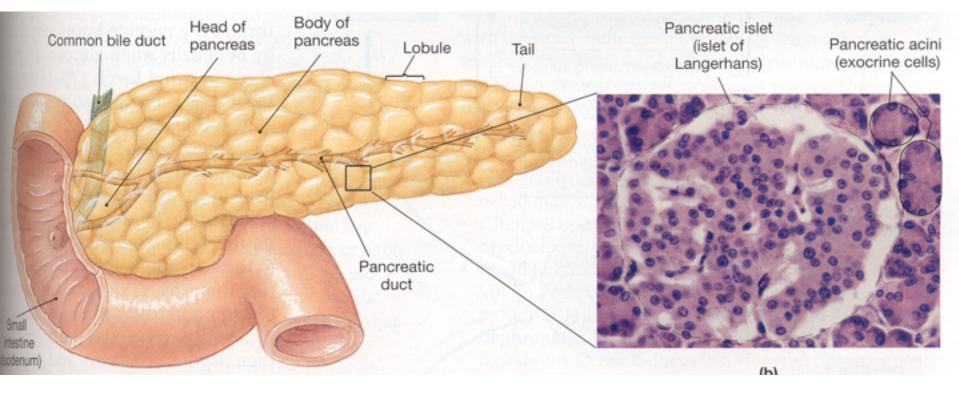


Diabetes Mellitus

- Types of diabetes
 - TI: insulin-dependent (IDDM)
 - Require insulin
 - TII: non-insulin-dependent (NIDDM)
 - Require oral medications or insulin
- A&P of diabetes
 - Pancreas
 - Insulin
 - Glucose

<u>Pancreas</u>



- α cells: produce glucagon, ↑'s blood glucose
- β cells: produce insulin, ↓'s blood glucose

Glucose

- Simple sugar
- Body's main energy source
- Brain cells extremely sensitive to a lack of glucose
 - Respond immediately to inadequate levels
 - Normal = 80-120 mg/dL

Insulin

- Secreted by the pancreas when BGL is elevated
- Facilitates the transport of glucose across cellular membranes
 - Excess glucose stored in the liver (glycogen)
- \(\)'s blood glucose by \(\) ing rate of glucose uptake in cells
 - Glucose can be stored or used immediately in cells
 - Brain does not require insulin!

Glucagon

- Hormone
 - Released when insulin levels low
- †'s blood glucose by †ing rate of glycogen breakdown in liver
 - Glycogenolysis
 - Glycogen is stored energy, ready to be used when needed!
- †'s breakdown of fats in adipose tissue

Epinephrine

- Secreted when BGLs are low
- Inhibits insulin secretion
- Promotes glycogenolysis & gluconeogenesis
- Epi is responsible for many of the S/S we see in hypoglycemia

1 1

Normal

glucose

utilization

Food is eaten.

Digestion begins in the stomach.

Food is broken down into glucose in the small intestine.

Glucose enters the bloodstream. Insulin is released by pancreas.

Glucose enters body cells with aid of insulin.

Normal Glucose Regulation



BGL 120 to 140 mg/dL

Liver releases glycogen Increased fat breakdown Noncarbohydrate glucose production

Insulin secreted

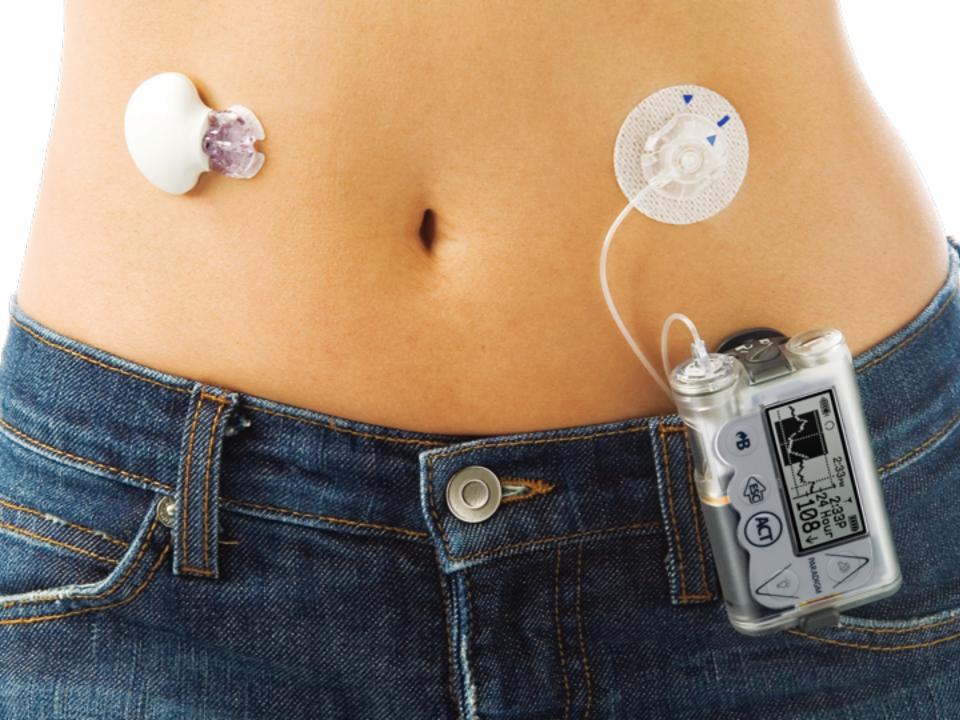
Glucagon secreted

Cells uptake glucose Liver creates glycogen

Type 1 DM

- AKA IDDM: "Juvenile-onset diabetes"
- Very little or no insulin production
 - Autoimmune response destroys β-cells
- Must take oral antihyperglycemic meds or insulin





Type 2 DM

- AKA NIDDM: "Adult onset diabetes"
- Moderate insulin production combined with \u00ed response to insulin at the cellular membrane
- Can be controlled with diet and exercise
- Pharmacological control
 - Oral antihyperglycemics



Various oral antihyperglycemics will:

- † insulin release
- ↓ glucose release from liver
- \ uptake of carbohydrate in gut



Problems related to diabetes:

- Peripheral neuropathy
- Decreased peripheral circulation

Can lead to soft tissue injuries and infection.





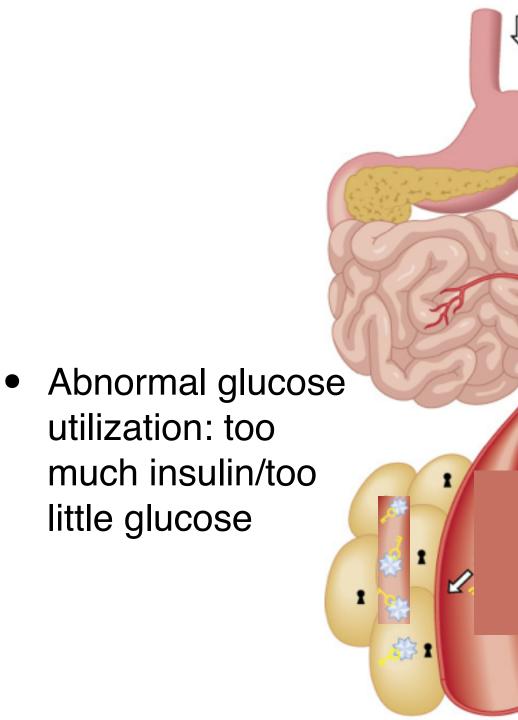






Causes of Hypoglycemia

- Diabetic took too much insulin or oral antihyperglycemic medication
- Diabetic has not eaten enough
 - or, has vomited!
- Diabetic has overexerted themselves
- Diabetic is stressed, has infection



utilization: too

little glucose

Food is eaten.

Digestion begins in the stomach.

Food is broken down into glucose in the small intestine.

Glucose enters the bloodstream. Insulin is released by pancreas.

Glucose enters body cells with aid of insulin.

S/S: Hypoglycemia

- Weakness, dizziness
- Disorientation, AMS, LOC
- Cool, pale, diaphoretic skin
- Seizures, stroke-like symptoms



Stroke Mimicker

- Hypoglycemia can present with S/S of CVA
- Pt is not having a stroke until blood glucose abnormalities are corrected!



Tx: Hypoglycemia

- Open the airway
- Assure adequate ventilation
- Administer oxygen
- ALS
- Assess BGL, if able
- Oral glucose





Pt Refusal?

- Is the cause explainable and does not include illness or OD?
- Can the pt eat a meal?
- Will there be someone with pt?
- Sign refusal?

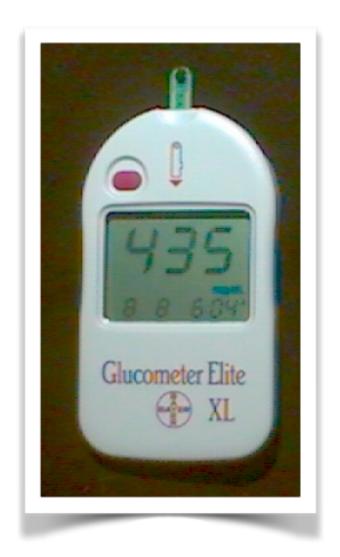






Hyperglycemia

- Two conditions that can arise from hyperglycemia:
 - DKA
 - HHNS



Diabetic Ketoacidosis

- Also called "diabetic coma", DKA
- Insulin deficiency with ↑ glucagon activity
- Significant amount of glucose in blood
 - Osmotic diuresis = dehydration, 'lyte imbalance
- Fats and proteins broken down for energy
 - Ketosis, then DKA occurs
- Death can occur

Causes of Hyperglycemia

- Patient with new onset diabetes
- Diabetic has not taken diabetic medications
 - oral meds, insulin
- Diabetic has overeaten
- Diabetic is stressed, has infection

DKA: S/S

- Onset over 12-24 hrs
- Initial phase:
 - Polyuria, polyphagia, polydipsia
 - Warm, dry skin, tachycardia, poss | BP, weakness, N/V, abd pn, hyperglycemia (>350 mg/dL)
- Late phase:
 - Kussmaul's respirations, fruity odor on breath, AMS, coma

Hyperglycemic Hyperosmolar Nonketotic Syndrome

- Serious complication associated with TII DM
 - Often precipitated by stress (illness, trauma, psych)
 - Stress results in decreased insulin levels
- HHNS present when 2 conditions occur
 - Sustained hyperglycemia- can be ↑ 1000 mg/dl
 - Osmotic diuresis = dehydration, 'lyte imbalance
- Insulin activity sufficient to prevent DKA
- 40 –70 % mortality

HHNS: S/S

- Onset over several days
- Polyuria, polydipsia, polyphagia
- Tachycardia, orthostatic hypotension
- AMS big indicator
- Hyperglycemia (greater than 600 mg/dL)
- Remember, will not have Kussmaul's respirations!

Tx: Hyperglycemia

- Tx for DKA & HHNS is same
- ABC's... duh
- 100% O₂ via appropriate device
- ALS
- What if you don't have a BGL? Is administering glucose bad?



Glucose

- Indications:
 - AMS with/without known Hx DM
- Contraindications:
 - Unresponsive, unable to swallow
 - Known Hx DM, has not taken insulin for days