# Acid-Base disorders

## Objectives:

- State the normal value for PH,PCO2,HCO3
- Understand the basic mechanism of acid base disturbance
- Interpret basic acid base disturbance
- List common differential diagnosis for different acid base disorder

[ Color index : Important | Notes | Extra ]

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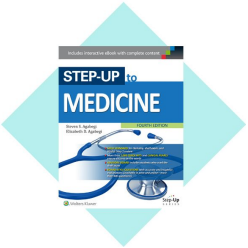
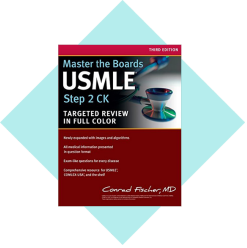
## Resources:

- 435 slides.
- Oxford medicine.
- Linda physiology fifth edition .
- 434 Team.







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"Medicine is an art, nobody can deny it."

# Introduction

3: 21 minutes

## ★ Normal value

- Arterial blood pH = 7.35 – 7.45 → 7.4
  - pH between 7.35 and 7.45 can be either normal or because of mixed acid-based disturbances..
- $PaCO_2$ = 35-45 → 40
- Serum $HCO_3$-= 22-26 → 24
- Anion gap = 8-12

## ★ Basic RECALL

- Acid-base balance is concerned with maintaining a normal hydrogen ion concentration in the body fluids. This balance is achieved by utilization of buffers in extracellular fluid and intracellular fluid, by respiratory mechanisms that excrete carbon dioxide, and by renal mechanisms that reabsorb bicarbonate and secrete hydrogen ions.
- Blood pH refers to the level of $H^+$ ions and maintained by several buffering systems.
  - A decrease in blood pH is called acidaemia and is caused by acidosis.
  - An increase in blood pH is called alkalemia and is caused by alkalosis.
- Disturbances of acid-base balance are described as either metabolic or respiratory, depending on whether the primary disturbance is in $HCO_3^-$ or $CO_2$.

## ★ Buffering

- A buffered solution resists a change in pH.
- Most importantly the **bicarbonate-carbonic acid buffer pair** that depends on the balance between bicarbonate ions and carbonic acid.

$$CO_2 + H_2O \rightleftarrows H_2CO_3 \rightleftarrows HCO_3^- + H^+$$

## ★ Primary disturbance ( read quickly to pick up the main point then after studying the lecture read it again )

| Primary disorder | Respiratory acidosis | Respiratory alkalosis | Metabolic acidosis | Metabolic alkalosis |
|---|---|---|---|---|
| Problem | Hypoventilation | Hyperventilation | Gain of $H^+$ or loss of $HCO_3$ | Gain of $HCO_3^-$ or loss of $H^+$ |
| PH | ↓ | ↑ | ↓ | ↑ |
| $HCO_3^-$ | ↑ | ↓ | ↓ ↓ | ↑ ↑ |
| $PaCO_2$ | ↑ ↑ | ↓ ↓ | ↓ | ↑ |

★ **Two arrows** indicate initial disturbance. Red arrows indicate Compensatory response.

# Respiratory Acidosis

## ★ Definition

Increased $PaCO_2$ and decreased pH.

## ★ Mechanism

- Alveolar **Hypoventilation** → Accumulation of $CO_2$ → Increases in $PaCO_2$ → Respiratory acidosis → pH decreases.
- $HCO_3$ will increase (Compensation) but it needs time (12 -24 h ) as the kidney need time to compensate.

## ★ Etiology **Hypoventilation** of any cause:

| | |
|---|---|
| **CNS** | Damage of the respiratory center in the brainstem Caused by: Stroke , Hemorrhage , Trauma , Tumor , Medication ( Commonly sleeping pills eg. benzodiazepines like lorazepam, other: morphine, anesthetics and narcotics ) |
| **Peripheral nervous system** | Demyelinating disease Of PNS ex. guillain-barré syndrome[1] |
| **Neuromuscular junction** | Myasthenia gravis |
| **Muscular disease** | Intercostal muscle atrophy, such as:<br>● Duchenne dystrophy ● Congenital muscle atrophy |
| **Chest wall** | Severe scoliosis |
| **Bronchial tree** | **COPD** [2](Emphysema, chronic bronchitis, severe asthma): (irreversible bronchoconstriction not responding to bronchodilators → retain $CO_2$ → exchange gases lung defect→ leading to acute/chronic Respiratory acidosis) |
| **Other** | Drowning , Sleep apnea and Morbid obesity. |

## ★ Clinical Features:

- **Symptoms** : Somnolence, confusion, myoclonus with asterixis (Flapping tremors
- **Signs** of acute CO2 retention: headaches, confusion, and papilledema[3][4].

[1] usually follow diarrhea or flu like illness → followed by ascending paralysis from legs going up (reach respiratory muscles)..
[2] Any disorder that reduces CO2 clearance (i.e., inhibits adequate ventilation) can lead to respiratory acidosis.
[3] Papilledema is optic disc swelling that is secondary to elevated intracranial pressure
[4] Pathophysiology :Increased PaCO2 causes increased cerebral blood flow which increases CSF pressure→ Resulting in generalized CNS depression.

## ★ Classification:

Each of the simple respiratory disorders has two ranges of expected values, one for the **acute** disorder and one for the chronic disorder. The **acute disorder** is present **before renal compensation has occurred,** and, therefore, values for blood **pH** tend to be **more abnormal**. The chronic disorder is present once renal compensation has occurred, which takes several days (starts within 24 hours). Renal mechanisms increase the excretion of H+ within 24 hours and may correct the resulting acidosis caused by chronic retention of CO2 to a certain extent. Because of the compensatory process, values for blood pH tend to be more normal in the chronic phase.

| | **Acute** Respiratory Acidosis | **Chronic** Respiratory Acidosis |
|---|---|---|
| **Causes** | 1. **Respiratory**: airway obstruction, severe pneumonia, chest trauma/pneumothorax<br>2. Acute **drug intoxication** : narcotics, sedatives<br>3. **Residual neuromuscular blockade**.<br>4. **CNS** disease (head trauma) | 1. Chronic lung disease (**COPD**)<br>2. Neuromuscular disease<br>3. Extreme **obesity**<br>4. Chest wall deformity<br>5. Muscular eg. Duchenne dystrophy |
| **pH** | **LOW** | **NORMAL** due compensatory mechanism. |
| **Compensation** | - **Immediate** compensatory ↑ of $HCO_3$.<br>- $HCO_3$ ↑ by 1 mEq/l for every 10 mmHg ↑ in PaCO2. | $HCO_3$ ↑ by 3-3.5 mEq/l for every 10 mmHg ↑ in $PaCO_2$ (Due to renal adaptation) |

Patients with COPD (with irreversible damage ) in their serious state they could have chronic respiratory acidosis ( ↑ $PaCO_2$, ↑ $HCO_3$ and pH is normal) but when they present acutely the homeostasis will be disturbed, pH will be low, ↑ $PaCO_2$, $HCO_3$ stay the same. (acute on top of chronic)

## ★ Treatment:

- Verify patency of airways.
- Give supplemental oxygen : If $PaO_2$ is low (<60 mmHg) , Oxygen is contraindicated in COPD patients as it can exacerbate symptoms.
- Treat underlying cause.
- Intubation and mechanical ventilation might be required for:
  - Severe acidosis (PH <7).
  - $PaCO_2$ > 60 or inability to increase $PaO_2$.
  - Mental deterioration.
  - Impending respiratory fatigue.

# Respiratory Alkalosis

## ★ Definition:

Decreased $PaCO_2$ and increased pH.

## ★ Mechanism:

- Alveolar **hyperventilation** → increased wash out $CO_2$ → decrease in $PaCO_2$ → increased pH.
- Compensation : $HCO_3$- will decrease after (12 -24 h).

## ★ Etiology Hyperventilation [5] of any Cause:

- Overaggressive mechanical ventilation.
- **Anxiety** (most common + severe respiratory alkalosis) , Fever ( not severe ) 2nd most common.
- Pain , Sepsis , Pregnancy[6](mild respiratory alkalosis) , Hepatic failure (cirrhosis)
- Hypoxemia , Restrictive lung disease
- Medication (salicylate toxicity eg.aspirin overdose[7][8])
- Severe congestive heart failure, Thyrotoxicosis.
- Pulmonary embolism , asthma , pneumonia.

## ★ Clinical Features:

- (lightheadedness, dizziness, anxiety, paresthesias, and perioral numbness) [9]
- Tetany [10] ,Arrhythmias, Trousseau's sign and Chvostek's sign may be positive

## ★ Classification:

| Acute Respiratory Alkalosis | Chronic Respiratory Alkalosis |
|---|---|
| $HCO_3$ ↓ by 2 mEq/l for every 10 mmHg ↓ in $PaCO_2$. | $HCO_3$ ↓ by 4-5 mEq/l for every 10 mmHg ↓ in $PaCO_2$. |

## ★ Treatment:

- Treat the underlying cause.
- Sometimes this does not need to be treated (e.g.,in the case of pregnancy).

[5] Any disorder that increases the respiratory rate inappropriately can lead to respiratory alkalosis
[6] increase serum prostaglandin → Hyperventilation.
[7] overstimulation of respiratory centre → Hyperventilation
[8] Aspirin can cause both respiratory alkalosis and metabolic acidosis
[9] Symptoms are mostly related to decreased cerebral blood flow (vasoconstriction)
[10] indistinguishable from hypocalcemia

- Breathe into paper bag to recycle the exhaled CO2 (especially who have anxiety ).

## Metabolic Acidosis

★ **Definition**: Decreased $HCO_3$ and decreased pH.

★ **Mechanism:**

- Increase acid gaining either Exogenous Intake or Endogenous production . Or Decrease acid excretion. Or Loss or decrease production of bicarbonate.
- Compensation[11]: Hyperventilation → decrease $PCO_2$ immediately. $PaCO_2$ ↓ by 1 mmHg for every 1 mEq/l ↓ in HCO3.
- If the kidneys are intact and the primary cause of acidosis is not renal in origin, the kidney can gradually increase acid secretion over days to weeks and restore a new steady state.

★ **The Anion gap:**

- The difference between primary measured cations (Na+ and K+) and the primary measured anions (Cl- and HCO3-) in serum :
  - Anion gap = cations - anions → AG= ([Na+] + [K+]) - ([Cl-] + [HCO3-])

  Or
  - Anion gap =Sodium - (Chloride+Bicarbonate)→AG = [Na+] - ([Cl-] + [HCO3-]).
- It is helpful in determining the cause of a metabolic acidosis

★ **Classification & Etiology:** The doctor said you only have to know the main etiology, don't dig deep in all the details. For example in Normal AG metabolic acidosis, we should know that it's caused by **diarrhea** and **renal conditions** (eg. Tubular acidosis)

- Normal AG Acidosis → The low $HCO_3$ is associated with high Cl–, so that the AG remains normal.

| | | |
|---|---|---|
| GI | loss of $HCO_3$ | **Diarrhea** , fistula in intestine or pancreas , Ureterosigmoidostomy: (colon secretes HCO3– in urine in exchange for Cl) |
| RENAL[12] | ↓ $HCO_3$ reabsorption. | Proximal Tubular Acidosis (RTA Type 2) |
| | ↓ production of $HCO_3$ | Distal Tubular Acidosis (RTA Type 1 ) |
| | Carbonic anhydrase inhibition | Due to diuretics as acetazolamide. |
| | Early renal failure | Impaired generation of ammonia |
| Other | Post- hypocapnia | Respiratory alkalosis→ renal wasting of HCO3 →rapid correction of |

[11] Metabolic problems always show compensation.

[12] To distinguish between RTA & Diarrhea we perform Urine Anion Gap (UAG= Sodium - Chloride) :
- In RTA there is a defect in acid secretion → so less Cl into urine→ result of UAG positive number.
- In Diarrhea Excretion acid is intact→ H+ is excreted with Cl- in urine → UAG negative number.

| | | |
|---|---|---|
| | | respiratory alkalosis → transient Acidosis until HCO3 regenerated |
| | Dilutional | Due to rapid infusion of bicarbonate - free IV fluids. |

- High Anion Gap Acidosis:

| Problem | | Causes |
|---|---|---|
| **Increased Endogenous production** | **Lactic acidosis** | • Low tissue perfusion (decreased oxygen delivery to tissues)<br>• Shock states (septic, cardiogenic, hypovolemic)<br>• Excessive expenditure of energy(e.g.,seizures) |
| | **Diabetic Ketoacidosis** | DM, Prolonged starvation and prolonged alcohol abuse |
| | **Uremia** | **Renal failure** ( accumulation of organic anions such as phosphate, sulfates,etc) |
| **Increased Exogenous Intake** | **Oxalic acid** | Ethylene glycol overdose/intoxication (manifistations include MS, cardiopulmonary failure calcium oxalate crystals and renal failure) |
| | **Formic acid** | Methanol overdose (manifistations include blurred vision) |
| | **Salicylates**[13] | Aspirin overdose |
| | **Other** | Paraldehyde , Acetaminophen , alcohol |

★ In summary: 1) Gain acid from A) Outside : alcohol "ethanol , methanol" or B) Inside: renal failure, lactic acidosis, ketoacidosis 2) Loss HCO3 from diarrhea or RTA

## ★ REMEMBER !

In metabolic acidosis you have to calculate the Anion Gap, and we have two types:

**1- High Anion Gap**

Remember that in metabolic acidosis with high anion gap the acid can be:

a. Endogenous
- +ve Ketones: ketoacidosis as a result of starvation or diabetic ketoacidosis
- -ve ketones: lactic acidosis as a result of ischemia or hypoxia, or uremia in renal failure.

b. Exogenous (eg. alcohol, ethanol, methanol, paraldehyde and aspirin overdose)

Here we calculate the osmolar gap to differentiate between the etiologies (**endogenous or exogenous**).osmolar gap is the difference between the **calculated osmolality** (2 x [**Na** mmol/L]+[**glucose** mmol/L]+[**urea** mmol/L] ) "fixed", and the **measured osmolality.**

OG = measured serum osmolality – calculated osmolality

They should be equal, the difference should not be more than 10 mOsm/kg unless you have added osmoles from outside, then the osmolar gap will be >10 mOsm/kg.

So if the cause is **exogenous (eg. Alcohol) the OG will be >10**

**2- Normal Anion Gap**

Remember two things : Diarrhea and Renal tubular acidosis, by history you'll be able to figure out which one.

[13] Salicylate overdose causes both primary respiratory alkalosis and primary metabolic acidosis.

## ★ Clinical Features:

- Hyperventilation ( deep rhythmic breathing ) also called Kussmaul respiration.
- Decreased in Cardiac output and tissue perfusion.

## ★ Treatment:

1. Treat the underlying cause.
2. Sodium bicarbonate is sometimes used in severe acidosis (esp. in normal AG acidosis).
3. Mechanical ventilation might be needed if the patient is fatigued (esp. in DKA)

# Metabolic Alkalosis

## ★ Definition:

Increased pH and increased $HCO_3$.

## ★ Mechanism:

- Initiating metabolic alkalosis by either:
  - Gaining of $HCO_3$-
  - Or Loss of acid ( $H^+$ ) ex: from **vomiting**.
- Maintaining Metabolic alkalosis due to the kidney inability to excrete the excess $HCO_3$[14]
- Compensation : Hypoventilation → increased $PCO_2$ (respiratory Acidosis) immediately ($PaCO_2$ ↑ by 0.6 mmHg for every 1 mEq/l ↑ in $HCO_3$ ).

## ★ Classification & causes:

| | **Saline Responsive Urine (cl-) <20 (Commonest )** | **Saline resistant Urine (cl-) >20** |
|---|---|---|
| **Definition** | Metabolic alkalosis with ECF contraction (due to → fluid loss). | Metabolic alkalosis with ECF volume expansion ( no fluid loss ) |
| **Causes** | - Gastric loss of H+ and generation of $HCO_3$ such as **vomiting** ( HCl loss ) , NGT drainage<br>- **Diuretic use** → These decrease the ECF volume, body $HCO_3$ content is normal, but plasma $HCO_3$ increases due to ECF contraction.<br>- Volume depletion<br>- Post-hypercapnia<br>- Villous adenoma of colon, diarrhea with high chloride content | **Hypertensive**:<br>- Hyperaldosteronism either primary or secondary.<br>- Non-mineralocorticoid, Cushing Syndrome<br>**Hypo/normo tensive:**<br>- Exogenous alkali load either IV or oral sodium bicarbonate<br>- Bartter's syndrome & Gitelman's syndrome, Severe hypokalemia |
| **Treatment** | **Treat by saline due to volume depletion** | **According to Etiology** |

[14] Uncomplicated metabolic alkalosis is typically transient , because kidney can normally excrete the excess $HCO_3$-

## ★ Clinical Features:

There is no characteristic signs and symptoms (most imp. Hx)

## ★ Treatment:

- Treat the underlying cause.
- Give normal saline plus potassium in saline responsive.
- Spironolactone (K+ sparing diuretic) might be considered in saline resistant.

# Steps in Acid-Base Analysis

8: 38 min

★ Normal value

- Arterial blood pH = 7.35 – 7.45 → 7.4
- $PaCO_2$ = 35-45 → 40
- Serum $HCO_3$-= 22-26 → 24
- Anion gap = 8-12

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➔ **Step 1**: Acidemic or Alkalemic?

- pH <7.35 → acidosis
- pH >7.45 → alkalosis.

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

- To determine whether the disturbance affects primarily the arterial $PaCO_2$ or the serum $HCO_3$.
- Primary disturbance is in $CO_2$ → Respiratory (normal value 35-45)
- Primary disturbance is in $HCO_3$ → Metabolic (normal value 22-26)

➔ **Step 3**: Is the respiratory disturbance acute or chronic?

- Acute respiratory acidosis: $HCO_3$ increase by 1 mEq/l for every 10 mmHg increase in $PaCO_2$.
- Chronic respiratory acidosis: $HCO_3$ increase by 3-3.5 mEq/l for every 10 mmHg increase in $PaCO_2$.
- Acute respiratory alkalosis: $HCO_3$ decrease by 2 mEq/l for every 10 mmHg decrease in $PaCO_2$.
- Chronic respiratory alkalosis: $HCO_3$ decrease by 4-5 mEq/l for every 10 mmHg decrease in $PaCO_2$.

➔ **Step 4:** For a metabolic acidosis, is there an increased anion gap?

- Anion gap = [Sodium] - ([Chloride] + [Bicarbonate]) (normal AG 8-12)
- Serum Osmolality = (2 x (Na + K)) + (BUN ) + (glucose )

➔ **Step 5:** Are there other metabolic processes present in a patient with an increased anion gap metabolic acidosis?

➔ **Step 6:** Is the respiratory system compensating adequately for a metabolic disturbance?

- Metabolic acidosis: $PCO_2$ decreases by 1 mmHg for every 1 mEq/l decrease in $HCO_3$
- Metabolic alkalosis: $PCO_2$ increases by 0.6 mmHg for every 1 mEq/l increases in $HCO_3$

## Important cases from the doctor slides

### Case study 1:

| | | |
|---|---|---|
| pH = 7.2 | $PaCO_2$ = 60 | $HCO_3$= 24 |

1) Is it acute or chronic ? Note that the pH is abnormal and the $HCO_3$ is within normal.
   Remember: Acute respiratory acidosis: $HCO_3$- ↑ by 1 mEq/l for every 10 mmHg increase in $PaCO_2$ while in Chronic respiratory acidosis: HCO3- ↑ by 3-3.5 mEq/l for every 10 mmHg increase in PaCO2
2) What is the primary problem ? Acute respiratory acidosis.
3) Is there compensation ? No
4) Your differential diagnosis ? Could be anything from the etiology box eg. tumor in the brain stem, stroke, guillain-barré syndrome..etc.
5) Treatment ? Treat underlying cause.

### Case study 2:

What do you expect the ABG in the following patient to be:

1) 24 years old male with acute shortness of breath and wheezes for two days: Acute respiratory acidosis
2) Past history of bronchial asthma : **Depends on the severity**, patient initially might come hyperventilated (respiratory alkalosis) then normalizing, after that might go to respiratory acidosis. Acidosis is the most severe stage because patient will collapse.
3) 67 years old women, HTN, DM II, COPD and presenting with cough and shortness of breath: Acute respiratory acidosis or chronic respiratory acidosis or acute in top of chronic.

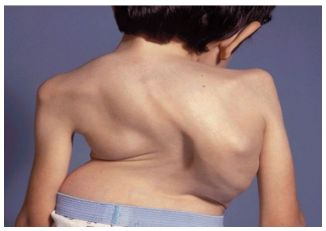
### Case study 3:

| | | |
|---|---|---|
| pH = 7.25 | $PaCO_2$ = 52 | $HCO_3$ = 20 |

1) What is the primary problem? Acute respiratory acidosis + Secondary problem (metabolic acidosis )
2) Is there compensation ? No
3) Your differential diagnosis ? Could be anything from the etiology box eg. tumor in the brain stem, stroke, guillain-barré syndrome..etc.

## Case study 4:

| | | |
|---|---|---|
| pH = 7.32 | $PaCO_2$ = 55 | $HCO_3$ = 19 |



1) What is the primary problem? Chronic respiratory acidosis + Secondary problem (metabolic acidosis )
2) Is there compensation ? Yes
3) Your differential diagnosis ? We have picture here so, scoliosis.
4) What other investigations you want to do? Anion gap

## Case study 5:

56 years old male with history of COPD is admitted with 1 week history of dyspnea, productive cough and diarrhea:

| | | | |
|---|---|---|---|
| Na = 125 | Cl = 103 | BUN = 42 | Glucose = 100 |
| K = 3.5 | $HCO_3$ = 10 | Creat = 1.4 | ABG, pH = 7.14 |
| $PCO_2$ = 30 | $pO_2$ = 50 | | |

1) What is the predominant acid base disorder ? Metabolic Acidosis
   Calculate the anion gap for your differential diagnosis.
   AG = [Sodium] -([Chloride] + [Bicarbonate])
   AG = [125] - (103 + 10)
   AG = 12
   it's normal so etiology is either diarrhea or RTA, but most likely diarrhea bc of the history..
2) What $pCO_2$ is expected with normal respiratory compensation ?
   40 – (1*(24-10)) = 26 This is not full compensation because pCO2 is 30 in this patient which indicates an underlying primary respiratory acidosis, suggested by the Hx of COPD, dyspnea, and productive cough (lungs not able to appropriately compensate)

## Case study 6:

32 years old male presented with two days history of intractable vomiting;

| | | | |
|---|---|---|---|
| pH = 7.51 | $PCO_2$ = 41 | Na = 132 | Cl = 90 |
| K = 3.4 | $HCO_3$ = 33 | Creatinine = 1.6 | |

What is the predominant acid-base disorder ? Metabolic Alkalosis secondary to vomiting, treat him with isotonic saline to correct his volume depletion.

What pCO2 is expected with normal respiratory compensation? 40 + (33 – 24) * (~ 0.6) = 45.4 mmHg; since the measured pCO2 < 44.8 ↔ 45.6, there is also a primary respiratory alkalosis (inappropriate hyperventilation)

## Case study 7:

A 58 year old man presents to the Emergency Department with abdominal pain and hypotension. Investigations reveal the following:

| | | | |
|---|---|---|---|
| Na = 140 | K = 4 | Cl = 90 | $HCO_3$ = 5 |
| pH = 6.8 | $PCO_2$ = 36 | $PO_2$ = 7 | |

Analyze the acid- base disorders seen in the patient.
Primary condition is Metabolic acidosis
AG = [Sodium] - ([Chloride] + [Bicarbonate])
AG = [140] - (90 + 5)
AG = 45 (high) most likely diagnosis based on the history Renal Failure or Lactic Acidosis.
If the scenario is changed to
12 year old boy presented with vomiting and abdominal pain → It could be DKA.

## Quiz from the doctor slides:

| | pH | $PCO_2$ | $HCO_3$ | Interpretation |
|---|---|---|---|---|
| 1 | 7.41 | 40 | 24 | |
| 2 | 7.5 | 42 | 35 | |
| 3 | 6.72 | 40 | 5 | |
| 4 | 7.26 | 63 | 25 | |
| 5 | 7.52 | 18 | 25 | |

Answers

1. Normal
2. Metabolic alkalosis
3. Metabolic acidosis
4. Respiratory acidosis
5. Respiratory alkalosis

## Summary

### Acid base balance disorders

| Primary disorder | Respiratory acidosis | Respiratory alkalosis | Metabolic acidosis | Metabolic alkalosis |
|---|---|---|---|---|
| PH | ↓ | ↑ | ↓ | ↑ |
| $HCO_3$ | ↑ | ↓ | ↓ ↓ | ↑ ↑ |
| $PaCO_2$ | ↑ ↑ | ↓ ↓ | ↓ | ↑ |
| Main problem and important note | Hypoventilation due to problems in ( CNS , PNS , muscles , chest wall and bronchial tree ) | Hyperventilation due to ( **Anxiety**, pregnancy and Thyrotoxicosis) | - Used AG to differentiate between the Etiology<br>- Gain H from **outside** "ethanol , methanol" or from **inside** "renal failure, lactic acidosis, ketoacidosis "<br>- loss $HCO_3$ from "diarrhea or RTA " | - Gain of $HCO_3$ due to medication (diuretic) , dehydration .<br>- loss of H " vomiting" |

Two arrows indicate initial disturbance. Red arrows indicate Compensatory response.

## Cases

1) **A 20 year old man presents with obtundation. Past medical history is unobtainable. Blood pressure is 120/70 without orthostatic change, and he is well perfused peripherally. The neurological examination is non focal. His laboratory values are as follows:**

**Na: 138 mEq/L**
**K: 4.2 mEq/L**
**HCO3: 5 mEq/L**
**Cl: 104 mEq/L**
**Creatinine: 1.0 mg/dL**
**BUN: 14 mg/dL**
**Ca: 10 mg/dL**
**Arterial blood gas on room air: PO2 96, PCO2 15, pH 7.02**
**Blood glucose: 90 mg/dL**
**Urinalysis: normal, without blood, protein, or crystals**
**Which of the following is the most likely acid-base disorder?**

a. Pure normal anion-gap metabolic acidosis
b. Respiratory acidosis
c. Pure high anion-gap metabolic acidosis

d. Combined high anion-gap metabolic acidosis and respiratory alkalosis
e. Combined high anion-gap metabolic acidosis and respiratory acidosis

**2) A 17-year-old man is brought to the emergency room with confusion and incoordination. He is uncooperative and refuses to provide further history. Physical examination reveals an RR of 30; the vital signs are otherwise normal as is the general physical examination. Laboratory values are as follows:**
**Na: 135 mEq/L**
**K: 2.7 mEq/L**
**HCO3: 15 mEq/L**
**Cl: 110 mEq/L**
**Arterial blood gases: PO2 92, PCO2 30, pH 7.28**
**Urine: pH 7.5, glucose—negative**
**Ca: 9.7 mg/dL**
**PO4: 4.0 mg/dL**
**Which of the following is the most likely cause of the acid base disorder?**
a. GI loss owing to diarrhea
b. Proximal renal tubular acidosis
c. Disorder of the renin-angiotensin system
d. Distal renal tubular acidosis
e. Respiratory acidosis

**3) A 73-year-old woman with arthritis presents with confusion. Neurologic examination is non focal, and CT of the head is normal. Laboratory data include:**
**Na: 140 mEq/L**
**K: 3.0 mEq/L**
**Cl: 107 mEq/L**
**HCO3: 12 mEq/L**
**Arterial blood gases: PO2 62, PCO2 24, pH 7.40**
**What is the acid-base disturbance?**
a. Respiratory alkalosis with appropriate metabolic compensation
b. High anion-gap metabolic acidosis with appropriate respiratory compensation
c. Combined metabolic acidosis and respiratory alkalosis
d. No acid-base disorder
e. Hyperchloremic (normal anion gap) metabolic acidosis with appropriate respiratory compensation

**4) A 27-year-old woman presents to the emergency room with a panic attack. She appears healthy except for tachycardia and a respiratory rate of 30. Electrolytes include calcium 10.0 mg/dL, albumin 4.0 g/dL, phosphorus 0.8 mg/dL, and magnesium 1.5 mEq/L. Arterial blood gases include pH of 7.56, PCO2 21 mm Hg, and PO2 99 mm Hg. Which of the following is the most likely cause of the hypophosphatemia?**
a. Hypomagnesemia
b. Hyperparathyroidism
c. Respiratory alkalosis with intracellular shift
d. Poor dietary intake
e. Vitamin D deficiency

**5) A 32-year-old builder presents in accident and emergency in a distressed state. He reports suffering from chest pain for the last 2 weeks, the pain is sharp and only occurs when he moves heavy objects. He has a family history of cardiovascular disease and is worried about a heart attack. His blood gas findings are as follows: pH = 7.47; PCO2 = 3.3; PO2 = 15.3; bicarbonate = 17.53. The most likely diagnosis is:**
a. Respiratory acidosis with metabolic compensation
b. Acute metabolic acidosis
c. Respiratory alkalosis with metabolic compensation
d. Metabolic acidosis with respiratory compensation

e. Acute respiratory alkalosis

## Answers

**1)** **C.** The first step in analyzing an acid-base disturbance is simply to look at the pH. This patient has an acidosis. Then look at the HCO3 and the PCO2 to determine the primary disturbance; that is, is it a metabolic acidosis or a respiratory acidosis? The serum HCO3 has decreased from 24 to 5 mEq/L, so this must be a metabolic acidosis. The PCO2 is below the normal value of 40 mm, so this cannot be a respiratory acidosis (the PCO2 would be above 40 in a respiratory acidosis). The first two steps are straightforward and unambiguous.
The third (and most difficult) step is to assess the compensatory response. This patient has a metabolic acidosis, so you need to assess the respiratory compensation. That is to say, has the PCO2 decreased appropriately to compensate for the metabolic acidosis? The normal compensatory response in metabolic acidosis is for the PCO2 to decrease by 1 to 1.5 mm Hg for each 1-mEq decrease in HCO3. This patient's 19 mEq/L drop in bicarbonate is matched by a 25-mm drop in the PCO2. Hence, this is a compensated metabolic acidosis. Another method of assessing compensation in a metabolic acidosis is to use the Winters formula, which says that the appropriate PCO2 equals 1.5 (HCO3) + 8. This would give an appropriate PCO2 of 15.5, very close to the measured PCO2. Again, the compensatory response is appropriate for the degree of acidosis; the patient does not have a respiratory acid-base disorder.
The fourth step is to calculate the anion gap. The normal anion gap is 8 to 12 mEq/L; in this case the value is 29 mEq/L. Therefore, this is an anion-gap metabolic acidosis with appropriate respiratory compensation. A brief differential of anion-gap metabolic acidosis is as follows:

- Diabetic ketoacidosis
- Lactic acidosis
- Ketoacidosis
- Toxic alcohol (methanol, ethylene glycol) ingestion
- Salicylate intoxication
- Renal failure

**2)** **D.** The patient has a metabolic acidosis. Respiratory compensation is appropriate, and the anion gap is normal. Therefore, he has a hyperchloremic (normal anion gap) metabolic acidosis. Common causes include renal tubular acidosis, bicarbonate loss owing to diarrhea, and mineralocorticoid deficiency.
In a metabolic acidosis, the urine pH should be low (ie, the patient should be trying to excrete the excess acid). This patient's high urine pH is therefore diagnostic of renal tubular acidosis (RTA).
Proximal RTA is associated with glycosuria, phosphaturia, and aminoaciduria (Fanconi syndrome). Since the serum phosphorus is normal, and glycosuria is absent, proximal RTA is unlikely. GI loss of bicarbonate caused by diarrhea would be associated with an appropriately acidic urine (pH < 5.5).
Disorders of the renin-angiotensin-aldosterone system are associated with hyperkalemia, not hypokalemia. The low PCO2 excludes respiratory acidosis. So, this patient has a distal RTA, probably because of toluene inhalation (glue sniffing). Toluene can lead to life-threatening metabolic acidosis and hypokalemia.

**3)** **C**. This patient's normal pH would initially suggest a normal acid-base status. However, the PCO2 is significantly low, indicating a respiratory alkalosis. If the pH is normal, there must be a superimposed metabolic acidosis; that is, metabolic compensation would not return the pH all the way back to 7.4. Indeed, the serum bicarbonate is too low for a compensatory response (metabolic compensation for respiratory alkalosis rarely drops the HCO3 below 17 mEq/L) and the anion gap is elevated at 21. The only cause of a substantially elevated anion gap is metabolic acidosis (the AG can be elevated to 16 or 17 in alkalosis). Therefore, this patient has a combined (mixed) disturbance, that is, combined respiratory alkalosis and metabolic acidosis. This is the classic acid-base disturbance associated with salicylate intoxication. Aspirin stimulates central respiratory drive; in addition, several metabolic substances (salicylic acid and lactic acid due to suppression of oxidative phosphorylation, among others) build up to widen the anion gap. Choices a, b, and e are wrong because compensation never normalizes the pH.

**4)** **C**. Respiratory alkalosis is one of the commonest causes of hypophosphatemia; it results from shift of phosphate from the extracellular to the intracellular space. Hypomagnesemia alone would increase phosphorus by decreasing parathormone effect. Hyperparathyroidism can decrease phosphorus, but not to this degree; also, calcium is not elevated. Severe hypophosphatemia is seen with malnutrition, especially during the refeeding stage when carbohydrate intake causes phosphate to shift into the intracellular space. Such patients have clear clinical evidence of malnutrition. In addition, malnutrition almost always causes hypoalbuminemia. Vitamin D deficiency is uncommon in this age group and would be associated with hypocalcemia.

**5)** **E**. The history in this case suggests the patient's chest pain is due to muscular injury rather than anything more sinister. The patient's anxiety about cardiovascular morbidity has ultimately resulted in hyperventilation causing an acute respiratory alkalosis (e). Acid base abnormalities can be solved by either considering the Henderson–Hassel Bach equation ($CO2 + H20 \leftrightarrow H2CO3 \leftrightarrow H+ + HCO3-$), whereby change in the product(s) on one side of the equation is balanced by a shift in equilibrium. For example, in this case the patient's hyperventilation causes a reduction in CO2, in order to increase the CO2, the equilibrium shifts towards CO2 + H2O which causes a reduction in H+ (alkalosis) and HCO3–. This process occurs in respiratory alkalosis with metabolic compensation (c). If the patient had a true cardiac arrest it would cause a surge in lactic acidosis hence H+ concentration increases causing a metabolic acidosis (b). In order to balance this change, the equilibrium shifts away from H+ and causes increased CO2 production which can manifest as an increased respiratory rate, otherwise called 'metabolic acidosis with respiratory compensation' (d). In a respiratory acidosis with metabolic compensation (a) scenario, a patient may have a respiratory abnormality such as chronic hypoventilation. The accumulation of CO2 which leads to increased H+ is compensated for by bicarbonate which is subsequently reduced. In more chronic conditions, the bicarbonate becomes elevated.