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Clinical study

Inpatient management of **diabetes mellitus**

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*There is now widespread appreciation of the importance of maintaining glucose levels as close to the normal range as possible among outpatients with **diabetes**. However, the importance of tight glucose control in inpatients is less well established. During the past several years, it has become apparent that hyperglycemia in hospitalized patients, especially those in the postoperative setting, is associated with poorer outcomes. In addition, two randomized trials have shown improved outcomes with intensive glucose management in acutely ill patients. Based on these studies and our own experience, we propose guidelines and a framework for improving the glycemic control of hospitalized patients.*

Keywords

Diabetes Mellitus

Hospital

Inpatient

Insulin

Perioperative

Adults with **diabetes** are six times more likely to be hospitalized than are those without **diabetes** ^[1], and nearly two thirds of the \$44 billion in direct medical costs related to **diabetes** is for inpatient care ^[2]. Hospitalized diabetic patients often have poor glycemic control ^[3], increasing their susceptibility to complications and lengthening their hospital stays. Although health care professionals in outpatient settings accept the importance of tight glucose control, its importance in hospitalized patients is not widely appreciated. In this review, we discuss the reasons for suboptimal **diabetes** management in the hospital, review the available data that support a more aggressive approach, and propose a framework by which glycemic control in diabetic inpatients can be attained.

Hospital barriers to glucose control

Poor glycemic control is common among inpatients with **diabetes**, particularly in those treated with insulin ^[4], for many reasons. The majority of diabetic patients are hospitalized for reasons other than **diabetes**, such as vascular complications. Thus, care of **diabetes** per se becomes subordinate to care for the primary diagnosis requiring admission. Infection, fevers, glucocorticoid therapy, surgical trauma, and general medical stress exacerbate hyperglycemia due to the release of counter-regulatory factors that impair insulin action ^[5]. Hyperglycemia may also result from decreased physical activity, particularly if the patient had exercised regularly. Conversely, regimentation of diet and supervised compliance with drug therapy may result in hypoglycemia in patients who had poor adherence to their management programs as outpatients. Complicating matters further, a bedridden patient with altered mental status is less able to seek assistance when symptoms of hypoglycemia develop.

Self-management techniques are emphasized for patients with **diabetes**. When hospitalized, however, diabetic patients typically lose personal control of their disease. The transfer of therapeutic responsibility to the health care team often introduces a less coordinated approach, particularly when the team is less familiar with aggressive multidose insulin regimens or use of an insulin pump. In contrast with individualized **diabetes** treatment programs for outpatients, most hospital activities remain fixed and scheduled ^[6]. In addition, there are frequent interruptions in meal delivery and medication administration because of examinations and procedures that may affect a previously well-coordinated regimen.

Common errors in glucose management

Admission orders

The outpatient treatment regimen for **diabetes** is often continued unchanged or withdrawn entirely upon admission. Although either of these choices may occasionally be indicated, patients more commonly will require some modification of their outpatient regimen to adapt to the effects of acute illness.

Overly high glycemic targets

Because of a general underappreciation of the importance of glycemic control in the hospital setting and concerns about hypoglycemia, blood glucose concentrations are commonly allowed to be >200 mg/dL without aggressive intervention.

Lack of therapeutic adjustment

The antihyperglycemic regimen is often left unchanged during the entire hospitalization, rather than being reassessed based on the results of monitoring blood glucose levels [7]. A common sequela is the realization by caregivers on the day of discharge that a patient has been treated with regular insulin alone during the entire hospital stay. Thus, there is little opportunity to observe a patient's response to a more conventional regimen that can be transferred to home.

Overutilization of "sliding scales"

A regular insulin "sliding scale" is used commonly to control blood glucose levels. Many, however, have suggested that sliding scales are illogical, because they are designed to correct the therapeutic inadequacies of the previous 6-hour period rather than anticipating future requirements [8] [9]. In addition, a sliding scale is used frequently as the sole means of insulin coverage, instead of in conjunction with longer-acting insulins. Such therapy does not provide any additional benefit compared with a fixed, standing insulin regimen [9]. Indeed, one study found that use of sliding scales was associated with greater difficulty in establishing glycemic control than was a fixed-dose regimen [4]. When used alone, without long-/intermediate-acting insulin preparations, sliding scales of short-/rapid-acting insulins (Table 1) may lead to peaks and valleys of systemic insulin supply. Erratic glucose control is a common sequela [8]. Depending on the glycemic threshold for scale initiation, some patients may not receive any insulin for prolonged periods, allowing the blood glucose level to climb back into the hyperglycemic range [10].

Insulin Type	Onset*	Peak*	Duration*
Rapid-acting			
Insulin lispro [†]	10–15 min	1–2 h	3–5 h
Insulin aspart [†]	10–15 min	1–2 h	3–5 h
Short-acting			
Regular insulin	0.5–1 h	2–4 h	4–8 h
Intermediate-acting			
NPH	1–3 h	4–10 h	10–18 h
Lente	2–4 h	4–12 h	12–20 h
Long-acting			
Ultralente	6–8 h	Variable	16–24 h
Insulin glargine [†]	2–3 h	None	24 h
Premixed insulin			

70/30 (70% NPH + 30% regular)	0.5–1 h	2–10 h	10–18 h
50/50 (50% NPH + 50% regular)	0.5–1 h	2–10 h	10–18 h
75/25 (75% NPL [‡] + 25% insulin lispro)	10–15 min	1–3 h	10–16 h

* Pharmacokinetics of insulin are influenced by dose, injection site, and certain factors that are not well defined in specific patients. Therefore, patients may experience variable onsets, peaks, and durations of action in their insulin from day to day.

† Insulin analog.

‡ The pharmacokinetics of NPL are very similar to that of NPH. NPL insulin is available only in this mixture.

Adapted from Inzucchi SE. *Int Anesthesiol Clin.* 2002;40:80. With permission from Lippincott Williams & Wilkins. © 2002. NPH = neutral protamine Hagedorn; NPL = neutral protamine lispro.

Sliding scales are sometimes useful. Many diabetologists recommend physiologic adjustment of preprandial insulin based, in part, on the premeal capillary glucose level and the anticipated carbohydrate consumption in their outpatients [\[11\]](#). With the introduction of basal insulin analogs, such as insulin glargine, sliding scales may become more common. Sliding scales may also be useful when evaluating a patient's initial response to insulin [\[8\]](#). They can also be justified in patients receiving parenteral nutrition, in whom each 6-hour period is similar to the last. Otherwise, using sliding scales as the sole form of insulin coverage is usually inappropriate and strongly discouraged. Adding long-/intermediate-acting insulin will improve control substantially. If used, sliding scales should also be adjusted continuously, based on glucose monitoring results.

Underutilization of insulin infusions

Intravenous insulin is underutilized in hospital settings, although it is an excellent method to attain glycemic control quickly. Intravenous insulin is recommended widely for patients with hyperglycemic emergencies [\[12\]](#) and can also be used in the perioperative setting or when glucose control has deteriorated with conventional subcutaneous insulin injections. When compared with subcutaneous administration, the intravenous route provides predictable insulin delivery and enables rapid control of glucose levels [\[13\]](#). The underutilization of insulin infusions results from institutional obstacles, such their restriction to intensive care units (ICU). Although adequate nurse training and supervision is required for their safe implementation, insulin infusions should be able to be administered properly in any well-staffed general medical or surgical ward.

How important is inpatient glycemic control?

Several studies have demonstrated the benefits of glucose control during hospitalizations, particularly in the postoperative setting [\[14\]](#) [\[15\]](#) [\[16\]](#) [\[17\]](#). There is a clear association between poorly controlled **diabetes** and increased susceptibility to infection. Several aspects of immune function are altered in **diabetes**, including leukocyte function and immunoglobulin complement fixation, both of which are impaired in ambient glucose concentrations between 200 and 250 mg/dL [\[18\]](#) [\[19\]](#) [\[20\]](#) [\[21\]](#) [\[22\]](#) [\[23\]](#). There is also evidence that improving glycemic control leads to improved immune status [\[24\]](#) [\[25\]](#) [\[26\]](#).

Perioperative patients are at high risk of infections due to the transient compromise of host defenses; this may be exacerbated in diabetic patients. One recent study found that hyperglycemia was an independent predictor of the short-term risk of infection when the mean plasma glucose concentration exceeded 200 mg/dL at 36 hours after surgery [\[16\]](#). Another investigation of diabetic

patients undergoing cardiac surgery concluded that glucose concentrations >200 mg/dL on the first or second postoperative day increased the incidence of deep sternal wound infections ^[27]. In a nonrandomized study, use of an aggressive insulin infusion to lower postoperative glucose levels to 150 to 200 mg/dL reduced the risk of infection by 60% ^[17]. Other similar investigations suggest that hyperglycemia after surgery contributes to infectious complications and that this may be mitigated with improved control ^{[28] [29]}.

Another view is that the high incidence of infections in diabetic patients is not principally due to hyperglycemia, but to vascular disease, with impaired ability to deliver nutrients and oxygen to damaged tissues ^[30]. However, short-term hyperglycemia impairs endothelium-dependent vasodilation acutely ^[31]. Thus, although it is likely that both hyperglycemia and vascular disease contribute to the risk of infection, some of the vascular insufficiency may be reversible with improved glycemic control.

Postoperative diabetic patients also have impaired wound healing. Blood flow to wound areas and growth factor production in diabetic men are decreased, delaying the restoration of a normal epithelial layer ^{[32] [33]}. In addition, skin fibroblasts from diabetic subjects proliferate less well in a hyperglycemic milieu, further impairing wound closure ^[34].

Hyperglycemia has several other deleterious effects, including alterations in intravascular volume, which may precipitate electrolyte shifts and decrease glomerular filtration; promotion of coagulation, accentuating the risk of thrombotic complications; and the potential cardiotoxicity of elevated free fatty acid concentrations that frequently coexist in poorly controlled diabetic patients ^[14].

Intervention trials

Whether hyperglycemia is a cause or effect of complications in diabetic patients has been controversial for many years. A partial answer to this question was provided by the **Diabetes** Insulin-Glucose Infusion in Acute Myocardial Infarction (DIGAMI) study, albeit in a selected group of patients ^{[14] [15]}. The DIGAMI investigators randomly assigned 620 diabetic patients with myocardial infarction to standard therapy or to standard therapy plus an insulin-glucose infusion for at least 24 hours. The goal of the infusion was to maintain a blood glucose level between 126 and 196 mg/dL. Subsequently, infusion patients were transferred to multidose insulin therapy for at least 3 months, aiming to maintain stable normoglycemia. Twenty-four hours after randomization, mean blood glucose concentrations were 211 mg/dL in the control group and 173 mg/dL in the infusion group. At hospital discharge, the values were 162 mg/dL in the control group and 148 mg/dL in the infusion group.

During 3.4 years of follow-up, there were 138 deaths in the control group compared with 102 in the infusion group, a 28% reduction ($P = 0.01$). Most of this decrease occurred after hospital discharge, so patients may have benefited more from the meticulous outpatient treatment than from the aggressive inpatient program. However, in a subgroup of insulin-naïve patients at low cardiovascular risk, the reduction in mortality was already significant during the hospital phase (12% vs. 5%; relative reduction, 58%; $P < 0.05$).

Benefits of inpatient glucose control were also seen in a study of 1548 predominantly postoperative surgical ICU patients with or without **diabetes** ^[35]. All patients were randomly assigned to intensive insulin infusion to maintain a blood glucose level between 80 and 110 mg/dL or conventional therapy (i.e., an insulin infusion was provided only if the blood glucose level exceeded 215 mg/dL, with a goal between 180 and 200 mg/dL). During ICU care, there were 35 deaths (4.6%) in the

intensive treatment group (mean fasting glucose, 103 mg/dL) and 63 deaths (8.0%) in the conventionally treated group (fasting glucose, 153 mg/dL), a risk reduction of 42%. ($P = 0.005$). Overall in-hospital mortality was reduced by 34% ($P = 0.01$). The greatest benefit occurred in patients with multiorgan failure and sepsis. However, intensive therapy also reduced the duration of mechanical ventilation, the incidence of acute renal failure, and the need for blood transfusions.

Hospital utilization

Diabetes increases hospital stay not only for diabetes-associated admissions but for seemingly unrelated conditions as well. In one report, the average stay was almost twice that among nondiabetic patients, and **diabetes** doubled the risk of requiring intensive care ^[36]. Although the reasons for the longer hospitalization are not known, the additional hospital days likely resulted from complications of **diabetes** or from the time required to achieve glycemic control before procedures or discharge. Length of stay for diabetic patients can be decreased by more aggressive management. In the DIGAMI study, for example, the duration of hospitalization was 16% shorter in the insulin infusion group ^[37]. In another report, hospitals that implemented specific **diabetes** clinical pathways reduced the average length of stay for diabetic patients by 26% ^[38]. At another center, patients admitted with a primary diagnosis of **diabetes** had a 56% shorter stay when consultation with a **diabetes** management team was arranged ^[39]. Although economic modeling of these interventions is lacking, it is likely that they are cost saving.

Recommendations

Based on the best available data, particularly in the postoperative setting, it seems rational for good glycemic control to be a goal in the management of inpatients with **diabetes**. Specific management protocols, treatment algorithms, and clinical pathways should be developed and implemented by each institution, coordinating the roles of physicians, nurses, nutritionists, pharmacists, and discharge planners. The following are guidelines we have developed based on our own experience and, when available, published evidence.

Diet

Diet should be individualized, based on body weight, current medical status, and other comorbid conditions, such as obesity, hyperlipidemia, and hypertension. Consultation with the hospital nutritionist should be considered in all patients, providing a good opportunity to review dietary guidelines with the patient and family.

Glucose monitoring

Bedside blood glucose monitoring should be performed four times per day in all patients (before meals and at bedtime if eating; every 6 hours if not eating) for at least the first 48 hours. If the patient is medically stable, under good glycemic control, and receiving oral agents or one insulin injection per day, the frequency of monitoring can be decreased to twice daily.

Glucose control

General recommendations

Patients with type 1 **diabetes** will require some insulin at all times to prevent ketosis, even when not eating. For patients treated with insulin, the regimen, including rapid-/short-acting insulin sliding

scales if used ([Table 2](#)), should be revised frequently (i.e., every 1 to 2 days) based on the results of glucose monitoring. Patients should not remain on a sliding scale as their sole therapy. Adding long-/intermediate-acting insulin once or twice daily, even at a small dose, will stabilize control. Particularly in the postoperative intensive care setting and after myocardial infarction, glucose levels should be maintained as close to the normal range as safely possible. In other patients, a target blood glucose level of <200 mg/dL is supported by the literature and can be attained in most patients with proper planning. Individualized glycemic targets are necessary for each patient. In certain settings, such as in patients prone to hypoglycemic reactions (e.g., "brittle **diabetes**," hypoglycemia unawareness), in patients who are very elderly or have a short life expectancy due to comorbid conditions, or when adequate nursing or monitoring support are not available, more conservative targets should be considered. If glucose control is difficult to achieve, consideration should be given to consulting an internist or endocrinologist.

Table 2. Regular Insulin "Sliding Scale"*

Blood Glucose (mg/dL)	Rapid -/Short - Acting Insulin Dose (U)			
	Highly Insulin Sensitive	Normal Insulin Sensitivity (for most patients)	Highly Insulin Resistant	
<100 [†]	0	0	0	
100–149	0–1	0–2	0–5	
150–199	1	2	5	
200–249	2	4	10	
250–299	3	6	15	0–2
300–349	4	8	20	2–4
≥350	5	10	25	4–6

* In patients who are eating, sliding scale insulin should be given in conjunction with meals (regular insulin, 30 to 45 minutes before meals; insulin lispro or aspart, 0 to 10 minutes before meals). In patients who are not eating, regular insulin should be given every 6 hours.

[†] To achieve tighter control in patients who are eating, lower thresholds may be used (e.g., 70 to 100 mg/dL), and scales can be added to fixed doses of rapid -/short-acting insulin. To avoid hypoglycemia in patients who are not eating, higher thresholds (e.g., 200 mg/dL) should be considered, especially in patients with type 2 **diabetes**.

Adapted with permission from Inzucchi SE. *Yale Diabetes Center Facts and Guidelines*. New Haven, Connecticut: Yale **Diabetes** Center;2001:10.

Situation -specific recommendations

The patient usually treated with oral agents who is not eating. . In a well -controlled patient treated with an oral agent that may cause hypoglycemia (sulfonylurea or other secretagogues), hold the medication and use a short-/rapid- acting insulin sliding scale temporarily. If insulin is needed for more than 24 to 48 hours, consider adding a long -/intermediate -acting insulin to promote smoother control. In a well -controlled patient treated with an oral agent that does not cause hypoglycemia, the recommendations differ: hold metformin because of concerns about altered renal function in the

acutely ill; hold α -glucosidase inhibitors because they are effective only when taken with food; and continue thiazolidinediones if oral medications are allowed, unless the patient has abnormal hepatic or cardiac function.

In the poorly controlled patient usually treated with oral agents, a short-/rapid-acting insulin may be tried for 24 to 48 hours to assess insulin requirements. If it is clear that the patient will require insulin upon discharge, start a more permanent regimen, including a long-/intermediate-acting insulin.

The patient usually treated with oral agents who is eating . In the well-controlled patient who is taking a hypoglycemic agent, continue the medication but consider a dosage reduction of 25% to 50%, due to the likelihood of better dietary adherence, especially in those who are suboptimally compliant with diet as outpatients. In the well-controlled patient on an oral agent that does cause hypoglycemia, discontinue metformin if the patient is perioperative, if radiocontrast studies are imminent, if renal or hepatic function is altered, or if hemodynamic instability, heart failure, or dehydration are apparent, suspected, or anticipated; continue α -glucosidase inhibitors, unless the patient was admitted for a gastrointestinal symptom or illness; and continue thiazolidinediones, unless the patient has abnormal hepatic or cardiac function.

In the poorly controlled patient on oral agents, if glycemia does not improve rapidly during hospitalization, add additional oral agents as tolerated or begin insulin. (The slow onset of action of thiazolidinediones makes them less attractive when rapid improvement in glucose levels is desired.)

The insulin-treated patient who is not eating . In patients with type 1 **diabetes**, strongly consider using an intravenous insulin infusion (see below). Alternatively, give one half to two thirds of the patient's usual dose of long-/intermediate-acting insulin, along with a short-/rapid-acting insulin sliding scale. Unless the patient is very hyperglycemic (>200 mg/dL), provide a 5% dextrose solution at 75 to 125 cc/h for safety and access purposes. Check blood glucose levels every 6 hours (every 1 to 2 hours during insulin infusions).

Some insulin-treated patients with type 2 **diabetes** may have improved glycemic control when diet-restricted alone and require only short-/rapid-acting insulin. These patients are frequently obese and poorly compliant with diet as outpatients. Alternatively, give one half of the patient's usual dose of long-/intermediate-acting insulin, with a short-/rapid-acting insulin sliding scale. If insulin has been given, provide a 5% dextrose solution intravenously at 75 to 125 cc/h for safety and access, unless the patient is very hyperglycemic (>200 mg/dL). Check blood glucose levels every 6 hours. (Very insulinopenic type 2 diabetic patients may be managed more easily as type 1 patients, above.)

The insulin-treated patient who is eating . Continue insulin, although consider dosage reduction (10% to 50%) in well-controlled patients because of the likelihood of more rigid dietary adherence, especially in those with type 2 **diabetes** or who are suboptimally compliant with diet as outpatients.

Perioperative instructions

In general, surgeries and procedures should be scheduled for the early morning, when they will have the least effect on the patient's treatment program. Blood glucose levels should be monitored every 1

to 2 hours before, during, and after surgery or procedure. Use of short-/rapid-acting insulin by sliding scale as the patient's only insulin is discouraged because of the greater likelihood of wider fluctuations in blood glucose levels, especially in type 1 diabetic patients.

Type 1 diabetes . Place on an insulin drip (maintenance rate, 1 to 2 U/h) with a 5% dextrose solution at 75 to 125 cc/h, adjusted to maintain blood glucose levels between 100 and 150 mg/dL. Alternatively, give one half to two thirds of the usual dose of long-/intermediate-acting insulin on the morning of procedure. Do not give short-/rapid-acting insulin unless the blood glucose level is >200 mg/dL; if it is, use small doses (1 to 4 U to achieve blood glucose levels of 100 to 150 mg/dL).

Type 2 diabetes . If the patient is taking an oral hypoglycemic agent, hold the medication on the day of procedure and resume when tolerating a normal diet. Metformin must be held for safety concerns (i.e., possible perioperative alteration in renal function) and may be resumed 48 hours postoperatively after normal renal function is secured; α -glucosidase inhibitors should be held because these drugs are effective only when taken with meals; if pills are allowed, thiazolidinediones can be continued, although, due to their prolonged action, missing a dose or two should not affect glycemic control.

If the patient is usually treated with insulin, give one half of long-/intermediate-acting insulin on the morning of procedure. Do not give short-/rapid-acting insulin unless the blood glucose level is >200 mg/dL, and then in small doses (1 to 4 U to achieve a blood glucose level of 100 to 150 mg/dL). Alternatively, an insulin infusion can be used.

Insulin infusions

The typical indications for an intravenous insulin infusion are diabetic ketoacidosis and hyperglycemic hyperosmolar nonketotic syndrome. However, it should also be considered in any patient with poorly controlled **diabetes** despite subcutaneous insulin injections (e.g., blood glucose level >350 mg/dL on at least two determinations over 6 to 12 hours), as well as in patients with type 1 **diabetes** who are fasting or perioperative. Based on recent data, insulin infusions should also be considered in hyperglycemic postoperative ICU patients and diabetic patients with myocardial infarction.

Infusions should be started at 1 to 5 U/h, based on the degree of hyperglycemia. Greater rates may be required in highly insulin-resistant patients, including those who are obese, those with severe hyperglycemia (glucotoxicity), and those treated with steroids. When switching a well-controlled patient from subcutaneous to intravenous insulin therapy, divide one half of the total daily dose by 24 for an hourly rate. When the patient is fasting, carbohydrate calories should be provided to prevent catabolism using a 5% dextrose solution at a rate of 75 to 125 cc/h. The target blood glucose level is between 100 to 200 mg/dL, although a more aggressive target (80 to 110 mg/dL) may be indicated in postoperative ICU patients.

After initiating the infusion, measure the blood glucose level every hour for 4 hours until stable, then every 2 hours. For prolonged infusions in stable patients, monitoring may be reduced to every 4 hours. In hyperglycemic patients, intravenous insulin should decrease blood glucose levels by approximately 75 mg/dL per hour; if the blood glucose level is increasing or unchanged after 1 to 2 hours, increase the infusion rate by 50% to 100%. If the blood glucose level decreases by more than 100 mg/dL per hour, decrease the rate by 25% to 50%. If greater decreases occur, consider holding the drip for 1 hour and restarting at a rate that is 25% to 50% of the original rate. In patients who are

relatively well controlled, increase the infusion rate by 0.5 to 2 U/h if the blood glucose level is >200 mg/dL. If the blood glucose level falls below 100 mg/dL, hold the drip for 1 hour, then restart the infusion after decreasing the dose by 0.5 to 2 U/h. When switching a patient back to subcutaneous insulin therapy, give regular insulin 30 to 60 minutes before terminating the infusion.

Hospitals should maintain written protocols for the use of insulin infusions. Adequate nurse training for the preparation and administration of intravenous insulin solutions should be ensured. Intravenous insulin should only be administered when frequent glucose monitoring and close nursing supervision are possible. This does not imply that intravenous insulin should only be used in the ICU setting.

Education

A hospital admission should be used as a teaching opportunity for patients who lack **diabetes** knowledge or who require improvement in self-management skills. Consultation with the hospital's **diabetes** clinical nurse specialist or **diabetes** educator should be considered. Proper glucose monitoring and insulin injection techniques should be reviewed by the nursing staff. Pertinent literature, videotapes, and information on how to access educational materials upon discharge should be provided.

Discharge planning

Attempts should be made to approximate the ultimate at-home regimen as long as possible before discharge. Patients (and their families) should be familiar with their glucose targets as outpatients and should understand any changes made in the diabetic regimen. Appropriate outpatient **diabetes** follow-up should be secured, with telephone contact numbers given in case questions regarding **diabetes** management arise. Prescriptions (or samples) should be given for an adequate supply of oral agents, insulin, syringes, meters, test strips, lancing devices, and lancets, and a medical alert bracelet.

Conclusion

Studies in the perioperative setting suggest that there are short-term benefits from strict glycemic management in hospitalized patients with **diabetes**. Until further information is available, generalizing such an approach to all diabetic inpatients seems reasonable. Although there are many barriers to achieving such control in hospitals, many of these can be overcome. We present recommendations for glycemic management of diabetic patients during their hospitalization to encourage a more aggressive approach in the hospital setting to match that for outpatients. Additional clinical trials exploring the optimal degree of glycemic control for inpatients with **diabetes** are needed.

References

1. Roman SH, Harris MI. Management of **diabetes mellitus** from a public health perspective. *Endocrinol Metab Clin North Am* 1997;26:443-74. [Full Text](#)
2. Rubin R, Altman W, Mendelson D. Health care expenditures for people with **diabetes mellitus**. *J Clin Endocrinol Metab* 1994;78:809A-F. [Abstract](#)
3. Courtney L, Gordon M, Romer L. A clinical path for adult **diabetes**. *Diabetes Educ* 1997;23:664-71. [Abstract](#)
4. Queale WS, Seidler AJ, Brancati FL. Glycemic control and sliding scale use in medical inpatients with **diabetes mellitus**. *Arch Intern Med* 1997;57:545-52. [Abstract](#)
5. Rossini AA, Hare JW. How to control the blood glucose level in the surgical diabetic patient. *Arch Surg* 1976;111:945-9. [Abstract](#)

6. Walker R. Care of people with **diabetes** in hospital. *Nurs Stand* 1992;6:25-7. [Abstract](#)
7. Sawin CT. Action without benefit. The sliding scale of insulin use. *Arch Intern Med* 1997;157:489. [Citation](#)
8. Shagan BP. Does anyone here know how to make insulin work backwards? *Pract Diabetes* 1990;9:1-4.
9. MacMillan DR. The fallacy of insulin adjustment by the sliding scale. *J Ky Med Assoc* 1970;68:577-9. [Citation](#)
10. Gill G, MacFarlane I. Are sliding scale insulin regimens a recipe for diabetic instability? *Lancet* 1997;349:1555. [Citation](#)
11. MacMillan DR. Insulin adjustment by the sliding scale method a straw man who won't stay down. *J Ky Med Assoc* 1991;89:211-2. [Citation](#)
12. American **Diabetes** Association. Hyperglycemic crisis in patients with **diabetes mellitus**. *Diabetes Care* 2001;24(suppl):S83-90.
13. Hirsch IB, Paauw DS. **Diabetes** management in special situations. *Endocrinol Metab Clin North Am* 1997;26:631-45. [Full Text](#)
14. Malmberg K. Prospective randomised study of intensive insulin treatment on long term survival after acute myocardial infarction in patients with **diabetes mellitus**. *BMJ* 1997;314:1512-5. [Abstract](#)
15. 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. *Circulation* 1999;99:2626-32. [Abstract](#)
16. Golden SH, Peart-Vigilance C, Kao WL, Brancati FL. Perioperative glycemic control and the risk of infectious complications in a cohort of adults with **diabetes**. *Diabetes Care* 1999;22:1408-14. [Abstract](#)
17. 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. *Ann Thorac Surg* 1999;67:352-62. [Abstract](#)
18. Mowat AG, Baum J. Chemotaxis of polymorphonuclear leukocytes from patients with **diabetes mellitus**. *N Engl J Med* 1971;284:621-6. [Citation](#)
19. Sima AA, O'Neil SJ, Naimark D, et al. Bacterial phagocytosis and intracellular killing by alveolar macrophages in BB rats. *Diabetes* 1988;37:544-9. [Abstract](#)
20. Nolan CM, Beaty HN, Bagade JD. Further characterization of the impaired bactericidal function of granulocytes in patients with poorly controlled **diabetes**. *Diabetes* 1978;27:889-94. [Citation](#)
21. Bagade JD, Stewart M, Walters E. Impaired granulocyte adherence. *Diabetes* 1978;27:677-81. [Abstract](#)
22. Bagade JD, Root RK, Bulger RJ. Impaired leukocyte function in patients with poorly controlled **diabetes**. *Diabetes* 1974;23:9-15. [Citation](#)
23. Black CT, Hennessey PJ, Andrassy RJ. Short-term hyperglycemia depresses immunity through nonenzymatic glycosylation of circulating immunoglobulin. *J Trauma* 1990;30:830-3. [Abstract](#)
24. Joshi N, Caputo GM, Weitekamp MR, Karcher AW. Infection in patients with **diabetes mellitus**. *N Engl J Med* 1999;341:1906-12. [Citation](#)
25. Tenenberg S, Finkenauer R, Dwevedi A. Absence of lipopolysaccharide-induced inhibition of neutrophil apoptosis in patients with **diabetes**. *Arch Surg* 1999;134:1229-34. [Abstract](#)
26. Zykova SN, Jenssen TG, Berdal M, et al. Altered cytokine and nitric oxide secretion in vitro by macrophages from diabetic type II-like db/db mice. *Diabetes* 2000;49:1451-8. [Abstract](#)
27. Zerr KJ, Furnary AP, Grunkemeier GL, et al. Glucose control lowers the risk of wound infections in diabetics after open heart operations. *Ann Thorac Surg* 1997;63:356-61. [Abstract](#)
28. Watts NB, Gebhart SS, Clark RV, Phillips LS. Postoperative management of **diabetes mellitus** steady-state glucose control with bedside algorithm for insulin adjustment. *Diabetes Care* 1987;10:722-8. [Abstract](#)
29. Lillienfeld D, Viahov D, Tennay J, McLaughlin J. Obesity and **diabetes** as risk factors for postoperative wound infections after cardiac surgery. *Am J Infect Control* 1988;16:3-6. [Abstract](#)
30. Pozzilli P, Leslie R. Infections and **diabetes**: mechanism and prospects for prevention. *Diabetes Med* 1994;11:935-41.
31. Cagliero E, Roth T, Roy S, Lorenzi M. Characteristics and mechanisms of high glucose-induced over expression of basement membrane components in cultured human endothelial cells. *Diabetes* 1991;40:102-10. [Abstract](#)
32. Loots MA, Lamme EN, Mekkes JR, et al. Cultured fibroblasts from chronic diabetic wounds on the lower extremity (non-insulin-dependent **diabetes mellitus**) show disturbed proliferation. *Arch Dermatol Res* 1999;291:93-9. [Abstract](#)
33. Blakytyn R, Jude EB, Martin GJ, et al. Lack of insulin-like growth factor 1 (IGF1) in the basal keratinocyte layer of diabetic skin and diabetic foot ulcers. *J Pathol* 2000;190:589-94. [Abstract](#)
34. Koivukangas V, Annala AP, Salmela PI, Oikarinen A. Delayed restoration of epidermal barrier function after suction blister injury in patients with **diabetes mellitus**. *Diabetes Med* 1999;16:563-7.
35. Van den Berghe G, Wouters P, Weekers F, et al. Intensive insulin therapy in critically ill patients. *N Engl J Med*

2001;345:1359-67. [Abstract](#)

36. Krop JS, Saudek CD, Weller WE, et al. Predicting expenditures for Medicare beneficiaries with **diabetes**. *Diabetes Care* 1999;22:1660-6. [Abstract](#)

37. Malmberg K, Ryden 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. [Abstract](#)

38. **Diabetes** pathway slashes length of stay by 26%. *Hosp Case Manage*. 1999;Jan:8-9

39. Levetan CS, Salas JR, Wilets IF, Zumoff B. Impact of endocrine and **diabetes** team consultation on hospital length of stay for patients with **diabetes**. *Am J Med* 1995;99:22-8. [Abstract](#)

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