Acid base balance disorders

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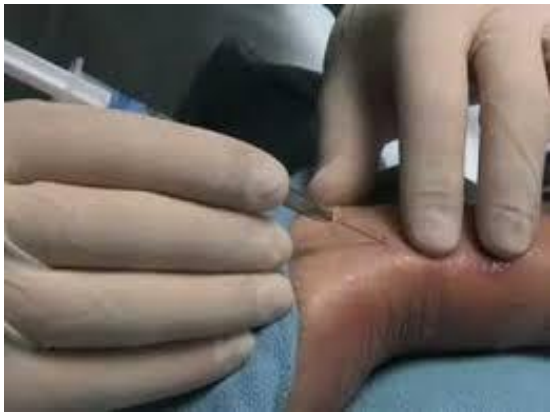
What we will talk about today

- The buffer systems functioning in blood plasma
- Respiratory regulation of acid-base balance
- Renal regulation of acid-base balance
- High and normal anion gap metabolic acidosis
- Metabolic alkalosis
- Respiratory acidosis
- Respiratory alkalosis



The Arterial Blood Gas (ABG)

Acid-base balance

pH/ $PaCO_2$/ PaO_2/ HCO_3^-/ O_2 Sat

$$pH = 6.1 + \log \left[\frac{HCO_3^-}{0.03 \times pCO_2} \right]$$

The Arterial Blood Gas (ABG)

Acid-base balance

pH/ $PaCO_2$/ PaO_2/ HCO_3^-/ O_2 Sat

Ventilation

The Arterial Blood Gas (ABG)

Acid-base balance

pH/ $PaCO_2$/ PaO_2/ HCO_3^-/ O_2 Sat

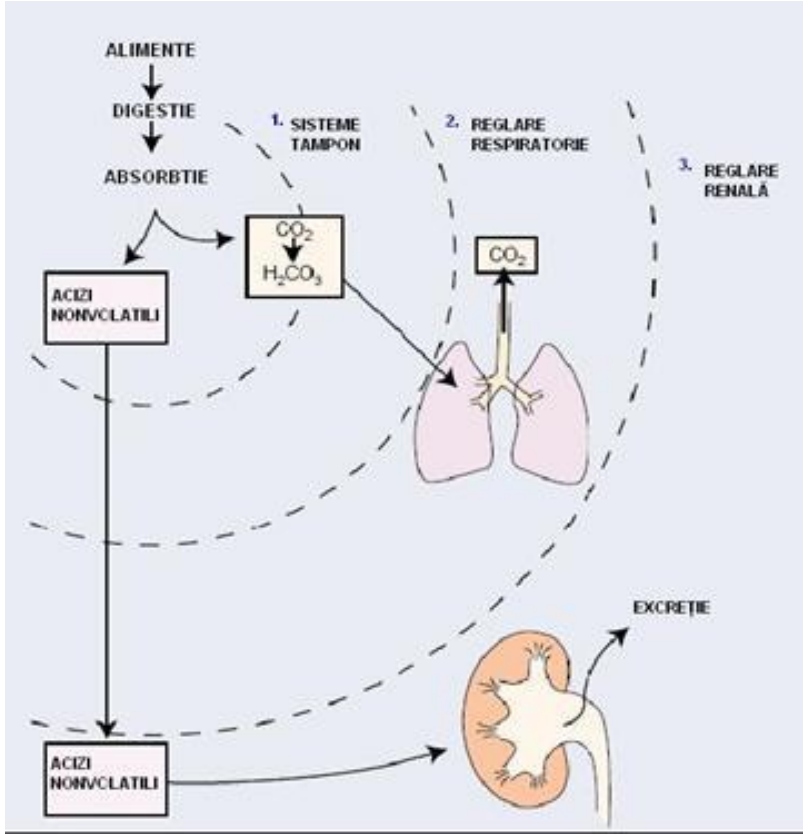
Ventilation Oxygenation

Maintenance of normal pH: buffers

- The most important buffer in blood is *bicarbonate/carbon dioxide* *(HCO_3^-/CO_2)*;

Other buffer systems are:

- *disodium phosphate/monosodium phosphate ($Na_2HPO_4^{2-}$/ $NaH_2PO_4^-$);*
- *plasma proteins;*
- erythrocytes contain the important *hemoglobin (Hb) system*, reduced Hb (HHb^-), and oxyhemoglobin (HbO_2^{2-});



P.S.: Bones also participate in buffering.

Bicarbonate-Carbonic Acid Buffer

- most important buffer in blood
- the HCO3−/CO2 system provides the first line of defense in protecting pH

- its role as a buffer can be described by incorporating this system into the Henderson–Hasselbalch equation

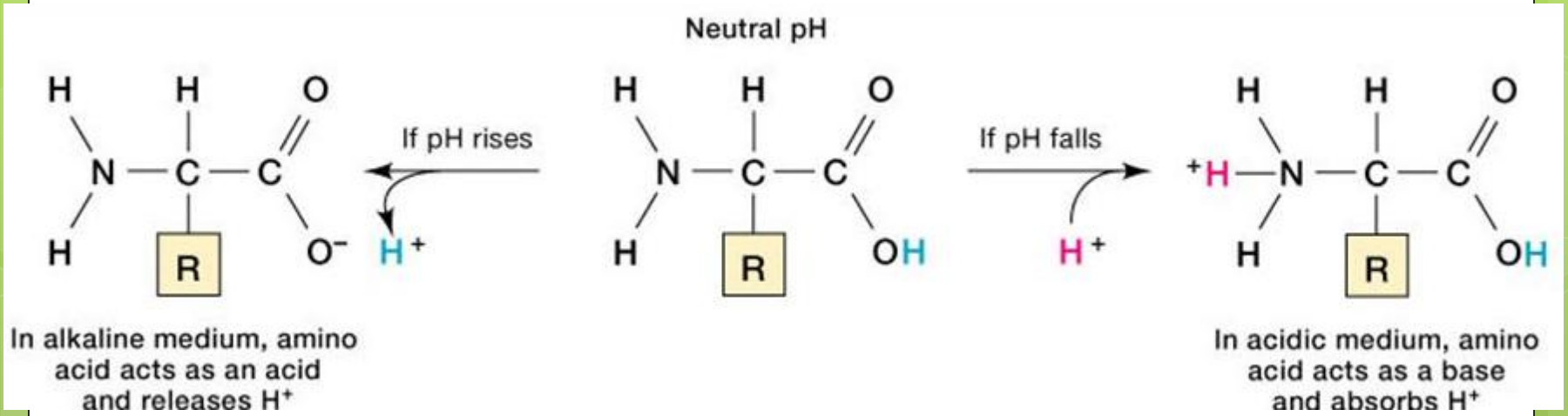
Phosphate Buffer

- effective in regulating intracellular pH more efficiently than extracellular pH (due to their higher concentrations inside the cell)
- the pKa of this system is 6.8, which is close to the intracellular pH
- alkalin component K2HPO4
- acid component KH2PO4

$$HCl + Na_2HPO_4 \rightarrow NaCl + NaH_2PO_4$$

$$NaOH + NaH_2PO_4 \rightarrow Na_2HPO_4 + H_2O$$

Protein Buffers in Blood Plasma



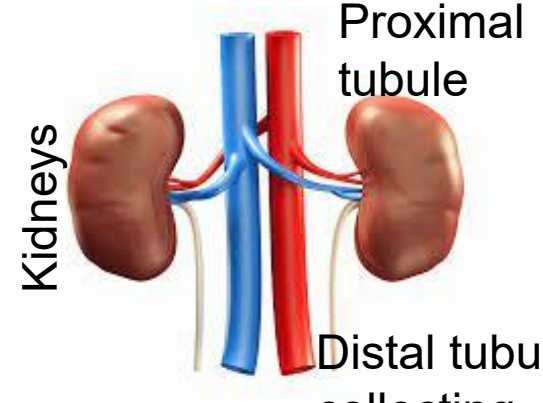
Hemoglobin as a Buffer

- principal protein inside of red blood cells;
- during the conversion of CO_2 into bicarbonate, H^+ is liberated in the reaction are buffered by Hb, which is reduced by the dissociation of oxygen;
- the process is reversed in the pulmonary capillaries to re-form CO_2, which then can diffuse into the air sacs to be exhaled into the atmosphere.

$$KOH + HHb \rightarrow KHb + H_2O$$

$$HCl + KHb \rightarrow HHb + KCl$$

Regulation of acid-base balance

Site	Mechanism
	Expiration of CO_2
	Reabsorption of HCO_3^-
	Excretion of H^+ as titrable acid (e.g. $H^+ + HPO_4^{2-} = H_2PO_4^-$) Excretion of $NH4^+$

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The lungs role and acid-base regulation

Peripheric

↓

↓arterial pH or↑$PaCO_2$

↓

Carotid body chemoreceptors

Central

↓

↓ CSF pH

↓

Medullary chemoreceptors

Medullary respiratory center

↓

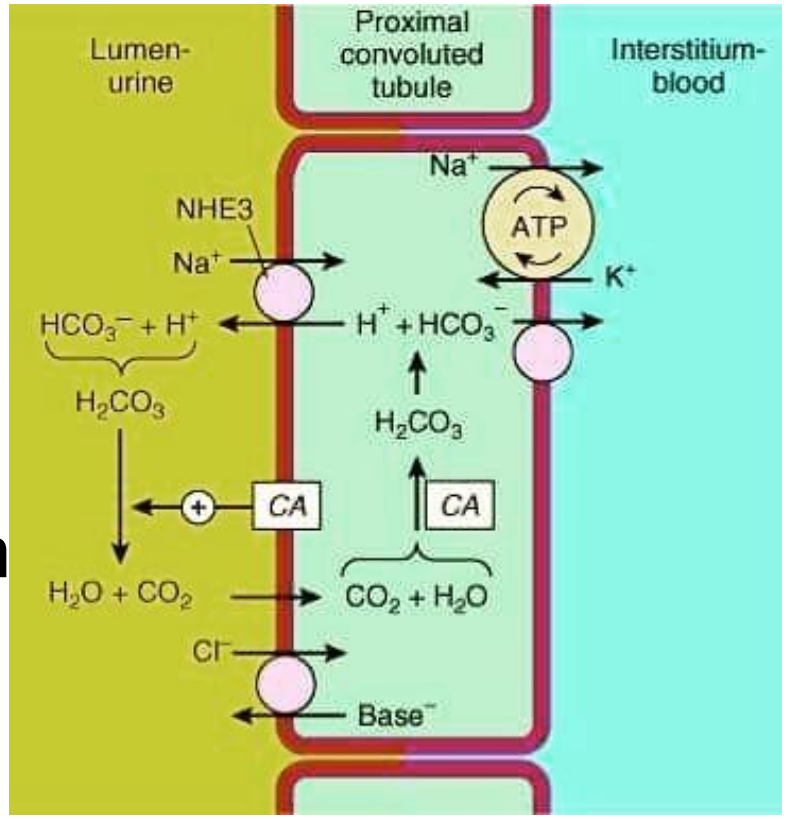
↑ Minute ventilation

expiration of CO_2

↑ pH ↓ $PaCO_2$

The kidney's role and acid-base regulation

- Reabsorption of filtered HCO_3^-
- Generation of new HCO_3^- by titratable acid excretion
- Formation of HCO_3^- from generation of NH_4^+



ABG terminology

- Acidemia: an increase in blood [H^+]
- Alkalemia: a decrease in blood [H^+]
- Acidosis: a pathophysiologic process that tends to acidify body fluids
- Alkalosis: a pathophysiologic process that tends to alkalinize body fluids
- Arterial blood gas (ABG): includes pH, pCO2, and calculated serum [HCO_3^-]
- Normocapnia: normal arterial pCO_2 (40 mmHg)
- Hypocapnia: a decrease in arterial pCO_2
- Hypercapnia: an increase in arterial pCO_2
- Normobicarbonatemia: normal serum [HCO_3^-] (24 mEq/L)
- Hypobicarbonatemia: a decrease in serum [HCO_3^-]
- Hyperbicarbonatemia: an increase in serum [HCO_3^-]

Acid–base terminology

- Primary change: an abnormality in either the serum [HCO_3^-] or arterial pCO_2 resulting from a primary change in body function/metabolism or additions to or losses from body fluids
- Secondary change: a compensatory (secondary) response that acts to minimize changes in pH produced by the primary disorder (compensation)
- Simple acid–base disorder: presence of one primary disorder with appropriate secondary response
- Mixed acid–base disorder: simultaneous occurrence of two or more primary disorders

Pathogenesis of Acid-Base Disorders

Metabolic acidosis develops because of the following conditions:

1. Loss of HCO_3^- either from the GI tract or kidney
2. Retention of H^+ due to impaired renal function
3. Addition of exogenous or endogenous of strong acids

Pathogenesis of Acid-Base Disorders

Metabolic alkalosis develops due to retention of HCO_3^- and/ or loss of H^+.

Respiratory acidosis results from retention of pCO_2.

Respiratory alkalosis develops from hyperventilation.

Normal compensatory responses to primary acid–base disorders

Acid–base disorder	Compensatory response
Metabolic acidosis	$pCO_2 = HCO_3^- \times 1.5+8 \pm 2$, or for each mEq/L decrease in HCO_3^-, pCO2 decreases by 1.2 mmHg
Metabolic alkalosis	For each mEq/L increase in HCO_3^-, pCO_2 increases by 0.7 mmHg
Respiratory acidosis	
Acute	For each mmHg increase in pCO_2, HCO_3^- increases by 0.1 mEq/L
Chronic	For each mmHg increase in pCO_2, HCO_3^- increases by 0.4 mEq/L
Respiratory alkalosis	
Acute	For each mmHg decrease in pCO_2, HCO_3^- decreases by 0.2 mEq/L
Chronic	For each mmHg decrease in pCO_2, HCO_3^- decreases by 0.4 mEq/L

Primary acid–base disturbances and their secondary response

Acid–base disorder	pH	Primary change	Secondary change	Mechanism of secondary change
Metabolic acidosis	< 7,40	↓ HCO_3^-	↓ pCO_2	Hyperventilation
Metabolic alkalosis	> 7,40	↑ HCO_3^-	↑ pCO_2	Hypoventilation
Respiratory acidosis	< 7,40	↑ pCO_2	↑ HCO_3^-	↑ reabsorbton HCO_3^-
Respiratory alkalosis	> 7,40	↓ pCO_2	↓ HCO_3^-	↓ reabsorbtion HCO_3^-

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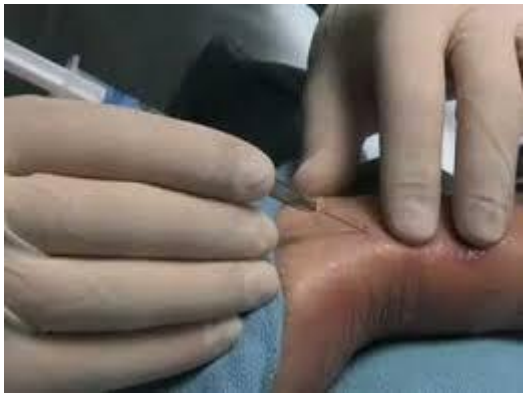
Arterial vs. Venous Blood Sample for ABG

ABG value	Arterial blood	Venus blood
[H+] (nmol/L)	40	44
pH	7,40	7,36
pCO2 (mmHg)	40	48
[HCO3−] (mEq/L)	24	26



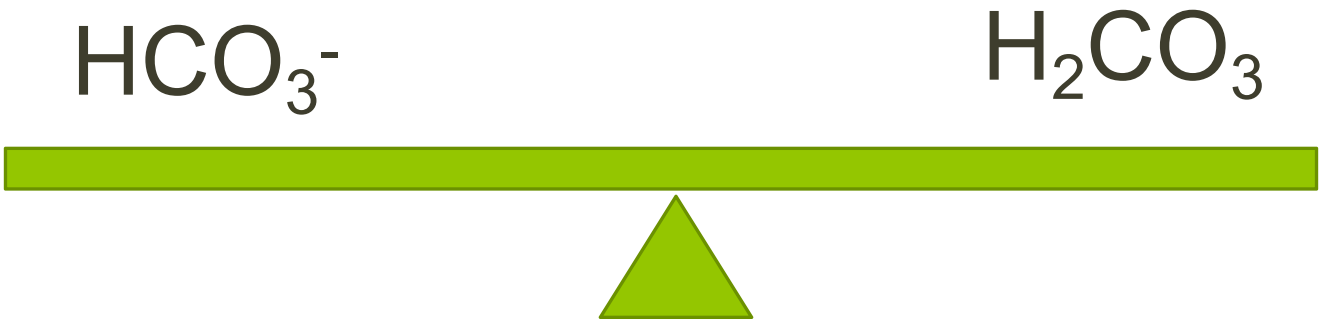
Metabolic acidosis

caused by decrease in HCO_3^- < 24 mmol/l in the blood

lowers pH < 7.35

(BE > -2mEq/l)

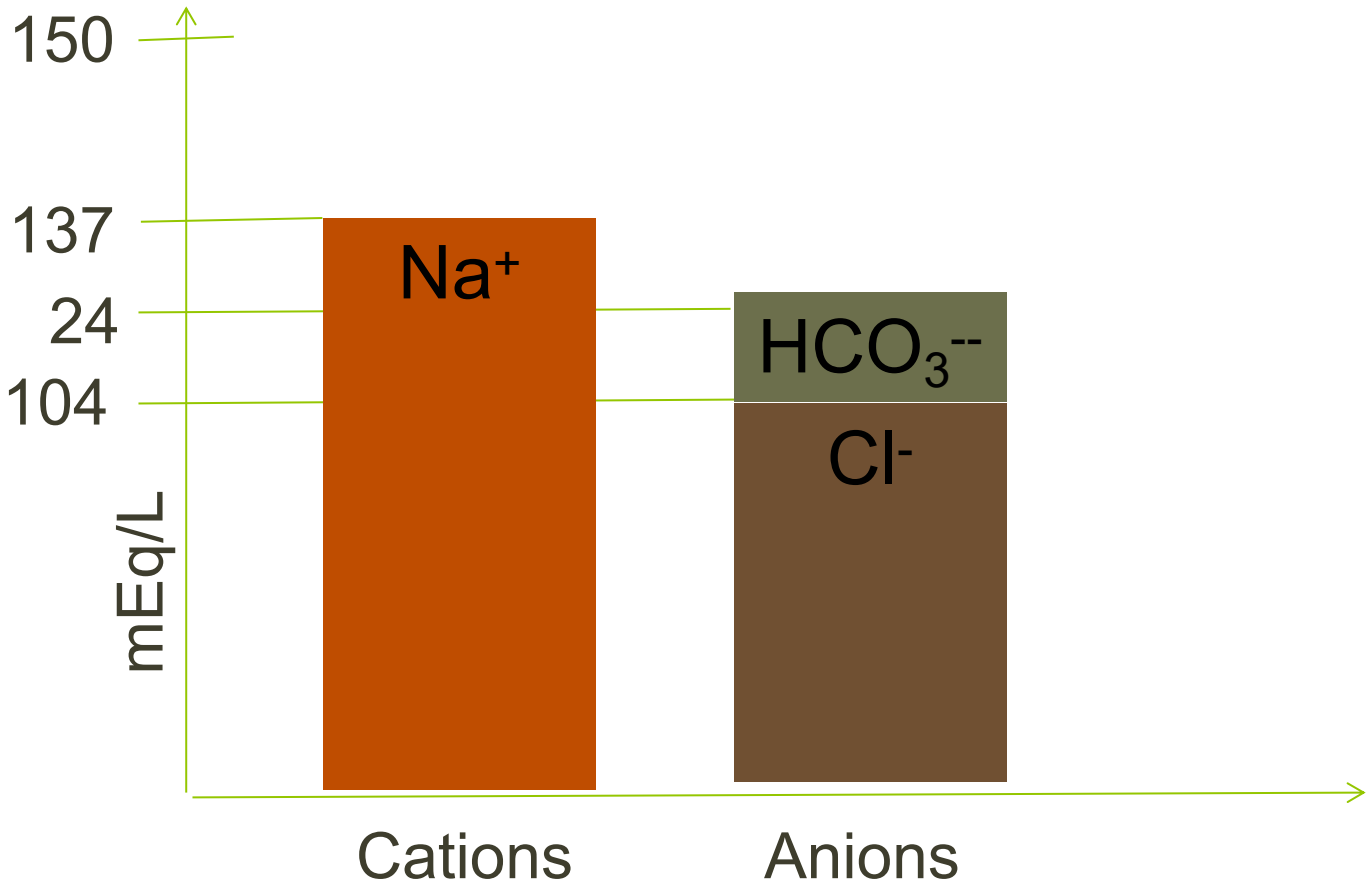
Blood pH



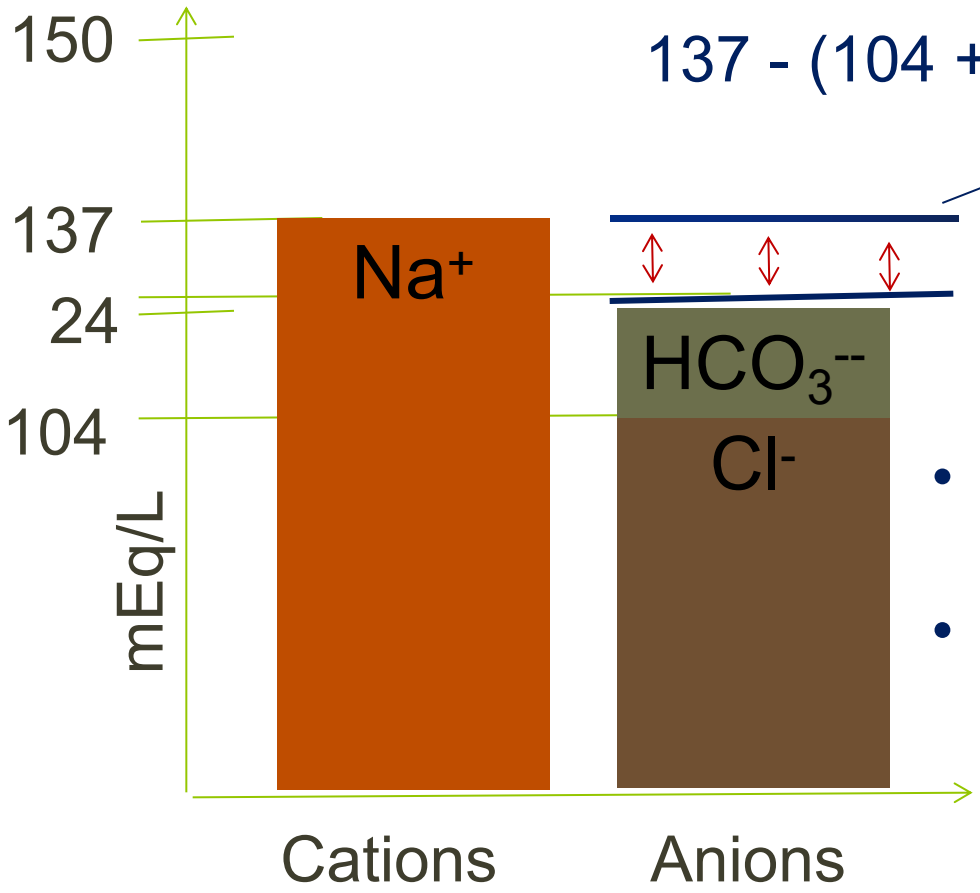
Blood: electrically neutral

pH: 7.35 - 7.45

Blood:electrically neutral



Blood:electrically neutral



137 - (104 +24) = 9 mEq/L

↓

Anion gap*

3 - 11 mEq/L

↓

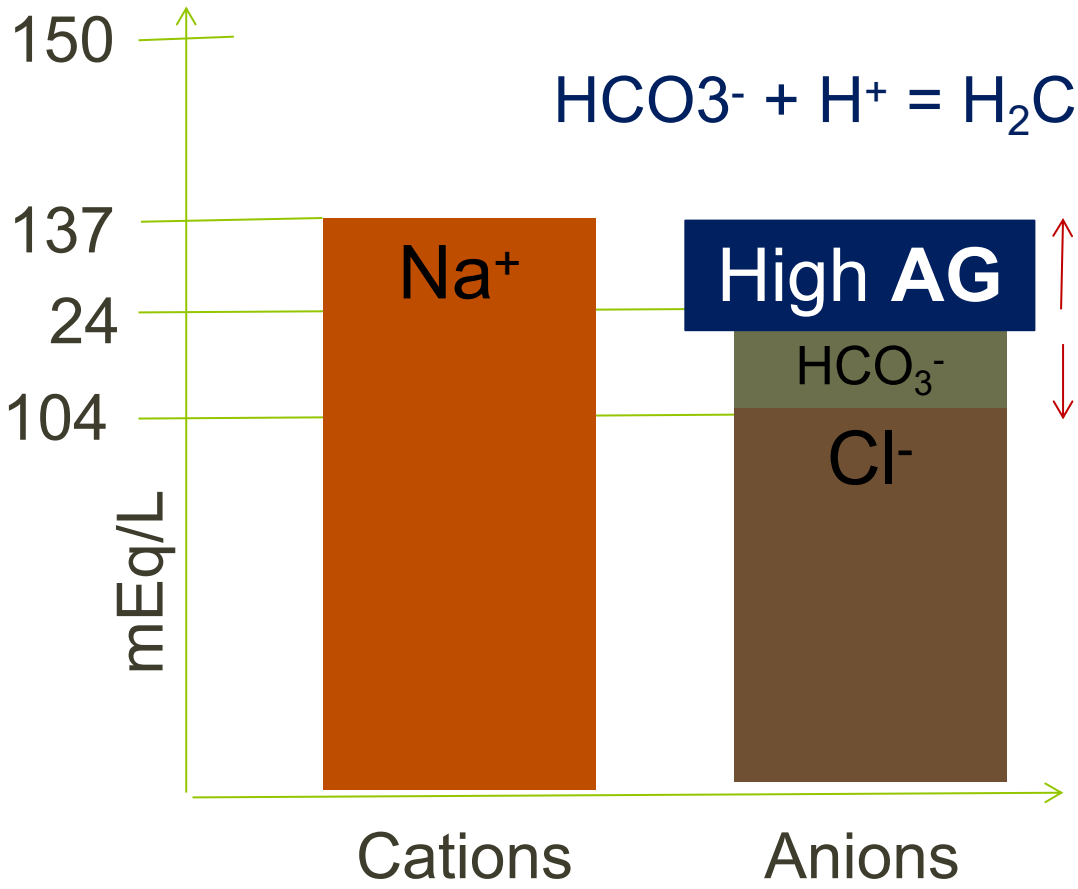
- unmeasured anions (organic acids)
- negatively charged plasma proteins (albumin)

*correct for low albumin, if indicated

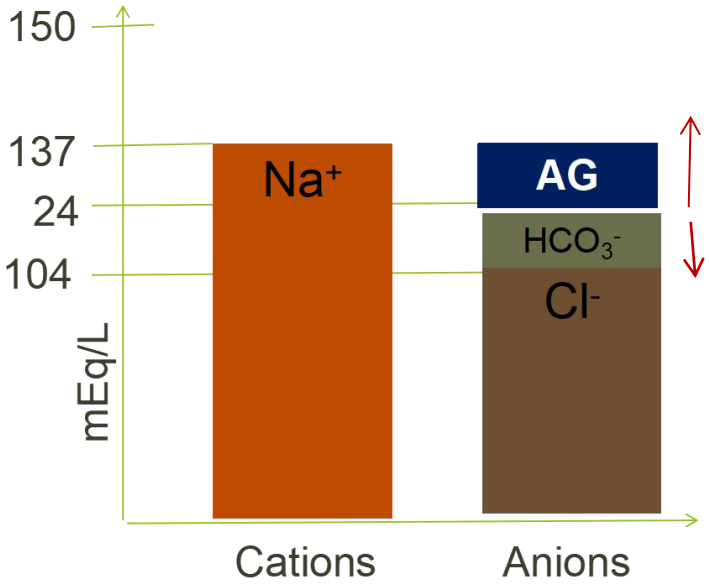
Metabolic acidosis: high or normal AG?

Metabolic acidosis: high AG

$HCO3^- + H^+ = H_2CO_3 = CO_2 + H_2O$



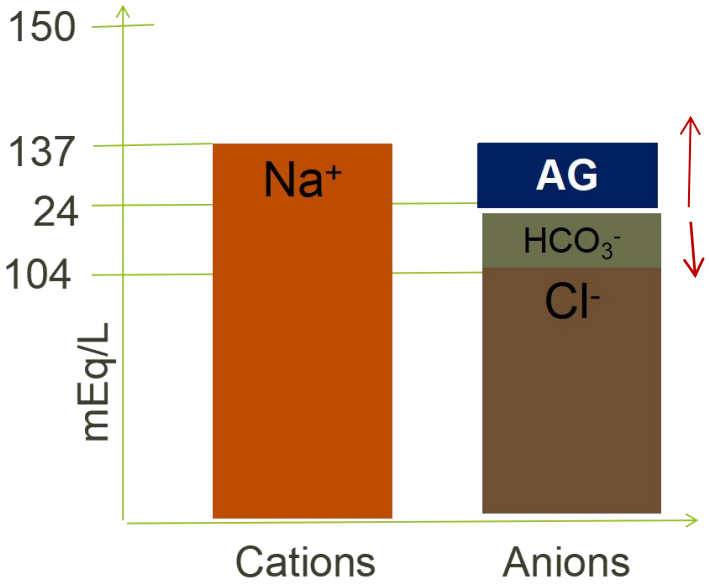
Metabolic acidosis: high AG



Lactic acidosis

Decreased oxygen delivery to the tissues = anaerobic metabolism = buildup of lactic acid

Metabolic acidosis: high AG

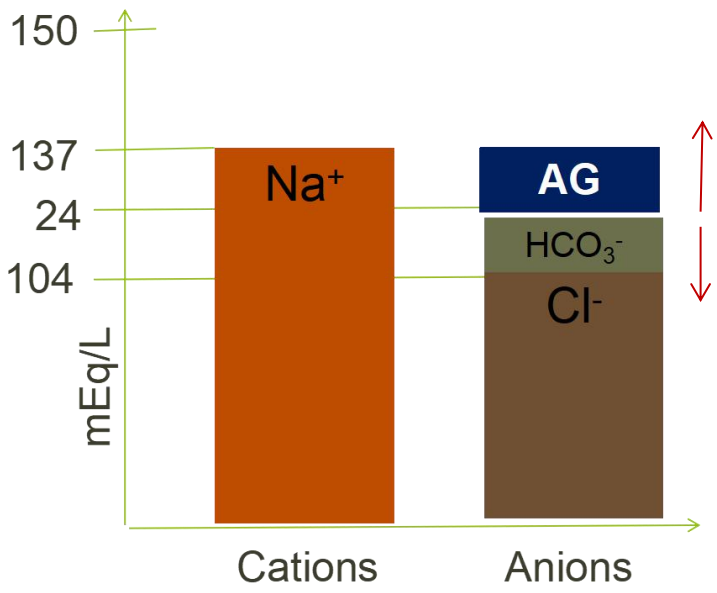


Uncontrolled diabetes mellitus

Lack of insulin = cells use fats as primary energy fuel instead of glucose

Fats = converted to ketoacids (acetoacetic acid and β-hydroxybutyric acid)

Metabolic acidosis: high AG

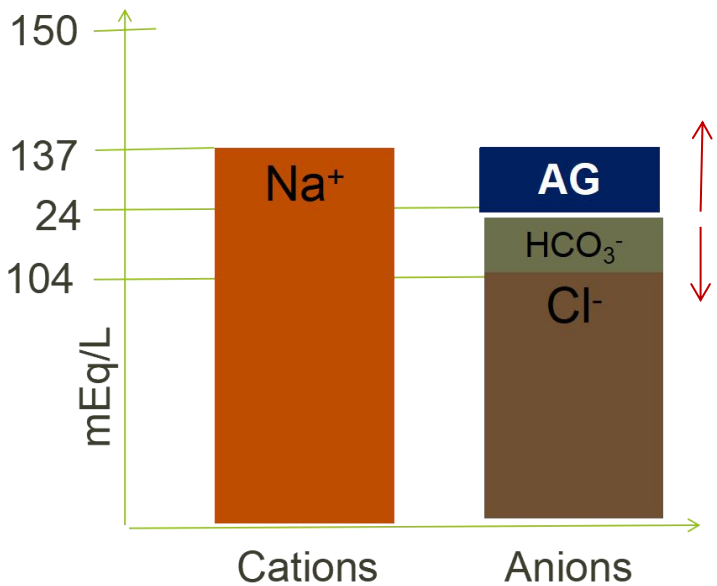


chronic renal failure

organic acids

(uric acid or sulfur- containing amino acids) accumulate

Metabolic acidosis: high AG

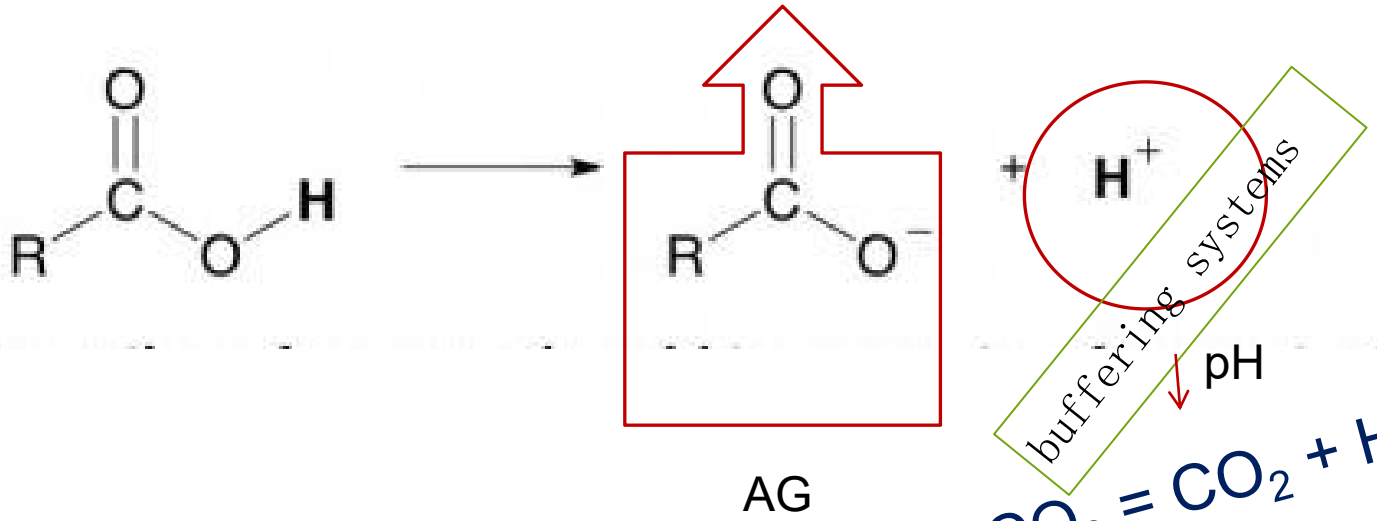


organic acids

accidentally ingested

- oxalic acid (build up after an accidental ingestion of ethylene glycol, which is a common antifreeze);
- formic acid (a metabolite of methanol, a highly toxic alcohol);
- hippuric acid (comes from toluene, which is found in paint and glue).

Metabolic acidosis: high AG



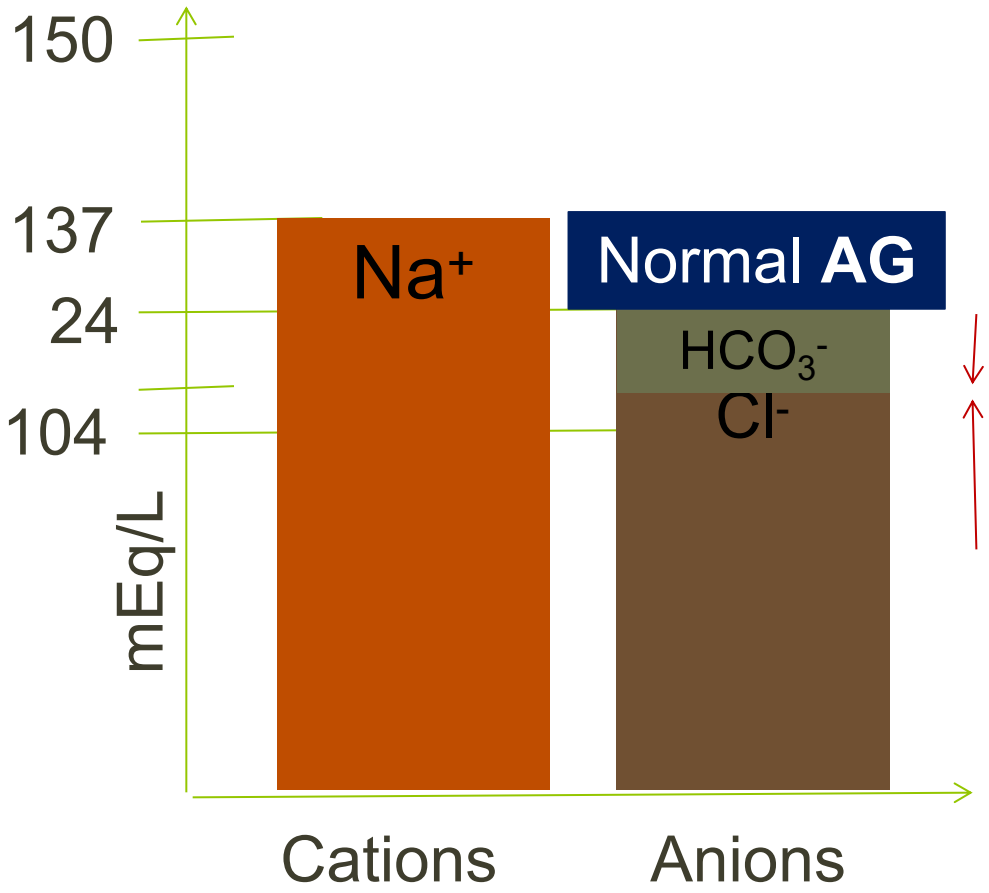
$HCO3^- + H^+ = H_2CO_3 = CO_2 + H_2O$

Metabolic acidosis: high AG

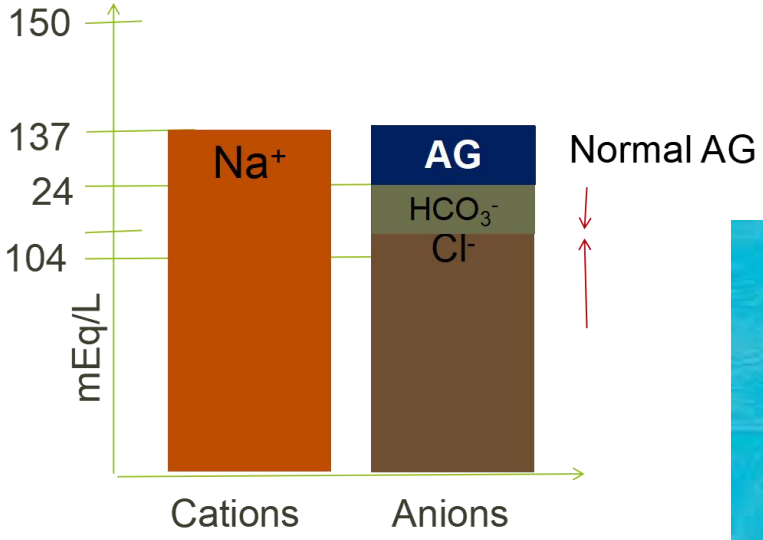
G glycols (ethylene and propylene glycols)
O oxoproline (pyroglutamic acid)
L L-lactate
D D-lactate

M methanol
A aspirin
R renal failure
K ketoacidosis

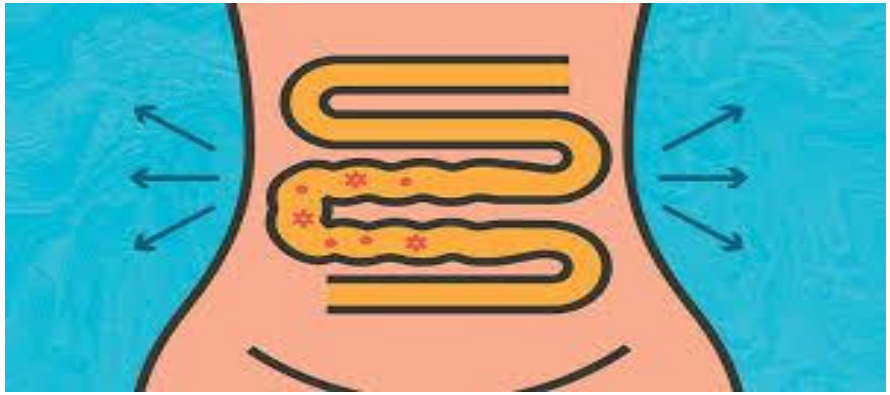
Blood: electrically neutral



Metabolic acidosis: normal AG

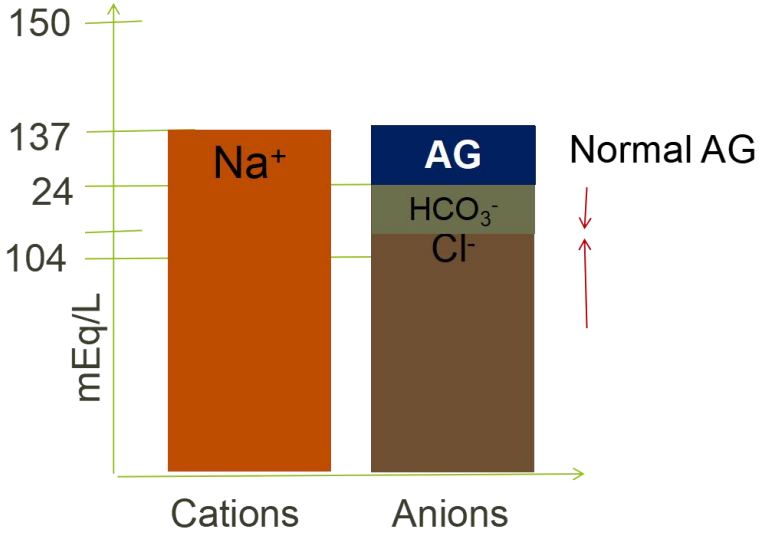


severe diarrhea

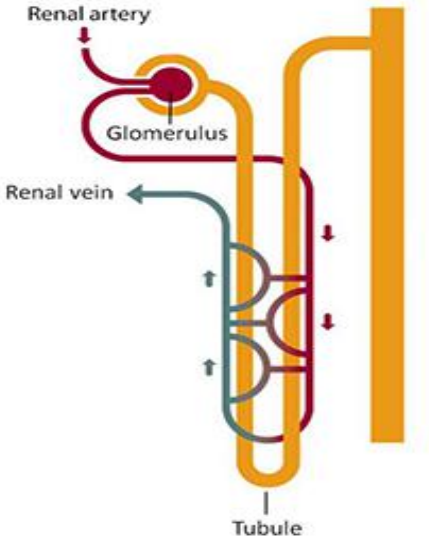


bicarbonate - rich intestinal and pancreatic secretions rush through the gastrointestinal tract before they can be reabsorbed

Metabolic acidosis: normal AG



type 2 renal tubular acidosis



- the most common type of renal tubular acidosis
- develops because the proximal convoluted tubule is unable to reabsorb bicarbonate HCO_3^-

Metabolic acidosis: normal AG

the proximal convoluted tubule is unable to reabsorb bicarbonate HCO_3^-

↓

type 2 renal tubular acidosis

inability to excrete protons H+ in the urine

↓

Other types of renal tubular acidosis

Hyperchloremic metabolic acidosis

UAG = (UNa+UK−UCl)

positive UAG = renal ; negative UAG = extrarenal

Metabolic acidosis: etiology

Increased H+ production

Lactic acidosis:
Type A: hypoperfusion (shoks);
Type B: severe liver diseases, metformin, malignancies, alchoolism, mitocondrial dysfunction,
antiretroviral drugs;

Ketoacidosis:
Diabetis

Toxins: methanol, ethylene glycol, salycilate posoning, chronic acetaminophen ingestion;
D-lacticcidosis;
Toluene ingestion.

Loss of bicarbonate

Diarrhea/ GI losses
RTA type 2 (proximal);

Post-treatment of ketoacidosis

Carbonic anhydrase inhibitors

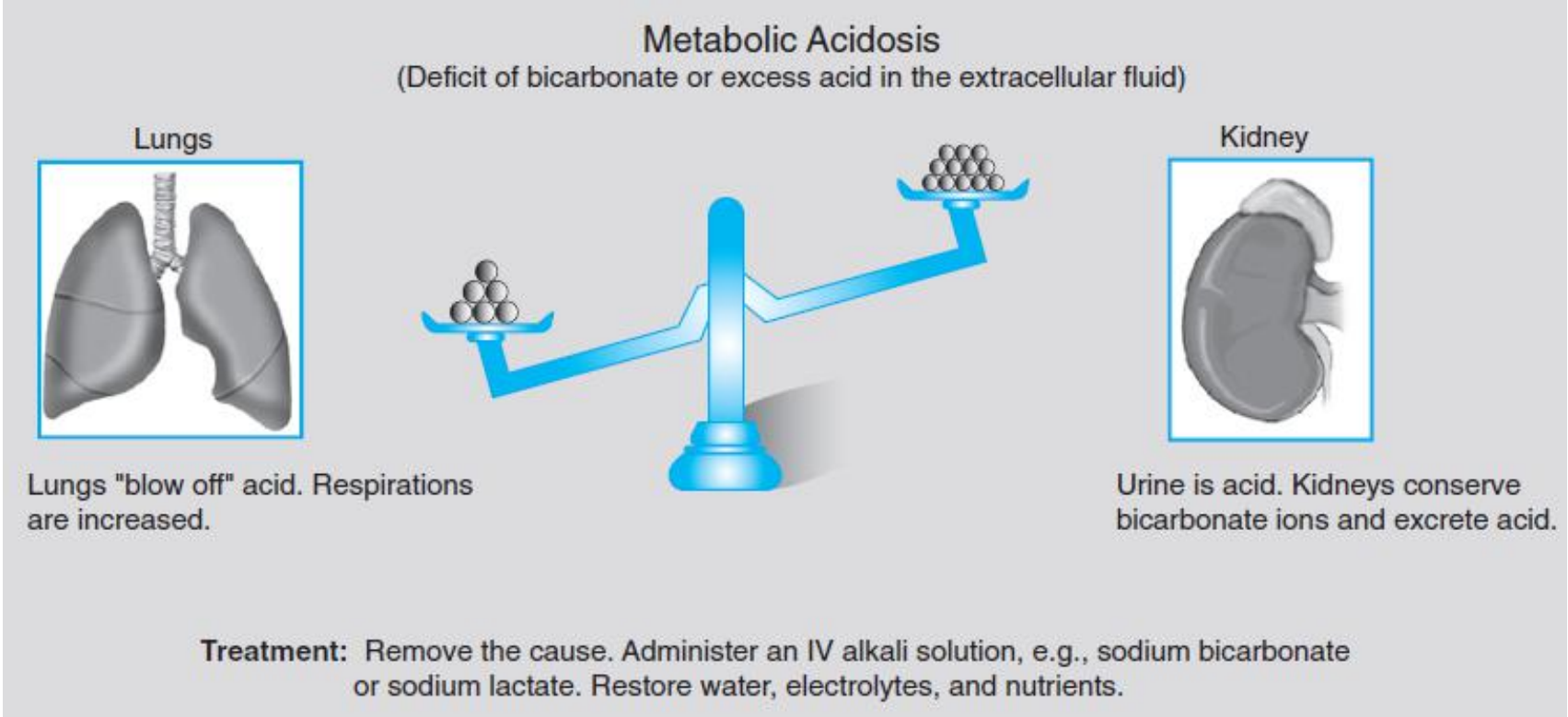
Decreased acid excretion

Chronic kidney disease;

RTA type 1 (distal, inability to secret H^+);

RTA type 4 (hyperkaliemic, decreased function of aldosteron).

Metabolic acidosis: physiopathology



Clinical manifestations of metabolic acidosis

Cardiovascular

- Increased heart rate and contractility at pH <7.2;
- Decreased contractility at pH <7.1; decreased cardiac responsiveness to catecholamines, renal and hepatic blood flow, fibrillation threshold;
- Increased peripheral vasodilation and hypotension.

Neurologic

- Increased sympathetic stimulation, increased cerebral blood flow
- Altered mental status, decreased cerebral metabolism

Clinical manifestations of metabolic acidosis

Respiratory

- Increased minute ventilation, dyspnea, decreased diaphragmatic contractility

Other

- Inhibition of anaerobic metabolism
- Increased protein catabolism
- Increased metabolic rate
- Impaired phagocytosis
- Decreased ATP production
- Impaired skeletal growth
- Bone pain

Metabolic acidosis: treatment

- Removing/ treating the underlying cause improves lactic acidosis
- Circulatory support
- Broad-spectrum antibiotic administration (extremely important for SIRS/sepsis syndrome with circulatory support)
- Alkali treatment of metabolic acidosis is important; however, there are several disadvantages with this treatment

Intravenous alkali treatment for metabolic (lactic) acidosis

Alkali	Advantages	Disadvantages
$NaHCO_3$	Rapid effect, inexpensive, easy to administer	Hypertonicity, hypernatremia, ↑ CO_2 production, ↑ intracellular acidosis, volume overload, no survival benefit
THAM (Tris-Hydroxymethyl Aminomethane)	No increase in CO2, penetrates cells to buffer intracellular pH, useful in the treatment of mixed metabolic and respiratory acidosis	Respiratory depression, hypoglycemia, hyperkalemia, liver necrosis in children. Avoid in renal failure
Carbicarb	A mixture of 0.33 M Na2CO3 and 0.33 M NaHCO3, less CO2 production	The same as NaHCO3, no clinical benefit and nonavailability

NaHCO3 requirements

- Decide how much serum [HCO_3^-] you need to raise, i.e., ΔHCO_3^-
- Estimate HCO_3^- space as 50% of body weight in metabolic acidosis*

*Some authors calculate as 40% (note that HCO_3^- space or deficit increases with an increase in [H^+] or a decrease in pH)

- Calculate the amount of $NaHCO_3$ that is needed to raise serum [HCO_3^-] to the desired level
- Calculations should be based on an ongoing pathologic process that is causing metabolic acidosis
- Administer slowly as an isotonic solution at a rate of ~0.1 mEq/kg/min
- Consider administration of calcium gluconate separately to prevent fall in ionized Ca_2^+ after alkali administration to improve cardiac function

Metabolic alkalosis

caused by increase in HCO_3^- > 26 mmol/l in the blood

pH > 7.35

(BE > +2 mEq/l)

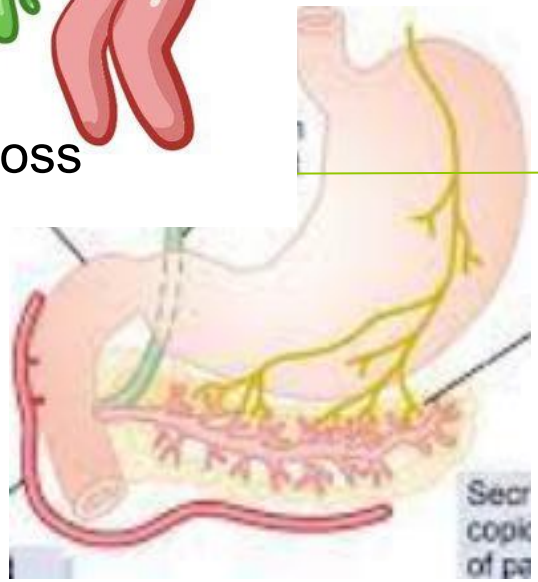
Etiologies of metabolic alkalosis

Primary issue	GI pathology	Renal problem
Loss of H+		
Gain of HCO3-		

Etiologies of metabolic alkalosis

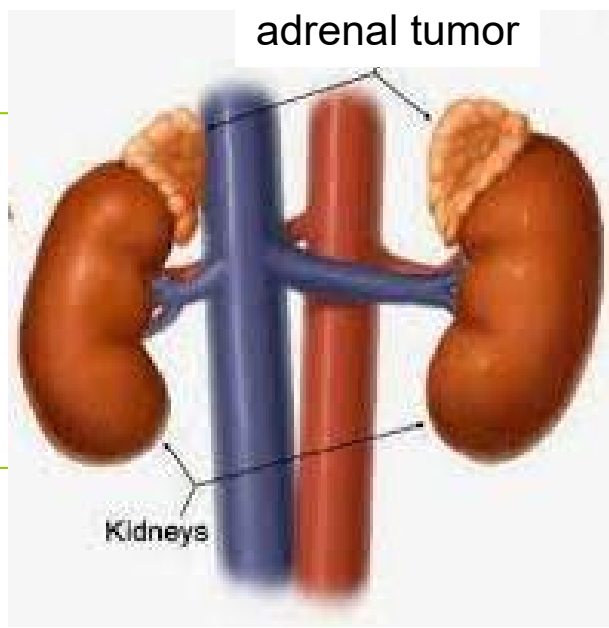
Primary issue	GI pathology
Loss of H+	**Vomitting** **NG sucction** Congenital chloride diarhhea
Gain of HCO3-	

H^+ loss



Etiologies of metabolic alkalosis

Primary issue		Renal problem
Loss of H+		**Mineralocorticoid excess** Loop/ Thiazide diutertics Contraction alkalosis Bartter/ Gitelman syndromes

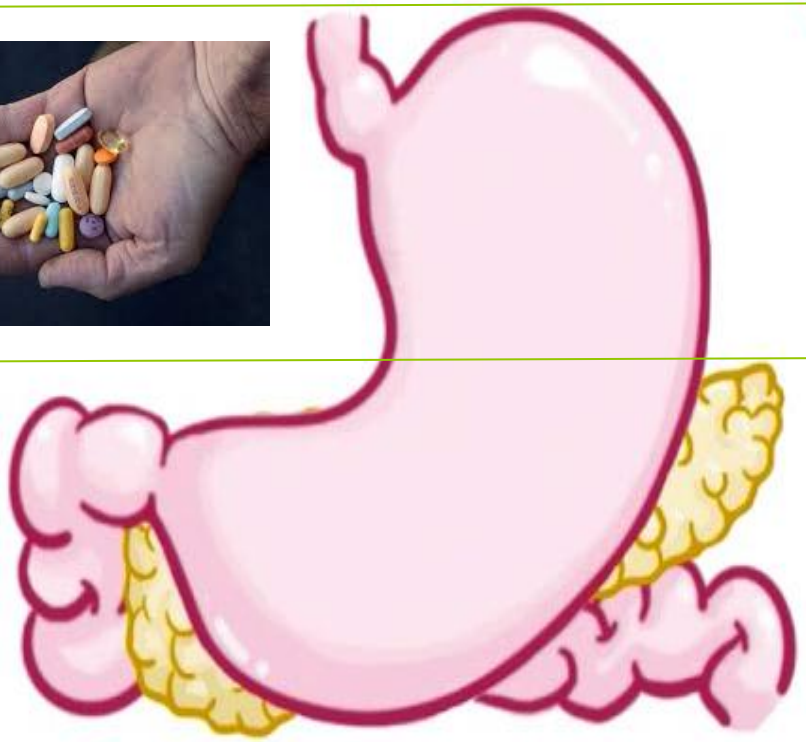


↑ aldosterone

α- intercalated cells of the distal convoluted tubule and collecting duct dump out H+ and reabsorb HCO3−

Etiologies of metabolic alkalosis

Primary issue	**GI pathology**
Loss of H+	
Gain of HCO3-	Milk-alkali syndrome NaHCO3 ingestion

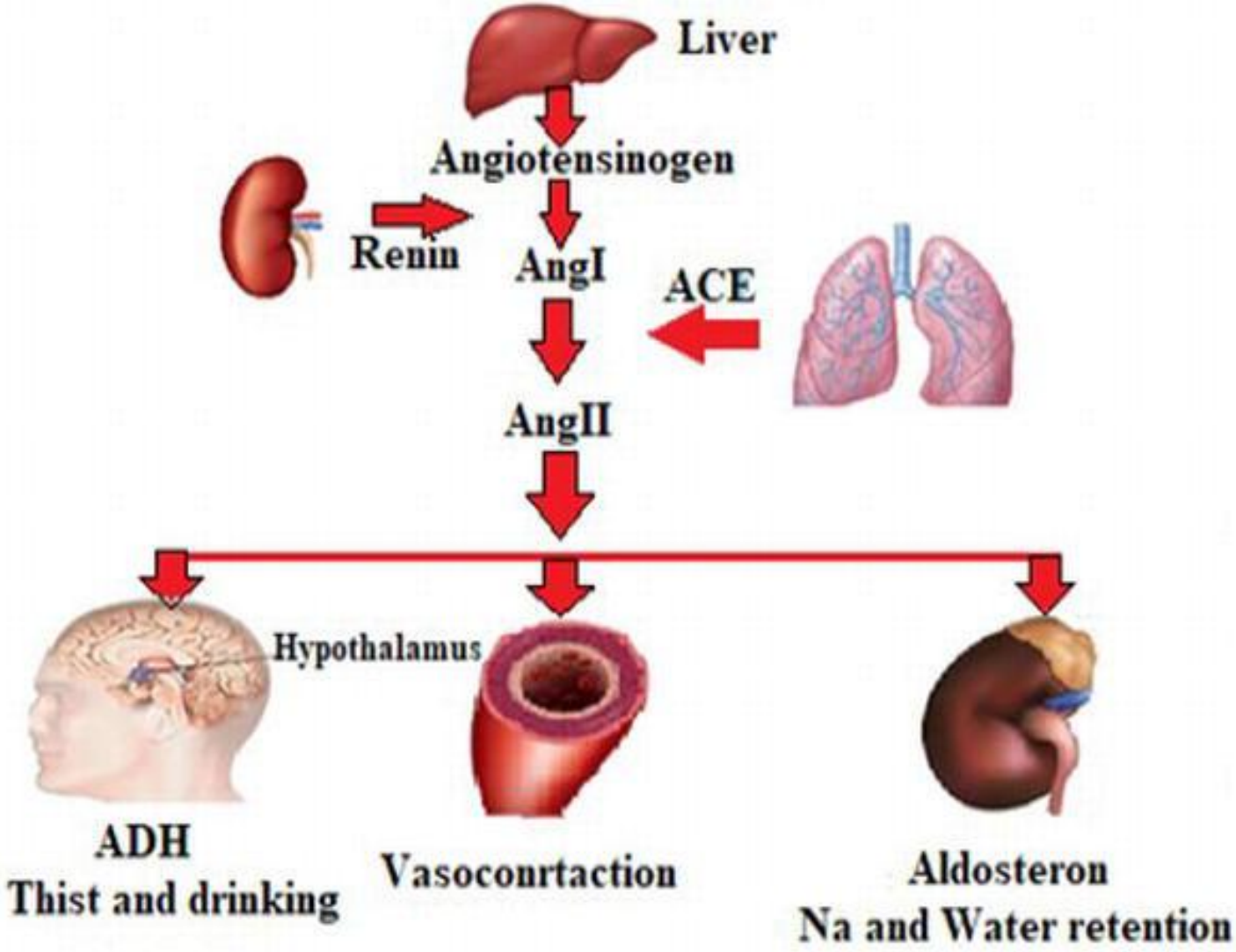


Etiologies of metabolic alkalosis

Primary issue	GI pathology	**Renal problem**
Loss of H+	Vomitting NG sucction Congenital chloride diarhhea	Mineralocorticoid excess **Loop/ Thiazide diutertics** **Contraction alkalosis** Bartter/ Gitelman syndromes
Gain of HCO3-		**Contraction alkalosis**

increased reabsorption of HCO_3^- from the kidneys

The kidney's role and acid-base regulation



Leading causes of metabolic alkalosis

Primary issue	GI pathology	Renal problem
Loss of H+	Vomitting NG sucction Congenital chloride diarhhea	Mineralocorticoid excess Loop/ Thiazide diutertics Contraction alkalosis Bartter/ Gitelman syndromes
Gain of HCO3-	Milk-alkali syndrome NaHCO3 ingestion	Contraction alkalosis

Clinical manifestations of metabolic alkalosis

Central nervous system. Reduction in blood flow, confusion, obtundation, reduced seizure threshold.

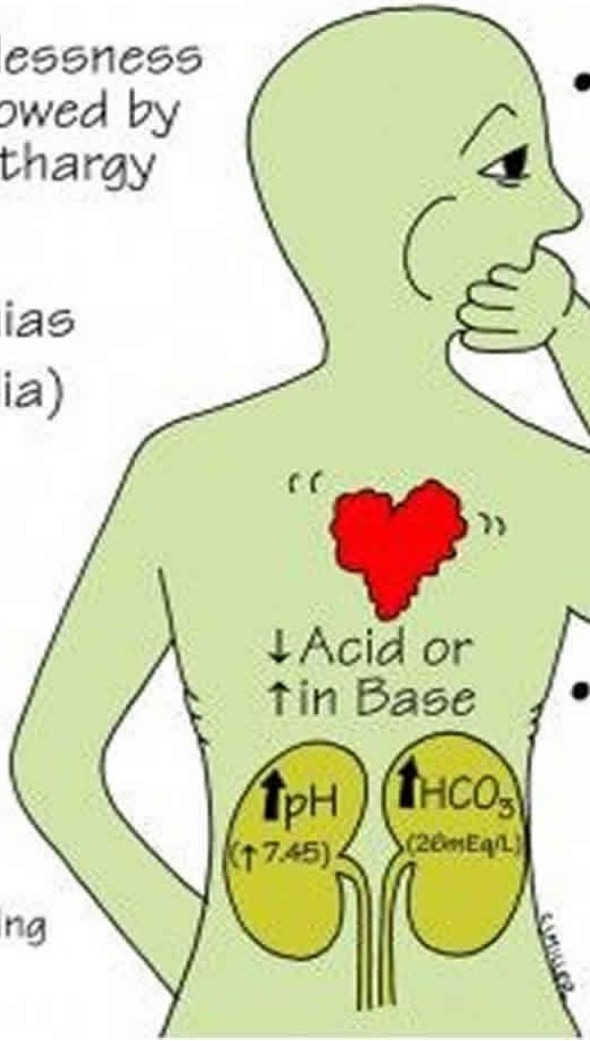
Neuromuscular. Increased excitability, tetany.

Cardiovascular. Reduced cardiac output and coronary blood flow, arteriolar vasoconstriction, increased heart rate, predisposition to refractory ventricular and supraventricular arrhythmias.

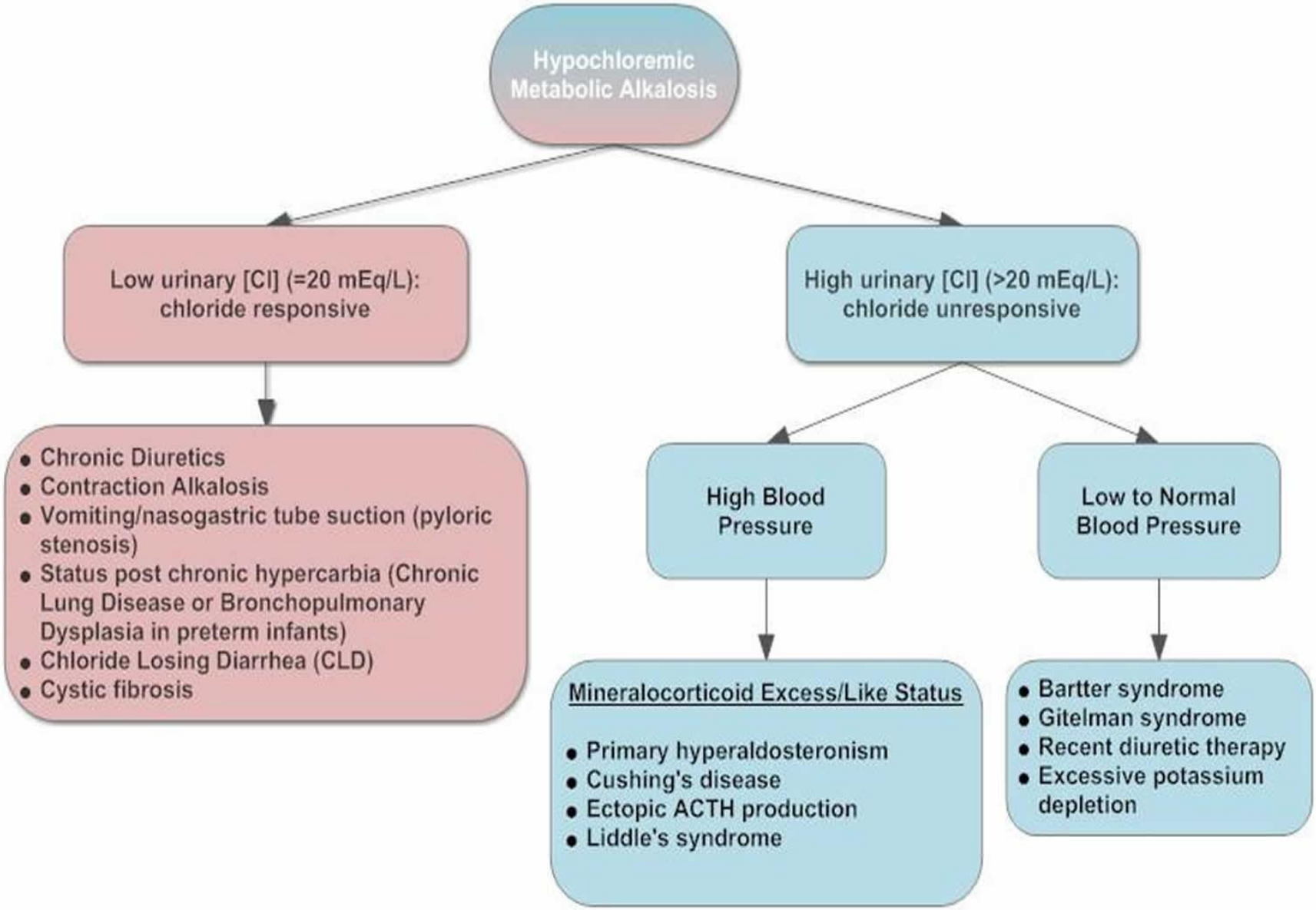
Pulmonary. Hypoventilation, hypercapnia, hypoxia.

Metabolic. Hypokalemia, decreased ionized Ca^{2+}, hypophosphatemia, hypomagnesemia, stimulation of glycolysis and production of lactate.

Metabolic alkalosis



- Restlessness Followed by Lethargy
- Confusion (↓LOC, Dizzy, Irritable)
- Dysrhythmias (Tachycardia)
- Nausea, Vomiting, Diarrhea
- Compensatory Hypoventilation
- Tremors, Muscle Cramps, Tingling of Fingers & Toes
- Causes:
  - Severe Vomiting
  - Excessive GI Suctioning
  - Diuretics
  - Excessive NaHCO3
- Hypokalemia


Hypochloremic Metabolic Alkalosis
Low urinary [Cl] (=20 mEq/L): chloride responsive
High urinary [Cl] (>20 mEq/L): chloride unresponsive
• Chronic Diuretics
• Contraction Alkalosis
• Vomiting/nasogastric tube suction (pyloric stenosis)
• Status post chronic hypercarbia (Chronic Lung Disease or Bronchopulmonary Dysplasia in preterm infants)
• Chloride Losing Diarrhea (CLD)
• Cystic fibrosis
High Blood Pressure
Low to Normal Blood Pressure
Mineralocorticoid Excess/Like Status
• Primary hyperaldosteronism
• Cushing's disease
• Ectopic ACTH production
• Liddle's syndrome
• Bartter syndrome
• Gitelman syndrome
• Recent diuretic therapy
• Excessive potassium depletion

Metabolic alkalosis: tratment

1. Address the specific cause, and correct it.
2. Administration of normal saline is the appropriate therapy in volume depletion patients (replaces volume and Cl^- loss).
3. Administer KCl orally or IV for severe hypokalemia.
4. Avoid Ringer's lactate(!), as lactate is converted into HCO_3^- and worsens alkalosis.
5. Intravenous HCl (0.1 or 0.2 M) indicated when NaCl and KCl are contraindicated, blood pH >7.5, or hypervolemic.
6. HCl should be infused via a large vein to avoid extravasation at <0.2 mEq/kg/h.
7. An alternative to HCl is NH_4Cl or arginine HCl.

(!) NH_4Cl is contraindicated in hepatic and renal failure.

Metabolic alkalosis: treatment

8. Acetazolamide (250 mg BID or TID) used in patients with volume overload and normal renal function.

9. When a renal failure patient fails to respond to Cl^- repletion, renal replacement therapy (hemodialysis, peritoneal dialysis, or hemodiafiltration) is indicated.

10.In patients with protracted vomiting or prolonged nasogastric suction, H_2 blockers (cimetidine, ranitidine) or H/K-ATPase inhibitor (omeprazole) can blunt acid production in the stomach and reduce HCl loss.

11. Omeprazole is effective in the treatment of alkalosis in patients with gastrocystoplasty.

12. Octreotide is used to control hypergastrinemia in Zollinger–Ellison syndrome.

Respiratory vs. metabolic disorders

Respiratory disorders	Pathologic processes which disrupt acid-base balance due to their effect on the lungs (i.e. alveolar ventilation).	
	Respiratory acidosis $PaCO_2$ is too high (hypoventilating)	Respiratory alkalosis $PaCO_2$ is too low (hyperventilating)
Metabolic disorders	Pathologic processes which disrupt acid-base balance due to their effects on anything other than the lungs (i.e. kidneys, GI tract or cellular respiration).	
	Metabolic acidosis HCO_3^- is too low	Metabolic alkalosis HCO_3^- is too high

Respiratory acidosis
(primary hypercapnia)

- $PaCO_2 > 45$ mmHg (hypercapnia)
- pH < 7.35 (acidosis)

- increase in serum $[HCO_3^-]$

Respiratory acidosis: etiology

Hypercapnic: PUMP FAILURE

Acute respiratory failure

Hypoxemic: LUNG FAILURE

Failure to ventilate

CNS

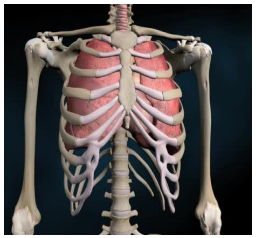
Cerebral trauma

Overdose of respiratory depressant

Cerebrovascular accident

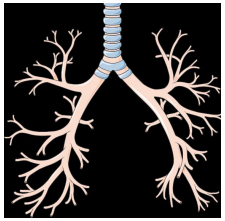
Subarachnoid hemorrhage

Peripheral NS & Chest Bellow components



Poliomyelitis
Guillain-Barre
Multiple sclerosis
Spinal cord injury etc.
Flail chest
Pleural effusion
Pneumothorax etc.

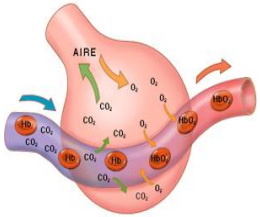
Airways component



Upper airway obstruction (epiglottitis, obstructive sleep apnea)
Asthma
Bronchospa
Foreign body
Postextubational laryngeal edema

Failure to oxygenate

Alveolar component



Pulmonary contusion

Pneumonia

Pulmonary edema

ARDSInsterstitial lung disease

Respiratory acidosis

(primary hypercapnia)

H^+ is immediately (minutes) buffered by nonbicarbonate buffers (Hb, phosphates, plas proteins), so th is not use

the within min, and a steady-state condition persists for 1 h;

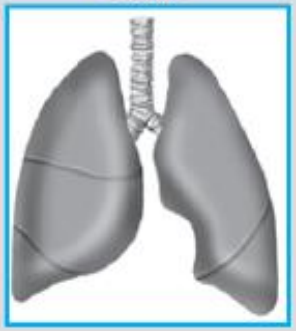
Thus, respiratory acidosis can be classified into acute (<12 h) or chronic (>5 days) types. Both acute and chronic hypercapnias are associated with hypoxemia

pnia continues for > 12 h, the kidneys generate a onal HCO_3^- by excretion of H^+ and the maximum level of HCO_3^- generation is completed within 3-5 days.

Respiratory acidosis

(Excess of carbonic acid in the extracellular fluid)

Lungs



Kidneys Compensate

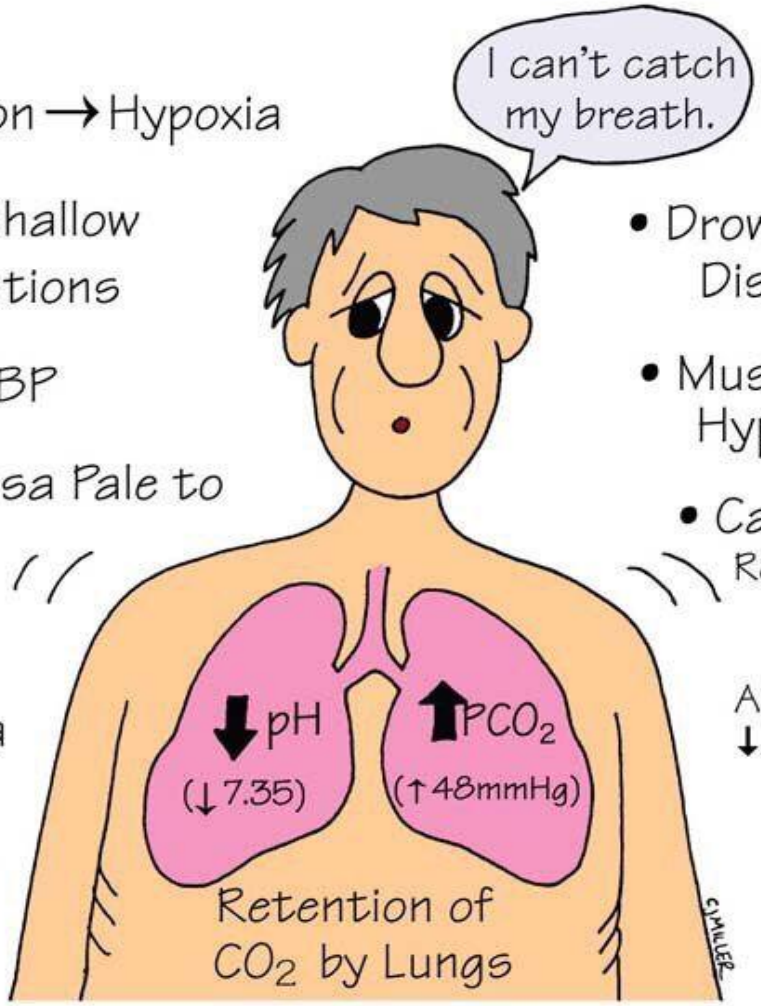
Lungs are affected: insufficient gas exchange and/or ventilation. High $PaCO_2$ causes a reflexive attempt to increase ventilation.

Urine is acid. Kidneys conserve base (bicarbonate) and excrete acid.

Treatment: Remove the cause. Administer an IV alkali solution. Deep breathing exercise or use of a ventilator.

RESPIRATORY ACIDOSIS

- Hypoventilation → Hypoxia
- Rapid, Shallow Respirations
- ↓ BP
- Skin/Mucosa Pale to Cyanotic
- Headache
- Hyperkalemia
- Dysrhythmias (↑ K^+)



- Drowsiness, Dizziness, Disorientation
- Muscle Weakness, Hyperreflexia
- Causes:
  - Respiratory Depression (Anesthesia, Overdose, ↑ICP)
  - Airway Obstruction
  - ↓Alveolar Capillary Diffusion (Pneumonia, COPD, ARDS, PE)

Respiratory acidosis: treatment

- Correction of the underlying cause
- Establishing a secure patent airway
- Oxygenoterapy to improve hypoxemia (more important than lowering $PaCO_2$ and raising pH !)
- Assisted ventilation for severely obtunded/ comatose patients/ patients with pH < 7.10
- In awake and hemodynamically stable, O_2 by nasal cannula/ by high-flow Venturi face mask (the aim: PaO_2 of 60–70 mmHg or SpO_2 > 88%)
- Mechanical ventilation in apneic/ obtunded/ hemodynamically unstable patients with pH <7.10 and $PaCO_2$ >80 mmHg
- Lowering $PaCO_2$ may be sufficient to raise pH, but NaHCO3 is needed in some patients

Respiratory alkalosis
(primary hypocapnia)

- $PaCO_2 < 35mmHg$ (hypocapnia)
- pH > 7.45
- decrease in serum $[HCO_3^-]$

Primary hypocapnia reflects pulmonary hyperventilation.

Secondary hypocapnia should be distinguished from primary hypocapnia, as the former occurs in response to metabolic acidosis.

Respiratory alkalosis

(primary hypocapnia)

Corrected by nonbicarbonate buffering as well as H^+ release from tissues;

lactate is also produced by alkalemia. This buffering from various sources persists for (several hours);

resultant acid–base disturbance is called acute respiratory alkalosis;

H^+ secretion in both proximal tubule and cortical collecting duct is suppressed.

When alkalemia persists, renal compensation starts with a decrease in both H^+ secretion and basolateral exit of HCO_3^- in the proximal tubule; this lowers serum $[HCO_3^-]$ even further, due to which the pH is maintained close to normal;

the full renal compensation takes 2–3 days for completion, and a new steady state is established, which is called chronic respiratory alkalosis.

Causes of respiratory alkalosis

1. Direct stimulation of medullary respiratory center:

- Voluntary or psychogenic hyperventilation
- CNS infection, tumor, or trauma
- Gram-negative sepsis
- Hypermetabolic state (fever, thyrotoxicosis)
- Drugs (salicylates, progesterone, nicotine, xanthine derivatives, catecholamines, antipsychotic drug quetiapine)
- Stroke
- Liver failure
- Pregnancy
- Pain

2. Hypoxemic stimulation of medullary respiratory center:

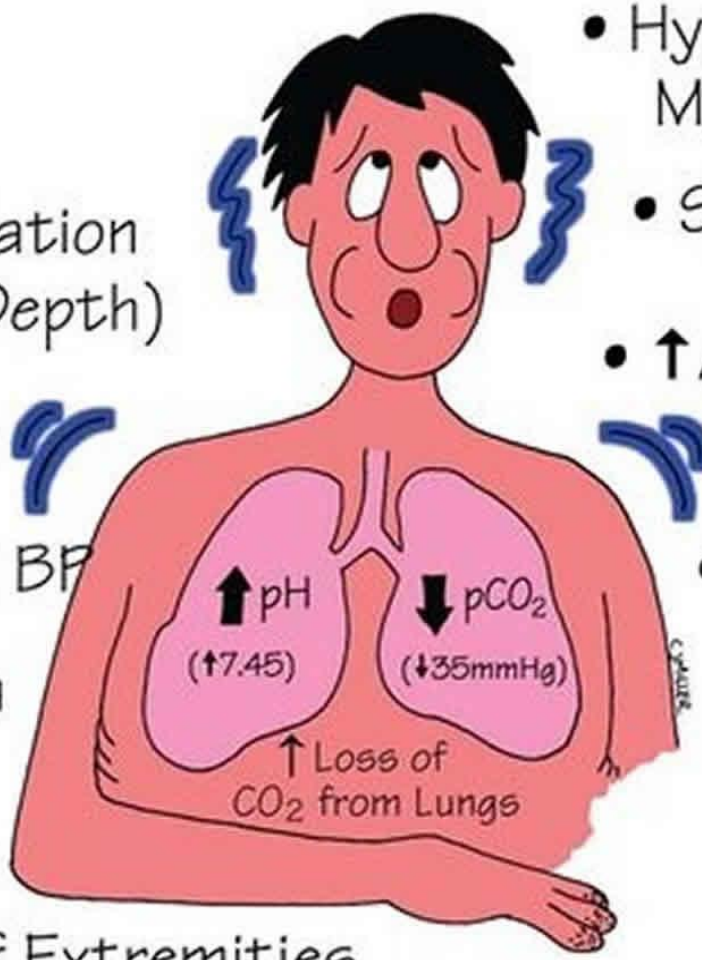
- Pulmonary diseases (pneumonia, asthma, pulmonary edema, pulmonary embolus, interstitial lung disease, high altitude, hypotension, severe anemia)

3. Mechanical ventilation:

- High minute ventilation

RESPIRATORY ALKALOSIS

- Hyperventilation (↑Rate & Depth)
- Tachycardia
- ↓or Normal BP
- Hypokalemia
- Numbness & Tingling of Extremities

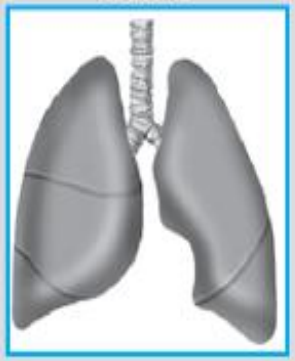


- Hyper Reflexes & Muscle Cramping
- Seizures
- ↑Anxiety, ↑Irritablity
- Causes:
  Hyperventilation (Anxiety, PE, Fear)
  Mechanical Ventilation

Respiratory alkalosis

(Deficit of carbonic acid in the extracellular fluid due to hyperventilation)

Lungs



Kidney

Ventilation is affected. Treatment would be recommended.

Urine is alkaline. Kidneys excrete base (bicarbonate) and retain acid.

Treatment: Remove the cause. Rebreathe expired air, e.g., CO_2, from a paper bag. Antianxiety drugs, e.g., Valium (diazepam), Ativan (lorazepam).

Respiratory alkalosis: treatment

Correction of the primary disorder of the respiratory alkalosis is extremely important:

Anxiety-hyperventilation syndromes = rebreathing into a paper or plastic bag, mild sedation and reassurance hypoxia O_2

Salicylates = urinary alkalinization, forced diuresis, dialysis
Sepsis = antibiotics
Hyperthyroidism = β-Blockers, antithyroid medications
Asthma = bronchodilators, corticosteroids
Pulmonary edema = diuretics, improvement in CHF
Pulmonary embolism = O_2, anticoagulation

Mechanical ventilation = ↓ Ventilatory rate and tidal volume, ↑ dead space, mild sedation without skeletal muscle paralysis

Acidemia vs. acidosis
Alkalemia vs. alkalosis

Acidemia and alkylemia refere to a physiologic state dependent solely upon arterial pH.

Acidemia: pH < 7.35

Alkalemia: pH > 7.45

Acidemia vs. acidosis
Alkalemia vs. alkalosis

Acidosis and alkalosis refer to the individual processes which tend to drive the pH towards acidemia and alkalemia, respectively.

Acidemia vs. acidosis
Alkalemia vs. alkalosis

A patient can be acidemic or alkalemic, but not both.

However,

a patient can have one or more acidosis and one or more alkalosis which can directly offset one another in some cases this may even result in an overall normal pH.

Approaching an ABG

1. Measure electrolytes and ABG simultaneously or within few minutes apart.
2. Check the validity of blood pH—use Henderson equation.
3. Identify the primary disorder.
4. Calculate the AG* (correct for low albumin, if indicated).
5. Identify the causes of the primary disorder.
6. Calculate the expected compensation (also called secondary response).
7. Identify the mixed acid–base disorder, if any.
8. Use $\Delta AG/\Delta HCO_3^-$ ratio appropriately.
9. Determine the appropriate treatment.

"Life is a struggle, not against sin, not against the Money Power, not against malicious animal magnetism, but against hydrogen ions."

H.L. MENCKEN