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, pCO_2 and O_2, 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 pCO_2 and low/not evaluable pO_2 compared to arterial blood. Capillary ABR (from heated heel) has pH and pCO_2 values closer to arterial blood than venous sample (values the same or slightly different), pO_2 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**	**p_aO_2**		**p_aCO_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:

  Anion gap (mmol/l) = Na (mmol/l) - [Cl (mmol/l) + HCO_3 (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 ↓ HCO_3 (normal/low pCO_2).
- 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 (↓ Na^+ and ↑ 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

- ↑ pH and ↑ 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-Barterra 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 (naso/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

- ↓ pH and ↑ pCO_2 (insufficient alveolar ventilation → ↑ 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

- ↑ pH and ↓ pCO_2.
- CAVE: may be accompanied by reduced cerebral blood flow and reduced tissue oxygen supply (→ 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 → 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 (↓ pCO_2 ⇒ ↓ cerebral blood flow ⇒ risk of periventricular leukomalacia);
- volume expansion is given only for signs of hypovolemia (severe MAC ⇒ reduced myocardial contractility);
- correction of MAC by administration of bicarbonate is controversial ($NaHCO_3$ ⇒ ↑ 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 (⇒ 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 × base deficit (mmol/l) × weight (kg) × 0.5
  - give slowly i.v. (max. 1 mmol/kg/h); ensure adequate CO_2 ventilation ($NaHCO_3$ → H_2CO_3 → CO_2 and H_2O);
- correction of MAC by administration of sodium acetate (or sodium ethanoate or sodium acetate; CH3COONa = 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 (↑ pH ⇒ ↓ potassium ⇒ risk of hypokalemia);
- in persistent MAC, congenital metabolic defects must be ruled out.[1]

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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.