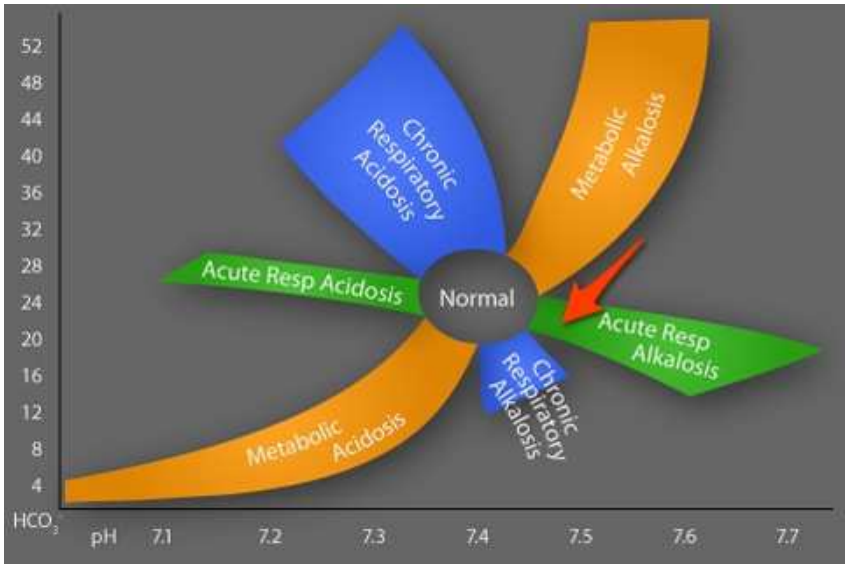
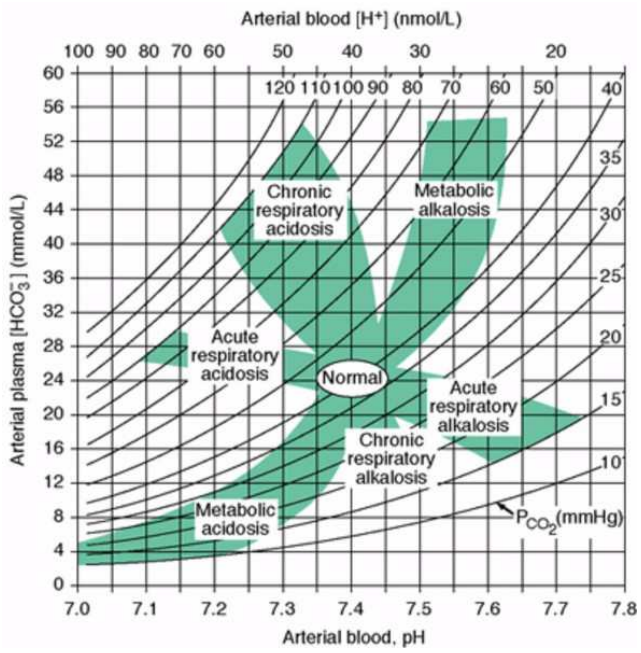
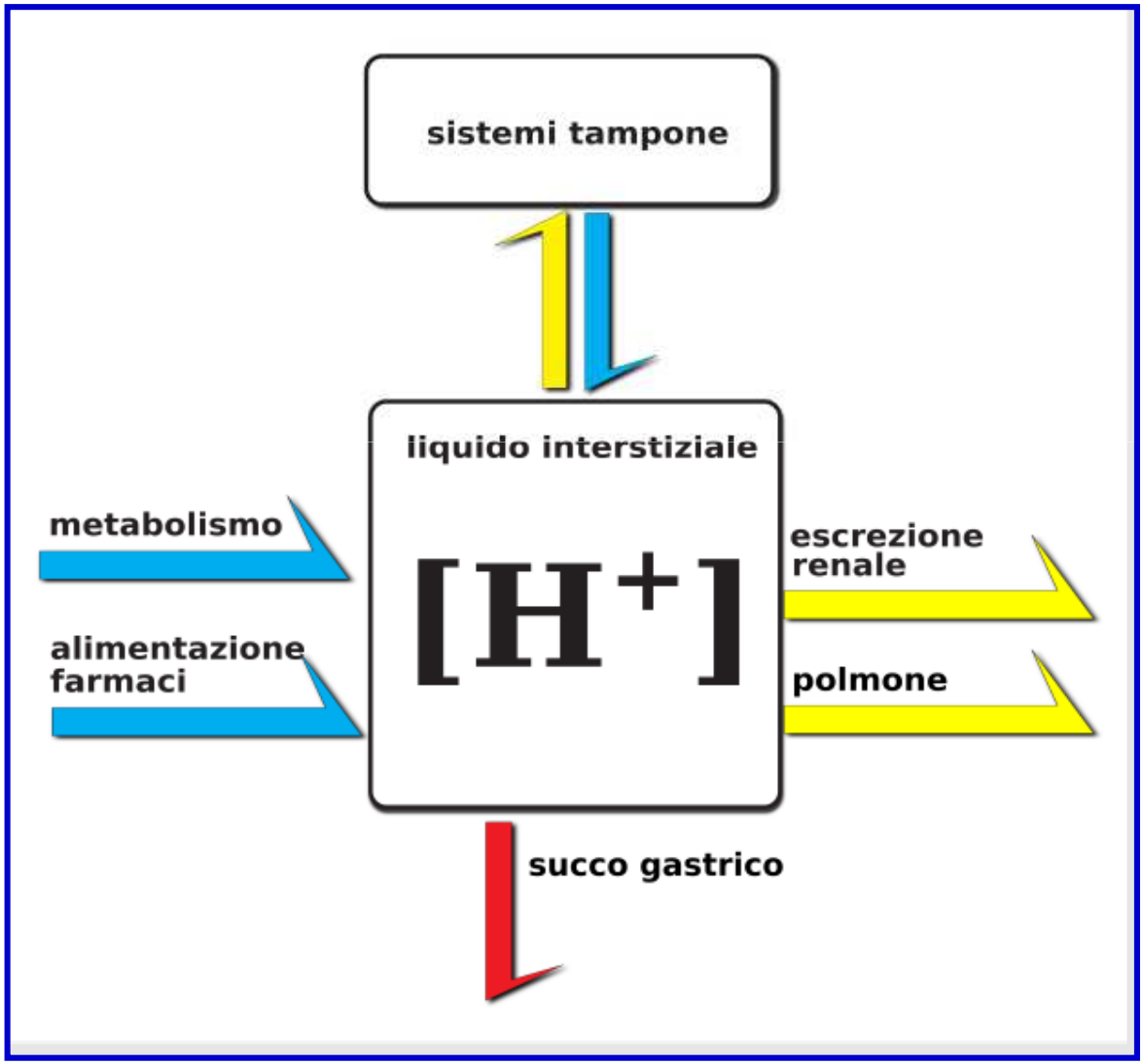
Acid Base Disorders

Prof. G. Zuliani





PREMESSA


sistemi tampone
liquido interstiziale
[H+]
metabolismo
alimentazione
farmaci
escrezione
renale
polmone
succo gastrico

$$pH = \log \frac{1}{[H+]}$$

La respirazione a livello tissutale è mantenuta da:

- ***Apporto di O_2 dai polmoni ai tessuti ottenuto con il trasporto nel sangue per mezzo della emoglobina***

- ***Trasporto di CO_2 dai tessuti ai polmoni ottenuto per lo più idratando la CO_2 ad acido carbonico = H_2CO_3 (che si dissocia in HCO_3^- + H^+) e successivamente viene scisso negli alveoli in CO_2 + H_2O.***

I bicarbonati: da una parte servono per eliminare la CO_2, dall'altro per mantenere il pH nei range.

Il mantenimento del **pH** nel range di normalità è funzione di una normale $PaCO_2$ (circa 40 mmHg) e di una normale quantità di bicarbonati (circa 24 mEq/L).
La **$PaCO_2$** varia al variare della **funzione respiratoria**, come l'**HCO_3^-** si modifica in base alla **funzionalità renale**: ne consegue che l'attività respiratoria e quella renale sono fondamentali nella omeostasi dell'equilibrio acido – base.

$$CO_2 + H_2O \leftrightarrow H_2CO_3 \leftrightarrow HCO_3^- + H^+$$

polmone **rene**

Esiste un equilibrio dinamico tra H_2CO_3 e CO_2 da una parte (insieme all'H_2O) e HCO_3^- e H^+ dall'altra.
Ogni variazione degli ioni di idrogeno e bicarbonato o dell'anidride carbonica determinano una alterazione dell'equilibrio con spostamenti a sinistra o a destra, che dovranno essere corretti dall'**apparato respiratorio** (che con un aumento della ventilazione smaltirà la quantità eccedente di CO_2) e dall'**apparato renale** (che aumenterà o ridurrà la eliminazione degli ioni H^+ e HCO_3^-)

Il metabolismo dell'organismo deve quindi mantenere costantemente in equilibrio il **pH,** attraverso alcuni principali ***meccanismi tampone***:

• **Escrezione renale di bicarbonati: $CO_2 + H_2O \Leftrightarrow H_2CO_3 \Leftrightarrow HCO_3^- + H^+$** (gli H^+ vengono escreti attraverso il rene, mentre i bicarbonati vengono riassorbiti nel tubulo renale – ***LENTO***)

• **Escrezione respiratoria CO2** (attraverso l'iperventilazione o l'ipoventilazione - ***RAPIDO***)

• **Escrezione renale di fosfati: $NaHPO_4 + H^+ + HCO_3^- \rightarrow H_2PO_4$** (escreto nelle urine) **+ $NaHCO_3$** (riassorbito) (nella pratica legano gli H^+ e li eliminano nelle urine = è il principale sistema tampone a livello renale)

• **Escrezione renale di ammoniaca: $Na^+R + NH_3 + H^+ + HCO3^- \rightarrow NaHCO3^-$** (riassorbito) **+ NH^+4R** (escreto nelle urine) (R è un qualunque acido non volatile

Un sistema tampone è costituito da un acido e dalla sua base coniugata ed è in grado di rilasciare o legare H+ a seconda delle necessità.

Buffers in the human body

Buffer	Acid	Conjugate base	Main buffering action
hemoglobin	HHb	Hb^-	erythrocytes
proteins	HProt	$Prot^-$	intracellular
phosphate buffer	$H_2PO_4^-$	HPO_4^{2-}	intracellular
bicarbonate	$CO_2 \rightarrow H_2CO_3$	HCO_3^-	extracellular

Questi meccanismi contribuiscono a garantire la condizione di equilibrio acido-base indispensabile alla omeostasi dell'organismo, secondo l'equazione di **Henderson-Hasselblach** ovvero:

$$pH = 6{,}1_{(K)} + \log \frac{HCO_3}{0{,}03 * PaCO_2}$$

(anione)

(acido indissociato)

Il rapporto tra HCO3 e CO2 deve restare costante per mantenere costante il pH; per ogni aumento/diminuzione della CO_2 dovremo avere un reciproco aumento o diminuzione di HCO_3^- e viceversa, se vogliamo mantenere costante il pH.

$$pH = 6.1 + \log \frac{HCO3^-}{0.030 \times PaCO_2}$$

$$pH = 6.1 + \log \frac{24\ mEq/L}{0.030 \times 40\ mmHg}$$

$$pH = 6.1 + \log \frac{24\ mEq/L}{1.2\ mEq/L}$$

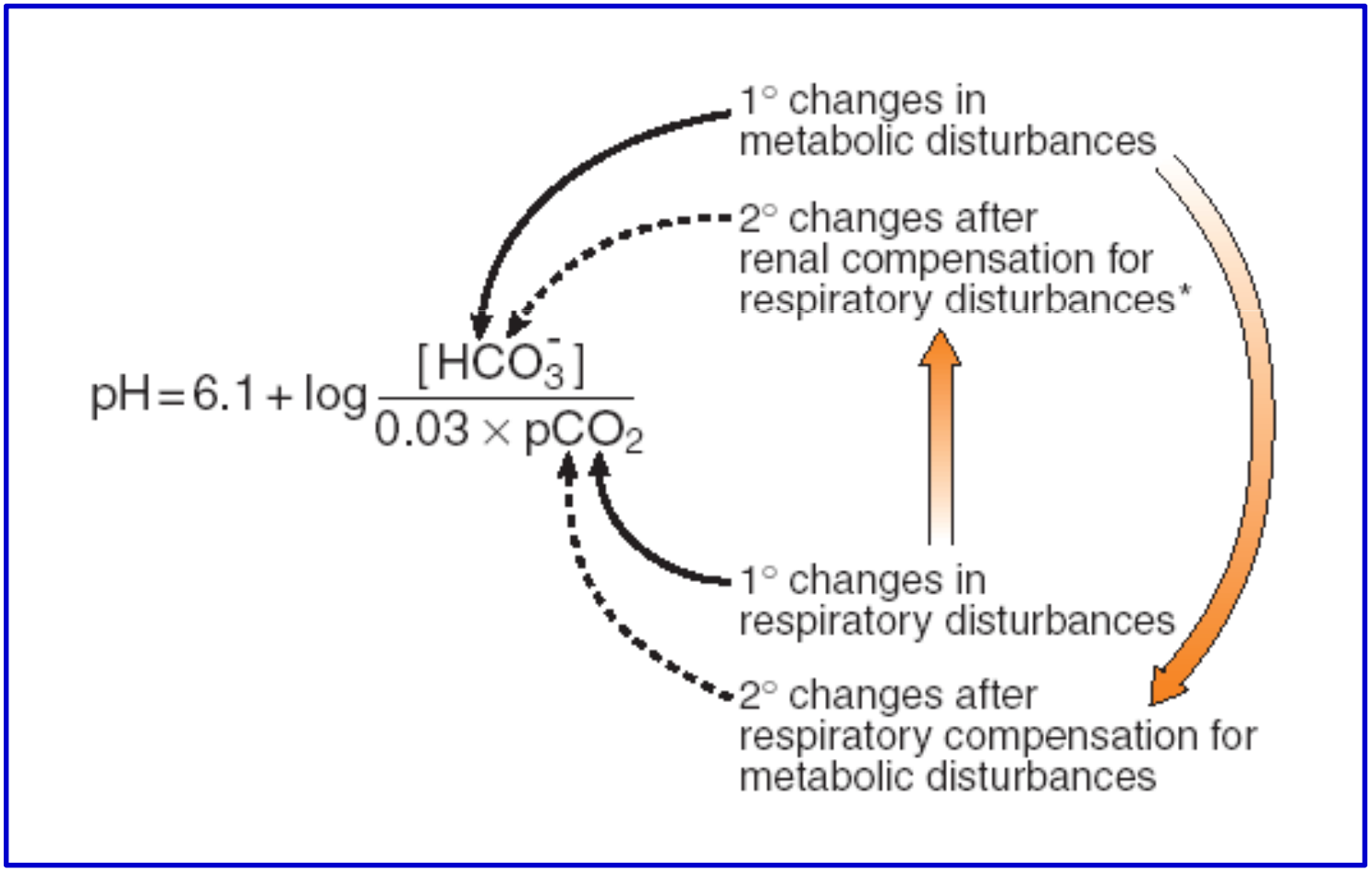
$$pH = 6.1 + \log (24/1.2)$$

$$pH = 6.1 + \log 20$$

$$pH = 6.1 + 1.3 = \mathbf{7.40}$$

Squilibrio primitivo respiratorio → compenso "metabolico"

Squilibrio primitivo metabolico → compenso "respiratorio"



1) Compenso respiratorio

I chemorecettori dei centri respiratori bulbari / glomo carotideo modificano la ventilazione: se acidosi metabolica (riduzione HCO3) si avrà iperventilazione, se alcalosi (aumento HCO3) si avrà ipoventilazione.

2) Compenso renale

Ad una alterazione del pH si avrà un movimento di HCO_3^- per tentare di ristabilire l'equilibrio. Questo movimento può avvenire per modifica del riassorbimento di HCO_3^- o modifica della rigenerazione di HCO_3^-.

2A) Riassorbimento di bicarbonati

Praticamente tutti i bicarbonati vengono riassorbiti (questo è correlato alla secrezione di H^+. Il riassorbimento è parzialmente modulato anche da K^+ e $PaCO_2$. Infatti l'aumento di CO_2 incrementa il riassorbimento di HCO_3^- e viceversa.

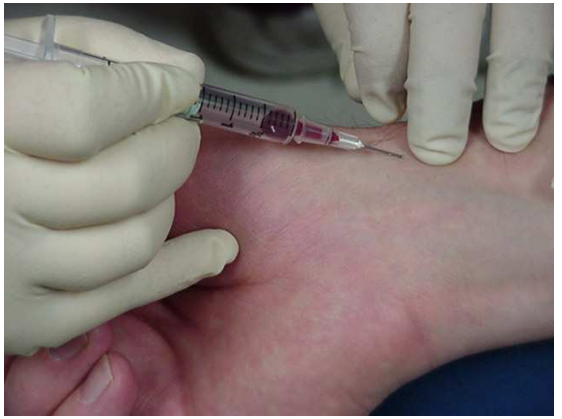
2B) Rigenerazione dei bicarbonati

Le cellule distali del nefrone infatti idratano l'anidride carbonica e la scindono in H^+ e HCO_3^- : il protone (H) viene escreto nelle urine assieme a fosfati o ammoniaca, mentre lo ione bicarbonato rientra nel circolo ematico.

When to get an arterial blood gas (ABG – EGA)

Most often indicated after finding:

- ***Acute or chronic respiratory failure***
- ***Abnormal electrolyte panel***
- ***Evaluate an ingestion***
- Always draw one at the same time as the electrolyte panel



The elements of the ABG

- pH
- pCO_2
- pO_2
- HCO_3^-
- Base excess or deficit
- O_2 saturation
- Methemoglobin, Carboxyhemoglobin

ABG - EGA

- **Desired Ranges**

  - pH: 7.35 - 7.45
  - $PaCO_2$: 35-45 mmHg
  - PaO_2: 80-100 mmHg
  - HCO_3: 21-27 (24 mean)
  - Base Excess: +/-2 mEq/L
  - O_2sat.: >96% (venous > 75%)

Important points for assessing tissue oxygenation

- This is the Oxigen that is available at the tissue level.

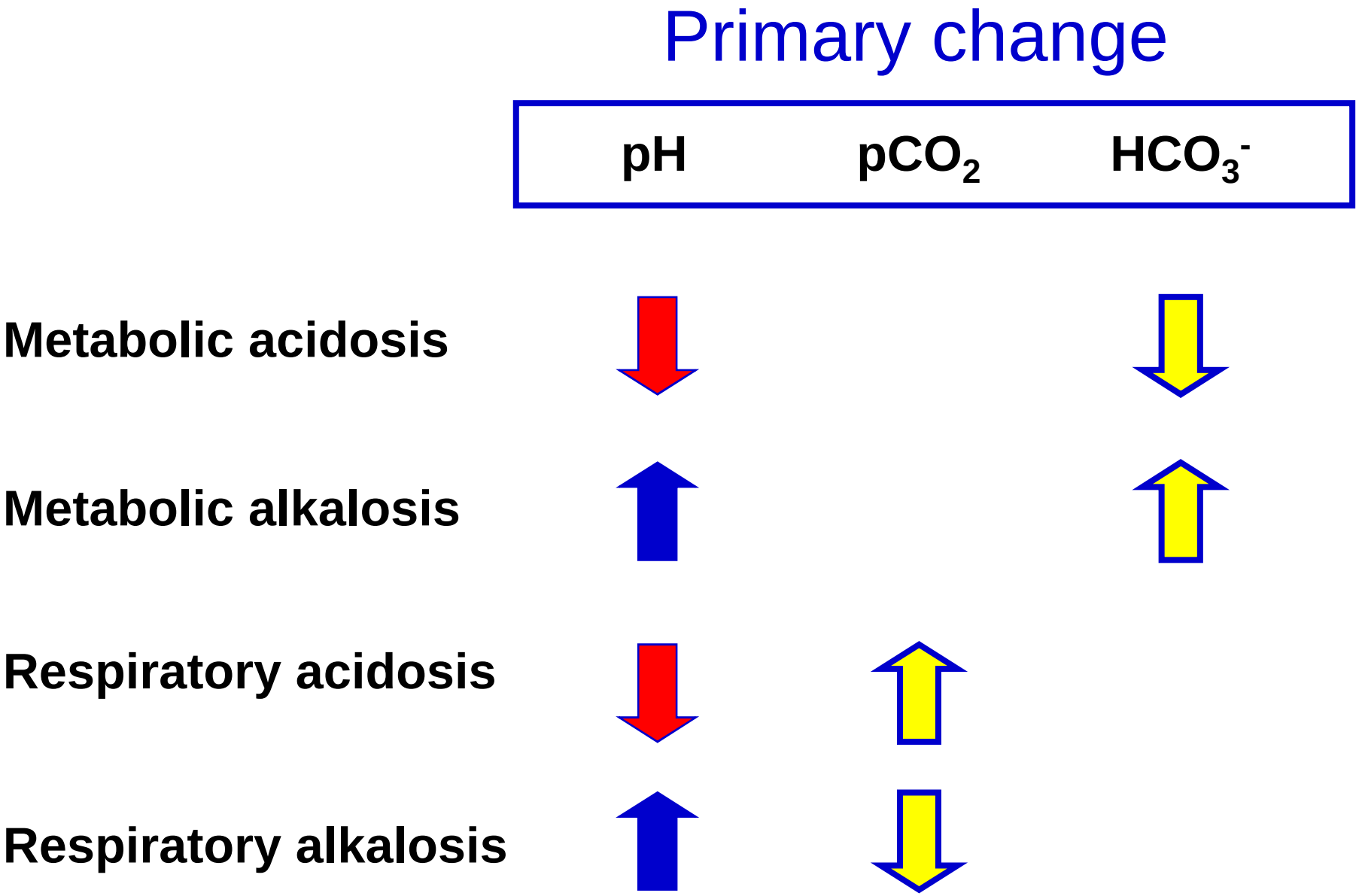
Also are important to check:

- Is **Haemoglobin** normal?
  - Low Hb means the ability of the blood to carry the O2 to the tissues is decreased
- Is **Perfusion** normal?
  - Low perfusion means the blood is not even getting to the tissues

The Terms

- **ACIDS**
  - Acidemia (pH↓)
  - Acidosis
    - Respiratory
      ↑CO_2
    - Metabolic
      ↓HCO_3

- **BASES**
  - Alkalemia (pH↑)
  - Alkalosis
    - Respiratory
      ↓CO_2
    - Metabolic
      ↑HCO_3


Primary change
pH
pCO_2
HCO_3^-
Metabolic acidosis
Metabolic alkalosis
Respiratory acidosis
Respiratory alkalosis

Acute vs Chronic

expected compensatory responses to acid–base disturbances

	For every	Expect	
Metabolic acidosis	1 ↓ HCO_3	1 ↓ pCO_2	FAST
Metabolic alkalosis	10 ↑ HCO_3	7 ↑ pCO_2	
Respiratory acidosis, acute	10 ↑ pCO_2	1 ↑ HCO_3	FAST
Respiratory acidosis chronic	10 ↑ pCO_2	4 ↑ HCO_3	SLOW
Respiratory alkalosis, acute	10 ↓ pCO_2	2 ↓ HCO_3	
Respiratory alkalosis, chronic	10 ↓ pCO_2	5 ↓ HCO_3	

Assuming a normal ABG of pH 7.4, pCO_2 40, HCO_3^- 24, and utilizing meq L^{-1} or mmol L^{-1} for bicarbonate and mmHg for pCO_2, the mnemonic is 1 for 1, 10 for 7, 1, 4, 2, 5

Anion Gap (Normale:12mEq/L +/- 4)

- **Anion Gap = (Na + UC) - (Cl + HCO3 + UA)**

Unmeasured cations (UC): Calcium, magnesium, and gamma globulins. These are very rarely increased enough to modify the anion gap (K)
Unmeasured anions (UA): **Lactate, phosphate, sulfate, ketones, metabolites of some poisons, plasma proteins.**

Se la carica elettrica è mantenuta “neutra”:

$$Na + UC = Cl + HCO3 + UA$$

$$UA - UC = Na - (Cl + HCO3)$$

Since UC= usually costant

$$\mathbf{UA\ (AG) = Na - (Cl + HCO3)}$$

Metabolic Acidosis = Anion Gap: ↑

(**acidi organici endogeni/esogeni** → **H+** → **consumo HCO3-**
rimpiazzato dall'anione legato al H+)

Diabetic Ketoacidosis (DKA)

Uremia

Lactic: CHF, hypoperfusion, metformin

Paraldehyde

Isoniazid

Methanol

Ethylene Glycol poisoning

Salicylates

AG = Na – (Cl + HCO3)

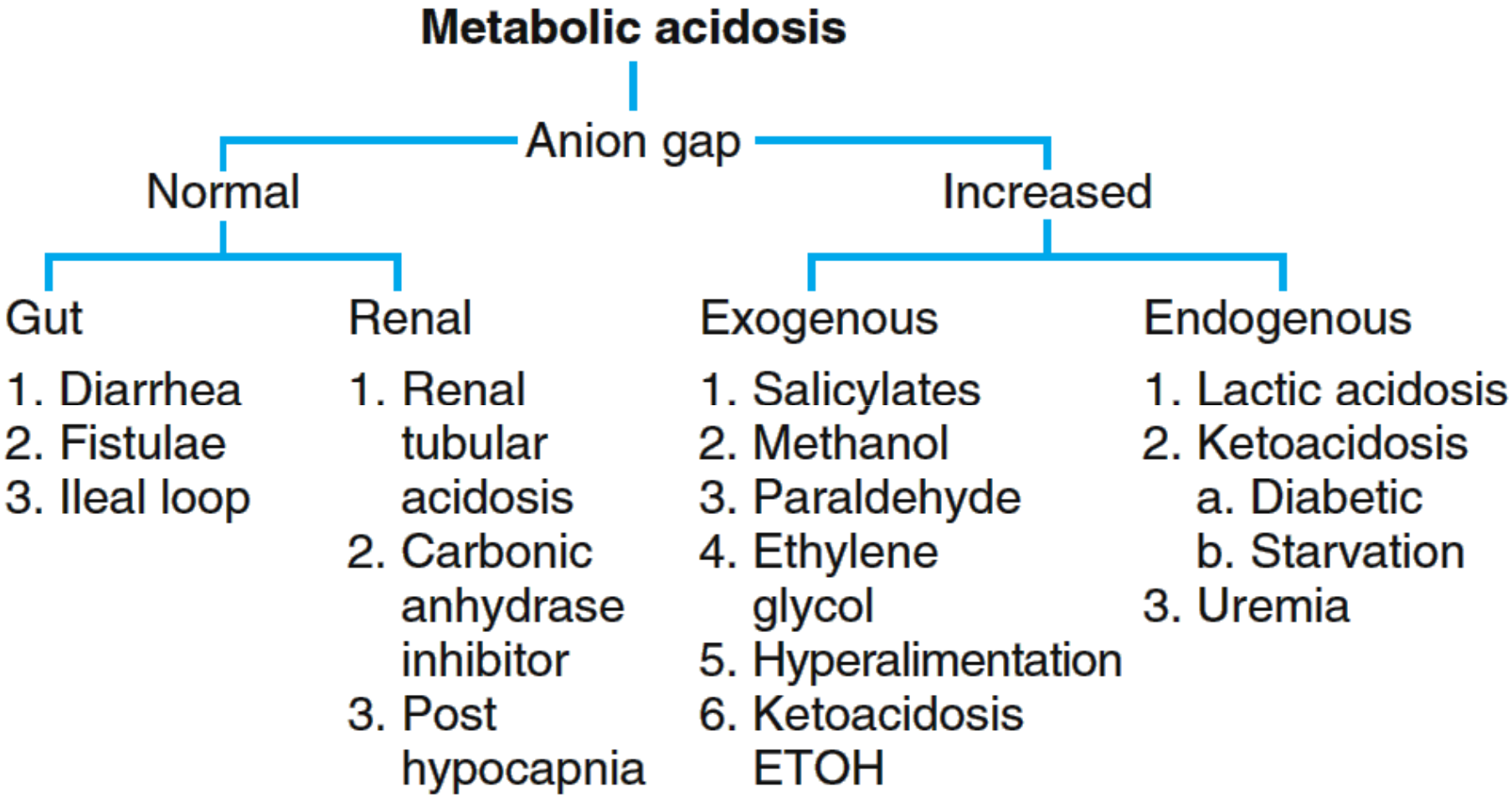
Metabolic Acidosis = Anion Gap: N

(**perdita primitiva di HCO3-** → rimpiazzato dal Cl- = ipercloremia)

Causes of non-AG acidosis

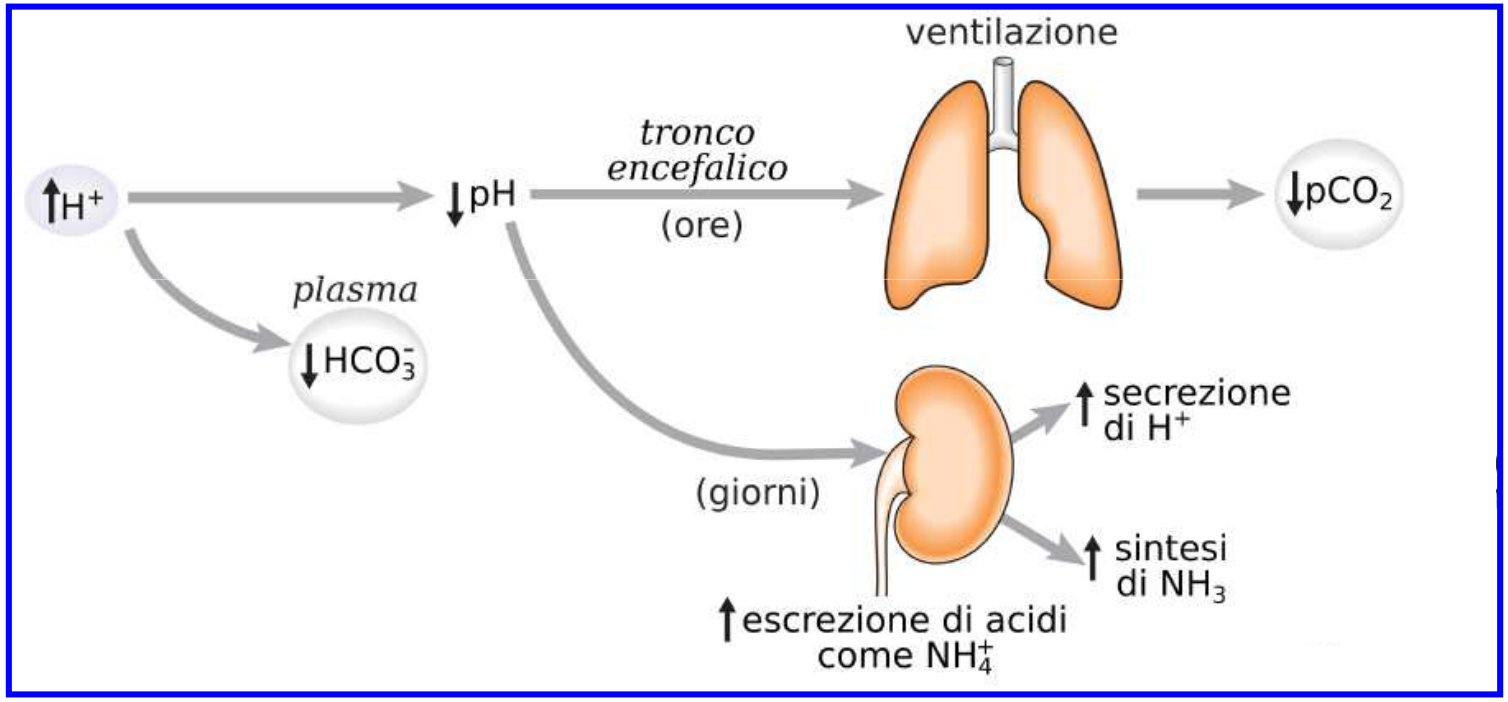
Ureterosigmoidostomy/fistulae
Saline
Early renal insufficiency
Diarrhea
Carbonic anhydrase inhibitors
Amino acids
Renal tubular acidosis
Supplements

AG = Na – (Cl + HCO3)

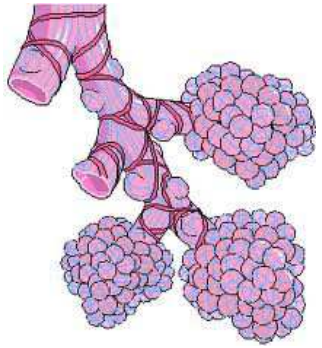

Metabolic acidosis
Anion gap
Normal
Increased
Gut
1. Diarrhea
2. Fistulae
3. Ileal loop
Renal
1. Renal tubular acidosis
2. Carbonic anhydrase inhibitor
3. Post hypocapnia
Exogenous
1. Salicylates
2. Methanol
3. Paraldehyde
4. Ethylene glycol
5. Hyperalimentation
6. Ketoacidosis ETOH
Endogenous
1. Lactic acidosis
2. Ketoacidosis
a. Diabetic
b. Starvation
3. Uremia

AG = Na – (Cl + HCO3)

Acidosi Metabolica: compenso

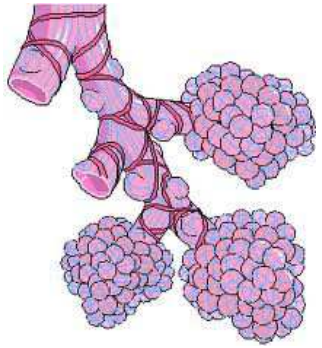


Respiratory Acidosis *(Hypoventilation)*



- CNS
  - Sedatives (iatrogenic or street)
  - Obesity: hypoventilation
- Pleural Disease
  - **Effusion, PTX**
- Intrinsic Lung Disease
  - **COPD**
  - **ARDS**
  - **Pulmonary Embolism**
  - **Pneumonia**
- Musculoskeletal or Neurologic
  - Kyphoscoliosis
  - Guillain-Barre (flaccid paralysis)
  - Myasthenia Gravis
  - Botulism
  - Polymyositis
  - Multiple sclerosis
  - ALS
- Other
  - **OSAS**

Causes of Chronic Respiratory Acidosis



Normal airway and lungs

Central nervous system depression
- Sedative overdose
- Methadone/heroin addiction
- Primary alveolar hypoventilation
- Obesity-hypoventilation syndrome
- Brain tumor
- Bulbar poliomyelitis

Neuromuscular impairment
- Poliomyelitis
- Multiple sclerosis
- Muscular dystrophy
- Amyotrophic lateral sclerosis
- Diaphragmatic paralysis
- Myxedema
- Myopathic disease

Ventilatory restriction
- Kyphoscoliosis, spinal arthritis
- Obesity
- Fibrothorax
- Hydrothorax
- Impaired diaphragmatic function

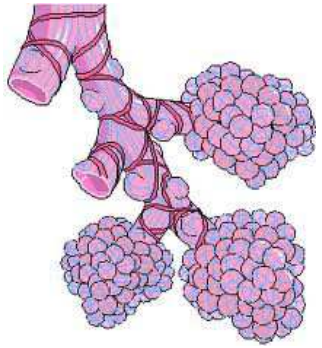
Abnormal airway and lungs

Upper airway obstruction
- Tonsillar and peritonsillar hypertrophy
- Paralysis of vocal cords
- Tumor of the cords or larynx
- Airway stenosis post prolonged intubation
- Thymoma, aortic aneurysm

Lower airway obstruction
- Chronic obstructive lung disease (bronchitis, bronchiolitis, bronchiectasis, emphysema)

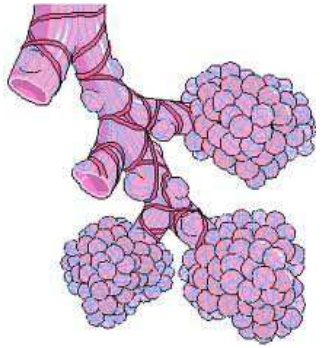
Disorders involving pulmonary alveoli
- Severe chronic pneumonitis
- Diffuse infiltrative disease
- Interstitial fibrosis

Respiratory Acidosis

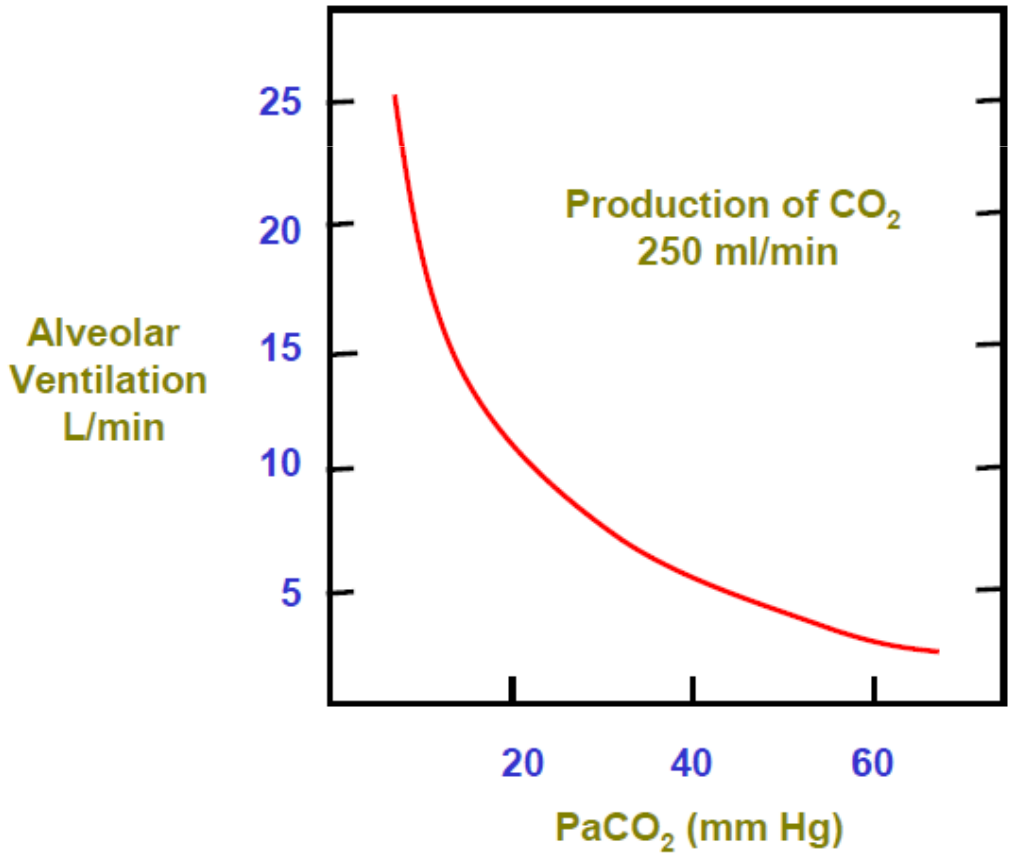


- Acute vs Chronic
  - Acute: Rapid buffering by *Hb* in red cells and *phosphate*, little-no kidney involvement.
    - pH ↓ by 0.08 for 10mmHg ↑ in CO_2
  - Chronic: Renal compensation by elimination of H2CO3 and *retention of* HCO_3- (↓Cl to balance charges ➲ hypochloremia)
    - pH ↓ by 0.03 for 10mmHg ↑in CO_2

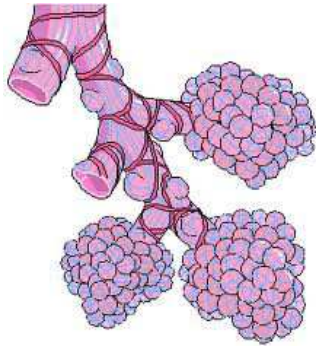
Respiratory Acidosis

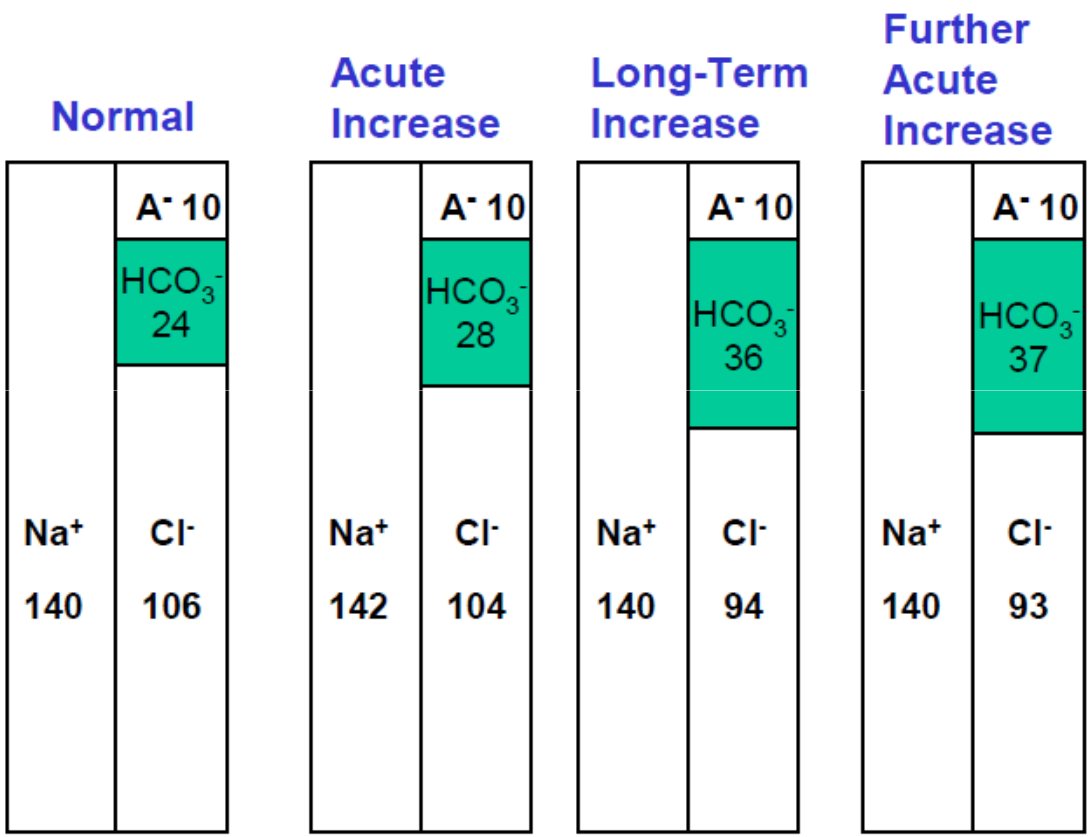


By far, most cases of respiratory acidosis reflect a decrease in alveolar ventilation.



Respiratory Acidosis





Respiratory Acidosis

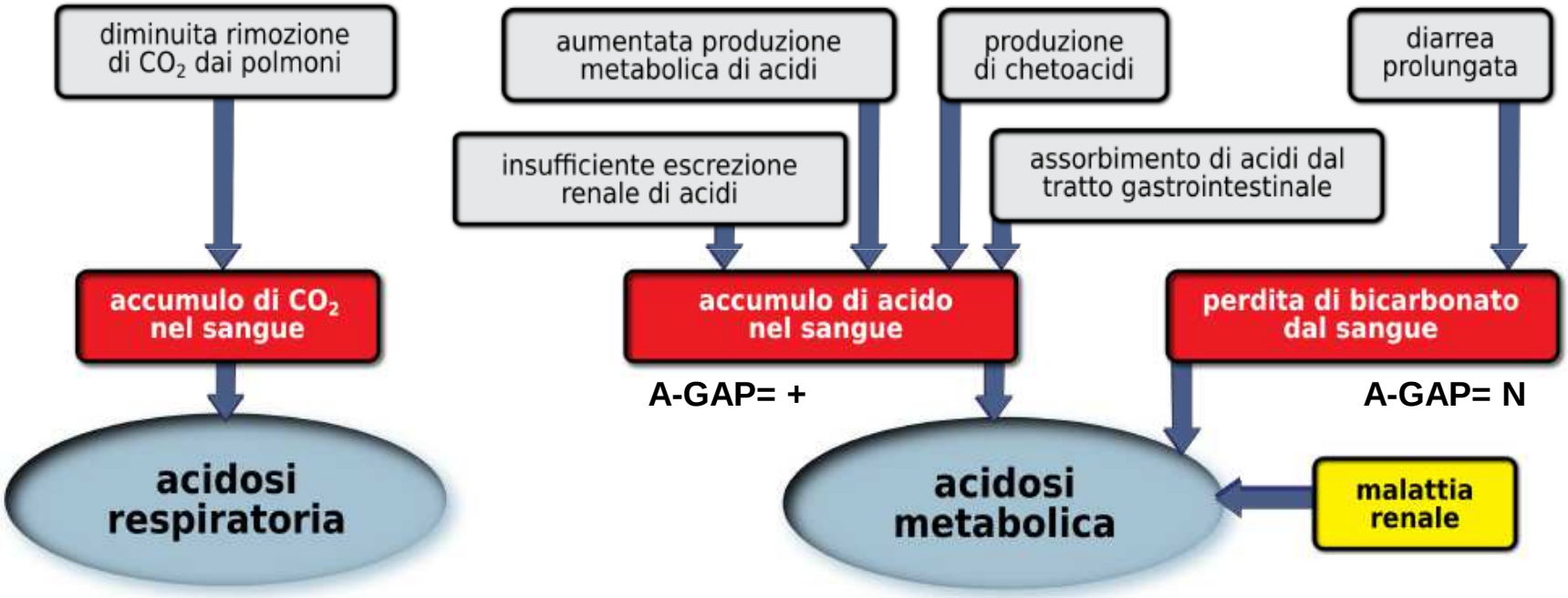
	$PaCO_2$ mm Hg	$[HCO_3^-]$ mEq/L	$[H^+]$ nEq/L	pH
Example #1	50 ↑	20 ↓	60	7.22
Example #2	80	24 **N**	80	7.10
Example #3	60	37 ↑	39	7.41

Diagnosis - Simple respiratory acidosis is characterized by hypercapnia, acidemi secondary increase in plasma bicarbonate concentration. However, respiratory a frequently associated with other acid-base disorders. Examples of such mixed a disorders follow:

	$PaCO_2$ mm Hg	$[HCO_3^-]$ mEq/L	$[H^+]$ nEq/L	pH
Example #1	50	20	60	7.22
Example #2	80	24	80	7.10
Example #3	60	37	39	7.41

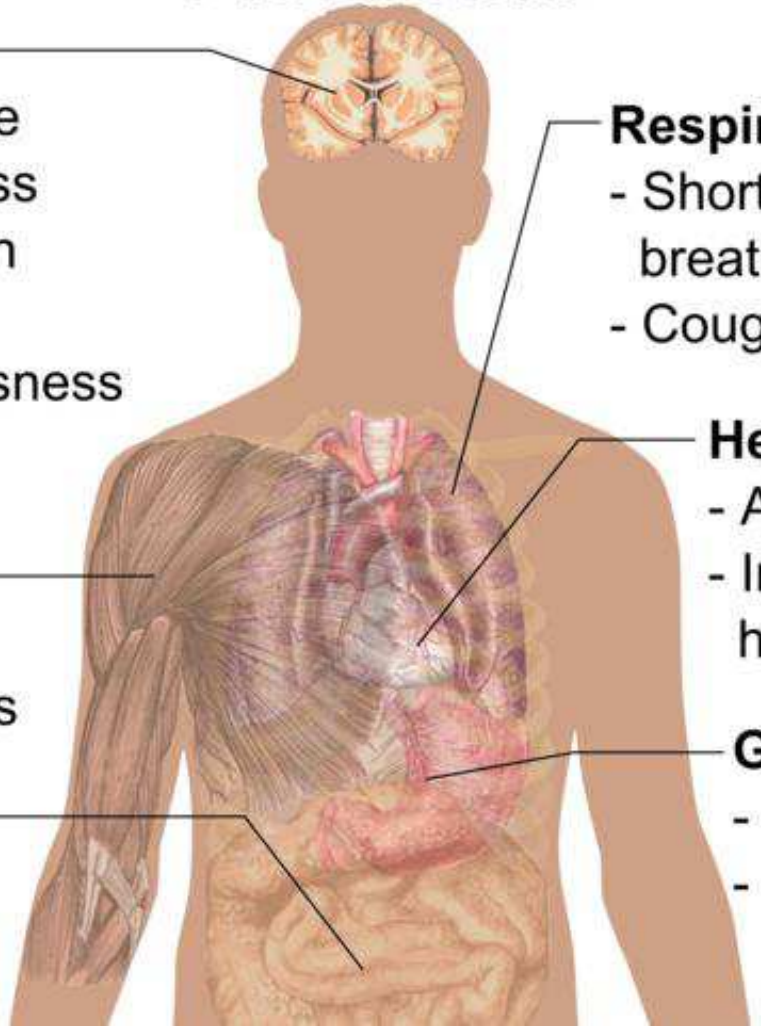
In example #1, hypercapnia is associated with a decreased, rather than an increased, level of plasma bicarbonate, indicating the presence of mixed respiratory acidosis and metabolic acidosis (e.g., a patient with respiratory insufficiency due to chronic obstructive pulmonary disease causing respiratory acidosis, and concomitant circulatory failure from an acute myocardial infarction resulting in lactic acidosis). Similarly, the absence of any increment in plasma bicarbonate concentration despite the prevailing hypercapnia (as in example #2 in which plasma bicarbonate is normal) signifies the presence of mixed respiratory acidosis and metabolic acidosis. In example #3, the increase in plasma bicarbonate level is considerably higher than that appropriate for this level of hypercapnia (even for chronic adaptation) and thus defines the coexistence of respiratory acidosis and metabolic alkalosis (e.g., a patient with respiratory failure due to severe bilateral pneumonia causing respiratory acidosis, and nasogastric suction resulting in metabolic alkalosis).

Summary Acidosis



Symptoms of

Acidosis



Central
- Headache
- Sleepiness
- Confusion
- Loss of consciousness
- Coma

Muscular
- Seizures
- Weakness

Intestinal
- Diarrhea

Respiratory
- Shortness of breath
- Coughing

Heart
- Arrhythmia
- Increased heart rate

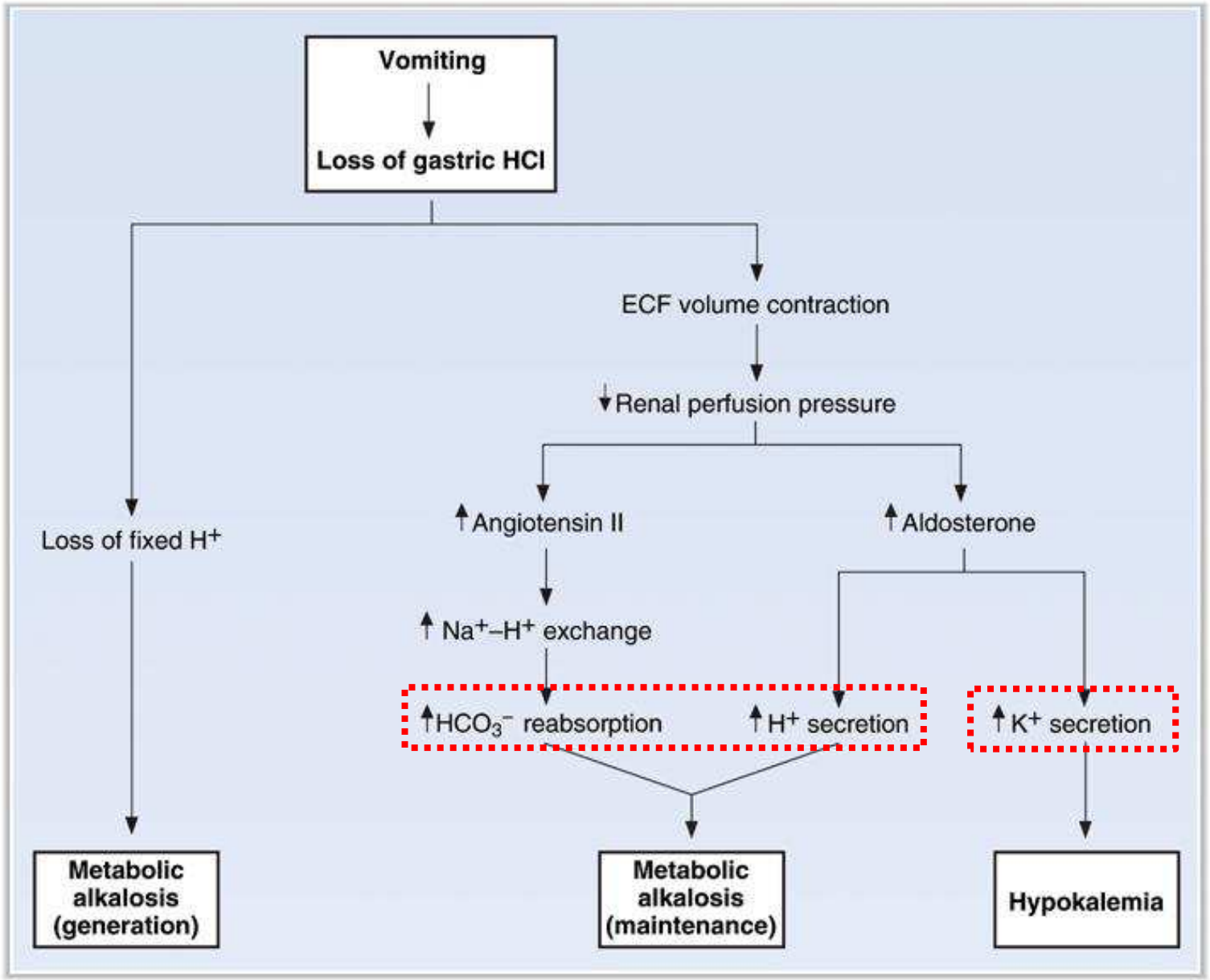
Gastric
- Nausea
- Vomiting

Metabolic Alkalosis

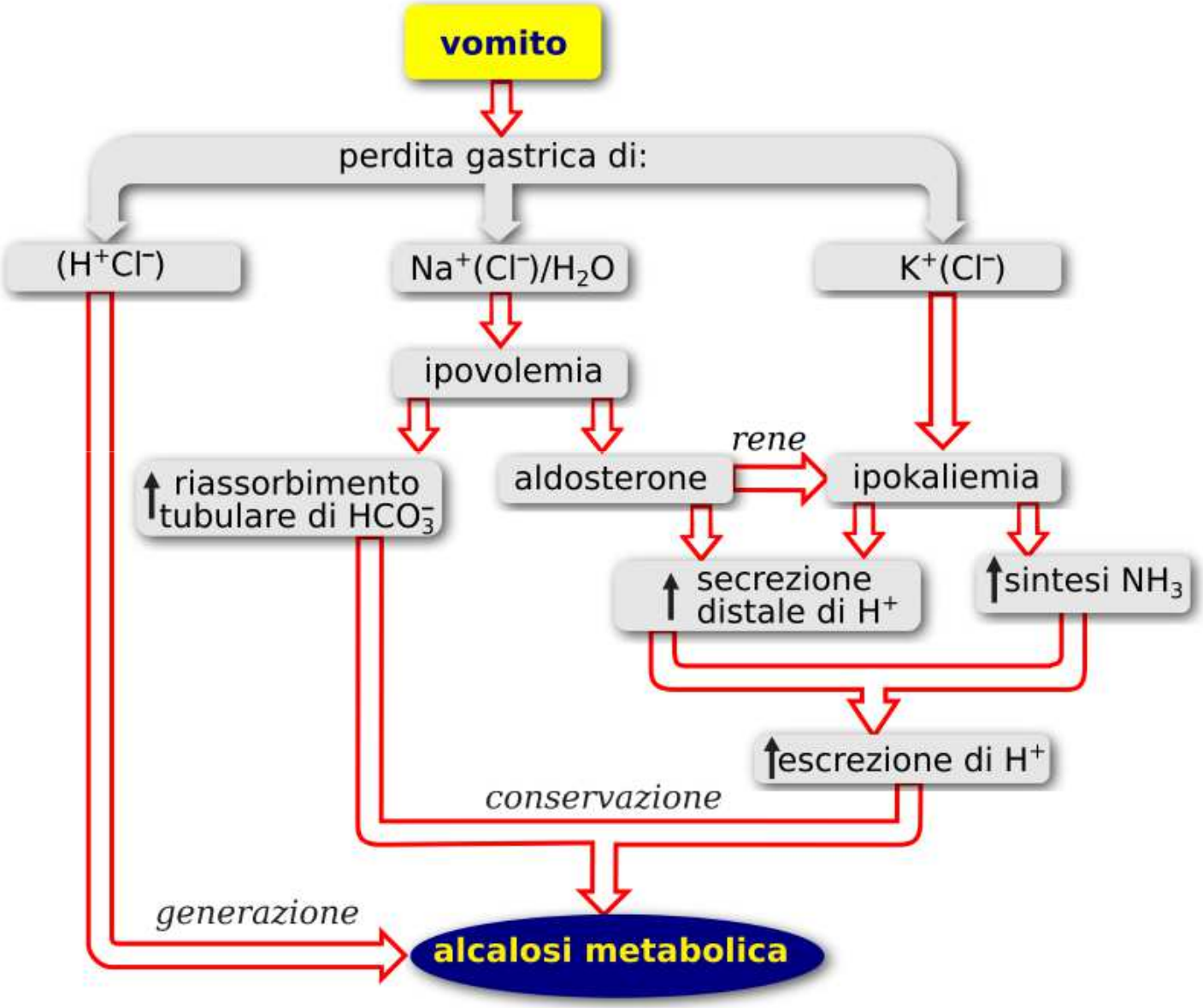
↑pH, ↑**HCO3**
↑PCO2 by 0.7 for every 1mEq/L ↑ in HCO3

- ***Vomiting or NG suction* (loss of H+)**
- ***Fluid volume contraction* (*"contraction alkalosis"*)**
- ***Hypokalemia* (*↑reabsorb HCO3*)**
- Ingestion or administration of bicarbonate
- Massive blood transfusion (citrate)
- Cushing's Disease, glucocorticoid, mineralcorticoid
- Post-chronic hypercapnia
- Bartter's or Gitelman's Syndrome
- "Milk-alkali Syndrome"

Metabolic Alkalosis



Metabolic Alkalosis



Respiratory Alkalosis

↑pH, ↓CO2, ↑**Ventilation**
↓ CO2 ➲ ↓ HCO3 (↑Cl to balance charges ➲ hyperchloremia)

- ***Catastrophic CNS events (brainstem)***
- ***Sepsis***
- ***Pulmonary embolism***
- ***Anxiety***
- ***Pain***
- Early asthma exacerbation or severe CHF
- Pulmonary Fibrosis
- Cirrhosis
- **Pregnancy**

Causes of Respiratory Alkalosis

Hypoxemia or tissue hypoxia
- Decreased inspired O_2 tension
- High altitude
- Bacterial or viral pneumonia
- Aspiration of food, foreign body, or vomitus
- Larygospasm
- Drowning
- Cyanotic heart disease
- Severe anemia
- Left shift deviation of HbO2 curve
- Hypotension
- Severe ciurculatory failure
- Pulmonary embolism

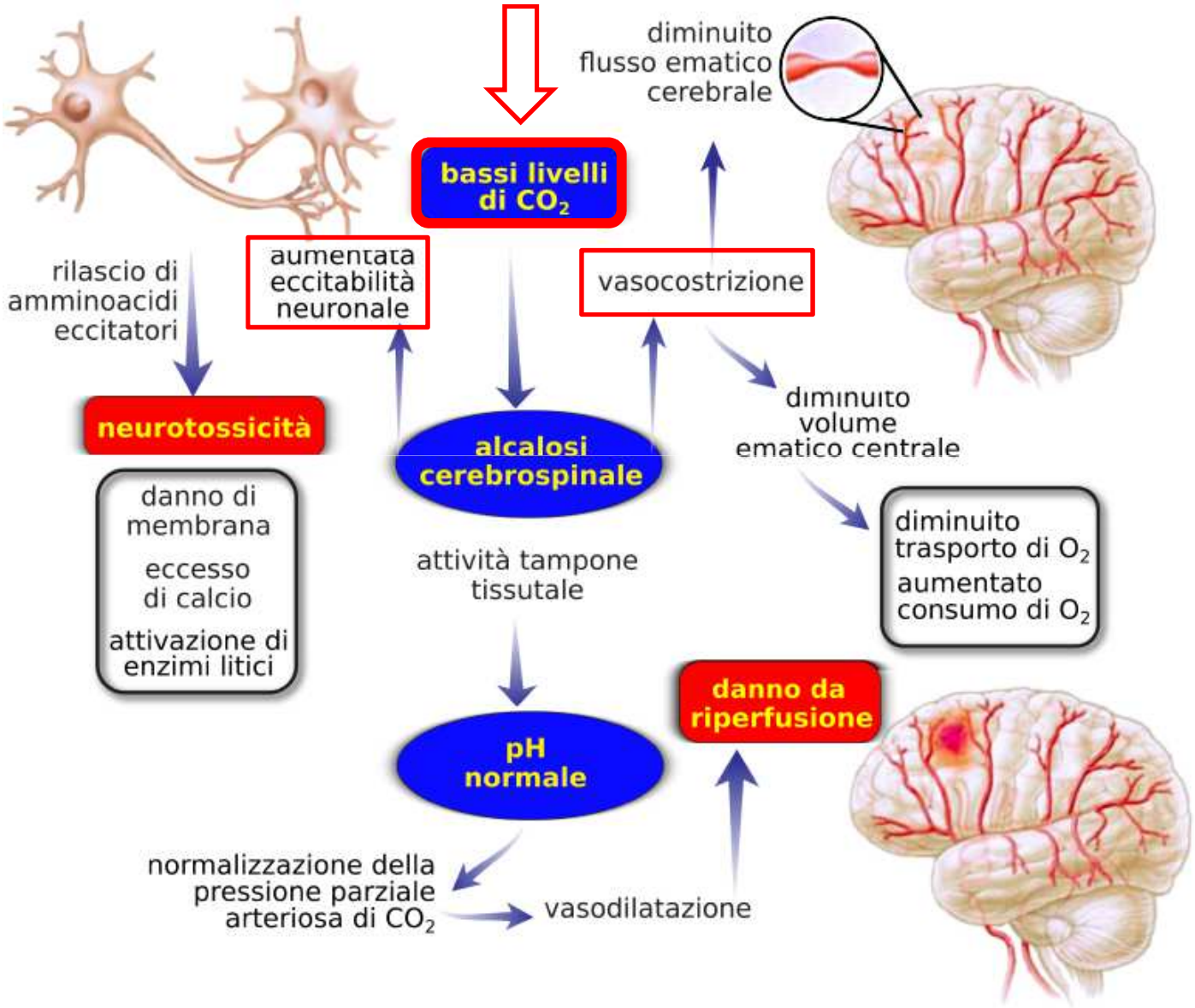
Stimulation of chest receptors
- Pneumonia
- Asthma
- Pneumothorax
- Hemothorax
- Flail chest
- Infant or adult respiratory distress syndrome
- Cardiac failure
- Noncardiogenic pulmonary edema
- Pulmonary embolism
- Interstitial lung disease

Central nervous system stimulation
- Voluntary
- Pain
- Anxiety
- Psychosis
- Fever
- Subarachnoid hemorrhage
- Cerebrovascular accident
- Meningoencephalitis
- Tumor
- Trauma

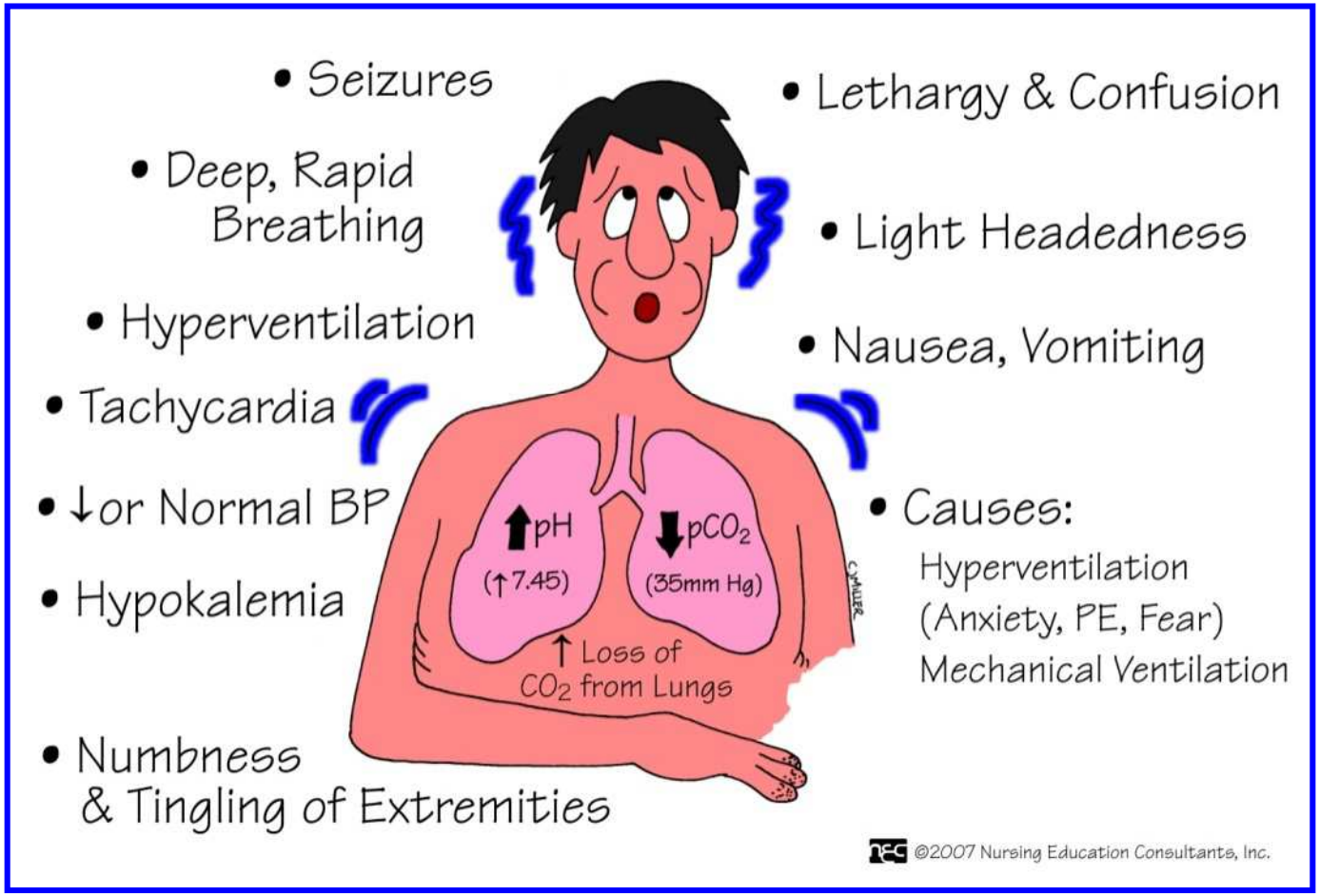
Drugs or hormones
- Nikethamide, ethamivan
- Doxapram
- Xanthines
- Salicylates
- Catecholamines
- Angiotensin II
- Vasopressor agents
- Progesterone
- Medroxyprogesterone
- Dinitrophenol
- Nicotine

Miscellaneous
- Pregnancy
- Sepsis
- Hepatic failure
- Mechanical hyperventilation
- Heat exposure
- Recovery form metabolic acidosis

Respiratory Alkalosis



Respiratory Alkalosis



Respiratory Alkalosis

	$PaCO_2$ mm Hg	$[HCO_3^-]$ mEq/L	$[H^+]$ nEq/L	pH
Example #1	30	36 ↑	20	7.70
Example #2	20	24 **N**	20	7.70
Example #3	15	9 ↓	40	7.40

Diagnosis - Simple respiratory alkalosis is characterized by hypocapnia, alkale secondary decrease in plasma bicarbonate concentration. However, respirator is frequently associated with other acid-base disorders. Examples of such mixe base disorders follow:

	$PaCO_2$ mm Hg	$[HCO_3^-]$ mEq/L	$[H^+]$ nEq/L	pH
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Example #2	20	24	20	7.70
Example #3	15	9	40	7.40

In example #1, hypocapnia is associated with an increased, rather than a decreased, level of plasma bicarbonate, indicating the presence of mixed respiratory alkalosis and metabolic alkalosis (e.g., a patient with hepatic failure causing respiratory alkalosis, and ongoing nasogastric suction resulting in metabolic alkalosis). Similarly, the absence of any decrement in plasma bicarbonate concentration despite the prevailing hypocapnia (as in example #2 in which plasma bicarbonate is normal) signifies the presence of mixed respiratory alkalosis and metabolic alkalosis. In example #3, the decrease in plasma bicarbonate level is considerably greater than that appropriate for this level of hypocapnia (even for chronic adaptation) and thus defines the coexistence of respiratory alkalosis and metabolic acidosis (e.g., a patient with salicylate intoxication in whom stimulation of the ventilatory center in the brain stem accounts for the respiratory alkalosis, whereas the accelerated production of organic acids is responsible for the metabolic acidosis).

Compensated or Uncompensated: what does this mean?

1. **If pH is normal → Compensated**

2. Next evaluate ***pCO2 and HCO3***

   - pH normal + increased pCO2 + increased HCO3
     = compensated respiratory acidosis

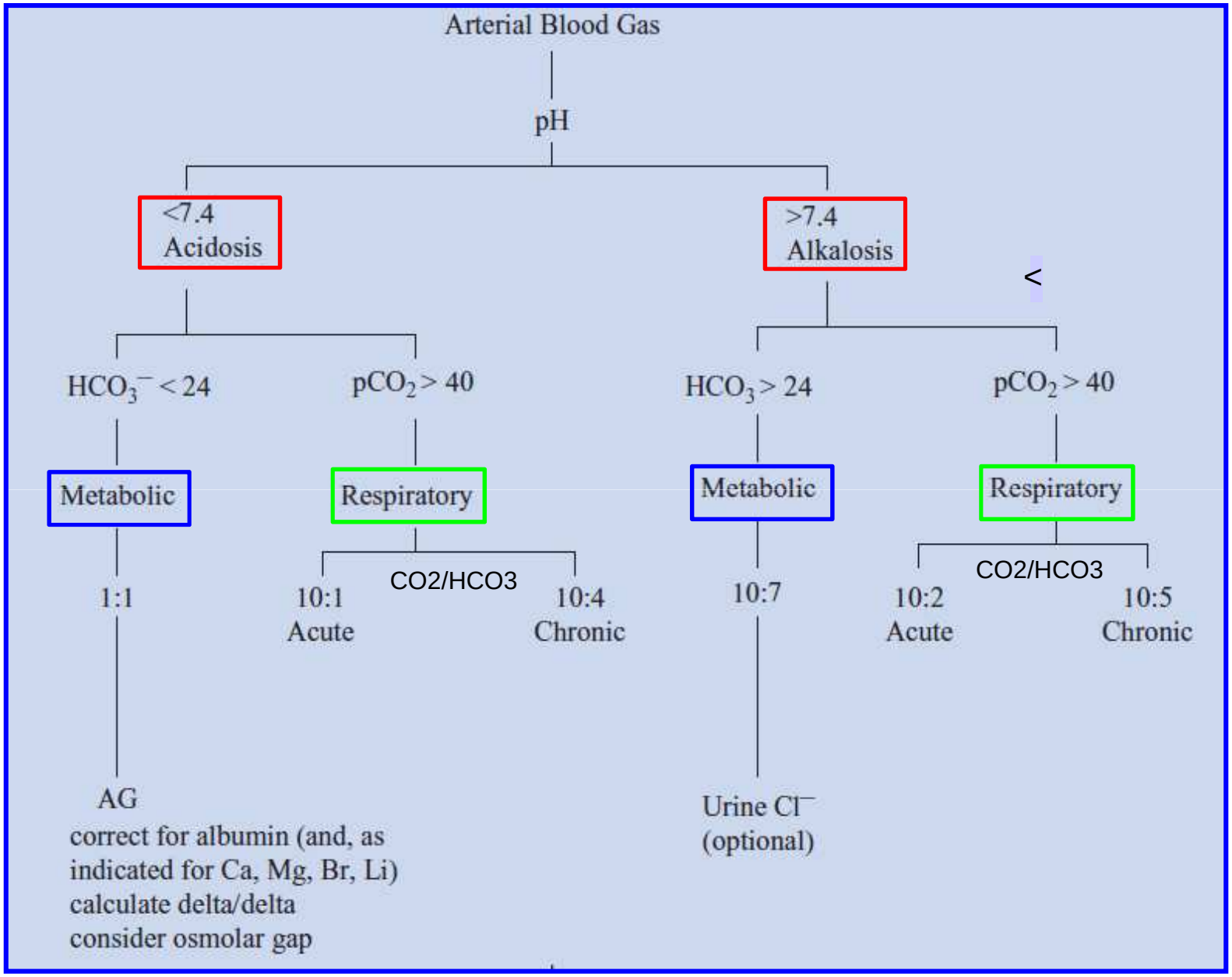
   - pH normal + decreased HCO3 + decreased pCO2
     = compensated metabolic acidosis

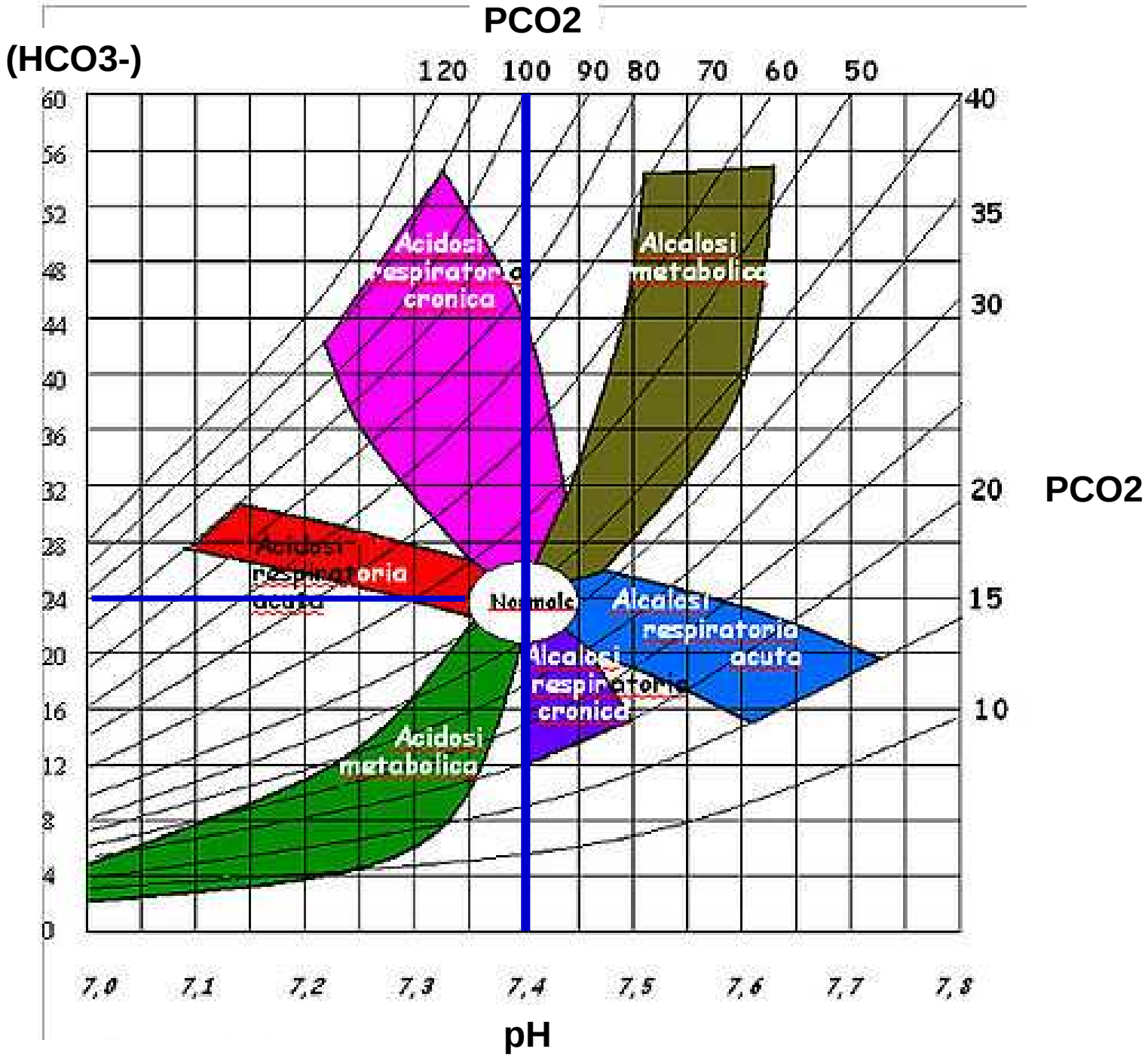
Compensated vs Uncompensated

1. **If pH is NOT normal → Uncompensated**
   - ***Acidosis vs Alkalosis***
   - ***Respiratory vs Metabolic***

- pH<7.30 + pCO2 >50 + high HCO3 = uncompensated respiratory acidosis
- pH<7.30 + HCO3 <18 + normal pCO2 = uncompensated metabolic acidosis

- pH>7.50 + pCO2 <30 + reduced HCO3 = uncompensated respiratory alkalosis
- pH>7.50 + HCO3 >30 + normal pCO2 = uncompensated metabolic alkalosis


Arterial Blood Gas
pH
<7.4
Acidosis
>7.4
Alkalosis
<
HCO_3^- < 24
pCO_2 > 40
HCO_3 > 24
pCO_2 > 40
Metabolic
Respiratory
Metabolic
Respiratory
CO2/HCO3
CO2/HCO3
1:1
10:1
Acute
10:4
Chronic
10:7
10:2
Acute
10:5
Chronic
AG
correct for albumin (and, as
indicated for Ca, Mg, Br, Li)
calculate delta/delta
consider osmolar gap
Urine Cl^-
(optional)


PCO2
(HCO3-)
120 100 90 80 70 60 50
60 56 52 48 44 40 36 32 28 24 20 16 12 8 4 0
40 35 30 20 15 10
PCO2
Acidosi respiratoria cronica
Alcalosi metabolica
Acidosi respiratoria acuta
Normale
Alcalosi respiratoria acuta
Alcalosi respiratoria cronica
Acidosi metabolica
7,0 7,1 7,2 7,3 7,4 7,5 7,6 7,7 7,8
pH

Consequences of acid–base disturbance by organ system

Organ	Acidosis	Alkalosis
Cardiac	Impaired myocardial contractility with decreased cardiac output and hypotension Reentrant dysrhythmias and ventricular fibrillation Catecholamine insensitivity	Reduction in ischemia threshold Refractory dysrhythmias
Peripheral vasculature	Arteriolar dilation Venoconstriction Centralization of blood volume Increased pulmonary vascular resistance (PVR)	Arteriolar constriction Reduction in coronary blood flow Reduction in PVR
Respiratory	Hyperventilation Skeletal muscle weakness Shift of the oxygen–hemoglobin dissociation curve to the right (resulting in desaturation)	Hypoventilation Impaired hypoxic pulmonary vasoconstriction and worsened ventilation–perfusion mismatch Increased hemoglobin affinity for oxygen
Metabolic	Increased metabolic demands Insulin resistance Hyperkalemia Increased protein degradation	Stimulation of organic acid production Decreased plasma electrolyte levels: hypokalemia, hypocalcemia (ionized), hypomagnesemia, and hypophosphatemia)
Central nervous system	Altered mental status and depressed level of consciousness	Reduction in cerebral blood flow if respiratory in origin Reduced seizure threshold Altered mental status and depressed level of consciousness

Case 1. Healthy 18 year old boy presents with Kussmaul breathing

- pH: 7.05
- pCO2: 12 mmHg
- pO2: 108 mmHg
- HCO3: 5 mEq/L
- BE: -30 mEq/L

Case 1. Healthy 18 year old boy presents with Kussmaul breathing

- pH: 7.05
- pCO2: 12 mmHg
- pO2: 108 mmHg
- HCO3: 5 mEq/L
- BE: -30 mEq/L

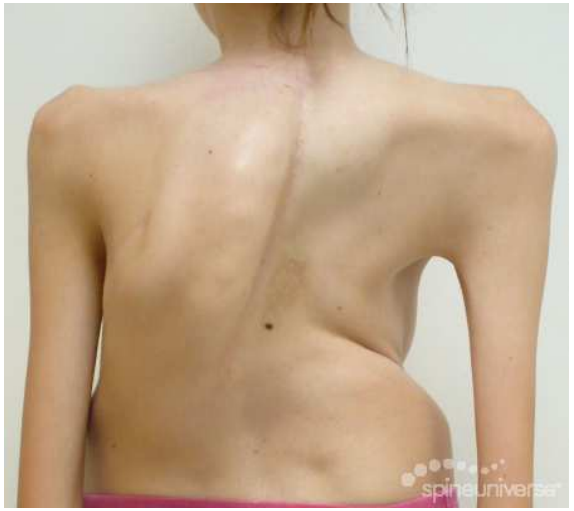
– Severe uncompensated metabolic acidosis due to ketoacidosis

Case 2. 49 year old, kyphoscoliosis, admitted for pneumonia

- pH: 7.40
- pCO2: 25 mmHg
- pO2: 60 mmHg
- HCO3: 14 mEq/L
- BE: -7 mEq/L

Case 2. 49 year old, kyphoscoliosis, admitted for pneumonia

- pH: 7.40
- pCO2: 25 mmHg
- pO2: 60 mmHg
- HCO3: 14 mEq/L
- BE: -7 mEq/L
  - Compensated respiratory alkalosis due to chronic hyperventilation secondary to hypoxia



Case 3. 23 year old, asthma; sitting up and using accessory muscles to breath; audible wheezes

- pH: 7.50
- pCO2: 26 mmHg
- pO2 35 mmHg
- HCO3: 22 mEq/L
- BE: -2 mEq/L

Case 3. 23 year old, asthma; sitting up and using accessory muscles to breath; audible wheezes

- pH: 7.50
- pCO2: 26 mmHg
- pO2 35 mmHg
- HCO3: 22 mEq/L
- BE: -2 mEq/L
  - Uncompensated acute respiratory alkalosis with severe hypoxia due to asthma exacerbation

Case 4. 78 year old post-surgery presenting with chills, fever and hypotension

- pH: 7.25
- pCO2: 32 mmHg
- pO2: 55 mmHg
- HCO3: 10 mEq/L
- BE: -15 mEq/L

Case 4. 78 year old post-surgery presenting with chills, fever and hypotension

- pH: 7.25
- pCO2: 32 mmHg
- pO2: 55 mmHg
- HCO3: 10 mEq/L
- BE: -15 mEq/L

- Uncompensated metabolic acidosis due to low perfusion state and hypoxia

Case 5

A 55 year old insulin-dependent **diabetic woman** was brought to hospital. She was semi-comatose and had been ill for several days. Past history of left ventricular failure; current medication was digoxin and a thiazide diuretic.

Results include: K^+ 2.7, glucose 720 mg/dl, anion gap 34 mmol/l

Arterial Blood Gases
pH 7.27
pCO2 32 mmHg
pO2 82 mmHg
HCO3 22 mmol/l (?)

AG = Na – (Cl + HCO3)

Case 5

A 55 year old insulin dependent **diabetic woman** was brought to hospital. She was semi-comatose and had been ill for several days. Past history of left ventricular failure; current medication was digoxin and a thiazide diuretic.

Results include: K^+ 2.7, glucose 720 mg/dl, anion gap 34 mmol/l

Severe diabetic ketoacidosis complicating a pre-existing metabolic alkalosis (due to thiazide use).

pO2	82 mmHg
HCO3	22 mmol/l (?)

AG = Na – (Cl + HCO3)

Case 6

A 69 year old patient had a **cardiac arrest** following an operation. Resuscitation was commenced including intubation and ventilation. Femoral arterial blood gases were collected about five minutes after.
Other results: **Anion gap 24, Lactate 12 mmol/l.**

Arterial Blood Gases

pH 6.85

pCO2 82 mmHg

pO2 214 mmHg

HCO3 14 mmol/l (?)

AG = Na – (Cl + HCO3)

Case 6

A 69 year old patient had a **cardiac arrest** following an operation. Resuscitation was commenced including intubation and ventilation. Femoral arterial blood gases were collected about five minutes after.
Other results: **Anion gap 24, Lactate 12 mmol/l.**

Cardiac arrest with tissue hypoperfusion causing a severe lactic acidosis. Ventilation is depressed causing a respiratory acidosis.

pO2 214 mmHg

HCO3 14 mmol/l (?)

AG = Na – (Cl + HCO3)

Case 7

A 44 year old moderately dehydrated man was admitted with a history of **acute severe diarrhoea**. Electrolyte results (in mmol/l): **Na^+ 134, K^+ 2.1, Cl^- 113, urea 12.3, creatinine 3.39 mmol/l. Anion gap 10.**

Arterial Blood Gases
pH 7.32
pCO2 33 mmHg
pO2 - not given
HCO3 16 mmol/l
K^+ 2.1 mmol/l

AG = Na – (Cl + HCO3)

Case 7

A 44 year old moderately dehydrated man was admitted with a history of **acute severe diarrhoea**. Electrolyte results (in mmol/l): **Na^+ 134, K^+ 2.1, Cl^- 113, urea 12.3, creatinine 3.39 mmol/l. Anion gap 10.**

This patient has acute diarrhoea causing a mild normal anion gap metabolic acidosis. The volume loss is responsible for pre-renal azotaemia.

HCO3 16 mmol/l
K^+ 2.1 mmol/l

AG = Na – (Cl + HCO3)

Case 8

An 80 year old lady was admitted to the ICU following a motor vehicle accident. She was haemodynamically stable but had **respiratory distress with paradoxical movement of her left anterior chest wall.** No head or neck injury. Past history was of hypertension for which she took propranolol 120 mg/day.

She was intubated and ventilated. Initial ventilation was tidal volume 1,000 mls at a rate of 10 breaths/min with 100% oxygen. Arterial gases (below) were obtained half an hour later. Peripheral perfusion was good. An intravenous infusion was commenced.

Arterial Blood Gases
pH 7.56
pCO2 23 mmHg
pO2 508 mmHg
HCO3 21 mmol/l

Case 8

An 80 year old lady was admitted to the ICU following a motor vehicle accident. She was haemodynamically stable but had **respiratory distress with paradoxical movement of her left anterior chest wall.** No head or neck injury. Past history was of hypertension for which she took propranolol 120 mg/day.

She was intubated and ventilated. Initial ventilation was tidal volume

The diagnosis is acute respiratory alkalosis due to mechanical hyperventilation

Arterial Blood Gases
pH 7.56
pCO2 23 mmHg
pO2 508 mmHg
HCO3 21 mmol/l

Case 9

A 60 year old woman was admitted with **lobar pneumonia.** She was on a thiazide diuretic for 9 months following a previous admission with congestive cardiac failure. The admission arterial blood results were:

Arterial Blood Gases
pH 7.64
pCO2 28 mmHg
pO2 75 mmHg
HCO3 33 mmol/l (?)
K^+ 2.2 mmol/l

Case 9

A 60 year old woman was admitted with **lobar pneumonia.** She was on a thiazide diuretic for 9 months following a previous admission with congestive cardiac failure. The admission arterial blood results were:

A mixed alkalosis: metabolic alkalosis due to to the thiazide diuretic therapy and respiratory alkalosis

pO2 75 mmHg
HCO3 33 mmol/l
K^+ 2.1 mmol/l

Case 10

An elderly woman from a nursing home was transferred to hospital because of **profound weakness and areflexia.** Her oral intake had been poor for a few days. Current medication was a sleeping tablet. Admission biochemistry (in mmol/l): **Na^+ 145, K^+ 1.9, Cl^- 86, bicarbonate >44, anion gap 14.**

Arterial Blood Gases
pH 7.58
pCO2 46 mmHg
pO2 - not given
HCO3 44.4 mmol/l

AG = Na – (Cl + HCO3)

Case 10

An elderly woman from a nursing home was transferred to hospital because of **profound weakness and areflexia.** Her oral intake had been poor for a few days. Current medication was a sleeping tablet. Admission biochemistry (in mmol/l): **Na^+ 145, K^+ 1.9, Cl^- 86, bicarbonate >44, anion gap 14.**

Severe metabolic alkalosis with life-threatening hypokalaemia.

pCO2 49 mmHg

pO2 - not given

HCO3 44.4 mmol/l

Common Clinical States and Associated Acid-Base Disturbances

Clinical State	Acid-Base Disorder
Pulmonary Embolus	Respiratory Alkalosis
Hypotension	Metabolic Acidosis
Vomiting	Metabolic Alkalosis
Severe Diarrhea	Metabolic Acidosis
Cirrhosis	Respiratory Alkalosis
Renal Failure	Metabolic Acidosis
Sepsis	Respiratory Alkalosis/Metabolic Acidosis
Pregnancy	Respiratory Alkalosis
Diuretic Use	Metabolic Alkalosis
COPD	Respiratory Acidosis