Pregnancy Induced Hypertension

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Definitions

1. Hypertension
2. Edema
3. Proteinuria

Hypertension

(SBP) Systolic - Sustained > 140 mm Hg

(DBP) Diastolic - Sustained > 90 mm Hg

$$MAP = \left(\frac{SBP + SBP + DBP}{3} \right)$$

(ACOG, 1996)

Hypertension

SBP rise of 30 mm Hg or DBP rise of 15 mm Hg is probably not significant provided sustained BP is < 140/90 mm Hg

(Villar and Sibai, 1989)

Proteinuria

1. Greater than 300 mg in 24 hour period
2. Greater than 100 mg/dl dipstick (sustainable)

Edema

Difficult Definition

(80 + % of normal gravidas exhibit edema)

Pregnancy Induced Hypertension (PIH)

1. Hypertension related to pregnancy
2. Hypertension returns to baseline by 6 weeks postpartum
3. PIH, by definition, after 20 weeks* gestation

(* exception = GTD)

PIH

1. Preeclampsia
2. Eclampsia
3. Late transient HTN

Preeclampsia	=	PIH with proteinuria
Eclampsia	=	PIH with seizure activity
Late transient HTN	=	HTN alone without other apparent organ involvement

Preeclampsia:

A. Mild
B. Severe
    1. HELLP Syndrome

Severe Preeclampsia

BP > 160/110 mmHg
Proteinuria > 5 gm/24hr
Azotemia/oliguria (< 500 mL/24 hr)
Microangiopathic hemolysis
Thrombocytopenia
End organ symptoms:
1. CNS
2. Visual
3. Hepatic

Intrauterine growth delay (oligohydramnios?)

HELLP Syndrome

Hemolysis

E **L** Elevated Liver Enzyme

L **P** Low Platelet

Special Circumstances (PIH)

1. HTN before 20 weeks gestation = chronic HTN
2. Superimposed PIH*

   Chronic HTN + Superimposed PIH

* Often difficult to ascertain

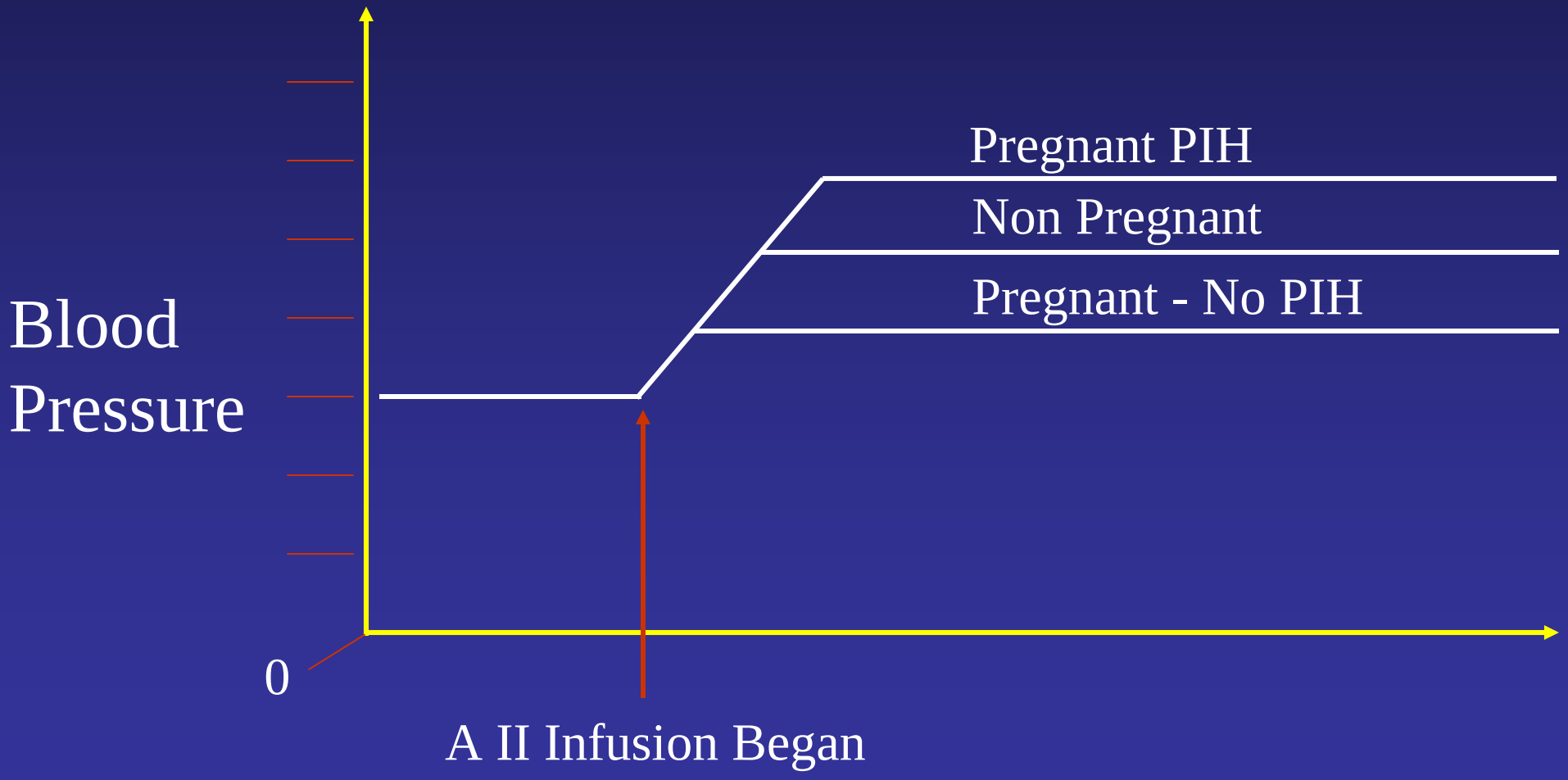
PIH Risk Factors

- Nulliparity
- Young or elderly gravidas
- Family history
- Chronic HTN
- Renal disease
- Antiphospholipid syndrome
- Diabetes
- Multiple gestation
- Angiotensinogen gene T235 (?)
- Previous severe PIH before 28 weeks

PIH Etiology - Uncertain?

1. Altered sensitivity to pressor effects of angiotensin II
2. EDRF?
3. Prostaglandin synthesis?

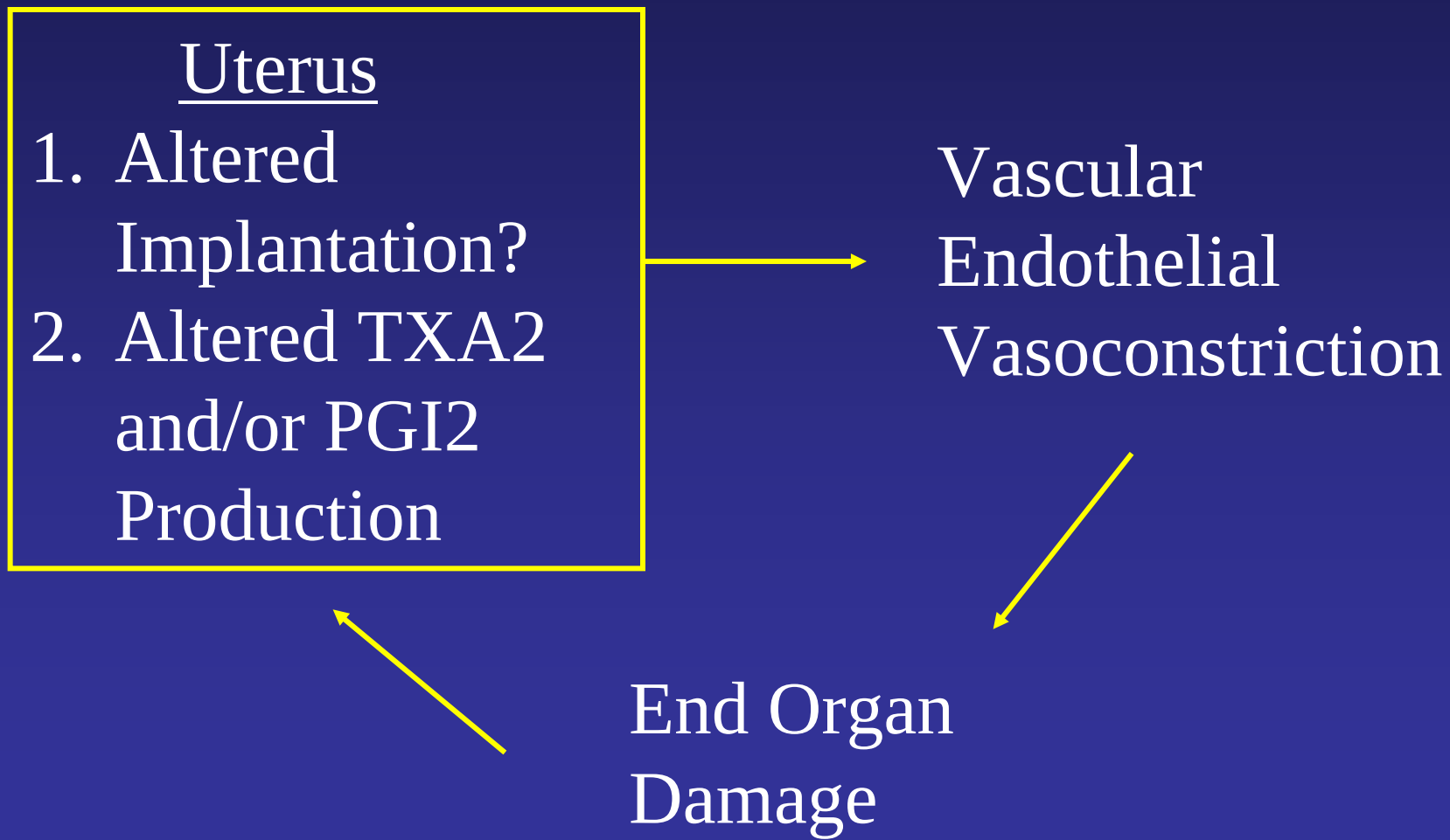
Sensitivity - Angiotensin II



PIH - Prostaglandin

1. Thromboxane (TXA2)
   - Aggregates platelets
   - Vasoconstricts
2. Prostacyclin (PGI2)
   - Inhibits platelet aggregation
   - Vasodilators

PIH Prostaglandins

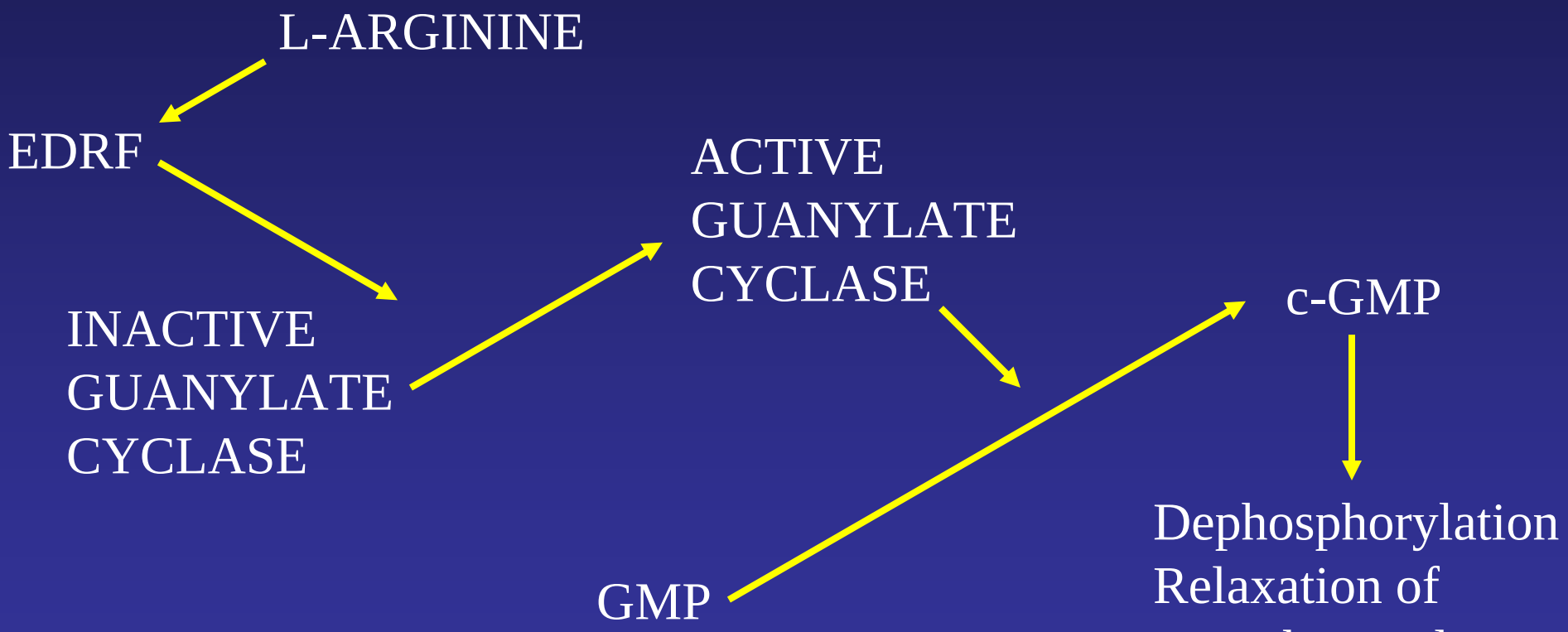


PIH Prostaglandin

1. Low dose ASA Inhibits TXA2
2. Low dose ASA did not improve outcome in normal gravidas or high risk patients

(Sibai, 1995; Hauth, 1998)

PIH - Nitric Oxide (NO or EDRF)



(Morris et al, 1996)

NO in PIH

1. NO may mediate AII refractoriness
2. NO may inhibit uterine contractility
3. NO may modulate uteroplacental blood flow

(Molnar and Hertelenay, 1992; Hull, 1994; Yallampalli, 1993)

NO in PIH

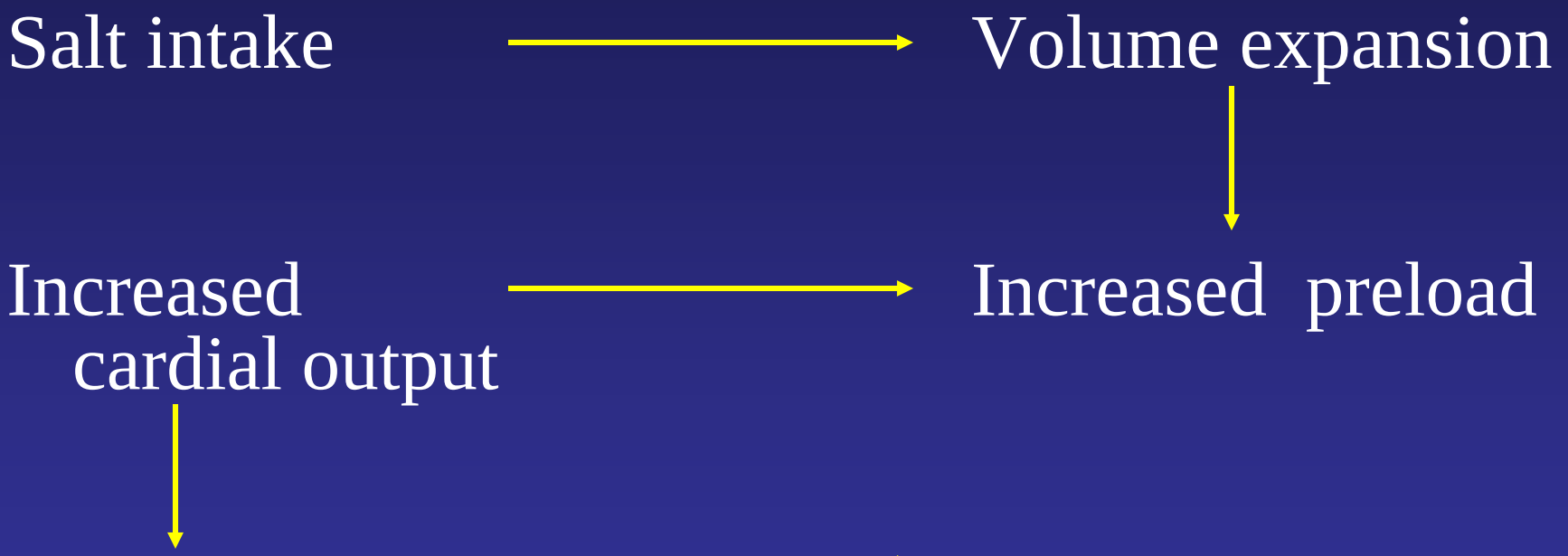
1. In murine model “FALSE” precursor diet (reduces substrate for NO) results in “preeclampsia”
2. Nitrovasodilators (precursors for NO) reduce BP in PIH patients

(Yallampalli et al, 1993; Gonzalez, 1997)

Etiology of PIH

1. Etiology still uncertain
2. Mediator responses may be effect or causal (??!!)

Guytonian Theory of Hypertension



(Easterling et al, 1989)

Management of PIH (1)

Goals:

1. “Termination of pregnancy with least possible trauma to mother and fetus”
2. “Birth of an infant who subsequently thrives”
3. “Complete restoration of health to the mother”

(Cunningham et al, Williams Obstetrics, 1997)

Management of PIH (2)

1. At term (“easy”) - delivery
2. Preterm - risk/benefit analysis

   mild disease - expectant care

   severe disease - controversial

Expectant Management-Severe Preeclampsia (1)

1. Some investigators note improved outcome (fetal/neonatal) without differences in outcome (maternal) by expectant management of the 24-32 (34?) week patient with severe preeclampsia

(Sibai et al, 1990; Olah et al, 1993; Visser and Wallenberg, 1995; Sibai et al, 1994)

Expectant Management-Severe Preeclampsia (2)

1. Neurodevelopmental outcome not impaired by expectant management
2. Daily AFS testing advocated and felt to be effective
3. Daily maternal evaluation necessary as inpatient treatment
4. TERTIARY CARE TREATMENT

(Chari, 1995; Sibai, 1994; Spinillo, 1994)

Expectant Management-Severe Preeclampsia (3)

1. Amniotic fluid volume - somewhat predictive of IUGR. NOT predictive of fetal distress
2. Proteinuria NOT correlated with outcome

(O'Brian 1993; Schucker, 1996; Schiff, 1996)

Expectant Management-Delivery Endpoints (4)

1. Gestational age < 24 weeks or > 32-34 weeks
2. H E L L P
3. Uncontrollable HTN
4. Fetal issues
5. Neurological symptoms
6. Treatment must be in tertiary care center

Delivery Route - Severe Preeclampsia

- Attempts at vaginal delivery supported
- Considerations for C-section
  - Fetal intolerance
  - Obstetric consideration (low threshold)
  - Previous C-section (VBAC?)

Gravida One Delivery With Preeclampsia (INDUCTION?)

- Length of labor and C-section rate higher in those induced with preeclampsia
- Majority do deliver vaginally
- PGE_2 gel probably safe
- "Off label" PGE_1 methyl analogue reported as safe
- Induction probably safe in low birth weight cohort

(Elly et al, 1997; ACOG, 1995; DeValle, 1996; Regenstein, 1995)

Anesthetic Technique - Preeclampsia

- Some controversy exists
- Risks from all techniques
- Recent U.S. study reports no difference in outcome with regional or general techniques if given correctly

(Wallace, 1995; ACOG, 1996)

Eclampsia

- Incidence in those with PIH varies between 2-5% (if not treated)
- Associated with what appears to be cerebral arterial vasospasm (etiology)
- May occur without appreciable HTN or proteinuria

Magnesium Sulfate

- Long empiric experience
- Several regimens are probably effective
- Magnesium treats vasospasm (?) versus other ill-defined mechanisms of action
- Efficacy recently validated as compared to phenytoin

(Lucas, 1995; Eclampsia Collaborative Trial, 1995)

Questions Regarding Magnesium Sulfate

- Do patients with mild PIH need prophylaxis?
- Does magnesium sulfate increase postpartum hemorrhage?
- Will other treatments of cerebral vasospasm work better?

(Burrows, 1993; Witlin, 1997; Belfort, 1995)

Treatment of HTN in Preeclampsia / Chronic HTN

1. Currently, no data suggests a preventive role for treatment of hypertension
2. HTN and treatment of HTN can both produce decreased uteroplacental blood flow
3. Chronic HTN - available data does not suggest outcome improved with treatment unless DBP > 90-100 mmHg.

(ACOG, 1996)

Treatment of HTN in Preeclampsia / Chronic HTN

1. Maternal consideration for treatment of HTN in labor

   SBP > 160-180

   DBP > 105-110

2. Choice of agents?

   Hydralazine

   Labetalol

Patients With Chronic HTN Who Conceive

1. Most agents not well studied - Aldomet - most data
2. Current recommendations are to continue already started therapy - (exception: ACE inhibitors)
3. Target BP in “Low” hypertension range
4. Third trimester AFS testing
5. Bedrest?
6. Observe for IUGR and superimposed PIH

HELLP Syndrome

Hemolysis

E **L** Elevated Hepatic Transaminases

L **P** Low Platelets (Thrombocytopenia < 100,000/mm^3)

HELLP

1. Usually an indication for delivery
2. May occur without appreciable HTN
3. Is indicative of multisystem disease

Differential Diagnosis - HELLP

1. Acute fatty liver of pregnancy
2. Thrombotic thrombocytopenia purpura
3. Systemic lupus erythematosis
4. Hemolytic uremia syndrome

Developing Therapies - HELLP

1. Plasmapheresis
2. Corticosteroids
3. Oxygen delivery therapy

(Belfort, 1993; Wheeler, 1996; Martin, 1997; Martin, 1995)

Pulmonary Artery Catheters - Preeclampsia

1. NOT usually needed
2. Indicative - Refractory HTN
   - Unresponsive oliguria
   - Underlying conditions
   - Unresponsive pulmonary edema

Conclusions

1. PIH is a multifaceted disease of uncertain etiology
2. At its worst, PIH is a multisystem disease
3. Expectant management of severe or worsening preeclampsia needs to be a tertiary care process
4. Developing trends may affect future outcome