Hypertension in Older Adults
A case-based discussion

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Dr. Chen has indicated that he has not had financial or other relationships with commercial interests, related to this presentation, within the past 12 months.

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Outline

- The problem (s)
- How to determine an appropriate management strategy
- Agents w/ benefits/drawbacks in OA
- Particular challenges in OA
  - Orthostatic Hypotension
  - Resistant HTN

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Age and HTN

- US: By 2050: 7.4% of people will be >80y
  - This = 2 x the % in 2010
- **HTN prevalence ~ 47% of the US population (>121 M)**
- Incidence HTN at a given age has been stable
- Overall population continues to grow
- 70% of those ≥ 65y have HTN
- Framingham Study observational data suggest the lifetime risk of developing HTN for 55-65y/o is >90%

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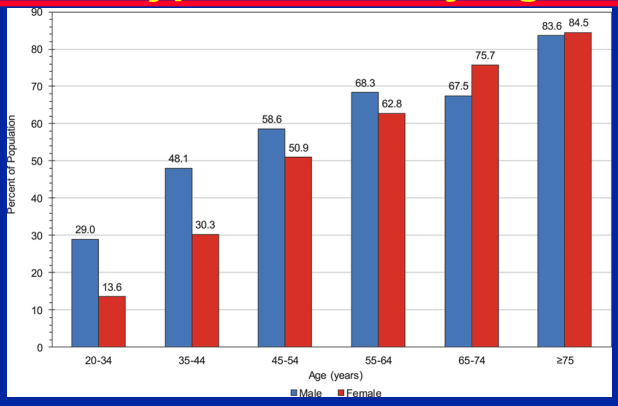
US Census Bureau: https://www.census.gov/newsroom/press-releases/2014/cb14-84.html. Accessed August 10, 2018;
Egan, et al. US trends in prevalence, awareness, treatment and control of hypertension, 1988-2008. JAMA 2010;303:2043;
Vasan RS , et al. Residual lifetime risk for developing hypertension in middle-aged women and men: the Framingham Heart Study. JAMA 2002;287:1003.

Prevalence of Hypertension by Age



Prevalence of hypertension in US adults ≥20 years of age by sex and age (NHANES, 2015–2018). Hypertension is defined in terms of NHANES blood pressure measurements and health interviews. A person was considered to have hypertension if he or she had systolic blood pressure ≥130 mm Hg or diastolic blood pressure ≥80 mm Hg, if he or she said "yes" to taking antihypertensive medication, or if the person was told on 2 occasions that he or she had hypertension. NHANES indicates National Health and Nutrition Examination Survey. Source: Unpublished National Heart, Lung, and Blood Institute tabulation using NHANES, 2015 to 2018.

Salim S. Virani. Circulation. Heart Disease and Stroke Statistics—2021 Update, Volume: 143, Issue: 8, Pages: e254-e743, DOI: (10.1161/CIR.0000000000000950)

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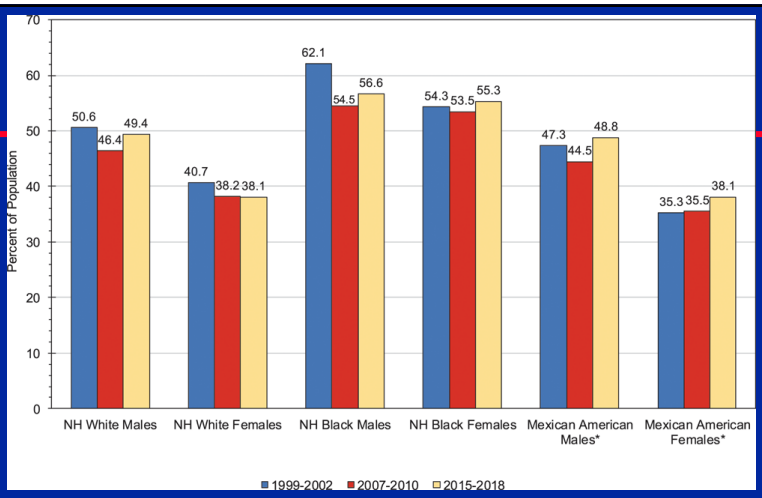

Age-adjusted prevalence trends for hypertension in US adults ≥20 years of age by race/ethnicity, sex, and survey year (NHANES, 1999–2002, 2007–2010, and 2015–2018). Hypertension is defined in terms of NHANES blood pressure measurements and health interviews. A person was considered to have hypertension if he or she had systolic blood pressure ≥130 mm Hg or diastolic blood pressure ≥80 mm Hg or if he or she said "yes" to taking antihypertensive medication. NH indicates non-Hispanic; and NHANES, National Health and Nutrition Examination Survey. *The category of Mexican American people was consistently collected in all NHANES years, but the combined category of Hispanic people was used only starting in 2007. Consequently, for long-term trend data, the category of Mexican American people is used. Source: Unpublished National Heart, Lung, and Blood Institute tabulation using NHANES, 1999 to 2018.

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Incidence Race/ethnicity

- CARDIA Study (3890; 18-30y) w/o HTN
- Incidence (SBP ≥130 mmHg, DBP ≥80 mmHg, or self-reported a-HTN med use) by 55y:
  - 75.7% in Black females
  - 75.5% in Black males
  - 54.5% in White males
  - 40.0% in White females

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Justin Thomas S, et al. Cumulative incidence of hypertension by 55 years of age in Blacks and Whites: the CARDIA study. **J Am Heart Assoc**. 2018; 7:e007988

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HTN and CVD Risk

- **CVD—Stroke, CAD, SCD, HF, PVD, ESRD**
- **The BP relationship to risk of CVD is continuous, consistent, and independent of other risk factors**
  - **Into the 80s age range in large Epidemiologic studies**
- **Each increment of 20/10 mmHg doubles the risk of CVD across the entire BP range starting from 115/75 mmHg**
  - **The lower end of this range varies depending on age and functional status of the population studied**

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BP and CV Risk (details)

- Systolic BP and Pulse Pressure are more indicative of CVD risk in older adults (>55-60y)
  - High SBP with a low DBP carry increased risk
  - SBP less informative for CVD risk in frail/multi-morbid
- In younger adults Diastolic BP is more so
  - More related to peripheral resistance
  - Tends to be less variable

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Vascular aging and hypertension

- Until `50s-60s, both Systolic and Diastolic BP increase
- Arterial stiffness causes the increase in systolic, decrease in diastolic BP (therefore increase in pulse pressure) with age
  - Each is associated with CVD events & all-cause mort
- Wall hypertrophy, calcification, atherosclerosis and changes in extracellular matrix
- Functional changes in endothelial function and smooth muscle cell function

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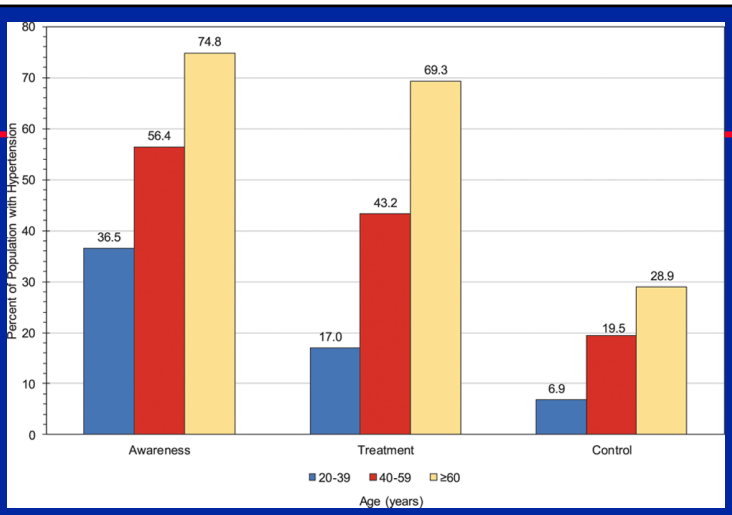

Extent of awareness, treatment, and control of high blood pressure by age, United States (NHANES, 2015–2018). Hypertension is defined in terms of NHANES blood pressure measurements and health interviews. A person was considered to have hypertension if he or she had systolic blood pressure≥130 mm Hg or diastolic blood pressure ≥80 mm Hg or if he or she said "yes" to taking antihypertensive medication. NHANES indicates National Health and Nutrition Examination Survey. Source: Unpublished National Heart, Lung, and Blood Institute tabulation using NHANES 2015 to 2018.

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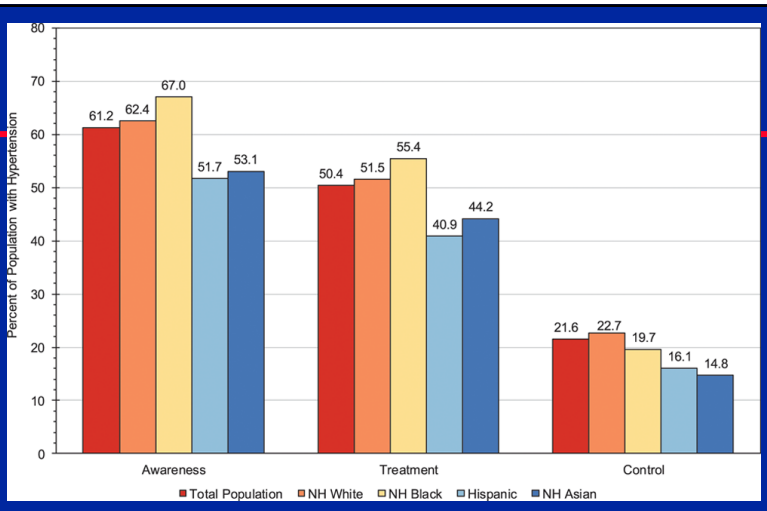

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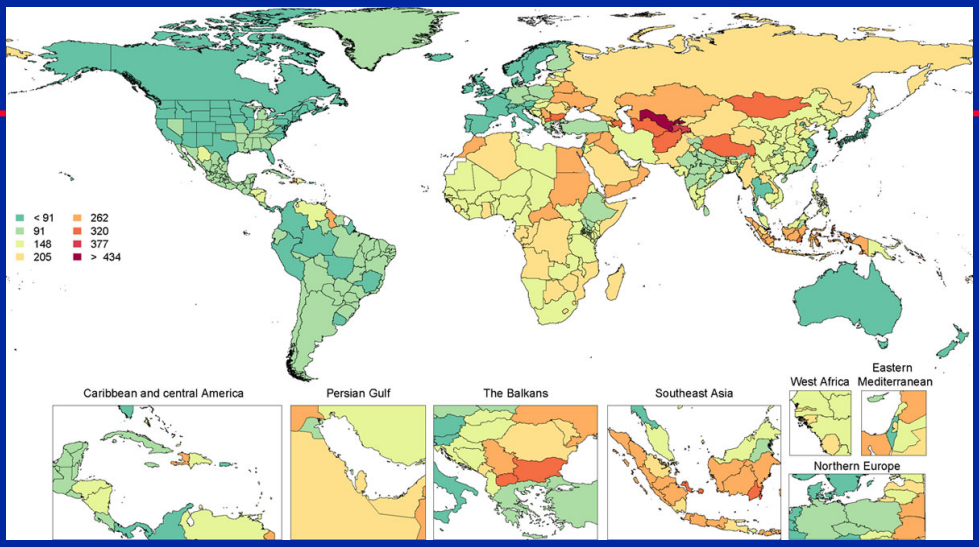

Age-standardized global mortality rates attributable to high systolic blood pressure per 100 000, both sexes, 2019. Source: Data derived from Global Burden of Disease Study 2019, Institute for Health Metrics and Evaluation, University of Washington.

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Classification and Management of BP for Adults

BP classification	SBP* mmHg	DBP* mmHg	Lifestyle modification	Initial drug therapy	
				Without compelling indication	With compelling indications
Normal	<120	and <80	Encourage		
Pre-HTN	**120–139**	or 80–89	Yes	No antihypertensive drug indicated.	Drug(s) for compelling indications. ‡
Stage 1 Hypertension	140–159	or 90–99	Yes	Thiazide-type diuretics for most. May consider ACEI, ARB, BB, CCB, or combination.	Drug(s) for the compelling indications.‡ Other antihypertensive drugs (diuretics, ACEI, ARB, BB, CCB) as needed.
Stage 2 Hypertension	≥160	or ≥100	Yes	Two-drug combination for most† (usually thiazide-type diuretic and ACEI or ARB or BB or CCB).	

Treatment determined by highest BP category.
†Initial combined therapy should be used cautiously in those at risk for orthostatic hypotension.
‡Treat patients with chronic kidney disease or diabetes to BP goal of <130/80 mmHg.

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Benefits of Lowering BP

	Average Percent Reduction
Stroke incidence	35 – 40%
Myocardial infarction	20 – 25%
Heart failure	50%

With Stage 1 HTN and additional CVD risk factors, achieving a sustained 12 mmHg reduction in SBP over 10 years prevents 1 death for every 11 patients treated!

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BP and "Reverese Causality"

- Some studies have shown an inverse relationship between BP (S & D) and CVD/All-cause mortality in the very old
  - Increased mortality with lower BP
  - Reasons: CV and neurologic comorbidities, weight loss, dehydration, polypharmacy
- Remaining HTN-ive may be a marker of better health
- Need for Anti-HTN Rx may decline over time

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INVEST Trial
Primary outcome: 1st occurrence of All cause death, nonfatal MI or nonfatal stroke

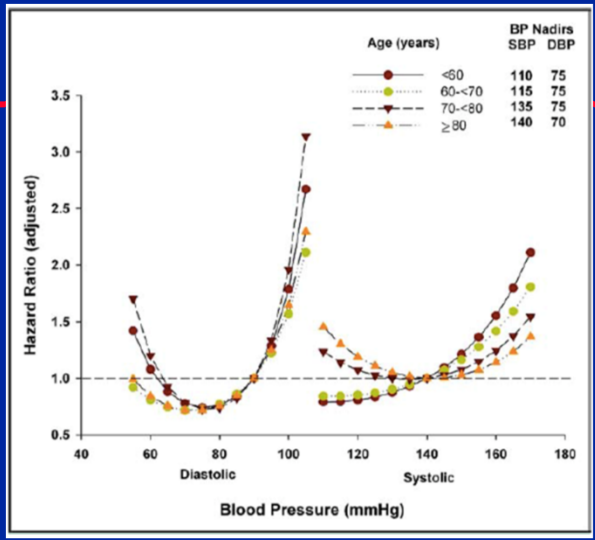

Figure 1: Adjusted hazard ratio as a function of age, systolic and diastolic blood pressure. Reference systolic and diastolic blood pressure for hazard ratio: 140 and 90mmHg, respectively. Blood pressures are the on-treatment average of all post baseline recordings. The quadratic terms for both systolic and diastolic blood pressures were statistically significant in all age groups (all P <0.001, except for diastolic blood pressure in 60-70-year-olds for whom P<0.006). *Figure reproduced from Denardo, et al. 2010 with permission from the publishers.*

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Frail patients

- Observational studies in very old frail patients
  - SBP <130mmHg, associated with higher morbidity & mortality in <u>treated</u> patients, but not in those w/ "naturally" lower BP
  - Because patients treated have had longer h/o HTN?
  - Because treated HTN in this age group worsens their prognosis?
  - Hypothesis: tissue hypoperfusion may result from treatment to these levels

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Differing guidelines over time

- 2013 European: ≥ 80y tx if SBP >160mmHg, target <150mmHg
- N. American guidelines 2017, 2018: Not modifying based on frailty
- 2017 Canada: Target <120mmHg for anyone >75y
- 2017 ACP/AAFP: Target <150/90mmHg, ≥60y
- 2017 ACC/AHA: Target & start tx at: <130/80mmHg, >65y
  - Clinical judgement & pt preference w/ low life expect or multiple comorbidities. Lack of RCT in pts w/ frequent falls, advanced cog impair, SNF residents. Utilize team-based approach
- 2018 ESC/ESH: Target <140/80 mmHg
  - 65-79y: Start treatment if ≥ 140/90 mmHg
  - ≥ 80y + "Fit": Start treatment if ≥ 160/90 mmHg

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Differing Guidelines

- What's "older" differs
- Simple "age" may not be the best way to help guide targets and therapy but loss of function and autonomy more common in the >80y

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To determine appropriate strategy

- Evaluate Frailty & Functional Status
- Frailty
  - Multidimensional (physical, cognitive, psychological) syndrome of loss of reserve
  - Predicts dependence, hospitalization, institutionalization, death
  - Can help determine the appropriateness of interventions

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Benetos A, et al. Hypertension. 2016;67:820.

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Frailty and Cardiovascular Disease

- Risk stratification for therapies using frailty & functional status have been shown useful in:
  - Heart failure
  - Transcatheter AV implantation
  - Cardiac Surgery
  - Atrial fibrillation
  - Diabetes

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HYVET & SPRINT STUDIES

- HYVET-Placebo controlled RCT on mortality and CV outcomes in >80 y/o
  - 2013 Eur Soc: initial SBP >160, decrease to <150mmHg; if treatment gets <140mmHg and tolerated, may continue
- SPRINT-Benefit of a <120mmHg goal in patients >75y

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HYVET & SPRINT STUDIES

- Post-hoc analysis did not find an impact of the results from frailty
- 2017 Canadian guidelines suggest no difference in treatment
  - Very frail subjects were excluded from both studies
  - Subjects were relatively fit, community dwelling, w/o dementia or significant cognitive decline, multiple CV RFs, other comorbidities, OH, metabolic disorders, loss of autonomy
  - SPRINT BP measurement method may have been misleading (more ~130-135mmHg)

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Exclusion Criteria HYVET & SPRINT

Age, y	SBP	DBP	BP Regulation Physiopathology	Main Risks	Better BP Risk Marker	Management
65–80	↑↑	↑	High PR and AS	CV complications, cognitive decline	High SBP	Physical activities, assess TOD and global CVR, medical tt (SBP <140)
65–80	↑	↔↓	High AS	CV complications, cognitive decline	High SBP, PP, low DBP	Physical activities, assess TOD and Global CVR, medical tt (SBP <140)
>80	↑↑	↔↓	High AS	CV complications, falls	High PP, low DBP, OH	CGA, medical tt (SBP <150 or SBP <140 according functional status)
>80	↔	↓↔	High AS and comorbidities	CV complications, falls, loss of autonomy	Normal/low SBP, low DBP, normal/high PP, OH	CGA, deprescribing if SBP<130 or OH, fight polypharmacy

AS indicates arterial stiffness; BP, blood pressure; CGA, Comprehensive Geriatric Assessment; CV, cardiovascular; CVR, cardiovascular risk; DBP, diastolic blood pressure; HYVET, Hypertension in the Very Elderly Trial; OH, orthostatic hypertension; PP, pulse pressure; PR, peripheral resistance; SBP, systolic blood pressure; SPRINT, Systolic Blood Pressure Intervention Trial; TOD, target organ damage; and tt, treatment.

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Integrating Frailty and Function in HTN mgmt

- Define degree past which treatment should be altered
- 2016 Expert review
  - Living in NH or needing daily assistance for basic activities
    - Up to 35% of >80y
    - Negative relationship btw BP and morbidity-mortality (especially when on a-HTN meds)
    - Always excluded from trials establishing benefits of tx

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Integrating Frailty and Function in HTN mgmt

- Comprehensive Geriatric Assessment
  - Likely too complex for non-geriatricians w/o a multidisciplinary team
- Clinical Frailty Scale more practical
  - Preserved function (1-3)
  - Loss of function but preserved autonomy for ADL (4-5)
  - Severe loss of function & autonomy for ADL (6-9)

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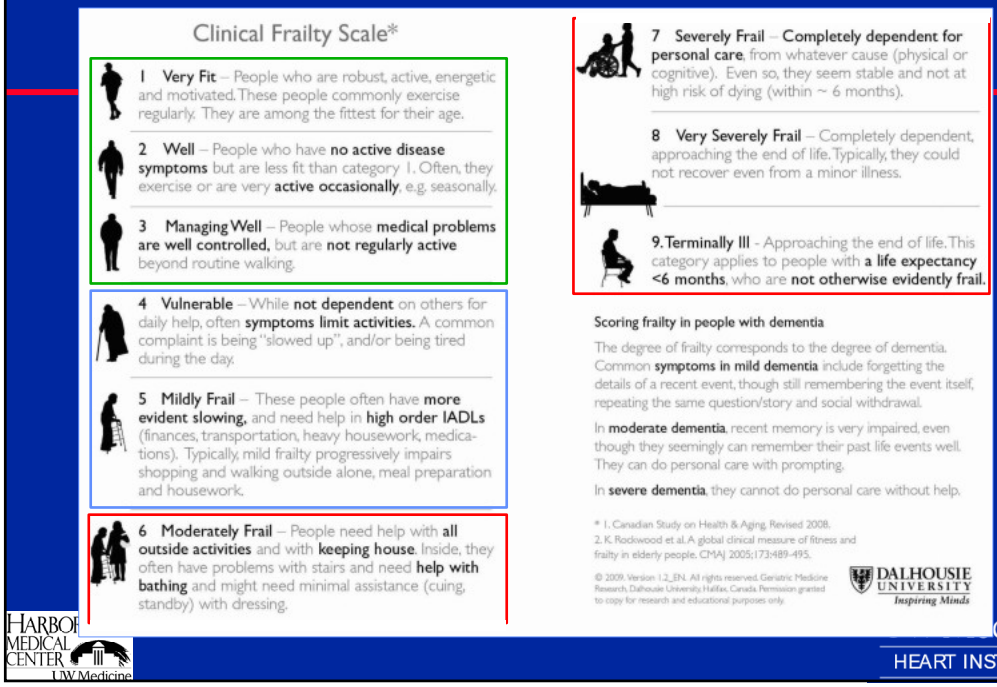

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Case

- 83 y/o M with diet-controlled DM and HTN w/o complications, HTN, and osteoarthritis presents for yearly physical, but hasn't been seen in person in 2 years due to the pandemic
- He lives independently in an apartment, is active and tries to walk ~30 min 3-4x week
- On exam he is mildly overweight, but walks without assistive devices and appears steady. Able to arise from chair w/o using arms to lift himself
- HR 86 BP 162/70 O2 Sat 96% on RA
- Exam is otherwise unremarkable

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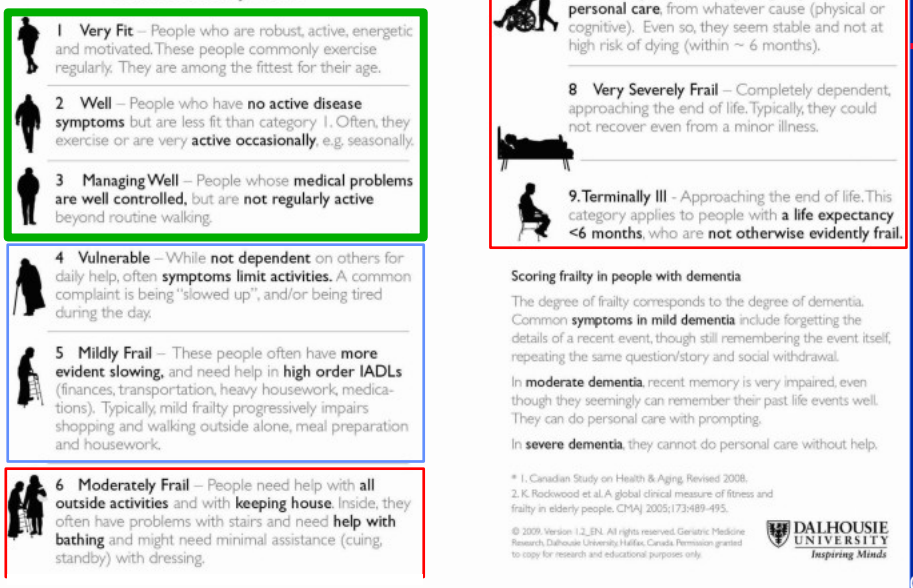
Clinical Frailty Scale*



1 **Very Fit** – People who are robust, active, energetic and motivated. These people commonly exercise regularly. They are among the fittest for their age.

2 **Well** – People who have **no active disease symptoms** but are less fit than category 1. Often, they exercise or are very **active occasionally**, e.g. seasonally.

3 **Managing Well** – People whose **medical problems are well controlled,** but are **not regularly active** beyond routine walking.

4 **Vulnerable** – While **not dependent** on others for daily help, often **symptoms limit activities.** A common complaint is being "slowed up", and/or being tired during the day.

5 **Mildly Frail** – These people often have **more evident slowing,** and need help in **high order IADLs** (finances, transportation, heavy housework, medications). Typically, mild frailty progressively impairs shopping and walking outside alone, meal preparation and housework.

6 **Moderately Frail** – People need help with **all outside activities** and with **keeping house**. Inside, they often have problems with stairs and need **help with bathing** and might need minimal assistance (cuing, standby) with dressing.

7 **Severely Frail** – **Completely dependent for personal care**, from whatever cause (physical or cognitive). Even so, they seem stable and not at high risk of dying (within ~ 6 months).

8 **Very Severely Frail** – Completely dependent, approaching the end of life. Typically, they could not recover even from a minor illness.

9. **Terminally Ill** - Approaching the end of life. This category applies to people with **a life expectancy <6 months**, who are **not otherwise evidently frail.**

Scoring frailty in people with dementia

The degree of frailty corresponds to the degree of dementia. Common **symptoms in mild dementia** include forgetting the details of a recent event, though still remembering the event itself, repeating the same question/story and social withdrawal.

In **moderate dementia**, recent memory is very impaired, even though they seemingly can remember their past life events well. They can do personal care with prompting.

In **severe dementia**, they cannot do personal care without help.

* 1. Canadian Study on Health & Aging, Revised 2008.
2. K. Rockwood et al. A global clinical measure of fitness and frailty in elderly people. CMAJ 2005;173:489-495.

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Activities of Daily Living (ADLs)

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Case

- 88 y/o W with mild to moderate dementia, a h/o afib and mild CAD w/o angina. She has had 2 falls in the last 2 years, which she attributes to tripping on throw rugs. She does use a walker. She lives with her daughter and son-in-law. They help her with preparing food, and bathing.

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Loss of Function/Preserved ADL Profile

- Usually have 1-2 comorbidities and
- Moderate cognitive and functional decline
- Consider tailoring therapy and deprescribing
- 25-40% of group 4 and the vast majority of group 5 were excluded from HYVET
- Further categorization may require CGA
  - Identify comorbidities, geriatric syndromes, degree of functional impairment & loss of autonomy (ADLs)

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Loss of Function/Preserved ADL Profile

- If: Few comorbidities and minor loss of autonomy
  - Treat as those with Preserved Function Profile, <130/80 mmHg
- If: Multiple comorbidities, geriatric syndromes, and dependence on multiple ADLs
  - Treat as Loss of Function & Altered ADL profile

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Case

- 96 y/o M with a h/o metatstatic prostate cancer, no longer being treated
- BP 160/65 HR 89
- Lives in an adult family home
  - Wheelchair bound, needing assistance for transfers, toileting, dressing, although he can eat w/o assistance
  - Mentally active; does the crossword daily

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Loss of function & Altered ADL Profile

- At least one:
  - Multiple comorbidities; Severe dementia, several geriatric syndromes, dependence in ADLs
- Most are at least 85 y/o
- Treatment should be tailored, emphasizing symptom relief and QOL
- Consider life expectancy calculator
- Multidisciplinary approach (PCP, specialists, pharmacists, caregivers)

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Loss of Function & Altered ADL Profile

- Consider keeping <150mmHg as a target
- Reduce doses/stop meds if <130mmHg
  - Or if there is Orthostatic Hypotension
- Monitor for and manage
  - Malnutrition, dehydration, other medications (alpha blockers for BPH)
- Smaller studies/trials with short f/u have evaluated deprescribing w/o harm and less cost in older adults

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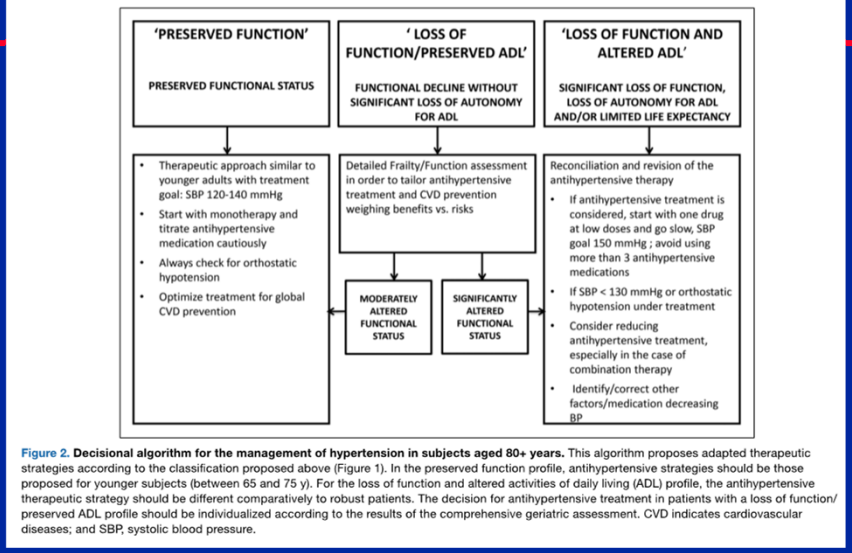

Figure 2. Decisional algorithm for the management of hypertension in subjects aged 80+ years. This algorithm proposes adapted therapeutic strategies according to the classification proposed above (Figure 1). In the preserved function profile, antihypertensive strategies should be those proposed for younger subjects (between 65 and 75 y). For the loss of function and altered activities of daily living (ADL) profile, the antihypertensive therapeutic strategy should be different comparatively to robust patients. The decision for antihypertensive treatment in patients with a loss of function/ preserved ADL profile should be individualized according to the results of the comprehensive geriatric assessment. CVD indicates cardiovascular diseases; and SBP, systolic blood pressure.

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Benetos A., et al. Circ Res. 2019;124:1045.

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Lifestyle Modification

Modification	~ SBP reduction (range)
Weight reduction	5–20 mmHg/10 kg Wt loss
Adopt DASH diet	8–14 mmHg
Sodium reduction	2–8 mmHg
Physical activity	4–9 mmHg
Moderation of EtOH consumption	2–4 mmHg

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Treatments

- Effective NON pharmacologic therapies may not apply to >80y (limited data) or may even be detrimental
  - Weight Reduction
  - Diets
    - Salt reduction: SSaSS Salt Trial
    - Mediterranean or DASH Diets
    - Physical activity
    - Moderate EtOH intake
  - Exercise (tai chi)

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Pharmacologic treatment

- Guidelines similar as to meds
  - Thiazide diuretics
  - Calcium channel blockers (CCB)
  - Angiotensin-converting enzyme (ACE) inhibitors
  - Angiotensin receptor blockers (ARB)
  - Beta blockers (BB)
- In general Diuretics, CCB and ACEi are well-tolerated and are first line
- Combination, if necessary, caution especially with >3 meds, >80y

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Adverse medication effects

- Common SE/Adverse reactions
  - Fatigue, confusion/delirium, OH, falls
- Nifedipine IR, peripheral alpha1-antagonists (doxazosin, prazosin, terazosin) increase risk of OH
- Central alpha2-agonists (clonidine, guanfacine, methyldopa) can cause CNS side effects
- Peripheral alpha1-antagonists + loop diuretics in Women can increase urinary incontinence
- RAAS agents (ACE-I, ARB, aliskiren) and potassium sparing diuretics (amiloride, triamterene) can increase risk of hyperkalemia

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Table 3. Antihypertensive Drugs: Adverse Effects and Precautions in Individuals Aged 80+ Years

Drug Class	Most Common Adverse Effects	Special Precautions/Considerations in Old Individuals
CCB Dihydropyridine CCB Non dihydropyridine CCB	Signs related to sympathetic activation (flushing, headache, tachycardia) are less frequent than in younger subjects. Lower limb edema (frequent since many other factors for LLE). Bradycardia, AV block, worsening heart failure, constipation (verapamil), fatigue, dyspnea.	LLE, which is relatively frequent with these drugs, can be erroneously interpreted as a clinical sign of heart failure. In addition, LLE can contribute to the decrease in social and physical activities for practical reasons (difficulties in walking with shoes). Second-line selection; diltiazem can also cause LLE. With verapamil, LLE is unusual, but constipation may be a major problem in very old individuals, as it can lead to fecal impaction, with nausea, anorexia, delirium, and functional decline. Never combine verapamil with β-blockers.
Diuretics Thiazide Loop diuretic	Hyponatremia, hypokalemia, hyperuricemia and gout attacks, hypotension, dehydration. Similar to Thiazides	For both thiazide and loop diuretics: Diuretic should be titrated according to the patient's volemic status, The latter may be difficult to assess in very old and frail individuals. Creatinine and electrolyte monitoring is warranted after each dose change, Association with SSRI antidepressants increases the risk of severe hyponatremia. Risk of aggravation of urine incontinence. For this reason, diuretics may have an impact on the social life of the patient and can contribute to his/her isolation. Other patients often do not take their treatment if they want to have outdoor activities. Thiazide-like indapamide has been tested in the only RCT specific for subjects >80 y. Small doses (up to 25 mg of HCTZ or equivalent) are safe and well tolerated. Loop diuretics are not indicated for hypertension unless there is severe renal insufficiency (estimated creatinine clearance <30 mL/(min·1.73 m²)). In the presence of both hypertension and heart failure, loop diuretics can be used for both diseases, either alone or in combination with thiazides.
ACE inhibitors	Dry cough, hyperkalemia, rash, angioedema, dizziness, fatigue, acute renal failure	ACE inhibitors have been tested in the only RCT specific for subjects >80 y. Avoid if you suspect dehydration, do not simultaneously increase diuretics to avoid a worsening in renal function. Regular control of creatinine and potassium levels.
Angiotensin II receptor antagonists	Hyperkalemia, rash, dizziness, fatigue, acute renal failure	The same as for ACE inhibitors: Do not combine ARB with ACE inhibitor or renin inhibitor. Be cautious with aldosterone antagonist because of increased risk of hyperkalemia.
β- adrenoreceptor antagonists (β-blockers)	Bradycardia, cardiac decompensation, peripheral vasoconstriction, bronchospasm, fatigue, depression, dizziness, confusion, hypoglycemia	Fatigue, which is multifactorial in older subjects, can be accentuated. Nightmares, sleep disturbances, depression, and confusion may be present especially for the β-blockers crossing the blood brain barrier. Cardiac conduction problems can also be aggravated. Caution when used in combination with acetylcholinesterase inhibitors (for Alzheimer disease): risk of major bradycardia.
Aldosterone antagonists	Hyperkalemia, hyponatremia, and gastrointestinal disturbances, including cramps and diarrhea, gynecomastia	Aldosterone antagonist should not be given in instances of severe renal insufficiency, estimated creatinine clearance <30 mL/(min·1.73 m²) or hyperkalemia. Creatinine and electrolyte monitoring is warranted after each dose change.
α-adrenoreceptor antagonists (α-blockers)	Dizziness, fatigue, nausea, urinary incontinence, orthostatic hypotension, syncope	Usually not indicated. Risk of hypotension (orthostatic, postprandial) and syncope.
Central α-adrenoreceptor agonists	Drowsiness, dry mouth, dizziness, constipation, depression, anxiety, fatigue, urinary retention or incontinence, orthostatic hypotension, confusion, and delirium	High risk of delirium and confusion. Depression, which is atypical and frequent in older subjects (and tricky to diagnose vs cognitive disorders), can be aggravated.

ACE indicates angiotensin-converting enzyme; ARB, angiotensin receptor blockers; AV, atrioventricular; CCB, calcium channel blockers; LLE, lower limb edema; HCTZ, hydrochlorothiazide; RCT, randomized controlled trial; and SSRI, selective serotonin reuptake inhibitors.

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Case

- 82 y/o M with h/o HTN, BPH comes to clinic c/o intermittent LH/Unsteadiness that began last week (he was visiting family in AZ and they were outside a lot).
- HCTZ 25mg po qd, Prazosin 2mg po tid
- BP 120/70 HR 68 sitting; 100/60 HR 81 standing

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Orthostatic Hypotension

- Orthostatic hypotension
  - Fall in SBP of ⩾20 mm Hg or DBP of ⩾10 mm Hg, within 3 min of standing (after supine for 5 min)
    - Fall in SBP of ⩾30 mm Hg for patients with an elevated baseline BP including those with supine hypertension (SH)
  - Categorized as Non-Neurogenic or Neurogenic
  - Non-Neurogenic OH is caused by reduced cardiac output and/or impaired vasoconstriction without a primary autonomic disorder and can include medications or volume depletion (most common)

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Orthostatic Hypotension

- Impaired baroreceptor function due to arterial stiffness cause inappropriate BP & HR responses to postural & other changes
  - Orthostatic hypotension
  - Increased BP variability with: exercise, post prandial increase/decrease, between check variability

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Orthostatic Hypotension--Etiologies

- Neurogenic OH (nOH)
  - Inadequate vasomotor sympathetic release of norepinephrine due to autonomic dysfunction
  - Reduction in sympathetic innervation also causes the heart rate (HR) to increase less than expected
  - nOH is not solely a disease of low BP but also of high BP (ie, supine hypertension) such that patients often display both OH and SH at differing times
  - From impaired central neural pathways that regulate sympathetic control, or
  - From deficient activation of vascular adrenoceptors due to degenerative postganglionic sympathetic neurons
- nOH is a debilitating disorder that carries significant morbidity and is an independent risk factor for mortality

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Orthostatic Hypotension

- Associated with
  - Syncope, falls (hospitalizations, functional decline)
  - CVD, all-cause mortality
- Better HTN control can actually reduce/improve OH

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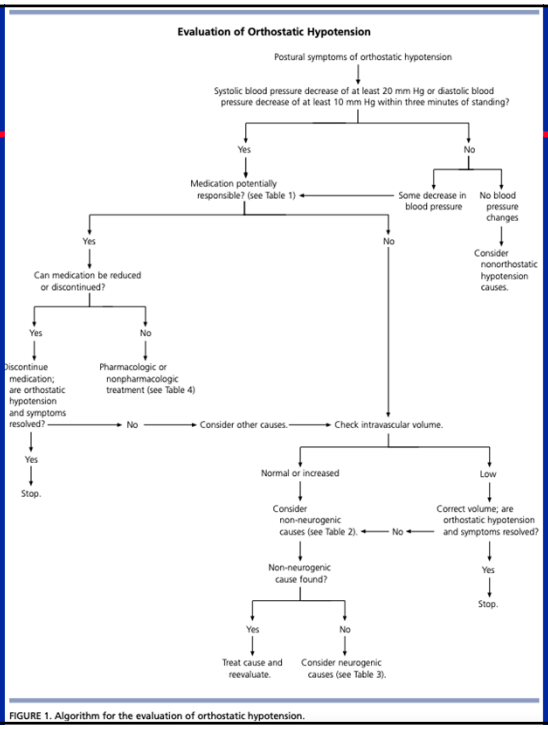

FIGURE 1. Algorithm for the evaluation of orthostatic hypotension.

TABLE 2
Clinical Clues to Non-Neurogenic Etiologies of Orthostatic Hypotension

Findings on history and physical examination	*Possible etiology*
Chest pain, palpations, shortness of breath, rales, edema, arrhythmia, murmur	Congestive heart failure, myocardial infarction, arrhythmia, pericarditis, or myocarditis
Swollen extremities, edema	Congestive heart failure, venous obstruction, prolonged sitting or standing (resulting in venous pooling)
Symptoms on awakening or after a meal	Venous pooling or postprandial hypotension
Vomiting, diarrhea, bleeding, burns, diuretic use, clinical signs of dehydration	Intravascular volume depletion
Various symptoms of endocrine diseases	Adrenal insufficiency, diabetes insipidus
Fever	Sepsis or other acute infectious process

TABLE 3
Clinical Clues to Neurogenic Etiologies of Orthostatic Hypotension

Findings on history and physical examination	*Possible etiology*
Autonomic failure with no other neurologic symptoms	Pure autonomic failure
Parkinsonian features, urinary incontinence or retention, cerebellar dysfunction, autonomic symptoms	Multiple system atrophy
Dysautonomia of acute onset or occurring over a few weeks (can occur with supine hypertension)	Guillain-Barré syndrome
Chronic alcohol abuse	Alcoholic polyneuropathy
Risk of sexually transmitted diseases	AIDS, tabes dorsalis
Various acute, subacute, or relapsing symptoms	Multiple sclerosis

AIDS = acquired immunodeficiency virus.
Information from references 1, 11, 15, and 16.

TABLE 1
Etiologies and Drugs That Can Cause Orthostatic Hypotension

Non-neurogenic etiologies	Neurogenic etiologies	Drugs
Cardiac pump failure	Spinal cord problems	Alpha and beta blockers
Aortic stenosis	Syringomyelia	Antihypertensives
Bradyarrhythmia	Tabes dorsalis	Bromocriptine (Parlodel)
Myocardial infarction	Transverse myelitis	Diuretics
Myocarditis	Tumors	Insulin
Pericarditis	Peripheral nervous system problems	MAO inhibitors
Tachyarrhythmia	HIV/AIDS	Marijuana
Reduced intravascular volume	Alcoholic polyneuropathy	Minor tranquilizers
Adrenal insufficiency	Amyloidosis	Narcotics/sedatives
Burns	Diabetes mellitus	Nitrates
Dehydration	Dopamine beta-hydroxylase deficiency	Phenothiazines
Diabetes insipidus	Guillain-Barré syndrome	Sildenafil (Viagra)
Diarrhea	Paraneoplastic syndrome	Sympatholytics
Hemorrhage	Renal failure	Sympathomimetics (with prolonged use)
Salt-losing nephropathy	Vitamin B_{12} or folate deficiency	Tricyclic antidepressants
Straining with heavy lifting, urination, or defecation	Other neurogenic etiologies	Vasodilators
Vomiting	Brain-stem lesions	Vincristine (Oncovin)
Venous pooling	Brain tumors	
Alcohol consumption	Carotid sinus hypersensitivity	
Fever	Cerebral vascular accidents	
Heat (e.g., hot environment, hot shower or bath)	Dysautonomias	
Postprandial dilation of splanchnic vessel beds	Multiple sclerosis	
Prolonged recumbency or standing	Multiple system atrophy	
Sepsis	Neurocardiogenic syncope	
Vigorous exercise with dilation of skeletal vessel beds	Parkinson's disease	
	Pure autonomic failure	
	Syringobulbia	

HIV = human immunodeficiency virus; AIDS = acquired immunodeficiency syndrome; MAO = monoamine oxidase.

Adapted with permission from Engstrom JW, Aminoff MJ. Evaluation and treatment of orthostatic hypotension. Am Fam Physician 1997;56:1379 with information from references 11 through 13.

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TABLE 4
Selected Nonpharmacologic Treatments for Orthostatic Hypotension

Implement	Avoid
Dorsiflex feet several times before standing	Standing motionless
Make slow, careful changes in position	Rising quickly after prolonged lying or sitting
Eat small, frequent meals	Large meals
Increase salt and fluid intake	Alcohol consumption
Elevate head of bed 5 to 20 degrees	Vigorous exercise
Schedule activities in the afternoon	Heat, hot baths, and hot environment
Wear compression stockings	Dehydration
	Working with arms above shoulders
	Straining with urination or defecation
	Coughing spells
	Rapid ascent to high altitude
	Hyperventilation
	Fever

Information from references 8 and 12 through 15.

Pharmacologic Therapies

- Nonsteroidal anti-inflammatory drugs can be used to increase intravascular volume.
- Mineralocorticoid fludrocortisone (Florinef) may be used in some patients to expand intravascular volume.
  - Used judiciously, risk of volume overload (edema), hypokalemia or hypomagnesemia.
  - Headache, supine hypertension.
- Midodrine (ProAmitine), a vasoconstrictor, is effective in some cases of orthostatic hypotension. RCT
  - Common Ses: pupillary dilation, piloerection, paresthesias, and pruritus.
  - Supine hypertension.
- Erythropoietin has been used to treat autonomic failure associated with decreased red cell mass or anemia.
  - Goal is to bring the hematocrit level within the normal range.
  - SE: Increased appetite, increased sense of well-being.

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Case

- 73 y/o M with Parkinson's Disease. As expected he is unsteady on his feet. His family is very attentive and has noticed that his BP is quite high at night (they first checked when he once had a headache)
- BPs in clinic: 127/64, 73-sitting; 100/53, 80-standing
- Supine BPs last week ranged from 173/80 to 208/93

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Orthostatic Hypotension with Supine Hypertension

- About ½ of patients with Neurogenic OH have supine HTN
- Underlying conditions include
  - Pure autonomic failure
  - Multiple system atrophy
  - Parkinson's Disease
  - Peripheral neuropathies
    - Diabetic neuropathy
    - Autoimmune neuropathies

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Jordan J, et al. J Hypertens. 2019. 37:1541.

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Orthostatic Hypotension with Supine Hypertension

- Consensus definition: Brachial SBP ≥ 140 mmHg and/or DBP ≥ 90 mmHg while supine
- BP may be normal when seated
- Majority have no night-time dip in BP
  - 1/3 of pts with SH have BP decrease after a normal dip

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Orthostatic Hypotension with Supine Hypertension

- Treatment of the HTN has to balance R & B
- Treat to lessen CV risk (LVH, Renal Fx, Brain white matter dz, cognitive impairment (PDz)
- Fear treatment will worsen OH, but evidence suggests treatment may improve OH
  - Supine HTN-->nocturnal pressure natriuresis
    - Nocturia disrupts sleep; 1-2kg/night

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Orthostatic Hypotension with Supine Hypertension

TABLE 1. Management of supine hypertension[a]

Avoid offending agents
Avoid lying down during the day; rest in the seated position
Beware 'Hidden' pressor agents
Ibuprofen
Indomethacin
Atomoxetine
Limit water ingestion near bedtime
Avoid fludrocortisone in favor of short-acting pressor agents when possible
Nonpharmacological treatments
Tilt the whole bed head-up by approximately 10°, in patients not tolerating this measure tilt only the head of the bed up 30°
Carbohydrate-rich snack at bedtime
If alcohol consumed, small amount at bedtime
Pharmacological treatments
Consider individualized antihypertensive treatment taken at bedtime
Monitor night-time blood pressure with 24 h ambulatory blood pressure monitors
Before starting treatment to see if patient 'dips' to a normal pressure later in the night
To monitor treatment efficacy

[a]These recommendations are based on expert opinion and results from small-scale studies. Larger-scale trials with hard clinical endpoints do not exist.

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Orthostatic Hypotension with Supine Hypertension

- Pharmacologic issues
  - If patient is a "dipper" may not need tx (ABPM)
  - Responses differ from pts with "essential HTN"
    - Autonomic Failure – sensitive to vasodilators; b-blockers may not work unless + vasodilatory. Clonidine may worsen HTN
    - Multisystem atrophy & central autonomic failure—more peripheral sympathetic nerves are preserved (driving the HTN)
      - Sympatholytics helpful (e.g. Clonidine)
  - Take in the evening

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Orthostatic Hypotension with Supine Hypertension

- Some trial & error, monitoring response w/ ABPM or taking supine BP at 4am. Some home BP cuffs can be programmed
- Pts with SH have intact Nitric Oxide vasodilating systems
  - Sildenafil—PDEi, has been shown to lower BP
  - Nitroglycerine—NO donor (night-time patch)
  - Nebivolol (vasodilating b-blocker)

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Orthostatic Hypotension with Supine Hypertension

- Renin-angiotensin-Aldosterone Agents
  - Losartan (ARB) effective vs. Captopril (ACEi)—not
    - Also decreased natriuresis (as does Clonidine)
  - Eplerenone (Aldosterone receptor blocker)
- Sympatholytic Agents
  - Caution w/ Clonidine
    - Effective in those with multi-system atrophy (residual sympathetic tone)
    - Can cause HTN in those w/ pure autonomic failure (alpha2-adrenoreceptor)

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Orthostatic Hypotension with Supine Hypertension

- Vasodilators, with caution
  - Short acting nifedipine (CCB)
  - Hydralazine
- Caution w/ all meds d/t possible carryover effects

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Case

Resistant HTN
Persists despite 3 adequate a-HTN meds (including a diuretic)

- 79 y/o W with HTN, DM presents frustrated by her HTN control
- Meds: Lisinopril 40mg po qd, HCTZ 25 mg po qd, Amlodipine 10mg po qd
- Her BP is 160-170/65-80mmHg; HR 95-105bpm on these
- What should be evaluated, by history (labs)

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George Bakris' approach to OA with Resistant HTN

- Ensure adherence to low salt diet (<2,300mg/d), including possibly of 24h urine sodium & total creatinine
  - RAAS agents less effective in setting of high Na+ intake
- Evaluate sleep quantity and quality (including OSA)
  - High resting HR (>86, often ~100bpm)
  - Consider sleep meds, avoiding benzodiazepines
  - Average 1mmHg drop per hour CPAP mask is worn
  - Sleep hygiene education and low Na+ diet
- Evaluate caffeine intake (>2 cups of coffee/day)
- Evaluate other meds: NSAIDS, decongestants

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https://www.practiceupdate.com/content/my-approach-to-the-elderly-patient-with-resistant-hypertension/53363, accessed 1/5/2022.

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George Bakris' approach to OA with resistant HTN

- Stage 3b or greater CKD (eGFR <45mL/min/1.73m^2), emphasize Na+ and diuretics for volume (and BP)
- A h/o persistent/difficult to correct hypokalemia
  - Which may be due to primary hyperaldosteronism
- Assess adherence (memory, cost, intolerances, complexity)
- Avoid certain medications: Clonidine, Hydralazine

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https://www.practiceupdate.com/content/my-approach-to-the-elderly-patient-with-resistant-hypertension/53363, accessed 1/5/2022.

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Treatment

George Bakris' approach to OA with resistant HTN

- CCB & Diuretics are cornerstone
- RAAS agents only in conjunction with those agents
- Beta blockers may be useful, particularly in patients with poor sleep (high adrenergic state)
- Dose at least 50% of aHTN meds at dinnertime if possible
  - This population are "no dippers" (nocturnal < 10% reduction) when evaluated w/ 24h ambulatory BP monitoring
  - Patients who are "no (nighttime) dippers" have no break from HTN; higher incidence of LVH on Echo and higher CVA risk
- Long acting qWeek patch Clonidine if this med is necessary
- Consider Guanfacine (long-acting clonidine-like med; also sedating) which may help w/ sleep initiation

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https://www.practiceupdate.com/content/my-approach-to-the-elderly-patient-with-resistant-hypertension/53363, accessed 1/5/2022.

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Case

- 74 y/o M with h/o CAD s/p MI with stenting, PVOD s/p Fem-Pop bypass as well as HTN c/o worsening BP in last few months
- BP used to be well-controlled on Amlodipine 5mg po qd and Metoprolol XL 100mg po qd
- In the last few months his BP readings range from 130-200/60-90 mmHg with an increase over that time

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Factors suggesting 2ndary HTN

- Acute rise in BP in a patient with previously stable readings
- Age of onset before puberty
- Age younger than 30 years in nonobese, nonblack patients with no family history of hypertension
- Malignant or accelerated hypertension (with signs of end-organ damage)
- Severe (SBP >180 mmHg and/or DBP >120 mmHg) or resistant hypertension

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Table 3. Signs and Symptoms That Suggest Specific Causes of Secondary Hypertension

Signs/symptoms	*Possible secondary hypertension cause*	*Diagnostic test options*
Increase in serum creatinine concentration of at least 50% (≥ 0.5 to 1 mg per dL [44 to 88 µmol per L]) after starting angiotensin-converting enzyme inhibitor or angiotensin receptor blocker Moderate to severe hypertension and unilateral small kidney/recurrent flash pulmonary edema Renal bruit	Renal artery stenosis	CT angiography Doppler ultrasonography of renal arteries Magnetic resonance angiography with gadolinium contrast media
Elevated serum creatinine Proteinuria	Renal diseases	Estimated glomerular filtration rate Renal ultrasonography
Hypokalemia	Primary hyperaldosteronism	Renin and aldosterone levels to calculate aldosterone-to-renin ratio
Apneic episodes during sleep Daytime sleepiness Snoring	Obstructive sleep apnea	Polysomnography (sleep study) Sleep Apnea Clinical Score with nighttime pulse oximetry
Flushing Headaches Labile blood pressures Orthostatic hypotension Palpitations Sweating Syncope	Pheochromocytoma	24-hour urinary fractionated metanephrines and normetanephrines Plasma free metanephrines
Arm to leg systolic blood pressure difference > 20 mm Hg Delayed or absent femoral pulses Murmur	Coarctation of the aorta	Magnetic resonance/CT angiography (adults) Transthoracic echocardiography (children)
Buffalo hump Central obesity Moon facies Striae	Cushing syndrome	24-hour urinary free cortisol Late-night salivary cortisol Low-dose dexamethasone suppression
Bradycardia/tachycardia Cold/heat intolerance Constipation/diarrhea Irregular, heavy, or absent menstrual cycle	Thyroid disorders	Thyroid-stimulating hormone

CT = computed tomography.

Adapted with permission from Viera AJ, Neutze DM. Diagnosis of secondary hypertension: an age-based approach. Am Fam Physician. 2010;82(12):1472, with additional information from references 3, 9, 13, and 23 through 28.

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Secondary HTN, sometimes the cause of resistant HTN

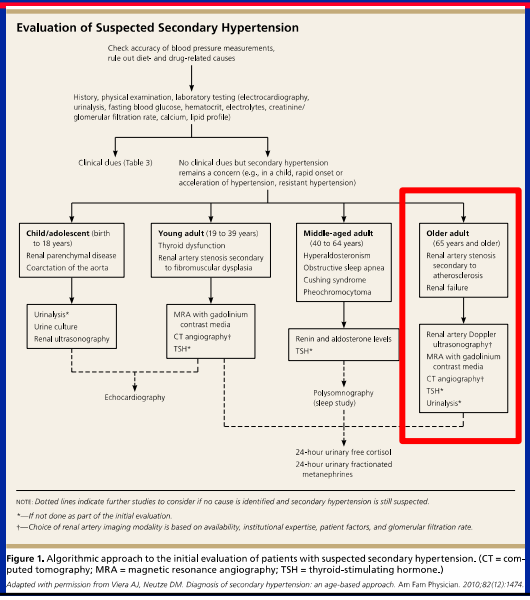

Figure 1. Algorithmic approach to the initial evaluation of patients with suspected secondary hypertension. (CT = computed tomography; MRA = magnetic resonance angiography; TSH = thyroid-stimulating hormone.)

Adapted with permission from Viera AJ, Neutze DM. Diagnosis of secondary hypertension: an age-based approach. Am Fam Physician. 2010;82(12):1474.

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HTN and Cognitive Function

- Epidemiologic studies tie HTN with Cognitive decline long-term
  - Epi studies show link between mid-life HTN and dementia
    - Framingham study (1993) showed HTN 20 years prior was associated with cognitive performance in untreated HTN
    - Honolulu-Asia Aging Study (30y); Skoog study (10-15y); Epidemiology of Vascular Aging Study (4y)
    - Other studies fail to find a link
      - HTN duration; testing methods; population differences
  - Demonstrating benefit in treatment trials is difficult
    - Likely due to relatively short study duration & older age at start of studies

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Elias MF, et al. Am J Epidemiol. 1993;138:353

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HTN and Cognitive Function

- Likely that midlife HTN is more important RF for later life cog impairment than late life BPs
  - Also may not simply be BP, but vascular changes due to longer term HTN
  - Uncertain target and duration needed to provide benefit
  - Lowering BP in frail older adults can even be worse for cognitive function

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Elias MF, et al. Am J Epidemiol. 1993;138:353

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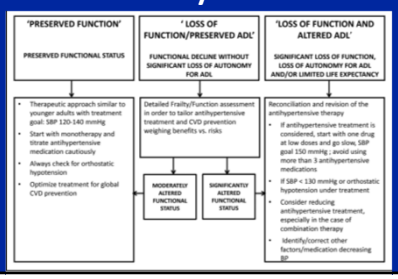
New Directions

- Controlled trials in those with significant frailty
  - In addition to registries, observational studies, longitudinal cohorts
- Evaluation of the HTN management algorithm which integrates Frailty into decision-making



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The End

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