- increase mortality

Diagnosis

- 3-5 minute rest
- Arm at level of heart
- Both side measure and take higher
- elderly 1st time ← supine sitting erect
- no caffine/stimulant 1hour before / no smoking 30 minutes before
- good weather
- cuff size width 40% of upper arm
- must REMOVE cloth
- 2 readings in 2 visits separated 5-10minutes => must be elevated in all 4 readings
- Technique
  - ↳ cuff on arm + stetoscope on brachial A. (NOT UNDERCLOTH)
  - ↳ blow upp until you hear nothing
  - ↳ deflate cuff 3mm Hg/sec. → make note hear of when you start hear any sound because difference btw (1st - sys) must be 10mmHg
  - ↳ 1st sound isn't systolic. Systolic is when you hear the sound consistently
  - ↳ continue to deflate until you hear no voices => diastolic BP

or else if more, pulsus paradoxis

- AMBULATORY BP
  - ↳ measures BP throughout Day
  - ↳ used when BP picture in clinic doesn't match clinical picture (patient with endorgan damage, but BP normal or BP is high with no symp.)
- Home BP
  - ↳ must teach
  - ↳ goal is less, because it is expected to be less in home

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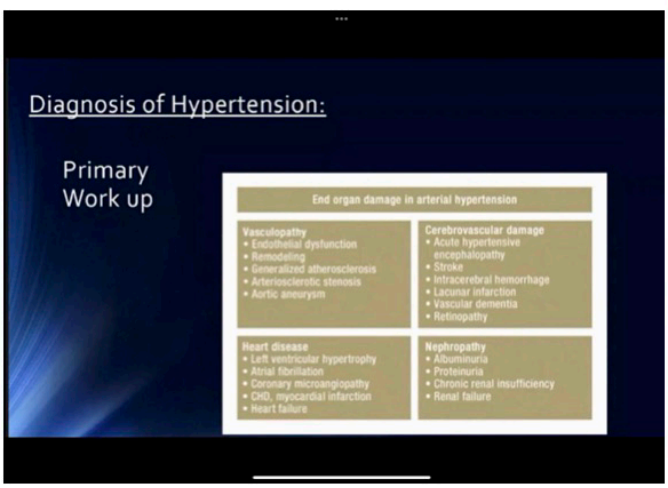
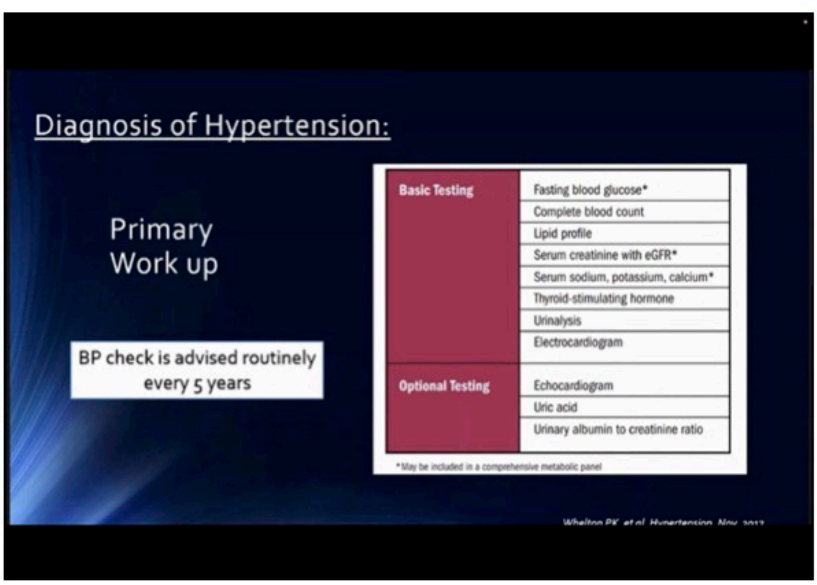
White coat HTN vs Masked HTN

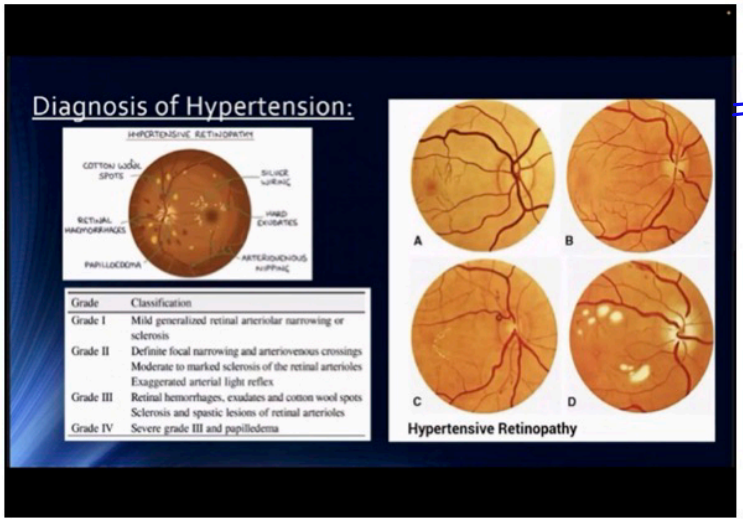
- White coat HTN
  - ↳ HTN in office but not in home
  - ↳ not bad as actual HTN, but must be treated
    - lifestyle modification
    - meds
- Masked HTN
  - ↳ No clinic HTN but is Hypertensive in Home
  - ↳ hard to catch because is normotensive in clinic
  - ↳ could find unexplained endorgan damage

Primary workup

- ↳ Eyeon workup
- ↳ basic testing







=> Retinopathy of HTN

Secondary HTN
↳ look at slides

- Renovascular
  - Fibromuscular: women + young => gives string of beads appearance on Renal angio
  - Atherosclerotic
    - ↳ flash pulmonary edema
    - ↳ bad prognosis
    - ↳ no ACE-I
- Primary Aldosteronism
  - ↳ hypokalemia
  - ↳ to diagnose => plasma aldosterone renin ratio
- OSA
  - ↳ obese, snoring, daytime sleeping
  - ↳ CPAP
- Aortic coarctation
  - ↳ Upper hypertensive with lower hypotensive (radiofemoral delay on exam)
  - ↳ Associated with bicuspid Aortic valve
  - ↳ rib notching on X Ray
- Alcohol / Amphetamines / caffiene / Immunosupp => cyclosporin / steroids / SSRI / OCP / decongestant.

Management

- normal BP < 120/80 → lifestyle modification
- grayzone BP 120-129/80 → lifestyle modification
- Stage 1 BP 130-139/80-89 → if CVD risk <10% → life style modifications; if CVD risk >10% → medications
- Stage 2 BP >140/90 → if CVD risk <10% → life style modifications; if CVD risk >10% → medications

  if CVD risk >10% or DM2 / Chronic venal insufficiency / PAD → medications

  => goal < 130/80
- severe HTN BP >160/100
  - ↳ must work quickly

- Nonpharm. Treatment
  - ↳ weight loss
  - ↳ ↓ salt
  - ↳ physical Activity
  - ↳ ↓ Alcohol

First choice is < CCB, ACEI } Because HF friendly
Thiazide → African American

Dihydroperidine (Never give nondihydroperidine CCB for patien with HF)

use 2 agent at lower dose than 1 agent at higher doses

2 agents for < Stage 2 HTN
Average BP >20/10 mmHg above their BP target

- Chlorthanidone is best Thiazide of BP
- ACE + ARBS → careful for kidneys + pregnancy
- Diltiazem + Verapamil (nondihydroperidine) → Avoide in HF
- Amlodipine (dihydroperidine) → v. good
- B blocker → not first line
  → used if secondary cause/diseases
  → don't use Atenolol
  - methyl dopa in pregnant
  - α_1 antagonist → v. hard to use in elderly

Comorbidities

HF
- must be aggressive in treatment of HTN
- <130/80

4 ways for HF with reduce Ejection fraction HFrEF
1. ACEI/ARB + Arni (neprolysin inhibitor)
2. BB
3. mineral corticoid receptor Antagonist (Spironolactone/eplerenone)
4. DM meds

- Avoid nondihydroperidine CCB < Diltiazem, Verapamil in HF

in HFpEF (HF with preserved Ejection Fraction)
↳ must treat HTN
↳ ACEI + ARB
↳ B Blocker

Stable coronary Disease
- B Blocker
- ACEI / ARB
- Dihydroperidine CCB (Amlodipine)
- could use Diuretic
- never use Atenolol

Acute coronary Syndrom
- b blocker
- nitro glycerine

CKD
- goal is <130/80
- If with Albuminuria → ACE / ARB

acute Intracerebral Hemorrhage

150-220 mmHg
↳ you might observe because Brain hemorrhage and < 140 mmHg is worse than leaving at BP elevated

>220 mmHg
↓
must lower

Ischemic stroke

could give Thrombolysis
↓
if acute and could give thrombolysis
↳ reduce BP less than <185/<110 mmHg before giving Thrombolytic

couldn't give thrombolysis
↓
no thrombolysis therapy
↳ reduce < 220/110 mmHg

don't introduce any HTN medication if BP is < 220 mmHg/110

DM
- ACE/ARB

AFib
- ACE ARB

Aortic Disease
- Never Bblocker

Hypertensive Crisis
↳ >180/>120

unless you have
- Ischemic stroke
- ICH
- Head Trauma

↳ must check their own guidline

if organ damage
↳ Hypertensive Emergency
↓
look at associated condition:
1. Aortic dissection
   ↳ reduce BP to <120 in 1 HOUR
   ↳ use Nitroprusside
2. pre Eclampsia
   ↳ mg SO_4
3. pheochromocytoma crome
   ↳ labetalol

} reduce BP <140 in 1 hour

if no organ damage
↳ hypertensive Ergency
↳ adjust drugs and educate + give clinic visit

} If emergency and not these:
1. Reduce BP by 25 % max over 1 hour
2. then 160/100 over 2-6 hour
3. then normal over 24-48 h

Drugs:
1. CCB IV
2. Nitroprusside
3. Nitroglycerine ← HF, Acute coronary syndrome, Pulm. edema
4. hydralazin
5. esmolol (B selective short acting)
6. Fenoldopam (selective dopamine)
   ↳ in nephritic

- Resistant HTN
  - 3 Agent (x Diuretics) with optimal doses
- Refractory HTN
  - 4 Agent (x Diuretics) with optimal doses
- Pseudoresistant
  - looks uncontrolled but there is problem with:
    - wrong BP reading
    - no compliant
    - Suboptimal treatment
    - white coat HTN
    - No lifestyle modification

Risk factor of Resistant, check slides

Risk Factors

PATIENT RELATED	POTENTIALLY REVERSIBLE
• Higher baseline BP (particularly systolic)	• Suboptimal therapy
• Presence of LVH	• Lifestyle and diet
• Older age	• Medications
• Obesity	• Extracellular volume expansion
• African-American race	• Secondary causes of hypertension
• Chronic kidney disease	• OSA
• Diabetes	

Risk Factors

- Medications
  - Can raise BP or reduce the response to antihypertensive drugs
  - Most commonly implicated agents are:
    - NSAIDs
      - NSAIDs can interfere with the antihypertensive effect of virtually any agent, except calcium channel blockers
    - Sympathomimetics
      - Diet pills
      - Decongestants
      - Amphetamine-like stimulants
      - Cocaine
      - Alcohol
    - Glucocorticoids
    - Estrogen-containing contraceptives
    - Erythropoietin
    - Herbal preparations (ephedra or ma huang)
    - Natural Licorice
    - Calcineurin inhibitors (cyclosporine and tacrolimus)
    - Antidepressants

Risk Factors

- Suboptimal therapy
- Lifestyle and diet
  - Obesity
  - High-salt diet
  - Physical inactivity
  - Heavy alcohol intake
- Extracellular volume expansion
  - Renal insufficiency
  - Sodium retention due to therapy with vasodilators
  - Ingestion of a high-salt diet (which can be assessed by measuring sodium excretion in a 24-hour urine collection)

management

steps
1. check if not pseudoresistant
2. substitute diuretic to thiazide – LIKE diuretic (chlorthalidone)
3. add mineralocorticoid receptor blocker (spironolactone)
4. add Bblocker (carvedilol / labetalol)
5. add clonidine
6. reevaluate for secondary
7. Add hydralazine ± isosorbide
   - minoxidil
   - renal Denervation