Reduce dose (e.g., lidocaine from 2.0% to 1.5%) for same effect.

Postoperative Period

- Sliding scale of insulin Rx based on blood glucose determinations every 1–3 h; tight control periop may decrease infections but side effects of hypoglycemia possibly negate benefit.

Anticipated Problems/Concerns

- Gastroparesis with presence of solid food 24 h after last meal if ANS dysfunction is present. Consider treating with metoclopramide 10 mg IM 1½ h prior to induction.
- ANS dysfunction is assoc with sudden death postop; pt can be kept in ICU/PACU overnight; vested adult who can measure blood glucose and call 911 if sent home postop.

Diabetes, Type II (Noninsulin Dependent)

Stanley H. Rosenbaum | Ranjit Deshpande

Risk

- Incidence in USA more than 25 million
- Highest prevalence: Hispanics and Native Americans
- Gender predominance: None
- Metabolic syndrome associated with obesity and sedentary lifestyle

Perioperative Risks

- Increased risk 5–10 times if end-stage renal disease, CV, CHF, or autonomic neuropathy; without renal, CV disease, or autonomic dysfunction, risk is 1–1.5 times normal.
- Metabolic abnormalities increased with perioperative insulin Rx.
- Unclear if same risks as for type I diabetes.

Worry About

- Autonomic neuropathy, gastroparesis, and sudden postop death.
- Myocardial ischemia; CV instability.
- Tight glucose control might be indicated in pregnancy (see Diabetes, Type III); difficult weaning from bypass (ECC), predictable global or focal CNS ischemia.
- Disordered autoregulation makes BP fluctuations dangerous.
- Fluid and electrolyte imbalance.
- Hyperosmolar hyperglycemic state and, less likely, diabetic ketoacidosis.

Overview

- Endocrinopathy that can cause same organ dysfunction as in diabetes type I: end-stage renal, myocardial, neuropathic disease, stiff joint syndrome, and retinopathy.
- Associated with deranged blood flow autoregulation to CNS (at blood sugar 250 mg/dL), renal (at blood sugar 200 mg/dL), and cardiac (at blood sugar 100 mg/dL) vessels.
- Ketosis is rare because there is some endogenous insulin.
- Primarily controlled by diet and/or oral agents, although insulin is more frequently used.
- Usually has high insulin levels for glucose level, but peripheral resistance to insulin effect. Can develop hyperosmolar nonketotic coma.
- Blood sugar control per se not associated with increased periop morbidity in absence of:
  - Hypoglycemia.
  - Hyperosmolar coma.
  - CNS ischemia.
  - Pregnancy.
  - Extracorporeal circulation.
- Preop HgBA1c levels (ideally <7%) indicate quality of recent blood sugar control. High levels correlate with chronic microvascular complications.

Etiology

- Familial predisposition with very high concordance in identical twins
- Autosomal dominant accentuated by conditions that increase peripheral insulin resistance (obesity, inactivity, hormones), increase glucose production or metabolic demands (glucocorticoids, pregnancy), or decrease insulin secretion (certain beta-adrenergic drugs)
- Increases nonenzymatic glycosylations
- Causes cell swelling
- Deranges autoregulation
- Increases viscous protein production
- Increases substrate for anaerobic metabolism

Usual Treatment

- Hypoglycemic agents (see oral hypoglycemic agents), diet, exercise, insulin
- BP control with ACE inhibitors being drugs of choice, especially in diabetic nephropathy

Assessment Points

System	Effect	Assessment by Hx	PE	Test
HEENT	Possible atlanto-occipital dislocation Cataracts, glaucoma, retinopathy Periodontal disease	Pain	Neck ROM, prayer sign Loose decaying teeth	
CV	Premature CAD Hypertension Peripheral arterial disease Resting tachycardia, orthostatic hypotension	Angina Claudication Symptoms of CHF	Peripheral pulses	ECG CAD-related tests, as indicated
RESP	Decreased pulm elastance	Exercise tolerance		
GI	Gastroparesis Diarrhea	Early satiety		
ENDO	Hyperglycemia Osmotic diuretic–caused hypokalemia	Polyuria		Blood glucose, K^+
HEME	Infection from decreased WBC phagocytic function		Site of infections	
RENAL	Nephropathy Type 4 RTA	Asymptomatic although often associated with neuropathy		BUN/Cr, UA for protein
CNS	Cerebrovascular disease Medication-induced hypoglycemia	TIAs, CVAs, long-acting oral hypoglycemic agents	CNS exam	
PNS	Distal sensory and motor neuropathy Autonomic neuropathy	Impotence Foot infections	PNS exam, especially prior to regional anesthetic	
MS	Impaired joint mobility		ROM of joints	

Key References: Barash PG, Cullen BF, Stoelting RK, et al, editors: *Clinical anesthesia*, ed 7, Philadelphia, PA, 2013, Lippincott Williams & Wilkins; Kadol Y: Anesthetic considerations in diabetic patients. Part I: preoperative considerations of patients with diabetes mellitus. *J Anesth* 24(5):739–747, 2010.

Perioperative Implications

Preoperative Preparation

- Metoclopramide (5–10 mg) if gastroparesis.
- Assess myocardial, autonomic function, and volume status.
- Formulate recommendations for preop insulin and long-acting hypoglycemic agents.

Monitoring

- Blood sugar and metabolic abnormalities
- Painless myocardial ischemia can cause CHF if volume overload and LV dysfunction
- Peripheral vasculature and nerves vulnerable to pressure ischemia

Airway

- Atlanto-occipital dislocation possible: See HEENT, do prayer sign test

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.