Adjuvants
- Regional: Diabetic nerves may be more prone to edema, especially if epinephrine is used. Reduce dose (e.g., lidocaine reduced from 1.5% to 1%) for same effect.

Postoperative Period
- Usually GDM cured by delivery.
- Women with GDM need a follow-up GTT at 6–12 wk after delivery.

Anticipated Problems/Concerns
- Fetal dysfunction, especially hypoglycemia and acidosis, if maternal hypoglycemia present
- Rapid changes in maternal blood glucose can accompany the pain and/or exertion of vaginal delivery of fetus and accompany the endocrine changes of uterine delivery.

Diabetes Insipidus

Natalie F. Holt

Risk
- Hereditary/familial (rare):
  - Nephrogenic DI due to mutations in the AVP receptor gene, with X-linked recessive transmission; or AQP2, usually with autosomal recessive transmission, but autosomal dominant transmission also occurs; overall, males at greater risk than females
  - Central (hypothalamic) DI due to mutations of the AVP gene; usually manifests in childhood; males equal risk as females
  - May also be part of developmental syndromes (Wolfram syndrome, Laurence-Moon-Biedl syndrome) or congenital septo-optic dysplasia
- Acquired:
  - Trauma/surgery, infarction, inflammatory, infectious, infiltrative, or neoplastic process affecting the hypothalamic-neurohypophyseal region (>80% of vasopressin-secreting neurons must be destroyed before symptoms of DI manifest)
  - Renal disease (chronic renal failure, polycystic kidney disease, obstructive uropathy, renal transplantation)
  - Systemic conditions (multiple myeloma, sickle cell disease, sarcoidosis)
  - Lyte imbalances (hypokalemia, hypercalcemia)
  - Medications (lithium, demeclocycline, vinblastine, amphotericin B, sulfonylureas) or toxins (methoxyflurane, ethanol)
  - Idiopathic (may be associated with lung, breast, and slow-growing intracranial cancers)
  - Gestational due to pregnancy-induced acceleration of vasopressin metabolism by placental cysteine aminopeptidase
  - Primary polydipsia (dipsogenic DI) due to fluid intake in excess of renal free water excretion capabilities

Perioperative Risks
- Dehydration, hyperosmolarity, hypernatremia
- Altered mental status/seizures
- Hemodynamic instability
- Bladder distention, hydroureter

Worry About
- Fluid and lyte imbalance.
- Contributing drugs/toxins.
- Postop onset, especially following pituitary surgery (1–6 d postop: Classic pattern is surgery followed by early SIADH and then DI.

Overview
- Polyuria due to either insufficient production of vasopressin or inadequate renal tubular response to vasopressin.
- Polyuria, excessive thirst, polydipsia; dehydration rarely present in competent pts with access to water.
- Inadequate fluid replacement leads to hypernatremia, hyperosmolarity, and dehydration, causing fatigue, weakness, altered sensorium, hemodynamic instability, seizures, and possible death.
- In the periop context, fluid over-replacement plus treatment with DDAVP can lead to hyponatremia, hypo-osmolality, and overhydration, causing seizures and possible death.

Etiology
- Neurogenic (central/hypothalamic):
  - Inadequate release of vasopressin from posterior pituitary
  - Primary genetic or secondary acquired condition due to trauma/surgery (especially hypophysectomy and basal skull fractures), inflammation/infiltration, infarction, neoplasm
- Nephrogenic:
  - Inadequate renal tubular response to vasopressin
  - Primary genetic or secondary acquired due to medications/intoxications, chronic renal disease, systemic diseases (multiple myeloma, sickle cell disease), lyte imbalances (hypokalemia, hypercalcemia)

Usual Treatment
- Central DI: Synthetic vasopressin or vasopressin analogue (desmopressin) supplementation; older treatments (chlorpropamide, carbamazepine, clofibrate) that increase ADH sensitivity or stimulate ADH release not commonly used due to systemic side effects
- Nephrogenic DI: Diuretics (e.g., hydrochlorothiazide, amiloride), salt restriction, nonsteroidal anti-inflammatory drugs
- Primary polydipsia: Fluid restriction

Assessment Points

System	Effect	Assessment by Hx	PE	Test
CV	Hypotension Tachycardia Myocardial ischemia	Fatigue Weakness Reduced exercise tolerance	Orthostatic hypotension Dry mucous membranes, poor skin turgor (especially in infants)	ECG
ENDO	Anterior pituitary dysfunction	Pituitary surgery, neoplastic or infiltrative disease	Multisystem effects due to hormone deficiencies	Tests of anterior pituitary function, hormone levels
RENAL	Polyuria	Excessive production of dilute urine (urine osmolality 50–200 mOsm/kg; urine volume >3 L/day)	Urine volume and specific gravity	24-h urine collection; simultaneous measurements of plasma and urine osmolality; exclude hyperglycemia, hypercalcemia, hypokalemia
CNS	Altered sensorium Seizures Visual disturbance	Excessive thirst (particularly for cold drinks) Polydipsia History of head injury or cranial surgery	Neurologic function, including visual fields Papilledema Hyperreflexia Fever	MRI

Key References: Leroy C, Karrouz W, Douillard C, et al.: Diabetes insipidus, *Ann Endocrinol (Paris)* 74(5–6):496–507, 2013; Lacassie HJ, Muir HA, Millar S, et al.: Perioperative anesthetic management for Cesarean section of a parturient with gestational diabetes insipidus, *Can J Anesth* 52(7):733–736, 2005.

Perioperative Implications

Preoperative Preparation
- Recognition and appropriate treatment–water deprivation test; desmopressin trial; rule out metabolic causes, such as hyperglycemia, hypokalemia, and hypercalcemia.
- Assess lytes, serum osmolality, and volume status; replace water deficit over 48–72 h and do not lower sodium concentration by more than by more than 0.5-1 mEq/L per h depending on the duration of hypernatremia.
- Rule out additional hormonal deficiencies (e.g., cortisol).
- Discontinue provocative medications (e.g., lithium, mannitol).

Monitoring
- Urine output
- Serum lytes
- Intravascular volume

Airway
- Not affected

Induction
- Pts may have exaggerated hypotensive response due to hypovolemia
- Arrhythmias may occur as a result of lyte abnormalities

Maintenance
- Fluid and lyte monitoring and replacement.
- Invasive hemodynamic monitoring.
- Variable sensitivity to neuromuscular relaxants depending on concomitant lyte imbalances (e.g., hypercalcemia, hypokalemia).

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.