Therapeutic Strategies for Interpreting Acid-Base Status with and Without Blood Gases

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and

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All planners, presenters, and reviewers of this session report no financial relationships relevant to this activity.
Learning Objectives

• Given a case scenario, determine acid-base status using blood gases and explain confounding factors to interpretation of the blood gases.

• Evaluate whether acetate should be added to parenteral nutrition in a given case scenario and determine the appropriate dose, if added, based on general guidelines.

• Discuss medications that are likely contributing to acid-base imbalance in a given case scenario.
Therapeutic Strategies for Interpreting Acid-Base Status Blood Gases

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Assistant Professor
West Coast University School of Pharmacy
Los Angeles, California
How often do you interpret ABGs in your current practice; how confident are you in interpreting ABGs?

A. None; I would like to learn more
B. Occasionally; I would like some more practice
C. Frequently; I’m pretty confident
D. All the time; I’m an expert
Outline

• ABGs in general
• One to practice, traditional
• Confounders type 1
• Same abg or different? As stewart
• Respiratory ABG?
• Confounders type 2?
Acid/Base Disorders

- Up to 90% of critically ill patients develop an acid/base disorder
  - May be self-limiting after management of the underlying condition
  - More commonly, they accompany complicated clinical conditions that obscure acid/base identification

- Correct identification is necessary to select the appropriate therapeutic intervention
Acid/Base Approaches

**Co₂/HCO₃ (Boston)**
- Entirely based on Henderson-Hasselbach equation
- Easy to use in stable patients

**BE/deficit (Danish)**
- BE is less influenced by changes in PaCO₂
- Allows better quantification of metabolic component

**Anion gap**
- Primary method for detecting unmeasured anions as the cause of metabolic acidosis
- Most critically ill patients have low albumin and phosphate, making AG less accurate

**Stewart (Physio-chemical)**
- Rejects Henderson-Hasselbach
- Independent variables are CO₂, SID, and total nonvolatile weak acids
Case: SZ

- SZ is a 46 y/o female admitted to MICU with sepsis
- Today is hospital day #3
- Acute renal failure, not yet on CRRT, not mechanically ventilated
- PMH: type 2 DM, hyperlipidemia, hypertension
Case: SZ

• SZ is currently receiving vancomycin, meropenem, NS, norepinephrine, and trophic enteral feeds.

• SZ’s hemodynamic status has improved and the team anticipates discontinuation of pressors in the next 24 hours.
Case: SZ

<table>
<thead>
<tr>
<th></th>
<th>Today</th>
<th></th>
<th>Today @ 0500</th>
</tr>
</thead>
<tbody>
<tr>
<td>Na</td>
<td>138</td>
<td>pH</td>
<td>7.142</td>
</tr>
<tr>
<td>K</td>
<td>4.5</td>
<td>pCO2</td>
<td>14.2</td>
</tr>
<tr>
<td>Cl</td>
<td>108</td>
<td>pO2</td>
<td>117</td>
</tr>
<tr>
<td>CO2</td>
<td>9</td>
<td>HCO3</td>
<td>4.9</td>
</tr>
<tr>
<td>BUN</td>
<td>64</td>
<td>BE</td>
<td>-24</td>
</tr>
<tr>
<td>SCr</td>
<td>6.2</td>
<td>O2SAT</td>
<td>97</td>
</tr>
<tr>
<td>Glucose</td>
<td>209</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lactate</td>
<td>5.2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Albumin</td>
<td>2.1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>WBC</td>
<td>16.7</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
How would you interpret SZ’s ABG?

A. Metabolic acidosis
B. Respiratory acidosis
C. Metabolic acidosis with respiratory compensation
D. Respiratory acidosis with metabolic compensation
Arterial Blood Gases (ABGs)

- Laboratory assessment of acid-base status most frequently measured by blood gases

<table>
<thead>
<tr>
<th></th>
<th>pH</th>
<th>pCO₂</th>
<th>P0₂</th>
<th>HCO₃</th>
<th>Base Excess</th>
<th>S0₂</th>
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</thead>
<tbody>
<tr>
<td>Arterial</td>
<td>7.4</td>
<td>35-45</td>
<td>80-100</td>
<td>22-26</td>
<td>-2 to +2</td>
<td>95-100</td>
</tr>
<tr>
<td></td>
<td>(7.35-7.45)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Venous</td>
<td>7.36</td>
<td>41-51</td>
<td>35-40</td>
<td>24-28</td>
<td>0 to +4</td>
<td>70-75</td>
</tr>
</tbody>
</table>
Acid/base – Traditional Approach

- Acid-base status is regulated by the
  - Lungs
  - Kidneys
  - Exogenous buffer system

**Carbonic acid/bicarbonate buffer system**

\[
\text{H}_2\text{O} + \text{CO}_2 \rightleftharpoons \text{H}_2\text{CO}_3 \rightleftharpoons \text{H}^+ + \text{HCO}_3^-
\]
<table>
<thead>
<tr>
<th>pH &gt; 7.45 (&gt; 7.4)</th>
<th>pH &lt; 7.35 (&lt; 7.4)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Respiratory alkalosis</strong> = ↓ PCO2</td>
<td><strong>Respiratory acidosis</strong> = ↑ PCO2</td>
</tr>
<tr>
<td>• Too much CO2 being removed from body (overbreathing)</td>
<td>• Lungs unable to remove CO2 from body</td>
</tr>
<tr>
<td><strong>Metabolic alkalosis</strong> = ↑ HCO3</td>
<td><strong>Metabolic acidosis</strong> = ↓ HCO3</td>
</tr>
<tr>
<td>• Too much base HCO3</td>
<td>• Consumption or loss of base (HCO3)</td>
</tr>
</tbody>
</table>
### pH

Assess presence of acidemia or alkalemia – Tightly regulated at 7.4

### pCO₂

- Acid
- Respiratory

### HCO₃⁻

- Base
- Metabolic

### Table: Arterial pH Values

<table>
<thead>
<tr>
<th></th>
<th>pH</th>
<th>pCO₂</th>
<th>PO₂</th>
<th>HCO₃⁻</th>
<th>Base Excess</th>
<th>SO₂</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arterial</td>
<td>7.4 (7.35-7.45)</td>
<td>35-45</td>
<td>80-100</td>
<td>22-26</td>
<td>-2 to +2</td>
<td>95-100</td>
</tr>
</tbody>
</table>
Interpreting ABGs – Traditional Approach

• 3 step approach:
  1. Determine acidosis or alkalosis
  2. Determine primary abnormality
  3. Identify other abnormalities → compensation or mixed?
Interpret SZ’s ABG

<table>
<thead>
<tr>
<th></th>
<th>Today @ 0500</th>
<th>Normal</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>7.142</td>
<td>7.4</td>
</tr>
<tr>
<td>pCO₂</td>
<td>14.2</td>
<td>35-45</td>
</tr>
<tr>
<td>pO₂</td>
<td>117</td>
<td></td>
</tr>
<tr>
<td>HCO₃</td>
<td>4.9</td>
<td>22-26</td>
</tr>
<tr>
<td>BE</td>
<td>-24</td>
<td></td>
</tr>
<tr>
<td>O₂SAT</td>
<td>97</td>
<td></td>
</tr>
</tbody>
</table>
How would you interpret SZ’s ABG?

A. Metabolic acidosis
B. Respiratory acidosis
C. Metabolic acidosis with respiratory compensation
D. Respiratory acidosis with metabolic compensation
SZ’s ABG

• 3 step approach:

1. Determine acidosis or alkalosis
   – pH 7.142 < 7.4, so **acidosis**

2. Determine primary abnormality
   – Is pCO₂ abnormal?
   – Would a low pCO₂ cause acidosis?

   – Is HCO₃ abnormal?
   – Would a low HCO₃ cause acidosis?

3. Identify other abnormalities → compensation or mixed?
SZ’s ABG

• SZ has metabolic acidosis
  – The low CO2 would cause alkalosis, so the lungs are compensating for the primary disorder

• Compensated metabolic acidosis

• Remember the lungs (pCO₂) can compensate in minutes, but the kidneys (HCO₃⁻) take days
<table>
<thead>
<tr>
<th>Disorder</th>
<th>Compensatory response and equation</th>
</tr>
</thead>
</table>
| Metabolic acidosis     | *Expected* response is 1.2 ↓ for each 1 ↓ in HCO3  
pCO2 = 40 – [1.2 x (24 – measured HCO3)] |
| Metabolic alkalosis    | *Expected* response is 0.7 ↑ for each 1 ↑ in HCO3  
pCO2 = 40 + [0.7 x (measured HCO3 – 24)] |
| Respiratory acidosis   | *Expected* response is 1-3 ↑ for each 10 ↑ in pCO2  
< 48 hours: pH ↓ = 0.08 x [(measured pCO2 – 40)/10]  
> 48 hours: pH ↓ = 0.03 x [(measured pCO2 – 40)/10] |
| Respiratory alkalosis  | *Expected* response is 2-4↓ for each 10 ↓ in pCO2  
< 48 hours: pH ↑ = 0.08 x [(40 - measured pCO2 )/10]  
> 48 hours: pH ↑ = 0.03 x [(40 - measured pCO2)/10] |
Confounding Factors

• Not enough information from traditional approach
  – Evaluate whether the ABG matches what you would expect from the clinical picture
  – Mixed or underlying disorder
Stewart or Physiochemical Approach

- Controversial
- Rejects Henderson-Hasselbach
- $\text{HCO}_3^-$ and $\text{H}^+$ are dependent variables
  - Independent variables are $\text{CO}_2$, SID, and total nonvolatile weak acids
- Original equations too complicated to perform at bedside
- There are methods to incorporate this approach into bedside evaluation

Kaplan LJ, Frangos S. Critical Care 2005;9(2):198
Unifying Base Excess and Stewart Approaches

• One method involves calculating the albumin and lactate corrected anion gap
  – Then if elevated, the delta ratio; if normal, calculate the urinary anion gap

• Another method calculates the effect of the strong ion difference (SID) on the base excess
  – And the effect of weak acids on the base excess

Interpret ABG - DV

• DV, 56 y/o male, admitted to CVICU after cardiac arrest and ROSC, day #1
• Mechanically ventilated:
  - pH: 7.122
  - pCO$_2$: 75
  - pO$_2$: 52
  - HCO$_3$: 24.5
  - BE: -5
  - SO$_2$: 73
How would you interpret DV’s ABG?

A. Metabolic acidosis
B. Respiratory acidosis
C. Metabolic alkalosis
D. Respiratory alkalosis
ABG #2 - DV

- DV, 56 y/o male, admitted to CVICU after cardiac arrest and ROSC
- Mechanically ventilated:
  - pH: 7.122
  - pCO₂: 75
  - pO₂: 52
  - HCO₃: 24.5
  - BE: -5
  - SO₂: 73

<table>
<thead>
<tr>
<th>Normal</th>
<th>7.4</th>
<th>35-45</th>
<th>22-26</th>
</tr>
</thead>
</table>
Confounding Factors

• Time when ABG measured
  – Respiratory vs metabolic time to compensation
  – Compared to past history or vent changes

• Nutrition/Medications
Respiratory Acid/Base Disorders and Time

• Respiratory changes to regulate acid/base status are rapid, in minutes to hours
  – Metabolic compensation takes 2-5 days to reach a new steady state
  – A respiratory disorder with a duration of less than 2-3 days is considered acute

• Review past ABGs, checking dates and times closely
  – When ventilator settings are being changed, there may be multiple ABGs per day
Respiratory Acid-Base Disorders

• Confounding factors
  – Respiratory acidosis may be caused by overfeeding
    • Consider obtaining RQ via indirect calorimetry or ventilator
    • May be caused by overfeeding, RQ > 1
      – RQ = $\frac{VCO_2}{VO_2}$
  – Salicylate toxicity may cause respiratory alkalosis (in addition to metabolic acidosis)
  – Caution needed if considering sodium bicarbonate to treat respiratory acidosis
Should buffers be used for respiratory acidosis?

**Table 4**

DIFFERENCES IN pHa, PaCO₂, AND BASE EXCESS BEFORE AND AFTER ADMINISTRATION OF SODIUM BICARBONATE (NaHCO₃) AMONG PATIENTS WITH ALI

<table>
<thead>
<tr>
<th>Case</th>
<th>pHa Pre-NaHCO₃</th>
<th>pHa Post-NaHCO₃</th>
<th>PaCO₂ Pre-NaHCO₃ (mm Hg)</th>
<th>PaCO₂ Post-NaHCO₃ (mm Hg)</th>
<th>Base Deficit Pre-NaHCO₃ (mEq/L)</th>
<th>Base Deficit Post-NaHCO₃ (mEq/L)</th>
<th>NaHCO₃ Dose (mEq)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>7.17</td>
<td>7.10</td>
<td>58</td>
<td>64</td>
<td>−7.8</td>
<td>−11.0</td>
<td>50</td>
</tr>
<tr>
<td>2</td>
<td>7.23</td>
<td>7.18</td>
<td>93</td>
<td>108</td>
<td>11.6</td>
<td>12.5</td>
<td>50</td>
</tr>
<tr>
<td>3</td>
<td>7.14</td>
<td>7.08</td>
<td>33</td>
<td>48</td>
<td>−16.8</td>
<td>−15.6</td>
<td>200</td>
</tr>
<tr>
<td>5</td>
<td>7.27</td>
<td>7.05</td>
<td>38</td>
<td>40</td>
<td>−9.2</td>
<td>−18.7</td>
<td>115</td>
</tr>
<tr>
<td>9</td>
<td>7.27</td>
<td>7.09</td>
<td>46</td>
<td>52</td>
<td>−6.5</td>
<td>−14.4</td>
<td>50</td>
</tr>
<tr>
<td>10</td>
<td>7.17</td>
<td>7.11</td>
<td>50</td>
<td>59</td>
<td>−9.9</td>
<td>−10.9</td>
<td>30</td>
</tr>
</tbody>
</table>

Mean ± SD: 7.21 ± 0.06 7.10 ± 0.04* 53 ± 19 62 ± 24* −6.4 ± 9.5 −9.7 ± 11.3 82.5 ± 64.5

* p < 0.05 Wilcoxon signed rank test for comparison of pre- and post-NaHCO₃ values.

**Carbonic acid/bicarbonate buffer system**

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

Sodium Bicarbonate to Treat Respiratory Acidosis

• Lack of evidence to suggest that sodium bicarbonate improves patient outcome in respiratory acidosis

• The ARDS Network trial allowed sodium bicarbonate infusions to maintain a pH greater than 7.30, but whether this had any effect on patient outcome is unclear

Key Takeaways

• Key Takeaway #1: ABGs should always be interpreted in light of the clinical condition of the patient

• Key Takeaway #2: Simple acid/base disorders can be interpreted using a three step traditional approach

• Key Takeaway #3: Complex acid/base disorders require further investigation
Therapeutic Strategies for Interpreting Acid-Base Status Without Blood Gases

Carol J. Rollins, Pharm.D., M.S., RD, BCNSP, CNSC
Clinical Professor
University of Arizona College of Pharmacy
Tucson, Arizona
Are there times when ABG’s would be helpful to you but they cannot be obtained?

A. YES

B. NO
Outline

1. Algorithm for assessment
2. Cases
   a) Factors contributing to metabolic alkalosis
   b) Factors contributing to metabolic acidosis
   c) Physiology/anatomy considerations
Acid-Base Balance is Complex

• Blood gases give objective data
• Must make assumptions without blood gases
  – Less accurate than ABGs
  – Simplified view of the many factors contributing to acid-base status
  – Confounding factors may be missed
Algorithm for Acid-Base Assessment Without ABG’s

Patient has respiratory problems → ABG’s needed

No respiratory problems → Likely metabolic disorder

Evaluate *laboratory data* that may substantiate clinical data

Evaluate *clinical factors* that affect acid-base status
Case 1

• 66 yo M admitted to the hospital with c/o abdominal pain, nausea, vomiting starting 12 hr PTA
• Diagnosed with SBO in ER
• Conservative therapy: fluids and NG suction
  – D5%/0.45% NaCl + 20 mEq KCl/L at 200 mL/h (60 mL/kg/d)
  – NG output of 4 L immediately in ER, then 2.5 – 3 L daily
• Day 6: Persistent SBO on imaging
Case 1: Labs

<table>
<thead>
<tr>
<th></th>
<th>Admit</th>
<th>Day 3</th>
<th>Day 6</th>
</tr>
</thead>
<tbody>
<tr>
<td>Na⁺</td>
<td>144</td>
<td>140</td>
<td>145</td>
</tr>
<tr>
<td>K⁺</td>
<td>4.2</td>
<td>3.9</td>
<td>3.3</td>
</tr>
<tr>
<td>Chloride</td>
<td>107</td>
<td>105</td>
<td>91</td>
</tr>
<tr>
<td>Bicarb (CO₂)</td>
<td>23</td>
<td>26</td>
<td>33</td>
</tr>
<tr>
<td>Glucose</td>
<td>98</td>
<td>114</td>
<td>99</td>
</tr>
<tr>
<td>BUN</td>
<td>18</td>
<td>10</td>
<td>22</td>
</tr>
<tr>
<td>Creatinine</td>
<td>1.1</td>
<td>0.9</td>
<td>0.7</td>
</tr>
</tbody>
</table>
What is your assessment on day 6 based on the history and labs shown?

A. Labs are “contaminated” by IV fluids
B. Fluids at 60 mL/kg/d caused hypervolemia
C. Metabolic acidosis from Cl⁻ loss
D. Metabolic alkalosis from H⁺ loss
**Case 1: Interpreting Labs**

<table>
<thead>
<tr>
<th></th>
<th>Day 6</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Na(^+)</td>
<td>145</td>
<td>High relative to Cl(^-)</td>
</tr>
<tr>
<td>K(^+)</td>
<td>3.3</td>
<td>Low</td>
</tr>
<tr>
<td>Chloride</td>
<td>91</td>
<td>Low</td>
</tr>
<tr>
<td>Bicarb (CO(_2))</td>
<td>33</td>
<td>High</td>
</tr>
<tr>
<td>BUN</td>
<td>22</td>
<td></td>
</tr>
<tr>
<td>Creatinine</td>
<td>0.7</td>
<td></td>
</tr>
</tbody>
</table>

Lab interpretation **MUST** be supported by **CLINICAL EVIDENCE**
GI Physiology

Liver
Bile ducts
Gall bladder
Duodenum

Stomach
Produces Hydrochloric Acid

Pancreas
Produces Bicarbonate

Jejunum
GI Physiology

Bicarbonate enters duodenum at common bile duct

- Duodenum
- Jejunum
- Ileum
- Colon

- Liver
- Stomach
- Pancreas

- Bile ducts
- Gall bladder

- EC Fistula, End Jejunostomy
- EC Fistula, Ileostomy

- Bicarb Losses
- Diarrhea

http://www.cancerresearchuk.org
Renal Physiology

Excretion
- Acid (H⁺) [~ 1 mmol/kg/d]

Reabsorption
- \(\text{NaHCO}_3\) [~ 4000-5000 mmol/d]
- \(\text{NaCl}\) [~ 650 g/d]

Acid (H⁺) Excretion
- Reabsorption of 85–90% of filtered bicarbonate

Reclaim 85–90% of Filtered Bicarbonate

Distal Convoluted Tubule
- \(\text{NaCl}\)

Medulla
- Cortex

Cortex Medulla
- Thiazides
- Aldosterone, K⁺-sparing diuretics

H₂O ADH

Loop of Henle

Ascending Loop of Henle

Proximal Tubule

Glomerulus

Collecting Tubules

Urine

\(\text{Na}_2\text{HPO}_4\)

\(\text{NaHCO}_3\)

\(\text{NaCl}\)
## Medications Frequently Associated with Acid-Base Disturbance

<table>
<thead>
<tr>
<th>Metabolic Acidosis</th>
<th>Mechanism</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spironolactone, Amiloride, Triamterene</td>
<td>Inhibit Na⁺ reabsorption → hyperchloremia, normal AG</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Metabolic Alkalosis</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Loop diuretics</td>
<td>Cl⁻ depletion, Na⁺ to DCT</td>
</tr>
<tr>
<td>Penicillin</td>
<td>Cl⁻ depletion, Nonreabsorbable anion</td>
</tr>
<tr>
<td>Aminoglycosides</td>
<td>Cl⁻ depletion, stimulates calcium-sensing receptor</td>
</tr>
</tbody>
</table>

Renal Physiology

Na^+ load stimulates pump

Aldosterone – Sensitive Na^+ Pump

SODIUM delivery

Distal Convoluted Tubules

SODIUM reabsorbed

SODIUM exchanged for H^+ and K^+

Collecting Duct

Urine Excretion

Cortex

Medulla

H_2O

+ ADH
# Metabolic Alkalosis

**Lose Acid**
- Gastric acid loss
- Renal $\text{H}^+$ loss
- Extracellular $\text{H}^+$ loss (intracellular shift)
- Decreased acid production (PPI, $\text{H}_2\text{RA}$)

**Gain Bicarb**
- Exogenous bicarb
- Bicarb precursor: Lactate, acetate, citrate
- Medications: Loop or thiazide diuretic, glucocorticoids, fludrocortisone, antacids
## Metabolic Acidosis

<table>
<thead>
<tr>
<th>Loss of Acid</th>
<th>Gain of Bicarb</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Reduce gastric acid loss</strong></td>
<td><strong>Reduce renal H⁺ loss (renal dysfunction)</strong></td>
</tr>
<tr>
<td><strong>Increase production – infection, DKA</strong></td>
<td><strong>GI tract losses – enterocutaneous fistula, jejunostomy, ileostomy, diarrhea</strong></td>
</tr>
<tr>
<td><strong>Stop/reduce administration of exogenous bicarb or bicarb precursor (lactate, acetate, citrate)</strong></td>
<td></td>
</tr>
</tbody>
</table>
Case 1: Factors Affecting Acid-Base Status

Hydrochloric Acid

Vomiting or NG suction

May have an affect on aldosterone

Na+ sensitive Na+ pump stimulated

↑ Na+ to DCT

↑ Na+ reabsorption

Exchange Na+ for K+ and H+  
Acid (H+ ) Loss
Case 1: Factors Affecting Acid-Base Status

• Hypokalemia – when intracellular $K^+$ is low, more $\text{HCO}_3^-$ is reabsorbed in the kidney
• Chloride deficiency – $\text{HCO}_3^-$ is reabsorbed with $\text{Na}^+$ rather than chloride to maintain electroneutrality
• Volume depletion – $\text{Na}^+$ is retained, which increases $\text{HCO}_3^-$ reabsorption (contraction alkalosis)
What is your assessment on day 6 based on the history and labs shown?

A. Labs are “contaminated” by IV fluids
B. Fluids at 60 mL/kg/d caused hypervolemia
C. Metabolic acidosis from Cl⁻ loss
D. Metabolic alkalosis from H⁺ loss
Metabolic Alkalosis Management

• What fluid issues should be considered?
  – Avoid bicarb, bicarb precursors
  – Lactate in LR, Acetate in PN, Citrate in CRRT
  – Adequate volume to prevent “contraction alkalosis”

• Any other therapies that impact alkalosis?
  – Adequate potassium to prevent hypokalemia
  – Reduce acid production in stomach (???)
    • PPI, H$_2$RA may reduce gastric acid removal with NG suction
Case 2

- 72 y.o. patient s/p colostomy after admission 12 days ago
- In ICU x 4 days; tube feeding advanced to goal on day 3
- Back to OR for end ileostomy hospital day 7
- Transferred from ICU to floor HD 9
- Ileostomy output HD 9 to 12: $1 \rightarrow 1.7 \rightarrow 3.6 \rightarrow 4.7$ liters/day
- Temperature to $38.4^\circ$ C last night, on broad spectrum abx
## Case 2: Labs

<table>
<thead>
<tr>
<th></th>
<th>Day 7</th>
<th>Day 10</th>
<th>Day 12</th>
</tr>
</thead>
<tbody>
<tr>
<td>Na+</td>
<td>139</td>
<td>135</td>
<td>138</td>
</tr>
<tr>
<td>K+</td>
<td>3.9</td>
<td>3.7</td>
<td>5.5</td>
</tr>
<tr>
<td>Chloride</td>
<td>106</td>
<td>108</td>
<td>114</td>
</tr>
<tr>
<td>Bicarb (CO₂)</td>
<td>25</td>
<td>20</td>
<td>14</td>
</tr>
<tr>
<td>Glucose</td>
<td>100</td>
<td>118</td>
<td>136</td>
</tr>
<tr>
<td>BUN</td>
<td>16</td>
<td>17</td>
<td>32</td>
</tr>
<tr>
<td>Creatinine</td>
<td>1</td>
<td>0.9</td>
<td>1.5</td>
</tr>
</tbody>
</table>
Case 2: Interpreting Labs

Lab interpretation **MUST** be supported by **CLINICAL EVIDENCE**

<table>
<thead>
<tr>
<th></th>
<th>Day 12</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Na⁺</strong></td>
<td>138</td>
<td>Low relative to Cl⁻</td>
</tr>
<tr>
<td><strong>K⁺</strong></td>
<td>5.5</td>
<td>High normal</td>
</tr>
<tr>
<td>Chloride</td>
<td>114</td>
<td>High</td>
</tr>
<tr>
<td>Bicarb (CO₂)</td>
<td>14</td>
<td>Low</td>
</tr>
<tr>
<td>BUN</td>
<td>32</td>
<td></td>
</tr>
<tr>
<td>Creatinine</td>
<td>1.5</td>
<td></td>
</tr>
</tbody>
</table>
What is your assessment on day 12 based on the history and labs shown?

A. Renal failure has resulted in hypervolemicia
B. Increased ileostomy output is due to tube feeding
C. Metabolic acidosis
D. Metabolic alkalosis
Factors Affecting Acid-Base Status

GI tract losses or outputs
- Gastric losses? No
- Post-pancreas losses? Yes – Ileostomy with high output

Gains or retention
- Renal H⁺ gain? Yes - Renal impairment/failure
- Other acids? Possible
  - Lactic acid with sepsis
  - Organic acids (kidney)

Potassium reflects H⁺ intra- and extra-cellular shifts
What is your assessment on day 12 based on the history and labs shown?

A. Renal failure has resulted in hypervolemia
B. Increased ileostomy output is due to tube feeding
C. Metabolic acidosis
D. Metabolic alkalosis
Metabolic Acidosis Management

• Reduce chloride provision
  – Fluids with lower NaCl content
  – Use bicarb precursors: lactate (LR), acetate, citrate

• Removal of organic acids
  – Renal replacement therapy (CRRT, HD)
  – Treatment that improves renal function
Metabolic Acidosis Management

How much bicarb precursor is needed?

• General rule: 1 mEq/kg/day of acetate for effect
  – If not excreting “usual” 1 mmol H⁺/kg/day, must neutralize this

• Calculate bicarbonate deficit (mEq HCO₃⁻)
  – Deficit = HCO₃⁻ Vd x (desired HCO₃⁻ – measured HCO₃⁻)
    * HCO₃⁻ Vd = (0.4 + 2.6/measured HCO₃⁻) - ideal body wt
  – Deficit = 0.6 x wt (kg) x (desired HCO₃⁻ - measured HCO₃⁻)
  – HCO₃⁻ end-point goal is typically 10-15

Case 3

- 50 y.o. readmitted to the hospital for the 6\textsuperscript{th} time in 5 months
- Tmax 39\degree C, tachycardia, mild hypotension; abdominal pain and distention
- Home PN x 1.5 years after complications of vascular surgery; usual jejunostomy output of 3 – 4 Liters/day
- Hx multiple abdominal surgeries leading to EC fistula and end jejunostomy; multiple septic episodes/CRBSI
- Hx poor diet and medication adherence
Case 3

What type of acid-base problem is expected given the patient history?

A. Mixed acid-base d/o
B. Metabolic acidosis
C. Metabolic alkalosis
D. No acid-base d/o

Do labs support this?

<table>
<thead>
<tr>
<th>Lab Test</th>
<th>3 days PTA</th>
<th>HD #1</th>
</tr>
</thead>
<tbody>
<tr>
<td>Na⁺</td>
<td>133</td>
<td>130</td>
</tr>
<tr>
<td>K⁺</td>
<td>3.9</td>
<td>3.3</td>
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<tr>
<td>Chloride</td>
<td>103</td>
<td>112</td>
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<tr>
<td>Bicarb (CO₂)</td>
<td>20</td>
<td>17</td>
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<tr>
<td>Glucose</td>
<td>86</td>
<td>115</td>
</tr>
<tr>
<td>BUN</td>
<td>15</td>
<td>14</td>
</tr>
</tbody>
</table>
Case 3: Hospital Day #1

- Antibiotics continued from start in ED
  - Rapid improvement in HR, BP, temp by transfer to floor
- MIV D5%-0.45%NaCl + 20 mEq KCl/L @ 125 mL/hr
- Hold PN
- Abdominal CT: large abscess, dilated loops small bowel, no free air
Case 3: Hospital Day #2

- Blood cx: no growth
- Restart PN **HPN Content**
  - Continuous infusion
- Stop MIV: D5% - 0.45%NaCl + 20 mEq KCl/L @ 125 mL/hr

<table>
<thead>
<tr>
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<th>Home PN</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>mEq/D</td>
</tr>
<tr>
<td>NaCl</td>
<td>230</td>
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<tr>
<td>NaAcetate</td>
<td>240</td>
</tr>
<tr>
<td>K-Acetate</td>
<td>40</td>
</tr>
<tr>
<td>KCl</td>
<td>68</td>
</tr>
<tr>
<td>Volume</td>
<td>3 L</td>
</tr>
</tbody>
</table>

**Volume: 3 L, 58 mL/kg**
Case 3: Hospital Day #3

- Continued clinical response to antibiotics started in ED
- Abdomen more distended on am rounds
- Emesis x 8 starting mid afternoon → NG refused
- Fistula + jejunostomy output only 200 mL after noon
  - Was 1.5 – 1.8 L/day while npo in hospital
  - Usual output at home 3 – 4 liters/day
- Abdominal imaging indicates SBO
  - Likely transition point near Ligament of Trietz
Case 3: Hospital Day #4

What factors contributed to the rise in $\text{HCO}_3^-$?

• Acid loss: Emesis (x 8)
• Bicarb retention: 200 mL jejunostomy output
• Administration of acetate in PN

<table>
<thead>
<tr>
<th></th>
<th>3 days PTA</th>
<th>HD#1</th>
<th>HD#4</th>
</tr>
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<tbody>
<tr>
<td>$\text{Na}^+$</td>
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<td>141</td>
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<tr>
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<td>107</td>
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<tr>
<td>Bicarb ($\text{CO}_2$)</td>
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<tr>
<td>Glucose</td>
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<td>18</td>
</tr>
<tr>
<td>Creatinine</td>
<td>1</td>
<td>0.9</td>
<td>1</td>
</tr>
</tbody>
</table>
Case 3: Hospital Day #5

- PN electrolyte content adjusted on HD #4 based on labs
- Patient admitted to ICU after emergent surgery
- Intubated → ABGs available to assess acid-base status
Key Takeaways

**Key Takeaway #1:** Acid-base status can be “simplistically” assessed using the clinical situation plus routine lab result when the patient’s respiratory status is not compromised.

**Key Takeaway #2:** Sodium and chloride generally “move together” when fluid is responsible and move in opposite directions when a metabolic acid-base disturbance occurs.

**Key Takeaway #3:** GI losses and renal function are keys to assessing metabolic acid-base status without ABGs.

**Key Takeaway #4:** Things change, sometimes rapidly.
Self-Assessment Questions: What is the Expected Effect on Acid-Base Status?

1. Patient with gastric fistula due to cancer. Output has been averaging 4 liters per day for over a week.
   A. Acidosis   B. Alkalosis   C. Need ABGs

2. The patient has taken only 2 liter 0.9% NaCl daily for the past 4 days due to increased edema.
   A. Worsen current acid-base disturbance   B. Normalize acid-base balance   C. Shift to the opposite acid-base status
Self-Assessment Question:
What is the Expected Effect on Acid-Base Status?

3. Patient with gastric fistula due to cancer. Continuing 2 L 0.9% NaCl daily, increased oral fluids to maintain hydration. Stopped H$_2$RA in PN a week ago.
   
   A. Worsen current acid-base disturbance
   B. Normalize acid-base balance
   C. Shift to the opposite acid-base status
   D. Need ABGs to assess acid-base status
Self-Assessment Questions: What is the Expected Effect on Acid-Base Status? The Preferred Therapeutic Option?

4. Patient with increased (> 3 L/d) jejunostomy output.
   A. Acidosis   B. Alkalosis   C. Need ABGs

5. Which “stock” fluid is most likely to help mitigate acid-base disruption in this patient?
   A. 0.9% NaCl
   B. D5W-0.45%NaCl
   C. Lactated Ringers
   D. All are equal if given in adequate volume
Self-Assessment Question:
What is the Preferred Therapeutic Option?

6. Patient with increased (> 3 L/d) jejunostomy output. PN to start. Wt 60 kg. Using the general guideline, how many mEq ACETATE would be added to PN?

A. 154
B. 77
C. 60
D. 30

Labs Results Today

<p>| | |</p>
<table>
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</tbody>
</table>
Self-Assessment Question:
What is the Expected Effect on Acid-Base Status?

7. A patient with large NG output for several days is most likely to develop what type of acid-base disorder?

A. Metabolic acidosis
B. Metabolic alkalosis
C. No acid-base disorder
D. Mixed acid-base disorder
Therapeutic Strategies for Interpreting Acid-Base Status with and Without Blood Gases

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Amber Verdell, Pharm.D., BCPS, BCNSP, CNSC


References

• http://www.medscape.com/viewarticle/808418