Hyperglycaemia in inpatients with type 2 diabetes mellitus

A national audit of NHS hospitals reported that approximately 15% of inpatients have diabetes mellitus, with the majority of these admissions being unrelated to diabetes (NHS Diabetes, 2010). The added cost to the NHS associated with the management of diabetes in hospital is estimated to be £573–686 million per year. People with diabetes are more likely to be admitted and have a longer average stay in hospital (Kerr, 2011). Despite the added cost, inpatient care for people with diabetes is often suboptimal, including medication errors, new complications (e.g. foot ulcers) and lack of input from the diabetes team (NHS Diabetes, 2010; Kerr, 2011).

Hyperglycaemia is commonly encountered among inpatients – this may occur in patients with known diabetes, previously undiagnosed diabetes or temporarily as part of an acute illness, also known as 'stress hyperglycaemia'. Pronounced hyperglycaemia is a predictor of poor hospital outcomes including increased risk of postoperative infections (Zerr et al, 1997) and intensive care unit admissions, and is an independent risk factor for higher mortality (Malmberg et al, 1999). In particular, hyperglycaemia on admission in previously normoglycaemic patients with myocardial infarction is associated with an increased risk of in-hospital mortality compared to those with established diabetes (Capes et al, 2000).

Dr Sindu Mohan *is Resident in Internal Medicine in the Department of Internal Medicine, London Health Sciences Centre, University of Western Ontario, London, Canada,* **Dr Daniel R Fenton** *is GPST1, Adelaide Medical Centre, London,* **Dr Andrew Solomon** *is Locum Consultant Physician and Endocrinologist, Princess Alexandra Hospital, Harlow, Essex and* **Dr Jacob F de Wolff** *is ST6 in Acute Medicine, Northwick Park Hospital, Harrow, Middlesex HA1 3UJ*

Correspondence to: Dr JF de Wolff (jfdwolff@doctors.org.uk)

It has been suggested that very tight (blood glucose below 6.3 mmol/litre) or reasonably tight glycaemic control (blood glucose below 8.3 mmol/litre) using insulin infusions might reduce inpatient morbidity and mortality as well as health-care costs. Various studies, mostly in intensive care, have looked at strict (keep glucose below 6.3 or 8.3 mmol/litre using insulin) *vs* a more lenient approach (start insulin if blood glucose exceeds 11.9 mmol/litre and maintain between 10 and 11 mmol/litre) to managing hospital hyperglycaemia. This was shown first in surgical and then in medical intensive care unit patients.

A subsequent meta-analysis of these trials revealed that while there was an improvement in the risk of sepsis, the risk of hypoglycaemia was increased and no mortality benefit could be demonstrated (Wiener et al, 2008). A further clinical trial comparing tight (4.5–6 mmol/litre) *vs* lenient (<10 mmol/litre) glycaemic control in 6000 patients in critical care found that tight glycaemic control led to an increased risk of death (27.5% *vs* 24.9%) (NICE SUGAR Study Investigators, 2009). The evidence for tight glycaemic control is therefore limited, but treatment of very high blood sugar levels remains important to prevent osmotic symptoms, which typically arise when blood sugars exceed 11.9 mmol/litre (Mesotten and Van den Berghe, 2012).

Pathophysiology

Hyperglycaemia in hospitalized patients is multifactorial.

Stress and insulin resistance

The initial phase of the stress response in acute illness such as trauma, burns, sepsis and postoperatively results in a state of transient hyperglycaemia. This is the result of insulin resistance from increased secretion of counter-regulatory hormones such as cortisol, catecholamines, growth hormone and cytokines (Dungan et al, 2009). The effect of these mediators ultimately leads to increased hepatic release and reduced peripheral uptake of glucose.

Steroid-related insulin resistance

Corticosteroids cause hyperglycaemia through a number of mechanisms, both acutely and subacutely. At a skeletal muscle level, where under normal circumstances 80% of postprandial glucose is taken up, glucocorticoids impair insulin receptor signalling-mediated glucose uptake. Receptor signalling is impaired, and migration of the glucose transporter 4 to the cell surface is reduced. Moreover, glycogen synthesis in myocytes is reduced. Skeletal muscle protein degradation into amino acids and glucocorticoid-mediated lipolysis appears to indirectly play a role in impairing insulin signalling (van Raalte et al, 2009). In the liver, glucocorticoids cause insulin resistance, endogenous glucose production, and unfavourable changes in lipid metabolism (van Raalte et al, 2009).

On a β-cell level, glucocorticoids impair insulin secretion in the short term, which explains why the peripheral insulin resistance is not matched by an appropriate insulin response (van Raalte et al, 2009).

Mechanism of hyperglycaemia-related complications

Various mechanisms have been proposed for the link between hyperglycaemia and poor outcomes. Hyperglycaemia relates to a state of relative insulin deficiency which promotes fatty acid metabolism and this response is exaggerated in acute illness (Capes et al, 2000). Hyperglycaemia promotes oxidative stress with the production of reactive oxygen species, and stimulates a pro-inflammatory state (Dungan et al, 2009). These inflammatory cytokines are toxic to organs in acute illness, for example exacerbating injury to an ischaemic myocardium (Capes et al, 2000). The inflammatory state results in further hyperglycaemia with the release of counter-regulatory hormones and ends in a vicious cycle.

Hyperglycaemia negatively affects wound healing. In animal models, high glucose suppresses the activity of runt-related transcription factor 2, which promotes wound healing by up-regulating

endothelial cell migration, remodelling processes and angiogenesis (D'Souza et al, 2009). Mouse models of diabetic ulcers show impaired wound closure, but with no real correlation between the severity of hyperglycaemia and wound closure variability (Trousdale et al, 2009).

Assessing the patient

In acutely admitted patients, it is common for the clinical team to focus on addressing the patient's admission problem, and to relegate his/her diabetes management to the background (Lleva and Inzucchi, 2011). This may be associated with the fact that in most inpatient observation systems, blood sugar charts are kept separate from the general charts that are used to document pulse, temperature, blood pressure, respiratory rate and early warning score. As a result, it is very easy to miss an opportunity to review blood glucose charts in known diabetic inpatients (Lleva and Inzucchi, 2011).

Frequently, the issue of a raised blood sugar is only addressed when mentioned briefly by a member of the nursing staff: 'Doctor, this patient has a high blood sugar, please review'. Often a junior doctor may simply prescribe a dose of short-acting insulin, only to be bleeped a few hours later to hear the phrase: 'The patient's blood sugar went down a bit, but it's gone up again'. Hyperglycaemic events are not necessarily handed over to the patient's usual team, who might discover the problem only when reviewing the prescription chart showing repeated doses of short-acting insulin and glucose charts showing peaks and troughs.

The first step of appropriate assessment should be a bedside impression: is this patient sick or well? Is the patient oriented or confused, and are there signs of sepsis? Could this be a hyperglycaemic hyperosmolar state or diabetic ketoacidosis? The latter is usually associated with type 1 diabetes, but may occur in type 2 diabetics, particularly in Afro-Caribbeans. Physical examination, including a fluid balance assessment, may reveals signs of volume depletion. A thorough look at the observation chart including assessing the trends in daily blood glucose readings is vital. Next, a review of diabetic medications (*Table 1*), steroids, nasogastric feeds and total parenteral nutrition should be undertaken to assess the compounding factors contributing to the hyperglycaemia (*Table 2*). If supplementary insulin has been given, the chart can give an impression as to the requirements.

Bedside tests can provide key information. A urine dipstick test can help assess for ketosis. If the patient is unwell, or urinary ketones are elevated, an arterial or venous blood gas can be used to check for acidaemia, and also give immediate electrolytes to allow calculation of the anion gap. In an unwell patient, determination of the renal function is important, particularly if the patient is currently taking metformin (which should be discontinued in those with a significantly impaired renal function or raised lactate levels).

Management

Various approaches have been taken to manage inpatient hyperglycaemia. This depends on the admission diagnosis and the clinical setting, but also importantly on whether the patient is stable or unstable, and whether the oral intake is predictable. Mild hyperglycaemia could be tolerated, but it is widely accepted that blood sugar levels persistently above 11.9 mmol/litre (the renal threshold) should be addressed (Mesotten and Van den Berghe, 2012).

Table 1. Oral medications used to treat type 2 diabetes mellitus

Drug class	Drug examples	Mechanism of action	Expected reduction in HbA_{1c} (%)	Potential side effects
Biguanides	Metformin	Inhibit hepatic gluconeogenesis via activation of AMP-activated protein kinase, decreases gastrointestinal glucose uptake and increases peripheral use of glucose	1.5–2.0	Common: gastrointestinal upset including nausea, diarrhoea and decreased appetite. Less common: lactic acidosis (risk factors include renal disease, cardiac disease, hypoperfusion and old age)
Sulfonylureas	Gliclazide, glipizide, glibencamide, glimepiride, tolbutamide	Augments insulin secretion via action on ATP-dependent potassium channels on the beta islet cells of the pancreas	0.8–2.0	Generally mild and infrequent but can include gastrointestinal disturbance, hypoglycaemia
Alpha-glucosidase inhibitors	Acarbose	Inhibit alpha glucosidase in the brush border of the small intestine which results in inhibition of digestion of starch and sucrose	0.7–1.0	Flatulence, abdominal distention and pain
Thiazolidinediones	Pioglitazone	Selectively activate peroxisome proliferator-activated receptor gamma, which causes increased insulin sensitivity	0.5–1.5	Fluid retention and weight gain, liver dysfunction, bladder neoplasia, heart failure
Meglitinides	Repaglinide, nateglinide	Increases insulin secretion via action on ATP-dependent potassium channels on the beta islet cells of the pancreas	0.5–2.0	Hypoglycaemia, hypersensitivity reactions including pruritus, rash and urticaria
DPP-4 inhibitors	Sitagliptin, saxagliptin, vildagliptin	Inhibit DPP-4 resulting in potentiation of the action of endogenous GLP-1 and a subsequent increase in insulin secretion and decrease in glucagon production	0.89	Gastrointestinal disturbances including nausea, dyspepsia and gastritis, peripheral oedema; rare finding of liver dysfunction and pancreatitis has been reported

AMP = adenosine monophosphate; ATP = adenosine triphosphate; DPP-4 = dipeptidyl peptidase 4; GLP = glucagon-like peptide; HbA_{1c} = glycated haemoglobin. From Nathan et al (2009), Ibrahim (2010), Joint Formulary Committee (2011)

Specific admission diagnoses

In patients admitted to hospital with severe sepsis, the 2008 Surviving Sepsis recommendations (Dellinger et al, 2008) still suggest a target blood sugar of 8.3 mmol/litre, to be maintained with a dose-adjusted insulin infusion or 'sliding scale'. In patients admitted with an acute coronary syndrome, a National Institute for Health and Clinical Excellence guideline recommends that blood sugars be kept below 11 mmol/litre, usually without intensive insulin therapy, using a dose-adjusted insulin infusion if required (National Institute for Health and Clinical Excellence, 2011).

Oral agents or insulin

If the patient is relatively well, with normal oral intake of food and drink, had reasonable glycaemic control before admission, and stable cardiac and renal function, it may be appropriate to introduce or titrate oral agents (Wesorick et al, 2008). However, oral agents are not easily titrated as a result of a combination of factors, including their mechanism of action, onset and duration of action as well as potential side effects. Sulphonylureas are relatively short-acting. However, in the hospital setting, a patient's nutritional status is not the same as when he/she is well. If calorific intake is significantly lower then there is a significant risk of hypoglycaemic events when altering these drugs.

Metformin has a slower mode of onset, and in unwell patients has been associated with a small risk of lactic acidosis in the context of hypoperfusion, hypoxia and exposure to contrast agents (Clement et al, 2004). Glitazones have a slow onset of action, and are contraindicated in patients with heart failure; moreover, their safety profile is increasingly in question. Acarbose has no role for patients who are fasting or have very poor nutritional intake. If a decision is made to introduce an oral agent, such as a sulphonylurea, this should be at a low dose followed by assessment of response. This may be chosen if the precipitant of the hyperglycaemia is likely to persist for some time, such as the use of steroids to treat temporal arteritis.

Table 2. Causes of hyperglycaemia in hospital inpatients

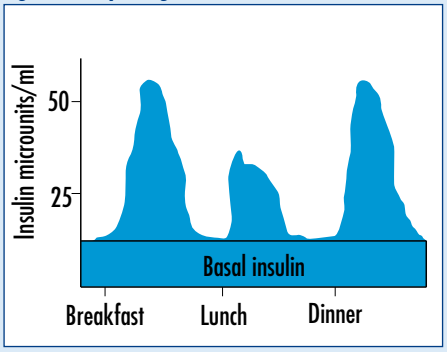
Stress response in acute illness
Drugs (e.g. glucocorticoid therapy)
Inappropriate use of dextrose-containing fluids
Total parenteral nutrition
Omission of diabetic medications
Inadequate monitoring
Unpredictable dietary intake
Decreased level of activity
Lack of knowledge among medical staff
Reluctance to treat high sugars for fear of inducing hypoglycaemia

Difficulty in controlling hyperglycaemia often prompts clinicians to prescribe insulin sliding scales. This could be seen as advantageous because of its convenience and simplicity, but it is not an appropriate approach to management. There is no good evidence to suggest that intravenous insulin has a role for non-critically ill patients (Clement et al, 2004). There is over-reliance on sliding scale 'coverage' as opposed to an active management plan for ward-based patients with hyperglycaemia (Lleva and Inzucchi, 2011).

A better answer in unwell patients: subcutaneous regimens

Insulin is undoubtedly easier to titrate than oral agents. Subcutaneous insulin is a reasonable approach for the vast majority of non-critically ill patients (Dungan et al, 2009). There are various advantages to using subcutaneous regimens. First, there is no need for dedicated intravenous access, avoiding the risks of nosocomial infection. Second, it allows clinicians to tailor a physiological regimen for the patient (*Figure 1*). In terms of glycaemic control, the prospective Randomized Study of Basal Bolus Insulin Therapy in the Inpatient Management of Patients with Type 2 Diabetes (RABBIT 2) by Umpierrez et al (2007), which compared subcutaneous insulin regimens with a sliding scale, showed no significant difference in patients with type 2 diabetes on general medical wards. A smaller study using historical controls found a significantly reduced risk of both hyper- and hypoglycaemia using a subcutaneous regimen (Perera et al, 2011) (*Table 3*). The introduction of a standardized subcutaneous insulin prescription chart that triggered daily reviews led to more time spent in an acceptable range (glucose 4–9.9 mmol/litre) and less hypoglycaemia (Cheung et al, 2011).

Figure 1. Physiological insulin secretion.



By using a subcutaneous regimen, one attempts to closely mimic the natural insulin secretion of a non-diabetic patient. The regimen consists of three components: a basal insulin, a prandial insulin and supplemental insulin (Clement et al, 2004). Total daily insulin requirement is typically 0.3–0.6 units/kg/day. The basal insulin accounts for approximately 50% of the total daily insulin requirement. The other 50% represents the prandial insulin – this is administered in equally divided doses in response to each meal. Supplemental insulin is then given as required to account for 'unexpected' rises in blood glucose (Wesorick et al, 2008).

To determine the dosing, an adequate assessment and some clinical judgment is required. A patient who is likely to be relatively insulin sensitive (as evidenced by a low body mass index, frailty or elderly) should be started at a conservative dose of 0.3 units/kg/day. On the other hand, a patient with sepsis, on high dose steroids, or obese with likely insulin resistance will require a larger dose.

To apply this principle to the case vignette in *Figure 2* Mr C weighs 85 kg. Given that he is unwell with an infective exacerbation of chronic obstructive pulmonary disease and has been commenced on steroids, his insulin requirements are significantly increased. He will require a total daily insulin dose of approximately 0.6 units/kg = 0.6 units x 85 kg/day = 51 units. This replaces his oral antidiabetics until his condition has improved. He should also be advised to avoid foods that are likely to lead to rapid rises in blood sugars.

Of this insulin, 50% should be given as a long-acting agent (first line Insulatard or Humulin I, or possibly second line Levemir

or Lantus) in the morning (basal). The remaining 25 units should be given in three divided doses of 7–8 units of short acting insulin (Novorapid, Humalog, or Humulin S) with each meal. As Mr C currently has an elevated blood glucose he should also receive a correction dose of insulin, i.e. 2–4 units of short-acting insulin. This regimen is flexible and will require up and down titration as the clinical condition changes, but avoids the 'roller coaster' of the insulin sliding scale, as well as the nuisance of hourly fingerprick blood sugar measurements. Subcutaneous insulin can be discontinued when the patient has improved clinically, and the sugars have stabilized. If the patient is known to be diabetic, previous treatment can often be resumed.

While all doctors looking after inpatients should have an appreciation of the basics of diabetes management, inpatient hyperglycaemia often warrants early involvement of the local diabetes team, often through the diabetes nurse specialist.

Conclusions

Inpatient hyperglycaemia is a common occurrence and has an important impact on outcomes. Insulin sliding scales are best avoided where possible, but judicious use of subcutaneous regimens can allow good blood sugar control. Better management of in-hospital hyperglycaemia can lead to reduced length of stay, increased patient safety and improved patient experience. BJHM

Conflict of interest: none.

Capes SE, Hunt D, Malmberg K, Gerstein HC (2000) Stress hyperglycaemia and increased risk of death after myocardial infarction in patients with and without diabetes: a systematic overview. *Lancet* **355**(9206): 773–8

Cheung NW, Cinnadaio N, O'Neill A, Koller L, Pratt HL, Zingle C, Chipps DR (2011) Implementation of a dedicated hospital subcutaneous insulin prescription chart: effect on glycaemic control. *Diab Res Clin Pract* **92**: 337–51

Clement S, Braithwaite SS, Magee MF et al (2004) Management of diabetes and hyperglycemia in hospitals. *Diabetes Care* **27**(2): 553–91

Dellinger RP, Levy MM, Carlet JM et al (2008)

TOP TIPS

- Most patients with high blood sugar levels, particularly when able to eat and drink, do not need an insulin sliding scale.
- If called to assess a patient for hyperglycaemia out-of-hours, ensure that the patient's own team is informed in due course so the patient's treatment can be adjusted and/or referred to the diabetes team.

Table 3. Insulins used for subcutaneous regimens

Insulin		Onset of action	Duration
Short-acting analogs	Insulin aspart (NovoRapid)	10–20 minutes	3–5 hours
	Insulin lispro (Humalog)	15–30 minutes	2–5 hours
	Insulin glulisine (Apidra)	10–20 minutes	3–5 hours
Short-acting animal insulins	Hypurin porcine neutral	30–60 minutes	6–8 hours
	Hypurin bovine neutral	30–60 minutes	6–8 hours
Short-acting human insulins	Humulin S	30 minutes	Up to 12 hours
	Actrapid	30 minutes	7–8 hours
	Insuman Rapid	30 minutes	7–9 hours
Intermediate-acting animal insulins	Hypurin porcine isophane	2 hours	18–24 hours
	Hypurin bovine isophane	2 hours	18–24 hours
Intermediate-acting human insulins	Humulin I	2–4 hours	10–16 hours
	Insuman basal	Within 60 minutes	11–20 hours
	Insulatard	Within 90 minutes	Up to 24 hours
Long-acting analogs	Insulin detemir (Levemir)	3–4 hours	Up to 24 hours
	Insulin glargine (Lantus)	3–4 hours	22–24 hours
Long-acting animal insulins	Hypurin bovine lente	2 hours	Up to 30 hours
	Hypurin bovine PZI	4–6 hours	24–36 hours
Pre-mixed analog insulins	Humalog Mix 25/50 (25/50% insulin lispro/75/50% insulin lispro protamine)	15 minutes (short-acting component)	15–22 hours (basal insulin)
	NovoMix 30 (30% soluble insulin aspart/ 70% protamine crystallized insulin aspart)	10–20 minutes	Up to 24 hours
Pre-mixed animal insulins	Hypurin porcine 30/70	2 hours	Up to 24 hours
Pre-mixed human insulins	Humulin M3 (insulin soluble human/ isophane human)	30 minutes	Up to 24 hours
	Insuman Comb 15/25/50 (a biphasic isophane insulin suspension with 15/25/ 50% dissolved insulin)	30–60 minutes	11–20 hours

From Electronic Medicines Compendium (2012)

Figure 2. Case scenario.

You are a junior doctor on the respiratory ward. On your round you see Mr C, who was admitted 2 days earlier with an infective exacerbation of chronic obstructive pulmonary disease. In addition he also suffers from type 2 diabetes and hypertension. The nurse attracts your attention to his blood sugar chart, which shows that since his admission his blood sugar has been markedly elevated. This morning, it was 29 mmol/litre, and it has not been below 15 mmol/litre. He reports polyuria and thirst, and is still quite breathless. His normal medication includes metformin 1 g twice daily and gliclazide 40 mg twice daily, as well as ramipril 5 mg once daily and salbutamol, tiotropium, salmeterol and fluticasone inhalers. His treatment in hospital has so far consisted of regular nebulisers, prednisolone, oral antibiotics and controlled oxygen therapy.

On examination, he is still quite wheezy and hypoxic, but not confused or dehydrated. He is centrally obese, weighs about 85 kg (body mass index 32 kg/m^2), and has a normal renal function. His last glycated haemoglobin was 8.0% (64 mmol/mol).

How do you approach the problem of Mr C's hyperglycaemia?

Surviving Sepsis Campaign: international guidelines for management of severe sepsis and septic shock: 2008. *Crit Care Med* **36**(1): 296–327
D'Souza DR, Salib MM, Bennett J et al (2009) Hyperglycemia regulates RUNX2 activation and cellular wound healing through the aldose reductase polyol pathway. *J Biol Chem* **284**(27): 17947–55
Dungan KM, Braithwaite SS, Preiser JC (2009) Stress hyperglycaemia. *Lancet* **373**(9677): 1798–807
Electronic Medicines Compendium (eMC) (2012) Summary of Product Characteristics. www.medicines.org.uk/EMC/ (accessed 3 May 2012)
Ibrahim R (2010) Diabetes mellitus type II: review of oral treatment options. *Int J Pharm Pharmaceut Sci* **2** (Suppl 1): 22–30
Joint Formulary Committee (2011) *British National Formulary 62.* Pharmaceutical Press, London
Kerr M (2011) Inpatient care for people with diabetes: the economic case for change. www.diabetes.nhs.uk/document.php?o=3034 (accessed 22 November 2011)
Lleva RR, Inzucchi SE (2011) Hospital management of hyperglycemia. *Curr Opin Endocrinol Diabetes Obes* **18**(2): 110–18
Malmberg K, Norhammar A, Wedel H, Ryden L (1999) Glycometabolic state at admission: important risk marker of mortality in conventionally treated patients with diabetes mellitus and acute myocardial infarction: long-term results from the Diabetes and Insulin-Glucose Infusion in Acute Myocardial Infarction (DIGAMI) study. *Circulation* **99**: 2626–32
Mesotten D, Van den Berghe G (2012) Glycemic targets and approaches to management of the patient with critical illness. *Curr Diab Rep* **12**: 101–7
Nathan DM, Buse JB, Davidson MB et al (2009) Medical management of hyperglycemia in type 2 diabetes: a consensus algorithm for the initiation and adjustment of therapy: a consensus statement of the American Diabetes Association and the European Association for the Study of Diabetes. *Diab Care* **32**: 193–203
National Institute for Health and Clinical Excellence (2011) Hyperglycaemia in acute coronary syndromes. Clinical Guideline 130. http://guidance.nice.org.uk/CG130 (accessed 4 December 2011)
NHS Diabetes (2010) Summary findings from the National Diabetes Inpatient Audit (NaDIA) 2010 (England). www.ic.nhs.uk/services/national-clinical-audit-support-programme-ncasp/audit-reports/diabetes (accessed 15 July 2011)
NICE-SUGAR Study Investigators (2009) Intensive versus conventional glucose control in critically ill patients. *N Engl J Med* **360**(13): 1283–97
Perera NJ, Harding AJ, Constantino MI et al (2011) Triple-B (basal-bolus-booster) subcutaneous insulin regimen: a pragmatic approach to managing hospital inpatient hyperglycaemia. *Pract Diabetes* **28**(6): 266–9
Trousdale RK, Jacobs S, Simhaee DA, Wu JK, Lustbader JW (2009) Wound closure and metabolic parameter variability in a db/db mouse model for diabetic ulcers. *J Surg Res* **151**(1): 100–7
Umpierrez GE, Smiley D, Zisman A et al (2007) Randomized study of basal-bolus insulin therapy in the inpatient management of patients with type 2 diabetes (RABBIT 2 trial). *Diabetes Care* **30**(9): 2181–6
van Raalte DH, Ouwens DM, Diamant M (2009) Novel insights into glucocorticoid-mediated diabetogenic effects: towards expansion of therapeutic options? *Eur J Clin Invest* **39**(2): 81–93
Wesorick D, O'Malley C, Rushakoff R, Larsen K, Magee M (2008) Management of diabetes and hyperglycemia in the hospital: a practical guide to subcutaneous insulin use in the non-critically ill, adult patient. *J Hosp Med* **3**(5 Suppl): 17–28
Wiener RS, Wiener DC, Larson RJ (2008) Benefits and risks of tight glucose control in critically ill adults: a meta-analysis. *JAMA* **300**(8): 933–44
Zerr KJ, Furnary AP, Grunkemeier GL, Bookin S, Kanhere V, Starr A (1997) Glucose control lowers the risk of wound infection in diabetics after open heart operations. *Ann Thorac Surg* **63**: 356–61

KEY POINTS

- Hyperglycaemia is common in people with diabetes admitted to hospital, and can have a significant impact on prognosis.
- Various mechanisms are responsible for hyperglycaemia, such as stress-induced insulin resistance and medication such as glucocorticoids.
- Patients with hyperglycaemia require a thorough assessment to decide how to treat the high blood sugars.
- Modern management of hyperglycaemia in hospitals involves the use of subcutaneous basal bolus regimens rather than intravenous insulin sliding scales.