Using ABGs to optimize mechanical ventilation

Three case studies illustrate how arterial blood gas analyses can guide appropriate ventilator strategy.

By Jin Xiong Lian, BSN, RN, CNS

An arterial blood gas (ABG) analysis can tell you about the patient’s oxygenation (via PaO₂ and SaO₂), acid-base balance, pulmonary function (through the PaCO₂), and metabolic status. This article focuses on translating ABG information into clinical benefits, with three case scenarios that focus on using ABGs to manage mechanical ventilation.

Endotracheal (ET) intubation and mechanical ventilation may be prescribed for patients who can’t maintain adequate oxygenation or ventilation, or who need airway protection. The goal of mechanical ventilation is to improve oxygenation and ventilation as well as rest fatigued respiratory muscles. Mechanical ventilation is supportive therapy because it doesn’t treat the causes of the illness and associated complications. However, ventilator support buys time for other therapeutic interventions to work and lets the body reestablish homeostasis.

When using this lifesaving intervention, clinicians should take steps to avoid or minimize
ventilator-induced lung injury (VILI—more on this later). Patients should be weaned from ventilatory support if their condition permits.

A critically ill patient’s condition can change rapidly and dramatically, and the need for ventilatory support in terms of oxygenation or minute ventilation can vary at different stages of the illness. ABG analysis is an indispensable diagnostic tool for monitoring the patient’s condition and evaluating the response to various interventions. By reviewing the patient’s ABGs and condition, clinicians can adjust ventilator settings to improve oxygenation, ventilation, and acid-base balance, or wean the patient from ventilatory support.1-7

Normal values for ABGs vary slightly among labs, but in general are:
- PaO2, 80 to 100 mm Hg
- SaO2, 95% to 100%
- pH, 7.35 to 7.45
- PaCO2, 35 to 45 mm Hg
- HCO3\(^{-}\), 22 to 26 mEq/L
- serum lactate, normally less than 2 mmol/L in critically ill patients.4-9

Types of acid-base imbalances presents an overview of various acid-base disorders.

For mechanically ventilated patients, the key means of improving oxygenation are to increase the fraction of inspired oxygen (FiO2) or increase positive end-expiratory pressure (PEEP). Remember that a patient’s minute ventilation equals respiratory rate times tidal volume (VT). Therefore, any intervention that alters respiratory rate or VT can help manage hypercapnia or hypocapnia and rectify acid-base imbalance.1-5,10-12

- In volume control mode ventilation, increasing the VT, respiratory rate, or both will reduce PaCO2 and improve ventilation.3,10-12
- In pressure control mode ventilation, interventions to improve ventilation include increasing the inspiratory pressure, respiratory rate, or both; prolonging inspiratory time; and decreasing airway resistance by administering bronchodilators, suctioning airway secretions, or using a larger diameter ET tube.3,10-14
- In pressure support mode ventilation, interventions to improve ventilation include increasing the pressure support level, and decreasing airway resistance by administering bronchodilators, suctioning airway secretions, or using a larger diameter ET tube.3,10-13

**Case 1: Meeting changing needs**

A 52-year-old man was admitted to the ICU via the ED due to respiratory distress and hypotension secondary to neutropenic sepsis. The patient required fluid resuscitation and I.V. positive inotropes. His medical history included diarrhea and fever for the past 3 weeks, diffuse large B-cell lymphoma treated with chemotherapy, hepatitis C virus, alcohol abuse, and cirrhosis.

Follow the five-step approach (see *Steps to interpreting ABGs*) to analyze his admission ABGs:

- PaO2 of 81.3 mm Hg (while on supplemental oxygen at 6 L/minute via simple face mask) indicates his oxygenation was adequate
- pH of 7.14 indicates acidosis
- PaCO2 of 41.8 mm Hg indicates his minute ventilation is adequate for his metabolic status
- HCO3\(^{-}\) of 13.8 mmol/L reflects a metabolic alteration toward acidosis
- serum lactate level of 5.8 mmol/L indicates hyperlactatemia.

The patient’s metabolic alteration moves his pH toward acidosis, but he has no respiratory derangement. Specifically, his PaCO2 and PaO2 values show that his respiratory system is able to maintain adequate ventilation and oxygenation with supplemental oxygen of 6 L/minute. This ABG profile shows uncompensated metabolic acidosis.

The patient’s metabolic acidosis was most likely caused by diarrhea, and aggravated significantly by sepsis and septic shock. With an acute episode of septic shock, the patient’s admission ABGs don’t demonstrate respiratory compensation for metabolic acidosis, although it usually occurs fairly quickly.

Lactic acidosis is characterized by hyperlactatemia (greater than 5 mmol/L) associated with metabolic acidosis.6 The patient’s lactate and pH values confirm the diagnosis of lactic acidosis. Hypotension decreases tissue perfusion and impairs oxygen delivery. Tissue hypoxia leads to anaerobic metabolism and increases lactate production. Hepatic dysfunction reduces lactate clearance.1,6 Therefore, the patient’s lactic acidosis was most likely caused by septic shock and the pre-existing cirrhosis.

In addition to antibiotics, the patient received continued I.V. fluid resuscitation and positive inotropes to maintain his mean arterial pressure (MAP) greater than 70 mm Hg. Because his oxygenation and ventilation were adequate,
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mechanical ventilatory support wasn’t initiated at this stage, but he continued to receive supplemental oxygen.

Three-and-a-half hours after his last ABG, a new ABG analysis showed: pH, 7.29; PaCO₂, 35.3 mm Hg; PaO₂, 99.7 mm Hg; HCO₃⁻, 17 mEq/L; and lactate, 5.77 mmol/L. He had less profound uncompensated metabolic acidosis with a marginal decrease in lactate level, but had developed signs of increased work of breathing: restlessness, shortness of breath, accessory muscle use, and diaphoresis. Because the patient was at increased risk for respiratory muscle fatigue, he was put on noninvasive ventilation with continuous positive airway pressure (CPAP) of 10 cm H₂O.

After the patient had been on CPAP for 4 hours, the ABG analysis showed: pH, 7.34; PaCO₂, 32.4 mm Hg; PaO₂, 95.9 mm Hg; HCO₃⁻, 19 mEq/L; and lactate, 6.7 mmol/L. The drop in the PaCO₂ level to below the normal range suggests that the patient was hyperventilating to blow off more carbon dioxide and raise pH. In other words, his respiratory system is compensating for the metabolic acidosis. Now, the diagnosis is partially compensated metabolic acidosis. However, his lactate level is still quite high. Hyperlactatemia may indicate inadequate tissue perfusion, but the patient’s MAP has been maintained greater than 70 mm Hg and his urine output is more than 0.5 mL/kg/hour, indicating that his tissue perfusion is adequate. The slow reduction of his hyperlactatemia is most likely due to impaired liver function.

The patient was weaned off CPAP and humidified oxygen at 6 L/minute was administered via simple face mask. On the next day, his ABGs were pH, 7.41; PaCO₂, 34.2 mm Hg; PaO₂, 90.7 mm Hg; HCO₃⁻, 20 mEq/L; and lactate, 5.41 mmol/L. His metabolic derangement had been ameliorated significantly by establishing and maintaining adequate tissue perfusion. Moreover, acid-base balance had been restored by his respiratory compensation. At this stage, the diagnosis was fully compensated metabolic acidosis.

On the fourth day postadmission, the patient’s temperature increased to 103.5°F (39.7°C) and his oxygenation dropped. An ABG analysis revealed pH, 7.34; PaCO₂, 52.6 mm Hg; PaO₂, 60.7 mm Hg (indicating hypoxemia); HCO₃⁻, 27.6 mEq/L; and lactate, 2.44 mmol/L. Fever increases oxygen consumption and carbon dioxide production. The patient’s respiratory system was unable to maintain adequate oxygenation and ventilation to meet his metabolic demand, as shown by the hypoxemia and hypercapnia. His carbon dioxide retention led to respiratory acidosis. On the other hand, his metabolic process

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<th>Types of acid-base imbalances⁶,⁷</th>
<th>pH</th>
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was attempting to elevate the pH, which suggested metabolic compensation for his respiratory acidosis. The above ABG profile is consistent with partially compensated respiratory acidosis. A marked reduction in lactate levels is the result of decreased lactate production and increased lactate clearance due to improved tissue perfusion since admission.

Bilevel positive airway pressure (BiPAP) refers to setting inspiratory positive airway pressure (IPAP) and expiratory positive airway pressure (EPAP) separately. The gap between IPAP and EPAP creates a pressure support. Compared with CPAP, BiPAP is more effective in eliminating carbon dioxide because of the pressure support generating by the gap between IPAP and EPAP. Therefore, BiPAP (IPAP, 15 cm H₂O; EPAP, 7 cm H₂O) with an F(IO₂) of 0.40, was started to treat his hypoxemia and hypercapnia as well as resting respiratory muscles. With the above settings, a pressure support of 8 cm H₂O is generated (15 cm H₂O – 7 cm H₂O = 8 cm H₂O). Also, antibiotics were changed based on the latest culture and sensitivity results.

After half an hour, the patient’s ABG showed pH, 7.39; PaCO₂, 42.1 mm Hg; PaO₂, 90.1 mm Hg; HCO₃⁻, 25.4 mEq/L; and lactate, 2.40 mmol/L, which suggested his hypoxemia, carbon dioxide retention, and respiratory acidosis had all been corrected.

Partially compensated respiratory acidosis recurred, which could be the result of inadequate spontaneous breathing and drowsiness. Because of the patient’s increasing risk of BiPAP intolerance, he was endotracheally intubated and ventilated with pressure support mode ventilation with the following settings: FiO₂, 0.40; PEEP, 10 cm H₂O; and pressure support, 16 cm H₂O.

The next day, his ABG analysis while on BiPAP was pH, 7.27; PaCO₂, 58.9 mm Hg; PaO₂, 89.5 mm Hg; HCO₃⁻, 26.8 mEq/L; and lactate, 1.78 mmol/L. Partially compensated respiratory acidosis recurred, which could be the result of inadequate spontaneous breathing and drowsiness. Because of the patient’s increasing risk of BiPAP intolerance, he was endotracheally intubated and ventilated with pressure support mode ventilation with the following settings: F(IO₂) 0.40; PEEP, 10 cm H₂O; and pressure support, 16 cm H₂O.

Thirteen hours later, the patient’s ABGs were pH, 7.51; PaCO₂, 34.1 mm Hg; PaO₂, 99.2 mm Hg; HCO₃⁻, 26.8 mEq/L; and lactate, 2.39 mmol/L. Pressure support ventilation augments spontaneous tidal volume and blows off more carbon dioxide, and his most recent ABGs showed that the ventilator support had turned his respiratory acidosis to alkalosis. Because both respiratory and metabolic alterations moved pH toward alkalosis, he developed mixed respiratory and metabolic alkalosis.

Two days later, the patient’s ventilator settings had been weaned to F(IO₂) 0.30; PEEP, 10 cm H₂O; and pressure support, 12 cm H₂O. An ABG showed pH, 7.59; PaCO₂, 28.4 mm Hg; PaO₂, 156.3 mm Hg; HCO₃⁻, 26.6 mEq/L; and lactate, 1.60 mmol/L. His mixed respiratory and metabolic alkalosis was worsening. The decrease of PaCO₂ levels from 34.1 to 28.4 mm Hg suggested he had been overventilated. Hypocapnia and respiratory alkalosis can be caused by pain, agitation, severe anemia, hypoxia, brainstem injury, or excessive mechanical ventilation. With this case, the absence of pain, agitation, anemia, and other conditions suggest the most likely cause for his hyperventilation would be over-ventilation caused by pressure support.

Consequently, both pressure support and PEEP were reduced to 5 cm H₂O. In less than 2 hours, a repeat ABG showed pH, 7.49; PaCO₂, 37.2 mm Hg; PaO₂, 96.9 mm Hg; HCO₃⁻, 28.2 mEq/L; and lactate, 1.53 mmol/L. Lowering the pressure support corrected his hypocapnia and eliminated respiratory alkalosis. Now, he only had uncompensated metabolic alkalosis.

Three days later, he was extubated and placed on an air-entrainment (Venturi) mask with an F(IO₂) of 0.30. The patient’s ABGs were now pH, 7.46; PaCO₂, 42.4 mm Hg; PaO₂, 114.8 mm Hg; HCO₃⁻, 29.8 mEq/L; and lactate, 1.10 mmol/L, reflecting a minor uncompensated metabolic alkalosis. Because hepatic dysfunction reduces the production

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Steps to interpreting ABGs

Follow this five-step approach to interpreting your patient’s ABGs.

1. Is the patient hypoxemic? Look at the PaO₂ and SaO₂.
2. What is the acid-base balance? Check the pH.
3. How is the patient’s pulmonary ventilation? Look at the PaCO₂.
4. What is the patient’s metabolic status? Review the HCO₃⁻.
5. Is there any compensation or other abnormalities? What is the primary cause of the acid-base imbalance, and which derangement is the result of secondary (compensatory) change? Matching PaCO₂ and HCO₃⁻ parameters with the pH can help you determine the primary cause and secondary change. Examine the serum lactate, hemoglobin, glucose, and electrolyte results.

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of \(\text{HCO}_3^-\) and proteins (buffers), the patient’s minor metabolic alkalosis most likely resulted from his cirrhosis.

**Case 2: Dual pathology and permissive hypercapnia**

An 84-year-old patient developed acute respiratory distress syndrome (ARDS). He had renal dysfunction and was receiving an I.V. furosemide infusion. Because of severe hypoxemia and profound hypercapnia, he was intubated and ventilated with high levels of \(F_{\text{O}_2}\), PEEP, and pressure support for a prolonged period.

When the patient was ventilated with pressure support mode ventilation at an \(F_{\text{O}_2}\) of 0.45, PEEP of 17.5 cm H\(_2\)O, and pressure support of 12 cm H\(_2\)O, his ABGs were:

- \(\text{PaO}_2\) of 85 mm Hg, indicating no hypoxemia with ventilatory support
- \(\text{pH}\) of 7.39 (within normal limits)
- \(\text{PaCO}_2\) of 65 mm Hg, indicating his minute ventilation was inadequate and causing hypercapnia and respiratory acidosis
- \(\text{HCO}_3^-\) of 38 mEq/L, reflecting a metabolic alteration toward alkalosis, most likely caused by the furosemide infusion or compensatory changes for hypercapnia and respiratory acidosis
- lactate of 1.21 mmol/L, indicating adequate tissue perfusion.

ARDS resulted in respiratory acidosis. On the other hand, the furosemide infusion and metabolic compensation for respiratory acidosis led to metabolic alkalosis. The above mixed acid-base disorders produce a normal pH.

Some patients may need a high level of ventilatory support to achieve and maintain optimal ABG values. This places them at risk of developing VILI from large tidal volumes or positive pressure, or oxygen toxicity from high \(F_{\text{O}_2}\) values, and may delay weaning. In patients with refractory hypoxemia or profound hypercapnia, mild hypoxemia or permissive hypercapnia are acceptable for a short period of time because this lung-protective ventilation strategy can minimize VILI.\(^3,17-22\)

The mechanical ventilation protocol summary developed by the National Institutes of Health; National Heart, Lung, and Blood Institute; and ARDS Clinical Network recommend maintaining \(\text{PaO}_2\) between 55 and 80 mm Hg or \(\text{SpO}_2\) between 88% and 95%, and pH between 7.30 and 7.45 in patients with ARDS.\(^17\)

To restore this patient’s acid-base balance and provide adequate oxygenation, we allowed permissive hypercapnia, making no change in ventilator settings despite his \(\text{PaCO}_2\) of 65 mm Hg. Eventually, the patient recovered, was extubated, and was discharged home.

**Case 3: Dehydration**

A 79-year-old woman was admitted to the ICU after a right hemicolectomy with the following ventilator settings: \(F_{\text{O}_2}\), 0.40; PEEP, 10 cm H\(_2\)O; and pressure support, 10 cm H\(_2\)O. Her ABGs were:

- \(\text{PaO}_2\) of 85.2 mm Hg, indicating no hypoxemia
- \(\text{pH}\) of 7.27, indicating acidosis
- \(\text{PaCO}_2\) of 41.5 mm Hg, indicating pulmonary ventilation was adequate for her metabolic status
- \(\text{HCO}_3^-\) of 18.6 mEq/L, reflecting a metabolic disturbance toward acidosis
- lactate of 1.57 mmol/L, a normal level suggesting that her tissue perfusion was adequate.

This is **uncompensated metabolic acidosis**. The patient subsequently developed sepsis and renal failure. Because of renal failure and severe metabolic acidosis, continuous veno-venous hemodi- lation (CVVHDF) was started with fluid removal at 150 mL/hour.

Five days later, her ABGs were pH, 7.49; \(\text{PaCO}_2\), 41.2 mm Hg; \(\text{PaO}_2\), 92.9 mm Hg; \(\text{HCO}_3^-\), 31.3 mEq/L; and lactate, 2.38 mmol/L. Ventilator settings were \(F_{\text{O}_2}\), 0.30; PEEP, 7.5 cm H\(_2\)O; and pressure support, 5 cm H\(_2\)O. Her metabolic acidosis had been rectified by CVVHDF, but she developed **uncompensated metabolic alkalosis**. Her lactate elevation was most likely due to sepsis. Because her normal serum creatinine level had been restored and her...
urine output was adequate, CVVHDF therapy was terminated.

After pressure support was increased from 5 to 12 cm H₂O, her systolic BP dropped from 140 mm Hg to less than 110 mm Hg [central venous pressure [CVP] was 5 mm Hg]. In addition, her urine output decreased significantly. Her ABGs were pH, 7.51; PaCO₂, 36.3 mm Hg; PaO₂, 106.2 mm Hg; HCO₃⁻, 28.3 mEq/L; and lactate, 1.30 mmol/L. This shows worsening metabolic alkalosis because increasing pressure support blew off more carbon dioxide and elevated pH.

PEEP affects the whole respiratory cycle [inspiration and expiration]. Pressure support, however, is only delivered during the inspiratory phase of spontaneous breaths. Therefore, compared with pressure support, PEEP has a more profound effect in decreasing cardiac output and lowering BP. With mechanically ventilated patients, BP often drops after PEEP is increased if the patient has inadequate intravascular volume. But a decrease in BP seldom occurs after elevating pressure support, unless the patient is profoundly dehydrated.

This patient’s marked reduction in BP and urine output as well as low CVP pointed to the possibility of profound dehydration, which made her very sensitive to increasing pressure support. Dehydration also can cause metabolic alkalosis.

Consequently, we lowered pressure support back to 5 cm H₂O. Her systolic BP immediately increased to between 140 and 160 mm Hg. We also administered a 250 mL I.V. bolus of 0.9% sodium chloride solution twice and increased the I.V. maintenance fluid infusion rate from 60 to 100 mL/hour. Consequently, her CVP increased to 7 mm Hg. Next, 500 mL of 4% albumin infusion was administered over 4 hours. Her urine output increased to 17 to 35 mL/hour.

After the albumin infusion was complete, the patient’s ABGs were pH, 7.46; PaCO₂, 36.1 mm Hg; PaO₂, 94.3 mm Hg; HCO₃⁻, 25.1 mEq/L; and lactate, 1.17 mmol/L. By treating her dehydration and lowering pressure support, her metabolic alkalosis was almost resolved in less than 6 hours.

**Staying on the case**

A critically ill patient’s condition can change rapidly and dramatically. Dynamic reviews of the patient’s condition and ABGs are essential. By understanding mechanical ventilation and how to use ABG information, ventilation strategies can be changed to accommodate your patient’s needs appropriately.

**REFERENCES**


*Jin Xiong Lian* is a clinical nurse specialist in the ICU at Concord Repatriation General Hospital, a teaching hospital for the University of Sydney in Australia. The author has disclosed that he has no financial relationships related to this article.

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