

Hypoglycemia in a Diabetic

Hypoglycemia in a diabetic can come from a number of causes. The usual suspects are **too little food**, **too much exercise**, and **too much medication**. Hypoglycemia is defined as a **bG < 70** or **symptoms of hypoglycemia** (palpitations, perspiration, presyncope); it's corrected with ingestion of sugar. Severe hypoglycemia may cause coma, anoxic brain injury, and death.

The most important thing to do is get the sugar up. Do it with an **oral glucose load** if the patient is awake, or with **IV D50** if the patient is in a coma. After the event has resolved, assess life-style and medications to determine exactly what happened and try to prevent it in the future.

Hypoglycemic events are potentially fatal and should be treated with significantly more acuity than a high reading.

Hypoglycemia in a Non-Diabetic

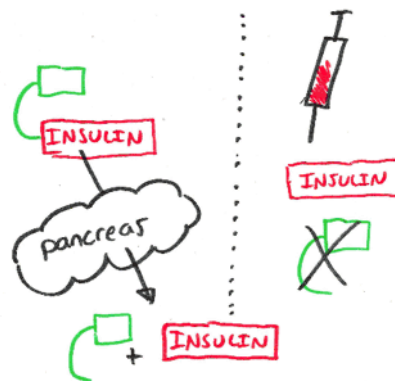
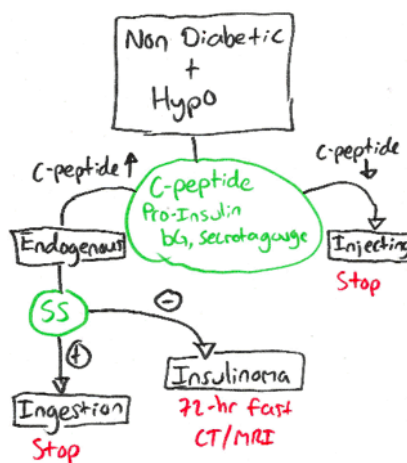
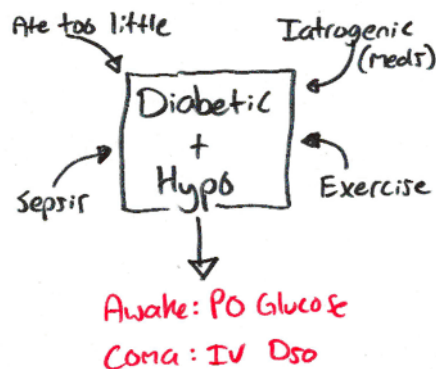
Hypoglycemia, a **bG < 60 AND symptoms**, in a non-diabetic is usually factitious disorder. Two potential disease states (that are quite rare) are **insulinoma** and **autoimmune hypoglycemia**. To discover if the patient is doing it to themselves, obtain a **C-peptide**, **Pro-Insulin**, **bG**, and **Secretagogue screen**.

Endogenous insulin comes from Pro-insulin, cleaving the C-peptide portion to result in insulin. Thus, if there's **NO rise in C-peptide**, the insulin must be exogenous (**self-injecting**). Your job is done if the C-peptide is normal.

Endogenous insulin secretion may be **induced by secretagogues** (like sulfonylureas) or **produced by insulinomas**. The only way to tell the two apart is to obtain a **secretagogue screen**. If positive, they're ingesting secretagogues - tell them to stop.

Only evaluate for insulinoma if the **C-peptide is elevated AND the secretagogue screen is negative**. Perform a 72-hour fast, retest for all the same things above, and if positive perform the CT scan or MRI of the abdomen to find + resect the tumor.

If all else fails, consider the option of looking at **insulin antibodies**.



Diabetic Ketoacidosis

DKA is a life-threatening emergency of Type I diabetics. There is plenty of sugar in the blood, but without insulin none of it can get into the cells. It's as if the patient is starving. The brain activates **ketones** from fatty acids, causing both **ketosis** and **acidosis**. Simultaneously, the high levels of sugar in the blood spill into the urine. With the T_m of the renal tubules at only about 180, excess glucose is spilled into the urine. Glucose is a potent osmotic diuretic; the patient becomes dehydrated.

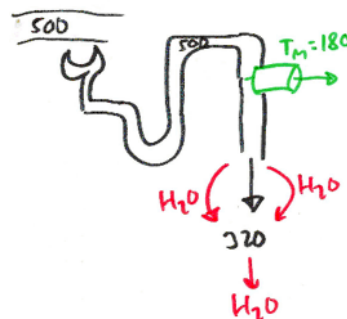
The patient will present **obtunded** or **in a coma**. They will be dry. Diagnose the condition by finding **elevation blood glucose** (Diabetic) **ketones in the urine and blood** (Keto) and **acidosis on ABG / anion gap on BMP** (Acidosis).

The treatment is threefold: **Replete Potassium** before giving Insulin, **IV Insulin**, and **Fluid**. Monitor therapy with hourly blood glucoses and BMPs every 4 hours. If the **gap hasn't closed** but the **glucose is approaching normal** (this value differs per protocol) switch to **D5 ½ NS**. As the anion gap resolves ("the gap closes") bridge to **subQ insulin** long acting and let them eat.

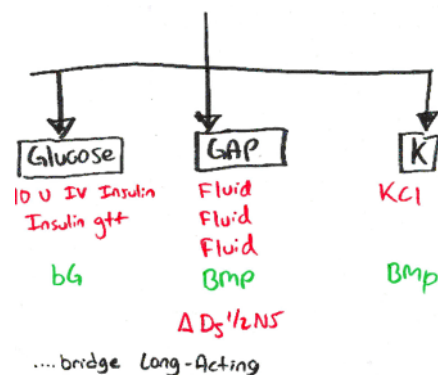
While insulin noncompliance is the most common cause of DKA, also look for NSTEMI, GI bleeds, and infections as precipitating causes.

Hyperosmolar Hyperglycemic Nonketotic Coma

HHNKC or HHS is the life-threatening emergency of Type II diabetics. **No ketones** are made because there's sufficient insulin to feed the brain. **No acidosis** occurs because fatty acids aren't accessed. However, the patient will still present with **coma** because of **profound dehydration**. The blood glucoses are often much more elevated in HHNKC than in DKA + the diuresis has gone on longer. This patient needs **fluids and IV insulin**.



Mechanism of dehydration in hyperglycemia. Excess glucose is lost in urine and draws with it water, leading to potent diuresis.



Treatment of DKA. Replete K, give insulin, then give fluids. HHS/HHNKC is the same thing, except no need to follow anion gap.

Characteristic	DKA	HHNKC / HHS
Path:	Type I, Insulin Dependent Diabetes Mellitus (IDDM)	Type II, Non-Insulin Dependent Diabetes Mellitus (NIDDM)
Pt:	+ Diabetic Coma + <u>K</u> etones + <u>A</u> cidosis	+ Diabetic Coma - Ketones - Acidosis
Dx:	bG 300-500 U/A: + Ketones ABG: + Acidosis BMP: + Gap	bG 800-1000 U/A: - Ketones ABG: - Acidosis BMP: - Gap
Tx:	Replete K IV Fluids – Bolus a lot IV insulin Follow the GAP	Replete K IV Fluids – Bolus a lot IV Insulin Follow the symptomatic improvement