

EMT Diabetic Ketoacidosis (DKA) Study Guide

By Forrest Munden, MundenInteractive.com

Diabetic ketoacidosis (DKA) is one of those calls where the patient looks like a lot of other things: the flu, a belly bug, an overdose, sometimes a drunk. Then you notice the deep, heavy breathing and the fruity smell on their breath, and the picture comes together. This guide covers what DKA is and why it makes the blood acidic, where ketones come from, why the patient breathes the way they do, how DKA differs from HHS, how insulin works, Type 1 versus Type 2 diabetes, what high and low blood sugar do to the body, and what an EMT does about it, including how to keep an IV dextrose line safe while you assist ALS. Throughout, I'll use the freeway analogy I teach in class, because it makes the physiology stick.

Guideline version verified: 2024 American Heart Association and American Red Cross Guidelines for First Aid (hypoglycemia), and 2025 AHA Guidelines for CPR and Emergency Cardiovascular Care (published October 22, 2025), Part 7 (Adult BLS) and Part 10 (Special Circumstances, hyperkalemia). Checked October 30, 2026: no 2026 AHA update has replaced them. AHA guidelines don't cover DKA treatment itself. The diabetes physiology here is standard textbook physiology, and DKA care is protocol-driven: follow your local protocol and medical direction.

Disclaimer: This is a study aid, not official. For PDF, copy into a word processor and export. Always follow current local protocols, medical direction, NREMT skill sheets, and the latest AHA guidelines. Blood sugar numbers in the freeway analogy are teaching approximations, not AHA treatment thresholds.

Section 1: The Freeway: How Blood Sugar and Insulin Work

Here's how I teach it. Picture the bloodstream as a **freeway loop**, a circular track that runs past every neighborhood in the body. The **cars are glucose** (sugar). The **houses are the body's cells**. Each car carries fuel to a house so the house can turn its lights on and do its

work. **Insulin is the off-ramps.** Without off-ramps, the cars just circle; they can't exit the freeway and get home.

- **Where insulin comes from:** the **beta cells** in the islets of Langerhans in the pancreas. When sugar rises after a meal, the beta cells release insulin. Insulin binds to receptors on the cells, especially in muscle, fat, and liver, and opens the way for glucose to move out of the blood and into the cell. It also tells the liver to store sugar and stop making more, and tells fat cells to stop breaking down fat.
- **Its counterpart: glucagon**, from the pancreatic alpha cells. When sugar runs low, glucagon tells the liver to release stored sugar. Think of it as an on-ramp that adds cars when traffic gets thin.
- **Healthy traffic:** in my freeway model, roughly **80–120 mg/dL**. There are enough cars to supply every house, and they move freely. (AHA's 2024 First Aid guideline describes a normal range of 70–120 mg/dL and defines hypoglycemia as below 70. Your protocol's numbers are the ones you treat by.)
- **Too few cars** (below about 80 in my model): nobody is getting home. The houses go dark. The brain notices first because it runs almost entirely on glucose. This is hypoglycemia.
- **Gridlock** (around 300–400 and up): so many cars that nothing moves. The fuel is all on the freeway, and the houses still aren't getting it.

Section 2: What DKA Is, and Why the Body Turns Acidic

The name tells the story. Diabetic: it happens in diabetes. **Keto:** ketones build up. **Acidosis:** the blood becomes too acidic.

In DKA there is essentially **no insulin**, which means **no off-ramps**. Sugar keeps piling onto the freeway into total gridlock, and the houses sit dark and starving with all that fuel stuck outside. Then two things make it worse:

1. **The body thinks it's starving, so it opens more on-ramps.** Glucagon and stress hormones (epinephrine, cortisol) tell the liver to dump even more sugar into traffic that's already gridlocked.
2. **The houses fire up the backup generator.** Cells that can't get sugar start burning **fat**. The liver breaks the fat down into **ketone bodies** (acetoacetate, beta-hydroxybutyrate, and acetone) as an emergency fuel. A little is normal. A flood is not.

Ketones are **acids**, and when they pile up faster than the body can buffer them, blood pH falls. That is the acidosis.

Meanwhile the kidneys try to clear the gridlock by spilling sugar into the urine. Sugar drags water with it, so the patient urinates heavily, gets thirsty, and becomes severely **dehydrated**, losing potassium and other electrolytes along the way. Vomiting makes all of it worse.

Section 3: Kussmaul Respirations and the Fruity Breath

- **Kussmaul respirations** are deep, labored, often rapid breaths. They are not a primary lung problem; they are **compensation**. CO₂ in the blood acts as an acid, so the body blows off as much CO₂ as it can to drag the pH back toward normal.
- **Fruity or acetone breath:** acetone, one of the ketones, is exhaled. It's a classic finding, but not everyone can smell it.
- **EMT takeaway:** this patient is breathing hard on purpose. Support the airway, give oxygen per protocol, and don't try to "calm" the breathing down. If the patient tires out and needs assisted ventilation, that's a critical patient. Call for ALS early and tell them.

Common DKA Signs and Symptoms:

- Excessive thirst, frequent urination, and hunger (the "three P's": polydipsia, polyuria, polyphagia)
- Nausea, vomiting, abdominal pain
- Weakness, confusion, or decreasing responsiveness
- Tachycardia; dry mouth and skin; signs of shock late
- Kussmaul respirations and fruity breath
- A history of missed insulin, an insulin pump problem, recent illness or infection, or no known diabetes at all (DKA can be how Type 1 is first discovered)

Section 4: DKA vs HHS

Hyperosmolar hyperglycemic state (HHS) is the other high-sugar emergency. In freeway terms: in HHS **a few off-ramps are still open**. Just enough insulin is working that the houses

never fully switch to the fat-burning backup generator, so there's no big ketone acid problem. But traffic backs up slowly over days into an extreme gridlock, and the kidneys flush out enormous amounts of water trying to clear it.

	DKA	HHS
Typical patient	Usually Type 1 (can occur in Type 2)	Usually Type 2, often older
Insulin	Essentially none: no off-ramps	Some: a few off-ramps open
Onset	Hours to a day or two	Days to weeks
Ketones / acidosis	Yes: the defining problem	Minimal or absent
Breathing	Kussmaul, fruity breath	Usually not Kussmaul
Blood sugar	High	Often much higher
Dehydration / mental status	Significant	Often profound; confusion, seizures, or coma are common

Section 5: Type 1 vs Type 2: Why Each Can Run High or Low

	Type 1	Type 2
Problem	The immune system destroys the beta cells, so little or no insulin is made. The off-ramps are torn out.	The cells resist insulin and the pancreas can't keep up. The off-ramps are narrowed and backed up.
Treatment	Insulin for life (injections or pump)	Diet, exercise, oral and injectable medications, sometimes insulin
Why it runs HIGH	Missed or insufficient insulin, a pump failure, illness or infection (stress hormones raise sugar)	Illness or infection, missed medications, steroids, and poor control over time
Why it runs LOW	Too much insulin, a missed or late meal, extra exercise, alcohol	Insulin or certain oral medications (some can cause prolonged lows), a missed meal, kidney problems, alcohol
Typical high-sugar emergency	DKA	HHS

Low sugar in freeway terms: too many off-ramps open at once (too much insulin or medication), or not enough cars entering (no food). The freeway empties and the houses go dark. The signs come fast: shakiness, sweating, a fast heart rate, confusion, slurred speech, and odd behavior, sometimes progressing to unresponsiveness or seizure.

Section 6: After-Effects of High vs Low Sugar

- **Low sugar is the faster killer.** AHA's First Aid guideline notes that untreated hypoglycemia can cause seizures, status epilepticus, permanent brain injury, and

death. It can also mimic a stroke. AHA lists hypoglycemia as a common stroke mimic and supports checking glucose when you can do it without delaying care.

- **High sugar in the short term:** dehydration, electrolyte loss, acidosis (DKA), confusion and coma (HHS), and dangerous potassium shifts that can cause dysrhythmias.
- **High sugar over years:** damage to blood vessels and nerves. That means heart attack and stroke risk, kidney failure, vision loss, numb feet, poor wound healing, and infections. This is why diabetic patients may have “silent” heart attacks with vague symptoms.

Section 7: EMT Management

Hypoglycemia (AHA 2024 First Aid):

- **Awake and able to swallow:** give oral glucose (Class 1). AHA notes that ≥ 20 g of oral glucose can rapidly restore blood sugar. Glucose tablets are preferred over gel or dietary sugars if available (2a). Simple dietary sugars are reasonable if there are no tablets or gel (2a).
- **Not awake or unable to swallow: no oral glucose** (Class 3: Harm), because of the risk of choking and aspiration. These patients need IV dextrose from ALS, which AHA calls potentially lifesaving.
- **Escalate** if the patient can't swallow, has a seizure, or doesn't improve within 10 minutes of oral glucose.
- Check blood glucose if it is in your scope and protocol. Reassess after treatment.

Suspected DKA or HHS:

- Manage the ABCs, give oxygen per protocol, and position for comfort or in the recovery position if responsiveness drops.
- **Request ALS early.** These patients need IV fluids, cardiac monitoring, and hospital insulin therapy. EMTs do not give insulin.
- Note the medications, insulin pump, last dose, last meal, recent illness, and urine output, and bring the glucose meter and medications to the hospital.
- Expect vomiting. Have suction ready.

- If the patient goes into cardiac arrest, deliver high-quality CPR and use the AED. AHA's 2025 Part 10 notes that acidemia can push potassium out of the cells, and high potassium can trigger lethal rhythms.

Section 8: IV Dextrose Safety: Recognize a Bad Site (EMT Awareness)

When ALS gives IV dextrose for a low sugar, you'll often be the one holding the arm, spiking the bag, or watching the site. **D50 is highly concentrated and irritating to tissue, and D10 is less so but still irritating.** If it leaks out of the vein into the surrounding tissue (**extravasation**), it can cause serious tissue damage, including tissue death (necrosis). This is a standard clinical safety principle, not an AHA recommendation.

- **Before the push, ALS should confirm a good line:** it flushes easily, there's no swelling, there's blood return or free flow, and ideally it's in a larger vein.
- **During the push, watch the site:** swelling, puffiness, blanching, coolness, pain or burning, or the fluid stopping or slowing.
- **If you see it, say it immediately and clearly:** "The site is swelling, stop the push."
- **Assist per protocol.** Typical steps include stopping the infusion right away, leaving the catheter in place initially per protocol, not flushing, and elevating the limb. The receiving facility is notified and everything is documented. ALS and medical direction make the treatment decisions.

Section 9: What Changed From 2020

- **First Aid:** AHA's first aid recommendations now come from the 2024 AHA/Red Cross First Aid guidelines, which supersede the 2015 guidelines and the 2019/2020 focused updates. Hypoglycemia recommendations include glucose tablets as preferred, dietary sugars as an alternative, a 10-minute escalation trigger, and a sugar-and-water slurry under the tongue that may be reasonable for children who are awake but won't swallow (2b).
- **2025 Part 10 hyperkalemia section:** for cardiac arrest from suspected hyperkalemia, the effectiveness of IV calcium, sodium bicarbonate, and insulin with

glucose is *not well established* (2b). This is ALS care, but it explains why the “potassium problem” in DKA matters.

- **DKA itself** isn’t addressed in the AHA guidelines in either edition. Physiology and DKA treatment come from standard references and your protocol.

Section 10: Putting It Together

Question: A 19-year-old Type 1 diabetic has vomited for a day and says she “couldn’t keep food down, so I skipped my insulin.” She’s confused, breathing deep and fast, and her breath smells fruity. Her glucose reads “HI.” What’s happening, and what do you do?

Answer: This is suspected DKA. Support the airway and breathing, give oxygen per protocol, and call ALS early. Don’t give oral glucose and don’t try to slow her breathing. Monitor and transport, and prepare to assist with IV access, watching the site.

Reasoning: No insulin means no off-ramps. Sugar is gridlocked in the blood while her cells starve, so her body burns fat and floods itself with acidic ketones. The deep breathing is her body blowing off CO₂ to fight the acid. Skipping insulin because she wasn’t eating is a classic trigger: illness raises sugar even without food.

Sources (American Heart Association only)

- 2024 American Heart Association and American Red Cross Guidelines for First Aid. *Circulation*. 2024;150(24):e519–e579. doi:10.1161/CIR.0000000000001281 (First Aid for Hypoglycemia; stroke recognition)
- Part 10: Adult and Pediatric Special Circumstances of Resuscitation: 2025 AHA Guidelines for CPR and ECC. *Circulation*. 2025;152(16 suppl 2):S578–S672. doi:10.1161/CIR.0000000000001380 (Hyperkalemia)
- Part 7: Adult Basic Life Support: 2025 AHA Guidelines for CPR and ECC. *Circulation*. 2025;152(suppl 2):S448–S478. doi:10.1161/CIR.0000000000001369

The diabetes physiology, DKA/HHS comparison, and IV dextrose extravasation guidance above are standard clinical teaching, not AHA recommendations. The freeway analogy is Forrest Munden’s teaching model.