Hypertensive Disorders in Pregnancy
Incidence

- Hypertensive disorders of pregnancy – 12-22%
- Gestational HTN
  - 6-17% nulliparous women
  - 2-4% multiparous women
- Preeclampsia – 5-8%

ACOG 2002
Risk Factors

• 1\textsuperscript{st} pregnancy
• Previous pregnancy with preeclampsia
• Age $\geq 35$ yrs
• Multiple pregnancy
• African American race
• Chronic medical conditions

ACOG 2002
Different Classifications

- Chronic hypertension
- Gestational hypertension
- Preeclampsia
- Superimposed Preeclampsia
Chronic Hypertension (NHBPEP)

- Chronic Hypertension
  - HTN prior to conception, or diagnosed before 20th week of gestation that does not resolve postpartum
Gestational HTN

• SBP ≥ 140 or DBP ≥ 90 on 2 occasions > 4 hours apart developing after 20 weeks gestation
• No changes in labs
• May be transient or chronic in nature
• Returns to normal postpartum

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Preeclampsia

- Preeclampsia
  - Systemic disease with hypertension accompanied by proteinuria ($\geq 300$mg in 24 hr or Protein/Creatinine ratio of $\geq 0.3$) or thrombocytopenia, impaired liver function, or new onset renal insufficiency without proteinuria

ACOG 2002, 2013
Superimposed Preeclampsia

- Preeclampsia that develops in the presence of preexisting hypertension or renal disease
Preeclampsia with Severe Features

- SBP ≥ 160 or DBP ≥ 110 on two occasions
  - Confirmation can occur quickly to help facilitate antihypertensive therapy within 4 hours of diagnosis
- Lab changes
- Symptomatic

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Preeclampsia with Severe Features

- Labs:
  - Proteinuria ≥ 5g in 24 hours or ≥ 3+ on dipstick on 2 occasions ≥ 4hrs apart
  - Oliguria <500cc in 24 hrs
  - Elevated liver enzymes (twice normal)
  - Thrombocytopenia (<100,000)
  - Progressive renal insufficiency – Cr 1.1mg/dl

ACOG 2002, 2013
Symptoms:
- Headache
- Visual disturbances
- Epigastric of RUQ pain unresponsive to meds
- Pulmonary edema

ACOG 2002, 2013
Preeclampsia with Severe Features

- Additional Considerations:
  - Fetal growth restriction may be present
  - HELLP
Eclampsia

• Eclampsia
  – Convulsive stage, when seizures cannot be attributed to other causes
Normal Pregnancy Changes

• Markedly vasodilated state
  – Peripheral resistance ↓ 25% due to ↑ in prostacyclin (potent vasodilator) and endothelium-derived relaxing factor
• 50% ↑ in total blood volume by end 2nd trimester
• CO ↑ in 1st trimester, peaking at 35% to 50% above non-pregnant levels
Normal Pregnancy Changes

- ↑ renal blood flow, ↑ GFR, activation of renin-aldosterone system (results in falling BP)
- DBP drops to an average of 10 mmHg below non-pregnant values by mid-pregnancy, then slowly approaches non-gravid levels in 3rd trimester
Pathophysiology of Pre-Eclampsia

• “Disease of theories” - no established cause

• 2 stages:
  – Disruptions in placental perfusion
  – Maternal syndrome

• Abnormal prostacyclin:thromboxane ratios
  • Prostacyclin $\rightarrow$ vasodilation and inhibits platelet aggregation
  • Thromboxane $\rightarrow$ vasoconstriction and platelet aggregation
Disruptions in Placental Perfusion

• Normal pregnancy: cells from placenta invade the maternal spiral arteries, causing the vessels to change from thick-walled vessels to flaccid sac-like vessels

• Results in \( \uparrow \) vessel diameter and expansion to accommodate the 10-fold \( \uparrow \) in uterine blood flow
In preeclampsia, the vessels remain thick-walled and a distinctive lesion (acute atherosis) noted.

These lesions, along with placental infarcts, lead to ↓ placental perfusion and hypoxia.

It is the placental hypoxia that is believed to be the initial cause of preeclampsia.
Maternal Syndrome

• Normal pregnancy:
  – Systemic vasorelaxation occurs, results in ↑ blood volume, HR, CO

• Preeclampsia
  – Blood vessels become hypersensitive to vasoactive hormones and vasoconstriction occurs
Widespread Vasospasm

- Arterial vasospasm
- Disrupts arterial & venous circulation due to alternating segments of constriction & dilatation, resulting in endothelial damage
- Vascular wall damage activates platelet aggregation, ↓ intravascular volume, results in tissue ischemia from impaired blood flow to organs
- BP rises as result of narrowed lumens (vasoconstriction) and high resistance state
Effects on Systems/Organs

- C = Cardiovascular
- H = Hematologic
- H = Hepatic
- U = Uteroplacental
- R = Renal
- N = Neurologic
Cardiovascular

• Hypertension
  – arterial vasospasm $\rightarrow$ ↑ SVR, HTN, edema
  – ↓ organ perfusion
R/O White Coat HTN

• ~ 15 to 30% of the general population exhibits “white coat” HTN, but a higher incidence is noted among the elderly and among pregnant women
Hematologic

- Vasospasm → vascular damage
- ↑ hct
  - inadequate plasma volume expansion
  - ↑ blood viscosity, ↓ maternal organ perfusion

- Thrombocytopenia
  - schistocytes, burr cells
  - ↑ bili
  - hemolysis compromises tissue O2 delivery
Normal RBC’s
Shistocytes
Burr Cells
Hematologic

• Change in plasma proteins
  – Colloid oncotic pressure -- “pulling pressure of proteins”

• Hydrostatic (water) pressure
  – pushing pressure of intravascular fluid against the vessel walls
  – balance of these 2 maintains fluid levels in capillaries
  – If COP < hydrostatic pressure, fluid moves rapidly out of capillaries
Hematologic

• Hemoconcentration
  – due to capillary permeability, ↓ plasma volume
  – ↓ tissue perfusion, reduced blood flow
  – norm is hemodilution & physiologic anemia

• DIC
  – rare, may occur if severe preeclampsia

• HELLP
  – clotting studies, liver function tests
HELLP Syndrome

• A variant of severe pre-eclampsia

• H = Hemolysis

• EL = Elevated Liver enzymes
  – 2x normal

• LP = Low Platelets
  – < 100,000
Assessment

• Look for s/s bleeding
• Monitor labs - watch for ↓ platelet count, lab value changes
• Observe for s/s of anemia
• Observe for adequate oxygenation
Hepatic

- Vasospasm & fibrin deposits may obstruct blood flow & cause ischemic changes in liver
- Ischemia, necrosis, and intrahepatic hemorrhage can lead to hepatic rupture
- ↑ liver enzymes due to poorly perfused liver
- Glucose metabolism, excretion of drugs may be affected
Assessments

• Assess for RUQ epigastric pain, substernal pain, tenderness to palpation

• N/V, malaise, viral-like syndrome

• Monitor labs for liver function
  – AST, ALT, LDH, bilirubin
Uteroplacental System

- **Physiology:**
  - Vasospasm causes ↓ in uteroplacental perfusion
  - ↓ in O2 and nutrients to fetus
  - ↑ risk placental abruption

- **Assessment:**
  - Evaluate FHR pattern, watch for signs of ↓ perfusion & O2
  - Assess UC activity/pattern
  - Fetal surveillance for activity, growth, AFV
Renal System

• Vasospasm & damage results in ↓ flow through kidneys, ↓ GFR, ↓ creatinine clearance, and ↓ urinary output
  – normal pregnancy, renal plasma volume & GFR ↑ 30-50%

• Kidneys unable to excrete waste products (urea, creatinine, uric acid)

• Proteinuria due to kidney damage, larger molecules leak out

• Important to remember when giving meds excreted by kidneys, ie, MgSO4
Renal (con’t)

• Proteinuria - classic sign

• Decreased urine output

• Edema
Renal Assessment/Care

- Weight gain
- Edema
- Breath sounds
- Pulse ox
- Strict I&O
- Infusion pumps
- Hourly output, call if < 30 cc/hr

- Watch Mg levels closely
- Monitor urine protein
- Monitor lab values:
  - ↓ H&H
  - ↑ BUN
  - ↑ creatinine
  - ↑ uric acid
Neurologic System

- ↓ cerebral perfusion due to HTN
- Vessel damage & vasospasm can cause ↑ intracranial pressure, cerebral edema, ischemia, hemorrhage
- S/S: drowsiness, tinnitus, visual disturbances (blurred vision, photophobia), persistent HAs, N/V
- Hyperreflexia & clonus
- Seizure
Neurologic Assessment

- Assess for HA, visual disturbances, drowsiness, N/V, altered LOC, hypersensitive to noise/light
- Assess DTR’s/clonus
- Monitor Mg level
- Assess for s/s of seizure, protective environment, seizure tray at bedside
DTR’s

• 0  No response, hypoactive, abnormal
• +1 Somewhat diminished, not abnormal
• 2+ Average response
• 3+ Brisker than average, not abnormal
• 4+ Hyperactive, very brisk, abnormal
Clonus

• Repetitive, rhythmic contractions of a muscle when attempting to hold it in a stretched state
• A strong, deep tendon reflex that occurs when the CNS fails to inhibit it
• Often tested for in the ankle, wrist, and kneecap
• Normal = no beats
• Video
  http://www.mult-sclerosis.org/clonus.html
Neurologic Assessments

- Pupil size (equal, size, reactive)
- Hand grasp
- Level of consciousness
- Response to painful stimuli
Other Nsg Assessments

• Emotional health assessment
  – communicate w/patient/family
  – referrals as necessary

• Provide education
  – disease process
  – procedures, pertinent policies
  – explain equipment to family
When to Deliver

• At diagnosis after 37 weeks for any preeclampsia or Gestational HTN
• At diagnosis after 34 weeks if severe features
• Prior to 34 weeks – give corticosteroids (Betamethasone) and seek expert care (MFM) for individualized plan of care
Goals

• Prevent preeclampsia
• Appropriately timed delivery
• Control of blood pressure
  – Goal is 140-159/90-109
“Okay... Daddy’s back in the shower. I’ll flush this time and you go up and watch.”
Common Medications

- Magnesium sulfate
- Hydralazine
- Nifedipine
- Labetalol
MgSO4

- Drug of choice for seizure precautions - CNS depressant
  - Not necessary in the absence of severe features
- Loading dose: 4-6 grams over 20-40 minutes
- Maintenance dose: 1-3 grams/hr
- Therapeutic range 5-8 mg/dl
- Antidote - calcium gluconate

# Mg Levels & Toxicity

<table>
<thead>
<tr>
<th>Sign or Symptom</th>
<th>Mg Level (mg/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Loss of patellar reflexes</td>
<td>8-12</td>
</tr>
<tr>
<td>Feeling of warmth/flushing</td>
<td>9-12</td>
</tr>
<tr>
<td>Somnolence</td>
<td>10-12</td>
</tr>
<tr>
<td>Slurred speech</td>
<td>10-12</td>
</tr>
<tr>
<td>Muscular paralysis</td>
<td>15-17</td>
</tr>
<tr>
<td>Respiratory depression</td>
<td>15-17</td>
</tr>
<tr>
<td>Cardiotoxicity</td>
<td>30-35</td>
</tr>
</tbody>
</table>
Hydralazine/Apresoline

• Antihypertensive, vasodilator
• Dosing: 5-10 mg IV
  – Administer over 2 min
  – Can repeat every 20 min
  – Max of 30mg

Labetalol

- Beta blocker - reduce arterial vasospasm, ↓ BP, prevent vascular injury
- Dosing: Initial dose 20mg IV
  - Recheck in 10 min: Give 40mg if needed
  - Recheck in 10 min: Give 80mg if needed
  - Maximum of 300mg

Procardia/Nifedipine

- Calcium channel blocker - ↓ intravascular resistance and ↑ C.O.
- Dosing: 10mg orally
- May repeat in 30 min if needed (can do 20mg)
- Max 50mg

Treatment of Eclampsia

- Goal is to protect the patient
- Avoid insertion of airways and padded tongue blades - can cause upper airway trauma and hemorrhage
- IV access
- Suction, O2
- MgSO4 2gm IV over 5 minutes
Alternative Anticonvulsants

- Lorazepam (Ativan) 2-4mg IV x1, may repeat x1 after 10-15 minutes
- Diazepam (Valium) 5-10 mg IV every 5-10 min to max of 30mg
- Phenytoin (Dilantin) 15-20mg/kg IV, may repeat 10mg/kg IV after 20 minutes if no response; avoid with hypotension, may cause cardiac arrhythmias
- Keppra 500mg IV or orally, may repeat in 12 hours, dose adjustment needed if renal impairment
Post Seizure Care

- Assess maternal labs
- Assess fetal well-being, inutero resuscitation
- Plan for delivery, induction okay if cervical readiness
- No need for immediate C/S
Other Complications

- **Pulmonary edema**
  - Fluid overload & ↓ COP
  - Occurs more often after delivery
  - Lasix 10-20mg IV

- **Oliguria**
  - Goal at least 30cc/hr
  - Small fluid boluses (250-500cc)
  - PP diuresis common
Other Complications

• Persistent hypertension
  – BP may remain ↑ for several days
  – by definition, preeclampsia resolves by 6 weeks

• DIC
  – rarely occurs without abruption
  – low platelets is not DIC
  – requires blood products & delivery
Postpartum Course

• Cardiogenic
  – Rapid improvement \(\rightarrow\) ? Hemorrhage
  – Blood loss not tolerated well
  – Use of methergine contraindicated
  – Watch for uterine hypotonus w/MgSO4
  – \(\downarrow\) in BP and U.O. may indicate hemorrhage and shock
Postpartum

- **Hematologic**
  - Thrombocytopenia resolves ~ 3 days
- **Hepatic**
  - Improvement in liver function studies
- **Uteroplacental**
  - Monitor vag flow closely, weigh pads
  - Watch uterine tone
Postpartum

• Renal
  – Improvement in urine output
  – Large diuresis sign of recovery
  – Improvement in BUN, creatinine, uric acid

• Neurologic
  – Regression of HA
  – Reflexes return to normal
Postpartum

• Subjective improvement
  – Treat until signs of improvement
  – If mom isn’t feeling well, she isn’t well
  – Keep MgSO4 running until signs the disease is regressing and mom feels better
Case Studies

• Examples of clinical errors when administering Magnesium Sulfate and the outcomes…
A woman in PTL was receiving IV MgSO4. Despite treatment, precipitous labor & birth occurred. Oxytocin was ordered after delivery of placenta. Instead of oxytocin, the MgSO4 was infused at a rapid rate. (The MgSO4 infusion had been DC’d and the IV line removed from the pump, but remained connected to the patient at the Y port.) Pt. returned to her LDR room and VS assessed. Approximately 30 min. later, pt. found unresponsive and not breathing. Code initiated, resuscitation difficult, defibrillation x 3 and 9 minutes to restore HR. Subsequently learned she had ~ 550 ml of the IV MgSO4 (22g). Despite aggressive Rx, calcium chloride, and ICU care, she did not regain consciousness and remains in a persistent vegetative state.

MCN, May/June 2004
A woman with preeclampsia being transferred to a tertiary care center was receiving IV MgSO4 at 2g/hr. Because the maternal transport team was not available, a critical care nurse was selected to accompany the patient. Illegibly written orders to continue the mag sulfate at 2 g/hr were interpreted by the nurse to be 7 g/hr, and the dosage was adjusted accordingly. The patient appeared stable, was noted to be sleeping during the last portion of the trip, and found to have respirations of 10/min. on arrival. The error in order interpretation and dosage was discovered by nurses at the accepting institution. Calcium gluconate was administered and respiratory status improved quickly.
A woman w/PTL had orders for 6g load of MgSO4 to be followed by 3g/hr. Mainline IV fluids of LR were TRA 300ml/hr. RN mixed 40 gm of MgSO4 in 1L LR and labeled the bag. The RN gave the 6g loading dose without an infusion device and then used a pump to give the MgSO4 and mainline fluids. Over next 3 hrs, woman reported feeling flushed/nauseated. DTRs not assessed. RR < to 10. RN assured woman/family these symptoms were expected. Woman eventually appeared to be sleeping deeply, RN assumed she needed rest after the stress of being admitted w/PTL. Pt’s family left for dinner, upon return found the woman not breathing. RN called to room and could not palpate a pulse. Code initiated but was unsuccessful. Mg toxicity suspected, IV fluids sent for analysis. Discovered the bags had been mislabeled, received 12g/hr x 3 hours.
Woman 4 days PP presented to ED in small rural hospital with c/o severe HA, blurred vision, epigastric pain. BP 168/110 and 4+ pitting edema in legs. ER MD ordered MgSO4 to be given (2 g load followed with 1 g/hr). A premixed bag of 40g/1000ml fluid sent to ED. Together, the ED MD and RN calculated the infusion rate for the pump to deliver 1g/hr. Jointly decided that 250 ml was the correct infusion rate. Two hrs after the infusion started, woman transferred to PP unit. Report abbreviated because ED was busy. The admitting nurse noted the 250 ml rate and assumed the MgSO4 must have been discontinued and that this rate was the mainline bag, because she knew 250 ml/hr was too high for MgSO4. Did not confirm rate and contents of each line. Approx. 30 min. later, pt’s husband called to say his wife was feeling weak & unable to move. Error noted when pt. reassessed. Pt. had received 25g MgSO4 over 2.5 hrs. Calcium gluconate given, pt. rapidly improved.
References