Hypertension

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Hypertension
Definition

- Hypertension is sustained elevation of BP
  - Systolic blood pressure ≥ 140 mm Hg
  - Diastolic blood pressure ≥ 90 mm Hg

Factors Influencing Blood Pressure

Blood Pressure = Cardiac Output x Systemic Vascular Resistance

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

Factors Influencing BP

- Hear rate
- Sympathatic/Parasympathatic
- Vasoconstriction/vasodilation
- Fluid volume
  - Renin-angiotensin
  - Aldosterone
  - ADH

Blood Pressure Classification

BP Classification	SBP mmHg		DBP mmHg
Normal	< 120	and	< 80
Pre-hypertension*	120-139	or	80-89
Stage 1 Hypertension	140-159	or	90-99
Stage 2 Hypertension	≥ 160	or	≥ 100
*newly recognized, requiring lifestyle modifications			

Blood Pressure Classification

BP Classification	SBP mmHg		DBP mmHg
Normal	< 120	and	< 80
Pre-hypertension* Elevated blood pressure	120-139 120-129	or	80-89 <80
Stage 1 Hypertension	140-159 130-139	or	90-99 80-89
Stage 2 Hypertension	≥ 160 ≥140	or	≥ 100 ≥ 90

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

Risk Factors for Primary Hypertension

- 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 $\geq$ 30)
- Ethnicity (African Americans)
- Sedentary lifestyle
- Socioeconomic status
- Stress

Hypertension
Clinical Manifestations

- Frequently asymptomatic until severe and target organ disease has occurred
  - Fatigue, reduced activity tolerance
  - Dizziness
  - Palpitations, angina
  - Dyspnea

Hypertension
Complications

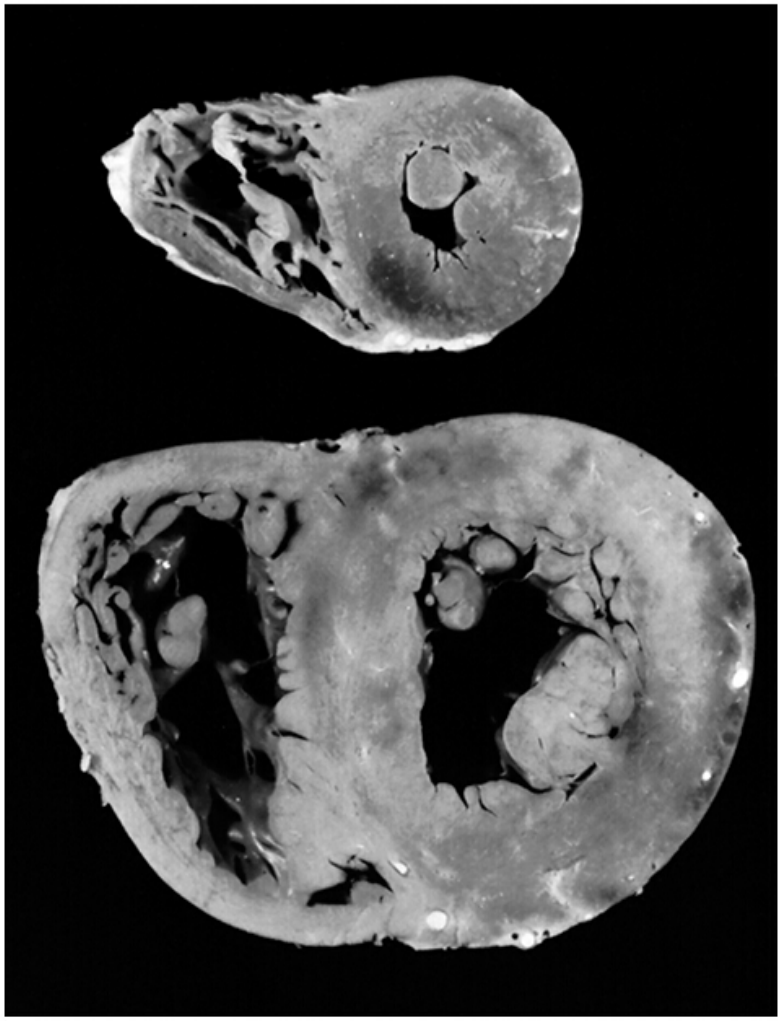
The common complications are target organ diseases occurring in the

- Heart
- Brain
- Kidney
- Eyes

Hypertension
Complications

- **Hypertensive Heart Disease**
  - Coronary artery disease
  - Left ventricular hypertrophy
  - Heart failure

Left Ventricular Hypertrophy



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

Fig. 32-3

Hypertension
Complications

- Cerebrovascular Disease
  - Stroke
- Peripheral Vascular Disease
- Nephrosclerosis
- Retinal Damage

Hypertension

- **Starting at 115/75 mmHg, CVD risk doubles with each increment of 20/10 mmHg throughout the BP range**
- Ambulatory BP Monitoring
  - For “white coat” phenomenon, hypotensive or hypertensive episodes, apparent drug resistance

Benefits of Lowering BP

	Average Percent Reduction
Stroke incidence	35–40%
Myocardial infarction	20–25%
Heart failure	50%

Treatment Goals

- **Goal** is to reduce overall cardiovascular risk factors and control
  - Diabetes, chronic kidney disease, and every one less than age 60 BP < 140/90 (<130/85)
  - For those 60 years and older < 150/90 (<140/90)

Hypertension

- **Lifestyle Modifications**

  - Weight reduction
  - Dietary changes (DASH diet)
  - Limitation of alcohol intake (≤ 2 drinks/day for men;
    ≤ 1/day for women)
  - 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

Hypertension

- Drug Therapy
  - Reduce Systemic Vascular Resistence
  - Decrease volume of circulating blood

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

Screening

- Testing can be expensive and requires clinical suspicion and knowledge of limitations of different tests
- General principles:
  - New onset HTN if <30 or >50 years of age
  - HTN refractory to medical Rx (>3-4 meds)
  - Specific clinical/lab features typical for disease
    - i.e., hypokalemia, epigastric bruits, differential BP between arm and leg, episodic HTN/flushing/palp, etc

Causes of Secondary HTN

- Common
  - Intrinsic Renal Disease
  - Renovascular Dz
  - Mineralocorticoid excess/ aldosteronism
  - OSA
- Uncommon
  - Pheochromocytoma
  - Glucocorticoid excess/ Cushing's dz
  - Coarctation of Aorta
  - Hyper/hypothyroidism

Renovascular HTN

- 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 - 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
- PE/Lab
  - Epigastric bruit, particulary systolic/diastolic
  - Azotemia induced by ACEI
  - Unilateral small kidney

Renovascular HTN - diagnosis

- Physical findings (bruit)
- Duplex U/S
- Magnetic Resonance Angiography or CTA
- Renal Angiography

Renovascular HTN – Medical Rx

- Aggressive risk fx modification (lipid, tobacco, etc)
- ACEI/ARB safe in unilateral RAS :careful titration and close monitoring
- ACEI/ARB Contraindicated in bilateral RAS or solitary kidney RAS

Primary Aldosteronism

- Prevalence .5- 2.0% (5-12% in referral centers)
- Etiology
  - Adrenal adenoma 33%
  - bilat adrenal hyperplasia 66%
- Clinical:
  - May be asymptomatic; headache, muscle cramps, polyuria
  - Retinopathy, edema uncommon
  - Hypokalemia (K normal in 40%-70%), metabolic alkalosis, high Na

Primary Aldosteronism - Treatment

- Surgical removal of adrenal tumor, can be done laparoscopically
- Pretreatment for 3-4 wks with spironolactone

Obstructive Sleep Apnea

- 30-80% of pt with essential HTN have OSA
- 50% pt with OSA have HTN[1]
- Clinical
  - Daytime somnolescence, am headaches, snoring or witnessed apneic episodes
- Dx – Sleep studies
- Rx – wt loss, CPAP, surgical

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 - treatment

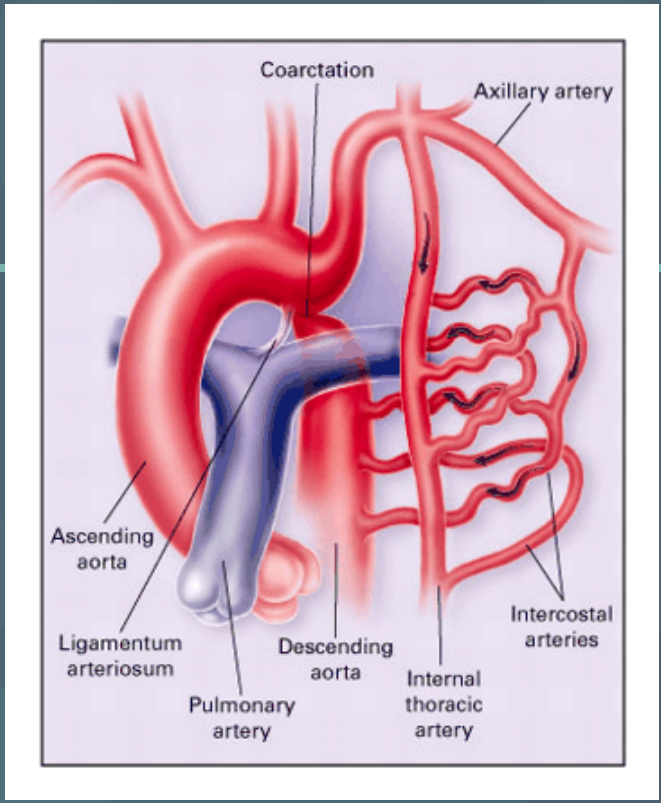
- Surgical removal of tumor
- Caution with BB – can cause unopposed alpha stimulation/pheo crisis
- BP control with alpha blockers (phentolamine, phenoxybenzamine, and prazosin) plus BB

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

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



Hyperthyroidism

- 33% of thyrotoxic pt develop HTN
- Usually obvious signs of thyrotoxicosis
- Dx: TSH, Free T4/3, thyroid RAIU
- Rx: radioactive ablation, propanolol

Hypothyroidism

- 25% hypothyroid pt develop HTN
- Mechanism mediated by local control, as basal metabolism falls so does accumulation of local metabolites; relative vasoconstriction ensues

Conclusions

- Remember clinical/diagnostic features of common forms of secondary HTN
- Important to appropriately screen pt suspected of having potentially correctable causes of HTN
- Understand limitations of screening/treatment (atherosclerotic RAS)

QUESTIONS

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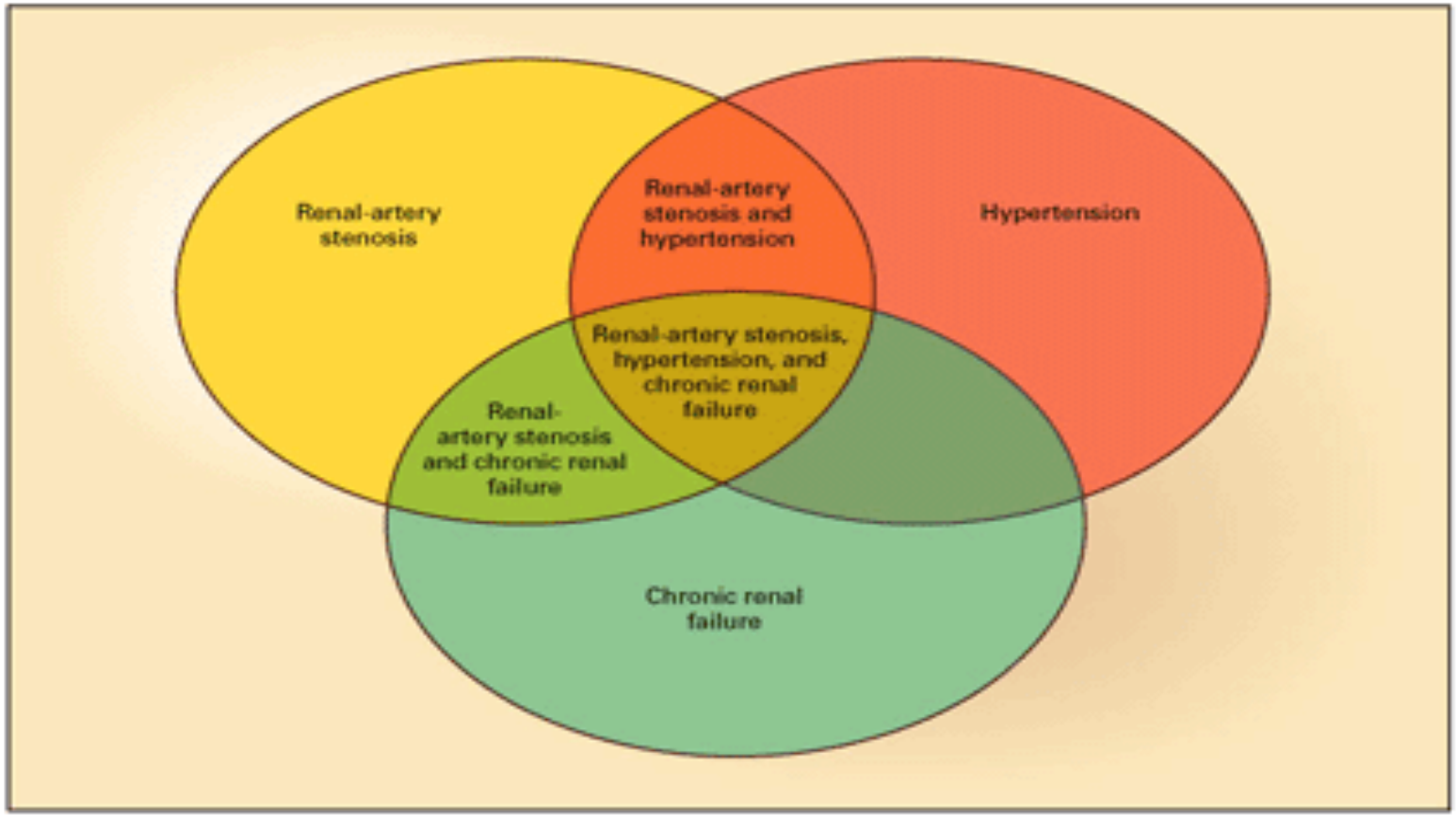
Renal Parenchymal Disease

- 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 or antagonism of vasodepressors/ prostaglandins, pressor effects on TPR
- Renal disease from multiple etiologies, treat underlying disease, dialysis/ transplant if necessary

Renovascular HTN



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 causes vasoconstriction (among other effects) which causes HTN and enhances adrenal release of aldosterone; leads to sodium and fluid retention
- Contralateral kidney (if unilateral RAS) responds with diuresis/ Na, H_2O excretion which can return plasma volume to normal
- with sustained HTN, plasma renin activity decreases (limited usefulness for dx
- Bilateral RAS or solitary kidney RAS leads to rapid volume expansion and ultimate decline in renin secretion

RAS screening/diagnostics

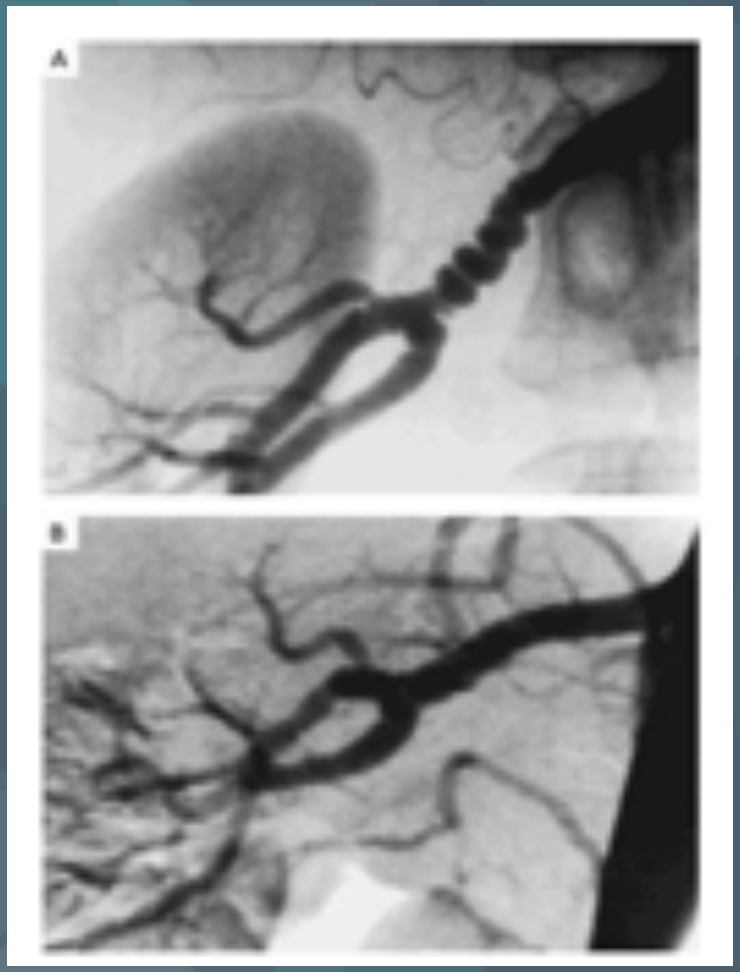
	Sens	Spec	Cost	Limitation/Etc
Duplex U/S	90-95%	60-90%	$117	Operator dependent, 10-20%
Captopril Renography	83-91%	87-93%	$968	Meds, accuracy reduced in pt with renal insufficiency, lacks anatomical info; good predictor of BP response
MRA	88-95%	95%	$572 ?	False positive artifact resp, peristalsis, tortuous vessels; cost
Bruit	39-65%	90-99%	-	Insensitive, severe stenosis may be silent
Angiography	Gold std	Gold std	?	Invasive, nephrotoxicity, little value in predicting BP response

Fibromuscular dysplasia

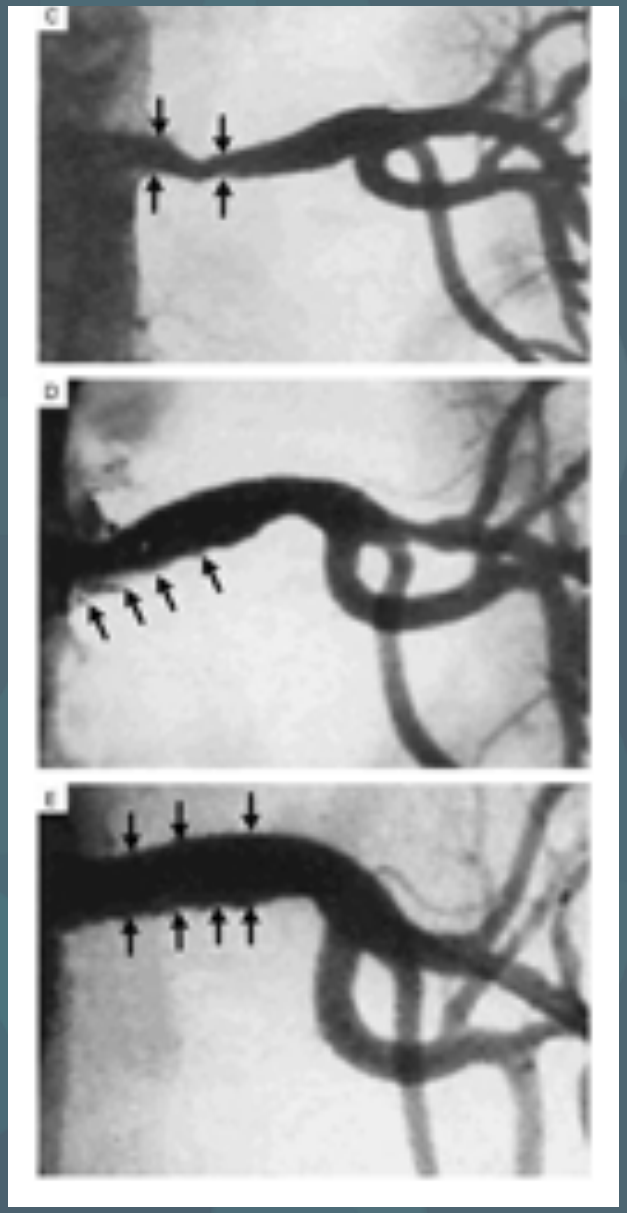
- 10-25% of all RAS
- Young female, age 15-40
- Medial disease 90%, often involves distal RA
- ~ 30% progressively worsen but total occlusion is rare
- Treatment – PTRA
  - Successful in 82-100% of patients
  - Restenosis in 5-11%
  - “Cure” of HTN in ~60%

Atherosclerotic RAS

- 75-90% of RAS
- Usually men, age>55, other atherosclerotic dz
- Progression of stenosis 51% @ 5years, 3-16% to occlusion, with renal atrophy noted in 21% of RAS lesions >60%
- ESRD in 11% (higher risk if >60%, baseline renal insufficiency, SBP>160)
- Treatment
  - PTRA success 60-80% with restenosis 10-47%
  - Stent success 94-100% with restenosis 11-23% (1yr)
  - "Cure" of RV HTN <30%



Fibromuscular Dysplasia, before and after PTRA



Atherosclerotic RAS before and after stent

Safian & Textor. NEJM 344:6;

Renovascular HTN - principles

- Not all RAS causes HTN or ischemic nephropathy
- Differing etiology of RAS has different outcomes in regards to treatment (FMD vs atherosclerosis)
- No current rationale for “drive-by” interventions
- Importance of medical rx

Primary Aldosteronism- Dx

- Aldosterone / Plasma Renin Activity ratio
  - Early am after ambulation ~10-15 min
  - Ratio >20-25 with PRA <1 and Aldo >15 should prompt further testing, endo referral
- Confirmatory/physiologic testing
  - Withold BP meds 2wks
  - High serum aldo after IV saline (1.25L x 2hr) load followed by low PRA after salt restricted diet (40mg/d) or diuretic (lasix up to 120mg)
  - serum aldo <8.5 ng/dL after IV saline rules out primary aldosteronism
  - Imaging – CT, scintography

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

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

Cushings syndrome - dx

- Screen:
  - 24 Hr Urine free cortisol
  - >90ug/day is 100% sens and 98% spec
  - false + in Polycystic Ovarian Syndrome, depression
- Confirm
  - Low dose dexamethasone suppression test
  - 1mg dexameth. midnight, measure am plasma cortisol (>100nmol is +)
  - Other tests include dexa/CRH suppresion test
- 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

Hypertension

- **For persons over age 50, SBP is more important than DBP as a CVD risk factor**
- **Starting at 115/75 mmHg, CVD risk doubles with each increment of 20/10 mmHg throughout the BP range**
- Ambulatory BP Monitoring
  - For “white coat” phenomenon, hypotensive or hypertensive episodes, apparent drug resistance

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

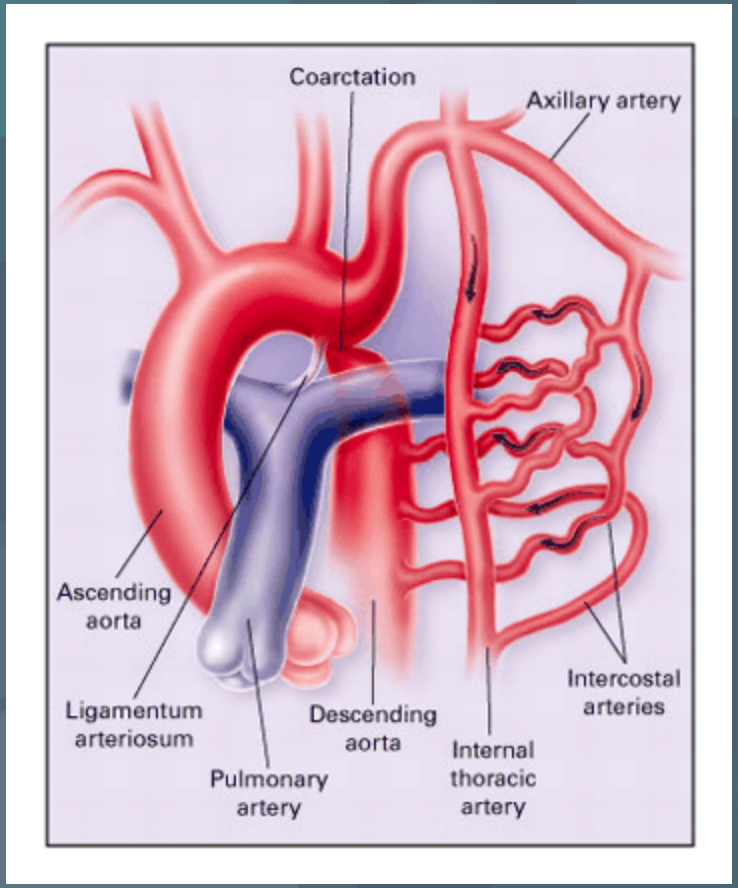
Obstructive Sleep Apnea

- 30-80% of pt with essential HTN have OSA
- 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
- Rx – wt loss, CPAP, surgical

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)

Coarctation of Aorta



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