Acid-base status assessment

Jiří Kofránek, Stanislav Matoušek

Outline

- Bicarbonate buffer
- Four basic types of disturbances
  - RAC, RAL, MAC and MAL
- Compensation of each disturbance
- Other buffers: BE, standard HCO_3^-
- Extras: Stewart theory or dilution acidosis

What do we take?

- Arterial blood gas measurement ("Astrup")
- Serum electrolytes
- Other

Arterial blood gas measurement

Apparatus measures:

- **pH** (7.35 – 7.45)
  Or analogous value: H+ = 35 – 45 **nmol**/l
- **pCO2** (40 Torr = 5.3 kPa)
- **pO2** (100 Torr = 13,3 kPa)

Apparatus calculates:

- **HCO_3^-** (24 mmol/l) *From Henderson-Hasselbalch Equation*
- **BE** (0 mmol/l) *From in-built Siggaard-Andersen nomogram*

Possible problems:

/ Visible air bubble stays in the syringe and dissolves in the sample

/ The sample is not analyzed right away. Metabolic processes cause changes in AB parameters. (If immediate analysis is not possible, the sample should be kept in ice-bath)

Serum electrolytes

- **Na+ (135 – 145 mmol/l)**
- **Cl- (97 – 108 mmol/l)**
- **Total CO2 or HCO_3^- (24 mmol/l)** .....should be equal to **HCO_3^-** from Astrup – can check the measurement validity

Additional:

- Phosphates >> $H_2PO_4^-$ => HPO_4^{2-} (1 – 1,5)
- K+ (3.5 – 5 mmol/l)
- Ca++ (2.4 mmol/l)
- SO_4^{2-}

Other

- **Hb** (120 – 170 g/L)

Additional:

Albumins (35 – 50 g/l)

Lactate (0.5 – 2.5)

Ketoacids (0)

Toxic substances: salycilates, methanol etc.

Why is pH (concentration of H^+) so important?

- Normal concentration of H^+ in plasma is 0.000 04 mmol/L = 40 nmol/L
- Very high chemical activity of hydrogen ions (protons) in solution
- Changes of pH influence spacial conformation of proteins
- however, in other fluids, it can different by many orders of magnitude
- **$pH = -\log_{10}(H^+)$**
- **40 nmol/L = pH 7.4**

change 2x .... -0.3

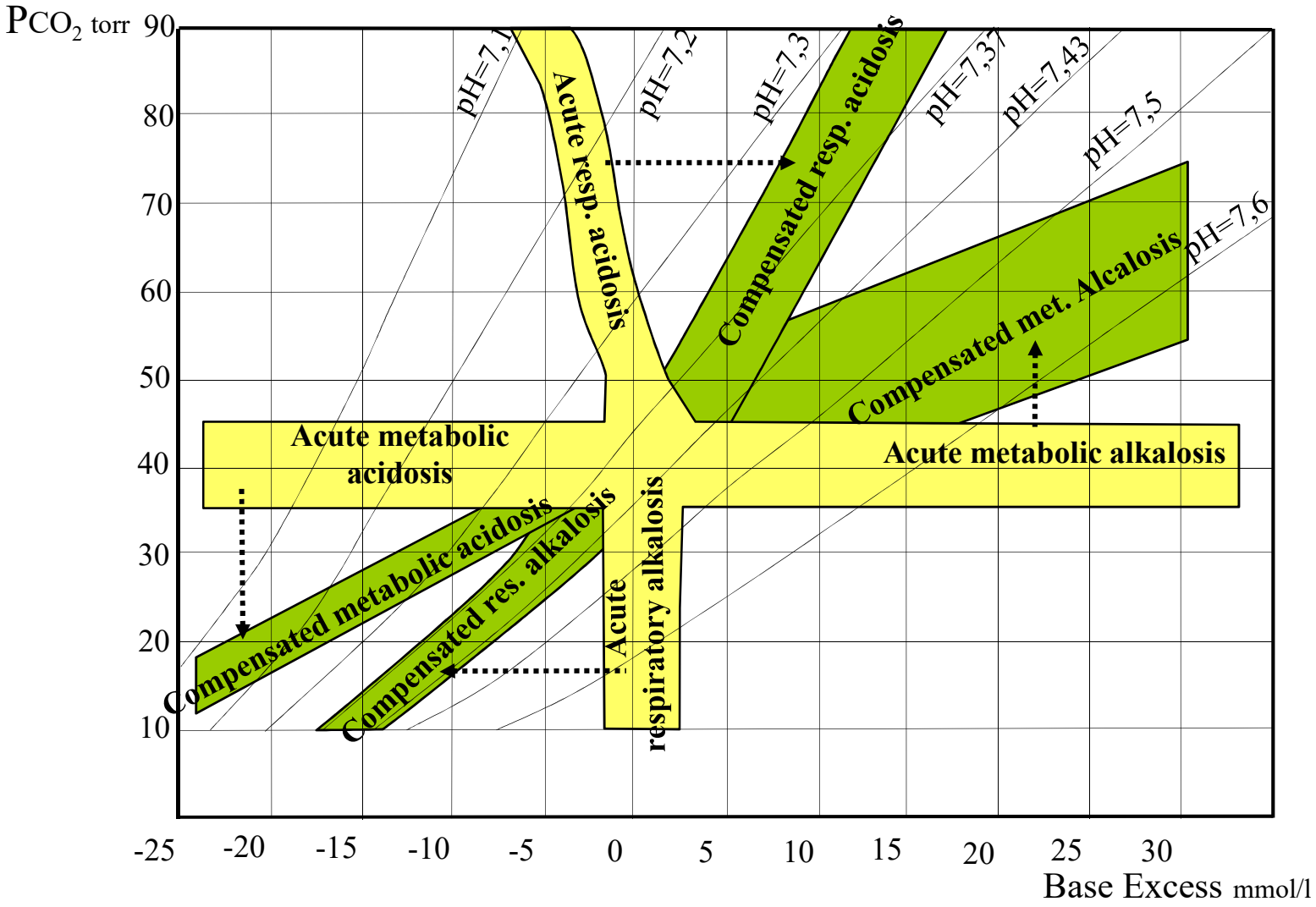
(change 1/2x..... +0.3)

change 4x .... -0.6

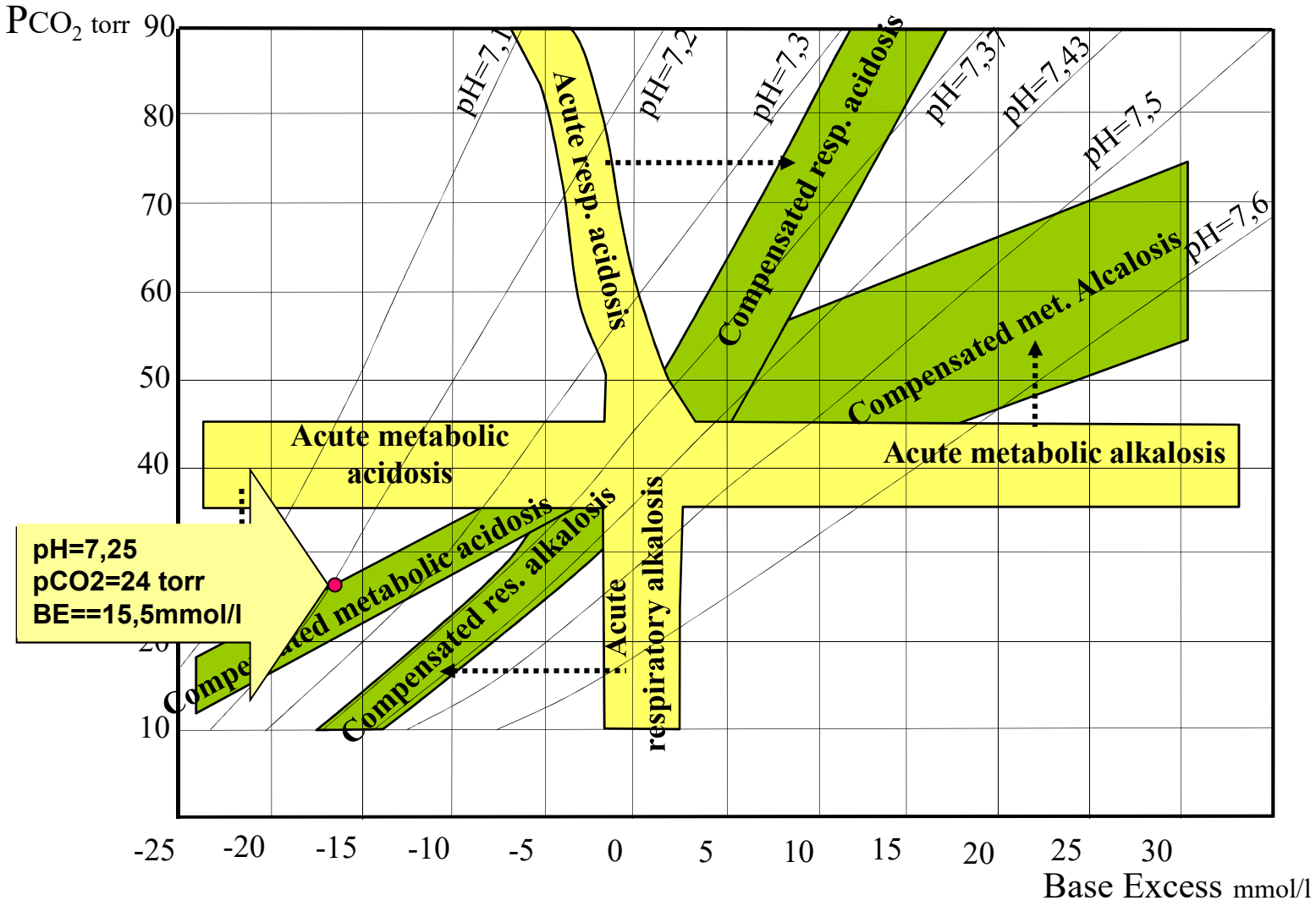
change 8x ..... -0.9

change 10x .....- 1.0

How is $[H^+]$ determined?


PCO2 torr
90
80
70
60
50
40
30
20
10
pH=7,1
pH=7,2
pH=7,3
pH=7,37
pH=7,43
pH=7,5
pH=7,6
Acute resp. acidosis
Compensated resp. acidosis
Compensated met. Alcalosis
Acute metabolic acidosis
Acute metabolic alkalosis
Compensated metabolic acidosis
Compensated res. alkalosis
Acute respiratory alkalosis
-25
-20
-15
-10
-5
0
5
10
15
20
25
30
Base Excess mmol/l

Compensated metabolic acidosis

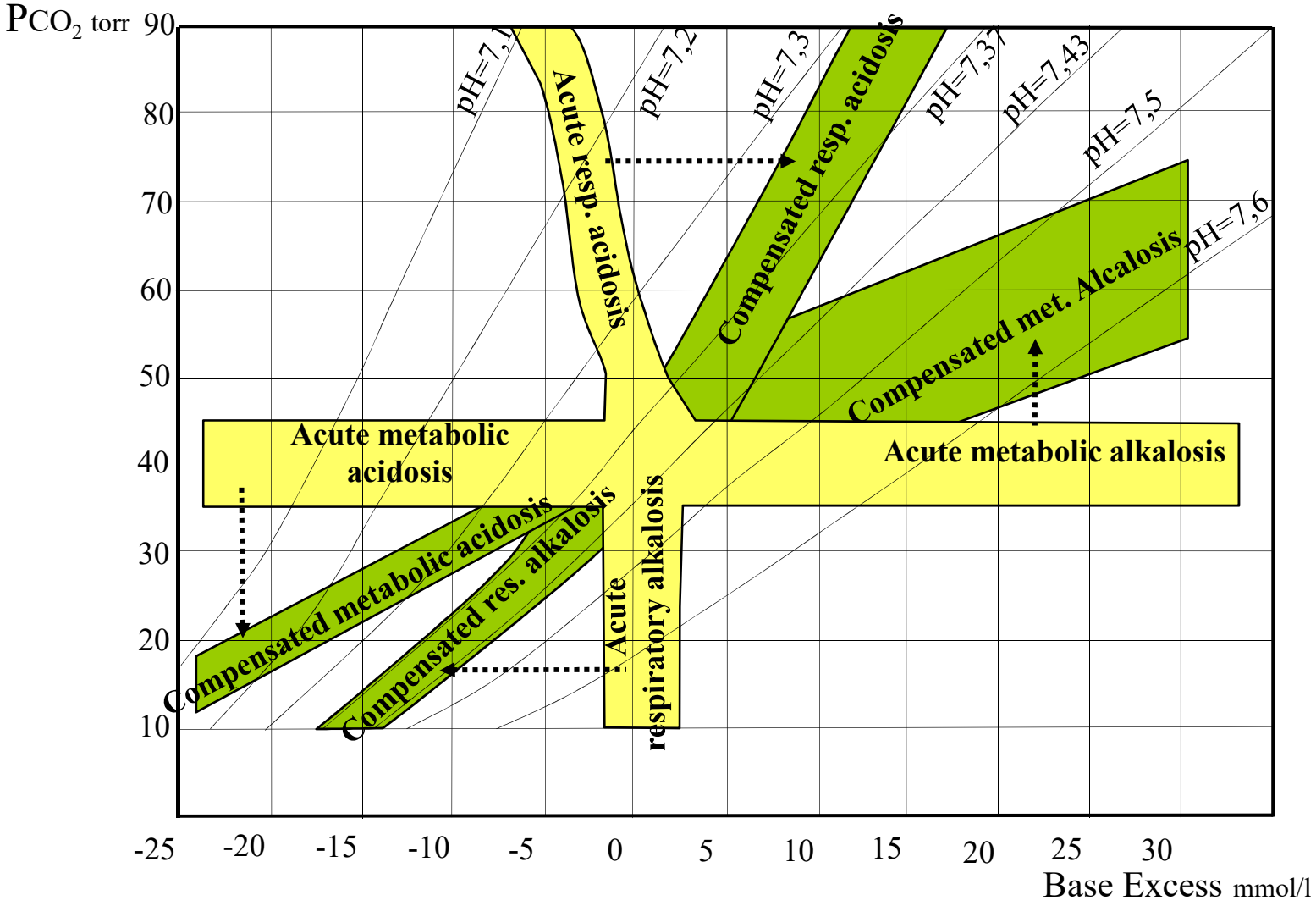


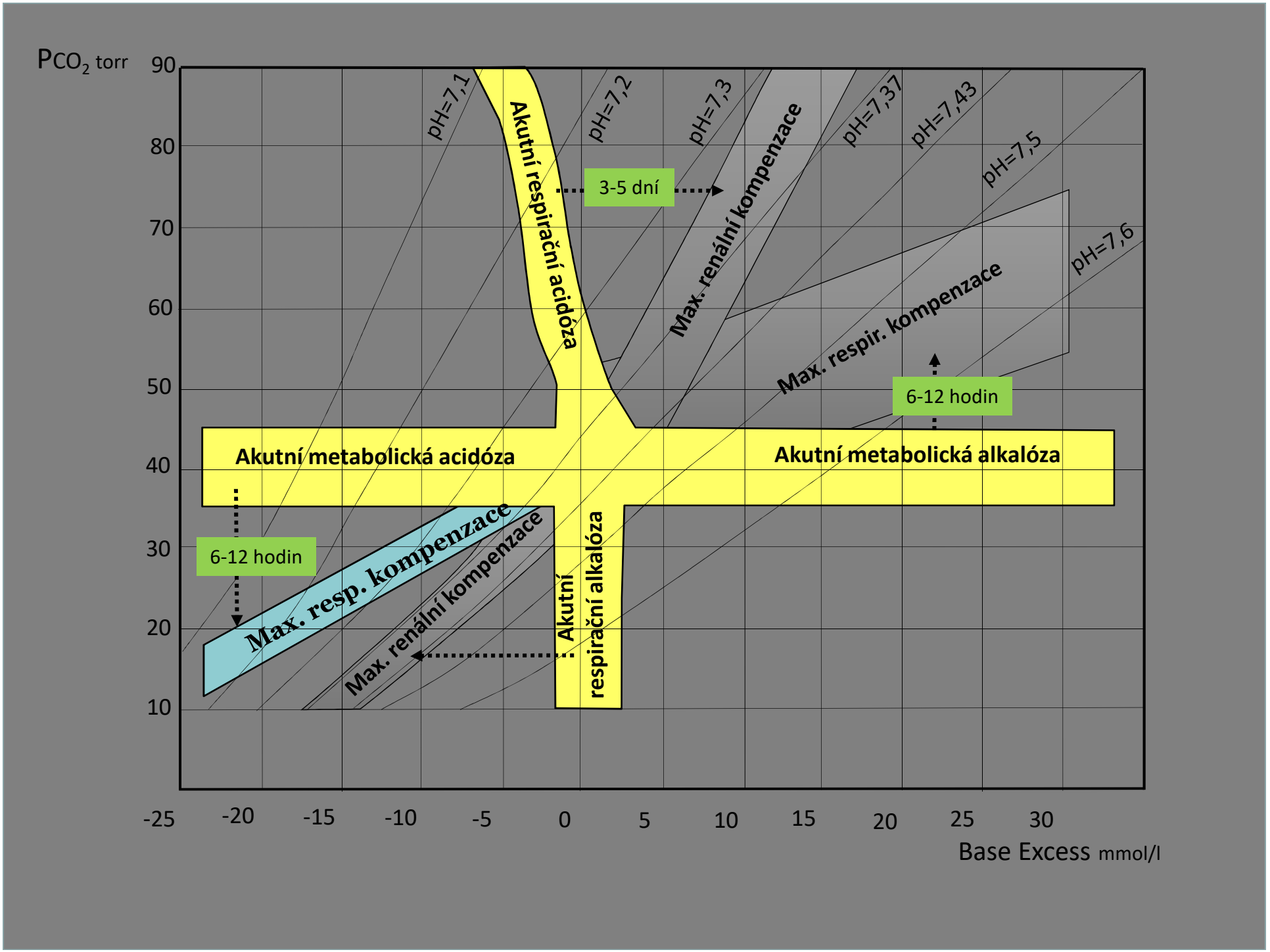
Base Excess -Solution by Siggaard-Andersen

BE = BB - normalBB

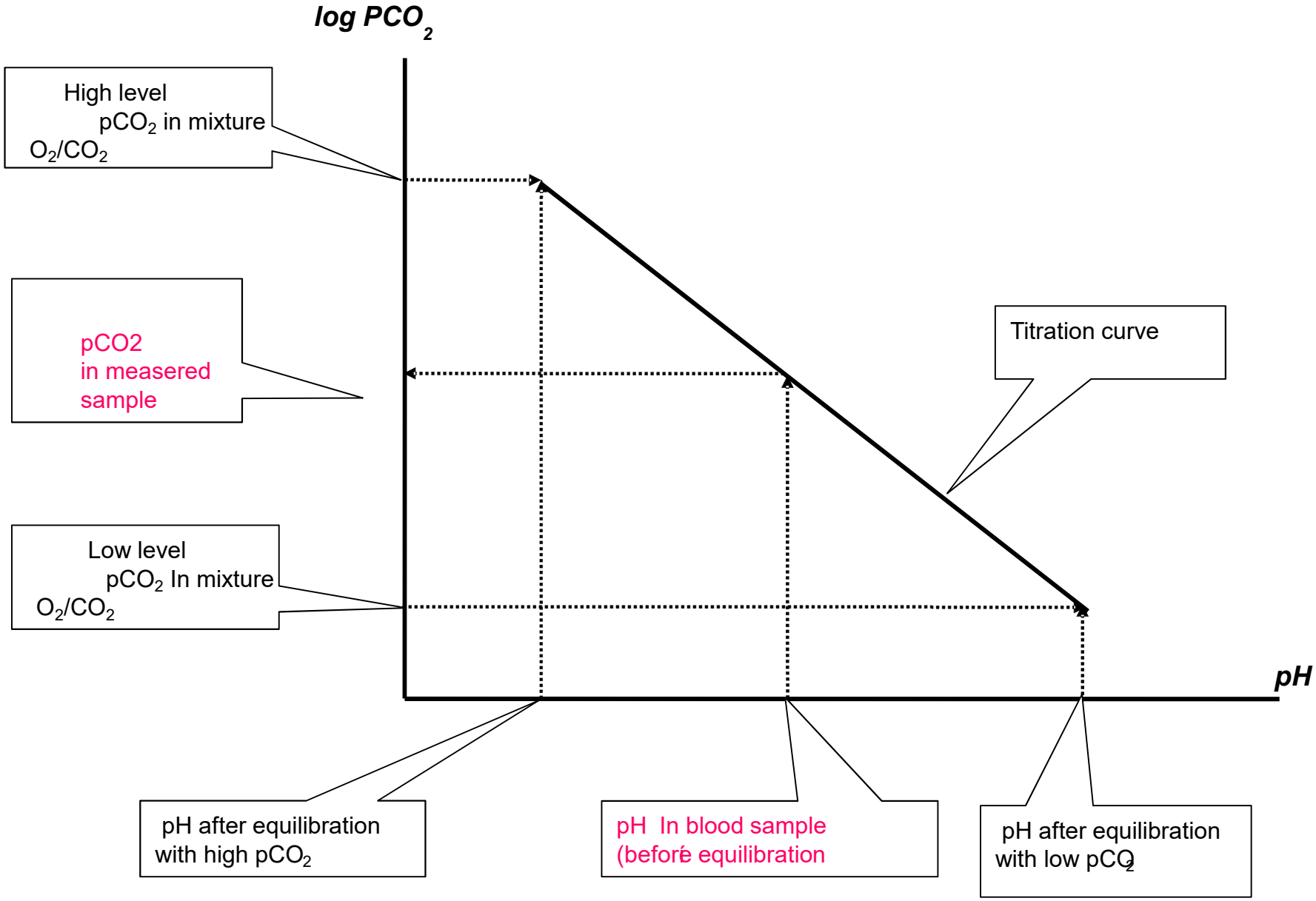
- **Normal BB** is the buffer base that the given **blood would have at pH=7.4** It varies with anemia, polycythemia, Albumin content etc.
- BE then only represents changes in BB due to changes in pH
- **BE** is independent of pCO_2

Compensation of respiratory acidosis

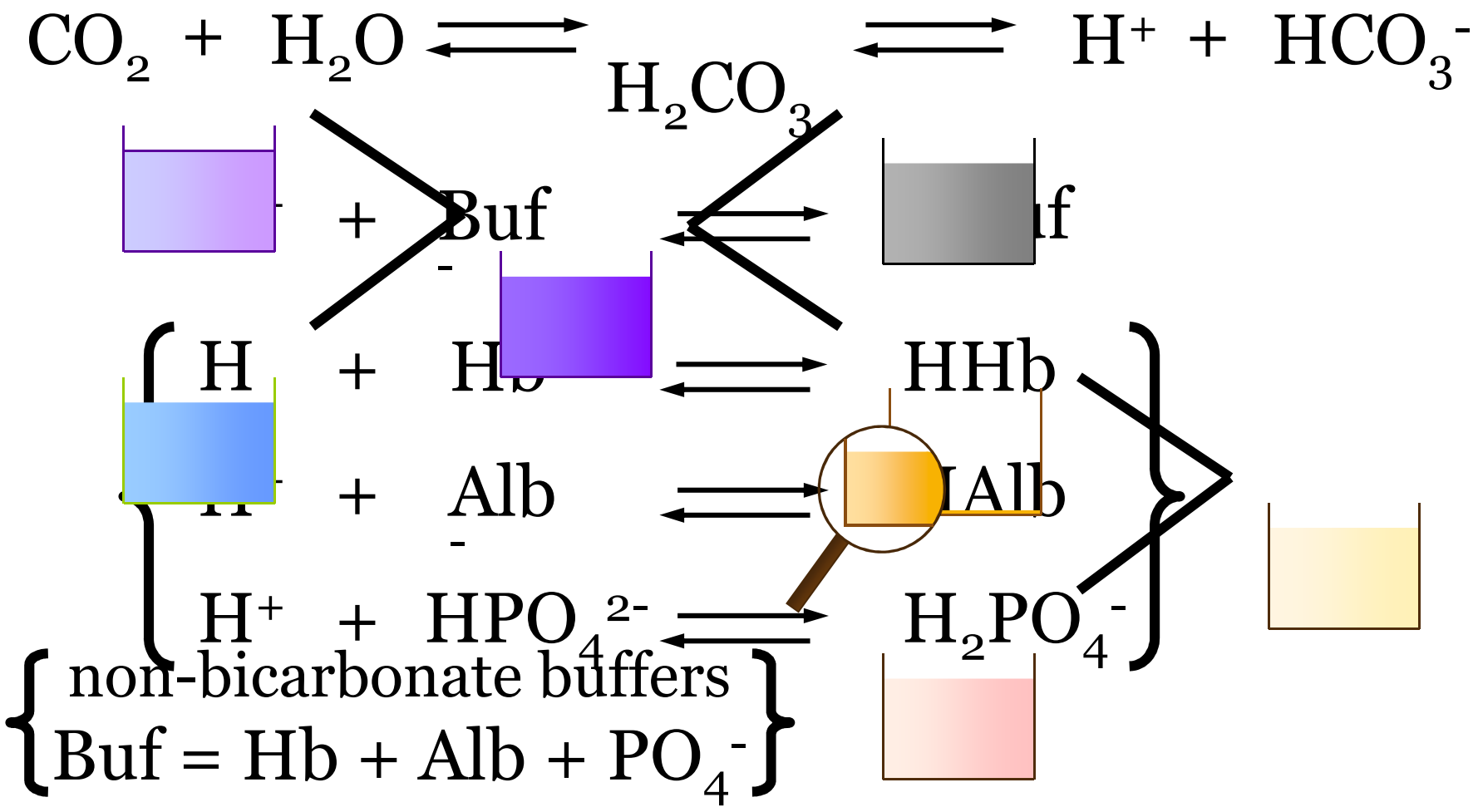



PCO2 torr
90
80
70
60
50
40
30
20
10
-25
-20
-15
-10
-5
0
5
10
15
20
25
30
Base Excess mmol/l
pH=7,1
pH=7,2
pH=7,3
pH=7,37
pH=7,43
pH=7,5
pH=7,6
Akutní respirační acidóza
Max. renální kompenzace
3-5 dní
Max. respir. kompenzace
6-12 hodin
Akutní metabolická acidóza
Akutní metabolická alkalóza
6-12 hodin
Max. resp. kompenzace
Max. renální kompenzace
Akutní respirační alkalóza

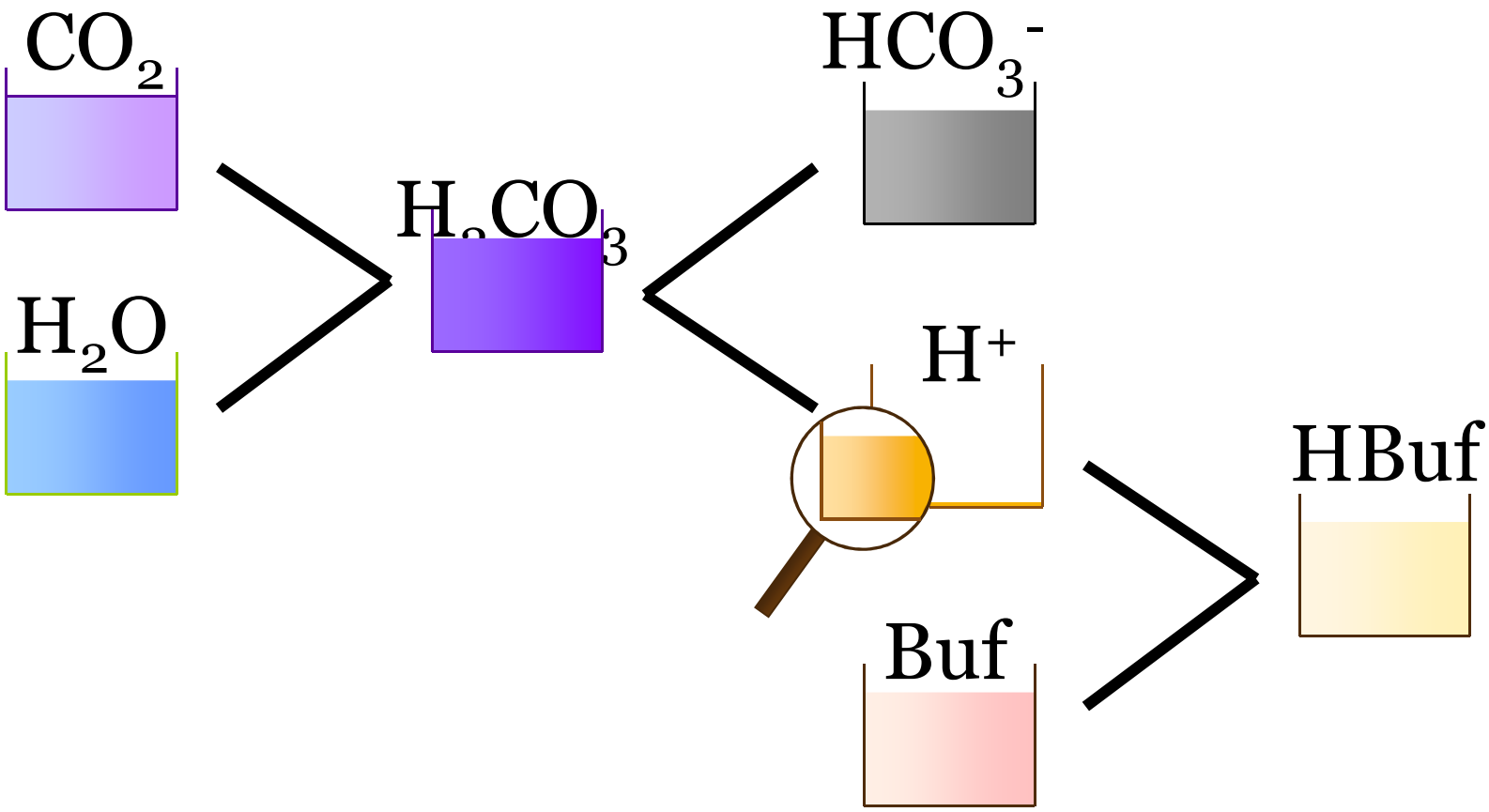
Equilibration method for pCO2 measurement by Astrup



Buffering systems of the blood



Buffering reactions



Bicarbonate buffer

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

Hendersson- Hasselbalch equation:

$[H^+] = 24 \cdot pCO2 / [HCO_3^-]$

or

$$pH = 6.1 + \log ([HCO_3^-] / [H_2CO_3])$$

$$= 6.1 + \log ([HCO_3^-] / 0.03\ pCO_2)$$

Basic division of acid-base disturbances

- **Respiratory acidosis**

  ↑ pCO_2 - alveolar hypoventilation

- **Respiratory alkalosis**

  ↓pCO_2 - alveolar hyperventilation

- **Metabolic acidosis**

  ↓ HCO_3^-

- **Metabolic alcalosis**

  ↑ HCO_3^-

Metabolic compensation of respiratory disorder

- Is carried out by **kidneys** that **increase** plasma concentration of **bicarbonate in resp. acidosis**., Kidneys also decrease bicarbonate reabsorption and their concentration in plasma in resp. alkalosis.
- It takes about **2.5 days** to fully develop

Respiratory compensation of metabolic disorder

- In **metabolic acidosis**, **lungs eliminate more pCO2** by deeper and faster breathing. This is called **Kussmaul breathing.**
- The respiratory compensation of the metabolic alkalosis is limited, because slower and more shallow breathing is limited by hypoxemia.
- Full compensation takes about **½ day** to develop.

Measure of metabolic disturbances

- Only bicarbonate buffer => Change in pCO2 causes no change in HCO_3^-
- Other buffers present => **Increase** in **pCO2** causes an **increase** in **HCO_3^-** as well, but at the same time **decrease** in **Buf^-** (non-bicarbonate buffers) =>

  BB (buffer base) = HCO_3^- + Buf^- stays constant
- Two possible approaches to a measure of metabolic disturbances :
  - USA: **Standard Bicarbonates -** measure of metabolic disturbances independent of pCO2
  - EU: **Base Excess** – measure derived from BB, thus independent of pCO2

Base excess

- At **pH = 7.4** different bloods can have different total BB (depending on Hb, albumines, phosphates), but **BE = 0**
- Amount of BB a sample has at pH 7.4 is called its normal buffer base (nBB)
- BE = BB – nBB
- If we add to all these samples 10 mmol/l of acid, the BB of each sample will decrease by 10 mmol/l. **Now** each **BE = - 10**

Causes of metabolic acidosis

- Extensive desequilibrating load on buffering system
  - Loss of bicarbonate from extensive buffering of acids
    - Ketoacidosis
      - Diabetic
      - Alcohol
      - Starving
    - Lactic Acidosis
    - Toxic substances
      - Salicylates
      - Ethylen glycol
      - methanol
  - Loss of bicarbonate by GIT
    - By diarrhea
    - By fistula and stomia
- Loss of kidney regulation
  - Renal tubular acidoses
  - Kidney failure

Causes of respiratory acidosis

- ↓ alveolar ventilation
  - Respiratory center depression
    - Drugs, medicaments
    - Respiratory centre hypoxia or damage
      - Trauma
      - Stroke
      - Tumor
      - Cerebral edema / increased intracranial pressure
  - Nerve of muscle disease
    - Myasthenia gravis
    - Polyradiculoneuritis
    - Serious obesity
  - Lung disease
    - Restrictive
      - ARDS
      - Fibroses
    - Trauma, pneumothorax, serial rib fractures
    - Obstructive
      - Astma
      - Tumor
      - Foreign body
    - Increase in dead space
      - Embolism
      - Emphysema
- Breathing CO_2 in the inspired air

Causes of respiratory alkalosis

- Hyperventilation
  - in mechanical ventilation
  - With hypoxemia
    - High altitude disease
    - Right-left shunting
- Respiratory centre irritation
  - Trauma, salicylates, inflammation

Causes of metabolic alkaloses

- Vomiting – losses of hydrochloric acid
- Hyper-aldosteronism
- Liver failure and insufficiency
- Kidney disorders
- Incorrect infusion therapy

Case History I

- 58 year old male comes to your ambulance.
- Chronic bronchitis and pulmonary emphysema
- His Lab. tests:
  - pH 7.3
  - pO_2 60 mmHg
  - pCO_2 80 mmHg
  - HCO_3^- 38 mmol/ l

Case History II

- 20 year old student is admitted to hospital for **acute anxiety** state
- Cannot concentrate, feeling of numbness or pins and needles in fingers
- She has split with her boyfriend recently
- Has not been seriously ill until now, no medication
- Physical examination – normal
- Lab. values:
  - pH 7,49
  - pO_2 100 mm Hg
  - pCO_2 30 mm Hg
  - HCO_3^- 22 mmol/l

Case History III

- 38 yo female, DM 1st type
- Chills and fever lasting several days
- She has not felt well => not eaten much and not taken much insulin
- During admission day: Abdominal cramps, vomited several times
- Physical exam: BF 30 min^{-1}, HF 112 min^{-1}, BP 110/70 lying and 100/60 standing, 37 °C,
- Dry mucosae and fruity breath odor

- Lab:
  - pH 7,20
  - pO2 96 mm Hg
  - pCO2 21 mm Hg
  - HCO3- 8 mmol/l
  - Glc 15 mmol/l
  - Na+ 148 mmol/l
  - K+ 5,5 mmol/l
  - Cl 110 mmol/l
  - Positive aceton in urine

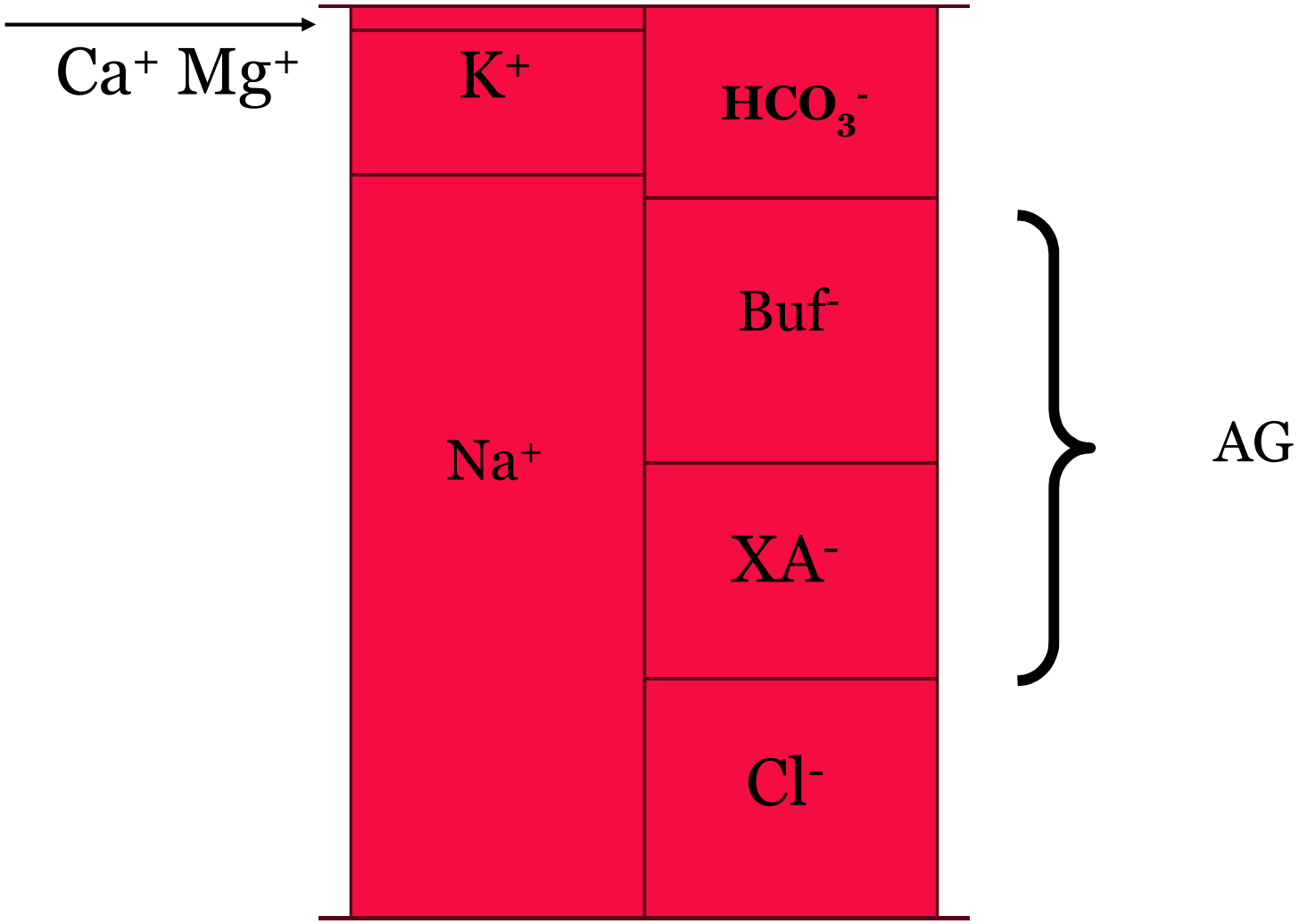
Case history IV

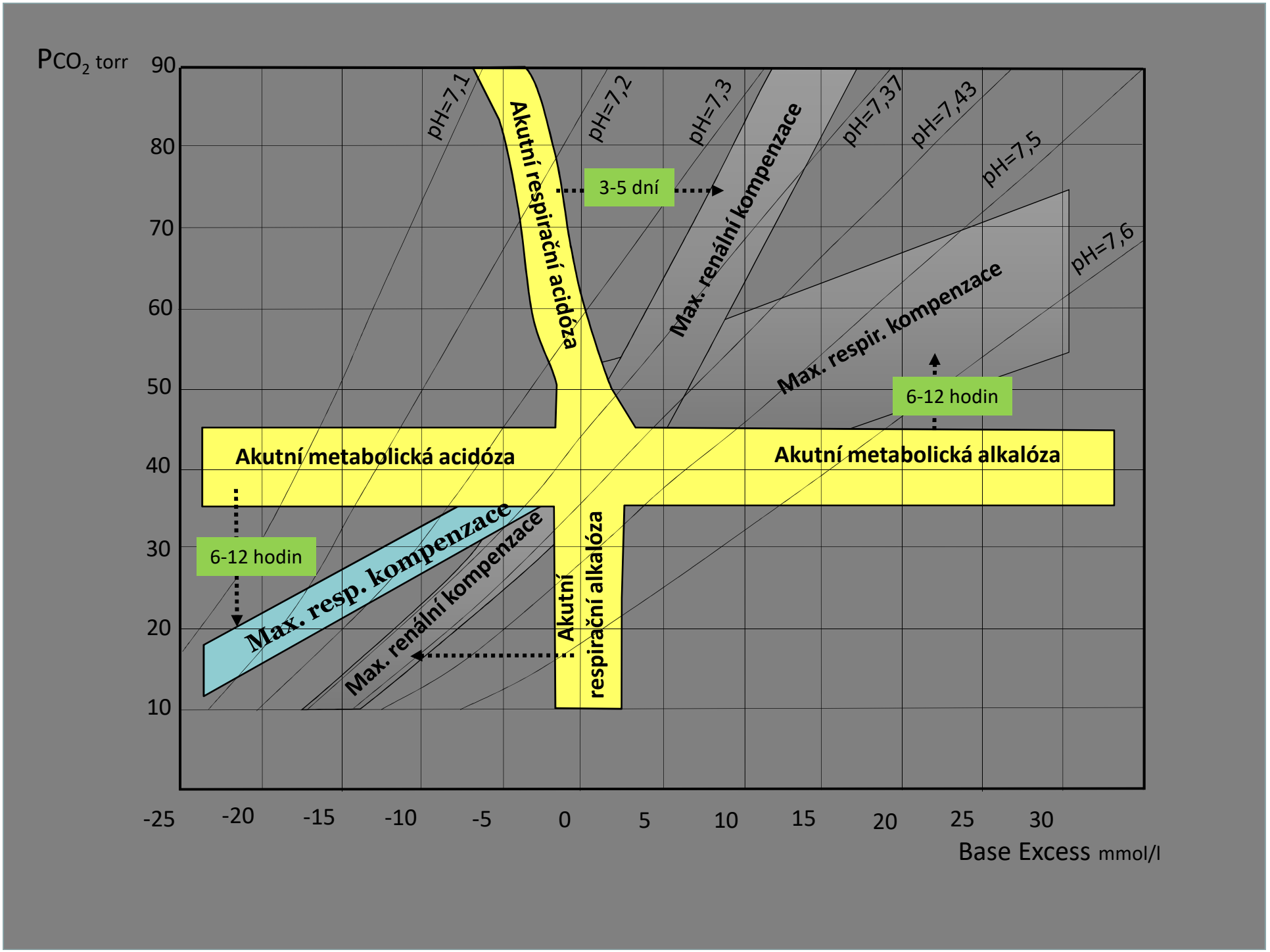
- 25 yo male, admitted for suicide attempt
- Has ingested large amount of aspirin
- At admission somnolent, difficult to make contact with
- BF 30 min^{-1} , HF 100 min^{-1}, BP 142/88, t = 36.8 °C
- Lab:
  - Toxic levels of salicylates,
  - pH 7.25
  - pCO2 14 mmHg
  - HCO_3^- 8 mmol/l

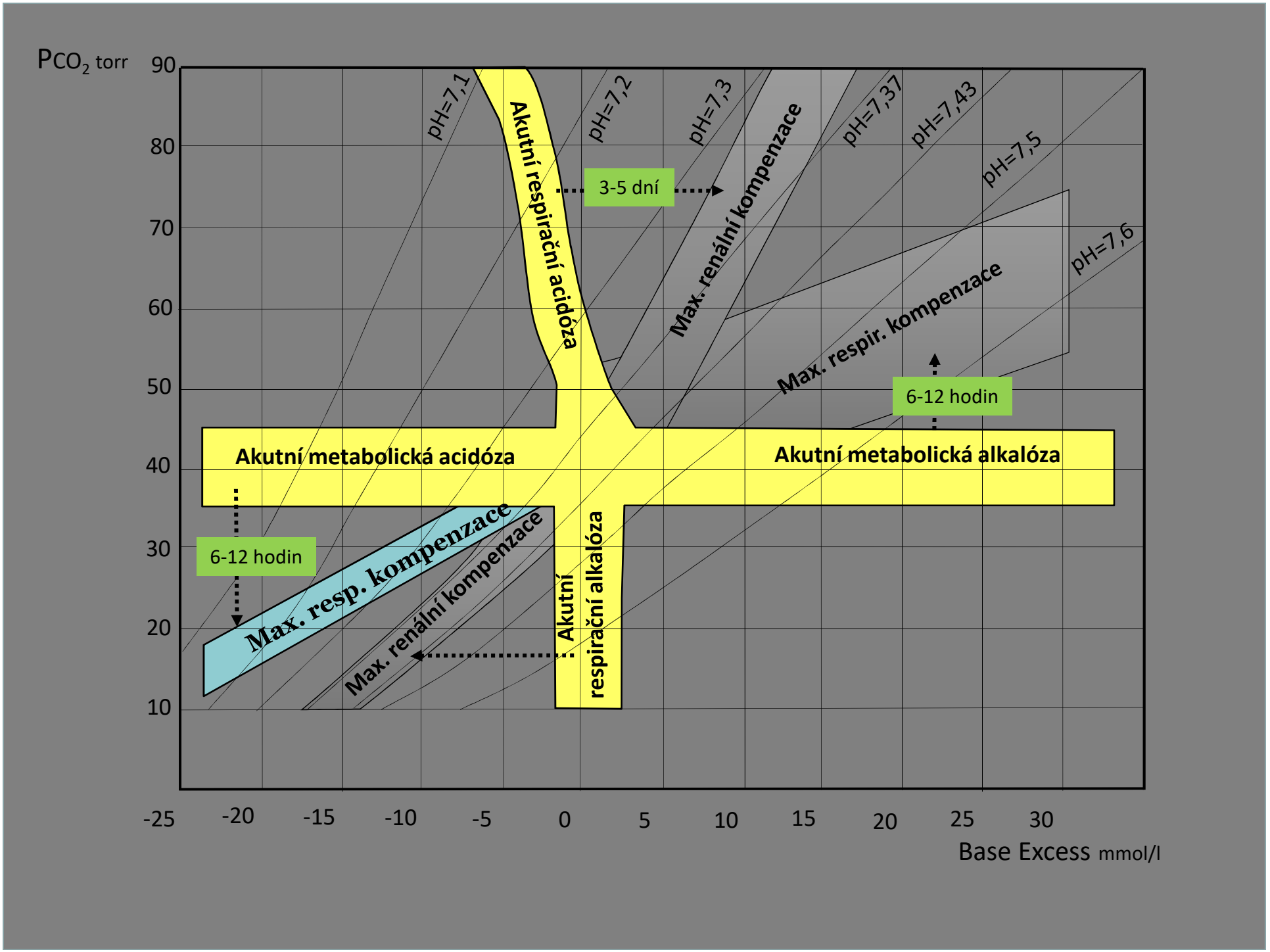
Anion gap

- Rozděluje metabolické acidózy podle příčiny
- **Zvyšuje** se, když jsou v plazmě přítomny ionty jako **laktát, anionty ketokyselin** nebo **sulfáty.**
- Znamená, že acidóza byla způsobena disociací vodíkového iontu z těchto látek
- AG = Na^+ - HCO_3^- - Cl^-
- norma: **10+/- 2** mmol/L

Anion gap




PCO2 torr
90
80
70
60
50
40
30
20
10
-25
-20
-15
-10
-5
0
5
10
15
20
25
30
Base Excess mmol/l
pH=7,1
pH=7,2
pH=7,3
pH=7,37
pH=7,43
pH=7,5
pH=7,6
Akutní respirační acidóza
Max. renální kompenzace
3-5 dní
Max. respir. kompenzace
6-12 hodin
Akutní metabolická acidóza
Akutní metabolická alkalóza
6-12 hodin
Max. resp. kompenzace
Max. renální kompenzace
Akutní respirační alkalóza


PCO2 torr
90
80
70
60
50
40
30
20
10
-25
-20
-15
-10
-5
0
5
10
15
20
25
30
Base Excess mmol/l
pH=7,1
pH=7,2
pH=7,3
pH=7,37
pH=7,43
pH=7,5
pH=7,6
Akutní respirační acidóza
Max. renální kompenzace
3-5 dní
Max. respir. kompenzace
6-12 hodin
Akutní metabolická acidóza
Akutní metabolická alkalóza
6-12 hodin
Max. resp. kompenzace
Max. renální kompenzace
Akutní respirační alkalóza