A GUIDE TO RISK ASSESSMENT FOR TYPE 1 DIABETES AND DISORDERED EATING (T1DE); medical, psychiatric, psychological and psychosocial considerations

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Abbreviations List

ALP	Alkaline phosphatase
ALT	Alanine transaminase
AN	Anorexia nervosa
AST	Aspartate aminotransferase
BMI	Body mass index
BN	Bulimia nervosa
BNF	British National Formulary
CBT	Cognitive behavioural therapy
CVD	Cardiovascular disease
DKA	Diabetic ketoacidosis
DSM-5	The Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition
ECG	Electrocardiogram
GAD-7	Generalized Anxiety Disorder-7
GGT	Gamma-glutamyl transpeptidase
GLP-1	Glucagon like peptide-1
Hb	Haemoglobin
HbA1c	Glycosylated haemoglobin
HCP	Health care professional
IV	Intravenous
JBDS	Joint British Diabetes Societies
MAOI	Monoamine oxidase inhibitor
MDT	Multidisciplinary team
MHA	Mental Health Act
NHS	National Health Service
NICE	National Institute for Health and Care Excellence
OCD	Obsessive compulsive disorder
PD	Personality disorder
PHQ-9	Patient Health Questionnaire-9
PTSD	Post-traumatic stress disorder
PWD	Person with diabetes
SIO	Syndrome of insulin omission
SSRI	Selective serotonin reuptake inhibitor
TCA	Tricyclic antidepressants
T1DE	Type 1 diabetes and disordered eating

The Wessex ComPASSION Pilot Project

Following an invitation to bid from the NHS Diabetes Treatment and Care Programme Board, NHS organisations across Hampshire and Dorset were accepted as one of two national pilots to trial, test and evidence the impact of an integrated diabetes and mental health pathway for the assessment, referral and treatment of people with both type 1 diabetes and disordered eating (T1DE).

The Wessex ComPASSION pilot has sought to establish patient pathways and protocols to best care for this patient group. This guide to risk assessment for the management of people with T1DE uses, where possible, published evidence to develop these recommendations but in the absence of such evidence, expert opinion and accumulated professional experience among the multidisciplinary

writing group have been utilised. The writing group aims to offer practical guidance and principles of care that can be employed to improve the early detection and quality of care for patients where both conditions co-exist. There is an emphasis on collaboration between clinical specialties to offer a joint physical and mental health clinical pathway that integrates effective, safe care and management of both conditions.

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This document will continue to be reviewed and re-issued to reflect new and emerging evidence. This guide is designed to offer information and advice to health care professionals in complement to and not to replace local guidance and professional judgement.

1.0 INTRODUCTION

1.1 Risk factors for people with type 1 diabetes developing an eating disorder

The requirements of self-management of type 1 diabetes and the biological and psychological consequences of living with this long term health condition are inherently tied to an increased risk of the development and maintenance of an eating disorder for a number of reasons:

- The need to carefully read food labels.
- The focus on weight and food intake at diabetes clinic appointments.
- The need to eat to treat hypoglycaemia, which can cause weight gain and can sometimes be counterintuitive (e.g. having to eat when not hungry), contributing to feelings of guilt.
- An increased awareness of the body through the need for self-monitoring of weight and glucose levels.
- A constant awareness of carbohydrates or calories in food in order to match insulin requirements for meals.
- The effort put into diabetes self-management does not always equate to the outcomes of these efforts causing frustration, guilt and shame, which may be influenced and reinforced by health care professionals' (HCPs') responses to diabetes data.
- Unrealistic expectations set by healthcare providers regarding blood glucose levels.
- The language of diabetes which can be polarised e.g. good versus poor control; in range versus out of range.
- A heavy focus on numbers and the assumptions about the link between numbers and effort or value / self- worth.
- Difficulty maintaining a desired weight.
- Weight loss prior to diagnosis and regain on starting insulin, cementing an association between insulin and weight gain.
- Others' scrutiny of and assumptions about diet and diabetes management e.g. family, friends and colleagues.

The interaction between these elements of living with type 1 diabetes and biological, psychological and social predisposing factors to developing an eating disorder[1] can precipitate the development of an eating disorder in the context of type 1 diabetes. In addition, the diagnosis of type 1 diabetes in someone who has previously had an eating disorder can act as a trigger for a re-emergence of the eating disorder because of the above factors.

Acknowledging the impact of some of these diabetes specific factors and addressing those that can be modified within diabetes health care could act as a preventative measure for future development of eating disorders within those with type 1 diabetes. For example, attention to the language used in diabetes consultations, the expectations set at diagnosis about management of glucose levels, the focus on weighing and the messages communicated in relation to weight. In order to minimise the potential for triggering previous eating disorders, it is also recommended that HCPs ask those newly-diagnosed with type 1 diabetes about previous eating disorder experiences.

Whilst there will be similarities with other eating disorders, if weight and shape concerns do develop within the context of type 1 diabetes the complexity of this presentation, most notably the unique

ability to control weight and shape by omitting insulin, means there are additional inherent risks including higher rates of short and long term complications of diabetes, a reduced quality of life and a reduced life expectancy[2].

As with those who have eating disorders but do not have a diagnosis of type 1 diabetes, the factors involved in the assessment of risk in people with T1DE include:[3]

- medical risk
- psychological and psychiatric risk
- psychosocial risk.

This document aims to bring together information relating to the risks associated with eating disorders in the context of adults with type 1 diabetes and give guidance on the appropriate management of these risks. It aims to provide information that is relevant to a range of health care professionals including diabetes specialists, eating disorder specialists, GPs, practice nurses, mental health nurses, dietitians and other allied health professionals working with this group of people. It can be applied across settings including outpatients, in both primary and secondary care, medical inpatients, general psychiatric inpatients and eating disorder specialist units. Whilst the document's scope does not include paediatrics it is acknowledged that many of the principles discussed will have relevance to paediatric care.

1.2 Definition and diagnostic criteria

For the purposes of this document the term Type 1 Diabetes and Disordered Eating (T1DE) has been used to describe the presence of an eating disorder in the context of type 1 diabetes. Proposed diagnostic criteria are outlined in Box 1.

Box 1: proposed T1DE diagnostic criteria

People with type 1 diabetes who present **with all 3 criteria:**

1. Disturbance in the way in which one's body weight or shape is experienced or intense fear of gaining weight or of becoming overweight.
2. Recurrent inappropriate direct or indirect* restriction of insulin (and/or other compensatory behaviour**) in order to prevent weight gain.

   *Indirect restriction of insulin refers to reduced insulin need/use due to significant carbohydrate restriction.

   ** Dietary restriction, self-induced vomiting, laxative use, excessive exercise.
3. Person must present 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 in areas of functioning.

A range of presentations of T1DE may occur dependent on the behaviours used to control weight. Omission of insulin, restriction of food, over exercise, self-induced vomiting and abuse of laxatives or diuretics may all be present.

1.3 Identification of T1DE

The risks associated with T1DE cannot be managed if it is not identified and engaging this group of people within diabetes clinics is vital. There are a number of factors that can impact upon the likelihood of identification of people with T1DE in diabetes services:

- Feelings of guilt, shame and judgement about eating habits, blood glucose levels and weight or body shape resulting in disengagement from services.
- Previous negative experiences with HCPs.
- Low confidence in diabetes HCPs to talk about emotional matters[4].
- Finding current habits rewarding e.g. compliments regarding weight loss.
- Denial of the seriousness of symptoms and conditions.
- Fear on the part of HCPs that asking about insulin restriction might inadvertently teach people this method of weight loss.
- Lack of education about T1DE for HCPs.
- No routine use of T1DE screening tools in diabetes healthcare settings.
- Concern that identification would open up a set of problems that the diabetes HCP has little confidence to contain or knowledge of where to refer onto[5].

The following approaches will improve the likelihood of identification of people living with T1DE in diabetes healthcare settings:

- Education for staff about T1DE, in particular red flags that might suggest T1DE (See Box 2).
- Building a respectful, non-judgemental, empathic relationship to create a safe environment for the person with an eating problem to open up and ask for support.
- Communication skills training for staff to increase confidence in being able to ask about T1DE.
- Consideration of language used[6].
- Being curious about why those who disengage from services have done so.
- Having information available within diabetes healthcare settings e.g. posters / leaflets / online resources to open up the conversation and provide more information.
- Keeping an open door to return to the conversation at another time.
- Building links between local diabetes services and eating disorder services.

Box 2: red flags for T1DE

- Increase in HbA1c or erratic blood glucose levels
- Multiple diabetic ketoacidosis (DKA) or near DKA episodes
- Dietary restriction or binge eating
- Secrecy about diabetes management
- Weight loss history or fear of weight gain
- Disengagement from services
- Diabetes distress
- Fear of hypoglycaemia
- Depression and anxiety
- Concerns about body image.

Screening tools can be considered for the identification of T1DE in diabetes healthcare settings however there continues to be debate over whether validated general screenings, modified screenings, or T1DE specific screening tools are the best approach when trying to identify eating disorders in those with type 1 diabetes[7].

The ComPASSION project amended a routine diabetes clinic questionnaire to include two questions that explore the possibility of weight and shape concern and used this as a T1DE specific screening tool. See Appendix A. It should be noted this tool has not been validated. Options for validated screening tools are outlined below.

Problem Areas in Diabetes (PAID) scale[8]
Diabetes Distress Scale (DDS) 2[9]
Diabetes Distress Scale (DDS) 17[10]
Diabetes Eating Problems Survey – Revised (DEPS-R)[11]
Generalized Anxiety Disorder 7-item (GAD-7) scale[12]
Patient Health Questionnaire-9 (PHQ-9)[13]
Eating Questionnaire (EDEQ 5.2)[14]
Eating Disorders Quality of Life (EDQOL)[15]
Work and Social Adjustment Scale[16]

2.0 MEDICAL RISK

The medical risk for an individual with T1DE arises from one or more of the following:

- insulin omission
- concomitant use of diabetes adjunctive therapies
- management of hypoglycaemia
- starvation
- alternative compensatory behaviours including vomiting, laxative misuse and exercise
- reinsulinisation / refeeding.

2.1 Insulin omission

Insulin is a hormone which plays a number of roles in the body's metabolism. Amongst these, insulin regulates how the body uses and stores glucose and fat. In type 1 diabetes, where the insulin producing cells of the pancreas have been destroyed and insulin is no longer produced, glucose can no longer be taken up by the cells of the body and without the provision of exogenous insulin blood glucose levels will rise. As carbohydrate is no longer available to the body as an energy source there is a switch to fat metabolism in order to ensure a continued supply of energy to the body. This results in the production of ketones which if left unchecked will result in, amongst other things, rapid weight loss due to the breakdown of the body's fat stores.

Syndrome of insulin omission (SIO) is a term used when a person with diabetes (PWD) deliberately and regularly reduces or misses their insulin with the specific aim of reducing weight or affecting their body shape leaving them at risk of the short term consequences of raised blood glucose levels and raised ketones as well as the long term consequences of raised blood glucose levels.

2.11 Short term risks of insulin omission

a) Raised blood glucose levels (hyperglycaemia)

Symptoms of hyperglycaemia include**:**

- thirst and dry mouth
- polyuria
- blurred vision
- tiredness
- irritability
- delayed wound healing
- weight loss
- genital itching / thrush.

Management of hyperglycaemia: 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)[17]. This is achieved through provision of background insulin, which is *always* required even when a PWD is not eating, alongside provision of rapid acting insulin, which is required when carbohydrates are eaten and for corrections of high glucose levels.

For those with SIO, 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 reinsulinisation (see section 2.6) and treatment induced retinopathy[18] and neuritis[19] 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[20,21,22,23]. Retinal screening and assessment should therefore have occurred prior to initiating treatment to achieve glycaemic control[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). As 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 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 (HCPs) to accept these high levels however it is a temporary measure with the aim of progressively increasing insulin doses to achieve recommended targets. See section 2.6 and Appendix B for further discussion and an example protocol for the reintroduction of insulin.

b) Raised ketones

Insulin deficiency is associated with the accumulation of ketoacids in the extra-cellular fluid and a loss of bicarbonate anions. A resulting metabolic acidosis ensues which is typically represented by a low pH, low bicarbonate level and a raised anion gap. This is usually coupled with hyperglycaemia which leads to the osmotic loss of sodium and water into the urine and a marked contraction of the extra-cellular fluid volume. If severe or prolonged, this can result in DKA which is associated with significant morbidity and mortality.

Measurement using ketone meters (e.g. Abbott Neo meter or Libre Reader) is recommended rather than urine ketone testing strips as the results are more accurate.

As ketone levels rise, the body attempts to remove them by excreting them in the urine. Consequently, the first signs of hyperketonaemia are increased urination and thirst. If ketone formation is not resolved by giving enough extra insulin the person can become acidotic which ultimately can result in DKA.

Initial symptoms of raised ketones include those outlined for hyperglycaemia above as well as nausea, loss of appetite and shortness of breath. Later symptoms requiring immediate attention are:

- vomiting (may start with nausea)
- weakness
- dehydration
- fruity odour on breath (may smell like 'pear drop' sweets or acetone)
- abdominal pain
- drowsiness
- confusion
- rapid, shallow breathing.

Clinically DKA is characterised by hyperglycaemia, ketonaemia and acidosis[26]:

- blood glucose > 11mmol/L or a known diagnosis of diabetes
- ketonaemia (blood ketones) > 3mmol/L
- bicarbonate < 15mmol/L and/or venous pH < 7.3.

If left untreated DKA can result in:

- acute kidney injury, as a result of severe dehydration
- cerebral oedema - symptoms include headache, bradycardia, rising blood pressure, change in neurological status, focal neurological signs, convulsions, papilloedema
- respiratory distress
- death.

DKA is a medical emergency and requires urgent evaluation in an emergency medical setting.

Management of raised ketones: Sick-day rules are commonly used to curtail or avert DKA. Local guidance available at https://www.bertieonline.org.uk/ advises[27]:

- <0.6 mmol/L – normal result.
- 0.6 - 1.5mmol/L – indicates need for extra insulin. The PWD requires their usual adjustment dose of rapid acting insulin (e.g. Novorapid). Recheck blood glucose and ketones in 1 - 2 hours after insulin dose. Increase sugar free fluid intake.
- >1.5mmol/L – indicates risk of DKA. The PWD requires double their usual adjustment dose of rapid acting insulin without delay. Increase sugar free fluid intake. Recheck blood glucose and ketone levels in 1 - 2 hours after insulin. If ketones remain >1.5mmol/L, repeat double adjustment dose without delay and continue to increase intake of sugar free fluids.

Alternative national guidance for those with type 1 diabetes when they are ill is also available[28].

For people with SIO asking them to follow this guidance in full and take appropriate doses of insulin to correct ketones may be challenging, or refused, even when they know it is for their own health and wellbeing. In many cases DKA can be averted by preventing blood ketones rising above 1.5mmol/L[19] and in cases of SIO the HCP 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 PWD *safe* whilst accepting it does not eliminate ketones to a

level usually recommended. For example: 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 PWD may feel able to accept this regime where the full recommended insulin dose would not be accepted. See Appendix C for example inpatient protocols for the management of hyperglycaemia and ketones.

Management of DKA: guidance for treating DKA has been published by the Joint British Diabetes Societies (JBDS) Inpatient Care Group[26].

There is universal agreement that the most important initial therapeutic intervention in DKA is appropriate fluid replacement followed by 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 < 0.6 mmol/L and venous pH > 7.3[26]. Blood glucose levels will reduce as the ketones reduce.

c) Dehydration

Dehydration is more likely to be seen in those with raised blood glucose and ketone levels and can rapidly lead to medical crisis through circulatory 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 an outpatient setting, in the absence of DKA oral replacement is preferable.

d) Hyperglycaemia and cognitive functioning

Hyperglycaemia (>15mmol/L) is associated with slowing of all cognitive performance tests and errors made[29]. For those with T1DE where starvation is a feature, this too is associated with impaired cognitive function (see section 2.42). Impaired cognitive function has the potential to interfere with utilisation of therapeutic sessions with the multidisciplinary team (MDT) because of impaired concentration, retention and processing as well as emotional numbness and disconnection. Consideration should also be given to the impact of changes in cognitive function on other aspects of daily living with psychiatric, psychological and psychosocial risks being assessed and addressed (see sections and 3.0 and 4.0).

2.12 Long term risks of insulin omission

The long term risks of insulin restriction or omission and subsequent complications that can develop in a person with T1DE are the same as in the general population with type 1 diabetes. The most

common physiological complications can be broadly divided into two categories; microvascular and macrovascular.

Microvascular:

- eyes (retinopathy)
- kidneys (nephropathy or renal disease)
- nerve tissue (neuropathy).

Macrovascular:

- blood vessels in the heart, brain and limbs (cardiovascular disease (CVD)).

In people with T1DE, especially where insulin is restricted or omitted, these micro and macrovascular complications may arise earlier. In particular there is an increased risk of microvascular complications occurring, including retinopathy and neuropathy[30,31,32]. This is most likely to be a consequence of long term exposure to high blood glucose levels, as eating disorders in people with type 1 diabetes are associated with blood glucose levels above recommended targets[33], an established risk factor for developing microvascular complications[25].

a) Microvascular complications

Retinopathy: diabetic retinopathy refers to damage of the blood vessels in the back of the eye (retina) caused by high blood glucose levels. These changes, if left undiagnosed and untreated, can cause blindness. The National Health Service (NHS) Diabetic Eye Screening programme recommends annual retinal screening for people with diabetes[34]. We do not currently know the optimal screening frequency for this cohort and should be guided by clinical need or significant deterioration in vision. Encouragement should be given to access retinal screening given the potential for this group of people to disengage from services and the potential for those with eating disorders to self-neglect (see sections 4.1 and 4.5).

In the management of T1DE consideration must also be given to the evidence that rapid improvement of blood glucose levels has been found to cause early worsening of diabetic retinopathy[18] (see section 2.11(a)).

Nephropathy (kidneys): high blood glucose levels cause damage to the blood vessels in the kidney. Damage to the kidneys can manifest in both the kidneys' ability to filter out toxins as well as their ability to keep in protein. Diabetes is one of the most common causes of end stage kidney failure requiring dialysis in developed countries.

The first sign of nephropathy is leakage of very small amounts of protein (microalbuminuria) into the urine. Screening and calculation of albumin : creatinine ratio (ACR) in type 1 diabetes is recommended[35]. Early nephropathy is usually treated with an angiotensin-converting enzyme (ACE) inhibitor and by improved blood glucose and blood pressure management. In the later stages of renal disease appropriate dietary advice should be provided by an appropriately qualified registered dietitian and should consider potassium, phosphate, salt and energy intake, and ensure malnutrition is prevented. Restriction of protein intake is not recommended[35].

Neuropathy: this refers to damage to nerves which can be in any part of the body. In the feet, this can be further complicated by any changes to blood flow. This combination of nerve problems and reduced blood flow can lead to reduced sensation in the feet and increased risk of wounds and ulcers developing as well as poor healing. In those with T1DE poor blood flow may be compounded by starvation where dietary restriction has resulted in low body mass index (BMI) and in extreme starvation peripheral neuropathy associated with malnutrition can further complicate the clinical presentation (see section 2.43(b)).

Symptoms of neuropathy include pins and needles, a tingling sensation, numbness or pain. The sensation in the person's feet should be checked at a minimum in annual review, more often if risk factors are present[36]. People with diabetes should be encouraged to examine their feet regularly to self-identify changes at an early stage[36]. As with retinal screening, encouragement should be given to those with T1DE to engage in annual foot checks and self-examination, given the potential for this group of people to disengage from services and the potential for those with eating disorders to self-neglect (see section 4.1 and 4.5). More information on diabetic foot care can be found elsewhere[36,37].

https://www.nice.org.uk/guidance/ng19
https://www.diabetes.org.uk/guide-to-diabetes/complications/feet/taking-care-of-your-feet

Diabetic gastroparesis: this is a chronic disorder of the stomach characterised by delayed gastric (stomach) emptying in the absence of a mechanical obstruction. It is thought up to 80% of severe cases in those with diabetes occur in women in their 20s and 30s[38,39] and is associated with suboptimal glucose levels and reduced quality of life[40]. The risk of other features of autonomic neuropathy is also higher in people with diagnosed gastroparesis[41]. In someone with diabetes, gastroparesis can be caused through two main mechanisms:

- High glucose levels (and ketoacidosis) slowing down gastric emptying (this has been demonstrated in people without diabetes).
- Over time high glucose levels causing damage to the vagus nerve which interrupts the usual coordination of gut movement and release of food from the stomach.

Common symptoms include:

- vomiting
- early satiety
- bloating
- poor oral intake resulting in an energy-deficient diet
- weight loss
- deficiencies in vitamins and minerals[38,39].

Hard masses of food (bezoars) causing potential gastrointestinal blockages can also occur.

Diagnosis is essential but can be challenging as it is often a diagnosis of exclusion. In T1DE further challenges present as many of the features of gastroparesis may be related to eating disorder behaviours including gastric stasis and its associated early satiety and bloating as a result of starvation, loss of bowel motility as a result of laxative abuse, weight loss and associated poor diet as a result of dietary restriction and vomiting as a purging behaviour. It is also important to rule out

other causes of gastroparesis that could co-exist such as some autoimmune endocrine disorders and common medications (opioids, glucagon like peptide -1 (GLP-1) agonist).

The management of someone with T1DE presenting with the symptoms of gastroparesis therefore requires careful consideration to elucidate the cause of the symptoms and the most appropriate flow of interventions and investigations. If gastroparesis is found to be present it is not possible to reverse any nerve damage, but normalisation of glucose levels may help with acute symptoms as can medications that promote food moving through the gut (pro-kinetics) and anti-emetics.

As hyperglycaemia and ketoacidosis can promote gastroparesis, optimisation of glycaemic levels[42] and consideration of continuous subcutaneous insulin infusion (CSII) or insulin pump therapy[34] are recommended. In T1DE however the benefits of taking a gradual approach to improving glycaemic control and the potential pitfalls of pump therapy need to be considered (see sections 2.11(a) and 2.51(a)).

Nutritional management should focus on assessment and correction of nutritional status, relief of symptoms, and improvement of gastric emptying. Dietary consensus[43,44] includes recommendations for:

- low-fat, low fibre meals
- small frequent meals
- complex carbohydrates
- meals with a high liquid content.

A small-particle-size diet may cause symptom relief for those experiencing vomiting[41]. Oral nutritional supplements may be required to fulfil energy requirements, and in those with severe gastroparesis, enteral nutrition can improve symptoms and reduce hospitalisations[45].

In T1DE any dietary assessment and advice for gastroparesis needs careful consideration of the eating disorder and referral to a registered dietitian is essential.

b) Macrovascular complications

Type 1 diabetes increases the risk of heart attacks, strokes and peripheral vascular disease due to a multiplicity of metabolic risk factors including predominantly hyperglycaemia but also high blood pressure and high blood cholesterol. Family history of heart disease is also an important factor[34]. The risk of cardiovascular complications appears to be similar in women with type 1 diabetes versus men[46] and in all those with type 1 diabetes cardiovascular risk factors should be assessed annually[34].

Given that eating disorders in people with type 1 diabetes can be associated with blood glucose levels above recommended targets[33], those with T1DE can be predicted to be at increased risk of CVD. Alongside working with a person with T1DE to optimise their blood glucose levels other steps can be taken 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 (see section 2.51(a) and (e)). 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[47].

2.2 Adjunctive therapies

Although not licensed in the UK for use in type 1 diabetes GLP-1 agonists have, in some studies, been demonstrated to reduce HbA1c, body weight and reduce total daily doses of insulin when used by people with type 1 diabetes alongside insulin. They have however been associated with a greater risk of hypoglycaemia and DKA[48]. To minimise these risks, careful glucose monitoring and adjustment of insulin doses is essential.

Sodium-glucose cotransporter-2 (SGLT-2) inhibitors are licenced for use in the United Kingdom where a person with type 1 diabetes is overweight and have been demonstrated to contribute to better glycaemic control, lower body weight and reduced total daily doses of insulin in people with type 1 diabetes without increasing the risk of hypoglycaemia. As with GLP-1 agonists they are however associated with an increased risk of DKA, particularly euglycaemic DKA where ketones and acidosis are present but at a much lower glucose level which can make identification and treatment difficult[48].

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

2.3 Low blood glucose levels (hypoglycaemia)

For a person with type 1 diabetes, hypoglycaemia is defined as a blood glucose below 4mmol/L, regardless of whether the person has symptoms of hypoglycaemia. It is caused when there is an imbalance between three key elements unless the person's insulin dose and/or carbohydrate intake is adjusted accordingly:

- The glucose lowering effect of the insulin dose.
- The glucose consumed from carbohydrate in food and drink.
- The glucose lowering effect of physical activity.

2.31 Symptoms of hypoglycaemia

Symptoms of hypoglycaemia are outlined in Box 3.

Box 3: symptoms of hypoglycaemia[49]

Physical symptoms of hypoglycaemia

- feeling shaky
- sweating
- looking pale
- palpitations and a fast pulse
- tingling lips
- blurred vision
- being hungry
- having a headache.

Cognitive symptoms of hypoglycaemia

- feeling disorientated
- mood changes, such as feeling tearful, anxious or irritable
- tiredness
- struggling to concentrate/think clearly.

If left untreated hypoglycaemia can progress to loss of consciousness, collapse and death.

2.32 Causes of hypoglycaemia[27]

Factors that can increase the risk of hypoglycaemia occurring in those administering insulin include:

- A mismatch between insulin and carbohydrate intake e.g. missing a meal or snack after injecting insulin.
- Accidentally injecting too much insulin e.g. by miscalculating, using the wrong insulin pen or dialling up the wrong dose of insulin.
- Physical activity without adjustment of food or insulin.
- Consuming alcohol the same day or the day before[50].
- Hot weather.
- Recent hypoglycaemia.
- Intentional overdose (see section 3.4).

For those with T1DE the following can also contribute to hypoglycaemia if insulin is being administered:

Excessive exercise: see section 2.51(e).

Low carbohydrate diets: see section 2.51(c).

Low glycogen stores: people with T1DE may be at higher risk of hypoglycaemia because factors such as food / carbohydrate restriction, excessive exercise and a low BMI can result in depletion of glycogen stores in the body. With inadequate glycogen stores to draw on between meals and overnight, hypoglycaemia can occur even in those without type 1 diabetes[51].

Delayed gastric emptying: a person experiencing delayed gastric emptying as a result of diabetes related gastroparesis, starvation or both may experience hypoglycaemia as insulin may be absorbed and start acting to reduce blood glucose levels before carbohydrates from the meal are digested and absorbed[52].

Purging: if a person with T1DE purges after a meal or binge and has taken rapid acting insulin the likelihood of hypoglycaemia will be increased.

Binge eating: a person may feel guilt and shame after an episode of binging and may overcompensate with a large insulin dose causing hypoglycaemia.

2.33 Consequences of hypoglycaemia

Regardless of the cause, hypoglycaemia can be dangerous. It can lead to seizures, poor balance and falls[50] and severe hypoglycaemia occasionally may lead to long term neurological sequelae or death. There is also a notable effect from hypoglycaemia on increasing risk of cognitive impairment later in life[53]. The behaviours motivated by weight and shape concern outlined above that increase the risks of hypoglycaemia therefore need to be addressed within the management of T1DE if insulin is being administered.

2.34 Treatment of hypoglycaemia

a) Oral treatment

Oral hypoglycaemia treatments are recommended for patients that are conscious and can safely swallow. They should provide 15-20g of rapidly absorbed carbohydrate (see Table 1).

Table 1: Oral hypoglycaemia treatments providing 15 – 20g rapid acting carbohydrate

Treatment	Portion
● Pure orange / apple juice	150 - 200ml
● Lucozade® Energy Original	170 - 225ml*
● Coca-Cola® Classic	140 - 190ml (mini can 150ml)
● Jelly babies	3 – 5 standard sweets
● Jelly beans	14 - 18g (12 - 16 jelly beans)**
● Marshmallows	3 - 4 (large)
● Dextro Energy	5 – 7 tablets
● GlucoTabs	4 – 5 tablets
● Hypoglycaemia treatment gel e.g. Glucogel	1 ½ - 2 tubes

*Note the new Lucozade recipe results in a higher volume as a treatment.

**Jelly babies are superior to jelly beans as the latter has a lower glycaemic index[54].

Hypoglycaemia can result in extreme hunger and lead to loss of control over eating. Frequent hypoglycaemia can therefore lead to weight gain and in those with T1DE this can result in engagement in compensatory behaviours, e.g. insulin omission. In some with T1DE they may feel the urge to compensate even if the treatment dose has been in line with the guidance outlined above.

Conversely a person with T1DE may consume hypoglycaemia treatments in excess without taking adequate insulin in order to keep blood glucose levels high enough to generate ketones in order to achieve weight loss. This may be particularly noticed in an inpatient setting and whilst a person with T1DE should always have treatment for hypoglycaemia available to them, consideration should be given to managing the amount the person has access to.

b) Treatment of hypoglycaemia with glucagon

Glucagon, e.g. GlucaGen® HypoKit® can be given by subcutaneous injection as a hypoglycaemia treatment by someone trained to administer it[55]. This may be a healthcare professional, family member, friend or carer. Glucagon triggers the liver to release glucose from glycogen stores, raising blood glucose levels. Once the person has responded to the treatment, oral carbohydrates should be given to restore the liver glycogen in order to prevent relapse of hypoglycaemia. This may be refused by those with T1DE and therefore close monitoring of blood glucose levels would be advised.

A person with T1DE may request glucagon as a hypoglycaemia treatment in order to avoid ingesting the calories in oral hypoglycaemia treatments. This however is not recommended if oral treatment is clinically indicated. The following risk factors may mean that the person with T1DE will have depleted liver glycogen stores and therefore 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:

- 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
- repeated purging.

c) Treatment of hypoglycaemia with intravenous (IV) dextrose

If a person with T1DE is unconscious, fitting, very aggressive or nil by mouth, IV glucose will be required. This would usually be administered urgently by a paramedic or doctor following checking of airway, breathing & circulation (ABC):

- 150ml 10% IV dextrose until blood glucose >4mmol/L.
- Recheck glucose level after 10-15 minutes, it should now be >4mmol/L.
- Follow up with long acting carbohydrate[56].

As with oral treatments, those with T1DE may feel the need to engage in compensatory strategies like insulin omission in response to receiving the calories from the IV dextrose.

2.35 Pseudo (or false) hypoglycaemia

If a person with T1DE has been running high blood glucose levels for a prolonged period of time they may experience symptoms related to hypoglycaemia at normal or high blood glucose levels, particularly when insulin is reintroduced and blood glucose levels begin to decrease. These symptoms occur in response to the release of counter regulatory hormones (stress hormones), such as adrenaline. It is therefore important to note that symptoms of anxiety can mimic symptoms of hypoglycaemia[57].

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 in the first instance should be conservative i.e. encouraging rest, offering reassurance and re-checking blood glucose levels ten minutes later. However, for symptomatic relief or to manage associated anxiety, if blood glucose levels are between 4-12 mmol/L, a small dose (e.g. 5g) rapid release carbohydrate can be offered. It is important not to over treat pseudo hypoglycaemia as 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 weight loss.

It is important to note that experiencing pseudo hypoglycaemia can produce the same psychological responses as typical hypoglycaemia, such as fear and anxiety. Checking and talking through the person's blood glucose level, reassuring the person who is experiencing a pseudo hypoglycaemic event that they are safe and supporting the person not to over treat are all key to its management (see section 3.3(e)). A flow chart for the management of hypoglycaemia, including pseudo hypoglycaemia for use on inpatient eating disorder units can be found in Appendix C.

2.36 Hypoglycaemia unawareness

Hypoglycaemia unawareness occurs in people with type 1 diabetes who experience episodes of severe or recurrent hypoglycaemia. The more frequently a person experiences hypoglycaemia, the more at risk they are of losing their symptom awareness, meaning they will no longer be able to detect when their blood glucose levels drop below 4mmol/L.

Hypoglycaemia and therefore hypoglycaemia unawareness is more likely to be seen in those with T1DE who use carbohydrate restriction or excessive exercise as primary mechanisms for weight loss than in those who restrict insulin and subsequently tend to run hyperglycaemic.

Hypoglycaemia unawareness puts the person at increased risk of being unable to treat hypoglycaemia until it is extremely low and increases the risk of severe hypoglycaemia, when a person requires external assistance for recovery, such as another person actively administering carbohydrates, glucagon or other corrective actions[58].

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'[59]. It may be that in order to achieve this, psychological work may be required for example to address fear of complications or challenge the perfectionism trait often seen in eating disorders with a 'good enough' approach (see section 3.3).

2.4 Starvation

Insulin omission is the most likely compensatory behaviour to be observed in those with T1DE. Alternative compensatory behaviours such as starvation, over-exercise and vomiting may also be present. These may already be a feature of the illness at diagnosis or may emerge as a person with T1DE addresses their insulin omission. Starvation and alternative compensatory behaviours carry their own risks as outlined in sections 2.4 and 2.5.

2.41 Short term risks of starvation

Although in our experience low BMI is a less common presentation in those with T1DE, it can occur either at presentation or as the illness progresses. Most risks associated with starvation will reflect those without type 1 diabetes who are in starvation. The following recommendations have therefore been adapted from previously published guidance for the assessment of risk in those with eating disorders[3].

BMI (weight(kg)/height(m)^2) can be considered a proxy measure of medical risk in starvation however its limitations include:

- The potential for deceit.
- A reduced reliability if a rapid change in weight is observed.
- A reduced reliability at extremes of height.
- A higher risk for each BMI range for men (taller).
- A reduced reliability if features of bulimia nervosa are present.
- A reduced reliability with fluid restriction or overload.
- A reduced reliability alongside some physical comorbidities.
- Not being critical with regards to risks associated with fluid and electrolyte balance.
- Not being critical with regards to risks associated with hyperglycaemia and raised ketones.

BMI alone is therefore an inadequate marker of the risks associated with starvation. Additional recommendations for screening using a brief essential medical examination are outlined in Box 4, to be repeated as necessary.

Box 4: essential medical examination for the assessment of risk in eating disorders

- Muscle Strength:
  - The sit up and squat stand test: the patient is asked to squat down on their haunches and is asked to stand up without using their arms as levers if at all possible.
  - The sit up test: the patient lies flat on a firm surface such as the floor and has to sit up without, if possible, using their hands.
- Circulation; blood pressure, pulse, electrocardiogram (ECG) if indicated. The signs to notice are:
  - dizziness or faintness standing up from sitting
  - postural drop, i.e. the difference between lying and standing blood pressure and heart rate.
- ECG monitoring; this is needed in people with an eating disorder, based on the following risk factors[60]:
  - BMI <15
  - rapid weight loss
  - excessive exercise
  - severe purging behaviours, such as laxative or diuretic use or vomiting
  - bradycardia
  - hypotension
  - excessive caffeine (including from energy drinks)
  - prescribed or non-prescribed medications
  - muscular weakness
  - electrolyte imbalance
  - previous abnormal heart rhythm
  - if drugs which have an effect on QT interval are prescribed.
- Temperature
- Hydration; reported intake alongside examination of the skin and mucous membranes for hydration status
- Blood tests; full blood count and chemistry (FBC, ESR, UE, Cr, CK, Glucose, LFTs) are necessary if:
  - patients are in a high risk category from a previous assessment
  - they have a BMI <15
  - the BMI is less reliable due to features outlined above
  - there is a history of purging
  - in those with T1DE HbA1c is also needed.
- Other investigations; any other appropriate physical investigation pertinent to physical state. e.g. infection (note can be with normal temperature in starvation) and signs of nutritional deficiency.
- **In T1DE assessment of diabetes related complications will also be relevant.**

Table 2: medical assessment parameters alert and concern[3]

	Red – Concern	Amber - Alert
Nutrition	BMI <12 Weight loss / week >1.0kg Skin breakdown >0.2cm Purpuric rash ++	BMI <14 Weight loss / week >0.5kg Skin breakdown <0.1cm Purpuric rash ++
Circulation	Heart rate (awake)<40 bpm Systolic blood pressure <80 Diastolic blood pressure <60 Postural drop (sit-stand)>20 mmHg	Heart rate (awake) <50 bpm Systolic blood pressure <90 Diastolic blood pressure <70 Postural drop (sit-stand)>10 mmHg
ECG	Corrected QT interval (QTC) >450 Arrythmias ++	Corrected QT interval (QTC) >450 Arrythmias ++
Temperature	<34.5°C	<35°C
Hydration status	Fluid refusal Severe dehydration (10%): reduced urine output, dry mouth, decreased skin turgor, sunken eyes, tachypnoea, tachycardiac	Severe fluid restriction Moderate dehydration (5–10%): reduced urine output, dry mouth, normal skin turgor, some tachypnoea, some tachycardiac
Bone Marrow *(MCV and MCH raised – no acute risk)	White cell count (WCC) <2.0 Neutrophil count <1.0 Haemoglobin (Hb) <9.0 Platelets<110 *Acute Hb drop ++ Platelets <110	WCC <4.0 Neutrophil count <1.5 Hb <11.0 Platelets <130 *Acute Hb drop++ Platelets <130
Salt/water balance	Hypophosphataemia <0.5 Hypomagnesaemia <0.5 Hypokalaemia <3.0 Hyponatraemia <130 Urea >10	Hypophosphataemia 0.5 – 0.8 Hypomagnesaemia 0.5 – 0.7 Hypokalaemia <3.5 Hyponatraemia <135 Urea >7
Liver	Bilirubin >40 ALP >200 AST >80 ALT >90 GGT >90	Bilirubin >20 ALP >110 AST >40 ALT >45 GGT >45
Nutrition abnormalities	Hypoalbuminaemia <32 Creatinine Kinase >250	Hypoalbuminaemia <35 Creatinine Kinase >170
Diabetes	Multiple episodes of DKA	Single episode of DKA
	Refer to sections 2.11 (Short term risks of insulin omission) and 2.3 (Hypoglycaemia) above.	
Sit up or Squat-stand test scoring 0: Unable 1: Able only using hands to help 2: Able with noticeable difficulty 3: Able with no difficulty	1 or less	2

- A tachycardia in the presence of signs and investigations of severe risk may indicate imminent cardiovascular collapse.
- Those with diabetes and cardiac autonomic dysfunction may have a resting tachycardia which may mask severity of starvation.
- Bradycardia. If <40 beats per minute (bpm) admit.
- The baselines for these tests vary between labs. Any abnormal result is an indication for concern and monitoring.

Table 2 gives values of concern for each part of the recommended assessment (Box 4) . Based on scores recommended actions follow:

Scores that do not fall into the risk areas:

- Stable. Regular review and monitoring of above parameters with routine referral to eating disorders service/secondary services depending on local resources.
- Unstable. If weight is falling, support the person to come up with a plan to ensure that the nutritional state does not fall into the risk areas. Regularly review the implementation of this plan.

Score/s in the alert area:

- Regular review of parameters (circa. weekly) and assessment of capacity with urgent referral to specialised eating disorders team and appropriate medical intervention if needed.
- As this signifies medical risk this should be shared with the carer.

Score/s in the concern area:

- If outpatient - immediate contact and referral to specialised eating disorders team and physicians as the patient will need urgent specialist and medical assessment. Consider assessment of capacity.
- If inpatient – immediate contact with on-call physicians and psychiatric liaison.

2.42 Starvation and cognitive function

In starvation there is atrophy of the brain and an associated reduction in brain activity with improvement in activity in those recovered from anorexia nervosa (AN)[61]. Cognitive impairment is known to occur in those with eating disorders[62] and it is known that starvation itself is associated with both cognitive and emotional changes[63] including:

- poor concentration
- reduced problem solving ability
- increased rigidity of thought
- obsessional thinking
- irritability
- depression
- anxiety
- emotional detachment.

Hyperglycaemia (>15mmol/L) has also been shown to be associated with slowing of all cognitive performance tests[29] so this is likely to affect those with T1DE where insulin omission is a feature (see sections 2.11(d) and 3.6).

Consideration should be given to the impact of cognitive impairment on treatment progression for those with T1DE and on other aspects of daily living. Potential psychiatric, psychological and psychosocial risks should be assessed and addressed (see sections and 3.0 and 4.0)

2.43 Long term risks of starvation

a) Osteopenia and osteoporosis

It can be assumed that the risks to bone health for those with T1DE are similar to those with an eating disorder who do not have a diagnosis of type 1 diabetes and therefore the same guidance is applicable[60] https://www.nice.org.uk/guidance/ng69.

b) Neuropathy

Peripheral neuropathy is an infrequent but known long term consequence of starvation and can result from a range of nutritional deficiencies[64]. In those with T1DE peripheral neuropathy may also occur as a long term complication of diabetes (see section 2.12(a)) or with aggressive reductions in glycaemic control[19] (see section 2.11(a)). In those with T1DE where peripheral neuropathy is present differential aetiology or the possibility of co-existing causes should be considered if long term starvation is a feature of the clinical presentation.

c) Brain function

It has been noted that there are similarities in brain configuration between acutely underweight patients and long term recovered patients[65,66]. This could be either a residual consequence of the illness or a premorbid feature, which represents an illness-related biomarker[66]. Further longitudinal studies are needed to clarify the role of pre-disease traits versus starvation on these observations[65].

2.5 Compensatory behaviours

2.51 Short term risks of compensatory behaviours

a) Maintaining high blood glucose levels and ketones

Those with T1DE may rely heavily on blood ketone testing to check that they remain in a ketotic, weight losing state. A range of strategies, outlined below, may be employed to achieve a persisting ketosis. Both the strategies and the frequent checking of ketones (which fuels a misplaced reassurance that this state persists) need to be addressed.

Insulin omission:
As a general principle we would advocate pens rather than pump for insulin administration during admissions to a specialist unit for the specific treatment of T1DE. This facilitates staff confidence in a mental health setting where the complexities of managing a pump may be challenging for non-

diabetes specialist staff. Attempts to avoid insulin administration by the PWD are also more easily identified and therefore the person can be better supported to give the advised dose.

Table 3 highlights some strategies which may be employed by those with T1DE to disguise insulin omission. The list is not exhaustive.

Table 3: strategies for insulin omission / reduction:

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. ● 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 full 10 seconds which is required to deliver the full dose of insulin. ● Continuing to pinch up the skin after injection to squeeze some of the insulin out again.
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 in order to avoid a correction dose. ● Frequent checking that ketones are present and if not taking steps to increase carbohydrate / decrease insulin.

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:

Excessive carbohydrate consumption: attempts may be made to keep blood glucose and ketone levels high in order to control weight through the over consumption of high carbohydrate foods and drinks. This strategy may be present in the absence of insulin but may only emerge as insulin is

restarted in an attempt to maintain the presence of raised blood glucose levels and ketones. Support should be provided to challenge this pattern of carbohydrate consumption.

Exercise: in a person without enough circulating insulin, exercise will raise blood glucose levels and increase ketone production[67] therefore exercise may be used in T1DE as a method of increasing ketone levels.

When someone with type 1 diabetes has a blood glucose of greater than 15mmol/L and their ketones are >0.5mmol/L, they are advised to take a correction dose of insulin, and depending on the level of ketonaemia, to consider refraining from exercising[68]. This advice would apply to those with T1DE however professionals should be mindful of the perceived benefits the presence of ketones may hold for those with T1DE. Support to challenge this potentially dangerous pattern of behaviour may therefore be needed and compromises compared to ideal treatment may be better accepted in the first instance, for example initially starting with a smaller correction dose of insulin in order to keep the person with T1DE safe and avoid DKA.

Discussion around other risks associated with exercise in T1DE can be found in section (e) below.

b) Vomiting, laxative abuse and diuretics

Hypokalaemia: vomiting and misuse of laxatives and / or diuretics can all result in hypokalaemia. Definitive treatment requires addressing the underlying behaviours. In some instances, plasma levels of potassium can be chronically low but acute changes are most dangerous. Regular eating with control of purging should be the goal to re-establish normal levels. If potassium replacement is required, follow the guidance in Table 4.

Refractory hypokalaemia may in part be secondary to concurrent low magnesium and therefore magnesium levels should be checked and replaced as necessary. See Table 4 for guidance on magnesium replacement.

If vomiting is a feature that persists and serum potassium levels remain low despite potassium supplementation, addition of a proton pump inhibitor such as lansoprazole may be considered. Proton pump inhibitors (PPI) work by inhibiting gastric acid secretion and may reduce metabolic alkalosis, helping to conserve potassium. This approach should only be considered as a second line measure[69] and clinicians should be aware of hypomagnesaemia as a potential side effect of PPIs[70].

Hypomagnesaemia and hypophosphataemia: although more commonly associated with refeeding, hypomagnesaemia and hypophosphataemia can both result from laxative and diuretic misuse due to losses in both faeces and urine. Patients who misuse alcohol seem to be at particular risk. If replacement is required, see example guidance in Table 4.

As described above, hypomagnesemia may be associated with hypokalaemia, in which case magnesium replacement will be required before hypokalaemia can be corrected; treatment-resistant hypokalaemia should therefore prompt measurement of plasma magnesium[69].

Blood glucose levels: vomiting can lead to unpredictable blood glucose levels, including the risk of hyper and hypoglycaemia and their associated complications (see sections 2.1 and 2.3). This may be further complicated by insulin omission in those with T1DE.

Acid-base changes: as well as the acidosis resulting from the accumulation of ketoacids that occurs with insulin omission, (see section 2.11(b)) a metabolic acidosis can also be seen in those who misuse laxatives due to the loss of potassium and bicarbonate in the faeces. Conversely vomiting and the use of diuretics typically leads to metabolic alkalosis through the loss of hydrogen and chloride in the vomit along with secondary hyperaldosteronism that can occur due to loss of sodium.

Depending on the behaviours of the individual with T1DE, a range of different acid-base balances can therefore be seen, and in some instances a mixed picture may be present.

Table 4: guidance for the correction of electrolyte abnormalities: adults (> 18 years)[71,72.]

Potassium	
3.5-5.0	Normal range
3.0-3.4	Replace orally with Sando K (12mmol K^+ per tablet), 2 tablets BD-TDS
<2.5	Requires intravenous replacement – refer patient to acute care
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

Oedema: when patients stop taking laxatives or discontinue purging they often develop oedema. Although the rapid weight gain associated with this can be very distressing for the individual it is temporary and diuretics are not typically recommended. In extreme cases however the accumulation of fluid may result in congestive cardiac failure (CCF), but this is rare. Signs of CCF can include shortness of breath that increases with activity and on lying flat, peripheral oedema, pulmonary crepitations on auscultation of the lung bases and a raised jugular venous pressure seen in the neck. If concerned about the potential for CCF, early review by a cardiologist should be sought and a chest x-ray, ECG, echocardiogram and brain naturetic peptide (BNP) level should be performed. A chest x-ray may show pulmonary venous redistribution and an echocardiogram may demonstrate reduced left ventricular function. An ECG may be normal, but it is important to do in order to screen for any abnormalities in the heart's electrical conduction which is not uncommon in people with severe eating disorders[73].

In those with T1DE, consideration should be given to the possibility that oedema can occur with the reintroduction of insulin. Discontinuation of laxatives or vomiting alongside the introduction of insulin therefore has the theoretical potential to exacerbate the severity of oedema seen.

Whilst 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.

Dehydration: diuretics and laxatives may be abused to achieve a temporary (fluid based) weight loss. Clinicians should be aware that in those with T1DE the resulting dehydration can result in

reduced effectiveness of circulating insulin, erratic blood glucose levels and an increased risk of DKA (see section 2.11 (c)).

c) Carbohydrate restriction

Although there is currently insufficient robust evidence to recommend low carbohydrate diets for those with type 1 diabetes, it is a dietary strategy that may be used by some with type 1 diabetes to manage their glycaemic control and reduce their requirement for insulin[74]. In those with T1DE they may use a low carbohydrate approach to inappropriately reduce their calorie intake, justify avoidance of insulin administration or in the instance of very low carbohydrate ketogenic diets (VLCKD) promote loss of body fat and weight through nutritional ketosis.

These strategies may result in good or very good glycaemic control (which may mask clinician concern) however can be associated with increased risk of severe hypoglycaemia without careful adjustment of type and amount of insulin[75]. Anyone with type 1 diabetes embarking on a ketogenic diet is encouraged to discuss this with their medical team[76] however due to the nature of the illness those with T1DE may well not seek advice.

In those with T1DE, dietary assessment should be undertaken to assess for carbohydrate manipulation and support provided to move towards a normalised pattern of balanced eating in line with mainstream nutrition recommendations for type 1 diabetes[77]. A gradual stepped approach to change may be better accepted than aiming for an ideal dietary intake in the first instance.

Carbohydrate restriction should be considered in relation to electrolyte shifts on refeeding, although in T1DE the rate of reinsulinisation is also key to managing this risk. For more information on the management of reinsulinisation / refeeding in T1DE (see section 2.6).

d) Excessive water ingestion

In 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. 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.

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).

In the case of thirst as a response to hyperglycaemia, fluids should not be restricted as this will increase the risk of DKA developing, particularly if ketones are present. Instead the priority should be on supporting the individual to take steps to take adequate insulin to improve the hyperglycaemic picture.

e) Exercise

The management of glucose levels in people with type 1 diabetes during exercise is complex with multiple physiological responses harmonising[78] and for people with T1DE the management of their blood glucose will be particularly challenging.

Hypoglycaemia: if a person with T1DE has a low BMI or is chronically restricting carbohydrates and does not reduce their insulin in response to this, they will be at risk of hypoglycaemia particularly when exercising as the glycogen stores in the liver may be depleted and unable to release sufficient glucagon to keep up with the increase in glucose required during exercise. People with eating disorders who are driven to exercise to manage weight and shape concerns may experience challenges with managing their insulin doses due to their heightened insulin sensitivity, which can put them at higher risk of experiencing hypoglycaemia for up to 24 hours after the exercise[79]. The effect physical activity has on blood glucose levels will vary depending on several factors including the duration and type of activity[80]. A person with type 1 diabetes should be supported to not exercise if blood glucose level is below 5.0 mmol/L[27], although if exercise is driven by strong eating disordered cognitions it may be hard for those with T1DE to accept this, again increasing the risk of hypoglycaemia.

After exercise muscle and liver glucose stores (glycogen) need to be replaced and glucose will be taken from the bloodstream to do this, known as "muscle filling effect". This occurs typically 4-8 hours after exercise. Blood glucose needs to be replenished with oral carbohydrate to prevent the ongoing risk of hypoglycaemia. Those with T1DE may find it difficult to do this and subsequently will continue to be at risk of hypoglycaemia.

Physical injury: Exercise levels in those with weight and shape concern can be high, resulting in friction burns, bruising and blisters, cracked or dry skin and increased callus 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. It is important to support all those with T1DE to engage with routine foot care, including daily checking for foot damage and as a minimum, annual foot examinations, more often if risk factors are present[29]. This is particularly important given that those with T1DE may disengage from services and that self-neglect can be a feature of eating disorders (see sections 4.1 and 4.5). More information on diabetic foot care can be found elsewhere[36,37].

https://www.nice.org.uk/guidance/ng19
https://www.diabetes.org.uk/guide-to-diabetes/complications/feet/taking-care-of-your-feet .

If weight is low there is a significant reduction in muscle mass and muscle strength, which reduces the support around the joints of the body. Continuing to exercise can result in:

- joint pain
- neck or back pain
- muscular and ligament injuries
- stress fractures.

If weight has been low for some time, osteopenia or osteoporosis may be present with its own inherent risks associated with particular forms of exercise.

Cardiovascular system: exercising at a low BMI and other compensatory behaviours such as purging can place extreme stress on already weakened cardiovascular and circulatory systems that can result in lowered blood pressure (hypotension), altered heart rhythms, dizziness and fainting.

Documents providing advice on managing a range of eating disorder related risks associated with exercise including osteoporosis and low BMI has been compiled by the Physiotherapy Eating Disorder Professional Network[81].

2.52 Long term risks of compensatory behaviours

a) Raised blood glucose

See section 2.12 above.

b) Laxative abuse

Long term abuse of stimulant laxatives can cause damage to the nerves controlling the colon which can result in a range of symptoms which include pain, bloating, faecal incontinence, nausea, vomiting and severe constipation[82,83]. These symptoms may be reversible with the discontinuation of stimulant laxatives however can sometimes progress to complete dysfunction of the colon requiring surgery[82,83].

In those with T1DE the presence of diabetes related gastroparesis or starvation, if it is present, may complicate the diagnostic picture by adding to symptomology (see section 2.12(a)).

Those with T1DE should be supported to discontinue laxatives, in particular stimulant laxatives if they are taking them. Symptoms may persist as it can take time for bowel function to recover[83]. Oedema and an associated temporary increase in weight are likely to occur[82,83] and support should be offered to encourage the person to persevere and not resort to restarting laxatives.

c) Vomiting

There is a range of medical concerns associated with the long term risks of vomiting. The following symptoms are the most commonly reported:

- Dental issues can arise due to the repeated exposure of teeth and gums to gastric acid. Dental erosion, tooth hypersensitivity and periodontal disease may all feature[84]. Until able to give up purging advise to[85]:
    - Avoid brushing teeth immediately after vomiting
    - Rinse with a non-acidic mouthwash after vomiting
    - Visit the dentist regularly
    - Avoid eating and drinking acidic foods, such as fruit juice both during a binge and after purging
    - Give up smoking.
- Skin abrasions to the fingers may be observed where the hand has been repeatedly inserted into the mouth to induce vomiting, ultimately resulting in callous formation[84]
- Recurrent nosebleeds should prompt inquiry about purging[84]

- Hoarseness, dysphagia, chronic cough, a burning sensation in the throat or repeated sore throats may occur due to repeated exposure of the larynx to regurgitated stomach contents[84]
- Parotid gland enlargement can occur alongside elevated salivary amylase. The enlargement of the salivary glands can be distressing for the patient due to the focus on body image. Reassurance can be given that this will resolve after discontinuing vomiting[82,84.]
- Excessive vomiting can lead to persistent gastro-oesophageal reflux disease and its associated signs and symptoms. Treatment is cessation of this behaviour and the administration of acid reduction therapies[82,84,86]
- Haematemesis due to Mallory-Weiss tears can occur or very rarely oesophageal rupture[82,86].

d) Carbohydrate restriction

There is a theoretical potential for long term carbohydrate restriction to impact on a range of health parameters, including adequacy of micronutrients and fibre, colorectal cancer risk, cardiovascular risk and kidney function. The current evidence highlights the need for further research to be undertaken in order to clarify the impact of this dietary strategy on these long term health parameters[87,88,89]. At present there is not enough robust evidence to support this dietary strategy in those with type 1 diabetes[74].

2.6 Reinsulinisation / refeeding

Refeeding syndrome is an insulin mediated, potentially fatal shift in fluids and electrolytes that can occur in malnourished patients on the reintroduction of energy in the form of carbohydrate[90].

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 refeeding syndrome. The term *reinsulinisation* describes the process of insulin reintroduction whilst *refeeding* describes the reintroduction of food, to include carbohydrate.

The severity of risk as a result of reinsulinisation / refeeding will be directly associated with the severity of starvation. It is important to recognise that there can be cellular starvation as a result of insulin omission regardless of weight, BMI or the other markers of starvation outlined in Table 2 (see section 2.41). For this reason it should be considered that all those with insulin omission are potentially at risk of electrolyte shifts on reinsulinisation / refeeding. 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[91]. There is no reason to assume that this is not the case in those with T1DE.

In those with T1DE, who have been omitting insulin, the risk of reinsulinisation / 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:

- Psychological; building up insulin and food slowly gives time to address psychological concerns which if not addressed will potentially deter the person with T1DE from continuing with treatment.
- It will minimise the risk of insulin / refeeding related oedema which although usually medically harmless has the potential to deter the person with T1DE from continuing with treatment as a result of rapid weight changes.
- It will allow adjustment to post-meal feelings of fullness as a result of delayed gastric emptying which can occur if food restriction has been part of the picture or if diabetes related gastroparesis is present. These feelings of fullness can be interpreted as 'feeling fat' and deter the person with T1DE from continuing with treatment.
- A gradual reduction in blood glucose levels will reduce the risk of complications i.e. treatment induced retinopathy and neuritis (see section 2.11(a))[9,10] .
- It will reduce the risk of hypoglycaemia or the frequency of pseudo hypoglycaemia which will potentially deter the person with T1DE from continuing with treatment.

Whilst a gradual approach is advocated, this needs to be balanced against the risks of DKA and underfeeding. Any protocol needs to ensure enough insulin is initially provided to ensure ketone production is switched off. The aim should then be incremental increases in food (to include carbohydrate) and insulin to achieve weight stabilisation or weight gain (if required) in order to prevent ongoing physical deterioration.

The requirement for certain vitamins is increased during refeeding, in particular thiamine and other B vitamins as there is an increased demand for these vitamins when metabolising energy from carbohydrate rich foods. Inadequate supply can on rare occasions lead to potential medical complications i.e. Korsakoff's syndrome and Wernicke's encephalopathy[90].

Taking all of the above into account an example protocol for reinsulinisation / refeeding can be found in Appendix B.

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.

NB: It is noted that in some instances nasogastric feeding may be required. Management guidelines for this intervention is beyond the scope of this document and advice should be sought from the treating nutrition and diabetes teams.

3.0 PSYCHOLOGICAL AND PSYCHIATRIC RISK

3.1 Psychological treatment of T1DE

It has been highlighted that those with type 1 diabetes show worse outcomes with conventional eating disorder treatment than those without type 1 diabetes[92] and at present this risk to those with T1DE remains, as it is not clear what the optimal psychological treatment model for T1DE is.

Theoretical models for T1DE are still in the very early stages of development. Three notable examples of theoretical models to date are Daneman et al's [93] model of the interaction between type 1 diabetes and eating disorders, Treasure et al's [94] theoretical maintenance model for disordered eating in people with type 1 diabetes and Goebel-Fabbri's [95] model of eating disorders in T1DE with insulin restriction.

The evidence base for psychological interventions effective in treating eating disorders in type 1 diabetes is also limited[96] and NICE guidance for eating disorders proposes that further research is needed regarding treatments for eating disorders in people with a comorbidity such as type 1 diabetes[60]. Specifically it notes the potential to investigate the effectiveness of a modified eating disorder therapy for those with a long term health condition that addresses both conditions and avoids the need for offering two different interventions (in parallel or one after the other). However, to date articles outlining treatment guidelines, although helpful, are limited as they are based upon clinical expertise rather than rigorous research, for example Goebel-Fabbri et al, 2009[97].

Psychological treatments for T1DE which have been tested include cognitive behavioural therapy (CBT), psychoeducation and integrated inpatient therapy[96]. Adaptations were made to existing treatments for eating disorders, with the most popular being to add extra education about type 1 diabetes to the pre-existing eating disorder treatment. The main conclusion drawn is that those delivering the intervention should ideally be knowledgeable and trained in the treatment of both eating disorders and type 1 diabetes management and that the duration of treatment offered should be addressed, with a longer treatment offering greater impact.

Currently the Safe Management of People with Type 1 Diabetes and Eating Disorder Study (STEADY) programme at King's College London is in the midst of designing a specialised treatment programme for T1DE based on patients' lived experiences offering both diabetes and mental health care with elements of education and psychotherapy.

In the absence of a robust evidence base for treatment of T1DE it is recommended that care plans and interventions are based on the principles outlined above alongside a biopsychosocial formulation that takes into account the interplay between the physical, emotional and cognitive elements of both type 1 diabetes and eating disorders. It is important that interventions are delivered by those with a working knowledge of both type 1 diabetes and eating disorders to avoid conflicting approaches that can occur between the usual working practices of these two specialties. Within the ComPASSION project example interventions have included:

- psychoeducation
- enhancement of self-efficacy
- building a sense of importance and confidence around making changes

- cognitive restructuring (e.g. diabetes management and body image)
- mood regulation, including understanding and coping with strong emotions
- developing compassion for oneself
- building acceptance (e.g. of diabetes as a long term health condition)
- identifying and living with personal values
- understanding and planning for high risk situations as part of relapse prevention work.

Practitioners need to be alert to developments in the evidence base for the psychological treatment of T1DE and revise practice in accordance with the emerging evidence base in order to optimise the efficacy of treatment interventions they are offering.

3.2 Access to psychological treatment

There is a significant risk that T1DE referrals to eating disorder services will be declined. Eating disorder services are oversubscribed nationally and as a result have referral criteria that often exclude those whose eating disorder behaviours are atypical. It is also likely that referrals are declined because of lack of evidence based interventions to offer (see section 3.1) and feelings of being ill equipped to modify the existing interventions to accommodate type 1 diabetes when this is not an area of expertise. The impact of declined referrals on individuals can be significant and can exacerbate feelings of difference from others and hopelessness, and might prevent them asking for help again in the future.

There are few psychologists attached to adult diabetes services nationally. Access to a psychologist for assessment in a diabetes team is rare and diabetes psychologists are unlikely to have the specialist eating disorder training required to work with this group.

Individuals with T1DE may be referred by the GP, or via self-referral, to primary care psychological services (Improving Access to Psychological Therapies (IAPT) that provide NICE recommended psychological treatment for depression and anxiety disorders within the NHS) or to local Community Mental Health Teams because of associated depressive or anxious symptomatology, dependent on severity. It is possible that due to low awareness of T1DE, the presence of an eating disorder and the relevance of type 1 diabetes could be missed.

Considering all of the above, it is our experience that starting the conversation and ongoing liaison between local diabetes and eating disorder teams has great potential to improve services available to those with T1DE through sharing expertise and offering shared care.

3.3 Psychiatric co-morbidities

Psychiatric comorbidities are the norm in people with eating disorders (>70%). The most common psychiatric comorbidities include mood and anxiety disorders, neurodevelopmental disorder, alcohol and substance use disorders, and personality disorders[98,99]. Type 1 diabetes has its own impact on emotional health, including depression, anxiety, diabetes distress and burnout, fear of long term complications, fear of needles, acceptance of diagnosis and fear of hypoglycaemia[100,101].

Many of these areas of psychological difficulty may form part of the initial development of an eating disorder in type 1 diabetes and / or serve to maintain it. They consequently can present a risk to

recovery and as such their assessment and treatment needs to be considered within any treatment model proposed.

a) Depression

As well as being a common co-morbidity in eating disorders, rates of moderate to severe depressive symptoms affect about 1 in 5 individuals with type 1 diabetes[102]. Depression can hinder recovery as it will impact upon motivation, energy and hope for change, as well as on perceived deservedness of treatment. A potential link between depression and self-neglect or suicide must also be considered (see sections 4.5 and 3.4). In those with type 1 diabetes depression is associated with an increase in cardiovascular risk factors[103], a 50% increase in mortality[104] as well as sub-optimal diabetes self-management and HbA1c levels and increased diabetes distress[105]. Additionally, poor metabolic control can worsen depression[106]. It is therefore vital to recognise and treat depression in patients presenting with T1DE.

Individuals with an eating disorder (and no diabetes) have been found to have greater depression than their T1DE matched peers however this may be because those with T1DE are not yet fully aware of the extent of their eating disorder and related consequences, and thus their psychological health may deteriorate with heightened awareness during the course of treatment[107]. HCPs working with those with T1DE should be alert to this possibility even if depressive symptoms are not initially present.

The nine-item Patient Health Questionnaire (PHQ-9)[13] is widely used to assess depression. It is quick to administer and freely available online (www.phqscreeners.com). Asking the person to complete the PHQ-9 can be a useful way to start a conversation about depressive symptoms and the effect they may have on the person's life, diabetes management and experience of the eating disorder. It can also be useful for systematically monitoring depressive symptoms (e.g. whether the symptoms are constant or changing over a period of time). However, a clinical interview by an appropriately qualified professional is needed to diagnose depression.

NICE guidance indicates a stepped-care model to treat depression, supporting patients, carers and practitioners in identifying and accessing the most effective interventions[108]. However, in T1DE the treatment of depression needs to be part of a broader psychological treatment package guided by an individual formulation that outlines the interaction between depression, diabetes and eating disorders.

b) Anxiety

As well as being a common co-morbidity in eating disorders, having type 1 diabetes places people at an increased risk of developing elevated anxiety symptoms or an anxiety disorder[109] with moderate to severe anxiety symptoms affecting about 1 in 6 people with type 1 diabetes[102].

As with depression it has been found that individuals with eating disorders (and no diabetes) reported greater state and trait anxiety than their T1DE matched peers[107]. Practitioners working with those with T1DE should however be alert to the possibility of emerging anxiety in those with T1DE as treatment progresses even if symptoms are not initially present.

Identification of anxiety in those with T1DE may be overlooked as severe anxiety and panic attacks share some similar physical symptoms to hypoglycaemia (e.g. sweating, increased heart rate, shaking and nausea). Consequently, elevated anxiety symptoms may be misinterpreted by people with diabetes and health care professionals (HCPs) and anxiety disorders may go undiagnosed.

Thorough clinical assessment allows for formulation of anxiety within the broader picture of T1DE to guide options for intervention. A brief questionnaire, such as the Generalized Anxiety Disorder Seven (GAD-7)[12], can be used for identifying people with elevated anxiety symptoms. It is quick to administer and freely available online (www.phqscreeners.com). As with depression a clinical interview by an appropriately qualified professional is needed to confirm an anxiety disorder. Follow on questionnaires to identify type 1 diabetes specific fears such as diabetes distress or fear of hypoglycaemia or complications are crucial and some of these are outlined in the subsequent co-morbidity sections.

It is likely that an untreated anxiety disorder could pose an obstacle to recovery from T1DE and therefore needs to be treated. NICE guidance outlines the evidence based psychological interventions for the specific anxiety disorders (e.g. PTSD, GAD and Panic Disorder, OCD etc)[110]. In the context of T1DE any treatment of an anxiety disorder should draw upon evidenced based interventions guided by an individualised formulation that outlines its interaction with both diabetes and the eating disorder psychopathology.

c) Pharmacological treatment of comorbid depression and anxiety

NICE recommends that for people with moderate or severe depression, a combination of antidepressant medication and a high-intensity psychological intervention should be provided. Similarly, the treatment for anxiety disorders associated with functional impairment generally involves psychological therapy on its own or in combination with medication[110].

A Cochrane review examining clinical trials on psychological treatments and antidepressant drugs in depressed patients with diabetes suggests that antidepressants are effective and moderately improve glycaemic control, and that serious or severe adverse effects were rare[111].

The Maudsley prescribing guidelines[112] recommend that in patients with diabetes, selective serotonin reuptake inhibitors (SSRIs) should be used as the first line in the treatment of depression in type 1 diabetes. Serotonin–noradrenaline reuptake inhibitors (SNRIs) are likely to be safe but there is less supporting evidence.

Blood glucose and HbA1c should be carefully monitored when antidepressant treatment is initiated, when the dose is changed and after discontinuation as some antidepressants, including SSRIs, are associated with insulin resistance[113].

In the British National Formulary (BNF), there are no reported drug interactions between insulins and any psychotropic drugs[114] however drug classes including monoamine oxidase inhibitor (MAOI) and tricyclic antidepressants (TCA) may increase appetite and weight and these side effects need to be considered when prescribing for T1DE patients who are likely to have significant weight and body image concerns. Maudsley prescribing guidelines advise that TCAs and MAOIs should be avoided in patients with diabetes due to their effects on weight and glucose homeostasis[112].

All patients treated with SSRIs should be monitored for the development of akathisia, increased anxiety and the emergence of suicidal ideation; the risk is thought to be greatest in those <30 years, those with co-morbid depression and those already known to be at higher risk of suicide[115,108] (see section 3.4).

d) Diabetes distress

Diabetes distress is the emotional distress resulting from living with diabetes and the burden of the relentless tasks of daily self-management[116] as well as the social impact of diabetes (e.g. stigma, dealing with other people's unhelpful reactions and lack of understanding)[10]. Severe diabetes distress affects about 1 in every 4 people with type 1diabetes[117] and is associated with sub-optimal diabetes self-management and impaired general emotional wellbeing. Importantly it is not that people do not understand the importance of diabetes self-management but that they feel unable to work with their diabetes any longer.

Diabetes distress (and other reasons for not taking required insulin doses such as depression, anxiety needle phobia, acceptance of diagnosis and fear of hypoglycaemia) may be mistaken for T1DE as presentation may include indicators associated with T1DE, including raised HbA1c, associated weight loss and disengagement with services. A careful history is required to identify if weight and shape concerns are part of the psychopathology in order to make a differential diagnosis and identify if T1DE is present or not.

In those with T1DE, it is our experience that diabetes distress and the related reasons highlighted above for not taking the required insulin doses commonly co-exist in those with T1DE and 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. Focus on precision, the need to count food and calculate insulin doses can also maintain the psychopathology of an eating disorder.

Diabetes distress can be assessed using a screening tool such as the Diabetes Distress Scale (DDS) 2[9], DDS 17[10] or the Problem Areas in Diabetes (PAID) scale[8]. The results can form a basis for a conversation around the person's experience of diabetes and an opportunity to consider what might help:

- e.g. simplification of the diabetes management plan, by for example using fixed doses of insulin and carbohydrate
- taking a planned, safe break from diabetes management for a few hours or a day, by relaxing targets a bit or reducing how many times bloods are checked each day[5]
- and realistic goal setting.

If the diabetes distress is significant a psychological intervention may be needed. Whilst intervention studies are scarce, a recent systematic review and meta-analysis found that elevated diabetes-distress is responsive to psychological treatment with a medium effect size[118]. Interventions in the review included CBT, problem-solving therapy, mindfulness and motivational interviewing (MI) however it was impossible to draw conclusions on the potentially differential effects of the interventions.

We have found that it is important for all members of the MDT to be aware of the impact of diabetes distress and that its psychological treatment cannot be separated out from treatment of the eating disorder.

e) Fear of hypoglycaemia

Fear of medical hypoglycaemia is a specific and extreme fear evoked by the risk and/or the occurrence of low blood glucose levels[119]. People fear losing consciousness in public, having an accident or injury, becoming emotionally upset or uncooperative and embarrassing themselves. They also worry about the very worst (but rare) scenario of sudden death. About 1 in every 7 people with type 1 diabetes report fearing hypoglycaemia[100]. Although being concerned about hypoglycaemia is rational and adaptive, if concerns develop into excessive fear this can result in the omission or reduction of insulin doses to avoid the risk of hypoglycaemia and is associated with impaired quality of life and emotional wellbeing, sub-optimal diabetes management and subsequently higher rates of diabetes related complications and symptoms.

As with diabetes distress the potential for HbA1c to be raised with or without associated weight loss, means that fear of hypoglycaemia can raise suspicion of T1DE and again a careful history is required to identify if weight and shape concerns are part of the psychopathology in order to make a differential diagnosis and identify if T1DE is present or not.

In the context of T1DE, fear of pseudo hypoglycaemia needs to be given consideration as during the early stages of treatment, when blood glucose levels are being gradually reduced, individuals can experience the symptoms of hypoglycaemia at normal or elevated blood glucose levels.

Fear of experiencing either pseudo hypoglycaemia or medical hypoglycaemia may be a barrier to treatment of T1DE as it may be an additional factor in the individual feeling unable to take recommended doses of insulin. It may also result in the overconsumption of carbohydrates to maintain blood glucose levels at a raised level.

Fear of hypoglycaemia can be assessed using the Hypoglycaemia Fear Survey II (HFS-II)[119]. It is important to assess if the fear is part of an existing anxiety disorder or PTSD, related to a traumatic experience of a previous hypoglycaemic event.

As fear of hypoglycaemia is intertwined with diabetes management, it is best addressed by an HCP with expertise in diabetes management. Psychological approaches to treating fear of hypoglycaemia follow:

- Psychoeducation about anxiety and fear and how the symptoms of this overlap with symptoms of hypoglycaemia.
- Information about actual risk of hypoglycaemia and its management; building skills to challenge unhelpful thoughts about perceived risk.
- Building confidence to take small steps in the direction of taking larger insulin doses and tolerating lower blood glucose levels heading towards ‘individualised targets’ which may be higher than standard targets.
- Addressing excessive safety behaviours.
- Involving family members who may have become part of the picture of maintaining excessive fear.

In the treatment of T1DE all of the above strategies are important in addressing fear of hypoglycaemia. For individuals with both T1DE and fear of hypoglycaemia there is a cumulative challenge of taking insulin (fear of hypoglycaemia and fear of weight gain). It is therefore helpful to work with the individual to take small steps towards an individualised target allowing the person to psychologically adjust to the sensation of pseudo hypoglycaemia and to build confidence and self-efficacy in their ability to cope with medical hypoglycaemia if and when it occurs (see section 2.3). It is very unlikely that having knowledge about the long term consequences of hyperglycaemia will motivate a person with fear of hypoglycaemia to reduce their blood glucose levels. Fear of hypoglycaemia is related to the 'here and now', not to long term health risks.

f) Fear of long term complications

Care should be taken not to assume that people with T1DE are not aware or concerned about the increased risks of long term complications that are associated with T1DE. In our experience this is in fact the opposite and significant anxiety, guilt and shame are commonly experienced. Diabetes education has a strong focus on the risk of long term complications and this may trigger (unrealistic) severe concerns, especially in people who do not feel able to maintain their blood glucose levels within recommended targets. It is important to shift the focus away from 'scary' messages about complications, instead discussing risk in an individualised way and more importantly encouraging the person to take small steps towards diabetes self-management, recognising this will positively impact upon the risks of long term complications. Worry about long term complications can be a motivator for taking steps towards self-care and engaging in T1DE treatment but do not assume this will be the case just because this seems obvious to the HCP. Instead take time to understand what would be the benefits of making change for each individual.

g) Fear of needles

Fear of needles, injections or finger pricks can have an impact on diabetes management (e.g. the person with diabetes (PWD) reducing the number of injections or blood glucose checks), diabetes outcomes (e.g. elevated HbA1c, greater risk of long term diabetes complications) and emotional wellbeing (e.g. impaired general wellbeing and diabetes distress). Modern insulin pens, finer needles and lancets, as well as technology such as insulin pumps and flash glucose monitoring, all help to minimise the pain of insulin injections and blood glucose checks. Needle phobia is a more extreme and debilitating form of fear. For people with a needle phobia, the sight of a needle or blood evokes anxiety and an increased heart rate, followed by a drop in blood pressure, dizziness, fainting, sweating, and nausea. Needle phobia is rare but, if it is present, it will complicate diabetes self-management.

Fear of needles, if present, could pose an obstacle to diabetes management in T1DE and should be especially considered in cases where it is proposed an individual is taken off a pump because of manipulation of the technology to reduce insulin dosing. A pump may have been initiated in the first place because of a fear of needles and so consideration of strategies to assist with the switch back to multiple daily injection (MDI) therapy is necessary. Strategies may include injecting education, behavioural therapy, desensitisation or distraction, and relaxation.

h) Alcohol and substance misuse

There is no known data about the use of alcohol and drugs in T1DE. As a rule, if an individual has alcohol or drug dependency and requires detoxification this would be delivered ahead of any other integrated T1DE intervention. Following detoxification, or if the substance use does not require detoxification, further interventions should be guided by a formulation that includes the previous drug and alcohol use, considering its function and interaction with diabetes and the eating disorder. Treatment should factor in strategies for relapse prevention and may require joint working with local addiction agencies which may often be non-statutory.

i) Personality disorders

A personality disorder (PD) diagnosis describes a long term pattern of behaviour and inner experiences that deviates from the expectations of the culture, causes distress or problems functioning, and lasts over time. There are 10 specific types of personality disorder defined by mental health diagnosis classification systems like the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) or International Classification of Diseases, Tenth Revision (ICD-10). Intrapersonal and interpersonal difficulties are central to the difficulties observed in personality disorder and attachment and trauma theories provide explanatory frameworks for understanding the development, maintenance, and treatment of the difficulties encountered by an individual who meets criteria for a diagnosis (or features of) a personality disorder[120].

A review of the literature surrounding personality disorder prevalence in those with eating disorders has found that there are specific personality trends amongst the eating disorder subtypes[121]. Obsessive compulsive and avoidant PD (Cluster C) are prominent in restrictive anorexia nervosa (AN), and borderline, histrionic PD (Cluster B) and dependent PD (Cluster C) are overrepresented in binge/purge disorders including AN binge/purge subtype.

There is a lack of research looking at any relationship between T1DE and personality disorder however one study found that borderline traits significantly predicted whether or not patients with type 1 diabetes would engage in eating disorder behaviours such as binge eating and insulin omission[122] and there is some evidence that cluster B personality disorders in general predict worse outcomes in glycaemic control[123]. Significant issues of treating those with co-morbid type 1 diabetes and borderline PD have also been noted including difficult interactions with treatment teams and risk of terminating therapeutic relationships prematurely[124].

In broad terms those with anxious, avoidant or obsessive compulsive PD have the potential to be more difficult to engage whilst those with externalising type PDs such as borderline PD are more likely to disengage from treatment prematurely and impact on team functioning. Where personality pathology is suspected in those with T1DE it is important to:

- Consider the process of engagement (see section 4.1) and support retention in treatment
- Provide assessment via a structured clinical interview and be mindful of the impact of physical health status on PD symptomatology (individuals who are very physically unwell because of an eating disorder are more likely to evidence higher scores on PD measures. PD features secondary to the eating disorder may well decrease with treatment of the eating disorder[125])

- Work with the patient to draw together a formulation that can help to understand the development and maintenance of the difficulties encountered (regarding T1DE but also incorporating elements of intra or interpersonal difficulties)
- Not to have too big a team offering multiple contacts, instead one or two key points of contact to facilitate interpersonal relationships
- Be clear and thoughtful about boundaries as a team and ensure an agreed and co-ordinated approach to treatment to minimise splitting of the treatment team
- Ensure regular team case reviews are in place
- Consider the impact on clinician well-being and utilise clinical supervision sessions (see section 5.0)
- Consider extra pre-admission preparation for inpatient admissions (e.g. sharing of a basic formulation with the inpatient team by the community team).

3.4 Suicide

In recent years there has been heightened interest in investigating the links between physical illness, mental illness, and suicidal behaviour. People with a physical health long term condition are more likely to have poor mental health, and vice versa. Suicide occurs more frequently with the coexistence of psychiatric and physical illness.

a) Suicide in eating disorders

A systematic review on suicidality in eating disorders concluded that suicide is the second leading cause of death in AN[126]. It is possible that the reported suicide rates are underestimates because of reluctance to report suicide as a cause of death. Between 3 - 20% of patients with AN report suicide attempts. Although the rate of completed suicide is low in bulimia nervosa (BN), the percentage of patients who attempt suicide is high, ranging from 25% to 35% in outpatient samples. Clinical correlates of suicidality include:

- Purging behaviours
- Comorbid disorders (e.g., depression, substance abuse)
- A history of physical and/or sexual abuse
- Certain personality features.

The review recommends that a thorough suicide assessment should be conducted routinely for individuals with past and current eating disorders, and that clinicians should be aware that this risk may be ongoing and occur throughout treatment.

b) Suicide in type 1 diabetes

A systematic review on suicide risk and type 1 diabetes indicated that, in general, patients with type 1 diabetes have a higher risk for suicide than the general population does. Patients with type 1 diabetes are 3 to 4 times more likely to attempt suicide than the general population and have a 61% higher risk of experiencing suicidal thoughts than individuals without type 1 diabetes[127]. Not all research reports a higher risk of suicide in patients with type 1 diabetes however most of the studies report rates of suicides in individuals with type 1 diabetes in the range of 5-15%[128]. Risk factors identified in the systematic review[127] of the literature include:

- Depression
- Childhood trauma
- Drug and alcohol abuse
- Disengagement with diabetes treatment plan.

In addition, it has been found that young adults admitted to hospital for diabetic ketoacidosis (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[129].

As the literature on risk factors for suicide in those with type 1 diabetes is limited it is suggested that the risk factors related to suicide in general apply in the context of diabetes as well[128]. General risk factors for suicide are outlined in Box 5.

Box 5: general risk factors for suicide[130]

Demographic and social:

- perception of lack of social support, living alone, no confidants
- males (may not disclose extent of distress or suicidal thoughts)
- stressful life events (e.g. recently bereaved, debt/financial worries, loss of attachment, major relationship instability, job loss, moving house)
- LGBTQ
- ethnic minority group.

Personal background:

- substance misuse: alcohol and/or illicit drug misuse especially if precipitated by a recent loss of relationship
- feeling close to someone who died by suicide (family or non-kin) or exposure to suicidal behaviour of key others (family, peers, favourite celebrity)
- use of suicide-promoting websites or social media
- **access to lethal means;** (if unable to remove lethal means ensure mitigation within a robust safety plan) (see section (c) overleaf).

Clinical factors in history:

- previous self-harm or suicide attempt(s) (regardless of intent, including cutting)
- mental illness, especially recent relapse or discharge from inpatient mental health care
- **eating disorder**
- disengagement from mental health services
- impulsivity or diagnosis of personality disorder
- **long-term medical conditions;** recent discharge from a general hospital; pain.

Mental state examination and suicidal thoughts:

- high degree of emotional pain and negative thoughts (hopelessness, helplessness, guilt – e.g. 'I'm a burden')
- sense of being trapped/unable to escape (sense of entrapment) and/or a strong sense of shame
- suicidal ideas becoming worse
- suicidal ideas with a well-formed plan and/or preparation
- psychotic phenomena, especially if distressing; persecutory and nihilistic delusions, command hallucinations perceived as omnipotent (pervasive).

c) Suicide in T1DE

Given that the risk for suicide is elevated in both eating disorders and type 1 diabetes, it is vital to consider this risk in those with T1DE, taking into account all of the risk factors outlined above.

It is important to highlight too that 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. HCPs 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 patients prescribed insulin has been proposed[131]:

- 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.

d) Assessment of suicidality

Suicide risk assessment can be conducted as a part of screening and assessment for depression (see section 3.3 (a)). Brief assessment tools like the Primary Health Questionnaire-9 (PHQ- 9) have questions specifically relating to suicidal ideation. Asking about current suicidal ideas and any plans based on these directly (and not in a roundabout indirect manner) is necessary. It has been a concern historically in HCPs that asking about suicidal ideas might insert such ideas into the mind of the person assessed, but hesitation on the part of the clinician may lead to missed opportunities to screen for suicidal behaviour.

e) Management of suicidality

A red flag is a risk factor with special significance in that it indicates that a person is at heightened risk of attempting suicide at this particular moment in time. This imminent risk requires an urgent, clinically appropriate and personalised intervention with a safety plan. 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. The safety plan should list activities and coping strategies and contain information on how to access social, psychological and emergency support[130].

3.5 Lack of insight

It is not uncommon for those with eating disorders to lack insight into or deny their illness. The phenomenon has been described and reviewed for those with anorexia nervosa[132,133,134] and the

DSM-5[135], states that 'individuals with anorexia nervosa frequently either lack insight into or deny the problem'.

Whilst lack of insight appears to be less prominent than in those with other eating disorder presentations[136] it is our experience that lack of insight or denial of the severity of the consequences of the illness can be a feature of T1DE. It is also our experience that this insight may fluctuate, with an understanding and fear of the consequences of the illness emerging intermittently.

Alongside shame, embarrassment and fear of criticism or judgement, lack of insight or denial of the illness may contribute to a lack of engagement in treatment. Building a relationship with the person with T1DE and gaining their trust will be key in this situation (see section 4.1). The process of engagement can take considerable time but should be considered a valuable and necessary intervention. The degree of presenting risk must also be assessed and where medical or psychiatric risks are high consideration given to capacity to consent to treatment (see section 3.8). Lack of insight may also need to be considered in relation to safeguarding of self and others, fitness to work and driving (see sections 4.4, 4.6 and 4.7). The impact on carers can also be considerable (see section 4.2).

3.6 Cognitive function

Both nutritional starvation and hyperglycaemia can impact on cognitive function (see sections 2.42 and 2.11(d)). This can impact upon treatment progression as a result of, for example, impaired concentration and difficulty with retention and processing. Both nutritional starvation and chronic hyperglycaemia can also be associated with emotional numbing, which can be negatively reinforcing of food and/or insulin restriction. Chronic hyperglycaemia can cause significant disruption to sleep which over time could impact upon everyday cognitive function (see section 3.7).

Consideration of these factors is important in the development of an individual formulation with the person to map out the maintaining processes in the eating disorder. Impaired cognitive function might impact upon the person's ability to utilise therapeutic interventions effectively. It may be necessary to make adaptations such as the use of repetition, summarising and writing down plans. The degree of impairment may guide choice of therapeutic intervention e.g. outpatient psychological therapy (which may require a higher level of cognitive functioning) versus inpatient treatment (where a focus might be on restorative eating and reinsulinisation).

The impact of reduced cognitive functioning might also have relevance in assessment of capacity (see section 3.8).

3.7 Sleep

Poor sleep is a commonly reported symptom in those with type 1 diabetes and can be decreased in both quality and length. This can be due to both physiological and psychological reasons. Poor sleep can impact on glycaemic levels (both higher and lower levels) and in turn suboptimal glycaemic levels can cause poor sleep[137].

Those with eating disorders have more disrupted sleep than those without eating disorders, with sleep disturbance being more commonly reported in those with AN than BN or Binge Eating Disorder

(BED) whose rates of sleep disturbance are reported similar to age matched controls[138,139,140]. For those with BN, rates of sleep disturbance are lower than those with depression[138]. The sleep disturbance seen in AN is thought likely to be due to hormonal changes, for example increased levels of orexin which drives wakefulness and feeding, that occur as a result of malnutrition[138] with markers of improved sleep positively correlated with ideal body weight (IBW) and BMI[139,140]. In those with T1DE where BMI is often not sub-optimal poor sleep may therefore relate to other factors such as suboptimal glycaemic control, fear of hypoglycaemia, polyuria or co-existing mood disorders.

Regardless of the cause, sleep deficit can cause problems with decision making, planning, use of information and problem-solving, all important to self-care and so can negatively impact someone with type 1 diabetes looking after their condition and making frequent decisions throughout the day about their health[141]. Poor sleep is also associated with decreased emotional well-being which then impacts on self-care behaviours[142].

Asking about and identifying poor sleep (quality, length, nocturnal waking etc.) in someone with T1DE is important in order to recognise it as a mechanism that may be maintaining the eating disorder.

3.8 Use of the Mental Health Act in the treatment of T1DE

Compulsory measures are generally unnecessary and, as patient autonomy is a long term objective in the management of eating disorders, they may be counter-productive.

As eating disorders can be accompanied by significant morbidity, especially if insulin restriction is used as a compensatory behaviour, there may however be occasions when clinicians may consider using the Mental Health Act (MHA) 1983[143] in the context of T1DE. This may be the case, for example, when a patient's physical health or survival is seriously threatened as a result of their mental illness.

In the MHA 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 and treatment for a physical condition may only be given without the patient’s consent therefore, if it is sufficiently connected to the treatment of the patient’s mental disorder.

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.

It may also be possible to justify under the Mental Capacity Act (MCA) 2005[144] action that is taken in an emergency as the minimum necessary to prevent serious injury or loss of life.

4.0 PSYCHOSOCIAL RISKS

4.1 Engagement

Individuals who struggle with a combination of type 1 diabetes and eating disorders often experience considerable shame and embarrassment about their situation and fear criticism and judgement from health care professionals (HCPs). These factors lead to reluctance to attend appointments and avoidance of contact with services. Their reluctance to engage exists in sharp contrast to their elevated level of risk, with T1DE patients more likely to drop out of treatment and show worse outcomes with conventional outpatient treatment for eating disorders[144,92].

Building a trusting and respectful therapeutic relationship through a focus on engagement is key in assisting people with T1DE to feel able to talk openly about their experiences and consider making changes. Principles of engagement include:

- Ensuring contact and communication is done in a way that reflects a supportive, non-judgemental, encouraging and empathic approach, which continues if avoidance of contact persists.
- Considering any communication needs e.g. for sight loss or learning disability.
- Carefully considering and adjusting language used in consultations.
- Considering any known relationship preferences of the person with different members of the multidisciplinary team (MDT).
- Seeking to understand the difficulties from the person's perspective and to come alongside them to envisage what change might look like and build hope for change.
- Holding empathy and validating the emotions of the person with diabetes (PWD), believing the person is doing the best they can, whilst also holding hope, offering encouragement and assisting the person to take the next steps that are needed.
- Developing realistic expectations and working at the individual's pace (whilst supporting each other as a team to contain our own anxieties about this).
- Recognising slips back as just that (rather than indicating failure).
- Noticing progress and celebrating effort.
- Keeping the person connected with their values and why they (not HCPs) feel it is important for them to make a change.
- Building a life beyond the eating disorder with a space for diabetes within it.

Two recent documents provide useful summaries of communication skills for HCPs[100,146].

The process of engagement takes time and several informal contacts may be needed before moving to formal assessment or discussing treatment options. This should be considered part of the treatment pathway and a good investment of clinical time.

Because of the potential for significant physical and mental health risks within this population, non-engagement needs to be carefully considered and managed empathically and appropriately. The ComPASSION pathway for engagement and non-engagement which takes into account management of the risks of non-engagement can be found in Appendix D. Note that assertive approaches to engaging and re-engaging individuals that are referred to within the pathway may be at odds with

typical adult diabetes clinics procedures. It may therefore be helpful to link with local mental health teams to consider these approaches further.

4.2 Disruption of social networks

Eating disorder symptoms can elicit emotionally driven reactions and behaviours from those close to the individual affected and these are accentuated in the context of diabetes[94]. The anxiety caused by being faced with someone who is clearly unwell, who may lack insight and shows little inclination to implement the obvious solution (to eat and / or take their insulin) can drive strong emotional reactions which in turn can play a part in the maintenance of the eating disorder.

For this reason, people with T1DE may find that the people around them find it difficult to understand why they behave the way that they do. Rather than fearing the possible complications of their condition, their driving fear may be that of gaining weight and in order to manage that fear the individual will behave in ways that put their life at risk.

Watching someone you care about effectively committing slow suicide[147] is likely to strain even the strongest relationship and the powerlessness experienced by friends and relatives to influence behaviour is likely to mean that these relationships become a source of conflict. That conflict will only increase the guilt and shame felt by the individual and increase any sense of isolation and alienation.

In order to help in these circumstances:

- Invite friends and relatives to diabetes education sessions.
- Offer appointments to parents, siblings and partners to talk about difficulties.
- Offer training to friends and relatives to increase their understanding of type1 diabetes and eating disorders.
- Offer training in communication skills and approaches to partners, parents and siblings.
- Share information about support services for friends and relatives.

In those with an eating disorder, including T1DE, social withdrawal with the person disconnecting from previously enjoyed social events and activities, is common. Feelings of low self-worth, believing they have nothing to contribute, a discomfort of being around others in the presence of food for fear of anxiety, discovery or judgement, difficulty trusting others and the drive to make time for the preoccupation with eating disorder led thoughts, all contribute to the perceived benefits of withdrawal.

In order to help the individual:

- Work with the individual to develop ways of managing fear and anxiety that do not have a negative impact on health.
- Explore with the individual other areas of their life which increase their self-worth and reduce the focus of body image as their only source of identity.
- Encourage engagement in social activities and opportunities.
- Offer peer support.

4.3 Media

a) Social media

This can be a helpful source of support and community for people, but conversely can drive unhappiness and unhealthy behaviour. In relation to eating disorders some specific risks include:

- Normalisation of unhelpful social constructs around weight and shape, exercise and diet, for example influencers talking about diets and exercise or promoting diet products, often glorifying being thin and encouraging restriction of food[148].
- Promotion of unhelpful perspectives that reinforce disordered behaviour and thoughts; particularly insidious are pro-eating disorder communities[149] posting content that directly encourages eating disorders and disordered behaviour[150].
- Editing of photos to distort bodies in unrealistic ways alongside a lack of regulation that compels the disclosure of editing of images presented[151] leading the person with T1DE to believe those bodies are real and aspirational.
- The opportunity to connect with other unwell individuals, further normalising disordered thoughts and behaviours.

People with diabetes are often members of diabetes related groups and communities online. Much of the content in these groups is positive and helpful but some content may pose a risk to people with T1DE whilst not being harmful to people with type 1 diabetes who do not have an eating disorder. Community members talk about diet, eating habits and exercise in detail and sometimes give advice to other people with diabetes on what they should eat.

b) Traditional media

TV programmes, magazines, newspapers and radio programmes often promote unhealthy standards for people's bodies[152]. Fat phobia is prolific with detailed discussion about how to lose weight while shaming overweight people. This weight stigma and the focus on exercise and dieting can reinforce unhealthy thoughts and behaviours[148].

c) Fitness apps and calorie trackers

These are designed to promote healthy behaviours, however whilst they may not cause harm to those without an eating disorder, for those that are vulnerable including those with T1DE, they can lead to maladaptive eating and exercise behaviours[153], for example promoting an obsessive focus on achieving a particular number of steps a day or counting calories consumed or burned.

d) The role of healthcare professionals

HCPs can help to mitigate some of the risks outlined above through promoting more appropriate ways to engage with media, for example by encouraging people with T1DE to unfollow influencers who promote diets, leave pro-eating disorder communities, delete calorie counting apps and fitness trackers and avoid consuming diet or weight related media. It may be appropriate for healthcare professionals to signpost to more helpful online destinations like T1DE peer support groups or body positive or Health at Every Size (HAES) social media influencers.

4.4 Safeguarding

Health providers are required under statute and regulation to have effective arrangements in place to safeguard and promote the welfare of children and adults at risk of harm and abuse in every service that they deliver[154].

Section 11 of the Children Act 2004, requires health services to ensure that they consider the need to safeguard and promote the welfare of children when carrying out their functions[155]. They also have a duty to safeguard all adult patients and provide additional measures for patients who are less able to protect themselves from harm or abuse*[156]. Forms of abuse and their descriptions for children and adults can be found elsewhere [157, 158].

The Care Act 2014 highlights the aims of safeguarding adults are to[158]:

- Prevent harm and reduce the risk of abuse or neglect to adults with care and support needs.
- Safeguard individuals in a way that supports them in making choices and having control in how they choose to live their lives "Making Safeguarding Personal".

If a clinician has a safeguarding concern they should follow their own local policies and procedures and seek advice from their organisation's safeguarding team if unsure.

The Care Act 2014 is clear that clinicians should safeguard adults in a way that supports them in making choices and having control in how they choose to live their lives and highlights that it is important to recognise that an adult who has capacity and is capable of giving their consent has the right to refuse treatment and that this right must be respected[158]. It is important to recognise that though an individual with capacity has the right to refuse care for themselves such a refusal may give rise to a safeguarding concern in respect of others[158].

Given this and the evidence that those with eating disorders can lack insight into their illness (section 3.5), health and social care professionals working with those with T1DE must consider the safeguarding risks to others as well as to the person with T1DE. If a clinician has any concerns of this nature they should follow their own local policies and procedures and seek advice from their organisation's safeguarding team if unsure.

Given the potential for those with T1DE to disengage from services (see section 4.1), health care professionals must also recognise their responsibility to those under 18 and adults at risk who regularly miss appointments. Local policies in relation to missed appointments to safeguard these groups should be followed.

* The definition of an adult at risk is an adult aged 18 years or over who may be in need of community care services by reason of mental or other disability, age or illness; and who is or may be unable to take care of him or herself, or unable to protect him or herself against significant harm or exploitation[158].

4.5 Self neglect

Self-neglect is a lack of self-care to an extent which will impact significantly on physical, emotional or social wellbeing. It is one of the categories of abuse identified in the Care Act 2014[158] and is known to be a feature of eating disorders equivalent to that of other mental health illnesses[159].

It may be useful to consider if the self-neglect is intentional or unintentional.

- Intentional self-neglect may suggest that an individual is in denial of or lacks insight into their condition (see section 3.5) or that they fear the consequences of self-care e.g. weight gain or social interaction. There may be avoidance in facing up to the reality of their condition or a mistrust of the care and advice they are being given. In these cases, the importance of relationship building and the development of a trusting therapeutic relationship is vital in supporting the individual to make changes. Once the trust is established this may form a basis on which changes in behaviour can be supported.
- Unintentional self-neglect may be a sign of intellectual or cognitive impairment but may also be evidence of depression and / or diabetes related "burn out" (see section 3.3 (d)). In the context of T1DE the extensive and complex self-care required in type 1 diabetes can exhaust the resources and resilience of the individual, resulting in them neglecting to take the actions they need in order to manage their condition (see section 3.3(d)). Empathic understanding and support are needed to enable these individuals to recharge their batteries and not to feel judged as they struggle to cope with the demands of the condition and the expectations of both professional and informal carers. Motivational approaches may help these individuals to find the reason to make changes.

In its extreme form, consideration of the impact of self-neglect on both safeguarding and mental capacity to consent to treatment needs to be given (see sections 4.4 and 3.8).

4.6 Work and education

Type 1 diabetes, eating disorders and therefore T1DE have the potential to impact on a person's education or work due to amongst other things:

- Discrimination in the workplace or educational establishment.
- Time away from work or education for appointments.
- Time away from work or education due to periods of illness or treatment.

This has the potential to impact on a person's achievement in education or progression at work with subsequent financial impact. Employment is crucial to maintain social role and status and remaining in work is an important long term prognostic indicator in severe mental illness[160].

The Equality Act 2010 protects the rights of those with a disability and states an employer, school, college or higher education institution should be expected to make reasonable adjustments to prevent discrimination from taking place[161,162]. Reasonable adjustments might include for example making accommodations for time away from work for appointments, allowing extensions on assignments due to an inpatient stay or facilitating a phased return to work.

Despite reasonable adjustments, some with T1DE may be physically or mentally unable to work or engage in education. Under these circumstances they may be entitled to financial support for example through the Employment and Support Allowance (ESA). Personal Independence Payment (PIP) is intended to cover some of the costs of having a long term health condition and is available to those either in or out of work[163]. Contact details for organisations that can offer advice around work and benefit related issues are available elsewhere[164] https://www.diabetes.org.uk/resources-s3/2019-10/EmploymentandDiabetes_A5_v7.pdf .

Where there are employer or educational establishment concerns regarding a person with T1DE's fitness for work or to be in education, HCPs may be contacted by the person's employer or educational establishment seeking a professional perspective. In these circumstances consent should be obtained from the person in question to release medical information and support offered if there are concerns around this.

An HCP may themselves have concerns regarding a person with T1DE's fitness to undertake their working duties safely or attend their place of education. HCPs have a duty to protect the patient and the public, with professional guidance from regulators around this. If there is an acute high risk to the public then breaching patient confidentiality can be considered. Concerns should be approached from a multidisciplinary team (MDT) perspective and, after discussion with the patient, advise of the need to contact their appropriate occupational health or human resources department or line manager. It is always better to encourage the patient to make the disclosure themselves but the HCP can do it if necessary.

The HCP's role is not to make an assessment of work or educational capability, but to recognise that someone may be struggling and may need support, and to consider the influence of behaviour and mental health on the ability to maintain a safe working environment.

4.7 Driving

The Driver and Vehicle Licensing Agency (DVLA) highlight applicants and licence holders have a legal duty to[165]:

- Notify the DVLA of any injury or illness that would have a likely impact on safe driving ability.
- Respond fully and accurately to any requests for information from either the DVLA or HCPs.
- Comply with the requirements of the issued licence, including any periodic medical reviews indicated by the DVLA.

They should also adhere, with ongoing consideration of fitness to drive, to prescribed medical treatment, and to monitor and manage the condition and any adaptations.

All those with type 1 diabetes need to inform the DVLA and additional circumstances which Group 1 and Group 2 drivers with type 1 diabetes must disclose are clearly outlined by the DVLA[166]. The DVLA [165] also gives comprehensive guidance on driving in relation to visual disorders and limb complications e.g. peripheral neuropathy and amputation which may be relevant to those with type 1 diabetes.

In relation to eating disorders there is no specific guidance issued by the DVLA however they do provide guidance on co-morbidities that may be relevant including ECG abnormalities, severe

anxiety and depression, drug and alcohol dependency and mild cognitive impairment[165] which can be a feature of both starvation and hyperglycaemia (See sections 2.11 (d), 2.42 and 3.6).

The DVLA[165] highlights clinician responsibilities, advising that doctors and other HCPs should:

- Advise the individual on the impact of their medical condition for safe driving ability.
- Advise the individual on their legal requirement to notify the DVLA of any relevant condition.
- Treat, manage and monitor the individual's condition with ongoing consideration of their fitness to drive.
- Notify the DVLA when fitness to drive requires notification but an individual cannot or will not notify the DVLA themselves.

In relation to T1DE HCPs therefore need to consider any risks that may affect the person's ability to drive safely in the context of the guidance from the DVLA and the General Medical Council (GMC). Although not exhaustive, risks might include deterioration in vision, impaired cognitive function, severe hypoglycaemia, fatigue and cardiac risk. If the person lacks insight into their situation (see section 3.5), it may mean that they are unable to see the presenting risks as they are and therefore be less likely to notify the DVLA themselves. The GMC and The College of Optometrists offer clear guidance about notifying the DVLA when the person cannot or will not exercise their own legal duty to do so[167].

As well as DVLA written guidance[165], the GMC highlights the need to seek the advice of an experienced colleague or the DVLA's medical adviser if you are not sure whether a condition or treatment might affect a patient's fitness to drive[167].

5.0 CONSIDERATIONS FOR HEALTH CARE PROFESSIONALS

Working with people with T1DE can be challenging at times and may raise significant frustration, doubt and anxiety for health care professionals (HCPs) relating to the physical and mental health risks to the individual, as well as the often slow pace of change. Perceptions of clinical effectiveness may be exacerbated by a lack of diagnostic criteria, theoretical models of T1DE and a very limited evidence base for effective treatment (see section 3.1). Professionals from both specialties (diabetes and eating disorders) can feel out of their depth, particularly related to issues surrounding the other comorbidity, which can drive strong emotional reactions and has the potential to impact the HCP's own well-being.

Concerns about the risks to the physical and mental health of the person with T1DE can impact upon the way interventions are delivered, for example triggering a natural response in HCPs to try to set things immediately right by offering advice and warning about the consequences of actions / inaction. This has the potential to impact on the effectiveness of treatment by damaging engagement.

There are some key features of working with individuals with T1DE that are likely to be protective of HCPs' well-being and therefore likely to benefit the effectiveness of treatment for the individual with T1DE:

a) Joint working

Diabetes and eating disorder services proactively making contact with each other facilitates learning about each other's services and clinical specialty. Longer term this could be the groundwork towards developing local T1DE services that encompass clinicians from both specialties who share expertise and work jointly.

b) Education

To be able to provide effective treatment to people with T1DE it is important that the healthcare teams each have a firm knowledge of both specialist areas. For example, people living with type 1 diabetes are specialists in their own self- management skills and struggle to engage if their HCP is not conversant with a reasonable depth of knowledge; similarly someone struggling with an eating disorder might find it difficult to open up about their thoughts and feelings about weight and shape if their diabetes HCP is unable to develop a safe, non-judgemental space to talk about this and have confidence to ask in the first place.

It is essential that educational opportunities are made available to both diabetes and eating disorder services to develop their understanding of T1DE. This could be through online learning packages or through opportunities for shadowing between the specialist areas. Other medical specialists from both primary and secondary care and allied health care professionals, who may support people with type 1 diabetes, would also benefit from educational opportunities and increased awareness, for example settings such as accident and emergency and acute medical units where they may see people with recurrent diabetic ketoacidosis which may be a manifestation of insulin restriction / omission, as well as eating disorder inpatient units who might be asked to admit people experiencing T1DE.

c) Health care professional peer supervision groups

Beyond joint case management and intervention delivery there is value in considering the use of a joint peer supervision group. The importance of not only training but of actively supporting staff to access supervision and mentoring has been highlighted[100].

In peer supervision, group members often bring with them expertise related to their respective specialities which can assist in the mutual development of knowledge and skills, raise awareness of the emotional impact of their work on them as a HCP as well as providing other perspectives which can be very useful when having to consider multiple variables while working with complex clients. Group members can work through thoughts and feelings in the process of reviewing their actions with the support of their colleagues who can provide challenges, different perspectives, and shared solutions.

These groups can be a space to assist in containing the anxieties about different ways of working, pace of work and risks faced by the person with T1DE. Peer supervision can act as an anchor providing a place of mental refuge from the stressors of this type of work and can give clinicians a greater sense of security in their clinical work.

Facilitator led or one to one clinical supervision are alternative models of supervision that can be considered.

APPENDICES

APPENDIX A: Questionnaire used in diabetes clinics adapted to include weight and shape concerns

Type 1 Diabetes Clinic Questionnaire

Name: _____

Date: _____

Today you will be seeing your consultant plus one of the team members either your nurse or dietitian. It can be difficult in the clinic room to remember what you wanted to discuss or you may not have thought about it.

A. Is there anything in particular you would like to discuss today?

……

……

Would you like to discuss any of the subjects below?
Tick as many or as few as you would like!

Topic	
Travel, things such as: how to adjust insulin for long flights, eating in different countries, storing insulin when you are travelling	
I would like to learn about how alcohol affects diabetes	
I would like to talk about how I feel about my diabetes (frustrated, fed-up?)	
How do I work out eating out with friends	
I would like to learn more about Insulin pumps	
I feel I could learn more about Carbohydrate counting	
I am really unhappy with my body image	
What tricks and techniques are there around managing exercise	
I want to think about planning for having a baby	
I am struggling with hypos	
I feel I am constantly worried about my weight	
How do I deal with snacks	
I think I could do with talking about sex and contraception	
What do I need to think about when starting college or University/ moving house	
I really struggle with my diabetes whilst at work	

B. It is important for us to think about and measure hypoglycaemia –please could you fill in the 3 questions below by circling the answers that describe how things are for you. (We can explain this more clearly in clinic if you prefer)

1. Do you know when your hypos are starting?

(Circle **1** if you are **always** aware and **7** if you can **never** feel them starting or somewhere along the line depending on how you feel about your own awareness of them)

1 2 3 4 5 6 7

2. How often in the last month have you had readings less than 3.9mmol/L without symptoms?

Never 1 to 3 times 1 time/week 2 to 3 times/week 4 to 5 times/week Almost daily

3. At what level do you start to feel symptoms of hypoglycaemia/ low blood sugar?

Less than 2.2mmol/L 2.2-3.3mmol/L 3.3-3.8 mmol/L 4-7 mmol/L 7-12mmol/L

C. Consider the degree to which each of the two items may have distressed or bothered you DURING THE PAST MONTH and circle the appropriate number:

	Not a problem	A slight problem	A moderate problem	Somewhat serious problem	A serious problem	A very serious problem
1. Feeling overwhelmed by the demands of living with diabetes	1	2	3	4	5	6
2. Feeling that I am often failing with my diabetes routine	1	2	3	4	5	6

APPENDIX B: Wessex ComPASSION pilot reinsulinisation and refeeding protocol for people with type 1 diabetes and eating disorders (T1DE)

All patients requiring support with reinsulinisation / refeeding should be considered at risk of shifts in electrolytes with the reintroduction of insulin and / or carbohydrate. The protocol outlined below has been developed by the ComPASSION TEAM. Dorset HealthCare University NHS Foundation Trust / Royal Bournemouth and Christchurch Hospitals NHS Foundation Trust.

Patients refeeding orally in a community setting:

A) FOOD

- In the first instance, if the person is eating very little, establishing a ½ portion refeeding meal plan approx. 1200 kcals alongside the suggested insulin protocol has in our experience been found to be safe. An example refeeding starter plan is outlined below.

- 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 as the refeeding risk is mediated by the amount of insulin provided, not by the amount of food.

- Refeeding vitamins are recommended as outlined below:

Over 18 years of age[91] ● Thiamine 100mg twice a day ● Vitamin B co-strong 2 tablets, three times a day ● Multivitamin and mineral supplement e.g. Forceval capsule or Forceval soluble tablet : one a day
Under 18 years of age[168] ● Evidence base is lacking however in older adolescents following adult guidelines on prescription of thiamine is justifiable.

Refeeding vitamins should be started as soon as possible but changes to food and insulin need not be delayed. Provision of these vitamins is particularly important if readiness for change is high and the person with T1DE is assessed as being likely to implement the advised changes at the pace proposed.

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

B) 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 0.2units/kg of body weight with planned incremental increases to meet the individual's requirements (expert opinion). This is alongside fixed doses of 2 units rapid acting insulin for meals (initially no insulin with snacks).

- Ketones should be tested pre-meal and if ketones are >1.5 an additional 2 units of rapid acting insulin should be given and sugar free fluids encouraged. Repeat hourly.

- 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 plan adjusted accordingly.

- 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.

- 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.

C) MONITORING

- Continuous or 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[169]. It is therefore recommended that refeeding bloods are taken at baseline and at day 3-4 (day 5 if days 3 & 4 both fall at a weekend). Then as clinically indicated. This is particularly important if readiness for change is high and the person with T1DE is assessed as being likely to implement the advised changes at the pace proposed.

- In the absence of robust evidence, if the planned pace of reinsulinisation / refeeding is faster than the proposed guidelines, if significantly more insulin is needed for the management of ketones or if there are other risk factors present such baseline electrolytes out of range, discuss with the medical team for further advice on frequency of monitoring of refeeding electrolytes.

- Local guidance for correcting blood levels if abnormal electrolytes are identified is outlined in Table 1 below. Always seek consultant 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. Whilst 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 1: guidance for the correction of electrolyte abnormalities: adults (> 18 years)[71,72]

Potassium	
3.5-5.0	Normal range
3.0-3.4	Replace orally with Sando K (12mmol K^+ per tablet), 2 tablets BD-TDS
<2.5	Requires intravenous replacement – refer patient to acute care
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

D) 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.

- In the community setting those close to the person e.g. family members, partners or friends may be able to offer support with meals and insulin administration. A T1DE service should include group or individual carers training and support. Alternatively, if the capacity is available, support with daily injections may be available from the local diabetes team. Even if this support cannot be offered at every injection it may help the person overcome their initial fear of re-engaging with appropriate insulin administration.

- If a person continues to struggle with re-engaging with eating and / or insulin administration a higher level of support may be required and consideration should be given to offering day service or specialist eating disorder inpatient unit support.

Additional notes for those attending day service or being refed orally in a specialist eating disorder unit (SEDU)

- **FOOD:** Follow guidance for food as documented for patients refeeding orally in a community setting.

- For those not completing meals and snacks, nutritionally complete supplements should be offered as an alternative in order 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 NB: that some nutritional supplements do not contain carbohydrate for example Calogen (Nutricia) and Fresubin 5kcal Shot (Fresenius Kabi).

- **INSULIN:** Follow guidance for insulin as documented for patients refeeding orally in a community setting. If ketones are present pre-meal follow hyperglycaemia / ketone management plan in Appendix C.

- **MONITORING:** As a minimum, check refeeding bloods at baseline, day 2, day 5 and thereafter as medically indicated.
- In the absence of robust evidence, if the planned pace of reinsulinisation / refeeding is faster than the proposed guidelines, if significantly more insulin is needed for the management of ketones or if there are other risk factors present such baseline electrolytes out of range, discuss with the medical team for further advice on monitoring of refeeding electrolytes.
- **SUPPORT:** In day service or SEDU settings it can be helpful to consider a staged approach in order to support the person to move forward with re-engaging with taking their insulin. A staged guide to achieving this has been developed. See Wessex ComPASSION pilot inpatient insulin management care plan for people with type 1 diabetes and eating disorders (T1DE) below.

Daily Meal plan

Refeeding starter plan

BREAKFAST:
Carbohydrate: 1 ½ cups Rice Crispies/Cornflakes, or 1 cup other cereal.
Add some protein: ¾ cup semi-skimmed milk or 2 tbsp whole milk yogurt

MORNING SNACK: 1 cup semi-skimmed milk

LUNCH:
Carbohydrate: 1 slice of bread or ½ bagel or 1 small pita bread or 1 small jacket potato (to fit in ½ the palm of your hand)
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 or fruit: Small bowl salad, ½ portion fruit
Add a fat source: 1 level tsp butter / margarine / mayonnaise or salad cream

Include a 2nd course e.g
Dairy option: ½ standard pot of whole milk yogurt *(e.g Muller Fruit Corner, Activia or Greek Style)* or ½ rice pudding/custard pot or 1 scoop of ice-cream.

AFTERNOON SNACK: 1 cup semi-skimmed milk

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, 6 chips/wedges.
Add some protein: ½ chicken breast or equivalent meat portion, 1 rounded serving spoon of Mince (1 ½ if a high vegetable content), x ½ baked fish fillet, 2 chipolata sausages or fish fingers, 1 egg, 1 rounded serving spoon quorn pieces/kidney beans/chick peas/cooked lentils (1 ½ if a high vegetable content).
Add some vegetables or fruit: ½ serving spoon of vegetables, small bowl salad
Add a fat source: 1 tsps 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).
¼ of a whole regular size thin crust pizza, plus a vegetable/salad portion.

Include a 2nd course e.g
Dairy option: ½ standard pot whole milk yogurt *(e.g Muller Fruit Corner, Activia or Greek Style),* or ½ rice pudding/custard pot or 1 scoop of ice-cream.

SUPPER SNACK: 1 cup semi-skimmed milk + 1 digestive biscuit

FLUIDS: Aim for 6-8 mugs of fluid spread over the day

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The Royal Bournemouth and Christchurch Hospitals NHS Foundation Trust

Wessex ComPASSION Pilot Inpatient Insulin Management Care Plan for people with Type 1 diabetes and eating disorders (T1DE)

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.	☐

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 x Unit of insulin for every ..... grams carbohydrate breakfast

  ..... grams carbohydrate lunch

  ..... grams carbohydrate evening meal

- **Adjustment doses**

  1 x Unit of insulin will reduce blood sugar by Units

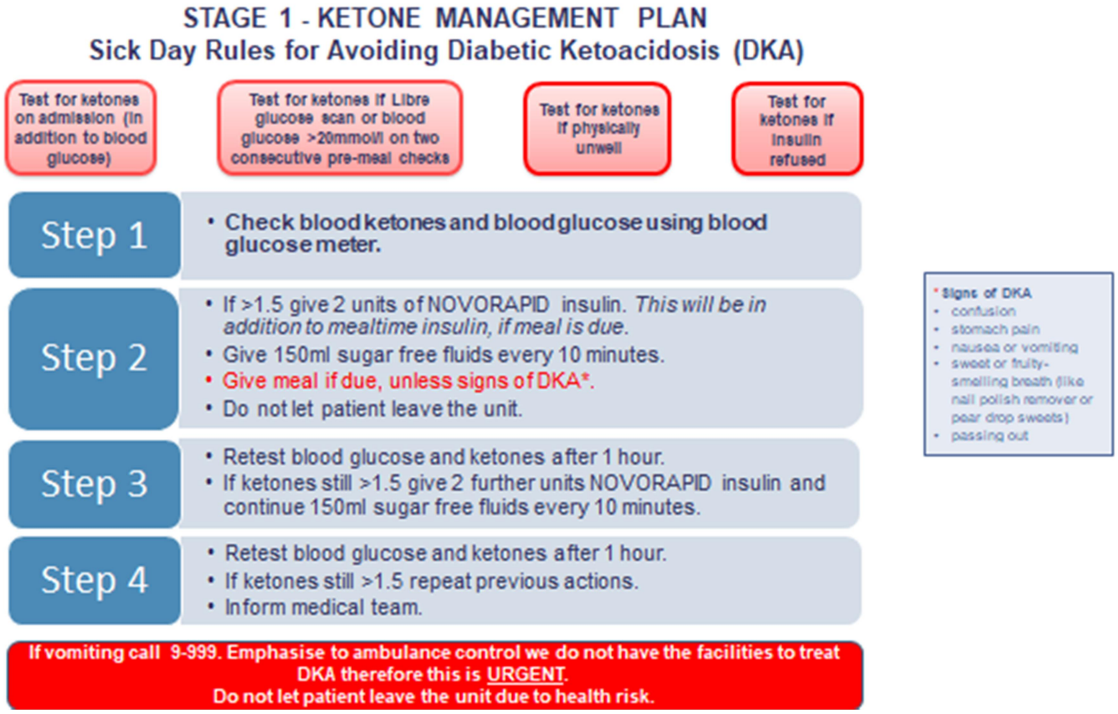
4. Insulin refusal

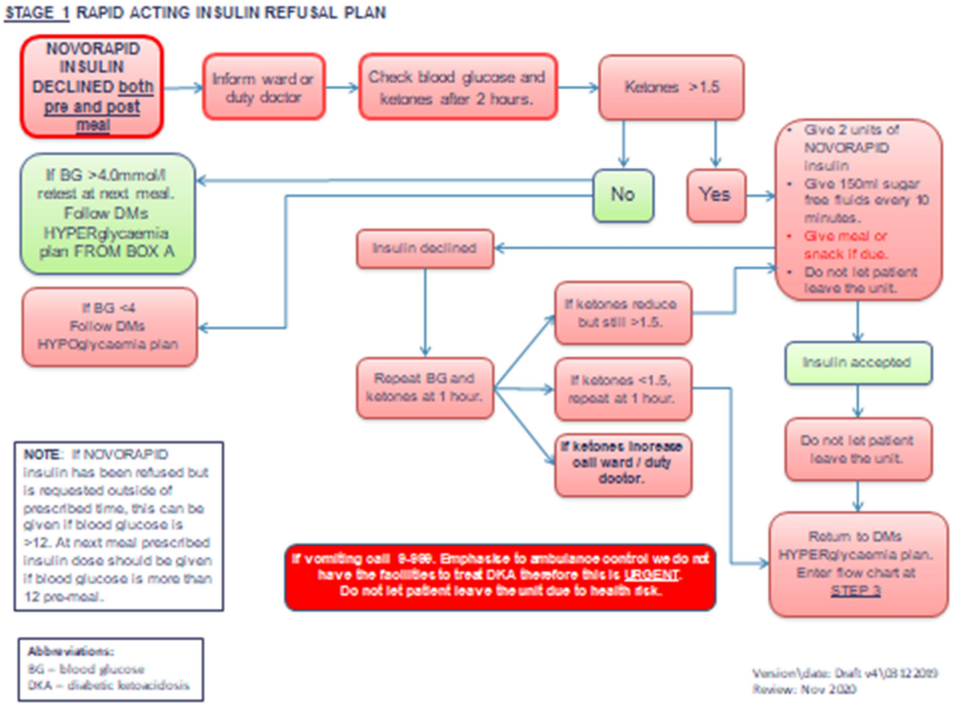
- Encourage insulin pre-meal as per prescription chart.
- If not accepted within 5 minutes continue with meal and offer again ONCE post meal.
- If insulin refused follow Ketone Management Plan.

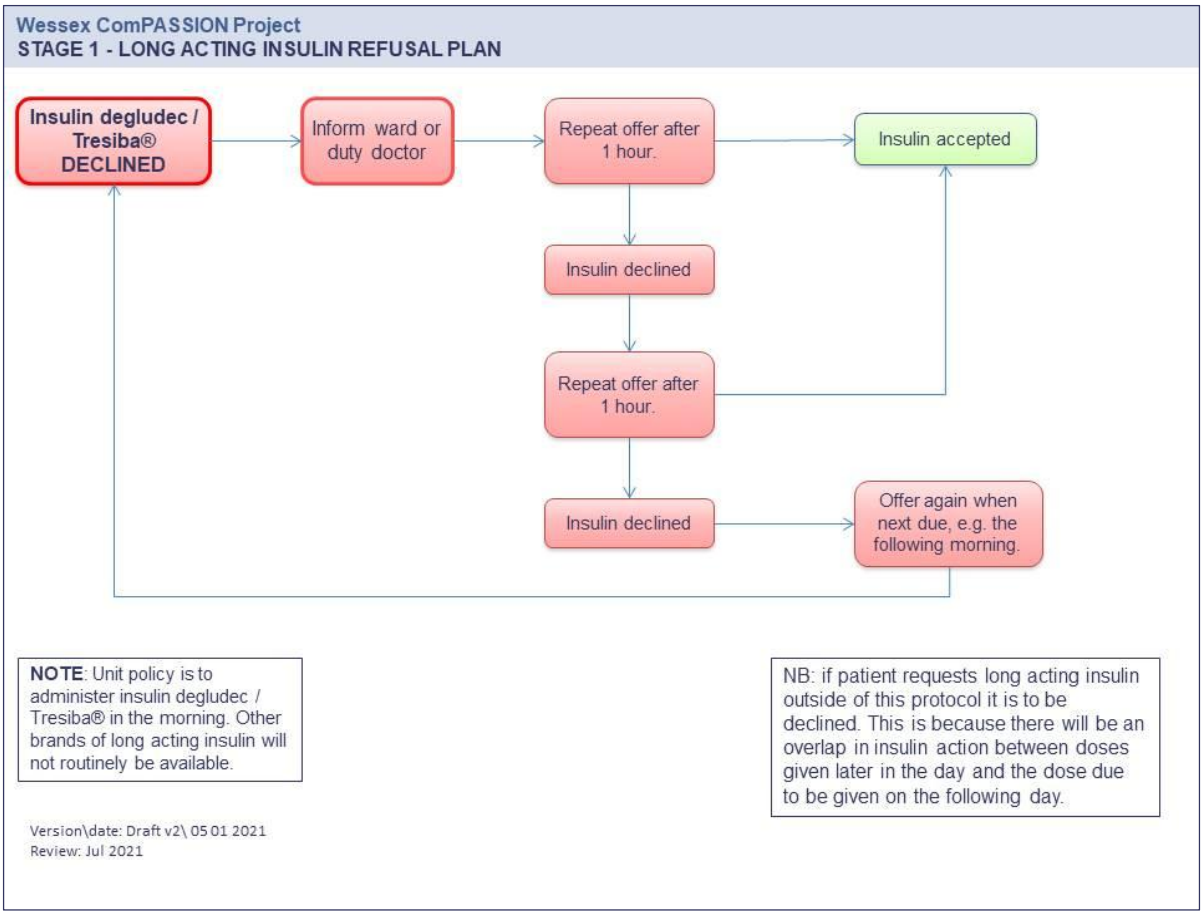
5. Carbohydrate refusal

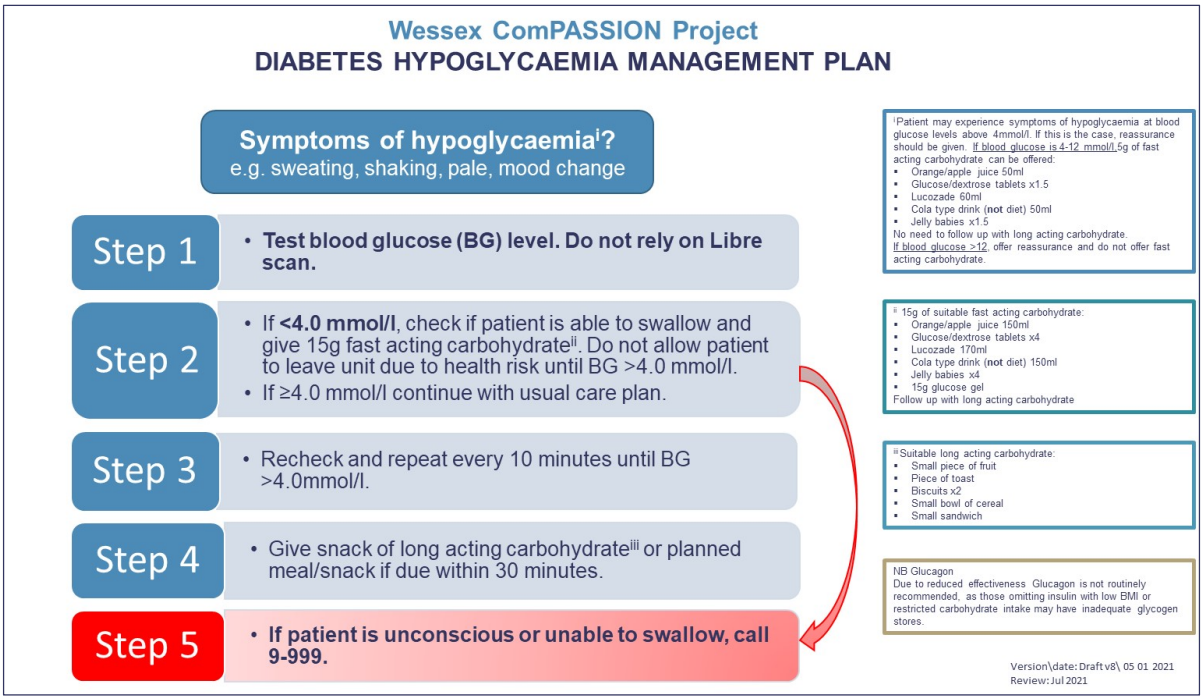
- If a person refuses their meal or the carbohydrate part of their meal or a snack and has taken their rapid acting insulin for that meal or snack, monitor their blood glucose levels every hour for the next four hours.
- If the person will accept carbohydrate in the interim this should be accommodated for example: 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.

APPENDIX C: Wessex ComPASSION pilot inpatient protocols for the management of ketones, insulin refusal and hypoglycaemia






Wessex ComPASSION Project
STAGE 1 - LONG ACTING INSULIN REFUSAL PLAN
Insulin degludec / Tresiba® DECLINED
Inform ward or duty doctor
Repeat offer after 1 hour.
Insulin accepted
Insulin declined
Repeat offer after 1 hour.
Insulin declined
Offer again when next due, e.g. the following morning.
NOTE: Unit policy is to administer insulin degludec / Tresiba® in the morning. Other brands of long acting insulin will not routinely be available.
NB: if patient requests long acting insulin outside of this protocol it is to be declined. This is because there will be an overlap in insulin action between doses given later in the day and the dose due to be given on the following day.
Version\date: Draft v2\ 05 01 2021
Review: Jul 2021


Wessex ComPASSION Project
DIABETES HYPOGLYCAEMIA MANAGEMENT PLAN
Symptoms of hypoglycaemia[i]?
e.g. sweating, shaking, pale, mood change
Step 1
• Test blood glucose (BG) level. Do not rely on Libre scan.
Step 2
• If <4.0 mmol/l, check if patient is able to swallow and give 15g fast acting carbohydrate[ii]. Do not allow patient to leave unit due to health risk until BG >4.0 mmol/l.
• If ≥4.0 mmol/l continue with usual care plan.
Step 3
• Recheck and repeat every 10 minutes until BG >4.0mmol/l.
Step 4
• Give snack of long acting carbohydrate[iii] or planned meal/snack if due within 30 minutes.
Step 5
• If patient is unconscious or unable to swallow, call 9-999.
[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,5g 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.
Version\date: Draft v8\ 05 01 2021
Review: Jul 2021

APPENDIX D: Wessex ComPASSION pilot pathway for engagement and non-engagement

ComPASSION Pathway for Engagement and Non-engagement

Initial contact

- Initial contact by phone to arrange a mutually convenient time.
- Use this opportunity to address anxieties and start to build trust.
- If requested, confirm any agreed appointment by letter.

Communication Tips

If unable to make contact by phone, send appointment letter including clear instructions for contacting the team if the appointment time is not convenient.

If possible this contact should not be an automated response, particularly within office hours.

Consider additional communication needs e.g. privacy, sight loss or learning disability. Adapt your contact style and resources.

Managing non-attendance

- Non-attendance at initial or follow-up appointments should trigger attempts to make timely contact by telephone / text / email / letter.
- Consider any preferred communication medium when deciding how to make this contact e.g. younger people may prefer the informality of a text. Also consider any known relationship preferences of the person with different members of the MDT.
- If contact is made use this as a further opportunity to address anxieties and to build trust. Several informal contacts may be helpful before moving to formal assessment or treatment options.

Consider language (verbal and body language) and writing style in your communications.

Escalating non-attendance

If it is not possible to initiate or sustain contact or where there is a pattern of non-attendance, discussion with the wider MDT should take place in a time appropriate manner and a decision made concerning next steps.

Next steps considered should include information from other agencies, e.g. GP, Social Care and Community Mental Health Teams and will be based on the known physical and mental health risks. If risks are high consider:

- Contact with relatives if consent to share has been gained to support engagement.
- Outreach visits to check on wellbeing and encourage engagement
- Considering contact with relatives irrespective of consent to share if risks are considered high enough.
- Safeguarding concerns
- Police welfare checks
- The right not to engage in treatment
- Assessment under the mental Health Act

In all cases the approach should remain empathic, non-judgemental and supportive with the aim of fostering a positive therapeutic relationship.

Ensure all attempts at contact and all discussions regarding engagement are documented in the patient notes.

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