- ETT sizes correlate better with weight than age; they have a smaller tube ready.

Induction

- No specific drug contraindications; limited data on dosages.
- Low functional residual capacity can lead to rapid desaturation with induction.
- Avoid hypoxia, hypercarbia, and acidosis, which can worsen pulmonary hypertension.

Maintenance

- Mechanical ventilation may require reduced tidal volume and higher rate.
- Pressure-controlled ventilation may be superior; careful attention to PAP.
- Careful positioning of hyperextensible joints.
- Consider OG tube for gastric decompression.
- Use peripheral nerve stimulator to guide NMBD dosage.

Postoperative Period

- Continuous pulse oximetry due to high incidence of sleep apnea.
- Prepare for prolonged resp insufficiency and mechanical ventilation.
- May need to remain intubated and/or monitored in an ICU.
- Pain control critical to postop resp status.

Anticipated Problems/Concerns

- Difficult airway and ventilation
- Neurologic impairment
- Resp insufficiency and postop ventilation
- Pain control

Acidosis, Lactic/Metabolic

Justin D. Ramos | Peter M. Schulman

Risk

- Incidence in USA: Unknown
- Present in a variety of disease states, from mild to severe systemic illness

Perioperative Risks

- Hemodynamic instability (due to arteriolar vasodilation and decreased cardiac output)
- Hyperkalemia
- Insulin resistance and hyperglycemia
- Stimulation of inflammation and suppression of immune response
- Acute resp failure

Worry About

- Decreased responsiveness to vasopressors and inotropes
- Decreased activity of local anesthetic agents
- Arrhythmias

Overview

- Physiologic disturbance resulting from excess acid production, failure of organic acid excretion, or inappropriate bicarbonate loss causing increased serum acidity.
- A marker of an underlying disease process.
- Severe when, in the presence of resp compensation, serum $[HCO_3]$ is ≤10 mmol/L or pH < 7.20.
- Acute metabolic acidosis is associated with increased morbidity and mortality.

Etiology

- Broadly differentiated by calculating the AG: AG = $[Na^+]$ – ($[Cl^-]$ + $[HCO_3^-]$). The AG corresponds to the presence of unmeasured anions in serum. The presence or absence of an elevated AG helps to determine the underlying cause and direct appropriate therapy. Normal AG is 7 ± 4 mEq/L and decreases 2.5 mEq/L for every 1 g/dL decrease in serum albumin. Corrected AG can be calculated:
  - Corrected AG = Calculated AG – {2.5 * (4.0 – [albumin])}.
- High AG metabolic acidosis: Results from an accumulation of excess acid in the serum. Specific causes are due to production of lactate or ketones (diabetic, alcoholic, or starvation ketoacidosis), toxic ingestion (methanol, ethylene glycol, salicylates), uremia, or medication side effects (propofol infusion syndrome, lactic acidosis associated with metformin).
- Normal AG (hyperchloremic) metabolic acidosis: Associated with excess HCO_3^- loss from the kidney or GI tract, failure of the kidney to excrete H^+, or rapid IV infusion of unbuffered solutions (e.g., normal saline).
- Delta gap (ΔΔ): Used to determine the presence of concomitant metabolic derangements and is calculated as ΔAG/Δ $[HCO_3^-]$, where ΔAG = (calculated AG – expected AG) and Δ $[HCO_3^-]$ = (24–$[HCO_3^-]$). ΔΔ < 1 indicates AG metabolic acidosis *and* concurrent non-AG acidosis. ΔΔ > 2 indicates AG metabolic acidosis and concurrent metabolic alkalosis. ΔΔ = 1 to 2 indicates a pure AG metabolic acidosis.

Usual Treatment

- Centered on rapid identification and treatment of the underlying physiologic disturbance (e.g., DKA, sepsis, inadequate resuscitation, CV failure, abdominal ischemia).
- In high AG metabolic acidosis, alkali therapy may be indicated as a temporizing measure for acute, severe acidemia (pH <7.20). In normal AG metabolic acidosis, alkali therapy may be indicated to replace bicarbonate losses.
  - Sodium bicarbonate remains the most widely used buffer; however, its use in correcting acute metabolic acidosis is controversial because it may increase $PaCO_2$ and paradoxically worsen intracellular acidosis. Other untoward effects of bicarbonate include hyperosmolarity and hypernatremia. Bicarbonate administration has not been proven to improve cellular function or reduce mortality in lactic or ketoacidosis.
  - THAM is an alternate buffer designed to limit CO_2 generation, offering theoretical benefits over bicarbonate. It buffers via the ammonia moiety, but elimination of protons is dependent on urinary excretion or removal via dialysis.
  - When alkali therapy is indicated, the bicarbonate deficit can be calculated to guide appropriate dosing. Bicarbonate should be administered as an isotonic infusion, rather than a bolus of hypertonic solution. Bicarbonate deficit (mEq) = 0.4 × body weight (kg) × (24 – $[HCO_3^-]$).
- In some instances (hyperventilation syndromes, high altitude), acidosis may be compensatory and not require treatment.

Assessment Points

System	Effect	Assessment by Hx	PE	Test
NEURO	Altered mental status, seizures	Level of consciousness, delirium, somnolence nausea/vomiting, seizures, toxic ingestion	Obtunded, confused, somnolent	Toxicology screen, osmolal gap, serum lytes
CV	Arteriolar vasodilation, hypotension, decreased response to vasopressors and inotropes, arrhythmias, hypocontractility	Signs of end-organ hypoperfusion	Tachycardia, hypotension, poor peripheral pulses, cold extremities, poor capillary refill	Invasive hemodynamic monitoring, ECHO, ECG
PULM	Hypoxemia, hyperventilation, resp failure	Tachypnea, dyspnea	Rapid and shallow breathing, accessory muscle use, hypoxia, hypercarbia	CXR, ABG, pulse oximetry
RENAL	Oliguria, acute kidney injury, ATN	Urine output, chronic renal disease	Signs of hypovolemia or hypervolemia	UO, Cr, BUN, urine lytes, UA, serum lytes
GI		Nausea, vomiting, diarrhea, melena, abdominal pain	Abdominal pain to palpation	Serum lactate, radiographic imaging, upper/lower endoscopy
ID		Fever, rigors	Hyperthermia or hypothermia, signs of focal infection	WBC with differential, cultures, radiographic imaging
ENDO	Hyperglycemia, insulin resistance	DM, polyuria, polydipsia, hyperphagia	Signs of dehydration	Blood glucose, serum ketones

Key References: Kraut JA, Madias NE: Metabolic acidosis: pathophysiology, diagnosis and management, *Nat Rev Nephrol* 6:274–285, 2010; Kraut JA, Madias NE: Treatment of acute metabolic acidosis: a pathophysiologic approach, *Nat Rev Nephrol* 8:589–601, 2012; Kimmoun A, Novy E, Auchet T, et al.: Hemodynamic consequences of severe lactic acidosis in shock states: from bench to bedside, *Crit Care* 19:175, 2015.

Perioperative Implications

Preoperative Preparation

- Pts with metabolic acidosis may be hemodynamically unstable and demonstrate decreased responsiveness to inotropes and vasopressors.
- Consider postponing surgery until the underlying cause is corrected, unless treatment requires immediate surgical intervention.
- If surgery is urgent or emergent, consider ways to optimize the pt preop.

Intraoperative

- Invasive monitoring may be indicated, depending on the severity of illness.
- Goal for induction is hemodynamic stability.
- Inotropes and vasopressors should be readily available.
- Consider the need for pt to remain intubated postop.

Postoperative Period

- Pt may require postop ICU care and prolonged mechanical ventilation.

Anticipated Problems/Concerns

- Hemodynamic instability with decreased responsiveness to inotropes and vasopressors.
- Compensation for profound metabolic acidosis may lead to acute resp failure.
- Treatment with bicarbonate may paradoxically increase $PaCO_2$ and worsen intracellular acidosis and resp status.

Acidosis, Renal Tubular

Amit Prabhakar | Alan David Kaye

Risk

- Incidence in USA: Unknown
- Present in a variety of disease states, from mild to severe systemic illness

Perioperative Risks

- Hemodynamic instability (related to arteriolar vasodilation, acidosis, and decreased cardiac output)
- Hyperkalemia
- Insulin resistance and hyperglycemia
- Acute respiratory failure

Worry About

- Decreased responsiveness to vasopressors and inotropes
- Decreased activity of local anesthetic agents
- Arrhythmias

Overview

- RTA is a type of metabolic acidosis that is due to either abnormal bicarbonate loss or acid excretion by the kidneys in presence of a normal or near normal glomerular filtration rate.
- Results in non-anion gap metabolic acidosis.
- Metabolic acidosis not due to gastrointestinal bicarbonate loss or acute/chronic renal insufficiency.
- Related to either proximal tubule dysfunction of bicarbonate reabsorption, failure of distal tubule excretion of acid, or mineralocorticoid deficiency.
- Other findings may include recurrent nephrocalcinosis, growth retardation, and osteomalacia/rickets in children.
- Can be either inherited, transient, or acquired.

Etiology

- Distal RTA (type 1) is due to defective distal tubular H^+ secretion.
  - Clinical features include impairment of growth, polyuria, hypercalciuria, lithiasis, nephrocalcinosis, and K^+ depletion.
  - Acquired forms related to hypergammaglobulinemia, autoimmune disorders such as SLE or Sjögren syndrome, and pts with chronic liver disease.
  - Can be associated with sensorineural hearing loss.
- Proximal RTA (type 2) is due to defective proximal tubule reabsorption of bicarbonate.
  - Manifests as stunted growth in children.
  - Can be associated with Fanconi syndrome, and if so, can manifest with osteomalacia and rickets.
  - Other causes include medications and toxins such as acetazolamide, aminoglycoside antibiotics, expired tetracyclines, lead, cadmium, and mercury.
- Type 3 RTA is a combination of types 1 and 2.
  - Can be transient in pediatric pts with type 1 RTA.
  - Carbonic anhydrase II deficiency is an AutoR syndrome associated with osteoporosis, RTA, cerebral calcification, and mental retardation.
- Hyperkalemic RTA (type 4): Due to either mineralocorticoid deficiency or hormone resistance
  - Most frequently observed in children with hypo- or pseudohypoaldosteronism
  - Also found to be related with diabetic nephropathy, SLE, and AIDS nephropathy
  - Drug induced causes include COX inhibitors, ACE-I's, heparin, K retaining diuretics, trimethoprim, and others

Diagnosis

- Should be suspected anytime metabolic acidosis is accompanied with hyperchloremia and a normal plasma anion gap without evidence of gastrointestinal bicarbonate loss or acid ingestion
- Differential diagnosis (common distal causes of RTA):
  - Hypokalemic or normokalemic: Primary, hypercalcemia, renal transplant rejection, multiple myeloma, SLE, nephrocalcinosis, hepatic cirrhosis, amphotericin B, lithium, and toluene
  - Hyperkalemic: Sickle cell nephropathy, obstructive nephropathy, hypoaldosteronism, and SLE
- Common proximal causes of RTA: Primary, Fanconi syndrome, Wilson disease, metals (mercury, lead, and cadmium), early renal transplant, nephrotic syndrome, and amyloidosis
- Tests used to aid in diagnosis include:
  - CMP to assess plasma electrolytes, baseline kidney function, and plasma anion gap
  - Urine pH and urine anion gap
  - Urine osmol gap and urine PCO_2
  - Urine calcium and citrate excretion
  - Renin: aldosterone ratio
  - Oral administration of acidifying salt (typically with ammonium chloride loading) employed to assess for ammonium secretion; normal individuals achieve a urine pH of less than 5.5, whereas pts with distal RTA are unable to acidify urine
  - Furosemide test: PO or IV dose given to assess distal tubule acidification function
- Metabolic derangements associated with each type
  - Distal: Pt will present with hyperchloremic metabolic acidosis with a positive anion gap or an osmol gap <100 mmol/L. Diagnosis is supported by either normal or decreased plasma K^+ concentration and inability to lower urine pH <5.5 after either acidifying salt (ammonium chloride) loading or furosemide test.
  - Proximal: Pt will present with hyperchloremic metabolic acidosis, negative anion gap, or osmol gap above 100 mmol/L. Definitive diagnosis is made with presence of low urine pH at low plasma bicarb concentration and the presence of normal urine PCO_2 and a high urine bicarb excretion at normal plasma bicarb concentration. GI or renal loss, previous intake of acidifying salt, or excessive use of laxatives must be ruled out.
  - Hyperkalemic RTA: Should be considered if K^+ is increased with urine pH <5.5. Renin and aldosterone levels must be assessed. Hypoaldosteronism is the most common cause of hyperkalemic distal RTA.

Usual Treatment

- Focused on preventing pediatric growth restriction, nephrocalcinosis, and development of chronic renal failure in all ages.
- Treatment based on disease specific alkali replacement using either bicarbonate or citrate.
- Proximal RTA alkali supplementation usually needed until 3 to 5 y of age.
- Distal RTA is more likely to be permanent with treatment needed throughout life.
- Hyperkalemic RTA can be treated with fludrocortisone, furosemide, and alkali supplements if needed.