CONSENSUS STATEMENT

Management of Hyperglycemia in Type 2 Diabetes: A Consensus Algorithm for the Initiation and Adjustment of Therapy

A consensus statement from the American Diabetes Association and the European Association for the Study of Diabetes

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The epidemic of type 2 diabetes in the latter part of the 20th and in the early 21st century, and the recognition that achieving specific glycemic goals can substantially reduce morbidity, have made the effective treatment of hyperglycemia a top priority (1–3). While the management of hyperglycemia, the hallmark metabolic abnormality associated with type 2 diabetes, has historically had center stage in the treatment of diabetes, therapies directed at other coincident features, such as dyslipidemia, hypertension, hypercoagulability, obesity, and insulin resistance, have also been a major focus of research and therapy. Maintaining glycemic levels as close to the nondiabetic range as possible has been demonstrated to have a powerful beneficial impact on diabetes-specific complications, including retinopathy, nephropathy, and neuropathy in the setting of type 1 diabetes (4,5); in type 2 diabetes, more intensive treatment strategies have likewise been demonstrated to reduce complications (6–8). Intensive glycemic management resulting in lower HbA_{1c} (A1C) levels has also been shown to have a beneficial effect on cardiovascular disease (CVD) complications in type 1 diabetes (9,10); however, the role of intensive diabetes therapy on CVD in type 2 diabetes remains under active investigation (11,12). Some therapies directed at lowering glucose levels have additional benefits with regard to CVD risk factors, while others lower glucose without additional benefits.

The development of new classes of blood glucose–lowering medications to supplement the older therapies, such as lifestyle-directed interventions, insulin, sulfonylureas, and metformin, has increased the treatment options for type 2 diabetes. Whether used alone or in combination with other blood glucose–lowering interventions, the availability of the newer agents has provided an increased number of choices for practitioners and patients and heightened uncertainty regarding the most appropriate means of treating this widespread disease. Although numerous reviews on the management of type 2 diabetes have been published in recent years (13–16), practitioners are often left without a clear pathway of therapy to follow. We developed the following consensus approach to the management of hyperglycemia in the nonpregnant adult to help guide health care providers in choosing the most appropriate interventions for their patients with type 2 diabetes.

Process

The guidelines and algorithm that follow are based on clinical trials that have examined different modalities of therapy of type 2 diabetes and on the authors' clinical experience and judgment, keeping in mind the primary goal of achieving and maintaining glucose levels as close to the

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Abbreviations: CVD, cardiovascular disease; DCCT, Diabetes Control and Complications Trial; GLP-1, glucagon-like peptide 1; SMBG, self-monitoring of blood glucose; TZD, thiazolidinedione; UKPDS, U.K. Prospective Diabetes Study.

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Table 1—*Summary of antidiabetic interventions as monotherapy*

Interventions	Expected decrease in A1C (%)	Advantages	Disadvantages
Step 1: initial			
Lifestyle to decrease weight and increase activity	1–2	Low cost, many benefits	Fails for most in 1st year
Metformin	1.5	Weight neutral, inexpensive	GI side effects, rare lactic acidosis
Step 2: additional therapy			
Insulin	1.5–2.5	No dose limit, inexpensive, improved lipid profile	Injections, monitoring, hypoglycemia, weight gain
Sulfonylureas	1.5	Inexpensive	Weight gain, hypoglycemia*
TZDs	0.5–1.4	Improved lipid profile	Fluid retention, weight gain, expensive
Other drugs			
α-Glucosidase inhibitors	0.5–0.8	Weight neutral	Frequent GI side effects, three times/day dosing, expensive
Exenatide	0.5–1.0	Weight loss	Injections, frequent GI side effects, expensive, little experience
Glinides	1–1.5†	Short duration	Three times/day dosing, expensive
Pramlintide	0.5–1.0	Weight loss	Injections, three times/day dosing, frequent GI side effects, expensive, little experience

*Severe hypoglycemia is relatively infrequent with sulfonylurea therapy. The longer-acting agents (e.g. chlorpropamide, glyburide [glibenclamide], and sustained-release glipizide) are more likely to cause hypoglycemia than glipizide, glimepiride, and gliclazide. †Repaglinide is more effective at lowering A1C than nateglinide. GI, gastrointestinal.

nondiabetic range as possible. The paucity of high-quality evidence in the form of clinical trials that directly compare different diabetes treatment regimens remains a major impediment to recommending one class of drugs, or a particular combination of therapies, over another. While the algorithm that we propose is likely to engender debate, we hope that the recommendations will help guide the therapy of type 2 diabetes and result in improved glycemic control and health status over time.

Glycemic goals of therapy

Controlled clinical trials, such as the Diabetes Control and Complications Trial (DCCT) (4) and the Stockholm Diabetes Intervention Study (5) in type 1 diabetes and the U.K. Prospective Diabetes Study (UKPDS) (6,7) and Kumamoto Study (8) in type 2 diabetes, have helped to establish the glycemic goals of therapy that result in improved long-term outcomes. Although the various clinical trials have had different designs, interventions, and measured outcomes, the trials, in concert with epidemiologic data (17,18), support decreasing glycemia as an effective means of reducing long-term microvascular and neuropathic complications. The most appropriate target levels for blood glucose, on a day-to-day basis, and A1C, as an index of chronic glycemia, have not been systematically studied. However, both the DCCT (4) and the UKPDS (6,7) had as their goals the achievement of glycemic levels in the nondiabetic range. Neither study was able to sustain A1C levels in the nondiabetic range in their intensive-treatment groups, achieving mean levels over time of ~7%, 4 SDs above the nondiabetic mean.

The most recent glycemic goal recommended by the American Diabetes Association, selected on the basis of practicality and the projected reduction in complications over time, is "in general" an A1C level <7% (19). For "the individual patient," the A1C should be "as close to normal (<6%) as possible without significant hypoglycemia." The most recent glycemic goal set by the European Union–International Diabetes Federation is an A1C level <6.5%. The upper limit of the nondiabetic range is 6.1% (mean A1C of 5% + 2 SD) with the DCCT-standardized assay, which has been promulgated through the National Glycohemoglobin Standardization Program (NGSP) and adopted by the vast majority of commercially available assays (20). Our consensus is that an A1C of ≥7% should serve as a call to action to initiate or change therapy with the goal of achieving an A1C level as close to the nondiabetic range as possible or, at a minimum, decreasing the A1C to <7%. We are mindful that this goal is not appropriate or practical for some patients, and clinical judgment, based on the potential benefits and risks of a more intensified regimen, needs to be applied for every patient. Factors such as life expectancy and risk for hypoglycemia need to be considered for every patient before intensifying therapeutic regimens.

Assiduous attention to abnormalities other than hyperglycemia that accompany type 2 diabetes, such as hypertension and dyslipidemia, has been shown to improve microvascular and cardiovascular complications. Readers are referred to published guidelines for a discussion of the rationale and goals of therapy for the nonglycemic risk factors, as well as recommendations as to how to achieve them (1,21,22).

Principles in selecting antihyperglycemic interventions

Choosing specific antihyperglycemic agents is predicated on their effectiveness in lowering glucose, extraglycemic effects that may reduce long-term complications, safety profiles, tolerability, and expense.

Effectiveness in lowering glycemia. Apart from their differential effects on glycemia, there are insufficient data at this time to support a recommendation of one class of glucose-lowering agents, or one combination of medications, over others with regard to effects on complications. In other words, the salutary effects of therapy on long-term complications appear to

be predicated predominantly on the level of glycemic control achieved rather than on any other specific attributes of the intervention(s) used to achieve glycemic goals. The UKPDS compared three classes of glucose-lowering medications (sulfonylurea, metformin, or insulin) but was unable to demonstrate clear superiority of any one drug over the others with regard to complications (6,7). However, the different classes do have variable effectiveness in decreasing glycemic levels (Table 1), and the overarching principle in selecting a particular intervention will be its ability to achieve and maintain glycemic goals. In addition to the intention-to-treat analyses demonstrating the superiority of intensive versus conventional interventions, the DCCT and UKPDS demonstrated a strong correlation between mean A1C levels over time and the development and progression of retinopathy and nephropathy (23,24). Therefore, we think it is reasonable to judge and compare blood glucose–lowering medications, and the combinations of such agents, primarily on the basis of the A1C levels that are achieved and on their specific side effects, tolerability, and expense.

Nonglycemic effects of medications. In addition to variable effects on glycemia, specific effects of individual therapies on CVD risk factors, such as hypertension or dyslipidemia, were also considered important. We also included the effects of interventions that may benefit or worsen the prospects for long-term glycemic control in our recommendations. Examples of these would be changes in body mass, insulin resistance, or insulin secretory capacity in type 2 diabetic patients.

Choosing specific diabetes interventions and their roles in treating type 2 diabetes

Numerous reviews have focused on the characteristics of the specific diabetes interventions listed below (25–33). The aim here is to provide enough information to justify the choices of medications, the order in which they are recommended, and the utility of combinations of therapies. Unfortunately, there is a dearth of high-quality studies that provide head-to-head comparisons of the ability of the medications to achieve the currently recommended glycemic levels. The authors highly recommend that such studies be conducted. However, even in the absence of rigorous, comprehensive studies that directly compare the efficacy of all available glucose-lowering treatments, and their combinations, we feel that there are enough data regarding the characteristics of the individual interventions to provide the guidelines below.

An important intervention that is likely to improve the probability that a patient will have better long-term control of diabetes is to make the diagnosis early, when the metabolic abnormalities of diabetes are usually less severe. Lower levels of glycemia at time of initial therapy are associated with lower A1C over time and decreased long-term complications (34).

Lifestyle interventions. The major environmental factors that increase the risk of type 2 diabetes, presumably in the setting of genetic risk, are overnutrition and a sedentary lifestyle, with consequent overweight and obesity (35). Not surprisingly, interventions that reverse or improve these factors have been demonstrated to have a beneficial effect on control of glycemia in established type 2 diabetes (36). While there is still active debate regarding the most beneficial types of diet and exercise, weight loss almost always improves glycemic levels. Unfortunately, the high rate of weight regain has limited the role of lifestyle interventions as an effective means of controlling glycemia long term. The most convincing long-term data that weight loss effectively lowers glycemia have been generated in the follow-up of type 2 diabetic patients who have had bariatric surgery (37,38). In this setting, diabetes is virtually erased, with a mean sustained weight loss of >20 kg (37,38). Studies of the pharmacologic treatment of obesity have been characterized by high drop-out rates, low sustainability, and side effects; weight loss medications cannot be recommended as a primary therapy for diabetes at this time. In addition to the beneficial effects of weight loss on glycemia, weight loss and exercise improve coincident CVD risk factors, such as blood pressure and atherogenic lipid profiles, and ameliorate other consequences of obesity (37–40). There are few adverse consequences of such lifestyle interventions other than the difficulty in incorporating them into usual lifestyle and sustaining them and the usually minor musculoskeletal injuries and potential problems associated with neuropathy, such as foot trauma and ulcers, that may occur with increased activity. Theoretically, effective weight loss, with its pleiotropic benefits, safety profile, and low cost, should be the most cost-effective means of controlling diabetes, if it could be achieved and maintained long term.

Given these beneficial effects, a lifestyle intervention program to promote weight loss and increase activity levels should, with rare exceptions, be included as part of diabetes management. The beneficial effects of such programs are usually seen rapidly, within weeks to months, and often before there has been substantial weight loss (41). Weight loss of as little as 4 kg will often ameliorate hyperglycemia. However, the limited long-term success of lifestyle programs to maintain glycemic goals in patients with type 2 diabetes suggests that a large majority of patients will require the addition of medications over the course of their diabetes.

Medications. The characteristics of currently available antidiabetic interventions, when used as monotherapy, are summarized in Table 1. The glucose-lowering effectiveness of individual therapies and combinations demonstrated in clinical trials is predicated not only on the intrinsic characteristics of the intervention, but also on the baseline glycemia, duration of diabetes, previous therapy, and other factors. A major factor in selecting a class of drugs, or a specific medication within a class, to initiate therapy or when changing therapy, is the ambient level of glycemic control. When levels of glycemia are high (e.g., A1C >8.5%), classes with greater and more rapid glucose-lowering effectiveness, or potentially earlier initiation of combination therapy, are recommended; conversely, when glycemic levels are closer to the target levels (e.g., A1C <7.5%), medications with lesser potential to lower glycemia and/or a slower onset of action may be considered. Obviously, the choice of glycemic goals and the medications used to achieve them must be individualized for each patient, balancing the potential for lowering A1C and anticipated long-term benefit with specific safety issues, as well as other characteristics of regimens, including side effects, tolerability, patient burden and long-term adherence, expense, and the nonglycemic effects of the medications. Finally, type 2 diabetes is a progressive disease with worsening glycemia over time. Therefore, addition of medications is the rule, not the exception, if treatment goals are to be met over time.

Metformin. Metformin is the only biguanide available in most of the world. Its major effect is to decrease hepatic glucose output and lower fasting glycemia. Typi-

cally, metformin monotherapy will lower A1C by ~1.5 percentage points (27,42). It is generally well tolerated, with the most common adverse effects being gastrointestinal. Although always a matter of concern because of its potentially fatal outcome, lactic acidosis is quite rare (<1 case per 100,000 treated patients) (43). Metformin monotherapy is usually not accompanied by hypoglycemia and has been used safely, without causing hypoglycemia, in patients with pre-diabetic hyperglycemia (44). The major nonglycemic effect of metformin is either weight stability or modest weight loss, in contrast to many of the other blood glucose–lowering medications. The UKPDS demonstrated a beneficial effect of metformin therapy on CVD outcomes that needs to be confirmed (7).

Sulfonylureas. Sulfonylureas lower glycemia by enhancing insulin secretion. They appear to have an effect similar to metformin, and they lower A1C by ~1.5 percentage points (26). The major adverse side effect is hypoglycemia, but severe episodes, characterized by need for assistance, coma, or seizure, are infrequent. However, such episodes are more frequent in the elderly. Episodes can be both prolonged and life threatening, although these are very rare. Several of the newer sulfonylureas have a relatively lower risk for hypoglycemia (Table 1) (45,46). In addition, weight gain of ~2 kg is common with the initiation of sulfonylurea therapy. This may have an adverse impact on CVD risk, although it has not been established. Finally, sulfonylurea therapy was implicated as a potential cause of increased CVD mortality in the University Group Diabetes Program (47). Concerns raised by the University Group Diabetes Program study that sulfonylurea therapy may increase CVD mortality in type 2 diabetes were not substantiated by the UKPDS (6).

Glinides. Like the sulfonylureas, the glinides stimulate insulin secretion, although they bind to a different site within the sulfonylurea receptor (28). They have a shorter circulating half-life than the sulfonylureas and must be administered more frequently. Of the two glinides currently available in the U.S., repaglinide is almost as effective as metformin or the sulfonylureas, decreasing A1C by ~1.5 percentage points. Nateglinide is somewhat less effective in lowering A1C than repaglinide when used as monotherapy or in combination therapy (48,49). The glinides have a similar risk for weight gain as the sulfonylureas, but hypoglycemia may be less frequent, at least with nateglinide, than with some sulfonylureas (49,50).

α-Glucosidase inhibitors. α-Glucosidase inhibitors reduce the rate of digestion of polysaccharides in the proximal small intestine, primarily lowering postprandial glucose levels without causing hypoglycemia. They are less effective in lowering glycemia than metformin or the sulfonylureas, reducing A1C by 0.5–0.8 percentage points (29). Since carbohydrate is absorbed more distally, malabsorption and weight loss do not occur; however, increased delivery of carbohydrate to the colon commonly results in increased gas production and gastrointestinal symptoms. This side effect has led to discontinuation of the α-glucosidase inhibitors by 25–45% of participants in clinical trials (29,51). One clinical trial examining acarbose as a means of preventing the development of diabetes in high-risk subjects with impaired glucose tolerance showed an unexpected reduction in severe CVD outcomes (51). This potential benefit of α-glucosidase inhibitors needs to be confirmed.

Thiazolidinediones. Thiazolidinediones (TZDs or glitazones) are peroxisome proliferator–activated receptor γ modulators; they increase the sensitivity of muscle, fat, and liver to endogenous and exogenous insulin ("insulin sensitizers") (31). The limited data regarding the blood glucose–lowering effectiveness of TZDs when used as monotherapy have demonstrated a 0.5–1.4% decrease in A1C. The most common adverse effects with TZDs are weight gain and fluid retention. There is an increase in adiposity, largely subcutaneous, with redistribution of fat from visceral deposits shown in some studies. The fluid retention usually manifests as peripheral edema, though new or worsened heart failure can occur. The TZDs either have a beneficial or neutral effect on atherogenic lipid profiles, with pioglitazone having a more beneficial effect than rosiglitazone (52,53). The PROactive (PROspective pioglitAzone Clinical Trial In macroVascular Events) study demonstrated no significant effects of pioglitazone compared with placebo on the primary CVD outcome (composite of all-cause mortality, nonfatal and silent myocardial infarction, stroke, major leg amputation, acute coronary syndrome, coronary artery bypass graft or percutaneous coronary intervention, and leg revascularization) after 3 years of follow-up, but a 16% reduction in death, myocardial infarction, and stroke, a secondary end point, was reported with marginal statistical significance (54).

Insulin. Insulin is the oldest of the currently available medications and has the most clinical experience. Although initially developed to treat the insulin-deficient type 1 diabetic patient, in whom it is life saving, insulin was used early on to treat the insulin-resistant form of diabetes recognized by Himsworth and Kerr (55). Insulin is the most effective of diabetes medications in lowering glycemia. It can, when used in adequate doses, decrease any level of elevated A1C to, or close to, the therapeutic goal. Unlike the other blood glucose–lowering medications, there is no maximum dose of insulin beyond which a therapeutic effect will not occur. Relatively large doses of insulin (≥1 unit/kg), compared with those required to treat type 1 diabetes, may be necessary to overcome the insulin resistance of type 2 diabetes and lower A1C to goal. Although initial therapy is aimed at increasing basal insulin supply, usually with intermediate- or long-acting insulins, patients may also require prandial therapy with short- or rapid-acting insulins as well (Fig. 1). Insulin therapy has beneficial effects on triglyceride and HDL cholesterol levels (56) but is associated with weight gain of ~2–4 kg, probably proportional to the correction of glycemia and owing predominantly to the reduction of glycosuria. As with sulfonylurea therapy, the weight gain may have an adverse effect on cardiovascular risk. Insulin therapy is also associated with hypoglycemia, albeit much less frequently than in type 1 diabetes. In clinical trials aimed at normoglycemia and achieving a mean A1C of ~7%, severe hypoglycemic episodes (defined as requiring help from another person to treat) occurred at a rate of between 1 and 3 per 100 patient-years (8,56–59) compared with 61 per 100 patient-years in the DCCT intensive-therapy group (4). Insulin analogs with longer, nonpeaking profiles may decrease the risk of hypoglycemia compared with NPH, and analogs with very short durations of action may reduce the risk of hypoglycemia compared with regular insulin (60,61). Inhaled insulin was approved in the U.S. in 2006 for the treatment of type 2 diabetes. Published clinical studies to date have not demonstrated whether inhaled insulin, given as monotherapy (62,63) or in combination with an injection of long-acting insulin (64), can lower A1C to ≤7%.

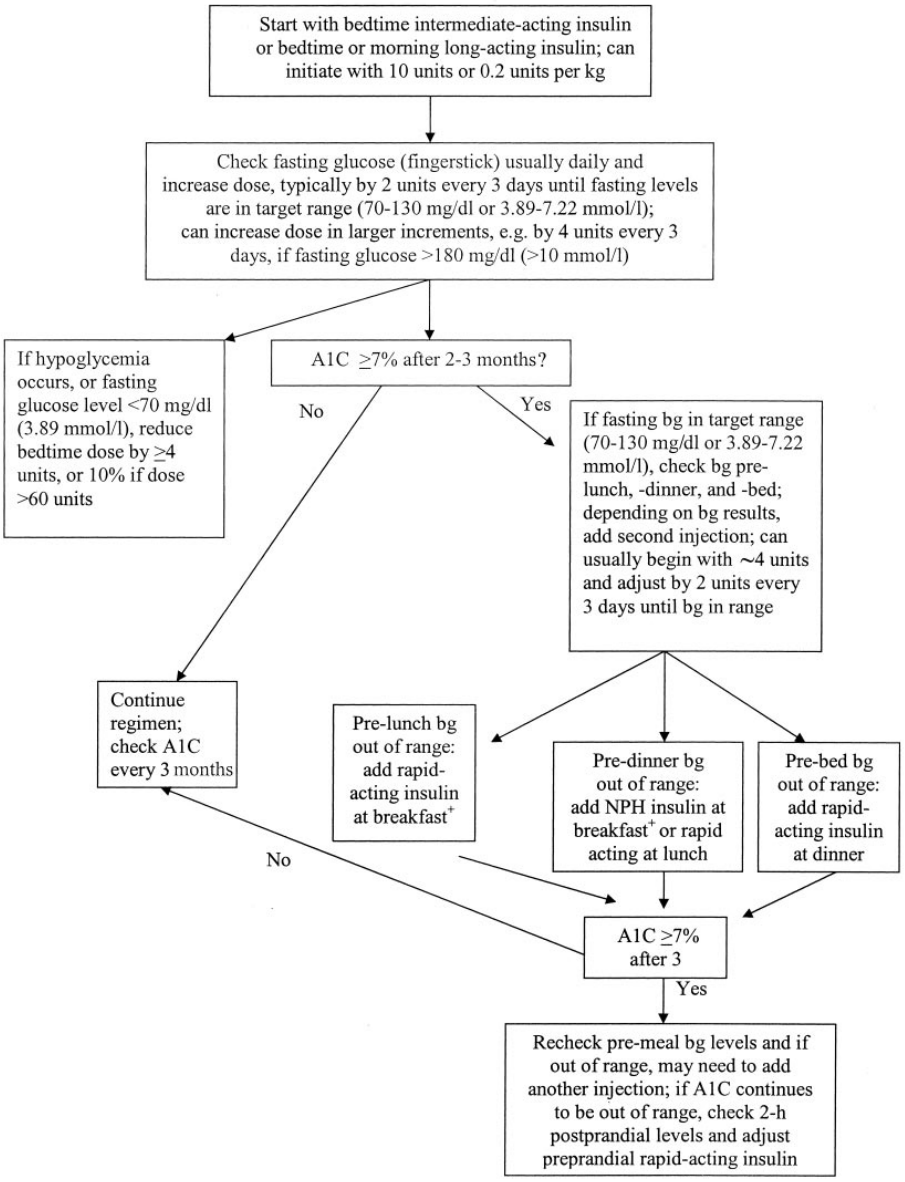

Figure 1—*Initiation and adjustment of insulin regimens. Insulin regimens should be designed taking lifestyle and meal schedule into account. The algorithm can only provide basic guidelines for initiation and adjustment of insulin. See ref. 71 for more detailed instructions. +Premixed insulins are not recommended during adjustment of doses; however, they can be used conveniently, usually before breakfast and/or dinner if proportion of rapid- and intermediate-acting insulins is similar to the fixed proportions available. bg, blood glucose.*

Glucagon-like peptide 1 agonists (exenatide). Glucagon-like peptide 1 (GLP-1) 7-37, a naturally occurring peptide produced by the L-cells of the small intestine, stimulates insulin secretion. Exendin-4 has homology with the human GLP-1 sequence but has a longer circulating half-life. It binds avidly to the GLP-1 receptor on the pancreatic β-cell and potentiates glucose-mediated insulin secretion (32). Synthetic exendin-4 (exenatide) was approved for use in the U.S. in 2005 and is administered twice per day by subcutaneous injection. Although there are far less published data on this new compound than the other blood glucose–lowering medications, exendin-4 appears to lower A1C by 0.5–1 percentage points, mainly by lowering postprandial blood glucose levels (65–68). Exenatide also suppresses glucagon secretion and slows gastric motility. It is not associated with hypoglycemia but has a relatively high frequency of gastrointestinal side effects, with 30–45% of treated patients experiencing one or more episodes of nausea, vomiting, or diarrhea

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