Hypertensive Disorders

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The previous basic classification was retained and describes **four** types of hypertensive disease:

1. **Preeclampsia and eclampsia** syndrome
2. **Chronic hypertension** of any etiology
3. **Preeclampsia superimposed** on chronic hypertension
4. **Gestational hypertension**—definitive evidence for the preeclampsia syndrome does not develop and hypertension resolves by 12 weeks postpartum.

Diagnosis of Hypertensive Disorders

Hypertension is diagnosed empirically when appropriately taken blood pressure **exceeds 140 mm Hg systolic or 90 mm Hg** diastolic. Previously, incremental increases of **30 mm** Hg systolic or **15 mm** Hg diastolic above blood pressure values taken at midpregnancy had also been used as diagnostic criteria, even when absolute values were <140/90 mm Hg. These incremental changes are no longer used to define hypertension, but it is recommended that such women be **observed more closely** because **eclamptic** seizures develop in some whose blood pressures have stayed below 140/90 mm Hg Also, a sudden rise in mean arterial pressure but still in a normal range—"**delta hypertension**"—may signify preeclampsia

Concept of “Delta Hypertension”

We use the term delta hypertension to describe this rather **acute rise in blood pressure, are still <140/90 mm Hg,** Some of these women will go on to have obvious preeclampsia, and some even develop eclamptic seizures or HELLP (hemolysis, elevated liver enzyme levels, low platelet count) syndrome while still normotensive

Gestational Hypertension

blood pressures reach 140/90 mm Hg or greater for the first time after midpregnancy, but in whom proteinuria is notidentified. Almost half of these women subsequently develop preeclampsia
syndrome.

gestational hypertension is reclassified by some as transient hypertension if evidence for preeclampsia does not develop and the blood pressure returns to normal by 12 weeks postpartum.

Preeclampsia Syndrome

Preeclampsia is best described as a **pregnancy-specific syndrome** that can affect **virtually every organ** system. In addition, it heralds a higher incidence of cardiovascular disease later in life . Although preeclampsia is much more than simply gestational hypertension with proteinuria, appearance of proteinuria **remains an important** diagnostic criterion.
Thus, proteinuria is an objective marker and reflects the system-wide endothelial leak that characterizes the preeclampsia syndrome.
In some women with the preeclampsia syndrome, **neither** overt proteinuria nor fetal-growth restriction are features.

TABLE 40-1. Classification and Diagnosis of Pregnancy-Associated Hypertension

Condition	Criteria Required
Gestational hypertension	BP >140/90 mm Hg after 20 weeks in previously normotensive women
Preeclampsia: Hypertension plus	
Proteinuria	• ≥300 mg/24 h, or • Urine protein: creatinine ratio ≥0.3, or • Dipstick 1+ persistent[a]
	or
Thrombocytopenia	• Platelet count <100,000/μL
Renal insufficiency	• Creatinine level >1.1 mg/dL or doubling of baseline[b]
Liver involvement	• Serum transaminase levels[c] twice normal
Cerebral symptoms	• Headache, visual disturbances, convulsions
Pulmonary edema	—

TABLE 40-2. Indicators of Severity of Gestational Hypertensive Disorders[a]

Abnormality	Nonsevere[b]	Severe
Diastolic BP	<110 mm Hg	≥110 mm Hg
Systolic BP	<160 mm Hg	≥160 mm Hg
Proteinuria[c]	None to positive	None to positive
Headache	Absent	Present
Visual disturbances	Absent	Present
Upper abdominal pain	Absent	Present
Oliguria	Absent	Present
Convulsion (eclampsia)	Absent	Present
Serum creatinine	Normal	Elevated
Thrombocytopenia (<100,000/μL)	Absent	Present
Serum transaminase elevation	Minimal	Marked
Fetal-growth restriction	Absent	Present
Pulmonary edema	Absent	Present
Gestational age	Late	Early

Preeclampsia Superimposed on Chronic

Chronic underlying hypertension is diagnosed in women with documented blood pressures >140/90 mm Hg before pregnancy or before 20 weeks' gestation, or both. If new-onset **or worsening baseline hypertension is accompanied by new-onset proteinuria or other findings** listed in then superimposed preeclampsia is diagnosed. Compared with "pure"preeclampsia, superimposed preeclampsia commonly develops earlier in pregnancy.

INCIDENCE AND RISK FACTORS

- **Young and nulliparous** women developing preeclampsia,
- older women are at greater risk for **chronic hypertension** with superimposed preeclampsia.
- **race and ethnicity**
- the **metabolic syndrome** and hyperhomocysteinemia
- Pregnancies with a **male fetus**
- **smoking** during pregnancy
- Other factors are **human immunodeficiency virus (HIV) seropositivity and sleep disordered.**

TABLE 40-3. Selected Clinical Risk Factors for Preeclampsia

Risk Factor	Pregnancies (millions)	Pooled Unadjusted Relative Risk (95% CI)
SLE	2.43	2.5 (1.0–6.3)
Nulliparity	2.98	2.1 (1.9–2.4)
Age >35	5.24	1.2 (1.1–1.3)
Prior stillbirth	0.063	2.4 (1.7–3.4)
CKD	0.97	1.8 (1.5–2.1)
ART	1.46	1.8 (1.6–2.1)
BMI >30	5.92	2.8 (2.6–3.1)
Multifetal	7.31	2.9 (2.6–3.1)
Prior abruption	0.29	2.0 (1.4–2.7)
Diabetes	2.55	3.7 (3.1–4.3)
Prior preeclampsia	3.72	8.4 (7.1–9.9)
CHTN	6.59	5.1 (4.0–6.5)
APA	0.22	2.8 (1.8–4.3)

ETIOPATHOGENESIS

- Are exposed to **chorionic villi for the first time**
- Are exposed to a **superabundance of chorionic villi**, as with twins or hydatidiform mole
- Have **preexisting conditions** associated with endothelial cell activation orinflammation, such as diabetes, obesity, cardiovascular or renal disease, immunological disorders, or hereditary influences
- Are **genetically predisposed** to hypertension developing during pregnancy.

A **fetus is not a requisite** for preeclampsia to develop. And, although chorionic villi are essential, they need not be intrauterine. For example, preeclampsia can develop with an abdominal pregnancy

Etiology

Those currently considered important include:

1. Placental implantation with **abnormal trophoblastic invasion** of uterine vessels
2. **Immunological maladaptive** tolerance between maternal, paternal (placental),
and fetal tissues
3. Maternal maladaptation to cardiovascular or inflammatory changes of normal pregnancy
4. Genetic factors including inherited predisposing genes and epigenetic influences.

Pathogenesis

- Vasospasm
- Endothelial Cell Injury
- Increased Pressor Responses
- Angiogenic and Antiangiogenic Proteins

Some Methods to Prevent Preeclampsia That Have Been Evaluated in Randomized Trials

Dietary manipulation—low-salt diet, calcium or fish oil supplementation

Exercise—physical activity, stretching

Cardiovascular drugs—diuretics, antihypertensive drugs

Antioxidants—ascorbic acid (vitamin C), α-tocopherol (vitamin E), vitamin D

Antithrombotic drugs—low-dose aspirin, aspirin/dipyridamole, aspirin + heparin, aspirin + ketanserin

PREECLAMPSI MANAGMENT

The basic management objectives for any pregnancy complicated by preeclampsia are:

(1) **termination** of pregnancy with the least possible trauma to mother and fetus

(2) **birth of a healthy newborn** that subsequently thrives

(3)complete restoration of **health to the mother**

Early Diagnosis of Preeclampsia

Women with overt new-onset hypertension—either diastolic pressures ≥90 mm Hg or systolic pressures ≥140 mm Hg—are admitted to determine if the increase is due to preeclampsia, and if so, to evaluate its severity

Evaluation

With hospitalization, a **systematic evaluation** is instituted to include:

- **Detailed examination**, which is coupled with daily scrutiny for clinical finding ssuch as headache, visual disturbances, epigastric pain, and rapid weight gain
- **Daily** weight measurement
- Quantification of proteinuria or **urine protein:creatinine ratio** on **admittance** andat least every **2 days** thereafter
- **Blood pressure** readings with an appropriate-size cuff every 4 hours, except between 2400 and 0600 unless previous readings are elevated
- Measurements of plasma or serum **creatinine** and **hepatic transaminase** levelsand a **hemogram** that includes a **platelet count**. The frequency of testing is determined by hypertension severity. Although some recommend measurement of serum uric acid and lactate dehydrogenase levels and coagulation studies,their value has been questioned .
- Evaluation of **fetal size and well-being and amnionic fluid volume**, by either physical examination or sonography

Consideration for Delivery

Termination of pregnancy is the only cure for preeclampsia. Headache, visual disturbances, or epigastric pain are indicative that convulsions may be imminent, and oliguria is another ominous sign. **Severe preeclampsia demands anticonvulsant and often antihypertensive therapy, followed by delivery.** Treatment for eclampsia is identical. The prime objectives are to forestall convulsions, to prevent intracranial hemorrhage and serious damage to other vital organs, and to deliver a healthy newborn.

Labor induction is carried out, usually with preinduction cervical ripening with a prostaglandin or
osmotic dilator

When the fetus is **preterm**, the tendency is to temporize in the hope that additional weeks in utero will reduce the risk of neonatal death or serious morbidity from prematurity. Such a policy certainly is justified in milder cases.

Assessments of fetal well-being and placental function are performed, especially when the fetus is immature. Most recommend frequent performance of **nonstress testing or biophysical profiles** to assess fetal well-being.

Hospitalization versus Outpatient Management

For women **with mild-to-moderate stable hypertension**—whether or not preeclampsia has been confirmed—monitoring is continued. During surveillance, reduced physical activity throughout much of the day, at least intuitively, seems beneficial. That said, **complete bed rest** is **not** recommended .this is pragmatically unachievable because of the severe restrictions it places on otherwise well women. Also, it likely predisposes to thromboembolism. disease.

further hospitalization is not warranted if hypertension abates within a few days. Outpatient management may continue as long as preeclampsia syndrome does not worsen and fetal jeopardy is not suspected. Sedentary activity throughout the greater part of the day is recommended. These women are instructed in detail to report symptoms. Home bood pressure and urine protein monitoring or frequent evaluations by a visiting nurse may prove beneficial.

Preterm delivery before 34 and before 37 weeks' gestation was increased twofold in the outpatient.

Antihypertensive Therapy for Mild-to-Moderate Hypertension

The use of antihypertensive drugs to prolong pregnancy or modify perinatal outcomes in pregnancies complicated by various hypertensive disorders has been of considerable interest.

Except for fewer episodes of severe hypertension, **none of these studies** showed any benefits from antihypertensive treatment

Expectant Management Recommendations

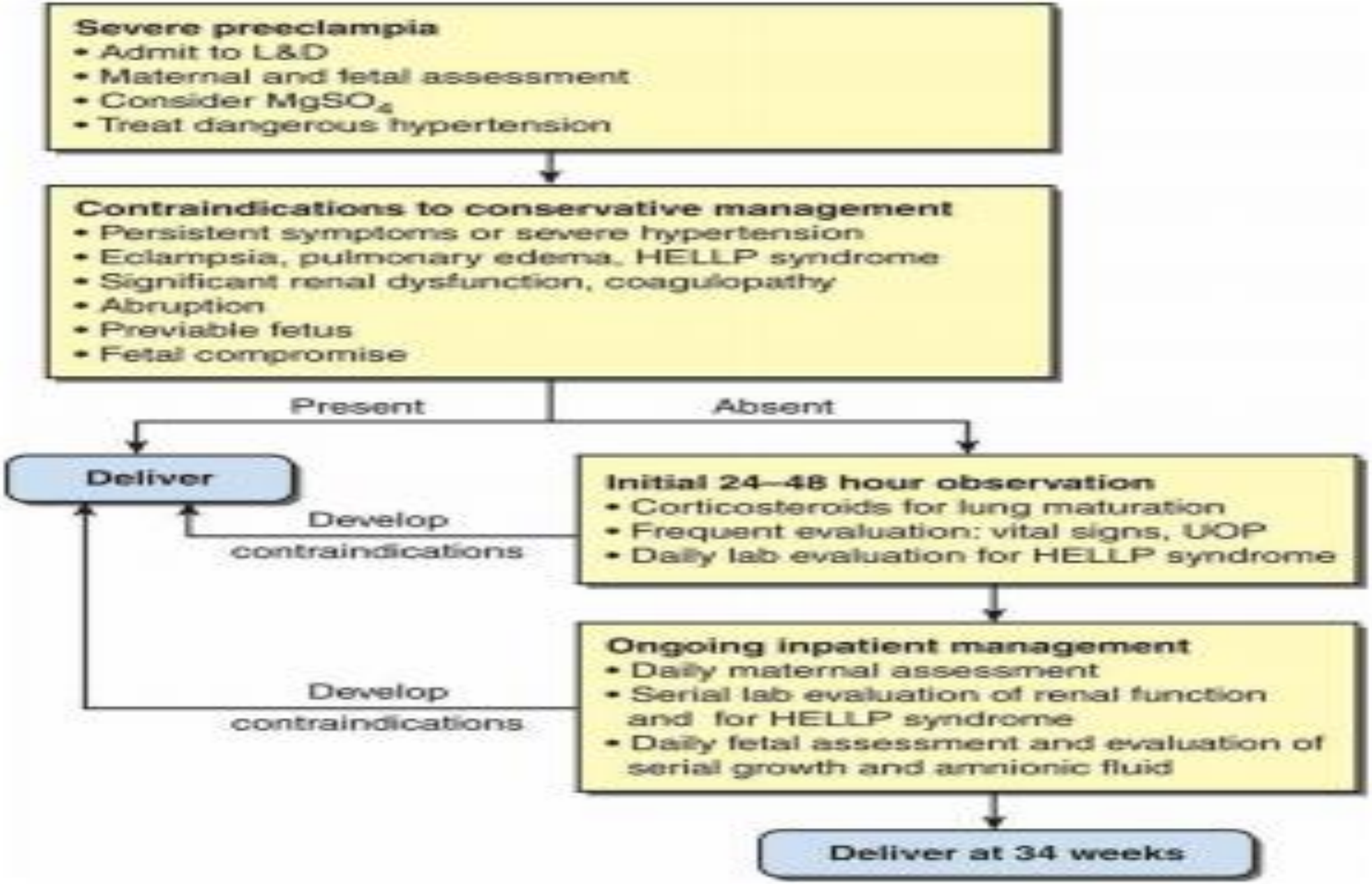
TABLE 40-10. Indications for Delivery in Women <34 Weeks' Gestation Managed Expectantly

Corticosteroid Therapy for Lung Maturation[a] and Delivery after Maternal Stabilization:

- Uncontrolled severe hypertension
- Eclampsia
- Pulmonary edema
- Placental abruption
- Disseminated intravascular coagulation
- Nonreassuring fetal status
- Fetal demise

Corticosteroid Therapy for Lung Maturation—Delay Delivery 48 hr If Possible:

- Preterm ruptured membranes or labor
- Thrombocytopenia <100,000/μL
- Hepatic transaminase levels twice upper limit of normal
- Fetal-growth restriction
- Oligohydramnios
- Reversed end-diastolic Doppler flow in umbilical artery
- Worsening renal dysfunction


Severe preeclampia
• Admit to L&D
• Maternal and fetal assessment
• Consider $MgSO_4$
• Treat dangerous hypertension
Contraindications to conservative management
• Persistent symptoms or severe hypertension
• Eclampsia, pulmonary edema, HELLP syndrome
• Significant renal dysfunction, coagulopathy
• Abruption
• Previable fetus
• Fetal compromise
Present
Absent
Deliver
Develop contraindications
Initial 24–48 hour observation
• Corticosteroids for lung maturation
• Frequent evaluation: vital signs, UOP
• Daily lab evaluation for HELLP syndrome
Develop contraindications
Ongoing inpatient management
• Daily maternal assessment
• Serial lab evaluation of renal function and for HELLP syndrome
• Daily fetal assessment and evaluation of serial growth and amnionic fluid
Deliver at 34 weeks

ECLAMPSIA

Preeclampsia complicated by generalized tonic-clonic convulsions

appreciably raises the risk to both mother and fetus.

clamptic seizures may be violent, and the woman must be protected, especially her airway. So forceful are the muscular movements that the woman may throw herself out of her bed, and if not protected, her tongue is bitten by the violent action of the jaws This phase, in which the muscles alternately contract and relax, may last approximately a minute. Gradually, the muscular movements become smaller and less frequent, and finally the woman lies motionless.

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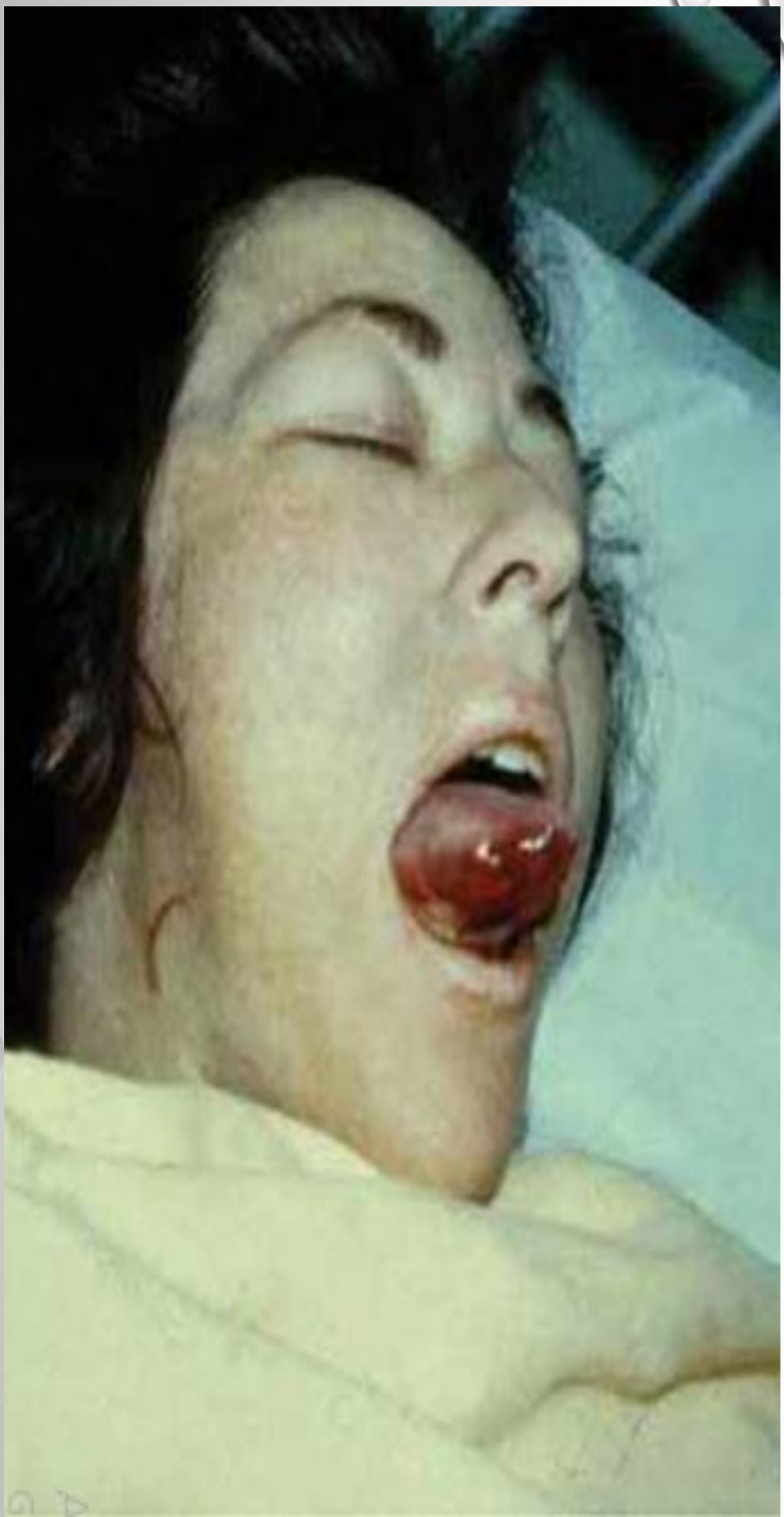

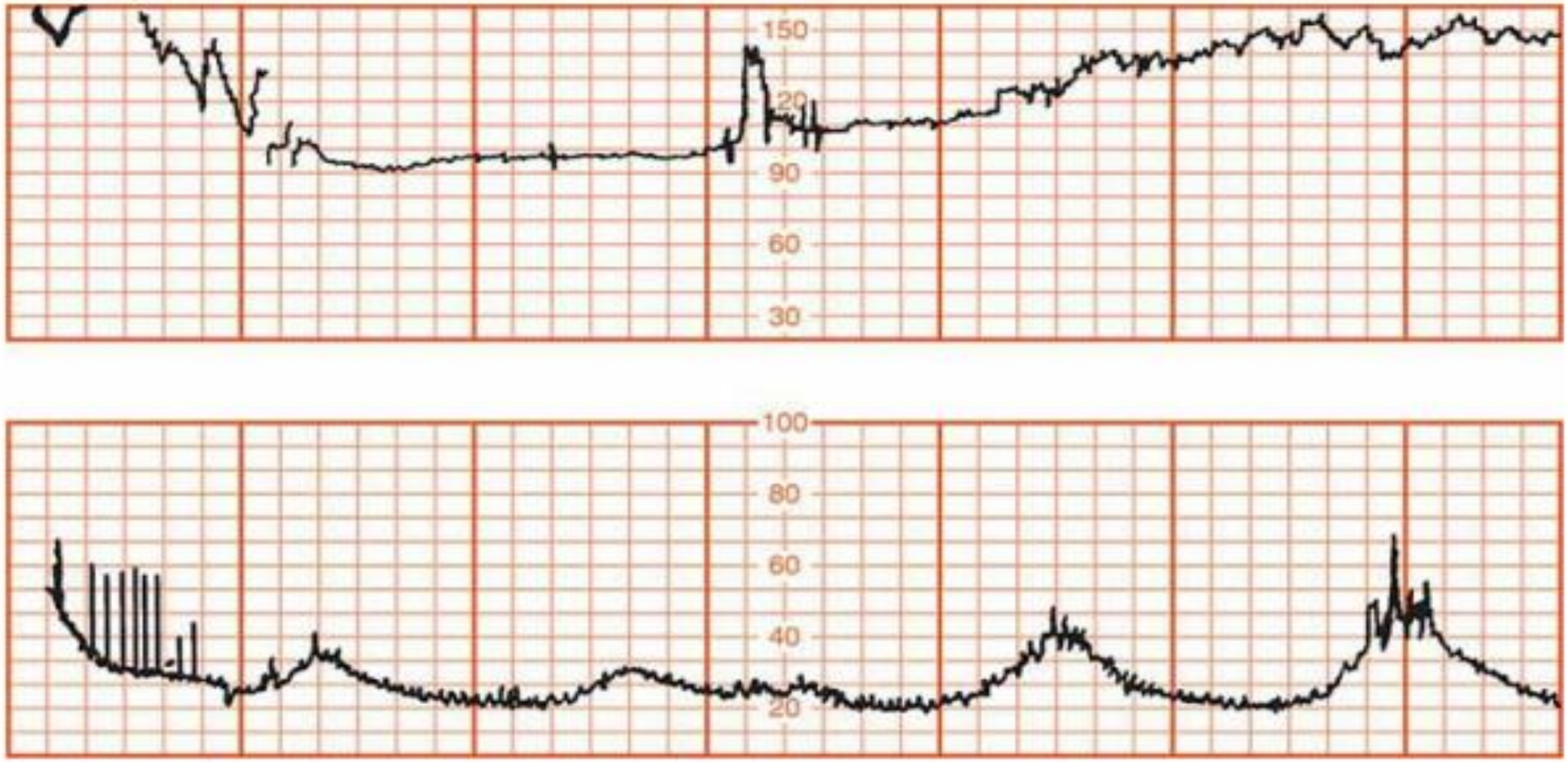

FIGURE 40-18 Fetal heart rate tracing shows fetal bradycardia following an intrapartum eclamptic convulsion. Bradycardia resolved and beat-to-beat variability returned approximately 5 minutes following the seizure.

Management of Eclampsia

Magnesium sulfate is highly effective to **prevent** convulsions in women with preeclampsia and to stop them in those with eclampsia.

it is still in use today:

1. Control of convulsions using an intravenously **administered loading dose of magnesium sulfate** that is followed by a maintenance dose, usually intravenous,of magnesium sulfate
2. **Intermittent administration of an antihypertensive medication** to lower blood pressure whenever it is considered dangerously high
3. **Avoidance of diuretics unless pulmonary edema is obvious**, limitation of intravenous fluid administration unless fluid loss is excessive, and avoidance of hyperosmotic agents
4. **Delivery of the fetus** to resolve preeclampsia.

. Magnesium Sulfate Dosage Schedule for Severe Preeclampsia and eclampsia

Continuous Intravenous (IV) Infusion

Give 4- to 6-g loading dose of magnesium sulfate diluted in 100 mL of IV fluid administered over 15–20 min
Begin 2 g/hr in 100 mL of IV maintenance infusion. Some recommend 1 g/hr
Monitor for magnesium toxicity:
- Assess deep tendon reflexes periodically
- Some measure serum magnesium level at 4–6 hr and adjust infusion to maintain levels between 4 and 7 mEq/L (4.8 to 8.4 mg/dL)
- Measure serum magnesium levels if serum creatinine ≥1.0 mg/dL

Magnesium sulfate is discontinued 24 hr after delivery

Intermittent Intramuscular Injections

Give 4 g of magnesium sulfate ($MgSO_4 \cdot 7H_2O$ USP) as a 20% solution intravenously at a rate not to exceed 1 g/min
Follow promptly with 10 g of 50% magnesium sulfate solution, one half (5 g) injected deeply in the upper outer quadrant of each buttock through a 3-inch-long 20-gauge needle. (Addition of 1.0 mL of 2% lidocaine minimizes discomfort.) If convulsions persist after 15 min, give up to 2 g more intravenously as a 20% solution at a rate not to exceed 1 g/min. If the woman is large, up to 4 g may be given slowly.
Every 4 hr thereafter, give 5 g of a 50% solution of magnesium sulfate injected deeply in the upper outer quadrant of alternate buttocks, but only after ensuring that:
- The patellar reflex is present,
- Respirations are not depressed, and
- Urine output the previous 4 hr exceeded 100 mL

Magnesium sulfate is discontinued 24 hr after delivery

Severe Hypertension Management

Dangerous hypertension can cause **cerebrovascular hemorrhage** and hypertensive encephalopathy, and it can trigger **eclamptic convulsions** in women with preeclampsia. Other complications include **placental abruption and congestive heart failure** induced by elevated hypertensive afterload.

Task Force recommend treatment to lower systolic pressures to or below 160 mm Hg and diastolic pressures to or below 110 mm Hg.

The three most commonly employed are **hydralazine, labetalol, and nifedipine.**

Hydralazine

This is probably still the most commonly used antihypertensive agent in the United States for treatment of women with severe gestational hypertension. Hydralazine is administered **intravenously with a 5- to 10-mg initial dose,** and this is followed by 10-mg doses at 15- to 20-minute intervals until a satisfactory response is achieved. Although we will administer a third dose, the American College of Obstetricians and Gynecologists (2017a) recommends labetalol therapy if severe hypertension persists after the second dose

Labetalol

This effective intravenous antihypertensive agent is an α- and nonselective βblocker. Some prefer its use over hydralazine **because of fewer side effects**. At Parkland Hospital, **we give 10 mg intravenously initially**. If the blood pressure has not decreased to the desirable level in 10 minutes, then 20 mg is given. The next 10-minute incremental dose is 40 mg and is followed by another 40 mg if needed.If a salutary response is not achieved, then an 80-mg dose is given.. If hypertension persists, hydralazine is then given

Nifedipine

This **orally** administered calcium-channel blocking agent has become popular because of its efficacy to control acute pregnancy-related hypertension.

Nifedipine given sublingually is **no** longer recommended.

Diuretics

We use antepartum furosemide or similar drugs solely to treat pulmonary edema.

Fluid Therapy

Lactated Ringer solution is administered routinely at a rate between **60 and 125 mL per hour**, unless fluid loss is unusual from vomiting, diarrhea, or diaphoresis, or, more likely, excessive blood loss with delivery.

TABLE 40-15. Selective versus Universal Magnesium Sulfate Prophylaxis: Parkland Hospital Criteria to Define Severe Gestational Hypertension or Preeclampsia

In a woman with new-onset proteinuric hypertension, at least one of the following criteria is required:
Systolic BP ≥160 or diastolic BP ≥110 mm Hg
Proteinuria ≥2+ by dipstick in a catheterized urine specimen
Serum creatinine >1.1 mg/dL
Platelet count <100,000/μL
Aspartate aminotransferase (AST) elevated two times above upper limit of normal range
Persistent headache or scotomata
Persistent midepigastric or right-upper quadrant pain


The End