BMI	**Total gain**
Underweight	<19.8	28-40 lbs. (12.5-18 kg)
Normal weight	19.8-26	25-35 lbs. (11.5-16 kg)
Overweight	>26-29	15-25 lbs. (7-11.5 kg)
Obese	>29	Minimum 15 lbs. (7.1 kg)

Reprinted with permission from American Diabetes Association. Gunderson, E.P., *Diabetes Care* 20: 223; 1997.

TABLE 53.21

Intensive Medical Nutrition Therapy for GDM

Meal or Snack	**Total Carbohydrate kcalories (%)**	**Carbohydrate (g)**
Breakfast	10-15	15-30
A.M. snack	10	15-30
Lunch	20-25	30-60
P.M. snack	15	15-45
Dinner	25	45-60
Bedtime snack	15	30 or more
Daily total	100	150-250

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Carbohydrate level should vary according to kcalorie prescription and the individual's tolerance for carbohydrate, which worsens as gestation progresses. The restricted levels listed above are characteristic of late gestation.

TABLE 53.22

Medical Nutrition Therapy Recommendations

Nutrient	Recommendation	Comments
Protein		
Sources: chicken, fish, meat, eggs, milk, tofu, nuts, peanut butter	10-20% of total kcalories should come from protein sources.	Research indicates needs are similar for people with or without diabetes. With onset of nephropathy, limit protein to adult RDA (0.8 g/kg/day). Some research studies suggest vegetable protein may not be as harmful to the kidneys as animal protein.
Carbohydrate		
Sources: starch (grains, bread, pasta, rice, potato, beans) milk, fruit, vegetables, sugar, honey, jam, molasses, etc.	80-90% of kcalories are divided between carbohydrate and fats based on individual risk factors and needs. Depending on nutritional assessment and medical nutritional therapy goals, this generally equates to 45-60% of total kcalories from carbohydrate.	Total carbohydrate intake has greater impact on blood glucose control than source of carbohydrate, i.e., whether complex carbohydrate or sucrose. Sucrose and sucrose-containing foods should be consumed within the context of a healthful diet. These foods are often high in total carbohydrate and fat and low in vitamins and minerals.
Sugars and Other Sweeteners		
Sources: sucrose, fructose, corn sweeteners such as corn syrup, fruit juice, or fruit juice concentrate, honey, molasses, dextrose, maltose; sorbitol, mannitol, xylitol (sugar alcohols)	% of kcalories will vary and is individualized based on usual eating habits, glucose and lipid goals. Sucrose and other sugars/sweeteners can be integrated into a healthy eating pattern for persons with diabetes.	Sucrose and sucrose-containing foods should be consumed within the context of a healthful diet. These foods are often high in total carbohydrate and fat and low in vitamins and minerals. Individuals can be taught to substitute sucrose-containing foods for other carbohydrate foods in their meal plans.
Nonnutritive sweeteners such as saccharin, aspartame, acesulfame K, and sucralose	All are approved by the FDA and the FDA determines an acceptable daily intake (ADI) which includes a 100-fold safety factor. Actual intake by persons with diabetes is well below the ADI.	
Fat		
Sources: monounsaturated: olive and canola oils, avocado, nuts polyunsaturated: safflower, sunflower, corn, and soy oils	60-70% of total kcalories should be divided between monounsaturated fats and carbohydrates. Up to 10% of kcalories should be from polyunsaturated fats.	Individuals with diabetes should limit total fat to 25-35% of total kcalories and <200 mg of dietary cholesterol per day. If obesity and weight management are the primary issues, reduced total fat to reduce total kcalories and increasing exercise should be recommended.

TABLE 53.22 *(Continued)*

Medical Nutrition Therapy Recommendations

Nutrient	Recommendation	Comments
saturated: butter, lard, shortening, animal fats, coconut, and palm oils	Less than 7% of total kcalories should be from saturated fats. Depending on nutritional assessment, total fat intake equates to 25-35% of total kcalories.	Guidelines for reducing cardiovascular risk are emphasized — nobody should exceed 7% of total kcalories from saturated fats. If elevated triglyceride and very low-density lipoprotein cholesterol are the primary concerns, a moderate increase in monounsaturated fat intake, with <7% of total kcalories from saturated fat, and a more moderate (slight decrease) in carbohydrate can be tried. Some studies have shown that a diet with increased total fat from monounsaturated fats can lower plasma triglycerides, glucose, and insulin levels more than a high-carbohydrate diet in some individuals. In individuals with triglycerides >1000, reduction of all types of dietary fats to reduce levels of plasma dietary fat in the form of chylomicrons should be implemented.
Fat replacers		
Typically fall into three categories based on their nutrient content: Carbohydrate-based: includes carrageenan, cellulose gum, corn syrup solids, dextrin, guar gum, hydrolyzed corn starch, maltodextrin, modified food starch, pectin, polydextrose, sugar beet fiber, tapioca dextrin, xanthan gum. Protein-based: includes microparticulated egg white and milk protein (Simplesse, K-Blazer), whey protein concentrate Fat-based: includes caprenin, olestra (Olean), salatrin (Benefat), and others.	Foods with fat replacers can be substituted in an individual's meal plan based on the nutrient profile of the food product.	Food products that contain <20 kcalories or 5 grams of carbohydrate per serving have a negligible effect on metabolic control. Foods containing 20 kcalories per serving should be limited to 3 servings spread throughout the day.

Dietary cholesterol	<200 mg per day	MNT typically reduces LDL Cholesterol 15-25 mg/dl (0.40-0.65 mmol/l)
Fiber	10-25 grams of soluble fiber per day — same recommendation as for individuals without diabetes.*	Research suggests that in the amounts typically consumed, fiber intake has very little impact on blood glucose levels.
Sodium	Same as for general population: <3000 mg per day. If hypertensive, individuals should reduce sodium intake to <2400 mg per day. Food selection guidelines: <400 mg sodium per single serving of food; <800 mg sodium per entree or convenience meal.	There is an association between hypertension and both IDDM and NIDDM, with an increase for people with NIDDM who are obese. There is also evidence that individuals with NIDDM are more salt sensitive.
Alcohol 1 drink = 12 oz. beer, 5 oz. wine, 1 oz. 80-proof liquor	Insulin users: limit to 2 drinks per day and do not cut back on food. Non-insulin users: substitute alcohol for fat.	Abstinence is recommended for those with history of alcohol abuse or alcohol-induced hypertriglyceridemia, and during pregnancy. Drink only with food. Alcohol can lead to hypoglycemia via inhibition of gluconeogenesis. Limit for weight loss and elevated triglycerides.
Micronutrients	The vitamin and mineral needs of people who are healthy appear to be adequately met by the RDAs, which include a generous safety factor.	Individuals at greatest risk for vitamin/mineral deficiency include those on weight loss diets, strict vegetarians, the elderly, pregnant or lactating women, those taking medications known to alter micronutrient metabolism, people with poor glycemic control (i.e., glycosuria), people with malabsorption disorders, and people with congestive heart failure or myocardial infarction.

* Exception in patients with autonomic neuropathy who should not have increased fiber in their diet.

Adapted and expanded from Karlsen, M., Khakpour, D., and Thomson, L.L. *Clinical Diabetes* 14: 54; 1996.

TABLE 53.23

Energy Nutrients and Their Absorption

Nutrient	Kcalories/Gram	% Nutrient Converted to Glucose	Estimated Time for Absorption*
Carbohydrate	4	100%	
Simple			5-30 min
Complex			1-3 h
Protein	4	50-60%	3-6 h
Fat	9	10%	3-8 h

* The absorption time is affected by the nutrient mix. For example, the sugar from a candy bar with high fat content is more slowly absorbed than a piece of candy than contains no fat.

TABLE 53.24

Meal Planning Approaches

Approach	Benefits	Drawbacks
Food guide pyramid	Well known by the general public	Little focus on meal spacing
Health food choices	Mixes guidelines with meal plan	Often perceived as diet
Exchange lists for meal planning	Places emphasis on all nutrient groups	Concept difficult to understand by the lay person
Counting plans	Good approach for specific nutrient intervention	Requires committed learner
Carbohydrate indications	Useful for adequate glucose control	Ignores other nutrients
Protein indications	Address diabetic nephropathy	Ignores other nutrients
Fat indications	Address weight or hyperlipidemia	Ignores other nutrients

Reprinted with permission from American Diabetes Association. Karlsen, M., Khakpour, D., and Thomson, L.L. *Clincal Diabetes,* May/June: 54; 1996.

Food Guides and Planning Food Intake for Persons with Diabetes

Historically, the approaches to planning food intake for persons with diabetes have ranged from starvation diets (during the pre-insulin era) to high-fat, low-carbohydrate diet plans, to our present system of more liberalized food intake. Various food guides and methods for planning food intake have been used. An overview of the meal planning approaches for providing MNT to persons with diabetes is reviewed in Table 53.24. A meal planning approach that provides the desired outcomes (decrease of complications and optimal health and satisfaction) is desirable. Carbohydrate counting is one method that allows maximum flexibility as well as excellent glycemic control. The different ways to count carbohydrates are reviewed in Table 53.25.

Food Labeling

Teaching patients with diabetes how to read a food label is especially important to those who count carbohydrate, fat, or protein in their meal plans. For persons using exchange

TABLE 53.25

Ways to Count Carbohydrates

Method	Description	Ease vs. Accuracy	Premeal or Bolus Dose Calculation*
Counting carbohydrate exchanges (interchanges)	Count each serving of starch, fruit, and milk as one carbohydrate exchange and consider them equal in carbohydrate value	Easiest method but also the least accurate Requires the least math skill	Calculate pre-meal dose units/exchange
Counting food exchanges	Add the carbohydrate values of all exchanges that contain carbohydrate (including vegetables) to obtain the carbohydrate total for a meal	Easy and fairly accurate	Calculate pre-meal dose as units/exchange, counting vegetables as carbohydrates, or calculate the bolus by dividing the total grams of carbohydrate in the meal by the insulin-to-carbohydrate ratio
Carbohydrate gram counting	Add carbohydrate gram values for all foods eaten to obtain the total carbohydrate intake per meal	More time-consuming than methods 1 and 2 but also quite accurate. Requires more math skill to add and divide 2- and 3-digit number	Calculate pre-meal dose by dividing the total grams of carbohydrate in the meal by the insulin-to-carbohydrate ratio
Calculating available glucose	Count grams of carbohydrate for all foods eaten, then calculate the glucose available from protein and add this value to the carbohydrate grams to obtain the meal total	Most difficult; requires the most math skill of all methods	Multiply grams of protein in meal by 0.6 to obtain available glucose, add to grams of carbohydrate; calculate the dose by dividing this total by the insulin-to-carbohydrate ratio

* Short-acting insulin adminstered before meals to control the meal-related glucose rise. The calibration of insulin to food intake is recommended for individuals with type 1 diabetes, especially those following intensive therapy.

lists for meal planning, information about how to use the nutrition information to fit foods into the exchange lists is helpful (and essential for combination foods). The key question for the patient to ask is "how does this food, based on the nutritional information, fit into my food plan for controlling diabetes?"[35]

Diabetes and Physical Activity

Exercise can be used as a therapeutic tool for controlling diabetes, and the person with diabetes needs to incorporate exercise into the lifestyle for healthy living with diabetes.

Patient Evaluation before Exercise

The person with diabetes should undergo a medical evaluation with appropriate diagnostic studies and should be screened for complications that may be worsened by the

exercise program.[36] If complications are present, the patient should have an individualized exercise program prescription that specifies the frequency, intensity, and duration of exercise, along with specific precautions for minimizing risks. The benefits of physical activity are many, including cardiovascular fitness and psychological wellbeing. However, the risks of exercise for the person with diabetes are many, including fluctuations in blood glucose control, ketosis, lower-extremity injury, and exacerbation of pre-existing complications.

Exercise Recommendations

For Persons with Cardiovascular Disease

Diabetics at risk or with diagnosed cardiovascular disease should undergo medical evaluation of cardiac status and special evaluation for exercise tolerance before participating in increased physical activities. Supervised cardiovascular risk reduction or rehabilitation programs often provide the patient and his family with increased support for increasing physical activities.

Positive effects of regular exercise on reducing blood pressure have been consistently demonstrated in hyperinsulinemic persons.

For Persons with Peripheral Arterial Disease

Following an evaluation of peripheral arterial disease, the basic treatment is a supervised exercise program and no smoking, carried out under the supervision of a physician. A walking program may improve muscle metabolism and collateral circulation for a person with intermittent claudication. If pain is severe and does not improve, further evaluation and possible limitation of exercises involving the lower extremities may be considered.

For Persons with Retinopathy

Following a dilated eye exam, if proliferative diabetic retinopathy is present, the person with diabetes may need to avoid anaerobic exercise and exercise that involves straining, jarring, or Valsalva maneuvers, and any other activities that increase systolic blood pressure. Medical status dictates the level of risks associated with exercise; however, low-impact cardiovascular conditioning such as swimming, walking, low-impact aerobics, stationary cycling, and endurance exercises are low risk.

For Persons with Nephropathy

Specific exercise recommendations have not been developed for persons with nephropathy, but some patients may self-limit exercise based on a reduced capacity for activity. High-intensity or strenuous exercises should probably be discouraged for persons with overt nephropathy, but other low intensity-physical activities may increase a sense of wellbeing and socialization.

For Persons with Neuropathy

Peripheral

For the person who has loss of protective sensation in the feet on testing, weight-bearing exercises are contraindicated. This includes use of treadmill, prolonged walking, jogging, and step exercises. Recommended exercises include swimming, bicycling, rowing, chair and arm exercises, along with other nonweight-bearing exercises.

Autonomic

Autonomic neuropathy increases the risks of exercise-related problems, and certain precautions need to be taken to tailor the exercise prescription to each individual patient following an in-depth evaluation. Thermoregulation may be difficult, so avoiding exercise in hot or cold environments, and special attention to adequate hydration are most important.

Exercise and Glycemic Control

Regular exercise activities (30 or more minutes on most days) have demonstrated consistent beneficial effects on carbohydrate metabolism and insulin sensitivity, as well as enhanced weight loss.

Exercise for Persons with Type 1 Diabetes

Persons with type 1 diabetes, who do not exhibit some of the limiting complications previously discussed or poor glycemic control, can enjoy all types of exercise.[37] The key is regulating the glycemic response to exercise. The person should avoid exercise if fasting glucose levels are >250 mg/dl with ketosis present or if glucose levels are >300 mg/dl. If glucose levels are <100 mg/dl prior to exercise, additional carbohydrates are recommended. Food adjustments for exercise for persons with type 1 diabetes are shown in Table 53.26. Food and fluids should be readily available for persons with type 1 diabetes

TABLE 53.26

Food Adjustments for Exercise for Persons with Type 1 Diabetes

	General Guidelines		
Types of Exercise and Examples	**If Blood Glucose Is:**	**Increase Food Intake By:**	**Suggestion of Food Exchanges to Use:**
Short duration, low-to-moderate intensity (walking a half mile or leisurely bicycling for <30 minutes)	<100 mg/dL ≥100 mg/dL	10 to 15 g of CHO None	1 Fruit or 1 starch
Moderate intensity (1 hour of tennis, swimming, jogging, leisurely bicycling, golfing, etc.)	<100 mg/dL	25 to 50 g CHO before exercise, then 10 to 15 g/h of exercise	1 Meat sandwich with a milk or fruit
	100-180 mg/dL 180-300 mg/dL >300 mg/dL	10 to 15 gm CHO None Do not begin exercise until blood glucose is under control	1 Fruit or 1 starch
Strenuous (about 1-2 hours of football, hockey, racquetball, or basketball; strenuous bicycling or swimming; shoveling heavy snow)	<100 mg/dL	50 g CHO, monitor blood glucose carefully	1 Meat sandwich (2 slices of bread) with a milk and fruit
	100-180 mg/dL	25 to 50 g CHO depending on intensity and duration	Meat sandwich with a milk and fruit
	180-300 mg/dL >300 mg/dL	10 to 15 g CHO Do not begin exercise until blood glucose is under better control	1 Fruit or 1 starch

Self-blood glucose monitoring is essential for all persons to determine their carbohydrate needs. Persons with type 2 diabetes usually do not need an exercise snack. During periods of exercise, all individuals need to increase fluid intake.

CHO = carbohydrates.

Adapted with permission from Franz, M.J., and Barry, B., *Diabetes and Exercise*: Guidelines for Safe and Enjoyable Activity, American Diabetes Association, Alexandria, 1996, p. 16.

during exercise. If the duration of exercise is 30 minutes or more during peak action time of insulin and blood glucose is in good control, reduction of insulin is recommended. The reduction of insulin is based on duration and intensity of exercise, and usually ranges from 5 to 60% of daily requirements. After exercise, an extra carabohydrate snack may be necessary. Frequent monitoring of blood glucose and adequate food/fluid intake to prevent hypoglycemia are essential for self-management and maintaining a healthy lifestyle.

Exercise for Persons with Type 2 Diabetes

Many persons with type 2 diabetes may have some of the previously mentioned complications at diagnosis, and also may have been sedentary for many years. Thus, before beginning an exercise program, an in-depth physical examination and recommendations for exercise frequency, intensity, and duration is recommended.[38] Beginning with 5 to 10 minute sessions with gradual increases usually is successful and safe. Unless treated with insulin or glucose-lowering medications, the person with type 2 diabetes does not usually need additional food before, during, or following exercise, except for exercise that is intense or of long duration. Recent attention has focused on the useful role of exercise in preventing or delaying the onset of type 2 diabetes.

Exercise for Older Adults with Diabetes

Exercise for older adults with diabetes is recommended, and may lead to an improved quality of life and less chronic disease. The same precautions should be taken with older adults with and without diabetes.

Hospital Admission Guidelines for Persons with Diabetes

If the standard of care is adequate, seldom will a diabetic patient require hospitalization. According to the guidelines of the ADA, inpatient care may be required in:

- Life-threatening acute metabolic complications of diabetes
- Newly diagnosed diabetes in children and adolescents
- Patients with chronic poor metabolic control that necessitates close monitoring to determine the problem behind the poor control and changes in therapy
- Patients with severe chronic complications that require intensive treatment either of diabetes or of conditions that significantly affect diabetes control and further development of complications
- Uncontrolled or newly discovered insulin-requiring diabetes during pregnancy
- Patients in whom institution of insulin-pump therapy or other intensive insulin regimens are being contemplated

Translation of Medical Nutrition Therapy for Diabetes to Health Care Institutions

Today's recommendations for MNT in health care facilities are based on individualized needs of the patients with diabetes. One of the approaches frequently used is the "con-

TABLE 53.27

Developing a Consistent Carbohydrate Diabetes Meal Plan Menu for Health Care Facilities

1. Establish the desired kcalorie range.
2. Determine the desired percentages of macronutrients (carbohydrate, protein, saturated fat, total fat).
3. Determine the numbers of CHO choices to be given at each meal, and if included, at bedtime snack.
4. Determine how often to include sucrose-containing desserts and the maximum number of CHO choices to be allotted to each dessert.
5. Analyze current fat-modified menus for distribution of macronutrients (% carbohydrate, protein, saturated fat, total fat) to determine if they meet goal ranges of new diabetic menus.
6. Determine how many grams of carbohydrate or CHO choices are in each item in the fat-modified menu (i.e., fruits, salads, starches, casseroles, desserts, milk, juices).
7. For nonselective menus, adjust the fat-modified menus to provide the established number of CHO choices, and include a bedtime snack if desired.
8. For facilities with menu selections, identify the CHO choices for each carbohydrate item, and include instructions on the menu regarding the number of carbohydrate choices to make at each meal.
9. For long-term care facilities that wish to base their diabetic diet (consistent carbohydrate diet) on regular menus, use the same process as for fat-modified menus.

Reprinted with permission from Schafer, R.G. *Practical Diabetology* 16:3, 48; 1997.

sistent carbohydrate diabetes meal plan;" a plan for developing a menu is shown in Table 53.27.

Acute Health Care Facilities

Approximately one out of seven hospital beds is occupied by a person with diabetes. In the acute care facility, many of the patients have complex health problems in addition to diabetes. Thus, the challenge is to maximize health potential and provide foods that are culturally acceptable to the patient. Each acute care facility has a different meal planning system that best meets its needs. The consistent carbohydrate menu plan can be used to improve metabolic control. The ideal meal plan reflects the diabetes nutrition recommendations and does not unnecessarily restrict sucrose.[39]

Long-Term Health Care Facilities

Since the risk of diabetes increases with age, the patient population in long-term care includes many individuals with diabetes. Additionally, malnutrition is a recognized challenge in the older adult population. Food intake should be adequate and not overly restricted. Regular menus, with consistent amounts of carbohydrate at meals and snacks, are the recommended approach. Monitoring of blood glucose and hemoglobin A_{1c} should be used to evaluate glycemic control, with individualized approaches to achieve goals of MNT.

Self-Management Education for Persons with Diabetes

Diabetes self-management education and continuing nutrition care is essential for meeting the goals of MNT and diabetes control.[23,30] The outpatient and home settings are the ideal environments. If the patient is hospitalized with multiple other priorities, usually the concern of the patient and the family is not focused on diabetes self-management education. Learning readiness is the cornerstone for self-management education. At discharge from the inpatient facility, plans are made for continuing education and followup by the health care team.

Education for Health Care Professionals and Administrators

The role of MNT in helping the team and person with diabetes attain the desired treatment goals is cost effective and leads to quality health services. One of the roles of the registered dietitian is to translate nutrition recommendations for the diabetes care team and to integrate these recommendations into the overall care of the person with diabetes. Team members should have access to simplified guidelines for patient nutrition care until a registered dietitian is available.

Third Party Reimbursement for Diabetes Care, Supplies, and Self-Management Education

Currently, 37 states have enacted diabetes reform laws that improve insurance coverage for supplies, equipment, and education for people with diabetes. States that have enacted these laws are listed in Table 53.28.[40] MNT counseling is usually included in diabetes education coverage; however, examination of each state's reform laws is necessary to determine the extent of coverage. New Medicare regulations are expected to be released in 2000, and diabetes advocates and nutrition professionals are working to assure inclusion of MNT in these regulations. Medicaid, a federal–state partnership program for persons unable to afford health care and private third-party insurance, offers coverage for MNT in some states. To determine if your state offers coverage, contact the state's Medicaid program.

The frequency of dietitian contact with the patient and the essential care processes for MNT have not been clearly delineated; yet quality health care today requires consistently applied, evidence-based care that leads to positive outcomes for most patients. In a research study conducted by the Diabetes Care and Education Practice Group of the ADA, use of guidelines resulted in changes in dietitian practices and produced greater improvement in patient blood glucose outcomes at three months compared to usual care.[41] Self-management education is critical to successful diabetes management, and medical treatment without self-management education is regarded as substandard and unethical. Numerous studies have demonstrated that self-management education and MNT improve outcomes for persons with diabetes.

TABLE 53.28

States that have Enacted Diabetes Reform Laws (as of January 2000)[27]

Arizona	Iowa	Nebraska	Rhode Island
Arkansas	Kansas	Nevada	South Carolina
California	Kentucky	New Hampshire	South Dakota
Colorado	Louisiana	New Jersey	Tennessee
Connecticut	Maine	New Mexico	Texas
Florida	Maryland	New York	Vermont
Georgia	Minnesota	North Carolina	Virginia
Illinois	Mississippi	Oklahoma	Washington
Indiana	Missouri	Pennsylvania	West Virginia
			Wisconsin

From Maggio, C.A., Pi-Sunyer, F.X. *Diabetes Care* 20: 1744; 1997.

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