SPAGG

Specialist Palliative Care Audit and Guidelines Group

Coversheet for Specialist Palliative Audit and Guideline Group Agreed Documentation

This sheet is to accompany all documentation agreed by SPAGG. This will assist maintenance of the guidelines as well as demonstrating the governance process undertaken prior to members seeking local approval in their areas of work.

Document Title	**A guideline for management of diabetes in Palliative Care**
Document Date	October 2022
Document Purpose and Intended Audience	Guideline to help avoid hypoglycaemia, limit symptomatic hyperglycaemia and avoid unnecessary blood sugar monitoring and complex insulin regimes. This guideline is intended for use by healthcare professionals working with palliative patients.
Authors	Dr Maddy Turley, Palliative Registrar, West Midlands
References	See section 17
Consultation Process	Reviewed by SPAGG and ratified
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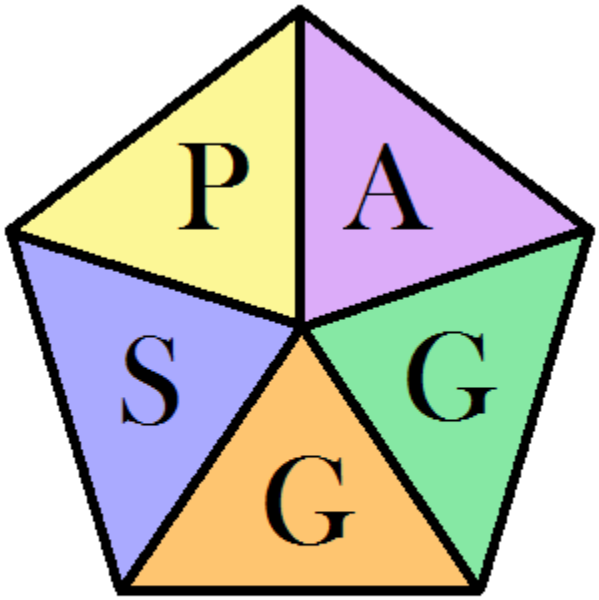
Version History

Version	Date	Summary of change/ process
1.1	13/11/2019	Reviewed by SPAGG and ratified



Specialist Palliative Care
Audit and Guidelines Group

A Guideline for the Management of Diabetes in Palliative Care

Dr Maddy Turley

Key Points

1. Provision of support to allow people to die with dignity, keeping them as comfortable as possible until the end, and assisting families to manage this often distressing experience
2. Highlight the awareness, identify training and educational needs for high quality end of life diabetes care
3. To foster partnerships in end of life diabetes care with established palliative care plans

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1. Introduction

1.1 There is very little gold standard evidence for managing diabetes in the palliative care population, mainly due to the vulnerability of this group and difficult recruitment. The following guidelines are, therefore, based on review of available literature and clinical experience shared by the diabetes and palliative care teams.

2. Purpose of the document

2.1 The aim of these guidelines for management of diabetes in end of life care is to

- Avoid hypoglycaemia
- Limit symptomatic hyperglycaemia
- Avoid unnecessary blood glucose checks and complex insulin regimes
- Prompt checking of CBG if symptoms of hypo or hyper glycaemia are present, or if a diabetic patient's condition changes.
- Consider treatment of diabetic emergencies
- Ensure the patient is on the lowest effective dose of steroid

2.2 Management will be different for each patient and will need to be reviewed as their condition changes – for example, as their oral intake and weight changes.

3. Scope

3.1 This is a regional guideline for all adults with palliative care needs, regardless of their care setting. It can be used in hospital, hospice and community. There will be specific situations which will demand slight alterations in the way the guidance is used, and this will be highlighted throughout the document.

4. Definitions

QDS – four times a day
TDS – three times a day
BD – twice daily
OD – once daily
ACEi – ACE inhibitor
ARB – Angiotensin receptor blocker
GP1 analogue – Glucagon like peptide
Normoglycaemia – normal blood glucose

CBG – capillary blood glucose
DSN – diabetes specialist nurse
DKA – diabetic ketoacidosis
HHS – Hyperglycaemic, hyperosmolar state
LA – long acting (insulin)
HPA-axis – Hypothalamic-pituitary-adrenal axis

Hypoglycaemia

- Defined as CBG below 4.0 mmol/L
- Symptoms include
  - pallor, sweating, tremor, tachycardia, loss of concentration, aggression/confusion, fits, transient neurological deficit, reduced conscious level
- Causes in palliative care population include
  - Weight loss
  - Anorexia
  - Renal failure (drugs not metabolised)
  - Liver failure (decreased glycogen and gluconeogenesis)
- Treatment should be instigated when identified, if appropriate to do so. (Appendix 4)

Hyperglycaemia

- Defined as CBG above 15.0 for the purpose of this guidance [1,3]
- Symptoms include
  - Thirst, dry mouth, confusion, drowsiness, polyuria and lethargy
- Causes in palliative care population include
  - Steroid use
  - Stress response to illness
  - Co-existent infection
  - Pre-existing diabetes
  - Pancreatic cancer
- Treatment should be instigated if there are symptoms, which generally occur when the CBG is >15[4]. Which treatment depends on the individual case.

5. Target Setting

5.1 Target setting has two aims:

a) To prevent distress from the acute metabolic complications of DKA, HHS and hypoglycaemia
b) To provide symptomatic comfort by preventing high blood glucose related symptoms of excessive thirst, polyuria and excessive tiredness

Whilst CBG targets can vary between patients and their disease duration, a target range of 6-15mmol/l will be appropriate in most cases [1,2].

5.2 A move away from rigid target based CBG control is advised, especially avoiding the use of rapid acting insulins if the CBG is 'high' in those T2DM or steroid induced diabetes. There is no evidence to show that the use of rapid acting insulins will offer any symptomatic benefit in this patient group. In these situations, an alteration to the existing diabetes regimen is preferable to ensure CBG readings are within the preferred range. However, in patients with T1DM with a prognosis of longer than weeks, use of rapid acting insulins may avoid ketoacidosis, which would add significant burden of illness. Therefore patient selection, that encompasses type of diabetes and predicted prognosis, is crucial when considering prescription of PRN rapid acting insulin.

6 Glucose Monitoring

6.1 Frequency and method of testing is likely to need to change. Ideally, CBG monitoring should be minimised where possible. Urinalysis may be sufficient in some cases, particularly where there is no hypoglycaemia risk, or within the community where access to a CBG machine may be limited. Stopping CBG monitoring altogether may be a reasonable option for some patients. Refer to appendices 1-3 for specific guidance depending on the type of diabetes and estimated prognosis.

7 Counselling with Patient and Family

7.1 Patients, families and carers will have often spent many years striving for tight glycaemic control in an attempt to reduce the risk of long-term complications. They may find it difficult to understand that when the end of life is approaching, maintenance of strict normoglycaemia, aggressive blood pressure and lipid management, and strict dietary restriction can become detrimental to quality of life. Avoidance of long-term complications becomes an irrelevant goal. They will require sensitive counselling from their clinicians to explain the shift in glycaemic goals.

8 Optimisation of insulin delivery

8.1 Insulin delivery pens may need to be reassessed if the physical capabilities of patient alters or carers/family becomes involved in insulin delivery.

8.2 Similarly, any change to the insulin regimen should be implemented near the beginning of the week if at all possible.

8.3 If there is isolated hyperglycaemia avoid stat doses of short acting insulins such as Novorapid (see 5.2 above). Instead, explore reasons for hyperglycaemia (consider DDDISH)

- Have the **D**rugs of diabetes been modified (ie insulin reduced or metformin stopped)?
- Have **D**rugs causing diabetes been introduced (steroids, immunosuppressives, check point inhibitors)
- Has the **D**iet been altered (nutritional supplements)?
- **I**nfection
- **S**tress hyperglycaemia
- **H**bA1c

8.4 If there is a persistent trend, maintenance therapy should be reviewed. Appendices 1-3 give further detail on how to up titrate medications depending on the type of diabetes and estimated prognosis of the patient. A DSN can be involved if required.

9 Diabetic Ketoacidosis (DKA) [6,7,8]

9.1 Usually presents following 2-3 day history of decline with polyuria, polydipsia, lethargy, anorexia, hyperventilation, ketotic breath, vomiting, and coma. But presentation can be even quicker than that. Precipitants include infection, non-compliance, and incorrect insulin dose.

9.2 This is a medical emergency and full active treatment would include IV rehydration with close monitoring and replacement of potassium. A fixed rate insulin infusion would also be considered routine. Refer to your local trust DKA protocol.

9.3 Diagnosis requires ketosis and acidosis and hence, we would be only able to presume such a diagnosis in the hospice/community setting. CBG usually >20mmol/L, but can be as low as 12mmol/L [8]

9.4 If a palliative patient in the hospice/community is suspected of suffering from this condition then referral to acute medical services should be considered and if the patient is conscious, discussion with them and/or relatives, regarding transfer should take place.

9.5 If it is felt inappropriate to transfer the patient or the patient/relatives make an informed decision not to transfer to the hospital, an

adapted regimen for rehydration (according to individual patient) and administration of a subcutaneous insulin regimen could be considered in the hospice setting, according to the needs of each patient. Sliding scales are no longer recommended in this circumstance. Discussion with DSN would be helpful in formulating a regimen in this rare circumstance.

10 Hyperglycaemic Hyperosmolar State (HHS) [6,9]

10.1 There is usually a 5-7 day history of decline with decreasing consciousness, focal neurological signs, marked dehydration and BG >35mmol/L. Blood osmolality would be high on testing, >340mmol/L [5].

10.2 This is a medical emergency and full active treatment would consist of IV rehydration and anticoagulation due to high risk DVT. Insulin therapy is often, but not always, needed. See your local trust HHS protocol [5].

10.3 If a palliative patient in the hospice/community is suspected of suffering from this condition, then referral to acute medical services should be considered and if patients are conscious, discussion with them and/or relative, regarding their transfer should take place.

10.4 If it is felt inappropriate to transfer the patient or the patient/relatives make an informed decision not to transfer to the hospital, an adapted regimen of rehydration could be considered in the hospice setting, according to the needs of each patient. Further advice from DSN may inform any SC insulin therapy options within the hospice.

11 Detailed management – appendices 1-3

11.1 Existing palliative care guidance for diabetes management at the end of life defines the approach depending on the prognosis of the patient. The guidance within this document (appendices 1-3) gives specific guidance with prognoses of months, weeks to days, and last days of life.

11.2 Within the hospice in patient unit setting, these forms can be held in the drug chart folder, to act as a prompt on managing the changing needs of a palliative diabetic patient. In this situation they can be used in conjunction with appendix 9 on the reverse of the form, to act as both guidance and a place to record the CBG regimen and readings. However, for hospital and community teams, these forms can simply act as a reference for some steps to consider when caring for these patients.

11.3 If it is felt that the patient has years to live, treatment would usually remain within national guidelines [1] and glycaemic targets should be individualised and potentially relaxed. Diabetes care should be provided by the primary or secondary care team, depending on patient need.

12 Type 1 Diabetes [1,3,6]

12.1.1 Type 1 diabetes accounts for 5-10% of diabetes. It is a disease of absolute insulin deficiency; therefore insulin withdrawal is likely to lead to death. Unless a patient is imminently dying, we would recommend the continuation of insulin, with the regimen simplified wherever possible, unless the patient specifies otherwise. CBG monitoring can also be reduced according to prognosis.

12.2 If a mentally competent patient requests withdrawal of their insulin, this should be respected but clinicians should first ensure that the patient is aware of the full implications. If such a request is because of undesirable symptoms such as pain and sickness, we should treat these and involve the palliative care team if not done so already. **Once the clinician has stopped the insulin either during the terminal phase of life or at the request of a mentally competent patient, CBG monitoring should cease as well.**

12.3 The types of commonly used insulin are listed in appendix 6.

12.4 Please see Appendix 1 for detail on how to manage palliative patients with type 1 diabetes.

13 Type 2 Diabetes

13.1 Type 2 diabetes accounts for 90% of diabetes. It occurs when there is resistance to the insulin being produced by the pancreas. It can initially be managed with dietary modification and/or oral anti-diabetic drugs. However, overtime the beta cells of the pancreas struggle to produce enough insulin and 20% of type 2 diabetics go on to develop insulin deficiency. Patients with type 2 diabetes can develop HHS or DKA.

13.2 The mechanism of action, dose range, efficacy and elimination route of non-insulin antidiabetic drugs for T2DM are listed in Appendix 5.

13.3 The types of commonly used insulin are listed in appendix 6

13.4 Refer to the following appendices (2 and 3) for detail on how to manage patients with T2DM in the last stages of life. Given the

various ways in which T2DM can be treated, the detailed guidance is divided into:

13.5 ***T2DM: diet or taking non-insulin anti diabetic drugs –*** see Appendix 2[1,3,6]

13.6 Gliclazide can be commenced at 40mg OD and be increased by 40mg every 3 days until the target CBG is achieved. Initially up titrate to 240mg in the morning and, if CBG still not in range, add 80mg in the evening [1].

13.7 Remember to check renal function before commencing/up titrating gliclazide to avoid prolonged hypoglycaemic episodes.

13.8 Metformin should be withdrawn if the creatinine is >150. Additionally metformin can cause unpleasant side effects and a low threshold for withdrawal should be considered if there is any concern regarding side effects (nausea, heartburn, diarrhoea, flatulence) [1]

AND

13.9 ***T2DM: on insulin –*** see Appendix 3 [1,3,6]

13.10 Insulin provides rapid, effective and more predictable control, is easier to titrate, and has less risk of hypoglycaemia compared with some oral hypoglycaemics [10]. It is a better choice in patients with [11]

- A short prognosis (<3 months)
- Poor or erratic oral nutritional intake
- Contra-indications to the use of oral hypoglycaemics
- Severe symptoms from hyperglycaemia

13.11 Where the guidance refers to once daily insulin, the choice would be a long acting (LA) insulin, eg lantus/levemir.

13.12 See appendix 7 for guidance on how to switch to a LA insulin from a pre-mixed or basal bolus regimen

14 Steroid Induced Diabetes/Hyperglycaemia

14.1 The use of steroids in high doses is common in advanced cancer. These agents have a direct hyperglycaemic effect which starts

very early after ingestion, and can also increase appetite.

14.2 Steroids can cause 'steroid induced hyperglycaemia' in known diabetic patients. One in ten non-diabetics on steroids develop 'steroid induced diabetes[12,14]. The hyperglycaemia in both conditions is dose dependent[13,14]

14.3 Steroids cause an increase in blood sugar levels 4-8 hours after ingestion. This leads to peak blood glucose levels between lunch and dinner[2,14]. In the hospice setting we recommend testing CBGs pre-dinner to look for the resultant high CBGs, but they can be checked any time between lunch and evening meal.

14.4 if a patient takes steroids twice daily, the resulting hyperglycaemia is more likely to persist and be more consistent throughout the day and night[2,14], although this can be hard to predict. In this situation, increasing CBG monitoring to TDS or QDS may be advisable.

14.5 However, given that there is no pharmacological benefit to splitting the dose of steroid (unless there is adrenal insufficiency) the recommendation is to give the full dose of steroid between once daily in the morning between 6am and 10am. This will minimise side effects, not only prolonged hyperglycaemia, but also HPA-axis suppression and insomnia.

14.6 Appendices 1, 2 and 3 all have a foot note to encourage clinicians to consider the effect of steroids on diabetes control.

14.7 When faced with steroid induced hyperglycaemia in a known diabetic, uptitrating the existing regimen is a good place to start, taking into consideration when the hyperglycaemia is likely to be most problematic.

14.8 When faced with a new diagnosis of steroid induced diabetes, the options are gliclazide or insulin[1,2,3,6,14]. Once the choice of drug is made, refer to appendix 2 and 3 for suggested uptitration regimens.

14.9 Initiating treatment for steroid induced diabetes[1,2]

	Gliclazide	**Insulin**
Once Daily Steroid	Once daily dose Uptitrate as per appendix 2	Once daily intermediate insulin (see appendix 6)

Twice Daily Steroid	Not advisable OD regimen unlikely to be effective BD regimen leads to an increased risk of night time hypoglycaemia	Once daily long acting insulin given in the morning BD intermediate insulin leads to an increased risk of night time hypoglycaemia

14.10 It is recognised that patients often move between OD and BD steroid regimens throughout an admission. Switching between various insulins based on the frequency of the steroid prescription could lead to confusion and complexity. It is advisable to respond to the pattern of hyperglycaemia observed rather than pre-emptively changing diabetic regimens based on the frequency of steroids. This illustrates the importance of increasing the frequency of CBG monitoring when on steroids. Contact the DSN for more support and guidance if needed.

14.11 *Considerations if the patient is already on a more complex regimen*

-If the patient is on a **twice daily insulin regimen**, an increase the morning dose (10-20%) will be required to treat any pre-dinner hyperglycaemia. However, if on twice daily steroids, there may be some benefit in increasing the evening dose additionally (although caution may be required regarding night time hypoglycaemia)[1]. Aim to increase every 2-3 days until the CBG is within target range and increase CBG monitoring up to a maximum of QDS until more stable[2,3]. Contact DSN for more support and guidance in this complex scenario.

- If the patient is on a **basal bolus regimen**, an increase (10-20%) in the rapid acting insulin dose at breakfast and lunch may be required to treat the pre-dinner hyperglycaemia. The basal insulin may also need to be increased due to any persistent hyperglycaemia[2]. Aim to increase every 2-3 days until the CBG is within the target range and increase CBG monitoring up to a maximum of QDS until more stable[2,3]. Contact the DSN for more support and guidance in this complex scenario.

15 Identifying steroid induced diabetes

15.1 The existing guidance for how best to screen for steroid induced diabetes is variable[1,2,3,6,14]. Pragmatically, the method used will depend on the care setting.

15.2 Guidance from Joint British Diabetes Societies[2] produced guidance for hospital inpatients which suggests first identifying those who are at high risk of developing steroid induced diabetes based on patient characteristics and venous blood sampling. The results inform risk stratification with various monitoring arms using CBGs. The guidance is for use for hospital in patients, encouraging tight glycaemic control and is logistically difficult to apply to a hospice or community setting.

15.3 A further guideline from Joint British Diabetes Societies and the UK Chemotherapy Board offers guidance for commencement of steroid therapy in the outpatient setting[14]. This focuses on CBG monitoring at each appointment and offers advice of treatment arms dependent on results obtained.

15.4 This guideline recommends a pragmatic approach in the hospice and community settings, informed by the existing guidance and applied to a palliative population.

15.5 The aim is to capture those who are at risk of developing diabetes, and those who are previously undiagnosed. Commence treatment only in those who have a CBGs of >15 or are symptomatic. See appendix 8 for the flow chart intended for the hospice setting. The flow chart is used whenever steroids are commenced *or* the steroid dose is increased.

15.6 The community setting can be particularly challenging when monitoring for steroid induced diabetes, predominantly due to the resources available and the logistics of who is responsible for testing and responding to results. If a CBG machine is available appendix 8 can be followed. The logistics of who would hold responsibility for the ongoing monitoring would depend on the local service framework and would need to be considered on an individual basis. If CBG testing is not possible for all those on steroids in the community, it may feel appropriate for the patient to have once weekly urine testing, looking for evidence of raised blood sugar. Urinary glucose sticks demonstrate high specificity (99.3) for ruling in patients with diabetes[15], making it a useful test in this population. There exists no information to make a direct comparison of glucose stick results (ie trace, 1+ etc) to CBG values. However, a pragmatic approach supports using a value of 1-2+ as a prompt to consider the development of steroid induced diabetes. In this situation, initiation of blood sugar monitoring with a home CBG machine and monitoring regimen should be considered. As per appendix 8 this would ideally be OD pre dinner, to establish whether anti diabetic medication may be required if

readings >15 persist.

15.7 Steroid induced diabetes and hyperglycaemia is dose dependent[11,13,14]. It is therefore important to keep the steroid dose at the minimum effective.

15.8 When steroid levels are reduced, there may be a risk of hypoglycaemia. Insulin requirements are likely to decline in parallel[11]. Remember to review diabetic regimens when titrating steroids up or down.

16 Vulnerable Populations and Nutrition

16.1 Trend[1] make note of certain clinical scenarios where special considerations may be necessary. These include

- Special populations
    - Care Home Residents
    - Frailty
    - Dementia
    - Cancer
    - Renal disease
- Nutrition
    - Poor swallow/reduced appetite
    - Enteral feeding
- Pumps/flash readers

Please refer to the Trend [1] national document for further exploration of these scenarios.

17 References

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Appendix 1: Type 1 diabetes

Aim to be symptom free, CBG range 6-15. If CBG<4 treat as per hypoglycaemia guidance.

Estimated Prognosis Months:

- Follow patient's normal glucose monitoring and insulin regimen.
  - If CBG <6 consider 20% reduction in basal insulin every three days until within target range
  - If CBG >15 consider 20% increase in basal insulin every three days until within target range
- Check CBG if condition changes or unwell
  - Treat diabetic emergencies as per guideline (see DKA)
- If oral intake reducing, reduce insulin regimen by 20% initially and then by 20% every 3 days until CBG within target range, to avoid hypoglycaemia

Estimated Prognosis weeks to days:

- Aim to avoid hypoglycaemia, limit symptoms of hyperglycaemia (polyuria, thirst, nausea, vomiting, blurred vision) and avoid unnecessary monitoring
- Consider switching insulin to OD regimen if not already done so (Appendix 7)
- Check CBG pre-dinner
  - If <6 reduce insulin by 50%. If remains <6 reduce insulin by 20% daily until within range
  - If >15 increase insulin by 20% initially and then every three days until CBG within range
- Consider reducing insulin levels in presence of reduced PO intake, weight loss, N+V and renal impairment.

Estimated Prognosis: Last days of life

- Stop all rapid acting and pre-mixed insulin and change to long acting insulin at 50% of the pre-existing basal insulin dose (Appendix 7)
- If already on LA insulin reduce by 50%
- Check CBG once daily
  - If 6-15 – continue at current dose and continue to monitor
  - If <6 reduce long acting insulin by 50% and continue to monitor
  - If >15 increase long acting insulin by 20% ad continue to monitor
- If imminently dying and burden of injections and monitoring outweighs benefits, insulin can be stopped after discussion with patient (if possible) and/or family

REMEMBER: when commencing steroids, hyperglycaemia more likely. Consider increasing frequency of CBG monitoring and be aware of likely need to adjust insulin doses. For more information on management of steroid induced hyperglycaemia, see full guidance

Appendix 2: Type 2 diabetes - diet or non-insulin anti diabetic drugs

Aim to be symptom free, CBG range 6-15. If CBG <4 treat as per hypoglycaemia guidance.

Estimated Prognosis Months:

- Only check CBG if unwell or symptoms of hypo/hyperglycaemia. However, if on existing CBG monitoring regimen, can continue.
- If oral intake reducing or variable consider reducing or stopping oral treatment
- Commence treatment for confirmed persistent hyperglycaemia with symptoms
    - Gliclazide 40mg OD, increasing by 40mg increments to a max dose of 360mg/24hr (be split depending on when hyperglycaemia is most problematic) until within range. Monitor pre-dinner CBGs
    - OR Long acting insulin OM 0.2units per kg (or approx. 10 units) and increase by 20% every three days until within range. Monitor pre-dinner CBGs
- Avoid metformin due to SE profile
- If deteriorating renal function, review doses of oral diabetic medications (appendix 5)

Estimated Prognosis weeks to days

Diet/Metformin

- If admission CBG <15 don't recheck unless symptoms of hyperglycaemia. If symptomatic hyperglycaemia identified, treat as above.
- Consider stopping metformin

On oral hypoglycaemic medication:

- Check CBG on admission, looking for hypoglycaemia if rapid decline
    - <6 consider stopping oral hypoglycaemics, or reduce by 50% and continue to monitor pre-dinner CBG, checking more frequently if concern regarding persistent hypoglycaemia.
    - 6-15 monitor pre-dinner CBG
    - >15
        - increase gliclazide by 40mg increments to a max of 320mg/24hrs which can be split to 240mg OM and 80mg ON.
        - OR consider commencing long acting insulin OM at 0.2units per kg (or approx. 10 units) and increase by 20% every 3 days until within range. Monitor pre-dinner CBGs
- No indication to treat high CBGs with rapid acting insulin. Instead either recommence or uptitrate hypoglycaemics/insulin. If concern regarding HHS, refer to full guidelines.

Estimated Prognosis: Last days of life

- Stop all oral diabetic medication if not already done so
- Stop all CBG monitoring

REMEMBER: if commencing steroids, hyperglycaemia is more likely to occur and uptitration of oral hyperglycaemics or switching to insulin may be necessary to maintain symptom control. Consider increasing CBG monitoring until CBGs are more stable. *For more information on managing steroid induced diabetes, see full guideline.*

Appendix 3: Type 2 diabetes - on insulin

Aim to be symptom free, CBG range 6-15. If CBG <4 treat as per hypoglycaemia guidance

Estimated Prognosis Months:

- Follow patients usual blood monitoring regime
  - If CBG <6 consider withholding basal insulin for next 24-48 hours and reassess, or consider 50% reduction in basal insulin every three days until within target range
  - If CBG >15 consider 20% increase in basal insulin every three days until within target range
- If oral intake reducing/variable consider adjusting regimen
- Consider switching insulin to once daily regimen for simplicity (Appendix 7)
- If acutely unwell check CBG and treat diabetic emergencies as per guideline.

Estimated Prognosis weeks to days:

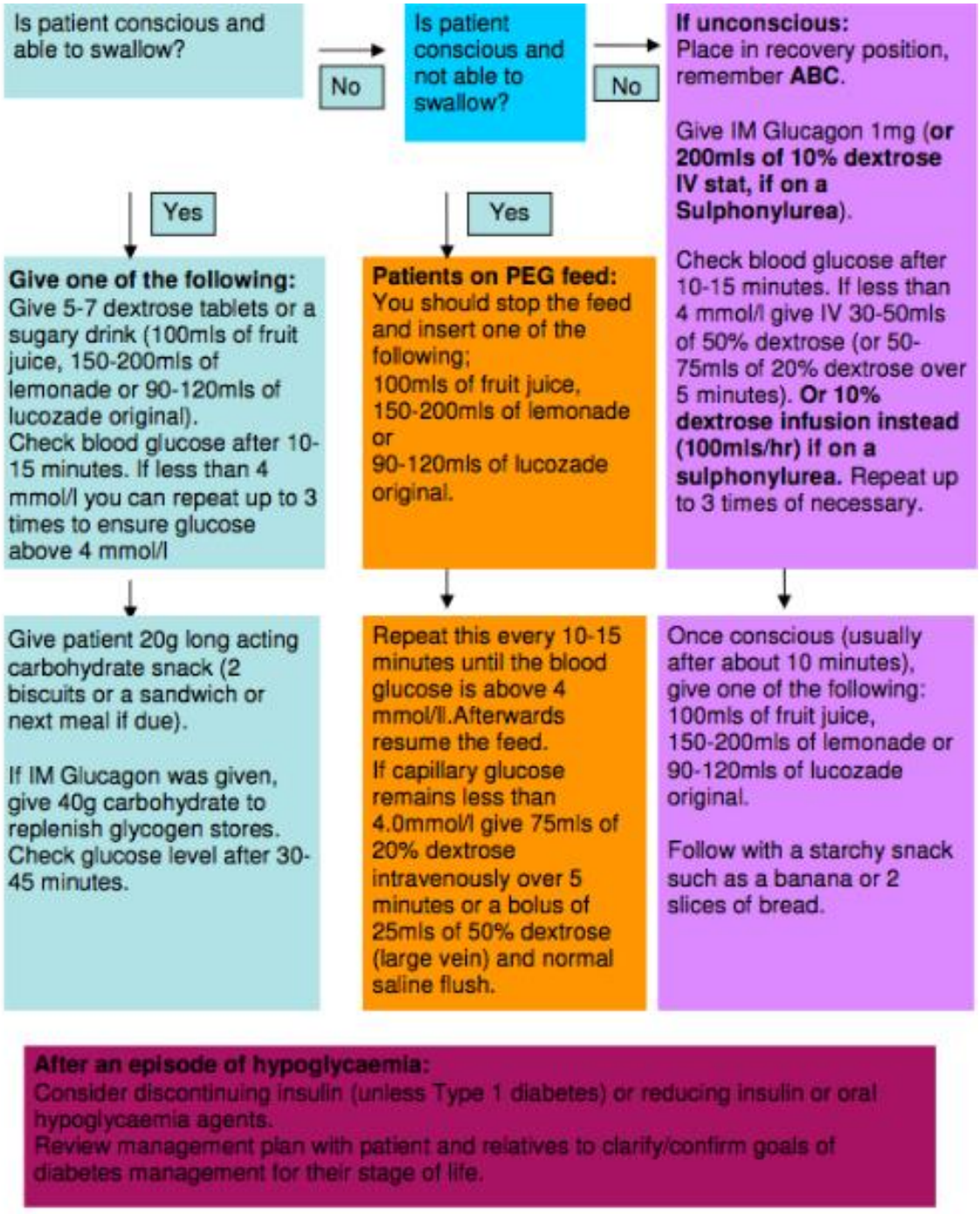
- Aim to avoid hypoglycaemia, limit symptoms of hyperglycaemia (polyuria, thirst, nausea, vomiting, blurred vision), and avoid unnecessary monitoring
- Consider switching insulin to OD regimen if not already done so, especially if CBGs running low (appendix 7)
- Check CBG on admission, looking for hypoglycaemia if rapid decline
  - If <6 consider withholding basal insulin for next 24-48 hours and reassess, or reduce by 50%, until CBG in target range. Monitor pre dinner CBGs
  - If 6-15 monitor pre dinner CBGs
  - If >15 increase basal insulin by 20% every 3 days until within target range. Monitor pre dinner CBGs
- Consider reducing insulin levels in presence of reduced PO intake, weight loss, N+V and renal impairment.

Estimated Prognosis: last days of life

- Stop insulin after discussion with patient (if possible) and/or family
- Stop all CBG monitoring

REMEMBER: if commencing steroids, hyperglycaemia is more likely to occur and uptitration of insulin may be necessary to maintain symptom control.
When using long acting insulin in presence of steroids, consider increasing CBG monitoring to BD until CBGs more stable, as there is a small risk of early morning hypoglycaemia. *See full guidance for more information on steroid induced diabetes and the management options.*

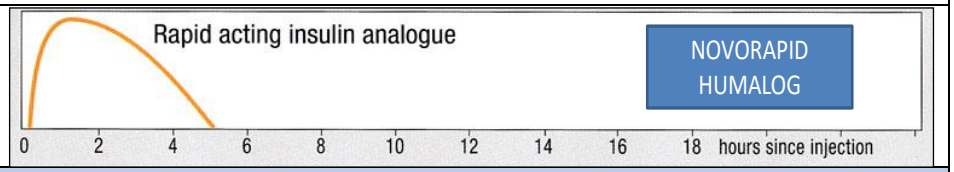
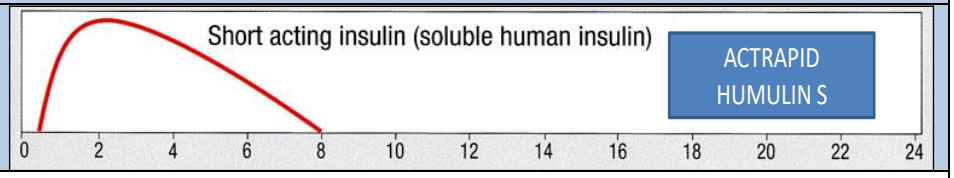
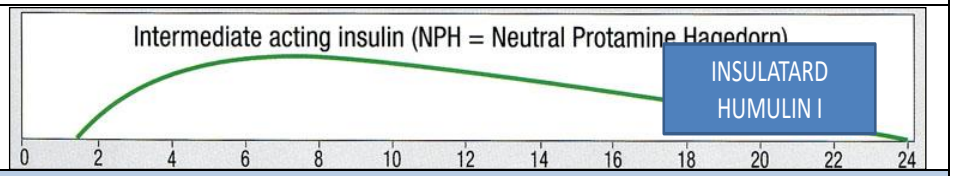
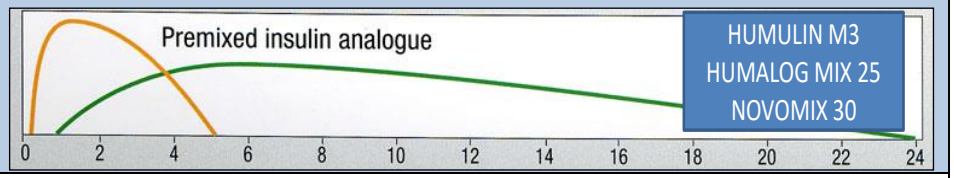
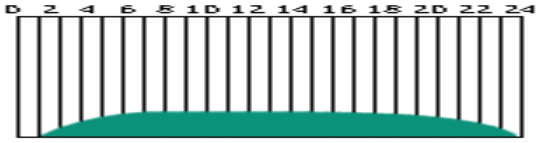
Appendix 4: Hypoglycaemia Guidelines



Appendix 5. Non-insulin anti diabetic medications[3]

Class of Agent	Names	Mechanism of Action	Starting Dose	Max Dose	Main Elimination Route	Precautions
Biguanides	Metformin	↓insulin resistance ↓ hepatic glucose output ↑ peripheral glucose utilisation ↑glucose turnover between intestine and liver	500mg bd	1g bd	Renal	GI intolerance Lactic acidosis (rare) Renal impairment, any hypoglycaemic condition
Sulphonylureas	Gliclazide Gliclazide SR	Directly ↑insulin secretion Binds to SUR1 - stimulates β-cells by closure of K+-ATP channels	40mg od 30mg od	320mg/24hrs 120mg od	Renal 60%	Hypoglycaemia Selection restricted by severe liver or renal disease, or porphyria
Meglitinides	Nateglinide Repaglinide	Directly ↑ insulin secretion Binds to benzamido site on SUR1 - stimulates β-cells by closure of K+-ATP channels Rapid onset, short duration of action	60 mg with each meal 0.5g with meals	540 mg 16g	Hepatic Hepatic	Lesser risk of hypoglycaemia (fewer and less severe than with sulphonylureas) Liver or severe renal disease
Gliptins (DPP-4 inhibitors)	Sitagliptin Linagliptin	↑ insulin secretion Inhibition of DPP-4 allows increased t½ for incretins, which potentiate nutrient-induced insulin secretion	50mg od 5mg od	100mg od	Renal Faecally excreted	Small risk of hypoglycaemia (seldom severe), mostly when used with other glucose-lowering agents Substantial renal or liver disease
Glitazones	Pioglitazone	↑ insulin action Stimulate PPARγ ↑adipogenesis Alter glucose-fatty acid cycle	30mg od	45mg od	Hepatic	Heart failure, oedema, fluid retention, anaemia, fractures Cardiac disease, severe liver or renal disease
Alpha-Glucosidase Inhibitors	Acarbose	Inhibiting the digestion of carbohydrates by inhibiting a-glucosidase	50mg od	100mg tds	Renal 35%	The major drawback of acarbose is the fact that it is often associated with a lot of flatulence.
SGLT-2 Inhibitors	Canagliflozin Dapagliflozin Empagliflozin	Reversibly inhibits SGLT-2 to reduce glucose reabsorption and increase urinary glucose excretion	100mg OD 10mg OD 10mg OD	300mg OD 25mg OD	Hepatic and Renal Hepatic and Renal Hepatic and Renal	Risk of DKA, use with caution conditions leading to restricted food intake. Renal impairment.
GLP-1 Receptor Agonists (SC injection)	Albiglutide Exenatide Liraglutide Lixisenatide	↑glucose dependent insulin secretion ↓gastric emptying	30mg weekly 5mcg BD 0.6mg OD 10mcg	50mg weekly 10mcg BD 3mg OD 20mcg	Renal	CCF, pancreatitis, Renal impairment

Appendix 6. Types of Insulin

Trade Name	Source	Delivery System	Taken	Time Action Profile Marked in hourly intervals
Rapid-acting Insulin Analogues				
Novorapid	Analogue	Vial and Syringe 3 ml cartridges in a re-usable pen Pre filled disposable pen system (Flexpen)	Just before / with / just after food	
Short-acting Human Insulins				
Actrapid	Human	Vial and Syringe	For use only in VRIII or FRIII	
Intermediate-acting Human Insulins				
Humulin I	Human	Vial and Syringe 3 ml cartridges in a re-usable pen Pre filled disposable pen system (Kwikpen)	30 mins before food	
Intermediate-acting Biphasic and Rapid-acting Analogue Insulins				
Humalog Mix25	Analogue	3 ml cartridges in a re-usable pen Pre filled disposable pen system (Kwikpen)	Just before / with / just after food	
NovoMix 30	Analogue	3 ml cartridges in a re-usable pen Pre filled disposable pen system (Flexpen)	Just before / with / just after food	
Long-acting Insulin Analogues				
Lantus Levemir	Analogue	Vial and Syringe 3 ml cartridges in a re-usable pen Pre filled disposable pen system (Solostar)	Once a day anytime but at the same time each day	 **No peak of action**

Appendix 7[3]:

Conversion of twice daily mixed insulin to once daily long-acting insulin:

Pre-mixed insulin has a proportion of intermediate-acting insulin in it. For example Novomix 30 has 70% intermediate-acting insulin. Humalog 25 has 75% intermediate-acting insulin. Calculate the total daily amount of intermediate-acting insulin and give 80%of this amount as LA insulin analogue such as Lantus once daily. If blood glucose is low i.e. 4.0- 6.0 mmol/L, use 50% of the original intermediate-acting dose.

For example

If the patient normally takes 20 units BD Novomix30, this totals 40 units of insulin in 24 hours as it is a BD preparation. This is a mixture of intermediate and short acting insulin. 70% of Novomix30 is intermediate-acting, so the patient takes 28 units of intermediate-acting insulin daily (0.70 x 40 units).

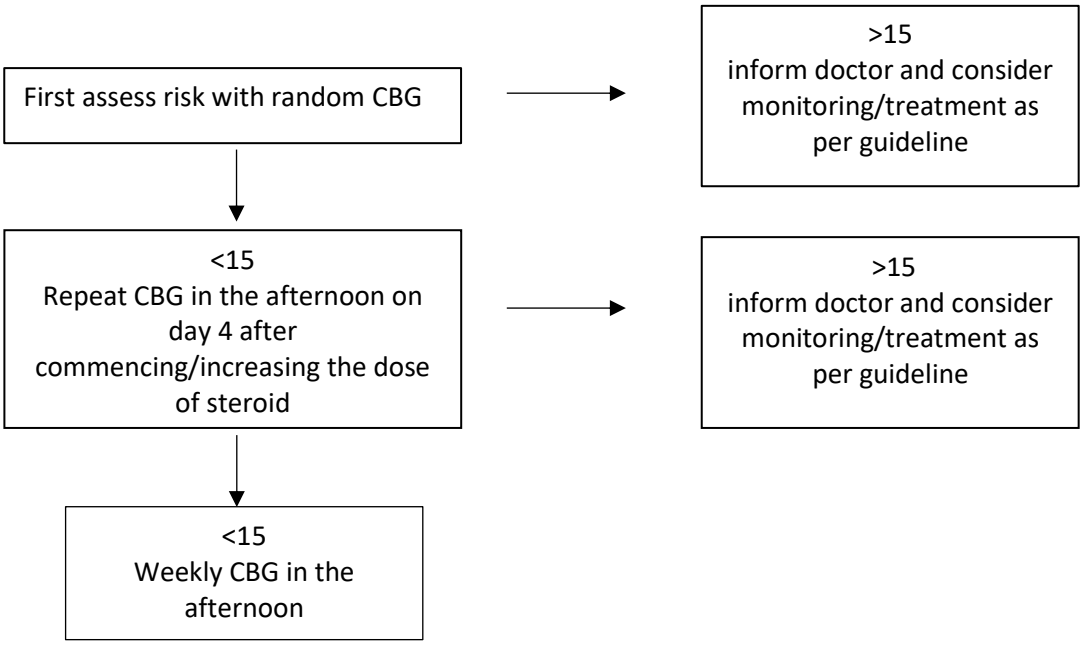
To calculate the new dose, we want 80% of the existing intermediate-acting daily insulin dose, which is 22 units (0.80 x 28 units = 22 units). If hypoglycaemic we need to give 50% of the daily intermediate-acting insulin dose. This equals 14 units (0.50 x 28 units = 14 units)

Appendix 8: Steroid initiation/monitoring form

Name:
DoB

Date	Dose of steroid	Indication	Review date	Comments

<u>If starting/increasing steroids or admitted already on steroids</u>



Remember…

- Consider treatment as per T2DM if CBG >15 using clinical judgement and reference to full guidance.
- If increasing dose of steroids then need to reassess from the top of the flow chart with a random CBG as the risk of steroid induced diabetes is dose dependant.
- Consider that if the dose of dexamethasone is reduced, and the patient is on antidiabetic medication, the dose may need to be reduced.
- Always try to keep the patient on the minimum possible steroid dose.
- Doctor to note on drug chart when 'Day 4' CBG needs to be tested after initiating or changing dose of dexamethasone.
- Consider treatment as per T2DM if CBG >15 but use clinical judgement and refer to full guidance

Monitoring CBG when taking steroids

Name:
DoB

Test in the afternoon.
If CBG >15 inform a doctor

Date	Pre dinner CBG	Comments
		Admission random CBG

Appendix 9: Diabetes monitoring chart for inpatients

Name: DoB

Estimated Prognosis	Date	Sig
Months		
Weeks		
Days		

CBG monitoring regime to be used

Random daily	Pre-breakfast	Pre-lunch	Pre-evening meal	Bed	Date/sign

Date	Pre breakfast CBG	Pre Lunch CBG	Pre evening meal CBG	Bed CBG	Comments
					Admission