HYPERTENSION STUDY GUIDE 2025

Family Medicine Course

FIFTH YEAR Mutah University

Definition

Hypertension is a sustained elevation of systemic arterial blood pressure, most commonly defined as a systolic blood pressure (BP) ≥ 140 mm Hg or diastolic BP ≥ 90 mm Hg, but definitions vary by professional organization and cardiovascular risk.

Ex. AHA/ACC 2017: 130/80 is the cut point for diagnosis

ESH 2024, NICE guidelines, JNC 8: 140/90 is the cut point for diagnosis

Types

Essential hypertension is also called idiopathic or primary hypertension; which represents most cases of hypertension.

Resistant hypertension is defined as elevated blood pressure despite concurrent use of maximum dose of 3 antihypertensive drugs of different classes, including a diuretic.

***Secondary hypertension** is defined as hypertension due to an identifiable, potentially curable cause.

Masked hypertension is defined as elevated blood pressure at home or on ambulatory blood pressure monitoring but normal office blood pressure.

White coat hypertension is defined as normal blood pressure at home or on ambulatory blood pressure monitoring but elevated office blood pressure. See High Blood Pressure - Differential Diagnosis for additional information.

Labile hypertension is characterized by sudden increases in blood pressure usually attributed to emotional stress, and is considered a clinical impression as opposed to a concrete diagnosis.

*Among secondary causes in general:

More common causes include:

Obstructive sleep apnea (OSA)

Hyperaldosteronism (primary aldosteronism or secondary hyperaldosteronism)

Renal parenchymal disease

Renal artery stenosis

Drugs and other substances, including but not limited to:

1) Alcohol
2) Caffeine

3) Nonsteroidal anti-inflammatory drugs (NSAIDs)
4) Decongestants (for example, phenylephrine and pseudoephedrine)
5) Systemic corticosteroids
6) Immunosuppressants
7) Oral contraceptives
8) Antidepressants
9) Second-generation antipsychotics
10) Amphetamines
11) Herbal supplements (for example, Ma Huang and St. John's wort)
12) Recreational drugs (for example, "bath salts," cocaine, and methamphetamine)
13) Angiogenesis inhibitor (for example, bevacizumab) or tyrosine kinase inhibitors (for example, sunitinib and sorafenib)

Less common causes include:

Thyroid disease

Cushing disease

Pheochromocytoma (or pseudopheochromocytoma)

Coarctation of aorta

Rare causes include:

Primary hyperparathyroidism

Congenital adrenal hyperplasia

Mineralocorticoid excess syndromes other than primary aldosteronism

Acromegaly

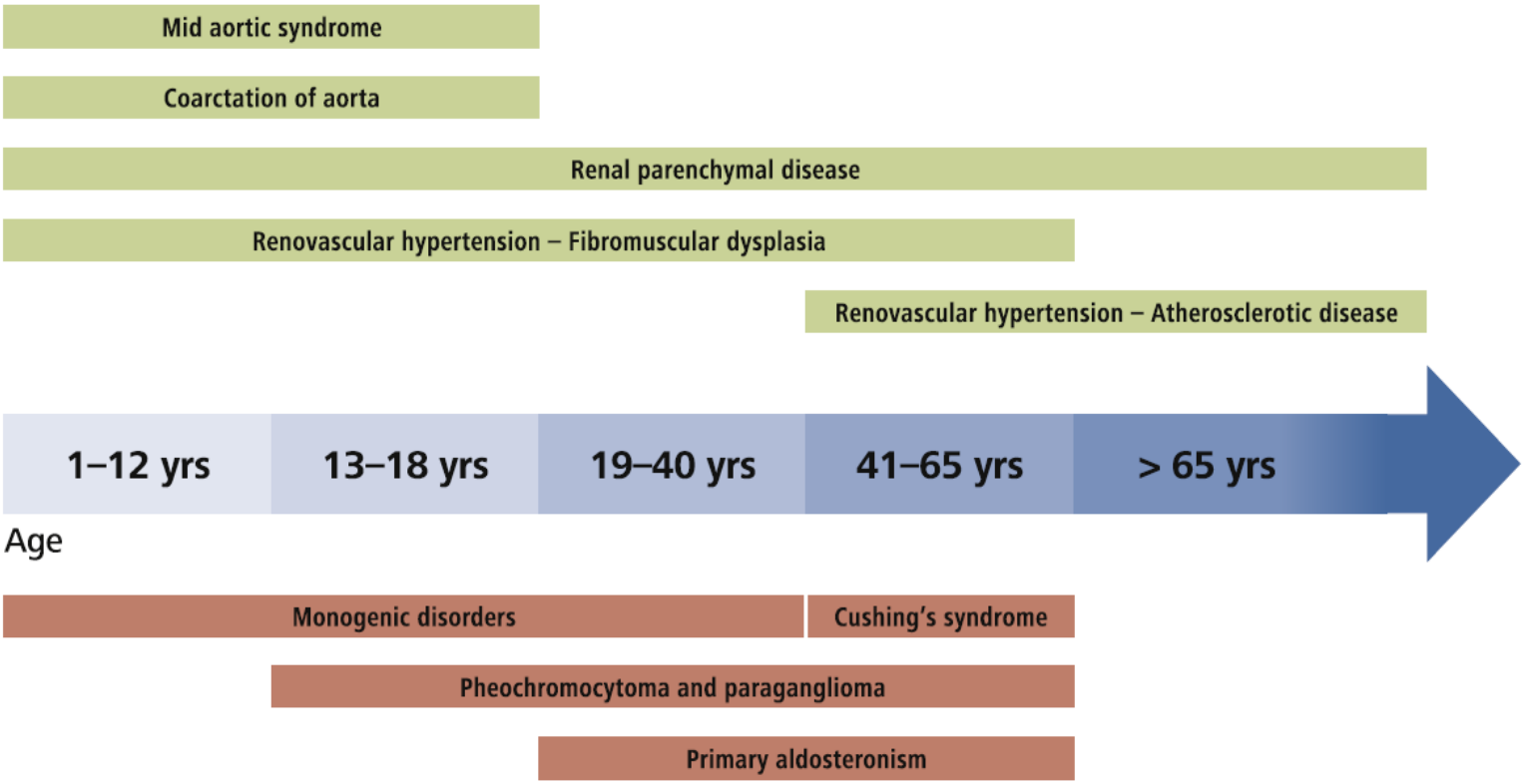


Fig. 4. Incidence of selected forms of secondary hypertension according to age.

Prevalence in Jordan

According to Khader et al. 2019; the crude prevalence of *hypertension was 41.4% among men and 28.3% among women. The age-standardized prevalence was 33.8% among men and 29.4% among women. The prevalence of hypertension increased significantly with increasing age among men and women. Only 30.7% of men and 35.1% of women who were on antihypertensive medications had their blood pressure controlled.

**(Hypertension in this study was defined as average measured blood pressure ≥140 mm Hg systolic and/or 90 mm Hg diastolic)*

Screening

USPSTF Recommendations

- United States Preventive Services Task Force (USPSTF) recommends screening for high blood pressure (BP) in all adults ≥ 18 years old (USPSTF Grade A).

- BP thresholds vary by professional organization, and range from ≥ 130/80 mm Hg to ≥ 140/90 mm Hg.

- Screening interval:

1. Annually for all of the following patients:

a) ≥ 40 years old
b) High-normal BP
c) Overweight or obese
d) Black persons

2. Every 3-5 years in patients aged 18-39 years with normal BP and not at increased risk for hypertension

Evaluation

1. Measure blood pressure–diagnose

The accurate measurement of blood pressure (BP) is the cornerstone for the diagnosis and management of hypertension. The measurement of BP to diagnose hypertension therefore represents the first pivotal step of the ESH **MASTER**plan for the management of hypertension.

• **Conventional attended office BP measurement (OBPM)** is the most well-studied method for assessing BP and the one by which the diagnosis of hypertension, BP classification, the role of BP as a cardiovascular (CV) risk factor, the protective effect of antihypertensive treatment and the BP thresholds and targets of therapeutic interventions have been established.

• **Ambulatory BP monitoring (ABPM) and home BP monitoring (HBPM)** are important methods for out-of-office BP monitoring, that provide important additional information for the management of hypertensive patients.

Measure blood pressure with the appropriate cuff size in a calm, seated position and with the patient's arm supported at the level of the heart.

A hypertension diagnosis is based on ≥ 2 blood pressure measurements per visit, at ≥ 2 visits, with systolic blood pressure (SBP) ≥ 140 mm Hg and/or diastolic blood pressure (DBP) ≥ 90 mm Hg when using manual measurement methods.

BP measurement technique:

- The patient should be seated and relaxed for > 5 minutes with the arm rested on a support at the level of the heart, with back supported, and with feet flat on the floor.

- The patient should avoid caffeine, exercise, and smoking for ≥ 30 minutes before measurement.

- Ensure patient has emptied their bladder, and neither patient nor observer should talk during rest period or during measurement.

- The inflatable bladder of the cuff should encircle 80% of the patients' arm circumference. Clothing should be removed from the location of cuff placement.

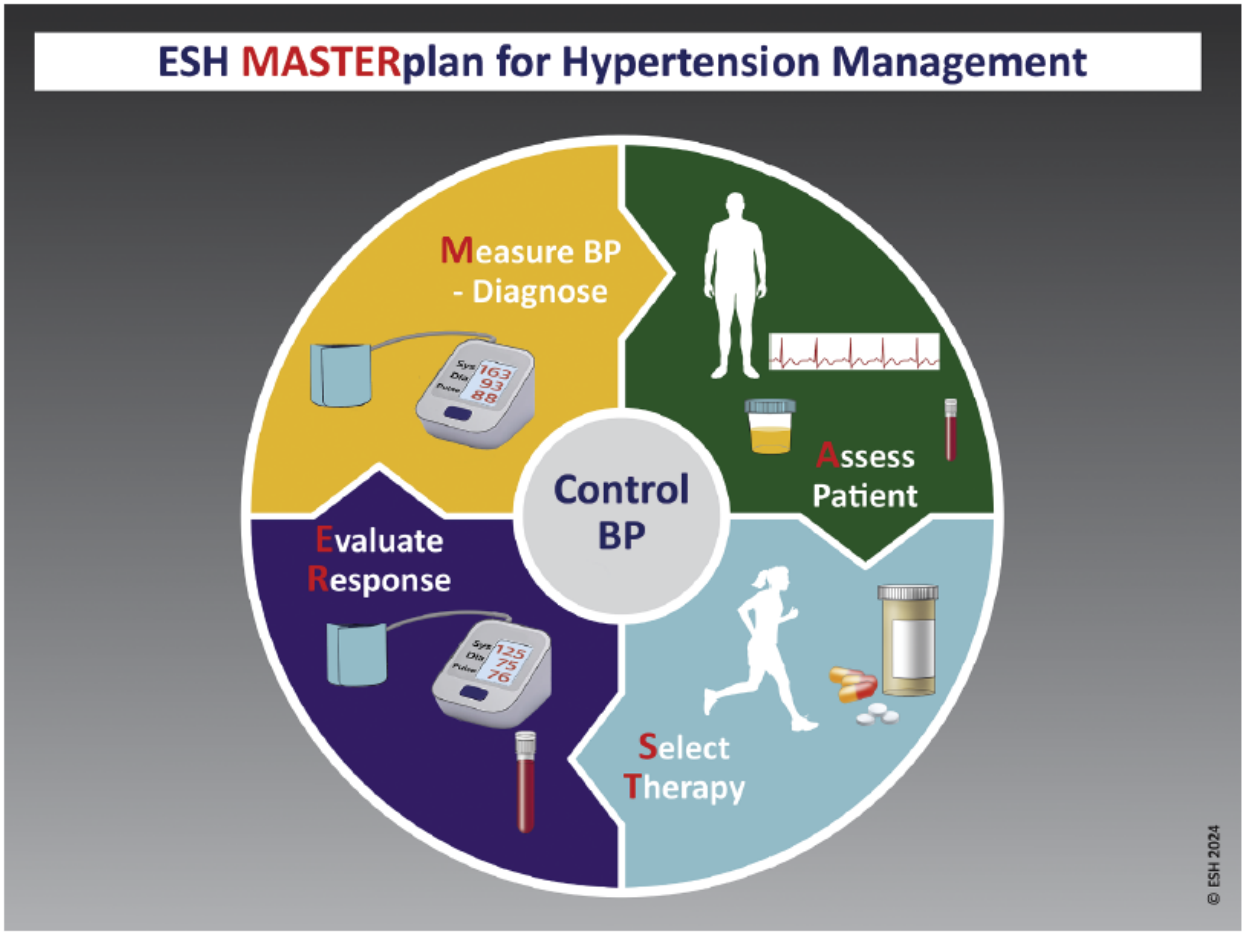
- Systolic and diastolic blood pressure is recorded as the onset of the first Korotkoff sound and the disappearance of all Korotkoff sounds, respectively. Use the nearest even number.

- Use a validated blood pressure measurement device and ensure the device is calibrated periodically.

- Separate repeated measurements by 1-2 minutes.

- Use an average of ≥ 2 readings obtained on ≥ 2 occasions to estimate blood pressure.

The ESH developed a **MASTER** plan for the management of hypertension:



Measure Blood Pressure - Diagnose

In Office

Office BP measurement (OBPM)

*SBP ≥140
and/or
DBP ≥90

Conditions

1. Use validated automated electronic upper-arm cuff device[a] (www.stridebp.org).
2. Select appropriate cuff to fit arm size according to instructions by device manufacturer[b].
3. Quiet room with comfortable temperature.
4. No smoking, caffeine, food, or exercise 30 min before measurement.
5. Start measurement after patient remained seated and relaxed for 3-5 min[c].
6. No talking during and between measurements.

Posture

7. Sitting with back supported on chair.
8. Legs uncrossed, feet flat on floor.
9. Bare arm resting on table with mid-arm at heart level.

Measurement

10. Take 3 readings with 1 min intervals between them. Use the average of the last 2 readings for BP and also for pulse rate[d].

Relevance

- Was used in outcome trials and provides the basis for diagnosis and BP targets.

Out-of-office

Home BP monitoring (HBPM)

*SBP ≥135
and/or
DBP ≥85

Conditions and Posture

1.-9. From OBPM apply also to HBPM.

Measurement

10. Propose a standardized protocol to the patient:
    - Educate the patient on how to use a validated device and report the data.
    - Take 2 readings with 1 min intervals between them.
    - Measure in the morning and the evening (before drug intake if treated).
    - Measure for 3–7 days before office visits.
    -Use the average of all readings excluding the first day for both BP and pulse rate.
11. For long-term follow-up of treated hypertension, make duplicate measurements once or twice per week or month.

Relevance

- Recommended for long-term follow-up of treated hypertension, because it improves BP control, especially when combined with education and counseling.
- Confirmation of hypertension diagnosis and of true resistant hypertension, particularly if ABPM is not available.

Ambulatory BP monitoring (ABPM)

*24-h mean BP:
SBP ≥130
and/or
DBP ≥80

*Daytime (awake):
SBP ≥135 mmHg
and/or
DBP t≥85

*Nighttime (asleep):
SBP ≥120 mmHg
and/or
DBP ≥70

Conditions

1.-2. From OBPM applies also to ABPM.
3. Use fully automated devices programmed to record BP automatically at preselected intervals for 24 h.

Measurement

4. The recommended optimal time interval between measurements should be 20 minutes during day (awake) and night (sleep).
5. Measure during a routine workday for 24 h.
6. Instruct patients to keep a diary of their activities, symptoms, meals, drug intake times, sleep times or any unusual problems.

Relevance

- Obtaining 24-h BP profile and especially BP during night (sleep) not captured by OBPM or HBPM
- Confirmation of hypertension diagnosis and of true resistant hypertension.

*Definition of hypertension [a]A device that takes triplicate readings automatically is preferred. [b]The selection of an appropriate cuff size is crucial. A smaller than required cuff overestimates BP and a larger underestimates BP. [c]Use of electronic devices allowing automated storage and data transfer is encouraged. [d]At initial visit measure on both arms. An interarm SBP difference >10 mmHg must be confirmed with repeated measurements. If confirmed, the arm with the higher BP should be used for all subsequent measurements. If any two sequential BP readings in one arm differ by >10 mmHg, additional measurements are recommended. See also Table 1.

Table 1
Clinical indications for home and ambulatory BP monitoring*.

Conditions in which white-coat hypertension is more common, e.g.
• Grade I hypertension on office BP measurement
• Marked office BP elevation without HMOD
Conditions in which masked hypertension is more common, e.g.
• High-normal office BP
• Normal office BP in individuals with HMOD or at high total CV risk
In treated individuals
• Confirmation of uncontrolled and true resistant hypertension
• Evaluation of 24 h BP control (especially in high-risk patients)
• Evaluating symptoms suggestive of hypotension (especially in older patients)
Suspected postural or postprandial hypotension in treated patients
Exaggerated BP response to exercise
Considerable variability in office BP measurements
Specific indications for ABPM rather than HBPM
• Assessment of nocturnal BP and dipping status (e.g. sleep apnea, CKD, diabetes, endocrine hypertension, or autonomic dysfunction)
• Patients incapable or unwilling to perform reliable HBPM, or anxious with self-measurement
• Evaluation of patients considered for renal denervation
• Children
• Pregnancy
Specific indications for HBPM rather than ABPM
• Long-term follow-up of treated individuals to improve adherence with treatment and hypertension control
• Patients unwilling to perform ABPM, or with considerable discomfort during the recording
Indications for repeat out-of-office BP evaluation (same or alternative method – HBPM/ABPM)
• Confirmation of white-coat hypertension or masked hypertension in untreated or treated individuals

2. Assess patient

A thorough patient work-up aims to gather essential information about the patient's personal and medical history, any other relevant factors and co-morbidities that may impact their BP, CV risk and management.

This information is critical in determining the initiation of the most appropriate treatment approach and the follow-up strategy.

A. Basic assessment

Due to the high prevalence of hypertension and thus the large number of individuals that will be managed by primary care providers, it is essential to prioritize the basic assessment to investigations that are effective and feasible in this setting, allowing widespread implementation.

Assess Patient

Assessment of patients should be adapted according to the severity of hypertension and clinical circumstances.

Basic

History

- Personal
  - -Hypertension related, including sex-specific aspects, e.g. HDP
  - -Co-morbidities
- Cardiovascular risk factors
- Symptoms of HMOD, CVD, stroke or CKD
- Possible secondary hypertension
- Other drug treatments or use including OTCs (See also Table 2 and Table 3)

Physical Examination

- Body habitus and BMI
- Signs of HMOD
- Signs of secondary hypertension
- Resting pulse rate (see BP measurement)
- Level of frailty/functionality in older persons (e.g. >80 years) (See also Table 4 and Table 5)

Lab Test

Blood (serum/plasma)

- Creatinine, eGFR
- Potassium and sodium
- (Fasting) glucose and HbAlc
- Total-, LDL, HDL-Cholesterol

Urine

- Urine analysis multicomponent dipstick test
- Urinary albumin-creatinine ratio (See also Table 6)

Other Investigations

- 12 lead ECG

Extended

HMOD
Select if deemed necessary and available

- Echocardiography
- MRI
- Coronary calcium score

- Ultrasound
- Doppler ultrasound

- Carotid ultrasound

- Pulse wave velocity
- Ankle-brachial index

- CT
- MRI
- Cognitive function tests

- Fundoscopy
- Retina microvasculature

(See also Table 3)

When to refer a patient

To a specialist

- Suspected secondary hypertension (depending on age, Figure 4)
- To exclude secondary hypertension in younger patients (<40 years) with grade 2 or 3 hypertension
- Sudden onset or aggravation of hypertension
- Patients with treatment resistant hypertension
- Need of more detailed assessment of HMOD, which might influence decision making (treatment and follow-up).
- Hypertension in pregnancy
- Requirement of more in-depth specialist evaluation from the referring physician

To a hospital

- Hypertensive emergencies, i.e. in severe hypertension (grade 3) associated with acute symptomatic HMOD
- Severe hypertension with conditions that need intensified BP management:
  - Acute stroke
  - Complicated aortic aneurysm
  - Acute heart failure
  - Acute coronary syndrome
  - Acute kidney failure
- Hypertension caused by phaeochromocytoma or exogenous sympathomimetic substances (e.g. substance abuse)
- Severe forms of HDP including preeclampsia/eclampsia

The basic assessment includes personal and medical history, physical examination, lab tests and the recording of a 12-lead resting electrocardiogram (ECG).

The recommended evaluation allows the diagnosis and staging of chronic kidney disease (CKD), by assessing estimated glomerular filtration rate (eGFR) and urinary albumin-creatinine ratio (UACR), and of left ventricular hypertrophy (LVH) by ECG (although with limited sensitivity).

An extended list of factors to be considered in the evaluation of patient's history is shown in Table 2.

Table 2

Medical and family history.

Personal history

• Time of the first diagnosis of hypertension, including records of any previous medical screening, hospitalization

• Stable or rapidly increasing BP

• Recordings of current and past HBPM values

• Current/past antihypertensive medications including their effectiveness and intolerance • Adherence to therapy

• Previous hypertension in pregnancy/preeclampsia

Risk factors

• Family history of hypertension, CVD, stroke or kidney disease

• Smoking history

• Dietary history, alcohol consumption

• High volume of sedentary behavior and lack of physical activity

• Weight gain or loss in the past • History of erectile dysfunction

• Sleep history, snoring, sleep apnea (information also from partner)

• Stress

• Long-term cancer survivor

History, signs and symptoms of HMOD, CVD, stroke and kidney disease

• Brain and eyes: headache, vertigo, syncope, impaired vision, TIA, sensory or motor deficit, stroke, carotid revascularization, cognitive impairment, memory loss, dementia (in older people) • Heart: chest pain, shortness of breath, edema, myocardial infarction, coronary revascularization, syncope, history of palpitations, arrhythmias (especially AF), heart failure

• Kidney: thirst, polyuria, nocturia, hematuria, urinary tract infections

• Peripheral arteries: cold extremities, intermittent claudication, pain-free walking distance, pain at rest, ulcer or necrosis, peripheral revascularization

• Patient or family history of CKD (e.g. polycystic kidney disease)

History of possible secondary hypertension

• Young onset of grade 2 or 3 hypertension (<40 years), or sudden development of hypertension or rapidly worsening BP in older patients

• History of repetitive renal/urinary tract disease

• Repetitive episodes of sweating, headache, anxiety or palpitations, suggestive of pheochromocytoma

• History of spontaneous or diuretic-provoked hypokalemia, episodes of muscle weakness and tetany (hyperaldosteronism)

• Symptoms suggestive of thyroid disease or hyperparathyroidism

• History of or current pregnancy, postmenopausal status and oral contraceptive use or hormonal substitution

Drug treatments or use (other than antihypertensive drugs)

• Recreational drug/substance abuse, concurrent therapies including on prescription drugs, e.g. glucocorticoids, NSAIDs/COX-2 inhibitors, immunosuppressive drugs, anticancer drugs, nasal decongestants.

An extended list of factors that influence CV risk in patients with hypertension is shown in Table 3. The American Heart Association (AHA) developed and recommends the PREVENT equations and the Pooled Cohort Equations to estimate 10-year ASCVD risk but SCORE2 and SCORE-OP is commonly used in Europe.

SCORE2: https://www.mdcalc.com/calc/10499/systematic-coronary-risk-evaluation-score2#next-steps

ASCVD risk calculator: https://www.mdcalc.com/calc/3398/ascvd-atherosclerotic-cardiovascular-disease-2013-risk-calculator-aha-acc

Table 3

Factors that influence CV risk in patients with hypertension.

Parameter for risk stratification, which are included in SCORE2 and SCORE2-OP

Sex (men >women)

Age

Level of SBP

Smoking – current or past history

Non-HDL cholesterol

Established and suggested novel factors

Family or parental history of early onset hypertension

Personal history of malignant hypertension

Family history of premature CVD (men aged <55 years; women aged <65 years)

Heart rate (resting values >80 bpm)

Low birth weight

Sedentary lifestyle

Overweight or Obesity

Diabetes

Dyslipidemia Lp(a)

Uric acid Adverse outcomes of pregnancy (recurrent pregnancy loss, preterm delivery, hypertensive disorders, gestational diabetes)

Early-onset menopause

Frailty, functional capacities and autonomy status

Psychosocial and socioeconomic factors

Migration

Environmental exposure to air pollution or noise

Additional clinical conditions or comorbidities

True resistant hypertension

Sleep disorders (including OSA)

COPD

Gout

Chronic inflammatory diseases

Metabolic dysfunction-associated fatty liver disease
Chronic infections (including long COVID-19)
Migraine
Depressive syndromes
Erectile dysfunction
Hypertension-mediated organ damage (HMOD)
Increased large artery stiffness
Pulse pressure (in older people) ≥60 mmHg
Presence of non-hemodynamically significant atheromatous plaque (stenosis) on imaging
ECG LVH
Echocardiographic LVH
Moderate increase of albuminuria 30–300 mg/24 h or elevated UACR (preferably in morning spot urine) 30 –300 mg/g
CKD stage 3 with eGFR 30–59 ml/min/1.73 m2
Ankle–brachial index <0.9 Advanced retinopathy: hemorrhages or exudates, papilledema
Established cardiovascular and kidney disease
Cerebrovascular disease: ischemic stroke, cerebral hemorrhage, TIA
Coronary artery disease: myocardial infarction, angina, myocardial revascularization
Presence of hemodynamically significant atheromatous plaque (stenosis) on imaging
Heart failure
Peripheral artery disease
Atrial fibrillation
Severe albuminuria > 300 mg/24 h or UACR (preferably in morning spot urine) >300 mg/g CKD stage 4 and 5, eGFR < 30 mL/min/1.73m2

Further details for a comprehensive physical examination for patients with hypertension are summarized in Table 4.

Table 4
Comprehensive physical examination for hypertension[a].

Body habitus
• Weight and height measured on a calibrated scale, with calculation of BMI
• Waist circumference
Signs of hypertension-mediated organ damage
• Neurological examination and cognitive status
• Fundoscopic examination for hypertensive retinopathy in emergencies
• Auscultation of heart and carotid arteries
• Palpation of carotid and peripheral arteries
• Ankle–brachial index
Signs of secondary hypertension
• Skin inspection: cafe-au-lait patches of neurofibromatosis (pheochromocytoma)
• Kidney palpation for signs of renal enlargement in polycystic kidney disease
• Auscultation of heart and renal arteries for murmurs or bruits indicative of aortic coarctation, or renovascular hypertension
• Signs of Cushing's disease or acromegaly
• Signs of thyroid disease

Table 6 shows selected basic and extended laboratory tests for assessment of hypertensive patients

Table 6
Selected standard laboratory tests for work-up of hypertensive patients[a].

Blood (serum/plasma)
• Hemoglobin and/or hematocrit
• Fasting blood glucose and HbA1c
• Lipids: total cholesterol, LDL cholesterol, HDL cholesterol, triglycerides
• Potassium and sodium
• Uric acid
• Creatinine (and/or cystatin C) for estimating GFR with eGFR formulas
• Calcium
Urine
• Multicomponent dipstick test, UACR (preferably early morning spot urine), microscopic examination in selected patients

B. Extended assessment

The extended assessment of hypertension mediated organ damage (HMOD) can be executed as deemed necessary and available to physicians as shown in the figure above.

C. When to refer a patient

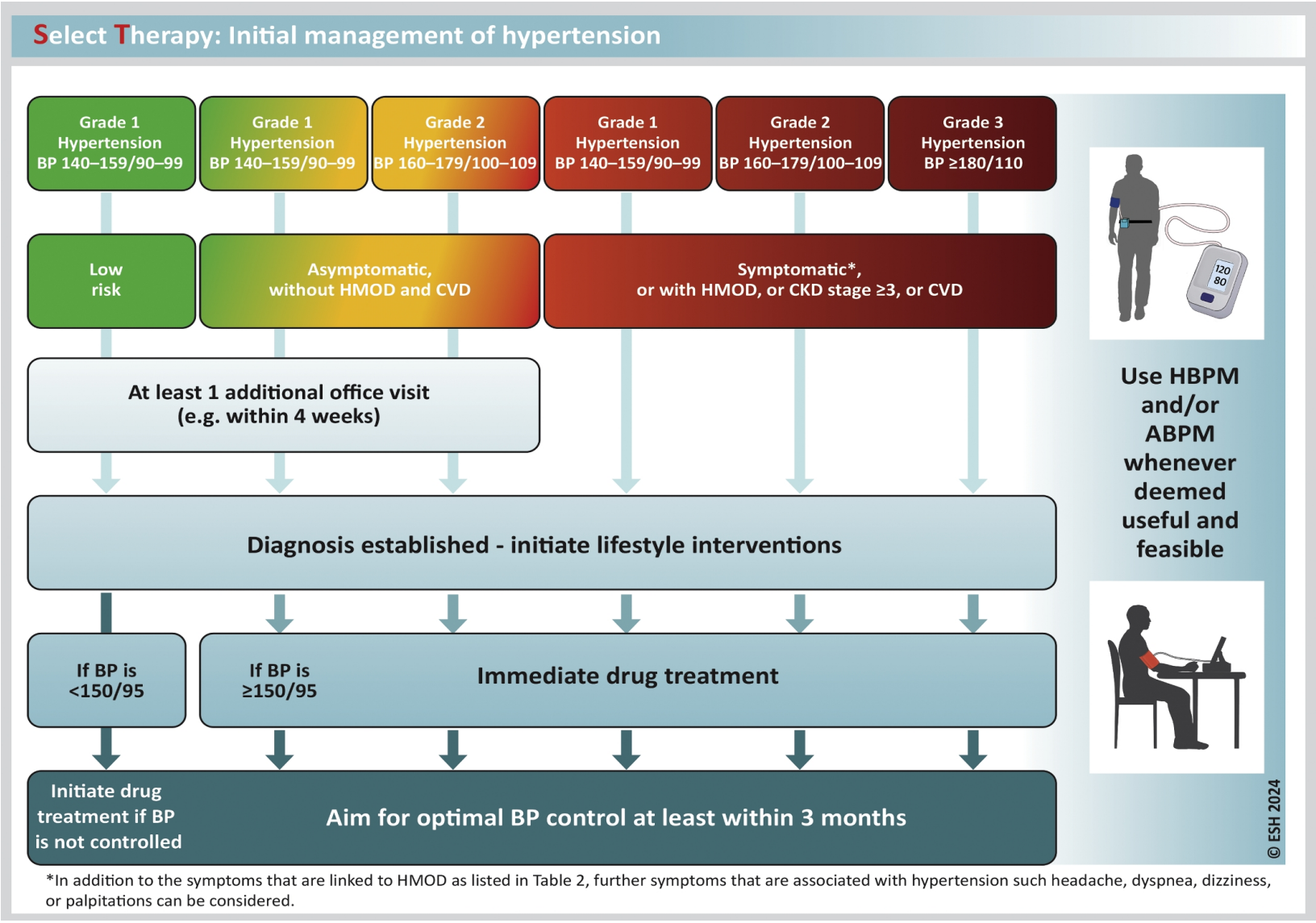
Basic and extended assessment of patients should support decision making about when a patient should be referred to a hypertension specialist or a hospital (including the need for inpatient treatment) as shown in the figure above.

3. Select therapy

A. General aspects

Lifestyle interventions have been shown to be effective in reducing BP in hypertensive patients and can also have additional benefits, such as increasing the efficacy of BP-lowering therapy, improving overall CV health and reducing the risk of other chronic diseases.

They are important in the prevention of hypertension and may control BP when used alone already in a fraction of patients, i.e. in patient with mildly elevated grade 1 hypertension with



systolic BP (SBP) and diastolic BP (DBP) <150/95 mmHg and low CV risk. However, most patients with hypertension should be treated with a combination of both lifestyle interventions and pharmacological treatment. The strategy for the initial management of hypertension with the aim to control BP within 3 months according to the risk of patients is shown in the figure below.

- According to AHA/ACC guidelines; in patients with stage 1 HTN (130-139/80-89), recommendations are to start antihypertensive medications if the patient has a **10-year**

ASCVD risk of 10% or higher with a target of BP less than 130/80 to prevent patients from cardiovascular events. The recommendation is for patients with stage 1 HTN and a **10-year ASCVD risk of less than 10%** to have lifestyle modification measures.

- All patients with **stage 2 HTN** (> or = 140/90) should start antihypertensive medications to lower BP to a target lower than 130/80 mm Hg, even if the 10-year ASCVD risk is less than 10%.

Guideline Similarities	2017 ACC/AHA	2023 ESH
Accurate Blood Pressure Measurement	Office-based BP measurements and use of validated, cuffed devices and home/ambulatory BP monitoring are recommended prior to diagnosing hypertension.	
Cardiovascular Risk Calculator for Treatment Thresholds	Pooled Cohort Equation and SCORE2/SCORE2-OP provide estimates for 10-year risk of fatal and non-fatal cardiovascular events and should be used to guide treatment decisions.	
Initial Pharmacotherapy Recommendations	Initial therapeutic choices include ACE inhibitors, angiotensin-receptor blockers, thiazide or thiazide-like diuretics, and calcium channel blockers. Single pill combination therapy is a first-line strategy for many patients.	
Guideline Differences	**2017 ACC/AHA**	**2023 ESH**
Hypertension Definition	≥ 130/80	≥ 140/90
Normal BP Ranges (mmHg)	Normal: < 120/80 Elevated: 120-129/<80	Optimal: < 120/80 Normal: 120-129/80-84 High-Normal: 130-139/85-89
Hypertensive BP Ranges (mmHg)	Hypertension Stage 1: 130-139/80-89 Hypertension Stage 2: ≥ 140/90	Hypertension Grade 1: 140-159/90-99 Hypertension Grade 2: 160-179/100-109 Hypertension Grade 3: ≥ 180/110
BP Targets for Treatment		
18 – 64 years (mmHg)	< 130/80	< 130/80
65-79 years (mmHg)	< 130/80	< 140/80*
≥ 80 years (mmHg)	< 130/80	140-150/<80
Pharmacotherapy	Initial therapy with beta-blockers reserved for specific conditions including ischemic heart disease or heart failure	Beta blockers included as first-line therapy for hypertension.

** Target < 130/80 if tolerated*

B. *Lifestyle interventions*

The recommended lifestyle interventions together with their relevance and prescribing patterns are shown below.

Select Therapy: Lifestyle Interventions

Relevance

- Prevent or delay onset of hypertension
- Improve overall/CV health and well-being
- Reduce BP
- Booster BP lowering effects of medications
- Reduce the number/dose of drugs needed for BP control

Prescribing

- To all patients with diagnosed hypertension
- To patients with white-coat or masked hypertension
- To patients with high-normal BP
- Individual patient counseling and support
- Prescribe with specific instructions, e.g. intensity and type of exercise
- Assess, adapt, and reinforce during follow-up

Supportive additional interventions

Smoking cessation

- Smoking cessation, supportive care and referral to smoking cessation programs are recommended for all smokers

Key interventions to reduce BP

Healthy diet

Prefer:

- DASH or Mediterranean type diets
- A healthy dietary pattern including more plant-based and less animal-based food
- Vegetables , fruits, beans, nuts, seeds, and vegetable oils
- Lean protein (e.g. fish, poultry)

Limit:

- Fatty meats, full-fat dairy
- Sugar, sweets and sweetened beverages

Daily physical activity and regular exercise

- Incorporate physical activity (e.g. walking, cycling) into everyday life and reduce sedentary behavior (e.g. sit less)
- Aim for:
  -150-300 min of aerobic exercise per week performed at a moderate intensity or
  -75-150 min of aerobic exercise per week performed at a vigorous intensity or
  -an equivalent combination of moderate and vigorous physical activities
- Add dynamic resistance (muscle strengthening) exercise 2-3 times per week
- Start slow and gradually to build up the amount/ intensity of activity

Weight reduction

- Combine a low-caloric diet with daily physical activity in patients with overweight or obesity
- Monitor waist circumference and weight

Restriction of sodium intake

- Sodium is mainly consumed as salt, which comes from processed foods or is added to the food during cooking or at the table
- Salt (NaCl) restriction to < 5 g (~2g sodium) or 1 teaspoon per day is recommended

Augmentation of potassium intake

- Increase potassium consumption, preferably via dietary modification, except for hypertensive patients with advanced CKD
- Foods high in potassium are for example white cannellini beans (1200 mg/cup), unsalted boiled spinach (840 mg/cup), avocado (708 mg/cup) and bananas (450 mg per medium fruit)
- Use salt substitutes replacing NaCl with KCl in patients consuming a high sodium diet

Limit alcohol intake

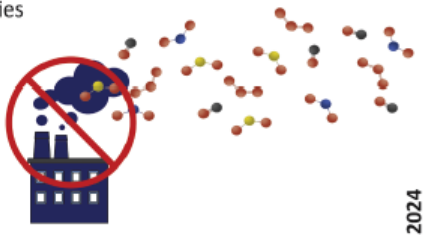
- Limit alcohol intake close to abstinence, particularly if intake is ≥3 drinks/day[a]
- Avoid excessive (binge) drinking

Improve stress management

- Reduce stress by use of
  -Regular physicial activity
  -Mindfulness-based exercise
  -Relaxation techniques, e.g. deep breathing, meditation, yoga or Tai Chi
- Get enough sleep (7-9 hours)
- Find individual ways to cope with stress, e.g. practicing mindfulness, engaging in hobbies or talking to a therapist
- Moderate alcohol and caffeine intake, avoid drugs

Minimize exposure to noise and air pollution

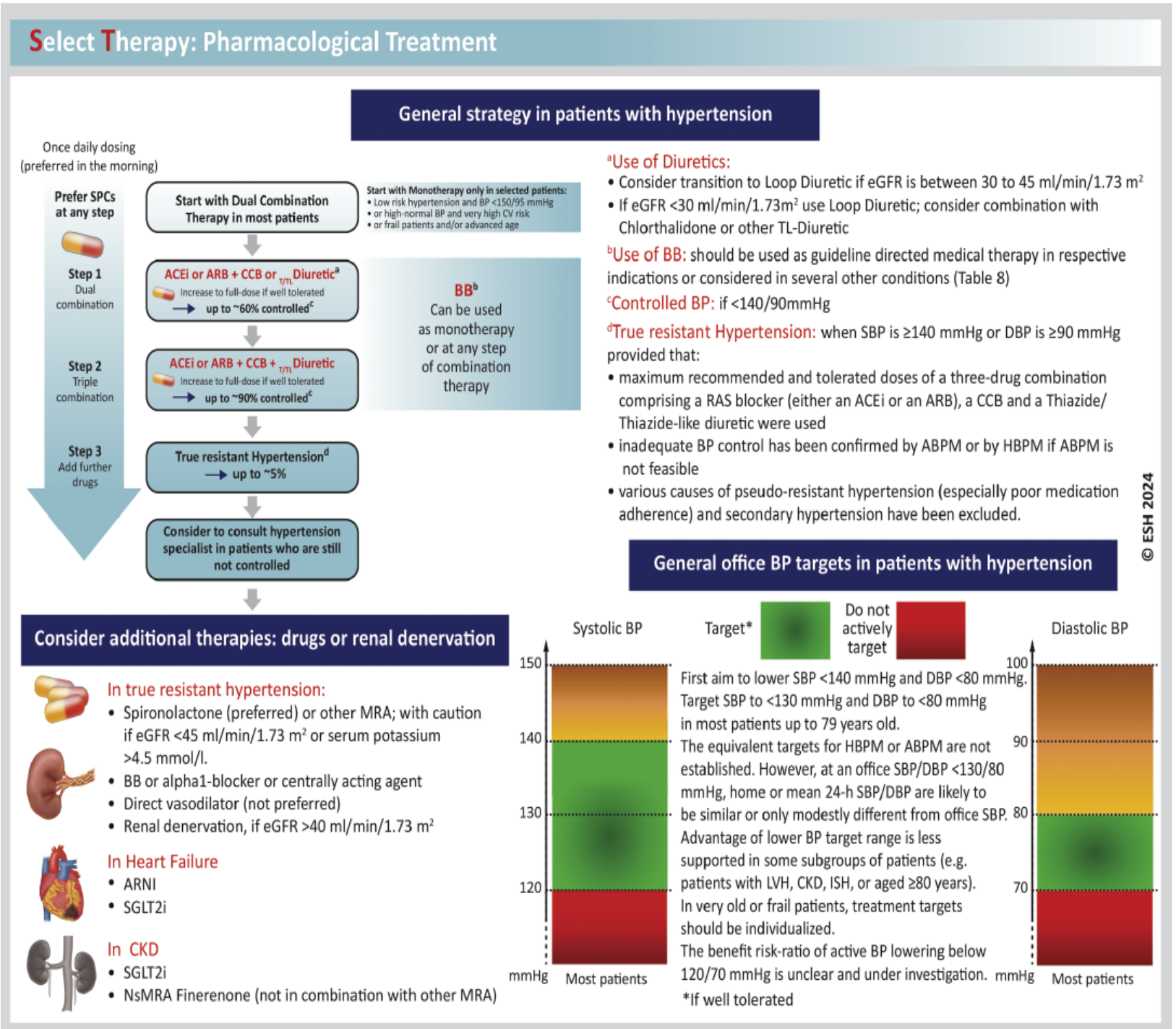
- Reduce indoor exposure to noise and air pollution.
- Consider to reduce exposure to air pollution by modifying the location, timing and type of outdoor activities



[a]About 350 ml of regular beer containing 5% alcohol by volume or 150 ml of wine containing 12% alcohol by volume per drink.

C. Pharmacological treatment

The general treatment strategy for patients with hypertension is shown below.



The recommended major BP-lowering drug classes include angiotensin-converting enzyme inhibitors (ACEi), angiotensin receptor blockers (ARB), beta-blockers (BB), calcium channel blockers (CCB) and thiazide/thiazide-like diuretics (T/TL-diuretics).

Additional therapies can be considered in patients with true resistant hypertension, heart failure and CKD as shown.

Furthermore, renal denervation can be considered in true-resistant hypertension and in patients who have uncontrolled BP despite the use of antihypertensive drug combination therapy, or if drug treatment elicits serious side effects and poor quality of life (if eGFR > 40 ml/min/1.73m2).

Antihypertensives

ACE-i (ramipril, enalapril, captopril...) ARBs (valsartan, candesartan...)

MOA: decrease blood pressure by inhibiting the angiotensin-converting enzyme; this causes a decline in the production of angiotensin II and increases the bradykinin level by inhibiting its degeneration, which leads to vasodilation.

Antihypertensives of choice for patients with heart failure, chronic kidney disease and diabetes mellitus. Independent of their antihypertensive effect, they are proven to have a cardioprotective effect in patients with a high risk of cardiovascular disease.

Both are recommended as first-line treatment for patients with left ventricular dysfunction and ST-elevation MI or non-ST elevation MI with the presence of diabetes, systolic dysfunction, or anterior infarct.

Side Effects: cough, hypotension, fatigue, and azotemia; reversible renal impairment is a common side effect, especially if the patient develops volume depletion due to diarrhea or vomiting.

Cough can occur in up to 20% of patients on ACE inhibitors. It takes up to 14 to 28 days after discontinuation for the cough to resolve. The incidence of cough is less common with ARB treatment; therefore, ARBs are safer in patients with asthma.

Mild hyperkalemia; even in patients with normal renal function. The risk of hyperkalemia increases in patients with renal failure, diabetes, or CHF.

Angioedema is a rare side effect of ACE inhibitors. ARBs are less associated with angioedema than ACE inhibitors.

CCBs (amlodipine, nifedipine....)

MOA: inhibition of Ca2+ entry to the cells can cause peripheral vasodilation, which is seen mainly in dihydropyridines, or a negative inotropic effect on the heart muscle in non-dihydropyridines, inhibiting the sinoatrial and atrioventricular nodes, leading to slow cardiac contractility and conduction.

CCBs divide into two groups: dihydropyridines and non-dihydropyridines

Dihydropyridines are more potent as vasodilators and are used more for HTN treatment. They have less effect on heart contractility and conduction. Nifedipine and amlodipine are the most used medications in this group.

Non-dihydropyridines are less potent as vasodilators and have a better effect on cardiac contractility and conduction. They are used more as antiarrhythmic medications and less for HTN treatment.

For African descent patients, initial treatment for hypertension (without evidence of heart failure or chronic kidney disease) should include CCB or a thiazide diuretic.

Dihydropyridines should not be a primary treatment for congestive heart failure (CHF) but represent a safe additional treatment in these patients for better blood pressure control or angina pectoris. Non-dihydropyridines are relatively contraindicated in patients with CHF with reduced ejection fraction, second and third-degree heart blocks, and in patients with sick sinus syndrome.

Side effects: dose-related peripheral edema. It is not related to sodium or fluid retention or developing heart failure; therefore, it does not improve with diuretics therapy; on the other hand, the combination of CCBs with ACE inhibitors or ARBs to a lesser effect showed a decreased risk of developing peripheral edema.

Dihydropyridines (amlodipine, nifedipine) can cause lightheadedness, flushing, headaches, and gingival hyperplasia.

Non-dihydropyridines (verapamil, diltiazem) are associated with bradycardia and can cause constipation in 25% of patients.

CCBs inhibit platelet aggregation and are associated with an increased risk of gastrointestinal bleeding; caution is necessary when prescribing these agents to older patients and patients with a high risk of bleeding.

Thiazide Diuretics (hydrochlorothiazide, chlorthalidone, indapamide...)

MOA: Thiazides inhibit sodium transport in the distal tubule by blocking the Na/Cl channels. Thiazides cause initial volume depletion associated with decreased cardiac output, which recovers within 6 to 8 weeks of starting the treatment in a reverse autoregulation mechanism while the blood pressure remains controlled; thiazide diuretics can acutely activate the renin-angiotensin system and cause systemic vascular resistance, which may resolve with chronic thiazide treatment, the addition of an ACE inhibitor or ARB can enhance the blood pressure control. Also, the thiazide-type diuretics have a modest vasodilation effect, although the mechanism is still unclear.

Thiazide diuretics can be used as the first-line treatment for HTN (either alone or in combination with other antihypertensives) in all age groups regardless of race unless the patient has evidence of chronic kidney disease where angiotensin-converting enzyme inhibitor or angiotensin II receptor blocker is indicated.

Chlorthalidone is the first choice for older patients with osteoporosis, as it was associated with a lower incidence of pelvic fractures.

Side effects: Most of side effects are directly related to the diuretic dose; hypokalemia and hyponatremia are the most common metabolic effects, followed by hyperuricemia, hypomagnesemia, hyperlipidemia, and increased glucose levels.

Beta-blockers (bisoprolol, metoprolol, atenolol…)

MOA: work by inhibiting the catecholamines from binding to the Beta 1, 2, and 3 receptors. Beta-1 receptors are found primarily in the heart muscle, beta-2 receptors are located in the bronchial and peripheral vascular smooth muscles, and beta-3 receptors appear in the adipose tissue of the heart. Cardio-selective beta-blockers (e.g., metoprolol succinate, metoprolol tartrate, atenolol, betaxolol, and acebutolol) inhibit only beta-1 receptors, causing fewer bronchospasms. By inhibiting the catecholamines binding to the beta receptors, the beta-blockers have a negative inotropic effect, which results in a decrease in the heart rate, which helps to reduce oxygen consumption.

Beta-blockers are preferred in patients with heart failure, myocardial infarction and tachycardia.

They are associated with decreased cardiovascular morbidity and mortality when used in younger patients but are less protective in patients older than 65.

Side effects: bradycardia, constipation, depression, fatigue, and sexual dysfunction. Additionally, they are associated with bronchospasm and worsening symptoms of peripheral vascular disease. They can cause a flare-up of Raynaud syndrome.

Combination therapy

One study showed the reduction in blood pressure when drugs from two different classes are combined is approximately five times greater than when the dose of one drug dose doubles.

The ACE inhibitor-ARB combination is not recommended; it showed a higher incidence of side effects with no added benefits.

The ACCOMPLISH trial demonstrated that the ACEi/CCB combination (specifically benazepril/amlodipine) was more effective than an ACEi/diuretic combination in reducing cardiovascular events in high-risk hypertensive patient.

Table 8: Selected Drug Classes and Corresponding Selected Indications to Consider for Use

Drug Class	Indications for Consideration	Contraindications
Thiazide or thiazide-like diuretics	ISH, elderly, stroke	Anuria
ACE inhibitor (or ARB if ACE intolerant)	Diabetes, CKD, CAD, heart failure, stroke	Pregnancy, renovascular disease
Calcium channel blocker (dihydropyridine)	ISH, elderly	--
Calcium channel blocker (non-dihydropyridines)	Angina	Heart block, heart failure
Beta blocker	Heart failure, recent MI, angina	Heart block, asthma for nonselective BBs
Aldosterone antagonists	Heart failure	Hyperkalemia, renal insufficiency

Abbreviations: ACE, angiotensin converting enzyme; ARB, angiotensin receptor blocker; BBs, beta blockers; CAD, coronary artery disease; CKD, chronic kidney disease; ISH, isolated systolic hypertension; MI, myocardial infarction.

Reference - J Hum Hypertens 2004 Mar;18(3):139, FDA DailyMed 2024 Jun 26 [hydrochlorothiazide], FDA DailyMed 2021 Oct 1 [eplerenone].

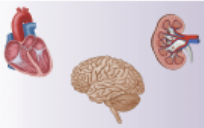
5. Evaluate response

It is important to evaluate the BP response after treatment initiation (3 months) as well as during short-term and long-term follow-up in patients with hypertension in order to monitor the effectiveness of the treatment and make any necessary adjustments.

To aim first for BP control with SBP and DBP below 140 and 80 mmHg in most patients in the general hypertensive population and subsequently for optimal BP control achieving the individual BP targets is the important goal that should be evaluated.

However, the evaluation of possible side effects (tolerability) and safety parameters, e.g. eGFR and serum potassium levels, in response to treatment and changes in the risk factor profile, HMOD or co-morbidities is also important.

Evaluate Response

	Initiation (3 months)	Short-term FU (3 months - 1 year)	Long-term FU (>1 year)
Objective	Aim for BP control	Establish optimal BP control	Maintain optimal BP control
	1-2 visits (4-6 weeks)	1-2 visits depending on CV risk (4-6 weeks) More frequently in patients with high-risk and difficult to control BP	Low-risk:1 visit per year High risk and difficult to control BP: more frequent visits (2-3/year)
	Office BP and Home BP	Office BP and Home BP (before visits); verify consistency of BP control; consider seasonal variability ABPM in apparent treatment resistance hypertension; consider to refer to a specialist	
	Selected lab tests to address safety of drug therapy or risk factors	Depending on baseline profile and condition periodic re-assessment of parameters with impact on drug safety and selection, e.g. eGFR, potassium or important risk factors, e.g. glucose, HbA1c, LDL-cholesterol	
	Re-Assess modifiable risk factors and HMOD (Table 2 and Table 3)	In patients with pre-existing HMOD verify BP-induced changes (depending on sensitivity to change), e.g. eGFR, albuminuria, pulse wave velocity or left ventricular hypertrophy.	In patients without pre-existing HMOD re-assess in longer intervals, e.g. every 3 years In patients with pre-existing HMOD more frequent re-assessments of BP-induced changes
	Verify and adapt lifestyle interventions and recommended drug therapy prescribing patterns	Support implementation of lifestyle interventions. Consider adjustment of medications depending on BP control, tolerability and change in co-morbidities, avoid inertia. Consider deprescribing in symptomatic very old and frail patients with low BP	
	Verify initiation and discuss adherence	Monitor adherence/persistence to drug therapy: assess barriers, e.g. changes in co-morbidiies, side-effects, polypharmacy including OTC use	
	Support individual needs and shared decision making	Organize and implement patient support: consider use of team-based care, telehealth, virtual visits, self-monitoring and patient empowerment	Maintain patient support

Complications

- **Hypertension is a risk factor for:**
    - **Coronary artery disease (CAD)**
    - **Heart failure**
    - **Chronic kidney disease**
    - **Stroke**
    - **Intracerebral hemorrhage**
    - **Transient ischemic attack (TIA)**
    - **Peripheral artery disease (PAD)**
    - **Aortic regurgitation**
    - **Atrial flutter**
    - **Mild cognitive impairment (MCI)**

HYPERTENSIVE EMERGENCY VS. URGENCY

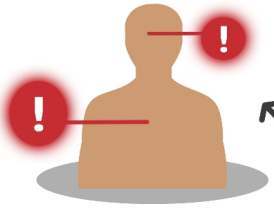
THE INTERN AT WORK

HYPERTENSIVE EMERGENCY

"Acute significant elevation of blood pressure, typically systolic BP greater than or equal to 180mmHg and/or diastolic BP greater than or equal to 120mmHg, with signs of new end-organ damage or dysfunction".

01 Symptoms

Headache, altered mental status, blurred vision, chest pain, dyspnea



*Dependent on symptoms

02 Initial Investigations*

Check the blood pressure in both arms to assess for aortic dissection, CBC, electrolytes, creatinine, urinalysis, serial troponin, blood gas, urine drug screen (if intoxication suspected), ECG, chest X-ray, non-contrast CT head

03 Long-term Investigations

Pheochromocytoma - urinary metanephrines; hyperthyroidism -TSH; hyperaldosteronism - aldosterone renin ratio; scleroderma renal crisis - autoimmune workup, including anti-Scl-70 antibody

*Start by taking a thorough history assessing for symptoms of pheochromocytoma, hyperthyroidism, hyperaldosteronism, and scleroderma renal crisis. History and physical examination trump investigations!

Management

(note: treatment of hypertensive emergency should be guided by etiology and will require admission to an ICU or step-down ICU setting)

- Target decrease in MAP by 10-20% within the first hour, followed by roughly 25% decrease in MAP in next 24-48h
  - Exceptions are aortic dissection and acute stroke, pre-eclampsia, and scleroderma renal crisis
- Type A or symptomatic Type B aortic dissections are surgical emergencies
- Asymptomatic Type B aortic dissections should be treated medically
- IV anti-hypertensives
  - Sodium nitroprusside (avoid in stroke, acute MI)
  - Nitroglycerin (avoid in inferior MI, contraindicated if using phosphodiesterase inhibitors)
  - Hydralazine (may cause rebound tachycardia, drug-induced lupus)
  - Labetalol (avoid in acute systolic heart failure)

HYPERTENSIVE URGENCY

"Acute significant elevation of blood pressure, typically systolic BP greater than or equal to 180mmHg and/or diastolic BP greater than or equal to 120mmHg, WITHOUT signs of new end-organ damage or dysfunction".

Management

- Common cause is discontinuation/inadequate dosing of anti-hypertensive medications
- Typically managed by restarting, modifying, or titrating pre-existing oral anti-hypertensive therapy
- Blood pressure should be reduced gradually over 1-2 days

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Infographic by: Hailey Pineda

References

ESH 2024 guidelines

https://www.ncbi.nlm.nih.gov/books/NBK554579/

Dynamed.com