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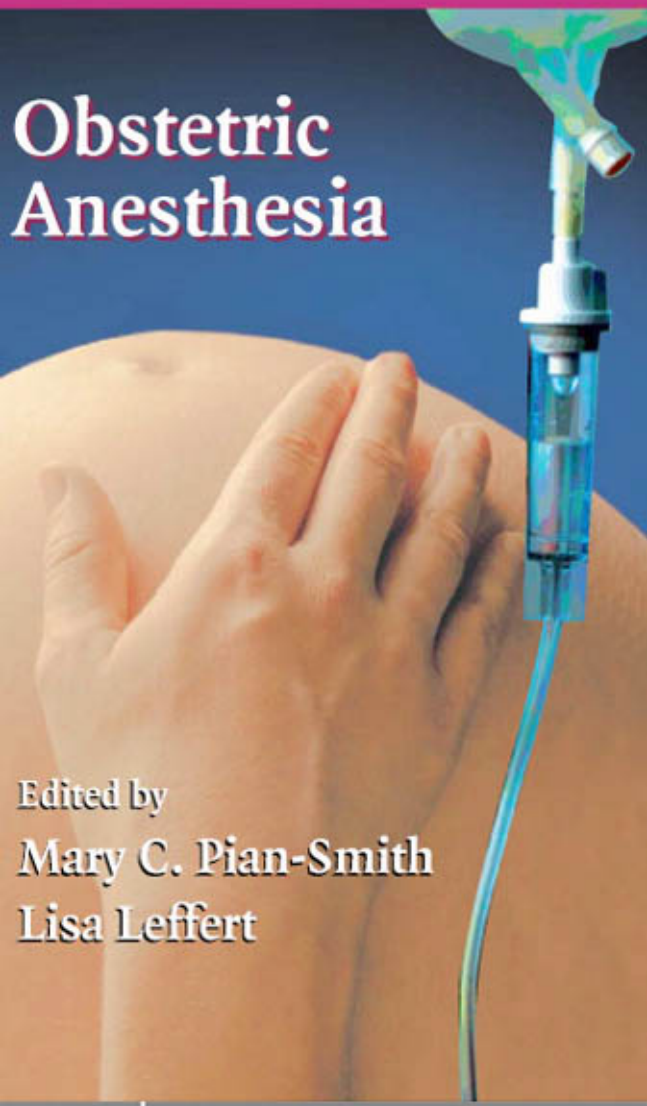
# Obstetric Anesthesia



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# Obstetric Anesthesia

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# Preface

*Obstetric Anesthesia* is a definitive, comprehensive and yet easily navigated reference for all anesthesia practitioners caring for parturients. With chapters written by Harvard anesthesia residents and fellows, it has been carefully edited and updated by faculty mentors and reflects the practice at our affiliated institutions.

The formatting was designed to offer rapid access to information, and thus is suited as a convenient reference on the labor floor. The first section focuses on the pharmacology, physiology and delivery of anesthesia as they relate to pregnancy. The second section offers both medical theory, as well as practical steps for the management of patients with co-existing disease (relevant physiology, pathology, obstetric management and anesthetic management). The third section focuses on the management of obstetric emergencies (physiology, pathology, step-by-step management and potential complications). We also include sections on anesthesia for non-delivery surgery and strategies for managing adverse outcomes such as neurologic complications and risk management. Finally, the book includes a detailed formulary of common medications, emphasizing indications, contraindications, and maternal and fetal effects of the drugs.

*Obstetric Anesthesia* is the result of a huge collaborative effort involving our trainees and our faculty colleagues. We thank the authors for their hard work and the contagious enthusiasm that they brought to the task. Indeed, it is working in this wonderful community that makes our clinical and teaching jobs so rewarding; thus it was not surprising that this writing project,

too, was stimulating and enjoyable. This book is a reflection of the intellectual curiosity, high standards and dedication to patient care that characterize this amazing team.

We also acknowledge gratefully the important administrative assistance of Linda Patten.

May C. M. Pian-Smith, and Lisa Leffert

PART ONE

# Obstetric Anesthesia

# ACHONDROPLASIA

STEPHEN PANARO, MD
EDWARD MICHNA, MD

## FUNDAMENTAL KNOWLEDGE

- Inherited disorder of bone metabolism that results in short stature, a small maxilla, large mandible & spinal abnormalities
- Lumbar lordosis & thoracic kyphosis are increased.
- The small vertebral pedicles & short anteroposterior & transverse diameters of the vertebral canal can result in spinal stenosis, which can worsen w/ age as scoliosis worsens & osteophytes form.

## STUDIES

- Physical exam
- Lumbosacral spine films may add information.

## MANAGEMENT/INTERVENTIONS

- Cervical mobility may be diminished; combined w/ other facial abnormalities, this may make intubation difficult.
- Regional anesthesia may be technically difficult but is not contraindicated.
- Because of the pt's short stature & spinal stenosis, the dosage for a single-shot spinal may be difficult to estimate.
- Insertion of an epidural or spinal catheter may be a better alternative to a single-shot spinal to allow careful titration of anesthetic.

## CAVEATS/PEARLS

- Consider using an epidural or spinal catheter instead of a single-shot spinal, which may be difficult to dose appropriately.
- Prepare for a difficult intubation if decreased cervical mobility or facial anomalies are present.

## CHECKLIST

- Physical exam, lumbosacral spine films

# ACROMEGALY

GRACE C. CHANG, MD, MBA; ROBERT A. PETERFREUND, MD, PhD; AND STEPHANIE L. LEE, MD, PhD

## FUNDAMENTAL KNOWLEDGE

### Definition

- Rare disfiguring & disabling disease caused by growth hormone hypersecretion
- Usually due to growth hormone-secreting anterior pituitary adenoma

### Epidemiology

- Prevalence 50–60 cases per million population
- Equal distribution btwn genders
- Very rare for acromegalic women to become pregnant because of impaired gonadotropic axis; 60% of acromegalic women are amenorrheic
- <100 pregnancies reported in women w/ acromegaly

### Signs/Symptoms

- Headache
- Papilledema
- Visual disturbances
- Enlargement of tongue & epiglottis
- Increased length of mandible
- Overgrowth of soft tissues of upper airway
- Hoarseness or change in voice
- Stridor
- Peripheral neuropathy
- Diabetes mellitus
- Systemic hypertension
- Ischemic heart disease
- Osteoarthritis/osteoporosis
- Thick & oily skin
- Skeletal muscle weakness

### Changes Associated w/ Pregnancy

- During early pregnancy, growth hormone (GH) is secreted by pituitary.
- In 2nd trimester, placenta starts producing variant of GH.
- GH levels continue to increase throughout pregnancy, peaking in 3rd trimester.

- Placental GH induces maternal hepatic IGF-1 production, which inhibits pituitary GH secretion.
- Serum IGF-1 levels increase in 2nd half of pregnancy.
- Conflicting evidence that pregnancy worsens pituitary macroadenoma growth; some case reports of worsening of symptoms during pregnancy; others showed improvement.
- In acromegalic women, pituitary GH secretion persists throughout pregnancy.

**Maternal Complications**
- Diabetes mellitus
- Hypertension
- Heart disease

**Fetal/Neonatal Complications**
- Newborns of untreated mothers have greater mean birthweights than newborns of treated mothers.

## STUDIES
- Plasma GH concentration >3 ng/mL
- Failure of plasma GH concentration to decrease 1–2 hours after administration of 75–100 g glucose
- Pituitary MRI w/ gadolinium for pituitary mass

## MANAGEMENT/INTERVENTIONS
- Transsphenoidal surgical excision of pituitary adenoma is definitive treatment.
  - ➢ May be required during pregnancy if pt has symptoms of tumor expansion or signs & symptoms not relieved by medical mgt
  - ➢ Perform surgery in 2nd trimester: surgery in 1st trimester associated w/ increased risk of spontaneous abortion; surgery in 3rd trimester may cause premature labor
- Medical mgt
  - ➢ Dopamine agonists
    - Bromocriptine or Dostinex; however, dopamine agonist treatment is successful in only 10% of cases
    - Dopamine agonists are more effective on tumors w/ GH & prolactin co-secretion.
  - ➢ Somatostatin analogs
    - Octreotide, octreotide LAR, lanreotide
    - Normalization of GH/IGF in 40–60% of pts
    - Limited experience during pregnancy

- ➢ GH receptor antagonists
  - Pegvisomant: no reported pregnancies on this medication
- Radiotherapy
  - ➢ Takes a long time to work
  - ➢ High incidence of hypopituitarism

**Anesthetic Management**

- Thorough evaluation of airway
  - ➢ Acromegalic pts have higher incidence of difficult intubation.
  - ➢ Distorted facial anatomy & increased length of mandible may lead to difficult mask airway.
  - ➢ Enlargement of tongue & epiglottis & overgrowth of soft tissue in upper airway can lead to obstruction.
  - ➢ Pt may require smaller endotracheal tube than normal because of subglottic narrowing & enlargement of vocal cords.
  - ➢ Nasal turbinate enlargement may make nasal intubation difficult.
  - ➢ Pt may require awake fiberoptic intubation.
- Use non-depolarizing muscle relaxants sparingly, especially if skeletal muscle weakness exists.
- Both regional anesthesia & general anesthesia can be used safely.
- Anticipate difficulty placing epidural or spinal because of skeletal changes.
- Document pre-op neuropathies & carefully pad all pressure points.
- Carefully monitor blood glucose, especially if pt has diabetes mellitus or glucose intolerance.

**CAVEATS/PEARLS**

- Pt may have various airway issues; anticipate difficult intubation if general anesthetic is required.

**CHECKLIST**

- Thorough preanesthetic evaluation, especially for hypertension, coronary artery disease, diabetes mellitus
- Thorough airway evaluation
- Anticipate difficult mask ventilation & intubation; may require fiberoptic bronchoscope.
- Monitor blood glucose.

# ACUTE FATTY LIVER OF PREGNANCY (AFLP)

LAWRENCE WEINSTEIN, MD
DOUG RAINES, MD

## FUNDAMENTAL KNOWLEDGE

### Epidemiology

- Approximate prevalence: 1/7,000 to 1/16,000 pregnancies
- Disorder of late pregnancy, w/ most cases diagnosed btwn 35 & 37 weeks gestation

### Pathogenesis

- Exact causal mechanisms are unknown, but there seems to be an association of AFLP w/ maternal long-chain 3-hydroxyacyl CoA dehydrogenase deficiency (LCHAD).
  - ➢ Long-chain 3-hydroxyacyl CoA dehydrogenase is an enzyme that is involved in mitochondrial beta-oxidation of fatty acids. It is thought that in mothers w/ this deficiency, fatty acid metabolites from the fetus & placenta can build up & overwhelm the mother's mitochondrial oxidation pathways. These metabolites can be hepatotoxic, & their accumulation is thought to be a possible causative mechanism for AFLP.
- Some evidence suggests a link between AFLP & pre-eclampsia.
  - ➢ There can be hepatic involvement in pre-eclampsia (HELLP syndrome).
  - ➢ Pre-eclampsia is often seen concurrently in pts w/ AFLP.
  - ➢ Liver biopsies in pre-eclamptic women w/ & w/o liver dysfunction both show microvesicular fat infiltration, suggesting that AFLP may be at the severe end of a pathologic spectrum encompassing pre-eclampsia, HELLP syndrome & AFLP.

### Clinical Manifestations

- AFLP occurs late, near term in the 3rd trimester.
- Symptoms of nausea, vomiting, lethargy, malaise & headache can be part of a prodromal period in AFLP.
- Pt may report RUQ abdominal pain.
- Jaundice or bleeding is sometimes the initial presentation.
- Hepatic encephalopathy can be a late finding.
- In contrast to intrahepatic cholestasis of pregnancy, pruritus is NOT a common symptom in AFLP.
- Over half of pts w/ AFLP have pre-eclampsia as well during their course.
- Coagulopathy & progression to DIC are possible in AFLP.

**Diagnosis**

- Diagnosis is usually made clinically through examination of symptoms & lab values (see "*Studies*").
- Work up the pt for pre-eclampsia & HELLP syndrome, since they are so often associated w/ AFLP.
  - ➢ HELLP syndrome consists of hemolysis, elevated liver enzymes & low platelet count.
- Imaging studies are not needed to diagnose AFLP but may be helpful to rule out other pathology, such as hepatic infarct or hematoma.

## STUDIES

**Laboratory Data & Studies**

- Serum transaminases are generally elevated in the neighborhood of 300–500 IU/L. Values may be as high as 1,000 but rarely exceed that.
- Alkaline phosphatase levels are markedly elevated (can be 10× normal).
- Leukocytosis w/ a left shift is a common but nonspecific finding.
- Hypoglycemia is common secondary to impaired hepatic gluconeogenesis.
- Acute renal failure may be a component of AFLP, w/ resultant rises in creatinine & BUN.
- If there has been progression to DIC, then thrombocytopenia is seen, along w/ elevations in PT & aPTT & a decrease in fibrinogen.
- Liver biopsy demonstrates microvesicular fatty infiltration of pericentral hepatocytes. There is also hepatocellular necrosis, portal inflammation & cholestasis. Biopsy is rarely needed to make the diagnosis & is in fact often contraindicated secondary to low platelets or abnormal coagulation studies.

## MANAGEMENT/INTERVENTIONS

- The ultimate treatment for AFLP is fetal delivery as soon as safely possible. Prompt attention is necessary because progressive hepatic failure & fetal death can occur within days.
- There is no evidence suggesting an advantage of cesarean delivery over prompt vaginal delivery.
- Prior to delivery, the pt should be stabilized, w/ attention to correcting metabolic & coagulation abnormalities.
  - ➢ Glucose infusion for hypoglycemia
  - ➢ Correction of coagulopathy w/ FFP, platelets or cryoprecipitate as needed (to avoid massive intrapartum hemorrhage). This is particularly important if neuraxial spinal/epidural anesthesia

is planned, because of the potential for a devastating epidural hematoma in a pt w/ coagulopathy &/or low platelets.
  - ➢ If acute renal failure complicates the clinical picture, dialysis can be necessary.
- Be aware of the renal status & potassium, & adjust your anesthetic plan accordingly (avoid potassium-containing replacement fluids if the pt is retaining K+, avoid succinylcholine if K+ is elevated).
- In the case of hepatic encephalopathy, blood ammonia levels should be obtained & lactulose given if appropriate.
  - ➢ If the encephalopathy is sufficiently bad to impair respiration, then mechanical ventilation in an ICU setting may be necessary.
- For either vaginal delivery or cesarean delivery, establish large-bore IV access in anticipation of the need for fluid resuscitation & administration of blood products.
- Be aware of intra-operative losses & have rapid access to appropriate replacement fluids & blood products.
- An arterial line can be an appropriate monitor to assist in frequent sampling of blood for Hct & coagulation studies & also to provide close BP monitoring should vasopressors become necessary.
- In the operating environment, especially if general anesthesia is required, it may be prudent to keep the pt warm to minimize coagulopathy.
- Anesthetic mgt for AFLP, & indeed for all liver disease of pregnancy, should be guided by the symptoms & manifestations of the liver process. Various manifestations will have differing effects on physiology & choice of anesthetic technique.
  - ➢ If pt is coagulopathic, see the section on "*General Concepts*."
  - ➢ If pt is cirrhotic, see sections on "*General Concepts*," "*Cirrhosis*," "*Portal Hypertension*," "*Ascites*," "*Hepatic Encephalopathy*."
- For general discussion of regional & general anesthesia considerations, see the section on "*General Concepts*."

**CAVEATS/PEARLS**

- AFLP is a rare but potentially devastating disorder of pregnancy.
- Presents in 3rd trimester
- Lab abnormalities are prominently elevated alkaline phosphatase, w/ a less marked rise in serum transaminases. Coagulation studies may also be abnormal, w/ potential for DIC.
- After diagnosis, treatment is maternal stabilization & delivery as soon as safely possible.
  - ➢ Anesthetic mgt should be guided by OB criteria & physiologic manifestations of the disease process.

➢ Special attention to coagulation status, as it has important implications for regional anesthesia & peripartum hemorrhage
➢ The maternal condition usually improves within 24 hours of delivery, w/ continued recovery over the next week.
  • There is generally no long-term liver dysfunction for survivors.

**CHECKLIST**
- Know OB plan.
- Degree of pt's compromise will dictate need for invasive monitoring, ICU care.
- Beware of coagulopathy, renal failure.

## ACUTE GLOMERULONEPHRITIS

JOSHUA WEBER, MD
PETER DUNN, MD

**FUNDAMENTAL KNOWLEDGE**
- Immunologic response to infection, usually group A beta-hemolytic streptococcal (although can be initiated by other bacterial or viral infections), that damages renal glomeruli
- Acute glomerulonephritis is rare during pregnancy.
- 3 urinary patterns are seen: focal nephritic, diffuse nephritic, nephrotic.

**STUDIES**
Lab tests commonly ordered to evaluate renal function in pregnancy include:
- Creatinine
- BUN
- Electrolytes
- Creatinine clearance
- CBC
- Urinalysis typically shows hematuria, proteinuria & red cell casts.
- In addition, consider:
- Renal biopsy

**Focal nephritic**
- Inflammatory lesions in less than half of glomeruli
- Urinalysis shows red cells, +/− red cell casts, & mild proteinuria.

**Diffuse nephritic**
- Affects most or all glomeruli

- Heavy proteinuria
- +/− renal insuffiency

**Nephrotic**
- Heavy proteinuria
- Few red cell casts

### MANAGEMENT/INTERVENTIONS

**Medical/OB mgt of acute glomerulonephritis:**
- Treatment of acute glomerulonephritis depends on the underlying etiology.
- For decreased renal function:
  - ➢ Increased frequency of prenatal visits (q2 wks in 1st & 2nd trimesters, then q1 week)
  - ➢ Monitoring: monthly measurements of serum creatinine, creatinine clearance, fetal development, BP
  - ➢ Erythropoietin may be used for maternal anemia.
  - ➢ Preterm delivery is considered for worsening renal function, fetal compromise or pre-eclampsia.
  - ➢ Renal biopsy is considered if rapid deterioration in renal function occurs prior to 32 weeks gestation.
  - ➢ If glomerulonephritis is responsive to steroids, these should be continued during pregnancy.
  - ➢ See "***Parturients on Dialysis***" for dialysis mgt.

**Anesthetic mgt of pts w/ acute glomerulonephritis**
**Pre-op**
- Evaluate degree of renal dysfunction & hypertension.
- Evaluate for anemia & electrolyte abnormalities.

**Intraoperative mgt**
- Monitors: standard monitoring + fetal heart rate (FHR) +/− arterial line +/− CVP
- Limit fluids in pts w/ marginal renal function to prevent volume overload.
- Use strict aseptic technique, as uremic pts are more prone to infection.
- Careful padding/protection of dialysis access is important.
- Consider promotility agents, as uremic pts may have impaired GI motility.

**Regional anesthesia**
- Uremia-induced platelet dysfunction leads to increased bleeding time.

- Pts may have thrombocytopenia from peripheral destruction of platelets.
- Increased toxicity from local anesthetics has been reported in pts w/ renal disease, but amide & ester local anesthetics can be used safely.
- Contraindications to regional technique: pt refusal, bacteremia, hypovolemia, hemorrhage, coagulopathy, neuropathy

**General anesthesia**

- Uremic pts may have hypersensitivity to CNS drugs due to increased permeability of blood-brain barrier.
- Uremia causes delayed gastric emptying & increased acidity, leading to increased risk of aspiration pneumonitis (consider sodium citrate, H2-receptor blocker, metoclopramide).
- Hypoalbuminemia leads to increased free drug concentration in drugs that are bound to albumin (ie, thiopental).
- Succinylcholine is relatively contraindicated, as it causes approx. 1-mEq/L increase in serum potassium, which may precipitate cardiac dysrhythmias.
- Use caution w/ drugs dependent on renal excretion (gallamine, vecuronium, pancuronium) & clearance (mivacurium, rocuronium).

## CAVEATS & PEARLS

- Succinylcholine is relatively contraindicated, as it causes approx. 1-mEq/L increase in serum potassium, which may precipitate cardiac dysrhythmias.
- Use caution w/ drugs dependent on renal excretion (gallamine, vecuronium, pancuronium) & clearance (mivacurium, rocuronium).
  - ➢ Cisatracurium is a good muscle relaxant for pts w/ renal dysfunction, as its clearance is independent of renal function (Hoffman degradation).
  - ➢ Standard doses of anticholinesterases are used for reversal of neuromuscular blockade.
  - ➢ Consider whether pt is likely to have platelet dysfunction (secondary to uremia) or significant peripheral neuropathy before doing regional anesthetic.

## CHECKLIST

- Check OB plan for pt.
- Document degree of renal dysfunction.
- Be prepared to manage hypertension.

# ACUTE INTERSTITIAL NEPHRITIS

JOSHUA WEBER, MD
PETER DUNN, MD

## FUNDAMENTAL KNOWLEDGE

Acute interstitial nephritis is usually characterized by development of acute renal failure after starting a known offending drug.

**Causes**

- Drugs: antibiotics, NSAIDs
- Infection
- Sarcoidosis
- Systemic lupus
- Idiopathic

**Presentation**

- Rash
- Fever
- Eosinophilia

## STUDIES

Lab tests commonly ordered to evaluate renal function in pregnancy include:

- Creatinine
- BUN

Expect acute elevations in BUN & creatinine

- Electrolytes
- Creatinine clearance
- CBC
- Urinalysis will show white cells, red cells & white cell casts.

In addition to labs commonly evaluated in pregnancy, the following tests may be performed:

- Decreased urine output
- Eosinophilia in serum
- Renal biopsy
- Gallium scan (high false-negative rate)

## MANAGEMENT/INTERVENTIONS

**Medical/OB mgt of AIN**

- Trial of corticosteroids (improvement usually seen in 1–2 weeks)

**Increased frequency of prenatal visits (q2 wks in 1st & 2nd trimesters, then q1 week)**

- Monitoring: monthly measurements of serum creatinine, creatinine clearance, fetal development, BP
- Erythropoietin may be used for maternal anemia.
- Preterm delivery is considered for worsening renal function, fetal compromise or pre-eclampsia.
- Renal biopsy is considered if rapid deterioration in renal function occurs prior to 32 weeks gestation.

**Anesthetic mgt in pts w/ renal dysfunction**

Pre-op

- Evaluate degree of renal dysfunction & hypertension.
- Evaluate for anemia & electrolyte abnormalities.
- Intraoperative mgt:
- Monitors: standard monitoring + fetal heart rate (FHR) +/− arterial line +/− CVP
- Limit fluids in pts w/ marginal renal function to prevent volume overload.

**Regional anesthesia**

- Increased toxicity from local anesthetics has been reported in pts w/ renal dysfunction, although esters & amides can be used safely.
- Contraindications to regional technique: pt refusal, bacteremia, significant hypovolemia, severe hemorrhage, coagulopathy, potentially preexisting neuropathy

**General anesthesia**

- Succinylcholine may be relatively contraindicated, as it causes approx. 1-mEq/L increase in serum potassium, which may precipitate cardiac dysrhythmias.
- Use caution w/ drugs dependent on renal excretion (gallamine, vecuronium, pancuronium) & clearance (mivacurium, rocuronium).

**CAVEATS & PEARLS**

- Trial of corticosteroids (improvement usually seen in 1–2 weeks) may be indicated.
- Succinylcholine may be relatively contraindicated, as it causes approx. 1-mEq/L increase in serum potassium, which may precipitate cardiac dysrhythmias.
- Use caution w/ drugs dependent on renal excretion (gallamine, vecuronium, pancuronium) & clearance (mivacurium, rocuronium).

- Cisatracurium is a good muscle relaxant for pts w/ renal dysfunction, as its clearance is independent of renal function (Hoffman degradation).
- Standard doses of anticholinesterases are used for reversal of neuromuscular blockade.
- Consider whether pt is likely to have platelet dysfunction (secondary to uremia) or significant peripheral neuropathy before doing regional anesthetic.

**CHECKLIST**
- Check OB plan for pt.
- Document degree of renal dysfunction.
- Be prepared to manage hypertension.

## ACUTE TUBULAR NECROSIS

JOSHUA WEBER, MD
PETER DUNN, MD

**FUNDAMENTAL KNOWLEDGE**
- Acute tubular necrosis (ATN) is an *intrarenal* cause of acute renal failure in pregnancy. Characterized by renal vasoconstriction, which leads to a 50% decrease in blood flow

**Pathogenesis**
- Ischemia: hypotension leads to decreased blood flow, which damages medullary cells
- Toxins: cause damage to proximal tubules

**Causes of ATN**
- Trauma/rhabdomyolysis
- Renal ischemia from septic/hemorrhagic/hypovolemic shock
- Nephrotoxic drugs
- Amniotic fluid embolism
- Intrauterine fetal death

**STUDIES**
- Creatinine (may see acute elevation)
- BUN (may see acute elevation)
- BUN/creatinine ratio 10:1
- Electrolytes
- Creatinine clearance

- CBC
- Urinalysis
- Decreased urine output
- Urinalysis -> brown epithelial cell casts
- Urine osmolality <350 mOsm/kg water
- Urine sodium >40 mEq/L
- Fractional sodium excretion (FENa) >1%
- Urinary/plasma creatinine <20

## MANAGEMENT/INTERVENTIONS

**Medical/OB mgt of ATN**

- Rule out DIC.
- For decreased renal function
  - ➢ Increased frequency of prenatal visits (q2 wks in 1st & 2nd trimesters, then q1 week)
  - ➢ Monitoring: monthly measurements of serum creatinine, creatinine clearance, fetal development, BP
  - ➢ Erythropoietin may be used for maternal anemia.
  - ➢ Preterm delivery is considered for worsening renal function, fetal compromise or pre-eclampsia.
  - ➢ Renal biopsy is considered if rapid deterioration in renal function occurs prior to 32 weeks gestation.
  - ➢ See "***Parturients on Dialysis***" for dialysis mgt.

**Anesthetic mgt for pts w/ new-onset renal dysfunction**

**Pre-op**

- Evaluate degree of renal dysfunction & hypertension.
- Evaluate for anemia & electrolyte abnormalities.

**Intraoperative mgt**

- Monitors: standard monitoring + fetal HR +/− arterial line +/− CVP
- Limit fluids in pts w/ marginal renal function to prevent volume overload.

**Regional anesthesia**

- Uremia-induced platelet dysfunction leads to increased bleeding time.
- Pts may have thrombocytopenia from peripheral destruction of platelets.
- Increased toxicity from local anesthetics has been reported in pts w/ renal disease, but amide & ester local anesthetics can be used safely.
- Contraindications to regional technique: pt refusal, bacteremia, hypovolemia, hemorrhage, coagulopathy, neuropathy

**General anesthesia**

- Uremic pts may have hypersensitivity to CNS drugs due to increased permeability of blood-brain barrier.
- Uremia causes delayed gastric emptying & increased acidity, leading to increased risk of aspiration pneumonitis (consider sodium citrate, H2-receptor blocker, metoclopramide).
- Hypoalbuminemia leads to increased free drug concentration of drugs that are bound to albumin (ie, thiopental).
- Succinylcholine is relatively contraindicated, as it causes approx. 1-mEq/L increase in serum potassium, which may precipitate cardiac dysrhythmias.
  - ➢ Use caution w/ drugs dependent on renal excretion (gallamine, vecuronium, pancuronium) & clearance (mivacurium, rocuronium).

**CAVEATS & PEARLS**

- BUN >18 & creatinine >0.8 are signs of renal dysfunction in pregnancy.
- Goals are to maintain renal perfusion & euvolemia.
- ATN may require diuretics, vasopressors & alkalinization.

**CHECKLIST**

- Check OB plan for pt.
- Document degree of renal dysfunction.
- Beware of electrolyte imbalances.

## ADRENAL INSUFFCIENCY

GRACE C. CHANG, MD, MBA; ROBERT A. PETERFREUND, MD, PhD; AND STEPHANIE L. LEE, MD, PhD

**FUNDAMENTAL KNOWLEDGE**

See "*Hypercortisolism.*"

**Physiology**

- Results from inadequate basal or stress level of plasma cortisol
- Diagnosis is imperative, as failure to initiate treatment can be fatal.

**Epidemiology**

- Rare endocrine condition; even more rare in pregnancy

**Signs/Symptoms**

- Weakness

- Altered consciousness, progressing from inattention to delirium to coma
- Malaise
- Slow respiration
- Nausea/vomiting
- Hypoglycemia
- Hypotension
- Hypothermia
- Bradycardia
- Cyclical fever
- Abdominal pain

**Etiology**

- Drugs (etomidate, ketoconazole, abrupt cessation of glucocorticoids or Megace therapy)
- Preexisting conditions that inhibit synthesis of steroid can suppress normal corticosteroid response & lead to adrenal insufficiency.
- Infections
- Tumors
- Adrenal hemorrhage
- Coagulopathy
- Postpartum pituitary necrosis (Sheehan's syndrome)

**Complications**

- Pregnancy & live birth can be expected in 87% of treated pts, compared w/ 54% of untreated pts.

**STUDIES**

- Initial tests should evaluate levels of ACTH & TSH.
- Use provocative tests such as ACTH (cosyntropin) test to provoke responses of cortisol & aldosterone.
- Plasma cortisol in nonpregnant pts should rise at least 12 mcg/dL above baseline, increase 2–3× over baseline & exceed 18 mcg/dL, & reach a maximal level at 60 minutes.
- Normal range of aldosterone: 7–35 ng/dL
- Primary adrenal insufficiency: baseline aldosterone levels are low & there is no response to cosyntropin
- Secondary adrenal insufficiency: baseline aldosterone levels may be low or normal, but then should rise to at least 4 ng/dL at 30 minutes after injection
- Check TSH w/ free T4 level in secondary adrenal insufficiency to exclude other pituitary deficiencies.

- Autoimmune primary adrenal insufficiency is associated w/ primary hypothyroidism.

### MANAGEMENT/INTERVENTIONS

- Hormone replacement therapy depends on type & extent of lesion & degree of hormone deficit.
- In acute severe adrenal crisis, give 100 mg hydrocortisone IV, w/ gradual taper (50 mg every 8 hours over 1–2 days), & then oral administration of usual steroid replacement.
- Use aggressive IV hydration w/ D5 to prevent hypoglycemia.
- Correct thyroid deficiency w/ L-thyroxine.
- Consider mineralocorticoid replacement w/ fludrocortisone, although secondary adrenal insufficiency does not require mineralocorticoid therapy.
- Adrenal insufficiency requires continued replacement of corticosteroid throughout pregnancy, w/ stress doses at delivery (50–100 mg IV q8h or as continuous infusion).
- During stressful events, such as labor, increased doses of corticosteroids are required to prevent adrenal crisis.

#### Anesthetic Management

- Both general & regional anesthesia can be safely used.
- Epidural anesthesia is recommended to decrease stress response from labor.
- Aggressive volume replacement
- Monitor urine output closely; decreased renal blood flow is associated w/ adrenal insufficiency.
- Give stress-dose steroids (100 mg hydrocortisone IV) pre-op, or prior to labor or C-section, or if sudden hypotension occurs.
- In life-threatening adrenal insufficiency, treat w/ hydrocortisone 100 mg IV bolus or continuous IV infusion of hydrocortisone, 10 mg/hr.
  - ➢ Invasive monitoring w/ arterial line & pulmonary artery catheter may be advisable.
- Use IV & inhalational anesthetics carefully, since they can cause myocardial depression & hypotension.
- Carefully monitor electrolytes; adrenal insufficiency is associated w/ hyponatremia, hypokalemia, hypoglycemia.
- Pt may have reduced non-depolarizing muscle relaxant requirement.
- Monitor muscle blockade carefully w/ peripheral nerve stimulator.
- Avoid etomidate, as it is associated w/ adrenal suppression.

### CAVEATS/PEARLS

- Adrenal insufficiency is associated w/ skeletal muscle weakness; pt may require less non-depolarizing neuromuscular blocking agents.
- Adrenal insufficiency w/ fever & abdominal pain can mimic an abdominal infectious process.

### CHECKLIST

- Give stress-dose steroids (100 mg hydrocortisone IV) pre-op, during labor & prior to C-section. With prolonged labor, continue with hydrocortisone 50–100 mg IV q8h.
- Monitor hemodynamics & fluid status closely.
- Be prepared to manage adrenal crisis.
- Monitor electrolytes closely.
- Use non-depolarizing neuromuscular blocking agents judiciously.

## ALCOHOLIC HEPATITIS

LAWRENCE WEINSTEIN, MD
DOUG RAINES, MD

### FUNDAMENTAL KNOWLEDGE

- Alcoholic hepatitis is characterized by acute or chronic inflammation & hepatic necrosis.
- Often reversible, but may be a precursor to chronic alcoholic cirrhosis
- Presentation can range from an asymptomatic pt w/ hepatomegaly to a critically ill person w/ a fulminant course.
- Physical symptoms & signs include abdominal pain, fever, ascites, jaundice & encephalopathy.
- Recent heavy drinking, hepatomegaly, jaundice & anorexia strongly support diagnosis.

### STUDIES

- Elevated transaminases
  - ➢ AST is usually elevated to a greater extent than ALT.
- Elevated alkaline phosphatase
- Bilirubin elevated in >50% of pts
- PT can be elevated w/ advanced hepatocellular compromise. High elevations in PT correlate w/ increased mortality; see the section on "General Concepts."
- Serum albumin decreased

- Macrocytic anemia may be present secondary to dietary deficits of folate & vitamin B12.
- Thrombocytopenia is seen in about 10% of pts.
- Liver biopsy is diagnostic & shows macrovesicular fat, neutrophilic infiltration, hepatic necrosis & Mallory bodies.
- Ultrasound can be useful to rule out biliary obstruction.

**MANAGEMENT/INTERVENTIONS**

- Abstinence from alcohol is essential. Pts should be provided w/ nutritional support (especially folate & thiamine).
- Corticosteroids may be of benefit in reducing short-term mortality.
- For pts w/ alcoholic hepatitis specifically, a history of alcohol use may have resulted in alcoholic cardiomyopathy w/ compromised ventricular function. For discussion of mgt of the parturient w/ cardiomyopathy, see "*Valvular Disease*" chapter.
- As w/ all liver disease pts, anesthetic decisions should be guided by the symptoms & manifestations of the liver process. Various manifestations will have differing effects on physiology & choice of anesthetic technique for labor or delivery.
  - ➢ If pt is coagulopathic, see the section on "*General Concepts*."
  - ➢ If pt is cirrhotic, see sections on "*General Concepts*," "*Cirrhosis*," "*Portal Hypertension*," "*Ascites*," "*Hepatic Encephalopathy*."
  - ➢ For general discussion of regional & general anesthesia considerations, see the section on "*General Concepts*."

**CAVEATS/PEARLS**

- Alcoholic hepatitis can be suspected in pts w/ elevated aminotransferases & a history of excessive alcohol use.
- Treatment is mostly supportive, w/ abstinence from alcohol being essential.
- Anesthetic decisions are based on physiologic manifestations present at time of surgery or delivery.
- Cardiac function may be compromised from long-term alcohol abuse; an echocardiogram may be prudent in this population.

**CHECKLIST**

- Watch for other alcohol-related complications, including those of withdrawal.
- Check coagulation status prior to initiation of regional anesthesia.

# AMNIOTIC FLUID EMBOLUS (AFE)

JASON JENKINS, MD
LEE WESNER, MD

## FUNDAMENTAL KNOWLEDGE

**Incidence, morbidity, mortality**

- While rare overall, true incidence is unknown due to difficulty in diagnosis. Clinical or fatal episodes occur in 1:8,000 to 1:80,000 pregnancies.
- High mortality: 80%, frequently within 5 hours of onset
- Only 15–25% of survivors remain neurologically intact.

**Risk factors**

- No specific risk factors other than pregnancy have been identified at this time.

**Etiology**

- Remains uncertain. Cardiovascular collapse does not appear to be due to an embolic process, as previously thought. Humoral mechanisms are currently suspected, & prognosis appears worse w/ meconium-stained amniotic fluid. Evidence does not support hypertonic uterine contractions as causative.

**Clinical presentation**

- General: classically described as sudden & profound cardiopulmonary collapse in a pregnant pt after an unexpected episode of dyspnea & restlessness. May occur throughout pregnancy, labor & immediate post-partum period.
- Cardiovascular: hypotension & tachycardia progressing to arrhythmias & cardiac arrest. Fatal right ventricular (RV) failure early due to pulmonary vasospasm, followed by left ventricular (LV) failure. Low cardiac output.
- Pulmonary: dyspnea, hypoxia, wheezing or bronchospasm, pulmonary edema, cough, ARDS
- Neurologic: anxiety, restlessness, headache, unconsciousness, convulsions
- Hematologic: consumptive coagulopathy progressing to disseminated intravascular coagulation (DIC). Severe DIC may lead to exsanguinating hemorrhage.
- Fetal: bradycardia, hypotension. Perinatal mortality near 80%, <40% of survivors neurologically intact.

## STUDIES

AFE is primarily a clinical diagnosis of exclusion. Do not delay CPR for confirmatory studies.

### Lab tests

- Hematologic: overall picture of coagulopathy
  - ➢ Thrombocytopenia
  - ➢ Decreased fibrinogen
  - ➢ Elevated d-dimer or fibrinogen split products
  - ➢ Prolonged PT or PTT
- ABGs may show metabolic acidosis.
- PA blood smear: presence of squamous cells is not diagnostic.

### Imaging

- Ultrasound
  - ➢ Echocardiography: may see LV or RV dysfunction, possibly pulmonary hypertension
- Radiographic
  - ➢ Angiography, CT, V-Q scans useful in eliminating other diagnoses

### Monitoring

- Standard noninvasive monitors (oximetry, ECG, etc.)
- Invasive monitoring guides fluid mgt.
  - ➢ Pulmonary artery (PA) line: monitor cardiac output, filling pressures, obtain mixed venous blood samples. Pulmonary hypertension often resolves by the time PA monitoring begins.
  - ➢ Arterial line: useful for pressure monitoring & frequent blood sampling

## MANAGEMENT/INTERVENTIONS

### General principles

- Treatment is generally supportive & symptom-directed. Since most pts are previously young & healthy, prompt & aggressive resuscitative measures are appropriate.

### Cardiovascular

- Vascular
  - ➢ Volume: rapid volume infusion to optimize preload
  - ➢ Systemic vasopressors (eg, phenylephrine) to support systemic perfusion
  - ➢ Pulmonary vasodilators (eg, NO, prostacyclin) may be useful adjuncts, but few data exist for their use in AFE.

- ➢ Diuretics may be required to treat pulmonary edema following major volume infusion.

- Cardiac
  - ➢ Inotropic support (eg, dopamine, dobutamine) is often needed to treat cardiogenic shock.
  - ➢ Full or partial cardiopulmonary bypass, though rare, has been used successfully.
  - ➢ Afterload reduction may be required to promote cardiac output, but must be balanced carefully w/ need to maintain systemic perfusion.

**Pulmonary**

- Goal is providing maximal oxygenation, & almost all pts require emergent endotracheal intubation. Initial hypoxia may be severe enough to result in permanent neurologic injury.
- Ventilatory support can be challenging, requiring CPAP or PEEP. ARDS can develop in severe cases, further complicating mgt. ECMO may be beneficial in the direst cases.

**Hematologic**

- PRBCs to treat massive hemorrhage often associated w/ AFE
- Platelets as appropriate for thrombocytopenia
- FFP is generally used for coagulopathy, but cryoprecipitate may be useful if volume overload is concerning.

**Neurologic**

- Maintain perfusion pressure & oxygenation. Hypoxic neurologic insult is common & devastating.

**Fetal**

- Prompt cesarean delivery may assist maternal survival.
- Perimortem delivery may allow fetal survival.

## CAVEATS/PEARLS

- AFE is devastating, not preventable & not predictable. It is a diagnosis of exclusion & must be considered quickly.
- Some suggest "anaphylactoid syndrome of pregnancy" & feel embolus is misnomer.
- Epithelial cells in maternal serum nondiagnostic for AFE, but presence of vernix/lanugo on PA blood smear may support diagnosis.
- Anaphylaxis-like picture in AFE; may consider epinephrine/corticosteroids if other modalities fail.
- Pulmonary hypertension may resolve before placement of PA catheter.

- Cardiopulmonary bypass, embolectomy, inhaled pulmonary vasodilators uncommon but successful use reported.

**CHECKLIST**
- High index of suspicion; consider & treat early.
- Cardiovascular support: volume, pressors, inotropes
- Pulmonary support: intubation & ventilation
- Hematologic treatment: large volumes of blood products
- Prompt fetal delivery

## AMYOTROPHIC LATERAL SCLEROSIS (ALS)

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**FUNDAMENTAL KNOWLEDGE**

**Definition**
- Progressive neurodegenerative motor neuron disease

**Epidemiology**
- Progressive respiratory paralysis; median survival 3–5 years
- Incidence 1–3 per 100,000
  Males > females
- Sporadic but 5–10% familial (autosomal dominant)

**Pathophysiology**
- Pathologic hallmark: death of both lower motor neurons (anterior horn cells) & upper motor neurons (corticospinal)
- Sensory apparatus is unaffected.

**Clinical Manifestations**
- Variable depending on degree of upper or lower motor neuron involvement
- Sensory, bowel, bladder & cognitive function are spared.

**Effect of Pregnancy on ALS**
- Mainly unaffected, although can increase respiratory compromise

**Effect of ALS on Pregnancy & the Fetus**
- ALS does not appear to have any effect on the fetus/pregnancy.
- Can increase the incidence of instrumented deliveries due to lack of muscle strength

## STUDIES

**History & Physical**

- Muscle weakness
- Hyperreflexia
  - ➢ Fasciculations
- Muscle atrophy
- History of bulbar weakness
- History of respiratory involvement

**Imaging**

- MRI
- Contrast myelography

**Other**

- Pulmonary function tests to assess baseline respiratory status

## MANAGEMENT/INTERVENTIONS

**Medical Treatment**

- Supportive
- Physical therapy
- Riluzole (glutamate antagonist): modest lengthening of trach-free survival
- Celecoxib (COX-2 inhibitor) & minocycline (tetracycline antibiotic) have been used.

**Anesthesia**

- Predelivery assessment
  - ➢ Evaluate respiratory status prior to & during labor.
  - ➢ Determine presence of bulbar weakness and increased risk for aspiration.
  - ➢ Document preexistent weakness.
- General anesthesia
  - ➢ Suggested if severe respiratory dysfunction exists
  - ➢ Increased incidence of aspiration; aspiration prophylaxis is recommended
  - ➢ Increased sensitivity to muscle relaxants
  - ➢ Avoid succinylcholine due to risk of hyperkalemia & rhabdomyolysis.
- Regional anesthesia
  - ➢ Epidural can be used w/o decreased neurologic function.
  - ➢ Spinal anesthesia: conflicting reports; has been associated w/ worsened neurologic function post-op in a few cases, but also no decrease in neurologic function in other cases.

**CAVEATS & PEARLS**
- Progressive neurodegenerative disease affecting both upper & lower motor neurons,
- Hallmarks are motor weakness & hyperreflexia.
- Pregnancy contributes to increased respiratory compromise.
- Bulbar paralysis increases risk of aspiration
- Increased sensitivity to muscle relaxants
- Avoid succinylcholine.
- May require post-op ventilation
- Regional anesthesia has been used without altering baseline neurologic function.

**CHECKLIST**
- Document preexisting neurologic dysfunction.
- Carefully assess pulmonary status at start of & during labor.
- Pulmonary function tests are recommended if pulmonary involvement is present.
- Aspiration prophylaxis
- General anesthesia if severe respiratory dysfunction present
- Avoid succinylcholine.

## ANEMIA

JEANNA VIOLA, MD
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**FUNDAMENTAL KNOWLEDGE**
- Anemia = a low erythrocyte count; it can be further classified as macrocytic, normocytic or microcytic.
- Causes vary from nutritional deficiency to disorders of hemoglobin production.
- Common diagnosis in pregnancy, usually due to hemodilutional state or deficiency of iron and/or folate
- Certain pts may have chronic anemia that precedes pregnancy: pts w/ renal disease or those taking drugs that impair erythropoiesis (certain anti-rheumatologic therapy).
- Most parturients tolerate anemia well.

**STUDIES**
- Hemoglobin, Hct, MCV
- Further studies are directed at determining the cause of anemia.

- ➢ Microcytic anemia: iron, ferritin, TIBC levels; consider hemoglobin electrophoresis to diagnose thalassemia
- ➢ Macrocytic anemia: check folate, vitamin B12 levels
- ➢ Normocytic anemia: may be a combination of nutritional deficiencies

**MANAGEMENT/INTERVENTIONS**

- Transfusion of PRBCs usually not warranted
  - ➢ Guide decision to transfuse by evidence of poor hemodynamic status, severe symptoms of anemia (shortness of breath, dyspnea on exertion, flow murmur), active bleeding or hemolysis.
  - ➢ Weigh benefits of transfusion against risks of clerical error, bacterial or viral infection or transfusion reaction.
- Give oxygen to pts w/ Hgb <8 during delivery.
- Maintain a warm environment for pts w/ cold agglutinin-induced hemolytic anemia.
- If anemia exists as part of pancytopenia, pt may also require a platelet transfusion or antibiotics for neutropenia-induced infections.
- Regional anesthesia is not contraindicated unless the anemia is associated w/ a thrombocytopenia or coagulopathy.

**CAVEATS AND PEARLS**

- Most parturients compensate for the anemia well.
- Regional anesthesia is not contraindicated unless there is a concomitant thrombocytopenia or coagulopathy.

**CHECKLIST**

- Check CBC to assess severity of anemia & to rule out other cytopenias.
- Evaluate how well the pt has compensated for the anemia:
  - ➢ Shortness of breath?
  - ➢ Dyspnea on exertion?
  - ➢ Presence of flow murmur?
- Administer supplemental oxygen.

Send blood sample for type & cross if transfusion may be warranted.

# ANESTHESIA FOR CESAREAN DELIVERY

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## FUNDAMENTAL KNOWLEDGE

*Cesarean delivery*: the delivery of a fetus by surgical incision through the abdominal wall & uterus

*Incidence*: In 2002, 26.1% of deliveries in the U.S. were via cesarean delivery. This has increased in recent years due to the lower rates of vaginal birth after cesarean delivery (VBAC), among other factors.

### Common indications

- Prior cesarean delivery (about 35%)
- Dystocia or cephalopelvic disproportion (30%)
- Breech presentation (12%)
- Non-reassuring fetal heart rate tracings (9%)

### Morbidity/mortality

- About 80% of cases of anesthesia-related maternal deaths occurred during cesarean deliveries.
- The case fatality rate associated w/ general anesthesia is about 15 times higher than w/ regional techniques.
- The increased use of regional anesthesia for cesarean delivery has led to a significant decrease in anesthesia-related maternal mortality.

### Surgical considerations

- Preferably performed by lower uterine incision (less blood loss, lower incidence of rupture during next pregnancies). Classic uterine incision is rarely used but is necessary in some pts (more blood loss, introduces need for a repeat cesarean delivery w/ subsequent pregnancies).
- Operative time varies according to obstetrician & clinical situation; usually 20–90 minutes, longer for classic incision.
- Usual blood loss 800–1,000 mL; may be more w/ repeat cesarean delivery, classic incision on uterus, previous abdominal surgery, multiple gestation, placenta accreta, abruption, etc.

### Anesthetic considerations

- Anesthesia choices: spinal, epidural, combined spinal epidural, general, local.

- Special concerns: airway, full stomach, placental transfer of medications, pre-existing conditions (obstetric or medical), potential for blood loss
- Requires constant communication w/ obstetric care provider, nurses & other personnel

**Classification (based on the level of urgency)**

- **Elective:** scheduled at a time to suit the maternity unit and/or the OB floor
- **Emergency:** not performed on a scheduled basis
  - ➢ Stable: Fetal & maternal physiology is stable, but surgery is required before destabilization occurs
  - ➢ Urgent: Maternal and/or fetal physiology is unstable but is not immediately life-threatening to either of them
  - ➢ Emergent (stat): Pt has a condition that is immediately life-threatening to mother or fetus

**Indications**

- **Elective**
  - ➢ Mother's request
  - ➢ Deteriorating maternal medical illness (cardiac or pulmonary disease)
  - ➢ Maternal infection w/ HIV
  - ➢ Active genital herpes simplex virus infection in mother
  - ➢ Previous classic cesarean delivery
  - ➢ Previous myomectomy or uterine or vaginal reconstruction
  - ➢ Cervical cancer
  - ➢ Mechanical obstruction to vaginal birth (large leiomyoma or condyloma acuminata, severely displaced pelvic fracture)
  - ➢ Placenta previa
  - ➢ Suspected placenta accreta
  - ➢ Fetopelvic disproportion
  - ➢ Transverse or oblique lie
  - ➢ Most breech presentations
  - ➢ High-order multiple pregnancy
  - ➢ Fetal macrosomia
  - ➢ Fetal bleeding diathesis

**Controversial**

- Twin pregnancy
- Maternal hepatitis C infection

- Severe preeclampsia/eclampsia remote from term
- Certain fetal congenital abnormalities (gastroschisis, neural tube defects)
- Previous cesarean delivery (see "*Management of TOLAC or VBAC*")
  - ➢ **Stable**
    - Chronic uteroplacental insufficiency
    - Abnormal fetal presentation w/ ruptured membranes but not in labor
    - Dysfunctional uterine activity
  - ➢ **Urgent**
    - Failure to progress in labor
    - Active herpes w/ rupture of membranes
    - Non-bleeding placenta previa in labor
    - Cord prolapse w/o fetal distress
    - Variable FHR decelerations w/ prompt recovery & normal FHR variability
  - ➢ **Stat**
    - Agonal fetal distress (prolonged severe fetal bradycardia, late FHR decelerations w/ no FHR variability)
    - Cord prolapse w/ fetal distress
    - Massive hemorrhage (placenta previa, abruption, etc.)
    - Ruptured uterus

**Anesthesia effects on the neonate**

- Some studies have concluded that regional or general anesthesia does not cause significant changes in the fetal acid-base status as long as maternal hypotension & hypoxia are avoided.
- There is disagreement among studies as to whether general anesthesia is associated w/ lower 1-min Apgar scores.
- Inhalational agents cause only mild fetal depression when given in doses <1 MAC & if delivery occurs <10 minutes from induction.
- Uterine incision-to-delivery (U-D) interval >3 minutes is associated w/ a higher incidence of low fetal blood pH & Apgar scores.
- In some studies, w/ general anesthesia, induction-to-delivery (I-D) interval >8 minutes is associated w/ a higher incidence of low fetal blood pH & Apgar scores.
- Induction agents in usual doses & local anesthetics given spinally or epidurally have minimal effects on the fetus. Benzodiazepines & opiates may cause fetal depression & their use is minimized before the delivery. Muscle relaxants do not cross the placenta.

**FHR monitoring**

- Used by obstetricians as an indicator of fetal status, although it has a relatively low positive-predictive value for abnormal fetal condition. The false-positive rate can be as high as 50%.
- An external Doppler placed on the abdomen can provide continuous monitoring of the fetal heart rate & pattern. A fetal scalp electrode may be placed on the baby's head to obtain a more accurate tracing. Normal FHR is 120–160 bpm w/ good beat-to-beat variability. Long-term variability is also considered.
- The tracing can be within normal limits (as above) or suggestive of fetal compromise (non-reassuring). Non-reassuring FHR (late decelerations, deep variable decelerations, fetal tachycardia w/ loss of beat-to-beat variability, fetal bradycardia, undulating baseline, etc.) may prompt emergency delivery or further testing.
- Fetal scalp blood sampling may clarify a suspicion of compromised fetal well-being. A fetal scalp pH $> 7.25$ is normal, whereas a pH $<$ 7.20 requires rapid delivery.
- The severity of the fetal compromise indicated by the abnormal FHR determines the urgency of the situation & the anesthetic choice.
- For chronic fetal distress (IUGR, etc.) the anesthetic choice of choice is regional, whereas acute severe fetal distress may require general anesthesia.
- Non-reassuring FHR may resolve or improve w/ certain maneuvers (maternal position change, adjustment of oxytocin, treatment of hypotension, etc.). Frequent monitoring & communication w/ the obstetric team may obviate the need for emergency induction of general anesthesia.

**STUDIES**

- Antepartum anesthesia consultation is recommended in some cases:
  - ➢ Massive obesity
  - ➢ Asthma requiring medications
  - ➢ Severe hypertensive disease
  - ➢ Maternal coagulopathy
  - ➢ Serious medical or obstetrical conditions
  - ➢ History of anesthetic complications
  - ➢ Severe facial or neck edema
  - ➢ Extremely short stature

- ➢ Decreased mobility of the neck, difficulty opening the mouth
- ➢ Anatomic abnormalities of face, mouth, neck or jaw

- Past medical/surgical history, anesthetic history
- Physical exam, airway exam
- Hemoglobin
- Type & screen; cross-match if significant loss of blood is anticipated
- Coagulation times (PT, PTT, fibrinogen) & platelet count if preeclampsia, heavy maternal bleeding, abruptio placentae or preexisting bleeding diathesis
- Check any recent imaging studies (previous ultrasound, MRI, etc.) to clarify any abnormalities that may affect the anesthetic plan (e.g., uterine or placental abnormalities).
- Other studies as indicated from history & physical

## MANAGEMENT/INTERVENTIONS

**Preparation for Anesthesia**

- **NPO status**
  - ➢ In the uncomplicated parturient, no solid food for at least 6, preferably 8 hours prior to elective cesarean delivery. Clear liquids can be ingested until 2 hours before surgery.
  - ➢ Laboring women should avoid solid food & restrict their oral intake to clear liquids.
  - ➢ All parturients are considered "full stomach" regardless of their NPO status.
- **Pt education, anxiolysis & reassurance**
  - ➢ A concise explanation of the anesthetic procedure & common side effects during & immediately after placement helps allay fears in most pts.
  - ➢ Constant reassurance as the regional anesthetic is evolving is also helpful.
  - ➢ Benzodiazepines & opiates in small doses can be given for severe anxiety (midazolam IV 0.5–2 mg, diazepam IV 2–5 mg, fentanyl IV 25–50 mcg). Amnesia & fetal depression are risks.
  - ➢ Support person (e.g., the father of the baby) present during cesarean delivery under regional anesthesia provides reassurance & emotional support.
- **Prophylactic IV fluid before induction**
  - ➢ Was found to reduce the incidence of maternal hypotension & to improve uteroplacental perfusion
  - ➢ Up to 15–20 mL/kg of crystalloid (Ringer's lactate, normal saline) in pts receiving regional anesthesia

- ➢ Avoid glucose or dextrose in the IV fluids because it causes maternal hyperinsulinemia w/ resultant fetal hypoglycemia in early postpartum.
- ➢ Colloids may be better than crystalloids because they remain in the intravascular space for a longer time, but they are more expensive & can alter the blood rheology & platelet function.
- ➢ Generally, the prophylactic administration of IV fluids should not delay the induction of anesthesia in cases that require immediate cesarean delivery.

- **Prevention of acid aspiration**
  - ➢ All parturients are considered at risk for aspiration regardless of the planned anesthetic.
  - ➢ Oral non-particulate antacids (sodium citrate 30 mL), given 15–30 minutes prior to induction, increase the pH of the gastric contents. Avoid particulate antacids because they may cause pulmonary injury similar to that caused by gastric acid, if aspirated.
  - ➢ Metoclopramide (10 mg IV) increases gastric emptying, acts as an antiemetic & increases the tone of the lower esophageal sphincter.
  - ➢ Intravenous H2-receptor antagonists or proton pump inhibitors combined w/ sodium citrate cause a greater increase in the gastric pH if administered at least 30 minutes before intubation.
  - ➢ Empty the stomach w/ an orogastric tube in all pts receiving general anesthesia.
- **Supplemental oxygen**
  - ➢ Indicated in cases of emergency cesarean delivery when the fetus and/or the mother is in distress.
  - ➢ No documented benefit was found in neonates of healthy parturients having an elective cesarean delivery.
- **Maternal positioning during & before cesarean delivery**
  - ➢ In the supine position the gravid uterus compresses the aorta & inferior vena cava, leading to decreased venous return, uterine artery perfusion & cardiac output.
  - ➢ Maintain left uterine displacement by placing a wedge (folded or rolled blankets) under the pt's right hip.

**Monitoring during anesthesia**

- Standard monitors (noninvasive BP, EKG, pulse oximeter), urine output
- Capnograph & temperature for general anesthesia

- Consider an arterial line for severe preeclampsia or massive blood loss.
- Consider central pressure monitoring as maternal condition warrants (e.g., significant maternal heart disease).

**Choice of anesthetic**

- Consider the indication for surgery, the urgency of the procedure, maternal coexisting disease & maternal preference. The anesthetic plan should be discussed w/ the obstetrician.
- Regional anesthesia is the preferred technique for both elective & emergency surgery due to its safety profile. A sensory level of T4 is desirable.
  - ➢ Spinal anesthesia
    - First-choice anesthetic technique for elective cesarean delivery
    - Good choice for many emergency cesarean deliveries due to rapid onset & simplicity
    - Negligible maternal risk of systemic local anesthetic toxicity or local anesthetic depression of the infant
    - Rapid onset of sympathetic blockade may result in abrupt hypotension w/ reduced uteroplacental perfusion.
    - Less ideal technique in pts w/ severe respiratory compromise, due to blockade of the thoracoabdominal segments.
    - May not provide long enough anesthesia if surgery lasts >90–120 minutes
  - ➢ Continuous spinal anesthesia
    - Not a first-line technique due to the high incidence of postdural puncture headache w/ the 17- or 18-gauge needle/20-gauge catheter
    - Feasible technique in cases of accidental dural puncture if the catheter has threaded into the spinal space & CSF can be easily aspirated
    - Spinal drugs can be given in small doses to the desired effect, minimizing exaggerated BP changes. In addition, respiratory changes are less severe since the level is easier to control.
    - May be the technique of choice in select complicated obstetric pts; evaluate the risk:benefit ratio.
  - ➢ Epidural anesthesia
    - Slower onset, higher incidence of failed block & technically more involved than spinal anesthesia

- Less ideal in emergency cesarean delivery when time is of the essence
- Requires higher doses of local anesthetic that may be associated w/ significant maternal & fetal toxicity if injected intravascularly
- Severe hypotension or weakness of respiratory muscles can often be avoided or better addressed due to the incremental administration of drugs.
- A labor epidural w/ a demonstrable level can be extended to provide anesthesia for emergency cesarean delivery, thus obviating the need for general anesthesia.

➢ Combined spinal epidural anesthesia (CSE)

- Combines the advantages of spinal anesthesia (speed of onset, reliability) w/ the utility of an epidural catheter (ability to extend the duration of anesthesia by administering additional epidural drug)
- Preferred for elective cesarean deliveries when the anticipated surgical time is expected to be longer
- Technically more involved & may be more time-consuming than spinal anesthesia, thus limiting its use in an emergency situation

■ Contraindications to regional anesthesia

➢ Absolute: severe, uncorrected hypovolemia, anticoagulants or bleeding diathesis, severe aortic or mitral stenosis, increased intracranial pressure, infection at the site of injection

➢ Relative: uncooperative pt, preexisting neurologic deficits, stenotic valvular heart lesions, severe spinal deformity

■ General anesthesia

➢ The most reliable & rapid means of achieving adequate anesthesia for emergency stat cesarean delivery

➢ Should be reserved for cases in which adequate anesthesia cannot be achieved w/ regional techniques in a timely manner

➢ The overall risk of maternal mortality w/ general anesthesia is 17× higher than w/ regional anesthesia; most deaths are due to airway problems.

➢ If the airway cannot be secured in a timely manner, the safety of the mother takes priority over delivery of the fetus.

➢ Urgency of delivery & the severity of fetal distress should always be weighed against the maternal risks of general anesthesia. Most emergency cesarean deliveries can be safely done under a regional technique.

- ➢ In pts w/ recognized difficult airway, placement of an epidural catheter early in labor provides a partial block that can be promptly extended if emergency delivery is required.
- ➢ Other indications: coagulopathy or other conditions that preclude the use of a regional technique, inadequate regional anesthesia or maternal refusal of regional anesthesia

■ Local anesthesia
- ➢ Very rarely employed as the sole anesthetic technique
- ➢ Involves local infiltration w/ large amounts of local anesthetic w/ high risk of systemic toxicity
- ➢ Indications: when rapid administration of regional or general anesthesia is not possible (severe coagulopathy & a known difficult airway); selected pts w/ extremely debilitating diseases; anesthesia personnel are not available
- ➢ Can be used as an adjuvant in pts w/ incomplete spinal or epidural anesthesia

■ Anesthetic options
- ➢ Elective cesarean delivery: spinal, epidural or CSE anesthesia, general anesthesia
- ➢ Emergency stable & urgent cesarean deliveries: spinal or extended epidural anesthesia, general anesthesia
- ➢ Emergent (stat) cesarean deliveries: general anesthesia or extended epidural (if a preexistent T10 or higher sensory level is present)

**Anesthetic management**

■ **Elective cesarean delivery w/ regional anesthesia**

1. Check NPO status.
2. Clarify maternal, fetal & obstetric issues w/ obstetrician.
3. Administer IV fluids.
4. Administer sodium citrate 30 ml PO, metoclopramide 10 mg IV.
5. Apply standard monitors.
6. Consider supplemental $O_2$ by face mask or nasal prongs.
7. Place pt in sitting or lateral decubitus position.

   **Spinal**
   - Should be administered in the surgical suite on the operative table
   - L3–L4 lumbar puncture w/ a 24- to 27-gauge non-cutting spinal needle
   - Medications: hyperbaric solution of bupivacaine (10–15 mg); may add fentanyl 10–25 mcg (to increase the intensity &

duration of the block) & preservative-free morphine 0.2–0.3 mg (for up to 24 hours postop analgesia)

**Combined spinal epidural**

- Should be administered in the surgical suite on the operative table
- Identify epidural space at L3–L4 w/ standard epidural needle. Use the spinal needle-through-epidural needle technique for dural puncture.
- Medications: same as above
- Remove the spinal needle & thread the epidural catheter.
- If the block is patchy or the pt becomes uncomfortable as the surgery is prolonged, the catheter can be tested w/ 3 mL of 2% lidocaine w/ epinephrine 1:200,000 w/ sodium bicarbonate (1 mEq/10 mL lidocaine). Additional local anesthetic can then be titrated to pt comfort.

**Epidural**

- May be administered outside the surgical suite (slower onset)
- Place L2–L3 or L3–L4 epidural catheter.
- Administer local anesthetic (lidocaine or bupivacaine) in 3- to 5-mL aliquots while monitoring maternal BP & ensuring fetal well-being.
  - 15–20 mL 2% lidocaine w/ epinephrine 1:200,000 w/ sodium bicarbonate (1 mEq/10 mL lidocaine)
  - 15–20 mL 0.5% bupivacaine (may add 0.05 mEq sodium bicarbonate/10 mL bupivacaine for a faster onset). Beware of potential for precipitation if more sodium bicarbonate is used.
  - Other adjuvants
    - Fentanyl 50–100 mcg (to increase intensity & duration of the block)
    - Preservative-free morphine 3–5 mg (to prolong postop analgesia up to 24 hours)

8. Place pt supine on operative table, maintain uterine displacement & treat hypotension aggressively.
9. Reassure the pt as the numbing effects of the regional anesthetic evolve.
10. Clarify the level of anesthesia prior to incision.

■ **Elective cesarean delivery w/ general anesthesia**

1. Check NPO status.
2. Clarify maternal, fetal & obstetric issues w/ obstetrician.

3. Administer IV fluids.
4. Administer sodium citrate 30 mL PO & metoclopramide 10 mg IV (consider H2 antagonist or proton pump inhibitor 30 minutes before induction).
5. Apply standard monitors.
6. Place pt supine, maintaining left uterine displacement.
7. OR personnel can proceed w/ surgical prep & drape (minimizes induction-delivery interval).
8. If difficult airway is not suspected, proceed w/ denitrogenation 3–5 minutes 100% $O_2$ face mask.
9. Ensure proper pt positioning (elevation of the shoulders, "sniffing" position).
10. Rapid sequence induction w/ cricoid pressure
    - Medications: propofol 2 mg/kg or thiopental 4 mg/kg or ketamine 1 mg/kg & succinylcholine 1–1.5 mg/kg.
11. Intubation w/ a smaller cuffed ETT (6.5, 7.0). A short-handled laryngoscope can make intubation easier. Surgery can begin after confirmation of the proper placement of the ETT.
12. Maintenance on 50% nitrous oxide, oxygen & up to 0.5 MAC of volatile anesthetic. Avoid hyperventilation as it reduces uterine blood flow. Minimize IV narcotics until after delivery.
13. Aspirate gastric contents w/ an orogastric tube.
14. Administer intermediate-acting muscle relaxant if necessary.
15. After delivery, the nitrous oxide can be increased & the volatile decreased or discontinued to facilitate uterine involution. Administer opioids & consider benzodiazepines to prevent awareness.
16. At the end of the surgery, reverse muscle relaxants if necessary, remove orogastric tube & extubate when pt is awake.

- **Emergency (stable, urgent, stat) cesarean delivery w/ regional anesthesia de novo**
  1. Clarify maternal, fetal & obstetric issues w/ obstetrician as well as the urgency of the surgery.
  2. Anesthetic of choice is spinal for more stable conditions, general for stat conditions.
  3. Principles & procedure of spinal & general anesthesia as listed above
  4. Clarify fetal-well being on arrival to operative suite.
- **Emergency (stable, urgent, stat) cesarean delivery w/ pre-existing regional labor epidural**
  ➢ Clarify w/ obstetrician the fetal status & the urgency of delivery.

**Stable cesarean delivery**

1. Administer sodium citrate 30 mL PO & metoclopramide 10 mg IV.
2. Supplemental $O_2$ by face mask or nasal prongs
3. Check the level of existing blockade.
4. Administer supplemental IV fluids.
5. Monitor maternal BP & FHR as epidural is reinforced.
6. Medications:
   - Administer local anesthetic (lidocaine or bupivacaine) in 3- to 5-mL aliquots while monitoring maternal BP & ensuring fetal well-being.
   - 15–20 mL 2% lidocaine w/ epinephrine 1:200,000 w/ sodium bicarbonate (1 mEq/10 mL lidocaine)
   - 15–20 mL 0.5% bupivacaine (may add 0.05 mEq sodium barcarbonate/10 mL bupivacaine for a faster onset). Beware of potential for precipitation if more sodium bicarbonate is used.
   - Other adjuvants:
     - Fentanyl 50–100 mcg (to increase intensity & duration of the block)
     - Preservative-free morphine 3–5 mg (to prolong postoperative analgesia up to 24 hour).
7. Maintain uterine displacement & aggressively treat hypotension.
8. Transfer to the surgical suite, reapply standard monitors, maintain supplemental oxygen & uterine displacement & ensure fetal well-being.
9. Clarify the level of anesthesia prior to incision.

**Urgent cesarean delivery**

1. Steps 1 through 4 as above, but do not delay the administration of anesthesia to give the fluid bolus.
2. Monitor maternal BP & FHR as epidural is reinforced.
3. Medications
   - Administer local anesthetic (lidocaine or chloroprocaine) in 5-mL aliquots while monitoring maternal BP & ensuring fetal well-being.
   - 15–20 mL 2% lidocaine w/ epinephrine 1:200,000 w/ sodium bicarbonate (1 mEq/10 mL lidocaine)
   - 15–20 mL 3% 2-chloroprocaine w/ sodium bicarbonate (1 mEq/10 mL chloroprocaine)
   - Other adjuvants:

- Fentanyl 50–100 mcg (to increase intensity & duration of the block)
- Preservative-free morphine 3–5 mg (to prolong postop analgesia up to 24 hours)

4. Monitor BP & fetal status.
5. Transfer to OR; maintain uterine displacement.
6. Clarify the level of anesthesia prior to incision.

**Emergent (stat) cesarean delivery**

1. Administer sodium citrate 30 ml PO & metoclopramide 10 mg IV if readily available & call for someone to assist w/ the multiple tasks ahead.
2. Administer IV fluids & rapidly (over 2–3 minutes) reinforce epidural w/ 15–20 mL 3% 2-chloroprocaine in 5-mL aliquots as pt is being transferred to the surgical suite.
3. With rapid epidural bolus administration, maintain communication w/ the pt to ensure maternal well-being.
4. Maintain uterine displacement & re-apply supplemental oxygen on transfer to operative table (ensure that airway position is ideal).
5. Quickly apply standard monitors & assess level of anesthesia while FHR is being reassessed & the surgical prep is being done.
6. Can administer ketamine IV 10–20 mg (if not contraindicated) as the block is evolving, but be prepared for general anesthesia as a backup if regional block is insufficient.
7. If difficult intubation is suspected, consider awake intubation despite fetal depression.
8. If difficult intubation is not suspected, proceed w/ general anesthesia as outlined above.
9. Chloroprocaine can affect the onset & efficacy of epidural narcotics & amide local anesthetics. Redose the epidural after the delivery w/ 5–10 mL 2% lidocaine w/ epinephrine 1:200,000 & sodium bicarbonate (1 mEq/10 mL lidocaine) to maintain an established level of anesthesia.

**Intraoperative mgt**

- **Difficult airway**
  - ➢ See "*General Anesthesia and Management of the Difficult Airway*."
- **Shortness of breath**
  - ➢ Mild dyspnea is common after the administration of the regional anesthetic & is caused by the high level of anesthesia concomitant

w/ the decreased pulmonary reserve w/ a gravid uterus. This usually improves after delivery. Recheck the sensory level, oxygen saturation, BP & heart rate. Oxygen & reassurance will usually suffice.

- ➢ In rare cases of total spinal or high spinal w/ severe respiratory compromise, consider gentle assistance w/ a fitted mask in the awake pt.
- ➢ Intubate if the pt is obtunded or adequate oxygenation cannot be maintained w/ spontaneous breathing.

■ **Hypotension**

- ➢ Defined as 20–30% decrease in BP or a systolic BP < 100 mm Hg. Hypertensive pts may need a higher BP to maintain uterine perfusion.
- ➢ Common side effect of regional anesthesia due to sympathetic blockade; more common in women who are not in labor than in women who are in labor
- ➢ May lead to decreased uteroplacental perfusion w/ non-reassuring FHR & fetal acidosis; pt may report nausea
- ➢ Measure BP every 1–2 minutes & aggressively treat it until it stabilizes.
- ➢ Prevention
  - IV boluses of fluid
  - Left uterine displacement
  - Prophylactic administration of ephedrine 25–50 mg IM before spinal anesthesia or 5–10 mg IV immediately after intrathecal injection has a questionable efficacy.
- ➢ Treatment
  - Ensure adequate uterine displacement.
  - IV boluses of fluid
  - Administration of supplemental $O_2$
  - Ephedrine (5- to 10-mg boluses IV)
  - Phenylephrine in small doses (25–50 mcg IV) is considered safe in healthy parturients having elective cesarean delivery. It may be indicated in conditions in which ephedrine-induced tachycardia is less desirable or in cases of refractory hypotension. Vasoconstriction w/ uteroplacental insufficiency may be of concern w/ large doses.

■ **Nausea & vomiting**

- ➢ Nausea that occurs immediately after a regional anesthetic is a very sensitive indicator of hypotension & should be treated as above. Visceral pain or neuraxial opioids (especially long-acting) are other causes of nausea in the peripartum period.

- ➢ Maintain adequate BP.
- ➢ Other medications
  - Ondansetron IV 1–4 mg
  - Metoclopramide IV 10–20 mg
  - If nausea is a side effect of the opioids: naloxone IV 40–160 mcg or nalbuphine IV 2.5–5 mg
  - Transdermal scopolamine (after the delivery) has a prolonged antiemetic effect.

■ **Failed block/Inadequate anesthesia**

- ➢ The failed block (complete absence or patchy sensory or motor blockade) is more common w/ epidural than w/ spinal anesthesia. When it occurs, the regional anesthetic procedure can be repeated, but if time is important, general anesthesia is required.
- ➢ During the surgery, pt may experience some abdominal discomfort even w/ adequate regional anesthesia. Severe discomfort & pain are common w/ incomplete (patchy) epidural block & can be treated with:
  - Ketamine IV in 10- to 20-mg increments
  - Opioids after delivery of the fetus
  - Induction of general anesthesia if pain cannot be controlled

■ **Antibiotic prophylaxis**

- ➢ Substantially reduces the risk of postop fever, endometritis, wound & urinary tract infection
- ➢ Optimal time of administration (after the cord is clamped vs. preop) is not known; currently is administered immediately after the cord clamping.
- ➢ Cefazolin (1 or 2 g IV) is most commonly used.

■ **Uterine atony**

- ➢ Uterine involution & increasing uterine tone significantly reduce the bleeding after delivery.
- ➢ Intravenous oxytocin is routinely given once the fetus is born & the cord is clamped (10–20 units oxytocin in 1,000 mL crystalloid, administered IV at 40–80 mU/min). Side effects: maternal hypotension & tachycardia.
- ➢ Communicate w/ obstetrician to clarify uterine tone. Verbally confirm each drug & preferred route of administration.
- ➢ If uterine atony & bleeding persists:
  - Methylergonovine: 0.2 mg IM or in cases of life-threatening uterine hemorrhage 0.2 mg IV over 1 minute or longer. Side effects: severe hypertension, esp. w/ IV administration.

- 15-methyl prostaglandin F2-alpha: 250 mcg IM or intramyometrially. Side effects: tachycardia, hypertension, bronchoconstriction, fever & vomiting.

- **Shortness of breath after delivery**
  - ➢ May be the result of venous air embolism. Found to occur subclinically in >60% of pts undergoing cesarean delivery. It is associated w/ chest pain, oxygen desaturation, hypotension & even cardiac arrest in severe cases.
  - ➢ Mild symptoms can be treated w/ supplemental oxygen, IV fluid bolus, flooding the surgical field & slight reverse Trendelenburg (places the heart above the level of the surgical field).
  - ➢ In severe cases, adequate circulatory & ventilatory support is warranted. May consider rapid insertion of a central venous catheter to aspirate air or hyperbaric oxygen therapy if cerebral air embolism is suspected.
- **Shoulder pain**
  - ➢ Blood within the peritoneum may trigger transient referred shoulder pain. Reassurance will usually suffice. Ensure adequate hemostasis & stability of maternal vital signs. IV opioids may be given to relieve severe discomfort.
- **Shivering**
  - ➢ Shivering is uncontrollable & can be considerable & quite disturbing to the parturient.
  - ➢ Thermogenic & non-thermogenic factors may be involved (pain, labor, cold room, cold IV fluids, regional/general anesthesia).
  - ➢ Use warm IV fluids. A warm blanket & verbal reassurance may reduce anxiety & help pt feel better. IV opioids may be given after delivery for persistent shivering.

**CAVEATS & PEARLS**

Advantages of elective cesarean delivery over vaginal birth include:

- A known endpoint to the pregnancy, facilitating issues related to work, childcare, etc.
- Possible reduction in the risk of pelvic floor injury & its sequelae (incontinence, prolapse)
- Avoidance of post-term pregnancy & prevention of stillbirth
- Avoidance of potential intrapartum problems (hypoxia, meconium aspiration, cord prolapse, etc.) & of perinatal transmission of maternal infections (HIV, herpes simplex virus, hepatitis B)
- Reduction of fetal complications related to vaginal birth (cranial or clavicle fractures, brachial plexus injuries, etc.) & possible reduction in perinatal mortality

**Complications**

- **Surgery-related**
  - ➢ Infection
  - ➢ Bladder & bowel injury
  - ➢ Pulmonary thromboembolism
  - ➢ Venous air embolism
  - ➢ Amniotic fluid embolism
- **Anesthesia-related**
  - ➢ Inability to oxygenate & ventilate the pt
  - ➢ Pulmonary aspiration
  - ➢ Local anesthetic toxicity
  - ➢ Cardiovascular instability
  - ➢ Nausea/vomiting
  - ➢ Post-dural puncture headache
  - ➢ Postop respiratory depression
  - ➢ Epidural hematoma
  - ➢ Nerve damage

**CHECKLIST**

1. Close communication w/ obstetrician is essential for choosing the appropriate anesthetic technique.
2. Antepartum anesthesia consultation is ideal for the high-risk pt.
3. Pt education & constant reassurance are vital components of complete anesthetic care.
4. For all pts in the surgical suite, attention should be given to the position of the airway, regardless of the anesthetic technique used. This ensures that w/ an unforeseen need for general anesthesia, optimal positioning is already addressed.
5. All obstetric pts are considered "full stomach" regardless of the time of last oral intake & are at risk for pulmonary aspiration.
6. Pts who are at increased risk for complications associated w/ general anesthesia should have epidural catheters placed early in labor.
7. FHR abnormalities may resolve; frequent monitoring may obviate the need for rapid induction of general anesthesia.
8. Spinal or epidural anesthesia is preferred to general anesthesia because regional anesthesia is associated w/ much lower maternal mortality.
9. Hypotension is very common during regional anesthesia & should be promptly treated to prevent fetal distress.
10. The incidence of failed intubation in parturients is 10x higher than in the non-obstetric population.

11. When general anesthesia is induced, if the airway is difficult to secure, the safety of the mother takes priority over the delivery of the fetus.
12. Neuraxial administration of long-acting narcotics (e.g., morphine) places the pt at risk for late respiratory depression. Close monitoring for up to 24 hours after administration is required.

## ANESTHESIA FOR NON-OBSTETRIC SURGERY IN THE PREGNANT PATIENT

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### FUNDAMENTAL KNOWLEDGE

- Up to 2% of pregnant women in the U.S. undergo non-obstetric surgery.
- Surgical conditions common to the maternal age group include appendicitis, cholelithiasis, ovarian cysts & torsion, breast tumor, trauma & cervical incompetence.
- In caring for a pregnant pt, take into consideration both maternal safety & fetal well-being.

**Maternal Considerations**

Pregnancy affects virtually every organ system in the mother. The following are major physiologic changes having anesthetic implications.

**Respiratory changes**

- Decreased functional residual capacity by 20%
- Increased oxygen consumption
- Increased alveolar ventilation (normal PCO2 is approx. 32–35 mm Hg at term)
- Increased reported incidence of difficult intubations

**Cardiovascular changes**

- Increased blood volume
  - ➢ By 10% during the 1st trimester
  - ➢ By 30% during the 2nd trimester
  - ➢ By 45% during the 3rd trimester
- Increased cardiac output (CO)
  - ➢ By 35–40% at end of 1st trimester
  - ➢ By 50% during 2nd trimester
  - ➢ No further increase during 3rd trimester

- Decreased systemic & pulmonary vascular resistances
- Presence of hyperdynamic flow murmurs on physical exam
- ECG changes (eg, left axis deviation) due to enlargement & cephalad rotation of the heart
- Aortocaval compression by gravid uterus in supine position

**GI changes**
- Decreased lower esophageal sphincter tone
- Increased intragastric pressure
- Increased gastric acid secretion
- Increased risk for gastroesophageal reflux

**Hematologic changes**
- Physiologic anemia (baseline hemoglobin approx. 11–12 g/dL by mid-gestation)
- Hypercoagulability

**Neurologic changes**
- Decreased MAC for inhalational agents by 25–40%
- Decreased local anesthetic requirement by 30% for regional blockade

**Fetal Considerations**

With regard to the fetus, the anesthesiologist's primary goals are to avoid teratogens, recognize the risk of preterm labor & prevent fetal asphyxia.

**Teratogenicity**
- The fetus is most vulnerable to teratogens during the 1st trimester, when organogenesis takes place.
- W/ the exception of cocaine, no anesthetic agent has been identified as a human teratogen.
  - ➢ Risks of cocaine in human pregnancy
    - Fetal growth retardation
    - Placental abruption
    - Uterine rupture
  - ➢ Regarding benzodiazepines
    - Use became controversial after several studies in the 1970s reported an association between 1st- trimester diazepam use & oral cleft anomalies.
    - Subsequently, large cohort & case-control investigations have shown no increased risk w/ benzodiazepine therapy.
    - No evidence suggests that a single benzodiazepine dose given preoperatively to a pregnant pt would be harmful to the fetus.
  - ➢ Regarding nitrous oxide
    - Despite being a weak teratogen in rodents, nitrous oxide has not shown adverse effects in human pregnancy.

- A cautious approach would be to avoid nitrous oxide during the 1st trimester, to administer it at concentrations of 50% or less, and to limit its use during long operations.

- Drugs w/ long history of safe use during pregnancy include:
  - ➢ Thiopental
  - ➢ Opiates
  - ➢ Inhalational agents
  - ➢ Muscle relaxants
  - ➢ Local anesthetics

**Preterm labor & delivery**

- Epidemiologic studies of non-obstetric surgery during pregnancy report an increased incidence of abortion & preterm delivery.
- Lowest risk occurs when surgery takes place during 2nd trimester & does not involve uterine manipulation.
- Evidence does not support that any particular anesthetic agent or technique influences the risk of preterm labor.

**Fetal asphyxia**

Intra- & perioperative factors that can compromise fetal oxygenation include:

- Maternal hypotension
- Maternal hypoxia
- Maternal hypocarbia causing maternal alkalosis
  - ➢ Leads to decreased umbilical blood flow due to vasoconstriction
  - ➢ Leads to decreased release of oxygen to fetus at placenta due to leftward shift of maternal oxyhemoglobin dissociation curve
- Maternal hypercarbia leads to fetal respiratory acidosis &, if severe, fetal myocardial depression.
- Uterine contractions
- Intra-abdominal surgical manipulation & retraction

**STUDIES**

Pregnancy testing if diagnosis is uncertain

Perioperative fetal heart rate (FHR) monitoring

- For gestations <24 weeks, consider pre-op & post-op documentation of fetal heart tones.
- For gestations >24 weeks, consider intraoperative FHR monitoring.
  - ➢ Loss of beat-to-beat variability occurs w/ anesthetic meds.
  - ➢ Fetal decelerations, however, may indicate need to optimize intrauterine environment (eg, by ensuring adequate maternal BP & oxygenation, instituting tocolysis or repositioning surgical retraction).

- ➢ May not be feasible in urgent situations or during abdominal procedures
- ➢ Requires personnel skilled at FHR interpretation
- ➢ Has not been shown to improve fetal outcomes

Perioperative monitoring of uterine activity considered for gestations >24 weeks

## MANAGEMENT/INTERVENTIONS

**Pre-op mgt**

- Obtain obstetric consultation regarding:
  - ➢ Perioperative FHR monitoring (see "Studies")
  - ➢ Perioperative tocolysis
    - Use is controversial. Side effects of terbutaline & ritodrine include cardiac arrhythmias & pulmonary edema, & it is unclear whether prophylactic tocolysis improves outcome.
    - Some recommend monitoring of uterine contractions intraoperatively (when technically feasible) & post-op, w/ the institution of tocolytic therapy if appropriate.
- Discuss w/ pt her concerns regarding anesthetic risks to fetus & pregnancy.
- If no increased risk to mother, consider delaying surgery until 2nd trimester to minimize or eliminate fetal exposure to drugs during 1st trimester.
- Starting at 18–20 weeks gestation, consider pt as having a "full stomach" & administer aspiration prophylaxis [some combination of a non-particulate antacid (eg, sodium citrate 30 mL PO), metoclopramide (10 mg IV) & H2-receptor antagonist (eg, ranitidine 50 mg IV)].
- Administer anxiolytics (eg, midazolam 1–2 mg IV) & analgesics (eg, fentanyl 50–100 mcg IV) as necessary to treat maternal anxiety & pain.

**Intraoperative mgt**

- Primary goals are to maintain maternal BP & oxygenation.
- Standard monitors include ECG, BP, pulse oximetry, end-tidal CO2 & temp.
- Regional anesthesia
  - ➢ Consider regional if appropriate for maternal condition & surgical procedure.
  - ➢ Advantages
    - Minimizes drug transfer to fetus

  - Provides good post-op analgesia, reducing need for sedating meds & allowing for earlier mobilization
  - Does not cause changes in FHR variability
- ➢ Prevent hypotension w/ sufficient preload & lateral uterine displacement.
- ➢ Treat hypotension w/ ephedrine boluses (5–15 mg IV) as necessary.
- ➢ Reduce regional local anesthetic dose by approximately 30% compared w/ nonpregnant pts.

■ General anesthesia
- ➢ Adequate preoxygenation prior to induction is essential, since pregnant pts are prone to desaturate rapidly during periods of apnea.
- ➢ Starting at 18–20 weeks gestation, consider pregnant pts as having a "full stomach" & use rapid sequence induction w/ cricoid pressure.
- ➢ If difficult intubation is anticipated, consider awake fiberoptic intubation & have emergency airway supplies readily available.
- ➢ Reduce volatile anesthetic concentration by approx. 40% compared w/ nonpregnant pts.

■ No matter what anesthetic technique is used, remember that the greatest risk to the fetus is from maternal hypoxia, hypotension & hypoventilation, which can compromise fetal blood flow & oxygenation.
- ➢ Identify & treat common causes of hypoxia, which are the same as for nonpregnant pts (including laryngospasm, airway obstruction, improperly positioned endotracheal tube, hypoventilation).
- ➢ Correct maternal hypotension w/ appropriate treatments:
  - Lighten anesthesia for deep general anesthetic
  - Vasopressors for sympathectomy secondary to regional anesthetic
  - Fluid for hypovolemia
  - Lateral uterine displacement for aortocaval compression
- ➢ Maintain normal maternal PCO2 w/ adequate ventilation.

**Post-op mgt**

■ Continue monitoring of FHR & uterine activity.

■ Encourage early mobilization, since pregnant pts are at higher risk for thromboembolic complications.

**Special considerations**

- Laparoscopy
  - ➢ Once considered contraindicated during pregnancy but now becoming increasingly common as alternative to laparotomy in pregnant pts
  - ➢ Advantages
    - Smaller, less painful incisions, reducing need for sedating analgesics post-op
    - Earlier post-op mobilization
  - ➢ Pregnant pts are particularly susceptible to effects of CO2 insufflation:
    - Hypercarbia secondary to CO2 absorption, decreased pulmonary compliance & inadequate ventilation
    - Hypoxia from further decreases in FRC
    - Hypotension from increased intra-abdominal pressure & sudden position changes
  - ➢ Recommendations
    - Use open technique for trocar insertion.
    - Minimize insufflation pressures (no higher than 12–15 mm Hg).
    - Make position changes gradually.
    - Maintain lateral uterine displacement.
    - Adjust ventilation to maintain end-tidal CO2 around 32 mm Hg.
- Neurosurgery
  - ➢ Conditions such as aneurysm or AVM repair may be required during pregnancy.
  - ➢ FHR monitoring during induced hypotension may be useful in evaluating intrauterine environment.
  - ➢ Hypotensive agents such as esmolol, hydralazine, nitroprusside, nitroglycerin & inhalational agents have been used successfully in pregnant pts.
  - ➢ Remember that hyperventilation shifts the maternal oxyhemoglobin dissociation curve to the left, thereby decreasing oxygen release to the fetus. However, this maneuver may still be necessary for optimal care of the mother.
  - ➢ During endovascular procedures, the fetus should be shielded from radiation.

**CAVEATS & PEARLS**

- Defer elective surgery until after delivery, when maternal physiologic changes have returned to normal.

- For essential surgery, consider delaying until after 1st trimester, if possible, to minimize fetal exposure to drugs.
- For emergency surgery, proceed w/ optimal care for mother, taking into account physiologic changes of pregnancy.
- Above all, for the safety of the mother & fetus, avoid maternal hypotension & hypoxia.

**CHECKLIST**
- Obstetric consultation
- Aspiration prophylaxis & rapid sequence induction
- Lateral uterine displacement

## ANKYLOSING SPONDYLITIS

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**FUNDAMENTAL KNOWLEDGE**

See *"Autoimmune Disease."*
- Systemic rheumatic disorder characterized by chronic inflammatory arthropathy of both the axial skeleton & the large peripheral joints; progresses to fibrosis, ossification & ankylosis
- Typically there is progressive flexion & fusion of the spine & fixation of the rib cage.
- Prevalence of ankylosing spondylitis in women is 0.3% to 0.6%.
- Wide range of symptoms
  - ➢ Initial signs typically begin in the 20s and progress slowly over years.
  - ➢ Although cervical involvement can occur early, it more commonly presents after the child-bearing years (classic chin-on-chest deformity).
  - ➢ Extra-articular manifestations of ankylosing spondylitis are rare in pregnancy but can include cardiovascular complications of aortic insufficiency & conduction abnormalities (more commonly AV conduction delays leading to complete heart block).
  - ➢ Pulmonary complications usually occur late in the disease process & include pulmonary fibrosis & restrictive lung disease.
  - ➢ Systemic manifestations include fever, fatigue, weight loss, uveitis & anemia.
  - ➢ Neurologic complications include cauda equina syndrome, peripheral nerve lesions & vertebrobasilar insufficiency.

**STUDIES**

- Careful & directed history & physical exam; few pts will have manifestations that complicate anesthetic mgt during child-bearing years
  - ➢ Attention to history of TMJ dysfunction, cervical spine involvement & cardiopulmonary manifestations
- Lumbosacral radiographs

**MANAGEMENT/INTERVENTIONS**

- See "*Autoimmune Disease*."
- Pt may be taking aspirin or NSAIDs chronically. In most instances, the use of these meds is not a contraindication to regional anesthesia.
- Epidural or spinal anesthesia: Consider the paramedian approach, as ossification of the interspinal ligaments or osteophyte formation may make placement of the epidural challenging
- "Early" epidural placement is reasonable given anticipated difficulty w/ placement.
- Consider awake fiberoptic intubation if pt requires general anesthesia & has significant involvement of cervical spine, TMJ & cricoarytenoids.

**CAVEATS/PEARLS**

- Spinal rigidity & deformity as well as extra-articular manifestations are rare in young pts.
- Although women w/ ankylosing spondylitis often report chronic back pain, which itself is a risk factor for post-partum back pain, the overall risk of complications w/ regional anesthesia is minimal.
- Ossification of the interspinal ligaments or osteophyte formation may make placement of an epidural challenging.
- Consider awake fiberoptic intubation if pt requires general anesthesia & has significant involvement of cervical spine, TMJ & cricoarytenoids.

**CHECKLIST**

- Examine the airway. Be prepared for potential awake fiberoptic intubation.
- Establish an early epidural.

# ANKYLOSING SPONDYLITIS, BACK PROBLEMS

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## FUNDAMENTAL KNOWLEDGE

### Definition

- Chronic, progressive inflammatory disease involving the sacroiliac & synovial joints of the spine

### Epidemiology

- Prevalence: 0.3–0.6% of women
- Peak age of onset btwn 15 & 29 years of age
- Male: female 3:1

### Clinical manifestations

- Fibrosis, ossification & ankylosis occur, leading to the characteristic radiographic finding of "bamboo spine."
- Generally confined to hips & spine
- Extra-articular manifestations
  - ➢ Systemic
    - Fever
    - Weight loss
    - Fatigue
  - ➢ Cardiac
    - Aortitis
    - Aortic insufficiency
    - Conduction disorders
  - ➢ Pulmonary
    - Restrictive lung disease
    - Interstitial fibrosis
  - ➢ Neurologic
    - Peripheral neuropathies
    - Cauda equina syndrome
    - Vertebrobasilar insufficiency
  - ➢ Hematologic
    - Anemia
  - ➢ Urologic
    - Prostatitis
  - ➢ Ophthalmic
    - Uveitis

**Effect of pregnancy on AS**
- No significant change during pregnancy
- Active AS during conception increases the risk of postpartum flare of anterior uveitis, peripheral arthritis

**Effect on pregnancy & fetus**
- Higher incidence of C-section
- AS is reported to be the indication for C-section in 58% of these pts

## STUDIES

**History & Physical**
- Airway: examine for indicators of potentially difficult intubation
  - ➢ Limited neck range of motion, complete cervical fusion in flexed position
  - ➢ If disease has been present >16 years, 75% of pts have cervical ankylosis & high risk of cervical fractures
  - ➢ TMJ involvement may significantly limit mouth opening
  - ➢ Cricoarytenoid involvement increases risk of trauma to vocal cords
- Cardiac
  - ➢ Aortic insufficiency due to proximal aortitis
  - ➢ Mitral valve involvement, but usually only in pts w/ disease >15 years
- Pulmonary
  - ➢ Restrictive pattern due to involvement of costovertebral angle & fixation of thoracic cage
  - ➢ Pulmonary fibrosis
- Musculoskeletal: examine back & hip anatomy
- Neurologic: evaluate for peripheral neuropathies

**Imaging**
- Cervical spine radiograph
- Chest radiograph

**Other**
- ECG
- Pulmonary function testing in all pts

## MANAGEMENT/INTERVENTIONS

**Medical treatment**
- Aspirin/NSAIDs

- ➢ No evidence of teratogenicity
- ➢ No need for prophylactic cessation, but pregnancy should be closely monitored
- ➢ Recommend discontinuation during 3rd trimester because of:
  - Inhibitory effect on platelets & hemostasis
  - Increased risk of fetal CNS hemorrhage
  - Possible premature closure of ductus arteriosus
  - Possible compromised fetal renal perfusion & abnormal amniotic fluid dynamics
  - Possible factor in necrotizing enterocolitis (NEC)
- ➢ Sulfasalazine
  - No evidence of teratogenicity or difference in fetal outcome
- ➢ Corticosteroids
  - No demonstrated benefit in AS

**Anesthesia**

- General anesthesia
  - ➢ Awake fiberoptic intubation is recommended for pts w/ significant cervical spine disease
- Regional anesthesia
  - ➢ Establish early epidural due to potential difficulties w/ emergent induction of general anesthesia
  - ➢ Regional technique may be difficult due to ossification of interspinous ligaments & limitation of flexion of lumbar vertebrae

**CAVEATS & PEARLS**

- Anticipate potentially difficult airway due to possible involvement of cervical spine, TMJ & cricoarytenoids
- Take care to avoid high regional block, particularly in pts w/ significant restrictive lung disease
- May need to consider paramedian instead of midline approach due to ossification of interspinous ligaments & limitation of flexion of lumbar vertebrae

**CHECKLIST**

- Thorough airway exam
- Immediate availability of special airway equipment: laryngeal mask airway, fiberoptic laryngoscope, transtracheal jet ventilation, emergency cricothyrotomy kit
- Document presence of preexisting cauda equina syndrome & peripheral neuropathies prior to general or regional anesthesia
- Establish early epidural

# ANTEPARTUM ASSESSMENT

ANJALI KAIMAL, MD
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## FUNDAMENTAL KNOWLEDGE

- Perinatal mortality rate in U.S. has fallen steadily since 1965. According to the National Center for Health Statistics Definition, rate in 1997 was 7.3/1,000.
- To determine a strategy for antepartum monitoring, the risk of perinatal mortality must be determined for a specific clinical scenario.
- Antepartum deaths may be divided into 4 categories:
  1. Chronic asphyxia (multiple origins)
  2. Congenital malformations
  3. Superimposed complications of pregnancy (abruption, infection, Rh disease)
  4. Deaths of unexplained cause
- Antenatal surveillance is designed to identify those at risk for poor outcome.
- Although no current data are available describing etiologies of fetal deaths in the U.S., available studies suggest that 30% of antepartum deaths may be attributed to asphyxia, 30% to maternal complications, 15% to congenital malformations & chromosomal abnormalities & 5% to infections. At least 20% are unexplained.
- Anecdotal experience suggests that antepartum fetal assessment can affect the frequency & causes of antenatal fetal deaths. Unfortunately, few of the tests commonly used today have been subjected to prospective, randomized evaluation.
- Indications for antepartum fetal monitoring:
  1. High risk for uteroplacental insufficiency
     a. Prolonged pregnancy
     b. Diabetes
     c. Hypertension
     d. Previous stillbirth
     e. Suspected IUGR
     f. Advanced maternal age
     g. Multiple gestation w/ discordant growth
     h. Antiphospholipid antibody syndrome
  2. Suspected IUGR
  3. Decreased fetal activity

4. Oligohydramnios
5. Routine antepartum surveillance

## STUDIES

**Maternal assessment of fetal activity**

- During the third trimester, the fetus makes about 30 gross body movements per hour. Approx. 70–80% of these are perceptible by the mother.
- Periods of fetal activity are generally approximately 40 minutes, w/ quiet periods lasting 20 minutes.
- Fetal movement peaks btwn 9 pm & 1 am, when maternal glucose levels are falling.
- Presence of fetal movements is a reassuring sign of fetal health. Absence of fetal movement requires further evaluation before a determination of fetal status can be made.
- Maternal ability to perceive fetal activity may be influenced by many factors, including maternal habitus, placental location, amniotic fluid volume, maternal position & duration of fetal movements.
- There is a wide range of normal fetal activity. Generally, 10 movements in 2 hours is considered a reassuring result.

**Fetal heart rate assessment**

See "*Non-reassuring Fetal Heart Rate Tracing*" chapter.

- FHR assessment consists of identifying patterns indicative of fetal well-being & patterns that may be associated w/ adverse neonatal outcomes.
- Reassuring patterns:
  - ➢ Baseline FHR 120–160 bpm
  - ➢ Absence of FHR decelerations: Mild, transient episodes of hypoxia lead to bradycardia, either as variable or late decelerations depending on the etiology (cord compression or fetoplacental insufficiency)
  - ➢ Age-appropriate fetal heart accelerations: Advancing gestational age is associated w/ increased frequency & amplitude of fetal heart rate increases. Before 30 weeks' gestation, accelerations are typically 10 bpm for 10 seconds as opposed to 15 bpm for 15 seconds. Hypoxemia leads to a loss of the normal sympathetic response to movement & accelerations are absent.
  - ➢ Normal FHR variability: FHR variability results from sympathetic & parasympathetic nervous system input. Parasympathetic

influence increases w/ gestational age, so absence of variability is abnormal after 28 weeks.

- Nonreassuring patterns:
  - ➢ Late decelerations: In this case, the nadir of the deceleration occurs after the peak of the contraction. Mild late decelerations are a response to hypoxia. Repetitive late decelerations with absent variability are particularly concerning.
  - ➢ Variable decelerations: Intermittent mild or moderate variable decelerations w/ a quick return to baseline likely result from cord compression & are not worrisome. In contrast, deep, repetitive, severe variables can be associated w/ a fall in pH.
  - ➢ Absent variability: Loss of variability is thought to result from cerebral hypoxia & acidosis & signifies that the compensatory mechanisms to maintain adequate oxygenation to the brain have failed.
- Distress patterns:
  - ➢ Severe bradycardia: FHR <100 bpm for a prolonged time in the absence of drugs, heart block, or hypothermia
  - ➢ Tachycardia w/ diminished variability, esp. when associated w/ other nonreassuring patterns or in the absence of maternal fever

**Nonstress test**

- A nonstress test is performed by monitoring the FHR using an external monitor.
- A reassuring heart rate tracing has a baseline of 120–160 bpm, the absence of decelerations, presence of age-appropriate accelerations & normal variability.
- The presence of accelerations almost always indicates a non-acidotic fetus.
- Nonstress tests can be performed as soon as the fetal cardiac & neurologic systems are mature enough to demonstrate accelerations: as early as 26–28 weeks, more reliably at 32 weeks.
- A test is reactive is there are at least 2 accelerations of 15 bpm above the baseline lasting 15 seconds in a 20-minute period.
- The stillbirth rate after a reactive nonstress test is approximately 1.9/1,000. A reactive nonstress test or a negative contraction stress test has been associated with fetal survival for 1 week in more than 99% of cases.

**Contraction stress test or oxytocin challenge test**

- Uterine contractions cause a reduction in blood flow to the intervillous space. Therefore, a fetus w/ inadequate placental reserve will

demonstrate late decelerations due to hypoxia in response to frequent contractions.

- A contraction stress test should be performed on Labor & Delivery or an Antenatal Diagnostic Unit as there is a small but real risk of need for immediate delivery for fetal distress.
- FHR is monitored for 10–20 minutes to provide a baseline. If adequate contractions are not occurring spontaneously, they can be induced by nipple stimulation or infusion of oxytocin. Adequate contractions are defined as at least 3 moderate-intensity contractions in 10 minutes, w/ each contraction lasting 40–60 seconds.
- Fetal & uterine monitoring is continued until contractions return to baseline.
- Results:
  - ➢ Negative: Adequate contractions, no late decelerations
  - ➢ Positive: Late decelerations w/ >50% of the contractions, w/o excessive uterine activity
  - ➢ Suspicious: Inconsistent late decelerations
  - ➢ Hyperstimulation: Uterine contractions more frequently than every 2 minutes or lasting >90 seconds, or 5 contractions in 10 minutes. If no late decelerations are seen, test is negative.
  - ➢ Unsatisfactory: Quality of fetal heart tracing is inadequate for interpretation, or adequate contractions are not obtained.
- Contraindications to contraction stress test: conditions w/ increased risk for preterm labor, contraindications to contractions such as prior classic cesarean section or uterine surgery, placental abruption, placenta previa
- The stillbirth rate after a negative contraction stress test is 0.3/1,000.

**Biophysical profile**

- The BPP consists of assessment of 5 biophysical variables: fetal movement, fetal tone, fetal breathing, amniotic fluid volume & nonstress testing.
- Each component is given a score of 0 or 2 points depending on if criteria are met:
  - ➢ Fetal movement: 2 points if 2 or more discrete body or limb movements in 30 minutes
  - ➢ Fetal tone: 2 points if one or more episodes of extension of a fetal extremity or fetal spine with return to flexion
  - ➢ Fetal breathing: 2 points for one or more episodes of rhythmic breathing movements of at least 20 seconds within 30 minutes

- ➢ Amniotic fluid: 2 points if there is a single pocket of fluid measuring at least 2 cm vertically
- ➢ Nonstress test: 2 points if reactive

- Any given variable reflects the integration of CNS signals. The presence of normal biophysical activity virtually ensures functional integrity of the regulatory systems.
- Progressive loss of brain regulation leads to loss of reactivity, fetal breathing, fetal movement, fetal tone & amniotic fluid, in a reverse ontologic order.
- The modified BPP consists of amniotic fluid & nonstress test, with complete testing for those with an abnormal result.
- The risk of fetal demise within 1 week of a normal BPP is 0.8/1,000.
- A BPP of 8/10 or 10/10 is equally predictive as long as the points are not deducted for amniotic fluid volume.
- The BPP may be falsely depressed by corticosteroid administration; these changes are transient & usually return to normal by 48–96 hours after steroid administration. Neonatal outcome is not affected.

**Doppler ultrasonography**

- Umbilical artery flow can be examined w/ Doppler velocimetry.
- In fetuses w/ placental vasculopathy, impedance in the placental bed should be increased, leading to decreased diastolic flow.
- Results are described w/ a quotient of systolic to diastolic flow; <3.0 is considered normal after 28 weeks.
- This is generally used in combination with the BPP and nonstress test, esp. in pregnancies complicated by IUGR, preeclampsia or oligohydramnios.

## MANAGEMENT/INTERVENTIONS

**Maternal assessment of fetal activity**

- Maternal perception of decreased fetal activity should lead to a nonstress test.

**Nonstress test**

- Mgt of a nonreactive nonstress test depends on the clinical context & gestational age; either further fetal evaluation or delivery is indicated.
- The false-positive rate of a nonreactive nonstress test may be as high as 50%, so in the setting of a preterm gestation, additional testing is useful to prevent an unnecessary iatrogenic preterm delivery.
- Repetitive late decelerations or severe variable decelerations generally require prompt delivery by cesarean section.

**Contraction stress test**

- A positive contraction stress test indicates decreased fetal reserve & is associated w/ abnormal FHR patterns in labor.
- An equivocal or suspicious test is also associated w/ abnormal FHR patterns in labor. The presence of repetitive late decelerations generally requires prompt delivery, often by cesarean section.

**Biophysical profile**

- BPP mgt protocol:
  - ➢ BPP 10/10: Normal infant; repeat testing weekly as indicated
  - ➢ BPP 8/10: Normal infant as long as amniotic fluid volume is normal; repeat as indicated
  - ➢ BPP 6/10: Suspect chronic asphyxia, repeat testing in 4–6 hours, deliver for oligohydramnios
  - ➢ BPP 4/10: Suspect chronic asphyxia. If >36 weeks or mature fetal lungs, deliver. If <36 weeks, repeat in 24 hours; if repeat <4, deliver.
  - ➢ BPP 0–2/10: Strong suspicion of chronic asphyxia. Extend testing to 120 minutes. If persistent score <4, deliver if viable.

**Doppler ultrasonography**

- A high Doppler index, absent end-diastolic flow and reversed end-diastolic flow are associated w/ an increased likelihood of poor perinatal outcome.
- Depending on the gestational age, these findings should prompt delivery or more intensive fetal monitoring.

**CAVEATS/PEARLS**

- Antepartum monitoring is designed to identify those at risk for asphyxia, as well as those w/ congenital anomalies.
- Few of the tests used for antepartum assessment have been subjected to prospective, randomized evaluation.
- The fetus makes about 30 gross body movements an hour during the third trimester. Fetal movement peaks btwn 9 pm & 1 am.
- FHR assessment consists of identifying patterns of fetal well-being & patterns that may be associated w/ adverse neonatal outcomes.
- A baseline of 120–160, the absence of decelerations, the presence of accelerations & normal heart rate variability are reassuring.
- Persistent late decelerations, variable decelerations & absent variability are nonreassuring patterns.
- Severe bradycardia or tachycardia w/ absent variability indicates fetal distress.

- BPP consists of ultrasound assessment of fetal movement, fetal tone, fetal breathing & amniotic fluid in combination w/ a nonstress test. Scores of 8/10 or 10/10 require no further intervention.
- Umbilical artery flow can be a useful adjunct to other modes of testing.
- Mgt of abnormal test results depends on the clinical scenario & gestational age.

**CHECKLIST**
- Identify fetuses at risk for asphyxia.
- Initiate appropriate testing.
- Obtain additional testing as necessary to confirm fetal well-being.

Proceed w/ delivery or plan for repeat testing based on gestational age, test results & clinical scenario.

## ANTIPHOSPHOLIPID SYNDROME

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**FUNDAMENTAL KNOWLEDGE**

**Definition**
- Antiphospholipid syndrome (APS) is a hypercoagulable state defined by recurrent miscarriage &/or late pregnancy loss in the presence of antiphospholipid antibodies (aPLs)
- Primary APS occurs in pts **without** SLE or other connective tissue disease
- Secondary APS occurs in pts w/ SLE or other connective tissue disease

**Epidemiology**
- Prevalence is unknown because the accepted diagnostic criteria are new & subject to frequent revision & debate

**Pathophysiology**
- Two most common aPLs associated w/ recurrent pregnancy loss, thromboembolism, thrombocytopenia
  - ➢ Lupus anticoagulant (LA): identified by abnormal coagulation assay & confirmed w/ a mixing test
    - Misnomer resulting from its prolongation of phospholipid-dependent clotting studies in vitro

- No true anticoagulant activity in vivo; actually promotes thrombosis
- Present in 34% of pts w/ SLE
- Only 35% of pts w/ LA have SLE

➢ Anticardiolipin (aCL): measured by ELISA
- Present in 44% of pts w/ SLE
- Antibodies may be induced in HIV-1-positive pts, but this does not appear to lead to APS

**Clinical manifestations**
- Most serious complication is thrombosis
  - ➢ 70% venous
  - ➢ Mostly in lower extremities
- Placental pathology: thrombosis of placental & decidual vessels, infarction
- Pulmonary embolism, myocardial infarction, TIA/CVA, amaurosis fugax may also occur
- Autoimmune thrombocytopenia & anemia linked to APS
- Renal insufficiency
- Pulmonary hypertension
- Diagnostic clinical criteria for APS
  1. One or more unexplained deaths of morphologically normal fetus (documented by ultrasound or direct fetal exam) at or beyond gestational age of 10 weeks
  2. One or more premature births of morphologically normal neonate at or before gestational age of 34 weeks due to severe pre-eclampsia or eclampsia, or severe placental insufficiency
  3. Three or more unexplained consecutive spontaneous abortions before the gestational age of 10 weeks w/ the exclusion of maternal anatomic/hormonal & parental chromosomal causes

**Effect on pregnancy & fetus**
- Increased risk of APS-related pregnancy loss, preeclampsia 16–50% & IUGR attributed to defective embryonic implantation

## STUDIES

**History & Physical**
- Evaluate for signs & symptoms of thromboembolism & pulmonary hypertension

**Other**
- Antiphospholipid antibodies
- Coagulation studies & platelet count

- Exclusion of anatomic, hormonal, chromosomal abnormalities as factors in recurrent pregnancy loss

## MANAGEMENT/INTERVENTIONS

**Medical treatment: focus is on anticoagulation**

- Aspirin
  - ➢ No evidence of teratogenicity
  - ➢ Should be started as soon as pregnancy test is positive
  - ➢ Discontinue at 36th week of gestation to avoid:
    - Premature closure of ductus arteriosus
    - Inhibitory effect on platelets & hemostasis
    - Increased risk of fetal CNS hemorrhage
- Heparin or LMWH
  - ➢ No evidence of teratogenicity
  - ➢ Should be started as soon as intrauterine pregnancy confirmed
  - ➢ Times to peak & half-life are shortened in pregnancy, so BID dosing is recommended
  - ➢ Chronic unfractionated heparin therapy is associated w/ maternal osteopenia/osteoporosis, but LMWH is associated w/ significantly less risk of bone loss
- Corticosteroids
  - ➢ No evidence of teratogenicity
  - ➢ Inactivation by placental 11-beta-OH-dehydrogenase results in low fetal levels of active drug
  - ➢ May precipitate gestational diabetes mellitus & hypertension
- Longer-term adverse effects
  - ➢ Osteoporosis
  - ➢ GI ulceration
  - ➢ Impaired immunity
  - ➢ Adrenal suppression
- IVIG & hydroxychloroquine
  - ➢ Under investigation

**Anesthesia**

- Hold morning dose of heparin when induction of labor or C-section is planned
- Hold anticoagulants & check for normalization of coagulation studies prior to regional technique
- Ideally, wait 24 hours since last dose of LMWH prior to regional technique

- If regional technique is requested in a pt still taking LMWH, may perform Hep test (anti-factor Xa activity)
  - ➢ Proceed if normal
  - ➢ If abnormal, wait until normalization; may prescribe pt-controlled anesthesia w/ fentanyl +/− ketamine in the meantime
- Recommend frequent neurologic evaluations for the first 6–12 hours after a regional technique for early detection of epidural hematoma formation

### CAVEATS & PEARLS

- Coagulopathy &/or thrombocytopenia may contraindicate regional anesthetic until corrected
- Increased risk for thromboembolic phenomena: DVT, pulmonary embolism, myocardial infarction, TIA/CVA, placental thrombosis
- Lupus anticoagulant (LA) is a misnomer because it has no true anticoagulant activity in vivo & actually promotes thrombosis

### CHECKLIST

- Hold anticoagulants, check platelet count & check for normalization of coagulopathy prior to administering regional anesthetic
- Hold morning dose of heparin when induction of labor or C-section is planned
- Frequent neurologic evaluations for the first 6–12 hours after a regional technique are recommended for early detection of epidural hematoma formation

## AORTIC REGURGITATION

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### FUNDAMENTAL KNOWLEDGE

- Multiple etiologies exist for aortic regurgitation (AR).
- Disease processes may lead to the destruction of the aortic valve. The etiology of AR is rheumatic heart disease in 75% of cases. Women w/ AR secondary to rheumatic heart disease often have a diseased mitral valve as well. Isolated AR is more often than not secondary to a process other than rheumatic heart disease. Infective endocarditis is another process that may lead to the destruction of the aortic valve w/ AR.

- Congenital deformities of the aortic valve may result in AR. Pts w/ a ventricular septal defect can develop a prolapse of an aortic cusp (15% of pts w/ a ventricular septal defect). Congenital fenestrations may be present. A bicuspid aortic valve may have isolated AR. Bicuspid aortic valve is highly associated w/ aortic root abnormalities leading to aortic root & annular dilatation.
- Several different processes may lead to widening of the aortic annulus, resulting in AR. Severe hypertension may widen the aortic annulus, leading to progressive AR. Syphilis, via cellular infiltration & scarring of the media of the thoracic aorta, may lead to aortic dilation, aortic annulus dilation & AR. Cystic medial necrosis may lead to widening of the ascending aorta & aortic annulus, which in turn leads to AR. Pts w/ Marfan syndrome may develop widened aortic annulus w/ AR. Idiopathic dilation of the aorta widens the aortic annulus & results in AR. In pts w/ aortic dissection, retrograde dissection may involve the aortic annulus & cause AR.
- Regurgitation leads to L ventricular dilatation & L ventricular hypertrophy, which eventually may promote L ventricular systolic dysfunction, diastolic dysfunction & myocardial ischemia. When the aortic valve leaflets fail to oppose due to leaflet, annulus or cusp deformity, blood regurgitates into the L ventricle from the aorta during diastole. The L ventricle dilates to accommodate the extra volume from regurgitation. As the L ventricle dilates, wall stress increases. Over time, the L ventricle hypertrophies to compensate for the increased wall stress (afterload). As the L ventricle hypertrophies, it becomes less compliant. With decreased compliance comes increased L ventricular end-diastolic pressures for a given L ventricular volume. L ventricular end-diastolic pressure may reach upwards of 40 mmHg. Increased L ventricular end-diastolic pressures may lead to subendocardial ischemia. An increased L ventricular end-diastolic pressure may promote increased L atrial pressure & its concomitant issues.
- Initially, a competent mitral valve protects the pt from pulmonary manifestations of increased ventricular end-diastolic volume & pressure. Eventually, L ventricular pressure may exceed L atrial pressure toward the end of diastole, causing premature closing of the mitral valve or diastolic mitral regurgitation. Increased L atrial pressure may lead to pulmonary edema & atrial arrhythmias.
- The decreased systemic vascular resistance (SVR) that accompanies pregnancy unloads the heart & may improve ventricular function in the setting of AR.

- There may come a time when the heart cannot sufficiently balance increased L ventricular volume, L ventricular pressure & L ventricular hypertrophy to yield the necessary cardiac output. The heart rate will increase to augment cardiac output. The increased heart rate may lead to myocardial ischemia. Increased myocardial oxygen demand accompanies an increased heart rate. At baseline, there is decreased coronary flow during diastole in the pt w/ AR, for the pressure gradient falls quickly: there is less time w/ a sufficient pressure head to enable coronary flow. An increased heart rate means less time is spent in diastole. Heart failure may ensue. This is most likely to occur in the parturient during labor & delivery.
- In the healthy parturient, cardiac output increases up to 60% from prepregnancy values during labor & delivery. Immediately after delivery, intravascular volume increases dramatically as the uterus contracts; the heart may be unable to accommodate the large increase in blood volume. Immediately after delivery, compression of the inferior vena cava decreases, which facilitates venous return.

**STUDIES**

- Several sources of information or studies may be used to arrive at a diagnosis of AR.
  - ➢ The history may provide evidence of cardiac dysfunction resulting from AR. The history may reveal symptoms consistent w/ L ventricular overload & dysfunction: exertional dyspnea & other symptoms of diminished cardiac reserve; orthopnea; paroxysmal nocturnal dyspnea; pounding sensation in the head and/or chest, especially when supine; diaphoresis; peripheral edema; congestive hepatomegaly; ascites.
    - Symptoms of L ventricular failure are more common than symptoms of myocardial ischemia, but these symptoms may occur & should be elicited: chest pain, anginal symptoms, excessive pounding of the heart on the chest wall, coronary insufficiency.
    - A history of symptoms consistent w/ L atrial volume overload causing pulmonary edema suggests that a pt may have involvement of the L atrium due to equalization of pressures or concomitant mitral valve disease. Pt may report dyspnea or orthopnea. Pt may report hemoptysis (occurs secondary to rupture of dilated bronchial veins), blood-tinged sputum secondary to pulmonary edema. Pt may report chest pain or palpitations.

- ➢ Auscultation may reveal evidence of AR.
  - Three murmurs are associated w/ AR:
    - High-pitched, blowing decrescendo murmur that occurs during diastole due to regurgitant blood through the aortic valve into the L ventricle
    - Systolic murmur secondary to increased forward flow across the aortic valve
    - Austin Flint murmur: This is a low-pitched murmur that occurs during diastole. The murmur occurs secondary to the incomplete opening of the mitral leaflets due to elevated L ventricular filling pressures.
  - An $S_3$ gallop may be present if pt has developed heart failure.
- ➢ Palpation may reveal evidence of AR.
  - A widened pulse pressure may be detected via palpation of the pulse. The pulse is bounding w/ a rapid upstroke & quick collapse ("water hammer pulse"). The cardiac impulse is hyperdynamic. A diastolic thrill may be palpated at the L sternal border, third intercostal space.
- ➢ EKG may reveal evidence of a dilated L ventricle diseased by AR: L axis deviation, increased QRS voltage. Voltage criteria for L ventricular hypertrophy include R wave in AVL >12 mm; R wave in I >15 mm; sum of the S wave in $V_1$ or $V_2$ plus R wave in $V_5$ or $V_6$ equal to or greater than 35 mm.
- ➢ EKG may reveal evidence of a dilated L atrium if disease is far progressed.
  - Atrial enlargement, characterized by:
    - A wide P wave in lead II that lasts >0.12 seconds
    - Deeply inverted terminal component in lead $V_1$
  - Atrial fib
  - R ventricular hypertrophy, characterized by:
    - Tall R waves in $V_1$ though $V_3$
    - Deep S waves in leads I, L, $V_5$ & $V_6$
    - R axis deviation
- ➢ Chest x-ray may reveal evidence of AR: cardiomegaly, L atrial enlargement, pulmonary edema.
- ➢ Echocardiography is the best study for evaluating & characterizing AR. The lesion can be examined in real-time to determine the mechanism. Leaflet morphology can be evaluated. Severity of aortic insufficiency can be assessed. The valve area can be calculated if mixed aortic stenosis & AR exist. A gradient can be estimated for prognostication if mixed aortic stenosis & AR exist.

- Echocardiography is indicated when a diastolic murmur, a continuous murmur or a loud systolic murmur is auscultated; when the murmur is associated w/ symptoms or when the murmur is associated w/ an abnormal EKG.

- Few sources of information or studies may be used to offer a prognosis for how the pregnant pt w/ AR will fare.
  - ➢ Pts are considered high risk when the L ventricular end-systolic diameter is >50 mm or the ejection fraction is <50%.
  - ➢ According to the American College of Cardiology/American Heart Association guidelines, pts predicted to have a high risk of abnormal outcomes are those w/ an abnormal functional capacity & those w/ L ventricular dysfunction.
  - ➢ The American College of Cardiology & the American Heart Association have classified maternal & fetal risk during pregnancy based on the type of valvular abnormality in conjunction w/ the New York Heart Association functional class.
    - NYHA functional classification for congestive heart failure:
      - Class I: Pts have no limitation of activities, no symptoms from ordinary activities.
      - Class II: Pts have slight, mild limitation of activity; pts are comfortable w/ mild exertion.
      - Class III: Pts have marked limitation of activity; they are comfortable only at rest.
      - Class IV: Any physical activity brings discomfort; symptoms occur at rest.
    - Associated w/ low maternal & fetal risk: AR in the setting of NYHA class I or II & normal L ventricular function
    - Associated w/ high maternal & fetal risk: AR w/ NYHA class III or IV symptoms; AR in the setting of pulmonary hypertension; AR in the setting of depressed L ventricular function (ejection fraction <40%)

## MANAGEMENT/INTERVENTIONS

### Medical mgt

- Time course for evaluation by a cardiologist
  - ➢ During pregnancy, women w/ valvular heart disease should be evaluated once each trimester for presence or worsening of symptoms & if there is a change in symptoms to assess for any deterioration in maternal cardiac status.
  - ➢ The American College of Cardiology & the American Heart Association recommend that women w/ high-risk cardiac lesions undergo full evaluation prior to & during pregnancy.

- During the antepartum period, treatment goals for the pt w/ mild or moderate symptoms focus on:
  - ➢ Treatment of volume overload to avoid or treat pulmonary edema:
    - Diuretic therapy w/ a loop or thiazide diuretic
    - Titrate diuretics to avoid hypovolemia & uteroplacental hypoperfusion.
    - Avoid excessive salt intake, which will increase intravascular volume.
  - ➢ Treat L ventricular systolic dysfunction:
    - Digoxin to increase contractility
    - Afterload reduction w/ vasodilators:
      - Hydralazine dilates arterioles, but not veins; associated reflex tachycardia is tolerated well by the pt w/ AR.
      - Avoid ACE inhibitors (unsafe for pregnancy; associated w/ IUGR, premature deliveries, neonatal renal failure, anemia, limb defects, patent ductus arteriosus).
    - Avoid bradycardia:
      - An increase in diastolic time will increase L ventricular filling & thus the regurgitant volume per cardiac cycle. Atrial fib should be aggressively treated, as the loss of the atrial contraction will decrease atrial emptying & may lead to pulmonary congestion. Atrial fib w/ hemodynamic instability and/or pulmonary edema requires cardioversion. Atrial fib w/o hemodynamic instability should be rate-controlled pharmacologically.
        - Beta blockers are safe for use in pregnancy.
        - Digoxin depresses atrioventricular conduction & thus an excessively high ventricular rate & is safe for use in pregnancy.
        - Calcium channel blockers block activated & inactivated calcium channels in the sinoatrial & atrioventricular node. Peripheral vasodilation is a side effect. They are safe for use in pregnancy.
        - Cardioversion often is performed when pharmacologic therapy fails to control the ventricular response to atrial fib.
      - Medications that have a suppressive effect on atrial fib may have been prescribed to the parturient in the antepartum period.
        - Procainamide, a sodium channel blocking drug (class I), may cause direct myocardial depression & may reduce

peripheral vascular resistance. It is safe for use in pregnancy.
- Quinidine, a sodium channel blocking drug (class I), depresses the pacemaker rate, depresses conduction & excitability & has alpha receptor-blocking properties & may cause vasodilatation & a reflex increase in sinoatrial node rate. It is safe for use in pregnancy.
- Amiodarone, a blocker of sodium, potassium & calcium channels, suppresses supraventricular & ventricular arrhythmias. It may cause peripheral vascular dilation through alpha-blocking effect & may cause bradycardia. It is *not* listed as safe for use in pregnancy due to the risk of hypothyroidism in the fetus as well as IUGR & prematurity.

- Pts in atrial fibrillation will need to be anticoagulated.
- When the onset of atrial fib cannot be determined, the pt should not be cardioverted until she has been anticoagulated for a sufficient period of time as determined by her cardiologist (unless she is hemodynamically unstable).

- Surgery during pregnancy usually is indicated for pts w/ refractory NYHA class III or IV symptoms.

**Obstetric mgt of the pt w/ AR**

- AR is often tolerated well during pregnancy; labor & delivery often proceed w/o complication.
- C-section is performed only when there are obstetrical indications for such a mode of delivery.
- The American College of Cardiology/American Heart Association Guidelines for the mgt of pts w/ valvular heart disease recommend that antibiotic prophylaxis be provided for the pt w/ aortic regurgitation to prevent endocarditis.
- Recommendations are that antibiotic prophylaxis be provided for dental procedures & certain respiratory, GI & GU procedures, as listed in the ACC/AHA guidelines. Antibiotic prophylaxis for endocarditis is not recommended for routine vaginal delivery or C-section.
- Anesthetic mgt should avoid conditions that exacerbate AR & facilitate conditions that improve the symptoms of AR. Maintain a normal to increased heart rate to decrease diastolic time. This will decrease the time during diastole for regurgitation into the L ventricle. Administer agents that have a favorable side effect profile & avoid agents with an unfavorable side effect profile in relation to heart rate (ephedrine instead of phenylephrine; meperidine may be the preferred opioid; although ketamine is not associated w/ bradycardia, it

is associated w/ increased SVR & as such would not offer a favorable side effect profile).

- Aggressively treat atrial fib (maintain normal sinus rhythm). Cardioversion is always indicated if new-onset atrial fib causes hemodynamic instability. Treat rapid ventricular rate: IV beta blockers, IV calcium channel blockers, IV (careful: narrow therapeutic window!)

➢ Maintain cardiac output. Maintain normal to increased heart rate to avoid L ventricular volume overload. Maintain venous return. Avoid aortocaval compression; maintain L uterine displacement. Avoid insufficient intravascular volume while avoiding volume overload. If a central venous catheter is in situ, optimize the central venous pressure to allow for sufficient systolic BP while avoiding pulmonary edema. If a pulmonary artery catheter is in situ, optimize pulmonary capillary wedge pressure to maximize cardiac output while avoiding pulmonary edema. Avoid an increase in pulmonary vascular resistance: avoid pain, hypoxia, hypercarbia, acidosis. Boluses of oxytocin, methylergonovine & 15 methyl prostaglandin F2-alpha may result in increased pulmonary vascular resistance.

- Maintain a decreased afterload. An increase in SVR may increase the regurgitant fraction & may decrease cardiac output. The decrease in SVR associated w/ pregnancy benefits pts w/ this lesion. Avoid uterine compression of the aorta, as this may increase SVR.
- Neuraxial anesthesia will provide a decrease in SVR. Neuraxial anesthesia is well tolerated by pts w/ AR. Early administration during labor prevents a pain-mediated increase in SVR. Maintain (but do not increase) SVR during the onset of sympathetic blockade w/ epidural administration of local anesthetic. General anesthesia w/ a volatile agent will provide a decrease in SVR & is well tolerated by the parturient w/ AR. *Isoflurane* & *sevoflurane* decrease SVR the most of the volatile agents. Halothane produces myocardial depression much more than a decrease in SVR & is a poor choice if other volatile agents are available.
  - Pharmacologic agents may be necessary to decrease SVR: sodium nitroprusside; hydralazine (dilates arterioles & not veins; reflex increase in heart rate is a favorable side effect for the parturient w/ AR); nitroglycerin (use w/ caution as this agent will cause uterine relaxation). Avoid ACE inhibitors

until after delivery as they are associated w/ several adverse fetal effects.

**CAVEATS/PEARLS**

- AR is usually well tolerated by the parturient. The increased heart rate & the increased intravascular volume that accompany pregnancy are favorable to the pt w/ AR. The decreased SVR that accompanies pregnancy is also favorable to the pt w/ AR.
- Early administration of epidural analgesia during labor prevents a pain-mediated increase in SVR, which may exacerbate AR.
- For a general anesthetic, halothane should not be the first choice of volatile agent as it is a direct myocardial depressant & it sensitizes the myocardium to catecholamines.
- Hydralazine is an excellent choice for afterload reduction, as it is associated w/ an increase in heart rate, which may benefit the pt w/ AR.
- Avoid ACE inhibitors for afterload reduction until after delivery as they are associated w/ several adverse fetal effects.
- Nitroglycerin will provide afterload reduction, but it may cause deleterious uterine relaxation.

**CHECKLIST**

- Ascertain if the pt has had symptoms of pulmonary congestion or heart failure during pregnancy.
- Review meds that the pt may be taking for rate control, atrial fib suppressive therapy & symptoms of heart failure.
- Obtain echocardiographic data to characterize severity of AR if there has been a significant change in the pt's symptoms.
- Pt is high risk if she has AR w/ NYHA class III or IV symptoms; aortic valve disease resulting in pulmonary hypertension; or aortic valve disease w/ L ventricular systolic dysfunction.
- Women who are at high risk should have invasive monitoring during labor & delivery, as this is the period w/ the greatest magnitude in change w/ regard to hemodynamics.
- If GU procedures are to be performed other than routine vaginal delivery or C-section, administer prophylactic antibiotics to prevent endocarditis from bacteremia.
- C-section is performed only when there are obstetrical indications for such a mode of delivery.
- During anesthetic mgt, maintain normal to increased heart rate to avoid L ventricular volume overload. Maintain venous return &

decreased afterload. Consider epidural labor analgesia for vaginal delivery. Consider epidural anesthesia for cesarean delivery. Consider isoflurane, sevoflurane & desflurane rather than halothane for general anesthesia. Consider vasodilators as necessary, but avoid ACE inhibitors.

## AORTIC STENOSIS

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### FUNDAMENTAL KNOWLEDGE

- Aortic stenosis (AS) may be congenital or rheumatic in origin. In pregnant women, the most common cause of AS is a congenital bicuspid aortic valve. Progressive fibrosis & calcification of the congenitally abnormal valve renders it stenotic.
- Rheumatic heart disease is another cause of AS in women of childbearing age. Disease occurs after fusion of the commissures; there is scarring & eventual calcification of the cusps.
- Severe AS is poorly tolerated during pregnancy.
- A stenotic aortic valve due to anatomic distortion alters the normal physiology of the parturient. Typically, hemodynamics in the pt w/ AS are altered when the aortic valve area is reduced from one-third to one-fourth its original area. The normal aortic valve area is 3.0–4.0 $cm^2$. Physiologic perturbations often are apparent when the valve area is $<1.0$ $cm^2$. A reduced valve area increases the pressure gradient between the L ventricle & the ascending aorta. A significantly large pressure gradient increase for a given increase in flow occurs when the valve area is 0.75 $cm^2$ or less. Severe AS is associated w/ a mean pressure gradient of 50 mmHg or greater.
  - ➢ The L ventricle must work harder to pump blood through a stenotic aortic valve. Increased pressure gradients across the stenotic valve present the L ventricle w/ an increased wall stress, or afterload. The L ventricle will hypertrophy to compensate for an increased afterload. Hypertrophy is often adequate to preserve ventricular performance as measured by the ejection fraction.
    - As disease progresses, the stroke volume will eventually become fixed. The degree of hypertrophy will be insufficient to