OBSTETRIC CATASTROPHES

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Lecture Organization

- Embolism
  - Pulmonary Embolism
  - Amniotic Fluid Embolism
- Eclampsia
- Hypertensive Crisis
- Local Anesthetic Toxicity
- Cardiopulmonary Arrest

Pulmonary Embolism

Pulmonary embolism, along with amniotic fluid embolism, accounts for the leading cause of maternal mortality in the United States (Koonin, et al; 1989 MMWR)

Virchow's Triad:

- Stasis
- Hypercoagulability
- Vascular Damage

DVT: Key Facts

- 40% of asymptomatic patients with DVT have radiographically documented pulmonary embolism
- DVT of pelvic venous system is often an asymptomatic condition until clinical pulmonary embolism develops
- Untreated pulmonary embolism mortality is up to 30%. Treated mortality is 3%

(Moser et al, 1994; Cunningham et al, 1997; Toglia & Weg, 1996)

Diagnosis of Pulmonary Embolism

- Chest X-ray
- ECG
- Arterial blood gas
- D-dimer
- Ventilation-perfusion scintography
- Angiography
- Thoracic enhanced CT
- Extremity Doppler

Chest X-Ray Findings in PE:

- ***Hampton's Hump*:**

  pleural based density at CPJ
- ***Westermark's Sign*:**

  peripheral aligemia with proximal vessel dilatation
- ***Most <u>common</u> finding is normal X-Ray (30%)!***

ECG Changes in PE:

- **p-pulmonale, RBBB, RAD**
- **S1 Q3 T3**
- **New Onset A-Fib**
- ***Most common finding is normal (or sinus tach) ECG***

Arterial blood Gas (ABG) in PE:

- **Hypoxemia typical of moderate to large PE (from shunt and V/Q mismatching**
- **Room air PaO2 > 85 mmHg is reassuring- 15% of angiographically detectable PE's have room air PaO2 greater than 85 mmHg.**
- **Hyperventilation of pregnancy confounding as both overlay of clinical presentation (pulmonary embolism presents with hyperventilation) and in the interpretation of PaO2 (lower alveolar PCO2 produces a higher PAO2 for a given FIO2**
- **Oxygen administration before ABG analysis confuses clinical picture!**

(Robin, 1977; Phelan, 1997)

D-Dimer in the Diagnosis of Pulmonary Embolism

- D-Dimer testing measures level of specific fibrin degradation product
- Some studies suggest that D-dimer is elevated in patients with deep venous thrombosis
- Data on non-pregnant patients is mixed - best results show high sensitivity and low specificity (good screening test?). Others show less favorable results
- Pregnancy associated changes in hemostatic system may make D-dimer less reliable during pregnancy

(Ginsburg et al, 1998; Kutinsky et al, 1999)

Etiology of PE:

- **Often from proximal lower extremity clots (i.e. above knee)**
- **Clots in other locations are not unheard of, especially in pregnancy!**

  1/2 of pulmonary emboli during pregnancy arise from pelvic veins

(Erdman, 1990; Williams OB, 1993; Dunmire, 1989)

Radiographic Diagnosis of Pulmonary Embolism During Pregnancy:

- Ventilation/Perfusion (V/Q) Scanning
- Pulmonary Angiography
- Spiral/Helical CT

V/Q Scintography:

- Q = Technetium 99m (16 mRad)
- V = Xenon 133 (10 mRad)
- Interpretation of results:

  Normal =no perfusion defects

  High Probability

  (> 85% PE with mismatched defect)

  2 Defects- segmental or greater

V/Q Continued...

- Intermediate Probability
- Low Probability
- Indeterminate

→ Segmental or subsegmental defects with or w/out vent. defects

Statistics- Normal and High Probability scans are very predictive. Intermediate and low probability scans are not accurate. *4% of those with low, indeterminate* and normal scans have emboli!
(PIOPED [Natl heart, Lung, and Blood inst.] trial, 1990)

V/Q Continued:

Bottom Line

If the *a priori* risk of PE is high, and the V/Q is indeterminate, low or moderate - consider either treatment (or angiography) anyway

V/Q is useful because -

If *a priori* low and scan normal, then PE not likely

High Probability is predictive of PE

Selective angiography can be used on suspicious V/Q areas - limiting dye use

Test has scant morbidity and 0 mortality

Lower Extremity Doppler- PE

- If lower extremity DVT is identified, then issue to anticoagulate is settled (DVT is treated by anticoagulation)
- Absence of lower extremity DVT does not exclude PE in non-pregnant patients
- Pregnancy has a higher rate of non lower extremity thrombosis - lower extremity DVT probably even less useful

Pulmonary Angiography in Dx of PE in Pregnancy:

- "Gold Standard" for diagnosis (95+% sensitivity and specificity)
- Invasive

  1-4% risk of arrest

  Mortality 0.1 - 0.4%
- Selective angiography may be performed (when one embolic site found, test terminated)
- Radiation exposure risks (and "pseudorisks") consideration in pregnancy

Helical-Spiral CT

- Technique uses high-speed contrast enhanced thoracic CT
- Preliminary results- great promise
- Very operator (radiologist) dependent
- Fairly specific (90-95%), but only moderately sensitive (50-60%)
- Best presently used as a confirmatory rather than a screening test

(Garg et al, 1998; Drucker et al, 1998)

Treatment- Pulmonary Embolism in Pregnancy

- Anticoagulation is mainstay of pharmacotherapy
- Supportive care should not be forgotten during the rush to diagnose and treat
- Likelihood of repeat events very high after initial embolism. Risk is not eliminated, even with anticoagulation.

Heparin

- Molecular Weight-15,000 d (4000 d if LMW Heparin)
- Activates AT III
- Increased fetal M/M reported-probably due to M/M in patients that was due to their underlying diseases requiring Heparin - not the Heparin itself

(Hall, 1980. Refuted by Ginsburg, 1989)

Heparin Continued (2)

- **Antidote - Protamine sulfate**

  1mg protamine reverses 1mg (approx 100 units heparin)

- **Complications**

  bleeding

  thrombocytopenia

  Osteoporosis (risk 1/50?? with prolonged use dose related effect argued [.20k/day for > 6 mos)

  (Rayburn, 1992; Hirsch, 1991)

Heparin Cont. (3)

Dosage- Acute Tx of Pulmonary Embolism

Intravenous- 5-10,000 unit bolus (65-75 U/kg)- followed by 1000 U/hr

(20,000 U/Liter = 1000 U/hr at 50 cc/hr)

Therapeutic Goal- APTT 1.5-2.5 X Control

Heparin Cont (4)

IV Heparin is continued for 7-10 days

Afterwards, conversion to SQ dosing

10-20,000 U q 8-12 hrs to maintain APTT *at middose* 1.5-2.0 X Control

If Postpartum, may convert to Coumadin from IV Heparin

(Williams OB, 1993, Hirsch, 1991 and others)

Low Molecular Weight Heparin

- More selectively inhibits X to Xa conversion
- Less theoretical risk of spontaneous bleeding
- When lower risk “abused”, reports of spontaneous epidural hematomas noted
- With proper use, may be better (albeit expensive) alternative to unfractionated heparin
- Use and indications should be equivalent to unfractionated heparin

(ACOG, 1998 and others)

Low Molecular Weight Heparin (2)

- Anticoagulation dose = 1 mg/kg every 12 hours in non-pregnant subjects
- Optimal dose in pregnancy not known
- Must monitor factor anti Xa effect (0.4-1.0 U/mL 3-4 hours after injection)
- aPTT will NOT be prolonged
- Large trials pending

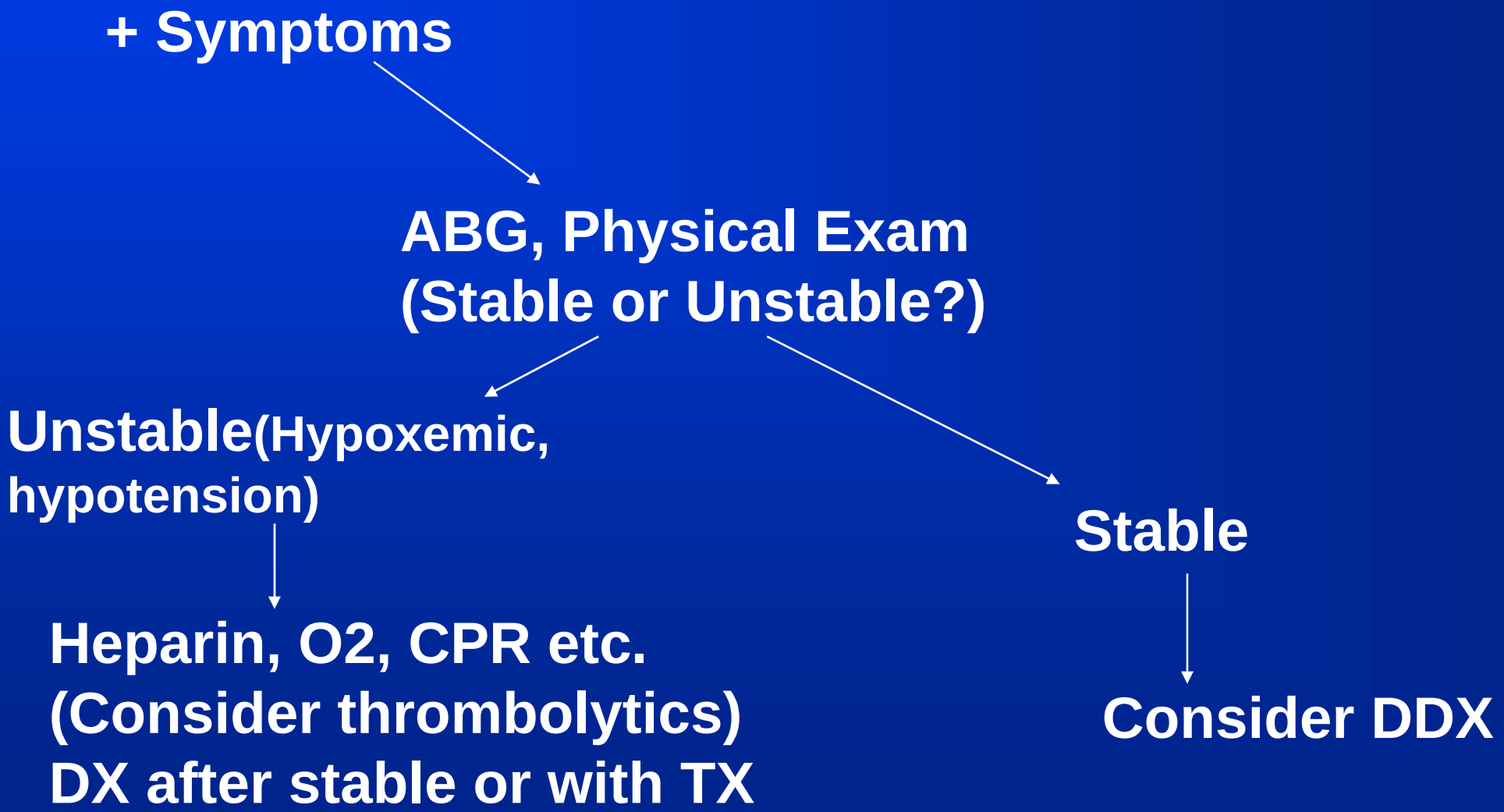
(Thompson et al, 1998; Sanson et al, 1999; Aguilar and Goldhaber, 1999)

Thrombolytic Therapy:

- **Relative contraindication AT DELIVERY**
- **Some case-report experience**
- **Significant bleeding with use**
- **USE AS ALTERNATIVE TO THORACOTOMY (OR DEATH) IN UNSTABLE patient.**
- **PA-catheter directed treatment may allow lower dose**

(Garite and Briggs, 1997; Nishimura et al, 1998)

Dx/Tx Algorithm for PE in Pregnancy (1)



Dx/Tx PE Continued (2)

Consider DDX

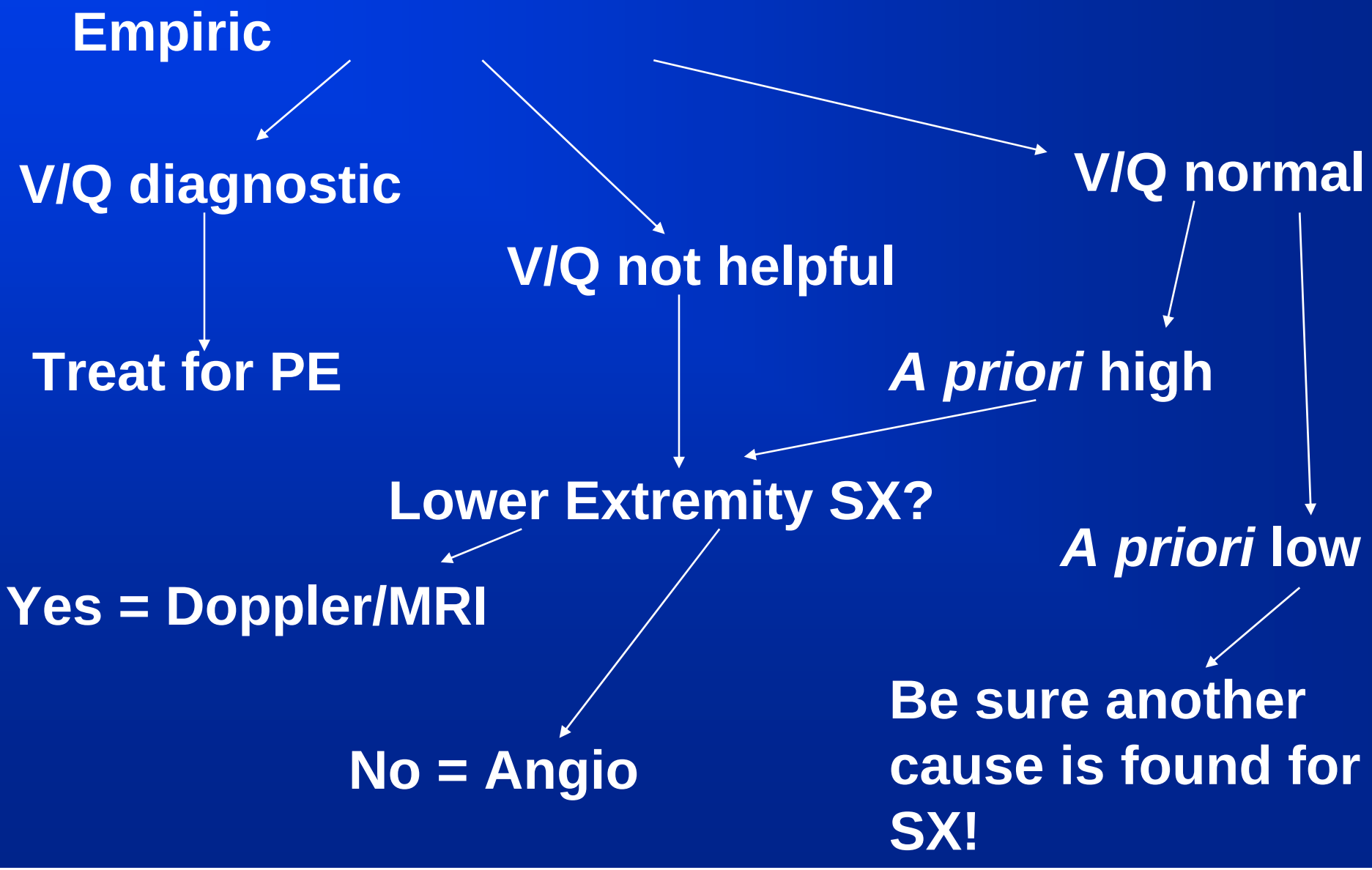
ABG (if not done), ECG, ± CXR
(no delay!)

Other Cause= TX

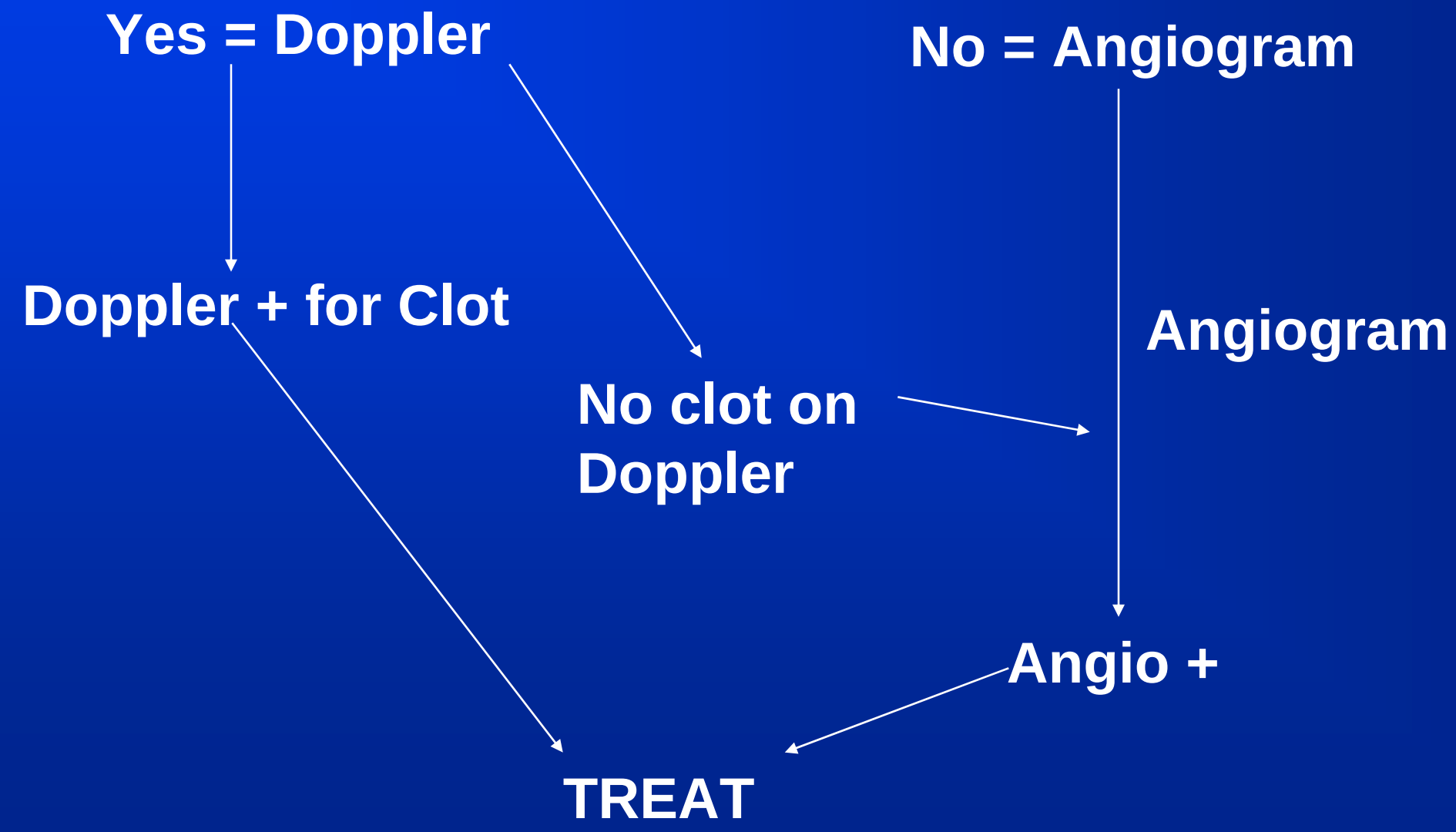
PE still not ruled out-
SUSPECTED

Consider Empiric Heparin
V/Q Scan (heparin if V/Q delayed)

DX/TX PE Cont. (3)



DX/TX Algorithm for PE Cont (4)



Management of Labor in Patient Who is Anticoagulated:

1. Stop full-dose heparin
2. Minidose heparin during labor, delivery and for 6-24 hours postpartum
3. If APTT does not normalize soon enough after full heparin stopped (i.e., patient near delivery), consider protamine
4. 6-24 hrs postpartum, restart full dose tx.- consider changeover to Coumadin when out of immediate danger for postop bleeding

Amniotic Fluid Embolism

- Frequency- 1/15,000 - 1/20,000 Pregnancies
- Catastrophic Consequences
- Multisystem Collapse
- Mortality Quoted as High as 80% (Probably Lower Now)

Pathophysiology- Animal Data:

- Amniotic fluid thought to be composed of some abnormal factor or mediator
- Factor is heat stable
- Factor is soluble?
- Possible relationship with anaphylactoid phenomenon

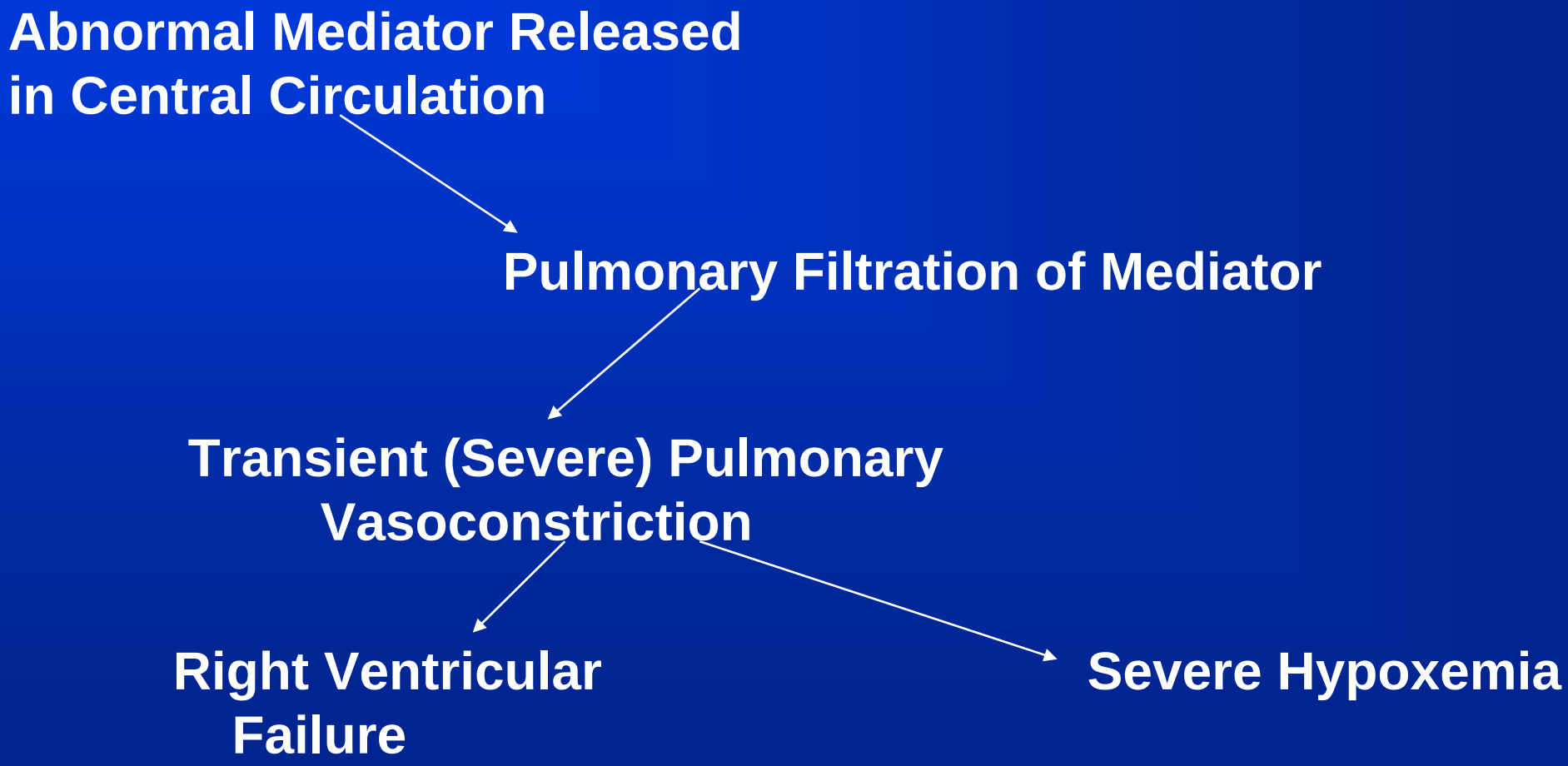
(Hankins, 1995; Hankins, et al, 1993; Clark, 1995)

Situations Related or NOT Related to AFE:

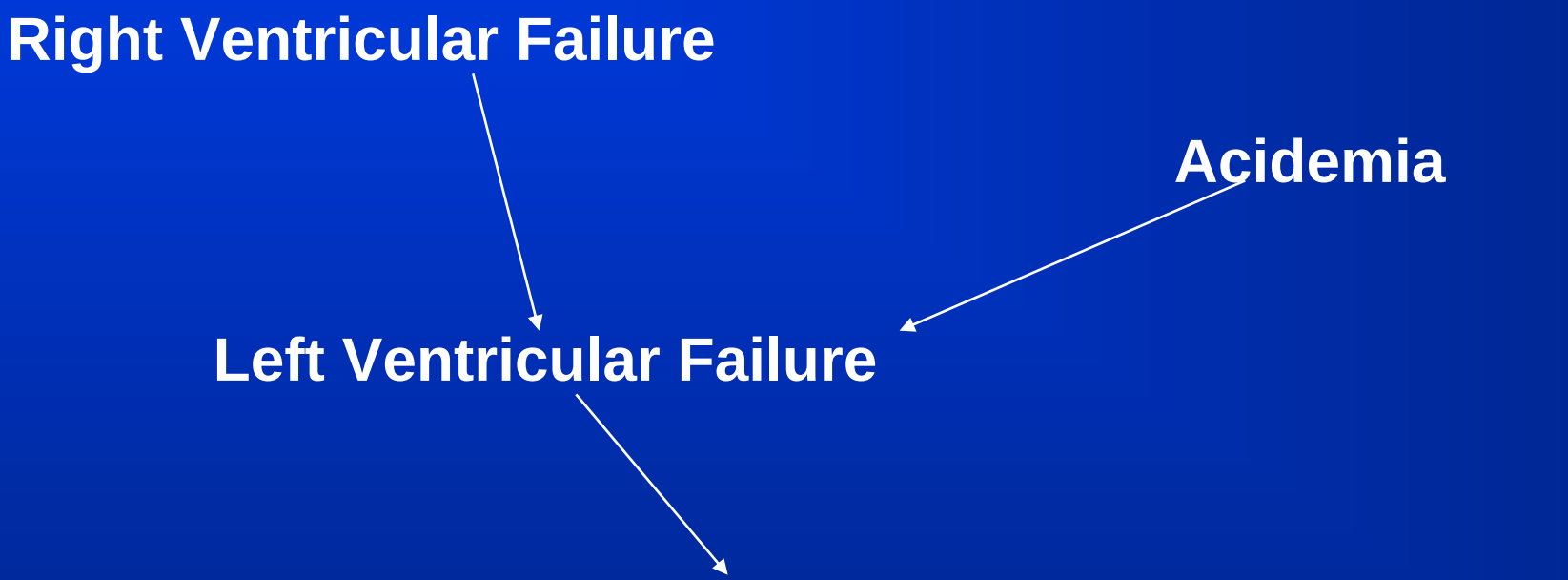
- **Uterine Hyperstimulation- AFE registry suggests that hyperstimulation is EFFECT rather than cause of hyperstimulation**
- **Oxytocin use- NOT RELATED**
- **Drug Allergy and/or Atopy- RELATED, with 41% of patients in AFE registry with allergies**
- **Normal labor!!??**

(Clark, 1997)

Amniotic Fluid Embolism (AFE)- Mechanism



Amniotic Fluid Embolism-Mechanism



AFE- Symptomatology

- **Unexplained Hemodynamic Collapse- Most Common**
- **10-15% Present With Coagulopathy- 40% Who Survive Initial Events Develop Coagulopathy**
- **20% Present With Seizure**
- **70% Develop Respiratory Insufficiency**

(Morgan, 1979; Clark, 1986)

AFE- Differential Diagnosis

- Pulmonary Embolism
- Venous Air Embolism
- Myocardial Infarction
- Eclampsia
- Anaphylaxis
- Local Anesthetic Toxicity

AFE- TREATMENT

- ***R*ecognition is First Step**
- ***E*liminate Other Causes**
- **Support- Hemodynamic and Respiratory Support (LEFT Ventricular Failure)**
- ***C*ontrol Coagulopathy**
- ***U*ndertake Delivery (If Not Delivered)**
- ***E*valuate Necessity For Prolonged Hemodynamic or Respiratory Support**

AFE- Hemodynamic Support

- LEFT ventricular failure predominates
- Pressor/inotrope agents may be necessary
- Oxygen/mechanical ventilation as necessary
- Aggressive correction of coagulopathy

Eclampsia

- Complicates approximately 5% of patients with preeclampsia (EPH) (untreated)
- Significant cause of M/M in patients with EPH
- Majority of cases occur intrapartum

May occur up to one month postpartum (but rare after 3-10 days postpartum)

Cerebral edema or pulmonary edema predict poor outcome

(Williams Obstetrics, 1997 and other sources)

Definitions Cont (4)

Severe Preeclampsia-

- SBP > 160 torr or DBP > 110 torr
- Proteinuria > 5.0 gm/ 24 hr
- Oliguria
- Preeclampsia-associated CNS symptoms
- Epigastric Pain
- Pulmonary Edema
- HELLP
- IUGR

Pregnancy Induced Hypertension (PIH) Factoids:

- No relationship between the degree of proteinuria and propensity for eclampsia
- In patients that develop proteinuria, it may be a delayed finding
- Renal lesion= *Glomeruloendotheliosis*
- Proteinuria and HTN together more significant markers for poor outcome than either alone
- Hypertension and Preeclampsia Risk(Nullipara):

  DBP > 15 torr = Doubled Risk

  SBP > 30 torr = Increased 2 1/2 Fold

  SBP and DBP = Nearly 4 1/2 Fold Increase
- New onset seizure activity in a pregnant or early postpartum pregnant patient is ECLAMPSIA until proven otherwise

(Various Sources- Including Williams Obstetrics- 20th ed, 1997)

Etiology of Eclampsia (EC)

- Etiology uncertain
- May be a manifestation of loss of cerebral autoregulation (hypertensive encephalopathy in a relatively normotensive person)*
- Cerebral findings- Cortical petechiae

*Does the physiologic hyperventilation of pregnancy alter cerebral autoregulation?)

Treatment of Eclampsia

- **Mainstays of treatment are:**
  - **Control of Seizure**
  - **Correction of hypoxemia and acidosis**
  - **Delivery (if undelivered)**
- **Consider the D/DX of Seizure**
  - **Cavernous Sinus**
  - **Local Anesthetic**
  - **CVA**
  - **Metabolic**
  - **Amniotic Fluid Embolism**
  - **Infection**
  - **Substance Abuse**

Eclampsia- Control of Seizure

- Magnesium sulfate most effective for treatment of seizure
- Magnesium sulfate most effective for prophylaxis (several regimens)
- Magnesium sulfate more effective than phenytoin
- Other agents useful for refractory seizure and/or non-availability of magnesium (example- non OB ER)

(Lucas et al, 1995; Eclampsia Collaborative Trial Group, 1995)

Treatment of Eclampsia

Airway Protection

Intravenous Magnesium= 2-4 gm IV- depending on preexisting Mag tx.

Success = Continuous Magnesium

Failure= Consider
- **Other Pharm tx**
- **Complicating Causes**

Treatment of Eclampsia (2)

Treatment Failure?

Consider Other Causes!
Neuro Exam
Cerebral Edema?
Hypertensive Crisis?

Consider CT
Consider Mechanical Ventilation
Consider Cerebral Protective or Edema Treatment
(Get Help!)

Magnesium Sulfate

- After IV loading, infuse with 2.0-3.0 gm/hr
- 4.0 - 7.0 mEq/L = therapeutic range
- Overdose = Calcium gluconate
- Treatment Failure = Consider other cause of seizure or cerebral complication
- DTR's, Urine Output, Respiration, etc., etc.

Eclampsia- Airway/Metabolic

- Control of Airway During Seizure
- Evaluation and correction of hypoxemia
- Diagnosis of Aspiration
- Neurologic and Opthamologic Evaluation
- Treatment of Hypertensive Crisis
- Evaluation of electrolytes, other manifestations of severe EPH and blood gas

Hypertensive Crisis in PIH

Diagnosis- Fetal Sx, Maternal Sx, SBP > 160 torr, DBP > 110 torr

Hydralazine 5-10 mg IV- repeat x 2
Eliminate false positives

Response- no further tx

No response or transient

Hypertensive Crisis in PIH Cont.(2)

No response/ transient response

Consider bolus labetalol tx.

Response- no further tx

No response- xient

Hypertensive Crisis in PIH Cont (3):

Transient or No Response

Arterial Line
Continue Fetal Monitoring
Continuous Labetalol

Response = Continue
or wean before/after delivery

Failure, or fetal or maternal
Symptoms despite tx

Consider Invasive-
Directed Tx

Cardiopulmonary Resuscitation in Pregnancy

- If you don't think that this will never happen to you, you are wrong!
- Being an Obstetrics provider is no excuse not to be CPR literate.
- Non-Obstetrics providers may know more than you do about CPR, but they may know little or nothing about pregnancy, fetal evaluation, etc.
- Even if CPR by non-OB, OB endeavored to function as an advocate ombudsman or resource person for the <u>OB</u> patient and her fetus!

Issues Specific to CPR in Pregnancy

- Pregnancy is a state of increased metabolic demands
- The placenta comprises a 20-30% shunt
- Buffering capacity is diminished during pregnancy
- Functional Residual Volume (FRC) is decreased in pregnancy-predisposing gravida to supine hypoxemia

Issues Specific to CPR in Pregnancy (2)

- **Aortocaval Compression- occurs during second 1/2 of pregnancy. Compression may significantly reduce effectiveness of CPR during second 1/2 of pregnancy**
- **Aspiration risk**
- **Pregnancy-associated causes of cardiopulmonary arrest**

Use of Medications During CPR of Pregnant Person

- When the alternative is death, very few things are absolutely contraindicated
- Most inotrope/vasopressors are either poorly studied or can cause reductions in uteroplacental blood flow
- Thrombolytics are relatively contraindicated if delivery imminent

Fetal Outcome in CPR

- **Limited data suggest intact fetal salvage if delivery afforded by 5 minutes of unsuccessful CPR**
- **Neonatal neurologic impairment increases significantly after 8-10 minutes of CPR**
- **CPR does NOT adequately perfuse the uterus**

(Katz, 1986; ACOG, and others)

IMPORTANT CAVEATS FOR CPR IN PREGNANCY

- **If CPR can be anticipated (sick gravida at risk), pre-arrest planning and counseling vital!**
- **In CPR during pregnancy, in addition to ABC's, immediate fetal evaluation should not delay primary maternal evaluation**

IMPORTANT CAVEATS FOR CPR IN PREGNANCY (2)

- Early Intubation recommended success (>20-25 weeks gestation)
- Thoracostomy tubes, if placed, should be placed with consideration of the fact that the diaphragm is elevated in pregnancy
- Pregnancy causes of arrest need to be considered

Treatment of CPR in Pregnancy

ABC's
Early Intubation
Gestational age?

< 20-25 weeks

Continue CPR
Evaluate fetus when able

> 20-25 weeks

Lateral tilt
Fetus alive?
Gest. age viable?

Treatment of CPR in Pregnancy (2)

Lateral tilt
Fetus alive?
Gest. age viable?

Alive/Viable

C/S by 5 min.
if CPR not working

Pre-viable/Demise

C/S by 5-10 min.
if CPR not working