



**TRAINING MANUAL ON
HYPERTENSIVE DISORDERS
IN PREGNANCY**

Revised 2014

**NATIONAL TECHNICAL COMMITTEE ON
CONFIDENTIAL ENQUIRIES INTO
MATERNAL DEATHS**

Coordinated by:

Division of Family Health Development Division

Ministry of Health, Malaysia



**TRAINING MANUAL ON
HYPERTENSIVE DISORDERS
IN PREGNANCY**
Revised 2014

**NATIONAL TECHNICAL COMMITTEE ON
CONFIDENTIAL ENQUIRIES INTO
MATERNAL DEATHS**

Coordinated by:

Division of Family Health Development Division

Ministry of Health, Malaysia

CONTENTS

Page

NATIONAL TECHNICAL COMMITTEE MEMBERS 2010

EDITORIAL COMMITTEE

INTRODUCTION

OBJECTIVES

SECTION 1 : UNDERSTANDING HYPERTENSIVE DISORDERS (HDP) IN PREGNANCY

SECTION 2 : IDENTIFYING THE PROBLEM

SECTION 3 : MANAGEMENT OF HYPERTENSIVE DISORDERS IN PREGNANCY

- 3.1 Management of Mild Hypertensive Disorders in Pregnancy
- 3.2 Management of Severe Hypertensive Disorders in Pregnancy
- 3.3 Management of Eclampsia
- 3.4 Management of HELLP Syndrome
- 3.5 Anaesthesia and Hypertensive Disorders in Pregnancy
- 3.6 Fetal Surveillance in Hypertensive Disorders in Pregnancy
- 3.7 Discharge and Follow-up Strategies

**SECTION 4 : REFERRAL PROCEDURES AND
MANAGEMENT IN TRANSIT**

SECTION 5 : DIFFERENTIAL DIAGNOSIS

- 5.1 Differential Diagnosis of Convulsions
In Pregnancy
- 5.2 Chronic Hypertension
- 5.3 Case Studies

SECTION 6 : APPENDICES

- 6.1 Basic Facts on Common Drugs Used In
The Management of Hypertension
- 6.2 Fluid Regimes for Patients with HDP
- 6.3 Recommended Techniques in Taking Blood Pressure
- 6.4 Recommended Resuscitation Equipment
- 6.5 Patient Information, Communication and Education (ICE)
- 6.6 Suggested Training Programme

NATIONAL TECHNICAL COMMITTEE MEMBERS 2010

Dato' Dr. Mukudan Krishnan

Senior Consultant O&G
& Head of Department of O&G
Hospital Raja Permaisuri Bainun
Ipoh, Perak

Dato' Dr. Bhupinder Singh a/l Jeswant Singh

Senior Consultant Forensic Pathologist
& Head of Department of Forensic Pathology
Hospital Pulau Pinang

Dato' Dr. Ravindran Jegasothy

Senior Consultant O&G
& Head of Department of O&G
Hospital Kuala Lumpur

Dato' Dr. Sapari bin Satwi

Senior Consultant Physician
& Head of Department of Medicine
Hospital Tengku Ampuan Afzan
Kuantan, Pahang

Dato' Dr. Ghazali b. Ismail

Senior Consultant O&G
& Head of Department of O&G
Hospital Sultan Ismail, Johor

Dr. Muhaini Othman

Senior Consultant Physician
& Head of Department of Medicine
Hospital Serdang, Selangor

Dr. J. Ravichandran

Senior Consultant O&G
& Head of Department of O&G
Hospital Sultanah Aminah, Johor Bahru

Datin Dr. V. Sivasakthi

Senior Consultant Anaesthetist
& Head of Department of Anaesthesiology
and Intensive Care
Hospital Melaka

Dr. Mohd Rohisham bin Zainal Abidin

Senior Consultant Anaesthetist
& Head of Department of Anaesthesiology
and Intensive Care
Hospital Sungai Buloh, Selangor

Dr. Tham Seng Woh

Senior Consultant O&G
& Head of Department of O&G
Hospital Tuanku Jaafar, Seremban, NS

Dr. Mohd. Farouk Abdullah

Senior Consultant O&G
& Head of Department of O&G
Hospital Tengku Ampuan Rahimah
Klang, Selangor

Dr. Soon Ruey

Senior Consultant O&G
& Head of Department of O&G
Hospital Likas, Kota Kinabalu, Sabah

Prof. (Dr.) Mohd. Shukri bin Othman

Deputy Dean & Senior Consultant of O&G
Hospital Universiti Sains Malaysia, Kelantan

Prof. (Dr.) Muhd. Abdul Jamil bin Mohd. Yassin

Deputy Dean & Senior Consultant of O&G
Universiti Kebangsaan Malaysia

Prof. (Dr.) Jamiyah bt Hassan

Deputy Dean & Senior Consultant of O&G
University Malaya Medical Centre

Dr. Nurdiana bte. Abdullah

Family Medicine Specialist
Pejabat Kesihatan Daerah Klang, Selangor

Dr. Daud bin Che Yusof

Family Medicine Specialist
Klinik Kesihatan Rompin
Pejabat Kesihatan Daerah Kuantan, Pahang

Dr. Harris Njoo Suharjono

Senior Consultant O&G
Hospital Umum Sarawak

Dr. Frederick Walter De Rozario

Consultant Physician
Hospital Umum Sarawak, Kuching

Dr. Chua Siew Kee

Senior Consultant Physician
Hospital Sungai Buloh, Selangor

Dr. Mohd Zulkifli bin Mohd Kassim

Consultant O&G
Hospital Sultanah Nur Zahirah,
KualaTerengganu

Dr. Noor Aziah bt. Zainal Abidin

Senior Principal Assistant Director
Medical Development Division
Ministry of Health

Dr. Safurah Jaafar

Director
Family Health Development Division
Ministry of Health

Dr. Mymoon Alias

Deputy Director
Family Health Development Division
Ministry of Health

Dr. Safiah bt. Bahrin

Senior Principal Assistant Director
Family Health Development Division
Ministry of Health

Dr. Rachel Koshy

Senior Principal Assistant Director
Family Health Development Division
Ministry of Health

Pn. Dasimah bt. Ahmad

Matron U 44
Division of Nursing, Ministry of Health

Pn. Tumerah Swandi

Public Health Sister
Family Health Development Division
Ministry of Health

Pn. Siti Dayang

Public Health Sister
Family Health Development Division
Ministry of Health

Editorial Committee

Chairman : **Dato' Dr. Ghazali b. Ismail**
Senior Consultant O&G
& Head of Department of O&G
Hospital Sultan Ismail, Johor

Members :

Dr. J. Ravichandran
Senior Consultant & Head of Department O&G
Hospital Sultanah Aminah, Johor Bahru

Dr. Wan Hamilton bt. Wan Hassan
Senior Consultant & Head of Department O&G
Hospital Serdang, Selangor

Dr. Mohd. Daud bin Che Yusof
Family Medicine Specialist
Klinik Kesihatan Bandar Kuantan
Pejabat Kesihatan Daerah Kuantan, Pahang

Dr Thohiroh Abdul Razak
Consultant Obstetric Anaesthetist
Department of Anaesthesiology and Intensive Care,
Hospital Kuala Lumpur

Dr. Safiah bt. Bahrin
Senior Principal Assistant Director
Family Health Development Division, MOH

Dr. Majdah bt. Mohamed
Senior Principal Assistant Director
Family Health Development Division, MOH

Dr. Zul Azuin bt Zulkifli
Principal Assistant Director
Family Health Development Division, MOH

Dr. Anis Iryani bt. Safiee
Assistant Director
Family Health Development Division, MOH

Puan Noor Aini bt Karimon
Public Health Matron
Family Health Development Division, MOH

Pn. Tumerah Swandi
Public Health Sister
Family Health Development Division, MOH

Puan Suzana bt Kipli
Public Health Sister, Family Health Development Division

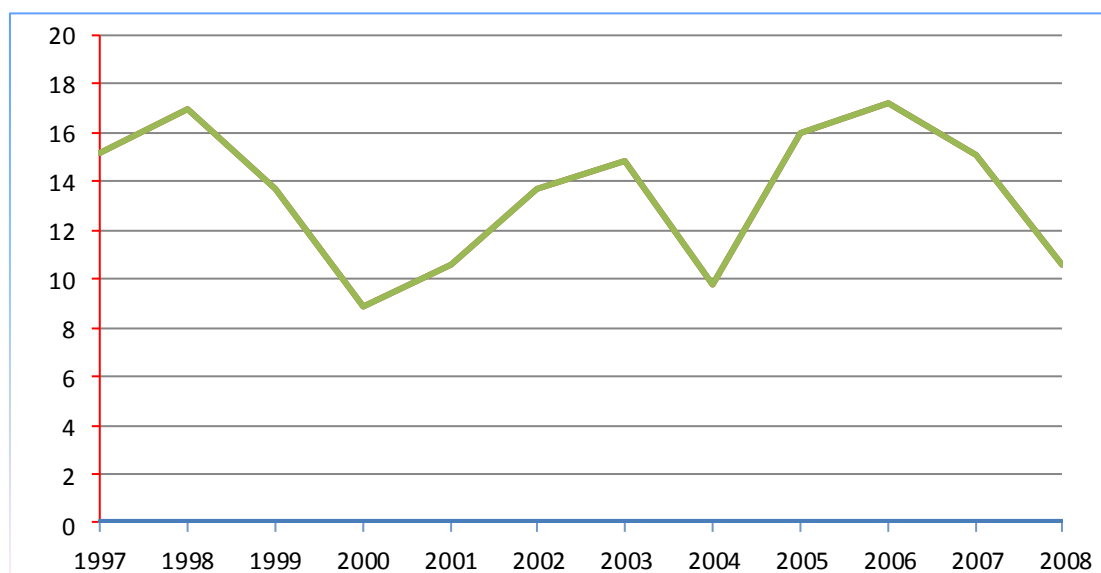
Introduction

Improving maternal health and reducing maternal mortality have been key concerns of several international summits and conferences since the late 1980s. In developing countries: preeclampsia/eclampsia impact 4.4% of all deliveries¹ and may be as high as 18% in some settings in Africa². If the rate of life threatening eclamptic convulsions (0.1% of all deliveries) is applied to all deliveries from countries considered to be the least developed, 50,000 cases of women experiencing this serious complication can be expected each year.

In the Global Burden Disease report 1990 by WHO, hypertensive disorders of pregnancy ranked 75th in terms of DALYs and were responsible for 6% of the burden of all maternal conditions. It was estimated that deaths due to hypertensive disorders of pregnancy represented 13% of all maternal deaths³.

Hypertensive pregnancy disorders complicate 10% of all pregnancies and cover a spectrum of conditions, namely preeclampsia, eclampsia, chronic and gestational. These disorders occur frequently among pregnant woman and are important contributors to maternal and perinatal mortality and morbidity worldwide. From 1997-2008, there were 238 cases of death related to HDP with an average incidence of 13.56% per year (excluding fortuitous deaths). There was no sign of declining trend over the stipulated period of time with the highest mortality incidence reported in 2006 and lowest in 2000⁴.

Graph 1: Percentage of maternal deaths related to Hypertensive Disorders in Pregnancy



Hypertensive disorders in pregnancy (HDP) is not a preventable condition but early recognition and prompt treatment can prevent the development of both maternal and fetal morbidity and mortality.

Eclampsia remain a severe complication of pre-eclampsia and is associated with increased maternal and fetal morbidity and mortality. Following the Confidential Enquiry into Maternal Deaths of 2006/2008 ⁴, out of 70 cases who died of HDP, 41 were due to eclampsia and the remaining died due to its complications eg. CVA, multi-organ failure.

Ministry of Health uses eclampsia as one of the Key Performance Indicators (KPI) which reflect the quality management of HDP ⁵. Since then, various strategies were adopted by the Ministry of Health to reduce morbidity and mortality.

Table 1: Causes of Maternal Deaths 2001-2006

CAUSES	2001 – 2006	Percentage
	Cases	%
Obstetric Embolism	138	29.54
Associated Medical Conditions	116	17.26
Postpartum Haemorrhage	106	15.76
Hypertensive Disorders in Pregnancy)	86	12.79
Obstetric Trauma	62	9.23
Abortion	39	5.50
Puerperal Sepsis	23	3.42
Antepartum Haemorrhage	18	2.77
Ectopic	14	2.08
Unspecified Complications of Pregnancy & Puerperium	20	2.97
Associated with Anaesthesia	2	0.30
Others	50	7.44
TOTAL	672	100%

When factors like age and parity are considered, more than a third (37.5%) of those who died of HDP were aged 20-24 years. Of the HDP cases studied⁴ the rate of occurrence of eclampsia was higher among the primigravid patients (7%) compared to the parous mothers (2.3%).

Prenatal counselling, early booking of pregnancy and quality antenatal care for the patient 'at risk' are the cornerstone to the appropriate management. Some of the remediable factors identified in previous audits³ point towards failure to recognise severity of the ailment, inadequate and a lack of understanding of HDP among health providers and the community at large.

Women who have been diagnosed with either preeclampsia or gestational hypertension are at increased risk of subsequent cardiovascular morbidity including hypertension and coronary heart disease. A recent systematic review and meta-analysis⁹ determined that the relative risks for hypertension were 3.70 after 14 years follow-up, for ischemic heart disease 2.16 after 12 years, for stroke 1.81 after 10 years, and for venous thromboembolism 1.87 after 5 years. Overall mortality after preeclampsia was increased 1.5 fold after 14 years. These associations are likely to reflect a common cause for preeclampsia and cardiovascular disease, or an effect of preeclampsia on vascular disease development, or both. It is reasonable to counsel patients who develop hypertension 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 preeclampsia or hypertension in pregnancy have an annual blood pressure check and regular (5 yearly or more frequent if indicated) assessment of other cardiovascular risk factors including serum lipids and blood glucose.

This 2nd edition teaching module is produced to supplement existing teaching materials available to health providers both in the primary care setting and at referral centres. It has been developed through discussion with clinicians and health care providers and reference is made to current opinions in the management of HDP in developing countries. It is primarily intended to be used for in-service training for health providers involved in the management of HDP. The manual is not intended to replace standard textbook teaching which will deal with other aspects of pregnancy and childcare.

There are 6 Sections in the manual. These include basic terminology used in HDP, management of HDP at home, domiciliary practice, health centres and in tertiary centres. While it is important to apply theory in practice, trainers are encouraged to use various teaching methods to suit local practice. Trainers are also expected to include pre and post-test questions to evaluate understanding of HDP.

References:

1. *"Revised 1990 Estimates of Maternal Mortality: A New Approach by WHO and UNICED". World Health Organization, Geneva, 1996.*
2. *Villar J, Betran AP, Gulmezoglu M. Epidemiological basis for the planning of maternal health services. WHO/RHR 2001.*
3. *Carmen Dolea, Carla AboZahr : Global burden of HDP in the year 2000 :Evidence and Policy WHO Geneva 2003*
4. *CEMD report 2006-2008 : Analysis on death related to HDP (unpublished)*
5. *Hypertensive disorders of Pregnancy 1985-1997. Quality Assurance Programme of the Family Health Division, Ministry of Health, 1997*
6. *Maharaj B, Moodley J. Management of hypertension in pregnancy. Cont. Med. Educ 1994; 12:1581-1589*
7. *CEMD report 1997-2005*
8. *Maternal Mortality Report 2005 :WHO*
9. *Bellamy L, Casas JP, Hingorani AD, Williams DJ. Pre-eclampsia and risk of cardiovascular disease and cancer in later life: systematic review and meta-analysis. BMJ. 2007;335(7627):974*

Objectives of The Training Module

General Objectives

To develop a comprehensive training manual on the management of hypertensive disorders in pregnancy to all health care providers.

Specific Objectives

- To provide adequate knowledge and skills in the management of hypertensive disorders in pregnancy for all health care providers
- To develop a simplified and standardised training manual in the management of hypertensive disorders at hospitals, health clinics and at home
- To ensure that all health care providers are able to understand hypertensive disorders in pregnancy and its associated complications so as to enable them to initiate an appropriate management plan.

SECTION 1

**UNDERSTANDING
HYPERTENSIVE DISORDERS
IN PREGNANCY**

SECTION 1

UNDERSTANDING HYPERTENSIVE DISORDERS IN PREGNANCY

1. AETIOLOGY OF PRE-ECLAMPSIA

HDP constitutes a group of diseases of distinct aetiology of which **hypertension** is the chief clinical manifestation. Pregnant women who are hypertensive will generally fall into two groups: normotensive women who develop the pre-eclampsia syndrome (PE), characterised by hypertension and proteinuria with or without oedema and women with chronic hypertension who are at a higher risk for superimposed PE. Pre-eclampsia is the more serious form of hypertension in pregnancy. The disease is present from the second half of pregnancy as a result of abnormalities of placentation. The cause of the latter is not known though it may be due to altered genetic and immunologic influences. Impaired trophoblastic invasion appears to be universally present in maternal spiral arterioles with poor trophoblastic perfusion, endothelial injury, altered endothelial permeability, utero-placental ischemia, with resultant activation of coagulation. Impairment of vasopressor function is usually present. PE is a syndrome of generalised endothelial dysfunction and the complications are associated with the vascular system. Untreated, complications of generalised vasospasm result in tissue hypoxia and organ failure.

2. TERMINOLOGY

No consensus exists on the definition of HDP, which has resulted in parallel classification in use around the world^{1,2}. Hypertension has been **defined** as an **absolute blood threshold**, as a **rise in blood pressure** or as a **combination of both**.

2.1 Definition of Hypertension:

Hypertension in pregnancy is defined as:

- BP of 140/90 mm Hg taken after a period of rest on two occasions.

OR

Rise of systolic blood pressure (SBP) of 30 mmHg and /or a rise in diastolic blood pressure (DBP) of 15 mmHg³ compared to pre pregnancy levels.

Measurement of blood pressure is important for accurate prediction of hypertension. This is dealt with in Section 6.3. Although the fifth Korotkoff sound is far more reliable, it may be significantly lower than Korotkoff IV in pregnancy. If there is confusion, both are best recorded.⁴

2.2 Classification of Hypertension in Pregnancy

A clinical approach to classification is best adopted taking into consideration that HDP is a multisystem disease. Although PE is more commonly seen primigravida mothers, the condition is also detected in the multiparous population. Patient factor including late booking and lack of information on pre-morbid status together with a lack of data made available to 'end-point' health care providers in the combined care approach afforded to pregnancy management in Malaysia may make it difficult to place HDP into particular categories. Some classifications include an '**Unknown Hypertension**' group. The latter is excluded in this manual as too many will fall into this category.

Classification of HDP used in this manual is:

i) **Pregnancy Induced Hypertension (PIH)**

Hypertension after the 20th week of pregnancy in a previously normotensive woman. It may be associated with proteinuria. The condition is expected to return to normal after puerperium.

- | | | |
|-----------------------------------|---|-------------------------|
| (a) Gestational Hypertension (GH) | - | PIH without proteinuria |
| (b) Pre-eclampsia (PE) | - | PIH with proteinuria |
| (c) Eclampsia | - | PIH with convulsions |

HELLP syndrome is a severe form of PE manifested by Haemolysis, Elevated Liver Enzymes and Low Platelets.

ii) **Chronic Hypertension** (includes essential and secondary hypertension)

iii) **Chronic Hypertension with superimposed Pre-eclampsia**

- **Chronic Hypertension** is defined as the presence of hypertension of at least 140/90 before 20 weeks of pregnancy OR beyond 6 weeks postpartum.
- **Chronic Hypertension with superimposed PE** refers to the development of PE in women who have pre-existing hypertension. Criteria used should include worsening hypertension, proteinuria and non-dependent oedema.
- **Essential Hypertension and Secondary Hypertension** are best categorised under chronic hypertension as pregnancy outcomes in all these categories are similar.^{5,6}
- **Proteinuria**
Urinary tract infection must be excluded. **Proteinuria** is defined as 300 mg/24 hours urine collection or 1 gm/L or more in two randomly collected urine samples 6 hours apart. Semiquantitative assessment of proteinuria using dipstix is convenient and has collaborated well with maternal outcome.⁽⁷⁾

Quantifying Proteinuria			
Dipstix	+	-	0.3 gm/L
(Albustix)	++	-	1.0 gm/L
	+++	-	3.0 gm/L
	++++	-	> 20 gm/L

(+) proteinuria carries a high false positive rate. Therefore the presence of proteinuria should be confirmed by measuring 24 hour protein excretion or a protein:creatinine ratio.⁸

- **Oedema**

Oedema is commonly seen in pregnancy and may not be a usual sign for early detection of PIH. In **severe PIH**, there is generalised accumulation of fluid largely due to endothelial damage resulting in accumulation of fluid evidenced by pitting oedema following 12 hours of recumbant bedrest. A weight gain of 1 kg within a week may point to increasing severity of PIH especially in the presence of proteinuria.

- **Severity of HDP**

HDP and related diseases should be classified further according to severity of disease. Eclampsia is easily identified by the presence of convulsion in a patient who has all the clinical signs of HDP. Severity of HDP would include:

(a) **Mild**

Characterised by BP >140/90 mmHg without albuminuria

OR a rise in SBP 30 mmHg or a DBP 15 mmHg

(b) **Severe**

Severe HDP is characterised by progressive deterioration in both maternal and foetal condition. It is characterised by:

- i) SBP >160 mmHg or DBP >110 mmHg on two occasions 6 hours apart
- ii) Proteinuria of (3+) or > 3 gm/L
- iii) Oliguria (< 400 ml/24 hours)
- iv) Headache
- v) Cerebral or visual disturbances
- vi) Epigastric pain
- vii) Hyper-reflexia
- viii) Pulmonary Oedema
- ix) Impaired liver function tests
- x) Increased serum creatinine (> 1.2 mg/dl)
- xi) Retinal haemorrhage, exudates or papilloedema
- xii) Thrombocytopenia
- xiii) IUGR

3. IDENTIFYING THE MOTHER AT RISK

HDP cannot be prevented. However, a certain subset of pregnant women are at risk of developing HDP. Identifying this group early, prenatally and during early booking will assist health providers to keep these patients under surveillance. The risk factors include:

- i) Maternal age <20 years and >35 years
- ii) Nulliparity
- iii) Previous history of HDP
- iv) Multiple gestation
- v) Polyhydramnios
- vi) Non-immune foetal hydrops
- vii) Underlying renal disease
- viii) Chronic hypertension
- ix) Diabetes mellitus
- x) Gestational Trophoblastic Disease (Molar pregnancy)
- xi) Low socio-economic group
- xii) Pregnancies with different partners
- xiii) Excessive weight gain
- xiv) Rh incompatibility.

4. COMPLICATIONS OF HDP

Pregnancy induced hypertension is a multisystem disease with evidence of universal vasoconstriction and vasospasm. Untreated, organ dysfunction and organ failure will be the sequelae. The commonly recognised complications are tabulated below.

Complications of Pre-Eclampsia

Central Nervous System

- Cerebral Oedema
- Cerebral Haemorrhage
- Transient Cortical blindness
- Serous retinal detachment

Cardiovascular System

- Hypertension
- Acute Pulmonary Oedema
- Cardiac Failure

Pulmonary System

- Acute Pulmonary Oedema
- Aspiration Pneumonia

Liver

- Congestion
- Haemorrhage
- Infarction
- Rupture

Kidney

- Glomerulendotheliosis
- Nephrotic Syndrome
- Acute Renal Failure

Blood

- Thrombocytopenia
- Microangiopathic haemolytic anemia
- Disseminated intravascular coagulation

Uterus, Skin & Mucosa

- Placental Abruption
- Oedema
- Petechia, ecchymosis
- Laryngeal oedema

Foetus

- Intrauterine growth retardation
- Prematurity
- Intrauterine death

References:

1. Davey DA, Mac Gillivray I. *The classification and definition of the hypertensive disorders of pregnancy. Am J Obstet Gynecol 1988; 158: 892-898*
2. *Australian Society for the Study of Hypertension in Pregnancy in : Pregnancy: Consensus statement. Med J Aust 1993; 158: 700-702.*
3. Douglas KA, Redman ML *Eclampsia in the United Kingdom BMJ 1994; 309: 1395-1400*
4. Grant JM: *Editorial: Defining Pre-Eclampsia: Br J Obstet Gynecol 1999; 106: vii-ix.*
5. Sibai BM *Management of Pre-eclampsia in Clinics in Perinatology 1991; 18: 793-808.*
6. Zuspan FP, MacGilivray I, Grant N et al, *National High Blood Pressure Education Program Working Group Report on High Blood Pressure in Pregnancy. Am J Obstet Gynecol 1999; 163: 1689-1712).*
7. North RA, Taylor RS, Schellenberg J C. *Evaluation of a definition of pre-eclampsia Br J Obstet & Gynecol 1999; 106: 767-773*
8. Saudan PJ, Brown MA Farrel KC, et al *Improved methods of assessing proteinuria in hypertensive pregnancy. Br J Obstet Gynecol 1997; 104: 1159-1164.*

SECTION 2

IDENTIFYING THE PROBLEM

SECTION 2

IDENTIFYING THE PROBLEM

1. EARLY IDENTIFICATION OF HYPERTENSIVE DISORDERS IN PREGNANCY

The early identification of hypertensive disorders in pregnancy (HDP) is an important strategy to prevent its complications.

The aim of this section is to enable health personnel to acquire the art of diagnosis and differential diagnosis in respect to HDP. Health personnel must be able to carry out:

- Early identification of high risk patient at community level
- Classification of HDP and recognition of its severity

1.1. Identification of high risk patient at community level

1.1.1 History and complaints

- Family history of hypertension
- History of pregnancy induced hypertension
- Primigravida
- Associated conditions
 - multiple pregnancy
 - diabetes mellitus
 - renal disease
 - SLE/APS (Anti-Phospholipid Syndrome)
 - obesity (>80 kg. BMI >27)
 - maternal age
 - pre existing chronic hypertension
- Symptoms:
 - headache
 - visual disturbance
 - nausea and vomiting
 - epigastric pain

1.1.2. Physical examination

- Excessive weight gain (> 1 kg per week)
- Oedema in the face and abdomen and/or non-dependant oedema
- Proteinuria
- Obesity (Body Mass Index >27 or 80 kg.)
- Abdominal examination - polyhydramnios
- multiple pregnancy

Blood pressure:

- BP of 140/90 mmHg taken on 2 occasions 6 hours apart
- If baseline BP is known :
 - ⇒ Increase in systolic BP by 30 mmHg
 - ⇒ Increase in diastolic BP by 15 mmHg

1.2 Recognition of Severity and Classification of HDP

All patients should be appropriately coded according to the severity of the condition based on the Ministry of Health Guideline on Management of High Risk Cases in Pregnancy: 2nd Edition, 1991

- **RED CODE**

Mild pre-eclampsia and **more** than 36 weeks gestation
Severe pre-eclampsia
Eclampsia

- **YELLOW CODE**

Mild pre-eclampsia and **less** than 36 weeks gestation
Proteinuria

2. MANAGEMENT OF PATIENT AT COMMUNITY LEVEL

2.1 Issues related to management of HDP at community Level

- Lack of awareness about HDP
- Lack of awareness on the importance of antenatal care

- Socio-economic factors
 - low socio-economic status
 - geographical barriers
 - transportation
 - lack of family support
- Socio-cultural factors
 - adverse traditional beliefs beliefs and taboos
 - influence of traditional healers and TBA's

2.2 Overcoming The Problems

- Educating patient, family and community through various modalities such as health talks in the clinic, pamphlets, posters etc.
- Counselling, group discussion about danger signs and symptoms of HDP and the importance of antenatal care

Information to be given to patient

Notify the doctor or nurse if any one or more of the following occur:

- Abnormal increase in weight gain
- Oedema or swelling of hands, feet, and/or face e.g. puffy eyes, tight rings on fingers, tight shoes
- Epigastric pain
- Nausea and vomiting
- Headache
- Blurred vision

- Establish rapport between the health personnel and community
 - health personnel to participate in community activities e.g. be a member of the village JKKK (Jawatankuasa Kemajuan dan Keselamatan Kampung)
 - smart partnership with NGO, TBA's and other care providers in the community
 - organise open clinic day
 - encourage family participation in care of patient (family support) – when patient is hospitalised or indisposed)
 - recognise people in the community who are able to provide transportation when required
 - employer support
 - home visiting for follow-up care and support

3. MANAGEMENT OF PROVIDER PROBLEM

3.1 Issues Related to Management of HDP at Provider Level

- failure to recognize disease and severity of disease
- delay in referral
- problems in transfer of patient

3.2 Overcoming the problems:

- Educate all category of staff through existing in-service training strategies and telemedicine
- Provide refresher courses yearly
- Effective supervision
- Provide checklist of symptoms for front-liners
- Early consultation with referral hospitals by telephone, fax or teleconsultation (telemedicine)
- Recognize those who are likely to give resistance for referral eq.TBA, husband, father, mother
- Explore reasons for resistance (if present) - counsel and give reassurance
- Involve other Medical staff in times of emergency for assistance
- Develop strategies to transfer ill patient in cases of emergency

Checklist for Identifying Severity of HDP

1. Blood Pressure:

- Initial reading after a period of rest
- Ideally at 6 hours later
- Increase in previous or pre-pregnancy BP (mmHg):
 - systolic
 - diastolic

2. Weight Gain (kg.)

- 1 week
- 2 weeks
- 3 weeks
- 1 month

3. Oedema:

- Legs
 - ankle
 - pretibial
- abdomen
- Hands
- Generalised

4. Proteinuria (dip stick)

- Trace
- +
- ++
- +++
- Solid (++++)

5. Patient's complaints:

- epigastric pain
- nausea
- vomiting
- headache
- blurring of vision

1. Interpretation

- i.
- ii.
- iii.

Name of Health Provider : _____ (Date/time)

4. INFORMATION, COMMUNICATION AND EDUCATION (ICE)

- 4.1 Healthcare providers should acquire knowledge and information through CME, conferences, peer review, audit, QA and supervision by seniors
- 4.2 Community should be reached through electronic and printed media, radio talk, pamphlet, exhibitions
- 4.3 Health provider should make available adequate supply of patient information leaflets (Section 6.5)

EDITOR'S COMMENTS

PREVENTING PREECLAMPSIA

i) **Dietary Sodium Restriction**

Although strict restriction of dietary sodium has been shown to be effective in long term management of chronic hypertension in men, there is no convincing evidence that salt restriction has any role in preventing or treating HDP.

ii) **Bed Rest**

Bed rest, either at home or in hospital has not been shown conclusively to prevent the development or alter the course of protenuric hypertension, to decrease the risk of preterm delivery or to reduce perinatal mortality.¹ In our context, whilst the above statements are true, limitation in household chores of normal activity must be emphasized in all women with HDP, if ambulatory care is instituted.

iii) **Dietary Supplementation**

***Eicosanoid** metabolism is a common element that is disordered in pre-eclampsia. Dietary supplementation of fish oil (rich in omega-3 polyunsaturated fatty acids) was considered to correct thromboxane/prostaglandin ratios and consequently influence vascular sensitivity. Although there are favourable reports to advising fish oil supplementation, it is unclear whether they decrease incidence of pre-eclampsia.²*

***Zinc and magnesium supplements** have also been considered but the evidence is not strong for their prescription*

Calcium Supplementation

Extracellular ionised calcium concentrations are crucial for production of endothelial nitric oxide (NO) and regulation of vascular tone. Decreased NO is implicated in pathophysiology of pre-eclampsia. Meta-analysis of initial trials in humans suggest an inverse relationship of calcium intake and maternal blood pressure, the effect being more pronounced in nulliparous women. Initial trials on dietary calcium have been shown to substantially decrease HDP during pregnancy. (OR 0.34%, (0.22-0.54)).^{3,4} Further studies are required to identify high risk groups who may benefit from calcium supplementation.

iv) **Diuretics**

There is no evidence to show diuretics are beneficial in preventing HDP. Diuretics should not be used in pregnancy as they can reduce renal and placental perfusion.

v) **Low-dose Aspirin**

Aspirin blocks the production of certain eicosanoids by inhibition of action of cyclo-oxygenase (COX). Low dose aspirin has been used to prevent pre-eclampsia at a dose of around 60 mg/day. Early clinical trials suggested that aspirin at this dose prevented pre-eclampsia with no risk to mother and fetus. More recent studies do not justify the use of low-dose aspirin for the prevention of PE in a low risk population. In high risk populations, aspirin may reduce the overall risk by about 13% (a reduction that is of questionable clinical importance)^{5,6}

References:

1. Crowther C, Chalmers. *Bed rest and hospitalization during pregnancy. In Chalmers 1, Enkin M, Keirse MJN eds. Effective Care in Pregnancy and Childbirth, Oxford : Oxford University Press, 1989 ; 624-632*
2. Adair CD, Sanchez-Ramas L, Briones DL et al. *The effect of high dietary n-3 fatty acid supplementation on angiotension 2 pressor response in human pregnancy Am J Obstet Gynecol 1996, 175: 688-691*
3. Bucher H, Guyatt GH, Cook RJ et al *Effect of calcium supplementation on pregnancy-induced hypertension and pre-eclampsia: A meta-analysis of randomised control trials. JAMA 1996; 275: 1113-1117*
4. Carolli G, Duley L, Belzian JM et al. *Calcium supplemetation during pregnancy: a systemic review of randomised controlled trials. Br J Obstet Gynecol 1994; 101: 753-758*
5. Caritis S, Sibai BM, Hauth j et al. *Low dose aspirin to prevent pre-eclampsia in women at high risk. N Engl J Med 1998; 338: 701-705*
6. CLASP (*Collaborative Low dose Aspirin Study in Pregnancy. A randomised trial of low-dose apsirin for the prevention and treatment of pre-eclampsia among 9,364 pregnant women. Lancet 1994; 343: 619-629.*

SECTION 3

**MANAGEMENT OF HYPERTENSIVE
DISORDERS IN PREGNANCY**

Section 3.1

Management of Mild Hypertensive Disorders in Pregnancy

1. INTRODUCTION

Being a multi-organ disease, the progress of HDP is often unpredictable and could lead to rapid deterioration in maternal and fetal condition. In view of this, the management of mild HDP requires continuous and close surveillance.

A decision must be made at the time of diagnosis whether to manage mild HDP as an out-patient or as an in-patient. The RCOG Consensus ⁽¹⁾ is of the opinion that mild HDP in the absence of proteinuria may be managed on an outpatient basis. It has been shown that this could reduce 60-80% of hospital stay, with no detrimental effects on maternal or fetal care. However, in our context we have to look at the cases on an individual basis, in terms of logistics, socio-economic factors and patient's educational level.

2. GOAL OF MANAGEMENT OF MILD HDP

In mild HDP, the aim is to prolong the pregnancy to near term as possible provided there are no evidence of maternal complications or fetal compromised (fetal distress, FGR/oligohydramnios)

3. AMBULATORY CARE (OUT-PATIENT MANAGEMENT)

a) Criteria for selection of patient for ambulatory care:

- B/P \geq 140/90 mmHg but less than 160/100mmHg
- NO proteinuria
- NO signs/symptoms of impending eclampsia
- NO excessive weight gain
- No signs of intrauterine growth retardation
- Normal biochemical investigation

b) Antenatal Care

- Mild HDP can be managed in health clinics.
- Every patient should be monitored to detect any deterioration in maternal and fetal condition
- The frequency of each visit should be individualized depending whether patient requires medication or in the presence of other complication
- In patient who do not require medication and absence of maternal or fetal complications, the patient should be attending the normal antenatal follow up
- Patient should be counsel with regards her condition, management option and need for regular antenatal care.
- *Maternal and fetal monitoring and surveillance is the mainstay of management of HDP.*

During visit to the doctor, the following should be monitored;

All decisions must be documented in the patient's antenatal card.

Maternal Surveillance

- Blood Pressure,
- Urine for protein,
- Weight gain
- Signs/symptoms of impending eclampsia should be elicited.
- Biochemical Investigation:
 - Platelet count
 - Heamatocrit
 - Serum Uric Acid
 - Serum Creatinine
 - 24 Urine Protein (if necessary)

Fetal Surveillance

- Fundal Height
- Fetal Heart
- Fetal Movement (Fetal Kick Chart)
- Serial Ultrasound - (if available)
 - growth parameters (BPD/FL/AC/HC)
 - Amniotic Fluid Index. (AFI)

c) Antihypertensive Therapy

Not all mild HDP require antihypertensive treatment. A majority of them may benefit from adequate rest. Patients with BP of 140/90mmHg, **without any complications** may not require antihypertensive treatment. Antihypertensive treatment may be considered when B/P is persistently above DBP 100 mmHg.²

d) Referral to Hospital with Specialist

- *At any time, referral should be made when there is any deviation observed from the above criteria.*
- *All cases of mild HDP must deliver in hospital.*

4. IN-PATIENT MANAGEMENT

(a) Indications for hospitalization

Generally the indication for in-patient management is for those who fail ambulatory care (out-patient) management. The reasons for admission are:-

- symptomatic patients
- maternal or fetal complications
- persistent diastolic blood pressure > 100 mmHg or systolic > 160 mm Hg for stabilization
- abnormal biochemical PE profile
- presence of severe proteinuria > 2+

(b) Management in the ward

All observations should be documented in HDP (PE) chart, which consist of the following:-

- Four hourly Blood Pressure and Pulse Rate monitoring
- Daily urine for protein
- Weekly weight
- Sign and symptoms of impending eclampsia.
- Investigations eg: FBC, Renal Profile, serum uric acid
- Fetal monitoring eg: fundal height, fetal movement, CTG

(c) Antihypertensive therapy

A decision to start antihypertensive therapy and the selection of the agent should be individualized. It should be based on an assessment of the relative risks and benefits for the mother and her fetus.

Antihypertensive therapy should start when the DBP is ≥ 100 mmHg, and or systolic BP is ≥ 160 mmHg

i) Medication used

In mild HDP, the following antihypertensive drugs may be considered:

- Alpha-Methyl dopa or
- Labetolol (see Section 6.1)

Start with monotherapy and increase gradually till maximum dose. Consult the specialist if there is a need to add the 2nd drug medication.

ii) Aim of treatment

The aim of treatment is to maintain a DBP around 90 -100 mmHg to:

- minimize the risk to the mother from events such as cerebral vasculo-accident , cardiac failure and placental abruption etc.
- avoid placental hypoperfusion which may lead to IUGR, fetal hypoxia and Intrauterine Death.

5. DISCHARGE AND FOLLOW-UP

Discharge may be considered when BP is stabilized (diastolic pressure between 90-100 mmHg) with no complications. A clear plan of management should be documented in the patient's antenatal card. The patient should be counselled on the importance of compliance to medication, adequate rest, frequent follow-up and to observe for warning signs of PE. The patient should be advised for hospital delivery. The nearest health facility should be notified for continued care by way of telephone (or fax).

6. TIMING OF DELIVERY

In the absence of maternal and fetal complication, pregnancy should not be allowed beyond dates i.e before 40 weeks. If at anytime the maternal and fetal condition is compromised, early delivery is mandatory and appropriate corticosteroid usage is necessary.

6.1 Intra-partum management

Mild HDP may become severe during labour, hence close vigilance monitoring is essential.

Maternal surveillance:

- Labour should be monitored using Partogram.
- Antihypertensives should be continued if patient is on such treatment.
- Intravenous line should be set-up.
- I.V. Hydrallazine should be considered if DBP is more than 110mmHg
- Adequate analgesia is essential
- USE only Syntocinon during third stage of labour
- Fluid regime therapy (Refer Section 6.2)
- Assist second stage if indicated.

Fetal surveillance:

- Auscultation of fetal heart rate every 15 minutes
- Institute electronic monitoring (cardiotocography) continuously or intermittently as indicated.

6.2 Immediate post-partum period

The blood pressure may settle after delivery, however the patient is still at risk to develop complications. Therefore, continuation of maternal monitoring is essential.

The patient should be observed in labour suite about one hour. If the BP remain high (diastolic > 100 or systolic > 160) then such patient should be observed at Obstetric HDU before transfer to the high risk post-natal ward.

6.3 First 24 hours after delivery

- Monitor B/P, P/R every 4 hours.
- Antihypertensive drugs should be continued after delivery as dictated by blood pressure.
- If the diastolic BP < 90 mmHg, the medication can be withhold.
- While rest is encouraged, patient should be assisted for early mobilization.
- Encourage and assist patient to breast-feed her baby.

6.4 After 24 hours post-partum

- Continue antihypertensive agent aiming for DBP between 90-100 mmHg.
- Counsel patient on the importance of:
 - Compliance to medication and follow up.
 - Symptom or signs of impending eclampsia
 - Effective contraception.
- Discharge criteria (refer Section 3.7)
- Upon discharge, a clear summary and plan of management should be written in the patient's antenatal card.
- The nearest health clinic should be notified either by phone, verbally, written letter, fax or through family members.

References:

1. *Collaborative Low-dose Aspirin Study in Pregnancy (CLASP) Collaborative Group (1994). CLASP: A randomized Trial of Low Dose Aspirin For The Prevention and Treatment of Pre-eclampsia Among 9364 pregnant women. Lancet 1994; 343: 619-29.*
2. *Redman C.W.G (1991). Drugs, hypertension and pregnancy. Progress in Obstetrics and Gynaecology Volume 9, Ed: John Studd, Churchill Livingstone, Medical Division of Longman Group UK Limited., page 83-84*
3. *Green Top Guidelines No: 10(A March 2006 RCOG)*
4. *Malaysian CPG : Management of Hypertension – 3rd Edition February 2008 MOH/P/PAK/156.08 (GU)*

Section 3.2

Management of Severe HDP

1. INTRODUCTION

Severe hypertension in pregnancy is an obstetric emergency state and generally acknowledged that it should be lowered promptly, albeit carefully, to prevent cerebral hemorrhage and hypertensive encephalopathy¹. This degree of hypertension therefore requires urgent assessment and management. It is important to acknowledge that systolic as well as diastolic hypertension increases the risk of cerebral hemorrhage.

Hypertension continues to be an important association with maternal and fetal morbidity and mortality. In almost all the Reports of the Confidential Enquiry into Maternal Deaths in Malaysia, hypertensive disease in pregnancy (HDP) has been the second most common condition associated with maternal deaths². The presence of substandard care or remediable clinical factors in the management of these cases reinforces the need for a multi-disciplinary approach and also the involvement of consultants in patient care.

2. DEFINITION

Severe HDP is characterized by progressive deterioration in both maternal and fetal conditions. The characteristics are:

- SBP \geq 170 mmHg or DBP \geq 110 mmHg on two occasions 6 hours apart
- Proteinuria of 3+ or $>$ 3 gm/L
- Oliguria ($<$ 400 ml/24 hours)
- Headache
- Cerebral or visual disturbances
- Epigastric pain
- Hyper-reflexia
- Pulmonary oedema
- Impaired liver function tests
- Increased serum creatinine ($>$ 1.2 mg/dl)
- Retinal haemorrhage, exudates or papilloedema
- Thrombocytopenia
- Impairment of fetal growth (IUGR)

3. MANAGEMENT

The aim of the management of severe HDP is to prevent a cerebro-vascular accident to the mother whilst trying to achieve a clinically useful prolongation of the pregnancy². This is because it is also aimed at delivering a live baby as mature as possible. Pre-eclampsia when diagnosed at term, mandates delivery as there is no advantage to either the fetus or mother in prolonging the pregnancy.³ Magnesium sulphate should be considered to prevent seizure in women with pre-eclampsia for whom there is concern about the risk of eclampsia. This is usually in the context of severe pre-eclampsia once a delivery decision has been made and in the immediate postpartum period⁴.

Compared with placebo or no treatment, the use of MgSO₄ more than halved the risk of eclampsia and the number needed to treat to prevent one seizure in this group of women was 50⁵.

4. MANAGEMENT AT HOME AND HEALTH CLINIC

- The patient should be referred to the hospital immediately with Code Red.
- Arrange for transport and accompany the patient to hospital (refer to **Section 4** on Transport). To inform the receiving hospital (labour room) prior to referral.
- Set up an IV drip with normal saline for emergency administration of drugs for resuscitation if the need arises. To give deep IM MgSO₄ 10g bolus (5g each buttock) to prevent eclampsia.
- To lower blood pressure give oral nifedipine (10mg stat) or IM hydralazine 6.25 mg. (preparation: 1 ampule contain 20mg hydralazine + 9ml distill water = 2mg/ml. Give 3.1 ml (equivalent to 6.2mg)

During Transfer

- Monitor and record the maternal BP, pulse rate and fetal heart rate every 15 minutes.
- If an acute situation arises, stop the vehicle to carry out resuscitative measures or divert to the nearest health facility.

5. MANAGEMENT AT HOSPITAL WITHOUT O&G SPECIALIST

- Being tagged Red, the patient should be admitted, managed and monitored in a high dependency area while awaiting transfer to a hospital with specialist.
- Maintain an IV drip of normal saline. To give IV MgSO₄ 4g slow bolus over 10 minutes if not given earlier. This is followed by maintenance IV infusion of MgSO₄ 1g per hour

to prevent eclampsia (refer Section 6.1). If bolus MgSO₄ has been given earlier to continue with the maintenance dose only.

- Monitor the maternal BP, pulse rate, respiratory rate and the fetal heart rate every 15 minutes.
- If DBP \geq 110 mmHg, set up an IV infusion of Hydralazine 20 mg in either 500 ml of Hartman's solution or normal saline. Starting at 5-10 drops per minute (dpm), increase by 5 dpm every 15 minutes until the DBP is around 90 mmHg.
- Continue oral antihypertensive medication.
- Insert a Foley's catheter and record urine output.
- Test for proteinuria.
- Consult the O&G Specialist in the nearest hospital and alert the hospital staff of the receiving hospital about transfer.
- Arrange for an ambulance and ensure that basic resuscitative equipments are available.
- The husband or next of kin should be informed. They should accompany the patient to the hospital.
- If the fetus is preterm, dexamethasone should be administered to improve lung maturity.

6. MANAGEMENT IN HOSPITAL WITH O&G SPECIALIST

- The patient should be admitted and managed in a high dependency area.
- Alert the O&G consultant immediately, so as to be involved in the patient management.
- Monitor the maternal BP, pulse rate, respiratory rate and fetal heart rate every 15 minutes until stabilized.
- Set up an IV drip of Hartman's solution or normal saline for emergency resuscitative therapy. To give IV MgSO₄ 4g slow bolus over 10 minutes if not given earlier. This is followed by maintenance IV infusion of MgSO₄ 1g per hour to prevent eclampsia. (Refer Section 6.1). If bolus MgSO₄ has been given earlier to continue with the maintenance dose only.
- Close monitoring of fluid balance is essential to prevent pulmonary oedema.
- If DBP is \geq 110 mmHg, set up another IV infusion of Hartman's or normal saline with Hydralazine 20 mg. Titrate at 5 dpm against the BP, increasing 5 dpm every 15 minutes until DBP is around 90 mmHg.

- Continue any oral antihypertensive previously started and consider increasing the dosage.
- Insert Foley's catheter and record urine output hourly
- Test for proteinuria
- If the fetus is preterm, administer Dexamethasone
- The fetus should be monitored.

6.1 Obstetric management

- If the gestation is below 34 weeks, there is a place to try to prolong the pregnancy to as near 36 weeks as possible provided there is no danger to the mother or the fetus. This is to reduce the problems of immaturity
- If the gestation is 34 weeks or more, consider delivery if crisis recur.
- In the presence of maternal or fetal complication, then delivery is indicated after stabilization.
- In the absence of obstetric contraindication, aim for vaginal delivery. Otherwise Caesarean section (CS) is recommended.
- The paediatrician should be informed and be present at delivery.

Maternal surveillance:

- Labour should be monitored using Partogram.
- Antihypertensives should be continued if patient is on such treatment.
- Intravenous line should be set-up.
- Adequate analgesia is essential
- USE Syntocinon in place of ergometrine and syntometrine during third stage of labour
- Fluid regime therapy (Refer Section 6.2)
- Paediatrician to be present during delivery.

Fetal surveillance:

- Auscultation of fetal heart rate every 15 minutes
- Institute electronic monitoring (cardiotocography) as indicated.

6.2 Role of Steroid

Antenatal administration of corticosteroids like Dexamethasone prior to preterm delivery reduces neonatal morbidity and mortality. Thus every effort should be made to initiate antenatal corticosteroid therapy in women between 24 – 36 weeks gestation provided there is no evidence of tuberculosis or intrauterine infection⁵

Dosage: 12 mg 12 hourly x 24 hours

6.3 Intrapartum Management

- Adequate analgesia preferably epidural
- To complete Magnesium sulphate infusion to at least 12 hours postpartum
- Strict input/output chart
- Monitor magnesium toxicity
- Paediatrician to standby at delivery

Maternal surveillance:

- Labour should be monitored using Partogram.
- Antihypertensives should be continued if patient is on such treatment.
- Intravenous line should be set-up.
- I.V. Hydrallazine should be considered if DBP is more than 110mmHg
- Adequate analgesia is essential
- USE only Syntocinon during third stage of labour
- Fluid regime therapy (Refer Section 6.2)
- Assist second stage if indicated.

Fetal surveillance:

- Auscultation of fetal heart rate every 15 minutes
- Institute electronic monitoring (cardiotocography) continuously or intermittently as indicated.

6.4 Postpartum Care

- Patient should be observed at OHDU
- Continue her oral antihypertensive agent
- BP control should be between diastolic 90-100 mmHg
- Magnesium infusion should be continue at least 12 hours after delivery
- Monitor input/output

7. CONCLUSION

The principle of management of severe HDP is to stabilise the BP, to prevent complication and eclampsia. Pregnancy can be prolonged to as near term as possible in the absence of maternal and fetal complication. Prophylaxis Magnesium Sulphate should be considered in severe HDP.

References:

1. *Report of the National High Blood Pressure Education Program Working Group on High Blood Pressure in Pregnancy. Am J Obstet Gynecol 2000; 183:1-22.*
2. *Confidential Enquiry of Maternal Deaths in Malaysia. Reports of 2001-2005, 2006-2008*
3. *Broughton-Pipkin F. The hypertensive disorders of pregnancy. BMJ 1995; 311: 609-613.*
4. *Tuffnell DJ, Shennan AH, Waugh JJ, Walker JJ. The management of severe pre-eclampsia/eclampsia. London (UK): Royal College of Obstetricians and Gynaecologists; 2006 Mar. 11 p. (Guideline; no. 10(A)). [52 references]*
5. *Duley L, Gülmezoglu AM, & Henderson-Smart DJ 2003a, 'Magnesium sulphate and other anticonvulsants for women with pre-eclampsia', Cochrane Database of Systematic Reviews, Issue 2. Art. No.: CD000025. DOI: 10.1002/14651858.CD000025.*
6. *Redman CWG. Hypertension in Pregnancy. In: de Sweit, ed. Medical Disorders in Obstetric Practice. Oxford: Blackwell Science, 1995.*
7. *Davey DA, MacGillivray I. The classification and definition of the hypertensive disorders of pregnancy. Am J Obstet Gynecol 1988 ; 158 : 892-*

Section 3.3

Management of Eclampsia

1. INTRODUCTION

Eclampsia is the occurrence of convulsions in a patient with HDP. A clear cut syndrome of pre-eclampsia (PE) nearly always precedes the convulsions, but it may occur even in a modest hypertension without proteinuria. It is thus crucial to recognize that any case of HDP is a potential prelude to eclampsia. In Malaysia, two third of deaths due to eclampsia occurred in antenatal mothers¹.

The pathophysiology of eclampsia is thought to involve cerebral vasospasm leading to ischaemia, disruption of the blood brain barrier and cerebral oedema. Neurological complications may include coma, focal motor deficits and cortical blindness. Cerebrovascular haemorrhage may complicate about 2% of cases.²

2. PREVENTION

Being of unclear aetiology, it is not easy to prevent pre-eclampsia. Currently, a simple and effective way of reducing the dangers of pre-eclampsia is to screen pregnant women for proteinuria and hypertension during antenatal care.³ Otherwise the following steps may be considered:

- The early recognition and treatment of mild HDP
- The early recognition and treatment of severe HDP

The signs of HDP usually appear over a period of several days in the following order:

- fluid retention (or excessive weight gain e.g. >1 kg per week)
- hypertension of 140/90 mmHg or more
- proteinuria

However, they can appear in any order or all together in less than 24 hours. The symptoms and signs of impending eclampsia should always be looked for in patients with HDP:

Severe frontal headache
Vomiting
Blurring of vision
Epigastric pain
Hyper-reflexia
Severe hypertension

Eclampsia is the occurrence of epileptiform convulsions. Four stages are described⁴:

- Premonitory stage: This lasts 10 – 20 seconds during which:
 - the eyes roll or stare
 - the face and hand muscles may twitch
 - there is loss of consciousness
- Tonic stage: This lasts 10 – 20 seconds during which:
 - the muscles go stiff or rigid
 - the diaphragm is in spasm so that breathing stops and colour of skin becomes cyanosed
 - the back may be arched
 - the teeth are clenched
 - the eyes bulge
- Clonic stage : This lasts 1 – 2 minutes and is marked by:
 - violent contraction and relaxation of muscles
 - increased saliva causes "foaming" at mouth
 - deep noisy breathing
 - inhalation of mucous or saliva
 - face looks congested and swollen
 - tongue is bitten by violent action of jaws
- Coma stage: This may last minutes or hours during which:
 - there is a deep state of unconsciousness
 - breathing is noisy and rapid
 - cyanosis fades but face remains congested
 - further fits may occur

3. TREATMENT

The goals of treatment are:

- To treat convulsions and prevent recurrence (3,4,5)
- To control the blood pressure
- To stabilise the mother
- To deliver the fetus

4. MANAGEMENT OF ECLAMPSIA AT HOME & HEALTH CLINICS

4.1 Immediate measures

- Call for medical assistance.
- The patient should be placed in the lateral position. Maintain airway, O2 given through nasal prong/ ventimask.
- 10 gm 50% solution (20 mls) is injected intramuscularly. One half is injected into upper outer quadrant of each buttock in zigzag manner (*preceded by local anaesthesia if necessary*) using a 21 gauge needle.
- Antihypertensive therapy eg. Hydralazine or Labetalol if available or Nifedipine, may be needed to be administered to control hypertension.
- Set up an IV drip with normal saline for emergency administration of drugs for further resuscitation.
- Suck out secretions/saliva
- Insert a Foley's catheter to record and monitor urine output
- Monitor and record the maternal BP, pulse rate, respiration rate and the fetal heart beat every 15 minutes using a Labour Progress Chart.
- Arrange for transport and accompany the patient to hospital (Refer Section 4). To inform the labour room personnel of the receiving hospital prior referral.

4.2 During Transfer

- Continue the monitoring of the mother and fetus as above.
- Maintain patient in lateral position.
- Maintain airway with oxygen
- Continue IV drip: normal saline .
- **To prepare IV MgSO₄ 2g or 5g/IM in a syringe in case patient threw recurrent seizure during transfer.**

5. MANAGEMENT OF ECLAMPSIA AT THE HOSPITAL WITHOUT O&G SPECIALIST

Eclampsia is an obstetric emergency, and although there is no O&G Specialist, the management of the ill patient has to be carried out appropriately:

5.1 Immediate measures

- Initiate **Red Alert System** by calling other doctors to help.
- Put the patient in the lateral position.
- Suck out secretions/saliva
- Insert an airway and give oxygen 6-8L/ min
- Give IV MgSO₄ 4g slow bolus over 10 minutes if not given earlier. This is followed by maintenance IV infusion of MgSO₄ 1g per hour to prevent eclampsia. (Refer Section 6.1).
- If **IV line is not secure**, 10 gm 50% solution (20 mls) is injected intramuscularly. One half is injected into upper outer quadrant of each buttock in zigzag manner (*preceded by local anaesthesia if necessary*) using a 21 gauge needle.

- To run normal saline (NS) infusion for further resuscitative measures.
- Insert a Foley's catheter and record urine output. Test for proteinuria.
- Monitor and record the maternal BP, pulse rate, respiratory rate and also fetal heartbeat every 15 minutes.
- If the DBP \geq 110 mmHg, Hydralazine infusion should be started : 20 mg in 500 ml of NS or Hartman's solution , starting at 5 dpm and increasing by 5 dpm every 15 minutes until the DBP is about 90 mmHg.
- Consult the O&G Specialist at the nearest hospital to transfer the patient and alert the hospital staff. The patient should be transferred **only after the initial stabilisation.**

5.2 During Transfer

- **The patient should be accompanied by a doctor.**
- An ambulance with basic resuscitative equipment is required for the transfer to the referral hospital.
- The husband or next of kin should be informed and they should accompany the patient too.
- Continue the monitoring of the vital signs as above during the transfer and document the readings.
- Continue normal saline infusion
- To be ready with IV MgSO₄ 2g or 5g/IM in a syringe in case patient develop recurrent convulsion during transfer.

6. MANAGEMENT OF ECLAMPSIA AT THE HOSPITAL WITH O&G SPECIALIST

Eclampsia is an obstetric emergency which should be managed by a team of doctors and nurses. The Red Alert system proposed by the Ministry of Health in 1992, should be initiated immediately:

- O&G consultant, specialist and Registrar
- Anaesthetic consultant, specialist and Registrar
- Matron/Sister on-call
- Blood bank specialist/technician

6.1 During convulsions

- The patient is managed in the lateral position.
- Suck out secretions/saliva. Maintain airway and O₂.
- Give MgSO₄:

10 gm 50% solution (20 mls) is injected intramuscularly. One half is injected into upper outer quadrant of each buttock in zigzag manner (*preceded by local anaesthesia if necessary*) using a 21 gauge needle **OR** intravenously as described in **Section 6.1** (Basic Facts on Common Drugs used in HDP).

- Antihypertensive therapy as described above (Refer Section 6.1).

NB: *Clinical monitoring for magnesium toxicity is an acceptable, reliable and safe technique. Hourly assessment of patellar reflex and respiratory rate should be carried out. If the reflexes are absent or respiratory rate less than 16 per minute, 1 gm IV calcium gluconate (over 10 minutes) should be given.*

6.2 After convulsions

- Continue maintain airway and oxygen administered at 6-8 L/min.
- Set up an IV line with Normal Saline.
- Insert Foley's catheter to check urine output.

- Monitor maternal vital signs: BP, pulse rate, respiration rate and also tendon reflexes.
- Continue MgSO₄ infusion 1 gm/hour and to be continue till 24 hours after delivery or convulsion whichever is later
- If DBP is more than 110mmHg, treat with Hydralazine infusion titrate to BP using syringe pump 20mg in 50 mls normal saline Start at 5 mls/hr and titrate every 15 minutes and aim blood pressure diastolic about 90 mmHg.

Or

- Using syringe pump, IV labetalol 50mg in 50 ml NS, start with 5 mls/hour after excluding heart failure and bronchial asthma. (refer protocol in the procedure)
- Alert the Anaesthesiologist for possible operative procedure, and the need for ICU or HDU care.
- Reassess the pregnancy to decide the timing and mode of delivery

6.3 Investigations

Eclampsia is a multisystem disorder and the following complications may occur: Haemolysis, elevated liver enzymes, low platelets (HELLP), DIVC, renal failure, acute pulmonary oedema, intracranial haemorrhage, adult respiratory distress syndrome.² Thus the following investigations should be sent for:

- Haemoglobin
- Platelet count
- Coagulation profile
- Transaminases (Liver function test)
- Renal profile, Uric acid
- CT scan in the presence of neurological deficits or recurrent fits

6.4 Obstetric Management

- The mainstay of treatment of eclampsia is delivery after stabilisation of the patient irrespective of gestational age. Delivery should be conducted in fully equipped hospital with ICU, HDU and NICU facilities.
- If patient is in advance stage of labour (cephalic and os 8cm and above) vaginal delivery is possible in the absence of fetal or maternal complication. Otherwise Caesarean section is advisable.
- The husband or next of kin should be informed on the progress and plan of management.
- The paediatrician should be informed and be present at the delivery
- After delivery, high dependency care should be continued **for at least 24 hours**.

6.5 Choice of anaesthesia/analgesia

If there are no contraindications, epidural analgesia is the preferred mode of providing pain relief during labour as this can be extended for any operative delivery/ procedure. (Refer Section 3.5).

6.6 Immediate postpartum care

After delivery, eclamptic patients should continue to be nursed and monitored in the high dependency area (HDU) for at least 24 hours since the risk of recurrence of convulsions is still very high. Once the condition is stabilised/optimised, the patient can be transferred to the general ward to be nursed according to the postnatal care of patients with HDP (Refer Section 3.1).

On discharge, it is preferred that the follow-up postnatal visit to be at the referral hospital.

7. CONCLUSION

Eclampsia is an obstetric emergency and requires multidisciplinary approach. It is a common and dangerous obstetric complication in Malaysia. Delivery is the mainstay of treatment after the maternal condition has been optimized. With the availability of Magnesium sulphate and training of its use at the peripheral clinic, a reduction and better management of eclampsia is anticipated in near future.

References:

1. *Confidential Enquiry of Maternal Deaths in Malaysia. Reports of 2006-2008.*
2. Douglas KA, Redman CWG. *Eclampsia in the United Kingdom. Br Med J 1994, 309: 1395-1400.*
3. Duley L & Henderson-Smart D 2003b, 'Magnesium sulphate versus diazepam for eclampsia', *Cochrane Database of Systematic Reviews, Issue 4. Art. No.: CD000127. DOI: 10.1002/14651858.CD000127.*
4. Duley L & Henderson-Smart D 2003c, 'Magnesium sulphate versus phenytoin for eclampsia', *Cochrane Database of Systematic Reviews, Issue 4. Art. No.: CD000128. DOI: 10.1002/14651858.CD000128.*
5. Duley L, Henderson-Smart DJ, & Meher S 2006, 'Drugs for treatment of very high blood pressure during pregnancy. *Cochrane Database of Systematic Reviews, Issue 3. Art. No.: CD001449. DOI: 10.1002/14651858.CD001449.pub2.*
6. *W.H.O. Teaching Manual on Midwifery. 2005. Eclampsia. WHO/FRH/MSM/96*

Section 3.4

Management of HELLP Syndrome

1. INTRODUCTION

The acronym HELLP Syndrome is a variant of severe preeclampsia and is characterised by:

- Haemolysis (H)
- Elevated Liver enzymes (EL)
- Low Platelets (thrombocytopenia) (LP)

The syndrome has been described in various gestational ages, ranging from mid-second trimester of pregnancy until several days postpartum.¹ It rarely occurs before 25 weeks pregnancy. Its clinical significance is its association with increased maternal and perinatal complications. The maternal mortality is high (24% in one series) and perinatal mortality ranges from 30-40%.²

3. CLINICAL RECOGNITION

The signs and symptoms of PE-Eclampsia must be present. In addition, the following derangements must be confirmed by laboratory tests.

- Microangiopathic haemolytic anemia
- Thrombocytopenia
- Hepatic Dysfunction

The exact levels of biochemical and haematological values and criteria that are used to make the diagnosis are debated in the literature³

4. INVESTIGATIONS

- i) Full blood count including peripheral blood smear for evidence of haemolysis
- ii) Liver function tests (SGOT, LDH)
- iii) Renal function test (Creatinine Clearance, uric acid)

- iv) Coagulation Profile (Prothrombin time, Partial Thromboplastin Time, INR, Fibrinogen)

(a) Thrombocytopenia is the principal and earliest coagulation abnormality detected. Maternal platelets continue to decrease immediately postpartum but increase by the third day. Failure of platelets to increase within 96 hours of delivery may indicate a serious disorder and multiorgan dysfunction .

(b) Increased LDH and decreased serum haptoglobin levels are sensitive early markers of HELLP Syndrome.

5. DIFFERENTIAL DIAGNOSIS

- i) Thrombotic thrombocytopenic purpura
- ii) Haemolytic-uremic syndrome
- iii) Acute fatty liver of pregnancy
- iv) Connective tissue disorders (SLE)
- v) Dengue Fever

6. COMPLICATIONS OF HELLP SYNDROME

- i) Disseminated intravascular coagulation
- ii) Abruption placenta
- iii) Acute pulmonary oedema
- iv) Acute renal failure
- v) Intracerebral haemorrhage/stroke
- vi) Subcapsular liver haematoma
- vii) Retinal detachment
- viii) Death

7. TREATMENT

- i) Early recognition and institution of appropriate therapy in HDU or ICU
- ii) Additional investigations may be necessary, depending on severity of condition *eg. arterial blood gas, chest x-ray*
- iii) LDH, SGOT, platelets, PCV, BUSE every 12-24 hours and a further 48 hours after delivery
- iv) Control Hypertension

80% have elevated BP necessitating antihypertensive treatment. Aim to reduce BP without compromising placenta perfusion (*if undelivered*).

Although hydralazine or labetalol may be prescribed, calcium channel antagonists have potent peripheral arterial vasodilation properties with renal and cardiac sparing effect. Urine output is improved and rapid normalisation of postpartum platelet count has been reported.

- v) If spontaneous haemorrhage from injection sites are noticed (or platelet count is <50,000/ul) platelets should be transfused
- vi) Role of corticosteroid in improving platelet count in the treatment of HELLP syndrome is rather limited. Although in some observational studies did show dexamethasone significantly increase the platelet count but this however did not translate to improvement in outcomes and the clinical relevance of this is unclear⁴. Postpartum use of dexamethasone was also compared with placebo in a well designed RCT and found no difference in key maternal morbidity and mortality indices and no difference in use of blood products between the two groups.⁵
- vii) Assess coagulation profile, determine if DIVC is present and treat for this disorder
- viii) Manage fluid and electrolytes
- ix) If epigastric pain is present, treat as for eclampsia.
- x) Assess fetal condition
 - Corticosteroids if fetus is 24-36 weeks gestation with caution
 - Deliver in 24-48 hours after steroids are administered and maternal condition is stable
- xi) Correct thrombocytopenia

- Transfuse platelets regardless of platelet count if there is bleeding from intravenous site
- PPH is seen in vaginal delivery if platelets count is $<40000/\text{cm}^3$
- Platelet transfusion indicated after delivery for first 24 hours to maintain counts $>50000/\text{cm}^3$ in Caesarean section and $>20000/\text{cm}^3$ in vaginal delivery.

xii) Labour and Delivery

- Aim for vaginal delivery and avoid episiotomy if possible
- Caesarean section done only for obstetric indication and vertical skin incision preferred to pfannensteil incision²
- Allow for spontaneous expulsion of placenta rather than manual extraction at caesarean section
- Uterine repair is done in-situ, rather than exteriorisation to minimize uterine and adnexal trauma
- Mass closure should be done for abdominal incision
- Antibiotics should be prescribed for 3 days.
- In the non-obstetric population a level of $<50 \times 10^9/\text{L}$ is considered significant in the context of surgery or major haemorrhage⁶

Aim for carefully controlled, skillfully executed non-operative vaginal delivery

xii) Postpartum Care

- Care as for severe PE-Eclampsia in HDU/ICU
- Watch for hepatic rupture/haemorrhage (PE-Eclampsia + HELLP + Right Hypochondrial pain + Hypotension)
- Watch for upward trend in platelets, downward trend of LDH, SGOT
- Dexamethasone 12 mg 12 hrly till platelets $>100,000/\text{ul}$, than Dexamethasone 5 mg 12 hrly for further 2 doses.

References:

1. *Matrin JN, Magann EF, Blake PG et al, Analysis of 454 pregnancies with severe preeclampsia/eclampsia, HELLP syndrome, using the 3-class system of classification, Am J Obstet Gynecol 1993 : 68 :386*
2. *Magann EF, Martin JN Twelve steps to optimal management of HELLP Syndrome in Clinical Obstetrics & Gynaecology 1999; 42; 532-550*
3. *Australian and New Zealand College of Anaesthetists 2008*
4. *O'Brien JM, Shumante SA, Satchwell SL, Milligan DA & Barton JR 2002, 'Maternal benefit of corticosteroid therapy in patients with HELLP (hemolysis, elevated liver enzymes, and low platelet count) syndrome: impact on the rate of regional anesthesia', Am J Obstet Gynecol, vol. 186, pp. 475-9.*
5. *Katz LM, de Amorim, MM, Figueiroa JN & Silva JL 2008, 'Postpartum dexamethasone for women with hemolysis, elevated liver enzymes, and low platelets (HELLP) syndrome: a double-blind, placebo-controlled, randomized clinical trial', Am J Obstet Gynecol, vol. 198. pp. 283 e1-8.*
6. *National Health Medical Research Council 2001, Clinical Practice Guidelines. Appropriate use of platelets, viewed October 2008, <http://www.nhmrc.gov.au/publications/synopses/_files/cp79.pdf>.*
7. *Roberts WE, Perry KG, Woods JB. The intrapartum platelets count in patients with HELLP Syndrome : Is it predictive of late haemorrhage complications Am J Obstet Gynecol 1994; 171; 7999-8*

Section 3.5

Anaesthesia and Hypertensive Disorders in Pregnancy

1. ROLE OF THE ANAESTHETIST

There should be a multidisciplinary approach to the management of patients with hypertensive disorders in pregnancy and the anaesthetist should be involved in the care of patients with severe pre-eclampsia at an early stage. The anaesthetist is an integral member of the "RED ALERT" team. There must be active communication between the obstetrician and the anaesthetist to successfully achieve a good outcome.

The anaesthetist may be involved in:

- Providing pain relief in labour
- Control and stabilization of blood pressure
- Optimization of intravascular volume status
- The prevention of convulsions
- Treatment of coagulation abnormalities.¹

In order to achieve these, the anaesthetist will have to actively contribute to:

- The placement of epidural catheter for pain relief during labour
- The placement of arterial
- The placement of central line (if no coagulopathy)
- Electively manage "the difficult airway" and prevent organ damage due to hypoxia
- And also plan the best time and the best anaesthetic technique to deliver the foetus.²

2. BLOOD PRESSURE (BP) CONTROL³

Anaesthetist is often asked the acceptable BP before patient is allowed to undergo anaesthesia. Below are some guidelines:

- In non-severe HTN, acceptable BP is SBP 140-159/DBP 90-109
- Patient must be on some treatment such as oral Labetalol unless contraindicated or (methyldopa, nifedipine, isradipine, metoprolol, pindolol, propranolol, low-dose diazoxide)

- **Atenolol and ACE inhibitors are contraindicated because the risk of fetal bradycardia and nephropathy**
- In severe HTN, BP $\geq 160/110$ should be treated. Any BP ≥ 180 is a medical emergency and reducing severe HPT decreases mortality risk and also reduces fetal morbidity
- To avoid precipitous falls in BP.
- Aim of BP reduction of Systolic BP 140-150, Diastolic BP 80-100, at a rate of 10-20mmHg every 10-20min.
- I/V Esmolol or Labetolol (oral or IV) should be avoided in asthmatic patient.
- Hydralazine (intermittent IV boluses or IV infusion 0.5-10mg/h), can cause maternal tachycardia.
- IV infusion Glyceryl trinitrate 5mcg/min (max 100mcg/min) is recommended in Hypertensive Crisis or in Pre-Eclampsia with APO.
- Anaesthesia (neuroaxial block or GA) should only be attempted when BP is back to normal or less than 160/100 in severe hypertensive crisis. Fetus may show sign of distress if BP is very high. Controlling of BP with left lateral tilt and oxygen supplement do improve fetal wellbeing.
- Continuous fetal heart monitoring is mandatory until BP is stable.

3. ANALGESIA AND ANAESTHESIA FOR PATIENTS WITH PRE-ECLAMPSIA

3.1 Analgesia for labour

If there is no contraindication, epidural analgesia is the preferred mode for providing pain relief, and this can be extended for Caesarean delivery if the need arises.⁴ Epidural also helps controlling blood pressure in severe HTN cases.

3.2 Anaesthesia for LSCS

3.2.1 General anaesthesia

Used for emergencies, or when regional anaesthesia is contraindicated.

(i) Risks of general anaesthesia:

- Intubation and extubation can cause a rise in both systolic and diastolic blood pressure.⁵
- Intubation can be difficult due to oedema of the larynx

- Aspiration of gastric content is higher, and intubation has to be carried out with rapid sequence induction with cricoid pressure.

(ii) Precautions:

- If MgSO₄ has been used to prevent convulsions then the dose of muscle relaxants must be reduced. MgSO₄ inhibits calcium facilitated pre-synaptic transmitter release hence enhancing sensitivity to non-depolarizing neuromuscular blockers. Suxamethonium fasciculations may not occur after administration of MgSO₄.¹ Newborn can also suffer from transient hypotonia.
- Extubation should only be undertaken when patient's protective reflexes are well established.

(iii) Technique of General Anaesthesia

- Rapid Sequence Induction (RSI) using short acting induction agent such as Sodium Thiopentone or Propofol and followed by Suxamethonium is well practised. However, in patients without any potential airway difficulty and normal renal function, Rocuronium can be used instead of Suxamethonium. This is provided the unit has Sugammadex reversal agent in case immediate reversal is necessary.
- Adequate pre-oxygenation is mandatory since Functional Residual Capacity (FRC) is usually compromised especially in obese pre-eclamptic patient. Risk of patient going into hypoxia is very high especially during period of apnea after RSI.
- Oxygen analyser showing Inspired O₂ (FiO₂) and Expired O₂ more than 95% are good parameters to show that FRC is adequately filled up before attempting intubation.
- Induction phase should be as smooth as possible. Sympathetic response towards laryngoscopy can be obtunded using intravenous Lignocaine 1mg/kg with/without intravenous Esmolol 1mg/kg (if not asthmatic) about 90 sec before intubation. Lignocaine 10% spray is also helpful to prevent post extubation sore throat and also obtund sympathetic response postoperatively. However the dose need to be calculated so as not to exceed toxic dose (max 3mg/kg).
- Maintenance involves ensuring good analgesia, optimum anaesthesia level and adequate lung ventilation. Judicious intravenous therapy in both

intraoperative and postoperative period is important as the patient can be easily tipped over to acute pulmonary odema.⁶

- The use of uterotonic is important to prevent postpartum haemorrhage. However uterotonic such as oxytocin can cause vasodilation and others like ergometrine and syntometrine can cause hypertensive crisis. Thus oxytocin needs to be used cautiously while ergometrine and syntometrine are contraindicated in pre-eclamptic patients. Oxytocin must be given as a slow bolus or infusion to prevent hypotension.
- Extubation is another critical period since high sympathetic response can cause hypertensive crisis. Another dose of intravenous Esmolol 1mg/kg 90 seconds before extubation can be given. Extubating is only done once all protective reflexes are returned and patient has good motor power as shown by ability to sustain a head lift for 5 seconds.
- Post operatively, good pain relief can be achieved with Patient Controlled Analgesia (PCA) using opioids and other modes of multimodal analgesia. NSAIDS are contraindicated due to risk of nephropathy.

3.2.2 Regional anaesthesia

Epidural or spinal anaesthesia would be far safer for the mother and the foetus. A new technique called Low Dose Combined Spinal Epidural (CSE) is increasingly popular as it can provide a faster onset of neuroaxial block while providing the option of extending the block in unanticipated prolonged operation.⁷ It is also safer compared to single shot spinal. Sudden drop in blood pressure which is associated with single shot spinal can be overcome with CSE since initial spinal dose is given in a smaller dose. The use of intrathecal morphine 0.1-0.15mg in addition to local anaesthetic cocktail can provide excellent post-operative pain relief.

- **Risks:**
 - Haematoma at epidural spaces
 - Severe hypotension after initiating the block
- **Precautions:**⁸
 - Coagulation screen should be normal
 - Platelet count $>80 \times 10^9/L$
 - Sufficient preloading should be done before initiating the block
 - Left lateral tilt to prevent supine hypotensive syndrome

- Early use of ephedrine hydrochloride or phenylephrine infusion after initiating the block.

3.3 Post-Operative Management

- The patient must be monitored in HDW or ICU post operatively since they are still at risk of developing eclampsia up to 48 hours.
- Good postoperative analgesia is important to promote early ambulation and faster recovery.
- Thromboprophylaxis can be started after 4-6hrs post operatively.
- Judicious fluid management is important during this period because the risk of acute pulmonary oedema. Invasive arterial line would show beat to beat blood pressure changes. Central venous line is an optional. Fluid restriction to 2L/d is acceptable.
- MgSO₄ should be discontinued after 24 hours. Monitoring should utilize clinical parameters (urine output, respiratory rate, SpO₂ & patellar reflexes) and serum Mg be measured to prevent toxicity especially in renal insufficiency. Therapeutic level Mg²⁺ (2-4mmol/l). Antidote is 10% Ca Gluconate 1g over 10 min³.

4. CRITERIA FOR ICU MANAGEMENT:

ICU admission is indicated in the following:

- Eclampsia
- Cerebrovascular accident
- Pulmonary oedema
- Aspiration pneumonitis/ARDS
- HELLP Syndrome
- Renal complications eg. AKI
- Poor recovery from anaesthesia post caesarean section

If delivery has not occurred, management should be directed at stabilising the patient haemodynamically, controlling any convulsions, protecting the airway and delivery of the foetus.

'Uncomplicated eclampsia' do not usually require prolonged ventilation. Once their cardiovascular, respiratory and renal systems are normalised they can be weaned off

sedation and extubated soon after their protective reflexes have returned. What is important is continued close monitoring in ICU or HDU for at least 48 hours. Transfer to general ward should only be done when clinical and biochemical parameters begin to return to normal.⁹

Patients who develop cerebro-vascular accidents may be more difficult to be weaned off, and these patients may require CT scan to establish the amount of cerebral damage.

References:

1. Mushambi MC, Halligan AW., Williamson K., Recent Developments in the Pathophysiology and Management of Pre-eclampsia. *Br J. Anaesth.* 1996; 76 : 133-148
2. Ramanathan J., Sibai BM., *Obstetric Anaesthesia.* Mark C. Norris. (ed), J.B. Lippincott Com. 1993; 109-21
3. A.T Dennis. Management of pre-eclampsia: issues for anaesthetists. *Anaesthesia* 2012, 67, 1009-1020
4. Robert Langer, MD., *Anaesthetic Management of Pre-eclampsia.*, New York Medical Centre
5. Ramanathan J., "Pathophysiology and Anaesthetic implications in pre-eclampsia", *Clinical Obstetrics and Gynaecology.* Ed. 1992; 35: 414-25
6. Wallace CH, Leveno KJ, Cunningham FG et al. Randomised comparison of general and regional anaesthesia for caesarean delivery in pregnancies complicated by severe Pre-eclampsia. *Obstet Gynaecol* 1996; 86: 193-199.
7. Berends N, Teunkens A, Vandermeersch E, Van de Velde M. A randomized trial comparing low-dose combined spinal-epidural anesthesia and conventional epidural anesthesia for cesarean section in severe preeclampsia. *Acta Anaesthesiol Belg.* 2005;56: 155–62.
8. Sujata C, Rashmi S, Subarachnoid block for caesarean section in severe preeclampsia. *Journal of Anaesthesiology and Clinical Pharmacology*, 2011;27,2,169-173
9. Walker JJ., *Recent Advances in Obstetrics and Gynaecology* 20., Bonnar J. (ed), Churchill Livingstone : 111

Section 3.6

Fetal Surveillance in HDP

1. INTRODUCTION

The assessment of the fetus is an integral part of the management of patients with hypertensive disorders in pregnancy. When there is mild disease which settles quickly on admission to hospital, intensive fetal monitoring is not indicated. Where the disease progresses, close fetal surveillance is necessary in order to determine the effect of HDP on the fetus and if necessary to establish the timing of delivery once the overall situation has been addressed. If there is severe HDP and intensive fetal monitoring is necessary, then it is best undertaken in a tertiary obstetric and perinatal centre, where if required, appropriate management of a critically ill mother or infant can be provided quickly and safely.

In HDP, the fetus is monitored for:

- Growth
- Well-being

2. FETAL MONITORING DURING THE ANTEPARTUM PERIOD

2.1 Fetal growth

- **Symphysis-fundal height** tape measurement should be performed routinely from 22-24 weeks onwards in all pregnancies. This would be more important in patients who are going to develop HDP. This measurement should be performed at every antenatal visit whether at home or at the clinic.
- **Maternal weight gain** may not be useful now with the widespread availability of ultrasound. However, it would be useful to know whether there is weight gain or loss. Static weight gain or weight loss might be indicating intrauterine growth restriction (IUGR) and subsequent increased risk to the fetus.
- **Ultrasound scanning:** Measurement of fetal crown-rump length (CRL) in the first trimester or the biparietal diameter (BPD) before 24 weeks are accurate measures of gestational age. For HDP patients, the biparietal diameter (BPD) FL, head circumference (HC), abdominal circumference (AC) and the amniotic fluid index (AFI) should be

measured monthly to ensure satisfactory growth of the fetus. HDP is commonly associated with IUGR. Plotting of the fetal growth chart is encourage to identify early onset of growth restriction

2.2 Fetal Well-Being

- **Cardiotocography (CTG):** The antenatal non-stress CTG is a recognised method of fetal assessment. It has a high specificity and has been shown to have a sensitivity of 85% in HDP.¹ It is more useful than Doppler studies because of the wide normal variability seen with the latter particularly in the second trimester.² The frequency of the non-stress test is based on the severity and stability of hypertension. Normally, if HDP is not severe, twice weekly CTG should be sufficient. In severe HDP, this may have to be done more frequently.
- **Fetal movement chart (FMC):** The 'count-to10' FMC is a cheap and easy method of assessing the well-being of the fetus. It will also make the mother feel that she is part of the team caring for her fetus. The FMC in Malaysia has been translated into 4 languages to cater for the various ethnic groups. It should be given to the mother with HDP from 28 - 30 weeks onwards. It allows the mother to feel that she is contributing to her fetal wellbeing
- **Fetal heart rate monitoring with the Pinard's** fetal stethoscope should be continued as usually practised.
- **Ultrasound scanning** can also assess fetal well being i.e by doing the Biophysical Profile. This includes: – FHR, fetal movements and tone, breathing movements, amniotic fluid index and also the CTG. Two controlled trials of biophysical profile testing showed no improvement in outcome when compared to non-stress test.^{3,4}
- **Doppler velocimetry studies:** This is available in tertiary centres. There is a wide range of variability. However, reverse end diastolic flow in the umbilical artery is associated with poor fetal perfusion and hypoxia. Presence of increase Doppler signals is an indication for closer fetal surveillance and anticipate early delivery. Presence of reverse end diastolic flow is an indicator for an immediate delivery

3. METHODS OF FETAL MONITORING DURING LABOUR

- **Cardiotocography:** In most, if not all the hospitals in Malaysia, the CTG is the main method of fetal monitoring of high risk pregnancies during labour, either continuously or intermittently. For the patients with HDP, continuous monitoring is preferred. The perfusion of the placental bed, which is already decreased in HDP, can be further decreased during uterine contractions leading to fetal hypoxia. This will be shown as *late deceleration pattern on CTG*.
- **Fetal Blood Sampling (FBS):** If this facility is available, it has a role in detecting gradually developing hypoxia. Special attention must be paid to those who are likely to get acidotic faster e.g. fetuses who are IUGR, HDP, preterm, scanty meconium and thick meconium liquor.⁵
- **Fetal Heart Rate:** This should be monitored using the Pinard's fetal stethoscope every **15** minutes. It is a cheap and easy method with the added advantage of direct contact with the patient.

4. CONCLUSION

Careful and regular monitoring of the growth and well being of the fetus in HDP will improve the perinatal outcome of the fetus, who may be growing restrictively and more prone to hypoxia. However, even the findings of the most sophisticated monitoring facility should be taken into consideration with the overall clinical picture of the patient. This will ensure favourable fetal and maternal outcome.

References:

1. *Devoe LD, Gardner P, Dear C, Castillo RA. The diagnostic values of concurrent nonstress testing, amniotic fluid measurement and Doppler velocimetry in screening a general high-risk population. Am J Obstet Gynecol 1990, 163: 1040-1048*
2. *Low JA. The current status of maternal and fetal blood flow velocimetry. Am J Obstet Gynecol 1991, 164: 1049-1063*
3. *Manning FA, Lange IR, Morrison I, Harman CR. Fetal biophysical profile score and the non-stress test. A comparative trial. Obstet Gynecol 1984; 64: 326-331*
4. *Platt LD, Walla CA, Paul PH, et al. A prospective trial of the fetal biophysical profile vs. the non-stress test in the management of high risk pregnancies. Am J Obstet Gynecol 1985; 153: 624-633.*
5. *Arulkumaran S, Montan S. The fetus at risk in labour – identification and management. In Contributions to Obstetrics & Gynecology, Vol. 1, Ratnam SS, Ng SC, Sen DK, Arulkumaran S eds. Churchill Livingstone, Singapore 1991, 179-190.*

Section 3.7

Discharge and Follow-up Strategies

Although the definitive management for HDP is said to be the delivery of the fetus and the critical period is the first 48 hours after delivery, its complications have been shown to also occur any time during the postnatal period. It is therefore vital for health care providers to continue monitoring patients during the postnatal period.

1. Discharge from Hospital

1.1 Discharge Criteria

- Blood Pressure has settled below 100 mmHg DBP
- NO end-organ dysfunction
- Patient:
 - understands the disease and its complications,
 - compliant to medication
 - accessible to a health center
 - has good support from family

1.2. Care Plan on Discharge

- **Counselling**

- (i) **Complication of HDP during puerperium.**

Mother and family members should be counselled on the signs and symptoms of HDP and its complications, prior to discharge. They should be warned that, HDP and its complications might occur any time during post-natal period. In many women with chronic hypertension or superimposed pre-eclampsia, the blood pressure is unstable for 1-2 weeks after delivery and may be difficult to control ²

(ii) Importance of Contraception

Mother and her spouse should be counselled on the importance of contraception in relation to HDP, the methods available and where to obtain the services. **Continuing exclusive breast-feeding should be encouraged as the method of contraceptive during the first six months.**

Among the complications to observe are:-

- i. Signs and symptoms of impending eclampsia
 - Increase in Blood Pressure
 - Proteinuria
 - Headache
 - Giddiness
 - Blurring of vision
 - Nausea
 - Vomiting
 - Epigastric pain
- ii. Signs and symptoms of pulmonary oedema
 - Cough with or blood stained sputum
 - Difficulty in breathing
 - Orthopnea (not able to lie flat)
 - Cyanosis
- iii. Signs and symptoms of Deep Vein Thrombosis
 - Swelling of leg (the calf muscle)
 - Pain/tenderness of the calf muscle
 - Redness/inflammation of leg

• Notification of Birth

The importance of continuing post-natal care either at the hospital or health center cannot be overemphasized. To facilitate this, notification of birth to the nearest peripheral health center is vital.

Notification of birth can be done either through:-

- telephone
- fax
- report by the family members (next-of-kin) to the nearest health center.

1.3 Management of HDP at community level.

(i) **Timing of post-natal nursing care**

Post-natal nursing care for mothers with history of mild HDP/severe HDP or eclampsia should be done *as soon as possible after* notification is received.

(ii) **Frequency of post-natal nursing care**

For a post-natal mother who is known to be normotensive during pregnancy, the routine scheduled postnatal visit will suffice, unless an increase of blood pressure is detected. Thereafter, frequent post-natal follow-up is required. Post-natal mother with history of mild HDP/severe HDP or eclampsia on the other hand, require more frequent post-natal care.

(iii) **Elements to be monitored during post-natal nursing or follow-up include:**

Every other day follow-up either at the health center or at home, where Blood Pressure, urine for albumin and eliciting for sign and symptoms of severe HDP are carried out by the nursing/paramedical staff.

The patient has to be seen and examined by a doctor either in the hospital or at health center **fortnightly** until six weeks post-natal period.

Elements to be monitored	Frequency of review by nurse	Frequency of review by doctor
<ul style="list-style-type: none"> • Blood Pressure • Urine for protein (if B/P \geq 140/90 mmHg) • Signs and symptoms of severe HDP and pre-eclampsia. • Signs and symptoms of Deep Vein Thrombosis – Post-natal mothers with history of HDP have a higher risk of developing DVT, as a complication of advocating long rest during antenatal period. 	<p>Every other day</p> <p>Every other day</p> <p>Every other day</p> <p>Every other day</p>	<p>Two weekly</p>

(iv) **Criteria for referral**

More often, the blood pressure of a majority of mothers who have history of mild HDP/severe HDP or eclampsia may fall to normal levels during the post-natal period. There is no specific time as to when the B/P will fall to normal levels, however as any other physiological changes related to pregnancy and labour, it may take as long as six weeks.

Referral to hospital may be considered, if:-

- BP > 140/100 mmHg with proteinuria and/ or
- With signs and symptoms of impending Eclampsia
- If hypertension and proteinuria persist beyond six weeks postnatal period.

2. At six weeks post-natal follow-up

- During this follow-up, counselling on the importance of contraception should further be reinforced.
- Mothers whose blood pressure has returned to normal, should be advised to have an early booking and regular antenatal care in the next pregnancy.
- If hypertension and proteinuria persists beyond six weeks post-natal period, the mother should be referred to a physician with regular follow up
- Counselling on the importance of effective contraception at least for 2 years.
- Pre- pregnancy counselling is necessary before next pregnancy.

References

1. *Guideline for management of high risk cases in pregnancy; Second edition: 1991 Ministry of Health Malaysia.*
2. *Guidelines for the management of HDP 2008: Society of Obstetric Medicine of Australia and New Zealand*

SECTION 4

**REFERRAL PROCEDURES &
MANAGEMENT IN TRANSIT**

Section 4

REFERRAL PROCEDURES AND MANAGEMENT IN TRANSIT

1. INTRODUCTION

Referral criteria according to model of good care in the appropriate management of HDP include:

1.1 Referral from community health clinic (KD) to health clinic when patient is found to have:

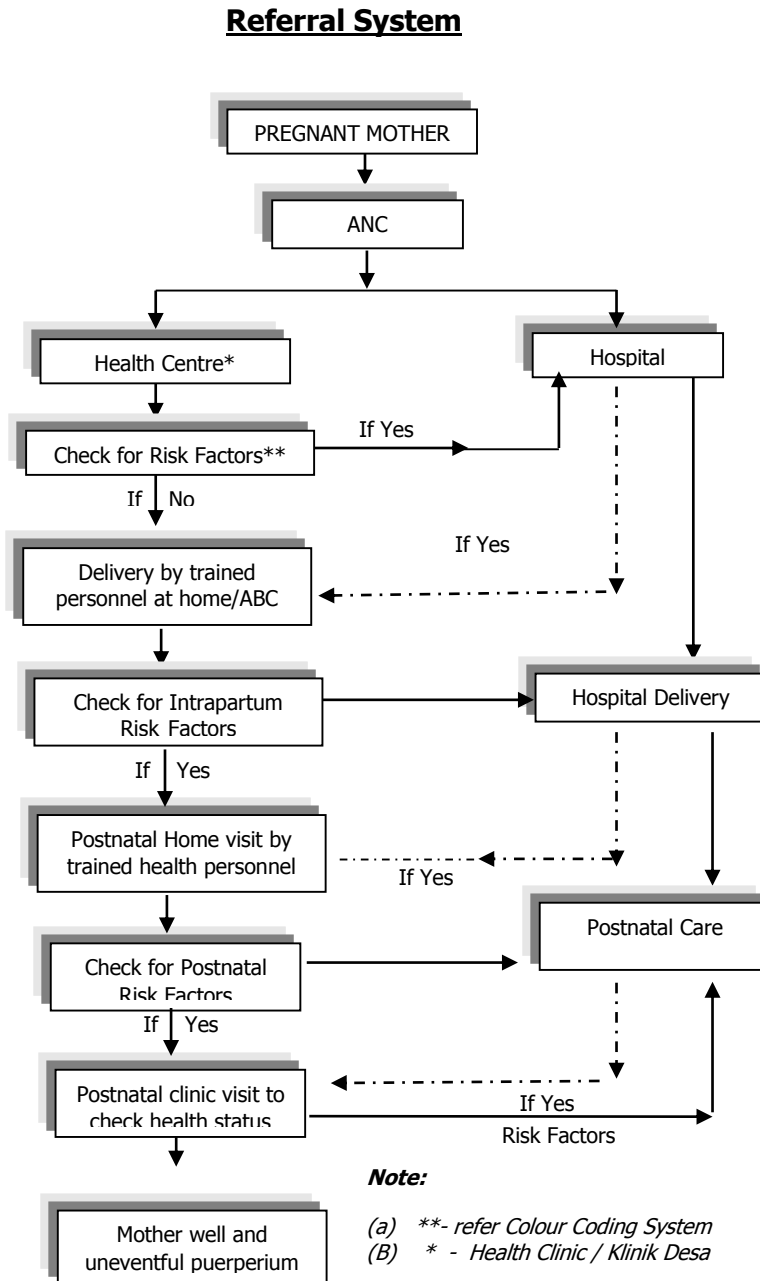
- Hypertension
- Excessive weight gain (weight gain of > 1 kg per week 2nd trimester onwards)
- Raised BP
- Proteinuria with or without hypertension

1.2 Referral from community health clinic (KD) and health clinic to the nearest hospital when patient is found to have:

- Hypertension with proteinuria
- Hypertension with IUGR (Evidence of fetal compromise)
- Hypertension not responding to drug treatment
- Hypertension with excessive weight gain
- Hypertension with symptoms (Headache, Blurring of vision, Epigastric pain, Nausea & vomiting)

(Evidence of maternal compromise as shown by deterioration of renal function/liver function/and coagulopathy)

2. The system of referral is as follows:



3. DATA TO BE INCLUDED IN THE REFERRAL NOTES

In general, symptomatic patients with HDP justify admission to a referral hospital. (Refer to the suggested referral form format)

4. TRANSPORTATION

The type of transport required depends on the situation and availability of vehicle. A fully equipped ambulance is preferable.

5. THE PRINCIPLES OF MANAGEMENT IN TRANSITS

In most instances it is advisable not to send the patient before optimisation of the patient with the idea of 'saving time'. Outcomes are much better if the patient is kept in the referring health centre and stabilised before transfer.

5.1 Pre-transfer

i) Meticulous planning and coordination

Cooperation and continual communication between the referral and receiving hospital is vital. Details of the case including diagnosis, status, details of therapy and treatment required on arrival should be relayed. Treatment advice may be given to the referring doctor before transferring the patient.

ii) Identify personnel, properties, mode of transport

Skilled medical and nursing staff should accompany the patient. The team must be trained and competent in certain skills eg. airway management including intubation and ventilation, setting up intravenous lines, defibrillation and use of resuscitative drugs. A team leader should be appointed who will supervise and coordinate the safe transfer of the patient.

iii) Resuscitate and Stabilise Patient

The principle is to resuscitate and stabilise the patient as far as possible before transfer. This includes fluid, resuscitative drugs, airway control and maintaining ventilatory support. All lines, drains, endotracheal tubes, portable oxygen tanks must be well secured to prevent accidental dislodgement.

iv) Coordinate safe embarkation onto the vehicle

Identify and take only essential equipment including basic life support equipments, other treatment (including drugs) and monitoring equipment.

5.2 **During Transfer**

- i) Maintain stability of patient
- ii) Constant monitoring and documentation of patient condition and treatment
- iii) Direct communication with receiving hospitals is encourage during transportation
- iv) If an acute problem arises, **stop the vehicle** to carry out resuscitative measures or divert to the nearest health facility

5.3 **On Arrival**

- Ensure safe disembarkation
- Hand over to appropriate person

6. FEEDBACK MECHANISM

In ensuring continuity of care of patient, the reply form and the home based card should be filled up on discharge. Patient and next of kin must be informed that the reply note be given to the nearest health centre as soon as possible to ensure continuation of care after discharge. In addition hospital staff should aim to contact or notify nearest health facility by phone, fax or e-mail to ensure that continued care is available. (Refer to the attached referral and feedback form).

SECTION 5

DIFFERENTIAL DIAGNOSIS

Section 5.1

Differential Diagnosis of Convulsions in Pregnancy

Convulsions occurring in women in the second half of pregnancy in the presence of hypertension for the first time are often due to ECLAMPSIA. However, convulsions have been seen following other disorders in pregnancy.

The following conditions must be considered:

- i. Epilepsy
- ii. Cerebral Malaria
- iii. Meningitis
- iv. Subarachnoid Haemorrhage and Intracranial Haemorrhage
- v. Cerebral Tumor
- vi. Septicaemia

The attending nurse or doctor must elicit a good history from relatives and friends especially that related to the premobid status of the patient. Some specific aspects of the above conditions are included to assist health providers.

i) **EPILEPSY**

- History of fits in childhood is common.
- Fits rarely appear for the first time in adult life.
- Patient may have been under medical care or on anticonvulsants.
- Other signs of PE (e.g. hypertension, proteinuria) may be absent after the convulsion.

Tests/Investigations

- Rule out other causes of fits
- Urine for proteinuria
- Blood Urea, serum electrolytes
- EEG

ii) CEREBRAL MALARIA

- Residence in 'malaria-endemic' areas.
- History of fever, headache, lethargy, coma within few hours of feeling unwell.
- Rapidly fatal if not treated urgently

Tests/Investigations

- Haemoglobin
- Total white count
- Blood film for malarial parasite

NB.

- 5% of circulating red cells will be parasited in cerebral malaria.
- If malaria is suspected, do not wait for laboratory results, manage with appropriate antimalaria/drugs and antipyretics.

iii) MENINGITIS

- Convulsions are seen with **pneumococcal meningitis**
- Coma may precede convulsions
- Hypertension is not a common feature
- Onset of meningitis in most cases is sudden (except in tuberculous meningitis)
- **Meningococcal meningitis** occurs in **epidemics** (look for history of other family members similarly affected)
- **Streptococcal** and **Staphylococcal** meningitis are secondary to other infections (eg. Otitis media, mastoiditis, sinusitis)

Test/Investigations

- Distinguish from subarachnoid haemorrhage
- CSF (Lumbar puncture)
 - CSF is under increased pressure
 - Fluid is cloudy in bacterial meningitis, clear in viral meningitis
 - Cell count is increased (bacterial)
 - Causal organism found on bacterial examination

Brudzinkin's neck sign positive

- Passive flexion of neck (chin to chest) leads to spontaneous flexion of legs

Kernig's sign

- Recumbent position, flex thighs close to abdomen and attempt to extend knee joint
- In meningitis, attempts to extend knee is resisted due to stretching of sacral nerve roots (and extension of inflamed meninges)

iv) SUBARACHNOID HAEMORRHAGE AND INTRACRANIAL HAEMORRHAGE

- (a) **Subarachnoid Haemorrhage** is the most common cause of haemorrhage in the brain of young or middle – aged person
- It occurs suddenly in a previously healthy person
 - There is severe headache, neck stiffness, nausea and vomiting (as a result of rupture of cerebral aneurysm)
 - Can occur as a result of trauma to the head
- (b) **Intracranial Haemorrhage** is due to ruptured aneurysm or hypertension.

Tests/Investigations

- Skull X-Ray
- CT/MRI Brain
- Carotid Angiogram
- CSF
 - heavily blood stained
 - not purulent
 - C&S will be negative

Nb:

- (i) Patients with subarachnoid and/or intracranial haemorrhage must be referred to the physician/neurosurgeon for skilled medical treatment
- (ii) Cerebral haemorrhage is a complication of eclampsia. It is important to establish whether the bleeding is the cause or effect of coma (if present) so that correct management can be given.

v) **CEREBRAL TUMOUR**

- Clinical picture will vary according to the location of tumor
- Mental and bodily function may be affected
- Elicit
 - behaviour/personality change
 - speech difficulty
 - visual disturbances
 - neurological deficit
- May occur as a secondary growth to a malignancy elsewhere in the body

Tests/Investigations

- Suspect cerebral tumour if coma deepens or there is no response to anticonvulsants

- Perform full medical/neurological assessment
- Perform CT/MRI Brain
- Lumbar Puncture CSF under increased intra-cranial pressure

Cerebral Abscess and space occupying lesions need to be excluded.

vi) **SEPTICAEMIA**

- Rapidly fatal if treatment is not given
- Puerperal sepsis can lead to septicaemia
- Elicit history of:
 - Prolonged rupture of membranes
 - Interference during labour/delivery
 - eg. Unsterile techniques
 - manual removal of placenta
 - foreign body in vagina
 - abortion by unqualified person or unhygienic circumstances.

Test/Investigations

- Hb, Blood count, platelets
- Coagulation profile
- BUSE
- Urine FEME
- Blood culture
- High vaginal swab for C&S

FITS MAY BE CAUSED by OTHER SEVERE INFECTIONS
eg. Typhoid, viral diseases

CONCLUSION

Convulsions may be a result of vascular, infective, traumatic or metabolic disorders. In pregnancy a clear history of circumstances preceding the seizures together with physical examination will enable the attending health provider to come to a diagnosis. Coma may ensue following the convulsion. A combined/team approach in a tertiary institute will be required. Most of these patients will need intensive care treatment.

Reference

i) WHO Teaching Manual on Midwifery 2008, Eclampsia WHO.FRH/MSM/96.6 Pg. 13-23

Section 5.2

Chronic Hypertension

- Chronic hypertension** in pregnancy is not uncommon, the diagnosis being made when hypertension is present before twenty weeks of pregnancy or beyond six weeks postpartum (refer to page 6).

Primary or Essential hypertension would be the diagnosis in most of these women. However, in a small group (10-20%) of women, a definite cause of hypertension can be identified i.e. **Secondary hypertension**. There are many causes for this (Table below), the most common being **renal parenchymal disease**.

The Causes of Secondary Hypertension

Congenital or Hereditary Problems
Coarctation of aorta Congenital renal artery stenosis Polycystic disease
Endocrine disease
Phaeochromocytoma Primary Aldosteronism (with adrenal tumour in Conn's syndrome or with adrenal hyperplasia) Cushing's syndrome Acromegaly Hyperparathyroidism
Renal disease
Chronic Glomerulonephritis Chronic pyelonephritis Adult polycystic kidney disease Diabetic nephrosclerosis Renin secreting tumors
Vasculitis
Systemic lupus erythematosus Progressive systemic sclerosis Takayashu's disease (pulseless disease) Polyarteritis nodosa

2. **Chronic glomerulonephritis** (idiopathic or post infectious GN) and **lupus nephritis** are the common renal problems encountered. In lupus nephritis other features of systemic lupus erythematosus may be found viz butterfly rash on the face, arthritis and vasculitic lesions over the limbs. Renal profile, autoimmune screening, ultrasound of the kidneys and occasionally a renal biopsy are necessary to conform the diagnosis.

3. **Renovascular disease** as a result of fibromuscular hyperplasia (idiopathic) or as part of Takashu's disease is another occasional cause of hypertension in pregnancy. The presence of abdominal bruit or absent pulses would clinch the diagnosis.

4. **Endocrine causes** of hypertension especially primary aldosteronism is occasionally seen, the characteristic feature of this being persistent hypokalemia. Cushing's syndrome with its typical clinical features of moon facies, truncal obesity and purplish abdominal striae may be seen but this can be difficult to diagnose in pregnancy. Phaeochromocytoma is another rare cause classically presenting with episodic headache, palpitations, sweating, anxiety, tremors and hypertension due to excessive secretion of adrenaline and noradrenaline. Urinary excretion of vanillylmandelic acid (a metabolite of catecholamines) is the usual test done to diagnose this condition.

The diagnosis of secondary hypertension can be difficult and a high index of suspicion is required. In summary, the features that should alert the clinician are:

- Persistent hypokalemia
- Abdominal bruit
- Variable pressures with tachycardia, sweating and tremor
- History of renal disease

Section 5.3

Case Studies

INSTRUCTION FOR STUDENTS

GUIDELINES FOR CASE STUDY	
YOUR CASE STUDY MUST CONCERN SOME ASPECT OF MANAGEMENT OF PRE-ECLAMPSIA OR ECLAMPSIA. It should include the following:	
Case number : (This will enable the case record to be traced if needed but will protect the confidentiality of the woman).	
Age :	
Parity :	
Date of the first day of the last menstrual period (LMP)	
Estimated date of delivery (EDD)	
Social background :	
Past obstetric history :	
Relevant medical and surgical history:	
History and course of present pregnancy, labour and puerperium	
SUMMARY OF CARE AND MANAGEMENT TO DATE	
You will be required to discuss the following important issues	
1. What happened? This will include details of the condition of the woman after delivery?	This is the outcome
2. What risk factors to eclampsia were present (eg. Primigravida, twin pregnancy, pre-eclampsia or other risk)?	This is the process
3. How were pregnancy, labour and postnatal care managed?	

4. Summarize the main points of midwifery practice. Emphasizing how to case was managed	This is considers the relationship between process and outcome
5. Where any oppotunities missed? Factors may have been overlooked which, in another woman, would have resulted in maternal death. In cases of death, ask: Was this avoidable?	This demonstrates what can be learned through experience.

CASE STUDY 1

A 19 year old Malay girl G1P0 at 32 weeks of gestation was referred to a general hospital. She was booked at 24 weeks at a government health clinic. Her booking BP was 140/90mmHg. She only had one more antenatal visit at 28 weeks when BP was noted to be 150/90mmHg, no proteinuria was noted.

On the day of the referral, the neighbour found her lying on the floor of her house unconscious. She was immediately sent to the hospital. On the way she threw a fit, with uprolling of the eyeballs and tonic clonic movements of both her limbs.

On arrival at the A&E of the general hospital, she fittted again. Her BP was 200/120mmHg and proteinuria 4+. She was disoriented. There was no fetal heart present. She then started gasping. She was immediately intubated and resuscitated. However she collapsed and despite active resuscitation was pronounced dead about 50 minutes after arrival.

Q1 What is your diagnosis?

Q2 How would you have managed her if you had seen her at 24 weeks of pregnancy?

Q3 What are the risk factors in this patient?

Q4 How would you have managed her if you had attended to her at home?

CASE STUDY 2

A 24 year old Chinese primigravida was referred from a Health Clinic at 39 weeks of gestation with history of giddiness of 3 days duration. She was booked at 23 weeks and was normotensive. She had a total of 6 antenatal visits, which were uneventful.

On arrival to the General Hospital she was comfortable and asymptomatic, BP was 150/90 mmHg and proteinuria 1+. CTG was reactive. It was decided for Prostin induction coming morning.

Q1 *Comment on the management so far.*

Four hours after admission the patient complained of headache (increasing in severity), blurring of vision and she vomited twice. Her BP was 180/120mmHg and a repeat proteinuria was 4+. Emergency LSCS was done for impending eclampsia. The operation was uneventful and a healthy baby boy of 3.2 kg AS 9@1 minute, was delivered. Patient was then monitored in the HDU (high dependency unit). She was started on Diazepam infusion and Tab. Labetolol 200mg tds.

Q2 *Was the management correct?*

Suddenly, 11 hours after the operation the patient threw a fit lasting about 3 minutes. It was tonic-clonic in nature with up rolling of the eyeballs. Fit was aborted with IV Diazepam. Patient remained drowsy and confused. Her BP was then 200/110mmHg. She remained drowsy and was referred to the anaesthetist.

Q3 *Discuss the role of the anaesthetist.*

She had 48 hours of cerebral resuscitation following which she made a slow but successful recovery. She was discharged well on 10th day after delivery.

Q4 *Discuss the subsequent management of this patient.*

CASE STUDY 3

A 30 year old Indian lady, G3P1+1 was booked at the district hospital at 20 weeks of gestation. Her booking BP was 150/90mmHg and there was no proteinuria. Blood investigations including Haemoglobin, Platelet count, BUSE, uric acid and creatinine were sent for.

She was seen 2 weeks later and her BP was then 150/100mmHg and asymptomatic. All her results were normal. She was started on Tab. Methyldopa 250mg tds. and given an appointment in two weeks.

Q1 If you saw the patient now, what would your management be?

However she defaulted treatment, and was only seen again at 32 weeks in the labour room with per vaginal bleeding and severe abdominal pain. Her B/P was 190/120mmHg and proteinuria 3+, pale and HR 132/min. The abdomen was tender and fetal heart was absent. She was dilated to 5 cm and ARM revealed blood-stained liquor.

Q2 What could be the contributing factors for the patient defaulting treatment?

Q3 What is your Diagnosis?

She was started on hydralazine infusion and a coagulation profile done. She delivered vaginally to a fresh stillbirth, baby boy weighing 1.3 kg. There was about 600mls of retroplacental clots evacuated. She was transfused with blood and FFP. She recovered well and was discharged on day 5 post delivery.

Q4 How would you manage the immediate post partum?

CASE STUDY 4

A 22 year old G2P0+1 at 32 weeks, not known to have HDP, was referred from a district hospital for impending eclampsia.

Q1 *What are the significant symptoms of impending eclampsia?*

Q2 *Discuss the role of antenatal steroids in this patient.*

On arrival she was complaining of headache, nausea and blurring of vision. Her BP was 170/120mmHg and the proteinuria was solid. She was planned for an emergency LSCS after her B/P was stabilised with Hydralazine infusion as the cervical (Bishop) score was unfavourable.

Q3 *How would you commence Hydralazine infusion in this patient?*

Half hour after the infusion was started, she suddenly threw a fit lasting about 1 min. It was tonic-clonic in nature and was aborted with iv. Diazepam 10mg. Patient was stabilised and had an uneventful LSCS, following which she was nursed in ICU and ventilated for 24 hours.

The hypertension was difficult to control and both Tab. Methyldopa 500mg and Tab. Labetolol 200mg tds were prescribed.

Q4 *When would you discharge this patient?*

She made a complete recovery and discharged on the 8th post-operative day with Tab.Labetolol 200mg tds. She was advised to be followed up at the district hospital.

Q5 *What are the side effects of Labetolol?*

CASE STUDY 5

A 32 year old pregnant lady was found fitting at a bus stand and was rushed to the A&E department of a general hospital. On arrival, the patient was arousable but disorientated and was bleeding from her lower lip.

Her BP 120/80mmHg, PR 98/^{min} and no proteinuria was noted. Physical examination revealed a gravid uterus of 28 weeks. A ⁹⁵er systems were normal. The patient regained consciousness about an hour later.

Q1 *What is your differential diagnosis?*

Q2 *What further history would you like to elicit from this patient?*

Patient then admitted that she has been an epileptic since 18 years of age and had been on medication, which she had stopped on her own since she got pregnant.

An ultrasound showed a viable pregnancy with a normal foetus. She was then referred to a neurologist and combined care provided.

She had an uneventful antenatal follow up and a successful outcome.

CASE STUDY 6

A 22 year old Malay lady, who was pregnant 18 weeks, fainted at her factory and was sent to the hospital.

On arrival she was disorientated and not arousable, her BP 100/60mmHg, PR 100/min. and urine protein was absent. A glucometer reading showed 1.9mmol/l.

*Q1 **What is your diagnosis?***

*Q2 **What are the possible causes of her condition?***

She was immediately given 50cc of 50% glucose. Following that she became alert and orientated. All systematic investigations were normal.

Retrospective history revealed that she had been fasting and had felt faint, following which she fainted which was tonic in nature i.e. flexion of her upper and lower limbs. No clonic phase was noted.

She made a complete recovery and had a FTSVD (full term spontaneous vertex delivery).

ANSWERS TO CASE STUDIES

CASE STUDY 1

Q1 Eclampsia

Q2 She should have been referred to a hospital

Q3

- i) young age*
- ii) primigravida*
- iii) had pregnancy induced hypertension (PIH) at booking*
- iv) poor antenatal follow up*

Q4 Refer to management in transit (Section 4)

CASE STUDY 2

Q1 Acceptable management plan

Q2 She had an emergency LSCS done for impending eclampsia, which is acceptable management. Use of MgSO₄ should be considered.

Q3 Refer to Section 3.5

Q4 Refer to Section 3.7

CASE STUDY 3

Q 1 Management plan is acceptable but patient should have been counselled on her condition and the need for close antenatal follow-up.

Q 2

- i) Patient unaware of the seriousness of her condition*
- ii) Lack of patient education*
- iii) Social problems*
- iv) Financial problems*

Q 3. Abruptio Placenta

- Q 4
- i) Initiate red alert***
 - ii) Resuscitation with blood and blood products***
 - iii) Consider delivery after stabilization***
 - iv) Priority is always the mother***

CASE STUDY 4

- Q 1
- i) Headache***
 - ii) Blurring of vision***
 - iii) Nausea***
 - iv) Vomiting***
 - v) Epigastric pain***
 - vi) Hypertension***
 - vii) Oedema***
 - viii) Proteinuria***
 - ix) Hypereflexia***

Q 2 ***Refer to Section 3.2***

Q 3 ***Refer to Section 6.1***

Q 4 ***Refer to Section 3.2***

Q 5 ***Refer to Section 6.1***

CASE STUDY 5

Q 1 *Refer to Section 5.1*

- Q 2**
- i.** *History of fits previously and if so what medication she is on.*
 - ii.** *Family history of fits*
 - iii.** *Similar incidents in previous pregnancies*

CASE STUDY 6

Q 1 *Hypoglycaemia*

- Q 2**
- i.** *Undiagnosed diabetes*
 - ii.** *Patient is fasting*
 - iii.** *Possible overdose of hypoglycaemic agents*

SECTION 6
APPENDICES

Section 6.1

Basic Facts on Common Drugs Used in the Management of Hypertension

1. **Methyldopa** is a centrally acting antihypertensive drug that is altered in the CNS to alpha-methylnorepinephrine, which stimulates inhibitory alpha-2 adrenergic receptors in the hypothalamus (inhibits sympathetic nervous system outflow from the vasomotor centre to the periphery). Available in tablets, suspension and injection.

Cardiovascular effects are a decrease in systemic vascular resistance and blood pressure, whereas cardiac output (renal, cerebral and myocardial blood flow) is maintained.

Contraindications are acute hepatic disease, history of depression, and phaeochromocytoma

Side-effects are sedation, depression nightmares, nasal congestion, hemolytic anemia and liver disorders.

2. **Hydralazine** decreases blood pressure by exerting a direct relaxant effect on vascular smooth muscle (arterioles greater than veins). Available in tablets and injections.

Cardiovascular effects include the preferential dilation of arterioles compared with veins minimizes orthostatic hypotension and promotes an increase in cardiac output (stroke volume and heart rate increases).

Contraindication is tachycardia.

Side-effects are reflex tachycardia, sodium and water retention, vertigo, myocardial stimulation, SLE-like syndrome.

3. **Labetolol** exhibits selective alpha-1 antagonist and nonselective beta-2 antagonist effects following oral or intravenous administration. Available in tablets and injections.

CVS effects : it acutely lowers BP by decreasing systemic vascular resistance and reflex tachycardia triggered by vasodilation is attenuated by simultaneous beta blockade

Contraindication is partial heart block, CCF.

Side-effects are fluid retention, orthostatic hypotension, bronchospasm, cardiac failure.

4. **Calcium Channel Blockers**

Two common drugs used in pregnancy:

- i) Nicardipine
- ii) Nifedipine

Mechanism of action

Calcium channel blocker inhibits the passage of calcium through the voltage gate L type of membrane channels of smooth and cardiac muscle, reduces available intracellular calcium and causes muscles to relax and hence vasodilation occurs.

Some of the group have weakly negative cardiac inotropic action and negative chronotropic effect via pacemaker cells and depress conducting tissue.

Pharmacokinetics

Generally all Calcium channel blockers are well absorbed from the gut and are metabolised by the liver.

Adverse Effects

Headache

Flushing

Dizziness

Palpitations

Lethargy

Hypotension – may occur during 1st few hours after dosing

Ankle oedema

Bradycardia

GIT effects - constipation

- nausea

- vomiting

Gum hypertrophy (Nifedipine)

Individual Calcium blockers:

Nifedipine has greater coronary and peripheral arterial vasodilator properties than verapamil.

There is minimal effect on venous capacitance vessels. Has little or no depressant activity on sinoatrial or atrioventricular nodal activity.

Peripheral vasodilation and resultant decrease in blood pressure produced by nifedipine activate baroreceptors leading to increased peripheral sympathetic nervous system activity, most often manifesting as tachycardia.

Side effects are headaches and flushing. Abrupt discontinuation of nifedipine has been associated with coronary artery spasm.

(Half-life) T_{1/2} : 2 hours

Selectively dilates arteries with little effect on veins

Can be given sublingually or orally

Dosage 30 – 60 mg daily

5. **Parenteral Antihypertensives**

Aim to keep diastolic blood pressure between 90-95 mmHg

Labetolol is contraindicated in Bronchial Asthma, CCF and Atrio-Ventricular Heart Blocks.

i) **Labetolol Infusion**

a) For rapid control

- I/V Labetolol 10 mg (2 mls) over 1 minute and repeat at 5 minute intervals (Maximum dose : 200 mg (40 mls))
- Effective dose : 20-150 mg/hr (4-30 mls/hr)
- Infusion syringe pump (put 200 mg or 40 mls Labetolol in 50 mls syringe and start at 20 mg/hr ie. 20 mg or 4 mls/hr and increase at 30 minutes. Stop infusion if rate exceeds 150 mg/hr (30 mls/hr) and inform specialist.

ii) **Hydralazine Infusion**

a) For rapid control

- I/V bolus 6.25 mg over 20 minutes and repeat every 20 minutes only if DBP >90 mmHg (1-10 mg/hr infusion is preferred)

b) Maintenance Dose

- Effective dose : 1-10 mg/hr
- Infusion Pump
- Dilute 50 mg Hydralazine in 50 mls Normal saline ie. 1 mg/ml and start at 5 mls/hr

Increase every 20 minutes by 1 ml/hr until maximum dose of 10 mls/hr or 10 mg/hr

- Infusion Drip Set

- Dilute 20 mg Hydralazine in 500 mls Normal Saline and start at 10 dpm.
Increase every 20 minutes at 10 dpm to titrate against blood pressure so as to maintain at 140/90 mmHg.
- Prevent fluid over load.

Antihypertensive Drugs Commonly Used In Pregnancy

Drug	Mode of action	Start. Dosage (mg/day)	Max. dosage (mg/day)	Half-life T1/2 (hours)	Adverse effects
Methyldopa	Centrally-acting (false transmitter precursor)	250	3000	1.8	Depression, drowsiness, lupus-like syndrome, blood dyscrasias, liver dysfunction
Labetolol	α/β - Blockers	100	2000	4	Complete heart. block, pulmonary odema, bronchoconstriction
Nifedipine	Ca-channel blocker	15	60	3.4	Headaches, flushing
Hydralazine	Vasodilator	25	300	2.2-2.6	Tachycardia, hypotension, headache tachyphlyaxis

Drugs and Fetal Risks

Drug	Fetal risk
Methyldopa	Positive Coomb's test in fetus, doses > 2g/day meconium ileus
Hydralazine	Fetal heart rate changes when given acutely at term
Magnesium sulphate	Bladder atony & myotonia but these are rare
β-Blockers	IUGR, hypotonia at birth, neonatal bradycardia, hypoglycaemia

Drugs Used In Eclampsia

Drug	Mode of action	Dosage	Adverse effects
Diazepam	GABA receptors, chloride gates	bolus 10mg, infusion 40mg & titrate	Respiratory. depression, tolerance, drowsiness
Magnesium sulphate	Cerebral depressant, reverses cerebral vasoconstriction	See text	Respiratory arrest, arrhythmias, oliguria

6. Anticonvulsant Therapy in HDP

MAGNESIUM SULPHATE

This drug has gained prominence in the management of convulsions in HDP.

It is available as $\text{MgSO}_4 \cdot \text{H}_2\text{O}$ – 50% solution. This contains 50 gm in 100ml solution i.e.: 5ml ampule contains 2.5 gm MgSO_4 ; 20% solutions are also available.

The drug can be administered intravenously or intramuscularly.

- 50% solution is suitable for intramuscular use
- 20% solution is suitable for intravenous route

Administration

- Intravenous
- Intramuscular

Intravenous route is preferred to intramuscular route which is painful and is complicated by local abscess formation.

a) **Loading dose:** I/V 4 gm MgSO_4 slow bolus

- An initial dose of 4 gm MgSO_4 is given over 10-15 mins (rapid injection causes cardiac arrest)
- 4 gms (8 mls) MgSO_4 is diluted in 12 mls Normal Saline or sterile water to a total volume of 20 mls
- If further convulsions persists after 15 minutes, a further 2 gms MgSO_4 is diluted and given over 15 minutes

b) **Maintenance Treatment** IV 1 gm/hour MgSO_4

- Syringe Pump
Ideally MgSO_4 is given by a syringe infusion pump

2 mls MgSO_4 is diluted in 48 mls of 5% Dextrose and infused at 50 ml/per hour

Drip Infusion Set

5 mg MgSO_4 (10 mls) in 500 mls 5% Dextrose is run at 33 drops per minute

This infusion is only continued if the following criteria are satisfied

- Patellar (knee jerk) reflex is present
- Respiratory rate > 16/min
- Urine Output >100 mls over 4 hours
- Serum Magnesium level are within therapeutic range of 1.7 – 3.5 mmol/L

- **Intramuscular Loading Dose**

10 gm 50% solution (20 mls) is injected intramuscularly. One half is injected into upper outer quadrant of each buttock in zigzag manner (preceded by local anaesthesia if necessary) using a 21 gauge needle and this is followed by a maintenance dose every 4 hours.

Caution: The loading dose of MgSO_4 must be reduced in oliguria.

- **Maintenance Dose for Intramuscular Route**

5 gm 50% solution is injected deep intramuscular in alternate buttocks every 4 hours after ascertaining that:

- a) Knee (patellar) jerk is present
- b) Respiratory rate is > 16/min
- c) Urine output >100 ml/4 hours

Caution

1. Patients who have received diazepam should not be administered intravenous loading dose. Only intramuscular loading dose should be given.
2. If convulsions recur, after loading 2 gm 20% solution (10 ml) intravenous can be repeated slowly over 3 minutes
3. If reflexes are absent, recheck at half hourly intervals.

The therapeutic plasma concentration is 4-7 mmol/l

Magnesium sulphate is eliminated by **the kidneys**.

c) **Mechanism of Action**

Magnesium sulphate is not an anticonvulsant but it does **relax vascular smooth muscle**, therefore it is likely that magnesium sulphate acts by **reversing cerebral vasoconstriction**.

Magnesium sulphate is an effective **cerebral depressant** and hence reduces neuromuscular irritability.

d) **Recurrent convulsions**

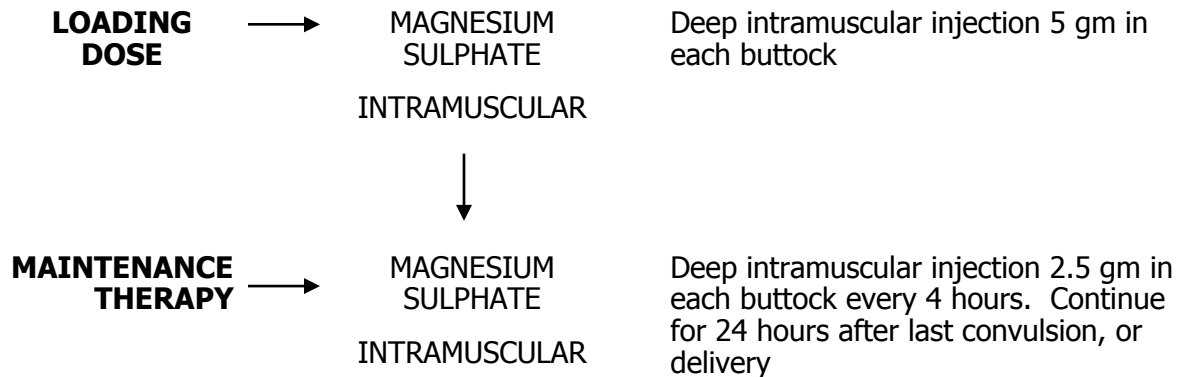
Both intramuscular and intravenous regimens:

Further 2-4 grams (depends on weight, 2 gram if <70 kg) to be given

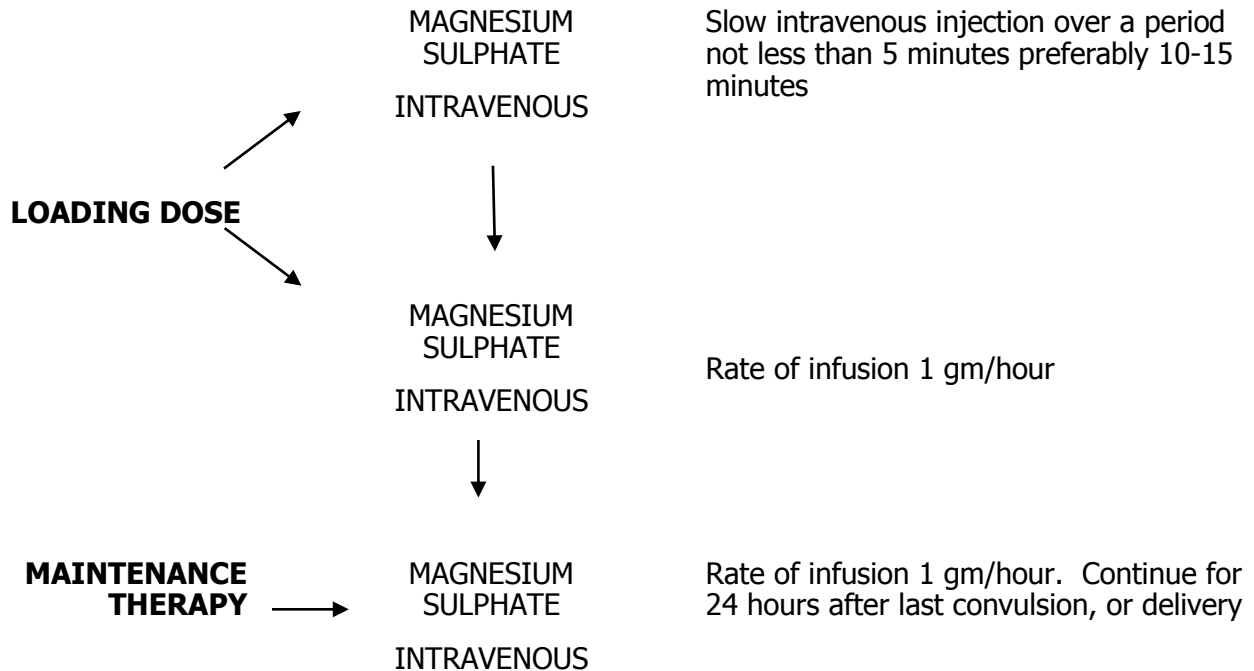
Intravenously over 5 min.

MAGNESIUM SULPHATE REGIMENS FOR WOMEN WITH ECLAMPSIA

INTRAMUSCULAR MAINTENANCE REGIMEN



INTRAVENOUS MAINTENANCE REGIMEN



e) **Monitoring During Magnesium Sulphate Therapy**

The next intra-muscular dose can only be given or the intravenous infusion can only be continued if:

- Respiratory rate > 16/min - check every 15 min
- Urine output > 25 ml/hr - check every hour
- Patellar reflexes are present - check every 15 min

Intravenous regime needs more frequent monitoring. In the first 2 hours monitoring should be every 10 min.

f) **Side-effects and Toxicity of Magnesium Sulphate**

Effects associated with various serum magnesium levels

Effects	Serum level mmol/l
Anticonvulsant prophylaxis	4 - 6
ECG changes	5 – 10
Loss of deep tendon reflexes	10
Respiratory paralysis	15
General anaesthesia	15
Cardiac arrest	>25

Other side effect : May enhance the action of curare-like drugs and Ca channel blocker

Effects on newborn : Magnesium sulphate crosses the placenta freely, however there is little evidence of toxicity eg. hypermagnesaemia associated with hyperreflexia and respiratory depression, provided maternal serum limits are observed.

g) Steps to be taken in Magnesium Toxicity

The following guidelines are provided for management of the potential complications of magnesium sulphate.

Respiratory arrest

- Intubate and ventilate immediately
- Stop magnesium therapy
- Give 1 g IV of calcium gluconate

Respiratory depression

- Give oxygen by mask
- Stop magnesium therapy
- Give 1g IV of calcium gluconate
- Maintain the airway
- Nurse in the recovery position

Absent patellar reflexes

- If respiration normal - withhold further doses of magnesium sulphate until the reflexes return
- If respiration depressed - manage as above

Urine output <100ml in 4 h

If no other signs of magnesium toxicity, reduce the next dose

IM dose to 2.5g OR

IV dose to 0.5g/h

If there are other signs of magnesium toxicity, manage as for the appropriate section above.

h) **Magnesium Sulphate Pack**

The entire components are kept in a readily available portable carrier container.

○ **Intravenous Infusion**

- i) 500 ml Normal Saline
- ii) Intravenous cannula
- iii) Infusion giving set
- iv) Tape to secure cannula
- v) Swab to clean skin

○ **Magnesium Sulphate Pack**

- i) 4 g loading dose
- ii) 5 x 5 gm maintenance dose
- iii) 5 gm (for recurrent convulsions)
- iv) Pharmacology of MgSO₄

- Available as MgSO₄ H₂O – 50% solution. This contains 50 mg 100 ml solution ie. : 5ml ampoule contains 2.5 gm MgSO₄
- Drug can be administered **intravenously** or intramuscularly. 50% solution is suitable for intramuscular use, 20% solution is suitable for intravenous route.
- Magnesium sulphate is cleared by the kidney, hence the dose must be reduced if there is impairment of renal function. In the presence of oliguria, cumulative toxicity can result with repeated doses; thus dose must be reduced or omitted in such situations.
- Serum levels of MgSO₄ correlate with clinical signs. MgSO₄ depresses neuromuscular transmission and can cause muscular paralysis. Loss of patellar (knee) reflex is usually the first clinical manifestation of toxicity. Respiratory depression follows if levels go higher. Thus laboratory testing of serum MgSO₄ may be unnecessary.

- **Calcium Gluconate**

- i) 1 gm (intravenous) for toxicity

- **Observation Chart**

- i) Fluid Chart
 - ii) BP/Pulse/Respiratory rate/knee jerk reflexes

- **Protocol**

- i) Summary Flow Chart
 - ii) Detailed Regime
 - iii) Guidelines for other aspects of care

- i) Route of administration of MgSO₄**

- There is no evidence of any difference between intramuscular and intravenous regimes in their effects on recurrent convulsion except that intramuscular injections are painful and have a 0.5% risk of abscess formation
- All staff must be familiar with both routes of administration. Repeat doses are only given when the respiratory rate is >16/minute and the knee reflexes are present
- The intramuscular route is especially convenient where infusion sets are not available.

DIAZEPAM

Diazepam is a minor tranquilliser that has been used as an anti-convulsant

Advantages:

- a) readily available
- b) cheap
- c) easy to administer

Disadvantages:

- a) Profound maternal sedation
- b) Respiratory depression
- c) Loss of fetal heart variability
- d) Neonatal hypotonia and poor sucking
- e) Tachyphylaxis

Dosage and Administration

- The recommended regime is
 - 40 mg Diazepam in 500 ml of 5% Dextrose/Normal saline (preferably glass container)
 - Infusion rate is titrated against patient's level of consciousness ie to keep her drowsy but arousable
 - Regime to continue for 24 hours after the last convulsion and to half the concentration for the next 24 hours
 - Recurrent convulsions can be managed with an additional IV injection of 5-10 mg over 1-2 min
 - Patient should be nursed in the HDU/ICU

Section 6.2

Fluid Regimen for Patients with HDP

In pre eclampsia/eclampsia there is diminished intravascular volume, high systemic vascular resistance and low colloid osmotic pressure. In view of this, infusion of fluids must be cautiously undertaken. The appropriate use of intravenous fluids in terms of both fluid type and quantity may influence morbidity and mortality. In the extreme, acute pulmonary oedema is a leading cause of death in women with pre-eclampsia and a frequent cause for admission to intensive care ¹. In observational studies the use of either crystalloid or colloid solutions has been associated with transient improvements in maternal cardiovascular system parameters. However in one large trial and a systematic review volume expansion demonstrated no advantages compared with no plasma volume expansion. ^{2, 3}

Also, with the available evidence the use of intravenous fluids to increase plasma volume or treat oliguria in a woman with normal renal function and stable serum creatinine levels cannot be recommended (Duley et al 1999, **Level I**)⁴

For practical purposes close monitoring of fluid intake and urine output is mandatory.

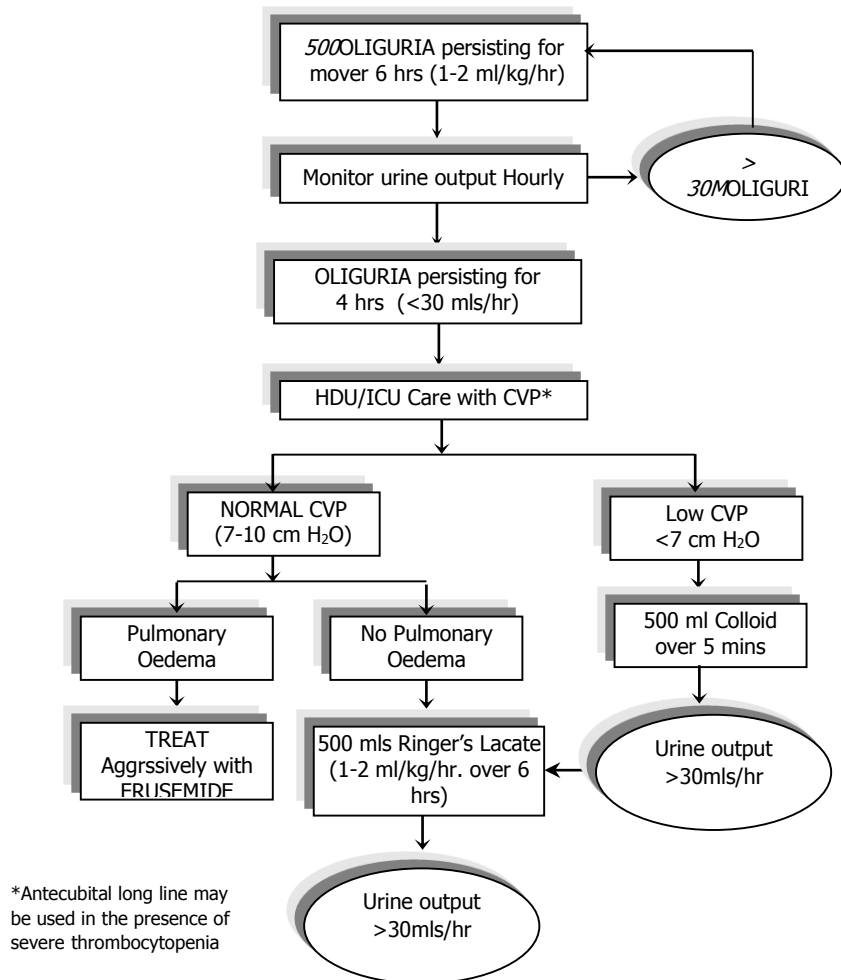
The presence or absence of pulmonary oedema can be assessed clinically by auscultation for basal crepitation, tachypnoea and continuous measurement of oxygen saturation using a pulse oximeter.

Suggested total fluid replacement: (See Fig. 1)

- Should not exceed 1-2 ml/kg/h¹ or 85 ml/hr whichever is lower.
- Urine output of more than 30 ml/h (0.5 ml – 1 ml/kg/h) should be maintained
- If CVP line is present, then the CVP level should not be higher than 7 cm of H₂O
- If the patient has started taking oral fluids, then this amount should be taken away from the intravenous fluid regimen of 1-2 ml/kg/h
- If pulmonary oedema develops i/v Frusemide 40 mg should be given, oxygen administered and patient managed in ICU
- If oliguria develops and urine output is less than 30 ml/h for 4 hours, then a fluid challenge with 200 ml of crystalloid solution over 5 minutes should be tried. Evaluation should be done at 4 hours period because in most cases the kidneys will recover. If oliguria persists and CVP is above 7-10 cm H₂O, then referral should be made to the Physician for further management.

Attention to fluid management is vital in view of the contracted plasma volume in PIH/Eclampsia. Central venous pressure measurements in an HDU/ICU setting may be warranted to assist fluid management. The following regime is suggested in addition to the usual treatment protocol outlined in the manual (see Editor's Comment **Section 3.5**).

Fig. 1: Fluid Regimen



Types of Intravenous Fluids

The types of intravenous fluids that can be used in the management of patients with HDP are:

- (i) crystalloids
- (ii) colloids

Composition of common intravenous fluids:

(i) Crystalloids:

Name	pH	Osmolality Mosmol /litre	Na+ mmol/litre	K+ mmol/litre	Cl-mmol/litre	HCO3 mmol/litre	Misc. mmol/litre	Carbohydrate /litre	Protein g/litre
Sodium Chloride 0.9%	5.0	308	154	0	154	0	0	0	0
Ringer's Lactate	6.5	280	131	5	112	29	Ca 1 Mg 1	0	0
Glucose 5% + NaCl 0.9%	4.5	585	154	0	154	0	0	50	0
Glucose 5%	4.0	280	0	0	0	0	0	50	0
Glucose 4% + NaCl 0.18%	4.5	286	31	0	31	0	0	40	0

Normal Saline:

Is essentially isotonic with human plasma and contains sodium as the primary osmotically active particle. It distributes evenly throughout the extracellular space. About $\frac{1}{4}$ of the infused volume remains in the intravascular space after one hour. After infusion equilibration with the extracellular space occurs within 20 to 30 minutes. Normal Saline is used as a replacement fluid.

Ringer's Lactate:

This is a balanced salt solution and it equilibrates with the extracellular space within 20 to 30 minutes and is mainly used as a replacement fluid. The lactate is metabolised in the liver to form bicarbonate, to counteract acidosis. If normal saline or Ringer's lactate solution is used to replace lost blood volume at least 3 times the volume of blood lost, must be infused.

Dextrose/Saline solutions:

These are usually maintenance fluids. Five percent dextrose solution is isotonic and may be used for fluid maintenance and to keep an intravenous route open for medication. When stored blood is followed by glucose solution, rouleaux formation takes place. This causes clumping in the drip set. Dextrose 5% is distributed throughout all body fluids and is ineffective as a volume expander.

(ii) Colloids:

Name	pH	Oncotic Pressure (cm H ₂ O)	Na+ mmol/litre	K+ mmol/litre	Cl-mmol/litre	Misc. mmol/litre	Carbo Hydrateg/litre	Protien g/litre	Half-life in Plasma (hours)
Gelatin (succinylated urea. Haemaccel)	7.4	37	145	5.1	145	Ca 6.25 PO ₄ trace SO ₄ trace	0	35	5 h
Gelatin (polygeline. Gelofusin)	7.4	46.5	154	0.4	125	Ca 0.4 Mg 0.4	0	40	4 h
Hetastarch (Hespan)	5.5	31	154	0	154	0	0	0	17 days

Colloids:

Colloids maintain or increase plasma oncotic pressure and so help to draw fluid into the intravascular space. Examples of colloids in clinical use are gelatins, starches and albumin.

- **Gelatins :**
These are produced by the hydrolysis of collagens. Gelatins have an incidence of adverse reactions (1 in 2,000 to 13,000 have been reported).
They have a long shelf- life and are of reasonable cost. Risk of transmissible diseases is not there.
 - Haemaccel:** is a 3.5% urea linked gelatin with a molecular weight of 35,000 in electrolyte solution. As Haemaccel contains calcium, it should not be given in the same infusion set as citrated blood and fresh frozen plasma.
 - Gelofusine :** is a 4% succinylated modified fluid gelatin in normal saline. It has a molecular weight of 30,000 and pH of 7.4. It has a biological half-life of about 4 hours. The duration of useful plasma expansion is about 2 hours and 85% is excreted by the kidneys.

- **Starch:**

Hetastarch: is a synthetic colloid derived from corn starch. Its molecular weight ranges from 10,000 to over one million. Intravascular volume is expanded to the same volume as that infused, and this state may last for about 3 hours. Smaller molecules, with molecular weight less than 50,000 will be excreted in urine, and the larger molecules will undergo hydroxyethylation before being eliminated. Its biological half-life is about 17 days.³ The recommended volume is 500ml to 1000ml per day for a 70 kg adult. Over-infusion can lead to pulmonary oedema. The incidence of anaphylatic reaction to hetastarch is less than 0.085%.^{3,4}

- **Albumin:** has a molecular weight of 66,300 to 69,000 and exerts 80% of the plasma colloid oncotic pressure. Its half-life is 16 hours. Albumin is used in the resuscitation of patients with an acutely diminished intravascular volume to draw in fluid from the extravascular space into the intravascular space. When 100ml of 25% albumin is infused, intravascular volume increases to 450ml in 30 to 60 minutes.³ Caution has to be exercised as over-infusion can easily lead to pulmonary oedema. Anaphylatic reaction to albumin is between 0.47% and 1.53%.^{3,4}

References:

1. *Sriram S & Robertson MS 2008, 'Critically ill obstetric patients in Australia: a retrospective audit of 8 years' experience in a tertiary intensive care unit', Crit Care Resusc, vol. 10, pp.124.*
2. *Duley L, Williams J & Henderson-Smart DJ 1999, 'Plasma volume expansion for treatment of pre-eclampsia. Cochrane Database of Systematic Reviews, Issue 4. Art. No.: CD001805. DOI: 10.1002/14651858.CD001805.*
3. *Ganzevoort W, Rep A, Bonse GJ, Fetter WP, van Sonderen, de Vries JI & Wolf H 2005, 'A randomised controlled trial comparing two temporising management strategies, one with and one without plasma volume expansion, for severe and early onset pre-eclampsia', BJOG, vol. 112, pp. 1358-68.*
4. *Mushambi MC., Halligan AW., Williamson K.. Recent Developments in the Pathophysiology and Management of Pre eclampsia. Br. J. Anaesth. 1996; 76: 133-148.*
5. *Shennan A., de Swiet M., Protocol for the Management of Severe Pre eclampsia/Eclampsia., Queen Charlotte's Hospital London 1999.*
6. *Pharmacologic Approach to the Critically Ill Patient., Baltimore: Williams & Wilkins; 1988: 219-40.*
4. *Clinical Pharmacology. 1993; 12: 415-28.*

Section 6.3

Recommended Techniques in Taking Blood Pressure

1. BLOOD PRESSURE

Blood pressure is the force exerted by the blood as it flows through the blood vessels.

2. EQUIPMENT

- Sphygmomanometer with cuff
- Stethoscope

Mercury sphygmomanometers remain the gold standard for measurement of blood pressure in pregnancy. While such automated devices may give similar mean blood pressure values to those obtained with mercury sphygmomanometry, there is wide intra-individual error and their accuracy may be further compromised in pre-eclamptic women^{1,2}. All devices should be calibrated on a regular basis (ideally monthly), as recommended by the British Hypertension Society

Cuff

The cuff is a rubber bag that can be filled with air. It is generally covered with a cloth and has two rubber tubes attached to it. One tube is attached to the rubber bulb that blows up the cuff. The other tube is attached to a manometer indicating the pressure of air within the cuff. Blood pressure cuffs come in six standard sizes. Some cuffs hook together, others wrap or snap in place. The use of appropriate size and length of cuff is important for accurate reading.

The cuff should be wide enough to cover about two-thirds of the arm or upper thigh or be 40% of the circumference of the mid-point of the limb. The cuff needs to be long enough to completely encircle the limb. Too narrow a cuff can result in erroneously high reading, while too wide a cuff can produce low readings.

3. RECOMMENDED STANDARD METHOD FOR THE MEASUREMENT OF BLOOD PRESSURE BY SPHYGMOMANOMETRY

3.1. Preparation

a) Preparation

Remove any tight clothing so that the upper arm is fully exposed and the sphygmomanometer cuff can be easily applied.

b) **Position**

Patient can be comfortably seated with the examination arm resting on a table or the patient rests comfortably on a couch or bed on her right side with 15-30° tilt and that the right upper arm is well supported at the same level as the heart.

c) **Sphygmomanometer**

Position the sphygmomanometer so that the 90mmHg mark is at eye-level when the blood pressure is taken with the stethoscope fixed in the ears and the bell applied to the antecubital fossa.

3.2. **Application of cuff**

a) **Position**

Place the center of the bladder in the sphygmomanometer cuff directly over the brachial artery on the inner side of the right upper arm with the cuff at the same level as the sternum at the 4th intercostal space.

b) **Application**

Apply the cuff evenly and firmly but not tightly around the arm with the connecting tubes pointing upward (toward the head) and the antecubital fossa free.

c) **Size of cuff**

If possible use a cuff with the acceptable range of arm circumference marked on the sleeve. If arm circumference is too large, use a large cuff. If no large cuff is available, note that the blood pressure reading is unsatisfactory and may be erroneous.

All clinics should have appropriate sized cuffs available.

3.3. **Taking the blood pressure**

a) **Position of stethoscope**

Palpate the brachial artery in the antecubital fossa and place the stethoscope directly over the artery and hold in place without undue pressure.

b) **Initial cuff pressure**

Rapidly pump up the pressure in the sphygmomanometer cuff to 20-30 mmHg above the point at which pulsation in the brachial and radial artery cease and the Korotkoff's sounds disappear.

c) **Rate of deflation of cuff**

Let the air out of cuff without delay so that mercury falls steadily at 2-3 mm/s

d) **Systolic blood pressure**

Take the systolic blood pressure as the point where first clear tapping sound is heard, read top of mercury meniscus, record to nearest 2 or 5 mmHg.

e) **Diastolic blood pressure**

Take diastolic blood pressure in pregnancy as the point where the Korotkoff's sounds first become muffled (Phase IV), read the top of mercury meniscus to nearest 2-5 mmHg. If no clear point of muffling is heard, take point of disappearance of sounds (Phase V) and record which point taken. Ideally, both point of muffling (Phase IV) and of disappearance (Phase V) should be recorded.

f) **Complete deflation of cuff**

Let down pressure in the cuff completely as soon as blood pressure is taken to minimise patient discomfort and to allow free flow of blood in and out of arm.

3.4. **Choice of arm**

a) **Right arm preference**

The blood pressure should normally be taken in the right arm as most observers stand on the right side of the patient when taking the blood pressure.

b) **Both arms at first visit (Preferable)**

At first visit take blood pressure in both arms. It can be in a comfortably seated or in the lying position. First take blood pressure in right arm and then turn patient on left side with 30° tilt with left arm well supported at level of heart. Ensure patient rests comfortably for 5 min and take blood pressure in left arm the same way as in right arm and record all measurements.

c) **If BP in left arm higher**

If blood pressure in left arm is 10 mmHg or more than in right arm, make special note in records that the left arm should be used for all future measurements.

3.5. **Repeat measurements if blood pressure is uncertain or high**

a) **If BP uncertain**

If the blood pressure reading is uncertain, always repeat measurement. Let cuff down completely and wait 5 min before re-inflating cuff and repeating measurement.

b) **If BP is taken in sitting position and found to be raised**

Arrange that patient lies down on couch on right side with 30° tilt, rests a few minutes and take blood pressure as described above.

c) **If BP is raised in lying position**

Ensure patient rests comfortably for 5 min, repeat blood pressure measurement and if necessary get second experienced observer to check. Record all measurements both initial and repeat and act on lowest DBP (phase IV) unless it is falsely low measurement.

4. **KOROTKOFF'S SOUNDS**

Phase I

The period initiated by the first faint clear tapping sounds. These sounds gradually become more intense. To ensure that one is not hearing an extraneous sound at least two consecutive tapping sounds should be identified

Phase II

The period during which the sounds have a swishing quality.

Phase III

The period during which the sounds are crisper and more intense.

Phase IV

The period during which the sounds become muffled and a soft blowing quality is heard.

Phase V

The point where the sounds disappear.

References:

1. *Brown MA, Robinson A, Buddle ML. Accuracy of automated blood pressure recorders in pregnancy. Aust NZ J Obstet Gynaecol 1998;38:262-5.*
2. *Gupta M, Shennan AH, Halligan A, Taylor DJ, de Swiet M. Accuracy of oscillometric blood pressure monitoring in pregnancy and pre-eclampsia. Br J Obstet Gynaecol 1997;104:350-55*
3. *Recommendations for Human Blood Pressure Determination by Sphgmomanometer; American Heart Association, 1990*
4. *James DK, Steer PJ, Weiner CD et al Hypertensive Disorders in Pregnancy in High Risk Pregnancies, WB Saunders, London. 1994 pp 270-271*

EDITOR'S COMMENTS

KOROTKOFF SOUNDS

There is controversy whether Korotkoff phase IV (muffling) or phase V (disappearance) should be used in defining diastolic blood pressure. The difference between phase IV and V sound in pregnancy is increased because of the hyperkinetic state of pregnancy.¹ As a result of the latter, phase V may occasionally reach 'O'. Phase IV is subject to more frequent inter-observer error. However the WHO² recommends use of phase IV. When in doubt it is best to include both Phase IV and V readings.

References:

1. *Villar J, Repke J, Markush L et. Al. The measuring of blood pressure during pregnancy. Am J Obstet. Gynecol 1989; 118: 25-28.*
2. *World Health Organisation Study Group. The hypertensive disorders of pregnancy. No. 758 of Technical reports series, Geneva: The Organisation. 1987.*

Section 6.4

Recommended Resuscitation Equipment

The list below is the recommended requirement for resuscitating patient with severe HDP and eclampsia at health center and during transfer.

Resuscitation equipment should be ready at all times for use. To ensure this, regular scheduled inventory check and maintenance should be carried out.

1. RESUSCITATION EQUIPMENT FOR DELIVERY¹

- a) Basic portable Adult Resuscitator Set
- b) Ventilator Resuscitator Set for Child
- c) Sterile child birth kit
 - Surgical scissors - 2
 - Umbilical cord clamps or tape - 4
 - Rubber bulb syringe - 1
 - 4 x 4 inch gauze pads - 12
 - Rubber gloves - 4
 - Towels - 4
 - Dressing towels - 4
 - Baby blanket
 - Sanitary napkins
 - Large plastic bags - 2
 - Disposable apron - 2
 - Surgical masks - 2
 - Mucous Extractor - 1
- d) Portable Incubator
- e) Disposable Vaginal Examination set
- f) Hibitane cream
- g) Normal Saline for Irrigation

2. **RECOMMENDED RESUSCITATION SET**

a) **Resuscitation Equipment**

- Oxygen Resuscitator set
 - Oxygen Regulator pin index with demand valve and continuous oxygen flow meter
 - Suction device oxygen operated
 - Ventimask with tubing
 - Suction tube with finger control
 - Oxygen cylinder key
- Oxygen Resuscitator Bag for adult
 - Adult airway size 2, 3, 4
 - Adult oxygen mask
 - Oxygen Reservoir Bag
- Oxygen Resuscitator for Infant
 - Infant airway 2.0, 0, 1
 - Infant and Neonatal mask
 - Oxygen Reservoir bag
- Laryngoscope with adult blade and spare bulb
- Laryngoscope with infant and neonatal blade
- Endotracheal tube child size 2.0, 2.5, 3.0, 3.5
- Endotracheal tube adult size 7.0, 7.5, 8.0
- Magill Forcep Adult/child
- Mouth Gag child/adult
- Lignocaine Jelly 2 %
- Intubation stylet (small, medium, large)
- Cardiac monitor with defibrillator AC and DC
 - Patient cable
 - Power cable
 - Chest electrode
 - Electrode jelly
- Suction Unit (Battery operated)
- Portable Ventilator (Oxygen operated)

- Pulse oximeter
- Foley's catheter (various sizes)
- Urine bag

b) **Miscellaneous:**

- Nebulizer machine

c) **Equipment for Examination:**

- Stethoscope
- Sphygmomanometer (Mercury)
- Torchlight with battery
- Diagnostic set
- Disposable gloves
- Sterile gloves
- Dextrostix/Glucostix
- Tourniquet

d) **Intravenous solutions**

- I/V drip set
- I/V Cannula size 16G, 18G, 20G, 22G, 24G
- Scalp vein size 19G, 21G, 23G, 25G
- Disposable syringes 2cc, 5cc, 10cc, 20cc, 50cc
- Disposable needles – 21G, 23G, 25G
- 2 bottles Normal Saline
- 2 bottles Dextrose 5%
- 2 bottles Dextrose Saline
- 2 bottles Hartman's Solution
- 2 bottles Haemacel (or other colloid)

e) **Injection**

- Inj. Adrenaline 1 : 1000
- Inj. Atropine 2 mg
- Inj. Sodium Bicarbonate
- Inj. Aminophylline 200 mg
- Inj. Chlorpheniramine 10 mg
- Inj. Frusemide 40 mg
- Inj. Dextrose 30%, 50%
- Inj. Metochlorpromide 10 mg
- Inj. Terbutaline 2.5 mg
- Inj. Salbutamol 5 mg
- Inj. Hydrocortisone 100 mg
- Inj. Dexamethasone 12 mg
- Inj. Syntocinon 10 mg
- Inj. Pethidine 50 mg
- Inj. Diazepam 10 mg
- Inj. Naloxone 0.4 mg
- Inj. MgSO₄ (see below)
- Inj. Hydrallazine 25 mg
- Inj. Calcium Gluconate 1 gm

3. **ECLAMPSIA KIT**

i) **Magnesium Sulphate Pack**

a) Intravenous infusion:

- Normal saline – 500 ml
- Intravenous cannula – size 16G, 18G
- Infusion 'giving' set
- Tape to secure cannula
- Swab to clean skin
- Spirit for swabbing the skin
- Tourniquet
- Magnesium Sulphate ²
- Loading dose – 4 grams
- Maintenance dose – 5x5 grams
- For recurrent convulsions – 5 grams

- b) Syringes – 5cc, 10cc, 20cc
- c) Needles – 21G, 23G, 25G
- d) Calcium Gluconate – 1 gram (for toxicity)
- e) Observation Chart
 - Fluid Balance
 - BP/PR/respiratory rate, knee jerk reflexes
- f) Protocol
 - Summary Flow Chart
 - Detailed Regime
 - Guidelines for other aspects of care

ii) **Diazepam Pack**

- a) Intravenous infusion ;
 - Dextrose 5% - 500 ml
 - Intravenous cannula – size 16G, 18G
 - Infusion 'giving' set
 - Tape to secure cannula
 - Swab to clean skin
 - Spirit for swabbing the skin
- b) Diazepam
 - Loading dose – 10 mg
 - Maintenance dose – 10-30 mg (infusion)
 - For recurrent convulsions – 10 mg
- c) Syringes – 5cc, 10cc, 20cc
- d) Needles – 21G, 23G, 25G
- e) Observation Chart
- f) Protocol
 - Summary Flow Chart
 - Detailed Regime
 - Guidelines for other aspects of care

iii) Adult airway size – 2,3,4

iv) Portable Sucker

References:

1. *'Garis panduan Peralatan Dalam Ambulans', Ministry of Health, 1994.*
2. *Collaborative Eclampsia Trial, Lancet 1995.*
3. *Davey D.A. 2007, Hypertensive Disorders In Pregnancy. Dewhurst's Textbook of Obstetrics and Gynaecology For Postgraduates. Seventh edition edited by Charles R. Whitfield, Blackwell Science Ltd, London.*

Section 6.5

Patient Information, Communication and Education (ICE)

What is Hypertension in pregnancy?

Hypertension in pregnancy is a condition where the blood pressure (B/P) of the pregnant woman is raised above normal limits. In addition to this, protein may be present in the urine (dirty urine), excessive weight gain and swelling of the legs (oedema).

Hypertension in pregnancy occurs in about 5% to 10% of pregnant women. This condition can be detected if you attend regular antenatal check up.

Is Hypertension in pregnancy a dangerous condition?

It is dangerous if left unchecked and untreated. However, when detected early, the condition can be controlled and treatment can be given to prevent complications.

Who are at risk of getting hypertension in pregnancy?

Certain groups of women are at risk of developing hypertension in pregnancy. By attending early antenatal check up you can assist health care providers to identify risk factors and keep you under surveillance. If you have any risk factors, you have a greater chance of developing hypertension in pregnancy.

The following are common risk factors for developing high blood pressure in pregnancy:

- Mothers age < 20 years and > 35 years.
- First pregnancy
- Previous history of hypertension during pregnancy
- Twin pregnancy
- Hypertension
- Diabetes Mellitus
- Excessive weight gain

What complications can occur?

Complications can occur to both mother and baby. The worst complications that can occur are death to the mother and the baby. Other complications are as follows: -

Mother

- Fits - this condition is called **eclampsia**.
- Risk of developing a stroke
- Severe uncontrolled hypertension and heart failure.
- Collection of excessive fluid in the lungs (pulmonary oedema).
- Functions of the kidneys and liver can be affected resulting in loss of protein in the urine.
- Early separation of the placenta from the wall of the womb.
- Failure of blood to clot due to reduction of blood clotting factors in the blood.

Baby

- Failure of baby to grow satisfactorily (Growth retardation in the womb)
- Tendency for baby to die in the womb i.e. being stillborn.

Are there any warning signs?

Yes, there may be several warning signs, some of which are as listed below:

- Sudden increase in blood pressure.
- Sudden increase in weight (> 1 kg per week)
- Generalised swelling (oedema), swelling of hands, feet and / or puffy eyes, tight rings, tight shoes.
- Protein in the urine
- Epigastric pain (Pain in the upper part of the abdomen)
- Frequent/or severe headache
- Visual disturbance such as blurred vision
- Nausea and vomiting
- Passing less urine than normal

What should be done if the warning signs occur?

SEE YOUR DOCTOR IMMEDIATELY or GO TO THE NEAREST CLINIC.

How is hypertension in pregnancy managed or treated?

The progression of hypertension is very unpredictable. Complications can develop without prior warning. Hence you should have frequent antenatal check ups. You may require admission to hospital for close observation. If you are prescribed medication it is very important that you take them as instructed.

If the disease becomes severe, early delivery may be required. Because of the seriousness of the disease HOSPITAL DELIVERY IS REQUIRED.

Can Hypertension in Pregnancy be treated at home?

Hypertension in pregnancy requires close monitoring by the medical staff as it can become worse. Hospital admission is usually advised.

Home management or outpatient treatment depends very much on your condition and your doctor is the best person to advise you on this. Home management may be acceptable in some instances, provided that you are able to come for continuous and close monitoring. From time to time you may still need hospital admission.

What steps should be taken to ensure early detection and prevent complications?

- Attend antenatal clinic as soon as you know you are pregnant.
- Keep scheduled appointments at the antenatal clinic. If you are one of those in the high-risk group it is advisable that you attend clinic early.
- Report to the clinic immediately or go to the hospital when you have any of the warning signs. **Do not wait for scheduled appointments.**
- Always carry your home-based maternal health record (Antenatal card) for all clinic visits so that records can be made into the card and your condition monitored.
- Assess your baby's activity daily. Maintain the Fetal Kick Chart as advised by your nurse or doctor.
- Make sure you have adequate rest.
- Ensure adequate sleep (6 to 8 hours)

Why rest is necessary and what else should I do?

Rest is important because when you have adequate rest, your blood pressure will settle and your baby will continue to grow better.

To help you rest, the following are some advice that may be useful: -

- To reduce boredom, you can keep yourself occupied with diversional activities like reading, handicrafts etc.
- Do gentle exercises such as circling of hands and feet or gently tensing and relaxing arms and leg muscles. This improves muscle tone, circulation and sense of wellbeing.
- In bed lie on the left side (alternate to the right side as needed).
- Use relaxation techniques to help cope with stress. Relax the body, one muscle at a time or imagine some pleasant scene or image. Soothing music can help in relaxation.
- Encourage family members to assist in house- work.
- Increase fluid intake to 8 glasses a day and add roughage to diet e.g. fruits and vegetables.

Will the medication that I take affect my baby?

The medication prescribed by the doctors is safe for you and your baby.

Do I need a Caesarian Section for delivery?

The method of delivery will depend on the severity of the disease and the stage (gestation) of your pregnancy. Your doctor will counsel you on the methods of delivery. Many women can have a normal vaginal delivery. Caesarean section may be required in a few.

Can I breast-feed my baby?

Yes, breastfeeding is best for you and your baby. Infact you should breastfeed exclusively for a period of 4 months. If your baby is premature he/she may require a special nutrition programme. You are encouraged to express your breast milk to feed your baby as part of the nutrition plan.

Will my blood pressure subside after my delivery?

This varies from individual to individual. The blood pressure settles rapidly after delivery. In some it may take several days to weeks. You will need close observation during the post-natal period. You will require monitoring and examination of your blood pressure till it returns to normal. Hypertension of pregnancy normal clears within six weeks of delivery.

Will the hypertension recur during my next pregnancy?

There is a small risk of recurrence of hypertension in your next pregnancy. Space out your pregnancy by using a suitable contraceptive method. Attend an antenatal clinic as soon as you know that you are pregnant again.

Make sure you inform the doctor/nurse regarding the history of your previous pregnancy. It would be useful if you bring along the antenatal card of your previous pregnancy.

Section 6.6

Suggested Training Programme

SESSIONS	TEACHING LEARNING METHOD	TIME FRAME
Introduction to the modules	Briefing	10 minutes
Technique of Blood Pressure Measurement	Lecture & Demostration	20 minutes
Understanding Hypertensive Disorders in Pregnancy	Lecture	30 minutes
Management of Hypertensive Disorders - Mild	Lecture	30 minutes
Management of Hypertensive Disorders in Pregnancy - Severe - Eclampsia - HELLP	Lecture	30 minutes
Anaesthesia in the Management of Hypertensive Disorders in Pregnancy	Lecture	30 minutes
Fetal Surveillance	Lecture	20 minutes
Referral procedures and documentation	Lecture and demonstration	30 minutes
TOTAL TIME		400 minutes

Quality Assurance in Management of Hypertensive Disorders of Pregnancy

The following factors have been identified as quality related issues in management of hypertension in pregnancy in Malaysia. These factors, causes and remediable steps have been derived following several quality assurance workshops held in the Ministry of Health. Trainers are encouraged to use these risk factors in identifying, evaluating and monitoring of hypertension management in pregnancy.

Risk Factors	Causes	Avoidable?	Steps To Avoid Occurrence
<p>Health Service Risk Factors</p> <p>1. Failure to monitor blood pressure and urine during prenatal care</p>	<p>1. Lack of proper guidelines on B/P monitoring related to severe pre-eclampsia.</p> <p>2. Lack of well defined job description in the ward</p> <p>3. Lack of supervision of junior staff by senior staff</p> <p>4. Attitude – non compliance to set criteria (slip shod work)</p>	<p>Yes</p>	<p>1. Check B/P and urine at every prenatal visit</p> <p>2. Guidelines for nurses to increase frequency of B/P taking – according to patients condition</p> <p>3. Assignment of specific nurse when patient needs close monitoring</p> <p>4. Educate staff/ retrain staff every year</p> <p>5. Closer supervision by senior staff</p> <p>6. Closer monitoring of problem staff</p>

Risk Factors	Causes	Avoidable?	Steps To Avoid Occurrence
<p>2. Failure to recognise signs and symptoms of PE & eclampsia</p>	<p>1. Lack of adequate knowledge on disease process</p>	<p>Yes</p>	<ol style="list-style-type: none"> 1. Criteria for diagnosis <ul style="list-style-type: none"> • B/P level 140/90 taken on 2 occasions • If B/P is known: <ul style="list-style-type: none"> - ↑ in diastolic 20 mm/Hg - ↑ in systolic 30 mm/Hg • Diastolic B/P of 110 mm/Hg 2. Monitor BP closely if weight gain >1 kg/week 3. Details that has to be noted when taking history <ul style="list-style-type: none"> ▪ Family history of hypertension • History of gestational hypertension • Primigravida • Low socio- economic group • Associated conditions; <ul style="list-style-type: none"> - Twins - Diabetes mellitus - Renal disease - SLE - Rh iso-immunization 4. Presence of albumin in urine 5. Complaints from patient such as: <ul style="list-style-type: none"> • Epigastric pain • Seeing 'stars' • Diarrhoea • Blurring of vision • Frontal headache

Risk Factors	Causes	Avoidable?	Steps To Avoid Occurrence
<p>3. Delay in referral of eclamptic women</p>	<ol style="list-style-type: none"> 1. Family resistance for admission <ul style="list-style-type: none"> • Illegal immigrant (fear of law) • To seek traditional treatment 2. Influence of traditional healers 3. Influence of other persons 4. Distance – interior (remote) 5. Transportation problems 6. No adult male available or out at work during eclampsia 	<p>Yes</p>	<ol style="list-style-type: none"> 1. Patient should be transferred to a hospital not more than ½ hr. after a fit 2. Recognize those who are likely to give resistance eg. TBA, husband, father mother and counsel them 3. Explore their reasons for resistance – give assurance 4. Call for other medical staff help 5. Each district to get to know usual means of transport 6. To develop strategies to transfer ill patient in cases of emergency 7. Recognise those who are likely to give resistance eg. TBA, husband, father mother and counsel them 8. Explore their reasons for resistance – give assurance 9. Call for other medical staff help

Risk Factors	Causes	Avoidable?	Steps To Avoid Occurrence
			10. Each district to get to know usual means of transport 11. To develop strategies to transfer ill patient in cases of emergency

Risk Factors	Causes	Avoidable?	Steps To Avoid Occurrence
<p>4. Lack of clear-cut treatment strategies for dealing with PE & eclampsia</p>		<p>Yes</p>	<ol style="list-style-type: none"> 1. At the clinic <ul style="list-style-type: none"> • If B/P high (140/90) • Recheck after rest • Still high → refer/admit 2. Presence of albumin <ul style="list-style-type: none"> • Urine FEME • Urine protein 3. Excessive weight gain <p>→ look for oedema of – abdomen, sacrum, face, hands</p> 4. Treatment: <p>Antihypertensive drugs only if diastolic pressure >90 mm/Hg</p> <p>Begin with oral medication</p> <ul style="list-style-type: none"> • Methyldopa • Trandate • Aldalat <p>I/v Hydralazine ≥ or sublingual Nifedine if DBP ≥110 mm/Hg</p> 5. Investigations <ul style="list-style-type: none"> • Hb • Platelet count • Renal profile – uric acid, urea, creatinine • LFT • BT, CT • Serial ultrasound

Risk Factors	Causes	Avoidable?	Steps To Avoid Occurrence
			<p>6. Timing of delivery</p> <ul style="list-style-type: none"> • Hypertension, no treatment → 40/52 • Hypertension with treatment → 38/52 • Symptoms with mild proteinuria → 38/52 with gross proteinuria → 34/52 • Severe with renal failure & eclampsia → deliver the baby whatever the gestational age <p>7. Labour</p> <ul style="list-style-type: none"> • 2nd stage – should not be prolonged, assist if necessary • 3rd stage – avoid Syntometrine & Ergometrine, uses syntocinon <p>8. Puerperium</p> <ul style="list-style-type: none"> • 4 hrly B/P & pulse • urine protein daily • stop oral anti-hypertensive treatment if B/P ≤ 90 mm/Hg. If B/P ≥ 110 mm/Hg – i/v hydralazine in incremental doses. <p>9. Postnatal follow up</p> <ul style="list-style-type: none"> - 6/52 if not on treatment - 1/52 if on treatment - if at 6/52 → refer medical if B/P still high, refer to physician

Risk Factors	Causes	Avoidable?	Steps To Avoid Occurrence
5. Lack of proper equipment and drugs to treat eclampsia		Yes	1. Ministry to ensure availability of equipment and drugs.

PATIENT AND COMMUNITY FACTORS IDENTIFIED

Risk Factors	Causes	Avoidable?	Steps To Avoid Occurrence
<p>COMMUNITY RISK FACTORS</p> <p>1. Lack of awareness about symptoms of pre-eclampsia and importance of antenatal care</p>	<p>1. Inadequate transfer of knowledge by health staff</p> <p>2. Traditional belief</p>	Yes	<p>1. Information to be included in educating patient:</p> <ul style="list-style-type: none"> • Pathophysiology of disease • Symptoms • Dangers • Signs of impending eclampsia • The management <p>2. Allow harmless traditional practice</p>
2. Low Socio-economic status (this is because teenage pregnancy is more common)		Yes	<p>1. Educate on birth spacing</p> <p>2. Food supplements</p> <p>3. Refer social welfare</p>

Risk Factors	Causes	Avoidable?	Steps To Avoid Occurrence
among the poor)			<ol style="list-style-type: none"> 4. Introduce reproductive health issues to school children
3. Transportation problems	<ol style="list-style-type: none"> 1. Inaccessible areas 2. Poverty 	Yes	<ol style="list-style-type: none"> 1. Admit mothers with B/P 140/90 and above early. 2. Home visits by Health staff. Self monitoring of B/P and urine for mothers who can afford it. Home supervision or self monitoring 3. Arrange for alternative modes of transport should an emergency arise eg. neighbour's car, police etc. 4. Half-way home in the hospital or close-by mother and children 5. Counselling on importance of antenatal care 6. Education 7. Teleconsultation and Hot line service for urgent advice

Risk Factors	Causes	Avoidable?	Steps To Avoid Occurrence
<p>4. Community distrust of Health Care Personnel</p>	<p>1. Un-approachable staff → fear and anxiety</p>	<p>Yes</p>	<ol style="list-style-type: none"> 1. Establish rapport between health care providers and community to build trust 2. Involve in community project 3. Counsel mothers and families about dangerous symptoms of PE and importance of antenatal care 4. Organise open clinic day once a year eg. Exhibition 5. Survey (questionnaire) 6. Phenomenological survey 7. Provide detailed information of risk to mother 8. Attitude problem → retrained staff 9. Provide Suggestion Box

