

ACID-BASE DISORDERS

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Disclosures

N/A

Objectives

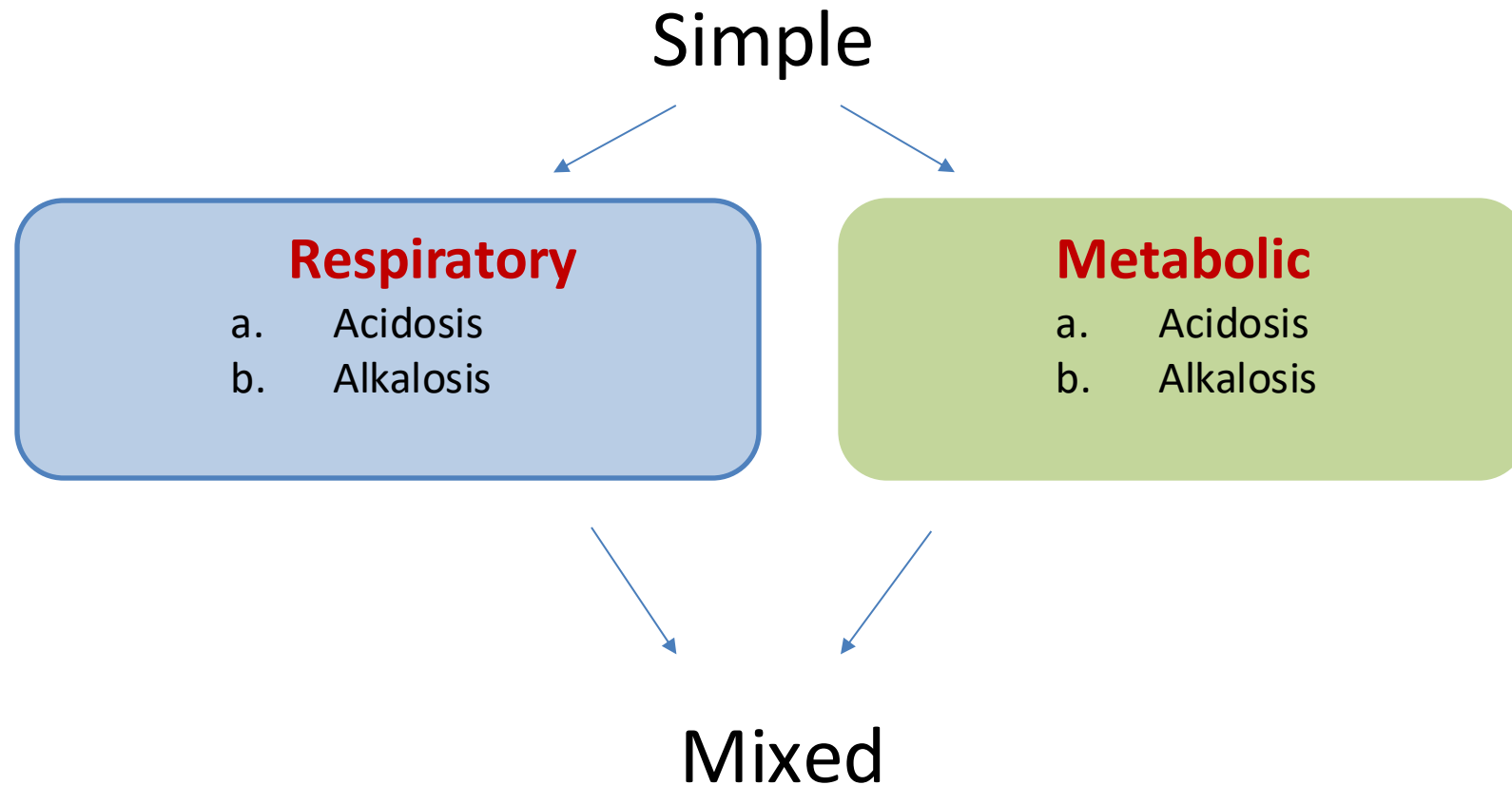
- **Define** acid-base balance and explain its physiological importance.
- **Describe** the normal ranges of blood pH, PaCO₂, and HCO₃⁻.
- **Classify** acid-base disorders into metabolic and respiratory categories.
- **Explain** the compensatory mechanisms involved in each disorder.
- **Interpret** arterial blood gas (ABG) results to identify specific disorders.
- **Discuss** common causes and clinical features of each acid-base disorder.
- **Outline** basic treatment strategies for correcting acid-base imbalances.

20 y/o F with severe asthma has received supplemental oxygen, nebulized albuterol continuously, IV methylprednisolone and IV Mg but fails to improve and gets intubated. Her laboratory 10 minutes earlier reveal the following
ABG: pH 7.17, PaCO₂ 54, PO₂ 80 . BMP: Na 136, Cl 99, K 2.8 HCO₃ 21

Which acid base disorder(s) is present?

- a. Respiratory acidosis
- b. Respiratory acidosis plus AG metabolic acidosis
- c. Respiratory acidosis plus NAG metabolic acidosis
- d. Respiratory acidosis plus AG metabolic acidosis plus NAG metabolic acidosis

Types of Acid – Base disorders



Step Wise Approach

1. Analyze acid base disturbance with both ABG (pH) and BMP (HCO₃)
 - a. Correct the venous blood gas add 0.05 to make it same as arterial
 - b. Correct the venous pCO₂ decrease 5 (-5) to make same as arterial
 - c. Serum bicarbonate to arterial bicarbonate difference of 5
2. Verify accuracy
Compared calculated HCO₃ on ABG vs. HCO₃ on electrolyte panel
3. Calculate Anion Gap $AG = [Na] - (Cl + HCO_3)$ →
Correct for serum albumin
 - a. Approx 80% of serum AG is due to negative charges on circulating protein
 - b. For each 1g/dl fall in albumin the anion gap raises by 2.5
4. Calculate predicted respiratory or metabolic compensation →
5. Know causes of HAG and NAG
6. Compare ΔAG with ΔHCO_3 (delta/delta) > Predicted HCO₃ equal to measure HCO₃ (change in AG)
<1 = AGMA + Non AGMA
1-2 = pure AGMA
>2 MA + Malk (look for expected change in HCO₃)
7. Calculate serum osmolar gap when cause of HAG not know or toxic alcohol ingestion suspected →

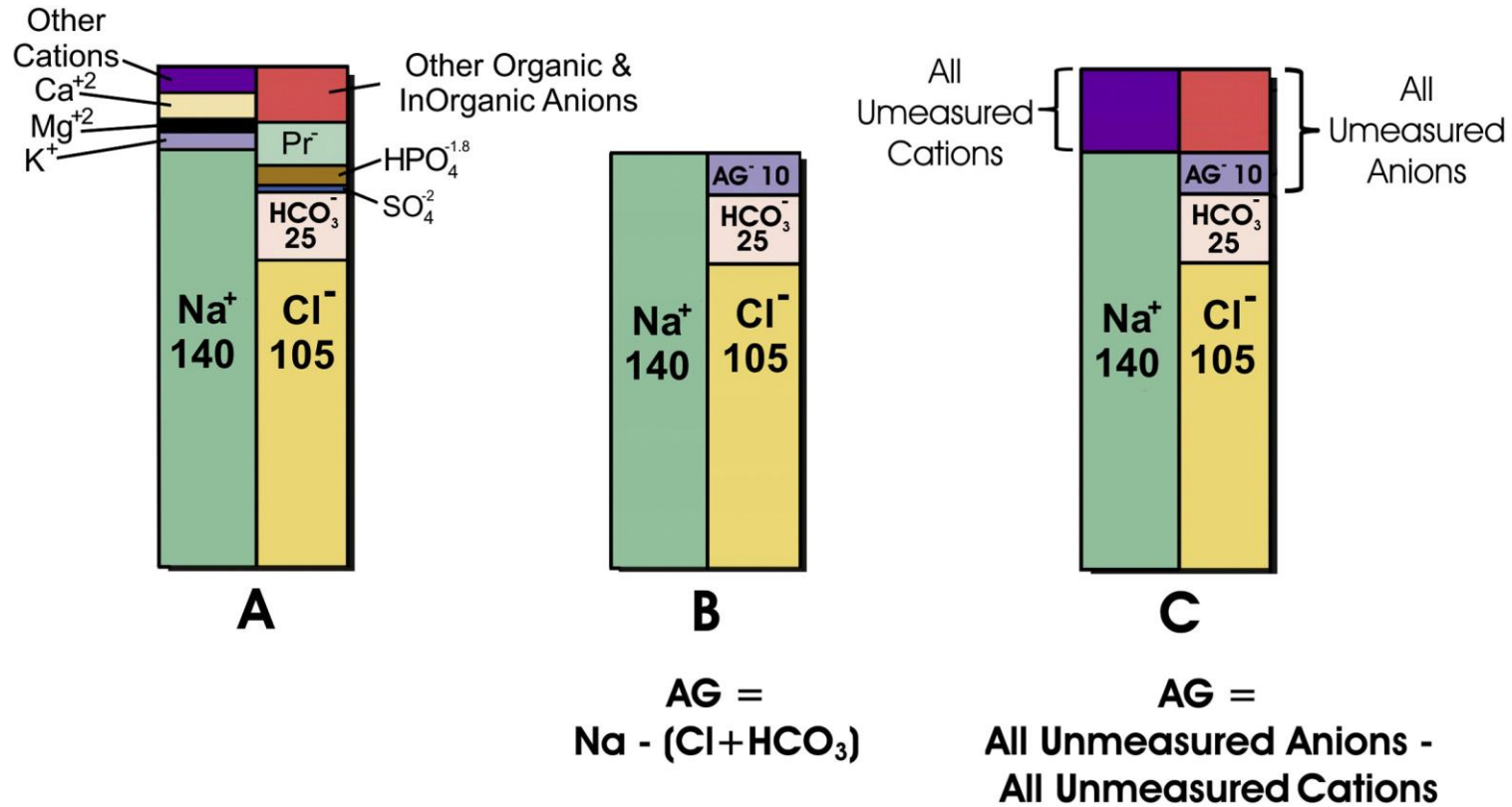
“Normal”

- pH 7.40
- HCO₃⁻ 24 mEq/L
- pCO₂ 40 mm Hg
- AG 12 mEq/L

$$AG = [Na] - (Cl + HCO_3) \text{ nl } 10-12 \text{ mEq/L}$$

Represents unmeasured anions present in the serum including anionic proteins, phosphate, sulfate and organic anions.

Correct for serum albumin



EXPECTED COMPENSATION (JASN 2010;21:920)

Metabolic acidosis: 2-24 hr

Winter's formula: $p\text{CO}_2 = 1.5 \times \text{HCO}_3 + 8 \pm 2$

Metabolic alkalosis: start 30 min, complete 24 hrs

$\text{PaCO}_2 = 0.7 \times (\text{HCO}_3 - 24) + 40 \pm 2 = \text{HCO}_3 + 15$

$\Delta \text{HCO}_3 \uparrow 1 \rightarrow \text{expect } \Delta p\text{CO}_2 \uparrow 0.7$

Respiratory acidosis:

Acute (<4-5days): $\Delta p\text{CO}_2 \uparrow 10 \rightarrow \Delta \text{HCO}_3 \uparrow 1 - \downarrow \text{pH } 0.1$

Chronic: $\Delta p\text{CO}_2 \uparrow 10 \rightarrow \Delta \text{HCO}_3 \uparrow 4 - \downarrow \text{pH } 0.015-0.03$

Respiratory alkalosis:

Acute: $\Delta p\text{CO}_2 \downarrow 10 \rightarrow \Delta \text{HCO}_3 \downarrow 2 \text{ or } \uparrow \text{pH } 0.08$

Chronic: $\Delta p\text{CO}_2 \downarrow 10 \rightarrow \Delta \text{HCO}_3 \downarrow 4 \text{ or } \uparrow \text{pH } 0.03$

Metabolic Acidosis

$$\downarrow \text{pH} \equiv \frac{\downarrow \text{HCO}_3^-}{\text{PaCO}_2}$$

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Respiratory Acidosis

$$\downarrow \text{pH} \equiv \frac{\text{HCO}_3^-}{\uparrow \text{PaCO}_2}$$

$$\downarrow \text{pH} \equiv \frac{\uparrow \text{HCO}_3^-}{\uparrow \text{PaCO}_2}$$



Osmolar Gap in diagnosis of Toxin-Induce AGMA

Tonicity = [effective osmoles]

Osmolality (osm)* = [effective osmoles] + [ineffective osmoles]

*Normal serum osm = 275 – 295 mosm/kg

Serum osm = $2 [\text{Na meq/L}] + [\text{BUN mg/dL}]/2.8 + [\text{glucose mg/dL}]/18$

$$\text{Gap}_{\text{osm}} = P_{\text{osm}}^{\text{det}} - P_{\text{osm}}^{\text{cal}}$$

Compared measures and calculated osmolality

Gap >10 setting of possible toxin ingestion suggest EgMP (ethylene glycol, Methyl alcohol or propylene glycol)

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$$\text{pH} \sim \frac{\text{HCO}_3^-}{\text{PaCO}_2}$$

“Normal”

- pH 7.40
- HCO₃⁻ 24 mEq/L
- pCO₂ 40 mm Hg
- AG 12 mEq/L

ABG: 7.17 / 54 / 80

Respiratory Acidosis

Na: 136 / Cl: 99 / HCO₃: 21

Expected HCO₃:

25.5

AG = [Na] – (Cl + HCO₃)

16 (nl 10-12)

Δ AG / Δ HCO₃ =

16-12 / 24 -21 = 1.3

“Normal”

- pH 7.40
- HCO₃⁻ 24 mEq/L
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Respiratory acidosis:

Acute (<4-5days): Δ pCO₂ \uparrow 10 \rightarrow Δ HCO₃ \uparrow 1 - \downarrow pH 0.1

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<1 =AGMA + Non AGMA

1-2 = pure AGMA

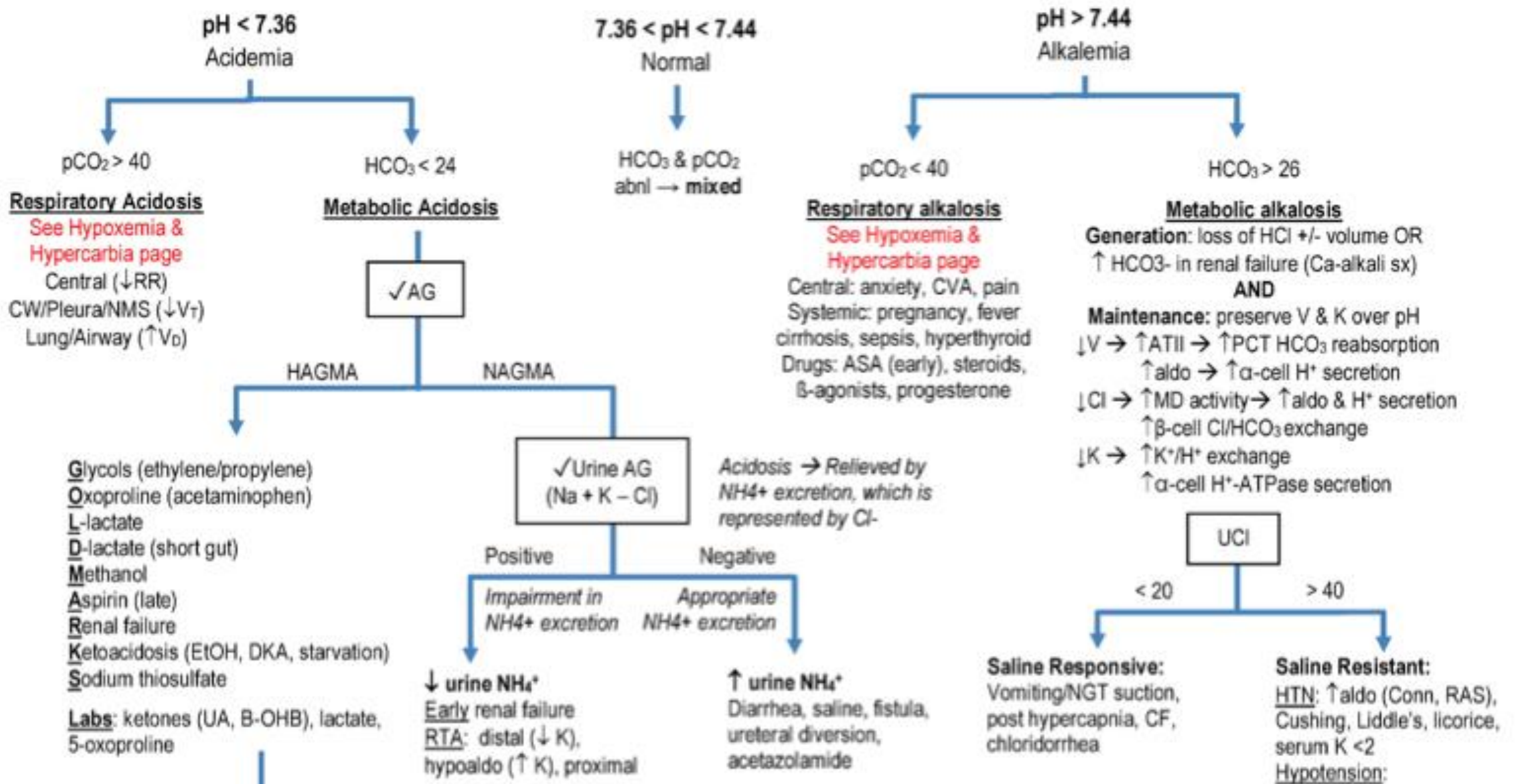
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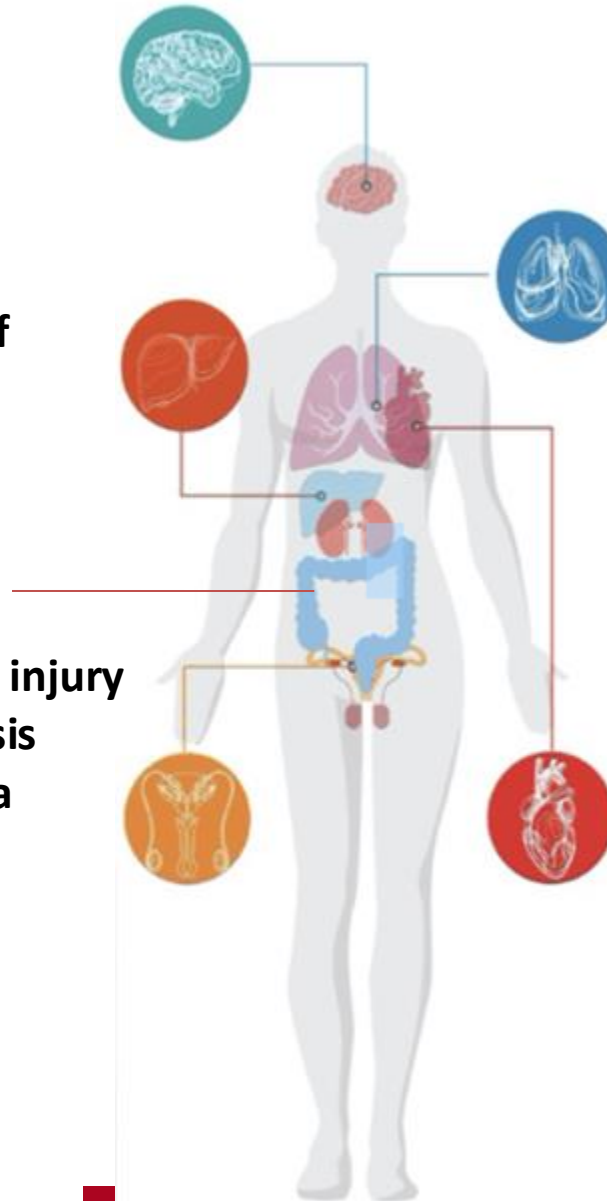


Metabolic Acidosis (AGMA)

Lethargy, coma,

Insulin resistance, inhibition of anaerobic glycolysis, protein degradation, decrease ATP synthesis,

Acute Kidney injury
Nephrolithiasis
Hyperkalemia



Decrease vaso & venoconstriction -> pulm. Congestion
Shift Hg-O₂ diss. Curve to Right -> decrease O₂ uptake and increase O₂ release
Hyperventilation, reps fatigue / failure

Impaired contractility, vasodilatation, venoconstriction, decrease CO, sensitization to arrhythmias, decrease responsiveness to pressors

Multisystem Failure

Causes of HAGMA

Differential diagnosis of HAGMA

- Glycolysis (EgMP)
- Oxyproline
- L - lactate
- D - lactate
- Methanol
- Aspirin
- Renal failure
- Ketoacidosis

Ketoacidosis

- Diabetic
- Alcoholic
- Starvation

Lactic acidosis

- Type A
- Type B
- D Lactic acidosis

39 y/o M found unresponsive (>12h). GCS 6. Initial ABG pH 6.83 PCO2 101, PO2 96
Intubated. BMP: Na 133, K 6.8, Cl 101, Bicarbonate 16, BUN 38, Creatinine 3.1, glucose 229.
UTox +ve for opiates, THC, cocaine. Urine ketones -ve.
Blood ASA and acetaminophen -ve.
Serum osmolality 314. LA 1,4.
Repeat ABG pH 7.22

Which of the following is the most appropriate next step

- a. Charcoal hemoperfusion
- b. Insulin infusion
- c. Fomepizole
- d. Urine alkalinization with Bicarbonate in D5W

ABG pH 6.83, PCO2 101 PO2 96 HCO3 16

Respiratory Acidosis

Na: 133 / Cl: 101 / HCO3: 16

Expected HCO3:

$(24+6) = 30 \sim$

Respiratory Acidosis + Metabolic acidosis

$AG = [Na] - (Cl + HCO3)$

16 (nl 10-12)

$\Delta AG / HCO3 =$

$16-12 / 24 - 16 = 0.5$

Respiratory Acidosis + HAGMA + NAGMA

Calculated osmolality:

$2Na + BUN/2.8 + Glucose/18$

$OG = SO - CO$

$OG = 314 - 292$

$OG = 21.7$

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Respiratory Acidosis

Na: 133 / Cl: 101 / HCO3: 16

Expected HCO3:

~30

Respiratory Acidosis + Metabolic acidosis

$AG = [Na] - (Cl + HCO3)$

16 (nl 10-12)

$\Delta AG / HCO3 =$

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Causes of HAGMA

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-Glycolysis (EgMP)

-Oxyproline

-L - lactate

-D - lactate

-Methanol

-Aspirin

-Renal failure

-Ketoacidosis

Ketoacidosis

- Diabetic

- Alcoholic

- Starvation

Lactic acidosis

- Type A

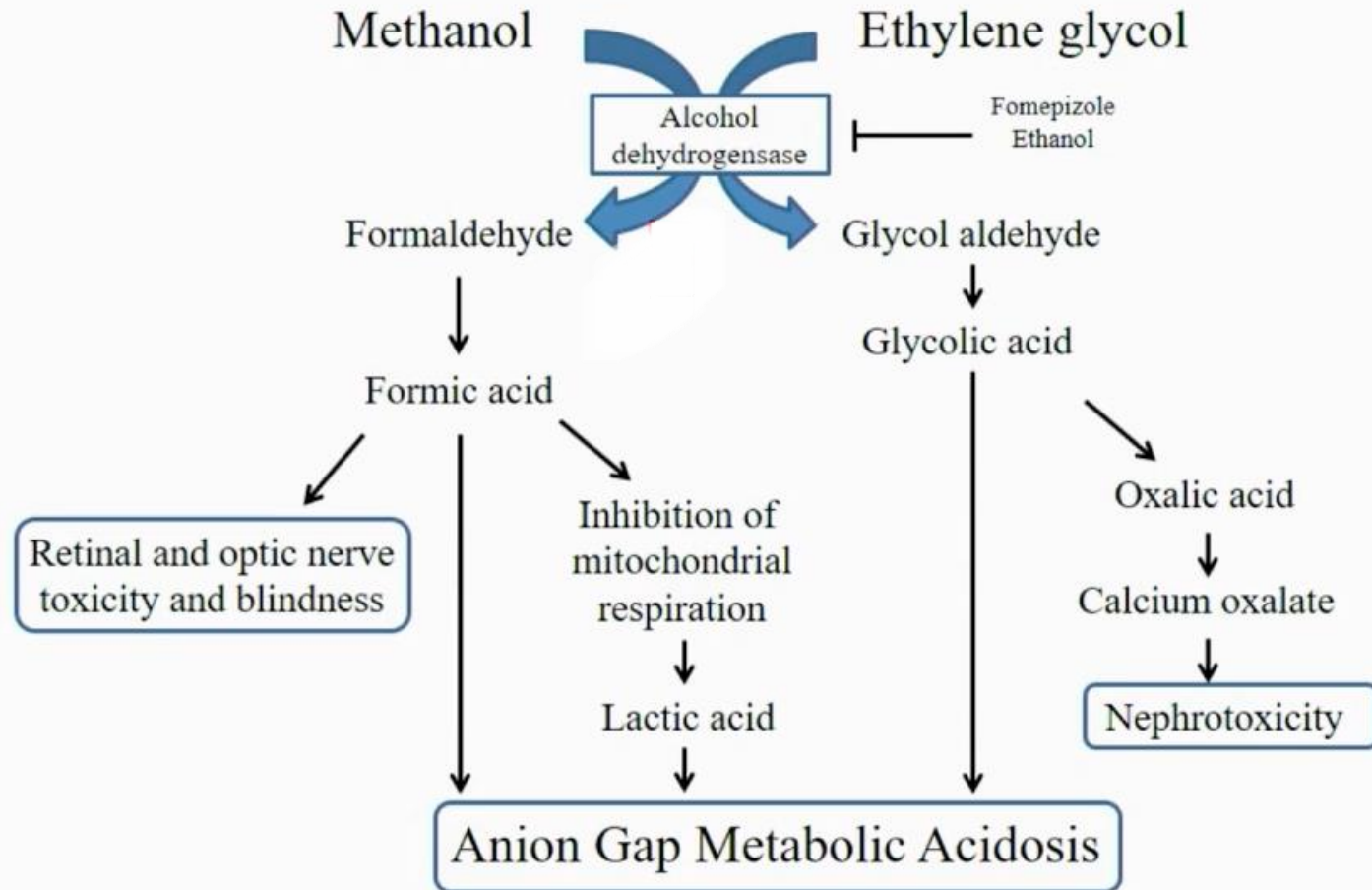
- Type B

- D Lactic acidosis

Toxins / drugs:

INCREASE OSMOLAR GAP

Methanol and Ethylene Glycol Poisoning



Increase AG (**Eg-M-P**)

Ethylene glycol

- Impaired conversion of lactate to glucose , increase NADH/NAD ratio, Converted to glycolic acid > glyoxylic acid > oxalic acid
- **Automobile antifreeze** (deicing / cooling fluid)
- Can have **lactate gap** (discrepancy between measure lactate and blood gas lactate, due to artificial elevation of LA in ABG)
- 3 stages of symptoms
 - Neuro (seizures, coma), N/V
 - Shock, pulmonary edema
 - AKI, Oliguria, Ca+OxC.

Tx:

- **Fomepizole** (competes with ETOH dehydrogenase & no conversion to acids; give every 4h if HD (gets dialyze)
- IV fluids containing HCO₃
 - Initial dose is 15 mg/kg, with subsequent doses of 10 mg/kg every 6 h until the toxin is cleared (EG <20)
- **HD** > if there is severe metabolic acidosis (**pH <7.25**), **AKI**, **ethylene glycol level >50 mg/dL**, or glycolate level >10 mg/dL (if available)
 - HD is generally continued until the EG level is <20 mg/dL.

Methanol > Blindness (retinal edema) , hyperemic optic disc, cerebral edema

- Windshield fluids, paint thinner, photocopier fluid.
- Methanol is metabolized to formic acid
- Emergent dialysis
Refractory acidosis (pH<7.2), neurological / ocular manifestation

Propylene glycol > **BZD ggt**, phenobarbital, phenytoin, **etomidate**

- It's a vehicle of IV sedatives
- Can be another form of D lactic acidosis > But can have normal AG
- Can produce AKI
- Role of fomepizole is unproven

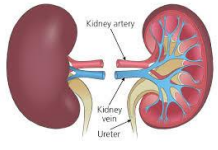



Diethyl glycol = automobile break fluid , **cerebral palsy**

Normal AG:

Isopropyl glycol

ketones and fruity odor , rubbing alcohol, mouthwash, solvents

Treatment is **supportive care**

	Ethylene Glycol	Methanol	Propylene Glycol	Isopropyl Glycol
OG	✓	✓	✓	✓
AG	✓	✓	✓	
				

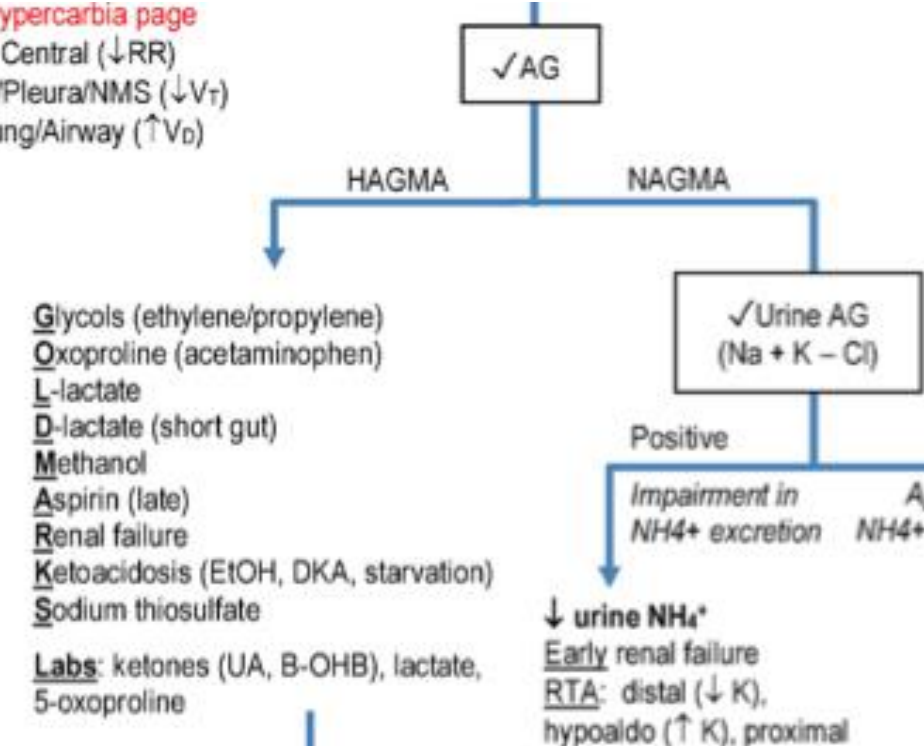
Hypercarbia page
 Central (\downarrow RR)
 CW/Pleura/NMS (\downarrow V_T)
 Lung/Airway (\uparrow V_D)

Hypercarbia page
 Central: anxiety, CVA, pain
 Systemic: pregnancy, fever
 cirrhosis, sepsis, hyperthyroid
 Drugs: ASA (early), steroids,
 β -agonists, progesterone

\uparrow HCO₃⁻ in renal failure (Ca-alkali sx)
AND
Maintenance: preserve V & K over pH
 \downarrow V \rightarrow \uparrow ATII \rightarrow \uparrow PCT HCO₃ reabsorption
 \uparrow aldo \rightarrow \uparrow α -cell H⁺ secretion
 \downarrow Cl \rightarrow \uparrow MD activity \rightarrow \uparrow aldo & H⁺ secretion
 \uparrow β -cell Cl/HCO₃ exchange
 \downarrow K \rightarrow \uparrow K⁺/H⁺ exchange
 \uparrow α -cell H⁺-ATPase secretion

Glycols (ethylene/propylene)
Oxoproline (acetaminophen)
L-lactate
D-lactate (short gut)
Methanol
Aspirin (late)
Renal failure
Ketoacidosis (EtOH, DKA, starvation)
Sodium thiosulfate

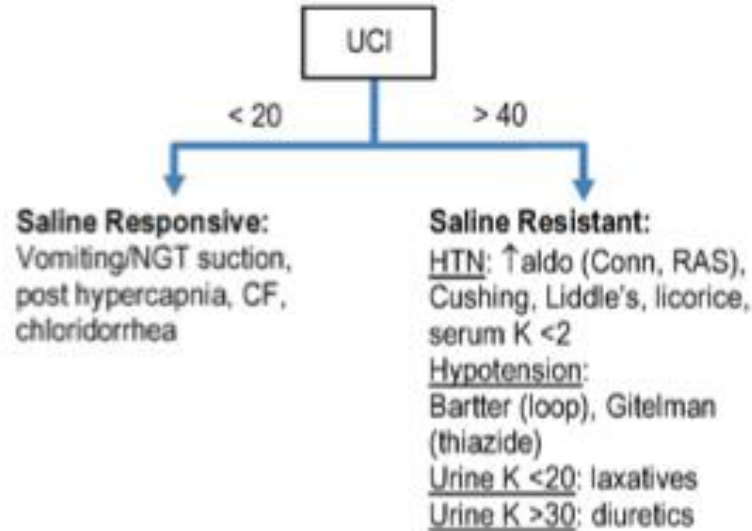
Lactic acidosis:
A tissue hypoxia
B no tissue hypoxia
 sepsis, cancer, EtOH,
 meds (metformin, NRTI,
 linezolid, salicylates)
D short-bowel



Acidosis \rightarrow Relieved by
 NH₄⁺ excretion, which is
 represented by Cl⁻

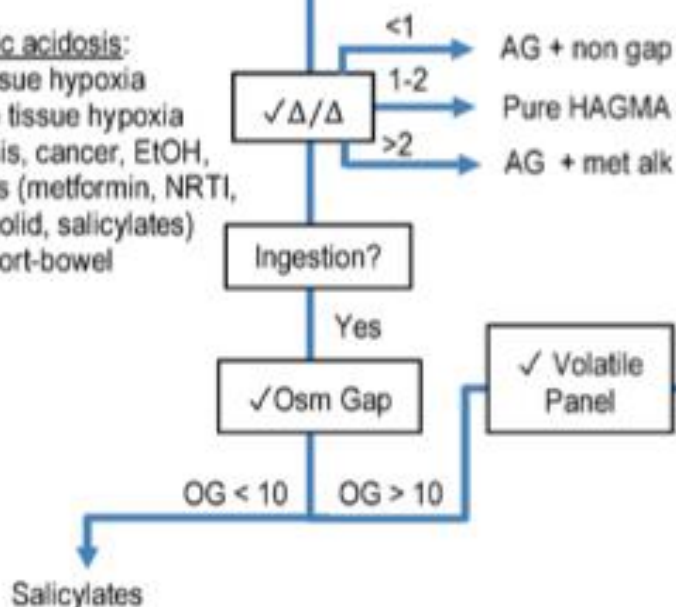
↓ urine NH₄⁺
 Early renal failure
 RTA: distal (\downarrow K),
 hypoaldo (\uparrow K), proximal

↑ urine NH₄⁺
 Diarrhea, saline, fistula,
 ureteral diversion,
 acetazolamide



Saline Responsive:
 Vomiting/NGT suction,
 post hypercapnia, CF,
 chloridorrhea

Saline Resistant:
HTN: \uparrow aldo (Conn, RAS),
 Cushing, Liddle's, licorice,
 serum K < 2
Hypotension:
 Bartter (loop), Gitelman
 (thiazide)
Urine K < 20: laxatives
Urine K > 30: diuretics



AG	OG	Ingestions	Toxin	Manifestations
↑	↑	Methanol	Formic acid	Δ MS, blurry vision, pupil dilation, papilledema
		Ethylene glycol	Oxalic acid	Δ MS, \downarrow Ca, Ca oxalate crystals \rightarrow AKI
		Propylene glycol	Lactic acid	AKI, liver injury
		Diethylene glycol	Diglycolic acid	AKI, n/v, pancreatitis, neuropathy, \uparrow lactate
nl/↑	↑	Isopropyl alcohol	Acetone	Δ MS, fruity breath, pancreatitis, \uparrow lactate
		Ethanol	Acetaldehyde	Keto/lactic acidosis \pm met. alk 2/2 emesis

Causes of HAGMA

Differential diagnosis of HAGMA

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- Oxyproline
- L - lactate**
- D - lactate**
- Methanol
- Aspirin
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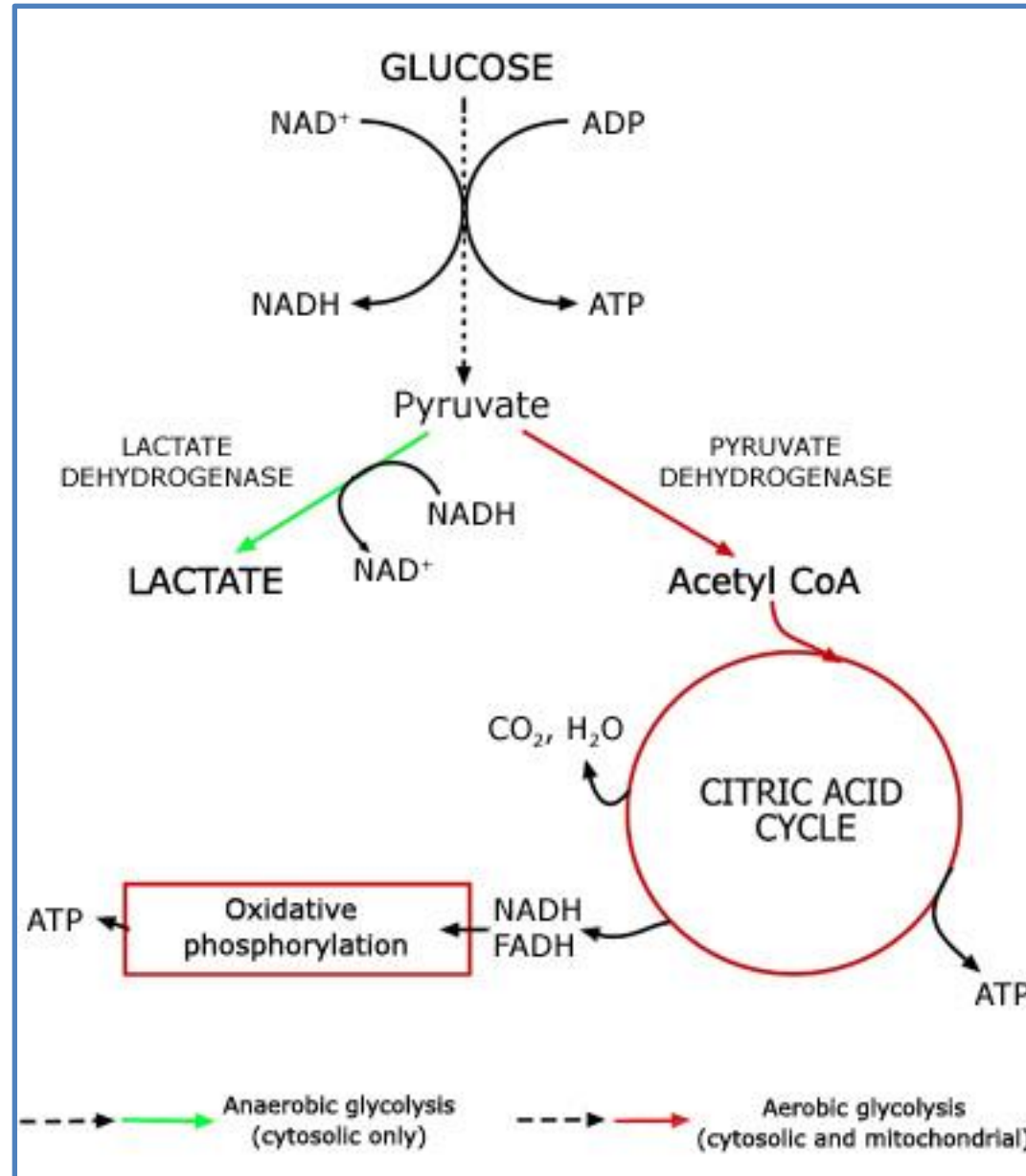
Ketoacidosis

- Diabetic
- Alcoholic
- Starvation

Lactic acidosis

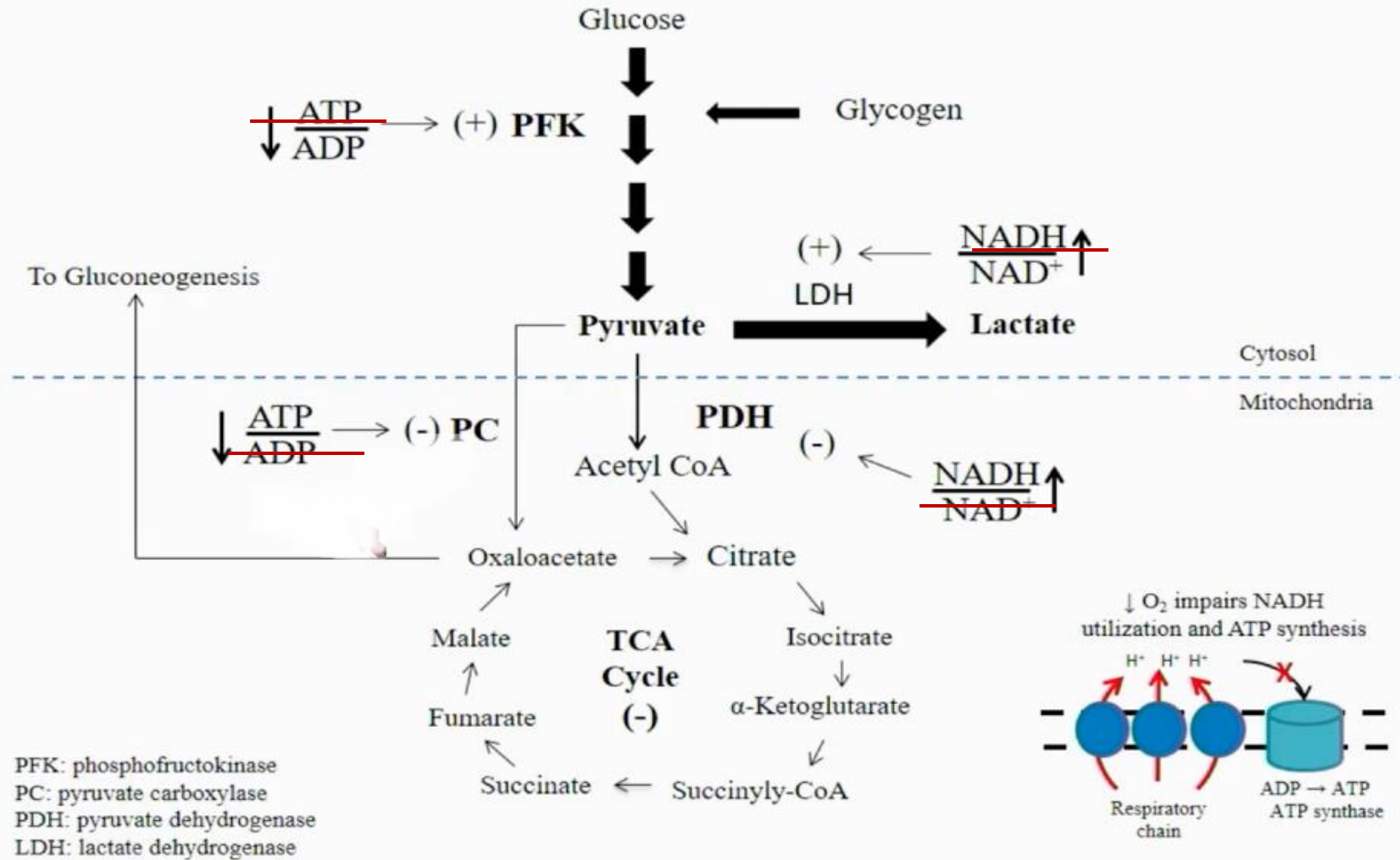
- Type A
- Type B
- D Lactic acidosis

Type A vs. Type B Lactic Acidosis



Type A

↑Lactate: Hypoxia-Induced ↓ATP, ↑[NADH/[NAD⁺]

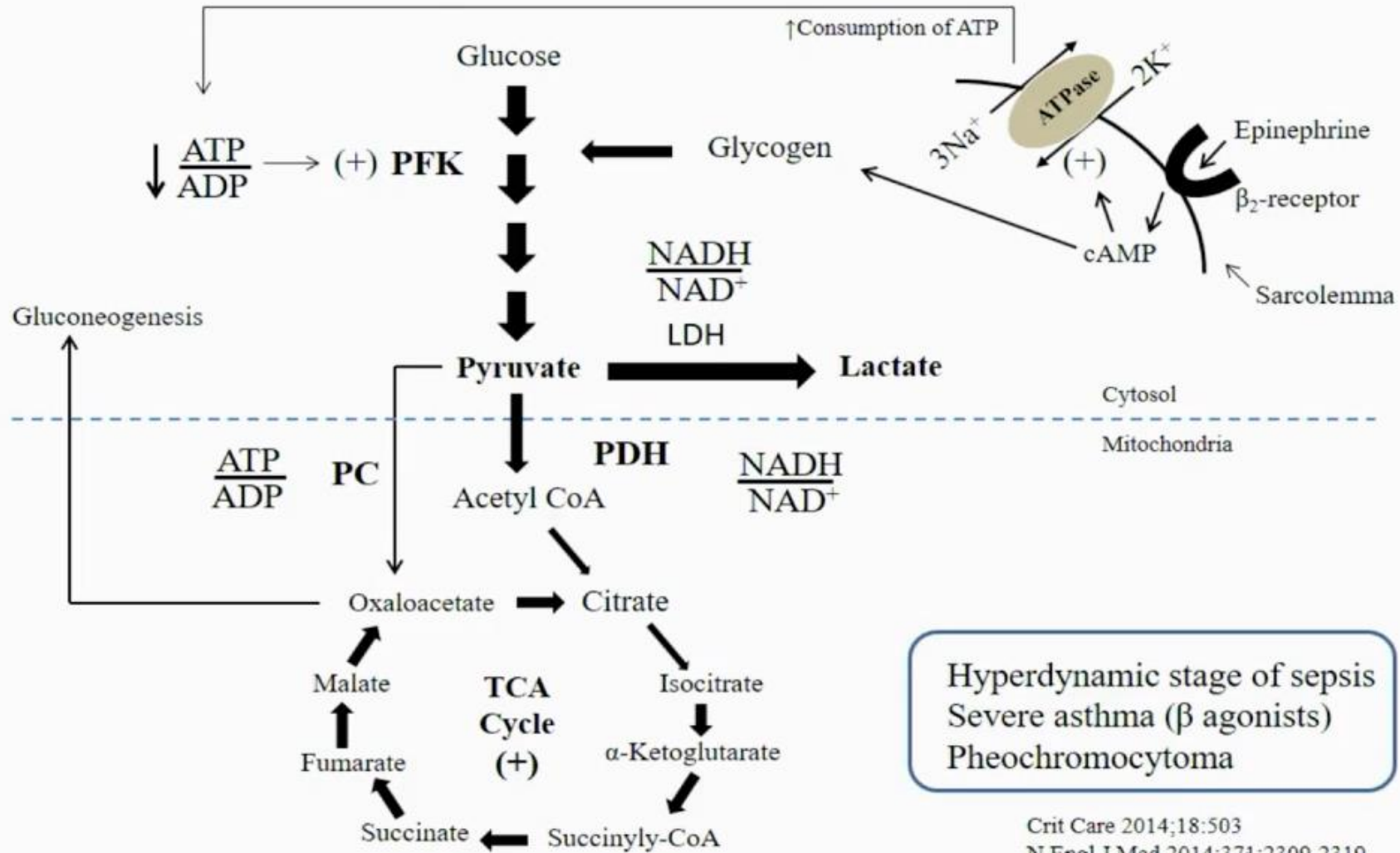


Type A (tissue hypoperfusion) = **INCREASE LACTATE - PYRUVATE RATIO**

- Hypoxic = imbalance between oxygen supply and oxygen demand
 - Anaerobic metabolism drives pyruvate toward lactate > **increasing lactate to pyruvate ratio**
- Seen in states if **low DO₂** (low cardiac output (HR/SV), severe anemia, CO poisoning, Hypoxia)
- Tx:
 - **CAREFUL** with administration of **NaHCO₃**
can increase lactate production (usually given with pH<7.1)
can **retain / produce more CO₂** worsening hypoventilation

Type B

Hyperlactatemia from ↑ Aerobic Glycolysis



Type B : (LACTATE - PYRUVATE RATIO NORMAL)

Oxygen delivery normal but phosphorylation impaired

- Seen more with catecholamines (anaerobic does NOT play a role)
- **Accelerated glycolysis** > (High catecholamines production which stimulate anaerobic, sepsis , seizures, cocaine)
- Reduce clearance (**Hepatic dysfunction**)
- **Mitochondrial dysfunction**

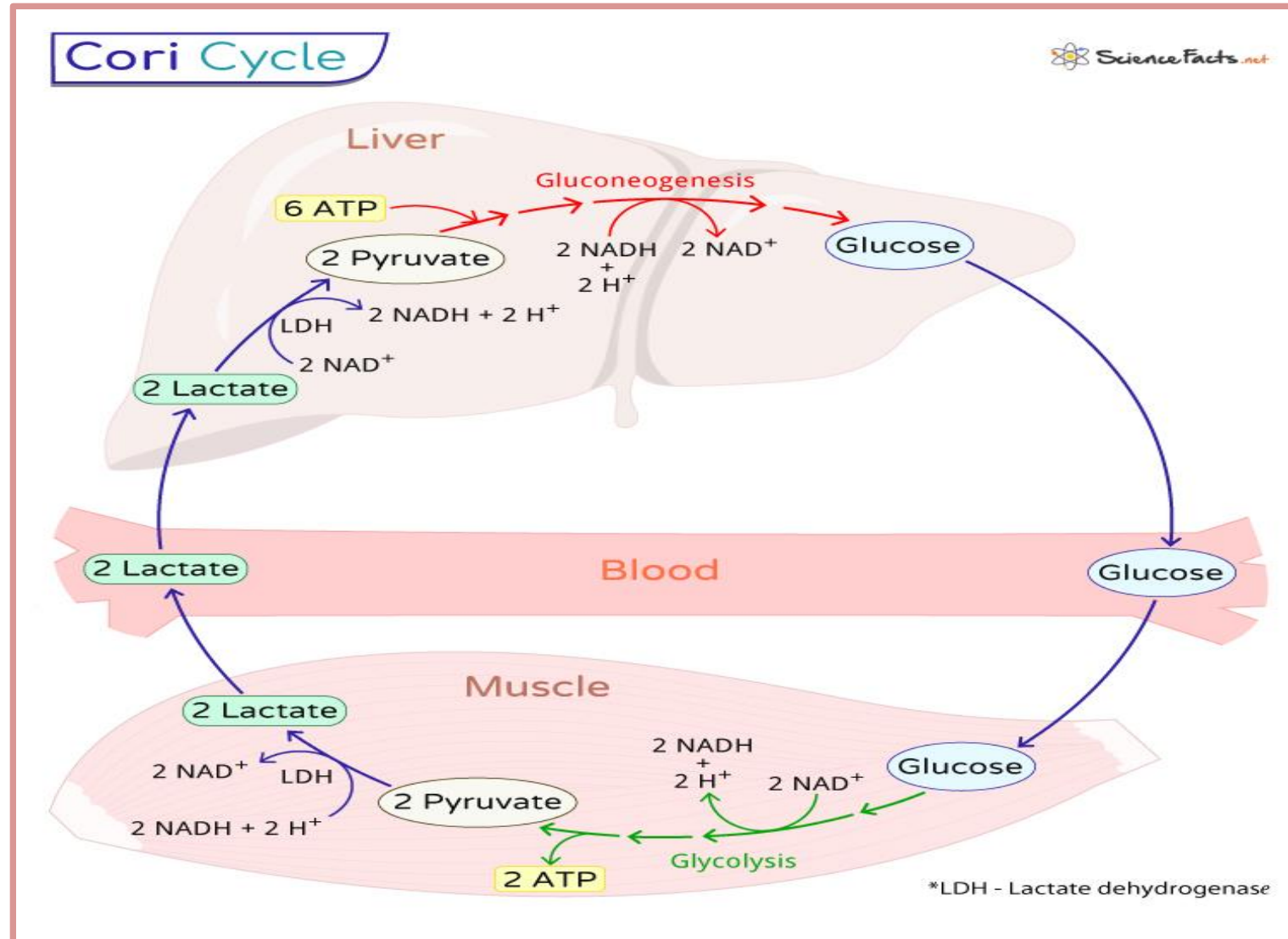
Drugs :

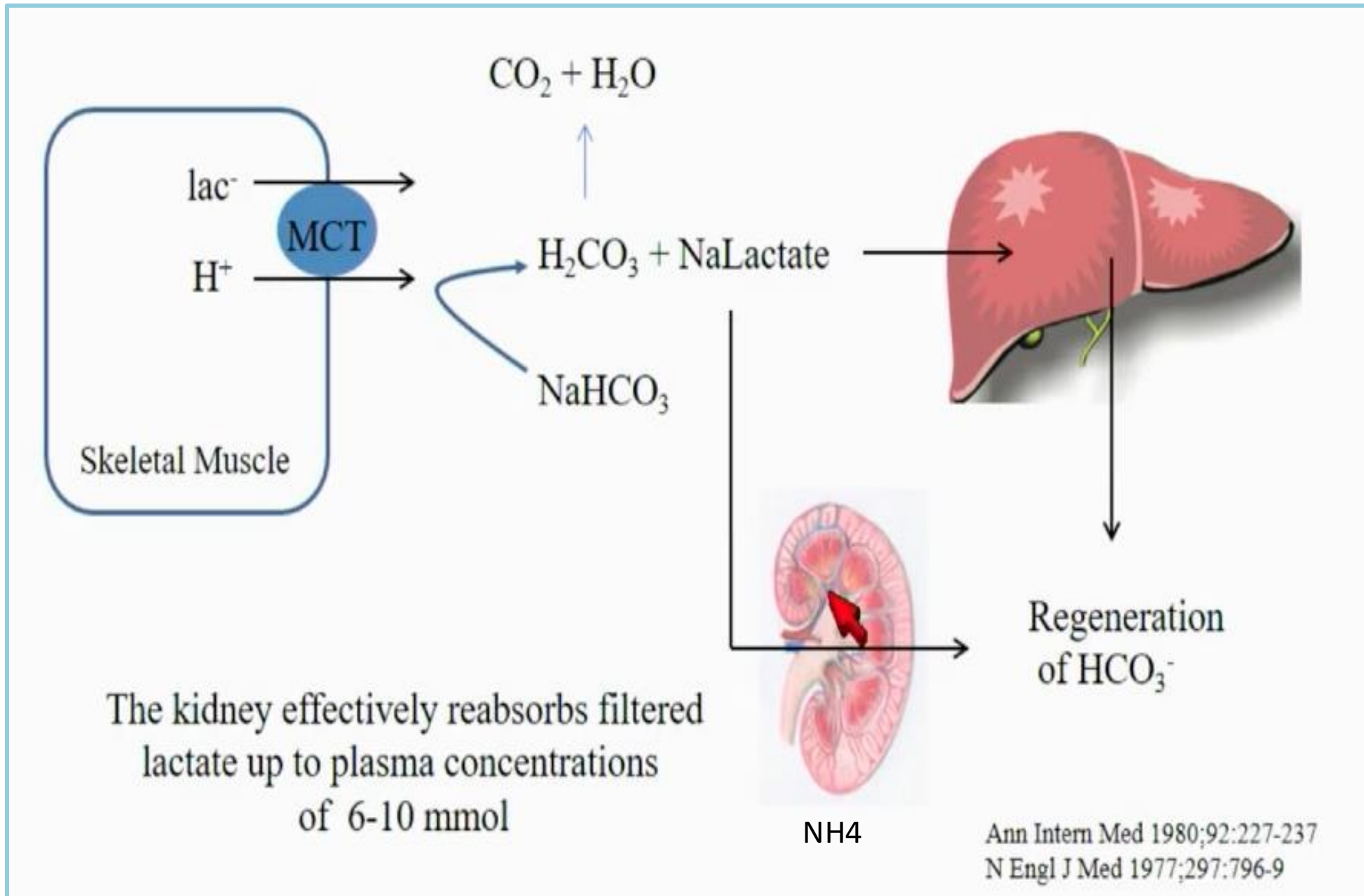
- HIV (zidovudine, didanosine, lamivudine) decrease oxidation of pyruvate with mitochondrial dysfunction
- **ASA**, ethanol (increase NADH/NAD ratio which increase lactate and ketoacid, exacerbated with thiamine deficiency)
- **Linezolid**
- **Thiamine deficiency** (essential enzyme cofactor in glycolysis (pyruvate dehydrogenase complex), the citric acid cycle (α -ketoglutarate dehydrogenase complex))
- Isoniazid
- **Propofol (mitochondrial dysfunction)**
- **Metformin**

Malignancy (large tumor burden and cell lysis)

Sorbitol , fructose (consumes ATP = stimulating glycolysis)

Lactic Acidosis clearance





Ann Intern Med 1980;92:227-237
 N Engl J Med 1977;297:796-9

55 y/o F with Hx of CHF, T2DM, HTN presents with decrease UOP, nausea, diarrhea and fatigue. Medications she has continued to take despite Nausea include (ASA, metformin, lisinopril, metoprolol). VS 90/70 HR 135 RR 13 Sat 99% RA.

Labs in Fig ; ABG ph 7.07, PCO2 15 PO2 120 HCO3 6

Serum tox: ASA , Tylenol and Ethanol level all normal.

Despite 1.5L of LR she has progressive hypotension and levophed is initiated.

New LA is 16.9.

Which of the following interventions is indicated

- a. Infusion of glucagon
- b. Infusion of D5W
- c. Hemodialysis
- d. Fomepizole

WBC	8,100/μL	8.1 x 10⁹/L
Hemoglobin	10.3 g/dL	103 g/L
Sodium	134 mEq/L	134 mmol/L
Potassium	5.9 mEq/L	5.9 mmol/L
Chloride	101 mEq/L	101 mmol/L
Carbon dioxide	11 mEq/L	11 mmol/L
BUN	86 mg/dL	30.7 mmol/L
Creatinine	10.44 mg/dL	922.9 μmol/L
Glucose	102 mg/dL	5.66 mmol/L
Serum osmolality	306 mOsm/kg	306 mmol/kg
Lactate	7.6 mmol/L	7.6 mmol/L
Lipase	55 U/L	0.92 μkat/L
Brain-type natriuretic peptide	519 pg/mL	519 ng/L

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Metabolic Acidosis

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22 (nl 10-12)

HAG Metabolic acidosis

Expected CO2:

Winters formula: $(1.5 \times \text{HCO}_3) + 8 \pm 2$

24.5 \pm 2

HAG Metabolic acidosis + Respiratory Alkalosis

$\Delta\text{AG} / \text{HCO}_3 =$

22-12 / 24 -11 = 0.7

HAG Metabolic acidosis + Respiratory Alkalosis + NAG metabolic acidosis

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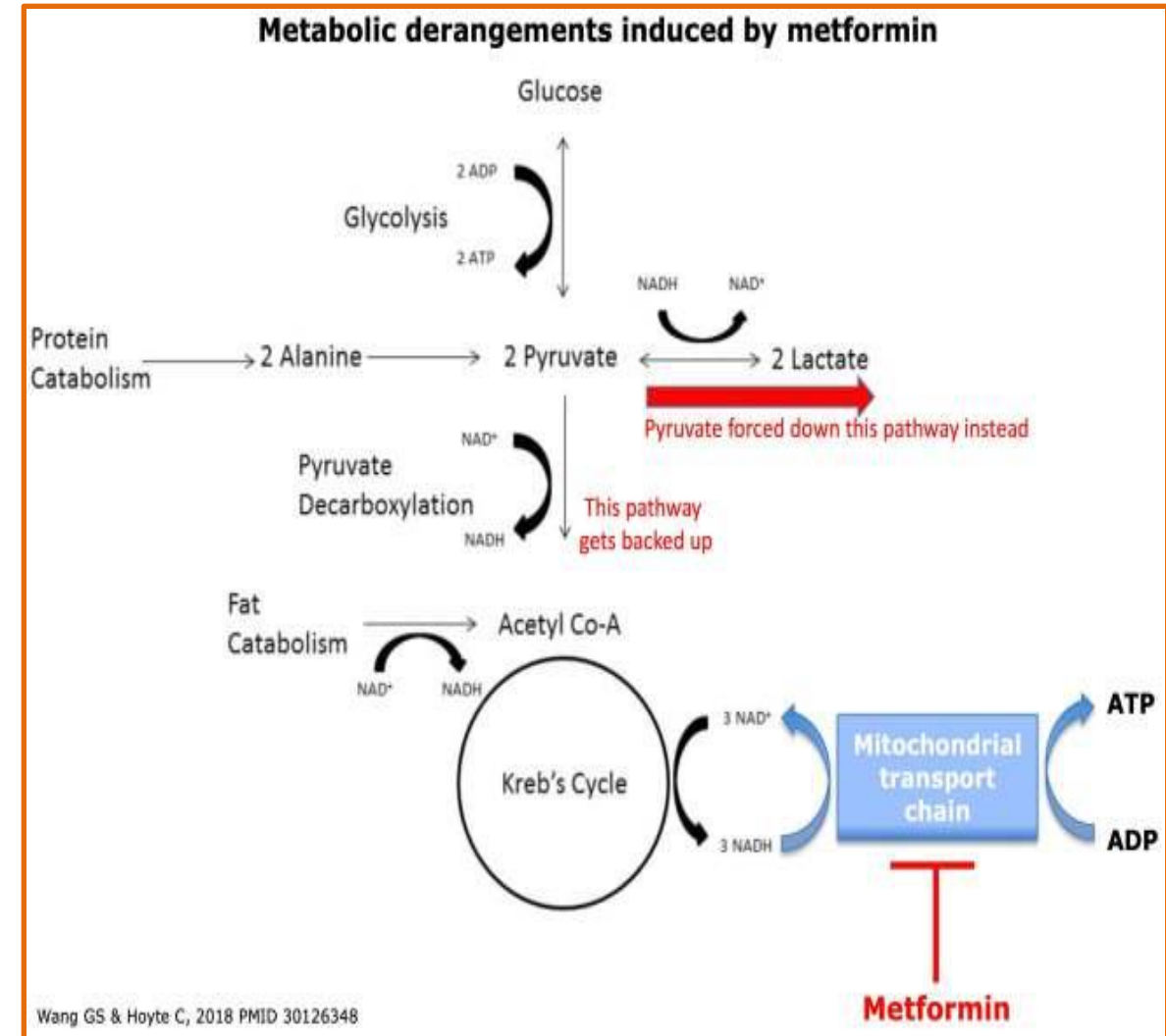
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Metformin

MA : decrease gluconeogenesis, **inhibits pyruvate dehydrogenase**, E: 100% renal) decrease 50% in CKD3 and stop in CKD4

- in Kidney failure (CKD) = more accumulation = binds to mitochondrial complex = increase LDH = increase lactate
- MALA (intoxication) = p/w **N,V,D** AMS, tachycardia, SOB , AGMA (>LA ,Ex: ~10 or more; pH <7.1)
- Tx:
 - RRT (small V/ distribution) : Failure to improve with supportive care (IV fluids, pressors), pH <7, and/or lactic acid >20 mmol/L
 - **5h of HD** and then CRRT with high dose
 - Usually does not result in hypoglycemia



19-y/o, admitted with acute hemiparesis following recreational cocaine use. CTH showed ICH. Patient was intubated and sedated with propofol and underwent craniectomy with hemorrhage evacuation. Systemic inflammation led to treatment with vancomycin and meropenem, but cultures were negative. Neurological findings were unimproved, New imaging showed worsened cerebral edema. On day 6, decompressive craniectomy was performed. The immediate postoperative VS : HR 130-140/min, BP 85/55 requiring vasoactive therapy. TTE EF 55%, but no regional wall abnormalities. SV 65 mL (with vasoactive meds). ScvO₂ 0.78. He became anuric, fever worsened, and laboratory values showed hyperkalemia, elevated creatine kinase (73,000 U/L [1,219.10 μ kat/L]), and lactic acidosis (9 mmol/L). Ventricular premature beats became increasingly frequent. Hydrocortisone was given, and renal replacement therapy was initiated to control serum potassium levels. Bradycardia led to transcutaneous pacing. Despite maximal supportive treatment, profound ventricular dysrhythmias ensued, and the patient died 24 h following the second craniectomy.

Which of one of the following mechanisms best explains this patient's postsurgical deterioration?

- a. Globally depressed oxygen transport to tissues
- b. Sepsis induce microvascular dysfunction
- c. Mitochondrial impairment
- d. Drug related Anaphylactic reaction

PRIS

Propofol related infusion syndrome

***Mitochondrial impairment** (The mechanism of PRIS involves inhibition of mitochondrial fatty acid oxidation and electron transport).

- Lactic acidosis 88%
- Cardiac arrhythmia / Brugada type coved ST elevation , BBB 70% , bradycardia
- Rhabdomyolysis 65% (elevated CK)
- Hypotension 50%
- Renal failure 47%, **Uric acid crystalluria** > just monitor
- Hyperkalemia 44%
- Hyperlipidemia 20%
- Pancreatitis , hypertriglyceridemia

Tx:

- Discontinuing propofol
- Supportive therapy
- Prolonged dialysis > **CRRT** (cuz is poorly cleared)

D lactic acidosis (lactate measured in lab is not D lactic acidosis)

- Mostly seen with small bowel resection > **carbohydrate meal reaches colon** > **bacterial fermentation** > generates organic acid including D lactate
- Presents with encephalopathy (ataxia, slurred speech) following carbohydrate meal where in colon bacterial fermentation generated organic acid to include D lactate

Dg

Confirmed by demonstrating high levels of D-lactate in urine and/or in serum. D-lactic acid level is usually greater than 3 mmol/L in D-lactic acidosis. D-lactate level is measured enzymatically using a D-lactic dehydrogenase specific assay.

57 y/o w is admitted for SOB and AMS. She has hx of polysubstance abuse, COPD, depression. 3 weeks ago, had a mechanical fall while intoxicated, sustaining a right radius fx. Current medications include oxycodone and Tylenol prn. She has been heavily drinking since the fall.

Patient requires to be intubated due to respiratory failure. Blood work shows WBC 14k, ALT 150/AST 61. Albumin 2. Toxicology + for opiates. Tylenol level 7ug/ml. Osmolar gap is nl. Rest of chemistry in fig 1. CXR with RUL consolidation. Started on ceftriaxone, azithromycin, thiamine, folate and IV fluids.

In addition to alcohol and starvation ketoacidosis, which process is contributing to severe metabolic acidosis.

- a. Paraldehyde
- b. D lactate
- c. 5 oxyproline
- d. Propylene glycol

pH	7.08
PCO ₂ , mm Hg	15
PO ₂ , mm Hg	109
Sodium, mEq	127 (127 mmol/L)
Potassium, mEq	3.2 (3.2 mmol/L)
Chloride, mEq	95 (95 mmol/L)
Bicarbonate, mEq	3 (3 mmol/L)
BUN, mg/dL	18 (6.43 mmol/L)
Creatinine, mg/dL	1.7 (150 μmol/L)
Glucose, mg/dL	68 (3.77 mmol/L)

ABG pH 7.08, PCO₂ 15 PO₂ 109 HCO₃ - 3

Acidosis

Na: 127 / Cl: 95 / HCO₃: 3

AG = [Na] - (Cl + HCO₃)

29 (nl 10-12)

29 + 5 = 34

HAG Metabolic acidosis

Expected CO₂:

Winters formula: $(1.5 \times \text{HCO}_3) + 8 \pm 2$

12.5 \pm 2

HAG Metabolic acidosis

$\Delta\text{AG} / \text{HCO}_3 =$

$34 - 12 / 24 - 3 = 1.04$

HAG Metabolic acidosis

Osmolar Gap is normal

pH	7.08
PCO ₂ , mm Hg	15
PO ₂ , mm Hg	109
Sodium, mEq	127 (127 mmol/L)
Potassium, mEq	3.2 (3.2 mmol/L)
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BUN, mg/dL	18 (6.43 mmol/L)
Creatinine, mg/dL	1.7 (150 $\mu\text{mol/L}$)
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Glucose, mg/dL	68 (3.77 mmol/L)

Causes of HAGMA

Differential diagnosis of HAGMA

-Glycolysis (EgMP)

-Oxyproline

-L - lactate

-D - lactate

-Methanol

-Aspirin

-Renal failure

-Ketoacidosis

Ketoacidosis

- Diabetic

- Alcoholic

- Starvation

Lactic acidosis

- Type A

- Type B

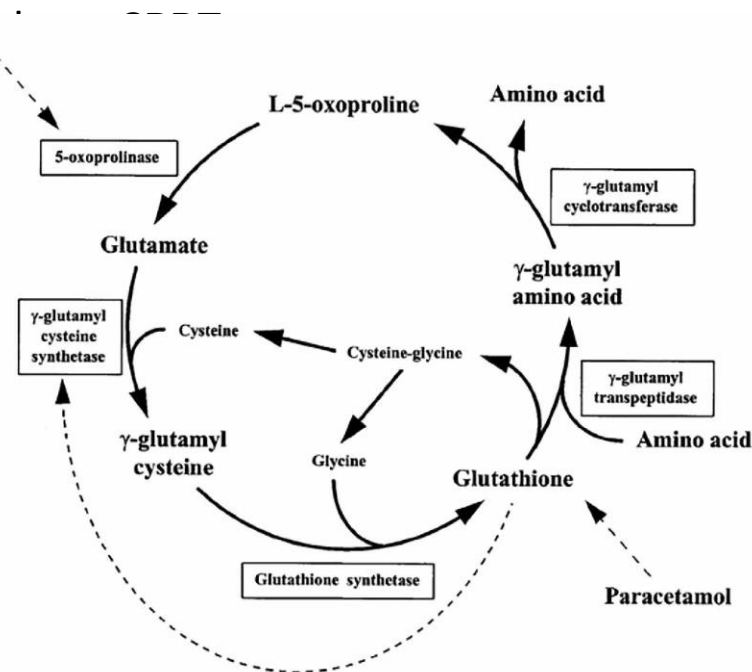
- D Lactic acidosis

5-Oxyproline (pyroglutamic acid)

- Decrease glutathione > glutamate unable to be used for synthesis and auto cyclizes in ATP depleting cycle to 5 oxyproline
- More seen in infection and use of **Acetaminophen** , poor nutrition, cirrhosis

Profound / severe AG elevation , w/ NO osmolar gap / Can have Normal LA

- Dg:
 - 5 oxyproline in serum
 - urine analyses for **organic acid screen** > pyroglutamic acids (Tylenol) > look for urinary organic acid screen
 - do if RRTx is **Flucloxacillin**



Causes of HAGMA

Differential diagnosis of HAGMA

- Glycolysis (EgMP)
- Oxyproline
- L - lactate
- D - lactate
- Methanol
- Aspirin**
- Renal failure**
- Ketoacidosis**

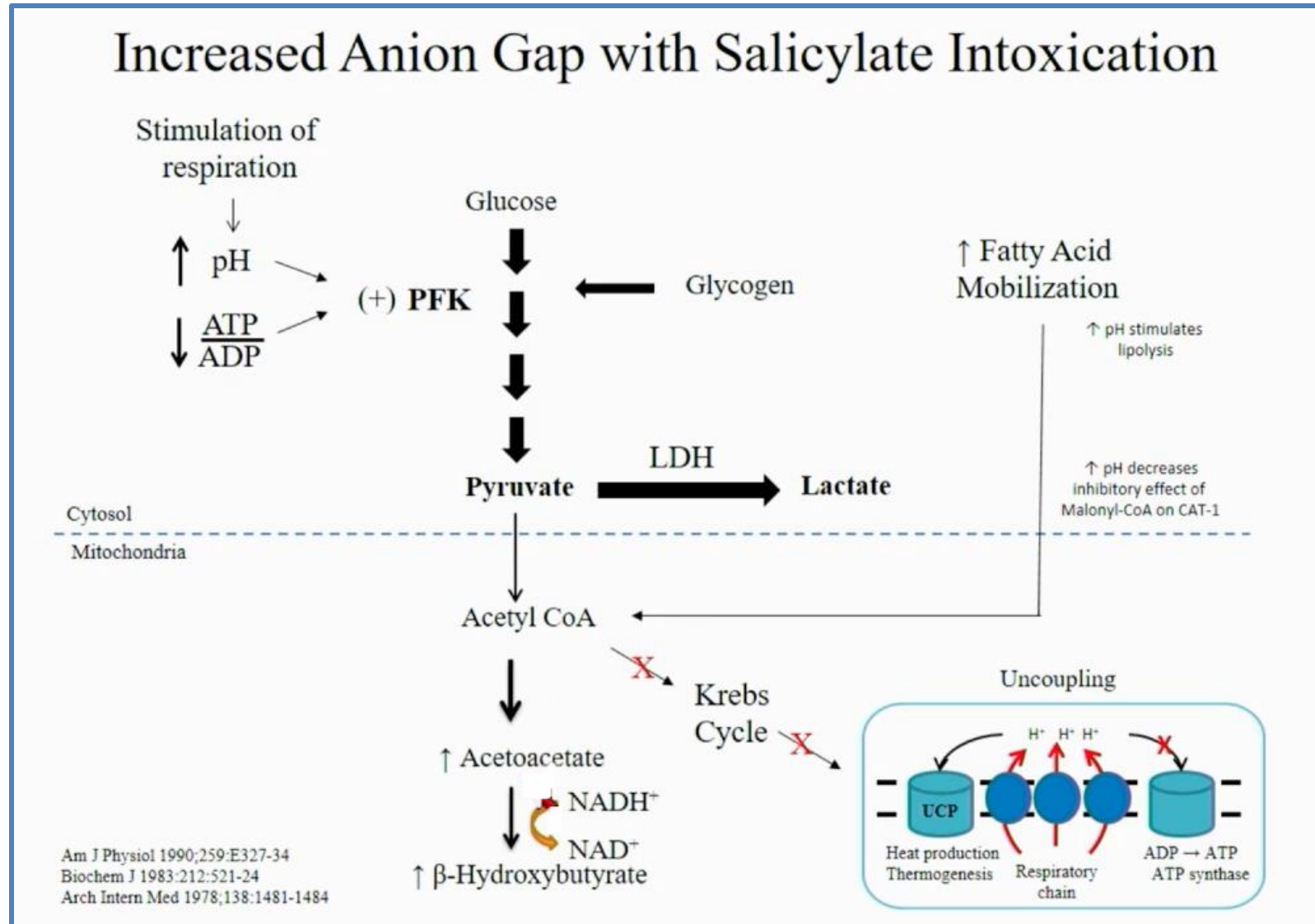
Ketoacidosis

- Diabetic
- Alcoholic
- Starvation

Lactic acidosis

- Type A
- Type B
- D Lactic acidosis

Salicylate Intoxication



Salicylate intoxication

Respiratory alkalosis + AG Metabolic Acidosis

○Symptoms

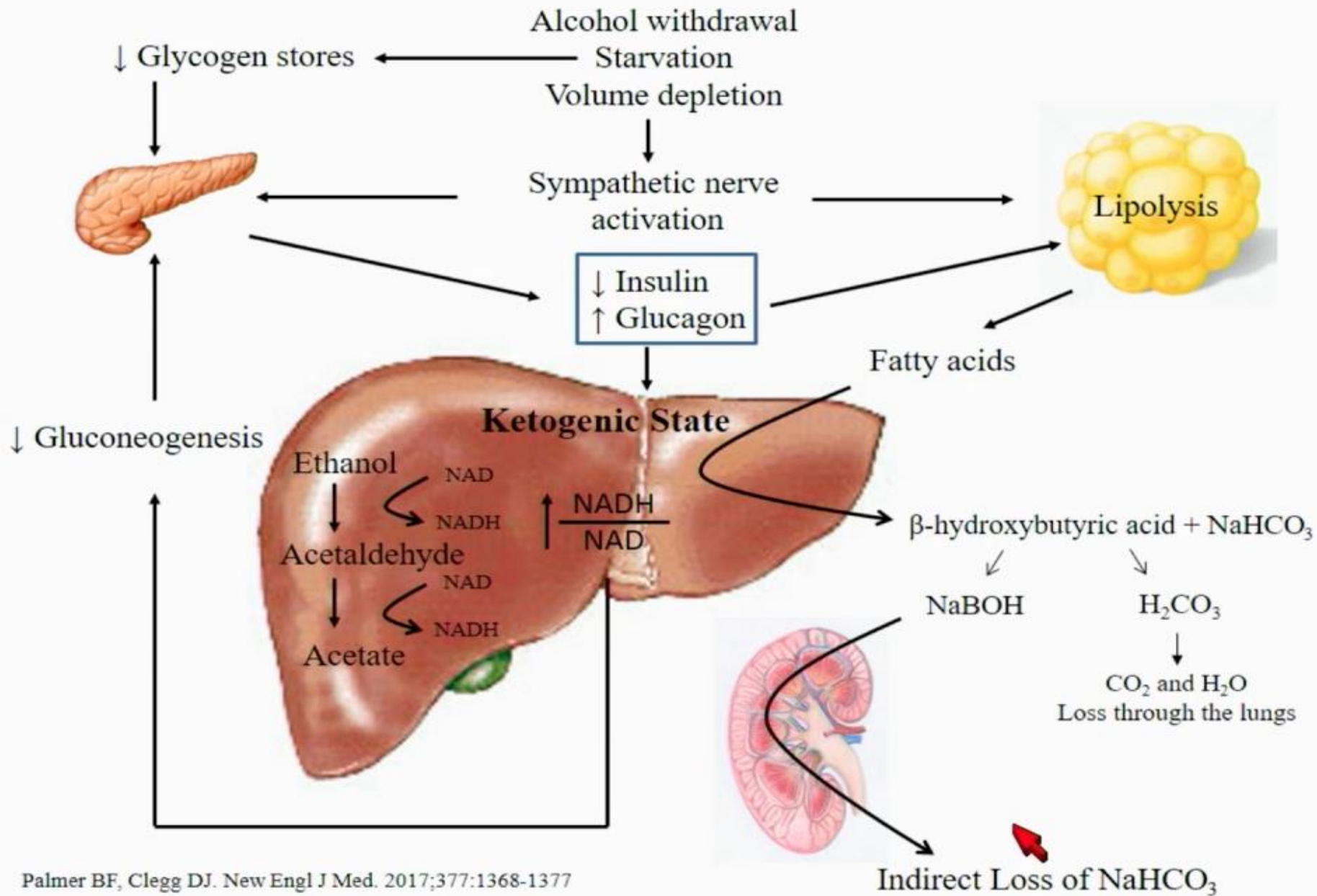
- Presents with CNS (cerebral edema, seizures , AMS, coma, neuroglycopenia)
- Volume overload (**non cardiogenic Pulmonary edema**, also seen in opioid intoxication) , AKI, tachypnea, tinnitus , N,V, hyperpyrexia
- Decrease reabsorption of uric acid in proximal tubule = **hypouricemia and hyperuricosuria**
- Hypokalemia
- **Elevated lactic acid , high production of ketones**

○Tto:

- **Alkalization** (IV sodium bicarbonate alone or + dialysis if indications) **goal keep pH 7.4-7.6**
Alkalinization will trap drug .
 - **Empiric use of dextrose (ASA can have CNS effect due to neuroglycopenia > even with normal serum glucose)**
- **Dialysis** (small V/ distribution) --->>>. At least 4-6h of **HD > then can do CRRT <<<-----**
ASA level **>100**,
ASA > 90 + persistent AKI,
Fluid overload/pulmonary edema,
AMS cerebral edema
Severe acidemia

Ketoacidosis

- Diabetic Ketoacidosis
- Alcoholic Ketoacidosis
- Starvation ketoacidosis



Palmer BF, Clegg DJ. *New Engl J Med.* 2017;377:1368-1377

• Diabetic ketoacidosis

- Potassium : If Serum K is low = really low K
 - Reduced level due to osmotic diuresis, but serum level can remain normal because potassium shifts out of cells in response to insulin deficiency and increased osmolality
 - Hyperglycemia solvent drag of K to serum
 - Hypovolemia : RAAS > aldosterone excrete K
 - Can also present as NAGMA at beginning & end
 - NAGMA: Beginning (when there is preserved kidney function there is loss of ketones in urine (ketones are converted into HCO₃ by liver if not excreted)
 - AGMA : when there is massive ketogenesis with volume contraction and AKI
 - NAGMA: fluids + insulin makes ketogenesis ceases due to increase ketones excretion
 - Tx:
 - Careful if you give bicarbonate cuz it can overshoot alkalosis (insulin/fluids will metabolize ketones to NaHCO₃) , Also can cause hypocalcemia
- Can be used in severe acidosis
NaHCO₃ only if pH <7.1
- Use LR as resuscitation (subgroup of SALT ED & SMART showed that DKA resolves 4h earlier with balance fluids (Acetate and lactate are metabolized to HCO₃)
 - Add D5W when glucose level 200-250mg/dl
- Don't stop insulin** , you can just cut it in 1/2 (you only stop for hypokalemia)

• Euglycemic DKA (SGLT2i, pregnancy, anorexia, chronic liver disease, ETOH ingestion)

- **Glucose level <250**, metabolic acidosis and ketosis (**LA is normal**)
For euglycemic DKA treatment is the same just need to more dextrose

Alcoholic ketoacidosis

- Decrease insulin secretion > increase other hormones (glucagon Epi, cortisol)> lipolysis > ketones production > acetoacetate > metabolize to B hydroxy by NADH = more lactate
 - Tto:
 1. **Thiamine** >to help metabolization of pyruvate to energy and also to decrease risk of Wernicke encephalopathy (if D5 is given it will consume thiamine)
 2. D5-NS
 - i. Treatment with **dextrose** will increase insulin and decrease glucagon secretion and will also stop ketosis, while saline will repair any volume deficit
 - Careful D5 can worsen hypophosphatemia given that will raise insulin and will increase PO4 intake
- Bicarb infusion almost never used, careful with hypocalcemia = can worsen **hypocalcemia**

Starvation ketoacidosis

- Decrease insulin secretion (starvation) > increase other hormones (glucagon Epi, cortisol)> lipolysis > ketones production > acetoacetate > metabolize to B hydroxy by NADH

Hypercarbia page
 Central (\downarrow RR)
 CW/Pleura/NMS (\downarrow V_T)
 Lung/Airway (\uparrow V_D)

Hypercarbia page
 Central: anxiety, CVA, pain
 Systemic: pregnancy, fever
 cirrhosis, sepsis, hyperthyroid
 Drugs: ASA (early), steroids,
 β -agonists, progesterone

\uparrow HCO₃⁻ in renal failure (Ca-alkali sx)
AND
Maintenance: preserve V & K over pH
 \downarrow V \rightarrow \uparrow ATII \rightarrow \uparrow PCT HCO₃ reabsorption
 \uparrow aldo \rightarrow \uparrow α -cell H⁺ secretion
 \downarrow Cl \rightarrow \uparrow MD activity \rightarrow \uparrow aldo & H⁺ secretion
 \uparrow β -cell Cl/HCO₃ exchange
 \downarrow K \rightarrow \uparrow K⁺/H⁺ exchange
 \uparrow α -cell H⁺-ATPase secretion

Glycols (ethylene/propylene)
Oxoproline (acetaminophen)
L-lactate
D-lactate (short gut)
Methanol
Aspirin (late)
Renal failure
Ketoacidosis (EtOH, DKA, starvation)
Sodium thiosulfate

Lactic acidosis:
A tissue hypoxia
B no tissue hypoxia
 sepsis, cancer, EtOH,
 meds (metformin, NRTI,
 linezolid, salicylates)
D short-bowel

Salicylates

\checkmark AG

HAGMA

NAGMA

\checkmark Urine AG
 (Na + K - Cl)

Acidosis \rightarrow Relieved by
 NH₄⁺ excretion, which is
 represented by Cl⁻

Positive

Negative

Impairment in
 NH₄⁺ excretion

Appropriate
 NH₄⁺ excretion

\downarrow urine NH₄⁺
Early renal failure
RTA: distal (\downarrow K),
 hypoaldo (\uparrow K), proximal

\uparrow urine NH₄⁺
 Diarrhea, saline, fistula,
 ureteral diversion,
 acetazolamide

UCI

< 20

> 40

Saline Responsive:
 Vomiting/NGT suction,
 post hypercapnia, CF,
 chloridorrhea

Saline Resistant:
HTN: \uparrow aldo (Conn, RAS),
 Cushing, Liddle's, licorice,
 serum K < 2
Hypotension:
 Bartter (loop), Gitelman
 (thiazide)
Urine K < 20: laxatives
Urine K > 30: diuretics

\checkmark Δ/Δ

<1

1-2

>2

AG + non gap

Pure HAGMA

AG + met alk

Ingestion?

Yes

\checkmark Osm Gap

OG < 10

OG > 10

\checkmark Volatile
 Panel

AG	OG	Ingestions	Toxin	Manifestations
\uparrow	\uparrow	Methanol	Formic acid	Δ MS, blurry vision, pupil dilation, papilledema
		Ethylene glycol	Oxalic acid	Δ MS, \downarrow Ca, Ca oxalate crystals \rightarrow AKI
		Propylene glycol	Lactic acid	AKI, liver injury
		Diethylene glycol	Diglycolic acid	AKI, n/v, pancreatitis, neuropathy, \uparrow lactate
nl/ \uparrow	\uparrow	Isopropyl alcohol	Acetone	Δ MS, fruity breath, pancreatitis, \uparrow lactate
		Ethanol	Acetaldehyde	Keto/lactic acidosis \pm met. alk 2/2 emesis

Non Anion Gap Metabolic Acidosis (NAGMA)

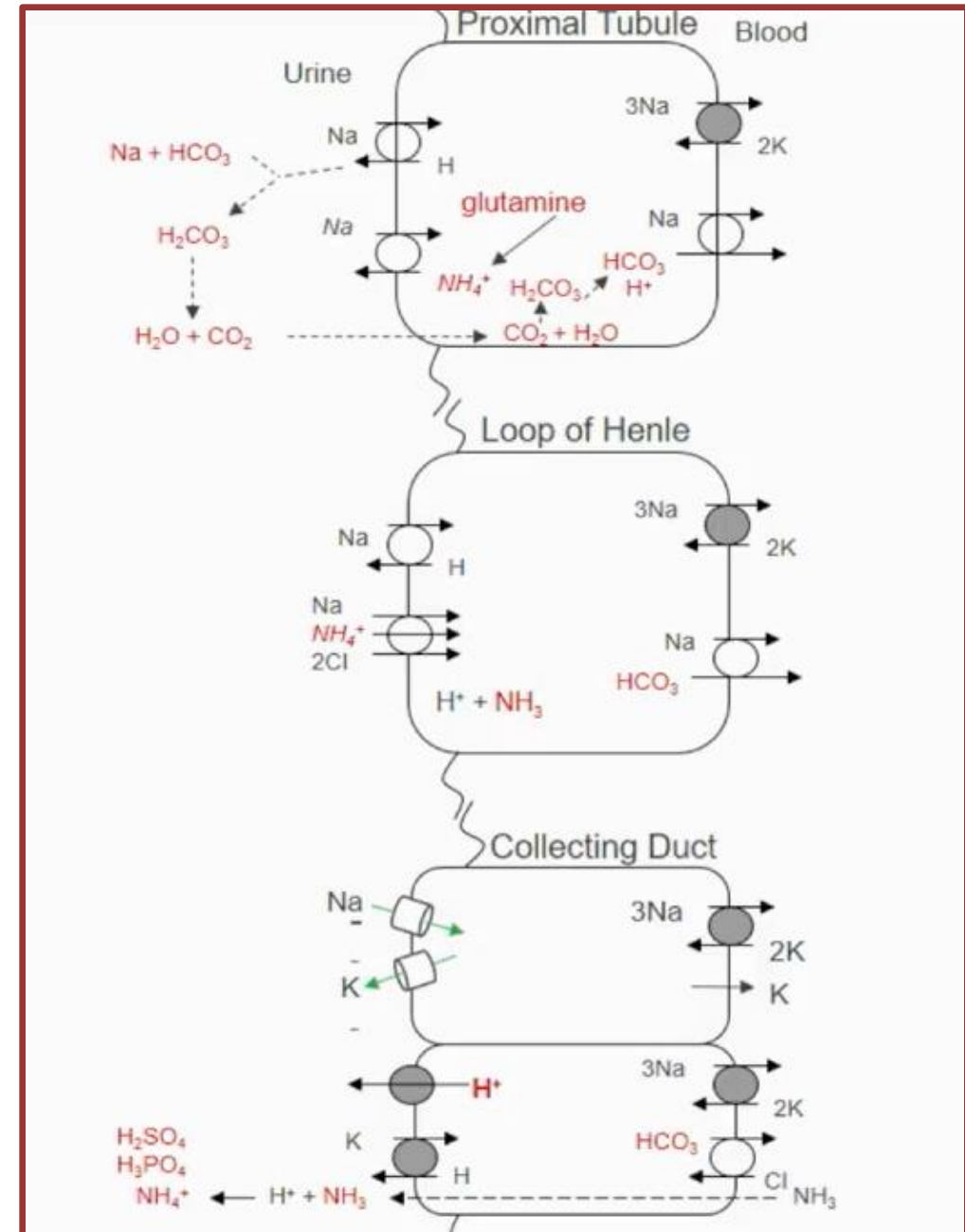
Causes **NAGMA**

- HAARDUPS
 - Hyperalimentation
 - Acetazolamide or any CA Inhibitor
 - Amphotericin B
 - RTA
 - Diarrhea
 - Ureterosigmoidostomy
 - Post hypocapneic state, pancreatic fistula
 - Sulfamylon

NAGMA

Role of the kidney (Renal vs. non-Renal)

- Reclamation of filtered bicarbonate (PCT / DCT)
- Impaired H secretion (DCT)
- Ammonia production and excretion increases with dietary acid load (protein)



68 y/o F w/hx of HTN, T2DM, p/w diarrhea for 3 days. Her medications include lisinopril, furosemide, spironolactone, and recent use of amoxicillin-clavulanate for sinusitis. Has been drinking just electrolytes solutions. She reports some decrease urine output over the last 24h. labs in fig.

ABG pH 7.22, PCO2 20, PO2 102, HCO3 5.

Urine studies: Una 31, UK 12, UCI 60

What can best explain the patient's acid base picture?

- a. Type 4 RTA
- b. Starvation ketosis
- c. Amoxicillin – clavulanate related LA
- d. Diarrhea

Sodium	133 mEq/L	133 mmol/L
Potassium	3.8 mEq/L	3.8 mmol/L
Chloride	118 mEq/L	118 mmol/L
Bicarbonate	5 mEq/L	5 mmol/L
BUN	50 mg/dL	17.9 mmol/L
Creatinine	3.5 mg/dL	309.4 mmol/L
Glucose	185 mg/dL	10.27 mmol/L

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ABG pH 7.22, PCO2 20 PO2 102 HCO3 5

Acidosis

Na: 133 / Cl: 118 / HCO3: 5

AG = [Na] – (Cl + HCO3)

10 (nl 10-12)

Non Anion Gap Metabolic Acidosis

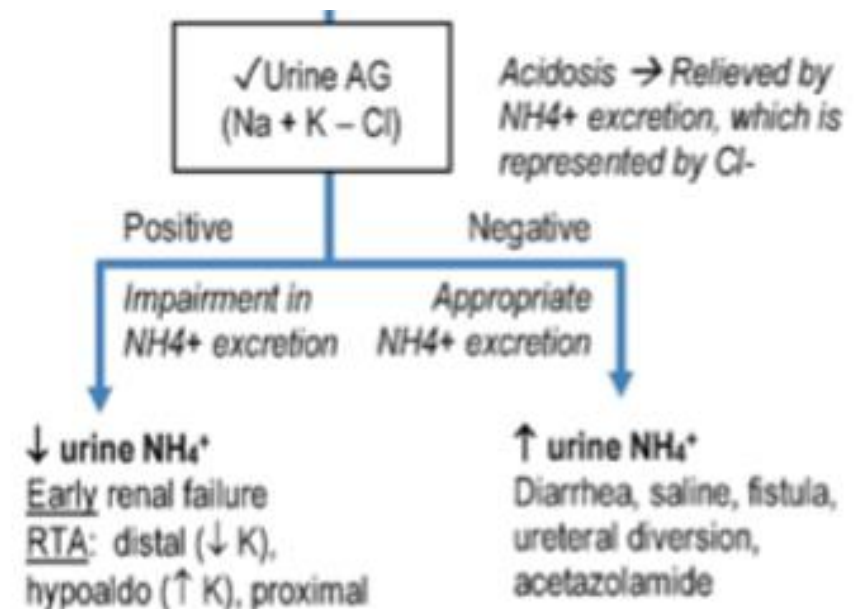
UAG= (Una + UK) – UCI

UAG = (31 + 12) – 60

UAG = -17

Diarrhea

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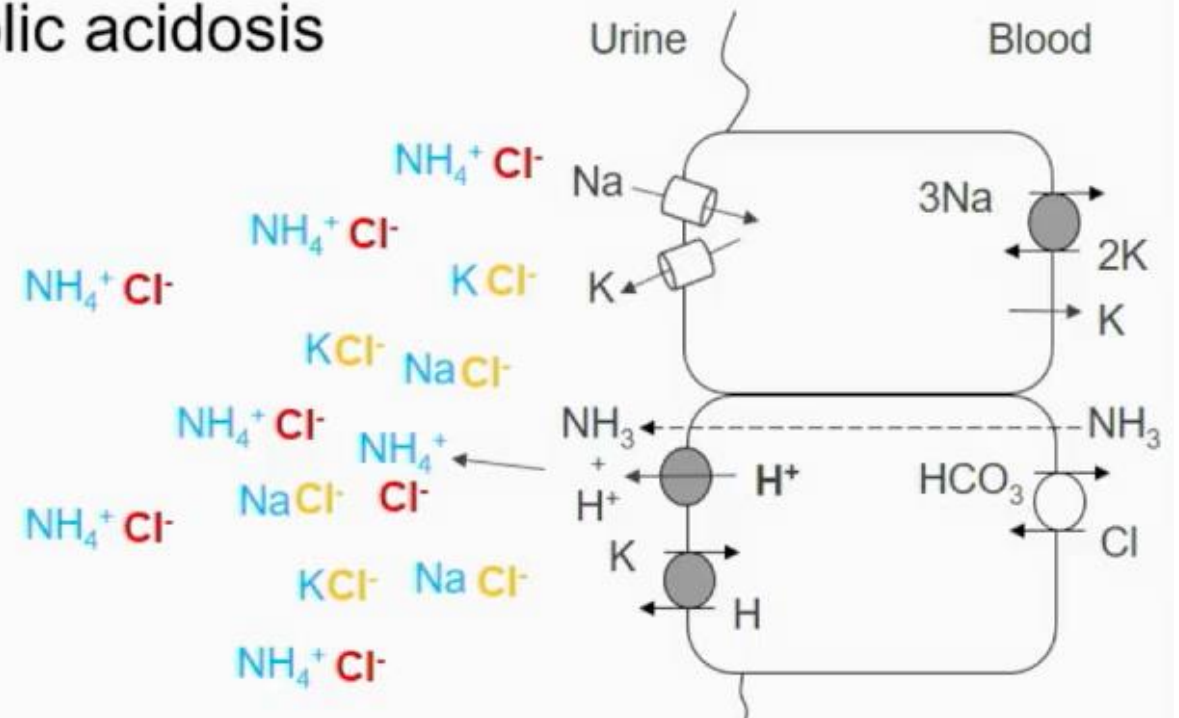


Renal vs non-Renal

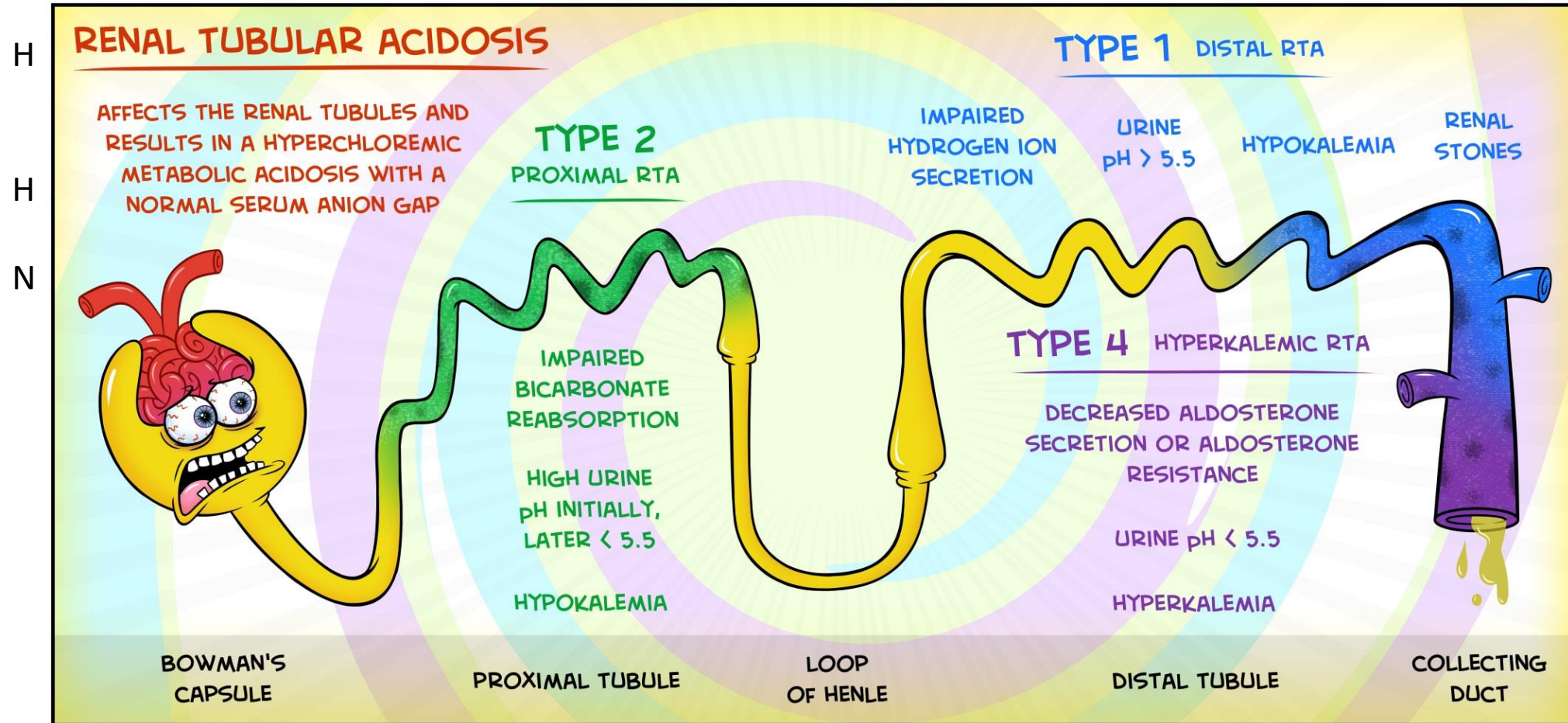
- Urine AG = $\text{Na} + \text{K} - \text{Cl}$
 - Reflects urinary NH_4^+ excretion (NH_4^+ coupled with Cl^-)

Interpretation: In the setting of a metabolic acidosis

- Positive urine AG
 - $(\text{Na} + \text{K}) > \text{Cl}$ “Low NH_4^+ ” excretion = Tubular dysfunction (Renal cause)
- Negative urine AG
 - $(\text{Na} + \text{K}) < \text{Cl}$ “High NH_4^+ ” excretion = Appropriate urinary NH_4^+ excretion (GI cause vs. urinary HCO_3^- loss)



Types of Renal acidosis



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A patient arrives to the ICU 1h after a Spinal Sx w/2.3 L of estimated blood loss and 4L of NS resuscitation during the procedure. ICU ABG: ph 7.28, PCO2 38, PO2 100. BMP in fig

What is the best way to approach this acid base disorder?

- Continue to monitor his electrolytes and pH
- Administer 50 mEq (1 amp) of NaHCO₃
- Administer an additional bolus of 1L NS
- Administer acetazolamide

ABG ph 7.28, PCO₂ 38 PO₂ 100

Acidosis

Na: 150 / Cl: 125 / HCO₃: 18

AG = [Na] – (Cl + HCO₃)

7 (nl 10-12)

Sodium	150 mEq/L	150 mmol/L
Potassium	4 mEq/L	4 mmol/L
Chloride	125 mEq/L	125 mmol/L
Carbon dioxide	18 mEq/L	18 mmol/L
Creatinine	1 mg/dL	88.4 μmol/L
Lactate	2 mmol/L	2 mmol/L

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Carbon dioxide	18 mEq/L	18 mmol/L
Creatinine	1 mg/dL	88.4 µmol/L
Lactate	2 mmol/L	2 mmol/L

Fluid composition	NS	LR	Plasmalyte
Na (mEq/L)	154	130	140
Cl (mEq/L)	154	109	98
K (mEq/L)	-	4	5
Ca (mEq/L)	-	2.7	-
Mg (mEq/L)	-	-	3
Buffer (mEq/L)	None	Lactate 28	Gluconate 23 Acetate 27
pH	5	6.5	7.4
SID	0	28	50
Price/L	\$1.30	\$1.66	\$7.12

Consideration	0.9% NS	LR	Plasma-Lyte
Can cause hyperkalemia*	✓	✗	✗
Can cause NAGMA	✓	✗	✗
Can cause lactic acidosis	✗	✗	✗

Metabolic alkalosis

- GI losses:
 - H⁺ losses (Vomiting, NGT), laxative abuse (hypokalemia), hereditary Cl losing diarrhea, use of PPI.
- Urinary:
 - Diuretic use / abuse
 - Loss ability to excrete HCO₃
 - Volume depletion w/ decreased HCO₃ filtration due to GFR decline
 - Proximal tubular Na and HCO₃ reabsorption increased
 - Cl depletion (inability to excrete HCO₃ in collecting duct)
 - K depletion (extracellular shift of K in exchange for H⁺)
- Exogenous administration alkali (milk alkali syndrome – Ca carbonate ingestion)
- Mineralocorticoid / glucocorticoid excess
- Hypokalemia
- Post hypercapnic metabolic alkalosis (seen in chronic CO₂ retainers with compensatory HCO₃ retention when MV increased)

Metabolic alkalosis

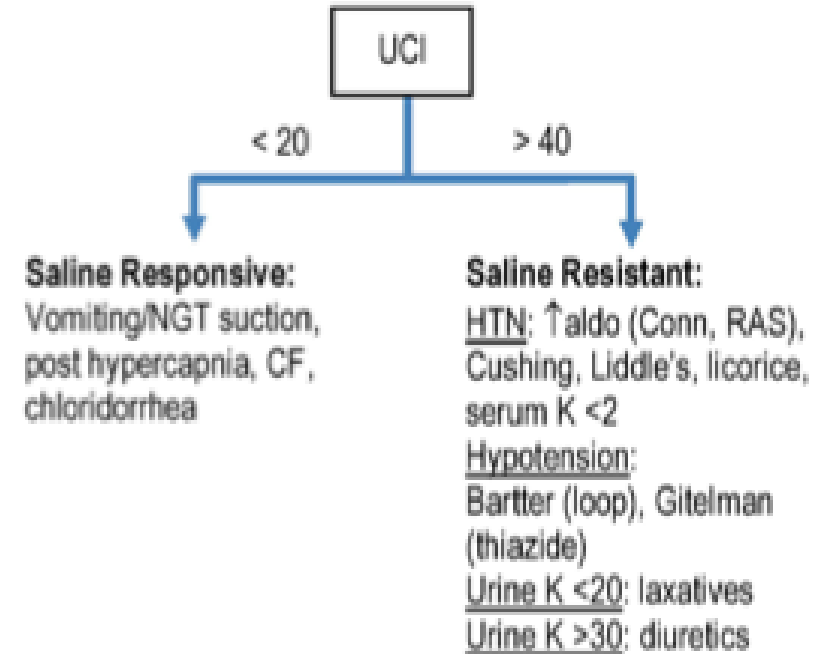
Interventions: Cl back?

Cl sensitive

Replace NaCl, replace Mg, K, stop offending agent diuretic / laxative, PPI for gastric losses, acetazolamide (ongoing diuretic requirement)

Cl resistant



Stop alkali , cause specific



Sodium Bicarbonate Therapy in Patients with Metabolic Acidosis

[María M. Adeva-Andany](#),* [Carlos Fernández-Fernández](#), [David Mouriño-Bayolo](#), [Elvira Castro-Quintela](#), and [Alberto Domínguez-Montero](#)

Sodium bicarbonate therapy for patients with severe metabolic acidaemia in the intensive care unit (BICAR-ICU): a multicentre, open-label, randomised controlled, phase 3 trial

[Prof Samir Jaber, MD](#)   • [Prof Catherine Paugam, MD](#) • [Prof Emmanuel Futier, MD](#) • [Prof Jean-Yves Lefrant, MD](#) • [Prof Sigismund Lasocki, MD](#) • [Prof Thomas Lescot, MD](#) • et al. [Show all authors](#) • [Show footnotes](#)

[Sanniya Khan Ghauri](#),^{✉1} [Arslaan Javaeed](#),² [Khawaja Junaid Mustafa](#),¹ [Anna Podlasek](#),³ and [Abdus Salam Khan](#)¹

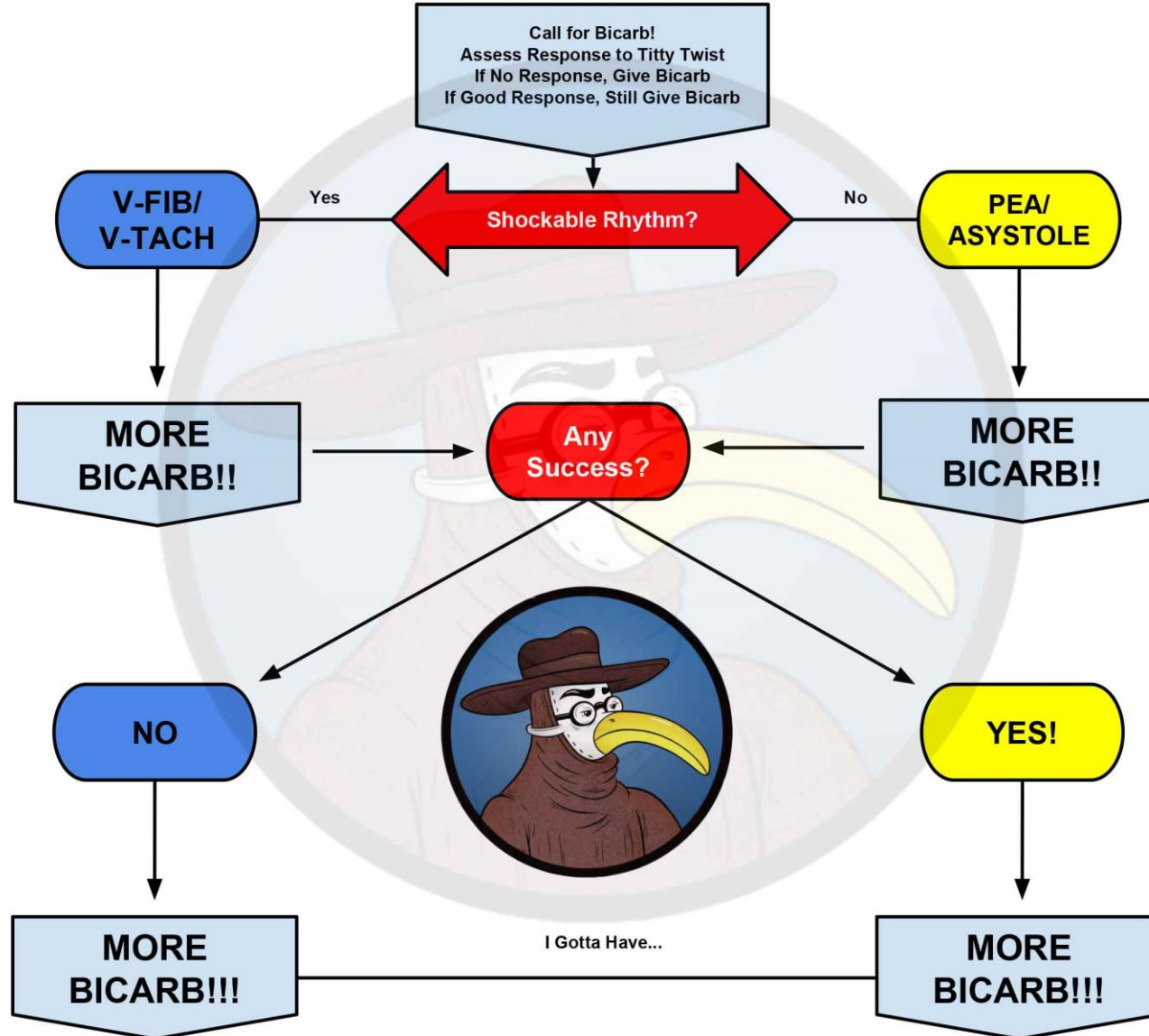
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- Sodium bicarbonate had no effect on the primary composite outcome
- Metabolic alkalosis, hypernatremia, and hypocalcemia were observed more frequently in the bicarbonate group than in the control group
- BICAR ICU trial: ICU patients with severe metabolic acidosis did not have improvement in all cause mortality at day 28 or the presence of >1 organ failure at day 7. However, there was a reduction of AKI (in pre specified group)

Sodium bicarbonate on severe metabolic acidosis during prolonged cardiac arrest randomized, placebo-controlled trial

Shin Ahn^{1*}, Youn-Jung Kim¹,
Donnino², Won Young Kim¹

CARDIAC ARREST ALGORITHM



	7/14/2022 1252	7/14/2022 1223	7/14/2022 1223
BASIC CHEMISTRY			
Sodium	169 ▲	150 ▲	150 ▲
Potassium	6.4 ▲		7.2 ▲
Chloride	122 ▲		110 ▲
Bicarbonate (TCO2)			9 ▼
Anion Gap (calc.)			31 ▲
Glucose	414 ▲		551 ▲
BUN			64 ▲
Creatinine	8.44 ▲		9.13 ▲
Glomerular Filtrat...	8* ▼		7* ▼
BUN/Creatinine			7.01
Osmolality, serum			
Osmolality (calc)			357
Calcium			6.3 ▼
Calcium, Ionized	3.1 ▼		
Phosphorus			11.1 ▲
Magnesium			
Bilirubin, Direct			
Bilirubin, Indirec...			
Bilirubin, Total			0.6
Alkaline Phosphatase			84
AST (SGOT)			636 ▲
ALT (SGPT)			166 ▲
Lipase			
Lactic Acid			18.0 ▲
Lactic Acid, POC	>20.0 ▲		
PROTEINS			
Protein, Total			4.8 ▼
Albumin			2.1 ▼
Albumin / Globulin...			0.78
Globulin (calc.)			2.7

	7/14/2022 0720	7/13/2022 2336	7/13/2022 1944	7/13/2022 1218
BASIC CHEMISTRY				
Sodium	146 ▲	138	132 ▼	125 ▼
Potassium	4.5	4.4	5.2 ▲	5.4 ▲
Chloride	106	93 ▼	84 ▼	75 ▼
Bicarbonate (TCO2)	17 ▼	19 ▼	23	27
Anion Gap (calc.)	23 ▲	26 ▲	25 ▲	23 ▲



Normal Solutions	Percent Solution	NaCl Concentration
	5% (50 g/L)	854 mEq/L
	3% (30 g/L)	513 mEq/L
	2% (20 g/L)	342 mEq/L
normal	0.9% (9 g/L)	154 mEq/L
half normal	0.45% (4.5 g/L)	77 mEq/L
quarter normal	0.225% (2.25 g/L)	38 mEq/L
	0.20% (2.0 g/L)	34 mEq/L

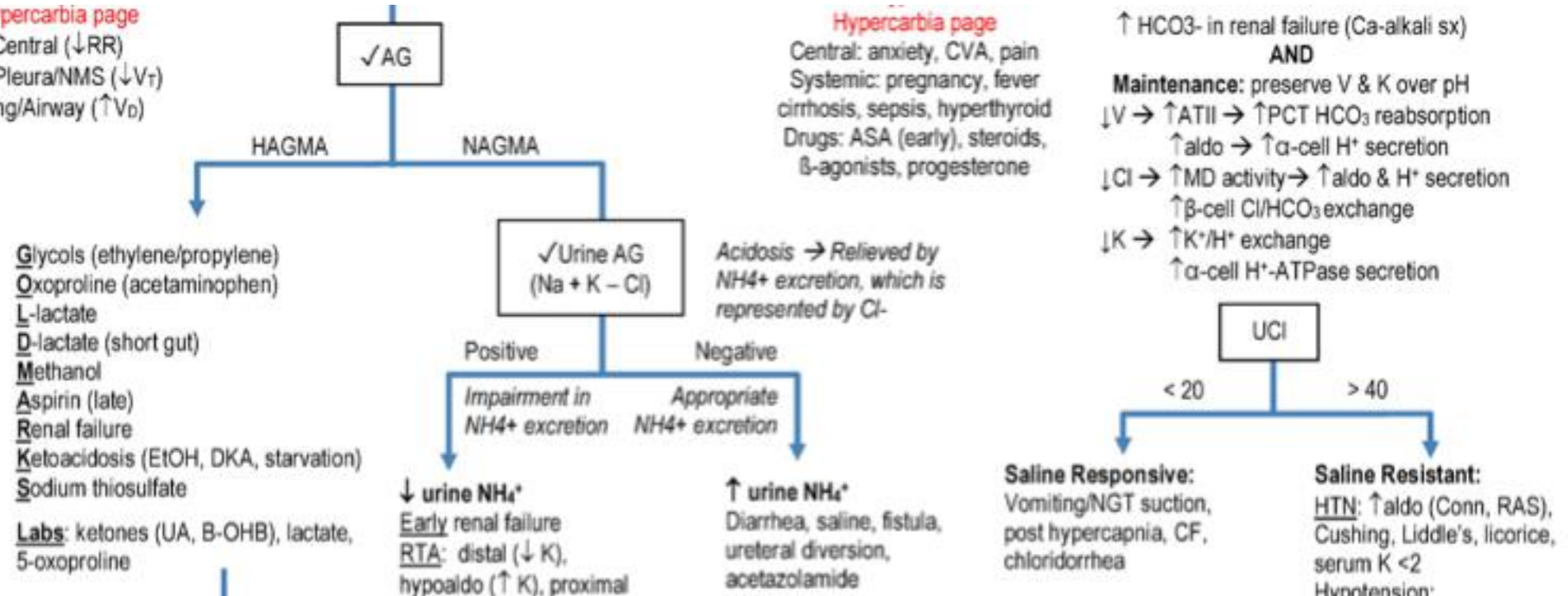
Complications of Bicarbonate Therapy

- Overshoot alkalosis
- Exogenously administered NaHCO_3 must be added to endogenously produced by metabolism of ketones, lactate, etc
- Increase in lactate generation
- Volume expansion with ARF and ESRD
- Increase CO_2 production
- Hypocalcemia

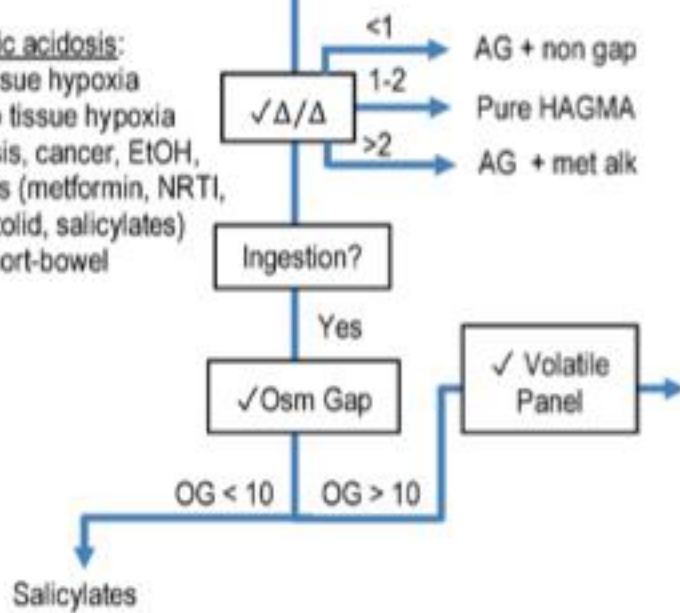
Hypercarbia page
 Central (\downarrow RR)
 CW/Pleura/NMS (\downarrow V_T)
 Lung/Airway (\uparrow V_D)

Hypercarbia page
 Central: anxiety, CVA, pain
 Systemic: pregnancy, fever, cirrhosis, sepsis, hyperthyroid
 Drugs: ASA (early), steroids, β -agonists, progesterone

\uparrow HCO₃⁻ in renal failure (Ca-alkali sx)
AND
Maintenance: preserve V & K over pH
 \downarrow V \rightarrow \uparrow ATII \rightarrow \uparrow PCT HCO₃ reabsorption
 \uparrow aldo \rightarrow \uparrow α -cell H⁺ secretion
 \downarrow Cl \rightarrow \uparrow MD activity \rightarrow \uparrow aldo & H⁺ secretion
 \uparrow β -cell Cl/HCO₃ exchange
 \downarrow K \rightarrow \uparrow K⁺/H⁺ exchange
 \uparrow α -cell H⁺-ATPase secretion



Lactic acidosis:
A tissue hypoxia
B no tissue hypoxia
 sepsis, cancer, EtOH, meds (metformin, NRTI, linezolid, salicylates)
D short-bowel



AG	OG	Ingestions	Toxin	Manifestations
\uparrow	\uparrow	Methanol	Formic acid	Δ MS, blurry vision, pupil dilation, papilledema
		Ethylene glycol	Oxalic acid	Δ MS, \downarrow Ca, Ca oxalate crystals \rightarrow AKI
		Propylene glycol	Lactic acid	AKI, liver injury
		Diethylene glycol	Diglycolic acid	AKI, n/v, pancreatitis, neuropathy, \uparrow lactate
nl/ \uparrow	\uparrow	Isopropyl alcohol	Acetone	Δ MS, fruity breath, pancreatitis, \uparrow lactate
		Ethanol	Acetaldehyde	Keto/lactic acidosis \pm met. alk 2/2 emesis

Thanks.