Assessment of Shortness of Breath in Pregnancy

T. Lee-Ann Hawkins, MD, MSc., FRCPC
Department of Medicine, University of Calgary
“Medical Disorders in Pregnancy”
No disclosures.
Objectives:

• recognize the hemodynamic and respiratory changes of pregnancy that impact symptomatology, physical examination findings and laboratory parameters

• develop a comprehensive approach to the late-gestation pregnant patient who presents with shortness of breath

• demonstrate knowledge of an appropriate evaluation and management plan for respiratory symptoms in pregnancy

• recognize the importance of collaborative care in the obstetrical patient
Case Presentation:

- 30-year old G₁P₀ with twin pregnancy at 36 weeks gestation presents short of breath over the past 2 hours
- Previously well.
- Uncomplicated pregnancy.
- Vitals: HR 110 RR 25
  BP 160/100 Sat 98% on 5 L
Case Presentation:

- 30-year old G1P0 with twin pregnancy at 36 weeks gestation presents short of breath over the past 2 hours.
- Previously well.
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Pregnancy Physiology

- Cardiac
  - Blood volume
  - Cardiac output
  - Blood pressure

- Pulmonary
  - Anatomical
  - Ventilatory

- Further physiological changes associated with labour and delivery
Cardiac Physiology

- **Blood Volume**
  - Increased ~50% over pregnancy
  - Changes start as early as 6 weeks gestation
  - Rapid increase until mid-gestation
  - Peak and plateau at ~32 weeks gestation

- **Cardiac Output**
  - Increased ~40% over pregnancy
  - Due to both increased heart rate and stroke volume
Cardiac Physiology

- Blood Pressure
  - Mean pressure decreases 10-15 mmHg; diastolic > systolic
  - Begins in the 1\textsuperscript{st} trimester, nadir in 2\textsuperscript{nd} trimester and return to baseline near term
  - Due to decreased systemic vascular resistance
Pulmonary Physiology

• Anatomic changes:
  • Upper airway hyperemia, increased edema and friability (increased nasal congestion, snoring, difficult airway)
  • Increased A-P diameter, increased circumference of chest
  • Elevation of diaphragm (excursion unchanged)

• Minute ventilation (gas exchange/1 min) is increased; up to 50% near term
  • Progesterone as a respiratory stimulant
  • Increased basal metabolic rate, increased O₂ consumption
Pulmonary Physiology

Non-pregnant

Pregnant

AARC. Respir Care 2001; 46:531–539.

ERV

RV

<ERV

<RV
Pulmonary Physiology

- Spirometry and peak flow assessment are unchanged by pregnancy
- No increase in respiratory rate but an increase in tidal volume
- Increase in minute ventilation leads to lowering of the maternal PaCO₂ and a mild respiratory alkalosis

<table>
<thead>
<tr>
<th></th>
<th>Non-pregnant</th>
<th>Pregnant</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>7.38-7.44</td>
<td>7.40-7.45</td>
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<tr>
<td>PaO₂</td>
<td>80-100</td>
<td>100-105</td>
</tr>
<tr>
<td>PaCO₂</td>
<td>35-45</td>
<td>28-32</td>
</tr>
<tr>
<td>HCO₃⁻</td>
<td>21-28</td>
<td>24-30</td>
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Labour & Delivery

- Cardiac output increased ~35% between contractions and a further 15-20% during them
- Both heart rate and blood pressure increase during contractions
- 300-500 mL of blood is “autotransfused” with each contraction
- Cardiac output increases 60-80% immediately postpartum then rapidly declines to antepartum levels
Implications on Examination

- Hyperventilation (tidal volume, not respiratory rate)
- Pulmonary crackles due to atelectasis
- Hyperdynamic circulation (bounding pulses)
- Prominent but not elevated JVP
- Brisk, sustained, displaced apex
- Loud S1, S3 (84%), grade I-II early mid-systolic ejection-type murmur (96%)
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• Uncomplicated pregnancy.

• Vitals: HR 110 RR 25
  BP 160/100 Sat 98% on 5 L
Case Presentation:

- Gradual onset of dyspnea over the past 2 hours, no chest pain
- No leg or groin swelling. No prior history of VTE.
- No infectious symptoms.
- No headache, visual changes, RUQ pain, nausea or vomiting.
- Medications: multivitamin, Vitamin D 2000 U. NKDA
- Non-smoker, non drinker. Born in Canada. Married.
Case Presentation:

Focused Examination:

- 2-3 word sentences and accessory muscle use
- JVP 5 cm. Normal heart sounds. Grad 2/6 mid-systolic murmur at RUSB. Brisk peripheral pulses.
- Decreased air entry to bases with coarse crackles bilaterally. No stridor or wheeze.
- Abdomen benign
- No edema. 4+ reflexes with 2 beats of clonus bilaterally.
Case Presentation:

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<tr>
<th>ABG</th>
<th></th>
<th>Electrolytes</th>
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<th>Creatinine</th>
<th></th>
<th>CBC</th>
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<tbody>
<tr>
<td>pH</td>
<td>7.33</td>
<td>Na</td>
<td>135</td>
<td>95</td>
<td>(prior 50)</td>
<td>Hg</td>
<td>151</td>
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<tr>
<td>PaCO₂</td>
<td>33</td>
<td>K</td>
<td>4.1</td>
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<td></td>
<td>Plts</td>
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<tr>
<td>PaO₂</td>
<td>60</td>
<td>Cl</td>
<td>104</td>
<td></td>
<td></td>
<td>WBC</td>
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<tr>
<td>lactate</td>
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<td>CO₂</td>
<td>24</td>
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<td></td>
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**Case Presentation:**

<table>
<thead>
<tr>
<th>Test</th>
<th>Value</th>
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<tbody>
<tr>
<td>INR</td>
<td>1.1</td>
</tr>
<tr>
<td>PTT</td>
<td>26.8</td>
</tr>
<tr>
<td>Total Bili</td>
<td>2</td>
</tr>
<tr>
<td>ALP</td>
<td>79</td>
</tr>
<tr>
<td>ALT</td>
<td>13</td>
</tr>
<tr>
<td>GGT</td>
<td>40</td>
</tr>
<tr>
<td>Lipase</td>
<td>30</td>
</tr>
<tr>
<td>LDH</td>
<td>175</td>
</tr>
<tr>
<td>Urate</td>
<td>607</td>
</tr>
</tbody>
</table>

24-hour urine protein 1.52g
- Vent rate: 114 BPM
- PR interval: 160 ms
- QRS duration: 88 ms
- QTc: 320/441 ms
- P-R-T axes: 57 32 15

Sinus tachycardia
Otherwise normal ECG

COMMENTS:

Referred by: 

Confirmed by:

25mm/s 10mm/mV 100Hz 7.1.1 12SL 237 CID: 12
SID: 795381200 EID: 1028 EDT: 10:13 01-OCT-2011 ORDER: 001PLCVFF ACCOUNT: 100035861295
Chest X-Ray Report:

FRONTAL CHEST

COMPARISON STUDY: None.

FINDINGS:

Bilateral patchy parenchymal opacities are seen worse on the left than right. There is involvement of the left upper and lower lobes with only involvement of the right lower lung. The appearance of this is nonspecific but may represent asymmetric pulmonary edema. In the appropriate clinical setting pneumonia is also a possibility.

Jason Kam Wong, MD, FRCPC/cp
Dictated but not read.

Final Report
## Pulmonary Edema in Pregnancy

<table>
<thead>
<tr>
<th>Category</th>
<th>Causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pregnancy associated pulmonary edema</td>
<td>- Preeclampsia/eclampsia&lt;br&gt;- Sepsis&lt;br&gt;- Anemia &amp; fluid overload&lt;br&gt;- Amniotic air embolism</td>
</tr>
<tr>
<td>Congestive Heart Failure</td>
<td>- Cardiomyopathy&lt;br&gt;- Valvular/ischemic/congenital heart disease</td>
</tr>
<tr>
<td>Thromboembolism</td>
<td></td>
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<tr>
<td>Pneumonia</td>
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<tr>
<td>Aspiration Pneumonitis</td>
<td></td>
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<tr>
<td>Transfusion-related</td>
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<tr>
<td>Venous Air Embolism</td>
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<tr>
<td>Anaphylaxis</td>
<td></td>
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<tr>
<td>Drug-related</td>
<td></td>
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<tr>
<td>Asthma</td>
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</tbody>
</table>
Physiology that Predisposes:

- 20% decrease in colloid osmotic pressure
- 50% increase in blood volume
- 40% increase in cardiac output
- decreased functional residual capacity

These changes make pregnant women susceptible but do not cause pulmonary edema.
Case Diagnosis?

- 30-year old G₁P₀ with twin pregnancy at 36 weeks gestation presents short of breath over the past 2 hours
- BP 160/100
- 98% on 5L with pulmonary edema
- Evidence of hyperreflexia
- Renal dysfunction (Cr 95), hyperuricemia (Urate 607), proteinuria (24hr urine protein 1.52 g)
Case Diagnosis?

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PREECLAMPSIA
Preeclampsia is more than hypertension!
Multisystem disorder unique to pregnancy
Cause and pathophysiology unknown and under intense investigation
Related to placental dysfunction with potential for severe maternal and fetal effects
Preeclampsia

Roberts JM & Hubel CA. Placenta. 2009:30;S32-37
in preeclampsia, invasion of maternal spiral arteries is shallow and results in small-caliber resistance vessels which may result in placental ischemia

= mismatch between uteroplacental supply and fetal demand

Preeclampsia & Pulmonary Edema

- disruption of normal endothelial barrier allows fluid to escape into alveoli and interstitium
- aggravated by reduced maternal plasma colloid osmotic pressure
- cardiac involvement via sudden increase in afterload caused by intense vasospasm & relative left ventricular systolic dysfunction
- Occurs in up to 3% of preeclamptic pregnancies
# Preeclampsia Risk Factors

<table>
<thead>
<tr>
<th><strong>Maternal</strong></th>
<th><strong>Maternal Medical</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>- primiparity</td>
<td>- chronic hypertension</td>
</tr>
<tr>
<td>- past history</td>
<td>- diabetes (higher if complications)</td>
</tr>
<tr>
<td>- family history</td>
<td>- renal disease</td>
</tr>
<tr>
<td>- &gt;4 cups coffee/day</td>
<td>- SLE, CTD</td>
</tr>
<tr>
<td>- Black</td>
<td>- obesity</td>
</tr>
<tr>
<td>- low maternal birthweight</td>
<td>- thrombophilia</td>
</tr>
<tr>
<td></td>
<td>- insulin resistance &amp; obesity</td>
</tr>
<tr>
<td><strong>Fetal</strong></td>
<td><strong>Paternal</strong></td>
</tr>
<tr>
<td>- multiple gestation</td>
<td>- prior preeclamptic pregnancy</td>
</tr>
<tr>
<td>- hydrops fetalis</td>
<td>- strong family history HDP</td>
</tr>
<tr>
<td>- gestational trophoblastic disease</td>
<td>- primipaternity</td>
</tr>
<tr>
<td>- triploidy, other genetic syndromes</td>
<td></td>
</tr>
</tbody>
</table>
Onset of hypertension (new or worsening) after 20 weeks gestation with at least one of:

- new or worsening proteinuria
- resistant hypertension
- one or more adverse conditions (maternal or fetal)
Preeclampsia - Proteinuria

- all pregnant women should be assessed for proteinuria
  - moderate/high suspicion: 24h *or* protein to creatinine ratio (PCr)
- Proteinuria defined as:
  - $\geq 0.3 \text{ g/d in a 24h urine sample}$
  - $\geq 30 \text{ mg/mmol in a random PCr}$
  - no conversion from PCr to 24h urine in pregnancy
“Severe” Preeclampsia:

- presentation prior to 34 weeks gestation
- heavy proteinuria (3-5 g/day)
- one or more adverse conditions:
  - CNS (visual disturbances, decreased consciousness, CVA, persistent headache)
  - RUQ pain, liver enzymes >2x normal
  - thrombocytopenia <100
  - pulmonary edema
  - placental abruption, fetal loss, severe IUGR
Management of Preeclampsia:

- preeclampsia = admission
- minimal to no IV fluids
- establish control of blood pressure to <160/110 or if symptomatic, 10-15% lower than presentation but be mindful of maintaining adequate diastolic pressure (fetal perfusion)
- preference is towards the use of oral antihypertensives as soon as feasible
Antihypertensive Use in Pregnancy

• For BP ≥160/110, start with:
  • Labetalol 20 mg IV, repeat q30min or 1-2mg/min
  • Nifedipine 5-10 mg capsule (bite and swallow) q30min
  • Hydralazine 5 mg IV, repeat 5-10 mg q30 min

• When able, the following PO medications:
  • Methyldopa 250-500 mg PO bid-qid (max 2g/day)
  • Labetalol 100-400 mg PO bid-tid (max 1200mg/day)
  • Nifedipine XL 20-60 mg PO bid (max 120 mg/day)
Preeclampsia & Pulmonary Edema

- goal $O_2$ saturation $>96\%$ to ensure fetal oxygenation
- iv lasix in small quantities might prove useful
- goal is to stabilize, control BP and prepare for delivery with OB & Anesthesia input!
- mortality risk: 10\% maternal & 50\% fetal
- majority improve within 24 hours
Magnesium Sulphate?

- Indicated for treatment of eclampsia
- Indicated for eclampsia prophylaxis with “severe” preeclampsia
  - Evidence of neurotoxicity
  - HELLP syndrome
  - Neuroprotection of pre-term infants (<32 weeks)
- Dose: 4-6 g bolus followed by 1-2 g per hour x 24-48 hours (regional variability)
- Mechanism of action not well established.
Summary

• Pulmonary edema is a serious but infrequent consequence of preeclampsia.

• The internists role should be to rapidly recognize this complication of preeclampsia, to support the patient with minimal fluids and diuretics if able, to stabilize the blood pressure, and to alert the Obstetrics team such that a delivery decision can be made.

• Patients with preeclampsia benefit from IM care.