Managing Hypertensive Crisis from Preeclampsia

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Hypertension in Pregnancy!

Task Force on Hypertension in Pregnancy

Why is it important?

- Complicates 10% pregnancies worldwide
- One of the greatest causes of maternal & perinatal morbidity and mortality
- \( \approx 50,000 - 60,000 \) preeclampsia –related deaths per year worldwide
- In the US:
  - Incidence has increased 25% in US during past 20 yrs
  - For every death from preeclampsia, 50 – 100 women have “near miss” events, significant health risks and costs

Improving Health Care Response to Preeclampsia: A California Quality Improvement Toolkit

CMQCC PREECLAMPSIA TOOLKIT/PREECLAMPSIA CARE GUIDELINES CDPH-MCAH Approved: 12/20/13
Available online at www.cmqcc.org

Grouped Cause of Death 2002-2004 (N=145)

<table>
<thead>
<tr>
<th>Grouped Cause of Death</th>
<th>Chance to Alter Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Strong (%), Good (%), Some (%), None (%), Total N (%)</td>
</tr>
<tr>
<td>Obstetric hemorrhage</td>
<td>69, 25, 6, 16 (11)</td>
</tr>
<tr>
<td>Deep vein thrombosis/pulmonary embolism</td>
<td>53, 40, 7, 15 (10)</td>
</tr>
<tr>
<td>Sepsis/infection</td>
<td>50, 40, 10, 10 (7)</td>
</tr>
<tr>
<td>Preeclampsia/eclampsia</td>
<td>30, 50, 0, 25 (17)</td>
</tr>
<tr>
<td>Cardiomyopathy &amp; other cardiovascular causes</td>
<td>25, 61, 14, 28 (19)</td>
</tr>
<tr>
<td>Cerebral vascular accident</td>
<td>22, 0, 78, 9 (6)</td>
</tr>
<tr>
<td>Amniotic fluid embolism</td>
<td>0, 87, 13, 15 (10)</td>
</tr>
<tr>
<td>All other causes of death</td>
<td>46, 46, 8, 26 (18)</td>
</tr>
</tbody>
</table>
**Maternal Mortality Rate, California Residents; 1970-2010**

Maternal Hypertension in California, 1999-2005

Maternal hypertension identified at time of hospitalization for labor and delivery (includes pre-gestational and gestational hypertension)


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**How Do Women Die Of Preeclampsia in CA?**

CA-PAMR Final Cause of Death Among Preeclampsia Cases, 2002-2004 (n=25)

<table>
<thead>
<tr>
<th>Final Cause of Death</th>
<th>Number</th>
<th>%</th>
<th>Rate/100,000</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stroke</td>
<td>16</td>
<td>64%</td>
<td>1</td>
</tr>
<tr>
<td>Hemorrhagic</td>
<td>14</td>
<td>87.5%</td>
<td>12.5%</td>
</tr>
<tr>
<td>Thrombotic</td>
<td>2</td>
<td>12.5%</td>
<td>1</td>
</tr>
<tr>
<td>Hepatic (liver) Failure</td>
<td>4</td>
<td>16.0%</td>
<td>25</td>
</tr>
<tr>
<td>Cardiac Failure</td>
<td>2</td>
<td>8.0%</td>
<td></td>
</tr>
<tr>
<td>Hemorrhage/DIC</td>
<td>1</td>
<td>4.0%</td>
<td></td>
</tr>
<tr>
<td>Multi-organ failure</td>
<td>1</td>
<td>4.0%</td>
<td></td>
</tr>
<tr>
<td>ARDS</td>
<td>1</td>
<td>4.0%</td>
<td></td>
</tr>
</tbody>
</table>

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**Why is it important? (Cont.)**

- Many Preeclamptic deaths in US and worldwide reported as “preventable”
- Major contributor to prematurity
- Risk factor for future CV disease and metabolic disease in women
- Etiology remains unclear
- The only cure is delivery (of the placenta)

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**Management Issues Warranting Special Attention**

- Failure of healthcare providers to appreciate the multi-systemic nature of preeclampsia
- Preeclampsia is a dynamic and a progressive process
  1. Appropriate management mandates frequent reevaluation
  2. Can worsen or present after delivery – which can create a venue for adverse maternal events

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**ACOG Executive Summary on Hypertension In Pregnancy, Nov 2013**

1. The term "mild" preeclampsia is discouraged for clinical classification. The recommended terminology is:
   a. "preeclampsia without severe features" (mild)
   b. "preeclampsia with severe features" (severe)
2. Proteinuria is not a requirement to diagnose preeclampsia with new onset hypertension.
3. The total amount of proteinuria > 5g in 24 hours has been eliminated from the diagnosis of severe preeclampsia.
4. Early treatment of severe hypertension is mandatory at the threshold levels of 160 mm Hg systolic or 110 mm Hg diastolic.

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**ACOG Executive Summary on Hypertension In Pregnancy, Nov 2013 (cont.)**

5. Magnesium sulfate for seizure prophylaxis is indicated for severe preeclampsia and should not be administered universally for preeclampsia without severe features (mild).
6. Preeclampsia with onset prior to 34 weeks is most often severe and should be managed at a facility with appropriate resources for management of serious maternal and neonatal complications.
7. Induction of labor at 37 weeks is indicated for preeclampsia and gestational hypertension.
8. The postpartum period is potentially dangerous. Patient education for early detection during and after pregnancy is important.
9. Long-term health effects should be discussed.

**New!**

**2013 Classification of Hypertensive Disorders of Pregnancy**

- Four Categories
  1. Preeclampsia-eclampsia
  2. Chronic hypertension (of any cause)
  3. Chronic hypertension with superimposed preeclampsia
  4. Gestational hypertension

**Key Change:**

Diagnosis of Preeclampsia:
**Proteinuria Not Required**

- Recognizes the syndromic nature of preeclampsia
- The disease affects all organ systems

**Hypertension**

<table>
<thead>
<tr>
<th>Diagnostic Criteria for Preeclampsia</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Blood Pressure</strong></td>
<td>Greater than or equal to 140 mm Hg systolic or greater than or equal to 90 mm Hg diastolic on two occasions at least 4 hours apart after 20 weeks of gestation in a woman with a previously normal blood pressure</td>
</tr>
<tr>
<td><strong>Severe Hypertension</strong></td>
<td>Greater than or equal to 160 mm Hg systolic or greater than or equal to 110 mm Hg diastolic, hypertension can be confirmed within a short interval (minutes) to facilitate timely antihypertensive therapy</td>
</tr>
</tbody>
</table>

**New!**

Hypertension: Systolic BP of 140 mm Hg or higher, OR Diastolic BP of 90 mm Hg or higher

<table>
<thead>
<tr>
<th>Feature</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>蛋白尿（Proteinuria）</td>
<td>( \geq 300 \text{ mg/g} ) on a 24-hour urine collection, or ( \text{ protein/creatinine ratio} \geq 0.3 ) (each measured by mg/dL)</td>
</tr>
</tbody>
</table>

*Collection may be extrapolated from timed tests;* |

| Thrombocytopenia | Platelets <100,000/microliter |
| Impaired liver function | Increased serum liver transaminases to twice normal values |
| New development of renal insufficiency | Elevated serum creatinine greater than 1.1 mg/dL, or a doubling of serum creatinine in the absence of other renal disease |
| Pulmonary edema | |
| New-onset cerebral or visual disturbances | |
Preeclampsia

Hypertension* + Evidence of Organ System Involvement

• Systolic ≥140 mmHg

Or

• Diastolic ≥90 mmHg

Evidence of Organ System Involvement

• Proteinuria

• Thrombocytopenia

• Impaired liver function

• New development of renal insufficiency

• Pulmonary edema

• New-onset cerebral or visual disturbances

*On two occasions at least 4 hours apart (in prev healthy pt after 20 wks)

Severe Features of Preeclampsia

ANY of these Findings

Severe Feature

Definition

• Hypertension

• Systolic BP of 160 mmHg or higher, or

• Diastolic BP of 110 mmHg or higher, on 2 occasions at least 4 hours apart while the patient is on bedrest (unless antihypertensive therapy is initiated before this time)

• Thrombocytopenia

Platelets <100,000/microliter

• Impaired liver function

Abnormally elevated blood concentrations of liver enzymes (to twice normal concentration), severe persistent right upper quadrant or epigastric pain unresponsive to medication and not accounted for by alternative diagnoses, or both

• Progressive renal insufficiency

Serum creatinine concentration greater than 1.1 mg/dL or a doubling of serum creatinine concentration in the absence of other renal disease

• Pulmonary edema

• New-onset cerebral or visual disturbances

Severe Features of Preeclampsia

Hypertension*

Evidence of Organ System Involvement

• Systolic ≥140 mmHg

Or

• Diastolic ≥90 mmHg

Evidence of Organ System Involvement

• Proteinuria

• Thrombocytopenia

• Impaired liver function

• New development of renal insufficiency

• Pulmonary edema

• New-onset cerebral or visual disturbances

*On two occasions at least 4 hours apart (in prev healthy pt after 20 wks)
2. **Chronic**: Hypertension that predates pregnancy  

3. **Gestational**: Hypertension is BP elevation after 20 weeks of gestation in the absence of proteinuria or the aforementioned systemic findings.  

4. **Superimposed Preeclampsia**: Chronic hypertension in association with preeclampsia.

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### Risk Factors for Preeclampsia

- Primiparity  
- Prior preeclamptic pregnancy  
- Chronic hypertension or chronic renal disease or both  
- History of thrombophilia  
- Multifetal pregnancy  
- In vitro fertilization  
- Family history of preeclampsia  
- Type 1 or 2 diabetes mellitus  
- Obesity  
- Systemic lupus erythematosus  
- Advanced maternal age (older than 40 years)

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### Key Observations

- ↑ Risk for Preeclampsia  
  - Twofold to fourfold if the pt has a first-degree relative with a medical Hx of preeclampsia  
  - Sevenfold if pt had preeclampsia in prior pregnancy  
- Triplet Gestation Greater Risk than Twin; twin greater than singleton pregnancy

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### Prediction

- Current attempts have only produced modest prediction value (risk factors)  
- No improvement in maternal or fetal outcome related to Uterine Artery Doppler screening (no randomized control trials)  
  - may be technique/standardization issues  
- Biomarkers for prediction said to be “integral” to disease stratification, targeted therapy

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### Angiogenesis-Related Biomarkers

- Several circulating anti-angiogenic proteins and pro-angiogenic proteins have been studied as possible biomarkers for preeclampsia  
- Maternal risk factors + Biomarkers = show future promise as algorithms for predicting the disease  
- However, the Task Force does not recommend using in clinical practice (no evidence that early screening improves outcomes)
**Preeclampsia**

- Current model of preeclampsia is one of **vascular mal-adaptations** stemming from implantation
- Results in **vessel epithelium injury** which may cause release of pro-inflammatory agents/mediators that produce cytokine release
- **Cytokines**, if released in large quantities, can produce the Cytokine Release Syndrome seen in some oncology patients when receiving chemotherapy using monoclonal antibodies.

**New Definition of Preeclampsia**

- Based on research findings from past 10 years
- Emphasis on identification of preeclampsia based on the presence of hypertension and evidence of organ system involvement from any of the systems most susceptible to specific insults

**Preeclampsia: Inflammatory Consequences**

- Vessel damage may trigger complex inflammatory changes to the arterial endothelial layer, resulting in
  - Third spacing of fluid in pulmonary system (non-cardiogenic pulmonary edema)
  - Leftward shift in the oxyhemoglobin dissociation curve
  - Potential decreased oxygen consumption
  - Activation of the coagulation cascade, stimulation of pro- and anti-coagulants, and fibrinolysis

**Cytokines**

- When cytokines are released into the circulation, systemic symptoms such as fever, nausea, chills, hypotension, tachycardia, asthenia, headache, rash, scratchy throat, and dyspnea can result.
- Abnormal vessel damage occurring in preeclampsia may have similar effects on pregnant patients, and lead to some of the signs of symptoms associated with HELLP syndrome.

**Diagnosis**

- Hypertension

  PLUS...

- Evidence of organ system involvement
  - Measure function of systems sensitive to hypoxemia, endothelial damage, reduced blood flow/O2 transport, etc.
Systemic Involvement
↓
Unpredictable Patterns of Cellular/Organ System Stress and Injury
↓
Diagnosis Challenge
↓
Preeclampsia or Something Else?

### Differential Diagnoses

<table>
<thead>
<tr>
<th>Condition</th>
<th>HELLP</th>
<th>Hypertension</th>
<th>AFLP</th>
<th>ITP</th>
<th>HUS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood Pressure</td>
<td>Increased</td>
<td>Variable or Increased</td>
<td>WNL</td>
<td>Variable</td>
<td>WNL</td>
</tr>
<tr>
<td>Platelets</td>
<td>Decreased</td>
<td>Variable or Increased</td>
<td>WNL</td>
<td>Variable</td>
<td>WNL</td>
</tr>
<tr>
<td>Liver enzymes</td>
<td>Decreased</td>
<td>Variable or Increased</td>
<td>WNL</td>
<td>Decreased</td>
<td>WNL</td>
</tr>
<tr>
<td>Clotting Factors</td>
<td>WNL, decreased</td>
<td>WNL, or decreased</td>
<td>Decreased</td>
<td>WNL</td>
<td>WNL</td>
</tr>
<tr>
<td>Liver enzymes</td>
<td>WNL</td>
<td>Increased</td>
<td>Increased</td>
<td>WNL</td>
<td>WNL</td>
</tr>
<tr>
<td>Hemoglobin/Hematocrit</td>
<td>Decreased</td>
<td>Decreased</td>
<td>Decreased</td>
<td>WNL</td>
<td>WNL</td>
</tr>
<tr>
<td>Clotting factors</td>
<td>WNL, decreased</td>
<td>WNL, or decreased</td>
<td>Decreased</td>
<td>WNL</td>
<td>WNL</td>
</tr>
<tr>
<td>Liver enzymes</td>
<td>WNL</td>
<td>Increased</td>
<td>Increased</td>
<td>WNL</td>
<td>WNL</td>
</tr>
<tr>
<td>Nausea &amp; Vomiting</td>
<td>None</td>
<td>Common</td>
<td>Common</td>
<td>None</td>
<td>None</td>
</tr>
</tbody>
</table>

### Stroke in Pregnancy

- **Incidence:**
  - Approximately 9 to 34 per 100,000
- **Types**
  - Intracerebral hemorrhage during pregnancy carries the highest morbidity and mortality, with an in-hospital mortality of 20%.

### Stroke Causes

#### Unique to Pregnancy
- Preeclampsia/eclampsia
- Postpartum angiopathy
- Amniotic fluid embolism
- Postpartum Cardiomyopathy

#### Non-Pregnant Women
- Hypertension
- Diabetes
- Vasculitis
- Arteriovenous malformations
- Aneurysms

### Stroke in Pregnancy: Risk Factors

- >35 years
- African-American
- Preeclampsia/eclampsia/gestational hypertension
- Thrombophilies
- Migraine headaches
- Diabetes
- Chronic hypertension
Key Clinical Pearl

Controlling blood pressure is the optimal intervention to prevent deaths due to stroke in women with preeclampsia.

Over the last decade, the UK has focused QI efforts on aggressive treatment of both systolic and diastolic blood pressure and has demonstrated a reduction in deaths.

Case Study PEMD.05

Question?
What is the lowest value of systolic blood pressure that would classify a patient as having “severe” preeclampsia?

A. Systolic > 180 mmHg
B. Systolic > 160 mmHg
C. Systolic > 140 mmHg
D. Systolic > 120 mmHg

ACOG Hypertensive Emergency Treatment Guidelines, CO #514

“Hypertensive Emergency”

- Acute-onset
- Severe Hypertension
  - Systolic ≥ 160 mm Hg, OR
  - Diastolic ≥ 110 mm Hg,
  - OR Both
- Accurately measured using standard techniques and
- Persistent for ≥ 15 minutes [is considered] a hypertensive emergency.
ACOG, 2015 HYTN: Box 3
Oral Nifedipine as First Line Agent

Antique Fire Hose Nozzles

Physiology of Blood Pressure

Blood Pressure = Flow x Resistance

MAP = Cardiac Output (CO) x Systemic Vascular Resistance (SVR)

Antihypertensive agents will typically reduce “flow” (cardiac output), OR “resistance” (SVR), or both.

Some side effects are therefore, consequences of too much reduction in cardiac output or SVR.
In all cases, treatment should be re-instituted once BP levels during pregnancy.

When BP < threshold, then every 15 min x 1 hour.

When BP > threshold, administer labetalol (80 mg IV over 2 minutes). If BP is below threshold, continue to monitor BP closely.

Repeat BP measurement in 10 minutes; record results.

Institute additional BP timing per specific order.

Once the aforementioned BP thresholds are achieved, repeat BP measurement every 10 minutes for 4 hours.

If either BP > threshold is, administer labetalol (40 mg IV over 2 minutes). If BP is below threshold, continue to monitor BP closely.

Repeat BP measurement in 10 minutes and record results.

If either BP > threshold is, administer labetalol (20 mg IV over 2 minutes). If BP is below threshold, continue to monitor BP closely.

Repeat BP measurement in 10 minutes and record results.

If either BP > threshold, administer hydralazine (10 mg IV over 2 minutes). If BP is below threshold, continue to monitor BP closely.

Repeat BP measurement in 20 minutes and record results.

If either BP > threshold, obtain emergency consultation from MFM, IM, anesthesia, or critical care specialists.

Close additional antihypertensive medication per specific order (Nicardipine).

Order Set for Severe Intrapartum or Postpartum Hypertension Initial First-Line Management with Labetalol®

1. Notify physician if systolic ≥ 160 mm Hg or if diastolic ≥ 110 mm Hg.
2. Institute fetal surveillance if undelivered fetus is viable.
3. Administer labetalol (20 mg IV over 2 minutes).
4. Repeat BP measurement in 10 minutes; record results.
5. If either BP > threshold, administer labetalol (40 mg IV over 2 minutes). If BP is below threshold, continue to monitor BP closely.
6. Repeat BP measurement in 10 minutes and record results.
7. If either BP > threshold is, administer labetalol (60 mg IV over 2 minutes). If BP is below threshold, continue to monitor BP closely.
8. Repeat BP measurement in 10 minutes and record results.
9. If either BP > threshold, administer hydralazine (10 mg IV over 2 minutes). If BP is below threshold, continue to monitor BP closely.
10. Repeat BP measurement in 20 minutes and record results.
11. If either BP > threshold, obtain emergency consultation from MFM, IM, anesthesia, or critical care specialists.
12. Close additional antihypertensive medication per specific order (Nicardipine).
13. Once the aforementioned BP-thresholds are achieved, repeat BP measurement every 10 minutes for 1 hour; then every 20 minutes for 1 hour; then every 30 minutes for 1 hour; and then every hour for 4 hours.
14. Institute additional BP timing per specific order.
Hypertensive Crisis Algorithm

Order Set for Severe IP or PP Hypertension
Initial First-Line Management with Hydralazine

1. Notify physician if systolic BP is greater than or equal to 160 mm Hg or if diastolic BP is greater than or equal to 110 mm Hg.
2. Institute fetal surveillance if undelivered and fetus is viable.
3. Administer hydralazine (5 mg or 10 mg IV over 2 minutes).
4. Repeat BP measurement in 20 minutes and record results.
5. If either BP threshold is still exceeded, administer hydralazine (10 mg IV over 2 minutes). If BP is below threshold, continue to monitor BP closely.
6. If either BP threshold is still exceeded, administer labetalol (20 mg IV over 2 minutes).
7. If either BP threshold is still exceeded, administer labetalol (20 mg IV over 2 minutes) and obtain emergency consultation from MFM, IM, anesthesia, or critical care specialists.
8. Give additional antihypertensive medication per specific order.
9. If the aforementioned BP thresholds are achieved, repeat BP measurement every 10 minutes for 1 hour, then every 15 minutes for 1 hour, then every 30 minutes for 1 hour, and then every hour for 4 hours.
10. Institute additional BP timing per specific order.

Hypertensive Crisis Algorithm

Box 2

12.
9.
8.
6.
5.
4.
3.
2.
1.

Hypertensive Crisis Algorithm

New 2015!

Hypertensive Crisis Algorithm

New 2015!
Hypertensive Medication Administration: Oral versus IV

- First line therapy recommendations for acute treatment of critically elevated BP in pregnant women (160/105-110) are either IV labetalol or IV hydralazine.
- If acute treatment needed in a patient without IV - oral nifedipine may be used (10 mg) and may be repeated in 30 minutes.
- PO (oral) nifedipine appears equally as efficacious as IV labetalol in correcting severe BP elevations.
- Oral labetalol would be expected to be less effective in acutely lowering the BP due to a slower onset to peak action; should be used only if oral nifedipine is not available in a patient without IV access.


Hypertensive Medication Administration: Oral versus IV

- IV Labetalol
  - Onset: 2-5 min
  - Peak: 5 min

- PO Labetalol:
  - Onset: 20 min-2 hrs
  - Peak: 1-4 hrs

- IV Hydralazine
  - Onset: 5-20 min
  - Peak: 15-30 min

- PO Nifedipine
  - Onset: 5-20 min* 
  - Peak: 30-60 min


http://www.epharmaconsult.com/pdf/2012/7/16/16843371776152


Hypertensive Medication Administration: Oral versus IV

- SHORTER Bars are “Better”

“First Line” Therapy

- Alternatives to consider:
  - Continuous intravenous infusion (pump) of Labetalol or Nicardipine
  - Minimal transplacental passage and changes in umbilical artery Doppler velocimetry have been noted

Nicardipine HCL

- Is a calcium ion influx inhibitor (slow channel blocker or calcium channel blocker).
- Produces significant decreases in systemic vascular resistance.
- Indicated for the short-term treatment of hypertension when oral therapy is not feasible or not desirable.
- Metabolized extensively by the liver - plasma concentrations are influenced by changes in hepatic function
- Contraindicated in patients with advanced aortic stenosis because of the reduced afterload. Reduction of diastolic pressure in these patients may worsen rather than improve myocardial oxygen balance.
- Pregnancy Category C
Nicardipine: Rapid Onset and Peak Action

<table>
<thead>
<tr>
<th>Drug</th>
<th>Half Life (time)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Labetalol</td>
<td>5.5 hours</td>
</tr>
<tr>
<td>Hydralazine</td>
<td>4 hours</td>
</tr>
<tr>
<td>Nicardipine*</td>
<td>2 to 5 minutes</td>
</tr>
<tr>
<td>Nifedipine</td>
<td>2 to 5 hours</td>
</tr>
</tbody>
</table>

*Contraindications to the use of nicardipine are hypersensitivity to nicardipine, severe aortic stenosis, hypotension, and shock.


Starting Dose and Titration

- **Non-pregnant patient:**
  - Starting dose 3 to 5 mg/hour
  - Increase rate by 2.5 mg/hour every 5 minutes to a maximum of 15 mg/hour

- **Pregnancy**
  - Starting dose 1 to 3 mg/hour
  - Increase by 0.5 to 1.0 mg/hour to maximum of 10 mg/hour until the target BP is reached


Maternal and Fetal/Neo Adverse Effects of Intravenous Nicardipine in 147 Patients

<table>
<thead>
<tr>
<th>Maternal</th>
<th>Fetal/Neonatal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Transient hypotension 8</td>
<td>Bradycardia 0</td>
</tr>
<tr>
<td>Nausea 3</td>
<td>Decelerations 2</td>
</tr>
<tr>
<td>Palpitations 3</td>
<td>Loss of variability 1</td>
</tr>
<tr>
<td>Headache 11</td>
<td>Preterm delivery 59</td>
</tr>
<tr>
<td>Flushing 8</td>
<td>Small for gestational age 24</td>
</tr>
<tr>
<td></td>
<td>Apgar score &lt;7 after 5 mins 3</td>
</tr>
</tbody>
</table>


Non-Responders: Sodium Nitroprusside (Nipride®)

“When Nothing Works . . .”

- Sodium nitroprusside should be reserved for extreme emergencies and used for the shortest amount of time possible.
- **Rationale/side effects:**
  - Cyanide and thiocyanate toxicity in the mother and fetus or newborn (monitor maternal levels during administration)
  - Increased intracranial pressure with potential worsening of cerebral edema in the mother.


HIGH RISK & CRITICAL CARE OB
A Forensic Case Studies Approach

Day One 8:00 AM to 4:00 PM

- **Background:** Distressing facts and stats on maternal deaths
- **Case Study:** “AP patient with severe hypotension unresponsive to fluid bolus”
- **Volume resuscitation quick “Rules” for treatment
- **Case Study:** “Post C-section patient with preeclampsia & new onset complaints of ‘tight chest’ and difficulty breathing”
- **Hypertension disorders**
  - Early recognition of pulmonary edema
  - Identification of HELLP,HELLP: Hemolysis, elevated liver enzymes, low platelet count
  - TTP: Thrombotic thrombocytopenic purpura
- **Case Study:** “IP patient with severe preeclampsia and hypertensive urgency”
- **Magnesium Sulfate: ‘High Alert Status’”
- **Magnesium Sulfate: Myths vs. Science
- **Case Study:** “IP patient with preterm hypertension: an option to magnesium”
- **Nicardipine ISM drug: New Directions”
- **Top 5 most common errors in prescribing antihypertensives

Post C-section patient with preeclampsia & new onset complaints of ‘tight chest’ and difficulty breathing
Post C/S fetal intolerance to labor

- L&D Recovery Room
- 27 year old, G1P1, delivered of a 33 2/7 weeks gestation, Dx: preeclampsia
- Cervidil, oxytocin induction
- EFM: Category I & II; persistent Category II with loss of accelerations; increasing FHR baseline
- Induction maternal blood pressures (BP): 145/88, 150/92, 142/80, 144/92, 157/90, 160/90

Preeclampsia Post C/S

- Operating Room I&O
  - EBL: 800 mL

<table>
<thead>
<tr>
<th>Intake</th>
<th>IV</th>
<th>Meds</th>
<th>Blood</th>
<th>TOTAL</th>
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<tbody>
<tr>
<td></td>
<td>2400</td>
<td>100</td>
<td>0</td>
<td>2500 mL</td>
</tr>
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<table>
<thead>
<tr>
<th>Output</th>
<th>Urine</th>
<th>EBL</th>
<th>Emesis</th>
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<tbody>
<tr>
<td></td>
<td>50</td>
<td>800</td>
<td>50</td>
<td>900 mL</td>
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</table>

L&D PACU – Page 1

<table>
<thead>
<tr>
<th>Time</th>
<th>BP</th>
<th>HR</th>
<th>RR</th>
<th>Temp</th>
<th>SaO2</th>
<th>UOP</th>
<th>Notes</th>
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</thead>
<tbody>
<tr>
<td>17:55</td>
<td>110/70</td>
<td>108</td>
<td>16</td>
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<td>.88</td>
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L&D PACU – Page 2

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</table>

Answer the following questions:

1. Why is the patient tachycardic?
2. Why is she tachypnic?
3. Why are her BP’s increasing?
4. Why is her SpO2 decreasing?
5. What other information do you need to answer the questions?
   - Assessments
   - Labs
   - Studies

What is your differential diagnosis at this point?
L&D PACU – Page 3

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Preeclampsia Post C/S

- Arterial Blood Gas (ABG)

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<th>Value</th>
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<td>7.39 – 7.45</td>
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<tr>
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<td>25 – 33</td>
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<tr>
<td>pO₂</td>
<td>68</td>
<td>92 – 107</td>
<td>mmHg</td>
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<tr>
<td>HCO₃</td>
<td>17</td>
<td>16 – 22</td>
<td>mEq/L</td>
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<tr>
<td>SaO₂</td>
<td>89</td>
<td>98-100 Percent (%)</td>
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Problem List

- Hypertension
- Uncompensated respiratory acidosis; loss of buffering capability; impending metabolic acidosis...
- Pulmonary insufficiency, respiratory compromise
- ? Heart failure
- ? Pulmonary edema
- ? End organ system derangements

Preeclampsia: Hemodynamics

- One pathway for hemodynamic alterations specific to preeclampsia is thought to begin with or shortly after implantation.
- Complex signaling of the abnormal placental vascular sites may also trigger increased maternal cardiac output at significantly higher volumes compared with normal pregnancy.

Hemodynamic Changes in Normal Pregnancy

- Early increase in CO results in a compensatory decreased in SVR, but likely exposes endothelial cells to sheering damage from flow.
- To compensate and protect end organs, the endothelial signal the arterial muscle cells to begin constricting to decrease sheering forces.
- Over time, the arterial constriction contributes to elevated SVR that ultimately decreases CO.
Hemodynamic Changes in Preeclampsia with High Output

Labs, Tests, Plan

- Auscultation of lungs (later sign)
- Chest x-ray (late sign)
- Renal function labs
- Chemistry
- CBC with differential
- Liver function labs
- Cardiac function (echo) and enzymes (R/O MI)

Urgent Actions

- Respiratory/pulmonary consult (stat)
- Bedside intubation and mechanical ventilation
- BP severely elevated at intubation -
  - Pink, frothy sputum when tube placed
  - Wide pulmonary shunt fraction
  - Decreased left ventricular contractility

Treatment and Outcome

- Non-cardiogenic pulmonary edema secondary to preeclampsia
- Mechanical ventilation x 3.5 days
- Slightly elevated cardiac enzymes; peaking in 1st 24 hours of intubation/cardiac failure
- Abnormal renal function – ATN; resolving prior to D/C on post partum day 11 (follow-up with nephrology)
- Echo WNL at D/C

Preeclampsia

- Abnormal vessels – stress
- Endothelial involvement
- Stimulation of inflammatory system – whole body
- Production of fibrin polymers, activation of fibrinolysis
- Perfusion challenges – mechanical/chemical/electrical
- Cellular consequences
- Organ system involvement
- Impaired oxygen transports and utilization

Physiology of Blood Pressure

Blood Pressure = Flow x Resistance

\[ \text{MAP} = \text{Cardiac Output (CO)} \times \text{Systemic Vascular Resistance (SVR)} \]
MAP = CO x SVR

- Elevated BP may be caused by
  - Increased CO and normal to low normal SVR
  - Increased SVR and decreased CO

**QUESTION:** How do you know which one your patient has?

**Measure SVR**

\[
SVR = \frac{MAP - CVP}{CO} \times 80
\]

**Example: BP = 170/88**

**"High CO, normal to low-normal SVR"**
- SAMPLE Pt #1
  - HR 100
  - RR 18
  - CVP 5 mmHg
  - PAP 26/10
  - PCOP 10
  - C.O. 8.7 L/minute
  - SVR - ???

**"High SVR, low CO"**
- SAMPLE Pt #2
  - HR 100
  - RR 18
  - CVP 5 mmHg
  - PAP 34/16
  - PCOP 16
  - C.O. 4.2 L/minute
  - SVR - ???

**What Type of Antihypertensive Drug Would Work BEST for Each Patient?**

**"High CO, normal to low-normal SVR"**
- SVR = ?
  - MAP = 115
  - \([\frac{MAP-CVP}{CO}] \times 80\) = 12.6 x 80
  - = 1012
  - SVR = 1012

**"High SVR, low CO"**
- SVR = ?
  - MAP = 115
  - \([\frac{MAP-CVP}{CO}] \times 80\) = 26.2 x 80
  - = 2095
  - SVR = 2095

**SVR**

**"High CO, normal to low-normal SVR"**
- SVR = ?
  - MAP = 115
  - \([\frac{MAP-CVP}{CO}] \times 80\) = 12.6 x 80
  - = 1012
  - SVR = 1012

**"High SVR, low CO"**
- SVR = ?
  - MAP = 115
  - \([\frac{MAP-CVP}{CO}] \times 80\) = 26.2 x 80
  - = 2095
  - SVR = 2095

**Labetalol v. Hydralazine?**
Summary