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Edited by Mary E. D'Alton, Errol Norwitz and Thomas F. McElrath  
Excerpt

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## ACUTE ASTHMA EXACERBATION

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### BACKGROUND

- 4% of adult population has asthma
- In general, sx often worsen during 28–36 wk of pregnancy; acute exacerbations rare in last 4 wk or in labor

### DIAGNOSIS

#### History

- Sx include cough, dyspnea, wheezing; fever, chills, malaise less common
- Ask about prior attacks, current Rx, baseline peak expiratory flow rate (PEFR), precipitating events (upper respiratory tract infection, allergen exposure)

#### Physical examination

- Check temp, pulse oximetry (note: pulse oximetry does *not* assess pt's ability to clear CO<sub>2</sub>)
- Examine for cyanosis, hyperinflation, use of accessory muscles, pulsus paradoxus

#### Diagnostic tests

- *Laboratory tests:* ABG, CBC
- *Specific diagnostic tests:* decrease in PEFR, FEV1
- *Imaging tests:* AP/lateral CXR
- *Screening tests:* daily PEFR

### DIFFERENTIAL DIAGNOSIS

- Pulmonary edema
- Pulmonary embolism
- Bronchitis
- Pneumonia

### COMPLICATIONS

- *Maternal complications:* respiratory failure, preterm birth

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## 2 Acute Asthma Exacerbation

- *Fetal complications:* prematurity, low birthweight, increased perinatal mortality (esp. w/ severe disease)

### PROGNOSIS

- Pregnancy represents a state of compensated respiratory alkalosis; maternal  $\text{PCO}_2 \geq 35$  mmHg in room air suggests impending respiratory failure

### MANAGEMENT

#### General measures

- $\text{O}_2$  supplementation to maintain  $\text{O}_2$  saturation  $\geq 95\%$ ,  $\text{PO}_2 \geq 70\%$
- Continuous pulse oximetry to follow oxygenation
- Adequate hydration
- Serial ABGs

#### Specific treatment

- Inhaled beta-2-agonist (bronchodilator) Rx q 20–30 min  $\times$  3 doses
- If initial response adequate (ie, increase in PEFR to  $\geq 70\%$  predicted or baseline, if known), continue bronchodilator Rx & follow as outpatient
- If response inadequate, continue Rx for 2–3 h; consider admission for measured PEFR  $< 70\%$  predicted
- Consider IV corticosteroid Rx for PEFR 40–70% baseline (or predicted) after 2–3 h
- Stress-dose steroids (IV hydrocortisone 80 mg q8h) in labor if history of steroid Rx in last 6 mo

#### Contraindications

- Cardiogenic disease relative contraindication to beta-agonist Rx

#### Side effects & complications of treatment

- Maternal adrenal suppression (can be avoided w/ stress-dose steroids in labor)

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Acute Asthma Exacerbation

Acute Cystitis

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### Follow-up care

- Regular outpatient visits
- Referral to pulmonologist

### SUBSEQUENT MANAGEMENT

- Severity & frequency of acute exacerbations similar in subsequent pregnancies

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## ACUTE CYSTITIS

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### BACKGROUND

- Most common medical complaint of pregnancy
- Incidence: 1–4% of all pregnancies
- Organisms: *Escherichia coli* (90%), *Staphylococcus saprophyticus* (4–7%)

### DIAGNOSIS

#### History

- Sx may include frequency, dysuria, urgency, suprapubic pain
- *Risk factors*: diabetes, urinary tract anomaly, prior urinary tract infection/pyelonephritis in index pregnancy, sickle cell trait/disease

#### Physical examination

- Suprapubic tenderness
- Flank pain, costovertebral angle tenderness, fever, systemic complaints usually absent

#### Diagnostic tests

- Urine dip can be positive for nitrates, leukocyte esterase
- Definitive Dx made by urinalysis ( $\geq 100,000$  colony-forming units/mL of single pathogenic organism in midstream clean-catch urine specimen)
- Imaging studies not indicated
- Check CBC if patient febrile

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#### 4 Acute Cystitis

##### **DIFFERENTIAL DIAGNOSIS**

- Mycotic/bacterial vaginosis w/ contamination of urine specimen
- Asymptomatic bacteriuria
- Pyelonephritis

##### **COMPLICATIONS**

- *Maternal complications:* progression to pyelonephritis, urosepsis, ARDS, preterm labor
- *Fetal complications:* preterm birth, low birthweight

##### **PROGNOSIS**

- Full resolution can be expected w/ adequate Rx; increased risk of pyelonephritis/urosepsis if Rx inadequate
- Screening/treatment prevents 80% of pyelonephritis in pregnancy

##### **MANAGEMENT**

###### **General measures**

- Aggressive oral hydration
- Outpatient Rx acceptable in absence of pyelonephritis

###### **Specific treatment**

- Antibiotic Rx for 3 d adequate for otherwise healthy women (consider 5-d course for women w/ concurrent chronic disease); single-dose Rx assoc. w/ increased failure rate in pregnancy
- Rx options include trimethoprim/sulfamethoxazole 160/180 mg po bid, nitrofurantoin monohydrate/macrocrystals 100 mg po bid, cephalexin 500 mg po qid
- Adjust Rx according to culture results, if indicated

###### **Prevention**

- Periodic screening urinalysis in women at high risk for urinary infections

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Acute Cystitis

Amniotic Fluid Embolism

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### SUBSEQUENT MANAGEMENT

- Repeat urine culture in 10 d after completion of Rx (“test of cure”)
- If Rx unsuccessful, consider noncompliance, failed Rx (poor antibiotic selection, antibiotic resistance)
- Consider suppressive Rx for 6 wk if repeat culture positive w/ same organism.

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## AMNIOTIC FLUID EMBOLISM

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### BACKGROUND

- Rare, unpredictable, catastrophic obstetric event
- 10% of maternal mortality in U.S.
- *Incidence:* 1/8,000–1/85,000 births

### DIAGNOSIS

#### History

- Prodromal sx may include sudden chills, sweating, anxiety
- *Risk factors:* multiparity, advanced maternal age, hypertonic labor, male fetus, intrauterine fetal demise, oxytocin, amniotomy, abruption, intrauterine pressure catheter, chorioamnionitis, cesarean, preeclampsia, intrauterine saline injection (abortion)

#### Physical examination

- *Clinical:* Dx characterized by acute-onset respiratory distress, cyanosis, hypotension, tachycardia, hypoxemia, neurologic manifestations (seizures, coma), hemorrhage in labor/delivery or early puerperium

#### Diagnostic tests

- *Laboratory tests:* check CBC, DIC panel
- *Specific diagnostic tests:* clinical Dx, identification of amniotic fluid (mucin, fetal squames) in pulmonary vasculature at postmortem not pathognomonic

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## 6 Amniotic Fluid Embolism

- *Imaging*: check CXR, V/Q scan (shows decreased perfusion); of little value in acute setting. Transesophageal ultrasound useful in acute assessment of pulmonary embolism in intubated pt.

### DIFFERENTIAL DIAGNOSIS

- Pulmonary embolism
- Pulmonary edema
- Venous air embolism (assoc w/ ruptured uterus, placenta previa, persistent atrial septal defect)
- Aspiration
- Eclampsia
- Drug overdose/withdrawal
- Other causes of DIC

### COMPLICATIONS

- *Maternal complications*: shock, DIC, blood transfusion; very high maternal mortality rate (60–90%), permanent neurologic sequelae (85% of survivors)
- *Fetal complications*: intrauterine fetal demise, hypoxic ischemic cerebral injury if fetus undelivered

### PROGNOSIS

- Death not inevitable if early Dx, aggressive management, including intubation and possible pulmonary bypass

### MANAGEMENT

#### General measures

- High index of suspicion, early Dx
- Monitor vital signs, O<sub>2</sub>
- Anesthesia consult, central hemodynamic monitoring, IV access
- Immediate delivery regardless of gestational age
- Rx primarily supportive

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### Specific treatment

- CPR, Rx hypoxemia (supplemental O<sub>2</sub>, mechanical ventilation)
- Control bleeding (correct DIC, uterotonic Rx)
- Correct anemia/coagulopathy w/ aggressive blood product transfusion
- Maintain arterial PO<sub>2</sub> >60 mmHg, O<sub>2</sub> saturation >90%; Rx bronchospasm (terbutaline, aminophylline, ? steroids)
- Maintain SBP >90 mmHg, urine output >25 mL/h; inotropic support (dopamine) as needed

### Contraindications

- Regional anesthesia contraindicated in acute setting; general endotracheal anesthesia for cesarean
  - Airway management crucial and intubation highly likely
  - Pressor support likely to be acutely needed
  - May require pulmonary bypass
- Avoid heparin in established DIC

### SUBSEQUENT MANAGEMENT

- Recurrence rate not clear, likely low.

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## ANTENATAL FETAL TESTING

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### BACKGROUND

- Goal: early identification of fetus at risk for preventable morbidity due to hypoxemia
- Assumptions: (1) hypoxemia leads to permanent injury; (2) tests discriminate between asphyxiated, nonasphyxiated fetuses; (3) early detection can prevent adverse outcome
- At most, 15% of cerebral palsy due to intrapartum hypoxemia

### DIAGNOSIS

#### History

Indications for testing:

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## 8 Antenatal Fetal Testing

1. *Maternal factors*: diabetes, hypertension, hyperthyroidism
2. *Fetal factors*: intrauterine growth restriction, increased fetal activity, oligo/polyhydramnios
3. *Pregnancy-associated*: placental abruption, postterm pregnancy

### Physical examination

- Usually unhelpful

### Diagnostic tests

1. *Fetal movement charts* (“kick counts”): count all movements in 1 h or count time for 10 kicks; 2–3 times/d; any decreased movement requires further evaluation
2. *Contraction stress test* (CST): measures response of fetal heart rate to contractions (3/10 min required to interpret test); (+) CST defined as decelerations w/  $\geq 50\%$  contractions
3. *Nonstress test* (NST): changes in fetal heart rate pattern w/ time; reflects maturity of fetal autonomic nervous system; absence of reactivity (2 accelerations of 15 bpm  $\times$  15 sec in 20 min) depends on gestational age: 50% at 24–28 wk, 15% at 28–32 wk
4. *Biophysical Profile* (BPP): NST + 4 sonographic variables: breathing  $\geq 30$  sec/30 min, movements  $\geq 3/30$  min, tone (flexion/extension)  $\geq 1/30$  min, amniotic fluid volume  $\geq 2$  cm single vertical pocket

### DIFFERENTIAL DIAGNOSIS

Causes of irreversible cerebral injury other than hypoxia:

- Congenital abnormalities
- Intracerebral hemorrhage
- Infection
- Drugs
- Trauma
- Hypotension
- Metabolic (thyroid, hypoglycemia)



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### COMPLICATIONS

- *Maternal complications:* increased cesarean delivery rate
- *Fetal complications:* iatrogenic prematurity due to false-positive testing

### PROGNOSIS

- Negative predictive value (intrauterine fetal demise < 1 wk following (-)/reassuring testing) consistent for all tests at 0.3–1.9/1,000 pregnancies
- Positive predictive value varies widely; severely abnormal fetal testing associated w/ adverse outcome in only 25–40% of cases
- Interpret testing in light of gestational age, underlying clinical risk factors, congenital anomalies

### MANAGEMENT

#### General measures

- All antenatal tests probably equally efficacious

#### Contraindications

- Contraindications to CST: preterm premature rupture of membranes, previa, preterm labor, prior cesarean

### SUBSEQUENT MANAGEMENT

- Specific to suspected pathology.

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## ANTIPHOSPHOLIPID ANTIBODY SYNDROME

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### BACKGROUND

- Autoimmune disorder characterized by circulating antibodies against membrane phospholipid & one or more specific clinical syndromes
- Incidence depends on population screened (0.5–3% of non-pregnant, 2–4% of pregnant, & 4–5% of women w/ prior pregnancy loss have low-titer anticardiolipin antibody [ACA] IgG;

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## 10 Antiphospholipid Antibody Syndrome

among women w/ recurrent pregnancy loss, 5–20% have moderate to high titer ACA, & 5–10% are + for lupus anticoagulant [LAC])

### DIAGNOSIS

Two elements are required for Dx:

#### 1. **Appropriate clinical setting:**

- Recurrent pregnancy loss
  - Unexplained thrombosis
  - Autoimmune thrombocytopenia
  - ? Preeclampsia
  - ? Intrauterine growth restriction
- AND

#### 2. **A confirmatory serologic test:**

- LAC is an unidentified antibody causing increases of phospholipid-dependent coagulation tests (aPTT, Russel Viper Venom test) by binding to prothrombin-activator complex; in vivo, LAC causes thrombosis; LAC results reported as present or absent (no titers)
- Specific antiphospholipid antibodies measured by ELISA (most commonly ACA) assoc. w/ anticoagulant activity in vitro but procoagulant activity in vivo; ACA IgM alone &/or low-positive IgG may be nonspecific; moderate to high levels of ACA IgG required for Dx
- ACA & LAC similar but not identical antibodies; may coexist in vivo (70–80% of women w/ LAC are ACA (+); 10–30% of ACA (+) women have LAC)
- False-positive test for syphilis common but not sufficient to make Dx of antiphospholipid antibody syndrome (APS)

### DIFFERENTIAL DIAGNOSIS

- SLE (10–30% of women w/ SLE have antiphospholipid antibodies; 60–90% of women w/ APS are ANA (+) but w/ insufficient criteria for Dx of SLE)
- Other causes of thrombocytopenia