Acid Base Physiology and Arterial Blood Gas Interpretation

Jackie Glenn, MD FACS
Outline

• Motivation
• Blood Gas Sampling
• Brief Overview of Acid-Base Physiology
• Acid-Base Nomograms
• Cases
  
  *Case 1* – Cyanotic Unresponsive Patient
  *Case 2* – Lung Transplant Patient
  *Case 3* – Patient with Severe Abdominal Pain
  *Case 4* – Pregnant Woman with Hyperemesis Graviderum
  *Case 5* – Ascent to Mount Everest
MOTIVATION FOR LEARNING ABOUT ARTERIAL BLOOD GAS INTERPRETATION
MOTIVATION

In a survey conducted at a university teaching hospital, 70% of the participating physicians claimed that they were well versed in the diagnosis of acid-base disorders and that they needed no assistance in the interpretation of arterial blood gases (ABGs).

These same physicians were then given a series of ABG measurements to interpret, and they correctly interpreted only 40% of the test samples.


From: THE ICU BOOK
MOTIVATION

A survey at another teaching hospital revealed that incorrect acid-base interpretations led to errors in patient management in one-third of the ABG samples analyzed.


From: THE ICU BOOK
MOTIVATION

These surveys reveal serious deficiencies in an area that tends to be ignored.

This can cause trouble in the ICU, where 9 of every 10 patients may have an acid-base disorder.


From: THE ICU BOOK
Some Acid-Base Web Sites

http://www.acid-base.com/

http://www.qldanaesthesia.com/AcidBaseBook/

http://www.virtual-anaesthesia-textbook.com/vat/acidbase.html#acidbase

http://ajrccm.atsjournals.org/cgi/content/full/162/6/2246

http://www.osa.suite.dk/OsaTextbook.htm

Google/uptodate/pubmed is your friend!!
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*** EXCEPT DURING INSERVICE/BOARD EXAMS OR CODES! ***
<table>
<thead>
<tr>
<th>Clinical state</th>
<th>Acid-base disorder</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pulmonary embolus</td>
<td>Respiratory alkalosis</td>
</tr>
<tr>
<td>Hypotension</td>
<td>Metabolic acidosis</td>
</tr>
<tr>
<td>Vomiting</td>
<td>Metabolic alkalosis</td>
</tr>
<tr>
<td>Severe diarrhea</td>
<td>Metabolic acidosis</td>
</tr>
<tr>
<td>Cirrhosis</td>
<td>Respiratory alkalosis</td>
</tr>
<tr>
<td>Renal failure</td>
<td>Metabolic acidosis</td>
</tr>
<tr>
<td>Sepsis</td>
<td>Respiratory alkalosis, metabolic acidosis</td>
</tr>
<tr>
<td>Pregnancy</td>
<td>Respiratory alkalosis</td>
</tr>
<tr>
<td>Diuretic use</td>
<td>Metabolic alkalosis</td>
</tr>
<tr>
<td>COPD</td>
<td>Respiratory acidosis</td>
</tr>
</tbody>
</table>
Getting an arterial blood gas sample
Allen’s Test!
Be sure the hand will be perfused prior to line placement or sampling.
Blood Gas Report

**Acid-Base Information**
- pH
- PCO₂
- HCO₃ [calculated vs measured]

**Oxygenation Information**
- PO₂ [oxygen tension]
- SO₂ [oxygen saturation]
Blood Gas Report

Acid-Base Information
• pH
• $\text{PCO}_2$
• $\text{HCO}_3$ [calculated vs measured]

Oxygenation Information
• $\text{PO}_2$ [oxygen tension]
• $\text{SO}_2$ [oxygen saturation]
PaO$_2$ [oxygen tension]
SaO$_2$ [oxygen saturation]
a = arterial
Pulse Oximeter Measures $\text{SaO}_2$
Pulse Oximeter Measures SaO2
**Left shifted**
- Decreased temp
- Decreased 2-3 DPG
- Decreased [H+]
- CO

**Right shifted** (reduced affinity)
- Increased temp
- Increased 2-3 DPG
- Increased [H+]
Hydrogen Ions

H+ is produced as a by-product of metabolism.

[H+] is maintained in a narrow range.

Normal arterial pH is around 7.4.

A pH under 7.0 or over 7.8 is compatible with life for only short periods.
pH and $[\text{H}^+]$

$[\text{H}^+]$ in nEq/L = $10^{(9-\text{pH})}$
A normal [H⁺] of 40 nEq/L corresponds to a pH of 7.40. Because the pH is a negative logarithm of the [H⁺], changes in pH are inversely related to changes in [H⁺] (e.g., a decrease in pH is associated with an increase in [H⁺]).

<table>
<thead>
<tr>
<th>pH</th>
<th>[H⁺]</th>
</tr>
</thead>
<tbody>
<tr>
<td>7.7</td>
<td>20</td>
</tr>
<tr>
<td>7.5</td>
<td>31</td>
</tr>
<tr>
<td>7.4</td>
<td>40</td>
</tr>
<tr>
<td>7.3</td>
<td>50</td>
</tr>
<tr>
<td>7.1</td>
<td>80</td>
</tr>
<tr>
<td>7.0</td>
<td>100</td>
</tr>
<tr>
<td>6.8</td>
<td>160</td>
</tr>
</tbody>
</table>
Hydrogen Ion Regulation

The body maintains a narrow pH range by 3 mechanisms:

1. **Chemical buffers** (extracellular and intracellular) react instantly to compensate for the addition or subtraction of H+ ions.

2. **CO2 elimination** is controlled by the lungs (respiratory system). Decreases (increases) in pH result in decreases (increases) in PCO2 within minutes.

3. **HCO3^- elimination** is controlled by the kidneys. Decreases (increases) in pH result in increases (decreases) in HCO3-. It takes hours to days for the renal system to compensate for changes in pH.
Buffers

- A buffer is a solution which has the ability to minimize changes in pH when an acid or base is added.
- A buffer typically consists of a solution which contains a weak acid HA mixed with the salt of that acid & a strong base e.g. NaA. The principle is that the salt provides a reservoir of A$^-$ to replenish [A$^-$] when A$^-$ is removed by reaction with H$^+$. 
CENTRAL EQUATION OF ACID-BASE PHYSIOLOGY

The hydrogen ion concentration \([H^+]\) in extracellular fluid is determined by the balance between the partial pressure of carbon dioxide (PCO2) and the concentration of bicarbonate \([HCO_3^-]\) in the fluid. This relationship is expressed as follows:

\[
[H^+] \text{ in nEq/L} = 24 \times \frac{\text{PCO2}}{[HCO_3^-]}
\]

where \([H^+]\) is related to pH by \([H^+] \text{ in nEq/L} = 10^{(9-\text{pH})}\)
NORMAL VALUES

Using a normal arterial PCO2 of 40 mm Hg and a normal serum [HCO3⁻] concentration of 24 mEq/L, the normal [H⁺] in arterial blood is

\[ 24 \times \left(\frac{40}{24}\right) = 40 \text{ nEq/L} \]
PCO2/[HCO3^-] Ratio

Since \([H^+] = 24 \times (PCO2 / [HCO3^-])\), the stability of the extracellular pH is determined by the stability of the PCO2/HCO3^- ratio.

Maintaining a constant PCO2/HCO3^- ratio will maintain a constant extracellular pH.
PCO2/[HCO3\textsuperscript{-}] Ratio

When a primary acid-base disturbance alters one component of the PCO2/[HCO3\textsuperscript{-}] ratio, the compensatory response alters the other component in the same direction to keep the PCO2/[HCO3\textsuperscript{-}] ratio constant.
COMPENSATORY CHANGES

When the primary disorder is metabolic (i.e., a change in $\left[\text{HCO}_3^-\right]$), the compensatory response is respiratory (i.e., a change in $\text{PCO}_2$), and vice-versa.

It is important to emphasize that compensatory responses limit rather than prevent changes in pH (i.e., compensation is not synonymous with correction).
PRIMARY AND SECONDARY ACID-BASE DERANGEMENTS

End-Point: A Constant PCO2/[HCO3⁻] Ratio

<table>
<thead>
<tr>
<th>Acid-Base Disorder</th>
<th>Primary Change</th>
<th>Compensatory Change</th>
</tr>
</thead>
<tbody>
<tr>
<td>Respiratory acidosis</td>
<td>PCO2 up</td>
<td>HCO3 up</td>
</tr>
<tr>
<td>Respiratory alkalosis</td>
<td>PCO2 down</td>
<td>HCO3 down</td>
</tr>
<tr>
<td>Metabolic acidosis</td>
<td>HCO3 down</td>
<td>PCO2 down</td>
</tr>
<tr>
<td>Metabolic alkalosis</td>
<td>HCO3 up</td>
<td>PCO2 up</td>
</tr>
</tbody>
</table>
# Simple Acid Base Disorders

<table>
<thead>
<tr>
<th>Acid Base Disorder</th>
<th>pH</th>
<th>pCO2</th>
<th>[HCO₃⁻]</th>
</tr>
</thead>
<tbody>
<tr>
<td>Metabolic Acidosis</td>
<td>Low</td>
<td>Low</td>
<td>Low</td>
</tr>
<tr>
<td>Respiratory Acidosis</td>
<td>Low</td>
<td>High</td>
<td>High</td>
</tr>
<tr>
<td>Metabolic Alkalosis</td>
<td>High</td>
<td>High</td>
<td>High</td>
</tr>
<tr>
<td>Respiratory Alkalosis</td>
<td>High</td>
<td>Low</td>
<td>Low</td>
</tr>
</tbody>
</table>

- **pH**: 7.35-7.45
- **pCO₂**: 35-45 mm Hg
- **[HCO₃⁻]**: 24 mEq/L
<table>
<thead>
<tr>
<th>Primary Disorder</th>
<th>Expected Changes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Metabolic acidosis</td>
<td>[ \text{PCO2} = 1.5 \times \text{HCO}_3 + (8 \pm 2) ]</td>
</tr>
<tr>
<td>Metabolic alkalosis</td>
<td>[ \text{PCO2} = 0.7 \times \text{HCO}_3 + (21 \pm 2) ]</td>
</tr>
<tr>
<td>Acute respiratory acidosis</td>
<td>[ \delta \text{pH} = 0.008 \times (\text{PCO2} - 40) ]</td>
</tr>
<tr>
<td>Chronic respiratory acidosis</td>
<td>[ \delta \text{pH} = 0.003 \times (\text{PCO2} - 40) ]</td>
</tr>
<tr>
<td>Acute respiratory alkalosis</td>
<td>[ \delta \text{pH} = 0.008 \times (40 - \text{PCO2}) ]</td>
</tr>
<tr>
<td>Chronic respiratory alkalosis</td>
<td>[ \delta \text{pH} = 0.003 \times (40 - \text{PCO2}) ]</td>
</tr>
</tbody>
</table>
Respiratory Compensation

The ventilatory control system provides the compensation for metabolic acid-base disturbances, and the response is prompt.

The changes in ventilation are mediated by H$^+$ sensitive chemoreceptors located in the carotid body (at the carotid bifurcation in the neck) and in the lower brainstem.
Respiratory Compensation

A **metabolic acidosis** excites the chemoreceptors and initiates a prompt increase in ventilation and a decrease in arterial PCO2.

A **metabolic alkalosis** silences the chemoreceptors and produces a prompt decrease in ventilation and increase in arterial PCO2.
**PaCO2 Equation**

PaCO2 = (VCO2/VA)*0.863
PaCO2= partial pressure of CO2 in the arterial blood
VCO2: metabolic production of CO2
VA: alveolar ventilation = VE - VD
VE: minute ventilation = tidal volume * respiratory rate
VD: dead space ventilation (area in the respiratory system which is ventilated but has no perfusion)

The constant 0.863 is necessary to equate dissimilar units for VCO2 (ml/min) and VA (L/min) to PACO2 pressure units (mm Hg).
The Six Step Approach to Solving Acid-Base Disorders
Step 1: Acidemic, alkalemic, or normal?

Step 2: Is the primary disturbance respiratory or metabolic?

Step 3: For a primary respiratory disturbance, is it acute or chronic?

Step 4: For a metabolic disturbance, is the respiratory system compensating OK?

Step 5: For a metabolic acidosis, is there an increased anion gap?

Step 6: For an increased anion gap metabolic acidosis, are there other derangements?
MedCalc: Acid-Base Calculator

### Arterial Blood Gas (ABG) values:
- **pH**: 7.4 (7.36 - 7.44)
- **\( P_{CO_2} \)**: 40 mm Hg (36 - 44 mm Hg)
- **\( HCO_3^- \)**: 23 mEq/L (22 - 26 mEq/L)

### Anion Gap values:
- **Sodium (\( Na^+ \))**: 137 mEq/L
- **Bicarbonate (\( HCO_3^- \))**: 23 mEq/L
- **Chloride (\( Cl^- \))**: 99 mEq/L
- **Albumin**: N/A (g/dL)

### Acid-Base Interpretation:
Normal ABG

- *expected pH = 7.38*
- *expected \( CO_2 = 38 \)*
- *expected \( HCO_3^- = 24 \)*
Case 1

A Man and His Pain Machine
Case 1

- Very healthy, fit, active 56 year old man for total hip replacement
- No regular meds, no allergies, unremarkable PMH
- Pain managed by self-administered morphine apparatus (Patient-Controlled Analgesia) *Abbott LifeCare 4100 PCA Plus II*
- When wife visits, patient is cyanotic and unresponsive. “Code Blue” is called. (At CCF Call 111 for all codes)
Case 1

You arrive on the scene with the crash cart.

What should you do?
Case 1

What should you do first?

A  Assess Airway
B  Assess Breathing
C  Assess Circulation
D  Administer Rescue Drugs
E  Evaluate the Situation in Detail
   (get patient chart, interview bystanders, etc.)
Case 1

• What is cyanosis?

• Why is the patient unresponsive?

• Could this be a medication-related problem?
Case 1

What should you do first?

A  Assess Airway
B  Assess Breathing
C  Assess Circulation
D  Administer Rescue Drugs
E  Evaluate the Situation in Detail
   (get patient chart, interview bystanders, etc.)
Assess Airway

Apply jaw thrust to open up the airway.
Assess Breathing

If patient is not breathing, institute rescue breathing (with 100% oxygen if possible)
Endotracheal Intubation
Assess Circulation

Check the patient’s carotid pulse
Administer Rescue Drugs

Drug
MORPHINE

Rescue Drug (Antidote)
NALOXONE (Narcan)
Drug molecule ‘locking’ onto a receptor site to form a drug–receptor complex.
Competitive inhibition of opiate receptors by opiate antagonist
Case 1

While he is being assessed and resuscitated, an arterial blood gas sample is taken, revealing the following:

- pH 7.00
- PCO2 100
- [HCO3 -] data unavailable
Case 1

What is the hydrogen ion concentration?

What is the bicarbonate ion concentration?

What is the acid-base disorder?
Case 1

What is the hydrogen ion concentration?

\[ [H^+] = 10^{(9-\text{pH})} \]

\[ = 10^{(9-7)} \]

\[ = 10^2 \]

\[ = 100 \text{ nEq/L} \]
Case 1

What is the bicarbonate ion concentration?

Remember that \([H^+] = 24 \times (PCO2 / [HCO3^-])\)

Thus,

\([HCO3^-] = 24 \times (PCO2 / [H^+])\)

\([HCO3^-] = 24 \times (100 / 100)\)

\([HCO3^-] = 24 \text{ mEq/L}\)
Case 1

What is the acid-base disorder?
Case 1

What is the acid-base disorder?
Case 1  What is the acid-base disorder?

Recall that for acute respiratory disturbances (where renal compensation does not have much time to occur) each arterial PCO2 shift of 10 mm Hg is accompanied by a pH shift of about 0.08, while for chronic respiratory disturbances (where renal compensation has time to occur) each PCO2 shift of 10 mm Hg is accompanied by a pH shift of about 0.03.
Case 1  What is the acid-base disorder?

In our case an arterial PCO2 shift of 60 mm Hg (from 40 to 100 mm Hg) is accompanied by a pH shift of 0.40 units (from 7.40 to 7.00), or a 0.067 pH shift for each PCO2 shift of 10 mm. Since 0.067 is reasonably close to the expected value of 0.08 for an acute respiratory disturbance, it is reasonable to say that the patient has an

ACUTE RESPIRATORY ACIDOSIS.
Case 1  What is the acid-base disorder?

ANSWER FROM  www.medcalc.com/acidbase.html

(1) partially compensated primary respiratory acidosis, or

(2) acute superimposed on chronic primary respiratory acidosis, or

(3) mixed acute respiratory acidosis with a small metabolic alkalosis
Case 2

Patient Evaluation for a Possible Double Lung Transplant
Case 2

• Very sick 56 year old man being evaluated for a possible double lung transplant
• Dyspnea on minimal exertion
• On home oxygen therapy (nasal prongs, 2 lpm)
• Numerous pulmonary medications
Oxygen therapy via nasal prongs (cannula)
Case 2

While he is being assessed an arterial blood gas sample is taken, revealing the following:

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>7.30</td>
</tr>
<tr>
<td>PCO2</td>
<td>65 mm Hg</td>
</tr>
</tbody>
</table>
Case 2

What is the hydrogen ion concentration?

What is the bicarbonate ion concentration?

What is the acid-base disorder?
Case 2

What is the hydrogen ion concentration?

\[
[H^+] = 10^{(9-pH)}
\]

\[
= 10^{(9-7.3)}
\]

\[
= 10^{(1.7)}
\]

\[
= 50.1 \text{ nEq/L}
\]
Case 2

What is the bicarbonate ion concentration?

Remember that $[H^+] = 24 \times (PCO2 / [HCO3^-])$

Thus,

$[HCO3^-] = 24 \times (PCO2 / [H^+])$

$[HCO3^-] = 24 \times (65 / 50.1)$

$[HCO3^-] = 31.1 \text{ mEq/L}$
Case 2

What is the acid-base disorder?
Case 2  What is the acid-base disorder?

Recall that for acute respiratory disturbances (where renal compensation does not have much time to occur) each arterial PCO2 shift of 10 mm Hg is accompanied by a pH shift of about 0.08, while for chronic respiratory disturbances (where renal compensation has time to occur) each PCO2 shift of 10 mm Hg is accompanied by a pH shift of about 0.03.
In our case an arterial PCO2 shift of 25 mm Hg (from 40 to 65 mm Hg) is accompanied by a pH shift of 0.10 units (from 7.40 to 7.30), or a 0.04 pH shift for each PCO2 shift of 10 mm. Since 0.04 is reasonably close to the expected value of 0.03 for an chronic respiratory disturbance, it is reasonable to say that the patient has a

CHRONIC RESPIRATORY ACIDOSIS.
Case 2  What is the acid-base disorder?

ANSWER FROM www.medcalc.com/acidbase.html

(1) partially compensated primary respiratory acidosis, or

(2) acute superimposed on chronic primary respiratory acidosis, or

(3) mixed acute respiratory acidosis with a small metabolic alkalosis

SAME ANSWER AS IN CASE 1 !!
Case 3 – Patient with Severe Abdominal Pain
Case 3 – Patient with Severe Abdominal Pain

An obese 70 year old man has diabetes of 25 years duration complicated by coronary artery disease (CABG x 4 vessels 10 years ago), cerebrovascular disease (carotid artery endarterectomy 3 years ago) and peripheral vascular disease (Aorto-bifem 2 years ago). [“VASCULOPATH”]
Case 3 – Patient with Severe Abdominal Pain

He now presents to the emergency department with severe, poorly localised abdominal pain with a relatively sudden onset.

To the surprise of the intern that examines him, the patient has a relatively normal abdominal examination. Just lots and lots of pain. Nor has the patient had vomiting, diarrhea, or other GI symptoms.
Case 3 – Patient with Severe Abdominal Pain

The intern considers the differential diagnosis of severe abdominal pain in the setting of a diabetic vasculopathy without much in the way of abdominal signs. She wonders if this might be another manifestation of vascular disease. Following a Google search she finds the following statement at emedicine.com:

The *sine qua non* of mesenteric ischemia is a relatively normal abdominal examination in the face of severe abdominal pain.
Case 3 – Patient with Ischemic Bowel

Following discussion with her attending, the patient is to be admitted to a regular nursing floor where he is to be worked up for his abdominal pain. However, he must remain in the emergency department until a bed can be found.

When the intern comes by 3 hours later to recheck on the patient he looks much worse. He now has abdominal distention, ileus (no bowel sounds), and signs of shock (BP 75/45).

He is rushed to the Intensive Care Unit (ICU).
Case 3 – Patient with Ischemic Bowel
Ischemic injury to the gastrointestinal tract can threaten bowel viability with potential catastrophic consequences, including intestinal necrosis and gangrene. The presenting symptoms and signs are relatively nonspecific and diagnosis requires a high index of clinical suspicion. Acute mesenteric ischemia (AMI) often results from an embolus or thrombus within the superior mesenteric artery (SMA), although a low-flow state through an area of profound atherosclerosis may also induce severe ischemia. Because most laboratory and radiologic studies are nonspecific in early ischemia an aggressive approach to diagnosis with imaging of the splanchnic vasculature by mesenteric angiography is advocated. Various therapeutic approaches, including the infusion of vasodilators and thrombolytics, may then be used. Proper diagnosis and management of patients with AMI requires vigilance and a readiness to pursue an aggressive course of action.
Case 3 – Patient with Ischemic Bowel
Case 3 – Patient with Ischemic Bowel

CLINICAL COMMENTS (emedicine.com)

The sine qua non of mesenteric ischemia is a relatively normal abdominal examination in the face of severe abdominal pain.

The pain generally is severe and may be relatively refractory to opiate analgesics.

Mortality rates of 70-90% have been reported with traditional methods of diagnosis and therapy; however, a more aggressive approach may reduce the mortality rate to 45%.

A survival rate of 90% may be obtained if angiography is obtained prior to the onset of peritonitis.
Case 3 – Patient with Ischemic Bowel

ABGs obtained in the ICU

pH      7.18
PCO2    20 mmHg
HCO3    7 mEq/L
Case 3 – Patient with Ischemic Bowel
Case 3 – Patient with Ischemic Bowel

ABGs obtained in the ICU

pH  7.18
PCO2  20 mmHg
HCO3  7 mEq/L
Case 3 – Patient with Ischemic Bowel

ABGs obtained in the ICU

pH 7.18

PCO2 20 mmHg

HCO3 7 mEq/L

What is the primary disorder?

What is the physiologic response to this disorder?
Case 3 – Patient with Ischemic Bowel

**Step 1:** Acidemic, alkalemic, or normal?

**Step 2:** Is the primary disturbance respiratory or metabolic?

**Step 3:** For a primary respiratory disturbance, is it acute or chronic?

**Step 4:** For a metabolic disturbance, is the respiratory system compensating OK?

**Step 5:** For a metabolic acidosis, is there an increased anion gap?

**Step 6:** For an increased anion gap metabolic acidosis, are there other derangements?
Case 3 – Patient with Ischemic Bowel

Step 1: Acidemic,alkalemic, or normal?

ACIDEMIC
Case 3 – Patient with Ischemic Bowel

Step 2: Is the primary disturbance respiratory or metabolic?

METABOLIC
Case 3 – Patient with Ischemic Bowel

Step 3: For a primary respiratory disturbance, is it acute or chronic?

NOT APPLICABLE
Case 3 – Patient with Ischemic Bowel

Step 4: For a metabolic disturbance, is the respiratory system compensating OK?

DISCUSSION

The physiological response to metabolic acidosis is hyperventilation, with a resulting compensatory drop in PCO2 according to "Winter's formula":

\[
\text{Expected PCO2 in metabolic acidosis} = 1.5 \times \text{HCO}_3 + 8 \quad \text{(range: +/- 2)}
\]

If the actual measured PCO2 is much greater than the expected PCO2 from Winter's formula, then the respiratory system is not fully compensating for the metabolic acidosis, and a respiratory acidosis is concurrently present. This may occur, for instance, when respiratory depressants like morphine or fentanyl are administered to the patient to reduce pain.
Case 3 – Patient with Ischemic Bowel

Step 4: For a metabolic disturbance, is the respiratory system compensating OK?

"Winter's formula":

Expected PCO2 in metabolic acidosis

\[
\text{Expected PCO2} = 1.5 \times \text{HCO3} + 8 \quad \text{(range: +/- 2)}
\]

\[
= 1.5 \times 7 + 8 = 18.5
\]

<table>
<thead>
<tr>
<th>pH</th>
<th>7.18</th>
</tr>
</thead>
<tbody>
<tr>
<td>PCO2</td>
<td>20 mm Hg</td>
</tr>
<tr>
<td>HOC3</td>
<td>7 mEq / L</td>
</tr>
</tbody>
</table>
Step 5: For a metabolic acidosis, is there an increased anion gap?

FOR THIS STEP ONE MUST OBTAIN SERUM ELECTROLYTE DATA
Case 3 – Patient with Ischemic Bowel

SERUM ELECTROLYTE DATA

Serum sodium 135 mEq/L
Serum bicarbonate 7 mEq/L
Serum chloride 98 mEq/L
Anion Gap =
Serum Sodium –
Serum Chloride –
Serum Bicarbonate

Anion Gap =
= 135 - 98 -7 mEq/L
= 30 mEq/L
(ELEVATED)

SERUM ELECTROLYTE DATA

<p>| | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
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</tr>
</thead>
<tbody>
<tr>
<td>Serum sodium</td>
<td>135</td>
<td>mEq/L</td>
</tr>
<tr>
<td>Serum bicarbonate</td>
<td>7</td>
<td>mEq/L</td>
</tr>
<tr>
<td>Serum chloride</td>
<td>98</td>
<td>mEq/L</td>
</tr>
</tbody>
</table>
Case 3 – Patient with Ischemic Bowel

Step 5: For a metabolic acidosis, is there an increased anion gap?

ANSWER: YES
Case 3 – Patient with Ischemic Bowel

Step 6: For an increased anion gap metabolic acidosis, are there other derangements?

To determine if there are other metabolic derangements present we start by determining the “corrected bicarbonate concentration”: **Corrected HCO3 = measured HCO3 + (Anion Gap - 12)**. If the corrected HCO3 is less than normal (under 22mEq/L) then there is an additional metabolic acidosis present. Corrected HCO3 values over 26 mEq/L reflect a co-existing metabolic alkalosis.
Case 3 – Patient with Ischemic Bowel

Corrected HCO₃ = measured HCO₃ + (Anion Gap - 12).

Corrected HCO₃ = 7 + (30 - 12) = 25

REMEMBER

If the corrected HCO₃ is less than normal (under 22mEq/L) then there is an additional metabolic acidosis present. Corrected HCO₃ values over 26 mEq/L reflect a co-existing metabolic alkalosis.
Case 3 – Patient with Ischemic Bowel

Step 6: For an increased anion gap metabolic acidosis, are there other derangements?

ANSWER: NO OTHER DERANGEMENTS NOTED
Case 3 – Patient with Ischemic Bowel

ANSWER FROM

www.medcalc.com/acidbase.html

“Primary metabolic acidosis, with increased anion gap, with full respiratory compensation”
BUT … What is the cause of the elevated anion-gap metabolic acidosis?
Case 3 – Patient with Ischemic Bowel

The most common etiologies of a metabolic acidosis with an increased anion gap are shown below:

- Lactic acidosis  
  *(from poor perfusion)*
- Starvation
- Renal failure
- Ketoacidosis (as in diabetic ketoacidosis)

- Ingestion of:
  - Ethylene glycol
  - Methanol
  - Salicylate
Notes on Lactic Acidosis

“Lactic acidosis is a disease characterized by a pH less than 7.25 and a plasma lactate greater than 5 mmol/L.”

“Hyperlactemia results from abnormal conversion of pyruvate into lactate. Lactic acidosis results from an increase in blood lactate levels when body buffer systems are overcome. This occurs when tissue oxygenation is inadequate to meet energy and oxygen need as a result of either hypoperfusion or hypoxia.”

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Case 3 – Patient with Ischemic Bowel
Case 3 – Patient with Ischemic Bowel

By the time the patient is admitted to the ICU he looks absolutely terrible. He is moaning in agony, having received no pain medications at all.

Vital signs in ICU

- BP: 82/50
- HR: 112
- RR: 35
- Temp: 35.5 Celsius
- O2 sat: 84%
- Pain Score: 10/10
Case 3 – Patient with Ischemic Bowel

Because of the extreme pain, the patient is given morphine 8 mg IV push, a somewhat generous dose. When reexamined 15 minutes later the patient appears to be more comfortable. New vital signs are obtained.

- BP: 75/45
- HR: 102
- RR: 22
- Temp: 35.5 Celsius
- O2 sat: 82%
- Pain Score: 7/10
<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>BP</td>
<td>75/45</td>
</tr>
<tr>
<td>HR</td>
<td>102</td>
</tr>
<tr>
<td>RR</td>
<td>22</td>
</tr>
<tr>
<td>Temp</td>
<td>35.5 Celsius</td>
</tr>
<tr>
<td>O2 sat</td>
<td>82%</td>
</tr>
<tr>
<td>Pain Score</td>
<td>7/10</td>
</tr>
</tbody>
</table>

What is the next thing we should do for this patient?
Pulse Oximeter

Normal saturation is over 95% or better
Saturations under 90% constitute hypoxemia
Case 3 – Patient with Ischemic Bowel

ABGs obtained in the ICU after morphine has been given

pH 7.00 (was 7.18)
PCO2 25 mmHg (was 20)
HCO3 7 mEq/L

REMEMBER THAT MORPHINE IS A RESPIRATORY DEPRESSION AND WILL ELEVATE PCO2
Case 3 – Patient with Ischemic Bowel

pH 7.00

PCO2 25 mmHg

HCO3 7 mEq/L

Here is what MEDCALC says

“Primary metabolic acidosis, with increased anion gap, with superimposed respiratory acidosis”
Case 3 – Patient with Ischemic Bowel

“Primary metabolic acidosis, with increased anion gap, with superimposed respiratory acidosis”

BUT …

How could there be a respiratory acidosis when the PCO2 is very much below 40 mm Hg?

Normal Values (arterial blood)
- pH = 7.35 to 7.45
- PCO2 = 35 to 45 mm Hg
- HCO3 = 22 to 26 mEq/L
Case 3 – Patient with Ischemic Bowel

How could there be a respiratory acidosis when the PCO2 is very much below 40 mm Hg?

ANSWER

The expected degree of respiratory compensation is not present.
The expected degree of respiratory compensation is not present.

Expected PCO2 in metabolic acidosis

\[
= 1.5 \times \text{HCO3} + 8 \quad (\text{range: +/- 2})
\]

\[
= 1.5 \times 7 + 8 = 18.5
\]

BUT … we got a PCO2 of 25 mm Hg (as a result of respiratory depression from morphine administration) so the expected degree of respiratory compensation is not present.
Case 3 – Patient with Ischemic Bowel

THERAPY FOR THIS PATIENT

Oxygen

Metabolic tuning (blood sugar etc.)

Mechanical ventilation

Fluid resuscitation

Hemodynamic monitoring

Surgical, anesthesia, ICU consultation
Case 4 – Pregnant Woman with Persistent Vomiting
Case 4 – Pregnant Woman with Persistent Vomiting

A 23-year-old woman is 12 weeks pregnant. For the last 10 days, she has had worsening nausea and vomiting. When seen by her physician, she is dehydrated and has shallow respirations. Arterial blood gas data is as follows:

- pH: 7.56
- PCO2: 54 mm Hg
Step 1: Acidemic, alkalemic, or normal?

Step 2: Is the primary disturbance respiratory or metabolic?

Step 3: For a primary respiratory disturbance, is it acute or chronic?

Step 4: For a metabolic disturbance, is the respiratory system compensating OK?

Step 5: For a metabolic acidosis, is there an increased anion gap?

Step 6: For an increased anion gap metabolic acidosis, are there other derangements?
Step 1: Acidemic, alkalemic, or normal?

The pH of the arterial blood gas identifies it as alkalemic.

(Recall that the “normal range” for arterial blood pH is 7.35 to 7.45).
Step 2: Is the primary disturbance respiratory or metabolic?

The primary disturbance is metabolic, with the HCO3 being elevated. Since the PCO2 is raised in the face of an alkalemia, there is obviously not a primary respiratory disturbance – the raised PCO2 merely indicates that respiratory compensation has occurred.
**Step 3:** For a primary respiratory disturbance, is it acute or chronic?

Not applicable in this case.
Step 4: For a metabolic disturbance, is the respiratory system compensating OK?

The expected PCO2 in metabolic alkalosis is $0.7 \times \text{HCO}_3 + 20$ mmHg $= [0.7 \times 45] + 20 = 52$ mm Hg.

Since the actual PCO2 (54) and the expected PCO2 (52) are approximately the same, this suggests that respiratory compensation is appropriate.
Step 5: For a metabolic acidosis, is there an increased anion gap?

Not applicable in this case.
**Step 6:** For an increased anion gap metabolic acidosis, are there other derangements?

Not applicable in this case.
DIAGNOSIS

Metabolic Alkalosis from Persistent Vomiting

pH 7.56
PCO2 54 mm Hg
DIAGNOSIS:

Metabolic Alkalosis from Persistent Vomiting
Metabolic Alkalosis from Persistent Vomiting
**Acid-Base Interpretation:**

Primary metabolic alkalosis, with full respiratory compensation

(expected $P_{CO_2} = 52 - 56$)

expected $pH = 7.56$
expected $CO_2 = 54$
MERTABOLIC ALKALOSIS

Metabolic alkalosis is a primary increase in serum bicarbonate (HCO₃⁻) concentration. This occurs as a consequence of a loss of H⁺ from the body or a gain in HCO₃⁻. In its pure form, it manifests as alkalemia (pH >7.40).

As a compensatory mechanism, metabolic alkalosis leads to alveolar hypoventilation with a rise in arterial carbon dioxide tension (PaCO₂), which diminishes the change in pH that would otherwise occur.

emedicine.com
Nausea and vomiting in pregnancy is extremely common. Studies estimate nausea occurs in 66-89% of pregnancies and vomiting in 38-57%. The nausea and vomiting associated with pregnancy almost always begins by 9-10 weeks of gestation, peaks at 11-13 weeks, and resolves (in 50% of cases) by 12-14 weeks. In 1-10% of pregnancies, symptoms may continue beyond 20-22 weeks.

The most severe form of nausea and vomiting in pregnancy is called hyperemesis gravidarum (HEG). HEG is characterized by persistent nausea and vomiting associated with ketosis and weight loss (>5% of prepregnancy weight). HEG may cause volume depletion, altered electrolytes, and even death.

emedicine.com
Charlotte Bronte, the famous 19th century author of *Jane Eyre*, died of hyperemesis in 1855 in her fourth month of pregnancy.
Case 5 – Expedition to the Top of Mount Everest
The atmospheric pressure at the summit of Mount Everest (29,028') is about a third that at sea level. When an ascent is made without oxygen, extreme hyperventilation is needed if there is to be any oxygen at all in the arterial blood (a direct consequence of the alveolar gas equation).

Typical summit data (West 1983)

\[
\begin{align*}
\text{pH} & = 7.7 \\
\text{PCO2} & = 7.5
\end{align*}
\]
Pulmonary gas exchange was studied on members of the American Medical Research Expedition to Everest at altitudes of 8,050 m (barometric pressure 284 Torr), 8,400 m (267 Torr) and 8,848 m (summit of Mt. Everest, 253 Torr). Thirty-four valid alveolar gas samples were taken using a special automatic sampler including 4 samples on the summit. Venous blood was collected from two subjects at an altitude of 8,050 m on the morning after their successful summit climb. Alveolar CO2 partial pressure (PCO2) fell approximately linearly with decreasing barometric pressure to a value of 7.5 Torr on the summit. For a respiratory exchange ratio of 0.85, this gave an alveolar O2 partial pressure (PO2) of 35 Torr. In two subjects who reached the summit, the mean base excess at 8,050 m was -7.2 meq/l, and assuming the same value on the previous day, the arterial pH on the summit was over 7.7. Arterial PO2 was calculated from changes along the pulmonary capillary to be 28 Torr. In spite of the severe arterial hypoxemia, high pH, and extremely low PCO2, subjects on the summit were able to perform simple tasks. The results allow us to construct for the first time an integrated picture of human gas exchange at the highest point on earth.
<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
<th>Normal Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>7.7</td>
<td>7.36 - 7.44</td>
</tr>
<tr>
<td>$P_{CO_2}$</td>
<td>7.5 mm Hg</td>
<td>36 - 44 mm Hg</td>
</tr>
<tr>
<td>$HCO_3^-$</td>
<td>9 mEq/L</td>
<td>22 - 26 mEq/L</td>
</tr>
<tr>
<td>Sodium ($Na^+$)</td>
<td></td>
<td>mEq/L</td>
</tr>
<tr>
<td>Bicarbonate ($HCO_3^-$)</td>
<td>9</td>
<td>mEq/L</td>
</tr>
<tr>
<td>Chloride ($Cl^-$)</td>
<td></td>
<td>mEq/L</td>
</tr>
<tr>
<td>Albumin</td>
<td></td>
<td>g/dL</td>
</tr>
<tr>
<td>Anion Gap</td>
<td></td>
<td>mEq/L</td>
</tr>
</tbody>
</table>

Acid-Base Interpretation:

Acute (uncompensated) primary respiratory alkalosis

- pH > 7.66 and HCO3 > 16, for acute (uncompensated)
- pH < 7.46 and HCO3 < 8, for chronic (compensated)

expected pH = 7.7
expected CO2 = 8
The End
<table>
<thead>
<tr>
<th></th>
<th>units</th>
<th>Arterial</th>
<th>Venous</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td></td>
<td>7.4</td>
<td>7.35</td>
</tr>
<tr>
<td>$\text{HCO}_3$</td>
<td>mEq/L</td>
<td>20-24</td>
<td>20-24</td>
</tr>
<tr>
<td>$P_{\text{CO}}$</td>
<td>mmHg</td>
<td>32-40</td>
<td>40-45</td>
</tr>
<tr>
<td>$\text{H}_2\text{CO}_3$</td>
<td>mMoles/L</td>
<td>$\text{PCO}_2 \times 0.03$</td>
<td>$\text{PCO}_2 \times 0.03$</td>
</tr>
<tr>
<td>$P_{\text{O}}$</td>
<td>mmHg</td>
<td>80-110</td>
<td>40</td>
</tr>
</tbody>
</table>
## Control System for Respiratory Regulation of Acid-base Balance

<table>
<thead>
<tr>
<th>Control Element</th>
<th>Physiological or Anatomical Correlate</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Controlled variable</td>
<td>Arterial pCO$_2$</td>
<td>A change in arterial pCO$_2$ alters arterial pH (as calculated by use of the Henderson-Hasselbalch Equation).</td>
</tr>
<tr>
<td>Sensors</td>
<td>Central and peripheral chemoreceptors</td>
<td>Both respond to changes in arterial pCO$_2$ (as well as some other factors)</td>
</tr>
<tr>
<td>Central integrator</td>
<td>The respiratory center in the medulla</td>
<td></td>
</tr>
<tr>
<td>Effectors</td>
<td>The respiratory muscles</td>
<td>An increase in minute ventilation increases alveolar ventilation and thus decreases arterial pCO$_2$ (the controlled variable).</td>
</tr>
</tbody>
</table>

# Metabolic Acidosis: Increased Anion Gap

<table>
<thead>
<tr>
<th>Disorder</th>
<th>Osmolar Gap</th>
<th>Blood Glucose</th>
<th>BUN/Cr</th>
<th>L-Lactate</th>
<th>Ketones</th>
<th>Blood Level</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ethylene Glycol Ingestion</td>
<td>Very High</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>Negative</td>
<td>Yes</td>
</tr>
<tr>
<td>Methanol Ingestion</td>
<td>Very High</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>Negative</td>
<td>Yes</td>
</tr>
<tr>
<td>Alcoholic Acidosis (ethanol)</td>
<td>High</td>
<td>Low-Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>Positive</td>
<td>Yes</td>
</tr>
<tr>
<td>Diabetic Acidosis</td>
<td>High</td>
<td>Elevated</td>
<td>Normal</td>
<td>Normal</td>
<td>Positive</td>
<td></td>
</tr>
<tr>
<td>Renal Failure (Severe)</td>
<td>High</td>
<td>Normal</td>
<td>High</td>
<td>Normal</td>
<td>Negative</td>
<td></td>
</tr>
<tr>
<td>Lactic Acidosis</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>High</td>
<td>Negative</td>
<td></td>
</tr>
<tr>
<td>D-Lactic Acidosis</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>Negative</td>
<td>Yes</td>
</tr>
<tr>
<td>Toluene Exposure</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>Negative</td>
<td></td>
</tr>
<tr>
<td>Salicylate Ingestion</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>Negative</td>
<td>Yes</td>
</tr>
</tbody>
</table>
## Metabolic Acidosis

**Normal Anion Gap**

<table>
<thead>
<tr>
<th>Disorder</th>
<th>FEHCO3-</th>
<th>Urine pH</th>
<th>UAG</th>
<th>[K+]</th>
<th>NaHCO3 Rx</th>
<th>BUN/Cr</th>
</tr>
</thead>
<tbody>
<tr>
<td>Distal RTA (Type I)</td>
<td>&lt;15%</td>
<td>&gt;5.5</td>
<td>Positive</td>
<td>Normal</td>
<td>Small Dose</td>
<td>Normal</td>
</tr>
<tr>
<td>Proximal RTA (Type II)</td>
<td>&gt;15%</td>
<td>&lt;5.5</td>
<td>Negative</td>
<td>Low</td>
<td>Large Dose</td>
<td>Normal</td>
</tr>
<tr>
<td>Generalized RTA (Type IV)</td>
<td>&lt;15%</td>
<td>&lt;5.5</td>
<td>Positive</td>
<td>High</td>
<td>Small Dose</td>
<td>Normal</td>
</tr>
<tr>
<td>Renal Failure (Mild)</td>
<td>&lt;15%</td>
<td>&lt;5.5</td>
<td>Negative</td>
<td>Normal</td>
<td>Small Dose</td>
<td>High</td>
</tr>
<tr>
<td>GI Losses of HCO3-</td>
<td>&lt;15%</td>
<td>&lt;5.5</td>
<td>Negative</td>
<td>Normal</td>
<td>Variable</td>
<td>Normal</td>
</tr>
<tr>
<td>Acid Load</td>
<td>&lt;15%</td>
<td>&lt;5.5</td>
<td>Negative</td>
<td>Normal</td>
<td>Normal</td>
<td></td>
</tr>
<tr>
<td>Dilutional Acidosis</td>
<td>&lt;15%</td>
<td>&lt;5.5</td>
<td>Negative</td>
<td>Normal</td>
<td>Normal</td>
<td></td>
</tr>
</tbody>
</table>
# Metabolic Acidosis

**Normal Anion Gap**

<table>
<thead>
<tr>
<th>Disorder</th>
<th>FEHCO3-</th>
<th>Urine pH</th>
<th>UAG</th>
<th>[K+]</th>
<th>NaHCO3 Rx</th>
<th>BUN/Cr</th>
</tr>
</thead>
<tbody>
<tr>
<td>Distal RTA (Type I)</td>
<td>&lt;15%</td>
<td>&gt;5.5</td>
<td>Positive</td>
<td>Normal</td>
<td>Small Dose</td>
<td>Normal</td>
</tr>
<tr>
<td>Proximal RTA (Type II)</td>
<td>&gt;15%</td>
<td>&lt;5.5</td>
<td>Negative</td>
<td>Low</td>
<td>Large Dose</td>
<td>Normal</td>
</tr>
<tr>
<td>Generalized RTA (Type IV)</td>
<td>&lt;15%</td>
<td>&lt;5.5</td>
<td>Positive</td>
<td>High</td>
<td>Small Dose</td>
<td>Normal</td>
</tr>
<tr>
<td>Renal Failure (Mild)</td>
<td>&lt;15%</td>
<td>&lt;5.5</td>
<td>Negative</td>
<td>Normal</td>
<td>Small Dose</td>
<td><strong>High</strong></td>
</tr>
<tr>
<td>GI Losses of HCO3-</td>
<td>&lt;15%</td>
<td>&lt;5.5</td>
<td>Negative</td>
<td>Normal</td>
<td>Variable</td>
<td>Normal</td>
</tr>
<tr>
<td>Acid Load</td>
<td>&lt;15%</td>
<td>&lt;5.5</td>
<td>Negative</td>
<td>Normal</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dilutional Acidosis</td>
<td>&lt;15%</td>
<td>&lt;5.5</td>
<td>Negative</td>
<td>Normal</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
# Metabolic Alkalosis

<table>
<thead>
<tr>
<th>Disorder</th>
<th>Urine [Cl(^-)]</th>
<th>Urine Ca</th>
<th>Serum Mg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gastrointestinal Losses of H(^+)</td>
<td>Low</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Contraction Alkalosis</td>
<td>Low</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Loop Diuretics</td>
<td>High</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Thiazide Diuretics</td>
<td>High</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bartter’s Syndrome</td>
<td>High</td>
<td>High</td>
<td>Normal</td>
</tr>
<tr>
<td>Gitelman’s Syndrome</td>
<td>High</td>
<td>Low</td>
<td>Low</td>
</tr>
<tr>
<td>Posthypercapnic Alkalosis</td>
<td>Variable</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
## Some Aids to Interpretation of Acid-Base Disorders

<table>
<thead>
<tr>
<th>&quot;Clue&quot;</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>High anion gap</td>
<td><em>Always</em> strongly suggests a metabolic acidosis.</td>
</tr>
<tr>
<td>Hyperglycaemia</td>
<td>If ketones present also diabetic ketoacidosis</td>
</tr>
<tr>
<td>Hypokalemia and/or hypochloremia</td>
<td>Suggests metabolic alkalosis</td>
</tr>
<tr>
<td>Hyperchloremia</td>
<td>Common with normal anion gap acidosis</td>
</tr>
<tr>
<td>Elevated creatinine and urea</td>
<td>Suggests uremic acidosis or hypovolemia (prerenal renal failure)</td>
</tr>
<tr>
<td>Elevated creatinine</td>
<td>Consider ketoacidosis: ketones interfere in the laboratory method (Jaffe reaction) used for creatinine measurement &amp; give a falsely elevated result; typically urea will be normal.</td>
</tr>
<tr>
<td>Elevated glucose</td>
<td>Consider ketoacidosis or hyperosmolar non-ketotic syndrome</td>
</tr>
<tr>
<td>Urine dipstick tests for glucose and ketones</td>
<td>Glucose detected if hyperglycaemia; ketones detected if ketoacidosis</td>
</tr>
</tbody>
</table>

http://www.anaesthesiamcq.com/AcidBaseBook/ab9_2.php