EDITORIAL

Arterial Blood Gas Analysis: A New Look at the Old Formula

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Arterial blood gas (ABG) analysis is one of the most common performed investigations in the intensive care unit (ICU) worldwide. Historically, it was mainly performed to assess oxygenation status and the severity of respiratory failure was classified depending on the partial pressure of oxygenation (PaO_2)/fraction of inspired oxygen (FiO_2) ratio. Pulse oximetry and oxygen saturation measured by pulse oximetry (SpO_2) as a reliable surrogate for PaO_2 has been validated and more commonly used Berlin.[1] In fact, there has been a suggestion to modify the current Berlin definition and convert PaO_2/FiO_2 to SpO_2/FiO_2 ratio to classify the severity of hypoxemia with <300 equivalent of the former to <350 for the latter, though this has not become a standard practice may be due to some limitations of pulse oximetry readings.[2] Arterial blood gas analysis nowadays is more commonly performed to detect acid-base disturbances in intensive care unit (ICU) patients. Both metabolic and respiratory acidosis and alkalosis are common in these patients. Analysis of these metabolic and respiratory disturbances should not remain an academic exercise only and identifying simple or mixed disturbances should help clinicians to suspect underlying pathophysiologic processes and intervene with appropriate therapy in a timely fashion. Customarily ABG analysis for acid-base disturbance has been performed by using the Boston compensation method, which takes into account pH, partial pressure of carbon dioxide (pCO_2), and actual bicarbonate (HCO_3) into consideration and applies equations to identify simple or mixed metabolic or respiratory disturbances. Another way of approaching this problem is to interpret base excess (Deficit) which to calculated by the ABG analyzer by normalizing the ABG for CO_2 of 40 mm Hg, and calculating the resultant HCO_3 value (standard bicarbonate) and compare this with the observed bicarbonate value (Copenhagen method).[3] In acute respiratory disturbance (acidosis or alkalosis) the base deficit is minimal whereas in pure metabolic disturbances, the delta bicarbonate (normal minus observed bicarbonate) should be roughly equal to base deficit (excess). In chronic respiratory disorders, a correction formula is applied to base excess (deficit) to arrive at the predicted bicarbonate value which is compared to the actual value to determine underlying mixed metabolic disturbance.[4] Over the last few years, Stewarts strong ion difference approach has been applied to ABG analysis based on sodium and chloride. A difference of less than 40 mEq/L between these ions reflects metabolic acidosis, and the gap has to be corrected by bicarbonate, albumin, and phosphate values. If not corrected fully one needs to look at unmeasured anions like lactate, ketones, etc. A difference of more than 40 mEq/L between sodium and potassium reflects metabolic alkalosis.[5] Due to complexity of this equation it has not been practiced widely. These various ways of analyzing ABG reflect room for improvement and devising more novel methods of bedside application of ABG analysis.[6]

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In this issue of the journal, the authors have utilized a novel approach to ABG analysis by analyzing change in pH which is equivalent to the sum of changes in the respiratory and non-respiratory (metabolic) components of pH.[6] Henderson–Hasselbalch equation (HHE) has been utilized in ABG analysis to predict the accuracy of the ABG results and to detect errors in machine measurements. HHE calculates hydrogen ions (H^+) by multiplying 24 into CO_2 and dividing the results with measured bicarbonate.

$H^+ = 24 \times PaCO_2/HCO_3$. The hydrogen ion is subsequently converted to pH which is a logarithm of H^+.[6] In order to determine the non-respiratory (metabolic) component of pH the authors have utilized HHE and replaced observed carbon dioxide (CO_2) with standard CO_2 (40) and replaced observed bicarbonate with standard bicarbonate (SB), which is derived from ABG to calculate base deficit (excess). Thus the formula comes to $24 \times 40/SB = 960/SB$. The H^+ derived from this equation denotes the metabolic component of acid-base balance. This H^+ is then converted to pH and the difference between this and normal pH (7.4) denoted by delta non-respiratory pH (NRpH) will denote the metabolic component of the equation. If positive it denotes metabolic alkalosis and if negative it denotes metabolic acidosis. A delta pH is calculated by noting the difference between the observed pH from the ABG and the normal pH.[7] As delta pH is a summation of metabolic and respiratory component, the delta respiratory component of acid-base (RpH) is then derived by subtracting delta NRpH and Delta pH. If positive it denotes respiratory alkalosis, if negative it denotes respiratory acidosis. Thus in three steps of calculation, one can have a reasonable idea of the respiratory and/or metabolic contribution to the acid-base imbalance. The authors have given a few case examples to illustrate their point.

As clinicians are averse to mathematical calculations, changing H^+ to pH may be a hindrance to the common use of this approach. Tables and nomograms are available to help in these conversions, but then it will add to the complexity of the procedure.[8] A simpler way of arriving at pH is by subtracting H^+ with 80, which will

denote the last two digits of pH. For example, normal H^+ is 40 which when subtracted from 80 comes to 40 which denotes the last two digits of normal pH 7.40. This formula is usually applicable in the physiological ranges of pH observed in clinical practice. Thus adding this equation to the authors' formula may ease the calculation of pH from H^+. Another limitation of the described approach is to differentiate acute and chronic respiratory disorders and one needs to go back to Boston compensation rules to estimate the time period of compensation. In clinical practice though, even when compensation rules are applied, they can not differentiate acute on chronic respiratory disorders, partially compensated chronic disorder, acute disorder of some duration and thus one has to fall back on clinical history and examinations to determine these. Thus, the information on respiratory acidosis or alkalosis may suffice for clinical practice, and the rest obtained from clinical presentation. Another difficulty in analyzing this formula is to determine presence of mixed and complicated disorder, especially triple disorder with anion and non-anion gap metabolic acidosis along with metabolic alkalosis and respiratory disturbances as may be seen in diabetic ketoacidosis and vomiting with aspiration, resuscitated with a large amount of normal saline. These patients can have anion gap metabolic acidosis due to ketosis, non-anion gap acidosis due to hyperchloremia induced by saline resuscitation, metabolic alkalosis due to vomiting and respiratory acidosis due to aspiration pneumonia. One will need to apply conventional Boston compensation rules and delta anion gap, delta bicarbonate gap, and Gap/Gap ratio to calculate underlying triple disorders.

The simple rules described by authors for ABG analysis need to be utilized in clinical practice and need to be compared with other conventional methods of analysis to know the accuracy and the time taken to arrive at the interpretation. Clinical application of the results in patient care and any change in management also need to be noted. Lastly, with the advent of the increasing use of artificial intelligence and machine learning in many other diagnostic areas like electrocardiogram interpretation, echocardiogram analysis, chest X-ray interpretation, and histology interpretation, hopefully, in built we will soon have a smart ABG analyzer which will have a built algorithm to describe the results and use the above-described tools for acid-base analysis. Finally, it will be left to us clinicians to interpret it with the help of natural intelligence to decide on the applicability of the results to the patient we are caring for.

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