Eliminating Inpatient Sliding-Scale Insulin

A reeducation project with medical house staff

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OBJECTIVE — We studied a systematic program to reeducate our medical house officers on how to manage inpatient hyperglycemia without the use of sliding-scale insulin (SSI).

RESEARCH DESIGN AND METHODS — Patients admitted to the general medical service with diabetes or a blood glucose >140 mg/dl were included. HbA_{1c} was measured in all patients, and therapy was modified if the HbA_{1c} was >7.0%. For each 24 h on call, two house officers were responsible for all glucose management for their team's patients and rounded with a teaching endocrinologist twice daily for 2 weeks. Oral agent or insulin therapy was modified using blood glucoses and HbA_{1c}. All patients who required insulin therapy were treated with basal and bolus insulin, usually NPH and regular, adjusted twice daily.

RESULTS — During 8 weeks, 88 patients were identified and 16 house officers were instructed. The mean duration of diabetes was 10.4 years. Mean HbA_{1c} level was 8.7%, and 48% of patients had HbA_{1c} >8%. All patients with HbA_{1c} >7% had diabetes therapy intensified. Overall 80% had their diabetes therapy changed by discharge. Compared with 98 historical control subjects, significantly fewer study patients had episodes of hyperglycemia, and a subgroup followed for 12 months showed a decrease in HbA_{1c} from 10.1 to 8%.

CONCLUSIONS — Medical history, blood glucose, and HbA_{1c} testing can effectively identify patients with inpatient hyperglycemia. Using direct ward-based teaching and a widely disseminated pocket set of guidelines, house officers can be taught to effectively and safely manage inpatient hyperglycemia without the use of SSI.

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Diabetes is one of the most common diagnoses encountered in hospitalized patients (1). In 1997, diabetes was the fourth most common comorbid condition in hospital discharges, and 9.5% of all hospital discharges listed diabetes as a diagnosis upon discharge (2). The majority of hospitalizations for patients with diabetes are due to comorbid conditions, and diabetes management is not a focus during inpatient stays (3,4). However, inpatient hyperglycemia has been associated with nosocomial infections, increased mortality, and increased length of stay (5).

As in most American hospitals, the use of sliding-scale insulin (SSI) for the treatment of inpatient hyperglycemia was common practice at Rush University Medical Center before our study. Typically an order is written for diabetic patients upon admission that provides a predetermined amount of subcutaneous regular insulin, usually beginning with 2 units for blood glucose >150 or 200 mg/dl. The order calls for the insulin dose to be increased by 2 units for every 50-mg/dl increase in blood glucose. Previous outpatient diabetes therapy is commonly discontinued on admission, especially if patients are NPO (nothing per os).

While it has long been recognized that SSI has many drawbacks, its use in the inpatient setting has been a reflex action passed down from attending physicians to residents to medical students for the past several generations despite a lack of evidence to support the practice. In December of 2003, the American Association of Clinical Endocrinologists and the American Diabetes Association sponsored a consensus conference on inpatient diabetes control (2). The conference position statement articulates that intensified glycemic control can improve short- and long-term outcomes for hyperglycemic inpatients. The new guidelines for inpatient therapy recommend targeting fasting blood glucose to 110 mg/dl and postprandial glucose to <180 mg/dl for non-ICU inpatients. The target range for glucose for ICU patients was 80–110 mg/dl. A technical review by Clement et al. (6) was published to coincide with the consensus conference and contains detailed suggestions on how to achieve the glucose targets identified by the consensus guidelines.

Our study attempted to achieve similar goals in hyperglycemic inpatients by using a standardized approach. First, we sought to identify all hyperglycemic inpatients and treat them with a uniform protocol regardless of whether they had a previous history of diabetes. Previous studies have shown that hyperglycemic inpatients without a history of diabetes receive less attention to glucose control and have higher mortality than previously diagnosed diabetic inpatients (7,8). Second, we sought to effectively and safely

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Abbreviations: SSI, sliding-scale insulin.
A table elsewhere in this issue shows conventional and Système International (SI) units and conversion factors for many substances.

Table 1—*Patient demographics and inpatient insulin regimens*

	Study patients (n = 88)	Control patients (n = 98)
Mean age years (range)	57 (19–86)	59 (24–87)
Female sex (%)	63	58
Type 2 diabetic (%)	95	93
Mean duration of diabetes (years)	10.4	14.6
Newly diagnosed diabetes (%)	10	9
Inpatient therapy		
No diabetic therapy (%)	2	16
Oral agent (%)	30	37
NPH and regular insulin (%)	68	32
Combination NPH/Regular and oral agent (%)	0	15
Sliding-scale regular (%)	0	100

implement a basal-bolus approach to insulin management in the inpatient setting. This involved teaching medical residents to feel comfortable managing blood glucose without the use of SSI. Importantly, we did not want to significantly increase the incidence of hypoglycemia with a protocol that decreased the incidence of hyperglycemia. Third, we sought to determine whether our approach of evaluating patients and intervening to modify glycemic control twice daily was better than SSI in the prevention of hyperglycemia. Fourth, we wanted to determine whether routinely checking HbA_{1c} levels on all hyperglycemic inpatients could lead to short- and long-term improvements in diabetes control.

RESEARCH DESIGN AND METHODS

— The study enrolled all patients with a history of diabetes or a blood glucose level >140 mg/dl who were admitted to two general medical house staff teams during an 8-week period of time. Patients whose length of stay was <2 days were excluded. Eight post–graduate year 1 house officers were targeted during each month of the study.

For each 24-h tour of duty, two house officers were responsible for the glucose management of all patients on their team. All patients in the study group had blood glucose monitored premeals and at bedtime. All patients had HbA_{1c} checked on admission. Preexisting diabetes therapy was continued for the first 12 h as long as the initial blood glucose was <200 mg/dl. Metformin was discontinued if serum creatinine was >1.5 mg/dl.

Thiazolidinediones were discontinued if new or worsened heart failure was present. Sulfonylureas were discontinued if the patient was unable to eat. If blood glucose was >200 mg/dl, oral agents were discontinued and basal/bolus insulin was given twice daily. If HbA_{1c} was >7%, therapy was intensified by one of the following methods: increasing the dose and number of oral medications, changing from oral medications to insulin, or increasing the doses of insulin.

Insulin-treated patients usually received NPH and regular twice daily. Insulin glargine and insulin lispro/aspart were used only if they were preexisting therapy. Combination insulin/oral agent therapy was not used. Premixed insulin such as 70/30 was not used. Standing orders were permitted for oral agents but not for insulin. SSI was not permitted. Doses were modified twice daily based on four daily blood glucose values. Our targets were as follows: fasting blood glucose 80–120 mg/dl and prelunch, predinner, and bedtime blood glucose 100–150 mg/dl.

A teaching endocrinologist rounded twice daily at 7:00 A.M. and 6:00 P.M. with the house officers. The nurse and medical student caring for each patient also participated in the exercise. (See inpatient guidelines, online appendix 1 [available from http://care.diabetesjournals.org].)

A group of historical control patients was retrospectively identified. These patients had been admitted to the same two teams, had the same attendings, and were on the same three-patient units as the study patients. The mean blood glucose of the study group, as well as the percentage of all blood glucose levels, which were in target ranges, were compared with those of the control patients. The frequency of hypoglycemic episodes (blood glucose level <60 mg/dl) in both groups was compared, as well as the necessity for intravenous 50% dextrose therapy. Since changing outpatient diabetes therapy guided by the inpatient HbA_{1c} was an important cornerstone of our approach, we compared how often HbA_{1c} was a part of inpatient data in the historical control patients and how often outpatient therapy was changed on hospital discharge. We had follow-up HbA_{1c} data on 34 study patients after 1 year.

Statistical analysis was performed using SPSS v. 11.5 (SPSS, Chicago, IL). Descriptive statistics were performed on all data. Comparisons between groups were tested using one-way ANOVA, Mann Whitney U tests, and χ^2 tests as appropriate. Results were considered statistically significant when $P < 0.05$.

RESULTS

— A total of 88 patients were analyzed in the study group and 98 in the control group. The groups were well matched (Table 1).

The hyperglycemic therapy for all patients is also shown in Table 1. Altogether, 100% of the control patients and none of the study patients had SSI orders written; 68% of the study group was on a twice-daily dosing of the NPH/regular as compared with 32% of the control patients; and 30% of the study patients were on an oral agent, while none were on combination NPH/regular insulin and oral agents together. This is in comparison with 37% of control patients on an oral agent and 15% of control patients on NPH/regular insulin/oral agent combinations.

The mean blood glucose for study patients was (mean ± SD) 150 ± 37 mg/dl versus 200 ± 51 mg/dl for control patients ($P < 0.01$) (Table 2). Only 6.5% of blood glucose levels in the study group were >250 mg/dl compared with 20.5% of blood glucose levels in control patients ($P < 0.01$). Also, 3.6% of blood glucose levels in study patients were <60 mg/dl compared with 1.4% of control patients ($P = 0.01$). Moreover, 26% of hypoglycemic episodes in study patients and 30% of hypoglycemic episodes in control patients required intravenous dextrose, usually because they were NPO. None of the episodes of hypoglycemia in either group were clinically severe or associated with adverse outcomes.

Two target windows for blood glucose were analyzed (Table 2). Of the study patients, 43% had blood glucose levels be-

Table 2—*Measurements of glycemic control*

	Study patients	Control patients	Significance
Mean glucose ± SD (mg/dl)	150 ± 37	200 ± 51	$P < 0.01$
Glucose <60 mG/dl (%)	3.60	1.40	$P = 0.01$
Low glucose levels requiring i.v. D-50 (%)	26	30	NS
Glucose >250 mg/dl (%)	6.50	20.50	$P < 0.01$
Glucose >250 mg/dl (%)	6.50	20.50	$P < 0.01$
Glucose 80–140 mg/dl (%)	43.80	22	$P < 0.01$
Glucose 80–180 mg/dl (%)	65.10	43.10	$P < 0.01$

tween 80–140 mg/dl compared with 22% of control patients ($P < 0.01$). When blood glucose parameters were expanded to 80–180 mg/dl, 65% of the study group was within these parameters compared with 43% of the control patients ($P < 0.01$).

HbA_{1c} values were obtained in 99% of the intervention patients but in only 32% of the control patients (Table 3). Mean HbA_{1c} was 8.7% in the study patients and 10.2% in the control patients. Diabetic therapy was changed on hospital discharge in 80% of the study patients versus 32% of control patients. After discharge from the hospital, 40 study patients had local follow-up and 34 of these patients had periodic HbA_{1c} levels obtained. The initial mean HbA_{1c} was of 10.1% for these 34 patients, and after 12 months follow-up, their mean HbA_{1c} decreased to 8% ($P < 0.01$) (Fig. 1).

At the end of each 4-week rotation, all house officers completed a survey, and all felt competent to manage inpatient hyperglycemia without SSI according to the new guidelines.

CONCLUSIONS — The management of inpatient hyperglycemia has been a controversial and problematic challenge for many years. The sliding-scale culture of tolerated hyperglycemia is deeply rooted in most residency training programs, ours included. To approach this problem, we studied the use of commonly used outpatient algorithms for the management of inpatient hyperglycemia.

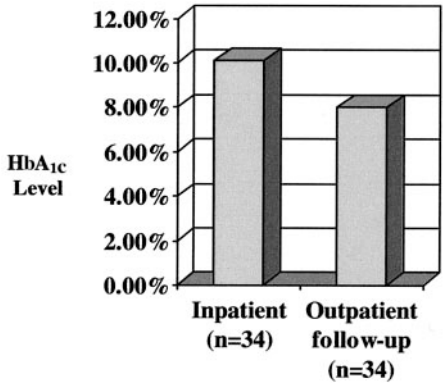

Figure 1—*HbA_{1c} outcomes: the effect of inpatient interventions after 1 year.*

There have been few published studies addressing SSI management in inpatients. A prospective cohort study by Queale et al. (9) looked at the effectiveness of SSI among 171 adult diabetic patients admitted to an inpatient medical service. Of these, 37% received NPH insulin, 25% received oral agents, and 76% were placed on SSI. They found that suboptimal glycemic control was common; patients treated solely with SSI were three times more likely to have blood glucoses >300 mg/dl. In 80% of patients, the diabetes control orders prescribed on admission were not changed during the inpatient stay, despite poor glucose control. A retrospective study compared glucose control in a cohort of inpatients after recovery from diabetic ketoacidosis (10). These authors found that the median blood glucose was 262 mg/dl and that the length of stay was 6.3 days in SSI-treated patients compared with 200 mg/dl and 4.4 days in patients treated with intermediate and short-acting insulin. SSI treats hyperglycemia once it has occurred but does nothing to prevent it from occurring or recurring, as often no basal insulin is provided. SSI attempts to treat basal hyperglycemia with short-acting bolus insulin. No bolus insulin is provided for meals. It is no wonder that such a backwards approach usually fails to control hyperglycemia. Indeed its use has been described as "Action without Benefit" (11).

Our study goals aimed at answering the following questions. *1*) Could medical house staff be taught an intensive basal-bolus approach to managing inpatient hyperglycemia? *2*) Could medical house staff improve the quality of glycemic control for inpatients while maintaining a high degree of patient safety? *3*) Does the routine measurement and utilization of HbA_{1c} on all inpatients with a history of diabetes or with hyperglycemia contribute to improvements in immediate inpatient management and/or to long-term improvement in glycemic control?

Our results indicate that we met our first goal. The two key ingredients to this success were individualized teaching of 1st-year medical house staff by an experienced diabetologist and a widely disseminated pocket set of guidelines that set forth a standardized approach for all inpatients. We emphasized the view that inpatient hospitalizations are often "windows of opportunity" for improvement of diabetes care, as espoused by Roman and Chassin (3). They compared the frequency of hyperglycemia among inpatients after initiation of quality improvement interventions. The frequency of severe and prolonged hyperglycemic episodes and nosocomial infections decreased significantly. We believe that we achieved a similar result in terms of glycemic control. Although we have not yet fully implemented an inpatient diabetes team with an educator and dietitian, other authors have clearly shown the additional benefits of adding inpatient education and coordination in reducing length of stay and the rate of readmission for patients with diabetes (3,12,13).

The skills and experience gained by our house staff in the management of inpatient hyperglycemia should be equally valuable in the management of outpatients, since we emphasized a common approach for all patients. Hopefully this approach will significantly improve the diabetes management skills of our trainees.

Since completing this 2-month study, we continued to educate each of our 1st-year medical house staff with the same approach and program. Over the subsequent 5 months, we educated the remaining 44 1st-year house staff in our program. Beginning in July 2003, SSI was no longer allowed on our medical inpatients. We have continued to educate the incoming 1st-year house staff in groups of four during their 1st month of general medical floor duty. This effort, which admittedly is intensive, requires 4 months of time in our program with ~60 1st-year trainees.

Table 3—*HbA_{1c}-guided change in therapy*

	Study patients	Control patients	Significance
HbA_{1c} level obtained (%)	99	32	$P < 0.01$
Mean HbA_{1c} (%)	8.7	10.2	NS
Diabetic therapy changed (%)	80	32	$P < 0.01$

We also met the second goal of the study: improvement in the glycemic control of inpatients without a clinically relevant increase in hypoglycemia. The number of blood glucose levels between 80–140 mg/dl doubled to 43%, and the number >250 mg/dl was reduced by ~75%. Although the use of a basal-bolus approach to insulin therapy in our study was associated with a slight increase in the frequency of hypoglycemia, the percentage of hypoglycemic events requiring i.v. dextrose was not different, and none were associated with adverse outcomes. Inpatients may have hypoglycemia due to many factors such as a decreased appetite, disruption of meals for medical tests, infection, renal failure, and antidiabetes therapy. Hypoglycemia in elderly inpatients has not been correlated with mortality (14).

Levatan et al. (7) found in their study of 130 hyperglycemic inpatients that 35.7% were not recognized to have a diagnosis of diabetes. Several authors have studied the use of HbA_{1c} for case finding of outpatients with diabetes (15). Greci et al. (16) studied hyperglycemic inpatients and found that HbA_{1c} >6% reliably diagnoses diabetes. Our approach emphasized the role of HbA_{1c}. We were able to evaluate patients with newly identified hyperglycemia during their inpatient stay and begin an educational and therapeutic program for those with an elevated HbA_{1c}. Since home blood glucose data are not available for most inpatient diabetic patients, we used HbA_{1c} as well as inpatient blood glucose levels to guide changes in therapy for hyperglycemia. With this approach, we increased the number of patients who were discharged on an improved therapeutic regimen from 32 to 80%. HbA_{1c} was significantly improved in a subset of patients for whom we had follow-up data after 1 year.

The main weakness of our study is the retrospective use of historical control patients. However, our dominant motivation for the program was to move our house staff out of the sliding-scale era, and this quality/safety goal took precedence over a controlled prospective study.

In summary, our medical house staff has adapted well to the use of a basal-bolus approach to inpatient therapy of hyperglycemia guided by blood glucose and HbA_{1c} levels. The successful teaching of this approach allowed for complete cessation of SSI. Interestingly, the use of SSI by our surgical house staff has dropped by 60% over the past several years, perhaps reflecting cross-pollination of these concepts by our medical students. Our approach mainly used NPH and regular insulin. At the time that our study was conducted, we had little experience with the use of the newer insulin analogues. Currently we are conducting studies comparing glargine to NPH insulin and lispro/aspart to regular insulin in inpatients. These newer analogues may prove to be valuable additions given the problems with insulin and meal timing in the inpatient setting. It may be difficult for our findings to be widely generalized. A significant staff commitment is required. Since a new group of interns arrive each July, the entire effort needs to be repeated annually. In the current climate of reduced work hours for interns, the success described in this study is not easy for our house staff to maintain. It is especially difficult for each of our on-call interns to evaluate and order evening insulin on a timely basis for four intern's load of patients while they are also receiving their new admissions. Some regression has been inevitable, and the addition of additional resources may be necessary for optimal success of such a program.

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