1. The Command Center: HPT Axis

The **Hypothalamic-Pituitary-Thyroid (HPT) Axis** is the feedback loop that maintains metabolic homeostasis.

- **TRH (Thyrotropin-Releasing Hormone):** Released by the Hypothalamus.
- **TSH (Thyroid-Stimulating Hormone):** Released by the Anterior Pituitary; tells the thyroid to get to work.
- **T3 & T4:** The actual hormones produced by the thyroid.
- **Negative Feedback:** When T3/T4 levels are high enough, they tell the brain to stop producing TRH and TSH.

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2. Hypothyroidism: The Slow Down

Hypothyroidism occurs when the thyroid is underactive, leading to a "slowed" metabolism.

- **Iodine Deficiency:** Iodine is a mandatory "ingredient" for making T3 and T4. Without it, the thyroid enlarges (Goiter) trying to compensate.
- **Hashimoto's Thyroiditis:** An **autoimmune** disorder where the body attacks its own thyroid. It is the most common cause of hypothyroidism in iodine-sufficient areas.
- **Cretinism (Congenital Hypothyroidism):** Severe hypothyroidism in infants. If untreated, it leads to stunted physical and mental growth.
- **Myxedema Coma:** The "Final Boss" of hypothyroidism. A life-threatening medical emergency characterized by extreme lethargy, hypothermia, and slowing of multiple organ systems.

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3. Hyperthyroidism: The Speed Up

Hyperthyroidism occurs when the thyroid is overactive, leading to an "accelerated" metabolism.

- **Hyperthyroidism/Thyroiditis:** Generally involves weight loss, rapid heart rate, heat intolerance, and anxiety. "Thyroiditis" specifically refers to inflammation that can leak excess hormone into the blood.
- **Thyroid Storm:** The "Final Boss" of hyperthyroidism. A life-threatening state of extreme hypermetabolism. Symptoms include high fever (hyperpyrexia), tachycardia (rapid heart rate), and agitation/delirium.

Summary Comparison Table

Feature	Hypothyroidism	Hyperthyroidism
Metabolic Rate	Decreased (Weight gain)	Increased (Weight loss)
Temperature	Cold intolerance	Heat intolerance
Energy	Fatigue/Lethargy	Anxiety/Insomnia
Emergency State	**Myxedema Coma**	**Thyroid Storm**
Key Cause	Hashimoto's / Iodine deficiency	Graves' Disease / Thyroiditis

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Quick Recall "Key Meanings"

- **HPT Axis:** The "Thermostat" (Brain → Thyroid).
- **Iodine:** The "Fuel" (No fuel = No hormone).
- **Hashimoto's:** "Self-Attack" (Autoimmune Hypo).
- **Cretinism:** "Developmental Delay" (Infant Hypo).
- **Myxedema Coma:** "The Deep Freeze" (Extreme Hypo).
- **Thyroid Storm:** "The Meltdown" (Extreme Hyper).

1. Hypothyroidism (Adding Fuel)

The goal is to replace the hormones the body isn't making.

- **Standard Treatment:**
  - **Levothyroxine (T4):** The most common medication (e.g., Synthroid). It is synthetic T4 that the body converts into active T3.
  - **Liothyronine (T3):** Sometimes added if T4 alone isn't enough, but usually T4 is the gold standard.
- **Hashimoto's & Iodine Deficiency:** Both are treated with lifelong **Levothyroxine**. For iodine deficiency specifically, iodine supplementation is the preventive cure, but once the thyroid fails, replacement hormone is needed.
- **Myxedema Coma (Emergency):**
  - **IV Levothyroxine:** High doses are given intravenously for rapid absorption.
  - **Hydrocortisone:** Often given alongside T4 to prevent an adrenal crisis (the body's stress response can't keep up).

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2. Hyperthyroidism (Putting Out the Fire)

The goal is to stop production or block the effects of excess hormones.

- **Antithyroid Drugs (Thionamides):**
  - **Methimazole:** The preferred first-line drug for most.
  - **Propylthiouracil (PTU):** Often preferred in the **first trimester of pregnancy** or in emergency "storms."
- **Beta-Blockers (e.g., Propranolol):**
  - These don't lower thyroid levels, but they **stop the symptoms** (racing heart, tremors, anxiety) by blocking adrenaline.
- **Thyroid Storm (Emergency):**
  - **High-dose PTU or Methimazole:** To stop new hormone synthesis.
  - **Iodine (Lugol's solution):** Given *after* antithyroid drugs to temporarily "stun" the thyroid and stop it from releasing stored hormones (Wolff-Chaikoff effect).
  - **Glucocorticoids (Dexamethasone):** To inhibit the conversion of T4 into the more active T3.

Medication Key Summary Table

Condition	Primary Medication	Mechanism "Meaning"
Hypothyroidism	**Levothyroxine (T4)**	Synthetic replacement hormone.
Cretinism	**Levothyroxine**	Critical for infant brain development.
Myxedema Coma	**IV Levothyroxine + Steroids**	Emergency "kickstart" for the system.
Hyperthyroidism	**Methimazole / PTU**	Inhibits hormone production.

Thyroid Storm	**PTU + Beta Blockers + Iodine**	"The Multi-Pronged Attack" to stop the heart and thyroid.
Thyroiditis	**NSAIDs / Steroids**	Reduces inflammation (hormone leak).

Pro-Tip for Review: Always remember that **Beta-blockers** are the "band-aid" for hyperthyroidism—they fix how the patient *feels* immediately, while the other drugs fix the actual hormone levels over weeks.

1. Diabetes Insipidus (DI)

The Problem: A deficiency of ADH (Antidiuretic Hormone) or a lack of response to it. Think of ADH as the "Anti-Pee Hormone." Without it, you pee excessively.

- **Key Concept:** "Dry Inside." The body cannot hold onto water.
- **Signs:** Polyuria (up to 20L/day), polydipsia (extreme thirst), very dilute urine (Low Specific Gravity <1.005).
- **Types:** * **Central:** The brain doesn't make ADH (usually head trauma or surgery).
    - **Nephrogenic:** The kidneys don't respond to ADH.
- **Medications:**
    - **Desmopressin (DDAVP):** A synthetic replacement for ADH (used for Central DI).
    - **Vasopressin:** The exogenous form of ADH.
    - **Thiazide Diuretics:** Paradoxically used for Nephrogenic DI to help the kidneys reabsorb water in a different segment.

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2. SIADH (Syndrome of Inappropriate Antidiuretic Hormone)

The Problem: The opposite of DI. Too much "Anti-Pee Hormone." The body holds onto too much water.

- **Key Concept:** "Soaked Inside." Fluid volume overload and **Dilutional Hyponatremia** (low sodium because it's watered down).
- **Signs:** Low urine output, concentrated urine (High Specific Gravity >1.030), weight gain without edema, and seizures (due to low sodium).
- **Medications:**
    - **Furosemide (Lasix):** To loop out the excess water.

- **Hypertonic Saline (3% NaCl):** Only in severe cases to slowly raise sodium levels.
- **Demeclocycline:** An antibiotic used "off-label" because it inhibits ADH action on the kidneys.
- **Vaptans (e.g., Tolvaptan):** Vasopressin receptor antagonists that block ADH.

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3. Diabetes Mellitus (DM)

The Problem: A "Sugar" issue. High blood glucose due to lack of insulin (Type 1) or insulin resistance (Type 2).

- **Key Concept:** "Sweet Blood." The name "Diabetes" refers to the polyuria, but here it is caused by glucose pulling water into the urine (osmotic diuresis).
- **Signs:** The 3 P's (Polyuria, Polydipsia, Polyphagia), fatigue, blurred vision.
- **Medications:**
  - **Insulin:** Essential for Type 1, used in Type 2 when oral meds fail. (Rapid, Short, Intermediate, and Long-acting).
  - **Metformin:** First-line for Type 2; decreases glucose production in the liver.
  - **Sulfonylureas (e.g., Glipizide):** Stimulate the pancreas to squirt out more insulin.
  - **SGLT2 Inhibitors (e.g., Jardiance):** Make the kidneys pee out glucose.

Comparison Summary

Feature	Diabetes Insipidus (DI)	SIADH	Diabetes Mellitus (DM)
Hormone at Fault	Low ADH	High ADH	Low Insulin / Resistance
Primary Issue	Water Loss	Water Retention	High Blood Sugar
Urine Concentration	Very Dilute (Clear)	Very Concentrated	Contains Glucose

Blood Sodium	High (Hypernatremia)	Low (Hyponatremia)	Normal to Low
Primary Med	**Desmopressin**	**Demeclocycline / Vaptans**	**Insulin / Metformin**

Quick Reviewer Tip:

- **DI = D**rain **I**nside (Lose water).
- **SIADH = S**tops **I**ncreased **A**mounts of **D**ischarge **H**2O (Keep water).
- **DM = D**igestive **M**etabolism (Sugar problem).

1. Type 1 Diabetes: The "Missing Key"

The Problem: An autoimmune destruction of the beta cells in the pancreas. The body produces **zero** insulin.

- **Key Phrase:** "Absolute Insulin Deficiency."
- **Patient Profile:** Usually develops in childhood/adolescence (formerly called Juvenile Diabetes).
- **The Emergency: DKA (Diabetic Ketoacidosis)**. Since there is no insulin, the body burns fat for energy, producing acidic ketones.
- **Medication: Lifelong Insulin** is mandatory. Oral meds (like Metformin) generally do not work because there is no insulin to optimize.

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2. Type 2 Diabetes: The "Jammed Lock"

The Problem: The body makes insulin, but the cells ignore it (**Insulin Resistance**). Eventually, the pancreas gets tired and stops making enough.

- **Key Phrase:** "Relative Insulin Deficiency & Resistance."
- **Patient Profile:** Often linked to obesity, sedentary lifestyle, and genetics.
- **The Emergency: HHS (Hyperglycemic Hyperosmolar State)**. Extremely high blood sugar without the ketones found in DKA.
- **Medications:** * **Metformin:** The "Gold Standard" (sensitizes the locks).
    - **Sulfonylureas (Glipizide):** Pushes the pancreas to work harder.
    - **GLP-1 Agonists (Ozempic/Trulicity):** Mimics gut hormones to slow digestion and trigger insulin.
    - **SGLT2 Inhibitors (Jardiance):** Flushes sugar out through the urine.

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3. Gestational Diabetes (GDM): The "Pregnancy Block"

The Problem: Hormones produced by the **placenta** block the action of insulin in the mother.

- **Key Phrase:** "Placental Insulin Resistance."
- **Screening:** Tested at **24–28 weeks** gestation via the Oral Glucose Tolerance Test (OGTT).
- **Risks:** Macrosomia (large baby) and neonatal hypoglycemia after birth.
- **Medications:** * **Diet & Exercise:** First-line treatment.
    - **Insulin:** Preferred if lifestyle changes fail (it does not cross the placenta).
    - **Glyburide/Metformin:** Sometimes used, but insulin is safer.

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4. Secondary Diabetes: The "Side Effect"

The Problem: High blood sugar caused by *another* medical condition or a specific medication.

- **Common Causes:**
    - **Cushing's Syndrome:** Excess cortisol increases glucose.
    - **Pancreatitis/Cystic Fibrosis:** Physical damage to the pancreas.
    - **Drug-Induced:** Long-term use of **Corticosteroids** (e.g., Prednisone) is the #1 culprit.
- **Management:** Treat the underlying cause. If it's steroid-induced, the patient may need temporary insulin until the steroid dose is lowered.

Quick Comparison for Review

Type	Onset	Primary Pathophysiology	Key Emergency
Type 1	Sudden/Young	Autoimmune (No Insulin)	**DKA**

Type 2	Gradual/Adult	Resistance (Jammed Lock)	**HHS**
Gestational	2nd-3rd Trimester	Placental Hormones	Macrosomia
Secondary	Variable	Meds (Steroids) or Disease	Variable

"Meaning" Key Words

- **Type 1:** Destruction / Absolute / DKA.
- **Type 2:** Resistance / Exhaustion / Lifestyle.
- **Gestational:** Placenta / Temporary / 24-28 Weeks.
- **Secondary:** Steroids / Damage / Underlying Cause.

Do you need a breakdown of the different types of **Insulin** (Rapid vs. Long-acting) next?

1. Diabetic Retinopathy (The Eyes)

The Problem: Damage to the retinal blood vessels, leading to vision loss and eventually blindness.

- **Pathophysiology:** Microaneurysms (small bulges) leak fluid, or new, fragile blood vessels grow (neovascularization) and bleed into the eye.
- **Key Signs:** Floaters, blurred vision, "dark spots" in the visual field.
- **Reviewer Lab/Exam:** Annual **Funduscopic (dilated eye) exam**.
- **Management:** Blood glucose and BP control; Laser photocoagulation to "seal" leaking vessels.

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2. Diabetic Nephropathy (The Kidneys)

The Problem: Damage to the filtering units (nephrons) of the kidney. This is the #1 cause of End-Stage Renal Disease (ESRD).

- **Pathophysiology:** High pressure and sugar damage the basement membrane of the glomerulus.
- **Key Lab Finding: Albuminuria** (protein in the urine). The presence of "Microalbumin" is the earliest warning sign.
- **Reviewer Lab/Exam: Gopher (GFR)**, Serum Creatinine, and 24-hour urine for albumin.
- **Meds: ACE Inhibitors (e.g., Lisinopril)** or **ARBs**. Even if the patient doesn't have high BP, these meds are given to protect the kidneys by reducing pressure in the renal arteries.

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3. Diabetic Neuropathy (The Nerves)

The Problem: Nerve damage caused by metabolic derangement and reduced blood flow to nerve tissues.

- **Types:**
  - **Peripheral:** "Stocking-glove" distribution (numbness/tingling in hands and feet).
  - **Autonomic:** Gastroparesis (slow stomach emptying), orthostatic hypotension, or "silent" MI (not feeling chest pain).
- **Key Signs:** Paresthesia (tingling), burning pain, or total loss of sensation.
- **Meds: Gabapentin** or **Pregabalin** (for nerve pain) and **Duloxetine**.

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4. Foot Ulcers (The Consequence)

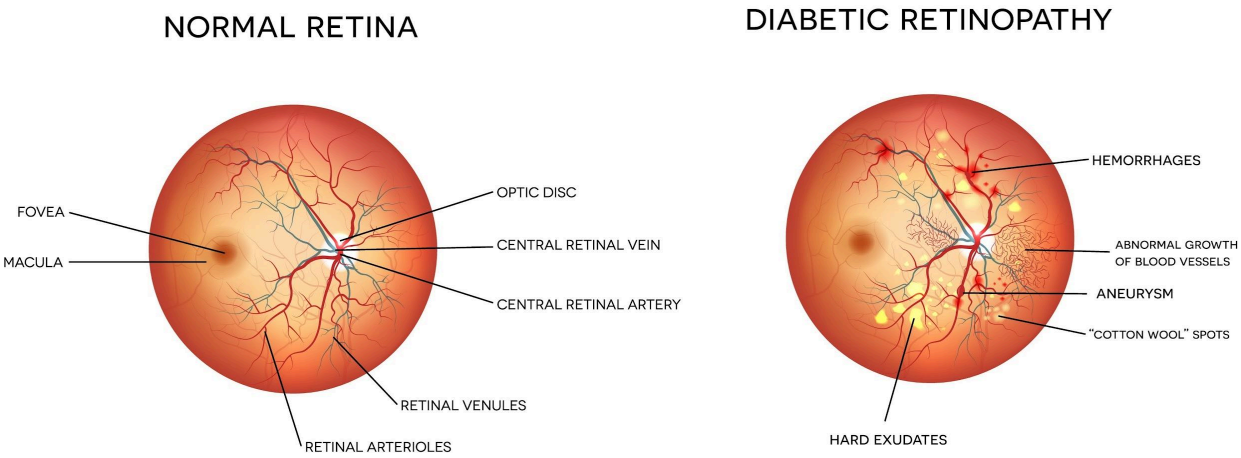
The Problem: A "Perfect Storm" of **Neuropathy** (can't feel the injury) + **Peripheral Vascular Disease** (poor blood flow to heal the injury) + **Hyperglycemia** (bacteria love sugar).

- **Pathophysiology:** A patient steps on a tack, doesn't feel it (neuropathy), the wound doesn't get oxygen/nutrients (ischemia), and it becomes infected (gangrene), often leading to amputation.
- **Management/Nursing Care:**
  - **Daily foot inspections** (use a mirror for the heels).
  - Never go barefoot; wear well-fitting shoes.
  - Cut toenails straight across (or see a podiatrist).
  - Test water temperature with the elbow/thermometer, not the feet.

Summary for Reviewer

Complication	Target Organ	Early Warning Sign	Key Prevention
Retinopathy	Eyes	Blurry vision/Floaters	Annual Dilated Eye Exam
Nephropathy	Kidneys	**Albuminuria**	ACE Inhibitors / ARBs
Neuropathy	Nerves	Tingling/Burning/Numbness	Tight Glucose Control
Foot Ulcer	Extremities	Redness/Blisters (unfelt)	Daily Inspection/Podiatry

DIABETIC RETINOPATHY



Meaning Key Words

- **Retinopathy:** "Hemorrhage / Neovascularization / Blindness."
- **Nephropathy:** "Albumin / ACE Inhibitors / Renal Failure."
- **Neuropathy:** "Stocking-Glove / Paresthesia / Gastroparesis."
- **Foot Ulcers:** "Insensate (no feeling) / Ischemia / Amputation."

1. Hypoglycemia (The "Insulin Shock")

The most common and immediate danger. This occurs when there is too much insulin relative to glucose.

- **The "Rule of 15":** 1. Give **15g** of rapid-acting carbs (4oz juice, 3-4 glucose tabs). 2. Wait **15 mins** and recheck blood sugar. 3. If still below 70 mg/dL, repeat. 4. Once stable, give a snack with **protein/complex carb** (e.g., peanut butter crackers) to keep it stable.
- **Emergency Treatment:** If the patient is unconscious, give **Glucagon (IM/SubQ)** or **Dextrose 50% (IV Push)**.

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2. Lipodystrophies (Injection Site Issues)

Local reactions at the site of repeated injections.

- **Lipoatrophy:** Loss of subcutaneous fat (appears as "dimpling" or pits in the skin).
- **Lipohypertrophy:** Accumulation of extra fat (appears as "rubbery" lumps).
    - **The Problem:** Injecting into these lumps significantly **delays insulin absorption**, leading to erratic blood sugar levels.
- **Prevention: Rotate sites!** Think of the abdomen like a clock or a grid and move 1 inch with every shot.

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3. The "Morning Highs" (Somogyi vs. Dawn)

If a patient wakes up with high blood sugar (Hyperglycemia), you have to figure out *why* before you change the dose.

Feature	Somogyi Effect	Dawn Phenomenon
The Cause	**Rebound:** Too much nighttime insulin causes a "dip" (hypo) at 3 AM. The body panics and releases stress hormones to spike sugar.	**Growth:** Normal release of Growth Hormone/Cortisol in the early morning spikes sugar.
3 AM Blood Sugar	**Low** ↓	**High** or Normal ↑
The Fix	**Decrease** bedtime insulin or give a bedtime snack.	**Increase** bedtime insulin or adjust timing.

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4. Systemic Reactions

- **Allergic Reactions:** Rare with modern "human" insulin, but local redness or swelling can occur.
- **Insulin Edema:** A temporary condition where the body retains fluid (usually in the legs) when starting intensive insulin therapy for the first time.
- **Weight Gain:** Because insulin is an **anabolic** (building) hormone, it moves sugar into cells for storage, which can lead to weight gain if diet isn't adjusted.

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5. Insulin Resistance

Over time, some patients (especially in Type 2) require massive doses (e.g., >200 units/day) because their cells become increasingly "deaf" to the insulin signal. This is often managed by adding non-insulin meds like Metformin.

Quick Reviewer Tip:

- **Hypoglycemia:** "Cool and Clammy, need some Candy."
- **Hyperglycemia:** "High and Dry, sugar High."
- **Somogyi:** S = **S**leeping **S**ugar is **S**inking (Low at 3 AM).
- **Dawn:** D = **D**ownright **D**angerous (High all night/morning).

1. Central Diabetes Insipidus (Neurogenic)

The most common form. The problem is at the "source"—the **Hypothalamus** or the **Posterior Pituitary**.

- **The Problem:** The body does not **produce or release** enough ADH (Vasopressin).
- **Common Causes:** Head trauma, brain tumors, neurosurgery (especially involving the pituitary gland), or infections like meningitis.
- **Key Treatment: Desmopressin (DDAVP)**. Since the body is just missing the hormone, we replace it.

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2. Nephrogenic Diabetes Insipidus

The problem is at the "destination"—the **Kidneys**.

- **The Problem:** There is plenty of ADH in the blood, but the kidneys are **resistant** to it. The "locks" on the renal tubules are broken.

- **Common Causes:**
    - **Lithium Toxicity:** (A classic board exam question).
    - **Hypercalcemia** or **Hypokalemia**.
    - Genetic mutations or chronic kidney disease.
- **Key Treatment:** Replacing ADH (DDAVP) **will not work** because the kidneys can't "see" it. Treatment involves a low-sodium diet and, paradoxically, **Thiazide diuretics**.

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3. Dipsogenic Diabetes Insipidus (Primary Polydipsia)

This is actually a problem of **intake**, not necessarily a physical hormone defect.

- **The Problem:** A defect in the thirst mechanism in the hypothalamus causes the person to feel pathologically thirsty.
- **The Result:** They drink so much water that they suppress their own ADH and pee constantly.
- **Common Causes:** Often associated with mental health conditions (Schizophrenia) or damage to the thirst center of the brain.
- **Key Treatment:** Behavioral modification and fluid restriction. Giving ADH is dangerous here as it can cause water intoxication.

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4. Gestational Diabetes Insipidus

A rare condition occurring only during pregnancy.

- **The Problem:** The **placenta** produces an enzyme (vasopressinase) that breaks down the mother's ADH.
- **Outcome:** Usually resolves on its own after delivery.
- **Key Treatment:** Desmopressin (DDAVP) is often effective because it is more resistant to the placental enzyme than natural ADH.

The "Water Deprivation Test" (Reviewer Gold)

How do doctors tell them apart? They stop the patient from drinking and then give a dose of Vasopressin (ADH).

Response to Vasopressin Injection	Diagnosis
Urine concentrates (Stops peeing)	**Central DI** (The kidney was just waiting for the signal).
Urine stays dilute (Keeps peeing)	**Nephrogenic DI** (The kidney doesn't care about the signal).

Meaning Key Words

- **Central:** Brain / Trauma / DDAVP.
- **Nephrogenic:** Kidney / Lithium / Resistance.
- **Dipsogenic:** Thirst / Behavioral / Intake.
- **Gestational:** Placenta / Enzyme / Temporary.

1. Acute Complications (The Emergencies)

These happen fast and require immediate clinical intervention.

- **Hypoglycemia (The "Insulin Shock"):** * **Cause:** Too much insulin, skipping meals, or excessive exercise.
    - **Signs:** Diaphoresis (sweating), tremors, tachycardia, confusion.
    - **Rule of 15:** 15g carbs, wait 15 mins, repeat if sugar is still <70.
- **Diabetic Ketoacidosis (DKA) - Primarily Type 1:** * **Cause:** No insulin → body burns fat → Ketones (acid) build up.
    - **Signs:** Kussmaul respirations (deep, rapid breathing), **fruity breath**, abdominal pain.
- **Hyperglycemic Hyperosmolar State (HHS) - Primarily Type 2:** * **Cause:** Extreme dehydration and high sugar (>600) without the acid.
    - **Signs:** Severe dehydration, altered mental status, but **no** fruity breath/ketones.

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2. Chronic Microvascular (Small Vessel Damage)

Think of these as the "three -opathies."

- **Retinopathy (Eyes):** Hemorrhages and new vessel growth in the retina. Can lead to retinal detachment and **blindness**.
- **Nephropathy (Kidneys):** Damage to the renal filters. Look for **Albumin (protein)** in the urine. It is the #1 cause of dialysis.
- **Neuropathy (Nerves):** * **Peripheral:** "Stocking-glove" numbness/pain in feet and hands.
    - **Autonomic:** Gastroparesis (stomach doesn't empty), silent MI, or erectile dysfunction.

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3. Chronic Macrovascular (Large Vessel Damage)

High sugar acts like "sandpaper" inside the large arteries, leading to plaque buildup (Atherosclerosis).

- **Cardiovascular:** High risk for Myocardial Infarction (Heart Attack) and Hypertension.
- **Cerebrovascular:** Increased risk for CVA (Stroke).
- **Peripheral Vascular Disease (PVD):** Poor circulation to the legs, leading to cold skin, decreased pulses, and slow-healing wounds.

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4. The Diabetic Foot (The "Perfect Storm")

This is a combination of **Neuropathy** (can't feel the injury) + **PVD** (can't heal the injury) + **Infection** (bacteria love high sugar).

- **Management:** Daily foot checks with a mirror, never walking barefoot, and proper shoe fitting.

Summary Reviewer Table

Complication	Type	Key Marker/Sign	Priority Action
Hypoglycemia	Acute	"Cool and Clammy"	15g Fast Carbs

DKA	Acute	Ketones / Acidosis	IV Fluids + Regular Insulin
Retinopathy	Chronic	Vision loss	Annual Dilated Eye Exam
Nephropathy	Chronic	Albuminuria	ACE Inhibitors ("-pril")
Neuropathy	Chronic	Burning/Numbness	Foot Safety / Glucose Control

"Meaning" Key Words

- **DKA:** "Acid / Fruit / Type 1."
- **HHS:** "Dehydration / Extreme Sugar / Type 2."
- **Microvascular:** "Eyes / Kidneys / Nerves."
- **Macrovascular:** "Heart / Brain / Circulation."

INSULIN THERAPY

Types.	**Onset.**	**Peak.**	**Duration**
Rapid.	**15min.**	**1hr.**	**3-5 hrs**
Storm Acting	**30 min**	**2-4hrs**	**6-8hrs**
Intermediate	**1-2 hours**	**6-12 hrs.**	**18hrs**
Long-Acting	**1-2 hours**	**None.**	**24hrs**