Clinical problems in acid-base balance: Parts I and II
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This article concludes the AANA Journal's presentation of the Postgraduate Course which was offered at the 43rd AANA Annual Meeting held in San Francisco during August, 1976. In the first part of the article, the author discusses the concepts of $T_{\text{40}}$ bicarbonate, which afford a simplified approach to acid-base balance. In the second part of the article, the author relates this methodology to actual clinical applications.

Part I: $T_{\text{40}}$ bicarbonate—A simplified approach to acid-base balance

The general availability of modern blood gas electrode systems has brought the diagnosis and treatment of clinical acid-base problems from the dark ages to the scientific approach available today. In 1966, Armstrong, et al.\textsuperscript{1} described the use of the in vivo carbon dioxide titration curve based on a measurement of a single arterial blood sample. Utilizing this system of determining extracellular $T_{\text{40}}$ bicarbonate, I shall attempt to simplify the approach to clinical acid-base problems for you. This is a "bedside approach" which has been successfully used in instruction at the LAC-USC Medical Center.

To begin with, let us lay the ground rules by some simple definitions and terminology. These may be at variance with those that have been previously presented to you, but for purposes of this presentation, let us accept them.

$\text{Acid}$. A proton donor. In solution an acid dissociates into $\text{H}^+$ plus a neutral molecule or an anion (called a base).

$\text{Base}$. An acceptor of $\text{H}^+$. In solution, a base can combine chemically with a hydrogen ion. A base neutralizes the effects of hydrogen ions by removing them from solution. As a result, the proton becomes part of another molecule.

Conjugate bases and acids. A conjugate base is an ion or neutral molecule formed when an acid dissociates in solution. A conjugate acid is an ion or neutral molecule formed when a base combines chemically with $\text{H}^+$. In other words, when an acid dissociates it yields a $\text{H}^+$ plus a conjugate base. When a base associates with a $\text{H}^+$ it forms a conjugate acid.

Weak acids versus strong acids. A strong acid dissociates almost completely into a hydrogen ion and its conjugate base when in solution. There are virtually no conjugate acid molecules remaining in solution. Hydrochloric acid is an example of a strong acid. A weak acid does not dissociate completely when in solution. Therefore, a solution of a weak acid contains hydrogen ions, conjugate base, and conjugate acid. Examples of weak acids are carbonic acid, acid hemoglobin, acid proteins, and ammonium ion. Some weak acids are
stronger than others and therefore release greater concentrations of hydrogen ions when in solution.

Volatile acids versus non-volatile acids (fixed acids). A volatile acid is an acid for a chemical product of the acid that can be excreted from the body as a gas. Carbonic acid is excreted from the body as CO₂.

\[ \text{H}_2\text{CO}_3 \rightleftharpoons \text{CO}_2 + \text{H}_2\text{O} \]
Carbon dioxide is excreted from the lungs during ventilation. A non-volatile acid is an acid that must be excreted from the body in water. All acids found in the body fluids other than carbonic acid are fixed acids and are excreted by the kidney.

Buffers. Buffers are substances that absorb some of the changes in hydrogen ion concentration that occur when acids or bases are added to, or are removed from the body fluids. Chemical buffers must be able to combine with or yield \( \text{H}^+ \) in response to appropriate body fluid composition. Buffers do not prevent a change in pH from occurring, rather, they reduce the magnitude of change that would have occurred without the buffers. Buffers in body fluids are conjugate acid-base pairs of weak acids.

The body has three modes of maintaining the acidity of body fluids within a normal range:

1. Buffer action.
   a. Of blood and extracellular fluids.
   b. Of cells.
2. By removal of \( \text{CO}_2 \) through respiration.
3. By the generation of bicarbonate in the kidney in the process of removing fixed acids in the urine.

Every day the body produces large quantities of acids as a result of chemical reactions of metabolism and digestion. In order to maintain acid-base balance, those acids which are not used in metabolism must be eliminated from the body at the same rate at which they are produced.

The largest amount of acid produced is the volatile carbonic acid. Approximately 13,000 millequivalents of \( \text{H}_2\text{CO}_3 \) are produced and must be excreted each day. Additionally, approximately 100 millequivalents of unusable fixed acids are produced and excreted each day.

These acids are produced as end products of metabolism in tissues throughout the body, but are excreted at only two sites, the lungs and the kidneys. They are buffered before being transported to the areas of excretion.

Body buffer systems. The most important buffering system is protein. Most protein is intracellular. Hemoglobin of the red blood cell is particularly important and exchanges hydrogen ions rapidly with the plasma. Extracellular protein is contained in the plasma but is not a particularly large portion of the body buffering power. The second most important buffering system is the bicarbonate-carbonic acid system. This is important only because \( \text{CO}_2 \) is volatile and can be removed from the system by ventilation. Phosphate and other buffer systems do exist but quantitatively are far less important than the two previously mentioned.

The most vital theory in our understanding is that each of the buffer systems will be in equilibrium with each other in the same compartment of the body as the \( \text{H}^+ \) concentration is uniform within that compartment. If the pH is uniform throughout the extracellular fluid, then all of the extracellular buffers must be in equilibrium with each other. This means we can evaluate all the acid-base systems by completely evaluating only one buffer system, usually the bicarbonate-carbonic acid system.

Volatile acids (\( \text{CO}_2 \)) are buffered by both the bicarbonate-carbonic acid and the protein-proteinate systems. Fixed acids, however, are buffered only by the bicarbonate-carbonic acid system. Therefore:

1. The protein-proteinate system buffers only volatile acids.
2. The bicarbonate-carbonic acid system buffers both volatile and non-volatile acids.
**CO₂ Transport.** CO₂ produced at the site of metabolism must be continuously transported to the lungs for excretion. There are three methods of transportation: (1) 5% is transported by being physically dissolved; (2) 15% is by protein transport; and (3) 80% is transported as bicarbonate.

If respiration is impaired and CO₂ accumulates, it dissolves in the blood and forms carbonic acid (H₂CO₃). However, this excess acid can be buffered by hemoglobin to form acid hemoglobin and bicarbonate.

\[ \text{H}_2\text{CO}_3 + \text{Hb} \rightleftharpoons \text{HHb} + \text{HCO}_3^- \]

Since the amount of bicarbonate liberated equals the amount of hemoglobin buffer utilized, the total buffer anion content of the blood will not be altered by changes in CO₂ content or in PCO₂. Buffer base changes, therefore, represent the effect essentially of metabolic (non-respiratory) disturbances in the body.²,³

The pH is in effect the summation of a balance between respiratory and metabolic components. In evaluating the acid-base status of a blood sample, and therefore indirectly the acid-base status of the body, one must attempt to dissect its components into respiratory and metabolic components. To do this, we must know the pH, PCO₂, and the bicarbonate. If one can correct for the effect of sudden changes in PCO₂ on the bicarbonate, the respiratory component, to establish what the metabolic component is.

Let us first establish normal values for pH, PCO₂, and HCO₃⁻, as well as the appropriate terminology for deviations:

- **pH:** 7.40 (7.35-7.45) = Normal
  - Acidemia < 7.35
  - Alkalalemia > 7.45
  - Note the suffix "-emia," referring to blood values.

- **Respiratory = PCO₂ (Lung)**
  - Normal = 40 TORR (35-45)
  - Respiratory acidosis > 45 TORR
  - Respiratory alkalosis < 35 TORR

**Metabolic = HCO₃⁻ (Kidney)**
- Normal = 24 mM/L (23-26)
- Metabolic acidosis < 23 mM/L
- Metabolic alkalosis > 26 mM/L

The so-called "T₄₀" approach to acid-base problems is easily viewed without complicated concepts and sentences, yet proves to be as useful, if not more so, than any other approach. Clinical acid-base problems can be looked at simply, so that one may seldom err.

The first basic concept one must realize in this approach is that PCO₂ reflects effective ventilation, often referred to as alveolar ventilation. (Alveolar ventilation = Total ventilation – Dead space ventilation).

\[ \dot{V}_A = K \frac{(\dot{V}\text{CO}_2)}{(\text{PCO}_2)} \]

*Where:* \( \dot{V}_A \) is alveolar ventilation

\[ K = \text{A constant which converts PCO₂ to % CO₂, and STPD to BTPS.} \]

\( \dot{V}\text{CO}_2 = \text{Rate of CO₂ production (metabolically controlled and at rest is assumed to be constant).} \)

\( \text{PCO}_2 = \text{estimated pressure of CO₂ in the alveolus (and is assumed equal to arterial tension, and is easily measurable).} \)

Any component that is normal or constant is arbitrarily given the numerical value of "1." Therefore:

\[ K = 1 \]

\( \dot{V}\text{CO}_2 \) is assumed to be constant and \( \dot{V}_A = K \text{ Rate of CO₂ production} \)

The normal PCO₂ at sea level is 40 TORR, therefore PCO₂ may be represented by "1" in the equation:

\[ \dot{V}_A = \frac{1}{\text{PCO}_2} = 1 \]

Thus alveolar ventilation is normal.

If the value for PCO₂ = 80 TORR, then PCO₂ is twice normal and the numerical value is equal to "2." Substituting in the equation for PCO₂:

\[ \dot{V}_A = \frac{1}{2} \]

alveolar ventilation is ½ normal.

If the value of PCO₂ = 20 TORR, the PCO₂ is ½ normal, and substituting in the equation for PCO₂:

\[ \dot{V}_A = \frac{1}{\frac{1}{2}} = 2 \]

and alveolar ventilation is twice normal.
It becomes clear that if one knows the arterial PCO$_2$, the relative effective (alveolar) ventilation is known. Restated, effective or alveolar ventilation is that part of the total pulmonary ventilation which takes part in gas exchange with the pulmonary blood. Knowing the PCO$_2$, then allows one to assess the alveolar ventilation as well as the respiratory acid-base status of the patient.

Since the range of pH in the body in usual clinical situations falls between 7.0 and 8.0, there is no need to utilize the Henderson-Hasselbach equation in the solution of acid-base problems. This obviates the necessity of working with pH and negative logarithms. Instead, we may use the original Henderson equation to evaluate acid-base problems.

Since carbonic acid is the end product of aerobic metabolism in the body it follows:

$$\text{CO}_2 + \text{H}_2\text{O} \longrightarrow \text{H}_2\text{CO}_3 \longrightarrow ([\text{H}^+] \times [\text{HCO}_3^-])$$

But, since CO$_2$ is essentially controlled by alveolar ventilation:

$$K\cdot\text{CO}_2 = [\text{H}^+] \times [\text{HCO}_3^-]$$

a. $$\text{PCO}_2 = \frac{[\text{H}^+] \cdot [\text{HCO}_3^-]}{K}$$

b. $$[\text{H}^+] = \frac{K\cdot\text{PCO}_2}{[\text{HCO}_3^-]}$$

c. $$[\text{HCO}_3^-] = \frac{K\cdot\text{PCO}_2}{[\text{H}^+]}$$

Where $K = 25$ (24.7)

The conversion of pH to [H$^+$] is simple, recalling these facts as anchor points: pH 7.0 = [H$^+$] of 100; pH 7.40 = [H$^+$] of 40; and pH 8.0 = [H$^+$] of 10. We can now construct a table for the remaining values as follows: (For each increase of pH of .3 and [H$^+$] changes by a factor of 2 X.)

<table>
<thead>
<tr>
<th>pH</th>
<th>[H$^+$] (nanoMols/L.)</th>
<th>pH</th>
<th>[H$^+$] (nanoMols/L.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>7.0</td>
<td>100</td>
<td>7.6</td>
<td>25</td>
</tr>
<tr>
<td>7.1</td>
<td>80</td>
<td>7.7</td>
<td>20</td>
</tr>
<tr>
<td>7.2</td>
<td>64</td>
<td>7.8</td>
<td>16</td>
</tr>
<tr>
<td>7.3</td>
<td>50</td>
<td>7.9</td>
<td>12.5</td>
</tr>
<tr>
<td>7.4</td>
<td>40</td>
<td>8.0</td>
<td>10</td>
</tr>
<tr>
<td>7.5</td>
<td>32</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Suppose you received from the laboratory these values on arterial blood in a patient whom you suspected was in ventilatory failure:

$$\text{pH} = 7.0$$

$$\text{PCO}_2 = 100 \text{ TORR}$$

Before an adequate interpretation can be made, we must know the bicarbonate.

Using the Henderson equation and the pH/[H$^+$] conversion table:

$$[\text{HCO}_3^-] = \frac{K\cdot\text{PCO}_2}{[\text{H}^+]} \text{ or } \frac{25 \times 100}{[\text{H}^+]} = 25 \text{ mM/L}$$

We can readily see that the effective ventilation is decreased $2\frac{1}{2}$ times. (PCO$_2 = 100$, Normal = 40). The patient has a marked acidemia (pH = 7.0, Normal = 7.4). However, this is not the total problem that the patient suffers.

Additional pertinent information can be obtained if we could actually, or abstractly make this patient ventilate normally (PCO$_2$ = 40). If this could be done to the patient without damage, then the ventilatory component of the acid-base problem would be normalized, and the values for pH and bicarbonate would represent the "metabolic component". Better yet, if we could somehow visualize these values in our mind, without altering the condition of the patient first, we would be further ahead in the diagnosis and in the ultimate treatment of this patient. Fortunately, we can do just that.

Concept of $T_{40}$ bicarbonate. For an acute change in PCO$_2$ of 15 TORR, the blood alters its bicarbonate level by about 1 millimole$^1$. If one were to lower the PCO$_2$ to 40 TORR acutely, the appropriate change in bicarbonate would represent the metabolic state of bicarbonate balance when the patient was breathing normally.

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If we were to answer the question: "What would be the values for arterial HCO₃⁻ and pH for this patient if he were ventilating normally (PCO₂ = 40)?" We would first have to calculate the change in [HCO₃⁻] that would occur if the PCO₂ were to fall from 100 to 40.

Since for every acute change of 15 TORR in PCO₂, there is a change of 1 millimole of HCO₃⁻, there would be a change of 4 millimoles of HCO₃⁻ if we lowered the PCO₂ from 100 to 40. Thus, the arterial [HCO₃⁻] would be lowered by 4 mM/L if the PCO₂ were quickly lowered by 60 TORR.

We know that the patient would have a PCO₂=40 TORR and a bicarbonate of (25-4) 21. Using the Henderson equation to solve for [H⁺], let us look at Example 1:

**Example 1.**

\[
\frac{[\text{H}^+] \times \text{PCO}_2}{[\text{HCO}_3^-]} = \frac{25 \times 40}{21}
\]

From the pH [H⁺] conversion table, pH = 7.32. Therefore:

<table>
<thead>
<tr>
<th>PCO₂</th>
<th>pH/[H⁺]</th>
<th>[HCO₃⁻]</th>
</tr>
</thead>
<tbody>
<tr>
<td>Given:</td>
<td>100</td>
<td>7.0/100</td>
</tr>
<tr>
<td>If:</td>
<td>40</td>
<td>7.32/48</td>
</tr>
</tbody>
</table>

The missing fact was that even if this patient were breathing effectively, he still would have had a bicarbonate deficit and an acidemia. This patient had severe ventilatory failure complicated by lactic acidosis. The ventilatory component was treated by assisted ventilation and the metabolic component was treated by infusion of bicarbonate solution.

The calculated bicarbonate, 21 mM/L in this case is given the name "T₄₀ bicarbonate" and is defined as that bicarbonate concentration that would be present in the arterial blood of a patient who is ventilating his lungs effectively to maintain a PCO₂ of 40 TORR¹. This concept differs from the standard bicarbonate in that the changes in pH and bicarbonate occur in vivo for T₄₀ rather than in vitro as for the standard bicarbonate. Thus, evolves the expression "in vivo carbon dioxide titration curve"¹. Let us look at Example 2:

**Example 2.**

Given values:

\[
\begin{align*}
\text{PCO}_2 &= 80 \text{ TORR}; \text{pH} = 7.15; [\text{HCO}_3^-] = 7 \\
[\text{HCO}_3^-] &= K \times \text{PCO}_2 = 25 \times 80 = 28 \text{ mM/L} \\
[\text{H}^+] &= \frac{70}{40} \quad \text{(from pH/[H⁺] Table)}
\end{align*}
\]

Interpretation can only be partially complete at this point, until we know the T₄₀ bicarbonate. Effective ventilation has been halved (PCO₂ = 80; normal = 40), and the patient has an acidemia. But is there a bicarbonate deficit? The normal [HCO₃⁻] is 24 mM/L. There seems to be about a 4mM/L excess. Is this bicarbonate excess due to PCO₂ retention or renal bicarbonate retention? To answer this question, we must calculate the T₄₀ bicarbonate.

**Example 3.**

Given values: PCO₂ = 10 TORR; pH = 7.30; [HCO₃⁻] = 7

\[
\begin{align*}
[\text{HCO}_3^-] &= K \times \text{PCO}_2 = 25 \times 10 = 50 \text{ mM/L} \\
[\text{H}^+] &= \frac{50}{40} \quad \text{(from pH/[H⁺] Table)}
\end{align*}
\]

Using pH/[H⁺] Table: pH = 7.40

It becomes immediately clear that there is no bicarbonate excess or deficit and that this patient has acute ventilatory failure. The treatment becomes obvious. Let us look at Example 3:

**Example 3.**

Given values: PCO₂ = 10 TORR; pH = 7.30; [HCO₃⁻] = 7

\[
\begin{align*}
[\text{HCO}_3^-] &= K \times \text{PCO}_2 = 25 \times 10 = 6 \text{ mM/L} \\
[\text{H}^+] &= \frac{6}{40} \quad \text{(from pH/[H⁺] Table)}
\end{align*}
\]

Where: Change in [HCO₃⁻] = (60-40)/15 = 2.7 or 3

and: T₄₀ [HCO₃⁻] = 25

and: [H⁺] = K × PCO₂ = 25 × 40 = 40

[HCO₃⁻] = 25

Therefore, there is a 24 - 7 = 17 mM/L base deficit which was apparently due to diabetic ketoacidosis. If this patient had continued to ventilate normally (PCO₂ = 40) rather than quadruple his effective ventilation (PCO₂ = 10), his pH would have been around 6.85.
his pH was 7.30, it appears that hyper-ventilation had a desirable effect.

The $T_{40}$ bicarbonate reflects metabolic bicarbonate levels. These change relatively slowly since they represent renal retention or excretion of bicarbonate. If $PCO_2$ and $T_{40}$ bicarbonate are both elevated, this represents chronic respiratory failure, since $PCO_2$ is elevated (respiratory) and bicarbonate is retained by the kidney (an attempt to adjust the pH toward normal).

A sudden drop in $T_{40}$ bicarbonate reflects:
1. A laboratory error.
2. An increase in [H+] in the body.

A sudden increase in $T_{40}$ bicarbonate reflects:
1. Most likely a laboratory error.
2. The patient has received exogenous bicarbonate.

Review. $T_{40}$ bicarbonate can be used to:
1. Evaluate the metabolic component of an acid-base problem.
2. Estimate the duration of ventilatory failure.
3. Check on laboratory accuracy. The $T_{40}$ bicarbonate should not vary more than 2-4 mM/L in 12 hours.
4. Serve as a sensitive indicator to the addition of [H+], since a drop in $T_{40}$ bicarbonate may precede a significant acidemia.
5. Allow you to derive these results with a rapid and simple method and paper and pencil calculations. It allows you to decide the proper therapeutic approach prior to treatment, rather than a “hit and miss” type of approach.

Approach to repair of a bicarbonate deficit. One can find many methods of calculating the bicarbonate repair needed for a patient based upon whole blood buffering or clinical empiricism. One such method is:

\[
\text{Repair} = (24 - \text{Standard Bicarbonate}) \times .5 \times \text{body weight (kilo)}
\]

which makes the following assumptions:
1. The body buffering capacity is 31 slykes.
2. Infused bicarbonate distributes to all body fluids.
3. The body is a static “test-tube”, that is, there are no dynamic changes in acids or bases being made by the body.

Assuming the unreasonable case of a normal distribution and volume of fluids in a patient. the fluid space infused bicarbonate will distribute itself in about 20 liters (blood + extravascular-extracellular volume), thus 44 mMoles of a bicarbonate solution will result in an increase of about 2.2 mMoles per liter in a patient, if no other acids are added and the kidney does not react. These are also unreasonable assumptions, and even if repair is calculated, one must titrate the patient to desired levels by repeatedly looking at the $T_{40}$ bicarbonate value for the end point. This end point may not be 24 mMoles/liter. For example, if the $T_{40}$ was 35 at one time, and later was found to be 20, one would suspect lactic acidosis or ketoacidosis (“anion gap”) and would begin base repair by titrating the patient to a $T_{40}$ of about 35, which is where the patient presumably was stable prior to the superimposed metabolic crisis.

Calculations of base repair must be approached cautiously, lest one be led to believe that infusing this base will provide the desired effects. Such a calculation is valuable when viewed against the results obtained when infusion of base is monitored by repeated calculations of the $T_{40}$ bicarbonate.

Now that we have learned the use of the Henderson equation and the pH/H+ conversion tables in calculating the $T_{40}$ bicarbonate in understanding clinical acid-base problems, it is appropriate to state that one may apply a nomogram devised by Drs. Bruce Armstrong and John Mohler. (Figure 1.) To simply solve these problems, the nomogram is used in the following manner.

A line is drawn between the “given $PCO_2$” (a) and the “given pH (b), and extended through the $T_{40}$ isobar (c) and...
Figure 1. T₄₀ bicarbonate nomogram for solving clinical acid-base problems

IN VIVO BALANCE

AM
Wn
Wr
25
5
6
1
8
9
10
15
-20
-25
-30
40
50
60
70
80
90
100
120
140
160
180
200

HCO₃⁻ PLASMA

PCO₂

5
6
7
8
9
10
15
20
25
30
40
50
60
70
80
90
100
120
140
160
180
200

Courtesy of Dr. John Mohier, LAC-USC Medical Center.

plasma HCO₃⁻ line (d). One may then read off the values for H⁺, actual bicarbonate and base excess or deficit under these conditions. If one then rotates a straight edge, using the intersect on the T₄₀ isobar (c) as a fulcrum so the right hand side of the rule now intersects line “a” at a PCO₂ value of 40, one can now draw a second line. Reading points along the intersects will reveal in sequence: “a”—PCO₂ = 40 (normal ventilation), “b”—pH and H⁺ when the patient has been corrected for normal ventilation, “d”—the T₄₀ bicarbonate
and base excess or deficit which represents the metabolic status of the patient when ventilation is normal.

Part II: Clinical assessment

In the first part of this article, we have become familiar with the concepts of $T_{40}$ bicarbonate, and the utilization of the Henderson equation in its determination. We have also been exposed to use of the $T_{40}$ nomogram, and its application in the interpretation of a blood gas report. We are now ready to apply these skills to practical bedside applications.

To properly assess their meaning in a clinical status, it is necessary to correlate the blood gas results with the clinical state of the patient. The following factors are of interest:

1. Etiology of the disease.
2. Duration of the disease process.
3. Condition of the patient (sensorium, hydration, and so on).
4. Treatment.

Evaluation of problems as presented in Part I of this article (that is, examples 2 and 3) represent rather straightforward situations, such as acute respiratory failure and diabetic ketoacidosis. More complicated is the evaluation of acid-base disorders found in chronic respiratory acidosis complicated by shock and lactic acidosis in patients who have been on long-term steroid and diuretic therapy, and who develop vomiting and diarrhea as well as gastrointestinal bleeding while on a respirator. It may be appreciated that, aside from the anticipated findings of chronic respiratory acidosis and metabolic acidosis, other problems arising from previous therapy and complications of that therapy will change the results of the blood gases from those that might be expected. Thus, it is of inestimable value to have as much clinical information available to aid in your assessment.

It must also be remembered that a single arterial blood gas determination is just that. It is a laboratory test and is only one of many assessments to be made in evaluating and treating the patient. You must be ready to assess whether this test, in fact, represents a steady state or simply a transient point in the patient's clinical course. Repeated blood gases are of course necessary to evaluate this, as well as continuing evaluation of the clinical status of the patient. These changes also must be correlated to what has happened to the patient's therapeutic response.

In our previous discussion, we have alluded to the appropriate description and interpretation of the values of blood gas analysis obtained from the laboratory. One such approach is outlined below:

**Description:**

- $	ext{pH}$: Normal, Acidemia, Alkalemia
- $\text{PCO}_2$: Ventilatory Status (Quantitate)
  - Respiratory Acidosis
  - Respiratory Alkalosis
- $\text{HCO}_3^-$: Metabolic Status
  - Metabolic Acidosis
  - Metabolic Alkalosis

Acute or Chronic?

Are these the results you expect? If not, why? (Laboratory error, iatrogenic influences, and so on.)

I will now present a series of representative examples to clarify the previously presented statements.

1. A patient with chronic lung disease suddenly becomes lethargic and cyanotic.

   $\text{pH} = 7.10$
   $\text{PCO}_2 = 90 \text{ TORR}$

   \[
   [\text{HCO}_3^-] = \frac{\text{K}_1 \times \text{PCO}_2}{\text{pH}/\text{H}^+} = 25 \times 90 = 28 \text{ mM/L}
   \]

   It would appear that the patient has an acidemia, respiratory acidosis, a decrease in alveolar ventilation of $2\frac{1}{4}$ times (90/40), and a metabolic alkalosis (therefore, representing a chronic respiratory failure). To properly evaluate this, we calculate the $T_{40}$ bicarbonate.

   \[
   \begin{array}{ccc}
   \text{PCO}_2 & \text{pH}/\text{H}^+ & [\text{HCO}_3^-] \\
   \hline
   \text{Given:} & 90 & 7.10/90 & 28 \\
   \text{If:} & 40 & 7.40/40 & 25 \\
   \end{array}
   \]
Thus, if the patient were ventilating effectively so that his \(PCO_2\) were 40 TORR, there would be a sudden change in \(PCO_2\) of 50 TORR. For every acute change of \(PCO_2\) of 15 TORR, there would be an appropriate change in bicarbonate of 1 mM/L. Consequently, there would be a fall of 3 mM/L of bicarbonate. The \(T_{40}\) bicarbonate is now seen to be 25, so that no metabolic abnormality exists, and this represents an acute respiratory failure. The treatment in turn becomes obvious: ventilation. It also suggests that, although the patient is known to have chronic lung disease, he was not in chronic respiratory failure. Obviously, the patient must also be treated for the precipitating cause of his acute respiratory failure (that is, an overdose, pneumonia, pleural effusion, pneumothorax, etc.).


\[
pH = 7.22 \\
PCO_2 = 58 \text{ TORR} \\
[HCO_3^-] = \frac{K \cdot PCO_2}{[H^+]} = \frac{25 \times 58}{61} = 24 \text{ mM/L} 
\]

It would appear that the patient has an acidemia, respiratory acidosis, a decrease in alveolar ventilation of \(2\frac{1}{2}\) times, and no metabolic disorder (therefore, representing an acute respiratory failure). Obviously, the patient must also be treated for the precipitating cause of his acute respiratory failure (that is, an overdose, pneumonia, pleural effusion, pneumothorax, etc.).

Thus, if the patient were ventilating effectively so that her \(PCO_2\) were 40 TORR, there would be a sudden change of \(PCO_2\) of 18 TORR and a fall of bicarbonate of approximately 1 mM/L. The \(T_{40}\) bicarbonate is 23 (within normal limits) and the pH would be within normal limits. It is apparent that the patient did indeed have an acute respiratory failure, which was appropriately treated by the relief of bronchospasm and with supplemental oxygen without the necessity of ventilator intervention.

3. An elderly man with pulmonary emphysema enters with \(CO_2\) narcoses.

\[
\begin{align*}
\text{pH} &= 7.20 \\
PCO_2 &= 90 \text{ TORR} \\
[HCO_3^-] \quad ? \quad = \frac{K \cdot PCO_2}{[H^+]} = \frac{25 \times 90}{64} = 35 \text{ mM/L} 
\end{align*}
\]

It would appear that the patient has an acidemia, respiratory acidosis, a decrease in alveolar ventilation of \(2\frac{1}{4}\) times, and a metabolic alkalosis (which would be the usual finding in a patient in chronic respiratory failure and is what we expected to find in the first patient). Let us see if we are right this time. Calculating the \(T_{40}\) bicarbonate:

\[
\begin{array}{ccc}
\text{PCO}_2 & \text{pH/}[H^+] & [HCO_3^-] \\
\hline
\text{Given:} & 90 & 7.20/64 & 35 \\
\text{If:} & 40 & 7.52/31 & 32 \\
\end{array}
\]

Thus, if the patient were suddenly ventilated to a \(PCO_2\) of 40 TORR, his metabolic state would be represented by a metabolic alkalosis and would exhibit an alkalemia. Should anyone be tempted to treat him with bicarbonate and ventilation (prior to determining his actual acid-base status), he would be susceptible to significant alkalemia which would be manifest after being ventilated. This might predispose to significant arrhythmias, particularly in the presence of significant hypoxemia which this patient is most certain to have.

4. Emphysema with respiratory failure with a massive GI bleed.

\[
\begin{align*}
\text{pH} &= 7.10 \\
PCO_2 &= 62 \text{ TORR} \\
[HCO_3^-] \quad ? \quad = \frac{K \cdot PCO_2}{[H^+]} = \frac{25 \times 62}{80} = 19 \text{ mM/L} 
\end{align*}
\]

The patient has a significant acidemia, respiratory acidosis, a decrease in alveolar ventilation of approximately \(1\frac{1}{2}\) times, and an apparent metabolic acidosis. This is certainly a different blood gas picture than we have seen in the previous example. With the given clinical history, we would expect this man to have a metabolic alkalosis, but alas he has metabolic acidosis in addi-

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tion to his respiratory acidosis. What are the causes that seem to be complicating his respiratory problem? First, to quantitate his acidemia, it is necessary to calculate his $T_{40}$ bicarbonate:

$$\begin{align*}
\text{PCO}_2 & \quad \text{pH}/[H^+] \\
\text{Given:} & \quad 62 \quad 7.10/80 \quad 19_2^4 \\
\text{If:} & \quad 40 \quad 7.26/56 \quad 18
\end{align*}$$

The patient does indeed have a metabolic acidosis complicating his clinical situation. Ventilation would significantly improve his acidicotic situation; but, in addition to this, other approaches must be utilized. Again, one would initially think in terms of bicarbonate therapy. However, if used, it will still not solve the basic problem which becomes obvious with assessment of the clinical history. The patient has had a major bleeding episode (not unusual in chronic lungers who have an increased frequency of ulcer diathesis), with a fall in blood pressure and poor tissue perfusion. Consequently, the patient has developed lactic acidosis. Treatment is thus modified by clinical data.

5. A 39-year-old female, drug overdose with heroin, aspiration, endotracheal intubation with continuous ventilation.

$$\begin{align*}
\text{pH} & = 7.60 \\
\text{PCO}_2 & = 20 \text{ TORR} \\
[HCO_3^-] & = K\cdot\text{PCO}_2 = 25 \times 20 = 20 \text{ mM/L}
\end{align*}$$

The patient has an alkalemia, respiratory alkalosis, alveolar hyperventilation of perhaps 9 L/min, and a metabolic acidosis. We know that a heroin overdose causes ventilatory depression; and one would therefore expect to find acute alveolar hypoventilation. Additionally, such an overdose can cause an adult respiratory distress syndrome, with severe unremitting hypoxemia and with early hyperventilation.

The patient also has aspirated, which can cause a significant right-to-left shunting physiologically. If severe enough, it can also cause the adult respiratory distress syndrome; as can high prolonged use of oxygen, which may be necessary in the supportive treatment of this patient. Don't forget the patient is being ventilated. What is her state of hydration? What is her tissue perfusion? Now, we are beginning to see how complicated these problems may become. Let us start to unravel it all by defining her $T_{40}$ bicarbonate, which will not only tell us what her metabolic status is, but also will be useful in monitoring the patient as we treat her.

$$\begin{align*}
\text{PCO}_2 & \quad \text{pH}/[H^+] \\
\text{Given:} & \quad 20 \quad 7.60/25 \quad 20 \quad 3 \quad 2 \\
\text{If:} & \quad 40 \quad 7.32/48 \quad 21
\end{align*}$$

She has only a mild base deficit of 3mM/L and, therefore, a very minimal metabolic acidosis. The respiratory alkalosis, then, certainly is not in response to this. It remains to be seen if it is secondary to hypoxemia, brain damage (cerebral edema), or whether it is iatrogenic. This patient's hypoxemia was easily controlled, thus eliminating that factor. By decreasing her minute ventilation, her ventilatory status was corrected. Remember, in making changes in alveolar ventilation, do not overcorrect, or correct too quickly. It may lead to arrhythmias or seizures. Also, do not ignore the obvious: overventilation is the most common complication of ventilator therapy. If we had not looked at this patient's entire acid-base evaluation, I dare say that one might have overestimated the importance of her metabolic acidosis and not have been aware of her major problem: hyperventilation.

6. A 45-year-old female diabetic patient with visual problems, urinary frequency, and shortness of breath.

$$\begin{align*}
\text{pH} & = 7.30 \\
\text{PCO}_2 & = 24 \text{ TORR} \\
[HCO_3^-] & = ? = K\cdot\text{PCO}_2 = 25 \times 24 = 12 \text{ mM/L}
\end{align*}$$

This represents a common clinical problem. The diabetic patient who enters the emergency room in a coma often presents the clinical dilemma of diabetic ketoacidosis versus insulin coma. The obvious is sometimes overlooked. The status of the patient's respiration is often
a clue to the answer. Here, the patient tells you she has significant diabetes as manifested by polyuria (perhaps with a superimposed infection). She has visual problems (which may be simply related to uncontrolled diabetes) and shortness of breath (caused by hyperventilation). Let us now assess her metabolic status.

\[
\begin{array}{ccc}
\text{PCO}_2 & \text{pH}/[\text{H}^+] & [\text{HCO}_3^-] \\
\text{Given:} & 24 & 7.30/50 & 12 \\
\text{If:} & 40 & 7.12/77 & 13 \\
\end{array}
\]

It is quite clear that this case represents a moderately severe primary metabolic acidosis secondary to diabetic ketoacidosis with secondary hyperventilation (an attempt to maintain pH homeostasis). It is not as dramatic as the third example in Part I of this article, but nevertheless, it is representative of a common clinical situation. Yet, if the clinical history of diabetes were unknown, the appropriate cause of the “shortness of breath” might not have been quite so obvious without the help of blood gases and the appropriate evaluation of the acid-base status of the patient.

**Summary**

In summary, I have attempted to review the mechanical application of \( T_{10} \) bicarbonate to clinical acid-base disorders and to show its utilization at the bedside for diagnostic purposes. By utilizing this simple concept, one is able to previsualize the results of ventilatory therapy without the potential hazards of actually using this modality. In conjunction with an appropriate clinical history and evaluation of the patient, one is able to assess the physiological status of the patient and, hopefully, approach therapy in a logical and decisive manner.

**REFERENCES**


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