MANAGING HYPERTENSION IN PREGNANCY

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Hypertension is the most common medical problem encountered in pregnancy and remains an important cause of maternal, and fetal, morbidity and mortality. It complicates up to 15% of pregnancies and accounts for approximately a quarter of all antenatal admissions. The hypertensive disorders of pregnancy cover a spectrum of conditions, of which pre-eclampsia poses the greatest potential risk and remains one of the most common causes of maternal death.

Normal Physiological Change In Blood Pressure During Pregnancy

Early in the first trimester there is a fall in blood pressure caused by active vasodilatation, achieved through the action of local mediators such as prostacyclin and nitric oxide. This reduction in blood pressure primarily affects the diastolic pressure and a drop of 10 mm Hg is usual by 13–20 weeks gestation.血 Pressure continues to fall until 22–24 weeks when a nadir is reached. After this, there is a gradual increase in blood pressure until term when pre-pregnancy levels are attained. Immediately after delivery blood pressure usually falls, then increases over the first five postnatal days. Even women whose blood pressure was normal throughout pregnancy may experience transient hypertension in the early post partum period, perhaps reflecting a degree of vasomotor instability.

CLASSIFICATION

Hypertensive disorders during pregnancy are classified into 4 categories, as recommended by the National High Blood Pressure Education Program Working Group on High Blood Pressure in Pregnancy.

- Chronic hypertension
- Preeclampsia-eclampsia
- Preeclampsia superimposed on chronic hypertension
- Gestational hypertension (transient hypertension of pregnancy)

CHRONIC HYPERTENSION

Chronic hypertension is defined as blood pressure exceeding 140/90 mm Hg before pregnancy or before 20 weeks’ gestation. When hypertension is first identified during a woman’s pregnancy and she is at less than 20 weeks’ gestation, blood pressure elevations usually represent chronic hypertension.

In contrast, new onset of elevated blood pressure readings after 20 weeks’ gestation mandates the consideration and exclusion of preeclampsia. Preeclampsia occurs in up to 5% of all pregnancies, in 10% of first pregnancies, and in 20-25% of women with a history of chronic hypertension. Hypertensive disorders in pregnancy may cause maternal and fetal morbidity, and they remain a leading source of maternal mortality.

Chronic hypertension is a primary disorder in 90-95% of cases and may be either essential (90%) or secondary to some identifiable underlying disorder, such as renal parenchymal disease (eg, polycystic kidneys, glomerular or interstitial disease), renal vascular disease (eg, renal artery stenosis, fibromuscular dysplasia), endocrine disorders (eg, adrenocorticosteroid or mineralocorticoid excess, pheochromocytoma, hyperthyroidism or hypothyroidism, growth hormone excess, hyperparathyroidism), coarctation of the aorta, or oral contraceptive use. About 20-25% of women
with chronic hypertension develop preeclampsia during pregnancy.

Chronic hypertension occurs in up to 22% of women of childbearing age, with the prevalence varying according to age, race, and body mass index (BMI). Population-based data indicate that approximately 1% of pregnancies are complicated by chronic hypertension, 5-6% by gestational hypertension (without proteinuria), and 1-2% by preeclampsia.

**PREECLAMPSIA**

Although the exact pathophysiologic mechanism is not clearly understood, preeclampsia is primarily a disorder of placental dysfunction leading to a syndrome of endothelial dysfunction with associated vasospasm. In most cases, pathology evaluation demonstrates evidence of placental insufficiency with associated abnormalities such as diffuse placental thrombosis, an inflammatory placental decidual vasculopathy, or abnormal trophoblastic invasion of the endometrium. These findings support abnormal placental development or placental damage from diffuse microthrombosis as being central to the development of this disorder.

The hypertension occurring in preeclampsia is due primarily to vasospasm, with arterial constriction and relatively reduced intravascular volume compared with that of a normal pregnancy. The vasculature of normal pregnant women typically demonstrates decreased responsiveness to vasoactive peptides such as angiotensin-II and epinephrine.

In contrast, women who develop preeclampsia typically show a hyperresponsiveness to these hormones, an alteration that may be seen even before the hypertension and other manifestations of preeclampsia become apparent. In addition, blood pressures in preeclampsia are labile, and the normal circadian blood pressure rhythms may be blunted or reversed. One study found increased arterial stiffness in women with preeclampsia, as well as in those with gestational hypertension, compared with normotensive controls; treatment with alpha methyl/dopa significantly improved the vascular stiffness in preeclampsia but did not normalize it.

**RISK FACTORS**

Preeclampsia is more common at the extremes of maternal age (< 18 y or >35 y). The increased prevalence of chronic hypertension and other comorbid medical illnesses in women older than 35 years may explain the increased frequency of preeclampsia among older gravids. In addition, black women have higher rates of preeclampsia complicating their pregnancies compared with other racial groups, mainly because they have a greater prevalence of underlying chronic hypertension.

Among women aged 30-39 years, chronic hypertension is present in 22.3% of black 4.6% of non-Hispanic white, and 6.2% of Mexican Americans.

Women who develop preeclampsia during pregnancy have an increased risk of recurrent preeclampsia during subsequent pregnancies. The overall risk is about 18%. The risk is higher (50%) in women who develop severe early preeclampsia (ie, before 27 weeks’ gestation). These women are also at increased risk for cardiovascular disease later in life.

**Maternal risk factors for preeclampsia**

- First pregnancy
- New partner/paternity
- Age younger than 18 years or older than 35 years
- History of preeclampsia
- Family history of preeclampsia in a first-degree relative
- Black race
- Obesity (BMI ≥30)
- Interpregnancy interval less than 2 years or longer than 10 years
- Chronic hypertension, especially when secondary to such disorders as hypercortisolism, hyperaldosteronism, pheochromocytoma, or renal artery stenosis
- Preexisting diabetes (type 1 or type 2), especially with microvascular disease
- Renal disease
- Systemic lupus erythematosus
- Thrombophilia
- History of migraine
- Use of selective serotonin uptake inhibitor antidepressants (SSRIs) beyond the first trimester

**Placental/fetal risk factors for preeclampsia**

- Multiple gestations
- Hydrops fetalis
- Gestational trophoblastic disease
- Triploidy

**GESTATIONAL HYPERTENSION**

Gestational hypertension refers to hypertension with onset in the latter part of pregnancy (>20 weeks’ gestation) without any other features of preeclampsia, and followed by normalization of the blood pressure postpartum. Of women who initially present with apparent gestational hypertension, about one third develops the syndrome of preeclampsia. As such, these patients should be observed carefully for this progression. The pathophysiology of gestational hypertension is unknown, but in the absence of features of preeclampsia, the maternal and fetal outcomes are usually normal. Gestational hypertension may be a harbinger of chronic hypertension later in life.
Hypertensive disorders in pregnancy are among the leading causes of maternal mortality, along with thromboembolism, hemorrhage and nonobstetric injuries. Furthermore, hypertension before pregnancy or during early pregnancy is associated with a twofold increased risk of gestational diabetes mellitus.

Although maternal diastolic blood pressure (DBP) greater than 110 mm Hg is associated with an increased risk for placental abruption and fetal growth restriction, superimposed preeclamptic disorders cause most of the morbidity due to chronic hypertension during pregnancy.

**DIAGNOSIS**

Determining whether elevated blood pressure identified during pregnancy is due to chronic hypertension or to preeclampsia is sometimes a challenge, especially if no recorded blood pressures from the first half of the gestation are available. Clinical characteristics obtained via history, physical examination, and certain laboratory investigations may be used to help clarify the diagnosis. Fetal well-being must also be considered with the workup of the mother.

Preeclampsia is rare before the third trimester, and the diagnosis of severe hypertension or preeclampsia in the first or early second trimester necessitates exclusion of gestational trophoblastic disease and/or molar pregnancy. Mild lower extremity edema is common in normal pregnancy, but rapidly increasing or nondependent edema may be a signal of developing preeclampsia. However, edema is no longer included among the criteria for the diagnosis of preeclampsia.

New seizures in pregnancy suggest preeclampsia-eclampsia, but primary neurologic disorders must be excluded.

Hyperaldosteronism and hypercortisolism are difficult to diagnose during pregnancy due to the high levels of progesterone and the normal increase in endogenous cortisol output.

**INVESTIGATIONS**

Include a CBC count, cases in which an incidental platelet count is less than 150,000/µL, 75% are secondary to dilutional thrombocytopenia of pregnancy, 24% are due to preeclampsia, and about 1% of cases are due to other platelet disorders not related to pregnancy. Platelet counts less than 100,000/µL suggest preeclampsia or immune thrombocytopenic purpura (ITP). Examination of the peripheral blood smear for evidence of microangiopathic hemolysis and thrombocytopenia may reveal the presence of red blood cell (RBC) fragments. In this setting, the diagnoses of hemolytic-uremic syndrome (HUS), thrombotic thrombocytopenic purpura (TTP), and HELLP syndrome (hemolysis, elevated liver enzymes, and low platelet count) should also be considered.

Hemoglobin levels greater than 13 g/dL suggest the presence of hemococoncentration. Low levels may be due to microangiopathic hemolysis or iron deficiency. Prothrombin time (PT) and activated partial prothrombin time (aPTT) results may be abnormal in consumptive coagulopathy and disseminated intravascular coagulopathy (DIC) complicating severe preeclampsia. However, checking the PT/INR/aPTT is not necessary in the absence of abnormal liver transaminases or thrombocytopenia.

Abnormal values of lactate dehydrogenase (LDH), bilirubin, haptoglobin, fibrinogen, and D-dimers may confirm the presence of hemolysis and DIC, along with coagulation testing. It is unnecessary to check levels of LDH, bilirubin, haptoglobin, fibrinogen, and D-dimers, unless the PT/INR/aPTT results are abnormal, thrombocytopenia is present, or the hemoglobin level is dropping.

**LABORATORY TESTS DURING PREGNANCY**

- Urinalysis: If >1+ proteinuria on dipstick arrange for 24 hour collection to quantify proteinuria
- Pre-eclampsia (“PET”) bloods:
  - FBC: thrombocytopenia &/or haemoconcentration suggest severe pre-eclampsia
  - U&Es: urea and creatinine are reduced in uncomplicated pregnancy, “normal” values may indicate renal impairment
  - LFTs: transaminase concentrations increase in the HELLP syndrome (a variant of pre-eclampsia)
  - Urate: levels are gestation related but rise in pre-eclampsia, largely due to reduced renal excretion
  - Clotting screen: when the platelet count <100×10⁹/l
- Blood film: microangiopathic haemolytic anaemia may occur in severe pre-eclampsia

FBC, full blood count; HELLP, haemolysis, elevated liver enzymes, low platelets; LFT, liver function test; U&E, urea and electrolytes.

**DOPPLER ASSESSMENT OF UTERINE ARTERIES**

Pulsed wave and colour flow Doppler ultrasound examination of the uterine arteries can demonstrate the increased placental vascular resistance which results from complete or partial failure of trophoblast invasion of the spiral arteries. In addition to a measurement of the resistance index, the presence of an abnormal uterine artery waveform is also sought (fig 11). Uterine artery “Dopplers” are offered to high risk women at between 20–24 weeks of pregnancy and have useful predictive power. A woman with normal uterine Doppler assessment at 20–24 weeks can be considered to be at low risk, whereas those with abnormal Dopplers have approximately a 20% chance of developing pre-eclampsia and require increased vigilance.

**MRI**

An MRI may be performed to evaluate for abnormalities of the cerebral cortex (ie, edema, infarction, hemorrhage) in
preeclamptic women with severe visual disturbance, seizures, or altered mental status. An MRI is more sensitive than a CT scan for detecting cerebral cortical abnormalities but less useful in detecting cerebral hemorrhage.

**USG**

Ultrasonography or CT scanning of the liver may be used to evaluate for subcapsular hemorrhage or infarction in the setting of persistent severe rt. hypochondriac pain or markedly elevated hepatic transaminases.

**Fetal USG**

Women with both chronic hypertension and pre-eclampsia are at risk of IUGR. Such women are offered regular fetal ultrasound scans to assess fetal growth, liquor volume, and umbilical artery blood flow. When pre-eclampsia is severe, and there is a significant risk of delivery before 34 weeks gestation, intramuscular steroids (dexamethasone or betamethasone) are given to the mother to enhance fetal lung maturity in anticipation of a premature delivery.

**HISTOLOGIC FINDINGS**

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

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

The kidneys may reveal glomerular endotheliosis that is associated with proteinuria greater than 300 mg in 24 hours or, more rarely, acute tubular necrosis (ATN) or cortical necrosis.

**MANAGEMENT**

**Pre-pregnancy counselling**

Up to 50% of pregnant women not attending ANC clinic, pre-pregnancy counselling may not be feasible. In women with chronic hypertension, assessment before conception permits exclusion of secondary causes of hypertension (for example, renal/endocrine), evaluation of their hypertensive control to ensure it is optimal, discussion of the increased risks of preeclampsia, and education about any drug alterations which would need to be made in the first trimester should they become pregnant. The majority of women with controlled chronic hypertension will, under close supervision and appropriate management, have a successful outcome. Poorly controlled hypertension in the first trimester will significantly increase maternal and fetal morbidity and mortality. It must be stressed that none of the many antihypertensive agents used in routine practice have been shown to be teratogenic and women can safely conceive while taking medication. However, angiotensin converting enzyme (ACE) inhibitors and angiotensin receptor blockers (ARBs) need to be withdrawn in the first trimester since they are fetotoxic. Women who have experienced poor obstetric outcomes in previous pregnancies because of severe pre-eclampsia, or those at particular risk, need to be counselled about the condition and offered prophylactic treatment with low dose aspirin. Since pre-eclampsia involves endothelial dysfunction and oxidative stress, there is interest in supplementation, with antioxidant vitamins C and E, in the second trimester. A preliminary trial of antioxidant vitamins, in high risk women, has reported improvements in biochemical markers of endothelial activation together with a reduction in pre-eclampsia and a large, nationwide, randomised trials are under evaluation.

**Bedrest and Hospitalization**

Women with worsening hypertension during pregnancy often are placed on bed rest or restricted activity, although no scientific evidence demonstrates that this is beneficial in prolonging gestation or reducing maternal or fetal morbidity/mortality.

Women with hypertension and suspected preeclampsia are typically admitted to a hospital for close observation and investigation. Those with established preeclampsia must be observed very closely, either in hospital or in a comprehensive home monitoring program under the care of an obstetrician.

**Antenatal care**

Blood pressure assessment and the search for proteinuria form the cornerstone of antenatal screening of all pregnant women for pre-eclampsia. If “white coat” hypertension is suspected, ambulatory monitoring can be helpful. Those women who have been defined as at increased risk of pre-eclampsia are monitored more closely, often in a specialised obstetric clinic. Doppler ultrasound evaluations of the uterine arteries around the time of the fetal anomaly scan at 20–22 weeks and blood analysis. Rising blood pressure, deranged blood results, and/or the development of significant proteinuria requires further evaluation. < 1+ proteinuria on dip stick needs to be formally quantified with a 24 hour urine collection or protein/creatinine ratios. The onset of significant proteinuria, in the absence of renal disease, is among the best indicators of superimposed pre-eclampsia. Depending on the severity of maternal symptoms and clinical findings and on the fetal growth pattern, a woman may be referred to ANC clinic, or be admitted. Many women are initially asymptomatic, or present with non-specific signs of malaise. However, headache, visual disturbance, or abdominal pain are well recognised signs of severe pre-eclampsia.
When to Treat Hypertension during Pregnancy

Significant hypertension must be treated in its own right, regardless of the assumed underlying pathology, largely to reduce the risk of maternal intracranial haemorrhage. The level at which antihypertensive treatment is initiated for non-severe hypertension remains controversial, depending on whether treatment is focused on maternal or fetal wellbeing. Most physicians commence antihypertensive medication when the systolic blood pressure >140-170 mm Hg or diastolic pressure >90-110 mm Hg. Treatment is mandatory for severe hypertension when the blood pressure is ≥170/110 mm Hg. Once treatment is started, targeted blood pressure is also controversial, but many practitioners would treat to keep the mean arterial pressure <125 mmHg—for example, a blood pressure 150/100 mm Hg. Rapidly blood pressure control may lead to placental hypoperfusion, as placental blood flow is not autoregulated, and this will compromise the fetus. Unfortunately there is no evidence that pharmacological treatment of chronic or gestational hypertension protects against the development of pre-eclampsia. Changes in diet or bed rest have not been shown to provide maternal or fetal benefit.18-20

Drug Treatment

All antihypertensive drugs have either been shown to cross the placenta and reach the fetal circulation. However, none of the antihypertensive agents in routine use have been documented to be teratogenic, although ACE inhibitors and ARBs are fetotoxic. The objective of treating hypertension in pregnancy is to protect the woman from dangerously high blood pressure and to permit continuation of the pregnancy, fetal growth and maturation.

MILD TO MODERATE HYPERTENSION

The evidence base for treatment of mild to moderate chronic hypertension in pregnancy resides in maternal benefit rather than clear evidence of an enhanced perinatal outcome for the baby.4 Some women with treated chronic hypertension are able to stop their medication in the first half of pregnancy, because of the physiological fall in blood pressure during this period. However, this is usually temporary, and women are monitored and treatment resumed as soon as necessary.

Although the primary risk of chronic hypertension in pregnancy is development of superimposed preeclampsia, no evidence suggests that pharmacologic treatment of mild hypertension reduces the incidence of preeclampsia in this population.

In normal pregnancy, women’s mean arterial pressure drops 10-15 mm Hg over the first half of pregnancy. Most women with mild chronic hypertension (ie, SBP 140-160 mm Hg, DBP 90-100 mm Hg) have a similar decrease in blood pressures and may not require any medication during this period. Conversely, DBP greater than 110 mm Hg has been associated with an increased risk of placental abruption and intrauterine growth restriction, and SBP greater than 160 mm Hg increases the risk of maternal intracerebral hemorrhage. Therefore, pregnant patients should be started on antihypertensive therapy if the SBP is greater than 160 mm Hg or the DBP is greater than 100-105 mmHg.

The goal of pharmacologic treatment should be a DBP of less than 100-105 mm Hg and an SBP less than 160 mm Hg. Women with preexisting end-organ damage from chronic hypertension should have a lower threshold for starting antihypertensive medication (ie, >139/89) and a lower target blood pressure (<140/90).

If a pregnant woman’s blood pressure is sustained greater than 160 mm Hg systolic and/or 110 mm Hg diastolic at any time, lowering the blood pressure quickly with rapid-acting agents is indicated for maternal safety. Anticonvulsant therapy may be undertaken in the setting of severe preeclampsia (primary prophylaxis) or in the setting of eclamptic seizures (secondary prophylaxis). The most effective agent is IV magnesium sulfate; phenytoin is an alternative, although less effective, therapy.

A healthy fetus depends on a healthy mother, so medications should be used when clear benefit to the mother exists. The US Food and Drug Administration (FDA) categorization for drug use during pregnancy is simplistic and sometimes misleading. To quote the FDA descriptions, any medication in class A through D may be used “when the potential benefit justifies the potential risk.”

Available data suggest that all studied agent’s are excreted into human breast milk, but most are excreted to a negligible degree. All antihypertensive medications are believed to be compatible with breastfeeding, but using medications with a well-established record is reasonable. Atenolol, as well as the other beta-blocking agent’s nadolol and metoprolol, appear to be concentrated in breast milk. Labetalol and propranolol do not share this property and are preferred agents if a beta-blocker is indicated.

Evidence-based guidelines from the American Association of Clinical Endocrinologists single out methyldopa or nifedipine as preferable antihypertensive medications in pregnancy, with magnesium sulfate for women with preeclampsia who are at high risk for seizures, but they recommend all major antihypertensive agents with the exception of angiotensin-converting enzyme (ACE) inhibitors and angiotensin II receptor blockers (ARBs).

ACE inhibitors should be avoided during pregnancy, as they are associated with fetal renal dysgenesis or death when used in the second and third trimesters, as well as with increased risk of cardiovascular and central nervous system malformations.
when used in the first trimester. Angiotensin II receptor antagonists/blockers are not used during pregnancy, because they have a mechanism of action that is similar to that of ACE inhibitors. Diuretics do not cause fetal malformations but are generally avoided in pregnancy, as they prevent the physiologic volume expansion seen in normal pregnancy. They may be used in states of volume-dependent hypertension, such as renal or cardiac disease.

**Drug treatment of severe hypertension**

The mortality and morbidity of women with severe hypertension (> 170/110 mm Hg), usually secondary to severe pre-eclampsia, remain considerable. Because of the circulating plasma volume contraction, women may be very sensitive to relatively small doses of antihypertensive agents (and diuretics), risking abrupt reductions in blood pressure. Good control of hypertension in severe pre-eclampsia does not halt the progression of the disease, only delivery can do this, but it can reduce the incidence of complications such as cerebral haemorrhage. Management of severe hypertension involves adequate blood pressure control, often using parenteral agents, and “expectant” management by trying to prolong the pregnancy without unduly risking the mother or fetus. Different units have their preferences for either parenteral hydralazine or labeltol, and some use oral nifedipine. Hydralazine should be given after a colloid challenge to reduce the reflex tachycardia, and abrupt hypotension, precipitated by vasodilatation of a volume contracted circulation. These women are high risk and should be managed in a high dependency unit setting. They are very sensitive to fluid overload and are at risk of developing non-cardiac pulmonary oedema; through capillary leak. Severe forms of pre-eclampsia require admission to intensive care, frequently for respiratory failure or the development of a severe systemic inflammatory response syndrome (SIRS). Seizure prophylaxis, with intravenous magnesium sulfate, may be required in these cases.

**Antihypertensive Drugs to Avoid in Pregnancy**

Both ACE inhibitors and ARBs are fetotoxic but there are no data to support teratogenicity. Women can thus be reassured that conceiving while taking such agents, particularly ACE inhibitors where the data are strongest, appears to be safe. However, all women of childbearing age treated with these drugs must be informed of the need for drug discontinuation within the first trimester should they become pregnant. The greatest risk to the fetus appears to be associated with exposure in the third trimester, but the earlier the discontinuation the better hence the need for patient education. A variety of malformations and adverse events have been reported for both ACE inhibitors and ARBs including:

- oligohydramnious
- IUGR
- joint contractures
- pulmonary hypoplasia
- hypocalvaria (incomplete ossification of the fetal skull)
- fetal renal tubular dysplasia and neonatal renal failure.

**Antihypertensive Treatment Post Partum And During Breastfeeding**

Post partum hypertension is common. Blood pressure typically rises after delivery over the first five days. Thus women who experienced hypertension during pregnancy may be normotensive immediately after the birth, but then become hypertensive again in the first postnatal week. The need to obtain hypertensive control may delay discharge. Methyldopa should be avoided post partum because of the risk of postnatal depression. Our first line agent is atenolol, plus nifedipine or an ACE inhibitor if another agent is required. Women with gestational hypertension, or pre-eclampsia, are usually able to stop all antihypertensives within six weeks post partum. Those with chronic hypertension can resume their pre-pregnancy drugs. Diuretics, however, are usually avoided if the woman wishes to breast feed because of increased thirst. Proteinuria in pre-eclamptic women will usually remit by three months post partum, in the absence of any underlying renal abnormality. Persistent proteinuria requires further renal investigation. An accurate estimation of drug passage into breast milk is difficult to individualise since it is influenced by many factors such as the lipid solubility of the drug, protein binding, ionisation, molecular weight, and the constituency of milk itself (fat, protein versus water content). However, most antihypertensive agents used in routine practice are compatible with breastfeeding, but safety data for doxazosin, amiodipine, and ARBs are lacking.

**Recurrence of Hypertension in Subsequent Pregnancy**

Women who experience hypertension in a first pregnancy are at increased risk in a subsequent pregnancy. Certain factors influence this risk. The earlier the onset of hypertension in the first pregnancy, the greater the risk of recurrence and the type of hypertensive disorder influences recurrence. One study reported a recurrence risk of 19% for gestational hypertension, 32% for pre-eclampsia, and 46% for pre-eclampsia superimposed on pre-existing chronic hypertension. In addition, severe isolated IUGR is also a risk factor for developing hypertension in a subsequent pregnancy.

**Pregnancy Induced Hypertensive & CVS complication**

Women who develop gestational hypertension or pre-eclampsia are at increased risk of hypertension and stroke in later adult life. Furthermore, there is evidence of an increased risk of ischaemic heart disease (IHD) in women who have experienced pre-eclampsia or isolated IUGR, together with increased death rates from IHD. At first an
association between pre-eclampsia and an increased risk of IHD in later adult life may appear rather tenuous. However, both conditions are associated with dyslipidaemia, insulin resistance, and endothelial dysfunction. It is of interest that the lipid abnormalities which occur in pre-eclampsia (raised low density lipoprotein (LDL), VLDL, free fatty acid, and triglyceride values) pre-date the clinical appearance of the condition and endothelial dysfunction continues to be impaired post partum.

Although there are methodological concerns with some of the data in this area, these apparent late sequelae may have important public health implications given the relative frequency of pregnancy induced hypertension and may, in future, dictate screening for cardiovascular disease in previously affected women. At the very least, it would seem prudent for such women to have an annual blood pressure measurement. Interestingly, women who go through a pregnancy without developing hypertension are at a reduced risk of becoming hypertensive in later life, when compared to nulliparous women. Pregnancy may offer a window into the future cardiovascular health of women, which is unavailable in men.

**SUMMARY**

- Main categories of hypertensive disease in pregnancy: chronic, gestational, pre-eclampsia
- Pre-eclampsia remains an important cause of maternal death in the UK
- No antihypertensive has been shown to be teratogenic, but angiotensin converting enzyme (ACE) inhibitors and angiotensin receptor blockers (ARBs) are fetotoxic
- First line antihypertensive during pregnancy: methyldopa
- First line antihypertensive post partum: atenolol
- Pregnancy induced hypertension increases the risk of cerebrovascular disease and ischaemic heart disease in later life

**REFERENCES**