HYPERTENSION

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Blood Pressure Cardiac Output Systemic Vascula Resistance



Hear rate

- Sympathatic/Parasympathatic
- Vasoconstriction/vasodilation

Fluid volume

- Renin-angiotensin
- Aldosterone
- > ADH

FACTORS INFLUENCING BP



HYPERTENSION DIAGNOSIS

- Diagnosis requires two reading at two different clinic visits
- BP measurement in both arms
 - Use arm with higher reading for subsequent measurements

Measure BP following 5min of rest in the sitting position with good back support



Office BP Readings: Checklist for Accurate Measurements

Key Points	Specific Instructions
Step 1: Prepare patient	 -Have patient relax, sitting in a chair (feet on floor, back supported) for >5 min. -Avoid caffeine, exercise, and smoking for ≥ 30 min before measurement. -Ensure bladder emptied. -No talking during rest period or measurement. -Remove clothing covering location of cuff placement. -Measurements while patient sitting/lying on exam table do not fulfill criteria.
Step 2: Use proper technique	-Use validated BP measurement device that is calibrated periodically. -Support patient's arm (e.g., resting on a desk). -Position middle of cuff on patient's upper arm at mid-sternum (right atrium). -Use correct cuff size, such that the bladder encircles 80% of the arm. -Either stethoscope diaphragm or bell may be used for auscultatory readings.
Step 3: Take proper measurements	-At first visit, record BP in both arms. Subsequently, use arm with higherBP. -Separate repeated measurements by 1–2 min. -For auscultatory readings, estimate SBP by palpation and inflate cuff 20–30 mm Hg above. Deflate 2 mm Hg per second and listen for Korotkoff sounds.
Step 4: Document BP readings	-Note time of most recent BP medication before measurements. -Record SBP and DBP.
Step 5: Average readings	-Use average of ≥ 2 readings obtained on ≥ 2 occasions to estimate level of BP.

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Whelton PK et al. Hypertension/J Am Coll Cardiol. 2017;Epub ahead of print

CLASSIFICATION OF HYPERTENSION

- Primary (Essential) Hypertension
 - Elevated BP with unknown cause
 - 90% to 95% of all cases
- Secondary Hypertension
 - Elevated BP with a specific cause
 - 5% to 10% in adults



Age (> 55 for men; > 65 for women)

- Alcohol
- Cigarette smoking
- Diabetes mellitus
- Elevated serum lipids
- Excess dietary sodium
- Gender

RISK FACTORS FOR PRIMARY HYPERTENSION



- Family history
- ▷ Obesity (BMI ≥ 30)
- Ethnicity (African Americans)
- Sedentary lifestyle
- Socioeconomic status
- Stress



CLINICAL MANIFESTATIONS

- > Asymptomatic
- > Non-specific symptoms
- Fatigue
- **Reduced activity tolerance**
- Dizziness
- **Palpitations**
- > End organ damage



BASIC AND OPTIONAL LABORATORY TESTS FOR PRIMARY HYPERTENSION

Basic testing	Fasting blood glucose*
	Complete blood count
	Lipid profile
	Serum creatinine with eGFR*
	Serum sodium, potassium, calcium*
	Thyroid-stimulating hormone
	Urinalysis
	Electrocardiogram
Optional testing	Echocardiogram
	Uric acid
	Urinary albumin to creatinine ratio
*May be included ir	n metabolic panel. eGFR indicates estimated glomerular filtration rate.

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Whelton PK et al. Hypertension/J Am Coll Cardiol. 2017 [Epub ahead of print].

BP Classification (JNC 7 and ACC/AHA Guidelines)

SBP		DBP	2003 JNC7	2017 ACC/AHA	
<120	and	<80	Normal BP	Normal BP	
120–129	and	<80		Elevated BP	Major area
130–139	or	80–89	Prehypertension	Stage 1 hypertension	- of difference
140–159	or	90-99	Stage 1 hypertension	Stage 2 hypertension	
≥160	or	≥100	Stage 2 hypertension	Stage 2 hypertension	

• Blood Pressure should be based on an average of ≥ 2 careful readings on ≥ 2 øccasions

• Adults with SBP or DBP in two categories should be designated to the higher BP category

Whelton PK et al. Hypertension/J Am Coll Cardiol. 2017; Epub ahead of print

Out of Office BP Readings

Greater use of out of office BP measurements (ABPM or HBPM) for confirmation of office hypertension and recognition of White Coat/Masked Hypertension

Confirmed (Sustained) Hypertension

- Elevated office and out of office average BP
- Require therapy (nonpharmacological or combined nonpharmacological and antihypertensive drug therapy)

White Coat Hypertension (WCH)

- Office Hypertension not confirmed by out of office BP readings
- Present in about 10-25% of adults with office hypertension
- CVD risk profile more like adults with normal BP than adults with sustained hypertension
- May not need treatment for hypertension (should be monitored for development of sustained hypertension)

Masked Hypertension (MH)

- Normal office BP but out of office BP hypertension
- Present in about 10-25% of adults with normal office BP
- CVD risk profile more like adults with sustained hypertension than adults without hypertension
- Should be considered for antihypertensive drug therapy

Whelton PK et al. Hypertension/J Am Coll Cardiol. 2017; Epub ahead of print

HYPERTENSION COMPLICATIONS

End organ damage involves:

Heart
Brain
Kidney
Eyes



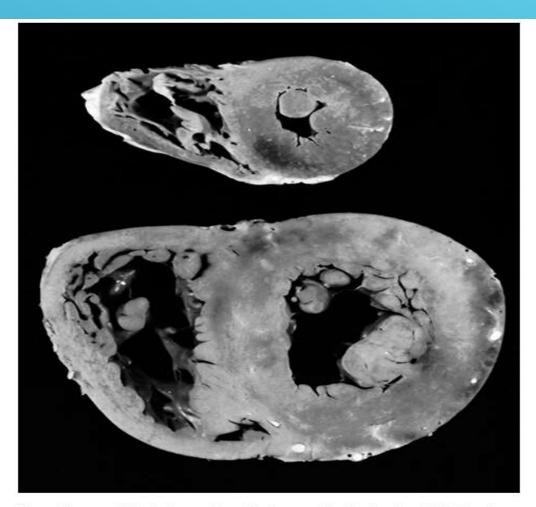
HYPERTENSION COMPLICATIONS

Cardiovascular Disease

- Coronary artery disease
- Left ventricular hypertrophy
- Diastolic dysfunction
- Heart failure
- Peripheral arterial disease
- Aneurysm and dissection



LEFT VENTRICULAR HYPERTROPHY



From Kissane JM: Anderson's pathology, ed 9, St. Louis, 1990, Mosby. Copyright © 2004, 2000, Mosby, Inc. All Rights Reserved.



HYPERTENSION COMPLICATIONS

CNS
 Ischemic stroke
 Hemrrhagic stroke
 Hypertensive Encephalopathy



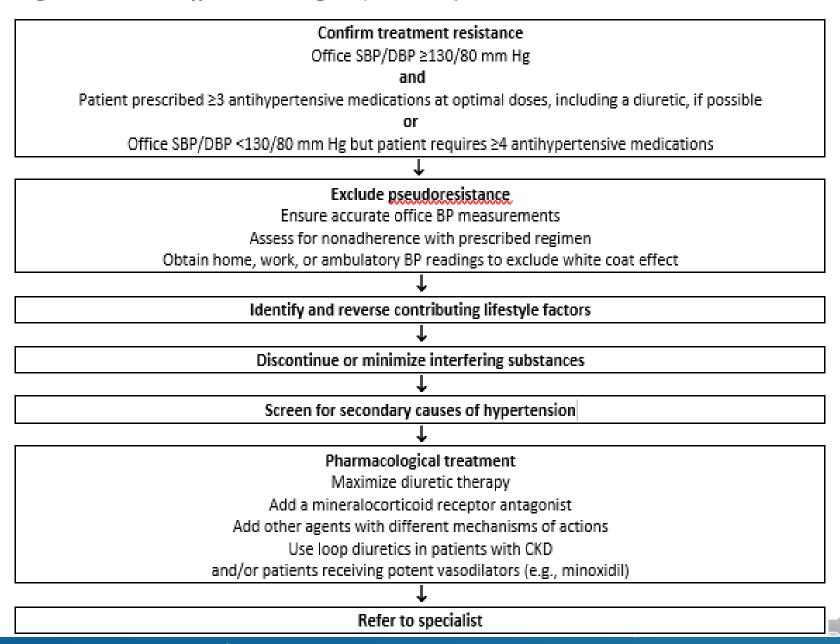
Kidney:
 Nephrosclerosis
 Major cause for End stage Renal Failure

> Ophthalmic:

Retinal complication including bleeding



RESISTANT HYPERTENSION: DIAGNOSIS, EVALUATION, AND TREATMENT



Whelton PK et al. Hypertension/J Am Coll Cardiol. 2017 [Epub ahead of print].

- "Secondary" HTN accounts for ~5-10% of other cases and represents potentially curable disease
- Often overlooked and underscreened
- Controversy over screening and treatment in some cases

SECONDARY HTN



Secondary Hypertension

Underlying cause of high BP in about 10% of adults with hypertension

Common causes

Renal parenchymal disease
Renovascular disease
Primary aldosteronism
Obstructive sleep apnea
Drug or alcohol induced
Uncommon causes
Pheochromocytoma/paraganglioma
Cushing's syndrome
Hypothyroidism
Hyperthyroidism
Aortic coarctation (undiagnosed or repaired)
Primary hyperparathyroidism
Congenital adrenal hyperplasia
Mineralocorticoid excess syndromes other than primary aldosteronism
Acromegaly

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Whelton PK et al. Hypertension/J Am Coll Cardiol. 2017 [Epub ahead of print].

General principles:

- New onset HTN if <30 or >50 years of age
- > HTN refractory to medical Rx (>3 meds)
- Specific clinical/lab features typical for certain disease entity:
 - > Hypokalemia,
 - Epigastric bruit
 - Differential BP between arm and leg
 - > Episodic HTN/flushing/palp, etc

SCREENING



- Common cause of secondary HTN
- HTN is both a cause and consequence of renal disease
- Multifactorial cause for HTN including disturbances in Na/water balance, depletion
- of vasodilators leading to highTPR
- Renal disease from multiple etiologies, treat underlying disease, dialysis/ transplant if necessary

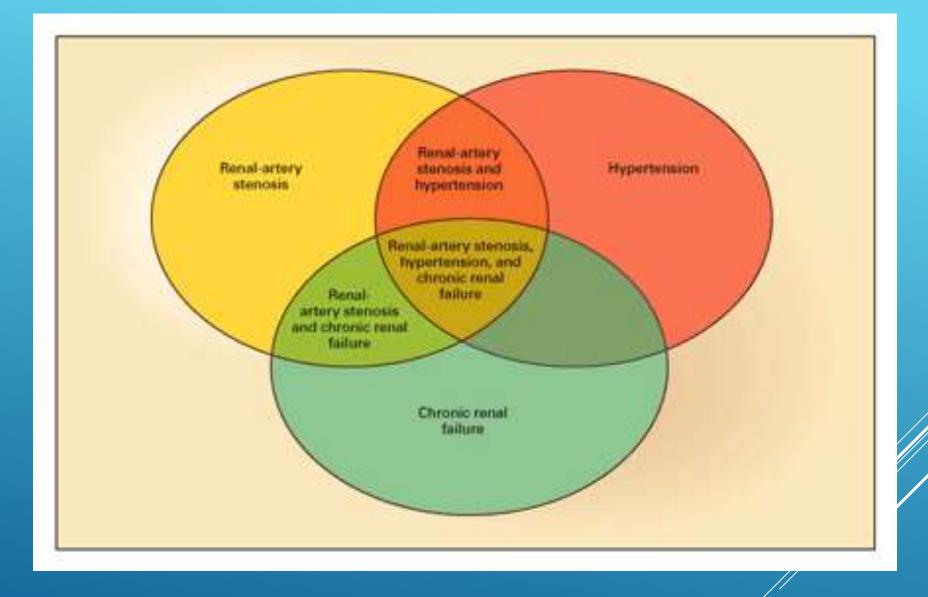
RENAL PARENCHYMAL DISEASE



- Incidence 1-30%
- Etiology
 - > Atherosclerosis 75-90%
 - > Fibromuscular dysplasia 10-25%
 - > Other
 - Aortic/renal dissection
 - Takayasu's arteritis
 - > Thrombotic/cholesterol emboli
 - > CVD
 - Post transplantation stenosis
 - Post radiation

RENOVASCULAR HTN





Safian & Textor. NEJM 344:6;p 432



 Decrease in renal perfusion pressure activates RAAS, renin release converts angiotensinogen -> Ang I; ACE converts Ang I-> Ang II

- Ang II causes vasoconstriction which causes HTN and enhances adrenal release of aldosterone; leads to sodium and fluid retention
- Contralateral kidney (if unilateral RAS) responds with diuresis/ Na, H2O excretion which can return plasma volume to normal
- Bilateral RAS or solitary kidney RAS leads to rapid volume expansion and ultimate decline in renin secretion

RENOVASCULAR HTN -PATHOPHYSIOLOGY



History

- onset HTN age <30 or >55
- Sudden onset uncontrolled HTN in previously well controlled pt
- Accelerated/malignant HTN
- Intermittent pulm edema with nl LV fxn
- > PE/Lab
 - > Epigastric bruit, particulary systolic/diastolic
 - > Azotemia induced by ACEI
 - Unilateral small kidney

RENOVASCULAR HTN - CLINICAL



- Physical findings (bruit)
- Duplex U/S
- Captopril renography
- ► CTA
- ► MRA
- Renal Angiography

RENOVASCULAR HTN -DIAGNOSIS



- ▶ 10-25% of all RAS
- > Young female, age 15-40
- Medial disease 90%, often involves distal RA
- Treatment PTCA
 - Successful in 82-100% of patients
 - Restenosis in 5-11%
 - "Cure" of HTN in ~60%

FIBROMUSCULAR DYSPLASIA



- > 75-90% of RAS
- Usually men, age>55
- Treatment

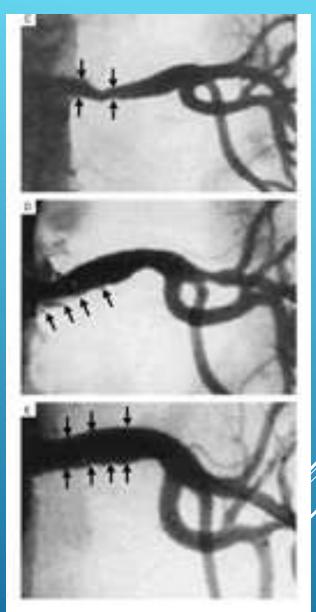
Stent success 94-100%

ATHEROSCLEROTIC RAS





Fibromuscular Dysplasia, before and after PTCA



Safian & Textor. NEJM 344:6;

Atherosclerotic RAS before and after stern

Aggressive risk fx modification (lipid, tobacco, etc)

 ACEI/ARB safe in unilateral RAS if careful titration and close monitoring

RENOVASCULAR HTN – MEDICAL RX



- Prevalence .5- 2.0%
- Etiology
 - > Adrenal adenoma 33%
 - bilat adrenal hyperplasia 66%
- Clinical:
 - May be asymptomatic; headache, muscle cramps, polyuria
 - Hypokalemia (K normal in 40%-70%), metabolic alkalosis, high Na

PRIMARY HYPERALDOSTERONISM



- Aldosterone / Plasma Renin Activity ratio
 Ratio >20
- Confirmatory/physiologic testing
 - > Withold BP meds 2wks
 - High serum aldo after IV saline (1.25L x 2hr) load
 - serum aldo <8.5 ng/dL after IV saline rules out primary aldosteronism
 - Imaging CT

PRIMARY ALDOSTERONISM- DX



- Surgical removal of adrenal tumor, can be done laparoscopically
- Pretreatment for 3-4 wks with spironolactone minimizes postoperative hypoaldosteronism and restores K to normal levels, response of BP to spiro treatment is predictor of surgical outcome

PRIMARY ALDOSTERONISM -TREATMENT



- Published reports estimate incidence of 30-80% of pt with essential HTN have OSA and 50% pt with OSA have HTN¹
- Prospective studies show link between OSA (apneic-hyponeic index) and development of HTN independent of other risk fx²
- Clinical
 - Daytime somnolescence, am headaches, snoring or witnessed apneic episodes
- Dx Sleep studies
- Rx wt loss, CPAP, surgical

OBSTRUCTIVE SLEEP APNEA



- Rare cause of HTN (.1-1.0%)
- Tumor containing chromaffin cells which secrete catecholamines
- > Young-middle age with female predominance
- Clinical
 - Intermittent HTN, palpitations, sweating, anxiety "spells"
 - May be provoked by triggers such as tyraminecontaining foods (beer,cheese,wine), pain, trauma, drugs (clonidine, TCA, opiates)

PHEOCHROMOCYTOMA



PHEOCHROMOCYTOMA - SCREEN

 Best detected during or immediately after episodes

	Sensitivity	Specificity
Plasma free metanephrine >.66nmol/L	99%	89%
24hr urine metanephrine (>3.7nmol/d)	77%	93%
24 urine VMA	64%	95%

Lenders, et al. JAMA 2002 Mar 20;287(11):1427-34

PHEOCHROMOCYTOMA - DIAGNOSIS

Imaging for localization of tumor

	Sens	Spec	PPV	NPV
(MIBG) scintigraphy	78%	100%	100%	87%
СТ	98%	70%	69%	98%
MRI	100%	67%	83%	100%

Akpunonu, et al. Dis Month.October 1996, p688



- Surgical removal of tumor
 - Anesthesia- avoid benzo, barbiturates or demerol which can trigger catechol release
 - Complications include ligation of renal artery, post op hypoglycemia, hemorrhage and volume loss
 - ▶ Mort 2%, 5 yr survival 95% with <10% recurrence
- Caution with BB can cause unopposed alpha stimulation/pheo crisis
- > BP control with alpha blockers (phentolamine, phenoxybenzamine, and prazosin)

PHEOCHROMOCYTOMA -TREATMENT



- Rare cause of secondary HTN (.1-.6%)
- Etiology: pituitary microadenoma, iatrogenic (steroid use), ectopic ACTH, adrenal adenoma
- Clinical
 - Sudden weight gain, truncal obesity, moon facies, abdominal striae, DM/glucose intolerance, HTN, prox muscle weakness, skin atrophy, hirsutism/acne

CUSHING'S SYNDROME/ HYPERCORTISOLISM



- Screen:
 - 24 Hr Urine free cortisol
- Confirm
 - Low dose dexamethasone suppression test
 - 1mg dexameth. midnight, measure am plasma cortisol
- Imaging
 - CT/MRI head (pit) chest (ectopic ACTH tumor)

CUSHINGS SYNDROME - DX



- Cushings dz/ pit adenoma
 - Transphenoidal resection
 - > Pituitary irradiation
 - > Bromocriptine, octreotide
- > Adrenal tumors adrenalectomy
- Removal of ACTH tumor

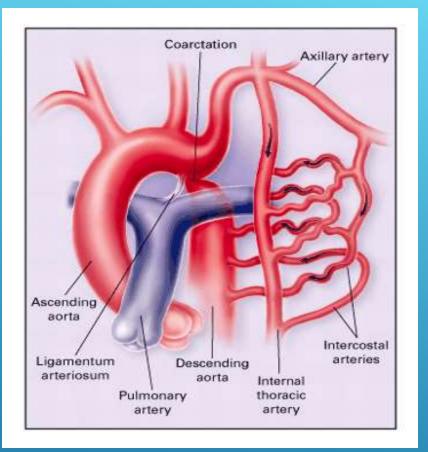
CUSHINGS SYNDROME - RX



- Congenital defect, male>female
- Clinical
 - Differential systolic BP arms vs legs
 - Diminished/absent femoral art pulse
 - > Often asymptomatic
 - > Assoc with Turners, bicuspid AV
- If uncorrected 67% will develop LV failure by age 40 and 75% will die by age 50
- Surgical Rx, long term survival better if corrected early

COARCTATION OF AORTA





COARCTATION OF AORTA

Brickner, et al. NEJM 2000;342:256-263



HYPERTENSION

- Lifestyle Modifications
 - Weight reduction
 - Limitation of alcohol intake
 - Regular physical activity
 - Avoidance of tobacco use
 - Stress management



HYPERTENSION

- Nutritional Therapy: DASH Diet = Dietary Approahes to Stop HTN
 - Sodium restriction
 - Rich in vegetables, fruit, and nonfat dairy products
 - Calorie restriction if overweight



Choice of Drug Therapy in Treatment of Hypertension

First-step agents:

- Compelling indication
 - Use agent(s) that concurrently lower BP (e.g. post-MI, SIHD, HF)
- > No compelling indication
 - > Achieving BP goal more important than choice of drug therapy
 - Diuretic or CCB often good choice, but
 - Drugs from following classes acceptable
 - > Diuretic (esp. long-acting thiazide-type agent such as chlorthalidone)
 - Calcium channel blocker (CCB)
 - > Angiotensin converting enzyme inhibitor (ACEI)
 - > Angiotensin receptor blocker (ARB)



ANTIHYPERTENSIVE DRUG TREATMENT: DIABETES MELLITUS

- In adults with hypertension and DM,
- If average BP ≥130/80 mm Hg, initiate antihypertensive drug therapy and treat to <130/80 mm Hg
- All first-line classes of antihypertensives (i.e., diuretics, ACE inhibitors, ARBs, and CCBs) useful and effective

Consider ACEI or ARBs in presence of albuminuria



ANTIHYPERTENSIVE DRUG TREATMENT: HEART FAILURE

Hypertension and heart failure with reduced ejection factor (HFrEF)

- Prescribe guideline directed medical therapy (GDMT)
 ACEI, ARB, BB, MRA
- Nondihydropyridine CCBs not recommended
- ▶ BP goal: <130/80 mm Hg

Hypertension and heart failure with preserved ejection factor (HFpEF)

- If symptoms of volume overload, prescribe diuretics
- If high BP persists, prescribe ACE inhibitors or ARBs and beta blockers
- ▶ BP goal: <130 /80 mm Hg



ANTIHYPERTENSIVE DRUG TREATMENT: ISCHEMIC HEART DISEASE

Adults with hypertension and stable ischemic heart disease (SIHD)

- Use GDMT medications (e.g., beta blockers, ACE inhibitors, or ARBs) for compelling indications (e.g., previous MI, stable angina)
- Add other drugs (e.g. dihydropyridine CCBs, thiazide diuretics, and/or mineralocorticoid receptor antagonists) as needed to control hypertension

BP target: <130/80 mm Hg



- A 22 year old medical student presented to the E/D with epistaxis, his BP in the right arm is 190/110mmHg and left arm 200/115mmHg.
- On exam he was found to have radio-femoral delay.
- What is the next step in your evaluation and what is the diagnosis ?





- A 30 year old female presents with muscle fatigue and was found to be hypertensive.
- Her BP 170/100mmHg, K=2.8meq
- What is the next step in your evaluation?
- > What is your diagnosis?





- A 27 year old female presents with palpitation, headache and hypertension.
- > These episodes come in paroxysmal pattern.
- > What is your clinical diagnosis?
- How do you confirm it?





- A 65 year old hypertensive gentleman, treated with amlodipine 5mg .Recently he noticed his BP readings to be out of control. Today on exam his BP is 190/105mmHG and there is a paraumbilical bruit.
- > What is the next step in your evaluation?
- > What is your diagnosis?





- A 50 year old non-compliant male patient who is to be hypertensive buy doesn't follow appropriate life style neither he takes his medications presents to the ED with chest pain, and was found to be hypertensive with BP 200/110 mmHG in the right arm and 140/80mmHG in the left arm.
- > What is your clinical diagnosis?
- > How to confirm ?

CASE 5



THANK YOU

