

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 scintigraphy
- Angiography
- Thoracic enhanced CT
- Extremity Doppler

Chest X-Ray Findings in PE:

- *Hampton's Hump:*
pleural based density at CPJ
- *Westermarck's Sign:*
peripheral oligemia with proximal vessel dilatation
- *Most common 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 Scintigraphy:

- 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

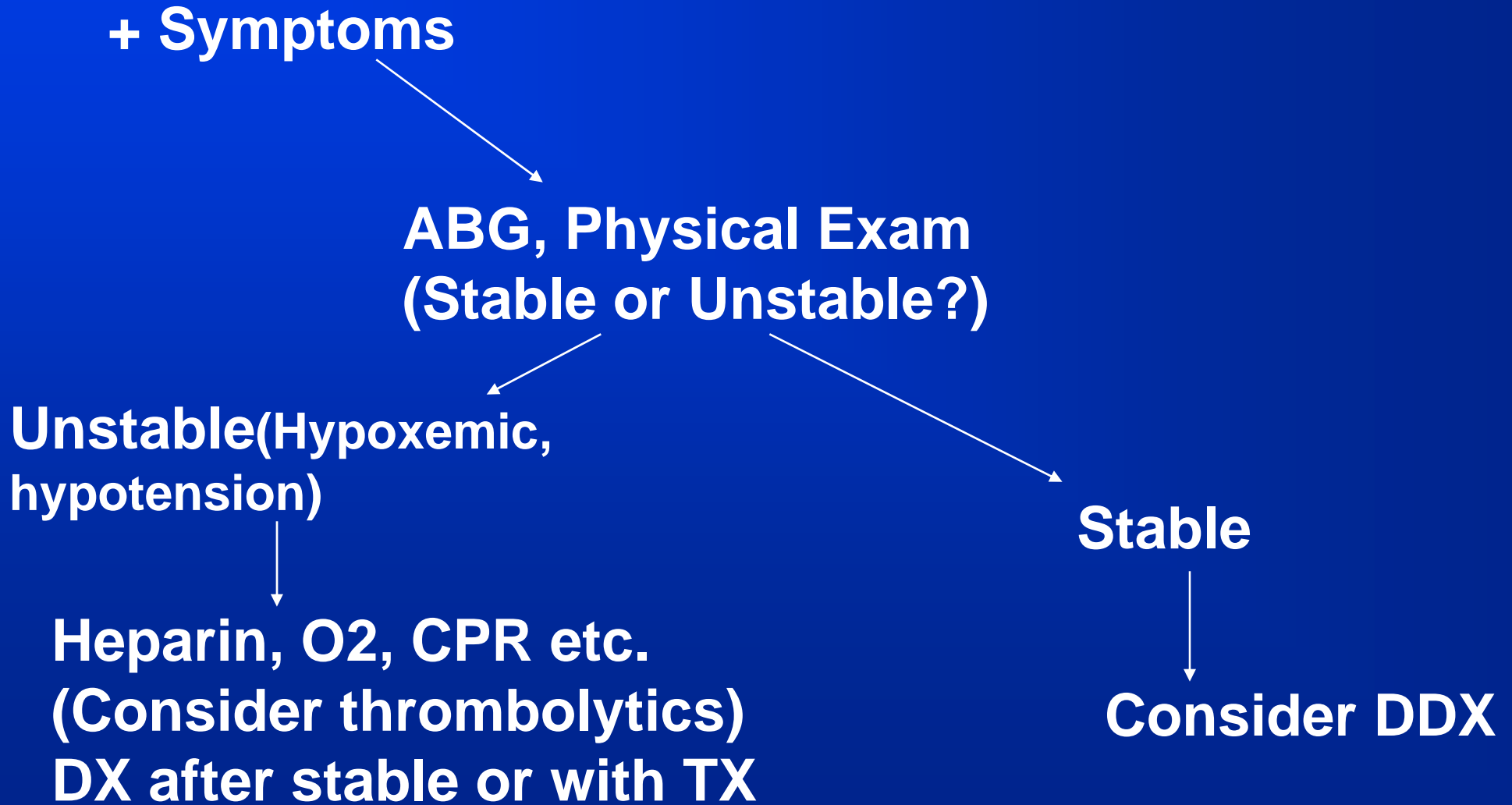
(Thompson et al, 1998; Sanson et al, 1999; Aguilar and Goldhaber, 1999)

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

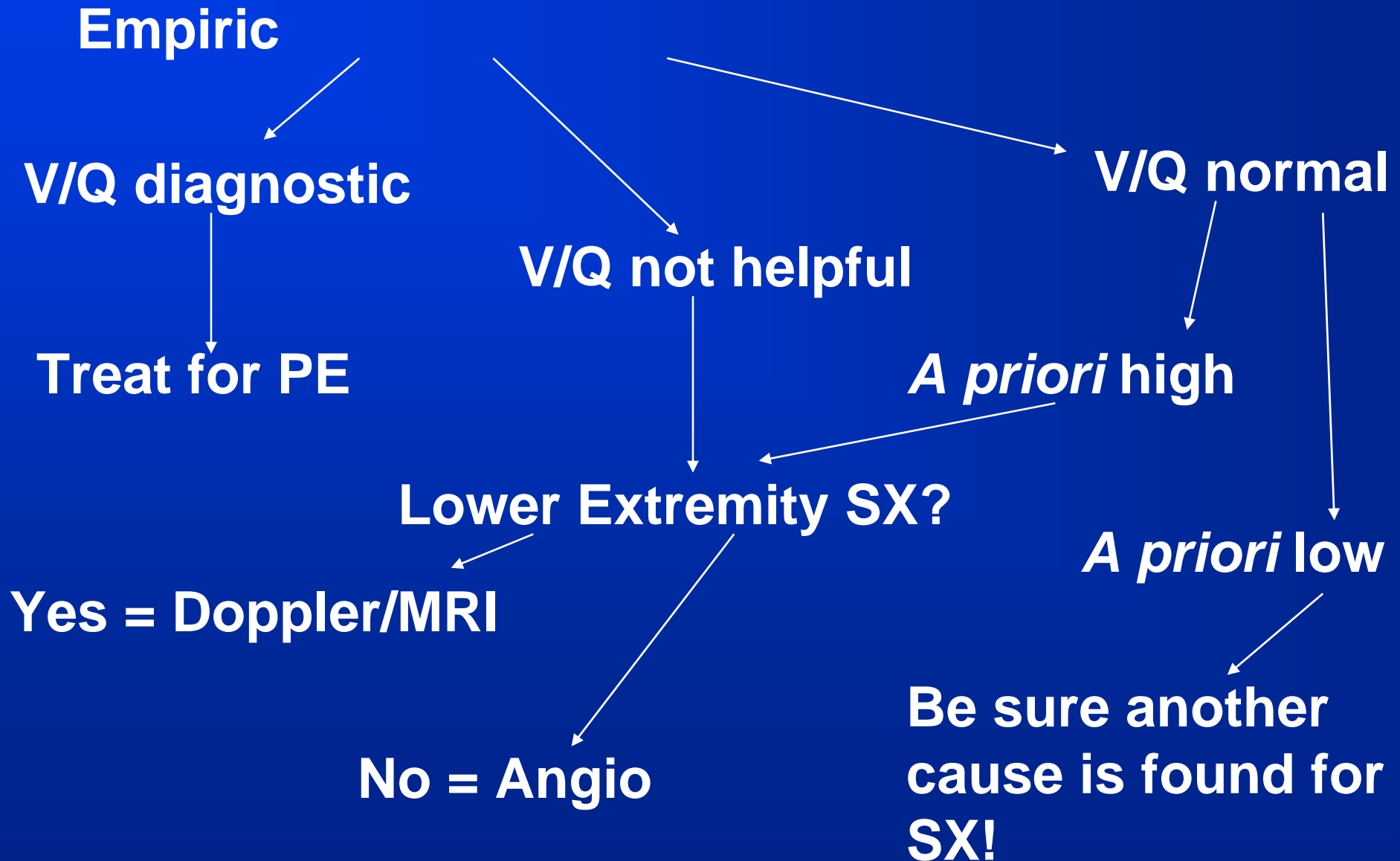
ABG (if not done), ECG, \pm CXR
(no delay!)

Other Cause= TX

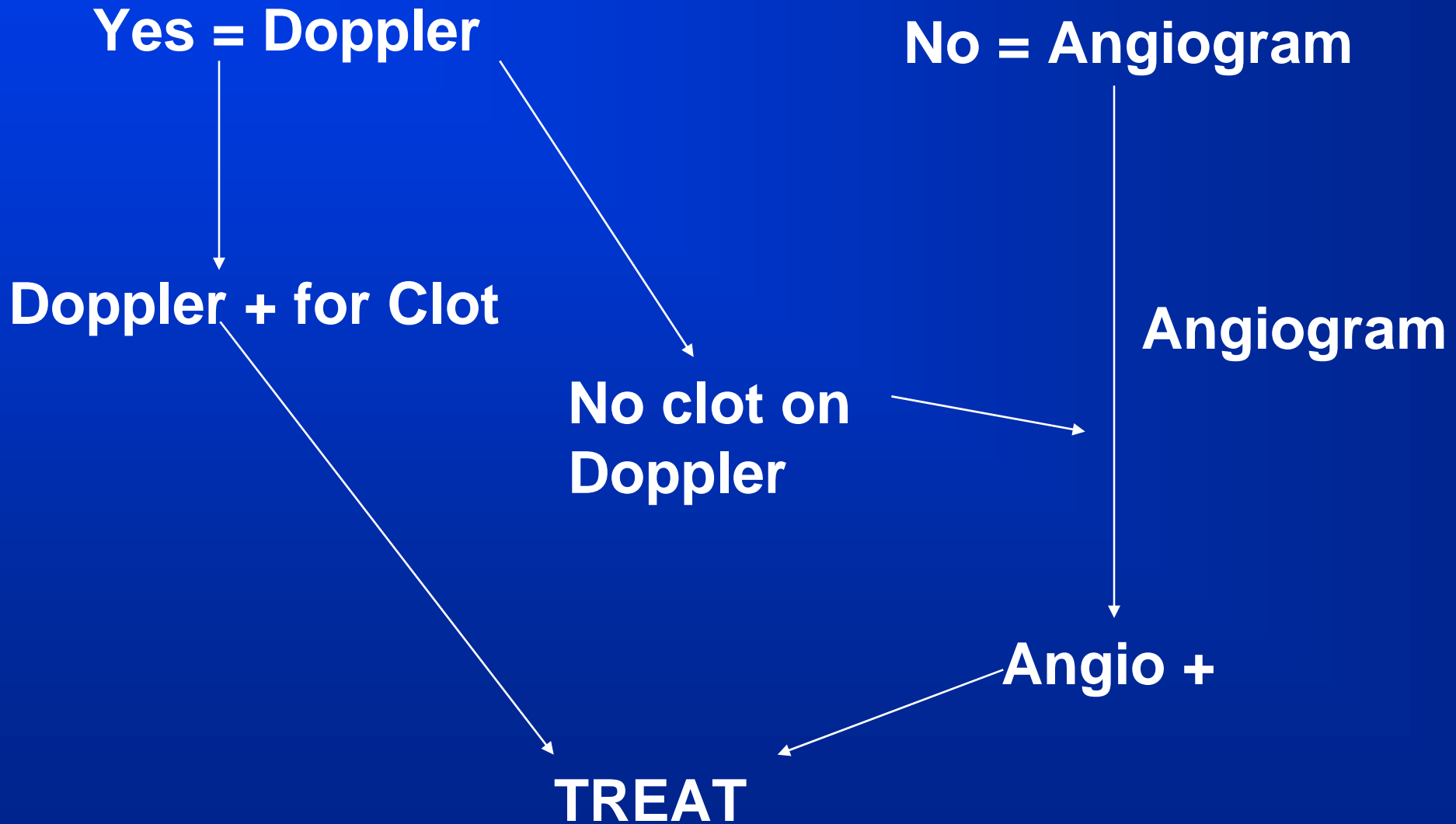
PE still not ruled out-
SUSPECTED

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

Abnormal Mediator Released
in Central Circulation

Pulmonary Filtration of Mediator

Transient (Severe) Pulmonary
Vasoconstriction

Right Ventricular
Failure

Severe Hypoxemia



Amniotic Fluid Embolism- Mechanism

Right Ventricular Failure

Acidemia

Left Ventricular Failure

**Resolution of Right Heart Findings
Continued Manifestations of Left
Ventricular Pump Failure**



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

- Recognition is First Step
- Eliminate Other Causes
- Support- Hemodynamic and Respiratory Support (LEFT Ventricular Failure)
- Control Coagulopathy
- Undertake Delivery (If Not Delivered)
- Evaluate 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 Ophthalmologic 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

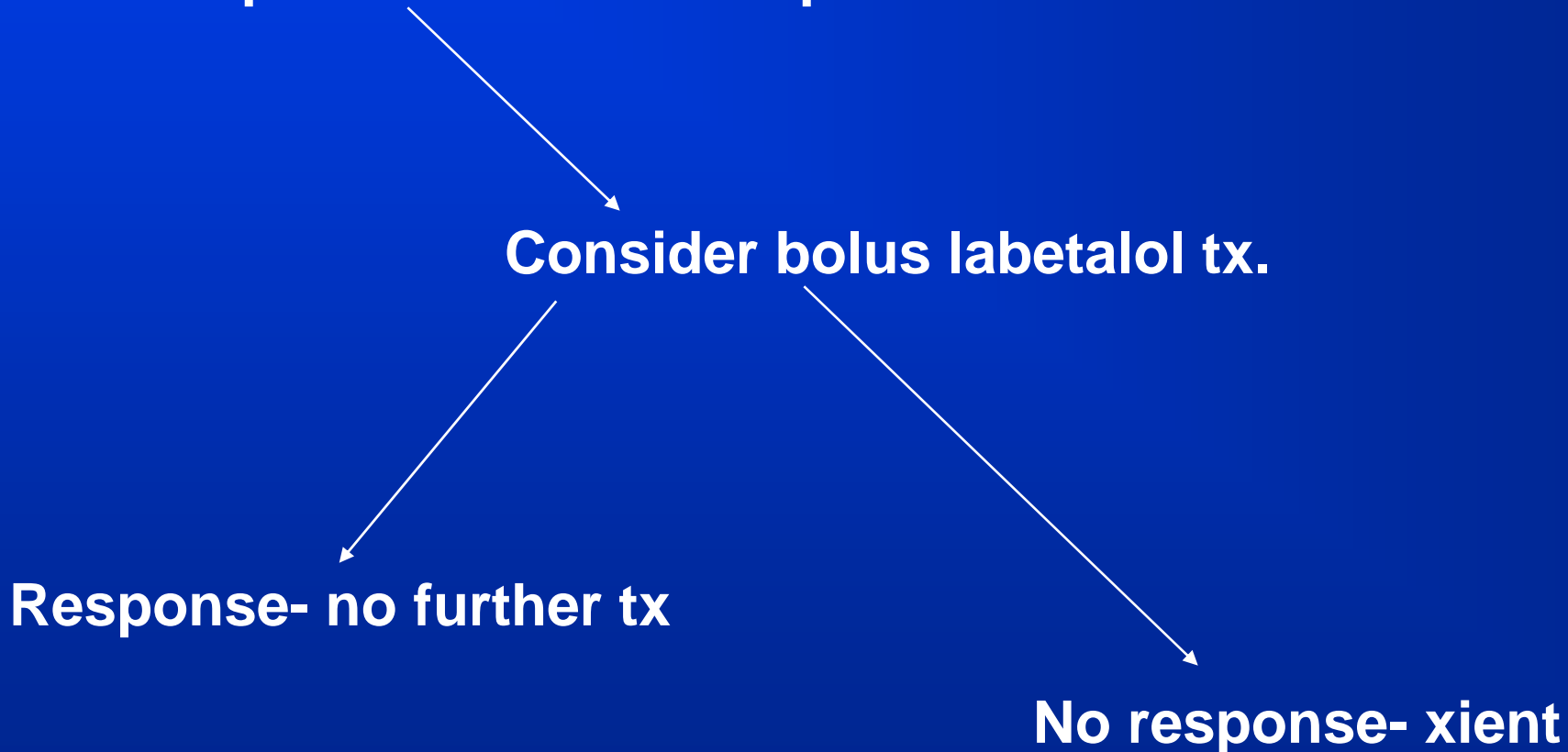
Hypertensive Crisis in PIH Cont.(2)

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

Consider Invasive-
Directed Tx

Failure, or fetal or maternal
Symptoms despite 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 OB 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