PLAN OF LECTURE

2. Early gestosis:
   • etiopathogenesis;
   • clinical symptoms;
   • diagnostics;
   • management of pregnant women with various forms of early gestosis.
3. Hypertensive disorders in pregnancy:
   • etiopathogenesis;
4. HELLP-syndrom
Gestoses in pregnant women are referred to pregnant induced diseases that occur in connection with the development of a fetal egg which disappear after childbirth or abortion.
The forms of gestoses are distinguished according to the time of their occurrence and clinical manifestations:

1. **Early gestoses**: vomiting (emesis gravidarum), severe vomiting (Hyperemesis gravidarum), hypersalivation.

2. **Rare forms of gestoses**: dermatoses gravidarum, chorea gravidarum, jaundice of pregnancy, acute fatty liver of pregnancy.

3. **Late gestoses**: hypertension, proteinuria, oedema, mild and severe preeclampsia, eclampsia, HEELP-syndrom.
Theories:

1) **Hormonal.** The peak level of HCG with higher biological activity provokes vomiting in early pregnancy. Progesterone excess to relaxes of the cardiac sphincter and impaired gastric motility and reflux.

2) **Allergic or autoimmunological** reaction of the body to bioactive substances that produce the ovaries (women with adrenal insufficiency and hypothyroidism have autoimmune polyglandular syndrome).

3) **Nutritional deficit.** An electrolyte imbalance happens in the morning, when pregnant woman is hungry (low carbohydrate reserve develops and deficiency of vitamin B6, Vit B1 and proteins).

4) **Decreased gastric motility.** is cause nausea. Whatever may be the cause of initiation of vomiting, it is probably aggravated by the neurogenic element.

5) **Psychogenic** target is aggravating factor of early gestosis.
### The 24-hour Pregnancy-Unique Quantification of Emesis (PUQE-24) Scale

<table>
<thead>
<tr>
<th>Question</th>
<th>1 hour or less (2)</th>
<th>2-3 hours (3)</th>
<th>4-6 hours (4)</th>
<th>more 6 hours (5)</th>
</tr>
</thead>
<tbody>
<tr>
<td>In the last 24 hours, for how long have you felt nauseated or sick to your stomach?</td>
<td>Not at all (1)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>In the last 24 hours have you vomited or thrown up?</td>
<td>1–2 times (2)</td>
<td>3–4 times (3)</td>
<td>5–6 times (4)</td>
<td>7 or more times (5)</td>
</tr>
<tr>
<td>In the last 24 hours how many times have you had retching or dry heaves without bringing anything up?</td>
<td>No time (1)</td>
<td>1–2 times (2)</td>
<td>3–4 times (3)</td>
<td>5–6 times (4)</td>
</tr>
</tbody>
</table>

Mild - 6 points; Moderate - 7–12 points; Severe -13–15 points.
Management.

- **Mild forms of early gestosis** allowed outpatient treatment under conditions of dynamic control body weight and regular **examination of urine for acetone**.

- Patient is advised to drink fluids (2,5 l in daily). If necessary, medical treatment is provided, including antiemetic drugs - trifluoperazine (Espazine), droperidol, methoproclamid.
Hospitalization. Whenever a patient is diagnosed as a case of hyperemesis gravidarum, she is admitted.

Treatment Algorithm

- A. PUQE score ≤6 (mild NVP (nausea vomiting in pregnancy))
- Emergency Room or Outpatients Department
- Weight patient and record in chart
- Advise on dietary management and adequate fluids
- Provide diet information leaflet and discharge home
Hypermesis gravidarum

is a severe type of vomiting of pregnancy which has got deleterious effects on the health of the mother:

- dehydration
- electrolyte imbalance (hypokalemia)
- metabolic acidosis (from starvation) or alkalosis (from loss of hydrochloric acid)
- weight loss $\geq 5\%$
Treatment Algorithm
B. PUQE score 7-12 (moderate NVP)

- Admission to the Day Care Unit or appropriate Ward
- Investigations
- Obtain full medical history
- Urine dipstick for ketonuria
- Full blood count, urea and electrolytes for hypokalaemia and hyponatraemia, liver function test and thyroid function
- Send MSSU to the laboratory for culture and sensitivity
- Obtain baseline weight/ BMI
D. PUQE score ≥13 (severe NVP/hyperemesis or transfer or transfer from day care) hospitalization

- Correct hydration.
- Check each urine sample for ketones
- Insulin-dependent diabetics must be managed carefully.
- Vitamin supplementation
- Diabetics requiring glucose infusion must be given Thiamine deficiency followed by glucose infusion precipitates Wernicke’s encephalopathy.
- Record full physiological observation at least daily on the IMEWS chart (more frequent depending on general wellbeing)
- If the serum potassium level is found to be less than 3.2mmol/l, potassium supplements should be given.
- Refer to dietitian for assessment of nutritional needs
- Provide psychological support
- Administer antiemetics as prescribed.
- Apply antiembolic stockings for women who are bed ridden, risk assess as per hospital guideline for venous thromboembolism
- Daily control electrolyte balance
- Assess bowel function daily
Antiemetic medications

**I line** of antiemetic drugs –
- promethazine 25 mg or rochlorperazine 5 mg
- trifluopromazine 10 mg 2-3 times intramuscularly.
- trifluoperazine 1 mg twice daily intramuscularly
- Vitamin B6
- doxylamine.

**II line**
- **Metoclopramide** stimulates gastric and intestinal motility without stimulating the secretions. It is found useful.

**III line** **Hydrocortisone 100 mg IV** in the drip is given in a case with hypotension or in intractable vomiting. Oral method prednisolone is also used in severe cases.

**Intravenous infusion therapy**
*The violation of infusion must be not less in than 1-3 liters of liquid*
- protein agents,
- glucose with insulin solution,
- Ringer-Locke solution,
- Rheosorbilact,
- Xylatum 5% sodium hydrocarbonate solution.
Dermatoses gravidarum

- Dermatoses gravidarum is a group of diseases that occur in connection with pregnancy and disappear after its termination. Skin diseases in pregnancy depend on functional imbalance between the cortex and the subcortex, increased excitability of the vegetative nervous system, accompanied by disturbances of skin innervation, metabolic, hemomicrocirculatory changes in it. Dermatoses gravidarum are manifested in the form of skin itching, and rarely in the form of eczema, erythema, and herpetic rash. Treatment of dermatoses gravidarum is conducted similar to treatment of vomiting of pregnancy (the appropriate regimen, meal with limited content of protein and fats, medications that regulate the function of the nervous system and metabolism).
Hypersalivation (ptyalism).

- Hypersalivation is observed in vomiting, and sometimes is an independent manifestation of gestosis. The amount of **secreted saliva can reach 1,0 l per day**. Salivation does not affect the body, but suppresses the patients’ psyche, causing **maceration of skin and labial mucosa**.

- Treatment similar to vomiting is carried out in salivation. Sometimes, in order to reduce the secretion of the salivary glands intramuscular administration of **1,0 ml 0,1% atropine solution** is prescribed. Rinsing the mouth with the tincture of sage, chamomile and other agents with astringent properties will be also appropriate. No termination of pregnancy is required in this pathology.
HYPERTENSIVE DISORDERS ARE THE MOST COMMON MEDICAL COMPLICATIONS OF PREGNANCY,

- affecting 5% to 10% of all pregnancies.
- These disorders are responsible for approximately 16% of maternal mortality in developed countries.
Maternal Mortality and Hypertension

Developing Nations

- Hemorrhage: 20%
- Sepsis: 40%
- HTN: 15%
- Other: 25%

100-800 / 100,000 (deaths/birth)

Developed Nations

- Hemorrhage: 13%
- Embolism: 20%
- Sepsis: 8%
- Abortion: 17%
- HTN: 17%
- Other: 25%

12 / 100,000 (deaths/birth)
Hypertension is defined as

- a systolic blood pressure (SBP) of 140 mm Hg or greater
- or
- a diastolic blood pressure (DBP) of 90 mm Hg or greater.

Pressure should be taken
- after a 10-minute or longer rest period.
- in the left lateral recumbent position with the patient's arm at the level of the heart.
## Monitoring of Blood Pressure

- **For early diagnosis of preeclampsia**

<table>
<thead>
<tr>
<th></th>
<th>Blood pressure</th>
<th>Proteinuria</th>
</tr>
</thead>
<tbody>
<tr>
<td>Till 20 weeks</td>
<td>1 time in 3 weeks</td>
<td></td>
</tr>
<tr>
<td>20 – 28 weeks</td>
<td>1 time in 2 weeks</td>
<td></td>
</tr>
<tr>
<td>After 28 weeks</td>
<td>Every week</td>
<td></td>
</tr>
</tbody>
</table>

- Consultation of ophthalmologist, ECG, biochemical analysis of blood, coagulogramma, USG of fetus
CLASSIFICATION OF HYPERTENSIVE DISORDERS

**Chronic hypertension**
Hypertension diagnosed prior to pregnancy, prior to 20 weeks gestation, or after 12 weeks postpartum

**Gestational hypertension**
Hypertension developing after 20 weeks gestation without proteinuria or other signs of preeclampsia

- **Transitional GH** - BP normalized till 12 weeks postpartum
- **Chronic GH** - high BP have place after 12 weeks postpartum

**Preeclampsia or eclampsia**
Hypertension typically developing after 20 weeks gestation with proteinuria;
eclampsia is attack of convulsion in women with preeclampsia
<table>
<thead>
<tr>
<th>Classification</th>
<th>SBP</th>
<th>DBP</th>
<th>Proteinuria</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Mild</strong></td>
<td>&gt;140 mm Hg</td>
<td>&gt;90 mm Hg</td>
<td>&lt; 0.3 g in a 24-hour urine collection or &lt; 0.3 g/L on two random sample urine dipsticks at least 6 hours apart</td>
</tr>
<tr>
<td><strong>Middle</strong></td>
<td>150-159</td>
<td>100-109</td>
<td>0.3 – 5 g in a 24-hour urine collection</td>
</tr>
<tr>
<td><strong>Severe</strong></td>
<td>≥ 160</td>
<td>≥ 110</td>
<td>≥ 5 g in a 24-hour urine collection</td>
</tr>
<tr>
<td><strong>Oliguria</strong></td>
<td></td>
<td></td>
<td>Oliguria &lt;500 ml in 24 hours</td>
</tr>
<tr>
<td><strong>Thrombocytopenia</strong></td>
<td></td>
<td></td>
<td>Thrombocytopenia &lt;100,000/mm³</td>
</tr>
<tr>
<td><strong>Elevated liver function test</strong></td>
<td></td>
<td></td>
<td>Elevated liver function test</td>
</tr>
<tr>
<td><strong>Persistent epigastric or right upper quadrant pain</strong></td>
<td></td>
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</tr>
<tr>
<td><strong>General and pulmonary edema</strong></td>
<td></td>
<td></td>
<td>General and pulmonary edema</td>
</tr>
<tr>
<td><strong>Persistent, severe cerebral or visual disturbances</strong></td>
<td></td>
<td></td>
<td>Persistent, severe cerebral or visual disturbances</td>
</tr>
</tbody>
</table>

**Preeclampsia superimposed**

The development of preeclampsia or eclampsia in a woman with preexisting or chronic hypertension.
Abnormal proteinuria in pregnancy is defined as the excretion of 0.3 g or more of protein in 24 hours or 0.3 g/L in middle portion of urine.
**Edema** is a common finding in the gravid patient, occurring in approximately 50% of women. Lower extremity edema is the most typical form. Pathologic edema is seen in nondependent regions such as the face, hands, or lungs.
New understanding of traditional **pre-eclampsia**

- **triad:**
  - (1) Hypertension arteriolar constriction (endothelial dysfunction).
  - (2) Proteinuria leaky glomerulus (capillary) (endothelial dysfunction).
  - (3) Edema leaky capillaries in skin, muscle, liver, brain, airway, nose. (endothelial dysfunction).

**4th component of endothelial dysfunction in pre-eclampsia**

(1) Muscular artery spasm
(2) Increased “augmentation index” (Alx)
(3) Increased Alx
(4) Modern concept of pre-eclampsia: symptoms are due to arterial, arteriolar and capillary endothelial damage
**Normal pregnancy**

- Vessel remodeling
- Blood flow increases
- Normoxia
- Normal organ function
  - No hypertension
  - Normal glomerular function
  - No proteinuria
  - No brain edema
  - No liver edema
  - No coagulation abnormalities
- sFlt1

**Preeclampsia**

- No vessel remodeling
- Blood flow decreases
- Hypoxia
- sFlt1
- Free VEGF and PIGF
- Endothelial dysfunction
- Multi-organ disease
  - Hypertension
  - Glomerular dysfunction
  - Proteinuria
  - Brain edema
  - Liver edema
  - Coagulation abnormalities
Complications.

- Neurologic complications:
  - Encephalopathy due to thiamine deficiency
  - Pontine myelinolysis
  - Peripheral neuritis
  - Psychosis
  - Convulsions
  - Coma
  - Gastrointestinal problems
  - Stress ulcer in stomach
  - Esophageal tear (Mallory-Weiss syndrome)
  - Jaundice
  - Renal failure.
Morphology

1. The placenta reveals various microscopic changes, most of which reflect malperfusion, ischemia, and vascular injury.

2. Placental infarcts—small, peripheral ones that may occur in normal full-term placentas—are larger and more numerous in preeclampsia. Ischemic changes in the chorionic villi and trophoblast. Increased syncytial knots and the appearance of accelerated villous maturity. There is increased frequency of retroplacental hematomas due to bleeding and instability of uteroplacental vessels.

3. Finding is in the decidual vessels, reflecting abnormal implantation. This can be in the form of thrombosis fibrinoid necrosis intraintimal lipid deposition (acute atherosis)
The liver lesions, when present, take the form of
1. irregular,
2. focal,
3. subcapsular,
4. intraparenchymal hemorrhages

On histologic examination there are fibrin thrombi in the portal capillaries and foci of hemorrhagic necrosis. 1. The kidney lesions are variable.
Pathophysiology of Preeclampsia

**Cause: Unknown**

Characterized by:

- **Vasospasm**
- **Activation of coagulation system**
- **Perturbations in humoral and autacoid systems related to volume and BP control**
- **Oxidative stress and inflammatory-like responses**
- **Pathologic changes ischemic in nature**
Pathophysiology: Placenta

- • Normal pregnancy:
  - Spiral arterioles invaded by endovascular trophoblast
  - diameter, flaccid,
  - sac-like vessels

- • Preeclampsia:
  - Invasion incomplete
  - Failure to re-model: thick walled, muscular arterioles
  - Acute atherosis in basal arteries: necrosis, foam cells
  - Decreased perfusion, early placental hypoxia, infarction
• **Pathophysiology: Renal**

- Glomerular capillary endotheliosis
- • GFR and renal blood flow
- • Proteinuria: nonselective, late in clinical course
- • Hyperuricemia (marker for preeclampsia)
- • Hypocalciuria, altered Ca+2 regulatory hormones
- • Impaired NA+ excretion, suppression of renin-angiotensin system: fluid retention, edema
- • Plasma volume, hemoconcentration
Pathophysiology: Coagulation System

- Activation coagulation system
  - Procoagulants
    - Thrombocytopenia
      - Most common hematologic abnormality
      - Platelets < 100,000 cells/mm³: serious disease
      - Fetal platelet count unaffected
  - Fibrinogen
  - Antithrombin III
  - FDP
  - Microthrombi
Pathophysiology: Cardiac

- Normal: CO, HR, AC
- TPR, BP, nl contractility
- • Preeclampsia:
  - CO, TPR, nl load-independent contractility (Wallenburg et al, Lan)
  - CO, TPR; “cross over” later (Easterling et al, Boslo et al)
- • HELLP: subendocardial hemorrhages
- • Cardiac decompensation: preexisting heart disease
Preeclampsia: Hemodynamic Changes

- extracellular fluid edema
  - Endothelial damage, capillary leakage
  - plasma colloid oncotic pressure
  - interstitial colloid osmotic pressure
- Pulmonary edema
  - left-sided filling pressures
  - CO
- capillary permeability
  - Associated with excessive crystalloid,
  - colloid, b-methasone
- More common: older, CHTN, obesity
Pathophysiology: Hepatic

- Hemorrhagic lesions
  - Infarction
  - ALT, AST, LDH
  - HELLP syndrome: hemolysis, LFT’s, platelets
    - Markedly ALT, AST, LDH
    - Subcapsular bleeding
    - Hepatic rupture
Pathophysiology: CNS

- Headache
  - Visual disturbances
    - Blurred vision
    - Scotomata
    - Cortical blindness
  - Convulsions: cerebral vasospasm
  - CT or MRI: normal vs transient
  - Abnormalities: cerebral edema, hemorrhage, global ischemia induced by vasospasm
  - Pathology: Hemorrhages, petechiae, vasculopathy with vessel wall damage, fibrinoid necrosis, ischemic damage and microinfarcts
MATERNAL AND FETAL COMPLICATIONS IN SEVERE PREECLAMPSIA

Maternal

- Abruptio placentae
- Disseminated coagulopathy
- HELLP syndrome
- Pulmonary edema/aspiration
- Acute renal failure
- Eclampsia
- Liver failure or hemorrhage
- Stroke
- Death
- Long-term cardiovascular morbidity

Fetal

- Preterm delivery
- Fetal growth restriction
- Hypoxia
- Perinatal death
- Long-term cardiovascular morbidity associated with low birth weight (fetal origin of adult disease)
MILD PREECLAMPSIA

- Till 37 week gestation –

- Investigation:
  - baseline laboratory evaluation, including a 24-hour urine collection for protein, hematocrit, platelet count, coagular blood analyses, serum creatinine value, electrolytes, ALT and AST level.
  - ultrasonography should be performed to evaluate amniotic fluid volume and estimated fetal weight and to confirm gestational age

- Medicine do not prescribe

- Waiting and delivery per vias naturals

- Indications for hospitalization:
  - Term of gestation after 37 weeks
  - Appearance 1 or more sign meddle preeclampsia
  - Disorders of fetus
- Hospitalisation in room of intensive therapy and measurement of BP, diures, Ps every hour
- Investigation in moment of hospitalisation
  - GBA, hematocrit, platelet count, coagular blood analyses, serum creatinine value, electrolytes, ALT and AST level, GUA, 24-hour urine collection for protein, ECG, consultation of terapeutist, neurologyst, oftalmologyst
- Maternal management:
  - Measurement of BP every 6 hour in first 24-hour, after then – 2 in day
  - Daily weight
  - Urine analyses and 24-hour protein daily
  - Lab tests (liver function tests, hematocrit, platelet count, createnyn, urine acid, proteinogram) twice per week
- Fetal management:
  - Daily fetal movement
  - Auscultation every 8 hour
  - Nonstress test twice per week or biophysical profile once per week
Till 37 week gestation

- Bed-rest
- Nutrition: high-protein diet without limitation of salt
- Hypotensive medicine when DBP > 100 mm Hg
  - Metyldopa 0,25-0,5 g 3-4 time in day
  - Nifedipine 10 mg 2-3 time in day
- Prevention of fetal RDS (28-34 week gestation)
  - Dexametazon (β-metazone) - 6 mg every 12 hour 4 time

- If the treatment is don’t effective and
- appearance 1 or more sing severe preeclampsia
- or sign of fetal distress
After 37 weeks

preparation to labor (prostaglandyns)

If in labor appearance 1 or more sing severe preeclampsia

MgSO$_4$ for eclamptic seizure prophylaxis

Cesarean section
**SEVERE PREECLAMPSIA**

- **Hospitalization** in room of intensive therapy with monitoring of BP, diureses, Ps

- **Investigation in moment of hospitalisation**
  - GBA, hematocrit, platelet count, coagular blood analyses, serum creatinine value, electrolytes, ALT and AST level, GUA, 24-hour urine collection for protein, ECG, consultation of therapeutist, neurologyst, ophtalmologyst

- **Catheterization of peripheral veins for infusion** (see late)
  - Recover blood volume
  - MgSO₄ for eclamptic seizure prophylaxis
  - Hypotensive medicine for prevention cephalic bloodstroke

- **Delivery in 24-hour after diagnosis of severe form of preeclampsia**

- **Maternal management (by obstetrics and anesthesiologist):**
  - measurement of BP every hour
  - Auscultation every 8 hour
  - Urine analyses every 4 hour
  - Catheterization of urine bladder and control diureses every hour
  - Lab tests (liver function tests, hematocrit, platelet count, createnyn, urine acid, proteinogram) every day
  - Continuous fetal monitoring for the first 48 hours, then daily biphasic profile. Ultrasound assessment; include amniotic fluid measurement with umbilical artery Dopplers
Recover blood volume

- NaCl 0.09%, sol Ringer, and refortan (stabizol) 2:1
- Volume – 2.5-3.0 L/d (35 ml/kg)
- Speed – no more 85 ml/h
- Not to be used sol. of albumin and glucose (hypoglycemia in fetus)
- Fresh frozen plasma (for prevention of coagulopathy bleeding)

### Hypotensive medicine

<table>
<thead>
<tr>
<th>Drug</th>
<th>Starting Dosage</th>
<th>Maximum Dosage</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute treatment of severe hypertension (till 150/90 – 160/100)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hydralazine</td>
<td>5 mg i.v. every 10 min till DBP=90-100</td>
<td>30 mg(^a)</td>
<td></td>
</tr>
<tr>
<td>Labetalol</td>
<td>10-20-40-80 mg- i.v. every 10-15 min</td>
<td>220 mg(^a)</td>
<td>Avoid in women with asthma or congestive heart failure</td>
</tr>
<tr>
<td>Nifedipine</td>
<td>5-10 mg p.o. every 30 min</td>
<td>50 mg(^a)</td>
<td></td>
</tr>
<tr>
<td>Long-term treatment of hypertension</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Methyldopa</td>
<td>250 mg *3-4 i/d</td>
<td>4 (2)g/d</td>
<td>Rarely indicated</td>
</tr>
<tr>
<td>Labetalol</td>
<td>100 mg *2-3 i/d</td>
<td>2,400 mg/d</td>
<td>First choice</td>
</tr>
<tr>
<td>Atenolol</td>
<td>50 mg *1 i/d</td>
<td>100 mg/d</td>
<td>Associated with IUGR</td>
</tr>
<tr>
<td>Nifedipine</td>
<td>10-20 mg *3-4 i/d</td>
<td>100 mg/d</td>
<td>To be used in women with diabetes</td>
</tr>
<tr>
<td>Nifedipine-long</td>
<td>20-40 mg *2 i/d</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Hypertensive therapy and infusion to be use with magnesium-therapy
Treatment of hypertension

- 
- *Nifedipine* 10–20 mg conventional release tablet Oral Onset: 30–45 minutes
- Repeat: after 45 minutes
- Maximum: 80 mg
- *Labetalol* Initially 20 mg
  - Repeat with 40–80 mg IV bolus over 2 minutes Onset: 5 minutes
  - Repeat: every 10 minutes
  - 20–160 mg/hour Infusion Titrate to BP response to a maximum of 300 mg
  
- *Hydralazine* 5–10 mg
  - (5 mg if fetal compromise) IV bolus over 3–10 minutes Onset: 20 minutes
  - Repeat: every 20 minutes
  - Maximum: 30 mg
  - 10–20 mg/hour Infusion Titrate to BP response
  - Refer to Appendix C: Hydralazine protocol
- *Diazoxide* 15–45 mg IV rapid bolus Onset: 3–5 minutes
- Repeat: after 5 minutes
- Maximum 150 mg/dose
- Monitor Blood Glucose Levels
**MgSO\textsubscript{4} for eclamptic seizure prophylaxis**

- **Start-Dosage** –
  - 4 mg (16 ml 25% sol. MgSO\textsubscript{4}) + 34 ml NaCl 0,09% i.v very slowly during 15 min (eclampsia – 5 min)

- **Support-Dosage** –
  - 7.5 mg (30 ml 25% sol. MgSO\textsubscript{4}) + 220 ml NaCl 0,09% i.v with speed 1-3 g/h (10-30 drop/h) with infusion therapy at the same time.

- **Sing of toxicity of MgSO\textsubscript{4}:** ↓ frequency of respiration, hypoactivity of knee reflex, AV – block
  - *stopped MgSO\textsubscript{4} + 10 ml 10\% Ca-gluconatis i/v*

- **Monitoring during magnesium-therapy:**
  - measurement of BP every 20 min
  - frequency of respiration (no less 14 /min)
  - Pulse and cardiomonitoring, ECG
  - Evaluation of knee reflex every 2 hour
  - control diureses every hour
  - (no less 50 ml/h)

*Sing of stopping of magnesium-therapy:*
- Stopping of convulsion
- Lack of sing of hyperactivity CNS
- Normalization DBP and diureses

MgSO\textsubscript{4} infusion should continue for 24 hours postpartum
Severe Preeclampsia. Delivery in 24-Hour

+ Prevention of fetal RDS (28-34 week gestation)
  - Dexametazon (β-metazone) - 6 mg every 12 hour 4 time

× if coli uteri is prepared for labor - amhiotomia and delivery per vias naturalis with effective anastesia (epidural)
× is not prepared for labor
× Prostaglandins prevention is not effective
× Progress of hypertension
× threat of convulsion
× Distress of fetus
★ Distressing of women and (or) fetus in II period of labor – vacuum extraction or forceps
★ In III period of labor - risk of bleeding – prevention by okcytocini not be use metylergometrin

× Intrapartum management
  × close blood pressure control, and intravenous MgSO₄ administration
Eclampsia

**Goals of treatment**
- Terminate the seizure
- Prevent recurrence
- Control hypertension
- Prevent maternal and fetal hypoxia

**Context**
- There are no reliable clinical markers that predict eclampsia
- Hypertension and proteinuria may be absent prior to the seizure
- Seizures may occur antepartum, intrapartum or postpartum usually within 24 hours of birth
- Reported incidence of eclampsia varies.
Imminent eclampsia

- Defined in the Magpie trial as at least two of the following symptoms
  - Frontal headache
  - Visual disturbance
  - Altered level of consciousness
  - Hyperreflexia
  - Epigastric tenderness
Treatment

• Magnesium Sulfate is the anticonvulsant drug of choice for the prevention and treatment of eclampsia

  o Diazepam 5–10 mg IV at a rate of 2–5 mg/minute (maximum dose of 10 mg) or
  o Midazalam 5–10 mg IV over 2–5 minutes or IM
  o Clonazepam 1–2 mg IV over 2–5 minutes

• Do not use Phenytoin for eclampsia prophylaxis or treatment unless there is a contraindication to Magnesium Sulfate or it is ineffective

• Aim for BP below 160/100 mmHg
Post seizure care

• If birth has not occurred, plan as soon as feasible and when the woman’s condition is stable
• Close clinical surveillance is required in an appropriately staffed area
HELLP Syndrome

- A variant of severe preeclampsia (Haemolysis, Elevated Liver enzymes and Low Platelet count). Elements include:
  - Thrombocytopenia (common)
  - Haemolysis (rare) and
  - Elevated liver enzymes (common)
  - Maternal platelet count of less than 100 x 109/L
  - Elevated transaminases
  - Microangiopathic haemolytic anaemia with fragments/schistocytes on blood film
Diagnostic criteria

- Liver transaminases (AST, ALT) should be elevated >70 IU/L,
- Total serum lactate dehydrogenase (LDH) should be 600 IU/L or more,
- The platelet count should be <100 x 10⁹/L (<100,000/microlitre).
- Haemolysis may be indicated by elevated total bilirubin (>1.2 mg/dL [>20.5 micromol/L]),
- LDH and AST elevations, and characteristic findings (schistocytes) on a peripheral blood smear, haematuria, worsening anaemia, and a low serum haptoglobin.
Management

- Liaise with consultant obstetrician, obstetric physician, physician haematologist or anaesthetist
  - Contact other facilities/services if necessary
  - If greater than 34 weeks gestation and/or condition deteriorating, plan birth
  - Magnesium Sulfate infusion may be indicated
  - Consider platelet transfusion if:
    - Thrombocytopenia presents a hazard to operative birth or
    - There is significant bleeding postpartum attributable to preeclamptic