dysglycemia, dyslipidemia), and this clustering has traditionally been referred to as the metabolic syndrome, which is associated with increased risk of CVD.[7] Metabolic syndrome, along with hypertension alone, is included in the AHA cardiovascular-kidney-metabolic (CKM) construct.[8,9] CKM syndrome includes both individuals at risk for CVD due to the presence of metabolic risk factors and/or CKD, and individuals with existing CVD that is potentially related to or complicates metabolic risk factors and/or CKD.[8,9] Metabolic syndrome has increased in recent years, with an estimated prevalence of 47% among U.S. adults.[10] Sex-specific risk factors for metabolic syndrome include gestational diabetes and hypertensive disorders of pregnancy (HDP).

As obesity is a major cause of hypertension, strategies that target the underlying pathophysiology of excess or dysfunctional adiposity should be considered in hypertension management, including intensive lifestyle intervention (Section 5.1, "Lifestyle and Psychosocial Approaches"), pharmacotherapies,[1-4,11] and bariatric surgery[12,13] for weight loss. Among lifestyle interventions, the efficacy and safety of time-restricted eating as a strategy to improve metabolic health and lower BP remain unclear.[14] While certain antihypertensive therapies have been suggested to adversely impact metabolic health (eg, thiazide-type diuretics, BB), outcome data do not demonstrate overt harm. Regardless of the weight loss strategy, weight regain is common and may lead to rebound worsening of BP.[11,12]

Recommendation-Specific Supportive Text

1. In a systematic review and meta-analysis of 6 RCTs of patients with excess weight and without diabetes, use of GLP-1 receptor agonists demonstrated significant reduction in BP, which was a prespecified secondary endpoint in the phase 3 STEP (Once-Weekly Semaglutide in Adults With Overweight or Obesity) trials.[13] In patients with overweight or obesity and without diabetes, the STEP 8 (Research Study to Investigate How Well Semaglutide Works Compared to liraglutide in People Living With Overweight or Obesity) trial demonstrated significant and similar reduction in SBP with semaglutide (−5.7 mm Hg [95% CI: −8.1 to −3.3 mm Hg]) and liraglutide (−2.9 mm Hg [95% CI: −5.3 to −0.5 mm Hg]); significantly greater reduction in DBP was achieved with semaglutide (−5.0 mm Hg [95% CI: −7.0 to −3.1 mm Hg]) compared with liraglutide (−0.5 mm Hg [95% CI: −2.3 to 1.3 mm Hg]).[3] In a prespecified substudy of the SURMOUNT-1 (Study of Tirzepatide in Participants With Obesity or Overweight) trial, 600 participants completed ambulatory BP monitoring with placebo-adjusted SBP change at 36 weeks of −8.0 mm Hg (95% CI: −10.6 to −5.4 mm Hg) for tirzepatide 15 mg, with similar changes in BP for 5- and 10-mg doses and with 70% of the change in BP mediated by change in weight.[5]
2. Bariatric surgery has demonstrated improvement in obesity-related risk factor levels, including BP. In a randomized single-center trial conducted in Brazil, 100 adults aged 18 to 65 years with a BMI 30.0 to 39.9 kg/m^2 were randomized to Roux-en-Y gastric bypass combined with antihypertensive therapy or antihypertensive therapy alone. At 5-year follow-up, there was greater reduction in number of antihypertensive medications, with 81% versus 14% achieving at least a 30% reduction in number of medications in the surgical compared with the medical therapy arm (primary endpoint: relative risk: 5.91 [95% CI: 2.58-13.52]). In addition, SBP was significantly lower in the surgical arm (124 mm Hg [95% CI: 119-128 mm Hg]) compared with medical therapy alone (131 mm Hg [95% CI: 126-136 mm Hg]).[6] Similar findings were observed for BP benefit in a prospective observational study of U.S. adults aged 18 to 72 years with a BMI >35.0 kg/m^2 in which 418 patients underwent Roux-en-Y gastric bypass and were compared with 738 patients who did not undergo surgery, demonstrating a significantly lower incidence in hypertension at 12 years follow-up.[15] In LABS-2 (Longitudinal Assessment of Bariatric Surgery-2), a prospective cohort study of adults aged ≥18 years from 10 hospitals in 6 U.S. cities who underwent Roux-en-Y gastric bypass, weight regain was frequent, with median rate of weight regain of 27% of the maximum weight loss at 5 years after reaching nadir weight.[12]

5.3.3. Chronic Coronary Disease

Synopsis

Adults with CCD and hypertension are at increased risk of death compared with adults with CCD who do not have hypertension.[1] Reducing SBP to <130 mm Hg can lower cardiovascular risk and mortality in adults with CCD and hypertension.[2-5] Although there are scarce data on the optimal treatment target for DBP, when SBP is <130 mm Hg, a DBP between 70 and 80 mm Hg is associated with reduced cardiovascular events without an increase in serious adverse events.[5,6] ACEi, ARB, and BB have been shown to reduce CVD events and all-cause death in adults with CCD and hypertension.[7] Conflicting evidence exists regarding the long-term use of BB therapy (>1 year) in adults with CCD (eg, post-MI or post-acute coronary syndrome [ACS]) and hypertension with preserved left ventricular ejection fraction.[5,8] If additional antihypertensive medications are needed to achieve BP control, CCB, thiazide-type diuretics, and/or MRA are