Pregnancy-Induced Hypertension

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Risk

- Although the true incidence of PIH remains unknown, it is believed to complicate about 6–10% of pregnancies, contributing to one of the major causes of maternal, fetal, and neonatal mortality and morbidity.

Perioperative Risks

- Short-term maternal risks: CNS dysfunction, hepatocellular injury/hemorrhage, thrombocytopenia, acute DIC, oliguria, pulmonary edema, cerebrovascular events (hemorrhage, encephalopathy), placental abruption, acute renal failure, progression to PEC, or EC and death.
- Short-term fetal risks: Severe intrauterine growth restriction, small for gestational age, preterm birth, low birth weight, oligohydramnios, hypoxia-acidosis, neurologic injury, and intrauterine and perinatal death.

Worry About

- Prompt recognition: When PIH is complicated by PEC, EC, or superimposed forms
- Timely response: With pharmacologic treatment or delivery of fetus/placenta when crucial
- High risk of uteroplacental insufficiency despite elevated maternal BP
- Multiple organ/system-based complications associated with PIH (see Assessment Points table)

Overview

- Broadly defined as BP ≥140/90 mm Hg obtained on at least two occasions at least 4 h apart. PIH is further classified into four categories:
- Chronic Htn: Htn before pregnancy, noted <20 wk gestation (suspect if Htn persists beyond 6 mo postpartum)
- Gestational Htn: BP >140/90 but <160/110 mm Hg after 20 wk gestation in previously normotensive pt. Increased BP in the first 24 h postpartum but normalization of BP within 10 d; no proteinuria, no other associated symptoms, no abnormal lab findings/blood tests
- PEC-EC:
  - PEC: Htn at ≥20 wk gestation + proteinuria (≥300 mg protein in 24-h urine collection)
  - PEC with severe features: Htn at ≥20 wk gestation + any of the following (new onset): Severe Htn (BP ≥160/110 mm Hg); persistently severe cerebral or visual disturbances; thrombocytopenia, <100,000/mm^3; HELLP syndrome; elevated liver enzymes, >2× upper limit normal; pulm edema; serum creatinine, >1.1 mg/dL; or doubling serum creatinine (in the absence of other renal disease)
- Superimposed PEC:
  - Superimposed PEC: Exacerbation of previously controlled CHTN (escalating BP meds) and/or new-onset/increased proteinuria
  - Superimposed PEC with severe features: Exacerbation of previously controlled CHTN despite treatment with severe features/symptoms (cerebral/visual changes, persistent epigastric pain, pulm edema, and lab findings, as discussed previously)

Etiology

- Multifactorial disease process with multiple theories about its pathogenesis. Mechanism is not fully understood. However, a central theory suggests abnormal placental implantation, vascularization, and function.
- Immune factors (auto-antibodies, oxidative stress, natural killer-cell abnormalities) cause placental dysfunction and impaired placental perfusion, stimulating the placental release of antiangiogenic and inflammatory mediators, which eventually initiate maternal endothelial dysfunction and organ damage.

Usual Treatment

- Primary goals include BP control (maintenance of uteroplacental perfusion) and prevention of seizures.
  - Mild-to-moderate Htn: Hypertensive disorders in pregnancy without evidence of severe features <37 wk of gestation (e.g., CHTN, gestational Htn, PE, or superimposed PE without severe features) call for expectant management. In the absence of severe features, antihypertensive therapy is only reserved for chronic Htn. Delivery is suggested if "category described previously" is ≥37 wk gestation (38 wk for chronic Htn).
  - Severe Htn: Hypertensive disorders in pregnancy *with* evidence of severe features ≤34 weeks of gestation call for aggressive management. Consider magnesium sulfate for seizure prophylaxis and corticosteroids for fetal lung maturity. Delivery is suggested if "category described previously" is >34 wk gestation. Delivery should not be delayed, regardless of gestational age, if maternal condition is unstable: this includes cases complicated by uncontrollable severe Htn, any of the severe features, evidence of nonreassuring fetal status, or fetal demise.
- Antihypertensive agents recommended in pregnancy include acute-lowering agents (for severe Htn):
  - Labetalol (onset: 5–10 min, dose: 20 mg IV, 40–80 mg every 10 min, max dose: 300 mg or continuous infusion: 1–2 mg/min)
  - Hydralazine (onset: 10–20 min, dose: 5–10 mg IV every 20 min, max dose: 30 mg)
  - Nifedipine (onset: 5–10 min, dose: 10 mg PO, repeat in 30 min, × 2 doses prn, 10–20 mg every 4–6 h up to max dose: 240 mg in 24 h)
- Antihypertensive agents recommended in pregnancy for long-term treatment include oral agents (for chronic Htn in outpatient settings):
  - Labetalol (dose: 200–2400 mg daily in 2–3 divided doses)
  - Nifedipine (dose: 30–120 mg daily)
  - Methyldopa (0.5–3 g daily in 2–3 divided doses)
  - Hydrochlorothiazide (25–100 mg daily): Second-line agent
- Prophylactic agents recommended for PIH in pregnancy include low-dose aspirin (which inhibits the synthesis of prostaglandins and the biosynthesis of plt thromboxane A_2).
- Agent recommended for seizure prophylaxis in PEC-EC is magnesium sulfate (loading dose: 4–6 g IV over 20–30 min, maintenance dose: 1–2 g/h, continued 24 h postpartum; therapeutic concentration range: 4–8 mEq/L).

Assessment Points*

System	Effect	Assessment by Hx	PE	Test
CV	Vasospasm, increased SVR Decreased circ BV, decreased IV volume Decreased oncotic pressure (decreased alb) Hyperdynamic, increased CO mostly	Decreased exercise tolerance Lightheadedness Decreased UO, dizziness	Increased BP + other signs of hypovolemia (increased HR, decreased mental status, increased RR, decreased UO)	BP cuff, pulse ox, ECG monitoring UA, 24-h output BUN, uric acid, Cr, albumin
RESP	Increased airway edema Increased risk of pulm edema	Hoarseness, stridor Dysphonia, SOB	Lung auscultation (rales, crackles), snoring	Airway exam CXR (if symptomatic)
HEME	Decreased plt count Hemolysis ± DIC (liver involvement)	Bleeding diathesis	Epistaxis Bleeding gums Easy bruisability Bleeding from sites	Hgb + coagulation studies: Plts, PT, PTT, fibrinogen; ± function if decreased plt (bleeding time, ±TEG) Peripheral blood smear (rule out HELLP), LDH, haptoglobin
RENAL	Oliguria or anuria Proteinuria Increased BUN, Cr, uric acid	Decreased UO		UA, 24-h urine protein collection Comprehensive metabolic panel (e.g., albumin, BUN, serum Cr)
HEPAT	Increased liver enzymes HELLP	Epigastric pain	Jaundice RUQ tenderness	Liver function tests (e.g., transaminases—ALT, AST; alkaline phosphatase)
CNS	Impaired CNS autoregulation; vasogenic edema, posterior reversible leukoencephalopathy syndrome	Persistent headaches Visual changes	Diplopia, blurry vision, scotomata Hyperreflexia (DTR)	Rule out posterior reversible leukoencephalopathy syndrome —MRI, CT (if concerning diagnosis)

*Especially in severe gestatational Htn, PEC, or EC.

Key References: Moussa HN, Arian SE, Sibai BM: Management of hypertensive disorders in pregnancy. *Womens Health (Lond)* 10(4):385–404, 2014; Lambert G, Brichant JF, Hartstein G, et al.: Preeclampsia: an update. *Acta Anaesthesiol Belg* 65(4):137–149, 2014.

Perioperative Implications

Preoperative Preparation

- Control BP with pharmacologic agents, and institute anticonvulsant therapy for seizure prophylaxis as needed.
- Examine the airway and evaluate coagulation status.
- Careful intravascular repletion could minimize the severe hypotension seen with regional or general anesthesia. If in doubt, consider TTE to quantify volume status.

Monitoring

- Basic monitoring should include measuring BP by noninvasive method, ECG, pulse rate, pulse oximetry, and UO measurements.
- Invasive BP monitoring with the use of an intra-arterial cath may be necessary in unstable pts with severe PEC or EC; pts needing frequent ABG measurements; pts requiring potent pharmacologic treatment for malignantly increasing BPs; or pts with significant body habitus (obesity) without the availability of an appropriate BP cuff size.
- Repetitive physical exam (e.g., DTRs) and measurement of Mg^{2+} blood levels for pts on magnesium sulfate for antiseizure prophylaxis.

Induction

- Early neuraxial analgesia (epidural) is often the preferred anesthetic technique for delivery (unless contraindicated or unstable); it may relieve vasospasm, decrease circulating catecholamines, improve uteroplacental perfusion, and decrease airway catastrophe risk.
- With the need for general anesthesia in the setting of severe gestational Htn, PEC, and/or EC:
  - Pharmacologic agents should be used to minimize the sympathetic response from direct laryngoscopy and endotracheal intubation.
  - Avoid the use of ketamine for induction due to its sympathetic effects.
  - Airway edema may complicate optimal mask ventilation and rapid-sequence intubation. Various sizes of endotracheal tubes should be available.
  - With the anticipation of a difficult intubation, consider the use of airway adjuncts (e.g., videolaryngoscopy) or an awake/anesthetized fiberoptic endotracheal tube placement. A difficult airway cart should always be in close proximity.

Maintenance

- Use of magnesium sulfate in the management of PEC-EC can contribute to the loss of uterine muscle tone, hence increasing the risk of uterine atony and postpartum hemorrhage. Consider the use of additional uterotonics after delivery. Avoid the use of methylergonovine maleate if possible.
- Reduce the dosage of muscular relaxants when used in conjunction with magnesium sulfate due to the potentiation of neuromuscular relaxant effects. Continuous monitoring with a peripheral nerve stimulator should be utilized.

Extubation

- If evidence of thrombocytopenia prior to placement of epidural, consider rechecking platelet count prior to removal of cath.
- Ensure that pt fulfills all "extubation criteria" prior to extubation.

Postoperative Period

- Pts with PIH are still at risk for progression to PEC and EC after delivery. Seizure prophylaxis with magnesium sulfate may be continued after delivery (48 h postpartum).
- Triage: Consider the need for ICU monitoring post delivery if concerned or if pt is unstable.

Preterm Infant

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Risk

- Births at less than 37 post conceptual wk (PCW) rose sharply from 1990 to 2006 in USA due to the increase in assisted reproductive technology and multiple gestation. It has been slowly declining in the ensuing years.
- In 2013, 11.4% of all live births were <37 PCW; 3.4% were <34 PCW.
- 67% of all infant deaths occur among the premature. Babies born at less than 32 wk are 88 times more likely to perish compared to full-term babies.
- Neonates have the highest periop morbidity and mortality among pediatric pts, with premature infants carrying the highest risk.

Perioperative Risks

- Laryngospasm
- Hypothermia
- Hypoglycemia
- Massive blood loss
- Rapid onset hypoxia
- Bradycardia, poor cardiac output
- Apnea

Worry About

- Occult congenital abnormalities
- Difficult intubation, vascular access
- Cardiac decompensation
- Persistent or reversion to fetal circulation and high pressures in the pulmonary vascular tree
- High airway pressures
- Oxygen toxicity
- Medication or dilution errors
- Pain control
- Transport disaster (extubation or hemodynamic compromise)

Overview

- Immature organ systems present very specific challenges to the anesthesiologist.
- Cardiac physiology is different in the premature and early neonate. The heart has fewer and disorganized contractile elements. Muscle cells contain fewer mitochondria. With low compliance, cardiac output is dependent upon heart rate. Yet, parasympathetics dominate predisposing to bradycardia. The premature heart is exquisitely sensitive to drops in serum calcium levels.
- Fetal lungs have inadequate surfactant production up until about 34–36 wk. The lungs are stiffer, harder to ventilate, and prone to atelectasis. They are vulnerable to volutrauma and barotrauma, which can lead to chronic pulmonary compromise.
- High pressure caused by an ill-fitting endotracheal tube against the trachea can lead to post extubation stridor or potentially subglottic stenosis.
- Babies have a higher oxygen demand and a lower oxygen reserve (FRC), but it is important not to over-oxygenate. Premature infants produce fewer antioxidants against the oxygen free radicals produced during oxygen therapy. Oxygen therapy is associated with retinopathy of prematurity and bronchopulmonary dysplasia.
- Hepatic metabolism of drugs is immature in the premature infant, which alters pharmacokinetics.
- Glucose homeostasis is immature. Glycogen stores are low, predisposing to hypoglycemia. These babies also frequently have dextrose or total parenteral nutrition infusions, which puts them at risk of iatrogenic hyperglycemia. These pts are also relatively insulin resistant.
- Hypothermia is common and can occur rapidly. Premature babies have immature mechanisms for heat homeostasis; they burn brown fat in "nonshivering thermogenesis."
- Premature kidneys have a lower glomerular filtration rate and a decreased ability to concentrate urine. Renal clearance of drugs is lower.
- The coagulation cascade of healthy neonates is immature but "functionally balanced." In sick babies, however, this immaturity may predispose them to coagulopathies leading to bleeding (intraventricular hemorrhage) or thrombotic events.
- Compared to an adult, the larynx is more cephalad, the epiglottis is omega shaped, the glottis lies at an angle, and the narrowest part of the airway is subglottic.
- The risk of postop apnea is high, especially with concomitant anemia (HCT <30%). It is usually mixed central and obstructive and made worse by anesthetics. The risk of apnea is greater than 1% in babies born before 35 wk who have not yet reached 54 PCW and in babies born before 32 wk who have not yet reached 56 PCW. It is important to monitor these babies postop. There is some evidence that spinal anesthesia without additional sedatives is somewhat protective against postop apnea.
- Common problems in critically ill premature infants include congenital abnormalities causing cardiac, respiratory, gastrointestinal, renal or hepatic insufficiency, intraventricular hemorrhage, necrotizing enterocolitis, hernias, or retinopathy of prematurity. These can be the reasons these babies come to the OR, or can complicate surgery done for another reason.

Etiology

- Etiology for prematurity is multifactorial and incompletely understood. Risk factors include:
  - Maternal factors including previous preterm birth, race, extremes of age, substance abuse, including smoking, multiple gestation, obesity, infection, and anemia
  - Fetal factors including congenital anomalies, intrauterine growth restriction, and male sex

Usual Treatment

- Optimization in a NICU setting, including respiratory care, nutrition, hemodynamic support, treatment of infection, and surgical correction or palliation of congenital abnormalities