IMPROVING GLYCEMIC CONTROL IN HOSPITALIZED PATIENTS:

Strategies to Enhance Inpatient Care and Facilitate Outpatient Transitions

A CLINICAL MEDICINE TODAY DIGITAL NEWSLETTER

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Target Audience
This newsletter is intended for primary healthcare professionals, including physicians, physician assistants, nurse practitioners, nurses, and pharmacists, as well as other specialists and healthcare providers who are interested in the management of patients with diabetes.

Series Overview/Statement of Need
Diabetes mellitus is a serious and costly disease that affects an estimated 20.8 million individuals, or 7% of the United States (US) population. It is estimated that the number of individuals in the US with diagnosed diabetes will increase by 165% from 2000 to 2050.

Despite widespread agreement among physicians about guidelines for the care of diabetes, treatment is often suboptimal. Compelling evidence suggests that poorly controlled glucose levels are associated with increased morbidity, mortality, and higher costs. The longer diabetes is uncontrolled, the greater the risk of developing vascular complications, retinopathy, end-stage renal disease, and neuropathy. Studies have found that improved glycemic control benefits patients, but, until recently, this has not been a major therapeutic focus.

In recent years, several position statements and practice guidelines have emphasized the importance of implementing adequate glycemic goals for hospitalized patients and maintaining those goals as patients are discharged from the inpatient setting. Large prospective studies have demonstrated that intensive treatment of diabetes can decrease the chronic complications of the disease. The availability of new insulin analogues should assist primary care physicians and their patients with diabetes in achieving the goal of physiologic insulin replacement and near-normal glucose levels.

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LEARNING OBJECTIVES

Upon completion, participants should be able to:

1. **Discuss the physiologic effects and clinical sequela of poorly controlled glycemia in hospitalized patients**
2. **Identify recommended glycemic targets and apply strategies for reaching and maintaining these targets in the hospital setting**
3. **Design individualized discharge plans for patients who are transitioning from inpatient to outpatient care**

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NEWSLETTER FACULTY

ADVISER
Jack L. Leahy, MD
Chief, Division of Endocrinology, Diabetes and Metabolism
Professor of Medicine
University of Vermont College of Medicine
Burlington, Vermont

AUTHOR
Terry Glauser, MD, MPH
Consulting Physician
Medical Education Solutions Group (MESG)
Baltimore, Maryland

This Clinical Medicine Today newsletter is published by:

Medical Education Solutions Group (MESG)
5523 Research Park Drive
Suite 220
Baltimore, MD 21228

INTRODUCTION

Diabetes mellitus (DM) is classified by the American Diabetes Association as "a group of metabolic diseases characterized by hyperglycemia resulting from defects in insulin secretion, insulin action, or both."[1] In the United States (US), an estimated 20.8 million individuals, or 7.0% of the population, have DM; of these, 14.6 million have been diagnosed and 6.2 million remain undiagnosed.[2] Of those who are diagnosed, 90% to 95% have type 2 DM and 5% to 10% have type 1 DM.[3] Another 54 million individuals have pre-diabetes.[2]

The lifetime risk that an individual born in the US in the year 2000 will develop DM is 38.5% for females and 32.8% for males.[4] One analysis used DM prevalence data from the year 2000 to project a 165% increase in the number of diagnosed diabetic patients by 2050.[5]

Patients in the early stages of DM may be asymptomatic, which largely accounts for the significant number of undiagnosed individuals.[6] This is of concern because chronic hyperglycemia leads to damage in a number of organs and body systems, including the eyes, kidneys, nerves, and cardiovascular system.[1,6] Outpatient hyperglycemic control is a crucial factor in decreasing these long-term complications.

In 2004, DM was the fourth most common comorbid condition noted in hospital discharge summaries.[7] DM increases the rate of hospitalization 2- to 4-fold and increases the length of hospital stay by 1 to 3 days.[7] Patients may present to the hospital with a prior diagnosis of DM or may be found to be hyperglycemic on admission. In either case, blood glucose levels should be appropriately controlled to optimize outcomes.

In this newsletter, we discuss methods for improving glycemic control in hospitalized patients with DM or newly recognized hyperglycemia. We also present strategies for transitioning these patients from in-hospital regimens to outpatient therapies that effectively keep blood glucose levels within recommended limits. In addition, we have woven a clinical vignette into the text, which is introduced below and revisited throughout the discussion to highlight important concepts.

Clinical Vignette

A 45-year-old Caucasian woman presents to the emergency department for increasing confusion. Two days ago, she saw her family physician for frequent urination and was diagnosed with a urinary tract infection (UTI). She is taking the prescribed antibiotic, but continues to experience frequent urination and is now drinking excessive fluids that "seem to go right through" her. She has a history of hypertension and takes hydrochlorothiazide 25 mg daily. She does not smoke and rarely drinks alcohol. She leads a sedentary lifestyle.

Upon examination, the patient is obese, lethargic, and somewhat confused. She has a temperature of 100.5ºF, blood pressure of 140/90 mm Hg, pulse rate of 96 beats per minute, and respirations of 24 breaths per minute. Her physical examination is remarkable for dry tongue and mucus membranes.

Her laboratory studies reveal a WBC count of 12.5 x10^9 cells/L, with normal RBC and platelet counts. Her blood glucose is 500 mg/dL, with other electrolyte values within normal limits. She has a UTI that does not appear to be responding to the initial antibiotics.

She is admitted to the hospital for blood glucose control and treatment of UTI. After her blood glucose level was reported, the admitting physician asked for a hemoglobin A_{1c} ($HbgA_{1c}$) level to be run.

THE EFFECTS OF HYPERGLYCEMIA ON HOSPITALIZED PATIENTS

Hyperglycemia caused by metabolic stress has a wide variety of adverse physiologic effects that can contribute to poor outcomes in hospitalized patients. In a recent review, Clement et al. noted that hyperglycemia[8]:

- Suppresses the immune system, increases reactive oxygen species, and increases the level of cytokines that favor inflammation
- Decreases the migration and bacterial-killing functions of white blood cells
- Leads to endothelial dysfunction
- Impairs ischemic preconditioning needed to protect the heart from insult, which can lead to increased infarct size
- Causes catecholamine elevation and blood pressure changes
- Leads to platelet abnormalities and a variety of changes in hemostasis that favor thrombosis
- Causes increased tissue acidosis and lactate levels, which lead to neurologic damage

These adverse physiologic effects take their toll on hospitalized patients who are hyperglycemic.

Critically Ill Patients

A number of studies have demonstrated the increase of in-hospital morbidity and mortality caused by hyperglycemia in critically ill medical patients.

In a meta-analysis of studies of patients admitted for an acute myocardial infarction, patients with hyperglycemia (defined as a blood glucose > 110 mg/dL) had increased in-hospital mortality and an increased incidence of congestive heart failure, regardless of whether these patients were previously diagnosed with DM.[9]

The effect of hyperglycemia on patients admitted to a combined medical-surgical intensive care unit (ICU) was examined in a retrospective review.[10] The investigators found that nonsurvivors had mean and maximum blood glucose levels that were significantly higher ($P < 0.001$) than survivors, and that in-hospital mortality increased as blood glucose levels increased.[10] Mortality was 42.5% in patients with mean blood glucose levels > 300 mg/dL.[10]

Non-Critically Ill Patients

Hyperglycemia also affects outcomes in patients who do not require intensive care. Capes et al. analyzed 32 studies that examined the relationship between acute poststroke blood glucose and outcomes.[11] Acute hyperglycemia predicted an increased risk of mortality and poor functional recovery in both diabetic and not-previously-known diabetic stroke survivors, although the nondiabetic patients fared worse. For patients with admission blood glucose > 108 mg/dL, the unadjusted relative risk of in-hospital or 30-day mortality was 3.07 in nondiabetic patients and 1.30 in diabetic patients. Nondiabetic stroke survivors with admission blood glucose > 121 mg/dL also had a greater risk of poor functional recovery (relative risk = 1.41) (data were not available for diabetic patients only), and studies of pooled relative risk of diabetic and nondiabetic patients showed that stress hyperglycemia was associated with an unadjusted relative risk of 1.07.[11]

Surgical patients who are hyperglycemic fare worse than normoglycemic patients; those with blood glucose levels > 220 mg/dL on the first postoperative day had a 5.7-fold increase in serious infections.[13]

A retrospective study examined the effect of hyperglycemia (defined as an admission or in-hospital fasting blood glucose of ≥ 126 mg/dL or a random blood glucose of ≥ 200 mg/dL on 2 or more occasions) on 2,030 patients admitted to a general hospital ward.[12] Patients with newly discovered hyperglycemia had an in-hospital mortality rate of 16% compared with patients who had a history of DM (3%; $P < 0.01$) and patients who were normoglycemic (1.7%; $P < 0.01$). Patients with newly discovered hyperglycemia had longer hospital stays, more frequent transfers to ICUs, and were more likely to be discharged to a nursing home.[12] Interestingly, this study found hyperglycemia in 38% of patients admitted to the hospital, with one-third having no history of DM.[12]

Surgical Patients

Surgical patients who are hyperglycemic fare worse than their normoglycemic counterparts as well. A prospective study evaluated 100 patients with DM who had elective surgery and did not have any infections prior to surgery.[13] After surgery, diabetic patients with blood glucose levels > 220 mg/dL on the first postoperative day had a 2.7-fold increase in infections.[13] When UTIs were excluded, these patients had a 5.7-fold increase in serious infections.[13]

BETTER GLYCEMIC CONTROL, BETTER OUTCOMES

A number of recent interventional studies have demonstrated that attaining and maintaining blood glucose levels between 80 mg/dL and 110 mg/dL in the hospital leads to improved clinical outcomes.[14]

One of the early studies to examine the effects of treating hyperglycemia in hospitalized patients was conducted by van den Berghe et al. in 2001—and the results were dramatic.[15] This prospective, randomized, controlled study enrolled 1,548 patients admitted to a surgical ICU, many of whom had not previously been diagnosed with DM.[15] Patients were randomized to one of two groups:

- Intensive insulin therapy—started if a single blood glucose level exceeded 110 mg/dL—with the goal of keeping blood glucose levels between 80 mg/dL and 110 mg/dL, or
- Conventional therapy of continuous insulin infusion—started only if blood glucose levels exceeded 215 mg/dL—with the goal of keeping blood glucose levels between 180 mg/dL and 200 mg/dL.[15]

Mortality for patients randomized to intensive insulin was 4.6% compared with 8.0% for patients treated conventionally ($P < 0.04$), with the greatest reduction involving deaths due to multiple-organ failure with sepsis.[15] Significantly more patients treated conventionally remained in the ICU for more than 5 days (20.2% vs. 10.6% of intensive insulin patients, $P = 0.005$). Intensive insulin therapy led to a 34% reduction in in-hospital mortality, a 46% decrease in bloodstream infections, a 41% reduction in acute renal failure requiring dialysis or hemofiltration, a 50% drop in the median number of red-cell transfusions, and a 44% reduction in critical-illness polyneuropathy.[15]

These striking results led the authors to perform a similar prospective, randomized, controlled study with patients in a medical ICU.[16] In this study, 1,200 patients, many of whom had not been previously diagnosed with DM, were randomized to either:

A FRONT-LINE PERSPECTIVE

The 1990s saw proof that intensive outpatient blood glucose management for type 1 and type 2 diabetes results in a marked reduction in microvascular complications. When combined with aggressive blood pressure and lipid control, macrovascular disease is also prevented to a major extent. As a result, we have national standards of care for the outpatient setting that are well known to most practitioners. However, until recently, there has been little agreement about the importance of blood glucose control for hospitalized patients. This is now changing, in part because of a growing number of papers that show improved outcomes for hospitalized patients when blood glucose levels are kept within the target range. Also, the newer analogue insulins and subcutaneous dosing protocols that are being explored at various institutions (such as those included in this monograph from my hospital, the University of Vermont) are increasingly making it possible to control blood glucose levels—safely—in most patients.

Many hospitals are developing subcutaneous dosing protocols, educating staff about appropriate blood glucose management, and putting specialty diabetes management teams in place for hard-to-manage patients. One hopes we will see the same focus on the importance of diabetes management in the hospital as typically occurs outside the hospital. Our patients will be the winners.

Jack L. Leahy, MD
Chief, Division of Endocrinology, Diabetes and Metabolism
Professor of Medicine
University of Vermont College of Medicine
Burlington, Vermont

- Intensive insulin infusion—started when blood glucose levels exceeded 110mg/dL—with the goal of keeping blood glucose levels between 80 mg/dL and 110 mg/dL, or
- Conventional therapy of insulin infusion—started when blood glucose levels exceeded 215 mg/dL and then tapered when levels dropped below 180 mg/dL — with the goal of keeping blood glucose levels between 180 mg/dL and 200 mg/dL.[16]

In this setting, intensive insulin therapy reduced blood glucose levels, but did not significantly reduce in-hospital mortality when compared with conventional therapy (37.3% vs. 40.0% respectively, $P = 0.33$).[16] However, the intensive insulin therapy led to a significant reduction in morbidity because it prevented renal injury, hastened weaning from mechanical ventilation, and allowed patients to be discharged from both the ICU and the hospital more rapidly.[16]

Other studies with varying designs (meta-analyses, retrospective cohort studies, and randomized clinical trials) have demonstrated that patients who receive intensive insulin therapy to achieve and maintain normal blood glucose levels experience reduced in-hospital mortality, mortality at 1 year, mortality at 3 years, and morbidity.[14]

STRATEGIES FOR IN-HOSPITAL TREATMENT

The goal of in-hospital blood glucose management is to provide patients with therapy that reflects normal physiology while maintaining a maximum blood glucose level of 110 mg/dL for patients in the ICU, a maximum preprandial blood glucose level of 110 mg/dL for patients in non-critical care units, and a maximum blood glucose level at any time of 180 mg/dL for patients in non-critical care units (Table 1).[7]

TABLE 1. Recommended Glycemic Targets for Inpatients

Inpatient	Target
In the intensive care unit	110 mg/dL (6.1 mmol/L)
In non-critical care units: preprandial	110 mg/dL (6.1 mmol/L)
In non-critical care units: maximum	180 mg/dL (10.0 mmol/L)

Data derived from American College of Endocrinology. Position statement on inpatient diabetes and metabolic control. *Endocr Pract*. 2004;10(1):77-82.

In normal individuals, insulin secretion by pancreatic β cells consists of basal insulin secretion (relatively constant throughout the whole day and night) and a prandial insulin surge (Figure 1). Newer insulin preparations have been developed with altered amino acid structures that provide a specific absorption and kinetic profile.[17] These synthetic insulins, called insulin analogues, have a similar ability to lower blood glucose as human insulins, but they more closely reflect physiologic insulin secretion because of their absorption and kinetic profiles.[17]

Insulins can be differentiated by time to onset of action, time to peak effect, and duration of effect (Figure 2). Long-acting insulin analogues are used to control basal blood glucose, while rapid-acting analogues are use to control postprandial blood glucose surges.[17]

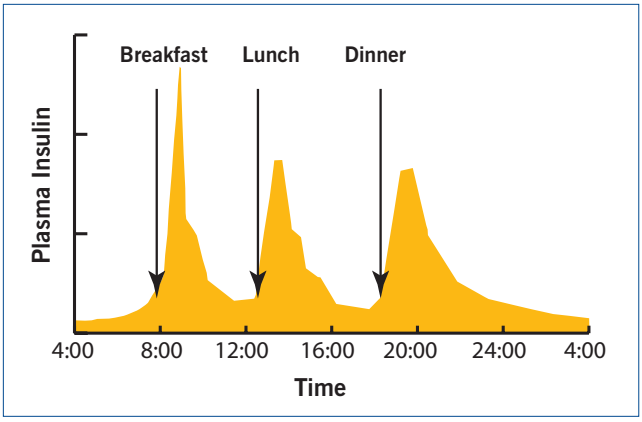

FIGURE 1. Physiologic Insulin Secretion. The 24-hour insulin profile of a healthy patient eating 3 meals a day shows 2 distinct patterns. The lower pattern of secretion (basal) is relatively constant throughout the day and the middle portion of the graph shows fast-peaking mealtime spikes (bolus).

Long-Acting Insulin

Long-acting insulin analogues are used once or twice daily, and include insulin glargine and detemir. They are often the only insulin needed by patients who are unable to eat or NPO, and it is easy to transition the use of these agents from an in-hospital to an outpatient setting. They are increasingly being used instead of NPH, a human insulin with a rising and falling pattern of action (a so-called "peaking" insulin) that does not well match the physiologic pattern of basal insulin secretion.

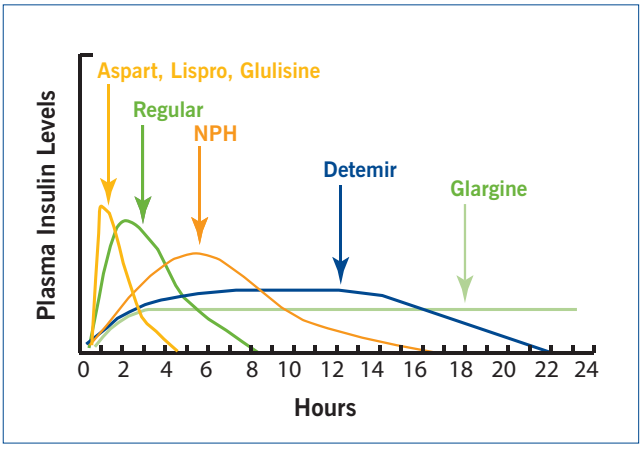

FIGURE 2. Profiles of Human Insulins and Insulin Analogues. There are several different types of insulin available, with varying onsets and durations of action, that can be used alone or in combination to improve glycemic control in patients.

Short-Acting Insulin

Short-acting (or rapid-acting) insulin analogues include aspart, lispro, and glulisine. Unlike regular insulin, which should be taken at least 30 minutes before eating, these analogues do not have to be taken before a meal. Instead, they reach peak effect quickly and can, therefore, be taken when patients begin to eat, or even after they have already started to eat (glulisine can be taken up to 20 minutes after beginning a meal), allowing greater flexibility in patient schedules.[18] This flexibility is quite valuable in the hospital

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because patients may be taken for X-rays, testing, or therapy without prior notice. Short-acting insulin analogues may, therefore, be used to prevent the potential situation where patients receive insulin and are then taken for a test before they can eat—a situation that can lead to symptomatic hypoglycemia. Moreover, waiting to administer insulin until patients have had their meal trays for several minutes can be extremely helpful in choosing the appropriate insulin dose for patients whose intake may still be variable because of situations such as nausea and recent surgery.

The rapid onset of action of short-acting insulin analogues is useful in treating patients who have high blood glucose levels, as they can help lower levels rapidly. These agents also carry a lower risk of late postprandial hypoglycemia than regular insulin.[18]

Tailoring the Treatment Plan

Patient insulin programs should be individualized and should match eating status. Sliding-scale insulin therapy has been shown to lead to suboptimal glycemic control and, therefore, its use is discouraged.[7,19] Indeed, the American College of Endocrinology's 2004 position statement advises, "Insulin, given either intravenously as a continuous infusion or subcutaneously, is currently the only available agent for effectively controlling glycemia in the hospital."[7]

Patients Who Are Able to Eat. Patients who are able to eat regularly should be treated with a long-acting insulin to meet their basal needs and a short-acting insulin at mealtime to cover postprandial blood glucose increases.[20] Specific dosing requires consideration of comorbid disorders, however, and the long-acting insulin analogue given to address basal needs must be adjusted accordingly.[20] For the elderly, patients with poor renal function, or those with significant cardiac or hepatic disease, treatment is typically begun by using a lower dose of long-acting insulin analogue.[20] In turn, for patients who are overweight, have metabolic syndrome, have an infection, have had coronary artery bypass surgery (CABG), or have open wounds, it is appropriate to prescribe a higher dose of long-acting insulin analogue.[20]

An exception to the use of basal-prandial insulin is when patients are receiving prednisone. It is well known that glucocorticoids increase glucose production, suppress insulin secretion, and inhibit glucose uptake, thereby promoting hyperglycemia.[8] However, the typical pattern for patients taking a single high dose of prednisone in the morning is a large rise in glycemia at supper and bedtime as opposed to throughout the entire 24 hours. This pattern of glucose elevation is often well matched by the profile of NPH insulin (Figure 3) and for this reason, it is recommended that patients on prednisone be treated with a dose of NPH insulin in the morning. Alternatively, where available, an intravenous insulin infusion that is adjusted hourly to a target glucose level can be effective as well.

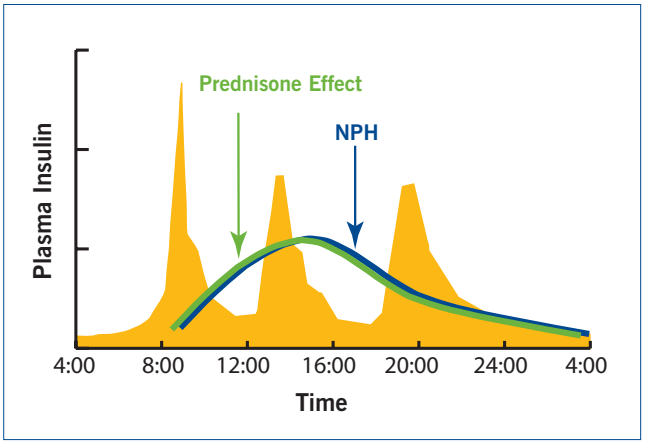

FIGURE 3. Prednisone Effect on Insulin Secretion. Hospitalized patients who are receiving once-daily prednisone (in the morning) typically experience a large rise in blood glucose levels at supper and bedtime, as opposed to throughout a 24-hour period. NPH can often well match this pattern of glucose elevation.

Clinical Vignette (cont.)

The patient is admitted to the hospital. She is lethargic, but able to eat. The admitting physician initially decides to lower her blood glucose level from 500 mg/dL to 200 mg/dL. He starts the patient on 40 U insulin glargine (she weighs 80 kg) and administers an additional 8 U insulin aspart immediately. He decides that this is the dose of aspart she will receive with meals. He also prescribes a different antibiotic for her UTI and orders another blood glucose test to be administered in 6 hours. Her $HbgA_{1c}$ level comes back at 8%.

One suggested dosing protocol, used at the University of Vermont College of Medicine, is to begin calculating starting insulin doses as follows[20]:

- Basal insulin (glargine at bedtime or twice-daily NPH):
  - 0.5 U/kg for patients without the concerns listed below (which makes up most patients)
  - 0.3 U/kg if concerned about a high risk of hypoglycemia because of renal, cardiac, or hepatic dysfunction, gastroparesis, or older age
  - 0.7 U/kg for states of presumed high insulin resistance such as obesity, metabolic syndrome, infections, open wounds, or post CABG
- Mealtime (lispro, aspart, glulisine): 0.1 U/kg with meals (adjust down to 0.05 U/kg or administer after meal for inconsistent eaters)
- Supplemental lispro/aspart/glulisine:
  - Blood glucose between 200 and 299 mg/dL: give an extra 0.075 U/kg (¾ of usual meal dose)
  - Blood glucose > 300 mg/dL: give an extra 0.1 U/kg (usual meal dose)

This regimen is adjusted by obtaining blood glucose levels at mealtime and bedtime and adjusting the glargine doses to attain fasting blood glucose < 110 mg/dL. Mealtime short-acting insulin analogue doses should be adjusted to achieve preprandial and bedtime blood glucose levels of 110 mg/dL to 180 mg/dL.[20]

An additional useful strategy for insulin dosing in patients who are already being managed with a basal-bolus insulin program is called the "Rule of 1800." This rule can help determine the additional number of units of short-acting insulin needed to lower blood glucose to the target value. This calculation helps estimate the individualized drop (in mg/dL) per unit of short-acting insulin analogue for a particular patient (note that for regular insulin, 1500 can be substituted for 1800). The calculation is:

$$\frac{1800}{\text{total daily dose of insulin in units (both long- and short-acting)}} = \text{mg/dL decrease for each unit given}$$

Clinical Vignette (cont.)

By the second hospital day, the patient has defervesced and is much less lethargic. Her blood glucose has responded well to the glargine and aspart insulin doses that were originally chosen. However, her blood glucose level before lunch is 325 mg/dL. To lower her blood glucose to the desired level of < 180 mg/dL, the physician decides to give her additional short-acting insulin. To calculate how much additional insulin he needs to give her, he uses the Rule of 1800 as follows:

- She is currently receiving 40 U glargine daily and 8 U aspart at each meal, for a total of 64 U of insulin daily
- The physician divides 1800 by 64 to get 28 mg/dL/U
- He would like to lower her blood glucose from 325 mg/dL to 180 mg/dL, a drop of 145 mg/dL. To calculate the aspart dose to give, he divides 145 mg/dL by 28 mg/dL/U to get 5 U of aspart needed to lower her blood glucose to 180 mg/dL

A number of recent interventional studies have demonstrated that attaining and maintaining a blood glucose level between 80 mg/dL and 110 mg/dL in the hospital leads to improved clinical outcomes.[14]

The physician orders an additional 5 U of aspart to be added to her usual mealtime dose of 8 U, for a total of 13 U.

Patients Who Are Unable to Eat. When hospitalized patients are not eating, their insulin needs can be met with a long-acting insulin analogue. If patients are unable to eat for short periods of time, intravenous glucose is administered, which is a factor that must be considered when calculating basal insulin needs. Patients who are unable to eat for longer periods of time are usually placed on continuous tube feeds or total parenteral nutrition (TPN). The blood glucose needs of these patients would be most effectively addressed with a continuous intravenous insulin infusion, although this is impractical in many hospital settings.

Alternatively, the University of Vermont uses a protocol where 70/30 NPH/regular premixed insulin is given every 8 hours, thereby providing a relatively constant level of insulin effect for a 24-hour period. This alternative protocol also helps healthcare professionals control patients who are on 16-hour or overnight tube feeds by reducing or eliminating the dose that is acting when the tube feeds are not running. The following protocol can be used to calculate the starting insulin dose:

- 70/30 NPH/regular insulin with a total daily dose of:
  - 0.8 U/kg for the average patient (note that this dose is the same dose recommended above for basal bolus insulin by combining the basal and mealtime amounts into a single insulin dose)
  - 0.6 U/kg for patients with renal, cardiac, or hepatic dysfunction; for elderly patients; and when tube feedings are just beginning and are being advanced slowly
  - 1 U/kg for patients who are obese, who have open wounds infections, etc.
- Divide by 3 to obtain the 70/30 dose; give every 8 hours

To monitor this therapy, bedside glucose measurements should be obtained every 4 to 6 hours. The 70/30 insulin doses are then adjusted daily to attain blood glucose levels of 110 mg/dL to 180 mg/dL.

A similar insulin program can also be used for patients who are receiving TPN, although much or all of the insulin is often placed in the TPN bottle. If tube feedings or TPN are discontinued suddenly, blood glucose levels should be monitored closely and intravenous glucose should be given if levels fall below 100 mg/dL.[20]

Transitioning Patients From Intravenous to Subcutaneous Insulin

Patients who are unable to eat and who have been on insulin infusions will eventually need to be transitioned to subcutaneous insulin. To calculate the subcutaneous basal insulin needs, the number of units of intravenous insulin

used over the past 24 hours can be converted to units of long-acting insulin analogue on a 0.8 to 1.0 conversion.[20] For example, a patient who has been well managed on 3 U/hour can be converted to 60 to 72 U of a long-acting insulin analogue. Basal insulin therapy should be started a minimum of 2 hours prior to stopping the intravenous insulin infusion.[20] If the patient is eating when receiving intravenous insulin, it is preferable to give a dose of short-acting insulin analogue at mealtime so that the insulin infusion rate still represents the patient's basal insulin needs.[20]

Hospital-Wide Initiatives

From the above discussion, it is clear that identification and treatment of hyperglycemia in hospitalized patients is an important part of patient management. Barriers to achieving optimal care, however, do exist. Competing priorities and limited resources are a challenge.[14] In particular, the nursing resources required to care for these patients may strain the system. Furthermore, many healthcare providers are not only skeptical about the benefits of good inpatient glycemic control, but also fearful of hypoglycemia.[14] There may also be a lack of knowledge about hyperglycemia and DM.[14]

Although such barriers exist, strategies can be developed to improve the inpatient care of individuals with DM. Multidisciplinary committees can be formed to promote initiatives for improved care, administrative support should be emphasized to make this level of care a priority, and current processes and barriers within institutions should be assessed.[14] Consider creating a hyperglycemia management team, with representatives from medicine, nursing, pharmacy, nutrition, and diabetes education.[14] Such teams can develop hospital-wide protocols for the management of hyperglycemia and hypoglycemia, with the goal of facilitating care.[14]

PREPARING FOR HOSPITAL DISCHARGE

Discharge planning for patients with DM must be individualized, and whether patients go home on their usual diabetes medicines or new/adjusted insulin programs will depend on their admitting diagnosis, length of hospitalization, comorbidities, and social situation. However, the most valuable information is often the admission $HbgA_{1c}$ value. If patients are at target before becoming ill, they should be able to return to their previous treatment programs once returned to their usual states of health. Alternatively, a high $HbgA_{1c}$ value with a short illness means that the outpatient treatment program should be adjusted. In particular, a value exceeding 8% or 9% often signifies a need for long-term insulin use. Planning, including patient education, should begin before the discharge date.

Clinical Vignette (cont.)

After one day in the hospital, the patient began to feel better. Her physician arranged for a predischarge consult with a diabetes educator and a nutritionist. The patient spoke with both, but still did not fully understand her plan of action. A second session with each was arranged prior to discharge, and the patient's husband agreed to be present at these sessions.

To determine if patients who were on insulin in the hospital should be discharged on insulin, healthcare providers should consider the following questions:

- Was the patient taking insulin prior to admission?
- What was the $HbgA_{1c}$ level on admission?
- What is the patient's medical history?
- How difficult was it to control the patient's blood glucose levels in the hospital?
- Does the patient have any contraindications for the use of oral hypoglycemic agents?

If patients do not have a prior history of DM, a follow-up outpatient physician visit should occur within 1 month of discharge to complete a full workup and confirm the diagnosis.[8] Patients who have a history of type 2 DM who were being treated with oral agents can be discharged on their previous therapy with additional insulin if needed, based on their admission diagnosis and in-hospital insulin requirements, and their medical regimen can be reassessed within a few weeks.

Discharge planning should include diabetes self-management education while in the hospital.[8] Prior to discharge, patients must be able to monitor their own blood glucose levels and take medications as prescribed.[8] If patients cannot complete these tasks on their own, providers should ascertain if family members will be available to assist them.[8] The need for continuing education and/or a visiting nurse after discharge should be assessed, as should the patient's ability to pay for DM medication and supplies.[8]

Clinical Vignette (cont.)

When the patient's blood glucose was well controlled, she was discharged from the hospital. Given her new diagnosis of diabetes and an $HbgA_{1c}$ level of 8%, she was taught home blood glucose monitoring and was discharged on oral diabetes medications. She followed up with her primary care physician 3 weeks later. She continues to take her discharge medications, monitors her blood glucose levels as directed, and is implementing a weight-loss plan that includes moderate exercise.

PUTTING KNOWLEDGE INTO PRACTICE

- Test patient $HbgA_{1c}$ levels upon admission to the hospital (a value exceeding 8% or 9% often signifies a need for long-term insulin use)
- Strive to attain and maintain patient blood glucose levels between 80 mg/dL and 110 mg/dL
- Design insulin programs that meet eating status and illness characteristics
- Calculate starting insulin doses based on individual patient requirements
- Monitor blood glucose levels daily and adjust the insulin doses and programs as needed
- Plan ahead for discharge by initiating diabetes self-management education while the patient is still in the hospital
- Encourage patients to follow up with outpatient providers within 1 month of hospital discharge

SUMMARY

Hyperglycemia remains underdiagnosed and undertreated in the general population, as well as in the hospital setting. Because it leads to damage in multiple organ systems, appropriate and timely management is crucial.

Tight glucose control has been shown to improve outcomes for hospitalized patients, and those with hyperglycemia should be managed with insulin therapy. Patients who can eat should receive a long-acting insulin analogue for basal coverage and a short-acting insulin analogue to address postprandial glucose spikes. Patients who are unable to eat can be managed with intravenous infusions of insulin or, if on TPN or tube feeds, 70/30 NPH/regular insulin in divided doses throughout the day. Patients known to have DM should receive predischarge education; those with hyperglycemia, but no prior diagnosis of DM, require an outpatient workup to assess for DM.

A multidisciplinary approach provides the best care for patients with hyperglycemia. Physicians, nurses, diabetes educators, and nutritionists can work collaboratively to create effective in-hospital protocols that optimize outcomes for patients admitted with hyperglycemia.

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IMPROVING GLYCEMIC CONTROL IN HOSPITALIZED PATIENTS:
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1. Discuss the physiologic effects and clinical sequela of poorly controlled glycemia in hospitalized patients	☐	☐	☐	☐	☐
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	A Model of Its Kind 7	6	5	Average 4	3	Needs Serious Improvement 2	1
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1. **From the years 2000 to 2050, it is expected that the number of diagnosed cases of DM will increase by:**
   A. 115%
   B. 135%
   C. 140%
   D. 165%

2. **Hyperglycemia is associated with all of the following adverse physiologic effects, *except*:**
   A. Endothelial dysfunction
   B. Bronchospasm
   C. Blood pressure changes
   D. Neurologic damage

3. **Non-critically ill patients with newly diagnosed hyperglycemia have a lower in-hospital mortality rate than non-critically ill patients with a history of DM.**
   A. True
   B. False

4. **In a recent study of hyperglycemic patients in the medical ICU, van den Berghe et al. found that, compared with conventional therapy, intensive insulin therapy:**
   A. Did not reduce blood glucose levels
   B. Significantly reduced in-hospital mortality
   C. Significantly reduced in-hospital morbidity
   D. All of the above

5. **Patients in the ICU should maintain a maximum blood glucose level of:**
   A. 100 mg/dL
   B. 110 mg/dL
   C. 120 mg/dL
   D. 130 mg/dL

6. **Short-acting insulin analogues offer greater flexibility than short-acting human insulin because they:**
   A. Do not have to be taken before a meal
   B. Can be taken up to 20 minutes after a meal
   C. Carry a lower risk of postprandial hypoglycemia
   D. All of the above

7. **The use of sliding-scale insulin therapy is not recommended because it has been shown to lead to suboptimal glycemic control.**
   A. True
   B. False

8. **It is appropriate to prescribe a lower dose of long-acting insulin to patients who have:**
   A. Poor renal function
   B. An infection
   C. Metabolic syndrome
   D. Experienced recent CABG

9. **When taking a multidisciplinary approach to creating a hyperglycemia management team, in addition to medicine and pharmacy, which of the following groups should be represented?**
   A. Nutrition
   B. Nursing
   C. Diabetes education
   D. All of the above

10. **Before discharge, the most valuable information for proper outpatient planning is often the:**
    A. Admission blood glucose levels
    B. Predischarge blood glucose levels
    C. Admission $HbgA_{1c}$ values
    D. Predischarge $HbgA_{1c}$ values

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