Keseimbangan asam basa dan elektrolit

Ninuk Dian Kurniawati
Asam dan basa

- Asam: substansi yang menyumbang ion H+, e.g. HCL & H2CO3
- Basa: substansi yang menurunkan konsentrasi ion H+ dalam larutan, e.g. NaOH
Asam dalam tubuh

- Yang bisa menguap: CO2
- Yang tidak bisa menguap: asam nukleat, DNA, RNA, asam laktat, asam urat, asam keton, asam fosfor, asam sulfur.
H ions produced in the body

3 sources steadily add H ions to body fluids:
1. Carbonic acid is formed from metabolically produced CO2. Cellular oxidation of nutrients yields energy with CO2 and H2O as end products.
2. Inorganic acids produced from the breakdown of nutrients. E.g. Sulphuric acid and phosphoric acid
3. Organic acids resulting from normal intermediate metabolism: e.g. Lactic acid partially dissociate
Lactate production

In Kreb’s cycle:

- Aerobic metabolism
- Anaerobic metabolism
  - Less efficient glycolysis and decreased production of ATP
  - Pyruvate and lactate are produced
  - Metabolic acidosis
- Without O2 and in the presence of H+, pyruvate can convert to lactate
Keseimbangan asam basa

Keseimbangan pengaturan konsentrasi ion hidrogen bebas di dalam cairan tubuh PH darah normal = 7,35 – 7,45
Penyimpangan dari PH normal bisa menyebabkan gangguan fungsi:
• Basic cellular functions
• Activity of critical enzymes
• Muscle contraction
• Electrolyte balance
• Quantifikasi keasaman
• H+ Dinyatakan dalam satuan PH
• Henderson-Hasselbach Equation
  Expresses the relationship between pH and the action as CO2 and HCO3- as a buffer pair
Henderson-Hasselbach equation

\[ \text{pH} = 6.1 + \log \frac{[\text{HCO}_3^-]}{[\text{CO}_2]} \]  
(6.1 is the pK of H\(^+\))
Defense against acid base imbalance

- Chemical buffer system
  - First line of defense
  - Active immediately to minimise pH change
  - Doesn't eliminate H+ from the body
  - Limited capacity
- Respiratory system
- Renal system
Chemical buffer system

Protein buffer system
- Most plentiful in the body
- Can give up or take away a H+
- Mostly works in the ICF where intracellular proteins are most plentiful

Haemoglobin buffer system
- Buffers H+ generated by metabolically produced CO2 so it doesn’t contribute to the acidity of body fluids
- Venous blood only slightly more acidity than arterial
Chemical buffer system

- Phosphate buffer system
  - Urinary buffer and ICF buffer
- Excess PO4 consumed is filtered through the kidneys. It buffers urine as it is being formed by removing the H ion secreted into tubular fluid
The Respiratory System

- The respiratory system has the ability to alter ventilation, thereby regulating $[\text{H}^+]$ by controlling CO2 elimination
- Second line of defence
Respiratory system response

- [H+] from non respiratory cause
  - Stimulation of brain stem
    - Pulmonary ventilation
      - CO2 removal
        - formation of H2CO3
          - Restoring [H+] to normal

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Respiratory system

- In changes of [H+] resulting from [CO2] alterations from respiratory disease, the respiratory system cannot contribute to pH control
  - Buffer systems and renal system must correct respiratory induced acid base disorders
Respiratory system

- Can’t return pH to normal by itself
- In response to pH,
  - Peripheral chemoreceptors ventilation in response to pH
  - Central chemoreceptors ventilation in response to in [CO2]
- Problem if acidosis due to a metabolic cause
  - Peripheral chemoreceptors stimulate the resp centre in response to pH causing CO2 to be blown off
  - Central chemoreceptors detect fall in CO2 and inhibit the respiratory centre
- Opposing action prevents full compensation
Role of Kidneys

- Third line of defense against acid base imbalance
- The kidneys adjust the rate of H+ excretion in response to plasma [CO2] or [H+]
- Eliminates H+ derived from sulphuric, phosphoric, lactic, carbonic and other acids from the body
- The kidneys control the pH of body fluids by altering
  - H+ excretion
  - HCO3- excretion
  - Ammonia (NH3) secretion
Renal H+ secretion

- Most H+ is actively secreted into the tubular lumen
- The H+ secretory process begins as
  - CO2 diffuses into the tubular cells from plasma, tubular fluid or CO2 produced from within the tubular cells
- In the presence of carbonic anhydrase, CO2 and H2O form H2CO3 which disassociates into HCO3− and H+
- H+ are then transported into the tubular lumen, while Na+ is transported back into the cell in exchange
Secretion of H+

- Kidneys can only secrete H+ they can’t reabsorb H+
- Factors that affect the rate of H+ secretion:
  - [H+] of plasma passing through peritubular capillaries
  - [H+] of plasma passing through peritubular capillaries
Pengendalian kecepatan pengeluaran H+ oleh tubular ginjal
Pengaturan konsentrasi HCO3-dalam plasma

2 interrelated mechanisms:

- Variable reabsorption of filtered HCO3 & back into the plasma
- Variable addition of new HCO3 & to the plasma
Pengaturan HCO₃⁻ pengeluaran H⁺ akan diikuti oleh reabsorbsi HCO₃⁻
Respons ginjal terhadap keseimbangan asam basa

Acidosis
↓ HCO₃⁻ filtered
↑ plasma [H⁺]
↑ H⁺ secretion

When all the HCO₃⁻ has been “reabsorbed” secreted H⁺ is excreted in the urine
The addition of new HCO₃⁻ into the plasma

Alkalosis
↑ HCO₃⁻ filtered
↓ plasma [H⁺]
↓ H⁺ secretion

HCO₃⁻ is excreted in the urine
Reduced plasma [HCO₃⁻ ] and alkaline urine
Produksi amonia

- The kidney secrete ammonia in acidotic states to buffer secreted H+.
- In acidotic states there is a point that H+ gradient is too big for it to be secreted into the tubular lumen.
- The kidneys can’t acidify urine beyond a certain point and H+ can’t be left unbuffered.
- Filtered phosphate or secreted ammonia will buffer H+.
Summary points

- Buffer systems and renal system must correct respiratory induced acid base disorders
- Normally there is a ratio between \([\text{HCO}_3^-]\) and \([\text{CO}_2]\) in the ECF
- Chemical buffer systems don't eliminate \(\text{H}^+\) from the body
- The respiratory system has the ability to alter ventilation, thereby regulating \([\text{H}^+]\) by controlling \(\text{CO}_2\) elimination
- The kidneys control \(\text{H}^+\) secretion and can generated new \(\text{HCO}_3^-\)
BGA

- Help to differentiate and assess
  - Acid base balance
  - Primary metabolic abnormalities
  - Oxygenation status
  - Ventilation status
BGA

- **PaO2** – Partial pressure of O2 dissolved in the blood
- **pH** – Measurement of acidity or alkalinity
- **PaCO2** – Partial pressure of CO2 dissolved in the blood
- **HCO3⁻** – Concentration of bicarbonate ions in the blood
- **Base Excess (BE)** – Calculated parameter that reflects the metabolic component of acid base disorders only -
Respiratory acidosis

- Ratio of HCO3- to CO2 less than 20:1
- pH < 7.35
- CO2 > 45 mmHg
- HCO3-
  - Acute 22-26 mmol/L
  - Chronic > 26 mmol/L Lung disease
- Late respiratory failure
- Depression of the respiratory centre
- Inadequate mechanical ventilation
**Respiratory acidosis**

Hypoventilation

↑ in CO2

CO2 combines with H2O to produce H2CO3

H+ and HCO3 produced, but more net H+

Chemical buffers respond immediately to absorb additional H+

Respiratory mechanism cannot respond

Kidney detect rise in [H+] 

Kidney conserve filtered HCO3& and add new HCO3&

H+ secreted and excreted in the urine

↑ pH
Respiratory Alkalosis

- Ratio of HCO₃⁻ to CO₂ greater than 20:1
- pH > 7.45
- CO₂ < 35 mmHg
- HCO₃⁻: Acute 22-26 mmol/L, Chronic <22 mmol/L
- Fever
- Anxiety
- Pain
- Aspirin poisoning
- Over mechanical ventilation
- Early respiratory failure: Hypoxaemia
Respiratory alkalosis

- Hyperventilation
  - Decrease in CO2
  - H2CO3 produced
  - Chemical buffers respond immediately to liberate additional H+

- Respiratory mechanism cannot respond
- Kidney detect fall in [H+]
- Kidney excretes HCO3−
- H+ conserved
- pH
Asidosis metabolik

- Failure of kidney function/ decreased availability of renal HCO3-
- Ratio of HCO3- to CO2 is less than 20:1
- pH < 7.35
- HCO3- < 22 mmol/L
- Expect CO2 < 35 mmHg

Causes:
- Severe diarrhea
- DKA
- Lactic acidosis
- Renal failure
Asidosis metabolik

\[ \text{HCO}_3^- \uparrow \text{of non-carbonic acids} \]

\[ \text{Buffers absorb extra H}^+ \]

\[ \text{Central chemoreceptors in the medulla detect an increase in } \text{[H}^+\text{]} \]

\[ \text{Ventilation increases and CO}_2 \text{ is blown off} \]

\[ \text{Kidney detect rise in } \text{[H}^+\text{]} \]

\[ \text{Kidney conserve filtered HCO}_3^- \text{ and add new HCO}_3^- \]

\[ \text{H}^+ \text{ secreted and excreted in the urine} \]

\[ \text{pH} \uparrow \]
Alkalosis metabolik

- Increase in plasma HCO3-
- Ratio of HCO3- to CO2 greater than 20:1
- pH > 7.45
- HCO3- > 26 mmol/L
- Expect CO2 > 35 mmHg or unchanged
Metabolik alkalosis

- Vomiting
- Nasogastric suction
- Ingestion of alkaline drugs
- Contraction alkalosis
- Bicarbonate administration
- Renal loss of H+
- Potassium depletion
Metabolik alkalosis

↑ in HCO3& or ↓ in non-carbonic acids

↓

Buffers liberate H+

↓

Central chemoreceptors in the medulla detect an increase in [H+]

↓

Ventilation decreases and CO2 is retained

↓

Kidney detect a fall in [H+]

↓

Kidney excretes filtered HCO3&

↓

H+ retained

↓

↓ pH
A 66-year-old woman is admitted to CCU following an AMI (V1-V5 ST elevation, Q waves in V2 – V4). Four hours later, she develops respiratory distress and demonstrates crackles half way up each lung field. The doctor made a presumptive diagnosis of cardiogenic pulmonary oedema.

Room air arterial blood gas values were:
- pH 7.10
- PCO2 25 mm Hg
- PO2 40 mm Hg
- HCO3 8 mmol/L
- O2Sat 79%
- BE -20 mmol/L

Vital signs were BP 60/? mm Hg, Pulse 140/min, thready, RR 40/min

What is your interpretation of the ABG?
Contoh

Patient A

- pH 7.11
- PCO2 46.6 mm Hg
- PO2 75.3 mm Hg
- HCO3 14.7 mmol/L
- BE -14.1 mmol/L
- O2Sat 92.6 %
Contoh

Patient B

- pH 7.49
- PCO2 32 mm Hg
- PO2 69.5 mm Hg
- HCO3 23.9 mmol/L
- BE 1.4 mmol/L
- O2Sat 95.3 %
Cairan dan elektrolit

Fluid regulation

- All exchanges between the intra cellular fluids and the external environment must occur through the extra cellular fluid.
- Plasma is the only fluid that we can act directly on to control its volume and composition.
- If the volume and composition of plasma is regulated then the interstitial fluid is also regulated which then influences the intracellular fluid.
Fluid Resuscitation

• Aim to restore intravascular volume, normalize tissue perfusion & avoid complications
• Effective expansion of IV volume can be achieved provided the correct fluid is used at an appropriate rate, internal fluid shifts are taken into consideration and the patient is closely monitored
• Rate of fluid administration may be limited by CVP>15 or PCWP>18
Regulation of fluid osmolarity and sodium concentration.

- The body's water is primarily determined by
  - Fluid intake
  - Renal excretion
Anti diuretic hormone

• The release of ADH is in response to an
  – increased plasma osmolarity.
• Stimulates osmoreceptors in hypothamus
  – Decreased Intravascular volume,
    Decreased blood pressure.
• Low pressure Baroreceptors R atria and carotid
Conservation of water

• Impulse sent to the posterior pituitary gland
• Secretes more antidiuretic hormone (ADH) into the blood stream.
• This increases the permeability of the distal tubules and collecting ducts to water
• More water is reabsorbed
• Solutes are excreted at normal rate
• Urine output decreases
• Urine is more concentrated
• Increases volume and decreases osmolarity of the intravascular fluid.
Cardiovascular control of ADH

• Atrial stretch reflex
  – If atria are over stretched causes decrease in the release ADH.
Elimination of excess water

- Excess fluid low serum osmolarity
- Decreased amount of ADH secreted
- Decreased amount of water reabsorbed
- Solute reabsorbed
- Can excrete 20/l per day
Thirst

- Sensation of thirst is also an important factor in maintaining fluid balance.
- Thirst is the physiological urge to drink water.
- Trigger when water loss equals 2% of body weight.
- Stimulus to thirst are
  - Cellular dehydration, via osmoreceptor
  - Hypovolemia and Hypotension baroreceptors
  - Angiotension II released in response to hypotension.
- Sensation turned off by mechanoreceptors in mouth and pharynx