RC PSYCH
ROYAL COLLEGE OF PSYCHIATRISTS

Guidance on Recognising and Managing Medical Emergencies in Eating Disorders

(Replacing MARSIPAN and Junior MARSIPAN)

Annexe 3: Type 1 diabetes and eating disorders (T1DE)

May 2022

The Royal College of Psychiatrists' Medical Emergencies in Eating Disorders: Guidance on Recognition and Management has been endorsed by the Council of the Academy of Medical Royal Colleges, which represents all the Medical Royal Colleges and Faculties in the UK.

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Editors

Simon Chapman, Consultant Paediatrician, King's College Hospital and South London and the Maudsley NHS Foundation Trusts

Helen Partridge, University Hospitals Dorset NHS Foundation Trust

Claire Pinder, Eating Disorders Dietitian, Dorset HealthCare University NHS Foundation Trust

Marietta Stadler, King's College Hospital NHS Foundation Trust

Contributors

Sarah Alicea, Children and Young People's Diabetes Specialist Dietitian, University Hospitals Dorset NHS Foundation Trust

Michelle Bennett, Clinical Specialist in Eating Disorders, Dorset HealthCare University NHS Foundation Trust

Dr Sarah Brewster, Diabetes and Endocrinology Registrar/Clinical Academic Fellow, Southern Health NHS Foundation Trust

Caroline Cross, ComPASSION Project Manager, Dorset HealthCare University NHS Foundation Trust

Dr Carla Figueiredo, Consultant Psychiatrist; Dorset HealthCare University NHS Foundation Trust

Linda Gerrard-Longworth, Eating Disorders Therapist, Southern Health NHS Foundation Trust

Kerri Hampton, Diabetes Nurse Specialist, Dorset County Hospital NHS Foundation Trust

Dr Eveleigh Nicholson, Consultant in Diabetes and Endocrinology, Portsmouth Hospitals University NHS Trust

Dr Helen Partridge, Consultant in Diabetes, University Hospitals Dorset NHS Foundation Trust

Simone Penfold, Diabetes Specialist Nurse, University Hospitals Dorset NHS Foundation Trust

Claire Pinder, Eating Disorders Dietitian, Dorset HealthCare University NHS Foundation Trust

Dr Lindsey Rouse, Clinical Psychologist, University Hospitals Dorset NHS Foundation Trust

Jacqueline Ryder, Diabetes Specialist Nurse, University Hospitals Dorset NHS Foundation Trust

Nicola Stacey, Diabetes Specialist Nurse, University Hospitals Dorset NHS Foundation Trust

Ariella Thompson, PhD Student, Bournemouth University

Dr Sebastian Zaidman, Dorset HealthCare University NHS Foundation Trust

Reviewers

Jacqueline Allan, Patient Public Involvement representative; DClinPsych Trainee at South London and the Maudsley Hospitals NHS Foundation Trust

Andrea Brown, Consultant Psychiatrist, Schoen Clinic, London

Helen Cope, Liaison Psychiatrist, Guy's and St Thomas NHS Foundation Trust

Sophie Harris, Consultant Diabetologist, King's College Hospital NHS Foundation Trust

Khalida Ismail, Professor of Psychiatry, King's College London

Dulmini Kariyawasam, Consultant Diabetologist, Guy's and St Thomas NHS Foundation Trust

Yuk-Fun Liu, Consultant Diabetologist, Guy's and St Thomas NHS Foundation Trust

Ruth Marshall, Child and Adolescent Psychiatrist, Central Manchester CAMHS

Contents

List of boxes and tables

1. Introduction

Interaction between elements of living with type 1 diabetes and the biological, psychological and social predisposing factors to developing an eating disorder can precipitate the development of an eating disorder in the context of type 1 diabetes[1].

1.a. Presentations

Type 1 diabetes and disordered eating (T1DE) refers to a range of presentations in those with a diagnosis of type 1 diabetes that use one or more of a range of behaviours to control their weight. These include omission or restriction of insulin, restriction of food, over-exercise, self-induced vomiting, and laxative or diuretic misuse. Thyroid hormones or diabetes medication believed to reduce body weight may also be used. Insulin over-injection, either to cover binge eating or out of fear that high blood-glucose levels may cause long-term complications, have been observed. Insulin restriction puts patients at higher risk of the short- and long-term complications of diabetes; insulin over-injection bears the risk of severe hypoglycaemia and loss of awareness of hypoglycaemia. Studies report that insulin restriction is associated with a threefold increase in mortality[2] and a reduced quality of life compared to those with type 1 diabetes who do not restrict insulin[3,4].

1.b. Diagnostic criteria

There is no consensus on how best to define this patient group. Diagnostic criteria are proposed in Box 1. Most patients are of a normal weight, with a body mass index (BMI) of 18.5–25 kg/m^2 in adults, or above 85% median BMI (mBMI) in children and young people aged under 18 (this would need to be adjusted in ethnic minority populations). While some patients omit insulin, others restrict their dietary intake of carbohydrates but use insulin appropriately. As a result, BMI/%mBMI and haemoglobin A1C (HbA1c) cannot be used exclusively in the identification or diagnosis of patients with T1DE. It is also important to recognise that other diabetes-related factors (such as diabetes distress, depression, anxiety, needle phobia, acceptance of diabetes diagnosis, fear of complications, and fear of hypo- or hyperglycaemia) may lead to diabetes-related behaviours that can be mistaken for T1DE. This is because presentation may include indicators associated with T1DE, such as raised HbA1c, weight loss and disengagement from services. A careful history is therefore required to identify if weight and shape concerns are part of the psychopathology, to make a differential diagnosis and ensure an appropriate treatment pathway.

Box 1: Proposed diagnostic criteria for T1DE

People with type 1 diabetes who present with all three criteria:

1. Intense fear of gaining weight, or body image concerns, or fear of insulin promoting weight gain.
2. Recurrent inappropriate direct or indirect* restriction of insulin (and/or other compensatory behaviour**) to prevent weight gain.
3. Presenting with a degree of insulin restriction, eating or compensatory behaviours that cause at least one of the following:
    - harm to health
    - clinically significant diabetes distress
    - impairment on daily functioning.

* Indirect restriction of insulin refers to reduced insulin need/use due to significant carbohydrate restriction.
** Dietary restriction, self-induced vomiting, laxative use, excessive exercise, over-use of thyroid hormones, over-use of diabetes medication believed to avoid weight gain or promote weight loss.

1.c. Red flags to indicate disordered eating in patients with type 1 diabetes

People with T1DE can be fearful of judgement by healthcare professionals, family and friends, and can experience considerable shame and guilt. Sometimes this can be rooted in past adverse experience with healthcare delivery related to their diabetes[5]. This cohort of patients may therefore disguise the difficulties they are experiencing or disengage with services or health care professionals. As a result, T1DE can be easily overlooked. It is recommended that staff in a range of healthcare settings (including diabetes clinics, ophthalmology, primary care, emergency departments, acute medical units, medical and paediatric wards, intensive care units, dental clinics and liaison psychiatry services) are alert to the potential red flags for T1DE (Box 2) and feel confident to initiate a supportive, non-judgemental conversation with the person, or, in the case of children and young people, also with their parents or teachers, to explore the possibility of T1DE being present.

Where there is concern, it is important to recognise that this cohort require ongoing treatment. Staff identifying someone at risk of T1DE should therefore refer to psychiatric liaison or make links with local eating disorder, mental health or diabetes teams to discuss their concerns.

Box 2: Red flags for T1DE

Any one of the following:

Biochemical

- Increase in HbA1c above 86mmol/mol or erratic blood glucose levels (e.g. high glycaemic variability, postprandial hyperglycaemia following bolus omission).
- Multiple emergency department or ward admissions with hyperglycaemia, diabetic ketoacidosis (DKA).
- Recurrent ketonaemia (>3mmol/L) – may have compensated metabolic acidosis.
- Recurrent severe hypoglycaemia (two or more episodes over 24 months).

Beliefs, behaviours and functioning

- Over-exercising.
- Impaired awareness of hypoglycaemia.
- Extreme dietary restriction or binge eating.
- Weight loss history (weight loss in line with Medical Emergencies in Eating Disorders guidance criteria) or fear of weight gain.
- Body image concerns.
- History of eating disorder diagnosis.
- Diabetes distress.
- Fear of hypoglycaemia.
- Mental health comorbidity (e.g. depression, generalised anxiety disorder).

Relationships

- Secrecy about diabetes management, failure to request regular prescriptions, disengagement from diabetes services
- Poor school/work performance/attendance
- Conflict at home around meals and eating/diabetes management

1.d. A shared-care model

Several factors will have historically resulted in the exclusion of this cohort from the support and expert treatment that they require. Both eating disorders and type 1 diabetes are specialist areas of medical and psychological expertise. Where T1DE is suspected or identified, it is essential that expertise is drawn from both specialities to inform the treatment offered. Good practice would recommend a shared-care model that provides continuity of care and a unified team approach with joint care plans[6]. Case discussion, professional shadowing, peer-to-peer learning, joint appointments (face-to-face or virtual), development of referral routes, shared treatment pathways, case conferences involving the service user, multidisciplinary team meetings and peer supervision all have the potential to inform improved care.

1.e. Acute hospital settings

In acute hospital settings, input in adults from the diabetes team alongside the treating physician is recommended. Or, in children and young people, by the paediatric diabetes team, as well as referral to psychiatric liaison within reach from local eating disorder services as needed:

- Registered mental health nurse (RMN): to support nursing care plan including the management of compensatory behaviours (including appropriate management of insulin administration, advice on level of observations, requirement for one-to-one support and appropriate restraint procedures).
- RMN/mental health support worker: to provide one-to-one support, and appropriate restraint if required.
- Eating disorders dietitian: liaison with the treating dietitians over the dietetic care plan.
- Consultant psychiatrist: liaison with the treating medical or paediatric team, advice regarding assessment of capacity, Mental Health Act assessment and advice on level of observations. If any degree of restraint is required, this would need to be discussed and advice given around the appropriate legal safeguards.

1.f. Specialist eating disorder unit settings

In specialist eating disorder units, care should be supported by the local diabetes team. Physical health assessment and medical stabilisation (if indicated, e.g. severe electrolyte disturbance, metabolic acidosis, dehydration) should be achieved prior to admission to the eating disorder unit in an acute medical setting:

- Adult or paediatric diabetes consultant:
    - advice on the insulin management plan and the safe management of hyperglycaemia, hypoglycaemia and ketones
    - advice on modified rehydration/ re-insulinisation protocols for not too rapid improvement of glycaemia to avoid too rapid weight gain and acute neuropathic complications.
- Adult or paediatric diabetes specialist nurse:
    - to support the RMNs with the implementation of the advised insulin plan and practical aspects of diabetes management
    - Initiating additional physical health investigations (bone health, endocrine disorders, autonomic neuropathy assessment) as appropriate.

2. Table for risk assessment in people with T1DE

The assessment of risk in people with T1DE will mirror those with other eating disorder presentations with some additional considerations. Table 1 shows the parameters, risks and links to relevant places in this document, including those that mirror more general eating disorder presentations that have been described in the main guidance document. See Table 2 for strategies for insulin omission reduction.

General principles for assessment and treatment of this patient group are discussed, which can be adapted to a range of healthcare settings including primary care, outpatient diabetes and eating disorder services, acute inpatient settings and specialist eating disorder units and beds (SEDUs and SEDBs). Additionally, clinicians should confirm that annual diabetes review checks are up to date, to include[7–9]:

- HbA1c measurement
- blood pressure measurement
- smoking status
- weight and BMI measurements, including %mBMI in those under 18 years
- albumin: creatinine ratio
- full lipid profile (including high-density lipoprotein and low-density lipoprotein, cholesterol and triglycerides)
- electrolytes, estimated glomerular filtration rate (or measured glomerular filtration rate if extremely underweight)
- thyroid hormone status
- liver enzymes
- electrocardiogram
- retinopathy screening
- foot health check
- Consider screening for cortisol deficiency and growth hormone deficiency (e.g., in case of problematic hypoglycaemia)
- Consider screening for coeliac disease (e.g. if underweight, or gastrointestinal symptoms)
- Pre-pregnancy counselling/contraception counselling.

Table 1: Risk assessment for T1DE

Parameter	Risks	Relevant links within this document
BMI/starvation (e.g., temperature, Sit-Up–Squat Stand [SUSS] Test)	**As for those in Medical Emergencies in Eating Disorders: Guidance on Recognition and Management (referred to as the Guidance)** **Additional considerations for all levels of risk:** Most T1DE patients fall within the low or moderate risk for BMI. This should not mask clinician concern because severity of overall risk lies in the consequences of insulin omission. Cellular starvation will be present in the omission of insulin regardless of BMI. Those with diabetes and cardiac autonomic dysfunction may have a resting tachycardia, which may mask severity of starvation. Insulin administration is the driver for refeeding syndrome in T1DE. Considerations for insulin during nasogastric tube feeding in T1DE.	1.b. Diagnostic criteria 3.a. BMI and starvation 3.b. Re-feeding/re-insulinisation 3.b. Re-feeding/re-insulinisation Appendix A: Example re-insulinisation and refeeding protocol…
Cardiovascular health/ electrocardiogram	**As for those in the Guidance** **Additional considerations at all levels of risk:** Assessment of long-term macrovascular complications of type 1 diabetes should form part of medical assessment in T1DE.	3.g. Biochemical risks

Parameter	Risks	Relevant links within this document
Hydration and oedema	**As for those in main Guidance**	
	Additional considerations at all levels of risk:	
	Insulin restriction/omission increases risk of dehydration as a result of hyperglycaemia/ketones.	3.d. Hydration/oedema
	Assessment of hydration status should therefore form part of the medical assessment in T1DE.	
	Reinsulinisation can be associated with oedema.	3.d. Hydration/oedema
Biochemical	Hyperglycaemia and raised ketones will occur with reduction or omission of insulin.	Appendix B: Inpatient protocols for the management of hyperglycaemia… 3.e.i. Management of raised ketones in T1DE
	Low risk = normal glycaemia.	
	Moderate risk = hyperglycaemia with ketones absent or recurrent episodes of ketones <1.5.	
	High risk = hyperglycaemia with recurrent episodes of ketones >1.5	
	Very high risk = one or more episodes of DKA associated with deliberate insulin omission	
	Hypoglycaemia may occur with carbohydrate restriction or excessive exercise alongside insulin administration.	Appendix B: Inpatient protocols for the management of hyperglycaemia…
	Low risk = infrequent episodes requiring self-management of hypoglycaemia.	
	Moderate risk = frequent episodes requiring self-management of hypoglycaemia.	

Parameter	Risks	Relevant links within this document
	High risk = Infrequent episodes of hypoglycaemia requiring external assistance for treatment.	3.g.ii. Hypoglycaemia and hypoglycaemia unawareness
	Very high risk = severe and recurrent episodes of hypoglycaemia requiring external assistance for treatment.	
	Electrolyte disturbance; as for those in main Guidance	
	Additional considerations at all levels of risk:	
	Assessment of renal function as a long-term complication of type 1 diabetes should form part of the medical assessment in T1DE. Consider the potential for renal impairment to impact on the biochemical picture. When considering renal function consider that urea will be reduced in the presence of starvation.	3.g.iii. Electrolyte imbalance
	Acid-base balance; as for those in main Guidance	
	Insulin omission may further complicate the metabolic picture.	3.g. Biochemical risks
Medication	**Additional considerations at all levels of risk:**	
	Adjunctive therapies; GLP-1[a] agonists and SGLT-2 inhibitors should be used with caution in those with T1DE due the associated weight loss and potential risks of hypoglycaemia and DKA with SGLT-2 inhibitors.	3.h. Medication

[a] Definitions of abbreviations used in this table are in the Abbreviations section.

Parameter	Risks	Relevant links within this document
	Glucagon – will be less effective in the management of hypoglycaemia where glycogen stores are reduced due to insulin omission, carbohydrate restriction or excessive exercise.	3.d. Nasogastric tube feeding
Disordered eating behaviours	**Food restriction in the presence of appropriate insulin administration; as for main Guidance**	
	Additional considerations at all levels of risk:	
	If food restriction is present in the absence or restriction of insulin the risks will be increased beyond those in main Guidance as availability of calories to the body will be further reduced.	3.i.i. Food restriction
	Assessment for the presence of gastroparesis as a long-term complication of type 1 diabetes should form part of the medical assessment in T1DE.	3.i.i. Food restriction
	High risk	
	Be aware of the potential for preferential avoidance of carbohydrate which if insulin is not adjusted increases the risk of hypoglycaemia.	3.i.i. Food restriction
	Be aware of the potential for the over consumption of carbohydrate, in the absence or restriction of insulin, to maintain raised blood glucose and ketone levels.	3.i.i. Food restriction
	Bingeing may result in raised blood glucose and ketone levels unless insulin is appropriately adjusted.	3.i.i. Food restriction
	Person may feel guilt and shame after an episode of bingeing and may overcompensate with a large insulin dose causing hypoglycaemia.	3.i.i. Food restriction
	Be aware of the potential for diabetic gastroparesis to impact on food patterns and eating disorder symptomology.	3.i.i. Food restriction

Parameter	Risks	Relevant links within this document
	Be aware that the delayed gastric emptying associated with diabetes related gastroparesis, can result in hypoglycaemia.	3.i.i. Food restriction
	Very high risk	
	Bingeing in context of pre-existing gastroparesis may lead to gastric dilatation, ischaemia and rupture.	3.i.i. Food restriction
Activity and exercise	**As for those in main Guidance**	
	Additional considerations at all levels of risk:	
	Increased risk of hypoglycaemia unless carbohydrate and/or insulin are adjusted.	3.i.i. Food restriction
	Increased risk of ketosis in those omitting or restricting insulin.	3.i.i. Food restriction
	Further impact on hydration and subsequent risk of DKA in those omitting or restricting insulin.	3.i.i. Food restriction
	Impact on foot health. Assessment of foot health should form part of the medical assessment in T1DE.	3.i.i. Food restriction
Compensatory behaviours	**As for those in main Guidance**	All points link to 3.i.iii. Compensatory behaviours
	Additional considerations at all levels of risk:	Table 2: Strategies for insulin omission/reduction...
	Strategies to disguise insulin omission/reduction.	
	Overconsumption of carbohydrate, and exercise in the absence or restriction of insulin can be used to maintain raised blood glucose and ketone levels.	Appendix C: Inpatient protocols for the management

Parameter	Risks	Relevant links within this document
	Emergence of other eating disorder behaviours as T1DE related compensatory behaviours are addressed.	of refusal of... insulin...
	Vomiting resulting in unpredictable blood glucose levels, further complicated by insulin omission or restriction if this is present.	Appendix D: Inpatient insulin management care plan...
Engagement with management plan	**Low risk** = has insight/motivation to engage in insulin management plan.	
	Moderate risk = some insight/motivation to engage in insulin management plan.	
	High risk = poor insight/motivation to engage in insulin management plan.	3.i.iv. Insulin omission or reduction
	Very high risk = active refusal to engage in/sabotage of insulin management plan.	3.i.iv. Insulin omission or reduction
	Additional considerations at all levels of risk:	
	Be aware that previous negative experiences with health care professionals can impact on engagement.	3.i.iv. Insulin omission or reduction
	Hyperglycaemia and ketones as well as recurrent hypoglycaemia can impair cognition and impact the ability to engage in treatment plans.	
	Be aware of the potential for diabetes-related psychological concerns to impact on treatment progression.	
	Be aware that lack of insight can impact on engagement with treatment.	
	Be aware that family dynamics can impact on treatment progression.	

Parameter	Risks	Relevant links within this document
Other mental health diagnoses	As for those in main Guidance	
	Additional considerations at all levels of risk:	
	Increased risk of depression and anxiety in those with type 1 diabetes compared to the general population.	All points link to 3.i.iv. Insulin omission or reduction
	Consider other diabetes related aspects of mental health including diabetes-related distress, needle phobia, fear of hypoglycaemia, fear of diabetes-related complications.	
Self-harm and suicide	**As for those in main Guidance**	
	Additional considerations at all levels of risk:	
	Suicide occurs more frequently with the coexistence of psychiatric and physical illness.	
	Be aware of insulin as an available and lethal means of harm.	All points link to 3.i.iv. Insulin omission or reduction
Use of the Mental Health Act	**As for those in main Guidance**	
	Additional considerations at all levels of risk:	
	Use of the Mental Health Act in physical illness.	3.i.iv. Insulin omission or reduction
Annual diabetes review checks	**Additional considerations at all levels of risk:**	
	Weight and body mass index measurements	

Parameter	Risks	Relevant links within this document
	blood pressure measurement	
	smoking status	
	HbA1c measurement	
	albumin: creatinine ratio	
	full lipid profile (including high-density and low-density lipoproteins, cholesterol and triglycerides)	
	retinopathy screening	
	foot check.	

3. Additional notes to support Table 1: Risk assessment table for T1DE

3.a. Body mass index and starvation

Risks associated with low BMI/%mBMI and starvation in T1DE will mirror those without type 1 diabetes. It should be noted that those with diabetes and cardiac autonomic dysfunction, or significant dehydration due to glucosuria/ polyuria may have a resting tachycardia which would invalidate heart rate as an indicator for severity of starvation. This is less likely to be a phenomenon seen in children and young people.

Most people with a diagnosis of T1DE fall within low or moderate risk for BMI/%mBMI. However, it is important that this does not mask clinician concern because it is important to recognise that the most acute risks result from metabolic sequelae associated with omission of insulin, where this is present, because these can occur at any weight (see Biochemical risks in this annexe).

3.b. Refeeding/re-insulinisation

Cellular starvation of insulin dependent tissue that is caused by insulin deficiency will occur regardless of weight, BMI or the other markers of starvation. In patients with T1DE, where insulin has been omitted and/or carbohydrate restricted, the reintroduction of insulin alongside carbohydrate will be the key driver for the refeeding syndrome. All those with insulin omission are potentially at risk of electrolyte shifts on re-insulinisation/refeeding. Re-insulinisation is the process of insulin reintroduction, while refeeding describes the reintroduction of food, particularly carbohydrates. Theory would suggest that the degree of risk will be associated with the severity of insulin omission and the rate of insulin reintroduction. Low initial electrolytes, vomiting, laxative, alcohol or drug misuse and infection all increase the risk of refeeding syndrome further in those without type 1 diabetes[10] and should be considered in this patient cohort. In children and young people, risks include hypophosphataemia before re-alimentation, core temperature below 36°C and low %mBMI[11]. Electrolyte shifts (e.g. hypokalaemia or hyponatraemia) will be more pronounced in a person with diabetes than in a person without diabetes, therefore electrolytes have to be monitored closely, with appropriate medical/diabetes/paediatric oversight.

In those with T1DE, who have been omitting insulin, the risk of re-insulinisation/refeeding syndrome can be managed by a gradual introduction of insulin (and carbohydrate if this has been restricted). There are other reasons for a gradual approach which, if ignored, all have the potential to deter the person with T1DE continuing with treatment:

- Building up insulin and food slowly gives time to address psychological concerns associated with this process. Some T1DE patients will find rapid increases in the carbohydrate content of their meal plan distressing. It is important that the patient is able to engage with the meal plan and a daily carbohydrate intake (distributed over three main meals and snacks, excluding hypoglycaemia treatment) of approximately 150g of carbohydrates as a starting point is deemed to be safe, and can be modified on a case-by-case basis.
- It will reduce the risk of insulin-/refeeding-related oedema which although usually medically harmless can have a psychological impact as a result of rapid weight changes.
- It will allow adjustment to post-meal feelings of fullness which can be interpreted as 'being fat'. This can be as a result of delayed gastric emptying which can occur if food restriction has been present or if diabetes-related gastroparesis is present. Another driver for delayed gastric emptying is hyperglycaemia per se, therefore investigations for gastroparesis should be conducted once stabile normoglycaemia has been safely achieved.
- A gradual reduction in blood glucose levels will reduce the risk of complications, i.e. treatment induced retinopathy and neuritis[12,13] (see Biochemical risks in this annexe).
- It will reduce the risk of hypoglycaemia or the frequency of pseudo hypoglycaemia (see Biochemical risks in this annexe).
- Counselling of the T1DE patient on all the above (in particular about the transient nature of the oedema-/fluid retention-related weight gain and about the rationale for a slow titration process) is important at all stages of this process.

While a gradual approach is advised, this needs to be balanced against the risks of DKA (see the section on Biochemical risks) and underfeeding. Any protocol needs to ensure enough insulin is initially provided to ensure ketone production is switched off. The aim should then be initial conservative glucose targets while gradually increasing insulin and food (including carbohydrate) to achieve weight stabilisation or weight gain (if required) and prevent ongoing physical deterioration. The requirement for certain vitamins, in particular thiamine and other B vitamins during the refeeding process can be assumed to mirror those without type 1 diabetes.

An example protocol for re-insulinisation/oral refeeding in T1DE that has so far been found to be safe and acceptable to patients can be found in Appendix A. Where re-insulinisation/refeeding is progressed more rapidly, or if other risk factors for the development of refeeding syndrome are present, such as infection

or abnormal baseline electrolytes, increased frequency of monitoring of electrolytes is advised, based on clinical judgement and reflecting levels that would be provided to those without type 1 diabetes as a minimum.

It should be noted that in atypical presentations of T1DE, where insulin is not omitted and HbA1c is not elevated, carbohydrate reintroduction alongside appropriate doses of insulin to maintain appropriate blood glucose levels would be advocated. Under these circumstances, the rate of carbohydrate introduction needs to be appropriately managed to reduce the risk of electrolyte shifts on refeeding. Standard refeeding protocols used for the management of refeeding syndrome in those who do not have type 1 diabetes would be appropriate to use under these circumstances, alongside matched insulin doses.

3.c. Principles of medical management

There are many factors to consider in the medical management of this patient group, and it is essential to ensure appropriate medical oversight is in place to monitor electrolyte and fluid imbalances, blood glucose and ketones.

- **Electrolyte shifts** (e.g. hypokalaemia or hyponatraemia) will be more pronounced in a person with diabetes than in a person without, therefore electrolytes have to be monitored closely, with appropriate medical/diabetes/paediatric oversight.
- **DKA** (see Biochemical risks in this annexe) must be excluded or appropriately managed prior to the initiation of either conventional refeeding or nasogastric tube feeding.
- Patients are likely to have significant **concerns around calorie/carbohydrate intake and insulin administration**, with risk of refusal of either or both. Anxiety levels are therefore likely to be extremely high. In an acute setting, referral to psychiatric liaison is essential and advice sought from local eating disorder/mental health services in order that appropriate management plans can be put in place, including sectioning under the Mental Health Act 1983 (updated 2007) and use of restraint if required (see Use of the Mental Health Act in this annexe).
- Initially, **blood glucose and ketone levels** are likely to be high (a combination of starvation effect and insulin deficiency); once DKA has been ruled out (or adequately treated), monitoring of ketones and glucose should be ongoing, with frequency determined by the clinical situation to ensure an appropriate and safe reduction. Rapid correction of blood glucose levels can precipitate acute, unpleasant symptoms of neuropathy (including autonomic neuropathy and gastroparesis), retinopathy and insulin oedema[9,10] (see section on Biochemical risks).

- Patients are likely to experience **symptoms of hypoglycaemia** at much higher glucose levels than would be expected. While medical management of pseudo hypoglycaemia (physical symptoms of hypoglycaemia but at glucose levels above those accepted as defining hypoglycaemia) is not required, it can be distressing for the patient, and a pre-defined quick-acting carbohydrate treatment portion can be offered to treat their symptoms (see Biochemical risks in this annexe).
- The rate of change of glucose should therefore be controlled, and it may be necessary to **start at very low insulin infusion rates initially** and accept higher blood glucose levels (see Biochemical risks in this annexe). We have some experience of using insulin at a total daily dose of 0.2–0.3 units/kg body weight as an initial oral refeeding/re-insulinisation protocol without significant detrimental shifts in electrolytes, hypoglycaemia or development of ketosis. Decisions over the starting dose of insulin should be undertaken with close supervision by a senior member of the diabetes team, dietitian and physician with clinical responsibility for the patient. A graduated approach to insulin reintroduction will also help to manage patient anxiety.
- Rate of insulin administration will be the main driver for **shifts in electrolytes** alongside initiation of the enteral feed. Frequency of monitoring of electrolytes should be based on clinical judgement and reflect levels that would be provided to those without type 1 diabetes as a minimum. Protocols for the provision of refeeding vitamins should also be mirrored.

3.d. Nasogastric tube feeding

Most settings will have had little experience of managing patients in this particular scenario, and a multidisciplinary approach is recommended in the management of this complex situation.

The use of nasogastric tube feeding in anorexia is already discussed in the Guidance, Chapter 5, but considerations specific to this particular patient group are detailed here.

Principles and competences

- It is recommended that nasogastric tube feeding is managed only where a highly skilled multidisciplinary team approach can be implemented. The team should include: a diabetes consultant supported by a specialist diabetes team, an eating disorders consultant, a nursing team with knowledge and skills of managing both enteral feeding, and type 1

diabetes and specialist dietetics with experience in both enteral feeding and type 1 diabetes.

- Patients receiving enteral tube feeding in SEDUs need to be medically stable.
- As well as demonstrating competency in the delivery of nasogastric tube feeding, staff need to be proficient in the following key areas (adapted from the Joint British Diabetes Societies for Inpatient Care guidance on enteral feeding for inpatients with diabetes[14]):
    - A working knowledge of the correct administration of subcutaneous insulin.
    - A working knowledge of the monitoring of type 1 diabetes.
    - A working knowledge of the action of different insulin products.
    - Knowledge of the definition and clinical signs of hyperglycaemia and hypoglycaemia.
    - Awareness of unit protocols to manage hyperglycaemia and hypoglycaemia.
    - Knowledge of the circumstances in which the diabetes team should be contacted.
    - Understanding of how to supervise the self-administration of insulin (to avoid manipulation of insulin doses).
- Insulin regime prescribed will be dependent on mode of delivery of the feed and should be advised by a senior member of the diabetes team, based on published clinical guidance[14] and clinical expertise. Starting doses should be decided under close supervision of a senior member of the diabetes inpatient team, with a dietitian and a physician with clinical responsibility for the patient.
- Due to the complexity of the clinical situation, it is recommended that staff administer the insulin regime. Independence of the person with T1DE around insulin administration is unlikely to be appropriate or safe at this point in treatment.
- Ready access to the diabetes team for clinical review is required. Pre-specify the thresholds and pathways at which the medical team and or on-call or diabetes team must be contacted out of hours, because not all ward staff may know which blood glucose or ketone levels might be dangerous.
- Clear guidance on blood glucose monitoring is required and must be dictated by clinical need rather than ward routine[14].

- Clear protocols for the management of hypoglycaemia need to be in place, including if insulin has been administered and the feed is subsequently stopped unexpectedly (e.g. due to nasogastric tube being pulled out or removed) (see Biochemical risks in this annexe).
- If post-bolus feed hypoglycaemia is a risk, the protocol may include giving insulin in reduced doses after the feed to minimise the risk of hypoglycaemia. Hypoglycaemia is also a risk if the patient self-induces vomiting after a nasogastric bolus or after eating.
- Treatment of hypoglycaemia may need to include feeding under restraint to treat the hypoglycaemia if insulin has already been given.
- Clear protocols for the management of hyperglycaemia and ketosis need to be in place (see Biochemical risks in this annexe).
- Clear guidance on when to escalate to the medical team is required in relation to hypoglycaemia, hyperglycaemia and ketosis.
- Consideration needs to be given to the potential for both feed and insulin to be delivered under restraint (see Use of the Mental Health Act in this annexe) and clear protocols developed should this be required.
- Consider the training requirements of medical ward staff on use of the Mental Capacity Act and Mental Health Act for these purposes and provide clear guidelines in the context of T1DE.
- Consider the training requirements of staff on diabetes management, including the administration of insulin, recognising and treating hypoglycaemia, capillary blood glucose checks and ketone checks. Staff training would include an understanding of risks associated with patients potentially tampering with nasogastric feeds and/or insulin infusions, particularly if these are administered continuously rather than as bolus.

Medical management while nasogastric tube feeding

Evidence to support specific glucose levels during nasogastric tube feeding is weak[15] and does not exist for this patient cohort.

A blood glucose level of 6–12mmol/L has been proposed as an appropriate target for the management of diabetes during enteral feeding[14], but in this cohort we propose an initial level of insulin infusion sufficient to switch off ketones, with an ongoing gradual increase in insulin to achieve an initial level of 10–17mmol/L (with ketones less than 0.6mmol/L). The recommended ongoing rate of reduction in blood glucose levels is unknown. It should be slow enough to prevent complications induced by rapid reductions in average glucose levels (see Biochemical risks in this annexe), but sufficient to allow adequate nutritional supplementation and weight gain (expert opinion).

As in oral refeeding, there is little guidance around the starting dose of insulin alongside nasogastric tube refeeding. We have some experience of using insulin at a total daily dose of 0.2–0.3 units/kg body weight as an initial oral refeeding/re-insulinisation protocol without significant detrimental shifts in electrolytes, hypoglycaemia or development of ketosis. There are two potential options for insulin administration:

1. Long-acting basal analogue insulin started immediately upon start of nasogastric tube feed and continued once (or twice) daily, plus an intravenous variable rate insulin infusion (with hourly blood glucose checks and a sliding scale) to maintain the insulin and ketone parameters above. Appropriate doses need determination by the medical multidisciplinary team prior to starting the nasogastric tube feed.
2. Insulins are administered subcutaneously to match the regime of the feed, i.e. continuous, intermittent or bolus and should follow the principles set out in the Joint British Diabetes Societies for Inpatient Care guidance[14] but with reduced insulin administration as described in the paragraph above. It is essential that these decisions are made by collaboration between experienced diabetes teams and dietitians, with regular review.

The ongoing insulin protocol will be dependent on the enteral feeding protocol; continuous, intermittent or bolus and should follow the principles set out in the Joint British Diabetes Societies for Inpatient Care[14]. Any adjunct meals or snacks must also be considered.

Frequency of blood glucose and ketone monitoring will be dependent on the enteral feeding protocol (continuous, intermittent or bolus) and should follow the principles set out in the Diabetes UK position statement[14].

People with type 1 diabetes are at risk of hypoglycaemia following insulin administration if the nasogastric tube feed is stopped for any reason (blockage or removal of the tube) or if the feed (or any adjunct meal or snack) is purged. Under these circumstances, regular monitoring of blood glucose in line with the principles set out in the Diabetes UK position statement, with availability of intravenous 10% glucose is essential[14]. People with type 1 diabetes and low BMI/%mBMI are at particularly high risk for severe hypoglycaemia (needing third-party assistance) due to reduced glycogen storage in liver and skeletal muscle; the treatment with intramuscular glucagon will not be very effective (although it remains part of emergency treatment plan of severe hypoglycaemia) and must be followed up with adequate carbohydrate replacement.

Use of diabetes technology

People on insulin infusion devices (insulin pumps) including those admitted for nasogastric tube feeding should be switched to subcutaneous multi-dose insulin. In exceptions, the insulin pump use can be continued on the ward, provided that ward staff supervising the insulin replacement on the ward around the clock have been adequately trained (completed competences on diabetes management, e.g., TREND-UK (Training, Research and Education for Nurses in Diabetes UK)

framework[16], the diabetes team has confirmed that the patient can use his device safely and the diabetes team regularly reviews downloads and settings; to prevent ketoacidosis due to cannula/infusion set problems, a low dose of long-acting basal insulin analogue alongside the insulin pump therapy can be considered.

Consider fitting a flash continuous glucose monitoring system for closer and less-invasive glucose monitoring (provided subcutaneous fat tissue allowing sensor insertion and not impacting on patient's distress) with the option for remote monitoring (which can be useful for liaising with the diabetes team for dose titration advice).

3.e. Cardiovascular health/electrocardiogram

Given that eating disorders in children, young people and adults with type 1 diabetes can be associated with blood glucose levels above recommended targets[17], adults with T1DE can be predicted to be at increased risk of cardiovascular disease (CVD) including heart disease, stroke and peripheral vascular disease. Risk factors for cardiovascular health and assessment for cardiovascular and other macrovascular complications should therefore form part of the medical assessment in T1DE.

Alongside working with a child, young person with their family or adult with T1DE to optimise their blood glucose levels, other steps can be taken in adults to reduce the risk of CVD including keeping blood pressure and cholesterol at recommended levels and working with the person to manage other modifiable risk factors such as smoking and physical activity, taking into consideration the risk that exercise may be used as a compensatory behaviour in T1DE. Routinely these measures should be started after the age of 40 for all patients with type 1 diabetes, but should be initiated earlier if other risk factors are present[17]. However, a person with T1DE may find additional medications stressful while trying to cope with incrementing insulin doses, and careful prioritisation (and potentially deferring additional CVD prevention medication to a later time in the therapy progress) with consensus of the patient's needs to be undertaken here.

3.f. Hydration/oedema

Dehydration is more likely to be seen in people with raised blood glucose and ketone levels. It can rapidly lead to medical crisis, through circulatory insufficiency and acute kidney and hepatic injury. The risk of DKA is increased in those with type 1 diabetes with profound fluid loss. All patients should be fully assessed for dehydration:

- Take a corroborative history for fluid intake and signs of decompensation (dizziness/fainting).
- Undertake physical examination to include assessment of skin turgidity and lying and standing blood pressure.

- Check electrolyte levels for high urea, creatinine, sodium and potassium.
- In the absence of DKA oral replacement is preferable.

In those with eating disorders, excessive water ingestion may occur for several reasons including a deliberate attempt to falsify weight, to mask hunger or because of difficulty differentiating between thirst and hunger. In these instances, a dilutional hyponatraemia is likely, although selective serotonin receptor inhibitors have been described to cause Syndrome of Inappropriate ADH (SIADH) as a side effect. In those with T1DE however increased thirst may be a sign of volume depletion and fluid loss from hyperglycaemia due to insufficient insulin to meet requirements. A concern about increased water ingestion should therefore be investigated carefully to understand the underlying cause so that this can be appropriately addressed. A fluid input/output chart should be kept, and daily electrolyte monitoring may be necessary in the initial phase of early stabilisation to monitor fluid balance.

In the event that excessive water ingestion is thought to be behavioural (and not due to hyperglycaemia or an underlying medical condition such as diabetes insipidus [lack of anti-diuretic hormone or renal resistance to anti-diuretic hormone]), then careful discussion with the individual should take place with goals to try and slowly reduce their water ingestion to a more reasonable total daily volume (e.g., 2.5–3 litres a day or using the Holliday-Segar equation in children and young people).

In the case of thirst as a response to hyperglycaemia, fluids should not be restricted because this will increase the risk of DKA, particularly if ketones are present. Instead, the priority should be on improving the hyperglycaemic picture.

In addition to other factors, in people with T1DE, consideration should be given to the possibility that oedema can occur with the reintroduction of insulin. It is important to physically examine the patient with oedema to rule out or treat other underlying medical causes that may have been unmasked by the rehydration (e.g. cardiomyopathy), including lung auscultation and echocardiography or chest x-ray when clinically indicated. A careful documentation of the fluid input and output balance is also helpful to monitor the oedema. While usually medically harmless, the potential psychological impact on the person with T1DE that the increase in weight and change in body shape from oedema can cause needs to be acknowledged and support provided for the person to continue with their treatment goals.

3.g. Biochemical risks

Insulin regulates how the body uses and stores glucose, protein and fat. In type 1 diabetes, the absolute deficiency of insulin results in a switch to a catabolic metabolism with increased breakdown of fat, protein and glycogen, which will also result in weight loss. The resulting production of ketone bodies as alternative fuel from fat breakdown causes increasing metabolic acidosis to the point of

metabolic decompensation (DKA), which constitutes a medical emergency, often needing intensive care treatment.

In type 1 diabetes, the absence of endogenous insulin results in a switch from carbohydrate to fat metabolism. The resulting production of ketones, if left unchecked, will result in, among other things, rapid weight loss due to the breakdown of the body's fat stores.

In T1DE, a person with diabetes may deliberately and regularly reduce or miss their exogenous insulin replacement with the specific aim of reducing weight or avoiding weight gain. This puts both their short- and long-term health at significant risk due to the consequences of the resulting hyperglycaemia and ketosis[2,18,19].

3.g.i. Management of hyperglycaemia in T1DE

Standard care of those with type 1 diabetes would aim to keep blood glucose readings between 4–10mmol/L both before and after meals, with 70% of blood glucose readings meeting this range (time in target)[20].

For those with insulin omission, giving physiologically appropriate amounts of insulin may cause psychological distress as it is likely that giving insulin will be equated with putting on weight. A stepped approach, gradually increasing the frequency and dose of insulin administered, is therefore recommended. This will allow time for psychological adjustment and reduce the risk of treatment-related complications including potential electrolyte shifts resulting from re-insulinisation (see BMI/starvation in this annexe) and treatment induced deterioration of pre-existing retinopathy and neuritis[12,13] which can occur if blood glucose levels are reduced rapidly.

Treatment induced retinopathy

Diabetic retinopathy refers to damage of the blood vessels in the back of the eye (retina). The most important factors linked with early worsening of retinopathy are the severity of pre-existing diabetic retinopathy at baseline, and a large reduction in HbA1c[21–23]. Retinal screening and assessment should ideally have occurred prior to initiating treatment to achieve tighter glycaemic control with regular follow-up screening visits planned[23].

Neuritis (treatment induced neuropathy of diabetes)

Insulin neuritis is a form of acute neuropathy. It presents as neuropathic pain, symptoms of autonomic dysfunction or a combination of both. Symptoms may include burning or shooting pains in the extremities (but may be more generalised) and orthostatic hypotension or syncope (fainting). Because insulin neuritis can lead to severe, disabling pain, there should be a focus on controlling symptoms while they gradually improve over the subsequent weeks or months.

To reduce the risk of these complications occurring, it is recommended that glycosylated haemoglobin (HbA1c) is not reduced any faster than 20mmol/mol (2%) in 2 months[24,25]. The 'normal' blood glucose targets are therefore temporarily

relaxed. Initial insulin doses are not intended to return the blood glucose levels back to 'normal' but it does serve to switch off the production of ketones reducing further weight loss and reducing the risk of DKA.

It is acknowledged that it may feel very uncomfortable for health care professionals to accept these high glucose levels; however, it is a temporary measure with the aim of progressively increasing insulin doses to achieve recommended glucose targets.

Screening for vitamin B12 and folate deficiency and multivitamin replacement should be part of standard treatment protocols in this group.

Management of raised ketones in T1DE

Insulin deficiency is associated with the accumulation of ketoacids in the extra-cellular fluid and a loss of bicarbonate anions in buffering to compensate the metabolic acidosis. The resulting metabolic acidosis can result in DKA which is associated with significant morbidity and mortality. Sick-day rules are commonly used to curtail or avert DKA and national guidance for those with type 1 diabetes to prevent DKA is available[16]. In people with T1DE, ketones also arise from prolonged starvation, thus further increasing the risk of DKA. Measuring blood pH and bicarbonate levels is essential to differentiate DKA from starvation ketones.

For those omitting insulin to manage their weight, following this guidance in full and taking appropriate doses of insulin to correct ketones may be challenging, or refused, even when they know it is for their own health and wellbeing. Some patients even take inappropriately large doses of short-acting insulin to avert having to go into hospital for DKA treatment, which can result in severe hypoglycaemia. In many cases DKA can be averted by preventing blood ketones rising above 1.5mmol/L[13] and the health care professional may need to accept that giving smaller doses of insulin than is usually recommended may be necessary with the revised aim being to reduce ketone levels to a point that prevents DKA and keeps the person with diabetes safe while accepting it does not eliminate ketones to a level usually recommended, e.g. 2 units of rapid-acting insulin for ketone levels >1.5mmol/L with repeat monitoring of ketones and blood glucose levels at 1 hour. This is not recommended best practice but the person with diabetes may feel able to accept this regime where the full recommended insulin dose would be refused. In children and young people this principle can also apply, but it may be necessary to be less compromising and offer more medically appropriate adjustment doses (up to 0.1 x total daily dose).

Training for staff on the rationale for the gradual treatment approach described above is required. Without this, the levels of hyperglycaemia and ketones that are likely to be present at the beginning of treatment can cause considerable anxiety for staff caring for the person with diabetes and might also trigger routine pathways for transfer into acute care, particularly in mental health settings. Clear treatment protocols for the management of hyperglycaemia and ketones specifically designed for this cohort of patients are therefore essential. An example inpatient management plan for this purpose can be found in Appendix B.

Monitoring of blood glucose level and ketones

Measurement of blood glucose and ketones using blood glucose and ketone meters are recommended in preference to urine testing strips.

Flash glucose monitors

Use of flash glucose monitoring can be helpful in the treatment of T1DE. Results from this method of monitoring correspond closely with capillary blood glucose readings and is approved to dose insulin without the need for an accompanying finger prick (except for flash glucose monitor readings below 4.0mmol/l, readings that are discrepant to clinical symptoms, or sensor malfunction). Data from the sensor can be uploaded and shared with healthcare professionals and allows for remote specialist advice, which is beneficial for multidisciplinary working, particularly in SEDUs.

Continuous glucose monitors

Some glucose monitoring systems are also equipped with alarms which can identify hypoglycaemia and offer reassurance to those with T1DE who are fearful of hypoglycaemia. The reduced need for finger prick testing to ascertain blood glucose readings can also reduce diabetes-related distress and be helpful for those with needle phobia. The decision as to whether continuous glucose monitor systems are supportive of recovery has to be taken on a case-to-case basis, as diabetes distress can increase, e.g. some patients are alarmed by high blood glucose levels they see on their continuous glucose monitor system in the erroneous belief that high blood glucose levels cause weight gain. (See Other mental health diagnoses in this annexe).

Management of diabetic ketoacidosis

Guidance for treating DKA has been published by the Joint British Diabetes Societies Inpatient Care Group[26]. There is universal agreement that the most important initial therapeutic intervention in DKA is appropriate fluid replacement along with insulin administration. The highest mortality in DKA is related to potassium abnormalities. Treatment with insulin promotes uptake of potassium intracellularly which can lead to hypokalaemia which is a potentially life-threatening complication during the management of DKA. Resolution of DKA is when blood ketones <0.6mmol/L and venous pH >7.3[26]. Blood glucose levels will reduce as the ketones reduce. Some local treatment protocols with fixed insulin rates are calibrated for normal weight adults, resulting in fast drops of blood glucose in underweight people with T1DE and will have to be modified on a case-to-case basis. It should be noted that gradual reduction in blood glucose is essential as this is a critical window for primary prevention of acute neuropathy. In children, standard guidelines are weight-based and would therefore apply.

3.g.ii. Hypoglycaemia and hypoglycaemia unawareness

Medical hypoglycaemia is defined as a blood glucose level of <4.0mmol/L (<3.5mmol/L is now used in adults, but not in hospital). If left untreated, it can progress to loss of consciousness, collapse and death. Hypoglycaemia is more likely to be seen in those with T1DE who administer insulin but use carbohydrate

restriction and/or excessive exercise as the primary mechanisms for controlling weight.

Causes of hypoglycaemia in those with type 1 diabetes are described elsewhere[b]. In those with T1DE, additional causes may be implicated if insulin is being administered including low glycogen stores as a result of carbohydrate restriction, low BMI/%mBMI or excessive exercise, delayed gastric emptying as a result of diabetes-related gastroparesis and/or starvation, purging, overcompensation with insulin following a binge, increased sensitivity to insulin as a result of excessive exercise and low carbohydrate diets.

Management of hypoglycaemia in T1DE

The factors outlined above that increase the risks of hypoglycaemia need to be addressed within the management of T1DE. Standard treatment guidelines for hypoglycaemia are described elsewhere[b]. It is important to recognise that those with T1DE may refuse any or adequate oral treatment of hypoglycaemia due to fear of the calories that will be consumed, putting them at risk of recurring episodes of hypoglycaemia or the need for alternative methods of treating their hypoglycaemia such as intravenous dextrose. People with type 1 diabetes have to have access their hypoglycaemia treatment at all times, which can represent challenges around managing binge eating and deliberately induced ketosis. It may be helpful to prescribe medicalised hypoglycaemia treatment (glucose in liquid or gel form and pre-portioned) to help avoid bingeing on hypoglycaemia treatment food items.

Clinicians should also be aware that a glucagon injection may not have the desired effect of raising blood glucose levels and would not be a recommended treatment under the following circumstances due to the potential for glycogen stores to be reduced:

- recurrent hypoglycaemia
- periods of sickness involving vomiting, diarrhoea and loss of appetite
- eating no or very little food
- chronically low carbohydrate intake
- excessive exercise
- low BMI/%mBMI
- repeated purging.

[b] Including: www.nice.org.uk/guidance/ng18/informationforpublic/type-1-diabetes-in-children-and-young-people/chapter/low-blood-glucose-hypoglycaemia, www.nice.org.uk/guidance/ng17/chapter/recommendations#hypoglycaemia-awareness-and-management, www.ispad.org/resource/resmgr/consensus_guidelines_2018_/12.assessment_and_management.pdf and https://diabetes-resources-production.s3.eu-west-1.amazonaws.com/resources-s3/2018-05/JBDS_HypoGuidelineRevised2.pdf%2008.05.18.pdf.

A person with T1DE may also request glucagon as a hypoglycaemia treatment, to avoid ingesting the calories in oral hypoglycaemia treatments. This, however, is not recommended if oral treatment is clinically indicated as it will leave glycogen stores depleted.

An example plan for the management of hypoglycaemia, including pseudo hypoglycaemia for use inpatient settings can be found in Appendix B. On SEDUs, guidance should also be available clearly indicating when transfer to an acute hospital setting for the treatment of hypoglycaemia is required.

Hypoglycaemia unawareness
Those who experience episodes of severe or recurrent hypoglycaemia can lose their symptom awareness of hypoglycaemia, meaning they will no longer be able to detect when their blood glucose levels drop below 4mmol/L. This will put the person at increased risk of requiring external assistance for recovery. Hypoglycaemia awareness may be partially restored by avoiding further hypoglycaemia and for this reason people with diabetes are encouraged to aim for their blood glucose levels to remain above 4mmol/L, with the commonly used motto 'make four the floor'[2,3]. To achieve this, psychological work may be required, e.g. to address fear of complications or challenge the perfectionism trait often seen in eating disorders with an alternative 'good enough' approach[5].

Pseudo (or false) hypoglycaemia
If a person with T1DE has been running high blood glucose levels for a prolonged period, they may experience symptoms related to hypoglycaemia at normal or high blood glucose levels, particularly when insulin is re-introduced and blood glucose levels begin to decrease. Blood glucose monitoring is essential to be able to distinguish a pseudo hypoglycaemic episode from medical hypoglycaemia where the blood glucose is <4mmol/L. Treatment for pseudo hypoglycaemia should be conservative, i.e. encouraging rest, offering reassurance and re-checking blood glucose levels 10 minutes later. However, for symptomatic relief or to manage associated anxiety, if blood glucose levels are between 4–12mmol/L, a small dose (e.g. 5g) of rapid release carbohydrate can be offered. It is important not to over-treat pseudo hypoglycaemia because repeated treatments will result in maintaining the status quo rather than enabling adjustment to the reduction in blood glucose levels. Over treatment of pseudo hypoglycaemia may also be used in a deliberate attempt to maintain high blood glucose levels and ketones to achieve an ongoing weight-losing state. It is important to note that experiencing pseudo hypoglycaemia can produce the same psychological responses as typical hypoglycaemia, such as fear and anxiety.

3.g.ii. Acid-base changes

Changes in acid-base balance observed with misuse of laxatives (typically a metabolic acidosis) or with vomiting and diuretic use (typically metabolic alkalosis) can be further complicated by the acidosis resulting from the accumulation of ketoacids that occurs with insulin omission. In T1DE a range of different acid-base balances can therefore be seen and in some instances a mixed picture may be present.

3.g.iii. Electrolyte imbalance

Important clinical factors that contribute to electrolyte imbalance in T1DE include:

- the use of laxatives
- purging
- increased fluid intake
- hyperglycaemia causing pseudo-hyponatraemia
- the use of SSRI
- malnutrition/malabsorption
- vomiting due to gastroparesis.

The potential for renal impairment, as a complication of type 1 diabetes to impact on the biochemical picture should be considered and assessment of renal function should form part of the medical assessment in T1DE. The first sign of nephropathy may be microalbuminuria. Screening and calculation of albumin: creatinine ratio in type 1 diabetes is recommended[8]. Early nephropathy is usually treated with an angiotensin-converting enzyme inhibitor and by improved blood glucose and blood pressure management. Management of chronic kidney disease is described in NICE clinical guideline 182[8]. In the acute crisis presentation of T1DE, acute kidney failure due to severe dehydration following hyperglycaemia induced glucosuria is often the dominant picture. Malnutrition can also influence renal markers (e.g. low creatinine).

3.h. Medication

Because of the associated weight loss, gastrointestinal symptoms and risks of hypoglycaemia and DKA we recommend that these adjunct therapies should be used with caution in those with T1DE.

- Glucagon-like peptide 1 (GLP-1) agonists and sodium-glucose cotransporter-2 (SGLT-2) inhibitors have, in some studies, been demonstrated to reduce HbA1c, body weight and total daily doses of insulin when used by people with type 1 diabetes alongside insulin[27].
    - GLP-1 agonists have, however, been associated with gastrointestinal side effects[28], which may be counterproductive for the recovery of a person with T1DE who tries to avoid purging by vomiting or who has gastroparesis.
    - SGLT-2 were associated with a greater risk for DKA and volume depletion, particularly euglycaemic DKA where ketones and acidosis are present but at a much lower glucose level which can make identification and treatment difficult[27]. SGLT-2 inhibitors are contraindicated in people with T1DE due to the increased risk for DKA in this group.

Where those with T1DE are already prescribed these medications, the physiological and psychological risks of continuing or discontinuing them needs to be carefully considered in conjunction with the person with T1DE.

Metformin also has gastrointestinal side effects and can be used to promote weight loss. It should be used with caution.

3.h.i. Other medicines

Olanzapine is commonly used at low doses in the management of eating disorders. It can be associated with insulin resistance, but the effect is rarely clinically significant.

There should be no diabetes contra-indications for the use of other psychotropic medicines.

3.i. Disordered eating behaviours

3.i.i. Food restriction

In presence of appropriate insulin administration food/carbohydrate restriction will result in the same risks as for those with restrictive anorexia nervosa. There is the potential for an increased risk of hypoglycaemia if insulin is not appropriately adjusted (see Hypoglycaemia in this annexe). Food restriction also results in starvation ketones, further increasing the risk of DKA.

Food restriction alongside omission or restriction of insulin will result in risks increased beyond those of restrictive anorexia nervosa as the availability of calories to the body will be further reduced.

Excessive carbohydrate consumption

Excessive carbohydrates may be consumed to keep blood glucose levels and ketones high, to promote weight loss. It is described in more detail below (see Compensatory behaviours in this annexe).

Bingeing

For those with T1DE, bingeing will have risks beyond those without T1DE. Without additional insulin being administered, blood glucose levels will rise, and ketones may ensue. Conversely, guilt, shame or fear of complications after an episode of bingeing may result in overcompensation with a large dose of insulin, potentially causing hypoglycaemia[29]. Where the person is driven to engage in compensatory behaviours after a binge, as such as restriction, purging and excessive exercise, the challenge of stabilising blood glucose levels is further complicated.

In the context of pre-existing gastroparesis, there is a small but real possibility that bingeing may lead to gastric dilatation, ischaemia and perforation.

Diabetic gastroparesis

Where it is present, diabetic gastroparesis can result in food patterns, behaviours and symptoms that may mirror or maintain eating disorder pathology, including

early satiety and bloating, loss of bowel motility and poor dietary intake with associated weight loss. It should be included in the medical assessment of people with T1DE. Gastroparesis can also be transient in the context of sustained hyperglycaemia, therefore assessments for gastroparesis diagnosis should be conducted once blood glucose levels are normalised. In a person with a binge-purge pattern eating disorder, it can be difficult to differentiate to what extent vomiting is due to gastroparesis and to what extent it is self-induced. Adjunct treatment with antiemetics may be considered.

3.i.ii. Activity and exercise

The impact of exercise on risk of hypoglycaemia and ketosis are described in the sections on Biochemical risks and Compensatory behaviours. Physical activity in the presence of incipient DKA can accelerate metabolic deterioration. Over-exercising can also contribute to hypoglycaemia risk and to weight loss.

Exercise can result in friction burns, bruising and blisters, cracked or dry skin and increased callous formation on the feet. In those with T1DE, the risk to foot health is of particular concern due to the circulatory and neurological complications potentially associated with both type 1 diabetes and starvation, if it is present. Fracture risk will also be increased in patients with reduced bone density. Foot assessment should form part of the medical assessment in those with T1DE including checks for signs of circulatory and neurological deficit. More information on diabetic foot care can be found in NICE guidance NG19 and on the Diabetes UK website[9,30].

3.i.iii. Compensatory behaviours

The risks associated with compensatory behaviours will mirror those of other eating disorder presentations. Additional considerations relating to T1DE follow.

3.i.iv. Insulin omission or reduction

When challenged to address the omission or reduction of insulin, people with T1DE may engage in a range of strategies to disguise this behaviour (see Table 2; this list is not exhaustive). Over-checking of blood glucose and ketone levels to gain inappropriate reassurance that a weight-losing state is maintained may also occur. Consideration needs to be given as to how best the person with diabetes can be supported to address these behaviours:

- Staff should have a good working knowledge of insulin administration.
- Staff should be aware of the potential ways in which insulin omission can be disguised.
- Insulin should be stored safely at an appropriate temperature to prevent deliberate deterioration.
- In the initial stages of treatment, it is suggested that equipment, including pens, needles and meters should be stored in a clinical area to minimise

the risk of deliberate damage, tampering or misuse and spare equipment should be readily available in the event of damage or loss.

- Staff should closely supervise insulin administration at least in the initial stages of treatment. It is our experience that close supervision is preferable to staff administration of the insulin however be guided by the individual situation.
- As a general principle we would advocate pens rather than pump for insulin administration. This facilitates staff confidence in settings where the complexities of managing a pump may be challenging for non-diabetes specialist staff. Attempts to avoid insulin administration by the person with diabetes are also more easily identified and therefore the person can be better supported to give the advised dose.
- Staff should be aware that the anxiety of insulin administration will be akin to that of eating food in people with other eating disorder presentations. Anxiety is likely to be high when insulin is being administered, so allowing time, taking an empathic approach and reassurance are key.

Table 2: Examples of behavioural symptoms that might alert clinicians to increased risk in people with T1DE

Strategy	
Secrecy	Injecting insulin in private/away from others.
Insulin	Leaving insulin in a warm environment to deteriorate, thereby reducing its effectiveness.
Pens	Leaving diabetes equipment elsewhere.
	Dialling up a smaller dose of insulin or dialling down dose just before injection.
	Omitting to 'prime' the insulin pen, resulting in the delivery of a reduced dose of insulin.
	'Squirting' the insulin prior to placing the needle into the injection site.
	Injecting insulin into sites where lipoatrophy is present to reduce the absorption of insulin into the body.
	Not fully depressing the insulin pen plunger.
	Releasing the plunger or pulling out the needle from the injection site before the end of the full 10 seconds required to deliver the full dose of insulin.
	Continuing to pinch up the skin after injection to squeeze insulin out.
	Injecting the insulin into clothes.
	Bending needle just before injection or not attaching needle properly.
	Leaving insulin pen on a radiator/in the sun to denature.
Pumps	Taking the pump off and not reconnecting it.

	Turning the pump off – disabling the alarm.
	Incorrect siting of the cannula resulting in a reduced or no delivery of insulin to the infusion site.
	Making a hole in the tubing from which the insulin can drip out.
	Altering the ratio doses programmed into the pump.
	Falsifying blood sugar readings in order that the pump does not advise a correction dose.
	Reducing the amount of carbohydrate entered to avoid the pump advising as much/any insulin.
	Changing the pump settings to facilitate under dosing of insulin, e.g. falsifying the time of insulin doses given so it appears that insulin remains on board.
Meters	Making up solutions using sugary substances and using this to falsify blood glucose readings to be lower, to avoid a correction dose.
	Frequent checking that ketones are present and if not taking steps to increase carbohydrate/decrease insulin.
	Using control solution, adding alcohol to test strips to obtain false low readings.

Example flow charts for the management of both long and short acting insulin refusal can be found in Appendix C. An example protocol for returning autonomy of insulin administration to the person with diabetes is also available in Appendix D.

As well as omitting insulin, other strategies may be employed to increase blood glucose levels and/or achieve a ketotic state with the purpose of achieving loss of body fat and weight. This puts at high risk for DKA, though many are able to maintain a subclinical state of ketoacidosis to avoid admission. These strategies include:

- Overconsumption of high carbohydrate foods and drinks alongside insulin omission may be present at diagnosis or may emerge as insulin is restarted to try to maintain the presence of raised blood glucose and ketone levels. Support should be provided to challenge this pattern of carbohydrate consumption and consideration given to access to high carbohydrate foods including sugar and high sugar drinks which are often readily accessible in inpatient environments. Overconsumption of hypoglycaemia treatments may also be used for this purpose. Overconsumption of high carbohydrate foods and drinks can also be triggered by pseudo or real hypoglycaemia that induces hunger and adrenergic symptoms as part of the counterregulatory response.
- In a person without enough circulating insulin, exercise will raise blood glucose levels and increase ketone production[29] therefore exercise may be

used in T1DE as a method of maintaining raised blood glucose and ketone levels. Fluid losses resulting from exercise will further progress the ketonaemia, increasing the risk of DKA. Support to challenge this potentially dangerous pattern of behaviour may therefore be needed, and consideration given to raised ketones as a potential consequence of covert exercise, particularly in the early stages of treatment when for the reasons described above insulin doses will be small (see Biochemical risks in this annexe).

Emergence of other eating disorder behaviours

Those with T1DE who are omitting or restricting insulin may present atypically, appearing relaxed around food choices and not engaging in other compensatory behaviours. Insulin reintroduction may result in the emergence of compensatory behaviours not previously observed, as the person's focus on how they can control weight and shape shifts elsewhere. Clinicians, particularly in inpatient settings where insulin administration is closely supervised should be alert to the emergence of behaviours more typical of other eating disorder presentations such as restriction, covert exercise and purging. Clinicians should also be aware of the potential for alternative self-harm behaviours to emerge and for deterioration in mood and increased anxiety symptoms (see Other mental health diagnoses in this annexe).

Vomiting

Vomiting can lead to unpredictable blood glucose levels, including the risk of hyperglycaemia and hypoglycaemia, and their associated complications (see Biochemical risks in this annexe). This may be further complicated by insulin omission or restriction in those with T1DE and in those who have gastroparesis. Clinical management of bolus insulin around this may be giving split insulin boluses (lower insulin bolus before the meal with a post-meal correction if the meal has been kept down) or low-dose post-meal injections.

Sham feeding

Other behaviours include chewing and spitting out food without swallowing it, also known as 'sham feeding'. Patients may over- or under-estimate the amount of energy absorbed and thus be at risk of hypo or hyperglycaemia. This behaviour is often experienced as deeply shameful and rarely spoken about.

Engagement with the management plan

In those with T1DE, barriers to engagement with the management plan will mirror those of other eating disorder presentations. Additional considerations for T1DE are:

Previous experiences with health services

Negative past experiences with healthcare teams can present a significant barrier to the engagement of this cohort. Contact and communication that reflects a supportive, non-judgemental, encouraging and empathic approach, which continues if avoidance of contact persists, is essential[6].

Language
Careful consideration and adjustment of the language used is key[31].

Cognitive function
It is known that starvation has a significant impact on cognitive function[32] and that refeeding is required for successful engagement in treatment plans and psychological therapy. Similarly, both the presence of hyperglycaemia and ketones[33] and severe or recurrent hypoglycaemia[34] have been demonstrated to impact on cognitive function and therefore have the potential to interfere with progression of treatment. Improvement in these aspects of diabetes management are therefore important for engagement with the treatment plan.

Diabetes-related psychological concerns
Diabetes-related distress and other diabetes-related psychological concerns can impact on progression of treatment (see Other mental health diagnoses in this annexe).

Lack of insight
Lack of insight has been described and reviewed for people with anorexia nervosa[34–36]. Research is lacking in relation to T1DE, but anecdotally it has been observed and may contribute to a lack of engagement in treatment. The degree of presenting risk must be assessed, and where medical or psychiatric risks are high, consideration should be given to capacity to consent to treatment (see Use of Mental Health Act in this annexe). Lack of insight may also need to be considered in relation to safeguarding of self and others, fitness to work and driving. The impact on carers can also be considerable.

Family and carers
The ethos of diabetes care is often to encourage autonomy. In contrast, the ethos within eating disorder services would be to encourage family and carers to offer support to their loved ones with T1DE, to address the challenges they face. Living with a person with T1DE can cause family members and partners anxiety; seeing that they are clearly unwell but may be lacking insight into their illness and struggle to eat and/or take their insulin can drive strong emotional reactions that, in turn, can play a part in the maintenance of the eating disorder. Consideration should therefore be given to how families and carers can best be supported.

Other mental health diagnoses
As well as being common, comorbidities in eating disorders, depression and anxiety also commonly occur in those with type 1 diabetes[37].

Depression in those with type 1 diabetes is associated with suboptimal diabetes self-management, raised HbA1c levels and increased diabetes distress[38]. The deterioration in metabolic control that occurs in those with T1DE who restrict or omit insulin can worsen depression[39].

Anxiety may be overlooked in those with type 1 diabetes because severe anxiety and panic attacks share some similar physical symptoms with hypoglycaemia (e.g. sweating, increased heart rate, shaking and nausea). Consequently, elevated

anxiety symptoms may be misinterpreted both by people with diabetes and by health care professionals, resulting in anxiety disorders remaining undiagnosed.

From clinical observation, mental health comorbidity and hyperglycaemia can be intertwined: in some patients persistent and chronic hyperglycaemia masks symptoms of their underlying depression or anxiety disorders and symptoms seem to re-emerge when glycaemic management improves. This makes frequent monitoring and re-assessment of the mental health status during therapy mandatory.

Diabetes distress is the emotional distress resulting from living with diabetes and the burden of the relentless tasks of daily self-management[40] as well as the social impact of diabetes[41]. It is observed to commonly coexist in those with T1DE. Diabetes distress can present a significant barrier to recovery as it requires the person to re-engage with all of the practical tasks and cognitive processing that is required of living with type 1 diabetes on a daily and ongoing basis. Its psychological treatment cannot be separated out from treatment of the eating disorder as the benefits of not having to engage in these tasks serves as a powerful maintaining factor in those who restrict or omit insulin to control their weight and shape.

Fear of medical hypoglycaemia (blood glucose level <4mmol/L) is a specific and extreme fear evoked by the risk and/or the occurrence of low blood glucose levels[42]. Being concerned about hypoglycaemia is rational and adaptive, but if this develops into excessive fear, it can result in the omission or reduction of insulin doses, or in the overconsumption of carbohydrates to maintain blood glucose at a raised level[5]. It may therefore emerge as a barrier to the treatment of T1DE as blood glucose levels normalise. Similarly, a fear of needles (or, in its extreme form, a needle phobia) can pose an obstacle to improved diabetes management in people with T1DE. Conversely, a fear of complications can result in overly tight management of blood glucose levels with its associated risk of severe or recurrent hypoglycaemia.

Self-harm and suicide

There is limited literature on the risk of self-harm in people with T1DE, but it is reasonable to assume that the potential for self-harm behaviours will mirror those of other eating disorder presentations, and their presence and management should be considered in this patient cohort. If not present at diagnosis, clinicians should be aware, as with other eating disorder presentations, that self-harm behaviours may emerge as weight and shape concerns are challenged by treatment. The act of ongoing insulin omission and the damage it causes can also in itself be viewed as a form of self-harm[43]. There is also a clinical subgroup with comorbid emotional instability, self-harm or history of trauma for whom insulin omission is a coping strategy.

Suicide occurs more frequently with the coexistence of psychiatric and physical illness[44]. The increased risk of suicide in eating disorders is well-documented and a systematic review on suicide risk and type 1 diabetes indicated that, in general, adult patients with type 1 diabetes have a higher risk for suicide than the general

population[45]. In addition, it has been found that young adults admitted to hospital for DKA have an increased risk of being admitted to hospital for a subsequent suicide attempt. The risk of a suicide attempt was highest in the 12 months following the ketoacidosis episode[46].

It is vital to consider the risk of suicide in those with T1DE. Importantly those with T1DE have access to insulin which can cause hypoglycaemia and subsequently death if it is severe and lasts for a prolonged period of time. Healthcare professionals working with those with T1DE must therefore be mindful of the potential for this lifesaving drug to be used in a deliberate attempt to end life. A four-point plan for the safe management of depressed adult patients prescribed insulin has been proposed[47]:

- Regularly assess for symptoms of depression.
- Refer to a mental health provider (sooner rather than later).
- Work closely with the mental health provider.
- Monitor thoughts of suicide when working with a patient with depression.
    - If there are red-flag warning signs/immediate risk of suicidal behaviour, the patient will require:
        - immediate discussion with or referral to mental health services
        - a robust safety plan
        - adequate support
        - removal of access to means.

The safety plan should be co-produced with the patient and should have explicit reference to removal and/or mitigation of means to harm themselves. Mental health providers will need to liaise with diabetes services to consider what realistic plans can be put in place surrounding availability of insulin as a means of harm, e.g. insulin injections under supervision only. The safety plan should list activities and coping strategies and contain information on how to access social, psychological and emergency support[44].

For children and young people, the ingredients will be the same, but early involvement of local child and adolescent mental health services is recommended for them to lead the care, supported by the children's diabetes team who know the young person and their family.

3.i.v. Use of the Mental Health Act

Because eating disorders can be accompanied by significant morbidity, especially if insulin restriction is used as a compensatory behaviour, there may be occasions when clinicians may consider using the Mental Health Act[48] in the context of T1DE (e.g., when a patient's physical health or survival is seriously threatened as a result of their mental illness).

The Mental Health Act section 63 ('medical treatment' in relation to a mental disorder) refers to medical treatment, the purpose of which is to alleviate or prevent a worsening of the mental disorder or one or more of its symptoms or manifestations. It applies only to medical treatment for the mental disorder. Therefore, treatment for a physical condition may only be given without the patient's consent if it is sufficiently connected to the treatment of the patient's mental disorder. Note that section 63 only applies to patients detained for assessment or treatment under the Mental Health Act of a Mental Disorder (e.g. section 2 or 3).

Considering the above in relation to T1DE, it would seem permissible to compel a person to receive treatment specifically for the management of their diabetes if it would prevent a serious deterioration in their health. Prolonged hyperglycaemia and ketosis will need to be taken into account during assessment as factors that impact on cognitive functioning and, therefore, potentially also mental capacity.

In patients over the age of 16, it may also be possible to justify action taken under the Mental Capacity Act[49] in an emergency as the minimum necessary to prevent serious injury or loss of life. For those under 16 years, the Mental Health Act applies.

Common law can be used to treat patients in emergencies if it is not possible to perform a capacity assessment, e.g. if the patient is unconscious due to diabetes[50].

Appendices

Appendix A: Example re-insulinisation and refeeding protocol for people with type 1 diabetes and disordered eating[c]

All patients requiring support with re-insulinisation/refeeding should be considered at risk of shifts in electrolytes with the reintroduction of insulin and/or carbohydrate.

Food

- In the first instance, the proposed fixed insulin doses will not match carbohydrate intake; therefore, strict carbohydrate counting is not required[2]. An awareness of carbohydrate content at different meals is, however, important to ensure the meal plan is safe and avoids unnecessary hypo or hyperglycaemic states. Carbohydrate estimates are provided in the example refeeding starter plan.
- People with T1DE may have selective concerns regarding the carbohydrate content of their plan and may require reassurance to normalise the balance of their intake. A daily carbohydrate intake of around 150g is deemed appropriate within the context of a refeeding starter plan (see example).
- Patients should have access to both quick- and long-acting carbohydrate as pre-defined treatment portions. Their consumption needs to be clearly documented.
- If eating more than the ½ portion plan, do not reduce food intake; instead, continue with the person's current dietary intake and follow the same insulin protocol below. This is agreed as safe because the refeeding risk is mediated by the amount of insulin provided, not by the amount of food.
- Refeeding vitamins are recommended as outlined in the text box below and should be started alongside the re-insulinisation/refeeding plan.

[c] The protocol outlined below has been developed by the ComPASSION TEAM. Dorset Health Care University NHS Foundation Trust/University Hospitals Dorset NHS Foundation Trust.

Multivitamin

Adults (18+ years): Multivitamin and mineral supplement, e.g. Forceval capsule or Forceval soluble tablet, 1 daily

12–17 years: *Forceval* 1 tablet daily

1–12 years: Abidec 0.6mL daily

Vitamin B

Vitamin B complex strong (one tablet contains 5mg B_1 [thiamine], 2mg B_2 [riboflavin], 20mg nicotinamide and 2mg B_6 [pyridoxine])

Adults (18+years): 2 tablets three times daily (TDS)

12–18 years: 3 tablets TDS

1–12 years: 2 tablets TDS

Thiamine 100mg BD*

Adults (18+ years): 100mg BD(51)

Under 18 years of age: (BNF 52) Evidence is lacking in the under 18s. Adult doses can be used for older adolescents.

**Note: If strong suspicion of thiamine deficiency: Pabrinex*

Vial 1 (5mLs contains B_1 [thiamine] 250mg and B_2 [riboflavin] 4mg, B_6 [pyridoxine) 50mg])

Vial 2 (5mLs contains C [ascorbic acid] 500mg, nicotinamide 160mg, glucose 1000mg)

Adult dose: 10mL of each ampoule diluted with 50–100ml of (0.9% saline or 5% dextrose) given over 30 minutes

Child dose:

6–10 years	Third of the adult dose
10–14 years	Half to two thirds of the adult dose
14 years and over	Adult dose

- Once the ½ portion meal plan is established, aim to increase one meal to full portion every 2–3 days to establish full portion breakfast, lunch and evening meal. It should be acknowledged that some people will potentially

be psychologically unable to tolerate this speed of increase in food and the plan should be adjusted accordingly.

- For those not completing meals and snacks, nutritionally complete supplements should be offered as an alternative, to manage the risk of underfeeding and minimise the risk of hypoglycaemia and pseudo hypoglycaemia. Amounts should be calculated based on approximations to the meal or snack equivalent carbohydrate content. Note that some nutritional supplements do not contain carbohydrate, e.g. Calogen (Nutricia) and Fresubin 5kcal Shot (Fresenius Kabi).
- In the first instance, the proposed fixed insulin doses will not match carbohydrate intake; therefore, strict carbohydrate counting is not required[d]. An awareness of carbohydrate content at different meals is, however, important to ensure that the meal plan is safe and avoids unnecessary hypo or hyperglycaemic states. Carbohydrate estimates are provided in the example refeeding starter plan.
- Those with T1DE may have selective concerns regarding the carbohydrate content of their plan and may require reassurance to normalise the balance of their intake. A daily carbohydrate intake of around 150g is deemed appropriate within the context of a refeeding starter plan.
- Patients should have access to both fast- and long-acting carbohydrate as pre-defined treatment portions. Their consumption needs to be clearly documented.

Insulin

- If omitting or taking only sporadic or minimal amounts of insulin, commence initially on 10 units of long-acting insulin. Some negotiation may be required around this initial dose, which can be calculated as approximately 0.2 units/kg of body weight with planned incremental increases to meet the individual's requirements (expert opinion). This is alongside fixed doses of two units of rapid-acting insulin for meals (initially no insulin with snacks).
- Ketones should be tested pre-meal if blood glucose is elevated (centres may vary on the level at which they intervene) and if ketones are present follow the hyperglycaemia/ketone management plan in Appendix B.
- With each meal increase, aim for a doubling of initial insulin dose. It should be acknowledged that some people will potentially psychologically be unable to tolerate this speed of increase in insulin and the insulin plan

[d] An alternative approach is to use carbohydrate to insulin ratios with, e.g., a starting insulin-to-carbohydrate ratio of 1 unit of rapid-acting insulin to 20g of carbohydrate.

adjusted accordingly. Alternatively, the insulin-to-carbohydrate ratio can be adjusted in a stepwise plan.

- It is acknowledged that blood glucose will remain elevated in the first instance, and this can be addressed after the refeeding period is completed, with insulin increases being made at meals and introduced at snacks to continue the process of reducing blood glucose levels gradually. See the Insulin management plan in Appendix D.
- Beyond the initial reintroduction of insulin and food as blood glucose levels normalise, options include a fixed carbohydrate meal plan alongside fixed doses of insulin **or** the reintroduction of carbohydrate counting alongside variable doses of insulin.

Monitoring

Flash monitoring is advised and will enable clinicians to monitor the change in blood glucose and make gradual appropriate adjustments to insulin accordingly.

Although there can be a risk of electrolyte shifts up to a fortnight later in the refeeding process, the risk is highest within the first 72 hours of initiating refeeding[53]. As a minimum, check refeeding bloods at baseline, day 2 and day 5, then as clinically indicated.

In the absence of robust evidence, where re-insulinisation/refeeding is progressed more rapidly or if other risk factors for the development of refeeding syndrome are present such as infection or abnormal baseline electrolytes, increased frequency of monitoring of electrolytes is advised based on clinical judgement and reflecting levels that would be provided to those without type 1 diabetes as a minimum.

Local guidance for correcting blood levels if abnormal electrolytes are identified is outlined in Table 3 below. Always seek consultant physician/paediatric, or diabetologist or psychiatrist guidance on management if abnormal electrolytes are identified.

Weight should be monitored weekly. Significant upward shifts in weight during the refeeding phase will reflect changes in fluid balance rather than a significant increase in lean tissue and body fat. While this shift in fluid is usually medically harmless the potential for psychological impact needs to be acknowledged and support provided for the person to continue with the refeeding plan despite the change in weight. Beyond the refeeding period more predictable weight change can be anticipated as physiology normalises. Ongoing support with weight and shape concern will be required.

Table 3: Guidance for the correction of electrolyte abnormalities

Potassium (mmol/l)	
3.5–5.0	Normal range
3.0–3.4	Replace orally with Sando K (12mmol K+ per tablet), 2 tablets BD–TDS or 2mmol/kg/d in divided doses in children and young people
<2.5	Requires intravenous replacement
Sodium	
	Standard guidance from Guidance (Chapter 2.d.iii.2) applies – note pseudohyponatraemia can occur in the context of hypoglycaemia
Phosphate	
0.8–1.6	Normal range
0.5–0.79	Replace orally with Phosphate Sandoz (16.1mmol PO_4 per tablet), 1–2 tablets BD
<0.5	Requires intravenous replacement – refer patient to acute care
Magnesium	
0.7–1.1	Normal range
0.5–0.69	Replace orally with magnesium citrate 150mg (6.2mmol/tablet). Two tablets BD
<0.5	Requires intravenous replacement – refer patient to acute care

Support

Undertaking the proposed protocol can be challenging for those with T1DE. Barriers to change include fear of weight gain and diabetes distress. Consideration therefore needs to be given to how the person with diabetes can be supported.

Staff should be aware that the anxiety of insulin administration will be akin to that of eating food in those with other eating disorder presentations. Anxiety will therefore be high at the times when insulin is administered. Allowing time, taking an empathic approach and reassurance are key.

It can be helpful to consider a staged approach, to support the person to move forward with re-engaging with taking their insulin. A staged guide to achieving this has been developed. See Appendix D.

Refeeding starter plan[e]

BREAKFAST 	**Carbohydrate:** 1 cup* Rice Krispies/Cornflakes, ¾ cup other cereal, or 2 Weetabix or Shredded Wheat	26–34g (carbo-hydrates)
	Add some protein: ¾ cup semi-skimmed milk	8g
MORNING SNACK:	1 cup semi-skimmed milk	11g
LUNCH 	**Carbohydrate:** 1 medium slice of bread or ½ bagel or 1 standard pita bread or 1 small jacket potato (to fit in ½ the palm of your hand)	16.5–24g
	Add some protein: 1 ½ slices cooked meat/chicken or ½ small can tuna/ baked beans or 1 cheese circle or equivalent cheese portion, 1 egg or 1 individual pot of hummus.	
	Add some vegetables: small bowl salad	
	Add a fat source: 1 level tsp butter/margarine/mayonnaise or salad cream	
Include a second course, e.g.:	**Dairy option:** ½ standard pot of whole milk yogurt (e.g. Müller Fruit Corner, Activia or Greek-style) or ½ rice pudding/custard pot or 1 scoop of ice cream.	6.5–11g
	Add some fruit: ½ a portion of fruit	3–10g
AFTERNOON SNACK:	1 cup semi-skimmed milk	11g
MAIN MEAL: 	**Carbohydrate**: ½ cup cooked rice, pasta, spaghetti or couscous, 1 jacket potato (to fit ½ the palm of your hand) or 2 egg-sized potatoes, 9 chips/wedges.	15–23g
	Add some protein: ½ chicken breast or equivalent meat portion, 1 rounded serving spoon of Mince (1 ½ if a high vegetable content), ½ baked fish fillet, 2 chipolata sausages, 1 egg, 1 rounded serving spoon Quorn pieces/kidney beans/chickpeas/cooked lentils (1 ½ if a high vegetable content).	
	Add some vegetables: ½ serving spoon of vegetables, small bowl salad	

[e] Developed by the Wessex ComPASSION Team.

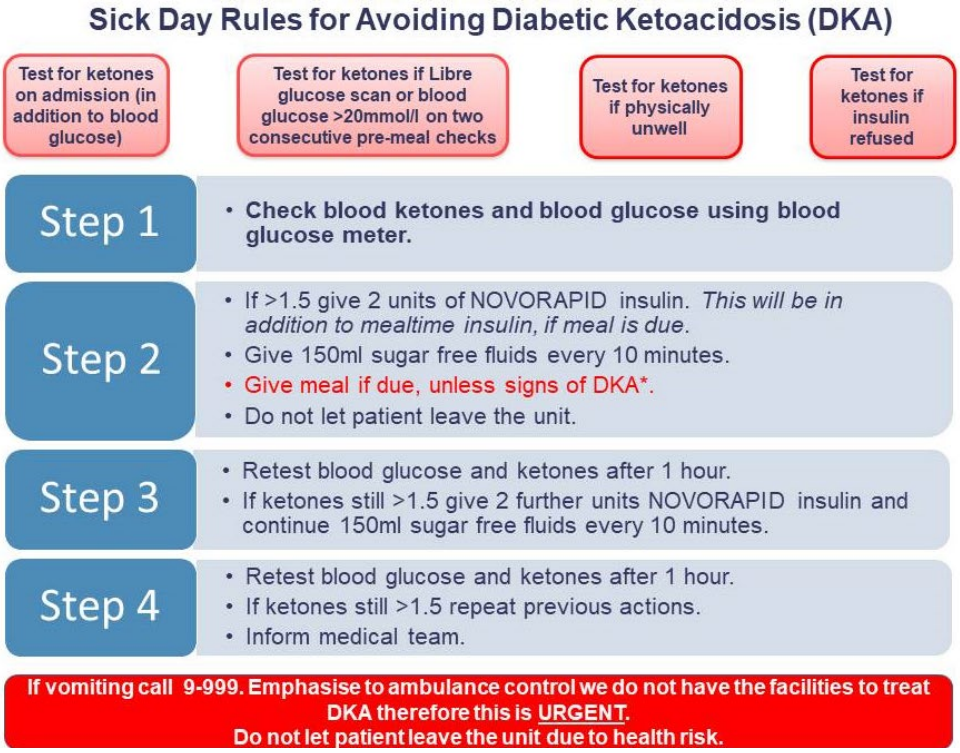
	Add a fat source: 1 tsp oil/1 level tsp butter or margarine	
	Add a flavour source: e.g. a sauce, herbs, spices, etc.	
OR:	½ portion of a homemade mixed meal: 1 ½–2 rounded serving spoons lasagne, shepherd's pie, pasta bake (includes the vegetable portion).	18.5–25.5g
	¼ of a whole regular size thin crust pizza, plus a vegetable/salad portion.	
Include a second course, e.g.: 6.5	**Dairy option**: ½ standard pot of whole milk yogurt (e.g. Müller Fruit Corner, Activia or Greek-style), or ½ rice pudding/custard pot, or 1 scoop of ice cream.	6.5–11g
	Add some fruit: ½ a portion of fruit	3–10g
SUPPER SNACK:	1 cup semi-skimmed milk + 1 digestive biscuit	20g

* Cup measures refer to UK measuring cups.

FLUIDS 	Aim for 6–8 mugs of fluid a day. Fluid requirements may be higher in hyperglycaemia due to polyuria, meaning affected patients may need access to more water. All fluid intake should be recorded.

Appendix B: Inpatient protocols for the management of hyperglycaemia and ketones in adults[f]

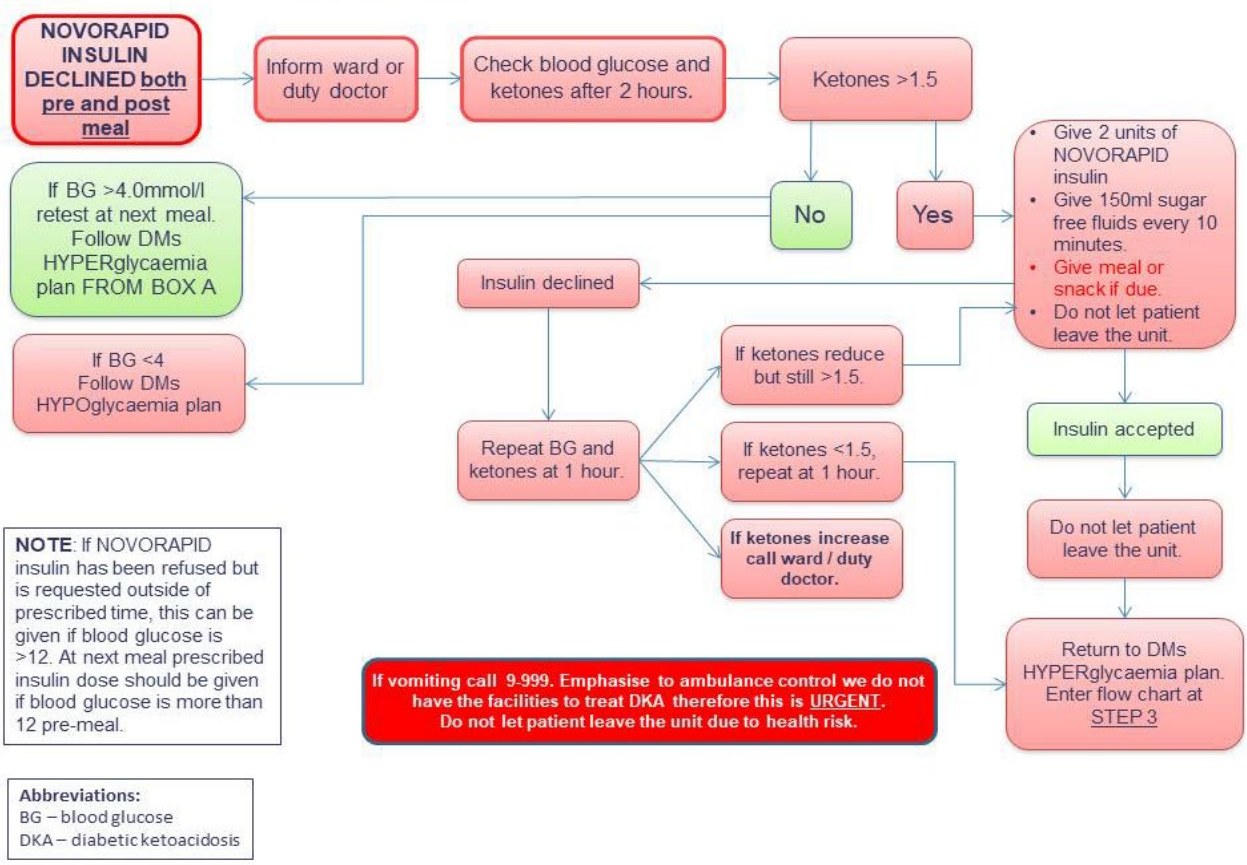
Stage 1: Ketone management plan



[f] Developed by the Wessex ComPASSION Team.

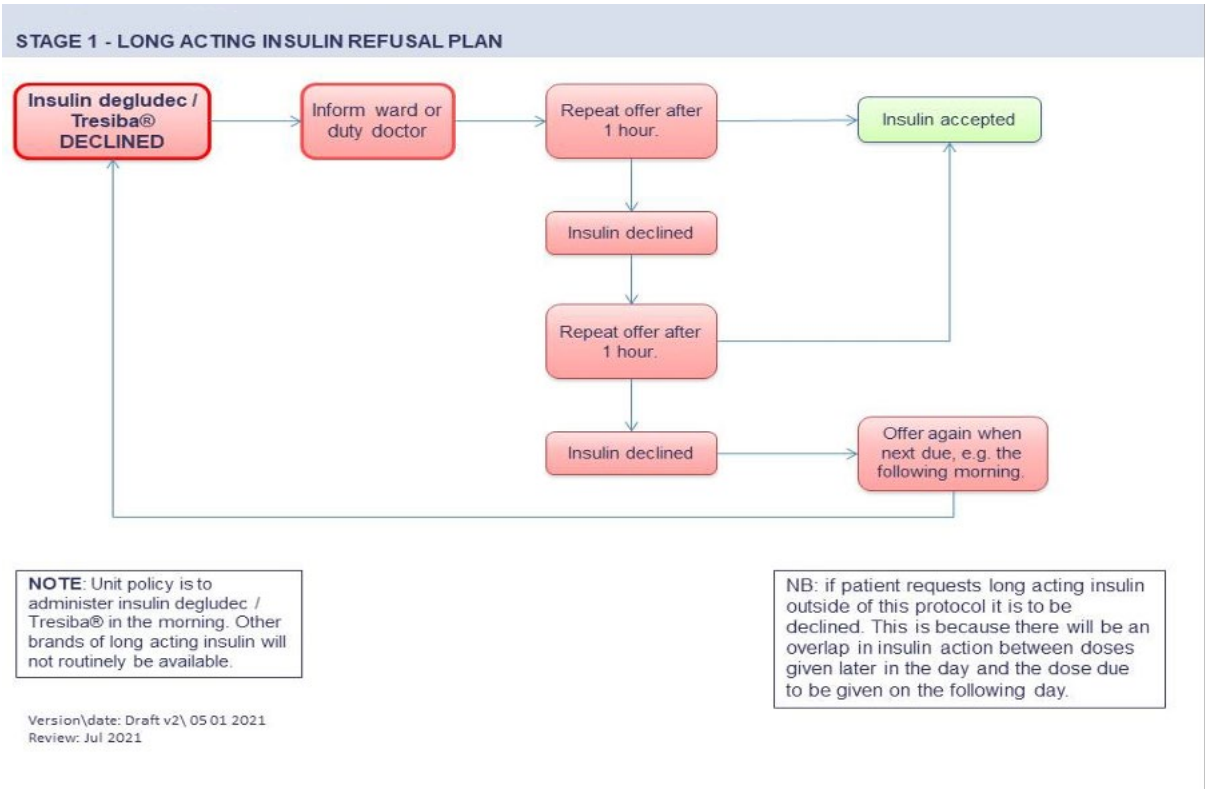
Appendix C: Inpatient protocols for the management of refusal of short- and long-acting insulin in adults[9]

Stage 1: Rapid-acting insulin refusal plan

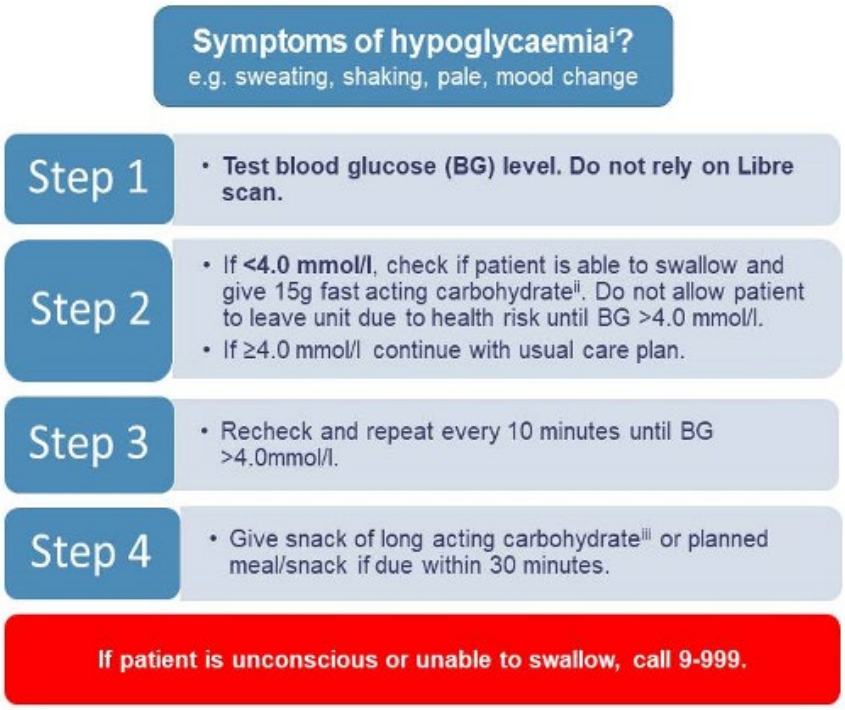


[9] Developed by and adapted from Wessex ComPASSION Project.

Stage 1: Long-acting insulin refusal plan



Diabetes hypoglycaemia management plan



[i] Patient may experience symptoms of hypoglycaemia at blood glucose levels above 4mmol/l. If this is the case, reassurance should be given. If blood glucose is 4-12 mmol/l 15g of fast acting carbohydrate can be offered:

- Orange/apple juice 50ml
- Glucose/dextrose tablets x1.5
- Lucozade 60ml
- Cola type drink (not diet) 50ml
- Jelly babies x1.5

No need to follow up with long acting carbohydrate.
If blood glucose >12, offer reassurance and do not offer fast acting carbohydrate.

[ii] 15g of suitable fast acting carbohydrate:

- Orange/apple juice 150ml
- Glucose/dextrose tablets x4
- Lucozade 170ml
- Cola type drink (not diet) 150ml
- Jelly babies x4
- 15g glucose gel

Follow up with long acting carbohydrate

[iii] Suitable long acting carbohydrate:

- Small piece of fruit
- Piece of toast
- Biscuits x2
- Small bowl of cereal
- Small sandwich

NB Glucagon
Due to reduced effectiveness Glucagon is not routinely recommended, as those omitting insulin with low BMI or restricted carbohydrate intake may have inadequate glycogen stores.

Appendix D: Inpatient insulin management care plan for adults with type 1 diabetes and eating disorders[h]

Date of care plan:

Patient details:

1. Stages of insulin management

Select (☑)

Stage 1	• Person with diabetes (PWD) to scan Libre before each meal. • Insulin administered by staff.	
Stage 2a	• PWD to scan Libre before each meal. • Staff dial up insulin dose. PWD administers insulin under staff supervision.	
Stage 2b	• PWD to scan Libre before each meal. • PWD dials up insulin dose. PWD administers insulin under staff supervision.	
Stage 3	• PWD to scan Libre before each meal and dials up insulin dose. • Insulin administered by PWD with staff support as required.	
Stage 4	• PWD to scan Libre before each meal and dials up insulin dose. • Insulin administered independently by the PWD.	

[h] Developed by and adapted by Wessex ComPASSION Team, The Royal Bournemouth and Christchurch Hospital NHS Foundation Trust, Dorset HealthCare University NHS Foundation Trust.

2. Stages of insulin dosing

		Select (☑)
Stage 1	• Fixed doses basal and bolus meals only (no snacks) no dose adjustments + sick day rules (see Ketone Management Plan)	☐
Stage 2	• Boluses added for snacks + fixed basal + bolus dosing + sick day rules (see Ketone Management Plan)	☐
Stage 3	• Dose adjustments added to fixed doses bolus and basal + sick day rules (see Ketone Management Plan)	☐
Stage 4	• Introduce carbohydrate counting + dose adjustments + sick day rules (see Ketone Management Plan)	☐
Other		☐

3. Current doses

Long-acting insulin

Specify:.....

No. of Units ☐

Rapid-acting insulin

Specify: .....

No. of Units: Breakfast ☐

Lunch ☐

Evening meal ☐

Snacks ☐

- **Carbohydrate ratios when appropriate**

  1 unit of insulin for every ..... grams carbohydrate breakfast

  ..... grams carbohydrate lunch

  ..... grams carbohydrate evening meal

- **Adjustment doses**

  1 unit of insulin will reduce blood sugar by ☐ Units

4. Insulin refusal

- Encourage insulin pre-meal as per prescription chart.
- If not accepted within 5 minutes, continue with meal and offer again ONCE post meal.

- If insulin refused, follow Ketone Management Plan.

5. Carbohydrate refusal

- If a person refuses their meal or the carbohydrate part of their meal or a snack and have taken their rapid-acting insulin for that meal or snack, monitor their blood glucose levels every hour for the next four hours.
- If the person will accept carbohydrate in the interim this should be accommodated, e.g.: A milky drink with 2–3 biscuits, 2 slices of toast with spread and jam or the snack previously declined. Adjust accordingly for ½ portions or the person's tolerance to accepting some carbohydrate.
- If the person's blood glucose levels fall below 4mmol/L treat as per the hypoglycaemia management plan.

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Abbreviations

BD	*bis die* (twice daily)
BMI	body mass index
CVD	cardiovascular disease
DKA	diabetic ketoacidosis
GLP-1	glucagon-like peptide 1
HbA1C	haemoglobin A1C
mBMI	mean body mass index
PWD	person with diabetes
RMN	registered mental health nurse
SEDU	specialist eating disorder unit
SGLT-2	sodium-glucose cotransporter-2
TDS	*ter die sumendum* (three times daily)
T1DE	type 1 diabetes and eating disorder
TREND-UK	Training, Research and Education for Nurses in Diabetes-UK