

Paramedic Diabetic Ketoacidosis (DKA) Study Guide

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DKA is a metabolic emergency that turns into a resuscitation problem when the acid, the dehydration, and the potassium get ahead of you. This paramedic and ACLS-level companion to the EMT DKA guide goes deeper into the pathophysiology: ketogenesis, the acid-base picture, why Kussmaul breathing matters if you take over the airway, total-body potassium, HHS versus DKA, Type 1 versus Type 2, and where the AHA guidelines actually apply. That means hypoglycemia, hyperkalemia, and the reversible causes of arrest. It also covers how to give IV dextrose without hurting the patient. I'll carry my freeway analogy through it, because it explains the "why" behind every finding.

Guideline version verified: 2025 American Heart Association Guidelines for CPR and Emergency Cardiovascular Care (published October 22, 2025), Part 9 (Adult ALS), Part 8 (PALS), and Part 10 (Special Circumstances: Hyperkalemia), plus the 2024 AHA/American Red Cross Guidelines for First Aid (hypoglycemia). Checked October 30, 2026: no 2026 AHA update has replaced them. AHA doesn't publish DKA treatment guidelines, so the fluid, insulin, and electrolyte strategy here is in-hospital and 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 2025 ACLS/PALS guidelines. Blood sugar numbers in the freeway analogy are teaching approximations, not AHA treatment thresholds; treat by your protocol's numbers.

Section 1: The Freeway Model of Glucose and Insulin

Picture the bloodstream as a **circular freeway** and glucose molecules as the **cars** on it. The **houses** along the route are the body's cells, and each car is fuel that lets a house turn on its lights. **Insulin builds the off-ramps.** It's secreted by the **beta cells** of the pancreatic islets and binds receptors on muscle, fat, and liver cells, letting glucose leave the blood and enter

the cell. Insulin also tells the liver to stop making new glucose (gluconeogenesis) and stop breaking down glycogen, and tells fat cells to stop breaking down fat (lipolysis). That last job is the one DKA turns on. **Glucagon**, from the alpha cells, and stress hormones (epinephrine, cortisol, growth hormone) are the **on-ramps**: they add cars.

Freeway state (teaching approximation)	Physiology
Healthy traffic, ~80–120 mg/dL	Insulin and glucagon in balance. AHA's 2024 First Aid guideline describes a normal range of 70–120 mg/dL.
Too few cars, below ~80	Hypoglycemia. AHA defines it as <70 mg/dL and notes symptoms typically develop below 50–60. The brain has almost no fuel reserve.
Gridlock, ~300–400 and up	Hyperglycemia. Plenty of fuel is in the blood, but the cells can't use it.

Section 2: DKA Pathophysiology: Gridlock With the Houses Starving

DKA is **absolute or near-absolute insulin deficiency**, so there are no off-ramps, made worse by a surge of counter-regulatory hormones that open every on-ramp. Three processes run at once:

1. **Hyperglycemia.** Cars pour in from the liver (gluconeogenesis and glycogen breakdown) while none can exit. Freeway gridlock.
2. **Ketogenesis.** The cells are starving in the middle of plenty. Without insulin's brake, fat cells release free fatty acids, and the liver converts them to **ketone bodies**: beta-hydroxybutyrate, acetoacetate, and acetone. The two main ones are acids. Their hydrogen ions use up the body's bicarbonate buffer, producing a **high anion gap metabolic acidosis**. In freeway terms, the houses fire up a smoky backup generator, and the exhaust is acid.
3. **Osmotic diuresis.** Glucose spills into the urine and drags water, sodium, potassium, magnesium, and phosphate with it. The result is profound volume depletion. Vomiting

adds to the losses, and falling kidney perfusion makes it harder to clear both glucose and acid. The gridlock tightens.

Common triggers: infection or illness, missed insulin, insulin pump failure (the catheter is kinked or dislodged, or the pump stops delivering), new-onset Type 1 diabetes, MI, pancreatitis, alcohol or drugs, pregnancy, and certain medications. Some diabetes drugs can cause DKA with only modestly elevated glucose (“euglycemic DKA”). A normal-looking sugar in a sick, acidotic diabetic doesn’t rule DKA out.

Section 3: Kussmaul Respirations: The Patient’s Own Bicarb

- Deep, labored, often rapid breathing is **respiratory compensation** for metabolic acidosis. The patient is blowing off CO_2 to push the pH back up. EtCO_2 will often run low. That reflects compensation, not necessarily hyperventilation that needs to be corrected.
- **Airway caution:** if you sedate, paralyze, or bag this patient, you take over their compensation. A ventilation rate that would be “normal” for another patient can let CO_2 rise and the pH crash, and the patient can arrest during or shortly after intubation. Avoid taking over the airway unless it is truly needed. If you must, plan ventilation to match the patient’s pre-intervention effort, per protocol and medical direction.
- **Fruity breath** is acetone, exhaled.

Section 4: Potassium: Low in the Body, Often High on the Monitor

This is the high-yield trap. Acidemia and insulin deficiency shift potassium **out of the cells into the blood**, so the serum level is often normal or high. Meanwhile the osmotic diuresis has been dumping potassium in the urine, so **total-body potassium is depleted**. Once insulin (and fluids) start in the hospital, potassium moves back into the cells and serum potassium can drop fast, risking dangerous rhythms. This is why potassium is checked before and during insulin therapy, and why prehospital insulin is not routine.

- **Watch the monitor and get a 12-lead.** AHA 2025 lists hyperkalemia ECG findings as peaked T waves, a flattened P wave, PR lengthening, QRS widening, and a sine-wave pattern. It also notes their sensitivity and specificity are low. Flat T waves and U waves suggest hypokalemia (textbook).
- AHA 2025 Part 10 states that acidemia can temporarily shift potassium extracellularly, which may confuse the cause of an arrest.

Section 5: DKA vs HHS

In freeway terms: **DKA** has no off-ramps, so the houses switch to the fat generator, and acid fills the air. **HHS** has a few off-ramps still open. Enough residual insulin is working to suppress fat breakdown, so there's little ketone acid. But the jam builds slowly over days into extreme gridlock with massive water loss and very high serum osmolality, which is what drives the neurologic picture.

	DKA	HHS
Insulin	Absolute or near-absolute deficiency	Relative deficiency; some insulin present
Typical patient	Type 1 (Type 2 possible)	Type 2, often older with comorbidities
Onset	Hours to 1–2 days	Days to weeks
Acidosis / ketones	Yes (high anion gap)	Minimal or none
Glucose	Elevated (occasionally near normal: euglycemic DKA)	Often markedly higher than in DKA
Volume loss / osmolality	Significant	Often profound; very high osmolality
Mental status	Varies with severity	Altered mental status, focal deficits, seizures, or coma are common
Breathing	Kussmaul	Usually not Kussmaul

Mixed DKA/HHS presentations occur. The hospital confirms the diagnosis with labs. Your job is to recognize the pattern, support perfusion, and protect the airway.

Section 6: Type 1 vs Type 2: Why Each Runs High or Low

- **Type 1:** autoimmune destruction of the beta cells. Without supplemental insulin there are **no off-ramps at all**, which is why DKA is the classic Type 1 emergency.
 - Runs **high** with missed insulin, pump failure, or illness.
 - Runs **low** when the insulin dose outpaces food or activity. Too many off-ramps open and the freeway empties.
- **Type 2:** insulin resistance with progressive beta-cell failure. The off-ramps exist but are narrowed and congested.
 - Runs **high** with illness, steroids, nonadherence, or progression; HHS is the classic Type 2 emergency.
 - Runs **low** with insulin or insulin-releasing oral agents, which can cause prolonged or recurrent lows, especially with kidney impairment. Missed meals and alcohol also contribute.

After-effects: hypoglycemia is the faster brain killer. AHA notes untreated hypoglycemia can cause seizures, status epilepticus, permanent brain injury, and death. Hypoglycemia is also a stroke mimic, so check glucose in every patient with altered mental status. Hyperglycemic crises kill through volume loss, acidosis, and electrolyte shifts. In children, cerebral edema is a feared complication during DKA treatment. Chronic hyperglycemia damages blood vessels and nerves, causing coronary disease, stroke, kidney disease, retinopathy, neuropathy, and atypical or “silent” MI presentations.

Section 7: Prehospital Management (Follow Your Local Protocol and Medical Direction)

- **Assessment:** ABCs, glucose, the cardiac monitor and 12-lead, EtCO₂, and a mental status trend. Check for infection, an insulin pump (note it; follow protocol on whether to suspend or disconnect), and medications.
- **Volume:** isotonic fluid resuscitation is the cornerstone of early DKA/HHS care. Volume, rate, and pediatric limits are protocol-specific and especially conservative in

children.

- **Insulin** is generally an in-hospital therapy, given after potassium is known.
- **Antiemetics and airway** per protocol. Remember the compensation caution in Section 3.
- **Hypoglycemia** (including overcorrection or an insulin-treated patient):
 - Oral glucose if the patient is awake and can swallow. AHA First Aid: ≥ 20 g, tablets preferred.
 - Nothing by mouth if they can't swallow (Class 3: Harm).
 - IV dextrose for patients unable to swallow. AHA calls it potentially lifesaving. Dextrose concentration and dose, and IM/intranasal glucagon if no access, are per your protocol.

Section 8: Dextrose Extravasation: Push It Safely

AHA's 2024 First Aid guideline supports IV dextrose for hypoglycemic patients who can't swallow. It doesn't address extravasation, so what follows is a **standard clinical safety principle**. **D50 is hypertonic and highly irritating to tissue**, and D10 is less so but still irritating. If it extravasates, it can cause tissue injury, including **necrosis**, which in a hand or antecubital space can mean a surgical problem for the patient.

Before the push:

- Confirm a patent, free-flowing line. It flushes easily, with no swelling, resistance, or pain.
- Confirm blood return or free gravity flow.
- Prefer a larger, proximal vein over a small hand vein when you have the choice.
- Secure the catheter, especially in a combative or seizing patient.

During the push:

- Keep your eyes and hand on the site.
- Stop at the first sign of swelling, blanching, coolness, pain or burning, new resistance, or loss of flow.
- A partner can watch the site while you push.

If extravasation happens (per local protocol and medical direction):

- **Stop the infusion immediately.**
- Leave the catheter in place initially per protocol, and **do not flush** the line.
- Elevate the limb.
- Notify the receiving facility, document the site, the estimated volume, and the time, and get new access elsewhere for any remaining dextrose.

Treatments beyond that are a medical-direction decision, not a field improvisation.

Section 9: When DKA Becomes an Arrest: AHA 2025

- **Standard ACLS/PALS comes first:** high-quality CPR, early defibrillation for VF/pVT, and epinephrine 1 mg IV/IO every 3–5 minutes in adults (as soon as feasible for nonshockable rhythms).
- **Reversible causes:** DKA hits several of the classic H's at once: **hypovolemia**, **hydrogen ion (acidosis)**, **hypo-/hyperkalemia**, and potentially hypoglycemia after treatment.
- **Suspected hyperkalemic arrest (Part 10, 2025):** the effectiveness of IV calcium (2b), IV sodium bicarbonate (2b), and IV insulin with glucose (2b) is *not well established*. Inhaled β 2-agonists are not recommended in arrest (Class 3: No Benefit). AHA warns that insulin with glucose risks hypoglycemia. Use them per protocol and medical direction.
- **Routine sodium bicarbonate, calcium, and magnesium** are not recommended in general cardiac arrest (Part 9, 2025). Bicarbonate in suspected hyperkalemia or specific poisonings is addressed separately in Part 10.

Section 10: What Changed From 2020

- **Part 10 (2025)** gives graded recommendations for cardiac arrest from suspected hyperkalemia: calcium, bicarbonate, and insulin/glucose are all 2b (not well established), and inhaled β 2-agonists are 3: No Benefit. It also explicitly notes that acidemia can temporarily shift potassium out of cells, which may confuse the cause of the arrest.

- **First Aid:** hypoglycemia recommendations now come from the 2024 AHA/Red Cross First Aid guidelines, which supersede the 2015 guidelines and the 2019/2020 updates. They include tablets preferred, dietary sugar as an alternative, a 10-minute escalation trigger, and no oral glucose for patients who can't swallow.
- **Part 9 (2025)** reaffirms that routine bicarbonate, calcium, and magnesium are not recommended in arrest.
- **DKA-specific care** isn't addressed in the AHA guidelines in either edition.

Section 11: Putting It Together

Question: A 34-year-old Type 1 diabetic with 3 days of vomiting is lethargic, breathing deep and fast, with EtCO₂ 18 mmHg, heart rate 128, and BP 88/50. Glucose reads 540. The monitor shows peaked T waves. Your partner suggests sedation and intubation for “airway protection.” What's your concern?

Answer: This is DKA with suspected hyperkalemia and shock.

- Prioritize fluids and the monitor, get a 12-lead, and contact medical direction early.
- Avoid taking over the airway unless it's truly needed. If intubation is unavoidable, match the patient's high minute ventilation per protocol.
- If the patient arrests, run standard ACLS, treat the reversible causes, and use hyperkalemia therapies per protocol and Part 10.

Reasoning: No off-ramps means gridlock outside and starving cells inside, so the body burns fat into ketoacids. The low EtCO₂ is his own compensation for that acid. Take it away and his pH falls further, potassium shifts out further, and the peaked T waves can become a sine wave and then an arrest.

Sources (American Heart Association only)

- Part 9: Adult Advanced Life Support: 2025 AHA Guidelines for CPR and ECC. *Circulation*. 2025;152(16 suppl 2):S538–S577. doi:10.1161/CIR.0000000000001376
- 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 8: Pediatric Advanced Life Support: 2025 AHA Guidelines for CPR and ECC. *Circulation*. 2025. doi:10.1161/CIR.0000000000001368
- 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)

Diabetes and DKA/HHS physiology, potassium handling, the airway/ventilation caution, and the dextrose extravasation guidance above are standard clinical teaching, not AHA recommendations. The freeway analogy is Forrest Munden's teaching model.