E. MEDICAL COMPLICATIONS OF PREGNANCY AND RELATED POSTPARTUM/NEONATAL PROBLEMS

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HYPERTENSIVE COMPLICATIONS OF PREGNANCY

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BACKGROUND

Hypertensive disease complicates 6-22% of all pregnancies, depending on the population studied, and is the third leading cause of maternal death worldwide. It accounts for 18% of maternal deaths in the United States and, because it often prompts the need for preterm delivery, it is a significant cause of perinatal mortality as well. Every provider caring for pregnant women needs to be familiar with the unique aspects of this disorder and understand that this is not necessarily the same entity familiar to them in the non-pregnant population.

DEFINITIONS

The National High Blood Pressure Education Program Working Group on High Blood Pressure in Pregnancy recently published new evidence-based guidelines on the classification, pathophysiology, and management of this disorder. This classification defines and stratifies the hypertensive disorders into four categories:

- Chronic hypertension
- Preeclampsia-eclampsia
- Preeclampsia superimposed on chronic hypertension
- Gestational hypertension

Chronic hypertension is defined as hypertension that was present prior to pregnancy or diagnosed prior to 20 weeks gestation. Hypertension is defined as blood pressure (BP) $\geq$140 mm Hg systolic or $\geq$90 mm Hg diastolic.

Preeclampsia-eclampsia is defined as gestational blood pressure elevation accompanied by proteinuria. This entity can be diagnosed after 20 weeks gestation in a woman with the same $\geq$140/90 BP criteria who also has $\geq$1+ proteinuria (>30 mg/dL) on dipstick, or $\geq$0.3 g on a 24 hour urine collection.

Preeclampsia superimposed on chronic hypertension is diagnosed in the woman with known non-proteinuric hypertension at <20 weeks who develops new onset proteinuria as defined above.

Gestational hypertension is an interesting entity diagnosed by blood pressure elevation after mid-pregnancy without proteinuria.

This classification has important clinical management implications that will be discussed in detail below. In general, preeclampsia should always be suspected, since it has the worst maternal and perinatal outcomes and, at the present time, can only be cured by delivery. The long-term outcome of women with each of these disorders is also different, and may have significant future long term public health implications as well.

There are some key details relevant to this classification. Diastolic blood pressure is defined as the disappearance of sound (Korotkoff phase V), not muffling of sound. Blood pressure should be measured with the patient in the sitting position and confirmed by repeating after a period of rest. The pressure will almost always decrease with the patient in the lateral decubitus position, and

while this is the therapeutic position, it will falsify the diagnosis. It was previously recommended that an increment of 30 mm Hg systolic or 15 mm Hg diastolic BP from first trimester reading should be used as a diagnostic criterion, but the evidence does not support that such women will have adverse outcomes and is no longer used. Proteinuria should not be diagnosed in a woman with a current urinary tract infection. 24-hour urine collections are more accurate than dipstick protein determinations, but are cumbersome, time-consuming, and do not always represent complete collections. A random spot urine collection, corrected for creatinine excretion, the protein/creatinine ratio, has a correlation coefficient of .90 with the 24-hour collection, is user-friendly, and expeditious. Values >0.20 have comparable diagnostic value for preeclampsia as 24-hour collections with >300 mg. Edema has also been eliminated as a diagnostic criterion for preeclampsia, as most normal women in late pregnancy will have it to some degree, thus making it of limited discriminant value. Nevertheless, rapid short-term weight gain may be an important first clue that the syndrome is about to manifest itself. Notice that besides proteinuria, there are *no* laboratory markers of preeclampsia. Some laboratory studies may be of key importance as ancillary guides to management (see below), but they cannot be used to make the actual clinical diagnosis. In particular, the diagnostic value of uric acid determinations, while classic teaching, has neither specificity, or sensitivity, or prognostic value for this disease.

PREECLAMPSIA-ECLAMPSIA

Classification

This entity will be discussed first, as it is the more common and most serious clinical problem. An understanding of its pathophysiology is very useful in order to understand its differentiation from the other classifications, as well as its appropriate management. Preeclampsia may be defined as mild or severe.

Mild preeclampsia is defined as above: systolic >140 and diastolic >90, accompanied by >1+ proteinuria. The majority of patients will only have mild disease, but an important, and largely unpredictable, subset will progress to the severe syndrome, thus mandating constant vigilance once the diagnosis is established. Severe preeclampsia is a life-threatening disease and requires delivery, regardless of gestational age (more on this later) in order to be reversed.

Severe preeclampsia is defined by one of the following criteria:

- Blood pressure$\geq$160 mm Hg systolic or $\geq$110 mm Hg diastolic
- Proteinuria of >5.0 g on a 24-hour collection (3-4+ on dipstick)
- Oliguria of <500 mL/24 hours and/or serum creatinine >1.2 mg/dL
- Thrombocytopenia (<100,000 mm^3) with elevated hepatic enzyme activities and/or evidence of microangiopathic hemolytic anemia (“HELLP” syndrome), usually accompanied by epigastric pain
- Cerebral or visual symptoms (headache, scotomata), seizures *(eclampsia)*
- Pulmonary edema
- Fetal growth restriction

You will notice that various end organs are targeted here: the vasculature itself, the kidney, the liver, the central nervous system, the cardiopulmonary system, and the uteroplacental unit. Severe preeclampsia is actually a systemic disorder with multi-organ dysfunction. Failure of any one of them can lead to maternal or fetal death. Therefore, once severe preeclampsia is diagnosed, the patient needs to be delivered. Reflective of this, most (but not all!) patients who are developing severe preeclampsia will feel ill, have symptoms, and show a change in their clinical picture from when they were diagnosed as having mild preeclampsia. This progression may never occur, occur slowly over days to weeks, or occur in a fulminant fashion over hours. A

major goal in the management of mild preeclampsia is the recognition and prevention of severe preeclampsia.

Pathophysiology and Etiology

The etiology of preeclampsia is still unknown, although many interesting hypotheses have been advanced. The pathophysiology of the disease however, has been fairly well elucidated. The final common pathway in the development of severe preeclampsia is currently thought to be generalized vascular endothelial dysfunction. This results in arterial vasospasm (clinically manifested as hypertension, oliguria, etc.), increased capillary permeability (clinically manifested as proteinuria, cerebral edema, pulmonary edema, etc.), and actual endothelial lesions with platelet activation (clinically manifested as HELLP syndrome, abruptio placentae, etc.). The net effect is downstream hypoperfusion with resultant hypoxic-ischemic damage to one or more target organs. Hypertension is really not the most significant abnormality of this entity; under-perfusion is what accounts for its most serious sequelae. This concept is very important in implementing appropriate management.

The inciting event is theorized to occur soon after conception and is the result of defective placentation with poor vascularization of the trophoblast. This may be the result of immune maladaptation between the maternal host and the fetal partial allograft, genetic predisposition, and vascular mediated factors. Preeclampsia primarily affects two subpopulations of women: young nulliparas and older multigravidas. There is evidence to suggest “primipaternity” rather than “primiparity” is the problem, as women who conceive soon after initiating sexual activity, and those later in life who acquire a new partner, seem to be the most susceptible, presumably as a result of not acquiring tolerance to new male antigens. Vascular factors may also play a key role in maternal susceptibility as women with chronic hypertension, diabetes, underlying renal disease, vasculitis (e.g., lupus), have a several-fold increased incidence of the disease compared to well women. Certain families also seem to have a genetic tendency to preeclampsia. This is well seen in certain Navajo kindreds where there exists a strong family history of the disease in multiple maternal female relatives. Whatever the predisposing factor, the trophoblast presumably fails to establish normal remodeling of the spiral arteries at the maternal interface, and responds to the relative ischemia by the production of cytokines that eventually damage the maternal vascular endothelium and result in the clinical syndrome we know as severe preeclampsia.

Management

- **Antenatal**

As noted above, preeclampsia will not resolve while the placenta still functions, thus delivery remains the only “cure”. The decision to deliver a woman with preeclampsia however depends on a balance of both the maternal and fetal risks. Continued observation is appropriate for the woman who is remote from term as long as her disease remains mild. This requires frequent periodic evaluations, usually twice weekly, and the woman may be managed at home if logistically feasible. Hypertensive disease is one of the few conditions where bed-rest is an evidence-based intervention, as the recumbent position usually will be helpful to optimize uteroplacental and renal blood flow and keep the blood pressure within acceptable limits. If adherence to a bed-rest regimen is difficult, hospitalization may be the best plan. Sodium restriction is futile as this mechanism is not operant in this disease the way it is in essential hypertension. The physiology of pregnancy is set up to raise cardiac output to meet the demands of the fetoplacental unit. This is accomplished by expanding stroke volume (preload) through retention of sodium and water, and this potent pregnancy-specific physiology cannot be readily reversed. Pharmacotherapy also has very little place in the management of this disorder. “Prophylactic” anticonvulsant therapy or maternal sedation with phenobarbital is of unproven benefit and may have adverse fetal effects. It is probably inappropriate to try to lower the

elevated BP with oral anti-hypertensive therapy. Again, this is not the same disease that non-pregnant hypertensive patients have! If the pressure is >160/110, the patient needs to be delivered. If the pressure remains in the non-severe range, and all else is stable, reduction of perfusion pressure will only have adverse effects on the already compromised uteroplacental perfusion. This concept is somewhat different for women with chronic hypertension, as will be discussed below.

In addition to frequent evaluation of maternal blood pressure, weight, proteinuria, reflexes, and symptoms, an initial baseline laboratory assessment including CBC (hemoconcentration?), platelet count (thrombocytopenia?), creatinine (normal renal function?), and liver enzymes (signs of early hepatic involvement?) is useful. This panel may be repeated weekly, but as noted above, the diagnosis of preeclampsia is largely clinical, and patients who are developing severe preeclampsia will usually manifest it by the development of symptoms, not early changes in biochemical parameters. Is there a place for "conservative" or expectant treatment of *severe* preeclampsia remote from term? It is controversial, but probably, yes, especially to give time for the administration of antenatal glucocorticoids for fetal lung maturation at <32-34 weeks gestation, but it should only be attempted at the Level III Center where the patient will be delivered. ✱

Recommendations for fetal surveillance depend on the severity of the disease and are largely formulated on the basis of "expert opinion" rather than the results of randomized trials. For women with mild preeclampsia, a baseline ultrasound examination for fetal growth and amniotic fluid index (AFI) should be obtained at diagnosis. Non-stress testing should be initiated and repeated on a weekly basis, but if fetal growth restriction or oligohydramnios are diagnosed (more common in women with chronic hypertensive, not "pure" preeclampsia), testing should be carried out twice weekly, or more frequently depending on fetal status. Testing should be repeated at any time there is an abrupt change in maternal condition. Placental abruption always remains a threat to the fetus of the hypertensive woman.

- **Intrapartum**

There is probably no benefit in delaying delivery once the patient has reached term. With the introduction of the current prostaglandin preparations, the "unripe" cervix is usually no longer a major obstacle to promptly establishing labor. Any deterioration of maternal or fetal condition with the development of severe preeclampsia prior to term is an indication for prompt delivery. There is no demonstrated benefit to cesarean delivery for other than the usual obstetric indications. Rushing to operation in the unstable severe preeclamptic may be hazardous. Induction of labor is safe with either oxytocin or prostaglandins. ✱

Magnesium sulfate ($MgSO_4$) is the mainstay of intrapartum therapy in the United States for both mild and severe preeclampsia. It is primarily used for the prevention of seizures, but has many other advantages and is key for stabilizing the patient. Contrary to traditional teaching, MgSO4 *is* a vasodilator, and, because it is a calcium-channel blocker, it is able to reverse vasospasm, lower blood pressure, and enhance perfusion. In addition to lowering the seizure threshold through its interference with the function of the excitatory neuronal NMDA (N-methyl d-aspartate) receptors, it is also an especially effective cerebral vasodilator and will help reverse the hypoxic-ischemic process that sets up the seizure focus. Other anticonvulsants, such as phenytoin, have been used for seizure prevention, but they do not have the other beneficial effects of magnesium, and the data clearly show an advantage to the use of the latter. Outside the United States, MgSO4 is only used for severe, not mild, preeclampsia, because it has been demonstrated that over 500 mild preeclamptic women have to be treated to prevent one seizure. (One in 30 *severe* preeclamptic women may seize if untreated.) Nevertheless, because of its ancillary salubrious effects on the disease process, it remains the drug of choice for all patients with the disease in this country.

Magnesium sulfate is usually administered intravenously. A loading dose of 4-6 g diluted in 100 mL D5W is given over 15 minutes, and is followed immediately by a maintenance drip of 1-3 g per hour. Urine output, deep tendon reflexes, respirations and O_2 saturation are probably better parameters to follow than serum magnesium levels. Magnesium is a simple cation and is almost completely eliminated by the kidneys in about 15 minutes in women with normal urine output, so the maintenance drip needs to be started immediately after the loading dose in order to achieve the therapeutic effect. Likewise, women with an elevated creatinine (normal in pregnancy is 0.4-0.6 mg/dL, secondary to enhanced glomerular filtration), or poor output, need more vigilance, and utilization of the lower end maintenance rate. Because it is a calcium channel blocker, MgSO4 will also have effects on skeletal muscle, particularly the accessory respiratory musculature, and O2 saturation monitoring is very helpful. This is especially true if renal function is compromised. MgSO4 may also be given intramuscularly (at a dose of 10 g mixed with 10 mL of 1% lidocaine and divided between the buttocks) if IV access is logistically problematic, but it is painful and levels may be somewhat unpredictable.

Despite popular wisdom, the evidence demonstrates that the length of labor is not different between preeclamptic women "on mag" and parturients being induced for other reasons. However, labor may be more difficult to initiate, and postpartum hemorrhage is definitely increased because magnesium also blocks calcium entry into uterine smooth muscle. Be prepared for postpartum hemorrhage and remember that ergot preparations, such as methergine, are potent vasoconstrictors and are contraindicated in preeclamptic women. Oxytocin or prostaglandins should be used for uterine atony. There is little evidence about the optimum length of time MgSO4 should be continued postpartum, but 12-24 hours is usually sufficient, depending on the severity of the disease process. Remember that in as many as 2-4% of women, eclampsia may occur for the first time in the 48 hours following delivery. Monitor urine output carefully as postpartum diuresis may be delayed for several days, and magnesium toxicity may develop if the dose is not adjusted appropriately.

Fluid balance is one of the most critical aspects of the care of the severe preeclamptic woman, because of the fluid compartment shifts so common to this disease. Starling's law of the capillary is helpful to remember here: the low oncotic pressure (secondary to heavy proteinuria) and the high hydrostatic pressure (secondary to hypertension) typical of this disease favor movement of fluid out of the intravascular space and into the interstitial space. On admission patients are thus typically intravascular volume depleted and hemoconcentrated with poor urine output and exaggerated vasospasm. Therefore initial rapid hydration with 1-2 L of crystalloid is usually helpful to reverse these effects and, often of itself, will foster lowering of the blood pressure. Thereafter, however, fluid will continue to leak into the interstitium and predispose the patient to cerebral or pulmonary edema, so cautious fluid administration is in order, usually 100 mL/hour total fluids, enough to maintain urine output at or slightly above 30 mL/hour. Administration of dextrose in water will only increase "third-spacing" and Ringers or saline should be the solution of choice. Administration of colloids would seem to be a logical choice in this setting, but they have not been shown to be superior to crystalloid and are significantly more costly. "Compulsive" I&O's are critical! Adequate hydration is especially important prior to administration of parenteral anti-hypertensive agents or regional anesthesia because a sudden drop in vascular resistance without volume expansion is very likely to provoke placental hypoperfusion and resultant fetal distress.

Parenteral anti-hypertensive agents are required in very few women if hydration and magnesium sulfate have been administered to treat the pathophysiology detailed above. For those women whose blood pressure has not been lowered below 160/110 by those measures, hydralazine or labetalol may be considered. Hydralazine is a direct arteriolar vasodilator and is the classic drug for this indication. It is administered in 5 mg IV boluses 20 minutes apart, or in a drip of 25 mg in 250 mL of saline in a pump at 15 mL/hr (0.025 mg/min), increased by 15 mL/h q20 min as needed. The goal is to lower the diastolic pressure to 95-100 mm Hg, not make the patient normotensive. Fetal distress is commonly produced by underestimating the cumulative effect of

this drug. Labetalol is a beta-1 blocker (lowers heart rate and inotropy), a beta-2 agonist (vasodilates), and an alpha-1-antagonist (reverses vasoconstriction). It is not associated with the abrupt drop in pressure commonly seen with hydralazine. Begin with an IV bolus of 10 mg and double the dose (20 mg, 40 mg) every 5-10 minutes until the diastolic pressure comes into the 95-100 range. Continue 40 mg doses until either 200 mg has produced no effect or 300 mg has been given total in 24 hours. About two thirds of patients will respond to this agent. Adding a small dose of hydralazine for those women to whom you have already administered labetalol without the desired effect will often be effective. Postpartum, any agent is usually acceptable. Angiotensin converting enzyme inhibitors probably work best for the pathophysiology of the disease, but are relatively contraindicated in breastfeeding mothers. Nifedipine 10 mg orally q 4-6 h prn diastolic BP>110 is also usually very effective, but remember, combining it with MgSO4, another calcium channel blocker, may potentiate the effects of the latter. *

The long-term prognosis for women who have experienced preeclampsia is somewhat population dependent. For Caucasian women, a 10% recurrence of the disease in the next pregnancy is possible, and the incidence of chronic hypertension later is also about 10%, no different than that of the general population. These numbers are doubled for African-American women however. No data are specifically available for Native American women. Women with early onset preeclampsia (late second or early third trimester occurrence) may have up to a 40% recurrence rate in a subsequent pregnancy. Multiparous women and women who have experienced recurrent preeclampsia have a much higher incidence of eventual chronic hypertension.

Is preeclampsia preventable? To date the answer is no. Large randomized trials of both low dose aspirin supplementation and calcium supplementation have shown no benefit. Negative results for fish oil supplementation have likewise been the result in three smaller trials. Antioxidants (vitamins C and E) have shown an effect in one trial, but this awaits confirmation. Smoking actually reduces the incidence of the disorder, but has obvious other negative general and fetal health aspects. A high protein diet has not been shown to be of benefit. These results are probably to be expected. A disease with this complex a pathophysiology that may commence at the time of conception and may have a genetic component would not be expected to respond to a single simple intervention late in gestation.

CHRONIC HYPERTENSION IN PREGNANCY

Women who enter pregnancy with high blood pressure are obviously different from those who later develop the preeclampsia syndrome. Nevertheless, their underlying disease may complicate their pregnancy, and up to one in three of them will develop superimposed preeclampsia. Likewise, a third of them may have a small for gestational age infant and they are at increased risk for preterm birth, placental abruption, and perinatal death. If significant renal disease is part of their picture (creatinine >1.4 mg/dL), these risks will all be exaggerated. The normal physiology of pregnancy may be expected to lower blood pressure in most of these women. As noted above, pregnancy's physiology is geared to enhancing cardiac output to meet the needs of the fetoplacental unit, and a reduction of systemic vascular resistance (afterload) is a sentinel event beginning in early gestation. Thus, most women with mild blood pressure elevation, who are already on anti-hypertensive medications, can usually have them stopped in early gestation without ill effect.

There is no evidence that anti-hypertensive therapy will improve perinatal outcome for chronically hypertensive women, and most studies show an increase in fetal growth restriction in women on such medications. This is especially true for the beta-blockers, with the exception of labetalol. Methyldopa and hydralazine have the best "track records" and are the preferred agents. ACE inhibitors have been implicated in causing fetal renal failure (after the first trimester) and are contraindicated. There is little data on calcium channel blockers, but what does exist shows neither deleterious nor advantageous effects from their use. Diuretics are not contraindicated,

but the aforementioned physiology of pregnancy with respect to volume expansion would seem to militate against them having much effect. The goal is to maintain perfusion and keep the diastolic pressure in the 90-100 mm Hg range. As already noted, the evidence does demonstrate that bed rest is an effective therapy if the patient is willing and able to adhere to such a regimen.

Appropriate baseline studies in women with chronic hypertension include an evaluation of preexisting target organ damage. Renal function can be assessed with a 24-hour urine protein and creatinine excretion, and/or a serum creatinine and a spot urine protein/creatinine ratio. If secondary hypertension is suspected, a renal ultrasound may have a small yield. Ophthalmoscopy can determine if any preexisting retinopathy is present. An EKG to look for the presence of left ventricular hypertrophy would be informative in order to anticipate the possible emergence of diastolic dysfunction later in the pregnancy. A baseline ultrasound for dating the pregnancy is important, and follow-up scans, beginning at 28 weeks and continuing monthly until term should be obtained to look for fetal growth restriction. Weekly non-stress testing after 32 weeks may be prudent, but there is little objective evidence of its benefit. If fetal growth restriction, oligohydramnios, or superimposed preeclampsia is diagnosed, however, twice weekly NST should be instituted at that time. The uncomplicated patient can be delivered at term, but if the above problems are diagnosed, timing of delivery will depend on their severity. The management of superimposed preeclampsia is the same as detailed above. Remember that preexisting renal or cardiac damage may significantly complicate the course and management of severe preeclampsia in these women. *

GESTATIONAL HYPERTENSION

The woman who has blood pressure elevation unaccompanied by proteinuria that develops in the second half of pregnancy is classified as having “gestational hypertension”. A significant proportion of these women will go on to develop the preeclampsia syndrome and so more frequent monitoring is essential. If proteinuria does not develop, these patients can be delivered at term. If blood pressure elevation persists beyond 3 months postpartum, the patient is diagnosed with chronic hypertension. In the absence of proteinuria, perinatal outcomes are good. Interestingly, and unlike typical preeclamptics, a significant number of these women will go on to develop essential hypertension later in life.

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ILLUSTRATIVE CASES

Case #1.

Angie Igaruk is a 17 y/o G1P0 at 37 weeks 4 days by her LMP and an early ultrasound. She presents to a Level I facility that offers obstetric services during a heavy snowstorm complaining of uterine contractions. She denies headache or visual changes. Her prenatal course to date has been uneventful. Examination reveals a BP of 154/94, 1+ proteinuria, 2+ edema, 3+ DTRs. The uterine fundus measures 36 cm and the cervix is 4 cm dilated, 90% effaced, with the vertex at –1 station. FHR is reassuring. Your BEST plan of management at this time would be:

a) bed rest and methyldopa 500 mg po tid and return when more active
b) 24-hour urine collection for total protein and overnight observation
c) corticosteroids for fetal lung maturation and transport to next level of care when the weather clears
d) hydration, AROM, and oxytocin and magnesium sulfate intravenously

Case #2.

Ida Joe is a 38 y/o G8P6 at 33 weeks 2 days by a "sure" LMP. She presents to your Level I facility that offers obstetric services for her first prenatal visit and has no complaints. Your exam reveals a BP of 171/98, 4+ proteinuria, 1+ edema, and 1+DTRs. The uterine fundus measures 28 cm and the cervix is floppy and fingertip dilated with a floating vertex. A normal FHR is recorded. Her creatinine is 1.3 mg/dL, hematocrit 29 vol %, platelets 305,000 mm^3. An ultrasound gives a gestational age of 30 weeks 5 days and the abdominal circumference is significantly smaller than the other parameters. AFI=8.9. She states she has been told last pregnancy she had high blood pressure, but doesn't know what all the fuss is about. Your BEST plan of management would be:

a) bed rest and methyldopa 500 mg po tid and return in 2 weeks
b) 24 hour urine collection for total protein and overnight observation
c) corticosteroids for fetal lung maturation and transport to tertiary care center
d) hydration, AROM, and oxytocin and magnesium sulfate intravenously

Case #3.

Mary Tall Boy is a 23 y/o G2P0 at 35 weeks 3 days by her LMP and an early ultrasound who has had a normal prenatal course to date. She presents to your Level II facility complaining of a severe retro-orbital headache of 2 days duration and states she is seeing spots in front of her eyes. Exam reveals a BP of 172/112, 3+ proteinuria, 2+ edema, and 4+ DTRs. The uterine fundus measures 34 cm and the cervix is closed with a floating breech presentation. The FHR strip is reassuring. Her hematocrit is 43 vol %; platelet count 135,000; creatinine 0.9 mg/dL; liver functions are normal. As the admission paperwork is being filled out, the patient is observed to have a generalized tonic-clonic seizure. A prolonged 5-minute deceleration of the fetal heart to a rate of 70 bpm occurs following the seizure, but eventually returns to baseline. Your best plan for IMMEDIATE management would be:

a) attempt external version and start oxytocin
b) immediate cesarean delivery
c) hydration, intravenous magnesium sulfate, type and screen
d) corticosteroids for fetal lung maturation and transport to level III care

CASE DISCUSSIONS

Case #1. **(D)**

Ms Igaruk is a mild term preeclamptic. While it would be ideal to be able to transport her to the next level of care, that is not feasible at this time and she is in active stage labor. At this time she will be best served by treatment with magnesium sulfate and expeditious delivery at your facility.

Case #2. **(C)**

Ms Joe is probably a chronic hypertensive with underlying renal disease. Her fetus is also probably suffering from growth restriction. Nevertheless, you cannot be sure from the data available that she is not a severe preeclamptic. Your best plan would be to begin steroids in anticipation of delivery and transport her to a facility equipped to care for an at risk fetus of this gestation age.

Case #3. **(C)**

Ms Tall Boy is a severe preeclamptic near term with a breech presentation who has now developed eclampsia. She does not appear to have HELLP syndrome or have a need for platelet transfusions at this point. Her fetus is at an age where steroids are not indicated because of the low risk of developing IRDS. She needs immediate stabilization to try to reverse her hypoxic-ischemic encephalopathy and prevent another seizure, and then be expeditiously delivered. During the maternal apnea and sustained muscle contraction that accompanies seizure activity it is common to see prolonged fetal bradycardia that eventually resolves as the seizure is terminated. The maternal cerebrovascular autoregulation is extremely unstable at this time, and the stress of rushing to “crash” cesarean under general anesthesia for fetal reasons could easily precipitate a fatal maternal cerebral hemorrhage. Her cardiovascular system needs to be stabilized by adequate hydration, and her central nervous system stabilized with MgSO4, prior to the surgical intervention. She is probably not the optimal candidate for transport, but if you were in a Level I instead of a Level II facility, that would of course be necessary. A MgSO4 drip would of course be critical during such a transport.