
بسم الله الرحمن الرحيم

# HYPOGLYCEMIA IN DIABETIC PATIENTS

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- ***Hypoglycemia is limiting factor in glycemic management of patients with diabetes, in whom risk of severe hypoglycemia increases as glycated hemoglobin (A1C) levels are reduced with intensive therapy.***

- ***Hypoglycemia can be a frightening, unpleasant, and potentially lethal complication of treatment of diabetes, and therefore, fear of hypoglycemia is understandable.***

- ***At its best, this concern should prompt diabetic patients to be aware of early autonomic symptoms and to ingest carbohydrate before symptoms progress.***

- ***In some cases, however, fear of hypoglycemia can become a major barrier to lowering blood glucose substantially.***

- ***Hypoglycemia is less common in patients with type 2 diabetes (T2DM) than in those with type 1 diabetes (T1DM), usually occur in those who are treated with insulins and insulin secretagouges (sulfonylureas, meglitinides).***

# SYMPTOMS



- ***Hypoglycemia causes neurogenic (autonomic) and neuroglycopenic symptoms.***

- ***Neurogenic symptoms include tremor, palpitations, and anxiety (catecholamine-mediated, adrenergic) and sweating, hunger, and paresthesias (acetylcholine-mediated, cholinergic).***

- ***Neuroglycopenic symptoms include dizziness, weakness, drowsiness, delirium, confusion, and, at lower plasma glucose concentrations, seizure and coma.***

- ***Although profound and prolonged hypoglycemia can cause brain death in the unobserved patient with DM, vast majority of episodes are reversed after glucose level is raised.***

- ***Rare fatal episodes are generally thought to be the result of ventricular arrhythmias.***

- ***Older adults and patients with long-term DM may have more neuroglycopenic than neurogenic manifestations of hypoglycemia.***

# Signs and symptoms of hypoglycemia



| Physical signs/ symptoms | Neuroglycopenic signs/symptoms | Behavioral/mood signs/symptoms |
|---|---|---|
| Pallor | Difficulty concentration | Emotional lability including anger |
| Diaphoresis | Hypothermia | Giddy |
| Tachycardia | Weakness | Tense |
| Blurred vision | Warmth | Anxiety |
| Elevated blood pressure | Hunger | Irritability |
| Palpitations | Fatigue | Feeling down/teary |
| Paresthesias | Motor impairment | |
| | Slurred speech | |
| | Seizures | |
| | Loss of consciousness | |

- ***Altuhogh lower limit of normal FPG (fasting plasma glucose) value is typically 70 mg/dL, and, according to the American Diabetes Association (ADA) guidelines, hypoglycemia in diabetic patients is defined as glucose levels ≤70 mg/dL, glycemic thresholds for developing hypoglycemic symptoms shift to higher plasma glucose concentrations in patients with poorly-controlled DM, and shift to lower plasma glucose concentrations in patients with repeated episodes of hypoglycemia, such as may be caused by intensive therapy of DM.***

- ***In 2017, The International Hypoglycaemia Study Group (IHSG) proposed a glucose level of <54 mg/dL as sufficiently low to indicate serious, clinically important hypoglycemia.***

- ***This value identifies an unequivocally low glucose level, one that occurs rarely, if at all, in nondiabetic individuals under physiologic conditions and one that should be avoided because of immediate and long-term consequences to patient.***

- *In diabetic patients, symptoms may be absent because of hypoglycemia unawareness (impaired hypoglycaemia awareness) which is thought to be the result of reduced sympathoadrenal (predominantly sympathetic neural) responses to hypoglycemia caused by:*

  *1) recent antecedent hypoglycemia,*

  *2) prior exercise, or*

  *3) sleep.*

- ***Hypoglycemia is the result of interplay of:***

  ***1) therapeutic hyperinsulinemia, and***

  ***2) compromised physiologic defense (defective glucose counterregulation) and impaired behavioural defense (intake of carbohydrate) against falling plasma glucose.***

- ***The American Diabetes Association (ADA) guidelines define hypoglycemia in patients with DM as all episodes of an abnormally low plasma glucose (with or without symptoms) that expose the individual to harm.***

- ***The ADA did not identify a specific glucose cut-off level that defines hypoglycemia, as glycemic thresholds that induce symptoms (and counterregulatory responses) vary within (intraindividual) and between (interindividual) individuals.***

# Classification of Hypoglycemia



- ***The ADA and the Endocrine Society Workgroups classify the severity of hypoglycemia in DM is as follows:***

- ***Severe hypoglycemia:***

***An event requiring assistance of another person to actively administer carbohydrate, glucagon, or other resuscitative actions.***

- ***Mild hypoglycaemia:***

***An event not requiring assistance of another person to actively administer carbohydrate, glucagon, or other resuscitative actions.***

**Table 6.4—Classification of hypoglycemia**

| | Glycemic criteria/description |
|---|---|
| Level 1 | Glucose <70 mg/dL (3.9 mmol/L) and ≥54 mg/dL (3.0 mmol/L) |
| Level 2 | Glucose <54 mg/dL (3.0 mmol/L) |
| Level 3 | A severe event characterized by altered mental and/or physical status requiring assistance for treatment of hypoglycemia |

Reprinted from Agiostratidou et al. (72).

# Pseudohypoglycemia



- *An event during which person with DM reports typical symptoms of hypoglycemia but has a measured glucose level >70 mg/dL.*

- *Patients with chronically poorly-controlled DM can experience symptoms of hypoglycemia as plasma glucose fall into physiologic/normal (not hypoglycemic) range.*

- ***Pseudohypoglycemia is also used to describe artifactually low plasma or serum glucose due to continued metabolism of glucose after sample is drawn as can occur when:***

  ***1) sample tube does not include an inhibitor of glycolysis, or***

  ***2) separation of plasma or serum is delayed.***

# RISK FACTORS



- *Hypoglycemia-associated autonomic failure (HAAF)*
- *Longer duration of diabetes*
- *Older age*
- *Lower levels of glycemia, when achieved with medications*
- *Erratic timing of meals, including missed meals and low carbohydrate content of meals (<100 g/day)*
- *History of recent severe hypoglycaemia (unawareness)*
- *Exercise (increased insulin sensitivity and glucose utilization)*
- *Alcohol ingestion (suppression of gluconeogenesis)*
- *Chronic kidney disease (decreased renal insulin clearance)*
- *Malnutrition with glycogen depletion*

# Frequency of Hypoglycemia



- Patients with T1DM report an average of up to 3 episodes of severe hypoglycemia (episodes requiring assistance of another person) per year.

- Studies using continuous glucose monitoring (CGM) show much more frequent episodes of clinically important hypoglycemia (<54 mg/dL), ranging from every 2-3 days to every 6 days.

- ***Hypoglycemia is substantially less frequent in T2DM, although patients with T2DM treated with insulins, sulfonylureas or glinides are generally at higher risk than those treated with diet or other medications.***

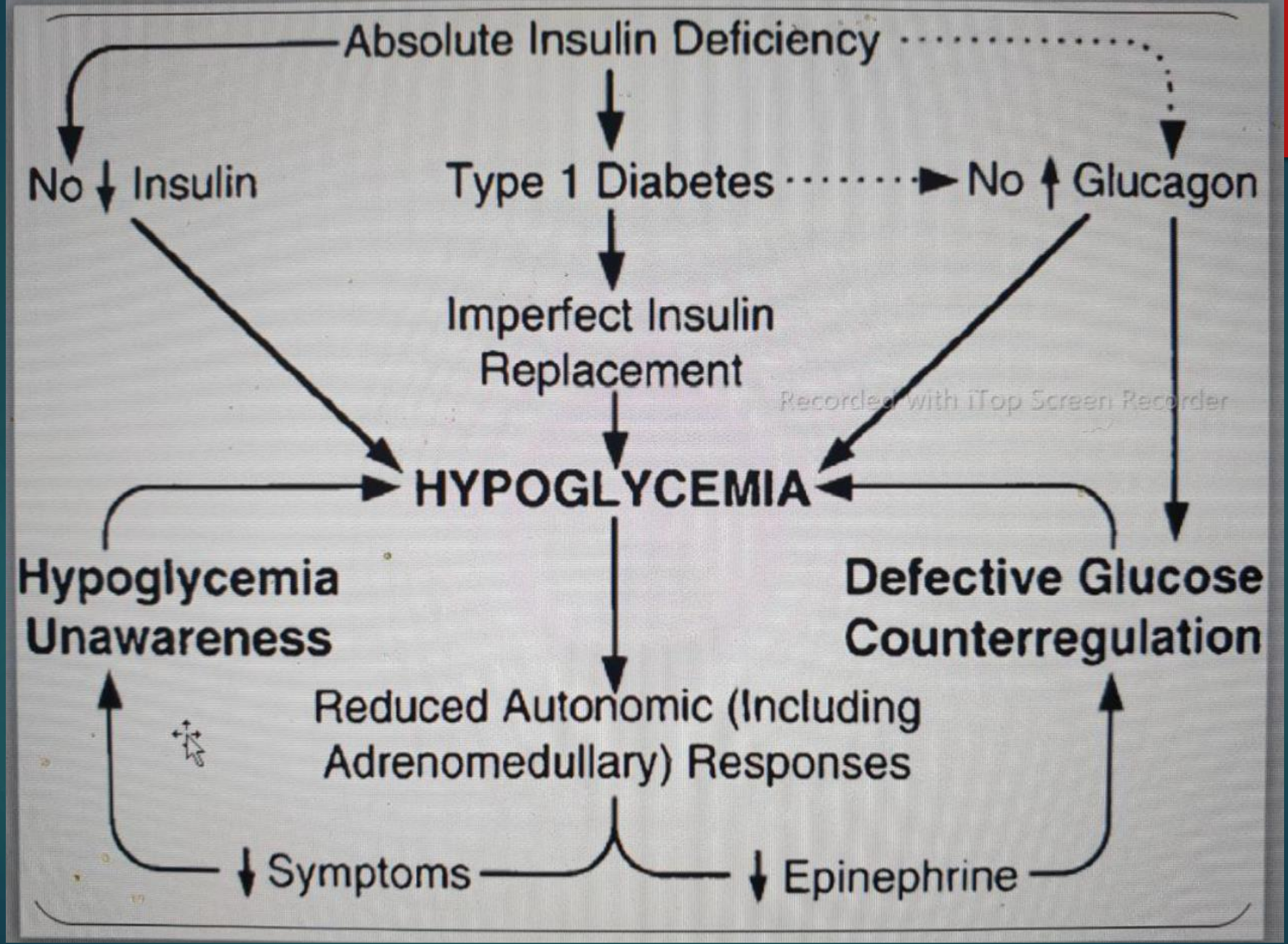
- ***Among commonly used insulin secretagogues (sulfonylureas, glinides), hypoglycemia is most often reported in patients taking long-acting drugs, such as glyburide (glibenclamide), compared with shorter-acting glipizide, glimepiride and gliclazide as well as glinides.***

- *In contrast to insulin and insulin secretagogues, agents that do not cause hyperinsulinemia, such as metformin, thiazolidinediones (TZDs), α-glucosidase inhibitors, glucagon-like peptide-1 (GLP-1) receptor agonists, dipeptidyl peptidase-4 (DPP-4) inhibitors (gliptins), and sodium-glucose cotransporter 2 (SGLT2) inhibitors (gliflozins) do not usually cause hypoglycaemia when they are used as monotherapy, however, they increase risk of hypoglycemia if used in combination with insulin or insulin secretagogues.*

- ***Reducing risk of hypoglycemia** while maintaining or improving glycemic control involves:*
- *patient education,*
- *frequent self-monitoring of blood glucose (**SMBG**):*

  *1) usually with fingerstick measurements (**glucometer**) in both T1DM and T2DM, or*

  *2) with continuous glucose monitoring (**CGM**) in **T1DM**,*
- *individualized glycemic goals (**targeted A1C**),*
- *flexible and rational insulin (and other drug) regimens, and*
- *ongoing professional guidance and support.*


Absolute Insulin Deficiency
No Insulin
Type 1 Diabetes
No Glucagon
Imperfect Insulin Replacement
HYPOGLYCEMIA
Hypoglycemia Unawareness
Defective Glucose Counterregulation
Reduced Autonomic (Including Adrenomedullary) Responses
Symptoms
Epinephrine

# Physiologic Response/Defense to Hypoglycemia in Normal Subjects and in Diabetic Patients



- ***Brain relies almost exclusively on glucose as a fuel**, but it cannot synthesize or store much of it (as **glycogen**), as a result, adequate uptake of glucose from plasma is essential for **normal brain function and survival**.*

- *Given survival value of maintenance of plasma glucose level, it is not surprising that **very effective physiologic and behavioral mechanisms** that **normally prevent or rapidly correct hypoglycemia** have evolved.*

- ***Hypoglycemia is a relatively uncommon clinical event except in patients who use drugs that lower glucose levels (insulins, sulfonylureas, or glinides).***

- ***In addition to being at increased risk for hypoglycemia, insulin-treated diabetic patients often have impaired neurohumoral responses to and few early (neurogenic) symptoms of hypoglycemia.***

- *In normal subjects, in fasting state (when glucose cannot be obtained from intestinal absorption of food) with falling glucose level, defensive mechanisms prevent or rapidly correct falling plasma glucose concentrations.*

- *Neurohormonal defense begins well before the onset of symptoms of hypoglycemia.*

# Counterregulatory Hormones



- The first defense is decrease in insulin secretion as plasma glucose decline within physiologic range, starting at plasma glucose threshold of 80-85 mg/dL.

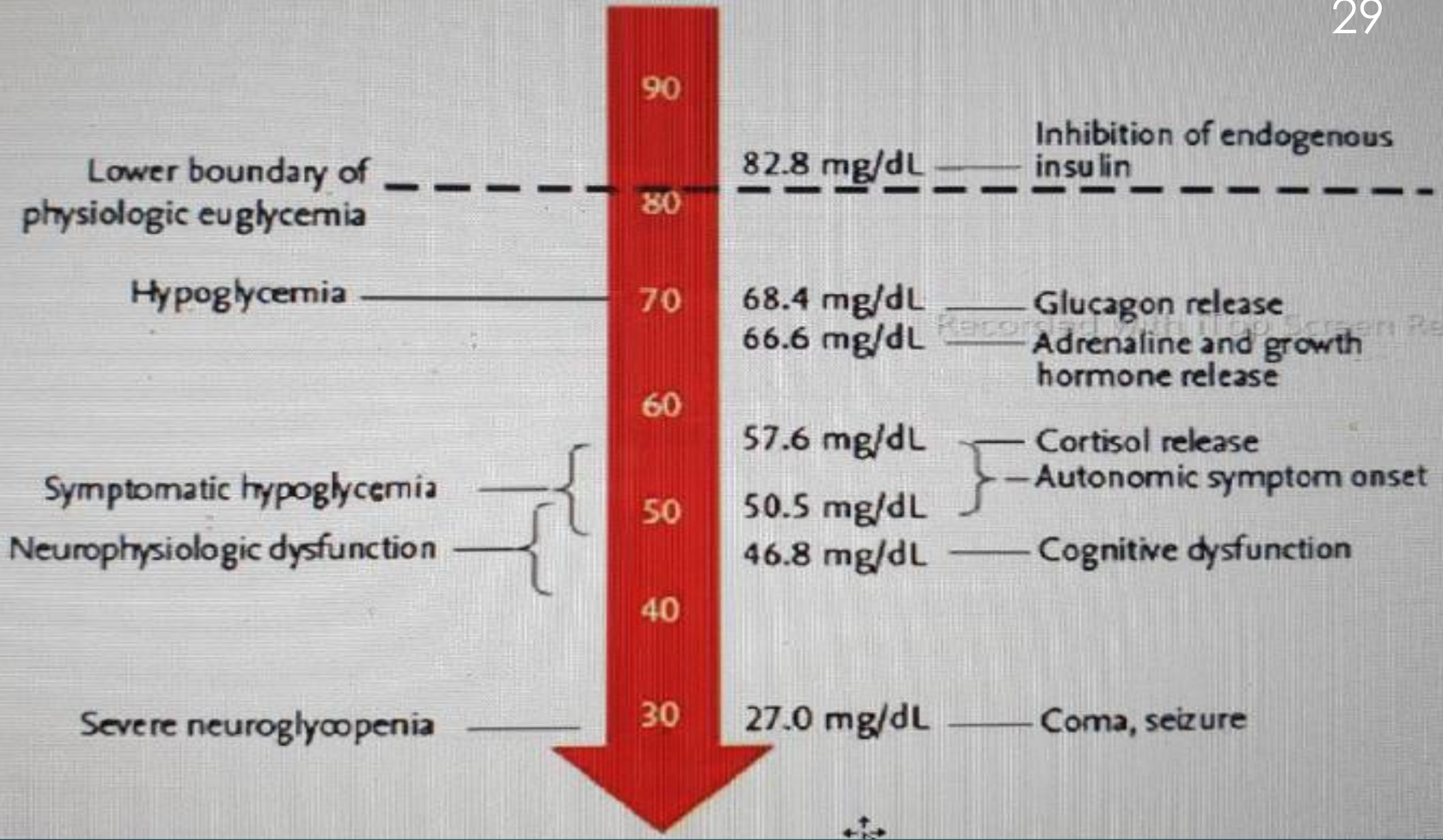
- The second defense is increase in glucagon secretion at glucose levels 65-70 mg/dL.
- Glucagon acts on liver, increasing glucose production by stimulating both glycogenolysis and gluconeogenesis from alanine, other amino acids, and glycerol.

- ***The third defense is increase in epinephrine secretion at glucose levels 65-70 mg/dL, acting mainly via β2 adrenergic receptors.***
- ***Epinephrine has similar hepatic effects as glucagon, but it also:***
  - ***1) increases delivery of gluconeogenic precursors from peripheral tissues (amino acids from skeletal muscle, glycerol from adipose tissue),***
  - ***2) inhibits glucose utilization by peripheral tissues, and***
  - ***3) via α-2-receptors, inhibits insulin secretion.***

- ***The last defense, cortisol and growth hormone (GH) secretion, contribute only if hypoglycemia persists for several hours.***

- ***Cortisol and GH limit peripheral glucose utilization and enhance hepatic glucose production.***

# Glycemic thresholds for secretion of counter-regulatory hormones



# Behavioral Defenses



- ***Initial neurogenic symptoms*** *of sweating, anxiety, palpitations, hunger, and tremor occur as plasma glucose falls to* ***≤55*** *mg/dL.*

- *Neurogenic symptoms trigger important* ***behavioral defense****, ie,* ***food ingestion****.*

# Neuroglycopenic Symptoms



- Hypoglycemia can also cause cognitive dysfunction, which occurs at plasma glucose of <50 mg/dL, which can impair behavioral defence (carbohydrate intake).

- Neuroglycopenic symptoms such as obtundation, seizures, and coma, occur with progressive hypoglycaemia, usually with plasma glucose of <30-40 mg/dL.

- Profound and prolonged hypoglycemia can cause brain death.

# *RESPONSE TO HYPOGLYCEMIA IN DIABETES*

# Impairment of Behavioral and Counterregulatory Responses



- ***Hypoglycemia in insulin/insulin secretagogue-treated patients is typically the result of interplay of:***

  ***1) therapeutic insulin excess, and***

  ***2) compromised physiologic (counterregulatory hormone release) and behavioral defences (food eating) against falling plasma glucose concentrations.***

- ***Protective/defensive response to hypoglycaemia (especially glucagon release) is impaired in many patients with long-standing DM.***

# Insulin:

- The first defense (ability to suppress insulin release) cannot occur in patients with:

  1) absolute β-cell failure (patients with T1DM and advanced T2DM who have negligible endogenous insulin), and

  2) hyperinsulinemia due to use of exogenous insulin/insulin secretaguges.

- The consequence is suppression of hepatic glucose production.

# Glucagon:

- Glucagon response to hypoglycaemia is lost in parallel with loss of insulin in T1DM and more slowly in T2DM.

- This may be the result of loss of hypoglycemia-induced decline in intra-islet insulin that signals increased glucagon secretion during hypoglycaemia.

- Such patients still have some glucagon responses to other stimuli such as amino acids.

# ***Epinephrine:***

- ***In the setting of absent insulin and glucagon responses, patients are dependent upon epinephrine to protect against hypoglycemia.***

- ***Epinephrine response to hypoglycaemia may also become attenuated in many diabetic patients, at least in part, because of recent antecedent hypoglycaemia.***

- ***An attenuated epinephrine response causes defective glucose counterregulation, which is associated with a ≥ 25-fold increased risk of severe hypoglycaemia.***

- ***Attenuated sympathoadrenal (largely sympathetic neural) response also causes "hypoglycemia unawareness."***

# Hypoglycemia-associated Autonomic Failure (HAAF)



- The concept of hypoglycemia-associated autonomic failure (HAAF) in T1DM and long-standing insulin-deficient T2DM posits that recent antecedent hypoglycemia causes both:

  1) defective glucose counterregulation, and

  2) hypoglycemia unawareness, and thus a "vicious cycle" of recurrent hypoglycaemia.

- ***HAAF** does so by **shifting** glycemic threshold for **sympathoadrenal response** to **subsequent hypoglycaemia** (symptomatic or asymptomatic) to a **lower plasma glucose concentration**.*

- ***This shift causes defective glucose counterregulation** by **reducing sympathoadrenal responses** in the setting of **absent insulin and glucagon responses** to hypoglycemia.*

- ***It also causes "hypoglycemia unawareness" by reducing neurogenic symptom responses.***

- ***"Sleep" and "prior exercise" can cause a similar phenomenon.***

- ***The precise mechanism(s) of key feature of HAAF, attenuated sympathoadrenal response to falling plasma glucose concentrations, is unknown.***

- ***One hypothesis is that hypoglycemia-induced alterations in hypothalamic functions, or even a cerebral network, reduce sympathoadrenal response to subsequent hypoglycaemia.***

- ***Another hypothesis is that an increase in cortisol (or some other factor) during hypoglycemia causes a reduced sympathoadrenal response to subsequent hypoglycemia.***

- ***HAAF is a "functional disorder" distinct from classical diabetic autonomic neuropathy, the result of nerve fiber loss.***

- ***Nonetheless, sympathoadrenal responses to hypoglycemia are reduced further in patients with diabetic autonomic neuropathy.***

- ***If there is a history of hypoglycemia awareness, a 2- to 3-week period of avoidance of hypoglycemia is advisable since that often restores hypoglycaemia awareness.***

- *Although HAAF was originally described in T1DM, it also applies to advanced T2DM patients treated with intensive (basal/bolus) insulin regimens.*

- *Insulin secretion decreases progressively over time in T2DM; as patients with T2DM develop absolute insulin deficiency and are treated with exogenous insulin, insulin secretion does not decrease and glucagon secretion does not increase when plasma glucose fall.*
- *Furthermore, antecedent hypoglycemia reduces sympatho-adrenal responses to subsequent falling glucose levels in T2DM.*

- *Compared with T1DM, features of HAAF develop later in natural history of T2DM (insulin-deficient phase of T2DM).*

- *This different temporal pattern of pathophysiology of glucose counterregulation likely explains why iatrogenic hypoglycemia is relatively uncommon early in the course of T2DM (even during treatment with insulin), when glucoregulatory defenses are intact, but occurs more frequently as patients approach insulin-deficient end of spectrum of T2DM, when defenses become compromised.*

- *If there is a history of hypoglycemia awareness, a 2- to 3-week period of avoidance of hypoglycemia is advisable since that often restores hypoglycaemia awareness.*

# Nocturnal Hypoglycemia



- ***Most episodes of severe hypoglycemia occur during sleep because overnight is typically the longest interprandial period, the time between self-monitoring of blood glucose (SMBG), and the time of maximal sensitivity to insulin.***

- ***Furthermore, sympathoadrenal responses to hypoglycemia are reduced during sleep, and therefore, patients are less likely to be awakened by symptoms of hypoglycaemia (presents as nighttime dreams and morning headache).***

- ***Unfortunately, nocturnal hypoglycemia is common, even with use of CSII (continuous subcutaneous insulin infusion) or basal-bolus regimen with insulin analogues.***

- ***Even asymptomatic nocturnal hypoglycemia impairs defenses against subsequent hypoglycemia.***

# Exercise



- Exercise increases glucose utilization by muscle and, therefore, can cause hypoglycemia in patients with insulin-deficient DM who have near-normal or elevated glucose levels at the start of exercise.

- In addition, exercise can cause HAAF hours later.

- ***Exercise-induced hypoglycemia can occur during, shortly after, or many hours after exercise (typically 6-15 hours, but up to 24 hours after strenuous exercise), and therefore, patients should remain vigilant for its occurrence, including frequent SMBG or CGM.***

- ***Exercise-induced Hypoglycemia can be prevented by frequent SMBG and reduced insulin doses, carbohydrate ingestion (eg, 1 g/kg/h) or both, prior to and during exercise.***

- ***In addition, exercise, can shift glycemic threshold for sympathoadrenal response to subsequent hypoglycemia to a lower plasma glucose hours later.***

- ***This shift causes defective glucose counterregulation by reducing epinephrine responses in the setting of absent insulin and glucagon responses.***

- ***Exercise also causes hypoglycemia awareness by reducing symptom responses.***

# Recommended carbohydrate intake with exercise

| Type of activity/duration | CHO intake | Insulin adjustments[a] |
|---|---|---|
| Low-intensity, short-duration activity (e.g. 30 min of walking) | 15 gm CHO if longer than 1–2 h after meal | Usually not needed |
| Moderate-intensity, intermediate-duration activity (e.g. competitive sports, running) for 30–60 min) | 15 gm CHO with 7–8 gm protein before exercise | Reduction in mealtime insulin pre-exercise by ≥ 30% and based on glucose readings |
| High-intensity, relatively long-duration activity (e.g. hiking for several hours, cross-country skiing for ≥ 60 min) | Snacks of 15–20 gm CHO with 7–8 gm protein every 60 min | Reduction in mealtime insulin by 50–100% and based on glucose readings |
| Regular water intake for any activity | | |

# Iatrogenic Hypoglycemia



- Iatrogenic hypoglycemia occurs in patients with both T1DM and T2DM treated with insulins, sulfonylureas, or glinides.

- Patients treated intensively to lower A1C levels in order to reduce microvascular complications have 2- to 3-fold higher rates of severe hypoglycaemia.

# Severe Intractable Hypoglycemia



- Patients with severe intractable hypoglycemia may be candidates for pancreas or islet transplantation.

- Pancreas/islet transplantation can result in:

  1) independence from exogenous insulin therapy,

  2) improvements in glucose metabolism and A1C values,

  3) improvement in counterregulatory responses of glucagon and epinephrine to hypoglycemia.

  - Islet transplantation can be performed currently only within the context of a controlled research study.

# Prevention of Hypoglycemia



- ***Prevention of hypoglycemia involves assessing for risk factors and tailoring treatment regimens to reduce risk.***
- ***At each visit, provider should assess about:***
  - ***Measured low glucose levels (patient's SMBG)***
  - ***Episodes requiring assistance of another person (severe hypoglycemia)***
  - ***Episodes of symptoms consistent with hypoglycaemia***
  - ***Patient education***
  - ***Frequent SMBG, usually with fingerstick measurements or with CGM (CGM primarily in T1DM or high-risk T2DM patients)***
  - ***Individualized glycemic goals***
  - ***Flexible and rational insulin (and other drug) regimens***
  - ***Ongoing professional guidance and support***

# Patient Education



- ***Patient education focused on implementation of flexible insulin therapy can reduce incidence of severe hypoglycemia.***
- ***Patients should be taught to adjust their drugs, meal plans, and exercise based on glycemic patterns.***
- ***Clinicians should review how to treat hypoglycemia with oral carbohydrate or glucagon by the patient.***
- ***Close associates, such as a spouse or a partner, should be taught to recognize severe hypoglycemia and treat it with glucagon.***

- ***Regular SMBG** is **critical** to glycemic management of **T1DM** as well as **intensively treated T2DM** (eg. basal/bolus insulin regimen).*

- ***CGM** is **usually** used in **T1DM**, but **some T2DM patients** may benefit as well.*

- *Use of CGM or SMBG **before** and **1-2 hours after each meal**, at **bedtime**, in the **middle of night**, and **before**, **during** and **after exercise** can help identify **glycemic patterns** and **hypoglycaemia**.*

- ***For patients with DM who may have asymptomatic hypoglycemia due to repeated episodes of hypoglycemia and/or hypoglycemia unawareness, intermittent use of CGM may be valuable for detection and management of hypoglycemia.***

# Treatment of Hypoglycemia



- ***The goal of treatment of hypoglycemia is to raise plasma glucose to normal by providing:***

  ***1) oral carbohydrate or IV glucose, or***

  ***2) in cases of severe hypoglycemia outside of a medical center, by stimulating endogenous glucose production by administering glucagon.***

- *In order to treat early symptoms of hypoglycemia, patients should be certain that fast-acting carbohydrate (such as glucose tablets or sweetened fruit juice) is available at all times (treatment with glucose tablets is more consistently effective).*

- *Patients with T1DM should have a glucagon kit, which should be checked regularly and replaced when it is beyond its expiration date.*

- ***For an asymptomatic patient, suggestion is defensive actions when SMBG reveals glucose ≤70 mg/dL.***

- ***Defensive options include:***

***1) repeating measurement within 15-60 minutes (depending on the setting),***

***2) avoiding critical tasks such as driving, and***

***3) adjusting treatment regimen.***

- ***Patients with symptomatic hypoglycemia should ingest 15-20 grams of fast-acting carbohydrate, which is usually sufficient to raise glucose into a safe range.***

- ***Patients should be instructed to retest blood glucose after 15 minutes and if glucose remains ≤70 mg/dL, repeat treatment may be necessary.***
- ***This can be followed by long-acting carbohydrate (a meal or a snack) to prevent recurrent symptoms.***

- *In patients taking insulin or insulin secretagogue in combination with α-glucosidase inhibitor (acarbose, miglitol, voglibose), only pure glucose (eg, glucose tablets) should be used to treat symptomatic hypoglycemia.*

- *In these patients, other forms of carbohydrates, such as table sugar (sucrose), will be less effective in raising blood sugar as α-glucosidase inhibitors slow digestion of disaccharides.*

- ***Patients already in hospital can usually be treated quickly by giving 25 g of 50% glucose (dextrose) intravenously.***

- ***For treatment of hypoglycemia in patients with impaired consciousness and no IV access, suggestion is immediate glucagon administration, rather than waiting to establish IV access.***

- ***Administration of glucagon (SC, IM, or nasal) will usually lead to recovery of consciousness within ~ 15 minutes, although it may be followed by marked nausea or even vomiting.***

- ***Glucagon dose should be followed promptly by intake of oral concentrated carbohydrates, immediately upon awakening from confused state and before development of nausea.***

- ***Glucagon therapy requires that close relative is able to recognize hypoglycemia and administer glucagon.***

- *There are no efficacy or safety data to guide management of severe hypoglycemia in patients with impaired consciousness and no immediate access to glucagon or IV dextrose.*

- *In the absence of other options for patients with severe hypoglycemia who are unconcious, some experts suggest that while awaiting emergency personnel, family members squeeze a glucose gel (eg, Insta-Glucose) or cake frosting in space between teeth and buccal mucosa, keeping patient's head tilted slightly to the side to prevent aspiration of these materials.*

- ***If glucose gel or cake frosting is unavailable, some advocate sprinkling table sugar under tongue as table sugar has been reported to raise plasma glucose to some extent in ill children with malaria.***

- ***Other experts would not administer buccal or sublingual preparations or foods, given lack of supporting evidence showing that buccal absorption of glucose occurs in humans and risk of aspiration.***

# Monitoring of Treatment



- ***Glycemic response to IV glucose and glucagon is transient, therefore, effective initial treatment of hypoglycemia often needs to be followed by continuous infusion of glucose (or food if patient is able to eat, often not possible by nausea often induced by glucagon).***

- ***Further treatment vary depending on the class of agent causing hypoglycemia.***

- ***Sulfonylurea-induced hypoglycemia may be particularly long lasting or recurrent since sulfonylurea is able to continue insulin secretion after initial carbohydrate treatment.***

- *There is little experience in treating "overdoses" of long-acting insulin analogues, such as degludec or glargine 300; durable effects of these insulins suggest that observation and therapy may need to be prolonged, compared with hypoglycemia associated with conventional shorter-acting insulins (such as NPH insulin).*

# Treatment of hypoglycaemia



| Duration of hypoglycaemia | Administrator | Treatment |
|---|---|---|
| minutes | Patient | Oral carbohydrate (>20 g) |
| hours | Caregiver | • Oral carbohydrate (liquid/solid)<br>• 1 mg glucagon[a] |
| | Primary healthcare setting | • 1 mg glucagon intramuscular or intravenous[a]<br>• 25 g dextrose intravenous |
| | Hospital setting | • 25 g dextrose intravenous<br>• 1 mg glucagon intravenous[a] |

# Treatment



## Rule of 15

- Take 15 g of glucose
- Wait 15 minutes
- If still low treat with another 15g glucose

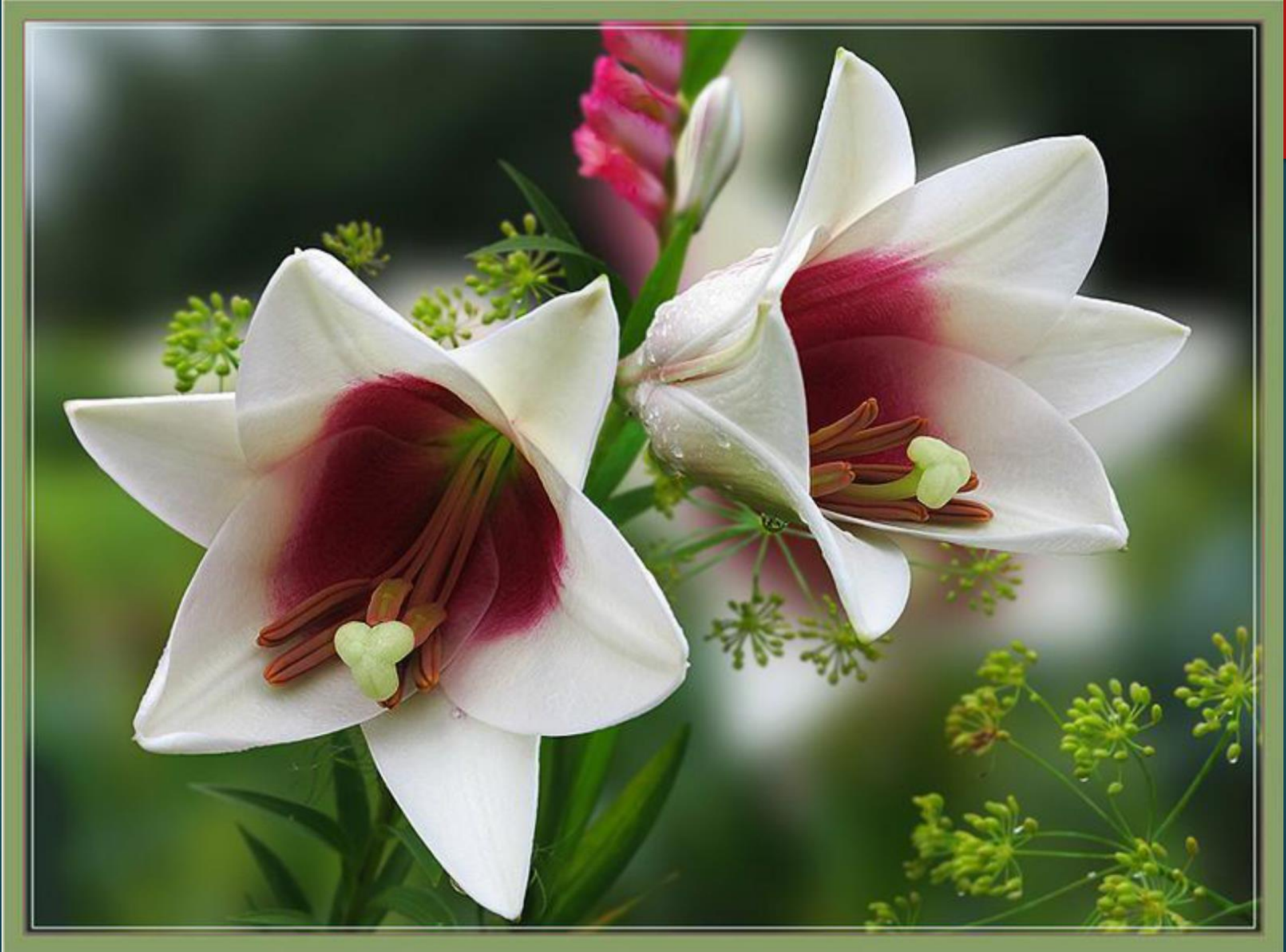
## Treatment

- Tablespoons sugar or honey
- Glucose tablets or gel
- 1/2 cup fruit juice
- 1/2cup soft drink



## Inappropriate treatment

- Milk
- Ice cream
- Chocolate



***Thank you for your attention and have a nice day***