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Diabetic Ketoacidosis in the Pediatric Patient

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Children's Hospital Colorado
Here, it's different.™

Disclosures

- None

Objectives

- Review the significance of diabetic ketoacidosis (DKA) in the pediatric population
- Provide basics of DKA management in the acute phase
- Educate to avoid mistakes in early DKA management
- Highlight differences between adult and pediatric management of DKA

Our patient

Previously healthy, thin and gaunt-appearing 10-year-old girl who presented to Urgent Care after mom noted she was short of breath.

At UC, pt noted to have significantly increased work of breathing.

Given albuterol/ipratropium x2 and dexamethasone x1 with some improvement but continued retractions.

Our patient

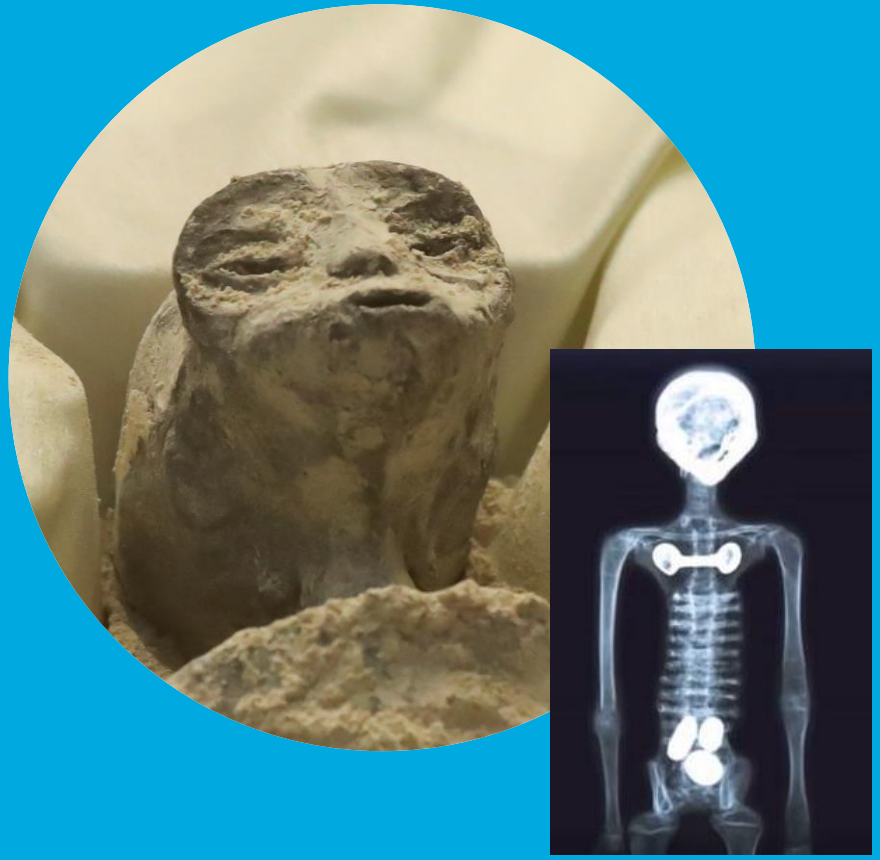
Transported to ED for treatment of status asthmaticus.

In the ED

…patient noted to have increased work of breathing.

HR 110, RR 24, BP 90/56, T 37.1. FLUVID negative.

Started on continuous albuterol 10 mg/hr. No improvement.




I WANT TO
BELIEVE
FBI

Sick or not sick?

Patient is looking worse.

Due to respiratory distress, concern patient may need intubation, so blood gas obtained.

The shocking conclusion…

iSTAT VBG+ lytes:

7.17/21/42/7.1/-11

Glucose 455


THOUGHT IT WAS ASTHMA
WAS DKA ALL ALONG
imgflip.com

Hyperglycemia: Blood glucose >200 mg/dl

AND

Metabolic Acidosis: Venous pH <7.3 or plasma bicarbonate <15 mEq/L

AND

Ketosis: ketones in urine or blood (beta hydroxybutyrate >1.0)

=

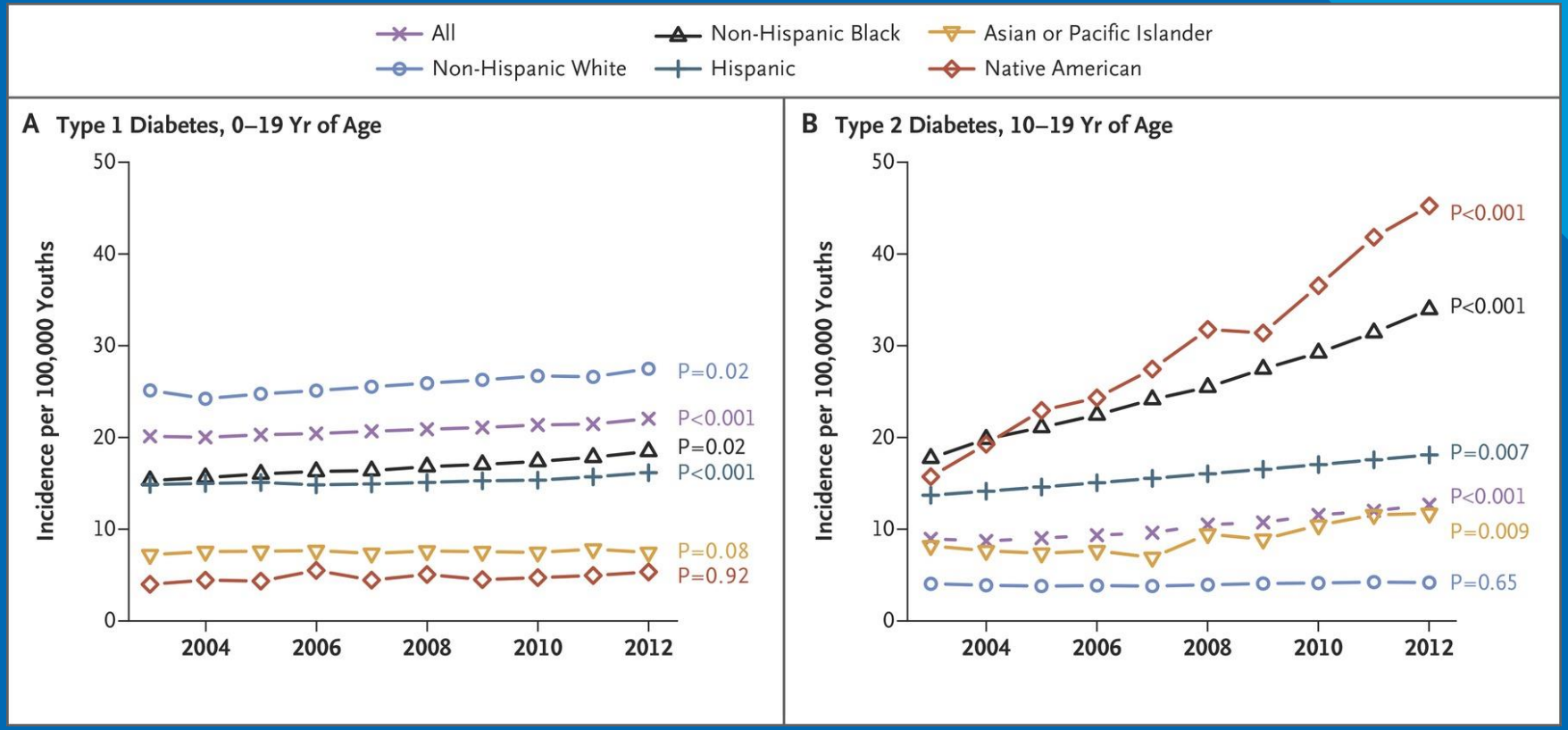
DIABETIC KETOACIDOSIS

Who (gets DKA)?

- Approximately 22 new T1DM per 100,000 youth <20 years of age
  - Incidence is increasing 3-5% annually
- Most between 5-14 years of age
- Slight male predominance
- White > Black > Hispanic > Asian > Native American
- Colorado has one of the highest incidences in the US
  - *(Also the most studied)*

Who (gets DKA)?

- Risk factors:
  - Public insurance
  - Rural address
- Often seek care multiple times prior to diagnosis (30%)



Mayer-Davis et al. NEJM, 2017

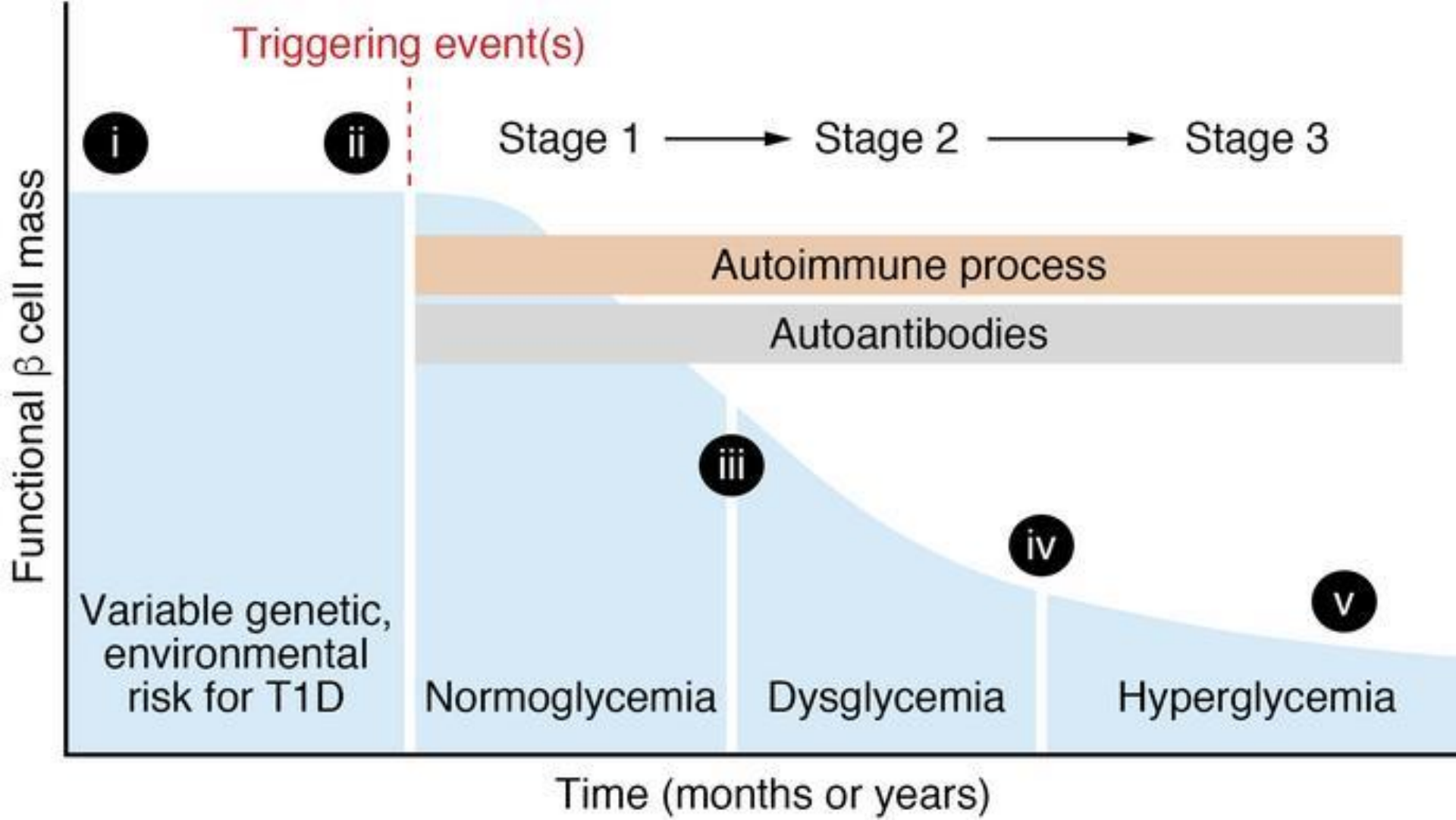
What (does the patient look like)?

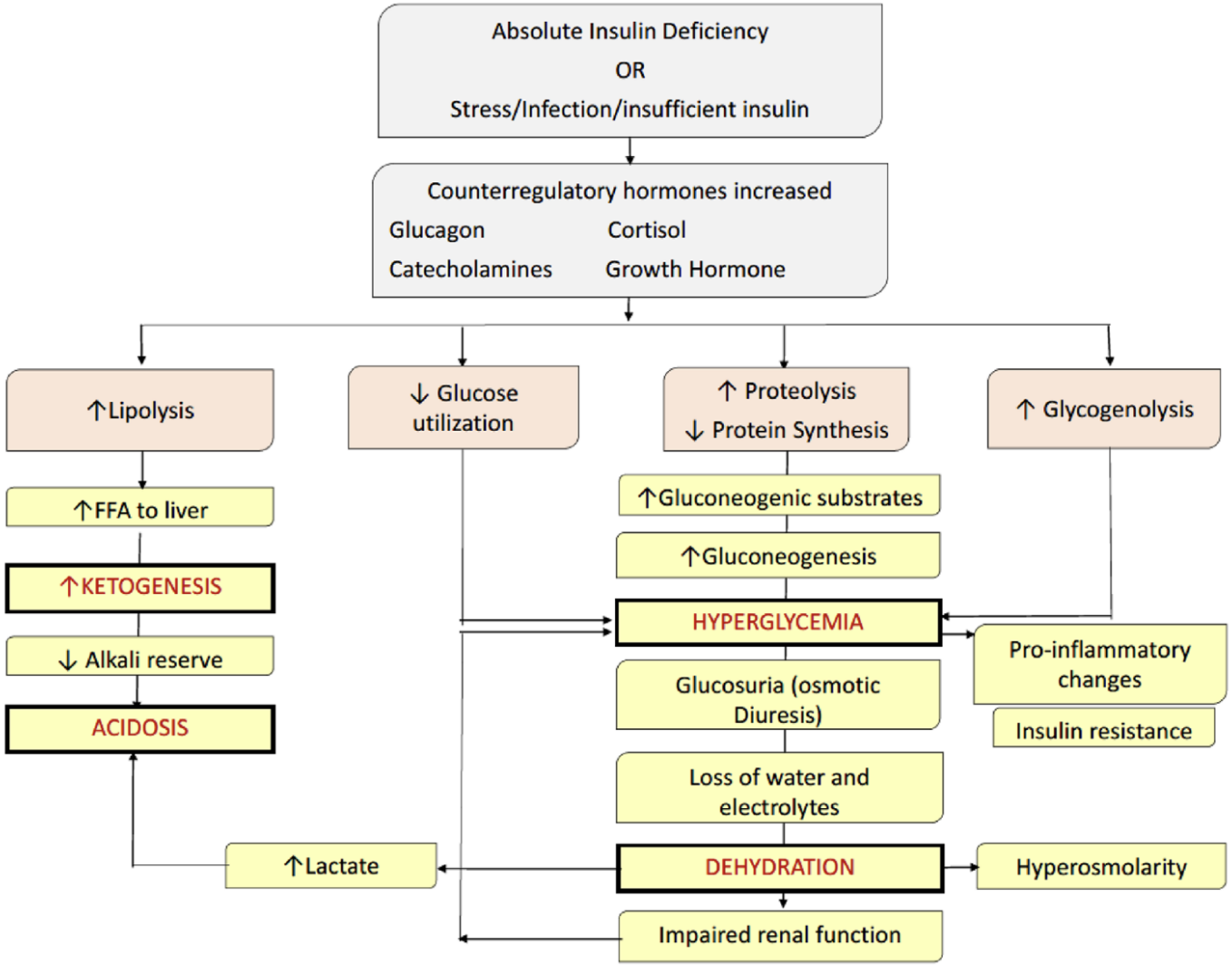
- Another clinical “great pretender”
  - Nausea/vomiting
  - Abdominal pain
  - Weight loss (72%)
  - Shortness of breath
  - Fatigue (74%)
  - Flu-like symptoms (30%)
  - “Polys” – polyuria (85%), polydipsia (90%)

Munoz et al., Clin Diabetes, 2019

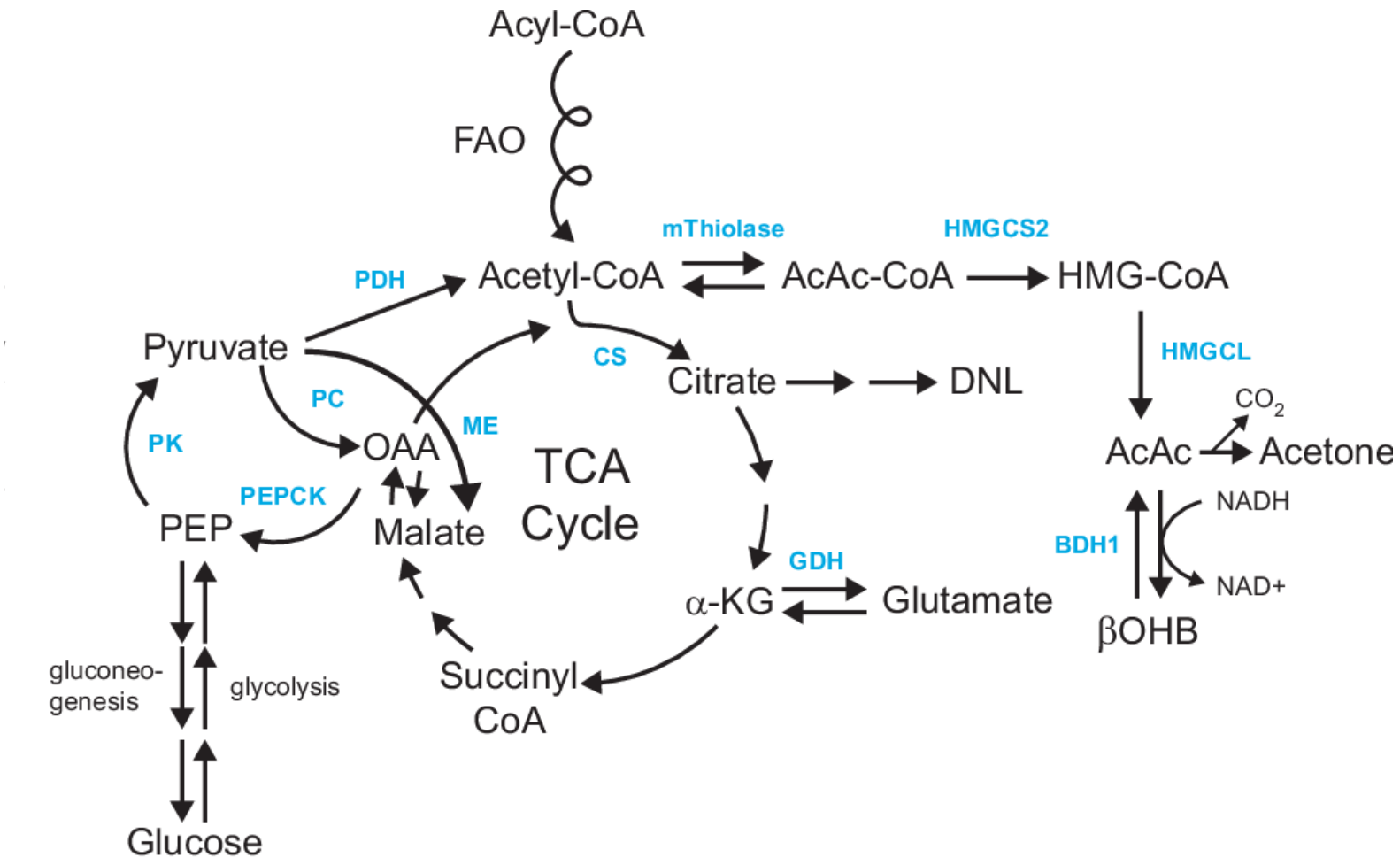
Why (do kids get DKA)?

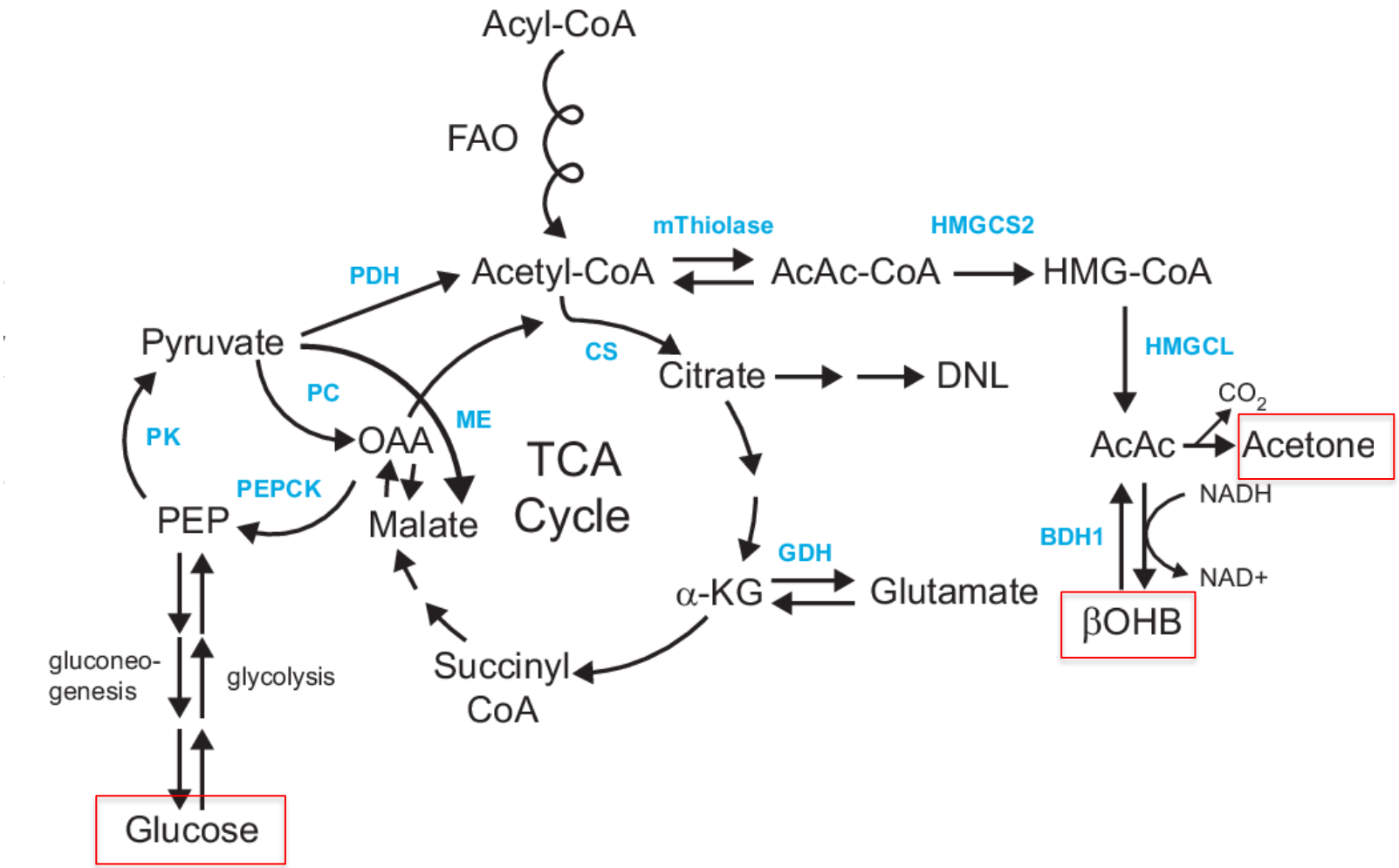
- Deficiency in circulating insulin and increase levels of counterregulatory hormones (glucagon, catecholamines, cortisol, growth hormone)
- **Etiology**: New onset Type 1 DM, failure of insulin pump/poor compliance, acute illness in otherwise stable diabetics
    - Relative insulin deficiency occurs in **trauma**, sepsis, febrile illness who can lead to imbalance of homeostatic mechanisms


Triggering event(s)
i
ii
Stage 1
Stage 2
Stage 3
Autoimmune process
Autoantibodies
iii
iv
v
Functional β cell mass
Variable genetic, environmental risk for T1D
Normoglycemia
Dysglycemia
Hyperglycemia
Time (months or years)



Glaser et al. Pediatr Diabetes, 2022.


Acyl-CoA
FAO
mThiolase
HMGCS2
PDH
Acetyl-CoA
AcAc-CoA
HMG-CoA
Pyruvate
CS
Citrate
DNL
HMGCL
PC
ME
CO2
PK
OAA
TCA
Cycle
AcAc
Acetone
PEPCK
NADH
PEP
Malate
BDH1
GDH
α-KG
Glutamate
NAD+
βOHB
gluconeo-
genesis
glycolysis
Succinyl
CoA
Glucose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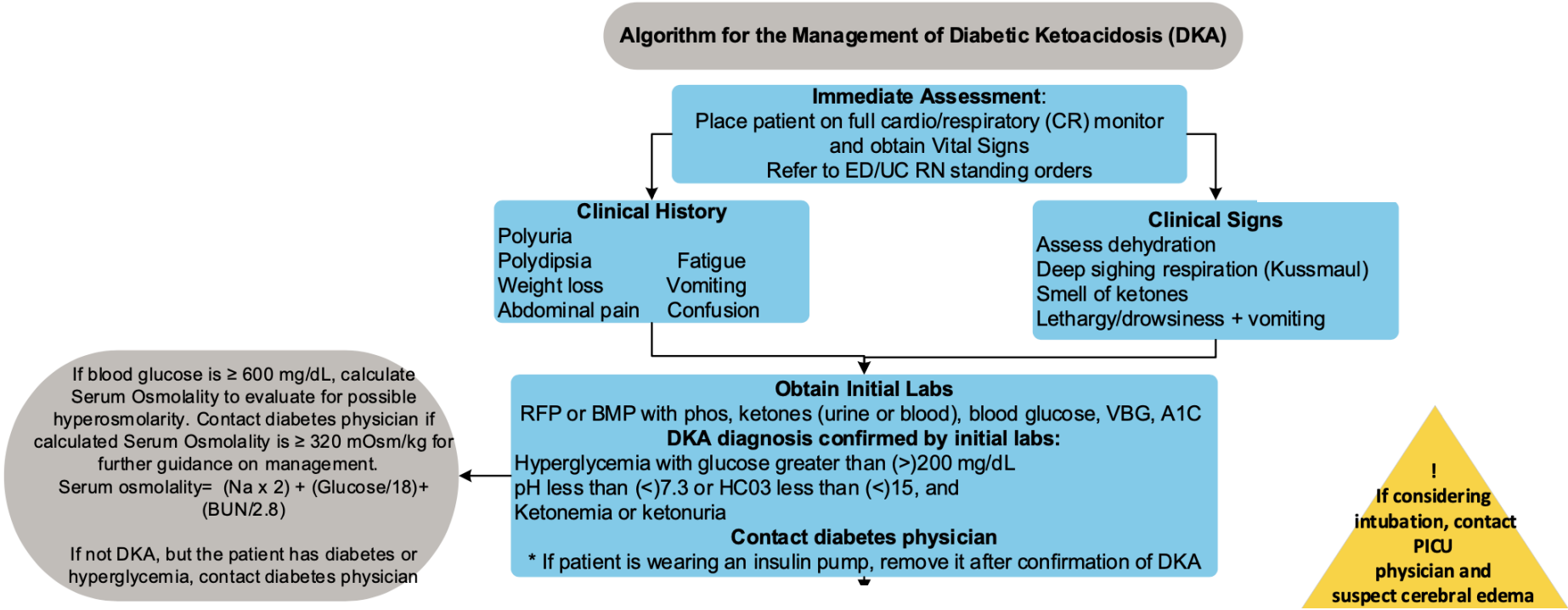
Acetone
PEPCK
NADH
PEP
Malate
BDH1
GDH
NAD+
α-KG
Glutamate
βOHB
gluconeo-
genesis
glycolysis
Succinyl
CoA
Glucose

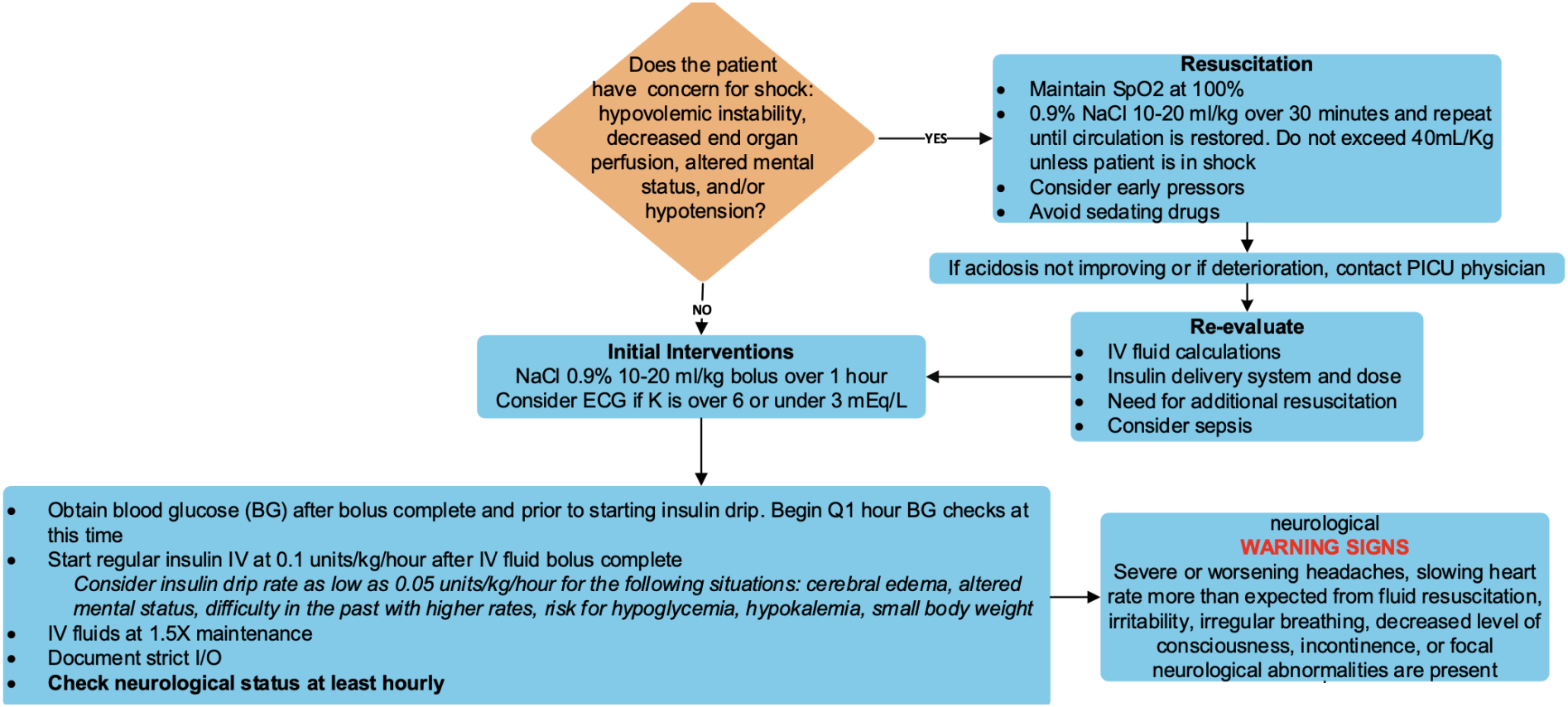
How (do we fix it)?

- Fluids
- Insulin

Target resolution of *acidosis*, NOT *hyperglycemia*

Pole, pole.


Algorithm for the Management of Diabetic Ketoacidosis (DKA)
Immediate Assessment:
Place patient on full cardio/respiratory (CR) monitor and obtain Vital Signs
Refer to ED/UC RN standing orders
Clinical History
Polyuria
Polydipsia
Weight loss
Abdominal pain
Fatigue
Vomiting
Confusion
Clinical Signs
Assess dehydration
Deep sighing respiration (Kussmaul)
Smell of ketones
Lethargy/drowsiness + vomiting
Obtain Initial Labs
RFP or BMP with phos, ketones (urine or blood), blood glucose, VBG, A1C
DKA diagnosis confirmed by initial labs:
Hyperglycemia with glucose greater than (>)200 mg/dL
pH less than (<)7.3 or HC03 less than (<)15, and
Ketonemia or ketonuria
Contact diabetes physician
* If patient is wearing an insulin pump, remove it after confirmation of DKA
If blood glucose is ≥ 600 mg/dL, calculate Serum Osmolality to evaluate for possible hyperosmolarity. Contact diabetes physician if calculated Serum Osmolality is ≥ 320 mOsm/kg for further guidance on management.
Serum osmolality= (Na x 2) + (Glucose/18)+ (BUN/2.8)
If not DKA, but the patient has diabetes or hyperglycemia, contact diabetes physician
!
If considering intubation, contact PICU physician and suspect cerebral edema


Does the patient have concern for shock: hypovolemic instability, decreased end organ perfusion, altered mental status, and/or hypotension?
YES
Resuscitation
Maintain SpO2 at 100%
0.9% NaCl 10-20 ml/kg over 30 minutes and repeat until circulation is restored. Do not exceed 40mL/Kg unless patient is in shock
Consider early pressors
Avoid sedating drugs
If acidosis not improving or if deterioration, contact PICU physician
Re-evaluate
IV fluid calculations
Insulin delivery system and dose
Need for additional resuscitation
Consider sepsis
NO
Initial Interventions
NaCl 0.9% 10-20 ml/kg bolus over 1 hour
Consider ECG if K is over 6 or under 3 mEq/L
Obtain blood glucose (BG) after bolus complete and prior to starting insulin drip. Begin Q1 hour BG checks at this time
Start regular insulin IV at 0.1 units/kg/hour after IV fluid bolus complete
Consider insulin drip rate as low as 0.05 units/kg/hour for the following situations: cerebral edema, altered mental status, difficulty in the past with higher rates, risk for hypoglycemia, hypokalemia, small body weight
IV fluids at 1.5X maintenance
Document strict I/O
Check neurological status at least hourly
neurological
WARNING SIGNS
Severe or worsening headaches, slowing heart rate more than expected from fluid resuscitation, irritability, irregular breathing, decreased level of consciousness, incontinence, or focal neurological abnormalities are present

Labs

BG Q1 hour
RFP (or BMP with phos) q2 hours
VBG initially, Q2 until pH at or above 7.15
Beta Hydroxybutyrate initially and as needed before transition to SC
Obtain ECG if K is over 6 or under 3

Blood Glucose (mg/dL)	% Rate NS ± Electrolytes	% Rate D10NS ± Electrolytes	Final Dextrose Concentration
>300	100%	0%	0
251-299	50%	50%	5
200-250	25%	75%	7.5
151-199	0	100%	10
< 150	Either decrease insulin drip as low as 0.05unit/kg/hour and/or increase GIR by increasing D10NS fluid rate (up to 2X maintenance) or change to D12.5 NS at 100% total rate		

*** See potassium supplementation table.**

DKA Resolution

Serum bicarbonate greater than or equal to (≥)18 mEq/L OR Beta Hydroxybutyrate less than 1 mmol/mL
AND
Clinically well, tolerates PO challenge with non-carbohydrate containing liquid

Transition to Subcutaneous (SC) insulin

Contact diabetes physician for doses and timing
*See page 7 for transition algorithm

Initial Serum Potassium	Potassium Supplementation
Greater than 5.5	None
4.5-5.5	20 mEq/L K-Acetate + 20 mEq/L Kphos*
Less than 4.5	30 mEq/L K-Acetate + 30 mEq/L Kphos* Hold Insulin drip until K above 3.
K supplementation is based on initial lab level. If K changes in management, patient may require repletion with potassium bolus	
*40 mEq/L KCL may be used if K-Acetate and Kphos unavailable	

Resolution of DKA

Serum bicarbonate >18 mEq/L

OR

beta-hydroxybutyrate < 1.0 mmol/L

AND

Clinically well, tolerates PO challenge

Resolution is correction of ACIDOSIS,
not correction of HYPERGLYCEMIA

Intensivist's dilemma:

What will kill this patient?

Actual intensivist in PPE circa 2020

$$\textit{Serum Osmolality (calculated)} = 2\,[Na^{+}] + \frac{[BUN]}{2.8} + \frac{[Glucose]}{18}$$

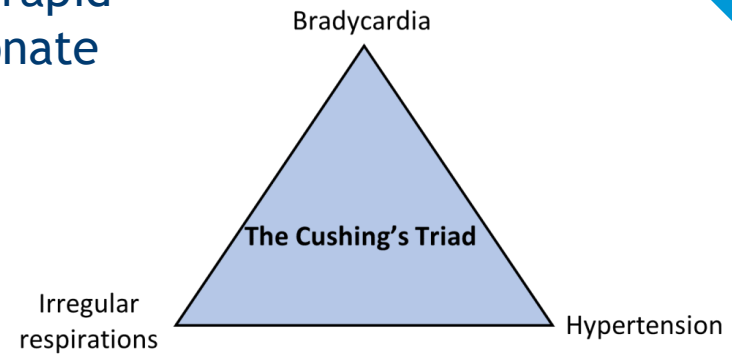
Cerebral edema

- Relatively rare (1%)
- High morbidity/mortality (20-25%)
- **Timing**: Usually within the first 24 hours, often in the first 4-12 hours *after* the start of treatment
- **Signs**: Confusion, lethargy, seizures, combativeness
- **Mechanism**: Osmotic *or* hypoxic-ischemic

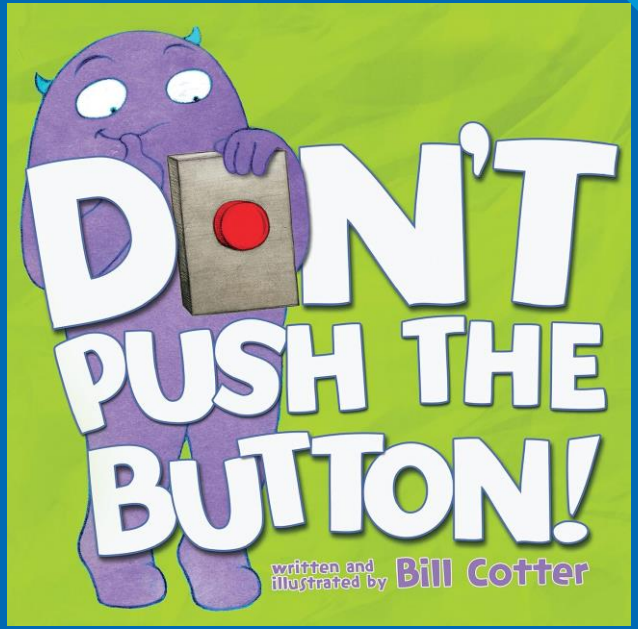
Cerebral edema

- **Independent risk factors:**
  - Younger age, more severe acidemia, rapid correction, administration of bicarbonate

- **Management:**
  - Elevate HOB, head midline
  - Hypertonic saline (or mannitol)
  - +/- CT head
  - Neuro checks, watch for Cushing's triad



What (not to do).



Don't: Bolus the insulin

- Insulin bolus = rapid drop in blood glucose
- Rapid shifts in osmolality = rapid shifts in free water in/out of tissue
- Cerebral edema, cerebral edema, cerebral edema


Opciones de vista
Hablando: salón Legisladores
Abandonar

Don't: Give bicarb

- Limited data
- Bicarbonate administration an independent factor associated with a greater risk of cerebral edema
- Bicarbonate in DKA associated with INCREASED CSF acidosis
- Decreases serum K^+ due to intracellular shift
- Shifts oxygen-hemoglobin dissociation curve to the left = tissue hypoxia/ischemia

Don't: Sedate (or intubate)

- Sedation will make neuro exam unreliable
- May alter native respiratory drive
- Intubating patient disturbs body's homeostatic mechanism for managing severe metabolic acidosis (Kussmaul respirations)

(*Don't*: Worry too much about the K^+)

- Many aspects of DKA pathophysiology effect serum potassium
  - Dehydration -> Aldosterone release -> Urine losses
  - Intracellular shift of H+ -> loss of intracellular K+
  - Insulin administration -> Intracellualar shift of K+ back into the cells
- Guidelines say to maintain > 3.0 mEq/L

(*Don't*: Worry too much about the K^+)

- DO NOT DELAY other therapies (fluid bolus, insulin administration) to replete potassium
  - Access issues in dehydrated kids are real

- UNLIKE ADULTS, children usually tolerate significant hypokalemia without arrythmia

CHILDREN ARE NOT LITTLE ADULTS

TRAINING FOR HEALTH CARE PROVIDERS
SECOND EDITION

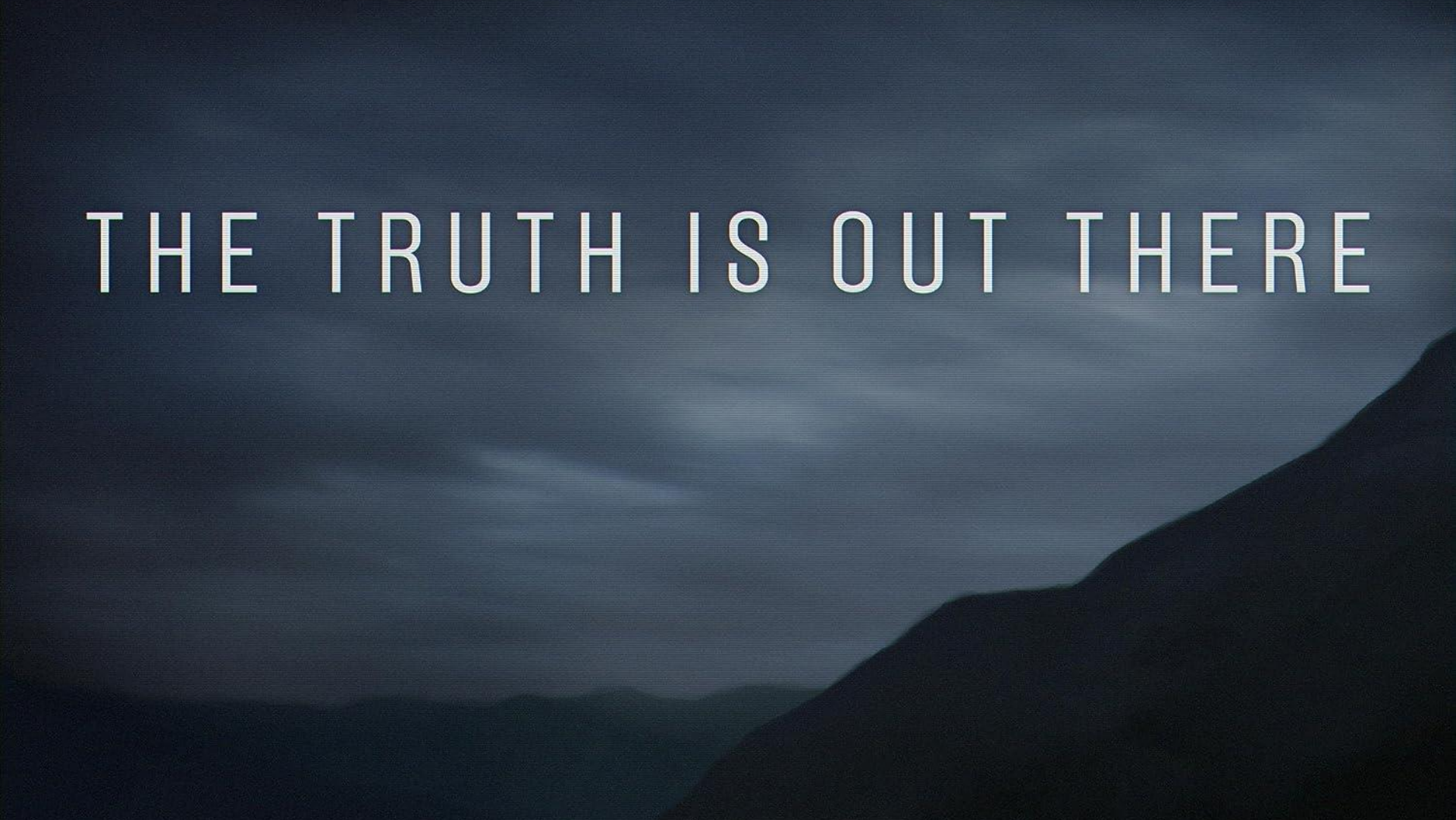
World Health Organization

Children's Health and the Environment
WHO Training Package for the Health Sector
World Health Organization
https://www.who.int/health-topics/children-environmental-health

WHO/CED/PHE/EPE/19.12.07

Conclusions

- DKA is common, often misdiagnosed, and can be life threatening
- The incidence of new onset T1DM is increasing in the pediatric population
- Volume resuscitation and slow insulin administration are the keys to fixing DKA
- Pole pole: don’t bolus insulin, don’t bolus bicarb, don’t sedate/intubate


THE TRUTH IS OUT THERE