

HOSPITAL HYPOGLYCEMIA: NOT ONLY TREATMENT BUT ALSO PREVENTION

*Susan S. Braithwaite, MD, FACE, FACP, Michelle M. Buie, BSN,
Cara L. Thompson, MSN, APRN, CDE, Douglas F. Baldwin, PharmD,
Maryanne D. Oertel, PharmD, BCPS, Beverly A. Robertson, MT(ASCP),
and Hetal P. Mehrotra, RD, MHA, CPHQ*

ABSTRACT

Objective: To propose a strategy, applicable on general hospital wards, for prevention of hypoglycemia in hospitalized patients.

Results: Although the mortality rate among hospitalized patients with hypoglycemia has been shown to be 22.2 to 27% in series that included patients with diabetes, some investigators have shown that hypoglycemia is not an independent predictor of mortality. Outside the critical care setting, the comparative risks of hyperglycemia and hypoglycemia and the relationship of hospital hypoglycemia to intensification of glycemic control have not been determined. The reported incidence of hospital hypoglycemia ranges from 1.2% for hospitalized adults to 20% for nonpregnant patients with diabetes admitted without a metabolic emergency. Among patients receiving antihyperglycemic therapy, the literature describes precipitating events—usually a sudden change of caloric exposure—and predisposing conditions for hypoglycemic episodes.

Conclusion: Hospital hypoglycemia is predictable, and it is preventable by measures other than undertreatment of hyperglycemia. Physician orders for antihyperglycemic therapy should be written and, if necessary, be revised so as to respond to the presence of predisposing conditions for hypoglycemia. A ward-based protocol or hospital-wide policy should establish the appropriate response to triggering events. Within the time frame of action of previously administered antihyperglycemic drugs (after abrupt interruption of caloric exposure), the threshold for preventive intravenous administration of dextrose is a glucose concentration of 120 mg/dL. (*Endocr Pract.* 2004;10[Suppl 2]:89-99)

Abbreviations:

DCCT = Diabetes Control and Complications Trial;
D50W = 50% dextrose in water; **NPO** = nothing by mouth; **TPN** = total parenteral nutrition

INTRODUCTION

At the time of completion of the Diabetes Control and Complications Trial (DCCT), Cryer et al (1-3) observed that, among patients having type 1 diabetes, iatrogenic hypoglycemia was the principal barrier that prevented attainment of normoglycemia. In some patients with both type 1 and type 2 diabetes, episodes of hypoglycemia develop during normal ambulatory living conditions, without warning symptoms and without major deviations from usual meal plans, activities, or insulin therapy (1-3). Understanding of the pathogenesis of hypoglycemia among these ambulatory patients sometimes requires subtle comprehension of physiologic processes involving the concept of alteration of the threshold for neurogenic responses to hypoglycemia, with resultant defects of counterregulation and hypoglycemia unawareness (4,5).

In contrast, among hospitalized patients, triggering events for hypoglycemia often are recognizable by laypersons and are readily understood to be a direct consequence of a mismatch between antihyperglycemic therapy and hospital routine. Patients are at risk, not from hypoglycemia unawareness but rather from anesthesia, analgesia, illnesses that alter consciousness, medical and surgical interventions, nothing by mouth (NPO) status, or separation from familiar resources used at home, resulting in inability to self-report symptoms or defend against hypoglycemia (or both). Prevention of hypoglycemia among hospitalized inpatients depends on matching antihyperglycemic therapy appropriate for the patient's medical condition to nutritional intake, coupled with conventional monitoring of blood glucose concentration and appropriate caregiver responses (6-10). The following discussion will focus on hypoglycemia—not as a primary disorder or spontaneous complication of another illness that might arise in the hospitalized patient but as a complication of antihyperglycemic therapy (11-43). Rather than focus on hypoglycemia as an indicator of patient safety or quality of care (44-58), the purpose of this report is to describe hypoglycemia as a familiar everyday impediment to the ability or willingness of caregivers to address a problem that seems to be a greater threat to patient safety—namely, hyperglycemia (59-83).

From the University of North Carolina, Chapel Hill, North Carolina.
Presented at the American College of Endocrinology Inpatient Diabetes and Metabolic Control Conference, Washington, DC, December 14 and 15, 2003.
© 2004 AACE.

Most likely, the greatest risk in the hospital for prolonged, unrecognized hypoglycemia exists on general wards among patients who are receiving subcutaneous insulin therapy. An argument will be made that hypoglycemia is both predictable in these patients and also preventable by means other than undertreatment of hyperglycemia. Preventive strategies should include the use of not only physician orders responsive to predisposing conditions but also ward-based protocols or a hospital policy by which nursing staff may be responsive to triggering events for hospital hypoglycemia.

HOSPITAL HYPOGLYCEMIA—A MARKER FOR COMORBIDITY

In four observational reports that are broadly inclusive of hospitalized patients, including patients without diabetes and admissions on general wards, hypoglycemia was associated with poor outcomes. In a study of 7,763 hospitalized adults reported by Fischer et al (19), 94 patients had 137 episodes of hypoglycemia, and the hospital mortality rate was 27% among the patients with hypoglycemia. In a study of 5,491 hospital admissions reported by Stagnaro-Green et al (48), 80 patients had 106 episodes of hypoglycemia in comparison with 104 patients having 166 episodes of severe hyperglycemia. The mortality rates were 22.2% and 11.1%, respectively. In a retrospective case-control study of hospitalized older adults without diabetes in whom hypoglycemia developed, Shilo et al (30) showed higher in-hospital mortality among 60 patients with hypoglycemia than in 83 control subjects (48% versus 18.1%, respectively). Hypoglycemia remained a predictor of mortality even after adjustment for other risk factors. The preceding three reports indicated that no case fatality was a direct consequence of hypoglycemia. Among 5,404 hospitalized elderly patients, Kagansky et al (43) found 281 patients with hypoglycemia. The mortality in the hypoglycemic group was 26% in comparison with 14% in the patients without hypoglycemia. In a multivariate analysis, hypoglycemia was not an independent predictor of mortality. Hospital hypoglycemia was judged by these investigators (43) to be a marker of poor health without a direct effect on survival.

In epidemiologic studies, hypoglycemia may fail to emerge as a major determinant of inpatient morbidity or mortality. In individual cases, however, adverse outcomes (seizures, alteration of vital signs, permanent neurologic injury, or death) may be traceable to iatrogenic hypoglycemia (11-13,18,20,26-28,31,55).

In hospitalized patients, we found no controlled randomized studies designed to assess the effect of a strategy of hypoglycemia avoidance on outcomes. Therefore, although hospital hypoglycemia has been associated with increased mortality, the causal relationship is unknown. One tenable hypothesis, supported by the aforementioned observational study reported by Kagansky et al (43), would be that hypoglycemia itself may not independently

cause an increase in mortality or morbidity within a hospital population, but rather that other conditions that increase mortality (such as hyperglycemia necessitating treatment, sepsis, heart failure, malnutrition, and renal failure) may contribute to the occurrence of hypoglycemia. Another tenable hypothesis would be that hypoglycemia is directly responsible for increased morbidity and mortality. Even in the absence of permanent neurologic effects, conceivably, transient alterations of sensorium could lead to injuries or events that ultimately affect outcomes. A third tenable hypothesis might be that hypoglycemia limits the ability or willingness of caregivers to control hyperglycemia. The adverse effects of hyperglycemia are demonstrable from outcome trials conducted in large populations of hospitalized patients, but in the case of an individual patient, these harms (being multifactorial in origin) can be said, at worst, to result from “acts of omission” with respect to glycemic management. In contrast, the adverse results attributable to severe hypoglycemia may be directly and uniquely traceable to iatrogenic causes. Severe hypoglycemia is a treatment-related complication that is dreaded by physicians and patients alike.

HOSPITAL HYPOGLYCEMIA—THE PRINCIPAL BARRIER TO HOSPITAL NORMOGLYCEMIA?

Uncertainty About Comparative Risks and Benefits on General Wards

In the care of critically ill patients and those who have undergone cardiac operations, hypoglycemia occurs but is promptly recognized and treated. In this setting, the benefits that result from prevention of mild hyperglycemia outweigh any risk of hypoglycemia (10,63-65,68,69,71-74,76,77,79-83). Indeed, reports from intensively monitored hospitalized patients who receive intravenous treatment with insulin infusion under protocol suggest that hypoglycemia is an insignificant problem. In striking contrast, during intensification of management of diabetes among ambulatory patients, both the DCCT and the United Kingdom Prospective Diabetes Study (UKPDS) determined that hypoglycemia was a clinically important or even prohibitive problem limiting the attainment of strict glycemic control and that hypoglycemia itself sometimes threatened the safety of intensively managed patients (59,60,62,66). Hypoglycemia associated with intensive diabetes care resulted in significant costs to a large midwestern health-care plan (42). In the care of patients on general hospital wards, no data are currently available from prospective trials on the comparative risks of adverse outcomes associated with hypoglycemia and hyperglycemia.

The frequency of hospital hypoglycemia cannot be readily determined. In comparison with intravenous insulin infusion therapy provided in critical care units, therapy with orally administered antihyperglycemic drugs or subcutaneous administration of insulin in general

hospital areas probably is associated with a greater likelihood of hypoglycemia. When staffing is limited, the likelihood that hypoglycemia will be undetected, prolonged, or severe may also be greater. The reported incidence of hypoglycemia differs on the basis of the specific hospital population being studied, ranging from 1.2% for adults (19) to 1.5% for a general hospital population (48), 5.2% for elderly patients (43), or 20% for nonpregnant patients with diabetes on general hospital wards admitted without a metabolic emergency (58).

One method of ascertaining hypoglycemic episodes is to monitor the frequency of hypoglycemia in a population at risk, such as a group of patients with the associated risk factors of advanced age (25,26), use of high-risk medications (11,25,26), renal failure (22,33,35,36,41), malnutrition or requirement for nutritional support (84), or hospital admission because of a hyperglycemic emergency (21).

The frequency of hypoglycemia, like any adverse event, is most likely underestimated by adverse event reporting. Removal of 50% dextrose from an emergency supply system without a physician order may be a useful signal to help quantify the problem of hypoglycemia (45,46,51,54,56,57). At the university of North Carolina Hospitals in Chapel Hill, a large University hospital that had 30,984 patient discharges for the year 2002, the records in the pharmacy department indicate that purchases of 50% dextrose in water (D50W) for all indications have been fairly consistent for the past 5 years. In a recent 12-month period (November 1, 2002, to October 30, 2003), the pharmacy department dispensed a total of 2,487 syringes or vials containing 25 g (50 mL) of D50W to patients, an average of approximately 200 per month (reviews of medical records have not been conducted to determine the purpose or to confirm that administration occurred).

Outside the intensive care setting, the task of relating the frequency of hypoglycemia to attempts to control hyperglycemia is not straightforward. Episodes of hypoglycemia are not necessarily detected by methods that are used for study of hyperglycemia, such as retrospective time-weighting of results, prospective sampling (followed by averaging), assessment of the distribution of the glucose concentration in each of several ranges, or ascertainment of the proportion of time above a specific glycemic threshold. In the general hospital ward setting, very few studies reporting on the frequency of clinical episodes of hypoglycemia have included any comment on the adequacy of control of hyperglycemia in the same general population or the same individual patients, except to record the frequency of episodes of severe hyperglycemia (47,48, 58,75).

The glycemic target range for critically ill patients has been discussed throughout this conference (76,80,83). Appropriate glycemic targets for various hospital populations outside the critical care unit, and the safety of subcu-

aneous insulin regimens aimed at those targets, have not been determined.

Fear of Hypoglycemia

Because of the potential for hypoglycemic episodes, hospitalization is feared by many patients with diabetes (17,19). It may be for fear of the occurrence of hypoglycemia during intensification of therapy that surgical services and other caregivers sometimes are reluctant to request an endocrine consultation.

With apparent reference to the risk of hypoglycemia, the pharmacy, medical informatics, and safety literature about diabetes identifies insulin as a "high alert" medication (44,49-52,54,56,57). Although the best quality improvement programs attempt to quantify both hyperglycemia and hypoglycemia (47,48), some such programs that consider diabetes probably examine mainly hypoglycemia.

Out-of-court settlements will likely preclude access to information about the relative prevalence of malpractice claims relating to hypoglycemia or hyperglycemia. Iatrogenic hypoglycemia results from "acts of commission" (50,85). It has been pointed out that "cases are especially hard to defend when adverse reactions are allowed to proceed in an institutionalized patient..." (85). Knowing that both hyperglycemia and hypoglycemia can be associated with adverse results, institutions and individual practitioners may suspect that liability is ascribed mainly to the latter.

The foregoing situations collectively exert a chilling effect upon willingness of staff to intensify antihyperglycemic therapy.

PREDICTABILITY OF HOSPITAL HYPOGLYCEMIA

Predisposing Conditions

Reported series of patients with hypoglycemia occurring initially in the ambulatory or hospital setting disclose patterns of predisposing conditions and treatments that are relevant to the hospitalized patient (18,25,26,28,31,40,43).

In the series of 137 inpatient episodes of hospital hypoglycemia with serum glucose levels of 49 mg/dL or less reported by Fischer et al (19), the following coexisting conditions were identified: (1) diabetes; (2) insulin therapy for diabetes, insulin therapy for hyperkalemia, or total parenteral nutrition (TPN)-associated hyperglycemia; (3) chronic renal insufficiency; (4) liver disease; (5) neoplasms; (6) burns; (7) infections; (8) shock; (9) pregnancy; and (10) alimentary disease. Of the 46 patients with chronic renal failure, 20 also had diabetes mellitus. In 90% of cases, insulin treatment had been used. Among the subgroup of patients treated with antihyperglycemic medication, inappropriate adjustment of the insulin dose was the proximate cause of 39% of episodes of hospital hypoglycemia. The inappropriate adjustments included treat-

ment of ketoacidosis or hyperosmolar coma, attempts at tighter metabolic control, “sliding-scale” treatment with use of doses too high for renal insufficiency, reduced requirement during resolution of infection, and failure to decrease the dose of insulin after one hypoglycemic episode. In a series of 106 episodes of hospital hypoglycemia that occurred among 80 patients described by Stagnaro-Green et al (48), risk factors for hypoglycemia included infection, sepsis, cancer, renal insufficiency, liver disease, neonatal status, and corticosteroids. Only 17 patients had diabetes, and the relationship to antihyperglycemic drug therapy was not determined. Inadequate oral intake was identified in 68% of the survivors and in 88% of the nonsurvivors. Among patients without diabetes, Shilo et al (30) identified hypoalbuminemia (serum albumin level less than 3 g/dL), liver disease, renal insufficiency, malignant condition, congestive heart failure, and sepsis as predictors of hypoglycemia. Kagansky et al (43) found that 70 of 281 patients with hospital hypoglycemia were taking insulin or sulfonylureas. Furthermore, they found that the concomitants of hypoglycemia included female sex, sepsis, malignant lesions, stroke, diabetes, treatment with insulin or a secretagogue, dementia, hypoalbuminemia, high serum creatinine concentration, and high serum alkaline phosphatase level.

Conversely, specific treatments or medical conditions have been examined for their relationship to hypoglycemia. Among patients treated with sulfonylureas, the highest rate of serious hypoglycemia has occurred among users of glyburide (25,34). Consequently, our health system (University of North Carolina Hospitals) has removed this drug from its formulary. Renal failure, peritoneal dialysis, hemodialysis, the coexistent presence of sepsis, and the use of antihyperglycemic agents and other drugs (for example, aspirin, β -adrenergic blocking agents, and propoxyphene) in the treatment of patients with renal failure have been linked to hypoglycemia (14,16,22,33,35-39,41). Conversions between intravenous therapy or NPO status and a transitional meal plan can impose a risk of hypoglycemia, especially among patients with uncertain oral intake. The risk of hypoglycemia at the time of termination of TPN perhaps has been overestimated; nevertheless, insulin therapy for hyperglycemia during enteral and parenteral nutrition creates the need for special prescribing precautions because of the possibility of sudden discontinuation of feedings (84,86-94).

Elderly patients are at special risk of hypoglycemia (25,26,41). Recovery from a metabolic emergency sometimes is complicated by hypoglycemia (21). Other conditions include those that predispose to hypoglycemia in the nonhospitalized patient with diabetes—such as alcoholism, hypoglycemia unawareness, tapering of glucocorticoid dose, polypharmacy or drug interactions, the use of certain drugs (23,34,53), adrenal or pituitary insufficiency, and pregnancy. A higher risk situation is created if, for any reason, the ability of the patient to self-report symptoms of hypoglycemia is altered.

Table 1
Predisposing Conditions and
Triggering Events for Occurrence of
Hospital Hypoglycemia
During Antihyperglycemic Therapy

Predisposing conditions

Renal insufficiency
Malnutrition
Liver disease
Sepsis
Shock
Pregnancy
Malignant lesion
Hyperkalemia
Total parenteral nutrition
Burns
Alimentary disease
Dementia
Congestive heart failure
Stroke
Alteration of ability of patient to self-report symptoms
Hypoglycemia unawareness or defective counterregulation
Old age
Recovery from metabolic emergency
Alcoholism
Concomitant drug interactions or polypharmacy
Tapering of glucocorticoid dose
Adrenal or pituitary insufficiency

Triggering events

Transportation off ward, causing meal delay
New “nothing by mouth” status
Interruption of intravenous dextrose therapy
Interruption of total parenteral nutrition
Interruption of enteral feedings
Interruption of continuous venovenous hemodialysis

Recognizing predisposing conditions that increase patient risk for iatrogenic hypoglycemia and making an appropriate therapeutic response are the responsibility of the primary physician or endocrinologist (Table 1).

Triggering Events

The only major study to undertake a direct assessment of proximate causes of hypoglycemia, as distinguished from predisposing risk factors, was the series reported by

Fischer et al (19). Among the patients with diabetes in that series, hypoglycemia frequently was attributable to decreased caloric intake related to illness or hospital routine. During treatment with insulin or orally administered agents, decreased intake of calories was the largest single proximate cause category, accounting for 45% of episodes of hypoglycemia. The causes included nausea, vomiting, anorexia, lethargy, NPO status for diagnostic tests or surgical procedures, interruption of enteral feedings for measurement of the residual, or nondelivery of meals (19). Of note, the possibility of hypoglycemia associated with each of these types of events might readily be addressed preventively by provision of calories in an alternative manner.

A current list of triggering events for hospital hypoglycemia resulting from decreased caloric exposure is shown in Table 1. The frontline observer of such triggering events often is the nurse.

PREVENTABILITY OF HOSPITAL HYPOGLYCEMIA

“Keeping patients a little sweet” is a precept that has been passed down in surgical training programs for decades as a preventive measure against hypoglycemia. Lacking results of research trials that would provide a risk-to-benefit analysis about tight glycemic control on general hospital wards, endocrinologists at least can emphasize that hypoglycemia in the hospital is preventable by measures other than undertreatment of hyperglycemia.

Monitoring

Currently available methods for hospital monitoring of glycemic control include laboratory determinations of glucose concentration in venous or arterial blood or point-of-care testing of venous, capillary, or arterial blood. Methods for continuous glucose monitoring have been developed (experimentally and in ambulatory care) that might eventually be applicable to the clinical care of hospitalized patients, especially those with altered consciousness (15,79,95).

Treatment

Articles from the nursing literature overwhelmingly discuss a reactive treatment strategy, not a proactive preventive strategy, for hypoglycemia (96-104). Similarly, other medical literature focuses on reactive treatment (29,92). Because hypoglycemia may cause seizures, alteration of vital signs, permanent neurologic injury, or even death, prompt treatment of incipient hypoglycemia is of critical importance. If the patient is conscious and able to eat, ingestion of 15 g of carbohydrate is less likely to result in overshoot hyperglycemia than the alternative therapy, 25 to 50 mL of 50% dextrose administered intravenously. Treatment is followed by retesting of the blood glucose level within approximately 15 to 20 minutes. For patients who are unconscious or who are in NPO status and lack

intravenous access, initial treatment may consist of glucagon, 1 mg intramuscularly, with appropriate follow-up testing and administration of carbohydrate. Institutional protocols may differ in detail. The principles are well known and are not the subject of the current discussion (92).

Hypoglycemia treatment orders from the physician, as well as nursing and hospital hypoglycemia treatment protocols, usually provide for administration of glucose at a threshold glucose level of 70 to 80 mg/dL.

Preventive Strategies

Patient self-management of diabetes in the hospital should be facilitated when appropriate. Staff understanding of new treatment modalities and the actions of new antihyperglycemic agents should be ensured through in-service training.

Orally administered antihyperglycemic agents should be prescribed with caution and with observance of changing organ function, potential drug interactions, and contraindications that might arise in the hospital. At the time of dismissal from the hospital, orally administered agents are associated with continued risk for hypoglycemia. Among patients 65 years of age or older who have a first episode of serious hypoglycemia during insulin or sulfonylurea use, Shorr et al (26) identified recent dismissal from the hospital as the major predictive factor for subsequent hypoglycemic episodes.

Caregivers, fearing hypoglycemia, may hope to prevent hypoglycemic episodes by substituting sliding-scale management for anticipatory insulin therapy. Standardization of such correction-dose algorithms may reduce the variability of physician practice, but an institutional strategy of issuing a standardized algorithm encourages the use of correction doses as monotherapy (sliding scale). This strategy will be ineffective or even harmful if used alone, without concomitant institutional encouragement of the use of a comprehensive, anticipatory treatment plan for hyperglycemia (88,105-107). The preferred approach would be to use measures for prevention of hypoglycemia that do not promote hyperglycemia (105-111).

As discussed in other sections of this conference, physicians should embed protections against hypoglycemia in their orders for scheduled insulin therapy. These protections might include the following: (1) appropriate selection of patients for use of intravenous insulin infusions (88); (2) provision of insulin intravenously or in the TPN bag to cover some or all of the requirements for insulin created by use of TPN (90); (3) inclusion of a short-acting insulin or a rapid-acting analogue to cover nutritional needs as part of a program of scheduled subcutaneous administration of insulin; (4) for patients with glycemic instability or patients in transition to a normal meal plan, the use of “hold” criteria for the short-acting insulin or rapid-acting analogue component of scheduled insulin therapy; (5) reliance on a structured program for enteral feedings or an individualized algorithm of graduat-

ed insulin doses that depend on tube feed rate (91,93); (6) avoidance of long-acting insulin for coverage of nutritional requirements (enteral feedings or intravenously administered dextrose); (7) restriction of the amount of peakless long-acting insulin to the estimated basal requirements of the patient; and (8) prescribing of a consistent carbohydrate diet, with matching of oral intake to the premeal dose of rapid-acting insulin analogue. For patients with transitional meal plans who have not yet advanced to a normal diet, or whose oral intake is not ensured, assessment of actual intake by a nutritionist is recommended. The plan for scheduled insulin treatment for hospitalized patients should be revised daily or twice daily on the basis of the clinical condition and the response to previous therapy.

Physicians need to react to *predisposing conditions* that create risk for hypoglycemia. While maintaining needed scheduled antihyperglycemic therapy, in the presence of predisposing conditions, physicians should order increased frequency of monitoring of blood glucose levels in an attempt to detect downward trending of glucose values that might be evident over a period of hours or several days. If the physician normally uses a specific threshold for withholding scheduled rapid-acting or regular insulin, the threshold for the withholding order should be increased during downward trending of glucose values. In response to a downward trend of blood glucose levels, doses of scheduled antihyperglycemic therapy should be reduced.

The nursing staff should be empowered under hospital policy or ward-based protocol, with minimal physician oversight, to respond with appropriate preventive actions after the occurrence of potential *triggering events* for hypoglycemia. Such triggering events usually involve abrupt interruption of caloric exposure. The aim of preventive actions is to maintain the blood glucose level in a target range of about 90 to 130 mg/dL.

Among patients who already have received orders for antihyperglycemic therapy, the protocol or policy might (1) define triggering events for hypoglycemia; (2) require interruption of antihyperglycemic therapy after a triggering event for hypoglycemia has occurred, until further physician orders are received (except for basal insulin for type 1 diabetes and correction doses of insulin for hyperglycemia); (3) require frequent blood glucose monitoring for the duration of action of previously administered antihyperglycemic drugs; and (4) provide for intravenous administration of dextrose during the time frame of action of previously administered antihyperglycemic drugs, before hypoglycemia actually occurs, at a threshold glucose level of 120 mg/dL.

A Multidisciplinary Approach to Prevention

The principal medical barrier to the prevention of hypoglycemia in the presence of triggering events is the need to define and maintain basal insulin therapy among patients having type 1 diabetes. A minor medical barrier is the need to establish strategies for dextrose replacement

that will meet variable patient volume restrictions, without producing overshoot hyperglycemia, and (in cases of transport of the patient off the assigned floor) provide a safety net for several hours. In most patients with glucose levels in the target range who are unable to eat, the prevention of hypoglycemia at the time of a triggering event will involve intravenous administration of dextrose (given as bolus therapy of concentrated dextrose or dextrose infusion) to maintain the target glucose range (90,91).

The principal administrative barrier to implementation of policies is the hospital-wide scope of the need for prevention of hypoglycemia, affecting many different types of patients, providers, and nursing units. Thus, a need exists for ward-based protocols and a hospital-wide policy. A multidisciplinary group should address medical and administrative barriers to implementation of a ward or hospital hypoglycemia prevention policy or protocol, including the following: (1) identification of patients as possibly having type 1 diabetes; (2) advance determination and recording of alternative basal insulin therapy for patients with type 1 diabetes; (3) determination of the time frame within which elements of the policy take effect, after occurrence of a triggering event (including initiation of alternative basal insulin therapy in relationship to previously scheduled insulin treatment); (4) calculation of the duration of action of previously administered antihyperglycemic therapy by the floor nurse in consultation with pharmacy; (5) determination of appropriate preventive dextrose therapy; (6) variation in physician practice patterns and existing ward-based or service-based protocols; (7) balancing physician adherence to protocol with physician autonomy and authority to override protocol; and (8) assessment of the comfort level of professional and administrative staff. The utility of a having a quality improvement team to answer and respond to concerns such as those in the foregoing itemization has been demonstrated in related contexts (47,75,110,112). Ideas that might be a springboard for discussion are shown in Appendices 1 and 2.

If the admitting physician or endocrine consultant indicates that a patient might have type 1 diabetes and records an alternative plan for basal insulin for the contingency of altered nutritional intake, this advance information will be helpful to cross-covering physicians, whose first impulse might otherwise be to cancel all scheduled antihyperglycemic therapy. Although paging systems within teaching hospitals probably are associated with relatively prompt house staff responses to calls (113), indirect paging systems in which a nurse or hospital operator is required to contact a physician's office or a private answering service, who then independently will contact the physician, are associated with slower responses than direct paging systems. In a study of two university-affiliated hospitals, 25% of pages originating from the intensive care unit that were placed through an indirect system were associated with a response time of 29 minutes or more

(114)—a delay that would be intolerable if a nurse recognized triggering events and foresaw an episode of hypoglycemia that might be prevented by timely action. It is appropriate for protection of nursing staff that their actions taken on an emergency basis on behalf of a patient should be covered under policy.

Using a ward-based protocol or hospital-based policy to prevent hypoglycemia is likely to improve upon even the best proactive efforts of individual physicians. Variations in physician practice patterns are likely to reduce quality, whereas standardization to excellence under policy is likely to ensure quality (115-118). In fact, at many hospitals and on many wards, at least some of the aforementioned components of a protocol for prevention of hypoglycemia already are the standard of practice.

CONCLUSION

Hospital hypoglycemia is a marker for comorbidities and can result in morbidity or mortality. The comparative risks of hypoglycemia and mild hyperglycemia on general hospital wards still need to be determined. Fear of hospital hypoglycemia is a barrier to prevention or correction of hyperglycemia. Many episodes of hypoglycemia in the hospital are predictable and preventable. Physician orders should include an appropriate response when the risk for hypoglycemia is increased by predisposing conditions. Ward-based protocols or hospital-wide policies should be developed that establish a response to triggering events. These triggering events usually involve a sudden change of caloric exposure among patients receiving antihyperglycemic therapy. The response to triggering events should include maintenance of basal insulin therapy for patients who may have type 1 diabetes and maintenance of correction doses of insulin for hyperglycemia, but otherwise interruption of antihyperglycemic therapy until further physician orders are received. In addition, for patients who are not eating during the time frame of action of previously administered antihyperglycemic drugs, the policy or protocol should require preventive intravenous administration of dextrose at a threshold glucose level in the mid- to high-target range, about 120 mg/dL.

REFERENCES

1. **Cryer PE.** Banting lecture: hypoglycemia; the limiting factor in the management of IDDM. *Diabetes.* 1994;43:1378-1389.
2. **Cryer PE, Childs BP.** Negotiating the barrier of hypoglycemia in diabetes. *Diabetes Spectrum.* 2002;15:20-27.
3. **Cryer PE, Davis SN, Shamooh H.** Hypoglycemia in diabetes. *Diabetes Care.* 2003;26:1902-1912.
4. **Boyle PJ, Kempers SF, O'Connor AM, Nagy RJ.** Brain glucose uptake and unawareness of hypoglycemia in patients with insulin-dependent diabetes mellitus. *N Engl J Med.* 1995;333:1726-1731.
5. **Smith D, Amiel SA.** Hypoglycaemia unawareness and the brain. *Diabetologia.* 2002;45:949-958.

6. **Hirsch IB, Paauw DS, Brunzell J.** Inpatient management of adults with diabetes. *Diabetes Care.* 1995;18:870-878.
7. **Ahmann A.** Comprehensive management of the hospitalized patient with diabetes. *Endocrinologist.* 1998;8:250-259.
8. **Levetan CS, Magee MF.** Hospital management of diabetes. *Endocrinol Metab Clin North Am.* 2000;29:745-770.
9. **Metchick LN, Petit WA Jr, Inzucchi SE.** Inpatient management of diabetes mellitus. *Am J Med.* 2002;113:317-323.
10. **Montori VM, Bistrian BR, McMahan MM.** Hyperglycemia in acutely ill patients. *JAMA.* 2002;288:2167-2169.
11. **Bauer HG.** Severe and prolonged hypoglycemic shock during sulfonylurea treatment. *Metabolism.* 1965;14:220-228.
12. **Arai J, Miyakawa Y, Kataoka K, Matsuki S, Asano S.** Case of fatal hypoglycemic coma during sulfonylurea administration for the therapy of steroid diabetes following adrenalectomy [article in Japanese]. *Saishin Igaku.* 1969;24:2524-2529.
13. **Seltzer HS.** Drug-induced hypoglycemia: a review based on 473 cases. *Diabetes.* 1972;21:955-966.
14. **Greenblatt DJ.** Fatal hypoglycaemia occurring after peritoneal dialysis. *Br Med J.* 1972;2:270-271.
15. **Pitkanen E, Koivula T.** Continuous blood glucose monitoring and characteristics of diabetes in patients on maintenance haemodialysis treatment. *Scand J Urol Nephrol.* 1979;13:309-312.
16. **D'Elia JA, Kaldany A, Miller DG, Rolla A, Weinrauch LA.** Elimination of requirement for exogenous insulin therapy in diabetic renal failure. *Clin Exp Dial Apheresis.* 1982;6:75-84.
17. **Goldgewicht C, Slama G, Papoz L, Tchobroutsky G.** Hypoglycaemic reactions in 172 type 1 (insulin-dependent) diabetic patients. *Diabetologia.* 1983;24:95-99.
18. **Malouf R, Brust JC.** Hypoglycemia: causes, neurological manifestations, and outcome. *Ann Neurol.* 1985;17:421-430.
19. **Fischer KF, Lees JA, Newman JH.** Hypoglycemia in hospitalized patients: causes and outcomes. *N Engl J Med.* 1986;315:1245-1250.
20. **Campbell I.** Dead in bed syndrome: a new manifestation of nocturnal hypoglycaemia? *Diabet Med.* 1991;8:3-4.
21. **Malone ML, Klos SE, Gennis VM, Goodwin JS.** Frequent hypoglycemic episodes in the treatment of patients with diabetic ketoacidosis. *Arch Intern Med.* 1992;152:2472-2477.
22. **Tzamaloukas AH, Murata GH, Eisenberg B, Murphy G, Avasthi PS.** Hypoglycemia in diabetics on dialysis with poor glycemic control: hemodialysis versus continuous ambulatory peritoneal dialysis. *Int J Artif Organs.* 1992;15:390-392.
23. **Pandit MK, Burke J, Gustafson AB, Minocha A, Peiris AN.** Drug-induced disorders of glucose tolerance. *Ann Intern Med.* 1993;118:529-539.
24. **Service FJ.** Hypoglycemic disorders. *N Engl J Med.* 1995;332:1144-1152.
25. **Shorr RI, Ray WA, Daugherty JR, Griffin MR.** Individual sulfonylureas and serious hypoglycemia in older people. *J Am Geriatr Soc.* 1996;44:751-755.
26. **Shorr RI, Ray WA, Daugherty JR, Griffin MR.** Incidence and risk factors for serious hypoglycemia in older persons using insulin or sulfonylureas. *Arch Intern Med.* 1997;157:1681-1686.
27. **Weston PJ, Gill GV.** Dead-in-bed syndrome in diabetes mellitus [letter]. *Lancet.* 1997;350:1032-1033.

28. **Hart SP, Frier BM.** Causes, management and morbidity of acute hypoglycaemia in adults requiring hospital admission. *QJM.* 1998;91:505-510.
29. **Littlefield D.** Ideas for treating low blood sugar for diabetics on dialysis. *J Ren Nutr.* 1998;8:107.
30. **Shilo S, Berezovsky S, Friedlander Y, Sonnenblick M.** Hypoglycemia in hospitalized nondiabetic older patients. *J Am Geriatr Soc.* 1998;46:978-982.
31. **Ben-Ami H, Nagachandran P, Mendelson A, Edoute Y.** Drug-induced hypoglycemic coma in 102 diabetic patients. *Arch Intern Med.* 1999;159:281-284.
32. **Virally ML, Guillausseau PJ.** Hypoglycemia in adults. *Diabetes Metab.* 1999;25:477-490.
33. **Haviv YS, Sharkia M, Safadi R.** Hypoglycemia in patients with renal failure. *Ren Fail.* 2000;22:219-223.
34. **Harrower AD.** Comparative tolerability of sulphonylureas in diabetes mellitus. *Drug Saf.* 2000;22:313-320.
35. **Jackson MA, Holland MR, Nicholas J, Lodwick R, Forster D, Macdonald IA.** Hemodialysis-induced hypoglycemia in diabetic patients. *Clin Nephrol.* 2000;54:30-34.
36. **Krepinsky J, Ingram AJ, Clase CM.** Prolonged sulphonylurea-induced hypoglycemia in diabetic patients with end-stage renal disease. *Am J Kidney Dis.* 2000;35:500-505.
37. **Mak RH.** Impact of end-stage renal disease and dialysis on glycemic control. *Semin Dial.* 2000;13:4-8.
38. **Akmal M.** Hemodialysis in diabetic patients. *Am J Kidney Dis.* 2001;38(Suppl 1):S195-S199.
39. **Mehmet S, Quan G, Thomas S, Goldsmith D.** Important causes of hypoglycaemia in patients with diabetes on peritoneal dialysis. *Diabet Med.* 2001;18:679-682.
40. **Miller CD, Phillips LS, Ziemer DC, Gallina DL, Cook CB, El-Kebbi IM.** Hypoglycemia in patients with type 2 diabetes mellitus. *Arch Intern Med.* 2001;161:1653-1659.
41. **Hasslacher C, Wittmann W.** Severe hypoglycemia in diabetics with impaired renal function [article in German]. *Dtsch Med Wochenschr.* 2003;128:253-256.
42. **Heaton A, Martin S, Brelje T.** The economic effect of hypoglycemia in a health plan. *Manag Care Interface.* 2003;16:23-27.
43. **Kagansky N, Levy S, Rimon E, et al.** Hypoglycemia as a predictor of mortality in hospitalized elderly patients. *Arch Intern Med.* 2003;163:1825-1829.
44. **Ponte CD.** Monitoring the patient with diabetes mellitus—how to avoid medication errors. *Hosp Pharm.* 1989;24:280-283, 289.
45. **Classen DC, Pestotnik SL, Evans RS, Burke JP.** Computerized surveillance of adverse drug events in hospital patients [erratum in *JAMA.* 1992;267:1922]. *JAMA.* 1991;266:2847-2851.
46. **Cullen DJ, Bates DW, Small SD, Cooper JB, Nemeskal AR, Leape LL.** The incident reporting system does not detect adverse drug events: a problem for quality improvement. *Jt Comm J Qual Improv.* 1995;21:541-548.
47. **Roman SH, Linekin PL, Stagnaro-Green A.** An inpatient diabetes QI program. *Jt Comm J Qual Improv.* 1995;21:693-699.
48. **Stagnaro-Green A, Barton MK, Linekin PL, Corkery E, deBeer K, Roman SH.** Mortality in hospitalized patients with hypoglycemia and severe hyperglycemia. *Mt Sinai J Med.* 1995;62:422-426.
49. **Lesar TS, Lomaestro BM, Pohl H.** Medication-prescribing errors in a teaching hospital: a 9-year experience. *Arch Intern Med.* 1997;157:1569-1576.
50. **Cohen MR, Proulx SM, Crawford SY.** Survey of hospital systems and common serious medication errors. *J Healthc Risk Manag.* 1998;18:16-27.
51. **Jha AK, Kuperman GJ, Teich JM, et al.** Identifying adverse drug events: development of a computer-based monitor and comparison with chart review and stimulated voluntary report. *J Am Med Inform Assoc.* 1998;5:305-314.
52. **Cohen MR.** *Medication Errors.* Washington, DC: Institute for Safe Medication Practices, American Pharmaceutical Association, 1999.
53. **Thamer M, Fay NF, Taylor T.** Association between anti-hypertensive drug use and hypoglycemia: a case-control study of diabetic users of insulin or sulfonylureas. *Clin Ther.* 1999;21:1387-1400.
54. **Bates DW.** Using information technology to reduce rates of medication errors in hospitals. *BMJ.* 2000;320:788-791.
55. **Bates DW.** Unexpected hypoglycemia in a critically ill patient. *Ann Intern Med.* 2002;137:110-116.
56. **Winterstein AG, Hattton RC, Gonzalez-Rothi R, Johns TE, Segal R.** Identifying clinically significant preventable adverse drug events through a hospital's database of adverse drug reaction reports. *Am J Health Syst Pharm.* 2002;59:1742-1749.
57. **Bates DW, Evans RS, Murff H, Stetson PD, Pizziferri L, Hripcsak G.** Detecting adverse events using information technology. *J Am Med Inform Assoc.* 2003;10:115-128.
58. **Deepak PJ, Sunitha K, Nagaraj J, Sanjukta A, Bhattacharyya A.** Inpatient management of diabetes: survey in a tertiary care centre. *Postgrad Med J.* 2003;79:585-587.
59. **DCCT Research Group.** Epidemiology of severe hypoglycemia in the Diabetes Control and Complications Trial. *Am J Med.* 1991;90:450-459.
60. **Diabetes Control and Complications Trial Research Group.** The effect of intensive treatment of diabetes on the development and progression of long-term complications in insulin-dependent diabetes mellitus. *N Engl J Med.* 1993;329:977-986.
61. **Mak KH, Mah PK, Tey BH, Sin FL, Chia G.** Fasting blood sugar level: a determinant for in-hospital outcome in patients with first myocardial infarction and without glucose intolerance. *Ann Acad Med Singapore.* 1993;22:291-295.
62. **U.K. Prospective Diabetes Study Group.** U.K. Prospective Diabetes Study 16: overview of 6 years' therapy of type II diabetes; a progressive disease [erratum in *Diabetes.* 1996;45:1655]. *Diabetes.* 1995;44:1249-1258.
63. **Malmberg K, Rydén L, Efendic S, et al.** 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.* 1995;26:57-65.
64. **Malmberg K (Diabetes Mellitus, Insulin Glucose Infusion in Acute Myocardial Infarction [DIGAMI] Study Group).** Prospective randomised study of intensive insulin treatment on long term survival after acute myocardial infarction in patients with diabetes mellitus. *BMJ.* 1997;314:1512-1515.
65. **Zerr KJ, Furnary AP, Grunkemeier GL, Bookin S, Kanhere V, Starr A.** Glucose control lowers the risk of wound infection in diabetics after open heart operations. *Ann Thorac Surg.* 1997;63:356-361.
66. **UK Prospective Diabetes Study (UKPDS) Group.** Effect of intensive blood-glucose control with metformin on complications in overweight patients with type 2 diabetes (UKPDS 34) [erratum in *Lancet.* 1998;352:1557]. *Lancet.* 1998;352:854-865.

67. **Pomposelli JJ, Baxter JK III, Babineau TJ, et al.** Early postoperative glucose control predicts nosocomial infection rate in diabetic patients. *JPEN J Parenter Enteral Nutr.* 1998;22:77-81.
68. **Furnary AP, Zerr KJ, Grunkemeier GL, Starr A.** Continuous intravenous insulin infusion reduces the incidence of deep sternal wound infection in diabetic patients after cardiac surgical procedures [with discussion]. *Ann Thorac Surg.* 1999;67:352-362.
69. **Golden SH, Peart-Vigilance C, Kao WH, Brancati FL.** Perioperative glycemic control and the risk of infectious complications in a cohort of adults with diabetes. *Diabetes Care.* 1999;22:1408-1414.
70. **Scott JF, Robinson GM, French JM, O'Connell JE, Alberti KG, Gray CS.** Prevalence of admission hyperglycaemia across clinical subtypes of acute stroke [letter]. *Lancet.* 1999;353:376-377.
71. **Scott JF, Robinson GM, French JM, O'Connell JE, Alberti KG, Gray CS.** Glucose potassium insulin infusions in the treatment of acute stroke patients with mild to moderate hyperglycemia: the Glucose Insulin in Stroke Trial (GIST). *Stroke.* 1999;30:793-799.
72. **Furnary AP, Chaugle H, Zerr K, Grunkemeier G.** Postoperative hyperglycemia prolongs length of stay in diabetic CABG patients. *Circulation.* 2000;102:II-556.
73. **Brown G, Dodek P.** Intravenous insulin nomogram improves blood glucose control in the critically ill. *Crit Care Med.* 2001;29:1714-1719.
74. **Latham R, Lancaster AD, Covington JF, Pirolo JS, Thomas CS.** The association of diabetes and glucose control with surgical-site infections among cardiothoracic surgery patients. *Infect Control Hosp Epidemiol.* 2001;22:607-612.
75. **Roman SH, Chassin MR.** Windows of opportunity to improve diabetes care when patients with diabetes are hospitalized for other conditions. *Diabetes Care.* 2001;24:1371-1376.
76. **Van den Berghe G, Wouters P, Weekers F, et al.** Intensive insulin therapy in critically ill patients. *N Engl J Med.* 2001;345:1359-1367.
77. **Markovitz LJ, Wiechmann RJ, Harris N, et al.** Description and evaluation of a glycemic management protocol for patients with diabetes undergoing heart surgery. *Endocr Pract.* 2002;8:10-18.
78. **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.* 2002;87:978-982.
79. **Baird TA, Parsons MW, Phan T, et al.** Persistent post-stroke hyperglycemia is independently associated with infarct expansion and worse clinical outcome. *Stroke.* 2003;34:2208-2214.
80. **Finney SJ, Zekveld C, Elia A, Evans TW.** Glucose control and mortality in critically ill patients. *JAMA.* 2003;290:2041-2047.
81. **Furnary AP, Gao G, Grunkemeier GL, et al.** Continuous insulin infusion reduces mortality in patients with diabetes undergoing coronary artery bypass grafting. *J Thorac Cardiovasc Surg.* 2003;125:1007-1021.
82. **McAlister FA, Man J, Bistritz L, Amad H, Tandon P.** Diabetes and coronary artery bypass surgery: an examination of perioperative glycemic control and outcomes. *Diabetes Care.* 2003;26:1518-1524.
83. **Van den Berghe G, Wouters PJ, Bouillon R, et al.** Outcome benefit of intensive insulin therapy in the critically ill: insulin dose versus glycemic control. *Crit Care Med.* 2003;31:359-366.
84. **Park RH, Hansell DT, Davidson LE, Henderson G, Legge V, Gray GR.** Management of diabetic patients requiring nutritional support. *Nutrition.* 1992;8:316-320.
85. **Fink S, Chaudhuri TK.** Stroke and malpractice claims. *South Med J.* 1997;90:901-902.
86. **Sheldon GF, Baker C.** Complications of nutritional support. *Crit Care Med.* 1980;8:35-37.
87. **Woolfson AM.** Control of blood glucose during nutritional support in ill patients. *Intensive Care Med.* 1980;7:11-14.
88. **Woolfson AM.** An improved method for blood glucose control during nutritional support. *JPEN J Parenter Enteral Nutr.* 1981;5:436-440.
89. **Sajbel TA, Dutro MP, Radway PR.** Use of separate insulin infusions with total parenteral nutrition. *JPEN J Parenter Enteral Nutr.* 1987;11:97-99.
90. **Knapke CM, Owens JP, Mirtallo JM.** Management of glucose abnormalities in patients receiving total parenteral nutrition. *Clin Pharm.* 1989;8:136-144.
91. **Pitts DM, Kilo KA, Pontious SL.** Nutritional support for the patient with diabetes. *Crit Care Nurs Clin North Am.* 1993;5:47-56.
92. **McMahon MM, Rizza RA.** Nutrition support in hospitalized patients with diabetes mellitus. *Mayo Clin Proc.* 1996;71:587-594.
93. **Kerr D, Hamilton P, Cavan DA.** Preventing glycaemic excursions in diabetic patients requiring percutaneous endoscopic gastrostomy (PEG) feeding after a stroke. *Diabet Med.* 2002;19:1006-1008.
94. **Putz D, Kabadi UM.** Insulin glargine in continuous enteric tube feeding [letter]. *Diabetes Care.* 2002;25:1889-1890.
95. **Choleau C, Dokladal P, Klein JC, Ward WK, Wilson GS, Reach G.** Prevention of hypoglycemia using risk assessment with a continuous glucose monitoring system. *Diabetes.* 2002;51:3263-3273.
96. **Young M.** Hypoglycaemia; a nursing care study. *Nurs Times.* 1970;66:915-916.
97. **Stock PL.** Action stat! insulin shock. *Nursing.* 1985;15:53.
98. **Arbour R.** Acute hypoglycemia. *Nursing.* 1994;24:33.
99. **Schaller J, Welsh JR.** Myths & facts about diabetic hypoglycemia. *Nursing.* 1994;24:67.
100. **Swithers C.** Avoiding hypoglycemia [letter]. *Nursing.* 1994;24:4, 6.
101. **Parker C.** Responding quickly to hypoglycemia. *Am J Nurs.* 1994;94:46.
102. **Peragallo-Dittko V.** Diabetes 2000: acute complications. *RN.* 1995;58:36-41.
103. **Reising DL.** Acute hypoglycemia: keeping the bottom from falling out. *Nursing.* 1995;25:41-48.
104. **Amiel SA.** Hypoglycaemia avoidance—technology and knowledge. *Lancet.* 1998;352:502-503.
105. **Raforth RJ.** Standardizing sliding scale insulin orders. *Am J Med Qual.* 2002;17:175-178.
106. **Hanish LR.** Standardizing regimens for sliding-scale insulin. *Am J Health Syst Pharm.* 1997;54:1046-1047.
107. **Dickerson LM, Ye X, Sack JL, Hueston WJ.** Glycemic control in medical inpatients with type 2 diabetes mellitus receiving sliding scale insulin regimens versus routine diabetes medications: a multicenter randomized controlled trial. *Ann Fam Med.* 2003;1:29-35.
108. **Gearhart JG, Duncan JL III, Replogle WH, Forbes RC, Walley EJ.** Efficacy of sliding-scale insulin therapy: a comparison with prospective regimens. *Fam Pract Res J.* 1994;14:313-322.
109. **Queale WS, Seidler AJ, Brancati FL.** Glycemic control and sliding scale insulin use in medical inpatients with diabetes mellitus. *Arch Intern Med.* 1997;157:545-552.

110. **Achtmeyer CE, Payne TH, Anawalt BD.** Computer order entry system decreased use of sliding scale insulin regimens. *Methods Inf Med.* 2002;41:277-281.
111. **Trence DL, Kelly JL, Hirsch IB.** The rationale and management of hyperglycemia for in-patients with cardiovascular disease: time for change. *J Clin Endocrinol Metab.* 2003;88:2430-2437.
112. **Gibney M, Cohen E, Roman SH.** Inpatient diabetes care: strategies for disease management. *Dis Manag.* 1999;2: 13-23.
113. **Harvey R, Jarrett PG, Peltekian KM.** Patterns of paging medical interns during night calls at two teaching hospitals. *CMAJ.* 1994;151:307-311.
114. **Caldemeyer C, Dowell AR.** Oral paging in a community hospital: is it used effectively? *Indiana Med.* 1994;87: 208-211.
115. **Chassin MR.** Is health care ready for Six Sigma quality? *Milbank Q.* 1998;76:565-591, 510.
116. **Kohn LT, Corrigan JM, Donaldson MS, eds (Committee on Quality of Health Care in America, Institute of Medicine).** *To Err Is Human: Building a Safer Health System.* Washington, DC: National Academies Press, 2000.
117. **Committee on Quality of Health Care in America, Institute of Medicine.** *Crossing the Quality Chasm: A New Health System for the 21st Century.* Washington, DC: National Academies Press, 2001.
118. **Quevedo SF, Sullivan E, Kington R, Rogers W.** Improving diabetes care in the hospital using guideline-directed orders. *Diabetes Spectrum.* 2001;14:226-233.

APPENDIX 1

Suggested Elements of a Nurse-Implemented Hypoglycemia Prevention Protocol

- Triggering events for hospital hypoglycemia are listed.
- Physicians ordering insulin are asked to identify a patient as possibly having type 1 diabetes.
- At the time insulin is ordered for patients with possible type 1 diabetes, an automatic query is generated that prompts the physician to select an alternative plan for subcutaneous basal insulin therapy, to be used in case a triggering event for hypoglycemia occurs.
- A provision exists under policy for nursing actions to be taken, if any of the listed triggering events for hypoglycemia should occur, to prevent glucose level from declining below 120 mg/dL:
 1. Withhold scheduled antihyperglycemic drug(s) until further orders are received.
 2. Substitute and administer the identified alternative basal insulin for type 1 diabetes.
 3. Continue correction doses of insulin for hyperglycemia.
 4. Obtain point-of-care blood glucose level every 2 hours for duration of action of the previously administered antihyperglycemic drug.
 5. Obtain needed assistance to restart intravenous line, if necessary.
 6. Give 15 to 30 g of carbohydrate orally or, as will usually be required, 50 mL of 50% dextrose in water for glucose levels <120 mg/dL.
 7. For patients to be transported off ward, deliver 5% dextrose-containing fluids or, if patient is volume-restricted, 10% dextrose-containing fluids, for the time frame of action of previously administered antihyperglycemic therapy.
 8. Notify physician and provide opportunity for physician override of policy.

APPENDIX 2

Sample Insulin Orders With Use of a Hypoglycemia Prevention Protocol

- Admit patient to General Ward 4.
- Maintain consistent dietary carbohydrate, 60 g at meals and 30 g at bedtime.
- Determine capillary blood glucose level 4 times daily.
- Insulin 70/30: Administer 20 U before breakfast and 10 U before supper daily.
- Correction-dose algorithm with insulin lispro
 1. (Query: Possible type 1 diabetes?)
- Answer: yes
 2. (Query and prompts for response: Alternative basal insulin order?)
 Enter total daily insulin dose during normal dietary intake, if known:
 (a) 30 U

Pharmacy computer proposes alternative basal orders (physician chooses among 3 regimens, each delivering 40% of total daily insulin dose over 24 hours, or free text entry; the number of units is soft-coded and is calculated for each patient):

- Regular insulin 3 U subcutaneously every 6 hours *or*
- Insulin NPH 6 U every 12 hours *or*
- Insulin glargine 12 U daily *or*
- (Free text) _____

If total daily insulin dose during normal dietary intake is unknown, enter weight in kilograms:

(b) 60 kg

Pharmacy computer proposes alternative basal orders (physician chooses among 3 regimens, each delivering 0.2 U/kg over 24 hours, or free text entry):

- Regular insulin 3 U subcutaneously every 6 hours *or*
- Insulin NPH 6 U every 12 hours *or*
- Insulin glargine 12 U daily *or*
- (Free text) _____

The patient's medication administration record is tagged with the alternative basal insulin orders until cancellation or ward transfer occurs. Instructions for nurse preventive management of hypoglycemia and use of alternative basal orders print/appear on medication administration record for nurse to carry out should such situations arise.