Consensus Guideline:

HYPERTENSION DURING PREGNANCY

CG012

Ministry of Public Health
General Directorate of Curative Medicine
Clinical Guideline Development Department

Kabul, Afghanistan

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Forward

Each year around the world a third of a million women die as a result of pregnancy and childbirth. Over 99% of these deaths occur in developing countries. Recent UN estimates suggest that progress is being made in reducing these deaths but the picture is very mixed, with some countries and regions having made impressive gains while others show a much more negative pattern of limited improvement and stagnation. Progress tends to be poorest in fragile states such as Afghanistan, which now has the highest maternal mortality ratio in the world. Reducing maternal deaths within a fragile or post-conflict environment where infrastructure is weak is extremely challenging, and specific dimensions of the Afghan context including insecurity, social and cultural factors and geographical barriers impose particular constraints. The main leading causes of maternal morbidity and mortality in Afghanistan are postpartum hemorrhage, hypertension during pregnancy (pre-eclampsia & eclampsia) and septicemia.

Hypertensive disorders during pregnancy occur in women with pre-existing primary or secondary chronic hypertension, and in women who develop new-onset hypertension in the second half of pregnancy.

Hypertensive disorders during pregnancy carry risks for the woman and the baby. Although the rate of eclampsia in Afghanistan appears to have fallen according to AMS 2010 but hypertension in pregnancy remains one of the leading causes of maternal death in Afghanistan. Hypertensive disorders also carry a risk for the baby too.

General directorate of curative medicine by technical and financial support of SEHAT/ Hospital thematic area remunerated two expertise of clinical guidelines and protocols development department and cohort of expertise from central hospitals to develop clinical guideline of diagnosis and management of hypertension during pregnancy for easy use of general practitioners, trainees and trainer to have unique approach on diagnosis and management of Hypertension during pregnancy.

This guideline covers diagnosing and managing hypertension (high blood pressure), including pre-eclampsia, during pregnancy, labour and birth. It also includes advice for women with hypertension who wish to conceive and women who have had a pregnancy complicated by hypertension. It aims to improve care during pregnancy, labour and birth for women and their babies.

Finally, I greatly appreciate and thank everyone involved for their active participation, collaboration, and valuable inputs. I would like to particularly acknowledge the strong leadership of the General Directorate of Curative Medicine, Clinical Guidelines & Protocols Development Department in directing the Gynecology and Obstetrics core group members.

Dr Feda Mohammad Paktan MD, MPH
Deputy Minister for Health Care Service Provision
**ABBREVIATIONS:**

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
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<tbody>
<tr>
<td>ACE</td>
<td>Angiotension converting enzyme inhibitors</td>
</tr>
<tr>
<td>ACR</td>
<td>Albumin: Creatinine Ratio</td>
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<tr>
<td>AFI</td>
<td>Amniotic Fluid Index</td>
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<tr>
<td>ALT</td>
<td>Alanine Aminotransferase</td>
</tr>
<tr>
<td>aPTT</td>
<td>activated partial thromboplastin</td>
</tr>
<tr>
<td>ARB</td>
<td>angiotensin receptor blockers</td>
</tr>
<tr>
<td>AST</td>
<td>Aspartate Aminotransferase</td>
</tr>
<tr>
<td>ATN</td>
<td>acute tubular necrosis</td>
</tr>
<tr>
<td>BMI</td>
<td>Body mass index</td>
</tr>
<tr>
<td>BP</td>
<td>Blood pressure</td>
</tr>
<tr>
<td>CBC</td>
<td>complete blood cell</td>
</tr>
<tr>
<td>CrCl</td>
<td>Creatinine clearance</td>
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<tr>
<td>CT</td>
<td>Computed tomography</td>
</tr>
<tr>
<td>CTG</td>
<td>cardiotocography</td>
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<tr>
<td>DIC</td>
<td>disseminated intravascular coagulation</td>
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<tr>
<td>DM</td>
<td>Diabetes mellitus</td>
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<tr>
<td>ECG</td>
<td>electrocardiogram</td>
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<td>EEG</td>
<td>electroencephalogram</td>
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<tr>
<td>e.g.,</td>
<td>exempli gratia [Latin: for example]</td>
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<tr>
<td>EmONC</td>
<td>Emergency Obstetric Newborn Care</td>
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<tr>
<td>GFR</td>
<td>glomerular filtration rate</td>
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<td>HDP</td>
<td>hypertensive disorders in pregnancy</td>
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<tr>
<td>HELLP Syndrome</td>
<td>Haemolysis, Elevated Liver enzymes and Low Platelet count Syndrome</td>
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<td>HTN</td>
<td>Hypertension</td>
</tr>
<tr>
<td>IUGR</td>
<td>intrauterine growth restriction</td>
</tr>
<tr>
<td>KEMH</td>
<td>King Edward Memorial Hospital</td>
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<tr>
<td>LDA</td>
<td>Low-dose Aspirin</td>
</tr>
<tr>
<td>LDH</td>
<td>Lactate Dehydrogenase</td>
</tr>
<tr>
<td>LDH cholesterol</td>
<td>Lactate Dehydrogenase or &quot;good&quot; cholesterol</td>
</tr>
<tr>
<td>LFT</td>
<td>Liver Function Tests</td>
</tr>
<tr>
<td>M&amp;E</td>
<td>Monitoring and Evaluation</td>
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<tr>
<td>μL</td>
<td>microliter</td>
</tr>
<tr>
<td>MgSO4</td>
<td>Magnesium Sulphat</td>
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<tr>
<td>Mmol</td>
<td>Millimeter Mol</td>
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<tr>
<td>MNH</td>
<td>Maternal Newborn Health</td>
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<tr>
<td>MRI</td>
<td>magnetic resonance imaging</td>
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<tr>
<td>NICU</td>
<td>Non Intensive Care Unit</td>
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<td>NST</td>
<td>non-stress tests</td>
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<tr>
<td>Abbr</td>
<td>Term</td>
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<tr>
<td>PEE</td>
<td>Pre-eclampsia</td>
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<tr>
<td>PIH</td>
<td>Pregnancy Induced Hypertension</td>
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<tr>
<td>PPH</td>
<td>Postpartum Hemorrhage</td>
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<tr>
<td>PRES</td>
<td>posterior reversible encephalopathy</td>
</tr>
<tr>
<td>PT</td>
<td>prothrombin Time</td>
</tr>
<tr>
<td>RUQ</td>
<td>Right Upper Quadrant</td>
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<tr>
<td>SGA</td>
<td>Small-for-gestational-age</td>
</tr>
<tr>
<td>SGOT</td>
<td>serum glutamic oxaloacetic transaminase</td>
</tr>
<tr>
<td>SGPT</td>
<td>serum glutamic pyruvic transaminase</td>
</tr>
<tr>
<td>WHO</td>
<td>World Health Organization</td>
</tr>
</tbody>
</table>
Table of Contents

WORKGROUP MEMBERS ........................................................................................................ II

ABBREVIATIONS: .................................................................................................................. IV

Epidemiology: ........................................................................................................................ 1

CHRONIC HYPERTENSION OF ALL CAUSES.................................................................... 2

- Essential (Primary) Hypertension ...................................................................................... 5
- Secondary Hypertension ..................................................................................................... 5
- White Coat Hypertension .................................................................................................. 6
- Transient Hypertensive Effect ............................................................................................ 6

Elevated blood pressure may be due to environmental stimuli (e.g.; the pain of labour). A transient hypertensive effect is not associated with an increased risk of adverse outcomes .......................................................................................................................... 6

Gestational Hypertension .................................................................................................... 6

Pre-eclampsia .......................................................................................................................... 6

A. Causes of pre-eclampsia .................................................................................................... 8
B. Risk Factors for Pre Eclampsia .......................................................................................... 8

Table 1. Diagnostic Criteria for Preeclampsia ...................................................................... 9

C. Symptoms of pre-eclampsia .............................................................................................. 9
D. HELLP syndrome ............................................................................................................. 10

SUPERIMPOSED PREECLAMPSIA ....................................................................................... 11

ECLAMPSIA ............................................................................................................................ 11

DIAGNOSTIC OVERVIEW ..................................................................................................... 12

1. Measurement of Blood Pressure .................................................................................... 13
2. Diagnosis of Hypertension .............................................................................................. 13

A. Routine Tests ..................................................................................................................... 13
B. Hematologic Evaluation .................................................................................................... 14
C. Renal and Hepatic Evaluation .......................................................................................... 14

1. Chronic hypertensive ....................................................................................................... 20
2. Gestational Hypertension ................................................................................................ 20
3. Management of preeclampsia and HELLP Syndrome ..................................................... 22

i. Maternal assessment ......................................................................................................... 25
ii. Fetal assessment ................................................................................................................ 25

Initial Evaluation of Women with superimposed Preeclampsia .............................................. 25

Indications for Delivery during Expectant Management ......................................................... 26

1. Maternal indications for delivery ..................................................................................... 26
2. Fetal indications for delivery ............................................................................................. 27
3. Elective cesarean delivery: ............................................................................................... 27

Table 2. Antihypertensive Drugs Recommended in Pregnancy ............................................. 29

Table 3. Flowchart for Hypertension in Pregnancy Management ......................................... 30

POSTNATAL MANAGEMENT ................................................................................................ 31

VI
• CARE IN THE FIRST 6 WEEKS POSTPARTUM ................................................................. 31
• CARE BEYOND 6 WEEKS POSTPARTUM ........................................................................ 32

Table 4. Flowchart for Postnatal Hypertension Management .................................................. 33
• LONG-TERM CONSEQUENCES ......................................................................................... 34

1. Resuscitation .................................................................................................................. 34
2. Prevention of further seizures ........................................................................................ 34
3. Control of hypertension ................................................................................................. 34
4. Delivery .......................................................................................................................... 35

i. ADMINISTRATION OF MAGNESIUM SULPHATE .......................................................... 35
ii. BEFORE DISCONTINUATION OF MgSO4 THERAPY: .................................................. 36

Antenatal management ....................................................................................................... 37
Annex 1: .............................................................................................................................. 38
Annex 2: .............................................................................................................................. 39
Annex 3: .............................................................................................................................. 40
Annex 4: .............................................................................................................................. 41
Annex 5: .............................................................................................................................. 42
Annex 6: .............................................................................................................................. 43
Aim/ Purpose of this Guideline

The purpose of this guideline is to improve the management of hypertension in pregnancy. These guidelines are intended for healthcare professionals, particularly those in training who are working in HSE-funded obstetric and gynecological services. They are designed to guide clinical judgment but not replace it. In individual cases a healthcare professional may, after careful consideration, decide not to follow a guideline if it is deemed to be in the best interests of the woman.

Background and Introduction

Definition

Hypertension is defined as the presence of a blood pressure (BP) elevation to a level that places patients at increased risk for target organ damage in several vascular beds, including the retina, brain, heart, kidneys, and large conduit arteries.

Hypertensive disorders of pregnancy remain a leading cause of maternal and neonatal morbidity and mortality. This guideline summarizes the existing evidence and provides a reasonable approach to the diagnosis, evaluation, and treatment of the hypertension in pregnancy.

Hypertensive disorders during pregnancy carry risks for the woman and the baby. Hypertension in pregnancy remains one of the leading causes of maternal death in the UK and Ireland, Europe and elsewhere. Detailed enquiries have examined standards of care, and substandard care (where different management might have been expected to prevent death) has been identified in the majority of cases. These failures of care occur throughout pregnancy and not just in the critical care environment.

Hypertensive disorders during pregnancy may result in substantial short-term maternal morbidity. More recently, the long-term consequences for women with a diagnosis of hypertension during pregnancy have become clear, in particular chronic hypertension and an increase in lifetime cardiovascular risk.

Hypertensive disorders also carry a risk for the baby. About 1 in 20 (5%) stillbirths in infants without congenital abnormality occur in women with pre-eclampsia. The contribution of pre-eclampsia to the overall preterm birth rate

1 Abalos E et al., 2014; Khan KS et al., 2006
2 Schutte JM et al., 2008; Knight M et al., 2014
3 Bellamy L et al., 2007; Smith GCS et al., 2001
4 Simpson LL., 2002.
is substantial: 8–10% of all preterm births result from hypertensive disorders. Small-for-gestational-age (SGA) babies (mainly because of intrauterine growth restriction (IUGR) arising from placental disease) are common, with 20–25% of preterm births and 14–19% of term births in women with pre-eclampsia being less than the tenth centile of birth weight for gestation.

There is national guidance on the care of women with severe pre-eclampsia or eclampsia. This clinical guideline contains recommendations for the diagnosis and CLINICAL PRACTICE GUIDELINE The Management of Hypertension in Pregnancy

Epidemiology:

Hypertensive disorders during pregnancy occur in women with pre-existing primary or secondary chronic hypertension, and in women who develop new-onset hypertension in the second half of pregnancy. Hypertensive disorders during pregnancy carry risks for the woman and the baby. Although the rate of preeclampsia and eclampsia in the developed countries appears to have fallen, but still hypertension in pregnancy remains one of the leading causes of maternal death in both developed and developing countries. And hypertensive disorders during pregnancy made also result in substantial maternal morbidity. But fortunately hypertensive disorders are preventable and avoidable disorders in health management.

Globally and in Afghanistan there has been significant progress in reducing maternal mortality, however, looking ahead to the ambitious post-2015 development agenda and ultimate goal of ending all preventable maternal deaths much work still needs to be done. Over 70% of maternal deaths occur as a result of complications of pregnancy and childbirth such as hemorrhage and hypertensive disorders; the time of childbirth and the period immediately after birth are particularly critical for maternal, fetal and neonatal survival and well-being. Therefore effective care to prevent and manage complications during this critical period is likely to have a significant impact on reducing maternal deaths, stillbirths and early neonatal deaths—a triple return on investment. Within this critical period, quality of care improvement efforts need to target essential maternal and newborn care and additional care for management of complications that can achieve the highest impact on maternal survival.

5 Pre-eclampsia and Eclampsia Clinical Practice Guideline no. 3, 2011
6 (Global Strategy for Women’s, Children’s and Adolescents’ Health 2015-2030).
The MoPH has stewardship role for service delivery approaches and specific interventions as outlined in below:

- Ensure BP checked in every antenatal care visit
- Every skilled birth attendant can detect, prevent and manage PEE.
- Every health facility where births take place will have adequate supplies, equipment and life-saving medicines for the prevention and treatment of PEE – such as; anticonvulsants (MgSO4), antihypertensive, Lignocaine 2% and Calcium Gluconate 10%.
- Testing implementation platform for calcium supplementation in pregnancy to inform scaling up
- Midwives are supported to perform according to their agreed scope of practice and administer life-saving drugs
- Community based MNH services focus on improving awareness of danger signs; complication readiness plans and mobilization for care seeking and referral
- Access to quality EmONC is improved including an effective referral system
- Relevant guidelines are updated in line with recent WHO recommendations, disseminated and operationalized.

In relation to PEE and PPH the professional associations can be mobilized to:

- Advocate for increasing access to EmONC including the availability of supplies and lifesaving medicines; of note Magnesium Sulphate, antihypertensive and utero tonics.
- Call upon national regulatory agencies and policy makers to approve misoprostol for PPH prevention and treatment and to ensure that current best-evidence regimens are adopted.
- Support quality improvement efforts of note strengthen compliance with recommended WHO guidelines
- Advocate for more focused M&E including tracking the correct indicators on PPH and PEE.

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7 National Symposium on Prevention and Management of Post-partum Hemorrhage (PPH), Pre-Eclampsia and Eclampsia (PEE), September 16th 2016
**Classification of Hypertension:**

It is imperative that every effort is made to accurately classify women with hypertension in pregnancy. The classification is as follows:

- Chronic hypertension of all causes
- Gestational (non-proteinuric) hypertension
- Pre-eclampsia
- Superimposed pre-eclampsia
- Eclampsia

This classification of the hypertensive disorders in pregnancy (HDP) reflects the pathophysiology of the constituent conditions as well as the risks and potential outcomes for both mother and baby. This clinical classification (or very similar) has been adopted by numerous national and international bodies.

**Pathophysiology:**

The pathophysiology of preeclampsia likely involves both maternal and fetal/placental factors. Abnormalities in the development of placental vasculature early in pregnancy may result in relative placental under perfusion/hypoxia/ischemia, which then leads to release of anti-angiogenic factors into the maternal circulation that alter maternal systemic endothelial function and cause hypertension and other manifestations of the disease. However, the molecular basis for abnormal placental development and placental dysregulation of these pathogenic factors remains unknown. The role of angiogenic proteins in early placental vascular development are under investigation.

**CHRONIC HYPERTENSION OF ALL CAUSES**

Chronic or pre-existing pregnancy hypertension predates the pregnancy or appears before 20 weeks’ gestation. A substantial number of pregnancies (0.2–5%) are complicated by pre-existing hypertension and the prevalence in western societies is likely to increase due to the advancing age of the prospective mother at conception and the rising tide of obesity. Approximately 90-95% of cases of chronic hypertension are considered to be essential in origin. Adverse outcomes of pregnancy are more common in women with pre-existing hypertension, regardless of the cause, but women with secondary hypertension and co-morbid conditions such as renal disease are at significantly increased risk of poor pregnancy outcome and require multidisciplinary care.
• **Essential (Primary) Hypertension**
  Defined by a blood pressure greater than or equal to 140 mmHg systolic and/or 90mmHg diastolic confirmed before pregnancy or before 20 completed weeks’ gestation without a known cause. The diagnosis can be difficult in women whose blood pressure before pregnancy or early in the first trimester is unknown as the physiological fall in blood pressure in the second trimester can obscure pre-existing hypertension. A diagnosis of essential hypertension can only be made after a thorough evaluation has eliminated secondary causes.

• **Secondary Hypertension**
  Hypertension occurring secondary to an underlying medical cause. Important secondary causes of chronic hypertension in pregnancy include:

  o Chronic kidney disease causes
    - Chronic Glomerulonephritis
    - Chronic renal insufficiency
    - Diabetic Nephropathy
  o Polycystic kidney disease
  o Acute Glomerulonephritis
  o Renal failure
  o Endocrine disorders
    - Diabetes mellitus
    - Cushing syndrome
    - Hyper aldosteronism
    - Pheo- chromocytoma
    - Thyrotoxicosis
  o Connective tissue disease
    - Lupus erythematosus
    - Periarterities nodosa
    - Anti phaspholipits
  o Arterial abnormalities
    - Renovascular Hypertension
    - Coarctation of the aorta
  o Essential familial hypertension
  o Obesity
• **White Coat Hypertension**

Some women with apparent essential hypertension may have white coat hypertension (raised blood pressure in the presence of a clinical attendant but normal blood pressure otherwise as assessed by ambulatory or home blood pressure monitoring). These women appear to have a lower risk of superimposed pre-eclampsia than women with true essential hypertension but are still at an increased risk compared with normotensive women. White-coat effect in early pregnancy is common. 40% of women progress to persistent hypertension at ≥ 20 weeks (i.e., gestational hypertension) and 8% to pre-eclampsia. Women with white-coat effect have risks (e.g., severe hypertension, preterm delivery, and NICU admission) intermediate between normotension and either chronic or gestational hypertension.

• **Transient Hypertensive Effect**

Elevated blood pressure may be due to environmental stimuli (e.g.; the pain of labour). A transient hypertensive effect is not associated with an increased risk of adverse outcomes.

**Gestational Hypertension**

- New onset of hypertension arising after 20 weeks gestation
- No additional maternal or fetal features of preeclampsia
- Resolves within 3 months postpartum

The earlier the gestation at presentation and the more severe the hypertension, the higher is the likelihood that the woman with gestational hypertension will progress to develop pre-eclampsia or an adverse pregnancy outcome.

The following women may have an increased risk of developing gestational hypertension:

- First time moms
- Women whose sister and mothers had PIH
- Women carrying multiples
- Women younger than age 20 or older than age 40
- Women who had high blood pressure or kidney disease prior to pregnancy

**Pre-eclampsia**

Pre-eclampsia is a multi-system disorder unique to human pregnancy characterised by hypertension and involvement of one or more other organ systems and/or the fetus. Raised blood pressure is commonly, but not always,
the first manifestation. Proteinuria is the most commonly recognized additional feature after hypertension but should no longer be considered mandatory to make the clinical diagnosis.

In other languages preeclampsia is a syndrome characterized by the onset of hypertension and either proteinuria or end-organ dysfunction after 20 weeks of gestation. Although most affected pregnancies deliver at term or near term with good maternal and fetal outcomes, these pregnancies are at increased risk for maternal and/or fetal mortality or serious morbidity. Additional signs and symptoms that can occur include visual disturbances, headache, epigastric pain, thrombocytopenia, and abnormal liver function. These clinical manifestations result from mild to severe microangiopathy of target organs, including the brain, liver, kidney, and placenta. Potential maternal sequelae include pulmonary edema, cerebral hemorrhage, hepatic failure, renal failure, and death. The fetal/neonatal burden of disease results from placental hypoperfusion and the frequent need for preterm delivery.

- Mild preeclampsia: diastolic blood pressure 90–99 mmHg, systolic blood pressure 140–149 mmHg.
- Moderate preeclampsia: diastolic blood pressure 100–109 mmHg, systolic blood pressure 150–159 mmHg.
- Severe preeclampsia: diastolic blood pressure 110 mmHg or greater, systolic blood pressure 160 mmHg or greater.

Preeclampsia is accompanied by one or more of the following signs of organ involvement:

- Proteinuria: spot urine protein/Creatinine >30 mg/ mmol or >300 mg/day or at least 1 g/L (++) on dipstick testing. OR in the absence of proteinuria
- Other maternal organ dysfunction:
  - Renal insufficiency: serum or plasma creatinine >90 μmol/L
  - Haematological involvement: Thrombocytopenia (<100,000 /μL), haemolysis or disseminated intravascular coagulation (DIC)
  - Liver involvement: Raised serum transaminases, severe epigastric and/or right upper quadrant pain
  - Neurological involvement: Seizure, hypereflexia with sustained clonus, persistent new headache, persistent visual disturbances

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8Lain KY, Roberts JM. Contemporary concepts of the pathogenesis and management of preeclampsia. JAMA 2002; 287:3183.
(photopsia, scotomata, cortical blindness, posterior reversible encephalopathy syndrome, retinal vasospasm), Stroke
  - Pulmonary edema
    ○ Fetal growth restriction

Rarely, pre-eclampsia presents before 20 weeks’ gestation; usually in the presence of a predisposing factor such as hydatidiform mole, multiple pregnancy, fetal triploidy, severe renal disease or anti phospholipid antibody syndrome.

**A. Causes of pre-eclampsia**

Any satisfactory theory concerning the etiology and pathophysiology of preeclampsia must account for the observation that hypertensive disorders due to pregnancy are very much more likely to develop in women who:

1. Are exposed to chorionic villi for the first time.
2. Are exposed to a superabundance of chorionic villi, as with twins or hydatiform mole
3. Have preexisting vascular disease
4. Are genetically predisposed to hypertension developing during pregnancy
5. Nutritional factors

**B. Risk Factors for Pre Eclampsia**

1. Maternal age < 20 years > 35 years
2. First pregnancy (nulliparity)
3. Multiple pregnancy
4. Interval since last pregnancy of more than 10 years
5. Hypertensive disease during a previous pregnancy
6. Body mass index (BMI) of 35kg / m2 or more at first visit
7. Family or previous story of preeclampsia
8. Anti-phospholipid anti bodies and systemic lupus erythromatosis
9. Chronic hypertension
10. Chronic kidney disease
11. Diabetes (Type I, II)
12. Auto immune disease
13. Social and economic causes
14. Race, ethnicity and environmental factors
Table 1. Diagnostic Criteria for Preeclampsia

| Blood pressure | • Greater than or equal to 140 mm Hg systolic or greater than or equal to 90 mmHg diastolic on two occasions at least 4 hours apart after 20 weeks of gestation in a woman with a previously normal blood pressure  
| | • Greater than or equal to 160 mm Hg systolic or greater than or equal to 110 mmHg diastolic, hypertension can be confirmed within a short interval (minutes) to facilitate timely antihypertensive therapy |
| and | |
| Proteinuria | • Greater than or equal to 300 mg per 24-hour urine collection (or this amount extrapolated from a timed collection)  
| | • Or  
| | • Protein/ creatinine ratio greater than or equal to 0.3*  
| | • Dipstick reading of 1+ (used only if other quantitative methods not available) |
| Or in the absence of proteinuria, new-onset hypertension with the new onset of any of the following: | |
| Thrombocytopenia | • Platelet count less than 100,000/microliter |
| Renal insufficiency | • Serum creatinine concentrations greater than 1.1mg/dL or a doubling of the serum creatinine concentration in the absence of other renal disease |
| Impaired liver function | • Elevated blood concentrations of liver transaminases to twice normal concentration |
| Pulmonary edema | |
| Cerebral or visual symptoms | |

*CEach measured as mg/dL

C. Symptoms of pre-eclampsia

Pregnant women should be made aware of the need to seek immediate advice from a healthcare professional if they experience symptoms of pre-eclampsia. Symptoms include:

- severe headache
- problems with vision, such as blurring or flashing before the eyes
- severe pain just below the ribs
- vomiting
- Sudden swelling of the face, hands or feet.
D. HELLP syndrome

HELPP Syndrome is a variant of severe preeclampsia (Haemolysis, Elevated Liver enzymes and Low Platelet count). Hemolysis, abnormal liver function tests, and thrombocytopenia have been recognized as complication of preeclampsia and eclampsia for many years. Maternal mortality is reported to be as high as 1-2%. Normal pregnancy is characterized by a fall in blood pressure, detectable in the first trimester and usually reaching a nadir in the second trimester. Blood pressure rises towards pre-conception levels towards the end of the third trimester. The development of HELLP syndrome may occur antepartum or postpartum.

A consensus of opinion is that prompt delivery is indicated if the syndrome develops beyond 34 weeks of gestation or earlier if there is disseminated intravascular coagulation, liver infarction or hemorrhage, renal failure, pulmonary edema, suspected abruption placenta, or non reassuring fetal status. Because the management of the patients with HELLP syndrome required the availability of neonatal and obstetric intensive care units and personnel with special expertise, patients with HELLP syndrome who are remote from term should receive care at a tertiary care center.

Epigastric or right upper quadrant pain in a woman with preeclampsia often represents hepatic involvement. This is called ‘Pre eclamptic Angina’. The pain responds poorly to analgesia but both the pain and associated increases in liver enzymes (AST, ALT) may subside (albeit temporarily) after blood pressure lowering, particularly with vasodilators. If the cause of epigastric or right upper quadrant pain is not clear, close ongoing assessment is required, with careful review of all indicators of maternal and fetal wellbeing and appropriate imaging of the liver and gallbladder.

Thrombocytopenia is the commonest hematologic abnormality seen in preeclampsia; the lower limit of the normal platelet count in pregnancy is approximately 140x10/L but the risk of spontaneous bleeding is not significantly increased until the count falls below 50 x 10/L. Even so, there are concerns with central neuraxial anaesthesia and analgesic techniques at higher levels (50-75 x 10 /L), and surgical bleeding may be increased even with moderate thrombocytopenia.

In a woman with pre eclampsia, the presence of any one of the following is an indicator of severe disease, even if not suggested on other criteria such as severity of hypertension:
- A maternal platelet count of 100000 microliter
- A transaminase level or LDH more than double the normal upper limit
- Haemolysis of any quantity

**SUPERIMPOSED PREECLAMPSIA**

Superimposed pre-eclampsia is diagnosed when a woman with chronic hypertension or pre-existing proteinuria develops one or more of the systemic features of pre-eclampsia after 20 weeks' gestation. Worsening or accelerated hypertension should increase surveillance for pre-eclampsia but it is not diagnostic.

Superimposed pre-eclampsia is likely when any of the following are present:

- A sudden increase in BP that was previously well controlled or escalation of antihypertensive medications to control BP.
- New onset of proteinuria or a sudden increase in proteinuria in a woman with known proteinuria before or early in pregnancy.

The diagnosis of superimposed preeclampsia with severe features is established when any of the following are present:

- Severe –range BP despite escalation of antihypertensive therapy
- Thrombocytopenia (platelet count less than 100 000 / microliter
- Elevated liver transaminases (two times the upper limit of normal concentration for a particular laboratory)
- New-onset and worsening renal insufficiency
- Pulmonary edema
- Persistent cerebral or visual disturbances

Clinicians should recognize that there is often ambiguity in the diagnosis of superimposed preeclampsia and that the clinical spectrum of disease is broad. Furthermore, women with superimposed preeclampsia can progress and develop end-organ involvement and adverse outcome. Therefore, increased surveillance but not intervention (e.g., delivery) is warranted even if the diagnosis is suspected and not definitive. Future investigation is needed to further refine the diagnosis, potentially including markers that are predictive of adverse outcome.

**ECLAMPSIA**

Eclampsia is defined as the presence of new-onset grand mal seizures in a woman with preeclampsia. Eclampsia is preceded by a wide range of signs and
symptoms, ranging from severe to absent or minimal hypertension, massive to no proteinuria, and prominent to no edema. Several clinical symptoms are potentially helpful in predicting impending eclampsia. These include persistent occipital or frontal headaches, blurred vision, photophobia, epigastric or right upper quadrant pain or both, and altered mental status. Eclamptic seizures contribute substantially to maternal morbidity and mortality, especially in developing countries. For many years, these were treated with several different anticonvulsants, and attempts to prevent eclampsia seizures were exercised sporadically.

There are no reliable clinical markers to predict eclampsia and conversely, the presence of neurological symptoms and/or signs is rarely associated with seizures. Seizures may occur antenatally, intra-partum or postnatally, usually within 24 hours of delivery but occasionally later. Hypertension and proteinuria may be absent prior to the seizure and not all women will have warning symptoms such as headache, visual disturbances or epigastric pain. If occurs at post-partum period the following conditions should be considered for differential diagnosis:

- Cerebral venous thrombosis
- Epilepsy
- Brain tumors
- Cerebral arterial aneurism
- Brain damage
- Encephalopathy

**DIAGNOSTIC OVERVIEW**

Clinical characteristics obtained via history, physical examination, and certain laboratory investigations may be used to help clarify the diagnosis. Fetal well-being must also be considered with the workup of the mother.

Preeclampsia is rare before the third trimester, and the diagnosis of severe hypertension or preeclampsia in the first or early second trimester necessitates exclusion of gestational trophoblastic disease and/or molar pregnancy. Mild lower extremity edema is common in normal pregnancy, but rapidly increasing or nondependent edema may be a signal of developing preeclampsia. However, edema is no longer included among the criteria for the diagnosis of preeclampsia.
New seizures in pregnancy suggest preeclampsia-eclampsia, but primary neurologic disorders must be excluded. Hyperaldosteronism and hypercortisolism are difficult to diagnose during pregnancy due to the high levels of progesterone and the normal increase in endogenous cortisol output.

1. Measurement of Blood Pressure

Accurate blood pressure measurement impacts on the diagnosis and management of hypertensive diseases in pregnancy. Blood pressure should be measured with the woman rested and in a sitting position with the arm at the level of the heart. An appropriately sized cuff should be used to avoid over or underestimation. If the mid-arm circumference is greater than 33cm, a large cuff should be used. The average of two blood pressure readings needs to be taken to properly diagnose hypertension.

2. Diagnosis of Hypertension

Hypertension in pregnancy should be defined as:

- A systolic blood pressure ≥ 140mmHg
- A diastolic blood pressure ≥ 90mmHg

These measurements should be based on the average of at least two measurements, taken using the same arm, several hours apart. Elevations of both systolic and diastolic blood pressures have been associated with adverse fetal outcome and therefore both are important. For severe hypertension, a repeat measurement should be taken for confirmation no more than 15 minutes later.

A. Routine Tests

Diagnostic proteinuria (described above).

- Decreased hematocrit secondary to severe hemolysis in HELLP syndrome.
- Elevated Prolonged prothrombin and partial thromboplastin times that may be due to primary coagulopathy, hepatic synthesis dysfunction, or abruptio placentae leading to disseminated intravascular coagulation.
- Decreased fibrinogen, increased fibrin degradation products, or both, as a result of coagulopathy or abruptio placentae.hematocrit resulting from decreased intravascular volume secondary to third spacing of fluid.
o Elevated serum uric acid level ≥5 mg/dL. Elevated serum creatinine ≥1.2 mg/dL.
o Remember that creatinine normally decreases in pregnancy, so even slight increases warrant investigation
o Elevated serum transaminases (AST >70 IU/L). Decreased platelet count ≤100,000/μL).
o Prolonged prothrombin and partial thromboplastin times that may be due to primary coagulopathy, hepatic synthesis dysfunction, or abruptio placentae leading to disseminated intravascular coagulation.

B. Hematologic Evaluation
A complete blood cell (CBC) count may reveal the following:
o Anemia due to microangiopathic hemolysis, hemoconcentration due to third spacing, or physiologic hemodilution of pregnancy
o Peripheral smear (schistocytes, burr cells, echinocytes)
o Increased bilirubin (>1.2 mg/dL)
o Thrombocytopenia (< 100,000) due to hemolysis and low platelet count associated with HELLP syndrome (seen in 20-25% of patients with eclampsia) [4]
o Low serum haptoglobin levels
o Elevated lactate dehydrogenase (LDH) levels (threshold of 180–600 U/L)

The coagulation profile may reveal normal prothrombin (PT) and activated partial thromboplastin (aPTT) times, fibrin split products, and fibrinogen levels. Rule out associated disseminated intravascular coagulation (DIC).

C. Renal and Hepatic Evaluation
1. Proteinuria Quantification Serum low

Urinary reagent-strip testing is simple, cheap and an appropriate screening test for proteinuria especially when the suspicion of pre-eclampsia is low.
Approximate equivalence is:
1+ = 0.3 g/l
2+ = 1 g/l
3+ = 3 g/l
There is considerable observer error with visual reagent-strip assessment. Consequently, automated reagent-strip readers, which significantly improve both false positive and negative rates, should be used. If an automated reagent-
strip reading device yields a result of 1+ or more, proteinuria should be formally quantified.

The gold standard for diagnosing abnormal proteinuria in pregnancy is a 24-hurinary protein >300 mg per day, although its accuracy is affected by numerous factors such as adequacy and accuracy of collection, and variations in protein excretion. Where 24-hour urine collection is used to quantify proteinuria, there should be a recognized method of evaluating the completeness of the sample.

A spot urine protein/Creatinine cut-off level of 30 mg/mmol equates to a 24-h urine protein >300 mg per day and this eliminates the inherent difficulties in undertaking the 24-h urine collections and speeds up the process of decision making.

1. Serum Creatinine level

The serum Creatinine level is elevated in eclampsia because of a decreased intravascular volume and a reduced glomerular filtration rate (GFR). Creatinine clearance (CrCl) may be less than 90 mL/min/1.73 m2

2. Urinalysis and Uric Acid levels

Proteinuria is typically one of the presenting symptoms in patients with eclampsia. A timed collection has been the criterion standard for urinalysis to detect proteinuria (>300 mg/24 h or >1 g/L). Protein per unit time measured over 24 hours has been used traditionally; however, 12-hour collections have proved to be as accurate.

Although investigations suggest that when measuring intact urinary albumin levels using high-performance liquid chromatography in an early and uncomplicated pregnancy, spot urinary albumin: creatinine ratio (ACR) values are higher. If measured early in the second trimester, an ACR of 35.5 mg/mmol or higher may predict preeclampsia before symptoms arise. Uric acid levels may be mildly increased.

3. Liver Function Tests (LFT)

Liver function test results may reveal the following (20-25% of patients with eclampsia):

- Aspartate aminotransferase (SGOT) level higher than 72 IU/L
- Total bilirubin levels higher than 1.2 mg/dL
- LDH level higher than 600 IU/L
- Elevated levels due to hepatocellular injury and HELLP syndrome
4. **Chest Radiography:**

Imaging studies should not be performed in an unstable patient, and they should not delay rapid facilitated delivery in a woman thought to have severe preeclampsia or eclampsia.

Obtain chest radiographs to evaluate for pulmonary edema in the setting of dyspnea or hypoxia occurring in a woman with preeclampsia, as shown in the following image.

Non cardiogenic pulmonary edema in a patient with preeclampsia. This is due to a capillary leak that can be a primary component of preeclampsia. The radiograph demonstrates a diffuse increase in lung markings without the cephalization or vascular redistribution that is seen in patients with pulmonary edema from systolic dysfunction. This patient had rapid clinical improvement after only 10 mg of intravenous furosemide.

The fetal ionizing radiation exposure of one maternal chest radiograph with abdominal shielding is only 0.001 rads. Although no dose of ionizing radiation is absolutely safe during pregnancy, a commonly held acceptable cumulative dose is 5 rads during pregnancy (approximately 5000 maternal chest radiographs could be performed before reaching this safety threshold).

5. **CT Scanning and MRI of the Head:**

Computed tomography (CT) scanning of the head, with or without contrast, can exclude cerebral venous thrombosis, intracranial hemorrhage, and central nervous system lesions, all of which can occur in pregnancy and present with seizures.

Although obtaining a CT scan in eclampsia is not routine, abnormalities have been observed in up to 50% of women imaged.
Characteristic CT scan findings include cortical hypodense areas, particularly in the occipital lobes, and diffuse cerebral edema, which is believed to correspond to petechial hemorrhages and diffuse edema noted in postmortem studies.

CT scan findings may include the following:

- Cerebral edema
- Diffuse white matter low-density areas
- Patchy area of low density
- Occipital white matter edema
- Loss of normal cortical sulci
- Reduced ventricular size
- Cerebral hemorrhage
- Intraventricular hemorrhage
- Parenchymal hemorrhage (high density)
- Cerebral infarction
- Low attenuation areas
- Basal ganglia infarctions

Abnormal magnetic resonance imaging (MRI) findings of the head have been reported in up to 90% of women imaged. These include an increased signal at the gray-white matter junction on T2-weighted images, as well as cortical edema and hemorrhage. The syndrome of posterior reversible encephalopathy (PRES), indicative of central vasogenic edema, has been increasingly recognized as a component of eclampsia.

Consider CT scanning or MRI in patients who have been involved in trauma, are refractory to magnesium sulfate therapy, or have atypical presentations (eg, seizures >24 h after delivery, absence of severe hypertension).
Non enhanced computed tomography scan of a woman's brain following an eclamptic seizure, showing hypo dense areas involving white matter of the occipital lobes and the high frontal/parietal lobes.

Findings in women with preeclampsia may include bilateral hypo dense areas, called venous infarcts or posterior reversible leukoencephalopathy, in the occipital and parietal regions. They represent focal and reversible areas of edema that are the result of capillary leak or focal areas of impaired venous flow. Generally, these areas resolve as the preeclamptic process reverses.

6. **Trans abdominal Ultrasonography**

Transabdominal ultrasonography is used to estimate gestational age. This may also be used to rule out Abruptio placenta, which can complicate eclampsia.
Ultrasonography or CT scanning of the liver may be used to evaluate for sub capsular hemorrhage or infarction in the setting of persistent severe RUQ pain or markedly elevated hepatic transaminases.

7. **Echocardiography and Electrocardiography**

   Limited echocardiography may be performed to evaluate for LVH in chronic hypertension and to exclude cardiomyopathy or occult valvular disease in pregnant women with pulmonary edema.

   Perform a 12-lead electrocardiogram (ECG) to evaluate for LVH in women with chronic hypertension

8. **Electroencephalography**

   An electroencephalogram (EEG) may be indicated to evaluate recurrent seizure activity, persistent altered level of consciousness, or altered mental status. Following eclampsia, the EEG may reveal epileptiform activity. More commonly, the test shows nonspecific diffuse slowing that may persist for several weeks after delivery

9. **Histological Findings**

   Endothelial dysfunction and vasospasm observed in preeclampsia affect multiple regions of the body, including the maternal brain, kidneys, liver, lungs, heart, and placenta. Pathology demonstrates areas of edema, micro infarctions, and micro hemorrhage in the affected organs.

   The placenta typically shows in situ thrombosis and decidual vasculopathy/incomplete decidualization of the spiral arterioles, which may be part of the pathogenesis of preeclampsia. This can affect the fetus via decreased utero placental blood flow. The decrease in flow can manifest clinically as non-reassuring fetal heart rate testing, low score on a biophysical profile, oligohydramnios, and fetal growth restriction.

   The kidneys may reveal glomerular endotheliosis that is associated with proteinuria greater than 300 mg in 24 hours or, more rarely, acute tubular necrosis (ATN) or cortical necrosis.
Management / treatment of the Hypertensive Disorders of Pregnancy

1. Chronic hypertensive

Women with chronic hypertension, whether essential or secondary, are at high risk of pregnancy complications and should therefore be observed frequently during the pregnancy by an obstetrician familiar with the management of hypertension in pregnancy. The frequency of review will be determined by such factors as how successfully blood pressure is controlled, the number of agents used, associated disorders (e.g. renal disease, proteinuria) and by the gestation but should be increased in the second half of pregnancy when complications are more likely. There may be a role for the use of home blood pressure monitoring equipment for this group of patients.

Low-dose Aspirin (LDA) should be initiated ideally at 12 weeks’ gestation and in any case prior to 16 weeks’ gestation. Serial surveillance for fetal growth restriction should be carried out as the risk for fetal growth restriction is higher in women with chronic hypertension9.

Clinicians must be vigilant for superimposed pre-eclampsia in women with chronic hypertension.

For pregnant women with chronic hypertension, treatment with anti-hypertensive should be considered if already on medication pre-pregnancy or if moderate to severe hypertension develops. Mild hypertension does not require treatment. There is some evidence that in the presence of end organ damage tighter blood pressure control is beneficial and therapy should be used to keep systolic blood pressure below 140 mmHg and diastolic blood pressure at 80-90 mmHg.

2. Gestational Hypertension

Women with new onset gestational hypertension should be cared for by an obstetrician. Combined antenatal care between the hospital and GP is acceptable.

9 Clinical Practice Guideline ‘Fetal Growth Restriction- Recognition, Diagnosis and Management’ published by the Institute of Obstetricians and Gynaecologists, Royal College of Physicians of Ireland and Clinical Strategy and Programmes Directorate, Health Service Executive.)
Women with mild hypertension do not need treatment but should be seen weekly for blood pressure assessment and screening for proteinuria. Blood tests should be performed at diagnosis and not repeated unless clinically indicated. Ultrasound for fetal assessment should be carried out at diagnosis, but need not be repeated if normal and clinical surveillance is satisfactory.

Women with moderate hypertension should be commenced on medication and be reviewed at least twice a week to assess blood pressure. At each visit urine should be checked for proteinuria. Bloods tests should be performed at diagnosis but not repeated unless clinically indicated. Ultrasound for fetal assessment should be carried out at diagnosis, but need not be repeated if normal and clinical surveillance is satisfactory.

Those with severe hypertension should be commenced on medication and admitted to hospital until blood pressure stabilises. While an inpatient blood pressure needs to be assessed regularly and urine should be checked for proteinuria daily but once stabilised and discharged home this can be reduced to twice weekly review and assessment.

Blood tests should be performed daily while inpatient and an ultrasound for fetal assessment should be carried out at diagnosis. Serial ultrasound surveillance should be every fortnight with daily CTG while inpatient. Consideration should be given to the use of corticosteroids for fetal lung maturation if less than 36 completed weeks’ gestation.

In the event intrauterine growth restriction (IUGR) is identified, the frequency of surveillance will need to be increased.

Clinicians must be vigilant for progression to pre-eclampsia in women with gestational hypertension and reassessment for pre-eclampsia needs to be considered if clinically indicated.

Instituting medical therapy of mild hypertension has not been shown to improve neonatal outcomes and may mask the diagnosis and recognition of progression to severe disease. Treatment should therefore be reserved for moderate to severe hypertension, with the goal of reducing maternal complications such as cerebral vascular accidents, and prolongation of the pregnancy. Severe hypertension requires urgent assessment and management. Increasing evidence exists that cerebral perfusion pressure is altered in pregnant women making them more susceptible to cerebral hemorrhage, posterior reversible encephalopathy syndrome and hypertensive encephalopathy. It is
universally agreed that severe hypertension should be lowered promptly, albeit carefully, to prevent such complications.10.

For women without underlying medical problems, antihypertensive drug therapy should be used to keep systolic blood pressure below 150 mmHg and diastolic blood pressure at 80-99 mmHg. For women with underlying medical problems, such as diabetes or renal disease, there is some evidence that tighter control is beneficial and therapy should be used to keep systolic blood pressure below 140 mmHg and diastolic blood pressure at 80-90 mmHg. Tighter control does not seem to be associated with adverse fetal or neonatal outcomes and is associated with a lower frequency of severe maternal hypertension.

There is insufficient evidence to identify a single preferred agent for non-acute, moderate-severe hypertension management. However, there is consistency across guidelines internationally regarding the acceptability of oral labetalol, nifedipine and methyldopa as first line agents for non-acute treatment of hypertension in pregnancy, based on good quality evidence. Oral labetalol should be considered as first line treatment, with a recommendation to consider alternatives methyldopa and nifedipine only after considering maternal, fetal and neonatal side effect profiles.

Second line agents include hydralazine and Prazocin. Angiotension converting enzyme (ACE) inhibitors, angiotensin receptor blockers (ARB) and renin inhibitors, have been associated with fetal renal abnormalities, and are contraindicated in pregnancy.

For those with moderate-severe hypertension, with medical therapy should be reviewed twice weekly to assess blood pressure levels. If the initial dose of any antihypertensive drug fails to adequately control blood pressure, the dose should be increased incrementally until the maximum dose is reached. If adequate control of blood pressure has still not been achieved, a second antihypertensive agent may be introduced. This drug should be prescribed in addition to and not instead of the first agent.

3. Management of preeclampsia and HELLP Syndrome

The first consideration in the management of women with mild gestational hypertension or preeclampsia without severe futures is always safety of the woman and her fetus. The second is delivery of a mature newborn that

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10 Clinical Practice Guideline: The Diagnosis and Management of Pre-eclampsia and Eclampsia
will not require intensive care or prolonged neonatal care. Once the diagnosis of mild gestational hypertension or preeclampsia without severe futures is established, subsequent management will depend on the results of maternal and fetal evaluation, gestational age, presence of labor or rupture of membranes, vaginal bleeding, and wishes of the woman (Fig. 1-1).

Figure 1-1. Management of mild gestational hypertension or preeclampsia without severe features
• Observe in labor and delivery for first 24–48hs
• Corticosteroids, magnesium sulfate prophylaxis, and antihypertensive medications
• Ultrasonography, monitoring of fetal heart rate, symptoms, and laboratory tests

**Contraindications to continued expectant management**
- Eclampsia
- Nonviable fetus
- Pulmonary edema
- Abnormal fetal test result
- Disseminated intravascular coagulation
- Intrapartum fetal demise
- Uncontrollable severe hypertension

**Are there additional expectant complications?**
- Greater than or equal to 33 5/7 weeks of gestation
- Persistent symptoms
- HELLP or partial HELLP syndrome
- Fetal growth restriction (less than fifth percentile)
- Severe oligohydramnios
- Reversed end-diastolic flow (umbilical artery Doppler studies)
- Labor or premature rupture of membranes
- Significant renal dysfunction

**Expectant management**
- Facilities with adequate maternal and neonatal intensive care resources
- Fetal viability - 33 6/7 weeks of gestation
- Inpatient only and stop magnesium sulfate
- Daily maternal – fetal tests
- Vital signs, symptoms, and blood test
- Oral antihypertensive drugs

**Delivery**
- Achievement of 34 0/7 weeks of gestation
- New-onset contraindications to expectant management
- Abnormal maternal – fetal test results
- Labor or premature rupture of membranes

**Corticosteroids for fetal maturation**
- Delivery after 48 hours

**Delivery once maternal condition is stable**
- Yes

**Figure 1-2. Management of severe preeclampsia at less than 34 weeks of gestation**
Maternal and Fetal Monitoring

During expectant management, maternal and fetal conditions should be frequently monitored as follows:

i. Maternal assessment

- Vital signs, fluid intake, and urine output should be monitored at least every 8 hours
- Symptoms of severe preeclampsia (headaches, visual changes, retrosternal pain or pressure, shortness of breath, nausea and vomiting, and epigastric pain) should be monitored at least every 8 hours
- Presence of contractions, rupture of membranes, abdominal pain, or bleeding should be monitored at least every 8 hours
- Laboratory testing (CBC and assessment of platelet count, liver enzyme, and serum creatinine levels) should be performed daily. (These tests can be then be spaced to every other day if they remain stable and patient remains asymptomatic.)

ii. Fetal assessment

- Kick count and NST with urine contraction monitored daily
- Biophysical profile twice weekly
- Serial fetal growth should be performed every 2 weeks and umbilical artery Doppler studies should be performed every 2 weeks if fetal growth restriction is suspected

Initial Evaluation of Women with superimposed Preeclampsia

Initial evaluation of women with superimposed preeclampsia should occur in a hospital sitting to confirm the diagnosis, evaluate maternal-fetal status, and monitor for progressive worsening of the disease. The clinical workup should include questions about symptoms associated with preeclampsia (neurologic symptoms, epigastric or right upper quadrant pain, nausea and vomiting, vaginal bleeding, and fetal movement). Serial BP measurements should be obtained. Physical examination should be performed with attention to signs of preeclampsia and associated complications. Proteinuria should be assessed by a protein/creatinine ratio or 24-hour urine collection. Laboratory evaluation should also include a complete blood count with platelets, liver
transaminases, lactic dehydrogenase, and creatinine assessment. Uric acid assessment also may be helpful if Uric acid concentrations are known from early pregnancy because hyperuricemia is associated with adverse outcomes in superimposed preeclampsia and also with early renal dysfunction, which may be present with chronic hypertension. Ideally, these laboratory results are compared with baseline information obtained in early pregnancy. If abnormalities are of new-onset then the diagnosis of superimposed preeclampsia; end-organ involvement; or hemolysis, elevated liver enzymes, and low platelet count (HELLP syndrome) can be confirmed. If abnormalities are long-standing or of unknown duration, result should be cautiously interpreted before a definitive diagnosis is established. Although not included in the diagnostic criteria for superimposed preeclampsia, fetal growth and well-being should be assessed when superimposed preeclampsia is suspected. Additional testing or investigations or both may warranted if there are concerns regarding fetal status.

**Indications for Delivery during Expectant Management**

In the published studies of preterm severe preeclampsia managed expectantly, delivery has typically been pursued at proximately 34 weeks of gestation. However, deterioration of maternal or fetal conditions before this gestational age is the most common reason for delivery. Maternal indications for delivery are delineated in figure 1-2. Delivery should also be considered for women whose health is declining or who are non-adherent with ongoing inpatient observation; those developing persistent epigastric or right upper quadrant pain, nausea, or vomiting; and those who develop preterm labor or premature rupture of membranes.

1. **Maternal indications for delivery**
   - Recurrent severe hypertension
   - Recurrent symptoms of severe preeclampsia
   - Progressive renal insufficiency (serum Creatinine concentration greater than 1.1 mg/dL or a doubling of the serum Creatinine concentration in the absence of other renal disease)
   - Persistent thrombocytopenia or HELLP syndrome
   - Pulmonary edema
   - Eclampsia
   - Suspected Abruptio placenta
   - Progressive labor or rupture of membranes
2. **Fetal indications for delivery**

- Gestational age of 34 0/7 weeks
- Severe fetal growth restriction (Ultrasonography estimate of fetal weight less than the fifth percentile)
- Persistent oligohydramnios (maximum vertical pocket less than 2 cm)
- BPP of 4/10 or less on at least two occasions 6 hours apart
- Reversed end-diastolic flow on umbilical artery Doppler studies
- Recurrent variable or late decelerations during NST
- Fetal death

3. **Elective cesarean delivery:**

In sense of urgency because of severity of preeclampsia and need to coordinate NICU, have led some practitioners to advocate C.S. The indication of labor was not successful in 35% of the women in induced group, but it was not harmful to their very low birth weight infants (750-1500 gram)

**Antepartum Management of Superimposed Preeclampsia**

General consideration in the Antepartum management of women with superimposed preeclampsia includes the administration of antenatal corticosteroids and use of Magnesium sulfate for seizure prophylaxis. Ongoing management and timing of delivery is based on gestational age and the severity of disease.

**Antenatal Corticosteroids**

Women with superimposed preeclampsia diagnosed before 37 weeks of gestation are at increased risk of preterm delivery. Therefore, antenatal corticosteroids should be administered at less than 34 weeks of gestation for fetal lung maturity benefit to decrease neonatal morbidity and mortality. For women with superimposed preeclampsia who receive expectant management at less than 34 0/7 weeks of gestation, the administration of corticosteroids for fetal lung maturity benefit is recommended.

**Some approved recommendations**

- It is suggested that corticosteroids be administered and delivery deferred for 48 hours if maternal and fetal conditions remain stable for women with severe preeclampsia and a viable fetus at 33 6/7 weeks or less of gestation with any of the following:
- Preterm premature rupture of membranes
- Labor
- Low platelet count (less than 100,000/ micro liter)
- Persistently abnormal hepatic enzyme concentrations (twice or more the upper normal values)
- Fetal growth restriction (less than the fifth percentile)
- Severe oligohydraminios (amniotic fluid index less than 5 cm)
- Reversed end-diastolic flow on umbilical artery Doppler studies
- New-onset renal dysfunction or increasing renal dysfunction
- It is recommended that corticosteroids be given if the fetus is viable and at 33 6/7 weeks or less of gestation, but delivery not be delayed after initial maternal stabilization regardless of gestational age for women with severe preeclampsia that is complicated further with any of the following:
  - Uncontrollable severe hypertension
  - Eclampsia
  - Pulmonary edema
  - Abruptio placenta
  - Disseminated intravascular coagulation
  - Evidence of non-reassuring fetal status
  - Intrapartum fetal demise
## Table 2. Antihypertensive Drugs Recommended in Pregnancy

<table>
<thead>
<tr>
<th>Drugs</th>
<th>Dosage Range</th>
<th>Action</th>
<th>Contraindication and Comments</th>
</tr>
</thead>
</table>
| Labetalol      | Standard dose: 200-600 mg orally per day in 2-4 divided doses  
Maximum dosage: 2,400 mg per day | Beta blocker with mild alpha vasodilator effect | Avoid in women with cardiac Conduction abnormalities, systolic heart failure or asthma.  
SI: Bradycardia, bronchospasm, nausea, headache which usually resolves within 24 hours |
| Nifedipine (extended release)  
i.e. Adalat LA | Standard dose: 30-60 mg orally per day  
Maximum dosage: 90 mg per day | Calcium channel antagonist | Ensure correct form prescribed; short acting is not recommended due to risk of hypotension  
Not recommended before 20 weeks’ gestation  
Caution regarding possible interaction with intravenous magnesium sulphate leading to severe hypotension  
Avoid in women with aortic stenosis  
SI: Severe headache, flushing, tachycardia, constipation |
| Methyldopa     | Standard dose: 250-1000 mg orally per day in 2-3 divided doses  
Maximum dosage: 3000 mg per day | Centrally acting | Slow onset over 24 hours  
SI: dry mouth, blurred vision, depression, and sedation (dose dependant)  
Associated with hepatitis, haemolytic anaemia  
Withdrawal effects: rebound hypertension  
Stop +/- substitute with other agents within 2 days post delivery |
### Table 3. Flowchart for Hypertension in Pregnancy Management

<table>
<thead>
<tr>
<th>Admission to Hospital:</th>
<th>Mild HTN 140/90-149/99</th>
<th>Moderate HTN 150/100-159/109</th>
<th>Severe HTN 160/110 or higher</th>
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<tbody>
<tr>
<td>HTN &gt;20/40</td>
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<tr>
<td>No proteinuria</td>
<td>No</td>
<td>Commence TX and monitor BP -</td>
<td>Yes –until BP stabilised</td>
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<tr>
<td>No symptoms</td>
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<td>Home/admit</td>
<td>&lt;159/109</td>
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<td>Treatment:</td>
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<td>Measurement of BP:</td>
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**Delivery**

**A. Timing of Delivery**

Timing of delivery is dependent on the severity of the maternal condition and the gestation at which the hypertension presents. A clinical assessment should include the woman’s symptoms, the severity of the hypertension, well-being of the fetus and the favorability of the cervix.\(^{11}\) suggests that in women with gestational hypertension, induction of labour after 37 weeks’ gestation is...
associated with a significant reduction in adverse maternal outcome including progression to pre-eclampsia and adverse neonatal outcomes without an increase in Caesarean section rates.

For women with uncomplicated chronic hypertension who are otherwise well with controlled blood pressure at ≥ 37+0 weeks’ gestation, delivery should be considered at 38+0 to 39+6 weeks’ gestation.

B. Mode of Delivery

For women with any hypertensive disorder of pregnancy, vaginal delivery should be considered unless a Caesarean delivery is required for the usual obstetric indications. Antihypertensive treatment should be continued throughout labour and delivery to maintain systolic blood pressure at < 160 mmHg and diastolic blood pressure at < 110 mmHg. The third stage of labour should be actively managed with oxytocics, however ergometrine maleate should not be administered to women with any hypertensive disorder of pregnancy.

POSTNATAL MANAGEMENT

• CARE IN THE FIRST 6 WEEKS POSTPARTUM

Blood pressure usually stabilises in the first two months following pregnancy. Appraisal and treatment should be based on the assumption that levels will decline. In many women with pre-existing hypertension blood pressure is often unstable immediately after delivery and may require a medication adjustment. Blood pressure should be measured during the time of peak postpartum blood pressure, at days 3 to 6 after delivery. Women with pre-existing hypertension who did not require treatment during the pregnancy often need treatment postpartum.

Severe postpartum hypertension must be treated with antihypertensive therapy to keep systolic blood pressure below 150 mmHg and diastolic blood pressure at 80-99 mmHg. For women with underlying medical problems, such as diabetes or renal disease, there is some evidence that tighter control is beneficial and therapy should be used to keep systolic blood pressure below 140 mmHg and diastolic blood pressure at 80-90 mmHg.
Non-steroidal anti-inflammatory drugs should not be given postpartum if hypertension is difficult to control, if there is evidence of kidney injury (oliguria and/or creatinine≥ 90 μM), or if platelets are <50 to 109/L.

Postpartum thromboprophylaxis should be considered in women with pre-eclampsia, particularly in the presence of other risk factors. Antihypertensive agents generally acceptable for use in breastfeeding include the following: labetalol, nifedipine XL, methyldopa, captopril, and enalapril.

- CARE BEYOND 6 WEEKS POSTPARTUM

Follow-up after 6 weeks is required to ensure resolution of pregnancy-related changes and ascertain the need for ongoing care. Women with chronic hypertension, a long duration of antihypertensive treatment in pregnancy, higher maximum systolic and diastolic blood pressures, higher body mass index, or occurrence of preterm pre-eclampsia are more likely to have sustained hypertension postpartum (exceeding 6 weeks).

Women with persistent hypertension not previously assessed should undergo routine work-up according to standard regimens. Advice regarding future lifestyle and optimization of risk factors in subsequent pregnancies may be required. This is particularly relevant for women who are obese, have cardiovascular risk factors, secondary hypertension, or end-organ disease.
<table>
<thead>
<tr>
<th>Table 4. Flowchart for Postnatal Hypertension Management</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Postnatal HTN</strong>&lt;6/52 postpartum</td>
</tr>
<tr>
<td>Treatment:</td>
</tr>
<tr>
<td></td>
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<td></td>
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<tr>
<td></td>
</tr>
<tr>
<td>Measurement of BP:</td>
</tr>
<tr>
<td></td>
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<tr>
<td></td>
</tr>
<tr>
<td>How often to review:</td>
</tr>
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<td></td>
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<td></td>
</tr>
</tbody>
</table>
• **LONG-TERM CONSEQUENCES**

Women who have been diagnosed with either pre-eclampsia or gestational hypertension are at increased risk of subsequent hypertension and cardiovascular disease. We recommend counselling women who have had hypertensive disorders in pregnancy that they will benefit from avoiding smoking, maintaining a healthy weight, exercising regularly and eating a healthy diet. It is recommended that all women with previous hypertensive disorders in pregnancy have an annual blood pressure check and regular assessment of other cardiovascular risk factors including serum lipids and blood glucose.

**Management of eclampsia**

There are four main aspects to care of the woman who sustains eclampsia.

**1. Resuscitation**

Resuscitation requires institution of intravenous access, oxygen by mask, assuring a patent airway and institution of intravenous access. Intravenous diazepam (2mg/min to maximum of 10mg) or Midazolam (0.1 – 0.2mg /kg IV or IM) may be given if the seizure is long. Intravenous Magnesium Sulphate is the agent of choice. See Loading dose- 4gm over 20 mins (rate 150 mL/hr for 20 mins only = 50mL).

**2. Prevention of further seizures**

Following appropriate resuscitation, treatment should be continued with Magnesium Sulphate. Ref. Administration of Magnesium Sulphate clinical guideline [Magnesium Sulphate Anticonvulsant Therapy](#) Annex 1

**3. Control of hypertension**

Control of severe hypertension to levels below 160/100 mmHg by parenteral therapy is essential as the threshold for further seizures is lowered after eclampsia, likely in association with vasogenic brain edema. In addition, the danger of cerebral hemorrhage is real.
4. Delivery

Arrangements for delivery should be decided once the woman’s condition is stable. In the meantime, close fetal monitoring (continuous CTG) should be maintained. There is no role, with currently available treatment, for continuation of pregnancy once eclampsia has occurred, even though many women may appear to be stable after control of the situation has been achieved.

Prevention of eclampsia in the woman with preeclampsia

The drug of choice for the prevention of eclampsia is magnesium sulphate. Although there is good evidence for the efficacy of this therapy, the case for its routine administration in women with preeclampsia in countries with low maternal and perinatal mortality rates is less than compelling.

Evidence indicates that magnesium sulphate is the superior drug to use in the prevention and the treatment of eclamptic seizures.

In the patient with known renal disease or myasthenia gravis however, phenytoin sodium is the anti-seizure medication of choice. Phenytoin sodium is administered in a total dose of 15mg/kg at an infusion rate of 40mg/min with continuous cardiac and blood pressure monitoring.

i. Administration of Magnesium Sulphate
See Clinical Guideline Magnesium Sulphate Anticonvulsant Therapy

Magnesium Sulphate should not be prescribed for the prevention of eclampsia without discussion with the Consultant Obstetrician on call.

Magnesium sulphate therapy is recommended for use Antepartum, Intrapartum and within the first 24 hours postpartum for severe preeclampsia (as defined on annex 1) when the following factors are present:
- persistently elevated blood pressure despite adequate hypotensive therapy and appropriate fluid management,
- Evidence of CNS dysfunction, thrombocytopenia or liver disease.

For women with chronic hypertension and superimposed preeclampsia with severe features, the administration of Intrapartum- postpartum parenteral magnesium sulfate to prevent Eclampsia is recommended.

ii. **BEFORE DISCONTINUATION OF MgSO4 THERAPY:**
- The blood pressure should be stable (consistently below 150/100)
- The patient should have adequate diuresis
- The patient should be clinically improved (absence of headache, epigastric pain).

**Postpartum Management**

Monitor the patient in the Adult Special Care Unit or Labour and Birth Suite until she begins to recover. Continue magnesium sulphate until stabilization and adequate diuresis is achieved. Antihypertensive therapy should be commenced if the BP is >150 mmHg systolic or >100 mmHg diastolic in the first four post-partum days. Options for antihypertensive therapy include:

- **Labetalol** 100mg TDS to start
- **Atenolol** 50mg daily. On rare occasions, may need increasing to 100mg/day.
- **Nifedipine SR** 20mg BD to start.
- **Enalapril** 5-10mg daily.

If there is significant bleeding attributed to preeclamptic thrombocytopenia at any time in the puerperium a platelet transfusion should be given (consult with Consultant Hematologist or Obstetric Physician). In the absence of bleeding, consider a platelet transfusion in the first 72 hours only if the count falls below 40,000 and there is concern of possible bleeding (e.g. after Caesarean Section).

If the count remains below 40,000 after 72 hours from delivery without significant bleeding and without sign of impending recovery, consultation with the Consultant Hematologist or Obstetric Physician is indicated.
Resolution of preeclampsia

After delivery, all clinical and laboratory derangements of preeclampsia recover, but there is often a delay of several days, and sometimes longer, in return to normality. On the first day or two after delivery, liver enzyme elevations and thrombocytopenia will often worsen before they reverse. Hypertension may persist for days, weeks or even up to three months and will require monitoring and slow withdrawal of antihypertensive therapy. Resolution is still assured if the diagnosis was pre-eclampsia and there is no other underlying medical disorder. The woman and her family are often overwhelmed and distressed from their experience and appropriate counseling post-partum should include psychological and family support.

All women who develop preeclampsia and gestational hypertension are at risk of these disorders in future pregnancies and should receive appropriate counseling before embarking upon another pregnancy

NOTE: Magnesium Sulphate may also be used antenatally prior to preterm birth for Neuro Protection of the fetus post birth- to reduce the incidence of cerebral palsy. See Preterm Birth guideline.

Management of HELLP Syndrome

Antenatal management

If the platelet count is sufficiently low to present a hazard for operative delivery, a platelet transfusion should be considered (consult with Consultant Hematologist or Obstetric Physician).
Annex 1:

**MEDICATION A-Z**

The information provided is for the use in Obstetrics and Gynecology only

<table>
<thead>
<tr>
<th>Drug</th>
<th>MAGNESIUM SULPHATE INFUSION</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Presentation</strong></td>
<td>IV Infusion bag: Magnesium Sulphate 8g in 100mL (approx. 32mmol Mg)</td>
</tr>
<tr>
<td><strong>WNHS Restrictions</strong></td>
<td>High Risk Medication List</td>
</tr>
<tr>
<td><strong>Dose</strong></td>
<td>NOTE: ALL INFUSIONS MUST BE GIVEN VIA AN INFUSION PUMP</td>
</tr>
<tr>
<td></td>
<td>Loading dose: Infuse 4g of MgSO4 over 20 minutes. This equates to an infusion rate of <strong>150mL per hour for 20 minutes</strong> (i.e. the woman receives only 50mL) where a solution of 8g of MgSO4 in 100mL bag is used at King Edward Memorial Hospital (KEMH).</td>
</tr>
<tr>
<td></td>
<td>Maintenance: The dose for maintenance infusion is 1g of MgSO4 per hour. This equates to an infusion rate of <strong>12.5mL per hour</strong> where a solution of 8g of MgSO4 in 100mL bag is used at KEMH.</td>
</tr>
<tr>
<td></td>
<td>Recurrent seizures/eclampsia occurring during prophylaxis: 2 - 4g of MgSO4 over 5 to 10 minutes. This equates to an infusion rate of <strong>300mL per hour for 5 minutes</strong> (i.e. the woman receives 25mL) where a solution of 8g of MgSO4 in 100mL bag is used at KEMH.</td>
</tr>
<tr>
<td><strong>Administration</strong></td>
<td>IV infusion: must be administered via a controlled infusion device</td>
</tr>
<tr>
<td><strong>Comments</strong></td>
<td>□ Monitor: BP, Deep tendon reflexes, urine output and respiratory function</td>
</tr>
<tr>
<td></td>
<td>□ Cease infusion if: Absent patella reflexes</td>
</tr>
<tr>
<td></td>
<td>• Urinary output less than 30mL/hour</td>
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<tr>
<td></td>
<td>• Respiratory depression ≤ 12 breaths/minute</td>
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<tr>
<td></td>
<td>• Respiratory arrest</td>
</tr>
<tr>
<td></td>
<td>□ <strong>Treatment of Toxicity:</strong> Calcium Gluconate 1g in 10mL IV over 3 to 10 minutes</td>
</tr>
</tbody>
</table>
Annex 2:

<table>
<thead>
<tr>
<th>Drug</th>
<th>DIAZOXIDE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Presentation</td>
<td>Ampoule: 15mg/mL</td>
</tr>
<tr>
<td>Dose</td>
<td><strong>Rapid IV Bolus:</strong> 15 - 45mg (1 - 3mL) given over 5 to 10 minutes, maximum of 300mg</td>
</tr>
<tr>
<td>Administration</td>
<td>Inject undiluted into a peripheral vein.</td>
</tr>
</tbody>
</table>
| Clinical guideline links | [Hypertension in Pregnancy](#)  
[Hypertension in Pregnancy - Medical Management](#) |
| Other guidelines or policy links | [Standard Procedures for Reconstitution and Administration of Intravenous Medications (Adults)](#) |
| Comments      | ☐  May be given as a single dose or divided into smaller doses and given every 5 to 15 minutes |
## Annex 3: HYDRALAZINE

<table>
<thead>
<tr>
<th>Drug</th>
<th>Presentation</th>
<th>Dose</th>
<th>Administration</th>
<th>Clinical guideline links</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Ampoule: 20mg Powder for Reconstitution</td>
<td>ACUTE TREATMENT OF SEVERE HYPERTENSION</td>
<td>IV: Dissolve hydralazine 20mg powder with 2mL of Sodium Chloride 0.9% in the ampoule. Further dilute to 20 mL with Sodium Chloride 0.9%. This equates to Hydralazine 1mg per mL</td>
<td>Hypertension in Pregnancy - Medical Management</td>
<td>□ Frequent monitoring of BP and continuous fetal monitoring is necessary</td>
</tr>
<tr>
<td></td>
<td>Tablet: 25mg Tablet</td>
<td>IV: Initially: 5 - 10mg by slow injection over 2 minutes (Administer a 5mg first dose if fetal compromise) If the desired BP is not obtained in the 20 to 30 minutes following the first dose notify the medical officer. A further dose of 5 - 10mg of intravenous hydralazine (equating to 5 - 10mL of the diluted mixture) may be ordered to be given slowly over 2 to 4 minutes</td>
<td>IV infusion: Initially: 200 - 300 micrograms/minute; Maintenance: 50 - 150 micrograms/minute</td>
<td>Midwifery Care of the Woman with Severe Pre eclampsia</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>IM: Initially: 5 - 10mg: 5 - 10mg may be repeated after 20 to 30 minutes if desired BP is not obtained; further doses are dependent on BP response</td>
<td>IV Infusion: Dissolve powder in ampoule in 2mL Sodium Chloride 0.9% for injection. Further dilute with Sodium Chloride 0.9% to 20mL</td>
<td>Midwifery care of the woman with severe pre eclampsia during labour</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>= 20mg (20000microgram) in 20mL</td>
<td>= 1mg (1000 microgram) in 1mL</td>
<td>Management of the woman with eclampsia</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>= 1mg (20000microgram) in 20mL</td>
<td>The recommended rate of administration is 50 to 300 microgram/minute.</td>
<td>Hydralazine Antihypertensive Therapy</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Using the above dilution</td>
<td>Labour and birth suite QRG for hydralazine therapy</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>50microgram/minute = 3mL/hr</td>
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<td></td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>150microgram/minute = 9mL/hr</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>200microgram/minute = 12mL/hr</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>300microgram/minute = 18mL/hr</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>IM: Reconstitute 20mg in 2mL water for injection to make a solution of 10mg per mL</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
## Annex 4:

<table>
<thead>
<tr>
<th><strong>Drug</strong></th>
<th><strong>LABETALOL INJECTION</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Presentation</strong></td>
<td>Vial: 100mg/20mL (SAS)</td>
</tr>
<tr>
<td><strong>WNHS Restrictions</strong></td>
<td><strong>SAS Category A</strong> (item requires approval by TGA)</td>
</tr>
</tbody>
</table>
| **Dose** | **IV injection**: 20 - 80mg IV bolus over 2 minutes. Onset of action is approximately 5 minutes. May be repeated after 10 minutes if required. Maximum total dose = 300mg  
**IV infusion**: Infuse with an infusion pump at an initial rate of 2mg/minute then adjust according to the blood pressure response. Maximum total dose = 300mg |
| **Administration** | **IV bolus**: Inject over 2 minutes  
**IV infusion**: Either add 40mL (200mg) to 160mL of a compatible fluid to make a concentration of 1mg/mL  
**OR**  
Add 40mL (200mg) to 250mL of a compatible fluid to make a concentration of approximately 2mg/3mL  
Compatible fluids include glucose 5%, glucose in sodium chloride solutions, Hartmann’s solution, sodium chloride 0.9% |
| **Clinical guideline links** | Hypertension in Pregnancy - Medical Management |
| **Other guidelines or policy links** | WNHS Policy: Administration of parenteral drugs  
Standard Procedures for Reconstitution and Administration of Intravenous drugs |
| **Comments** | - Blood pressure should be monitored during and after completion of the infusion or intravenous injections  
- Patients should always be kept in a supine position during IV drug administration |
### Annex 5:

<table>
<thead>
<tr>
<th>Drug</th>
<th>METHYLDOPA</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Presentation</strong></td>
<td>Tablet: 250mg</td>
</tr>
<tr>
<td><strong>Dose</strong></td>
<td>250 - 750mg three times a day</td>
</tr>
<tr>
<td><strong>Administration</strong></td>
<td>Oral</td>
</tr>
</tbody>
</table>
| **Clinical guideline links** | Hypertension in Pregnancy - Medical Management  
Midwifery care of the woman with Pre eclampsia – Antenatal ward  
Hydralazine Antihypertensive Therapy |
| **Comments** | □ Relatively slow onset of action of 3-6 hours and peak response of 6-9 hours.  
□ The sedating effect of methyldopa is exacerbated by dose increases; increase dosage at night to minimize inconvenience of increased sedation. Monitor blood count and liver function in first 6-12 weeks of treatment. |
## Annex 6:

<table>
<thead>
<tr>
<th>Drug</th>
<th>NIFEDIPINE</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>PRESENTATION</strong></td>
<td>Tablet Immediate Release</td>
</tr>
<tr>
<td></td>
<td>10mg, 20mg</td>
</tr>
<tr>
<td></td>
<td>Tablet SLOW RELEASE (OROS)</td>
</tr>
<tr>
<td></td>
<td>20mg, 30mg</td>
</tr>
<tr>
<td><strong>ACTION</strong></td>
<td>Blocks inward current of calcium into cells causing smooth muscle relaxation</td>
</tr>
<tr>
<td></td>
<td>Potent relaxant of arterial smooth muscle, peripheral vasodilator</td>
</tr>
<tr>
<td><strong>INDICATION</strong></td>
<td>Hypertension</td>
</tr>
<tr>
<td></td>
<td>Tocolysis in preterm labour</td>
</tr>
<tr>
<td><strong>CONTRAINDICATIONS</strong></td>
<td>Cardiogenic shock</td>
</tr>
<tr>
<td></td>
<td>APH, PE, chorioamnionitis, fetal distress, cardiac disease, hypotension,</td>
</tr>
<tr>
<td></td>
<td>Use with betamimetics, such as Salbutamol, Contra-</td>
</tr>
<tr>
<td><strong>PRECAUTIONS</strong></td>
<td>Congestive heart failure – nifedipine may precipitate or worsen heart failure.</td>
</tr>
<tr>
<td></td>
<td>May increase effects of magnesium sulphate and risk of hypotension. Do not given bolus doses of magnesium sulphate to women treated with nifedipine</td>
</tr>
<tr>
<td><strong>Drug Interactions</strong></td>
<td>Enzyme inducing epileptics (carbamazepine, phenytoin, phenobarbitone) – may increase metabolism of nifedipine</td>
</tr>
<tr>
<td><strong>DOSE</strong></td>
<td><strong>Immediate Release Only</strong></td>
</tr>
<tr>
<td></td>
<td><strong>Acute Treatment of Severe Hypertension:</strong></td>
</tr>
<tr>
<td></td>
<td>10mg orally initially, repeat dose if inadequate response after 30 mins</td>
</tr>
<tr>
<td></td>
<td><strong>Post Partum Management of Hypertension</strong></td>
</tr>
<tr>
<td></td>
<td>10mg twice a day increasing to 20mg three times a day</td>
</tr>
<tr>
<td></td>
<td><strong>Tocolysis</strong></td>
</tr>
<tr>
<td></td>
<td>Initially: 20mg orally.</td>
</tr>
<tr>
<td></td>
<td>After 30mins if contractions persist give another 20mg. Repeat dose if required.</td>
</tr>
<tr>
<td></td>
<td>Maintenance: 20mg three times a day for 48 hours after which discussion needs to be with a Consultant.</td>
</tr>
<tr>
<td></td>
<td>Maximum daily dose: 160mg</td>
</tr>
<tr>
<td></td>
<td><strong>Slow Release Only</strong></td>
</tr>
<tr>
<td></td>
<td>Initially 30mg once a day, up to maximum 120mg once daily</td>
</tr>
<tr>
<td><strong>ADMINISTRATION</strong></td>
<td>Crush or chew the first 2 doses to increase rate of absorption.</td>
</tr>
<tr>
<td></td>
<td>Swallow controlled release tablet whole, do not crush or chew</td>
</tr>
<tr>
<td><strong>ADVERSE EFFECTS</strong></td>
<td>Peripheral oedema, flushing, headache</td>
</tr>
<tr>
<td></td>
<td>Hypotension, nausea/vomiting</td>
</tr>
<tr>
<td></td>
<td>Dizziness, tiredness, weakness, light headedness</td>
</tr>
<tr>
<td><strong>COMMENTS</strong></td>
<td>Nifedipine is highly light sensitive. <strong>Do not</strong> break tablets</td>
</tr>
<tr>
<td><strong>PREGNANCY</strong></td>
<td>Safe to use. Care should be taken to avoid maternal hypotension and prevent the possibility of fetal hypoxia</td>
</tr>
<tr>
<td><strong>LACTATION</strong></td>
<td>Safe to use</td>
</tr>
</tbody>
</table>
Bibliography
Management of hypertension during pregnancy ROCOG UK.
Hypertension in pregnancy: diagnosis Hypertension in pregnancy: diagnosis and management, Clinical guideline Published: 25 August 2010
nice.org.uk/guidance/cg107
WHO recommendation for management of Induced Hypertension
ACOG (American College for Obstetrics and Gynecology)
Name of the guideline: Hypertension during pregnancy

First editor: Dr Karima Mayar Amiri / Clinical Guidelines and Protocols
Development Department specialist and Gyne/Osbtetric core group members

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Place of publication: Kabul-Afghanistan

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Series of publication: CG012

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