I. OVERVIEW OF PREGNANCY INDUCED HYPERTENSION, PREECLAMPSIA AND ECLAMPSIA

Preeclampsia is a self-limiting disease in that the only "cure" is delivery. Similarly, and by definition, so is Pregnancy Induced Hypertension (PIH). There is no proven way to prevent preeclampsia from developing, and care focuses on increased surveillance through prenatal visits, monitoring blood pressure, urine protein, edema and other findings. Obviously, the choice to initiate labor is based on fetal maturity. Women may be hospitalized solely for bedrest and blood pressure and lab monitoring, aJlowing more time before delivery of a premature fetus in a more controlled environment. Many women are maintained on bedrest at home. Medication to control blood pressure is not routinely prescribed or advised unless the diastolic blood pressure consistently exceeds 100mm Hg and the gestational age is 30 weeks or less.

Progression to eclampsia cannot always be prevented, and patients may deteriorate suddenly and without warning. However, a large body of evidence indicates that eclampsia is less common with adequate prenatal care and effective monitoring. Sources cite a period of up to two weeks postpartum as the "danger zone," and up to 48 hours postpartum for seizure activity. Some sources suggest an alternative cause if seizures are noted 48 hours after delivery. One source says approximately *75%* of eclamptic seizures occur before delivery. Postpartum, 50% of seizures occur in the first 48 hours after delivery, but can occur up to *6* weeks postpartum. Similarly, effects on other body systems have been observed up to 2 weeks or longer.

A. Pregnancy Induced Hypertension (PIH)

Hypertension in pregnancy was not well-defined in the past, and current literature does not consider individual blood pressure increases compared to pre-pregnancy readings valid. According to ACOG Technical Bulletin# 219, *Hypertension in Pregnancy,* January 0000, the definition of PIH remains a “sustained blood pressure increase to levels of 140/90mm Hg." This value is also used as the reference in *Williams Obstetric , 20th Edition,* 0000, p. 694. Hypertension that appears after 20 weeks gestation is considered PIH; chronic hypertension develops prior to 20 weeks.

Note that *Williams* takes the position that increases in systolic and diastolic blood pressure can be normal or signs of developing pathology. "The prudent physician can only increase surveillance and be aware of changes in blood pressure and lab values" and monitor for developing signs and symptoms of preeclampsia.” PIH is further divided into three categories:

*This is a literature review prepared in support of a case involving a brain-injured baby.
The literature cited (eg: guidelines, textbook references) in this review are here excluded.

1. Hypertension alone (sustained readings of 140/90 or greater).
2. Preeclampsia (hypertension plus proteinuria, generalized edema or both).
   a. Mild.
   b. Severe.
3. Eclampsia (convulsions).

B. Preeclampsia and Eclampsia

Preeclampsia has been further categorized as mild or severe based on the presentation or the extent of end-organ damage. Clinical criteria for severe preeclampsia have been set by *Williams Obstetrics; Guidelines for Perinatal Care;* Association of Women's Health, Obstetric and Neonatal Nurses' (AWHONN) publications *High-Risk and Critical Care: lntrapartum Nursing, Perinatal Nursing, Liability Issues in Perinatal Nursing and Critical Care Obstetric Nursing.*

The criteria are as follows:

1. Blood pressure - > 160 systolic or > 100 diastolic two occasions 6 hours apart with the patient on bed rest
2. Proteinuria - > 5g protein in 24 hours or +3-+4 on qualitative assessment.
3. Oliguria - < 400ml of urine in 24 hours.
4. Cerebral or visual disturbances.
5. Epigastric or right upper quadrant pain.
6. Pulmonary edema or cyanosis.
7. Impaired liver function of unclear etiology.
8. Thrombocytopenia.

Proteinuria is defined as 300mg or more of urine protein over 24 hours, or 100mg/dL in at least two random urine specimens collected 6 hours or more apart. The presence of protein in the urine is considered a sign of progressive or worsening disease, yet in some patients it is a late developing sign. Visual disturbances are another frequent finding in patients with severe preeclampsia and are primarily felt to be caused by vasospasm of the retinal artery.

C. Pathogenesis of PIH and Preeclampsia

Although the exact cause of PIH and preeclampsia is unknown, several theories have been recognized and studied:

- Endothelial cell injury.
- Rejection or insufficient production of blocking antibodies.
- Compromised placental perfusion.
- Altered vascular reactivity.
- Imbalance of prostacyclin and thromboxane.
- Decreased glomerular :filtration rate with retention of salt and water.
- Decreased intravascular volume.
- Increased central nervous system irritability.
- Disseminated intravascular coagulation.
- Uterine muscle stretch with ischemia.

- Dietary and genetic factors.

The most widely accepted theory that is consistent with many of the clinical findings and the complications that may ensue is endothelial injury and vasospasm. Vasospasm may cause elevations in the arterial blood pressure and increased resistance to blood flow. What is not clear is whether vasospasm damages the vessels, or damage to the vessels causes vasospasm. The overall effect of restricting blood flow produces endothelial cell damage which in turn stimulates platelet and fibrinogen utilization. The effect of this process, in simple terms, is to cause changes in the blood flow resulting in hypoxic damage to end organ systems.

Although the exact mechanism of this process is not clear, the vascular effects include decreased blood flow, increased microvascular obstruction and cell hypoxia. What follows is a summary of the various organs that may be affected in the patient with PIH and preeclampsia. A wealth of medical literature addresses each of the body and organ systems listed below:

Cardiovascular

Pregnant women experience a 40-50% increase in circulating blood volume. With severe preeclampsia, vasoconstriction and increased vascular permeability cause a significant decrease in plasma volume that results in decreased perfusion to the organs. This constriction or its effects on the intravascular compartment have been noted well into the postpartum period. This places these women at risk for developing pulmonary edema immediately after delivery.

Renal

Some patients with preeclampsia experience a decrease in renal blood flow that decreases the glomerular filtration rate (GFR), clearance of uric acid and sodium retention. This results in increased plasma creatinine levels, up to two to three times the normal levels found in nonpregnant women. The proteinuria is the result of glomerular damage. With severe preeclampsia, renal damage can also occur from severe vasospasm. These effects are not usually permanent, and recovery of renal function can be expected after delivery.

Hematologic

Endothelial damage prompts a cascade of events, resulting in platelet aggregation at the site of injury with destruction of platelets and red blood cells as they try to pass through narrowed vessels. Laboratory findings include thrombocytopenia (platelet count < 100,000) and hemolysis (break down) of red blood cells. Transfusion of platelets is usually not indicated unless platelet counts drop to < 20,000 or surgery is anticipated. Likewise, anemia is not usually significant enough to mandate red blood cell transfusion, but may affect the oxygen-carrying capacity of the blood.

Hepatic

Vasospasm of the liver may cause hemorrhage and rupture, or necrosis from ischemic changes. Laboratory findings include an elevated SGOT (serum glutamic-oxaloatic transaminase) level. Signs and symptoms of worsening preeclampsia include epigastric and right upper quadrant pain, nausea and vomiting, or continued hypertension. These symptoms warrant further evaluation. The presence of abdomen pain in preeclampsia has also been noted as a warning sign of imminent seizures (eclampsia).

Neurologic

As noted previously, visual disturbances are commonly found with severe preeclampsia, with up to 3% of patients reporting temporary blindness. Eclamptic seizures are rare and are usually grand mal

in nature. They have been noted to occur in the antepartum, intrapartum and postpartum periods, up to several weeks after delivery. The appearance of seizures is the diagnostic criterion for eclampsia and is associated with more serious complications for mother and fetus, including maternal hypoxia, cerebral hemorrhage and death.

Placental Effects

During early pregnancy, the pathophysiology in preeclampsia is a failure of the normal process of placentation involving trophoblast invasion of the uterine wall. A series of processes involving the arteries, the musculoelastic wall structure of placental arteries and acute atherosis result in compromised blood flow to the fetus in early gestation and throughout pregnancy. Medication to reduce hypertension may further decrease blood flow through the placenta and to the fetus if vasodilation occurs. This is the rationale behind maintenance of higher diastolic blood pressure readings during pregnancy.

D. Treatment

The treatment for preeclampsia and PIH is delivery of the fetus and placenta. If delivery is remote from term, steroids (betamethasone) are often prescribed to accelerate fetal lung maturity. When the fetus is previable or borderline viable, there is little consensus on the best course of management Unless fetal or maternal distress is indicated, vaginal delivery is acceptable.

1. Magnesium Sulfate

This remains the drug of choice for preventing and controlling seizure activity in patients with PIH and preeclampsia. While the exact mechanism remains unknown, magnesium slows or blocks neuromuscular conduction and increases the seizure threshold. The clinical evidence is clear that its use, either IM or IV in appropriate doses, decreases the incidence of eclampsia and improves perinatal outcomes. The goal of therapy is to maintain a therapeutic serum level of 4-8mg/dL. The two generally accepted methods or protocols for magnesium sulfate administration are IV/IM combination or just IV administration. Continuous IV administration is preferred as it provides more control over serum levels and is less painful for the patient.

A loading dose of 4-6g magnesium sulfate is given over 15-30 minutes, followed by a continuous infusion of 1, 2 or 3 grams per hour based on serum levels. Traditionally, nurses have monitored respiratory status and deep tendon reflexes as evidence of toxicity or therapeutic doses. This has not been found to correlate well with serum levels, and therapeutic serum levels should be checked periodically. *Williams Obstetrics* states that all women with PIH should be given magnesium sulfate during labor and for 24 hours postpartum.

If a seizure occurs in a patient receiving magnesium sulfate, an additional 2 grams IV is recommended over three to :five minutes. If the patient is not on magnesium sulfate, a loading dose of 4 grams is given IV over five minutes, with additional doses of 2 grams over three to five minutes if seizure activity continues.

Antiseizure medications such as benzodiazepines and phenytoin are more commonly used outside the U.S. Several randomized trials have found administering magnesium sulfate to be superior for both mother and fetus. The Eclampsia Trial Collaborative Group study, published in 0000 in *The Lancet,* is an excellent source for the research to support this.

2. Antihypertensive Medications

During pregnancy complicated by PIH, the use of antihypertensive medications has been reserved for patients with a diastolic blood pressure that reaches or exceeds 110mm Hg. The treatment goal is reducing the risk of cerebrovascular accident (CVA) but maintaining perfusion to the organs and the placenta. Ideally, the diastolic blood pressure should remain greater than 80-100, but less than 110mm Hg, which is a narrow window. In general, diuretics are not recommended for the acute management of PIH unless hypervolemia is noted with invasive hemodynamic findings. Alpha-methyldopa (such as Aldomet) has been used in long-term treatment during pregnancy·with favorable results, but is not appropriate during the acute phase due to the delayed onset of therapeutic action.

3. Other Treatment or Therapy

Prevention of preeclampsia with a variety of drugs and management protocols has been studied. No benefit has been found in dietary salt restriction, diuretic therapy or high protein diets. In addition, calcium supplements and aspirin therapy have been studied, with no positive clinical evidence to support their routine use.

II. PRENATAL CARE

The prevailing literature delineates the following as standard of care for prenatal surveillance in non high-risk pregnancies:

- At 0-28 weeks gestation, doctor visits once every four weeks.
- At 28-36 weeks, doctor visits once every two weeks.
- From 36 weeks to delivery, weekly visits.

Keep in mind Kate presented for initial prenatal care at approximately 30 weeks gestation. Based on the determination of a high-risk pregnancy, more frequent weekly visits (with NST or other fetal surveillance testing) may have been indicated.

Each visit should record weight gain, blood pressure, fundal height, abdominal examination and fetal heart tones. Urine should be checked for glucose, ketones and protein. Most importantly, these measures must be compared to results from previous visits.

Proteinuria of more than 300mg/24 hours (or at least 2+ on standard dipstick testing) may indicate renal dysfunction, or if associated with hypertension, the onset or progression of preeclampsia-eclampsia. At this point additional blood and mine tests may be ordered.

III. AUGMENTING LABOR AND PITOCIN USE

The most complete and current practice recommendations for the use of Pitocin in labor are inACOG Technical Bulletins# *218,Dystocia and the Augmentation of Labor,* December 0000, and# 217, *Induction of Labor,* December 0000. This information is also covered in ACOG Technical Bulletin # 207, *Fetal Heart Rate Patterns: Monitoring, Interpretation, and Management,* July 0000.

A. Indications for Use of Pitocin

The following (from ACOG Technical Bulletin# 217) are generally accepted in the literature as indications for the induction of labor:

- Pregnancy induced hypertension.
- Premature rupture of membranes.
- Chorioamnionitis.
- Suspected fetal jeopardy involving growth retardation or isoimmunization disorders.
- Maternal medical conditions, such as diabetes, renal, pulmonary or cardiac conditions.
- Intrauterine fetal demise.
- Postdated pregnancy.

None of the contraindications to labor induction involve clinical conditions related to this case. Special monitoring with induction of labor may be appropriate for multiple fetus deliveries, polyhydramnios, maternal cardiac disease, abnormal fetal heart rate patterns not requiring urgent or emergency delivery. severe hypertension and breech presentation. Based on the documentation of prenatal care and NST results, Kate was an appropriate candidate for a trial of induction or augmentation of labor with Pitocin.

B. Fetal Monitoring

Pitocin administration and the induction or augmentation of labor may cause uterine hyperstimulation or contractility and requires more vigilant monitoring of fetal and uterine activity. This is usually achieved by a 1:1 nurse/patient ratio, which appears to have been the case during Kate's course of labor.

1. Heart Rate Patterns

The following descriptions of fetal well-being as observed during electronic fetal monitoring (EFM) are summarized from several references and provide a basis for understanding the course of labor in this case.

Reassuring. A reassuring EFM pattern with short-term variability and spontaneous accelerations indicates a nonhypoxic fetus. EFM characteristics include a normal baseline (120- 160s), presence of short-term and long-term variability, although long-term variability may diminish if the fetus is in a sleep state. Spontaneous accelerations and early decelerations may be observed. If the baseline fetal heart rate is stable, some practitioners see mild variable decelerations as a reassuring pattern if short- and long-term variability are also present.

Compensatory fetal heart rate patterns include features of a normal baseline with tachycardia or bradycardia with spontaneous accelerations, short-term variability, minimal long-term variability and mild variable decelerations. Specific interventions should be initiated to improve fetal oxygenation and investigate the cause of hypoxia. Scalp stimulation and other measures to provoke accelerations should be attempted. If an acceleration is not initiated and hypoxia is not minimized, this pattern may progress to a nonreassuring fetal heart rate pattern.

Nonreassuring. This term describes patterns that reflect a deterioration of fetal status due to hypoxia and depletion of oxygen reserves. Such patterns increase the risk of progression to metabolic acidosis. Specific interventions again are recommended to correct hypoxia and further evaluate fetal well-being with fetal scalp sampling or stimulation to assist in determining acid- base status.

Features of nonreassuring fetal patterns include a tachycardic or bradycardic baseline, absent short-term variability, absent or minimal long-term variability, no accelerations and early decelerations, late decelerations or a mixed variable and late deceleration pattern. Fetal movement often decreases, and variability diminishes or decreases over time. If this pattern persists more than 30 minutes, further evaluation is required, including assessing maternal vital signs, pain status and vagina)l bleeding; and amniotic fluid color, amount and odor. A vaginal examination should be done to determine if expedited delivery is indicated and, if so, by what route.

Recognized interventions for a persistent nonreassuring fetal heart rate pattern (often termed intrauterine resuscitation measures) include assessing the pattern to determine etiology (as described above), improving fetal oxygenation by placing mother in lateral position and applying oxygen, measuring fetal scalp pH if EFM status does not improve, and doing a vaginal examination to assist in determining if operative intervention is required.

Ominous. Fetal heart rate patterns that are labeled ominous include persistent (terminal) bradycardia, repetitive severe variable or late decelerations. Ominous patterns are primarily noted when there is no heart rate variability combined with metabolic acidosis or asphyxia.

Features of ominous patterns include absent short- and long-term variability or minimal long-term variability, no accelerations and variable, late, prolonged or spontaneous decelerations. These patterns may be referred to as "flat lines" and often require neonatal resuscitation. Interventions include immediate notification of the physician and delivery as soon as possible, preferably with a skilled physician in attendance.

2. Variability Terminology

The difficulty with EFM strip interpretation may lie in the definitions of short- and long-term variability and their significance in determining whether the fetus has an intact and appropriately functioning neurological status. Variability does not apply to accelerations or decelerations, but to the baseline fetal heart rate. Variability is present when the baseline is chaotic and fluctuating; it is absent when the baseline is relatively smooth. It may be documented as absent, reduced or normal. Community General Hospital documents variability in terms of absent (0-2 beats per minute), minimal (2-5), average (5-15) or marked(> 15).

Short-term variability (STV), also known as beat-to-beat variability (BTBV), is usually recorded as present or absent. STV is best monitored with an internal or spiral electrode. In general, the presence of short-term variability indicates oxygenation of the brain, autonomic nervous system and heart and is a sign of fetal well-being.

Long-term variability (LTV) refers to the heart rate pattern fluctuating around the baseline and is measured in cycles per minute (increase and decrease from baseline). Terms used to describe LTV

include absent (0-1 beat or cycle per minute), minimal or decreased (3-5), average (6-10), moderate (11-25) or increase d (> 25). Community General Hospital does not distinguish between the LTVs and STVs on their labor and delivery flowsheet.

Unlike STV, LTV is not generally an indicator of fetal well-being but is considered nonreassuring if absent. Minimal LTV is often observed during fetal sleep cycles, if the mother has received narcotics or if the fetus is not adequately oxygenated. Minimal LTV must be evaluated over a period of time (such as 60 minutes prior to onset) to determine the etiology. Average LTV is a sign of an intact nervous system in the fetus, indicating adequate oxygenation to the brainstem if spontaneous accelerations and fetal movement are also present.

Based on these definitions and the documentation by the physicians and nurse monitoring the EFM strip, I do not see any gross misinterpretation or misapplication of the terms, or any significant deviations from what is seen on the strip. Since no ominous patterns or significant deceleration patterns were present, it may be difficult to determine the exact time that any intervention (repositioning, oxygen application, fetal scalp pH determination or cesarean section) would have made any difference in the outcome at birth. You will need to have an obstetrician expert as well as a labor and delivery nurse expert review this area in more detail.

C. Uterine Hypercontractility

The physician's orders upon admission (Bates 000036) included parameters for Pitocin and monitoring the contraction pattern. The beginning rate of Pitocin ordered was 2mu/min with incremental increases by 2mu/min every 20 minutes to achieve the desired contraction pattern. Measurement of contraction intensity by an intrauterine pressure monitor (Intran) was ordered with a goal of 60-80mm Hg and a desired frequency of contractions every 2-3 minutes. Several sources indicate that uterine activity goals should include contractions every 2-3 minutes, lasting 40-90 seconds with an intensity of 40-90mm Hg by intrauterine monitoring. The resting uterine tone should be less than 20mm Hg by intrauterine monitoring.

Uterine hypercontractility or hyperstimulation is defined as a resting tone of 20mm Hg or greater, more than *5* contractions in 10 minutes or contractions lasting longer than 90 seconds. Hypercontractility of the uterus may result in uteroplacental insufficiency, fetal compromise, uterine rupture and cervical or lower uterine lacerations. If a nonreassuring fetal heart rate pattern or hyperstimulation is noted, the Pitocin should be decreased or discontinued and intrauterine resuscitation measures initiated. If the patterns improve with these measures, Pitocin may be

restarted.

The nursing and physician documentation clearly indicate that uterine hyperstimulation was a factor in this case, but the continuing fetal monitoring indicates that this condition was promptly recognized and monitored. Based on the fetal monitor tracings and documentation of the interpretation of the strip in the progress notes, no gross deviations from the standard of care are apparent. It could be argued that Pitocin should have been stopped completely, or fetal well-being should have been evaluated more quickly. But no drastic change occurred in the fetal monitor strip to indicate an obvious sudden "crash" or deterioration.

D. Interventions

Specific nursing actions that are part of intrauterine resuscitation include:

- Notifying the physician.
- Stopping the Pitocin (if administered).
- Placing the mother on either side (left lateral is preferred) to reduce pressure on the inferior vena cava (which in this case could also have contributed to the decreased maternal cardiac output, hypotension - also called supine hypotension syndrome - and decreased uterine blood flow, although the maternal heart rate remained elevated).
- Administering I00% oxygen by face mask at 8-10 liters per minute.
- Administering IV fluid boluses without dextrose.
- Continuous monitoring of the patient

Additional actions after these interventions include scalp, acoustic or abdominal stimulation to determine fetal well-being as evidenced by EFM accelerations; beginning amnioinfusion if umbilical cord compression is suspected; and if acute fetal distress is severe or progressively worsening, initiating preparations for delivery by cesarean section.

IV. DELIVERY AND FETAL STATUS

A. Apgar Scores and Fetal or Neonatal Neurologic Injury

Apgar scores describe fetal distress, hypoxia, asphyxia, hypoxemia, acidemia, etc. It is most important to note ACOG committee opinions have long held that a low Apgar score of l at *5* minutes is not predictive or indicative of future neurologic sequelae. An Apgar score of 0-3 at *5* minutes is associated with a risk of cerebral palsy in term infants, but not a significantly higher risk percentage-wise. Briefly, neonatal asphyxia severe enough to be related to acute neurological injury should demonstrate all of the following:

- Umbilical cord arterial sample pH< 7.00 (profound metabolic or mixed acidemia).
- Apgar score 0-3 for more than *5* minutes.
- Neonatal seizures, coma or hypotonia.
- Multisystem organ dysfunction (affecting cardiovascular, gastrointestinal, hematologic, pulmonary or renal systems).

The ACOG definition, widely used in determining if a birth event resulted in cerebral palsy or neurologic delays, has been disputed in certain cases by pediatric neurologists. I am aware of pediatric neurologists who do not embrace the above requirements to define a birth asphyxia event. However, based on the discharge MRI and EEG, it is not clear what future neurologic or developmental delays Heather may experience as she reaches developmental and physical milestones or even begins a formal education program. ACOG Technical Bulletin #163, *Fetal and Neonatal Neurologic lnjury,* January 0000, is relied upon heavily in evaluating birth injury cases. It contains pertinent references to neonatal acidosis, cerebral palsy and other neurologic abnormalities commonly associated with fetal distress at birth.

V. PERIPARTUM CARDIOMYOPATHY AND PULMONARY EDEMA

The term, peripartum cardiomyopathy, has been used to describe cardiac failure during the last months of pregnancy and up to six months postpartum when there is no specific etiology or previous history of heart disease. The most common diagnostic findings include right and left ventricular failure with pulmonary congestion, hepatomegaly, low cardiac output, chest pain, hemoptysis and cough, fatigue, dyspnea, edema, murmurs, pulmonary rales and evidence of cardiomegaly. ECG changes and arrhythmias may occur. Pulmonary and systemic emboli are also common and will appear on an echocardiogram as mural thrombi (and give rise to cerebral infarcts and ischemia).

Treatment is similar to that for congestive heart failure and includes administration of digoxin and diuretics, salt restriction and prolonged bed rest. Vasodilators that reduce afterload of the heart may be used, but medications contraindicated in pregnancy, including Angiotensin-converting enzyme inhibitors (ACE inhibitors), should not be used. For patients who have embolic episodes or continuing heart failure, anticoagulants are indicated as prophylaxis for emboli.

Underlying hypertension with superimposed preeclampsia has been a common finding in patients with no other explanation for peripartum cardiomyopathy. More than 50% of patients return to baseline cardiac function with appropriate treatment but have significant risk of recurrence with future pregnancies, which is not advised for these women. Many of these patients opt for permanent sterilization since taking the risk of pregnancy can result in serious cardiac problems, including death, and fetal disorders.