HYPERTENSION

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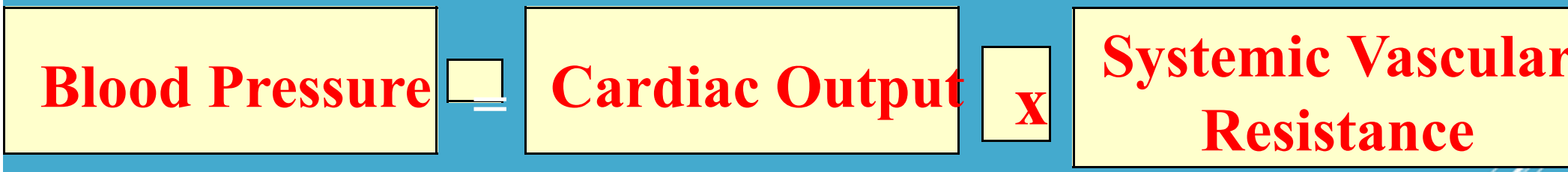
Blood Pressure
=
Cardiac Output
x
Systemic Vascular Resistance

- 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.
Step 2: Use proper technique	-Measurements while patient sitting, legs uncrossed, feet flat on floor, back supported, arm resting on table at heart level. -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.

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

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 in metabolic panel. eGFR indicates estimated glomerular filtration rate.**

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	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

Major area of difference

- 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

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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**

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

HYPERTENSION

COMPLICATIONS

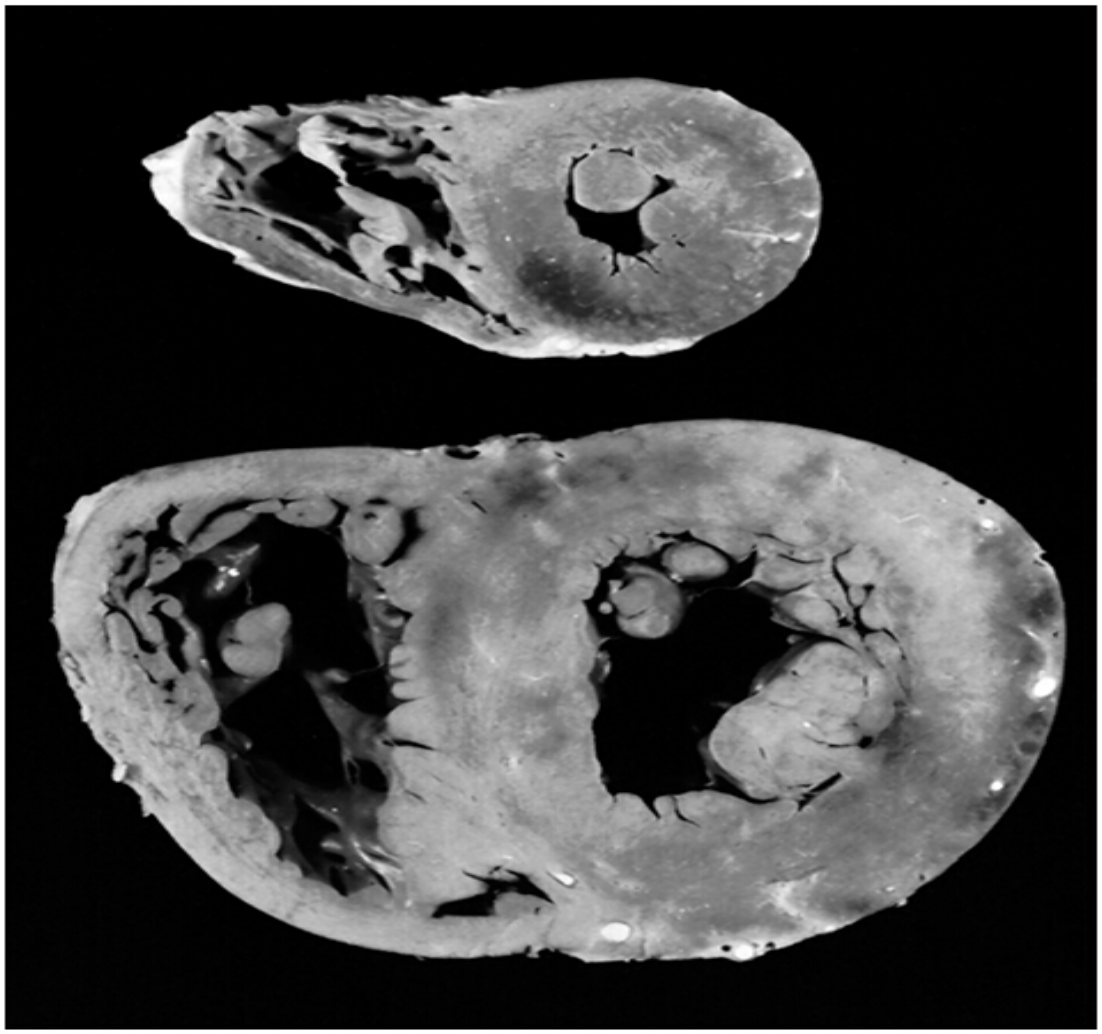
End organ damage involves:

- **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

- Kidney:

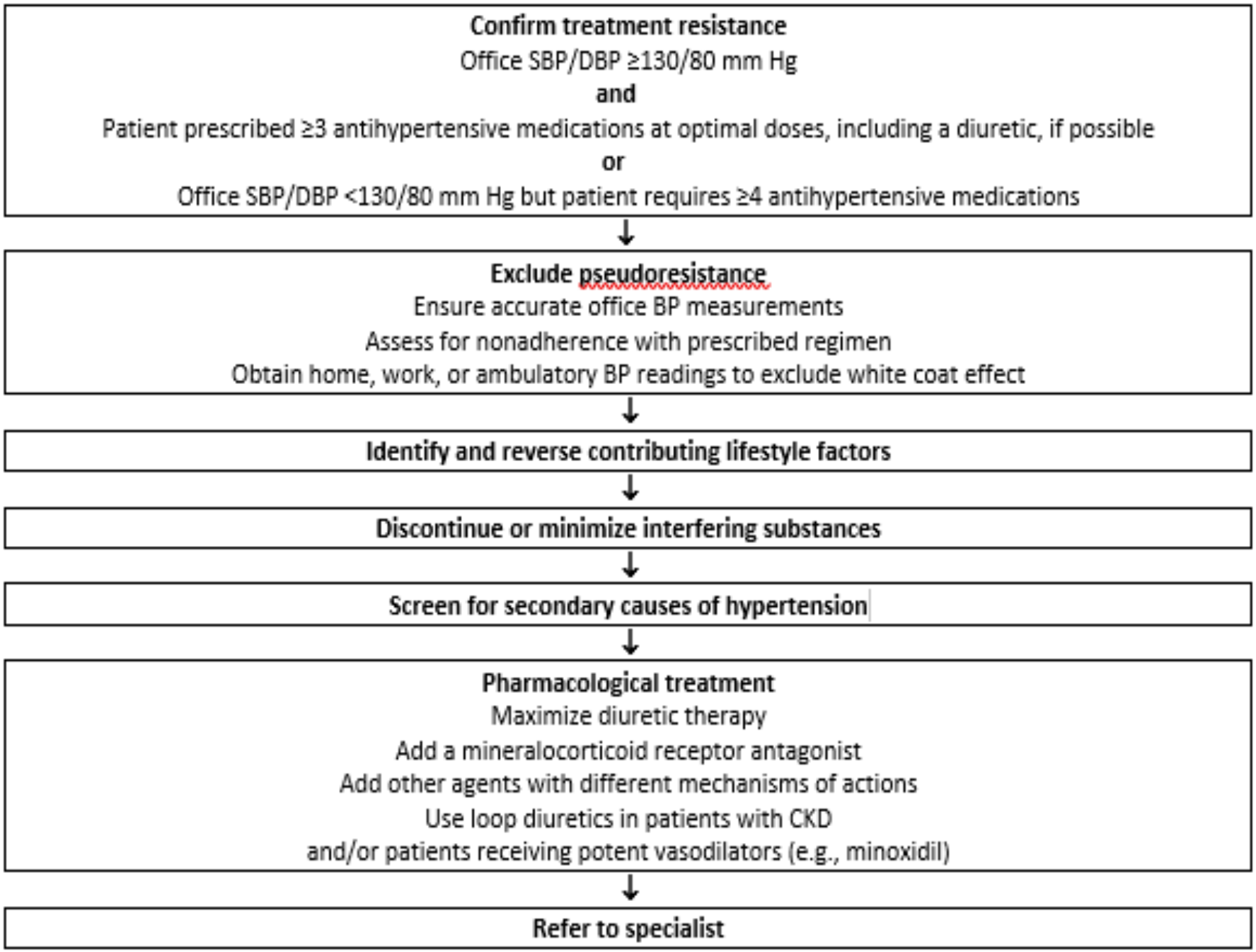
  Nephrosclerosis

  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

- " 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

- 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

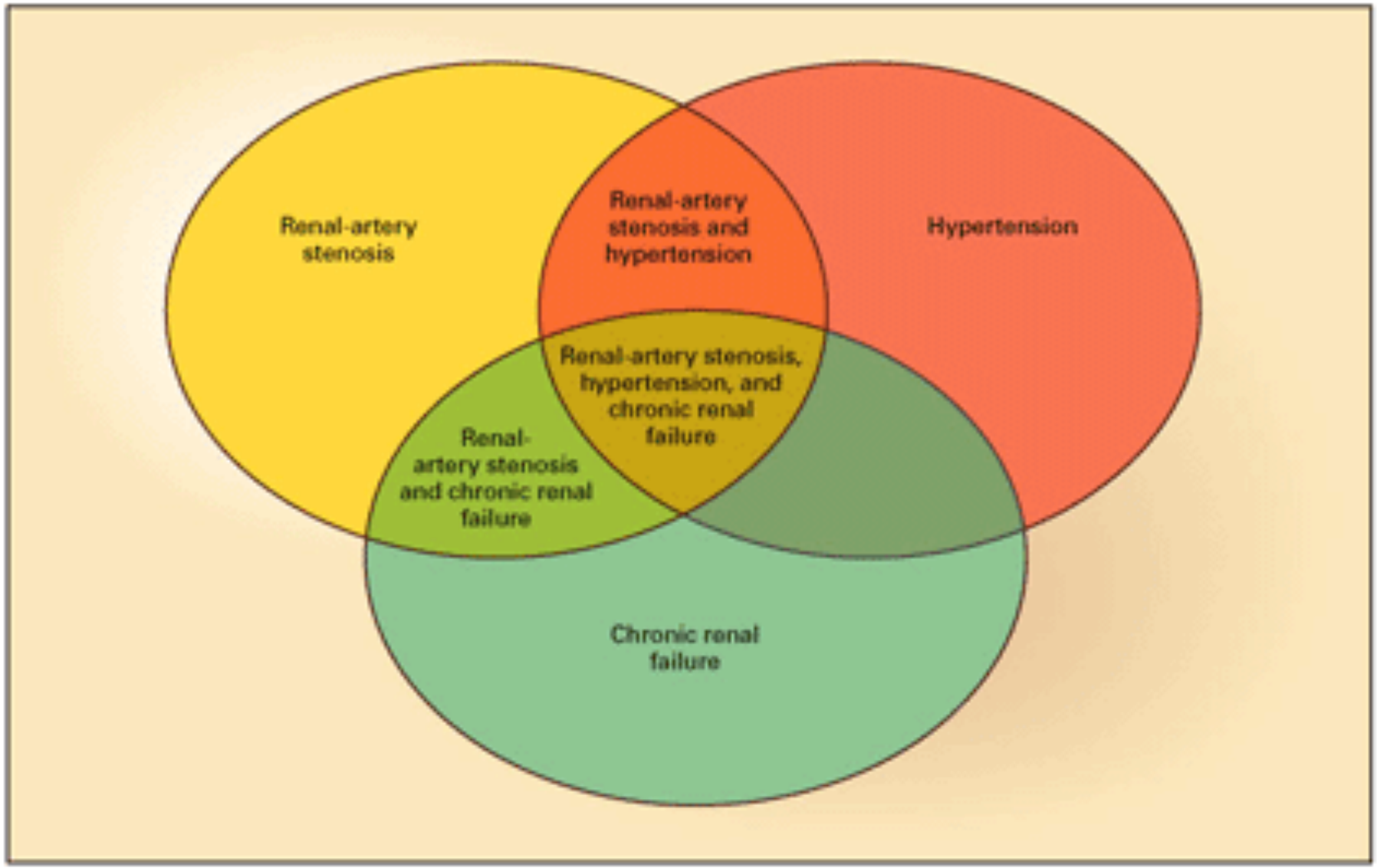
- Common cause of secondary HTN
- HTN is both a cause and consequence of renal disease
- Multifactorial cause for HTN including disturbances in Na/water balance, depletion

  of vasodilators leading to highTPR
- Renal disease from multiple etiologies, treat underlying disease, dialysis/ transplant if necessary

RENAL PARENCHYMAL DISEASE

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



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 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 - 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

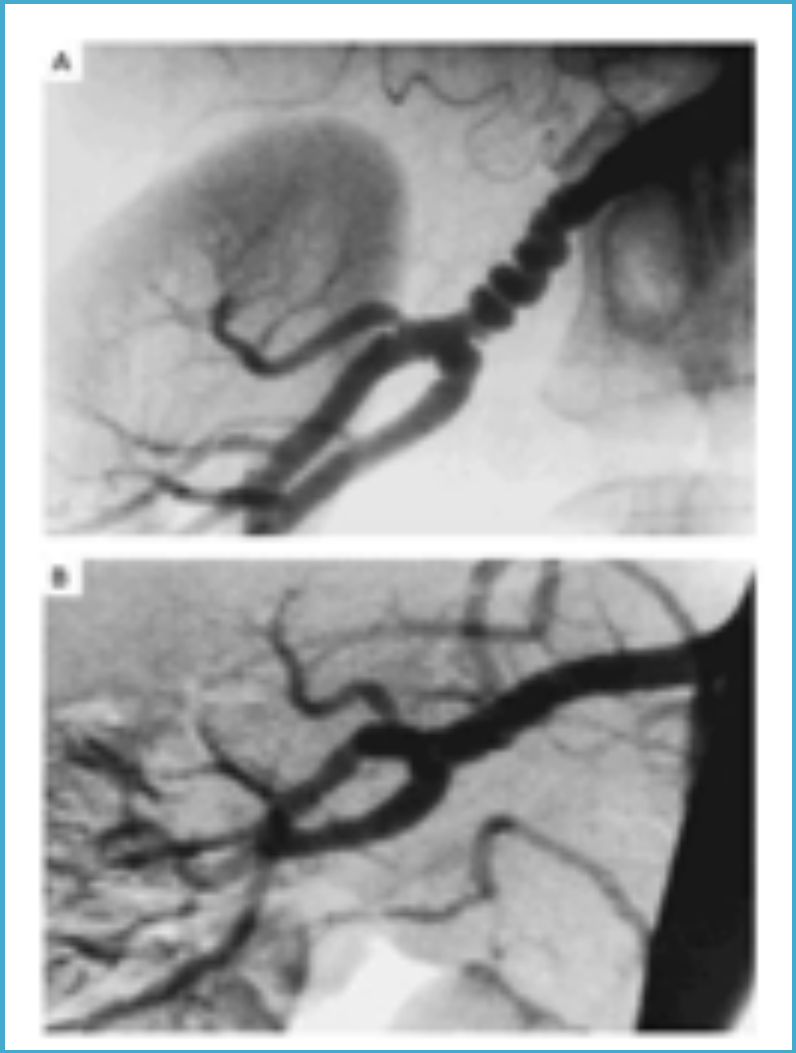
- Physical findings (bruit)
- Duplex U/S
- Captopril renography
- CTA
- MRA
- Renal Angiography

FIBROMUSCULAR DYSPLASIA

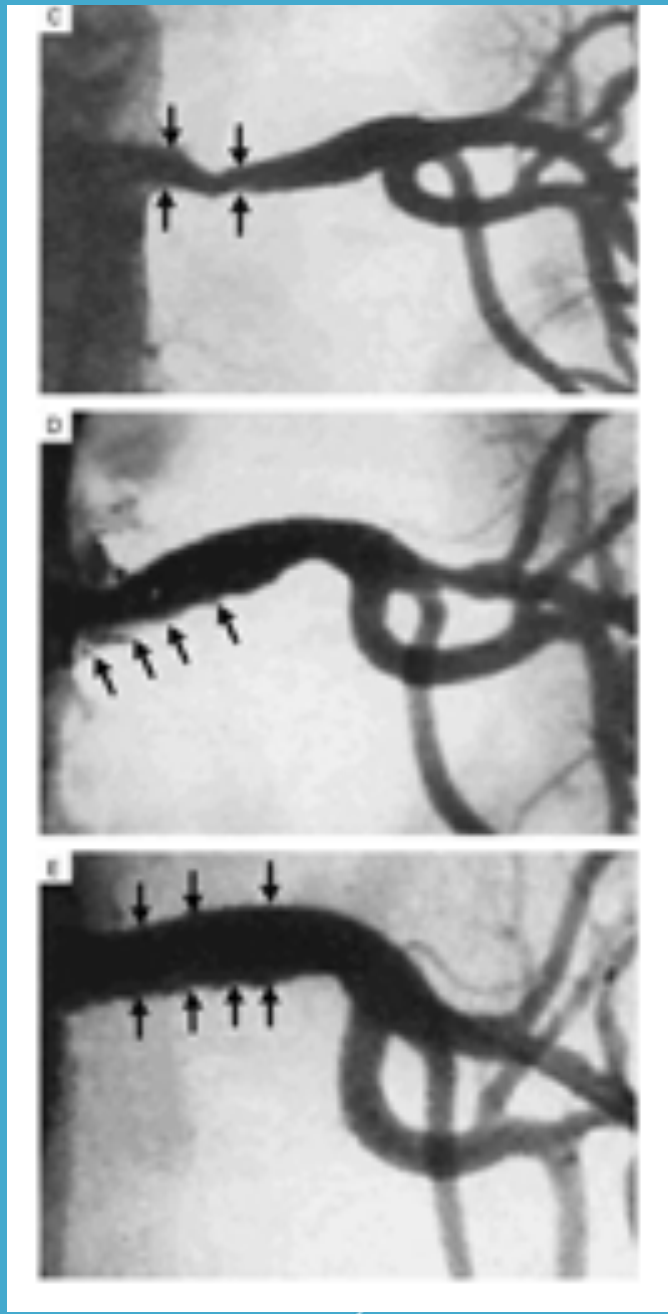
- 10-25% of all RAS
- Young female, age 15-40
- Medial disease 90%, often involves distal RA
- Treatment – PTCA
  - Successful in 82-100% of patients
  - Restenosis in 5-11%
  - “Cure” of HTN in ~60%

ATHEROSCLEROTIC RAS

- 75-90% of RAS
- Usually men, age>55
- Treatment
  - Stent success 94-100%



Fibromuscular Dysplasia, before and after PTCA



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

PRIMARY HYPERALDOSTERONISM

- 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 ALDOSTERONISM- DX

- 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 - 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 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

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

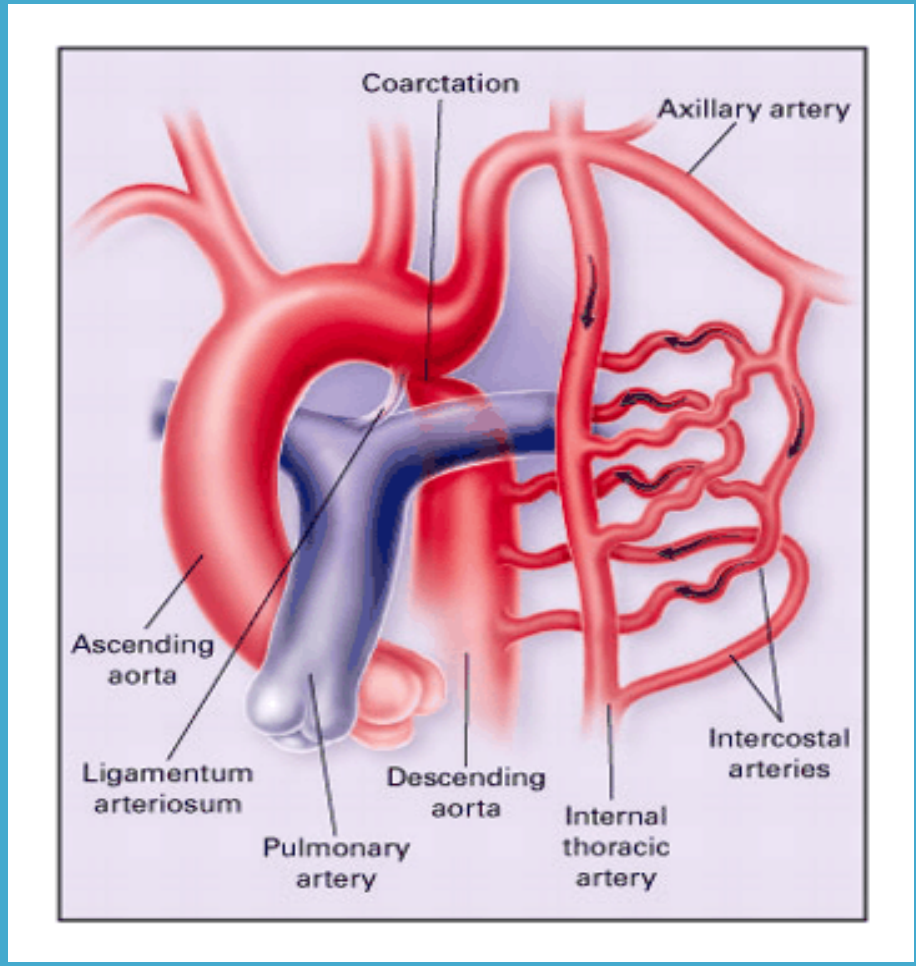
- Screen:
- 24 Hr Urine free cortisol
- Confirm
- Low dose dexamethasone suppression test
- 1mg dexameth. midnight, measure am plasma cortisol
- Imaging
- CT/MRI head (pit) chest (ectopic ACTH tumor)

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



COARCTATION OF AORTA

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

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

HYPERTHYROIDISM

HYPOTHYROIDISM

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

- 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)

CONCLUSIONS

2017 ACC/AHA BP GUIDELINE: THRESHOLDS FOR TREATMENT

SBP		DBP	+ CVD Risk/other circumstances	→ Recommended Treatment
<120	and	<80	N/A	Healthy Lifestyle
120–129	and	<80	N/A	Nonpharmacological therapy
130-139	or	80-89	No CVD/10-yr ASCVD risk <10%*	Nonpharmacological therapy
130–139	or	80–89	CVD/10-year ASCVD risk ≥ 10%	Antihypertensive drug therapy (plus nonpharmacological therapy)
≥130	or	≥80	Diabetes or CKD	
≥130			Age ≥65 years	
≥140	or	≥90	N/A	

* AHA/ACC 2013 Pooled Cohort CVD Risk Equations

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

2017 ACC/AHA BP GUIDELINE: TREATMENT TARGETS

SBP		DBP	CVD Risk	Recommended Treatment
<120	and	<80	N/A	N/A
120–129	and	<80	N/A	N/A
130-139	or	80-89	No CVD and 10-year ASCVD risk <10%	SBP <130 and DBP <80 mm Hg
130–139	or	80–89	Clinical CVD or 10-year ASCVD risk ≥ 10%	
≥130	or	≥80	Diabetes or CKD	
≥140	or	≥90	N/A	
≥130			Age ≥65 years	SBP <130 mm Hg

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

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
  - 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)*

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

Choice of Drug Therapy in Treatment of Hypertension

Combination drug therapy

Initial treatment with two drugs in most patients
esp. in blacks and adults with stage 2 hypertension with BP ≥20/10 above target

Use agents with complimentary modes of action
e.g. diuretic or CCB with ACEI or ARB

Use combination pill when feasible

In blacks with hypertension but without HF or CKD (including those with DM):
Initial treatment should include thiazide-type diuretic or CCB

Simultaneous use of ACEI and ARB <u>not</u> recommended
Potentially harmful

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

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**

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

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*

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

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

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

ANTIHYPERTENSIVE DRUG TREATMENT: CKD

Adults with hypertension and CKD

Treatment with ACE inhibitors reasonable to slow kidney disease progression:

Stage 3 (eGFR 30 - 59 mL/min/1.73 M2) or higher

Stage 1 or 2 with albuminuria ≥300 mg/d

Use of ARBs reasonable if ACE inhibitors not tolerated

BP goal: SBP <130/80 mm Hg

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

THANK YOU