

# Arterial Blood Gas Case Questions and Answers

In the space that follows you will find a series of cases that include arterial blood gases. Each case is then followed by an explanation of the acid-base status, the oxygenation status and a summary of the patient's clinical picture.

The explanations of the acid-base status utilize the 5-step approach to interpreting acid-base status that is laid out in the Arterial Blood Gas Primer, which you can access by clicking [here](#). In some of the cases below, information is not available to calculate the anion gap or the delta delta. In such cases, you should focus solely on identifying the primary and compensatory processes.

## Case 1:

**A 24 year-old woman is found down in Pioneer Square by some bystanders. The medics are called and, upon arrival, find her with an oxygen saturation of 88% on room air and pinpoint pupils on exam. She is brought into the Harborview ER where a room air arterial blood gas is performed and reveals: pH 7.25, PCO<sub>2</sub> 60, PO<sub>2</sub> 65, HCO<sub>3</sub><sup>-</sup> 26, Base Excess 1. On his chemistry panel, her sodium is 137, chloride 100, bicarbonate 27.**

## Acid-base status:

- The patient has a low pH (acidemia)
- The PCO<sub>2</sub> is high (respiratory acidosis) and the bicarbonate is at the upper end of normal. The low pH and high PCO<sub>2</sub> imply that the respiratory acidosis is the primary process
- The anion gap is 10 and is, therefore, normal. The patient does not have an elevated anion gap acidosis.
- There is no compensatory process. Although the measured bicarbonate is just above normal, the base excess of 1 tells us that there is no metabolic alkalosis
- The delta gap is  $10 - 12 = -2$  and the delta-delta is  $-2 + 27 = 25$ . There is, therefore, no metabolic process.
- Summary: An acute, uncompensated respiratory acidosis leading to acidemia.

**Alveolar-arterial oxygen difference:** The alveolar-arterial difference is 10 mmHg, a normal value, which tells us that her hypoxemia is entirely due to hypoventilation.

**Explanation for the clinical picture:** The respiratory acidosis implies that the patient is hypoventilating. This fact, in combination with the pinpoint pupils suggests the patient is suffering from an acute narcotic overdose. In this case, the narcotic is most likely heroin.

### Case 2:

A 60 year-old man with amyotrophic lateral sclerosis is brought into clinic by his family who are concerned that he is more somnolent than normal. On further history, they report that he has been having problems with morning headaches and does not feel very refreshed when he wakes up. An arterial blood gas is performed and reveals: pH 7.37, PCO<sub>2</sub> 57, PO<sub>2</sub> 70, HCO<sub>3</sub><sup>-</sup> 32.

### Acid-base status:

- The patient has a low pH (acidemia)
- The PCO<sub>2</sub> is high (respiratory acidosis) and the bicarbonate is high (metabolic alkalosis). The low pH in combination with the high PCO<sub>2</sub> tells us that the respiratory acidosis is the primary process.
- The metabolic alkalosis is the compensatory process.
- Summary: A chronic respiratory acidosis with a compensatory metabolic alkalosis.

**Alveolar-arterial oxygen difference:** The alveolar-arterial oxygen difference is 9 mmHg, a normal value, which tells us that the hypoxemia is entirely due to hypoventilation.

**Explanation for the clinical picture:** The patient has a respiratory acidosis with a compensatory metabolic alkalosis. The respiratory acidosis tells us that the patient is hypoventilating while the compensatory metabolic alkalosis tells us that this is a chronic process. The patient is likely hypoventilating due to progression of his amyotrophic lateral sclerosis, a neurodegenerative disorder associated with progressive muscle weakness that eventually involves the muscles of respiration.

### Case 3:

A 65 year-old man is brought into the VA hospital with complaints of severe nausea and weakness. He has had problems with peptic ulcer disease in the past and has been having similar pain for the past two weeks. Rather than see a physician about this, he opted to deal with the problem on his own and, over the past week, has been drinking significant quantities of milk and consuming large quantities of TUMS (calcium carbonate). On his initial laboratory studies, he is found to have a calcium level of 11.5 mg/dL, a creatinine of 1.4 and bicarbonate of 35. The resident working in the ER decides to draw a room air arterial blood gas, which reveals: pH 7.45, PCO<sub>2</sub> 49, PO<sub>2</sub> 68, HCO<sub>3</sub><sup>-</sup> 34. On his chemistry panel, the sodium is 139, chloride 95, HCO<sub>3</sub><sup>-</sup> 34.

### **Acid-base status:**

- The patient has a high pH (alkalemia)
- The  $PCO_2$  is high (respiratory acidosis) and the bicarbonate is high (metabolic alkalosis). The high pH and the high bicarbonate tell us that the metabolic alkalosis is the primary process.
- The anion gap is 10. This is a normal value
- The respiratory acidosis is the compensatory process
- The delta gap is  $10 - 12 = -2$ . The delta delta is  $-2 + 34 = 32$ . This value is above 26 and tells us that a metabolic alkalosis is present. This is the same process that was identified in the second step above.
- Summary: A primary metabolic alkalosis with respiratory compensation.

**Alveolar-arterial oxygen difference:** The alveolar-arterial difference is 14 mmHg. This value is mildly elevated but still within the normal range for someone of this age. This suggests that his hypoxemia is likely due to hypoventilation.

**Explanation for the clinical picture:** The patient has hypercalcemia and a metabolic alkalosis. In conjunction with a clinical history of heavy milk and calcium carbonate consumption, these abnormalities suggest the patient is suffering from milk-alkali syndrome. In response to metabolic alkaloses, patients develop hypoventilation. This explains his elevated  $PCO_2$  and respiratory acidosis that, in turn, explains his hypoxemia.

### **Case 4:**

**A 45 year-old woman with a history of inhalant abuse presents to the emergency room complaining of dyspnea. She has an  $S_pO_2$  of 99% on room air and is obviously tachypneic on exam with what appears to be Kussmaul's respirations. A room air arterial blood gas is performed and reveals: pH 6.95,  $PCO_2$  9,  $PO_2$  128,  $HCO_3^-$  2. A chemistry panel revealed sodium of 130, chloride 98,  $HCO_3^-$  2.**

### **Acid-base status:**

- The patient has a very low pH (acidemia)
- The patient has a low  $PCO_2$  (respiratory alkalosis) and a very low bicarbonate (metabolic acidosis). The low pH in conjunction with the low bicarbonate tells us that the metabolic acidosis is the primary process
- The anion gap is elevated at 30. This tells us that the patient has a primary elevated anion gap metabolic acidosis.
- The respiratory alkalosis is the compensatory process, although in this case, despite a huge compensatory increase in minute ventilation, the patient still has a very low pH.

- The delta gap is  $30 - 12 = 18$  and the delta-delta is  $18 + 2 = 20$ . Since the delta-delta is below 22, we know that there is an additional non-gap metabolic acidosis as well.
- Summary: Combined elevated anion gap and non-gap metabolic acidoses with compensatory respiratory alkalosis.

**Alveolar-arterial oxygen difference:** The alveolar-arterial oxygen difference is 11 mmHg. This tells us that the patient does not have any shunt or low V/Q areas and, therefore, likely does not have any pulmonary parenchymal pathology as the source of her dyspnea. Her  $P_aO_2$  is actually well above 100 mmHg even though she is breathing room air at sea-level. This is a result of her extreme degree of hyperventilation, which leads to a rise in the alveolar  $PO_2$ .

**Explanation for the clinical picture:** The patient has concurrent elevated anion gap and non-gap acidoses with respiratory compensation. The acidoses are so severe that, despite the high minute ventilation, the pH remains very low. This patient has a history of inhalant abuse and one of the commonly abused inhalants, toluene, can present with severe elevated anion gap acidosis with respiratory compensation. The severe elevated anion gap acidosis is due to accumulation of one of the main toluene metabolites, hippuric acid.

#### Case 5:

**A 68 year-old man with a history of very severe COPD ( $FEV_1 \sim 1.0L$ ,  $< 25\%$  predicted) and chronic carbon dioxide retention (Baseline  $PCO_2$  58) presents to the emergency room complaining of worsening dyspnea and an increase in the frequency and purulence of his sputum production over the past 2 days. His oxygen saturation is 78% on room air. Before he is placed on supplemental oxygen, a room air arterial blood gas is drawn and reveals: pH 7.25,  $PCO_2$  68,  $PO_2$  48,  $HCO_3^-$  31.**

#### Acid-base status:

- The patient has a low pH (acidemia)
- The patient has a high  $PCO_2$  (respiratory acidosis) and a high bicarbonate (metabolic alkalosis). The combination of the low pH and the high  $PCO_2$  tells us that the respiratory acidosis is the primary process.
- The metabolic alkalosis is the compensatory process. The pH is still low despite this metabolic compensation
- Summary: Primary respiratory acidosis with compensatory metabolic alkalosis.

**Alveolar-arterial oxygen difference:** The alveolar-arterial oxygen difference is 17 mmHg. This value is elevated, suggesting that the hypoxemia is due to either

shunt or areas of low V/Q (the more likely explanation in a patient with COPD) and cannot be explained by hypoventilation alone.

**Explanation for the clinical picture:** The patient has very severe COPD and chronic carbon dioxide retention. As a result, you expect that at baseline, they will have a chronic respiratory acidosis (his baseline  $\text{PCO}_2$  was 58) with a compensatory metabolic alkalosis. In this case, the clinical history suggests the patient is in an exacerbation. When the patient presents to the ER, his  $\text{PCO}_2$  is elevated above his baseline. Because this is an acute change, the bicarbonate has not had time to adjust and the pH falls. This case is, therefore, an example of an *acute on chronic* respiratory acidosis.

#### Case 6:

**A climber is coming down from the summit of Mt. Everest. At an altitude of 8,400 m ( $P_B \sim 272$  mmHg), he has a blood gas drawn while breathing ambient air as part of a research project. The blood gas reveals pH 7.55,  $\text{PCO}_2$  12,  $\text{PO}_2$  30 and  $\text{HCO}_3^-$  10.5.**

#### Acid-base status:

- The patient has a high pH (alkalemia)
- The  $\text{PCO}_2$  is low (respiratory alkalosis) and the bicarbonate is low (metabolic acidosis). The high pH in conjunction with the low  $\text{PCO}_2$  tells us that the respiratory alkalosis is the primary process.
- The metabolic acidosis is the compensatory process
- Summary: Primary respiratory alkalosis with metabolic compensation.

**Alveolar-arterial oxygen difference:** The alveolar-arterial oxygen difference is only 2.3! When you get to very low barometric pressures and low alveolar oxygen tensions like the values seen in this case, the alveolar-arterial oxygen difference can be very small. This is a normal value at this elevation. Hypoxemia with a normal alveolar-arterial oxygen difference is due to either hypoventilation or a low inspired  $\text{PO}_2$ . In this particular case, it is the low inspired  $\text{PO}_2$  that is responsible for the profound – but well-tolerated – hypoxemia.

**Explanation for the clinical picture:** The patient is climbing at extremely high altitude where the low oxygen tensions in the arterial blood trigger the hypoxic ventilatory response. This response is, in turn, responsible for the respiratory alkalosis. As the patient spends increasing time at these high altitudes, metabolic compensation occurs and allows the ventilatory response to increase even further in magnitude. It is important to note that the person could not achieve this degree of minute ventilation on acute exposure to this altitude and, as a result, would not be able to maintain an adequate  $P_a\text{O}_2$  to support life. It is only with long periods spent acclimatizing to the environment that the person can achieve

sufficient levels of minute ventilation and maintain adequate levels of oxygenation.

**Case 7:**

**A 57 year-old woman presents with 2 days of fevers, dyspnea and a cough productive of rust-colored sputum. Her room air oxygen saturation in the emergency room is found to be 85% and the intern decides to obtain a room air arterial blood gas while they are waiting for the chest x-ray to be done. The blood gas reveals: pH 7.54, PCO<sub>2</sub> 25, PO<sub>2</sub> 65, HCO<sub>3</sub><sup>-</sup> 22, Base excess -1.**

**Acid-base status:**

- The patient has a high pH (alkalemia)
- The PCO<sub>2</sub> is low (respiratory alkalosis). The high pH in conjunction with the low PCO<sub>2</sub> tells us that the respiratory alkalosis is the primary process.
- Although the bicarbonate is on the low side, the base excess of -1 tells us that the patient does not have a metabolic acidosis. The patient, therefore, has not mounted a compensatory response yet.
- Summary: Acute, uncompensated respiratory alkalosis

**Alveolar-arterial oxygen difference:** The alveolar-arterial oxygen difference is 54 mmHg. This value is elevated and tells us that the patient's hypoxemia is due to either shunt or areas of low V/Q

**Explanation for the clinical picture:** The patient has a history suggestive of pneumonia. The pneumonia has led to areas of shunt and/or low V/Q that have led to her arterial hypoxemia. The arterial hypoxemia will, in turn, lead to the hypoxic ventilatory response that is responsible for her hyperventilation and respiratory alkalosis. Because the hypoxemia may have only been present for a short time, there has not been adequate time for metabolic compensation to occur.

**Case 8:**

**A 47 year-old man with a history of heavy alcohol use presents with a two-day history of severe abdominal pain, nausea and vomiting. On exam, his blood pressure is 90/50 and he is markedly tender in his epigastrium. His initial laboratory studies reveal a sodium of 132, chloride 92, HCO<sub>3</sub><sup>-</sup> 16, creatinine 1.5, amylase 400 and lipase 250. A room air arterial blood gas is drawn and reveals pH 7.28, PCO<sub>2</sub> 34, PO<sub>2</sub> 88, HCO<sub>3</sub><sup>-</sup> 16.**

**Acid-base status:**

- The patient has a low pH (acidemia)
- The  $\text{PCO}_2$  is low (respiratory alkalosis) and the bicarbonate is low (metabolic acidosis). The combination of the low pH and the low bicarbonate tells us that the metabolic acidosis is the primary process
- The anion gap is elevated at 24. This tells us that the patient has a primary elevated anion gap metabolic acidosis
- The respiratory alkalosis is the compensatory process
- The delta gap is  $24 - 12 = 12$ . The delta delta is  $12 + 17 = 29$ . Because the delta-delta is greater than 26, we know that the patient has a concurrent metabolic alkalosis.
- Summary: Primary elevated anion gap metabolic acidosis with respiratory compensation and a concurrent metabolic alkalosis.

**Alveolar-arterial oxygen difference:** The alveolar-arterial oxygen difference is 27. This suggests that the patient's hypoxemia is due to either shunt or areas of low V/Q.

**Explanation for the clinical picture:** A history of epigastric pain, nausea and vomiting in conjunction with elevated lipase and amylase on laboratory studies is consistent with the diagnosis of pancreatitis. As a result of the pancreatitis, the patient has developed an elevated anion gap acidosis with respiratory compensation. The concurrent metabolic alkalosis is likely due to vomiting, which leads to hydrogen ion loss via the upper gastrointestinal tract.