Blood Gas Analysis

WWW.RN.ORG®
Reviewed July 2024, Expires July 2026
Provider Information and Specifics available on our Website

By Wanda Lockwood, RN, BA, MA

Purpose

This course describes blood gas analysis, including the types of tests, expected results, sampling, nursing considerations, and acid-base imbalances.

Goals

Upon completion of this course, one should be able to do the following:

- Describe normal range of values for at least 6 components of blood gas analysis.
- Explain the relationship of pH to acidosis and alkalosis.
- Explain how the partial pressure of oxygen (pO2) relates to oxygen bound in hemoglobin.
- Explain how the partial pressure of carbon dioxide (pCO_2) relates to respiratory function and production of carbonic acid.
- Describe how saturated oxygen ($SO_{2)}$ relates to the oxygen-hemoglobin dissociation curve.
- Explain the ratio of carbonic acid to bicarbonate (HCO_3^-).
- Explain base excess and list a respiratory acid and at least 2 metabolic acids.
- Describe at least 6 factors that can interfere with arterial blood gas values.
- Describe procedures for obtaining arterial, venous, capillary, and cord blood samples.
- Explain 3 mechanisms by which the body regulates acid-base balance.
- Describe 4 primary forms of acid-base imbalance, including causes and symptoms.

Introduction

Blood gas analysis is done to determine the acid-base balance in the blood. The acid-base balance is maintained by the respiratory, cardiovascular, and renal systems. The body, under normal circumstances, can maintain a balance between the acids produced as part of metabolism and the bases necessary to neutralize and promote the excretion of acids. Renal, respiratory, and metabolic disorders, such as kidney failure, COPD, and diabetes, may lead to acid-base imbalances. Additionally, loss of body fluids through vomiting or diarrhea can

lead to acid-base imbalances and fluid and electrolyte imbalances. The most commonly used measures for arterial blood gas (ABG) analysis include hydrogen ion concentration (pH), partial pressure of oxygen in the blood (pO_2), partial pressure of carbon dioxide in the blood (pCO_2), bicarbonate (HCO_3^-), oxygen saturation (SO_2), and base excess (BE) or (base deficit (BD).

pH

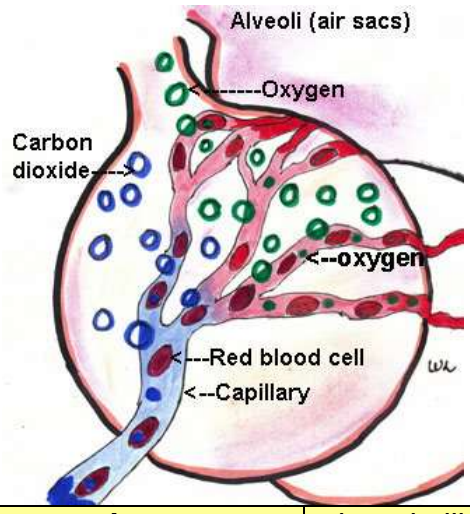
The hydrogen (H^+) ion concentration in the blood determines whether it is acid or base. The more hydrogen there is, the more acidotic the blood. Chemical solutions range in pH from 1 to 14 with a neutral pH being 7. A pH of less than 7 is acidotic and more than 7 is alkalotic. Blood is normally slightly alkalotic. The pH is expressed as a negative logarithm, meaning that the lower the number, the higher the concentration of ions. Thus, a normal pH ranges from 7.35 to 7.45 and as that number falls below 7.35, the concentration of hydrogen ions increases and the blood becomes more acidotic. As the pH number increases, the concentration of hydrogen ions decreases, causing the blood to become more alkalotic. Since the normal blood is alkalotic, a decrease in pH below 7.35 results in the *condition* of acidosis even though the acid-balance may remain slightly alkalotic (above 7 pH). The normal pH is maintained by a ratio of 1 part carbonic acid to 20 parts bicarbonate. Capillary blood provides an adequate sample for pH measurement.

<6.8	<7.35	7.35 – 7.45	>7.45	>7.8
Death	Acidosis	Normal pH	Alkalosis	**Death**

pO_2/paO_2

PO_2	**Arterial (paO_2)**	**Venous (pvO_2)**	**Capillary**
Cord blood	8 – 24 mm Hg	17 – 41 mm Hg	--
Adult/child	80 – 95 mm Hg	20 – 49 mm Hg	80 – 95 mm Hg

The partial pressure of oxygen (pO_2) in the blood, usually expressed as partial pressure in arterial blood (paO_2), represents the oxygen dissolved in the plasma. Unless otherwise specified, pO_2 and paO_2 both are used to represent arterial sampling. Oxygen enters the lungs as a gas, but when the oxygen diffuses into the capillaries at the alveolar level, it changes form and becomes a solution. This oxygen in solution exerts pressure. Some of the oxygen remains as free oxygen in the plasma while some oxygen binds to hemoglobin. The pO_2 measures the pressure of only the free oxygen in solution. PO_2 is measured in mm Hg and doesn't provide complete information about how much oxygen is in the blood because it doesn't include the oxygen bound to hemoglobin. Venous blood has picked up carbon dioxide and lost much of its oxygen so the pvO_2 pressure is lower than the paO_2 pressure. Capillary blood can provide adequate information about the partial pressure of oxygen but is usually only used to exclude hypoxia. Venous sampling is not as successful as arterial sampling, so it should not be relied up for accuracy.



Drugs that increase pO_2	Theophylline. Urokinase.
Drugs that decrease pO_2	Althesin. Barbiturates. Granulocyte-macrophage colony-stimulating factor. Meperidine. Isoproterenol.

pCO_2/$paCO_2$

PCO_2	Arterial ($paCO_2$)	Venous ($pvCO_2$)	Capillary
Cord blood	32 – 66 mm Hg	27 – 49 mm Hg	--
Adult/child	35 – 45 mm Hg	41 – 51 mm Hg	26 – 41 mm Hg

The partial pressure of carbon dioxide (pCO_2 or $paCO_2$) is important for the evaluation of ventilation as this level is controlled primarily by the lungs. The $paCO_2$ is usually evaluated after the pH to determine if respiratory function is adequate. Capillary blood provides an adequate sample for pCO_2 testing.

<35 mm Hg	35 -45 mm Hg	>45 mm Hg
Increases pH and increases alkalinity.	Normal	Decreases pH and increases acidity

Carbon dioxide forms carbonic acid when it dissolves in blood. Note that a 10 mm Hg increase in $paCO_2$ associated with acute respiratory acidosis lowers the pH about 0.08 while with chronic respiratory acidosis the pH lowers only 0.03. Conversely, a 10 mm Hg decrease in $paCO_2$ associated with acute respiratory alkalosis increases the pH about 0.08 while chronic respiratory alkalosis increases the pH only about 0.03.

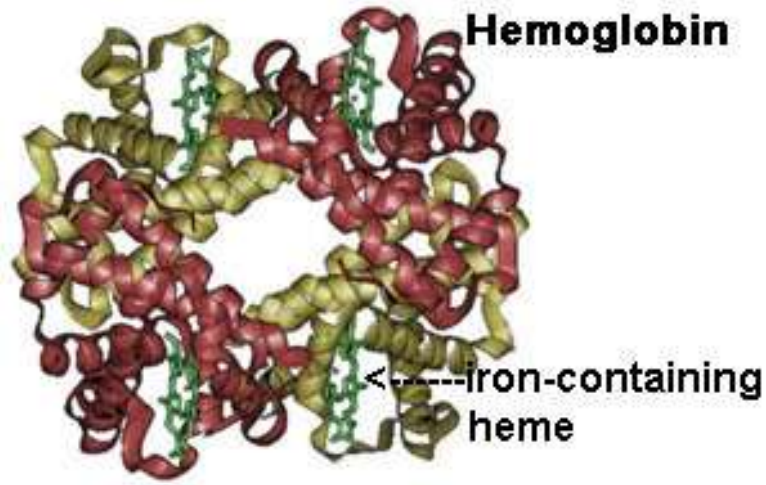
Drugs that increase pCO_2	Acetylsalicylic acid. Aldosterone bicarbonate. Carbenicillin, Corticosteroids. Dexamethasone. Ethacrynic acid. Laxatives (used chronically).

	X-ray contrast agents. Carbenoxolone.
Drugs that decrease pCO_2	Acetazolamide. Acetylsalicylic acid. Ethamivan, Neuromuscular relaxants (2ndry to post-operative hyperventilation). NSD 3004. Theophylline. Tromethamine. Xylitol.

Oxygen saturation (sO_2/saO_2)

SO_2	Arterial (SaO_2)	Venous (SvO_2)	Capillary
Cord blood	40 – 90%	40 – 70%	--
Adult/child	95 – 99%	70 – 75%	95 – 98%

Each molecule of hemoglobin contains four iron-containing heme sites to which oxygen can bind. As some of the free oxygen in solution binds to the heme, the hemoglobin becomes "saturated" with oxygen.

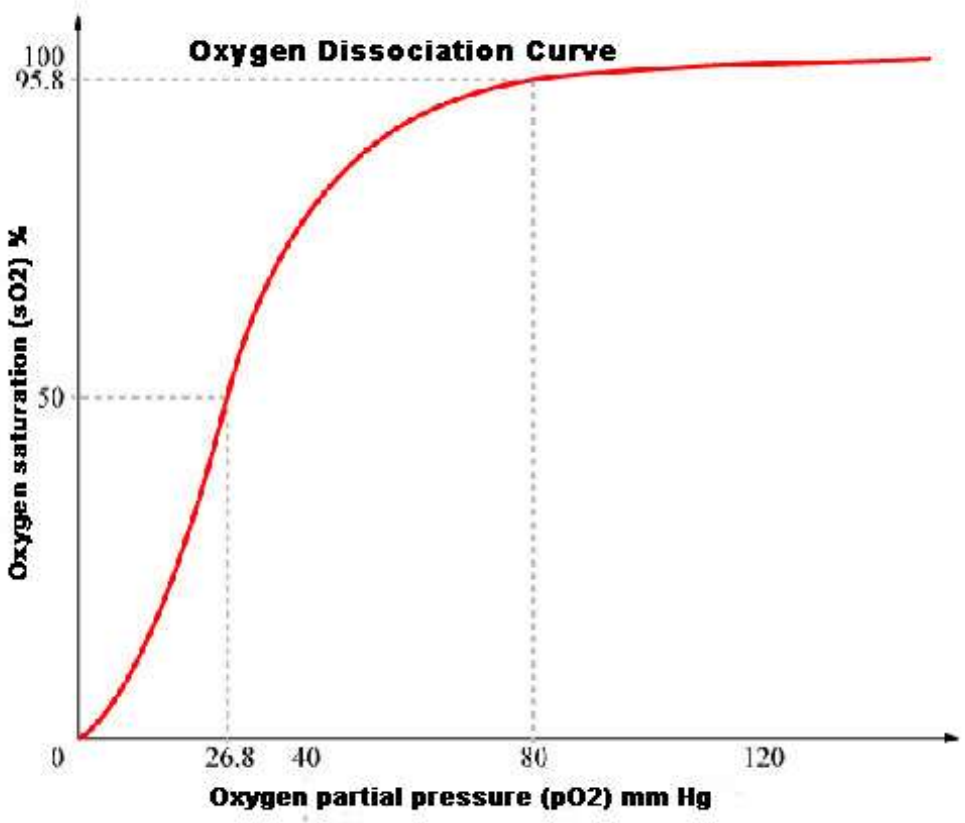


Wikimedia Commons, CC 3.0.

The saO_2 level is calculated as a percentage of the total heme sites saturated with oxygen. So, if there are 100 available heme sites and 97 have bound to oxygen, then the saO_2 level would be 97%. While the saO_2 level gives information about the effectiveness of the hemoglobin in binding to oxygen, it doesn't provide adequate information about the total amount of oxygen, because this is dependent on the hemoglobin count. If hemoglobin is very low, even a saturation of 100% may not provide adequate oxygenation to the tissues. The oxygen saturation level is influenced by the paO_2 level because if the paO_2 level falls significantly, there is not enough oxygen in solution to bind to available heme.

SO_2 is based on 100% normal hemoglobin A. Hemoglobin can bind 1.36 – 1. 37 mL per gram of hemoglobin, markedly increasing the content of oxygen in the blood. If hemoglobin S (sickle cell) is present, then there will be decreased oxygen binding, causing a shift in the right of the oxygen dissociation curve (lower saturation per given pO_2). Both fetal hemoglobin and methemoglobin can cause a shift to the left (higher saturation per given pO_2) with increased oxygen binding.

The oxygen-hemoglobin dissociation curve shows the relationship between sO_2 and pO_2. At 80 - 90 pO_2, hemoglobin is fully saturated so increased pO_2 won't further increase saturation. The critical point for pO_2 is 60 because below this point, there is a marked decrease in saturation, as demonstrated by the curve. PO_2 of 60 usually corresponds to sO_2 of 91%, the "ICU" point.



Diberri, Wikimedia Commons, CC 3.0

Bicarbonate (HCO_3^-)

HCO_3^-	Arterial	Venous	Capillary
Cord blood	17 – 24 mEq/L	17 – 24 mEq/L	--
Adult/child	18 – 23 mEq/L	24 – 28 mEq/L	18 – 23 mEq/L

Bicarbonate is produced by a reaction of carbon dioxide and water. The $paCO_2$ level can be used to indirectly calculate both the carbonic and the bicarbonate levels. First, the paO_2 is used to calculate the carbonic level: pCO_2 X 3% = carbonic acid. Thus, if the $paCO_2$ is 40, the carbonic acid level is 1.2 (40 X .03).

Since the carbonic acid to HCO_3^- ratio is 1:20, then the HCO_3^- level can be estimated as 1.2 (carbonic acid) X 20 = 24. The HCO_3^- level can also be measured directly or estimated from the total carbon dioxide (tCO_2).

Drugs that increase HCO_3^-	Acetylsalicylic acid (initially). Antacids. Carbenicillin. Carbenoxolone. Ethacrynic acid. Glycyrrhiza (licorice). Laxatives. Mafenide. Sodium bicarbonate.
Drugs that decrease HCO_3^-	Acetazolamide. Acetylsalicylic acid (high doses or chronic use). Citrates. Dimethadione. Ether. Ethylene glycol. Fluorides. Mercury compounds found in laxatives. Methylenedioxyamphetamine. Paraldehyde. Xylitol.

Total carbon dioxide (tCO_2)

tCO_2	Arterial	Venous
Cord blood	13 – 22 mEq/L	14 – 22 mEq/L
Adult/child	22 – 29 mEq/L	25 – 30 mEq/L

While the pCO_2 only measures the pressure of dissolved CO_2 in solution, total carbon dioxide (tCO_2) measures the total of all forms of carbon dioxide, including carbonic acid, carbonate, and bicarbonate. During the test for tCO_2, an acid is added to the serum sample. This acid converts all of the carbonic acid, carbonate, and bicarbonate into water and CO_2 so that the CO_2 can be measured.

Base excess (BE)

Base excess	Arterial
Cord blood	(-10) – (-2) mEq/L
Adult/child	(-2) – (+3) mEq/L

Base excess is the amount of acid needed to maintain the pH at a normal level with CO_2 at standard value. Medical calculators and formulas (Henderson-Hasselbalch equation) are used to calculate the base excess based on the pH and the $paCO_2$. Acids are produced by the body's metabolism. Acids include

respiratory acid (carbonic acid derived from CO_2) and metabolic acids (all other). About 288 liters of carbon dioxide (12 moles) are produced daily and eliminated through the lungs. Metabolic acids are produced in much smaller amounts (0.1 mole) daily. Metabolic acids are excreted by the kidneys or metabolized by the liver, but the liver and kidneys cannot eliminate acids as rapidly as the lungs because of smaller capacity so compensation is less effective. Metabolic acids include lactic pyruvic, and keto-acids (produced during diabetic acidosis). Bicarbonate is the primary base in the body.

Respiratory imbalances can be caused by carbonic acid excess, leading to acidosis, or carbonic acid deficit, leading to alkalosis. *Metabolic* imbalances can be caused by base bicarbonate deficit, leading to acidosis, or base bicarbonate excess, leading to alkalosis.

<7.35	7.35 – 7.45	>7.45
Acidosis	Normal pH	Alkalosis
Excess carbonic acid OR bicarbonate deficit		Carbonic acid deficit OR bicarbonate excess

Anion gap

The anion gap reflects anions in plasma that are not usually measured. These anions include phosphates, sulfates, and proteins. Calculation of the anion gap requires analysis of electrolytes—sodium (Na^+), potassium (K^+), chloride (Cl^-)—as well as bicarbonate (HCO_3^-). Two formulas can be used to calculate the anion gap, one including potassium and the other omitting it (because potassium has a low level in plasma):

Formula	**Normal anion gap**
$Na^+ + K^+ - (Cl^- + HCO_3^-)$ = anion gap	12 – 16 mEq/L
$Na^+ - (Cl^- + HCO_3^-)$ = anion gap	8 – 12 mEq/L

An anion gap >16 mEq/L (using the first formula) suggests excessive accumulation of anions. The anion gap is important for diagnosis of metabolic acidosis.

Factors that interfere with ABG results

Care must be used to follow procedures for obtaining ABG samples. There are a number of factors that can affect test results:

Interfering factor	**Discussion**
Delay in processing	Values remain stable for 1 – 2 hours with iced (slurry) specimens. Room temperature specimens degrade rapidly: • $PaCO_2$ rates increase 3 – 10 mm Hg/hr. • PaO_2 rates decrease. • pH decreases slightly.
Air bubbles in serum	PaO_2 increases 0-30 mm Hg.

sample **Clots**	$PaCO_2$ may decrease slightly. Samples with air bubble or clots should be discarded.
Hyper- or hypothermia	Alters oxyhemoglobin curve. Analyzers measure samples at 37° C, so the lab should be notified of abnormal patient temperature. Fever increases pO_2 and pCO_2 levels, but when measured at 37° C, the results will show a decrease. The converse is true of hypothermia.
Hyperventilation/holding breath	May alter results.
Heparin excess	May dilute sample and alter readings. May decrease pH, pO_2, and pCO_2 levels.
Recent blood transfusion	May present misleading results.
Respiratory passage suctioning/respiratory therapy	Alters values for 20 – 30 minutes.
Citrates (as anticoagulant in collection tube)	Causes marked decrease in pH.
Carbon monoxide in blood	Falsely increases saO_2.
Inspired oxygen	May affect results if the sample is taken immediately after a change in inspired oxygen.

Collecting a sample

The most common site for collection of a sample for ABGs is the radial artery in the wrist. Circulation can be assessed by Doppler or by the Allen test to ensure that there is collateral circulation in the hand in the event that a thrombosis occurs, occluding the artery. In the Allen test, both the radial and ulnar artery are compressed and the patient is asked to clench the hand repeatedly until it blanches, and then one artery is released, and the tissue on that side should flush. The procedure is repeated for the other artery. A modified Allen Test involves having the patient extend the wrist over a rolled towel and make a fist:

- Palpate the ulnar and radial pulses and then apply pressure over the arteries, having the patient open and close the hand until it blanches.
- Release the ulnar artery only while maintaining pressure on the radial artery. The hand such regain color within 5 seconds, indicating adequate collateral circulation so that the radial artery can be use.

Nursing considerations	
Preparation	• Provide information to the patient about the test. • Obtain history, including allergies, recent tests, disorders, previous diagnostic procedures, and surgical procedures.

	• Obtain list of medications. • Record temperature and note oxygen use, including type, mode of delivery and changes. • Perform Allen test if sample to be collected from radial artery.
Taking arterial sample	• Avoid equipment with latex if patient is sensitive. • Follow standard precautions. • Advise patient to avoid movement. • Use an air-free heparinized syringe to collect the arterial sample, being careful to avoid introducing room air into the collection sample. • Apply needle stopper immediately after withdrawing needle. • Apply pressure dressing to puncture site. • Gently roll syringe to ensure that heparin and blood are evenly mixed to prevent formation of clots. • Immediately place syringe in plastic bag to protect label and place in ice slurry for transport to lab.
Taking venous sample	• Collect central venous sample in a heparinized syringe. • Collect percutaneous sample in a 5-mL heparinized (green) tube for adults or heparinized microcontainer for children. • Remove vacuum collection tube before withdrawing needle. • Apply dressing, roll syringe, place in ice slurry and transport as for arterial sample.
Taking capillary sample	• Do capillary puncture. • Neonates: Collect sample (heel or scalp) with two 250 μL heparinized capillary tubes). Fill tubes as much as possible before capping both ends. Some institutions recommend inserting metal fleas into the capillary tubes before capping and running a magnet up and down the tube during transport to mix and prevent clot formation. Be sure to notify lab staff of the fleas so they can be removed prior to testing of the sample as they may damage the gas analyzer. • Pediatric patients: Collect sample in heparinized microcontainer. Roll the tube gently to mix the sample with the heparin and transpq2ort immediately to the lab.
Taking cord blood sample	• Take sample directly from cord blood using a syringe. • Some institutions recommend insertion of fleas before capping (see above). • Immediately transport to lab.

Body's regulation of acid-base

Because the body's metabolism produces acids, the body must maintain the acid-base balance and the optimal pH by neutralizing and excreting excess acid. There are 3 mechanisms by which the body maintains the acid-base balance:

- ***Buffer system:*** This is a chemical reaction that changes strong acids into weak acids or to neutralize them. Buffers comprise weakly ionized acid or base and its salt. The most common buffer system is the carbonic acid-bicarbonate buffer, which attempts to maintain the 1:20 ratio. Since carbon dioxide produces carbonic acid (H_2CO_3) in solution ($H_2O + CO_2 = H_2CO_3$), an increase in carbon dioxide results in increased carbonic acid. The body compensates by increasing bicarbonate (HCO_3^-). If compensation fails, then an acid-base imbalance occurs. Other buffer systems include monohydrogen-dihydrogen phosphate, intracellular and plasma protein, and hemoglobin buffers that shift chloride and bicarbonate in and out of red blood cells in relation to the amount of oxygen in the blood.

- ***Respiratory system:*** The lungs regulate pH by controlling the excretion of carbon dioxide and water (and thus carbonic acid). The respiratory center of the medulla regulates respiration. The respiratory rate increases if the CO_2 or hydrogen levels elevate and decreases if levels fall. Both the rate and depth of respiration alter (hyperventilation vs hypoventilation) to blow off or retain CO_2 and hydrogen and maintain pH. If respiratory failure occurs, the respiratory system is unable to regulate the acid-base balance.

- ***Renal system:*** The kidneys excrete acids produced by cellular metabolism, usually resulting in acidotic urine (pH 6), but during compensation, urine pH may range from 4 to 8. As with other systems, kidney failure may prevent acid-base regulation. Renal compensation tends to be slow, needing hours or days to compensate. Renal regulation occurs by 3 mechanisms:
    - Secretion of hydrogen through the renal tubules.
    - Combining hydrogen (H^+) with ammonia (NH_3) to form ammonium (NH_4^+).
    - Excretion of weak acids.

Acid-base imbalances

Respiratory acidosis (Carbonic acid excess)

Hypoventilation causes increased CO_2 and carbonic acid in the blood with a decrease in pH. The kidneys compensate by conserving bicarbonate and excreting more hydrogen ions in the urine. If the patient is in acute respiratory failure, the initial bicarbonate levels are normal but should increase within 24 hours as the kidneys begin to compensate. Lab findings:

- Decreased serum pH < 7.35
- Increased $paCO_2$ >42 mm Hg.

- HCO_3^- normal if compensated.
- HCO_3^- decreased if uncompensated.
- Urine pH <6 if compensated.
- Electrolytes: Hyperkalemia if compensation fails.

Causes	Symptoms
• COPD • Overdose of sedative or barbiturate. • Obesity. • Severe pneumonia/atelectasis. • Muscle weakness (Guillain-Barré). • Mechanical hypoventilation. • Increased pulse, respirations, and BP.	**Neuro/muscular:** • Cerebral vasodilation, increased intracranial pressure (if severe). • Drowsiness • Dizziness • Headache • Coma • Disorientation • Seizures. **Cardiac:** • Peripheral vasodilation, flushing. • Ventricular fibrillation. • Hypotension. • (Acute) Increased pulse, respirations, & BP. **GI:** None. **Respiratory:** • Hypoventilation with hypoxia. • Cyanosis and tachypnea with increased ICP.

Medical management of respiratory acidosis aims primarily to improve ventilation. Treatment includes:

- Bronchodilators to improve ventilation and reduce bronchial spasms.
- Antibiotics to treat infections.
- Thrombolytics/anticoagulants for pulmonary emboli.
- Pulmonary hygiene.
- Hydration (2 – 3 L/day) to loosen secretions.
- Supplemental oxygen. Note: if the $paCO_2$ level is chronically over 50 mm Hg, the respiratory center becomes insensitive to CO_2 levels as a stimulus for respiration, and respirations are triggered by hypoxemia, so providing oxygen may result in carbon dioxide narcosis.
- Mechanical ventilation. Note: Increased $paCO_2$ level should be decreased slowly as too rapid decrease may result in alkalosis and seizures as the kidneys cannot excrete excess bicarbonate fast enough.

Respiratory alkalosis (Carbonic acid deficit)

Respiratory alkalosis almost always results from hyperventilation. Hyperventilation decreases pCO_2 level and carbonic acid level, leading to

alkalosis and increased pH. Compensated respiratory alkalosis (rare) during which the kidneys increase excretion of bicarbonate is characterized by a decreased bicarbonate level.
Lab findings:

- Increased serum pH >7.45.
- Decreased $PaCO_2$ <38 mm Hg.
- HCO_3^- normal if uncompensated and decreased if compensated.
- Urine pH >6 if compensated.
- Electrolytes: Hypokalemia and hypocalcemia.

Causes	Symptoms
• Hyperventilation associated with hypoxia. • Pulmonary embolus. • Exercise. • Anxiety. • Pain. • Fever. • Encephalopathy. • Septicemia. • Brain injury. • Salicylate overdose. • Gram-negative bacteremia. • Mechanical hyperventilation (inappropriate ventilator settings).	**Neuro/muscular:** • Cerebral vasoconstriction reduces cerebral blood flow. • Light-headed. • Confused. • Lethargic. • Tetany. • Numbness, tingling. • Hyperreflexia. • Seizures. • Tinnitus. **Cardiac:** • Tachycardia. • Ventricular and atrial dysrhythmias. • Vasoconstriction. **GI:** • Epigastric pain • Nausea and vomiting. **Respiratory:** • Hyperventilation.

Treatment aims to identify and correct the underlying cause. If hyperventilation is related to anxiety, sedatives may be of value, but often helping patients to slow breathing will reduce symptoms.

Metabolic acidosis (Base bicarbonate deficit)

Base bicarbonate deficit occurs when acids other than carbonic acid (such as ketoacids from DKA or lactic acid from shock) begin to accumulate in the body or when bicarbonate decreases because of fluid loss (such as with severe diarrhea). The body attempts to compensate by excreting additional CO_2 through the lungs with Kussmaul (deep rapid) respirations, and the kidneys try to excrete additional acid.
Laboratory findings include:

- Decreased serum pH.
- PCO_2 normal if compensated, and decreased if uncompensated.
- Decreased HCO_3^-.
- Urine pH <6 if compensated.
- Electrolytes: Hyperkalemia and hypocalcemia.
- Anion gap >16 mEq/L or <12 mEq/L (using the formula that includes potassium).

Metabolic acidosis is classified according to the type of anion gap:

- ***Normal anion gap (Hyperchloremic):*** Usually results from direct loss of bicarbonate as through diarrhea, ureterostomy, diuretics, excess administration of chloride, intestinal (lower) fistulas, parenteral nutrition without bicarbonate.
- ***Decreased anion gap:*** Rare.
- ***Increased anion gap:*** Results from excessive accumulation of fixed acid as in ketoacidosis, lactic acidosis, late salicylate poisoning, and methanol or ethylene toxicity. If the anion gap is ≥30 mEq/L, then metabolic acidosis is present regardless of other findings.

Causes	Symptoms
• Diabetic ketoacidosis. • Lactic acidosis. • Diarrhea. • Starvation. • Renal failure. • Shock. • Renal tubular acidosis. • Lower intestinal fistulas. • Salicylate poisoning. • Methanol/ethylene toxicity. • Excess administration of chloride.	**Neuro/muscular:** • Drowsiness. • Confusion. • Headache. • Coma. **Cardiac:** • Hypotension. • Arrhythmias, • Peripheral vasodilation with flushing. **GI:** • Nausea and vomiting. • Abdominal pain • Diarrhea. **Respiratory:** • Deep inspired tachypnea.

Treatment aims to correct the underlying cause of metabolic acidosis. If pH is <7.1 and bicarbonate <10, bicarbonate is administered. If acidosis is reversed too quickly, hypokalemia can occur, so serum potassium must be monitored carefully during treatment. If metabolic acidosis is chronic, resulting in hypocalcemia, then calcium levels should be raised prior to treatment to prevent tetany. Other treatments may include alkalizing agents (for bicarbonate level < 12 mEq/L), and hemodialysis or peritoneal dialysis.

Metabolic alkalosis (Base bicarbonate excess)

Base bicarbonate (HCO_3^-) excess occurs when the body loses acid (such as with excessive vomiting or gastric suctioning) or gains bicarbonate (such as with intake of baking soda). The body compensates through hypoventilation in an

attempt to increase CO_2 and carbonic acid levels and through increased excretion of bicarbonate through the kidneys. Most symptoms results from decreased ionization of calcium as more calcium combines with serum proteins. Laboratory findings include:

- Increased serum pH.
- PCO_2 normal if uncompensated and increased if compensated.
- Increased HCO_3^-.
- Urine pH >6 if compensated.
- Electrolytes: Hypocalcemia, hypokalemia, and hypochloremia (especially with vomiting or cystic fibrosis).

Causes	Symptoms
• Excessive vomiting. • Gastric suctioning. • Diuretics (long-term use), especially thiazides or furosemide. • Potassium depletion. • Cystic fibrosis. • Chronic ingestion of milk and calcium carbonate. • Excessive mineralocorticoids. • Excessive sodium bicarbonate intake.	**Neuro/muscular:** • Dizziness. • Confusion. • Nervousness. • Anxiety. • Tremors. • Muscle cramping. • Tetany. • Tingling. • Seizures. **Cardiac:** • Atrial tachycardia • Ventricular disturbances (with pH >7.6 and hypokalemia). **GI:** • Nausea and vomiting • Anorexia. **Respiratory:** • Compensatory hypoventilation.

Treatment aims to identify and reverse the underlying cause of metabolic alkalosis. Fluid deficit should be replaced and sufficient chloride provided. Electrolyte imbalances are corrected as necessary. If related to gastric suctioning, histamine-2 receptor antagonists, such as cimetidine, may reduce stomach hydrochloric acid. If patients can't tolerate rapid fluids, then carbonic anhydrase inhibitors may be administered. Fluid intake and output should be monitored carefully.

Mixed

More than one type of acid-base imbalance may occur simultaneously. For example, those with COPD or chronic use of diuretics may develop a combination of respiratory acidosis and metabolic acidosis, resulting in a near-normal pH level. Mixed acidosis (both respiratory and metabolic) may occur with

cardiopulmonary arrest. Symptoms can vary widely, depending on the combination of imbalances.

Steps in diagnosing acid-base disturbances (acidosis vs alkalosis)		
1	Evaluate pH	Identify whether pH is acidotic or alkalotic (with 7.4 as the starting point). • <7.4 is becoming more acidotic. • >7.5 is becoming more alkalotic.
2	Evaluate pCO_2 (forms carbonic acid)	High levels indicate acidosis. Low levels indicate alkalosis.
3	Evaluate HCO_3^- (a base)	High levels indicate alkalosis. Low levels indicate acidosis.
4	Evaluate pH in relation to pCO_2 and HCO_{3-}	Determine which matches alteration in pH. If, for example, the pH is acidotic and $paCO_2$ is high (respiratory acidosis) but the HCO_3^- is low (metabolic alkalosis), $paCO_2$ matches the alteration of pH (both acidotic) and the disturbance is diagnosed as respiratory acidosis.
5	Evaluate for compensation	Determine if the parameter that doesn't match the pH is moving in the opposite direction. For example, (see step 4), if the pH is acidotic and the $paCO_2$ is acidotic, then HCO_3- should be increasing to compensate and to increase the pH, bringing it back to normal.

Summary

Blood gas analysis determines the acid-base balance in the blood. Tests included include hydrogen ion concentration (pH), partial pressure of oxygen in the blood (pO_2), partial pressure of carbon dioxide in the blood (pCO_2), bicarbonate (HCO_3^-), oxygen saturation (SO_2), and base excess (BE) or (base deficit (BD). Calculating the anion gap is also important for diagnosis of metabolic acidosis. Care must be taken to avoid interfering factors when obtaining a blood sample for testing. Interfering factors include delay in processing, air bubbles, clots, hyper- or hypothermia, hyperventilation, heparin excess, recent blood transfusion, respiratory suctioning, citrates, carbon monoxide, and inspired oxygen. Samples for ABGs are usually taken from the radial artery, so the Allen test should be performed before to ensure that collateral circulation is adequate in case thrombosis occurs at the test site. In some cases, cord blood, venous blood, or capillary blood may be used as well. The body has 3 systems that maintain the acid-base balance: buffer system, respiratory system, and renal system. The 4 primary types of acid-base balance include respiratory acidosis (carbonic acid excess), respiratory alkalosis (carbonic acid deficit), metabolic

acidosis (base bicarbonate deficit), and metabolic alkalosis (base bicarbonate excess). Some patients may also have mixed forms of acid-base imbalance.

References

Arterial blood gas. (2018). Family Practice Notebook. Retrieved November 3, 2019, from http://www.fpnotebook.com/Renal/Lab/ArtrlBldGs.htm

Grogono, A.W. (2018, March). Acid-base tutorial homepage. Tulane University Department of Anesthesiology.

Kee, J.LeF., Paulanka, B.J., & Purnell, L.D. (2015). Handbook of Fluid, Electrolyte, and Acid-Base Imbalances 2nd ed. Clifton Park, NY: Thomson, Delmar Learning.

Lewis, III, J.L. (2008, July). Acid-base balance. The Merck Manuals. Retrieved November 3, 2019, from http://www.merck.com/mmhe/sec12/ch159/ch159a.html

Lewis, S.M., Heitkemper, M.McL., & Dirksen, S.R. (2015). Medical Surgical Nursing 6th ed. St. Louis: Mosby.

Nelligan, P. (2002) Oxygen: What is the oxyhemoglobin dissociation curve and why is it important? University of Pennsylvania: Critical Care Medicine Tutorials. Retrieved November 3, 2019, from http://www.ccmtutorials.com/rs/oxygen/page06.htm

Shier, D., Butler, J., & Lewis, R. (2017). Holes Human Anatomy & Physiology 11th ed. New York: McGraw-Hill.

Smeltzer, S.C., Bare, B., Hinkle, J.L, & Cheever, K.H. (2019). Brunner & Suddarth's Medical-Surgical Nursing 11th ed. Philadelphia: Lippincott, Williams, & Wilkins.