

Arterial Blood Gases



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Introduction



- ❧ Arterial blood gases analysis is an essential component of diagnosing & managing critically ill patients in ICU.
- ❧ Proper knowledge, understanding & application of concepts of acid-base disorders will help clinician to follow the course of progress patients & to evaluate effectiveness of treatment.

Procedure



- ❧ Explain the procedure & its need beforehand to patient.
- ❧ Radial artery is preferred for collection.
- ❧ Wrist is positioned in hyper-extension.
- ❧ 22-gauge needle is used.
- ❧ Artery palpated with first three fingers of one hand, then remove middle finger & with the syringe (already flushed with heparin) in other hand like pencil and skin is entered at 45° .

Allen's test

9780702061905, "Arterial Blood Gases Made Easy", 2nd Edition, ©2016 Elsevier

ABG SAMPLING TECHNIQUE

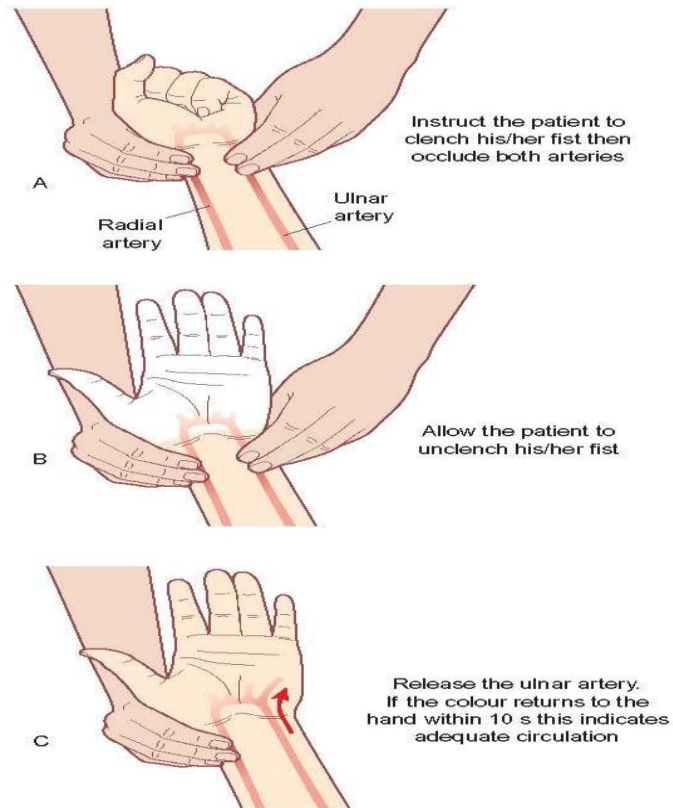


Figure 20 Modified Allen test.

Procedure



- ❧ Slow advancement yield pulsatile blood flow in the syringe.
- ❧ Never redirect needle without withdrawing to subcutaneous tissue.
- ❧ 2 to 4 ml blood should be obtained.
- ❧ Pressure applied for at least 2 min.



Potential Errors



❧ Sampling Error

Air contamination – spurious increase in pO₂

Duration of exposure is more important than volume of air bubbles.

Expel air immediately

Discard sample if froth is present.

❧ Venous admixture

Absence of flash of blood & pulsation on entry into vessel. Absence of auto-filling of syringe.

Discard such sample.

Potential Errors



❧ Anticoagulant effect

Dilution error leads to drop in $p\text{CO}_2$ & $p\text{O}_2$. pH usually remains unchanged.

❧ Metabolism effect

if sample is kept for long (>10 min) without cooling adequately, then RBCs consume O_2 , produce CO_2 & lower pH.

Normal Values



	Normal Range	For Calculation
pH	7.35-7.45	7.40
pCO ₂	35-45	40
pO ₂	>80	95
HCO ₃	22-26	24
SaO ₂	>92%	100%

All the values are at **BTPS** i.e. Body temperature (37 deg celsius), P is atmospheric pressure at which body is exposed, S is saturated with maximum water vapour.

Difference B/W ABG & VBG



NORMAL BLOOD GAS VALUES

	ARTERIAL BLOOD	MIXED VENOUS BLOOD
pH	7.40 (7.35-7.45)	7.38 (7.33-7.43)
PO ₂	80-100 mm Hg	35-40 mm Hg
O ₂ Sat	95% or greater	70-75%
PCO ₂	35-45 mm Hg	41-51 mm Hg
HCO ₂	22-26 mEq/L.	24-28 mEq/L.
Base Excess (B.E.)	-2 - +2	0 - +4 -

Basic Terms



- ❧ **Acidosis:** is the process which leads to decrease in pH, unless there is a compensatory process or an opposite disturbance. This **leads to Acidemia**.
- ❧ **Acidemia:** is the state of low blood pH. (Tissue pH cannot be calculated)
- ❧ **Alkalosis:** is the process which leads to increase in pH, unless there is a compensatory process or an opposite disturbance. This **leads to Alkalemia**.
- ❧ **Alkalemia:** is the state of high blood pH.

Remember



- ❧ So “Acidosis can exist without producing Acidemia, but Acidemia cannot exist in absence of Acidosis.”
- ❧ The same corollary holds true for Alkalosis & Alkalemia.
- ❧ Acidemia & Alkalemia are surrogate markers for their respective processes.

Buffer System

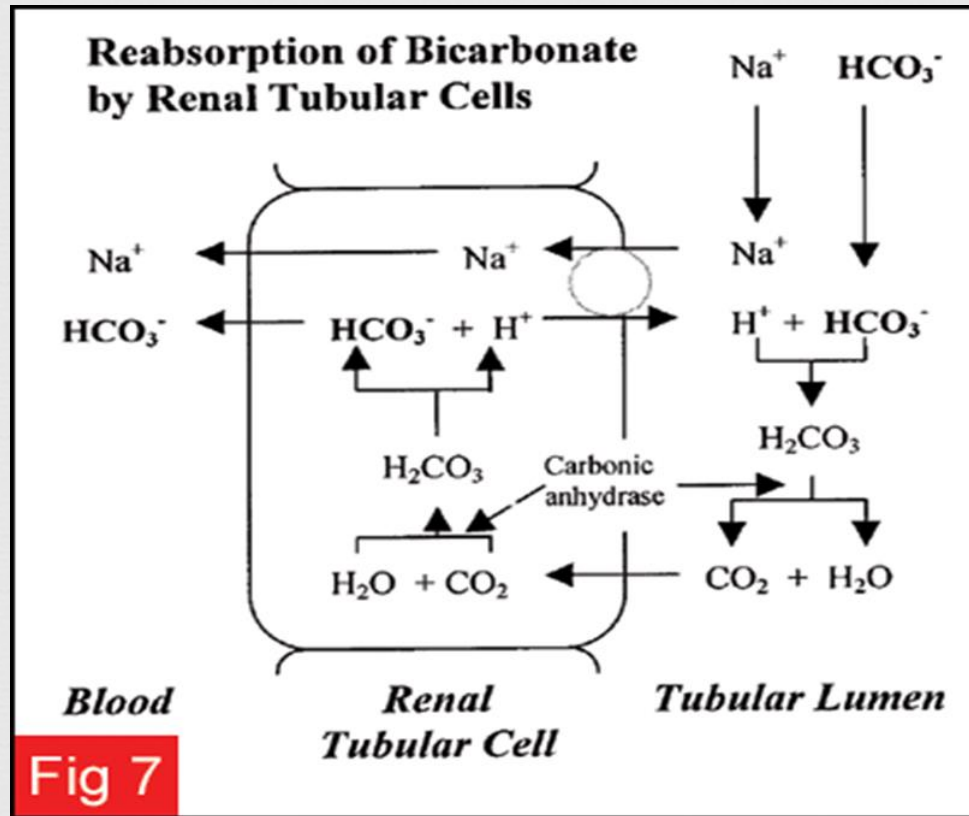


- ❧ A Buffer is a Substance that readily accept or donate hydrogen ions.
- ❧ The bicarbonate and non-bicarbonate buffers(hemoglobin, proteins, and phosphates) .
- ❧ Role of Bicarbonate is important, as it is the pre-dominant buffer.

Renal Buffering mechanism

1. Active exchange of sodium ions for hydrogen ions.
2. Carbonic Anhydrase.

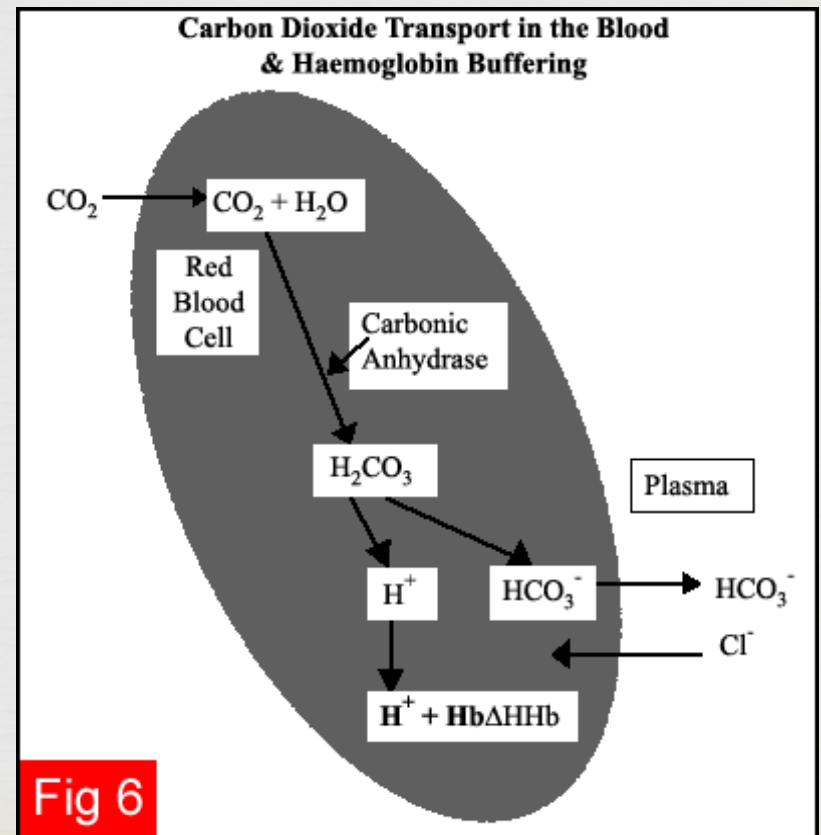
Re-absorption of HCO_3^- ions



Respiratory Component



- ❧ The major role of the lungs is to excrete the CO_2 produced.
- ❧ 98% of acid load is in the form of CO_2 .
- ❧ Seen in 3 forms
 1. Dissolved CO_2 (5%)
 2. Carbamino-Hb (25-30%)
 3. Bicarbonate (65-70%)



Interpretation of ABG



☞ Assess Oxygenation

1. Observe values of pO_2 & SaO_2 .
2. Note the PaO_2 / FiO_2 ratio.

Normal ratio is 1:400-1:500.

Less than 1:400 suggests V/Q mismatch or diffusion defect or shunt.

Less than 1:300 suggestive of ARDS.

3. A-a gradient: $PAO_2 - PaO_2$

Interpretation of ABG



Here pAO_2 is alveolar pO_2 (calculated by alveolar gas equation) & paO_2 is arterial pO_2 (measured value in ABG).

Normal A-a gradient: 5-10 mmHg (for young, non-smoker adult)

Conservative Approach is = $\text{Age}(\text{in yrs})/4 + 4$

Alveolar gas equation:

$$PAO_2 = [FiO_2 \times (P_{\text{atm}} - P_{H_2O}) - (PaCO_2)/0.8]$$

Under normal condition: $PAO_2 = 150 - PaCO_2/0.8$

Interpretation of ABG



☞ Assess Acid-base disorder

1. Look at pH – is it acidemia or alkalemia
2. Check CO₂ & HCO₃ to determine whether the primary problem is metabolic or respiratory.
3. If the primary disorder is respiratory, determine whether it is acute or chronic
4. Apply rules of compensation to know whether it is a simple or mixed disorder.
5. Mind the gaps – anion, delta & osmolar gap.

Interpretation of ABG



1. Look at pH

Acidemia can be due to low HCO_3 or high pCO_2

Alkalemia can be due to high HCO_3 /low pCO_2

2. Look at CO_2 & HCO_3 to determine whether the primary problem is respiratory or metabolic. **The change which is in the direction of pH is the primary change in most of the cases.**

Eg. $\text{pH}=7.29$, $\text{pCO}_2=72$, $\text{pO}_2=58$, $\text{HCO}_3=29.4$, $\text{SaO}_2=84\%$
 pH shows acidemia, pCO_2 change is in that direction.
So primary problem is **Respiratory Acidosis (uncompensated)**.

Normal pH is possible under three circumstances:

No acid-base disturbance exists

A single acid-base disturbance is being fully compensated

At least two acid-base disorders co-exist:

A primary acidemia is being balanced out by a primary alkalemia

CO_2 and HCO_3^- are both in the normal range.

CO_2 and HCO_3^- are both low:

Either of the two following disturbances is present:

CO_2 and HCO_3^- are both high:

Either of the two following disturbances is present:

CO_2 and HCO_3^- are both high

CO_2 and HCO_3^- are both low

CO_2 and HCO_3^- are both normal:

Compensated respiratory alkalosis

Compensated metabolic acidosis

Compensated respiratory acidosis

Compensated metabolic alkalosis

A primary respiratory acidosis + a primary metabolic alkalosis

A primary respiratory alkalosis + a primary metabolic acidosis

A primary metabolic acidosis is balancing out a primary metabolic alkalosis. Because the HCO_3^- is normal, CO_2 is normal

Respiratory Change



- ❧ Respiratory change can be either Respiratory Acidosis or Respiratory Alkalosis
- ❧ Respiratory Acidosis (high $p\text{CO}_2$): is due to primary rise in blood $p\text{CO}_2$ level. The change can be compensated or remain uncompensated.
- ❧ Hypercapnia (increased blood $p\text{CO}_2$) is primarily due to alveolar **hypoventilation**.

Respiratory Acidosis



❧ Etiology

- 1. Respiratory center involvement:** Opioid medication intoxication, other sedative drugs or due to anaesthetic drugs effect. Head trauma involving brain stem, severe metabolic encephalopathy & Bulbar Polio
- 2. Neural pathway involvement:** Traumatic transection of spinal cord, Guillain-Barré syndrome, Amyotrophic Lateral sclerosis.

Respiratory Acidosis



3. **N-M junction involvement:** Myasthenia Gravis, Botulism, due NM blocker drugs.
4. **Chest Wall involvement:** Flail chest, Kyphoscoliosis
5. **Upper Airway involvement:** Foreign body aspiration, OSAS, Extra- or Intra-luminal obstruction (LN/tumorous growth).
6. **Lung Parenchyma involvement:** OAD eg. AECOPD or Acute severe asthma in later stages.

Respiratory Alkalosis



✧ Respiratory Alkalosis (low $p\text{CO}_2$): is due to primary fall in blood $p\text{CO}_2$ level. The change can be compensated or remain uncompensated.

Respiratory Alkalosis



∞ Etiology

1. Early stages of an **asthma** exacerbation
2. **Panic** attack
3. Acute hypoxia (hypoxic ventilatory response)
4. **Pregnancy** or other cases of **elevated progesterone**
5. Cirrhosis and/or hepatic encephalopathy
6. **Salicylate intoxication**

Compensatory Mechanism



✧ **Respiratory Acidosis:** it is imperative to distinguish whether process is Acute or Chronic.

1. **Acute Resp. Acid.:**

↑PaCO₂ by 10mmHg then ↑HCO₃ by 1 mEq/L

2. **Chronic Resp. Acid.:**

↑PaCO₂ by 10mmHg then ↑HCO₃ by 3 mEq/L

Compensatory Mechanism



❧ **Respiratory Alkalosis:** it is imperative to distinguish whether process is Acute or Chronic.

1. Acute Resp. Alk.:

↓PaCO₂ by 10mmHg then ↓HCO₃ by 2 mEq/L

2. Chronic Resp. Alk.:

↓PaCO₂ by 10mmHg then ↓HCO₃ by 4 mEq/L

Metabolic Change



- ❧ Metabolic Change can be either Metabolic Acidosis or Metabolic Alkalosis.
- ❧ **Metabolic Acidosis** (low HCO_3): is due to low HCO_3 level in blood. The change can be compensated or remain uncompensated.
- ❧ **Metabolic Alkalosis** (high HCO_3): is due to high HCO_3 level in blood. The change can be compensated or remain uncompensated.

Metabolic Acidosis



- ✧ In all cases of Metabolic Acidosis it is important to calculate Anion Gap.
- ✧ **Anion Gap** = $\text{Na} - (\text{HCO}_3 + \text{Cl})$
Normal range: $12 \pm 2 \text{ mEq/L}$
- ✧ Anion Gap denotes the **Unmeasured Anions** in the body.
- ✧ Anion gap increases when either **unmeasured anions in body decrease** or unmeasured cations increase.

Always Remember



- ✧ In Body there is always Electro-neutrality maintained.
- ✧ All Cations (M & UM) = All Anions (M & UM).
- ✧ M/c cause for **High anion gap acidosis** is $\uparrow$ UM anions, due to loss of HCO_3 ions (Renal/Extrarenal) or addition of Extrinsic Acid (proton ions).
- ✧ In **Normal Anion Gap Met. Acidosis**: the reason for maintenance of AG is, that the **insulting acid is HCl**, so with addition of one H ion there is addition of one Cl ion cancelling the net effect & in **case were there is HCO_3 loss it accompanied by H ion loss.**

Metabolic Acidosis



⌘ Etiology: **High Anion Gap**

1. Methanol
2. Uremia (chronic)
3. Diabetic ketoacidosis
4. Paraldehyde
5. Infection, Isoniazid, Iron
6. Lactate
7. Ethanol, Ethylene glycol
8. Rhabdomyolysis, Renal Failure
9. Salicylate

Metabolic Acidosis



∞ Etiology: **Normal Anion Gap**

- | | | |
|---------------------------------|---|-----------------------------|
| 1. Hypoaldosteronism | } | Renal HCO ₃ loss |
| 2. Acetazolamide | | |
| 3. Renal Tubular Acidosis (RTA) | | |
| 4. Diarrhea | } | G.I. HCO ₃ loss |
| 5. Uretero-sigmoidostomy | | |
| 6. Post-ventilation Hypocapnia | | |

Metabolic Acidosis



- ✧ In case of Hypoalbuminemia with every 1 gm% decrease in albumin below 4gm%, addition of 2-3 to gap.
- ✧ Adjusted AG = Calculated AG + 2.5 (4 – S. Alb)

Mind The Gaps



✧ In case of metabolic acidosis after calculating Anion Gap, Calculation of Delta Gap & Delta Ratio is highly helpful to know whether there is singular pure AGMA or NAGMA or combined AGMA+NAGMA or concurrent AGMA+Met. Alk.

✧ **Delta Gap** = Delta Anion Gap - Delta HCO₃

In this Delta AG = Pt's AG - 12

Delta HCO₃ = 24 - Pt's HCO₃

Delta Gap



∞ Delta Ratio (Δ/Δ) = $\Delta\text{AG}/\Delta\text{HCO}_3$

Δ/Δ : 1-2, uncomplicated AGMA, eg Lactic acidosis = 1.6 & DKA = 1 as unmeasured anions i.e ketones are washed in urine.

Δ/Δ : <0.4, Hyperchloremic NAGMA

Δ/Δ : 0.4-0.8, combined AGMA+NAGMA

Δ/Δ : >2, AGMA & concurrent Met. Alk.

Base Excess



Under standard conditions...

"Positive" base excess: metabolic alkalosis

The amount of acid that returns the pH of the blood sample to normal.

"Negative" base excess: metabolic acidosis

The amount of alkali that returns the pH of a blood sample to normal.

Standard base excess

Calculated as for blood with a hemoglobin of 5 gm/dL.

The buffering capacity of hemoglobin is averaged out for the whole body including the extracellular fluid

Base Excess/Deficit



⌘ Normal range of Base Excess is: -2 to +2

Metabolic Alkalosis



- ❧ Metabolic Alkalosis is a condition in which blood HCO_3 level increases.
- ❧ It can be due to HCO_3 gain or due to volume contraction.
- ❧ It can be divided into Saline responsive & Saline unresponsive, based on Urine spot Cl level.

Metabolic Alkalosis



❧ Chloride/Saline Responsive Alkalosis:

- Vomiting/Nasogastric suction
- Chloride wasting Diarrhoea
- Diuretic therapy

❧ Chloride/Saline Un-responsive Alkalosis:

- Hyperaldosteronism
- Cushing's syndrome
- Exogenous Steroids, Licorice ingestion
- Bartter's syndrome, Liddle's syndrome
- Excess alkali intake (eg. milk alkali syndrome)

Case-1



⌘ A patient presents with a one-day history of productive cough, fevers and increasing dyspnea. In the ER, the chest x-ray shows a right middle lobe opacity. His oxygen saturation is 90% on room air and an arterial blood gas is obtained. It reveals a pH 7.55, PCO₂ 30, PO₂ 63, HCO₃⁻ 22.

Case-1



- ❧ Step 1: The pH is high (alkalemia)
- ❧ Step 2: The PCO₂ is low (respiratory alkalosis) and the bicarbonate is on the low side of normal. A high pH with a low PCO₂ indicates that the primary process is a respiratory alkalosis.
- ❧ Summary: In this case, the patient is likely hyperventilating because he is hypoxemic. This is a good example of the hypoxemic ventilatory response.

Case-2



⌘ A patient with Type I diabetes presents to the ER complaining of feeling poorly two days after running out of his insulin. An arterial blood gas is obtained and shows pH 7.25, PCO₂ 28, PO₂ 95, HCO₃⁻ 15.

Case-2



- ❧ Step 1: The pH is low (acidemia)
- ❧ Step 2: The PCO₂ is low (respiratory alkalosis) and the bicarbonate is low (metabolic acidosis). A low pH and low bicarbonate signifies that the metabolic acidosis is the primary process.
- ❧ Summary: In this case, the patient is likely in diabetic ketoacidosis because he was not taking insulin.

Case-3



❧ A 30 year-old woman is brought into the emergency room with altered mental status. Her friends found her at home with an empty bottle of aspirin by her bedside. Her SpO₂ is 99% and she is obviously hyperventilating on exam. An arterial blood gas is obtained as part of her initial work-up and reveals: pH 7.56, PCO₂ 22, PO₂ 110, HCO₃ ⁻17. On her chemistry panel, her sodium is 137, chloride 99, bicarbonate 18.

Case-3



- ❧ Step 1: The pH is high (alkalemia)
- ❧ Step 2: The PCO₂ is low (respiratory alkalosis) and the bicarbonate is low (metabolic acidosis). A high pH with a low PCO₂ indicates that the primary process is a respiratory alkalosis.
- ❧ Step 3: The serum anion gap is elevated at 20. As a result, she also has an elevated anion gap acidosis going on at the same time.
- ❧ Summary: This combination of alkalemia due to a primary respiratory alkalosis with a concurrent elevated anion gap metabolic acidosis is classic for salicylate intoxication.

Case-4



✧ A 40 year-old mountain climber ascends to an elevation of 15,000 feet. She remains there for a period of 3 weeks as part of a research project. At the end of that time, she has an arterial blood gas drawn which shows pH 7.44, PCO₂ 24 PO₂ 55, HCO₃⁻ 16. The serum anion gap was normal (11).

Case-4



- ❧ Step 1: The pH is high (alkalemia)
- ❧ Step 2: The PCO₂ is low (respiratory alkalosis) and the bicarbonate is low (metabolic acidosis). Therefore, the respiratory alkalosis is the primary process.
- ❧ Step 3: The serum anion gap normal.
- ❧ Step 4: The metabolic acidosis is the compensatory process for the respiratory alkalosis.
- ❧ Summary: If you had measured the ABG immediately upon arrival, the bicarbonate would have been close to normal and the pH would have been much higher. This is because it takes several days for metabolic compensation to occur.

Case-5



⌘ A 65 year-old man with very severe COPD (FEV1 = 25% predicted) has an arterial blood gas done as part of routine pulmonary function testing to help determine if he requires home oxygen therapy. The sample shows pH 7.36, PCO₂ 60, PO₂ 60, HCO₃⁻ 36. The anion gap is 8

Case-5



- ❧ Step 1: The pH is low (acidemia)
- ❧ Step 2: The PCO₂ is high(respiratory acidosis) and the bicarbonate is high (metabolic alkalosis). Therefore, the respiratory acidosis is the primary process.
- ❧ Step 3: The serum anion gap is normal at 8.
- ❧ Step 4: The metabolic alkalosis is the compensatory process for the respiratory acidosis.
- ❧ Summary: This patient likely has chronic carbon dioxide retention due to his very severe COPD. Because this is a long-standing process, he has had adequate time for metabolic compensation to occur.



☞ JUST THE LAST ONE LEFT.

☞ Please Do Not CURSE.

Case-6



⌘ A 40 year-old woman develops a severe case of diarrhea with multiple loose bowel movements over the course of a one-day period. When she presents to the ER, an arterial blood gas is obtained and shows pH 7.35, PCO₂ 32, PO₂ 75, HCO₃⁻ 18. The anion gap is 10

Case-6



- ❧ Step 1: The pH is low (acidemia)
- ❧ Step 2: The PCO₂ is low (respiratory alkalosis) and the bicarbonate is low (metabolic acidosis). Therefore, the metabolic acidosis is the primary process.
- ❧ Step 3: The serum anion gap is normal at 10. The metabolic acidosis from Step 2, therefore, is a non-gap acidosis.
- ❧ Step 4: The respiratory alkalosis is the compensatory process for the metabolic acidosis.

Case-6



Summary: The patient's metabolic acidosis is likely due to her diarrhea as diarrhea leads to bicarbonate loss through the lower gastrointestinal tract. Even though her symptoms have been present for only one day, her pH has come back up towards normal already. That is because respiratory compensation for metabolic processes occurs almost immediately. In severe acidosis, this may not be enough to bring the pH back close to normal but at a minimum respiratory compensation starts quickly.



"Either write something
worth reading or do
something worth writing."

Benjamin Franklin