

Acid-Base Disorders (Neonatology)

Normal values of acid-base balance (ABR) or *examination of blood gases and internal environment according to Astrup* depend on the sampling site (arterial/venous/capillary) and the gestational age of the newborn. The measured values include pH, pCO₂ and O₂, other values are added (excess/deficiency of bases, bicarbonate, oxygen saturation).

The gold standard is ABR examination from arterial blood (from a peripheral arterial catheter or an umbilical arterial catheter). Venous ABR has lower pH, higher pCO₂ and low/not evaluable pO₂ compared to arterial blood. Capillary ABR (from heated heel) has pH and pCO₂ values closer to arterial blood than venous sample (values the same or slightly different), pO₂ is low/not evaluable. Normal arterial pH is 7.30-7.40 and normal capillary pH is 7.25-7.35. It is not possible to draw conclusions from a single ABR examination, the development of ABR over time and the context (clinical condition, etc.) are important.^[1]

Normal arterial blood ABR values of newborns (at normal body temperature and normal hemoglobin) ^[1]							
Gestational age	pH	PaO ₂		PaCO ₂		HCO ₃ (mmol/l)	BE/BD
		(mm Hg)	(kPa)	(mm Hg)	(kPa)		
A full-term newborn	7.32-7.38	80-95	10.7-12.7	35-45	4.7-6.0	24-26	± 3.0
30-36 week of gestation	7.30-7.35	60-80	8.0-10.7	35-45	4.7-6.0	22-25	± 3.0
< 30th week of gestation	7.27-7.32	45-60	6.0-8.0	38-50	5.0-6.7	19-22	± 4.0

BE/BD = base excess/deficiency; body temperature 37 °C; hemoglobin 148-155 mg/l.

Anion gap

- the difference between measured cations and anions in serum or plasma:

$$\text{Anion gap (mmol/l)} = \text{Na (mmol/l)} - [\text{Cl (mmol/l)} + \text{HCO}_3 \text{ (mmol/l)}]$$

- normal values: 8-16 mmol/l (up to 18 mmol/l in premature newborns < 1000 g).^[1]

Differential diagnosis

Metabolic acidosis (MAC)

- ↓ pH and ↓ HCO₃ (normal/low pCO₂).
- Main causes: loss of bases (bicarbonate) from renal or gastrointestinal causes, reduced renal acid excretion or increased acid production.
- occurs in severely ill newborns;
- mild metabolic acidosis is common shortly after birth.
- MAC + increased anion gap'** (> 16 mmol/l; in newborns < 1000 g: > 18 mmol/l); normal chlorides ⇒ another acid is additionally present:
 - Lactic acidosis'**
 - causes: asphyxia, hypoxia, respiratory distress syndrome, sepsis, impaired cardiac output (cardiogenic, septic and hypovolemic shock), circulatory or ventilatory failure, massive bleeding, severe anemia, intracranial bleeding, hypothermia, hypotension, PDA, necrotizing enterocolitis, intestinal ischemia, excessive ventilation pressures compromising cardiac output, convulsions, ascites, leakage of fluids into the third space.
 - Inborn metabolic defects** (lactic acidosis without signs of impaired tissue perfusion)
 - causes: organic acidemia (most common), galactosemia, hereditary fructose intolerance, maple syrup disease, congenital/primary lactic acidosis, glycogenosis type I, pyruvate dehydrogenase/carboxylase deficiency, defects of the mitochondrial respiratory chain, fatty acid oxidation disorders (persistent MAC, increased anion gap, negative ketones in urine, HELLP syndrome mother).
 - Acute kidney damage, chronic kidney disease** (failure to excrete hydrogen ions and other acidic anions)
 - Late metabolic acidosis of immaturity** (1st to 3rd week of life, impaired growth; with excess protein intake)
 - Toxins and drugs:** maternal use of salicylates, maternal acidosis; alcohols and glycols, acetaminophen, α-adrenergic agent, cocaine, ibuprofen, iron, valproate,...
- MAC + hyperchloremia;** normal anion gap
 - Most common causes: renal tubular acidosis and diarrhea. Low potassium ⇒ loss of bases. High potassium ⇒ renal tubular acidosis.
 - Renal bicarbonate losses:** immature kidneys, renal tubular acidosis (impairment of bicarbonate reabsorption or hydrogen ion secretion), acute kidney injury, renal dysplasia, drugs (spironolactone, etc.),

hypoaldosteronism (\downarrow Na^+ and \uparrow K^+), hyperparathyroidism.

- **Gastrointestinal bicarbonate losses**: diarrhea (secretory), urological and GIT procedures (surgical intervention in NEC, ileostomy, enterocutaneous or intestinal fistula, drainage of the small intestine or pancreas...), drugs (cholestyramine, CaCl_2 , MgSO_4 ,...).
- **Dilution acidosis** - rapid volume expansion with solutions of ringer lactate, physiological solution, glucose with dilution of bicarbonates.
- **Excess chlorides in IV fluids**.
- False acidosis - with excess heparin in the syringe, with air admixture in the sample.
- Hyperalimentation acidosis.
- Potassium-sparing diuretics and hyperkalemia.^[1]

Metabolic alkalosis

- \uparrow pH and \uparrow HCO_3^- (pH > 7.45 and BE > 5).
- Infrequent in newborns, usually iatrogenic with excessive supply of HCO_3^- (e.g. chronic alkalinization) or with excessive losses of H^+ (e.g. with losses of gastric juices or diuretic therapy) .
- **MAL chloride-resistant** (high chloride content in urine, > 20 mmol/l); increased volume of extracellular fluids:
 - causes: excessive administration of alkaline solutions (acetate, bicarbonate, malate), early diuretic therapy (furosemide), hypokalemia, transfusion of a large volume of blood derivatives, Bartter's syndrome (administration of mineralocorticoids), exogenous administration steroids, Cushing's, Conn's or Liddle's syndrome, primary aldosteronism, variants of congenital adrenal hyperplasia (excess syndrome deoxycorticosterone), milk-alkali syndrome (excess calcium and alkali intake), neonatal pseudo-Barter syndrome (secondary to maternal eating disorders);
- **MAL chloride-responsive** (low urinary chloride content, < 10 mmol/l); low serum chloride level, reduced extracellular fluid volume:
 - losses of gastric fluid (persistent vomiting, continuous suction from the stomach), secretory diarrhea (congenital chloride-wasting diarrhea), acute correction of chronic compensated respiratory acidosis, late diuretic therapy, post hypercapnic syndrome;
- **common causes of MAL in newborns**:
 - prolonged suction from the stomach (nasal/orogastric tube);
 - diuretic therapy (furosemide in children with BPD);
 - excessive administration of bases (sodium bicarbonate, citrate, acetate, lactate) in parenteral nutrition or increased enteral supply of bases
 - potassium deficiency;
 - compensation of respiratory acidosis (children with BPD/CLD, chronic ventilation).^[1]

Respiratory acidosis

- \downarrow pH and \uparrow pCO_2 (insufficient alveolar ventilation \rightarrow \uparrow pCO_2).
- In children with BPD, a higher pCO_2 level is sometimes tolerated, so-called permissive hypercapnia.
- Main causes:
 - obstruction of the endotracheal tube (e.g. mucus);
 - wrong position of the endotracheal cannula (end in the oropharynx, or vice versa in the right bronchus or on the carina);
 - insufficient ventilation support or ventilator failure (circuit disconnection, etc.);
 - progression of respiratory failure (RDS, transient tachypnea of the newborn, pneumonia, BPD/CLD, pleural effusion, atelectasis, ...);
 - pneumothorax;
 - hypoventilation, insufficient respiratory effort (effect of maternal anesthesia, medication, neuromuscular disorders, congenital central hypoventilation syndrome, sepsis, intracranial hemorrhage, hypoglycemia);
 - open duct of Botall (PDA) with pulmonary edema;
 - pulmonary hypoplasia, congenital diaphragmatic hernia, paralysis of the phrenic nerve etc.^[1]

Respiratory alkalosis

- \uparrow pH and \downarrow pCO_2 .
- CAVE: may be accompanied by reduced cerebral blood flow and reduced tissue oxygen supply (\rightarrow hypocapnia is associated with periventricular leukomalacia and deafness).
- Main causes:
 - "overventilation" pulmonary ventilator;
 - air bubble in the analyzed blood sample;
 - heparin in the analyzed blood sample (heparin falsely reduces pCO_2);
 - central hyperventilation (increased respiratory drive, e.g. in case of CNS damage or transient hyperammonemia);
 - hypoxemia (hypoxemia stimulates the respiratory center through chemoreceptors \rightarrow hyperventilation);
 - hyperventilation during sepsis, fever, aspiration pneumonia, etc.
 - compensation of metabolic acidosis.^[1]

Notes on ABR correction

Do the ABR values correspond to the development of the clinical condition? If not, repeat the examination. (CAVE: bubble or heparin in the analyzed sample → false RAL)

MAC Correction

- diagnosis and treatment of the cause (eg, hypoxia, hypovolemia, sepsis, bleeding, low cardiac output, severe anemia);
- MAC is not corrected by hyperventilation (\downarrow $p\text{CO}_2 \Rightarrow \downarrow$ cerebral blood flow \Rightarrow risk of periventricular leukomalacia);
- volume expansion is given only for signs of hypovolemia (severe MAC \Rightarrow reduced myocardial contractility);
- correction of MAC by administration of bicarbonate is controversial ($\text{NaHCO}_3 \Rightarrow \uparrow$ natremia, transient fluctuations of cerebral blood flow, risk of intracranial bleeding, worsening of cardiac functions, reduced tissue oxygen supply, progression of intracellular acidosis); the administration of NaHCO_3 can be considered in the substitution of chronic GIT/kidney losses, in the urgent treatment of severe hyperkalemia accompanied by changes on the ECG, acute decompensation of some congenital metabolic defects, or severe, life-threatening metabolic acidosis;
 - 8.4% solution of NaHCO_3 (sodium bicarbonate) is a one-molar solution - this means that there is 1 mmol of NaHCO_3 in 1 ml of solution;
 - NaHCO_3 is a hypertonic solution (\Rightarrow dilute sufficiently with Aqua for injection, administer into a large vein; max. IV concentration 0.5 mmol/ml = max. 4.2% NaHCO_3);
 - "half" correction according to ABR: HCO_3^- (mmol) = $0.3 \times \text{base deficit (mmol/l)} \times \text{weight (kg)} \times 0.5$
 - give slowly i.v. (max. 1 mmol/kg/h); ensure adequate CO_2 ventilation ($\text{NaHCO}_3 \rightarrow \text{H}_2\text{CO}_3 \rightarrow \text{CO}_2$ and H_2O);
- correction of MAC by administration of sodium acetate (or sodium ethanoate or sodium acetate; CH_3COONa = organic acid salt; after metabolizing organic anions, a strong cation remains in excess and bicarbonate is generated to maintain electroneutrality and its concentration in the blood increases);
 - dosage same as bicarbonate;
- during MAC correction, it is necessary to monitor potassium (\uparrow pH $\Rightarrow \downarrow$ potassium \Rightarrow risk of hypokalemia);
- in persistent MAC, congenital metabolic defects must be ruled out.^[1]

Template:Working

References

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External links

References

1. GOMELLA, Tricia – EYAL, Fabien – BANY-MOHAMMED, Fayez. *Gomella's Neonatology*. 8. edition. McGraw-Hill Education, 2020. 1472 pp. pp. 98, 431-442. ISBN 9781259644818.