

Interpretation and Clinical Significance of Arterial Blood Gas Analysis in Respiratory and Laboratory Medicine-An Updated Review

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Abstract:

Background: Arterial blood gas (ABG) analysis is a cornerstone diagnostic tool that provides direct assessment of ventilation, oxygenation, and acid–base balance. Despite advancements in non-invasive monitoring, ABG remains the gold standard for evaluating critically ill patients and guiding therapeutic decision-making.

Aim: This review aims to summarize updated principles, specimen requirements, analytical procedures, interpretive strategies, and clinical applications of ABG analysis in respiratory and laboratory medicine.

Methods: A comprehensive narrative review of current practices in ABG analysis was undertaken, focusing on physiological principles, pre-analytical variables, analyzer technology, quality control, and interpretative frameworks. Clinical examples were included to illustrate systematic interpretation.

Results: ABG interpretation integrates pH, PaCO₂, PaO₂, bicarbonate, and oxygen saturation to distinguish respiratory and metabolic disorders, assess compensation, and evaluate oxygenation. Accurate results require strict sample handling, anaerobic collection, rapid processing, and appropriate anticoagulation. Modern analyzers enable precise measurement through advanced calibration systems, barometric correction, and automated quality control. Pre-analytical errors—including air exposure, improper heparin use, and delays in processing—remain major sources of inaccuracy. Clinically, ABG findings guide management of acute respiratory failure, shock, metabolic derangements, and ventilation strategies. Tools such as the A–a gradient, P/F ratio, and structured interpretation methods (e.g., Romanski method) enhance diagnostic reliability.

Conclusion: ABG analysis is indispensable for evaluating respiratory and metabolic status across emergency, critical care, and chronic disease management. Accurate sampling, rigorous analytical processes, and structured interpretation significantly improve clinical outcomes.

Key Words: Arterial blood gas, pH, PaCO₂, PaO₂, acid–base balance, ventilation, oxygenation, A–a gradient, P/F ratio, quality control.

INTRODUCTION:

Arterial blood gas (ABG) analysis is a fundamental diagnostic tool in modern clinical practice, providing direct insight into a patient's respiratory function, oxygenation status, and acid-base balance. By quantifying partial pressures of oxygen (PaO₂) and carbon dioxide (PaCO₂) and assessing blood pH, ABG analysis allows healthcare providers to identify and manage respiratory, circulatory, and metabolic disorders with precision [1]. While blood gas analysis can theoretically be performed using arterial, venous, or capillary blood, ABG specifically refers to measurements obtained from arterial blood, providing the most accurate assessment of systemic oxygenation and ventilation [2]. PaO₂ reflects the efficacy of oxygen transport and diffusion from the lungs into the bloodstream, while PaCO₂ serves as a marker of alveolar ventilation. Abnormal PaCO₂ values indicate either hypoventilation, commonly observed in chronic obstructive pulmonary disease or neuromuscular weakness, or hyperventilation, which may occur in metabolic acidosis or anxiety states [3]. Although non-invasive modalities, such as pulse oximetry for oxygen saturation and end-tidal carbon dioxide monitoring for ventilation, have become increasingly widespread, ABG analysis remains the gold standard for evaluating gas exchange and acid-base disturbances [4]. The value of ABG extends beyond oxygenation and ventilation; it provides essential information for diagnosing and managing conditions that disrupt acid-base homeostasis. Most modern ABG analyzers measure pH and PaCO₂ directly, while serum bicarbonate (HCO₃⁻) and base deficit or excess are derived from the Henderson-Hasselbalch equation [2]. The calculated bicarbonate reflects the CO₂ dissolved in plasma but does not fully account for CO₂ bound in carbamino compounds or carbonic acid, which is measured in total serum CO₂ during laboratory analysis [5][6]. Consequently, calculated HCO₃⁻ typically differs slightly from directly measured total CO₂ by approximately 1.2 mmol/L, although discrepancies can be more pronounced in critically ill patients due to altered CO₂ transport dynamics [7]. Interpreting these values requires careful consideration of the analyzer, calibration, and institutional standards, as the accuracy of calculations may vary across devices and methodologies [6].

ABG analysis is utilized across a range of clinical specialties. In emergency medicine and critical care, ABG provides rapid evaluation in patients presenting with acute respiratory distress syndrome (ARDS), severe sepsis, septic shock, hypovolemic shock, and cardiac arrest. In endocrinology and metabolic medicine, it aids in diagnosing diabetic ketoacidosis, renal tubular acidosis, and inborn errors of metabolism [3]. Pulmonologists and anesthesiologists frequently rely on ABG to guide ventilation strategies during mechanical ventilation or anesthesia, while intensivists use it to titrate oxygen therapy, monitor acid-base derangements, and assess response to interventions. ABG results influence immediate treatment decisions, such as initiating or adjusting mechanical ventilation, administering bicarbonate therapy, or correcting electrolyte imbalances. Beyond acute care, ABG analysis is a valuable tool for longitudinal monitoring of patients with chronic respiratory or metabolic disorders. Repeated measurements can detect early deterioration, guide adjustments in therapy, and evaluate the effectiveness of interventions over time. The ability to simultaneously assess oxygenation, ventilation, and acid-base status in a single test underlines the comprehensive utility of ABG analysis. For laboratory specialists and respiratory clinicians, understanding the principles,

limitations, and clinical applications of ABG is critical for accurate interpretation and informed decision-making. Its integration into patient care pathways enhances diagnostic accuracy, supports timely interventions, and ultimately improves patient outcomes across a wide spectrum of acute and chronic conditions. In summary, ABG analysis represents a cornerstone in the assessment of respiratory, metabolic, and circulatory status. Its precise measurement of PaO₂, PaCO₂, and pH, combined with calculated and measured bicarbonate values, provides a robust framework for diagnosing and managing complex physiological derangements. Accurate interpretation requires familiarity with analytical principles, potential discrepancies between calculated and measured values, and the clinical context in which the test is performed. ABG remains indispensable in critical care, emergency medicine, anesthesiology, and pulmonology, guiding timely interventions, optimizing patient management, and improving both short- and long-term outcomes [1][2][3][4][5][6][7].

Pathophysiology

Arterial blood gas analysis provides a direct assessment of a patient's respiratory and metabolic status, enabling the identification of diverse pathological conditions. The pH, PaO₂, and PaCO₂ values reflect the balance between acid production, bicarbonate buffering, and pulmonary ventilation, while comparison with measured serum bicarbonate can reveal compensatory mechanisms or underlying metabolic disturbances [1]. Deviations from normal ranges indicate either respiratory or metabolic dysfunction, or a combination of both, allowing clinicians to distinguish between primary disorders and compensatory responses. The alveolar-arterial (A-a) oxygen gradient, calculated from ABG results, is a critical tool in evaluating pulmonary gas exchange efficiency. This gradient quantifies the difference between oxygen concentration in the alveoli and arterial blood, highlighting abnormalities in oxygen transfer across the alveolar-capillary membrane. An elevated A-a gradient often indicates ventilation-perfusion mismatch, as seen in conditions such as pulmonary embolism, acute respiratory distress syndrome, or chronic obstructive pulmonary disease. It can also signal intrapulmonary shunting or diffusion limitations due to parenchymal lung disease [8]. ABG interpretation provides insight into compensatory responses, such as renal bicarbonate retention during chronic respiratory acidosis or increased respiratory rate in metabolic acidosis, reflecting the body's attempt to restore homeostasis. By integrating PaO₂, PaCO₂, pH, serum bicarbonate, and the A-a gradient, clinicians can not only diagnose the primary disorder but also assess severity, guide therapy, and monitor response to interventions. This comprehensive physiological information makes ABG analysis a cornerstone in the evaluation of critically ill patients and those with complex metabolic or respiratory disorders.

Specimen Requirements and Procedure

Arterial blood gas (ABG) analysis requires whole blood as the specimen, obtained either through direct arterial puncture or via an indwelling arterial catheter. While detailed procedural techniques for arterial cannulation and puncture are covered extensively in specialized references, including the StatPearls article "Arterial Lines," the critical aspect for accurate ABG measurement lies in proper handling, storage, and rapid processing of the collected sample [9]. Once the specimen is drawn, it must be kept on ice and analyzed promptly to minimize metabolic changes and gas diffusion that could result in erroneous readings. Delays in analysis, exposure to room air, or improper handling can significantly alter measured partial pressures and pH values, compromising the diagnostic utility of the ABG. Differences between arterial and venous blood gas values are most clinically significant for oxygenation. PaO₂ is the primary reason for arterial sampling, as venous oxygen levels are generally

approximately 60 mm Hg lower than arterial levels due to oxygen extraction at the tissue level [10]. In contrast, venous PaCO₂ is slightly higher, typically by 2 to 8 mm Hg, and venous pH is marginally lower by 0.02 to 0.05 units [11]. Therefore, reliance on venous blood for oxygenation assessment is limited. Proper specimen collection is essential for ensuring accurate results. Indwelling catheters with heparin locks, commonly used for short- or long-term intravenous therapies, must be flushed thoroughly; failure to do so can introduce heparin or residual fluids that distort blood gas measurements, producing non-physiologic or inconsistent values [12].

Specimens, whether arterial or venous, must be collected anaerobically in sterile syringes containing lyophilized heparin, typically 1 to 3 mL in volume. Evacuated tubes, such as lithium heparin tubes used for plasma collection, are unsuitable due to residual oxygen that can artificially elevate PaO₂ values [13]. Syringes with lyophilized heparin are preferred over liquid heparin, as liquid heparin contains atmospheric gases that dilute the sample, with the effect magnified if the syringe is underfilled. Inappropriate ratios of liquid heparin to blood significantly alter measured PCO₂ and derived parameters, while manufacturing variability in syringes can further affect pre-analytical accuracy [14][15]. Anaerobic technique is crucial because exposure of the specimen to atmospheric air alters gas tensions. Atmospheric PCO₂ is approximately 0.25 mm Hg, far below physiological levels, and exposure reduces blood CO₂ content and increases pH. Conversely, atmospheric PO₂ of roughly 155 mm Hg is higher than arterial blood, and exposure may artificially elevate oxygen tension, or conversely lead to oxygen loss in hyperoxic blood from patients receiving supplemental oxygen [16][17]. Even minimal air exposure from needle or syringe dead space can influence results. Studies have demonstrated that a small bubble introduced into a 2 mL blood sample can increase PaO₂ by several mm Hg and decrease PaCO₂ within minutes [18]. Gentle ejection of bubbles and immediate mixing of the sample by rolling between the hands ensures a homogeneous specimen and minimizes pre-analytical error [19]. Arterialized capillary blood is sometimes used as an alternative when arterial puncture is impractical, although its reliability is contingent on appropriate warming and collection technique [20].

Modern ABG analysis is typically performed with automated analyzers, providing results within 10 to 15 minutes. These analyzers directly measure pH, PaO₂, and PaCO₂, while calculating bicarbonate concentration, base excess or deficit, and arterial oxygen saturation (SaO₂), except in cases where co-oximetry is used for direct SaO₂ measurement [1]. Accurate analysis requires meticulous sample handling, rapid processing, and awareness of pre-analytical variables. Prior to radial artery sampling, evaluation of collateral blood flow via the modified Allen test is standard practice to ensure adequate perfusion through the ulnar artery. The patient flexes the selected upper extremity and clenches the fist while pressure is applied to both radial and ulnar arteries. Following release of the ulnar artery, prompt reperfusion of the palm confirms sufficient collateral flow, indicating it is safe to proceed with radial artery puncture. Failure to demonstrate adequate reperfusion suggests the risk of ischemic complications, necessitating alternative sampling sites such as the femoral artery [21]. This precaution, along with strict adherence to anaerobic collection and anticoagulant use, underpins the reliability and clinical utility of ABG analysis in guiding respiratory, metabolic, and critical care management. In conclusion, specimen integrity and collection technique are paramount to obtaining reliable arterial blood gas measurements. Whole blood must be collected under anaerobic conditions with appropriate anticoagulants, analyzed rapidly, and handled to minimize exposure to atmospheric gases. Knowledge of site selection, pre-

procedural assessment, and sample handling ensures accurate assessment of oxygenation, ventilation, and acid-base balance. Adherence to these principles enables ABG analysis to remain a cornerstone in the diagnosis and management of respiratory, metabolic, and systemic disorders.

Testing Procedures

Blood gas analysis relies on precise measurement of physiological parameters such as pH, partial pressures of oxygen (PaO₂) and carbon dioxide (PaCO₂), bicarbonate (HCO₃), base excess or deficit, and oxygen saturation (SaO₂). The procedure begins with the operator introducing a whole blood specimen into the instrument's sample probe. A peristaltic pump draws the specimen into the measurement chamber, where it remains long enough to achieve thermal equilibrium, allowing stable and reproducible readings. After measurement, the pump directs the sample to a waste collection system, preparing the instrument for subsequent testing [22]. The accurate performance of this process depends on meticulous attention to equipment function, sample handling, and adherence to operational protocols. Electrodes, which are the core sensors for pH, PCO₂, and PO₂, are inherently unstable over time. Consequently, routine and frequent calibration is required to ensure measurement accuracy [23]. Most blood gas instruments incorporate an integrated barometer to account for barometric pressure (P[Amb]), which is necessary for precise calculation of gas tensions. Without proper barometric correction, readings can be significantly inaccurate, particularly in facilities at high altitudes or areas with fluctuating atmospheric pressure. The evolution of blood gas analyzers has led to the development of point-of-care and bedside testing devices. Portable, stand-alone instruments are now widely available, providing rapid results with minimal operator training. Some hand-held analyzers utilize disposable electrodes, further simplifying operation while reducing contamination and maintenance concerns [24].

Contemporary blood gas analysis systems achieve reliable and precise measurements due to a combination of sophisticated technology and the use of high-quality calibrator materials. However, the reliability of the results is contingent upon several factors: strict adherence to the manufacturer's protocols, proper equipment maintenance, correct specimen collection and handling, and the consistent use of quality control measures [22]. Microprocessor-controlled analyzers often include software routines that monitor electrode performance, display warnings, and assist in troubleshooting deviations from expected readings. Operators are guided by these automated diagnostics, though practical experience is crucial in determining maintenance frequency and interpreting subtle anomalies [25]. Maintaining the cleanliness of the sample chamber and measurement pathway is critical for accurate results. Most modern analyzers include automatic flushing routines that cleanse the chamber after each measurement. Despite these safeguards, partial or complete clogging can still occur, primarily due to the presence of fibrin strands, microclots, or protein aggregates in the blood specimen [1]. These obstructions can interfere with electrode contact with the sample and buffers, potentially producing inaccurate readings or affecting subsequent calibrations [18]. The operator must visually inspect the chamber and sample path, where heat sinks or transparent sections allow detection of clogs, debris, or residual air bubbles [26]. Air bubbles are particularly problematic because they can adhere to electrode surfaces, alter gas tensions, and distort measured values. Prompt removal of bubbles and proper mixing of the specimen are essential to ensure representative measurements.

Specimens must be handled with care to avoid pre-analytical errors that may compromise instrument readings. Thermal equilibration is required because electrode response can vary

with temperature. Blood samples that remain in the chamber for extended periods may undergo metabolic activity, altering PaO₂, PaCO₂, and pH. Additionally, repeated exposure to heat can accelerate fibrin polymerization, increasing the likelihood of clogs and inaccuracies. Operators must be vigilant in observing both the physical and chemical integrity of the sample throughout the testing process. The introduction of portable blood gas analyzers and point-of-care devices has revolutionized clinical decision-making, particularly in critical care, emergency, and perioperative settings. These devices provide rapid results, enabling immediate adjustments in ventilation strategies, oxygen supplementation, and acid-base management. Despite their ease of use, the fundamental principles of accurate ABG measurement remain the same: meticulous sample collection, proper anticoagulation, anaerobic handling, thermal stabilization, and consistent instrument calibration. Neglect of any of these factors can significantly compromise the clinical utility of the results, potentially leading to inappropriate therapeutic interventions.

In addition to routine measurements, advanced blood gas analyzers can incorporate co-oximetry and metabolic calculations, offering comprehensive assessment of oxygen delivery and acid-base balance. The integration of microprocessor-based diagnostics enhances operator confidence and reduces the risk of procedural errors. Nevertheless, the human element remains essential. Experienced operators recognize artifacts caused by bubbles, clots, or incomplete mixing, and can implement corrective actions before analysis. Proper training in sample handling, awareness of pre-analytical factors, and understanding instrument mechanics are fundamental to obtaining reliable results. In summary, the testing procedure for arterial blood gas analysis is a complex interplay between advanced instrumentation, precise specimen handling, and operator expertise. Specimens are drawn into the analyzer via a peristaltic pump, thermally equilibrated, and measured while minimizing interference from bubbles, fibrin, or clots. Frequent calibration, barometric correction, and proper maintenance of the instrument ensure reproducibility and accuracy. Modern point-of-care and portable devices facilitate rapid testing in diverse clinical environments, yet the principles of specimen integrity, anaerobic collection, and meticulous handling remain critical. A comprehensive understanding of these procedural elements enables healthcare providers to interpret ABG results accurately, guide therapeutic interventions, and optimize patient outcomes in acute and critical care settings [22][23][24][25][26].

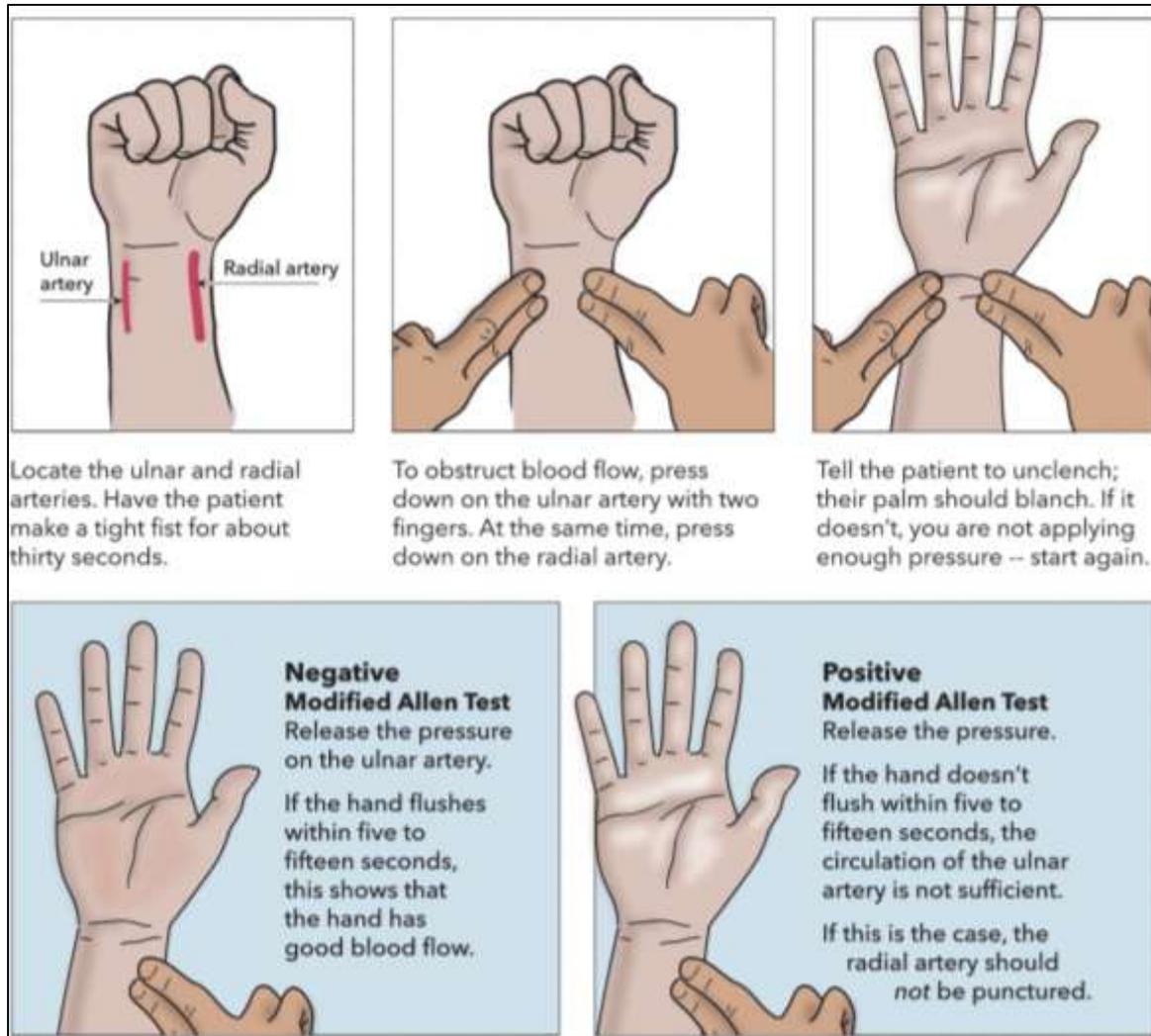


Fig. 1: Modified Allen Test. This test is used to check the overall blood supply to the hand.

RESULTS, REPORTING, AND CRITICAL FINDINGS

Arterial blood gas (ABG) analysis provides a comprehensive assessment of a patient's acid-base balance, oxygenation, and ventilation status, forming a critical component of diagnostic evaluation in acute and chronic medical conditions. Standard reference ranges for ABG components are well established, although variations may occur depending on laboratory methods, patient age, or comorbid conditions [27][28]. Normal arterial blood gas values include a pH of 7.35 to 7.45, PaO₂ of 75 to 100 mm Hg, PaCO₂ of 35 to 45 mm Hg, bicarbonate (HCO₃) of 22 to 26 mEq/L, base excess or deficit of -4 to +2, and arterial oxygen saturation (SaO₂) of 95 to 100 percent. Interpreting these results systematically allows clinicians to determine the severity of deviations, differentiate acute versus chronic disturbances, and identify the primary etiology as respiratory or metabolic [29]. The initial step in ABG interpretation involves evaluating the pH to determine whether acidemia or alkalemia is present. A pH below 7.35 indicates acidemia, whereas a pH above 7.45 reflects alkalemia. In cases where the pH remains within the normal range, a nominal cutoff of 7.40 is often applied for analytical purposes, classifying values slightly below 7.40 as acidosis and values above as alkalosis. This approach ensures subtle deviations are not overlooked and facilitates

early detection of clinically relevant abnormalities. Following assessment of pH, the respiratory and metabolic components are examined using PaCO₂ and HCO₃, respectively. Elevated PaCO₂ in conjunction with a low pH is indicative of primary respiratory acidosis, whereas reduced PaCO₂ with a high pH suggests primary respiratory alkalosis. Conversely, alterations in HCO₃ are typically representative of metabolic acid-base disturbances. The clinician must distinguish whether the observed deviations are primary or represent compensatory responses to a disorder in the other system [29].

Compensation analysis is an essential aspect of ABG interpretation, as the body attempts to restore homeostasis by partially correcting the primary disturbance. For instance, in metabolic acidosis, a respiratory compensation manifests as hyperventilation, reducing PaCO₂ to mitigate the acidotic state. In metabolic alkalosis, hypoventilation may increase PaCO₂, though this response is often limited by hypoxic drive. Conversely, in respiratory disorders, renal compensation occurs gradually through retention or excretion of bicarbonate. Determining whether compensation is appropriate requires comparing expected physiological responses with the actual ABG values. Discrepancies may indicate mixed acid-base disorders, which are associated with more complex pathophysiology and may require targeted interventions [29]. The oxygenation status, represented by PaO₂ and SaO₂, provides crucial information about the patient's respiratory function and tissue oxygen delivery. Hypoxemia is defined as a PaO₂ below 75 mm Hg, and it may occur due to ventilation-perfusion mismatch, diffusion defects, hypoventilation, or shunting. Interpretation of PaO₂ must consider supplemental oxygen, hemoglobin concentration, and cardiac output, as these factors directly influence oxygen delivery. Clinicians must also evaluate the alveolar-arterial oxygen gradient to detect subtle impairments in pulmonary gas exchange. In critically ill patients, low PaO₂ may indicate acute respiratory distress syndrome, sepsis-related hypoxemia, or cardiopulmonary compromise, prompting immediate intervention [29].

The Romanski method is a practical approach for systematically interpreting ABG results across all levels of healthcare providers. This methodology focuses on stepwise evaluation: first, assess pH; second, determine whether the primary disturbance is respiratory or metabolic using PaCO₂ and HCO₃; third, evaluate the presence and adequacy of compensation; and fourth, assess oxygenation and potential hypoxemia [30]. By applying this systematic approach, clinicians can identify subtle derangements, distinguish between acute and chronic processes, and detect mixed disorders that may otherwise be overlooked. The method ensures consistent interpretation, facilitating rapid decision-making in emergency, critical care, and perioperative settings. In addition to diagnostic interpretation, ABG reporting requires careful documentation of critical values. Abnormal results, such as severe acidemia (pH < 7.25), severe alkalemia (pH > 7.55), marked hypoxemia (PaO₂ < 60 mm Hg), or significant hypercapnia (PaCO₂ > 60 mm Hg), should be communicated immediately to the attending healthcare team. These values often indicate life-threatening derangements requiring urgent intervention, including ventilatory support, hemodynamic stabilization, or correction of metabolic abnormalities. Rapid and accurate reporting enhances patient safety, guides therapy, and informs prognosis, particularly in acute care settings such as intensive care units, emergency departments, or perioperative monitoring [29].

Finally, clinicians must recognize that ABG interpretation is not isolated from clinical context. Integration with patient history, physical examination, and additional laboratory and imaging studies is essential. For example, a low PaO₂ in a patient with dyspnea and diffuse infiltrates may suggest acute respiratory distress syndrome, whereas the same PaO₂ in a patient with

chronic obstructive pulmonary disease may represent baseline hypoxemia. Similarly, metabolic acidosis in diabetic ketoacidosis presents with distinct compensatory respiratory patterns, emphasizing the importance of correlating ABG findings with underlying pathophysiology [29]. In summary, arterial blood gas analysis provides an indispensable tool for evaluating acid-base balance, ventilation, and oxygenation. A systematic interpretation that includes assessment of pH, PaCO₂, HCO₃⁻, compensation, and oxygenation allows clinicians to identify primary and secondary disorders, detect mixed abnormalities, and guide appropriate interventions. Adherence to established reference ranges, consideration of patient-specific variables, and prompt reporting of critical values are crucial for optimal clinical outcomes. The Romanski method offers a structured approach suitable for diverse clinical settings, supporting rapid, accurate, and reproducible interpretation of ABG results [27][28][29][30].

ABG Interpretation: Clinical Examples

Arterial blood gas (ABG) interpretation requires a structured approach to determine the primary acid-base disturbance, the presence of compensatory mechanisms, and the status of oxygenation. In the first example [28], the ABG values are as follows: pH 7.39, PaCO₂ 51 mm Hg, PaO₂ 59 mm Hg, HCO₃⁻ 30 mEq/L, and SaO₂ 90% on room air. The pH falls within the standard reference range (7.35–7.45); however, using 7.40 as the analytic cutoff, the pH is below 7.40, indicating acidemia. The elevated PaCO₂ signifies respiratory acidosis, as CO₂ retention contributes directly to the acidotic state. Concurrently, the elevated HCO₃⁻ reflects a metabolic alkalosis, inconsistent with the primary acid-base disturbance, indicating compensatory renal adaptation. Since renal compensation requires several days to develop, the presence of increased HCO₃⁻ suggests a chronic or non-acute respiratory acidosis. Finally, the decreased PaO₂ and SaO₂ reveal impaired oxygenation, which may result from alveolar hypoventilation, ventilation-perfusion mismatch, or diffusion abnormalities. Correlation with patient history, physical findings, and underlying pulmonary or systemic pathology is necessary to determine the severity and guide potential interventions. In the second example [28], the ABG is as follows: pH 7.45, PaCO₂ 32 mm Hg, PaO₂ 138 mm Hg, HCO₃⁻ 23 mEq/L, base deficit 1 mEq/L, and SaO₂ 92% on room air. The pH is slightly above 7.40, indicating alkalemia. The PaCO₂ is below the normal range, confirming a primary respiratory alkalosis, often arising from hyperventilation due to anxiety, hypoxemia, or compensatory responses to metabolic acidosis. The HCO₃⁻ is within the lower end of normal and not inconsistent with the pH, indicating the absence of significant metabolic compensation. The PaO₂ is elevated, reflecting adequate oxygenation and possible supplemental oxygen exposure, although the SaO₂ remains within acceptable limits. This scenario demonstrates an acute respiratory alkalosis without compensatory renal adaptation, as metabolic mechanisms have not yet engaged.

Evaluating ABG results in isolation may not capture the full clinical picture. Integrating serum electrolytes, including the anion gap, provides additional insight, particularly in conditions such as diabetic ketoacidosis. In such cases, an initially elevated anion gap due to ketone accumulation may normalize as ketones are metabolized, yet persistent metabolic acidosis can remain due to hyperchloremia, which is beyond the scope of basic ABG interpretation but illustrates the importance of a comprehensive evaluation [31]. These examples emphasize the critical value of systematic ABG analysis, combining the assessment of pH, PaCO₂, HCO₃⁻, and oxygenation parameters to identify primary disorders, determine the adequacy of compensatory mechanisms, and guide clinical decision-making. Proper interpretation ensures

timely recognition of acute and chronic disturbances, informs appropriate interventions, and helps predict prognosis in complex medical scenarios.

Clinical Significance

Arterial blood gas (ABG) analysis remains a cornerstone in the assessment of oxygenation, ventilation, and acid-base status, despite the increasing use of non-invasive monitoring techniques. While pulse oximetry and capnography provide valuable continuous data, ABG analysis offers direct measurement of PaO₂, PaCO₂, and pH, allowing confirmation and calibration of non-invasive monitoring systems [1]. This is particularly critical in critically ill patients where precise oxygenation and ventilatory parameters guide therapeutic decisions. ABG evaluation is frequently employed in settings such as the intensive care unit (ICU) and emergency department, especially in cases of acute respiratory failure, acute respiratory distress syndrome (ARDS), and severe sepsis. In these contexts, calculation of the alveolar-arterial (A-a) oxygen gradient provides a refined assessment of gas exchange abnormalities, helping differentiate between hypoventilation, shunting, ventilation-perfusion mismatch, and diffusion impairment [25]. Although the A-a gradient is less accurate at high fractions of inspired oxygen, it remains a useful diagnostic adjunct. For practical bedside evaluation, clinicians often use the PaO₂/FiO₂ (P/F) ratio to assess oxygenation and classify ARDS severity [32]. The P/F ratio is easily derived but has limitations, as discrepancies between venous admixture and the ratio increase at varying FiO₂ levels. In neonates and pediatric populations, the oxygenation index (OI) is frequently used, integrating the mean airway pressure (Paw) required to maintain oxygenation with FiO₂ and PaO₂. The OI provides a more sensitive measure of pulmonary injury than the P/F ratio and guides clinical interventions such as administration of inhaled nitric oxide, surfactant therapy, or initiation of extracorporeal membrane oxygenation [33][34]. Despite a normal PaO₂, respiratory failure may still be present, particularly in patients receiving supplemental oxygen, highlighting the need for comprehensive ABG interpretation rather than reliance on isolated oxygen values.

PaCO₂ is a critical marker of alveolar ventilation and cellular CO₂ production. Hypercapnia and hypocapnia reflect hypoventilation and hyperventilation, respectively, and serve as sensitive indicators of respiratory failure even when oxygenation is maintained [27]. Pulmonary dead space fraction, calculated from the difference between PaCO₂ and mixed expired CO₂ or end-tidal CO₂, is a robust measure of ventilatory efficiency. Increased dead space indicates a mismatch between ventilation and perfusion and is associated with shunting. Elevated pulmonary dead space serves as a prognostic marker in ARDS and can assist in diagnosing conditions such as pulmonary embolism [1][35]. ABG analysis is also integral to assessing acid-base disorders. Respiratory disturbances, such as hypoventilation or hyperventilation, directly influence pH, resulting in respiratory acidosis or alkalosis. Hypoxemic conditions can induce anaerobic metabolism, generating metabolic acidosis. Metabolic disturbances, independent of respiratory function, also contribute to acid-base imbalances. Acute metabolic acidosis can occur in diabetic ketoacidosis, septic shock, renal failure, ingestion of toxins, and gastrointestinal or renal bicarbonate loss [28]. Conversely, metabolic alkalosis arises from kidney disease, electrolyte disturbances, prolonged vomiting, hypovolemia, diuretic use, or hypokalemia [36]. Recognizing the interplay between respiratory and metabolic components is essential for accurate interpretation of ABG results and for guiding targeted therapeutic interventions. Overall, ABG monitoring provides precise, real-time insights into oxygenation, ventilation, and acid-base status that are critical for managing critically ill patients. Beyond initial diagnosis, ABG analysis allows evaluation of disease progression, guides ventilatory and

pharmacologic management, and aids in prognostication. It remains indispensable for the assessment of complex interactions between respiratory, metabolic, and circulatory systems, providing a foundation for clinical decision-making in emergency and intensive care settings.

Quality Control and Lab Safety

Accurate arterial blood gas (ABG) and electrolyte analysis is fundamental to patient care, particularly when these tests are performed as point-of-care assessments in emergency or intensive care settings. Devices used for ABG testing must be properly calibrated and standardized to ensure the reliability of the results, as clinical decision-making frequently relies on subtle changes in measured parameters [37]. Device calibration involves following manufacturer instructions precisely and collaborating with the clinical laboratory team to confirm accuracy. This collaboration is critical because inconsistencies in calibration, maintenance, or electrode performance can lead to clinically significant errors, potentially compromising patient safety. Effective quality assurance in ABG analysis incorporates multiple elements. Instrument maintenance must be systematic, including routine cleaning, calibration, and electrode verification. The use of control materials, such as standard solutions with known pH, PaO₂, and PaCO₂ values, allows verification of instrument accuracy. Electrode linearity should be periodically assessed to ensure that the sensors respond consistently across the physiological range. Barometer function is another essential component, as variations in ambient pressure affect gas measurements. Temperature compensation and precise measurement are also critical, since pH and gas solubility are temperature-dependent [38]. Regulatory requirements in the United States, particularly the Clinical Laboratory Improvement Amendments (CLIA) of 1988, emphasize external quality assurance through proficiency testing [39]. CLIA mandates specific criteria for interlaboratory performance: pH within ± 0.04 of the target value, PaO₂ within ± 3 standard deviations, and PaCO₂ within $\pm 8\%$ or ± 5 mm Hg, whichever is greater [40]. These standards ensure consistent, accurate performance across laboratories and place legal and professional accountability on clinical teams. Failure to meet these standards carries potential penalties, reinforcing the need for stringent internal quality control measures and timely corrective actions [41].

Practical implementation of quality control must also balance cost and operational efficiency. CLIA regulations suggest that one concentration of control material should be analyzed every eight hours, with the full range of control concentrations tested every 24 hours [22]. Many laboratories adopt more rigorous schedules, running multiple concentrations of control for pH, PaO₂, and PaCO₂ at least once per shift or following maintenance and troubleshooting. Smaller, portable analyzers often incorporate auto quality control (QC) functions, which automatically test onboard QC materials at defined intervals to meet regulatory requirements. Electronic QC is increasingly common in devices with disposable electrode cartridges, verifying the instrument's electronic specifications prior to use [42][43]. Strict adherence to quality control protocols is not only a regulatory requirement but also a critical patient safety measure. Errors in ABG analysis can arise from improper sample handling, electrode deterioration, or instrument malfunction, leading to misinterpretation of oxygenation, ventilation, or acid-base status. By implementing comprehensive QC measures, including instrument maintenance, use of control materials, verification of electrode linearity, and adherence to regulatory mandates, laboratories ensure the integrity of ABG results. Effective lab safety protocols also extend to personnel, requiring proper handling of blood specimens, avoidance of sample contamination, and adherence to standard precautions to reduce

exposure to infectious material. Ultimately, maintaining high-quality control and lab safety in ABG testing enhances clinical reliability, supports precise therapeutic interventions, and contributes to overall patient outcomes. The combination of regulatory compliance, rigorous internal quality assurance, and modern automated QC features allows healthcare providers to confidently interpret ABG results, guide interventions in critical care, and minimize the risk of iatrogenic errors.

Enhancing Healthcare Team Outcomes

Arterial blood gas (ABG) analysis remains a cornerstone in evaluating a patient's ventilatory status, acid-base balance, and oxygenation. Beyond diagnosis, ABG testing is essential for assessing the efficacy of therapeutic interventions and monitoring disease progression in cardiopulmonary conditions, including acute respiratory failure, chronic obstructive pulmonary disease, and sepsis [44]. Despite its clinical utility, ABG analysis carries inherent risks of erroneous or inconsistent results. These discrepancies can arise from improper sample collection, handling, or pre-analytical errors, underscoring the critical need for meticulous attention to procedure and technique [27]. Accurate interpretation of ABG results depends on a combination of precise specimen collection, proper handling, and rigorous quality control of automated analyzers. Advances in machine design and quality assurance have minimized technical errors, making most inaccuracies attributable to human factors in the clinical setting. Key pre-analytical steps include using appropriate syringes with lyophilized heparin, avoiding air bubbles, ensuring adequate blood volume, and maintaining the specimen at appropriate temperatures. Failure in any of these steps can result in significant deviations in pH, PaO₂, PaCO₂, and oxygen saturation measurements, potentially affecting clinical decision-making [27].

In most hospital settings, ABG analysis is a collaborative process involving multiple healthcare professionals, such as physicians, nurses, and respiratory therapists. This interprofessional involvement necessitates effective communication, coordination, and teamwork to ensure sample integrity and accurate results. Each provider plays a distinct role in the pre-analytical, analytical, and post-analytical phases of ABG testing, from sample collection to result interpretation, emphasizing that errors often reflect breakdowns in the healthcare team's coordination rather than the technology itself [44]. Professional guidelines, such as those from the American Association for Respiratory Care, highlight best practices for ABG sampling, handling, and analysis. These guidelines address common sources of error, including inaccurate reporting of FiO₂, improper calibration for barometric pressure, temperature discrepancies, and physiological variations like hyperleukocytosis or dyshemoglobinemias, all of which may affect PaO₂ and oxygen saturation readings [44][45]. Additionally, the mode of sample transport can influence ABG accuracy. Pneumatic tube systems, for instance, can introduce air bubbles and lead to falsely elevated or decreased readings compared to manually transported specimens. Prompt delivery, correct storage, and avoidance of sample contamination are therefore essential for reliable measurements [45]. Optimizing ABG analysis through standardized protocols, interprofessional collaboration, and adherence to quality control measures enhances patient safety and clinical outcomes. Accurate blood gas assessment allows clinicians to titrate oxygen therapy, adjust ventilatory support, and guide interventions in real time. Beyond technical precision, effective team communication and role clarity are critical for ensuring that each phase of the ABG process contributes to valid, clinically actionable results. By integrating these practices, healthcare teams can minimize

errors, maximize reliability, and ultimately improve the quality of care delivered to patients with respiratory and metabolic disorders.

CONCLUSION:

Arterial blood gas analysis remains one of the most valuable tools for assessing a patient's physiological stability, particularly in acute and critical care settings. Its unique ability to simultaneously evaluate ventilation, oxygenation, and acid–base balance provides clinicians with rapid, actionable insights essential for diagnosing respiratory failure, metabolic disturbances, and circulatory compromise. Although modern non-invasive technologies have expanded monitoring capabilities, they cannot fully replace the precision of ABG measurements. The reliability of ABG results depends heavily on meticulous pre-analytical practices. Proper sample collection, avoidance of air contamination, appropriate anticoagulant use, rapid transport, and strict adherence to anaerobic conditions all play critical roles in ensuring accurate measurements. Advances in analyzer technology—including automated calibration, barometric correction, and internal quality control—have reduced technical variability, but human factors remain a key source of error. Strengthening interprofessional collaboration among clinicians, nurses, and laboratory personnel enhances the consistency and reliability of ABG interpretation. Clinically, structured interpretative approaches allow practitioners to distinguish primary disorders from compensatory responses, detect mixed acid–base abnormalities, and identify hypoxemia or ventilation defects with greater precision. By integrating ABG data with clinical context, patient history, and additional investigations, healthcare teams can optimize therapeutic interventions and improve patient outcomes across a wide spectrum of respiratory and metabolic conditions.

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