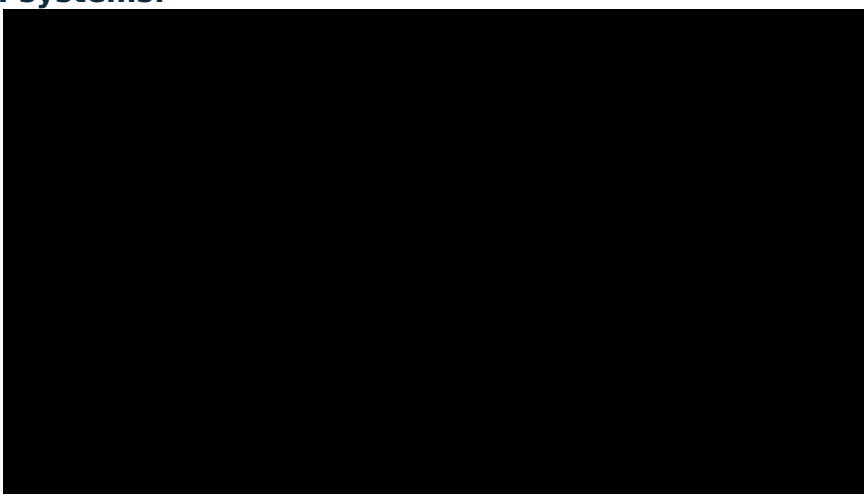
Acid-Base balance

Acid-base balance (ABB) is a **dynamic balance between acidic and basic substances within the organism**, the balance between their formation and excretion.

ABB Intro:

Handerson-Hasselbach equation:

Compensatory pH systems:



The acidity of the indoor environment **must be very precisely regulated**. Physiologically, t**he pH of blood** and many other body fluids is kept in a very narrow range of values **around 7.40 (from 7.36 to 7.44)**[1], so the

fluctuations are almost negligible. Such precise regulation is important because changes in pH change the properties of proteins, including enzyme activity, transport mechanisms, membrane channel properties, etc.

Larger pH deviation necessarily disturbs the regulation of a large number of metabolic pathways and physiological processes and gradually leads to general metabolic disruption.

ABB is maintained by means of so-called **buffers**, which compensate for immediate fluctuations in ABB, and by means of the lungs, kidneys and liver, which enable long-term compensation of ABB disorders.

The imbalance in favor of acids is called **acidosis**, and in favor of alkalis is called **alkalosis**. These disorders **can be caused by** *metabolic* disorders, then we talk about *metabolic acidosis* or *alkalosis*, or *respiratory* disorders, then we talk about *respiratory acidosis* or *alkalosis*.

The body tries to deal with ABB disorders and minimize pH deviation; therefore, we often encounter a situation where, despite the ABB disorder, the pH of the blood remains within the reference limits or deviates only minimally from them. Therefore, if the pH of the internal environment deviates (the concentration of hydrogen ions changes), we speak of *acidemia* or *alkalemia*.

Terms *acidosis* and *alkalosis* refer to the retention or depletion of strong acids. In other words, not every acidosis is accompanied by acidemia, but the body makes some effort to maintain pH and the effort to compensate may be at the expense of over-regulation of some physiological processes. They characterize the pH value of plasma.

ABB maintenance mechanisms

ABB maintenance mechanisms

ABB laboratory tests

Examination of blood gases and internal environment according to Astrup, more recently examination of acid-base regulation provides information on:

- blood pH ;
- oxygen partial pressure (pO 2);
- carbon dioxide partial pressure (pCO 2);
- percentage of oxygenated blood in the arteries (sO 2).

It is also possible to examine individual hemoglobin fractions (oxyhemoglobin, methemoglobin...) and calculate other values of acid-base balance. Arterial, arterialized capillary or central venous blood is used for the examination. The sample must be taken anaerobically.

Indication

Examination of blood gases and acid-base balance is one of the basic methods for ventilation and respiratory disorders (eg COPD, bronchial asthma, heart defects, severe lung diseases, heart disease), internal environmental disorders (kidney and liver diseases, some poisonings, intensive infusion therapy, drug-induced internal environment disorders), etc.

Preanalytical phase

The most suitable sample is arterial blood. It is most often taken from the radial artery into a capillary on a thin needle or into a modified syringe, lithium heparin is used as an anticoagulant. An arterial catheter is often introduced in intensive care units, which allows for repeated donations. In any case, it must be ensured that the sample is taken without air bubbles.

Another option is to take arterialized capillary blood, most often from the fingertip or earlobe. The composition of the capillary sample should correspond as closely as possible to the arterial blood. It is therefore necessary to increase the blood flow through the capillaries at the place from which the collection is performed ("arterialization") - by warming, massage, etc. Capillary blood collection is performed into the heparinized capillary, the sample must again be free of bubbles.

When examining venous blood, the sample should be taken from the central venous bed (central venous catheter, port). Peripheral venous blood does not provide sufficient information about the overall metabolic state of the body, especially in patients with severe centralized circulation. Central venous blood is collected in a balanced lithium heparin syringe, in which case the collection must be anaerobic.

The sample should be processed within 15 minutes of collection. It is necessary to indicate the type of consumption in the request.

Analytical phase

The material is processed using automatic analyzers. The following parameters are measured:

Current pH
The actual pH is determined electrochemically, usually with a miniaturized glass electrode.
Carbon dioxide partial pressure.

The partial pressure of carbon dioxide (pCO 2) is determined electrochemically by a Severinghaus electrode. It is also a glass electrode, but it is coated with a layer of water and separated from the sample by a gas-permeable membrane. CO 2 from the sample diffuses through a semipermeable membrane into distilled water, the pH of the resulting solution depends on pCO 2.

Oxygen partial pressure

Oxygen partial pressure is measured electrochemically with a Clark oxygen electrode.

Further examination

At the same time, hemoglobin oxygen saturation and hemoglobin concentration are usually measured. Depending on the type of analyzer available, it is possible to determine the concentration of glucose, lactate, sodium, potassium, chlorides, ionized calcium (Ca 2+) in the same sample. For neonatological purposes, fetal hemoglobin and "neonatal" bilirubin can be determined simultaneously.

Calculated parameters

Current and standard bicarbonates, base excess and possibly other parameters are calculated from the measured pH and pCO 2 values.

Significance and interpretation of acid-base balance parameters

pH

Normal values: 7.36-7.44

Deviation of blood pH from the norm is called acidemia or alkalemia.

The resulting pH informs about the severity of the indoor environment disorder and the degree of compensation or correction of any ABB disorder. In the case of compensated and corrected disorders, it is almost always the case that if the primary disorder is acidosis, the actual pH is lower than 7.4, and conversely, if the primary disorder is alkalosis, the actual pH is higher than 7.4.

pCO 2

Normal values: 5.3 ± 0.5 kPa

Informs about the respiratory component of acid-base balance. Hypocapnia accompanies hyperventilation and respiratory alkalosis, hypercapnia, on the other hand, respiratory insufficiency and respiratory acidosis.

Topical bicarbonates

Normal values: 24 ± 2 mmol / l

This parameter indicates the current concentration of bicarbonates in the examined blood. Due to the fact that it depends on the metabolic and respiratory components of acid-base balance, its interpretation is complicated.

Standard bicarbonates

Normal values: 24 ± 2 mmol / l

The calculated parameter expresses what the concentration of bicarbonates in the examined blood sample would be after the exclusion of the respiratory disorder, ie after the blood saturation at pCO 2 = 5.3 kPa. It therefore informs only about the metabolic component of the acid-base balance. Metabolic acidosis is characterized by a decrease in standard bicarbonates, and metabolic alkalosis by their increase.

Base excess **(BE)**

Normal values: 0 ± 2 mmol / l

Another calculated parameter that evaluates only the metabolic component of acid-base balance. It is defined as the amount of strong acid that would need to be added to the test sample to reach a pH of 7.4, provided that the ABB respiratory distress is excluded (ie pCO 2 = 5.3 kPa). In metabolic acidosis, a strong base would need to be added; the corresponding parameter is referred to as base deficiency, *base deficit*, BD, or (more often) is expressed as negative BE.

It is clear from the definition that a negative BE corresponds to metabolic acidosis and a positive BE corresponds to metabolic alkalosis. The parameter is easy to evaluate. In addition, the appropriate composition of infusion solutions for the treatment of the internal environment can be directly calculated from it, especially in metabolic acidoses.

Other ABB parameters

In addition to these values, a number of other calculated parameters are defined: strong ion difference (SID), *anion* gap (AG), serum buffer bases (BBS) and others. These are parameters that describe the relationship between ABB and mineral management. All are defined as the sums and differences of the concentrations of the selected major ions. According to some authors, however, it is more advantageous to

directly evaluate the concentrations of individual components of the mineralogram, because the calculation of these parameters loses information.

Evaluation of ABB examination

In general, the following decision algorithm can be used to evaluate the acid-base balance test:

1. **Is this an ABB disorder?**

   Is any ABB value outside the reference range?
   Can't this be a combined ABB fault (see below)?

2. **Is the primary ABB disorder acidosis or alkalosis?**

   If the actual pH is <7.4 (even within the reference range), it will be acidosis, and conversely, if the pH is> 7.4, the primary disorder will be alkalosis.

3. **Is the primary disorder metabolic or respiratory?**

   Respiratory failure corresponds to pCO 2 deviation, metabolic change of standard bicarbonates and BE. The direction must correspond to the previous point. For example, in the previous step, we determined that the primary disorder is acidosis. If the pCO 2 is > 5.3 kPa, it is respiratory acidosis; if standard bicarbonates are <24 mmol / l and BE is negative, it is metabolic acidosis. It can also be a combination of respiratory and metabolic acidosis, but we do not evaluate deviations in the opposite direction in this step.

4. **Are compensation mechanisms involved?**

   For example, if we have determined that the primary ABB disorder is metabolic acidosis, we expect the body to compensate for it by respiratory alkalosis after a period of time. If pCO 2 is in the reference range, we evaluate the disorder as *acute metabolic acidosis* (no respiratory compensation yet). Hypocapnia may be subacute or chronic metabolic acidosis (see below).

5. **Is the compensation complete?**

   If we determined in the previous step that the ABB compensation deviation is already developing, we evaluate the extent to which it can face the primary failure. If the current blood pH has returned to the reference range, we speak of a *chronic* ABB disorder. If the compensatory mechanisms are obvious, but the pH differs significantly from the norm, we speak of a *subacute* disorder.

Combined ABB disorders

In practice, we often encounter combined ABB disorders. It should be borne in mind that especially the combination of metabolic acidosis and metabolic alkalosis may remain hidden in the evaluation of the ABB test "according to Astrup", or the severity of the disorder may be underestimated (both disorders correct each other). Information about ABB is essential, as most treatments affect one component of the combined disorder more quickly than the other; it can then quickly prevail and the patient can get into a severe disruption of the internal environment in a short time.

Therefore, laboratory tests are never evaluated separately - it should always be related to other laboratory findings, anamnesis and clinical condition. In principle:

- any deviation in the concentration of major ions (Na +, K +, Cl -) causes ABB failure;
- any change in total protein and albumin concentration causes ABB impairment;
- every organ failure (renal insufficiency, more severe hepatopathy, heart failure) is accompanied by ABB.

If we expect an ABB disorder based on the anamnesis, clinical picture or other examinations, but we do not "see" the ABB examination, it is a combined disorder and we must look for further deviations! If we quickly affect one component of a combined ABB disorder, we must carefully monitor the internal environment and expect it to change rapidly.

ABB disorders

Acid -base balance disorders (ABB) are conditions in which:

- The pH of the indoor environment is deviated from the norm (acidemia, alkalemia)

or

- there is an excess or deficiency of acids or bases in the organism, ie there is a change in the composition of the buffers (which may or may not be accompanied by a change in the resulting pH; acidosis, alkalosis).

Bicarbonate buffer is of the utmost importance for rapid pH maintenance. One of its components, (HCO 3 -), has a charge and forms a relatively significant item in the ionogram. Acid-base balance is therefore closely linked to the metabolism of major ions. In practice, any major acid-base imbalance will also be accompanied by a disorder in the

mineralogram. Conversely, more pronounced changes in the ionogram are usually accompanied by an acid-base imbalance. You can find more about the relationship between acid-base balance and ionogram here.

Respiratory disorders of acid-base balance

If ventilation changes, the partial pressure of carbon dioxide in the blood changes, and thus the concentration of bicarbonate buffer conjugate. Specifically:

- hyperventilation accompanied by hypocapnia leads to respiratory alkalosis

and conversely

- hypercapnia caused by a ventilation disorder will result in respiratory acidosis.

Metabolic disorders of acid-base balance

Ionogram ABB metabolic disorders are conditions in which the concentration of bicarbonates changes significantly (more precisely: standard bicarbonates - see Examination of acid-base balance). At the same time, the concentration of one or more major ions always changes, because the bicarbonate anion must be in balance with other ions of body fluids (more in the chapter Relationships between acid-base balance and ionogram).

Metabolic acidosis

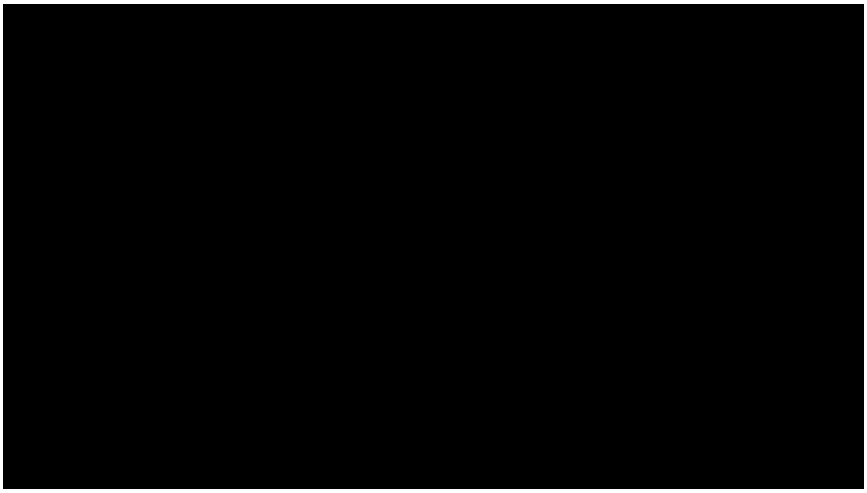
Metabolic acidosis is a condition in which the **concentration of standard bicarbonates falls below reference values**. This can happen:

- due to the accumulation of an anion that "pushes" the bicarbonates out of the mineralogram;
- due to the loss of bicarbonates (accompanied by a cation, most likely as sodium bicarbonate);
- more rarely: due to losses of some cations, most likely sodium, which are compensated by a decrease in bicarbonate concentrations.

MAC:

Acidosis due to ESRD or ARF:

MAC 2:



Metabolic acidosis from anion accumulation

Lactic acidosis
lactic acid in a medium close to 7.4 dissociates almost completely into the lactate anion. Lactate concentration increases significantly, especially in tissue hypoxia.

Ketoacidosis
(in terms of ABR accumulation of β-hydroxybutyrate and acetate). It develops when glucose is not a sufficient source of energy and fats are broken down to an increased extent: during starvation, type 1 diabetes, extreme exercise, etc.

Renal acidosis
in renal failure, sulphates, phosphates and other anions accumulate that would normally be excreted in the urine.

Acidosis in some poisonings

- **ethanol intoxication - ethanol is metabolized to acetate.**

  In addition to acetate overproduction, NADH production plays an important role in ethanol degradation. The high concentration of reducing equivalents inhibits the breakdown of lactate that accumulates. Similarly, NADH inhibits glycolysis, which ultimately leads to stimulation of ketogenesis and accumulation of β-hydroxybutyrate and acetate.

- **methanol intoxication - methanol is metabolized to formate;**
- **ethylene glycol intoxication - metabolized to oxalate.**

Metabolic acidosis from bicarbonate losses

It is most often due to the loss of bicarbonates from the **gastrointestinal tract**. Duodenal and pancreatic juices are rich in bicarbonates, which are supposed to neutralize the digestion coming from the stomach. Normally, bicarbonates are resorbed back in the small intestine. In some GIT diseases (diarrhea, short bowel syndrome), resorption may be so low that blood bicarbonate levels drop significantly.

Renal loss of bicarbonate may be another cause (renal tubular acidosis, side effect of diuretic therapy, etc.). We can also include the so-called **dilution acidosis** in the group of metabolic acidoses from bicarbonate losses. It occurs during rapid infusions. Bicarbonates dilute in the blood faster than can be supplemented by metabolism. The processes that maintain the carbon dioxide partial pressure are much faster, so pCO2 does not change.

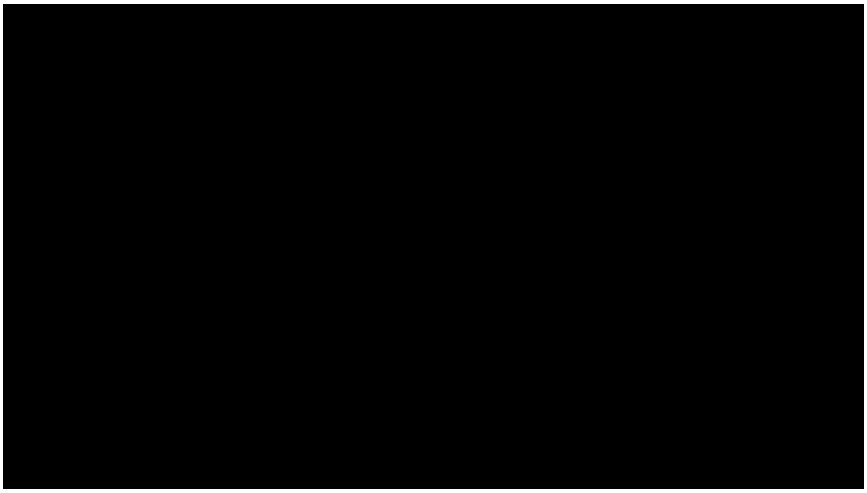
Renal failure

Metabolic acidosis typically develops in renal failure. There are several disorders that affect the acid-base balance in the same direction:

- accumulation of **sulphates,**
- accumulation of **phosphates**,
- **hyperuricaemia** - uric acid behaves like an anion at a pH close to physiological,
- **bicarbonate reabsorption fails** while maintaining diuresis and tubular damage.

Metabolic alkalosis

MAL:



Metabolic alkalosis is characterized by an increase in the concentration of standard bicarbonates. In principle, this may be due to:

- losses of an anion, usually chlorides or proteins, which are compensated in the ionogram by the addition of bicarbonates;
- an increase in the concentration of a cation, most often sodium.

Alkalosis from anion losses

Hypochloraemic alkalosis
: It is accompanied, for example, by prolonged vomiting, in which a large amount of chloride anion is lost through vomiting gastric juice. Diuretics may be another cause of hypochloraemic alkalosis.

Hypoproteinemia
: Proteins behave like polyanions, so the decrease in their concentration is also compensated by the addition of bicarbonates. Typical examples may be liver proteosynthesis failure, protein loss in nephrotic syndrome, or malnutrition.

Hypernatremic alkalosis

It is most often the result of hyperaldosteronism. Some adrenal tumors or other tumors producing this hormone lead to *primary hyperaldosteronism*. *Secondary hyperaldosteronism* is more common as a consequence of liver failure, as aldosterone is broken down in the liver. Another cause of secondary hyperaldosteronism may be overactivation of the renin-angiotensin-aldosterone system.

Elevated aldosterone levels cause the kidneys to retain more sodium, which is compensated in the ionogram by the addition of bicarbonate anion. In addition, sodium is being saved at the expense of increased urinary potassium and proton losses, leading to further deepening of alkalosis.

Sodium retention is also caused by corticosteroids, so metabolic alkalosis is accompanied by Cushing's syndrome.

Alkalosis from an excess of other cations

Rarely, metabolic alkalosis can be caused by an excess of another cation, such as ionized calcium. It occurs, for example, in bone tumors (multiple myeloma, metastases of breast cancer, prostate cancer, etc.). During the breakdown of bone tissue, a large amount of Ca2 + as well as HCO3- is released.

Liver failure

Liver failure is typically accompanied by metabolic alkalosis. Its causes are:

- hypoproteinemia in proteosynthesis failure;
- secondary hyperaldosteronism with sodium retention - aldosterone is normally broken down by the liver;
- slowing down the ureasynthetic cycle - a metabolic process that produces a proton for each molecule of urea formed.

Respiratory acidosis

RAC:

- We distinguish between acute and chronic;
- imbalance between CO 2 production by tissues and its excretion
- we create 13,000-15,000 mmol per day.

Acute respiratory acidosis

- Hypercapnia, always accompanied by hypoxemia and lactic acidosis;
- respiratory distress, restlessness, tachypnoea, dyspnoea, up to stupor and coma .

Causes

- Airway obstruction
- neuromuscular causes (strain damage, respiratory depression, myasthenia, botulism, tetanus ,...);
- respiratory reduction - pneumothorax, hemothorax, pneumonia ,...;
- circulatory disorders - embolism;
- poorly performed artificial respiration.

Chronic respiratory acidosis

- Chronic reduction in effective alveolar ventilation;
- renal compensation occurs - Na + and HCO 3 - retention, Cl - is increasingly excreted;
- erythrocyte count increases, Hb increases;
- hypercapnia initially stimulates the respiratory center, at pCO 2 above 9 kPa - attenuation - as stimulation at that moment is mainly hypoxemia → do not give pure oxygen!!!

Causes

- COPD;
- chronic sedative overdose;
- primary alveolar hypoventilation;
- Pickwick syndrome;
- neuromuscular impairment;
- anatomical deformity of the chest - kyphoscoliosis ,...;
- terminal stages of pulmonary fibrosis.

Category: Chemistry | Physiology | Pathobiochemistry | Pathophysiology | Clinical Biochemistry | Internal Medicine

Respiratory alkalosis

RAL:

Tissue carbon dioxide production is relatively constant. Respiratory alkalosis is therefore mainly caused by **increased excretion of CO_2 lungs**. This reduces the pCO_2 and thus the carbonic acid concentration in the system and deviates the ratio of bicarbonate and carbonic acid concentrations in the Henderson-Hasselbalch equation.

Causes

The cause of respiratory alkalosis is **hyperventilation**, which leads to **hypocapnia***:*

- central **breathing center stimulation**- fear, pain, fever, pregnancy, trauma, head injuries, bleeding into the CNS, mental illness;
- peripheral respiratory center stimulation - pulmonary embolization (minor), congestive heart failure, high altitudes;
- liver failure with hyperammonaemia;
- G^- sepsis;
- heart rhythm disorders;
- partial **respiratory insufficiency**, where efforts to maintain hyperventilation oxygenation lead to hypocapnia.

Links

Related Articles

- Acid-base balance parameters
- Acid-base balance mechanism
- Laboratory examination of acid-base balance
- Acid-base imbalances
  - Metabolic acidosis
  - Metabolic alkalosis
  - Respiratory acidosis
  - Combined acid-base imbalance
- Correction and compensation of acid-base imbalances
- Principles of treatment of acid-base balance disorders
- Relationships between acid-base balance and ionogram

Used literature

- SCHNEIDERKA, Petr, et al. *Kapitoly z klinické biochemie.* 2. vydání. Praha : Karolinum, 2004. ISBN 80-246-0678-X.

Combined ABB disorders

In practice, a combination of several acid-base imbalances can often be encountered. The following types of acid-base disorders can be combined: metabolic acidosis and alkalosis and respiratory acidosis and alkalosis. Individual faults can add up or cancel out. When acidosis is combined with alkalosis, the resulting pH may be normal, but severe ABB may still occur.

The combination of metabolic acidosis with metabolic alkalosis is especially important : in the ABB examination according to Astrup, individual parameters may be normal or only slightly deviated. Therefore, the combined ABB failure may not be recognized or may be underestimated. At the same time, a treatment that affects one of the sub-disorders can cause the other disorder to prevail quickly. This can lead to a sharp change in the pH of the internal environment and severe metabolic breakdown.

Conditions leading to combined ABB disorders are not uncommon. Typical examples are:

vomiting and diarrhea
: vomiting leads to hypochloraemic alkalosis, diarrhea to acidosis due to bicarbonate losses

prolonged vomiting
: hypochloraemic alkalosis in vomiting is combined with fasting ketoacidosis and lactic acidosis due to insufficient tissue perfusion in hypovolemia

hepatorenal failure
: combines hepatic metabolic alkalosis with renal acidosis

liver failure with respiratory insufficiency
: severe hypoproteinemia with liver failure leads to pulmonary edema, hypoxia develops lactic acidosis

renal failure with nephrotic syndrome and severe hypoproteinemia
: renal acidosis from sulfate and phosphate accumulation is accompanied by alkalosis in hypoproteinemia

ABB fault correction and compensation

If ABB fails for any reason, the body begins to strive to maintain the pH of the indoor environment. In essence, ABB struggles with another disorder that deflects the pH in the opposite direction. We distinguish two groups of such mechanisms:

Compensation

It means that in the case of a metabolic disorder, the pH of the internal environment is maintained by changing respiration. For example, metabolic acidosis is compensated by respiratory alkalosis ; the patient will breathe hard deeply (" Kussmaull's breathing ").

Correction

- We talk about correction only in the case of ABB metabolic disorders: one metabolic deviation is corrected by another. E.g. a patient with liver failure (and therefore metabolic alkalosis) will excrete more bicarbonate in the kidneys and less acidify the urine.

The development of correction and compensation mechanisms takes some time. Respiration changes almost immediately after an ABB disorder occurs. Respiratory compensation mechanisms then deepen, reaching a maximum in about 12-24 hours. Compensation and correction at the kidney level are much slower - some transport mechanisms have to be re-regulated, which often requires protein synthesis. These mechanisms reach their maximum in five days.

When arriving at high altitudes, it is necessary to count on about five days of acclimatization. The cause of alpine disease is hyperventilation, which the body tries to deal with hypoxia. However, strenuous breathing does not improve oxygen saturation of hemoglobin too much - the O 2 partial pressure in the surrounding atmosphere is too low for this, but it leads to respiratory alkalosis. It is alkalosis and ionic imbalance that is the cause of alpine disease, including brain swelling, lung swellingand tachycardia. Acclimatization consists in over-regulation of the kidneys - basically in the development of metabolic acidosis, which lasts the mentioned 5 days. It can be accelerated by the intake of large amounts of fluids, as it increases urinary bicarbonate losses. As part of the treatment of alpine disease, the administration of acetazolamide, a carbonic anhydrase inhibitor that reduces urinary acidification, is sometimes recommended (however, recent work considers the administration of acetazolamide to be ineffective).

Sources

Related Articles

- Disorders of acid-base balance
  - Metabolic acidosis
  - Metabolic alkalosis
  - Respiratory acidosis
  - Respiratory alkalosis
  - Combined acid-base disorders
- Correction and compensation of acid-base disorders
- Principles of treatment of acid-base disorders
- Relationships between acid-base balance and ionogram

References

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Original Article - https://www.wikiskripta.eu/index.php?curid=2070

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- SCHNEIDERKA, Petr. *Kapitoly z klinické biochemie.* 2. edition. Karolinum, 2004. ISBN 80-246-0678-X.

- NEČAS, E. *Obecná patologická fyziologie.* 1. edition. Karolinum, 2007. pp. 284–297. ISBN 80-246-0051-X.
- VOKURKA, Martin. *Patofyziologie pro nelékařské směry.* 3. edition. Karolinum, 2013. pp. 103. ISBN 978-80-246-2032-9.