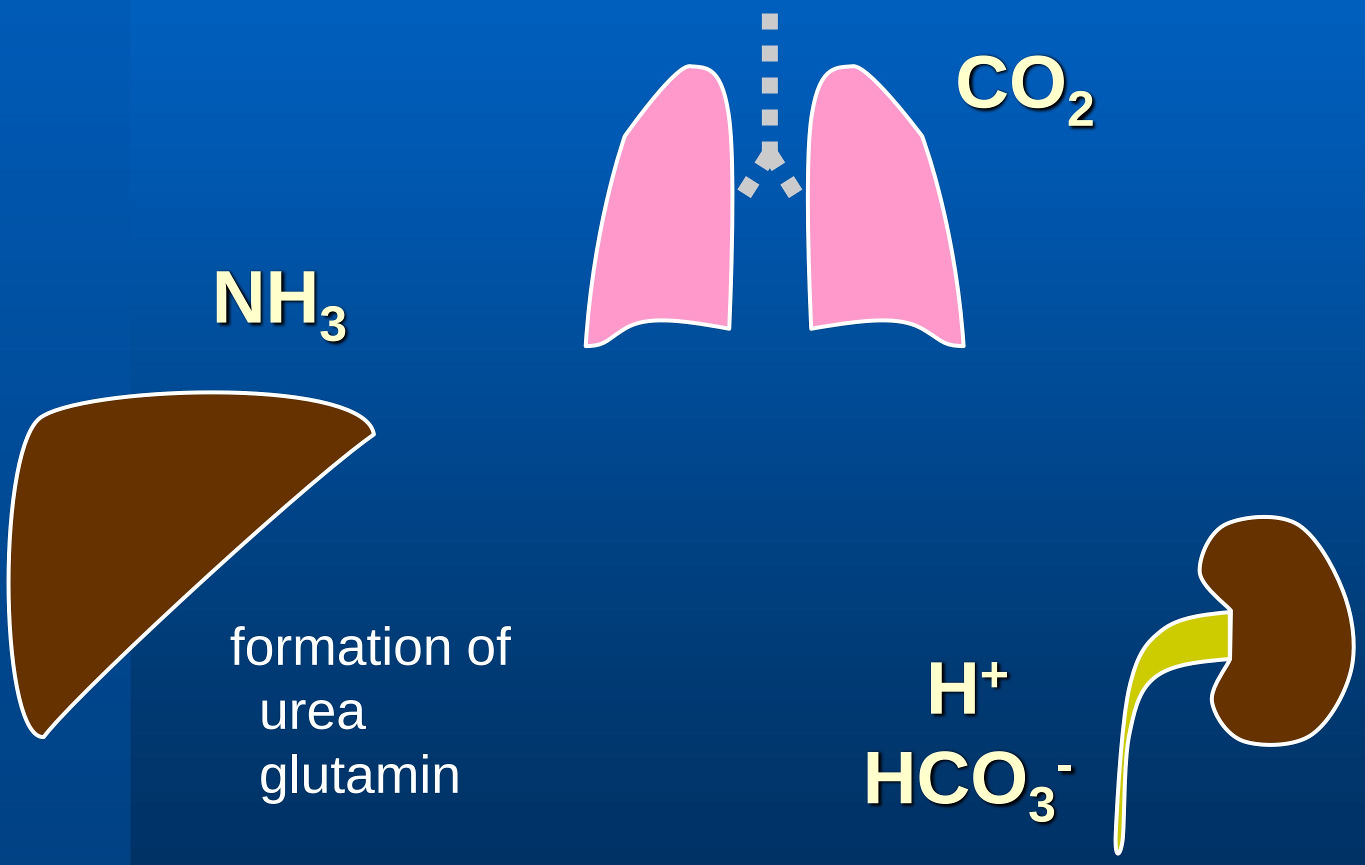
Disorders of acid-base equilibrium

Pathobiochemistry and diagnostics of acid-base and mineral metabolism

Maintaining acidity of inner environment



hydrogencarbonate buffer

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

Henderson-Hasselbalch equation

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

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

$$\frac{[HCO_3^-]}{[H_2CO_3]} = 20$$

Henderson-Hasselbalch equation

$$pH = pK_a + \log \frac{[HCO_3^-]}{\alpha \cdot pCO_2}$$

- $pK_a = 6,1$
- $[HCO_3^-] = 24\ mmol.l^{-1}$
- $\alpha = 0,224\ mmol.l^{-1} / kPa$ $pCO_2 = 5,3\ kPa$

Hydrogencarbonate buffer

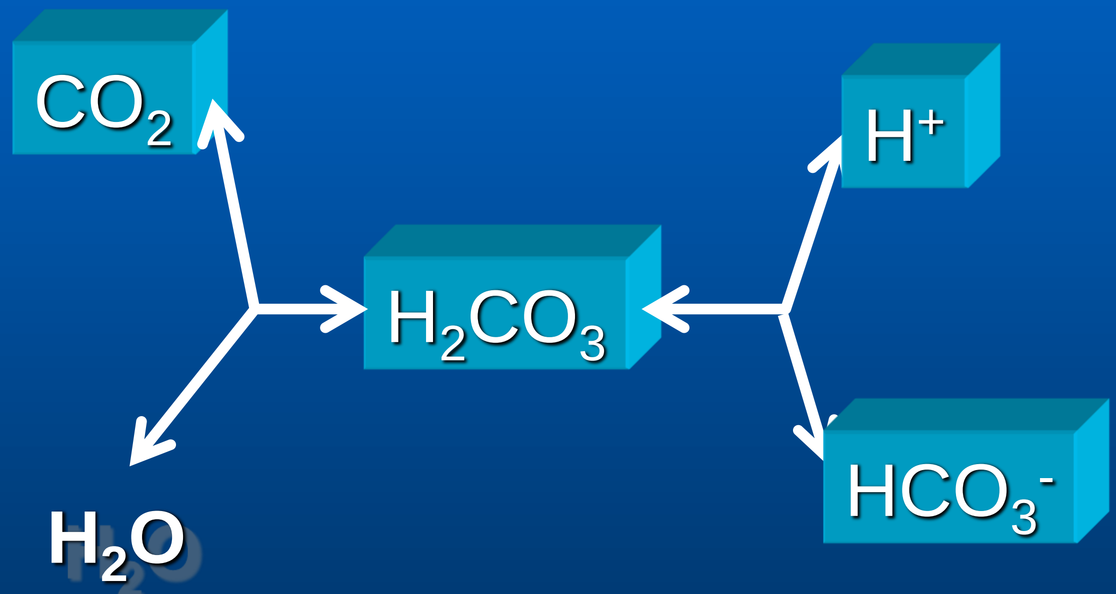
tissue

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

lungs

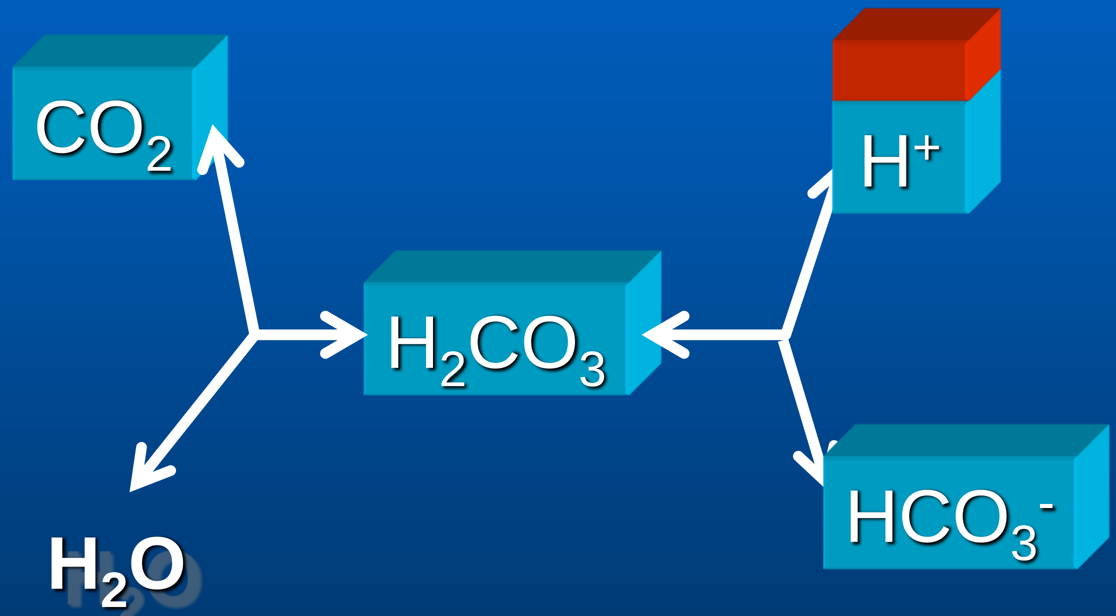
kidney

Hydrogencarbonate buffer

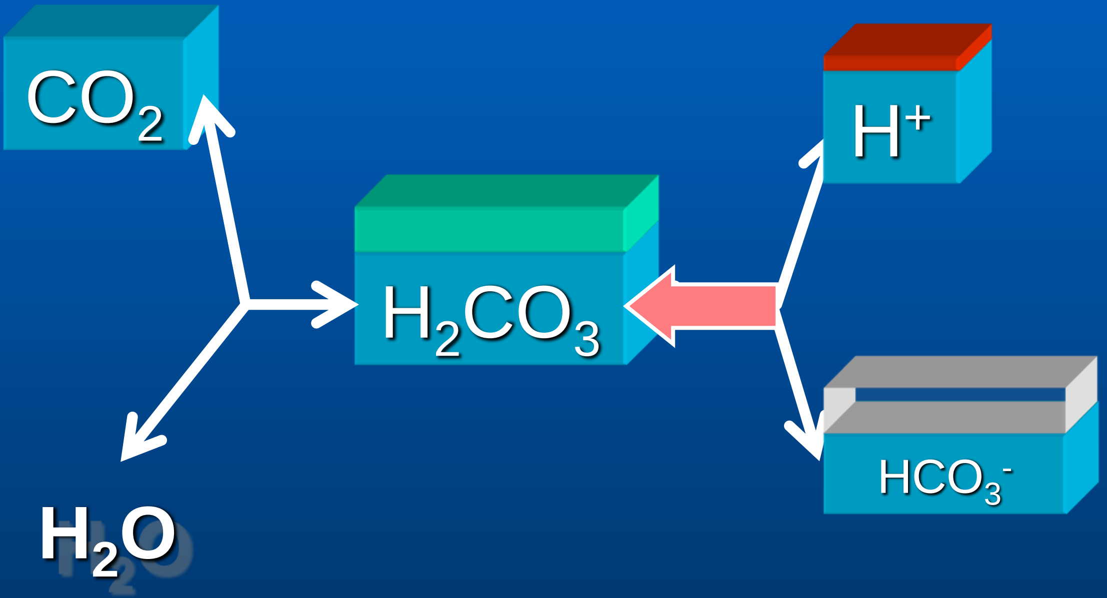


NB: box size does not correspond to concentration!

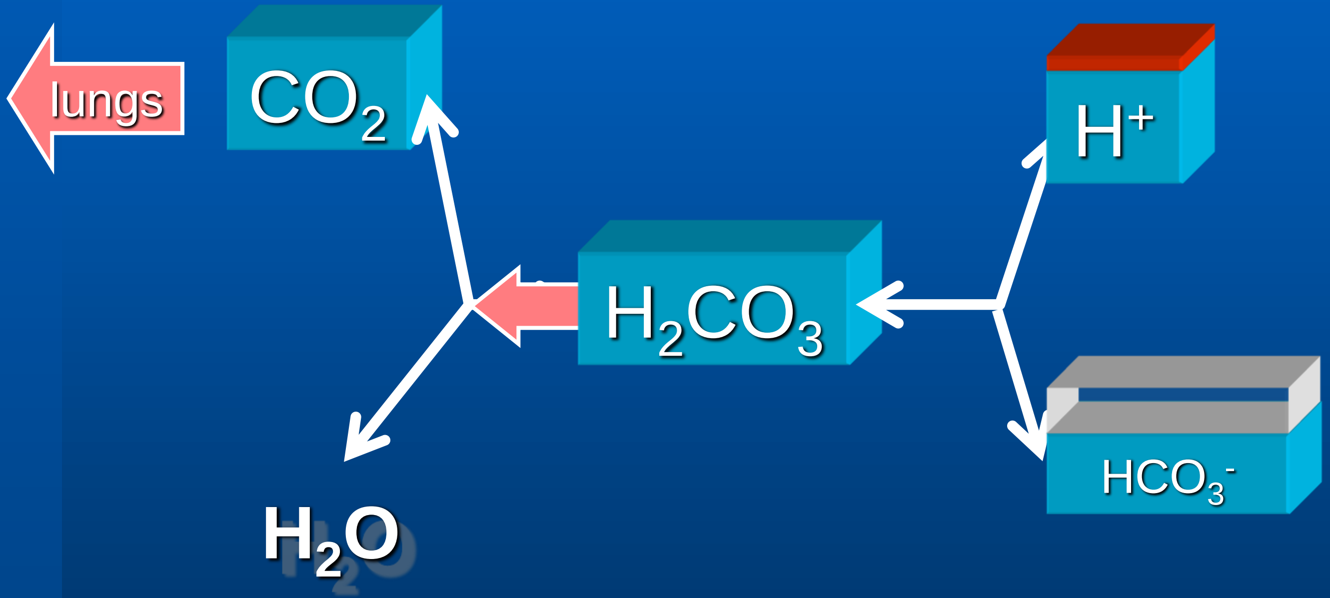
Hydrogencarbonate buffer



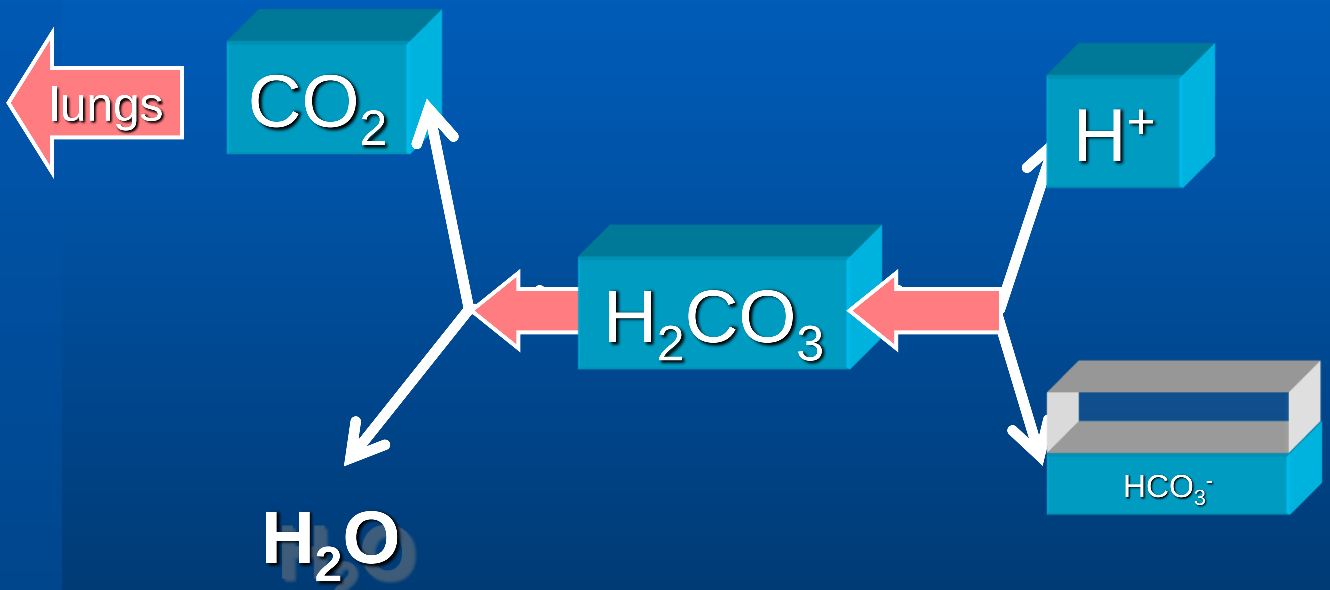
Hydrogencarbonate buffer



Hydrogencarbonate buffer



Hydrogencarbonate buffer



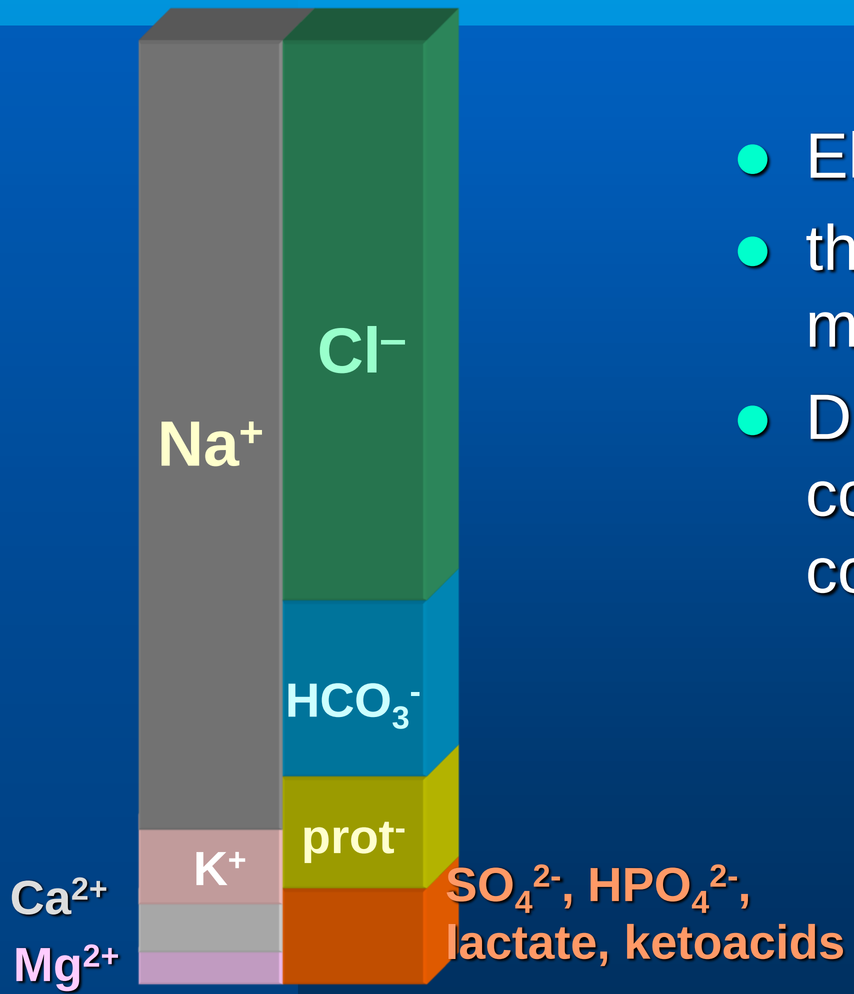
ABE and potassium

- **Exchange of K^+ a H^+ on cell membrane**
  - acidemia → hyperkaliemia
  - alkalemia → hypokaliemia
  - hyperkaliemia → acidemia
  - hypokaliemia → alkalemia

ABE and calcium

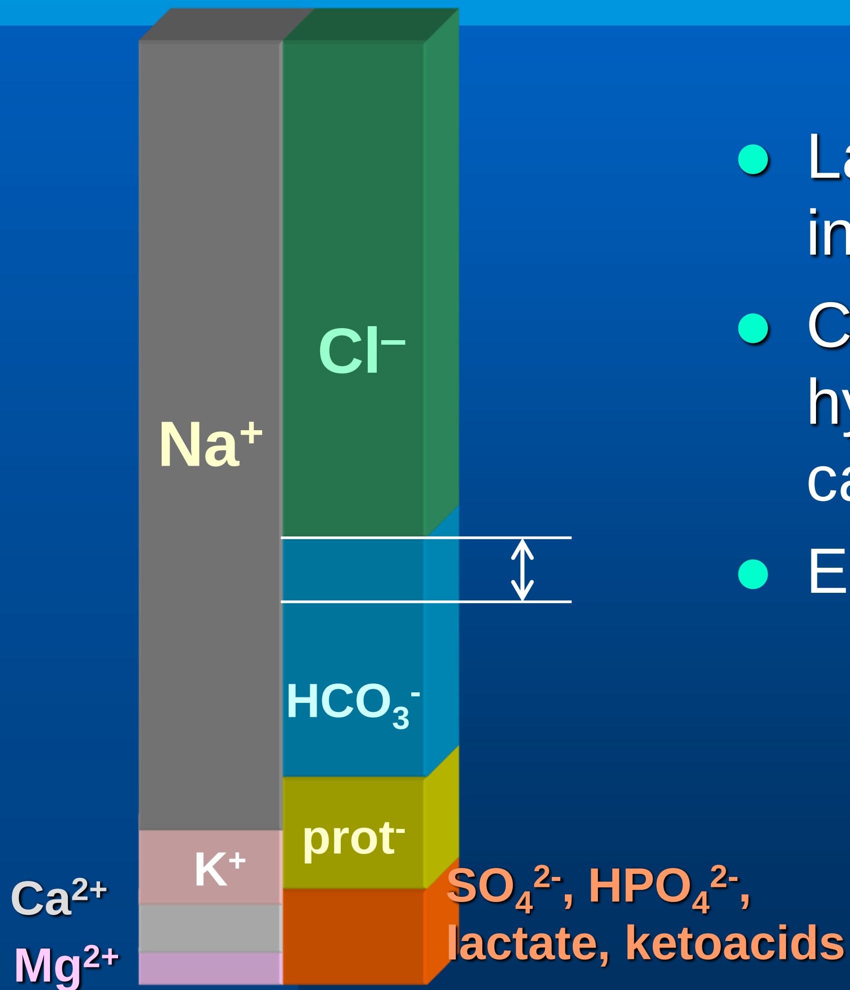
- Exchange of H^+ and Ca^{2+} on plasma proteins
- acidemia → hypercalcemia
- alkalemia → hypocalcemia

ABE and ions



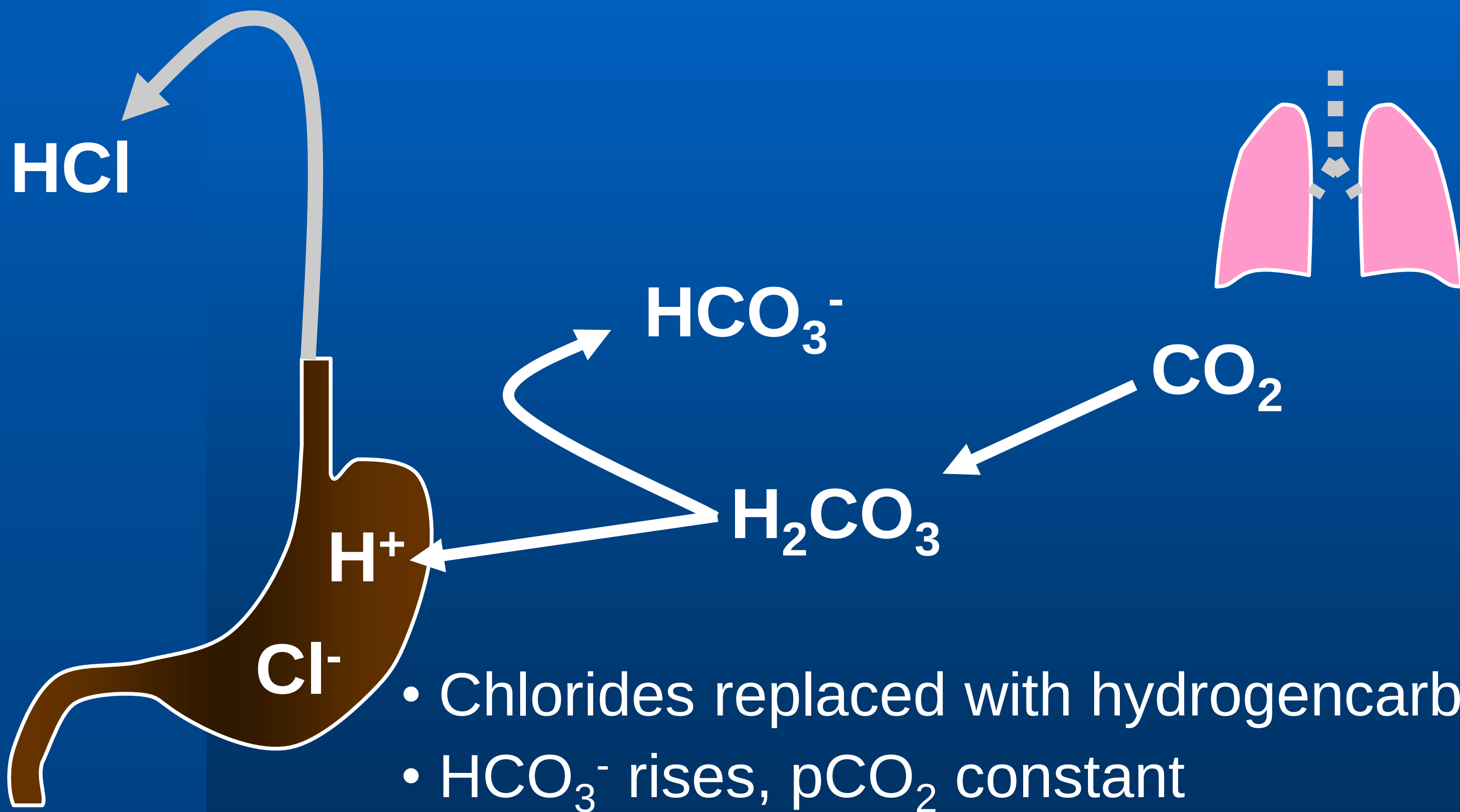
- Electroneutrality must be kept
- thus: ABE influences mineral metabolism
- Deviations in ion concentrations are most easily compensated by HCO_3^-

Hypochloremic alkalosis



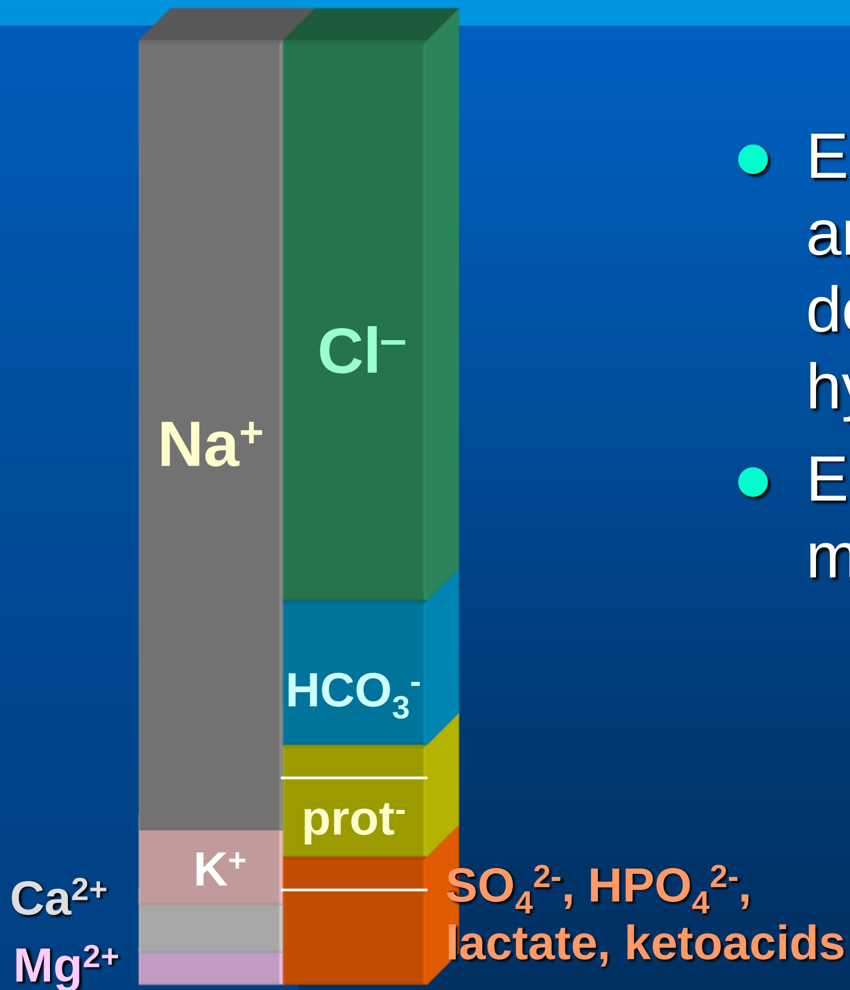
- Lack of Cl^- is compensated by increased HCO_3^-
- Changed ratio hydrogencarbonate / CO_2 causes alkalosis
- E.g. in vomiting

Hypochloremic alkalosis



- Chlorides replaced with hydrogencarbonates
- HCO_3^- rises, pCO_2 constant

Ketoacidosis



- Excess of β-hydroxybutyric and acetacetic acid leads to decreased hydrogencarbonate
- E.g. decompensed diabetes mellitus, starvation…

Acid-base equilibrium

Don‘t think about change in

~~H^+ or OH^-~~

but about changes in

<u>concentration of major ions</u>

pH change is secondary to change of HCO_3^- / pCO_2 ratio

Metabolic acidosis (MAC)

- **Lactate acidosis**
  – hypoxia, poor lactate degradation
- **Ketoacidosis**
  – diabetes, starvation, alcoholism…
- **Renal acidosis**
  – accumulation of sulphates, phosphates…
- **Intoxication**

MAC in loss of HCO_3^-

- **Diarrhoea and othe loss from GIT**
- **Renal tubular acidosis**
  - – disorder of HCO_3^- reabsorption in tubuli
- **Dilution acidosis**
  - – large amount of infusions lacking buffering system
    (pCO_2 constant, HCO_3^- quickly diluted)

Metabolic alkalosis (MAL)

- **Excessive loss of chlorides**
  - vomiting, diuretics
- **Dehydratation (concentration alkalosis)**
- **Hypoproteinemia**
- **Hyperaldosteronism**
  - retention of Na^+ at the expense of K^+ & H^+

Astrup

7,4

5,3

24,0

24,0

0,0

12,0

98,0

27,0

Astrup

pH	**7,4**
	5,3
	24,0
	24,0
	0,0
	12,0
	98,0
	27,0

- Actual pH of blood

  NV: 7,40 ± 0,04

Astrup

pH	**7,4**	
pCO_2	**5,3**	kPa
	24,0	
	24,0	
	0,0	
	12,0	
	98,0	
	27,0	

- Partial CO_2 pressure

  NV: 5,3 ± 0,5 kPa

  – Respiration

Astrup

pH	**7,4**	
pCO_2	**5,3**	kPa
HCO_3^- act.	**24,0**	$mmol.l^{-1}$
	24,0	
	0,0	
	12,0	
	98,0	
	27,0	

- Actual hydrogencarbonates

  NV: 24 ± 2 $mmol.l^{-1}$

Astrup

pH	**7,4**	
pCO_2	**5,3**	kPa
HCO_3^- act.	**24,0**	$mmol.l^{-1}$
HCO_3^- std.	**24,0**	$mmol.l^{-1}$
	0,0	
	12,0	
	98,0	
	27,0	

- Standard HCO_3^-

  what would HCO_3^- be in blood saturated to 5.3 kPa CO_2

  NV: 24 ± 2 $mmol.l^{-1}$

Astrup

pH	**7,4**	
pCO_2	**5,3**	kPa
HCO_3^- act.	**24,0**	$mmol.l^{-1}$
HCO_3^- std.	**24,0**	$mmol.l^{-1}$
BE	**0,0**	$mmol.l^{-1}$
	12,0	
	98,0	
	27,0	

- Base excess

  how much strong acid should be added to blood at pCO_2 = 5,3 kPa in order to get pH = 7,4

  NV: 0 ± 2,5 $mmol.l^{-1}$

  – Metabolic

Astrup

pH	**7,4**	
pCO_2	**5,3**	kPa
HCO_3^- act.	**24,0**	$mmol.l^{-1}$
HCO_3^- std.	**24,0**	$mmol.l^{-1}$
BE	**0,0**	$mmol.l^{-1}$
pO_2	**12,0**	kPa
	98,0	
	27,0	

- Partial pressure of O_2

  NV: 10,0 - 13,3 kPa

  – Respiration

Astrup

pH	7,4	
pCO_2	5,3	kPa
HCO_3^- act.	24,0	$mmol.l^{-1}$
HCO_3^- std.	24,0	$mmol.l^{-1}$
BE	0,0	$mmol.l^{-1}$
pO_2	12,0	kPa
sat. O_2	98,0	%
	27,0	

- Haemoglobin saturation with oxygen
  NV: 94 - 99 %
  - Useful to check results

Astrup

pH	**7,4**	
pCO_2	**5,3**	kPa
HCO_3^- act.	**24,0**	$mmol.l^{-1}$
HCO_3^- std.	**24,0**	$mmol.l^{-1}$
BE	**0,0**	$mmol.l^{-1}$
pO_2	**12,0**	kPa
sat. O_2	**98,0**	%
tot. CO_2	**27,0**	$mmol.l^{-1}$

- Total carbonate, i.e. $CO_2 + H_2CO_3 + HCO_3^-$

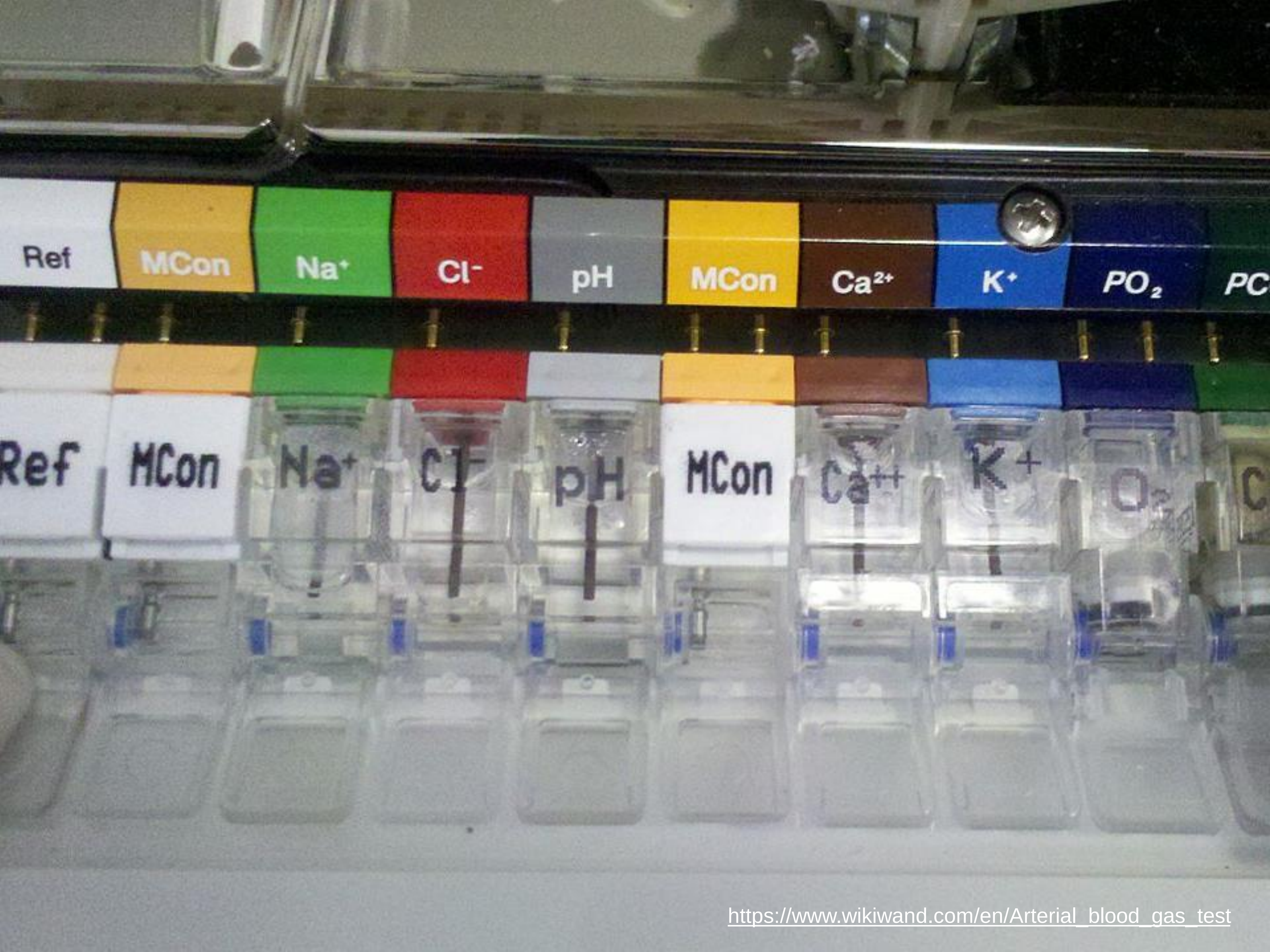
Astrup

- **Full heparinised blood**
  - **capillary (arterialised)**
  - **arteriary**
  - **venous**

Vyšetření podle Astrupa

pH	**7,4**	
pCO_2	**5,3**	kPa
HCO_3^- akt.	**24,0**	$mmol \cdot l^{-1}$
HCO_3^- std.	**24,0**	$mmol \cdot l^{-1}$
BE	**0,0**	$mmol \cdot l^{-1}$
pO_2	**12,0**	kPa
sat. O_2	**98,0**	%
celk. CO_2	**27,0**	$mmol \cdot l^{-1}$

https://www.healthcare.siemens.it/point-of-care/blood-gas/rapidlab-248-348-systems
https://en.wikipedia.org/wiki/File:Arterial_blood_gas_device.jpg



https://www.wikiwand.com/en/Arterial_blood_gas_test

Liver failure

- **Alkalosis**
  - – hypoproteinaemia
  - – hyperaldosteronism
  - – ↓ of ureasynthesis from ammonia

Renal failure

- **Acidosis**
  - – phosphate and sulphate retention
  - – poor urine acidification

Combined ABE disorders

- **MAC + MAL**
  - vomiting + starvation
  - vomiting + diarrhoea
  - renal failure + uraemic vomiting
  - hepatorenal failure

Case 1

pH	**7,156**	
pCO_2	**4,15**	kPa
HCO_3^- act.	**11,1**	$mmol.l^{-1}$
HCO_3^- std.	**12,4**	$mmol.l^{-1}$
BE	**-15,7**	$mmol.l^{-1}$
pO_2	**10,1**	kPa
sat. O_2	**90,8**	%
tot. CO_2	**12,1**	$mmol.l^{-1}$

Na	**141**	$mmol.l^{-1}$
K	**6,2**	$mmol.l^{-1}$
Cl	**110**	$mmol.l^{-1}$
TP	**58,8**	$g.l^{-1}$

Case 2

pH	**7,378**	
pCO_2	**4,49**	kPa
HCO_3^- act.	**19,4**	$mmol.l^{-1}$
HCO_3^- std.	**20,7**	$mmol.l^{-1}$
BE	**-4,4**	$mmol.l^{-1}$
pO_2	**8,8**	kPa
sat. O_2	**92,4**	%
tot. CO_2	**16,9**	$mmol.l^{-1}$

Na	**141**	$mmol.l^{-1}$
K	**4,6**	$mmol.l^{-1}$
Cl	**106**	$mmol.l^{-1}$
TP	**65,7**	$g.l^{-1}$

Case 3

pH	**7,454**	
pCO_2	**5,0**	kPa
HCO_3^- act.	**25,9**	$mmol.l^{-1}$
HCO_3^- std.	**26,1**	$mmol.l^{-1}$
BE	**2,6**	$mmol.l^{-1}$
pO_2	**5,2**	kPa
sat. O_2	**76,4**	%
tot. CO_2	**22,4**	$mmol.l^{-1}$

Na	**137**	$mmol.l^{-1}$
K	**4,6**	$mmol.l^{-1}$
Cl	**107**	$mmol.l^{-1}$
TP	**48,8**	$g.l^{-1}$

Case 4

pH	**7,39**	
pCO_2	**3,94**	kPa
HCO_3^- act.	**17,5**	$mmol.l^{-1}$
HCO_3^- std.	**19,6**	$mmol.l^{-1}$
BE	**-5,7**	$mmol.l^{-1}$
pO_2	**7,1**	kPa
sat. O_2	**87,2**	%
tot. CO_2	**15,3**	$mmol.l^{-1}$

Na	**137**	$mmol.l^{-1}$
K	**5,8**	$mmol.l^{-1}$
Cl		$mmol.l^{-1}$
TP	**62,3**	$g.l^{-1}$

Disorders of acid-base equilibrium

Pathobiochemistry and diagnostics of acid-base and mineral metabolism