

## Chapter

## 4

APPLYING STANDARDS  
OF CARE*Vivian Fonseca, MD*

The major risk factors associated with the development of the microvascular and macrovascular complications of diabetes are hyperglycemia, dyslipidemia, and hypertension. Controlled clinical trials have demonstrated that controlling these risk factors can prevent complications or lessen their severity.

**GLYCEMIC CONTROL**

Glycemic control is critical and can be optimized through individualized treatments targeting a patient's specific characteristics and point of disease progression:

- ▲ Patients with type 1 diabetes, having no endogenous insulin secretion, must be treated with a basal-bolus insulin regimen to reproduce normal physiology
- ▲ Patients with type 2 diabetes, who vary in their insulin secretion and insulin resistance, may begin with lifestyle modifications to reduce their insulin resistance, and/or oral agents, but because of their progressive deterioration of insulin secretion, often ultimately require insulin

The A1C level is the generally accepted measure of glycemic control. It reflects average glycemia over the previous 2 to 3 months. The normal A1C range in nondiabetic individuals is 4% to 6%.

- ▲ Treatment goals must be tailored to individual patients and allow for risk factors such as age and propensity for hypoglycemia. The latter is particularly important in children, whose mental development can be affected by hypoglycemia, or in the elderly (and others) who may not recognize the symptoms of hypoglycemia.

- Organizations such as the American Diabetes Association (ADA), the American Association of Clinical Endocrinologists (AACE), and CADRE have published different targets, depending on their interpretation of clinical trial results.

### Glycemic Targets

Clinical trials indicate that achievement of A1C levels within the normal range with certain pharmacologic agents increases a patient's risk of hypoglycemia. CADRE takes the position that it is prudent to aim for lower A1C levels than those currently recommended, allowing for exceptions in individual patients. Tables 4-1 and 4-2 outline targets for glycemic control in nonpregnant patients with diabetes.

**Table 4-1** A1C Targets for Nonpregnant Patients With Diabetes

Normal	ADA	AACE	CADRE
4-6	<7 (Near-normal targets may be considered for individual patients)	≤6.5	Lowest possible without unacceptable hypoglycemia Action suggested at A1C>7

Sources: ADA, 2004<sup>3</sup>; AACE, 2002<sup>13</sup>; CADRE.<sup>14</sup>

**Table 4-2** Other Glycemic Targets for Nonpregnant Patients With Diabetes

Parameter	Normal	ADA	AACE
Preprandial plasma glucose (mg/dL)	<100 (mean ~90)	90-130	<110
Postprandial (2-h) plasma glucose (mg/dL)	<140	<180	<140

Sources: ADA, 2004<sup>3</sup>; AACE, 2002.<sup>13</sup>

The ADA bases its targets on data from the Diabetes Control and Complications Trial (DCCT)<sup>1</sup> and the United Kingdom Prospective Diabetes Study (UKPDS)<sup>2</sup> demonstrating that the risk of long-term microvascular and macrovascular complications can be minimized with A1C <7%—although there is no threshold level for benefit. The AACE level of  $\leq 6.5\%$  is based on its own reading of data indicating that reducing the risk of macrovascular complications requires lower levels.

The intervals at which A1C levels should be measured depend largely on the patient's degree of glycemic control, with those who are poorly controlled requiring more frequent assessment. Table 4-3 outlines suggested parameters for testing.

There are some limitations of A1C testing, such as for certain populations. These limitations, and recommendations for overcoming them, are explained in Table 4-4.

### *Monitoring of Blood Glucose by the Clinician and Patient*

To complement the long-term assessment provided by A1C tracking, most patients with diabetes should self-monitor their blood glucose levels. As noted in Table 4-4, A1C does not specifically track fasting, postprandial, or nocturnal plasma glucose levels the way self-monitored blood glucose (SMBG) does. Clinical evidence indicates that SMBG can improve glycemic control because it provides:

- ▲ Feedback about how well they are responding to a given therapeutic strategy

**Table 4-3** Guidelines for Frequency of A1C Testing

Patient Status	Testing Interval
New patient	At diabetes diagnosis
Patient with poor glycemic control	Every 2–3 months
Patient meeting glycemic targets	At each visit

Source: ADA, 2004.<sup>3</sup>

**Table 4-4** Limitations of A1C Testing and Recommendations for Overcoming Them

Limitation	Recommendation
Patients with abnormal hemoglobin (eg, those with hemolytic anemias)	Testing of total glycated serum protein (GSP) or glycated serum albumin (GSA)—eg, fructosamine assay—provides an index of glycemic status over 1–2 weeks <sup>a</sup>
Pregnant patients or those changing therapies	Above recommendation
Lack of standardization in assay methods	Use laboratory that has passed A1C certification testing <sup>b</sup> Use the same lab consistently so results are comparable
A1C does not specifically track fasting, postprandial, or nocturnal plasma glucoses	Self-monitored blood glucose (SMBG) and office-based plasma glucose assessment

Source: ADA, 2004.<sup>3</sup>

<sup>a</sup>GSP measurement should be conducted at least monthly. It has not been shown to predict complication risk as A1C does.

<sup>b</sup>As sponsored by the ADA and the National Glycohemoglobin Standardization Program. The gold standard is the high-performance liquid chromatography (HPLC) procedure now used in most clinical trials.

- Information on how to avoid hypoglycemia and make necessary adjustments to medications, diet, and exercise<sup>3,4</sup>

Other considerations about SMBG include the following:

- The frequency of monitoring should reflect the patient's overall glycemic control and individual needs.
- Patients with type 1 diabetes, as well as patients with type 2 diabetes who are being treated intensively, should use

preprandial SMBG for insulin dose adjustment, and as a result, they should self-test three or more times per day preprandially, before going to bed, and occasionally overnight.

- ▲ Patients should keep track of results and review these logs with a member of their diabetes treatment team via phone, fax, E-mail, or personal visit.

### The clinician's role in SMBG

Clinicians should take an active role in their patient's SMBG. Those who accept less than optimal daily blood glucose targets have patients with significantly higher A1C levels (7.8% vs 7%) than are found in patients of clinicians who more aggressively promote lower blood glucose levels.<sup>5</sup>

Clinicians and their assistants must provide the means for<sup>2</sup>:

- ▲ Introducing patients to the necessary equipment and techniques; evaluating their progress at subsequent appointments
- ▲ Helping patients overcome psychological barriers to issues such as inconvenience, complexity, and discomfort with fingerprick methods
- ▲ Teaching patients to interpret the data properly in order to implement changes in medical nutrition therapy (MNT), exercise, or drug therapy<sup>4</sup>

See Chapter 5 for instruction on glucose monitoring techniques.

### MANAGEMENT OF DYSLIPIDEMIA

Clinicians should assess lipid levels in their patients with diabetes at least annually. Elevated triglycerides and/or low HDL cholesterol are common in people with diabetes. For patients who have dyslipidemia, the basic strategy is to:

- ▲ Lower low-density lipoproteins (LDL)
- ▲ Raise high-density lipoproteins (HDL)
- ▲ Lower triglycerides

Table 4-5 outlines the National Cholesterol Education Program (NCEP)<sup>6</sup> Adult Treatment Panel III (NCEP ATPIII) target lipid levels for persons with diabetes, which the ADA follows as well.

The ADA lists some additional recommendations regarding these guidelines. Generally, pharmacologic therapy should be started after lifestyle intervention has been initiated. However, if a patient has cardiovascular disease and LDL >100 mg/dL, pharmacologic therapy should be started at the same time as lifestyle intervention. For patients without existing cardiovascular disease, pharmacologic therapy should be started when LDL cholesterol is  $\geq 130$  mg/dL, with a goal of <100 mg/dL. Regarding triglyceridemia, it is the clinician's judgment whether to start pharmacologic therapy when triglyceride levels are between 200 mg/dL and 400 mg/dL. When levels are above 400 mg/dL, pharmacologic treatment is highly recommended to avoid the risk of pancreatitis. It is generally difficult to raise HDL cholesterol without pharmacologic intervention.

**Table 4-5** NCEP ATPIII Target Lipid Levels for Adult Patients With Diabetes

Lipid	Goal
LDL cholesterol	<100 mg/dL
HDL cholesterol	Men: >40 mg/dL Women: >50 mg/dL
Triglycerides	<150 mg/dL

*Source:* National Cholesterol Education Program (NCEP).<sup>6</sup> NCEP ATPIII, National Cholesterol Education Program, Adult Treatment Panel III; LDL, low-density lipoprotein; HDL, high-density lipoprotein.

### **Achieving Lipid Goals**

The primary means of achieving lipid goals include:

- ▲ Glycemic control
- ▲ Nutritional therapy (diets low in saturated fat and cholesterol and high in fiber)
- ▲ Exercise/weight loss
- ▲ Pharmaceutical approaches (eg, statins for lowering LDL; fibrates for lowering triglycerides; nicotinic acid for raising HDL and lowering triglycerides and LDL; combination therapy where indicated)

The order of priorities for treating dyslipidemia in patients with diabetes is detailed in Table 4-6.

MNT and drug therapy guidelines based on LDL cholesterol level are found in Table 4-7. Tables 4-8 and 4-9 describe specific nutrition and pharmacologic guidelines for the treatment of diabetic dyslipidemia.

### **MANAGING BLOOD PRESSURE**

Hypertension (blood pressure [BP]  $\geq 140/90$  mm Hg) affects 20% to 60% of patients with diabetes and increases the risk of both CV disease and of microvascular complications such as kidney disease and retinopathy:

- ▲ Patients with type 1 or type 2 diabetes may develop hypertension due to nephropathy.
- ▲ Patients with type 2 diabetes may also develop hypertension due to insulin resistance.

### **Blood Pressure Goals and Their Achievement**

Detailed blood pressure goals for patients with diabetes are found in Table 4-10. Clinicians can employ a number of therapeutic approaches to reach these targets, including the strategies outlined in Table 4-11 for mild to moderate hypertension.<sup>7-9</sup>

**Table 4-6** Priorities for Treating Diabetic Dyslipidemia in Adults

1. LDL cholesterol lowering<sup>a</sup>
  - ▲ Lifestyle intervention (weight loss, exercise, and smoking cessation)
  - ▲ First choice is a statin (HMG-CoA reductase inhibitor)
  - ▲ Second choice is a bile acid-binding resin; ezetimibe, which inhibits the intestinal absorption of cholesterol and related phyosterols; or nicotinic acid
  - ▲ Ezetimibe may be added to a statin to lower LDL cholesterol further
2. HDL cholesterol raising
  - ▲ Lifestyle intervention
  - ▲ Fibrates and/or nicotinic acid
3. Triglyceride lowering
  - ▲ Lifestyle intervention
  - ▲ Glycemic control is the first priority; may need to add high-dose statin
  - ▲ Fibrates (gemfibrozil, fenofibrate) or nicotinic acid may be useful
4. Addressing combined hyperlipidemia (increased LDL cholesterol and triglycerides)
  - ▲ Glycemic control plus add high-dose statin
  - ▲ Add a fibrate if necessary
  - ▲ Add nicotinic acid, bile acid-binding resin, or ezetimibe<sup>b</sup>
  - ▲ Omega-3 fatty acids may be used for very high triglycerides, if other measures fail

Source: ADA, 2004.<sup>15</sup>

LDL, low-density lipoprotein; HMG-CoA, coenzyme A.

<sup>a</sup>Decision for treatment of high LDL before elevated triglycerides is based on clinical trial data indicating safety as well as efficacy of the available agents.

<sup>b</sup>The combination of statins with nicotinic acid or a fibrate may carry an increased risk of myositis.

**Table 4-7 Treatment Decisions Based on LDL Cholesterol Levels in Adults with Diabetes**

Concurrent Condition	Lifestyle Intervention <sup>a</sup>		Drug Therapy	
	Initiation Level (mg/dL)	LDL Goal (mg/dL)	Initiation Level (mg/dL)	LDL Goal (mg/dL)
With CHD, PVD, or CVD	≥100	<100	≥100	<100
Without CHD, PVD, or CVD	≥100	<100	≥130 <sup>b</sup>	<100

Source: ADA 2004.<sup>15</sup>

LDL, low-density lipoprotein; HDL, high-density lipoprotein; CHD, coronary heart disease; CVD, cardiovascular disease; PVD, peripheral vascular disease.

<sup>a</sup>Lifestyle intervention consists of medical nutrition therapy (MNT) and physical activity.

<sup>b</sup>Several strategies are available for patients with LDL 100–129 mg/dL, including more aggressive MNT and treatment with a statin; if these patients also have HDL of <40 mg/dL, consider using a fibric acid such as fenofibrate.

**Table 4-8 Nutritional Recommendations for Management of Diabetic Dyslipidemia**

Dietary Component	Strategy
Calories	Restrict if patient is overweight
Carbohydrates	50–60% of daily energy intake, primarily complex carbohydrates <sup>a</sup>
Fiber	Intake should be high; ~20 g/1 Kcal
Fat	30% of daily energy intake; <10% saturated
Cholesterol	<300 mg/day
Omega-3 fatty acids	Should be reserved for patients with high triglycerides resistant to other therapy
Protein	10–15% of daily intake

Source: Horowitz, 1998.<sup>16</sup>

<sup>a</sup>If glycemic control or dyslipidemia worsens, dietary carbohydrate content should be reduced to 40–45% of total daily energy intake. Monounsaturated and polyunsaturated fats should replace saturated fats.

**Table 4.9** Pharmacologic Management of Dyslipidemia in Adults

	Effect on Lipoprotein		
	LDL	HDL	Triglycerides
First-line agents			
LDL lowering			
▲ HMG-CoA reductase inhibitor	↓↓	↔↑	↔↓
Triglyceride lowering			
▲ Fibric acid derivative	Variable	↑	↓↓
Second-line agents			
LDL lowering			
▲ Bile acid resins	↓	↔	↑
▲ Ezetimibe	↓	↑	↓
LDL and triglyceride lowering			
▲ Nicotinic acid	↓	↑↑	↓↓
▲ Omega-3 fatty acids	↔	↔	↓↓

↓, decrease; ↑, increase; ↔, no change.

LDL, low-density lipoprotein.

**Table 4.10** Blood Pressure Treatment Goals for Patients With Diabetes

Condition	Treatment Strategy
BP <130/80 mm Hg	No treatment necessary
sBP 130–139 or dBp 80–89 mm Hg	Lifestyle/behavioral therapy for 3 months; proceed to pharmacologic therapy if goals not achieved

(table continues)

**Table 4-10** (continued)

Condition	Treatment Strategy
sBP $\geq$ 140 or dBP $\geq$ 90 mm Hg (ie, hypertension)	Initiate drug therapy (ACE inhibitors, ARBs, $\beta$ -blockers, diuretics, or calcium channel blockers)
Hypertension with microalbuminuria or clinical albuminuria/nephropathy	Initiate drug therapy with ACE inhibitors or ARBs
Age $>$ 55 with or without hypertension, but with other CV risk factors	ACE inhibitor or ARB for cardiovascular protective effects
Recent MI	$\beta$ -Blockers in addition to other therapies (ACE inhibitors or ARBs)

Source: ADA, 2004.<sup>7</sup>

sBP, systolic blood pressure; dBP, diastolic blood pressure; ACE, angiotensin-converting enzyme; ARB, angiotensin receptor blockers; CV, cardiovascular; MI, myocardial infarction.

**Table 4-11** Lifestyle Modifications for Hypertensive Patients With Diabetes

Modification	Goal
Stop smoking	Overall CV health
Lose weight	As near-normal BMI as possible
Reduce sodium intake	$<$ 100 mmol/day (2.5 g sodium or 6 g sodium chloride)
Limit alcohol intake	$<$ 2 drinks/day
Maintain adequate intake of potassium, calcium, and magnesium	When possible, emphasize natural food sources, rather than supplements

Sources: Kaplan, 1998;<sup>8</sup> Kaplan, 2001.<sup>9</sup>

BMI, body mass index; CV, cardiovascular.

**Table 4-12** Antihypertensive Agents in the Context of Diabetes \*

Agent	Advantages	Disadvantages
Angiotensin-converting enzyme (ACE) inhibitors	<ul style="list-style-type: none"> <li>▲ Renoprotective</li> <li>▲ Reduce CV risk</li> <li>▲ Neutral or positive effect on lipids, glucose</li> </ul>	<ul style="list-style-type: none"> <li>▲ Cough</li> <li>▲ Hyperkalemia</li> <li>▲ Should not be used in pregnant women</li> </ul>
Angiotensin receptor blockers (ARBs)	<ul style="list-style-type: none"> <li>▲ Renoprotective</li> <li>▲ Reduce CV risk</li> <li>▲ Neutral effect on lipids, glucose</li> </ul>	<ul style="list-style-type: none"> <li>▲ Should not be used in pregnant women</li> </ul>
Thiazide diuretics	<ul style="list-style-type: none"> <li>▲ Reduce plasma volume</li> <li>▲ Act synergistically with other antihypertensive drugs</li> <li>▲ Reduce CV risk</li> </ul>	<ul style="list-style-type: none"> <li>▲ Adversely effect glucose and lipid levels at high doses</li> <li>▲ Reduced muscle blood flow during exercise</li> <li>▲ Impotence</li> </ul>
β-Blockers	<ul style="list-style-type: none"> <li>▲ Reduce CV risk, especially after acute MI</li> </ul>	<ul style="list-style-type: none"> <li>▲ Nonselective β-blockers may interfere with awareness of hypoglycemia</li> <li>▲ Worsen insulin resistance and glucose tolerance</li> <li>▲ Raise triglycerides; lower HDL</li> <li>▲ Reduce peripheral blood flow</li> <li>▲ Reduce exercise ability by limiting cardiac output</li> </ul>

(table continues)

**Table 4-12** (continued)

Agent	Advantages	Disadvantages
Calcium-channel blockers	<ul style="list-style-type: none"> <li>▲ Reduce CV risk</li> <li>▲ Neutral effect on glycemic control and lipid levels</li> </ul>	<ul style="list-style-type: none"> <li>▲ May not offer as much CV protection as ACE inhibitors</li> <li>▲ Constipation</li> <li>▲ Pedal edema</li> </ul>
$\alpha$ -Blockers	<ul style="list-style-type: none"> <li>▲ Neutral effect on glycemic control and lipid levels</li> </ul>	<ul style="list-style-type: none"> <li>▲ Significantly less effective in reducing congestive heart failure than other approaches</li> <li>▲ May cause orthostatic hypotension</li> </ul>

Sources: ADA, 2004<sup>7</sup>; Kaplan, 2001, 1998.<sup>8,9</sup>

ACE, angiotensin-converting enzyme; ARB, angiotensin receptor blocker; CV, cardiovascular; MI, myocardial infarct; HDL, high-density lipoprotein.

\*Vasodilators such as hydralazine, clonidine, and minoxidil may also be used if needed to reach goal.

Table 4-12 outlines the advantages and disadvantages of available drugs. This table considers current clinical opinion by starting with angiotensin-converting enzyme (ACE) inhibitors and angiotensin receptor blockers (ARBs)—often considered the antihypertensive drugs of first choice for diabetes. However, the Anti-hypertensive and Lipid-Lowering Treatment to Prevent Heart Attack (ALLHAT) trial indicates that thiazide diuretics are the treatment of choice for uncomplicated hypertension, although the effectiveness has not been specifically assessed in the diabetic population. Tailoring any such therapy to the individual patient is paramount. Note that some patients may have concurrent conditions that contraindicate some antihypertensive drugs, as detailed in Table 4-13.

**Table 4-13** Concurrent Conditions That Affect Antihypertensive Drug Choices

Condition	Drugs of Choice	Drugs to Avoid
<i>Cardiac</i>		
Coronary heart disease	ACE inhibitor; calcium-channel blocker; cardioselective $\beta$ -blocker; diuretic	None
Heart failure	ACE inhibitor; ARB; diuretic; vasodilator	$\beta$ -blocker; calcium-channel blocker
Left ventricular hypertrophy	ACE inhibitor; ARB; $\alpha$ -blocker; cardioselective $\beta$ -blocker	Hydralazine
<i>Metabolic</i>		
Frequent hypoglycemia	ACE inhibitor; calcium-channel blocker	Nonselective $\beta$ -blocker
Hyperlipidemia	ACE inhibitor; $\alpha$ -blocker; calcium-channel blocker	Nonselective $\beta$ -blocker; diuretic
Hypoaldosteronism	Calcium-channel blocker; $\alpha$ -blocker	ACE inhibitor; $\beta$ -blocker; potassium-sparing diuretic
<i>Neuropathic</i>		
Impotence	ACE inhibitor; ARB; $\alpha$ -blocker; vasodilator	$\beta$ -blocker; central or peripheral adrenergic inhibitor; diuretic
Orthostatic hypotension	ACE inhibitor; ARB; calcium-channel blocker; vasodilator	$\alpha$ -blocker; central or peripheral adrenergic inhibitor

(table continues)

**Table 4-13** (continued)

Condition	Drugs of Choice	Drugs to Avoid
<i>Renal</i>		
Nephropathy	ARBs, ACE inhibitor; calcium-channel blocker	None
<i>Vascular</i>		
Peripheral vascular disease	ACE inhibitor; $\alpha$ -blocker; calcium- channel blocker	$\beta$ -Blocker

Sources: Kaplan, 1998<sup>8</sup>; Christlieb, 1990.<sup>17</sup>

ACE, angiotensin-converting enzyme; ARB, angiotensin receptor blocker.

### ***Impaired Glucose Tolerance, Impaired Fasting Glucose, and Type 2 Diabetes***

The standard ADA diagnostic criterion for diabetes is fasting plasma glucose (FPG)  $\geq 126$  mg/dL. However, patients considered hyperglycemic but not clinically diabetic (“pre-diabetic”) fall into another category that currently has two names, depending on the testing method used:

- ▲ *Impaired fasting glucose (IFG)*: 100 to 125 mg/dL
- ▲ *Impaired glucose tolerance (IGT)*: 2-h plasma glucose 140 to 199 mg/dL

CADRE takes the position that lifestyle modifications affecting diet and exercise are likely to be effective in preventing progression to clinical diabetes in these patients, as indicated by the Diabetes Prevention Program (DPP) study, the Da Qing IGT and Diabetes Study, and the Finnish Study.<sup>10-12</sup>

The goals of treatment for IFG/IGT include:

- ▲ Lowering glycemia to normal ranges

- ▲ Weight loss, if needed
- ▲ Lowering blood pressure if hypertension is present
- ▲ Monitoring patients for deterioration in these or other risk indicators

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