OB Oral Sheet

The answers for these common oral board questions may not be appropriate. Please just use it as a reference. All suggestions are welcome!

—— *JP Wang, June 2007*

1. Maternal Physiology
2. Fetal Assessment and Monitoring
3. Antepartum Bleeding
4. Postpartum Bleeding
5. Pregnancy Induced Hypertension

Maternal Physiology

Preop:

1. What physiology chances of pregnancy affect your anesthetic management?

Almost all do:

a. Respiratory - increased edema and friability of the nasopharyngeal mucosa predisposes to airway bleeding and swelling, especially during nasal intubation; intubation itself may be more difficult; the hyperventilation causes a respiratory alkalosis, metabolic acidosis, and hyperoxia; the decreased FRC and elevated VO2 predispose to rapid artial desaturation anytime during e case.

b. Cardiac- the elevated CO predisposes to CHF, especially postpartum; venous return is likely compromised due to aortocaval compression in the supine position, making adequate hydration and LUD essential.

c. Hemo - hypervolemia and relative anemia are expected findings, along with a hypercoaguable state.

d. GI - upward displacement of the pylorus, LES incompetence, increased gastric volume, decrease gastric pH, and possibly delayed gastric emptying predispose to aspiration. Aspiration prophylaxis and airway techniques that reduce aspiration risk should always be considered.

e. Renal - increased RBF and GFR lower the expected BUN/Cr to 8/0.5.

f. Endocrine - since pregnancy is a diabetogenic state, blood sugar should be monitored as part of routine care.

g. Neuromuscular - MAC and the required dose of LA for epidural and spinal are all decreased (possibly due to changes such as chronic progesterone exposure or venous plexus compression of the epidural and Intrathecal spaces).

2. The patient's ABG is 7.35/40/86. Is this OK?

I would be slightly concerned since the normal ABG at term is 7.44/30/103. This patient has a slight respiratory acidosis and relative hypoxemia.

3. What accounts for the increase in minute ventilation during pregnancy?

Minute ventilation equals RR X TV. The increase in minute ventilation is

primarily due to an increase in TV (40% by term). The RR increases 15%. Progesterone may mediate the increase the respiratory sensitivity to CO2.

4. What is the significance of the decreased FRC?

It leads to more rapid arterial desaturation and may decrease the time required for inhalation induction. It also decreases the time required for preoxygenation to 2-3 minutes as measured by E'I'N2 concentrations. It may be prudent to extend preoxygenation times a bit longer, however, to increase the time to apneic desaturation and the fetal 02 stores.

5. What accounts for the increase in cardiac output during pregnancy?

CO equals HR x SV. The increase in CO is primarily due to an increase in SV (40% by term). The HR also increases 25%.

6. What changes of coagulation characterize pregnancy?

Anemia, leukocytosis, and hypercoagulability. There is no change in platelet count.

7. Do yon need to know the patient's NPO status?

Probably not since all parturient are considered full stomachs, especially if the planned procedure were emergent. However, if the procedure were elective, it may be reasonable to wait 6-8 hours to reduce (but no completely eliminate) the risk for aspiration.

8. The BUN and Cr are 19 and 1.3. Are you concerned?

Yes. Normal increase in RBF and GFR during pregnancy lowers the BUN/Cr to approximately 8/0.5. The patient's values are twice normal, suggesting that the GFR may be half normal. (Alternately, the BUN may be high from increase blood or protein in the GI tract and the Cr may be high from muscle breakdown).

9. When do the earliest physiologic chances of pregnancy occur?

Many begin in the first trimester, the effects of progesterone, estrogen, and human chronic somatotropin. The increase in minute’s ventilation begins by 6-7th weeks gestation, increase in blood volume and cardiac output by the 5th week, and LES pressure decrease in the first trimester. The decrease in FRC, the mechanical effect of upward displacement of the diaphragm, is not apparent until 5 months gestation.

Intraop:

1. Would you change your anesthetic dose for the parturient?

Yes. MAC is reduced by 40%. It is less clear whether dose requirements for IV anesthetics are similarly reduced.

2. Would you change your dose of epidural or spinal anesthetic?

Yes. The required dose for both spinal and epidural LA is reduced, due to the decreased epidural and Intrathecal spaces or increases sensitivity to LA.

3. Are parturient more difficult to intubate?

Statistically, yes. It has been suggested that intubation is 8 times more difficult than in the nonparturient.

Postop:

1. Why does the greatest risk for CHF occur in the postpartum period?

Because that is when the CO reaches its peak (150% above prepregnant values).

2. The patient is scheduled for a postpartum tubal ligation. Would you recommend a spinal or general anesthetic? Is she still at risk for aspiration?

There is no right, wrong, or best technique. The patient may still be at risk for aspiration since risk for aspiration (gastric volume, acidity, and emptying) may not have returned to normal. She may be relatively hypovolemic and anemic from blood loss. And due to the possible persistence of a low FRC she may be at risk for hypoxia. Due to these concerns, my first choice for most patients may be to perform a spinal, assuming no contraindications were present, but care should always be individualized.

Fetal Assessment and Monitoring

Preop:

1. When can FHRs be detected?

16 weeds gestation.

2. How would you assess fetal well being preoperatively?

a. First, I would obtain a history and physical, focusing on prenatal Care, the last medical assessment, and any prenatal problems.

b. The next simplest method is ask for a history of fetal body movements.

c. Finally, I would obtain a FHR tracing, looking for rate and variability.

d. Other methods include a NST OCT, or biophysical profile.

3. What if the FHR were 200?

This is fetal tachycardia (>160, the differential diagnosis for which includes hypoxemia (sympathetic stimulation), maternal fever (#1), terbutaline, maternal thyrotoxicosis, atropine, tachydysrhythmias, but not prematurity.

4. What if the FHR were 100?

This is fetal bradycardia (<120), the differential diagnosis for which includes severe hypoxia, uterine hypertonus, complete heart block, continuous head compression, hypothermia, postparparacervical block bradycardia (from direct fetal LA toxicity), but probably not propranolol or esmolol.

lntraop:

1. What is the significance of early deceleration?

They are normal findings that result from fetal head compressions causing parasympathetically mediated bradycardia.

2. Late decelerations are seen in the FHR tracing. What would you do?

Even though false alarms are common, late decelerations are worrisome, suggesting uteroplacental insufficiency.

a. First, I would determine if they were real as well as their severity. A FHR of 100 requires prompt treatment. A prolonged FHR of 60 requires emergency obstetric intervention (e.g., possible C/S). Loss of variability makes any degree of bradycardia more ominous.

b. Next, I would check ABCs. Is she receiving 100% O2 and well oxygenated? ls she in LUD and is her blood pressure adequate? Should fluids or ephedrine be given?

c. Then I would quickly determine any aspect of her condition had suddenly changed. Was a dose of LA just given through the epidural? Did the patient suddenly become SOB? Was an oxytocin infusion just begun?

d. The obstetrician should be informed and preparations made for a stat C/S.

3. What does loss of short-term variability mean?

It means that the normal 3-6-beat/min variation between every 2-3 beats from sympathetic/parasympathetic interaction is lost, consistent with asphyxia. The differential, however, includes atropine, Mg, benzodiazepiens, narcotics, phenothiazines, systemic absorption of epidural lidocaine, and anencephaly.

4. What is the significance of variable decelerations?

They suggest umbilical cord compression. They may be quite serious, especially if the decrease in HR is >60 beats/min, actual IIR is <60 beats/min, or deceleration lasts >60 sec duration (Robert Goodlin's rule of 60s"). The significance of variability between decelerations can be determined by looking at the variability and obtaining a scalp pH.

Antepartum Bleeding

Preop:

1. A parturient presents with painless vaginal bleeding. What are your concerns?

I am concerned about the possibility of placenta previa, which results from placental implantation in the lower uterine segment in advance of the presenting fetal part. The status of the mother and fetus should be immediately evaluated and the amount of bleeding determined.

a. If the mother and fetus are stable, conservative therapy (bed rest, transfusion, tocolytics) may be employed to allow fetal lung maturity.

b. If there is fetal distress in a viable fetus, a stat C/S may need to be performed. My role in that setting would be to obtain large-bore IV access, order cross-matched blood and blood products, and prepare for GA.

2. What if she presented instead with painful bleeding?

The diagnosis is then more likely abruptio placental premature separation of the placenta from the deciduas basalis or uterine rupture, both of which can cause pain as well as maternal and fetal instability. It may be necessary to obtain large bore IV access, transfuse blood, and prepare for an emergent C/S.

3. What are the risk factors for placental abruption?

HTN trauma, low-lying placenta, placenta previa, fibroids, trauma, cocaine abuse, cigarette smoking, lmultiparity, advanced age, physical work, stress, and previous abruption.

4. What are the risk factors for uterine rupture?

Previous uterine surgery, trauma (direct or indirect), excess oxytocin, multiparity, uterine anomalies or tumors, placenta percreta, and fetal macrosomia or malposition.

5. What are the risk factors for placenta previa?

Advanced age, multiparity, and prior C/S or uterine surgery. .

6. Hypotension and fetal distress are noted. What would you do?

It is likely that the fetal distress is secondary to the hypotension.

a. First, I would assess her respiratory and mental status. Is the patient awake, alert, well oxygenated, and breathing? 100% 02 should be provided.

b. Next, I would determine whether the hypotension is due to epidural LA, a sudden bleeding episode (uterine rupture. abruptio), an amniotic fluid embolus, seizure, intracranial hemorrhage, dysrhythmia, PE, drug reaction or anaphylaxis. LUD should be provided.

c. Whatever the cause, ephedrine and fluids should be given.

d. The obstetricians should be called and preparations should be made for a stat C/S under GA.

Intraop:

1. A stat C/S is required for fetal distress in a mother with placenta previa. What anesthesia technique would you use? .

If the fetal distress is due to bleeding from the previa, it is likely that the mother is hypovolemic, in which case GA is preferred. After quick assessment of the airway, I would preoxygenate, administer Ketamine and succinylcholine, and intubate with cricoid pressure. Any hypotension should be immediately treated with ephedrine, fluids, and, if associated with severe hemorrhage, blood transfusion

2. What would you use for maintenance of GA in the parturient with severe antepartum hemorrhage?

If the patient were moribund, I would use no anesthetic at all. But if she had only mild hypotension, I would use a narcotic based anesthetic with vecuronium. Low dose isoflurane could be added once the bleeding was controlled. Scopolamine may be given

to reduce the risk of recall.

3. Severe, unremitting bleeding occurs after delivery in a patient with placenta previa. What are your options?

a. First, make sure the patient's BP is adequate.

b. Next, determine if uterine atony is present, and if it is, administer oxytocin. IV methylergonovine or intramyometrial 15-methyl-PGF2-a may be considered next.

c. Any ongoing coagulopathy should be ruled out. (Even though DIC is more likely with abruptio, 10% of patients with placenta previa may also have abruptio.)

d. As a last resort, bilateral uterine artery or hypogastric artery ligation or hysterectomy may need to be considered, especially if severe placenta accreta is the problem. The decision to perform a hysterectomy is difficult, but cannot be delayed for long. Shock and massive blood transfusion, both potentially lethal, may ensue. (Despite the controversy of continuation of RA vs. inducing GA for accents, I would prefer GA if severe bleeding were expected.)

Postop:

1. What other serious complications may result from abruptio placenta?

a. Those relating to hypotension and hypoperfusion, such as renal failure.

b. Those relating to massive blood transfusion, such as coagulopathy, electrolyte abnormalities, and ARDS.

c. And those relating to tissue tltromboplastin release, such as DIC.

Postpartum Bleeding

Postop:

1. What is the most likely cause of postpartum bleeding?

Uterine stony, which may result from high parity; uterine overdistention (multiparity, polyhydrnmnios); chorionmnionitis; placenta previa; a prolonged, oxytocin-augmented, or precipitate labor; >1.0 MAC of potent invitational agent; uterine relaxants (isoxsuprine, terbutaline, Mg+, nitrites, nitroprusside, dantrolene, Ca channel blockers), or a full bladder.

2. After delivery, the uterine has increasing bleeding. What do you do?

a. First, make sure the patient is stable. If she is not, I would consider fluids, blood, and vasoactive agents as necessary.

b. If the cause of the bleeding is uterine atony, she may need oxytocin, methylergonovine (Methergine), and/or 15-methyl-prostaglantin F2-alpha (Hemabate).

c. Depending on the extent of the bleeding, I may need additional large-bore IV access, rapid infusing devices, or a cell-saver.

d. The surgeon may have to ligate the interal iliac or uterine arteries, and/or perform an emergency hysterectomy.

3. How do oxytocin and methylergonovine work?

Oxytocin promotes phosphorylation of myosin, allowing it to interact with actin. Ergot derivatives increase the intercellular Ca concentration.

4. Are there any contraindications to the use of methylergonovine?

Possibly. Methergine causes HTN, bronchocons, and coronary artery spasm, it should be used cautiously, if at all, in patients with preexisting HTN, asthma, or Cardiac disease.

5. After intramyometrial injection of methylergonovine. the patient develops chest pain. What is your response?

a. First, ABCs. She could be experiencing other side effects of which include HTN and coronary artery spasm.

b. Is vasospasm were occurring, I would administer' IV nitroglycerine. If severe HTN was also present, alternatives include nitroprusside and chlorpromazine.

c. I would also auscultate the chest because methylergonovine can cause bronchospasm.

6. After lntramyometrial injection of 15-methyl PGF2-a for uterine atony, the patient becomes cyanotic. What is your response?

First, ABCs. I would give 100% O2, listen to breath sounds, and check the vital signs. She could be experiencing bronchoconstriction from systemic absorption of inadvertent IV injection of the 15-methyl PGF2-alpha.

7. A patient with uterine atone develops pulmonary edema. What are you thoughts?

There are many etiologies, including those due to cardiologic and those due to noncardiogenic causes. One remote possibility that relates to the treatment of uterine atony is CHF from methergonovine-induced hypertension. Alternately, depending on the time course of the edema formation, excess oxytocin may, predispose to fluid retention and later pulmonary edema, especially with overzealous fluid administration.

8. Describe your anesthetic management for removal of retained placenta.

It depends on the severity of any postpartum bleeding.

a. If the bleeding is minor, IV NTG with/without sedation may be all that is required. Extension of a preexisting epidural to achieve a T8-l0 level allows uterine exploration without discomfort.

b. If the relaxation with NTG is insufficient, induction of GA with administration of potent invitational agents may become necessary.

c. If the hypotension does not allow administration of either nitroglycerine or potent incubational agents, Mg2+ may be chosen. Fluid and vasopressor necessary. B2 agonist tocolytics should be avoided became they may worsen the hypotension.

d. After removal of the fragments, the inhalational agents should discontinued (or at least reduced) and an oxytocin infusion begun (and continued postoperatively).

9. Following suspected amniotic fluid embolism, the pt continues to bleed. What tests should you order?

A CBC, platelet count, PT, PTT, fibrinogen, and FSP to rule out DIC and primary fibrinolysis.

Pregnancy Induced Hypertension

Preop:

1. What are your goals in managing a patient with PIH?

a. Preop: I would assess the fetus and mother control BP, administer antiseizure prophylaxis, assure adequate hydration and U/O, avoid overhydration and pulmonary edema, and assess coagulation status.

b. Intraop: I would establish epidural for labor or C/S, anticipate and try to avoid the exaggerated pressor responses, and avoid GA if possible.

c. Postop: I would remain vigilant for complications, including seizures and pulmonary edema.

2. What causes PIH?

The exact pathophysiology is uncertain, but the disease is one of maternal endothelial damage and placental ischemia, associated with vasospasm and abnormalities in at least 3 major cell types:

a. Trophoblasts - which fail to migrate into the maternal deciduas, allowing the maternal arteries to retain their adrenergic enervation.

b. Endothelial - which fail to produce prostacyclin and nitric oxide.

c. Platelets - which show increased aggregation, possibly due to an increase in the thromboxane/prostacyclin ratio.

3. What physiologic effects of PIH are you concerned about?

a. Cardiovascular - patients with mild or moderate disease may have an elevated CO and normal SVRs, severe cases with pulmonary edema may have a low CO and elevated SVR. Most patients are hypovolemic. The low colloid osmotic pressure, and increased vascular permeability predisposed to edema and hypovolemia. The CVP and PCWP may correlate, but imprecisely.

b. Hematologic - platelet activation, hypocoagulability, and increased fibrinolysis.

c. Renal - oligulia results from reductions in GFR. Proteinurina results from increased glomerular permeability. Rarely does renal failure occur.

d. Neurologic - HA, visual disturbances, and CNS hyperexcitability (hyperreflexia) occur. Intracranial hemorrhage is the #1 cause of death. The presence of seizures or coma suggests eclampsia.

e. Respiratory - upper airway and laryngeal edema predispose to a difficult airway. Pulmonary edema occurs due to the elevations in PCWP, decrease in COP, and excess fluid administration.

f. Hepatic - usually mild involvement, but distention, hemorrhage, ischemia, and rupture can occur win the HELLP syndrome

g. Fetal - may suffer from uteroplacental insufficiency.

4. What laboratories would you require for a patient with PIH?

It depends on the severity of the disease.

a. For mild disease, an H/H, platelet counts, electrolytes with BUN and Cr, LFTS (up to 20% of pts have elevated transaminase levels), UA.

b. For more advanced disease, PT/PTT, fibrinogen and FSPs, CXR may be helpful. A type and cross may be required if a C/S is planned.

5. The patient is hypertensive and oliguric. Is administration of fluids a good idea?

Perhaps. Often after restoration of adequate intramuscular volume, vasoconstriction decreases and BP declines. I would administer fluids in small increments while assassin vital signs, saturation, and listening for pulmonary crackles.

6. The UO is only 20 cc/hr in a pt with PIH. What would you do?

a. First, I would obtain a history and physical, looking for signs of dehydration, hemodynamic instability, and pulmonary edema.

b. If the pt seemed dehydrated and no pulmonary edema were present, I would consider a fluid challenge, say 250 cc of crystalloid.

c. Additional boluses could be given gradually, with continual checks of the VS, saturation, and lungs for crackles.

d. If, after 1000-2000 cc there were still no urine output, I would discuss the case with the obstetrician, who may want to perform a C/S for severe PIH.

e. If, at any time, pulmonary edema became a concern, I could insert a CVP or obtain an echocardiogram. If the CVP were low (or echo suggested hypovolemia with good LV function), I would give more fluid.

f. If pulmonary edema were to develop, especially with hypotension, I may consider inserting a PAC, although the condition would have to be severe. Actually, I would prefer to obtain a less invasive and more informative echocardiogram to assess volume status and contractility instead.

7. Why is the patient receiving Mg ? Any anesthetic implications?

Mg^{2+} is the prophylactic anticonvulsant of choice. Its use has several anesthetic implications, including:

a. Potentiation of neuromuscular blockade (for depolarizing and nondepolarizing relaxants), weakness,

b. Decreased uterine tone, prolonged labor,

c. Excessive blood loss,

d. Decreased FHR variability and neonatal depression, and

e. Exaggerated hypotension with epidural that does not adversely affect uterine blood now.

f. Respiratory arrest, asystole at toxic doses,

8. The BP is 200/100. What antihypertensive would you recommend?

a. Assuming this value is not an emergency, I would start with hydralazine, an arteriolar dilator with years of proven efficacy, the major side effect for which are reflex tachycardia.

b. If that were ineffective, I would consider (abetolol, a mixed alpha- and beta-

blocker (1:7 ratio IV) that also offers some HR control.

c. Other choices include nitroprusside, nifedipine, atenolol, and metoprolol.

9. Is a labor epidural a good idea for the parturient with PIH?

Yes. It provides:

a. Superior analgesia,

b. Attenuates hypertensive responses to pain,

c. Reduces circulating catecholamines,

d. Improves intervillous blood flow, and

e. A route for providing anesthesia for a possible C/S.

10. Would you prehydrate the patient with PIH before epidural placement?

Yes, although the fluid should be give more cautiously due to the concern of pulmonary edema (e.g., give 250-500 cc at a time instead of 500-1000 cc). (Paradoxically, the higher the diastolic BP, the lower the intravascular volume and greater the fluid requirements of the patient. Such patients may also be predisposed to developing pulmonary edema.)

11. Do you have a minimum platelet count below which you would refuse to place an epidural?

No. Placing an epidural catheter in a patient with PIH is a risk/benefit decision.b I don't have an absolute cutoff, but 100K is probably too high. Instead of relying on an absolute number, I prefer to use the change in plt count, signs of clinical coagulopathy, and need for regional anesthesia to decide.

12. So it's safe to proceed with an epidural if the plt count were <100?

Perhaps. In favor of proceeding at lower values (50-100K) is the fact that there are few reported cases of a neuraxial bleeding from neruaxial technique occurring as a result of thrombocytopenia in patient with PIH. I'm not saying there's no risk; it's just that the 100K cutoff is probably too high and the decision to place an epidural should be individualized.

13. What local anesthetic would you use for the epidural?

I would use bupivacaine or ropivicaine because they causes less motor block than lidocaine at equipotent concentrations and dilute solutions (0.125% and 0.1%, respectively) cause little hypotension.

14. What would you give to treat hypotension after an epidural?

Because of possible exaggerated pressor responses in the pt with PIH, I would give a small dose of ephedrine (2.5-5 mg) if the blood pressure drop were severe.

Intraop:

1. During an C/S under epidural anesthesia, the pt with PIH complains of LLO incisional pain despite prior epidural administration of 20 cc of 2% lidocaine. What would you do?

If there was time, additional LA and/or fentanyl could be administered through

the epidural. In addition, I would ask the surgeon to infiltrate the incision with LA, administer Ketamine and then consider using N2O. Ketamine given in small doses (0.25mg/kg) should not cause additional hypertension. If these measures were ineffective, I may have to induce GA.

2. How would you induce GA in a pt with PIH for an elective C/S?

My main concern is the potential for difficult intubation and prevention of aspiration and hypertension. After prior assessment, I would preoxygenate administer lidocaine 1.5 mg/kg, alfentanil 10 mg/kg, thiopental 5-7 mg/kg, succinylcholine, cricoid pressure, and intubate once the patient was relaxed. If the BP were extremely high(>160/110) for an elective case, I may try to lower it with labetolol and fluid before induction. If it were high at the time of an urgent or stat induction, I may insert an intra-arterial catheter before proceeding and titrate additional doses of thiopental or nitropprusside as required following intubation.

3. The BP rises to 300/150 following intubation of a pt with PIH. Would you give labetolol?

I would consider it, but its peak effects take 10 minutes, the necessary dose is hard to predict, and it is unreliable. A BP of 300/150 is critically high, and if it were real and due to inadequate anesthesia, I would prefer to give a bolus of thiopental or propofol, followed by 25-50 mcg doses of SNP as necessary. As both an alpha and beta-blocker (ratio l :7), labetolol can often be used to treat hypertension following intubation.

4. The pt has a seizure. How would you respond?

The treatment consists of:

a. Airway/breathing-- I would bag/mask ventilation with cricoid pressure and the assess adequacy of oxygenation and ventilation. If the seizures were refractory, I may need to intubate the pt's airway and control ventilation to prevent aspiration.

b. Circulation management -- I would control of BP with fluids, pressors, or hydralazine.

c. Seizure control -- Seizures in the pt with PIH should be assumed to be eclampsia until proven otherwise. I would administration of Mg2+, 4-6 g over 20 min, then 1-2g/hr with additional doses of 2-4g over 5 min as necessary. More acutely, I would use thiopental or midazolam.

5. The seizure continues, do you intubate her? Do you give muscle relaxant to stop seizure?

Yes, I would intubate her to provide oxygenation and ventilation and protect her airway from aspiration. If the seizure were prolonged, I would also administer relaxants to decrease the metabolic consequences of sustained muscle contraction, which include hyperthermia, metabolic acidosis, and hyperkalemia. However, I would obtain a stat EEG or keep the patient at one twitch to assess the effectiveness of continued seizure treatment.

6. The patient is intubated. 30 min later, she desaturates to 90%, there is pink frothy secretions coming out from ETT. How so you evaluate and manage?

I would suction out the froth, ventilate with high peak airway pressures, add PEEP, assess the BP and HR, and consider LV failure vs aspiration vs negative pressure pulmonary edema. Depending on the severity of any hyper-/hypotension and amount of fluids given, I may consider a dose of furosemide with invasive monitoring or an echocardiogram.

7. CXR comes back, showing bilateral infiltrates, pulmonary edema (PEd). What do you think and how do you manage it?

It seems like the patient has pulmonary edema, the treatment for with depends on the severity and cause. If the pulmonary edema were severe, I would provide mechanical ventilation with PEEP. If my evaluation pointed to a cardiogenic cause (e.g., myocardial ischemia, poor myocardial function), I may provide us diuretics, inotropic agents, beta-blockers, and afterload reducing agents (if HTN were present). If a noncardiogenic cause were more likely. I may simply provide diuretic therapy and fluid restriction.

Postop:

1. Is pulmonary edema more likely to occur pre- or postpartum?

Postpartum. This could be due to decompression of the IVC by the formerly gravid uterus, resulting in increased venous return to the heart. According to one estimate, 70% of cases of pulmonary edema occurred after delivery.

2. The pt still seems weak at the end of GA. Why?

There are many causes for apparent postop weakness, but 2 that relate specifically to PIH are Mg 2+ toxicity and a neurologic event, such as CVA or post-ictal state. The patient's neuromuscular response to twitch stimulation and neurologic status should be assessed.

3. Why does Mg 2 + cause weakness?

It inhibits presynaptic acetylcholine release, motor endplate depolarization, and muscle fiber excitability.

4. After delivery the baby, the neonate is not crying and becomes apneic. The adult patient is stable. Now you are requested for the neonate resuscitation, what do you do?

If the mother were stable, then I would have the nurse watch the mother and call out her vital signs while I helped in resuscitating the neonate.

5. Do you intubate the neonate?

I would intubate the neonate if bag mask ventilation were ineffective, prolonged mechanical ventilation were anticipated, chest compressions became necessary, drugs had to be given down the ETT, and significant meconium aspiration.

6. The neonate's HR is less than 60. There is no IV. Do you give epi via ETT? How much?

Yes, I would give epinephrine down the ETT if, after 30 seconds of adequate chest compression and effective ventilation with 100% O2, the HR were still <80. The

dose is 10-30 mc/kg (or 0.1-0.3 cc/kg of a 1:10,000 solution, which contains 0.1 mg/ml…in other words, rounding up, it's about 1cc containing 100 mc for a 3 kg neonate). To ensure proper distribution, the epi can be diluted with 1-2 cc of NS.

7. After extubation, the patient is transferred to PACU. Suddenly, the patient develops seizure. What do you think and what do you do?

My first thought is eclampsia, but hypoxia, hypotension, intracranial hemorrhage, local anesthetic toxicity (if a dose had just been inadvertently given IV) should be considered. I would provide oxygen, support the airway, treat with propofol, thiopental, or midazolam (depending on what was available and her hemodynamic status), provide bag-mask ventilation and consider intubation, as necessary.