Australian Diabetes Society

Guidelines for Routine Glucose Control in Hospital

2012

Contents

Introduction

Diabetes is estimated to affect 7.4% of the Australian population[1], and is increasing annually by 0.8%[2]. People with diabetes have a higher utilisation of both primary and tertiary health services. In 2004-05, 9% of all hospital admissions were recorded as having diabetes[3]. This is likely to be an underestimate as clinical audits from Australia and overseas have found hospital rates of diabetes of 11-25%[4-9] and furthermore, many cases are undiagnosed at the time[10]. Australian data indicate that the proportion of people with diabetes as a diagnosis in hospital has been increasing, with a 35% increase in numbers between 2000-01 and 2004-05[3]. They also have longer lengths of hospital stay, being about 2 days longer than people without diabetes[3,9]. The Australian Institute of Health and Welfare has estimated the cost of diabetes to hospital services in 2004-05 was $371M[3].

Diabetes and hyperglycaemia has been shown in a number of observational studies to be associated with poorer outcomes and are markers of morbidity and mortality. Reasons for the increased morbidity and mortality may be related to poor immune response, delayed healing, inflammation and thrombosis associated with hyperglycaemia as well as a higher rate of co-morbid conditions in this patient group[11].

Independent of diabetes, hyperglycaemia per se is also associated with worse hospital outcomes. This is the case whether the person has diabetes or not, but the relationship is stronger for people who do not have diabetes. The relationship between hyperglycaemia and adverse hospital outcomes, in particular mortality, has been clearly demonstrated in many different hospital settings, including myocardial infarction, stroke, general medical and surgical wards, trauma, cardiothoracic surgery, TPN, intensive care, and emergency admissions. For hyperglycaemic people who are not known to have diabetes, it is unclear if the higher mortality is due to the hyperglycaemia, or if the hyperglycaemia is but a marker of underlying critical illness. Most of the high quality studies demonstrating benefit of tight glycaemic control have come from critical care situations, and even these have produced conflicting results.

For patients with hyperglycaemia that is newly discovered in hospital, there is a high probability of undiagnosed diabetes, or future diabetes. However, at present follow-up is often haphazard, and the opportunity for early diagnosis and treatment of diabetes and thereby prevention of acute and long-term complications may be missed.

The aim of this document is to provide guidance for the management of hyperglycaemia in a range of hospital situations. The ADS has focused on the management of hyperglycaemia in patients with myocardial infarction and stroke, on general hospital wards, receiving enteral and parenteral nutrition, with steroid-induced or exacerbated hyperglycaemia, and in end of life situations. The optimal means of achieving tight control, the role of the specialist inpatient diabetes team, inpatient management of insulin pump therapy, and general measures for diabetes management have also been examined. We also provide guidance for the follow-up of patients with newly discovered hyperglycaemia. The recommendations were based on evidence obtained from systematic reviews where trials had been performed; otherwise they were made by consensus.

It is not the intention of these guidelines to deal with screening for diabetes, the management of diabetic emergencies such as diabetic ketoacidosis, hyperglycaemic hyperosmolar state, and hypoglycaemia, nor do they cover paediatrics, obstetrics or intensive care. Otherwise they should provide guidance for the management of patients with hyperglycaemia in the majority of hospital wards, and are complementary to the Australian Diabetes Society Perioperative Diabetes Management Guidelines.

We sought to achieve concordance in our recommendation to a single target glucose level for the majority of clinical situations, although there are some differences in the limited data for different scenarios. The overall recommendation is that for most hospital patients with hyperglycaemia, treatment should be instituted to achieve and maintain blood glucose (BG) levels below 10 mmol/L, but because of the potential dangers of hypoglycaemia, treatment should not aim to lower glucose levels below 5 mmol/L.

Section 1: Methodology and Process

Systematic reviews were conducted to provide the best possible evidence base for the recommendations. PICO searches of the Cochrane Database for Systematic Reviews, and Pubmed Clinical Queries were undertaken. Systematic reviews, meta-analyses and existing guidelines relating to our questions were reviewed by a member of the Writing Group, and summarised (Appendix 1). Key cited articles were also reviewed. Where systematic reviews were not available, general searches of the literature were undertaken. The evidence was discussed in an ADS workshop comprising an expert panel of Endocrinologists and Diabetes Educators, held in July 2011. At this workshop, recommendations for each section of the guidelines, and overall recommendations were agreed upon. Where there was little or no evidence, then the committee relied on experience, judgment and consensus to make their recommendations. Issues arising from the discussion, for which there is no evidence base, are included as practice points. The Writing Group drafted this document, which was circulated for further feedback from the participants of the Workshop, and others who were unable to attend.

Section 2: What Glucose Target Should be Aimed for in Acute Myocardial Infarction?

Hyperglycaemia and Cardiac Mortality

Hyperglycaemia is common with myocardial infarction. Data from numerous observational studies show a clear and consistent association between the initial admission glucose level and infarct outcomes, in particular mortality. A meta-analysis by Capes et al[12] showed that amongst patients without diabetes, those with an admission blood glucose level (BGL) ≥6.1-8.0 mmol/L had a 3.9 fold (95%CI 2.9-5.4) higher risk of death than those with lower BGL. For patients with diabetes, those with a BGL ≥10-11.0 mmol/L had a 1.7 fold (95%CI 1.2-2.4) increased risk of death. The majority of studies in this publication were performed in the pre-thrombolytic era, but newer publications show similar results (Appendix 2, Table 2.1). Virtually all have shown a dose relationship and a glucose threshold for increased mortality of around 6-8 mmol/L. In addition, there are observational data demonstrating a relationship between glucose levels in the first 24 hours after myocardial infarction and mortality (Appendix 2, table 2.2). These indicate that persistent hyperglycaemia, even if mild, is also associated with increased mortality following myocardial infarction.

Hypoglycaemia

Most studies have concentrated on the relationship between hyperglycaemia and increased mortality. There are also some data that hypoglycaemia is associated with adverse outcomes, with a U-shaped relationship being described in several observational studies[15,23,25]. The increased risk was seen in patients with admission BGLs ranging from <3.3 to <7 mmol/L. In the DIGAMI Study where there was active lowering of glucose, there was no increase in mortality or other major outcomes amongst subjects who developed hypoglycaemia <3 mmol/L, after adjustment for confounding variables[31].

Clinical Trial Data and Existing Recommendations

Five systematic reviews with specific analysis (in some cases subanalysis) of whether tight glucose control in myocardial infarction improves survival were identified[32-36]. One older systematic review which predated a number of the more recent trials with negative results, found a reduction in mortality with tight glucose control[32]. A more recent review suggested that tight glycaemic control can reduce mortality but did not make this conclusion on the basis of a meta-analysis[35], whilst another one decided that the evidence is inconclusive[34]. Two recent high quality systematic reviews concluded that tight glycaemic control did not reduce mortality[33,36], but one included cardiac conditions other than myocardial infarction.

Four of the randomised controlled studies identified in the systematic reviews had set specific glucose targets for their intervention (Appendix 2, Table 2.4)[28,31,44,48]. There was improvement in survival in the intensive treatment arm only in the oldest of these studies, where the glucose target was 7-10 mmol/L[31]. It has been postulated that the failure to demonstrate an effect in the more recent studies may be due to i) failure to

achieve a large enough differential in glucose levels between the arms of the study, ii) glucose levels in the control arm being only minimally elevated, iii) the advent of modern treatments for AMI (PTCA, thrombolysis, anti-platelet therapy, beta-blockade, statin therapy), overwhelming any benefit of glucose control[53].

Existing guidelines covering glucose control in myocardial infarction have given diverse recommendations (Appendix 2, Table 2.5)[54-57]. Two of the 4 guidelines did not have specific recommendations for myocardial infarction, but encompassed myocardial infarction within broader guidelines for hospital glucose control[55,57]. Two of the guidelines recommended target BGs <10 mmol/L[55,56], one recommended "normal" levels[54], and one recommended against tight control[57].

Conclusions

Observational data indicate a clear association between hyperglycaemia and mortality in myocardial infarction. However, only one RCT of patients with myocardial infarction has shown a benefit of glycaemic control, with a glucose target of 7-10 mmol/L. In the other studies, no mortality benefit of tight control was seen. Despite this, most professional organizations have recommended a glucose target of <10 mmol/L, provided that this can be achieved safely.

Recommendations and Practice Points

1. Patients admitted to hospital with myocardial infarction who have hyperglycaemia, should be treated to achieve and maintain glucose levels less than 10 mmol/L.
2. Hypoglycaemia must be avoided. It would be prudent to avoid treatment which lowers the glucose below 5 mmol/L.
3. Insulin infusion therapy may allow for tighter targets but this requires frequent monitoring and high level staff training.

Section 3: What Glucose Target Should be Aimed for in Acute Stroke

Hyperglycaemia and Stroke Mortality

Data from numerous observational studies show an association between initial glucose levels and outcomes of stroke, in particular mortality. Another meta-analysis by Capes et al showed that amongst patients without diabetes, those with an admission BGL ≥6.1-8.0 mmol/L had a 3.07 fold (95%CI 2.50-3.79) higher risk of death than those with lower BGL[58]. There was no increase in risk amongst patients with diabetes at these levels (RR 1.3, 95%CI 0.49-3.43) increased risk of death. Mortality from haemorrhagic stroke mortality was not associated with admission hyperglycaemia. More recent publications show similar results (Appendix 3, Table 3.1). Observational data also indicate that there is a relationship between glucose levels during the first 24 hours after stroke and mortality or infarct size (Appendix 3, Table 3.2).

Clinical Trial Data and Existing Recommendations

The 3 systematic reviews examining studies of tight glucose control in stroke came to divergent conclusions (Appendix 3, Table 3.3)[36,75,76]. Although none of the studies reviewed demonstrated a benefit of glucose control, one review recommended insulin therapy if glucose levels exceed 10 mmol/L[75]. There were 7 randomised controlled trials of tight glycaemic control for stroke. One had a large sample size but was discontinued early due to slow recruitment and failed to demonstrate a benefit of glucose control[78]. Most of the other trials were more of a pilot nature (Appendix 3, Table 3.4). An additional recent Australian study where there was a glucose control target of 4-8 demonstrated a 16% reduction in mortality with the intervention arm[85]. However glucose control was only one of 3 factors in the intervention package (the others being management of swallowing and fever), and it is difficult to determine the contribution of glucose control to the outcome. This study had not been included in any of the above systematic reviews.
Two sets of stroke guidelines which provided some recommendations regarding glucose control were identified (Appendix 3, Table 3.4). Both suggested aiming to keep BGs below a level around 10 mmol/L, but admit that the evidence for this is weak.

Conclusions

Observational data indicate a clear association between hyperglycaemia and mortality in acute thrombotic stroke. There is a lack of clinical trial evidence regarding appropriate glucose targets in stroke, and the recommendation is made on the basis of extrapolation from other clinical situations, and consensus.

Recommendations and Practice Points

1. Patients admitted to hospital with acute thrombotic stroke who have hyperglycaemia, should be treated to achieve and maintain glucose levels less than 10 mmol/L.
2. Hypoglycaemia must be avoided, and therefore it would be prudent to avoid treatment which lowers the glucose below 5 mmol/L.

Section 4: What are Appropriate Glucose Targets for Patients in General Hospital Wards?

Hyperglycaemia and Complications in General Hospital Wards

A number of observational studies have demonstrated an association between glucose levels and adverse outcomes in patients in general hospital wards. These have shown a higher risk of adverse outcomes above a random glucose level of 8-12.2 mmol/L (Appendix 4, Table 4.1). The adverse outcomes include infection, mortality, and longer length of stay. There is also a dose relationship between glucose levels and mortality[91-93]. The relationship between hyperglycaemia and mortality in the general wards is much stronger among those with newly discovered hyperglycaemia than among those with known diabetes.

Systematic Reviews and Existing Guidelines

Three systematic reviews have examined clinical trials of tight glycaemic control outside of the intensive care situation, and not specifically focusing on myocardial infarction or stroke (Appendix 4, Table 4.2). Most studies included in these reviews were in the perioperative context, or included subjects with myocardial infarction. The findings have been mixed, with one review finding a reduction in mortality with tight glycaemic control with cardiac surgery[94], one finding no benefit in the non-ICU or peri-operative settings[36], and a third finding a reduction in infection rate only[95]. There is a recent study in general surgical patients which found that treating to a pre-meal glucose target of <7.8 mmol/L with basal, bolus and supplemental insulin resulted in better glycaemic control and fewer wound infections and total complications than using sliding scale insulin with the same target[104]. However this study was designed to compare the 2 insulin regimes, rather than the effect of treating to their target. No trials have as their primary objective, examined the effect of treating to specific glucose targets in general medical wards.

Three existing guidelines for glucose control in non-critically ill hospital patients have recommended glucose levels below 10 mmol/L (Appendix 4, Table 4.3)[55,105,107]. A fourth guideline maintains that there is no evidence for strict control in non-ICU patients[106]. The American Association of Clinical Endocrinologists / American Diabetes Association and Endocrine Society of America guidelines also recommend pre-meal glucose levels of 3.9-7.8 mmol/L, without giving the rationale for different pre-meal and random glucose targets[55,107]. The caveat that these should only be the targets if they can be safely achieved has also been stated.

Conclusions

As the evidence is limited, our recommendations are based on existing guidelines and extrapolations from other clinical situations. Having the same glucose targets as for myocardial infarction and stroke was considered important for uniformity across the hospital, and to avoid confusion. Although one would not regard glucose levels as being in the hypoglycaemic range until they are below 4 mmol/L, active intervention

should not aim to reduce the glucose levels below 5 mmol/L, which allows for an added margin of safety. If aiming for tight glycaemic control, frequent glucose testing is required.

Recommendations and Practice Points

1. Most patients in general hospital wards with hyperglycaemia should be treated to achieve and maintain glucose levels less than 10 mmol/L.
2. Hypoglycaemia must be avoided. It would be prudent to avoid treatment which lowers the glucose below 5 mmol/L.
3. To achieve tight glucose control safely, frequent glucose monitoring is recommended

Section 5: What Special Measures Need to be Undertaken for People on Enteral or Parenteral Nutrition?

Hyperglycaemia and Enteral and Parenteral Feeding

Hyperglycaemia is a common occurrence in patients receiving nutritional support either in the form of enteral or parenteral nutrition. The specific effect of hyperglycaemia on clinical outcomes in patients receiving nutrition support has only been reported by one observational study. A retrospective study of 111 patients receiving total parenteral nutrition (TPN) found that increased blood glucose levels were associated with an increased risk of cardiac complications, infection, sepsis, acute renal failure and death[91]. Those receiving TPN with mean glucose levels >9.1 mmol/l had a 10-fold greater risk of mortality than those with mean glucose levels $\leq$6.9 mmol/l. This association was independent of age, sex and presence of pre-existing diabetes. This adds further weight to the overwhelming evidence of a clear relationship between high blood glucose levels and adverse outcomes in critically ill or hospitalised patients, as reviewed in the earlier sections of this guideline.

A major goal in the management of patients with diabetes receiving nutritional support is the achievement of good glycaemic control, avoiding both hyperglycaemia and hypoglycaemia, with their associated risks of fluid imbalance and dehydration, ketoacidosis and hyperosmolar coma, infection and neurological events. However, how best to achieve good glycaemic control in these patients remains unclear. A critical factor for consideration is where the patient will be cared for: in the ICU or general ward. Other important considerations include the method of nutritional therapy (enteral vs parenteral) and composition of the feeds particularly carbohydrate/dextrose content. In general, diabetic enteral formulas (low carbohydrate high monounsaturated fatty acid formulas) are preferable to standard high carbohydrate formulas in patients with diabetes[107]. Close monitoring of BGLs and review of diabetes management is essential when enteral/parenteral feeds cease and oral intake resumes.

Clinical Trials

No studies investigating the effects of oral glucose lowering agents on blood glucose levels and outcomes in patients receiving enteral or parenteral nutrition were identified. There are 2 studies, both of poor quality and at high risk of bias, which have investigated the effects of different insulin regimens in patients receiving enteral nutrition (Appendix 5, Table 5.1), but none in the situation of parenteral nutrition. One compared the effects of sliding scale insulin to sliding scale insulin and regular subcutaneous glargine insulin, showing no differences in blood glucose levels, adverse outcomes or length of stay[108]. However, a significant proportion of the patients in the sliding scale alone group also received NPH insulin during follow up. This suggests that a basal insulin on top of a correctional insulin regimen, has a role in achieving adequate glycaemic control in patients receiving enteral nutrition. A second (nonrandomized) pilot study with a retrospective control group found that a basal bolus insulin protocol achieved lower mean glucose levels than a variable dose preprandial

insulin regime, at the expense of a small increase in hypoglycaemia[109]. The nurse led insulin protocol was implemented in the ICU setting which limits its generalisability.

Conclusions

On the balance of the limited evidence, insulin therapy is likely to be the most effective agent for immediate control of blood glucose levels in patients receiving enteral and parenteral nutritional support. The recommendations made are based on experience and consensus.

Recommendations and Practice Points

1. Individualised nutritional plans should be provided as insulin therapy will depend on the nature of the feeding cycle.

2. Sliding scale insulin should not be used alone to optimize glucose control in patients receiving enteral or parenteral nutrition.

3. Insulin therapy should include regular basal insulin (intermediate or long acting insulin) with prandial and correctional insulin if required.

4. Perform BG testing 4-6 hourly. With bolus enteral or parenteral nutrition perform BG testing before each bolus is given.

6. Patients with unstable metabolic control or variable parenteral feeding may benefit from an intravenous insulin infusion therapy.

7. Close liaison with the dietitian or team managing the enteral or parenteral nutrition is critical particularly if calorie intake is changing, as insulin doses will need to be adjusted.

Section 6: How is Steroid-Induced Hyperglycaemia Best Managed?

Prevalence and risk factors

Hyperglycaemia is common amongst inpatients who are receiving glucocorticoids (GC), with reported incidences of 64-71%[110,111]. Risk factors for development of hyperglycaemia amongst inpatients include a pre-existing diagnosis of diabetes[110,112], higher HbA1c[113], increasing age[111], steroid dose[114], and family history of diabetes[115, 116].

There is little data on temporal BG profile of individuals receiving GC. An open prospective observational trial performed on acute hospital wards examined the interstitial glucose profiles of pts admitted with COPD treated with at least prednisone 20mg/day as compared to pts with COPD, not known to have diabetes, admitted for another indication who did not receive GC[117]. Patients receiving GC in the morning had higher BGLs in the afternoon and evening, as compared to those not receiving GCs (with the greatest elevation seen in those with known diabetes). A rise in fasting glucose is also seen when extremely high dose GC (e.g. methylprednisone 250-1000mg/day) are administered[113]. Based on ambulatory data, the effect of GC on BG profile is rapid, with a change seen within 2-3 hours of administration of GC[118,119]. This is also rapidly reversible, in that lower glucose levels are seen on GC free days in patients who receive alternate day GC[120].

Screening for development of hyperglycaemia and monitoring in those with DM

Prior to or upon the initiation of GC, it is prudent to exclude the presence of undiagnosed diabetes through measurement of serum glucose (see section 11). Screening for development of steroid-induced hyperglycaemia by afternoon fingerprick BG assessment is likely to detect the development of most cases of hyperglycaemia[112], and twice daily GC induced hyperglycaemia should still be detected. Reliance on fasting glucose is inadequate. If hyperglycaemia is detected, BG monitoring should occur as per the general diabetes protocol.

Management of glucocorticoid induced hyperglycaemia

There are no prospective trials on the use of any anti-diabetic medication for the management of GC related hyperglycaemia. The limited observational data are outlined in Appendix 6, Table 6.2. Sulphonylureas have a limited role in the treatment of steroid-induced hyperglycaemia in hospital. There are reports of thiazolidinedione use in the setting of organ transplantation, but these agents are also unsuitable for most patients in hospital. The management of new onset diabetes after transplantation has been addressed in other guidelines[140] and will not be further discussed in this document.

Although there are no trials of its use in steroid-induced hyperglycaemia, insulin is considered to be the agent of choice for the management of steroid-induced hyperglycaemia in hospital. Benefits provided by insulin include greater dose flexibility, more rapid onset of action and titration and that there is usually no dose ceiling as compared to other glucose lowering agents. Insulin dose requirements will always need to be individualised, and require pre-emptive titration as the GC dose is adjusted, usually on a daily basis. The insulin

regimen should predominantly target post-prandial control, and with morning GC administration, the afternoon hyperglycaemia. The use of isophane insulin for management of steroid-induced hyperglycaemia has been advocated, with the initial dose determined according to GC dose and patient weight[124,139]. Isophane type insulin can be supplemented with ultraquick insulin analogue with meals[139]. With twice, thrice or 4 times a day GC regimens, isophane insulin twice daily with prandial rapid acting analogue can be initiated. A regime that controlled glycaemia on previous occasions can be re-initiated, e.g. when cyclical GCs are required, as long as there has been no major interval change in weight or renal function. For those with pre-existing insulin requiring diabetes, a pre-emptive increase in insulin will be required, and further adjustment based on blood glucose response.

Recommendations and Practice Points

1. In patients receiving glucocorticoids, undiagnosed diabetes should be excluded. Those free of diabetes should be screened for the development of hyperglycaemia by random blood glucose monitoring performed in the afternoon following morning administration of GC.
2. Hyperglycaemia is best managed with insulin: basal insulin as isophane type insulin, and rapid acting analogue with meals as required.
3. In individuals already on insulin the likely need for increased insulin should be recognised. Dose requirements need to be individualised and require daily review.

Section 7: What is the Optimal Means of Achieving and Maintaining Glycaemic Control in Hospitalised Patients who are not Critically Ill?

This section examines the optimal methods for achieving and maintaining good routine glycaemic control in hospital. It does not discuss the use of insulin infusion therapy, or perioperative management. For the latter, we refer the reader to the Australian Diabetes Society Perioperative Diabetes Management Guidelines[141].

There is a paucity of data in the non-critically ill patient group as to the best method of maintaining glycaemic control. This group of patients differs greatly from those critically ill as they are often eating. Intensive insulin therapy has been shown to be beneficial in a critically ill patient population, but there have been no studies evaluating outcomes in general medical wards. The main adverse event with intensive subcutaneous insulin therapy is hypoglycaemia which can be quite severe.

Intensive insulin therapy requires frequent monitoring and should not just be reactive to changes in glucose loads, e.g. food. Its application requires a specific skill set for staff to maintain. Traditionally sliding scales have been used to maintain blood glucose levels in non-critical hospitalized patients. This method of injecting a set dose of insulin at set times is often reactive to high levels of blood glucose. BGs often fluctuate from high to low, which can potentially be detrimental. Sliding scale administration of insulin is not recommended, and American guidelines recommend that an insulin regimen with basal, nutritional and supplemental (correction) components be utilized for hospitalised patients with diabetes or stress hyperglycaemia[142].

There are few studies that have examined different subcutaneous insulin regimens in non-critical hospitalised patients (Appendix 7). Most studies have moderate to high risk of bias and outcome measures have been inconsistent between the different studies. Basal bolus regimens have been shown to be superior to sliding scale regimens for glucose control[102,104], and sliding scale insulin alone has been no more effective than continuation of the patient's usual diabetes medication[101]. Effective use of basal bolus insulin requires frequent and regular blood glucose monitoring (at least 4 and preferably 6-8 times daily). Based on clinical expertise, current practices and the limited literature, the following consensus recommendations were made.

Recommendations and Practice Points

1. Sliding scale insulin should not be used to optimise glucose control in the inpatient general medical or surgical setting.
2. Oral hypoglycaemic agents or pre-mixed insulin can be used in certain stable hospitalised patients who are eating regularly. Supplemental insulin should be written up in addition.
3. Insulin therapy in hospitalised patients should otherwise consist of a basal insulin, prandial and supplemental insulin.

Section 8: How Should Patients on Insulin Pump Therapy be Managed in Hospital?

Continuous Subcutaneous Insulin Infusion Therapy in Hospital

Continuous subcutaneous insulin infusion (CSII) or insulin pump therapy is used in the management of growing numbers of patients with Type 1 diabetes in Australia. Anecdotal reports suggest that patients established on CSII usually prefer to continue on their pumps during hospital admissions. Hospital health care providers will increasingly be faced with the issue of how to manage such inpatients. A number of publications have detailed guidelines regarding inpatient management of patients previously established on CSII[144-147]. Whilst there are no data from randomised trials available, observational reports indicate that patients admitted to hospital continued on CSII who are managed with best-practice consensus protocols fare at least as well as those changed over to subcutaneous insulin injections and managed by the endocrinology team. The data regarding hypoglycaemia is conflicting with one study indicating a lower incidence in those inpatients continued on CSII which was not confirmed with a subsequent study[148,149]. A caveat is that these reports have stemmed from tertiary academic medical centres in the United States and their applicability to a spectrum of hospitals (including community hospitals) in Australia is yet to be determined. The recommendations below are based upon a consensus opinion.

Management of CSII in Hospital

General recommendations for CSII therapy in hospital are outlined in Appendix 8, Table 8.1. In appropriate circumstances, CSII may be the preferred method of insulin delivery. However, device operating menus and programs vary not only according to the manufacturer but also from model to model. It is highly unlikely that non-specialised medical and nursing staff will be familiar with the operation of all available devices. We therefore recommend that CSII therapy is be continued in hospital only in those situations where the patient (or guardian) has the ability to safely self-manage their insulin dosing and the pump. The competency requirements are outlined in Appendix 8, Table 8.2. If these criteria are not met CSII must be substituted with either a subcutaneous insulin regimen or an intravenous insulin infusion. Contraindications to CSII therapy are listed in Appendix 8, Table 8.3. All aspects of CSII management should be documented (Appendix 8, Table 8.4) and it is recommended that the Endocrine team be involved.

CSII and Surgery

Surgery itself is not an absolute contraindication to continuation of CSII. If CSII is to be continued intra-operatively this decision must be made in conjunction with the anaesthetist, surgeon/proceduralist, and endocrinology team with the documented consent of the patient or their guardian. CSII and an intravenous insulin infusion should not be used simultaneously for any extended period[150]. The situations appropriate for intraoperative CSII or for its substitution with an intravenous insulin infusion are outlined in Appendix 8, Table 8.5. When CSII is being used intraoperatively, it is important for there is a protocol for its management (Appendix 8, Table 8.6.). Appropriate overlap and timing is important when switching a patient from CSII to insulin infusion or multiple subcutaneous insulin injections, and vice versa (Table 8.6.).

Recommendations and Practice Points

1. In general, CSII should be continued in hospital where the patient can competently and safely self-manage the pump and self-dosing.
2. Details of pump therapy should be documented, and supported by the endocrine team
3. CSII may be continued for short operative procedures if those responsible for the patient's intraoperative care are comfortable with its use.

Section 9: What is Appropriate Glucose Control in End of Life Situations?

Diabetes and End of Life

For patients with diabetes and advanced disease limiting their life expectancy there is no body of evidence available regarding the impact of tight glycaemic control on outcomes. Life-limiting disease includes, but is not limited to, cancer and includes any disease process such as advanced dementia, end stage cardiac and respiratory failure, which is incurable and significantly shortens the patient's life expectancy. As the patient with diabetes approaches the end of their life the guidelines regarding glucose monitoring and glycaemic targets detailed earlier in this document may no longer be appropriate with a potential for discomfort, inconvenience and significant morbidity relating to hypoglycaemia. Tight glycaemic control is questionable benefit and the avoidance of long-term complications is no longer relevant. Conversely it is important to maintain a level of glycaemia to prevent hyperglycaemia associated thirst, dehydration, polyuria associated urinary frequency, altered conscious state and symptomatic hypoglycaemia. Treatment regimens need to be individualised according to the patient's circumstances.

Palliative care is defined as medical or comfort care that reduces the severity of a disease or slows its progress rather than providing a cure. Currow et al[151] have described 4 phases in the end of life pathway: Stable, unstable, deteriorating, and terminal (see Appendix 9, Table 9.1 for details). Palliative patients may be admitted to hospital for management of an acute illness, either intercurrent or related to their primary underlying disorder or for terminal care. There is an absence of level I data though there are a number of valuable consensus based guidelines addressing the global management of palliative patients with diabetes[151-153]. The following represents a consensus of opinion in the absence of a suitable evidence base, and is in part based on the 2010 Guidelines for Managing Diabetes at the End of Life[152]. This consensus document focuses on the inpatient management of hyperglycaemia in those patients with diabetes deemed as requiring palliative care. As management should be individualised to each patient's needs this document provides general principles for the inpatient management of palliative care patients with diabetes and detailed protocols cannot be provided.

Glucose Management in End of Life Situations

Glucose management during inpatient admissions will depend on the type of diabetes and the phase of the end of life pathway (see Appendix 9, Table 9.2 for details). In general, in the earlier stages of end of life, the person's usual diabetes medication would be continued, with adjustments based on the many situational factors which would affect glycaemic stability (Appendix 9, Table 9.3). The decision to simplify and rationalise treatment regimes and targets would need to be made on an individual basis. As the person progresses through the phases of end of life, the emphasis shifts towards maintenance of comfort, with corresponding reductions in medication and glucose testing, and some liberalisation of food restriction. This does not imply a nihilistic approach in the metabolic management of palliative patients. Avoidance of marked hyperglycaemia is still relevant, particularly in hospital, to avoid symptoms of hyperglycaemia, and improve wound healing and

resistance to infection. Hypoglycaemia must also be avoided. With type 1 diabetes, ketoacidosis is likely to precipitate death, so it should be prevented until a decision is made to withdraw all treatment in the terminal phase. Therefore until then, some glucose testing and insulin administration may remain necessary. It is reasonable to continue on insulin pump therapy in those patients established on these devices.

The views of the patient and their family need to be determined. They may require advice and counseling regarding the management of the patient's glucose levels as many years may have been spent where glucose levels have been diligently maintained in a target range. The realisation that long-term survival is no longer a viable proposition and that maintenance of tight glycaemic control is of dubious value and could even adversely impact quality of life can be confronting. Ultimately the decision of the patient and their family will take precedence. The status of the patient may be evolving continuously requiring the ongoing reassessment of glycaemic management strategies by the medical team.

Recommendations and Practice Points

1. Palliative care patients may still benefit from a level of glucose control in hospital so diabetes treatment remains relevant.
2. The level of intervention would generally be less intensive than for other hospital patients, and needs to be individualised, depending on the phase of end of life, and other situational factors.

Section 10: At What Level is Hyperglycaemia in Hospital Predictive of Diabetes and How Should Patients with Newly Discovered Hyperglycaemia be Followed up?

Stress Hyperglycaemia

Patients with a known history of diabetes commonly have hyperglycaemia in hospital, but patients without a history of diabetes may also be found to have elevated blood glucose levels. Hyperglycaemia in patients not known to have diabetes may be secondary to stress or to undiagnosed diabetes. It is often difficult to distinguish the cause of hyperglycaemia in a short hospital stay.

Stress hyperglycaemia most commonly occurs in patients with acute or critical illness and is more likely to occur in a more critically ill patient. Hyperglycaemia is postulated to be mediated through cytokines, the sympathetic nervous system and the hypothalamic-pituitary-adrenal axis[155]. It is not clear whether patients who manifest stress hyperglycaemia have an underlying impairment in their glucose metabolism, but in the long term, inpatient hyperglycaemia may herald undiagnosed diabetes or the development of diabetes in the future. The prevalence of undiagnosed diabetes varies in different inpatient settings and can be up to 60% (Appendix 10, table 10.1). It is important to diagnose patients with diabetes early to ensure appropriate management, both lifestyle and medication to prevent the development of long term complications.

There is limited literature to guide the level of hyperglycaemia predictive of diabetes or to suggest an appropriate algorithm for detection of diabetes in the acute hospital setting. The American Association of Clinical Endocrinologists / American Diabetes Association consensus recommendations defines a BSL >7.8mmol/L as inpatient hyperglycaemia and suggest an HbA1c may assist in diagnosis of diabetes. HbA1c >6.5% (48 mmol/mol) is strongly suggestive of underlying diabetes[55,160]. However, there is considerable heterogeneity amongst studies looking at predictors of diabetes in inpatient populations (Appendix 10, table 10.1). Different glucose values have been used to define hyperglycaemia. HbA1c levels used to define a diagnosis of diabetes and the populations studied have also been quite variable. Whilst HbA1c has not been ratified for the general diagnosis of diabetes in Australia, there is no doubt that for a patient with hyperglycaemia, it is a strong indicator of underlying diabetes.

Whilst in hospital, patients with newly diagnosed diabetes should be referred to the Specialist Diabetes Inpatient Team (section 12) or the Endocrine Team for management. Irrespective of whether diabetes is definitively diagnosed in hospital, patients with inpatient hyperglycaemia should receive follow-up to ensure that the diagnosis is clarified, and appropriate counseling and management instituted. Notification of the general practitioner is vital to this process.

A suggested algorithm for the approach for the diagnosis and follow-up of an inpatient with newly discovered hyperglycaemia is given in Appendix 11, Figure 11.1.

Recommendations and Practice Points

1. All inpatients with newly discovered hyperglycaemia (random plasma glucose >7.8mmol/L) should have an HbA1c performed.
2. All inpatients who are newly diagnosed with diabetes should be managed appropriately for diabetes. If there is diabetes expertise available, an early referral should be made.
3. All patients with abnormal glucose metabolism detected in hospital should have adequate follow up arranged, and the findings should be communicated to the usual care practitioner.

Section 11: What is the Role of a Specialist Inpatient Diabetes Team?

Improving glycaemic control has been shown to reduce the risk of adverse outcomes associated with hyperglycaemia, but the evidence for these clinical benefits have been obtained in and limited to specific individual clinical units. Translating these improved outcomes to a whole hospital is more challenging and requires a different approach. Rather than focusing on improved clinical outcomes, or on specific blood glucose targets, hospital-wide approaches have focused on reducing the difference in length of stay for people with diabetes by improving overall diabetes management. The drivers for this approach are not so much an improvement in quality of care or clinical outcomes, but rather reductions in associated costs and improved bed utilisation. The factors contributing to increased length of stay and poorer outcomes associated with diabetes that are potentially modifiable include blood glucose control, inappropriate diabetes management and delayed involvement of specialist diabetes services.

Different approaches to this problem have been utilised, with varying levels of evidence to support the intervention. These vary from the traditional consultative service, to systematic hospital wide diabetes programmes, to the newer concept of the Specialist Diabetes Inpatient Management Team (Appendix 11, table 11.1). There has now been one randomised controlled trial[164] and a number of comparative studies which have demonstrated improved outcomes with the latter approach (Appendix 11, Table 11.2).

These teams usually comprise dedicated Diabetes Inpatient Specialist Nurses (DISN), usually led by a consultant in diabetes. The role of such teams has included improving diabetes management expertise throughout the hospital, the development and implementation of diabetes management protocols, direct management of diabetes with specific referral criteria, ward liaison, troubleshooting, management advice, and discharge planning (Appendix 11, Table 11.3). DISNs are currently involved in 30-50% of UK hospitals[171], with Diabetes UK recommending a ratio of one Diabetes DISN for every 300 beds[172]. The NHS (UK) has adopted this approach to improve diabetes inpatient management through the whole health system, resulting in reductions in adverse outcomes and length of hospital stay[9]. In Australia, the introduction of Specialist Diabetes Inpatient Management Teams will require additional resources, but the long-term economic argument is compelling. The literature suggests that hospitals which have introduced these teams have realised shorter lengths of stay and significant cost-savings[165,166,167,170]. Health administrators need to invest in such teams which should result in better inpatient diabetes care, shorter lengths of hospital stay, and cost-savings to the health system. Forward planning is also needed for the training of the specialised workforce required for Diabetes Inpatient Management Teams.

Recommendation

1. Hospitals should consider the introduction of Specialist Diabetes Inpatient Management Teams

Section 12: What Routine Measures Should be Undertaken for People with Diabetes Admitted to Hospital?

Effective inpatient diabetes management should be provided early and continuously throughout the hospital admission. To support optimal glycaemic control in hospital and diabetes management after discharge, it is important to have established routine processes and protocols for the care of people with diabetes in hospital. These recommendations are generally based on good general hospital practice, experience, and common sense. General recommendations include: clear identification of diabetes in the medical record, blood glucose monitoring, a hypoglycaemia management protocol, HbA1c testing, a multidisciplinary team approach, dietetic assessment, diabetes self-management education when appropriate, and discharge planning[142]. Insulin is a common source of medication error[171,172], and must be minimised by mechanisms such as staff education, pharmacist oversight, and dedicated insulin prescription charts[173].

Blood Glucose Monitoring

Where tight glycaemic control is desired, and particularly for patients on insulin, it is important for blood glucose monitoring to occur before and after meals. This is critical to facilitate appropriate adjustments to the patient's insulin dosage, and monitor for hypoglycaemia. Additional testing at bed-time and overnight is often also helpful. For stable patients, or those where tight glucose control is not an aim, testing can be reduced accordingly.

Discharge Planning and Diabetes Education

Whilst this document focuses on the management in hospital, it is important to take the opportunity to improve the post-discharge management of diabetes as well. Liaison with the general practitioner is an important component of this. Not only might this improve patient outcomes, but it may reduce the need for readmission to hospital. The various team members participating in inpatient management also have a role in promoting and facilitating better diabetes care post-discharge (Appendix 12, Table 12.1). Appropriate diabetes education is a critical component of inpatient patient care and discharge planning. A focus on the continuity of care where the patient is the central member in the management of diabetes is important, and their family members may need to be brought into the discussion.

Recommendations and Practice Points

1. Ensure clear processes and protocols are implemented in the hospital for routine diabetes care.
2. Ensure discharge planning which facilitates optimal long-term diabetes management.

Appendices

Appendix 1: Search Methodology of Systematic Reviews

Table 1.1. PICO search questions and search terms used for each of the chapters.

Question	Search Terms
What glucose target should be aimed for in acute myocardial infarction?	hyperglyc(a)emia, diabetes, intensive glucose control, tight glucose control, intensive glyc(a)emic control, tight glyc(a)emic control ,myocardial infarction, acute coronary syndrome, with the outcomes of mortality or death.
What glucose target should be aimed for in acute stroke?	hyperglyc(a)emia, diabetes, intensive glucose control, tight glucose control, intensive glyc(a)emic control, tight glyc(a)emic control ,myocardial infarction, stroke, cerebrovascular accident, with the outcomes of mortality or death.
What are appropriate glucose targets for patients in general hospital wards?	intensive glucose control, tight glucose control, intensive glyc(a)emic control, tight glyc(a)emic control, hospital, surgery, medicine
What special measures need to be undertaken for people on enteral + parenteral nutrition?	Diabetes and (enteral nutrition or parenteral nutrition)
How is steroid-induced hyperglycaemia best managed?	(Metformin or sulphonylurea or incretins or Dipeptidyl-Peptidase IV Inhibitors or thiazolidinediones or insulin) and (glucocorticoids or prednisone) and (hyperglycaemia or diabetes)
What is the optimal means of achieving routine glucose control in hospital?	hyperglyc(a)emia, diabetes, intensive glucose control, blood glucose/sugar control, intensive glyc(a)emic control, tight glyc(a)emic control, hospital, inpatient
How should patients on insulin pump therapy be managed in hospital?	diabetes, guidelines, hyperglyc(a)emia, hypoglyc(a)emia, hospital admission, acute care, inpatient care, perioperative management, CSII, insulin pump, insulin pump therapy, IPT
What is appropriate glucose control in end of life situations	diabetes, guidelines, hyperglyc(a)emia, hypoglyc(a)emia, hospital admission, inpatient care, end-of-life, palliative care, terminal illness, advanced cancer, hospice, insulin, oral hypoglyc(a)emic agents, sliding scale, blood glucose, therapy, and management
How should patients with newly discovered hyperglycaemia be followed up?	hyperglyc(a)emia, diabetes, intensive glucose control, blood glucose/sugar control, intensive glyc(a)emic control, tight glyc(a)emic control, hospital, inpatient
What is the role of a specialist diabetes inpatient team?	Diabetes, hospital, inpatient
What routine measures should be undertaken for people with diabetes admitted to hospital?	Consensus only

Appendix 2: Literature reviewed for "What Glucose Target Should be Aimed for in Acute Myocardial Infarction?"

Table 2.1. Recent studies examining the relationship between admission glucose levels and mortality following myocardial infarction

Study	Subjects	Characteristics	Elevated admission glucose predictive of mortality?	Threshold level for effect?	Comments	Methodology
Wong 2004[13]	158	STEMI	Yes	8 mmol/L	Similar relationship for both inpatient and 6 month mortality Relationship between BG and death present with and without reperfusion therapy	Clinical cohort study
Stranders 2004[14]	846	Any AMI	Yes	11.1 mmol/L for non-diabetics	Above 11.1 mmol/L, non-diabetics had same risk as those with diabetes.	Retrospective clinical cohort study
Timmer 2004[15]	356	STEMI with PTCA or reperfusion	Yes	7.8 mmol/L	Also association with larger infarct size and reduced LV function	Post-hoc subanalysis of a clinical trial cohort
Kosiborod 2005[16]	141680	Age >65	Yes	6.1 mmol/L for non-diabetes 13.3 mmol/L for diabetes	Similar results for 30 day and one year mortality	Analysis of database
Straumann 2005[17]	978	All had PTCA	Yes	7.8 mmol/L	Similar results for 30 day and longer-term mortality	Analysis of database
Meier 2005[18]	227	All AMI	Yes	7.4 mmol/L for non-diabetics, 7.9 mmol/L for diabetes	Survival >3.5 years was assessed	Clinical cohort study
Goyal 2006[19]	1469	Subanalysis of CARDINAL Trial	Yes (only for non-diabetics)		Lower mortality amongst non-diabetics where there was a greater drop in BG over 24 hrs	Post-hoc subanalysis of a clinical trial cohort
Bhadriraju 2006[20]	9020	Subanalysis of OPUS-TIMI trial	Yes	5.6 mmol/L	Relationship stronger for non-diabetics. Results also validated in subanalysis of TACTICS-TIMI trial	Post-hoc subanalysis of a clinical trial cohort
Naber 2009[21]	5866	Non-diabetic STEMI (ACOS Registry)	Yes	8.3 mmol/L	Inpatient and 1 year mortality	Cohort study
Sinnaeve	13526	Global registry	Yes	6.9 mmol/L	Random BGL associated with inpatient	Analysis of

2009[22]				(random) 5.6 mmol/L (fasting)	mortality only, fasting BGL associated with both inpatient and 6 month mortality.	database
Ishihara 2009[23]	3750	Within 48 hrs of AMI	Yes	7 mmol/L	U-shaped curve for patients with diabetes, increased mortality if BGL<7 or >11 mmol/L	Cohort study
Dziewiercz 2009[24]	763	Non-STEMI treated conservatively	Yes	5 mmol/L	Relationship stronger for non-diabetic subjects	Analysis of database
Goyal 2009[25]	30536	CREATE ECLA and OASIS-6 cohorts	Yes	7.8 mmol/L	Hypoglycaemia < 3.3 also predicted mortality	Post-hoc analysis of clinical trial cohorts
De Mulder 2010[26]	1185	Both preinvasive and PTCA eras	Yes	11 mmol/L	Each mmol/L increase corresponded to a 7% increased mortality (adjusted HR 1.07, 95% CI 1.04-1.10)	Cohort study
Timmer 2011[27]	4176	Non-diabetic STEMI	Yes	8.2 mmol/L	30 day and 1 year mortality assessed. U-shaped curve for mortality with increased mortality for those with BGL ≤6.9 mmol/L	Cohort study

Table 2.2. Observational data of a relationship between average glucose levels or glucose levels achieved in the first 24 hours after myocardial infarction and mortality.

Study	**Subjects**	**Characteristics**	**Glucose parameter**	**Elevated glucose predictive of mortality?**	**Threshold level for effect?**	**Comments**	**Methodology**
Cheung 2006[28], 2008[29]	240	Myocardial infarct with known diabetes or admission BG ≥7.8 mmol/L	1-2 hourly capillary BGs	Yes	8 mmol/L		Post-hoc subanalysis of a clinical trial cohort
Kosiborod 2008[30]	7820	All AMI	Mean glucose measurements	Yes	6.1 mmol/L	Mortality lower in insulin treated patients	Analysis of database

Table 2.3. Systematic reviews of randomized controlled trials of tight glucose control in myocardial infarction, where the primary outcome was death.

Review	Search Method	Selection Question	Studies	Subjects	Result/Conclusion	Comment
Pittas 2004[32]	Medline, Cochrane Controlled Clinical Trials Register	Studies of Insulin in critically ill hospitalized adult patients. Subanalysis: those aiming for glucose control	AMI subanalysis: 8 studies. **Davies 1991[37], Malmberg 1995[31], Scott 1999[38]**, Lazar 2000[39], Szabo 2001[40], van den Berghe 2001[41], Groban 2002[42], Smith 2002[43]	Subanalysis: 2772	Subanalysis: 29% reduction in mortality (RR 0.71, 95%CI 0.54-0.93)	Subanalysis included studies of coronary surgery and ICU patients
Zhao 2010[33]	Medline, CENTRAL, EMBASE	RCTs of GIK or insulin-glucose. Subanalysis: insulin-glucose only	AMI subanalyis: 3 studies. **Malmberg 1995[31], Malmberg 2005[44], Cheung 2006[28]**	Subanalysis: 2113	Subanalysis: No reduction in mortality (RR 1.07, 95%CI 0.85-1.36)	
Devine 2010[34]	Medline and Embase	Randomised trials of ACS with hyperglycaemia comparing insulin infusion or GIK with active controls which assessed mortality and morbidity	**Malmberg 1995[31],** Diaz 1998[45], Ceremuzynski 1999[46], **Malmberg 2005[44]**, CREATE-ECLA 2005[47], **Cheung 2006[28]**		Current evidence that insulin therapy reduces mortality and morbidity in ACS is inconclusive	Meta-analysis not done
Lipton 2011[35]	Pubmed	Trials with insulin in patients with unstable angina or AMI. Subanalysis: those aiming for glucose normalization	AMI subanalysis: 3 studies. **Malmberg 1995[31], Malmberg 2005[44], Cheung 2006[28]**		Intensive glucose lowering insulin therapy can reduce mortality	Meta-analysis not done
Kansagara 2011[36]	MEDLINE, Cochrane Database of Systematic Reviews, ClinicalTrials.gov	RCTs using insulin to achieve strict glycaemic control. Subanalysis: AMI.	AMI subanalysis: 6 studies **Malmberg 1995[31], Van der Horst 2003[48], Malmberg 2005[44]**, **Cheung 2006[28],** Rasoul 2007[49], Oksanen 2007[50],	Subanalysis: 4007	Non-ICU: 9 studies, no reduction in short-term mortality (RR 1.0, 95%CI 0.94-1.07) AMI: No mortality reduction	6-fold risk of hypos (BGL<2.2 mmol/L) in all settings RR 6.0, 95%CI 4.06-8.87, p<0.001). Oksanen[50]=study of subjects following VF

Bold=studies with specified objective of intensive glucose control in AMI

Table 2.4. Randomised controlled trials of myocardial infarction with a specific glucose target.

Trial	Subjects	Entry Criteria	Insulin Regimen	Glucose Target	Primary Outcome	Secondary Findings	Comments
DIGAMI, Malmberg 1995[31], 1997[51]	620	Myocardial infarct and admission BG >11.0 mmol/L.	Variable rate glucose-insulin solution for at least 24 hrs.	7-10 mmol/L	Reduced one year mortality in insulin infusion group (18.6% vs 26.1%, p=0.027).	Greatest benefit to patients with low premorbid cardiovascular risk profile.	Amongst first 327 subjects, blood glucose at 24 hours lower in insulin than in control subjects (9.2±2.9 vs 12±4.4 mmol/L). Insulin group received regular subcutaneous insulin after discharge, which may have contributed to better outcomes.
GIPS, van der Horst 2003[48]	940	Within 24 hrs of ST-segment elevation infarct (all had PTCA).	20% glucose-potassium solution at 3mls/kg/hr with insulin at variable rate.	7-11 mmol/L.	No significant reduction in 30 day mortality (4.8% vs 5.8%).		Median BG at 16 hours 7.7 mmol/L for GIK group and 8.1 mmol/L for controls (NS).
DIGAMI-2, Malmberg 2005[44]	1253	Myocardial infarct and either known type 2 diabetes or admission BG >11.0 mmol/L.	Variable rate glucose-insulin solution for at least 24 hrs.	7-10 mmol/L	No reduction in mortality with insulin infusion.		BG at 24 hours in insulin treated groups only 0.9 mmol/L lower than for conventional treatment group (9.1±3.0 and 9.1±2.8 vs 10±3.6 mmol/L, p=0.0001)
HI-5, Cheung 2006[28]	240	Myocardial infarct with known diabetes or admission BG ≥7.8 mmol/L.	Variable rate insulin with 5% dextrose 80 mls/hr.	4-10 mmol/L	No reduction in mortality with insulin infusion.	Mortality higher in subjects with mean 24 hour blood glucose level >8.0 mmol/L.	Mean 24 hour BG in insulin treated group only 0.7 mmol/L lower than for conventional treatment group (8.3±2.2 vs 9.0±2.8 mmol/L, NS)

Other trials of insulin glucose therapy where there were no glucose targets were excluded from consideration: ECLA (1998)[45], POL-GIK (1999)[46], CREATE-ECLA (2005)[47], GIPS II (2006)[52]

Table 2.5. Guidelines regarding glucose control in myocardial infarction

Guideline	Population	Recommendation
ESC and EASD guidelines on diabetes, prediabetes and cardiovascular disease,	People with diabetes and AMI	There is reasonable evidence to initiate glucose control by means of insulin infusion in diabetic patients who are admitted for AMIs with significantly elevated

2007[54]		blood glucose levels in order to reach normoglycaemia as soon as possible (Class IIa, Level B)
AACE/ADA consensus statement on inpatient glycemic control, 2009[55]	All hospitalized patients	Insulin infusion should be used to control hyperglycaemia in the majority of critically ill patients in the ICU setting, with a starting threshold of no higher than 10 mmoml/L. Target 7.8 – 10 mmol/L, and greater benefit may be realized at the lower end of this range. For majority of non-critically ill patients, premeal <7.8 mmol/L, random < 10 mmol/L. as long as this can be safely achieved.
ACC/AHA guidelines for the management of patients with ST elevation myocardial infarction, 2009[56]	ST elevation AMI	Reasonable to use insulin based regimen to achieve and maintain BG <10 mmol/L whilst avoiding hypoglycaemia
ACP guidelines for intensive insulin therapy for the management of glycemic control in hospitalized patients, 2011[57]	All hospitalized patients	Do not use intensive insulin therapy to strictly control glucose in non-ICU patients with or without diabetes mellitus

Appendix 3: Literature reviewed for "What Glucose Target Should be Aimed for in Acute Stroke?"

Table 3.1. Recent studies examining the relationship between admission glucose level and stroke outcomes.

Study	Subjects	Characteristics	Relation between admission glucose and mortality?	Relation between admission glucose and other outcome?	Threshold level for effect?	Comment
Baird 2003[59]	25	Ischaemic stroke	N/A	Yes, infarct size, NIHSS and mRS	7 mmol/L	
Allport 2004[60]	31	Acute ischaemic stroke	N/A	Yes, insular cortical ischaemia	N/A	
Alvarez-Sabin 2004[61]	138	MCA territory treated with tPA	N/A	Yes with NIHSS and mRS	N/A	Detrimental association greatest with early reperfusion therapy.
Farrokhnia 2005[62]	447	Acute stroke	Yes (only for non-diabetic)	N/A	Diabetes: 10.3 mmol/L, non-diabetics: 6.3 mmol/L	Threshold determined from ROC
Stollberger 2005[63]	992	All acute stroke	Yes	N/A	9.2 mmol/L for non-diabetics	
Gentile 2006[64]	960	Ischaemic stroke	Yes	N/A	7.2 mmol/L	
Yong 2008[65]	748	Received tPA for acute hemispheric stroke	Yes	Yes, with BI, mRS, 7 day neurological improvement	7.8 mmol/L	Effect not seen among subjects with known diabetes
Fuentes 2009[66]	476	Acute ischaemic stroke	Yes	Yes with mRS	8.6 mmol/L	Threshold determined from ROC
Stead 2009[67]	447	Acute ischaemic stroke	Yes	Yes with stroke severity and functional impairment	7.2 mmol/L	Relationship stronger for patients without diabetes
Poppe 2009[68]	1098	Acute ischaemic stroke treated with t-PA	Yes	Yes, with symptomatic intracerebral haemorrhage and mRS	N/A	
Ntaios 2010[69]	1446	Ischaemic stroke	N/A	Yes, with NIHSS and mRS	<3.7 and >7.2 mmol/L	
Ahmed 2010[70]	16049	Ischaemic stroke treated with thrombolysis	Yes	Yes, with NIHSS and mRS	6.7 mmol/L	similar threshold for diabetes and non-diabetics

Dziedzic 2010[71]	302	Ischaemic stroke	Yes	N/A	N/A	
Saposnik 2011[72]	8223	Acute ischaemic stroke in registry	Yes	N/A	7.5 mmol/L	
Kimura 2011[73]	97	Received tPA within 3 hours of stroke onset	N/A	Infarct volume larger and worse mRS	7.2 mmol/L	Relationship not present amongst those with successful early recanalisation.
Hu 2012[74]	774	Acute stroke	Not reported	Yes, with NIHSS, BI and mRS	Diabetes: 8.9 mmol/L, non-diabetics: 6.8 mmol/L	

NIHSS = National Institutes of Health Stroke Scale, BI = Barthel Index, mRS = modified Rankin Score

Table 3.2. Studies examining the relationship between mean glucose levels and stroke outcomes:

Study	Subjects	Characteristics	Glucose parameter	Elevated glucose predictive of mortality?	Relation between admission glucose and other outcome?	Threshold level for effect?
Baird 2003[59]	25	Ischaemic stroke	Mean capillary and mean CGMS	N/A	Yes, infarct size, NIHSS and mRS	7 mmol/L
Farrokhnia, 2005[62]	447	Acute stroke	Mean capillary	Yes	N/A	10.3 mmol/L for diabetes, 6.3 mmol/L for non-diabetics (based on ROC)
Yong 2008[65]	748	Received tPA for acute hemispheric stroke	Glucose at 24 hrs	Yes	Yes, BI. mRS, 7 day neurological recovery.	7.8 mmol/L
Fuentes 2009[66]	476	Ischaemic stroke	Maximum capillary glucose	Yes	Yes, mRS	8.6 mmol/L

NIHSS = National Institutes of Health Stroke Scale, BI = Barthel Index, mRS = modified Rankin Score

Table 3.3. Systematic reviews of randomized controlled trials of tight glucose control in stroke, where the primary outcome was death or a measure of disability.

Review	Search Method	Selection Question	Studies	Subjects	Result/Conclusion	Comment
Kansagara 2011[36]	MEDLINE, Cochrane Database of Systematic Reviews, ClinicalTrials.gov	RCTs using insulin to achieve strict glycaemic control. Subanalysis: stroke and acute brain injury	**Walters 2006**[77], **Gray 2007**[78], Azevedo 2007[79], **Bruno 2008**[80], Yang 2009[81]		Non-ICU setting: 9 studies, no reduction in short-term mortality (RR 1.0, 95%CI 0.94-1.07) Stroke and acute brain injury: No reduction in mortality	Increased risk of hypoglycaemia in all settings.
Kruyt 2010[75]	Not stated	Studies investigating the feasibility and efficacy of tight glycaemic control in patients with ischaemic stroke	**Walters 2006**[77], **Gray 2007**[78], **Bruno 2008**[80]		BG >10 mmol/L should trigger insulin administration.	Review not restricted to RCTs
Bellolio 2011[76]	Cochrane Stroke Group Trials Register, CENTRAL, MEDLINE, EMBASE, CINAHL, Science Citation Index, Web of Science, Scopus	RCTs comparing intensively monitored insulin therapy versus usual care in adult patients with acute ischaemic stroke.	**Vinychuk 2005**[82], **Walters 2006**[77], **Gray 2007**[78], **Staszewski 2007***, **Bruno 2008**[80], **Kreisel 2009**[83], **Johnston 2009**[84]	1296	No difference in death or disability and dependence (OR 1.00, 95% CI 0.78 to 1.28) or final neurological deficit (SMD -0.12, 95% CI -0.23 to 0.00).	Increased hypoglycaemia with intervention. Some studies in this review were not designed to assess neuro outcomes.

Bold=studies of stroke only, *Unpublished

Table 3.4. Randomised controlled trials of stroke with a specific glucose target.

Trial	Subjects	Entry Criteria	Insulin Regimen	Glucose Target	Glucose Achieved	Primary Outcome	Secondary Findings	Comments	Hypos
Vinychuk 2005[82]	128	Within 24 hours of ischaemic stroke onset, admission BG 7-16 mmol/L	Glucose potassium insulin infusion	<7 mmol/L	Diabetes: 7 vs 11.2 mmol/L at 12-24 hrs No diabetes: 5.8 vs 8.1 mmol/L	Improvement in NHISS compared to		4 arms to study	Not reported
Walters 2006[77]	25	Within 24 hours of ischaemic stroke onset, admission BG 8-20 mmol/L	Variable rate insulin infusion	5-8 mmol/L	In target 87% of the time vs 71% of the time (p<0.001)	-	1 death insulin group, 0 in control group	A pilot study	1 in insulin group, 0 in controls
GIST-UK, Gray 2007[78]	933	Within 24 hours of stroke onset, admission BG 6-17 mmol/L	Glucose potassium insulin infusion	4-7 mmol/L	GKI group 0.57 mmol/L lower (p<0.001)	No reduction in death at 90 days with GKI	No reduction in residual disability or functional recovery	Trial discontinued early due to slow recruitment. BG dropped spontaneously in control group.	41.2% GKI patients had BG <4 mmol/L and 15.7% required rescue iv glucose
Bruno 2008[80]	46	Within 12 hours of cerebral infarct and BG ≥8.3 mmol/L	Variable rate insulin infusion for 72 hrs	<7.2 mmol/L	7.4 vs 10.5 mmol/L	Diff in glucose achieved	2 death insulin group, 0 in control group	A pilot study	35% vs 13% had hypos <3.3
Kreisel 2009[83]	40	Within 24 hours of ischaemic stroke onset	Variable rate insulin infusion	4.4-6.1 mmol/L	6.5 vs 8.0 mmol/L, p<0.0005	Diff in glucose achieved	No difference in death or functional outcome	Not powered to detect difference in death and disability	25 vs 2 hypo events (p<0.05)
Johnston 2009[84]	74	Within 12 hours of cerebral infarct and BG ≥6.1 mmol/L	Variable rate insulin infusion	3.9-6.1 mmol/L	6.2 vs 8.4 mmol/L	Diff in glucose achieved	No difference in death or functional outcome	3 arms to study Not powered to detect difference in death and disability	30% vs 4% had at least one hypo

Middleton 2011[85]	1126	Within 48 hours of acute stroke	Variable rate insulin infusion if BG >=11 mmol/L if diabetes, >=16 for non-diabetics, up to 72 hours	4-8 mmol/L or local guidelines once insulin infusion commenced	7.0 vs 6.8 mmol/L, p=0.02 (Note small difference only)	Difference in mortality or dependency 42% vs 58% (p=0.002)	No difference in Barthel index, but higher SF-36 Physical Health Score in intervention	Intervention was package of glucose, fever, and swallowing management. Not possible to determine contribution of glucose control.	Not reported.

Table 3.4. Guidelines regarding glucose control in stroke

Guideline	**Population**	**Recommendation**
AHA/ASA guidelines for the early management of adults with ischemic stroke, 2007[86]	Ischaemic stroke	Serum glucose concentrations (possibly >7.8 to 10.3 mmol/L) probably should trigger administration of insulin (Class IIa, Level of Evidence C).
European Stroke Guidelines, 2008[87]	Ischaemic stroke	Treatment of serum glucose levels > 10 mol/L with insulin titration is recommended (Class IV evidence). Severe hypoglycaemia [<2.8 mmol/L should be treated with intravenous dextrose or infusion of 10–20% glucose (Class IV evidence)

Appendix 4: Literature Reviewed for Question "What are Appropriate Glucose Targets for Patients in General Hospital Wards?"

Table 4.1. Observational studies examining the relationship between hyperglycaemia and outcomes in hospital outside of the situations of intensive care, myocardial infarction and stroke.

Study	Subjects	Finding	Comment
Pomposelli 1999 Study[88]	97 patients undergoing general surgery	Single BGL >12.2 mmol/L predictive of nosocomial infection	
Golden 1999[89]	411 CABG patients	Mean capillary BGL ≥11.5 mmol/L associated with increased infection	
Umpierrez, 2002[90]	2030 admissions to a community teaching hospital	2x Fasting BGL ≥7.0 mmol/L or rBGL ≥11.1 mmol/L associated with increased mortality, need for ICU and longer LOS	Risk higher for new hyperglycaemia than diabetes
Cheung 2005[91]	122 subjects on TPN	Mean BGL ≥7.9 mmol/L associated with increased infection Mean BGL ≥9.1 mmol/L associated with increased mortality	
Baker 2008[92]	903 patients in general medical ward	Admission BGL ≥5.6 mmol/L associated with increased mortality	No association between BG levels and mortality amongst people with known diabetes. HbA1c also predictive of mortality in non-diabetics.
Cheung, 2008[93]	6187 admissions to a teaching hospital	Admission BGL >8.0 mmol/L predictive of mortality and longer LOS	Risk higher for new hyperglycaemia than diabetes

Table 4.2. Systematic reviews of studies examining trials of tight glycaemic control outside of the intensive care setting, and not specifically focusing on myocardial infarction or stroke

Review	Selection Question	Studies	Total subjects	Result/Conclusion	Comment
Haga 2011[94]	Effects of "tight" versus "normal" glycaemic control, peri and post-operatively, in patients undergoing cardiac surgery.	3 studies: Groban 2002[42], Lazar 2004[96], Ingels 2006[97]	686	48% reduction in mortality (OR 0.52, 95%CI ¯0.3 - ¯0.91, p=0.02). May be some benefit to tight glycaemic control during and after cardiac surgery.	
Kansagara 2011[36]	RCTs using insulin to achieve strict glycaemic control. Subanalysis: Non-ICU studies Subanalysis: Perioperative	9 non-ICU studies: Malmberg 1995[31], Van der Horst 2003[41], Butterworth 2005, Li 2006, Cheung 2006[28], Oksanen 2007[50], Azevedo 2007[79], Yang 2009[81], 5 perioperative studies: Smith 2002[43], Lazar 2004[39], Butterworth 2005[98], Li 2006[99], Barcellos 2007[100]	Non-ICU: 2677	Non-ICU setting: no reduction in short-term mortality (RR 1.0, 95%CI 0.94-1.07). Perioperative: no reduction in short-term mortality.	No trials in general medical wards Perioperative studies mostly poor quality RR hypoglycaemia 6.0, 95%CI 4.06-8.87, p<0.001).
Murad 2012[95]	Observational or randomized studies that compared the effect of intensive glycaemic control to a control group seeking less aggressive normalization of glycaemic levels. Intensive care setting excluded.	19 studies: RCTs – Malmberg 1995[31], Dickerson 2003[101], van der Horst 2003[48], Malmberg 2005[31], Cheung 2006[28], Walters 2006[77], Umpierrez 2007[102], Umpierrez 2009[103], Umpierrez 2011[104]	Varied for different analyses	No association between intensive glucose control and risk of death, myocardial infarction or stroke. Association with reduced risk of infection (RR 0.41, 95%CI 0.21-0.77). Trend to increased hypoglycaemia.	Inclusion of observational studies is questionable.

Table 4.3. Existing guidelines for glucose targets in non-critically ill patients in hospital.

Guideline	Population	Recommendation
AACE/ADA, 2009[55]	All hospitalized patients	Insulin infusion should be used to control hyperglycaemia in the majority of critically ill patients in the ICU setting, with a starting threshold of no higher than 10 mmol/L. Target 7.8 – 10 mmol/L, and greater benefit may be realized at the lower end of this range. For majority of non-critically ill patients, premeal <7.8 mmol/L, random < 10 mmol/L, as long as this can be safely achieved.
Society for Ambulatory Anesthesia, 2010[105]	Pre and intra-operative	Insufficient data to recommend the level of preoperative fasting blood glucose above which elective ambulatory surgery should be postponed. No evidence that any particular blood glucose level is either beneficial or harmful for patients undergoing ambulatory surgical procedures. Suggest that in patients with well controlled diabetes, intraoperative blood glucose levels be maintained <10 mmol/L.
ACP, 2011[106]	All hospitalized patients	Do not use intensive insulin therapy to strictly control glucose in non-ICU patients with or without diabetes mellitus.
Endocrine Society, 2012[107]		Recommend premeal target <7.8 mmol/L, random target <10 mmol/L, but modify according to clinical status. Reassess therapy if BG values fall below 5.6 mmol/L. Modify therapy when BG values are <3.9 mmol/L.

Appendix 5: Literature Reviewed for Question "What is the best method to maintain glycaemic control in a hospitalized patient who is receiving parenteral or enteral nutrition?"

Table 5.1. Clinical trials of glucose control among patients receiving enteral or parenteral feeding.

Paper	Design	Quality	Level of evidence	Statistical precision	Size and direction of effect	Relevance
Koryt-kowski 2009[108]	Sliding scale insulin (4-6 hourly if BGL >7.1 mmol/l) vs sliding scale insulin and glargine. Target glucose range (5.6-10 mmol/l). Non-critically ill hospitalized patients with ≥2 BGLs over 7.2 mmol/l (with or without prior diagnosis of diabetes) Receiving enteral nutrition therapy formula and delivery at discretion of nutrition team with majority receiving ≥50% carbohydrate.	Selection bias, randomization process not described. Information bias, open label study. Financed by Sanofi Aventis; clearly disclosed and stated that sponsor did not influence study design conduct. Inclusion/exclusion criteria described. Baseline characteristics similar between groups. Large percentage (55%) of patients in SSI arm also received NPH insulin.	II	No difference between the treatment groups in mean study glucose levels p=NS.	No effect. Both regimens effective in lowering mean glucose levels. Similar rates of hypoglycaemia on BG measures, total adverse events and LOS. Similar total daily insulin doses.	Inpatient population receiving enteral nutrition therapy.
Grainger 2007[109]	Variable dose preprandial insulin (standard of care) vs nurse led insulin protocol (variable dose lispro, regular + correctional dose) and fixed dose glargine (weight dependent). Critically ill hospitalised patients with known type 2 diabetes or FBG> 11.1. Receiving bolus enteral nutrition (TwoCal HN or Nepro renal failure) q4 hours to provide 6 feeds/day.	Selection bias, high. Information bias, high. Inclusion/exclusion criteria described. Used historical controls from before protocol implementation. Excluded NIRDM patients. Baseline characteristics, few reported but similar.	III-3	Lower mean glucose in intervention cf control group p<0.001. Greater proportion in intervention group achieved goal glucose range p<0.01, + shorter time to achieve glucose control 21 vs 60 hrs, p not reported. Increased hypoglycaemia p=0.02.	Insulin protocol increased proportion of patients achieving target blood glucose range. Modest increase in hypoglycaemia.	ICU inpatient population receiving enteral nutrition therapy.

Appendix 6: Literature reviewed for the Question "How is steroid-induced hyperglycaemia best managed?"

Table 6.1. Incidence of steroid-induced hyperglycaemia.

Author	Study Design	Steroid	Incidence of steroid-induced hyperglycaemia	Risk factors for steroid-induced hyperglycaemia	Comment
Fajans 1954[115]	steroid given prior to a GTT	CRH or cortisone	N/A	First-degree relative with diabetes	
Gurwitz 1994[114]	Case control study	Any oral glucocorticoids	N/A	Steroid dose (odds ratio rose from 1.77 for 39 mg/day hydrocortisone equivalent, rising to 10.34 for 120mg/day or more	Hyperglycaemia based on prescription of oral antidiabetic agent
Feldman-Billard 2005[113]	Retrospective audit	Pulse methylprednisolone	64% had at least one BG ≥14 mmol/L	Higher HbA1c	
Donihi 2006[110]	Retrospective audit	At least prednisone 40 mg/day or equivalent for 2 days	Overall 64% had at least one BG ≥11.1mmol/L. 56% amongst non-diabetics.	Pre-existing diabetes	21% patients not screened
Fong 2011[111]	Prospective audit	At least prednisone 25 mg/day for 2 days	71% had at least one BG ≥10mmol/L	Age	
Burt, 2011[112]	Prospective study	At least prednisone 20 mg/day for 2 days	53% had at least one BG ≥10mmol/L	Pre-existing diabetes	Used CGMS

Table 6.2. Evidence regarding the effectiveness of anti-diabetic medication in the management of steroid-induced hyperglycaemia.

Medication Class	**Intervention Studies**	**Other Literature and Comments**
Metformin	No trials identified	Limitations to its use in the inpatient setting may include the presence of risk factors for lactic acidosis, or variable oral intake
Sulphonyl-ureas (SU)	No prospective trials. Studies in ambulatory setting only. A retrospective review of 40 ambulatory pts with diabetes at baseline who had received prednisone (dose range 5-40mg/day for management of non-endocrine conditions such as COPD and SLE) described 5 patients who had received repaglinide. However insulin was also required to achieve glycaemic control in 4 of the 5[121]. Kasayama reported 3 adult ambulatory patients with immunological conditions, initially requiring prednisone 20-40mg/day, and maintained on 5-10mg/day, who developed hyperglycaemia after 1-2 yrs of maintenance GC therapy[122]. All 3 were able to be controlled with glimepiride monotherapy (1-3mg), achieving Hba1c <7%.	Although predominant effect of GCs is a reduction in insulin sensitivity, individuals who develop hyperglycaemia with GC administration, have a reduced insulin secretory capacity[123]. Hence insulin secretogogues are not an ideal choice. Risk of hypoglycaemia as GC tapered if long acting agents are used[124], where there is meal omission[125] or if renal function is reduced[126]. SU use has been suggested to be limited to those with milder degrees of GC induced hyperglycaemia, where fasting BG <7mmol/L[126]. Since inpatients usually receive high GC doses and may have variable oral intake or renal function, SUs have a limited role.
Thiazolidine-diones	Published reports of use for steroid- induced hyperglycaemia in setting of organ transplantation only. Pioglitazone improved glycaemic control as an adjunct to insulin (mean HbA1c falling by 1.28%) in a series of renal transplant recipients managed with prednisone (4-20mg/day) and sirolimus or mycophenolate, 6 of the 10 patients had diabetes predating their transplant[127]. Efficacy of other thiazolidinediones for post transplant diabetes or GC related DM has also been described[128-130].	Delayed onset of action makes this group generally unsuitable for acute inpatient management of hyperglycaemia.
Incretins	No completed trials identified. A trial of vildagliptin in the management of post transplant diabetes is in progress[131].	Potential role has been explored by van Raalte[132].
Insulin	No trials of insulin regimens in management of steroid-induced hyperglycaemia in hospital other than intravenous insulin[133]. Observational report suggests that use of morning isophane insulin versus long acting insulin (either insulin with rapid acting insulin at mealtime) achieved glycaemic control more quickly[134]. Preliminary report suggests that early use of basal insulin in the	Where marked fasting hyperglycaemia (>10mmol/L) is present, insulin is thought to provide better management than oral agents[126,137]. Insulin provides greater dose flexibility, more rapid onset of action and titration and there is usually no dose ceiling. Insulin dose requirements need to be individualised, due to variations in insulin sensitivity, insulin secretory capacity, GC regimen and dosing, oral intake, renal function

	setting of renal transplantation, prevents the development of subsequent new onset diabetes[135]. In response to prednisone 60mg/day a series of 10 patients with T1DM managed with subcutaneous insulin pump required an increase of between 30-100%[136].	and prior control. Pre-emptive increases and decreases in insulin required as GC dose is adjusted. This usually requires daily adjustment. Generally the insulin regimen or adjustments to a pre-existing regimen should predominantly target post-prandial control, and with morning GC administration, the afternoon hyperglycaemia. Clore has advocated the use of isophane insulin for management of steroid hyperglycaemia[124], the initial dose determined according to GC dose and patient weight (e.g. prednisone 10mg daily requires 0.1unit/kg/day as isophane insulin; prednisone 40mg daily requires 0.4 unit/kg/day), and titrated according to response. For a patient already on insulin, this may be added to the existing regimen. This titration schedule is easier to continue upon discharge than more complex regimens, and more likely to be successful if patients have a consistent routine and carbohydrate consumption from day to day[138]. Isophane insulin can be supplemented with ultraquick insulin analogue with meals[139]. Basal plus prandial insulin is likely to be required in patients receiving high dose GC (eg >50mg prednisone/day) where prior glycaemic control was poor, GCs had been initiated without pre-emptive consideration of glycaemia or GC are given as split dose. With multiple daily dose GC regimens, isophane insulin twice daily with prandial rapid acting analogue can be initiated. A regime that controlled glycaemia on previous occasions can be re-initiated when cyclical GCs are required, as long as there has been no major interval change in weight or renal function.

Appendix 7: Literature Reviewed for Question "What is the best method to maintain glycaemic control in a hospitalized patient who is not critically ill?"

Table 7.1. Randomised controlled trials comparing insulin regimes for glucose control in hospital.

Author	Study design	Quality and risk of Bias	Level of evidence	Statistical precision and significance	Size and direction of effect	Relevance
Dickerson 2003[101]	153 subjects. Randomised trial comparing glycaemic control in medical patients with type 2 diabetes receiving sliding scale insulin versus routine diabetes medications.	Medium–high risk of bias. Bias that population chosen from convenience sample. Randomisation procedure clearly described. Open label. Clear exclusion criteria. Baseline characteristics mostly similar. May have been treatment changes during study.	III-1	Statistical calculations included in study design. Hyperglycaemic and hypoglycaemic events.	Primary outcome was frequency of hyperglycaemia (>16.7) and hypoglycaemia (<2.8). No difference between the two groups. No difference in secondary outcome which was LOS.	General medical patients, similar to Australian general medical wards
Yeldandi 2006[143]	94 patients. Randomised trial comparing once-daily glargine insulin with twice daily NPH/regular insulin for control of hyperglycaemia in inpatients post-cardiovascular surgery.	Medium risk of bias. Randomisation procedure not described. Open label. No exclusion criteria. Baseline characteristics similar.	III-1	No calculations were done to show power. Mean BG 124mg/dL v 131mg/dL p=0.065 Hypoglycaemia less with glargine p=0.036.	Minimal effect. Calculated % of BGs within target range. Similar BGs achieved with both regimens in whole cohort. Subgroup with diabetes: bd NPH had lower mean BG than glargine. Subgroup without diabetes: no difference in mean BGs. % of BGs within target range similar between the groups. Hypoglycaemia less common with glargine p=0.036.	Restricted to post operative cardiac surgery. Patients could have diagnosed diabetes or hyperglycaemia. Caloric intake very diminished therefore patients not requiring a prandial insulin component.
Umpierrez 2007 (RABBIT 2 Trial)[102]	130 patients. Randomised trial of basal-bolus insulin therapy compared to sliding scale insulin in the inpatient	Medium risk of bias. Not clearly defined what the randomization process was. Open label. Inclusion and exclusion criteria detailed. Baseline	III-1	Unclear if study appropriately powered. FBG p<0.001, mean random blood glucose	Large effect. Significant difference in favour of BBI in regards glycaemic control, p<0.001, lower mean daily glucose, mean random BG and FPG. No difference in	Tertiary hospital. Similar patient population and treating teams. Difference is ceasing all oral

	management of general medical patients with type 2 diabetes.	characteristics same. Unrestricted educational event from Sanofi Aventis		p<0.001, mean glucose throughout hospital stay p<0.001.	hypoglycaemia. No difference in secondary endpoints (LOS and mortality). Significant difference in the units of insulin used.	hypoglycaemic agents on admission. Sliding scale not appropriate comparator.
Umpierrez 2009[103]	130 subjects. Randomised trial comparing regimens with detemir and aspart versus neutral protamine hagedorn and regular in medical patients with type 2 diabetes.	Medium risk of bias. Selection bias, randomisation process unclear. Information bias, open label study. Inclusion and exclusion criteria detailed. Baseline characteristics the same. Financed by Novo-Nordisk (disclosed and had no influence on study design).	III	No difference between the treatment groups, p=NS	No effect. Both regimens equally as effective in lowering mean BGs and lead to similar rates of hypoglycaemia. BG <140mg/dL was achieved in 45% of BBI group and 48% in split mixed group.	Tertiary hospital. Similar patient population and treating teams. Difference is ceasing all oral hypoglycaemic agents on admission.
Umpierrez 2011 (RABBIT 2 Surgery)[104]	211 subjects. Randomised trial of basal-bolus insulin therapy compared to sliding scale insulin in the inpatient management of patients with type 2 diabetes undergoing general surgery	Medium risk of bias. Computer generated randomisation. Open label. Inclusion and exclusion criteria detailed. Baseline characteristics the same. Unrestricted educational event from Sanofi Aventis	II	Appropriately powered. FBG p=0.037, mean BG p<0.001, BG <140mg/dL p<0.001. Secondary outcomes p=0.003, wound infection p=0.05.	Large effect. BBI better than SSI in treating hyperglycaemia. Significant difference between groups in regards glycaemic control, p<0.001, lower mean daily BG and FPG. More hypoglycaemia in BBI group. Difference in secondary endpoints favour BBI, with reduced wound infections and ICU stay. No difference in LOS or mortality. Significant difference in units of insulin used.	Tertiary hospital. Similar patient population and treating teams. Difference is ceasing all oral hypoglycaemic agents on admission. Sliding scale not appropriate comparator.

Appendix 8: How Should Patients on Insulin Pump Therapy be Managed in Hospital?

Table 8.1. General recommendations for diabetic patients who continue continuous subcutaneous insulin infusion therapy in hospital.

- CSII therapy is to be continued in hospital only in those situations where the patient or their guardian have the ability to self-manage their insulin dosing and the pump (button pushing and set changes) safely.
- **CSII should never substitute for an intravenous insulin infusion to treat patients with diabetic ketoacidosis**.
- In a metabolically stable patient, who is able to eat, CSII may be more appropriate than an intravenous insulin infusion or a basal + top up injection regimen.
- Regardless as to whether CSII is to be continued or ceased during the patient's hospitalization it is strongly recommended that an endocrine service consultation (if available) is obtained for all patients at the time of admission. The endocrinologist usually responsible for the care of the patient should be notified at the time of admission.
- The patient will be responsible (in consultation with the endocrine team) for setting basal rates, determining bolus doses administered with meals or to correct elevated glucose levels and for set changes.
- Comprehensive documentation all aspects of CSII management is required.
- CSII therapy must be substituted with either a subcutaneous insulin regimen or an intravenous insulin infusion if:

  1/ The patient or guardian is not able to demonstrate that they are able to safely and reliably manage the insulin pump.

  2/ A severe acute illness is present.

  3/ A procedure or investigation is planned potentially rendering CSII therapy ineffective or the anaesthetic staff are not confident regarding the management of the pump.

  4/ There are concerns regarding a malfunction in the pump.
- Should there be concerns regarding the technical functioning of the pump manufacturer's help line should be contacted.
- CSII therapy should never be discontinued without first ensuring the provision of insulin via an alternative route (IV infusion or subcutaneous injection)
- The patient's admission to hospital should be used as an opportunity to review all aspects of CSII management by the Endocrinology team.

Table 8.2. Minimum patient competency requirements for continued inpatient CSII therapy.

- Ability to operate the management menu of the device to alter basal rates.
- Ability to operate the management menu of the device to modify parameters of and operate the bolus calculator (including a basic level of proficiency in carbohydrate counting)
- Ability to perform a set change and manage line occlusions or leaks and have relevant supplies to implement a set change.

Table 8.3. Contraindications to continued inpatient CSII therapy.

- Patient is unable to demonstrate a basic level of competency in the operation of their insulin pump.
- Impaired or fluctuating conscious state.
- Major psychiatric disorder (psychosis)
- Severe acute illness (including diabetic ketoacidosis) requiring an insulin infusion
- Lack of supplies (infusion sets, batteries and other equipment required to continue the patient on CSII therapy)
- Extensive skin infections or inflammation.
- Concerns regarding technical malfunction of the pump.
- Numerous radiological procedures (CT and MRI). The pump should be suspended and disconnected prior to the patient entering a CT or MRI scanner.
- Patient undergoing lengthy or complicated surgery, or serious medical illness likely to be accompanied by significant metabolic disturbance.

Table 8.4. Hospital documentation recommended for inpatients continuing CSII.

• The model of the pump and duration of CSII. • Date current pump purchased. • Details of insulin delivery line. • The name of the insulin infused with an indication that it is being delivered via a pump. • Insulin delivery parameters including basal rates, insulin to carbohydrate ratios, correction factors, duration of insulin action and glucose targets. • Any changes to the pump insulin delivery settings should be clearly documented at the time they are implemented. It should also be documented that these changes have been clearly conveyed to and confirmed by the patient or their guardian. • The date and time of set changes. A follow-up fingerprick glucose should be performed 2 hrs later and documented. • Fingerprick glucose readings. At least 4 (pre-meal and bed-time) and preferably 7 (pre-meal, 2 hour post meal and bed-time) finger-prick glucose readings are recommended. These should be documented on the glucose monitoring chart. • Ketone readings. Blood ketones are preferable to urinary ketone measurements. • A signed agreement with the patient that clearly documents the patient's responsibilities with regard to inpatient CSII management is recommended.

Table 8.5. Intra-operative conditions appropriate for CSII or switch to temporary insulin infusion

Situations appropriate for intra-operative CSII	**Indications for intraoperative intravenous insulin infusion**
• Procedure of short duration (e.g. D&C). • Medical and anaesthetic staff that are familiar with pumps. • Patient awake and alert intraoperatively. • Patient metabolically stable. • Patient alert and to resume eating shortly after completion of the procedure.	• Prolonged and complicated procedure (eg coronary bypass surgery). • Medical and anaesthetic staff unfamiliar with CSII. • Patient critically unwell and metabolically unstable. • Prolonged post-operative recovery period.

Table 8.6. Recommendations for perioperative management of CSII.

Situation	Recommendations
Preoperative	• Perform a set change on the morning or afternoon of the day prior to the procedure. Ensure that the insertion site is well away from the operative field. • In fasting patients consider infusing insulin at a reduced temporary basal rate eg 70%. • IV dextrose (eg 5% at 80ml/hr) should be infused to prevent ketosis. • No boluses are necessary unless they are used to correct elevated glucose levels. • Monitor glucose levels and ketones with increased frequency as per the hospital's established protocols. • If CSII is to be continued intra-operatively consent must be obtained from the patient or their guardian. • If CSII is to be continued intra-operatively a label which is clearly visible must be attached to the patient stating that they have Type 1 diabetes and are using an insulin pump.
Intraoperative	• Ensure that the anaesthetist is aware that CSII is to be continued during surgery and is able to disconnect the pump if necessary. • Ensure that the pump is accessible to the anaesthetist intraoperatively. • Glucose levels should be monitored frequently (at least hourly) and ketones as determined by the anaesthetist. • In the event of an unexplained elevation in glucose levels or frank ketosis an IV insulin infusion should be commenced and the insulin pump suspended and disconnected. • In the event of intraoperative hypoglycaemia, the pump should be suspended immediately and a bolus of IV dextrose administered. Once hypoglycaemia has been abolished the insulin pump can be recommenced at a reduced basal rate. Alternatively the IV dextrose can be infused at a greater rate. In the face of unstable glucose levels, and an anaesthetist with limited CSII experience, CSII should be ceased and a formal IV insulin infusion commenced. • Bipolar diathermy is contraindicated. Unipolar diathermy can be used.
Ceasing and Recommencing CSII	• When commencing patients managed with CSII on a subcutaneous insulin regimen the first injection(s) should be given at the time CSII is ceased and should include a long acting insulin analogue. • An intravenous insulin infusion should be commenced within 2 hours of cessation of CSII. • In hospitalized patients where CSII has been ceased with the patient managed on an insulin infusion or multiple daily injections, when recommencing CSII is preferable that CSII be recommenced in the morning and with the insertion of a new line. • In those managed with multiple daily injections while an inpatient, if they are on a long acting analogue administered in the evening, half the dose should be given on the night with CSII commenced the next morning. • CSII should be re-commenced immediately prior to cessation of an insulin infusion.

Appendix 9: Appropriate Glucose Control in End of Life Situations

Table 9.1. Phases of end of life pathway

Phase	Description
Stable	The person's symptoms are adequately controlled on their established management plan but interventions to maintain symptom control and quality of life have been planned. This phase may last for several years.
Unstable	The person develops a new unexpected problem or a rapid increase in the severity of existing problems.
Deteriorating	The person's existing symptoms gradually worsen or they develop new but unexpected problems.
Terminal	Death is likely in a matter of days and no acute intervention is planned or required.

Table 9.2. Suggested inpatient management of type 1 and type 2 diabetes in the phases of the end of life pathway

Phase	Type 1 diabetes	Type 2 diabetes
Stable	Inpatient management of glycaemia as per standard care. Insulin should not be ceased. Hospitalisation may provide an opportunity to review the appropriateness of the patient's current insulin regimen, glycaemia targets, need for any non-diabetes medications exacerbating glycaemia and general diabetes education. Stable Phase palliative patients are usually discharged home from hospital. Post-discharge follow-up should be organized.	Inpatient management of glycaemia as per standard care. Hospitalisation may provide an opportunity to review the appropriateness of the patient's current diabetes regimen, glycaemia targets, need for any non-diabetes medications exacerbating glycaemia and general diabetes education. Stable Phase palliative patients are usually discharged home from hospital. Post-discharge follow-up should be organised.
Unstable	As for "Stable Phase". If the patient is to be discharged home consider simplifying their insulin regimen if appropriate. Liberalisation of the patient's diet should be considered.	As for "Stable Phase". If the patient is to be discharged home from hospital, a review of the current medication regimen should be undertaken aiming for simplicity, minimisation of the risk for hypoglycaemia and other adverse effects associated with some oral agents. Liberalisation of the patient's diet should be considered.
Deteriorating	Adjustments to usual insulin regimen are likely to be required at the time of hospitalisation particularly if nutritional intake is reduced with cachexia, renal and hepatic impairment, delirium or altered conscious state, increasing pain, mucositis, nausea and vomiting. Insulin should not be withdrawn completely unless it is at the request the patient or their family. Consider simplification of	If the patient is on oral hypoglycaemic agents at the time of hospitalisation a decision is required as to whether these are still appropriate and the need for each reviewed. If hyperglycaemia is present and responsible for symptoms consider commencing a simple insulin regimen e.g. a single dose of basal insulin to promote comfort. If the patient is already on insulin, simplification of the

	the insulin regimen e.g. single basal insulin injection with top-ups of short acting insulin analogue to maintain comfort. Less frequent BG monitoring (1-2 per day) and ketone checks are recommended. Aim for BGs between 5.0-15.0 mmol/L. Remove food restrictions. Review the need for any non-diabetes medications exacerbating hyperglycaemia or hypoglycaemia. If patient is to be discharged home from hospital, consider modifying insulin regimen aiming for simplicity and minimisation of the risk for hypoglycaemia. Ensure follow-up and support of the patient post discharge.	current insulin regimen should be implemented if possible with less frequent BG monitoring (1-2 per day). Aim for BGs between 5.0-15.0 mmol/L. Remove food restrictions. Review the need for any non-diabetes medications exacerbating hyperglycaemia or hypoglycaemia. Ensure follow-up and support of the patient post discharge.
Terminal	The patient's preferences or those of their carer take precedence. The primary objective is to maintain patient comfort. A single daily injection of basal insulin administration may be required to maintain comfort by addressing severe hyperglycaemia and to prevent frank ketoacidosis. Consider minimising/ceasing all glucose and ketone monitoring after the appropriate discussion with the patient or their carer.	The primary objective is to maintain comfort. Consider ceasing all glucose monitoring. Consider ceasing all insulin and oral hypoglycaemic agents. If severe hyperglycaemia and the patient symptomatic from hyperglycaemia consider commencing a daily injection of basal insulin.

Table 9.3. Factors potentially influencing management of glycaemia.

Anorexia and weight loss. Confusion and altered conscious state. The stress response to pain, anxiety, infection and unrelated intercurrent illness. Disturbance in glucose metabolism resulting from some malignant tumours. Use of corticosteroids and other diabetogenic medications. Metabolic derangement including renal and hepatic dysfunction.

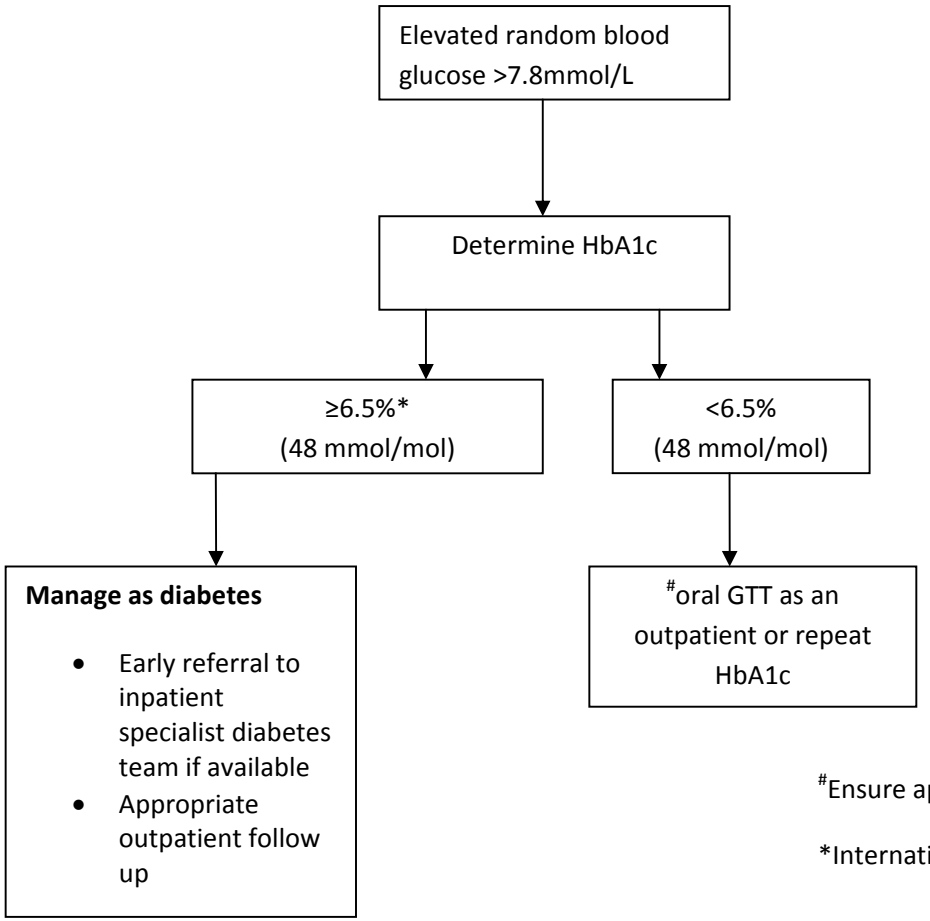
Appendix 10: How Should Patients with Newly Discovered Hyperglycaemia be Followed-Up?

Table 10.1. Incidence of newly diagnosed diabetes at various glucose and HbA1c screening thresholds

Study	Aim	Glucose threshold and diabetes prevalence	Subjects above Threshold	Quality	Level of evidence	Risk of Bias	Measures of accuracy for HbA1c	Relevance
Krebs 2000[156]	Prevalence study. Retrospect-ive trial.	22% prevalence of undiagnosed diabetes at random plasma glucose ≥7.8 mmol/l.	88 Subjects not described	No declaration of conflict of interest. Inclusion / exclusion criteria unclear.	IV	Mode-rate	At plasma glucose of ≥7.8mmol/L and HbA1c ≥6.0%. Sensitivity 47%. Specificity unable to calculate from the data.	New Zealand hospital. Diabetes diagnosed by various methods.
Greci 2003[157]	Study of diagnostic yield.	60% prevalence of undiagnosed diabetes at random plasma glucose ≥6.9 mmol/l.	35 Subjects were described	No declaration of conflict of interest. Small number of patients. Inclusion / exclusion criteria detailed.	IV	High	Sensitivity and specificity at random BG ≥6.9 mmol/l and HbA1c>6.0% were 57% and 100% respectively. Sensitivity and specificity at random BG ≥6.9 mmol/l and HbA1c 5.2% were 100% and 50% respectively. PPV at >6% 100%, NPV at <5.2% 100%.	Similar patient population. Diabetes diagnosed on basis of fasting BG x 2 or GTT. Better HbA1c assay available (not HPLC)
Gray 2004[158]	Study of diagnostic yield, but also prevalence study.	21% prevalence of undiagnosed diabetes at random plasma glucose ≥6.1 mmol/l.	62 Subjects were described	Declarations of conflict of interest. Inclusion / exclusion criteria detailed previously.	IV	Mode-rate	With random BG of ≥6.1 mmol/l and HbA1c ≥6.2% Sensitivity=86%, specificity=94% PPV 80%, NPV 96%	Patient group confined to those with acute stroke. UK setting. Diabetes diagnosed on basis of GTT. Hba1c ≥6.2%, HbA1c assay HPLC
George 2005[159]	Prevalence study.	33% prevalence of undiagnosed diabetes at random plasma glucose ≥7.0	36 Subjects not described	No declarations of conflict of interest. Inclusion / exclusion criteria	IV	Mode-rate	HbA1c not done.	UK hospital emergency department. Diabetes diagnosed on basis of fasting plasma glucose x2

		mmol/l.		detailed.				
Wong 2010[155]	Prevalence substudy of RCT of tight glucose control for myocardial infarction.	8% prevalence of undiagnosed diabetes at random plasma glucose ≥7.8 mmol/l.	55 Subjects were described	Declaration that there were no conflicts of interest. Inclusion / exclusion criteria detailed previously.	IV	Mode-rate	HbA1c not reported.	Australian population. Patient group confined to hyperglycaemic patients with myocardial infarction. Diabetes diagnosed mainly on basis of GTT.
Valentine 2011[160]	Study of diagnostic yield, but also prevalence study.	11% prevalence of undiagnosed diabetes at random plasma glucose ≥5.5 mmol/l.	2672 Subjects were described	Declaration of conflict of interest from authors. No inclusion / exclusion criteria	IV	Mode-rate	No measures of accuracy.	Similar patient population, general medical patients in Australian setting. Diabetes diagnosed on basis of HbA1c ≥6.5% (HPLC) only. Poor uptake of oGTT post discharge.
De Mulder 2011[161]	Study of diagnostic yield, but also prevalence study.	25% prevalence of undiagnosed diabetes at random plasma glucose ≥7.8 mmol/l.	109 Subjects were described	Declaration that there were no conflicts of interest. Inclusion / exclusion criteria detailed.	IV	Mode-rate	At random BG of ≥7.8mmol/L and HbA1c ≥6.5%. Sensitivity 29%, specificity 100%, PPV 100%, NPV 71%	Dutch hospital. Patient group confined to hyperglycaemic patients with acute coronary syndrome. Diabetes diagnosed by GTT.

Figure 11.1. Suggested approach to diagnosis of diabetes and follow-up of inpatient with newly discovered hyperglycaemia.



#Ensure appropriate communication to the GP

*International expert committee

Appendix 11: The Specialist Diabetes Inpatient Management Team.

Table 11.1. Hospital approaches to diabetes management

Approach	Description	Evidence
Consultant Service.	The traditional hospital model of care, whereby specialised diabetes services are invited, at the discretion of the admitting team, to assist with specific patients' diabetes management.	There is no evidence that improving this model has resulted in any substantial benefits. Anecdotal evidence suggests that this is akin to "shutting the gate once the horse has bolted".
Systematic Hospital-wide Diabetes Programme	These programmes aim to improve the identification of patients with diabetes and to enhance the diabetes management skills of all staff, by education and implementation of diabetes management and prescription guidelines. The responsibility of managing the patient's diabetes remains with the admitting team.	The evidence supporting such an institution-wide approach in improving diabetes-related outcomes is limited to one comparative study[162] which demonstrated a reduction in length of stay of 1.8 days for patients with primary diabetes following the intervention.
Specialist Diabetes Inpatient Management Team	This involves a multidisciplinary team approach, with the role of the Inpatient Diabetes Management Team varying from an advisory function to active management of the patient's diabetes, for all patients with diabetes and usually commences at the time of the patient's admission.	Several comparative trials (4-9) have shown reductions in ALOS of 0.26-5.6 days following intervention by an inpatient diabetes management team, primarily involving a specialist diabetes nurse (some with prescribing capabilities). See table 11.2.

Table 11.2. Studies examining effectiveness of Specialist Diabetes Inpatient Teams

	Study Design	Subjects	Team	Finding	Comment	Setting
Koprosky 1997[163]	RCT of Diabetes Team Care vs standard care	179	Diabetes nurse educator and Endocrinologist	Primary diagnosis diabetes: median LOS 5.5 days (Team) vs 7.5 days (control) Secondary diagnosis diabetes: median LOS 10 days (Team) vs 10.5 days (control) Post-discharge control better with Team Care: 75% good control vs 46%. Readmission lower with Team Care: 15% vs 32%, p=0.01.	Failure to achieve significance due to small sample size?	Single US medical centre
Davies 2001[164]	RCT of Diabetes Specialist Nurse Care vs standard care	300	Diabetes specialist nurse	Median LOS 8 days (intervention) vs 11 days (control), p<0.001. No difference readmission rate. Intervention group more satisfied with diabetes care (p<0.001). Control group £436 more expensive per patient.		Single UK teaching hospital
Cavan	Retrospective	1811	Diabetes	Median LOS decreased from 11 to 8 days (p<0.001)	Net saving of 4171	Single UK

2001[165]	analysis of diabetic admissions pre and post intervention		specialist nurse	amongst medical patients and 8 to 5 days (p<0.001) in surgical patients	bed days over one year	hospital
Sampson 2006[166]	Retrospective analysis of diabetic admissions pre and post intervention	14722	Diabetes inpatient specialist nurse service	Mean excess bed days for diabetes admissions reduced from 1.9 days to 1.2 days after introduction of the service	Estimated 700 bed days saved per 1000 inpatients with diabetes	Single UK teaching hospital
Newton 2006[167]	Random chart audit pre and post intervention	Not reported	Diabetes clinical nurse specialist, Diabetes nurse case managers, medical director	Reduction in mean glucose levels from 9.8 mmol/L to 8.4 mmol/L (p<0.0001). Reduction in LOS for patients with diabetes from 6.01±0.32 to 5.75±0.38 days (p=0.01).	Estimated saving of $2.2M per year for the hospital	Single US tertiary care hospital
Courtney 2007[168]	Prospective study comparing diabetic admissions with intervention vs historical controls	452	Diabetes Specialist Nurse with prescribing rights	Reduction in medication errors from median 6 to 4 (p<0.01). Reduction in LOS from median from 9 to 7 days (p<0.05)		6 wards in UK general hospital
Flanagan 2008[169]	Retrospective analysis of diabetic admissions pre and post intervention	28,016	5 specialist nurses, consultant and specialty registrar	LOS fell from 8.3 0±18 to 7.7±0.10 (p=0.002). Effect predominantly in medical patients. Emergency admissions fell from 9.7±0.2 to 9.2±0.2 (p<0.001). No change in elective admissions.		Single UK teaching hospital
Brooks 2011[170]	Retrospective audit	1140	Endocrinologist, diabetes nurse specialist, junior doctor	Reduction in average LOS for all patients with diabetes from 9.39 to 3.76 days. No change in average LOS for patients with primary diagnosis of diabetes	Estimated cost saving £132,500 over 3 months	Single UK district hospital

Table 11.3. The role of the Specialist Diabetes Inpatient Team

Improving diabetes management expertise throughout the hospital, Development and implementation of specific diabetes management protocols, Direct management of diabetes with specific referral criteria, Ward liaison, troubleshooting, management advice, Discharge planning,

Appendix 12: What Routine Measures Should be Undertaken for People with Diabetes Admitted to Hospital?

Table 12.1: Role of various team members in ensuring optimal routine diabetes care in hospital and after discharge.

Team member	Role in hospital management	Role in discharge planning
Diabetes Educator	Ensure appropriate blood glucose testing and quality control of glucose testing kits, support to ward nursing staff. Provide clinical leadership and continuing stable care in an environment of transitional and rotating medical, nursing and allied health workforce. Report back to the diabetes team if input should be offered to the referring unit.	Assess and consolidate knowledge and skills regarding eating plan, physical activity, self-monitoring, medication usage, insulin adjustment, sick day management, foot care. Qualified professionals are "ADEA Credentialled Diabetes Educators"[176]. If available, the services of a diabetes educator are useful in the early stages and a continuing liaison can be established.
Dietitian	Ensure appropriate diet in hospital and nutritional needs are met. Liaise with Diabetes Team regarding changes in diet particularly in the situation of enteral and parenteral nutrition.	Assess readiness to change eating behaviour, and dietary counseling that is innovative and specific to the requirements of the individual. Provide dietetic intervention – recommendations that include consistency in day-to-day carbohydrate intake, substitution of sucrose-containing foods, usual protein intake, cardio protective nutrition interventions, weight management strategies, regular physical activity
Pharmacist	Medicines review	Home medicines review on discharge for patients with co – morbidities.
Podiatrist	Podiatric advice and initial management of high risk foot	Organise follow up management of high risk foot. Note Medicare Enhanced Primary Care item available for GP referrals.
Aboriginal Health Worker		Providing culturally appropriate and practical support and counseling for ongoing care, thus improving patient understanding and adherence to treatment programs.
Social Worker	Liaison with family and social services	Without ensuring other aspects of the patient and his/her family welfare are catered for, good diabetes management may be challenging to achieve.

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GLOSSARY OF TERMS USED RELATING TO INSULIN

Sliding scale insulin	The prescription of insulin given in a variable dose depending on the glucose level at the time. Often this is the sole insulin prescribed.
Supplemental insulin	The administration of variable dose insulin to correct hyperglycaemia, given in conjunction with appropriate adjustments to the patient's scheduled anti-diabetic therapy.
Correctional insulin	This term is used interchangeably with "supplemental insulin"
Basal insulin	Intermediate or long acting insulin providing background insulin (eg Protaphane®, Humulin NPH®, Lantus®, Levemir®)
Prandial insulin	Rapid acting insulin given at mealtimes (eg Novorapid®, Humalog®, Apidra®)
Basal bolus insulin	Insulin regime comprising the combination of a basal insulin with multiple daily doses of prandial insulin
Ispohane insulin	Intermediate acting insulin (eg Protaphane®, Humulin NPH®)
Continuous subcutaneous insulin infusion (CSII)	Insulin administered by continuous subcutaneous infusion via an patient self-managed insulin pump

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