© 2009 International Society of Nephrology

Assessing acid–base disorders

Horacio J. Adrogué[1,2,3], F. John Gennari[4], John H. Galla[5] and Nicolaos E. Madias[6,7]

[1]Department of Medicine, Baylor College of Medicine, Houston, TX, USA; [2]Department of Medicine, Methodist Hospital, Houston, TX, USA; [3]Renal Section, Veterans Affairs Medical Center, Houston, TX, USA; [4]Department of Medicine, University of Vermont College of Medicine, Burlington, VT, USA; [5]Department of Medicine, University of Cincinnati College of Medicine, Cincinnati, OH, USA; [6]Department of Medicine, Tufts University School of Medicine, Boston, MA, USA and [7]Division of Nephrology, Department of Medicine, Caritas St Elizabeth's Medical Center, Tufts University School of Medicine, Boston, MA, USA

Effective management of acid–base disorders depends on accurate diagnosis. Three distinct approaches are currently used in assessing acid–base disorders: the physiological approach, the base-excess approach, and the physicochemical approach. There are considerable differences among the three approaches. In this review, we first describe the conceptual framework of each approach, and comment on its attributes and drawbacks. We then highlight the application of each approach to patient care. We conclude with a brief synthesis and our recommendations for choosing an approach.

Kidney International (2009) **76,** 1239–1247; doi:10.1038/ki.2009.359; published online 7 October 2009

KEYWORDS: base-excess approach; physicochemical approach; physiological approach; Stewart approach

Correspondence: *Nicolaos E. Madias, Department of Medicine, Caritas St Elizabeth's Medical Center, 736 Cambridge Street, Boston, MA 02135, USA. E-mail: nicolaos.madias@caritaschristi.org*

Received 24 June 2009; revised 14 July 2009; accepted 14 July 2009; published online 7 October 2009

Management of acid–base disorders begins with accurate diagnosis, a process requiring two tasks: First, reliable measurement of acid–base variables in the blood, a complex fluid containing multiple ions and buffers; this task is an exercise in chemistry. Second, proper interpretation of the data in relation to human health and disease allowing definition of the patient's acid–base status; this is an exercise in pathophysiology. The patient's history, physical examination, and additional laboratory testing and imaging, as appropriate, then help the clinician to identify the specific cause(s) of the acid–base disturbance, and from that information to undertake appropriate intervention.[1]

Three distinct approaches are currently used in assessing acid–base disorders, each with a considerable following worldwide. For the purposes of this review, we name them the physiological approach, pioneered by Van Slyke and co-workers;[2,3] the base-excess approach, developed by Astrup and co-workers;[4,5] and the physicochemical approach, proposed by Stewart and extended by his followers.[6–9] The last and newest approach has steadily gained acceptance, especially among critical-care physicians and anesthesiologists.

The three approaches differ considerably. In this review, we first describe the conceptual framework of each approach, and its attributes and drawbacks. We then highlight the application of each approach to patient care. We conclude with a brief synthesis and our recommendations for choosing an approach.

PHYSIOLOGICAL APPROACH

Conceptual framework

The physiological approach considers acids as hydrogen ion (H^+) donors and bases as H^+ acceptors.[10] It uses solely the carbonic acid/bicarbonate buffer system for assessing acid-base status, a position rooted in the isohydric principle. Adoption of this buffer system reflects its abundance, physiological preeminence, and the fact that its two components undergo homeostatic control.[1–3] Blood pH is viewed as being determined by the prevailing levels of carbonic acid (that is, $PaCO_2$, the respiratory component) and plasma bicarbonate concentration ($[HCO_3^-]$, the metabolic component, Table 1), as stipulated by the Henderson equation, $[H^+] = 24 \times PaCO_2/[HCO_3^-]$.

Table 1 | Assessment of the metabolic component of acid-base status

Approach	Variable	Determination	Remarks
Physiological	Plasma [HCO_3^-]	Measured pH and PCO_2	Interpretation complemented by evaluation of plasma anion gap, $[Na^+]-([Cl^-]+[\text{Total } CO_2])$
Base excess	Blood base excess (BE)	CO_2 equilibration method or calculated from measured pH and PCO_2	BE is a measure of the metabolic component of acid–base status as reflected in whole blood Interpretation complemented by evaluation of plasma anion gap
	Standard BE (SBE)	Calculated from measured pH, PCO_2, and hemoglobin	SBE is a measure of the metabolic component of acid-base status as reflected in the extracellular compartment. It is usually calculated automatically from arterial blood gas results, but it can also be obtained using the blood acid–base nomogram with the hemoglobin set at 5 g/dl[42] Interpretation complemented by evaluation of plasma anion gap
Physicochemical	SID_a (apparent strong ion difference)	$([Na^+]+[K^+]+[Ca^{++}]+[Mg^{++}])-([Cl^-]+[\text{lactate}^-])$ $([Na^+]+[K^+])-([Cl^-]+[\text{lactate}^-]+[\text{other strong anions}])$ $([Na^+]+[K^+])-[Cl^-]$	These three formulas for SID_a, as well as additional variants, are currently in use. SID_a is mathematically equivalent to the plasma buffer base of Singer and Hastings[64]
	SID_e (effective strong ion difference)	$[HCO_3^-]+[Alb^-]+[Pi^-]$ where: $[Alb^-]=[\text{Alb, g/l}]\times[(0.123\times pH)-0.631]$ $[Pi^-]=[\text{Pi, mmol/l}]\times[(0.309\times pH)-0.469]$	Represents the sum of plasma [HCO_3^-] and non-bicarbonate buffers (anionic equivalency of albumin and phosphate)
	SIG (strong ion gap)	$SID_a - SID_e$	An estimate of the concentration of unmeasured anions in plasma that resembles the plasma anion gap Value depends upon the variant of SID_a used
	A_{Tot} (total concentration of weak acids in plasma)	$2.43\times$ [total protein, g/dl]	Primarily related to albumin concentration For clinical purposes, approximated by the concentration of total protein

All variables and electrolytes listed are expressed in mEq/l, unless otherwise indicated.

The physiological approach recognizes four acid–base disorders[1,11–13] (Table 2). Metabolic disorders are expressed as primary changes in plasma [HCO_3^-], whereas respiratory disorders are expressed as primary changes in $PaCO_2$. Each primary change in either plasma [HCO_3^-] or $PaCO_2$ elicits *in vivo* a secondary response in the other variable that tends to minimize the change in acidity.[1,11] These secondary responses, otherwise referred to as compensatory, have been quantitated in animals and humans.[14–24] We discourage use of the term compensatory, because the secondary responses occasionally can yield a maladaptive effect on blood pH.[25,26] Absence of an appropriate secondary response denotes the co-existence of an additional simple acid–base disorder. Use of ventilator support in critically ill patients can, of course, alter or prevent expression of the secondary changes in $PaCO_2$ in response to metabolic acid–base disorders. These ventilator-induced alterations are viewed as complicating primary respiratory acid–base disorders. The simultaneous presence of two or more simple acid–base disorders defines a mixed acid–base disorder.

Assessment of the metabolic component is complemented by evaluating the plasma anion gap (AG), defined as $[Na^+]-([Cl^-]+[TCO_2])$,[27] where $[TCO_2]$ indicates venous total CO_2 concentration (Table 1 and Figure 1). The average normal value for plasma AG differs among health-care facilities because of methodological variation.[27] Normally, approximately 75% of the plasma AG is determined by plasma albumin concentration.[27] Thus, the plasma AG must be adjusted by subtracting or adding 2.5 mEq/l from the calculated value for each 1 g/dl of plasma albumin below or above the average normal value of 4.5 g/dl, respectively. Changes in blood pH elicit small, directional changes in the anionic charge of plasma albumin and thus the AG, but these changes are ignored in clinical practice.[28,29] The anionic charge of plasma albumin decreases by only 1.5 mEq/l when blood pH decreases from 7.40 to 7.10.[30]

Attributes and drawbacks

The physiological approach considers the acid–base status of body fluids as being determined by net H^+ balance (that is, influx minus efflux) and the prevailing complement of body buffers.[31,32] The chemistry of acids and bases is blended with the empirically derived secondary responses of the intact organism to primary changes in $PaCO_2$ or plasma [HCO_3^-]. This approach is simple regarding data collection and clinical application. The standard blood gas analyzer measures pH and PCO_2, from which plasma [HCO_3^-] is calculated (Table 1). Comparing plasma [HCO_3^-] with measured $[TCO_2]$ in venous blood validates this derived variable.[1] Furthermore, most acid–base disorders are first recognized by clinicians through abnormalities in venous $[TCO_2]$.

Although PCO_2 is universally considered as an appropriate index of the respiratory component, plasma [HCO_3^-] has been viewed by some as an unsuitable indicator of the metabolic component.[33,34] Criticisms include lack of independence of plasma [HCO_3^-] from the respiratory component and failure of quantitation of buffers other than bicarbonate. Plasma [HCO_3^-] is certainly affected by changes

Download English Version:

https://daneshyari.com/en/article/3884114

Download Persian Version:

https://daneshyari.com/article/3884114

Daneshyari.com