Management of Diabetes in the Hospital

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Hyperglycemia is a common complication in hospitalized patients, particularly among patients with acute myocardial infarction. It is an independent predictor of cardiovascular mortality and morbidity. Management of hyperglycemia with intensive insulin therapy has been shown to improve survival, reduce length of stay in intensive care, and decrease complications such as renal failure or prolonged mechanical ventilation in critically ill patients. Insulin infusions are now recommended for the treatment of hyperglycemia in several groups of patients, including patients in the intensive care unit and those undergoing major surgery. Implementation of protocols and standard orders may be useful to ensure the optimal use of insulin in the management of hyperglycemia. Fewer data are available to guide the management of hyperglycemia outside the intensive care setting. A variety of subcutaneous insulin regimens are now available with different pharmacokinetic profiles. These agents are preferred to controlling blood sugar with sliding scale regimens alone. Oral therapies may also have a role, but many agents may be contraindicated in the acute setting. As hyperglycemia has been shown to have significant adverse impact on patient outcomes in a variety of settings, cardiologists need to play a role in efforts to achieve adequate glycemic control in hospitalized patients with hyperglycemia in an effort to improve patient outcomes.
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There is now convincing evidence that insulin has many other properties beyond lowering glucose. In particular, insulin has anti-inflammatory properties, and it suppresses oxidative stress in patients whose blood glucose is normal or near normal.[1] However, in the setting of elevated blood

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glucose or possibly insulin resistance, these benefits of insulin may not be observed.[2,3]

Hyperglycemia plays a significant role in the pathogenesis of cardiovascular disease. It promotes the formation of reactive oxygen species, and it increases the production of intracellular advanced glycation end products.[4] This increase in oxidative stress contributes to a lower bioavailability of nitric oxide, the hallmark of endothelial dysfunction. In addition, vascular cell adhesion molecules are activated by the glycation end products, a process that stimulates the inflammatory cascade. Hyperglycemia is an independent predictor of cardiovascular morbidity and mortality.[5-7] The problem is compounded by the fact that even short-term elevations in blood glucose, such as post-prandial hyperglycemia, promote the formation of reactive oxygen species, which may exacerbate cardiac and vascular disease.[8] Several potential mechanisms lead to the independent relationship of hyperglycemia and cardiovascular disease (CVD) including glycosylation end products, stimulation of inflammation, and endothelial dysfunction.

The high prevalence of hyperglycemia among hospitalized patients may be due to several factors, including stress, carbohydrate intolerance, increased circulating catecholemines, or previously undiagnosed diabetes. Regardless of the cause, hyperglycemia is a negative prognostic factor in such patients.

Hyperglycemia is very common in the hospital setting, particularly after acute myocardial infarction (AMI). The high prevalence of hyperglycemia among hospitalized patients may be due to several factors, including stress, carbohydrate intolerance, increased circulating catecholamines, or previously undiagnosed diabetes. Regardless of the cause, hyperglycemia is a negative prognostic factor in such patients. However, the risk appears to be greater among hyperglycemic patients without a prior diabetes diagnosis, presumably because of the likelihood of increased endothelial damage associated with years of uncontrolled blood glucose.[9,10] Data are conflicting as to the value of treating hyperglycemia to goal in such patients, particularly after AMI. More controversial is the methodology used to achieve glycemic targets in such patients, as different studies have used a variety of protocols with different insulin dosing strategies, routes of administration, and monitoring parameters, many of which work in some settings but not in others. Intravenous insulin appears to achieve the best results due to its rapid onset of action and predictable pharmacokinetics. However, intravenous insulin therapy carries certain risks including hypoglycemia and the potential for dosing errors, and its administrative challenge–particularly outside the setting of intensive care units. On the wards, the majority of patients are treated with subcutaneous insulin, and many are not treated to goal. Subcutaneous insulin may fail to achieve blood glucose goals because of the tendency to rely on sliding scales without providing appropriate basal coverage, and because of failure to adjust the sliding scale dose according to the patient's response on the preceding days.[11] Both hyperglycemic and hypoglycemic events are quite common and blood glucose targets are often not achieved.

Some of the best evidence supporting the need for optimizing glycemic control in the inpatient setting comes from studies performed in the intensive care unit (ICU) setting.[12,13]

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Critically ill patients were treated with intravenous insulin to maintain a blood glucose level of 80 to 110 mg/dL, with 50% reductions in mortality, kidney failure, and a shorter hospital stay. Intensive insulin therapy was associated with a 42% risk reduction in mortality as compared to conventional therapy.[12] Intensive therapy was also associated with earlier weaning from mechanical ventilation (HR 1.21; 95% CI, 1.02-1.44, $P = .03$) and earlier discharge from the ICU (HR 1.15; 95% CI, 1.01-1.32, $P = .04$).[13] In patients with AMI, elevated blood glucose levels are a predictor of mortality, and they are associated with a larger infarct size and a greater propensity to develop congestive heart failure (CHF). Hyperglycemia may induce changes in the myocardium that lead to heart failure development through increased oxidative stress, glycosylation of proteins, alteration in protein kinase C isoforms, and cardioneuropathy. Activation of the sympathetic nervous system and increased cytokines may also play a role.[14] In addition, the insulin deficiency associated with hyperglycemia results in increased lipolysis and higher levels

of circulating free fatty acids. Free fatty acids increase myocardial oxygen demand, and as a result, they may lead to further myocardial cell damage during myocardial ischemia.[15] Ischemic preconditioning is also impaired, leading to increased infarct size.[16] Over the last few years, an attempt has been made to develop a consensus on glycemic targets in hospitalized patients. The American Association of Clinical Endocrinologists (AACE) has recommended that in the ICU setting, a blood glucose level of 110 mg/dL is desirable, whereas on medical and surgical wards, the preprandial blood glucose target should be 100 mg/dL and the maximum glucose level should be < 180 mg/dL.[17]

The American Association of Clinical Endocrinologists has recommended that in the ICU setting, a blood glucose level of 110 mg/dL is desirable, whereas on medical and surgical wards, the pre-prandial blood glucose target should be 100 mg/dL and the maximum glucose level should be less than 180 mg/dL.

The American Diabetes Association (ADA), in a technical review,[16] recommended an ICU target of 80 to 110 mg/dL and a non-ICU target of less than 110 mg/dL before meals, and a peak postprandial value of less than 180 mg/dL. Clement and colleagues recommend that intravenous insulin should initially be used to achieve these goals, followed by multi-dose subcutaneous insulin in patients post-AMI. They emphasize that IV or subcutaneous insulin infusion is safe and effective for achieving metabolic control during major surgery and in hemodynamic instability while a patient is not eating.[16] With the discontinuation of intravenous insulin, transition to a meal-related insulin schedule is preferable to sliding-scales.[16] The ADA and AACE have issued a joint consensus statement on the implementation of strategies to manage diabetes in hospital.[17]

Barriers to Improving Hyperglycemia Management

Considerable barriers exist to good management of hyperglycemia in the hospital setting. It requires incremental nursing time and effort, and some degree of skepticism remains about the benefits of good glycemic control in the hospital. However, reversal of hyperglycemia is associated with improved clinical outcomes. This improvement in outcomes has been most evident in the setting of post-MI, cardiac surgery, infection, and critical illness.[17] Cardiologists encounter these patients on a daily basis, and effectively managing hyperglycemia is an important intervention that can positively impact long-term outcomes. In addition, ensuring adequate glycemic control will become even more important as performance measures are developed to evaluate the extent to which hospitals are achieving glycemic goals.[17] The major obstacle to change in this area is the fear of hypoglycemia. A broad educational effort and process implementation, including integrated information systems to allow tracking and monitoring trends in glycemia data, are needed to overcome these barriers.

Table 1 outlines the strategy for inpatient diabetes management that includes a multidisciplinary team, established with the support of hospital administration, along with staff education. This team will enable the development and implementation of standardized dose titration protocols, metrics for evaluating achievement of glycemic goals, and standardized methods for post-discharge follow-up.

Table 1
Strategies to Improve Inpatient Diabetes Management

- Appropriate administrative support
- Formation of a multidisciplinary team
- Assessment of barriers to care
- System-wide change and acceptance
- Staff education
- Development and implementation of standardized and dose titration protocols
- Metrics for evaluation
- Patient participation
- Post-discharge follow-up

Pathophysiological Barriers for Achieving Good Optimal Glucose Control

Metchick and colleagues outlined several important changes in the pathophysiology of diabetes that contribute to poor glycemic control in the hospital (Table 2). These factors

Table 2
Changes in the Pathophysiology of Diabetes That Contribute to Poor Glycemic Control in the Hospital

Causing Hyperglycemia	
	"Stress hyperglycemia"
	Corticosteroid therapy
	Decreased physical activity
	Discontinuation of O/P regimen
	Medicine/insulin errors, interruptions
	"Insulinophobia"
Causing Hypoglycemia	
	Decreased caloric intake
	Meal interruptions
	Monitored compliance
	Medicine/insulin errors
	Insulin "stacking"
	Altered cognitive status

need to be addressed in order to achieve normal glycemia.[18] Abnormalities that lead to hypoglycemia include stress, drug therapy, decreased physical activity, discontinuation of outpatient treatment regimens, errors in medicine and insulin administration (including interruption in treatment), and, finally, a fear of using insulin. On the other hand, several factors tend to decrease blood glucose values and create separate problems. These include decreased calorie intake, meal interruptions, errors in insulin dosage, erratic absorption from subcutaneous tissue, and, finally, the altered cognitive state of the patient. Queale and colleagues found several predictors of both high and low blood glucose in the hospital setting.[11] Independent predictors of hyperglycemia included female gender, APACHE III score, severe diabetic complications, admission glucose, infectious disease as the admitting diagnosis, use of a conservative sliding scale (vs no sliding scale), and corticosteroid use. Use of either an oral agent or NPH insulin versus no standing regimen and dialysis conferred a lower risk of hyperglycemia. Low serum albumin was an independent predictor of hypoglycemic events, whereas corticosteroid use was associated with a lower risk of hypoglycemia.[11]

Stress hyperglycemia is an important mediator of glucose abnormalities in the hospital, and it has severe consequences for the patient, including osmotic diuresis, change in renal blood flow, halted wound healing and immune function, electrolyte shift, decreased endothelial function, inflammation, and secretion of a wide variety of potentially detrimental cytokines. In stressful conditions, the hypothalamic-pituitary-adrenal (HPA) axis is activated, resulting in cortisol release from the adrenal gland.[19] Norepinephrine, epinephrine, glucagon, and growth hormone release also increases. Peripheral insulin resistance is present, and insulin release may be further suppressed by interleukin-1 or tumor necrosis factor-α. Stress hyperglycemia results from the combination of low insulin levels, insulin resistance, and increased levels of counter-regulatory hormones.[19]

Insulin Infusion Protocols

Several protocols to guide inpatient insulin use have been published (Figures 1 and 2).[20-22] First, intravenous insulin has many advantages, and it can either be given mixed with glucose to provide nutrition or administered through a separate infusion line. Goldberg and associates described a protocol that is relatively simple to use, and it targets a blood

Figure 1. *An example of intravenous insulin infusion. BG, blood glucose. Reprinted with permission from Trence DL et al.*[20]

General Guidelines: **Goal BG = _____** (Usually 80-180 mg/dL)

- **Standard drip**: 100 Units/100 ml 0.9% NaCl via an infusion device.
- Surgical patients who have received an oral diabetes medication within 24hrs should start when BG>120 mg/dL. All other patients can start when BG≥70
- Insulin infusions should be discontinued when a patient is eating AND has received 1st dose of subcutaneous insulin.

Intravenous Fluids:

- Most patients will need 5-10GM of glucose per hour
  - D_5W or $D_5W1\backslash 2NS$ at 100-200 ml\hr or equivalent (TPN, enteral feeds, etc)

Initiating the Infusion:

- **Algorithm 1**: Start here for most patients.
- **Algorithm 2**: For patients not controlled with Algorithm 1, or start here if s/p CABG, s/p solid organ transplant or islet cell transplant, receiving glucocorticoids, or patient with diabetes receiving >80 units/day of insulin as an outpatient.
- **Algorithm 3**: For patients not controlled on Algorithm 2. NO PATIENTS START HERE without authorization from the endocrine service
- **Algorithm 4**: For patients not controlled on Algorithm 3. NO PATIENTS START HERE.
- Patients not controlled with the above algorithms need an endocrine consult.

Algorithm 1		Algorithm 2		Algorithm 3		Algorithm 4	
BG	Units/hr	BG	Units/hr	BG	Units/hr	BG	Units/hr
<60 = Hypoglycemia (See below for treatment)							
<70	Off	<70	Off	<70	Off	<70	Off
70-109	0.2	70-109	0.5	70-109	1	70-109	1.5
110-119	0.5	110-119	1	110-119	2	110-119	3
120-149	1	120-149	1.5	120-149	3	120-149	5
150-179	1.5	150-179	2	150-179	4	150-179	7
180-209	2	180-209	3	180-209	5	180-209	9
210-239	2	210-239	4	210-239	6	210-239	12
240-269	3	240-269	5	240-269	8	240-269	16
270-299	3	270-299	6	270-299	10	270-299	20
300-329	4	300-329	7	300-329	12	300-329	24
330-359	4	330-359	8	330-359	14	>330	28
>360	6	>360	12	>360	16		

Moving from Algorithm to Algorithm:

- Moving Up: An algorithm failure is defined as blood glucose outside the goal range (see above goal), and the blood glucose does not change by at least 60mg/dL within 1 hour.
- Moving Down: When blood glucose is <70 mg/dL X 2

Patient Monitoring

- Check capillary BG every hour until it is within goal range for 4 hours, then decrease to every 2 hours for 4hrs, and if remains stable may decrease to every 4 hours.
- Hourly monitoring may be indicated for critically ill patients even if they have stable blood glucose

Treatment of Hypoglycemia **(BG<60 mg/dL)**

- Discontinue insulin drip AND
- Give $D_{50}W$ IV
  - ❖ Patient awake: 25 ml (1/2 amp)
  - ❖ Patient not awake: 50ml (1 amp)]
- Recheck BG every 20 minutes and repeat 25ml of $D_{50}W$ IV if <60mg/dL. Restart drip once blood glucose is >70 mg/dl X2 checks. Restart drip with lower algorithm (see moving down)

Notify the physician:

- For any blood glucose change greater than 100 mg/dL in one hour.
- For blood glucose >360 mg/dL

For hypoglycemia which has not resolved within 20 min of administering 50ml of $D_{50}W$ IV and discontinuing the insulin drip.

Figure 2. *Subcutaneous insulin orders. Reprinted with permission from Trence DL et al.*[20]

Blood Glucose (BG) Monitoring: ☐ Before meals and at bedtime. ☐ _____Hrs after meals. ☐ 2-3 am

Goal Premeal BG = _____(80-150 mg/dL for most patients)

	Breakfast	*Lunch*	*Dinner*	*Bedtime*
Prandial Orders	Give _____units of: ☐ Lispro (Humalog ®) ☐ Aspart (Novolog®) ☐ Regular	Give _____units of: ☐ Lispro (Humalog ®) ☐ Aspart (Novolog®) ☐ Regular	Give _____units of: ☐ Lispro (Humalog ®) ☐ Aspart (Novolog®) ☐ Regular	
Basal Insulin Orders	Give _____units of: ☐ NPH ☐ Lente ☐ Ultralente ☐ Glargine	Give _____units of: ☐ NPH ☐ Lente ☐ Ultralente ☐ Glargine	Give _____units of: ☐ NPH ☐ Lente ☐ Ultralente ☐ Glargine	Give _____units of: ☐ NPH ☐ Lente ☐ Ultralente ☐ Glargine

Suggested Lag Times for Prandial Insulin:
Aspart/Lispro: 0-15 minutes before eating
Regular: 30 minutes before eating

For BG<60 mg/dL
A. If patient can take PO, give 15 grams of fast acting carbohydrate (4oz fruit juice/non diet soda, 8oz nonfat milk, or 3-4 glucose tablets)
B. If patient cannot take PO, give 25ml of D50 as IV push
C. Check finger capillary glucose q15 minutes and repeat above if BG<80

Premeal "correction dose" algorithm for Hyperglycemia: To be administered in addition to scheduled insulin dose to correct premeal hyperglycemia.
☐ Lispro
☐ Aspart

☐ **Low Dose Algorithm**
(For pts requiring ≤ 40 units of insulin/day)

Premeal BG	Additional Insulin
150-199	1 unit
200-249	2 units
250-299	3 units
300-349	4 units
>349	5 units

☐ **Medium Dose Algorithm**
(For pts requiring 40-80 units of insulin/day)

Premeal BG	Additional Insulin
150-199	1 unit
200-249	3 units
250-299	5 units
300-349	7 units
>349	8 units

☐ **High Dose Algorithm**
(For pts requiring >80 units of insulin/day)

Premeal BG	Additional Insulin
150-199	2 unit
200-249	4 units
250-299	7 units
300-349	10 units
>349	12 units

☐ **Individualized Algorithm**

Premeal BG	Additional Insulin
150-199	
200-249	
250-299	
300-349	
>349	

General Insulin Dosing Recommendations:
A. Patients with Type 1 Diabetes
This patient must have insulin to prevent ketosis. Even if the patient is not eating, he/she will need at least basal insulin (NPH/Lente/Ultralente/Glargine) to prevent ketosis.
1. When admitting a patient with Type 1 diabetes, continue the basal insulin that they were taking at home at the same dose. If the patient will be NPO, use an insulin drip rather than subcutaneous insulin. The prandial insulin (Regular/Lispro/Aspart) may require adjustment depending on the patient's situation. If the patient is eating much less the prandial insulin will need to be reduced. Many hospitalized patients are under significant metabolic stress (infection, glucocorticoids, etc.) and may require larger doses of prandial insulin despite eating less.
2. If a patient is newly diagnosed the usual daily insulin requirement is 0.5–0.7 units/kg/day. Half or 50% should be given as basal insulin and the remainder as prandial insulin.

B. Patients with Type 2 Diabetes
1. If patient is using insulin at home, continue the outpatient regimen and adjust as needed.
2. If patient has not been using insulin previously, the usual total daily insulin requirement is 0.4–1.0 units/kg/day.

Note: Individual insulin doses vary widely and adjustments should be made based on the bedside and laboratory glucose levels.

glucose of 100 to 139 mg/dL. Insulin infusion rates are altered based on frequent blood glucose monitoring.[21] Others have described a protocol that has the advantage of changing infusion rates for different levels of glucose, depending on the patient's insulin-resistant status, which in turn may reflect the severity of illness.[20,22] It is appropriate for hospitals to develop their own infusion protocols based on resources that are available. Blood glucose levels must be monitored frequently regardless of the protocol used, a requirement that may be challenging until better methods for such monitoring become available.[23] Clement and colleagues recommend that capillary blood glucose be checked every hour until it is within the goal range for 4 hours. Monitoring can then be decreased to every 2 hours for 4 hours, and if it remains stable, then monitoring may be decreased to every 4 hours. However, hourly monitoring may be indicated for critically ill patients even if the blood glucose has been stable.[16]

Management of Patients Outside the Intensive Care Unit

The management of a patient on the ward is more complex, in that there are no clearcut approaches to manage every patient, and multiple clinical situations must be considered. Very little research has been done to assess treatment protocols in this setting and to relate them to important patient outcomes. The appropriate strategy to maintain optimal glycemic control depends on the type of diabetes (type 1 vs type 2), which regimen the patient has been using as an outpatient, and whether adequate glycemic control has been achieved with the regimen. It is also dependent on the current glucose, the patient's dietary status, and the reason for hospitalization. These factors must be considered because they may influence the risk of hypoglycemia, the pharmacokinetics of subcutaneous insulin, or the patient's oral intake. For patients whose stay is very short it may be impossible to institute insulin regimens for optimal glycemic control. On the other hand, for a patient whose stay is likely to be more than 2 to 3 days it may be possible to start a treatment regimen that is based on the patient's nutritional status and level of glycemic control. In addition, some oral therapies may be contraindicated depending on the patient's comorbidities and/or reason for hospitalization. These variables are ultimately used to determine whether subcutaneous or intravenous insulin, or oral agent administration is most appropriate. As previously described, insulin infusions are preferred in severely decompensated type 1 diabetes (with or without ketoacidosis), type 2 diabetes with hyperglycemic hyperosmolar state, intensive care settings, and in patients who require prolonged periods without oral intake after surgical procedures.[16]

Many oral agents are available to treat type 2 diabetes, and patients are frequently prescribed combinations of these medications. Some of these therapies may be contraindicated in the acute setting. For example, acutely ill patients may be at greater risk of developing lactic acidosis on metformin therapy, particularly in clinical situations such as heart failure or MI. Similarly, thiazolidinediones (TZD) may need to be discontinued in the presence of edema or congestive heart failure. In general, sulfonylureas may be continued, with changes in dose or interval as needed based on formulary considerations. Theoretical considerations against using sulfonylureas include the propensity of some first generation sulfonylureas to block ischemic preconditioning.[24] However, this concept has not been supported by data from long-term outcome studies, and it is possibly not an issue with newer sulfonylureas.[24] In general, insulin is recommended in place of oral agents in the ICU.[16]

Insulin sliding scales are frequently used in the hospital, mainly because they are easy to order and seem to be ingrained in medical practice. However, they have many disadvantages including marked fluctuations in blood sugars because the insulin dose is calculated retrospectively. Absorption of subcutaneous insulin may be altered in edematous states or in the setting of decreased perfusion. In addition, insulin requirements often rapidly change in the acute setting, and sliding scales may be inadequate to respond to the patient's changing insulin needs. Protracted hypoglycemia may also be problematic.[16] In particular, sliding scales are not evidenced based, and in fact, most studies argue against their usage. They are labor intensive and not proactive, and they frequently dose with too little insulin. Several alternatives exist to sliding scales. These include basal insulin therapy alone, preferably with long-acting analogs, given once or twice a day; or basal bolus therapy with a long-acting or intermediate-acting analog, given once or twice a day, with a short-acting analog of insulin given with meals. It may also be possible to manage some patients on premixed insulin or self-mixed insulin. Over and above the scheduled doses, "correction doses" may also be given.

For well-controlled patients, it is generally acceptable to continue with the home insulin regimen. If the patient is using a non-formulary insulin, it may be appropriate to switch to a formulary insulin with similar pharmacokinetics. For patients with

poorly controlled diabetes, the home regimen may need to be adjusted with higher doses of insulin, or the patient could be switched to a more physiological basal insulin regimen.

For patients who were not being treated with insulin prior to admission, it is usually appropriate to start with low doses of basal insulin (0.2 to 0.3 units/kg per day, along with 0.05 units/kg per meals as a prandial bolus). For example, a 100-kg male could be started on a long-acting insulin in a dose of 20 to 30 units at bedtime and 5 units of rapid-acting insulin with each meal. In addition, a pre-meal correction may be appropriate, based on a blood glucose at that time.

In order to optimally prescribe insulin, physicians must become familiar with the pharmacokinetics of various insulin preparations. These range from rapid-acting analogs (lispro, aspart, and glulisine), to long-acting analogs, such as glargine and detemir. Long-acting analogs are routinely administered at night, and short-acting insulin is typically administered with meals. Table 3 summarizes the pharmacodynamics of several available insulin products.

It is also important to recognize that the insulin requirement may change in a hospitalized patient and, in fact, patients who are acutely ill often require higher doses of insulin, despite the fact that their caloric intake is low. Several special situations need to be addressed in hospitals. Perioperative care may often require an insulin infusion as described above and frequent blood glucose monitoring. Enteral feeding is also a special circumstance that may warrant consideration. These patients can often be treated with a basal insulin dose once or twice a day. Treatment during states of iatrogenic insulin resistance is of particular importance, including steroid therapy and the use of total parenteral nutrition (TPN), both of which induce severe insulin resistance that will require higher doses of intravenous insulin.

Hypoglycemia management in the hospital is important in order to prevent a rebound rise in blood glucose due to counter-regulatory hormone secretion or overzealous treatment. Either directly or indirectly, hypoglycemia may be associated with cardiac ischemia and increased mortality.[16-18] Hypoglycemia in conscious patients is best treated with glucose tablets or fruit juice, with repeat blood glucose monitoring in 15 minutes. Intravenous glucose or glucagon may be used in patients who are unable to eat, but the possibility of severe rebound hyperglycemia should be considered.

Finally, a plan for maintenance and follow-up is needed for patients prior to hospital discharge. The discharge plan should emphasize the correct diagnosis, particularly for patients who are newly diagnosed with diabetes. When a diagnosis is unclear, a plan to establish the diagnosis over the next few weeks should be formulated. The discharge plan should have clear recommendations for the therapeutic approaches to achieve short and long-term glycemic control and for follow up diabetes education. Insulin may be needed in the short-term for a patient with infection, or steroid therapy might be recommended, but then the patient could be switched to oral agents upon full recovery. It is also important to recognize that patients with newly diagnosed type 2 diabetes may have other complications of diabetes, and they should be screened for diabetic retinopathy

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Table 3
Insulin Treatment:
Comparison of Human Insulins and Analogues

Preparation	Onset	Peak	Duration
Lispro/Aspart	5-15 min	1-2 h	4-6 h
Regular	30-60 min	2-4 h	6-10 h
NPH/Lente	1-2 h	4-8 h	10-20 h
Ultralente	2-4 h	Unpredictable	16-20 h
Glargine	1-2 h	Flat	~24 h
Detemir	1-2 h	Less flat	12 h

The time course of action of any insulin may vary in different individuals, or at different times in the same individual. Because of this variation, time periods indicated here should be considered general guidelines only.

and nephropathy. Consideration should also be given to the use of aspirin, statins, and ACE inhibitors, as per treatment guidelines.

Conclusion

A greater focus has been placed on inpatient diabetes management following publication of a variety of clinical trials demonstrating general benefit, but occasionally lack of benefit, with insulin infusions. The effect appears to be greatest in patients in intensive care units. A greater degree of controversy exists surrounding the appropriate treatment in patients on hospital wards, and in patients whose stay in the hospital is relatively short. Significant advances in the management of diabetes have been made in the last few years, with the development of insulin analogs with different pharmacokinetic profiles. As a result, therapy can now be tailored to each patient's unique clinical status and requirements. These developments should allow most patients to be treated to individualized goals. ■

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Main Points

- Hyperglycemia is very common in the hospital setting, particularly after acute myocardial infarction.
- Hyperglycemia in the hospital setting is an independent predictor of cardiovascular morbidity and mortality.
- Intensive insulin therapy used to achieve normal blood glucose in critically ill patients has been associated with improved outcomes.
- Insulin infusions are the preferred method of insulin administration for several groups of patients, including patients in the intensive care unit, severely decompensated diabetics, and patients without oral intake for a prolonged period post-operatively.
- Subcutaneous insulin using combinations of long-acting and short-acting basal insulin is preferred to sliding scale regimens for the management of hyperglycemia in patients outside the intensive care unit setting.