100 ABG Practice Questions
With Step-by-Step Solutions

Master Arterial Blood Gas Interpretation
Respiratory Acidosis • Respiratory Alkalosis
Metabolic Acidosis • Metabolic Alkalosis • Mixed Disorders

Clinical Excellence Series
Nursing & Allied Health Education

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Introduction & How to Use This Ebook

Arterial Blood Gas (ABG) interpretation is one of the most essential clinical skills for nurses, respiratory therapists, intensivists, and medical students. A single ABG result can reveal life-threatening respiratory failure, dangerous electrolyte imbalances, or guide critical ventilator management — all in seconds.

This ebook provides 100 carefully crafted clinical scenarios with detailed, step-by-step solutions. Each question includes a patient scenario, a full ABG panel, and a systematic analysis walking through pH assessment, primary disorder identification, compensation verification, and clinical management.

How to Use This Book: For maximum learning, cover the answer section and attempt your own interpretation before reading the solution. Use the 5-Step Method below as your consistent framework.

The 5-Step ABG Interpretation Method

Step 1: Assess the pH — Is it acidosis (<7.35), normal (7.35–7.45), or alkalosis (>7.45)?

Step 2: Identify the primary disorder — Is PaCO2 or HCO3 the culprit moving pH in the same direction?

Step 3: Check for compensation — Is the other parameter changing appropriately? Use Winter's formula or compensation rules.

Step 4: Calculate Anion Gap (if metabolic acidosis) — AG = Na – (Cl + HCO3). Normal = 8–12 mEq/L.

Step 5: Assess oxygenation — PaO2 (normal ≥80 mmHg on room air), SaO2, A-a gradient.

ABG Normal Values at a Glance

Parameter	Normal Range	Acidosis	Alkalosis
pH	7.35 – 7.45	< 7.35	> 7.45
PaCO2	35 – 45 mmHg	> 45	< 35
HCO3–	22 – 26 mEq/L	< 22	> 26
PaO2	80 – 100 mmHg	—	—
SaO2	95 – 100%	—	—
BE	-2 to +2	< -2	> +2

Section 1: Respiratory Acidosis

Questions 1–20 | CO2 Retention & Hypoventilation

Question 1

A 68-year-old COPD patient presents with acute dyspnea, confusion, and cyanosis. He is breathing shallowly at 8 breaths/min.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.28	58 mmHg	28 mEq/L	52 mmHg	88%

What acid-base disturbance is present, and is compensation occurring?

✔ ANSWER: Acute Respiratory Acidosis with partial metabolic compensation

Step 1 – Assess pH

pH 7.28 is < 7.35 → ACIDOSIS.

Step 2 – Identify primary disorder

PaCO2 58 mmHg is > 45 → CO2 is retained → RESPIRATORY ACIDOSIS.

Step 3 – Check HCO3–

HCO3– 28 mEq/L is mildly elevated (normal 22–26). Kidneys are retaining bicarbonate to compensate.

Step 4 – Determine compensation

Expected HCO3– rise = 1 mEq/L per 10 mmHg rise in PaCO2. Rise in CO2 = 18 → expected HCO3 = 24+1.8 = 25.8. Actual 28 → slightly over-compensated but consistent with early chronic compensation.

Step 5 – Clinical correlation

Hypoventilation from COPD exacerbation. Supplemental O2, bronchodilators, possible NIV.

Question 2

A 35-year-old heroin overdose patient is unresponsive, GCS 6, respiratory rate 4/min.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.18	72 mmHg	24 mEq/L	44 mmHg	79%

Interpret the ABG. What is the most urgent intervention?

✔ ANSWER: Acute Respiratory Acidosis — no compensation yet

Step 1 – pH

7.18 → severe acidosis.

Step 2 – PaCO2

72 mmHg → markedly elevated → primary respiratory acidosis.

Step 3 – HCO3–

24 mEq/L (normal) → kidneys have NOT had time to compensate (acute).

Step 4 – Why no compensation?

Metabolic compensation takes 12–24 hours minimum.

Step 5 – Intervention

Naloxone (Narcan) IV immediately + bag-mask ventilation + prepare for intubation.

Question 3

Post-op day 1 patient after abdominal surgery. On PCA opioids. Nurse finds patient somnolent, RR 6.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.25	65 mmHg	25 mEq/L	58 mmHg	91%

What does this ABG represent?

✔ ANSWER: Acute Respiratory Acidosis (opioid-induced hypoventilation)

Step 1 – pH

7.25 → acidosis.

Step 2 – PaCO2

65 mmHg → elevated → respiratory cause.

Step 3 – HCO3–

25 mEq/L → normal → acute event, no renal compensation.

Step 4 – Conclusion

Acute respiratory acidosis from opioid-suppressed respiratory drive.

Step 5 – Action

Stimulate patient, reduce PCA dose, consider naloxone, increase monitoring frequency.

Question 4

A morbidly obese patient (BMI 52) presents sleepy in the morning, spouse reports loud snoring and apnea episodes.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.32	55 mmHg	30 mEq/L	61 mmHg	92%

What acid-base disorder is this? What syndrome do you suspect?

✔ ANSWER: Chronic Respiratory Acidosis — Obesity Hypoventilation Syndrome

Step 1 – pH

7.32 → acidosis, but closer to normal than acute cases → chronic.

Step 2 – PaCO2

55 mmHg → elevated → CO2 retention.

Step 3 – HCO3–

30 mEq/L → elevated → kidneys have compensated (chronic = HCO3 rises 3.5/10 mmHg CO2).

Step 4 – Expected HCO3–

Rise in CO2 = 15 → 3.5×1.5 = 5.25 → 24+5 = 29 ≈ 30 ✓

Step 5 – Suspect

Obesity Hypoventilation Syndrome (Pickwickian Syndrome). Refer for sleep study, CPAP/BiPAP.

Question 5

A 72-year-old with severe kyphoscoliosis and restrictive lung disease presents with worsening dyspnea.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.34	50 mmHg	27 mEq/L	65 mmHg	93%

What acid-base imbalance is present?

✔ ANSWER: Chronic Respiratory Acidosis — fully compensated

Step 1 – pH

7.34 → just within acidosis range but nearly compensated.

Step 2 – PaCO2

50 mmHg → elevated → primary respiratory.

Step 3 – HCO3–

27 mEq/L → elevated = renal compensation.

Step 4 – Expected

Rise in CO2 = 10 → HCO3 rise = 3.5 → expected 27.5 ≈ 27 ✓ = fully compensated.

Step 5 – Clinical

Restrictive pattern from chest wall deformity. Long-standing CO2 retention.

Question 6

A 58-year-old with CHF exacerbation has crackles to mid-lungs, O2 sat 85% on room air, RR 28.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.30	50 mmHg	24 mEq/L	48 mmHg	85%

Interpret the ABG and explain the low PaO2.

✔ ANSWER: Acute Respiratory Acidosis with hypoxemia (V/Q mismatch)

Step 1 – pH

7.30 → acidosis.

Step 2 – PaCO2

50 mmHg → elevated → respiratory acidosis.

Step 3 – HCO3–

24 mEq/L → normal → acute process.

Step 4 – PaO2

48 mmHg → severe hypoxemia. Normal ≥ 80 mmHg on room air.

Step 5 – Mechanism

Pulmonary edema causes V/Q mismatch → impaired gas exchange → hypoxemia AND CO2 retention.

Step 6 – Action

High-flow O2, diuretics, possible BiPAP.

Question 7

A 44-year-old with Guillain-Barré syndrome has declining NIF (negative inspiratory force). RR 22, accessory muscle use.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.29	58 mmHg	26 mEq/L	60 mmHg	91%

What does this ABG indicate? What is your primary concern?

✔ ANSWER: Acute Respiratory Acidosis — impending respiratory failure

Step 1 – pH

7.29 → acidosis.

Step 2 – PaCO2

58 → respiratory cause; neuromuscular weakness = cannot exhale CO2.

Step 3 – HCO3–

26 → normal → acute event.

Step 4 – Primary concern

Neuromuscular paralysis will worsen → complete respiratory failure.

Step 5 – Action

Prepare for intubation. NIF < -20 cmH2O or FVC < 1L = intubation threshold.

Question 8

A 19-year-old with status asthmaticus, wheeze throughout, using accessory muscles. Previous ABG 2 hours ago showed pH 7.48.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.38	44 mmHg	26 mEq/L	68 mmHg	94%

This ABG appears normal. Is that reassuring? Explain.

✔ ANSWER: WARNING: 'Pseudo-normal' ABG — critical sign in status asthmaticus

Step 1 – pH

7.38 → normal

Step 2 – PaCO2

44 → normal

Step 3 – Why alarming?

Early asthma = respiratory alkalosis (hyperventilation). A NORMAL PaCO2 in an asthmatic who was previously alkalotic means they are TIRING — cannot hyperventilate anymore.

Step 4 – Implication

CO2 normalizing = CO2 beginning to RISE = impending respiratory failure.

Step 5 – Action

Escalate treatment immediately. Prepare for intubation. Do NOT be reassured.

Question 9

A 77-year-old nursing home resident with aspiration pneumonia. CXR shows right lower lobe infiltrate. RR 30.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.26	60 mmHg	26 mEq/L	50 mmHg	87%

Classify the acid-base disorder and identify the hypoxemia severity.

✔ ANSWER: Acute Respiratory Acidosis with severe hypoxemia

Step 1 – pH

7.26 → acidosis

Step 2 – PaCO2

60 → elevated → respiratory cause

Step 3 – HCO3–

26 → normal → acute

Step 4 – PaO2

50 mmHg → severe hypoxemia (50-59 = moderate; <50 = severe — borderline here)

Step 5 – A-a gradient

If on room air: A-a = [150 - (PaCO2/0.8)] - PaO2 = [150-75] - 50 = 25 (elevated → V/Q mismatch from consolidation)

Step 6 – Management

Antibiotics, supplemental O2, consider NIV or intubation.

Question 10

A 62-year-old with end-stage COPD on home O2. Admitted for productive cough. Currently on 2L O2.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.30	62 mmHg	32 mEq/L	55 mmHg	89%

What does the elevated HCO3 tell you? What type of respiratory acidosis is this?

✔ ANSWER: Acute-on-Chronic Respiratory Acidosis

Step 1 – pH

7.30 → acidosis

Step 2 – PaCO2

62 → elevated

Step 3 – HCO3–

32 → elevated → pre-existing chronic compensation

Step 4 – Interpretation

Elevated HCO3 shows chronic compensation (COPD baseline). But pH still acidotic → acute worsening on top of chronic = Acute-on-Chronic Respiratory Acidosis.

Step 5 – Key teaching

If chronic alone, pH would be near normal. The drop in pH signals the NEW acute component.

Step 6 – Management

Treat exacerbation: steroids, bronchodilators, antibiotics, controlled O2 therapy.

Question 11

A 50-year-old patient is sedated on mechanical ventilator. Vent settings: RR 10, TV 500mL.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.28	58 mmHg	25 mEq/L	88 mmHg	97%

What does this ABG indicate about ventilator settings?

✔ ANSWER: Respiratory Acidosis due to inadequate ventilation

Step 1 – pH

7.28 → acidosis

Step 2 – PaCO2

58 → elevated → hypoventilation

Step 3 – HCO3–

25 → normal (HCO3 and PaO2 fine on vent)

Step 4 – Conclusion

Ventilator is not removing enough CO2. Minute ventilation too low.

Step 5 – Fix

Increase RR or tidal volume (or both). Target PaCO2 35-45 mmHg.

Question 12

A 25-year-old with a traumatic C3 spinal cord injury is on a ventilator but desatting after vent disconnection attempt.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.22	70 mmHg	25 mEq/L	50 mmHg	86%

Classify the ABG. Why can't this patient breathe independently?

✔ ANSWER: Acute Respiratory Acidosis — high SCI with total ventilator dependence

Step 1 – pH

7.22 → severe acidosis

Step 2 – PaCO2

70 → severe CO2 retention

Step 3 – Reason

C3 SCI = loss of phrenic nerve function (C3-C5 innervate diaphragm). Cannot breathe without vent.

Step 4 – Mnemonic

'C3, 4, 5 keeps the diaphragm alive' — injury above C5 = vent-dependent.

Step 5 – Management

Reinstate ventilation immediately. No spontaneous breathing trials.

Question 13

A 3-year-old child swallowed a toy and is presenting with stridor, retractions, and RR 40.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.24	62 mmHg	23 mEq/L	54 mmHg	86%

What is this ABG indicating? What is the priority action?

✔ ANSWER: Acute Respiratory Acidosis — upper airway obstruction

Step 1 – pH

7.24 → acidosis

Step 2 – PaCO2

62 → elevated → cannot ventilate

Step 3 – HCO3–

23 → normal → acute

Step 4 – Priority

Airway, airway, airway. Foreign body removal (back blows/Heimlich for children). Prepare for emergency airway.

Step 5 – Note

Pediatric normal PaCO2 is 35-45 — same interpretation applies.

Question 14

A 30-year-old with myasthenic crisis, diplopia, ptosis, and difficulty swallowing. RR 28, SpO2 92%.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.27	60 mmHg	26 mEq/L	60 mmHg	92%

What ABG pattern do you expect and why?

✔ ANSWER: Acute Respiratory Acidosis — neuromuscular failure (myasthenic crisis)

Step 1 – pH

7.27 → acidosis

Step 2 – PaCO2

60 → elevated → can't clear CO2 (respiratory muscle weakness)

Step 3 – HCO3–

26 → normal → acute event

Step 4 – Pathophysiology

Myasthenia gravis → antibodies against acetylcholine receptors → respiratory muscle weakness → hypoventilation → CO2 rises.

Step 5 – Management

ICU admission, plasmapheresis or IVIG, pyridostigmine, consider intubation.

Question 15

A 68-year-old post-cardiac arrest patient. ROSC achieved. Currently intubated with RR 18. ABG drawn 10 min post-ROSC.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.18	35 mmHg	13 mEq/L	88 mmHg	97%

Is this respiratory or metabolic? Explain.

✔ ANSWER: Metabolic Acidosis (lactic acidosis post-arrest) — NOT respiratory

Step 1 – pH

7.18 → severe acidosis

Step 2 – PaCO2

35 → NORMAL → respiratory is adequate on vent

Step 3 – HCO3–

13 → severely LOW → metabolic acidosis

Step 4 – Conclusion

Primary metabolic acidosis. The respiratory is NORMAL — do not change vent settings.

Step 5 – Cause

Ischemia during arrest → anaerobic metabolism → lactic acid production.

Step 6 – Management

Treat underlying ischemia, ensure adequate perfusion; bicarbonate rarely used unless pH < 7.1 + hemodynamic instability.

Question 16

A 45-year-old with severe sleep apnea, no CPAP compliance. Morning ABG drawn.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.33	54 mmHg	29 mEq/L	64 mmHg	93%

Classify the ABG disturbance.

✔ ANSWER: Chronic Respiratory Acidosis — partially compensated

Step 1 – pH

7.33 → acidosis

Step 2 – PaCO2

54 → elevated

Step 3 – HCO3–

29 → elevated (renal retention) → chronic compensation

Step 4 – Expected HCO3

Rise PaCO2 = 14 → 14/10 × 3.5 = 4.9 → expected HCO3 = 24+5 = 29 ✓ Full chronic compensation.

Step 5 – But pH

7.33 still acidotic → not fully normalized → partially compensated.

Step 6 – Teaching point

Chronic respiratory acidosis: HCO3 rises ~3.5 per 10 mmHg rise in CO2.

Question 17

A 55-year-old smoker with a large right-sided pleural effusion. Dull percussion, absent breath sounds R base.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.31	55 mmHg	27 mEq/L	58 mmHg	90%

What acid-base disorder is this? What is the mechanism?

✔ ANSWER: Acute Respiratory Acidosis — large pleural effusion

Step 1 – pH

7.31 → acidosis

Step 2 – PaCO2

55 → elevated → respiratory

Step 3 – HCO3–

27 → slight elevation → early/partial compensation

Step 4 – Mechanism

Large pleural effusion compresses lung → reduces functional lung volume → impairs ventilation → CO2 retention.

Step 5 – Management

Thoracentesis to drain effusion → improved ventilation.

Question 18

A 78-year-old with bilateral pneumonia, RR 34, using accessory muscles.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.20	68 mmHg	26 mEq/L	42 mmHg	80%

Classify the severity and urgency.

✔ ANSWER: Severe Acute Respiratory Acidosis — critical, intubation likely needed

Step 1 – pH

7.20 → severe acidosis

Step 2 – PaCO2

68 → markedly elevated

Step 3 – HCO3–

26 → normal → acute

Step 4 – PaO2

42 → severe hypoxemia

Step 5 – Severity classification

pH < 7.20 = severe acidosis; PaO2 < 50 = critical hypoxemia.

Step 6 – Urgency

Immediate escalation of care: High-flow O2, NIV trial (CPAP/BiPAP). If failing → intubation. Alert ICU.

Question 19

A 29-year-old with flail chest after MVA. Paradoxical chest wall movement noted.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.26	60 mmHg	25 mEq/L	56 mmHg	89%

Interpret the ABG and explain the paradoxical breathing.

✔ ANSWER: Acute Respiratory Acidosis — flail chest with paradoxical breathing

Step 1 – pH

7.26 → acidosis

Step 2 – PaCO2

60 → hypoventilation

Step 3 – HCO3–

25 → normal → acute

Step 4 – Mechanism

Multiple adjacent rib fractures create unstable chest wall segment. During inspiration, segment moves IN (paradoxically) → effective tidal volume drastically reduced → CO2 rises.

Step 5 – Management

Positive pressure ventilation (intubation), adequate analgesia, possible surgical stabilization.

Question 20

A 40-year-old with botulism after eating home-canned food. Progressive descending weakness noted.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.28	57 mmHg	25 mEq/L	62 mmHg	91%

What is the ABG pattern and mechanism?

✔ ANSWER: Acute Respiratory Acidosis — botulism-induced neuromuscular blockade

Step 1 – pH

7.28 → acidosis

Step 2 – PaCO2

57 → elevated → hypoventilation

Step 3 – HCO3–

25 → normal → acute process

Step 4 – Mechanism

Botulinum toxin blocks acetylcholine release at NMJ → flaccid paralysis → respiratory muscles fail → CO2 retention.

Step 5 – Key feature

Descending paralysis: cranial nerves first (diplopia, dysarthria) → respiratory muscles.

Step 6 – Management

Antitoxin, supportive care, early intubation, ICU monitoring.

Section 2: Respiratory Alkalosis

Questions 21–35 | CO2 Loss & Hyperventilation

Question 21

A 22-year-old hyperventilating after a panic attack. Tingling in hands and feet, carpopedal spasm.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.55	25 mmHg	22 mEq/L	98 mmHg	99%

Identify the ABG disorder. Why does carpopedal spasm occur?

✔ ANSWER: Acute Respiratory Alkalosis — hyperventilation/panic attack

Step 1 – pH

7.55 → alkalosis

Step 2 – PaCO2

25 → low → CO2 blown off → respiratory alkalosis

Step 3 – HCO3–

22 → normal → acute event (no renal compensation yet)

Step 4 – Carpopedal spasm mechanism

Low PaCO2 → respiratory alkalosis → calcium binds more to albumin → decreased ionized calcium → tetany.

Step 5 – Management

Reassurance, rebreathing techniques, address underlying anxiety.

Question 22

A 32-year-old at high altitude (14,000 feet) for 2 days. Feeling well, no mountain sickness.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.47	30 mmHg	20 mEq/L	55 mmHg	88%

What does this ABG show? Is this normal at altitude?

✔ ANSWER: Chronic Respiratory Alkalosis — normal altitude acclimatization

Step 1 – pH

7.47 → mild alkalosis

Step 2 – PaCO2

30 → low → deliberate hyperventilation to compensate for low O2

Step 3 – HCO3–

20 → low → kidneys excreted HCO3 to compensate for alkalosis (metabolic compensation)

Step 4 – Altitude physiology

Low atmospheric O2 → hypoxic drive → hyperventilation → respiratory alkalosis → kidneys lower HCO3.

Step 5 – Conclusion

Expected physiologic response to altitude. Not pathologic.

Question 23

A 28-year-old septic patient with fever 39.8°C, tachycardia, hypotension, RR 28.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.50	28 mmHg	21 mEq/L	75 mmHg	96%

Interpret the ABG in the context of sepsis.

✔ ANSWER: Acute Respiratory Alkalosis — early sepsis

Step 1 – pH

7.50 → alkalosis

Step 2 – PaCO2

28 → hyperventilation from fever/sepsis stimulating respiratory center

Step 3 – HCO3–

21 → slightly low (early renal compensation or mild lactic acid)

Step 4 – Sepsis ABG pattern

Early sepsis = respiratory alkalosis (hyperventilation). Late sepsis = metabolic acidosis (lactic acid).

Step 5 – Clinical significance

Respiratory alkalosis in a febrile patient = early warning of sepsis. Reassess often.

Question 24

A 7-month-pregnant woman presents for routine prenatal care. She feels well.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.44	30 mmHg	20 mEq/L	98 mmHg	99%

Is this ABG normal for pregnancy?

✔ ANSWER: Normal ABG in pregnancy — physiologic respiratory alkalosis

Step 1 – pH

7.44 → high-normal

Step 2 – PaCO2

30 → low (normal in pregnancy: 28-32)

Step 3 – HCO3–

20 → low (renal compensates)

Step 4 – Why?

Progesterone stimulates respiratory center → hyperventilation → intentional respiratory alkalosis.

Step 5 – Normal pregnancy ABG

pH 7.40-7.45, PaCO2 28-32, HCO3 18-22.

Step 6 – Implication

A 'normal' PaCO2 of 40 in a pregnant woman may actually represent CO2 retention!

Question 25

A 30-year-old mechanical ventilation patient. Settings: RR 20, TV 600 mL.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.52	28 mmHg	22 mEq/L	95 mmHg	99%

What is this? How do you correct it?

✔ ANSWER: Iatrogenic Respiratory Alkalosis — over-ventilation

Step 1 – pH

7.52 → alkalosis

Step 2 – PaCO2

28 → too low → too much CO2 being eliminated

Step 3 – HCO3–

22 → normal

Step 4 – Cause

Vent rate or tidal volume too high → excessive minute ventilation.

Step 5 – Correction

Decrease RR or tidal volume. Target PaCO2 35-45 mmHg. Reassess ABG after changes.

Question 26

A 45-year-old with hepatic encephalopathy and liver cirrhosis. Mental status changes, asterixis.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.49	30 mmHg	22 mEq/L	88 mmHg	97%

What ABG pattern is seen in liver failure?

✔ ANSWER: Chronic Respiratory Alkalosis — hyperammonemia stimulating respiratory center

Step 1 – pH

7.49 → alkalosis

Step 2 – PaCO2

30 → low → hyperventilation

Step 3 – HCO3–

22 → slightly low → early renal compensation

Step 4 – Mechanism

Liver fails to detoxify ammonia → ammonia crosses blood-brain barrier → directly stimulates central respiratory center → hyperventilation.

Step 5 – Clinical note

Respiratory alkalosis in cirrhosis is common and not always treated directly; focus on treating liver failure.

Question 27

A 60-year-old post-op patient with fever 38.5°C, pain 8/10, RR 26.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.50	27 mmHg	21 mEq/L	94 mmHg	98%

What is causing the respiratory alkalosis?

✔ ANSWER: Acute Respiratory Alkalosis — pain and fever-driven hyperventilation

Step 1 – pH

7.50 → alkalosis

Step 2 – PaCO2

27 → hyperventilation from pain/fever

Step 3 – HCO3–

21 → normal/slightly low

Step 4 – Triggers

Pain and fever are both direct stimulators of the respiratory center.

Step 5 – Management

Treat the underlying cause: adequate analgesia, antipyretics. ABG will normalize.

Question 28

A 34-year-old with pulmonary embolism confirmed on CT-PA. RR 32, pleuritic chest pain.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.49	29 mmHg	21 mEq/L	60 mmHg	92%

Explain why respiratory alkalosis occurs in PE.

✔ ANSWER: Acute Respiratory Alkalosis with hypoxemia — pulmonary embolism

Step 1 – pH

7.49 → alkalosis

Step 2 – PaCO2

29 → hyperventilation

Step 3 – PaO2

60 → hypoxemia → drives respiratory center

Step 4 – Mechanism

PE causes dead space (ventilated but not perfused) → hypoxemia → reflexive hyperventilation to maintain O2 → CO2 blown off.

Step 5 – Classic PE triad

Hypoxemia + respiratory alkalosis + hypocapnia (low PaCO2)

Step 6 – Note

Normal ABG does NOT rule out PE.

Question 29

A 19-year-old salicylate (aspirin) overdose patient. Tinnitus, diaphoresis, vomiting.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.52	22 mmHg	17 mEq/L	95 mmHg	98%

What complex acid-base disorder is present in salicylate toxicity?

✔ ANSWER: Mixed: Respiratory Alkalosis AND Metabolic Acidosis (classic salicylate toxicity)

Step 1 – pH

7.52 → alkalosis

Step 2 – PaCO2

22 → very low → primary respiratory alkalosis (salicylate stimulates CNS respiratory center)

Step 3 – HCO3–

17 → low → also metabolic acidosis (salicylic acid accumulation)

Step 4 – Classic pattern

Salicylate toxicity = BOTH respiratory alkalosis AND metabolic acidosis simultaneously.

Step 5 – AG

Calculate anion gap: if elevated → high-AG metabolic acidosis from salicylate.

Step 6 – Management

Urinary alkalinization with NaHCO3, hemodialysis if severe.

Question 30

A 55-year-old anxious patient in the ER with chest pain. ECG normal. RR 28, SpO2 99%.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.53	24 mmHg	20 mEq/L	99 mmHg	99%

Interpret the ABG. How would you manage this?

✔ ANSWER: Acute Respiratory Alkalosis — anxiety/hyperventilation syndrome

Step 1 – pH

7.53 → alkalosis

Step 2 – PaCO2

24 → hyperventilation

Step 3 – HCO3–

20 → normal/slightly low → acute event

Step 4 – Diagnosis

Hyperventilation syndrome — a diagnosis of exclusion after cardiac and PE workup is negative.

Step 5 – Management

Calm reassurance, controlled breathing exercises, address anxiety source. Paper bag rebreathing is controversial — use with caution.

Question 31

A 26-year-old with anemia (Hgb 5 g/dL) from GI bleeding. Pale, tachycardic, tachypneic.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.50	28 mmHg	21 mEq/L	86 mmHg	96%

Why does severe anemia cause respiratory alkalosis?

✔ ANSWER: Acute Respiratory Alkalosis — compensatory response to tissue hypoxia from anemia

Step 1 – Mechanism

Low Hgb → reduced O2-carrying capacity → tissue hypoxia despite adequate PaO2 → chemoreceptors sense hypoxia → hyperventilation.

Step 2 – pH

7.50 → alkalosis

Step 3 – PaCO2

28 → reduced

Step 4 – Key teaching

PaO2 can be normal in anemia — it's oxygen CONTENT (CaO2 = Hgb × 1.34 × SaO2) that is low.

Step 5 – Management

Blood transfusion, stop bleeding source.

Question 32

A 65-year-old with brain tumor and elevated ICP. GCS declining. Cushing's triad beginning.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.48	30 mmHg	22 mEq/L	85 mmHg	97%

Why might respiratory alkalosis be present with raised ICP?

✔ ANSWER: Respiratory Alkalosis — central neurogenic hyperventilation

Step 1 – Mechanism

Mass lesion/raised ICP stimulates brain stem respiratory centers → central neurogenic hyperventilation.

Step 2 – pH

7.48 → alkalosis

Step 3 – PaCO2

30 → low

Step 4 – Therapeutic note

Controlled mild hyperventilation (PaCO2 30-35) is used SHORT-TERM to reduce ICP (CO2 vasoconstriction). But chronic hyperventilation harmful.

Step 5 – Management

Treat raised ICP: osmotherapy (mannitol), elevation, neurosurgical consult.

Question 33

A 40-year-old patient receiving total parenteral nutrition (TPN). No other complaints.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.47	32 mmHg	23 mEq/L	95 mmHg	99%

Is this ABG normal? What is the most likely cause?

✔ ANSWER: Mild Respiratory Alkalosis — may be iatrogenic or positional

Step 1 – pH

7.47 → mild alkalosis

Step 2 – PaCO2

32 → slightly low

Step 3 – HCO3–

23 → normal

Step 4 – Causes in TPN

High dextrose loading → increased CO2 production → hyperventilation; also pain, anxiety from line.

Step 5 – Management

Review TPN formula (fat:carb ratio), reassess if worsening.

Question 34

A 32-year-old recently returned from trekking in the Himalayas. Presents with fatigue. ABG done.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.45	33 mmHg	22 mEq/L	80 mmHg	97%

What residual ABG pattern might persist post-altitude?

✔ ANSWER: Resolving Respiratory Alkalosis — post-altitude acclimatization

Step 1 – pH

7.45 → high normal

Step 2 – PaCO2

33 → still slightly low → lungs still hyperventilating or not fully normalized

Step 3 – HCO3–

22 → slightly low → renal HCO3 excretion hasn't fully reversed

Step 4 – Teaching

Acclimatization changes normalize over days to weeks after return to sea level.

Step 5 – Conclusion

Expected residual changes; clinically benign.

Question 35

A 22-year-old sprinting. ABG drawn immediately post-exercise.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.46	34 mmHg	23 mEq/L	105 mmHg	100%

Is this pathologic?

✔ ANSWER: Physiologic Respiratory Alkalosis — normal exercise response

Step 1 – pH

7.46 → mild alkalosis

Step 2 – PaCO2

34 → slightly low → increased ventilation during exercise

Step 3 – PaO2

105 → supranormal — possible mild hyperoxia

Step 4 – Conclusion

Entirely physiologic. Exercise drives ventilation up. No treatment needed.

Step 5 – Teaching point

Always interpret ABGs in clinical context. 'Abnormal' values can be normal physiologically.

Section 3: Metabolic Acidosis

Questions 36–60 | Bicarbonate Depletion & Acid Gain

Question 36

A 24-year-old Type 1 diabetic presents with vomiting, abdominal pain, fruity breath, RR 28 (Kussmaul), glucose 480 mg/dL.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.10	18 mmHg	6 mEq/L	95 mmHg	98%

Identify the acid-base disorder and calculate the anion gap.

✔ ANSWER: Severe Metabolic Acidosis — Diabetic Ketoacidosis (DKA), High Anion Gap

Step 1 – pH

7.10 → severe acidosis

Step 2 – PaCO2

18 → low (respiratory compensation = Kussmaul breathing)

Step 3 – HCO3–

6 → severely low → primary metabolic acidosis

Step 4 – Anion Gap

AG = Na – (Cl + HCO3) = 138 – (100 + 6) = 32 (elevated; normal 8-12)

Step 5 – Delta Ratio

(AG–12) / (24–HCO3) = 20/18 = 1.1 → pure high-AG metabolic acidosis

Step 6 – Compensation check

Expected PaCO2 = 1.5×HCO3 + 8 ± 2 = 1.5×6+8 = 17 ± 2 ✓ Appropriate compensation

Step 7 – Management

IV fluids, insulin infusion, electrolyte replacement (K+), monitor closely.

Question 37

A 66-year-old septic patient in ICU. Lactic acid 8 mmol/L.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.12	20 mmHg	7 mEq/L	78 mmHg	95%

What is causing the metabolic acidosis? Calculate and interpret AG.

✔ ANSWER: Severe Metabolic Acidosis — Lactic Acidosis (Type A), High Anion Gap

Step 1 – pH

7.12 → severe acidosis

Step 2 – PaCO2

20 → respiratory compensation

Step 3 – HCO3–

7 → severely low

Step 4 – AG

Na(140) - (Cl 98 + HCO3 7) = 35 → severely elevated AG

Step 5 – Type A lactic acidosis

Tissue hypoperfusion/hypoxia → anaerobic metabolism → lactate production

Step 6 – Lactate

Normal < 2 mmol/L; > 4 = severe; > 8 = life-threatening

Step 7 – Management

Treat sepsis source, fluid resuscitation, vasopressors if needed, antibiotics.

Question 38

A 55-year-old found unresponsive. Smells of alcohol. Serum osmolality 340 mOsm/kg.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.20	20 mmHg	8 mEq/L	88 mmHg	97%

How does the osmol gap help distinguish toxic alcohol ingestion?

✔ ANSWER: High-AG Metabolic Acidosis — toxic alcohol (methanol/ethylene glycol) poisoning

Step 1 – pH

7.20 → acidosis

Step 2 – AG

140 - (105+8) = 27 → elevated

Step 3 – Osmol gap

Measured osm – calculated osm = 340 – [2×Na + glucose/18 + BUN/2.8] = 340-298 = 42 (elevated; normal < 10)

Step 4 – Interpretation

Elevated osmol gap + high AG = toxic alcohol ingestion until proven otherwise

Step 5 – Key toxins

Methanol → formic acid → blindness; Ethylene glycol → oxalic acid → renal failure

Step 6 – Management

Fomepizole (antidote), hemodialysis, avoid ethanol infusion if fomepizole available.

Question 39

A 68-year-old with chronic kidney disease stage 5 (GFR < 10). Fatigue, anorexia.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.27	28 mmHg	13 mEq/L	85 mmHg	97%

What type of metabolic acidosis is seen in renal failure?

✔ ANSWER: High-AG Metabolic Acidosis — uremic acidosis

Step 1 – pH

7.27 → acidosis

Step 2 – PaCO2

28 → respiratory compensation

Step 3 – HCO3–

13 → low

Step 4 – AG

140 - (100 + 13) = 27 → elevated

Step 5 – Mechanism

Failed kidneys cannot excrete H+ or regenerate HCO3; uremic acids (sulfates, phosphates) accumulate

Step 6 – Management

Oral sodium bicarbonate supplementation, dialysis (hemodialysis/PD) when GFR < 10-15.

Question 40

A 45-year-old with chronic diarrhea (15 loose stools/day) for 2 weeks. Weak, crampy.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.28	30 mmHg	14 mEq/L	90 mmHg	98%

What type of metabolic acidosis and why is AG normal here?

✔ ANSWER: Normal-AG (Hyperchloremic) Metabolic Acidosis — GI bicarbonate loss

Step 1 – pH

7.28 → acidosis

Step 2 – PaCO2

30 → compensation

Step 3 – HCO3–

14 → low

Step 4 – AG

Na(138) - (Cl 110 + HCO3 14) = 14 → slightly elevated but near normal

Step 5 – Why normal AG?

HCO3 lost in stool → kidneys retain Cl to maintain electroneutrality → hyperchloremia → AG stays normal

Step 6 – Teaching

GI losses below pylorus = hyperchloremic metabolic acidosis (diarrhea, fistulas, ileostomy)

Step 7 – Management

IV fluid replacement with NaCl, potassium and bicarbonate repletion.

Question 41

A 35-year-old with Type 4 RTA (renal tubular acidosis) on ACE inhibitor for diabetic nephropathy.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.31	30 mmHg	16 mEq/L	88 mmHg	98%

Explain how RTA causes metabolic acidosis without elevating the AG.

✔ ANSWER: Normal-AG Metabolic Acidosis — Renal Tubular Acidosis Type 4

Step 1 – pH

7.31 → acidosis

Step 2 – PaCO2

30 → compensation

Step 3 – HCO3–

16 → low

Step 4 – AG

Normal — kidneys fail to excrete H+ but don't accumulate unmeasured anions

Step 5 – Type 4 RTA

Hypoaldosteronism (from ACE-I, DM) → failure of H+ and K+ excretion → hyperkalemia + acidosis

Step 6 – Key feature

Hyperkalemia is a hallmark of Type 4 RTA (vs Types 1 and 2 which cause hypokalemia)

Step 7 – Management

Fludrocortisone or furosemide, dietary K+ restriction, sometimes NaHCO3.

Question 42

A 40-year-old post-ureteroileal conduit (urinary diversion). Routine labs.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.28	28 mmHg	14 mEq/L	90 mmHg	98%

Why does urinary diversion cause hyperchloremic metabolic acidosis?

✔ ANSWER: Normal-AG Metabolic Acidosis — urinary diversion (ileal conduit)

Step 1 – pH

7.28 → acidosis

Step 2 – PaCO2

28 → compensation

Step 3 – HCO3–

14 → low, AG normal

Step 4 – Mechanism

Ileal segment absorbs urinary Cl– and excretes HCO3 into urine (ileum exchanges Cl for HCO3) → HCO3 is lost + Cl rises → hyperchloremic acidosis

Step 5 – Management

Oral NaHCO3 supplementation, monitor electrolytes regularly.

Question 43

A 22-year-old with bulimia nervosa is found to have a serum K+ of 2.8 and bicarbonate of 34.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.52	46 mmHg	36 mEq/L	92 mmHg	98%

This doesn't seem like acidosis — explain this ABG in context of bulimia.

✔ ANSWER: Metabolic Alkalosis — recurrent purging (see Q61 section); taught here for contrast

Step 1 – pH

7.52 → alkalosis

Step 2 – HCO3–

36 → elevated → metabolic alkalosis

Step 3 – PaCO2

46 → mildly elevated → respiratory compensation (hypoventilation)

Step 4 – Mechanism

Vomiting → HCl loss → net gain of HCO3 → metabolic alkalosis + hypokalemia

Step 5 – Teaching

This ABG contrasts with diarrhea (which causes metabolic ACIDOSIS). Distinguish upper GI loss (vomiting = alkalosis) vs lower GI loss (diarrhea = acidosis).

Question 44

A 50-year-old started on metformin 2 weeks ago. Now presents obtunded. Creatinine 4.2 mg/dL.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.15	16 mmHg	6 mEq/L	88 mmHg	97%

What dangerous metabolic complication has occurred?

✔ ANSWER: Severe High-AG Metabolic Acidosis — Metformin-Associated Lactic Acidosis (MALA)

Step 1 – pH

7.15 → severe acidosis

Step 2 – PaCO2

16 → compensatory hyperventilation

Step 3 – HCO3–

6 → severely low

Step 4 – AG

140-(104+6) = 30 → very high AG

Step 5 – Mechanism

Metformin accumulates in renal failure → inhibits mitochondrial respiration → L-lactic acidosis

Step 6 – Risk factor

Contraindicated when GFR < 30 or creatinine > 1.5 (males) / 1.4 (females)

Step 7 – Management

Stop metformin, supportive care, emergent hemodialysis if severe.

Question 45

A 30-year-old with HIV on antiretroviral therapy (NRTIs). Presents with nausea, weakness.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.22	18 mmHg	8 mEq/L	86 mmHg	97%

What metabolic complication of NRTI therapy is this?

✔ ANSWER: High-AG Metabolic Acidosis — NRTI-induced lactic acidosis

Step 1 – pH

7.22 → acidosis

Step 2 – AG

Elevated

Step 3 – Mechanism

NRTIs (e.g., stavudine, didanosine) inhibit mitochondrial DNA polymerase gamma → impaired oxidative phosphorylation → lactic acidosis

Step 4 – Management

Stop NRTIs, supportive care, IV fluids. Switch to safer ART regimen.

Question 46

A 28-year-old ingested unknown pills. Blood test: anion gap 28, lactate 2, glucose normal.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.25	20 mmHg	9 mEq/L	92 mmHg	98%

What toxin class causes high AG with unmeasured anions but not high lactate?

✔ ANSWER: High-AG Metabolic Acidosis — drug/toxin ingestion (check salicylate, iron, INH levels)

Step 1 – AG

28 → significantly elevated

Step 2 – Lactate

Normal — NOT lactic acidosis

Step 3 – MUDPILES mnemonic

M=Methanol, U=Uremia, D=DKA, P=Propylene glycol, I=Infection/Isoniazid/Iron, L=Lactic acidosis, E=Ethylene glycol, S=Salicylates

Step 4 – Approach

With elevated AG and normal lactate/glucose → check: salicylate level, iron level, osmol gap (for methanol/ethylene glycol), renal function

Step 5 – Management

Based on identified toxin.

Question 47

A 55-year-old is given large volumes of normal saline (0.9% NaCl) during resuscitation — 8 liters over 4 hours.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.28	30 mmHg	14 mEq/L	90 mmHg	99%

What iatrogenic acid-base disorder has occurred?

✔ ANSWER: Normal-AG Metabolic Acidosis — dilutional/hyperchloremic acidosis from excess NS

Step 1 – pH

7.28 → acidosis

Step 2 – HCO3–

14 → low

Step 3 – AG

Normal — no unmeasured anions

Step 4 – Mechanism

0.9% NaCl contains 154 mEq/L Cl (higher than normal plasma Cl of 100) → large volumes → hyperchloremia → HCO3 diluted + Cl rises → AG stays normal

Step 5 – Prevention

Use balanced crystalloids (LR, PlasmaLyte) for large-volume resuscitation

Step 6 – Management

Switch to balanced solution; acidosis usually self-corrects.

Question 48

A 70-year-old on TPN for 3 weeks. No other acute issues.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.30	28 mmHg	14 mEq/L	88 mmHg	97%

What metabolic acid-base issue can TPN cause?

✔ ANSWER: Normal-AG Metabolic Acidosis — TPN-associated (amino acid metabolism)

Step 1 – pH

7.30 → acidosis

Step 2 – AG

Normal

Step 3 – Mechanism

Amino acids in TPN (e.g., arginine, lysine, histidine) are cationic — their metabolism produces H+ → metabolic acidosis

Step 4 – Also

Acetate in TPN formulas can be adjusted to buffer H+ — if acetate content is low, acidosis occurs

Step 5 – Management

Add acetate to TPN formulation, adjust amino acid profile.

Question 49

A 10-year-old child with severe persistent diarrhea and dehydration.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.23	25 mmHg	10 mEq/L	90 mmHg	98%

What are the key differences in interpreting ABG in children?

✔ ANSWER: Metabolic Acidosis (Normal-AG) — pediatric diarrheal dehydration

Step 1 – Normal pediatric ABG

pH 7.35-7.45, PaCO2 35-45, HCO3 22-26 — same as adults

Step 2 – pH

7.23 → acidosis

Step 3 – HCO3–

10 → severely low

Step 4 – PaCO2

25 → compensatory

Step 5 – Compensation formula

Same Winters formula: expected PaCO2 = 1.5×10 + 8 = 23 ± 2 → actual 25 ✓ appropriate

Step 6 – Pediatric note

Dehydration + diarrhea are the #1 cause of metabolic acidosis in children

Step 7 – Management

Oral rehydration (mild), IV fluids with Na and K (moderate-severe).

Question 50

A 33-year-old marathon runner collapses at mile 24. Cramping, confusion.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.18	18 mmHg	7 mEq/L	90 mmHg	97%

What type of metabolic acidosis occurs in extreme exercise?

✔ ANSWER: High-AG Metabolic Acidosis — exercise-induced lactic acidosis

Step 1 – pH

7.18 → severe acidosis

Step 2 – AG

Elevated — lactate is the unmeasured anion

Step 3 – PaCO2

18 → respiratory compensation working hard

Step 4 – Mechanism

During extreme exertion, O2 demand exceeds supply → anaerobic glycolysis → lactate production → H+ accumulation

Step 5 – Management

Rest, IV fluids, glucose, cooling (if heat stroke component). Self-limiting — lactate clears with rest.

Question 51

A 65-year-old diabetic with new AKI from contrast nephropathy. Oliguric. BUN 80, Cr 5.8.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.24	26 mmHg	11 mEq/L	84 mmHg	96%

What is the mechanism of metabolic acidosis in AKI?

✔ ANSWER: High-AG Metabolic Acidosis — AKI with uremia

Step 1 – pH

7.24 → acidosis

Step 2 – HCO3–

11 → low

Step 3 – AG

Elevated from retained uremic acids

Step 4 – Mechanism

Acute kidney injury → impaired H+ excretion + failure to regenerate HCO3 → H+ accumulates with sulfates, phosphates, urates (unmeasured anions) → AG rises

Step 5 – Distinction

Early AKI = AG metabolic acidosis; CKD (long-standing) = may also have AG acidosis

Step 6 – Management

Treat AKI cause, fluid management, dialysis if severe acidosis or fluid overload.

Question 52

A 4-year-old ingested iron tablets (found empty pill bottle). Vomiting, lethargy.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.20	22 mmHg	9 mEq/L	88 mmHg	97%

How does iron toxicity cause metabolic acidosis?

✔ ANSWER: High-AG Metabolic Acidosis — iron poisoning

Step 1 – pH

7.20 → acidosis

Step 2 – AG

Elevated

Step 3 – Mechanism

Free iron → mitochondrial toxicity → impairs oxidative phosphorylation → lactic acidosis + direct tissue injury

Step 4 – Stages of iron poisoning

Stage 1 (0-6h): GI toxicity; Stage 2 (6-24h): apparent recovery; Stage 3 (12-48h): systemic toxicity with metabolic acidosis

Step 5 – Management

Deferoxamine chelation, supportive care, GI decontamination if early.

Question 53

A 30-year-old ingested aspirin (salicylate) — 30 tablets. Hyperventilating, tinnitus.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.48	22 mmHg	16 mEq/L	98 mmHg	99%

This is a repeat salicylate question focusing on the mixed pattern. Explain.

✔ ANSWER: Mixed: Primary Respiratory Alkalosis + Metabolic Acidosis (salicylate toxicity)

Step 1 – pH

7.48 → alkalosis — but this is a mixed disorder

Step 2 – PaCO2

22 → severely low → RESPIRATORY ALKALOSIS (salicylate stimulates respiratory center directly)

Step 3 – HCO3–

16 → low → METABOLIC ACIDOSIS (salicylic acid + lactic acid production)

Step 4 – Why alkalosis wins pH?

Respiratory alkalosis is the dominant process here

Step 5 – Key teaching

NEVER see this combination normally. Simultaneous respiratory alkalosis + metabolic acidosis = SALICYLATE until proven otherwise

Step 6 – Management

Alkaline diuresis, hemodialysis if severe, monitor electrolytes.

Question 54

A 45-year-old with severe pancreatitis requiring large fluid resuscitation.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.29	25 mmHg	12 mEq/L	82 mmHg	96%

What metabolic disorder can occur in severe pancreatitis?

✔ ANSWER: High-AG Metabolic Acidosis — severe pancreatitis (multifactorial)

Step 1 – Mechanisms

(1) Lactic acidosis from shock/hypoperfusion; (2) Loss of HCO3 in pancreatic fluid; (3) Splanchnic ischemia; (4) Saline dilution from resuscitation

Step 2 – pH

7.29 → acidosis

Step 3 – AG

Elevated

Step 4 – Management

Aggressive fluid resuscitation, NPO, pain management, treat complications including ARDS, AKI.

Question 55

A 20-year-old uses a 'huffing' substance (toluene from paint thinner). Presents drowsy.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.26	28 mmHg	12 mEq/L	88 mmHg	97%

What unique acid-base pattern does toluene (solvent) abuse cause?

✔ ANSWER: Normal-AG Metabolic Acidosis — toluene (solvent) abuse

Step 1 – pH

7.26 → acidosis

Step 2 – AG

Normal — unlike most toxic ingestions

Step 3 – Mechanism

Toluene → metabolized to hippurate and benzoate → excreted renally → while in body, displaces Cl (normal AG), but once excreted, AG normalizes and hyperchloremia persists

Step 4 – Electrolytes

Severe hypokalemia common; also hypophosphatemia

Step 5 – Teaching

Toluene = NORMAL AG acidosis with hypokalemia and high osmol gap initially

Step 6 – Management

Supportive, electrolyte replacement, avoid further exposure.

Question 56

A 60-year-old with chronic alcohol abuse found with fruity breath (not diabetes). Glucose 80. Ketones positive.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.25	22 mmHg	10 mEq/L	88 mmHg	97%

What is alcoholic ketoacidosis (AKA)?

✔ ANSWER: High-AG Metabolic Acidosis — Alcoholic Ketoacidosis

Step 1 – pH

7.25 → acidosis

Step 2 – AG

Elevated — ketones are unmeasured anions

Step 3 – PaCO2

22 → compensatory

Step 4 – Mechanism

Chronic alcohol + starvation → low insulin → high glucagon → increased lipolysis → ketone bodies (beta-hydroxybutyrate, acetoacetate)

Step 5 – Distinction from DKA

Glucose NORMAL or LOW in AKA; elevated in DKA

Step 6 – Urine ketones

May be falsely negative if mostly beta-hydroxybutyrate (not detected by typical dipstick)

Step 7 – Management

IV dextrose-containing fluids (D5NS or D5 0.45NS), thiamine before glucose, electrolytes.

Question 57

A 72-year-old given acetazolamide for glaucoma. Routine labs show metabolic acidosis.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.32	30 mmHg	16 mEq/L	86 mmHg	97%

How does acetazolamide cause metabolic acidosis?

✔ ANSWER: Normal-AG Metabolic Acidosis — acetazolamide (carbonic anhydrase inhibitor)

Step 1 – Mechanism

Acetazolamide inhibits carbonic anhydrase in proximal tubule → impaired HCO3 reabsorption → bicarbonaturia → HCO3 is lost in urine → metabolic acidosis (Type 2 RTA-like)

Step 2 – pH

7.32 → acidosis

Step 3 – AG

Normal

Step 4 – Teaching

Any drug that blocks carbonic anhydrase or inhibits HCO3 handling → Type 2 RTA picture

Step 5 – Management

Usually mild; if problematic, add potassium bicarbonate supplementation.

Question 58

A 52-year-old with ileostomy output of 2.5 L/day. Presents with weakness and cramping.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.27	27 mmHg	13 mEq/L	88 mmHg	98%

What electrolyte and acid-base problems occur with high-output ileostomy?

✔ ANSWER: Normal-AG Metabolic Acidosis — ileostomy bicarbonate loss

Step 1 – pH

7.27 → acidosis

Step 2 – AG

Normal

Step 3 – Mechanism

Ileal fluid is rich in HCO3 (30-60 mEq/L) and Na → losses → hyperchloremic metabolic acidosis + hyponatremia + dehydration

Step 4 – Electrolytes

Hyponatremia, hypokalemia, hypomagnesemia common

Step 5 – Management

Fluid replacement with Na and K, add HCO3 or acetate. Loperamide to reduce output.

Question 59

A 55-year-old with multiple myeloma and creatinine 3.5.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.28	28 mmHg	14 mEq/L	88 mmHg	98%

Explain the two mechanisms by which myeloma causes metabolic acidosis.

✔ ANSWER: Mixed Normal-AG and High-AG Metabolic Acidosis — multiple myeloma

Step 1 – Mechanism 1

Paraprotein (immunoglobulins) are cationic → act as unmeasured cations → can cause a DECREASED AG (or mask high AG). Alternatively, they don't register as anions → pseudo-normal AG

Step 2 – Mechanism 2

Renal failure from myeloma (cast nephropathy, amyloid) → uremic acid accumulation → AG metabolic acidosis

Step 3 – Teaching

In myeloma, AG may be misleadingly low — always check for acidosis even with normal AG

Step 4 – Management

Treat myeloma, manage renal failure, consider dialysis.

Question 60

A 65-year-old with type 2 diabetes has HbA1c 11%, Cr 1.9, glucose 390.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.30	24 mmHg	12 mEq/L	84 mmHg	97%

Is this DKA? Compare with hyperosmolar hyperglycemic state (HHS).

✔ ANSWER: Metabolic Acidosis (mild) — likely HHS with some element of ketoacidosis; compare DKA vs HHS

Step 1 – pH

7.30 → acidosis, but not as severe as typical DKA

Step 2 – HCO3–

12 → low

Step 3 – AG

Elevated → some ketones present

Step 4 – DKA vs HHS comparison

DKA: younger T1DM, pH < 7.3, HCO3 < 18, ketones +++, glucose usually < 600 / HHS: older T2DM, pH > 7.3, HCO3 > 18, minimal ketones, glucose > 600, severe dehydration

Step 5 – This case

Overlap — mixed DKA/HHS in T2DM. Treat: IVF, insulin, electrolytes.

Section 4: Metabolic Alkalosis

Questions 61–75 | Bicarbonate Excess & H+ Loss

Question 61

A 25-year-old with cyclic vomiting presents with weakness, palpitations. Na 138, K 2.8, Cl 88.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.54	48 mmHg	40 mEq/L	90 mmHg	98%

Identify the disorder and explain the chloride connection.

✔ ANSWER: Metabolic Alkalosis — contraction alkalosis from vomiting

Step 1 – pH

7.54 → alkalosis

Step 2 – HCO3–

40 → elevated → primary metabolic alkalosis

Step 3 – PaCO2

48 → mild elevation (respiratory compensation = hypoventilation)

Step 4 – Mechanism

Vomiting → HCl loss → H+ lost → HCO3 rises; also Cl depletion → kidneys reabsorb more HCO3 to maintain electrical neutrality

Step 5 – Chloride-responsive alkalosis

Urine Cl < 20 mEq/L → responds to Cl replacement

Step 6 – Management

IV NS (saline-responsive), KCl replacement, treat vomiting cause.

Question 62

A 60-year-old on furosemide 80mg daily for CHF. K+ 3.0, Cl 92.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.50	46 mmHg	36 mEq/L	88 mmHg	97%

How does furosemide cause metabolic alkalosis?

✔ ANSWER: Metabolic Alkalosis — diuretic-induced (loop diuretic)

Step 1 – pH

7.50 → alkalosis

Step 2 – HCO3–

36 → elevated

Step 3 – PaCO2

46 → compensatory hypoventilation

Step 4 – Mechanism

Furosemide → Na/K/2Cl cotransporter inhibition → Cl excretion → contraction alkalosis; also K+ and H+ excretion increases → metabolic alkalosis

Step 5 – Key feature

Hypokalemia is common and perpetuates alkalosis (H+ shifts intracellularly when K+ low)

Step 6 – Management

KCl replacement, consider switching to K-sparing diuretic (spironolactone). Saline if volume depleted.

Question 63

A 45-year-old with primary hyperaldosteronism (Conn syndrome). Hypertension, K+ 2.5.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.50	46 mmHg	36 mEq/L	90 mmHg	97%

What is the ABG pattern in hyperaldosteronism and why?

✔ ANSWER: Metabolic Alkalosis — hyperaldosteronism (chloride-resistant)

Step 1 – pH

7.50 → alkalosis

Step 2 – HCO3–

36 → elevated

Step 3 – Mechanism

Aldosterone acts on collecting duct → Na reabsorption → K+ and H+ excretion → H+ loss → HCO3 rises → metabolic alkalosis

Step 4 – Chloride-resistant

Urine Cl > 20 mEq/L; does NOT respond to saline — need to address aldosterone excess

Step 5 – Management

Spironolactone (aldosterone antagonist), surgical removal of adenoma if Conn's, treat hypokalemia.

Question 64

A 30-year-old with Cushing's syndrome on long-term high-dose corticosteroids.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.48	46 mmHg	34 mEq/L	90 mmHg	98%

Why does Cushing's cause metabolic alkalosis?

✔ ANSWER: Metabolic Alkalosis — glucocorticoid excess (mineralocorticoid effect)

Step 1 – pH

7.48 → alkalosis

Step 2 – HCO3–

34 → elevated

Step 3 – Mechanism

Cortisol in high concentrations has mineralocorticoid (aldosterone-like) effects → H+ and K+ excretion → metabolic alkalosis + hypokalemia

Step 4 – Management

Treat underlying cause (taper steroids, remove tumor). Potassium repletion.

Question 65

A 55-year-old post-cardiac surgery received 10 units of blood transfusion overnight.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.50	44 mmHg	34 mEq/L	92 mmHg	98%

What causes metabolic alkalosis post-massive transfusion?

✔ ANSWER: Metabolic Alkalosis — massive transfusion (citrate metabolism)

Step 1 – Mechanism

Blood products contain citrate as anticoagulant. Liver metabolizes citrate → bicarbonate → massive transfusion → HCO3 load → metabolic alkalosis

Step 2 – Additional factor

If acidosis was present pre-transfusion and NaHCO3 was given → 'over-correction'

Step 3 – pH

7.50 → alkalosis

Step 4 – HCO3–

34 → elevated

Step 5 – Management

Usually self-limiting. Monitor electrolytes, especially calcium (citrate chelates Ca2+).

Question 66

A 68-year-old is on nasogastric suction for 5 days post-op bowel obstruction.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.52	48 mmHg	38 mEq/L	86 mmHg	96%

How does NG suction lead to metabolic alkalosis?

✔ ANSWER: Metabolic Alkalosis — NG suction (gastric acid loss)

Step 1 – Mechanism

NG suction removes gastric HCl → H+ loss → relative HCO3 excess → alkalosis; also Cl depletion perpetuates it (kidneys retain HCO3 to replace lost Cl)

Step 2 – pH

7.52 → alkalosis

Step 3 – HCO3–

38 → elevated

Step 4 – PaCO2

48 → compensatory

Step 5 – Management

Cl replacement (isotonic saline), KCl. If suction is continued, may need IV HCl in severe cases.

Question 67

A 70-year-old with CHF on digoxin and furosemide. K+ 2.2, presents with nausea, PVCs on ECG.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.52	46 mmHg	38 mEq/L	86 mmHg	96%

How does hypokalemia perpetuate metabolic alkalosis?

✔ ANSWER: Metabolic Alkalosis perpetuated by hypokalemia — digoxin toxicity risk

Step 1 – Mechanism

Low K+ → K+ shifts out of cells → H+ shifts IN (to maintain neutrality) → intracellular acidosis + extracellular alkalosis. Also kidneys try to retain K+ → secrete more H+ → more alkalosis

Step 2 – Digoxin toxicity

Hypokalemia sensitizes myocardium to digoxin toxicity — key drug interaction

Step 3 – Management

Aggressive KCl IV replacement. Hold digoxin until K+ corrected. Monitor ECG.

Question 68

A 78-year-old with severe COPD and PaCO2 baseline 60 is given high-flow O2 and IV furosemide.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.50	54 mmHg	42 mEq/L	70 mmHg	92%

What combined disturbance is present?

✔ ANSWER: Mixed Respiratory Acidosis + Metabolic Alkalosis (COPD + diuretics)

Step 1 – pH

7.50 → alkalosis

Step 2 – PaCO2

54 → elevated → respiratory acidosis component

Step 3 – HCO3–

42 → markedly elevated → metabolic alkalosis (from furosemide + pre-existing compensation)

Step 4 – Mixed disorder

Primary respiratory acidosis (COPD, elevated PaCO2) PLUS metabolic alkalosis (diuretics) — these oppose each other, resulting in near-normal or alkalemic pH

Step 5 – Teaching

Elevated HCO3 in COPD can be compensation OR superimposed metabolic alkalosis — check if HCO3 is too high for expected compensation

Step 6 – Management

Stop or reduce diuretics, treat COPD exacerbation, add spironolactone.

Question 69

A 28-year-old athlete uses sodium bicarbonate (baking soda) as an ergogenic aid before a race.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.50	44 mmHg	34 mEq/L	96 mmHg	99%

What ABG pattern does exogenous bicarbonate loading cause?

✔ ANSWER: Metabolic Alkalosis — exogenous bicarbonate loading

Step 1 – Mechanism

Ingested NaHCO3 → directly raises serum HCO3 → metabolic alkalosis

Step 2 – pH

7.50 → alkalosis

Step 3 – HCO3–

34 → elevated

Step 4 – Clinical use

Some athletes use NaHCO3 to buffer exercise-induced lactic acidosis — minimal evidence, GI side effects common

Step 5 – Risk

Tetany (low ionized Ca), nausea, vomiting

Question 70

A 60-year-old with Bartter syndrome. Normal BP, hypokalemia, metabolic alkalosis.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.50	44 mmHg	36 mEq/L	90 mmHg	98%

How does Bartter syndrome differ from primary hyperaldosteronism in causing alkalosis?

✔ ANSWER: Metabolic Alkalosis — Bartter syndrome (loop of Henle defect)

Step 1 – Mechanism

Bartter = defect in Na/K/2Cl transporter in thick ascending limb (same target as furosemide) → salt wasting → secondary hyperaldosteronism → H+ and K+ excretion → alkalosis

Step 2 – Key features

Normal or low BP (unlike primary hyperaldosteronism which has hypertension)

Step 3 – Comparison

Primary hyperaldosteronism: hypertensive + alkalosis / Bartter: normotensive + alkalosis / Gitelman: similar to Bartter but hypomagnesemia, hypocalciuria

Step 4 – Management

KCl supplementation, NSAIDs (indomethacin), K-sparing diuretics.

Question 71

A 55-year-old given IV sodium bicarbonate for a metabolic acidosis. Nurse notices RR has decreased.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.50	50 mmHg	38 mEq/L	90 mmHg	98%

What has happened and is this expected?

✔ ANSWER: Metabolic Alkalosis — over-correction with NaHCO3 administration

Step 1 – pH

7.50 → now alkalotic (overcorrected)

Step 2 – HCO3–

38 → elevated

Step 3 – PaCO2

50 → rising (respiratory compensation for alkalosis = hypoventilation)

Step 4 – Complication

NaHCO3 can also raise CO2 (as bicarbonate generates CO2 when it buffers H+) — in closed-circuit patients (intubated) this matters more

Step 5 – Management

Reassess; may need to reduce NaHCO3 dose. Ensure adequate ventilation.

Question 72

A 46-year-old with primary adrenal insufficiency (Addison's) on fludrocortisone.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.42	38 mmHg	24 mEq/L	90 mmHg	98%

Is this ABG abnormal? What if fludrocortisone dose is excessive?

✔ ANSWER: Normal ABG — but excess fludrocortisone would cause metabolic alkalosis

Step 1 – Current ABG

Normal — patient is adequately replaced

Step 2 – If fludrocortisone excessive

Excess mineralocorticoid → Na retention → H+ and K+ excretion → metabolic alkalosis + hypokalemia (same mechanism as primary hyperaldosteronism)

Step 3 – If fludrocortisone insufficient

Opposite: hyperkalemia, metabolic acidosis (hypoaldosteronism = Type 4 RTA)

Step 4 – Teaching

Fludrocortisone dose titration guided by BP, K+, and sometimes ABG.

Question 73

A 33-year-old on antacids (calcium carbonate) chronically for GERD. Presents with muscle weakness.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.48	45 mmHg	33 mEq/L	88 mmHg	97%

What syndrome can chronic antacid use cause?

✔ ANSWER: Metabolic Alkalosis — Milk-Alkali Syndrome (antacid overuse)

Step 1 – Mechanism

Chronic ingestion of Ca-based antacids → hypercalcemia + alkalosis → Milk-Alkali Syndrome (Burnett Syndrome)

Step 2 – Triad

Hypercalcemia + metabolic alkalosis + renal insufficiency

Step 3 – pH

7.48 → alkalosis

Step 4 – HCO3–

33 → elevated

Step 5 – Management

Stop antacid, IV NS to treat hypercalcemia, monitor renal function.

Question 74

A 56-year-old post-pyloric surgery with retained gastric secretions.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.50	48 mmHg	37 mEq/L	88 mmHg	97%

Why does pyloric obstruction cause severe metabolic alkalosis?

✔ ANSWER: Metabolic Alkalosis — pyloric obstruction (projectile vomiting)

Step 1 – Mechanism

Pyloric obstruction → vomiting of ONLY gastric contents (HCl) → massive HCl loss → H+ depletion → HCO3 rises markedly → paradoxical aciduria (kidneys excrete H+ to retain K+ in hypokalemia → urine acidic despite alkalemia)

Step 2 – Paradoxical aciduria

Classic finding in pyloric obstruction alkalosis

Step 3 – Electrolytes

Hypokalemia, hypochloremia

Step 4 – Management

IV NS + KCl to correct volume and electrolytes; surgical correction.

Question 75

A 50-year-old with refeeding syndrome after aggressive nutrition in malnourished state.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.48	44 mmHg	32 mEq/L	90 mmHg	98%

What electrolyte shifts in refeeding syndrome affect acid-base balance?

✔ ANSWER: Metabolic Alkalosis — refeeding syndrome (phosphate, potassium, and magnesium depletion)

Step 1 – Mechanism

Refeeding → insulin surge → intracellular shift of K+, Mg2+, PO4 → hypokalemia → promotes metabolic alkalosis (same K-related mechanism)

Step 2 – Hypophosphatemia

Most dangerous → impairs ATP production, respiratory muscle weakness → may cause respiratory acidosis if severe

Step 3 – This ABG

Metabolic alkalosis from K+ shifts

Step 4 – Prevention

Slow refeeding, monitor electrolytes daily, supplement K/Mg/PO4.

Section 5: Mixed Disorders & Advanced

Questions 76–100 | Complex & Combined Disturbances

Question 76

ICU patient: pH 7.40, PaCO2 60, HCO3 36. On mechanical ventilation for COPD.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.40	60 mmHg	36 mEq/L	68 mmHg	94%

pH is 7.40 — is this normal? Or a mixed disorder?

✔ ANSWER: Mixed: Chronic Respiratory Acidosis + Metabolic Alkalosis — 'normal' pH is deceptive

Step 1 – pH

7.40 → appears normal → but this is NOT normal

Step 2 – PaCO2

60 → markedly elevated → respiratory acidosis

Step 3 – HCO3–

36 → markedly elevated → metabolic alkalosis

Step 4 – Expected compensation for PaCO2 60

Chronic: HCO3 should be ~29. Actual 36 → EXTRA elevation beyond compensation → metabolic alkalosis is ALSO present

Step 5 – Conclusion

Two disorders (respiratory acidosis + metabolic alkalosis) are canceling each other out → 'pseudo-normal' pH

Step 6 – Teaching

ALWAYS check if PaCO2 and HCO3 changes are consistent with pure compensation before calling an ABG 'normal'.

Question 77

A 50-year-old with COPD and congestive heart failure on furosemide. pH 7.43.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.43	58 mmHg	38 mEq/L	62 mmHg	92%

What mixed disorder is present?

✔ ANSWER: Mixed Respiratory Acidosis + Metabolic Alkalosis (COPD + diuretic use)

Step 1 – PaCO2

58 → elevated → respiratory acidosis

Step 2 – HCO3–

38 → elevated beyond what chronic COPD compensation would give

Step 3 – Expected HCO3 for PaCO2 58

3.5 × (58-40)/10 + 24 = 3.5 × 1.8 + 24 = 30.3. Actual 38 >> 30 → metabolic alkalosis ON TOP

Step 4 – pH

7.43 → nearly normal → both disorders oppose each other

Step 5 – Management

Treat both: COPD management + reduce diuretics/add spironolactone to correct alkalosis.

Question 78

A 35-year-old with sepsis develops AKI and starts hyperventilating.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.38	25 mmHg	14 mEq/L	80 mmHg	97%

This pH appears normal — is it? Explain the triple acid-base disorder.

✔ ANSWER: Triple Disorder: High-AG Metabolic Acidosis + Respiratory Alkalosis + Metabolic Alkalosis

Step 1 – Step 1

pH 7.38 — seemingly normal

Step 2 – Step 2

PaCO2 25 → low → respiratory alkalosis

Step 3 – Step 3

HCO3 14 → low → metabolic acidosis

Step 4 – Step 4 — AG

If AG = 30 → delta ratio = (30-12)/(24-14) = 18/10 = 1.8 → delta HCO3 suggests ADDITIONAL metabolic alkalosis

Step 5 – Delta ratio interpretation

< 1 = normal AG also present; 1-2 = pure high-AG; > 2 = metabolic alkalosis ALSO present

Step 6 – Conclusion

Sepsis (lactic acidosis) + hyperventilation + prior diuretic use (metabolic alkalosis) = triple disorder

Question 79

A 26-year-old with anorexia nervosa has persistent vomiting and laxative abuse.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.50	46 mmHg	36 mEq/L	90 mmHg	98%

How do vomiting and laxative abuse create opposing acid-base effects?

✔ ANSWER: Predominant Metabolic Alkalosis (from vomiting) with blunted effect from laxative acidosis

Step 1 – Vomiting

HCl loss → metabolic alkalosis → pH rises, HCO3 rises

Step 2 – Laxative abuse

HCO3 loss from colon → metabolic acidosis → opposes alkalosis

Step 3 – Net effect

Usually alkalosis predominates as gastric acid loss is more potent

Step 4 – This ABG

pH 7.50 → net alkalosis; HCO3 36 → elevated

Step 5 – Teaching

Mixed disorders may partially cancel — assess context and specific behaviors

Question 80

A 70-year-old COPD patient given NaHCO3 for metabolic acidosis related to CHF exacerbation.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.45	64 mmHg	43 mEq/L	60 mmHg	92%

What triple disorder might present?

✔ ANSWER: Mixed: Respiratory Acidosis + Metabolic Alkalosis (from NaHCO3) + Metabolic Acidosis (from CHF lactic acidosis)

Step 1 – Clinical context

COPD = chronic respiratory acidosis (high PaCO2, compensatory high HCO3). CHF = tissue hypoperfusion = lactic acidosis. NaHCO3 administration = iatrogenic alkalosis

Step 2 – PaCO2

64 → elevated → respiratory acidosis

Step 3 – HCO3–

43 → extremely elevated → combination of chronic compensation + NaHCO3 + CHF lactic alkalosis

Step 4 – Warning

NaHCO3 in COPD patients can be dangerous — raises PaCO2 further and worsens respiratory acidosis

Step 5 – Management

Address primary causes; avoid unnecessary bicarbonate in COPD.

Question 81

A 60-year-old on peritoneal dialysis. ABG sent as routine check.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.40	38 mmHg	23 mEq/L	90 mmHg	98%

What parameters do you monitor in dialysis patients' ABG?

✔ ANSWER: Normal ABG — peritoneal dialysis patient with adequate acid-base management

Step 1 – Interpretation

Normal ABG — dialysate is bicarbonate or acetate buffered, correcting uremic acidosis

Step 2 – Key monitoring

PaCO2 (ensure no respiratory compensation needed), HCO3 (target 22-26 on dialysis), K+ (critical in renal failure)

Step 3 – Teaching

Dialysis aims to normalize acid-base. If HCO3 low despite dialysis → check adequacy, diet, residual renal function

Step 4 – Dialysis target

HCO3 22-26 pre-dialysis; K+ 3.5-5.5; pH 7.35-7.45.

Question 82

A 55-year-old post-liver transplant. ICU day 2. On tacrolimus.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.32	28 mmHg	14 mEq/L	85 mmHg	97%

What renal acid-base complication can calcineurin inhibitors cause?

✔ ANSWER: Normal-AG Metabolic Acidosis — calcineurin inhibitor-induced RTA Type 4

Step 1 – Mechanism

Tacrolimus/cyclosporine → distal tubule dysfunction + hypoaldosteronism-like state → Type 4 RTA → hyperchloremic metabolic acidosis + hyperkalemia

Step 2 – pH

7.32 → acidosis

Step 3 – AG

Normal

Step 4 – Management

Fludrocortisone, NaHCO3 supplementation, check tacrolimus levels, monitor K+.

Question 83

An 80-year-old with altered mental status. Na 128, K 5.5, Cl 95, HCO3 16.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.26	26 mmHg	16 mEq/L	86 mmHg	97%

Calculate and interpret the anion gap with the serum electrolytes given.

✔ ANSWER: High-AG Metabolic Acidosis — with correction for hyponatremia

Step 1 – AG calculation

Na - (Cl + HCO3) = 128 - (95+16) = 17 → slightly elevated

Step 2 – Hyponatremia correction

In low sodium states, AG may appear lower. Corrected AG = AG + 2.5 × (4.0 - albumin) if albumin low

Step 3 – Albumin correction

If albumin = 2.0 g/dL: corrected AG = 17 + 2.5×(4-2) = 17+5 = 22 → clearly elevated

Step 4 – Teaching

Always correct AG for albumin! Low albumin falsely lowers AG and can mask high-AG acidosis

Step 5 – Cause

Likely uremia or lactic acidosis in this elderly patient with altered MS.

Question 84

A 40-year-old with DKA admitted with pH 7.10. After 6 hours of treatment pH is 7.35 but HCO3 is 15.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.35	22 mmHg	15 mEq/L	92 mmHg	98%

Is the DKA resolved? Explain the 'hyperchloremic metabolic acidosis' post-DKA treatment.

✔ ANSWER: Persistent Metabolic Acidosis — hyperchloremic phase after DKA treatment

Step 1 – pH

7.35 → improving but still acidotic

Step 2 – HCO3–

15 → still low

Step 3 – AG

If now normal → ketones cleared

Step 4 – Explanation

As ketones are metabolized (or excreted), the anion gap closes. BUT large volumes of NS given during DKA treatment → hyperchloremia → replaces high-AG acidosis with normal-AG acidosis

Step 5 – This is expected

Hyperchloremic acidosis post-DKA is not an emergency — resolves as kidneys excrete Cl over days

Step 6 – Key teaching

In DKA treatment, track BOTH AG (for ketosis resolution) AND total HCO3 (not just one).

Question 85

A 33-year-old has pH 7.45, PaCO2 40, HCO3 28. The physician asks if there's a hidden disorder.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.45	40 mmHg	28 mEq/L	92 mmHg	98%

Is this a normal ABG or could a mixed disorder be hiding?

✔ ANSWER: Possible hidden Metabolic Alkalosis — need AG and clinical context

Step 1 – Apparent normal

pH, PaCO2 both normal — seems fine

Step 2 – But HCO3 = 28

Normal HCO3 is 22-26. HCO3 28 is MILDLY elevated — could be metabolic alkalosis

Step 3 – PaCO2 is NOT elevated

If it were pure metabolic alkalosis, expected PaCO2 = 0.7×(28-24)+40 = 42.8 → actual 40 → slightly undercompensated

Step 4 – Hidden disorder?

Possible metabolic alkalosis with incomplete respiratory compensation, OR recent resolution of respiratory acidosis (COPD)

Step 5 – Teaching

Never declare 'normal' just because pH is 7.35-7.45. Check each component.

Question 86

A 65-year-old with an ABG: pH 7.42, PaCO2 50, HCO3 32. O2 sat 90% on room air.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.42	50 mmHg	32 mEq/L	60 mmHg	90%

How do you know if the HCO3 elevation is compensation or a second disorder?

✔ ANSWER: Mixed Respiratory Acidosis + Metabolic Alkalosis vs Pure Chronic Respiratory Acidosis

Step 1 – Step 1

PaCO2 50 → respiratory acidosis

Step 2 – Step 2 — expected HCO3 (chronic)

3.5 × (50-40)/10 + 24 = 3.5 + 24 = 27.5. Actual HCO3 = 32 → HIGHER than expected → additional metabolic alkalosis

Step 3 – Step 3

If HCO3 32 were pure chronic compensation, expected PaCO2 = (32-24)/0.35 + 40 = 62.9 → actual 50 → less acidosis than expected

Step 4 – Conclusion

Mixed disorder: respiratory acidosis + metabolic alkalosis (likely COPD + diuretics or vomiting)

Step 5 – Teaching

Always compare actual HCO3 to expected compensation using the formula.

Question 87

A 55-year-old with toxic ingestion: pH 7.35, PaCO2 45, HCO3 24. Seems normal. Osmol gap is 22.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.35	45 mmHg	24 mEq/L	88 mmHg	97%

The ABG is 'normal' but the osmol gap is elevated. What does this mean?

✔ ANSWER: Hidden toxic ingestion — normal ABG with elevated osmol gap = early toxic alcohol poisoning

Step 1 – ABG

Normal — but this is an early snapshot

Step 2 – Osmol gap

Normal < 10; here 22 → elevated → suggests osmotically active substance present (not accounted for in calculated osmolality)

Step 3 – Suspected toxins

Methanol, ethylene glycol, isopropyl alcohol

Step 4 – Why ABG normal early?

The parent compound (alcohol) causes osmol gap BEFORE metabolism to toxic acid → once metabolized → high AG acidosis appears

Step 5 – Teaching

Elevated osmol gap = early toxic alcohol ingestion. DO NOT wait for acidosis to develop. Check levels and treat immediately.

Step 6 – Management

Fomepizole, check methanol/ethylene glycol levels, prepare for dialysis.

Question 88

A 45-year-old post-CABG day 1 in ICU. On mechanical ventilation. ABG: pH 7.55, PaCO2 28, HCO3 24.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.55	28 mmHg	24 mEq/L	98 mmHg	99%

Classify and troubleshoot this post-cardiac surgery ABG.

✔ ANSWER: Acute Respiratory Alkalosis — over-ventilation post-CABG

Step 1 – pH

7.55 → alkalosis

Step 2 – PaCO2

28 → low → over-ventilated

Step 3 – HCO3–

24 → normal → acute event

Step 4 – Harm of alkalosis post-CABG

Respiratory alkalosis causes coronary artery vasoconstriction, shifts O2-Hgb curve left (less O2 delivery), can cause arrhythmias

Step 5 – Action

Reduce ventilator RR or TV to allow PaCO2 to rise to 35-45 mmHg. Reassess in 30 min.

Question 89

Neonatal ICU: 30-week premature infant, RDS. On surfactant and CPAP.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.27	55 mmHg	24 mEq/L	50 mmHg	86%

How does neonatal respiratory distress syndrome (RDS) affect ABG?

✔ ANSWER: Acute Respiratory Acidosis + Hypoxemia — neonatal RDS

Step 1 – Neonatal normal values

pH 7.35-7.45, PaCO2 35-45, HCO3 20-24, PaO2 50-80 on room air (lower than adult)

Step 2 – pH

7.27 → acidosis

Step 3 – PaCO2

55 → elevated → respiratory — surfactant deficiency → alveolar collapse → hypoventilation

Step 4 – PaO2

50 → at lower limit for neonate — hypoxemia

Step 5 – Management

Surfactant replacement, CPAP/ventilator, supportive care, temperature management

Question 90

A 66-year-old with COPD on home O2. PaO2 on 2L nasal cannula is 95 mmHg.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.38	55 mmHg	32 mEq/L	95 mmHg	97%

What is the risk of giving too much O2 to a COPD patient?

✔ ANSWER: Chronic Respiratory Acidosis — risk of hypoxic drive loss with high-flow O2

Step 1 – pH

7.38 → near normal — chronic compensation

Step 2 – PaCO2

55 → chronically elevated

Step 3 – HCO3–

32 → compensated

Step 4 – Hypoxic drive concept

COPD patients rely on hypoxia to stimulate breathing ('hypoxic drive') because their chemoreceptors are 'reset' to tolerate high CO2

Step 5 – Danger

High-flow O2 → relieves hypoxia → removes the drive to breathe → CO2 rises further → acute respiratory acidosis

Step 6 – Target

In COPD: target SpO2 88-92% (not 100%). Use controlled O2 therapy.

Question 91

A 40-year-old runner's ABG at VO2 max: pH 7.20, PaCO2 22, HCO3 9.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.20	22 mmHg	9 mEq/L	100 mmHg	99%

Perform a complete step-by-step ABG interpretation.

✔ ANSWER: Severe Metabolic Acidosis with respiratory compensation — extreme exercise (lactic acidosis)

Step 1 – Step 1 — pH

7.20 → ACIDOSIS

Step 2 – Step 2 — Primary disorder

PaCO2 22 (low) → moves pH UP (toward alkalosis). HCO3 9 (low) → moves pH DOWN (toward acidosis). Since pH is acidotic and HCO3 is low → PRIMARY METABOLIC ACIDOSIS

Step 3 – Step 3 — Compensation

Expected PaCO2 = 1.5×9 + 8 = 21.5 ± 2. Actual PaCO2 = 22 → APPROPRIATE compensation

Step 4 – Step 4 — AG

If Na 140, Cl 100: AG = 140-(100+9) = 31 → HIGH-AG → lactic acidosis from anaerobic exercise

Step 5 – Step 5 — PaO2

100 → normal/good oxygenation

Step 6 – Conclusion

Severe high-AG metabolic acidosis from exercise-induced lactic acidosis, appropriately compensated.

Question 92

A patient's ABG: pH 7.40, PaCO2 20, HCO3 12. Anion gap 28.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.40	20 mmHg	12 mEq/L	90 mmHg	98%

pH is normal — is this truly normal? Use delta-delta analysis.

✔ ANSWER: Mixed High-AG Metabolic Acidosis + Metabolic Alkalosis (delta-delta reveals hidden alkalosis)

Step 1 – Step 1

pH 7.40 → seems normal

Step 2 – Step 2

PaCO2 20 → low; HCO3 12 → low → metabolic acidosis with respiratory compensation

Step 3 – Step 3 — AG

28 → elevated → high-AG metabolic acidosis

Step 4 – Step 4 — Delta Ratio

(AG - 12) / (24 - HCO3) = (28-12)/(24-12) = 16/12 = 1.33 → expected range 1-2 for pure high-AG

Step 5 – Delta-delta analysis

Expected HCO3 if ONLY high-AG acidosis present = 24 - (AG-12) = 24-16 = 8. Actual HCO3 = 12 → HIGHER than expected → extra HCO3 → metabolic alkalosis also present

Step 6 – Conclusion

High-AG metabolic acidosis (lactic acidosis/DKA) + metabolic alkalosis (vomiting/diuretics) simultaneously. pH appears normal because they cancel.

Question 93

A 25-year-old overdosed on tricyclic antidepressants (TCA). Wide complex tachycardia on ECG.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.18	35 mmHg	13 mEq/L	88 mmHg	97%

What ABG is expected in TCA overdose and how is it treated with bicarbonate?

✔ ANSWER: Metabolic Acidosis — TCA toxicity with sodium bicarbonate as antidote

Step 1 – TCA mechanism

Na+ channel blockade → widened QRS, hypotension; alpha blockade → hypotension; anticholinergic effects

Step 2 – ABG

Metabolic acidosis from shock/lactic acidosis; may also have respiratory depression

Step 3 – pH

7.18 → severe acidosis

Step 4 – Bicarbonate treatment

IV NaHCO3 raises pH AND increases Na+ load → reverses Na+ channel blockade → narrows QRS → reduces arrhythmia risk

Step 5 – Target

pH 7.45-7.55 with NaHCO3 (intentionally alkalotic to treat TCA toxicity)

Step 6 – Management

IV NaHCO3 bolus for wide QRS > 100ms or acidosis; lipid emulsion; intubation if needed.

Question 94

A 38-year-old with cystic fibrosis. Chronic sputum production, FEV1 25% predicted.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.33	60 mmHg	31 mEq/L	56 mmHg	90%

What acid-base pattern is seen in severe cystic fibrosis?

✔ ANSWER: Chronic Respiratory Acidosis — CF with severe obstructive/restrictive disease

Step 1 – pH

7.33 → acidosis

Step 2 – PaCO2

60 → elevated → chronic CO2 retention from severe airflow obstruction

Step 3 – HCO3–

31 → elevated → chronic renal compensation

Step 4 – Expected HCO3

3.5 × (60-40)/10 + 24 = 3.5×2 + 24 = 31 ✓ appropriate chronic compensation

Step 5 – CF lung disease

Mucus plugging → obstruction → ventilation-perfusion mismatch → hypoxia + hypercapnia

Step 6 – Management

Airway clearance, bronchodilators, CFTR modulators (e.g., ivacaftor/elexacaftor), O2 therapy, lung transplant evaluation.

Question 95

A 72-year-old admitted for elective hip surgery. Pre-op ABG.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.43	36 mmHg	24 mEq/L	92 mmHg	98%

This looks normal. What pre-op ABG findings would concern you?

✔ ANSWER: Normal Pre-op ABG — understanding the concerning patterns

Step 1 – This ABG

Completely normal — good surgical candidate from pulmonary standpoint

Step 2 – Concerning findings

(1) Low PaO2 (< 70) on room air → pulmonary reserve limited; (2) Elevated PaCO2 (> 45) → CO2 retainer, high-risk for post-op respiratory failure; (3) Low HCO3 → metabolic acidosis → investigate cause; (4) pH < 7.35 → acidosis → need to optimize before surgery

Step 3 – Pre-op cutoffs

PaO2 > 60 and PaCO2 < 45 usually acceptable. FEV1 matters more for pulmonary reserve.

Question 96

A 60-year-old is being weaned from the ventilator. Current settings: PS 5/PEEP 5. ABG done.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.38	42 mmHg	24 mEq/L	88 mmHg	97%

Is this patient ready for extubation based on the ABG?

✔ ANSWER: Normal ABG on minimal vent support — likely ready for extubation trial

Step 1 – pH, PaCO2, HCO3

All normal — gas exchange adequate on minimal support

Step 2 – Additional weaning criteria

RSBI (RR/TV) < 105, NIF < -20 cmH2O, adequate mentation, minimal secretions, hemodynamically stable, O2 sat > 90% on FiO2 ≤ 0.4

Step 3 – ABG alone insufficient

ABG tells you about gas exchange but NOT about airway protection or secretion management

Step 4 – Extubation decision

Multifactorial — ABG + clinical criteria. This ABG is SUPPORTIVE but not the only factor.

Question 97

A 55-year-old patient has ABGA at 3 PM and at 9 PM. At 3 PM: pH 7.42, PaCO2 38, HCO3 24. At 9 PM: pH 7.30, PaCO2 42, HCO3 20.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.30	42 mmHg	20 mEq/L	82 mmHg	96%

What has changed in 6 hours? Is the respiratory component the issue?

✔ ANSWER: Developing Metabolic Acidosis — new acute process in 6 hours

Step 1 – 3 PM ABG

Normal

Step 2 – 9 PM ABG

pH 7.30 → acidosis; PaCO2 42 → NORMAL → respiratory is NOT the cause; HCO3 20 → decreased → metabolic acidosis developing

Step 3 – Compensation check

If pure metabolic: expected PaCO2 = 1.5×20+8 = 38. Actual 42 → slightly higher → compensation NOT fully established OR mixed disorder emerging

Step 4 – Clinical concern

Rapid development of metabolic acidosis in 6 hours = investigate for sepsis, lactic acidosis, AKI, bowel ischemia

Step 5 – Action

Urgent workup: lactate, cultures, fluid balance, renal function.

Question 98

A 36-year-old pregnant woman at 34 weeks presents with severe headache. BP 170/110, proteinuria 3+.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.50	27 mmHg	21 mEq/L	96 mmHg	99%

How does preeclampsia modify the normal pregnant ABG?

✔ ANSWER: Respiratory Alkalosis — increased from normal pregnancy baseline (preeclampsia-related hyperventilation)

Step 1 – Normal pregnancy

pH 7.40-7.45, PaCO2 28-32, HCO3 18-22

Step 2 – This patient

pH 7.50, PaCO2 27 → even more alkalotic and hypocapnic than expected for pregnancy

Step 3 – Cause

Severe preeclampsia/pain/anxiety → further hyperventilation above pregnancy baseline

Step 4 – Concern

The primary danger is eclamptic seizure, not the ABG change. Hepatic rupture risk.

Step 5 – Management

Magnesium sulfate (seizure prophylaxis), antihypertensives (labetalol, hydralazine), plan delivery.

Question 99

A 68-year-old intubated patient with ARDS on ventilator. FiO2 0.8, PEEP 14. P/F ratio = 80.

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.30	52 mmHg	25 mEq/L	65 mmHg	90%

What is permissive hypercapnia and when is it used?

✔ ANSWER: Respiratory Acidosis — intentional in ARDS (lung-protective ventilation strategy)

Step 1 – pH

7.30 → acidosis — this is ACCEPTED in ARDS

Step 2 – Strategy

Lung-protective ventilation (ARMA trial): TV 6 mL/kg IBW, plateau pressure < 30 cmH2O → may not clear all CO2 → PaCO2 rises

Step 3 – Permissive hypercapnia

Allowing PaCO2 to rise above normal (up to 60-65 mmHg) and pH to fall to 7.20-7.25 to avoid ventilator-induced lung injury (VILI)

Step 4 – Why acceptable?

Barotrauma/volutrauma from high pressures is more dangerous than mild acidosis

Step 5 – Limits

pH should not fall below 7.20. If so: increase RR (but watch for auto-PEEP), consider prone positioning.

Question 100

A 45-year-old nurse asks: 'My patient's ABG shows pH 7.36, PaCO2 44, HCO3 24, PaO2 78, SaO2 97%. Are there any abnormalities?'

pH	$PaCO_2$	HCO_3^-	PaO_2	SaO_2
7.36	44 mmHg	24 mEq/L	78 mmHg	97%

Perform a complete, systematic 5-step ABG interpretation.

✔ ANSWER: Normal ABG — complete systematic interpretation

Step 1 – Step 1 — pH

7.36 → normal (7.35-7.45). Tendency toward acidosis end of normal.

Step 2 – Step 2 — PaCO2

44 mmHg → normal (35-45). No respiratory disturbance.

Step 3 – Step 3 — HCO3–

24 mEq/L → normal (22-26). No metabolic disturbance.

Step 4 – Step 4 — Compensation

Not applicable — both primary values normal.

Step 5 – Step 5 — Oxygenation

PaO2 78 mmHg → borderline low (normal ≥80 on room air). If age 45, age-adjusted minimum PaO2 = 100 - (age/4) = 100-11 = 89 → 78 is BELOW adjusted normal.

Step 6 – Conclusion

Technically normal acid-base status, but PaO2 78 is mildly below age-adjusted normal — worth noting. Reassess with age, altitude, and FiO2 in mind.

Step 7 – ABG Interpretation Template

1. pH (7.35-7.45); 2. PaCO2 (35-45): respiratory; 3. HCO3 (22-26): metabolic; 4. Compensation (Winters/chronic formulas); 5. Oxygenation (PaO2, A-a gradient).

Appendix: Quick Reference Guide

Compensation Formulas

Metabolic Acidosis: Expected PaCO2 = 1.5 × HCO3 + 8 ± 2 (Winter's Formula)

Metabolic Alkalosis: Expected PaCO2 = 0.7 × (HCO3 – 24) + 40 ± 5

Acute Resp. Acidosis: HCO3 rises 1 mEq/L per 10 mmHg rise in PaCO2

Chronic Resp. Acidosis: HCO3 rises 3.5 mEq/L per 10 mmHg rise in PaCO2

Acute Resp. Alkalosis: HCO3 falls 2 mEq/L per 10 mmHg fall in PaCO2

Chronic Resp. Alkalosis: HCO3 falls 5 mEq/L per 10 mmHg fall in PaCO2

MUDPILES — High Anion Gap Metabolic Acidosis Causes

M	Methanol
U	Uremia (renal failure)
D	Diabetic Ketoacidosis (DKA) / Alcoholic KA / Starvation KA
P	Propylene glycol / Paracetamol (Acetaminophen)
I	Isoniazid / Iron / Infection (sepsis — lactic acidosis)
L	Lactic Acidosis (Type A: hypoperfusion; Type B: drugs/toxins)
E	Ethylene Glycol
S	Salicylates (aspirin overdose)

Delta Ratio (Delta-Delta) Interpretation

Delta Ratio	Interpretation
< 0.4	Normal anion gap metabolic acidosis (+ possible high AG)
0.4 – 1.0	Mixed: High AG + normal AG metabolic acidosis
1.0 – 2.0	Pure high anion gap metabolic acidosis
> 2.0	High AG metabolic acidosis + metabolic alkalosis

This ebook is intended for educational purposes. Always correlate ABG findings with the full clinical picture, patient history, and physical examination before making clinical decisions.