- There is a lot of evidence that many poorly controlled hypertensives have actually hyperaldosteronism hence Aldo blockers esp Aldactozide should always be tried in pts with refractory hypertension
- in children BP increases with age, RR decreases with age, and HR starts at 120 then peaks at 150 at 2mo of age and then back to nl range as a teenager
- SBP increases with age and DBP increases with age to 50yo then decreases with age hence old pts have wide pulse pressure
- DBP is more important for younger pts vs SBP is more important for older pts b/c vessels calcify making DBP and MAP unreliable
- for every increase in stage (20/10) you have a double increase in CV risk
- measure BP in AM b/c that is when highest therefore if in control in AM then likely in control the rest of the day
- if HTN goal BP is <140/<90 (you want to avoid hypotension) or if pt has DM/CKD then <130/<80 (more a aggressive) BUT remember that nl BP is <120/<80
- don't use CCB for BP control post-MI b/c it has been found to increase r/o death
- In AA don't use ACE-I/ARB b/c they are already in a low renin state, CCB/Thiazides work great in AA
- There is evidence that hyperuricemia is a predictor for the development of HTN and animal studies suggest that increased uric acid causes HTN. A JAMA article found that giving allopurinol to adolescents with newly diagnosed hypertensive pts resulted in a reduction of BP.
- Independent of BMI and fat content pts w/ HTN have higher insulin levels and more insulin resistance
- White coat HTN seen in 15% of pts
- Pt should be resting for 5min in a chair (not exam table) before checking BP with arm at level of heart
- The key to Tx is being aggressive meaning titrate meds on a weekly basis by having pts report BP to you via email
- ALLHAT (Antihypertensive and Lipid Lowering to prevent Heart Attack Trial) compared thiazides, ACE-I, D-CCB, and AB, the trial showed that there was no difference b/t all the drugs and b/c thiazides were cheapest at the time they were then considered first line agents, the problem with this study was that it did not look at the long term effects of these meds and what is now known is that thiazides have lots of problems including hypoNa, DL, hyperglycemia, etc.
- In general don't ever use scheduled hydralazine, minoxidil, or clonidine
- New secondary causes include increased renal salt absorption
- Thiazides are particularly effective in salt sensitive HTN like AA patients
- ? that long term use of diuretics is associated w/ T2DM therefore avoid in young pts
- CKD causes HTN b/c of increased renal sodium retention and increased peripheral vascular resistance and RAAS activation
- CKD most common cause of secondary HTN

Hypertension in Pregnancy

- Why important?
  - occurs in 10% of pregnancy w/ 50% 2/2 a specific pregnancy related disorder
  - leading cause of maternal/fetal M&M
  - normally progesterone mediates smooth muscle relaxation → ↓BP up to mid 2nd trimester, plateaus, then rises back to normal during 3rd trimester
- Types
  - **Chronic Pre-Existing HTN** (HTN b/f 20wks GA, NB 1/3 will develop preeclampsia as such always follow proteinuria, if <160/<110 then no Tx but if > then Tx w/ methyldopa (most studied but least effective), few BB (labetalol, et al but not atenolol b/c IUGR), CCB (nifedipine), hydralazine, AVOID clonidine, NEVER use ACE-I/ARB/DRI and diuretics
  - **Pregnancy Induced HTN (PIH) aka Gestational HTN (GH) → Pre-Eclampsia → Eclampsia**
    - **Preeclampsia**: PIH + Nondependent Edema + End Organ Damage w/o Seizures ((1) UPI 2/2 decreased bld flow to placenta eventually causing IUGR if chronic or placental infarct, abruption, stillbirth if acute (2) Severe Proteinuria: >5000mg on 24hr urine OR >4+ protein on urine dipstick, (3) ARF w/ Oliguria, (4) Stroke, (5) HA and Scotomata, (6) Pulmonary Edema, (7) RUQ pain due to subcapsular hematoma with subsequent elevated LFTs, (8) Hemolytic Anemia, Thrombocytopenia and DIC), Tx: bedrest/betamethasone until delivery and MgSO4 (watch for hypermagnesemia) and antihypertensives (only if >160/110 b/c you can compromise fetal blood flow if too low) deliver if possible if signs of impending eclampsia (hyperreflexia, headaches, epigastric pain)
    - **Eclampsia**: Severe PIH + Severe Nondependent Edema + Severe End Organ Damage (" ") + Grand Mal Tonic-Clonic Seizures THAT CANNOT BE ATTRIBUTED TO ANY OTHER CAUSE, Tx: same, Classic Presentation: preeclampsia usually manifests after 20wk but more commonly in 3rd trimester particularly near term (if HTN is seen b/f 20wk suspect a molar pregnancy or undiagnosed chronic HTN) even though delivery is the cure for preeclampsia some patients actually worsen acutely after delivery and then get better due to increased placental Ag exposure during L&D process (hence seizure prophylaxis for up to 24hrs after delivery) some pts maintain HTN for wks after delivery (hence antihypertensives are given for weeks after delivery) after first preeclamptic pregnancy there is a 30% recurrence (70% if pt also has chronic HTN), Etiology: Genetics (possible paternal) → Abnormal Placentation (normally when the placenta invades the uterine wall, spiral arteries remodel into a low pressure / high volume system BUT when placentation is bad this remodeling does not occur such that as blood runs through the unremodeled arteries they are damaged releasing toxins which cause widespread vasospasm and vascular injury), RFs: Hx, chronic HTN, "Big Placentas" (DM, Twins, Molar Pregnancies), chronic RF, SLE, African American, Advanced (>35yo) or Early (<20yo), Maternal Age, Nulliparity, Multiple Gestation, Abnormal Placentation, New Paternity (if mother is a multip with one father but then

conceives a another baby with a NEW father she is back to nulliparity so to speak such that she is back to an increased risk for preeclampsia), cohabitation (nulliparous mothers who live father for >1yr have lower risk vs mothers who conceive <1yr of living with father), Hx in a female relative of the father (like mother-in-law)

Hypertensive Urgency = acute rise in BP (>180/>120) = decrease MAP by <25% over a period of hours on day one with ORAL agents then on subsequent days bring BP to wnl

- captopril (Capoten) (onset in 15-30min) 25mg PO Q2hrs until desired MAP achieved
- nicardipine (Cardene) (onset in 30-60min) 30mg PO Q8hrs until desired MAP achieved
- labetalol (Trandate) (onset in 60-120min) 200mg PO Q4hrs until desired MAP achieved
- clonidine (Catapres) (onset in 15-30min) 0.1-0.2mg PO Q1hr until desired MAP achieved

Hypertensive Emergency aka **Malignant Hypertension** = acute rise in BP + evidence of organ dysfunction = decrease MAP by <10% in the first few minutes then <15% more over a period of few hours on day one with IV agents then on subsequent days bring BP to wnl w/ PO agents

- If
    - Cocaine then Nitroglycerine
    - HTN Encephalopathy then Nitroprusside and if that fails then Beta-Blocker or Nicardipine
    - Subarachnoid Hemorrhage then Nimodipine and if that fails then Beta-Blocker or Nicardipine
    - CVA then Beta-Blocker and if that fails then Nitroprusside
    - ARF then Nicardipine and if that fails then Fenoldopan
    - Cardiac Ischemia then Nitroglycerine + Beta-Blocker and if that fails then Nitroprusside
    - Surgical Pt then Fenoldopan
- labetalol (Trandate) (onset in 5-10min) 20-80mg IV Q10min until desired MAP achieved and then 0.5-2mg/min and titrate to keep desired MAP
- esmolol (Brevibloc) (onset in 1-2min) 500mcg/kg IV Q5min until desired MAP achieved and then 100mcg/kg/min and titrate to keep desired MAP
- nitroprusside (Nipride) (onset immediately) 0.25-10mcg/kg/min IV and titrate to keep desired MAP (remember SEs)
- nicardipine (Cardene) (onset in 1-5min) 5-15mg/hr IV and titrate to keep desired MAP
- nimodipine (Nimotop) ?
- fenoldopam (Carlopam) (onset in 1-5min) 0.1-0.3mcg/kg/min IV and titrate to keep desired MAP
- nitroglycerine (Tridyl) (onset in 2-5min) 5-100mcg/hr IV and titrate to keep desired MAP

Reference Card From the

Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC 7)

Evaluation

Classification of Blood Pressure (BP)*

Category	SBP† (mm Hg)		DBP‡ (mm Hg)
Normal	<120	and	<80
Prehypertension	120–139	or	80–89
Hypertension, Stage 1	140–159	or	90–99
Hypertension, Stage 2	≥160	or	≥100

*See Blood Pressure Measurement Techniques (reverse side).
†SBP=systolic blood pressure; ‡DBP=diastolic blood pressure.

Diagnostic Workup of Hypertension

- Assess risk factors and comorbidities
- Reveal identifiable causes of hypertension
- Assess presence of target organ damage
- Conduct history and physical examination
- Obtain laboratory tests: urinalysis, blood glucose, hematocrit and lipid panels, serum potassium, creatinine, and calcium. Optional: urinary albumin/creatinine ratio
- Obtain electrocardiogram

Assess for Major Cardiovascular Disease (CVD) Risk Factors

- Hypertension
- Obesity (body mass index ≥30 kg/m²)
- Dyslipidemia
- Diabetes mellitus
- Cigarette smoking
- Physical inactivity
- Microalbuminuria, estimated glomerular filtration rate <60 mL/min
- Age (>55 years for men, >65 years for women)
- Family history of premature CVD (men age <55 years, women age <65 years)

Assess for Identifiable Causes of Hypertension

- Sleep apnea
- Drug induced/related
- Chronic kidney disease
- Primary aldosteronism
- Renovascular disease
- Cushing's syndrome or steroid therapy
- Pheochromocytoma
- Coarctation of aorta
- Thyroid/parathyroid disease

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Treatment

Principles of Hypertension Treatment

- Goal BP <140/90 mm Hg or BP <130/80 mm Hg in patients who have diabetes or chronic kidney disease
- Majority of patients will require two medications to reach goal

Algorithm for Treatment of Hypertension

Lifestyle Modifications

Not at Goal Blood Pressure (<140/90 mm Hg) (<130/80 mm Hg for patients who have diabetes or chronic kidney disease)
- See Strategies for Improving Adherence to Therapy

Initial Drug Choices

Without Compelling Indications		With Compelling Indications
Stage 1 Hypertension (SBP 140–159 or DBP 90–99 mm Hg) Thiazide-type diuretics for most. May consider ACEI, ARB, BB, CCB, or combination	**Stage 2 Hypertension** (SBP ≥160 or DBP ≥100 mm Hg) Two-drug combination for most (usually thiazide-type diuretic and ACEI, ARB, BB, or CCB)	**Drug(s) for the Compelling Indications** See Compelling Indications for Individual Drug Classes Other antihypertensive drugs (diuretics, ACEI, ARB, BB, CCB) as needed

Not at Goal Blood Pressure

Optimize dosages or add additional drugs until goal blood pressure is achieved. Consider consultations with a hypertension specialist.
- See Strategies for Improving Adherence to Therapy

Blood Pressure Measurement Techniques

Method	Notes
In-office	Two readings, 5 minutes apart, sitting in chair. Confirm elevated reading in contralateral arm.
Ambulatory BP Monitoring	Indicated for evaluation of "white coat hypertension." Absence of 10–20% BP decrease during sleep may indicate increased CVD risk.
Patient Self-check	Provides information on response to therapy. May help improve adherence to therapy and is useful for evaluating "white coat hypertension."

Causes of Resistant Hypertension

- Improper BP measurement
- Excess sodium intake
- Inadequate diuretic therapy
- Medication
  - —Inadequate doses
  - —Drug actions and interactions (eg, nonsteroidal anti-inflammatory drugs [NSAIDS], illicit drugs, sympathomimetics, oral contraceptives)
  - —Over-the-counter (OTC) drugs and herbal supplements
- Excess alcohol intake
- Identifiable causes of hypertension (see reverse side)

Compelling Indications for Individual Drug Classes

Compelling Indication	Initial Therapy Options
Heart failure	THIAZ, BB, ACEI, ARB, ALDO ANT
Postmyocardial infarction	BB, ACEI, ALDO ANT
High CVD risk	THIAZ, BB, ACEI, CCB
Diabetes	THIAZ, BB, ACEI, ARB, CCB
Chronic kidney disease	ACEI, ARB
Recurrent stroke prevention	THIAZ, ACEI

THIAZ = thiazide diuretic; ACEI = angiotensin converting enzyme inhibitor; ARB = angiotensin receptor blocker; BB = beta blocker; CCB = calcium channel blocker; ALDO ANT = aldosterone antagonist.

The National High Blood Pressure Education Program is coordinated by the National Heart, Lung, and Blood Institute (NHLBI) at the National Institutes of Health. Copies of the JNC 7 Report are available on the NHLBI Web site at **http://www.nhlbi.nih.gov** or from the NHLBI Health Information Center, P.O. Box 30105, Bethesda, MD 20824-0105; Phone: 301-592-8573 or 240-629-3255 (TTY); Fax: 301-592-8563.

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Strategies for Improving Adherence to Therapy

- Clinician empathy increases patient trust, motivation, and adherence to therapy
- Physicians should consider their patients' cultural beliefs and individual attitudes in formulating therapy

Principles of Lifestyle Modification

- Encourage healthy lifestyles for all individuals
- Prescribe lifestyle modifications for all patients with prehypertension and hypertension
- Components of lifestyle modifications include weight reductions, DASH eating plan, dietary sodium reduction, aerobic physical activity, and moderation of alcohol consumption

Lifestyle Modification Recommendations

Modification	Recommendation	Average SBP Reduction Range*
Weight reduction	Maintain normal body weight (body mass index 18.5–24.9 kg/m²)	5–20 mm Hg/10 kg
DASH eating plan	Adopt a diet rich in fruits, vegetables, and lowfat dairy products with reduced content of saturated fat	8–14 mm Hg
Dietary sodium reduction	Reduce dietary sodium intake to ≤100 mmol (2.4 g sodium or 6 g sodium chloride)	2–8 mm Hg
Aerobic physical activity	Regular aerobic physical activity (eg, brisk walking) at least 30 minutes per day, most days of the week	4–9 mm Hg
Moderation of alcohol consumption	Men: limit to ≤2 drinks† per day. Women and lighter-weight persons: limit to ≤1 drink† per day	2–4 mm Hg

*Effects are dose and time dependent.
†1 drink = 1/2 oz or 15 mL ethanol (eg, 12 oz beer, 5 oz wine, 1.5 oz 80-proof whiskey).