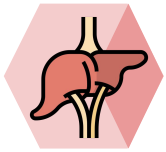
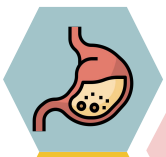
Medicine
TEAM 437

MED437
KING SAUD UNIVERSITY

Acid-Base disorders


Objectives :

- Develop an approach to acid base problems
- Identify the primary acid base disturbance
- Solve simple acid base cases

Done by :

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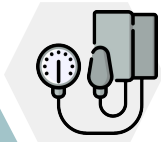


Revised by :

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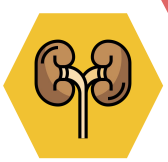
Resources :

- Doctor ‘s slides - Team 436 - Davidsons

Lecturer: Dr. Talal Alfaadhel

Same 436 lecture Slides: Yes




Introduction

VIDEO

Normal value

- Arterial blood pH = 7.35 – 7.45 while normal pH of venous blood is 7.31-7.41
- PaCO2 = 35-45 → 40
- Serum HCO3-= 22-26 → 24
- Anion gap = 8-12

Normal values are different due to different references. Check the given reference in the test. maintaining normal pH is important to maintain the integrity of protein function (Enzyme function and membrane proteins).

Rule of 4 for the normal values:-

pH	**7.4**	7.35-7.45
HCO3	**24**	22-26
CO2	**40**	35-45

Basic RECALL

O Acid-base balance is concerned with maintaining a normal hydrogen ion concentration in the body fluids. This balance is achieved by buffers utilization of in extracellular fluid and intracellular fluid, by respiratory mechanisms that excrete carbon dioxide, and by renal mechanisms that reabsorb bicarbonate and secrete hydrogen ions.

O Blood pH refers to the level of H+ ions and maintained by several buffering systems.

- A decrease in blood pH is called acidemia and is caused by acidosis.
- An increase in blood pH is called alkalemia and is caused by alkalosis.

O Disturbances of acid-base balance are described as either metabolic or respiratory, depending on whether the primary disturbance is in HCO3 − or CO2.

- The assessment of acid base abnormalities is typically done using arterial blood gases (ABG)
- Given the ease of obtaining venous blood gases (VBG) and capillary blood gases (CBG) these are often used in clinical practice
- Always check the reference range in your local laboratory

Buffering

O A buffered solution resists a change in pH.

O Most importantly the bicarbonate-carbonic acid buffer pair that depends on the balance between bicarbonate ions and carbonic acid.

- In compensation, Ph gets close to the normal value but does not reach normal. our bodies compensate by 3 mechanisms: Breathing in or out (if it is metabolic), Excreting / retaining hydrogen (respiratory) and Buffers in blood.
- In cases of normal Ph levels, look at the patient's health. if the patient has a disease with a normal pH level think of mixed acidosis and alkalosis. If the patient appears healthy with a normal pH they're most probably normal

$$CO2 + H2O \rightleftarrows H2CO3 \rightleftarrows HCO3- + H+$$

Definitions

Disorder	Definition & Mechanism
Metabolic acidosis	**loss of [HCO3] or addition of [H+]** Process that primarily reduces bicarbonate: Excessive H+ formation e.g. lactic acidosis, ketoacidosis. Reduced H+ excretion e.g. renal failure. Excessive HCO3 loss e.g. diarrhoea. • Increase acid gaining either Exogenous Intake or Endogenous production. Or Decrease acid excretion. Or Loss or decrease production of bicarbonate. • Compensation[10]: Hyperventilation → decrease PCO2 immediately. • 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
Metabolic alkalosis	**loss of [H+] or addition of [HCO3]** Process that primarily raises bicarbonate: Extracellular fluid volume loss e.g. due to vomiting or diuretics. Excessive potassium loss with subsequent hyperaldosteronism. • Initiating metabolic alkalosis by either: - Gaining of HCO3- - Or Loss of acid (H+) ex: from vomiting. • Maintaining Metabolic alkalosis due to the kidney inability to excrete the excess HCO3[13] • Compensation: Hypoventilation → increased PCO2 (respiratory Acidosis) immediately (PaCO2 ↑ by 0.6 mmHg for every 1 mEq/l ↑ in HCO3).
Respiratory acidosis	**increase in pCO2** Process that primarily causes elevation in PaCO2: Reduced effective ventilation e.g. many chronic respiratory diseases or drugs depressing the respiratory centre. • Alveolar Hypoventilation → Accumulation of CO2 → Increases in PaCO2 → Respiratory acidosis → pH decreases. • HCO3 will increase (Compensation) but it needs time (12 -24 h) as the kidney need time to compensate.
Respiratory alkalosis	**decrease in pCO2** Process that primarily causes reduction in PaCO2: Increased ventilation e.g. in response to hypoxia or secondary to a metabolic acidosis. • Alveolar hyperventilation → increased wash out CO2 → decrease in PaCO2 → increased pH. • Compensation: HCO3- will decrease after (12 -24 h).

[10] Metabolic problems always show compensation.
[13] Uncomplicated metabolic alkalosis is typically transient, because kidney can normally excrete the excess HCO3-

Respiratory Acidosis

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- **Etiology Hypoventilation of any cause:**

- CNS: Damage of the respiratory center in the brainstem Caused by: Stroke, Hemorrhage, Trauma, Tumor, Medication (Commonly sleeping pills, other: morphine, anesthetics and narcotics)
- Peripheral nervous system: Demyelinating disease Of PNS ex. Guillain-Barre syndrome[1]
- Neuromuscular junction: Myasthenia gravis
- Muscular disease: Intercostal muscle atrophy, such as:

1) Duchenne dystrophy.
2) Congenital muscle atrophy

- Chest wall: Severe scoliosis
- Bronchial tree: COPD[2] → retain CO2 → exchange gases lung defect→ leading to acute/chronic Respiratory acidosis)
- Other: Drowning, Sleep apnea and Morbid obesity.
- ALS

- **Clinical Features:**

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

- **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 2. Acute drug intoxication: narcotics, sedatives. 3. Residual neuromuscular blockade. 4. CNS disease (head trauma)	1. Chronic lung disease (COPD) 2. Neuromuscular disease 3. Extreme obesity 4. Chest wall deformity 5. Muscular e.g. Duchenne dystrophy
pH	LOW	Almost NORMAL due compensatory mechanism.
Compensation	• Immediate compensatory ↑ of HCO3. • HCO3 ↑ by 1 mEq/l for every 10 mmHg ↑ in PaCO2.	HCO3 ↑ by 3-3.5 mEq/l for every 10 mmHg ↑ in PaCO2 (Due to renal adaptation)

[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] Drowsiness or sleepiness
[4] Papilledema is optic disc swelling that is secondary to elevated intracranial pressure. Pathophysiology: Increased PaCO2 causes increased cerebral blood flow which increases CSF pressure→ Resulting in generalized CNS depression.

Respiratory Acidosis

- **Treatment:**

- Verify patency of airways.
- Give supplemental oxygen: If PaO2 is low (<60 mmHg), Oxygen is contraindicated in COPD patients (CO2 retention) as it can exacerbate symptoms.
  The explanation is that hypoxia drives breathing, so when the patient is no longer hypoxic, hypoventilation can result and hypercapnia worsens, causing a respiratory acidosis.
- Treat underlying cause.
- Intubation and mechanical ventilation might be required for: 1-Severe acidosis (PH <7).
  2-PaCO2 > 60 or inability to increase PaO2. 3-Mental deterioration. 4-Impending respiratory fatigue.

Respiratory Alkalosis

- **Etiology Hyperventilation[5] of any cause:**

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

Respiratory alkalosis can be caused by anything that causes hyperventilation (anxiety causing panic attack)

- **Clinical Features:**

- (lightheadedness, dizziness, anxiety, paresthesia, and perioral numbness)[8]
- Tetany[9], Arrhythmias, Trousseau's sign and Chvostek's sign may be positive

- **Classification:**

Acute Respiratory Alkalosis	Chronic Respiratory Alkalosis
HCO3 ↓ by 2 mEq/l for every 10 mmHg ↓ in PaCO2	HCO3 ↓ by 4-5 mEq/l for every 10 mmHg ↓ in PaCO2

- **Treatment:**

- Treat the underlying cause.
- Sometimes this does not need to be treated (e.g., in the case of pregnancy).
- Breathe into paper bag to recycle the exhaled CO2 (especially who have anxiety).

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

Metabolic Acidosis

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- **The Anion gap:**

VIDEO

- The difference between primary measured cations (Na+ and K+) and the primary measured anions (Cland 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

- **Calcification & Etiology:**

- It's mainly divided into normal Anion gap acidosis and High Anion gap acidosis
- Causes of High Anion Gap Metabolic Acidosis (MUD PILES):
  - Methanol
  - Uremia
  - DKA (check glucose)
  - Propylene glycol (not paraldehyde)
  - INH (impaired hepatic clearance of lactate)
  - Lactic acidosis
  - Ethanol/Ethylene Glycol
  - Salicylates

Problem		Causes
Increased Endogenous production	Lactic acidosis	• Low tissue perfusion (decreased oxygen delivery to tissues) • Shock states (septic, cardiogenic, hypovolemic)
	Diabetic Ketoacidosis	DM, Prolonged starvation and prolonged alcohol abuse
	Uremia	Renal failure→decreased NH4 + excretion (thus decreasing net acid) → Decreased excretion of organic anions, sulfate, and phosphate increases AG.
Increased Exogenous Intake	Oxalic acid	Ethylene glycol overdose/intoxication (manifestations include cardiopulmonary failure, calcium oxalate crystals and renal failure)
	Formic acid	Methanol overdose (manifestations include blurred vision)
	Other	Salicylates[11] (Aspirin overdose), aldehyde, Acetaminophen, alcohol

In lactic acidosis, the body switches from aerobic metabolism to anaerobic metabolism due to a decrease in O2, leading to an increase in lactic acid (which is an anion). Therefore, we have a high anion gap metabolic acidosis.
Aspirin overdose induces the respiratory center to breathe fast → increased CO2 excretion

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

Metabolic Acidosis

- Normal AG Acidosis → The low HCO3 is associated with high Cl− , so that the AG remains normal.

GI	loss of HCO3	Diarrhea, fistula in intestine or pancreas, Ureterosigmoidostomy: (colon secretes HCO3− in urine in exchange for Cl)
RENAL[12]	↓ HCO3 reabsorption.	Proximal Tubular Acidosis (RTA Type 2)
	↓ production of HCO3	Distal Tubular Acidosis (RTA Type 1)
	Carbonic anhydrase inhibition	Due to diuretics as acetazolamide.
Other	Post- hypocapnia	Respiratory alkalosis→ renal wasting of HCO3 →rapid correction of respiratory alkalosis → transient Acidosis until HCO3 regenerated
	Dilutional	Due to rapid infusion of bicarbonate - free IV fluids (0.9% saline is acidic)

★ One of the causes of metabolic acidosis with normal anion gap is Urinary diversion procedure. In this procedure the ureter is taken from kidney and attached to gut, urine is excreted with stool. With that, there is retention of chloride from urine leading to hyperchloremic acidosis

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!**

1) High Anion Gap
a. Endogenous:
- +ve Ketones: ketoacidosis because of starvation or diabetic ketoacidosis
- -ve ketones: lactic acidosis because of ischemia or hypoxia, or uremia in renal failure.

b. Exogenous: (e.g. alcohol, ethanol, methanol, paraldehyde and aspirin overdose)
2) Normal Anion Gap Diarrhea and Renal tubular acidosis

- **Clinical Features:**

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

a. Occurs with severe metabolic acidosis (blood pH < 7.2)
b. Acidosis diminishes tissue responsiveness to catecholamines. This can lead to an undesirable chain of events: poor tissue perfusion → lactic acidosis → decreased cardiac output → hypotension → further decrease in 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)

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

Metabolic Alkalosis

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- **Calcification & Etiology:**

	Saline Responsive Urine (cl-) <20	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 HCO3 such as vomiting (HCl loss), NGT drainage • Diuretic use → These decrease the ECF volume, body HCO3 content is normal, but plasma HCO3 increases due to ECF contraction. • Volume depletion: Loss of sodium and fluid leads to hypovolemia and secondary hyperaldosteronism, triggering proximal sodium bicarbonate reabsorption and additional acid secretion by the distal tubule Hypokalemia occurs due to potassium loss in the vomitus and by the kidney as the result of secondary hyperaldosteronism, and itself is a stimulus to acid secretion. • Post-hypercapnia • Villous adenoma of colon, diarrhea with high chloride content	Hypertensive: • Primary Hyperaldosteronism either • Non-mineralocorticoid, Cushing Syndrome Hypo/normo tensive: • Exogenous alkali load either IV or oral sodium bicarbonate • Bartter's syndrome & Gitelman's syndrome, Severe hypokalemia
Treatment	Treat by saline due to volume depletion	According to Etiology

- **Clinical Features:**
  There are 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.

- **In metabolic alkalosis, the body compensates by reducing the RR, which can't happen for long because it will lead to loss of consciousness**

Step 1

• Take a thorough history and physical examination, look for clues that may lead to the abnormalities in pH

Respiratory Acidosis

- Hypoventilation
- Respiratory disease

Metabolic Acidosis

- Diarrhea
- Diabetes
- Medications (laxatives)

Respiratory Alkalosis

- Hyperventilation
- Respiratory disease

Metabolic Alkalosis

- Vomiting
- Medications (diuretics)

Step 2

- **Look at the pH:**
- Determine if this is
- Normal 7.35 – 7.45 (No abnormality or mixed acidosis and alkalosis)
  For example, a very ill patient who is on diuretics(metabolic acidosis) and had diarrhea(metabolic alkalosis)
- Low <7.35 (acidemic)
- High >7.45 (alkalemic)

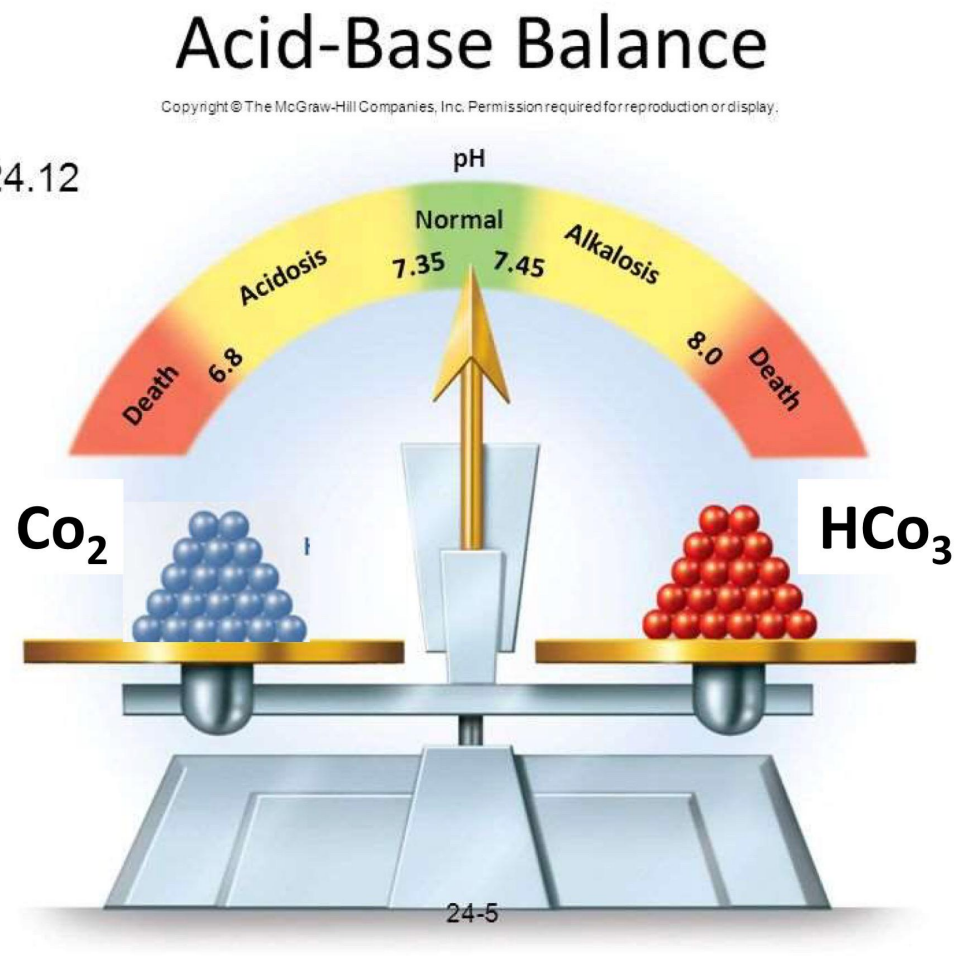


Figure 24.12

Rule of 4 for the normal values:-

pH	7.4	7.35-7.45
HCO3	24	22-26
CO2	40	35-45

Step 3 A&B

A. Determine the primary abnormality that is causing the abnormal pH

- If the pH is acidemic (<7.35), then look for:-
- Low HCO3 (Metabolic)
- High PCO2 (Respiratory)

- If the pH is alkalemic (>7.45), then look for:-
- High HCO3 (Metabolic)
- Low PCO2 (Respiratory)

B. If pH is normal, rule out mixed acidosis and alkalosis

- Look for high or low PCO2
- Look for high or low HCO3

Determine what is being mixed

- Low PCO2 suggests respiratory alkalosis
- High PCO2 suggests respiratory acidosis
- Low HCO3 suggests metabolic acidosis
- High HCO3 suggests metabolic alkalosis

Example:- If the pH was 7.15 and PCO2 was 55 and HCO3 was 10 It considered as:
Metabolic acidosis (due to Low HCO3) and Respiratory acidosis (due to High PCO2) at the same time

Step 4

After determining the primary abnormality, check for compensation

- Compensation is the mechanism by which the body adapts to either acidosis or alkalosis, it will fully correct the abnormality

- For example:-
- A patient has diabetic ketoacidosis, pH is 7.29, HCO3 is 15
- Expected PCO2 by using Winter's formula
- PCO2 = 1.5 x HCO3 + 8 (±2) = 1.5 x 15 + 8 = 30.5

So you expect the PCO2 in this patient to be in the range of 28.5– 32.5

- If the PCO2 in this patient is higher than 32.5 —> consider additional respiratory acidosis
  (because the body is supposed to compensate by hyperventilating and reducing CO2,.if the CO2 is higher than expected, this means there's respiratory acidosis on top of metabolic acidosis)
- If the PCO2 in the patient is lower than 28.5 —> consider additional respiratory alkalosis

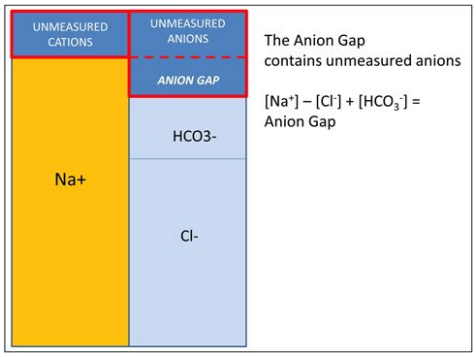
- **Compensation calculations:-**

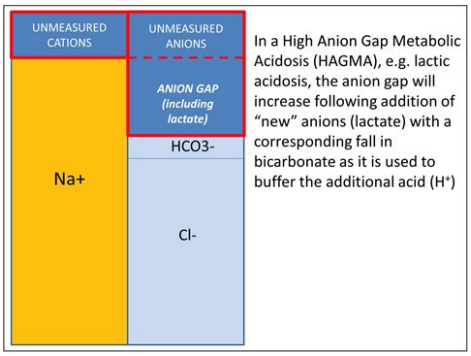
Memorize one formula for each disorder

Primary Disorder	Expected Compensation	
Metabolic Acidosis	↓ PaCO2 = 1.2 ×△ HCO3	Or
	PaCO2 = 1.5 × HCO3 + 8 ± 2	Or
	PaCO2 ~ last two digits of pH.	
Metabolic Alkalosis	↑ PaCO2 = 0.7 ×△ HCO3	
Acute Respiratory Acidosis	↑ HCO3 = 0.1 ×△ PaCO2	
Chronic Respiratory Acidosis	↑ HCO3 = 0.35 ×△ PaCO2	Also
	↓ pH = 0.003 ×△ PaCO2	
Acute Respiratory Alkalosis	↓ HCO3 = 0.2 ×△ PaCO2	
Chronic Respiratory Alkalosis	↓ HCO3 = 0.4 ×△ PaCO2	

Step 5

- **Calculate the anion gap (AG):** AG = Na – (Cl + HCO3)
- Albumin is the main unmeasured anion. To overcome the effects of the hypoalbuminemia on the AG, the corrected AG can be used which is AG + (0.25 X (40-albumin) expressed in g/L.
- when we see increase in anion gap that's mean there's additional acids like lactic acid and keto acid.





Metabolic acidosis with <u>normal</u> anion gap suggests:
Gastrointestinal losses of bicarbonate
Renal tubular acidosis
Treatment with carbonic anhydrase inhibitors
Urinary diversion procedures
Excessive administration of 0.9% saline

Causes of High Anion Gap Metabolic Acidosis (MUD PILES)

- Methanol
- Uremia
- DKA
- Propylene glycol (not paraldehyde)
- INH (impaired hepatic clearance of lactate)
- Lactic acidosis
- Ethanol/Ethylene Glycol
- Salicylates

Cases

❖ **Normal reference range:**

- pH (7.35-7.45), PCO2 (35-45 mmHg), PO2 (82-105 mmHg), HCO3 (22-26 mmol/L), AG (8- 12)
- Creatinine (40-110 umol/L), Urea (2.5-7.8 mmol/L), Na (136-145 mmol/L), K (3.5-5 mmol/L).

A 75-year-old man is admitted with septic shock. Shortly after admission, blood tests reveal the following:

- pH 7.18, PO2= 150 mmHg, PaCO2= 16 mmHg, HCO3 7 mmol/L
- Na 138 mmol/L, K 3.9 mmol/L, Cl 95 mmol/L, Urea 8.2 mmol/L, Creatinine 102 µmol/L

★ Please identify the acid base disturbance:

1.The disturbance is metabolic acidosis. Why? Because the pH is less than the normal range (7.35- 7.45) and the bicarbonate is less than 22 mmol/L.

2. Is the body properly compensating?
1.5 x 7 + 8= 18.5 ± 2. The patient's value is 16 Which almost falls within the range, that means that the metabolic acidosis is being compensated properly with respiratory alkalosis.

3. Anion gap: Na – (Cl+HCO3) = 138 – (95+7) = 36 The anion gap is higher than normal. which indicates metabolic acidosis.

★ Please indicate what is causing the acid base disturbance:

The cause is Lactic acidosis because which associated with shock.

A 68-year-old woman is being treated for congestive heart failure in the coronary care unit. After several days of treatment, the following results are returned:

- pH 7.49, PO2= 86 mmHg, PaCO2= 48.5 mmHg, HCO3 39 mmol/L
- Na 142 mmol/L, K 3.0 mmol/L, Cl 85 mmol/L, Urea 9.3 mmol/L, Creatinine 84 µmol/L

★ Please identify the acid base disturbance:

1. The disturbance is metabolic alkalosis. Why? Because the pH is higher than the normal range (7.35-7.45) and the bicarbonate is also higher than 26 mmol/L.

2. Is the body properly compensating?
Here the calculation is different: 0.7 x (39-24) = 10.5 → (10.5 + 40) = 50.5 ±2 (this is the estimated increase in PaCO2). So it should be between 48.5-52.2 mmHg which means that the metabolic alkalosis is compensating properly with respiratory acidosis.

3. Anion gap: Na – (Cl+HCO3) = 142 – (85+39) = 18
The anion gap is higher than normal here so the patient might have metabolic acidosis aswell. but the doctor said to neglect it in this case for simplicity*

★ Please indicate what is causing the acid base disturbance:

The cause of alkalosis here is the use of Diuretics (diuretics decrease blood volume so as a response to that, the kidneys increase reabsorption of sodium bicarbonate)

Cases

A 70-year-old man with chronic obstructive pulmonary disease (COPD) is admitted with increasing confusion. Shortly after admission, blood tests reveal the following:

- pH 7.21, PO 61.5 mmHg, PaCO2 83 mmHg, HCO3 34 mmol/L
- Na 140 mmol/L, K 4.7 mmol/L, Cl 94 mmol/L Urea 8.2 mmol/L, Creatinine 66 µmol/L

★ Please identify the acid base disturbance:

1.The disturbance is respiratory acidosis. Why? Because the pH is less than the normal range (7.35-7.45) and the PaCO2 is also significantly higher than 40 mmHg.

2. Is the body properly compensating?
First we have to determine whether the cause is Acute or Chronic, this mainly depends on the clinical scenario. Which means that if a patient presents with a stroke 2 or 3 hours ago and he cannot breathe so he developed Respiratory acidosis, this is Acute. A chronic scenario (like the case mentioned here) where the patient has COPD and he chronically retains CO2 which eventually lead to respiratory acidosis. 0.35 x (83-40) = 15 → (15+24=39 ±2) which means that 37-41 mmol/L is the expected HCO3. The doctor said this was a tough call because it's close to the expected range but if you want to go by the book it means that the patient's body isn't compensating properly. So, the patient has respiratory acidosis with metabolic acidosis.

3. Anion gap: Na – (Cl+HCO3) =140– (94+34) = 12 (normal anion gap)

★ Please indicate what is causing the acid base disturbance:

The cause here is CO2 retention caused by COPD

A 40-year-old man developed profuse diarrhea following antibiotic treatment of a chest infection. He is thirsty, and light headed. Shortly after admission, blood tests reveal the following:

- pH 7.25, PO 101 mmHg, PaCO2 31.5 mmHg, HCO3 17 mmol/L
- Na 134 mmol/L, K 3.4 mmol/L, Cl 104 mmol/L, Urea 9.3 mmol/L, Creatinine 102 µmol/L

★ Please identify the acid base disturbance:

1.The disturbance is metabolic acidosis. Why? Because the pH is less than the normal range (7.35-7.45) and the bicarbonate is less than 22 mmol/L.

2. Is the body properly compensating?
The calculation in this case is similar to the first case 1.5 x 17 + 8= 33.5 ± 2 mmHg (so, expected PaCO2 should be between 31.5-35.5) so the body here is compensating properly with respiratory alkalosis.

3.Anion gap: Na – (Cl+HCO3) =134– (104+17) = 13 (normal anion gap metabolic acidosis)

★ Please indicate what is causing the acid base disturbance:

The cause here is diarrhea, which is one of the causes of metabolic acidosis with a normal anion gap. Because the body retains chloride in response to the loss of bicarbonate through the GIT.

Summary

Five Steps of Acid-Base Analysis 1-5:

Step 1 & 2:
- Look for clues that may lead to the abnormalities in pH.
-Acidemia (pH <7.38) or alkalemia (pH >7.42)?

Step 3:
-Determine the primary abnormality that is causing the abnormal pH.
-If pH is normal, that doesn't rule out mixed acidosis and alkalosis.

Step 4:
After determining the primary abnormality, check for compensation: Compensation is the mechanism by which the body adapts to either acidosis or alkalosis.

Step 5:
- Calculate the anion gap (AG):
AG = Na – (Cl + HCO3).
- **Causes of High Anion Gap Metabolic Acidosis (MUD PILES):**
  - **M**ethanol.
  - **U**remia.
  - **D**KA.
  - **P**ropylene glycol (not paraldehyde).
  - **I**NH (impaired hepatic clearance of lactate).
  - **L**actic acidosis.
  - **E**thanol/Ethylene Glycol.
  - **S**alicylates.

Summary

Acid-Base Disorders:

Metabolic Acidosis:

- loss of [HCO3] or addition of [H+].
- Causes:
  - Shock states.
  - Renal failure.
  - Methanol overdose.
  - DM.
- **Expected Compensation:**
  1) ↓ PaCO2 = 1.2 ×△ HCO3.
  2) PaCO2 = 1.5 × HCO3 + 8 ± 2.
  3) PaCO2 ~ last two digits of pH.

Metabolic alkalosis:

- loss of [H+] or addition of [HCO3]
- Causes:
  - Primary Hyperaldosteronism.
  - Volume depletion.
  - Diuretic use.
  - Gastric loss of H+.
- **Expected Compensation:**
  1) ↑ PaCO2 = 0.7 ×△ HCO3.

Respiratory Acidosis:

- Increase in pCO2.
- Causes:
  - **Acute:**
  1) Respiratory: airway obstruction, severe pneumonia, chest trauma/pneumothorax.
  2) Acute drug intoxication: narcotics, sedatives.
  - **Chronic:**
  1) COPD.
  2) Neuromuscular disease.
- **Expected Compensation:**
  1) **Acute:** ↑ HCO3 = 0.1 ×△ PaCO2.
  2) **Chronic:** ↑ HCO3 = 0.35 ×△ PaCO2
     Or pH = 0.003 ×△ PaCO2.

Respiratory Alkalosis:

- Decrease in pCO2.
- Causes:
  - Pulmonary embolism.
  - Severe congestive heart failure.
  - Thyrotoxicosis.
  - Anxiety.
- **Expected Compensation:**
  1) **Acute:** ↓ HCO3 = 0.2 ×△ PaCO2.
  2) **Chronic:** ↓ HCO3 = 0.4 ×△ PaCO2

Questions

Q1: A 73 years old woman with arthritis presents with confusion. Neurologic examination is nonfocal, laboratory data include Na: 140 mEq/L K: 3 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 disorders.

Q2: A 20-year old student suffered a panic attack while awaiting an exam. Her ABGs showed: pH = 7.6 , PCO2 = 24 mmHg , HCO3 = 23 mEq/L.

A) Uncompensated Respiratory alkalosis.
B) Compensated Respiratory alkalosis.
C) Metabolic acidosis.
D) Respiratory acidosis.

Q3: A person was admitted in a coma. Analysis of the arterial blood gave the following values: PCO2 16 mm Hg, HCO3- 5 mmol/l and pH 7.1. What is the underlying acid-base disorder?

A) Metabolic Acidosis.
B) Metabolic Alkalosis.
C) Respiratory Acidosis.
D) Respiratory Alkalosis.

Q4: In a man undergoing surgery, it was necessary to aspirate the contents of the upper gastrointestinal tract. After surgery, the following values were obtained from an arterial blood sample: pH 7.55, PCO2 52 mm Hg and HCO3- 40 mmol/l. What is the underlying disorder?

A) Metabolic Acidosis.
B) Metabolic Alkalosis.
C) Respiratory Acidosis.
D) Respiratory Alkalosis.

Answers:

1. C
2. A
3. A
4. B

Questions

Q5: A young woman is found comatose, having taken an unknown number of sleeping pills an unknown time before. An arterial blood sample yields the following values: pH – 6.90, HCO3- 13 meq/liter, PCO2 68 mmHg. This patient's acid-base status is most accurately described as:

A) Uncompensated metabolic acidosis.
B) Uncompensated respiratory acidosis.
C) Simultaneous respiratory and metabolic acidosis.
D) Respiratory acidosis with partial renal compensation.

Answer: C

Q6: A 22 year-old woman presents with 4 hours of numbness in both hands typical of previous episodes of anxiety. ABG: pH 7.48, pCO2 30 mmHg, pO2 86 mmHg Metabolic panel: Na 140, Cl 110, HCO3 22.

Answer:

Step 1: Alkalosis.

Step 2: Respiratory.

Step 3:
Acute. Drop in the pCO2 by 10 corresponds to a drop in the HCO3 by 2 if acute and 5 if chronic. 24-22 = 2 and therefore, as would be expected by the clinical history, an acute disorder is diagnosed.

Step 4: AG 140-(110+22) = 8

Step 5: XX

This question was taken from Toxicology Team 436