Managing Hyperglycemia During Critical Illness

New Opportunities to Improve Clinical Outcomes

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It is well known that tight glycemic control decreases the risk of long-term complications of diabetes mellitus. Multiple guidelines advocating near-normal blood glucose levels have been published for the outpatient management of diabetes. Much less attention had been paid, however, to the consequences of acute hyperglycemia during illness. Despite the common belief that stress-induced hyperglycemia is harmless, a strong correlation has recently been demonstrated between in-hospital hyperglycemia and poor clinical outcomes. Aggressive treatment of diabetes and hyperglycemia with the use of intensive insulin regimens significantly reduces morbidity and mortality among critically ill patients with a variety of medical and surgical conditions.

Pathogenesis of Stress Hyperglycemia

Stress hyperglycemia, defined as a transient increase in blood glucose concentration during acute physiologic illness, is a common problem among critically ill patients, even those who have not been diagnosed with diabetes. Critical illness is associated with increases in the principal insulin counterregulatory hormones, including catecholamines, glucocorticoids, glucagon, and growth hormone. This elevation in stress hormones accelerates catabolism, hepatic gluconeogenesis, and lipolysis, all of which lead to insulin resistance and hyperglycemia. In addition, several cytokines (tumor necrosis factor-alpha, interleukin-6, and interleukin-1) that are increased during acute illnesses may impair insulin signaling. As a result of these changes, insulin-mediated glucose uptake by skeletal and cardiac muscle is impaired, and insulin fails to suppress gluconeogenesis by the liver. The ongoing increase in glucose production by the liver, despite decreased glucose clearance, is considered the major mechanism responsible for stress-induced hyperglycemia. Additional factors contributing to hyperglycemia in the intensive care unit (ICU) include treatment with vasopressors, treatment with corticosteroids, provision of enteral or parenteral nutrition or excess amounts of dextrose, and bed rest itself.

The significance and underlying mechanisms of stress hyperglycemia in the absence of a history of diabetes are not fully understood. For years, stress hyperglycemia was considered merely a marker of severe disease and of the resulting increase in concentrations of stress hormones. Although a correlation does exist between the severity of critical illness and levels of counterregulatory hormones, not all patients in the ICU develop hyperglycemia. It has been proposed that stress hyperglycemia is a reflection not only of insulin resistance, but also of relative insulin deficiency. Hypoinsulinemia might result from several independent conditions, such as the blunting of insulin secretion via glucose toxicity, the unmasking of preexisting pancreatic beta-cell dysfunction during stress, or the direct effects of counterregulatory factors on islet function.

When 181 nondiabetic patients with acute myocardial infarction (AMI) and blood glucose levels less than 200 mg/dl on admission to the coronary unit were tested with a standard 75-g glucose tolerance test 3 months after discharge, approximately 40% were diagnosed with impaired glucose tolerance and 25% with diabetes. If patients with blood glucose greater than 200 mg/dl had been included in this study, the percentage of patients with undiagnosed diabetes would certainly have been even higher. The high prevalence of undiagnosed abnormal glucose metabolism in this setting supports the hypothesis that unrecognized diabetes or pre-diabetes is a major underlying mechanism of poor glycemic control in some patients with newly detected hyperglycemia during hospitalization.

Hyperglycemia in Hospitalized Patients

Recent observational studies have shown a high prevalence of in-hospital hyperglycemia, especially among critically ill patients. In a retrospective analysis of 2,030 consecutive adults admitted to a community teaching hos-

pital in Atlanta, hyperglycemia was present in 38% of patients on the general medical and surgical units. Of these patients, 26% were known to have diabetes, but the remaining 12% had no history of diabetes prior to admission (Figure 1). These results are consistent with other studies reporting an 8% to 12% prevalence of newly diagnosed hyperglycemia among hospitalized patients.

No consensus exists on what level of admission hyperglycemia should be considered abnormal in the absence of a history of diabetes. In most of the observational studies, admission hyperglycemia was defined as a random blood glucose level of 200 mg/dl (11.1 mmol/ liter) or higher on at least two occasions. This threshold is derived from the criteria for the diagnosis of diabetes mellitus as per the American Diabetes Association (ADA). The use of different thresholds in the literature to define admission hyperglycemia makes it difficult to determine its true prevalence.

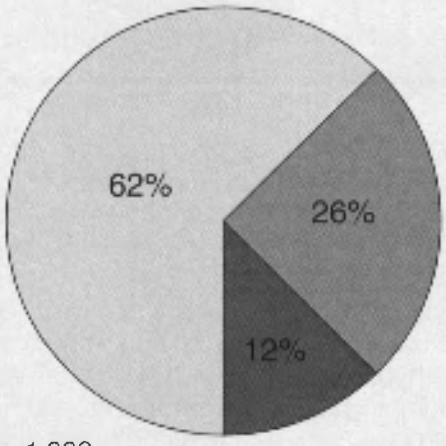
Admission Hyperglycemia Predicts Poor Outcomes

Substantial evidence suggests that admission hyperglycemia, apart from a history of diabetes, is a strong and independent risk factor for adverse short-term (in-hospital) and long-term outcomes in a variety of clinical settings. In patients hospitalized for AMI, admission hyperglycemia, defined as blood glucose greater than 110 mg/dl (6.1 mmol/liter), has been associated with increased risk of adverse events including congestive heart failure, cardiogenic shock, and death in patients with and without diabetes. Additionally, nondiabetic patients with AMI and admission blood glucose level of 200 mg/dl or higher had a similarly high risk of death as patients with AMI and previously diagnosed diabetes. Hyperglycemia in patients with cerebrovascular accidents also predicted a worse prognosis in terms of both mortality and residual neurological disability.

Several studies have demonstrated poorer outcomes among hyperglycemic patients without a history of diabetes than among patients with known diabetes. Patients admitted to the ICU with newly diagnosed hyperglycemia had a three-fold higher mortality rate (31%) than patients with a history of diabetes or with normoglycemia. (Although the mortality rate in diabetic patients was higher than that in the normoglycemic group, the difference did not reach statistical significance.) The higher mortality rate in patients with new hyperglycemia was mainly due to infectious disorders and acute neurologic events. In the same study, patients with new hyperglycemia

FIGURE 1.

PREVALENCE OF IN-HOSPITAL HYPERGLYCEMIA



Total = 1,886

Normoglycemia

Known diabetes

New hyperglycemia

Adapted from Umpierrez GE, Isaacs SD, Bazargan N, You X, Thaler LM, Kitabchi AE: Hyperglycemia: An independent marker of in-hospital mortality in patients with undiagnosed diabetes. *J Clin Endocrinol Metab* 87:978–982, 2002.

TABLE 1.

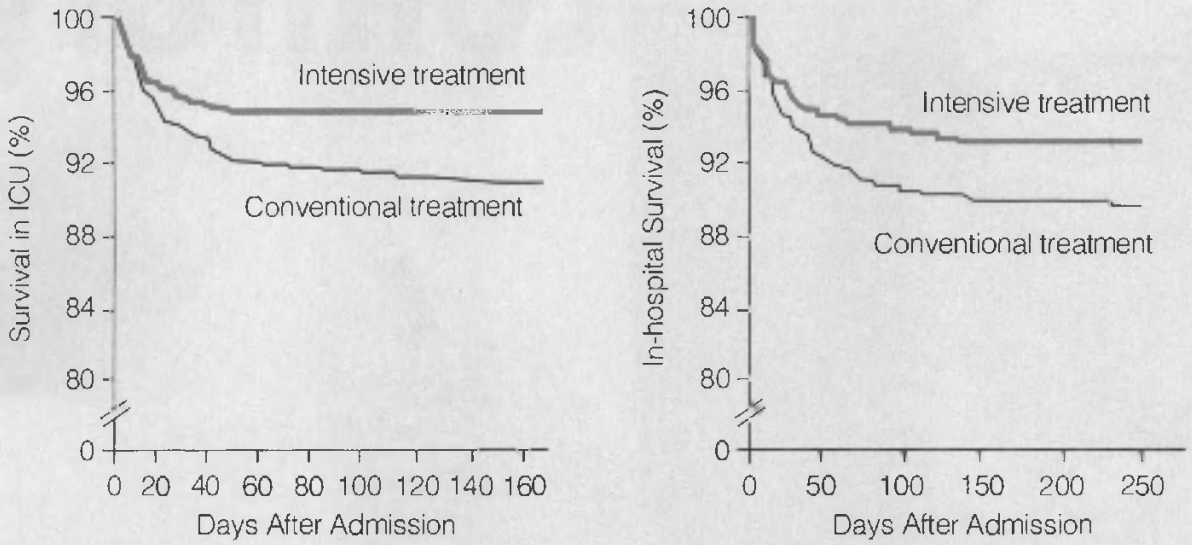
HOSPITAL MORTALITY RATE AND MEAN BLOOD GLUCOSE LEVEL

Mean Blood Glucose (mg/dl)	Mortality Rate (%)	Number of Patients
80–99	9.6	264
100–119	12.2	491
120–139	15.1	338
140–159	18.8	202
160–179	28.4	141
180–199	29.4	102
200–249	37.5	144
250–299	32.9	70
>300	42.5	40

Reprinted with permission from Krinsley JS: Association between hyperglycemia and increased hospital mortality in a heterogeneous population of critically ill patients. *Mayo Clin Proc* 78:1471–1478, 2003.

FIGURE 2.

SURVIVAL WITH INTENSIVE VERSUS CONVENTIONAL INSULIN TREATMENT IN THE ICU



ICU indicates intensive care unit.

on the general medical and surgical floors (not in the ICU) had an 18-fold increase in in-hospital mortality, compared with a 2.7-fold increase in patients with known diabetes. Patients with new hyperglycemia also had a longer hospital stay, a higher rate of admission to the ICU, and a higher rate of transfer to a transitional care unit or nursing home facility. Similarly, among 1,664 patients hospitalized with AMI in Nova Scotia, Canada, those with blood glucose greater than198 mg/dl and no history of diabetes had much worse outcomes than all diabetic patients admitted with AMI, with more than a two-fold higher risk of in-hospital mortality. In the same study, all diabetic patients and those with blood glucose higher than 198 mg/dl but without diabetes continued to fare worse than nondiabetic newly hyperglycemic patients whose admission blood glucose was below 198 mg/dl, even up to 1 year after discharge.

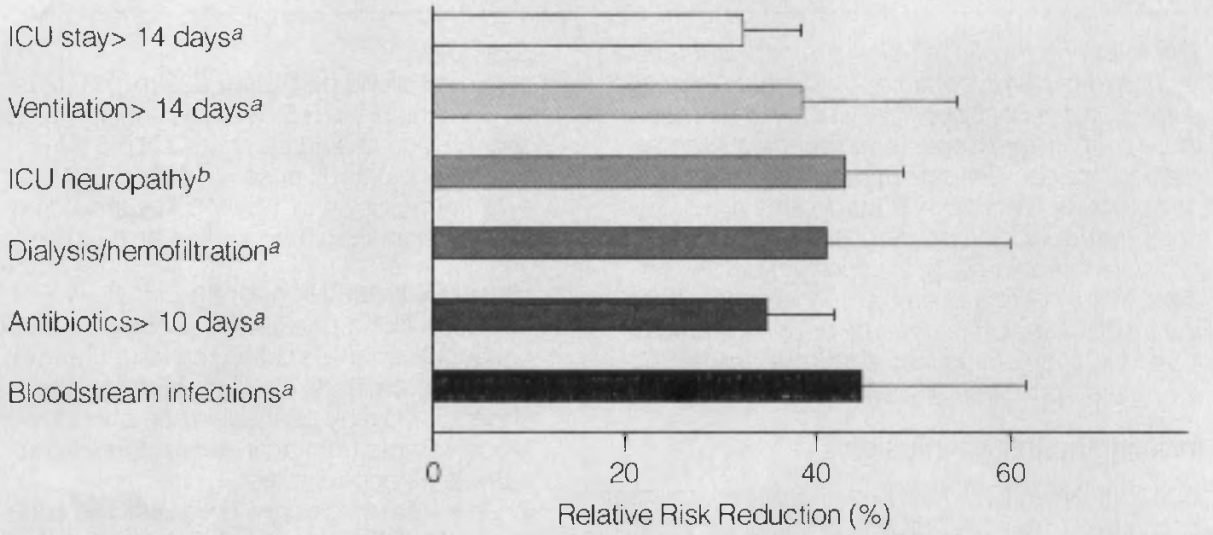
A continuous relationship has been demonstrated between admission hyperglycemia and poor clinical outcomes. A retrospective analysis of 1,826 patients admitted to the Stamford Hospital ICU, Stamford, Connecticut, showed that even a modest degree of hyperglycemia was associated with a substantial increase in hospital mortality (Table 1). The lowest hospital mortality rate, 9.6%, occurred among patients with mean blood glucose values between 80 mg/dl and 99 mg/dl. Further increases in mean blood glucose had a progressively deleterious effect, with hospital mortality reaching 42.5% among patients with mean blood glucose values exceeding 300 mg/dl. Initial, maximum, and mean blood glucose values were all significantly higher among nonsurvivors than among survivors for the entire population and for most of the patient subgroups. Similarly, among the patients admitted with AMI, each increase in admission blood glucose level of 18 mg/dl (1 mmol/liter) was associated with a 5% increase in mortality risk among diabetic patients and a 4% increase among nondiabetic patients. Finally, in the cardiac surgery population, hospital stay was reduced by 1 day for each 50-mg/dl lowering of the average 3-day postoperative blood glucose level.

Intensive Glucose Control with Insulin Infusion in Critically Ill Patients

Although epidemiologic studies have demonstrated a high prevalence of in-hospital hyperglycemia and a strong correlation between hyperglycemia and poor clinical outcomes, they could not establish whether hyperglycemia per se was a contributor to worse clinical outcomes or was merely a surrogate marker

FIGURE 3.

REDUCTION OF ICU MORBIDITY WITH INTENSIVE INSULIN TREATMENT



ICU indicates intensive care unit; error bars indicate 95% confidence intervals.

[a] $P<0.01$.

[b] $P<0.0001$.

Adapted with permission from Van den Berghe G: Beyond diabetes: Saving lives with insulin in the ICU. *Int J Obes* 26(Suppl 3):S3–S8, 2002.

of disease severity. To answer this question, a growing number of intervention studies are reporting decreased mortality and morbidity when hyperglycemia is treated aggressively.

The best-designed randomized controlled trial was performed in a heterogeneous population of surgical ICU patients undergoing surgical, trauma, transplantation, and coronary procedures in Belgium. At admission to the ICU, 1,548 patients were randomly assigned to receive either intensive or conventional insulin treatment. In the conventional-treatment group, a continuous infusion was started only if blood glucose exceeded 215 mg/dl. In the intensive-treatment group, an insulin infusion was started if blood glucose exceeded 110 mg/dl. Compared with conventional therapy, adjusted to a mean blood glucose of 153 ± 33 mg/dl, intensive insulin therapy kept blood glucose at 103 ± 19 mg/dl and was associated with an increase in overall survival of 34% (Figure 2). This effect was observed exclusively in those patients who stayed in the ICU for more than 5 days. The greatest reduction in mortality involved deaths due to multiple-organ failure with a septic focus, documented on postmortem examination.

Intensive insulin treatment also dramatically reduced the risk of several morbid clinical outcomes when compared with conventional treatment (Figure 3). The patients in the intensive-therapy group were less likely to require prolonged use of antibiotics, an effect that was largely attributable to the 46% reduction in the incidence of bloodstream infections. Fewer patients in the intensive-therapy group required prolonged ventilatory support; this decrease might be partly explained by the 44% reduction in the severity of critical-illness polyneuropathy. This study demonstrated a linear correlation between the risk of critical-illness polyneuropathy and mean blood glucose level. A 50% reduction in median number of red blood cell transfusions and a 41% reduction in the incidence of acute renal failure requiring dialysis or hemofiltration were also demonstrated among the patients in the intensive insulin-treatment group. The benefits of intensive insulin treatment were not limited to patients with a known history of diabetes, as only 13% of the study population had a history of diabetes in this study.

In the Diabetes and Insulin–Glucose Infusion in Acute Myocardial Infarction (DIGAMI) study, a randomized, multicenter trial, aggressive treatment of hyperglycemia among diabetic patients reduced short-term and long-term mortality, with a benefit that extended to at least 3.4 years. Of 620 patients with diabetes and AMI, 306 were randomly

FIGURE 4.

YALE INSULIN INFUSION PROTOCOL

The following insulin infusion protocol is intended for use in hyperglycemic adult patients in an ICU setting, but is not specifically tailored for those individuals with diabetic emergencies such as diabetic ketoacidosis or hyperglycemic hyperosmolar states. When these diagnoses are being considered, or if blood glucose is ≥500 mg/dl, a physician should be consulted for specific orders. Also, notify a physician if the response to the insulin infusion is unusual or unexpected or if any situation arises that is not adequately addressed by these guidelines.

Initiating an Insulin Infusion

INSULIN INFUSION: Mix 1 unit (U) Regular human insulin per 1 ml 0.9% NaCl. Administer by infusion pump (in increments of 0.5 U/hour).

PRIMING: Flush 50 ml of infusion through all IV tubing before infusion begins (to saturate the insulin-binding sites in the tubing).

TARGET BLOOD GLUCOSE LEVELS at 100–139 mg/dl.

BOLUS AND INITIAL INSULIN INFUSION RATE: Divide the initial blood glucose level by 100, then round to the nearest 0.5 U for the bolus and initial infusion rate.

Examples:
a. If initial blood glucose = 325 mg/dl: 325 ÷ 100 = 3.25; round up to 3.5; IV bolus should be 3.5 U and infusion should be started at 3.5 U/hour.
b. If initial blood glucose = 174 mg/dl: 174 ÷ 100 = 1.74; round down to 1.5; IV bolus should be 1.5 U and infusion should be started at 1.5 U/hour.

Blood Glucose Monitoring

1. Check blood glucose hourly until stable (three consecutive values within the target range). In hypotensive patients, capillary blood glucose (i.e., fingersticks) may be inaccurate, but obtaining the blood sample from an indwelling vascular catheter is acceptable.
2. Check blood glucose every 2 hours once stable for 12–24 hours. Blood glucose checks can then be spaced to every 4 hours IF:
a. the patient has no significant change in clinical condition and
b. no significant change in nutritional intake.
3. If any of the following occur, consider temporarily resuming hourly blood glucose monitoring until blood glucose is again stable (two to three consecutive blood glucose values within the target range):
a. any change in insulin infusion rate (i.e., blood glucose out of target range)
b. significant changes in clinical condition

TABLE A.

INSTRUCTIONS FOR CHANGING THE INSULIN INFUSION RATE

BG 75–99 mg/dl	BG 100–139 mg/dl	BG 140–199 mg/dl
		BG ↑ by >50 mg/dl/hr
	BG ↑ by >25 mg/dl/hr	BG ↑ by 1–50 mg/dl/hr or BG unchanged
BG ↑	BG ↑ by 1–25 mg/dl/hr, BG unchanged, or BG ↓ by 1–25 mg/dl/hr	BG ↓ by 1–50 mg/dl/hr
BG unchanged or BG ↓ by 1–25 mg/dl/hr	BG ↓ by 26–50 mg/dl/hr	BG ↓ by 51–75 mg/dl/hr
BG ↓ by >25 mg/dl/hr: see below[b]	BG ↓ by >50 mg/dl/hr	BG ↓ by >75 mg/dl/hr

BG indicates blood glucose.

[a] For specific changes (Δ) in infusion rate, consult Table B.

[b] Discontinue insulin infusion and check blood glucose every 30 minutes. When blood glucose is ≥100 mg/dl, restart infusion at 75% of most recent rate.

c. initiation or cessation of pressor or steroid therapy
d. initiation or cessation of renal replacement therapy (e.g., hemodialysis, continuous venovenous hemofiltration)
e. initiation, cessation, or rate change in nutritional support (e.g., total or partial parenteral nutrition, tube feedings)

Changing the Insulin Infusion Rate

If blood glucose is <50 mg/dl:

Discontinue insulin infusion

Give 1 amp (25 g) D50 IV; recheck blood glucose every 15 minutes.

When blood glucose ≥100 mg/dl, wait 1 hour, then restart the insulin infusion at 50% of the original rate.

If blood glucose is 50–74 mg/dl:

Discontinue insulin infusion

If *symptomatic* (or unable to assess), give 1 amp (25 g) D50 IV; recheck blood glucose every 15 minutes.

If *asymptomatic*, give 1/2 amp (12.5 g) D50 IV or 8 ounces juice; recheck blood glucose every 15–30 minutes.

When blood glucose ≥100 mg/dl, wait 1 hour, then restart the insulin infusion at 75% of the original rate.

If blood glucose is ≥75 mg/dl:

Determine the *current blood glucose level*, identified as a column in Table A (75–99, 100–139, 140–199, or ≥200 mg/dl).

Determine the *rate of change* from the previous blood glucose level, identified as a cell in Table A, then move to the right column for instructions. Refer to Table B as needed.

Note: If the last blood glucose was measured 2–4 hours before the current blood glucose, calculate the *hourly* rate of change. Example: If the blood glucose at 2 P.M. was 150 mg/dl and the blood glucose at 4 P.M. is now 120 mg/dl, the total change over 2 hours is –30 mg/dl; however, the hourly change is –30 mg/dl ÷ 2 hours = –15 mg/dl/hour.

Adapted with permission from Goldberg PA, et al: Implementation of a safe and effective insulin infusion protocol in a medical intensive care unit. *Diabetes Care* 27(2):461–467.

BG ≥200 mg/dl	INSTRUCTIONS[a]
BG ↑	↑ INFUSION by "2Δ"
BG unchanged or BG ↓ by 1–25 mg/dl/hr	↑ INFUSION by "Δ"
BG ↓ by 26–75 mg/dl/hr	NO INFUSION CHANGE
BG ↓ by 76–100 mg/dl/hr	↓ INFUSION by "Δ"
BG ↓ by >100 mg/dl/hr	HOLD x 30 min, then ↓ INFUSION by "2Δ"

TABLE B.

CHANGES IN INFUSION RATE ("Δ") AS DETERMINED BY THE CURRENT RATE

Current Rate (U/hour)	Δ = Rate Change (U/hour)	2Δ = 2 × Rate Change (U/hour)
<3.0	0.5	1
3.0–6.0	1	2
6.5–9.5	1.5	3
10–14.5	2	4
15–19.5	3	6
20–24.5	4	8
≥25	≥5	10 (consult M.D.)

assigned to intensive treatment, including infusion of glucose–insulin–potassium to maintain blood glucose at less than 200 mg/dl, followed by a regimen of subcutaneous insulin injections for 3 or more months after discharge. The remaining 314 patients didn't receive insulin unless it was deemed clinically indicated, and then only in a conventional fashion. At 1 year, mortality was reduced by 29% in the intensive-therapy group in comparison with the conventional-treatment group. At 3.4 years of follow-up, the absolute reduction in mortality was 11%, meaning that one life was saved for every nine patients treated.

In the DIGAMI study, the significant difference in mortality between groups was found despite a relatively modest difference in blood glucose between the groups. This suggests that the beneficial effect of insulin treatment may be mediated through mechanisms other than the restoration of euglycemia. However, studies have also shown that control of blood glucose levels, rather than absolute levels of exogenous insulin, explains the mortality benefit associated with intensive insulin therapy. At present, it is unclear to what extent restoring euglycemia or providing insulin therapy is responsible for the observed beneficial effects. It is also difficult to determine from the DIGAMI study whether it was the short-term insulin–glucose infusion or the outpatient multi-dose insulin regimen (or both) that imparted the benefit. The ongoing DIGAMI-2 study will hopefully answer this important question.

Observational studies from Portland, Oregon have demonstrated a strong correlation between the introduction of a continuous intravenous insulin infusion protocol (IIP) and improved clinical outcomes among cardiac surgery patients. During a 15-year period, management of hyperglycemia after cardiothoracic surgery at the Providence St. Vincent Medical Center in Portland was intensified from intermittent subcutaneous insulin injections to a continuous insulin infusion. After IIP was implemented in 1991, the target for blood glucose dropped sequentially, reaching 125–175 mg/dl in 1999 and 100–150 mg/dl in 2001. Initially, these authors reported a 59% reduction in the incidence of deep sternal wound infections among patients treated with an intensive IIP in comparison with historic controls. In an updated report on 2,612 diabetic patients in 2003, hyperglycemic cardiac surgery patients treated with IIP during the first 3 postoperative days demonstrated reductions in absolute and risk-adjusted mortality of 57% and 50%, respectively. Although this was mainly a retrospective study, with the inherent limitations of comparing a new intervention with outcomes in historic controls, the Portland protocol is widely acknowledged for increasing awareness of the need for better glycemic control after cardiothoracic surgery.

In light of this new evidence, which is mainly in critically ill, postoperative patients, the ADA and the American College of Endocrinology have released new recommendations for the management of diabetes and hyperglycemia in the hospital. For ICU patients, the recommended goal for blood glucose is lower than 110 mg/dl (6.1 mmol/liter), and a continuous intravenous insulin infusion is the preferred choice of therapy. Because of the continuous relationship between blood glucose level and ICU mortality, it is reasonable to conclude that intensive insulin treatment among *all* critically ill patients should be targeted to near-normal levels. Admittedly, however, no outcome data are yet available in critically ill patients in medical ICUs. Because such patients tend to be older than patients in surgical ICUs with more multisystem dysfunction, the benefits (and potentially even the risks) of intravenous insulin therapy may be greater. Ongoing trials will shed further light on this issue. Until more data is available, however, the optimal glycemic target in medical ICU patients is yet to be defined.

Despite the common fear of hypoglycemia, multiple IIPs have proven safe and effective in medical and surgical ICUs. We recently described a nurse-driven protocol that was validated in the medical ICU to significantly improve glucose control with minimal risk of hypoglycemia (Figure 4, page 34).

Conclusions

Patients who are hyperglycemic at hospital admission, independent of a history of diabetes, represent a high-risk population. Strict glycemic control among these and other critically ill patients significantly reduces mortality and morbidity. Achieving euglycemia in the ICU is not easy and requires active collaboration among critical-care physicians, endocrinologists, nurses, and pharmacy staff. The development of a standard IIP can facilitate this process and improve outcomes among the critically ill.

Drs. Sakharova and Goldberg are postdoctoral fellows in endocrinology and metabolism and Dr. Inzucchi is Professor of Medicine and Clinical Director of the Section of Endocrinology at Yale University School of Medicine in New Haven, Connecticut.

Suggested Reading

1. Clement S, Braithwaite SS, Magee MF, Ahmann A, Smith EP, Schafer RG, Hirsh IB; American Diabetes Association Diabetes in Hospitals Writing Committee: Management of diabetes and hyperglycemia in hospitals. *Diabetes Care* 27(2):553–597, 2004.
2. American College of Endocrinology Task Force on Inpatient Diabetes and Metabolic Control: Position statement on inpatient diabetes and metabolic control. Available at: www.aace.com/pub/ICC/ACEPosiStat.pdf. Last accessed: August 10, 2004.
3. Umpierrez GE, Isaacs SD, Bazargan N, You X, Thaler LM, Kitabchi AE: Hyperglycemia: An independent marker of in-hospital mortality in patients with undiagnosed diabetes. *J Clin Endocrinol Metab* 87:978–982, 2002.
4. Krinsley JS: Association between hyperglycemia and increased hospital mortality in a heterogeneous population of critically ill patients. *Mayo Clin Proc* 78:1471–1478, 2003.
5. Capes SE, Hunt D, Malmberg K, Gerstein HC: Stress hyperglycaemia and increased risk of death after myocardial infarction in patients with and without diabetes: A systematic overview. *Lancet* 355(9206):773–778, 2000.
6. Stranders I, Diamant M, Van Gelder RE, Spruijt HJ, Twisk JW, Heine RJ, Visser FC: Admission blood glucose level as risk indicator of death after myocardial infarction in patients with and without diabetes mellitus. *Arch Intern Med* 164:982–988, 2004.
7. Wahab NN, Cowden EA, Pearce NJ, Gardner MJ, Merry H, Cox JL; ICINS Investigators: Is blood glucose an independent predictor of mortality in acute myocardial infarction in the thrombolytic era? *J Am Coll Cardiol* 40:1748–1754, 2002.
8. Norhammar A, Tenerz A, Nilsson G, Hamsten A, Efendic S, Ryden L, Malmberg K: Glucose metabolism in patients with acute myocardial infarction and no previous diagnosis of diabetes mellitus: A prospective study. *Lancet* 359(9324):2140–2144, 2002.
9. Van den Berghe G, Wouters P, Weekers F, et al: Intensive insulin therapy in critically ill patients. *N Engl J Med* 345: 1359–1367, 2001.
10. Zerr KJ, Starr A: Continuous intravenous insulin infusion reduces the incidence of deep sternal wound infection in diabetic patients after cardiac surgical procedures. *Ann Thorac Surg* 67:352–362, 1999.
11. Furnary AP, Gao G, Grunkemeier GL, Wu Y, Zerr KJ, Bookin SO, Floten HS, Starr A: Continuous insulin infusion reduces mortality in patients with diabetes undergoing coronary artery bypass grafting. *J Thorac Cardiovasc Surg* 125:1007–1021, 2003.
12. Malmberg K, Norhammer A, Wedel H, Ryden L: Randomized trial of insulin-glucose infusion followed by subcutaneous insulin treatment in diabetic patients with acute myocardial infarction (DIGAMI study): Effects on mortality at 1 year. *J Am Coll Cardiol* 26(1):57–65, 1995.
13. Malmberg K, Norhammar A, Wedel H, Ryden L: Glycometabolic state at admission: Important risk marker of mortality in conventionally treated patients with diabetes mellitus and acute myocardial infarction: Long-term results from the Diabetes and Insulin-Glucose Infusion in Acute Myocardial Infarction (DIGAMI) study. *Circulation* 99(20): 2626–2632, 1999.
14. Goldberg PA, Siegel MD, Sherwin RS, Halickman JI, Lee M, Bailey VA, Lee SL, Dziura JD, Inzucchi SE: Implementation of a safe and effective insulin infusion protocol in a medical intensive care unit. *Diabetes Care* 27:461–467, 2004.

Diabetes Care and the Risk of Malpractice Suits

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A recent article in the journal *Trial* stated that malpractice cases related to the care of diabetes are rare (1). Our experience with a large New York State malpractice firm gives the antithesis of this impression. Any disease that can cause serious outcomes in a large patient population is a fertile area for malpractice litigation. Diabetes is such a disease.

Why Suits Arise

Diabetic complications occur with the greatest frequency in poorly managed patients, but they can occur even in scrupulously managed patients. The development and management of complications provide attorneys with a fertile field for litigation. A bad outcome is often the only criterion necessary to initiate a lawsuit. Attorneys employ highly qualified physicians to review charts and compile a list of major and minor deviations in patient care. Nonphysician juries must then relate the alleged deviations to the alleged injuries in complex medical cases.

The number of lawsuits against diabetologists is amplified because patients with diabetes, especially elderly patients, visit multiple physicians. Attorneys spread a "wide net" in malpractice cases to avoid missing any physician who might be involved in the patients' care before the running of the statute of limitations rule. Thus, when the cardiologist, nephrologist, podiatrist, or neurologist of a patient with diabetes is sued, it is very likely that the diabetologist will be sued also. Multiple defendants increase the prospect of maximizing the patient's (and attorney's) dollar award. Even if the diabetologist is eliminated from the suit as it progresses, the malpractice company will have spent a significant amount of money preparing to protect the insured.

Types of Suits

Some common clinical problems are particularly likely to precipitate lawsuits against diabetologists by plaintiffs with diabetes. Each problem has a unique set of community standards and guideline recommendations. The failure to meet "established modes of behavior" in a reasonable and often exquisite fashion results in a long list of allegations, which the physician receives in a Bill of Particulars.

A review of malpractice suits against diabetologists and nondiabetologists caring for patients with diabetes reveals a broad and consistent pattern of allegations. These include the following:

History
- Inadequate initial history, with failure to record and act on important information
- Failure to update history at follow-up visits (e.g., hospitalizations, other physician care, changes in medication)
- Misinterpretation of historical information
- Lack of focused history of systems commonly involved in diabetes (e.g., eyes, heart, feet, nervous system, vascular system) at follow-up visits

Patient Education
- Failure to follow standard educational guidelines (including issues related to home glucose monitoring and the diabetologist's review of the patient's monitoring schedule)
- Patient injury as a result of lack of information or misinformation

Cerebrovascular Disease
- Failure to evaluate symptoms in a timely fashion
- Failure to refer to consultants for appropriate studies and management in a timely manner
- Erroneous interpretation of signs and symptoms
- Absence of a neurologic and vascular examination
- Failure to control cholesterol and triglycerides
- Inadequate control of hypertension

Cardiovascular Disease
- Misinterpretation of history
- Failure to perform cardiac system review at follow-up visits
- Failure to treat elevated cholesterol and triglyceride levels before the development of clinically symptomatic arteriosclerosis
- Inadequate treatment of hypertension
- Delay or absence of immediate cardiac evaluation with onset of new symptoms, resulting in disability or death
- Erroneous telephone instructions given by office staff or physician to a patient with unrecognized cardiac symptoms