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ACID-BASE BALANCE INTERPRETATION

ISPITIVANJE POREMEĆAJA ACIDOBAZNOG STATUSA

Natalija Vuković¹, Milan Elenkov² ¹Clinic for Anesthesiology and Intensive Care, Clinical Center Niš, ²Department for Anesthesiology and Intensive Care, General hospital Pirot

Summary: Acid-base analysis is important diagnostic method in the therapeutic approach of many diseases. Accurate and timely diagnosis of acid-base disorders can be lifesaving or produce crucial changes in the disease course. Although technological innovations have brought better quality and a number of new information's, acid-base interpretation still presents challenge. Physiological acid base method uses carbonic acid-bicarbonate system as basis for interpretation. In this way, changes in partial pressure of carbon dioxide produces changes in bicarbonate in the typical order and vice versa. This method implicit two basic metabolic and respiratory disorders. **Keywords:** Acid-base, acidosis, alkalosis



INTRODUCTION

Blood gas analysis can be driven from capillary, peripheral arterial, peripheral and central venous blood sampling and from pulmonary artery. [1] It provides the assessment of acid-base disorders, ventilation and oxygenation in patients. [2] Furthermore, the comparison of acid-base results from different blood samples provides a comprehensive approach to metabolism derangements and hemodynamic characteristics. [3,4]. Advanced analyzers measure and calculate the pH, partial pressure of carbon dioxide (PCO2), partial pressure of oxygen (PO2), concentration of bicarbonate (HCO3), base excess (BE), lactate and anion gap (AG). All of the above obliges us to regularly revise our knowledge about acid base.

In this review we will revise three basic steps in the interpretation of the acid-base analysis of peripheral arterial blood gas sample. The physiological ranges of acid-base parameters differ between different analyzers but there are average values (Table 1). For the interpretation it is easier to assume that the normal pH is 7.40 and the PaCO2 is 40mmHg.

Acid base parameters	Normal ranges
pH ¹	7.35-7.45
PaCO2 ²	35-45mmHg
PaO2 ³	80-100mmHg
HCO3-4	22-26mmol/L
BE ⁵	±2
Lactate	<2mmol/L
AG ⁶	12±3mmol/L

¹pH negative logarithm of the [H⁺]; ²PaCO2 arterial partial pressure of carbon dioxide; ³PaO2 arterial partial pressure of oxygen; ⁴HCO3 concentration of bicarbonate; ⁵BE base excess; ⁶AG Anion gap

Table 1. Physiological ranges of acid-base parameters

Step one

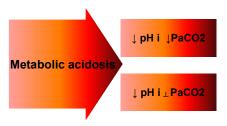
What is the primary abnormality, metabolic or respiratory?

The primary metabolic disorder occurs in these two cases [5]:

- a) If the pH and PaCO2 change in the same direction
- b) If the pH changes, while the PaCO2 stays normal

↑ pH i ↑PaCO2

↑ pH i ⊥ PaCO2





In this way, the patient will have metabolic acidosis if the pH and PaCO2 fall, or the pH falls and the PaCO2 is normal (Figure 1).

If the pH and PaCO2 rise, or the pH rises and the PaCO2 is normal, the patient will have metabolic alkalosis (Figure 2).

The primary respiratory disorder is present if the pH and PaCO2 change in the opposite direction. An increase in PaCO2 is respiratory acidosis, while a decrease in PaCO2 is respiratory alkalosis.

Step two

Metabolic alkalosis

Figure 2. Metabolic alkalosis

In the situation where the patient has metabolic acidosis the anion gap should be calculated. The anion gap is the assumption that the negatively charged anions and positively charged cations in the serum equal electrical neutrality. [6.7] It represents the difference between unmeasured anions and cations. [8] It is calculated on the basis of electrolyte measurements as follows in the equation1.1:

1.1. $AG = (Na^+ + K^+) - (Cl^- + HCO3^-)$



The normal value of AG is 12±3mmol/L. [6] Nowadays, with more accurately measured electrolytes, the normal range of AG has decreased up to 7 ± 4mmol/L. [9]

Metabolic acidosis can be divided into those having an elevated AG and those with a normal AG (10). The high AG acidosis occurs after the addition of acids or decreased excretion of endogenous acids. In those situations hydrogen ions combine with bicarbonate, decrease it and produce an increase in the AG (Figure 3).

The normal AG acidosis is characterized by the loss of bicarbonate (diarrhea etc.) or addition of chloride (rapid administration of isotonic saline etc.), (Figure 4).

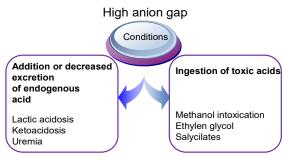


Figure 3. High anion gap

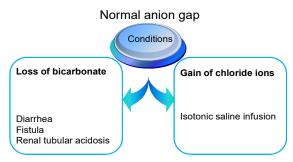


Figure 4. Normal anion gap

Step three

Is there a compensation or superimposed secondary abnormality?

This question should be answered for all the primary disorders. So, if the patient has metabolic alkalosis we should follow the explanation for compensation in metabolic alkalosis, for the patient with respiratory acidosis, we follow the interpretation for respiratory acidosis etc.

Metabolic acidosis

Respiratory compensation for metabolic acidosis is hyperventilation with reduction of Pa-CO2. [10]. The expected PaCO2 for each patient is calculated from equation 1.2. [11] In this equation, the concentration of bicarbonate that is used is from the patient's acid-base result.

1.2. Expected PaCO2= $(1.5 \times HCO3) + 8 (\pm 2)$

If the PaCO2 from the analysis is the same as the expected one, the patient has <u>compensated</u> <u>metabolic acidosis</u>. If the measured PaCO2 exceeds the expected one, then the patient has metabolic acidosis as the first derangement and the respiratory acidosis as the second one. If the measured PaCO2 is lower than the expected one, then the patient has primary metabolic acidosis with secondary respiratory alkalosis.

Metabolic alkalosis

The expected PaCO2 in patients that have metabolic alkalosis as primary disorder is calculated from the equation 1.2. [12] Again, like in the previous case, the concentration of bicarbonate is the one we have from the patient's blood gas analysis.

1.3. Expected PaCO2= (0.7 x HCO3⁻) +20 (± 2)

In the situation where the PaCO2 from the analysis is the same as the expected one, the patient has <u>compensated metabolic alkalosis</u>. If the measured PaCO2 exceeds the expected one, there is no compensation and metabolic alkalosis occurs as the first disorder and the respiratory acidosis as the second one. If the measured PaCO2 is lower than the expected one, then the patient has primary metabolic alkalosis with secondary respiratory alkalosis.

Respiratory acidosis

Compensation for raised PaCO2 in respiratory acidosis is the increased reabsorption of HCO3in the kidney proximal tubules. [5] The relationship is analyzed by calculating Δ pH/ Δ PaCO2. The results of Δ pH/ Δ PaCO2 or (7.40-measured pH) / (40-measured PaCO2) are then compared to given:



- ΔpH/ΔPaCO2
- If the result is > 0.008 then there is associated metabolic acidosis
- If the result is between 0.003-0.008 that is compensated respiratory acidosis
- If the result is < 0.003 there is associated metabolic alkalosis

If the result of $\Delta pH/\Delta PaCO2$ is 0.003-0.008 the patient has <u>partially compensated respiratory</u> <u>acidosis</u>. If the calculation is greater than 0.008 then there is respiratory acidosis with secondary metabolic acidosis. In the situation where $\Delta pH/\Delta PaCO2$ is less than 0.003 the patient has respiratory acidosis with associated metabolic alkalosis.

Respiratory alkalosis

Compensation in respiratory alkalosis is decreased reabsorption of HCO3⁻ in the kidney with decreased concentration in plasma HCO3⁻. As in the case of respiratory acidosis, this compensation is slow and needs time to reach its full realization. The respiratory disorder will be partially compensated during the time of compensation. The calculation of the Δ pH/ Δ PaCO2 is again accomplished with normal values of pH of 7.40 and PaCO2 of 40mmHg and the results are compared to those given:

REFERENCES

- Toftegaard M, Rees S, Andreassen S. Correlation between acid-base parameters measured in arterial blood and venous blood sampled peripherally, from vena cavae superior, and from the pulmonary artery. Eur J Emerg Med 2008; 15(2): 86-91. doi: 10.1097/MEJ.0b013e3282e6f5c5.
- Byrne A, Bennett M, Chatterji R, Symons R, Pace N, Thomas P. Peripheral venous and arterial blood gas analysis in adults: are they comparable? A systematic review and meta-analysis. Respirology 2014; 19(2): 168-175. doi: 10.1111/resp.12225.
- 3. De Backer D. Detailing the cardiovascular profile in shock patients. Critical Care 2017; 21(Suppl 3):36-41. doi: 10.1186/s13054-017-1908-6.
- Tipping R, Nicoll A. Mechanisms of hypoxaemia and the interpretation of arterial blood gases. Surgery 2018; 36 (12): 675-681. doi.org/10.1016/j.mpsur.2018.10.001.
- Marino P. Algorithms for acid-base interpretations. In: The ICU Book. 1st ed. Pennsylvania, Kent: Lea & Febiger; 1991. p. 415-26.

- $\Delta pH/\Delta PaCO2$
- If the result is > 0.008 then there is associated metabolic alkalosis
- If the result is between 0.002-0.008 that is compensated respiratory alkalosis
- If the result is < 0.002 there is associated metabolic acidosis

If the $\Delta pH/\Delta PaCO2$ result is between 0.002-0.008 the patient has <u>partially compensated</u> <u>respiratory alkalosis</u>. If the calculation is greater than 0.008 then there is respiratory alkalosis with secondary metabolic alkalosis. In the situation where $\Delta pH/\Delta PaCO2$ is less than 0.002 the patient has respiratory alkalosis with associated metabolic acidosis.

CONCLUSION

This was just one way of interpreting results of the acid-base analysis. For complete comprehensive interpretation clinical anamnesis and examination with history of illness are of utmost importance. New elements of acid-base analysis like Lactate, Alb and electrolytes are additional parameters which can be analyzed. All of the previous enhance diagnostic process and better treatment plan of diseases.

- Emmet M, Narins RG. Clinical use of the anion gap. Medicine 1977; 56: 38-54.
- Cassaleto JJ. Differential diagnosis of metabolic acidosis. Emerg Med Clin North Am.2005; 23: 117-62. doi: 10.1016/j.emc.2005.03.007.
- Lim S. Metabolic acidosis. Acta Med Indones 2007; 39 (3): 145-50.
- Winter D, Pearson R, Gabow A, Schultz L, Lepoff B. The fall of the serum anion gap. Arch Intern Med 1990; 150: 311-3. doi:10.1001/archinte.1990.00390140057012.
- Marino P, Sutin K. Acid-base interpretations. In: The ICU Book. 3rd ed. Philadelphia: Lippincot Williams&Wilkins; 2007. p. 531-45.
- Albert M, Dell R, Winters R. Quantitative displacement of acid-base equilibrium in metabolic acidosis. Ann Intern Med 1967; 66: 312-5. doi:10.7326/0003-4819-66-2-312.
- Javaheri S, Kazemi H. Metabolic alkalosis and hypoventilation in humans. Am Rev Respir Dis 1987; 136: 1011-6. doi:10.1164/ajrccm/136.4.1011.



ISPITIVANJE POREMEĆAJA ACIDOBAZNOG STATUSA

Natalija Vuković¹, Milan Elenkov²

¹Klinički centar Niš, Klinika za anesteziju i intenzivnu terapiju, ²Opšta bolnica Pirot, Odeljenje anestezije i intenzivne terapije, Pirot

Sažetak: Ispitivanje poremećaja acidobaznog statusa predstavlja važan dijagnostički metod u lečenju čitavog niza oboljenja. Pravovremeno i adekvatno lečenje poremećaja acidobaznog statusa može dovesti do spašavanja života i krucijalnih promena u toku lečenja. Iako je napredak tehnologije doveo do unapređenja kvaliteta i obima informacija dobijenih iz jedinstvenog uzorka krvi za acidobazni status, analiza acidobaznog poremećaja predstavlja izazov za mnoge lekare različitih specijalnosti. Fiziološki metod interpretacije acidobaznih poremećaja koristi kao osnovu sistem ugljena kiselina-bikarbonat. Na osnovu ovog sistema, promene u parcijalnom pritisku ugljen dioksida dovođe do promena u koncentraciji bikarbonata na određen svrsishodan način i obrnuto. U fiziološkom metodu interpretacije postoje dva osnovna metabolička i dva respiratorna poremećaja. *Ključne reči:* acidobazni, acidoza, alkaloza

Korespondencija/Correspondence

Natalija VUKOVIĆ, MD, PhD

Clinical Centre Niš Clinic for Anesthesiology and Intensive Care Bulevar Zorana Djindjića 48, Niš e-mail: <u>massha.vukovic@gmail.com</u>

www.seejournal.rs seejournal.office@gmail.com