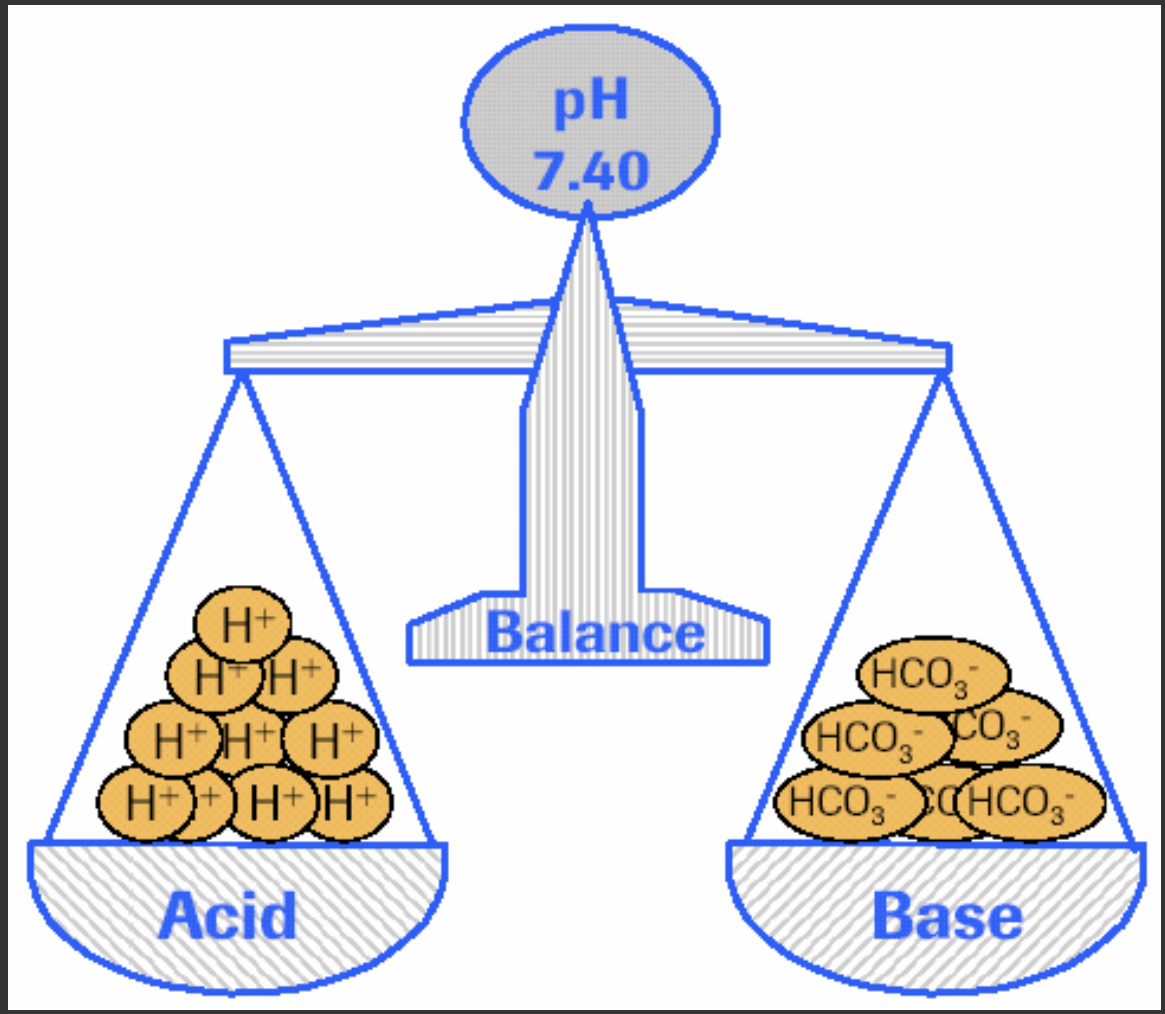
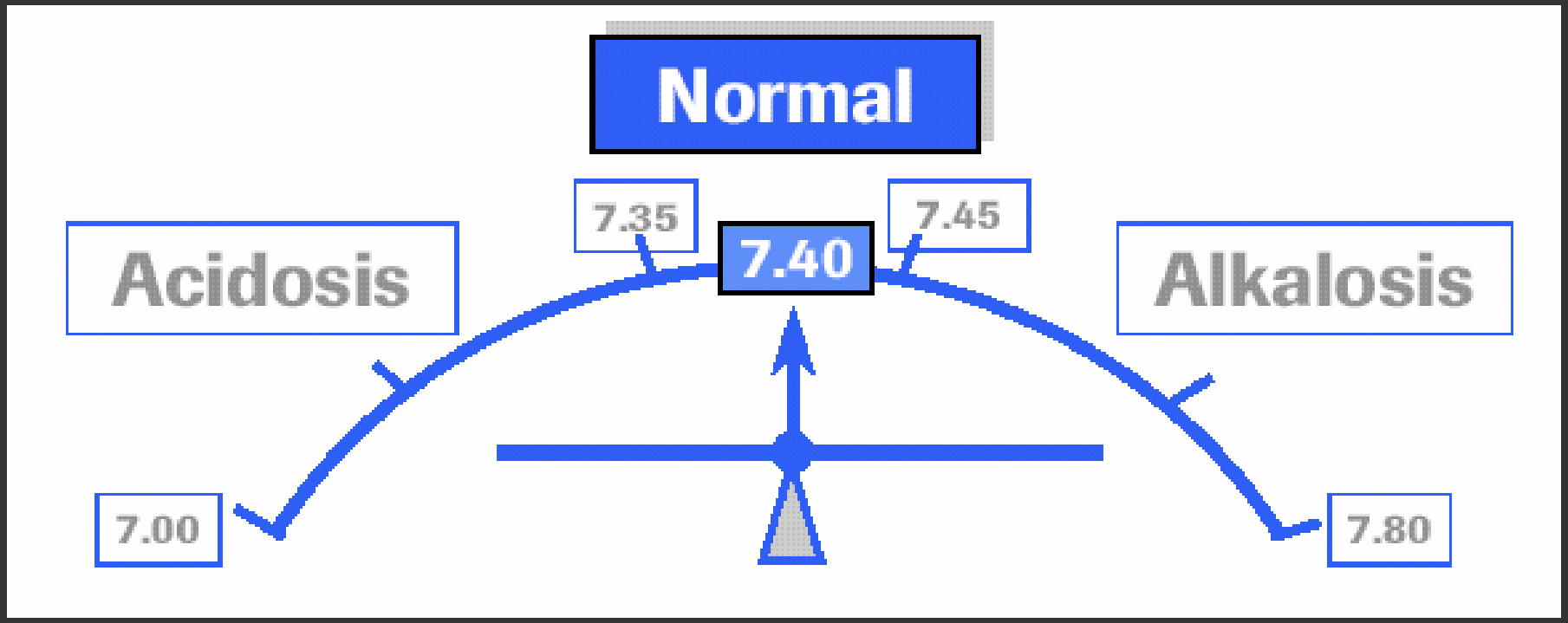
ABR: acid-base balance



Normal pH - very close ranges



Analyte	Unit	Reference intervale
pH		7.35 - 7.45
pCO_2	kPa	4.8 - 5.6
pO_2	kPa	10 - 13
actual HCO_3	mmol/l	22 - 26
standart HCO_3	mmol/l	22 - 26
BE (base exces)	mmol/l	$\pm$ 2

pH - activity of hydrogen ions

- pH: 7.35-7.45

- Acidosis: pH < 7.35
  - „common“: 7.35 - 7.10
  - serious: 7.10 - 6.80
  - extreme: < 6.80 (life threatening)

- Alkalosis: > 7.45
  - serious: 7.45 - 7.70
  - extreme: > 7.70

Not serious acid-base disorders

- „Common“ acidosis: pH 7.35-7.10
  - „Physiologic" situation
- Physiologic occur acids
  - Ketonic acids
  - Lactic acid
- There is no „common“ alkalosis
  - Alkalosis is always serious disorder
  - It often results from inadequate therapy of acidosis
- Normal pH
  - Aide-base disorder may be present

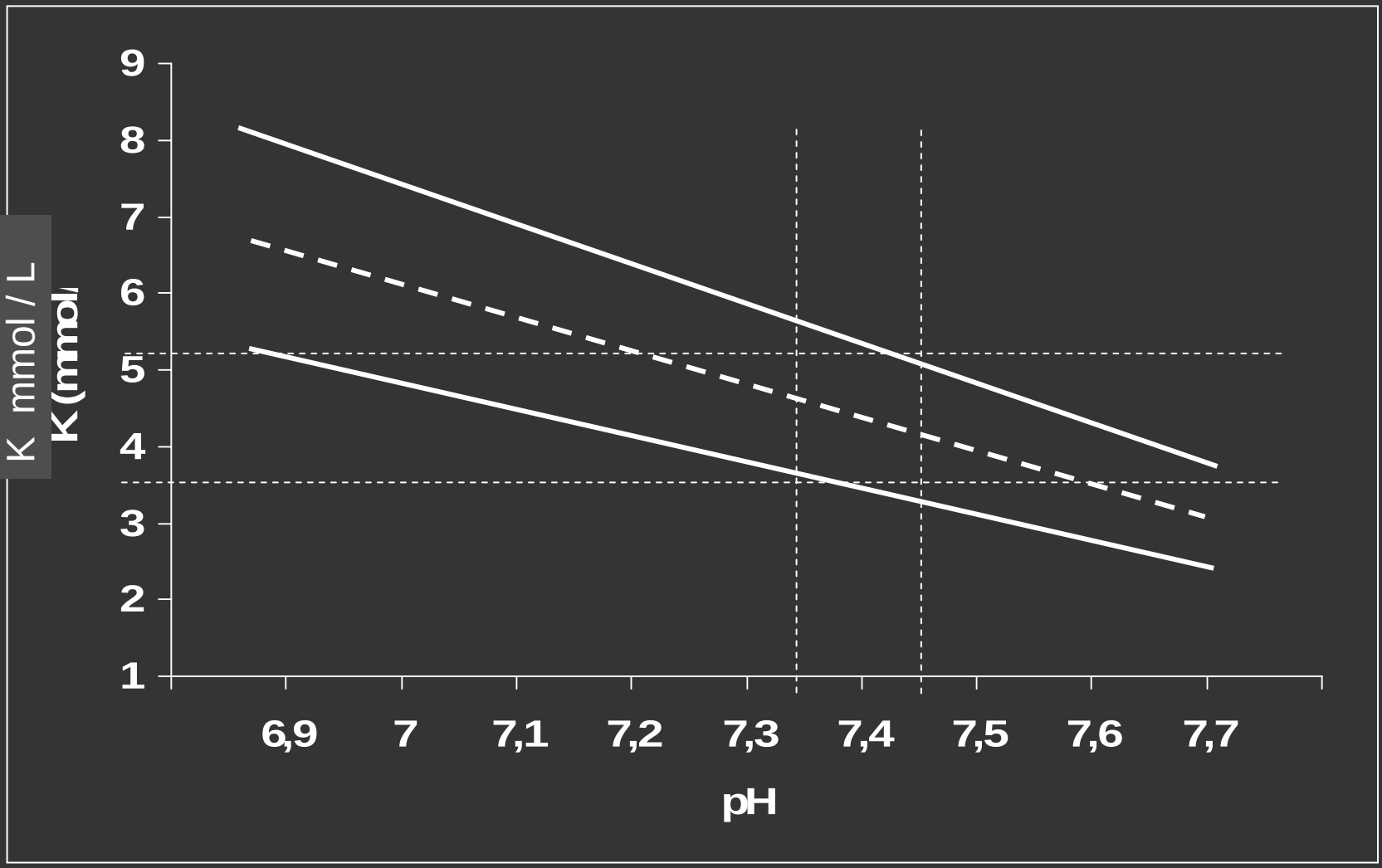
Correction of pH changes

- Buffers
  - To correct the influence of acids and bases
- Reaction of buffers
  - Immediately
- Compensation
  - With delay
  - Lungs and/or kidney

Buffers

- Bicarbonate
  - Extracellular fluid (blood, interstitial fluid)
  - Cerebrospinal fluid - activity of the respiratory centre
- Haemoglobin
  - Intravascular fluid
- Phosphate
  - Intracellular fluid
  - Connection to the concentration of K^+ !
- Proteins
  - Both intra and extracellular fluid
  - Small capacity

Relationship between K a pH



K and pH

- Acidosis = excess of H^+ ions
  - H^+ move to ICF, where is bound to phosphate buffer, K^+ is released from buffer and move out to maintain electroneutrality
  - Concentration of K^+ increase

K and pH

- Alcalosis = lack of H^+ ions
  - H^+ is released in ICF from phosphate buffer, K^+ move into cells and is bound to P buffer
  - Concentration of K^+ decrease

Buffers

- Blood
  - Bicarbonate 53 %
  - Phosphate 5 %
  - Haemoglobin 35 %
  - Proteins 7 %

HCO_3^- a Hb system

- Bicarbonate
  - Concentration: 24 ± 2 mmol/l
  - Regulation of HCO_3^- level: kidney

- Haemoglobin
  - Similar capacity as HCO_3^-
  - Oxidized HbO is stronger acid: it releases H^+
    - ★Tissues: $HbO \rightarrow Hb$: binding of H^+
    - ★Lungs: $Hb \rightarrow HbO$: release of H^+

Compensation of A-B disorders

- Respiratory A-B disorders
  - Renal compensation
- „Metabolic“ A-B disorders
  - Lung and renal compensation

Respiratory compensation of A-B disorders
↑ or ↓ of ventilation

- Metabolic acidosis: hyperventilation
  - Stimulation of respiratory centre by low pH
  - Very effective mechanism to ↓ pCO_2
  - Good tissues saturation with O_2

- Metabolic alkalosis: hypoventilation = hypoxia !!
  - Inhibition of respiratory centre by elevated pH
  - Ineffective mechanism
  - ↑ pCO_2, ↓ pO_2, hypoxia = hypoventilation is cancelled

Respiratory compensation of A-B disorders
↑ or ↓ of ventilation

- Respiratory compensation started immediately, but
  - Maximal compensation: 24 hours
  - Hyperventilation persists after adjustment of acidosis
    - ★Risk of respiratory alkalosis development

Renal compensation of A-B disorders

- Acidosis
  - Synthesis of HCO_3^-
  - Excretion of H^+ to urine
    - ★Ammonium ions (NH_4^+)
    - ★Phosphate ions ($H_2PO_4^-$)
- Alkalosis:
  - Excretion of HCO_3^- to the urine
  - Inhibition of H^+ excretion
    - ★Stop of NH_4 synthesis
    - ★HPO_4^{2-} synthesis is started

Renal compensation of A-B disorders

- Renal compensation starts during 24 hours
  - It is complete is after 1 week
  - When is acidosis or alkalosis removed (therapy), compensation continues several days
    - ★Risk of reverse A-B disorder !

Acid-base disorders

- Simple disorders
  1. Metabolic acidosis
  2. Metabolic alkalosis
  3. Respiratory acidosis
  4. Respiratory alkalosis

- Combined acid-base disorders
  - Result of compensation mechanisms
  - Primary combined disorders

Metabolic acidosis

- SOURCES OF HYDROGEN IONS
  - Cell Metabolism
  - Food Products
  - Medications
  - Metabolic Intermediate by-products
  - Some disease processes

METABOLIC ACIDOSIS

- Metabolic acidosis is always characterized by a reduction in plasma **HCO_3^-** while **CO_2** remains normal

Metabolic acidosis

- Laboratory diagnosis:
  - ↓ pH
  - ↓ HCO_3^-
  - ↔ pCO_2 (acute), ↓ (respiratory compensation)

- Causes:
  a) Without HCO_3^- loss
     ★ ↑ acids
  b) Due to loss of HCO_3^-

Acidosis without HCO_3^- loss

- ↑ production of acids
  - Kenotic acids
    - ★Starvation
    - ★Unregulated DM
    - ★High grade fever
  - Ketones
    - ★Acetone
    - ★Acetoacetic acid
    - ★β-hydroxybutyrous acid

Acidosis without HCO_3^- loss

- Others acids
  - Ingestion of
    - ★Ethylene glycol (antifreeze) → oxalic acid
    - ★Methanol → formate acid
    - ★Salicylate

Acidosis without HCO_3^- loss

- Acid retention
  - Acute renal failure, chronic renal failure
  - Kidneys are unable
    - ★To rid the plasma of even the normal amounts of **H^+** generated from metabolic acids
    - ★To conserve an adequate amount of **HCO_3^-**

Acidosis without HCO_3^- loss

- Lactic acidosis type A (hypoxia)
  - Respiratory insufficiency, shock
  - Anaemia (Hb < 70 g/l), carbon monoxide poisoning, extreme muscular activity
  - Blood stagnation

Acidosis without HCO_3^- loss

- Type B (insufficient utilization of lactic acid)
  - Hepatic failure
  - Biguanide poisoning
  - Sepsis

Acidosis due to bicarbonate loss

- Real bicarbonates loss
  - Severe diarrhoea
  - Pancreatic fistula

- Decline of bicarbonates: Hyperchloremic acidosis
  - ↑ chloride intake
    - ★KCl, NaCl, NH_4Cl

Therapy of the metabolic acidosis

- $NaHCO_3$
  - How much ?
- Calculation
  - mmol HCO_3 = BE x 0.3 x weight (kg)

Therapy of the metabolic acidosis

The rules for therapy of met. ac.

- Therapy have to be causal (if possible) !
  - If the acidosis is mild, treatment for the underlying disorder may be all that's needed

Therapy of the metabolic acidosis

The rules for therapy of met. ac.

- HCO_3^- may be given only when
  - Causal therapy is not possible
  - Acidosis is severe: pH < 7,1

- Maximal dose of bicarbonate
  - 1/3 - 1/2 of the calculated dose

Metabolic alkalosis

- Dg:
  - ↑ pH
  - ↑ HCO_3
  - ↔ pCO_2 (acute), ↑ (pulmonary compensation)
    - ★ Breathing suppressed to hold CO_2
- Types of MAL:
  - Responding for treatment with chlorides
    - ★ MAL due to loss of Cl
  - Not responding for treatment with chlorides

MAL due to loss of Cl

- Vomiting
- Drainage of gastric juice
- Diuretic use (thiazides)

MAL due to lack of Cl: therapy

- Cl^- (HCO_3^- ions replaced by Cl^- ions)
  - NaCl, KCl, NH_4Cl, arginin hydrochlorid

- How much of Cl^- ?
  - BE x 0.3 x weignt (kg)
  - Deficiency of Cl^- x 0.3 x weight (kg)

MAL due to lack of Cl: therapy

- The rules for therapy of met. alkalosis
  - Therapy of alkalosis should be started in all cases
  - The full calculated dose of Cl^- should be given

- Alkalosis is more dangerous than acidosis!

MAL not responding for treatment with Cl^-

- Hyperaldosteronism
- Long-term therapy with glukocorticoides
- Iatrogenic
  - ↑ supplementation of HCO_3^-

MAL not responding for treatment with Cl^-:

- Therapy have to be causal
- Hypokalemia - KCl
- Live- threatening MAL - Haemodialysis

Respiratory acidosis (RAC)

- It is characterized by retention of CO_2
- Dg:
  - $\downarrow$ pH
  - $\uparrow$ pCO_2
  - $\leftrightarrow$ HCO_3, then $\uparrow$ (renal compensation)

- Cause of RAC: retention CO_2
  - Central
  - Pulmonary
  - Cardiac

Respiratory acidosis (RAC)

- Central (depression of respiratory centre)
  - Drug induced - sedatives, narcotics
  - Lesions of resp. centre - tumour, trauma, ...

Respiratory acidosis (RAC)

- Pulmonary
  - Neuromuscular (myastenia gravis, botulism)
  - Muscles (myositis, muscular dystrophy)
  - Thorax (pneumothorax)
  - Respiratory tract (asthma, bronchostenosis, tumour)
  - Pulmonary parenchyma (pulmonary edema, ARDS, pneumonia)

Respiratory acidosis (RAC)

- Cardiac
  - Low minute volume of cardiac output

Respiratory acidosis: Therapy

- Therapy must be causal !!
- Hypoxia is more serious problem then acidosis !!!
- Improvement of respiration, sometime oxygen
- In the life threatening RAC
  - Artificial ventilation
- Bicarbonate is contraindicated !!!

- Oxygen must be done with caution !
  - Hypoxia stimulate respiratory centre
    - ★Increased pO_2 may inhibite respiration

Respiratory alkalosis (RAL)

- Stimulation of the respiratory center
- Dg:
  - ↑ pH
  - ↓ pCO_2
  - ↔ HCO_3, then ↓ (kidney compenzation)

Respiratory alkalosis (RAL)

- Causes of RAL
  - Anxiety, emotional disturbances, hysteria
  - Pulmonary embolism
  - Lesions of the CNS (respiratory center)
    - Encephalitis, meningitis, tumours, trauma
  - Pregnancy
  - Fever
  - High altitude (low pO_2)
    - Too much CO_2 is “blown off”

Respiratory alkalosis (RAL): Therapy

- Depression of the respiratory center
  - Sedatives

- Life threatening RAL
  - Arteficial ventilation

Combined A-B disorders

- Primary combined A-B disorders

- Result of the compensation
  - Metabolic acidosis is compensated by respiratory alkalosis
  - Metabolic alkalosis is compensated by respiratory acidosis
  - Respiratory acidosis is compensated by metabolic alkalosis
  - Respiratory alkalosis is compensated by metabolic acidosis

How to recognized combined A-B disorders ?

- Respiratory component is present, if
  - pCO_2 is changed

- Sometimes it may be difficult to detect metabolic components, as both acidosis and alkalosis may be present, resulting in the normal laboratory values (pH, HCO_3, pCO_2, pO_2):
  - Concentration of Cl^- , K^+, Na^+ must be measured !
  - Some calculation may be useful

Calculations that help recognize combined disorders

- Buffer Base = $Na^+ + K^+ - Cl^-$
  - Normal result: 42 mmol/l
  - ↑ = metabolic alkalosis is probably present

Calculations that help recognize combined disorders

- Anion gap = $(Na^+ + K^+) - (Cl^- + HCO_3^-)$
- Normal result = 18 mmol/l
- ↑ = metabolic acidosis is probably present (↑concentration of organics anions)
  - Lactate, kenotic acids, multiple acid radicals

How to recognized combined A-B disorders ?

- Normal pH do not exclude A-B disorders !

- What we need for correct interpretation of A-B status ?
  - pH, HCO_3^-, pCO_2, pO_2
  - Na, K, Cl
  - **Patients´s history and clinical examination!**

How to recognized combined A-B disorders ?

- An interpretation of the blood´s A-B status must take into account the electrolyte status
- ***Cl and K deserve special attention!***
- Changes in Cl^- conc. are followed by the changes in A-B status
  - ↑ of Cl^- results in ↓ of HCO_3^- and it is followed by metabolic acidosis (hyperchloremic acidosis)
  - ↓ of Cl^- results in ↑ of HCO_3^- and it is followed by metabolic alkalosis (hypochloremic alkalosis)