Saudi Journal of Medicine and Public Health

https://saudijmph.com/index.php/pub

https://doi.org/10.64483/202522530

Arterial Blood Gas–Guided Optimization of Mechanical Ventilation in Nursing Practice

Ruwaidat Al-Ardh Hospital Jamla Omar Hady (1) , Halimah Obaid Alradhady (2) , Samiha Saeed Moshbab Alasmari (3) , Munirah Aql Almutairi (3) , Munirah Aql Almutairi (4) , Safiah Mohammed Saghir Maghfuri (5) , Itidal Mohammed Alnam (6) , Mona Aqlaa Alenazi (7) , Sharifa Ahmed Khormi (8) , Taher Hassan Ali Sumaily (9) , Abdulrahman Zaal Saad Albalawi (10)

(1) Alqahtani, Saudi Arabia,
(2) halemaalr1212@gmail.com, Saudi Arabia,
(3) Al-Rawdah Primary Health Care Center 1, Riyadh, Ministry of Health, Saudi Arabia,
(4) Damad General Hospital – Jazan Health Cluster – Ministry of Health, Saudi Arabia,
(5) Prince Mohammed Bin Nasser Hospital – Jazan – Ministry of Health, Saudi Arabia,
(6) Itidal Mohammed Alnam, Ministry of Health, Saudi Arabia,
(7) Al Rawdah Primary Health Care Center – Ministry of Health, Saudi Arabia,
(8) Virtual Health Hospital – Ministry of Health, Saudi Arabia,
(9) Al-Najamiyah Health Center – Jazan Health Cluster – Ministry of Health, Saudi Arabia,
(10) Medina General Hospital, Ministry of Health, Saudi Arabia

Abstract

Background: Arterial blood gas (ABG) analysis is a cornerstone in managing mechanically ventilated patients, offering critical insights into oxygenation, ventilation, and acid–base balance. Nurses play a pivotal role in interpreting ABG results and adjusting ventilator settings to optimize patient outcomes.

Aim: This study aims to highlight the significance of ABG-guided ventilator optimization within nursing practice and its impact on patient safety and clinical outcomes.

Methods: A comprehensive review of physiological principles, ventilator parameters, and ABG interpretation was conducted, emphasizing evidence-based strategies for individualized ventilator adjustments. Special considerations for conditions such as COPD, ARDS, asthma, restrictive lung disease, and cardiogenic pulmonary edema were analyzed.

Results: ABG-guided interventions enable precise ventilator adjustments, improving oxygenation and ventilation while minimizing complications like volutrauma, barotrauma, and oxygen toxicity. Disease-specific strategies, including low tidal volumes for ARDS and prolonged expiratory times for obstructive disorders, were identified as critical for safe ventilation.

Conclusion: Integrating ABG analysis with ventilator management enhances patient safety, reduces complications, and supports individualized care. Interprofessional collaboration and continuous monitoring are essential for effective implementation.

Keywords: Arterial blood gas, mechanical ventilation, ventilator settings, nursing practice, critical care, oxygenation, respiratory management..

Introduction

Arterial blood gas (ABG) analysis represents a central component in the clinical management of critically ill patients receiving mechanical ventilation. Its value lies in the objective assessment of acid–base equilibrium, alveolar ventilation, and systemic oxygenation, all of which are essential parameters for evaluating respiratory function and guiding therapeutic decisions in intensive care settings.[1] Through direct measurement of arterial pH, partial pressure of carbon dioxide ($PaCO_2$), and partial pressure of oxygen (PaO_2), ABG analysis allows clinicians and nurses to determine the adequacy of ventilatory support and to identify early signs of respiratory or metabolic derangement that may compromise patient stability. In mechanically ventilated patients, deviations in $PaCO_2$ serve as a primary indicator of ventilation efficiency. Elevated $PaCO_2$ levels reflect hypoventilation and impaired carbon dioxide elimination, conditions that may necessitate modifications to ventilator parameters such as respiratory rate or tidal volume to enhance minute ventilation and restore normocapnia.[1] Conversely, abnormally low $PaCO_2$ values indicate hyperventilation, which may contribute to respiratory alkalosis and reduced cerebral blood flow, thereby requiring careful reduction of ventilatory support. ABG interpretation therefore supports precise titration of ventilation to meet individual patient needs while minimizing the risks associated with overventilation

*Corresponding author e-mail: joalqhtani@moh.gov.sa (Jamla Omar Hady Alqahtani).
Receive Date: 06 December 2025, Revise Date: 28 December 2025, Accept Date: 31 December 2025

or underventilation. Oxygenation status, as reflected by PaO2 values, is another critical determinant of ventilator management. Reduced PaO2 levels signal inadequate oxygen transfer across the alveolar-capillary membrane and may arise from conditions such as atelectasis, ventilation–perfusion mismatch, or acute lung injury. In such cases, ABG findings inform targeted adjustments, including increases in the fraction of inspired oxygen (FiO2) or the application of positive end-expiratory pressure (PEEP) to recruit collapsed alveoli and improve functional residual capacity.[1] These interventions must be carefully balanced, as excessive FiO2 exposure or inappropriate PEEP levels can contribute to oxygen toxicity or barotrauma, highlighting the importance of ongoing ABG-guided reassessment [1].

Continuous interpretation of ABG results plays a pivotal role in preventing complications associated with mechanical ventilation and in optimizing clinical outcomes. Regular monitoring enables early detection of deteriorating gas exchange, acid–base imbalance, or ventilator-induced lung injury, allowing timely corrective action.[2] Within nursing practice, the ability to correlate ABG values with ventilator settings, patient assessment, and underlying pathophysiology is fundamental to delivering safe and effective care. This process supports an individualized approach to ventilation management, ensuring that adjustments are evidence based and responsive to dynamic changes in patient condition. This activity focuses on the systematic adjustment of ventilator parameters guided by ABG analysis, underscoring the integration of physiological principles, clinical judgment, and evidence-based practice. By emphasizing individualized care and continuous evaluation, ABG-guided ventilator management contributes significantly to improved patient safety, reduced complications, and enhanced outcomes in the critical care environment.[2]

Basic Ventilator Parameters

Mechanical ventilation depends on precise adjustment of ventilator parameters to support gas exchange while minimizing harm. Each parameter influences oxygenation, ventilation, and lung mechanics. Effective management requires understanding how these settings interact with patient physiology and disease processes. In clinical practice, especially in critical care, inappropriate ventilator adjustments can worsen lung injury, prolong ventilation, or increase mortality. For this reason, mastery of basic ventilator parameters remains a core competency for healthcare professionals involved in ventilatory care. Tidal volume represents the volume of air delivered to the lungs with each mechanical breath. Clinicians usually calculate tidal volume according to predicted body weight rather than actual body weight to reduce the risk of lung overdistension. Excessive tidal volumes increase alveolar stretch and promote ventilator induced lung injury, including volutrauma and biotrauma. Evidence strongly supports the use of lower tidal volumes, particularly in patients with acute respiratory distress syndrome, where lung compliance is reduced and susceptibility to injury is high.[3] Maintaining appropriate tidal volume protects alveolar integrity and supports safer ventilation over prolonged periods. Respiratory frequency refers to the number of breaths delivered per minute. This parameter directly affects minute ventilation and carbon dioxide elimination. Increasing the respiratory rate raises minute ventilation and can reduce elevated PaCO2 levels in patients with hypoventilation. However, excessive rates shorten expiratory time and increase the risk of air trapping and intrinsic PEEP. This is especially relevant in obstructive lung diseases where expiratory flow limitation is present.[4] Effective adjustment of respiratory frequency therefore requires balancing carbon dioxide clearance with adequate time for lung emptying.

The fraction of inspired oxygen defines the concentration of oxygen delivered to the patient. It ranges from 0.21 in room air to 1.0 when delivering pure oxygen. FiO2 is often increased initially to correct hypoxemia, but prolonged exposure to high oxygen concentrations can damage alveolar cells and lead to oxygen toxicity. For this reason, clinicians aim to use the lowest FiO2 that maintains acceptable oxygenation.[5] Continuous reassessment ensures that oxygen therapy remains both effective and safe. Positive end expiratory pressure is the pressure maintained in the lungs at the end of expiration. PEEP prevents alveolar collapse, increases functional residual capacity, and improves oxygenation by enhancing alveolar recruitment. Appropriate PEEP reduces shunt and improves ventilation perfusion matching. Excessive PEEP, however, can overdistend alveoli, impair venous return, and increase the risk of barotrauma.[6] Selecting the optimal PEEP level requires careful assessment of oxygenation response and hemodynamic tolerance. Inspiratory time defines the duration of gas delivery during the inspiratory phase. Modifying inspiratory time influences mean airway pressure and gas distribution. Prolonged inspiratory time can enhance oxygen diffusion and improve oxygenation in patients with severe lung disease. At the same time, extended inspiratory periods reduce expiratory time and may promote auto PEEP if not closely monitored. Adjusting inspiratory time must therefore consider lung mechanics, disease state, and patient comfort.

Inspiratory flow represents the speed at which gas enters the lungs during inspiration. Higher flow rates shorten inspiratory time and may improve patient comfort in those with high ventilatory demand. They also increase peak airway pressures and may worsen lung stress. Lower flow rates allow more even gas distribution but may increase the work of breathing in spontaneously breathing patients. Careful adjustment of inspiratory flow helps synchronize the ventilator with patient effort and optimizes pressure dynamics within the airway. The inspiratory to expiratory ratio describes the relative duration of inspiration compared with expiration. A ratio of one to two approximates normal breathing patterns and supports adequate expiration. Altering this ratio can improve oxygenation and ventilation in selected cases. Inverse ratios increase mean airway pressure and may enhance oxygenation in severe hypoxemia. This approach carries a high risk of air trapping and hemodynamic compromise and requires close monitoring.[7]

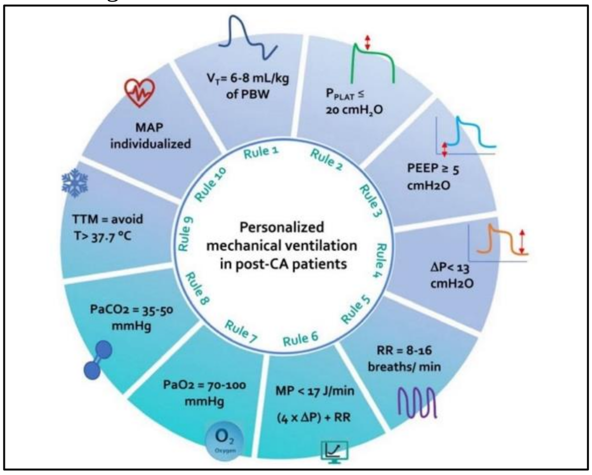

Fig. 1: Basic components for optimization of ventilator.

Basic Components of Arterial Blood Gas Analysis

Arterial blood gas analysis provides a direct assessment of oxygenation, ventilation, and acid base balance. The key variables include pH, PaCO2, PaO2, bicarbonate, and base excess. Together, these measurements offer a comprehensive picture of respiratory and metabolic function and guide clinical decision making in acute and chronic illness. Blood pH reflects hydrogen ion concentration and indicates whether the blood is acidic or alkaline. Normal values range from 7.35 to 7.45. Deviations outside this range signal acid base imbalance that can impair enzyme activity, myocardial function, and cellular metabolism. Accurate interpretation of pH forms the foundation of ABG analysis and directs further evaluation of respiratory and metabolic contributions. PaCO2 measures the partial pressure of carbon dioxide dissolved in arterial blood. Normal values range from 35 to 45 mm Hg. PaCO2 serves as the primary indicator of alveolar ventilation. Elevated levels indicate hypoventilation and carbon dioxide retention, while reduced levels indicate hyperventilation. Changes in PaCO2 directly influence pH and reflect the respiratory component of acid base regulation. PaO2 measures the partial pressure of oxygen in arterial blood and reflects the effectiveness of oxygen transfer from the alveoli to the bloodstream. Normal values range from 80 to 100 mm Hg. Reduced PaO2 indicates hypoxemia and may result from diffusion impairment, shunt, or ventilation perfusion mismatch. PaO2 assessment is essential for evaluating oxygenation status and guiding oxygen and ventilator therapy. Bicarbonate acts as a major buffer in the blood and represents the metabolic component of acid base balance. Normal values range from 22 to 26 mmol per liter. Alterations in bicarbonate concentration indicate metabolic disorders or renal compensation for respiratory disturbances. Monitoring bicarbonate trends helps distinguish acute from chronic conditions. Base excess quantifies the amount of excess or deficient base in the blood. Normal values range from minus two to plus two mmol per liter. A positive base excess reflects metabolic alkalosis, while a negative value reflects metabolic acidosis. Base excess offers a clear estimate of metabolic contribution independent of respiratory effects and supports precise diagnosis.[8]

Basic Interpretation of ABG Results

Interpreting ABG results involves identifying acid base disturbances and understanding compensatory mechanisms. The body uses respiratory and renal systems to restore equilibrium. ABG analysis allows clinicians to determine the nature and severity of imbalance and to guide appropriate intervention. Acidosis occurs when blood pH falls below normal. Respiratory acidosis presents with low pH and elevated PaCO2 and results from inadequate ventilation. Common causes include chronic obstructive pulmonary disease, central respiratory depression, and neuromuscular weakness. Metabolic acidosis presents with low pH and reduced bicarbonate and arises from conditions such as diabetic ketoacidosis, lactic acidosis, and renal failure. Alkalosis occurs when blood pH rises above normal. Respiratory alkalosis is characterized by high pH and low PaCO2 and often results from hyperventilation due to pain, anxiety, or hypoxemia. Metabolic alkalosis presents with high pH and elevated bicarbonate and may result from vomiting, diuretic therapy, or electrolyte imbalance. Compensation represents the body’s attempt to normalize pH. Respiratory compensation adjusts ventilation to correct metabolic disorders. Increased ventilation reduces PaCO2 in metabolic acidosis. Decreased ventilation raises PaCO2 in metabolic alkalosis. Renal compensation adjusts bicarbonate reabsorption and hydrogen ion excretion to correct respiratory disturbances. In chronic respiratory acidosis, the kidneys retain bicarbonate to buffer excess acid.[9] Understanding these mechanisms allows clinicians to distinguish primary disorders from compensatory responses and to deliver targeted, effective care.

Issues of Concern

Adjusting Ventilator Settings Based on Arterial Blood Gas Analysis Results

Adjustment of ventilator settings based on arterial blood gas analysis represents a critical aspect of managing mechanically ventilated patients. ABG values provide objective data on oxygenation, ventilation, and acid base status, allowing clinicians to tailor ventilator support to evolving physiological needs. Misinterpretation or delayed response to ABG abnormalities can worsen gas exchange, increase ventilator associated complications, and prolong critical illness. For this reason, careful integration of ABG findings with ventilator management remains a central concern in critical care practice. Hypoxemia is defined by a reduced arterial partial pressure of oxygen and reflects impaired oxygen transfer at the alveolar level. One of the most direct interventions for hypoxemia is increasing the fraction of inspired oxygen. This approach raises the oxygen gradient between alveoli and blood and often produces a rapid improvement in PaO2. Despite its effectiveness, sustained exposure to high oxygen concentrations can damage pulmonary tissue and promote oxygen toxicity. Clinical practice therefore emphasizes maintaining PaO2 within an acceptable target range while using the lowest FiO2 necessary. Positive end expiratory pressure offers an additional and often more protective strategy. By preventing alveolar collapse at end expiration, PEEP improves alveolar recruitment and enhances ventilation perfusion matching. Appropriate PEEP levels reduce atelectasis and improve oxygenation efficiency. Excessive PEEP, however, may overdistend alveoli and impair venous return, making titration essential. Adjustments to inspiratory time may also support oxygenation by increasing mean airway pressure and allowing more time for oxygen diffusion. Prolonged inspiratory phases shorten expiratory time and increase the risk of intrinsic PEEP if not monitored closely. In severe hypoxemia, altering the inspiratory to expiratory ratio to favor longer inspiration may further improve oxygenation. This strategy increases mean airway pressure but carries significant risks and requires vigilant monitoring to prevent air trapping and hemodynamic compromise.[10][11]

Acidosis represents another major concern identified through ABG analysis. A low arterial pH may originate from respiratory or metabolic causes, each requiring a distinct ventilatory approach. Respiratory acidosis results from inadequate alveolar ventilation and elevated PaCO2. Increasing the respiratory rate enhances minute ventilation and promotes carbon dioxide elimination, thereby improving pH. This intervention must be balanced carefully, as excessive rates reduce expiratory time and increase the likelihood of auto PEEP, particularly in patients with obstructive lung disease. Adjusting tidal volume may also support carbon dioxide removal by increasing alveolar ventilation. Larger tidal volumes improve PaCO2 clearance but raise the risk of volutrauma and barotrauma. Lung protective strategies favor modest tidal volume increases only when clearly indicated and when plateau pressures remain within safe limits. Metabolic acidosis presents with reduced bicarbonate levels and reflects systemic conditions such as renal failure or lactic acidosis. In these cases, ventilator management focuses on supporting compensatory hyperventilation while definitive treatment targets the underlying metabolic disorder. Excessive suppression of respiratory compensation may worsen acidemia and compromise cellular function. Alkalosis is characterized by an elevated arterial pH and may also be respiratory or metabolic in origin. Respiratory alkalosis develops from excessive ventilation and reduced PaCO2. This pattern often occurs in patients receiving high respiratory rates or large tidal volumes. Reducing the respiratory rate allows carbon dioxide to accumulate, correcting PaCO2 and stabilizing pH. Careful reduction is required to maintain adequate oxygenation and prevent patient discomfort or dyssynchrony. Decreasing tidal volume may further increase PaCO2, though this adjustment must ensure sufficient alveolar ventilation. Metabolic alkalosis involves elevated bicarbonate levels and commonly results from gastrointestinal losses or diuretic therapy. Ventilator adjustments play a supportive role by avoiding unnecessary hyperventilation that could exacerbate alkalemia. Correction of the underlying metabolic disturbance remains the primary treatment strategy.[12][13] Overall, ventilator adjustments guided by arterial blood gas analysis demand continuous reassessment and clinical judgment. Each change in ventilator settings influences pulmonary mechanics and systemic physiology. Effective management balances correction of ABG abnormalities with prevention of ventilator induced injury, supporting safe and individualized patient care.

Clinical Significance

Considerations for Adjusting Ventilator Settings in Special Circumstances

Adjustment of ventilator settings according to the underlying respiratory condition of the patient has major clinical significance in critical care practice. Mechanical ventilation does not represent a uniform intervention that can be applied identically to all patients. Instead, ventilator management must reflect the specific pathophysiology, lung mechanics, and gas exchange abnormalities associated with each disease state. Failure to individualize ventilator settings may worsen lung injury, increase complications such as barotrauma or hemodynamic instability, and prolong the duration of mechanical ventilation. In special respiratory circumstances, careful interpretation of clinical findings and arterial blood gas results is essential to guide safe and effective ventilatory support. Chronic obstructive pulmonary disease is a progressive inflammatory disorder characterized by persistent airflow limitation and abnormal inflammatory responses of the lungs to noxious particles or gases. The disease includes emphysema, which involves destruction of alveolar walls and loss of elastic recoil, and chronic bronchitis, which is defined by mucus hypersecretion and airway inflammation. Patients with COPD who require mechanical ventilation present unique challenges due to increased airway resistance, prolonged expiratory time constants, and high physiological dead space. These factors predispose patients to hypercapnia, air trapping, and dynamic hyperinflation. During mechanical ventilation, inadequate expiratory time can result in intrinsic PEEP, increased intrathoracic pressure, and compromised venous return. These changes increase the work of breathing and may precipitate hypotension or ventilator induced lung injury. Appropriate adjustment of ventilator settings is therefore critical to reduce these risks and support effective gas exchange.[14]

In mechanically ventilated patients with chronic obstructive pulmonary disease, tidal volume selection must prioritize lung protection. Lower tidal volumes based on ideal body weight reduce the risk of alveolar overdistension and barotrauma, particularly in lungs with heterogeneous compliance. Excessive tidal volumes increase peak and plateau pressures and worsen dynamic hyperinflation. Management of PEEP in COPD requires careful titration. Although external PEEP can help counterbalance intrinsic PEEP and reduce inspiratory effort, excessive PEEP may exacerbate air trapping and increase intrathoracic pressure. Determining optimal PEEP involves balancing improvements in oxygenation against the risk of worsening hyperinflation. Respiratory rate adjustment plays a central role in COPD ventilation. Lower respiratory rates allow prolonged expiratory times, facilitating more complete lung emptying and reducing intrinsic PEEP. This strategy improves carbon dioxide clearance while minimizing the mechanical complications associated with rapid ventilation.[15] Acute respiratory distress syndrome represents a distinct and severe form of respiratory failure characterized by diffuse alveolar damage, increased pulmonary capillary permeability, and reduced lung compliance. ARDS results in profound hypoxemia that is often refractory to conventional oxygen therapy. The pathological process involves inflammatory injury to the alveolar-capillary membrane, leading to alveolar flooding, surfactant dysfunction, and widespread atelectasis. Patients with ARDS are particularly vulnerable to ventilator induced lung injury due to the presence of nonuniform lung involvement, where relatively normal lung regions coexist with collapsed or consolidated areas. Mechanical ventilation in this setting must achieve adequate oxygenation while minimizing further lung damage.[16]

Lung protective ventilation strategies form the foundation of ventilator management in ARDS. The use of low tidal volumes based on predicted body weight reduces alveolar overdistension and limits volutrauma. This approach has been shown to reduce mortality and improve clinical outcomes in ARDS patients. Application of PEEP is essential to prevent repetitive alveolar collapse and reopening, a phenomenon known as atelectrauma. Appropriate PEEP improves functional residual capacity and oxygenation by recruiting collapsed alveoli. However, excessive PEEP may overdistend compliant lung units and impair cardiac output. Monitoring plateau pressure provides insight into lung compliance and helps guide safe ventilator settings. Maintaining plateau pressures below 30 cm H2O reduces the risk of barotrauma. Driving pressure, defined as the difference between plateau pressure and PEEP, has emerged as a key determinant of ventilator induced lung injury. Lower driving pressures are associated with improved survival, highlighting the importance of minimizing stress applied to the lung parenchyma during ventilation.[17][18] Bronchial asthma is a chronic inflammatory airway disease characterized by reversible airflow obstruction, airway hyperresponsiveness, and bronchial smooth muscle constriction. Acute severe asthma may progress to respiratory failure requiring mechanical ventilation. During mechanical ventilation, asthmatic patients are highly susceptible to dynamic hyperinflation due to severe expiratory flow limitation. Air trapping leads to intrinsic PEEP, elevated intrathoracic pressures, and increased risk of barotrauma and hemodynamic compromise. Ventilator management in asthma focuses on preventing excessive airway pressures while allowing adequate time for exhalation.[19]

In mechanically ventilated patients with bronchial asthma, lower tidal volumes and reduced respiratory rates are essential to prolong expiratory time and limit air trapping. Shorter inspiratory times further support adequate exhalation and reduce mean airway pressure. High levels of PEEP are generally avoided, as they can worsen hyperinflation and increase the risk of hypotension. Ventilator settings must be continuously reassessed in conjunction with bronchodilator therapy and clinical improvement to avoid complications associated with overventilation.[20] Restrictive lung diseases encompass a broad group of disorders characterized by reduced lung compliance and limited lung expansion. These conditions may arise from intrinsic lung pathology, such as interstitial lung diseases, or from extrinsic factors including neuromuscular disorders, pleural disease, obesity, or chest wall deformities. Reduced compliance leads to decreased lung volumes and impaired oxygenation. Patients with restrictive lung disease often exhibit rapid shallow breathing patterns and increased breathing work. Mechanical ventilation aims to support oxygenation while minimizing pressure related lung injury.[21] Ventilator management in restrictive lung disease typically requires lower tidal volumes to avoid overdistension of stiff lungs. Even modest increases in volume can result in significant pressure changes due to poor compliance. Higher levels of PEEP may be necessary to maintain alveolar recruitment and improve oxygenation. Careful monitoring of plateau pressure and oxygenation response is essential to prevent barotrauma. Ventilator settings must reflect the balance between improving gas exchange and avoiding excessive airway pressures that could worsen lung injury.[22]

Cardiogenic pulmonary edema is a condition characterized by accumulation of fluid within the alveolar spaces due to elevated hydrostatic pressures in the pulmonary circulation. This process commonly results from left ventricular dysfunction, valvular heart disease, or acute cardiac events. Fluid accumulation impairs gas exchange by increasing diffusion distance and reducing alveolar ventilation. Patients often present with severe hypoxemia and respiratory distress that may require ventilatory support.[23] Ventilator management in cardiogenic pulmonary edema focuses on improving oxygenation and reducing pulmonary congestion. Application of higher PEEP levels increases intrathoracic pressure, which can reduce venous return and lower pulmonary capillary hydrostatic pressure, thereby decreasing fluid transudation into the alveoli. PEEP also improves oxygenation by recruiting flooded alveoli and enhancing ventilation perfusion matching. Noninvasive ventilation plays a significant role in this population by reducing the need for endotracheal intubation, decreasing work of breathing, and improving cardiac function. When invasive ventilation is required, careful hemodynamic monitoring is essential to avoid compromising cardiac output.[24] Overall, the clinical significance of adjusting ventilator settings in special respiratory circumstances lies in the need for individualized, physiology driven management. Each disease state presents distinct challenges that influence lung mechanics, gas exchange, and hemodynamic stability. Effective ventilator adjustment requires continuous assessment, integration of arterial blood gas analysis, and understanding of disease specific pathophysiology. Tailoring ventilatory support to these special conditions improves patient safety, reduces ventilator associated complications, and enhances overall outcomes in critically ill patients.

Nursing, Allied Health, and Interprofessional Team Interventions

Mechanical ventilation is a complex, high-risk therapy that demands coordinated intervention from an interprofessional healthcare team to ensure patient safety and optimal outcomes. Nurses, respiratory therapists, physicians, and advanced practice providers each contribute distinct yet complementary expertise. Among these professionals, critical care nurses and respiratory therapists hold a central operational role, as they are continuously present at the bedside and directly responsible for implementing ventilator adjustments based on patient assessment and arterial blood gas interpretation. Their ability to recognize early physiological changes and respond promptly is essential for maintaining effective gas exchange and preventing deterioration. Critical care nurses play a key role in translating ABG results into practical bedside actions. They routinely assess pH, $PaCO_2$, and PaO_2 values in relation to ventilator settings and patient condition. A rising $PaCO_2$ may prompt collaborative discussion regarding respiratory rate or tidal volume adjustments, while declining PaO_2 values may lead to coordinated interventions involving FiO_2 or PEEP modification. Nurses also integrate ABG data with clinical findings such as chest expansion, breath sounds, work of breathing, and hemodynamic stability. This holistic approach allows for individualized care that reflects both numerical data and patient response [25].

Respiratory therapists provide specialized expertise in ventilator mechanics and lung physiology. Their role includes setting up ventilators, optimizing modes of ventilation, and fine-tuning parameters to align with disease-specific goals. In collaboration with nurses, respiratory therapists assess ventilator waveforms, airway pressures, and patient-ventilator synchrony. Their understanding of how ventilator changes influence alveolar ventilation and oxygenation is critical when responding to abnormal ABG results. Together, nurses and respiratory therapists ensure that ventilator adjustments are precise, timely, and physiologically appropriate. Advanced practice nurses and physicians provide clinical oversight and are responsible for higher-level decision making. They evaluate trends in ABG values, imaging findings, and overall clinical trajectory to guide long-term ventilator strategies. Decisions regarding escalation or de-escalation of ventilatory support, changes in ventilation mode, or readiness for weaning are made collaboratively. This shared decision-making process reduces error, promotes consistency, and ensures that ventilator management aligns with evidence-based standards and patient goals. Implementation of safety protocols is a critical component of interprofessional intervention. Mechanical ventilation carries risks such as barotrauma, volutrauma, oxygen toxicity, and ventilator-associated lung injury. Preventive strategies include adherence to lung-protective ventilation principles, avoidance of excessive airway pressures, and judicious use of FiO_2. Nurses and respiratory therapists are instrumental in enforcing these safeguards through continuous monitoring and prompt reporting of concerning trends. Protocol-driven care enhances consistency and reduces variability in ventilator management [25].

Education and training underpin effective interprofessional interventions. Regular competency-based training ensures that team members remain proficient in ventilator management, ABG interpretation, and emergency response. Simulation-based education allows healthcare professionals to practice managing acute ventilatory crises in a controlled environment, improving confidence and team communication. Ongoing professional development supports integration of emerging evidence into clinical practice, ensuring that ventilator care reflects current standards.[25] Ultimately, interprofessional interventions in mechanical ventilation rely on shared responsibility, mutual respect, and clear communication. When nurses, respiratory therapists, and clinicians function as a cohesive unit, ventilator adjustments based on ABG results become safer, more effective, and more responsive to patient needs.

Nursing, Allied Health, and Interprofessional Team Monitoring

Continuous monitoring is a cornerstone of safe and effective mechanical ventilation and requires close collaboration among nursing, allied health, and medical professionals. Patients receiving ventilatory support are physiologically unstable and vulnerable to rapid changes in respiratory and hemodynamic status. Effective monitoring ensures early detection of deterioration, timely adjustment of ventilator settings, and prevention of complications. Nurses, respiratory therapists, and critical care clinicians each play a vital role in this ongoing surveillance process. Critical care nurses are primarily responsible for continuous bedside monitoring. They assess vital signs, oxygen saturation, respiratory effort, and level of consciousness while simultaneously evaluating ventilator parameters and alarms. Nurses correlate these observations with ABG results to identify trends that may indicate worsening ventilation or oxygenation. For example, increasing $PaCO_2$ values combined with rising respiratory effort may signal inadequate ventilatory support or patient-ventilator dyssynchrony. Early recognition allows for prompt intervention and escalation when necessary. Respiratory therapists contribute focused monitoring of ventilator performance and pulmonary mechanics. They assess airway pressures, tidal volumes, minute ventilation, and flow patterns to ensure that ventilator delivery matches prescribed settings. Analysis of ventilator waveforms provides insight into lung compliance, airway resistance, and the presence of auto-PEEP. When ABG abnormalities arise, respiratory therapists help determine whether ventilator mechanics or patient factors are contributing to the imbalance. Their expertise supports precise and evidence-based adjustments [26].

Arterial blood gas monitoring remains central to evaluating ventilation effectiveness. Interprofessional collaboration is essential in determining the frequency of ABG sampling, interpreting results, and responding appropriately. Nurses often obtain arterial samples and recognize critical values, while respiratory therapists and clinicians assist in interpretation and planning. Serial ABG measurements allow the team to assess response to ventilator changes and to distinguish transient abnormalities from persistent dysfunction. This iterative process supports dynamic and individualized ventilator management.[26] Advanced practice providers and physicians synthesize monitoring data to guide broader clinical decisions. They evaluate ABG trends alongside radiographic findings, laboratory results, and patient trajectory. Monitoring informs decisions related to sedation adjustment, ventilator weaning, or escalation to advanced therapies. Clear communication of monitoring findings among team members ensures that decisions are timely and coordinated. Interprofessional monitoring also includes vigilance for ventilator-associated complications. Nurses monitor for signs of barotrauma, infection, and oxygen toxicity, while respiratory therapists track airway pressures and lung compliance. Early detection of complications allows for rapid intervention and mitigation of harm. Consistent documentation and structured handover communication ensure continuity of monitoring across shifts and disciplines. Effective monitoring depends on teamwork, standardized protocols, and shared situational awareness. When nurses, respiratory therapists, and clinicians actively exchange information and collaborate in interpreting data, patient safety improves. Interprofessional monitoring transforms raw physiological data into meaningful clinical action, supporting stable ventilation, reducing complications, and improving outcomes for mechanically ventilated patients.[26]

Conclusion:

ABG-guided ventilator management represents a vital component of critical care, ensuring that ventilatory support aligns with dynamic patient needs. By interpreting ABG values in conjunction with clinical assessment, nurses and respiratory therapists can implement timely and precise adjustments to ventilator parameters, thereby optimizing gas exchange and preventing complications. Disease-specific strategies, such as lung-protective ventilation in ARDS and prolonged expiratory times in obstructive disorders, underscore the necessity of individualized care. Interprofessional collaboration, continuous monitoring, and adherence to evidence-based protocols further enhance safety and effectiveness. Ultimately, ABG-guided ventilator optimization not only improves physiological stability but also reduces the incidence of ventilator-induced lung injury, shortens the duration of mechanical ventilation, and promotes better patient outcomes. This approach exemplifies the integration of scientific principles with clinical judgment, reinforcing its indispensable role in modern critical care nursing practice.

References:

1. Kelly AM, Klim S, Rees SE. Agreement between mathematically arterialised venous versus arterial blood gas values in patients undergoing non-invasive ventilation: a cohort study. Emergency medicine journal : EMJ. 2014 Oct:31(e1):e46-9. doi: 10.1136/emermed-2013-202879.

2. Menzel M, Soukup J, Henze D, Engelbrecht K, Senderreck M, Scharf A, Rieger A, Grond S. Experiences with continuous intra-arterial blood gas monitoring: precision and drift of a pure optode-system. Intensive care medicine. 2003 Dec:29(12):2180-2186. doi: 10.1007/s00134-003-1962-1.
3. Villar J, Kacmarek RM, Pérez-Méndez L, Aguirre-Jaime A. A high positive end-expiratory pressure, low tidal volume ventilatory strategy improves outcome in persistent acute respiratory distress syndrome: a randomized, controlled trial. Critical care medicine. 2006 May:34(5):1311-8
4. de Durante G, del Turco M, Rustichini L, Cosimini P, Giunta F, Hudson LD, Slutsky AS, Ranieri VM. ARDSNet lower tidal volume ventilatory strategy may generate intrinsic positive end-expiratory pressure in patients with acute respiratory distress syndrome. American journal of respiratory and critical care medicine. 2002 May 1:165(9):1271-4
5. Kor CT, Lin KH, Wang CH, Lin JF, Kuo CD. Ventilator Parameters in the Diagnosis and Prognosis of Acute Respiratory Distress Syndrome in Postoperative Patients: A Preliminary Study. Diagnostics (Basel, Switzerland). 2021 Apr 3:11(4):. doi: 10.3390/diagnostics11040648.
6. Blankman P, Hasan D, Erik G, Gommers D. Detection of 'best' positive end-expiratory pressure derived from electrical impedance tomography parameters during a decremental positive end-expiratory pressure trial. Critical care (London, England). 2014 May 10:18(3):R95. doi: 10.1186/cc13866.
7. Liyanage I, Dassanayaka DARK, Chellapillai FMD, Liyanage E, Rathnayake S, Rikas M, Mayooran S. Manual and ventilator hyperinflation parameters used by intensive care physiotherapists in Sri Lanka: An online survey. PloS one. 2024:19(5):e0297880. doi: 10.1371/journal.pone.0297880.
8. Balzanelli MG, Distratis P, Lazzaro R, Pham VH, Del Prete R, Dipalma G, Inchingolo F, Aityan SK, Hoang LT, Palermo A, Nguyen KCD, Gargiulo Isacco C. The importance of arterial blood gas analysis as a systemic diagnosis approach in assessing and preventing chronic diseases, from emergency medicine to the daily practice. European review for medical and pharmacological sciences. 2023 Dec:27(23):11653-11663. doi: 10.26355/eurrev_202312_34603.
9. Zadek F, Danieli A, Brusatori S, Giosa L, Krbec M, Antolini L, Fumagalli R, Langer T. Combining the Physical-Chemical Approach with Standard Base Excess to Understand the Compensation of Respiratory Acid-Base Derangements: An Individual Participant Meta-analysis Approach to Data from Multiple Canine and Human Experiments. Anesthesiology. 2024 Jan 1:140(1):116-125. doi: 10.1097/ALN.0000000000004751.
10. Florio G, Zanella A, Slobod D, Guzzardella A, Protti I, Carlesso E, Canakoglu A, Fumagalli J, Scaravilli V, Colombo SM, Caccioppola A, Brioni M, Pesenti AM, Grasselli G. Impact of Positive End-Expiratory Pressure and FiO(2) on Lung Mechanics and Intrapulmonary Shunt in Mechanically Ventilated Patients with ARDS Due to COVID-19 Pneumonia. Journal of intensive care medicine. 2024 May:39(5):420-428. doi: 10.1177/08850666231210485.
11. Pan L, Yang L, Gao L, Zhao Z, Zhang J. Individualized PEEP without Recruitment Maneuvers Improves Intraoperative Oxygenation: A Randomized Controlled Study. Bioengineering (Basel, Switzerland). 2023 Oct 9:10(10):. doi: 10.3390/bioengineering10101172.
12. Arena A, Miller E. Respiratory Acid-Base Disorders. Emergency medicine clinics of North America. 2023 Nov:41(4):863-875. doi: 10.1016/j.emc.2023.06.009.
13. Sanghavi SF, Swenson ER. Arterial Blood Gases and Acid-Base Regulation. Seminars in respiratory and critical care medicine. 2023 Oct:44(5):612-626. doi: 10.1055/s-0043-1770341.
14. Cornelius T. Clinical guideline highlights for the hospitalist: GOLD COPD update 2024. Journal of hospital medicine. 2024 Sep:19(9):818-820. doi: 10.1002/jhm.13416.
15. Liao KM, Lu HY, Chen CY, Kuo LT, Tang BR. The impact of comorbidities on prolonged mechanical ventilation in patients with chronic obstructive pulmonary disease. BMC pulmonary medicine. 2024 May 25:24(1):257. doi: 10.1186/s12890-024-03068-9.
16. Camporota L, Calfee CS, Grasselli G. Acute respiratory distress syndrome guidelines: oceans apart or rivers that merge? Intensive care medicine. 2024 Jun:50(6):941-943. doi: 10.1007/s00134-024-07435-2.
17. Qadir N, Sahetya S, Munshi L, Summers C, Abrams D, Beitler J, Bellani G, Brower RG, Burry L, Chen JT, Hodgson C, Hough CL, Lamontagne F, Law A, Papazian L, Pham T, Rubin E, Siuba M, Telias I, Patolia S, Chaudhuri D, Walkey A, Rochwerg B, Fan E. An Update on Management of Adult Patients

with Acute Respiratory Distress Syndrome: An Official American Thoracic Society Clinical Practice Guideline. American journal of respiratory and critical care medicine. 2024 Jan 1:209(1):24-36. doi: 10.1164/rccm.202311-2011ST.

18. Grasselli G, Calfee CS, Camporota L, Poole D, Amato MBP, Antonelli M, Arabi YM, Baroncelli F, Beitler JR, Bellani G, Bellingan G, Blackwood B, Bos LDJ, Brochard L, Brodie D, Burns KEA, Combes A, D'Arrigo S, De Backer D, Demoule A, Einav S, Fan E, Ferguson ND, Frat JP, Gattinoni L, Guérin C, Herridge MS, Hodgson C, Hough CL, Jaber S, Juffermans NP, Karagiannidis C, Kesecioglu J, Kwizera A, Laffey JG, Mancebo J, Matthay MA, McAuley DF, Mercat A, Meyer NJ, Moss M, Munshi L, Myatra SN, Ng Gong M, Papazian L, Patel BK, Pellegrini M, Perner A, Pesenti A, Piquilloud L, Qiu H, Ranieri MV, Riviello E, Slutsky AS, Stapleton RD, Summers C, Thompson TB, Valente Barbas CS, Villar J, Ware LB, Weiss B, Zampieri FG, Azoulay E, Cecconi M, European Society of Intensive Care Medicine Taskforce on ARDS. ESICM guidelines on acute respiratory distress syndrome: definition, phenotyping and respiratory support strategies. Intensive care medicine. 2023 Jul:49(7):727-759. doi: 10.1007/s00134-023-07050-7.
19. Gayen S, Dachert S, Lashari BH, Gordon M, Desai P, Criner GJ, Cardet JC, Shenoy K. Critical Care Management of Severe Asthma Exacerbations. Journal of clinical medicine. 2024 Feb 1:13(3):. doi: 10.3390/jcm13030859.
20. Oddo M, Feihl F, Schaller MD, Perret C. Management of mechanical ventilation in acute severe asthma: practical aspects. Intensive care medicine. 2006 Apr:32(4):501-10
21. Martinez-Pitre PJ, Sabbula BR, Cascella M. Restrictive Lung Disease. StatPearls. 2025 Jan
22. Luo F, Annane D, Orlikowski D, He L, Yang M, Zhou M, Liu GJ. Invasive versus non-invasive ventilation for acute respiratory failure in neuromuscular disease and chest wall disorders. The Cochrane database of systematic reviews. 2017 Dec 4:12(12):CD008380. doi: 10.1002/14651858.CD008380.pub2.
23. Malek R, Soufi S. Pulmonary Edema. StatPearls. 2025 Jan
24. Zanza C, Saglietti F, Tesauro M, Longhitano Y, Savioli G, Balzanelli MG, Romenskaya T, Cofone L, Pindinello I, Racca G, Racca F. Cardiogenic Pulmonary Edema in Emergency Medicine. Advances in respiratory medicine. 2023 Oct 13:91(5):445-463. doi: 10.3390/arm91050034.
25. Tesseyman S, Poulsen T, Rainsdon-Meek S, Leary H, Sorensen U, Plummer K. Decision-based learning for teaching arterial blood gas analysis. International journal of nursing education scholarship. 2023 Jan 1:20(1):. doi: 10.1515/ijnes-2023-0028.
26. Pruitt B. Strategies for interpreting arterial blood gases. Nursing. 2024 Jan 1:54(1):16-21. doi: 10.1097/01.NURSE.0000995560.71478.3f