

METABOLIC ACIDOSIS & ANION GAP *David Kissin, BS, RRT*



With the increasing sophistication of analyzers and laboratory techniques, many of the measurements cursorily used in the past for diagnoses are now giving more definitive information to the caregiver team. The sensitivity of these devices can detect previously unmeasurable ions and substances that had complicated patient diagnoses. Measuring the unmeasurable has more clearly differentiated dysfunction, especially in patients with acid-base imbalances. This can be seen with the increased utility of anion gap and the ability to separate metabolic acidoses into treatable categories.

Anion gap..... mention that to most practitioners and you get a nod of understanding with a somewhat quizzical look. Is this the cause of ALL metabolic aberrations displayed by arterial blood gases? Do clinicians really have a grasp on the causative factors for anion gap and how it is manifested? I am not sure that they do and will try to explain it to some degree.

Metabolic acidosis is an extremely common finding in the Intensive Care Unit (ICU). It is defined as the result of either increased acid production, decreased acid excretion, or increased alkali excretion. This is often caused by renal failure or insufficiency. The kidneys' function is impaired and, therefore, not efficiently regulating bicarbonate ion in the blood. This phenomenon can be seen relatively easily by following arterial blood gas results. Some clinicians attempt to compensate for this by "controlling" the patient's ventilation to decrease the PaCO₂ in the intubated, mechanically ventilated patient. Other patients require dialysis to filter the patient's blood and regulate their acid-base balance.

Another cause is lactic acidosis. This is usually a by-product of anaerobic metabolism. Muscle fatigue and sepsis are common factors. This is one of the examples of an anion gap acidosis. Anion gaps in intensive care unit patients can have multiple underlying mechanisms. Hyperchloremia and hyperlactatemia are very common findings. Sepsis in addition to renal failure can cause high anion gap acidosis and can be further complicated by the metabolism of some drugs. These drugs can cause a pyroglutamic acidemia. Rocktaeschel and his colleagues have looked at unmeasurable anions to predict high lactate levels and mortality. They found that base excess and anion gap can adequately predict hyperlactatemia, but not mortality. Moe and Fuster describe the classification of metabolic acidosis with the measurement of anion gap to delineate it better than direct measurement of strong acid anions.

Anion gap is a direct measure of the difference between the positively charged ion (cation) sodium (Na⁺) less the sum of the negatively charged ions (anions) bicarbonate (HCO₃⁻) and chloride (Cl⁻) expressed as the following equation:

$$Na^+ - (HCO_3^- + Cl^-)$$

A normal result is 12 mmol/liter. If the result of the equation is greater than 12, it is indicative of an anion gap acidosis. Conversely, if the result is less than 12, it is deemed a non-anion gap acidosis. A lactic acidosis is a measured level of lactate greater than 5 mmol/liter in the blood. Murray and her colleagues at the Royal Bristol Hospital for Children in Bristol, United Kingdom, looked at post-cardiac surgery metabolic acidosis and found that corrected anion gap was the most accurate bedside calculation for assessing tissue acidosis. They found that this was due to hyperchloremia as a common occurrence with cardiopulmonary bypass and less commonly due to hyperlactatemia. Shoal and his group of B'nai Zion Medical Center in Haifa, Israel found that, along with measurement of urea, anion gap was the most sensitive laboratory finding in pediatric dehydration patients. Kaplan and Kellum of Yale noted strong

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correlation between ion gap and mortality in vascular trauma patients. Dr. Martin and his team from Los Angeles found anion gap to be a strong predictor of mortality in patients with normal lactate levels. Forni's group from Worthing Hospital in West Sussex, United Kingdom, conclude that unmeasured anions are the strongest contributors to metabolic acidosis and may be a complicating factor in such acidoses as diabetic ketoacidosis. These anions were found not to be elevated if the patient exhibited normal anion gap. Bowling and Morgan of Australia determined that these anions, especially in those patients with sepsis and shock are intermediates of the Krebs cycle. Dr. Howard Corey of the Goryeb Children's Hospital in New Jersey states that unmeasured anions that cause metabolic acidosis are only indirectly measured using anion gap calculations. He also concludes that the anion gap is influenced by non-carbonate buffers albumin and phosphate in the plasma, plasma acid-base balance and how the anion gap is measured. Feldman, Soni and Dickson found a strong inverse correlation between serum anion concentrations in patients with fluid imbalances and anion gap metabolic acidosis with electrolyte disorders. Humphreys' group at Brigham and Womens Hospital in Boston describe a patient who was admitted for chemotherapy treatment for lymphoma that develops fever and neutropenia and is treated with 10 days of acetaminophen. An anion gap of up to 30 mmol/liter and altered mentation followed and they found this to be due to the production of pyroglutamic acid confounded by the patient's sepsis and renal impairment.

Not all acidosis is ventilation related, with elevated carbon dioxide levels. Metabolic acidoses are common, especially in intensive care units, with trauma patients, septic patients and patients who have undergone cardiopulmonary bypass. While base deficit results from an arterial blood gas can give some indication of the causative factors for the acidosis, it is not a directly measured value. Anion gap, the difference between active ions in the blood, is a direct measurement and is a much more useful tool for diagnosis and treatment of metabolic acidosis. Treatment options, of course, center around and are directed at the underlying cause of the metabolic acidosis. When clinically indicated, such treatments may include hemodialysis, insulin therapy (for diabetic ketoacidosis) and, in cases of severe acidosis, an intravenous bicarbonate infusion. Treating an acidosis needs to be done cautiously. If an acidosis is corrected too slowly, sequelae include inactivity of many pharmaceuticals, which are inactive in acidotic environments. If an acidosis is corrected too quickly, this may lead to seizures and ischemia. Keeping an especially close watch on renal function is imperative. The kidneys are the acid-base "balancer" of the body along with the lungs. In the non-intubated patient, the kidneys and lungs try to work symbiotically to maintain acid-base stasis. When one or the other becomes impaired, its partner tries to compensate. When either organ is overwhelmed, the other can quickly become overwhelmed as well and the patient will require more intensive intervention. Renal insufficiency usually leads to dialysis and pulmonary insufficiency can lead to intubation and mechanical ventilation. Both interventions are aimed at taking the burden off of the organs and allowing them to "play catch up". However, artificially manipulating acid-base balance can also lead to the impairment in the organs' function and the inherent compensatory mechanisms. Caution should always be used and repeated assessment of the patient's progress, especially the anion gap and acid-base status should be done vigilantly.



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