



Hypertensive Disorders of Pregnancy for Midwives

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Course Description:

Hypertensive disorders in pregnancy remain one of the leading causes of maternal death. The Maternal 911 Hypertension in Pregnancy module will give you a basis of knowledge to better recognize and treat preeclampsia. This knowledge base will help with communication to the patient and her family. The goal would be to increase the maternal safety where she will undergo care and delivery.

Approximate Time to Complete: 120 minutes



Click here to download a print version of this course.



The purpose of this module is for the participant to:

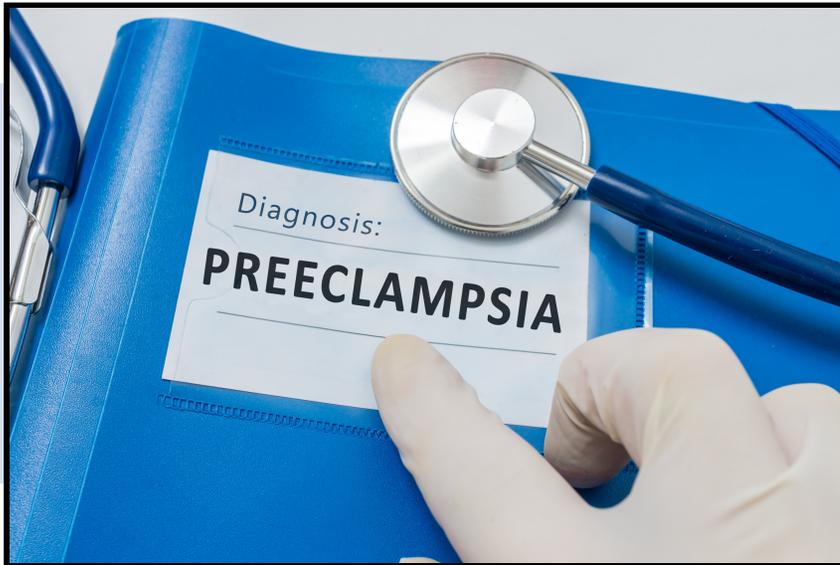
- Explain criteria for preeclampsia, severe preeclampsia, and eclampsia.
- Identify risks associated with causing preeclampsia.
- Recognize the signs and symptoms of worsening preeclampsia so prompt health care delivery can be implemented.
- Describe the pathogenesis of preeclampsia.
- Identify clinical features and pathophysiology by organ system.
- Describe the medications used for resuscitation and how they may affect the woman and expected outcomes.





- Table of Contents
- How to use the module
- Preeclampsia
- Diagnosis of Preeclampsia
- Diagnosis of Gestational Hypertension
- Diagnosis of HELLP Syndrome
- Diagnosis of Preeclampsia with Severe Features
- Eclampsia
- Occurrence Rates
- Spectrum of Preeclampsia
- Respectful Care Element
- Pathophysiology
- Clinical Features and Pathophysiology by Organ System
- Risk Factors
- Planning and Prevention
- Management and Treatment
- Recommendations of National and International Societies
- Complications
- Course Conclusion





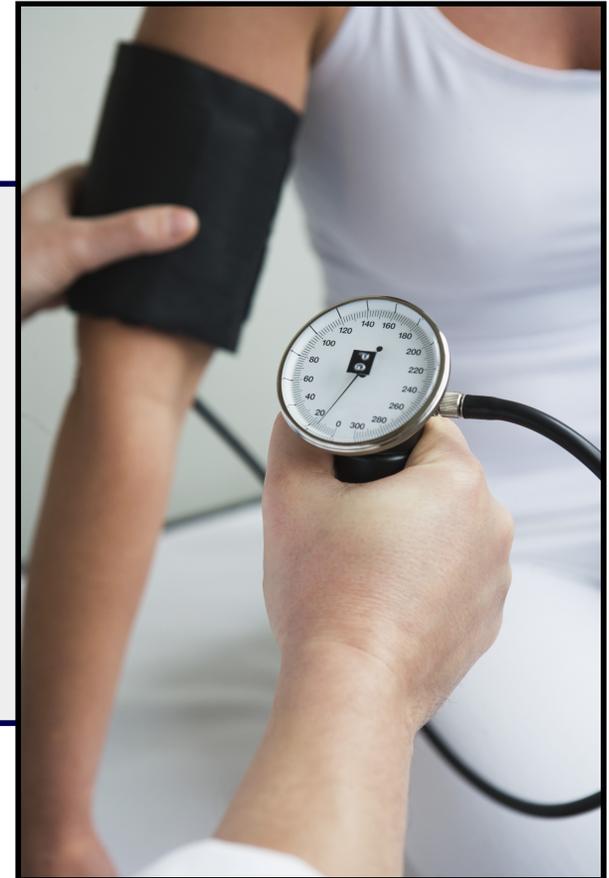
Preeclampsia is a multi-system progressive disorder characterized by new onset hypertension and proteinuria, or hypertension and end-organ dysfunction with or without proteinuria, in the last half of pregnancy or postpartum, often in a previously normotensive woman.



Diagnosis of Preeclampsia

Preeclampsia is defined as systolic blood pressure (BP) > 140 mmHg or diastolic BP > 90 mmHg on two occasions, at least four hours apart, after 20 weeks of gestation in previously normotensive woman with proteinuria >0.3 grams in a 24-hour urine specimen or protein(mg/dL)/creatinine (mg/dL) ratio > 0.3.

The diagnosis also needs to be considered in the postpartum hypertensive patient.



Diagnosis of Gestational Hypertension

Gestational Hypertension is defined as a systolic blood pressure of 140 mmHg or more or a diastolic blood pressure of 90 mmHg or more, or both, on two occasions at least 4 hours apart after 20 weeks gestation in a woman with previously normal blood pressure.





Diagnosis of HELLP Syndrome

The HELLP is an abbreviation for Hemolysis, Elevated Liver enzymes and Low Platelet count syndrome.

Hemolysis

- LDH >600 IU/L

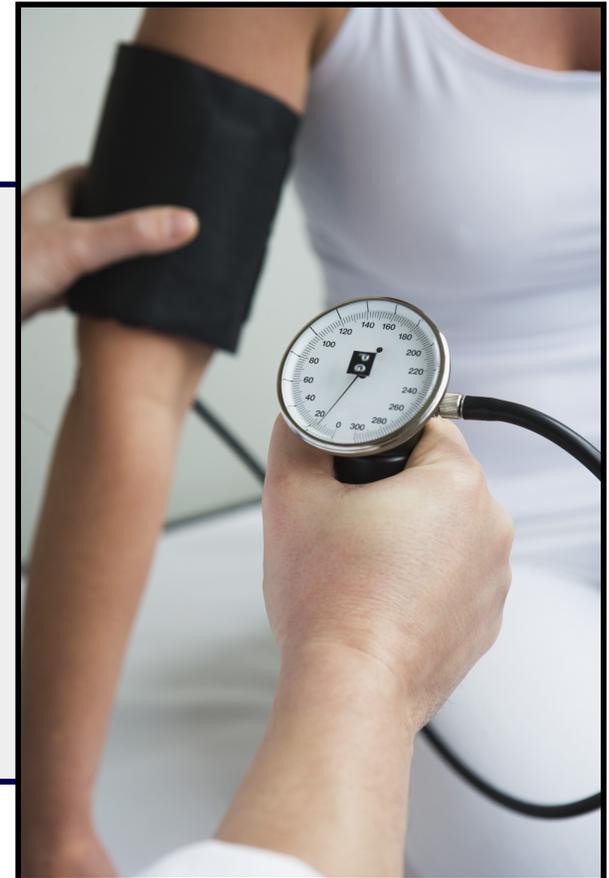
Elevated Liver Enzymes

- AST, ALT > 2x the upper limit of normal

Low Platelets

- Platelets <100,000

Does not require the presence of hypertension or proteinuria
30% of cases present or worsen postpartum



Diagnosis of Preeclampsia with Severe Features

Hypertension

- New onset systolic blood pressure of 160 mmHg or more, or diastolic blood pressure of 110 mmHg or more
- Treatment of severe range BP should not be withheld for the purposes of diagnosis

Pre-eclampsia with severe features should be diagnosed in any patient with gestational hypertension in conjunction with any of the following:

- Thrombocytopenia
 - Platelet count <100,000
- Impaired liver function with no alternative etiology
 - 2x the upper limit of normal or persistent right upper quadrant or epigastric pain unresponsive to medication



[Click here for more on the diagnosis of preeclampsia with severe features](#)





Diagnosis of Preeclampsia with Severe Features

Renal insufficiency

- Creatinine >1.1 mm/dL or doubling of a baseline creatinine in the absence of other renal disease

Pulmonary edema

- Maternal symptoms
 - New onset headache unresponsive to medication and not accounted for by alternative diagnoses
 - New onset visual disturbances

Preeclampsia with severe features does not require proteinuria





Eclampsia is diagnosed when grand mal seizures occur in a woman with no history of neurological conditions and evidence of hypertension or other evidence of preeclampsia.

Seizures can occur before, during, or after delivery of the fetus.



Preeclampsia

- Complicates 2-8% of pregnancies worldwide [1].
- The prevalence of preeclampsia in the United States (U.S.) is about 3.4% but 1.5- to 2-fold higher in first pregnancies [2].



Slide 1 of 3



Click the above arrows to see more information.



Eclampsia

- Occurs in 2 to 3% of women with severe features not receiving anti-seizure prophylaxis and up to 0.6% of women with preeclampsia without severe features [3].
- The incidence of eclampsia has been stable at 1.6-10 cases per 10,000 deliveries in developed countries [4-9].



 *Slide 2 of 3* 



Click the above arrows to see more information.





PREECLAMPSIA NEED TO KNOW

Any Pregnancy, Any Person

Preeclampsia is a serious disease related to high blood pressure. It can happen to any pregnant person during the second half of pregnancy or up to six weeks after delivery. In extreme cases, mothers and babies have died from preeclampsia.

If it's discovered early, most mothers with preeclampsia can still have a healthy baby.

SIGNS AND SYMPTOMS TO REPORT TO YOUR DOCTOR OR MIDWIFE IMMEDIATELY

Migraine-like Headaches

Chronic headaches that will not go away after taking medications are a concern.

Upper belly pain or stomach pain

Right sided abdominal pain that mimics indigestion or the flu. Nausea and vomiting may occur.

Feeling generally unwell

Blurry vision or seeing flashing lights

Seeing flashing lights, bright spots or auras. Partial or total loss of vision may occur.

Baby moving less

You know your baby's movement patterns. When the baby moves less this could be concerning.

Hands or face swelling

Preeclampsia can cause water retention leading to the hands and face to swell.

Gaining more than 5 pounds (2.3kg) in a week.

FACTS ABOUT PREECLAMPSIA

Affects 5-8 percent of all pregnancies. That is 1 in every 12 pregnancies



Preeclampsia doubles a woman's risk for developing heart disease or having a stroke over their next 5-15 years.



maternal911.com



African-American women are four times as likely to die as a result of preeclampsia. Two out of three women who experience preeclampsia will die from heart disease.



Over an eight year period of time, African American women had 56% more preeclampsia than Caucasian women.

Spectrum of Preeclampsia



Click the image for a full sized infographic



PREECLAMPSIA NEED TO KNOW

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Respectful Care Element



Open, transparent, and empathetic communication with pregnant and postpartum people and their support network.

- Establish and maintain a mechanism for to identify inequitable care episodes of miscommunication or disrespect.
- Develop a plan to address reported cases of inequitable care, miscommunication or disrespect.

Include the patient as part of the multidisciplinary team to establish trust, and bidirectional shared decision making with patient values and goals as the primary driver of this process.

As Black, Indigenous, and Hispanic people experience maternal mortality and severe maternal morbidity at disproportionately higher rates because of systemic racism, but not race itself, it is necessary to mitigate bias by having a high index of suspicion for a contributing clinical condition, such as severe hypertension, in these populations.

Patient support networks may include nonfamilial supports, such as doulas and home visitors, who, with the postpartum person's permission, should be welcomed when any teaching or planning is provided.





Hypertensive complications of pregnancy is 1 of the 3 leading causes of maternal death in the United States [12-14].

Approximately one maternal death per 100,000 live births is due to preeclampsia or eclampsia with a case fatality rate of 6.4 deaths in 10,000 cases [15-17].

It is imperative to educate women during their pregnancy regarding the signs and symptoms of preeclampsia and empower her to reach out for help if these occur.



Slide 3 of 3



Click the above arrows to see more information.



Pathophysiology of Preeclampsia



Maternal and fetal/placental factors are involved in the pathophysiology of preeclampsia with both affecting the severity of the disease.

Studies on placental pathology have demonstrated that preeclampsia is associated with abnormal uterine spiral artery remodeling and trophoblastic invasion, but the cause of this remains unknown [23,24].

It has been found that the spiral artery remodeling and trophoblastic invasion have defects leading to characteristic hypertensive disorders of pregnancy and fetal growth restriction [18,19].

Pathophysiology of Preeclampsia

Critical components in the pathogenesis of preeclampsia include:

- Hypoperfusion
- Hypoxia
- Ischemia

The release of factors into the maternal blood stream alters the maternal endothelial cell function, leading to characteristic systemic signs and symptoms of preeclampsia [28-32].

Hypoperfusion becomes more pronounced as pregnancy progresses, since the abnormal uterine vasculature is unable to accommodate the normal rise in blood flow to the fetus/placenta with increasing gestational age [20-22].



It is unknown why the normal development of uteroplacental circulation, does not occur in some pregnancies.

The following are suspected to play a role:

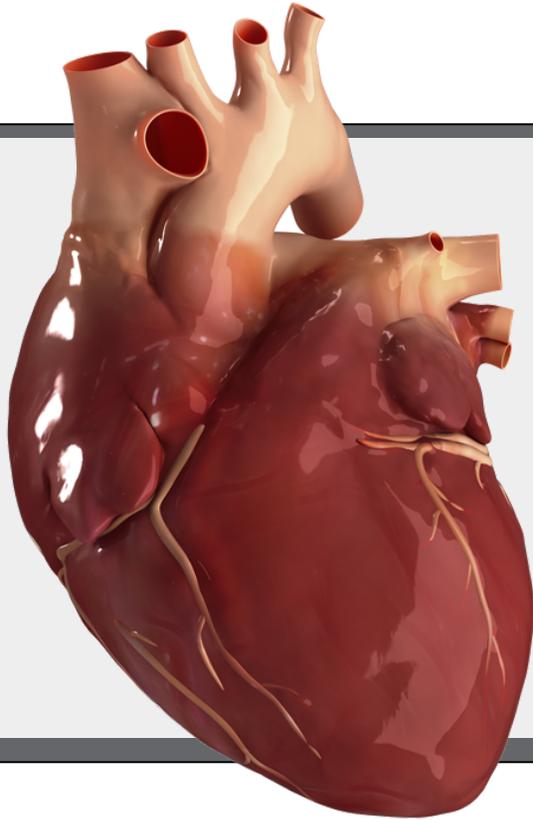
- Vascular
- Environmental
- Immunological
- Genetic factors [35]

The aphorism "Delivery is the cure" for preeclampsia is widely believed in medicine, but it is clear in many cases the multisystem pathology of hypertension in pregnancy continues for a variable amount of time after delivery and after hospital discharge. For some women, delivery is not the cure, so it is critical to keep evaluating, asking and listening. to women about their health through the postpartum period as serious clinical consequences may persist for days and weeks after delivery [35, 102].

[Click for a video on preeclampsia](#)

www.youtube.com/watch?v=70tpqg58Oug





CARDIAC PHYSIOLOGY

Hypertension is the earliest clinical finding of preeclampsia and is the most common clinical indicator of disease.

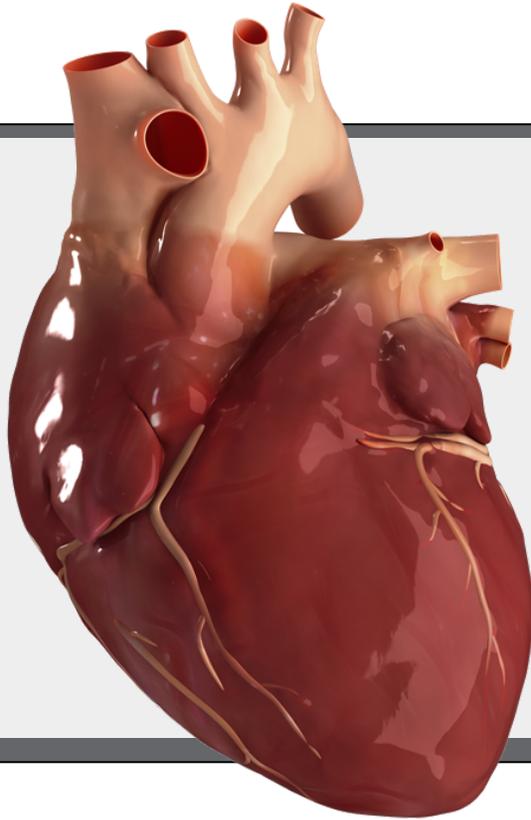
Hypertension can develop at any point after 20 weeks gestation or in the postpartum period.

Historically, obstetric (OB) practice has placed less emphasis on acute control of blood pressure to prevent stroke, yet this has recently been identified as a major gap in knowledge and application of proven therapeutic interventions [102].

In addition to preventing cerebrovascular accidents, control of severe hypertension is associated with reduced severe maternal mortality [102].

Slide 1 of 5





CARDIAC PHYSIOLOGY

The BP usually rises gradually to $\geq 140/90$ mmHg.

- Often in the third trimester and after the 37th week of gestation [28].

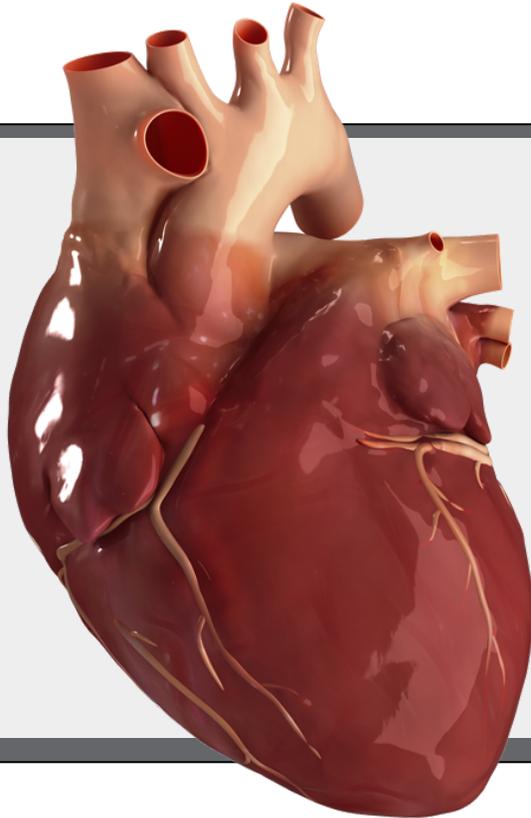
A woman with a systolic blood pressure greater than or equal to 140 mmHg or a diastolic blood pressure greater than or equal to 90 mmHg on 2 occasions at least 4 hours apart after 20 weeks gestation is concerning for pre-eclampsia.

A woman with a systolic blood pressure greater than or equal to 160 mmHg or a diastolic blood pressure greater than or equal to 110 mmHg is concerning for pre-eclampsia with severe features.

The diagnosis is confirmed if the severe range BP is noted on two occasions at least 4 hours apart; however, antihypertensive therapy should not be withheld for the purposes of confirming the diagnosis.



CARDIAC PHYSIOLOGY

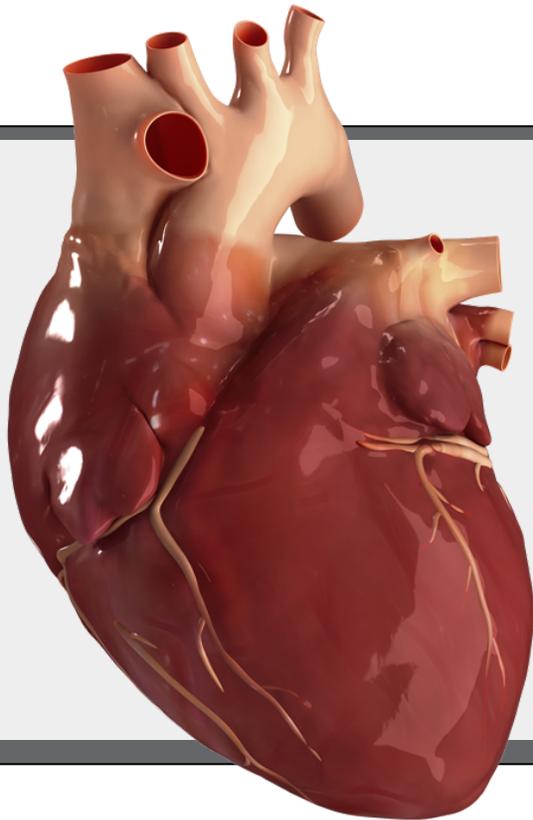


Vascular Changes

- Typical vascular changes include:
 1. Vasospasm
 2. Leaky capillaries
- Vasospasm is caused by inappropriate ratios of the vasoconstrictors - thromboxane A2 and endothelins - and vasodilators - prostacyclin and nitric oxide
- Leaky capillaries are caused by innate differences of the vasculature, in addition to decreased colloid oncotic pressure
- The vascular changes are responsible for the common finding of edema, as well as the cause of the increased risk of pulmonary edema



CARDIAC PHYSIOLOGY



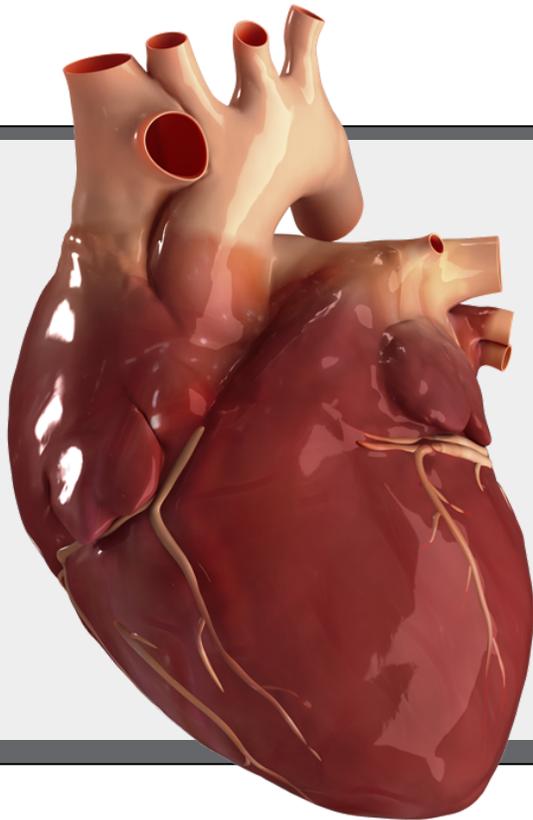
Edema

- Edema is one of the most common findings in preeclampsia
- The finding is likely related to leaky capillaries and decreased colloid oncotic pressure.
- Edema is not part of the diagnostic criteria for preeclampsia due to the frequency of this finding in most pregnant women; however, if rapid onset edema or weight gain is noted, it can be a harbinger for the future development of preeclampsia.

[Click to see a picture of edema in the lower extremities](#)







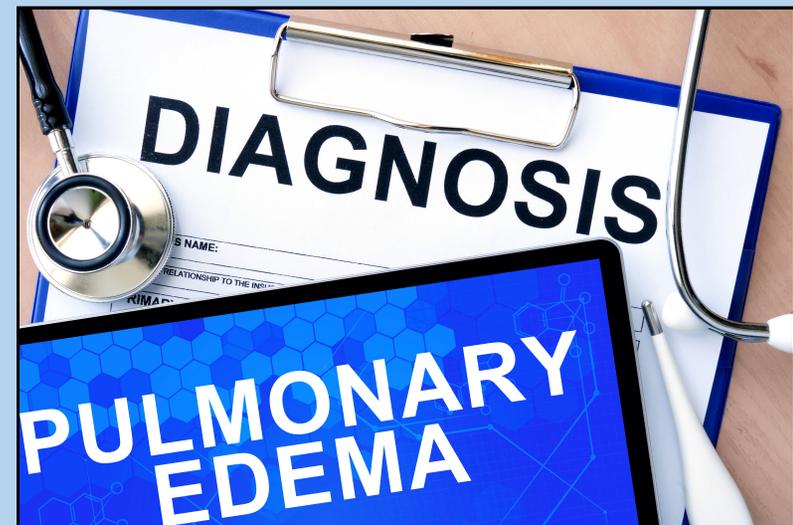
CARDIAC PHYSIOLOGY

- Changes can be associated with a highly variable hemodynamic profile [36-41].
 - A small subgroup of women develops myocardial damage or diastolic dysfunction [41].

◀ Slide 5 of 5

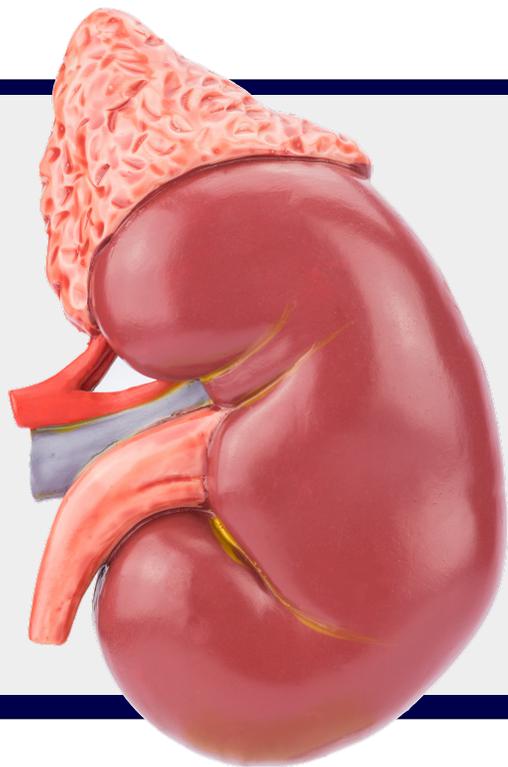
Pulmonary Physiology

- Pulmonary edema is diagnosed by chest x-ray showing typical features of pulmonary edema.
- Caused by capillary leak and decreased colloid oncotic pressure.
- If present, indicative of preeclampsia with severe features.
- More common in the postpartum period [54, 57].





RENAL FUNCTION



Glomerular filtration rate (GFR) decreases by 30 to 40% in preeclampsia.

Plasma creatinine is typically normal or slightly elevated (0.8-1.2 mg/dL).

Creatinine > 1.1mg/dL or doubling of a baseline value indicates severe disease.

- Caused by renal vasoconstriction and sodium retention due to reduced plasma volume and systemic vasoconstriction.

Urine output may decrease to <500mL/24 hours.



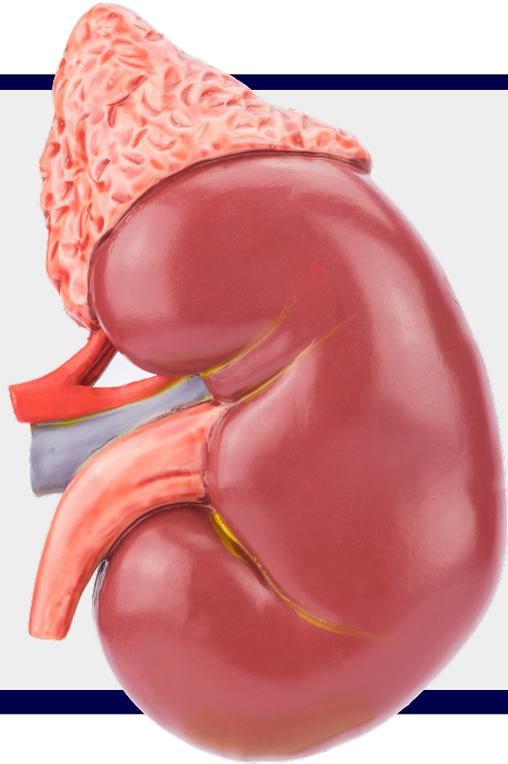
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Slide 1 of 5





RENAL FUNCTION



The role of serum uric acid levels remains controversial as a predictor of complications associated with preeclampsia [43,44].

An international prospective study of women with preeclampsia demonstrated that serum uric acid, based upon gestational age:

- Is not clinically useful in predicting adverse maternal outcomes.
- Is useful in predicting perinatal outcomes [45].



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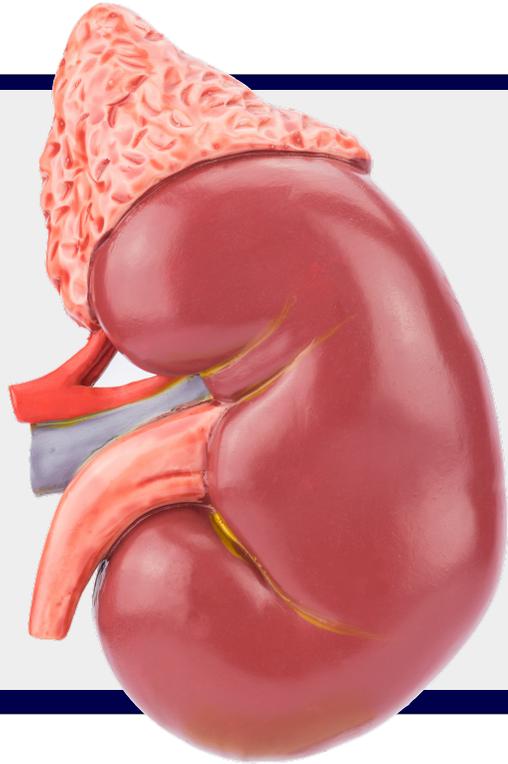


Slide 2 of 5





RENAL FUNCTION



Histology findings include:

- Endothelial cell swelling
- Loss of fenestrations
- Occlusion of capillary lumens [46]

Glomerular Endotheliosis

- Shares histologic features with non-preeclamptic thrombotic microangiopathies [46].
- Histopathologic finding due to preeclampsia



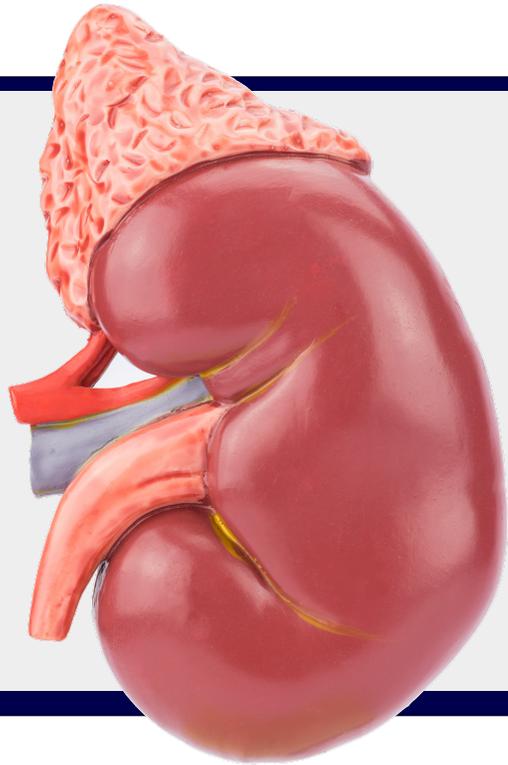
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Slide 3 of 5



RENAL FUNCTION



Clinical Features of the Renal System

The kidney is the organ most likely to manifest endothelial injury related to preeclampsia.

Renal - Proteinuria

The most common cause of proteinuria in pregnant women is preeclampsia.

Click for more on renal changes



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Slide 4 of 5

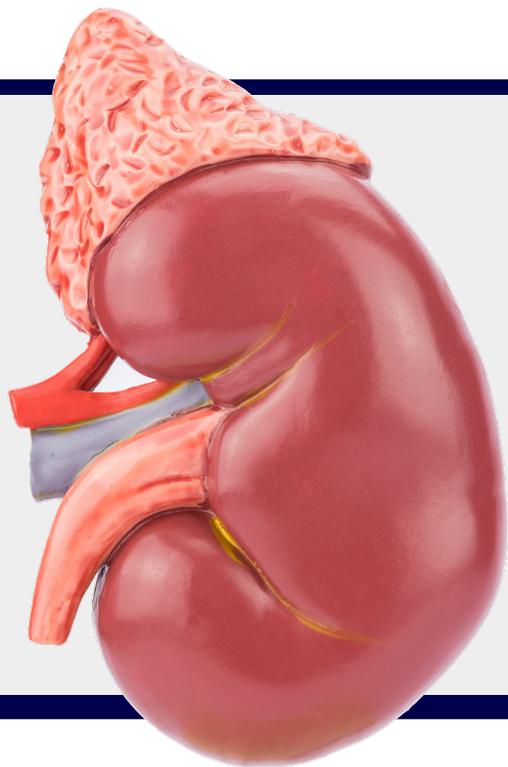


Typical renal changes include:

1. Proteinuria
 2. Elevated creatinine
- The histopathologic renal finding is glomerular endotheliosis.
 - Proteinuria is caused by increased tubular permeability.
 - Increased creatinine is caused by acute renal deterioration, which is likely related to vasospasm causing a decrease in the glomerular filtration rate.



RENAL FUNCTION



Proteinuria is defined as:

- >0.3 mg protein on urine protein:creatinine ratio
- >300 mg of protein in a 24-hour specimen.
- Persistent 1+ (30mg/dL) on dipstick.

Proteinuria

- Once diagnostic proteinuria is noted, there is typically no reason to continue to trend values.
- Proteinuria is caused by impaired integrity of the glomerular filtration barrier.
- Protein excretion increases with hypofiltration caused by altered tubular function [42].



Click through all 5 slides before moving on to the next page.



Slide 5 of 5





Hematologic Changes

- Typical hematologic changes include:
 1. Hemoconcentration
 2. Thrombocytopenia
 3. Possible hemolysis
- Hemoconcentration is caused by a lack of hypervolemia, which is attributed to the leaky capillaries.
- Thrombocytopenia is caused by increased platelet activation, aggregation and consumption.
- The hemoglobin level should take into account the possibility of hemoconcentration and hemolysis.
- Lactate dehydrogenase is highly concentrated in red blood cells; therefore, an elevated LDH (>600) is indicative of hemolysis.

Slide 1 of 2





When hemolysis and reduced plasma volume are both present, the hematocrit may be normal.

White blood cell (WBC) count may be slightly elevated due to neutrophilia.

Concentrations of the following are not affected unless abruptio placenta or severe liver dysfunction is also present:

- Prothrombin time (PT)
- Partial thromboplastin time (PTT)
- Fibrinogen [47]

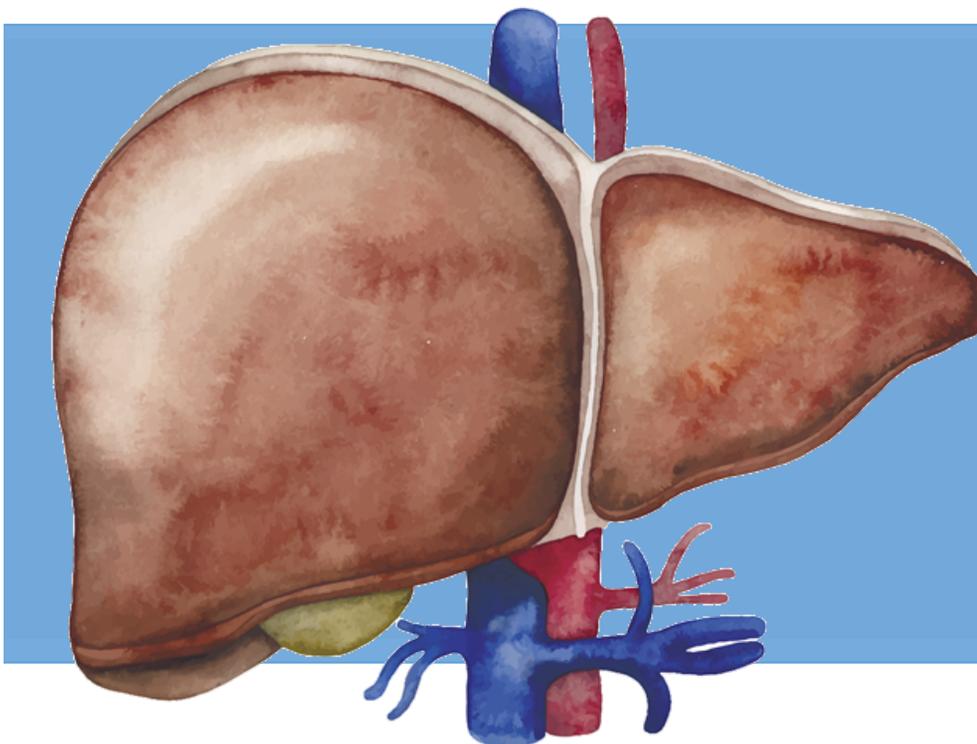


Slide 2 of 2





HEPATIC



Hepatic Changes

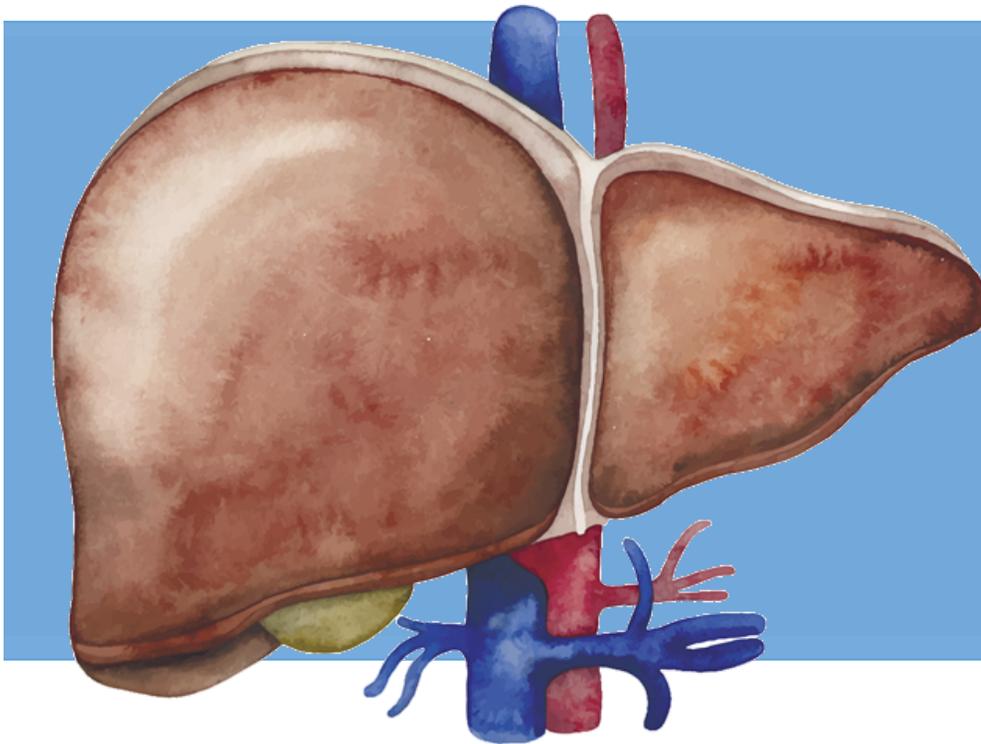
- Typical hepatic changes include:
 1. Elevated AST, ALT
 2. Elevated LDH
- AST is the dominant transaminase that is released into peripheral circulation secondary to periportal necrosis.
- Typically, early in the course of the disease, AST is elevated more significantly than ALT.
- LDH can be elevated from hemolysis, but also from hepatic dysfunction if there is liver ischemia or necrosis.
- In rare cases, elevated bilirubin can be seen if there is significant hemolysis in the late stage of disease.
- If hepatic function is severely compromised, there can be abnormalities in coagulation studies including PT, PTT and fibrinogen.

Slide 1 of 4





HEPATIC



Hepatic Function

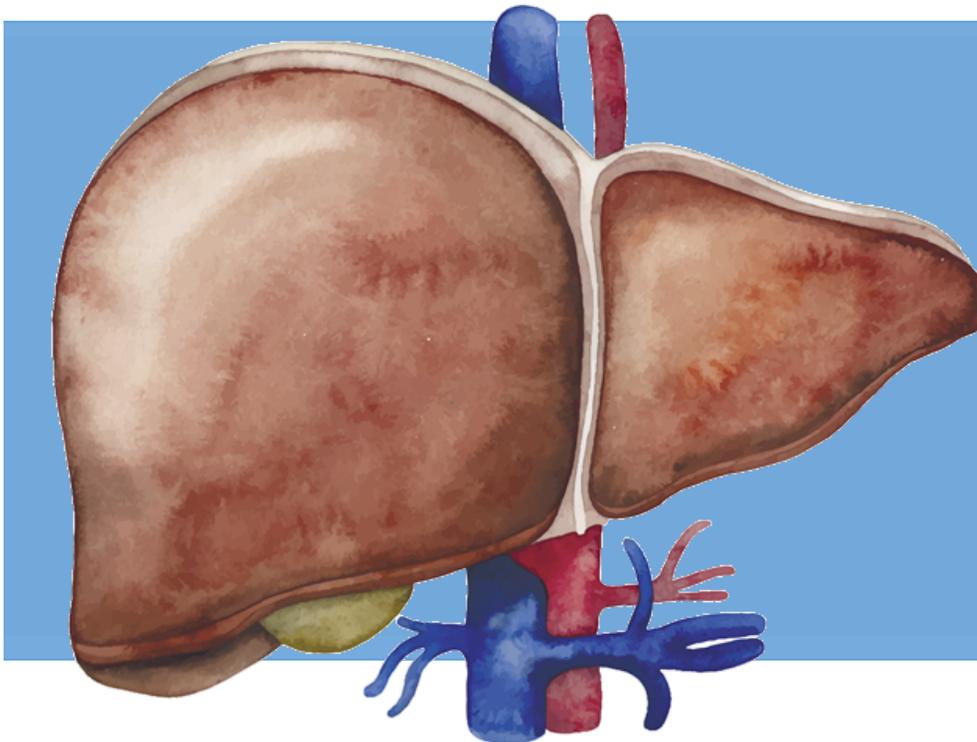
- Impaired liver function that is not accounted for by alternative etiology and is consistent with transaminases that are 2x the upper limit of normal is consistent with preeclampsia with severe features.
- The diagnosis can also be made with severe, persistent right upper quadrant pain or epigastric pain unresponsive to medications in the setting of hypertension.

◀ Slide 2 of 4 ▶





HEPATIC



Epigastric Pain

Thought to be related to stretching of the Glisson's capsule due to hepatic swelling or bleeding.

May be the only presenting symptom of preeclampsia

- Can be mistaken for gastroesophageal reflux.
- Liver irritation can also present as new onset nausea or vomiting
- GERD is common for pregnant women and occurs more often at night.

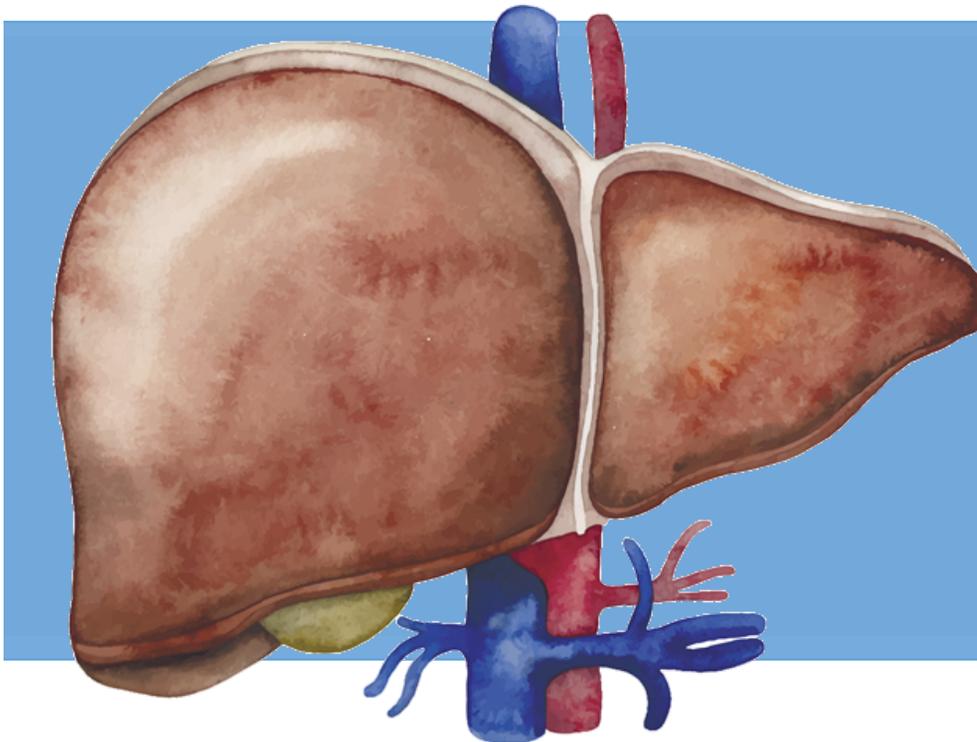
Palpation of the liver may cause her discomfort.

◀ Slide 3 of 4 ▶





HEPATIC



Epigastric Pain

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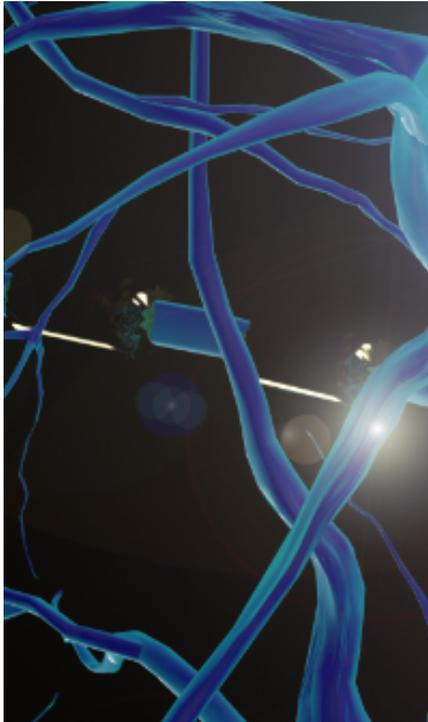
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◀ Slide 3 of 4 ▶

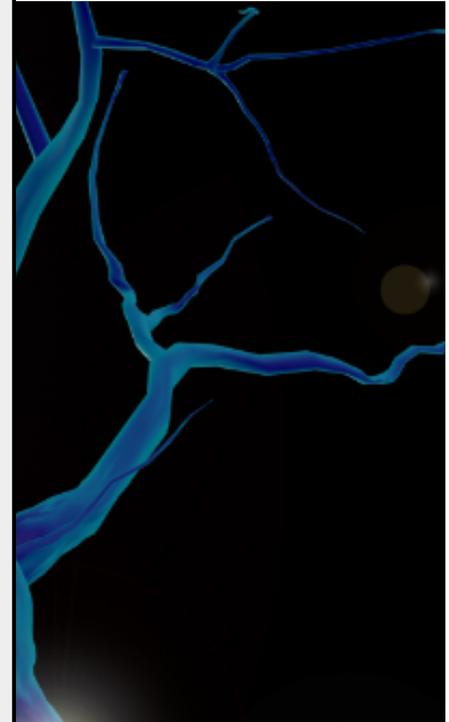




Central Nervous System (CNS)

Central nervous system manifestations of preeclampsia include:

- Headache
- Visual symptoms
- Generalized hyperreflexia
- Sustained ankle clonus





CNS

Headache may be:

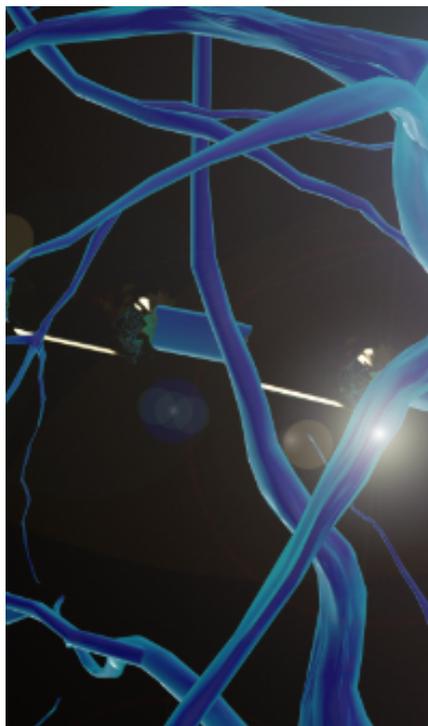
- Temporal
- Frontal, the most common site
- Occipital
- Diffuse [50,51]

Frontal is most common.

Pain described as:

- Throbbing
- Pounding
- Piercing

The headache is not relieved with over-the-counter analgesics and persists despite sleep, caffeine and eating.

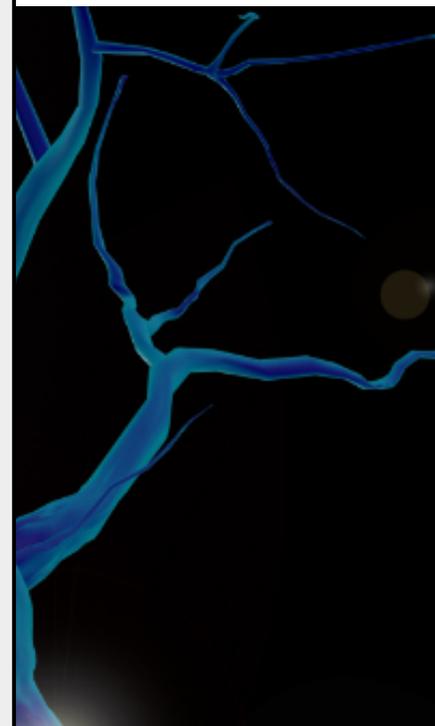


CNS

Visual symptoms are caused by retinal arteriolar spasm [52].

Symptoms include:

- Blurred vision
- Flashing lights or sparks (photopsia)
- Scotomata (dark area or gaps in the visual field [53-55])
- Diplopia (blindness in one eye)
- Cortical blindness is rare and typically transient [56]





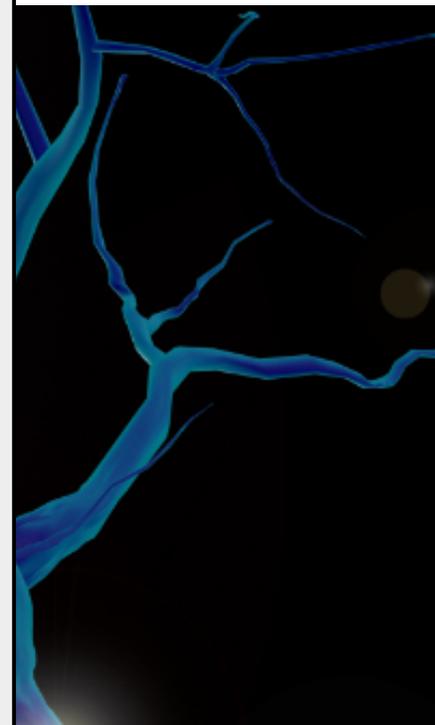
CNS Seizures

When a seizure occurs in a woman with preeclampsia, it signifies eclampsia.

- 1 in 400 women with preeclampsia without severe features develop eclamptic seizures [3].
- 1 in 50 with preeclampsia with severe features women will develop eclamptic seizures [5-11].

Risks associated with an eclamptic seizure include:

- Hemorrhage
- Petechiae
- Cerebral edema
- Vasculopathy
- Ischemic brain damage
- Microinfarcts [87]

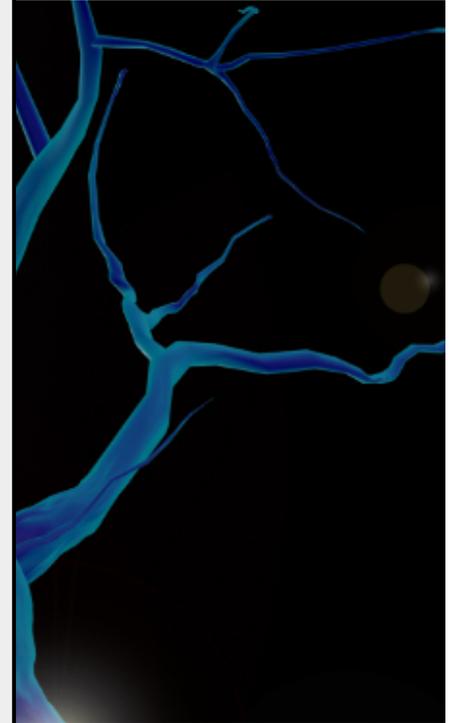




CNS

Computed Tomography (CT) or Magnetic Resonance Imaging (MRI) findings may include:

- Cerebral edema
- Cerebral ischemia
- Hemorrhagic changes [58,59]
- Posterior reversible leukoencephalopathy syndrome (PRES):
 - Brain abnormalities associated with preeclampsia are caused by generalized endothelial cell dysfunction and loss of cerebrovascular autoregulation [92].
- Reversible cerebral vasoconstriction syndrome



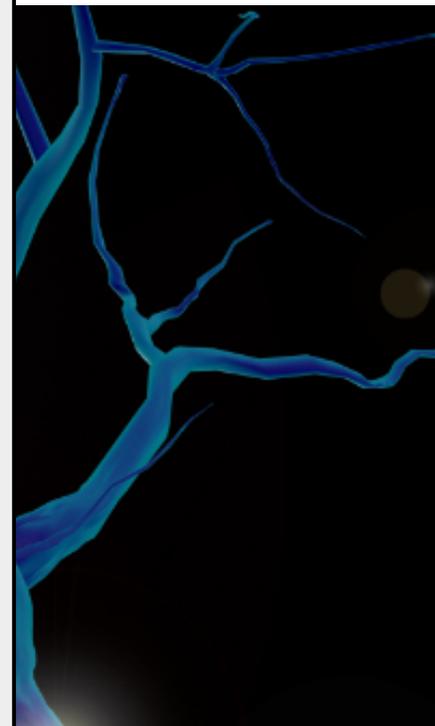


CNS – Stroke

Leading cause of death or disability related to severe preeclampsia or eclampsia [93].

Most strokes caused by preeclampsia or eclampsia are:

- Hemorrhagic
- Preceded by severe headache
- Sustained severe range blood pressure



CNS – Stroke

Eclamptic seizures do not occur in all cases.

Risk factors for hemorrhagic stroke in women with preeclampsia include:

- Systolic BP persistently > 160mmHg and/or
- Diastolic BP persistently > 110mmHg
- Severe headache
- Seizures

Prompt treatment of severe range blood pressures in the setting of preeclampsia is indicated in order to prevent stroke

Pathophysiology of Preeclampsia

Fetus

Fetal Growth Restriction:

- Severe and early onset of preeclampsia affects birth weight [60].
- Although fetal growth restriction is not part of the diagnosis of preeclampsia, the finding of fetal growth restriction is a risk factor for the subsequent development of preeclampsia

Oligohydramnios

- Can develop in the setting of preeclampsia due to placental insufficiency

Early Onset Severe Preeclampsia Increases the Risk of:

- Fetal death
- Perinatal death
- Severe neonatal morbidity [61,62]



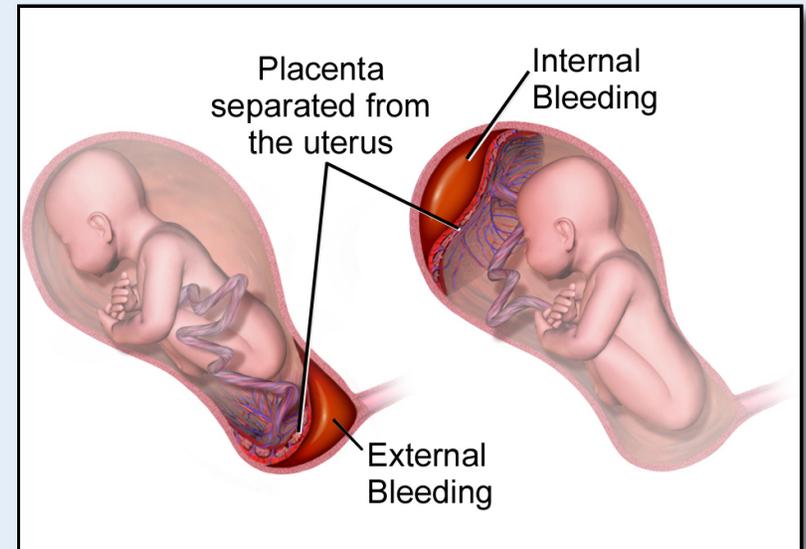
Placental Abruption

Occurs in < 1% of women with preeclampsia without severe features.

Occurs in 3% of women who have severe features [64].

If placental abruption occurs in the setting of preeclampsia, the patient has a high risk of fetal demise, in addition to a high risk for coagulopathy, including disseminated coagulopathy.

This [summary table](#) reviews features of preeclampsia with adverse and severe features.



System	Severe Feature
Central Nervous System (CNS)	Headache Visual Changes
Cardiac	SBP>160 or DBP>110
Hematologic	Platelets <100,000
Hepatic	AST, ALT > 2x upper limit of normal
Renal	Creatinine >1.1 or 2x baseline



If pre-eclampsia with severe features develops prior to 37 weeks, preterm delivery is indicated.

- This may be indicated within hours to days of diagnosis

Preeclampsia does not accelerate fetal maturation.

In preeclamptic women with age-matched normotensive controls, frequency of the following neonatal morbidities is not increased:

- Neonatal respiratory distress
- Intraventricular hemorrhage
- Necrotizing enterocolitis (NEC) [63]



A critical factor in reducing maternal mortality is early recognition and treatment of worsening signs and symptoms of preeclampsia. Health care providers often fail to recognize and respond to clinical signs and symptoms in a timely manner.

In fact, missed vital sign 'triggers' occurred in 60% of preeclampsia deaths [99].

This module will help with the development of this critical thinking, but each center must develop a process for recognition and response to patient's deteriorating conditions with written criteria describing early warning signs and indicating when to seek further assistance [100, 101].





Readiness

Drills to simulate patients that emphasize:

- all elements of the facility severe hypertension emergency management plan
- patient centered, empathetic, trauma-informed care

1 of 2 **➤➤**



Click the arrows to display more information about readiness

Readiness





Readiness

Referral resources and communication to ensure that:

- maternal and neonatal transfer protocol is in place
- implementation of resource mapping to identify local resources and support services so that this information is available.
- consider providing a blood pressure cuff; do provide educational materials and information on who to call for concerns for the patient to take home

◀ 2 of 2





It is imperative to the health of the mother and fetus to monitor for signs and symptoms of worsening or severe preeclampsia:

- Headache
- Increasing BP
- Altered consciousness: restless, agitation, hallucinations, lethargy, confusion
- Visual disturbances: floaters, blurred vision, spots, blind spots
- Upper abdominal pain
- Urine output < 30mL/hour
- Shortness of breath
- Complaints of chest pain
- Pulse oximetry < 95%
- Cough
- Tachypnea > 26 breaths/min
- Tachycardia > 100 bpm
- Adventitious breath sounds
- Eclamptic seizure [101]

This link provides a tool for preeclampsia early recognition (PERT) to help:
<https://www.health.ny.gov/publications/2036.pdf>

Consider printing the PERT tool and carry with you to deliveries.

Signs and Symptoms



New York State Department of Health Preeclampsia Early Recognition Tool (PERT)

ASSESS	NORMAL (GREEN)	WORRISOME (YELLOW)	SEVERE (RED)
Awareness	Alert/oriented	<ul style="list-style-type: none"> Agitated/confused Drowsy Difficulty speaking 	<ul style="list-style-type: none"> Unresponsive
Headache	None	<ul style="list-style-type: none"> Mild headache Nausea, vomiting 	<ul style="list-style-type: none"> Unrelieved headache
Vision	None	<ul style="list-style-type: none"> Blurred or impaired 	<ul style="list-style-type: none"> Temporary blindness or blind spots
Systolic BP (mm Hg)	100-139	140-159	≥160
Diastolic BP (mm Hg)	50-89	90-109	≥110
HR	61-110	111-129	≥130
Respiration	11-24	25-30	<10 or >30
SOB	Absent	Present	Present
O2 Sat (%)	≥95	91-94	≤90
Pain: Abdomen or Chest	None	<ul style="list-style-type: none"> Nausea, vomiting Chest pain Abdominal pain 	<ul style="list-style-type: none"> Nausea, vomiting Chest pain Abdominal pain
Fetal Signs	<ul style="list-style-type: none"> Category I Reactive NST 	<ul style="list-style-type: none"> Category II IUGR Non-reactive NST 	<ul style="list-style-type: none"> Category III
Urine Output (ml/hr)	≥50	30-49	≤30 (in 2 hrs)
Proteinuria (Level of proteinuria is not an accurate predictor of pregnancy outcome)	Trace	<ul style="list-style-type: none"> ≥ 1+** ≥300mg/24 hours 	<ul style="list-style-type: none"> 3+ or greater on 2 samples 4 hours apart*** ≥ 5 Gms/24 hours***
Platelets	>100	50-100	<50
AST/ALT	normal	1-<2x normal	2x normal or greater
Creatinine	≤0.8	0.9-1.1	≥1.2
Magnesium Sulfate Toxicity	<ul style="list-style-type: none"> DTR +1 Respiration 16-20 	<ul style="list-style-type: none"> Depression of patellar reflexes 	<ul style="list-style-type: none"> Respiration <12

GREEN = NORMAL

Proceed with Protocol

YELLOW = WORRISOME

Increase assessment frequency	
# Triggers	TO DO
1	<ul style="list-style-type: none"> Notify provider & charge nurse
≥2	<ul style="list-style-type: none"> Notify provider & charge nurse In-person evaluation Order labs/tests Anesthesia consult Consider magnesium sulfate Supplemental oxygen

** Physician should be made aware of worsening or new-onset proteinuria.

RED = SEVERE

Trigger: 1 of any type listed below	TO DO
1 of any type	<ul style="list-style-type: none"> Notify provider & charge nurse Immediate evaluation Transfer to higher acuity level 1:1 staff ratio
Awareness	<ul style="list-style-type: none"> Consider Neurology consult
Headache	<ul style="list-style-type: none"> CT Scan
Visual	<ul style="list-style-type: none"> R/O SAH/Intracranial hemorrhage
BP	<ul style="list-style-type: none"> Labetalol/hydralazine in 15 minutes Magesium sulfate loading or maintenance infusion
Chest pain	<ul style="list-style-type: none"> EKG, Consider CT angiogram
Respiration	<ul style="list-style-type: none"> O2 at 10L per rebreather mask
SOB	<ul style="list-style-type: none"> R/O pulmonary edema
O2 SAT	<ul style="list-style-type: none"> Chest x-ray



Risk Factors for Preeclampsia

Nulliparity

Exposure to paternal antigens is associated with the pathogenesis of preeclampsia. The primigravid woman, in theory, may be at risk due to limited exposure [65].



Risk Factors for Preeclampsia are Related to:

- Ethnicity: Nordic, Black, South Asian, and Pacific Island
- Low socioeconomic class
- Advanced maternal age
- Obesity
- Assisted reproductive technology
- Obstructive sleep apnea



Risk Factors for Preeclampsia

Preeclampsia in a previous pregnancy

- The severity of preeclampsia strongly impacts this risk.

If a woman developed preeclampsia with severe features in the second trimester of a prior pregnancy, the risk of recurrence is 25-65% [66-69].

If a woman has a history of pre-eclampsia without severe features, the risk of recurrence is 5-7% [70,71].

Women who had a normotensive first pregnancy develop preeclampsia in less than 1% of second pregnancies [65].



Risk Factors for Preeclampsia

Preexisting Medical Conditions

- Pregestational diabetes
- Chronic hypertension
- Renal disease
- Antiphospholipid antibody syndrome (APS)
- Systemic lupus erythematosus

Risk Factors Cont'd

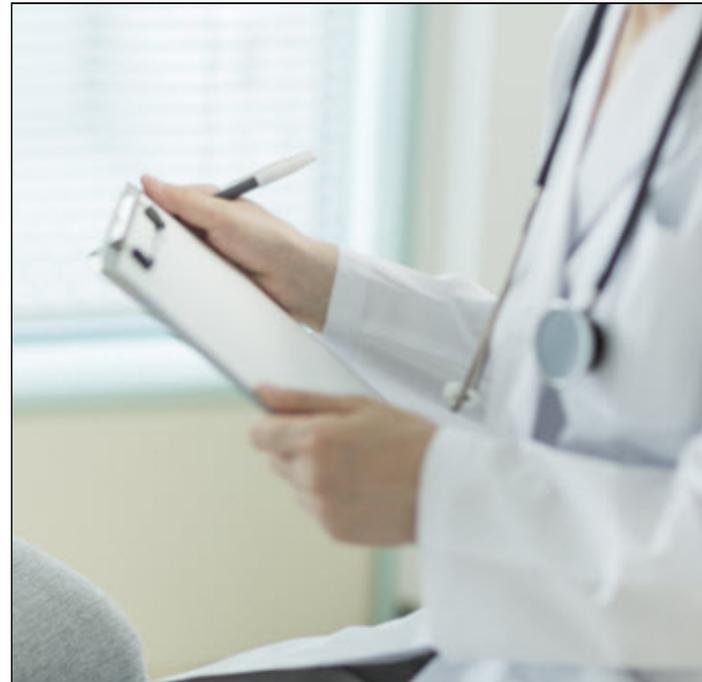




Risk Factors for Preeclampsia

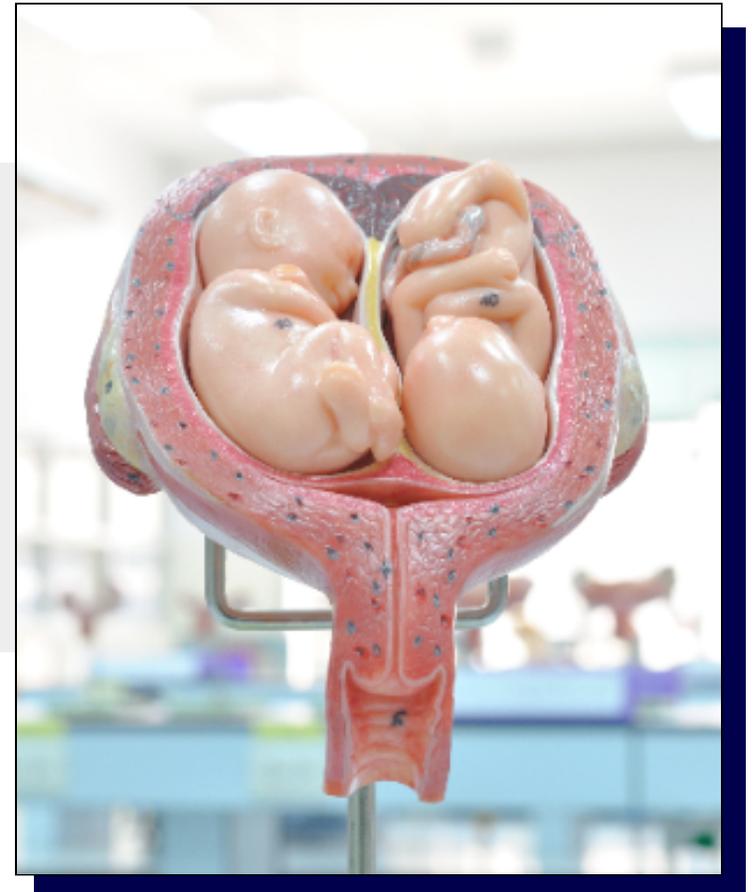
APS:

- 11-17% of women with pre-eclampsia will test positive for APS antibodies.
- The risk is highest in women who develop preeclampsia prior to 34 weeks.



Risk Factors for Preeclampsia

- Preeclampsia occurs at higher rates with multi-order gestations [77]:
 - Twins
 - Triplets
 - Quadruplets
 - The risk increases with each additional fetus





Diagnosis of Preeclampsia

- Any pregnant patient who is noted to have an elevated blood pressure after 20 weeks gestation should be screened for possible preeclampsia. The screening needs to include women that are postpartum.
- History
 - Ask about headaches, visual changes, right upper quadrant pain, epigastric pain, nausea and vomiting.
- Physical Exam
 - Evaluate for altered mental status, pulmonary edema, abdominal tenderness, edema, reflexes and clonus.





Laboratory evaluation

- CBC
- CMP
- Urine protein:creatinine ratio

Consider

- Non-stress test or biophysical profile
- Obstetric growth US

These studies would be appropriate to order in the outpatient setting when elevated BP is recognized. However, if the BP is in the severe range, hospital evaluation would be appropriate.





Management of Preeclampsia

Goals in health care delivery are to prevent:

- Organ damage
- Seizures
- Cerebral vascular accidents (CVA)
- Maternal and/or fetal death



Order recommended labs:

- protein/creatinine
- CBC with platelets
- LDH
- AST
- ALT

Screen the patient for her:

- medical needs
- mental and behavioral needs
- structural and social drivers of health

Patient education to include:

- who to contact
- review of warning signs
- reinforce value of outpatient follow up
- all should align with health literacy, cultural needs and language preference.

Recognition





Management of Preeclampsia

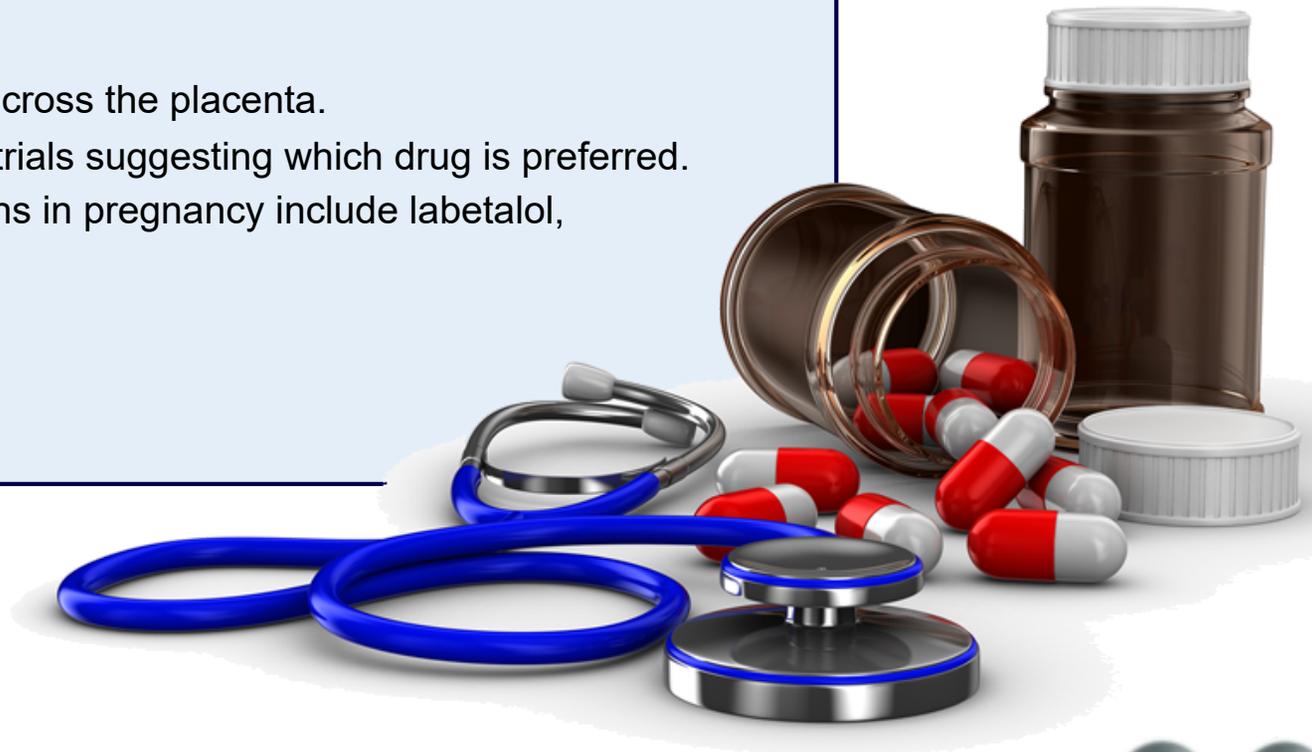
Treatment of severe hypertension is necessary when the systolic BP ≥ 160 mmHg and/or diastolic BP ≥ 110 mmHg, to reduce the risk of maternal stroke.

The goal is lower the BP, not to normal, but to a less severe range between 140-160/90-100mmHg.

If a patient meets criteria for gestational hypertension or preeclampsia without severe features, antihypertensive therapy is **not recommended** given that it can mask the diagnosis of severe blood pressures

Antihypertensive Therapy

- All antihypertensive drugs cross the placenta.
- There are no randomized trials suggesting which drug is preferred.
- Typical first-line medications in pregnancy include labetalol, hydralazine or nifedipine.





Treatment Options

- The choice of drug depends on the severity of hypertension, location of the patient (home, birthing center or hospital) and the route of administration:
 - Parenteral
 - Oral

For those delivering in the hospital or birthing center location, let's review the medications.

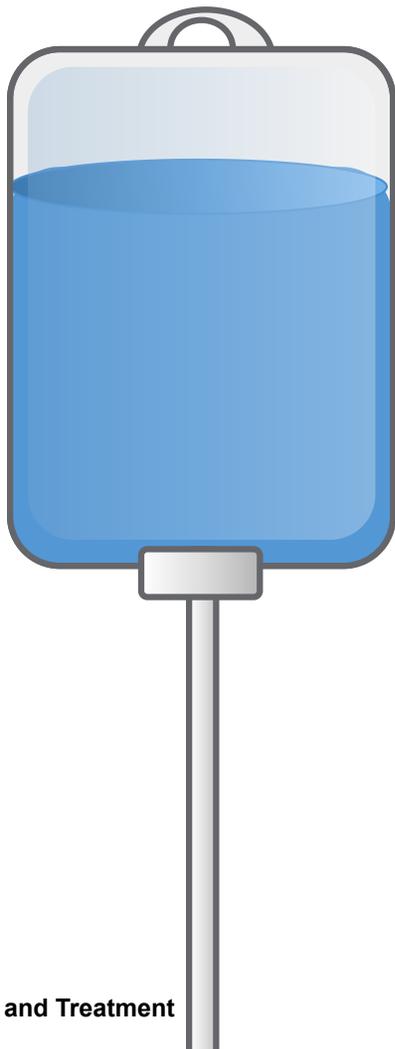
Acute Therapy

First-line agents for treatment of severe hypertension:

- Labetalol
- Hydralazine
- Nifedipine, immediate release, is acceptable [79].

Treatment with first line agents should be expeditious and occur as soon as possible within 30-60 min of confirmed severe hypertension to reduce the risk of maternal stroke [98].





Labetalol

- First-line therapy because it is:
 - Effective
 - Rapid onset of action
 - Good safety profile

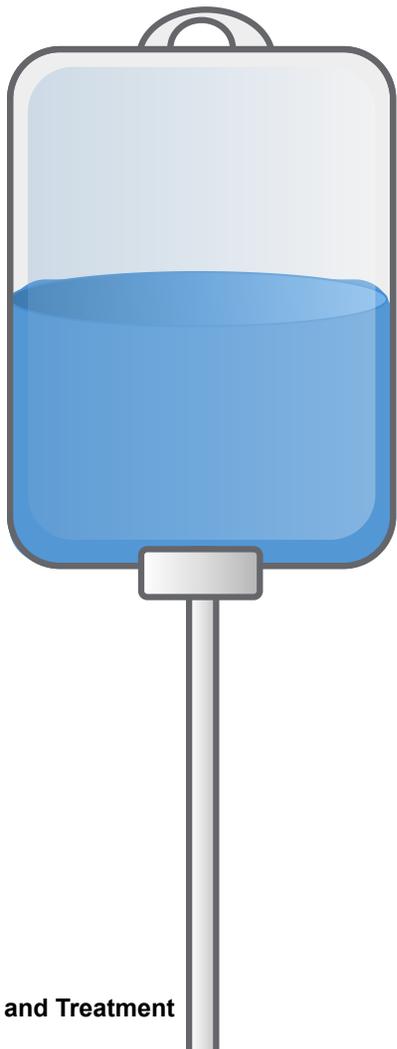
Begin with 20mg intravenously (IV) over 2 minutes followed at 10-minute intervals with incrementally larger doses up to 80 mg, up to a maximum total cumulative dose of 300 mg.

Labetalol should not be used in women with asthma, heart disease, or congestive heart failure (CHF) [98].



Click the I.V. bag to see types of treatment.





Hydralazine

Begin with 5mg IV over 1 to 2 minutes:

- If BP goal is not achieved within 20 minutes, give a repeat dose of 5 or 10 mg. The maximum cumulative dose is 20 mg.

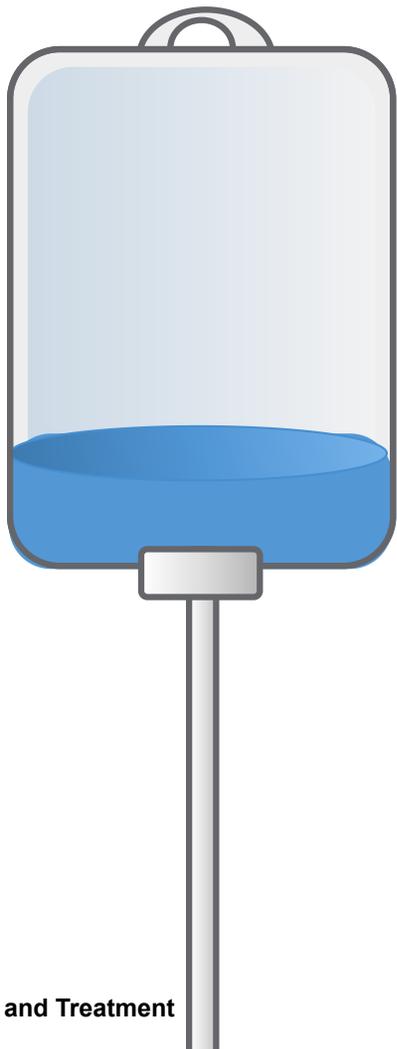
If a total dose of 30mg does not achieve optimal BP control, another agent should be used.

The fall in BP begins within 10 to 30 minutes and lasts for 2 to 4 hours.



Click the I.V. bag to see types of treatment.





Nifedipine - Calcium Channel Blocker (CCB)

Immediate release oral nifedipine capsules should be administered orally [98].

Typical dosing is 10-20 mg orally, repeated in 20 minutes, if needed, then 10-20 mg every 2-6 hours.

- Maximum daily dose is 180 mg.

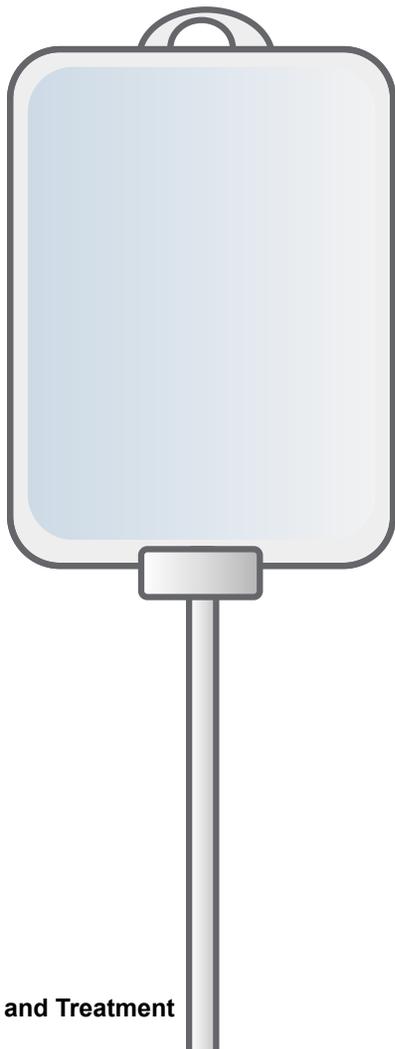
Sustained release nifedipine is not recommended for acute treatment of severe range blood pressures, but can be effective in long term management.

Target BP reached within 23 mins in 70% of women with severe hypertension and 91% reach target within 130 mins.



Click the I.V. bag to see types of treatment.





Nitroglycerin

A good option for treatment of hypertension associated with pulmonary edema [80].

Administer 5mcg/min IV and gradually increase every 3 to 5 minutes to maximum dose of 100mcg/min.

Typically only administered in ICU settings.

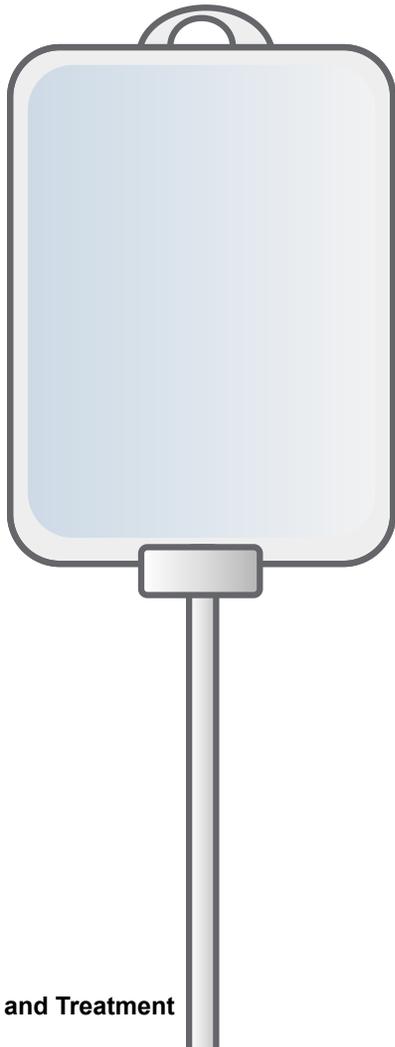
Nicardipine

- If IV labetalol, IV hydralazine and PO instant-release nifedipine are not effective in controlling a patient's blood pressure, a nicardipine drip can be started; however, this is typically only managed in an ER or ICU setting.



*Click here to see
more information.*





Rarely is BP not controlled with the drugs discussed previously.

Nitroprusside is administered as a last resort.

Requesting assistance from maternal fetal medicine, anesthesia or critical care may be required if typical antihypertensive management fails.



Maintenance Antihypertensive Therapy

If a woman is diagnosed with pre-eclampsia with severe features based on severe range BP alone (no other severe features are present), she is a candidate for expectant management and should be started on oral antihypertensive therapy.

Options for oral antihypertensive therapy and typical starting doses include:

- Labetalol 200 mg BID
- Nifedipine XL 30 mg daily

[Click Here to view a video from AJOG: Failure of Physical Transformation and Spiral Artery Atherosclerosis: Their Role in Preeclampsia](#)



<https://www.facebook.com/AJOGthegray/videos/ajog-expert-reviews-failure-of-physiological-transformation-and-spiral-artery-at/397377551523695/>





Target BP

- Systolic 130 to 150mmHg
- Diastolic 80 to 100mmHg

How quickly the BP should be brought to safe levels is controversial.

- Cerebral, myocardial ischemia or infarction can be induced by aggressively reducing the BP.
- It is reasonable to reduce the mean arterial pressure by no more than 25% over two hours and achieving a target of 130-150mmHg systolic and 80-100mmHg diastolic [80].
- Appropriate and prompt management of severe systolic and severe diastolic hypertension is required to reduce risk and have successful, safe clinical outcomes for women with preeclampsia or eclampsia [98].
- There is mounting evidence that patient outcomes improve when standardization of care occurs [98].
- Adverse maternal outcomes have been reduced when introducing standardization of evidence based clinical guidelines for the management of patients with preeclampsia and eclampsia [98].





Review of the use of magnesium sulfate occurs in the next 6 slides for those delivering in the appropriate locations for use of magnesium.



MAGNESIUM SULFATE

- Given for the prevention and treatment of seizures in women with severe features of preeclampsia and eclampsia.
- A loading dose of 6 grams intravenous over 15-20 minutes followed by 2 grams per hour as a continuous infusion is the most common regimen [82-85].
- An alternative regimen is 5 grams intramuscularly into each buttock (total of 10 grams) followed by 5 grams intramuscularly every 4 hours.
- A clear threshold concentration has not been determined but there is a recommendation based on retrospective data to help insure the prevention of convulsions with a therapeutic range of 4.8 - 8.4mg/dL (2-3.5mmol/L) [86].
- Loading doses less than 6 grams are more likely to result in subtherapeutic magnesium levels (less than 4.5mg/dL) [84,87].





MAGNESIUM SULFATE

- Since magnesium sulfate is excreted by the kidneys, dosing should be adjusted in women with renal insufficiency (defined as a serum creatinine greater than 1.0mg/dL).
- Consideration should be given to administer a 4 gram bolus (rather than 6 gram) and the maintenance rate should be reduced to 1 gram/hour.
- Labs for creatinine and magnesium levels should be followed every 6-12 hours.
- Clinical assessments should be performed hourly.
- The maintenance phase is given only if a patellar reflex is present (loss of reflexes being the first manifestation of symptomatic hypermagnesemia), respirations exceed 12 per minute, and the urine output exceeds 100mL per 4 hours.
- Following serum magnesium levels is not required if the woman's clinical status is closely monitored for evidence of potential magnesium toxicity.
- The maintenance dose should be decreased if there is clinical evidence of magnesium toxicity.



Slide 2 of 6



MAGNESIUM SULFATE

Duration of Therapy:

- Magnesium sulfate is typically initiated at the time of diagnosis of preeclampsia with severe features; however, there are no specific guidelines for the duration of therapy if expectant management is planned.
- Continuing the infusion for 12-24 hours after initial diagnosis is typical.
- If a patient has been expectantly managed, magnesium sulfate is restarted whenever delivery is initiated and it is continued for 24 hours postpartum.
- If immediate delivery is planned, magnesium sulfate is started and continued until 24 hours postpartum.
- Diuresis (greater than 4L/day) is believed to be the most accurate clinical indicator of resolution of preeclampsia/eclampsia, but is not a guarantee against the development of seizures [88].

Mechanism of Anticonvulsant Action:

- The mechanism for the anticonvulsant effects of magnesium sulfate has not been clearly defined.
- The primary effect is thought to be central.
- Another theory is that it promotes vasodilatation of constricted cerebral vessels by opposing calcium-dependent arterial vasospasm, thereby reducing cerebral barotrauma [89].



MAGNESIUM SULFATE

Complications and Side Effects:

- Rapid infusion of magnesium sulfate causes diaphoresis, flushing, and warmth, probably related to peripheral vasodilation.
- Nausea, vomiting, headache, muscle weakness, visual disturbances, and palpitations can also occur.
- Dyspnea or chest pain may be symptoms of pulmonary edema, which is a rare side effect.
- Magnesium toxicity is uncommon in women with normal renal function [90].

Toxicity is Related to Serum Magnesium Concentration:

- Loss of deep tendon reflex (DTR) occurs at 7 to 10mEq/L (8.5 to 12mg/dL or 3.5 to 5.0mmol/L).
- Respiratory paralysis at 10 to 13mEq/L (12 to 16mg/dL or 5.0 to 6.5mmol/L).
- Cardiac conduction is altered at >15mEq/L (>18mg/dL or >7.5mmol/L).
- Cardiac arrest occurs at >25mEq/L (>30mg/dL or >12.5mmol/L) [150].
- Calcium gluconate (1 gram IV over 5 to 10 minutes) should be administered only to counteract life-threatening symptoms of magnesium toxicity, such as cardiorespiratory compromise.



MAGNESIUM SULFATE

- Magnesium sulfate is contraindicated in women with myasthenia gravis since it can precipitate a severe myasthenic crisis.
- Neuromuscular blockade and hypotension due to concurrent use of magnesium sulfate and calcium channel blockers have been described in case reports, but the risk appears to be minimal [92].
- Although magnesium sulfate is a weak tocolytic, labor duration does not appear to be affected by magnesium sulfate administration [93].



MAGNESIUM SULFATE

When a patient is therapeutic on magnesium sulfate and has a seizure, the first recommendation is an additional loading dose of magnesium sulfate 2g IV over five minutes. If she continues to have seizure activity, alternative anti-convulsants should be considered:

- Lorazepam 4mg IV over 2-5 minutes (repeating in 5-15 min prn) to maximum of 8mg in 12 hours
- Diazepam 5-10mg IV slowly (repeating Q15 minutes up to 30mg)
- Midazolam 1-2mg IV (may repeat in 5-10 minutes)
- Phenytoin 1000mg IV over 20 minutes
- Involvement of the anesthesia team for further options [101]





Response

Standardize protocols should include [24]:

- onset and duration of magnesium therapy
- advance preparation for seizure prophylaxis and magnesium toxicity
- transfer to a proper facility if BP systolic is 160mm Hg or more or diastolic is 110mm Hg or more two measurements within 15 minutes.
- monitor cases of borderline severe hypertension (150-159 mm Hg/ 105-109mm Hg) for progression to severe ranges
- initiate treatment within 60 minutes of verification after first severe blood pressure range
- escalation measures for ongoing observation and management

Postpartum follow up

- schedule a follow up within three days of discharge date and this visit should include:
 - blood pressure check
 - discussion of signs/symptoms of worsening hypertension
 - who to contact if signs/symptoms continue
 - information about where to go such as urgent care or Emergency Department, if signs/symptoms worsen

Aligning this above information with the patient's health literacy, culture, language and accessibility needs will always be necessary.



ACOG

SOGC

First-Line Therapy

- Labetalol
- Nifedipine

Avoiding:

- Angiotensin-converting enzyme inhibitors
- Angiotensin receptor blockers
- Renin inhibitors
- Mineralocorticoid receptor antagonists

Treatment Goal: BP between 120/80 to 160/105mmHg.



*Click the grey arrow
for more information.*





ACOG

SOGC

The American College of Obstetricians and Gynecologists (ACOG) Committee Opinion on emergent therapy acute onset of severe hypertension in pregnancy and postpartum recommends treatment when:

- Severe systolic >160mmHg
- Severe diastolic >110mmHg
- Or both



To achieve BP of 140-150/90-100mmHg [81].
ACOG Committee Opinion on acute onset of severe hypertension in pregnancy and postpartum **recommends first-line treatment with:**

- Labetalol
- Hydralazine
- Oral Nifedipine

Recommend using short-acting preparation of oral nifedipine.





ACOG

SOGC

The Society of Obstetricians and Gynaecologists of Canada (SOGC) guidelines recommend ***antihypertensive treatment for new onset:***

- Systolic BP >160mmHg or
- Diastolic BP >110mmHg

Goal BP <160/110mmHg [95].





For those delivering in the hospital, here is a common order set for severe hypertension

- 1 Notify physician if systolic BP measurement is greater than or equal to 160mmHg or if diastolic BP measurement is greater than or equal to 110mmHg.
- 2 Fetal surveillance if undelivered and fetus is viable.
- 3 If severe BP elevations persist for 15 minutes or longer administer labetalol 20mg IV over 2 minutes.
- 4 Repeat BP measurements in 10 minutes and record results.
- 5 If either BP threshold is still exceeded, administer labetalol 40mg over 2 minutes.
- 6 If BP is below threshold, continue BP monitoring closely. Repeat BP measurement in 10 minutes and record results.
- 7 If either BP threshold is still exceeded, administer labetalol 80mg IV over 2 minutes. If BP is below threshold, continue to monitor BP closely.
- 8 Repeat BP measurement in 10 minutes and record results.
- 9 If either BP threshold is still exceeded, administer hydralazine 10mg IV over 2 minutes. If BP is below threshold, continue to monitor BP closely.
- 10 Repeat BP measurement in 10 minutes and record results.
- 11 If either BP threshold is still exceeded, obtain emergency consultation from Maternal Fetal Medicine (MFM), Internal Medicine (IM), anesthesia or Critical Care subspecialty.
- 12 Give specific antihypertensive meds per order.
- 13 Once BP thresholds are achieved repeat BP measurements every 10 minutes for an hour, then every 15 minutes for an hour, then every 30 minutes for an hour, then every hour for 4 hours [96].





Multi-organ involvement may result in fetal, perinatal and maternal morbidity and mortality [97].

Maternal complications include but are not limited to:

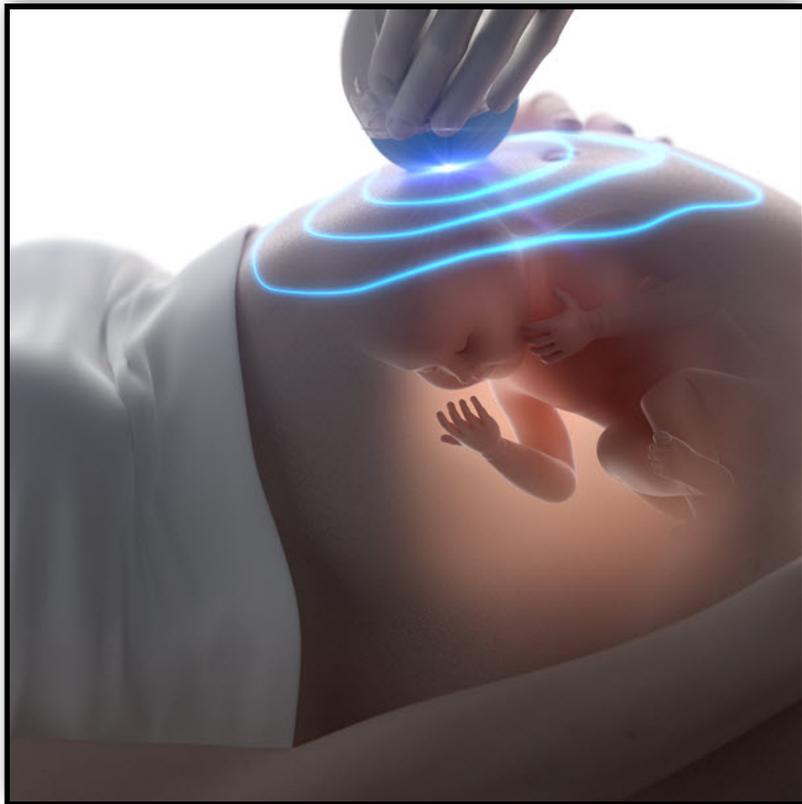
- Stroke
- Pulmonary edema
- Hepatic failure
- Jaundice
- Eclampsia



Women developing eclampsia are at great risk for:

- Maternal death
- Need for assisted ventilation
- Adult respiratory distress syndrome (ARDS)
- Embolism
- Placenta abruption
- Acute renal failure [98]
- Posterior Reversible Encephalopathy Syndrome (PRES)





Fetal complications

- Oligohydramnios
- Fetal growth restriction
 - Up to 30% of fetus' of women with preeclampsia
- Metabolic acidosis
- APGAR score <3 at 5 minutes
- Umbilical artery pH <7
- Positive pressure ventilation (PPV) for <5 minutes



Delivery Recommendations

- Gestational Hypertension - 37 weeks
- Preeclampsia without severe features - 37 weeks
- Preeclampsia with severe features - 34 weeks



In a pregnant patient, preeclampsia is the most common cause of:

- Hypertension
- Thrombocytopenia
- Liver abnormalities
- Renal abnormalities

Other health care conditions should be considered and excluded:

- Acute fatty liver of pregnancy (AFLP)
- Thrombotic thrombocytopenic purpura (TTP)
- Systemic Lupus Erythematosus (SLE) exacerbation

The [California Maternal Quality Care Collaborative](#) developed a tool to help differentiate between these disorders. It is a free download from their website [101].





Table 5. Differentiation between Preeclampsia, HELLP Syndrome, Acute Fatty Liver Disease of Pregnancy (AFLD), Thrombotic Thrombocytopenia Purpura (TTP), Hemolytic Uremia Syndrome (HUS) *

	Plts	LFTs	Bili	Cr	LDH	Glu	DIC	CNS
Preeclampsia	±	±	±	±	±	→	±	±
HELLP	↓/↓↓	↑↑	↑	±	↑	→	±	±
AFLD	↓↓	↑↑	↑↑↑	↑	↑	↓↓↓	↑↑↑	±
TTP	↓↓↓	↑	↑	↑	↑↑	→	±	++
HUS	↓	↑↑	↑↑	↑↑↑	↑	→	±	±

AFLD: Acute Fatty Liver Disease of Pregnancy; TTP: Thrombotic Thrombocytopenic Purpura; HUS: Hemolytic Uremia Syndrome; Plts: platelet count; LFTs: liver function test; Bili: total bilirubin level; LDH: Lactate Dehydrogenase; Glu: glucose; DIC: Disseminated Intravascular Coagulation; CNS: Central Nervous System symptoms (confusion, visual changes, headache)

*Arrows represent relative changes: one arrow equals some increase; two arrows indicate moderate increase, and three arrows equal very high increase.

Lyndon A, Lagrew D, Shields L, Main E, Cape V. Improving Health Care Response to Obstetric Hemorrhage. (California Maternal Quality Care Collaborative Toolkit to Transform Maternity Care) Developed under contract #11-10006 with the California Department of Public Health; Maternal, Child and Adolescent Health Division; Published by the California Maternal Quality Care Collaborative, 3/17/15. Reprinted with permission.

Another issue with maternal morbidity and mortality arises from lack of communication. Misdiagnosis and denial of severity of women's illness along with delays or failures in treatment are key factors contributing to fatal outcomes. Lack of listening skills or responsiveness to concern and failure in communication including silence in the face of clinical concern are likely to have contributed to delays, misdiagnosis, and treatment failures.

Teams are found to be effective with collective monitoring, crosschecking one another, and clinical processes in place to proactively identify potential problems. Empowering all staff to "stop the line" or formally interrupt the plan of care and check safety when they observe potential for harm. Listening to a peer whether or not they agree and establishing strategies for communication and teamwork will help to build trust in these complex settings [101].



A thorough history, physical exam, and data collection should be completed in a timely fashion upon admission of any pregnant woman who presents with hypertension or symptoms concerning for preeclampsia.

The ability to predict preeclampsia is limited.

Accurate identification of women at risk, early diagnosis, and prompt management can improve outcomes.

Consideration for implementing a Preeclampsia Early Recognition Tool (PERT) is detrimental to maternal and fetal health. Please review the PERT at this link:

<https://www.pdfFiller.com/101064292-2036pdf-Preeclampsia-Early-Recognition-Tool-PERT-Publication-2036-Preeclampsia-Early-Recognition-Tool-PERT-Publication-2036-health-ny->



It is imperative for the health and well-being of pregnant and postpartum women to have an integrated system set forth by the Alliance for Innovation on Maternal Health (AIM) [24].

The following slides review this information of actionable items.

Conclusion





Readiness

Drills to simulate patients that emphasize:

- all elements of the facility severe hypertension emergency management plan
- patient centered, empathetic, trauma-informed care

1 of 2 **➤➤**



Click the arrows to display more information about readiness

Readiness





Readiness

Referral resources and communication to ensure that:

- maternal and neonatal transfer protocol is in place
- implementation of resource mapping to identify local resources and support services so that this information is available.
- consider providing a blood pressure cuff; do provide educational materials and information on who to call for concerns for the patient to take home

◀ 2 of 2 ▶





Order recommended labs:

- protein/creatinine
- CBC with platelets
- LDH
- AST
- ALT

Screen the patient for her:

- medical needs
- mental and behavioral needs
- structural and social drivers of health

Patient education to include:

- who to contact
- review of warning signs
- reinforce value of outpatient follow up
- all should align with health literacy, cultural needs and language preference.

Recognition





Response

Standardize protocols should include [24]:

- onset and duration of magnesium therapy
- advance preparation for seizure prophylaxis and magnesium toxicity
- transfer to a proper facility if BP systolic is 160mm Hg or more or diastolic is 110mm Hg or more two measurements within 15 minutes.
- monitor cases of borderline severe hypertension (150-159 mm Hg/ 105-109mm Hg) for progression to severe ranges
- initiate treatment within 60 minutes of verification after first severe blood pressure range
- escalation measures for ongoing observation and management

Postpartum follow up

- schedule a follow up within three days of discharge date and this visit should include:
 - blood pressure check
 - discussion of signs/symptoms of worsening hypertension
 - who to contact if signs/symptoms continue
 - information about where to go such as urgent care or Emergency Department, if signs/symptoms worsen

Aligning this above information with the patient's health literacy, culture, language and accessibility needs will always be necessary.



Reporting

Multidisciplinary case review which may identify:

- alignment with standard policy and procedures
- appropriate updates to standard policies and procedures for future events
- other opportunities for improvement
- consistent issues should be reported via established pathways



Respectful Care Element



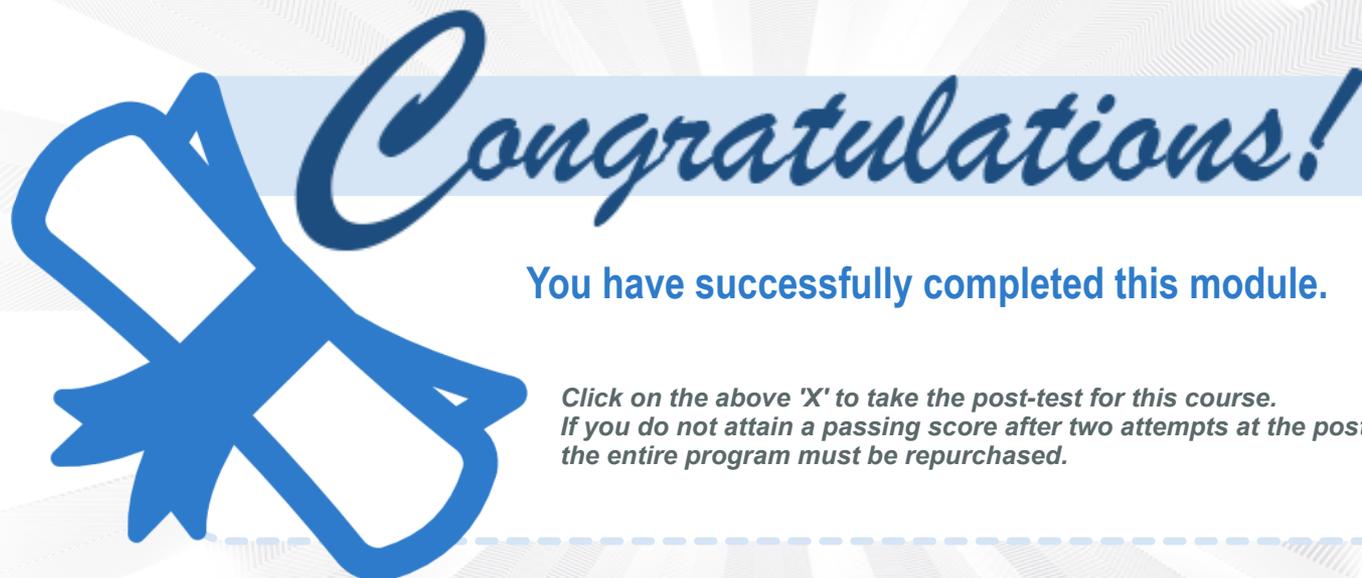
Open, transparent, and empathetic communication with pregnant and postpartum people and their support network.

- Establish and maintain a mechanism for patient and staff to identify inequitable care episodes of miscommunication or disrespect.
- Develop a plan to address reported cases of inequitable care, miscommunication or disrespect.

Include the patient as part of the multidisciplinary team to establish trust, and bidirectional shared decision making with patient values and goals as the primary driver of this process.

As Black, Indigenous, and Hispanic people experience maternal mortality and severe maternal morbidity at disproportionately higher rates because of systemic racism, but not race itself, it is necessary to mitigate bias by having a high index of suspicion for a contributing clinical condition, such as severe hypertension, in these populations.

Patient support networks may include nonfamilial supports, such as doulas and home visitors, who, with the postpartum person's permission, should be welcomed when any teaching or planning is provided.



Congratulations!

You have successfully completed this module.

*Click on the above 'X' to take the post-test for this course.
If you do not attain a passing score after two attempts at the post-test
the entire program must be repurchased.*

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