Н.А. Ходорович, Э.В. Величко

ПАТОФИЗИОЛОГИЯ НАРУШЕНИЙ КИСЛОТНО-ОСНОВНОГО СОСТОЯНИЯ

АЛГОРИТМ И ПРИМЕРЫ РЕШЕНИЯ КЛИНИЧЕСКИХ ЗАДАЧ

ACID-BASE DISORDERS

ALGORITHM AND CASE STUDIES

Учебно-методическое пособие

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Ходорович, Н. А.

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Пособие предназначено для англоговорящих студентов, обучающихся в медицинских вузах по специальностям: «Лечебное дело», «Стоматология», «Фармация».

Также может быть использовано для обучения интернов, ординаторов и аспирантов медицинских специальностей.

This study guide is for English-speaking medical students in the field of: «General Medicine», «Dentistry», «Pharmacy».

Also recommended for interns, residents, and graduate students in medical specialties.

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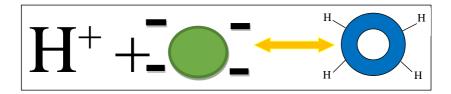
INTRODUCTION

Acid-base balance is one of the most important body constants and features.

Numerous biochemical and physiological processes are dependent on the hydrogen ion concentration: oxidation-reduction; protein breakdown and synthesis; the rate of glycolysis; state of colloid systems; enzymatic activity; cell growth and proliferation; genetic mechanisms.

Acid-base balance (ABB) is the relative constancy of the hydrogen index (pH) of the internal environment of the body, due to the interaction of buffer systems that determine the usefulness of metabolic reactions in the body.

THE BIOLOGICAL SIGNIFICANCE OF NORMAL ACID-BASE PHYSIOLOGY



- H⁺ bound to the organic molecules change their structure and properties: Proteins (mainly enzymes) Nucleate acids Carbohydrates Lipids (amphiphilic molecules) and so on.
- 2. There is an accumulation of acids and bases in the body. Generally, acid accumulation is faster than bases.

CO₂ TRANSPORT

1 liter of venous blood transports 2 mmol of CO_2 . This CO_2 is called "exchange" because it is coming from the tissues into the blood, then from the blood into the lungs and finally it is further egested during ventilation.

• 10% CO₂ (approx. 0.2 mmol) is transported by venous blood in a physically dissolved state, in equal proportions between blood plasma and red blood cells (RBC);

- 10% CO₂ (approx. 0.2 mmol) is transported as a part of carbaminohemoglobin;
- 80% CO₂ (approx. 1.6 mmol) is transported as bicarbonate;
- 0.9% CO₂ is transferred by plasma in the form of HCO₃;
- 0.7% CO₂ is transferred by RBC in the form of HCO₃⁻.

As you know, pH ("Hydrogen power") is the negative logarithm of the hydrogen ion concentration [H⁺] in solution. So, for example, if pH = 7.0, then this means that [H⁺] is equal to 1/10000000 (10-7) equivalent in liter. In the case of hydrogen, one gram is equal to one equivalent and thus, at pH = 7.0, 0.0000001 g of hydrogen is contained in a liter of solution. Accordingly, for example, at pH = 8.0 in a liter of solution contains 0.00000001 g of hydrogen, etc.

Blood pH is one of the most tightly regulated physiological constants. The physiological blood pH is normally maintained between 7.36 and 7.42. Any deviation of 0.1 from this range can develop into severe pathology. A deviation of blood pH of more than 0.2 from the normal range indicates a coma state. If the blood pH changes more than 0.3 from the physiological range, its life-threatening.

MAIN ACID-BASE BALANCE INDICATORS

- *Actual blood pH* is arterial blood pH, measured without air access at a temperature of 37 ° C.
- The actual (true) partial pressure of carbon dioxide (Pco_2) is the value of the arterial carbon dioxide pressure (P_aco_2), defined without air access at a temperature of 37 ° C.
- *Standard bicarbonate (SB)* is the content of bicarbonates in blood plasma (mmol / L) when it is fully saturated with oxygen and with *Pco*₂ = 40 mmHg, defined at a temperature of 37 ° C.
- *Actual (true) bicarbonate (AB)* is the concentration of bicarbonate in blood plasma (in mmol / L) with true *Pco*₂, measured at a temperature of 37 ° C.
- Excess (lack) of buffer bases (*BE* ''*Base Excess*'') is the difference between the average normal content of the buffer bases (whole blood pH = 7.40 and *Pco*₂ = 40 mmHg) and the known value of buffer base concentration.

NORMAL VALUES OF THE MAIN ACID-BASE BALANCE INDICATORS OF THE ARTERIAL BLOOD

INDICATOR	VALUE
рН	M = 7.36-7.42; F = 7.37-7.42
Pco ₂ (mm Hg)	M = 35.8- 46.6; F = 32.5-43.7
SB	21.3-24.8 mmol / L
AB	18.8-24.0 mmol / L
BE	M = -2.4 + 2.3; F = -3.3 + 1.2

NORMAL VALUES OF OTHER ADDITIONAL BLOOD AND URINE ABB INDICATORS

INDICATOR	VALUE
Lactic acid (blood)	0.9 - 1.75 mmol / L (6-16 mg %)
Ketone bodies (blood)	0.4 - 1.72 mmol / L (0,5-5 mg %)
TA (titratable acidity of the daily	10-30 ml of alkali
urine)	
Urinary ammonia (NH4 ⁺)	20-50 mmol / L

BUFFER SYSTEMS OF THE BODY

The bicarbonate buffer system (Bicarbonate-carbonic acid buffer pair) is clinically most relevant and determined as carbonic acid/its acid salt equation, e.g.: $H_2CO_3/NaHCO_3$. Disproportion in this buffer pair reveals changes in all other buffer systems. This buffer pair is continuously maintained in a proportion of 1/20. When a strong acid is generated or enters the body (using HCI as an example), the following reaction occurs:

$NaHCO_3 + HCI \leftrightarrow NaCI + H_2CO_3$

In this case, excess sodium chloride is easily excreted by the kidneys, and carbonic acid, induced by the carbonic anhydrase enzyme, decomposes into water and carbon dioxide, the excess of which is rapidly breathed out by the lungs.

When an excess of alkaline substances enters the internal environment of the body (using NaOH as an example), the reaction proceeds differently:

$H_2CO_3 + NaOH \leftrightarrow NaHCO_3 + H_2O$

A decrease in the carbonic acid concentration is compensated by a decrease in the carbon dioxide (CO_2) removal in the lungs.

The bicarbonate used in the buffering process must be regenerated to maintain normal acid-base balance.

The phosphate buffer system acts by maintaining a constant ratio of the oneand two-metal salt of phosphoric acid. In the case of sodium salts (dihydrogen phosphate and sodium hydrogen phosphate), this ratio is as follows: NaH_2PO_4 / Na_2HPO_4 . This ratio is maintained in a proportion of 1/4.

When this system interacts with acidic substances, sodium dihydrogen phosphate and sodium chloride are generated:

$Na_{2}HPO_{4} + HCI \leftarrow \rightarrow NaH_{2}PO_{4} + NaCI$

And when it reacts with alkaline substances, monosubstituted sodium hydrogen phosphate and water are generated.

$Na_2HPO_4+NaOH \leftarrow \rightarrow Na_2HPO_4+H_2O$

Excess products of both reactions are removed by the kidneys.

The protein buffer system can manifest its properties due to the protein amphoteric quality. In one instance proteins can react with bases as acids (as a result of the reaction, alkaline albumin is generated), and in another instance, with acids as bases (with acidic albumin generation). In general, in a very schematic form, this pattern can be illustrated as follows:

PROTEIN

$$NH_2 + HCI \rightarrow NH_4 CI$$

The hemoglobin buffer system to a large extent provides the buffer capacity of the blood. This is because oxyhemoglobin (HbO₂) is a lot of stronger acid than reduced hemoglobin (Hb). In the venous capillaries, a large amount of acidic decomposition substances enters the blood, and it is enriched with carbon dioxide, which shifts this reaction to the acidic band. But at the same time, hemoglobin is being reduced in the same areas of the microvasculature, and becoming a weaker acid, gives up a significant part of the alkaline substances bound to Hb. These alkaline substances, reacting with carbonic acid, generate bicarbonates.

And so, the most capacious buffer is bicarbonate buffer; and the "fastest" – hemoglobin buffer. In addition to the blood buffer mechanisms, processes occurring in the kidneys take part in the regulation of acid-base balance. The role of the kidneys is especially great in the excretion of acidic decomposition substances and the body alkaline maintenance.

Kidneys keep maintaining of acid-base balance by the following mechanisms:

1. Resorption of sodium bicarbonate in the renal (mainly proximal) tubule;

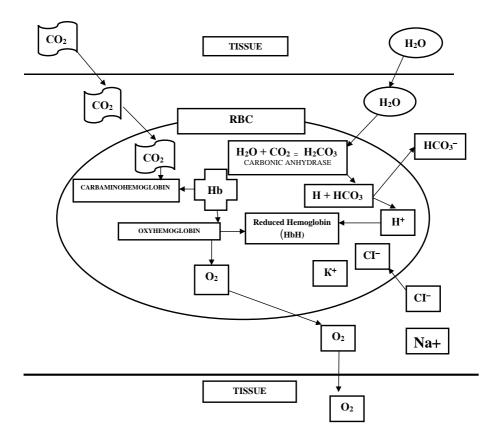
2. Converting less acidic compounds to more acidic;

3. Generation of free weak organic acids;

4. Ammoniogenesis.

BINDING OF PHYSIOLOGICAL MECHANISMS IN MAINTAINING AN ACID-BASE BALANCE (by Michel A. Grippy, 1997)

The picture illustrates the carbon dioxide transport, the generation of bicarbonates, the chlorine shift, and the binding of hydrogen ions in tissue capillaries (in pulmonary capillaries, when the oxygen is absorbed and carbon dioxide is released, this reaction proceeds in the reverse order).



TRANSPORT OF CARBON DIOXIDE, GENERATION OF BICARBONATES, BINDING OF HYDROGEN IONS

The main amount of carbon dioxide (90%) in arterial blood is contained in the state of bicarbonate, which is generated due to the reaction between carbon dioxide and water (with an appearance of H_2CO_3) and subsequent dissociation into a hydrogen cation [H⁺] and an HCO₃⁻ anion. This reaction induced by the intracellular enzyme *carbonic anhydrase* proceeds extremely quickly in red blood cells.

The HCO₃⁻ anion passes freely through the cell membrane and accumulates in the blood plasma, generating sodium bicarbonate.

Hydrogen cations [H⁺], like any other cations, pass poorly through the RBC membrane and accumulate in the cell. Their excess is eliminated by combining with reduced hemoglobin. The last one is created in tissue capillaries after oxygen detachments and its diffusion into the tissue.

On the other hand, the cell electroneutrality is provided by the diffusion of chlorine anions into the RBC (the so-called *chloride shift*).

A small amount of carbon dioxide is transported from tissue to pulmonary capillaries due to the binding with hemoglobin (in the form of *carbaminohemoglobin*).

When acid-base disorders are compensated, the buffer systems of RBC, plasma and intercellular fluid react as a cohesive whole, since:

• The blood buffer capacity is distributed almost evenly between plasma and red blood cells.

•The hemoglobin buffering properties have the first and greatest importance in cells, and bicarbonates are in second place.

• In plasma, on the contrary, bicarbonates come first, and proteins come second.

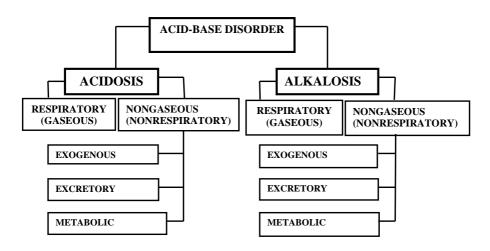
• The intercellular (interstitial) fluid, which is functionally connected with the blood, contains only bicarbonate buffer. (Its concentration is about 2 times less than in plasma, but since the extracellular fluid is about 2 times more than blood, their total buffer capabilities are the same).

The acid-base disturbance may be caused by:

- Abnormal CO₂ removal in the lungs ("respiratory" acidosis and alkalosis);
- Abnormalities in the regulation of bicarbonate and other buffers in the blood (for example, "metabolic" acidosis and alkalosis).

An increase in the $[H^+]$ (a fall in pH) is determined as *acidaemia* (the disorders caused by these changes in the blood pH are called **acidosis**). A decrease in the $[H^+]$ (a rise in the blood pH) is termed as *alkalemia* (the disorders caused by these changes in the blood pH are called **alkalosis**).

CLASSIFICATION OF ACID-BASE DISORDERS



TYPES OF ACID-BASE DISORDERS

ACIDOSIS

I. RESPIRATORY or GASEOUS (retention of CO₂):

- reduction of CO₂ removal due to a disorder of external respiration;
- high concentration of CO₂ in the environment (closed rooms, mines, submarines and so on);
- defective anesthesia machine and breathing equipment (rare!).

II. NONGASEOUS (accumulation of nonvolatile acids).

1. Metabolic:

• ketosis (ketoacidosis) – is due to acid generation, or oxidation disorders and resynthesis of ketone bodies (diabetes mellitus, fasting, liver functional abnormalities, fever, hypoxia, and others);

• lactic acidosis – is due to an increased lactic acid generation, reduced oxidation, and its resynthesis (e.g., hypoxia, hepatic dysfunction, infections, etc.).

• acidosis with an accumulation of other organic and inorganic acids (extensive inflammation, burns, injuries, etc.).

2. Excretory:

• Retention of acids due to impaired acid excretion by the kidneys in renal failure (diffuse nephritis, uremia);

• loss of alkalis;

- Renal (renal tubular acidosis, salt-losing nephritis, hypoxia, intoxication by sulfonamides);

- Gastro-enteric (diarrhea, hypersalivation).

3. Exogenous:

- long-term acidic food ingestion;
- ingestion of certain medications (eg, NH4Cl);
- acid ingestion (rare!).

III. MIXED

respiratory and nongaseous acidosis:

- develops due to asphyxia,
- cardiovascular disease,
- severe disorders of the cardiovascular and respiratory systems, etc.

OR a combination of different types:

- lactic acidosis and ketosis;
- metabolic and excretory;
- and other combinations.

ALKALOSIS

I. RESPIRATORY (gaseous) alkalosis

• increased elimination of CO₂ due to the external breathing hyperventilation nature (neurosis, altitude sickness);

• hyperventilation controlled breathing.

II. NONGASEOUS alkalosis

1. Excretory:

• Retention of bases (increased reabsorption of alkaline ions (bases) by the kidneys);

• loss of acids (vomiting at pyloric stenosis, intestinal obstruction, toxemia of pregnancy, hypersecretion of gastric juice);

• electrolyte imbalance: hypokalemia, hyponatremia and hypochloremia (blocked renal compensation) lead to sub- and decompensated types of alkalosis (60-65%).

2. Exogenous:

• prolonged bicarbonate ingestion or recovery from starvation;

• drug administration (ampicillin, penicillin therapy, bicarbonate, and other alkaline substances);

• intensive treatment with corticosteroids (mineralocorticoid excess) and diuretics (potassium depletion).

3. <u>Metabolic</u> (rare). Hypercalcemia states: hypofunction of parathyroid glands, hypercalcemia of malignancy, acute or chronic milk-alkali syndrome.

III. MIXED

gaseous (respiratory) and nongaseous alkalosis

• respiratory and metabolic alkalosis (in brain injuries, chronic anemia, and renal diseases);

• respiratory alkalosis and renal tubular acidosis (heart failure and treatment with carbonic anhydrase inhibitors).

Each buffer system has a specific capacity. All systems can be arranged in the order of decreasing capacity of the buffer properties, as a percentage of the entire blood capacity.

DISTRIBUTION OF BUFFER SYSTEMS AS A PERCENTAGE OF THE TOTAL BLOOD CAPACITY

RBC - 57%

Including:

- Hemoglobin 35%
- Bicarbonates 28%
- Organophosphate acids 3%
- Inorganic phosphorus 1%

Plasma - 43%

Including:

- Bicarbonates 35%
- Plasma proteins 7%
- Inorganic phosphorus 1%

ALGORITHM OF CASE STUDIES "ACID-BASE DISORDERS"

- 1. Determine how the pH value given in the task differs from the physiological one. The physiological blood pH range lies from 7.36 to 7.42 on average for men and women. If this value is way beyond the normal one, there is uncompensated acidosis or alkalosis. Borderline values of the norm (7.36; 7.35 or 7.42; 7.41) may indicate the presence of compensated acidosis or alkalosis.
- 2. Determine how the value of carbon dioxide partial pressure (PCO_2) corresponds to the normal. The average PCO_2 range lies within 32.5 to 46.6 mmHg for men and women. PCO_2 values below 32.5 or above 46.6 mmHg mean either CO₂ removal from the body or, conversely, its retention in the body. In both cases, this will indicate certain disorders of external respiration (hyperventilation or hypoventilation).

These disorders can be associated with any lung diseases, changes in the concentration of oxygen and carbon dioxide in the environment, forced lung ventilation, or other causes. It is important that a decrease in the arterial carbon dioxide pressure, as a rule, leads to the development of respiratory alkalosis, and an increase of PCO_2 - to the development of respiratory acidosis.

3. Define the values of SB and AB, keeping in mind that the excess of AB over SB is typical for respiratory acidosis, and the excess of SB over AB is for respiratory alkalosis.

If there are acidosis and SB<AB than it is respiratory.

If there are alkalosis and SB>AB than it is respiratory.

- 4. Determine the BE value (average normal value for men and women ranges from -3.3 to +2.3). Out of lower normal limit BE value (negative) means that the body in attempts to compensate an acidosis has exhausted its base reserves. Out of upper normal limit BE value (positive) means that the body has an abnormally increased accumulation or retention of buffer bases (base reserves), which characterize alkalosis developing.
- 5. If the problem contains data on the lactic acid concentration in the blood (normal value is 0.9 1.75 mmol / L), and if this indicator is higher than the normal one, then one should assume an intensification of the

glycolysis process, which is typical for the development of hypoxia and metabolic acidosis.

- 6. The presence of elevated blood ketone body concentration (normal value ranges 0.4 1.72 mmol / L) indicates the development of metabolic acidosis, most likely of diabetic origin.
- 7. Titratable acidity of the daily urine (TA) and urinary ammonia are two indicators that can also be given in the conditions of a case because they are involved in acid excretion.

A conjugated anion of a titratable acid (such as phosphate) can be bond to the hydrogen ion/ions, titrated within the pH range, and physiologically excreted in the urine. Each hydrogen ion is secreted as a titratable urinary buffer. The normal value of TA of the daily urine is 10-30 ml of bases.

In the presence of metabolic acidosis, titratable acids cannot increase greatly because their capacity is limited by the plasma concentration. Controversy, the ammonia buffering system is able when necessary. The normal value of urinary ammonia is 20-50 mmol / L.

As a rule, an increase of these parameters indicates that the body intensively removes acidic substances, that is, it tries to compensate acidosis (most often metabolic) in this way. However, a decrease in these parameters (compared with the normal values), when the blood pH is shifted to the acid side and there are other signs of compensated or uncompensated acidosis, indicates the excretory renal dysfunction and the development of excretory acidosis. Especially when urine-pH is lower the normal range (5-7).

8. It is very important to keep in mind that many cases have additional notes that at least briefly describe the state caused the acid-base disorder of the body. You should carefully read these notes since the "key" to solving the problem is often contained in them.

"ACID-BASE DISORDERS" CASE STUDIES

rmal range (7.36-
35 is a lower
erline. So, there is the
ndency to acidosis
osis).
of PCO ₂ is 32.5 to
e case, PCO ₂ is 52
tor is greatly above
herefore, there is CO ₂
e blood. Indirectly,
an increase in AB.
icantly arises the
o less important SB
ory acidosis is
ch a state.
r the physiological
so there is great
the blood.

Case № 1

The answer is *<u>compensated respiratory</u> (gaseous) acidosis* caused by impairment of respiratory function.

• pH 7.24	The pH of 7.24 is significantly lowered (physiological pH range is 7.36-7.42) to the acidic side. 7.36
• <i>PCO</i> ₂ 69 mm Hg	 7.24 0.12 Deviation of blood pH more than 0.1 from the normal range indicates severe state or pathology. So, there is the right to assume uncompensated acidosis. In the case, <i>PCO</i>₂ is 69 mm Hg. The normal is 32.5 to 46.6 mm Hg. The <i>PCO</i>₂ indicator is greatly above the normal limit. Therefore, there is CO₂ accumulation in the blood. Indirectly, this should lead to an
• SB 18.5 mmol/L	increase in AB. SB indicator is below the normal (21.3-24.8 mmol/L), and AB
• AB 28.0 mmol/L	indicator significantly arises the normal value (18.8 – 24.0 mmol/L) and no less important more than SB. Respiratory acidosis is characteristic of such a state.
• BE -8.0 mmol/L	The BE indicator (- 8.0 mmol/L) is greatly lower than the physiological borderline (- 3.3), so there is a great use of buffer bases due to the removal of alkaline reserves of blood.
Note: patient is under surgery with the use of mechanical ventilation.	In a view of the note, the situation has arisen from lung hypoventilation. This led to the CO ₂ retention in the body, increased glycolysis, and the accumulation of acidic metabolic substances in the blood.

The answer is *uncompensated mixed respiratory and metabolic acidosis*.

• pH 7.42	The pH of 7.42 is the upper physiological borderline. So, compensated alkalosis can be considered.
• <i>PCO</i> ₂ 30 mm Hg	PCO ₂ is below normal (physiological range is 32.5 to 46.6 mm Hg). There is CO ₂ removal from the lungs due to hyperventilation. It results in a decrease of H ₂ CO ₃ generation into the blood. However, the same circumstance will reduce the ability of the bicarbonate generation in the blood.
• SB 20.5 mmol/L	Pay attention to AB less than SB, and this is the indicator of respiratory alkalosis.
 AB 18.0 mmol/L BE - 4,0 mmol/L 	It is proved by AB decrease (the physiological range is $18.8 - 24.0$ mmol/L) and defined exhaustion of alkaline reserves of the body (BE - 4.0 mmol / L at a rate of -3.3
Note: patient is under surgery with the use of mechanical ventilation.	- +2.3).

The answer is *compensated respiratory alkalosis*.

	1
• pH 7.17	The pH of 7.17 is significantly shifted to the acid side and is outside the normal range (7.36-7.42). We
	can assume that the patient has
	uncompensated acidosis.
	7.36
	-
	7.17
	0.19 Deviation of blood pH of
	approximately 0.2 from the
	normal range indicates a coma
	state.
• <i>PCO</i> ₂ 50 mm Hg	The PCO ₂ (50 mm Hg) significantly
	arises the normal (32.5-46.6 mm
	Hg), which indicates the carbon
	dioxide accumulation in the patient's
	blood. Coma is the reason for
	dyspnea and CO ₂ retention.
• SB 15.5 mmol/L	SB indicator is below the normal
• 3B 13.3 IIIII01/L	(21.3-24.8 mmol/L).
	(21.3-24.8 mm0/L).
• AB 38.0 mmol/L	AB indicator significantly arises the
	normal value $(18.8 - 24.0 \text{ mmol/L})$.
	Respiratory acidosis is
	characteristic of such a state.
• BE - 13.0 mmol/L	Since a part of the carbonic acid is
	used for the bicarbonate generation,
	which, however, cannot compensate
	for this severe acidosis, alkaline
	reserves are completely exhausted
	(BE –13.0 mmol / L).
	Even though the excess of AB over
	SB indicates the respiratory genesis
	of acidosis, such a great decrease in
	pH cannot be explained only by this
	reason.
Note: A patient is in a coma.	The cause of the patient's coma is
	metabolic acidosis.
The answer is <i>uncompensated mixed respiratory and metabolic acidosis</i> .	

Case № 5

• pH 7.23	The pH of 7.23 is significantly shifted to the acid side and is outside the normal range (7.36-7.42). 7.36 - 7.23 0.13 Deviation of blood pH more than 0.1 from the normal range indicates severe state or
	pathology. So, there is the right to assume
• <i>PCO</i> ₂ 34 mm Hg	uncompensated acidosis. The PCO_2 of 34 mm Hg is within the normal (32.5 to 46.6 mm Hg). Therefore, there are no signs of
• SB 16 mmol/L	respiratory type of acidosis. The AB and SB indicators are below the normal.
• AB 13 mmol/L	A great decrease in the AB level indicates the inability of the bicarbonate buffer to compensate acidosis.
• BE - 11.0 mmol/L	Data on the BE indicator of -11.0 mmol / L (at the rate of (-3. 3) to (+ 2.3)) indicate complete exhaustion of alkaline reserves of blood.
• TA of the urine: 8 ml of alkali	The TA and Urinary Ammonia indicators are significantly below
Urinary ammonia:	the normal. When there is so expressed acidosis, this can only
8 mmol/L	mean one thing - the kidneys are not able to fulfill their excretory function.
Note: a patient has diffuse glomerulonephritis.	This conclusion is confirmed by the information indicated in the note: there is the presence of diffuse glomerulonephritis in the patient.

The answer is *uncompensated excretory acidosis*.

Case № 6

	٦
• pH 7.56	The physiological pH range is 7.36 to 7.42. The pH of 7.56 is significantly upper borderline.
	7.56
	7.42
	0.14 Deviation of blood pH more
	than 0.1 from the normal range
	indicates severe state or
	pathology.
	So, there is the right to assume
	uncompensated alkalosis.
• <i>PCO</i> ₂ 26 mm Hg	The physiological PCO_2 range is 32.5 to 46.4 mm Hg. The PCO_2
	indicator in the case is 26 mm Hg.
	There is CO ₂ removal from blood
	caused by some reasons, as in an
	example, lung
	hyperventilation/shortness of breathing.
• SB 24 mmol/L	A decrease of PCO_2 leads to
	depletion of the AB level which is
- AD 17	less than the SB indicator in the case.
• AB 17 mmol/L	This is characteristic of respiratory alkalosis.
• BE +4.0 mmol/L	The note indicates that the patient
	has uncontrollable vomiting,
	therefore, in the body losing large
	amounts of gastric acid, the amount
	of acidic substances in the blood also
	decreases. This fact, despite a decrease in blood bicarbonates (low
	AB level), leads to a relative
	increase in alkaline reserves of blood
	(BE + 4.0).
Note: a patient has a concussion	Thus, uncompensated alkalosis has
accompanied by uncontrollable	two reasons: respiratory and
vomiting and shortness of breath.	excretory.

The answer is *uncompensated mixed respiratory and excretory alkalosis*.

	1
• pH 7.42	The physiological pH ranges from 7,36 to 7,42. The pH of 7.42 is an
	upper limit of the normal.
	Therefore, there is a compensated
	alkalosis.
• <i>PCO</i> ₂ 40.5 mm Hg	The normal value of PCO₂ is 32.5 to
	46.4 mm Hg. In the case, PCO ₂ is
	40.5 mm Hg. The indicator is within
	the normal. Therefore, this
	compensated alkalosis is not
	respiratory.
• SB 28 mmol/L	The AB и SB levels are over the
	normal. There is the bicarbonate
• AB 31 mmol/L	retention in the blood.
• BE $\pm 6.5 \text{ mmol/L}$	The BE of +6.5 mmol/L is over the
• BE $+0.5$ IIIII01/L	normal. It indicates the increase of
	alkaline blood reserves.
Note: a patient was intravenously	What is the reason for the
injected with a sodium bicarbonate	bicarbonate accumulation and an
solution.	increase of alkaline blood reserves?
Solution.	Turn to the note. It says that the
	patient was injected with a sodium
	bicarbonate intravenously. Perhaps
	it was due to the need to neutralize
	his acidosis. The sodium
	bicarbonate was introduced in
	excessively large quantities.
	his acidosis. The sodium bicarbonate was introduced in

The answer is *compensated exogenous alkalosis*.

" ACID-BASE DISORDERS" CASE STUDIES FOR INDIVIDUAL WORK

Case № 1

• pH	7.20	
• <i>Pco</i> ₂	90 mm Hg	
• SB	24 mmol/L	
• AB	29 mmol/L	
• BE	+6.0 mmol/L	

• pH	7.55	
• <i>Pco</i> ₂	20 mm Hg	
• SB	24 mmol/L	
• AB	20 mmol/L	
• BE	-7.0 mmol/L	

• pH 7.28	
• <i>Pco</i> ₂ 26.4 mm Hg	
• SB 15 mmol/L	
• AB 12 mmol/L	
• BE -13 mmol/L	
Note: patient has chronic renal failure.	

• pH	7.37	
• <i>Pco</i> ₂	66 mm Hg	
• SB	31 mmol/L	
• AB	39.5 mmol/L	
• BE	+8.0 mmol/L	
Note: patient has a chronic respiratory failure.		

• pH 7.30	
• <i>Pco</i> ₂ 40 mm Hg	
• SB 15 mmol/L	
• AB 15 mmol/L	
• BE -13 mmol/L	

• pH	7.60	
• Pco ₂	40 mm Hg	
• SB	40 mmol/L	
• AB	40 mmol/L	
• BE	+12 mmol/L	

• pH 7.25	
• <i>Pco</i> ² 45 mm Hg	
• SB 18 mmol/L	
• AB 18 mmol/L	
• BE -7.0 mmol/L	
• Blood lactate	
21.0 mmol/L	
• TA 8 ml of alkali	
Urinary Ammonia	
15 mmol/L	
Note: patient has toxic damage to the liver and kidneys.	

• pH	7.64	
• <i>Pco</i> ₂	28 mm Hg	
• SB	32.5 mmol/L	
• AB	29 mmol/L	
• BE	+9.3 mmol/L	
• Urine pH 4.3		
Note: patient is under surgery with the use of mechanical ventilation and intravenous sodium bicarbonate administration.		

• pH 7.22	
• <i>Pco</i> ₂ 39 mm Hg	
• SB 18 mmol/L	
• AB 18 mmol/L	
• BE -7.8 mmol/L	
 Blood lactate 22.0 mmol/L 	
• TA 60 ml of alkali	

• pH 7.50	
• <i>Pco</i> ₂ 18 mm Hg	
• SB 20.5 mmol/L	
• AB 14 mmol/L	
• BE +4.5 mmol/L	
Note: patient has a concussion.	

• pH 7.17	
• <i>Pco</i> ₂ 66 mm Hg	
• SB 31 mmol/L	
• AB 39.5 mmol/L	
• BE +8.0 mmol/L	
 Blood ketone bodies 9.1 mmol/L 	
• TA 60 ml of alkali	
Note: patient is in a coma.	

7.26	
36 mm Hg	
14 mmol/L	
14 mmol/L	
-8.0 mmol/L	
utient has chronic	
	36 mm Hg 14 mmol/L 14 mmol/L -8.0 mmol/L

• pH	7.19	
• <i>Pco</i> ₂	25 mm Hg	
• SB	14 mmol/L	
• AB	11 mmol/L	
• BE	-17.0 mmol/L	
Note: patient has acute posthemorrhagic anemia.		

• pH	7.24	
• P CO ₂	55 mm Hg	
• SB	17 mmol/L	
• AB	18 mmol/L	
• BE	-12 mmol/L	
Note: pat sided hear	tient has acute left- t failure.	

Учебное издание

Ходорович Надежда Анатольевна Величко Эллина Валериевна

ПАТОФИЗИОЛОГИЯ НАРУШЕНИЙ КИСЛОТНО-ОСНОВНОГО СОСТОЯНИЯ

АЛГОРИТМ И ПРИМЕРЫ РЕШЕНИЯ КЛИНИЧЕСКИХ ЗАДАЧ

На английском языке

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Российский университет дружбы народов 115419, ГСП-1, г. Москва, ул. Орджоникидзе, д. 3

Типография РУДН 115419, ГСП-1, г. Москва, ул. Орджоникидзе, д. 3, тел. 952-04-41