Hypertension Cases

Katharine Dahl, MD

January 10, 2017

Dr. Dahl arrives late for her AHD hypertension lecture and you are asked to present the epidemiology of hypertension until she arrives. Which of the following statements about the epidemiology of hypertension is correct?

A. The prevalence of HTN has increased over the past decade
B. About 75% of people with HTN are controlled
C. About 50% of people with HTN are controlled
D. About 50% of people with HTN are receiving treatment
E. About 50% of people with HTN are aware of a diagnosis of hypertension

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Prevalence, Awareness, and Control

- 1 in 3 US adults has HTN (80 million) which has remained steady
  - Hispanics: 27% men and 29% women
  - Whites: 30% men and 27% women
  - Blacks: 40% men and 43% women
- 83% aware of a diagnosis of HTN (awareness has increased)
- 76.5% being treated for HTN (treatment rates fairly steady since 2012)
- 54% controlled

AHA statistical update 2016, *Circulation*

CASE

A 30yo black woman has come to the her primary care office for a health maintenance visit. She is asymptomatic. BP is 128/80 and her BMI is 30. Otherwise her physical exam is normal.

According to the US preventative Services Task Force, when should her BP be rechecked?

A. 6 months
B. 1 year
C. 3-5 years
D. As needed, based on symptoms

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USPSTF BP Screening Recommendations

- All individuals >40 yo
- High risk for developing HTN despite age need to be rescreened in 1 year

Risk Factors for HTN

- Age
- Obesity (BMI >30)
- Black Race
- Family history
- Lifestyle (inactivity, sodium intake, alcohol consumption)
- High-normal BP (130-139/85-89)
- Other possible contributing factors
  - Stress
  - Smoking
  - Air pollution

CASE

A 65yo woman with primary hypertension is seen in the office for routine follow up. She feels well and is asymptomatic, but she is worried that her BP may be too low. She does not have diabetes or coronary artery disease. BP medications include Lisinopril 10mg qd and hctz 12.5mg qd. Current BP is 130/70. Physical exam is normal. Labs show creatinine of 0.7.

Which one of the following would be the most appropriate management according to JNC 8 guidelines?

A. Discontinue HCTZ
B. Discontinue Lisinopril
C. Discontinue HCTZ and Lisinopril
D. Increase Lisinopril to 20mg qd
E. Make no changes to the current regimen.

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JNC 8 Guidelines

- Elderly (>60 years old): goal < 150/90 BUT, don't need to increase the BP if it is <140/90 and the patient is asymptomatic
- DM and CKD: goal <140/90
- Choice of antihypertensive:
  - Black patients: Thiazide or CCB as initial agent
    - 50% increased risk of CVA with ACE-I/ARB
  - CKD: ACE-I or ARB (if patient is Black, can start with thiazide or CCB but 2nd agent added should be ACE-I/ARB)
  - Avoid beta blockers or alpha blockers as initial agents (unless has another indication such as afib or BPH)

KDIGO guidelines

- CKD with no proteinuria and no diabetes: <140/90
- CKD with proteinuria and no diabetes: <130/80
- DM: 120-125/<90

CASE

40yo man referred to you for evaluation of elevated diastolic BP that has persisted despite lifestyle changes over 6 months. He is overweight and has a family history of premature CAD. Otherwise he has no comorbid conditions. BP is 130/105 in the office and average of 125/95 at home.

In addition to lifestyle modification, how should you counsel him?

A. Start drug treatment
B. Do not treat until home DBP >100
C. Advise him that his cardiovascular risk is increased, but isolated diastolic hypertension does not require treatment
D. Reassure him that diastolic hypertension does not increase cardiovascular risk

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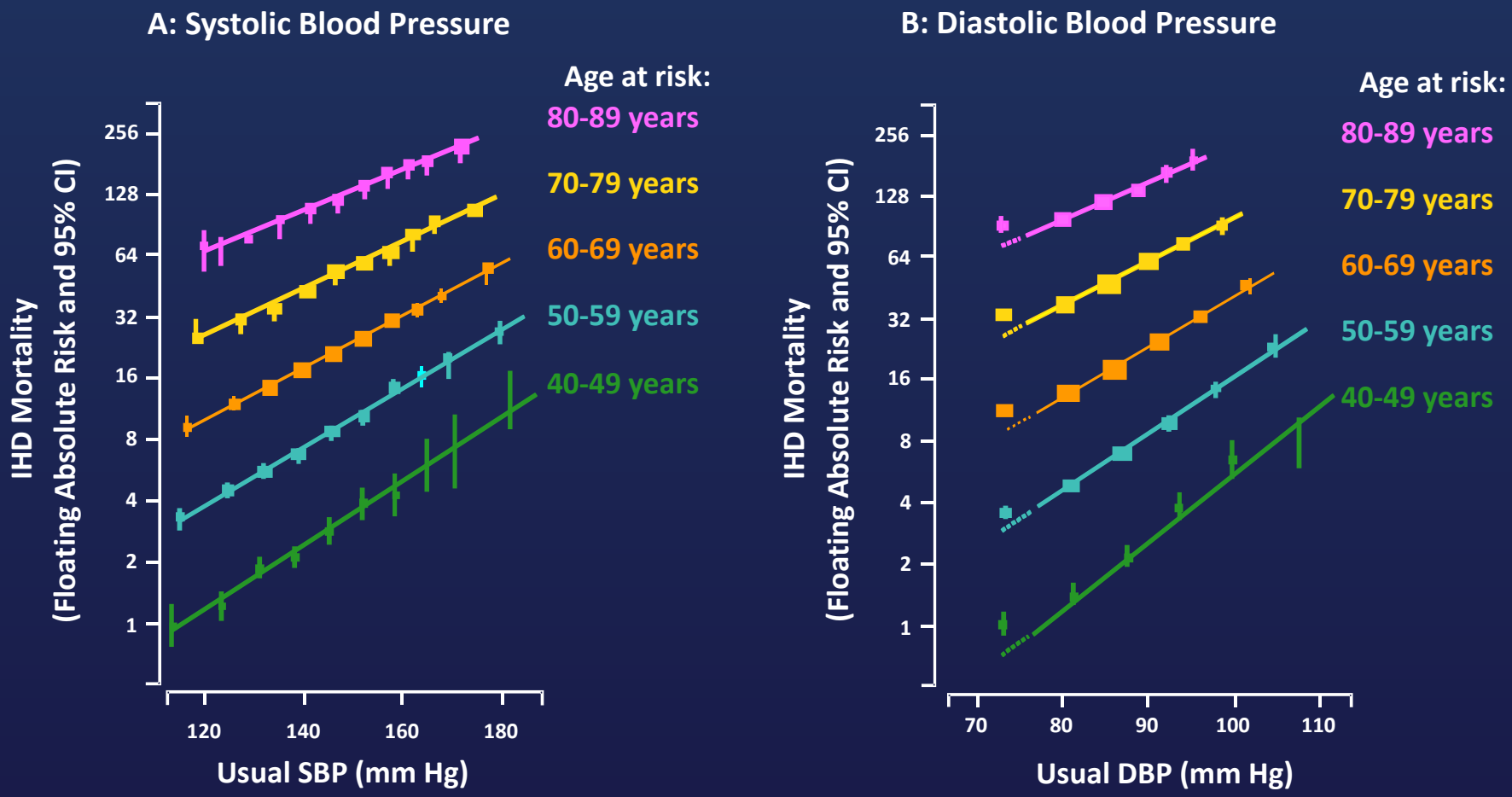
Relationship Between HTN and CV Mortality



Lewington et al. *Lancet.* 2002;360:1903-1913.

CV risk and treatment of diastolic HTN

- Isolated diastolic HTN is associated with increased cardiovascular events and requires treatment
- CV risk of diastolic HTN is less than in isolated systolic or systo-diastolic HTN
- Treatment of diastolic BP is recommended for DBP >100 in the office
- If office DBP 90-99, long-term benefits in the absence of diabetes is uncertain

CASE

48yo man with HTN is in the office for follow up. His antihypertensive regimen includes: amlodipine 10mg qd, telmisartan 80mg qd, chlorthalidone 25mg qd, and metoprolol 50mg bid. He has been adherent to his medications.

Exam shows BP 158/96. HR is 58. His BMI is 30. Labs show potassium 4.2, creatinine 0.8, urine albumin:creatinine ratio is 28mg/g.

24 hour ambulatory BP monitoring is done with the following averages:

24hr: 135/82

Daytime: 140/88

Nighttime: 122/76

What medication would you add next?

A. No change
B. Increase metoprolol
C. Add spironolactone
D. Add doxazosin

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Resistant Hypertension

- BP above goal on 3 medications including a diuretic
  - Need to r/o pseudoresistance (non-adherence, white coat HTN, proper BP technique, sclerotic non-compressible arteries)

- Risk factors
  - Obesity (BMI >30)
  - Primary aldosteronism
  - Black race
  - Obstructive sleep apnea

- Though HTN prevalence increases with age, age is not associated with resistant HTN

White Coat HTN

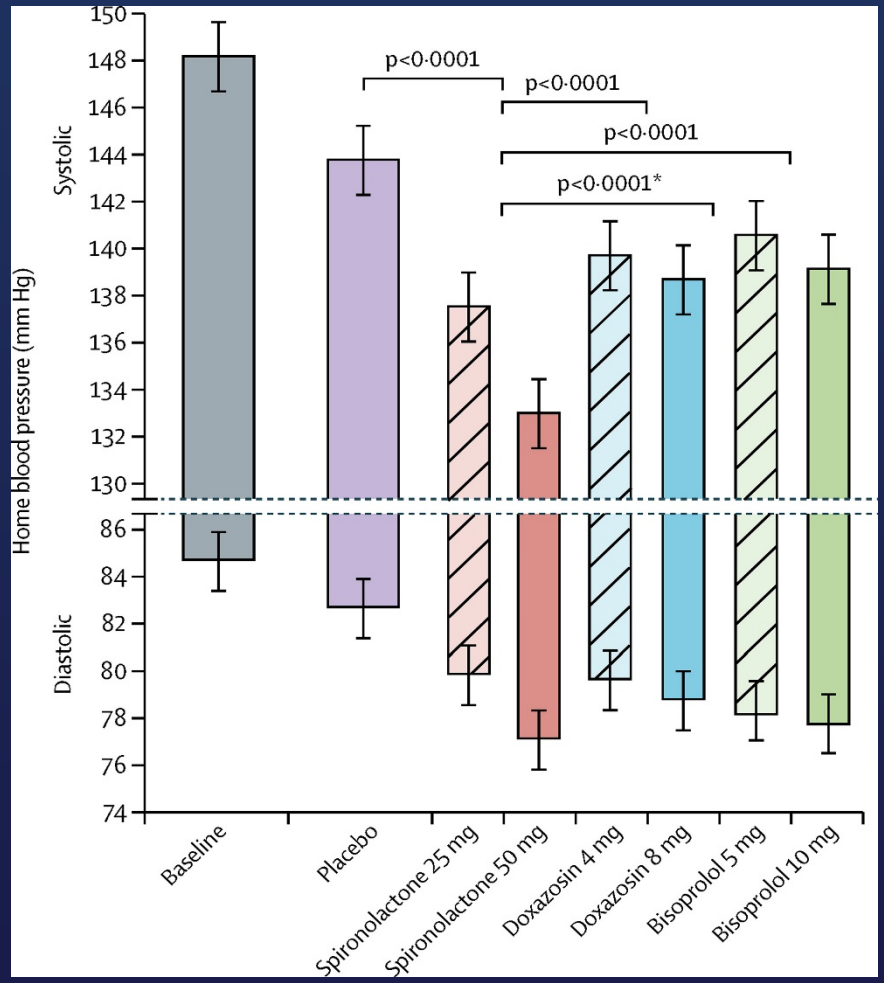
- BP > 140/90 in the office and <135/85 at home
- There is a possible increased risk of CVA seen in small studies but meta-analysis did not show increased risk
- Could check echo to check for end-organ manifestations of HTN, which may guide treatment.

24hr Ambulatory BP Monitoring

- More predictive of cardiovascular death compared to office or home BP monitoring
- Useful to diagnose white coat HTN, resistant HTN, episodic HTN, and masked HTN
- Targets: 24hr average <135/85, daytime <140/90, night <125/75
- Dipping: 15% drop overnight
  - Absence of dipping associated with LVH and increased CV death

Spironolactone Lowers BP in Resistant HTN

- Patients with resistant HTN despite optimally-dosed angiotensin blockade, calcium channel blocker, and a diuretic
- Compared spironolactone, doxazosin, and bisoprolol.
- Spironolactone decreased mean home SBP by 10mmHg compared with the doxazosin and bisoprolol



Williams B et al: British Hypertension Society's PATHWAY Studies Group: *Lancet* 386:2059-2068

CASE

53yo woman with DM2, HTN, and proteinuria. Antihypertensive regimen includes: chlorthalidone, lisinopril, diltiazem, doxazosin. Spironolactone was tried and worked well for her BP, but she stopped taking it due to GI side effects. Home BP readings average 145/90. On exam: BP 163/92, HR 59, 1+ leg edema. Labs show GFR 78ml/min and potassium 3.8, and urine albumin/creatinine ratio of 1182mg/g creatinine.

Which medication would you add next?

A. Amiloride
B. Atenolol
C. Clonidine
D. Minoxidil

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Amiloride Lowers BP and Proteinuria in Resistant Hypertension

- Proteinuria induces activation of the epithelial sodium channel via plasminogen activation
- Amiloride attenuates urine plasminogen activation and lowers BP in patients with resistant HTN

Oxlund CS et al: *J Am Soc Hypertens* 8(12):872-81, 2014

CASE

65yo man with obesity, obstructive sleep apnea, and poorly controlled HTN is seen in the office for HTN management. Ambulatory BP monitoring shows: daytime mean 160/92 and nighttime mean of 154/90 He finds it difficult to tolerate his CPAP machine. Medications include Lisinopril 40mg qd, amlodipine 10mg qd, and chlorthalidone 25mg qd. BP is 166/96 and HR 66 in the office. Physical exam is notable for BMI of 40 and an obese abdomen.

Which one of the following would be the most effective non-pharmalogical intervention to lower his BP and improvethe nocturnal BP pattern?

A. Psychotherapy
B. Device-guided breathing
C. Nocturnal oxygen supplementation
D. Regular use of CPAP for at least 4 hours per night

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CPAP Lowers BP in Patients with OSA and Restores Nocturnal Dipping Patterns

- Obesity – increased risk of OSA and resistant HTN
- Weight loss and CPAP can reverse abnormal dipping patterns

Martinez-Garcia MA et al: HIPARCO randomized clinical trial *JAMA* 310(22):2407-15, 2013

CASE

58yo woman with HTN, hyperlipidemia and active tobacco use is seen in the office for HTN management. BP is 135/75. Physical exam and laboratory tests are normal. Which one of the following would be the most appropriate next step in her management?

A. Change Lisinopril to amlodipine
B. Add amlodipine, targeting SBP <140
C. Add amlodipine, targeting SBP <120
D. Continue the current treatment regimen

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Treatment of Patients with High Cardiovascular Risk (SPRINT study)

- Non-diabetic patients, >50 years old, high cardiovascular risk and HTN
- Targeting SBP <120 (vs SBP <140) resulted in decreased cardiovascular risk and all-cause death
- 25% reduction in composite endpoint of MI, Coronary syndroms, stroke, heart failure, and cardiovascular death.
- 38% less heart failure
- 27% decrease in all-cause death
- All subgroups benefited, including patients older than 75
- Risk of AKI increased by 71% in the low BP group but absolute risk only 4.4% (2.6% in the standard group).

SPRINT Research Group: *NEJM* 373:2103-2115, 2015

CASE

28yo man with autosomal dominant polycystic kidney disease. He has liver cysts but no other extrarenal manifestations of ADPKD. BP is 134/82. Home BP monitoring shows feadings that average 130/82. Physical exam is normal. Labs show eGFR 84ml/min and potassium level of 4.0

Which one of the following would be the most appropriate BP to target to slow increases in kidney volume?

A. 110/75
B. 120/75
C. 130/80
D. 140/80

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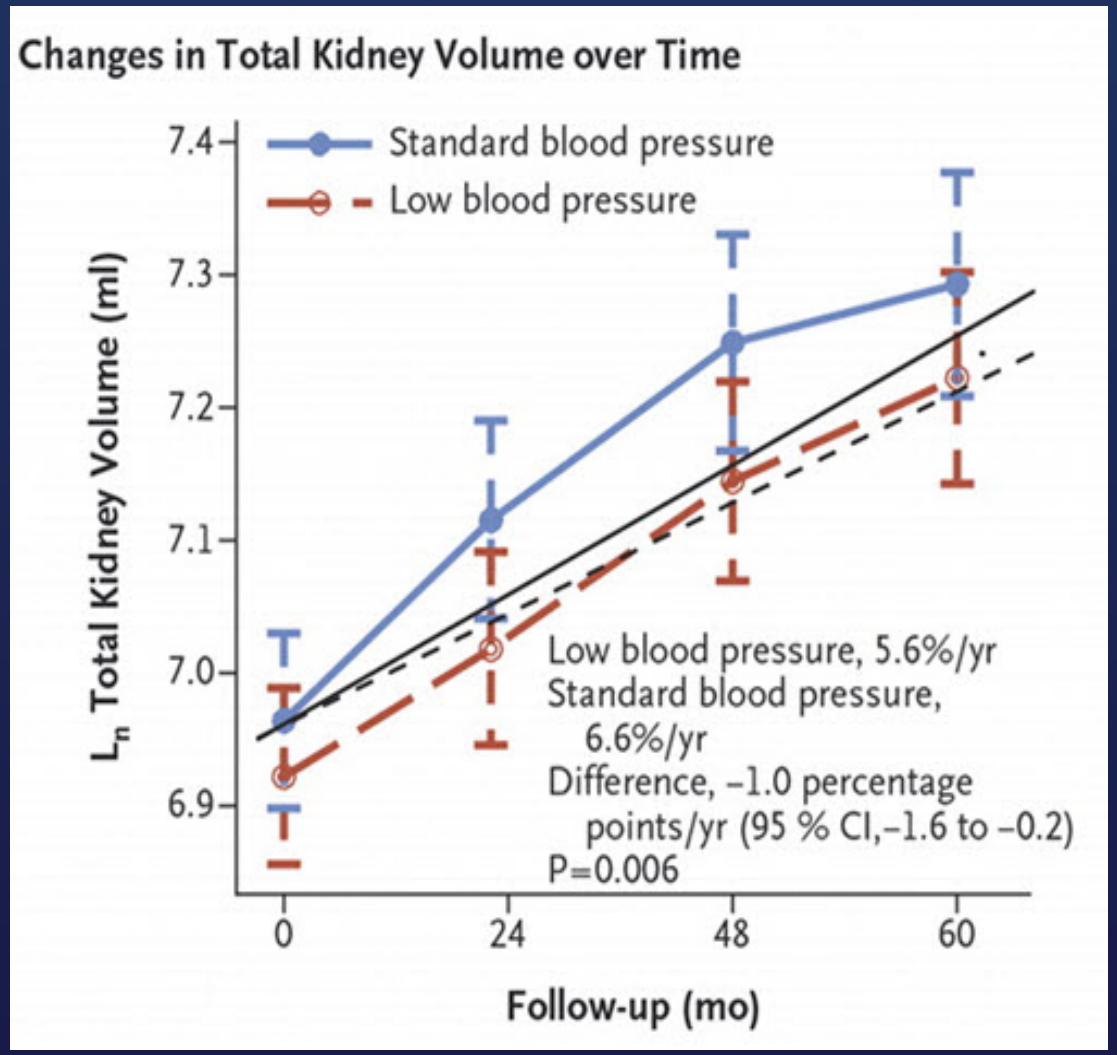
Lower BP Target Slows Increase in Kidney Volume in ADPKD

- Patients with ADPKD and GFR >60ml/min
- Targeting BP 110/75 superior to 130/80 in slowing increases in kidney volume in patients with ADPKD
- ACE-inhibitor based therapy was used to achieve these goals



Schrier RW et al: Blood pressure in early autosomal dominant polycystic kidney disease: *NEJM* 371(24):2255-66, 2014.2068

CASE

A 65-year-old woman is evaluated for resistant hypertension. Despite use of antihypertensive therapy for over 20 years, her blood pressure usually is approximately 160/90 mm Hg. For several years she has been taking amlodipine, 10 mg/d, and metoprolol, 100 mg/d. However, her regimen recently was changed to lisinopril, 20 mg/d, and sustained-release verapamil, 180 mg/d.

On physical examination, pulse rate is 68/min and blood pressure is 178/100 mm Hg. On cardiac examination, the point of maximal impulse is prominent and displaced laterally. The lungs are clear to auscultation. The remainder of the examination is normal.

CASE

Laboratory Studies

BUN 18 mg/dL

Creatinine 0.9 mg/dL

Sodium 147 meq/L

Potassium 3.3 meq/L

Chloride100 meq/L

Bicarbonate28 meq/L

An echocardiogram reveals increased left ventricular mass.

Which of the following is the most appropriate next step in this patient's management?

A Magnetic resonance angiography

B Hydrochlorothiazide, 25 mg/d

C Aldosterone–renin ratio

D CT scanning

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Hyperaldosteronism: Clinical Features

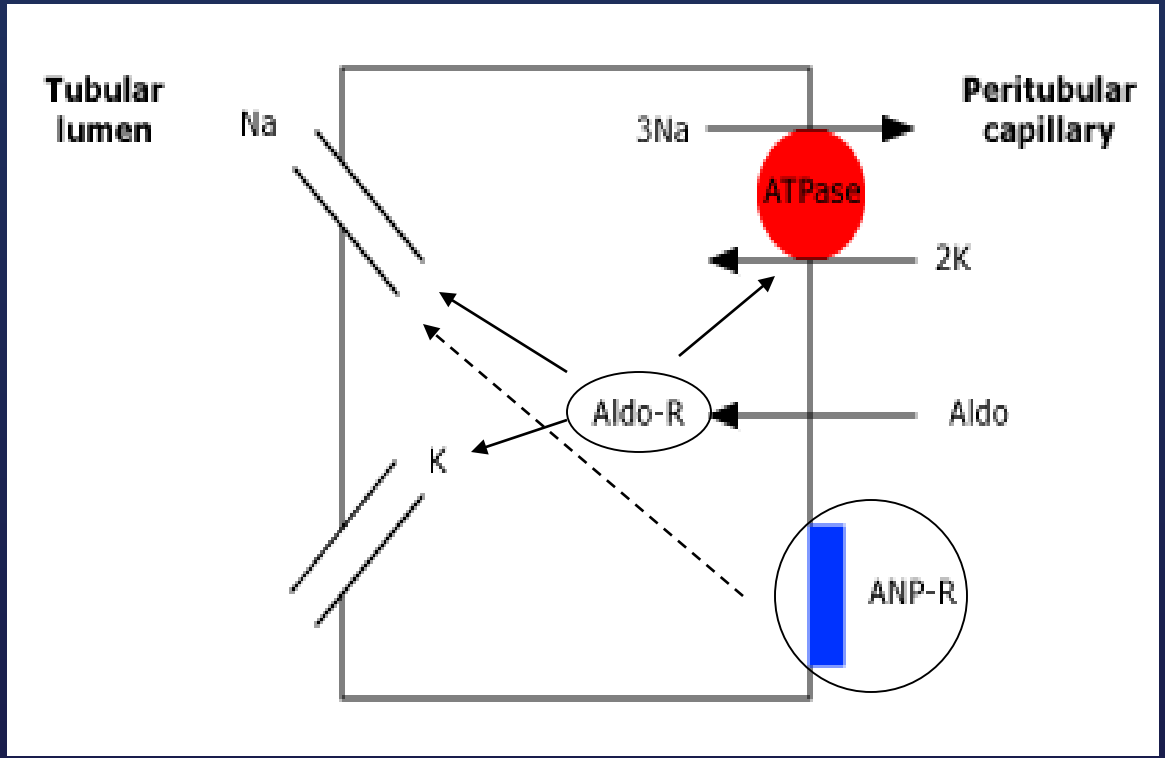
- Consider if unprovoked hypokalemia and metabolic alkalosis or resistant hypertension are present

  (though most patients lack hypokalemia)

- Solitary adenoma or bilateral adrenal hyperplasia

Hyperaldosteronism: Mechanism

Sodium and Potassium Handling in the Collecting Tubule



Hyperaldosteronism:diagnosis

- Increased aldosterone-renin ratio (ARR) PAC/PRA of > 30

AND

- PAC >20ng/dl

(can try to suppress aldosterone with volume expansion – i.e. 2L NS over 4hrs, or NaCl tabs 10g/day x 3 days)

- Sensitivity and specificity of 90% for diagnosis of aldosterone-producing adenoma
- Confirm with 24hr urine aldosterone >12mcg/day

Weinberger MH; Fineberg NS Arch Intern Med 1993 Sep 27;153(18):2125-9.

Hyperaldosteronism: workup/treatment

- CT scan with thin cuts through adrenal glands
- If unilateral adenoma in patient < 40 yrs old with hyperaldosteronism – surgical removal of adenoma indicated.
- If > 40 yrs old, more likely to have bilateral adrenal hyperplasia, → adrenal vein sampling to document unilateral secretion prior to surgery.
- 60-70% cure rate with laproscopic removal of adrenal gland

Hyperaldosteronism: workup/treatment

- If CT negative – could consider adrenal vein sampling (many adenomas < 1cm)
- If unilateral secretion, consider surgery

- If bilateral adrenal hyperplasia, aldosterone blockade (spironolactone)
- Calcium channel blockers can help with blood pressure, but still need to replete K

CASE

A 79-year-old man is evaluated for poorly controlled hypertension. He has had hypertension for 30 years, but his condition has become more difficult to control during the past 2 years. Over the last 6 months, his blood pressure measurements have ranged from 150/70 mm Hg to 170/90 mm Hg. Medications are atenolol, 50 mg/d; enalapril, 20 mg twice daily; and hydrochlorothiazide, 25 mg/d.

On physical examination, pulse rate is 66/min and blood pressure is 168/80 mm Hg; these results were the same on two previous office visits. Cardiac examination reveals a faint midline abdominal bruit and a left femoral bruit.

Creatinine level is 1.1 mg/dL, which has not changed for 2 years.

Low-density lipoprotein cholesterol level is 160 mg/dL.

CASE

Which of the following is the most appropriate management at this time?

A Renal angiography

B Amlodipine

C Plasma renin activity measurement

D Renal vein renin sampling

E Magnetic resonance angiography of the renal arteries

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Renovascular Hypertension: clinical features

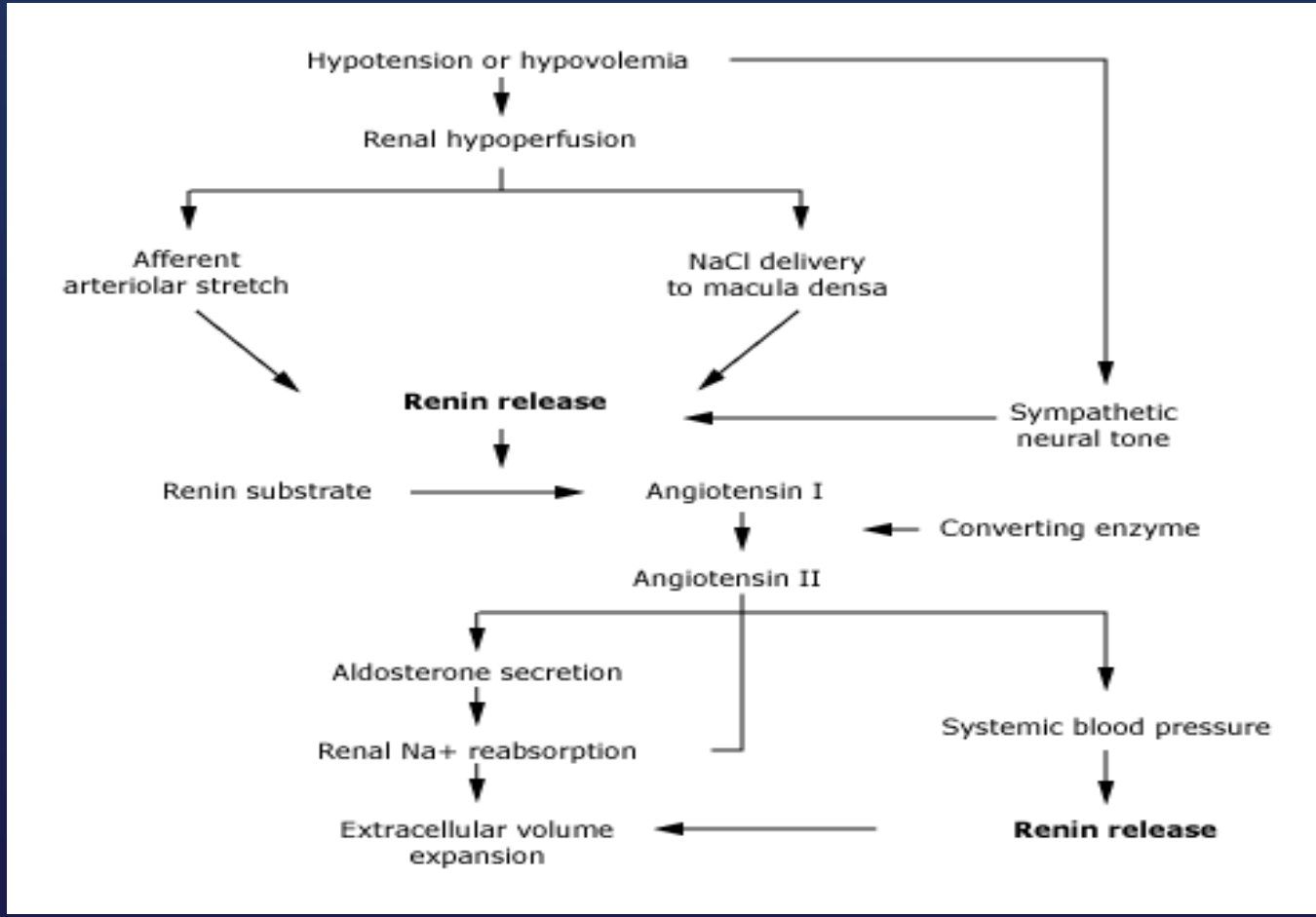
Clinical features	Essential HTN (%)	Renovascular HTN (%)
Duration < 1 year	12	24
Age of onset > 50	9	15
Family Hx of HTN	71	46
Grade 3-4 retinopathy	7	15
Abdominal bruit	9	46
BUN >20mg/L	8	16
Potassium <3.4	8	16
Urinary casts	9	20
Proteinuria	32	46

Simon N, et al. *JAMA.* 1972;220:1209-18.

Renovascular Hypertension: mechanism

- Occurs in the setting of hemodynamically significant (at least 72-80%) unilateral or bilateral renal artery stenosis via:
  - Stimulation of renin-angiotensin system
  - Sodium retention leading to excess plasma volume

Renovascular Hypertension: mechanism – stimulation of RAAS



Renovascular Hypertension: diagnosis

- Gold standard: angiography
- MRA less invasive but risk of nephrogenic systemic fibrosis if GFR <30 (and controversial (GFR 30-60)
- Risks associated with renal artery revascularization (bleeding, vessel dissection, branch occlusion, atheroembolic renal failure, MI, CVA, contrast nephropathy) make establishing diagnosis important.

Renovascular Hypertension:
Atherosclerotic RAS - treatment

- Less clear whether or not revascularization will be helpful
- Coexistent essential HTN confounds diagnosis
- Angioplasty improves BP control in only 30%-50% of patients
- Renal function improves in 25%-29%, but deteriorates in 19-25%(artheroemboli).
- Antihypertensive therapy effectively controls BP in many patients with renovascular hypertension

Van Jaarsveld BC, et al. NEJM 2000 Apr 6;342:1007-14. Plouin PF et al. Hypertension 1998 Mar;31:823-9. Webster J et al. J Hum Hypertens. 1998 May;12:329-35.

Features of Improvement in BP and Renal function after Revascularization

- Sudden acceleration of HTN or renal failure
- Size discrepancy
- Renal resistive index <0.8 on ultrasound

(RI = peak syst velocity – end diastolic velocity/peak syst velocity)

- Decrease in GFR on radionucleotide scanning
- Increase in creatinine after ACE inhibitor
- Lateralization of renin secretion on renal vein renin sampling
- Increased gradient across stenosis at time of angiogram

CASE

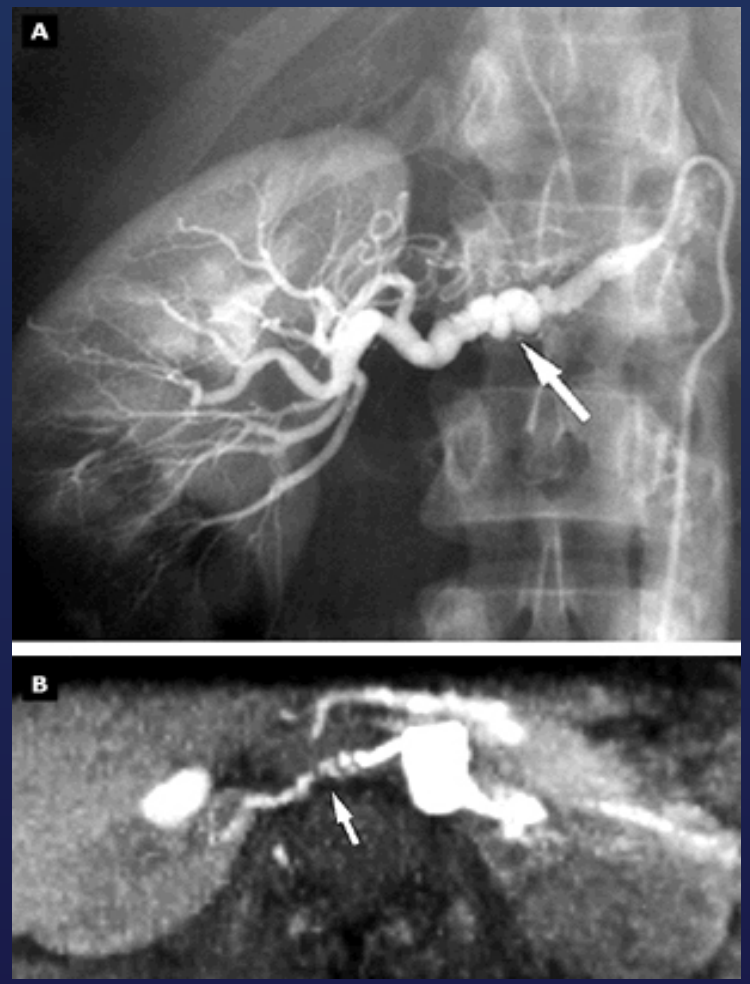
- 33 yo woman referred for evaluation of recent-onset severe HTN. She denies evening snoring or gasping for air, headaches, palpitations, or sweating. Her BP medications are amlodipine 10mg qd, metoprolol 100mg bid, and chlorthalidone 25mg qd. Physical exam: BMP 24, BP 170/100, HR 60. No abdominal bruits. Labs: sodium 138, potassium 3.5, co2 28, creatinine 1.2, aldosterone 18, renin 12. MRI of the abdomen shows no adrenal mass.

- Which is the most appropritate next test?

A. Refer for polysomnography
B. Measure plasma metanephrines
C. Refer for adrenal vein sampling
D. Order CT angiogram

Renovascular Hypertension:
Fibromuscular dysplasia - diagnosis

- Young women <50
- Affects carotid arteries (30%) and renal arteries (60%)
- Pathogenesis unclear ?hormonal
- HTN, TIA, Stroke



Renovascular Hypertension:
Fibromuscular dysplasia - treatment

- Young patients with fibromuscular dysplasia with HTN should be revascularized.
- Angioplasty successful, stent not needed. Reports of stent fracture in FMD.
- 30-50% chance of cure of HTN
- Surgery if angioplasty unsuccessful
- Need future monitoring with renal artery dopplers to monitor for recurrence/progression.

CASE

- 75 yo white man with a history of CKD presents for evaluation of resistant HTN. Average home BP is 170/94. His BP medications are: losartan 100mg qd, amlodipine 10mg qd, chlorthalidone 25mg qd, and carvedilol 25mg bid. On exam, his BMI is 23, BP 174/96. He has hypertensive retinopathy and a S4 gallop. No edema. Labs show creatinine of 2.1 (eGFR 1.73)

Which of the following is the most likely to be associated with his resistant hypertension?

A. His race

B. His age

C. Underlying CKD

D. His BMI

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HTN from Renal Disease: epidemiology

- Most common cause of secondary HTN
- Hypertension eventually occurs in 85-90% of CKD patients

HTN from Renal Disease: mechanism

- 80% - volume expansion is primarily responsible for HTN

- ≅ 20% - increased renin release is important

HTN from Renal Disease: mechanism of renin-mediated HTN

- Primary vascular disease from vasculitis, hypertensive nephrosclerosis, or atherosclerotic renal artery stenosis

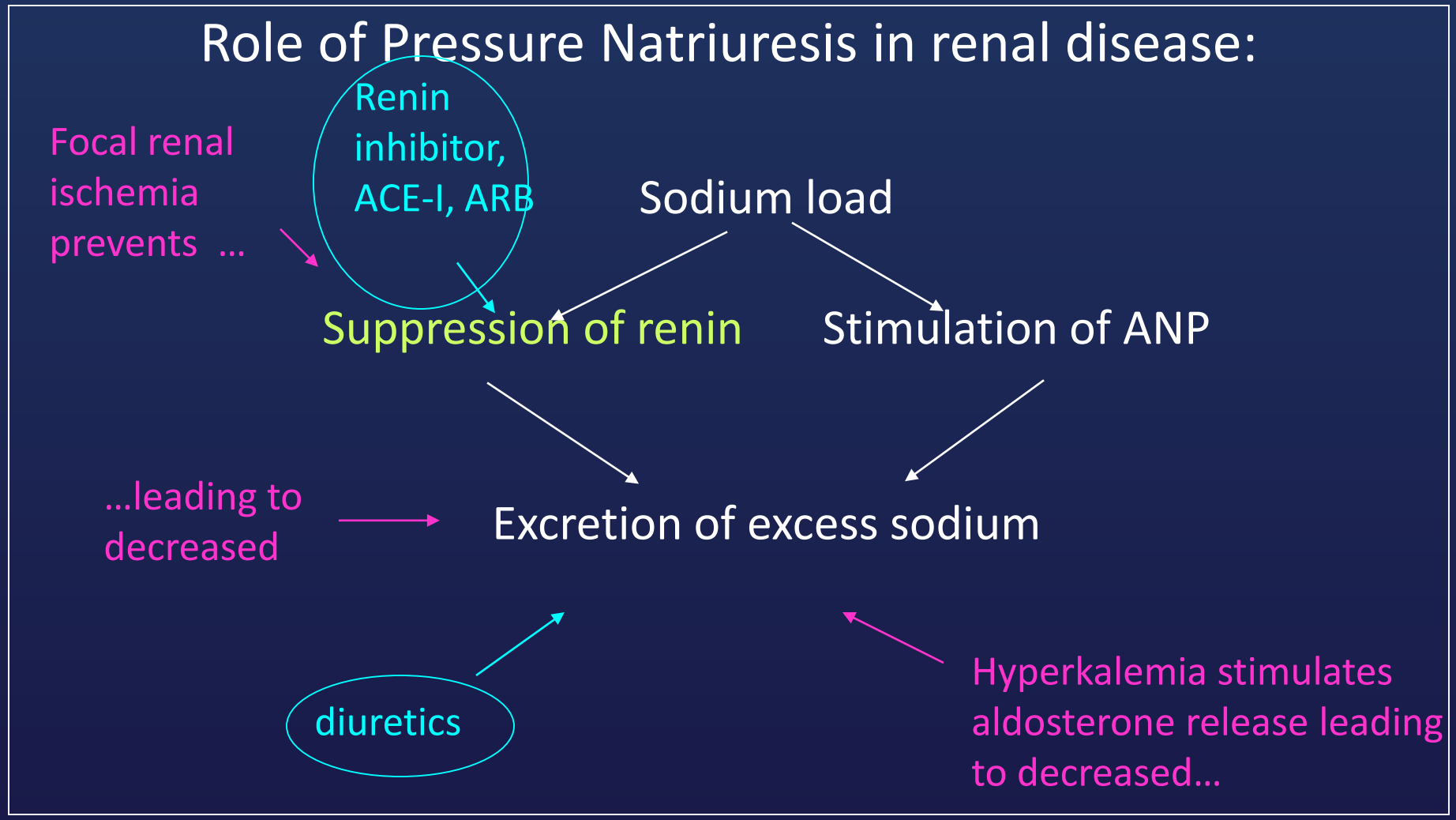
- Disordered renal architecture causing focal ischemia leading to increased renin release

(extrinsic and intrinsic renin stimulation)

Renal Disease: HTN is necessary for maintenance of homeostasis

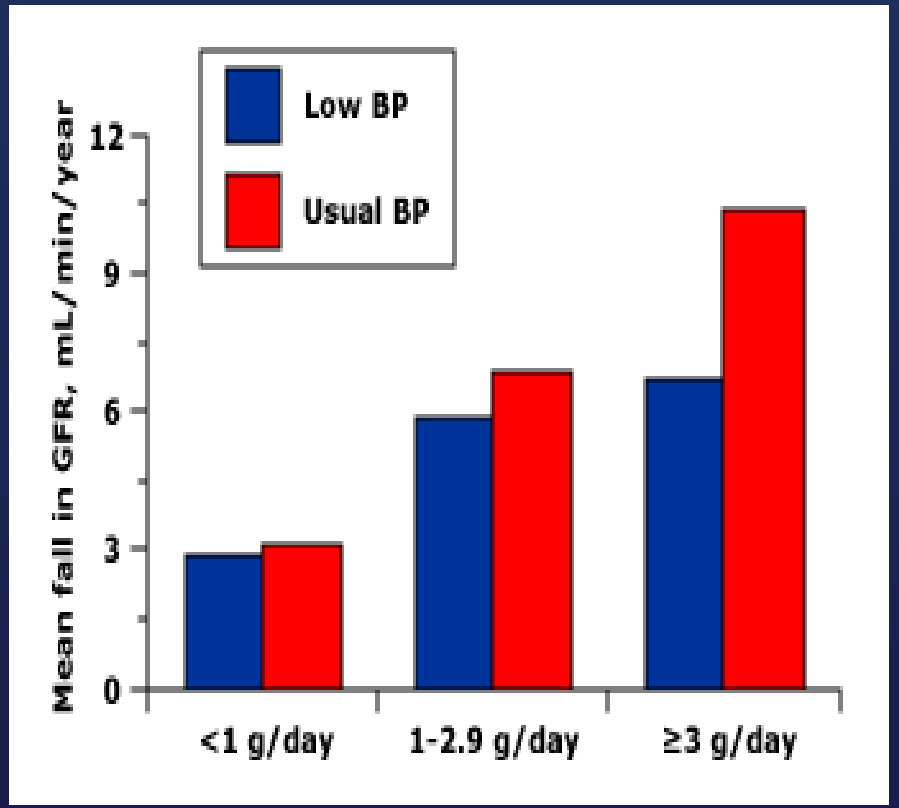
- Decreased number of nephrons requires that each nephron secrete more sodium
- HTN leads to increased renal perfusion pressure which increases sodium excretion
- Thus, HTN is the “price paid” for preventing sodium accumulation

HTN from Renal Disease: Treatment



Aggressive BP control in proteinuric patients

- Usual BP control
  - <130/80
- Aggressive BP control
  - 4.7mmHg lower than usual BP
- Aggressive BP control only benefited pts. with >1g/day proteinuria



Klahr S, et al. *N Engl J Med* 1994;330:877.

CASE

74 yo man with a history of poorly controlled HTN is evaluated 3 months after a left-sided middle cerebral artery stroke. He wants to avoid a future stroke and asks about optimal BP goal. On exam, BP 155/80. He does not have carotid bruits or a cardiac murmur. He has mild right arm weakness.

Which of the following is associated with the highest 5 year risk of stroke, MI or death?

A. A discharge SBP of >140

B. Maintenance of his SBP <140

C. Maintenance of his systolic BP at <130

D. Maintenance of his SBP <120

CASE

25 year old man with a history of hypertension since age 15.

BP in the office is 180/90. Labs reveal potassium of 3.0, bicarbonate of 32. Renin is 0.4, aldosterone is 3.

The best treatment of this disorder is:

A. Spironolactone

B. HCTZ

C. Triamterene

D. Furosemide

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The best medication for his HTN is:

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B. HCTZ

C. Triamterene

D. Furosemide

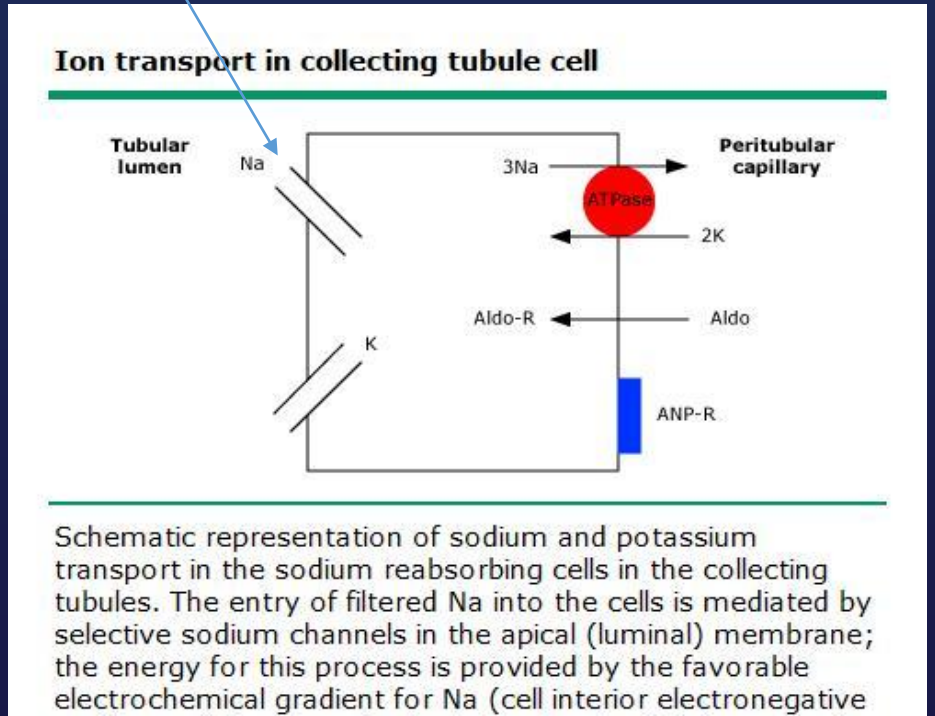
BP Management after CVA

- SBP <120 associated with worse long-term outcome in patients
- AHA/ASA recommends BP <140/90
  - SBP <130 for lacunar stroke
- CATIS trial: randomized: sbp lower 10-25% within 24hrs, <140/90 within 7 days vs discontinuation of all antihypertensive medications
  - No difference in death or disability at 14 days or hospital discharge

Liddle Syndrome

- Autosomal dominant
- Defect localized to chromosome 16q, gene that encodes the β or γ subunit of the renal epithelial Na+ channel at the apical membrane of the principal cells in the cortical collecting duct
  - Effect of mutation is to increase the expression of sodium channels in the luminal membrane
- Clinical manifestations
  - Severe hypertension, presenting in childhood
  - Hypokalemic metabolic alkalosis
  - Pseudohyperaldosteroism: Renin and aldosterone are both suppressed

  (unlike primary hyperaldosteronism-Conn Syndrome)
- Treatment
  - Triamterene
  - Dietary sodium restriction



CASE

45 year old man with hyperkalemia and hypertension.

BP is 165/86

Potassium 5.8, bicarbonate 18, magnesium 2.0,

Renin 0.01

Aldosterone 2

The best treatment for his HTN is:

A. Chlorthalidone
B. Spironolactone
C. Acetazolamide
D. Amiloride

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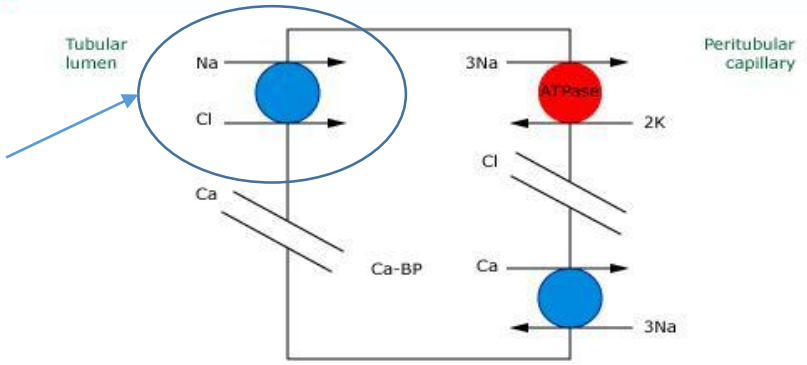
Gordon's Syndrome

- Autosomal dominant
  - WNK4 – mutation increases activity of the thiazide-sensitive sodium chloride cotransporter (salt retention) and the ROMK channel (hyperkalemia)
  - WNK1 – mutation blocks down-regulation of WNK4
- Clinical manifestations
  - Hypertension
  - Hyperkalemia
  - Metabolic acidosis
  - Hypercalciuria
  - Pseudohypoaldosteroism: Renin and aldosterone are suppressed (unlike Gitelman's syndrome)
- Treatment
  - Thiazide diuretic
  - Dietary sodium restriction

Affects of Mutations in Gordon Syndrome

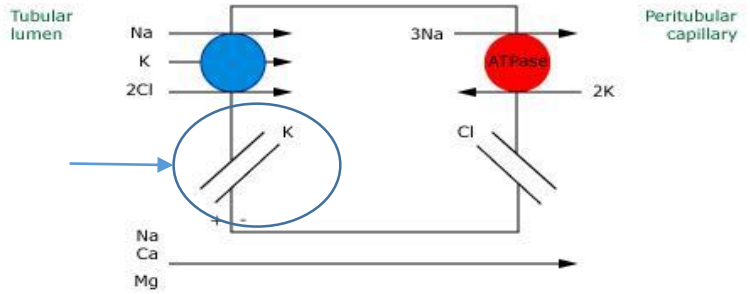
Ion transport in the distal tubule



Schematic representation of the mechanisms of Na, Cl, and Ca reabsorption in the distal tubule. The entry of filtered Na chloride into the cell is mediated by a neutral Na-Cl cotransporter in the apical (luminal) membrane; the energy for this process is provided by the favorable electrochemical gradient for Na (low cell sodium concentration and cell interior electronegative). At the basolateral (peritubular) membrane, reabsorbed Na is pumped out of the cell by the Na-K-ATPase pump, while reabsorbed Cl exits via a chloride channel. **Thiazide diuretics** inhibit NaCl reabsorption by competing for the Cl site on the apical Na-Cl cotransporter. The distal tubule is also the major site of active Ca reabsorption. Ca enters the cell via a Ca transporter that is probably a Ca channel. Reabsorbed Ca combines with a vitamin D-induced Ca binding protein (Ca-BP), moves across the cell, and is then extruded at the basolateral membrane by a Ca-ATPase (not shown) and, to a greater degree, a 3Na:1Ca exchanger that again uses the energy provided by the favorable inward gradient for Na.

UpToDate

Ion transport in loop of Henle



Schematic representation of the transport mechanisms in the thick ascending limb of the loop of Henle. The entry of filtered NaCl into the cells is mediated by a neutral Na-K-2Cl cotransporter in the apical (luminal) membrane; the energy for this process is provided by the favorable inward electrochemical gradient for Na (low cell Na concentration and cell interior electronegative). Reabsorbed Na is pumped out of the cell by the Na-K-ATPase pump in the basolateral (peritubular) membrane. Although K plays an important role in this process, the concentration of K in the filtrate and tubular fluid is much less than that of Na and Cl; thus, K must recycle back into the lumen through K channels in the apical membrane to allow continued NaCl reabsorption. This movement of cationic K into the lumen plus the movement of reabsorbed Cl (via a Cl channel) out of the cell into the peritubular capillary generates a net positive current from the capillary to the lumen. The ensuing lumen electropositivity creates an electrical gradient that promotes the passive reabsorption of cations - Na, and, to a lesser degree, Ca, and Mg - via the paracellular pathway between the cells. The loop diuretics inhibit Na, K, and Cl (and Ca and Mg) reabsorption by competing for the Cl site on this transporter.

UpToDate

CASE

57yo woman presents to the ER with chest pain, dyspnea, and is found to have acute pulmonary edema. Creatinine is 1.7, and she has proteinuria and microhematuria on urinalysis. BP is 220/110.

Which is false about optimal treatent:

A. BP should be lowered 10-20% within the 1st hour
B. BP should be lowered 25% within the 1st 24 hours
C. Diuretics are contraindicated in this patient
D. Nitroprusside should not be used for a prolonged period in this patient

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