Induction
- Osmotic diuresis, autonomic nervous system, and CV dysfunction can make BP/HR fluctuate

Maintenance
- CV instability: Volume status and avoidance of hypertension key to avoiding renal and myocardial dysfunction periop

Extubation
- CV and pulm drive insufficiencies common with neuropathies

Adjuvants
- Regional: Diabetic nerves may be more prone to edema, especially if epinephrine used. Reduce dose (e.g., lidocaine from 2.0% to 1.5%) for same effect.
- Oral hypoglycemics may ablate preconditioning.

Postoperative Period
- Current ADA guidelines recommend IV administration of insulin for critically ill pts in ICU settings with a goal of maintaining plasma glucose concentration between 140–180 mg/dL.
- For noncritically ill pts, it is accepted to have targets below 140 mg/dL for fasting and <180 mg/dL postprandial.
- Debate as to whether control to tighter than 60–250 mg/dL is of value in absence of Htn.

Anticipated Problems/Concerns
- Autonomic nervous system dysfunction associated with sudden death postop; can monitor for resp function in ICU/PACU overnight; presence of adult at home who can measure blood glucose and call 911.
- Infections and end-organ risk substantially increased with blood sugar >250 mg/dL. Hypoglycemic symptoms hidden by autonomic nervous system dysfunction, effects of regional, sedative-narcotic, and beta-adrenergic blocking agents.

Diabetes, Type III (Gestational Diabetes Mellitus)

Kristen L. Lienhart | Danny Wilkerson

Risk
- Incidence of GDM approximately 5–6% of all pregnancies.
- Increased in African American, Hispanic, Asian, Native American, or Pacific Islander women.
- Risk factors:
  - Maternal age >25 y.
  - Previous delivery of macrosomic infant.
  - Glucosuria.
  - History of polycystic ovarian syndrome.
  - Previous unexplained fetal demise.
  - Previous pregnancy with GDM.
  - Strong immediate family history of NIDDM or GDM.
  - Obesity.
- Dx: Two-step approach:
  1. Fasting glucose >95 mg/dL or a glucose >130 mg/dL (identifies ~90% of women with GDM) 1 h after a 50-g OGTT.
  2. If initial screening meets or exceeds threshold, perform a 100-g, 3-h diagnostic OGTT on a separate day.

Perioperative Risks
- Increased frequency of gestational Htn, preeclampsia, and cesarean delivery
- Unlikely renal, ocular, neurologic, or orthopedic complications in GDM
- Hypoglycemia if insulin is used
- Fetal risk (if not controlled: Polyhydramnios or macrosomia [6 times normal])
- RDS (2–3 times normal); preeclampsia, neonatal hypoglycemia, prematurity

Worry About
- Hyperglycemia and hypoglycemia

Overview
- GDM is defined as a carbohydrate intolerance that occurs (or is first recognized) during pregnancy.
- Universal screening between 24-28 wk gestation.
- A glucose tolerance test is used to identify GDM. For details of the test, see the Key References.
- Maternal complications with GDM are few, but the fetus is at risk.
- Complications, such as fetal polyhydramnios, macrosomia (6 times normal), prematurity, birth trauma, RDS (2–3 times normal rate), neonatal hypoglycemia, or morbidity, are as common with type III diabetes (GDM) as with type I diabetes (insulin dependent).

Etiology
- Occurs in genetically susceptible individuals.
- Pregnancy, through secretion of substances from uterus, exerts diabetogenic effects.

Usual Treatment
- Many clinicians obtain a single HbA_{1c} level at 6–12 wk gestation. In pts with mildly elevated plasma glucose levels and normal concentration of HbA_{1c}, dietary modification alone and a modest increase in exercise are often sufficient to normalize plasma glucose levels.
- Use of insulin in GDM is now more common as tighter control seems beneficial.
- Insulin can be started if fasting glucose exceeds 95 mg/dL despite diet control.
- Glyburide and metformin are appropriate as first line therapy for diet failure in women with GDM.

Assessment Points

System	Effect	Assessment by Hx	PE	Test
HEENT	Possible facial/pharyngeal edema	Snoring	Neck ROM, Mallampati exam	
CV	CV changes of pregnancy—Possible worse hypovolemia from osmotic diuresis		BP/HR with orthostatic maneuvers	
RESP	Resp changes of pregnancy, decreased FRC, etc.			
GI	Gastroparesis of pregnancy	Early satiety		
ENDO	Neonatal hypoglycemia if maternal hyperglycemia, obesity			Blood sugar, glucose levels, acid-base status of fetus, HbA_{1c} in mother
HEME	No change, unless type I diabetes			
RENAL	Decreased renal function			BUN/Cr
CNS	ANS dysfunction	Gastroparesis, early satiety	Orthostatic BP	Tilt table test
PNS	Neuropathy not present unless type I diabetes			

Key References: Cunningham FG, Leveno KJ, Bloom SL, et al, editors: *Williams obstetrics*, ed 24, New York, NY, 2014, McGraw-Hill, pp 1–40; Garrison A: Screening, diagnosis, and management of gestational diabetes mellitus, *Am Fam Physician* 91(7):460–467, 2015.

Perioperative Implications

Preoperative Preparation
- Full-stomach precautions: Nonparticulate antacid administration usual

Monitoring
- Blood sugar in maternal and umbilical vein blood

Airway
- Examine for edema.

Induction
- Regional anesthesia is preferred to general anesthetic due to risks of aspiration and failed airway attainment if C-section is performed.
- Osmotic diuresis can cause hypovolemia and increase BP and HR fluctuations.

Maintenance
- CV instability: Volume status is key to maintenance of uterine and other organ perfusion.

Extubation
- Ensure patient is awake before extubation.

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).