hypertension trials (31 043 participants with 275 098 assessments for OH), have together shown that more intensive BP treatment and active antihypertension treatment lower the risk for OH.[1–3] In SPRINT, there was an increase in self-reported syncope for intensively treated participants; however, the intensively treated group experienced no increase in falls or injurious falls but rather a nonstatistically significant decrease in injurious falls. This finding was the same for the intensively treated arm in the CRHCP (goal for both trials <120 mm Hg).[4,18]

2. The impact of OH on the CVD and mortality benefit from intensive BP control in the SPRINT trial found no difference in the risk reduction for CVD or all-cause mortality, regardless of OH status, and no evidence for harm among those with standing hypotension (SBP <110 mm Hg).[3] A similar post-hoc analysis of the NAILED (Nurse-Based Age-Independent Intervention to Limit Evolution of Disease) study population (n=814, 35% experiencing OH at least once) showed that intensification of BP control was not associated with an increased risk of cardiovascular events or death in this stroke/TIA population.[5] Systematic titration of antihypertensive treatment did not increase the prevalence of OH compared with usual care. Thus, there is emerging evidence that OH does not reduce the gains of intensive antihypertensive treatment.
3. Assessment for OH prior to initiation of treatment is equitable as this was part of the eligibility process for the most valid trials of BP lowering.[1–4,18] An assessment for OH in symptomatic patients, particularly after initiation of treatment or adding a new class of antihypertensive medication, is acceptable to detect unmasked autonomic system dysfunction or other acute or chronic conditions.[6,7]

6.2. Hypertensive Emergencies and Severe Hypertension in Nonpregnant and Nonstroke Patients

Recommendations for Hypertensive Emergencies and Severe Hypertension in Nonpregnant and Nonstroke Patients* References that support recommendations are summarized in the Evidence Table.

COR	LOE	Recommendations
1	B-NR	1. In adults with a hypertensive emergency (BP >180 and/or >120 mm Hg and evidence of acute target organ damage), admission to an intensive care unit is recommended for continuous monitoring of BP and target organ damage and for consideration of parenteral administration of appropriate therapy (Tables 26 and 27, Figure 9).[1–3]
1	C-LD	2. For adults with a hypertensive emergency related to a compelling condition (eg, acute aortic syndrome or acute aortic dissection), SBP should be reduced to <140 mm Hg for most conditions and to <120 mm Hg in aortic dissection during the first hour, while monitoring for other target organ dysfunction.[4–7]
1	C-LD	3. For adults with a hypertensive emergency but without a compelling condition, SBP should be reduced with oral or parenteral therapy by no more than 25% within the first hour; then, if stable, to <160/100 mm Hg within the next 2 to 6 hours; and then cautiously to 130 to 140 mm Hg during the next 24 to 48 hours to limit target organ injury.[2,8,9]
3: Harm	B-NR	4. For adults with severe hypertension (>180/120 mm Hg) who are hospitalized for noncardiac conditions without evidence of acute target organ damage, intermittent use of additional IV or oral antihypertensive medications are not recommended to acutely reduce BP.[8,10,11]

*Hypertensive emergencies in patients with acute ICH and acute ischemic stroke are discussed in Section 5.3.9 ("Cerebrovascular Disease") and in pregnant adults in Section 11.5 ("Hypertension and Pregnancy").

Synopsis

Hypertensive emergencies are defined as severe elevations in BP (>180/120 mm Hg) associated with evidence of acute target organ damage. Patients with hypertensive emergencies experience a high in-hospital mortality rate of 10% with a 1-year cardiovascular morbidity and mortality rate of 20% to 30%.[12,13] Common forms of acute hypertension-related target organ damage include acute HF/pulmonary edema, neurologic disorders (encephalopathy, ICH, acute ischemic stroke), and AKI, with aortic dissection the least common presentation.[12] Hypertensive emergencies demand immediate reduction of BP to prevent or limit further target organ damage. However, the rapid correction of BP in patients with longstanding hypertension to normal range may result in vital organ hypoperfusion due to loss of autoregulation.[6,7] In contrast, patients with severe hypertension without evidence of acute target organ damage (previously called hypertensive urgency) should not have aggressive BP lowering in the short-term or be given parenteral antihypertensive drug therapy. Reinstitution or intensification of oral antihypertensive medications, preferably in the outpatient setting, is recommended for these patients.

Recommendation-Specific Supportive Text

1. There is no RCT evidence that antihypertensive drugs reduce morbidity or mortality in patients with hypertensive emergencies.[3] There is also no high-quality RCT evidence to inform clinicians as to which first-line

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antihypertensive drug class provides more benefit than harm in hypertensive emergencies. This lack of high-quality RCT evidence is related to the small size of trials, lack of double-blinding design, lack of long-term follow-up, and failure to report outcomes. The therapeutic goal is to minimize target organ damage safely by rapid recognition of the problem and early initiation of appropriate antihypertensive treatment. To achieve rapid BP control and avoid large swings in BP, continuous infusion of short-acting titratable antihypertensive agents is often preferable in the intensive care setting. Antihypertensive agents available for the treatment of hypertensive emergencies are shown in Tables 26 and 27. Clinical trials suggested that IV nicardipine is more effective than labetalol in reaching short-term BP target, while clevidipine was shown to induce faster reduction in BP than nicardipine.[1,2] However, selection of an antihypertensive agent should be based on the drug's pharmacology, underlying mechanisms of hypertension, degree of progression of target organ damage, the desirable rate of BP decline, and the presence of comorbidities.

2. Other than for stroke/ICH (Section 5.3.9, "Cerebrovascular Disease"), there is no RCT evidence comparing different strategies to reduce BP acutely. Observational studies have indicated exponential dose-response relationship between SBP and an increasing risk of aortic dissection and death, with an HR of more than 2-fold for SBP >120 mm Hg.[4] However, BP measurement was not performed during hospitalization or IV drug treatment. Nevertheless, the current guideline endorses SBP target to <130 mm Hg in most patients with AD and <120 mm Hg in selected high-risk patients in the chronic setting.[14] Pheochromocytoma hypertensive crisis is also considered a medical emergency that requires prompt lowering of BP. However, the relationship between BP and mortality risk has not been characterized during adrenergic crisis. Despite potential cardiovascular complications of pheochromocytoma crisis, including a takotsubo-like cardiomyopathy,[5] pheochromocytoma should not be considered a compelling indication to reduce SBP immediately to less than <120 or 140 mm Hg in the first hour. In addition, clinical trials in patients without aortic dissection or pheochromocytoma showed increased risk of adverse kidney events associated with early intensive lowering of SBP to 110 to 139 mm Hg, particularly among patients with extremely high initial SBP of >220 mm Hg.[6,7] Thus, an attempt should be made to reduce SBP to <120 mm Hg in aortic dissection during the first hour and <140 mm Hg for most other conditions while monitoring for the presence and extent of other target organ function.
3. There is no RCT evidence for the treatment of other forms of hypertensive emergency without compelling indication. Small clinical trials in patients with acute HF and severe hypertension showed improvement in dyspnea when SBP was reduced by 15% (ie, 20 to 40 mm Hg) within 30 minutes without increased adverse events.[2,9] Thus, strategies to reduce BP should be more conservative to achieve 25% reduction in the first hour, followed by gradual further reduction within 24 to 48 hours.
4. Antihypertensive drug treatment should be used with extreme caution in hospitalized patients with asymptomatic severe hypertension because spontaneous falls in BP without any antihypertensive agents occur commonly, at a rate of 40% to 50%.[10] Observational studies have shown that initiation or intermittent use of additional IV or oral antihypertensive medications in patients hospitalized for noncardiac conditions are associated with increased risk of in-hospital mortality, AKI, and prolonged hospital stay.[8,11] Asymptomatic patients with severe hypertension can be treated with careful and frequent monitoring using standing medications and avoiding as-needed medications.

6.2.1. Medications for Hypertensive Emergencies

Treatment of hypertensive emergencies requires rapid recognition of the condition and knowledge of the unique treatment approaches appropriate for the causal condition or emergency consequences. A full discussion of these factors are in Sections 6.2 ("Hypertensive Emergencies and Severe Hypertension in Nonpregnant and Nonstroke Patients") and 5.3.9 ("Cerebrovascular Disease"). Tables 26 and 27 provide preferred agents for specific conditions.

6.3. Sexual Dysfunction

Synopsis

Sexual dysfunction frequently occurs in individuals with hypertension and has been more commonly reported by women than men.[1–6] Sexual dysfunction is defined as a person's inability to participate in sexual relationships as they would wish[7] and can be assessed in men and women using sex-specific validated tools that query emotional and physical symptoms in several domains, including thoughts and desires, arousal, frequency of sexual activity, pleasure and orgasm, and problems affecting sexual function.[8,9] The association between hypertension and sexual dysfunction may be due to shared mechanistic pathways of impaired vascular function and atherosclerosis, in addition to common risk factors of increasing age, hormonal shifts, diabetes, and depression.[10] Antihypertensive medications may also contribute as treated patients with hypertension are more likely to report sexual dysfunction than untreated ones.[11] Discussing sexual function with patients is essential as concerns about antihypertensive medications' negative impact on sexual function can lead to decreased adherence.[12] Diuretics and BBs, except nebivolol, are most commonly associated with erectile dysfunction in men,[10] and BBs are associated with a worsening of sexual function in women.[13,14] ARBs have the most favorable profile in men and women.[6,10] Phospho-

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Table 26. Intravenous Antihypertensive Drugs for Treatment of Hypertensive Emergencies

Class	Drug(s)	Usual Dose Range	Comments
CCB–dihydropyridines	Nicardipine	Initial 5 mg/h, increasing every 5 min by 2.5 mg/h to maximum 15 mg/h	Contraindicated in advanced aortic stenosis; no dose adjustment needed for persons aged ≥65 y. No negative inotropic or chronotropic effects.
	Clevidipine	Initial 1-2 mg/h, doubling every 90 s until BP approaches target, then increasing by less than double every 5-10 min; maximum dose 21 mg/h; maximum duration 72 h	Contraindicated in patients with soybean, soy product, egg, and egg product allergy and in patients with defective lipid metabolism (eg, pathological hyperlipidemia, lipoid nephrosis [minimal change disease] or acute pancreatitis). No negative inotropic or chronotropic effects. Decreased risk of reflex tachycardia.
Vasodilators–nitric-oxide dependent	Sodium nitroprusside	Initial 0.3-0.5 mcg/kg/min; increase in increments of 0.5 mcg/kg/min every 5 min to achieve BP target; maximum dose 10 mcg/kg/min; duration of treatment as short as possible	Due to potency, intra-arterial BP monitoring is recommended to prevent "overshoot." Lower dose required for older adults. Tachyphylaxis is common with extended use. No negative inotropic or chronotropic effects. Due to increased mortality risk, should be avoided in acute cerebrovascular disease unless other agents are not available. Use cautiously in pregnancy or older adults. Cyanide toxicity (increased risk in liver dysfunction and chronic kidney disease) and thiocyanate toxicity (increased risk in kidney dysfunction, sCr >3) may occur for infusion rates ≥3 mcg/kg/min and/or duration ≥3 d. Cyanide toxicity and thiocyanate toxicity may present similarly with metabolic acidosis, altered mental status, and cardiac arrhythmia. For either toxicity state, nitroprusside should be discontinued and sodium thiosulfate or cyanocobalamin should be administered.
	Nitroglycerin	Initial 5 mcg/min; increase in increments of 5 mcg/min every 3-5 min to a maximum rate of 200 mcg/min	Use only in patients with acute coronary syndrome and/or acute pulmonary edema. Do not use in volume-depleted patients. Tachyphylaxis is common with extended use.
Vasodilators–direct	Hydralazine	Initial 10 mg via slow IV infusion (maximum initial dose 20 mg); repeat every 4-6 h as needed. Adjust rate up to total cumulative dose of 200 mg/24 h	BP begins to decrease within 10-30 min, and the fall lasts 2-4 h. Hydralazine is an undesirable first-line agent for acute treatment in most patients due to unpredictability of response and prolonged duration of action.
Adrenergic blockers–beta-1 receptor selective antagonist	Esmolol	Loading dose 500-1000 mcg/kg/min over 1 min followed by a 50-mcg/kg/min infusion. For additional dosing, the bolus dose is repeated, and the infusion increased in 50-mcg/kg/min increments as needed to a maximum of 300 mcg/kg/min	Contraindicated in patients with concurrent beta-blocker therapy, bradycardia, or decompensated HF. Monitor for bradycardia. Higher doses may block beta-2 receptors and impact lung function in reactive airway and obstructive pulmonary disease.
Adrenergic blockers–combined alpha-1 and nonselective beta receptor antagonist	Labetalol	Initial 0.3- to 1.0-mg/kg dose (maximum 20 mg) slow IV injection at 10-min intervals or 0.4-1.0-mg/kg/h IV infusion up to 3 mg/kg/h. Adjust rate up to total cumulative dose of 300 mg/24 h	Contraindicated in reactive airway or obstructive pulmonary disease. Especially useful in hyperadrenergic syndromes. May worsen HF and should not be given in patients with second- or third-degree heart block or bradycardia.
Adrenergic blockers–nonselective alpha receptor antagonist	Phentolamine	IV bolus dose 5 mg. Additional bolus doses every 10 min as needed to lower BP to target. Adjust rate up to total cumulative dose of 50 mg/24 h	Used in hypertensive emergencies induced by catecholamine excess (pheochromocytoma, interactions between monoamine oxidase inhibitors and other drugs or food, cocaine toxicity, amphetamine overdose, or clonidine withdrawal).
Dopamine-1-receptor selective agonist	Fenoldopam	Initial 0.1-0.3 mcg/kg/min; may be increased in increments of 0.05-0.1 mcg/kg/min every 15 min until target BP is reached. Maximum infusion rate 1.6 mcg/kg/min	Contraindicated in patients at risk of increased intraocular pressure (glaucoma) or intracranial pressure and those with sulfite allergy.
ACE inhibitor	Enalaprilat	Initial 1.25 mg over a 5-min period. Doses can be increased up to 5 mg every 6 h as needed to achieve BP target. Adjust rate up to total cumulative dose of 50 mg/24 h	Contraindicated in pregnancy and should not be used in acute MI or bilateral renal artery stenosis. Mainly useful in hypertensive emergencies associated with high plasma renin activity. Poorly defined dose adjustments for kidney failure and may worsen kidney injury in those with CKD. Relatively slow onset of action (15 min) and unpredictability of BP response.

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BP indicates blood pressure; CCB, calcium channel blocker; CKD, chronic kidney disease; HF, heart failure; IV, intravenous; and MI, myocardial infarction.

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diesterase-5 inhibitors are a safe and effective therapy for erectile dysfunction and can be administered with antihypertensive medications. Caution should be exercised when sildenafil, tadalafil, vardenafil, and avanafil are coadministered with CYP3A4 inhibitors (such as diltiazem, verapamil; Section 5.2.6, "Medication Interactions") or ingested with grapefruit juice or alcohol, which may increase the risk of hypotension. These drugs should not be taken with nitrates due to the risk of severe hypotension.

6.4. Patients Scheduled for Surgical Procedures

Recommendations for Patients Scheduled for Surgical Procedures Referenced studies that support the recommendations are summarized in the Evidence Table.		
COR	**LOE**	**Recommendations**
1	B-NR	1. In patients with hypertension scheduled for major surgery who have been on BBs chronically, BBs should be continued throughout the perioperative period to assist with BP control.[1–5]
2a	C-EO	2. In patients with hypertension scheduled for elective major surgery, it is reasonable to continue most medications for hypertension throughout the perioperative period.
2b	B-R	3. In patients with hypertension scheduled for major surgery, discontinuation of ACEi or ARB preoperatively may be considered to prevent hypotension during surgery.[6–10]
Recommendations for Patients Scheduled for Surgical Procedures (Continued)		
COR	**LOE**	**Recommendations**
2b	C-LD	4. In patients scheduled for elective major surgery with SBP ≥180 mm Hg or DBP ≥110 mm Hg, deferring surgery may be considered especially in high-risk patients to minimize perioperative complications.[11–13]
3: Harm	B-NR	5. In patients with hypertension scheduled for surgery, abrupt preoperative discontinuation of BB or clonidine may result in rebound hypertension and is potentially harmful.[14]
3: Harm	B-R	6. For patients with hypertension scheduled for surgery, BB should not be started on the day of surgery in BB-naïve patients because of increased risk of postoperative mortality.[4,15,16]

Synopsis

Hypertension in the perioperative period increases the risk of cardiovascular and cerebrovascular events and bleeding.[17,18] As many as 25% of patients who undergo major noncardiac surgery and 80% of patients who have cardiac surgery experience perioperative hypertension.[18–20] In general, the level of risk is related to the severity of the hypertension. Uncontrolled hypertension is associated with increased perioperative and postoperative complications. Certain medications (eg, BB, clonidine) may be associated with rebound hypertension if discontinued abruptly. In addition to RCT results, several general strategies and

Table 27. Intravenous Antihypertensive Drugs for Treatment of Hypertensive Emergencies in Patients With Selected Comorbidities

Comorbidity	Preferred Drug(s)*	Comments
Acute aortic dissection	Esmolol, labetalol	Requires rapid lowering of SBP to ≤120 mm Hg. Beta blockade should precede vasodilator (eg, nicardipine or nitroprusside) administration, if needed for BP control or to prevent reflex tachycardia or inotropic effect; SBP ≤120 mm Hg should be achieved within 20 min.
Acute pulmonary edema	Clevidipine, nitroglycerin, nitroprusside	Beta blockers contraindicated.
Acute coronary syndromes	Esmolol†, labetalol, nicardipine, nitroglycerin†	Nitrates given in the presence of PDE-5 inhibitors may induce profound hypotension. Contraindications to beta blockers include moderate-to-severe LV failure with pulmonary edema, bradycardia (<60 beats/min), hypotension (SBP <100 mm Hg), poor peripheral perfusion, second- or third-degree heart block, and reactive airways disease.
Acute kidney injury	Clevidipine, fenoldopam, nicardipine	N/A
Eclampsia or preeclampsia	Hydralazine, labetalol, nicardipine, nifedipine	Requires rapid BP lowering. ACE inhibitors, ARB, renin inhibitors, and nitroprusside contraindicated.
Perioperative hypertension (BP ≥160/90 mm Hg or SBP elevation ≥20% of the preoperative value that persists for >15 min)	Clevidipine, esmolol, nicardipine, nitroglycerin	Intraoperative hypertension is most frequently seen during anesthesia induction and airway manipulation.
Acute sympathetic discharge or catecholamine excess states (eg, pheochromocytoma, postcarotid endarterectomy status)	Clevidipine, nicardipine, phentolamine	Requires rapid lowering of BP.
Acute ICH	Clevidipine, nicardipine, esmolol, labetalol, hydralazine	Section 5.3.9.1
Acute ischemic stroke	Clevidipine, nicardipine, esmolol, labetalol, hydralazine	Section 5.3.9.2

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*Agents are listed in alphabetical order, not in order of preference.

†Agent of choice for acute coronary syndromes.

ACE indicates angiotensin-converting enzyme; ARB, angiotensin receptor blocker; BP, blood pressure; ICH, intracerebral hemorrhage; LV, left ventricular; PDE-5, phosphodiesterase type-5; and SBP, systolic blood pressure.

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principles based on experience and observation are recommended for this section. In the management of patients with perioperative hypertension, it is important to assess other potential contributing factors, such as volume status, pain control, oxygenation, and bladder distention, when the use of pharmacological therapy to control BP is under consideration. For additional recommendations on perioperative hypertension management for noncardiac surgery, including the use of BB, ACEi, and ARB, please refer to the "2024 AHA/ACC/ACS/ASNC/HRS/SCA/SCCT/SCMR/SVM Guideline for Perioperative Cardiovascular Management for Noncardiac Surgery."[21]

Recommendation-Specific Supportive Text

1. If well tolerated, BBs should be continued in patients who are currently receiving them for GDMT indications (eg, recent MI, hypertension, arrhythmias). Multiple observational studies support the benefits of continuing BBs in patients who are undergoing surgery and who are taking these agents for GDMT.[1,3,4] Clinical judgment is useful in titrating BB during the perioperative period, focusing on continuing the medication through the hospital stay and at discharge unless clear contraindications arise.
2. In the absence of conclusive RCTs, the expert opinion of this writing committee is that control of BP to levels recommended by this guideline (BP <130/80 mm Hg) or other target levels specified for a particular individual is reasonable before undertaking major elective procedures in either the inpatient or outpatient setting. If the patient is unable to take oral medications, it is reasonable to use IV medications as necessary to control BP. Special consideration of parenteral therapy usually occurs for patients taking clonidine or BB because of the risk associated with stopping these medications acutely. Withdrawal syndromes, accompanied by sympathetic discharge and acute hypertension, can occur on cessation of these agents.[16] Caution is advised when continuing antihypertensive therapy in patients with low perioperative BPs, older adults (age ≥65 years),[22] and patients in whom the risk for perioperative hypotension is high.
3. Data on the potential risk and benefit of ACEi in the perioperative setting have been mostly limited to observational analyses and are controversial. Evidence from a large cohort study demonstrates that patients who stopped their ACEi or ARB 24 hours before noncardiac surgery were less likely to suffer the primary composite outcome (all-cause death, stroke, or myocardial injury) and intraoperative hypotension than were those continuing these medications until surgery.[10] However, the results from POISE-3 (Perioperative Ischemic Evaluation-3), which randomized 7490 patients undergoing noncardiac surgery with at least 1 high-risk factor, did not find a significant difference between patients randomized to a strategy that involved perioperative discontinuation of ACEi/ARB therapy and those with maintenance of such therapy during the operative and immediate perioperative periods.[9] Omitting RAASi before surgery has been shown to reduce intraoperative hypotension, whereas RCTs have failed to prove their continuation or implementation improves clinical outcomes.[6,7,23]
4. There is conflicting evidence for patients with DBP >110 mm Hg regarding recommending delay of surgery to provide for gradual reduction in DBP before proceeding with surgery.[24] In a systematic review and meta-analysis, preoperative hypertension was associated with a 35% increase in cardiovascular complications.[12] An increase in cardiovascular and cerebrovascular complications and renal failure has been reported in patients with DBP >110 mm Hg immediately before surgery.[25] In contrast, patients with DBP <110 mm Hg do not appear to be at significantly increased risk.[26] The relationship of systolic hypertension to surgical risk is less certain. During induction of anesthesia for surgery, a sympathetic reaction can result in a 20 to 30 mm Hg increase in BP among patients with normal BP.[25] Lability in BP appears more likely in patients with poorly controlled hypertension, whereas studies have observed that patients with controlled hypertension respond similarly to those who are normotensive.[26] An elevated BP on the day of surgery may represent a situational ("white-coat hypertension") response if there was evidence of controlled hypertension or normotension prior to surgery.[27] Therefore, referring to patients' baseline ambulatory BP is recommended to guide BP management. Without evidence for increased risk for perioperative complications in patients whose preoperative SBP/DBP is <180/110 mm Hg, there is little evidence to defer surgery, and the patient can be evaluated and BP can be controlled postoperatively or after discharge.[12]
5. Although few studies describe risks of withdrawing BB in the perioperative period, longstanding evidence from other settings suggests that abrupt withdrawal of long-term BB is harmful and should be avoided.[1,2,28] There are fewer data to describe whether short-term (1 to 2 days) perioperative use of BB, followed by rapid discontinuation, is harmful.[21,29] Importantly, abrupt discontinuation of clonidine can result in rebound hypertension associated with norepinephrine surge.[30]
6. This guideline recommends against starting a BB on the day of surgery in BB-naïve patients, particularly at high initial doses, in long-acting form, and if there are no plans for dose titration or monitoring for adverse events.[4,15,16] Evidence has been summarized in at least

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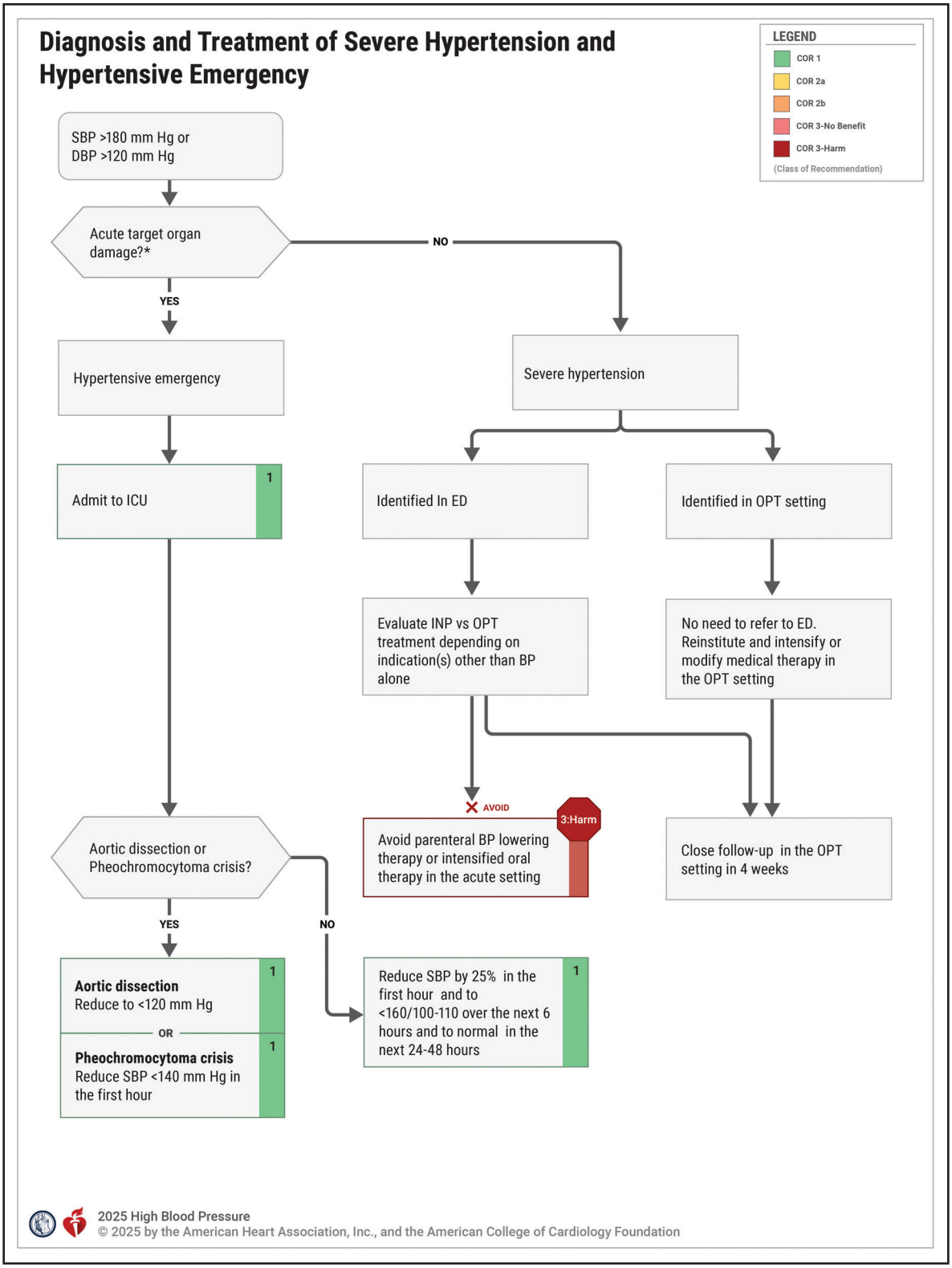


Figure 9. Diagnosis and Treatment of Severe Hypertension and Hypertensive Emergency.
*Defined as acute heart failure/pulmonary edema, neurologic disorders (posterior reversible encephalopathy syndrome, encephalopathy, retinal hemorrhage, papilledema, intracranial hemorrhage, acute ischemic stroke), acute decompensated heart failure, acute coronary syndrome, acute kidney injury, acute aortic syndrome (penetrating aortic ulcer, aortic dissection). DBP indicates diastolic blood pressure; ED, emergency department; ICU, intensive care unit; INP, inpatient; OPT, outpatient; and SBP, systolic blood pressure. For reinstitution, modification or intensification of medical therapy, refer to Sections 5.2.2. through 5.2.4. Modified with permission from Whelton et al.[15] Copyright 2018 American College of Cardiology Foundation and American Heart Association, Inc.

3 well-performed meta-analyses.[15,21,31] The first 2 of these meta-analyses called into question the strength of evidence for benefit of BB, while raising concerns about harms related to hypotension and stroke.[21,31] A more recent review confirmed earlier meta-analytic results while assessing that the certainty of evidence was low given biases and heterogeneity among the studies reviewed; each of these meta-analyses results were driven in part by the large POISE (Perioperative Ischemic Evaluation)[29] sample size.

7. EVIDENCE GAPS AND FUTURE DIRECTIONS

Since the 2017 high BP guideline was published, there have been numerous advancements in high BP management. However, key questions on the awareness of high BP and optimal management of high BP remain, and these knowledge gaps suggest areas for future research as described in this section.

Among adults with hypertension in the United States, most are not controlled to <130/80 mm Hg. More than 50% are unaware that they have hypertension. Population management strategies are just beginning to utilize the EHR and consider community-engaged strategies to identify those with undiagnosed or uncontrolled hypertension to focus resources. There is a need for research regarding effective screening methods and for more effective implementation strategies within and outside the health care system to control BP and reduce CVD risk.

Observational data demonstrate increased risk of CVD among younger adults with stage 1 hypertension, and elevated BP can be associated with evidence of target organ damage. There is a need to clarify those areas where current clinical trial evidence is sufficient on which to base our treatment decisions for younger adults, and where the needs for additional research are greatest. There is a lack of evidence to support BP targets for diastolic hypertension, which is more common in younger adults who have low short-term CVD risk but have a longer time horizon to consider for prevention. Research related to this issue should include detection of CVD endpoints organized outside academic centers, such as by pragmatic trial designs, to allow financial feasibility, use of surrogate endpoints such as left ventricular hypertrophy, which are not widely measured clinically, and evaluations for target organ damage performed at baseline and during longitudinal follow-up.

Accurate BP measurement remains a major challenge. Trials are needed that compare measurements taken by attended AOBP and unattended AOBP methods. Continued studies are needed in the realm of alternative methods for measurements, including accurate wearable and cuffless devices to provide near-continuous monitoring, HBPM, ABPM, and other novel approaches to measuring or estimating BP load. Additional studies comparing home and ambulatory BP measurements in estimating CVD risk are greatly needed to reduce treatment disparities due to lack of availability of ABPM to all populations. There is a need for studies utilizing HBPM combined with interventions to effectively achieve and maintain BP control utilizing health technology and to minimize nonadherence.

Further studies are needed at the intersection of BP, race/ethnicity, and SDOH, which includes those who are underinsured or uninsured to allow more precise predictions of needs and better focused prevention and management. To ensure optimal application of guidelines and evidence-based approaches that are effective and equitable for all groups, the BP treatment thresholds and treatment targets for different subpopulations need to be further clarified. With these efforts, there will be a need for additional data monitoring to evaluate new approaches and their effects on BP control and CVD outcomes across differences in sex/gender, race/ethnicity, socioeconomic status, education level, and access to medical care.

Further studies are needed to help us understand the role of genetic and epigenetic factors in BP. Using risk estimates that consider the influence of environmental and behavioral factors on genetic risk may add clarity to the areas of greatest need and potential benefit. Overall, further studies of environmental and lifestyle issues are needed, including nutrition, physical activity, and especially obesity and the role of emerging interventions for weight management in persons living with obesity and hypertension. We need additional studies of patients with white-coat hypertension to determine whether this condition carries additional long-term risk.

Using risk estimates that incorporate genetic risk, but that also consider the influence of environmental and behavioral factors on this risk, may add clarity to areas of greatest need and potential benefit.

In the realm of treatment approaches, there is prior evidence to support the contribution of sleep apnea to hypertension and resistant hypertension, but evidence of BP lowering from sleep apnea treatment is limited. This is an area ripe for research, particularly as sleep apnea treatment includes serial monitoring, which could incorporate BP tracking.

Early studies using stress management techniques have shown promise but need to be tested across a greater breadth of patients with adverse SDOH. We need additional trials comparing combinations of medications, dosed as separate agents and using SPCs to improve adherence and therefore effectiveness while monitoring for patient tolerance.

While pregnancy is an area where different concepts apply and decisions must account for optimal management of both the pregnant individual and the fetus, pregnancy planning and management have important implications for women with or at risk for high BP. Among women with preexisting hypertension or at risk for hypertension, safety and effectiveness of antihypertensive therapies should be considered, along with counseling on appropriate contraction options and recommendations for prophylactic aspirin therapy among those at greatest risk for acute onset or worsening of prior hypertension.

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