



# The Internet Book of Critical Care

## Anatomy of a DKA resuscitation

November 6, 2016 by [Josh Farkas](#)



(<https://i0.wp.com/emcrit.org/wp-content/uploads/2016/11/Mind-the-gap4.jpg>)

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### definition & diagnosis of DKA

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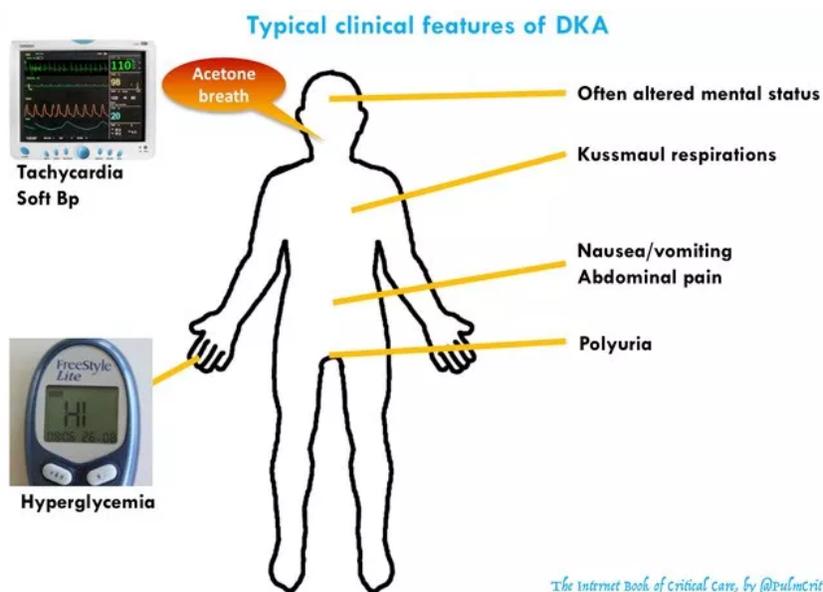
**Anion Gap** = Na – Cl – Bicarbonate

(<https://i2.wp.com/emcrit.org/wp-content/uploads/2016/11/ag4.jpg>) **defining anion gap**

- Using this formula, an elevated anion gap is above 10-12 mEq/L.<sup>1</sup>
- Please don't correct for albumin, glucose, or potassium. Don't make this unnecessarily complicated.<sup>2</sup>

### gold-standard definition of DKA?

- Many definitions of DKA may be found in the literature, most of which are antiquated. According to the Canadian DKA guidelines, "there are no definitive criteria for the diagnosis of DKA."<sup>3</sup>
- My preferred definition: any patient with diabetes plus a significantly elevated serum beta-hydroxybutyrate level (above 2-3 mM/L).<sup>4 5</sup>
- Please note the following:
  1. DKA patients can have a *normal* glucose (euglycemic DKA, more on this [below](#) (#euglycemic\_DKA)).
  2. DKA patients can have a *normal* pH and a normal bicarbonate. This usually occurs due to a combination of ketoacidosis plus metabolic alkalosis from vomiting.
  3. That's right: DKA patients can have a totally stone-cold normal ABG.



(<https://i1.wp.com/emcrit.org/wp-content/uploads/2016/11/typicaldka.jpg>) **various ways to diagnose DKA**

- **Obvious DKA:**
  - Patient has known diabetes.
  - Anion gap is  $\gg 12$  mEq/L with positive urinary ketones.
  - History & physical exam are consistent with DKA (figure above) and don't suggest that anything else is going on
- **Non-obvious DKA:** In situations where the diagnosis is unclear, check lactate and beta-hydroxybutyrate levels.
  - A significantly elevated beta-hydroxybutyrate level strongly supports the diagnosis of DKA.<sup>6</sup>
  - If the patient has a *markedly* elevated anion gap with only a *mildly* elevated beta-hydroxybutyrate level, consider the possibility that something else is going on (e.g. mild DKA plus toxic alcohol poisoning).

### ABG/VBG is neither required nor particularly helpful

- If you look throughout this chapter, neither pH nor pCO<sub>2</sub> are mentioned much. These aren't required for the diagnosis or management of DKA.
- As explored above, DKA is diagnosed purely on the basis of venous blood chemistries (chem-7, anion gap, and if necessary beta-hydroxybutyrate).
- The vast majority of DKA patients can be diagnosed and managed perfectly without ever checking an ABG or VBG.<sup>7</sup>

## step 1- evaluation

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## precipitating cause

DKA is occasionally the initial manifestation of diabetes, but it usually occurs in the context of known diabetes plus a trigger. Most triggers of DKA are benign (e.g. noncompliance, viral gastroenteritis). However, DKA can be caused by *any* source of physiologic stress. Occasionally, DKA is the presentation of a serious underlying problem, such as occult sepsis. Common causes of DKA include:

- Insulin non-adherence, inadequate dosing, or insulin pump failure
- Infection (e.g. gastroenteritis, pneumonia, urinary tract infection, diabetic foot infection)
- Pancreatitis
- Pregnancy
- Trauma, surgery
- Substance abuse or alcoholism
- Medications (e.g. steroid, atypical antipsychotics, sympathomimetics, SGLT-2 inhibitors, HIV protease inhibitors, anti-calcineurin immunosuppressives<sup>8</sup>)

## evaluation for the cause of DKA

- History and physical examination are the key here. If there is clear history of non-adherence, a big workup isn't necessary.
- **Infectious trigger?**
  - DKA *itself* may cause leukocytosis, so a WBC elevation alone is nonspecific.
  - Infection is suggested by fever, bandemia (>10%), marked left-shift, or severe leukocytosis (>20,000-25,000).<sup>9 10</sup>
- **Primary abdominal problem?**
  - DKA *itself* can cause abdominal pain. This creates diagnosis confusion – we must sort out whether the pain is due to DKA, or whether the pain represents an underlying problem (appendicitis, cholecystitis, etc). This may be sorted out in two ways:
    - (1) Severe pain with only mild ketoacidosis argues against DKA causing the pain.<sup>11</sup>
    - (2) When in doubt about the need for an abdominal CT scan, aggressively treat the DKA and follow serial abdominal examinations. If the abdominal pain is due to DKA, it will resolve as the ketoacidosis improves. If pain fails to resolve or gets worse, then further investigation is warranted.
- **Primary neurologic problem?**
  - DKA *itself* cause mental status changes, but this usually occurs when the [calculated serum osmolality](https://www.mdcalc.com/serum-osmolality-osmolarity) (<https://www.mdcalc.com/serum-osmolality-osmolarity>) is >320 mOsm/kg. Abnormal mental status despite normal serum osmolality should trigger suspicion for a primary neurologic problem (e.g. meningitis, intracranial hemorrhage).<sup>12</sup>
  - Another sign of a primary neurologic problem is if the mental status doesn't improve with treatment of the DKA.<sup>13</sup>

## initial evaluation panel (<https://i2.wp.com/emcrit.org/wp-content/uploads/2016/12/checklist3.gif>)

- EKG
- Electrolytes (including Ca/Mg/Phos), blood count
- If diagnosis of DKA is unclear: beta-hydroxybutyrate level & lactate
- If infection possible: blood cultures, urinalysis, chest X-ray
- If pregnancy is possible: urine pregnancy test or serum beta-HCG
- If significant abdominal pain/tenderness: lipase (note, however, that DKA itself can increase lipase substantially)<sup>14</sup>
- Troponin level *only* if EKG or history suggests ischemia (i.e., if you are genuinely concerned about MI).
  - Routinely checking troponin on every DKA patient is a common cause of mass hysteria and unnecessary evaluations.
- Additional workup as clinically appropriate (e.g. toxicology evaluation, CT scan to evaluate for septic focus).



## step 2- initial resuscitation

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## IV access

- The vast majority of patients with DKA can be treated with peripheral IV access, but for the sickest patients central access may be needed.
- Femoral site is often best for DKA patients:
  - In a severely acidotic patient with respiratory compensation, pneumothorax would be poorly tolerated.

- Patients may be delirious and unable to stay still enough to facilitate safe placement of a jugular/subclavian line.
- The line will only be needed for 24-48 hours (until DKA resolves), so infection risk is minimal.

### insulin infusion: getting started

- Unless the patient is hypokalemic, insulin infusion should be started immediately.
  - For hypokalemia, hold insulin until potassium level is raised. The maximal infusion rate of potassium is often regarded as 20 mEq/hr, but in hypokalemic DKA giving 40 mEq/hr is reasonable with careful monitoring.<sup>3 15 16</sup> To avoid damaging the veins this may be given via a central line or multiple peripheral lines (e.g. 20 mEq/hr simultaneously through two peripheral IVs).
- Insulin bolus (10 units IV) should be considered if setting up the infusion will take >30 minutes. The main advantage of an insulin bolus is that this can usually be given *immediately* (most units have 10-unit insulin vials immediately available, whereas an insulin infusion needs to be mixed up in pharmacy).
- Insulin infusion is usually started at 0.1 U/kg/hour (up to a max of 15 units/hour in morbid obesity). However, for patients with severe acidosis (e.g. bicarbonate <5 mEq/L) or marked insulin resistance (with high chronic insulin requirements), higher doses will often be needed (e.g. 0.2-0.3 U/kg/hr).
  - The insulin infusion should be up-titrated as needed to drop the glucose by 50-70 mg/dL (2.8-3.9 mM) per hour.

### potassium

- DKA resuscitation will cause the potassium to drop like a stone.
- If the patient is hyperkalemic, then this should resolve rapidly. For critical hyperkalemia IV calcium may be considered, but the real key here is *IV insulin*.
  - Critical hyperkalemia is an indication for an immediate 10 unit IV insulin bolus.
- Aggressive potassium repletion is generally needed. Shoot for a potassium >5.3 mM (targeting a high potassium prevents you from falling behind).

### fluid resuscitation

- DKA patients are often profoundly volume depleted (e.g. due to vomiting, reduced PO intake, and osmotic diuresis). Hypovolemia triggers the release of stress hormones (e.g. catecholamines, cortisol) which cause insulin resistance and thereby make it *harder* to treat the DKA. Thus, prompt reversal of hypovolemia is one of the key treatments for DKA.
- Most patients will require ~2-4 liters of crystalloid up front.
  - For young DKA patients with normal cardiorenal function, if the patient's heart rate is >100 b/m then they probably need more fluid.
  - Ultrasound-guided fluid resuscitation is useful for patients with heart failure or patients on hemodialysis.
- Balanced crystalloid is preferred here (e.g. LR or plasmalyte).
  - Normal saline will worsen (<https://emcrit.org/pulmcrit/four-dka-pearls/#saline>), the patient's acidosis.

### management of severe acidosis<sup>17</sup>

- Scary acidosis may be defined in various ways (e.g. pH<6.9 or bicarb <5 mM). Patients generally tolerate this surprisingly well.
- Avoid giving bicarbonate here for management of acidosis. If you're worried about the acidosis, the most effective strategy is to increase the *insulin dose* (usually along with administration of additional glucose and potassium).
  - a) Don't wait for the insulin to arrive from pharmacy: bolus 10 units IV immediately.
  - b) Consider starting at 0.2 U/kg/hr in the sickest patients.

### high-flow nasal cannula (HFNC) to support work of breathing

- Intubation should be avoided (<https://emcrit.org/pulmcrit/four-dka-pearls/#tube>) if at all possible (more on this [here \(#intubating\\_if\\_you\\_must\)](#)). BiPAP should also be avoided, because DKA patients often have gastroparesis causing emesis into the mask.
- High-flow nasal cannula is a safe way to support the patient's breathing. By facilitating CO<sub>2</sub> elimination, HFNC can help the patient compensate for their metabolic acidosis.
- HFNC should be set as follows:
  - FiO<sub>2</sub> titrated to achieve a saturation >92% (usually a low FiO<sub>2</sub> will be needed e.g. 30-40%)
  - Increase the flow rate as high as the patient can tolerate (e.g. 60 liters/minute). The *flow rate* is what does the work of reducing dead space and thereby blowing off CO<sub>2</sub>. If the patient is very sick and air-hungry, they will tolerate high flow rates.
- Indications for HFNC may include: increased work of breathing, severe acidosis (e.g. bicarbonate <5 mM).<sup>18</sup>

## step 3- closing the gap

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### things to follow

- Glucose Q1hr
- Extended electrolytes (including Phos & Mg) Q2-Q4 hrs
- Urine output. Glucose >250 mg/dL (>14 mM) functions as a diuretic, so patients *should* produce lots of urine. Poor urine output raises concerns about shock or renal failure.

### start long-acting insulin

- Long-acting insulin should be started *early* (well in advance of discontinuing the infusion). Glargine has a delayed onset compared to some older forms of insulin (e.g. NPH), so the traditional two-hour overlap may not work well with glargine.<sup>19</sup> Early initiation of long-acting insulin facilitates transitioning off the insulin infusion, reduces the incidence of hyperglycemia, and might decrease hospital length of stay.<sup>20</sup>
- Patients can generally be treated with their home insulin regimen (ideally a single daily dose of glargine). For a patient naive to insulin, a starting dose of 0.25 units/kg daily of glargine (Lantus) may be given.
- Common pitfalls with long-acting insulin:
  1. Practitioners who are nervous about giving early glargine may sometimes give a reduced dose, which leads to tremendous confusion.
  2. Some patients are on twice daily glargine (for reasons which aren't entirely clear to me). If such patients are continued on twice daily glargine, the insulin infusion shouldn't be stopped until after they receive their *second* dose of glargine. Alternatively, both doses can be compiled into a single daily dose.<sup>21</sup>
  3. Glargine should be ordered "q24 hours," not "daily." If the glargine is electronically ordered as "daily" then it may default to every morning at 9 AM – which will cause some patients to receive their daily dose in the evening, and then *another* dose the following morning.

### crystalloid infusion

- After bolusing the patient to a point of euvoemia, start a maintenance fluid infusion.
- If the patient's glucose is >300 mg/dL (>16.6 mM), a good choice is often LR at ~200 ml/hr.
- Once the glucose falls <300 mg/dL (<16.6 mM), IV glucose should be added. One strategy to achieve this is to *drop and split*:
  - Cut the LR rate in half (e.g. from 200 ml/hr to 100 ml/hr)
  - Add a D10w infusion at an equal rate (e.g. 100 ml/hr LR plus 100 ml/hr D10W).<sup>22</sup>
  - Combining LR with an equal volume of D10W effectively creates a solution of "D5 1/2 LR" (a solution which doesn't exist in pre-mixed bags). The two fluids are compatible and can be given through a single IV line. The advantage of giving the components separately is that it provides you more control with regards to adjusting the amount of sodium you are giving versus the amount of dextrose. For example, if you want to give additional dextrose you can up-titrate the D10W infusion (without giving the patient more sodium and causing volume overload).
- An alternative approach is to switch to D5 1/2 NS at ~200 ml/hr.

### keep repleting the lytes....

- **Potassium:**
  - As above, replete aggressively to shoot for  $K > 5.3$  mM. This will often involve repeated doses of IV potassium.
  - Oral potassium can be used, but patients are often nauseous and unable to tolerate this.
  - In renal failure, be more conservative with potassium repletion.
- **Phosphate**
  - Phosphate will drop during treatment, especially in patients with severe DKA.
  - Follow the phosphate and replete if severe hypophosphatemia occurs ( $< 1$  mg/dL or  $< 0.32$  mM).
- **Magnesium**
  - Maintaining a high-normal magnesium level may tend to protect against hypokalemia-induced arrhythmia if the potassium falls too low (isolated hypokalemia is usually well tolerated, whereas the *combination* of hypokalemia plus hypomagnesemia is more dangerous).

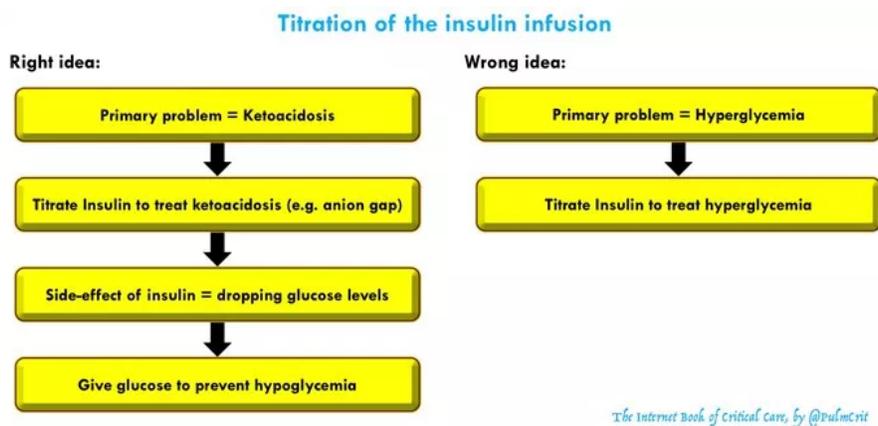
### make sure the anion gap is closing

- If the anion gap isn't closing, consider the following possibilities:

- Inadequate fluid resuscitation.
- Inadequately low insulin dose.
- Malfunction of insulin infusion.
- Festering, underlying problem which hasn't been addressed.
- Interventions if the anion gap isn't closing:
  - Evaluate fluid status (e.g. with ultrasonography), provide additional crystalloid if necessary.
  - Consider increasing the insulin infusion rate (see next section).
  - Re-evaluate for a missed underlying problem (see [Step #1 \(#step\\_1- evaluation\)](#)).
  - Consider checking beta-hydroxybutyrate & lactate levels, to exclude an occult/worsening *lactic* acidosis.

### insulin titration

- Your hospital should have a DKA-protocol insulin infusion. Make sure to order the DKA protocol. Using the *non-DKA protocol* may cause the insulin to be shut off prematurely.
- DKA insulin protocols generally work fairly well and don't require a lot of adjustment. So you can often ignore this. However, if things are going sideways then consider the following:
  - The primary problem with DKA is *ketoacidosis* (not hyperglycemia). Therefore, our overall goal is to titrate insulin as needed to treat the ketoacidosis (figure below). Unfortunately, it's a bit more complicated than this. Glucose levels are easier to repeat than measurements of anion gap. Thus, glucose levels are often used as a *surrogate measurement* of the biological efficacy of insulin (for example, during the initial phase of resuscitation, if the glucose level isn't falling that indicates that insulin isn't working and should be up-titrated).
  - Occasionally, if the patient's anion gap isn't clearing, you might need to simultaneously increase *both* the insulin infusion rate *and* the glucose infusion rate. However, once the glucose falls to ~250 mg/dL (14 mM) the insulin infusion rate is typically reduced considerably.



<https://i2.wp.com/emcrit.org/wp-content/uploads/2016/11/titratett.jpg>

### treat non-anion gap metabolic acidosis (NAGMA) with bicarb

$$\text{Predicted Final Bicarb} = (\text{Na} - \text{Cl} - 10)$$

<https://i1.wp.com/emcrit.org/wp-content/uploads/2016/11/pfb.jpg>

- NAGMA commonly develops at this phase, caused by two factors:
  - (a) Resuscitation with normal saline or half-normal saline
  - (b) Excretion of ketoacid in the urine (once ketoacid is in the sewer system, it can no longer be converted back into bicarbonate)
- Development of NAGMA may be revealed by the following:
  - a) The anion gap is closing but the patient's bicarbonate remains low
  - b) The predicted final bicarbonate (equation above) is falling  $\ll 20$  mEq/L. The predicted final bicarbonate is an estimate of where the bicarbonate will end up after all the ketoacid is converted into bicarbonate. This doesn't work perfectly, but it can be a rough estimate of whether there is significant NAGMA.

- The best time to treat NAGMA is as the anion gap is *beginning* to close (e.g. when the anion gap is ~12-18 mEq/L). This facilitates prompt discontinuation of the insulin infusion, without delay in order to treat the NAGMA.
- NAGMA should be treated with IV bicarbonate to achieve a bicarbonate level above 18-20 mEq/L prior to discontinuing the insulin infusion. Acidosis causes insulin resistance, so treating NAGMA will facilitate transitioning off the insulin infusion without recurrent DKA (more on this [here](https://emcrit.org/pulmcrit/bicarbonate-dka/#nagma) (<https://emcrit.org/pulmcrit/bicarbonate-dka/#nagma>)).
  - The bicarbonate deficit can be estimated using [this formula](https://www.mdcalc.com/bicarbonate-deficit) (<https://www.mdcalc.com/bicarbonate-deficit>). While the anion gap is still open, use the *predicted final bicarbonate* to get a rough concept of the bicarbonate deficit. Keep in mind, however, that you're only shooting for a bicarbonate of ~20 mEq/L (not 24 mEq/L). 100-150 mEq of bicarbonate is usually adequate.
  - If the patient is hyponatremic, then a couple of hypertonic bicarbonate ampules can be used (each ampule contains 50 mEq sodium bicarbonate in 50 ml water).
  - If the patient's sodium is normal or elevated, then isotonic bicarbonate may be used (e.g. one liter of D5W with three ampules of bicarbonate, to generate a 150 mEq/L bicarbonate solution, infused over 3-4 hours). This will cause the glucose to increase a bit, but that can actually be useful in closing the anion gap (because it will trigger an increase in the insulin infusion).

### DVT prophylaxis

- DKA patients are at relatively high risk of venous thromboembolic disease, comparable to other cohorts of critically ill patients.<sup>23</sup>
- DVT prophylaxis should be provided unless contraindicated.

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## step 4- stopping the drip

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### don't stop the drip until the following criteria are met:

- 1) Resolution of ketoacidosis (anion gap < 10-12 mEq/L).
  - An exception here is a patient with end-stage renal disease, who may chronically have an elevated anion gap due to uremia which never normalizes. In this situation, normalization of the *beta-hydroxybutyrate* level (<0.6 mM) is a more useful way to determine that ketoacidosis has resolved.
- 2) The patient isn't significantly acidotic (bicarbonate > 18-20 mEq/L).
  - If the patient has developed NAGMA then treat with IV bicarbonate as described [above](#) ([#nagma](#)).
- 3) The patient has received the full daily dose of long-acting insulin >2 hours previously.
- 4) Glucose is reasonably well controlled (e.g., <250 mg/dL or <14 mM)
- 5) The patient should *ideally* be hungry (this is an excellent sign suggesting that the ketoacidosis has resolved).
  - If the insulin infusion is stopped and the patient doesn't eat anything or receive any IV glucose, this increases the risk of recurrent DKA.
  - An exception can be made for patients with gastroenteritis or diabetic gastroparesis, who may not be hungry for a while. In this situation, the insulin infusion can be stopped, but patients should remain on low-dose intravenous glucose (e.g. D5W at 75 ml/hr). If the patient's glucose level increases, they should be treated with PRN short-acting insulin. Ongoing administration of carbohydrate plus PRN insulin will help keep the anion gap closed.

### start meal-associated & PRN insulin

- Start meal-associated and sliding-scale insulin.
  - If the patient isn't already on a prescribed regimen of meal-associated insulin, a dose of ~0.08 U/kg rapid-acting insulin per meal may be reasonable. Follow glucose carefully and titrate to effect.
- Encourage patients to eat. Carbohydrate intake (along with meal-associated and sliding-scale insulin) is important at this step to prevent recurrent DKA.

### monitor for recurrence of DKA

- (1) Follow the glucose level.
  - Development of progressively severe hyperglycemia may be an early sign of recurrent DKA.
  - Since glucose levels are often checked frequently, skyrocketing glucose may pre-date the development of a widening anion gap by a few hours.
- (2) Consider repeat electrolytes

- A set of electrolytes ~6 hours after stopping the drip is a reasonable idea to make sure that the anion gap is remaining closed (if there is any doubt about this clinically).

### management of recurrent DKA

- Causes of recurrent DKA (anion gap re-opens after stopping drip)
  - Insulin infusion was stopped despite not meeting all five of the criteria above.
  - Inadequate long-acting insulin dose.
  - Patient isn't eating enough (which causes insufficient meal-associated & PRN insulin doses)
  - Ongoing systemic inflammation (e.g. DKA caused by infection, with persistent infection).
- Treatment:
  - Restart the insulin infusion.
  - Continue long-acting insulin (consider up-titrating the dose).
  - Address any reversible causes of DKA.
  - Aggressively [treat NAGMA \(#nagma\)](#) to get the serum bicarb >20 mEq/L (this will improve insulin sensitivity).
  - Sometimes patients just need a bit longer on the insulin infusion (especially if they were severely ill on admission).

## DKA in the hemodialysis patient

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### differences compared to the usual DKA resuscitation

- Patients generally aren't severely volume depleted (because they never develop polyuria).
- If you give excessive fluid this will create a persistent problem, requiring dialysis.
- Potassium repletion should be less aggressive, because patients cannot excrete excess potassium.
- Many patients really just need *insulin* (without a lot of fluid or potassium).

### role of hemodialysis

- Hemodialysis will remove ketoacid, replace bicarbonate, and basically fix everything.
- The risk of hemodialysis is that it may cause rapid osmotic shifts. For patients with severe hypertonicity (serum osmolality >>330 mOsm/kg), this could carry a risk of causing cerebral edema.
- Even if hemodialysis fixes everything, don't forget the insulin – the patient still needs insulin to prevent slipping back into DKA.

## DKA in the heart failure patient

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### differences

1. Heart failure patients may be hypervolemic prior to developing DKA. Furthermore they may respond less strongly to the diuretic effect of hyperglycemia. Overall, the patient may not be severely hypovolemic at baseline.
2. If excessive fluid is administered, heart failure patients will tend to retain this fluid (unlike young DKA patients, who will eliminate any excess fluid via urination).

### management

- Serial assessment of volume status is needed (e.g. with ultrasonography).
- Standard DKA protocols may not work. Specifically, these protocols will recommend excessive fluid administration.

## avoiding cerebral edema

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### who is at risk

- Younger patients (almost all affected are <25 YO)

- Patients with marked baseline hyperosmolality (e.g. [calculated serum osmolality](https://www.mdcalc.com/serum-osmolality-osmolality))  $> \sim 330$  mOsm).
  - Among older adults this is mostly an issue among patients with a hyperosmolar hyperglycemic state (HHS), rather than DKA. However, there can be some overlap between HHS and DKA, so it's worth considering the risk of cerebral edema in patients with marked hyperosmolality.

#### how to avoid cerebral edema:

- Avoid over-aggressive fluid administration.
- Don't drop the glucose too fast. Avoid reducing the glucose below  $< 200$  mg/dL ( $< 11.1$  mM).
- Replace water gradually (slower correction is safer, there's no rush here).
  - Try to use only isotonic fluids (e.g. D5 LR can be used as a source of glucose-containing IV fluid, rather than hypotonic fluids such as D10W or D5 1/2 NS).
  - Avoid dropping the serum osmolality by more than 3 mOsm/kg/hour.<sup>3</sup>
- Note that the sodium will often initially *increase* during resuscitation due to glucose entering the cells. This *doesn't* reflect an increase in serum osmolality, and *doesn't* require treatment with free water.
  - The key parameter to track is the measured or [estimated](https://www.mdcalc.com/serum-osmolality-osmolality) serum osmolality – not the sodium.

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## intubating, if you must

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#### avoid intubation

- Whenever possible, [avoid intubation](https://emcrit.org/pulmcrit/four-dka-pearls/#tube).
- Intubating a patient due to altered mental status is usually a mistake. The mental status should improve over several hours, so careful observation is generally the best approach.
- Indications for intubation may include:
  - Frank inability to protect airway (e.g. gurgling, inability to control secretions).
  - Intubation needed to facilitate surgical procedure (e.g. patient has DKA plus perforated viscus).
  - Respiratory arrest or impending arrest (e.g. patient in extremis).
- If intubation *is* necessary (e.g. for a surgical procedure), it may be wise to delay it for a few hours to allow vigorous treatment of DKA first.

#### risks involved with intubation

1. Hemodynamic collapse: If hypovolemia isn't corrected prior to intubation
2. Vomiting/aspiration: These folks often have gastroparesis and ileus.
3. Decompensation of acidosis: Most patients have severe metabolic acidosis with a compensatory respiratory alkalosis. Paralysis takes away their respiratory compensation, potentially leading to profound acidosis.

#### mitigating the risks

- Hemodynamic collapse:
  - Volume resuscitate prior to intubation.
  - If necessary start a vasopressor infusion to establish MAP  $> 75$ - $80$  mm before the procedure.
  - Use hemodynamically stable induction drugs (e.g. ketamine).
- Avoid regurgitation
  - Visualize the stomach with [ultrasound](https://emcrit.org/pulmcrit/drowned-airway-algorithm/#sono), if it's distended consider NG drainage prior to intubation.
  - If the patient is intermittently vomiting, encourage them to vomit immediately before anesthetic induction (while they can still protect their airway).
- Consider giving some bicarbonate prior to intubation if the bicarbonate level is  $< 10$  mEq/L.
  - For example, *slowly* push 2-3 ampules (100-150 mEq) of bicarbonate over 10-15 minutes, at least  $\sim 10$  minutes prior to intubation.

- Bicarbonate contains dissolved CO<sub>2</sub>, which the patient must blow off. In order to benefit from the bicarbonate, the patient should have enough time to blow off additional CO<sub>2</sub> *prior* to intubation.<sup>24</sup>
- Consider using mechanically controlled *apneic ventilation* (with [BiPAP](https://emcrit.org/pulmcrit/apneic-ventilation-using-pressure-limited-ventilation/) (<https://emcrit.org/pulmcrit/apneic-ventilation-using-pressure-limited-ventilation/>), or a [ventilator](https://emcrit.org/emcrit/emcrit-wee-vapox/) (<https://emcrit.org/emcrit/emcrit-wee-vapox/>)) during induction of anesthesia if you're adept at this. If you're not, then it's probably best to perform pure RSI to minimize risk of regurgitation (without any breaths interposed between paralytic and intubation).
- Use a relatively large ETT to minimize airway resistance (ideally nothing smaller than a 7.5-mm ETT).
- Use rocuronium, so that after intubation the patient will be paralyzed and sync perfectly with the ventilator.<sup>25</sup>
- As soon as the ETT is secured, increase tidal volume & respiratory rate to hyperventilate the patient (thus restoring respiratory compensation).
  - Set the tidal volume at 8 cc/kg.
  - Crank the respiratory rate as high as possible without causing autoPEEP (will often end up around ~24-28 breaths/minute).
  - Shoot for a very high minute ventilation (e.g. 12-18 liters/minute).

## euglycemic DKA

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### definition & diagnosis

- **Definition:** Euglycemic DKA is defined as DKA with a glucose <250 mg/dL (<13.9 mM).<sup>26</sup>
- **Diagnosis:** This can present in an atypical fashion. Remember to consider the possibility of DKA whenever the anion gap is elevated or ketones are present in the urine (even if the glucose is normal).

### causes

- SGLT2 inhibitors (empagliflozin, canagliflozin, dapagliflozin)
- Anything that exhausts the liver's ability to synthesize glucose:
  - Starvation, prolonged nausea/vomiting
  - Pregnancy
- Partial treatment with insulin before admission (either intentionally or unintentionally via an insulin pump)

### treatment

- Overall similar to usual treatment described above.
- IV glucose will need to be started immediately (e.g. D10W or D5LR infusion). These patients will require a combination of both IV glucose plus IV insulin to resolve their ketoacidosis.

## with an insulin pump

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Most of the time when these patients present there won't be an insulin pump guru available. The following approach describes a safe strategy for folks without high-level knowledge of insulin pumps (e.g. 98% of practitioners).

- First, disconnect the insulin pump (including removal of the needle from the skin). It's unclear whether the pump is working. The safest approach is to remove this variable from the equation until the patient is stabilized.
- Initial evaluation & resuscitation (steps #1-2) are exactly the same as usual.
- In step #3 above, give the patient a dose of glargine which is equal to the 24-hour basal insulin provided via the pump.
  - The basal rate can usually be obtained from notes, or the patient will know their basal rate. Multiply this out times 24 hours to obtain the total daily basal requirement.
- Proceed with the remainder of DKA therapy per usual.
- If the patient and their endocrinologist decide to resume pump therapy, the transition from glargine back onto the pump can be made at a later date.

## checklist

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## DKA resuscitation: Getting started

- **Diagnostic evaluation**
  - EKG, Electrolytes (including Ca/Mg/Phos), CBC
  - If diagnosis of DKA is unclear: beta-hydroxybutyrate level & lactate
  - If cause of DKA is unclear: blood cultures, urinalysis, CXR
  - If significant abdominal pain: lipase
  - If pregnancy possible: urine pregnancy test or serum beta-HCG
  - Troponin level ONLY if you truly suspect ischemia based on history & EKG review
- **Insulin infusion**
  - Hold if  $K < 3.3$ , otherwise start 0.1 U/kg/hr
  - For sick patient if infusion isn't immediately available at bedside, bolus 10 units IV insulin.
  - Sicker patients may need higher insulin infusion rates (0.2-0.3 U/kg/hr).
- **Crystalloid**
  1. Bolus with lactated ringers until euvoletic (based on echo, heart rate resolution to  $< 100$  b/m).
  2. Infuse LR ~200 ml/hr until glucose  $< 300$ mg/dL ( $< 16.6$  mM)
  3. Infuse dextrose-containing solution after glucose  $< 300$ mg/dL (e.g. D10W plus LR, both at 100 ml/hr)
- **Electrolyte repletion**
  - Aggressive repletion of potassium (target  $K > 5.3$  mEq/L if patient has normal renal function)
  - Magnesium repletion if needed
  - Follow phosphate, replete if severe hypophosphatemia.
- **High-flow nasal cannula** if patient has elevated work of breathing or severe acidosis (Bicarb  $< 5$  mEq/L)
- **Scary Acidosis?**
  - Do not give bicarb in the initial resuscitation phase for management of scary acidosis.
  - If you're worried about the severity of the acidosis, give more insulin / dextrose / potassium.

*The Internet Book of Critical Care, by @pulmcrit*<https://i0.wp.com/emcrit.org/wp-content/uploads/2016/11/dkachk2.jpg>

## IBCC podcast on DKA

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<https://i1.wp.com/emcrit.org/wp-content/uploads/2016/11/apps.40518.14127333176902609.7be7b901-15fe-4c27-863c-7c0dbfc26c5c.5c278f58-912b-4af9-88f8-a65fff2da477.jpg> Follow us on iTunes (<https://itunes.apple.com/ca/podcast/the-internet-book-of-critical-care-podcast/id1435679111>)

### The Podcast Episode

00:00

00:00

[\(view transcript\)](#)

Want to Download the Episode?

[Right Click Here and Choose Save-As](http://traffic.libsyn.com/ibccpodcast/IBCC_EP1_DKA_final.mp3) ([http://traffic.libsyn.com/ibccpodcast/IBCC\\_EP1\\_DKA\\_final.mp3](http://traffic.libsyn.com/ibccpodcast/IBCC_EP1_DKA_final.mp3))

## questions & discussion

[\(back to contents\)](#),[\(#top\)](#)To keep this page small and fast, questions & discussion about this post can be found on another page [here](https://emcrit.org/pulmcrit/dka/) (<https://emcrit.org/pulmcrit/dka/>).

(<https://i1.wp.com/emcrit.org/wp-content/uploads/2016/11/pitfalls2.gif>)

- Missing an [underlying cause \(#step\\_1- evaluation\)](#) of DKA, especially sepsis. The primary cause of death among patients admitted with DKA isn't the DKA itself, but rather associated conditions.
- Bolusing large volumes of normal saline will make the patient *more acidotic*. This is generally not a major problem, but it can be for the sickest patients who present with severe acidosis.
- Avoid intubation of DKA patient if possible (it's a trap!). If you do need to intubate, proceed with extreme caution & [preparation \(#intubating\\_if\\_you\\_must\)](#).
- BiPAP should be avoided as well, as patients will often vomit. To provide some additional respiratory support, consider [high-flow nasal cannula \(http://<section class="ibanchor" id=""></section>\)](#).
- Please don't measure a troponin on every DKA patient (older DKA patients will usually have a measurable troponin level, which may trigger unnecessary and harmful workups). Be a doctor. Check a troponin if you are genuinely concerned about ischemia, based on symptoms and EKG evaluation.
- Don't stop the insulin infusion until the patient [meets criteria \(#step\\_4- stopping\\_the\\_drip\)](#) to do so.
- Don't exclude the diagnosis of DKA because a patient has normal glucose, or normal bicarb/pH. Remember, instead, to *mind the gap*.

### Going further:

#### FOAMed

- PulmCrit DKA series
  - [DKA I: the pearls \(https://emcrit.org/pulmcrit/four-dka-pearls/\)](#)
  - [DKA II: Dominating the acidosis \(https://emcrit.org/pulmcrit/bicarbonate-dka/\)](#)
  - [Why ABG & VBG are unhelpful in DKA \(https://emcrit.org/pulmcrit/blood-gas-measurements-dka-searching-unicorn/\)](#)
- Euglycemic DKA
  - [REBEL EM \(http://rebelem.com/euglycemic-dka-not-myth/\)](#): Euglycemic DKA isn't a myth
  - [EMDOCS \(http://www.emdocs.net/euglycemic-dka-secondary-sglit2-inhibitors/\)](#): Euglycemic DKA secondary to SGLT2 inhibitors
- Bicarb in DKA? See Chris Nickson on [LITFL \(http://lifeinthefastlane.com/ccs/sodium-bicarbonate-and-diabetic-ketoacidosis/\)](#), Anand Swaminathan on [EMDOCS \(http://www.emdocs.net/myths-dka-management/\)](#), Salim Rezaei on [RebeLEM \(http://rebelem.com/diabetic-ketoacidosis-dka-myths/\)](#), Darrel Hughes on [RebeLEM \(http://rebelem.com/benefit-sodium-bicarbonate-dka/\)](#).
- Insulin bolus? Darrel Hughes on [RebeLEM \(http://rebelem.com/benefit-initial-insulin-bolus-diabetic-ketoacidosis/\)](#)
- [DKA myths \(http://rebelem.com/diabetic-ketoacidosis-dka-myths/\)](#) by RebeLEM, and also by Anand Swaminathan on [EMDOCS \(http://www.emdocs.net/myths-dka-management/\)](#).

#### Guidelines

- Canadian 2013 guidelines.<sup>3</sup>
- [British guidelines \(2013 update\) \(https://www.diabetes.org.uk/resources-s3/2017-09/Management-of-DKA-241013.pdf\)](#) – perhaps the best guideline available currently.
- American Diabetic Association 2009 guidelines – rather outdated.<sup>27</sup>

Image credits: [Gap \(https://commons.wikimedia.org/wiki/File:Mind-the-gap.jpg\)](https://commons.wikimedia.org/wiki/File:Mind-the-gap.jpg)

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2. Farkas J. PulmCrit. Correcting the anion gap for albumin is not helpful. <https://emcrit.org/pulmcrit/mythbusting-correcting-the-anion-gap-for-albumin-is-not-helpful/> (<https://emcrit.org/pulmcrit/mythbusting-correcting-the-anion-gap-for-albumin-is-not-helpful/>).
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6. Among non-diabetics, ketoacidosis can be caused by starvation or alcoholism. In the context of diabetes, ketoacidosis is basically diagnostic of DKA.
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10. Bandemia is the best predictor in this study, but different hospitals may have difference performance regarding the definition of band forms. Get acquainted with your lab's performance for detecting left-shift and pay attention to it (e.g. some labs tend to report bandemia, others report more metamyelocytes etc).
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18. I'm not aware of any high-quality evidence on this, but it seems to work and HFNC is extremely safe. .
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21. If the glargine is given at an inopportune time (e.g. it's given in the evening and the patient prefers taking it in the morning), the timing can be slowly shifted by an hour each day. .
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23. Keenan C, Murin S, White R. High risk for venous thromboembolism in diabetics with hyperosmolar state: comparison with other acute medical illnesses. *J Thromb Haemost*. 2007;5(6):1185-1190. [PubMed (<https://www.ncbi.nlm.nih.gov/pubmed/17403099>)]
24. Even if the patient is already breathing as hard as they can, giving amps of bicarb will increase the pCO2 and thus increase the \*gradient\* driving CO2 excretion from the body. So the CO2 administered with the bicarb won't be "trapped" in the patient's body.
25. If you're extremely adept at vent management, there are some theoretical advantages to using succinylcholine and then using a pressure-cycled vent strategy (this allows for active exhalation).
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The Internet Book of Critical Care is an online textbook written by Josh Farkas (@PulmCrit), an associate professor of Pulmonary and Critical Care Medicine at the University of Vermont.