Acid-Base Disorders

Clinical Evaluation and Management

Alluru S. Reddi

Springer

Acid-Base Disorders

Alluru S. Reddi

Acid-Base Disorders

Clinical Evaluation and Management

Springer

Alluru S. Reddi
Professor of Medicine, Department of Medicine, Chief, Division of Nephrology and Hypertension
Rutgers New Jersey Medical School
Newark, NJ, USA

ISBN 978-3-030-28894-5 ISBN 978-3-030-28895-2 (eBook)
https://doi.org/10.1007/978-3-030-28895-2

This Springer imprint is published by the registered company Springer Nature Switzerland AG
The registered company address is: Gewerbestrasse 11, 6330 Cham, Switzerland

Preface

Acid-base physiology is a difficult topic in medicine because of its complexity. The purpose of writing this book is to present a clear and concise understanding of the fundamentals of acid-base physiology and its associated disorders that are frequently encountered in clinical practice. Each clinical acid-base disorder begins with pathophysiology followed by case studies and questions with explanations. I believe that this kind of approach will increase the knowledge of a physician in managing acid-base disturbances.

This book would not have been possible without the help of many students, house staff, and colleagues who made me understand acid-base disorders and manage patients appropriately. I am grateful to all of them. I am extremely thankful and grateful to my family for their immense support and patience. I extend my thanks to Andy Kwan and Gregory Sutorius, Springer, New York, for their continued support, help, and advice. Constructive critique for improvement of the book is gratefully acknowledged.

Newark, NJ, USA — Alluru S. Reddi

Contents

Chapter 1
Introduction to Acid–Base

The arterial blood gas (ABG) determination is an important laboratory test in the evaluation of oxygenation and acid–base status of the body. This ABG test is most frequently done in the emergency department and critical care units. Also, this test is a valuable tool during operative procedures. When an ABG is ordered, four important values are reported: pH, partial pressure of oxygen (pO_2), partial pressure of carbon dioxide (pCO_2), and bicarbonate (HCO_3^-). Base excess (BE) is also reported (see Chap. 3), which is used by some clinicians. The percent saturation of hemoglobin with oxygen in the arterial blood (SaO_2) is done by either direct measurement using CO-oximetry or estimated from pO_2. Only some blood gas analyzers are equipped with CO-oximeter for measurement of SaO_2 directly, and other laboratories report calculated value. Mean SaO_2 is 98%.

Technique of ABG Measurement

After collection of either arterial or venous blood, it is introduced into the blood gas analyzer (BGA). The BGA aspirates the blood into a measuring chamber which contains ion-specific electrodes for pH, pO_2, and pCO_2. The pH is measured by two electrodes: a pH-measuring electrode and a reference electrode. The reference electrode contains a saturated solution of KCl, and the current flow compares the voltage of the unknown blood with a reference voltage, and the difference in voltage is displayed on a voltmeter calibrated in pH units.

pO_2 is measured with Clark electrode or polarographic electrode. O_2 diffuses across polypropylene membrane through the electrode immersed in phosphate buffer. O_2 then reacts with water in the buffer and generates voltage (current) that is proportional to the number of O_2 molecules in the solution. The current is measured and expressed as pO_2.

A. S. Reddi, *Acid-Base Disorders*, https://doi.org/10.1007/978-3-030-28895-2_1

The pCO_2 electrode is a modified pH electrode with a silicone or Teflon rubber CO_2 semipermeable membrane covering the tip of the electrode. The electrode is bathed in a solution containing $NaHCO_3$. The CO_2 diffuses from the blood across the semipermeable membrane, and the reaction between CO_2 and water generates free H^+ in proportion to the pCO_2

A brief description of each of these components of ABG is described below.

pH

pH is measured by a specific pH electrode, and it indicates either acidity or alkalinity of blood. Actually the pH is an indirect measurement of hydrogen ion concentration (abbreviated as $[H^+]$).The normal $[H^+]$ in the extracellular fluid is about 40 nmol/L or 40 nEq/L (range 38–42 nmol/L), which is precisely regulated by an interplay between body buffers, lungs, and kidneys. Since many functions of the cell are dependent on the optimum $[H^+]$, it is extremely important to maintain $[H^+]$ in blood ~40 nmol/L. Any deviation from this $[H^+]$ results either in acidemia ($[H^+]$ >40 nmol/L) or alkalemia ($[H^+]$ <40 nmol/L). The $[H^+]$ in blood is so low that it is not measured routinely. However, the $[H^+]$ is measured as pH, which is expressed as:

$$pH = -\log\left[H^+\right] \tag{1.1}$$

Thus, pH is defined as the negative logarithm of the $[H^+]$. An inverse relationship exists between pH and $[H^+]$. In other words, as the pH increases, the $[H^+]$ decreases and vice versa (Table 1.1). Cells cannot function at a pH below 6.8 and above 7.8. The normal arterial pH ranges from 7.38 to 7.42, which translates to a $[H^+]$ of 38–42 nmol/L. Mean pH is 7.40.

Table 1.1 Relationship between pH and $[H^+]$

pH (Units)	$[H^+]$ (nmol/L)
7.90	13
7.80	16
7.70	20
7.60	25
7.50	32
7.40	40
7.30	50
7.20	63
7.10	79
7.00	100
6.90	126
6.80	159
6.70	199
6.60	251

pO_2

pO_2 refers to the partial pressure of oxygen (tension) dissolved in blood. As mentioned, it is measured specifically by a pO_2 electrode. The mean value of pO_2 in a normal young man is approximately 97 mmHg at sea level. Various formulas have been developed to predict approximate values of pO_2 in individuals of varying ages. Clinically, however, it is cumbersome to use these formulas on daily basis. One suggested way of estimating approximate pO_2 is to assume 100 mmHg in a 10-year-old child and a decrease of 5 mmHg for every 10 years up to 90 years of age. For example, a 20-year-old man will have a pO_2 of 95 mmHg, and it is 60 mmHg for a 90-year-old man.

pCO_2

pCO_2 indicates the partial pressure of carbon dioxide (tension) dissolved in blood. It reflects alveolar ventilation and represents respiratory component of ABG. The normal values range from 35 to 46 mmHg with a mean value of 40 mmHg.

HCO_3^-

HCO_3^- represents the bicarbonate concentration ($[HCO_3^-]$) of the blood sample that is sent for the analysis of ABG. It is not a measured value but calculated from Henderson–Hasselbalch equation (see Chap. 2). This calculated $[HCO_3^-]$ is lower by 1–2 mEq/L than the $[HCO_3^-]$ from chemistry panel, which is measured as total CO_2. Total CO_2 comprises three components: HCO_3^-, dissolved CO_2, and carbonic acid. For this reason, total CO_2 concentration is higher than the calculated $HCO3^-$. Total CO_2, calculated HCO_3^-, and base excess (see Chap. 3) are indicators of metabolic components of ABG.

Normal ABG Values

Mean and range values of normal ABG are shown in Table 1.2.

Table 1.2 Mean and range values of normal ABG

Component	Mean	Range
pH	7.40	7.36–7.44
$[H^+]$ (nnol/L)	40	36–44
pO_2 (mmHg)	97	80–100
pCO_2 (mmHg)	40	36–44
$[HCO_3^-]$ (mEq/L)	24	22–26
BE (mEq/L)	0	0 ± 2
SaO_2 (%)	97	97–98

BE base excess, *SaO_2* saturation of hemoglobin with oxygen

Arterial vs. Venous Blood Sample for ABG

Arterial blood is used most of the time to evaluate an acid–base disorder. However, venous blood samples can be used because there is insignificant difference in ABG values between these two samples (Table 1.3).

Although there is not much difference between the two samples in normal individuals, significant difference can be observed in pathological conditions. For example, large arteriovenous difference can be found in a patient with decreased cardiac output and on mechanical ventilation. In such a patient, the arterial pCO_2 remains normal, but central venous pCO_2 may be extremely elevated, as more CO_2 is added to the perfusing tissue. In low cardiac output states, an arterial ABG is useful in assessing pulmonary gas exchange, and central venous ABG is useful in assessing pH and tissue oxygenation.

Primary Acid–Base Disorders

As stated above, a change in plasma $[HCO_3^-]$ results in a metabolic acid–base disturbance, whereas a change in arterial pCO_2 results in a respiratory acid–base disorder. Clinically, four primary acid–base disorders can be recognized: (1) *metabolic acidosis*, (2) *metabolic alkalosis*, (3) *respiratory acidosis*, and (4) *respiratory alkalosis*. Changes in pH, HCO_3^-, and pCO_2 for each primary acid–base disorder are shown in Table 1.4. In addition, the respiratory acid–base disorders are classified into either acute or chronic based on the buffering mechanism. Buffering for acute disorder is complete in minutes to few hours, whereas for chronic disorder complete buffering takes a few days (see Chap. 2). Systemic disorders cause primary acid–base disorders, and the resultant pH changes are minimized by appropriate secondary physiologic response, as shown in Table 1.5.

Table 1.3 Differences between arterial and venous blood samples

ABG value	Arterial blood	Venous blood
pH	7.40	7.36
$[H^+]$ (nmol/L)	40	44
pCO_2 (mmHg)	40	48
$[HCO_3^-]$ (mEq/L)	24	26
pO_2 (mmHg)	~97	~50

Table 1.4 Primary acid–base disturbances

Acid–base disorder	pH	Primary change
Metabolic acidosis	<7.40	↓ HCO_3^-
Metabolic alkalosis	>7.40	↑ HCO_3^-
Respiratory acidosis	<7.40	↑ pCO_2
Respiratory alkalosis	>7.40	↓ pCO_2

↑ increase ↓ decrease