Acido-basic disorders

Spac Jiri

Hendersonova-Hasselbalchova equation

$$pH = pK_a + \log \frac{[HCO_3^-]}{[H_2CO_3]}$$

- $pK_a = 6,1$
- $[HCO_3^-] = 24\ mmol.l^{-1}$
- $[H_2CO_3] = 1,2\ mmol.l^{-1}$

Systemic arterial pH is maintained between 7.35 and 7.45 by extracellular and intracellular chemical buffering together with respiratory and renal regulatory mechanisms. The control of arterial CO2 tension (Paco2) by the central nervous system (CNS) and respiratory system and the control of plasma bicarbonate by the kidneys stabilize the arterial pH by excretion or retention of acid or alkali. The metabolic and respiratory components that regulate systemic pH are described by the Henderson-Hasselbalch equation:

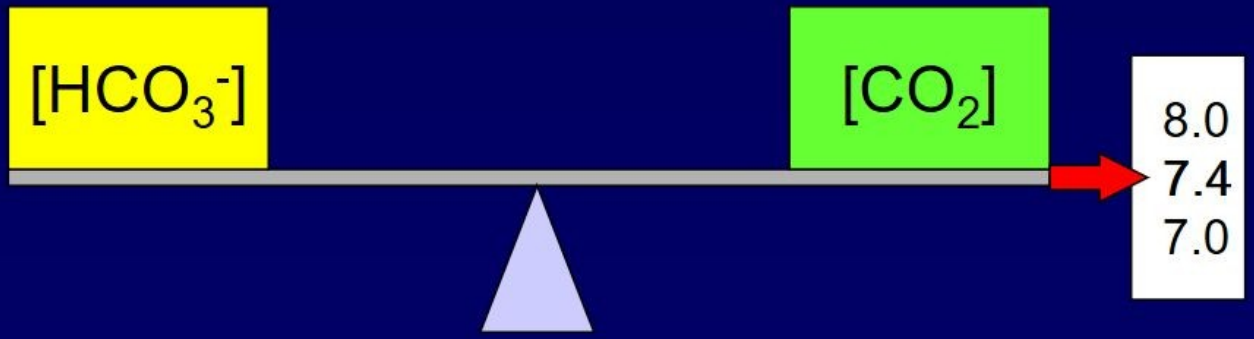
$$[HCO_3^-] \quad 20$$
$$[H_2CO_3]$$

Henderson - Hasselbach

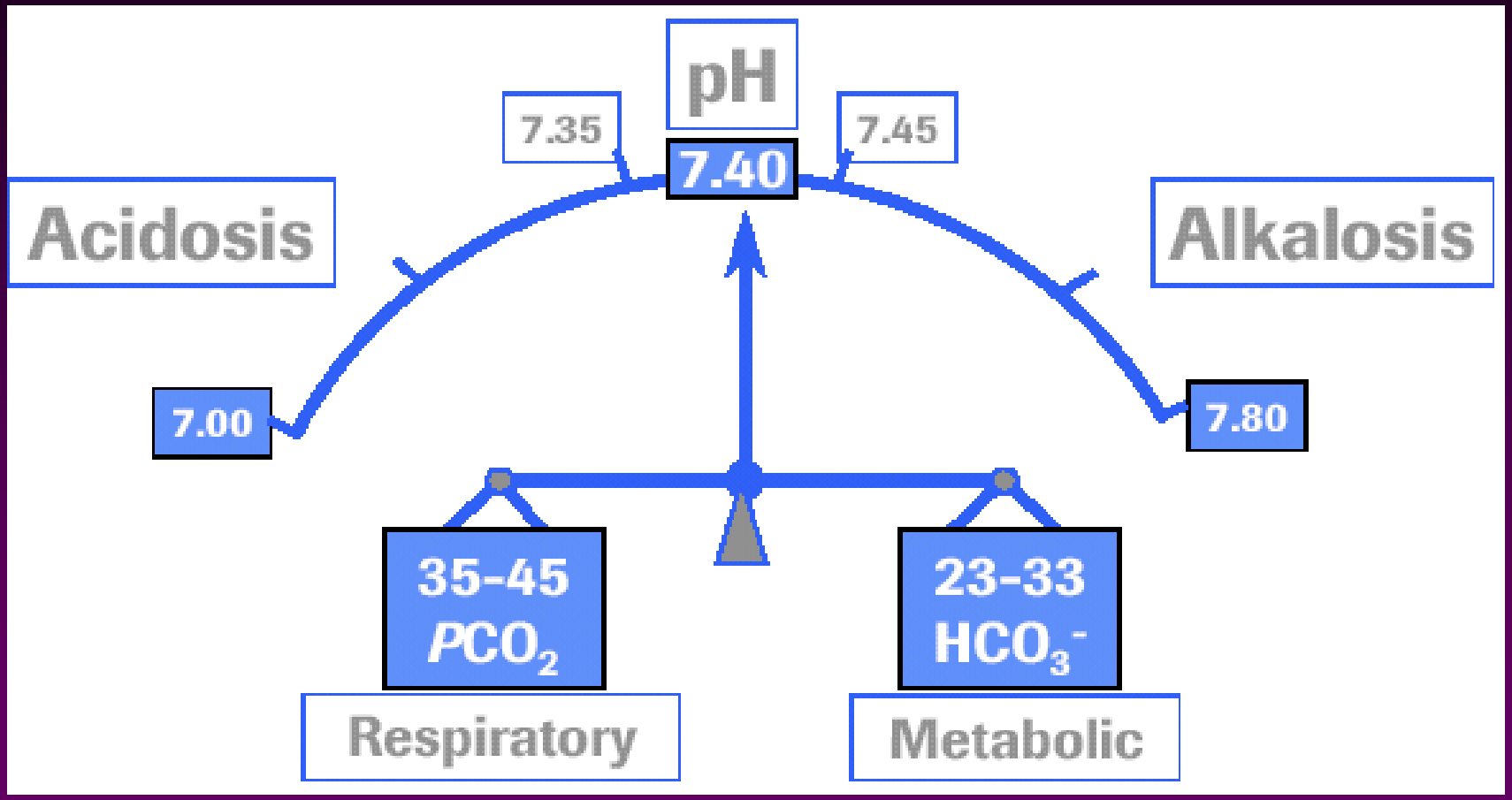
$$- \log [H^+] = \mathbf{pH}$$

$$H^+ + HCO_3^- = CO_2 + H_2O$$

$$\mathbf{pH} = 6.1 + \log \frac{[HCO_3^-]}{P_{CO_2}}$$



Normal acido-basic homeostasis



Systemic arterial pH is maintained between 7.35 and 7.45 by extracellular and intracellular chemical buffering together with respiratory and renal regulatory mechanisms

Preanalytic phase

The most suitable sample is arterial blood. Most often it is taken from a. Radialis into a capillary on a thin needle or into a modified syringe; lithium heparin is used as an anticoagulant. In the intensive care units, an arterial catheter is often inserted to allow repeated donations. In any case, ensure that the sample is taken without air bubbles.

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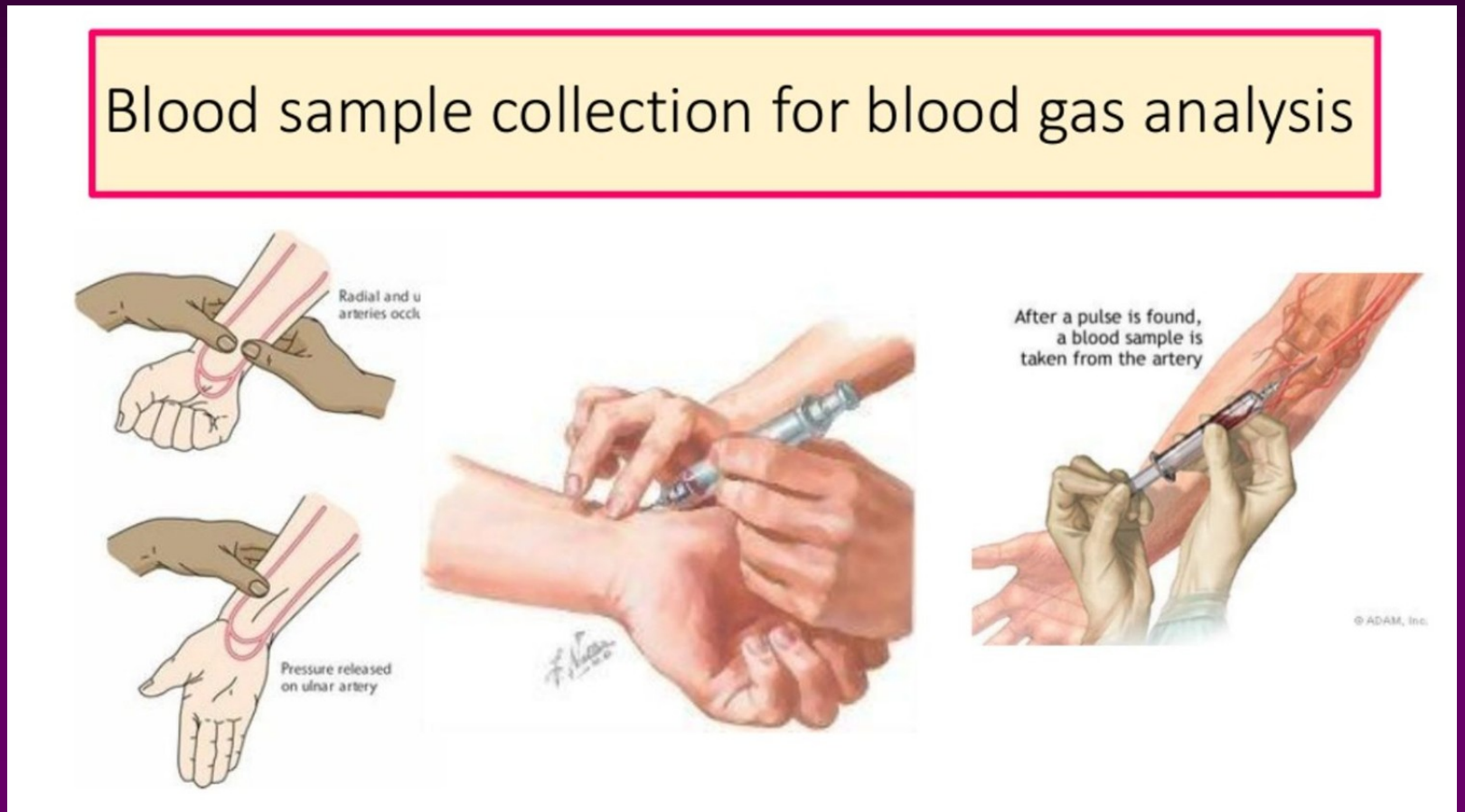
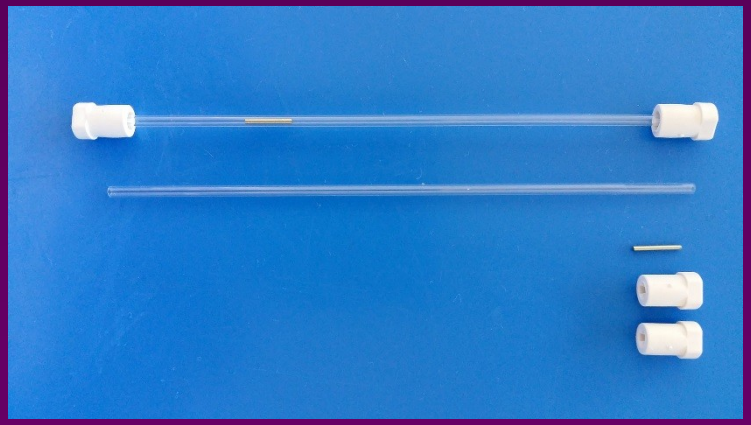


Preanalytic phase

arterialized capillary blood, most commonly from the fingertip or the earlobe. The capillary sample should be composed as much as possible of arterial blood. Therefore, it is necessary to increase the blood flow through the capillaries at the place of collection ("arterialization") - by heating, massages, etc. as much as possible

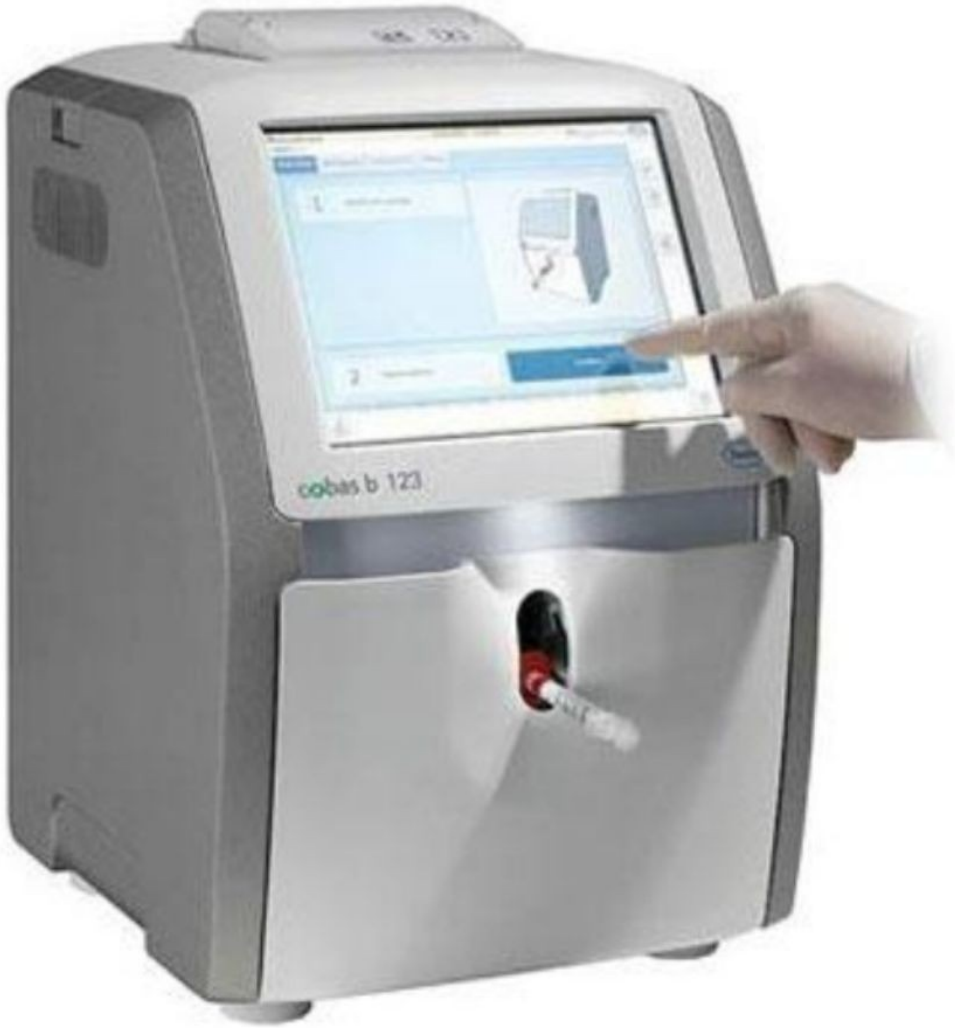
venous blood should be sampled from the central venous bed (central venous catheter, port). Peripheral venous blood does not adequately report the overall metabolic status of the organism, especially in patients with severe centralized circulation. Central venous blood is collected into a syringe with balanced lithium heparin, even in this case the collection must be anaerobic.



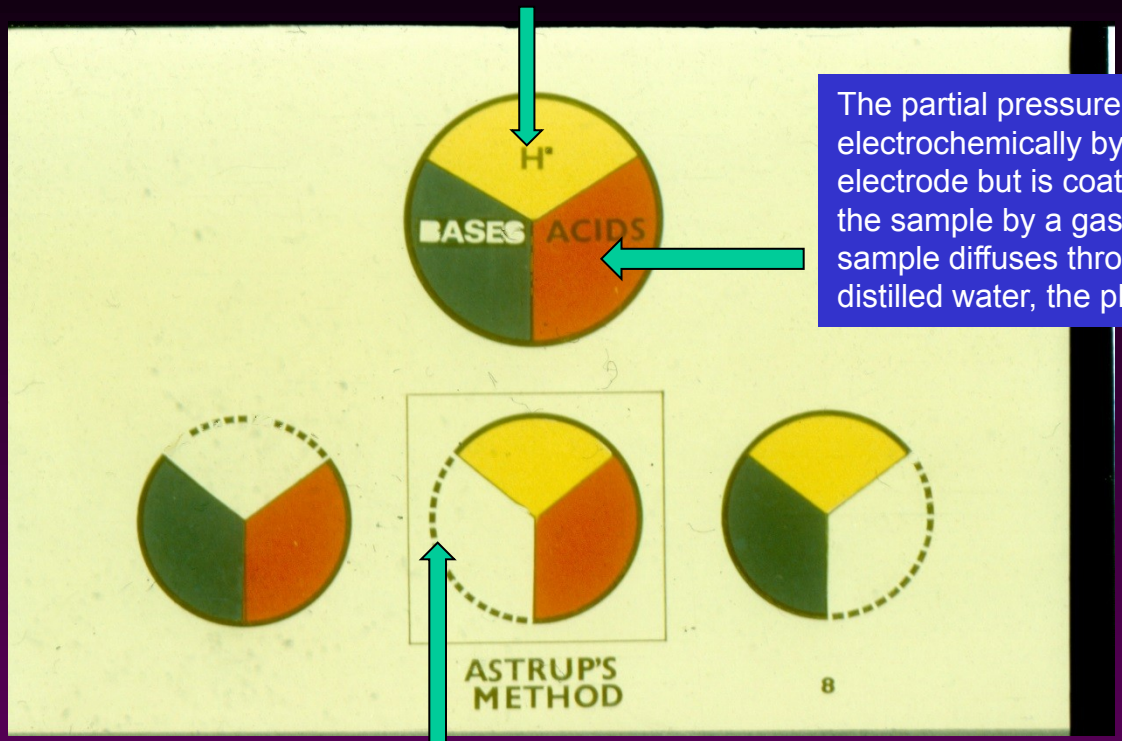
PRINCIPLE OF BLOOD GAS ANALYSER

BLOOD GAS ANALYSER works with three in-built electrodes

1. **pco_2 electrode**
2. **pO_2 electrode**
3. **pH sensitive glass electrode**



The actual pH is determined electrochemically, typically by a miniaturized glass electrode.



The partial pressure of carbon dioxide (pCO2) is determined electrochemically by a Severinghaus electrode. It is also a glass electrode but is coated with a layer of water and separated from the sample by a gas permeable membrane. CO2 from the sample diffuses through the semipermeable membrane into distilled water, the pH of the resulting solution depends on pCO2.

Current and standard bicarbonates are calculated from the measured pH and pCO2 values,

Current HCO3 - This parameter indicates the current concentration of bicarbonate in the blood being examined. Since it depends on both the metabolic and respiratory components of acid-base balance, its interpretation is complicated.

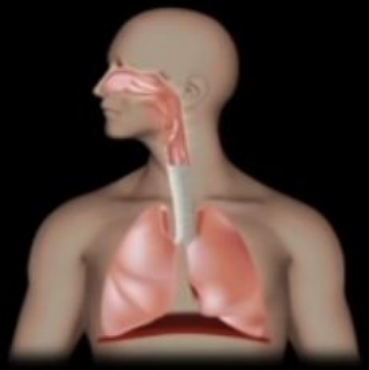
Standard HCO3 - what would be the concentration of bicarbonate in the blood sample examined after elimination of the respiratory disorder, ie after saturation of the blood to pCO2 = 5.3 kPa.
It therefore only informs about the metabolic component of the acid-base balance.

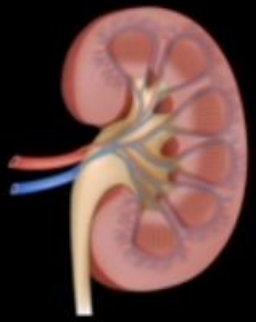
***Base excess*, BE** evaluates only the metabolic component of the acid-base balance.
It is defined as the amount of strong acid that would need to be added to the sample to reach a pH of 7.4, provided that the respiratory disorder ABR (ie pCO2 = 5.3 kPa) is excluded. In metabolic acidosis, a strong base would have to be add the corresponding parameter is referred to as base deficiency, base deficiency, BD, or (more often) expressed as negative BE.

Acid-Base Disturbances

Acidosis

- *Respiratory*
- *Metabolic:* HCO_3^-

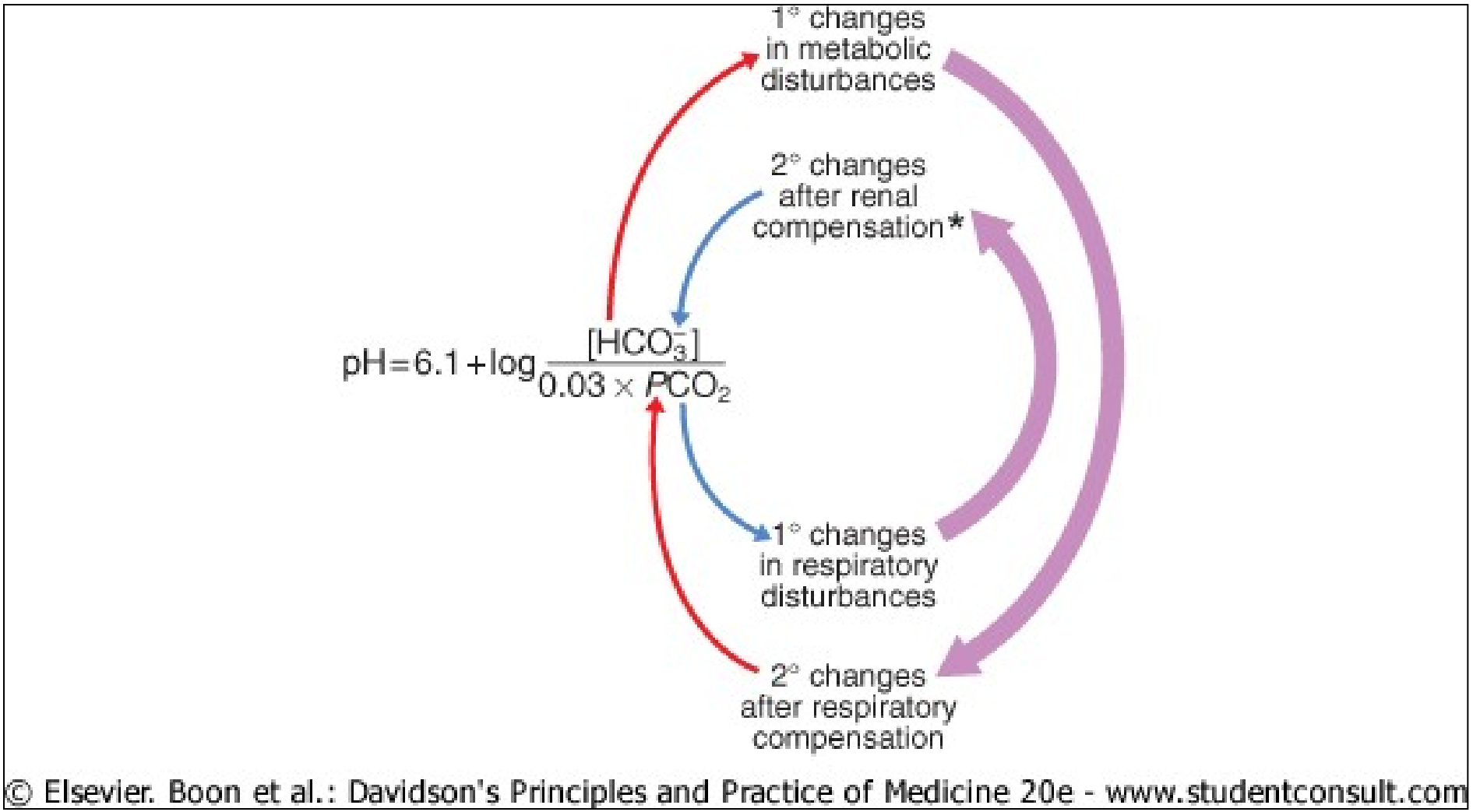


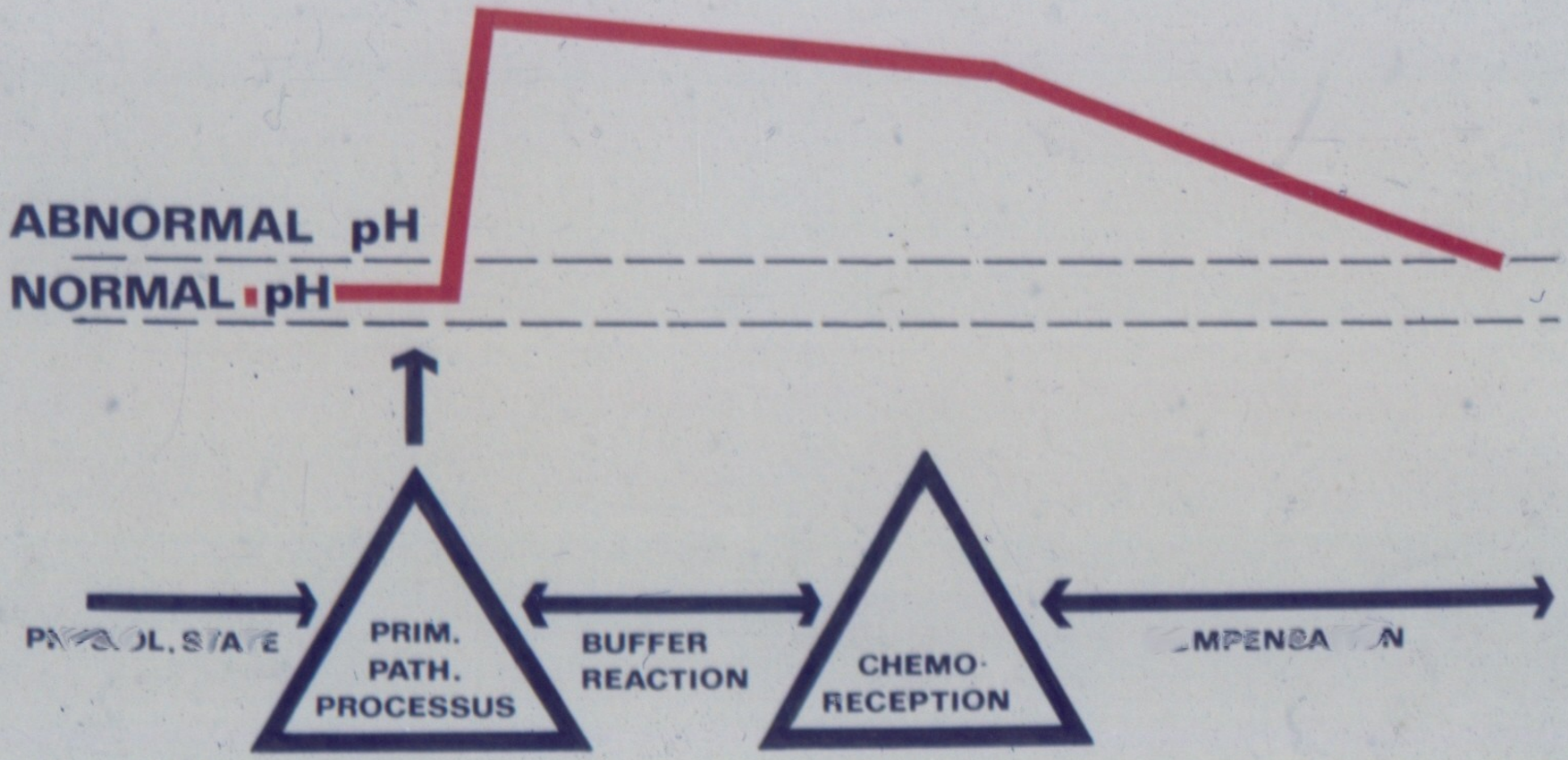


- *production/ingestion of acids*
- *renal failure, renal tubular acidosis*
- *loss of alkali*

Alkalosis

- *Respiratory*
- *Metabolic:* HCO_3^-
- *excess loss of acids*
- *bicarbonate retention*
- *ingestion of alkali*




ABNORMAL pH
NORMAL pH
PRIM.
PATH.
PROCESSUS
BUFFER
REACTION
CHEMO-
RECEPTION
ONCENTRATION OF
BUFFER COMPONENTS
NORMAL
DIFFERENT
DIFFERENT

SYMPTOMS OF ACIDOSIS

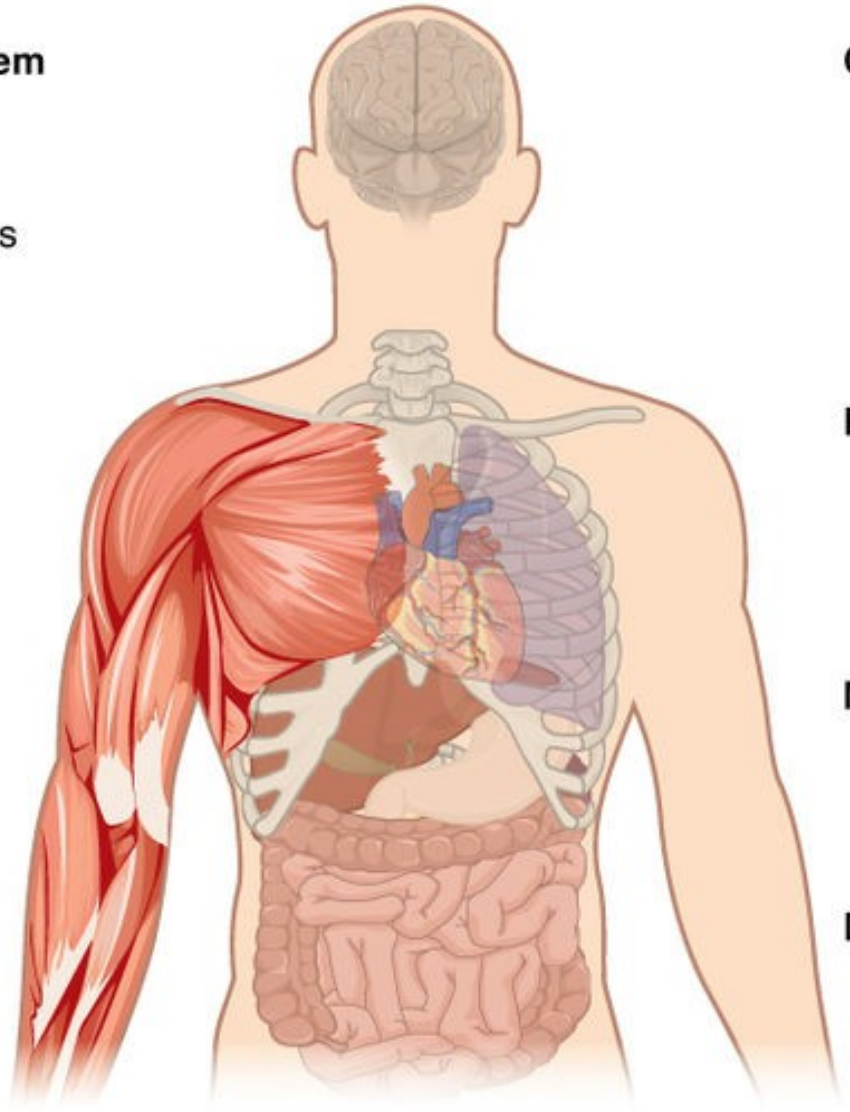
Central Nervous System
- Headache
- Sleepiness
- Confusion
- Loss of consciousness
- Coma

Respiratory System
- Shortness of breath
- Coughing

Heart
- Arrhythmia
- Increased heart rate

Muscular System
- Seizures
- Weakness

Digestive System
- Nausea
- Vomiting
- Diarrhea



SYMPTOMS OF ALKALOSIS

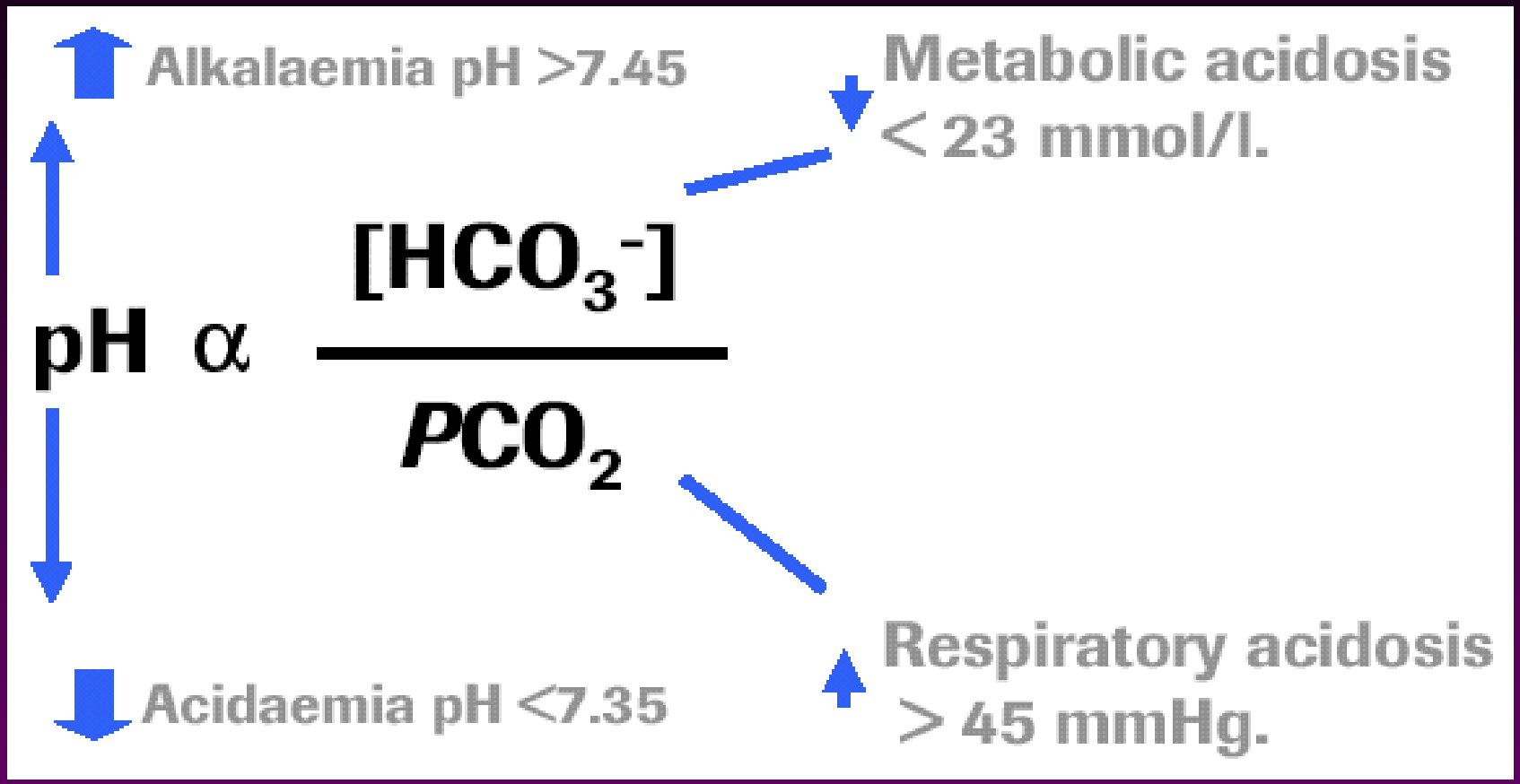
Central Nervous System
- Confusion
- Light-headedness
- Stupor
- Coma

Peripheral Nervous System
- Hand tremor
- Numbness or tingling in the face, hands, or feet

Muscular System
- Twitching
- Prolonged spasms

Digestive System
- Nausea
- Vomiting

Acidosis



Metabolic acidosis – increased H^+, decreased HCO_3^-

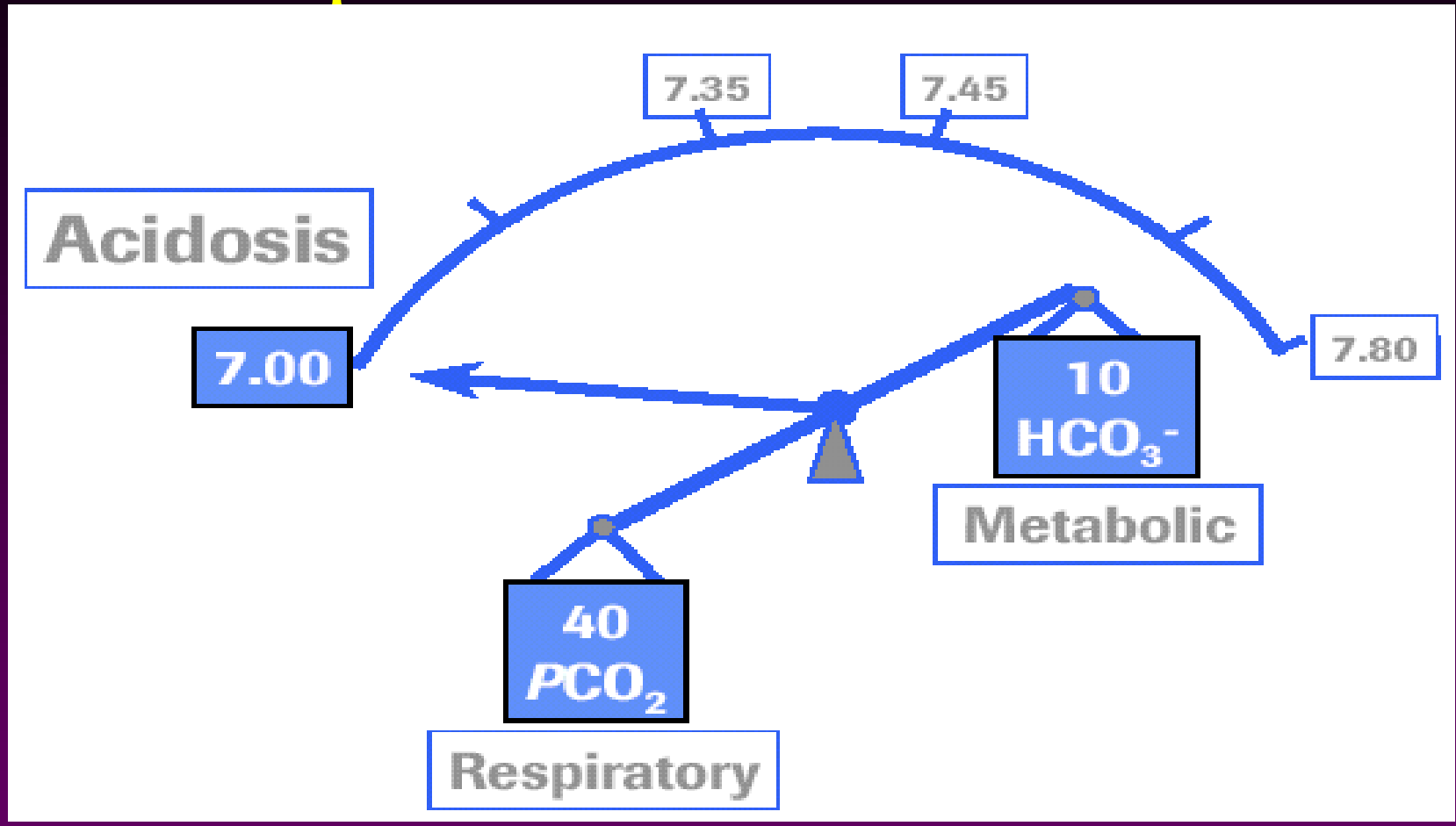
Respiratory acidosis – increased H+, increased $PaCO_2$

Primary Acid-base Disorders: Metabolic Acidosis

- **Metabolic acidosis** - A primary acid-base disorder where the first change is a lowering of HCO_3^-, resulting in decreased pH. Compensation (bringing pH back up toward normal) is a secondary hyperventilation; this lowering of $PaCO_2$ is not respiratory alkalosis since it is not a primary process.

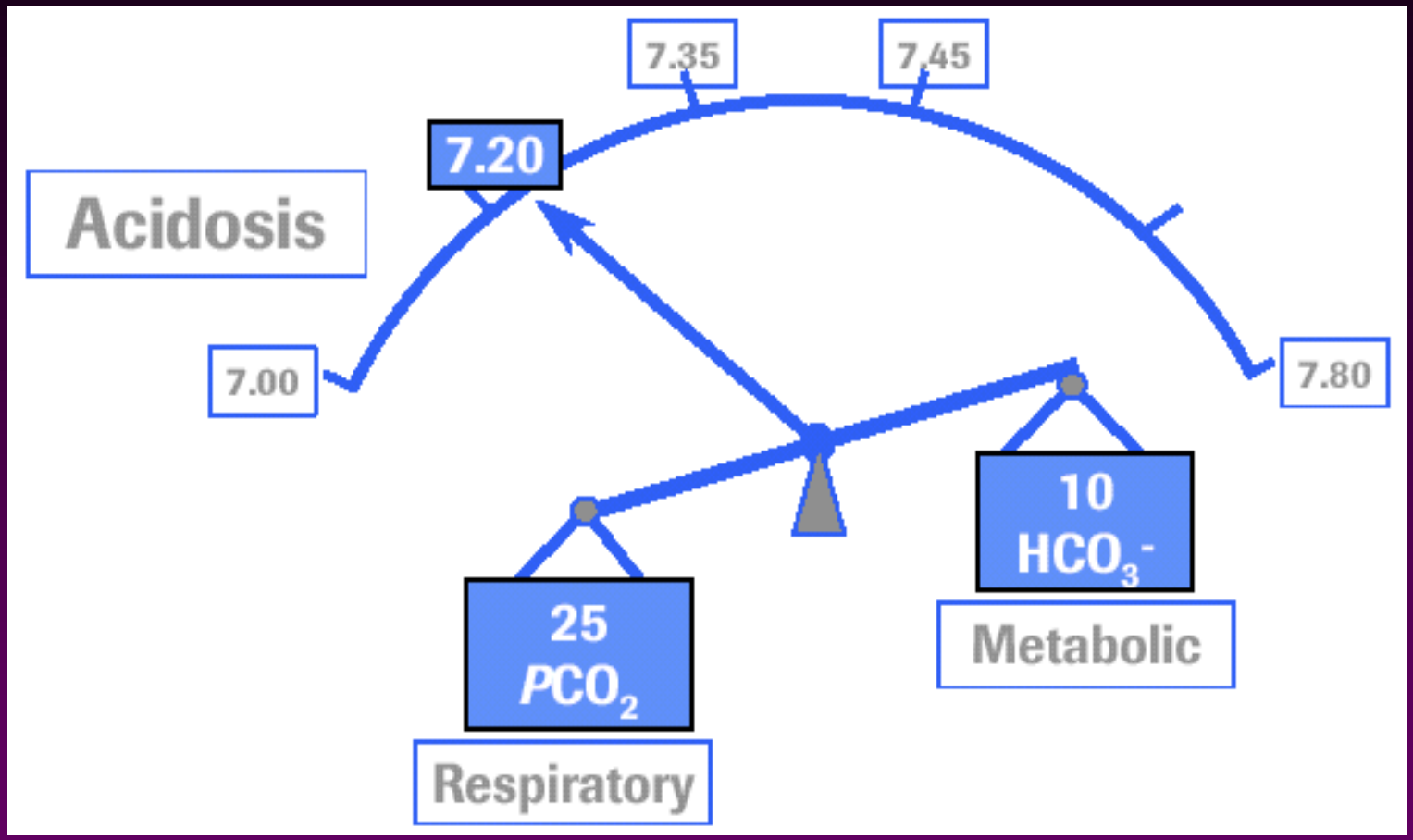
Primary Event	Compensatory Event
$\downarrow HCO_3^-$	$\downarrow HCO_3^-$
$\downarrow$ pH ~ ----------	$\downarrow$ pH ~ ----------
$PaCO_2$	$\downarrow PaCO_2$

Simple metabolic acidosis



Disturbance	Blood PH	Primary change	Compensatory response	Predicted compensation
Metabolic acidosis	< 7,40	HCO_3 < 24 mmo/l	PCO_2 < 5,33 kPa	PCO_2 fall in kPa = 0,16 x HCO_3 fall in mmol/l

Compensation of metabolic acidosis

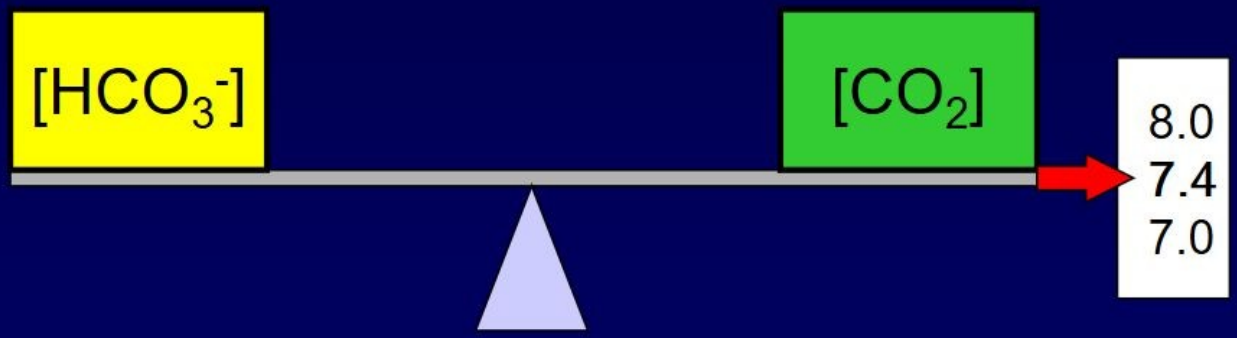


Disturbance	Blood PH	Primary change	Compensatory response	Predicted compensation
Metabolic acidosis	< 7,40	HCO_3 < 24 mmo/l	PCO_2 < 5,33 kPa	PCO_2 fall in kPa = 0,16 x HCO_3 fall in mmol/l

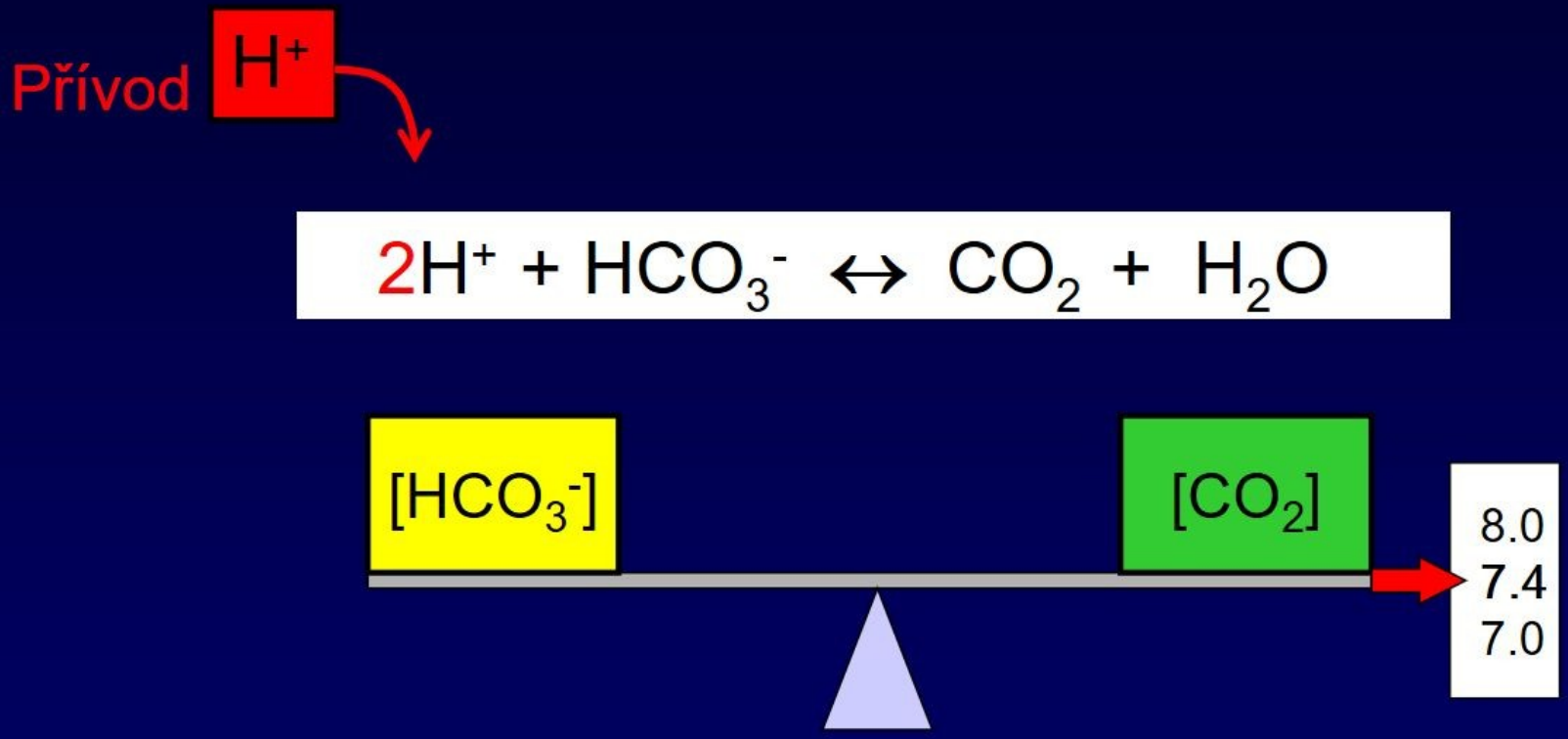
MAC

Přívod H^+

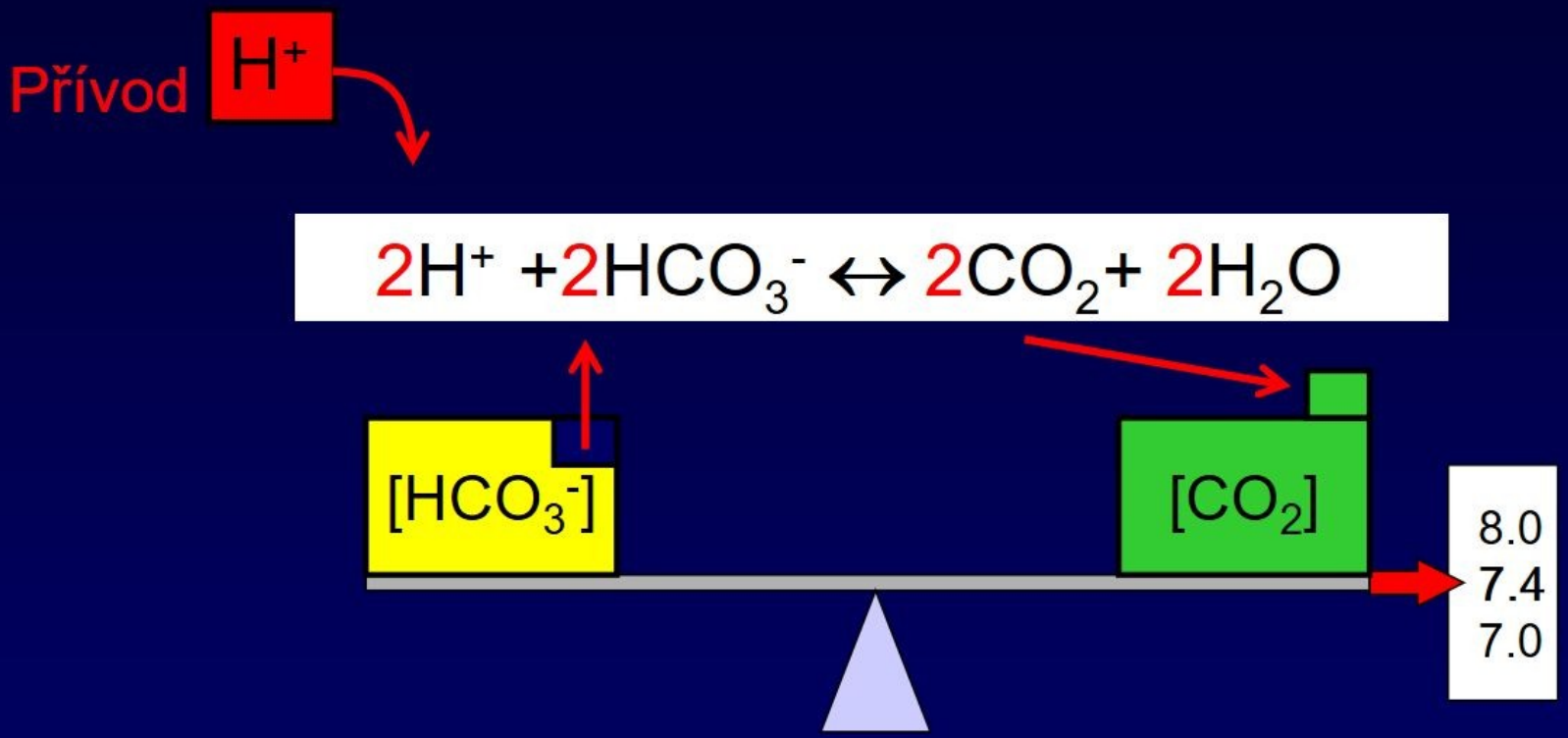
$$H^+ + HCO_3^- \leftrightarrow CO_2 + H_2O$$



MAC



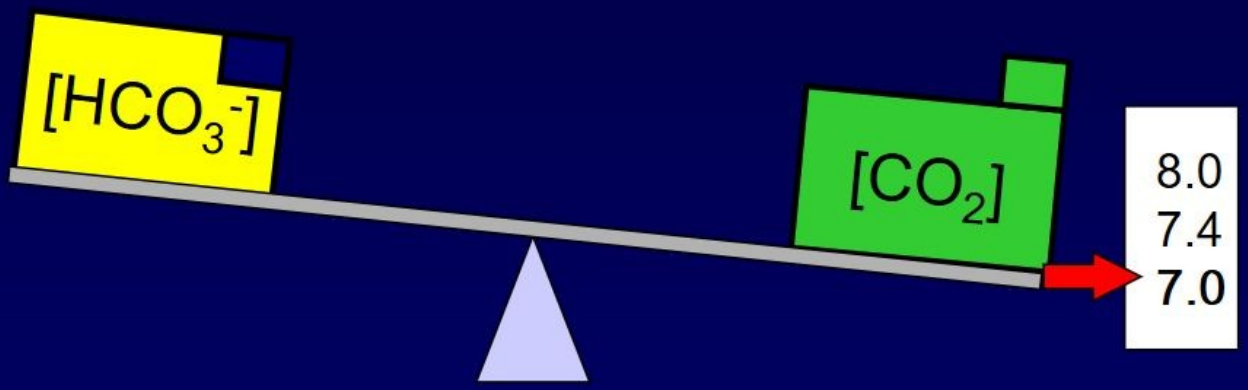
MAC



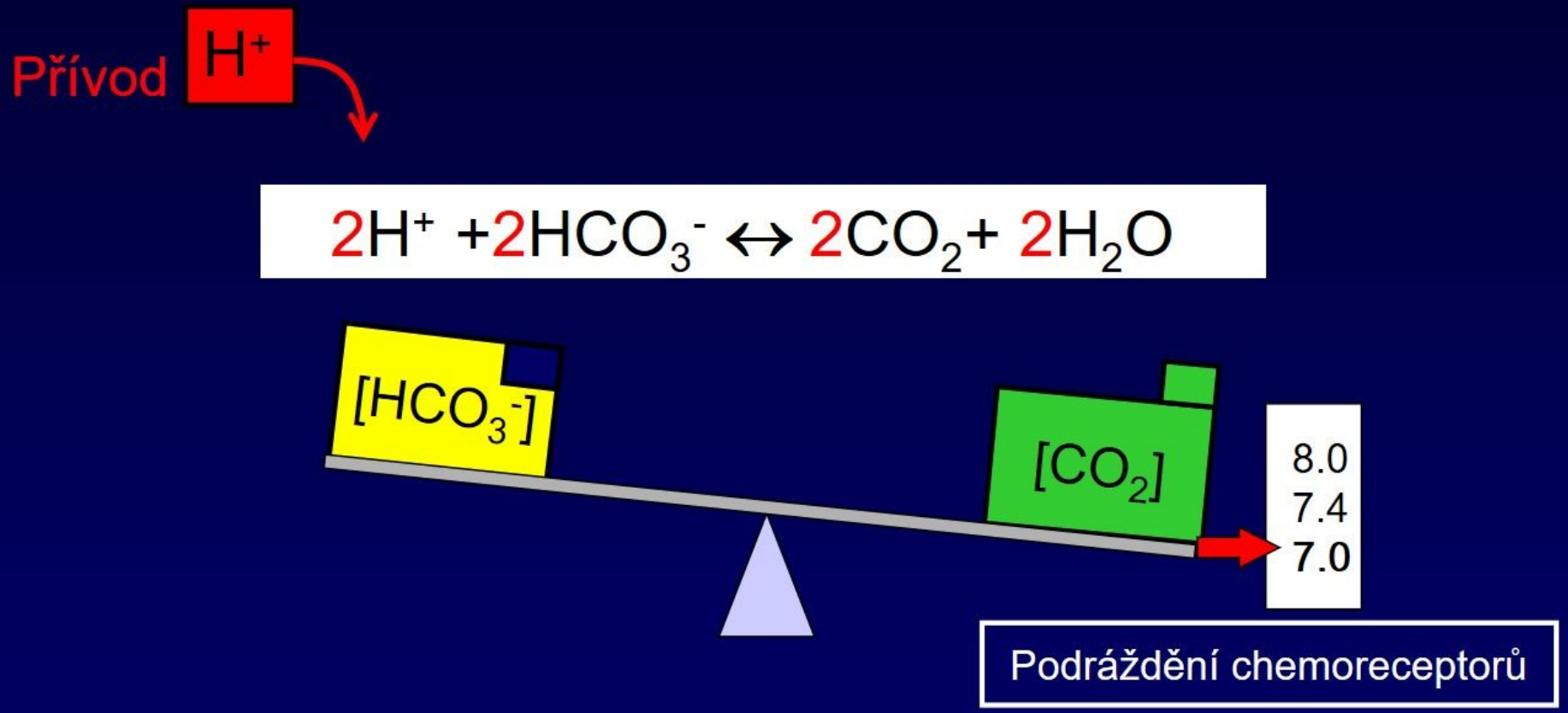
MAC

Přívod H^+

$$2H^+ + 2HCO_3^- \leftrightarrow 2CO_2 + 2H_2O$$



MAC


Přívod
H+
2H+ +2HCO3- ↔ 2CO2+ 2H2O
[HCO3-]
[CO2]
8.0
7.4
7.0
Podráždění chemoreceptorů

MAC

Přívod H^+

Plícemi ven z těla

$$2H^+ + 2HCO_3^- \leftrightarrow 2CO_2 + 2H_2O$$

$[HCO_3^-]$

$[CO_2]$

8.0
7.4
7.0

Podráždění chemoreceptorů

Kompenzace MAC

Přívod H^+

Plícemi ven z těla

$$2H^+ + 2HCO_3^- \leftrightarrow 2CO_2 + 2H_2O$$

$[HCO_3^-]$

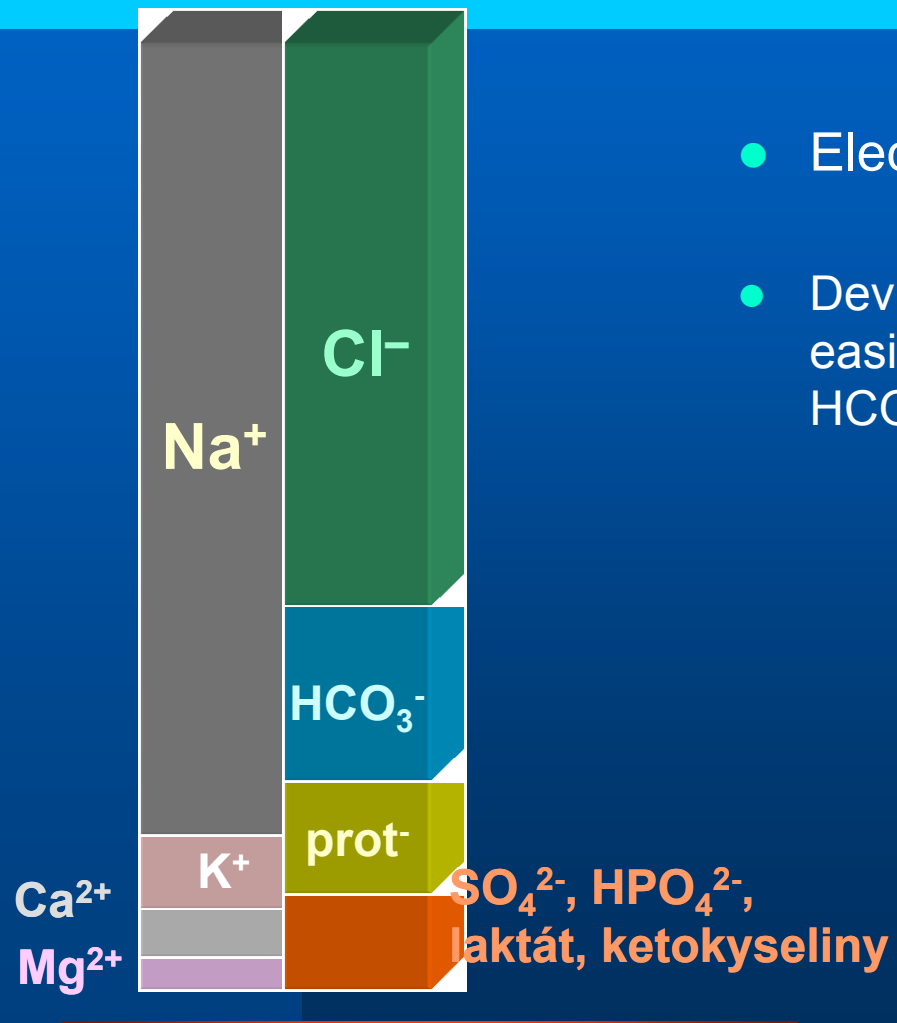
$[CO_2]$

8.0
7.4
7.0

1) Pufr: bikarbonátový

2) Plíce: hyperventilace

ABR and electroneutrality



- Electroneutrality must be maintained
- Deviations in ion concentration are most easily compensated by changing the HCO3-

Anion gap (AG)

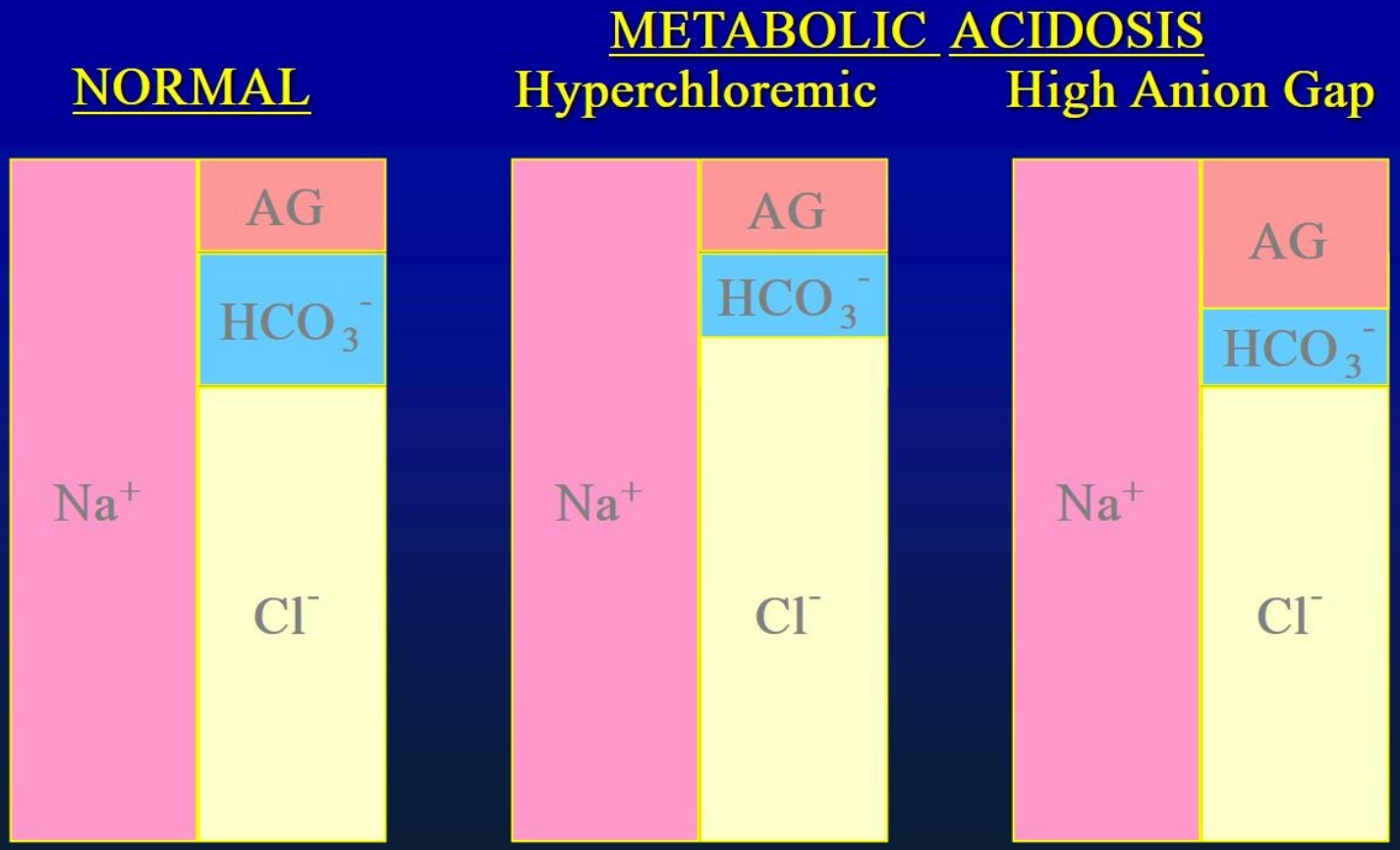
- The difference between main measured cations (Na + K) and the measured anions (Cl + HCO_3)
- Normally 13 to 15 mmol/l
- Unmeasured anions (P^- - albumin, SO_4, PO_3, ORG^-)

Na – (Cl+HCO3) = anion gap

Anion Gap

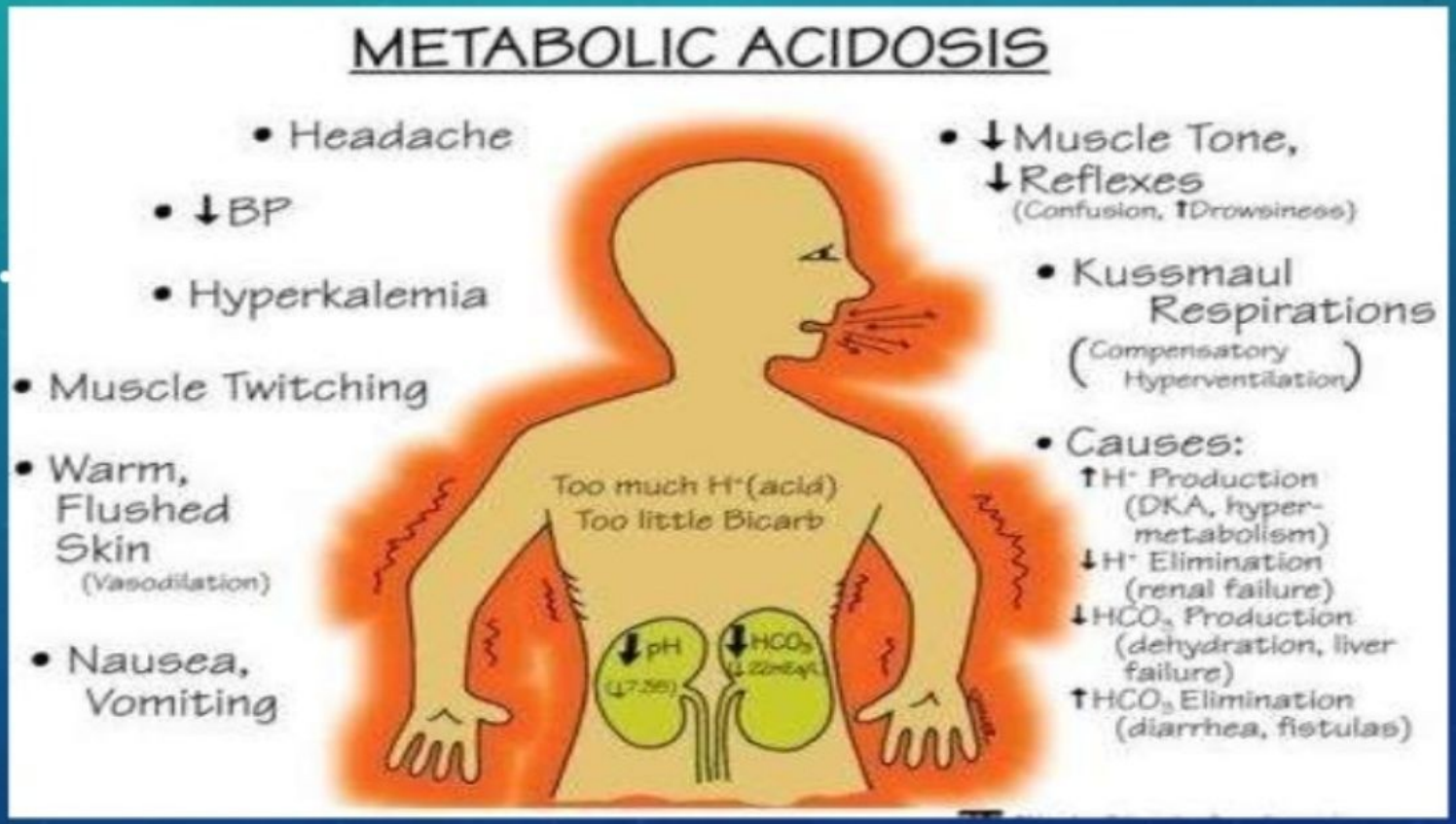
- AG = $[Na^+ + K^+] - [Cl^- + HCO_3^-]$
- Norma: 14 ± 2 mmol/L
- Hlavní „neměřitelné“ anionty, zahrnuté v AG:
  - albumin
  - fosfáty
  - sulfáty
  - organické anionty
- Slouží k posouzení příčin metabolické acidozy

Anion Gap



- High anion gap (normal Cl)
  - Increased unmeasured anions (albumine, inorganic, organic)

SIGNS AND SYMPTOMS



Acid-base disorders

- Metabolic acidosis lead to hyperkalaemia
  - Shift H/K or Na

- Decrease of 0,1 PH increase plasma K by 0,6 mmol/l
  - Diabetic ketoacidosis, lactic acidosis, diarrhea and RTA often associated with low K intracellulary !!

Metabolic acidosis – clinical features

- Increased ventilation (Kussmauls)
- Increased tachycardia
- Decreased cardiac contractility
- Periferal arterial dilatation + central venous contraction → pulmonary edema with minimal overload
- CNS – headache, lethargy, stupor, coma

Causes of metabolic acidosis 1

Disorder	Mechanism
Normal anion gap	
Inorganic acid addition	Therapy or poisoning with NH_4Cl, HCl
Gastrointestinal base loss	Loss of HCO_3 in diarrhoea, small bowel fistula, urinary diversion procedure
Renal tubular acidosis	Urinary loss of HCO_3 in proximal RTA, impaired tubular acid secretion in distal RTA

Causes of renal tubular acidosis

Type	Examples
Proximal RTA (type 2)	Congenital (Fanocon, cystinosis, Wilsons disease) Paraproteinaemia, amyloidosis Heavy metal toxicity (Pb, Cd, Hg) Hyperparathyreosis Carboanhydrase inhibitors (ifosfamide)
Classical distal RTA (type 1)	Congenital, hyperglobulinaemia, Autoimunne connective tissue disease (SLE) Toxins and drugs (toluene, lithium, amphotericin)
Hyperkalemic distal RTA (type 4)	Hypoaldosteronism, Obstructive nephropathy Drugs (amiloride, spironolactone) Renal transplant rejection

Acid-base disorders

Disturbance	Blood PH	Primary change	Compensatory response	Predicted compensation
Metabolic acidosis	< 7,40	HCO_3 < 24 mmo/l	PCO_2 < 5,33 kPa	PCO_2 fall in kPa = 0,16 x HCO_3 fall in mmol/l
Metabolic alkalosis	> 7,40	HCO_3 > 24 mmol/l	PCO_2 > 5,33 kPa	PCO_2 rise in kPa = 0,08 x HCO_3 rise in mmol/l
Respiratory acidosis	< 7,40	PCO_2 > 5,33 kPa	HCO_3 > 24 mmol/l	Acute: HCO_3 rise in mmol/l = 0,75 x PCO_2 rise in kPa Chronic: HCO_3 rise in mmol/l = 2,62 x PCO_2 rise in kPa
Respiratory alkalosis	> 7,40	PCO_2 < 5,33 kPa	HCO_3 < 24 mmo/l	Acute: HCO_3 fall in mmol/l = 1,50 x PCO_2 fall in kPa Chronic: HCO_3 fall in mmol/l = 3,75 x PCO_2 fall in kPa

PH of 7,4 = H^+ of 40 nmol/l

PCO_2 of 5,33 kPa = 40 mmHg, PCO_2 does not rise above 7,33 kPa (55 mmHg), not adequate oxygenation

Primary Acid-base Disorders: Respiratory Acidosis

- **Respiratory acidosis** - A primary disorder where the first change is an elevation of $PaCO_2$, resulting in decreased pH. Compensation (bringing pH back up toward normal) is a secondary retention of bicarbonate by the kidneys; this elevation of HCO_3^- is not metabolic alkalosis since it is not a primary process.

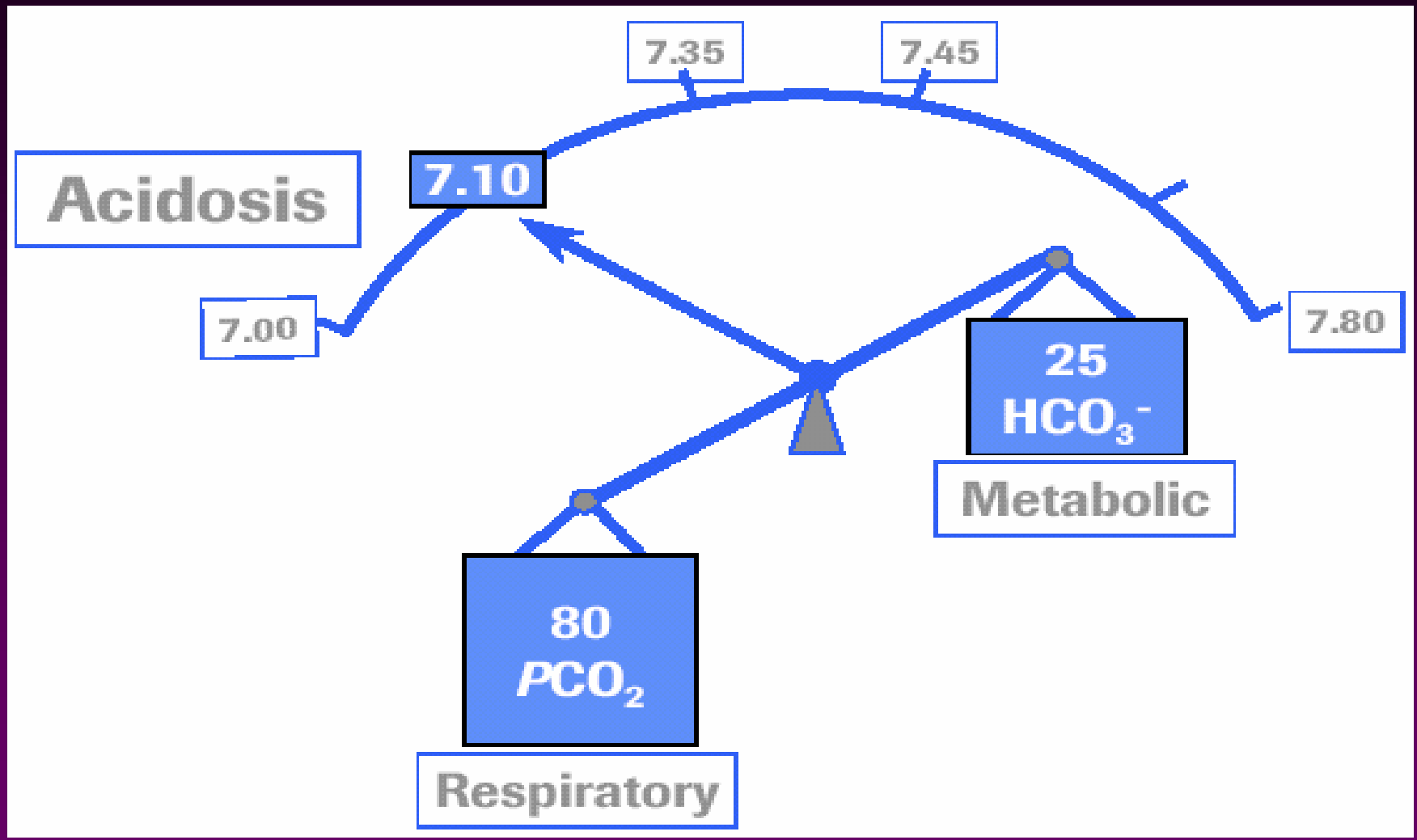
Primary Event	Compensatory Event
$\downarrow$ pH ~ $\frac{HCO_3^-}{\uparrow PaCO_2}$	$\downarrow$ pH ~ $\frac{\uparrow HCO_3^-}{\uparrow PaCO_2}$

Respirační acidóza (RA)

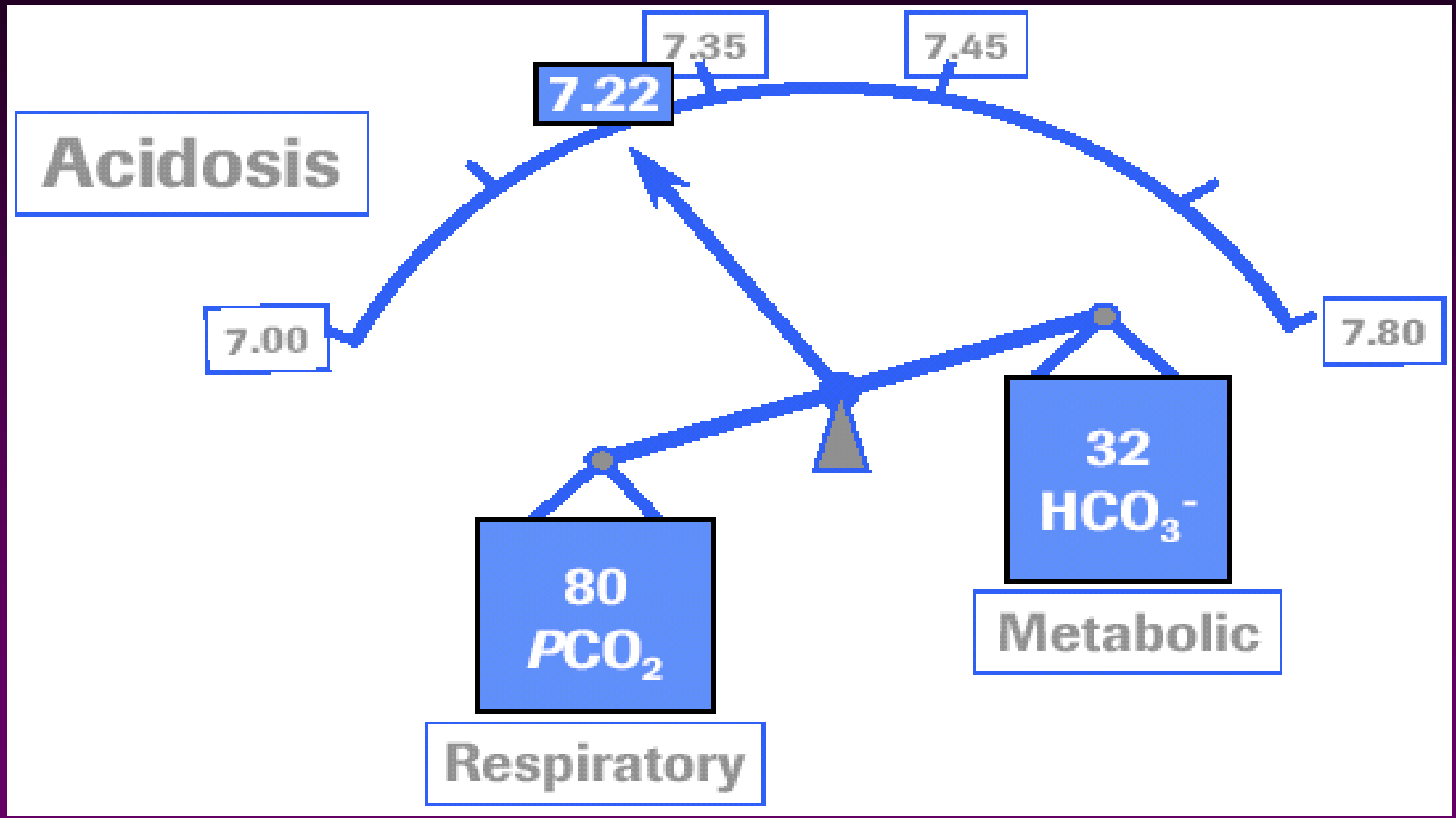
- primární změnou je ↓pH v důsledku ↑$PaCO_2$ (>40 mmHg), tj. hyperkapnie
  - akutní (↓pH)
  - chronická (↓pH nebo normální pH)
    - renální kompenzace – retence HCO_3^-, 3-4 dny
- příčiny:
  - pokles alveolární ventilace
  - (zvýš. koncentrace CO_2 ve vdechovaném vzduchu)
  - (zvýšená produkce CO_2)

$paCO_2 = VCO_2 / VA$

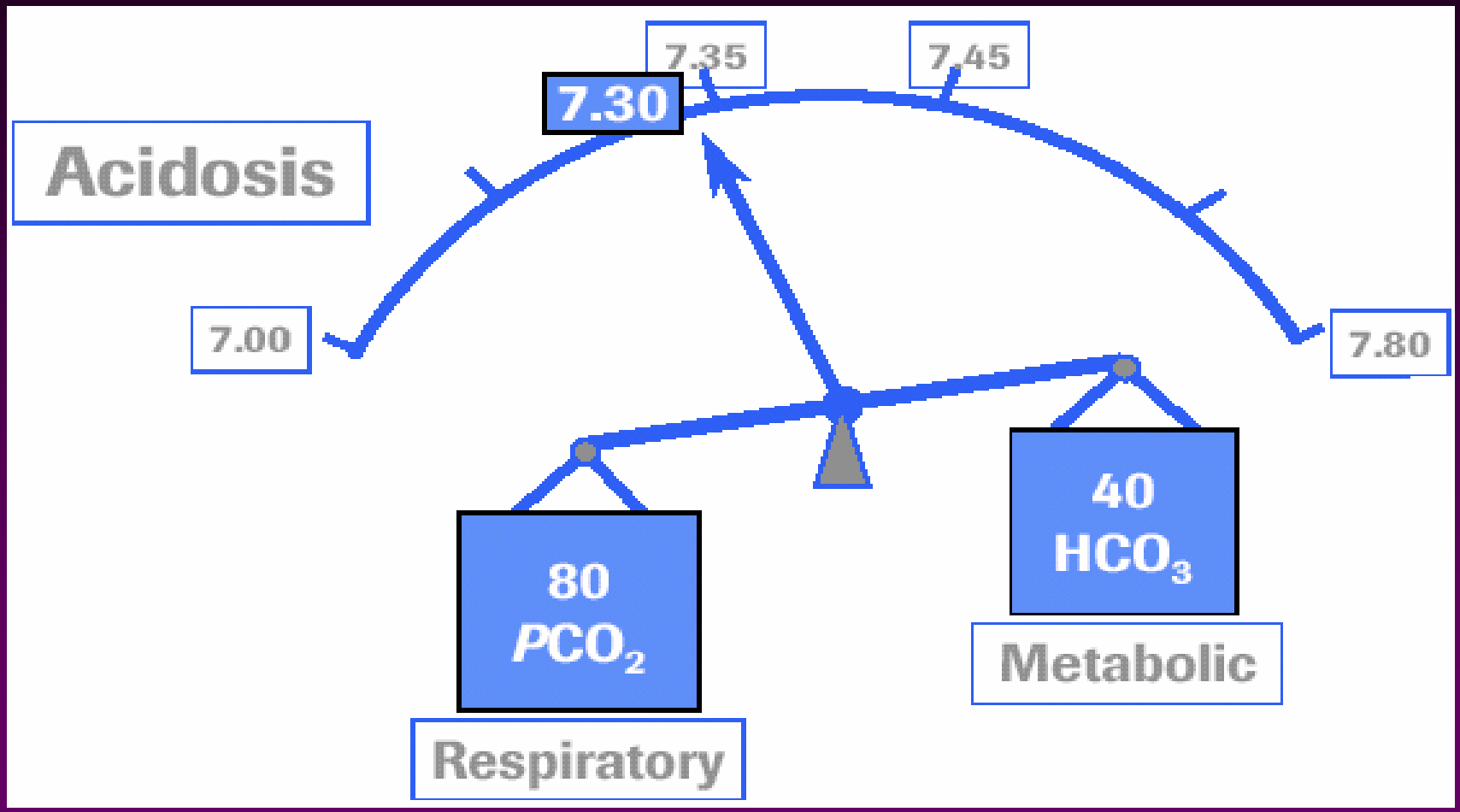
Simple respiratory acidosis



Acute compensation of respiratory acidosis



Chronic compensation of respiratory acidosis



Causes of respiratory acidosis

- Severe pulmonary disease
- Respiratory muscle fatigue
- Increase of $PaCO_2$ (central respiratory control)
- Renal compensation by reabsorbtion of bicarbonate (about 3 days)

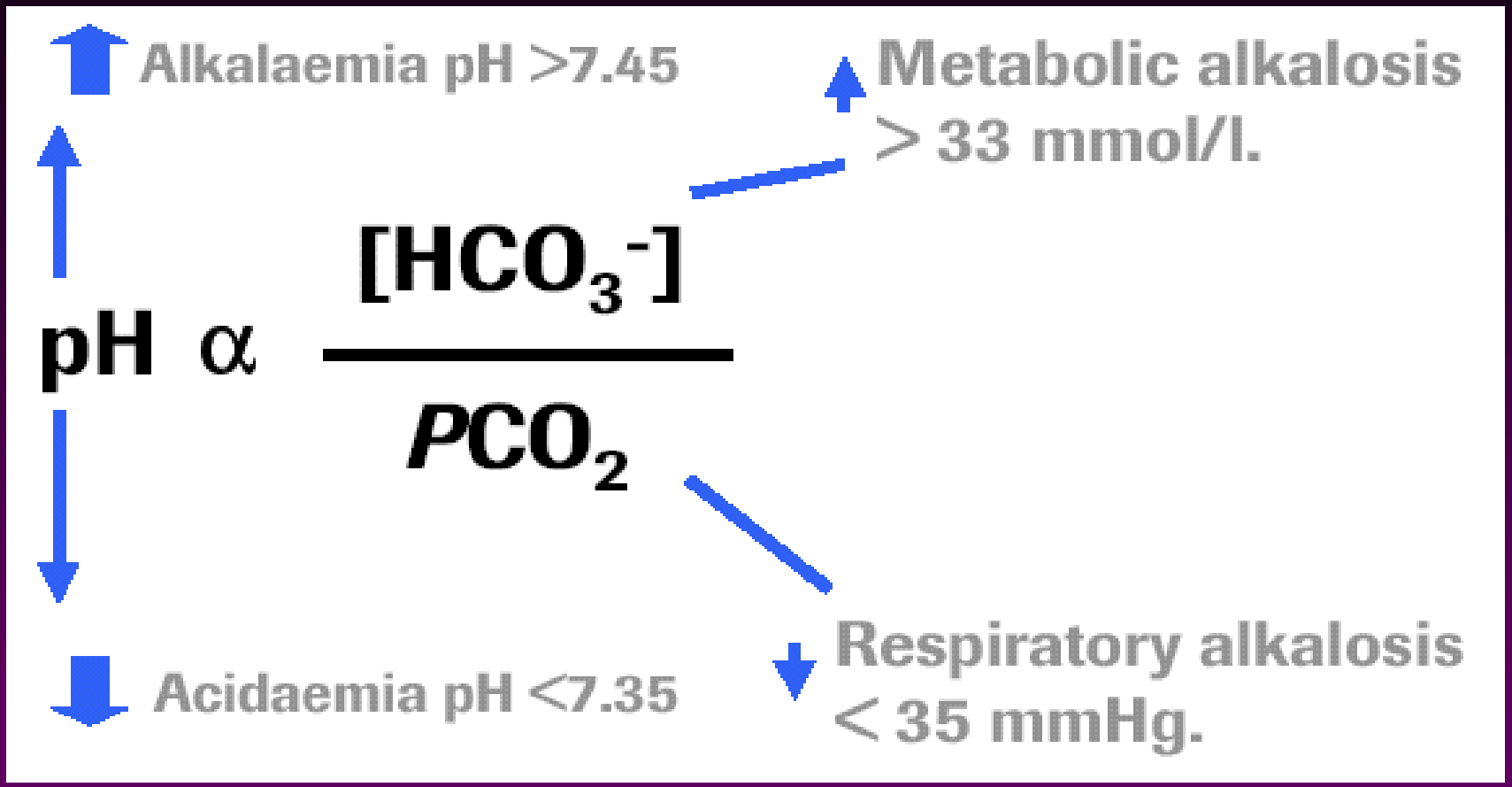
Respiratory acidosis clinical features

- Acute
  - Anxiety, dyspnoea, halucination, coma
- Chronic
  - Sleep disturbances, sleep inversion somnolence, loss of memory, tremor, myoclonics jerks, asterixis

Respiratory acidosis treatment

- Acute
  - Adequate alveolar ventilation
  - Intubation, mechanical ventilation
  - Oxygen administration titrate carefully in CHOPD
  - No rapid correction (respiratory alcalosis symptoms)
  - Sufficient Cl and K to enhance renal excretion of bicarbonate
- Chronic
  - Improve lung function (bronchodilatans, glucocorticoids, diuretics)

Alkalosis



Metabolic alkalosis – decreased H^+, increased HCO_3^-

Respiratory alkalosis – decreased H+, decreased $PaCO_2$

Primary Acid-base Disorders: Metabolic Alkalosis

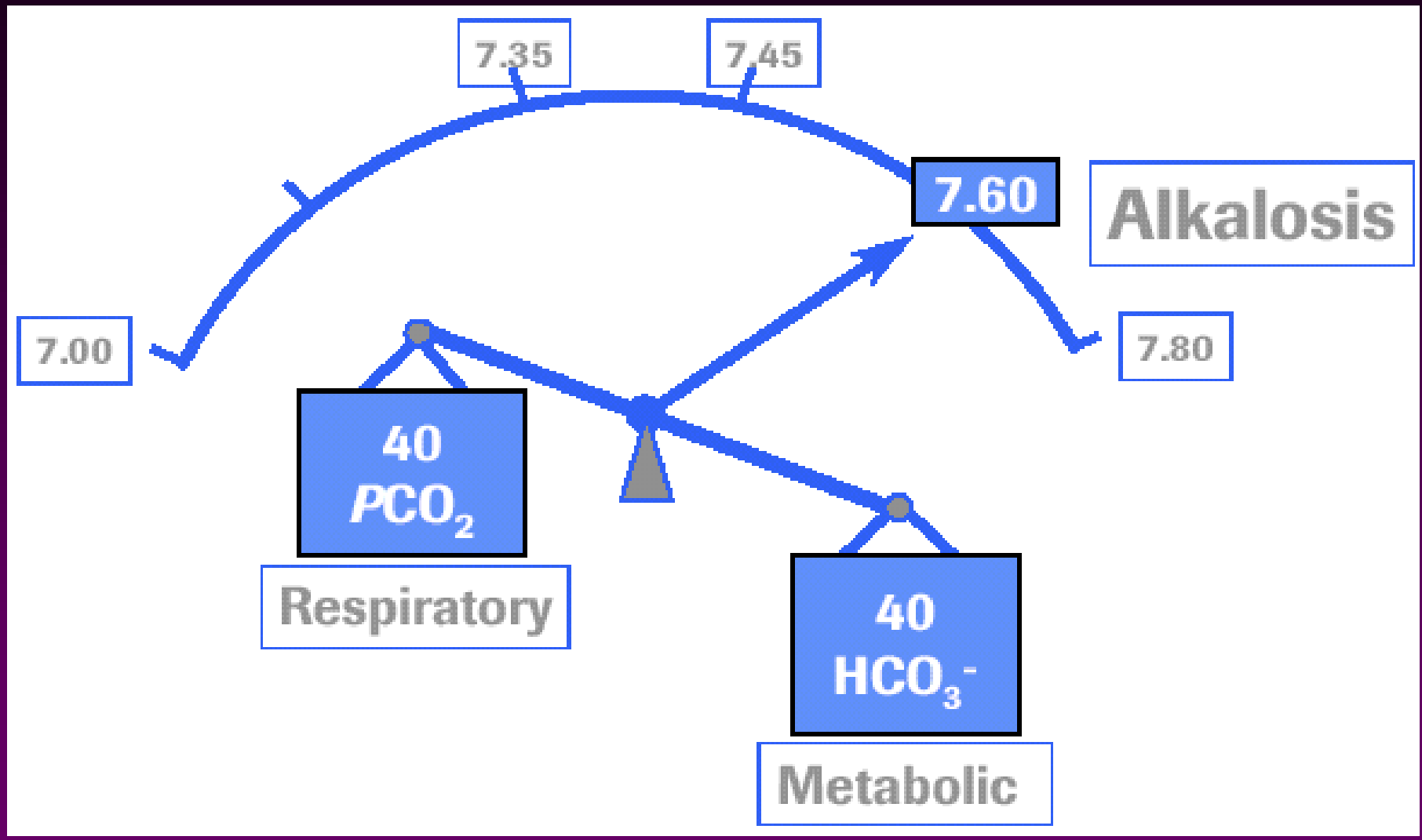
- **Metabolic alkalosis** - A primary acid-base disorder where the first change is an elevation of HCO_3^-, resulting in increased pH. Compensation is a secondary hypoventilation (increased $PaCO_2$), which is not respiratory acidosis since it is not a primary process. Compensation for metabolic alkalosis (attempting to bring pH back down toward normal) is less predictable than for the other three acid-base disorders.

	Primary Event	Compensatory Event
	$\uparrow HCO_3^-$	$\uparrow HCO_3^-$
$\uparrow$ pH ~	---	---
	$PaCO_2$	$\uparrow PaCO_2$

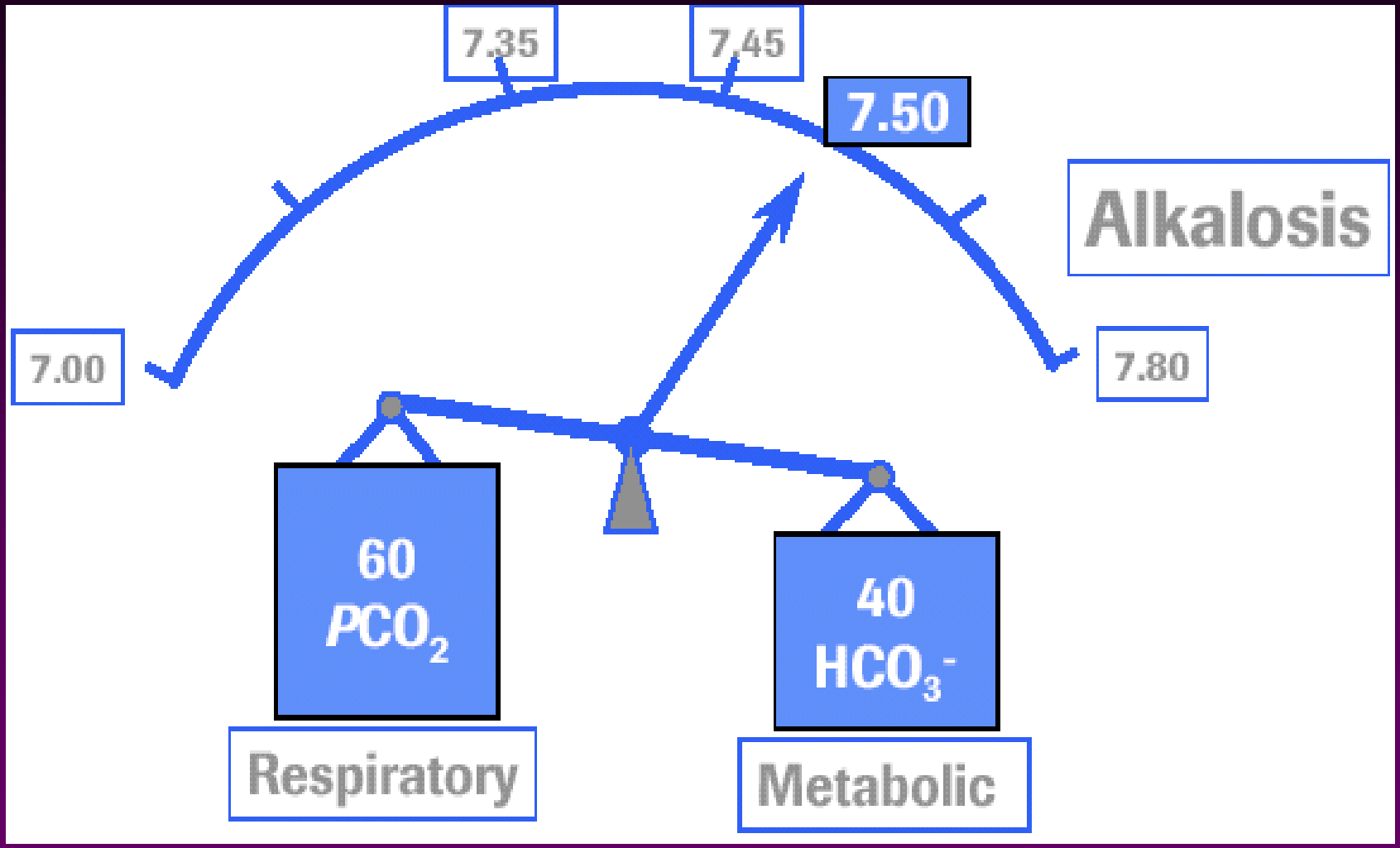
Causes of respiratory alcalosis

- Hyperventilation
- Critically ill patients
- Mechanical ventilation
- 2 to 6 hour hypocapnia → decrease of renal amonium and titrable acid excretion
- Full adaptation take several days
- Many causes and diseases
  - Drugs (salycilates, methylxantines) direct stimulation of respiration
  - Progesterone (gravidity)
  - Liver failure
  - Gramnegative septikaemia before fever with hypotension and hypoxemia

Simple metabolic alkalosis



Compensation of metabolic alkalosis

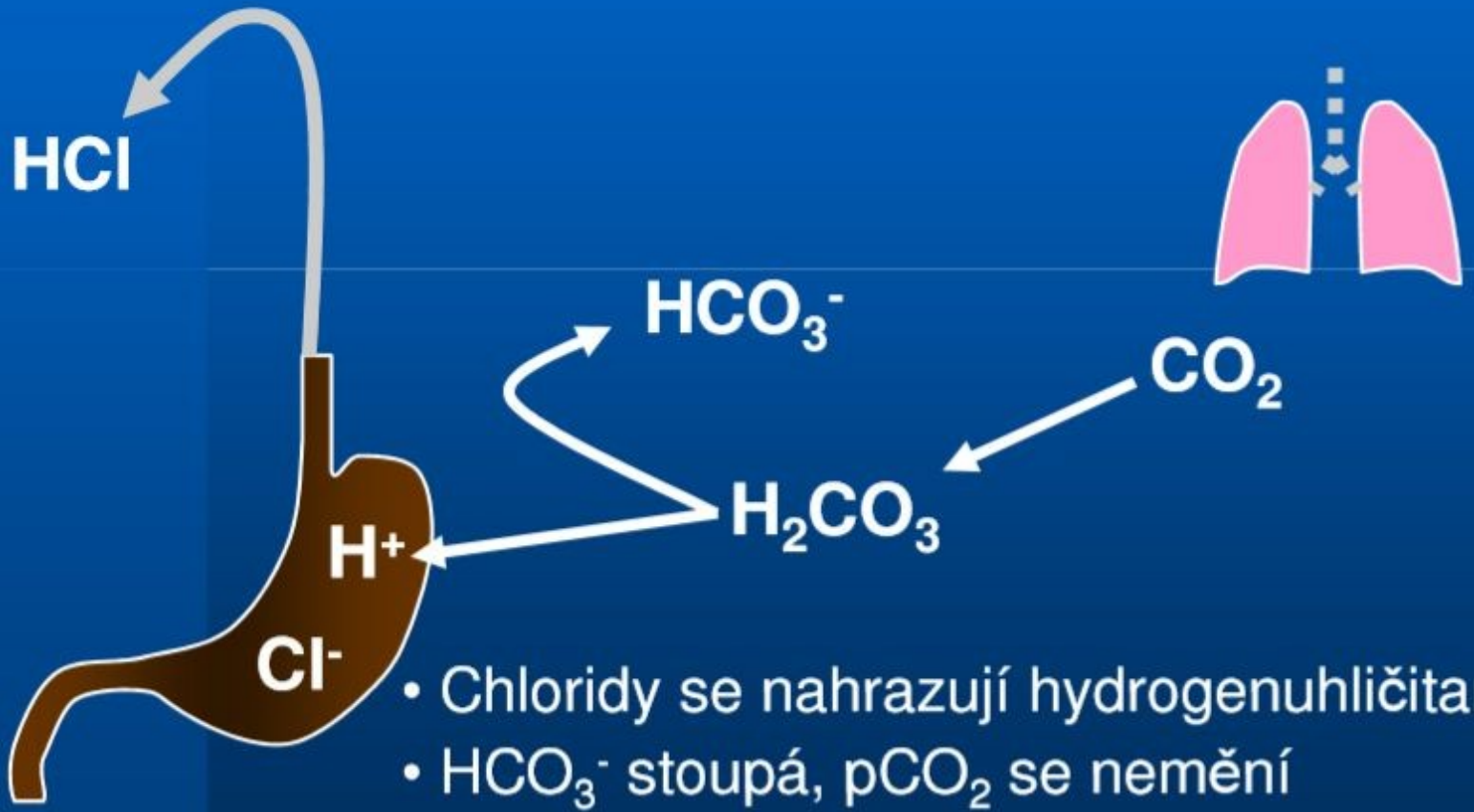


Causes of metabolic alkalosis 1

- Exogenous bicarbonate load
  - Milk alkali syndrome
  - Acute alkali administration

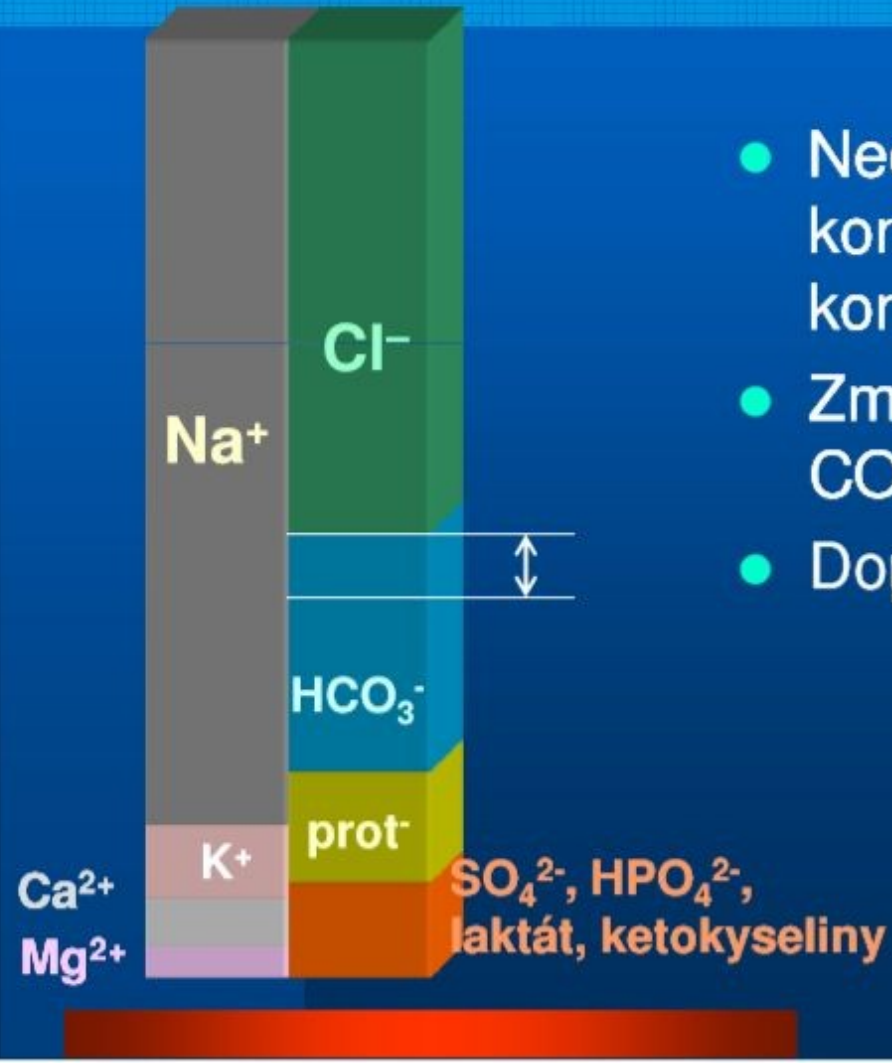
- Effective ECFV contraction, normotension, K deficiency and secondary hyperreninemic hyperaldosteronism
  - Gastrointestinal origin
    - Vomiting, gastric aspiration
    - Congenital chloridorhea
    - Villous adenoma
  - Renal origin
    - Diuretic, edematous states, posthypercapnic state, recovery of acidosis
    - Hypercalcemia/hypoparthyreoidism, Mg, K deficiency
    - Barttters syndrome
  - Gitemans syndrome

Hypochloremická alkalóza



- Chloridy se nahrazují hydrogenuhličitany
- HCO_3^- stoupá, pCO_2 se nemění

Hypochloremická alkalóza



- Nedostatek Cl^- je kompenzován zvýšením koncentrace HCO_3^-
- Změna poměru bikarbonátu a CO_2 vede k alkalóze
- Doprovází např. zvracení

Causes of metabolic alkalosis 1

- ECFV expansion, low K, hypertension
  - High renin
    - Renal artery stenosis, accelerated hypertension
    - Renin secreting tumors, estrogen therapy
  - Low renin
    - Primary aldosteronism
      - Adenoma, primary hyperplasia, carcinoma
    - Adrenal enzyme defects
      - 11 beta hydrochylase deficiency
      - 17 alfa hydroxylase deficiency
    - Cushings syndrome
      - Ectopic corticotropin
      - Adrenal adenoma
      - Primary pituitary
    - Other
      - Licorice, carbenoxolone, chewers tobaco
      - Liddles syndrome

Metabolic alkalosis clinical features

- Changes in central and peripheral nervous system
  - Similar to those hypocalcemia
  - Mental confusion, obtundation, seizures
  - Paresthesia, muscular cramping, tetany, aggravation of arrythmias
- Hypoxemia in chronic obstructive pulmonary disease
- Hypokalemia and hypophosphatemia

Metabolic alkalosis treatment

- Correcting the underlying stimulus for generating bicarbonate
  - Discontinuation of diuretics
  - H_2 blockers or proton pump blockers
- Remove the factors that sustain bicarbonate reabsorption
  - ECF contraction
  - K deficiency

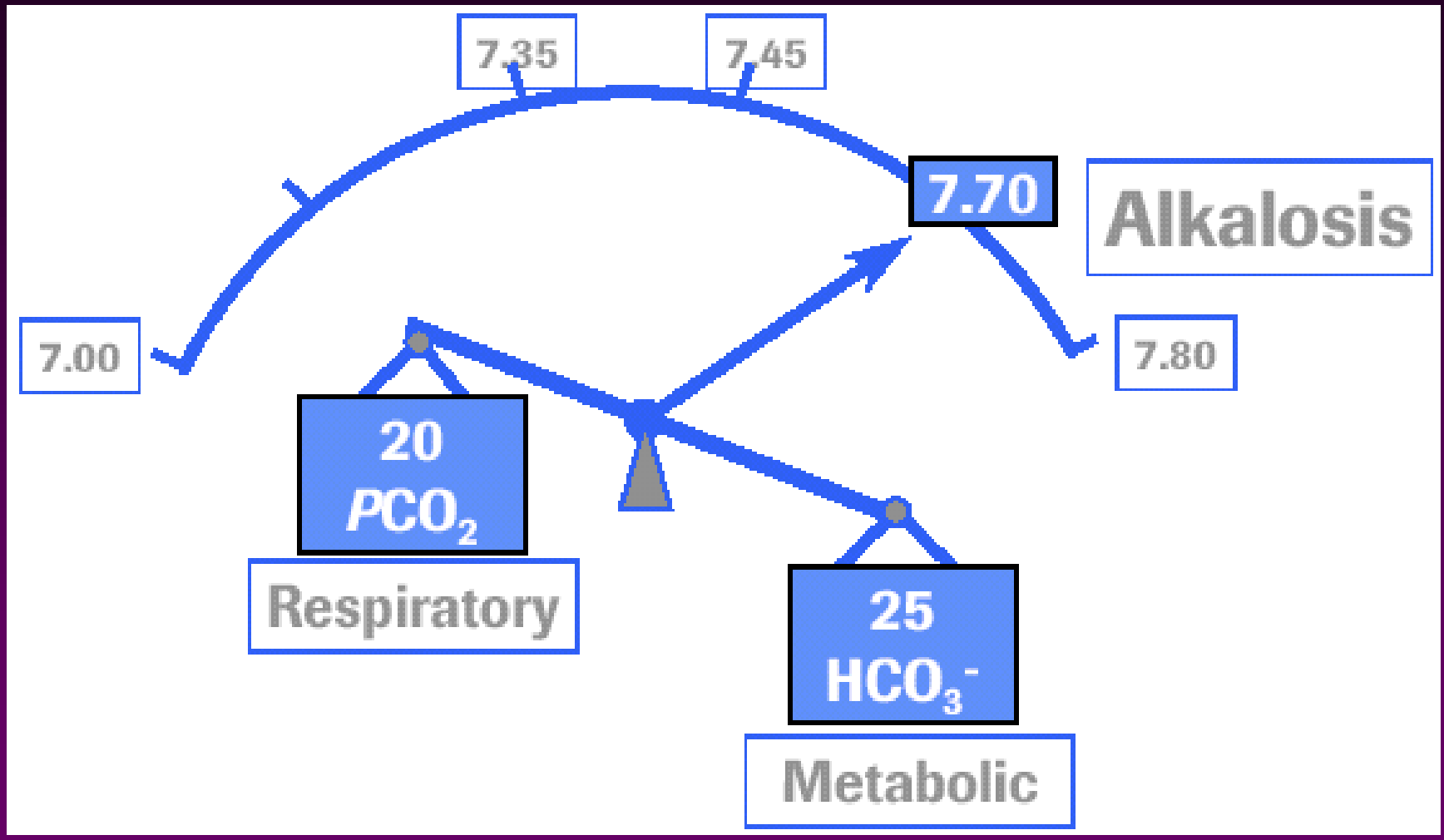
- ECF expansion (NaCl, KCl)
- Acetazolamide
- Arginin hydrochloride
- NH_4Cl oraly
- Hemodialysis

Primary Acid-base Disorders: Respiratory Alkalosis

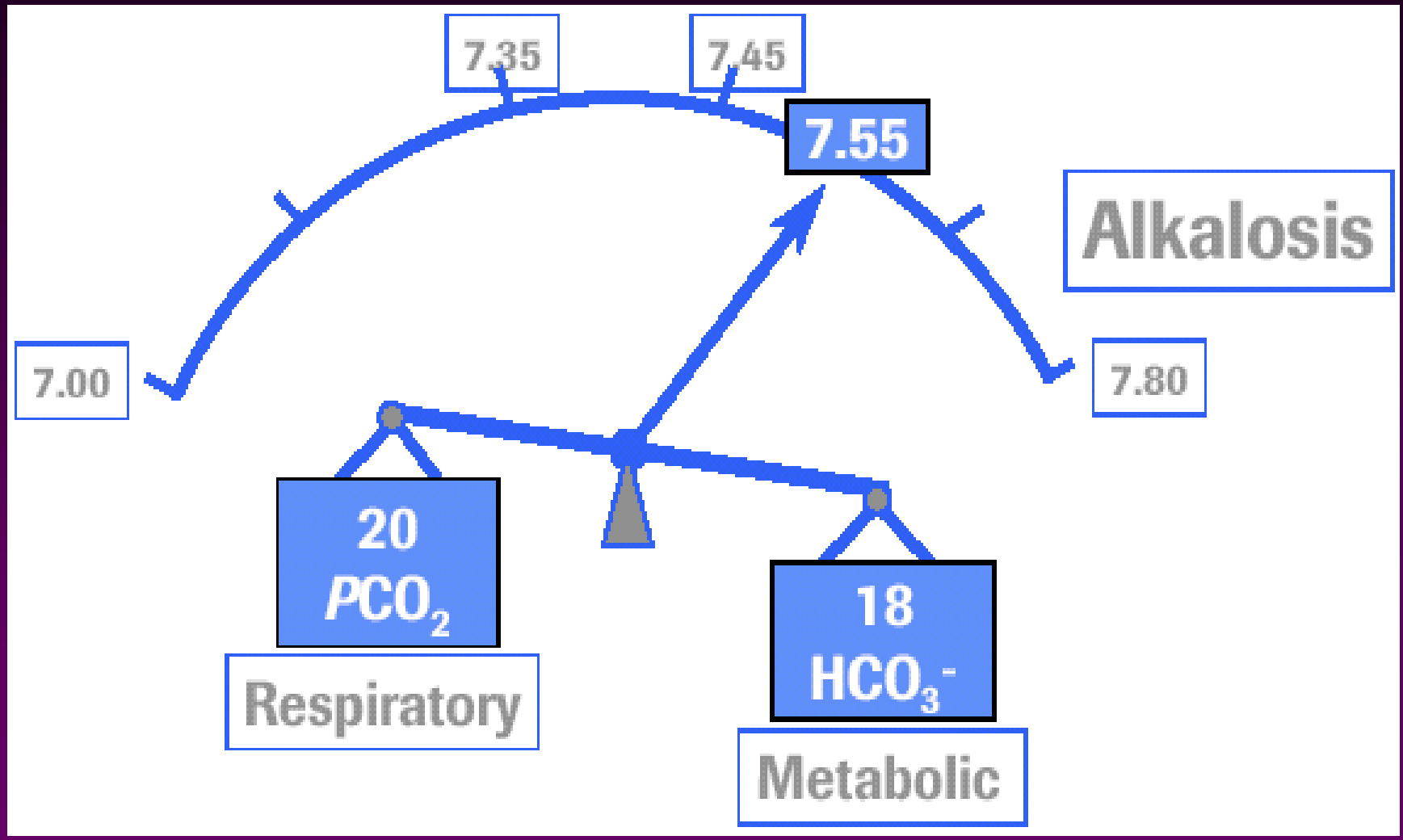
- **Respiratory alkalosis** - A primary disorder where the first change is a lowering of $PaCO_2$, resulting in an elevated pH. Compensation (bringing the pH back down toward normal) is a secondary lowering of bicarbonate (HCO_3) by the kidneys; this reduction in HCO_3^- is not metabolic acidosis, since it is not a primary process.

Primary Event	Compensatory Event
HCO_3^-	$\downarrow HCO_3^-$
$\uparrow$ pH ~ -------	$\uparrow$ pH ~ -------
$\downarrow PaCO_2$	$\downarrow PaCO_2$

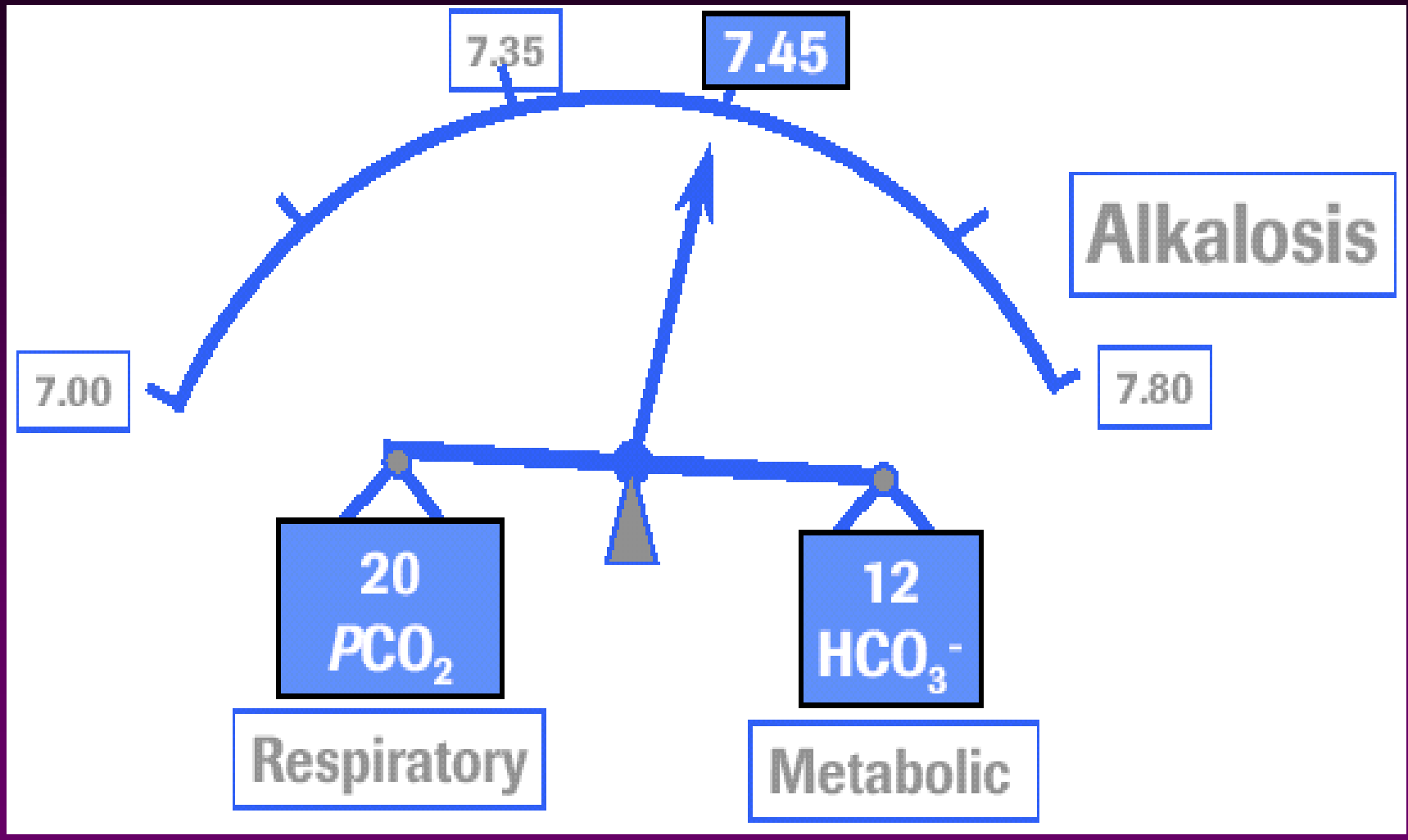
Simple respiratory alkalosis



Acute compensation of respiratory alkalosis



Chronic compensation of respiratory alkalosis



Respiratory alkalosis clinical features

- Reduced cerebral blood flow
  - Dizziness, mental confusion, seizures
- Cardiac arrhythmias
  - Intracellular shift of Na, K
  - Decreased Ca^{2+}
- Paresthesia, circumoral numbness, tetany

Respiratory alkalosis treatment

- Ventilator management (dead space, tidal volume and frequency)
- Rebreathing from paper bag
- Betablockers in hyperadrenergic states

STEP-BY-STEP ANALYSIS OF ACID-BASE STATUS

1. Look at the pO_2 (<80 mm Hg) and O_2 *saturation* (<90%) for *hypoxemia*

2. Look at the *pH*

- < 7.35 : ACIDOSIS
- > 7.45 : ALKALOSIS
- 7.35 – 7.45 : normal/mixed disorder

3. Look at pCO_2

- > 45 mm Hg : Increased (Acidic)
- < 35 mm Hg : Decreased (Alkalotic)

4. Look at the HCO_3^-

- > 26 mEq/L : Increased (Alkalotic)
- < 22 mEq/L : Decreased (Acidic)

5. Determine the acid-base disorder, match either the pCO_2 or the HCO_3^- with the pH

6. ***Compensation***… are the CO_2 or HCO_3^- of opposite type ?

7. Calculate the *anion gap* if it is more there is Metabolic acidosis

$$AG = [Na^+] - [Cl^- + HCO_3^-]$$

Na (140) + K (5) = Cl (105) – HCO3 –(25) + Gap (15)

If Gap > 30 – clinically important acidosis

Treat the patient not the ABG!!!

Thank you...

Summary

- Individualize patient and correcting underlying stimulus
- ECF volume clinical examination (contraction or edema)
- Laboratory plasma
  - Na, K, Cl, Ca, Mg,
  - Proteins and albumin, urea, kreatinin, AST,ALT
  - Bicarbonate
  - $PaCO_2$
- Laboratory urine
  - Na, K, Cl, free water, Ca
  - Poisoning