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Hypertension

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-HTN is one of most common Dx
-its prevalence is around 30%
-increase in either CO or SVR will increase BP

Blood Pressure = **Cardiac Output** x **Systemic Vascular Resistance**

Factors Influencing BP

- Hear rate
- Sympathatic/Parasympathatic Influencing:
- Vasoconstriction/vasodilation
- Fluid volume Controlled by:
  - Renin-angiotensin
  - Aldosterone
  - ADH

Hypertension
Diagnosis

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

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

-we take the higher reading, ex: rt arm systolic BP is 140 and Lt arm is 130, we take the rt arm measurement.

-the monitoring happens in the office, so we call the patient after 1 week to recheck the BP and initiate treatment if HTN is confirmed.

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.*
Step 6: Provide readings to patient	*-Provide patients SBP/DBP readings both verbally and in writing.*

Classification of Hypertension

- Primary (Essential) Hypertension
  - Elevated BP with unknown cause
  - 90% to 95% of all cases

- Secondary Hypertension: treating the underlying cause resolves the HTN
  - Elevated BP with a specific cause
  - 5% to 10% in adults

Risk Factors for Primary Hypertension

- Age (> 55 for men; > 65 for women)
- Alcohol
- Cigarette smoking
- Diabetes mellitus
- Elevated serum lipids
- Excess dietary sodium
- Gender

- **Family history**
- **Obesity (BMI $\geq$ 30)**
- **Ethnicity (African Americans) more than Caucasians**
- **Sedentary lifestyle**
- **Socioeconomic status**
- **Stress**

Clinical Manifestations

- **Asymptomatic** (it is called the silent killer)
- **Non-specific symptoms (if symptomatic)**

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

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	Prehypertension	Elevated BP
130–139	or	80–89		Stage 1 hypertension
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 occasions

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

- **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**
- **In adults not taking antihypertensive medication**
- **• Confirmed (Sustained) Hypertension**
- **□ Elevated office and out of office average BP**
- **□ Substantially higher risk of CVD compared to adults with normal office and out of office BPs**
- **□ 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**

- ABPM(AMBULATORY), HBPM(HOME) blood pressure measurements
- -sustained HTN :increase anywhere(office(clinic), home, work)
- -WCH: only in the office, so good prognosis hence their clinical visits are infrequent resulting in brief BP increase in their lives.
- - masked HTN: at home only

Hypertension
Complications

End organ damage involves:

- **Heart**
- **Brain**
- **Kidney**
- **Eyes**

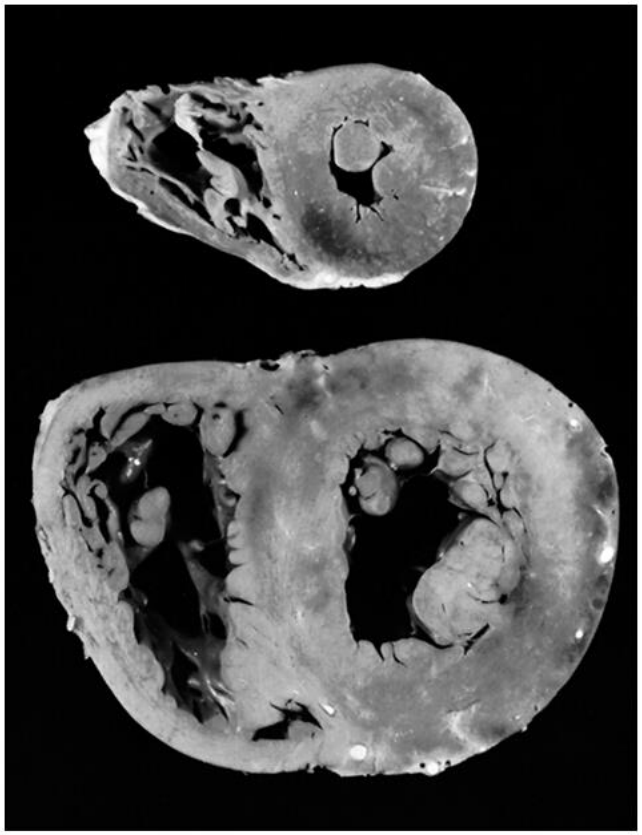
Hypertension
Complications

- **Cardiovascular Disease**

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

Left Ventricular Hypertrophy

Autopsy from a hypertensive patient, who has Lt ventricular hypertrophy
-here, it is concentric hypertrophy, bulging to the the lumen of Lt ventricle



From Kissane JM: *Anderson's pathology*, ed 9, St. Louis, 1990, Mosby.

Fig. 32-3

Hypertension
Complications

- **CNS**

Ischemic stroke

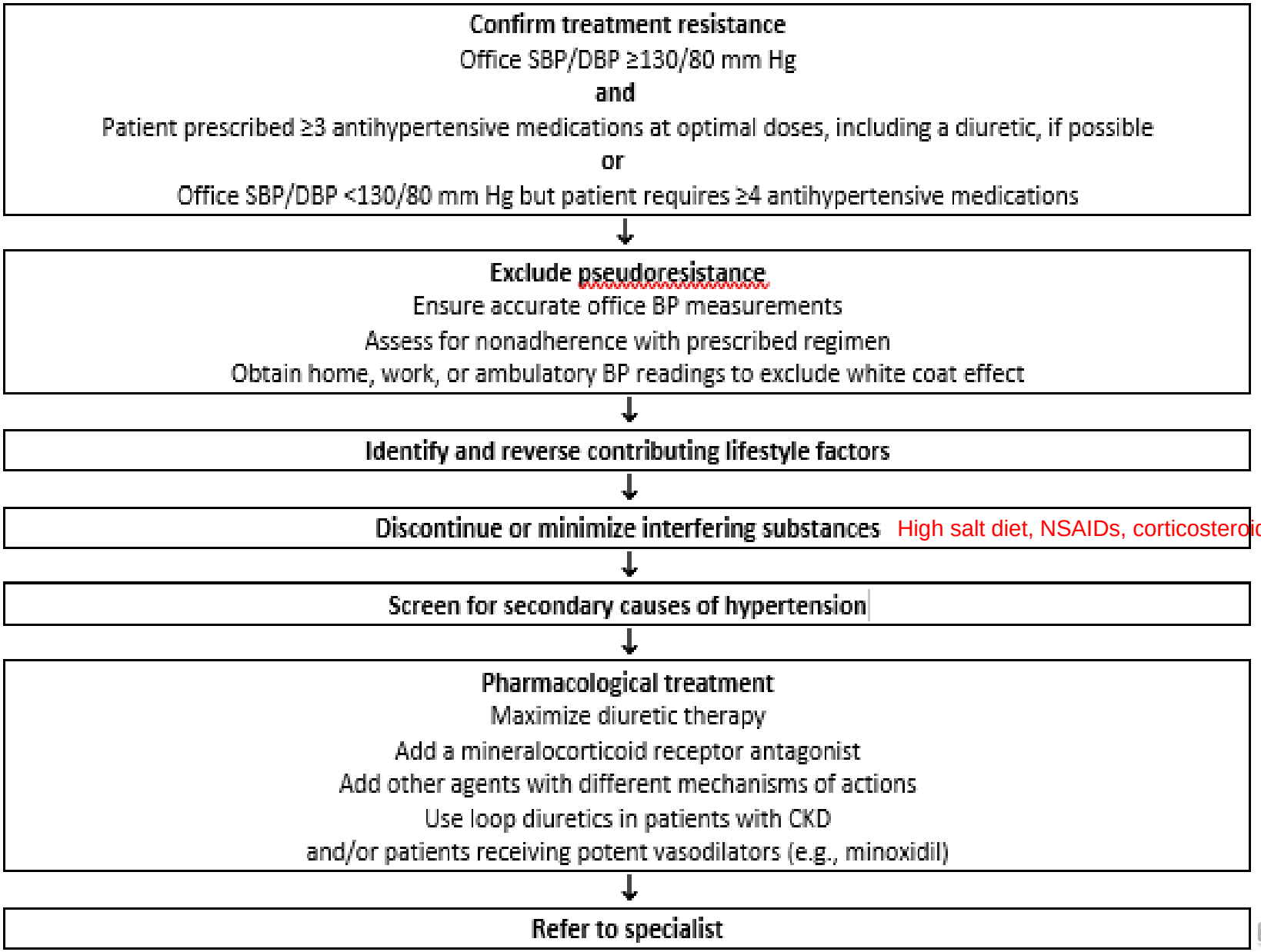
Hemrrhagic stroke

Hypertensive Encephalopathy

-Kidney:
Nephrosclerosis
Major cause for End stage Renal Failure

-Ophthalmic:
Retinal complication including bleeding

Resistant Hypertension: Diagnosis, Evaluation, and Treatment



resistant Htn: persistance of Htn despite medications

* conditions: ① ≥3 different classes of antihypertensives at max dose including a diuretic
② ≥4 antihypertensives needed to control office Htn
* usally 2° Htn if pseudo Htn is excluded
* pharmacological treatment:
① maximize diuretic
② add mineralocorticoid agonist
③ add agents with different MOA
④ use loop diuretics for CKD patients & patients that take potent vasodilators (ex: minoxidil)

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pseudo Htn: is Htn affected by:

① lifestyle (ex: ↑ salt intake)
② if the patient isn't taking perscribed doses
③ if the patient takes medications that interfere with perscribed antihypertensives (ex: NSAIDs, steroids, decongestants)

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Secondary HTN

- “ 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 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

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

Screening

- General principles:
  - New onset HTN if <30 or >50 years of age very young or very old
  - HTN refractory to medical Rx (>3 meds) resistant HTN
  - Specific clinical/lab features typical for certain disease entity:
    - Hypokalemia,
    - Epigastric bruit (turbulence blood flow caused by stenosis, RAS)
    - Differential BP between arm and leg
    - Episodic HTN/flushing/palp, etc, ex:pheochromocytoma

Renal Parenchymal Disease

=chronic kidney disease

- Common cause of secondary HTN
- HTN is both a cause and consequence of renal disease
- -does CKD cause HTN or the opposite?
- Multifactorial cause for HTN including disturbances in Na/water balance, depletion

of vasodilators leading to highTPR

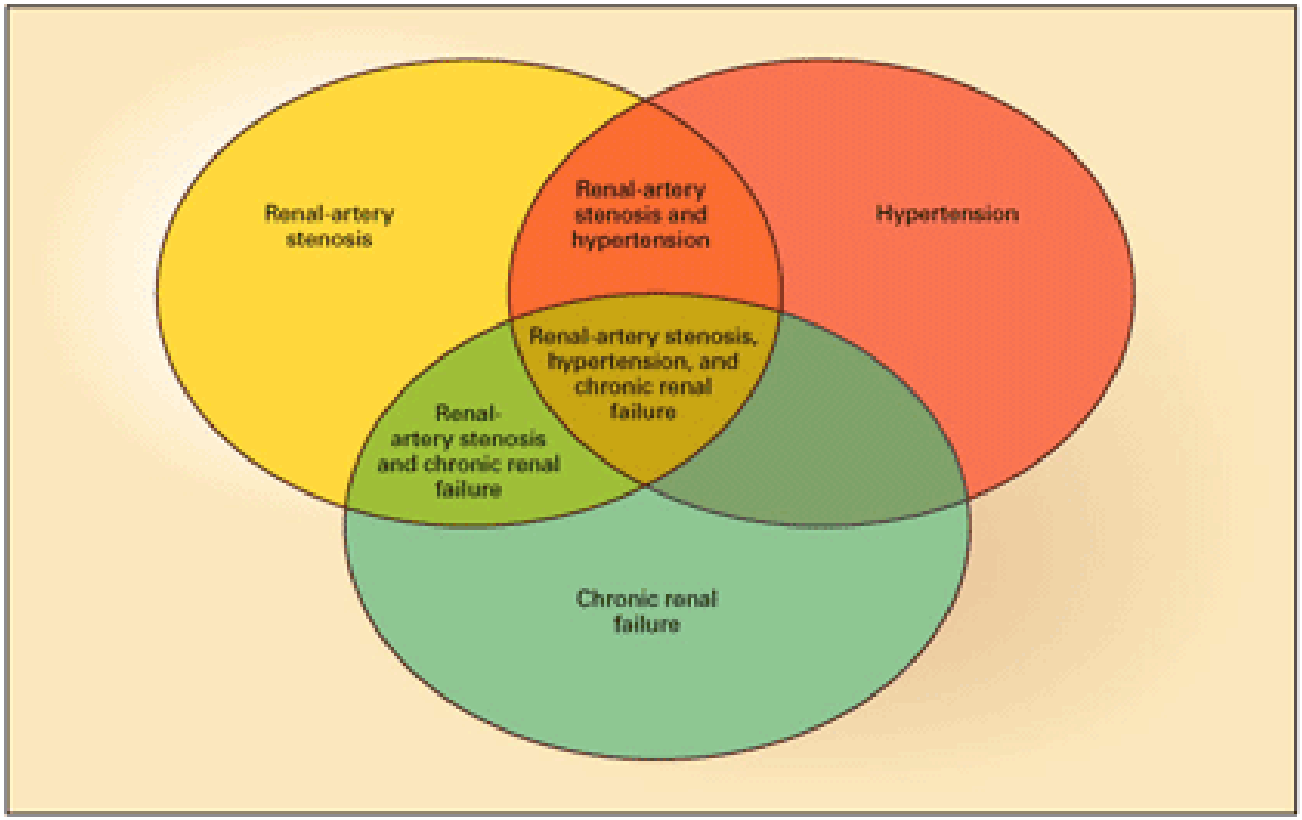
- Renal disease from multiple etiologies, treat underlying disease, dialysis/ transplant if necessary

Renovascular HTN

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

*atheroSclerosis: usually in the proximal segment of renal A (vessel ostium)

* FMD: usually in middle to distal segment of renal A, some lesions develop thicker tunica media (beading pattern)
(في المسبحة: توسع تكيس توسع تكيس ...)



Overlapping between HTN, RAS(renal artery stenosis) and CKD
-CKD is a cause of HTN, also HTN can cause CKD,.
-RAS which is also a cause for 2ry HTN, can lead to ischemia, nephropathy, and CKF.

Safian & Textor. NEJM 344:6;p 432

Renovascular HTN - Pathophysiology

- Decrease in renal perfusion pressure activates RAAS, renin release converts angiotensinogen→ Ang I; ACE converts Ang I→ Ang II
- Ang II (bad player)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(bilateral RAS can lead to pulmonary edema)

Renovascular HTN - Clinical

- 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(normal left ventricular function)
- PE/Lab
  - Epigastric bruit, particulary systolic/diastolic
  - Azotemia induced by ACEI (azotemia=acute kidney injury→sudden creatinine elevation after use of ACEI)
  - Unilateral small kidney→ischemic nephropathy

Renovascular HTN - diagnosis

- Physical findings (bruit)
- Duplex U/S (ultrasound)
- Captopril renography
- CTA (CT scan with angiography)
- MRA (MR scan with angiography)
- Renal Angiography (cath)

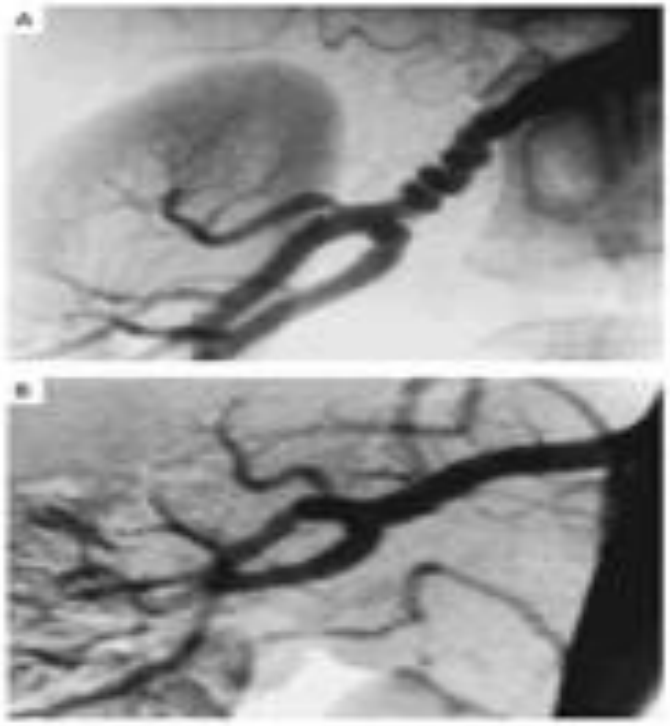
Fibromuscular dysplasia

- 10-25% of all RAS
- Young female, age 15-40
- Medial disease 90% (disease of the medial layer of the vessel), often involves distal RA
- Treatment – PTCA (balloon angiography)
  - Successful in 82-100% of patients
  - Restenosis in 5-11%
  - “Cure” of HTN in ~60%

Atherosclerotic RAS

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

  Stent success 94-100%

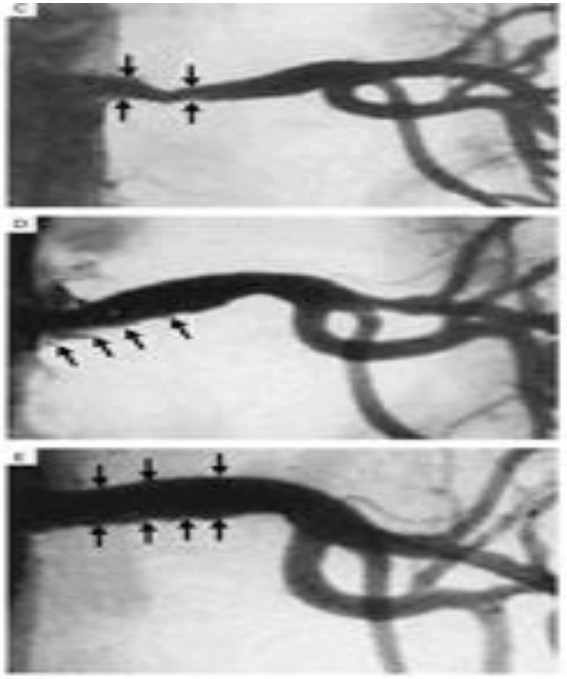


Fibromuscular Dysplasia, before and after PTCA

-blow ballon in vessel to dilate it)

Fig A –beading pattern of distal segment Of RA

-Fig B:AFTER resolution



Atherosclerotic RAS before and after stent

Fig E shows good expansion in the stent

Safian & Textor. NEJM 344:6;

Renovascular HTN – Medical Rx

- Aggressive risk fx modification (lipid, tobacco, etc)
- ACEI/ARB safe in unilateral RAS if careful titration and close monitoring

Treatment is a combination of lifestyle modification with balloon or stent in the RAS

Primary HyperAldosteronism

- Prevalence .5- 2.0%
- Etiology
  - Adrenal adenoma 33%
  - bilateral adrenal hyperplasia 66% (more common)
- Clinical:
  - May be asymptomatic; headache, muscle cramps, polyuria
  - Hypokalemia (K normal in 40%-70%), metabolic alkalosis, high Na (hypokalemia(if symptomatic) is presented in up to 60% of pop)

Primary Aldosteronism- Dx

- Aldosterone / Plasma Renin Activity ratio

  Ratio >20 then we do:

- Confirmatory/physiologic testing (CT images)
  - 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 - Treatment

- 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
- -response to spironolactone gives good implication that the patient will respond to the surgery

Obstructive Sleep Apnea

=especially in obese and with short necks

- Published reports estimate incidence of 30-80% of pt with essential HTN have OSA and 50% pt with OSA have HTN[1]
- Prospective studies show link between OSA (apneic-hyponeic index) and development of HTN independent of other risk fx[2]
- Clinical
  - Daytime somnolescence, am headaches, snoring or witnessed apneic episodes
- Dx – Sleep studies sleep lab, Apneic hyponeic index)
- Rx – wt loss, CPAP, surgical (CPAP: continuous positive airway pressure for severe OSP)

Pheochromocytoma

- 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 tyramine-containing foods (beer,cheese,wine), pain, trauma, drugs (clonidine, TCA, opiates)

Pheochromocytoma – Screen

=for serum or urine metanephrine, or VMA(Vanillmandelic acid)

- 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%
CT	98%	70%	69%	98%
MRI	100%	67%	83%	100%

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

Pheochromocytoma - treatment

- 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)

Cushing's syndrome/ hypercortisolism

- 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

Cushings syndrome - dx

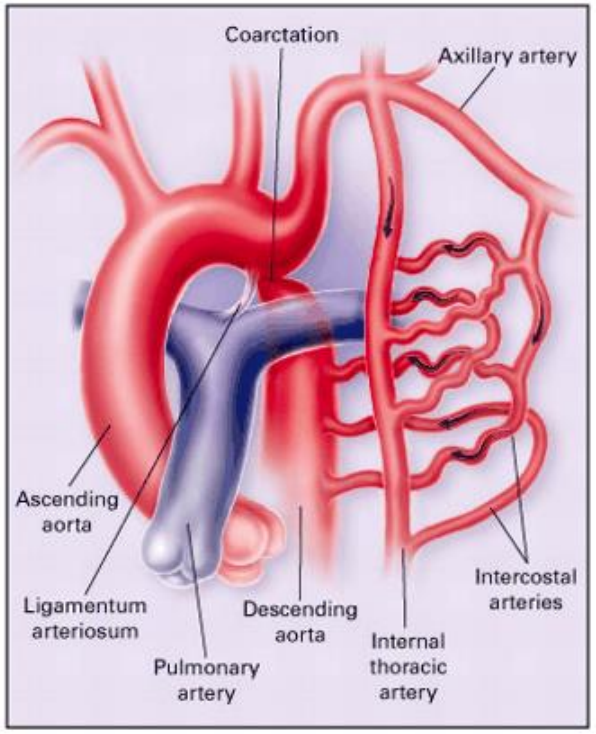
- Screen:
  - 24 Hr Urine free cortisol
- Confirm
  - Low dose dexamethasone suppression test
  - 1mg dexameth. midnight, measure am plasma cortisol
- Imaging
  - CT/MRI head (pit) chest (ectopic ACTH tumor)

Cushings syndrome - Rx

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

Coarctation of Aorta

- 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
- -POOR PROGNOSIS IF UNTREATED



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 (HTN+other diseases)*
    - *Use agent(s) that concurrently lower BP (e.g. post-MI, SIHD, HF)*
  - *No compelling indication (only HTN)*
    - *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)*
      - *-WE CAN DO DRUGS COMBINATION EXCEPT BETWEEN ARBS and ACEIs to prevent hyperkalemia*

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 (mineralocorticoid receptor antagonist (MRA or MCRA) or aldosterone antagonist)
- *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 and CCBs*
- *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**

Case 1

- 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 ?
- -Chest CT , to find out the coarctation segment.

Case 2

- 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?
- -severely low K (fatigue)+severe HTN=hyperaldosteronism)
- The next step is to perform the renin aldosterone ratio, and if it is>20, we do imagery confirmatory tests.

Case 3

- 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?
- -for serum or urine metanephrine, or VMA(Vanillmandelic acid), then
- Imagery confirmatory tests

Case 4

- 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(epigastric) bruit.
- What is the next step in your evaluation?
- What is your diagnosis?
- -RAS, it has 2 types(atherosclerosis and FMA), this case is more likely atherosclerosis, testing by ultrasound especially in obese, or cath(renal angiogram)

Case 5

- 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 ?
- -it is hypertensive emergency, (BP>180/120), +end organ damage
- -here there is vascular damage in thoracic aorta→aortic dissection
- -with disciplinary between the right and left upper extremities, confirmed by CTA, or transesophageal echo(if the patient is unstable)

Patient comes with a pressure 149/70 mmHg, what is his blood pressure according to ACC/AHA guidelines?

a) Stage 1
b) Stage 2
c) Stage 3
d) Stage 4
e) Unclassified hypertension

Thank You