Management of Inpatient Hyperglycemia

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Abstract

Hyperglycemia occurs frequently in hospitalized patients and affects patient outcomes, including mortality, inpatient complications, length of stay, and overall hospital costs. Various degrees of glycemic control have been studied and a recent consensus statement from American Diabetes Association (ADA)/American Association of Clinical Endocrinologists (AACE) recommends a target glucose range of 140-180 mg/dL in most hospitalized patients. Insulin is first-line therapy for hyperglycemia as it is adaptable to the changing patient physiology over the course of hospitalization. Critically ill patients should receive intravenous (IV) insulin infusion, and all non-critically ill patients with hyperglycemia should be managed using a subcutaneous (SC) insulin algorithm with basal, nutritional, and correctional dose components. The limiting factor to achieving a near euglycemic state is hypoglycemia. Similar to hyperglycemia, hypoglycemia is an independent risk factor for poor outcomes in the hospitalized patient. Institutions can increase safe insulin use by utilizing insulin algorithms, pre-printed order sets, and hypoglycemia protocols as well as by supporting patient and health care provider education.

Keywords: glycemic control, inpatient, insulin, hyperglycemia

After reading this article, readers should be able to review new guidelines and provide an update on current management and issues of inpatient hyperglycemia management.

Over the past decade, hyperglycemia in the hospitalized patient has gained attention due to the association with increased mortality, inpatient complications, and negative economic impact. Hospitals have invested tremendous efforts in addressing glycemic control, developing insulin protocols, and educating health care providers. Recently, multiple national organizations and professional societies have published and revised guidelines regarding the management of inpatient hyperglycemia to improve patient care. Hypoglycemia is also associated with poor hospital outcomes and remains the limiting factor in controlling hyperglycemia. Improved glycemic control throughout the hospital stay, preventing hyperglycemia and hypoglycemia, is associated with decreases in short- and long-term mortality, inpatient complications, and hospital lengths of stay. Financial benefits of glycemic control are significant, not just in reducing direct hospital costs, but also in reducing length of stay and readmission rates.

Conservative estimates of the incidence of diabetes in adult hospitalized patients range from 12% to 26%. At 1 academic center, hyperglycemia in the non-diabetic patient at the time of hospital admission was 12%, which translates into 1 out of every 8 hospital admissions. Stress hyperglycemia historically was felt to be part of the natural course of acute illness and not treated unless glucose levels exceeded 200 mg/dL or were symptomatic. However, we now know that stress hyperglycemia has been associated with longer hospital stays, higher rates of intensive care unit (ICU) admission, greater need for rehabilitation services at the time of discharge, and higher mortality rates. In the largest review of hospital glucose data of more than 12 million blood sugars at 126 U.S. hospitals, 46% of all blood sugars in the ICU setting and 31.7% of all blood sugars in non-ICU patients were in the hyperglycemic range (defined as a glucose >180 mg/dL).

Abbreviations

IV, intravenous; SC, subcutaneous; ICU, intensive care unit; BG, blood glucose; AMI, acute myocardial infarction; CAP, community-acquired pneumonia; COPD, chronic obstructive pulmonary disease; NICE-SUGAR, Normoglycemia in Intensive Care Evaluation and Survival Using Glucose Algorithm Regulation; AACE, American Association of Clinical Endocrinologists; ADA, American Diabetes Association; OADs, oral antidiabetic drugs; GLP-1, glucagon-like peptide 1; SHM, Society of Hospital Medicine; TDD, total daily dose; SSI, sliding-scale insulin; HbA1C, hemoglobin A1c; JCAHO, Joint Commission on Accreditation of Healthcare Organizations; TPN, total parenteral nutrition; EMR, electronic medical record; POCT, point-of-care glucose testing

Chemistry exam 21102 questions and corresponding answer form are located after this CE Update on page 435.
Improving the treatment of hyperglycemia in hospitals is gaining inertia, but it still has yet to be universally accepted.

The link between hyperglycemia and adverse hospital outcomes is multi-factorial. Elevated blood glucose (BG) concentrations produce a proinflammatory cytokine predominance, leading to a multitude of downstream effects, including capillary basement membrane thickening, impaired phagocytosis and immunity, oxidative stress, abnormal lipid metabolism, decreased vascular contractility, increased platelet adhesiveness, increased concentrations of coagulation factors, and increased C-reactive protein levels. Contributing factors to hyperglycemia include elevations in stress-related hormones (growth hormone, catecholamines, cortisol, glucagon), pharmacologic agents, enteral and total parenteral nutrition (TPN), and glucocorticoid therapy. As a result, a glycemic control plan should be in place for all hospitalized patients with hyperglycemia.

Hospialized patients with diabetes have higher perioperative mortality, deep sternal wound infections, postoperative strokes, and longer length of hospital stays. Tighter glucose control has been identified as a predictor of improved outcomes in a variety of patient settings, including acute myocardial infarction (AMI), strokes, community-acquired pneumonia (CAP), chronic obstrucive pulmonary disease (COPD) exacerbations, and in non-ICU postsurgical settings such as renal transplantation, total joint arthroplasty, and colorectal surgery. Morbidity and mortality are correlated with the presence and degree of hyperglycemia in the postoperative period and are independent of a prior diagnosis of diabetes.

Interventional outcomes studies have shown benefits in inpatient morbidity and hospital mortality from intensive inpatient hyperglycemia management. In Van den Berghe’s landmark study in a single center Belgian surgical ICU, tight glycemic control was associated with a 34% reduction in hospital mortality, 46% reduction in sepsis, 41% reduction in renal impairment requiring dialysis, 50% reduction in blood transfusions, and 44% reduction in the incidence of ICU polyneuropathy compared with standard hyperglycemia management. Based on these results, the standard of care changed in the glucose management of ICU patients, but the optimal glycemic target remained controversial. Similar studies in different patient populations were unable to reproduce Van Den Berghe’s success. In March 2009, the largest randomized ICU trial assessing intensive glycemic control—the Normoglycemia in Intensive Care Evaluation and Survival Using Glucose Algorithm Regulation (NICE-SUGAR) study, reported higher mortality and hypoglycemia rates in ICU patients treated with intensive glycemic control (80-110 mg/dL) compared to less tight glycemic control (glucose <180 mg/dL). The conventional group in NICE-SUGAR, however, required insulin 69% of the time in order to achieve the target glucose below 180 mg/dL, indicating a continued need for insulin therapy in the majority of critically ill patients just with a less intensive glucose target range. The goal target range for all critically ill patients remains controversial, but it is likely population specific and should be individualized based on the clinical situation, training of ICU personnel with insulin protocols, and risk of hypoglycemia.

Glycemic variability has emerged as an additional component affecting inpatient hyperglycemia outcomes. In the outpatient setting, wider glucose fluctuations (defined as the amplitude from peak to trough in glucose) corresponds to an increased risk of diabetic microvascular complications in patients with diabetes. In the ICU setting, Krinsley and colleagues previously identified the near linear relationship between ICU mortality and mean glucose. Further data from his mixed medical, cardiac, and surgical ICU shows glucose variability is also a predictor of ICU mortality. In patients with the best glycemic control (mean glucose 70-99 mg/dL), the patients with the largest glycemic variability had a 5-fold increase in mortality compared to patients with the least glycemic variability. A larger European ICU study had similar results.

AACE/ADA Guidelines for Optimal Glycemic Control

Guidelines for optimal glycemic control in the hospital setting have been developed by the American Association of Clinical Endocrinologists (AACE) and the American Diabetes Association (ADA). The AACE/ADA recommendations can be summarized as 1) identify elevated BG in all hospitalized patients; 2) establish a multidisciplinary team approach to diabetes management in all hospitals; 3) implement structured protocols for aggressive control of BG in both ICUs and other hospital settings; 4) create educational programs for all hospital personnel caring for people with diabetes; and 5) plan for a smooth transition to outpatient care with appropriate diabetes management. These guidelines give institutions structure to develop protocols that achieve BG goals yet allow for individualization of algorithms and policies to fit with the hospital’s culture and environment. In May 2009, AACE/ADA revised their inpatient glycemic targets to 140-180 mg/dL in the ICU and non-ICU preprandial glucose levels below 140 mg/dL and all random glucose levels below 180 mg/dL. (Table 1).

Hyperglycemia Treatment Options in the Hospital Setting

The goal of insulin therapy is to reach acceptable glucose targets in the shortest duration of time and with minimal incidence of hypoglycemia. Once admitted to the hospital, patients with hyperglycemia should be managed using either intravenous (IV) or subcutaneous (SC) insulin algorithms. Most treatment algorithms recommend discontinuation of oral antidiabetic drugs (OADs) and initiation of insulin analog therapy. Insulin secretagogues, such as sulfonylureas and glinides (nateglinide, repaglinide), are associated with an increased risk of hypoglycemia. Metformin should not be used for inpatients because of the increased risk of acute changes in renal function (due to volume shifts, medications, or contrast-induced nephropathy). Thiazolidinediones (rosiglitazone, pioglitazone) are insulin sensitizers that can increase circulating plasma volume by 6% to 7%, and therefore should not be used in patients with edema or heart failure. Incretin-based agents (sitagliptin, saxagliptin, exenatide, liraglutide, and pramlintide) may increase the risk of gastrointestinal adverse effects, which may slow the recovery of a hospitalized patient, although they have not been frequently studied in the inpatient setting. Incretins may have a role in the hospital as they affect endogenous insulin secretion in a glucose-dependent mechanism, thereby minimizing the risk of hypoglycemia. Intravenous infusion of a glucagon-like peptide 1 (GLP-1) agonist
was recently compared to IV insulin infusions in non-critically ill patients and controlled blood sugars with less hypoglycemia.42 This proof of concept study raises the hope for non-insulin based mechanisms of glycemic control to be further investigated.

**Insulin: The Standard of Care**

Insulin is the preferred agent for glycemic control in hospitalized patients. The pharmacodynamics of insulin allow it to be adaptable to the changing physiology of the sick patient, is easily titrated, and has no dosage threshold. Furthermore, insulin has a rapid onset of action, minimal side effects except for hypoglycemia, and has minimal drug-drug interactions. The ideal insulin protocol will help reach the glucose target range timely, effectively treat all degrees of hyperglycemia, minimize glycemic variation and the risk of hypoglycemia, and is easy for nurses to carry out in a timely fashion.43

The Society of Hospital Medicine (SHM) has created a workbook to guide hospitals in creating safe and effective glycemic control plans. Included in the workbook appendix are multiple successful inpatient insulin protocols. The common themes in these protocols include the use of regular insulin for continuous insulin infusion and a basal/bolus SC insulin regimen including a long-acting insulin analog (insulin glargine or detemir) and prandial and correctional doses of a rapid-acting insulin (insulin aspart, glulisine, or lispro).44-47

Continuous infusion of regular insulin is suggested for critically ill ICU patients, pre- and postoperative patients, peripartum women with hyperglycemia, severe hyperglycemia with metabolic decompensation (diabetic ketoacidosis and hyperosmolar non-ketotic states), and any patient in whom tight glycemic control is clinically indicated. Paper-based and computer-based insulin infusion algorithms are available to help clinicians achieve optimal glycemic control.44,48,49

Conversion from IV to SC insulin commonly occurs when the critical illness resolves when the patient is extubated, off vasopressors, and ready to begin eating, or is at a stable tubefeed rate. When the patient is being converted from an IV insulin drip, the drip rate is used as a guide to determine total daily insulin requirements. The insulin drip rate over the preceding 6 hours is averaged to obtain a stable hourly rate. The average hourly rate is multiplied by 24 hours to calculate the total daily dose (TDD) of insulin required. The basal insulin dose ordered is 60%-80% of the TDD, and the prandial insulin dose for each meal is 10% of the TDD. The prandial insulin dose is adjusted accordingly as the patient’s appetite improves. The proportion of insulin given for prandial dosing is substantially less because these patients are generally consuming only a clear liquid diet initially with a reduced caloric content. The insulin infusion should be continued for 4 hours after the first injection of basal insulin is given (if insulin glargine or detemir; 2 hours if NPH insulin). Practical necessities sometimes outweigh physiologic reasoning in that the conversion from IV to SC insulin often coincides with the transfer of the patient out of the ICU rapidly. Basic insulin may be given, and the insulin infusion is simply stopped without an overlap period. In this scenario, a conversion dose of a rapid-acting insulin (10% of TDD) can be given simultaneously with the basal insulin, and the insulin infusion can be discontinued without an overlap period.50

For all non-critically ill patients, a basal/bolus insulin regimen is the preferred method of glycemic control. Basal insulin suppresses hepatic gluconeogenesis between meals and overnight. During illness, basal insulin requirements rise with any physical stress, including surgery, infection, infarction, or fever. For patients who are eating, a scheduled mealtime insulin dose with a rapid-acting insulin analog helps prevent the glucose from rising from carbohydrate intake. Whether eating or not, when blood sugars are outside the glycemic target range, a correctional dose of rapid-acting insulin should be administered.

Hospitalized patients have unpredictable eating and diagnostically unpredictable testing schedules and thus are more susceptible to an insulin-food dyssynchrony; hypoglycemia occurs if insulin peaks before the patient has eaten or consumed enough carbohydrates; hyperglycemia results if the insulin peak is insufficient to meet glucose intake or metabolic stress needs. Rapid-acting insulin analogs can be given immediately before or up to 20 minutes following food consumption and thus are more flexible and less likely to cause hypoglycemia compared to regular insulin.51

Insulin analogs are preferred for basal, mealtime, and correction doses instead of human insulins (regular and NPH). Insulin analogs have a more predictable absorption and action profile in addition to less pharmacokinetic fluctuations in patients with renal insufficiency. High doses of human insulins have not only a greater peak effect than lower doses but also result in a longer duration of action. Overlap of insulin doses (known as insulin stacking) increases the risk of a hypoglycemic event. The duration of action of rapid-acting insulin analogs are predictable at low and high doses, thereby decreasing the risk of insulin stacking. Insulin analogs have a more consistent pharmacokinetic and pharmacodynamic profile (less inter- and intra-individual variability), and it is easier to predict the effect the dose will have on an individual’s BG concentration. Table 2 describes the types of insulin recommended for hospital use.54,55

<table>
<thead>
<tr>
<th>Table 1_Glycemic Goals in the Hospital</th>
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<tbody>
<tr>
<td>Patient Type</td>
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<td>---------------------------------------</td>
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<tr>
<td>Critically ill patients</td>
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<td></td>
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<tr>
<td>Non-critically ill patients</td>
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Regular insulin should be avoided for SC postprandial BG correction and should not be used as monotherapy in a sliding-scale regimen. Numerous studies over the past 50 years show that sliding-scale insulin (SSI) alone is not effective for inpatient glycemic control (Figure 1) and more recently has been associated with increased inpatient mortality.56 Sliding-scale insulin regimens do not allow for basal or mealtime insulin requirements and grossly underestimate total daily insulin requirements. Furthermore, SSI regimens respond to hyperglycemia after it has happened, rather than preventing it, and the sliding scale depends on the inaccurate assumption that insulin sensitivity is uniform among all patients.

In a randomized, prospective study, the use of a basal/bolus regimen vs SSI regimen in diabetic patients naïve to insulin (on oral agents only) was superior in achieving glycemic control with almost no hypoglycemia (RABBIT-2).57 In this study and the subsequent RABBIT surgery study,58 the TDD was calculated by multiplying the patient weight in kilograms by either 0.4 or 0.5 units/kg and ordering 50% of TDD as basal insulin and the other 50% of TDD as mealtime insulin divided equally into 3 mealtime doses. Glucose targets were reached in the majority of patients in the basal bolus intervention group with minimal hypoglycemia.

Premixed insulins are generally not recommended for use in the hospital setting, as there is an increased risk of hypoglycemia in patients with variable oral intake. Some hospitals have eliminated premix insulins via therapeutic interchange so patients do not get mealtime insulin if at a procedure or NPO, therefore reducing the risk of hypoglycemia. The TDD can be calculated by adding up the total daily home dose of premixed insulin. This allows basal and pre-meal insulins to be ordered separately; thereby, mealtime insulin can be administered only when the patient is eating. Subsequently, at the time of discharge, these patients can be converted back to their premixed insulin if appropriate.

All hyperglycemic patients should have their hemoglobin A1c (HbA1C) checked on admission to help differentiate between pre-hospital or acute onset of hyperglycemia. The Joint Commission on Accreditation of Healthcare Organizations (JCAHO) recommends a A1c during the hospital stay if one is not documented in the past 60 days to identify previously unrecognized diabetes or to help guide optimization of the outpatient diabetes regimen if the A1c is elevated. Recently, an international expert committee recommended an A1c ≥ 6.5% indicates a diagnosis of diabetes.59

### Table 2 Insulins Used in Hospitalized Patients (ref 68)

<table>
<thead>
<tr>
<th>Generic (Brand)</th>
<th>Onset/Duration</th>
<th>IV/SC Use</th>
<th>Recommended Use in Hospital</th>
</tr>
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<tbody>
<tr>
<td><strong>RAPID-ACTING INSULINS</strong></td>
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<td></td>
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<tr>
<td>Insulin lispro (Humalog)</td>
<td>5-15 min</td>
<td>SC</td>
<td>15 min before or immediately after meal</td>
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<tr>
<td></td>
<td>3-5 h</td>
<td></td>
<td></td>
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<tr>
<td>Insulin aspart (NovoLog)</td>
<td>5-15 min</td>
<td>SC, IV*</td>
<td>5-10 min before meal</td>
</tr>
<tr>
<td></td>
<td>3-5 h</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Insulin glulisine (Apidra)</td>
<td>5-15 min</td>
<td>SC, IV*</td>
<td>15 min before or 20 min after starting meal</td>
</tr>
<tr>
<td></td>
<td>3-5 h</td>
<td></td>
<td></td>
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<tr>
<td><strong>SHORT-ACTING INSULINS</strong></td>
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<td></td>
<td></td>
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<tr>
<td>Regular insulin (Humulin R, Novolin R)</td>
<td>30-60 min</td>
<td>SC, IV</td>
<td>Preferred for insulin drips; avoid for postprandial use; do not use sliding scale</td>
</tr>
<tr>
<td></td>
<td>6-8 h (longer with U-500 or renal insufficiency)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>INTERMEDIATE-ACTING INSULINS</strong></td>
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<tr>
<td>NPH insulin (Novolin N, Humulin N)</td>
<td>2-4 h</td>
<td>SC</td>
<td>Can be used as a twice-daily substitute for basal insulin regimen</td>
</tr>
<tr>
<td></td>
<td>8-12 h (longer with renal insufficiency)</td>
<td></td>
<td></td>
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<tr>
<td><strong>LONG-ACTING INSULINS</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Insulin detemir (Levemir)</td>
<td>3-8 h</td>
<td>SC</td>
<td>Preferred for basal insulin use</td>
</tr>
<tr>
<td></td>
<td>16-24 h</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Insulin glargine (Lantus)</td>
<td>2-4 h</td>
<td>SC</td>
<td>Preferred for basal insulin use</td>
</tr>
<tr>
<td></td>
<td>24 h</td>
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IV, intravenous; SC, subcutaneous.
*Insulin analogs have no benefit over regular insulin for intravenous infusions.

### Insulin Protocols Provide Consistent, Effective Glycemic Control

Implementation of insulin protocols may seem like an easy step, but it requires a hospital-wide effort at the clinician, nursing, and pharmacy levels.45 The first step in changing the glycemic control culture at an institution is to create a multidisciplinary committee led by a physician champion. Administrative support is critical at all levels for change to be accepted. Bad habits die hard; overcoming the popular use of
SSI regimens and adjusting nursing habits formed over a career require frequent re-education and support from hospital leaders.

Not all protocol implementations or adjustments are successful, however, and a reassessment of the effectiveness of a protocol is required. As an example, in January 2007, the Detroit Medical Center removed regular insulin sliding scales (SC regular insulin orders in non-TPN or for patients receiving enteral nutrition) from the hospital formulary and replaced these with low-, moderate-, and high-dose insulin aspart correction scales. The goal was to change current practice patterns, promote basal/bolus insulin regimens, and improve overall glycemic control. In the following 4 months, a review of 301,549 capillary glucose values did not show an improvement in glycemic control (and hypoglycemia rates remained unchanged).60 Practice patterns of clinicians were not changed at this institution; sliding-scale-only insulin regimens remained the most common insulin regimen ordered. Education is the key to success; however, a stepped implementation of SC insulin protocols (paper-based or via electronic medical record [EMR]) leading to mandatory use of basal/bolus insulin on a hospital-wide basis is optimal. One community hospital’s successful conversion to a basal/bolus insulin protocol in the majority of hospital patients has led to an increase in all blood sugars in the 70-180 mg/dL range from 56.6% to 72.2% over a 3-year period while hypoglycemia rates remained 2% (unpublished data, DMC Huron Valley-Sinai Hospital). Similar improvements have been shown at Boston Medical Center.62

Quality control monitoring of glycemic control by hospital administration helps measure success (by showing improvements in glycemic control) or trouble spots in the hospital to target further education and/or process improvement. Glucometrics, a Web site-based benchmarking tool from Yale University (http://metrics.med.yale.edu), recently compiled glucose data from 31 U.S. hospitals to document real-world glycemic control in the hospital setting. Analysis of more than 1.5 million blood sugars showed an average per patient-day mean glucose of 151.8 mg/dL in ICU patients and 152.7 mg/dL in non-ICU patients. Outside the ICU, a mean target glucose of 70-149 mg/dL was achieved in 55.8% of the patients, and 9.5% of patient-days had at least 1 hypoglycemic episode.63

Minimizing Hypoglycemia

Hypoglycemia is the limiting factor to aggressively normalizing blood sugars in all patients. Hypoglycemia is an independent predictor of hospital mortality. Spontaneous hypoglycemic events (not induced by insulin) are associated with significantly higher mortality rates when compared with iatrogenic or insulin-induced hypoglycemic events.64,65 In the largest review of hospital glucose data of more than 12 million blood sugars at 126 U.S. hospitals, 10.1% of all blood sugars in the ICU setting were in the hypoglycemic range (defined as a glucose <70 mg/dL) and 3.5% of all blood sugars in non-ICU patients indicated hypoglycemia.11 In a study of more
than 100,000 inpatient admissions in patients with diabetes, patients who experienced hypoglycemic episodes had longer hospital stays, a 7% higher risk of inpatient mortality, a 39% increase in hospital costs, and a 58% increase likelihood of discharge to a skilled nursing facility.66

Root cause analysis of more than 1000 hypoglycemic events at a large academic medical center found that 67% of the documented hypoglycemic episodes were associated with a change in nutritional status (diet, enteral, or parenteral rate) within the previous 24 hours (unpublished data, Northwestern Medical Center, Chicago, IL). Additional factors increasing the risk of hypoglycemia include a lack of coordination between feeding and insulin administration (insulin-food dysynchrony), insufficient frequency of BG testing, orders not clearly or uniformly written, and failure to adjust insulin requirements in patients with advanced age, renal failure, liver disease, or changing clinical status. With the addition of a nursing EMR-based hypoglycemia event form to document each event at this center, the incidence of hypoglycemia was decreased 50%.

Preventing and minimizing the incidence and severity of hypoglycemia is possible with the use of standardized insulin protocols, hypoglycemia protocols, and the use of insulin analogs.67 Hypoglycemia protocols should be nurse driven and supported by point-of-care glucose testing (POCT). Ongoing review process of insulin-related errors and near misses should be conducted at the hospital level.

**The Role of Point-of-Care Testing**

Point-of-care testing is a convenient method to acquire glucose levels in a timely manner allowing for rapid insulin titration and clinical decision making. Glucometers, however, may not be the most accurate and reliable method to monitor glucose. An international standard is currently under development that recommends improving the accuracy of POCT devices to ±20 mg/dL for glucose values under 100 mg/dL and ±20% for higher glucose values. This is quite a large variation given the narrow therapeutic window.68 Common reasons for erroneous POCT BG readings, such as improper handling of the test strips and equipment leading to analytic error, underscore that care must be used in following the manufacturer’s instructions regarding appropriate quality-control measures. The POCT BG results may be inaccurate in patients with extremes in hematocrit, glucose, PO2, values and body temperature.69 Specimens from arterial blood have higher glucose concentrations than venous samples; glucose levels in plasma are generally 10%-15% higher than glucose measurements in whole blood. Capillary specimens may not be representative of central BG concentrations in patients with shock or diabetic ketoacidosis. Certain medications may interact with the POCT equipment and produce a faulty reading (eg, maltose- and fructose-containing medications can interfere with meters utilizing the dehydrogenase method). In these cases, POCT should be used with caution. In general, it is a good idea to confirm unexpected POCT BG results with a specimen sent to a central laboratory for analysis.

Continuous glucose monitoring systems have been developed for the outpatient setting, but none have been approved for use in the hospital. Given the limitations of POCT, the need for more accurate and real-time glucose monitoring is apparent. Development of SC, transdermal, and IV central line glucose monitoring is under way at various phases of study.70-72

**Hospital System Enhancements for Safe Insulin Use**

Standardized protocols promote the safe use of insulin in hospitals.68 Complicated insulin regimens can lead to confusion and medication errors, so it is recommended to simplify insulin regimens as much as possible. Simplification of the hospital insulin formulary is the first step, limiting clinicians to a single basal insulin and a single rapid-acting insulin choice. Monitoring and documentation of insulin administration and glucose measurement should be together as a single document (either paper flowsheet or a single EMR screen). Computerized glycemic control algorithms have been shown to improve efficacy and decrease insulin administration error rates.73 Revisiting protocols and assessing provider adherence to pre-printed orders and protocol directions should be conducted regularly or whenever a discrepancy is found. It is important to identify and then resolve any inconsistencies or errors within the protocols.

**Summary**

Insulin is the preferred therapy for treating all hospitalized patients with hyperglycemia, independent of their diabetes status. With recent outcomes studies like NICE-SUGAR, we have transitioned from the era of “tight” glycemic control to one of “less-tight” glycemic control, focusing intensely on the safety and efficacy of our glycemic control plan. Although optimal target glucose ranges remain controversial, the consensus is that glycemic control is important and hospitals should continue to manage blood sugars with insulin. Those hospitals that are successful with lower glycemic targets may choose to continue current practice; others may choose higher glycemic targets to balance the risk of hypoglycemic events and ensure patient safety.

Improvement in glycemic control throughout the hospital includes efforts at every level: physician education and ordering of appropriate insulin and medication regimens, nursing coordination on the timing of insulin administration and treatment of hypoglycemia, patient-care associates measuring capillary BGs and communicating results promptly, dietary teams notifying the nurse or unit clerk when trays arrive on the floor, and, finally, the patient being his/her own advocate in treating diabetes and hyperglycemia. At every step of the process, glycemic control can be affected. As with most things in life, education is the key to success.1m


54. Schmelz L. System-wide formulary change from regular insulin sliding scales to insulin aspart sliding scales did not improve overall glycemic control [abstract]. Diabetes Pro. 2008;57(suppl1):A162.


