

Hypertensive Crisis

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"In this case, a new high score is not a good thing."

Objectives

1. State the pathophysiology and potential etiologies of a hypertensive crisis.
2. Describe the pharmacologic management of hypertensive crisis.
3. Describe components of patient education in the prevention of a hypertensive crisis.

Conflict of Interest Disclosure

- I have no conflicts of interest to disclose.



"You've got the blood pressure of a teenager – who lives on junk food, TV and the computer."

Hypertension and Cardiovascular Disease

- Cardiovascular disease (CVD) is the leading cause of death for both men and women in the **world**
- Approximately one in three of American adults have one or more types of CVD including hypertension (HTN), coronary artery disease (CAD), myocardial infarction (MI), heart failure (HF), and stroke
- People with HTN develop CVD 5.0 years earlier than their counterparts without HTN
- The elimination of hypertension could reduce CVD mortality by 30.4% in males and 38.0% in females.
- At age 60 years, the lifetime risk for CVD is 60.2% for those with HTN and 44.6% for those without HTN

American Heart Association. 2016 Heart Disease and Stroke Statistical Update, 2016.

ACC/AHA 2017 Hypertension Guideline

"While the updated guideline means that more people will be diagnosed with high BP, nearly all of these newly categorized patients can treat their hypertension with lifestyle changes instead of medication."

Whelton PK, Carey RM, Aronow WS, et al. 2017 Guideline for the Prevention, Detection, Evaluation and Management of High Blood Pressure in Adults. American College of Cardiology Foundation and American Heart Association. <http://professional.heart.org/hypertension>.

What is New?

- New data shows cardiovascular benefit from aggressive BP lowering
- New BP goal: less than 120/80 mm Hg (individualized)
- Focus is on a healthier lifestyle – diet and exercise have the greatest impact on BP
- Revised classification of blood pressure
- Revised management of blood pressure
- Use of atherosclerotic cardiovascular disease (ASCVD) risk calculator
- Accurate blood pressure measurement in the office and at home (self-monitoring)

2017 Classification of Blood Pressure

Blood Pressure Category	Systolic (mm Hg)		Diastolic (mm Hg)
Normal	Less than 120	and	less than 80
Elevated	120-129	and	less than 80
Hypertension Stage 1	130-139	or	80-89
Hypertension Stage 2	140 or higher	or	90 or higher

Essential (Primary) Hypertension

- “Silent killer” as it usually has no symptoms
- Most common risk factor for MI and stroke
- 34% of American adults have HTN
- Of these with HTN, about 76% are using anti-hypertensive medications, but only 54.4% have their condition controlled
- 90-95% of cases of HTN have no known cause, but risk factors have been identified

American Heart Association. 2018 Heart Disease and Stroke Statistical Update, 2018.

Nonmodifiable Risk Factors

• Race

African Americans develop HTN more often, earlier, and more severe than Caucasians

• Family history

The risk of HTN increases if parents, brothers, sisters, or children have the disease.

• Age

The risk of HTN increases with age. A higher percentage of men have HTN until age 45. From age 45-60, the percentage of men and women are similar. After age 60, a higher percentage of women have HTN.

American Heart Association. 2018 Heart Disease and Stroke Statistical Update, 2018.

Modifiable Risk Factors

• Obesity

BMI \geq 30 are more likely to develop HTN

• High-sodium diet

Excess sodium intake (e.g. >3000 mg/day) increases risk for HTN

• Lack of physical activity

Inactive lifestyle promotes weight gain and increases for HTN

• Stress/hostile attitudes/impatience

• Dyslipidemia and diabetes

• Excessive alcohol consumption

American Heart Association. 2018 Heart Disease and Stroke Statistical Update, 2018.

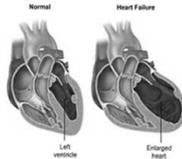
Pathophysiology of HTN

- Poorly understood but a variety of factors have been implicated
- Increased sympathetic nervous system activity
 - \uparrow HR
 - \uparrow peripheral resistance (vasoconstriction)
 - Vascular remodeling with narrowing and vasospasm of arteries
 - \uparrow insulin resistance contributing to endothelial dysfunction and \downarrow production of vasodilators
- Increased renin-angiotensin-aldosterone activity
 - Sodium and water retention
 - Vasoconstriction
 - Hypertrophy of myocardium associated with HTN
- Genetics

HTN and CAD

- HTN causes microscopic tears in the artery walls that then turn into scar tissue
- Scar tissue provides a place for plaque to develop
- Arteries slowly harden and narrow resulting in peripheral artery disease (PAD) and CAD
- As one ages, arteries harden and become less elastic
- Ongoing HTN accelerates this process
- Blood flow impaired to major organs leading to risk of blood clots, CAD, stroke, and renal damage

HTN Complications



- Left ventricular hypertrophy with ↑risk for heart failure, ventricular arrhythmias, MI, and death
- Most common and important risk factor for the development of stroke and intracerebral hemorrhage
- Chronic and end-stage kidney disease
- Retinopathy
- **Marked elevations in BP can cause acute, life-threatening emergencies**

Definitions

- **Resistant HTN:** BP greater than goal despite the concurrent use of 3 antihypertensive agents of different classes, including a diuretic
- **Hypertensive urgency:** acute increase in BP in the absence of symptoms suggesting acute organ damage
- **Hypertensive emergency:** acute increase in BP associated with severe, potentially life-threatening target organ damage
- “Acute increase in BP”:
 - systolic BP > 180 mm Hg
 - diastolic BP >120 mm Hg

Taylor, DA. Hypertensive crisis: a review of the pathophysiology and treatment. Crit Care Clin N Am. 2015;27:439-447.

Hypertensive Urgency

- **Acute increase in BP in the absence of symptoms suggesting acute organ damage:**
- Headache
- Shortness of breath
- Epistaxis
- Usually noncompliant with prescribed medications or have not followed up and have not had their medications dosages titrated
- Optimal treatment is close outpatient follow up; American College of Emergency Physicians (2013) discourages initiation of BP medications in the ED for asymptomatic patients with HTN

Wolf SJ, Lo B, Shih RD, et al. Clinical policy: critical issues in the evaluation and management of adult patients in the emergency department with asymptomatic blood pressure. Ann Emerg Med 2013;62:59-68.

Hypertensive Emergency

- **Acute increase in BP associated with severe, potentially life-threatening target organ damage:**
- Myocardial infarction/ischemia
- Acute left ventricular failure with pulmonary edema
- Acute aortic dissection
- Encephalopathy
- Retinopathy
- Ischemic/hemorrhagic stroke
- Acute kidney injury

Hypertensive Emergency

- “Acute increase in BP” varies with the clinical scenario
- Associated with significant short-term and long-term morbidity and mortality
- 5 year mortality close to 100%
- Approximately 1-2% of patients with HTN will develop an HTN emergency over their lifetime
- Hospitalization, preferably in an ICU required for prompt (minutes or a few hours) BP management
- IV antihypertensive medications

Deshmukh A, Kumar G, Kumar N, et al. Effect of the Joint National Committee VII Report on hospitalizations for hypertensive emergencies in the United States. Am J Cardiol 2011;108(9):1271-82.

Risk Factors

- Low socioeconomic status
- Poor access to health care
- Non-adherence to prescribed anti-hypertensive medications
- Substance (particularly cocaine) or alcohol use disorders
- Oral contraceptive use
- Cigarette smoking

Deshmukh A, Kumar G, Kumar N, et al. Effect of the Joint National Committee VII Report on hospitalizations for hypertensive emergencies in the United States. Am J Cardiol 2011;108(9): 1277-82.

Pathophysiology of Hypertensive Emergency

- Initial event is an abrupt rise in BP from an unknown stimulus followed by compensatory mechanisms from the vascular endothelium
- Endothelium release nitric oxide (vasodilator)
- Arterioles sense the rise in BP and arterial smooth muscle contracts to reduce the rise in BP
- Vicious cycle with prolonged arterial smooth muscle contraction leads to endothelial dysfunction and the inability to release more nitric oxide resulting in further increase in BP
- Shearing forces on the vascular wall result in further endothelial damage and dysfunction

Pathophysiology of Hypertensive Emergency

- Endothelial dysfunction results in the release of inflammatory markers (cytokines, endothelin-1, endothelial adhesion molecules)
- Promotes platelet aggregation, coagulation, and endothelial permeability
- Vasoconstriction and thrombosis
- Hypoperfusion and end organ ischemia
- Patients with chronic HTN have adapted to the higher BP
- Patients who are normotensive and have an abrupt increase in BP do not have any adaptive mechanisms. Even a small abrupt rise in BP can induce a hypertensive emergency

Etiology of Hypertensive Emergencies

- Hypertensive encephalopathy
- Pulmonary edema/acute heart failure
- Myocardial ischemia
- Aortic dissection
- Pregnancy

Assessment

- History
- Physical examination
- Laboratory testing
- Imaging

History

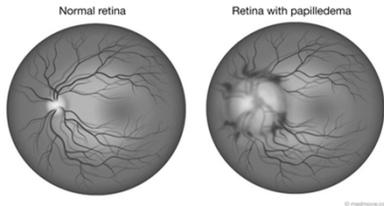
- Chest pain, shortness of breath, dyspnea on exertion, orthopnea
- Neurologic: agitation, acute confusion, delirium, stupor, transient loss of consciousness, visual disturbances, seizures, weakness, sensory deficits, headache (suggests hypertensive encephalopathy)
- Acute head trauma or falls
- Acute, severe back or neck pain
- Anxiety, nausea or vomiting
- Medication, dietary, and hemodialysis adherence and/or nonadherence

Medications/Substance Use

- Cocaine
- Methamphetamines
- Thyroid replacement hormones
- Monoamine oxidase inhibitors
- Abrupt withdrawal of clonidine
- Alcohol

Physical Examination

- Retinal hemorrhage, blurred vision, and/or papilledema (optic disc swelling caused by increased intracranial pressure)



Physical Examination

- Pulmonary: crackles, hypoxia, tachypnea (suggests pulmonary edema)
- Cardiac: murmurs (new diastolic decrescendo suggests aortic regurgitation and Type A aortic dissection)
- Vascular: Proximal and distal pulses; blood pressure
- Volume status:
 - Acute increase in BP results in natriuresis (excretion of large amounts of sodium in urine)
 - Appropriate response that decreases blood volume to decrease BP

BP Measurement

- Initially check for postural changes by taking readings after 5 mins supine, then 2 mins after standing (important in persons over age 65, diabetics or those taking antihypertensive medications)
- Arm at level of heart
- Do not measure BP over clothes

Whelton PK, Carey RM, Aronow WS, et al. 2017 Guideline for the Prevention, Detection, Evaluation and Management of High Blood Pressure in Adults. American College of Cardiology Foundation and American Heart Association. <http://professional.heart.org/hypertension>.

BP Measurement

- Ensure BP devices calibrated and maintained
- Patients should sit comfortably with back supported, legs uncrossed ideally for 5 minutes before BP measurement
- Take at least 3-4 readings
- Initially take BP in both arms; use the higher reading
- Cuff size: length of bladder should be 80% and width of bladder should be at least 40% of the circumference of the upper arm
- Document the BP reading, patient position, arm, and cuff size: 140/90 mm Hg, seated, right arm, large adult cuff

Whelton PK, Carey RM, Aronow WS, et al. 2017 Guideline for the Prevention, Detection, Evaluation and Management of High Blood Pressure in Adults. American College of Cardiology Foundation and American Heart Association. <http://professional.heart.org/hypertension>.

Laboratory Testing and Imaging

- Assessment for end-organ damage
- Serum electrolytes and creatinine
- Urinalysis
- Cardiac enzymes (if chest pain present)
- 12 lead ECG
- Chest x-ray
- CT or MRI of brain (if head injury, neurologic symptoms, hypertensive retinopathy, nausea or vomiting present)
- Contrast CT, transesophageal echo (TEE), or MRI chest if aortic dissection suspected

Initial Management

- Goal is to reduce BP rapidly to prevent organ damage
- Therapy decisions weigh risks and benefits, which organs are compromised, and comorbidities
- Patient is admitted to an ICU or intermediate care unit for IV titration of antihypertensive medications and cardiopulmonary monitoring
- **Goal is to ↓BP 15-25% of initial values in the first minutes of treatment or about 110 mmHg DBP; goal is < 140/90 mmHg gradually achieved over several hours and days**

Chobanian AV, Bakris GL, Black HR, et al. The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure: the JNC report. JAMA 2003;289:2560-72.

Initial Management

- Oral antihypertensive medications started for long-acting therapy before IV medications are titrated down and discontinued
- If patient already on an oral antihypertensive at home, restart that medication. Remember side effects, onset of action, and half-life as iatrogenic hypotension is common

Medication Management

- Nitrates
 - Sodium Nitroprusside
 - Nitroglycerin
- Calcium Channel Blockers
 - Celvedipine
 - Nicardipine
- Dopamine-1 Agonist
 - Fenoldopam
- Adrenergic-Blockers
 - Labetolol
 - Esmolol

Nitrates: Sodium Nitroprusside

- Arterial and venous dilation that ↓ preload and afterload
- Initial dose 0.5 mcg/kg/min, increase 0.5 mcg/kg/min until goal BP reached
- Onset of action < 2 min
- Duration 1-10 min
- Max dose 10 mcg/kg/min for no longer than 10 min
- BP monitoring in ICU with arterial catheter required
- Protect infusion bag from light

Sodium Nitroprusside Concerns

- Hypotension, restlessness, headache
- Nausea and vomiting
- “Coronary steal” in patients with CAD
 - Shunting of blood from diseased coronary vessels to well perfused coronary vessels may produce coronary ischemia
- May ↑ ICP and ↓ CBF (impairment of autoregulation)
- Do not use in patients with impaired kidney function or use very carefully

Sodium Nitroprusside Concerns

- Thiocyanate/cyanide toxicity when used for more than 72 hours
- Symptoms:
 - Odor of almonds on breath
 - Tachycardia
 - Mental status changes (lethargy to agitation)
 - Metabolic acidosis
- Management
 - Stop medication!
 - 100% oxygen
 - Sodium thiosulfate or hydroxocobalmin (B12)

Nitroglycerin

- Venous dilation that ↓ preload, ↑ coronary blood flow, inhibits coronary vasospasm, and ↓ cardiac oxygen demands
- Arterial dilation but only at higher doses
- Preferred medication in patients with ischemic heart disease, after CABG, pulmonary edema or in the management of aortic dissection (along with a beta-blocker)
- Initial dose 5 mcg/kg/min, which can be increased 5 mcg/kg/min every 3-5 min; after 20 mcg/kg/min it can be incrementally increased by 20 mcg/kg/min
- No dosing limits but the risk of hypotension greater after 200 mcg/kg/min
- Onset of action 2-5 min
- Duration 5-10 min

Nitroglycerin Concerns

- Headache (most common side effect)
- Hypotension (assess volume status prior to initiating to ensure adequate preload)
- Tachycardia
- Methemoglobinemia
- Tachyphylaxis (rapidly diminishing response to successive doses of a drug, rendering it less effective)
- Avoid if recent use of Viagra in previous 24 hrs or Cialis in previous 48 hrs
- Vomiting
- Adheres to tubing?

Altavilla JL, Haas CE, Nowak DR, et al. Clinical response to intravenous nitroglycerin infused through polyethylene or polyvinyl chloride tubing. Am J Hosp Pharm. 1994;51:490-494.

Calcium Channel Blocker: Clevidipine

- Direct coronary vasodilator → ↑ coronary blood flow, stroke volume and cardiac output, ↓ peripheral vascular resistance (PVR)
- Protects against perfusion/reperfusion injury, maintains splenic and renal blood flow
- Initial dose 1-2 mg/hr, usual dose 4-6 mg/hr or 0.4 mcg/kg/min and titrated up by doubling increments every 90 seconds to a max of 3.2 mcg/kg/min
- Onset of action 2-4 min
- Duration 5-15 min
- Very short half-life of 2-4 min

Espina M, Varon J. Clevidipine: a state-of-the-art antihypertensive drug under the scope. Expert Opin Pharmacother. 2012;13(3):387-93.

Clevidipine Concerns

- Tachycardia
- Headache
- Flushing
- Hypotension
- Use cautiously in patients with severe aortic stenosis and acute heart failure
- Lipid emulsion; IV tubing changed every 12 hrs to prevent bacterial contamination
- Avoid in patients with soy or egg allergies

Nicardipine

- Coronary and cerebral vasodilation (CAD: ↑ SV and coronary blood flow)
- Initial infusion dose is 5 mg/hr and titrated up by 2.5 mg/hr every 5 min for a max dose of 15 mg/hr (30 mg/hr can be tolerated)
- Onset of action 5-15 min
- Duration of action 15-30 min; may last up to 3-4 hrs
- IV nicardipine compared to nitroprusside: nicardipine provided a greater reduction in SBP and DBP and was more effective in patients with severe HTN
- IV nicardipine compared to labetalol use in the emergency department: nicardipine was more effective in reaching SBP within 30 min than labetalol

Neutel JM, Smith DH, Wallin D, et al. A comparison of intravenous nicardipine and sodium nitroprusside in the immediate treatment of severe hypertension. Am J Hypertens. 1994;7(4 Pt 1):1023-6.

Peacock WF, Varon J, Baumgart BM, et al. CLUE: a randomized comparative effectiveness trial IV nicardipine versus labetalol use in the emergency department. Crit Care. 2011;15(3):R157.

Nicardipine Concerns

- Flushing
- Hypotension
- Palpitations
- Angina
- Syncope
- Peripheral edema
- Headache
- Vomiting
- Avoid in acute heart failure

Dopamine-1 Agonist: Fenoldopam

- Renal artery vasodilation that promotes natriuresis (inhibits sodium reabsorption) and diuresis
- Initial dose 0.1 mcg/kg/min and titrated up by 0.1 – 0.2 mcg/kg/min every 20 min to target BP
- Onset of action 5-10 min
- Duration of action 30-60 min
- Use for short term BP reduction for up to 48 hrs
- Useful in patients with renal impairment, acute heart failure, and patients undergoing vascular surgery

Fenoldopam Concerns

- Tachycardia, chest pain
- Hypotension
- Flushing
- Headache
- Hypokalemia
- Nasal congestion
- ↑ intraocular pressure, blurred vision
- Avoid in patients with myocardial ischemia, glaucoma, or intracranial HTN

Adrenergic-Blocker: Labetalol

- Alpha 1 and beta receptor blocker (↓BP without reflex tachycardia)
- Administer as a bolus or continuous infusion
- Initial dose 20 mg bolus followed by either bolus of 20-80 mg every 10 min or titrated to a continuous infusion rate of 1-2 mg/min
- Onset of action 2-5 min
- Duration of action 2-4 hrs
- Max dose is 300 mg

Labetalol Indications

- Aortic dissection
- Acute coronary syndrome
- Hypertensive encephalopathy
- Adrenergic crises
- Preeclampsia related crises (limited ability to cross maternal-fetal barrier)

Labetalol Concerns

- Avoid in patients with heart block, bradycardia, asthma, COPD, and acute heart failure
- May be used for BP control in conjunction with a vasodilator in patients with hypertensive crisis due to cocaine
- Side effects: AV conduction disturbances, headache, bronchospasm, nasal congestion, scalp tingling, nausea, vomiting
- Do not stop abruptly (may precipitate ischemic chest pain in patients with CAD)

Adrenergic Blocker: Esmolol

- Cardioselective beta blocker: ↓HR and myocardial contractility without vasodilation
- Initial dose 0.5 mg/kg loading dose over 60 seconds followed by continuous infusion starting at 50 mcg/kg/min and titrated up to 300 mcg/kg/min
- Onset of action 1-2 min (ultra short acting)
- Duration of action 10-30 min
- Indications: aortic dissection, intra- and postoperative HTN
- Can be used in patients with renal or hepatic disorders

Esmolol Concerns

- Avoid in patients who are already on a beta blocker, have bradycardia, or have heart failure exacerbation
- Side effects: hypotension, nausea, asthma exacerbation, first degree heart block, heart failure

Etiology of Hypertensive Emergencies

- Hypertensive encephalopathy
- Pulmonary edema/acute heart failure
- Myocardial ischemia
- Aortic dissection
- Pregnancy

Hypertensive Encephalopathy

- Brain has autoregulatory system to maintain a certain cerebral perfusion pressure through vasoconstriction and dilation
- Normotensive patient maintains a mean arterial pressure (MAP) of 60-120 mm Hg
- Normotensive patient suddenly develops HTN → autoregulatory system becomes overwhelmed → at risk cerebral edema and hypertensive encephalopathy
- Patients with long-standing HTN → autoregulatory system adapts gradually to severely increased BP with a decreased likelihood of hypertensive emergency with moderately increased BP

Hypertensive Encephalopathy Presentation

- Acute delirium
- Lethargy
- Confusion
- Severe headache
- Seizures
- Consider other causes of altered mental status (infection, stroke)

Hypertensive Encephalopathy Management

- Symptom control
- Delirium and seizure control: benzodiazepines, fosphenytoin, phenytoin, barbituates (will reduce BP as well)
- Absence of stroke: reduce MAP by 20-25% in the first hour with IV nitroprusside, labetalol, nicardipine, and/or enalapril
- Avoid clonidine as the sedative side effects can make it difficult to assess resolution of neurologic symptoms

Adebayo O, Rogers RL. Hypertensive emergencies in the emergency department. Emerg Med Clin N Am 2015; 33:539-551.
Schwartz RB. Hyperperfusion encephalopathies: hypertensive encephalopathy and related conditions. Neurologist 2002; 8:22-34.

Pulmonary Edema/Acute Heart Failure

- Acute HF can occur in hypertensive crisis, but it can also be a risk factor for the development of hypertensive crisis
- Unclear if this is a result of systolic (HFrEF) or diastolic dysfunction (HFpEF)
- Patients with HFrEF are not able to tolerate fluctuations in BP resulting in volume overload and pulmonary edema
- Chest x-ray, 12 lead ECG, TEE, serum electrolytes and creatinine

Pulmonary Edema/ Acute Heart Failure Management

- Treatment focuses on preload and afterload reduction
- Oxygen (face mask, bipap mask, intubation)
- Nitroglycerin: venous dilation → preload reduction
- Nitroprusside: rapid onset, venous and arterial dilation → preload and afterload reduction
- Clevidipine: calcium channel blocker that ↓ PVR
- Lasix: loop diuretic to induce diuresis (if patient hypervolemic) and to treat dyspnea; be aware that many of these patients may be hypovolemic
- Avoid hydralazine (↑ HR and cardiac workload), beta blockers (↓ cardiac contractility)

Myocardial Ischemia

- Hypertensive emergency can cause myocardial ischemia especially in patients with acute coronary syndrome (ACS)
- Symptoms of cardiac ischemia: chest pain, dyspnea, diaphoresis, vomiting, and fatigue
- Severe HTN along with cardiac ischemia is associated with ↑myocardial work and ↑oxygen demand

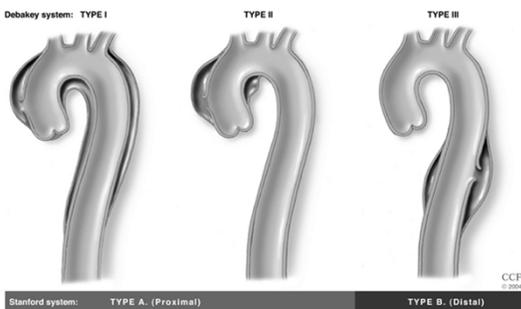
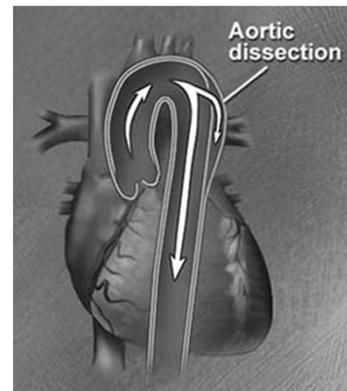
Myocardial Ischemia Management

- Treatment of ischemic chest pain focuses on ↓ myocardial work, myocardial oxygen consumption, and ↑coronary artery perfusion
- IV beta-blocker (labetalol, esmolol): ↓HR, ↓afterload, and improve coronary artery perfusion
- Nitroglycerin: ↓LV preload
- Avoid hydralazine (↑HR and cardiac workload)

Aortic Dissection

- Sudden onset of chest pain: **R**apid onset, **R**ipping, **R**adiating to chest, back or both (3 R's)
- Pain in any extremity, neurologic symptoms
- Type A and Type B
- Measure BP in both arms
- Assess pulses in all four extremities
- New diastolic murmur (aortic regurgitation)
- Chest x-ray, CT scan, MRI and/or TTE/TEE

Hiratzka LF, Bakris GL, Beckman JA, et al. ACCF/AHA/AATS/ACR/ASA/SCA/SCAI/SIR/STS/SVM guidelines for the diagnosis and management of thoracic aortic disease. Circulation 2010;121:e268.



Aortic Dissection Management

- Goal: rapid and large reduction in vascular shear stress and BP to prevent further dissection and rupture
- Target BP: SBP 100-120 mmHg; MAP <80 mmHg in 20 minutes if possible
- Target HR: < 65 bpm
- Beta blocker (labetalol, esmolol, propranolol, metoprolol); reduces shear stress by ↓HR and prevents reflex tachycardia when arterial vasodilators given
- Nitroprusside: reduces systolic BP quickly
- Clevidipine: calcium channel blocker that ↓PVR
- Avoid hydralazine as it ↑myocardial contractility

Braverman AC. Acute aortic dissection: clinician update. Circulation 2010; 122:184-8.

Pregnancy



- HTN is a complication of 5-7% of all pregnancies and is still a leading cause of fetal and maternal morbidity
- HTN of pregnancy: systolic BP > 140 mmHg and/or diastolic BP > 90 mmHg
- Preeclampsia: HTN and proteinuria, but can involve multiple organ systems

Report of the National High Blood Pressure Education Program Working Group on high blood pressure in pregnancy. Am J Obstet Gynecol 2000;183:51-22

Pregnancy

- Consensus that HTN of pregnancy should be treated in patients with a sustained systolic BP >160 mmHg and/or diastolic BP >110 mmHg
- No consensus on when and how to treat systolic BP 140-159 mm Hg and diastolic BP 90-105 mmHg
- Labetalol (safe to use when breast feeding), nifedipine, magnesium, and nitroprusside (as a last resort)

American College of Obstetricians and Gynecologists. Diagnosis and management of preeclampsia and eclampsia. Obstet Gynecol. 2005;105:402-10.

Evaluation After Persistent HTN Diagnosed

- Determine precipitating factors including medications and risk factors for CVD
- Physical examination for signs of organ dysfunction (e.g. retinopathy)
- Laboratory testing
 - Hematocrit, urinalysis, blood chemistries (glucose, creatinine, electrolytes) and estimated glomerular filtration rate (GFR)
 - Fasting lipid profile (total and HDL cholesterol, triglycerides)
 - ECG

Management

- Lifestyle modification!
 - Low salt diet
 - Weight loss
 - Reduce alcohol intake to 1-2 drinks per day
 - Stop smoking
- Continue antihypertensive medication if systolic BP \geq 140 mmHg and diastolic BP \geq 90 mmHg
- The incidence of stroke can be lowered with more aggressive lowering of BP in patients with diabetes and HTN
- **Amount of blood pressure reduction is the major determinant in reducing CV risk in persons with HTN, not the choice of the antihypertensive medication**

Medications

- Thiazide diuretics (initially)
 - Chlorthalidone
- Calcium channel blockers (long acting)
 - Amlodipine
- Angiotensin converting enzyme inhibitors (ACE-I)
 - Captopril, enalapril, lisinopril
- Angiotensin II receptor blockers (ARB)
 - Candesartan, losartan
- Thiazide diuretics and calcium channel blockers may have greater BP-lowering efficacy than other classes in African-Americans
- Beta blockers not first line therapy for HTN in persons older than 60

Strategies to Promote Lifestyle Changes

- **Goal setting**
People who target a specific behavior change are more likely to be successful
Goals must be specific and realistic
Focus on behavior (\uparrow whole grain intake) rather than a lab value
Need to provide regular feedback on goal attainment e.g. weight loss

American Heart Association. Interventions to promote physical activity and dietary lifestyle changes for cardiovascular risk reduction in adults. 2010. Whelton PK, Carey RM, Aronow WS, et al. 2017 Guideline for the Prevention, Detection, Evaluation and Management of High Blood Pressure in Adults. American College of Cardiology Foundation and American Heart Association. <http://professional.heart.org/hypertension>

Strategies to Promote Lifestyle Changes

- Assess readiness to learn
- Assess current knowledge
- Determine obstacles to learning
- Determine reading level
- Determine best learning style
 - Written materials
 - Videos
 - Individual learning
 - Group learning
 - Computer based

Strategies to Promote Lifestyle Changes

- **Frequent and prolonged contact**
Follow up sessions, face-to-face contact, telephone calls, email, internet
Provides social support from peer group
- **Feedback and reinforcement**
- **Incentives**
- **Role modeling**
- **Problem solving**
How to get support from family when attempting dietary changes

American Heart Association. Interventions to promote physical activity and dietary lifestyle changes for cardiovascular risk reduction in adults. 2010
Whelton PK, Carey RM, Aronow WS, et al. 2017 Guideline for the Prevention, Detection, Evaluation and Management of High Blood Pressure in
American College of Cardiology Foundation and American Heart Association. <http://professional.heart.org/hypertension>.

Case Study

- 67 year old male transferred via ambulance from a primary care clinic to the ED with a BP 228/120 mmHg and confusion.
- Patient has a long history of uncontrolled HTN, type 2 DM, and hypercholesterolemia.
- On admission to ED: HR 78 bpm, RR 18 breaths/min, BP 228/116 mmHg, room air oxygen saturation 96%
- Lethargic but easily arousable
- Disoriented to time and place

- Wife shares that the previous night he had been acting strangely; this morning he was confused about the time and year.
- She took him to their primary care office where the NP called 911.
- She reports no history of recent falls, no trauma, and no substance use.
- She shares that he stopped taking his blood pressure medication about 6 months ago because they made him "very tired" .
- She share his current medications: Lipitor 10 mg daily; Metformin 500 mg twice daily

- Per the EMR he is prescribed amlodipine (calcium channel blocker) 10 mg daily and Toprol (beta blocker) 50 mg daily but has not filled his prescriptions for the past 6 months.
- Weight: 90 kg
- Physical exam: retinopathy, pulses present and equal bilaterally (femoral, posterior tibial, dorsalis pedis), not following commands, equal BP on both arms
- Denies chest pain, SOB
- No focal neurological deficits

- **What are possible etiologies?**
- **What diagnostic tests should be performed?**

- **Possible etiologies:**
 - Acute coronary syndrome (ACS)
 - Stroke
 - Hypertensive encephalopathy
- **Diagnostic tests:**
 - Head CT to rule out stroke
 - 12 lead ECG to rule out ACS
 - Troponin levels
 - Chest x-ray to rule out thoracic aortic aneurysm
 - CBC and BMP to rule out electrolyte derangement
 - Urinalysis and urine culture to rule out infection

Results

- Head CT negative for hemorrhagic stroke, but white matter changes present that would suggest hypertensive encephalopathy
- Troponin levels normal
- 12 lead ECG: NSR with LV hypertrophy
- Creatinine 2.5 mg/dL which is 2.5 times greater than 6 months ago
- BUN 40 mg/dL which is elevated compared to 23 mg/dL 6 months ago
- Urinalysis normal

Suspected Diagnosis?

Suspected Diagnosis

- **Hypertensive emergency causing target organ damage (kidney, brain, heart)**
- Renal ultrasound performed to evaluate for kidney stones, an enlarged prostate, or a mass which could obstruct flow of urine and contribute to acute kidney injury
- Result: shrunken left kidney with **renal artery stenosis**
- Brain: Cerebral perfusion pressure maintained in HTN by vasoconstriction; higher MAP needed → contribute to vasodilation, hyperperfusion, and cerebral edema → **hypertensive encephalopathy**

Risk Factors for CVD

- Obese
- Diabetes
- Older age
- Hypertension

Management

- Goal: reduce MAP by 15-25% within the first hour; DBP to 110 mmHg
- Will improve/eliminate confusion
- Over the next 24 hrs, goal is SBP 140 mmHg or less and DBP 90 mmHg or less
- Admit to ICU
- IV antihypertensive infusions:
 - Fenoldopam: vasodilation of systemic and renal circulation
 - Nicardipine
- Transition to oral antihypertensive medications
- Patient/family education

Questions?

