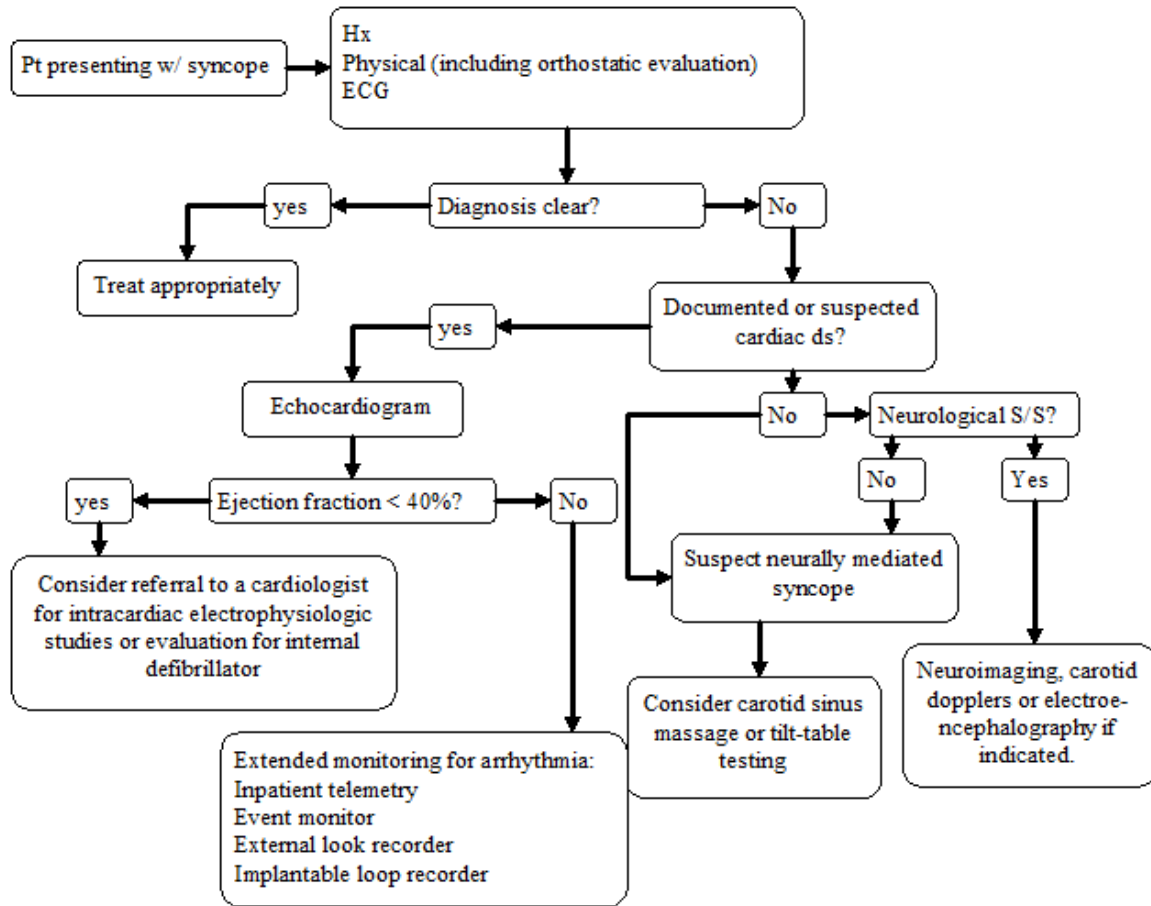


Chapter 20 Syncope

- I. Prevalence and Impact
 - a. Definition
 - i. Sudden loss of consciousness and postural tone with spontaneous recovery
 - b. During a 10 year period approximately 6% of adults have syncope. The prevalence rises steeply, however, after age 70.
 - c. Approximately 1.3% of all US hospitalizations had the discharge diagnosis of syncope, which translates to an annual cost of \$2.4 billion.
 - d. Diagnostic testing is applied inconsistently, specific therapies are often not begun and a large number of syncopal events remain undiagnosed.
 - e. A more stringent use of algorithms and evidence-based guidelines in the evaluation and management of patients with syncope are needed.
- II. Risk Factors & Pathophysiology
 - a. Underlying pathophysiology is inadequate oxygenation of the cerebral cortex and Reticular Activating System (RAS)
 - i. A variety of mechanisms may lead to this condition.
 - b. General Categories
 - i. Neurally mediated (reflex) causes
 - ii. Orthostatic hypotension
 - iii. Cardiac causes
 - iv. Central nervous system diseases
 - v. Psychiatric disorders
 - c. History of stroke or transient ischemic attack was, by far the greatest potential risk factor.
 - i. Use of cardiac medication and diagnosis of HBP were also significant predictors
 - d. Age-related physiological changes that increase risk for syncope
 - i. Atherosclerosis
 1. (impairing dilation of cerebral blood vessels in the face of reduced blood flow)
 - ii. Increased endothelin production
 1. increasing vasoconstriction of cerebral arterioles
 - iii. Left ventricular dysfunction, due to longstanding hypertension and/or heart disease
 1. causing decreased cardiac output
 - iv. blunting of autonomic response
 1. predisposing the person to orthostatic hypotension
- III. Differential Diagnosis & Assessment
 - a. Dif-dx is long and many cases go undiagnosed.
 - i. In geriatrics perhaps half are multifactorial
 - b. Neurally mediated (reflex): involves the initiation of reflexes that lower BP or slow HR usually through vagal stimulation.
 - i. May be triggered by urination, defecation, cough, GI stimulation (especially pain) and stimulation of the carotid (tight collar) and intense emotions.

- ii. Considered benign but has a high prevalence.
 - iii. May be difficult to diagnose due to multiple factors
 - iv. Vasovagal or neurocardiogenic is the most common type: including the common faint.
1. typically involves prolonged standing, emotional distress or exertion in warm environment causing peripheral pooling and a drop in blood return to the heart. Heart tires to compensate for decreased preload by contracting harder thereby activating ventricular mechanoreceptors that start a reflex causing the CNS to stimulate vasodilation and bradycardia.
 - v. Symptoms include: nausea/vomiting, prolonged standing, hot environments and unpleasant situations.
 - vi. be cautious not to assume this diagnosis in pts with heart disease or repetitive episodes of syncope.
 - vii. Carotid sinus syndrome is often listed as a cause
 1. In most cases the provocation of syncope with carotid sinus massage indicates a susceptibility to neurally mediated syncope rather than a diagnosis
 - c. Orthostatic Hypotension
 - i. a drop in arterial pressure that occurs when an individual moves to an upright position. Typically, the ANS rapidly compensates for this by increasing the venous tone in the legs, when this symptom fails, syncope may occur.
 - ii. when circulating blood volume is low, as in dehydration, orthostatic hypotension and syncope may occur even with appropriate autonomic compensation.
 - iii. should be considered in individuals on meds that predispose to hypotension, have reason to be dehydrated or have autonomic insufficiency from a neurological disorder such as parkinson's.
 - iv. typically happens after standing up or after prolonged sitting in a hot, crowded environment.
 - v. Defined as a drop ≥ 20 mmHg in systolic BP or a ≥ 10 mmHg in diastolic BP. 3 minutes after assuming the upright position.
 - vi. 3 general mechanisms can lead to this orthostatic syncope (or near-syncope) that does not meet the definition of orthostatic hypotension:
 1. Early orthostatic hypotension (i.e. a drop in blood pressure immediately upon standing that becomes normalized within 3 mins)
 2. Late orthostatic hypotension (i.e. a drop in BP that occurs after prolonged standing)
 3. Impaired cerebral perfusion and syncope with mild decreases in systemic blood pressure (often due to atherosclerotic narrowing of the carotid and/or vertebral arteries)
 - d. Cardiac syncope
 - i. Occurs when reduction in cardiac functioning by arrhythmia, death of myocardium, or outflow obstruction leads to decreased blood flow to the brain.
 - ii. most deadly of syncope types

- iii. cardiac cause should be considered when syncope is preceded by palpitations or chest pain, or when it occurs during exertion
 - iv. pts with known severe structural heart disease should be considered to have cardiac syncope until proven otherwise.
 - e. Cerebrovascular disease
 - i. rare but plausible cause of syncope.
 - ii. most TIA or strokes do not cause LOC
 - f. Psychiatric causes
 - i. should be considered in pts with repetitive syncope of unknown origin after cardiac causes have been ruled out.
 - ii. more common in younger patients.
 - iii. Prodromal symptoms, such as dizziness, are common.
 - iv. Hyperventilation may increase susceptibility to neurally mediated syncope.
 - v. Also pseudopsyncope,; describes pts with syncope of unknown but presumed psychiatric origin
- 1. no pathological changes, no change in BP or pulse
- IV. Evaluation of the patient with syncope
 - a. Most common cause of syncope in elderly is one of the neurally mediated syndromes. However, cardiac syncope is also quite common and, if untreated, is associated with a higher risk of death.
 - i. **Evaluation of the patient involves parallel process of seeking a specific diagnosis and ruling out cardiac causes.**
 - ii. In most cases hx, examination and ECG are sufficient
 - iii. Pts w/out a dx, but with a high likelihood of cardiac disease should have additional studies.
 - b. Hx, exam and ECG has high diagnostic yield and is cost effective
 - i. Hx
 - 1. can identify symptoms and situations surrounding syncope that can help diagnose 3 common etiologies: neurally mediated (vasovagal), orthostasis and drug related.
 - ii. Physical exam
 - 1. may uncover signs of cardiovascular or neurologic process as well as orthostasis.
 - iii. ECG (electrocardiogram)
 - 1. has little yield alone, due to intermittent nature of arrhythmias, but is important to use along with hx and physical risk..
 - iv. Hx and Physical alone may be diagnostic in 1/3 of cases.



- V. Ruling out Cardiac Causes
- a. Structural heart ds, defined as coronary heart disease, CHF, valvular ds or congenital heart ds, is the only independent risk factor for a cardiac cause.
 - b. ECG and echocardiograms may not be diagnostic but can stratify risk by identifying who has structural ds.
 - c. ECG is an inexpensive and noninvasive test that can diagnose some cases of syncope and guide further testing in others
 - d. Abnormal findings include previous myocardial infarction, BBB, evidence of ventricular caridas, premature ventricular contractions, pacemaker or significant ST abnormalities.
 - e. Pts with cardiac hx or abnormal ECG should have an echocardiogram.
 - i. Similar to ECG it can diagnose a few rare causes of syncope: critical aortic stenosis, myxoma or tamponade.
 - ii. Otherwise systolic function can be sued as a marker for arrhythmia risk. An ejection fraction <40% places pt at a significantly high risk of arrhythmia.
 - f. Continued search for arrhythmia in undiagnosed syncope may include more inefficient, more expensive tests
 - i. 24hr Holter monitoring
 - ii. Implantable loop recoders
 1. can be used for 18 mos. Can store data for up to 20 minutes prior to activation

- iii. Cardiac stress testing is rarely diagnostic
 - 1. should be considered in pts who have syncope during exertion or experiencing chest pain
 - iv. Intracardiac electrophysiologic studies are invasive and very expensive.
 - 1. Should be reserved for patients with known structural heart ds who have a very high risk for arrhythmias due to depressed ventricular function.
- VI. Neurological evaluations
 - a. Neuroimaging and electroencephalography (EEG) have been used frequently in the routine work-up of patients with unexplained syncope. But they have poor yield (2%) and high cost.
 - b. **Neuroimaging and EEG can be limited to patients with symptoms or signs of acute stroke or seizure.**
- VII. Evaluation for Neurally Mediated Syncope (NMSy)
 - a. Many cases will be diagnosed by the initial evaluation alone, usually with a classic hx.
 - i. When etiology is unknown and events become repetitive or dangerous, tilt table testing and carotid massage can be useful as confirmatory tests.
 - b. Tilt table evaluates whether a pt is susceptible to NMSy.
 - i. Multiple protocols, including drug mediated (isoproterenol or nitroglycerin) to increase susceptibility and improve sensitivity.
 - ii. Involves baseline measurement of BP and HR while supine, then quickly bringing the pt to an upright position by tilting to approx 60°.
 - iii. Pt is kept in tilted position for 45 mins while being observed for symptoms and HR & BP are measured.
 - iv. If symptoms occur during testing and correlate with a quick drop in BP or HR it is considered positive. If syncope occurs w/out change in vitals other etiology should be considered.
 - c. Standardized Carotid Sinus Massage (CSM), supine or upright can help differentiate carotid sinus syndrome or carotid sinus hypersensitivity.
 - i. Positive cardioinhibitory result is present if a cardiac pause (asystole) of >3s occurs during or immediately after CSM; a positive vasopressor result is present if the systolic BP drops >50mmHg and is accompanied by symptoms
 - d. Performed as follows
 - i. Confirm no carotid bruits & no significant cerebral vascular ds, if present consider carotid Doppler US to evaluate for plaque.
 - ii. Have pt supine, on ECG monitoring and beat-to-beat BP monitoring. An IV line should be in place, and atropine and transcutaneous pacing available.
 - iii. Turn pts head to left in supine and find the maximum impulse in the right carotid at the level of the thyroid cartilage.
 - iv. Press down and firmly massage longitudinally with 2 fingers for 5-10s. Wait a few minutes and repeat on left carotid.

- v. Repeat in head-up tilt position if symptoms do not occur in the supine position.
- VIII. Diagnosing Orthostatic Hypotension
- a. Have pt lie supine for 3 min, check BP and pulse. Have pt stand for 3min while monitoring pulse and BP.
 - b. Positive result if pt experiences a drop of at least 20mmHg in diastolic BP within 3min after standing from the recumbent position.
 - c. Can also be demonstrated using the tilt table in the head-up position at a minimum of 60° angle.
 - d. Multiple factors can affect the BP readings: time of day, ambient temp, postural deconditioning and meds.
 - i. **some pts may not show diagnostic drop in BP until they have are you listening room and have been standing for at least 10 mins.**
 - e. It is important to repeatedly check for significant orthostatic BP reduction while keeping mind when meds are taken, time of meals and any other factors that may have contributed to the syncopal episode under evaluation.
 - f. Many pts who demonstrate significant drop in BP after standing will also have an increase in pulse rate, but it is not necessarily for the diagnosis of orthostasis.
- IX. Psychiatric evaluation
- a. Clues to psychiatric origin include a hx of anxiety or depression, repetitive syncope of unknown origin, a repeatedly negative cardiac work-up, and multiple episodes w/o of syncope without injury.
 - b. Consulting with a psychiatrist is important both to identify or rule out a psychiatric diagnosis and to review the potential psychotropic effects of medications.
- X. Management
- a. The aggressiveness of evaluation and treatment of syncope will largely depend on the presence of cardiac disease and other comorbidities.
 - b. First time syncope in patients without known or suspected heart disease usually warrants the reduction of risk factors for further Syncope.
 - i. Includes reducing polypharmacy and medication misuse, treating underlying illness and educating regarding avoidance of triggers.
- XI. When to hospitalize
- a. Individuals for whom cardiac Syncope is not suspected can usually be safely managed as an out patient.
 - b. Hospitalization gives rapid access to evaluation tools that can quickly assess the risk of cardiac Syncope, and can change the patients medications in a monitored environment.
 - c. General principles about hospitalization
 - i. Consider hospitalization of older patients with multiple comorbidities when the etiology seems multifactorial and a monitored environment is needed to sort it out.

- ii. Hospitalize pts with known or suspected potentially fatal arrhythmias
 - iii. Hospitalize patients with unknown etiology of syncope when cardiac ds is known or suspected by initial evaluation.
 - iv. Hospitalize when the cause is identified and requires admission (eg myocardial infarction or pulmonary embolism)
- XII. Permanent cardiac pacemakers
 - a. When syncope is a sign of symptomatic bradycardia, not caused by medications, a pacemaker will be the treatment of choice.
 - b. The cardioinhibitory form of neurally mediated syncope may also be an indication, especially if it is repetitive and dangerous, though evidence to support this approach has been mixed.
- XIII. Implantable Defibrillators
 - a. Patients with unexplained syncope, documented structural heart disease and left ventricular ejection fractions >30% are at high risk of sudden death due to arrhythmia.
 - b. Refer to cardiac specialists to evaluate for implantable cardiac defibrillators.
- XIV. Tx of orthostatic hypotension and neurally mediated syncope w/out cardiac abnormalities.
 - a. 1st step is elimination of meds that may contribute to orthostasis, and an admonition to refrain from alcohol
 - b. Encourage lifestyle changes to reduce recurrence, including
 - i. Instruction to stay adequately hydrated by drinking at least 8 glasses of water a day.
 - ii. Prescription compression stockings to reduce venous pooling in the legs (many pts find them uncomfortable, difficult to use and butt-ugly).
 - iii. Education on safer ways of assuming an upright position by rising
 - 1. doing so in 2 stages – sitting and then standing – and using counterpressure maneuvers to avoid a drop in blood pressure.
 - c. If these fail pharmacological tx should be considered.
 - i. Any new meds should be monitored for adverse effects and interactions.

Chapter 35: Hypertension (HTN)

- I. Prevalence and Impact
 - a. Intro
 - i. HTN is one of the most common medical diagnoses in persons over 65
 - 1. Although common, not everyone with an elevated blood pressure reading has hypertension. But many patients with

BP Classification	SBP mmHg	DBP mmHg
Normal	<120	and <80
Prehypertension	120-139	or 80-89
Stage I hypertension	140-159	or 90-99
Stage II hypertension	≥160	or ≥100
Hypertensive crisis	>220	>120

hypertension are unaware of the diagnosis or are under-controlled.

The association among age, HTN and cardiovascular death underlies the importance of understanding HTN diagnosis and management.

Recent changes in HTN management

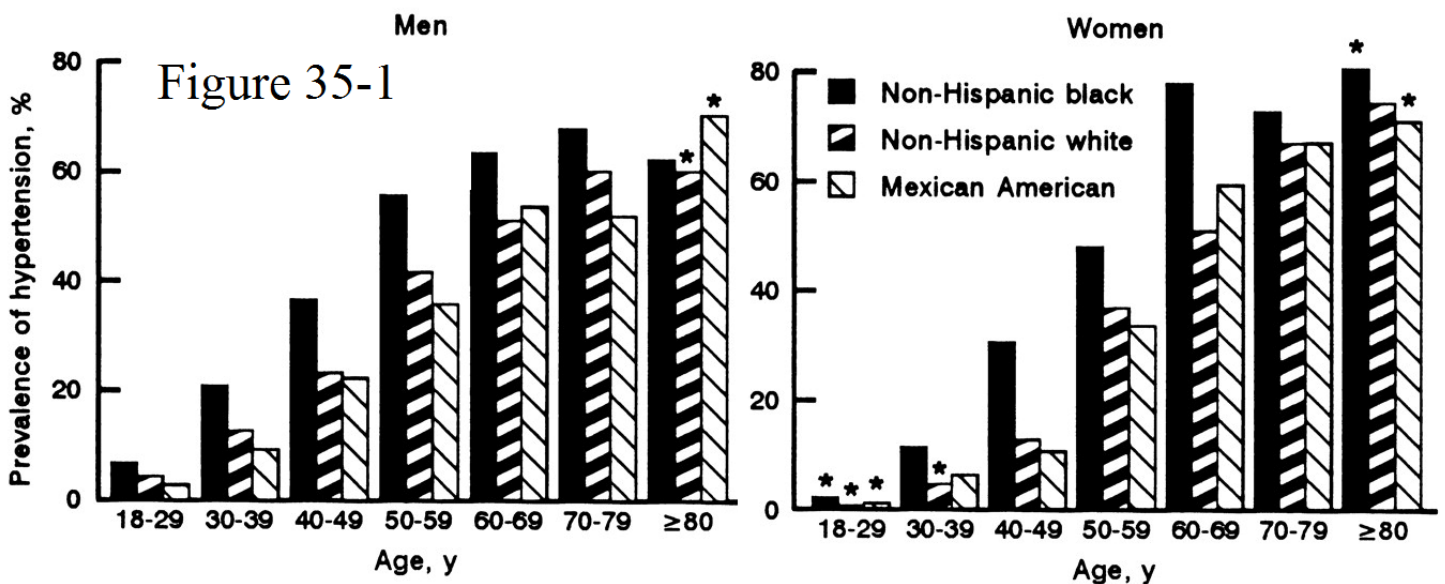
1. Isolated systolic hypertension and widened pulse pressure are important predictors of cardiovascular morbidity and mortality.
2. There are consistent benefits of hypertension treatment even in octogenarians.
3. Many different classes of drugs are appropriate for tx, and a rational approach to choosing drugs involves the consideration of comorbid conditions and existing target organ damage.

There are many elements of appropriate diagnosis and management of hypertension.

1. Distinguishing essential from secondary HTN
2. Recognizing when HTN BP poses an urgen problem
3. Recommending appropriate lifestyle changes
4. Recognizing when to initiate meds, being familiar with the classes of drugs and knowing the circumstances under which one class of drugs may be better suited.
 - a. choosing the appropriate meds and deciding how aggressively to treat the older pt requires considerable clinical judgment.

b. General Definition

- i. Defined as >90mmHg diastolic or >140mmHg systolic read over 3 separate occasions.
- ii. *Isolated Systolic Hypertension (ISH)* is <140mmHg systolic w/ a normal diastolic BP.
- iii.



- c. Prevalence
 - i. Across age groups (3rd national health and nutrition examination survey: figure 35-1)
 - 1. 51% in 60-69
 - 2. 66% in 70-79
 - 3. 72% in >80
 - 4. more recent data suggests 70% of >75 in the US
 - ii. Among elderly w/ hypertension, 60% to 87% have only elevated systolic BP (ISH), making it the most common form in the geriatric population.
 - 1. systolic HTN is more predictive of adverse outcomes compared to diastolic
 - 2. Unfortunately ISH is the least likely form of hypertension to be treated.
 - iii. Individuals with normal BP at age 55 have a lifetime risk of 90% or greater for developing HTN.
 - 1. only 2/3 of Americans are aware that they are hypertensive and only 1/3 of those have their BP under control
- d. Adverse Outcomes
 - i. General risks of HTN
 - 1. those over the age of 65 with HTN have a significantly higher risk for MI, sudden death, stroke, PAD and heart failure.
 - 2. according to the joint national committee on Prevention, Detection, Evaluation and Treatment of Hypertension (JNC) the relationship of BP to cardiovascular ds (CVD) is **“continuous, consistent and independent of other risks”**
 - 3. for persons aged 40-70 starting at a BP of 115/75 there is a doubling in the risk for CVD for each rise in systolic BP of 20 mmHg or diastolic of 10mmHg.
 - 4. **hypertension is linked to multiple medical issues affecting function and quality of life, including vascular dementia, erectile dysfunction, nephropathy and retinopathy.**
 - ii. Isolated systolic Hypertension
 - 1. between 50-60, systolic BP (SBP) assumes a superior role over diastolic BP (DBP) as a predictor of adverse cardiovascular outcomes including stroke and cardiac events
 - iii. Widened Pulse Pressure. (PP)
 - 1. $PP = SBP - DBP$
 - 2. **is a powerful and independent predictor of CVD risk.**
 - 3. a 10mmHg wider pulse pressure at baseline is associated with a 20% increased risk for Cardiovascular mortality.
- e. Rational for Tx

- i. Studies show treatment of ISH can:
 - 1. reduce risk of stroke 30-36%
 - 2. reduce risk of MI 22-55%
 - 3. reduce heart failure 39-54%
 - ii. in octogenarians tx can:
 - 1. Reduce risk of cardiovascular events by 22%
 - 2. reduce risk of stroke 34%
 - 3. reduce risk of heart failure 34%
 - 4. however there is only an insignificant (6%) reduction in all-cause mortality
 - 5. A different study showed similar reductions in stroke, CV events and heart failure with an 11-12% increase in overall mortality
 - iii. For now, given the risks associated with age and HTN, and given the evidence of cardiovascular event reduction with treatment, **limits on attempts at treatment should not be based on age.**
- II. Pathophysiology and Risk Factors
 - a. The elasticity of large blood vessels decreases normally with age and HTN hastens this process.
 - b. The difference in pathogenesis of diastolic versus systolic HTN involves the type of vessel undergoing change
 - i. Diastolic HTN is associated w/ an increase in resistance of small peripheral vessels
 - ii. Systolic HTN is associated with an increase in large vessels resistance. This changes the timing of the arterial pressure wave and ultimately leads to an increased afterload and compensatory left ventricular hypertrophy. The larger heart leads to increased myocardial oxygen consumption and decrease coronary perfusion pressure due to the drop in DBP.
 - c. With age, vessels become less responsive to vasodilating β -adrenergic stimulation while response to vasoconstrictive α -adrenergic stimulation remains the same.
 - d. With age plasma renin levels decline and renin response to sodium depletion, diuretic administration and upright posture declines as well. Therefore, although RAAS is still important to pressure regulation, it may play less of a role in HTN development.
 - e. Finally Tobacco use, excessive alcohol intake and excessive weight all contribute to elevations in BP.
- III. Differential Diagnosis and Assessment
 - a. Definitions and Criteria
 - i. Hypertension
 - 1. average BP of 140/90 on at least three readings taken on 3 separate occasions.
 - ii. Essential hypertension
 - 1. not directly attributable to an underlying medical disorder.
 - iii. Secondary Hypertension

1. attributable to an underlying medical disorder:
2. Factors suggesting secondary HTN
 - a. sudden rise of DBP to >105mmHg in a person known to be normotensive
 - b. DBP >100mmHg or SBP >160mmHg on a tational 3 drug therapy
 - c. Accelerated HTN (rapid worsening) in a person previously known to be hypertensive
 - d. Acute decline in renal function after starting ACE inhibitor
 - e. Unexplained hypocalcemia

3. Causes and screening techniques for secondary HTN

Causes	Tip-Offs	Screening/Evaluation Methods
Sleep Apnea	Excessive daytime sleepiness, snoring, lack of refreshing sleep	Sleep study
Drugs (illicit or prescribed)		Careful hx taking Urine toxicology screen
Primary aldosteronism	Unexplained low potassium	Per endocrinology consult
Renal parenchymal disease	elevated creatinine, abnormal urinalysis (hematuria, cell casts, proteinuria)	24-hour free urine for protein and creatinine clearance, renal US, additional imaging studies per nephrology consult
Renal vascular ds	Elevated creatinine, burit, acutge drop in renal fucntion with use of ACE inhibitors	Gold standard: digital subtraction angiography Other: MRA, CTA, Doppler
Chronic Steroid therapy	Cushingoid stigmata	Careful hx taking
Cushing's syndrome	Cushingoid stigmata	24-hour urine for metanephrines and/or urinary catecholamines
Coarctation of the aorta	discrepancy in SBP and pulse between upper and lower extremities. Notching of the posterior 1/3 to 1/8 ribs on chest radiograph	MRI and/or echocardiogram
thyroid ds	Altered HR, weight changes, palpitations	thyroid function test
Parathyroid ds	Elevated calcium level	Parathyroid hormone levels
plycythemia vera	Elevated RBC count	Bone marrow biopsy

4. This is less likely in elderly pts. Therefore when assessing accelerated or poorly controlled BP on should evaluate for poorly controlled essential HPN
 - i. Poor compliance
 - ii. Inadequate drug prescribing
 - iii. Meds that raside BP (decongestants, NSAIDs)
 - iv. Spuriously elevated BP
 - v. Increased volume expansion
 - vi. Excessive sodium or alcohol intake.

- iv. Hypertensive urgency
 - 1. markedly elevated blood pressure in the absence of acute target organ damage. (table 35-3)

(Table 35-3) Organ System	Acute Findings	Chronic Findings
Heart	Unstable angina pectoris Acute MI Acute left ventricular heart failure Pulmonary Edema	Hx of MI or revascularization Left ventricular hypertrophy CHF
Brain	Encephalopathy Intracranial hemorrhage Subarachnoid hemorrhage	hx of stroke or TIA
Kidneys	Acute Renal insufficiency	chronic renal insufficiency Proteinuria
Vasculature	Dissecting Aortic Aneurysm	PAD
Eyes	Papilledema	Retinopathy

- v. Hypertensive emergency
 - 1. markedly elevated blood pressure in the presence of acute target organ damage, requiring emergency parenteral drug tx to lower BP in order to limit damage. (table 35-3)
 - 2. **BP level is not a criterion for diagnosis of hypertensive urgency or emergency**

b. Assessment of a new case of hypertension

i. Making the dx

- 1. BP > 140/90 x 3 different visits
- 2. there is significant variability in BP btwn 1st and subsequent visits, so you must take more than 1.
- 3. measurements outside the office should be encouraged but **self-measurement > 135/85 is considered abnormal**
- 4. in ambulatory readings awake BP > 135/85 or sleeping >120/75 would be considered hypertensive.
 - a. BP decrease <10-20% during sleep would be concerning for an associated risk .

ii. Blood pressure measurement techniques

- 1. Mercury sphygmomanometers seem to be the most accurate.
- 2. Appropriate cuff size is essential
 - a. too small a cuff can artificially elevate SBP
- 3. BP should be measured after pt is comfortably seated for at least 5 mins w/ arm @ heart level.
- 4. More than 1 test with 1-2 minutes separation
 - a. if values differ by <5mmHg on same arm take subsequent readings until an average is reached
- 5. cuff should be inflated 30mmHg greater than palpable systolic pressure
- 6. Beware Auscultatory Gaps.

7. Cuff should not be deflated faster than 2-3mmHg per heartbeat.
 8. BP should be taken in both arms, arm with higher BP should be used for tx decisions.
- iii. BP measurement: Additional considerations
 1. in the elderly, rigidly calcified or sclerosed blood vessels can lead to overestimation of BP when measurements are made by a cuff. Called: **pseudohypertension**
- c. Initial evaluation
- i. Once diagnosed an assessment of contributing factors, target organ damage and associated cardiovascular risk factors should be done. This allows the doctor to broaden treatment goals and direct treatment (lifestyle change w/ or w/out meds)
 - ii. Past medical hx, fam hx, & ROS
 1. Should target additional cardiovascular risk factors
 - a. diabetes, hyperlipidemia, fam hx of cardiovascular ds.
 - b. past or present evidence of atherosclerotic ds
 - c. renal and cardiovascular ds.
 2. A careful meds hx including over the counter and herbal preparations should be taken for agents associated with elevated BP
 - iii. Social Hx
 1. An assessment for drug use, alcohol and tobacco use should be done.
 2. Diet, exercise routines, support networks and meal preparation abilities will be useful when counseling about nonpharmacological therapies.
 - iv. Physical exam
 1. Optic fundus should be examined for papilledema and hypertensive retinopathy (AV nicking, arterial narrowing, hemorrhages and exudates)
 2. Carotid, abdominal, and femoral arteries should be examined for bruits. The heart should be auscultated for murmurs or heaves, S3/4 and/or displaced point of maximal impulse.
 3. Lungs should be assessed for pulmonary edema.
 4. The abdomen should be palpated for passive congestion of liver and aortic dilation.
 5. Extremities should be examined for edema and evidence of PAD (diminished pulses, atrophic changes, changes in color and temperature)
 6. Nervous system should be assessed for local deficits that could indicate prior stroke.
 - v. Laboratory examinations

1. EKG if one has not been done in the last 3-5 years to assess left ventricular hypertrophy, evidence of prior ischemia and to establish a baseline for future events.
 2. BUN, creatinine, basic electrolytes, fasting lipid panel & glucose should be measured.
 3. UA
 4. If secondary HPT is suspected additional labs may be appropriate.
 5. Echocardiography to assess cardiac structural problems.
- d. Office Evaluation of the chronic Hypertensive
- i. Blood pressure measurements
 1. Same assessment as acute
 2. If pt complains of dizziness, lightheadedness, or postural unsteadiness an evaluation for orthostatic hypotension should be conducted. Especially before prescribing BP lowering meds.
 3. Pts on BP meds should have a BP measurement before the next dose to assess overall control (effectiveness).
 - ii. Persistently elevated BP: Resistant Hypertension
 1. a pt with hypertension stubbornly above goal despite 3 or more meds may need specific exploration to account for the resistance:
 - a. inadequate or ineffective therapy, excessive volume expansion, poor adherence to tx, meds or agents that raise BP (decongestants, NSAIDS, estrogen preparations, anabolic steroids, licorice-containing products, illicit drugs, alcohol) or artificially elevated BP due to “white coat hypertension.”
 - b. Suboptimal therapy and poor compliance tend to be the most common causes.
 - iii. Cardiovascular risk reduction
 1. many hypertensives have additional modifiable cardiac risk factors (DM, or impaired fasting glucose, hyperlipidemia, obesity, tobacco use). The addition of HPT compounds the risk.
 2. Concomitant risk factors such as hyperlipidemia, obesity, tobacco use should be dealt with aggressively.
 3. assessment for chest pain, SOB, dyspnea on exertion, exercise intolerance, calf claudication, and/or other symptoms should be done on a regular basis.
 - iv. Additional care
 1. patients on anti-hypertensives will need periodic electrolyte and renal function screening
 2. All patients should be encouraged to reduce sodium intake, exercise regularly and maintain healthy weight.
 3. Adherence to drug therapy is of the utmost importance!

a. Yeah it actually says that!

IV. Management

a. Management of Hypertensive urgency and emergency

- i. Only effect 1-2% of hypertensives but require immediate recognition and action.
- ii. *These are all drug therapies, I can't imagine he will quiz us on these. In real life if you have this patient walk-in get them to the ER.*

b. Office management of chronic hypertension

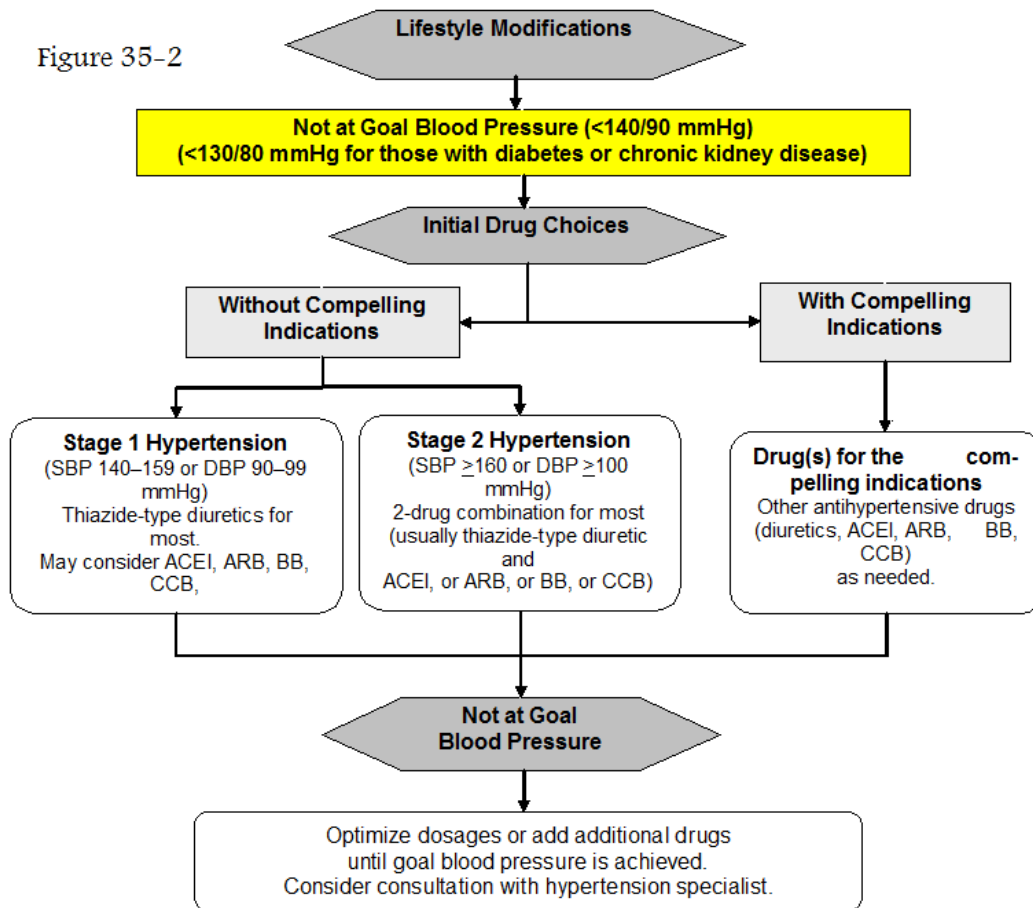
- i. Goal Blood pressure reduction
 1. The same as in younger pts (130/90), however, **reduction should not be at the expense of orthostasis.**
 2. the DM pt the goal should be less than 130/80
 3. pt w/ proteinuria >1g/24hrs goal BP is 125/75
 4. for some SBP <140 may be impossible in this case remember there is significant reduction of strokes, MI and cardiovascular event-associated deaths.
- ii. J-curve hypothesis
 1. Controversial belief that there is a DBP below which coronary perfusion is compromised. The literature seems to be mixed on this point.
 2. In general it is advisable to monitor diastolic pressure reductions cautiously in pts with existing cardiovascular ds.

c. General strategies

- i. **See figure 35-2**
- ii. Nonpharmacological techniques
 1. It may be helpful to enlist the help of a spouse or even a dietitian.
 2. schedule follow-up office visits to assess effectiveness.
 3. It should be noted that there is no evidence that lifestyle change alone reduces morbidity and mortality of hypertension, unlike studies using drug treatment. However, reductions in blood pressure can occur with lifestyle change and can augment drug treatment if latter is necessary.
 4. Effects of lifestyle modification on BP.
 - a. Weight loss: 5-20 mmHg/10kg lost
 - b. DASH diet: 8-14 mmHg
 - c. Sodium reduction: 2-8 mmHg
 - d. Physical Activity: 4-9 mmHg
 - e. Moderation of alcohol intake 2-4mmHg
- iii. Smoking & alcohol cessation
 1. smoking cessation is helpful to reduce risk of cardiovascular events
 2. Excessive alcohol consumption elevates BP of all pts

- a. No more than 2 drinks/day for men, 1 drink/day for women
- iv. Weight
 - 1. hypertension pts who are overweight (BMI <25-27) should be encouraged to get within 10% of their ideal body weight.
 - 2. It may be difficult for elderly pts who may depend on cafeteria food or programs like meals on wheels to make significant diet changes.
 - 3. the key is that dietary advice regarding weight loss must be individualized to the person's circumstances.
- v. Physical Activity
 - 1. Most pts find it easier to lose weight if they also engage in physical activity (optimally, aerobic exercise at least 30 mins per day, most days of the week)
 - 2. Consider pts physical limitations before prescribing an exercise program.

Figure 35-2



vi. Sodium restrictions

1. elderly hypertensives tend to be more salt-sensitive than younger hypertensives, limiting is effective in controlling BP in elderly.
 2. recommend <2.4g of sodium or 6g of NaCl/day
 3. Lower sodium foods may be unpalatable and may result in decline in pts overall nutritional status. In this case salt substitutes can be recommended assuming pts do not have impaired renal function or are taking ACE-inhibitors, angiotensin receptor blockers, or potassium sparing diuretics.
- vii. Dietary Approaches to Stopping Hypertension (DASH)**
1. low in cholesterol and high in fiber, potassium, calcium and magnesium.
 2. associated with reductions in SBP & DBP.
 3. Composition
 - a. 7-8 daily servings of grain
 - b. 4-5 daily servings of vegetables & fruits
 - c. 2-3 daily servings of low/non-fat dairy
 - d. <2 daily servings of meats, fish or poultry
 - e. 4-5 weekly servings of nuts, seeds and legumes.
- d. Medications**
- i. When and why to treat**
 1. The risk v. benefit of drug treatment for HTN needs to be weighed carefully in frail older patients, and it is important to be aware of factors that may increase the risk of adverse drug reactions.
 - a. Diminished baroreceptoractivity and decreased intravascular volume can lead to orthostatic hypotension
 - b. Impaired cerebral autoregulation can produce cerebral ischemia with small drops in systolic pressure..
 - c. There is a risk of dehydration, hyponatremia, hypokalemia, depression, and confusion related to the effects of certain BP meds.
 - d. Decreased renal and hepatic function can cause drug accumulation
 - e. Polypharmacy can raise the risk of drug interactions.
 2. See fig 35-2 regarding when to initiate pharmacological interventions
 - ii. Classes of medications**
 1. Diuretics
 - a. Thiazide are generally the first choice agents
 - b. Effective in lowering SBP & DBP
 - c. Taken 1x/day and generally inexpensive

- d.** Risk of hypocalcemia, hyponatremia, hyperuricemia (with increased risk of gout), dehydration and worsened urinary incontinence.
 - e.** Generally not as effective if creatinine clearance is less than 30 ml/min
 - f.** High doses (>25mg/day) are not recommended, as adverse effects outweigh increases in effectiveness
- 2.** β -blockers
 - a.** Have been shown to reduce cardiovascular mortality, left ventricular hypertrophy, stroke & heart failure.
 - b.** Increase peripheral vascular resistance, therefore, may worsen symptoms of claudication in patients with peripheral vascular disease.
 - c.** Negative inotropic effect and should be used carefully in pts with conduction abnormalities.
 - d.** Exacerbation of reactive airway diseases
 - e.** CNS symptoms, including depression
 - f.** Fatigue or poor exercise tolerance
- 3.** Calcium Channel Blockers
 - a.** Decrease peripheral vascular resistance making them, theoretically ideal in the elderly.
 - b.** No adverse effects on serum lipids
 - c.** Less likely than β -blockers to cause fatigue
 - d.** Generally well tolerated, can be taken 1x/day
 - e.** May cause cardiac conduction defects & may impair myocardial contractility; thus should be avoided if pt has impaired ejection fraction.
 - f.** May cause peripheral edema.
 - g.** Also associated with gingival hyperplasia and constipation
- 4.** ACE inhibitors
 - a.** Although geriatrics generally have low renin activity, these can act effectively as adjunct to diuretics for HTN and are preferential in the settings of diabetes, congestive heart failure, left ventricular hypertrophy, and renal insufficiency.
 - b.** Generally less effective than diuretics
 - c.** Associated with persistent cough
 - d.** Increase serum creatinine
 - e.** Can lead to precipitous drop in renal function in pts with B/L renal artery stenosis.
 - i.** Improve intrarenal blood flow and decrease proteinuria in pts with DM.
 - f.** Been shown to prolong life in pts with CHF and post MI pts

- g. Serum electrolytes (esp. potassium) should be monitored
5. Angiotensin Receptor Blockers
 - a. Similar benefits, indications and precautions as ACE inhibitors.
 - i. More expensive but less persistent cough
 6. Vasodilators
 - a. Directly lower peripheral vascular resistance. Most useful in hypertensive urgency
 - b. Associated w/ reflex tachycardia & postural hypotension
 - c. Typically used if the more standard agents are contraindicated or not tolerated, or if a 4th agent needs to be added to the regimen.
 7. α -blockers
 - a. lower peripheral vascular resistance
 - b. frowned upon as first line therapy due to their great propensity to cause orthostatic hypotension.
 - c. An increase in CHF risk in pts taking these drugs
 - d. Decrease symptoms of prostatic outlet obstruction
 8. Centrally acting alpha 2-agonists
 - a. Depress sympathetic outflow, thus lowering BP.
 - b. Expensive but effective in hypertensive urgency
 - c. Long-term side effects include: dry mouth, fatigue, sedation, depression, orthostatic hypotension and rebound hypertension if doses are missed.
 - d. >1x/day dosing
 - e. Generally not recommended unless hypertension cannot be controlled with any of the above agents in combination.

Comorbid Condition	Diuretic	β -blocker	Calcium Channel Blocker	ACE inhibitor	Angiotensin Receptor blocker	Alpha-1-blocker
Heart failure	+	+		+	+	
Prior MI		+		+	+	
Prior stroke	+			+		
High risk for CAD	+	+	+	+	+	
Type 2 DM	+	+	+	+	+	
Chronic Renal Failure				+	+	
Benign Prostatic Hypertrophy						+

Pre/post tests**Chapter 20****Pretests**

1. A 71yo woman with hypertension, diabetes mellitus and ischemic cardiomyopathy presents after a witnessed episode of syncope at a church gathering. She does not remember the event, but her daughter describes her suddenly appearing pale while sitting and then slumping over and hitting her head on the table. After about 30 seconds she came to on her own. She has a normal physical exam except for an abrasion on her forehead, and her only electrocardiogram abnormalities are the changes from her previous MI. What type of syncope is most likely in your differential diagnosis?
 - a. Neurological
 - b. Neurally mediated
 - c. Cardiac
 - d. Orthostatic
 - e. Psychiatric
2. Which of the following evaluation methods in *not* considered part of the routine workup for a first episode of syncope?
 - a. Electrocardiogram
 - b. Complete History and Physical Exam
 - c. CT scan of the brain
 - d. Review of medication use
3. A 65yo man presents to you after an episode of feeling lightheaded and nauseous, and then passing out briefly. A friend was with him at the time and caught him as he was sliding against a wall to the floor. He has a history only of longstanding hypertension. His comprehensive physical exam and electrocardiogram are normal. Are carotid Dopplers indicated as part of the evaluation to determine the etiology of his syncope?
 - a. Yes
 - b. No

Post-test

1. An 87yo man gets up at night to urinate, something he has done for years. However, this time, after urinating, he feels nauseated and “woozy,” and passes out as he tries to return to his bed. Given this brief history, what is the most likely cause of this syncopal episode?
 - a. Micturition
 - b. Cerebrovascular disease
 - c. Medication-induced postural hypotension
 - d. Multiple factors
 - e. Cardiac arrhythmia
2. A positive response to carotid sinus massage indicates a high likelihood of which one of the following types of syncope?
 - a. Neurally mediated
 - b. Cardiac
 - c. Neurological
 - d. Orthostatic

- e. Psychiatric
3. A 74yo patient comes to see you because she is frustrated with her previous physician, who was unable to diagnose her recurrent syncope. She describes multiple episodes of passing out and near passing out, which have occurred over several years. She often feels dizzy before the syncope. She lives at home, golfs regularly, and is active in her church. Medications include hydrochlorothiazide, a multi-vitamin, and PRN loratadine. She has no history of cardiac disease, and previous evaluations have included several normal electrocardiograms, a normal Holter monitor, and a normal echocardiogram. Your neurological examination is normal. Which of the following would be a logical next step in evaluating this patient?
- Event monitor
 - Tilt table testing
 - CT scan
 - Orthostatic blood pressure evaluation
 - Referral to a neurologist
4. A 65yo female patient who is new to your practice presents after one episode of syncope that was sudden and without prodromal symptoms. She has hypertension, hypothyroidism, and depression, all controlled on her medication per her report. Her physical exam reveals an obese woman with 2+ pretibial edema and a normal cardiovascular and neurological exam. Her electrocardiogram shows left ventricular hypertrophy. Which of the following would be reasonable as the next step in determining the etiology of her syncope?
- Carotid Dopplers
 - Transthoracic echocardiogram
 - CT scan of head
 - Tilt table testing
 - Referral to a psychiatrist
5. A 67yo man with no significant medical problems passes out at a cookout soon after standing up. He had recently finished his barbeque dinner and admits to having consumed three beers. He denies any previous syncopal episodes. His complete physical exam reveals a very healthy-appearing man with no abnormal findings. His electrocardiogram is normal. What should you do?
- Admit him to the hospital for telemetry monitoring
 - Refer him to a cardiologist for intracardiac electrophysiologic studies
 - Schedule an exercise treadmill cardiac stress test.
 - Reassure him and follow up with him in the clinic

Pretest Answers:

- c
- c
- b

Posttest Answers:

- d
- a
- b
- b
- d

Chapter 35

Pretest

1. You diagnose your 86yo patient, Ms. G, with hypertension. She asks you how common hypertension is in her age group. You respond by quoting the following statistic regarding prevalence of hypertension in older individuals:
 - a. 15%
 - b. 33%
 - c. 50%
 - d. 65%
 - e. 80%
2. The most common change in one might see in the blood vessels of an older patient like Ms. G is:
 - a. Increased peripheral vascular resistance
 - b. Decreased peripheral vascular resistance
 - c. Increased response to β -adrenergic stimulation
 - d. Increased response to the renin-angiotensin system
3. Mr. J comes to see you for an annual exam, and you notice that he has a systolic blood pressure of 160mmHg and a diastolic BP of 80mmHg. What will you tell him about this blood pressure?
 - a. "Nothing to worry about." (the diastolic blood pressure is normal.)
 - b. "You have hypertension."
 - c. "The blood pressure reading is concerning, and we need to follow this and get additional readings."
 - d. "You need treatment with medications immediately to bring down your blood pressure."
4. You see Mr J in you office several times and obtain the following blood pressure readings on 3 different occasions: 160/80 mmHg, 150/75 mmHg, and 165/80 mmHg, 150/75 mmHg and 165/80 mmHg. You diagnose him with isolated systolic hypertension. He asks you what risks isolated systolic hypertension pose to his health. You identify all of the following except:
 - a. Stroke
 - b. Myocardial infarction
 - c. Congestive heart failure
 - d. Left ventricular hypertrophy
 - e. Hyperlipidemia
5. Ms. G fails to bring down her blood pressure adequately with lifestyle changes alone. You discuss and decide to initiate drug treatment, while also encouraging her to continue with lifestyle change. Which class of drugs is the best studied and most often-used first-line agent in treating hypertension in older patients?
 - a. Beta-blockers
 - b. Alpha-blockers
 - c. Calcium-channel blockers
 - d. Angiotensin-converting enzyme inhibitors
 - e. Thiazide diuretics

6. What should Ms. G's goal blood pressure be?
 - a. $<160/90$ mmHg
 - b. $<150/90$ mmHg
 - c. $<140/90$ mmHg
 - d. Ten points lower than the starting systolic blood pressure

Posttest

1. Mr. S is a 72yo white male who has a long history of chronic obstructive pulmonary ds and now requires pharmacologic therapy for hypertension. The class of drugs that should be avoided when choosing an agent for him is:
 - a. Central adrenergic inhibitors
 - b. B-blockers
 - c. Calcium-channel blockers
 - d. ACE inhibitors
 - e. Diuretics
2. Ms. M is an 82yo black woman who has had hypertension for many years. Recently she has developed congestive heart failure. The class of drugs that would be best for her antihypertensive therapy now is:
 - a. thiazide diuretics
 - b. α -blockers
 - c. ACE inhibitors
 - d. Calcium-channel blockers
3. Mr. M is a 70yo white man who has had hypertension for years and now has type 2 DM. He is a retired physician and is worried about the new finding of diabetes mellitus. He wants to be treated with a drug that may limit the impact this condition, and his hypertension will have on his renal function. An agent from which of the following classes is most likely to be beneficial?
 - a. α -blockers
 - b. Diuretics
 - c. Calcium-channel blockers
 - d. Vasodilators
 - e. ACE inhibitors

Pretest

1. d
2. a
3. c
4. e
5. e
6. c

Posttest

1. b
2. c
3. e