

ISPAD Clinical Practice Consensus Guidelines 2009 Compendium

Assessment and management of hypoglycemia in children and adolescents with diabetes

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Introduction

Hypoglycemia is one of the most common acute complications of the treatment of type 1 diabetes

Hypoglycemia is the result of a mismatch between insulin dose, food consumed, and recent exercise and is rarely, if ever, a spontaneous event. A careful review of only blood glucose (BG) records will yield a retrospective prediction of the hypoglycemic event for at least 50% of events (1, 2). Because it can be accompanied by unpleasant, embarrassing, and potentially dangerous symptoms and because it causes significant anxiety and fear in the patient and their caregivers, its occurrence is a major limiting factor in attempts to achieve near normal BG levels (3, 4). Additionally, in its extreme manifestations, hypoglycemia can lead to permanent sequelae and even death (5–8).

Epidemiology

Intensive diabetes management initially resulted in a dramatic increase in the rate of hypoglycemia in

adolescents (9, 10). Greater experience with intensive therapy and use of analogue insulins decreased the rates of severe hypoglycemia to 8–30 episodes per 100 patient-years of diabetes exposure (11–15), with the exception of very low incidence (4/100 person-years) in a Finnish study (16).

Non-modifiable predictors of severe hypoglycemia are:

- Age (infancy and adolescence) (11, 14);
- Increased duration of diabetes (12, 13, 15).

Modifiable predictors are:

- Lower hemoglobinA1c (HbA1c) and
- Higher insulin dose.

Direct health care cost of severe hypoglycemic events is estimated at 7 400 euros per 100 patients per year in the 1990s (17). Further studies are needed to update these figures and to estimate, in addition, indirect costs (e.g., lost productivity and diminished quality of life).

Signs and symptoms

Hypoglycemia is often accompanied by signs and symptoms of autonomic (adrenergic) activation and/or neurological dysfunction (neuroglycopenia). Children may also exhibit behavioral or mood changes when their BG falls but remains within or above the normal range (18, 19).

Autonomic signs and symptoms

- Trembling;
- Pounding heart;
- Cold sweatiness;
- Pallor.
- Neuroglycopenic signs and symptoms
- Difficulty concentrating;

Blurred vision or double vision;

- Disturbed color vision;
- Difficulty hearing;
- Slurred speech;
- Poor judgment and confusion;
- Problems with short-term memory;
- Dizziness and unsteady gait;
- Loss of consciousness;
- Seizure;
- Death.

Behavioral signs and symptoms

- Irritability;
- Erratic behavior;
- Nightmares;
- Inconsolable crying.

Non-specific symptoms (associated with low, high, or normal BG)

- Hunger;
- Headache;
- Nausea;
- Tiredness.

Definition

There is no consistent or agreed upon numerical definition of hypoglycemia for the child with diabetes. Nevertheless, BG values below 3.3–3.9 mmol/L (60–70 mg/dL) are generally agreed to place the individual at risk for severe hypoglycemia because BG values in this range are associated with alterations in the counterregulatory hormones essential to the spontaneous reversal of hypoglycemia (14, 20, 21). For clinical use, the value of <3.6 mmol/L (65 mg/dL) has been most often used as the level for defining hypoglycemia in the pediatric population. However, a recent American Association (ADA)

Working Group report suggested using 3.9 mmol/L (70 mg/dL) as the definition in all age groups for research purposes in evaluating therapies designed to alter frequency of hypoglycemia (21). Therefore, in the interest of avoiding hypoglycemia and maintaining consistency in reporting hypoglycemia, 3.9 mmol/L (70 mg/dL) is the recommended lower target for BG levels in children and adults with insulin-treated diabetes (14, 21).

Hypoglycemia is that low BG level that exposes a child to possible harm

Alterations in activation of autonomic symptoms or neuroglycopenia (cognitive dysfunction) have been documented to occur at BG levels as high as 3.6 mmol/L (22). However, the absolute BG level at which signs and symptoms begin to occur may vary among individuals and within the same individual at different times and in different situations (23).

Young children and their parents are not as accurate at recognizing early warning signs and symptoms of low BG, as are adults with diabetes (24). Therefore, frequent BG monitoring should be encouraged especially during times at which extreme BG levels might be anticipated (overnight, at the peak of insulin actions, during and after strenuous exercise, when insulin doses are adjusted, during stressful situations, during illnesses, etc.).

The *BG threshold for activation of autonomic signs and symptoms* is related to activation of counterregulatory hormones and has been shown to be higher in children than in adults and to vary directly with the level of BG control (higher HbA1c, higher BG threshold) (14, 25). The BG threshold for symptoms may be affected by antecedent hypoglycemia or hyperglycemia (26). This may be accompanied by reduced intensity of symptoms over 24 h following a hypoglycemic event, leading to reduced awareness of hypoglycemia during this time. Low or moderate exercise one day may result in a decrease in symptoms of hypoglycemia and decreased hormonal reactions the next day (27). Also, the BG threshold is reduced during sleep (28, 29).

The *BG threshold for neuroglycopenia* neither varies as much with the level of glucose control nor with antecedent hypoglycemia (14, 30–33). Therefore, neuroglycopenia may occur before autonomic activation and be associated with reduced awareness of the onset of hypoglycemia (3). This phenomenon is termed *hypoglycemia unawareness* and is an important cause of severe hypoglycemia, accounting for 36% of the hypoglycemia that occurred during the Diabetes Control and Complications Trial (DCCT) while subjects were awake (12). A single hypoglycemic episode can lead

to significant decrease in neurohormonal counter-regulatory responses and cause an unawareness of hypoglycemia (26). The syndrome is usually associated with decreased glucagon and/or epinephrine output (24, 34). There is evidence that loss of awareness of hypoglycemia can be reversed by avoiding hypoglycemia for 2–3 wk (33, 35, 36), although this may be difficult to accomplish in young children who are less able to identify hypoglycemia.

- Avoidance or reversal of hypoglycemia unawareness is crucial to achieving optimal glycemic control without an unacceptable risk for hypoglycemia (36) (C).

Severity of hypoglycemia

Mild/moderate hypoglycemia

Hypoglycemia has often been described as mild, moderate, or severe based on the individual's ability to treat him/herself. However, there are no clinically important reasons to distinguish between mild and moderate hypoglycemia, and younger children will almost always need to be treated by a parent or caregiver. Therefore, mild and moderate hypoglycemia are considered together.

- The child or parent is aware of, responds to, and treats the hypoglycemia orally after documenting a BG level of ≤ 3.9 mmol/L (70 mg/dL). The ADA (21) has suggested using the terminology of 'Documented Symptomatic Hypoglycemia' for this category.
- Asymptomatic hypoglycemia applies when the child is not symptomatic with hypoglycemia but the BG is documented to be ≤ 3.9 mmol/L (21).
- The category of asymptomatic hypoglycemia, especially if <3.6 mmol/L (65 mg/dL), is suggested because it is important to recognize the frequency of hypoglycemia unawareness or glucose values that place an individual at risk for hypoglycemia unawareness.

Severe hypoglycemia

- The child is having altered mental status and cannot assist in their care, is semiconscious or unconscious, or in coma \pm convulsions and may require parenteral therapy (glucagon or i.v. glucose).

Treatment

Goal

The goal is to restore the BG level to euglycemia [BG = 5.6 mmol/L (100 mg/dL)].

It should be remembered that capillary BG estimation at low BG levels is less accurate than at higher levels, and therefore, low BG numbers should be interpreted with caution.

Mild/moderate

If the BG is 3.5–3.9 mmol/L and the child does not have uncomfortable symptoms, immediate intake of most carbohydrates will raise the BG sufficiently. In adults, 20 g of carbohydrate in the form of glucose tablets raised BG by approximately 2.5–3.6 mmol/L (45–65 mg/dL) (37, 38, 39). This has been extrapolated to 0.3 g/kg in children (40). However, it is important to remember that the amount of carbohydrate required will depend on the size of the child, type of insulin therapy, proximity to recent insulin dosage, the intensity of antecedent exercise as well as other factors (37, 41). The type of carbohydrate is also important as 40 g of carbohydrate in the form of juice was needed to give approximately the same rise as 20 g in the form of glucose tablets (37), sucrose likewise requires a greater amount to provide the same increase in blood glucose (38). Milk containing 20 g of carbohydrate gave only a rise of approximately 1 mmol/L (20 mg/dL). This has been explained by delayed emptying of the stomach because of the fat content (42).

Treatment of hypoglycemia should be provided promptly and should provide immediate oral, rapidly absorbed, simple carbohydrate calculated to raise BG level to 5.6 mmol/L (100 mg/dL), [1 g glucose should raise BG by approximately 0.17 mmol/L (3 mg/dL) for the average adult]. The amount of carbohydrate required will depend on the size of the child, type of insulin therapy, and proximity to recent insulin dosage as well as the vigorousness of the immediate antecedent exercise, if any (41). To increase the BG approximately 3–4 mmol/L (55–70 mg/dL) give glucose tablets/sugar lumps or a sweet drink (glucose/sucrose drinks, cola etc.), approximately 10 grams of glucose is needed for a 30 kg child and 15 grams for a 30 kg child. If sucrose or fructose are used, slightly higher amounts are required compared to pure glucose (38, 37).

- Chocolate, milk and other foods containing fat will cause the sugar to be absorbed more slowly and should be avoided as the initial treatment of hypoglycemia (37, 43).
- After treatment, wait 10–15 min, retest BG, if no response or an inadequate response, then repeat oral intake as above. Retest the BG in another 20–30 min to confirm that target glucose has been maintained and not exceeded (E).

- For initially lower glucose values, as symptoms improve or euglycemia is restored, the next meal or snack may be ingested (e.g., fruit, bread, cereal, and milk) to prevent recurrence of hypoglycemia.

Additional recommendations are given in the exercise section and in the International Society for Pediatric and Adolescent Diabetes (ISPAD) Guidelines for Exercise.

Improvement in signs and symptoms may lag behind improvement in BG level

Retesting to document normalization of glucose is essential to ensure adequate, and not excessive, treatment.

Severe

- Urgent treatment is required.
- Severe hypoglycemia with loss of consciousness \pm convulsions (particularly if there is vomiting) is most safely and rapidly reversed by injection of
- glucagon 0.5 mg for age <12 yr, 1.0 mg for ages >12 yr, or 10–30 mcg/kg body weight (44).

Glucagon is given intramuscularly or subcutaneously. In a hospital setting, i.v. glucagon may be given.

- if glucagon is unavailable or recovery is inadequate.
 - Intravenous dextrose should be administered slowly by trained personnel over several minutes, e.g., dextrose 10–30% at a dose of 200–500 mg/kg (dextrose 10% is 100 mg/mL) to reverse the hypoglycemia. Rapid administration, or excessive concentration, i.e., dextrose 50%, may result in an excessive rate of osmotic change.
 - When glucagon is not available, a common practice is to administer a rapid acting source of glucose (e.g. glucose gel or honey) into the buccal pouch (E). However, the efficacy of this practice is anecdotal and there is no scientific evidence for absorption of glucose from the buccal mucosa. On the contrary, there is one study in adults showing no buccal absorption of glucose (45).

In the recovery phase after treatment of severe hypoglycemia, close observation and BG monitoring is essential because vomiting is common and recurrent hypoglycemia may occur.

Should recurrent hypoglycemia occur, the child will require additional oral carbohydrate and/or i.v. infusion of glucose, e.g., dextrose 10%, 2–5 mg/kg/min (1.2–3.0 mL/kg/h).

Additional categories of hypoglycemia for clinical and research purposes

To fully categorize a patient's frequency of hypoglycemia, it should be recognized that individuals with diabetes may not always document an episode of hypoglycemia but may treat the event resulting in resolution of symptoms. These events should be recognized and may be categorized as *probable symptomatic hypoglycemia* (21). In addition, an event may occur when symptoms typical of hypoglycemia occur and are relieved by treatment but the BG value is >3.9 mmol/L. This may occur if the BG level falls rapidly or may occur in patients with chronically poor glycemic control (46). This is termed *relative hypoglycemia* (21). There is no evidence of any neurocognitive harm from relative hypoglycemia but, without appropriate patient education, it may become a barrier to achievement of more optimal glycemia.

Prevention of hypoglycemia—special considerations

Risk factors for hypoglycemia

Families should receive education about the times when hypoglycemia is more likely to occur so that more frequent glucose monitoring may be initiated. Hypoglycemia occurs more frequently:

- (i) when the treatment regimen is altered (more insulin, less food, and more exercise);
- (ii) in younger children;
- (iii) with lower HbA1c levels [The relation between severe hypoglycemia and lower HbA1c had been extensively explored (33), especially in children (11, 17, 35, 47, 48).];
- (iv) when there are frequent low BG levels (25);
- (v) when awareness of autonomic symptoms is reduced (49);
- (vi) during sleep (14, 28);
- (vii) after the ingestion of alcohol (50). Alcohol suppresses gluconeogenesis (51) and may induce hypoglycemia unawareness (52). In addition, alcohol ingestion acutely improves insulin sensitivity. In combination with exercise, drinking can lead to severe hypoglycemia, which may occur 10–12 h after the exercise or alcohol ingestion (53).

Comorbidities

The comorbidities of celiac disease, present in 4–10% of children with type 1 diabetes, and Addison's disease, present much less commonly (54), may also increase the risk for hypoglycemia (55, 56). The introduction

of a gluten-free diet and appropriate treatment of Addison's Disease may reduce the frequency of hypoglycemia (57, 58).

Exercise

The risk for hypoglycemia is increased during, immediately after, as well as 2–12 h after exercise. This effect is variable and depends on many factors including duration and intensity of exercise, type of insulin, and site of injection (41, 59, 60).

Evidence suggests that BG levels below 6.7–8.3 mmol/L (120–150 mg/dL), prior to sustained aerobic exercise (75 min) in the afternoon, is associated with a high probability of hypoglycemia within 60–75 min (61). Discontinuing continuous insulin infusion therapy for up to 2 h during exercise may help to prevent exercise-related hypoglycemia (61). During prolonged exercise, 15 g of carbohydrate will raise the BG by approximately 1 mmol/L for a 50-kg child (according to the Brodow extrapolation mentioned earlier) (37); therefore, 30–45 g of oral carbohydrate may be required to prevent hypoglycemia for a 30 kg child and 50–75 grams for 50 kg child, additional carbohydrate will usually be required if exercise occurs at the peak of insulin action (59–61). Likewise carbohydrate requirement will be lower if the premeal bolus prior to the exercise is lowered or if the exercise occurs several hours after the last meal bolus has been given. In many individuals, a lowering of the insulin dose after intense exercise should be considered to prevent nocturnal hypoglycemia.

Nocturnal hypoglycemia

Nocturnal hypoglycemia is often asymptomatic, does not necessarily disturb sleep patterns, and may be prolonged (62, 63). Cross-sectional studies found nocturnal hypoglycemia in 30–45% of children treated with the combination of evening regular (soluble) and intermediate-acting insulin (56, 64). When continuous glucose monitoring system (CGMS) was repeated every fortnight for 6 months, every participant had at least one nocturnal episode of low subcutaneous glucose (65). It should be suspected if prebreakfast BG is low, and/or confusional states, nightmares, or seizures occur during the night, or if impaired thinking, lethargy, altered mood, or headaches are experienced on waking. Research has shown that counterregulatory responses to low BG may be impaired during sleep (28).

Nocturnal hypoglycemia is not regularly predictable on the basis of a bedtime BG level and can only be confirmed by BG tests at regular intervals during the night or continuous glucose monitoring (62) (66, 67). A bedtime snack containing carbohydrate as well

as fat and protein may be useful in preventing nocturnal hypoglycemia, but this should not be at the expense of high overnight BG levels. Including protein in the bedtime snack seems to provide better overnight protection from hypoglycemia than simple carbohydrates alone (68). However, one study in adults indicated that the addition of protein (bread with meat) did not give better protection against hypoglycemia 3 h after the snack (69). Extra slowly absorbed complex carbohydrate at bedtime may be helpful especially following strenuous exercise in the afternoon or evening (63, 70–73).

Short- and long-acting insulin analogues and continuous insulin infusion therapy may decrease risk for nocturnal hypoglycemia (74, 75). Continuous glucose monitoring has been helpful in identifying the frequency and duration of nocturnal hypoglycemia (66, 67).

Brain dysfunction and neurological sequelae of hypoglycemia

Studies have shown an association between hypoglycemia and decrease in cognitive functioning in children with type 1 diabetes (76–78), particularly those diagnosed before the age of 5–6 yr (78, 79). Recently, the role of early-onset diabetes and chronic hyperglycemia to decrease cognitive functioning in very young children has received increased attention (80–82). On a practical basis, episodes of mild–moderate hypoglycemia, even if asymptomatic, have important implications for school and social well being. These include cognitive dysfunction (76, 83), reduced awareness of low BG, possible injury or accident, and significant fear of hypoglycemia, resulting in intentional decreases in insulin dosing causing elevated glucose levels and increased HbA1c (4, 84, 85).

Repeated episodes of hypoglycemic seizures in young children may cause permanent changes (66, 86–89), and brain imaging studies show that both hypoglycemia and hyperglycemia cause changes in the white and gray matter of developing brains (90). Hypoglycemic seizures lead to significant declines in verbal abilities (8), memory skills (67), and ability to organize and recall information (91). The latter may be impaired even after mild hypoglycemia (88). However, in one study, severe hypoglycemia did not have adverse effects on cognition in 6–15 yr olds during 18 months followup when comparing with diabetic children without severe hypoglycemia (92). Severe hypoglycemia in children may result in persistent electroencephalographic (EEG) changes; EEG abnormalities were found in 80% of diabetic children with a history of severe hypoglycemia compared with only 30% of those without and 24% of healthy control children (66). However, intensive insulin treatment in the DCCT cohort (aged 13–39

yr at baseline), while increasing the incidence of hypoglycemia, has not led to a significant worsening of neuropsychological or cognitive functioning during the trial (93, 94) as well as 18 yr after entry into the trial (95). This observation may be further evidence that the effect of severe hypoglycemia on long-term neuropsychological functioning is age dependent. However, there is increasing evidence that chronic hyperglycemia may be detrimental to the development of the brain. The intellectual development in boys with diagnosis, 6 yr of age correlated to metabolic deterioration at diagnosis and long-term HbA1c but not to hypoglycemia (80). A study in type 1 diabetic patients aged 25–40 years with disease duration of 15–25 years and minimal diabetes complications showed that both higher HbA1c levels and severe hypoglycemic events were associated with lower density of gray matter in brain regions responsible for language processing and memory (96). High blood glucose levels early in life may affect the brain's structure and development negatively, making the brain more vulnerable to any subsequent insult (hypoglycemia, head injury, alcoholism, other central nervous system conditions) that occurs later in the child's life (97) [E].

Severe hypoglycemia can also lead to increased worry, poor sleep, hospital visits, hospitalizations, excessive lowering of daily insulin dose, and worsening of subsequent glycemic control (98). Patients with severe hypoglycemia also reported lower global quality of life (99).

Death

Hypoglycemia is a significant factor in excess mortality in patients with diabetes (100). Despite recent improvements in therapy, diabetes-related mortality among children has not declined for 14 yr (101), and a recent US report suggests a slight increase in diabetes-associated mortality beginning in 1993–1994 (102). Sudden nocturnal death in young persons with type 1 diabetes has been described and is known as the 'dead in bed' syndrome (103). It appears to be responsible for about 6% of deaths in diabetic patients aged below 40 years (104). Nocturnal hypoglycemia has been implicated as the cause for these deaths, consistent with demonstrated impairment of counter-regulatory hormone response during sleep (28), the high frequency of nocturnal hypoglycemia reported by the DCCT (12), and more recent studies using continuous glucose monitoring (105). Nighttime severe hypoglycemia can lead to hypokalemia, arrhythmia, and death (18).

Hypoglycemia is potentially preventable because its occurrence is frequently predictable. But because it is often associated with significant psychosocial dysfunction, prevention may be difficult. Most importantly, the

knowledge that it can in rare cases lead to permanent long-term sequelae and is potentially life threatening makes education for children/adolescents, their parents, and other caregivers essential. Particular attention should be given to training children and their caregivers to recognize the early warning signs of hypoglycemia and treat low BG immediately and appropriately. To quickly reverse hypoglycemia unawareness, patients and their parents should be trained to contact their diabetes care provider if hypoglycemia is documented without symptoms or if the symptoms are those of neuroglucopenia and not autonomic symptoms (i.e., hypoglycemia unawareness)

Assessment of hypoglycemic episodes

Every hypoglycemic episode should be assessed carefully to determine its cause evaluating

- the insulin action profile (time of insulin administration, peak insulin action, and intensity of insulin action);
- recent food intake (timing and amount of carbohydrates eaten and peak BG effect of recent food);
- recent physical activity (timing, duration, and intensity).

To determine if changes in the treatment regimen are indicated, additional evaluation should include

- possible missed signs and symptoms of early hypoglycemia;
- the method of determination of meal insulin doses;
- if a BG measurement was performed at the time of the hypoglycemic symptoms and repeated after treatment

These steps in the evaluation are especially helpful for adolescents who may not be meticulous in determining or administering their insulin doses or attending to signs of hypoglycemia.

Review of the diabetes treatment regimen and goals of therapy

Insulin

After reviewing insulin action profiles, consideration should be given to using insulin regimens that have been shown to reduce the occurrence of hypoglycemia including continuous insulin infusion systems (pumps) and long-acting and rapid-acting analogues (70, 72, 106 64, 66, 100). The most consistent advantage of both rapid acting and long-acting analogue insulins has been a significant decrease in hypoglycemic episodes. Most studies have not been powered to show a difference in severe events but have documented a

difference in mild to moderate events and in nocturnal hypoglycemic events (99–101).

Food

Food intake (timing and content) should be adjusted so that glycemic peaks are more closely matched to insulin action peaks. Daytime and bedtime snacks may need to be added to the meal plan, especially in younger children or if intermediate-acting insulin is used. Adjusting the meal insulin dose for the BG value and the carbohydrate content of the meal may be helpful in decreasing the risk for postprandial hypoglycemia (103, 107).

Exercise

The timing, duration, and intensity of routine exercise should be reviewed so that food intake and insulin dose adjustments can effectively prevent marked reductions in BG levels. Pre- and postexercise snacks may be required. A carbohydrate snack of 15–30 g prior to exercise in well-controlled teenagers has been shown to decrease exercise-related hypoglycemia, as has suspension of pump basal rate during exercise, but if exercise occurs at the peak action of insulin, or is prolonged, additional carbohydrates may be required (59, 61, 62)(E). Additional recommendations for carbohydrate intake during exercise are given in a review by Riddell (4) and the ISPAD Exercise Guidelines.

BG goals

BG goals may need to be adjusted upward in patients with recurrent hypoglycemia and/or hypoglycemia unawareness (33). Suggested BG goals can be found in several sources (28, 108) and in the ISPAD Guidelines on Metabolic Monitoring.

BG monitoring

Frequent BG monitoring, with special attention to overnight (01:00–05:00 hours) levels, is one of the most important ways to detect mild hypoglycemia and prevent serious and severe episodes. Continuous glucose monitoring has revealed that prolonged hypoglycemia may occur during the night, and this technology appears to offer a significant advance in detection and avoidance of hypoglycemia (68, 109–111). Clinical trials of continuous glucose monitoring have given reason to believe that tighter glycemic control may not necessarily lead to increased risk of hypoglycemia when this technology is used (107). The ultimate goal remains the development of a noninvasive sensor or

an implantable sensor with long lifetime and capability to control automatic ‘close-loop’ insulin delivery system (112) (E).

Recommendations

- The aim of diabetes treatment should be to maintain BG levels above 3.9 mmol/L while striving to achieve the best possible glycemic control without the occurrence of severe hypoglycemia.
- Education about the risk factors for hypoglycemia should be given to patients and families to alert them as to times and situations when increased glucose monitoring is required and when treatment regimens need to be changed.
- Hypoglycemia should be prevented because its occurrence is frequently predictable, and it is often associated with significant psychosocial dysfunction; more importantly, it can in rare cases lead to permanent long-term sequelae and may be potentially life threatening.
- Particular attention should be given to training children, parents, schoolteachers, and other caregivers to recognize the early warning signs of hypoglycemia and treat low BG immediately and appropriately.
- Children and adolescents with diabetes should wear some form of identification or alert of their diabetes (E).
- An immediate source of glucose or sucrose must always be immediately available to young people with diabetes (A).
- Equipment for BG measurement must be available to all children with diabetes for immediate confirmation and safe management of hypoglycemia (B, E).
- Glucagon should be readily accessible to all parents and caregivers, especially when there is a high risk of severe hypoglycemia. Education on administration of glucagon is essential (E).
- Treatment of hypoglycemia should increase the BG approximately 3–4 mmol/L (55–70 mg/dL). This can be accomplished by giving glucose tablets/sugar lumps or a sweet drink (glucose/sucrose drinks, cola etc.), approximately 10 grams of glucose is needed for a 30 kg child and 15 grams for a 30 kg child (approximately 0.3g/kg).
- If sucrose or fructose are used, slightly higher amounts are required compared to pure glucose. Chocolate, whole milk and other foods containing fat will cause the sugar to be absorbed more slowly and should be avoided as the initial treatment of hypoglycemia.
- Following treatment BG should be retested in 10–15 min, if no response or inadequate response, repeat intake as above. Retest the BG in 20–30 min to confirm that target glucose has been maintained and not exceeded.

- BG monitoring should be performed prior to exercise, and extra carbohydrates should be eaten based on the BG level and the expected intensity and duration of the exercise.
- Patients and their parents should be trained to contact their diabetes care provider if hypoglycemia is documented without symptoms or if the symptoms are those of neuroglucopenia and not autonomic symptoms (i.e., hypoglycemia unawareness).
- Blood glucose goals may need to be adjusted upward in patients with recurrent hypoglycemia and/or hypoglycemia unawareness.
- If unexplained hypoglycemia is frequent, evaluation for unrecognized celiac and Addison's disease should be considered.

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