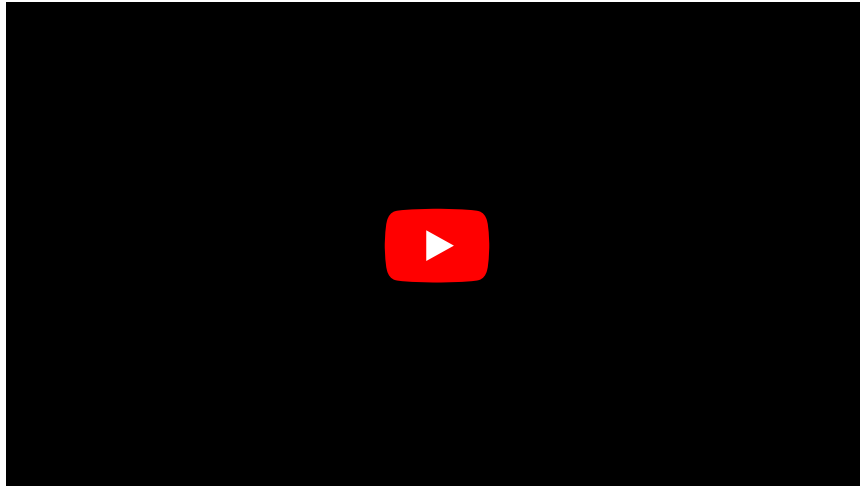


Metabolic acidosis

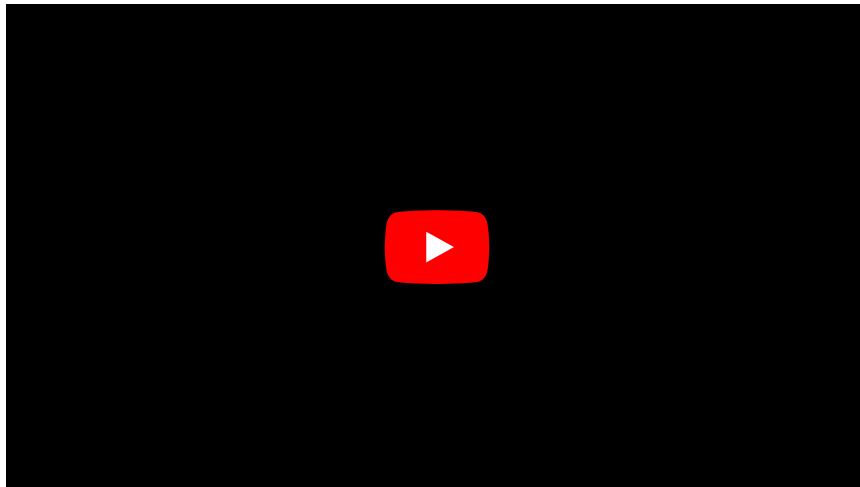
Metabolic acidosis is a condition in which the **concentration of standard bicarbonates falls below reference values**. This can happen:

- 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.

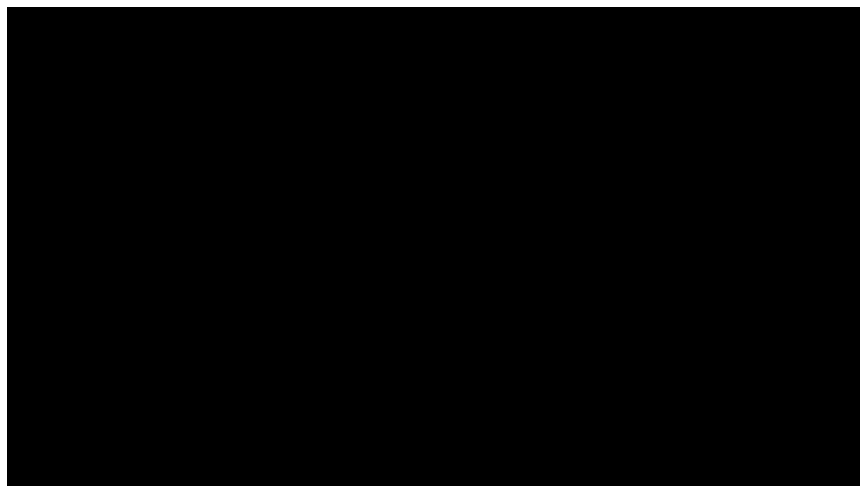
MAC:



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.

Links

Related articles

- Parametry acidobazické rovnováhy
- Mechanismus udržování acidobazické rovnováhy
- Laboratorní vyšetření acidobazické rovnováhy
- Poruchy acidobazické rovnováhy
 - Metabolická alkalóza
 - Respirační acidóza
 - Respirační alkalóza
 - Kombinované poruchy acidobazické rovnováhy
- Korekce a kompenzace poruch acidobazické rovnováhy
- Principy léčby poruch acidobazické rovnováhy
- Vztahy mezi acidobazickou rovnováhou a ionogramem

References

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