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Diagnosis, Evaluation, and Management of the Hypertensive Disorders of Pregnancy: Executive Summary

The guideline summarized here has been prepared by the Canadian Hypertensive Disorders of Pregnancy Working Group, reviewed and approved by the Hypertension Guideline Committee, reviewed by the Maternal Fetal Medicine and Family Physician Advisory committees, and approved by the Executive and Council of the Society of Obstetricians and Gynaecologists of Canada.

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Abstract

Objective: This executive summary presents in brief the current evidence assessed in the clinical practice guideline prepared by the Canadian Hypertensive Disorders of Pregnancy Working Group and published by *Pregnancy Hypertension* (http://www.pregnancyhypertension.org/article/S2210-7789(14)00004-X/fulltext) to provide a reasonable approach to the diagnosis, evaluation, and treatment of the hypertensive disorders of pregnancy.

Evidence: Published literature was retrieved through searches of Medline, CINAHL, and The Cochrane Library in March 2012 using appropriate controlled vocabulary (e.g., pregnancy, hypertension, pre-eclampsia, pregnancy toxemias) and key words (e.g., diagnosis, evaluation, classification, prediction, prevention, prognosis, treatment, postpartum follow-up). Results were restricted to systematic reviews, randomized control trials, controlled clinical trials, and observational studies published in French or English between January 2006 and February 2012. Searches were updated on a regular basis and incorporated in the guideline to September 2013. Grey (unpublished) literature was identified through searching the websites of health technology assessment and health technology-related agencies, clinical practice guideline collections, clinical trial registries, and national and international medical specialty societies.

Values: The quality of evidence in the guideline summarized here was rated using the criteria described in the Report of the Canadian Task Force on Preventative Health Care (Table 1).

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Table 1. Key to evidence statements and grading of recommendations, using the ranking of the Canadian Task Force on Preventive Health Care

Quality of evidence assessment*	Classification of recommendations†
I: Evidence obtained from at least one properly randomized controlled trial	A. There is good evidence to recommend the clinical preventive action
II-1: Evidence from well-designed controlled trials without randomization	B. There is fair evidence to recommend the clinical preventive action
II-2: Evidence from well-designed cohort (prospective or retrospective) or case–control studies, preferably from more than one centre or research group	C. The existing evidence is conflicting and does not allow to make a recommendation for or against use of the clinical preventive action; however, other factors may influence decision-making
II-3: Evidence obtained from comparisons between times or places with or without the intervention. Dramatic results in uncontrolled experiments (such as the results of treatment with penicillin in the 1940s) could also be included in this category	D. There is fair evidence to recommend against the clinical preventive action
	E. There is good evidence to recommend against the clinical preventive action
III: Opinions of respected authorities, based on clinical experience, descriptive studies, or reports of expert committees	L. There is insufficient evidence (in quantity or quality) to make a recommendation; however, other factors may influence decision-making

*The quality of evidence reported in these guidelines has been adapted from The Evaluation of Evidence criteria described in the Canadian Task Force on Preventive Health Care.[39]

†Recommendations included in these guidelines have been adapted from the Classification of Recommendations criteria described in the Canadian Task Force on Preventive Health Care.[39]

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ABBREVIATIONS

BP	blood pressure
HDP	hypertensive disorder of pregnancy
HELLP	hemolysis, elevated liver enzymes, low platelets
IUGR	intrauterine growth restriction
NICU	neonatal intensive care unit
RCT	randomized control trial
RDS	respiratory distress syndrome

INTRODUCTION

Hypertensive disorders of pregnancy remain leading causes of maternal and perinatal morbidity and mortality.[1,2] The guideline summarized here assesses the quality of the relevant existing evidence and provides a reasonable approach to the diagnosis, evaluation, and treatment of the HDP, focusing on Canadian context.

Our purpose is to support evidence-based maternity care of women who are planning pregnancy and are at risk of an HDP, have an HDP in the current pregnancy, or are postpartum and had an HDP. When necessary, we have provided expert opinion about reasonable clinical care. Our health intent and aim is to improve short- and long-term maternal, perinatal, and paediatric outcomes and the cost-effectiveness of related interventions in pregnancies complicated by an HDP. The expected benefit of this guideline is improved outcomes for mother, baby, and child through evidence-advised practice. Our target users are multidisciplinary maternity care providers from primary to tertiary levels of health care.

The questions that this guideline seeks to address are:

- How, and in what setting, should BP be measured in pregnancy, and what is an abnormal BP?
- How should proteinuria be measured in pregnancy? What constitutes significant proteinuria? Is heavy proteinuria an indication for delivery?
- How should the HDPs be diagnosed and classified? What constitutes severe preeclampsia?
- What are the prognoses of pregnancies complicated by pre-existing hypertension, gestational hypertension, or preeclampsia?
- How can preeclampsia and its complications be predicted and/or prevented by lifestyle changes, medication, and/or care of a specific type or in a specific location?
- How should women with an HDP be managed regarding initial investigations, dietary and lifestyle changes, place of care, antihypertensive therapy, aspects of care specific to women with preeclampsia (such as magnesium sulphate), mode and timing of delivery, intrapartum care (including BP monitoring and analgesia/anaesthesia), and postpartum monitoring, treatment, and counselling regarding the impact of an HDP on both future pregnancy outcomes and long-term maternal and paediatric outcomes?
- What is the patient's perspective on her diagnosis and evaluation?
- How can this guideline be implemented into clinical practice?

This document presents a summary of the recommendations, along with supporting text for the new classification of the HDPs, and all of the tables provided in the full guideline. Because of the breadth of the topic and the volume of material covered, the methods, supporting text for all recommendations, and the full list of references, including those for the tables, have been published separately as an open-access article in *Pregnancy Hypertension*.[3]

Important changes affect all aspects of care covered in the 2008 guidelines. Notable examples include the addition of systolic BP in the definition of pregnancy hypertension, revised HDP classification, new information on prevention, more direction with respect to timing of delivery in women with any HDP, information about magnesium sulphate for fetal neuroprotection at < 32 weeks, a new gestational age cut-off (≤ 34+6 instead of ≤ 33+6 weeks) for administration of steroids, and a section on knowledge translation with links to useful tools for women and practitioners.

CHAPTER 1: DIAGNOSIS OF HDP AND CLASSIFICATION OF BP MEASUREMENTS

BP Measurement

Recommendations

1. Blood pressure should be measured with the woman in the sitting position with the arm at the level of the heart. (II-2A)
2. An appropriately sized cuff (i.e., length 1.5 times the circumference of the arm) should be used. (II-2A)
3. Korotkoff phase V should be used to designate diastolic blood pressure. (I-A)
4. If blood pressure is consistently higher in one arm, the arm with the higher values should be used for all blood pressure measurements. (III-B)
5. Blood pressure can be measured using a mercury sphygmomanometer, a calibrated aneroid device, or an automated blood pressure machine that has been validated for use in preeclampsia. (II-2A)
6. Automated blood pressure machines that have not been validated for use in women with preeclampsia may underestimate or overestimate blood pressure in those women; a comparison of readings using mercury sphygmomanometry or a calibrated aneroid device is recommended. (II-2A)

7. In the office setting, when blood pressure elevation is non-severe and preeclampsia is not suspected, ambulatory blood pressure monitoring or home blood pressure monitoring is useful to confirm persistently elevated blood pressure. (II-2C)
8. When home blood pressure monitoring is used, maternity care providers should ensure that patients have adequate training in measuring their blood pressure and interpreting the readings. (III-C)
9. The accuracy of all blood pressure measurement devices used in hospitals or offices should be checked regularly against a calibrated device. (II-3C)
10. The accuracy of all automated devices used for home blood pressure monitoring should be checked regularly against a calibrated device. (III-C)

Diagnosis of Hypertension

Recommendations

11. The diagnosis of hypertension should be based on office or in-hospital blood pressure measurements. (II-B)
12. Hypertension in pregnancy should be defined as an office (or in-hospital) systolic blood pressure ≥ 140 mmHg and/or diastolic blood pressure ≥ 90 mmHg, based on the average of *at least* 2 measurements, taken at least 15 minutes apart, using the same arm. (II-2B)
13. Resistant hypertension should be defined as the need for 3 antihypertensive medications for blood pressure control at ≥ 20 weeks' gestation. (III-C)
14. A transient hypertensive effect should be defined as an office systolic blood pressure ≥ 140 mmHg or a diastolic blood pressure ≥ 90 mmHg that is not confirmed after rest, on repeat measurement, on the same or on subsequent visits. (II-2B)
15. A white-coat hypertensive effect refers to blood pressure that is elevated in the office (i.e., systolic ≥ 140 mmHg or diastolic ≥ 90 mmHg), but < 135 mmHg (systolic) and < 85 mmHg (diastolic) on ambulatory or home blood pressure monitoring. (II-2B)
16. A masked hypertensive effect refers to blood pressure that is normal in the office (i.e., systolic < 140 mmHg and diastolic < 90 mmHg) but elevated on ambulatory or home blood pressure monitoring (i.e., systolic ≥ 135 mmHg or diastolic ≥ 85 mmHg). (II-2B)
17. Severe hypertension should be defined, in any setting, as a systolic blood pressure of ≥ 160 mmHg or a diastolic blood pressure of ≥ 110 mmHg based on the average of *at least* 2 measurements, taken at least 15 minutes apart, using the same arm. (II-2B)

Measurement of Proteinuria

Recommendations

18. All pregnant women should be assessed for proteinuria. (II-2B)
19. Urinary dipstick testing (by visual or automated testing) may be used for screening for proteinuria when the suspicion of preeclampsia is low. (II-2B)
20. Significant proteinuria should be defined as ≥ 0.3 g/d in a complete 24-hour urine collection or ≥ 30 mg/mmol urinary creatinine in a spot (random) urine sample. (II-2B)
21. Significant proteinuria should be suspected when urinary dipstick proteinuria is ≥ 1+. (II-2A)
22. More definitive testing for proteinuria (by urinary protein:creatinine ratio or 24-hour urine collection) is encouraged when there is a suspicion of preeclampsia, including: ≥ 1+ dipstick proteinuria in women with hypertension and risingblood pressure and in women withnormal blood pressure, but symptoms or signs suggestive of preeclampsia. (II-2A)
23. Proteinuria testing does not need to be repeated once significant proteinuria of preeclampsia has been confirmed. (II-2A)
24. There is insufficient information to make a recommendation about the accuracy of the urinary albumin:creatinine ratio. (II-2L)

Classification of HDPs

Recommendations

25. Hypertensive disorders of pregnancy should be classified as pre-existing hypertension, gestational hypertension, preeclampsia, or "other hypertensive effects" on the basis of different diagnostic and therapeutic considerations. (II-2B) (Table 2)
26. The presence or absence of preeclampsia must be ascertained, given its clear association with more adverse maternal and perinatal outcomes. (II-2B)
27. In women with pre-existing hypertension, preeclampsia should be defined as resistant hypertension, new *or* worsening proteinuria, one or more adverse conditions, or one or more severe complications. (II-2B)
28. In women with gestational hypertension, preeclampsia should be defined as new-onset proteinuria, one or more adverse conditions, or one or more severe complications. (II-2B)
29. Severe preeclampsia should be defined as preeclampsia complicated by one or more severe complications. (II-2B)

Table 2. Classification of the hypertensive disorders of pregnancy

Disorder	Comments
Pre-existing (chronic) hypertension	This is defined as hypertension that develops either pre-pregnancy or at < 20+0 weeks' gestation
With comorbid condition(s)	Comorbid conditions (e.g., pre-gestational type I or II diabetes mellitus or kidney disease) warrant tighter BP control outside of pregnancy because of their association with heightened cardiovascular risk.
With evidence of preeclampsia	This is also known as superimposed preeclampsia, and is defined by the development of one or more of the following at ≥ 20 weeks: • resistant hypertension, *or* • new or worsening proteinuria, *or* • one or more adverse conditions,* *or* • one or more severe complications.* Severe preeclampsia is defined as preeclampsia with one or more severe complications.
Gestational hypertension	This is defined as hypertension that develops for the first time at ≥ 20+0 weeks' gestation.
With comorbid condition(s)	Comorbid conditions (e.g., pre-gestational type I or II diabetes mellitus or kidney disease) warrant tighter BP control outside of pregnancy because of their association with heightened cardiovascular risk.
With evidence of preeclampsia	Evidence of preeclampsia may appear only many weeks after the onset of gestational hypertension. Preeclampsia is defined as gestational hypertension with one or more of the following: • new proteinuria, *or* • one or more adverse conditions,* *or* • one or more severe complications.* Severe preeclampsia is defined as preeclampsia with one or more severe complications.
Preeclampsia	Preeclampsia may arise de novo. It is defined as gestational hypertension with one or more of the following: • new proteinuria, *or* • one or more adverse conditions,* *or* • one or more severe complications.* Severe preeclampsia is defined as preeclampsia with one or more severe complications.
Other hypertensive effects†	
Transient hypertensive effect	Elevated BP may be due to environmental stimuli, e.g., the pain of labour.
White-coat hypertensive effect	This is defined as BP that is elevated in the office (sBP ≥ 140 mmHg or dBP ≥ 90 mmHg), but consistently normal outside of the office (< 135/85 mmHg) by ABPM or HBPM
Masked hypertensive effect	This is defined as BP that is consistently normal in the office (sBP < 140 mmHg or dBP < 90 mmHg), but elevated outside of the office (≥ 135/85 mmHg) by ABPM or repeated HBPM.

sBP: systolic BP; dBP diastolic BP; ABPM: ambulatory BP monitoring; HBPM: home BP monitoring

*Adverse conditions and severe complications of preeclampsia are defined in Table 3.

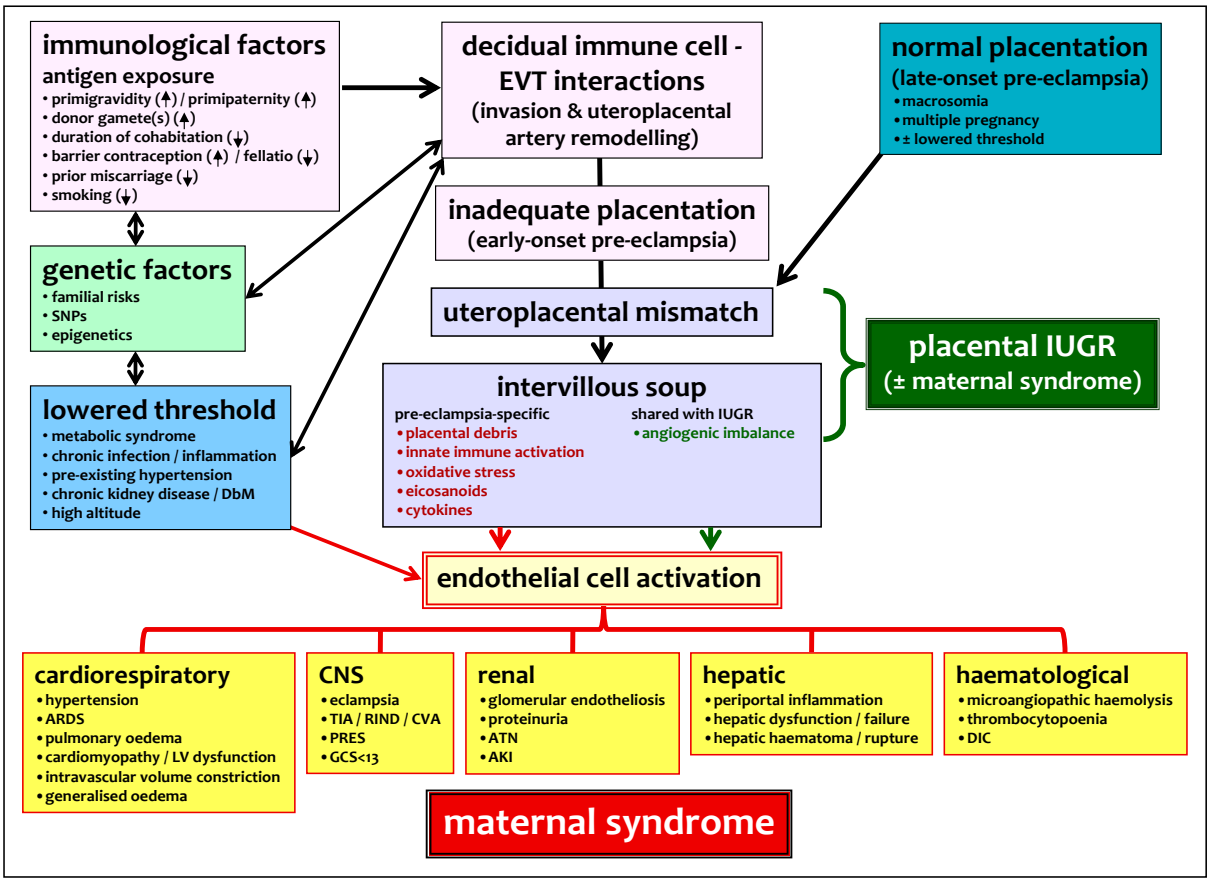
†These may occur in women whose BP is elevated at < 20+0 or ≥ 20+0 weeks who are suspected of having pre-existing or gestational hypertension/preeclampsia, respectively.

30. Severe preeclampsia, as defined in this guideline, warrants delivery. (II-2B)
31. The term PIH (pregnancy-induced hypertension) should be abandoned, as its meaning in clinical practice is unclear. (III-D)

Definition of Preeclampsia

Preeclampsia is most commonly defined by new-onset proteinuria and, potentially, other end-organ dysfunction. Hypertension and proteinuria are discussed above under "Diagnosis of Hypertension"" and "Management of Proteinuria." Women with preeclampsia may have a diminished, or no, nocturnal BP decrease.[4] Maternal end-organ dysfunction and fetal manifestations of preeclampsia illustrated in the Figure are all non-specific. In this model of its origins we describe preeclampsia that arises primarily through imperfect placentation (early-onset or "placental" preeclampsia [pink]) or through either a lowered maternal threshold or excessive physiological placentation (late-onset or "maternal" preeclampsia [blue]). Some aspects of the preeclampsia process are specific to it, while others are shared with normotensive IUGR. A lowered maternal threshold may also influence the development of early-onset preeclampsia through

The origins and consequences of preeclampsia



EVT: extravillous trophoblast; SNP: single nucleotide polymorphism; ARDS: acute respiratory distress syndrome; CNS: central nervous system; TIA: transient ischemic attack; RIND: reversible ischemic neurological deficit; CVA: cerebrovascular accident; PRES: posterior reversible encephalopathy syndrome; GCS: Glasgow Coma Scale; ATN: acute tubular necrosis; AKI: acute kidney injury; DIC: disseminated intravascular coagulation; DbM: diabetes mellitus; LV: left ventricle

direct endothelial cell activation. The consequences of endothelial cell activation that appear consistent between all women with preeclampsia include a variable impact on multiple vulnerable organ systems. Disease severity generally correlates with the degree and number of organ dysfunctions. Fetal manifestations may occur before, with, or in the absence of maternal manifestations.[5]

Table 3 outlines the end-organ dysfunctions of preeclampsia: adverse conditions and severe complications. Adverse conditions consist of maternal symptoms, signs, and abnormal laboratory results, and abnormal fetal monitoring results that may herald the development of severe maternal or fetal complications (including stillbirth). The adverse conditions are those that we wait for and respond to (e.g., low oxygen saturation) in an effort to avoid entirely the severe complications (e.g., pulmonary edema). That response could be more intensive maternal or fetal monitoring, specific treatment, or delivery. Severe maternal complications of preeclampsia warrant delivery.

The adverse conditions are manifestations of preeclampsia that increase the risk of adverse maternal or perinatal outcomes.[6] Table 3 lists the adverse conditions by maternal organ system. Of particular importance are preterm preeclampsia, chest pain or dyspnea, and abnormality of one or more of oxygen saturation by pulse oximetry, platelet count, serum creatinine, or aspartate transaminase.[6] Proteinuria predicts neither short-term adverse outcomes nor long-term maternal renal prognosis.[7,8] HELLP syndrome is represented by its component parts (hemolysis, elevated liver enzymes, and low platelets), to which we react to by initiating delivery.

How maternal adverse conditions may predict fetal or neonatal outcomes in preeclampsia is unclear. The perinatal literature suggests that abnormal fetal monitoring of various types may identify increased fetal risk. The biophysical profile has unproven utility in high risk women,[9,10] and may falsely reassure with early-onset IUGR[11] or preeclampsia.[12]

Currently, there is no single fetal monitoring test to accurately predict fetal compromise in women with

Table 3. Adverse conditions and severe complications of preeclampsia

Organ system affected	Adverse conditions (that increase the risk of severe complications)	Severe complications (that warrant delivery)
Central nervous system	Headache/visual symptoms	Eclampsia
		PRES
		Cortical blindness or retinal detachment
		Glasgow coma scale < 13
		Stroke, TIA, or RIND
Cardiorespiratory	Chest pain/dyspnea	Uncontrolled severe hypertension (over a period of 12 h despite use of three antihypertensive agents)
	Oxygen saturation < 97%	Oxygen saturation < 90%, need for ≥ 50% oxygen for > 1 h, intubation (other than for Caesarean section), pulmonary edema
		Positive inotropic support
		Myocardial ischemia or infarction
Haematological	Elevated WBC count	Platelet count < 50 × 10^9/L
	Elevated INR or aPTT	Transfusion of any blood product
	Low platelet count	
Renal	Elevated serum creatinine	Acute kidney injury (creatinine > 150 μM with no prior renal disease)
	Elevated serum uric acid	
		New indication for dialysis
Hepatic	Nausea or vomiting	Hepatic dysfunction (INR > 2 in absence of DIC or warfarin)
	RUQ or epigastric pain	
	Elevated serum AST, ALT, LDH, or bilirubin	Hepatic haematoma or rupture
	Low plasma albumin	
Feto-placental	Abnormal FHR	Abruption with evidence of maternal or fetal compromise
	IUGR	Reverse ductus venosus A wave
	Oligohydramnios	Stillbirth
	Absent or reversed end-diastolic flow by Doppler velocimetry	

PRES: posterior reversible leukoencephalopathy syndrome; TIA: transient ischemic attack; RIND: reversible ischemic neurological deficit (< 48 hr); WBC: white blood cell; INR: international normalized ratio; aPTT: activated partial thromboplastin time; RUQ: right upper quadrant; AST: aspartate aminotransferase; ALT: alanine aminotransferase; LDH: lactate dehydrogenase; DIC: disseminated intravascular coagulation; FHR: fetal heart rate.

preeclampsia. Most experts suggest a combination of tests, with emphasis on umbilical artery Doppler when there is IUGR.[9]

Other non-specific risk factors for severe complications of preeclampsia are immigrant status, young maternal age, nulliparity, lower maternal weight, and in the index pregnancy, multiple pregnancy and early-onset preeclampsia.[13]

Definitions of severe preeclampsia vary, but most include multi-organ involvement[14–16] We modified our definition of severe preeclampsia to preeclampsia associated with one or more severe complications. Severe preeclampsia now warrants delivery regardless of gestational age. Our definition excludes heavy proteinuria and HELLP syndrome, which are not absolute indications for delivery, and includes stroke[17] and pulmonary edema, which are leading causes of maternal death in preeclampsia.[2]

Other

A transient hypertensive effect is not associated with an increased risk of adverse outcomes. White-coat effect in early pregnancy (~30%) is common.[18] Forty percent of women progress to persistent hypertension at ≥ 20 weeks (i.e., gestational hypertension) and 8% to preeclampsia. Women with white-coat effect have risks (e.g., severe hypertension, preterm delivery, and NICU admission) intermediate between normotension and either pre-existing or gestational hypertension.[19–24]

Masked hypertension in early pregnancy (~30%) is also common,[18] but associated perinatal risks are unknown. Outcomes with masked hypertension at ≥ 20 weeks (~10%) equate to those of gestational hypertension.[25,26] Masked hypertension could be considered (and ambulatory or home BP monitoring performed) if there are unexplained maternal or perinatal complications typically associated with the HDPs.

Table 4. Investigations to diagnosis or monitor women with a hypertensive disorder of pregnancy

Investigations for diagnosis	Description in women with preeclampsia	Description in women with other conditions
MATERNAL TESTING		
Urine testing		
Urinalysis (routine and microscopy with/ without additional tests for proteinuria)	Proteinuria without RBCs or casts	Hemoglobinuria (dipstick "hematuria" without RBCs): hemolytic anemia RBCs alone: renal stones, renal cortical necrosis (also associated with back pain and oliguria/anuria) RBCs and/or casts are associated with other glomerular disease, with scleroderma renal crisis. and with about half of TTP-HUS Bacteria: UTI or asymptomatic bacteruria Proteinuria is usually absent in secondary causes of hypertension such as pheochromocytoma, hyperaldosteronism, thyrotoxicosis, coarctation of the aorta, and withdrawal syndromes
Oxygen saturation		
Pulse oximetry	SpO_2 < 97% associated with a heightened risk of severe complications (including non-respiratory)	May be decreased in any cardiorespiratory complication (e.g., pulmonary embolism)
CBC and blood film		
Hemoglobin	↑ due to intravascular volume depletion ↓ if microangiopathic hemolysis (with HELLP)	↑ due to volume depletion from any cause (e.g., vomiting) ↓ if microangiopathic hemolysis from other cause ↓ with any chronic anemia (nutritional or myelodysplasia) ↓ with acute bleeding of any cause
WBC and differential	↔	↑ due to neutrophilia of normal pregnancy ↑ with inflammation/infection ↑ with corticosteroids
Platelet count	↓ associated with adverse maternal outcome)	↓ with gestational, immune, or thrombotic thrombocytopenia, APS, AFLP, myelodysplasia
Blood film	RBC fragmentation	Microangiopathy due to mechanical causes (e.g., cardiac valvopathy, cavernous haemangioma), DIC or other disorders of endothelial function (e.g., APS, TTP-HUS, vasculitis, malignant hypertension)
Tests of coagulation*		
INR and aPTT	↑ with DIC, which is usually associated with placental abruption ↑ is associated with adverse maternal outcome	May be ↑ in APS or in DIC from other causes including sepsis, amniotic fluid embolism, stillbirth, massive haemorrhage, haemangiomas, or shock ↑ is prominent in AFLP
Fibrinogen	↔↓	↓ with all causes of DIC including massive haemorrhage, genetic disorders ↓ more profound with AFLP than with HELLP Usually normal in TTP-HUS (ADAMTS13 vWF cleaving protein may be moderately decreased in HELLP, but ADAMTS13 antibody should be absent)

Continued

Table 4. *Continued*

Investigations for diagnosis	Description in women with preeclampsia	Description in women with other conditions
Serum chemistry		
Serum creatinine	↑ due to hemoconcentration and/or renal failure	↑ with other acute or chronic kidney disease
	↑ associated with adverse maternal outcome	Renal failure prominent in malignant hypertension, TTP-HUS (along with thrombocytopenia), AFLP (along with liver dysfunction)
Serum uric acid	↑ associated with adverse maternal outcome	↑ with dehydration, medication (e.g., HCTZ), genetic causes
Glucose	↔	↓ with AFLP, insulin therapy
AST or ALT	↑ associated with adverse maternal outcome	↑ with AFLP and other PET imitators,† but to a lesser degree, and usually normal in TTP-HUS
		May be increased in other pregnancy-related conditions (e.g., intrahepatic cholestasis of pregnancy) or conditions not associated with pregnancy (e.g., viral hepatitis or cholecystitis)
LDH	↑ may be prominent; the ↑ is associated with adverse maternal outcome	↑ with AFLP, intravascular hemolysis
		↑ LDH/AST ratio (> 22) with TTP-HUS
Bilirubin	↑ unconjugated from hemolysis or conjugated from liver dysfunction	(early) ↑ in AFLP, ↑ with hemolytic anemia, other liver disease with dysfunction, genetic diseases
Albumin	↓ associated with adverse maternal and perinatal outcomes	↓ as negative acute phase reactant with acute severe illness, malnutrition, nephritic syndrome, crystalloid infusion
FETAL TESTING	Abnormalities are not specific to the cause of poor placentation and/or placental dysfunction	
Uterine artery Doppler velocimetry‡	Unilateral/bilateral notching, or elevated pulsatility index or resistance index may support a diagnosis of placental insufficiency including preeclampsia	
Fetal monitoring	Abnormal or atypical FHR tracing (e.g., decreased variability)	
Deepest amniotic fluid pocket	Oligohydramnios associated with adverse perinatal outcomes	
Ultrasonographic assessment of fetal growth	Usually intrauterine fetal growth restriction (typically asymmetrical but can be symmetrical if early and/or severe)	
Umbilical artery Doppler	Increased resistance, absent or reversed end-diastolic flow	
Ductus venosus Doppler	Increased resistance, especially absent or reverse A wave	
Middle cerebral artery Doppler	Cerebral redistribution (decreased resistance or “brain-sparing effect”). May be lost in extreme cases prior to fetal death	

RBC: red blood cell; TTP-HUS: thrombotic thrombocytopenic purpur–hemolytic uremic syndrome; UTI: urinary tract infection; SpO_2: oxygen saturation; CBC: complete blood count; HELLP: hemolysis, elevated liver enzyme, low platelet syndrome; WBC: white blood cell; APS: antiphopholipid antibody syndrome; AFLP: acute fatty liver of pregnancy; DIC: disseminated intravascular coagulation; INR international normalized ratio; aPTT: activated partial thromboplastin time; vWF: von Willebrand Factor; HCTZ: hydrochlorothiazide; AST: aspartate aminotransferase; ALT: alanine aminotransferase; PET: preeclampsia-eclampsia; LDH: lactate dehydrogenase; FHR: fetal heart rate

*Tests of coagulation are recommended if there is thrombocytopenia or placental abruption.

†“PET imitators” include AFLP, catastrophic APS, TTP-HUS, malignant hypertension, and scleroderma renal crisis.

‡Abnormal uterine artery Doppler velocimetry is practically defined at 22 to 24 weeks as bilateral notching with mean resistance index (RI) > 0.55 (i.e., > 50th centile), unilateral notching with mean RI > 0.65 (> 90th centile), or no notching with mean RI > 0.70 (> 95th centile).

Investigations to Classify HDPs

Recommendations

32. For women with pre-existing hypertension, the following should be performed in early pregnancy (if not previously documented): serum creatinine, fasting blood glucose, serum potassium, and urinalysis (III-D), and EKG. (II-2C)
33. Among women with pre-existing hypertension or those with a strong clinical risk marker for preeclampsia, additional baseline laboratory testing may be based on other considerations deemed important by health care providers. (III-C)
34. Women with suspected preeclampsia should undergo the maternal laboratory (II-2B) and pertinent fetal (II-1B) testing. (Table 4)
35. Doppler velocimetry-based assessment of the fetal circulation may be useful to support a placental origin for hypertension, proteinuria, and/or adverse conditions including intrauterine growth restriction, (II-2B) and for the timing of delivery. (I-A)
36. There is insufficient evidence to recommend use of the biophysical profile as part of a schedule of fetal testing in women with a hypertensive disorder of pregnancy. (II-2L)
37. If initial testing is reassuring, but there is ongoing concern about preeclampsia (e.g., change in maternal and/or fetal condition), maternal and fetal testing should be repeated. (III-C)

Comments

Most abnormalities found in maternal and fetal testing are non-specific. When preeclampsia is suspected, interpretation relies on findings of multiple (not single) abnormalities.

Preeclampsia imitators share manifestations with preeclampsia, but require different treatment.[27–31] (Table 5)

CHAPTER 2: PREDICTION AND PREVENTION

Predicting Preeclampsia

Recommendations

38. Women should be screened for clinical risk markers of preeclampsia from early pregnancy. (II-2C) (Table 6)
39. Consultation with an obstetrician or an obstetric internist, by telephone if necessary, should be considered for women with a history of previous preeclampsia or another strong clinical marker of increased preeclampsia risk, particularly multiple pregnancy, antiphospholipid antibody syndrome, significant proteinuria at first antenatal visit (usually early in pregnancy), or a pre-existing condition of hypertension, diabetes mellitus, or renal disease. (II-2B)
40. Screening using biomarkers or Doppler ultrasound velocimetry of the uteroplacental circulation cannot be recommended routinely at present for women at low or increased risk of preeclampsia until such screening has been shown to improve pregnancy outcome. (II-2C)

Preventing Preeclampsia and its Complications in Women at Low Risk

We based our recommendations on the need to prevent preeclampsia and/or its associated complications. Pregnant women are classified as being at either low or increased risk of preeclampsia, usually by the presence of one or more of the risk markers in Table 6 (see Predicting Preeclampsia).

Preventative interventions may be best started before 16 weeks' gestation when most of the physiologic transformation of uterine spiral arteries occurs. Such early intervention has the greatest potential to decrease early forms of preeclampsia.[32]

Women at low risk of preeclampsia have usually been from unselected populations of nulliparous and multiparous women.

Recommendations

41. Calcium supplementation of at least 1 g/d, orally, is recommended for women with low dietary intake of calcium (< 600 mg/d). (I-A)
42. The following are recommended for other established beneficial effects in pregnancy: abstention from alcohol for prevention of fetal alcohol effects (II-2E), exercise for maintenance of fitness (I-A), periconceptual use of a folate-containing multivitamin for prevention of neural tube defects (I-A), and smoking cessation for prevention of low birthweight and preterm birth. (I-E)
43. Periconceptual and ongoing use of a folate-containing multivitamin (I-B) or exercise (II-2B) may be useful in preventing preeclampsia.
44. Prostaglandin precursors and supplementation with magnesium or zinc are **not** recommended for preeclampsia prevention, but may be useful for prevention of other pregnancy complications. (I-C)
45. Dietary salt restriction during pregnancy (I-D), calorie restriction during pregnancy for overweight women (I-D), low-dose acetylsalicylic acid (I-E), vitamins C and E (based on current evidence) (I-E), and thiazide diuretics (I-E) are not recommended.

Table 5. Preeclampsia imitators

Pregnancy related	Not pregnancy related
Preeclampsia/HELLP syndrome	Malignant hypertension regardless of the cause
Acute fatty liver of pregnancy	Secondary causes of hypertension when associated with end-organ involvement (e.g., renal disease, pheochromocytoma)
	Disseminated intravascular coagulation (from any cause)
	Thrombotic thrombocytopenic purpura
	Hemolytic uremic syndrome
	Vasculitis or other systemic rheumatic condition (systemic lupus erythematous, scleroderma, cryoglobulinemia, catastrophic antiphospholipids syndrome)
	Sepsis
	Medications
	Cavernous hemangiomas
	Malignancy

46. There is insufficient evidence to make a recommendation about a heart-healthy diet (II-2L); workload or stress reduction (including bedrest) (II-2L); supplementation with iron with or without folate (I-L); vitamin D (I-L); pyridoxine (I-L); or food rich in flavonoids. (I-L)

Preventing Preeclampsia and its Complications in Women at Increased Risk

Women at increased risk of preeclampsia are most commonly identified by a personal or family history of an HDP, chronic medical disease, and/or abnormal uterine artery Doppler before 24 weeks' gestation. Combining clinical, biochemical, and/or ultrasonographic risk markers may better identify women at increased preeclampsia risk (see "Prediction"); however, no intervention trial has used such an approach to evaluate preventative therapy.[33,34]

Recommendations

47. Low-dose acetylsalicylic acid and calcium supplementation (of at least 1 g/d) for women with low calcium intake are recommended for preventions of preeclampsia in women at high risk. (I-A)
48. Acetylsalicylic acid should be: taken in a low dose (75–162 mg/d), (III-B) administered at bedtime, (I-B) initiated after diagnosis of pregnancy but before 16 weeks' gestation, (I-B) and considered for continuation until delivery. (I-C)
49. Prophylactic doses of low-molecular-weight heparin may be discussed in women with previous placental complications (including preeclampsia) to prevent the recurrence of severe or early-onset preeclampsia, preterm delivery, and/or infants that are small for gestational age. (I-B)
50. The following may be useful: L-arginine (I-B), increased rest at home in the third trimester (I-C), and reduction of workload or stress. (III-C)
51. The following may be useful for prevention of other pregnancy complications: prostaglandin precursors (I-B), magnesium supplementation (I-C), and heparin to prevent venous thromboembolic disease. (I-B)
52. The following are recommended for other established beneficial effects in pregnancy (as discussed for women at low risk of preeclampsia): abstention from alcohol (II-2E), periconceptual use of a folate-containing multivitamin (I-A), and smoking cessation. (I-E)
53. The following are **not** recommended: calorie restriction in overweight women during pregnancy (I-D), weight maintenance in obese women during pregnancy (III-D), antihypertensive therapy specifically to prevent preeclampsia (I-D), and vitamins C and E. (I-E)
54. There is insufficient evidence to make a recommendation about the usefulness of the following: the heart-healthy diet (III-L); exercise (I-L); selenium (I-L); garlic (I-L); zinc, pyridoxine, iron (with or without folate), vitamin D, or multivitamins with/without micronutrients. (III-L)

CHAPTER 3: TREATMENT OF THE HDPs

Dietary and Lifestyle Changes

Recommendations

55. There is insufficient evidence to make a recommendation about the usefulness of the following: new severe dietary salt restriction for

Table 6. Risk markers for preeclampsia

		Current pregnancy	
Demographics and family history	Past medical or obstetric history*	First trimester	Second or third trimester
	Previous preeclampsia **Anti-phospholipid antibody syndrome** **Pre-existing medical condition(s)** • Pre-existing hypertension or booking† diastolic BP ≥ 90 mmHg • Pre-existing renal disease or booking† proteinuria • Pre-existing diabetes mellitus	**Multiple pregnancy**	
Maternal age ≥ 40 years‡ Family history of preeclampsia (mother or sister) Family history of early-onset cardiovascular disease	Lower maternal birthweight and/or preterm delivery Heritable thrombophilias§ Increased pre-pregnancy triglycerides Non-smoking Cocaine and metamphetamine use Previous miscarriage at ≤ 10 weeks with same partner	Overweight/obesity First ongoing pregnancy New partner Short duration of sexual relationship with current partner Reproductive technologies‖ Inter-pregnancy interval ≥ 10 years Booking† sBP ≥ 130 mmHg, or booking dBP ≥ 80 mmHg Vaginal bleeding in early pregnancy Gestational trophoblastic disease Abnormal PAPP-A or free βhCG	Elevated BP (gestational hypertension)¶ Abnormal AFP, hCG, inhA, or E_3# Excessive weight gain in pregnancy Infection during pregnancy (e.g., UTI, periodontal disease) Abnormal uterine artery Doppler** IUGR Investigational laboratory markers††

AFP: alfafetoprotein; inhA: inhibin A; E_3: estradiol; UTI: urinary tract infection; sBP: systolic BP; dBP: diastolic BP; PAPP-A: pregnancy-associated plasma protein A.

*Women at increased risk (who should be considered for specialty referral) are those with one of the bolded factors, or two or more of the unbolded markers.

†First antenatal visit, usually early in pregnancy.

‡Maternal age was considered as a continuous variable in the SCOPE study.

§Heritable thrombophilia includes factor V Leiden gene mutation and protein S deficiency.

‖Subfertility and its treatment (especially the use of donor eggs, sperm and/or gametes), after correction for multiple gestations.

¶Elevated BP is defined as dBP ≥ 110 mmHg before 20 weeks, 2nd trimester mean arterial pressure of ≥ 85 mmHg, or a 2nd trimester sBP ≥ 120 mmHg. standardized cut-offs for 24-hour ambulatory BP or home BP monitoring have not been established.

#Decreased first trimester PAPP-A ≤ 5th centile, 110 decreased first or second trimester placental growth factor, unexplained increased second trimester AFP, increased second trimester hCG, increased first or second trimester inhA, increased second trimester activin.

**Abnormal uterine artery Doppler velocimetry is practically defined at 22 to 24 weeks as bilateral notching with mean resistance index (RI) > 0.55 (i.e., > 50th centile), unilateral notching with mean RI > 0.65 (> 90th centile), or no notching with mean RI > 0.70 (> 95th centile).

††Investigational markers include, in the first trimester: PAPP-A, PlGF, PP-13, and in the second trimester: elevated sFlt-1/PlGF (soluble fms-like tyrosine kinase, placental growth factor), PAI-1/PAI-2 (plasminogen activator inhibitor) von Willebrand factor, and leptin.

Table 7. The most commonly used agents for treatment of blood pressure ≥ 160/110 mmHg

Agent	Dosage	Onset	Peak	Duration	Comments
Labetalol	Start with 20 mg IV; repeat 20 to 80 mg IV q 30 min, or 1 to 2 mg/min, max 300 mg (then switch to oral)	5 min	30 min	4 hr	Best avoided in women with asthma or heart failure. Neonatology should be informed if the woman is in labour, as parenteral labetalol may cause neonatal bradycardia.
Nifedipine	5 to 10 mg capsule to be swallowed, or bitten then swallowed, every 30 min	5 to 10 min	30 min	~6 hr	Staff should be aware of the distinction between short-acting nifedipine capsules used to treat severe hypertension and both the intermediate-acting PA tablet (which can be used for treatment of non-severe or severe hypertension), and the slow-release tablets (XL) that are used for non-severe hypertension.
Hydralazine	Start with 5 mg IV; repeat 5 to 10 mg IV every 30 min, or 0.5 to 10 mg/hr IV, to a maximum of 20 mg IV (or 30 mg IM)	5 min	30 min		May increase the risk of maternal hypotension.

IV: intravenous; IM: intramuscular; PA: prolonged action; XL: slow release

women with any HDP, ongoing salt restriction among women with pre-existing hypertension, heart-healthy diet, and calorie restriction for obese women. (III-L)

56. There is insufficient evidence to make a recommendation about the usefulness of exercise, workload reduction, or stress reduction. (III-L)
57. For women with gestational hypertension (without preeclampsia), some bed rest in hospital (vs. unrestricted activity at home) may be useful to decrease severe hypertension and preterm birth. (I-B)
58. For women with preeclampsia who are hospitalized, strict bed rest is **not** recommended. (I-D)
59. For all other women with an HDP, the evidence is insufficient to make a recommendation about the usefulness of some bed rest, which may nevertheless be advised based on practical considerations. (III-C)

The following recommendations apply to women with either pre-existing or gestational hypertension.

Place of Care

Recommendations

60. In-patient care should be provided for women with severe hypertension or severe preeclampsia. (II-2B).
61. A component of care through hospital day units or home care can be considered for women with non-severe preeclampsia or non-severe pre-existing or gestational hypertension. (I-B, II-2B)

Antihypertensive Therapy for Severe Hypertension

Recommendations

62. Blood pressure should be lowered to < 160 mmHg systolic and < 110 mmHg diastolic. (I-A)
63. Initial antihypertensive therapy in the hospital setting should be with nifedipine short-acting capsules, parenteral hydralazine, or parenteral labetalol. (I-A) (Table 7)
64. Alternative antihypertensive medications include a nitroglycerin infusion (I-B), oral methyldopa (I-B), oral labetalol (I-B), oral clonidine (III-B), or postpartum, oral captopril. (III-B)
65. Refractory hypertension may be treated with sodium nitroprusside. (III-B)
66. Nifedipine and magnesium sulphate can be used contemporaneously. (II-2B)
67. Magnesium sulphate is not recommended solely as an antihypertensive agent. (I-E)
68. Continuous fetal heart rate monitoring is advised until blood pressure is stable. (III-L)

Antihypertensive Therapy for Non-Severe Hypertension *Without* Comorbid Conditions

Recommendations

69. Antihypertensive drug therapy may be used to keep systolic blood pressure at 130 to 155 mmHg and diastolic blood pressure at 80–105 mmHg. (I-B)

Table 8. Doses of the most commonly used agents for treatment of blood pressures 149 to 159/90 to 105 mmHg

Agent	Dosage	Comments
Methyldopa	250 to 500 mg po bid-qid (max 2 g/d)	There is no evidence to support a loading dose of methyldopa.
Labetalol	100 to 400 mg po bid-tid (max 1200 mg/d)	Some experts recommend a starting dose of 200 mg po bid.
Nifedipine*	XL preparation (20 to 60 mg po OD, max 120 mg/d)	Ensure that the correct form of nifedipine has been prescribed so that the XL preparation is not confused with the capsules.

XL: slow release

*The prolonged action nifedipine tablet is no longer available in Canada.

70. The choice of antihypertensive agent for initial treatment should be based on characteristics of the patient, contraindications to a particular drug, and physician and patient preference. (III-C)
71. Initial therapy in pregnancy can be with one of a variety of antihypertensive agents available in Canada: methyldopa (I-A), labetalol (I-A), other beta-blockers (acebutolol, metoprolol, pindolol, and propranolol), (I-B) and calcium channel blockers (nifedipine). (I-A) (Table 8)
72. Angiotensin-converting enzyme inhibitors and angiotensin receptor blockers should not be used during pregnancy. (II-2E)
73. Atenolol and prazosin are not recommended prior to delivery. (I-D)

For Non-Severe Hypertension (BP of 140–159/90–109 mmHg) *With* Comorbid Conditions

Recommendations

74. For women with comorbid conditions, antihypertensive drug therapy should be used to keep systolic blood pressure at < 140 mmHg and diastolic blood pressure at < 90 mmHg. (III-C)
75. Initial therapy in pregnancy can be with one of a variety of antihypertensive agents as listed for women without co-morbidities. (III-C)
76. Captopril, enalapril, or quinapril may be used postpartum, even during breastfeeding. (III-B)

Corticosteroids for Acceleration of Fetal Pulmonary Maturity

Recommendations

77. Antenatal corticosteroid therapy should be considered for all women who present with preeclampsia at ≤ 34+6 weeks' gestation. (I-A)
78. Antenatal corticosteroid therapy should be considered for women who present at ≤ 34+6 weeks' gestation with gestational hypertension (despite the absence of proteinuria or adverse conditions) only if delivery is contemplated within the next 7 days. (III-L)
79. A rescue dose of corticosteroids may be considered for women at ≤ 34+6 weeks' gestation who remain at high risk of preterm delivery 7 days or more after an initial course of antenatal corticosteroids. (I-C)
80. Antenatal corticosteroids may be considered for women delivered by elective Caesarean delivery at ≤ 38+6 weeks' gestation to reduce respiratory morbidity. (I-B)

Comments

When administered at ≤ 34+6 weeks' gestation, antenatal corticosteroids accelerate fetal pulmonary maturity and decrease neonatal mortality and morbidity, including among women with HDPs.[35] RCTs that administered steroids at 33+0 to 34+6 weeks resulted in reduced neonatal RDS.[35]

Prior to elective Caesarean section at ≤ 38+6 weeks' gestation, antenatal corticosteroids decrease the excess neonatal respiratory morbidity and NICU admissions.[36,37] All subgroup analyses have not necessarily revealed such benefits following Caesarean or vaginal delivery.[35]

Timing of Delivery for Women With Preeclampsia

Delivery is the only intervention that initiates resolution of preeclampsia, and women with gestational hypertension or pre-existing hypertension may develop preeclampsia.

Recommendations

81. Consultation with an obstetrician (by telephone if necessary) is mandatory in women with severe preeclampsia. (III-B)
82. All women with severe preeclampsia should be delivered immediately (either vaginally or by Caesarean), regardless of gestational age. (III-C)
83. For women with non-severe preeclampsia at < 24+0 weeks' gestation, counselling should include, as an option, information about delivery within days. (II-2B)
84. For women with non-severe preeclampsia at 24+0 to 33+6 weeks' gestation, expectant management

should be considered, but only in perinatal centres capable of caring for very preterm infants. (I-B)

85. For women with non-severe preeclampsia at 34+0 to 36+6 weeks' gestation, there is insufficient evidence to make a recommendation about the benefits or risks of expectant management. (III-L)
86. For women with preeclampsia at ≥ 37+0 weeks' gestation, immediate delivery is recommended. (I-A)
87. For women with non-severe preeclampsia complicated by hemolysis, elevated liver enzymes, low platelets syndrome at 24+0 to 34+6 weeks' gestation, consider delaying delivery long enough to administer antenatal corticosteroids for acceleration of fetal pulmonary maturity if there is temporary improvement in maternal laboratory testing. (II-2B)
88. All women with hemolysis, elevated liver enzymes, low platelets syndrome at ≥ 35+0 weeks' gestation should be considered for immediate delivery. (II-2B)

Timing of Delivery for Women With Gestational Hypertension

Recommendations

89. For women with gestational hypertension (without preeclampsia) at ≥ 37+0 weeks' gestation, delivery within days should be discussed. (I-B)
90. For women with gestational hypertension (without preeclampsia) at < 37+0 weeks' gestation, there is insufficient evidence to make a recommendation about the benefits or risks of expectant management. (III-L)

Timing of Delivery for Women With Pre-Existing Hypertension

Recommendation

91. For women with uncomplicated pre-existing hypertension who are otherwise well at ≥ 37+0 weeks' gestation, delivery should be considered at 38+0 to 39+6 weeks' gestation. (II-1B)

Mode of Delivery

Recommendations

92. For women with any hypertensive disorder of pregnancy, vaginal delivery should be considered unless a Caesarean delivery is required for the usual obstetric indications. (II-2B)
93. If vaginal delivery is planned and the cervix is unfavourable, then cervical ripening should be used to increase the chance of a successful vaginal delivery. (1-A)
94. At a gestational age remote from term, women with a hypertensive disorder of pregnancy with evidence of fetal compromise may benefit from delivery by emergency Caesarean section. (II-2B)
95. Antihypertensive treatment should be continued throughout labour and delivery to maintain systolic blood pressure at < 160 mmHg and diastolic blood pressure at < 110 mmHg. (II-2B)
96. The third stage of labour should be actively managed with oxytocin, 5 units intravenous or 10 units intramuscular, particularly in the presence of thrombocytopenia or coagulopathy. (I-A)
97. Ergometrine maleate should not be administered to women with any hypertensive disorder of pregnancy, particularly preeclampsia or gestational hypertension; alternative oxytocics should be considered. (II-3D)

Anaesthesia: General Principles

Recommendations

98. The anaesthesiologist should be informed when a woman with preeclampsia is admitted to the delivery suite. (II-3B)
99. Early insertion of an epidural catheter (in the absence of contraindications) is recommended for control of labour pain. (I-A)
100. In the absence of contraindications, all of the following are acceptable methods of anaesthesia for women undergoing Caesarean delivery: epidural, spinal, combined spinal-epidural, and general anaesthesia. (I-A)
101. A routine fixed intravenous fluid bolus should not be administered prior to neuraxialanaesthesia. (I-E)

Anaesthesia: Fluid Administration

Recommendations

102. Intravenous and oral fluid intake should be minimized in women with preeclampsia, to avoid pulmonary edema. (II-2B)
103. Fluid should not be routinely administered to treat oliguria (< 15 mL/hr for 6 consecutive hours). (III-D)
104. For treatment of persistent oliguria, neither dopamine nor furosemide is recommended. (I-E)

105. Phenylephrine or ephedrine may be used to prevent or treat hypotension during neuraxial anaesthesia. (I-A)

Monitoring

Recommendations

106. Arterial line insertion may be used for continuous arterial blood pressure monitoring when blood pressure control is difficult or there is severe bleeding. (II-3B)
107. Central venous pressure monitoring is not routinely recommended, and if a central venous catheter is inserted, it should be used to monitor trends and not absolute values. (II-2D)
108. Pulmonary artery catheterization is not recommended unless there is a specific associated indication (III-D), and then only in an intensive care unit setting. (III-B)

Coagulation

Recommendations

109. Upon admission to delivery suite, women with preeclampsia should have a platelet count done. (II-1A)
110. Neuraxial analgesia and/or anaesthesia are appropriate in women:
    a. with preeclampsia, provided there are no associated coagulation concerns (II-2E) (Table 9);
    b. with a platelet count $\geq 75 \times 10^9$/L, (II-2B);
    c. taking low-dose acetylsaliclylic acid in the presence of an adequate platelet count (I-A);
    d. receiving unfractionated heparin in a dose of $\leq$ 10 000 IU/d subcutaneously, 4 hours after the last dose and possibly immediately after the last dose without any delay (III-B);
    e. receiving unfractionated heparin in a dose $>$ 10 000 IU/d subcutaneously if they have a normal activated partial thromboplastin time 4hours after the last dose (III-B);
    f. receiving intravenous heparin in a therapeutic dose if they have a normal activated partial thromboplastin time 4 hours after the last dose (III-B); or
    g. receiving low-molecular-weight heparin 10 to 12 hours after a prophylactic dose, or 24 hours after a therapeutic dose. (III-B)

Aspects of Care Specific to Women With Pre-Existing Hypertension

Recommendations

111. Pre-conceptual counselling for women with pre-existing hypertension is recommended. (III-C)
112. The following antihypertensive drugs are all acceptable for use in the first trimester of pregnancy: methyldopa, labetalol, and nifedipine. (II-2B)
113. Angiotensin-converting enzyme inhibitors and angiotensin receptor blockers should be discontinued when planning pregnancy or as soon as pregnancy is diagnosed. (II-2D)
114. Atenolol should be discontinued when pregnancy is diagnosed. (I-D)
115. Planned changes in antihypertensive agent(s) for care in pregnancy should be made while the woman is planning pregnancy if the woman has uncomplicated pre-existing hypertension, or, if in the presence of comorbid conditions, she is likely to conceive easily (within 12 months). (III-L)

Aspects of Care for Women With Preeclampsia: Magnesium Sulphate for Preventing or Treating Eclampsia

Recommendations

116. Magnesium sulphate is recommended for first-line treatment of eclampsia. (I-A)
117. Magnesium sulphate is recommended as prophylaxis against eclampsia in women with severe preeclampsia. (I-A)
118. Magnesium sulphate may be considered as prophylaxis against eclampsia in women with non-severe preeclampsia but with severe hypertension, headaches/visual symptoms, right upper quadrant/epigastric pain, platelet count $< 100\,000 \times 10^9$/L, progressive renal insufficiency, and/or elevated liver enzymes, based on cost considerations. (I-C)
119. Magnesium sulphate should be used in standard dosing, usually 4 g intravenous loading dose followed by 1 g/hour. (I-A)
120. Routine monitoring of serum magnesium levels is not recommended. (I-E)
121. Phenytoin and benzodiazepines should not be used for eclampsia prophylaxis or treatment, unless there is a contraindication to magnesium sulphate or it is ineffective. (I-E)

Table 9. Eligibility for neuraxial anaesthesia

Treatment with ASA or heparin	Normal platelet count	Low platelet count (normal INR and aPTT)	Abnormal INR or aPTT (regardless of platelet count)*
None *or* low-dose ASA	✓	✓ if platelet count > 75 × 10^9/L Unclear if platelet count 50 to 75 × 10^9/L X if platelet count < 50 × 10^9/L	× Contraindicated
UFH			
≤ 10 000 IU/d (SC)†	✓ 0 to 4 hr after last dose	Unclear	
> 10 000 IU/d (SC)‡	✓ 4 hr after last dose and a normal aPTT	Unclear	
Therapeutic dose (IV)	✓ 4 hr after last dose and a normal aPTT	Unclear	
LMWH			
Prophylactic dose	✓ 10 to 12 hr after last dose	Unclear	
Therapeutic dose	✓ 24 hr after last dose	Unclear	
Low dose ASA + prophylactic UFH or LMWH§	Unclear‖	Unclear	

INR: international normalized ratio; aPTT: activated partial thromboplastin time; UFH: unfractionated heparin; SC: subcutaneous; IV: intravenous; LMWH: low-molecular-weight heparin

Note: These recommendations are based on the absence of a rapidly falling platelet count or a known platelet dysfunction (e.g., von Willebrand's disease).

*Other than a lupus anticoagulant

†Prophylactic dosing is defined as ≤ 10 000 IU/d

‡Therapeutic dosing (SC) is defined as > 10 000 IU/d

§Prophylactic doses of UFH are defined as ≤ 10 000 IU/d

‖Unless ASA is stopped 7 days or more before delivery

122. In women with pre-existing or gestational hypertension, magnesium sulphate should be considered for fetal neuroprotection in the setting of imminent preterm birth (within the next 24 hours) at ≤ 31+6 weeks. (1-A)
123. Delivery should not be delayed in order to administer antenatal magnesium sulphate for fetal neuroprotection if there are maternal and/or fetal indications of emergency delivery. (III-E)

There is no international consensus on what defines severe preeclampsia. This document defines it as preeclampsia requiring delivery due to serious maternal end-organ involvement and/or fetal compromise (see "Classification of HDPs"). For eclampsia prevention in the setting of non-severe preeclampsia, we have added to the indication for magnesium sulphate (in Recommendation 120) the following symptoms/signs as these are included in the definition of severe preeclampsia by other organizations: severe hypertension, headaches/visual symptoms, right upper quadrant/epigastric pain, platelet count < 100 000 × 10^9/L, progressive renal insufficiency, and/or elevated liver enzymes.

Aspects of Care for Women With Preeclampsia: Plasma Volume Expansion

Recommendation

124. Plasma volume expansion is not recommended for women with preeclampsia. (I-E)

Therapies for HELLP Syndrome

Recommendations

125. Every obstetrical centre should be aware of the local delay between ordering and receiving platelets units. (III-B)
126. For a platelet count of < 20 × 10^9/L with hemolysis, elevated liver enzymes, low platelets

Table 10. Recommendations for the transfusion of platelets related to mode of delivery in HELLP

	Mode of delivery	
Platelet count	Caesarean delivery	Vaginal delivery
< 20 × 10^9/L	✓	✓
20 to 49 × 10^9/L	✓	Consider in presence of: • excessive active bleeding • known platelet dysfunction • platelet count falling rapidly • coagulopathy
≥ 50 × 10^9/L	Consider in presence of: • excessive active bleeding • known platelet dysfunction • platelet count falling rapidly • coagulopathy	Consider in presence of: • excessive active bleeding • known platelet dysfunction • platelet count falling rapidly • coagulopathy
Regardless of the platelet count	No platelets should be transfused if there is a strong suspicion of HIT or TTP-HUS	

HIT: heparin-induced thrombocytopenia; TTP-HUS: thrombotic thrombocytopenic purpura–hemolytic uremic syndrome

syndrome, platelet transfusion is recommended regardless of mode of delivery. (III-B) (Table 9)

127. For a platelet count of 20 × 10^9 to 49 × 10^9/L with hemolysis, elevated liver enzymes, low platelets syndrome, platelet transfusion is recommended prior to Caesarean delivery. (III-B) (Table 9)
128. For a platelet count of 20 × 10^9 to 49 × 10^9/L with hemolysis, elevated liver enzymes, low platelets syndrome, platelet transfusion should be considered prior to vaginal delivery if there is excessive active bleeding, known platelet dysfunction, a rapidly falling platelet count, or coagulopathy. (II-2D) (Table 10)
129. For a platelet count of ≥ 50 × 10^9/L with hemolysis, elevated liver enzymes, low platelets syndrome, platelet transfusion and/or packed red blood cells should be considered prior to either Caesarean or vaginal delivery only if there is excessive active bleeding, known platelet dysfunction, a rapidly falling platelet count, or coagulopathy. (III-B)
130. We do not recommend corticosteroids for treatment of hemolysis, elevated liver enzymes, low platelets syndrome until they have been proven to decrease maternal morbidity. (II-3L)
131. We recommend against plasma exchange or plasmapheresis for hemolysis, elevated liver enzymes, low platelets syndrome, particularly within the first 4 days postpartum. (II-3E)

Care in the 6 Weeks Postpartum

Recommendations

132. Blood pressure should be measured during the time of peak postpartum blood pressure, at days 3 to 6 after delivery. (III-B)
133. Women with postpartum hypertension should be evaluated for preeclampsia (either arising de novo or worsening from the antenatal period). (II-2B)
134. Consideration should be given to continuing antihypertensive therapy postpartum, particularly in women with antenatal preeclampsia and those who delivered preterm. (II-2L)
135. Severe postpartum hypertension must be treated with antihypertensive therapy to keep systolic blood pressure < 160 mmHg and diastolic blood pressure < 110 mmHg. (I-A)
136. In women without co-morbidities, antihypertensive therapy should be considered to treat non-severe postpartum hypertension to keep blood pressure < 140/90 mmHg. (III-L)
137. Women with co-morbidities other than pre-gestational diabetes mellitus should be treated to keep blood pressure < 140/90 mmHg (III-C)
138. Women with pre-gestational diabetes mellitus should be treated to keep blood pressure < 130/80 mmHg. (III-C)
139. Antihypertensive agents generally acceptable for use in breastfeeding include the following: nifedipine XL, labetalol, methyldopa, captopril, and enalapril. (III-B)

140. There should be confirmation that end-organ dysfunction of preeclampsia has resolved. (III-C)
141. Non-steroidal anti-inflammatory drugs should not be given postpartum if hypertension is difficult to control, there is evidence of kidney injury (oliguria and/or creatinine≥ 90 μM), or platelets are < 50 to 10^9/L. (III-C)
142. Postpartum thromboprophylaxis should be considered in women with preeclampsia, particularly in the presence of other risk factors. (II-2B)

Care Beyond 6 Weeks Postpartum

Recommendations

143. Women with a history of severe preeclampsia (particularly those who presented or delivered before 34 weeks' gestation) should be screened for pre-existing hypertension and underlying renal disease. (II-2B)
144. Referral for internal medicine or nephrology consultation (by telephone if necessary) should be considered for women with:
    (i) postpartum hypertension that is difficult to control, or
    (ii) women who had preeclampsia and have at 3-6 months postpartum either ongoing proteinuria, decreased estimated glomerular filtration rate (eGFR) (< 60 mL/min), or another indication of renal disease, such as abnormal urinary sediment. (III-A)
145. Women who are overweight should be encouraged to attain a healthy body mass index to decrease risk in future pregnancy (II-2A) and for long-term health. (I-A)
146. Women with pre-existing hypertension or persistent postpartum hypertension should undergo the following investigations (if not done previously) at least six weeks postpartum: urinalysis; serum sodium, potassium and creatinine; fasting glucose; fasting lipid profile; and standard 12-lead electrocardiography. (III-L)
147. Women who are normotensive but who have had a hypertensive disorder of pregnancy, may benefit from assessment of traditional cardiovascular risk markers. (II-2B)
148. All women who have had a hypertensive disorder of pregnancy should pursue a healthy diet and lifestyle. (I-B)

Effects of Maternal Hypertension and Its Therapies on Child Neurobehavioural Development

Recommendations

149. Clinicians should be aware that gestational hypertension and preeclampsia may each be associated with an increase in adverse paediatric neurodevelopmental effects, such as inattention and externalizing behaviours (e.g., aggressiveness). (II-2B).
150. Clinicians should be reassured that there is no compelling evidence that antihypertensive medications (specifically labetalol, nifedipine, or methyldopa) are themselves associated with clear adverse neurodevelopmental effects. (I-B)

CHAPTER 4: PATIENT PERSPECTIVE

Recommendations

151. Health care providers should be alert to symptoms of posttraumatic stress following a hypertensive disorder of pregnancy and refer women for appropriate evaluation and treatment. (II-2B)
152. Health care providers should inform their patients, antepartum and postpartum, about preeclampsia, its signs and symptoms, and the importance of timely reporting of symptoms to health care providers. (II-2B)
153. Information should be re-emphasized at subsequent visits. (III-C)

CHAPTER 5: KNOWLEDGE TRANSLATION TOOLS AND IMPLEMENTATION OF THE GUIDELINE

The Appendix (Table 10 in the full document[3]) lists tools to support the application of this guideline. Some websites provide general information about BP measurement for non-pregnant patients, but the recommendations are similar enough to those for pregnant women to be useful. Patients, their partners, and their care providers should be well educated about the HDP, and relevant sites are listed.

Implementation of any evidence depends on individual knowledge and beliefs, as well as institutional culture. Strong recommendations should be incorporated into clinical practice. In well-resourced settings, almost all preeclampsia-related maternal deaths involve substandard care.[38]

Some updates to the 2008 SOGC guidelines on the HDP may require additional effort to implement.

Recommendation 9 states that all measurement devices used in hospitals or offices should be checked regularly against a calibrated device may not be possible for all Canadian hospitals and offices to do on a regular basis.

Physicians should consider the category "other HDP" (white-coat and masked hypertension) as part of the classification of hypertensive women and consider using some form of out-of-office BP measurement to evaluate women with non-severe pre-existing or gestational hypertension.

Health care providers should inform pregnant women about the symptoms and signs of the HDPs and refer them to appropriate knowledge translation tools.

We recommend the use of corticosteroids for women ≤ 34+6 weeks' gestation who are at high risk of delivery within the next seven days. This gestational age cut-off represents a fundamental change in practice that will require discussion.

Physicians should be familiar with the blood bank policies of their own hospital.

Physicians should be aware of postpartum signs of maternal posttraumatic stress disorder and the maternal and perinatal long-term effects of HDPs, especially at this vulnerable time in maternal care when the maternity care provider is often handing back care to the primary care physician.

The reader is reminded to refer to the full open-access guideline published in *Pregnancy Hypertension*,[3] which contains not only the recommendations and tables presented here, but also all explanatory text and additional references.

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APPENDIX. KNOWLEDGE TRANSLATION TOOLS FOR HDP

Tool	Resource	Comment
PATIENT INFORMATION		
BP measurement by patients		
Canadian Hypertension Education Program (CHEP)	http://www.hypertension.ca/measuring-blood-pressure (English) http://www.hypertension.ca/fr/mesures-dp1 (French)	This website gives patients basic information about BP measurement and gives instructions on self-measurement.
National Heart Foundation of Australia	http://www.heartfoundation.org.au/SiteCollectionDocuments/Self-Management-BP.pdf	This website gives information about the self-measurement of BP by patients and advice about buying a machine.
Heart and Stroke Foundation	https://ehealth.heartandstroke.ca/heartstroke/bpap.net/vid_measure_bp.html	This link refers to a movie that gives instructions for self-measurement of BP.
Société Canadienne d/hypertension	http://hypertension.ca/measuring-blood-pressure http://hypertension.ca/fr/mesures-dp1	This website gives detailed information in English and French (with a poster in English) although the images are of older patients.
Canadian Hypertension Education Program (CHEP)	https://www.youtube.com/watch?v=eqajdX5XU9Y&feasture=plcp	This website gives detailed video on home BP measurement (outside pregnancy).
Brochure	http://www.RCOG.org.uk	This also includes your risk of recurrence.
BP measurement and pre-existing hypertension		
Heart and Stroke Foundation	http://www.heartandstroke.ca	This website gives information about hypertension outside of pregnancy, blood pressure monitoring and medication.
Impact of pre-existing hypertension on pregnancy		
American Heart Association document: Chronic Hypertension in Pregnancy	https://circ.ahajournals.org/content/115/7/e188.full	This document explains in an understandable way how chronic hypertension and pregnancy influence each other and what the symptoms of preeclampsia that women should be aware of.
Preeclampsia awareness		
Preeclampsia Foundation: Preeclampsia Education Tool	http://www.preeclampsia.org/market-place	This tool explains the risks and symptoms of preeclampsia and how to act on them. This tool has shown to be effective in improving patient knowledge in an RCT (120 women).
Preeclampsia Foundation: Educational magnets and symptom Pads	http://www.preeclampsia.org/market-place	This website gives quick checklists of signs and symptoms of preeclampsia.

Continued

APPENDIX. *Continued*

Tool	Resource	Comment
Patient education once preeclampsia develops		
Preeclampsia Foundation Brochures: • HELLP syndrome • Preeclampsia FAQ • Preeclampsia and heart diseases	http://www.preeclampsia.org/market-place	These are available in English and Spanish.
Hôpital Maisonneuve-Rosemont, Centre affilié à l'Université de Montréal: Brochure on preeclampsia.	http://biblio.hmr.qc.ca/Publications_pdf/H/hypertension_sfe080.pdf	French brochure for patients about preeclampsia.
Patient education after preeclampsia		
Preeclampsia Foundation; APEC: Educational pamphlet	http://www.preeclampsia.org/market-place	Educational brochure about cardiovascular risks associated with preeclampsia.
HEALTH CARE PROVIDER INFORMATION		
BP measurement		
WHO document: detecting preeclampsia, a practical guide, 2005	http://www.who.int/reproductivehealth/publications/maternal_perinatal_health/MSM_92_3_/en/index.html	This document contains instructions how to measure blood pressure and proteinuria in pregnant women, and how to diagnose hypertensive disorders in pregnancy. This tool is for health care providers.
Approved BP measurement devices		
Canadian Hypertension Education Program (CHEP)	http://www.hypertension.ca/devices-endorsed-by-hypertension-canada-dp1	This website gives an oversight of recommended blood pressure devices.
Educational Trust	http://www.dableducational.org/sphygmomanometers/devices_1_clinical.html	This website gives an oversight of recommended blood pressure devices, outside of and during pregnancy.
Clinical practice guidelines from other countries		
NICE guidelines (UK, 2010)	http://www.nice.org.uk/nicemedia/live/13098/50475/50475.pdf	Graded recommendations
Australasian guidelines (Australia and New Zealand, 2008)	http://www.somanz.org/pdfs/somanz_guidelines_2008.pdf	Very practical but evidence not graded
College of Obstetricians and Gynecologists	http://www.acog.org/~/media/Task%20Force%20and%20Work%20Group%20Reports/HypertensioninPregnancy.pdf	Graded recommendations
WHO guidelines	http://whqlibdoc.who.int/publications/2011/9789241548335_eng.pdf	