## Obstetric Review

- **Physiologic changes of pregnancy**
  
  - **Cardiovascular**

<table>
<thead>
<tr>
<th>parameter</th>
<th>effect</th>
<th>causes/effects</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>cardiac output</strong></td>
<td>↑ particular to stage of pregnancy or labor: greatest increase (60-80%) occurs with umbilical cord clamping</td>
<td>↑HR (10-15 bpm by mid 2nd trimester)</td>
</tr>
<tr>
<td>10% 1st trimester</td>
<td>↑ 40% 3rd trimester</td>
<td>↑SV</td>
</tr>
<tr>
<td></td>
<td></td>
<td>↓SVR</td>
</tr>
<tr>
<td><strong>blood volume</strong></td>
<td>↑ RBC 15-20%</td>
<td>*disproportionate rise in plasma volume to RBC mass; causes “dilutional anemia”</td>
</tr>
<tr>
<td></td>
<td>↑ plasma volume 45-50%</td>
<td>Increased H/H may signify low volume state</td>
</tr>
<tr>
<td></td>
<td>↑ total volume by 1500 ml</td>
<td></td>
</tr>
<tr>
<td></td>
<td>↓Hgb/HCT (11/33%)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>↑ vessel engorgement</td>
<td></td>
</tr>
<tr>
<td><strong>coagulation</strong></td>
<td>↑ platelet size/↓ count</td>
<td>parturient considered hypercoaguable</td>
</tr>
<tr>
<td></td>
<td>↑ coagulation factors, especially fibrinogen</td>
<td>PIH may result in hypocoaguable state</td>
</tr>
</tbody>
</table>

- **Respiratory**

<table>
<thead>
<tr>
<th>parameter</th>
<th>effect</th>
<th>causative factors</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>minute ventilation</strong></td>
<td>↑50%</td>
<td>↑RR: progesterone causes increased sensitivity to CO₂; metabolic rate increased</td>
</tr>
<tr>
<td><strong>V&lt;sub&gt;E&lt;/sub&gt;</strong></td>
<td>↓ paCO₂ (from 40 to 30)</td>
<td>↑V&lt;sub&gt;T&lt;/sub&gt; (1&lt;sup&gt;st&lt;/sup&gt; causative factor)</td>
</tr>
<tr>
<td></td>
<td>pH remains unchanged due to increased renal excretion of HCO₃ ions</td>
<td></td>
</tr>
<tr>
<td><strong>O&lt;sub&gt;2&lt;/sub&gt; consumption</strong></td>
<td>↑ 20%</td>
<td>↑ metabolic rate</td>
</tr>
<tr>
<td><strong>O&lt;sub&gt;2&lt;/sub&gt; delivery</strong></td>
<td>↑</td>
<td>↑ 2,3 DPG; facilitates unloading of O&lt;sub&gt;2&lt;/sub&gt; to fetus</td>
</tr>
<tr>
<td><strong>FRC</strong></td>
<td>↓ 20% (largest Δ of all)</td>
<td>abdominal distention from gravid uterus; pushes diaphragm cephalad</td>
</tr>
<tr>
<td><strong>diameter of thorax</strong></td>
<td>(VC, TLC, and CV remain unchanged)</td>
<td></td>
</tr>
<tr>
<td><strong>airway resistance</strong></td>
<td>↓</td>
<td>progesterone causes bronchiolar and pulmonary vessel dilation</td>
</tr>
<tr>
<td><strong>pulmonary vascular resistance</strong></td>
<td>↓</td>
<td></td>
</tr>
</tbody>
</table>
Other systems

<table>
<thead>
<tr>
<th>system</th>
<th>effect</th>
<th>cause/effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>neurologic</td>
<td>↑ sedation, ↓ in MAC</td>
<td>effect of progesterone</td>
</tr>
<tr>
<td>renal</td>
<td>↑ RBF, ↑ GFR</td>
<td>↓ in BUN and creatinine by 50%</td>
</tr>
<tr>
<td>gastrointestinal</td>
<td>pyloric displacement incompetent GE sphincter (from as early as 12 weeks) gastric emptying delayed only during pain of labor</td>
<td>from gravid uterus progesterone **parturient highly prone to regurgitation/aspiration</td>
</tr>
<tr>
<td>hepatic</td>
<td>↓ plasma cholinesterase levels, ↑ hepatic enzymes, ↓ albumin</td>
<td>SCH duration clinically unchanged 2° large initial V₆ of drug</td>
</tr>
</tbody>
</table>

Determinants of uteroplacental flow

- Uterine vessels have α and β receptors
- Placental vessels are devoid of receptors
- Uterine perfusion (therefore placental perfusion) is directly proportional to maternal MAP and inversely proportional to uterine vascular resistance
  - uterine blood flow = \( \text{uterine arterial pressure} - \text{uterine venous pressure} \) \( / \text{uterine vascular resistance} \)
- At term, UBF is 500-700 ml/min or ~ 10% of CO

Factors which interfere with UBF

- Maternal hypotension
  - From hemorrhage, supine hypotensive syndrome, GA or regional
    - Supine hypotensive syndrome
      - Occurs from 18-20th week of gestation on
        - Compression of the vena cava (and eventually aorta) causes hypotension, ↓ placental perfusion from ↓ venous return and CO
- S/S: lightheadedness, n/v, diaphoresis
- Primary tx: institution of left uterine displacement (LUD) by 10-15 cm elevation of the R hip; pressors and fluid
  - Increased catecholamines
    - From pain of labor, stress, PIH
  - Seizures
  - Uterine tetany
    - From oxytocin administration
  - Hypocarbia
  - Phenylphrine once thought to cause uterine hypoperfusion
    - Now widely accepted for use in the parturient
    - Overall increase in MAP outweighs uterine vessel constriction
    - Fetal pH improved after use of phenylephrine vs. ephedrine

➢ The placenta

- Uterine arteries perforate and deliver maternal blood to the intervillous spaces; drain via uterine veins
- Chorionic villi from fetus is bathed in maternal blood from uterine arteries
- Exchange of nutrients and wastes occurs via diffusion
  - There is no direct contact between maternal and fetal blood
- Umbilical arteries (2) deliver fetal blood to the placenta
- Umbilical vein (1) delivers O2 and nutrient-rich blood to the fetus

Placental transfer of drugs
- Determined by
  - placental perfusion
  - maternal to fetal concentration gradient
  - total dose of drug administered
  - vascularity of administration site
  - maternal pH
  - maternal protein binding
  - metabolism of drug
  - Pharmocologic determinants
    - dependent on drug diffusion constant (Fick)
      - placental transfer will be greater if:
        - ↓ degree of ionization of drug
        - ↓ molecular size of drug
        - ↓ protein binding of drug
        - ↑ lipid solubility of drug
    - NMBs, glycopyrrolate, insulin, heparin do not X placenta

- Transfer of local anesthetic across placenta
  - ion trapping—unionized LA crosses the placenta and becomes ionized/”trapped” in the relatively acidemic fetus—may be noted with the use of paracervical anesthesia
  - more highly bound LA less likely to cross placenta
    - protein binding
      - bupivacaine > ropivacaine > lidocaine > mepivacaine
  - Lower doses of LA needed in the parturient
    - engorgement of epidural vessels
    - ↑ sensitivity to the effects of LA (progesterone)
maternal CSF pH favors unionized portion; enhanced diffusion across nerve cell membrane

Fetal circulation

- allows nutrients and oxygen to be transferred from the mother to the fetus, and for deoxygenated blood to be returned to the mother
- shunts are in place to allow the majority of blood flow to bypass the fluid-filled lungs
  - intracardiac shunting from R→L occurs 2º ↑ fetal pulmonary vascular resistance from:
    - relative hypoxemia (HPV)
    - compression of pulmonary vasculature by fluid-filled alveoli
- 3 major shunts:
  - Ductus venosus
    - Allows blood returning from the uterine vein to bypass the liver; delivers O2-rich blood directly to vital organs
    - Becomes ligamentum teres and venosum after birth
  - Foramen ovale
    - Opening between R and L atria; blood is shunted across as a result of high fetal PVR
      - Most blood entering the RA follows this route; remainder enters RV
    - Becomes fossa ovalis
  - Ductus arteriosus
    - Communication between the pulmonary artery and the aorta
    - Blood entering from the RV into the PA is shunted across to the aorta
    - Becomes ligamentum arteriosum
    - May remain patent in premature infants
      - Closure either pharmacologic with indomethacin or via surgical ligation

**L recurrent laryngeal nerve most commonly injured during PDA ligation surgery**
Fetal PaO$_2$ is 23-25mm Hg in the carotid and descending aorta.
Progression of labor and analgesics

- T4 level necessary for Cesarean section
- Fetal monitoring
  - parameters followed:
    - FHR (120-160 BPM considered normal)
      - patterns (i.e. beat-to-beat variability, accelerations, decelerations)
        - loss of baseline variability may indicate beginning distress (fetal hypoxia/acidosis); may treat with O₂ and ephedrine
        - other factors which may ∫ variability include narcotics, locals, benzodiazepenes, barbiturates, inhalational agents, and anticholinergics
    - uterine activity (contractions)
      - remember “VEAL CHOP”

<table>
<thead>
<tr>
<th>Stage of labor</th>
<th>pain</th>
<th>analgesia</th>
</tr>
</thead>
<tbody>
<tr>
<td>1st</td>
<td>visceral and cervical</td>
<td>parenteral (opioids)</td>
</tr>
<tr>
<td></td>
<td>T₁₀  ⇒  L₁</td>
<td>epidural combo spinal/epid</td>
</tr>
<tr>
<td></td>
<td>A.K.A. latent and active phases</td>
<td></td>
</tr>
<tr>
<td>2nd</td>
<td>lower vagina, perineum</td>
<td>paracervical caudal pudendal</td>
</tr>
<tr>
<td></td>
<td>S₂  ⇒  S₄</td>
<td></td>
</tr>
<tr>
<td>3rd</td>
<td>delivery of placenta</td>
<td></td>
</tr>
</tbody>
</table>

- *T4 level necessary for Cesarean section*
Fetal Monitor Patterns

Reassuring Pattern
Baseline fetal heart rate is 120-160, preserved beat-to-beat and long-term variability. Accelerations last for 15 or more seconds above baseline, and peak to 15 or more bpm.

Late Deceleration with Variability Loss
Fetal heart rate lags behind contractions, with little or no variability in line. Persistent late decelerations associated with decreased variability is an ominous pattern.

Variable Decelerations
Variable decelerations are variable in duration, intensity, and timing. Acceleration-deceleration-acceleration is due to compression and decompression of cord.

Severe Variable Decelerations
Severe decelerations have depth below 70 bpm, and a duration longer than 1 minute. Persistent variable decelerations may lead to acidosis and fetal distress.

Fetal Monitor Patterns

Reassuring Pattern
Baseline fetal heart rate is 120-160, preserved beat-to-beat and long-term variability. Accelerations last for 15 or more seconds above baseline, and peak to 15 or more bpm.

Elevated Heart Rate: Tachycardia
Baseline fetal heart rate is above 160, possible onset of decreased variability. Usually due to fetus lacking nourishing blood supply, or resultant effects of some drugs.

Early Deceleration
The onset and the return of the deceleration coincides with the start and the end of the contraction. Decelerations are associated with fetal movement, stimulation, and uterine contractions.

Late Deceleration with Preserved Variability
Fetal heart rate returns to baseline AFTER the contraction has ended. Late decelerations are associated with uteroplacental insufficiency, or decreased uterine bloodflow.
Fetal monitoring patterns

- Accelerations and variability
  - Normal; indicates a healthy fetus who is responding well to the stress of labor and transient decrease in placental perfusion from contractions
  - Decelerations
    - **Early** (A.K.A. Type I)
      - Signifies fetal head compression
        - Benign; may be noted as fetal head engages or during pushing
        - Vagally mediated
        - Decels U-shaped, do not drop below 100 BPM
        - May treat with atropine (controversial)
    - **Late** (A.K.A. Type II)
      - Signifies uteroplacental insufficiency
        - Ominous
        - HR slows as a result of fetal myocardial ischemia
        - U-shaped; > 100 BPS
        - May tx initially with ephedrine, O₂, fluids
          - *Definitive tx = delivery of the fetus*
    - **Variable** (A.K.A. Type III)
      - Signifies cord compression
        - Look for prolapse
      - Shape irregular; FHR may drop below 100 BPM
      - May resolve with maternal position change
      - Generally regarded as benign unless HR remains low or cord prolapse is present
        - Indicates emergency C/S

- Fetal distress/scalp pH
  - Obtained if fetal monitoring indicates distress (A.K.A. “non-reassuring fetal tracing” or “fetal asphyxia”)
    - Early s/s
      - Loss of beat-to-beat variability
      - Decelerations
Meconium staining of amniotic fluid

- Scalp pH values

<table>
<thead>
<tr>
<th>pH Range</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>7.25-7.45</td>
<td>normal</td>
</tr>
<tr>
<td>7.20-7.24</td>
<td>indicates mild distress</td>
</tr>
<tr>
<td>&lt;7.20</td>
<td>indicates severe distress; immediate delivery required</td>
</tr>
</tbody>
</table>

Transition from intrauterine to extrauterine life

- Lungs transition from fluid to air filled with first few breaths
  - Neonate must generate tremendous (-) inspiratory pressure to inflate lungs
  - Surfactant necessary to maintain alveolar inflation
  - Fluid begins to drain during labor process; enhanced expulsion during vaginal delivery
  - C/S neonate may require postural drainage to facilitate drainage
  - Remainder of fluid is drained via the lymphatics over 24-48⁰

- Circulatory changes
  - Pulmonary vascular resistance drops by 80%
    - Expansion of neonatal lungs
    - Presence of O₂-rich environment
    - Release of arachadonic acid and other vasoactive metabolites
      - R-sided heart pressures decrease
  - Systemic vascular resistance increases dramatically
    - Umbilical cord clamping
      - L-sided heart pressures increase
  - Changes in heart pressures cause cessation of flow through FO
  - PDA begins to constrict
    - Presence of O₂-rich environment; ↑ pH
    - Loss of prostaglandins from the placenta
    - Functionally closed within ~ 3 days
      - Permanent closure 2-3 weeks
Apgar Scoring

Scores obtained at 1 and 5 minutes after birth
- 1 minute score directs need for resuscitation
- 5 minute score prognostic for long-term sequelae of anoxia
- Score 8-10
  - Most neonates fall into this category
    - Loss of points usually for presence of acrocyanosis
  - Keep neonate dry and warm; monitor for decline in condition
  - Suction PRN
- Apgar score 4-7
  - Indicates mild asphyxia
  - Stimulate
  - May need transient $O_2$ (BBO$_2$ vs. BMV vs. LMA)
  - Keep dry/warm; monitor for decline

<table>
<thead>
<tr>
<th></th>
<th>0</th>
<th>1</th>
<th>2</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>color</strong></td>
<td>completely cyanotic and/or pale</td>
<td>body pink with cyanotic extremities</td>
<td>completely pink</td>
</tr>
<tr>
<td><strong>heart rate (BPM)</strong></td>
<td>absent</td>
<td>&lt;100 BPM</td>
<td>&gt;100 BPM</td>
</tr>
<tr>
<td><strong>reflex irritability</strong></td>
<td>no response</td>
<td>grimace</td>
<td>cry, cough, sneeze</td>
</tr>
<tr>
<td><strong>breathing</strong></td>
<td>apneic</td>
<td>slow; weak cry</td>
<td>crying</td>
</tr>
<tr>
<td><strong>muscle tone</strong></td>
<td>limp</td>
<td>minor flexion of extremities</td>
<td>active; extremities well-flexed</td>
</tr>
</tbody>
</table>
- **Apgar score 0-3**
  - Indicates severe asphyxia
  - bag/mask ventilation; LMA insertion vs. tracheal intubation
  - external cardiac compressions if HR < 60 BPM
  - may administer drugs via umbilical vein or ETT*
    - intraosseous (tibia) reintroduced in PALS
  - *Remember NAVEL:
    - Naloxone, Atropine, Vasopressin (not indicated for pedi pts), Epinephrine, Lidocaine

- **Neonatal resuscitation medications/treatments**

<table>
<thead>
<tr>
<th>Medication</th>
<th>Dosage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Epinephrine</td>
<td>1:10,000 dilution → 0.1 ml/Kg</td>
</tr>
<tr>
<td>NaHCO₃</td>
<td>Dilute 1:1 → 1 mEq/Kg slowly</td>
</tr>
<tr>
<td>Calcium gluconate</td>
<td>50 mg/Kg</td>
</tr>
<tr>
<td>Atropine</td>
<td>0.02 mg/Kg</td>
</tr>
<tr>
<td>naloxone</td>
<td>10 – 100 mcg/Kg</td>
</tr>
<tr>
<td>Lidocaine</td>
<td>1 mg/Kg</td>
</tr>
<tr>
<td>Defibrillation</td>
<td>2j/Kg</td>
</tr>
<tr>
<td>Volume expansion</td>
<td>Albumin, PRBC (10 ml/Kg); D₁₀ 0.2g/Kg (hypoglycemia)</td>
</tr>
</tbody>
</table>

- **Maternal Considerations/Comorbidities**
  - **Pregnancy-induced hypertension (PIH, preeclampsia, toxemia)**
    - Secondary to a defect in the endovascular system
    - Etiology unknown
    - manifests after the 20th week of pregnancy; approximately 10% occurrence rate
    - multisystemic involvement
    - major cause of premature labor 2° uterine hyper reactivity
    - major cause of obstetric and perinatal m/m
    - primary causes of maternal death:
      - cerebral hemorrhage
      - pulmonary edema
    - S/S
      - HTN—BP 140/90 or greater
- proteinuria
- edema
  - peripheral, airway
- intravascular depletion
- ↑↑ sensitivity to endogenous and exogenous catecholamines
- CNS disturbances
- headache
- visual field disturbances
- hyperreflexia
- *↓ uteroplacental perfusion

### Complications
- Premature labor
- Seizures (eclampsia)
- HELLP syndrome
  - Heralded by Hemolysis, Elevated LF, Low Platelets
    - Precursor to DIC
    - Definitive tx – delivery of the fetus
- acute hypertension with laryngoscopy, addition of epinephrine to local for regional anesthesia may lead to seizures, cerebral hemorrhage, pulmonary edema
- *hydralazine, labetalol usually the antihypertensives of choice; may use small dose of esmolol pre-intubation
- difficult airway 2º edema
- coagulopathy may be present
  - check coags; able to decline precipitously; may preclude the use of regional anesthesia
  - may need to administer FFP, platelets, cryoprecipitate
- regional (specifically spinal) anesthesia may ppt profound hypotension
  - preload with 1-2 liter crystalloid (LR, normosol)
- may need invasive monitoring
- follow U/O carefully
should be > than 1ml/Kg/hour
may administer loop diuretics, mannitol

- ↓ plasma cholinesterase
  - Probably clinically insignificant

### Treatment

- Magnesium sulfate (MgSO₄)
  - relaxes smooth muscle of the vessels, uterus, and bronchioles
  - interferes with Ca²⁺ transport
  - ↓ muscle membrane excitability
  - ↓ motor end plate sensitivity
  - inhibits release of ACh; enhances effects of NDMBs
  - may also be utilized in the treatment of pre-term labor as a tocolytic
  - *therapeutic plasma level ⇒ 4-6 mEq/L

- s/s magnesium toxicity
  - hyporeflexia/loss of DTRs (earliest sign of impending toxicity)
  - skeletal muscle weakness
  - uterine atony
  - vasodilation/hypotension
  - AV block, prolongation of P-Q intervals and widened QRS
    - may lead to cardiac arrest
  - CNS depression
    - apnea
    - paralysis
  - MgSO₄ crosses the placenta with ease; all effects, especially hypotonia, may be seen in the fetus
  - treatment is with Ca²⁺, supportive measures

- Maternal (A.K.A. Postpartum) Hemorrhage
  - Typical EBL for vaginal delivery 400-600 ml; C/S 800-1000 ml
  - Most common cause of maternal morbidity in developed countries
  - Causes
Remember “The Four Ts”

<table>
<thead>
<tr>
<th>Tone</th>
<th>Uterine atonia 1º cause of PP hemorrhage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trauma</td>
<td>Cervical, vaginal lacerations; uterine inversion, placental abruption</td>
</tr>
<tr>
<td>Tissue</td>
<td>Retained placental fragments; placental malimplantation</td>
</tr>
<tr>
<td>Thrombin</td>
<td>Coagulopathy, either intrinsic or acquired</td>
</tr>
</tbody>
</table>

- Placenta previa
  - Abnormally low implantation of the placenta either partially or completely obscuring the cervical os
  - Heralded on/around the 32ª week of gestation
    - *Painless* vaginal bleeding
  - Necessitates C/S for delivery

- Abruptio placentae (A.K.A. placental abruption)
  - premature separation of placenta from uterine wall
  - risk factors:
    - HTN, abdominal trauma, ETOH or cocaine use, multiparity
  - *1º cause of DIC in the parturient*
  - vaginal bleeding may be overt or occult
  - may manifest as sudden maternal decompensation
  - *severe abdominal pain*
  - quickly leads to shock, fetal distress
  - necessitates emergency C/S

- Retained placenta
  - usually from fragmentation of the placenta
    - bleeding occurs from resultant uterine atony (2-5% of vaginal deliveries)
    - EBL may be as much as 2 liters/5mins
  - necessitates manual uterine exploration/removal of retained fragments
    - Epidural (if in place) vs. GA vs. IV sedation
    - NTG 80-120 mcg IV given to relax uterus
  - may be 2º abnormal implantation
- accreta, increta, percreta
  - may necessitate uterine artery embolization, emergency hysterectomy

- Treatment of uterine atony
  - Uterine massage
  - Uterotonics the mainstay of treatment
    - Hormones
      - Oxytocin
        - posterior pituitary hormone which increases uterine tone
        - may cause vasodilation, hypotension, tachycardia, flushing if rapidly administered
        - original formulation had potential to cause SIADH
          - resulted in $H_2O$ intoxication and hyponatremia
          - unlikely with the use of synthetic oxytocins which do not contain vasopressin
      - Ergot alkaloids
        - Methylergonamine (Methergine)
may cause hypertension, N/V
*contraindicated in parturient with PIH

Prostaglandins
- PGF 2α Carboprost (Hemabate)
  - May cause bronchospasm
  - *contraindicated in the asthmatic parturient

Obstetric emergencies

Uterine inversion
- necessitates GA with RSI
  - ketamine preferred indx agent (supports BP)
- large bore IV, blood products
- uterine relaxation
  - volatile anesthetics
  - NTG 50-100 mcg IV boluses
- oxytocin gtt is started after uterus is replaced; prevents recurrence

Uterine rupture
- 80% occur spontaneously with no predisposing factors
- 1% incidence
- High risk groups
  - grand multiparous pts
  - precipitous delivery
  - uterine overstimulation
  - VBAC (1º concern)

S/S
- sudden, continuous, intense abdominal pain, even despite epidural anesthesia
- change in uterine tone/contraction pattern; loss of tracing on the tocometer
- maternal hypotension
- fetal bradycardia/distress
- **incidence of perinatal mortality (fetal mortality ~ 80%)
Amniotic fluid embolus

- manifests as a sudden onset of respiratory distress and ↓ BP
  - ↓SaO₂, ↓ETCO₂,
  - leads to complete CV collapse
- greater incidence in multiparous pts during precipitous delivery
  - dx confirmed by the presence of amniotic fluid/fetal material in maternal blood sample
- may lead to development of DIC, uterine atony, hemorrhage
- treatment is supportive (CPR, pressors, aminocaproic acid to tx DIC, NaHCO₃, steroids, correction of hypoxemia, HOB slightly ↑ with 15° L lateral tilt, LUD)
- mortality rate 86% (50% during 1st hour)
- 3rd leading cause of maternal mortality

Anesthesia for non-obstetric surgery

- Common surgeries during pregnancy: cholecystectomy, ovarian cystectomy, appendectomy, cervical cerclage
- primary concerns
  - avoidance of fetal hypoxia/acidosis
    - 1º fetal risk ⇒ uterine asphyxia
  - avoidance of potentially teratogenic anesthetic agents, especially in 1st trimester during period of organogenesis
    - N₂O
    - benzodiazepines
  - 1º overall risk ⇒ development of premature labor
    - usually 2º underlying pathology/surgical procedure
  - treat with β agonists (terbutaline, ritodrine), MgSO₄, CCBs
- anesthetic goals
  - delay surgery if possible until 2nd trimester
  - left uterine displacement (LUD) after 20th week gestation
- maintenance of maternal BP
  - fluids
  - *epinephrine vs. phenylephrine
• monitoring of FHR and uterine activity
• conduction anesthesia if possible
  ◦ limits fetal exposure to anesthetics

❖ Emergency C/S
  ➢ may be done under spinal anesthesia in certain cases; indwelling epidural
catheter may be dosed with carbonated lidocaine 2% or 2-3%
chloroprocaine (rapid onset)
  ➢ must achieve a T₄ level
  ➢ GETA necessary for truly emergent scenario
    ▪ maternal hemorrhage, persistent fetal bradycardia
  ➢ preoxygenate with FiO₂ 1.0
    ▪ anticipate airway difficulties
    ▪ have a variety of blades, small (#5.5 – 6.5) ETT, stubby laryngoscope
      handle, bougie, Proseal LMA, Fastrach or ILA, cricothyrotomy set,
      Glidescope, fiberoptic bronchoscope
  ➢ place pt supine with (+) LUD
  ➢ pt is prepped/draped before induction
    ▪ surgeon makes incision immediately after verification of ETT placement;
      allows for minimal fetal exposure to anesthetics
      • Propofol may result in neonatal hypotonicity
      • NMBs do not cross the placenta
  ➢ rapid sequence induction
  ➢ HOB ↑ 30°
  ➢ suction available
  ➢ stylet in ETT
  ➢ (+)/(-) use of Na⁺ citrate
    ▪ Pt is at high risk for aspiration
    ▪ Pt is at high risk for failed airway
  ➢ *maintain cricoid pressure until placement of ETT is verified by (+) ETCO₂
    and BBS
  ➢ do not hyperventilate pt
    ▪ hypocarbia results in placental hypoperfusion
anticipate need for volume resuscitation
VAE a risk, especially if the uterus is exteriorized and above the level of the heart during repair
- S/S
  - Chest pain, SOB, ↓ETCO₂
- Treatment
  - O₂ and supportive measures (i.e. pressors), HOB ↓with L lateral tilt
- may need to assist with neonatal resuscitation as well

Other fun OB factoids
- parturient is *always* considered a “full stomach” and at risk for aspiration
  - always needs RSI, even for non-obstetric procedures
- parturient has airway edema and friable tissues
  - magnified in the 3rd trimester
  - may lead to difficult airway scenario
    - *reassess airway in the laboring parturient who is in need of C/S*
      - Mallampati may drastically change after the process of labor (fluid administration, ↑CVP from pushing, etc...)
- infant born to mom with gestational diabetes at ↑risk for macrosomia, hypoglycemia
- ? use of atropine; may mask fetal distress
- all tocolytics cross the placenta; β agonists may lead to hyperglycemia, tachycardia, hypokalemia from potentiation of the Na/K⁺ pump, pulmonary edema
- most common musculoskeletal complaint of the parturient is low back pain
- *lumbosacral nerve is most commonly injured after vaginal delivery*
- *In the parturient, the incidence of post-dural-puncture headache after an inadvertent “wet tap” with a 17 g epidural needle is 70-80%!!!************!
  - Definitive tx – epidural blood patch (EBP)
- most drugs are safe during lactation
  - exceptions are lithium and ergotamine

Airway scenarios
- Routine C/S, GETA planned, no fetal distress, can ventilate but can’t intubate?
  - Wake pt up; attempt AFI
- Emergency C/S, GETA necessary, fetal distress, can ventilate but can’t intubate?
  - LMA (Pro seal, fast trach, ILA good choices)
- Can’t ventilate/can’t intubate?
- **LMA 1** st, cricothyrotomy