Hypertension

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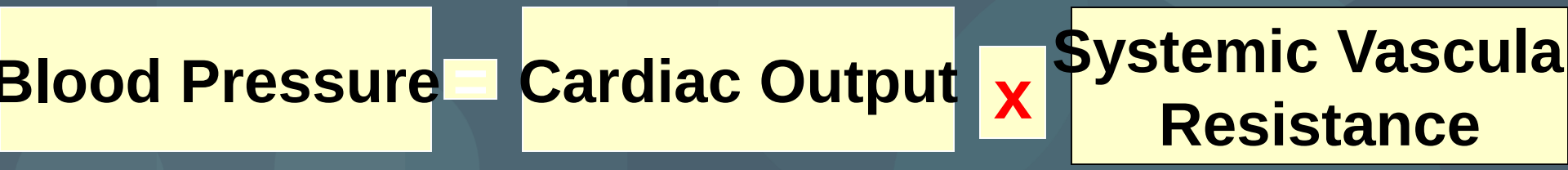
Hypertension
Definition

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

ACC/AHA 2017 guidelines

- Normal <120/80 mmHg
- Elevated 120-129/80-89mmHg
- Stage 1 130-139/90-99mmHg
- Stage 2 ≥140/100mmHg

Factors Influencing Blood Pressure



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

JNC VII
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		

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

- Asymptomatic
- Non-specific symptoms

Fatigue

Reduced activity tolerance

Dizziness

Palpitations

- End organ damage

Hypertension
Complications

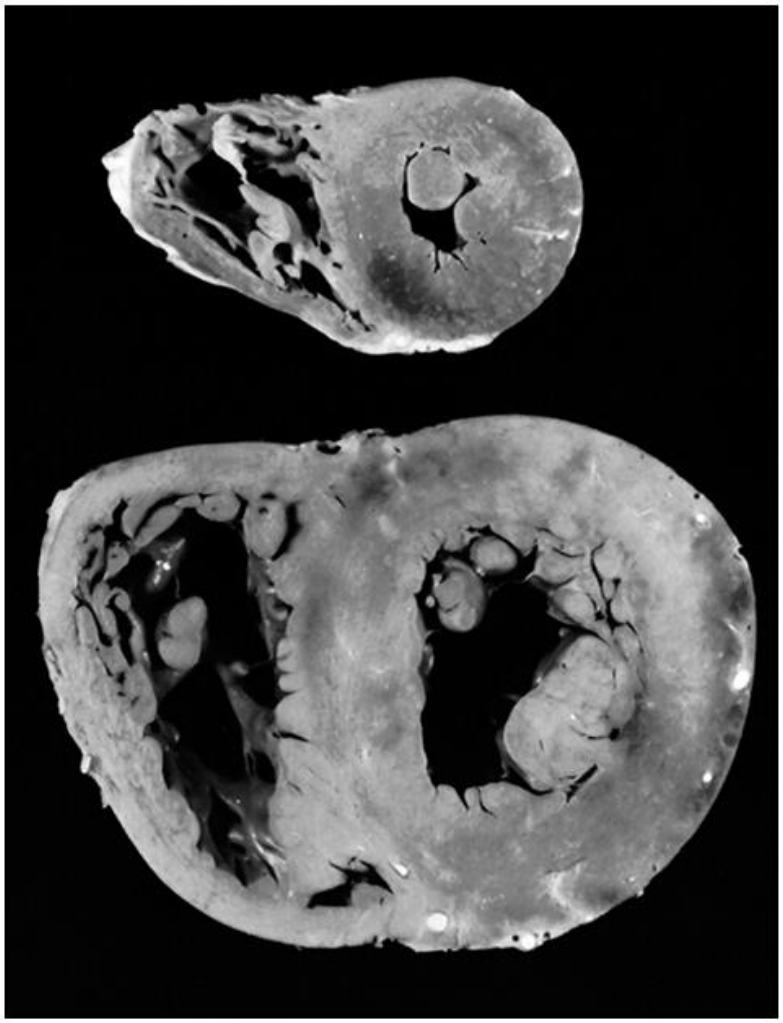
End organ damage involves:

- **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.

Fig. 32-3

Hypertension
Complications

- CNS

Ischemic stroke

Hemrrhagic stroke

Hypertensive Encephalopathy

Hypertension
Complications

- Kidney:

Nephrosclerosis

Major cause for End stage Renal Failure

- Ophthalmic:

Retinal complication including bleeding

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

Benefits of Lowering BP

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

Hypertension

- **Lifestyle Modifications**

  - Weight reduction
  - 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

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

- 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

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

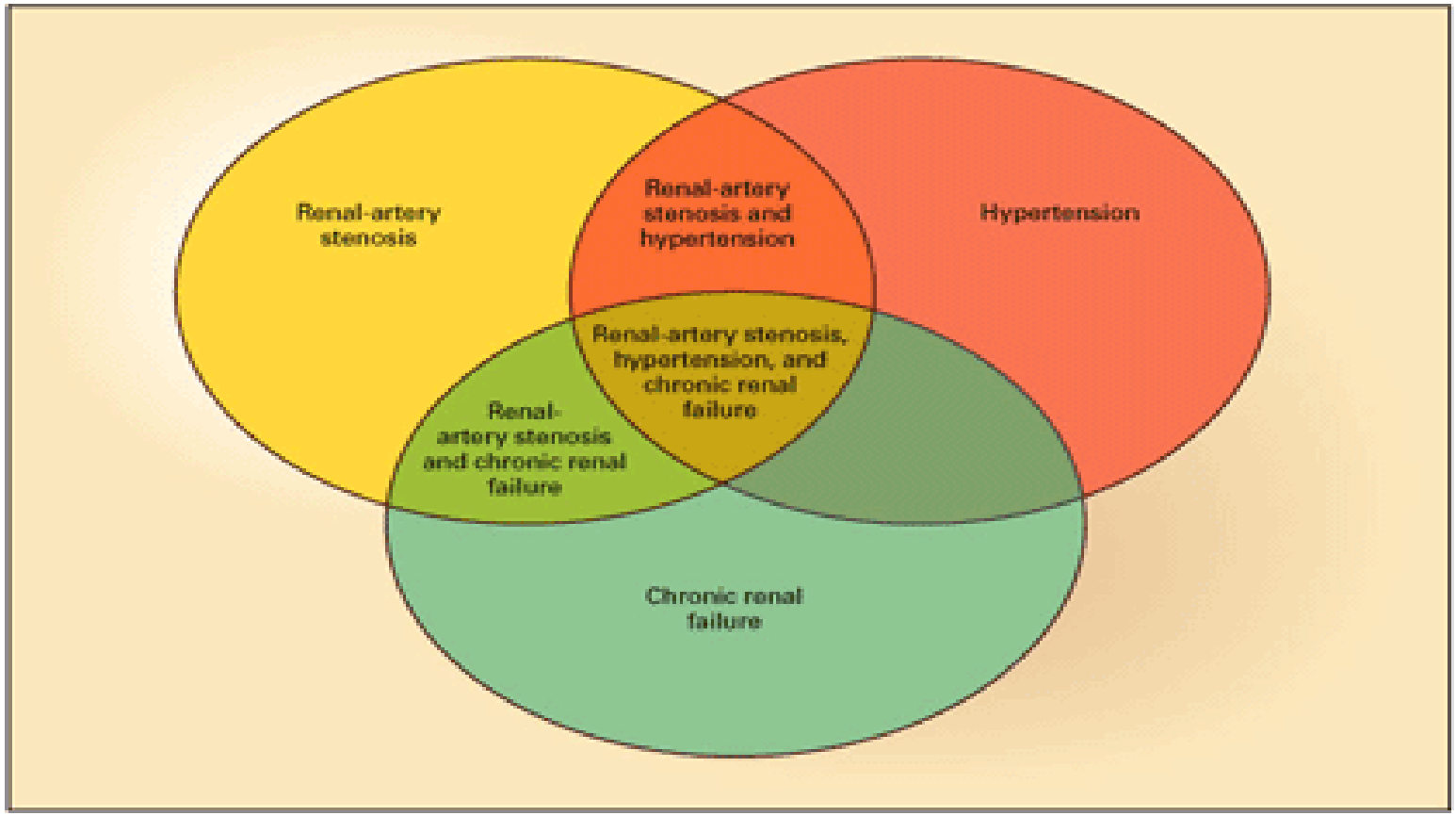
Renal Parenchymal Disease

- 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

- 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

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
- Bilateral RAS or solitary kidney RAS leads to rapid volume expansion and ultimate decline in renin secretion

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
- Captopril renography
- Magnetic Resonance Angiography
- Renal Angiography

RAS screening/diagnostics

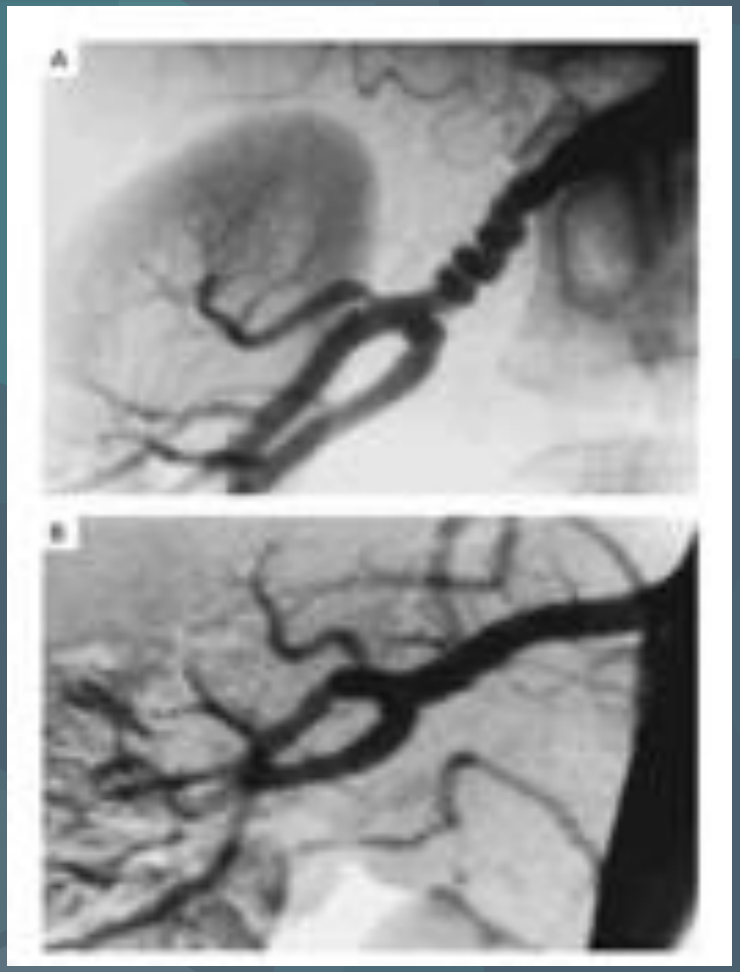
	Sens	Spec	Cost	Limitation/Etc
Duplex U/S	90-95%	60-90%	$117	Operator dependent, 10-20%
Captopril Renography	83-91%	87-93%	$968	Meds, accuracy reduced in pt with renal insufficiency, lacks anatomical info; good predictor of BP response
MRA	88-95%	95%	$572 ?	False positive artifact resp, peristalsis, tortuous 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

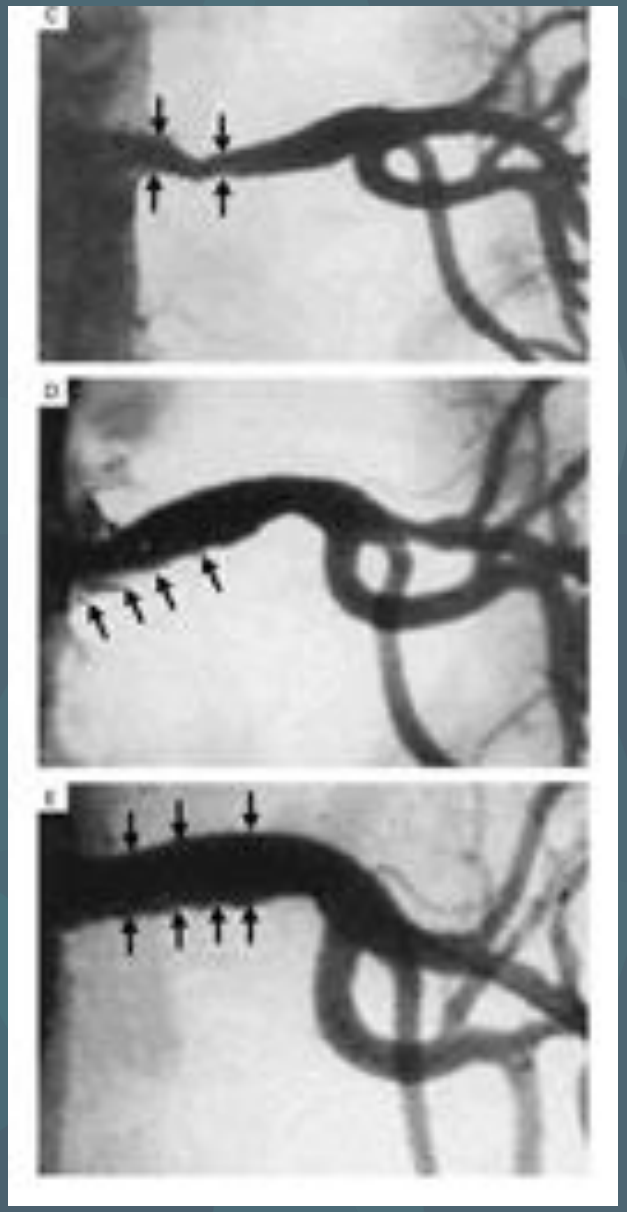
- 10-25% of all RAS
- Young female, age 15-40
- Medial disease 90%, often involves distal RA
- ~ 30% progressively worsen but total occlusion is rare
- Treatment – PTRA
  - Successful in 82-100% of patients
  - Restenosis in 5-11%
  - “Cure” of HTN in ~60%

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 – Medical Rx

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

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

- 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- 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
  - serum aldo <8.5 ng/dL after IV saline rules out primary aldosteronism
  - Imaging – CT, scintography

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

- 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
- 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 - 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

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
  - >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)

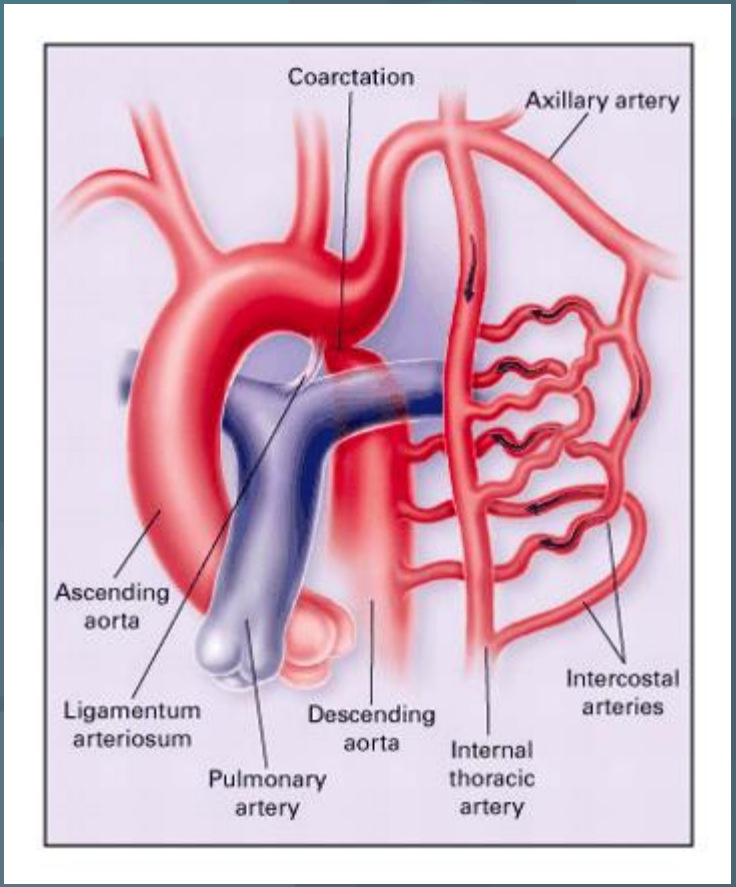
Cushings syndrome - Rx

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

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

Coarctation of Aorta



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

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)

HYPERTENSIVE EMERGENCIES

DEFINITIONS

- Systolic blood pressure >180 and diastolic >120mmHg.
- -**HYPERTENSIVE EMERGENCY**: also called hypertensive crisis, is severe hypertension with acute impairment of an organ

MALIGNANT HYPERTENSION: papilloedema present

ACCELERATED HYPERTENSION: No papilloedema

-**HYPERTENSIVE URGENCY**: the BP is a potential risk but has not yet caused acute end-organ damage.

ETIOLOGY

- **Essential hypertension** : Inadequate blood pressure control and noncompliance are common precipitants
- Renovascular
- Eclampsia/pre-eclampsia
- Acute glomerulonephritis
- Pheochromocytoma
- Anti-hypertensive withdrawal syndromes
- Head injuries and CNS trauma

Etiology

- Renin-secreting tumors
- Drug-induced hypertension
- Burns
- Vasculitis
- TTP
- Post-op hypertension
- Coarctation of aorta

PATHOPHYSIOLOGY

NORMAL AUTOREGULATION

RISE IN BP

↓

ARTERIAL AND ARTERIOLAR CONSTRICTION

↓

Normal flow.(flow=P/r)

AUTOREGULATION FAILURE

RISE IN BP

↓

FAILURE OF VASO CONSTRICTION

↓

ENDOTHELIAL DAMAGE

(due to shear stress on the wall)

PATHOPHYSIOLOGY

- BP=PVR*CO(SV*HR)
- Rate at which MAP rises more important than absolute rise.

Acute rise in BP → Failure of vasoconstriction by autoregulation → Endothelial damage → Depsn. of proteins/ fibrinogen in vessel wall → Activates coagn and inflammation → FIBRINOID NECROSIS

- RAAS plays an important role in initiating and perpetuating BP rise by causing vasoconstriction and fluid retention.

CENTRAL NERVOUS SYSTEM

- CENTRAL NERVOUS SYSTEM:
- ⇑ BP ⇒ Loss of arteriolar control over vasoconstriction and autoregulation of CBF

⇒ transudate leak across capillaries and continued arteriolar damage and subsequent fibrinoid necrosis

It causes normal autoregulatory mechanisms to fail, leading to clinically apparent papilledema, the sine qua non of malignant hypertension. The end result of loss of autoregulation is hypertensive encephalopathy.

CARDIOVASCULAR SYSTEM

- The cardiovascular system is affected as increased cardiac workload leads to cardiac failure; this is accompanied by pulmonary edema, myocardial ischemia, or myocardial infarction and aortic dissection.

RENAL SYSTEM

- The renal system is impaired when high BP leads to arteriosclerosis, fibrinoid necrosis, and an overall impairment of renal protective autoregulation mechanisms. This may manifest as worsening renal function, hematuria, red blood cell (RBC) cast formation, and/or proteinuria.

EPIDEMIOLOGY

- **In the US:** More than 60 million Americans, about 25-30% of the population, have hypertension. Of these individuals, 70% have mild disease, 20% moderate, and 10% severe hypertension (diastolic BP [DBP] >110 mm Hg). Approximately 1-2% develop a hypertensive emergency with end-organ damage.
- **Mortality/Morbidity:** Morbidity and mortality depend on the extent of end-organ damage on presentation and the degree to which BP is controlled subsequently. BP control may prevent progression to end-organ impairment. I yr mortality in untreated pts. >90%.

EPIDEMIOLOGY

- **Race:** African Americans have a higher incidence of hypertensive emergencies than Caucasians.
- **Sex:** Males are at greater risk of hypertensive emergencies than females.
- **Age:**Most commonly in middle-aged people.Peak age:40-50yrs.

HISTORY

- **Focus on circumstances surrounding hypertension & etiology :**

  -Medications: esp. hypertensive drugs/their compliance,illicit drugs

  -Duration of hypertension

  -Duration of current symptoms

  -Date of LMP

  -Other medical problems:prior hypertension,thyrotoxicosis,Cushing's,SLE, renal disease

HISTORY

- **Focus on complications :**

  -CNS:headaches,blurred vision,wt. loss,nausea,vomiting,weakness,fatigue, confusion and mental status changes.

  -CVS:symptoms of CHF,angina,dissection,SOB

  -Renal:hematuria,oliguria.

PHYSICAL

- Use an approach based on organ systems to identify signs of end-organ damage

  -CNS: focal neuro deficits,seizures,stupor,coma, papilledema, hemorrhages, exudates

CVS:JVD,lung auscultaion for crackles,peripheral edema,extra heart sounds, equal and symmetric BP and pulses bilaterally.

  -Check for abdominal masses and bruits.

Work-up

- CBC,Chem 8
- Urinanalysis:hematuria,proteinuria,RBCs,RBC casts.
- Toxicology,pregnancy,endocrine causes.
- Imaging:Chest X-ray,Head CT,Chest CT,aortic angiogram
- ECG,cardiac enzymes

TREATMENT

- Weight risks of decreasing end-organ perfusion v.s benefits. Important steps include:

  -Appropriately evaluating patients with an elevated BP

  -Correctly classifying the hypertension

  -Determining aggressiveness of therapy

  An important point to remember in the management of the patient with any degree of BP elevation is to "treat the patient and not the number."

Treatment

- Initial considerations: Place patient who is not in distress in a quiet room and reevaluate after an initial interview. Consider the context of the elevated BP (eg, severe pain)
- Screen for end-organ damage- Patients with end-organ damage usually require admission and rapid lowering of BP using iv meds.Suggested meds depend on the end-organ system damaged.

Treatment

- Patients without evidence of end-organ effects may be discharged with follow–up.It is a misconception that a patient should not be discharged from the ER with elevated BP.Giving oral meds such as nifedipine to rapidly lower BP may be dangerous as the BP may have been elevated for sometime and there may be organ hypoperfusion.Acute control has not improved long term mortality and morbidity rates.

DRUGS

- Once the diagnosis of hypertension is made and end-organ damage confirmed,the BP should be lowered by about 25% of the mean arterial pressure in 4-6 hours.
- There are 2 main classes of drugs:

  -Vasodilators

  -Adrenergic inhibitors

VASODILATORS

DRUG	DOSAGE	ONSET/DUR	ADV.EFFE
Nitroprusside	0.25-10mcg/kg/min	Instant/1-2min.	Thiocyanate,cyanide poisoning
Nitroglycerine	5-100mcg/min	1-5min/3-5min	Flushing,headache,methemoglobin
Nicardipine	5-15mg/hr	5-10min/1-4hr	Tachycardia,flushing.avoid-heart failure
Hydralazine	10-20mg	5-15min/3-8hr	Flushing,tachy,avoid-A.diss,MI
Enalapril	10-40mg IM,1.25-5MG1Vq6hr	20-30min/6hr	Hypotension,renal failure,hyperkalemia
Fenoldopam	0.1-0.3mcg/kg/min	5min/10-15min	Flushing,headache,tachy

ADRENERGIC INHIBITORS

DRUG	DOSAGE	ONSET/DUR	ADV.EFF
Labetalol (a+b blocker)	20-80mgiv bolus every 10 min,2mg.min iv infusion	5-10min/3-6hrs	Heart block,ortho hypotension.avoid-heart failure,asthma
Esmolol (b-1 selective blocker)	200-500 mcg/kg/min for 4min,then 150-300mcg/kg/min	1-2min/10-20min	Hypotension,avoid-heart failure,asthma
Phentolamine (a1 blocker)	5-15mg iv	1-2min/3-10min	Tachycardia,flushing ,headache

ORAL DRUGS

DRUG	DOSAGE	ONSET/DURATION	ADV. EFF.
CAPTOPRIL (ACE inhibitor)	6.25-25MG q 6hrs.	15-30min/6 hrs.	Hypotension in high renin states
CLONIDINE (a2 agonist-centrally acting)	0.1-0.2 mg hrly, Upto max 0.8mg in 24hrs.	30-60min/6-12hrs.	Sedation,bradycardia,dry mouth

RAPID BP REDUCTION

- Acute myocardial ischemia:

  IV NTG,b-blockers,ACE inhibitors

- CHF with pulmonary edema:

  IV NTG,furosemide,morphine

- Acute aortic dissection:

  IV nitroprusside and b-blockers

RAPID BP REDUCTION

- Hypertensive encephalopathy ,ischemic or hemorarhgic stroke sub-arachnoid hemorrhage:

  IV labetalol, nicardipine

- Cocain, Pheochromocytoma MAO-tyramine interactions with acute hypertension:

  phentolamine and benzodiazepine.

SPECIFIC TREATMENT

- **Hypertensive Encephalopathy:** Goal is to reduce MAP by no more than 25% over 8 hours **Labetalol,fenoldopam** .
- **Intracerebral Hemorrhage:** CPP=MAP-ICP.As ICP rises,MAP must rise for perfusion but this raises risk of bleeding from small arteries and arterioles. guidelines:If there are signs of increased ICP decrease when MAP>130 or SBP>220.**Labetalol,esmolol** agents of choice.

SPECIFIC TREATMENT

- **SAH: Nimodipine** decreases vasospasm that occurs due to chemical irritation of arteries by blood.Labetalol,esmolol agents of choice.Maintain SBP < 160mmHg

- **Acute Ischemic Stroke:** High BP can cause hemorrhagic transformation of infarct ,cerebral edema..AHA guidelines:BP be reduced only if SBP>220 .**Labetalol,nicardipine**-agents of choice.For thrombolysis,BP<185/110.

Specific Treatment

- **Aortic dissection:** Immediate redn. In BP and mainly,shear stress is essential to limit the extension of damage as surgery is being considered.**Labetalol or nitroprusside+b-blocker like propranolol** agents of choice.

- **MI: NTG,b-blockers,ACE inhibitors.**

Specific Treatment

- Acute LVF: usually associated with pulm edema and diastolic/systolic dysfx.

  IV nitroprusside,NTG agents of choice.Titrate until BP controlled and signs of heart failure alleviated.

- Renal insufficiency: is a cause and effect of high BP.Goal is to prevent further renal damage by maintaining adequate blood flow.**Nitroprusside** effective.

COMPLICATIONS

- CHF
- Myocardial infarction
- Renal failure
- Retinopathy
- CVA
- Abrupt lowering of the BP may result in inadequate cerebral or cardiac blood flow leading to stroke or myocardial infarction.

If untreated mortality is 90% at one year.

HTN-JNC 8

Recommendation 1

In the general population aged ≥60 years, initiate pharmacologic treatment to lower blood pressure (BP) at systolic blood pressure (SBP) ≥150 mm Hg or diastolic blood pressure (DBP) ≥90 mm Hg and treat to a goal SBP <150 mm Hg and goal DBP <90 mm Hg. (Strong Recommendation – Grade A

Recommendation 2

In the general population <60 years, initiate pharmacologic treatment to lower BP at DBP ≥90 mm Hg and treat to a goal DBP <90 mm Hg. (For ages 30-59 years, Strong Recommendation – Grade A;
For ages 18-29 years, Expert Opinion – Grade E

Recommendation 3

In the general population <60 years, initiate pharmacologic treatment to lower BP at SBP ≥140 mm Hg and treat to a goal SBP <140 mm Hg. (Expert Opinion – Grade E

Recommendation 4

In the population aged ≥18 years with chronic kidney disease (CKD), initiate pharmacologic treatment to lower BP at SBP ≥140 mm Hg or DBP ≥90 mm Hg and treat to goal SBP <140 mm Hg and goal DBP <90 mm Hg. (Expert Opinion – Grade E)

Recommendation 5

In the population aged ≥18 years with diabetes, initiate pharmacologic treatment to lower BP at SBP ≥140 mm Hg or DBP ≥90 mm Hg and treat to a goal SBP <140 mm Hg and goal DBP <90 mm Hg. (Expert Opinion – Grade E)

Recommendation 6

In the general nonblack population, including those with diabetes, initial antihypertensive treatment should include a thiazide-type diuretic, calcium channel blocker (CCB), angiotensin-converting enzyme inhibitor (ACEI), or angiotensin receptor blocker (ARB). (Moderate Recommendation – Grade B)

Recommendation 7

In the general black population, including those with diabetes, initial antihypertensive treatment should include a thiazide-type diuretic or CCB. (For general black population: Moderate Recommendation – Grade B; for black patients with diabetes: Weak Recommendation – Grade C

Recommendation 8

In the population aged ≥18 years with CKD, initial (or add-on) antihypertensive treatment should include an ACEI or ARB to improve kidney outcomes. This applies to all CKD patients with hypertension regardless of race or diabetes status. (Moderate Recommendation – Grade B

Recommendation 9

The main objective of hypertension treatment is to attain and maintain goal BP. If goal BP is not reached within a month of treatment, increase the dose of the initial drug or add a second drug from one of the classes in recommendation 6 (thiazide-type diuretic, CCB, ACEI, or ARB). The clinician should continue to assess BP and adjust the treatment regimen until goal BP is reached. If goal BP cannot be reached with 2 drugs, add and titrate a third drug from the list provided. Do not use an ACEI and an ARB together in the same patient. If goal BP cannot be reached using only the drugs in recommendation 6 because of a contraindication or the need to use more than 3 drugs to reach goal BP, antihypertensive drugs from other classes can be used. Referral to a hypertension specialist may be indicated for patients in whom goal BP cannot be attained using the above strategy or for the management of complicated patients for whom additional clinical consultation is needed. (Expert Opinion – Grade E

From: **2014 Evidence-Based Guideline for the Management of High Blood Pressure in Adults: Report From the Panel Members Appointed to the Eighth Joint National Committee (JNC 8)**

JAMA. 2014;311(5):507-520. doi:10.1001/jama.2013.284427

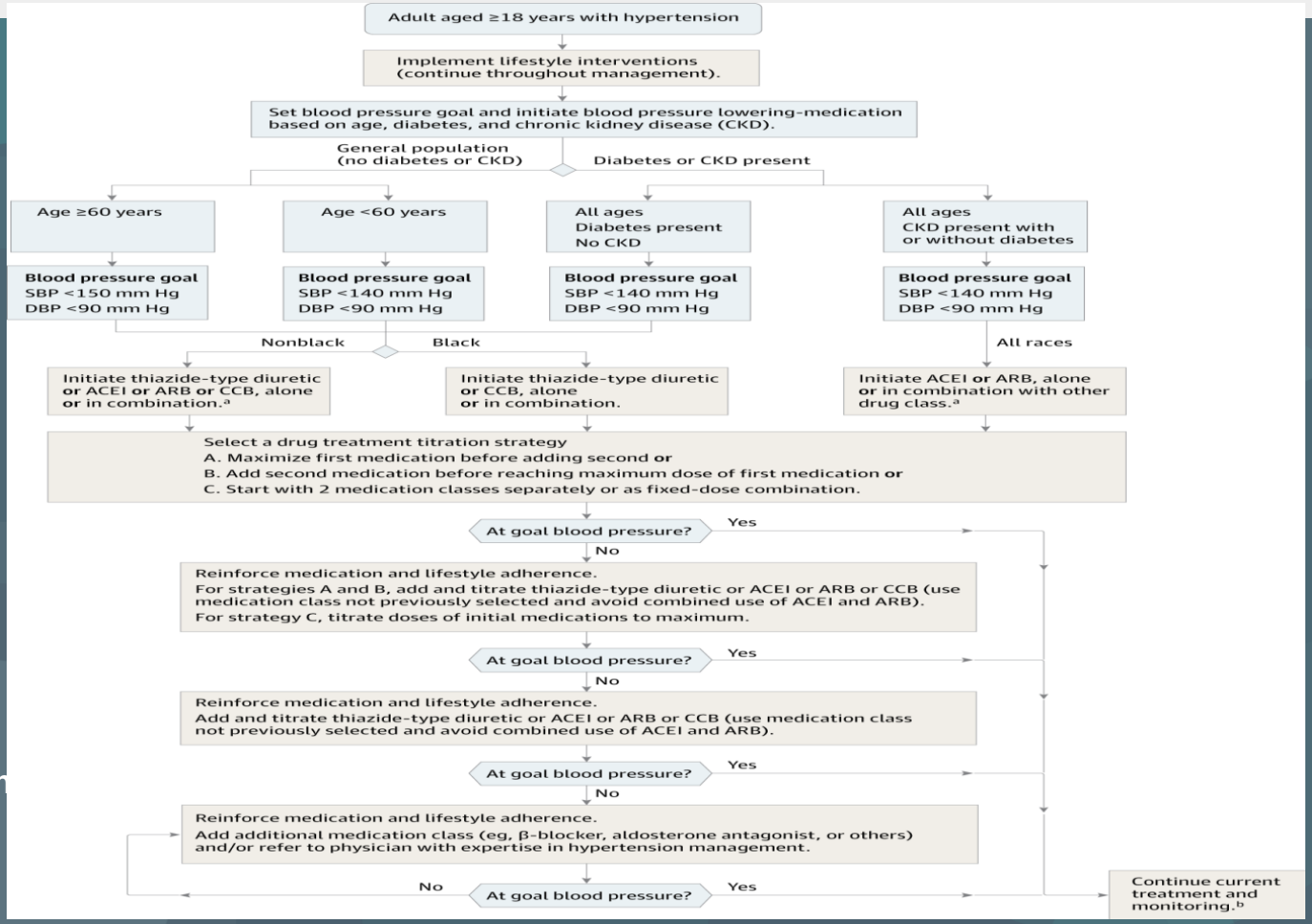


Figure Legen

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THANK YOU