Extubation
- Altered sensorium may impair airway protective reflexes.

Adjuvants
- Early consideration for initiating vasopressin replacement therapy.
- Administration of hypotonic IV fluids if oral intake inadequate to maintain normal plasma osmolality.
- Supplemental corticosteroid therapy if anterior pituitary deficiency present.
- Chlorpropamide treatment for DI may cause hypoglycemia.

Anticipated Problems/Concerns
- High-dose vasopressin therapy may cause vasoconstriction and precipitate myocardial ischemia in pts with preexisting CAD.
- Postop DI following pituitary surgery/traumatic brain injury usually manifests within 24–48 h but may be delayed.
- Vasopressin therapy will not increase urine osmolality in pts with nephrogenic DI and should not be used in pts with primary polydipsia.

Diabetic Ketoacidosis

Shamsuddin Akhtar

Risk
- Typically seen in pts with type I diabetes mellitus; can occur in pts with ketosis-prone type II diabetes.
- Stress related to acute infection, trauma, surgery, MI, pulm embolism, pancreatitis, alcohol abuse, stroke, emotional trauma, or drugs (steroids, thiazides, sodium-glucose transporter-2 inhibitors) can precipitate DKA in diabetic pts.
- Poor compliance with insulin therapy or inadequate outpatient insulin regimen.

Perioperative Risks
- CV collapse secondary to severe dehydration (diuresis, fluid deprivation, fever) and/or myocardial depression due to severe metabolic acidosis
- Cerebral edema and injury with rapid correction of DKA, especially in children
- ARDS and bronchial mucus plugging
- Worsening of preexisting renal dysfunction or periop MI in pts with preexisting CAD
- Malignant hyperthermia-like syndrome due solely to DKA (extremely rare)

Worry About
- Fluid deficit of 5–10 L in established DKA (100 mL/kg)
- Cardiac arrest, severe shock, or arrhythmias with onset of general anesthesia or regional anesthesia due to hypovolemia, acidosis, and lyte disturbances
- Severe lyte derangements and significant total body deficits of potassium (3–5 mEq/kg), sodium (7–10 mEq/kg), phosphate (5–7 mmol/kg), calcium (1–2 mEq/kg), and magnesium (1–2 mEq/kg)
- Necessity of surgical therapy to treat etiology of DKA (sepsis, abscess, gangrene)

Overview
- DKA is the most common acute metabolic emergency with significant mortality (3–5%).
- Two primary hormonal abnormalities: Absolute or relative deficiency of insulin; and glucagon excess, causing increased gluconeogenesis, increased breakdown of glycogen and decreased use of glucose by liver, muscle, and fat.
- Characterized by hyperglycemia (>250 mg/dL), ketosis (positive ketones in serum and urine), anion-gap metabolic acidosis (anion gap >10, HCO_3 <18, pH <7.3).
- Intensive periop hemodynamic and metabolic management essential for favorable outcome.

Etiology
- Type I diabetes with insulin deficiency caused by cessation or inadequate dosing of insulin therapy, with or without significant pathologic cause (infection, surgery) or emotional stress.
- Elevation of counter-regulatory hormones (glucagon, epinephrine, cortisol, and growth hormone) causes significant alteration in carbohydrate, fat, and protein metabolism and drive the catabolic and ketogenic state.
- Osmotic diuresis secondary to sustained hyperglycemia leads to volume and lyte depletion.
- Metabolic acidosis is a product of unrestrained free fatty acid release from adipose tissue and subsequent hepatic oxidation of fatty acids to ketone bodies (due to lack of insulin and glucagon excess).

Usual Treatment
- Search and treat initiating cause.
- Insulin, rehydration, correction of lyte derangements, and hemodynamic support.

Assessment Points

System	Effect	Assessment by Hx	PE	Test
CV	Hypovolemia	Duration of initiating event, postural symptoms	BP, HR, JVD, skin turgor, mucous membranes, tilt table test, orthostatic hypotension, shock	US, CVP, ABG
RESP	Hyperventilation (Kussmaul respiration)		Ventilatory rate and depth, fruity odor of acetone	ABG
GI	Anorexia, N/V	Appetite, N/V, abdominal pain	Abdominal distension, ileus, tenderness without rebound	
RENAL	Diuresis	Urinary frequency, thirst (polyuria, polydipsia)		UO, BUN/Cr, lytes (especially potassium), serum osmolality
ENDO	Insulin deficiency, glucagon excess during severe catabolic stress	Type I diabetes		Blood glucose ABG (anion gap) Ketones (urine, blood)
CNS	Confusion, drowsiness, lethargy to coma; late cerebral edema in children		Assess LOC Signs of increased ICP	ABG, serum osmolarity

Key References: Kamel KS, Halperin ML: Acid-base problems in diabetic ketoacidosis, *N Engl J Med* 372(20):1969–1970, 2015; Gosmanov AR, Gosmanova EO, Kitabchi AE: Hyperglycemic crises: diabetic ketoacidosis (DKA), and hyperglycemic hyperosmolar state (HHS). In De Groot LJ, Beck-Peccoz P, Chrousos G, editors: *Endotext* [internet]. South Dartmouth, MA, 2015, MDText.com, Inc. <http://www.ncbi.nlm.nih.gov/books/NBK279052/>.

Perioperative Implications

Perioperative Preparation
- Vigorous 0.9 normal saline infusion (15–20 mL/kg/h or 1–1.5 L in the first h) to restore hemodynamic stability, then 0.5 normal saline, especially if serum osmolality is >310 mOsm/L.
- Insulin Rx usually begins after first h of fluid therapy with 0.1 U of regular insulin/kg IV bolus (in adults) followed by infusion of 0.1 U/kg/h (as long as serum potassium is >3.3 mEq/L). Adjust insulin infusion to decrease glucose by 10% or 50–70 mg/dL per h. In children, fluid glucose content is adjusted prior to decreasing insulin infusion.
- Sodium bicarbonate not generally indicated, administer 100 mmol over 2 h if pH <6.9, hyperkalemia, or pt hemodynamically unstable with pH <7.1.

Monitoring
- Check glucose and lytes hourly (especially potassium); check pH frequently: Foley catheter to determine urine output reliably during periop period; CVP catheter for fluid management, possibly PA cath if pt has preexisting myocardial dysfunction or CAD; consider TTE/TEE in hemodynamically unstable pt

Airway
- Potential stiff joint syndrome with difficult intubation; at risk for aspiration

Induction
- Hemodynamic instability likely if intravascular volume depletion not corrected; pts frequently have preexisting autonomic neuropathy and CV dysfunction.

Maintenance
- Protect end organs, especially heart, renal, and CNS, because they are often compromised by DM.

Extubation
- Awake. May require mechanical ventilation and ICU admission if pH less than 7.2, compromised mental status, and/or high risk of aspiration.

Adjuvants
- Same as for diabetes

Postoperative Period
- Potential for hypoglycemic injury from rapid increase in insulin sensitivity when surgical cause of DKA corrected.
- Subsequent medical management should be continued by physician with expertise in diabetes.

Anticipated Problems/Concerns
- Hemodynamic instability from combined volume deficiency, acidosis, and pre-existing CV disease
- CNS dysfunction from metabolic and lyte abnormalities (hypokalemia, hypophosphatemia), both early and late

Diaphragmatic Hernia (Congenital)

N. James Halliday | Jibin V. Samuel

Risk
- Occurs in approximately 1 in 2500–5000 births; 12–25% have associated anomalies, in particular cardiac (20%), chromosomal (5–16%), and neurologic.
- Parents who have one child with isolated defect have 2% chance with next child.
- Usually left sided (90%) due to defect in foramen of Bochdalek and are more common in boys. Morgagni hernias (2–5%) located anterior are more common in girls. Remainder through esophageal hiatus.

Perioperative Risks
- 30–60% mortality despite improved diagnosis and management
- Degree of pulm hypoplasia and associated CNS and CV malformations affect mortality
- Timing of diagnosis associated with the prognosis

Worry About
- Hypoxemia and acidosis
- Pulm Htn and CHD
- Shock
- Tension pneumothorax

Overview
- Classified by site of herniation
  - Posterolateral defects (Bochdalek) are left sided (largest and associated with greatest degree of pulm hypoplasia). Morgagni hernias rare; parasternal, less symptomatic, and therefore diagnosed at later age
- Between 4–9 wk of age the pleuroperitoneal membrane forms with the left closing after the right. In Bochdalek and Morgagni, normal development of the diaphragm and digestive tract does not occur
- Degree of lung hypoplasia determined by time of defect during fetal development and amount of abdominal contents in chest. Although ipsilateral lung is most affected, both lungs are abnormal and result in decreased numbers and function of alveoli; hypoplastic lung with smaller pulm artery and decreased arterial branching causes high vascular resistance

Usual Treatment
- Initial treatment involves determining the severity of associated congenital anomalies and degree of illness.
- Goal is semielective surgery when pt is medically stable.
- Posterolateral defects require surgical repair (does not resolve the pulmonary dysfunction).
- Small defects closed primarily; larger defects use artificial diaphragm, which contributes to postop resp failure.
- In most cases, abdomen is closed primarily after correction but a silastic pouch may be used with increased intra-abdominal pressures.
- Fetal surgery has been accomplished in those severely affected with increased degree of pulm hypoplasia. FETO can be done to trigger lung growth. The Cochrane group reviewed two studies looking at a total of 97 women who underwent FETO and found insufficient data indicating improvement in perinatal mortality.

Assessment Points

System	Effect	Assessment by Hx	PE	Test
CV	Mediastinal shift, associated ASD, VSD, coarctation, tetralogy of Fallot (23%)	Displaced cardiac impulse	CV exam	ECHO
RESP	Resp distress, pulm Htn	Decreased breath sounds on affected side Prominent ipsilateral chest	Pulm exam	CXR, ABG
GI	Malrotation, atresia (20%)	Scaphoid abdomen	Abdominal exam	
GU	Hypospadias		Inspection	
CNS	Spina bifida, hydrocephalus, anencephaly (28%)		Inspection and neurologic exam	US, CT
METAB	Acidosis, hypoxemia, hypercarbia			ABG

Key References: Aggarwal S, Stockmann P, Klein MD, et al.: Echocardiographic measures of ventricular function and pulmonary artery size: prognostic markers of congenital diaphragmatic hernia? *J Perinatol* 31(8):561–566, 2011; Crivell RM, Andersen C, Dodd JM: Prenatal treatments for babies with congenital diaphragmatic hernia, *Cochrane Database Syst Rev* 11:CD008925, 2015.

Perioperative Implications

Perioperative Management
- ECMO provides temporary support until perinatal circulation matures and less sensitive to vasoconstrictive stimuli (1–2 wk).

Preoperative Preparation
- Avoid triggers for pulm vasoconstriction.
- Goals include a PaO_2 >80, $PaCO_2$ 25–30, normal or elevated pH, and normothermia (hypothermia increases O_2 consumption).
- For pulm Htn, can also use nitric oxide 20–80 ppm, sildenafil.
- Avoid gaseous distension of stomach with early placement of NG tube.
- With compromised neonates, ET intubation, sedation, paralysis, and ECMO may be required if conventional or high-frequency ventilation fails.
- All neonates with resp distress require invasive monitoring using preductal right radial a-line. If severe, consider preductal and postductal A-line and pulse oximetry.
- IV access best in upper extremities to avoid possible IVC obstruction from increased intra-abdominal pressure.
- Watch for pneumothorax (sudden deterioration in BP or oxygenation); consider prophylactic contralateral chest tube; equipment needed should be available.

Anesthetic Technique
- Opioids well tolerated; inhaled halogenated anesthetics may cause significant hypotension; avoid N_2O, which distends gas-filled intestines.
- Avoid peak pressures more than 25 cm H_2O.
- High frequency, low tidal volume preferred.
- Continue nitric oxide if given preop.
- Lung mechanics change during surgery; may require hand ventilation.

Postoperative Period
- Continued muscle relaxation/opioids and ventilatory support.
- Once stable, assess need for continued resp support.
- If A-aDO_2 gradient >400 mm Hg or if cardiopulmonary deterioration, continue resp assistance.
- Persistent hypoxemia while on high FIO_2 suggests persistent pulm Htn with R-to-L shunting.
- Minimize ET suctioning, correct metabolic acidosis.
- Deliver adequate nutrition.
- High degree of neurologic problems, whether or not infants placed on ECMO; seizures, developmental delay, and hearing loss in 20–30%, but pulm outcomes are usually good.