



LUMA EDUCATIONAL APPS

ABG Interpretation Made Simple

A step-by-step course for nurses and medical providers

2026 Edition | Luma Educational Apps

Every Value Explained · Step-by-Step Method · Clinical Scenarios

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Educational resource for nurses and medical providers

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Work through the modules in order the first time. After that, use the Bedside Pocket Card and the Common Clinical Patterns module as quick references at the bedside.

WELCOME

How to Use This Course

This course teaches arterial blood gas (ABG) interpretation from the ground up. Every value on a standard ABG panel is defined, given a normal range, and explained clinically. You will finish able to read an ABG in five steps, name the disorder, judge compensation, and decide what to do next.

Who this course is for

- Bedside nurses (med-surg, telemetry, ED, ICU, PACU, L&D)
- Nursing students and new graduates
- Nurse practitioners, PAs, medical students, and residents
- Respiratory therapists reviewing acid-base fundamentals
- CRNAs and SRNAs who want a clean bedside framework

How each module is built

- **Plain-language explanation** — what the value means at the bedside
- **Normal range and units** — the numbers you must know
- **Why it moves** — the clinical reasons for high or low results
- **What to do** — the next step at the bedside

A note on safety

This is an educational resource. It is not a substitute for clinical judgment, your facility's protocols, or the guidance of a licensed prescriber. Always correlate ABG results with the patient in front of you.

MODULE 1

What an ABG Is and Why It Matters

An arterial blood gas is a small sample of arterial blood analyzed for oxygen, carbon dioxide, and acid–base chemistry. In one panel it tells you how well the patient is ventilating, oxygenating, and buffering.

The three questions an ABG answers

- **Is the patient acidotic or alkalotic?** (pH)
- **Is the problem respiratory, metabolic, or both?** (PaCO_2 and HCO_3^-)
- **Is the patient oxygenating adequately?** (PaO_2 , SaO_2 , P/F, A–a gradient)

When to draw an ABG

- New or worsening respiratory distress
- Altered mental status of unclear cause
- Sepsis, shock, or severe hemodynamic instability
- Suspected metabolic acidosis (DKA, lactic acidosis, toxic ingestion)
- Titrating mechanical ventilation or non-invasive ventilation (BiPAP)
- Post-arrest and post-resuscitation assessment

ABG vs. VBG in one sentence

A venous blood gas (VBG) is easier to obtain and pH and bicarbonate correlate closely with arterial values, but PaCO_2 and PaO_2 from a venous sample cannot be used to judge oxygenation or true respiratory status — use an ABG when those questions matter.

Key takeaway

An ABG is the fastest bedside window into acid–base balance and gas exchange. Learn to read it in five steps and you can act on the result within minutes, not hours.

MODULE 2

Every Value on an ABG, Explained

A standard ABG panel reports the values below. Each one is defined in plain language, given a normal range, and paired with clinical causes of high and low results.

pH — the acidity of the blood

pH is a measure of how acidic or basic arterial blood is. It is tightly regulated because enzymes, electrolyte balance, and cell function depend on it. A low pH is acidemia; a high pH is alkalemia. The body will do almost anything to keep pH near 7.40.

Normal	7.35 – 7.45
Acidemia	< 7.35 (below normal)
Alkalemia	> 7.45 (above normal)
Life threat	< 7.20 or > 7.60 (call provider now)

PaCO₂ — the respiratory component

PaCO₂ is the partial pressure of carbon dioxide dissolved in arterial blood. CO₂ is an acid — every breath is the body's chance to blow it off. PaCO₂ is controlled almost entirely by the lungs, so it is the **respiratory** number on the ABG.

Normal	35 – 45 mmHg
High PaCO₂ (>45)	Hypoventilation — COPD flare, opioid overdose, sedation, respiratory muscle fatigue, obesity hypoventilation, severe asthma late in course
Low PaCO₂ (<35)	Hyperventilation — pain, anxiety, sepsis, PE, early asthma, pregnancy, high altitude, compensation for metabolic acidosis

MODULE 2 · CONTINUED

Every Value on an ABG, Explained

HCO_3^- — the metabolic component

Bicarbonate is the body's main buffer against acid. It is controlled by the kidneys, so HCO_3^- is the **metabolic** number on the ABG. It rises when the kidneys hold on to bicarbonate and falls when bicarbonate is lost or consumed neutralizing acid.

Normal	22 – 26 mEq/L
High HCO_3^- (>26)	Metabolic alkalosis — vomiting, NG suction, diuretics, hypokalemia, over-correction with bicarbonate, chronic respiratory acidosis (renal compensation)
Low HCO_3^- (<22)	Metabolic acidosis — DKA, lactic acidosis (sepsis, shock, ischemia), renal failure, diarrhea, toxic ingestions (methanol, ethylene glycol, salicylates)

PaO_2 — oxygen dissolved in arterial blood

PaO_2 is the partial pressure of oxygen in arterial blood. It reflects how well oxygen is moving from the alveoli into the blood. It is influenced by the amount of oxygen the patient is breathing (FiO_2), altitude, and lung disease.

Normal on room air	80 – 100 mmHg
Mild hypoxemia	60 – 79 mmHg
Moderate hypoxemia	40 – 59 mmHg
Severe hypoxemia	< 40 mmHg (emergency)

SaO_2 — oxygen saturation of hemoglobin

SaO_2 is the percentage of hemoglobin molecules carrying oxygen, measured from the arterial blood sample. It is the most clinically useful oxygen number for most patients.

Normal	95 – 100%
Hypoxemia threshold	< 90% (below the steep part of the oxyhemoglobin curve — small drops in PaO_2 cause large drops in SaO_2)
Life threat	< 85% sustained

MODULE 2 · CONTINUED

Every Value on an ABG, Explained

Base excess (BE) — how much acid or base is out of balance

Base excess estimates the amount of acid or base that would need to be added to return the blood to a normal pH. A positive number suggests too much base (metabolic alkalosis); a negative number suggests too much acid (metabolic acidosis). It reinforces what HCO_3^- tells you.

Normal	−2 to +2 mEq/L
BE > +2	Metabolic alkalosis (base excess)
BE < −2	Metabolic acidosis (base deficit)

Lactate — a marker of tissue oxygen delivery

Lactate rises when tissues do not have enough oxygen for normal aerobic metabolism (shock, sepsis, ischemia, seizure, severe hypoxemia) or when clearance is impaired (liver failure, some medications). Not officially part of every ABG panel, but nearly always reported alongside.

Normal	0.5 – 2.0 mmol/L
Elevated	> 2 mmol/L — investigate perfusion, oxygenation, sepsis, and organ function
Severe	> 4 mmol/L — resuscitate aggressively; associated with high mortality in sepsis

Electrolytes reported with an ABG

Modern blood gas analyzers usually report a small panel of electrolytes and other values with each ABG. You will not always act on them directly, but they help interpret the acid–base picture.

Value	Normal and clinical meaning
Na^+ (sodium)	135–145 mEq/L. Needed to calculate anion gap.
K^+ (potassium)	3.5–5.0 mEq/L. Shifts with acidosis and alkalosis and drives arrhythmia risk.
Cl^- (chloride)	98–108 mEq/L. Needed for anion gap; often high in non-gap acidoses.

Value	Normal and clinical meaning
iCa ²⁺ (ionized calcium)	1.15–1.32 mmol/L. Drops with rapid transfusion, sepsis, and alkalosis.
Glucose	70–140 mg/dL (fasting/random). Critical in DKA, HHS, and hypoglycemia.
Hgb / Hct	Anemia limits oxygen carrying capacity even when PaO ₂ looks normal.
COHb (carboxyhemoglobin)	< 3% non-smoker, < 10% smoker. Elevated in CO poisoning; pulse ox is falsely normal.
MetHb (methemoglobin)	< 2%. Elevated in benzocaine, dapsone, nitrates; pulse ox reads ~85%.

MODULE 3

Normal Values Cheat Sheet

Memorize these numbers. They are the anchor for every ABG you will ever read.

Core acid–base values

Value	Normal range
pH	7.35 – 7.45
PaCO ₂	35 – 45 mmHg
HCO ₃ [–]	22 – 26 mEq/L
Base excess	–2 to +2 mEq/L

Oxygenation values

Value	Normal range
PaO ₂ (room air)	80 – 100 mmHg
SaO ₂	95 – 100%
P/F ratio (PaO ₂ /FiO ₂)	> 400 normal; < 300 acute lung injury; < 200 ARDS
A–a gradient	Roughly Age/4 + 4 on room air (younger < 15 mmHg)

Common companion labs

Value	Normal range
Lactate	0.5 – 2.0 mmol/L
Sodium (Na^+)	135 – 145 mEq/L
Potassium (K^+)	3.5 – 5.0 mEq/L
Chloride (Cl^-)	98 – 108 mEq/L
Anion gap	8 – 12 mEq/L (with normal albumin)
Ionized calcium	1.15 – 1.32 mmol/L

MODULE 4

The 5-Step ABG Method

Use this exact order every time. Once it is muscle memory you can interpret any ABG in about a minute.

Step 1 • Look at the pH

Is it acidemic (< 7.35), alkalemic (> 7.45), or normal ($7.35\text{--}7.45$)? Write acid, base, or normal on the sheet before moving on.

Step 2 • Look at the PaCO_2

Is it high (> 45), low (< 35), or normal? Remember: CO_2 is an acid. High CO_2 pushes pH down; low CO_2 pushes pH up.

Step 3 • Look at the HCO_3^-

Is it high (> 26), low (< 22), or normal? Bicarb is a base. High bicarb pushes pH up; low bicarb pushes pH down.

Step 4 • Match the primary disorder

Whichever value (PaCO_2 or HCO_3^-) moves in the same direction as the pH abnormality is the **primary problem**. Respiratory if it is PaCO_2 ; metabolic if it is bicarb.

Step 5 • Assess compensation and oxygenation

Is the opposite system moving to correct the pH? Then compensation is present. Finally, check PaO_2 and SaO_2 to judge oxygenation.

MODULE 4 · CONTINUED

The 5–Step ABG Method

The ROME memory tool

Respiratory **O**pposite, **M**etabolic **E**qual. In a respiratory problem, the pH and PaCO_2 move in opposite directions. In a metabolic problem, the pH and HCO_3^- move in the same direction.

pH	PaCO_2	HCO_3^-	Primary disorder
↓ (<7.35)	↑ (>45)	normal or ↑ (compensating)	Respiratory acidosis
↑ (>7.45)	↓ (<35)	normal or ↓ (compensating)	Respiratory alkalosis
↓ (<7.35)	normal or ↓ (compensating)	↓ (<22)	Metabolic acidosis
↑ (>7.45)	normal or ↑ (compensating)	↑ (>26)	Metabolic alkalosis

Worked example

ABG: pH 7.28 | PaCO_2 60 | HCO_3^- 26 | PaO_2 62.

- **Step 1:** pH 7.28 → acidemia.
- **Step 2:** PaCO_2 60 → high (acid).
- **Step 3:** HCO_3^- 26 → normal.
- **Step 4:** PaCO_2 matches the acidemia — **respiratory acidosis**.
- **Step 5:** Bicarb has not risen yet, so this is **acute, uncompensated**. PaO_2 62 is mild hypoxemia — support ventilation and oxygenation.

MODULE 5

The Four Primary Disorders

Each disorder has a fingerprint: a direction of pH change, the value that drives it, and a short list of causes and clinical clues.

Respiratory acidosis · the lungs are not blowing off CO₂

pH	< 7.35
Driving value	PaCO ₂ > 45 mmHg
Common causes	COPD exacerbation, asthma late in course, opioid or sedative overdose, neuromuscular disease (myasthenia, Guillain-Barré, ALS), obesity hypoventilation, chest wall trauma, oversedation, airway obstruction, inadequate ventilator settings.
Clinical clues	Somnolence, headache, flushed skin, tachycardia, tremor, CO ₂ narcosis at very high levels. Pulse ox may be near normal on supplemental oxygen even while CO ₂ climbs.

Respiratory alkalosis · the lungs are blowing off too much CO₂

pH	> 7.45
Driving value	PaCO ₂ < 35 mmHg
Common causes	Pain, anxiety, fever, sepsis (early), pulmonary embolism, early asthma or pneumonia, high altitude, pregnancy, salicylate toxicity, mechanical ventilator set too aggressively.
Clinical clues	Dizziness, perioral or fingertip tingling, carpopedal spasm, chest tightness, palpitations. Ionized calcium falls with alkalosis, which explains the paresthesias.

MODULE 5 · CONTINUED

The Four Primary Disorders

Metabolic acidosis · too much acid or not enough base

pH	< 7.35
Driving value	$\text{HCO}_3^- < 22 \text{ mEq/L}$
Common causes	High anion gap: DKA, lactic acidosis (sepsis, shock, ischemia), renal failure (uremia), toxic ingestions (methanol, ethylene glycol, salicylates, propylene glycol). Normal anion gap: diarrhea, renal tubular acidosis, ureteral diversion, early kidney disease.
Clinical clues	Deep, rapid breathing (Kussmaul respirations) as the lungs try to blow off CO_2 . Confusion, hypotension, arrhythmia, hyperkalemia. In DKA look for fruity breath and dehydration.

Metabolic alkalosis · too much base or too little acid

pH	> 7.45
Driving value	$\text{HCO}_3^- > 26 \text{ mEq/L}$
Common causes	Vomiting or continuous NG suction (loss of HCl), loop or thiazide diuretics, hypokalemia, hypochloremia, over-correction with sodium bicarbonate, massive citrate load from transfusion, Cushing syndrome, primary hyperaldosteronism, milk-alkali syndrome.
Clinical clues	Slow, shallow breathing (respiratory compensation), muscle cramps, weakness, arrhythmias from hypokalemia, seizures at severe pH elevation, tetany from low ionized calcium.

Bedside anchor

If pH and PaCO_2 move opposite ways, think **respiratory**. If pH and HCO_3^- move the same way, think **metabolic**.

MODULE 6

Compensation, Simply

The body always tries to bring pH back to 7.40. The lungs respond in minutes; the kidneys take hours to days. Compensation never overshoots — the pH remains on the abnormal side, just closer to normal.

Three stages of compensation

Stage	What you see on the ABG
Uncompensated	pH is abnormal. The opposite system (PaCO_2 if metabolic, HCO_3^- if respiratory) is still normal. Usually acute.
Partially compensated	pH is still abnormal, but the opposite system has moved in the correcting direction. The body is responding but has not caught up.
Fully compensated	pH has returned to the normal range (7.35–7.45). Both PaCO_2 and HCO_3^- are abnormal, moved in opposite directions.

Which side is primary when pH is normal?

In a fully compensated gas, pH lands inside 7.35–7.45. Split the range at 7.40 to find the primary problem:

- **pH 7.35 – 7.39** → the primary disorder is an **acidosis** (respiratory or metabolic).
- **pH 7.41 – 7.45** → the primary disorder is an **alkalosis** (respiratory or metabolic).
- Then look at which value (PaCO_2 or HCO_3^-) is abnormal in that same direction — that is the primary system.

A worked compensated example

ABG: pH 7.36 | PaCO_2 60 | HCO_3^- 33

- pH is on the acid side of 7.40 → the primary problem is an **acidosis**.
- PaCO_2 60 is high (acid) → the primary system is **respiratory**.
- HCO_3^- 33 is high (base) → the kidneys are compensating.
- Interpretation: **fully compensated respiratory acidosis**, typical of chronic COPD.

MODULE 7

Anion Gap and Mixed Disorders

The anion gap sorts metabolic acidosis into two groups: acids the body is making or accumulating (high gap) versus bicarbonate lost through the gut or kidney (normal gap). It sharpens the differential and points to the treatment.

How to calculate

Anion gap formula

$$\text{Anion gap} = \text{Na}^+ - (\text{Cl}^- + \text{HCO}_3^-)$$

Normal: 8 – 12 mEq/L. Correct for low albumin: add 2.5 mEq/L for every 1 g/dL that albumin is below 4.

High anion gap acidosis — remember MUDPILES

Letter	Cause
M	Methanol
U	Uremia (renal failure)
D	Diabetic ketoacidosis (also alcoholic and starvation ketoacidosis)
P	Propylene glycol (found in some IV drug diluents)
I	Iron, Isoniazid, Ibuprofen (toxic doses)
L	Lactic acidosis (sepsis, shock, ischemia, metformin)
E	Ethylene glycol (antifreeze)
S	Salicylates (aspirin toxicity)

Normal anion gap acidosis — remember HARDUP

Letter	Cause
H	Hyperalimentation (TPN)
A	Acetazolamide, Addison disease
R	Renal tubular acidosis
D	Diarrhea
U	Ureteral diversion
P	Pancreatic fistula

MODULE 7 · CONTINUED

Anion Gap and Mixed Disorders

Mixed disorders — when two problems overlap

A mixed disorder exists when the compensation is bigger than expected, or when the pH is normal but both PaCO_2 and HCO_3^- are grossly abnormal in opposite directions. Common examples:

- **Salicylate toxicity** — respiratory alkalosis (drug-induced hyperventilation) + high-gap metabolic acidosis.
- **Vomiting in DKA** — metabolic acidosis (ketones) + metabolic alkalosis (HCl loss); anion gap reveals the acidosis.
- **Cardiac arrest** — respiratory acidosis (poor ventilation) + lactic acidosis (poor perfusion).
- **Sepsis** — respiratory alkalosis (early hyperventilation) + lactic acidosis as shock develops.

Bedside anchor

Always calculate the anion gap on every metabolic acidosis. If the gap is elevated, the differential shifts immediately to MUDPILES and the treatment path (insulin, fluids, dialysis, antidote) changes with it.

MODULE 8

Oxygenation: PaO_2 , SaO_2 , P/F Ratio, A–a Gradient

Acid–base tells you how the patient is buffering. Oxygenation tells you whether the tissues are getting the oxygen they need. Both belong on every ABG interpretation.

PaO_2 vs SaO_2 — do not confuse them

PaO_2	SaO_2
Partial pressure of dissolved O_2 (mmHg).	Percent of hemoglobin carrying O_2 (%).
Reflects how well O_2 crosses the alveolar–capillary membrane.	Reflects how much O_2 the blood is actually delivering.
Small changes matter above 60 mmHg.	Small changes matter below 90%.

P/F ratio — grading oxygenation quickly

$\text{P/F} = \text{PaO}_2 \div \text{FiO}_2$ (expressed as a decimal). It corrects PaO_2 for the amount of oxygen the patient is receiving. Especially useful in ICU and on ventilators.

P/F ratio	Interpretation
> 400	Normal oxygenation
300 – 400	Mild impairment
200 – 300	Acute lung injury / mild ARDS
100 – 200	Moderate ARDS
< 100	Severe ARDS

A-a gradient — is the lung the problem?

The alveolar–arterial (A–a) oxygen gradient shows how efficiently oxygen moves from the alveolus into the blood. A widened gradient points to a lung problem (shunt, V/Q mismatch, diffusion defect). A normal gradient in a hypoxic patient points to hypoventilation or low inspired oxygen.

Estimated normal	Roughly $\text{Age}/4 + 4$ (on room air). Younger patients under 15 mmHg.
Widened gradient	Pneumonia, PE, ARDS, pulmonary edema, atelectasis, shunt
Normal gradient	Hypoventilation (opioids, sedation, neuromuscular disease), high altitude, low FiO_2

MODULE 9

Common Clinical Patterns

Pattern recognition beats memorization. Here are the pictures you will see most often.

COPD exacerbation

ABG: pH 7.30 | PaCO₂ 65 | HCO₃⁻ 32 | PaO₂ 55

Partially compensated respiratory acidosis with hypoxemia. Chronic CO₂ retention, kidneys have raised bicarb.

Opioid overdose

ABG: pH 7.22 | PaCO₂ 68 | HCO₃⁻ 24 | PaO₂ 58

Acute uncompensated respiratory acidosis. Reverse with naloxone and support ventilation.

Anxiety / pain hyperventilation

ABG: pH 7.50 | PaCO₂ 28 | HCO₃⁻ 24 | PaO₂ 99

Acute uncompensated respiratory alkalosis. Treat the cause; slow the breathing.

Diabetic ketoacidosis

ABG: pH 7.15 | PaCO₂ 22 | HCO₃⁻ 8 | Na 140 | Cl 100 | Glucose 480

High anion gap metabolic acidosis (gap 32) with respiratory compensation. Fluids, insulin, electrolytes.

Septic shock

ABG: pH 7.24 | PaCO₂ 28 | HCO₃⁻ 12 | Lactate 6.5

High anion gap metabolic (lactic) acidosis with respiratory compensation. Resuscitate, cultures, antibiotics, source control.

Prolonged vomiting

ABG: pH 7.52 | PaCO₂ 46 | HCO₃⁻ 34 | K 3.1 | Cl 92

Partially compensated metabolic alkalosis with hypokalemia and hypochloremia. Give IV normal saline and replace potassium.

Salicylate toxicity

ABG: pH 7.44 | PaCO₂ 20 | HCO₃⁻ 14 | Anion gap 22

Mixed respiratory alkalosis and high-gap metabolic acidosis. Classic salicylate pattern.

Cardiac arrest

ABG: pH 7.05 | PaCO₂ 60 | HCO₃⁻ 14 | Lactate 9

Mixed respiratory and metabolic acidosis. High-quality CPR, ventilation, and reversible-cause work-up.

MODULE 10

Bedside Actions by Disorder

Interpretation is only half the job. These are the immediate bedside moves that stabilize the patient while the underlying cause is treated. Always follow facility protocol and prescriber orders.

Respiratory acidosis

- Sit the patient up; encourage deep breathing and coughing if awake.
- Suction airway secretions; check for a compromised airway.
- Support ventilation — bag-valve-mask, BiPAP, or intubation if severe or trending down.
- Reverse contributing agents — naloxone for opioids, flumazenil (rarely) for benzodiazepines.
- Recheck ABG within 20–30 minutes of any intervention.

Respiratory alkalosis

- Treat the underlying cause — pain, anxiety, sepsis, PE, fever.
- Coach breathing to a slower rate; avoid rebreathing bags without an order.
- Correct hypoxemia if present — it drives hyperventilation.
- Recheck ABG once the cause is addressed.

Metabolic acidosis

- Restore perfusion — IV fluids for hypovolemia, vasopressors as needed for shock.
- Treat the specific cause — insulin and fluids for DKA, antidote for toxic ingestion, dialysis for uremia.
- Monitor potassium closely; acidosis often masks a low total-body potassium.
- IV bicarbonate is reserved for severe acidemia ($\text{pH} < 7.10$) or specific ingestions.

Metabolic alkalosis

- Give IV normal saline for chloride-responsive causes (vomiting, NG suction, diuretics).
- Replace potassium and magnesium as ordered — arrhythmia risk rises with alkalosis and hypokalemia.
- Reduce ongoing acid losses — treat nausea, hold offending diuretics if appropriate.
- Recheck ABG and basic metabolic panel after resuscitation.

MODULE 11

Practice Cases with Full Explanations

Work through each case using the 5-Step Method before reading the explanation. Cover the interpretation with your hand if it helps.

Case 1 — 68-year-old man with COPD

Scenario. Presents with worsening dyspnea over 3 days. On 2 L nasal cannula.

ABG. pH 7.34 | PaCO₂ 58 | HCO₃⁻ 30 | PaO₂ 60 | SaO₂ 90%

Interpretation and plan. Partially compensated respiratory acidosis with mild hypoxemia. Chronic CO₂ retention (bicarb 30 is elevated from renal compensation). Consider BiPAP, bronchodilators, steroids, and titrate oxygen carefully to target SaO₂ 88–92% to avoid worsening hypercarbia.

Case 2 — 24-year-old woman with type 1 diabetes

Scenario. Nausea, abdominal pain, deep and rapid respirations. Glucose 520 mg/dL.

ABG. pH 7.10 | PaCO₂ 18 | HCO₃⁻ 6 | Na 138 | Cl 100 | K 5.4

Interpretation and plan. High anion gap metabolic acidosis (gap = 138 – (100 + 6) = 32) with respiratory compensation. Consistent with DKA. Initiate IV fluids, insulin infusion per protocol, and correct potassium before insulin drops it further.

Case 3 — 55-year-old man 30 minutes post-op

Scenario. Slow shallow breathing, RR 6, drowsy. Received IV opioid in PACU.

ABG. pH 7.22 | PaCO₂ 68 | HCO₃⁻ 25 | PaO₂ 60

Interpretation and plan. Acute uncompensated respiratory acidosis. Consistent with opioid-induced hypoventilation. Stimulate, support ventilation with bag-valve-mask, give naloxone per order, monitor closely.

MODULE 11 • CONTINUED

Practice Cases with Full Explanations

Case 4 — 40-year-old woman with severe vomiting

Scenario. Five days of vomiting from bowel obstruction. Weak, cramping.

ABG. pH 7.52 | PaCO₂ 48 | HCO₃⁻ 36 | K 3.0 | Cl 90

Interpretation and plan. Partially compensated metabolic alkalosis (loss of HCl in vomit). Hypokalemia and hypochloremia sustain it. Give IV normal saline, replace potassium, treat nausea, and address the obstruction.

Case 5 — 70-year-old woman with pneumonia and sepsis

Scenario. Fever, tachypnea, hypotension, warm extremities.

ABG. pH 7.28 | PaCO₂ 24 | HCO₃⁻ 12 | Lactate 5.2

Interpretation and plan. High anion gap metabolic (lactic) acidosis with respiratory compensation. Consistent with septic shock. Give IV fluids, blood cultures, broad-spectrum antibiotics within 1 hour, and start vasopressors if hypotension persists.

Case 6 — 30-year-old with panic attack

Scenario. Sudden shortness of breath, chest tightness, tingling fingers.

ABG. pH 7.52 | PaCO₂ 26 | HCO₃⁻ 22 | PaO₂ 105

Interpretation and plan. Acute uncompensated respiratory alkalosis. Reassure, coach slow breathing, evaluate for underlying causes like PE if risk factors present, and treat anxiety.

MODULE 11 · CONTINUED

Practice Cases with Full Explanations

Case 7 — 62-year-old on chronic diuretics

Scenario. Weakness and confusion. Long-standing furosemide use for heart failure.

ABG. pH 7.48 | PaCO₂ 44 | HCO₃⁻ 34 | K 2.9

Interpretation and plan. Metabolic alkalosis with hypokalemia (diuretic-induced). Replace potassium and magnesium, reassess diuretic regimen with the prescriber, and monitor for arrhythmias.

Case 8 — 45-year-old after aspirin overdose

Scenario. Tinnitus, hyperventilation, nausea. Salicylate level pending.

ABG. pH 7.42 | PaCO₂ 20 | HCO₃⁻ 13 | Na 140 | Cl 100

Interpretation and plan. Mixed disorder: respiratory alkalosis (salicylate stimulates the medullary respiratory center) plus high-gap metabolic acidosis (gap 27). Classic salicylate toxicity. IV fluids, urine alkalinization with bicarbonate per toxicology, and dialysis for severe cases.

QUICK REFERENCE

Bedside Pocket Card

Print this page and slip it in a badge holder. Every core value and step on one sheet.

Normal values

Value	Normal range
pH	7.35 – 7.45
PaCO ₂	35 – 45 mmHg
HCO ₃ ⁻	22 – 26 mEq/L
PaO ₂	80 – 100 mmHg
SaO ₂	95 – 100%
Base excess	-2 to +2 mEq/L
Anion gap	8 – 12 mEq/L
Lactate	0.5 – 2.0 mmol/L

The 5-step method

- **Step 1.** pH — acid, base, or normal?
- **Step 2.** PaCO₂ — high, low, or normal?
- **Step 3.** HCO₃⁻ — high, low, or normal?
- **Step 4.** Which value moves with the pH? That is the primary disorder (respiratory or metabolic).
- **Step 5.** Is the opposite system compensating? Then check PaO₂ and SaO₂.

ROME reminder

Respiratory **O**pposite · Metabolic **E**qual (pH vs. driving value).

GLOSSARY

Terms Used in This Course

Acidemia	A blood pH below 7.35.
Alkalemia	A blood pH above 7.45.
Anion gap	$\text{Na}^+ - (\text{Cl}^- + \text{HCO}_3^-)$. Normal 8–12 mEq/L. Rises when unmeasured acids accumulate.
Base excess	The estimated amount of acid or base needed to return the blood to a normal pH.
Compensation	The body's response by the opposite system (lungs or kidneys) to bring pH toward normal.
FiO₂	Fraction of inspired oxygen (21% on room air, up to 100% on non-rebreather or ventilator).
HCO₃⁻	Bicarbonate — the primary blood buffer, regulated by the kidneys.
Hypercapnia / hypercarbia	Elevated arterial CO ₂ (PaCO ₂ > 45 mmHg).
Hypoxemia	Low arterial oxygen content (PaO ₂ < 80 mmHg or SaO ₂ < 95%).
Lactate	Byproduct of anaerobic metabolism; a marker of impaired oxygen delivery to tissues.
P/F ratio	PaO ₂ divided by FiO ₂ . Screens for acute lung injury and ARDS.
PaCO₂	Partial pressure of arterial carbon dioxide; the respiratory value on the ABG.

PaO₂	Partial pressure of dissolved arterial oxygen.
pH	A measure of acidity of the blood. Narrowly regulated between 7.35 and 7.45.
ROME	R espiratory O pposite, M etabolic E qual — a memory tool for matching the primary disorder.
SaO₂	Percent of hemoglobin bound to oxygen (from an arterial sample).
Shunt	Blood that passes through the lungs without picking up oxygen (e.g., atelectasis, pneumonia).
VBG	Venous blood gas — a peripheral vein sample; pH and HCO ₃ ⁻ approximate arterial values.

REFERENCES

Suggested Sources for Deeper Study

The following widely used references informed the normal values, formulas, and clinical patterns in this course. Consult them and your facility's protocols for any clinical decision.

American Association of Critical-Care Nurses (AACN). Core Curriculum for High Acuity, Progressive, and Critical Care Nursing.

Standard nursing reference for acid–base and oxygenation assessment.

Marino P. The ICU Book, 4th ed. Wolters Kluwer.

Deep dive into ABG interpretation, oxygen transport, and mechanical ventilation.

Berne & Levy Physiology, latest ed. Elsevier.

Physiology of the buffer system, renal and respiratory compensation.

Adrogué HJ, Madias NE. Management of Life-Threatening Acid–Base Disorders. New England Journal of Medicine.

Classic clinical review of primary acid–base disorders and mixed presentations.

KDIGO and ARDS Berlin Definition (JAMA).

Reference thresholds for P/F ratio and ARDS grading used in Module 8.

Institute for Safe Medication Practices (ISMP) and manufacturer prescribing information.

Guidance on naloxone, insulin infusions, sodium bicarbonate, and other agents referenced in bedside actions.

Disclaimer

This course is an educational resource created through Luma Educational Apps. It is not a substitute for individualized clinical judgment, prescriber orders, facility policy, or accredited continuing education. Always verify medication doses, formulas, and interventions against current authoritative sources.