5.5.1. Gestational Hypertension

Synopsis

Gestational hypertension is the de novo development of hypertension after 20 weeks' gestation in the absence of new proteinuria or target organ damage (Table 20).[1] Gestational hypertension is associated with an increased risk of maternal and fetal/neonatal adverse events, and up to 30% of women with gestational hypertension ultimately develop preeclampsia.[2] Following delivery, individuals with gestational hypertension have an increased risk of future hypertension and CVD. Most RCTs examining BP targets for pregnant individuals with gestational hypertension have been small and of poor to moderate quality. The highest-quality randomized trial that included women with nonproteinuric hypertension examined tight versus less-tight DBP targets (85 mm Hg and 100 mm Hg, respectively) and demonstrated a reduction in severe maternal hypertension with tight DBP control.[3,4] There were no other significant differences in maternal, fetal, or neonatal complications or pregnancy loss between treatment groups. In post-hoc analyses, the development of severe hypertension was also associated with an increased risk of pregnancy loss, neonatal intensive care unit admission, preterm delivery, and low birthweight.[4] Current treatment recommendations by ACOG in the 2020 Practice Bulletin advocate that individuals with gestational hypertension who present with severe-range blood pressures, which is defined as persistent SBP ≥160 mm Hg or DBP ≥110 mm Hg, be managed with the same approach as those with preeclampsia and severe-range blood pressures, highlighting the overlap in risks associated with both of these HDP (Table 23).

5.5.2. Preeclampsia and Eclampsia, Including Preeclampsia Superimposed on Chronic Hypertension

Synopsis

Preeclampsia, a multiorgan system inflammatory syndrome, is an HDP characterized by hypertension, as well as proteinuria or target organ dysfunction (Table 24).[1] Preeclampsia also develops in 20% to 50% of individuals with chronic hypertension and is termed *superimposed preeclampsia* in that scenario, which often presents as an increase in baseline hypertension or proteinuria. In an individual with preeclampsia, the development of severe features or hemolysis, elevated liver enzymes, and low platelet count (HELLP) syndrome are associated with increased rates of maternal and fetal/neonatal morbidity and mortality. Eclampsia, the occurrence of convulsive seizures, is one of the most severe forms of preeclampsia. Both preeclampsia and eclampsia can occur before, during, or after delivery, and magnesium sulfate in addition to antihypertensive medications are the mainstay of treatment. Low-dose aspirin is the only routinely recommended intervention that has been demonstrated to reduce the risk of preeclampsia and its sequelae when taken from 12 weeks of gestation in pregnant people at moderate and greater risk.[2-4] Pravastatin has been investigated in small studies as a potential therapy for the treatment of preeclampsia, but larger prospective studies are needed to confirm safety and efficacy.[5] The measurement of the antiangiogenic markers soluble fms-like tyrosine kinase -1 (sFlt-1), placental growth factor (PlGF), and their ratio is emerging as a diagnostic test with high negative predictive value to rule out preeclampsia.[6-9] To date, the heterogeneity in prospective studies limits definitive conclusions about their clinical utility for diagnosing preeclampsia.

TABLE 24 Diagnostic Criteria for Preeclampsia

Blood pressure	SBP ≥140 mm Hg or DBP ≥90 mm Hg on 2 occasions at least 4 h apart after 20 wks of gestation in a woman with previously normal BP *or* SBP ≥160 mm Hg or DBP ≥110 mm Hg (severe hypertension can be confirmed within a short interval [min] to facilitate timely antihypertensive therapy).
AND	
Proteinuria	≥300 mg per 24-h urine collection (or this amount extrapolated from a timed collection) *or* Protein/creatinine ratio ≥0.3 *or* Dipstick reading of 2+ (used only if other quantitative methods are not available)
OR in the absence of proteinuria, new onset hypertension with the new onset of any of the following:	
Thrombocytopenia: Platelet count <100 × 10^9/L	
Renal insufficiency: Serum creatinine concentrations >1.1 mg/dL or a doubling of serum creatinine concentration in the absence of other renal disease	
Impaired liver function: Elevated blood concentration of liver transaminases to twice normal concentration	
Pulmonary edema	
New-onset headache unresponsive to medication and not accounted for by the alternative diagnoses or visual symptoms	

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BP indicates blood pressure; DBP, diastolic blood pressure; and SBP, systolic blood pressure.

5.5.3. Short- and Long-Term Follow-Up of Pregnancy-Associated Hypertension

Synopsis

BP measurement and medication titration in the early postpartum period should be individualized and patient centered. ACOG recommends a BP check for individuals with an HDP within 3 to 10 days of discharge,[1] and HBPM has been shown to improve BP ascertainment. When combined in a team-based approach, including medication self-management and telehealth, HBPM for postpartum individuals with a history of HDP has been associated with lower BP and improved measures of cardiac structure and function at 6 and 9 months' postpartum compared with usual care.[2,3] These remote strategies may also help compensate for racial disparities in office-centric follow-up strategies, although there is insufficient