In December, 2013 The American College of Obstetricians and Gynecologists issued a report summarizing the findings and recommendations of their Task Force on Hypertension in Pregnancy. With respect to preeclampsia several pertinent revisions to the diagnostic criteria were made including: proteinuria is no longer an absolute requirement for making the diagnosis; the terms mild and severe preeclampsia have been replaced, and the disease is now termed preeclampsia either with or without severe features; removal of fetal growth restriction as a defining criterion for severe preeclampsia.

Preeclampsia with severe features is the presence of new-onset hypertension and proteinuria accompanied by end-organ damage. Features may include severe elevation of blood pressure (>160/110 mmHg), evidence of central nervous system (CNS) dysfunction (headaches, blurred vision, seizures, coma), renal dysfunction (oliguria or creatinine >1.5 mg/dL), pulmonary edema, hepatocellular injury (serum alanine aminotransferase level more than twofold the upper limit of normal), hematologic dysfunction (platelet count <100,000/L or disseminated intravascular coagulation [DIC]). The *HELLP syndrome* (*h*emolysis, *e*levated *l*iver enzymes, *l*ow *p*latelets) is a special subtype of severe preeclampsia and is a major cause of morbidity and mortality in this disease. Platelet dysfunction and coagulation disorders further increase the risk of stroke.

TREATMENT PREECLAMPSIA

Preeclampsia resolves within a few weeks after delivery. For pregnant women with preeclampsia prior to 37 weeks of gestation, delivery reduces the mother's morbidity but exposes the fetus to the risk of premature birth. The management of preeclampsia is challenging because it requires the clinician to balance the health of the mother and fetus simultaneously. In general, prior to term, women with *mild* preeclampsia without severe features may be managed conservatively with limited physical activity, although bed rest is not recommended, close monitoring of blood pressure and renal function, and careful fetal surveillance. For women with preeclampsia with severe features, delivery is recommended unless the patient is eligible for expectant management in a tertiary hospital setting. Expectant management of preeclampsia with severe features remote from term affords some benefits for the fetus but significant risks for the mother.

The definitive treatment of preeclampsia is delivery of the fetus and placenta. For women with preeclampsia with severe features, aggressive management of blood pressures >160/110 mmHg reduces the risk of cerebrovascular accidents. IV labetalol or hydralazine is most commonly used to acutely manage severe hypertension in preeclampsia; labetalol is associated with fewer episodes of maternal hypotension. Oral nifedipine and labetalol are commonly used to manage hypertension in pregnancy. Elevated arterial pressure should be reduced slowly to avoid hypotension and a decrease in blood flow to the fetus. Angiotensin-converting enzyme (ACE) inhibitors as well as angiotensin-receptor blockers should be avoided in the second and third trimesters of pregnancy because of their adverse effects on fetal development.

Magnesium sulfate is the preferred agent for the prevention and treatment of eclamptic seizures. Large, randomized clinical trials have demonstrated the superiority of magnesium sulfate over phenytoin and diazepam in reducing the risk of seizure and, possibly, the risk of maternal death. Magnesium may prevent seizures by interacting with *N*-methyl-D-aspartate (NMDA) receptors in the CNS. Given the difficulty of predicting eclamptic seizures on the basis of disease severity, once the decision to proceed with delivery is made, most patients carrying a diagnosis of preeclampsia should be treated with magnesium sulfate. Women who have had preeclampsia appear to be at increased risk of cardiovascular and renal disease later in life.

CHRONIC ESSENTIAL HYPERTENSION

Pregnancy complicated by chronic essential hypertension is associated with intrauterine growth restriction and increased perinatal mortality. Pregnant women with chronic hypertension are at increased risk for superimposed preeclampsia and abruptio placentae. Women with chronic hypertension should have a thorough prepregnancy evaluation, both to identify remediable causes of hypertension and to ensure that the prescribed antihypertensive agents (e.g., ACE inhibitors, angiotensin-receptor blockers) are not associated with an adverse outcome of pregnancy. α-Methyldopa, labetalol, and nifedipine are the most commonly used medications for the treatment of chronic hypertension in pregnancy. The target blood pressure is in the range of 130–150 mmHg systolic and 80–100 mmHg diastolic. Should hypertension worsen during pregnancy, baseline evaluation of renal function (see below) is necessary to help differentiate the effects of chronic hypertension from those of superimposed preeclampsia. There are no convincing data that the treatment of mild chronic hypertension improves perinatal outcome.

GESTATIONAL HYPERTENSION

The development of elevated blood pressure during pregnancy or in the first 24 h post-partum in the absence of preexisting chronic hypertension or proteinuria is referred to as *gestational hypertension*. Mild gestational hypertension that does not progress to preeclampsia has not been associated with adverse pregnancy outcome or adverse long-term prognosis.

RENAL DISEASE

(See also Chaps. 333 and 341) Normal pregnancy is characterized by an increase in glomerular filtration rate and creatinine clearance. This increase occurs secondary to a rise in renal plasma flow and increased glomerular filtration pressures. Patients with underlying renal disease and hypertension may expect a worsening of hypertension during pregnancy. If superimposed preeclampsia develops, the additional endothelial injury results in a capillary leak syndrome that may make management challenging. In general, patients with underlying renal disease and hypertension benefit from aggressive management of blood pressure. Preconception counseling is also essential for these patients so that accurate risk assessment and medication changes can occur prior to pregnancy. In general, a prepregnancy serum creatinine level <133 μmol/L (<1.5 mg/dL) is associated with a favorable prognosis. When renal disease worsens during pregnancy, close collaboration between the internist and the maternal-fetal medicine specialist is essential so that decisions regarding delivery can be weighed to balance the sequelae of prematurity for the neonate versus long-term sequelae for the mother with respect to future renal function.

CARDIAC DISEASE

VALVULAR HEART DISEASE

(See also Chaps. 283–286) Valvular heart disease is the most common cardiac problem complicating pregnancy.

Mitral Stenosis This is the valvular disease most likely to cause death during pregnancy. The pregnancy-induced increase in blood volume, cardiac output, and tachycardia can increase the transmitral pressure gradient and cause pulmonary edema in women with mitral stenosis. Women with moderate to severe mitral stenosis who are planning pregnancy and have either symptomatic disease or pulmonary hypertension should undergo valvuloplasty prior to conception. Pregnancy associated with long-standing mitral stenosis may result in pulmonary hypertension. Sudden death has been reported when hypovolemia occurs. Careful control of heart rate, especially during labor and delivery, minimizes the impact of tachycardia and reduced ventricular filling times on cardiac function. Pregnant women with mitral stenosis are at increased risk for the development of atrial fibrillation and other tachyarrhythmias. Medical management of severe mitral stenosis and atrial fibrillation with digoxin and beta blockers is recommended. Balloon valvulotomy can be carried out during pregnancy. The immediate postpartum period is a time of particular concern secondary to rapid volume shifts. Careful monitoring of cardiac and fluid status should be observed.

Mitral Regurgitation and Aortic Regurgitation and Stenosis The pregnancy-induced decrease in systemic vascular resistance reduces