APPROACH TO AN ELDERLY PATIENT WITH HYPERTENSION

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Last reviewed: October 2026

I INTRODUCTION AND THE FRAILTY PARADIGM

- **Definitions:** Hypertension in older adults (chronologically defined as **65 years or older**, and the **"very old" as 80 years or older**) is the leading modifiable driver of stroke, heart failure, atrial fibrillation, chronic kidney disease, and vascular dementia.
- **Biological Age vs. Chronological Age:** Contemporary guidelines (2024 ESC and 2023 ESH Guidelines) shift decision-making from chronological age alone to **biological age, functional autonomy, and Frailty** (quantified using tools such as the **Clinical Frailty Scale [CFS 1 to 9]**), balancing major cardiovascular event reduction against the iatrogenic hazards of orthostatic hypotension, injurious falls, syncope, and acute kidney injury.

II CORE PATHOPHYSIOLOGY IN THE AGING VASCULATURE

- **Isolated Systolic Hypertension (ISH) and Loss of the Windkessel Effect:**
  - **Structural Mechanism:** Age-related fragmentation of medial elastin lamellae, replacement by stiff cross-linked collagen, and medial calcification markedly increase **large-artery (aortic) stiffness** and **Pulse Wave Velocity (PWV)**, abolishing the physiological **aortic Windkessel cushioning effect**.
  - **Hemodynamic Consequence:** The rigid aorta cannot distend during systole (**augmenting peak Systolic Blood Pressure [SBP]** via early return of reflected pulse waves in late systole rather than diastole) and fails to recoil during diastole (**lowering Diastolic Blood Pressure [DBP]**), resulting in a **markedly widened Pulse Pressure (SBP minus DBP >=60 mmHg)** and impaired diastolic coronary perfusion.
  - **The "Low-Renin, Salt-Sensitive" Volume Phenotype:**
  - **Mechanism:** Progressive nephrosclerosis and age-related decline in functioning nephrons and juxtaglomerular apparatus activity produce **low plasma renin activity** and impaired renal sodium excretion.
  - **Clinical Implication:** Older patients exhibit pronounced **salt sensitivity** and respond most effectively to **Calcium Channel Blockers (CCBs)** and **Thiazide/Thiazide-like diuretics**, as well as dietary sodium restriction.
  - **Arterial Baroreceptor Reflex Blunting and Autonomic Dysregulation:**
  - **Mechanism:** Carotid sinus and aortic arch baroreceptor stiffening blunts reflex cardioacceleration and sympathetic vasoconstriction upon standing, predisposing older patients to **Orthostatic Hypotension**, **Postprandial Hypotension**, and marked **24-hour blood pressure variability**.

III CLINICAL EVALUATION AND BEDSIDE PITFALLS

- **Mandatory Orthostatic Blood Pressure Screening (Class I):** Blood pressure must be recorded in the seated/supine position and at **1 minute and 3 minutes after standing** at baseline and after every dose titration. **Orthostatic Hypotension** is defined as a **drop in SBP of 20 mmHg or greater or DBP of 10 mmHg or greater within 3 minutes of standing**.
- **Out-of-Office Confirmation (HBPM or ABPM — Class I):** Because **White-Coat Hypertension** (common in older patients with ISH), **nocturnal non-dipping**, and autonomic hypotensive episodes are common in the elderly, **Home (HBPM) or 24-hour Ambulatory Blood Pressure Monitoring (ABPM)** is strongly recommended prior to escalating therapy.
- **Pseudohypertension (Osler Maneuver) and the Auscultatory Gap:**
  - **Pseudohypertension:** Severe medial brachial artery calcification resists cuff compression, yielding falsely elevated cuff pressures relative to true intra-arterial pressure (suspected when high BP is accompanied by postural dizziness on minimal medication and absence of target-organ damage).
  - **Auscultatory Gap:** Inflate the cuff at least 30 mmHg above the disappearance of the radial pulse (palpatory systolic pressure) to avoid underestimating SBP due to a silent auscultatory gap in severe ISH.

IV GUIDELINE-DIRECTED MANAGEMENT (ESC 2024 / ESH 2023 GUIDELINES)

Patient Category & Functional Status	Office BP Threshold to Initiate Drug Therapy	Treated Systolic / Diastolic BP Target (ESC 2024 / ESH 2023)	Key Prescribing Strategy
Fit Older Adults (Age 65–79 yr, Non-Frail)	**>= 140/90 mmHg** (>=130/80 mmHg if high CVD risk)	**SBP 120–129 mmHg** (if well tolerated; otherwise **130–139 mmHg**) and **DBP 70–79 mmHg**	Dual low-dose combination therapy (CCB + RAAS blocker or Diuretic); monitor eGFR/K+
Fit "Very Old" Adults (Age >=80 yr, Non-Frail)	**SBP >= 160 mmHg** (consider at **140–159 mmHg** if robust)	**SBP 140–150 mmHg** (ESH 2023) or **130–139 mmHg if tolerated** (ESC 2024); avoid SBP <120 or DBP <70 mmHg	Often initiate **monotherapy** or low-dose dual therapy ("Start Low, Go Slow")
Moderately-to-Severely Frail (CFS >=6), Institutionalized, or Orthostatic Hypotension	Individualized (typically **SBP >= 160 mmHg**)	**Individualized symptom-safe target (<150 mmHg)**; **Deprescribe / step-down** if SBP <120–130 mmHg or falls occur	Monotherapy preferred; avoid polypharmacy (>3 drugs)

- **Pharmacological Drug Selection:**
  - **First-Line Agents (Ideal for ISH & Low-Renin State):** Long-acting **Dihydropyridine Calcium Channel Blockers (Amlodipine 2.5 to 10 mg)** and **Thiazide-like Diuretics (Chlorthalidone 12.5 to 25 mg or Indapamide 1.5 mg SR)**—both proven in landmark ISH trials (**SHEP, Syst-Eur, HYVET**)—combined with an **ACE Inhibitor or ARB**. (Monitor serum **sodium** closely on thiazides, as elderly patients—especially elderly women—are highly susceptible to **thiazide-induced hyponatremia**.)
  - **Agents to Avoid as Routine Antihypertensives: Alpha-1 Blockers (Prazosin, Doxazosin)** and **Centrally Acting Sympatholytics (Clonidine, Methyldopa)** due to severe **orthostatic hypotension, syncope, sedation, and hip-fracture falls** (unless alpha-blockers are specifically required for benign prostatic hyperplasia, with bedtime dosing caution).
  - **The Diastolic Blood Pressure "J-Curve" Caveat:** In older patients with wide-pulse-pressure ISH and obstructive coronary artery disease, lowering SBP aggressively can drive **DBP below 60 to 70 mmHg**; because the left ventricular subendocardium is perfused exclusively during diastole, excessive diastolic lowering can compromise coronary perfusion pressure and precipitate myocardial ischemia.

V LANDMARK CLINICAL TRIALS IN OLDER ADULTS

- **SHEP (1991 — Chlorthalidone) and Syst-Eur (1997 — Nitrendipine) Trials:** Definitively proved that treating **Isolated Systolic Hypertension (SBP >=160 mmHg with DBP <90 mmHg)** in patients aged 60 years or older reduced stroke by **36 to 42 percent**, and SHEP roughly halved heart failure.
- **HYVET Trial (*NEJM* 2008 — Hypertension in the Very Elderly Trial):** Randomized 3,845 patients aged **80 years or older** with sustained SBP >=160 mmHg to **Indapamide SR (1.5 mg) +/- Perindopril** versus placebo (targeting <150/80 mmHg). Halted early after demonstrating a **30 percent reduction in fatal/non-fatal stroke**, a **64 percent reduction in heart failure**, and a **21 percent reduction in all-cause mortality**—proving that chronological age >=80 years is never a barrier to treatment in ambulatory seniors.
- **SPRINT-Senior (*JAMA* 2016 — Age >=75 Years) and STEP Trial (*NEJM* 2021 — Age 60 to 80 Years):**
- **SPRINT-Senior (n = 2,636, age >=75 yr):** Intensive BP lowering (**target SBP <120 mmHg** vs. <140 mmHg) reduced primary MACE by **34 percent** and all-cause mortality by **33 percent**, including in mildly frail community-dwelling seniors, without increasing injurious falls.
- **STEP Trial (n = 8,511 Chinese patients aged 60–80 yr):** Targeting **SBP 110 to <130 mmHg** versus 130 to <150 mmHg reduced composite cardiovascular events by **26 percent**.

KEY REFERENCES

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- **Williamson JD, Supiano MA, Applegate WB, et al. (SPRINT Research Group):** Intensive vs Standard Blood Pressure Control and Cardiovascular Disease Outcomes in Adults Aged >=75 Years, *JAMA*, 2016.