Hypertension: Diagnosis and Management

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Scope of the Problem—Prevalence

- Most common reason for office visits in non-pregnant pts in US.
- ~30% of adults >18 y.o.
- 58-65 million hypertensive adults in US.
- Hypertension on the rise:
  - Obesity epidemic.
  - Elderly (>1/2 of pts >65 – systolic or systolic + diastolic).

Prevalence – 2

- African-Americans >45 y.o.:
  - 35% of women
  - ~40% of men >45 y.o.
- European Americans >45 y.o.:
  - 15% of women
  - 25% of men
- Worldwide: 1 billion.

Scope of the Problem – Control

<table>
<thead>
<tr>
<th></th>
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</tr>
</thead>
<tbody>
<tr>
<td>Aware of HTN</td>
<td>73</td>
<td>68.4</td>
<td>70</td>
<td>78</td>
</tr>
<tr>
<td>Treated for HTN</td>
<td>55</td>
<td>53.6</td>
<td>59</td>
<td>68</td>
</tr>
<tr>
<td>HTN controlled</td>
<td>29</td>
<td>27.4</td>
<td>34</td>
<td>43.5</td>
</tr>
</tbody>
</table>

- We’re finally making some progress.
- More than half of hypertensive pts still uncontrolled.
- Another study showed 44% of men & 55% of women w/HTN have adequate control.
Scope of the Problem—Complications

- CAD/MI
- Heart failure
- LVH (independent risk factor)
- Stroke
- Peripheral vascular (arterial) disease
- Dysrhythmias
- Chronic kidney disease (also a risk factor for CAD)
- Hypertensive retinopathy

Making the Diagnosis of HTN

- 2 or more “properly measured” readings at each of 2 or more visits after an initial screen. In other words, persistent elevation on 3 separate visits.
  - Elevation of either SBP (>139) or DBP (>89) or both.
  - If SBP and DBP fall into different categories, the higher value is used.
  - For adults who are not acutely ill and not on HTN meds.

How to Measure BP

- JNC 7 says:
  - Pt seated ≥ 5 min in chair, w/feet on floor, arm at heart level.
  - 2 readings, 5 min apart.
  - Confirm elevated BP in contralateral arm.
  - No caffeine, exercise, or smoking for ≥ 30 min before measurement.
  - Important to use appropriately sized cuff (bladder encircles ≥ 80% of arm).

Classification of BP in Adults

<table>
<thead>
<tr>
<th>BP Classification</th>
<th>SBP</th>
<th>DBP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>&lt;120</td>
<td>&lt;80</td>
</tr>
<tr>
<td>Prehypertension</td>
<td>120-139</td>
<td>OR</td>
</tr>
<tr>
<td>Stage 1 HTN</td>
<td>140-159</td>
<td>OR</td>
</tr>
<tr>
<td>Stage 2 HTN</td>
<td>≥160</td>
<td>OR</td>
</tr>
</tbody>
</table>

- Treat pre-HTN only if compelling indication (DM, CAD, CKD, etc).
- Goal in pre-HTN w/o compelling indication = reduce BP to normal range, & prevent progressive incr in BP, w/lifestyle modification.

JNC 7—2003
BP Changes & CV Risk

- In people aged 40 – 70, and from BP range 115/75 – 185/115:
  - For every increase of 20 mm Hg in SBP, OR 10 mm Hg in DBP, there is a doubling (2-fold) of the risk for cardiovascular disease.
  - Pre-HTN doubles risk vs normal.
  - Stage 1 doubles risk vs pre-HTN ⇒ 4-fold > normal.
  - Stage 2 doubles risk vs stage 1 ⇒ 8-fold > normal.

- Treatment goal for pts w/HTN and no compelling indications: < 140/90.

Compelling Indications

<table>
<thead>
<tr>
<th>Compelling Indication</th>
<th>Diu</th>
<th>Beta-blocker</th>
<th>ACEI</th>
<th>ARB</th>
<th>CCB</th>
<th>Ald Ant</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart failure</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Post-MI</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td></td>
<td></td>
<td>X</td>
</tr>
<tr>
<td>High coronary dz risk</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td></td>
<td></td>
<td>X</td>
</tr>
<tr>
<td>Diabetes</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td></td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>Chronic kidney dz</td>
<td></td>
<td></td>
<td>X</td>
<td>X</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Recurrent stroke prevention</td>
<td>X</td>
<td></td>
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</tbody>
</table>

Target BP Goals

- General population: < 140/90
- Diabetes: < 130/80
- Chronic kidney disease: < 130/80

Follow Up of BP

<table>
<thead>
<tr>
<th>Initial BP Reading</th>
<th>Recommended F/U</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>Recheck in 2 yrs</td>
</tr>
<tr>
<td>Pre-HTN</td>
<td>Recheck in 1 yr, lifestyle mod</td>
</tr>
<tr>
<td>Stage 1 HTN</td>
<td>Confirm w/in 2 mo</td>
</tr>
<tr>
<td>Stage 2 HTN</td>
<td>Treat w/in 1 month; if &gt;180/110 ⇒ treat immediately or w/in 1 week (depending on clinical situation &amp; complications)</td>
</tr>
</tbody>
</table>
Why We Work-up HTN

3 objectives:
- Assess lifestyle & identify other cardiovascular risk factors or coexisting disorders that may affect prognosis & guide treatment.
- Reveal identifiable causes of HTN.
- Assess presence of target organ damage & cardiovascular disease.

Target Organ Damage

Heart:
- CAD—angina, MI, h/o revascularization.
- Heart failure.
- LVH.

Brain:
- Stroke, TIA.
- Dementia.

Kidney: chronic kidney disease.
- GFR is a better indicator than serum Cr.

Eye: retinopathy.

Vascular: peripheral arterial disease.

Mnemonic: Target Organ Damage

“Heart, Brain
Extremity Pain
Kidneys Fail
Vision wanes”

- (Extremity pain ➔ PAD)
- Any musical genre

Identifiable Causes of HTN—1 (i.e., Secondary HTN)

2 – 10% of hypertensive pts.
- Chronic kidney disease (2.5 – 6%)
- Primary aldosteronism and other mineralocorticoid excess states (1 – 10%)
- Renovascular hypertension (0.2 – 4%)
- Drug-induced or drug-related
  - Mnemonic: CARD or KARD

JNC 7: http://emedicine.medscape.com/article/241381-overview#aw2aab6b2b3aa
Identifiable Causes of HTN—2
- Obstructive uropathy
- Sleep apnea
- Thyroid or parathyroid disease
- Cushing’s syndrome and other glucocorticoid excess states, including chronic steroid therapy
- Coarctation of the aorta
- Pheochromocytoma (rare)

“DOG & CAT PARK”
- Drugs
- Obstructive uropathy
- Glucocorticoid excess
- Renovascular
- Pheochromocytoma
- Apnea (sleep)
- Coarctation of aorta
- Aldosterone
- Thyroid/parathyroid

Identify Cardiovascular Risk Factors
- HTN (Duh!)
- Obesity
- Dyslipidemia
- Diabetes mellitus
- Cigarette smoking
- Physical inactivity
- Renal disease
- Microalbuminuria
- GFR < 60
- Age (>55 for men, >65 for women)
- Family history of premature CVD (men age <55, women age <65)

Mnemonic: CV Risk Factors
- Per JNC-7:
  - Age—65 F (or premature menopause), 55 M
  - BP (HTN)
  - Cigarettes (smoking)
  - DM
  - Exercise lack (physical inactivity)
  - FH—65 F, 55 M
  - Good cholesterol too low (HDL-C <40)
  - High LDL-C
  - Kidney dz
  - Obesity (BMI > 30)—OK, it doesn’t fit in the alphabet, so you just have to remember it!
Diagnostic Workup of HTN

- Assess risk factors and comorbidities.
- Reveal identifiable causes of hypertension.
- Assess presence of target organ damage.
- Conduct history and physical examination.

Diagnostic Workup of HTN – 2

- Laboratory tests:
  - UA
  - Hematocrit
  - Lipid panel
  - Blood chemistry tests:
    - Blood glucose
    - Serum potassium, creatinine, and calcium
  - Optional (unless DM or CKD): urinary albumin/creatinine ratio.
  - Obtain electrocardiogram

Whom to Screen for 2° HTN

- Severe or resistant HTN (uncontrolled on 3 meds of different classes).
- An acute rise in blood pressure over a previously stable value.
- Age <30 years in non-obese, non-black patients with a confirmed negative family history of and no other risk factors (e.g., obesity) for hypertension.
- Malignant HTN (severe HTN + signs of end-organ damage).
- Proven age of onset before puberty.

Additional Clues to 2° HTN

- Hypokalemia—aldosteronism.
- ↑ Cr, abnormal UA – renal dz.
- Snoring, fatigue, daytime somnolence – sleep apnea.

http://www.uptodate.com/contents/who-should-be-screened-for-renovascular-or-other-causes-of-secondary-hypertension?source=see_link
Clinical Clues to Renovascular HTN

- Acute elevation in the plasma creatinine (>30%) after starting ACEI or ARB.
- Systolic-diastolic abdominal bruit that lateralizes to one side.
- And more....

Clinical Clues to Renovascular HTN—2

- Severe HTN w/4 A’s (Age, Atherosclerosis, Atrophic kidney, Acute pulmonary edema).
- Onset of stage II HTN (BP ≥160/100) after age 55.
- Moderate to severe HTN in pts w/diffuse atherosclerosis, esp > age 50.
- Moderate to severe HTN in a pt with an unexplained atrophic kidney or asymmetry in renal sizes of >1.5 cm.
- Moderate to severe HTN in patients with recurrent episodes of acute (flash) pulmonary edema or otherwise unexplained heart failure.

Drug Related Causes of HTN

- Nonadherence
- Inadequate doses
- Inappropriate combinations
- Oral contraceptives
- NSAID’s, COX-2 inhibitors
- Sympathomimetics (decongestants, anorectics)
- Cocaine, amphetamines, other illicit drugs
- Adrenal steroid hormones
- Cyclosporine, tacrolimus
- Erythropoetin
- Licorice (including some chewing tobacco)
- Some OTC supplements and medicines (e.g., ephedra, ma huang, bitter orange)

Screening Tests for 2° HTN

<table>
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<tr>
<th>Diagnosis</th>
<th>Test</th>
</tr>
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<tbody>
<tr>
<td>CKD</td>
<td>eGFR</td>
</tr>
<tr>
<td>Coarctation of aorta</td>
<td>CT angio</td>
</tr>
<tr>
<td>Cushing’s, glucocorticoid excess</td>
<td>Hx, dexamethasone suppression test</td>
</tr>
<tr>
<td>Drugs</td>
<td>Hx, drug screening</td>
</tr>
<tr>
<td>Pheochromocytoma (RARE)</td>
<td>24-hr urinary metanephrine and normetanephrine</td>
</tr>
<tr>
<td>Primary aldosteronism and other mineralocorticoid excess states</td>
<td>24-hr urinary aldosterone level or specific measurements of other mineralocorticoids</td>
</tr>
<tr>
<td>Renovascular HTN</td>
<td>Doppler flow study, MRA</td>
</tr>
<tr>
<td>Sleep apnea</td>
<td>Sleep study with O₂ saturation</td>
</tr>
<tr>
<td>Thyroid/parathyroid disease</td>
<td>TSH, PTH</td>
</tr>
</tbody>
</table>
Why Should We Treat HTN?

- DASH diet has been shown to reduce CAD & stroke.
- Pharmacologic treatment of HTN has been shown to reduce cardiovascular events (NNT to prevent 1 death = 11; NNT = 9 if CAD or target organ damage).
- In unselected population, outcomes are improved regardless of drug regimen.
- More intensive BP lowering leads to more favorable outcomes (↓ stroke & major CV events) – regardless of regimen (for the most part—ACEI + CCB may be more beneficial per one study).

Algorithm for Tx of HTN—1

Algorithm—2a
3 Simple Steps to HTN Tx

1. Lifestyle
2. Compelling indication?
   1. Yes: use appropriate med.
   2. No:
      1. Stage 1: 1 drug
      2. Stage 2: 2 drugs
3. Increase or add.

Non-Pharmacologic Therapy for HTN

<table>
<thead>
<tr>
<th>Modification</th>
<th>Recommendation</th>
<th>~ SBP Reduction</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight reduction</td>
<td>Maintain NI body wt (BMI 18.5 – 24.9)</td>
<td>5 – 20 mm/10kg wt loss</td>
</tr>
<tr>
<td>DASH diet</td>
<td>Fruits, veges, lowfat dairy, low saturated &amp; total fat</td>
<td>8 – 14 mm</td>
</tr>
<tr>
<td>Dietary Na restriction</td>
<td>Max 2.4 g Na = 6 g NaCl</td>
<td>2 – 8 mm</td>
</tr>
<tr>
<td>Physical activity</td>
<td>Regular aerobic activity, ≥ 30 min/day, most days</td>
<td>4 – 9 mm</td>
</tr>
<tr>
<td>Moderate ETOH (vs higher intake)</td>
<td>Max 2/day in men, 1/day in women or lighter persons</td>
<td>2 – 4 mm</td>
</tr>
</tbody>
</table>
Pharmacologic Therapy of HTN

- Monotherapy will control 30-50% of pts.
- Majority of pts require ≥ 2 meds for control.
- The average hypertensive pt is on 2-3 meds (depending on study).
- Vast majority of hypertensives w/ diabetes will require 2 or more meds.
- Key points to consider:
  - How high is the BP?
  - Other conditions or risk factors?
  - Target organ damage?

Choosing a Medication

- ABCD’s:
  - ACE inhibitors & ARB’s
  - Beta blockers
  - Calcium channel blockers
  - Diuretics
- Other “forgotten” meds:
  - Aldosterone receptor antagonists.
  - Central sympatholytics.
  - Alpha blockers.
  - Direct vasodilators.
  - Loop diuretics (better for CKD).

Diuretics (Thiazides)

- Usually the first choice antihypertensive agent.
- If pt is on a med from a different class, thiazides are usually the top choice for the 2nd med to add.
- Reduce morbidity & mortality from CAD.
- “Virtually unsurpassed in preventing the cardiovascular complications of HTN.” – JNC 7
- Inexpensive.
- “Despite the various benefits of diuretics, they remain underutilized.” – JNC 7

Diuretics – Who?

- All pts
- Elderly
- African-Americans
- Isolated systolic HTN
- CHF
- CAD/CAD risk
- Stroke
- Pt already on another med
Diuretics in CKD

- In pts with chronic kidney disease (CKD), use loop diuretic if estimated GFR is below ~ 30 (thiazides less effective).

Diuretic Adverse Effects

- Hypokalemia.
- Hyponatremia.
- Sexual dysfunction in men.

Diuretic Adverse Effects – 2

- Hyperuricemia – though gout is less common.
- Hyperglycemia – but not contraindicated in DM.
  - Minimal increase in glc w/low dose thiazides.
  - Proven beneficial outcomes in DM.
- Hyperlipidemia
  - Little effect at 12.5 mg HCTZ.
  - May be temporary (< 1 yr).
  - Proven beneficial outcomes – ↓ risk of CV events.

Commonly Used Thiazide Diuretics

- HCTZ 12.5 – 25 mg
- Chlorthalidone 12.5 mg
  - Equivalent to half the HCTZ dose (i.e., 12.5 mg chlorthalidone = 25 mg HCTZ).
  - May have longer duration of action & may be more effective.
  - Many outcomes studies done w/this drug.
Thiazide Contraindications
- Drug allergy
- RARE crossover w/sulfa
- Anything else (gout, hypoK, h/o arrhythmia, etc) is a caution, not absolute contraindication.

Angiotensin Converting Enzyme Inhibitors
- Preferred med in:
  - CHF
  - DM 1 & 2
  - CAD
  - High risk for CAD
  - Nephropathy

ACEI Adverse Effects
- Dry cough (5 – 20%)
- Reduced GFR (consider D/C med if >30% reduction).
  - More common in renovascular HTN, CHF, polycystic kidney disease, or CKD.
- Hyperkalemia (3.3 – 11%)
- Hypotension, dizziness, syncope.
  - More likely in CHF, volume depletion (diuretics).

Oddball ACEI Side Effects
- Skin rash (a possibility with just about any med).
- Dysgeusia (taste disturbance) – esp captopril.
- Angioedema (rare)
  - Swelling of lips, tongue, mouth, face.
  - May be more common in elderly (?), African-Americans.
- Neutropenia (rarer)
Angiotensin Receptor Blockers

- "ACE inhibitors without the cough."
- Similar efficacy to ACEI's (both are slightly less potent antihypertensives than other agents like thiazides, CCB's).
- Similar side effect profile to ACEI's.
  - Lower incidence of cough (rare, but it happens).
  - Most pts w/ACEI-induced cough tolerate ARB's.
  - ~1/3 the (already rare) incidence of angioedema.
  - More hypotension than ACEI's (~2X).


Beta Blockers

- Not usually a 1st choice any more unless a compelling or other indication:
  - Post-MI (non-intrinsic sympathomimetic)
  - CHF
  - Rate control in A fib
  - CAD—control of angina

Beta Blockers – 2

- Still useful:
  - Resting tachycardia
  - LVH
  - Migraine
  - Essential tremor (non-cardioselective)
- Labetolol – alpha + beta blocker
- Hypertensive emergencies.
- Pregnancy – preexisting HTN or pre-eclampsia (unlabeled).


β-Blocker Adverse Effects

- AV block.
- Bronchospasm.
- Increased PAD symptoms.
- CHF exacerbation if given in acute stage.
- CNS – overstated, but probably more common in elderly.
  - Fatigue – NNH = 57
  - Depression – no significant increase
- Sexual dysfunction – NNH = 199

http://www.uptodate.com/contents/major-side-effects-of-beta-blockers?source=search_result&selectedTitle=1~150
**β-Blocker Adverse Effects – 2**

- May increase stroke or overall mortality, esp > age 60.
- Impaired glc tolerance; ↑ risk of new onset diabetes.
- Vasodilating β-blockers like carvedilol appear OK.
- Effect may be temporary.
- Still given to post-MI diabetics.
- Adverse lipid effects (labetolol may be least likely).

**β-blocker Contraindications**

- Active bronchospasm
- Severe bradycardia
- Heart block > 1st (if no pacemaker)
- Pulmonary edema
- Hypotension with or without shock
- Overt heart failure should be brought under medical control 1st
- Most pts w/MI d/t cocaine should not be treated with beta blockers (risk of coronary artery spasm)

http://www.uptodate.com/online/content/topic.do?topicKey=acute_co/7620&sourc=see_link

**Common/Significant Drug Interactions With β-Blockers**

<table>
<thead>
<tr>
<th>Drug</th>
<th>Effects</th>
<th>Recommendation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amiodarone</td>
<td>Cardiac arrest</td>
<td>Extreme caution</td>
</tr>
<tr>
<td>Antidiabetic agents</td>
<td>HTN, poss ↓ glc</td>
<td>Monitor</td>
</tr>
<tr>
<td>Rate-sparing CCB (diltiazem, verapamil)</td>
<td>Brady, CHF, hypotension</td>
<td>Avoid (few clinical issues, however)</td>
</tr>
<tr>
<td>Digoxin</td>
<td>Worsening bradycardia</td>
<td>Monitor. OK in angina + low EF (? benefit)</td>
</tr>
<tr>
<td>Epinephrine, sympathomimetics</td>
<td>HTN crisis</td>
<td>Avoid if possible</td>
</tr>
<tr>
<td>Lidocaine</td>
<td>↑ lido level</td>
<td>↓ lido dose (NOT listed in ACLS protocol)</td>
</tr>
</tbody>
</table>

**Calcium Channel Blockers (CCB)**

- Dihydropyridines: use long acting meds
  - Amlodipine
  - Felodipine
- Non-dihydropyridines:
  - Diltiazem
  - Verapamil
- Avoid short acting dihydropyridines
  - Nifedipine (this is often an incorrect answer, esp if another CCB option).
**CCB Candidates**
- African-Americans
- Elderly
- Angina, incl Prinzmetal's (amlodipine, felodipine).
- Caution:
  - DM – 1 study showed incr MI risk vs ACEI, but may have actually been due to greater ACEI benefit.

**CCB Adverse Effects**
- Edema
  - More likely w/DHP's
- Dizziness
- HA
  - BUT: Verapamil may be used in migraine prophylaxis.
- Reflex tachycardia – DHP’s
- Non-DHP SE’s:
  - Bradycardia
  - AV block
  - CHF exacerbation

**CCB Comparison**

<table>
<thead>
<tr>
<th>Drug</th>
<th>Periph Resis</th>
<th>HR</th>
<th>Conduction</th>
<th>Contractility</th>
</tr>
</thead>
<tbody>
<tr>
<td>DHP's</td>
<td>↓↓↓</td>
<td>↑</td>
<td>+/-</td>
<td>+/-</td>
</tr>
<tr>
<td>Diltiazem</td>
<td>↓↓</td>
<td>↓</td>
<td>↓</td>
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<tr>
<td>Verapamil</td>
<td>↓↓↓</td>
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</tr>
</tbody>
</table>

•DHP’s ➔ more peripheral effects.
•Non-DHP’s ➔ more cardiac effects.

**Alpha-Adrenergic Blockers**
- Prazosin, terazosin, doxazosin.
  - Orthostatic hypotension & syncope, esp 1st dose.
  - Not usually used in initial monotherapy, except sometimes in men w/BPH, esp if low-mod CV risk.
    - More CV events & CHF than thiazide—but there was no control (untreated) group.
    - May enhance hypotensive effects of PDE-5 drugs for erectile dysfunction (sildenafil, vardenafil, tadalafil).
Central Sympatholytic Agents

- Clonidine
  - Dry mouth, constipation, sedation.
  - Can cause bradycardia, heart block.
  - Rebound HTN upon withdrawal.
- Methyldopa
  - Can be used in pregnancy.
  - Rare lupus-like syndrome.
  - Rare hemolytic anemia.
  - Can cause hepatitis, esp in pts with liver dz.

Direct Vasodilators

- Mainly for resistant HTN.
- Hydralazine
  - Can be used in pregnancy (unlabeled).
  - ANA + lupus-like syndrome.
  - HA, tachycardia (caution in angina/CAD).
  - May be useful for CHF in African-Americans (unlabeled).
- Minoxidil
  - Hirsutism.
  - Pericardial effusion.
  - EKG changes (T wave changes common).

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Reverse-Engineering Compelling Indications

- CAD/Angina:
  - Beta-blocker.
  - Add if needed: long acting DHP CCB, ACEI, diuretic
    - Long acting DHP OK if contraindication to β-blocker.
  - Caution w/β-blocker + non-DHP CCB (verapamil, diltiazem) – bradycardia, heart block, CHF.
- Post-MI: β-blocker; may add ACEI.
- CHF: Diuretic, ACEI or ARB, β-blocker (carvedilol, metoprolol succinate, bisoprolol), aldosterone antagonist.
Reverse-Engineering Compelling Indications – 2

- DM:
  - Diuretics (yes, they are safe and reduce mortality), ACEI or ARB, CCB.
  - β-blocker OK if CAD.
- CKD: ACEI or ARB.
- 2° prevention of stroke: diuretic or diuretic + ACEI.

Demographic Considerations

- Elderly & African-Americans respond best to thiazide diuretics or CCB’s.
- Less responsive to ACEI or ARB.
- ACEI/ARB responsiveness improves if given diuretic.
- Still use β-blocker after MI.
- African-Americans may respond better to Na⁺ restriction.
- Young pts may respond better to ACEI’s & beta-blockers.

Combination Therapy

- Preferred combinations:
  - ACEI or ARB + diuretic.
  - ACEI or ARB + CCB.
- Acceptable combinations:
  - Thiazide + most others (β-blocker, CCB, K-sparing diuretic).
  - β-blocker + DHP CCB.

Hypertensive Emergencies

- Def: acutely elevated BP with end organ damage (also called malignant HTN).
- Eye: retinal hemorrhages, exudates, or papilledema.
- Brain: hypertensive encephalopathy.
- Heart: angina, ischemia, CHF, dissecting aortic aneurysm.
- Kidney: hematuria, proteinuria, ARF.
- Goal: reduce BP by 25% within 1 hr, then to 160/100-110 within next 2-6 hrs.
Treatment of HTN Emergencies

- **Na' nitroprusside.**
  - Rapid action, short duration = titratable drip.
  - Cyanide toxicity with prolonged use, esp w/renal failure.
  - Caution w/high intracranial pressure.
  - Continuous BP monitoring.
- **Nicardipine**—longer half-life, harder to titrate.
  - Avoid in acute CHF.
  - Caution in coronary ischemia.
- **Clevidipine** – shorter acting, more titratable.


Treatment of HTN Emergencies

- **Labetolol**—safe in CAD; caution/avoid in asthma, COPD, CHF (avoid in acute HF), bradycardia, >1° heart block.
- **Esmolol**—ultra-short acting β-blocker.
  - Aortic dissection.
- **Fenoldopam**—dopamine receptor agonist.
  - Useful in renal failure.
  - Contraindicated in glaucoma.
- **Phentolamine**—catecholamine excess (pheochromocytoma).