Hypertensive disorders in pregnancy are the third leading cause of maternal mortality, after thromboembolism and nonobstetric injuries. Preeclampsia is a pregnancy-specific syndrome. It occurs in approximately 5% of all pregnancies, 10% of first pregnancies, and 20-25% of women with a history of chronic hypertension. There are only isolated documentations of hypertension in pregnancy in India. The article by Jai Prakash et al is a good attempt to study the prevalence and causes of hypertension and its impact on fetal and maternal outcome during pregnancy. In the present study, preeclampsia was the most common cause of hypertension during pregnancy.

Maternal diastolic blood pressure (DBP) greater than 110 mm Hg is associated with an increased risk for abruptio placentae, intrauterine fetal growth retardation, premature delivery, and intrauterine fetal death. The authors also noted similar findings. Only 33.33% fetuses had normal birth weight. Superimposed preeclamptic disorders cause most of the morbidity due to chronic hypertension during pregnancy. Severe maternal complications include eclamptic seizures, intracerebral hemorrhage, pulmonary edema due to capillary leak or myocardial dysfunction, acute renal failure due to vasospasm, proteinuria greater than 4-5 g/d, hepatic swelling with or without liver dysfunction, and disseminated intravascular coagulation and/or consumptive coagulopathy (This is rare, HELLP syndrome is more common). Consumptive coagulopathy usually is associated with placental abruption and is uncommon as a primary manifestation of preeclampsia. Ophthalmoscopy generally reveals a normal fundus in preeclampsia. Hypertensive retinopathy more often than not suggests chronic hypertension.

Gestational age is an important distinguishing feature. Hypertension prior to 20 weeks’ gestation almost always is due to chronic hypertension; preeclampsia is rare prior to the third trimester. New-onset or worsening hypertension after 20 weeks’ gestation should lead to a careful evaluation for manifestations of preeclampsia. The diagnosis of severe hypertension or preeclampsia in the first or early second trimester necessitates exclusion of gestational trophoblastic disease and/or molar pregnancy. Women diagnosed with severe or early preeclampsia (in the second trimester or early third trimester) have a higher prevalence of thrombophilias, but no studies to date have demonstrated that administering anticoagulants in subsequent pregnancies decreases the risk of recurrent preeclampsia.

Maternal risk factors for preeclampsia are first pregnancy, new partner or paternity, age younger than 18 years or older than 35 years, past history of preeclampsia, family history of preeclampsia in a first-degree relative. 51% patients in the current study were primigravida and 43% were multigravida and the age of the patients ranged from 19 to 32 years. The increased prevalence of chronic hypertension in women older than 35 years can explain the increased frequency of preeclampsia among older gravidas. The increasing incidence of divorce and live-in relationships in our country may also become an important risk factor for preeclampsia in the future.

Blood pressure should be measured in the sitting position, with the cuff at the level of the heart. Inferior venacaval compression by the gravid uterus while the patient is supine can alter readings substantially, leading to an underestimation of the blood pressure. Blood pressures measured in the left lateral position similarly may yield falsely low values if the blood pressure is measured in the higher arm and the cuff is not maintained at heart level.

Certain clinical and investigative features are important. They determine the severity of the disease and ensuing complications. Maternal SBP > 160 mm Hg or DBP > 110 mm Hg denotes severe disease. Retinal vasospasm is a severe manifestation of maternal disease. Delivery should be considered in these situations. Severely impaired vision and retinal edema depicts retinal detachment. The condition resolves completely, but is an indication for delivery. Right upper quadrant abdominal tenderness stems from liver swelling and capsular stretch and also is an indication for delivery. Certain features that raise concern and warrant further evaluation are a sudden change in dependent edema, edema in nondependent areas such as the face and hands, or rapid weight gain suggests a pathologic process; and clonus which is a sign of neuromuscular irritability. It should be noted that brisk, or hyperactive, reflexes are common during pregnancy, but clonus is not.

Laboratory investigations may offer clues about the
underlying pathology. 75% of cases with a platelet count less than 150,000/µL 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. Counts less than 100,000/µL suggest preeclampsia. Hemoglobin levels greater than 13 g/dL suggest the presence of hemoconcentration. Low levels may be due to microangiopathic hemolysis. Serum creatinine usually is less than 0.8 mg/dL during pregnancy. Higher levels suggest intravascular volume contraction or renal involvement in preeclampsia. A serum uric acid level greater than 5 mg/dL is abnormal and is a sensitive, but nonspecific marker of tubular dysfunction in preeclampsia. The reference range for protein excretion in pregnancy is up to 300 mg/24 hours. Higher levels are abnormal and may reflect renal involvement in preeclampsia. Creatinine clearance increases approximately 50% during pregnancy, and levels less than 100 mL/min suggest renal dysfunction that is either chronic or due to preeclampsia.

Research has shown that women who were at risk for developing preeclampsia had an abnormal uterine blood flow. Among the 43 women with abnormal uterine blood flow, 10 went on to develop pre-eclampsia, 14 delivered underweight babies due to growth restriction in the womb and 19 had normal pregnancies. Second-trimester testing also showed that half of the women with abnormal uterine blood flow had elevated blood levels of asymmetric dimethylarginine, or ADMA, which impairs the ability of blood vessels to widen. This happens because ADMA inhibits the effects of nitric oxide, which helps keep blood vessels relaxed and blood pressure in check. Additional ultrasound testing in the second trimester showed that, compared with women who did not develop pre-eclampsia, those who did had more restricted blood flow in their arms. This suggested that the presence of blood vessel dysfunction occurs before pre-eclampsia has been diagnosed, and the more restricted the blood flow, the higher the blood levels of ADMA.

These findings suggest that it may be possible to predict pre-eclampsia by checking blood flow in the arms in addition to the uterus. It is also known that the amino acid L-arginine can help counter the effects of ADMA; it may be worth exploring whether L-arginine can help stave off pre-eclampsia in high-risk women.

**REFERENCES**
