Using ABGs to optimize

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Illustration by Roy Scott

AN ARTERIAL BLOOD GAS (ABG) analysis can tell you about the patient's oxygenation (via Pao_2 and Sao_2), acid-base balance, pulmonary function (through the $Paco_2$), and metabolic status. This article focuses on translating ABG information into clinical benefits, with three case studies 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 and to 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), which will be discussed in detail later. Patients

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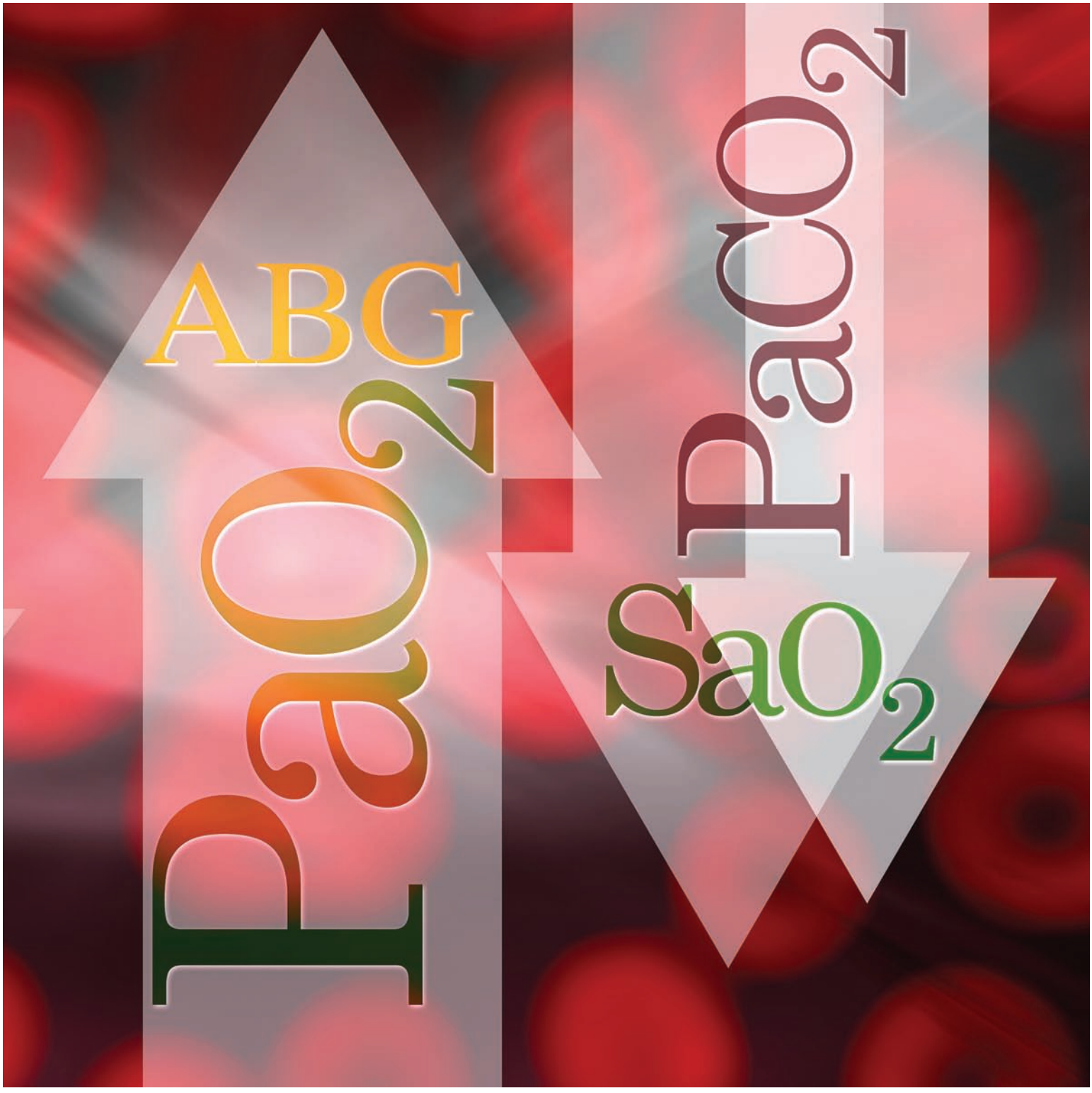
mechanical ventilation



should be weaned from ventilatory support if their condition permits.

A critically ill patient's clinical status 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 interventions. By reviewing the patient's ABGs and clinical status, 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:

- Pa_{O_2}, 80 to 100 mm Hg
- Sa_{O_2}, 95% to 100%
- pH, 7.35 to 7.45
- Pa_{CO_2}, 35 to 45 mm Hg
- HCO_3^-, 22 to 26 mEq/L.

Serum lactate is often obtained along with ABGs; results are normally less than 2 mmol/L in critically ill patients.[4-9]

See *Types of acid-base imbalances* for an overview of various acid-base disorders.

For mechanically ventilated patients, the key means of improving oxygenation are to increase the Fi_{O_2} or increase positive end-expiratory pressure (PEEP). Remember that a patient's minute ventilation equals respiratory rate times tidal volume (V_T). Therefore, any intervention that alters respiratory rate or V_T can help manage hypercapnia or hypocapnia and rectify acid-base imbalance.[1-5,10-12]

- In *volume control* mode ventilation, increasing the V_T, respiratory rate, or both will reduce Pa_{CO_2} 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 male patient, age 52, 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 history included diarrhea and fever for the last 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:

- Pa_{O_2} 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.
- Pa_{CO_2} of 41.8 mm Hg indicates his minute ventilation is adequate for his metabolic status.
- HCO_3^- of 13.8 mmol/L reflects a metabolic alteration toward acidosis.

Types of acid-base imbalances[6,7]

		pH	$Paco_2$	HCO_3^-
Respiratory acidosis	Uncompensated	<7.35	>45 mm Hg	Normal
	Partially compensated	<7.35	>45 mm Hg	>26 mEq/L
	Fully compensated	7.35 to 7.39	>45 mm Hg	>26 mEq/L
Metabolic acidosis	Uncompensated	<7.35	Normal	<22 mEq/L
	Partially compensated	<7.35	<35 mm Hg	<22 mEq/L
	Fully compensated	7.35 to 7.39	<35 mm Hg	<22 mEq/L
Respiratory alkalosis	Uncompensated	>7.45	<35 mm Hg	Normal
	Partially compensated	>7.45	<35 mm Hg	<22 mEq/L
	Fully compensated	7.41 to 7.45	<35 mm Hg	<22 mEq/L
Metabolic alkalosis	Uncompensated	>7.45	Normal	>26 mEq/L
	Partially compensated	>7.45	>45 mm Hg	>26 mEq/L
	Fully compensated	7.41 to 7.45	>45 mm Hg	>26 mEq/L
Mixed respiratory and metabolic acidosis		<7.35	>45 mm Hg	<22 mEq/L
Mixed respiratory and metabolic alkalosis		>7.45	<35 mm Hg	>26 mEq/L

• serum lactate level of 5.8 mmol/L indicates hyperlactatemia.

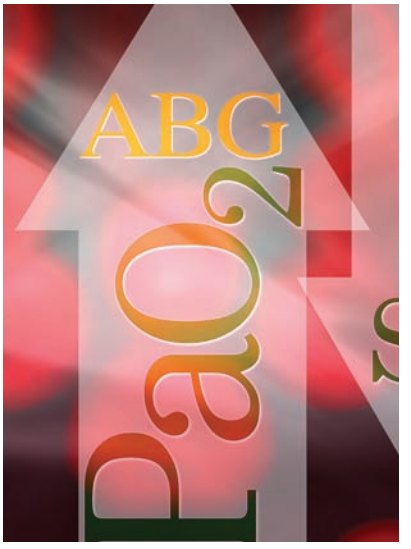
The patient's metabolic alteration moved his pH toward acidosis, but he had no respiratory derangement. Specifically, his $Paco_2$ and Pao_2 values showed that his respiratory system could maintain adequate ventilation and oxygenation with supplemental oxygen of 6 L/minute. This ABG profile shows ***uncompensated 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 didn't demonstrate respiratory compensation for metabolic acidosis, although it usually occurs fairly quickly.

Lactic acidosis is characterized by hyperlactatemia associated with metabolic acidosis.[6] The patient's lactate and pH values confirmed the diagnosis of lactic acidosis. Hypotension decreases tissue perfusion and impairs oxygen delivery, causing tissue hypoxia.

Tissue hypoxia leads to anaerobic metabolism and increases lactate production. Hepatic dysfunction reduces lactate clearance.[1,6] This patient's lactic acidosis was most likely caused by septic shock and the preexisting cirrhosis.

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



ABG analysis is an indispensable diagnostic tool for monitoring the patient's condition and evaluating the response to interventions.

Three-and-a-half hours after the previous ABG results, a new ABG analysis showed: pH, 7.29; $Paco_2$, 35.3 mm Hg; Pao_2, 99.7 mm Hg; HCO_3^-, 17 mEq/L; and lactate, 5.77 mmol/L. The patient 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 he was at increased risk for respiratory muscle fatigue, he was put on noninvasive ventilation with continuous positive airway pressure (CPAP) of 10 cm H_2O.

After the patient had been on CPAP for 4 hours, the ABG analysis showed: pH, 7.34; $Paco_2$, 32.4 mm Hg; Pao_2, 95.9 mm Hg; HCO_3^-, 19 mEq/L; and lactate, 6.7 mmol/L. The drop in the $Paco_2$ 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 was compensating for the metabolic acidosis. Now, the diagnosis was ***partially compensated metabolic acidosis***. However, his lactate level was still quite high.

Hyperlactatemia may indicate inadequate tissue perfusion, but the patient's MAP had been maintained above 70 mm Hg and his urine output was more than 0.5 mL/kg/hour, indicating adequate tissue perfusion. The slow reduction of his hyperlactatemia was 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; $Paco_2$, 34.2 mm Hg; Pao_2, 90.7 mm Hg; HCO_3^-, 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; $Paco_2$, 52.6 mm Hg; Pao_2, 60.7 mm Hg (indicating hypoxemia); HCO_3^-, 27.6 mEq/L; and lactate, 2.44 mmol/L. Fever increases oxygen consumption and carbon dioxide

Steps to interpreting ABGs

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

1. Is the patient hypoxemic? Look at the Pao_2 and Sao_2.
2. What's the acid-base balance? Check the pH.
3. How is the patient's pulmonary status? Look at the $Paco_2$.
4. What's the patient's metabolic status? Review the HCO_3^-.
5. Do you detect any abnormalities or compensation? What's the primary cause of the acid-base imbalance, and which derangement is the result of secondary (compensatory) change? Matching $Paco_2$ and HCO_3^- parameters with the pH can help you determine the primary cause and secondary change. Examine the serum lactate, hemoglobin, glucose, and electrolyte results.

production. The patient's respiratory system was unable to maintain adequate oxygenation and ventilation to meet his metabolic demand, as indicated by his hypoxemia and hypercapnia. His carbon dioxide retention led to respiratory acidosis. On the other hand, his metabolic process was attempting to elevate the pH, which suggested metabolic compensation for his respiratory acidosis.

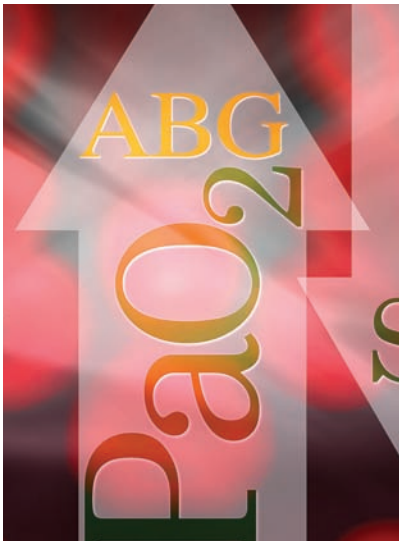
The above ABG profile is consistent with ***partially compensated respiratory acidosis***. A marked reduction in lactate levels is the result of decreased lactate production and increased lactate clearance due to improved tissue perfusion since admission.

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 generated by the gap between IPAP and EPAP.[15,16] BiPAP (IPAP, 15 cm H_2O; EPAP, 7 cm H_2O) with an Fio_2 of 0.40 was started to treat the patient's hypoxemia and hypercapnia as well as to rest respiratory muscles.

With the above settings, a pressure support of 8 cm H_2O was generated (15 cm H_2O – 7 cm H_2O = 8 cm H_2O). Also, antibiotics were changed based on the latest culture and sensitivity results.

After half an hour, the patient's ABG analysis showed: pH, 7.39; $Paco_2$, 42.1 mm Hg; Pao_2, 90.1 mm Hg; HCO_3^-, 25.4 mEq/L; and lactate, 2.40 mmol/L, which suggested his hypoxemia, carbon dioxide retention, and respiratory acidosis had all been corrected.

The next day, his ABG analysis while on BiPAP was pH, 7.27; $Paco_2$, 58.9 mm Hg; Pao_2, 89.5 mm Hg; HCO_3^-, 26.8 mEq/L; and lactate, 1.78 mmol/L.



Ventilator support buys time for other therapeutic interventions to work and lets the body reestablish homeostasis.

Partially compensated respiratory acidosis recurred, which could have been 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: Fio_2, 0.40; PEEP, 10 cm H_2O; and pressure support, 16 cm H_2O.

Thirteen hours later, the patient's ABGs were pH, 7.51; $Paco_2$, 34.1 mm Hg; Pao_2, 99.2 mm Hg; HCO_3^-, 26.8 mEq/L; and lactate, 2.39 mmol/L.

Pressure support ventilation augments spontaneous tidal volume and blows off more carbon dioxide. This patient's most recent ABG values 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 Fio_2, 0.30; PEEP, 10 cm H_2O; and pressure support, 12 cm H_2O. ABG values were pH, 7.59; $Paco_2$, 28.4 mm Hg; Pao_2, 156.3 mm Hg; HCO_3^-, 26.6 mEq/L; and lactate, 1.60 mmol/L. His ***mixed respiratory and metabolic alkalosis*** was worsening. The decrease of $Paco_2$ levels from 34.1 to 28.4 mm Hg suggested he'd been overventilated.

Hypocapnia and respiratory alkalosis can be caused by pain, agitation, severe anemia, hypoxia, brainstem injury, or excessive mechanical ventilation.[1,4,5] With this patient, the absence of pain, agitation, anemia, and other conditions suggest the most likely cause for his hyperventilation would be overventilation caused by pressure support. Consequently, both pressure support and PEEP were reduced to 5 cm H_2O. In less than 2 hours, a repeat ABG showed: pH, 7.49; $Paco_2$, 37.2 mm Hg; Pao_2, 96.9 mm Hg; HCO_3^-, 28.2 mEq/L; and lactate, 1.53 mmol/L. Lowering the pressure support corrected his hypocapnia and eliminated respiratory alkalosis. Now, he had only ***uncompensated metabolic alkalosis***.

Three days later, the patient was extubated and placed on an air-entrainment (Venturi) mask with an Fio_2 of 0.30. His ABGs were now pH, 7.46; $Paco_2$, 42.4 mm Hg; Pao_2, 114.8 mm Hg; HCO_3^-, 29.8 mEq/L; and lactate, 1.10 mol/L, reflecting minor ***uncompensated metabolic alkalosis***.

Because hepatic dysfunction reduces the production of 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 male patient with renal dysfunction developed acute respiratory distress syndrome (ARDS). He was receiving an I.V. furosemide infusion. Because of severe hypoxemia and profound hypercapnia, he was intubated and ventilated with high levels of Fio_2, PEEP, and pressure support for a prolonged period.

When the patient was ventilated with pressure support mode

ventilation at an FiO_2 of 0.45, PEEP of 17.5 cm H_2O, and pressure support of 12 cm H_2O, his ABGs were:

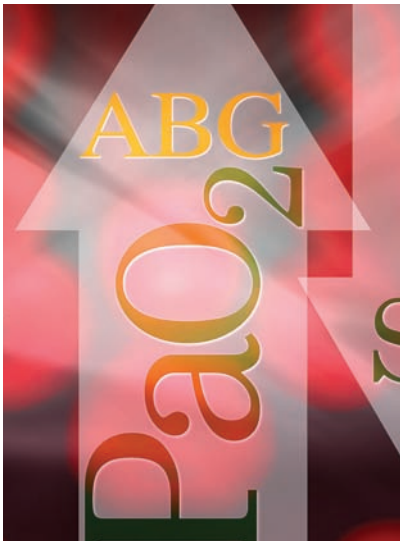
- PaO_2 of 85 mm Hg, indicating no hypoxemia with ventilatory support.
- pH of 7.39 (within normal limits).
- $PaCO_2$ of 65 mm Hg, indicating his minute ventilation was inadequate and causing hypercapnia and respiratory acidosis.
- HCO_3^- of 38 mEq/L, reflecting a metabolic alteration toward alkalosis, 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 for developing VILI from large tidal volumes or positive pressure, or oxygen toxicity from high FiO_2 values, and may delay weaning. In patients with refractory hypoxemia or profound hypercapnia, mild hypoxemia or permissive hypercapnia are acceptable for a short 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 PaO_2 between 55 and 80 mm Hg or 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, clinicians allowed permissive hypercapnia, making no change in ventilator settings despite his $PaCO_2$ of 65 mm Hg. Eventually, the patient recovered, was extubated, and was discharged home.



Because a critically ill patient's condition can change rapidly and dramatically, dynamic reviews of ABGs are essential.

Case 3: Dehydration

A 79-year-old woman was admitted to the ICU after a right hemicolectomy with the following ventilator settings: FiO_2, 0.40; PEEP, 10 cm H_2O; and pressure support, 10 cm H_2O. Her ABGs were:

- PaO_2 of 85.2 mm Hg, indicating no hypoxemia with ventilatory support.
- pH of 7.27, indicating acidosis.
- $PaCO_2$ of 41.5 mm Hg, indicating pulmonary ventilation was adequate for her metabolic status.
- 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 venovenous hemodiafiltration (CVVHDF) was started with fluid removal at 150 mL/hour.

Five days later, her ABGs were: pH, 7.49; $PaCO_2$, 41.2 mm Hg; PaO_2, 92.9 mm Hg; HCO_3^-, 31.3 mEq/L; and lactate, 2.38 mmol/L. Ventilator settings were FiO_2, 0.30; PEEP, 7.5 cm H_2O; and pressure support, 5 cm H_2O. Her metabolic acidosis had been corrected by CVVHDF, but she developed ***uncompensated metabolic alkalosis***. Her lactate elevation was most likely due to sepsis. Because her serum creatinine level had returned to normal and her urine output was adequate, CVVHDF therapy was terminated.

After pressure support was increased from 5 to 12 cm H_2O, her systolic BP dropped from 140 mm Hg to less than 110 mm Hg. Her central venous pressure (CVP) was 5 mm Hg. (During the first 6 hours of resuscitation, the goals of sepsis-induced hypoperfusion include a CVP of 8 to 12 mm Hg).[23] In addition, her urine output decreased significantly. Her ABGs were pH, 7.51; $PaCO_2$, 36.3 mm Hg; PaO_2, 106.2 mm Hg; HCO_3^-, 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 delivered only 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.[1,3] 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, pressure support was lowered back to 5 cm H_2O. Her systolic BP

immediately increased to between 140 and 160 mm Hg. She also received a 250 mL I.V. bolus of 0.9% sodium chloride solution twice, and the I.V. maintenance fluid infusion rate was increased from 60 to 100 mL/hour. Consequently, her CVP rose to 7 mm Hg.

Next, 500 mL of 4% albumin was infused 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; $Paco_2$, 36.1 mm Hg; Pao_2, 94.3 mm Hg; HCO_3^-, 25.1 mEq/L; and lactate, 1.17 mmol/L.

Treating dehydration and lowering pressure support nearly resolved this patient's metabolic alkalosis in less than 6 hours.

Dynamic approach

Because a critically ill patient's condition can change rapidly and dramatically, dynamic reviews of ABGs are essential. By understanding mechanical ventilation and how to use ABG results, ventilation strategies can be changed as needed to address changes in the patient's condition. ■

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