King Saud University

Acid base balance

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Objectives

At the end of this tutorial participant will be able to:

- State the normal value for PH,PCO2,HCO3
- Understand the basic mechanism of acid base disturbance
- Interpret basic acid base disturbance
- List common differential diagnosis for different acid base disorder

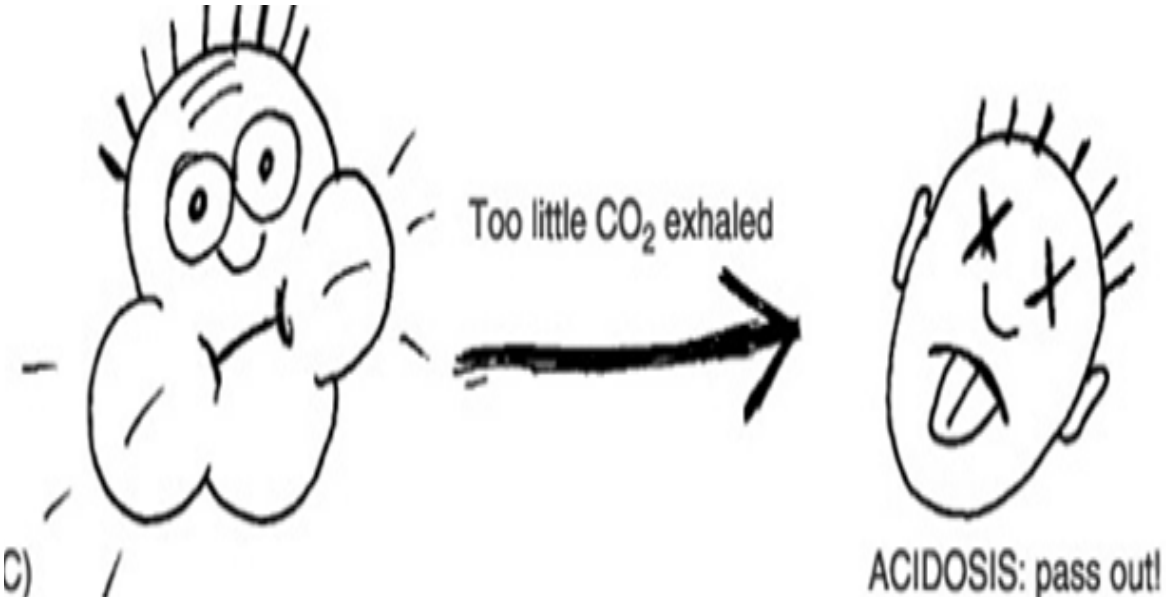
Normal Value

- Normal arterial blood pH = 7.35 – 7.45
- P_aCO_2 = 35-45
- Serum HCO_3^-.= 22-26
- Anion gap = 8-12

primary disturbance

Primary Disorder				
Primary Disorder	Problem	pH	HCO_3	P_aCO_2
Metabolic acidosis	gain of H^+ or loss of HCO_3	↓	↓	↓
Metabolic alkalosis	gain of HCO_3 or loss of H^+	↑	↑	↑
Respiratory acidosis	hypoventilation	↓	↑	↑
Respiratory alkalosis	hyperventilation	↑	↓	↓

Respiratory acidosis

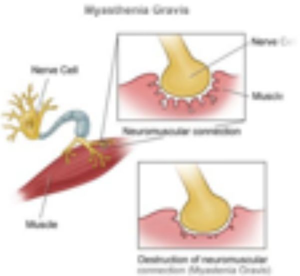


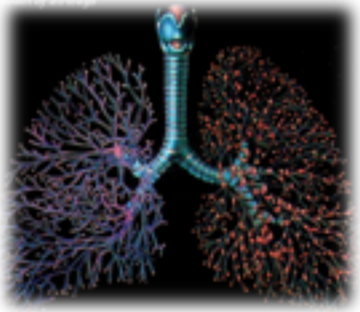
Respiratory acidosis

- Primary mechanism:

  Hypoventilation

- CNS
- Peripheral nerve
- Neuro muscular junction
- Chest wall
- Bronchial tree





Acute respiratory acidosis

Causes:

- Respiratory pathophysiology - airway obstruction, severe pneumonia, chest trauma/pneumothorax
- Acute drug intoxication (narcotics, sedatives)
- Residual neuromuscular blockade
- CNS disease (head trauma)

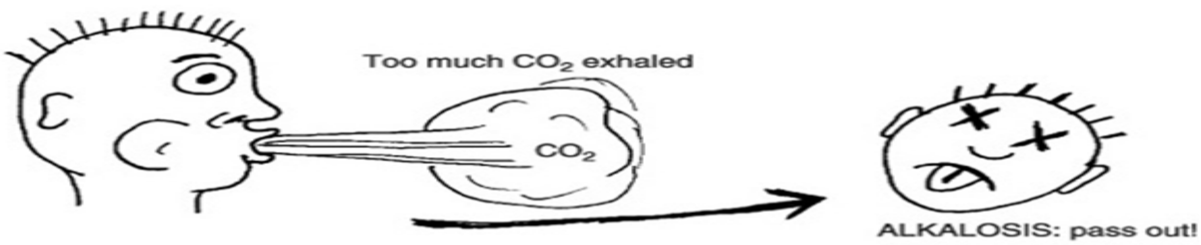
Chronic Respiratory Acidosis

- paCO_2 is elevated with a pH in the acceptable range
- Renal mechanisms increase the excretion of H^+ within 24 hours and may correct the resulting acidosis caused by chronic retention of CO_2 to a certain extent

Chronic Respiratory Acidosis
Causes

- Chronic lung disease (COPD)
- Neuromuscular disease
- Extreme obesity
- Chest wall deformity

Respiratory alkalosis

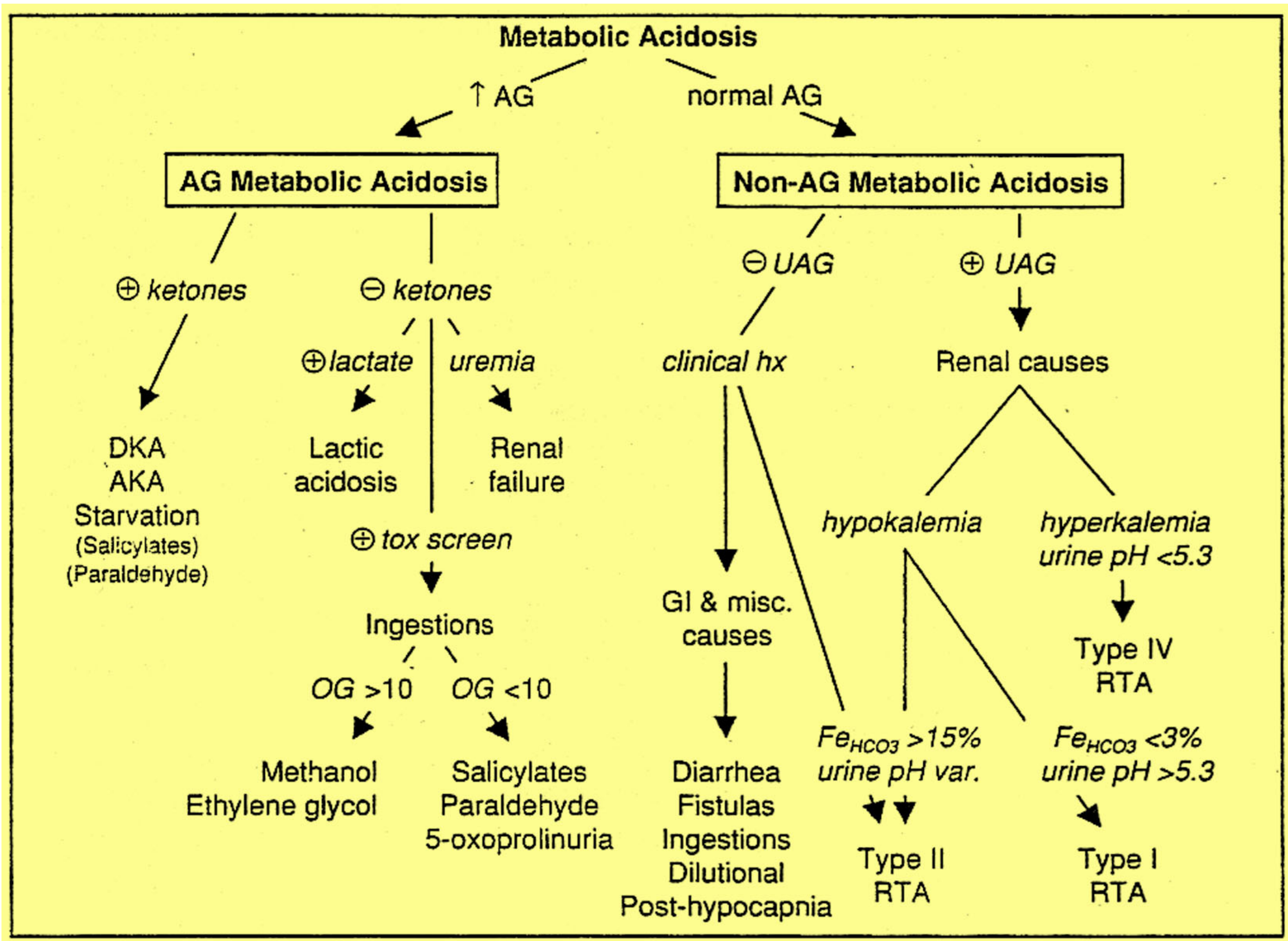


Respiratory Alkalosis

- Pain
- Drugs
- Sepsis
- Fever
- Thyrotoxicosis
- Pregnancy
- Overaggressive mechanical ventilation
- Hepatic failure
- Anxiety
- Hypoxemia
- Restrictive lung disease

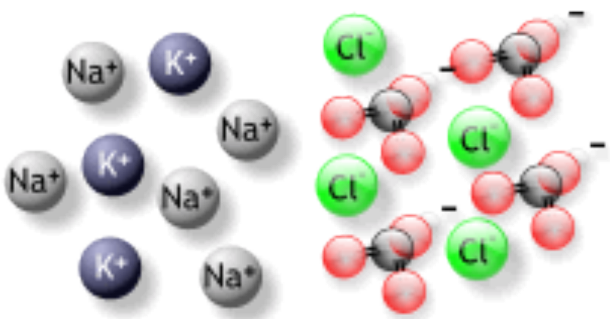
Metabolic acidosis

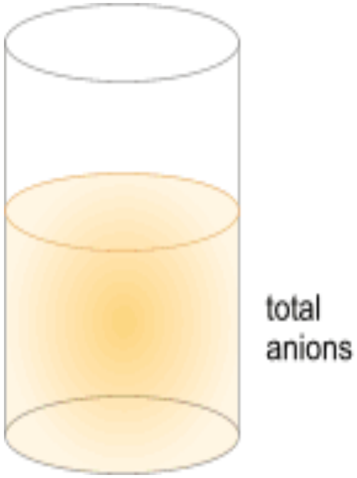
- Increase acid production
- Decrease acid excretion
- Loss of bicarbonate


Metabolic Acidosis
↑ AG
normal AG
AG Metabolic Acidosis
Non-AG Metabolic Acidosis
⊕ ketones
⊖ ketones
⊖ UAG
⊕ UAG
⊕ lactate
uremia
clinical hx
Renal causes
DKA
AKA
Starvation
(Salicylates)
(Paraldehyde)
Lactic acidosis
Renal failure
⊕ tox screen
Ingestions
OG >10
OG <10
Methanol
Ethylene glycol
Salicylates
Paraldehyde
5-oxoprolinuria
hypokalemia
hyperkalemia
urine pH <5.3
Type IV RTA
GI & misc. causes
Diarrhea
Fistulas
Ingestions
Dilutional
Post-hypocapnia
Fe_{HCO3} >15%
urine pH var.
Fe_{HCO3} <3%
urine pH >5.3
Type II RTA
Type I RTA

Metabolic acidosis

- Anion gap = [Sodium] - ([Chloride] + [Bicarbonate]) Or
  AG = $[Na^+] - ([Cl^-] + [HCO_3^-])$.
- OR
  Anion gap = $([Na^+] + [K^+]) - ([Cl^-] + [HCO_3^-])$
  Anion gap = cations - anions



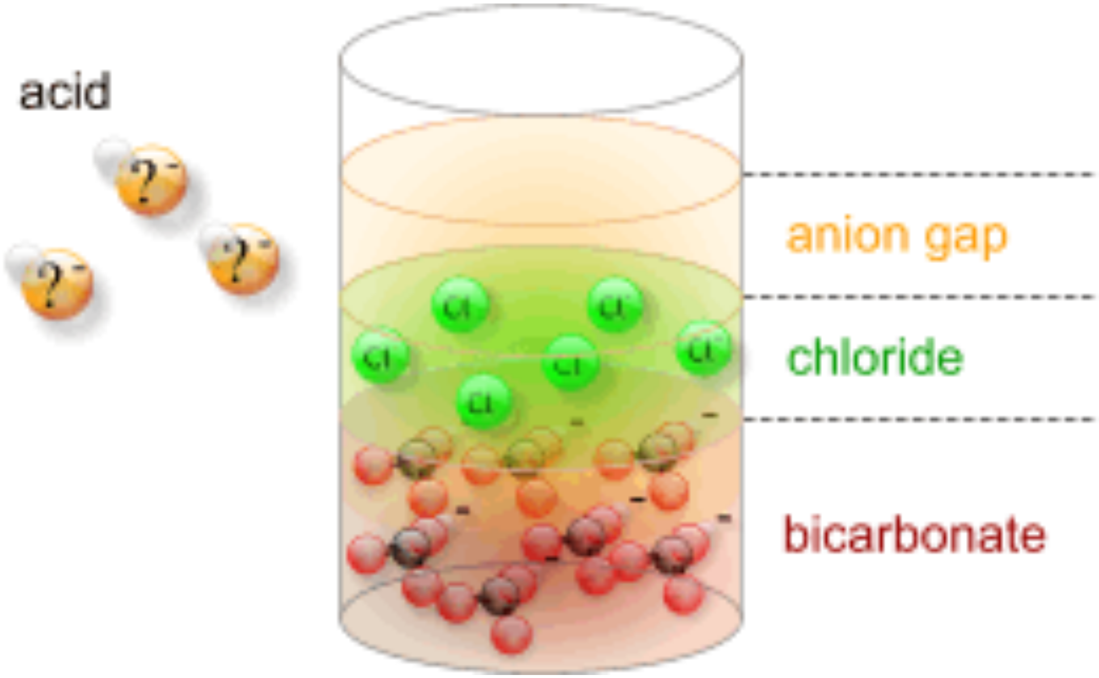


Increased anion gap metabolic acidosis

- Methanol other alcohols, and ethylene glycol intoxication
  Uremia (renal failure)
  Lactic acidosis
  Ethanol
  Paraldehyde and other drugs
  Aspirin
  Ketones (starvation, alcoholic and diabetic ketoacidosis)

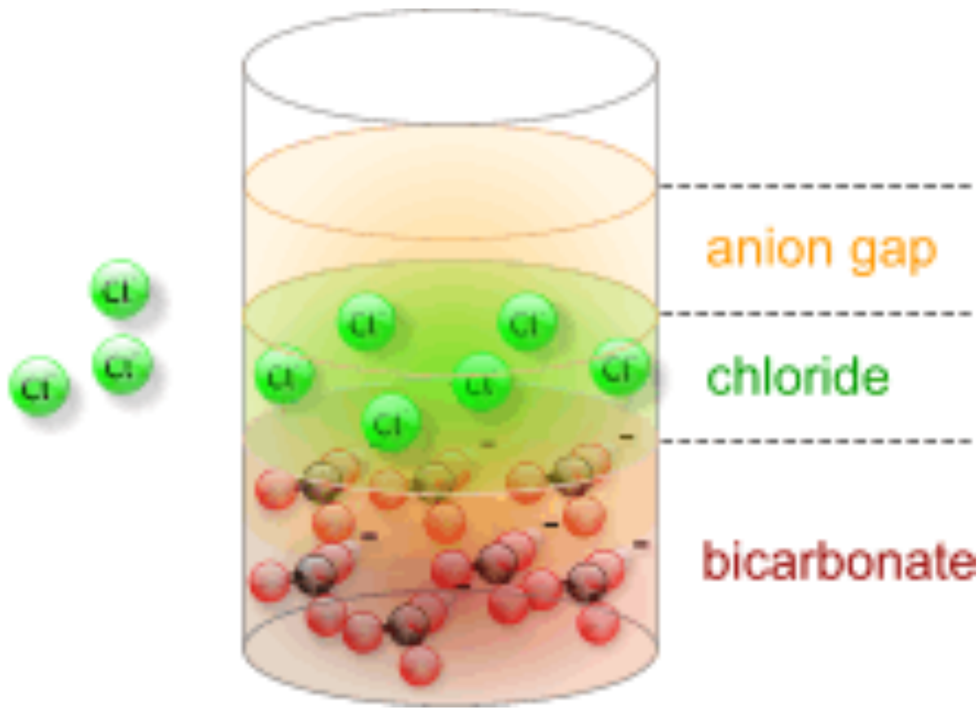
Anion Gap

High AG



Anion Gap

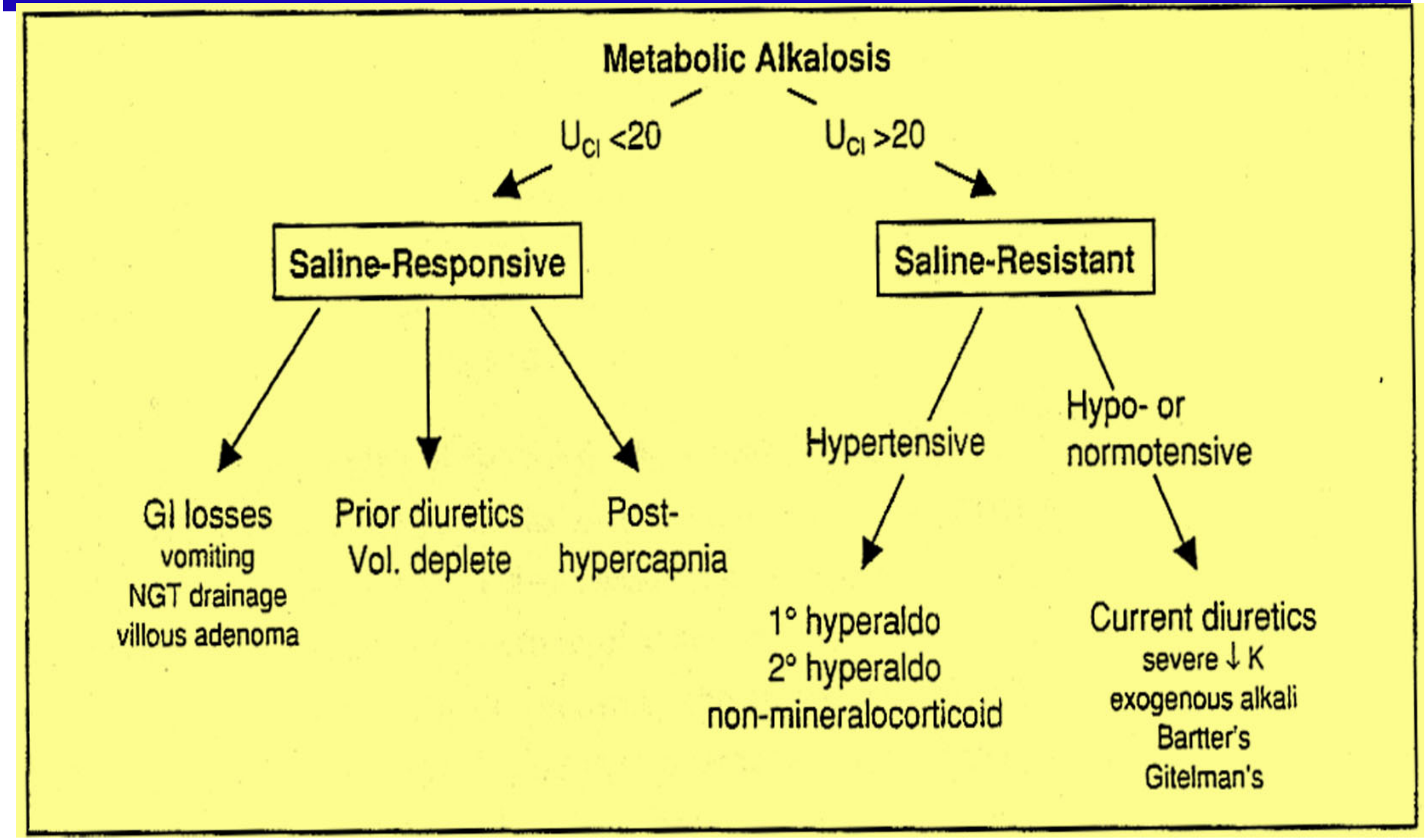
Normal AG



Etiologies of AG Metabolic Acidosis	
Ketoacidosis	Diabetes mellitus**, alcoholism, starvation**
Lactic acidosis	Type A**: impairment in tissue oxygenation, eg.** Circulatory or Respiratory failure, sepsis, **ischemic bowel, carbon monoxide** Type B: **no impairment in tissue oxygenation, eg. Malignancy, alcoholism,** **meds (metformin, NRTIs, salicylates)** D-lactic acidosis: **short bowel syndrome → glc metab by colonic bacteria** **To D-lactate, which is absorbed; not detected by standard lactate assay**
Renal failure	**Accumulation of organic anions such as phosphates, sulfates, etc.**
Ingestions	Methanol: **manifestations include blurred vision** Ethylene glycol: **manifestations include ∆MS, cardiopulmonary failure,** **calcium oxalate crystals and renal failure** Paraldehyde Salicylates: **metabolic acidosis (from lactate, ketones) + respiratory** **alkalosis due to stimulation of CNS respiratory center** Acetaminophen: **glutathione depletion accumulation of the** **Endogenous organic acid 5-oxoproline in susceptible host**

Etiologies of Non-AG Metabolic Acidosis	
GI losses of HCO_3	Diarrhea, intestinal or pancreatic fistulas or drainage
RTAs	See section on renal tubular acidoses below
Early renal failure	Impaired generation of ammonia
Ingestions	Acetazolamide, sevelamer, cholestyramine, toluene
Dilutional	Due to rapid infusion of bicarbonate-free intravenous fluids
Post-hypocapnia	Respiratory alkalosis → renal wasting of HCO_3 rapid correction → Of resp. alk. Transient acidosis until HCO_3; regenerated
Ureteral diversion	Colonic Cl^- /HCO_3^- exchange, ammonium reabsorption

Metabolic alkalosis



Etiologies of Metabolic Alkalosis

Etiologies of Metabolic Alkalosis	
Saline – responsive	GI loss of H^+ **: vomiting, NGT drainage, villous adenoma** Diuretic use **posthypercapina**
Saline – resistant	**Hypertensive** (mineralocorticoid excess) **1^0 hyperaldosteronism (eg. Conn's)** **2^0 hyperaldosteronism (eg, renovascular dis. Rennin-secreting tumor)** **Non-aldo (eg. Cushing's, Liddle's, exogenousmineralocorticoids)** **Normotensive** **Severe hypokalemia** **Exogenous alkali load** **Bartter's syndrome, Gitelman's syndrome**

primary disturbance

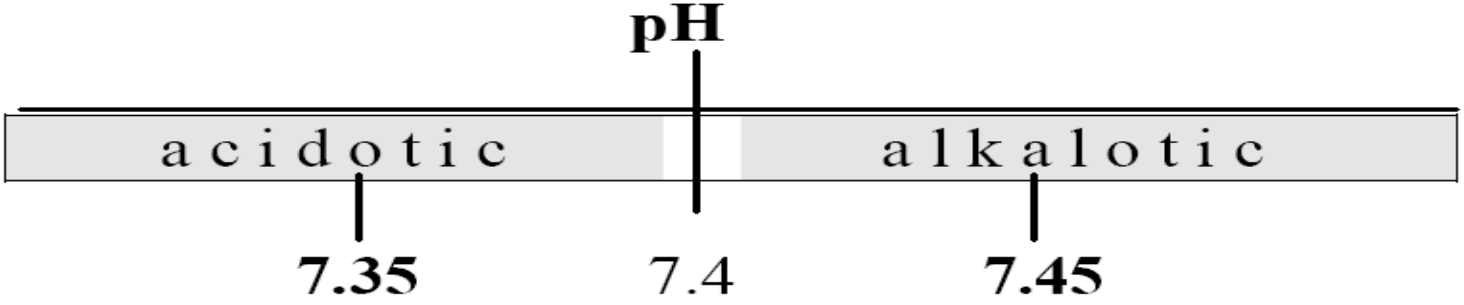
Primary Disorder				
Primary Disorder	Problem	pH	HCO_3	P_aCO_2
Metabolic acidosis	gain of H^+ or loss of HCO_3	↓	↓	↓
Metabolic alkalosis	gain of HCO_3 or loss of H^+	↑	↑	↑
Respiratory acidosis	hypoventilation	↓	↑	↑
Respiratory alkalosis	hyperventilation	↑	↓	↓

Steps in Acid-Base Analysis

- Step 1: Acidemic or Alkalemic?
- Step 2: Is the primary disturbance respiratory or metabolic?
- Step 3: Is the respiratory disturbance acute or chronic?
- Step 4: For a metabolic acidosis, is there an increased anion gap?
- Step 5: Are there other metabolic processes present in a patient with an increased anion gap metabolic acidosis?
- Step 6: Is the respiratory system compensating adequately for a metabolic disturbance

Step 1: Acidemic or Alkalemic?

- The pH of the arterial blood gas measurement identifies the disorder as alkalemic or acidemic.
- Normal arterial blood pH = 7.35 – 7.45
- Acidemic: pH < 7.35



Step 2: Is the primary disturbance respiratory or metabolic?

- To determine whether the disturbance affects primarily
  - The arterial P_aCO_2 or
  - The serum HCO_3^-.
- Respiratory disturbances alter the arterial P_aCO_2 (normal value 35-45)
- Metabolic disturbances alter the serum HCO_3^- (normal value 22-26)

Quiz?

	pH	pCO2	HCO3	Interpretation
1	7.41	40	24	
2	7.5	42	35	
3	6.72	40	5	
4	7.26	63	25	
5	7.52	18	25	

Quiz?

	pH	pCO2	HCO3	Interpretation
1	7.41	40	24	normal
2	7.5	42	35	metabolic alkalosis
3	6.72	40	5	metabolic acidosis
4	7.26	63	25	respiratory acidosis
5	7.52	18	25	respiratory alkalosis

Step 3: Is the respiratory disturbance acute or chronic?

- Acute respiratory acidosis:
  - HCO_3^- **increase by 1 mEq/l for every 10 mmHg increase in** PaCO2
- Chronic respiratory acidosis:
  - HCO_3^- **increase by 3-3.5 mEq/l for every 10 mmHg increase in** PaCO2
- Acute respiratory alkalosis:
  - HCO_3^- **decrease by 2 mEq/l for every 10 mmHg decrease in** PaCO2
- Chronic respiratory alkalosis:
  - HCO_3^- **decrease by 4-5 mEq/l for every 10 mmHg decrease in** PaCO2

Case study -1

- pH =7.2, pCO_2 = 60, HCO_2 = 24.

- What it is the primary problem? Compensation?
- Differential diagnosis?
- Treatment ?

Respiratory acidosis

- Is it acute or chronic?
- Note that the PH is abnormal
- Note the HCO2 is with in normal
- Remember:
- Acute respiratory acidosis:
  - HCO_3^- **increase by 1 mEq/l for every 10 mmHg increase in** PaCO2
- Chronic respiratory acidosis:
  - HCO_3^- **increase by 3-3.5 mEq/l for every 10 mmHg increase in** PaCO2

Case 2

- What do you expect the ABG in the following patients to be:
- 24 years old male with acute SOB, and wheezes for 2days.
- Past hx: Bronchial asthma
- 67 years old women, HTN,DMII, COPD presenting with cough and SOB

Case3

- pH: 7.25
- $[HCO_3^-]$: 20 mEq/L
- $PaCO_2$: 52 mmHg

- What it is the primary problem? Compensation?
- Differential diagnosis?

Case 4

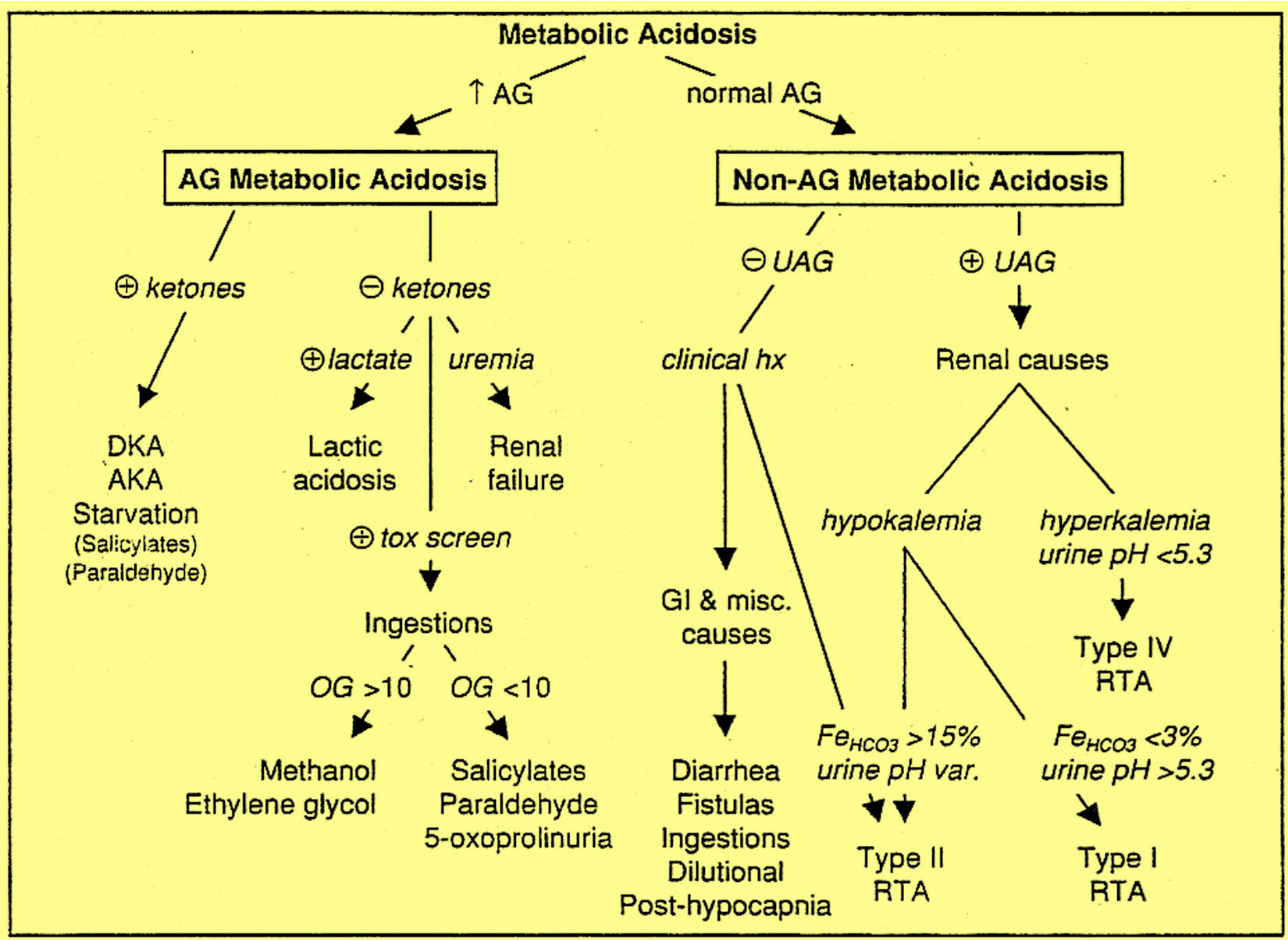
- pH: 7.32
- $[HCO_3^-]$: 19 mEq/L
- $PaCO_2$: 55 mmHg

- What it is the primary problem? Compensation?
- Differential diagnosis?
- What other investigation you want to do?

Step 4: For a metabolic acidosis, is there an increased anion gap?

- Anion gap = [Sodium] - ([Chloride] + [Bicarbonate]) Or
  AG = $[Na^+] - ([Cl^-] + [HCO_3^-])$.
- Normal AG 8-16

- Serum Osmolality = (2 x (Na + K)) + (BUN) + (glucose)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Ingestions
OG >10
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Ethylene glycol
Salicylates
Paraldehyde
5-oxoprolinuria
GI & misc. causes
Diarrhea
Fistulas
Ingestions
Dilutional
Post-hypocapnia
hypokalemia
hyperkalemia
urine pH <5.3
Type IV RTA
Fe_{HCO3} >15%
urine pH var.
Fe_{HCO3} <3%
urine pH >5.3
Type II RTA
Type I RTA

Step 5: Are there other metabolic processes present in a patient with an increased anion gap metabolic acidosis?

Step 6: Is the respiratory system compensating adequately for a metabolic disturbance

- Metabolic acidosis:
  - PCO2 decreases by 1 mmHg for every 1 mEq/l decrease in HCO3
- Metabolic alkalosis:

- PCO2 increases by 0.6 mmHg for every 1 mEq/l increases in HCO3

Case 5

- 56 yo M with Hx of COPD is admitted with 1-wk Hx of dyspnea, productive cough and diarrhea
  (Na) 125, (Cl) 103 , (BUN) 42, (Glucose) 100, (K) 3.5, (HCO3-) 10, (Creat) 1.4
- ABG 7.14 pCO_2 30 pO_2 50
- What is the predominant acid base disorder ?

Case 5 continue

- What pCO_2 is expected with normal respiratory compensation ? $40 – (1.2 * (24-10)) = 23.2$, this is not full compensation b/c pCO_2 is 30 – indicates an underlying primary respiratory acidosis, suggested by the Hx of COPD, dyspnea, and productive cough (lungs not able to appropriately compensate)
- What is the Anion Gap ? $125 – (103+10) = 12$ – normal AG ∴ etiology is either diarrhea or RTA – most likely diarrhea b/c of the history

Case 6

- 32 y/o male present w/ 2d Hx of intractable vomiting. ; pH 7.51, pCO_2 41

Na132, Cl 90 32 K3.4 HCo2= 33 creatinine1.6

- What is the predominant acid-base disorder? Alkalosis (Metabolic)

Case 6 continue

- What pCO_2 is expected w/ normal respiratory compensation? = 40 + (32 – 24) * (~ 0.6 ⇔ 0.7) = 44.8 ⇔ 45.6 mmHg; since the measured pCO_2 < 44.8 ⇔ 45.6, there is also a primary respiratory alkalosis (inappropriate hyperventilation)
- Tx: Isotonic saline to correct for volume depletion –

Case 7

- A 58- year old man presents to the Emergency Department with abdominal pain and hypotension. Investigation reveal the following:
- Na 140 K 4 Cl 90 HCO_3 = 5 PH 6.8 PCO_2 36 PO_2 7
- Analyze the acid-base disorder(s) seen in the patient.
-

Summary

- First, does the patient have an acidosis or an alkalosis
  - Look at the pH
- Second, what is the primary problem – metabolic or respiratory
  - Look at the pCO_2
  - If the pCO_2 change is in the opposite direction of the pH change, the primary problem is respiratory

Summary

- Third, is there any compensation by the patient - do the calculations
  - For a primary respiratory problem, is the pH change completely accounted for by the change in pCO_2
    - if yes, then there is no metabolic compensation
    - if not, then there is either partial compensation or concomitant metabolic problem