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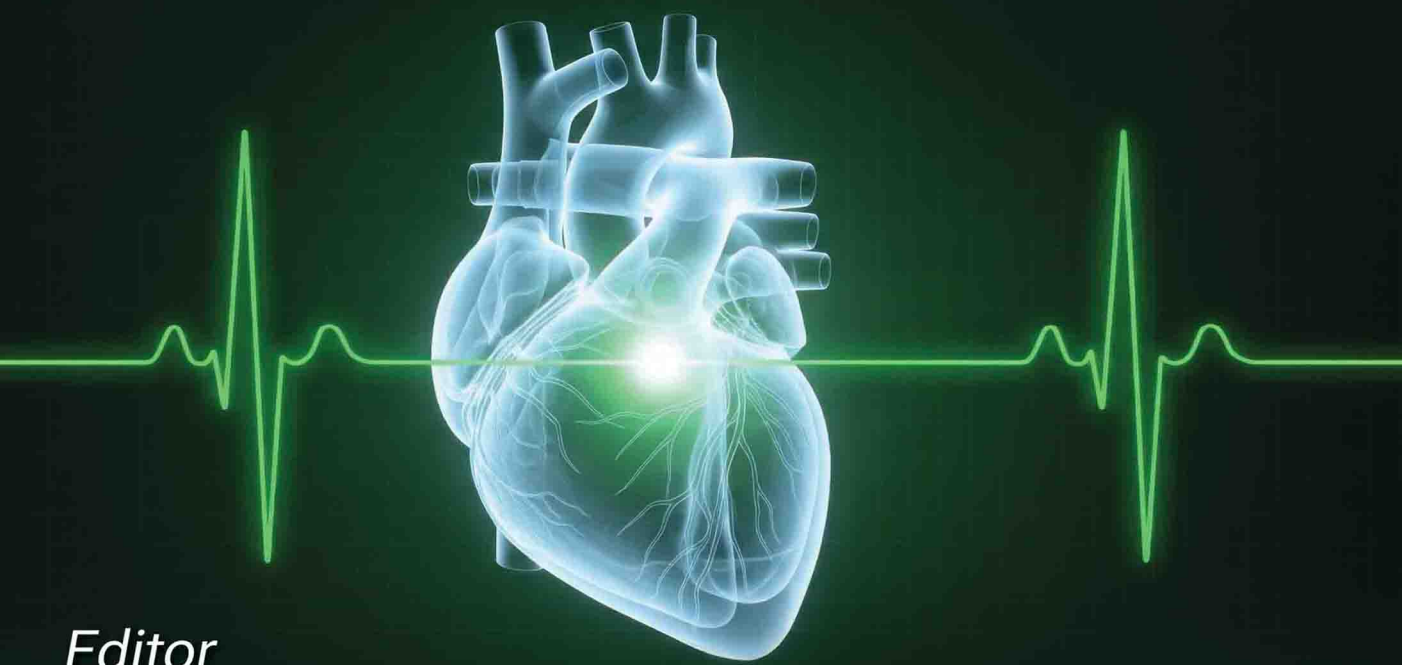

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CHAPTER 8

Hypertension: Diagnosis and Current Management

Suman Bhandari, Bhavana Mastebhakti

INTRODUCTION

Hypertension: The European Society of Cardiology (ESC) defines hypertension as systolic blood pressure (SBP) in office of ≥140 mm Hg or diastolic blood pressure (DBP) of ≥90 mm Hg.[1]

Elevated blood pressure: This is a new blood pressure (BP) category that has been introduced in the ESC 2024 guidelines and is defined as an SBP or DBP of 120–139 mm Hg or DBP of 70–89 mm Hg in office.[1]

EPIDEMIOLOGY

Hypertension is the most prevalent cardiovascular (CV) disease in the world. The World Health Organization (WHO) estimates that nearly 1.3 billion adults aged 30–79 years have hypertension, with two-third of them living in middle- to low-income countries and only one in five of these have their BP under control.[2] In <50 years age group, there is male predominance for hypertension, whereas a steep increase of SBP is seen in women from their third decade especially after menopause), thus making the prevalence of hypertension greater in women older than 65 years of age.[3] SBP increases gradually with age while DBP increases until the age of 50–60 years, followed by a short period of plateau and a subsequent mild decrease, causing the pulse pressure to increase with age.[4]

TYPES OF HYPERTENSION

Essential/primary hypertension—where the exact cause is unknown and is seen in 90% of the individuals.[5]

Secondary hypertension—where an identifiable cause exists and is seen in up to 10% of the individuals.[5]

PATHOPHYSIOLOGY

The pathophysiology of hypertension includes a complex interplay between various mechanisms including renal, neural, hormonal, and vascular mechanisms; and various factors including genetic, behavioral, environmental, socioeconomic, and psychosocial factors[1] **(Fig. 1)**.

- *Renal mechanisms:*
  - Salt sensitivity
  - Pressure-natriuresis
  - Renin–angiotensin-aldosterone system (RAAS)
  - Renal ischemia
- *Neural mechanisms:*
  - Autonomic nervous system
  - Baroreceptor reflex
- *Vascular mechanisms:*
  - Endothelial dysfunction
  - Small artery remodeling
  - Large artery stiffness
- *Hormonal mechanisms:*
  - RAAS
  - Endothelin system
  - Sex hormones
- *Behavioral factors:*
  - Exercise
  - Sleep
  - Dietary habits
  - Sodium and potassium intake
  - Obesity

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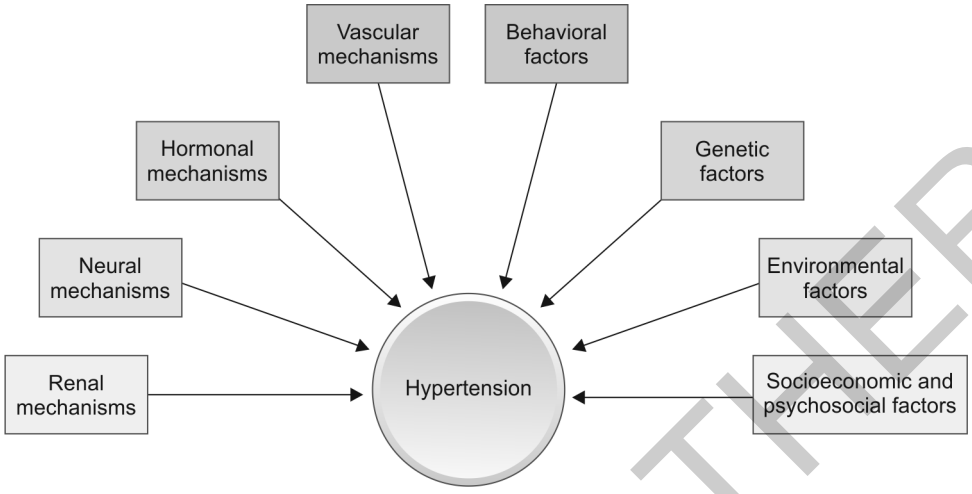

Fig. 1: Pathophysiological factors and mechanisms leading to hypertension.

- Alcohol consumption
- Drug abuse

- *Genetic factors:*
  - Gender
  - Single nucleotide polymorphisms (SNPs) determining blood pressure
  - Single gene mutations of hypertension
  - Epigenetics
- *Environmental factors:*
  - Geopolitics
  - Pollution
  - Climatic change
- *Socioeconomic and psychosocial factors:*
  - Psychosocial Stress
  - Poor socioeconomic status
  - Social deprivation
  - Inaccessibility to healthcare
  - Gender-based discrimination

MEASUREMENT OF BLOOD PRESSURE

Office BP/clinic BP measurement: It is measured manually or by using an automated device, in office/clinic, either in an unattended or an unattended fashion.[1] **Table 1** illustrates the practical recommendations for measuring office BP.

Home blood pressure measurement (HBPM): BP is measured in an out-of-office setting wherein the patient usually measures their own BP at home using a validated BP device.[1] **Box 1** illustrates the practical recommendations for measuring home BP.

Ambulatory blood pressure measurement (ABPM): It is an out-of-office BP measurement, in which BP is measured usually over a 24-hour period, at set intervals using a fully automated oscillometric device.[1] **Box 2** illustrates the practical recommendations for measuring ABPM.

BLOOD PRESSURE CATEGORIES

The cutoff values of office, home, and ambulatory blood pressure thresholds for BP are summarized in **Tables 2A to C**.[1]

White-coat hypertension: An office BP of ≥140/90 mm Hg but a home BP of <135/85 mm Hg or an ambulatory BP of <130/80 mm Hg.[1]

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TABLE 1: Practical recommendations for measuring office blood pressure (BP).

Patient preparation	• Rest for 5 minutes • Avoid exercise/coffee/tobacco within half an hour of measurement • Urinary bladder should be voided • Legs unfolded, back and arms supported • The cuff should not be tied above the clothing
BP measurement technique	• Use a validated device • Auscultatory or oscillometric techniques can be used
BP cuff selection and positioning	• *Length*: 75–100% and width 35–50% of the arm circumference • The cuff should be positioned on the patient's arm at the level of the heart with the lower edge of the cuff a few centimeters above the antecubital fossa while the stethoscope not being placed under the cuff
BP measurement by manual auscultation	• Measure BP at least three times, with each reading taken 1–2 minutes apart, at the first visit and average the last two readings • Further measurements need to be taken if the readings differ by >10 mm Hg
BP measurement using AOBP monitor	• AOBP monitors take three or six readings at 1-min intervals and provide an average • AOBP correlates more closely with mean ABPM than with the manual auscultatory technique
Inter-arm BP difference	• Measure BP in both arms at the first visit to detect inter-arm BP difference • An inter-arm BP difference of >10 mm Hg may need further evaluation
Postural/orthostatic hypotension	• Patients should be assessed for orthostatic hypotension at the first visit • A drop in SBP/DBP by ≥20/10 mm Hg within 3 minutes after standing, suggests classical orthostatic hypotension
Pulse assessment	Record heart rate and exclude arrhythmia by pulse palpation

(ABPM: ambulatory blood pressure monitoring; AOBP: automated blood pressure; BP: blood pressure; DBP: diastolic blood pressure; SBP: systolic blood pressure)

BOX 1: Practical recommendations for measuring home blood pressure measurement (HBPM).

- Use a validated blood pressure device
- Rest for 5 minutes
- Arm and back should be supported
- Obtain two readings on each occasion, 1–2 minutes apart
- Obtain readings twice a day (morning and evening) for at least 3 and ideally 7 days
- Record and average all readings and present results to clinician
- Devices older than 4 years may be inaccurate and if inaccurate should be replaced

BOX 2: Practical recommendations for measuring ambulatory blood pressure measurement (ABPM).

- Use a validated BP device
- BP is measured at 15–30 minutes intervals during the day and 30–60 minutes using night
- A minimum of 70% usable BP recordings is required to give a conclusive report
- The timing of patient's activities, intake of medications and sleep time should be enlisted

Masked hypertension: An office BP of <140/90 mm Hg but home BP of ≥135/85 mm Hg or an ambulatory BP of ≥130/80 mm Hg.[1]

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TABLE 2A: Blood pressure categories (ESC 2024).

(mm Hg)	*Office BP*	*Home BP*	*Daytime ABPM*	*24 h ABPM*	*Night-time ABPM*
Non-elevated BP	<120/70	<120/70	<120/70	<115/65	<110/60
Elevated BP	120/70–<140/90	120/70–<135/85	120/70–<135/85	115/65–<130/80	110/60–<120/70
Hypertension	≥140/90	≥135/85	≥135/85	≥130/80	≥120/70

(ABPM: ambulatory blood pressure monitoring; BP: blood pressure)

TABLE 2B: Blood pressure categories (ISH 2020).

(mm Hg)	*Office BP*	*Home BP*	*Daytime ABPM*	*24 h ABPM*	*Night-time ABPM*
Hypertension	≥140/90	≥135/85	≥135/85	≥130/80	≥120/70

(ABPM: ambulatory blood pressure monitoring; BP: blood pressure)

TABLE 2C: Classification of hypertension based on office BP measurement (ISH 2020).

Category	*Systolic (mm Hg)*		*Diastolic (mm Hg)*
Normal BP	<130	and	<85
High normal BP	130–139	and/or	85–89
Grade 1 hypertension	140–159	and/or	90–99
Grade 2 hypertension	≥160	and/or	≥100

HISTORY AND PHYSICAL EXAMINATION

Patients with hypertension or elevated BP have to be evaluated for risk factors of hypertension **(Table 3)**, hypertension-mediated organ damage (HMOD) **(Table 3)**, secondary causes of hypertension **(Table 4)**, and drugs causing hypertension **(Table 5)**.[1]

SCREENING AND CONFIRMATION OF HYPERTENSION

- A single office BP is not specific for confirmation of hypertension.[6] Home BP/ABPM is measured to confirm the diagnosis of hypertension.
- *Non-elevated BP and <40 years:* Opportunistic BP screening every 3 years
- *Non-elevated BP and ≥40 years:* Opportunistic BP screening every year
- *Elevated BP:* Follow **Table 3**
- *Hypertension with BP 140–159/90–99 mm Hg:* Confirm preferably with home or ambulatory measurements.
- *Hypertension with BP 160–179/100–109 mm Hg:* Confirm promptly (within a month) with home or ambulatory measurements.
- *Hypertension with BP ≥180/110 mm Hg:* Evaluate for hypertensive emergency.

CONSEQUENCES OF HYPERTENSION

Cardiovascular Effects of Elevated Blood Pressure

- High BP and CV and renal events share a direct relationship above an office BP of >115 mm Hg/75 mm Hg. The estimated 10-year risk for

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TABLE 3: Risk factors and symptoms suggestive of hypertension.

Risk factors	*Symptoms suggestive of HMOD*
Family/personal history of hypertension, CVD, or renal diseases	*Central nervous system (CNS):* Syncope, vision disturbances, TIA and CVA, focal neurological deficit, carotid stenosis, cognitive impairment, and dementia
Smoking/vaping history	*Cardiovascular system (CVS):* Chest pain, dyspnea, shortness of breath, edema, MI, coronary revascularization, history of palpitations, arrhythmias (AF), and heart failure
Dietary history	*Renovascular:* Polyuria, nocturia, hematuria, UTI, CKD, and nephropathy
Alcohol consumption	*Peripheral arteries:* Cold extremities, intermittent claudication, leg ulcers, and peripheral revascularization
Physical activity/sedentary lifestyle	
History of erectile dysfunction	
Migraine with aura	
Autoimmune inflammatory diseases	
HIV	
Sleep history and snoring	
Psychosocial factors—stress, depression, social deprivation, low SES, gender-based violence	
Obstetric history—gestational hypertension, pre-eclampsia, gestational diabetes, miscarriages/stillbirth, and pre-term labor	
Early menopause, PCOD	

(AF: atrial fibrillation; CKD: chronic kidney disease; CVD: cardiovascular disease; HIV: human immunodeficiency; MI: myocardial infarction; PCOD: polycystic kidney disease; SES: socioeconomic status; TIA: transient ischemic attack; UTI: urinary tract infection)

TABLE 4: Symptoms suggestive of secondary causes of hypertension.

All causes	• BP >160/100 mm Hg in young adults <40 years • BP >180/110 mm Hg irrespective of age • Sudden development of hypertension • Rapidly worsening BP • Resistant hypertension • Hypertensive emergency
Primary aldosteronism	• Spontaneous or diuretic provoked hypokalemia • Episodes of muscle weakness or tetany • Adrenal incidentaloma on abdominal imaging • History of AF not due to attributable cause • HMOD or CVD and renal disease disproportionate for the observed BP • Family history of early onset (<40 years) hypertension/CVA • First degree relatives with primary aldosteronism

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Obstructive sleep apnea syndrome	• Witnessed apnea • Heavy snoring • Disturbed sleep • Recurrent awakening from sleep with choking, gasping, and sweating • Daytime sleepiness, fatigue, and impaired concentration • Non-dipping or reverse dipping BP pattern at 24-hour BP monitoring
Renovascular hypertension	• *Fibromuscular dysplasia:* Hypertension in females <40 years, migraine, pulsatile tinnitus, history of arterial dissections/aneurysms • *Atherosclerosis:* Multiple CVD risk factors • *Both:* Reduced eGFR/presence of albuminuria/markedly elevated renin concentration, acute worsening of renal function after administration of ACEI or ARBs, unexplained small kidneys, or size discrepancy between kidneys of >1.5 cm • Sudden unexplained pulmonary edema
Renoparenchymal hypertension	• Known CKD • History of UTI • Microalbuminuria/reduced eGFR
Phaeochromocytoma	• Repetitive effects of sweating, pallor, headache, anxiety, and palpitations
Paraganglioma	• Neurofibromata • Features of MEN-2 • Von Hippel-Lindau features
Others	• Hypo/hyperthyroidism • Hyperparathyroidism • Cushing's disease • Substance abuse

(ACEIs: angiotensin-converting enzyme inhibitors; AF: atrial fibrillation; ARBs: angiotensin receptor blockers; BP: blood pressure; CKD: chronic kidney disease; CVA: cerebrovascular accident; CVD: cardiovascular disease; eGFR: estimated glomerular filtration rate; HMOD: hypertension mediated organ damage; MEN-2: multiple endocrine neoplasia, type 2; UTI: urinary tract infection)

TABLE 5: Drugs and substances which may increase blood pressure.

Contraceptive drugs	OCPs and vaginal ring
Sympathomimetics	• Phenylpropanolamine and sibutramine • Phenylephrine hydrochloride and naphazoline hydrochloride • Methylphenidate • Amphetamines, cocaine, ecstasy • Ephedra and ma-Huang
NSAIDs	• Indomethacin • Naproxen • Piroxicam • Ibuprofen
Paracetamol (acetaminophen)	Chronic use at high doses (4 g/day)
Corticosteroids	Increase BP in a dose-dependent manner

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Immunosuppressive medications	Cyclosporin A and tacrolimus
Anticancer drugs	• Vascular endothelial growth factors • Tyrosine kinase inhibitors
Antidepressant drugs	• Venlafaxine and • Monoamine oxidase inhibitors
Psychiatric drugs	• Clozapine • Carbamazepine • Lithium
Others	• Liquorice • Anabolic steroids (testosterone, growth hormone) • Highly active anti-retroviral therapy • Caffeinated drinks

cardiovascular disease (CVD) events is ≥10% in hypertensive individuals.[7,8]

- Blood pressure-lowering treatment is prescribed for patients with elevated BP as the efficacy of BP lowering therapy extends even to a BP of 120 mm Hg/70 mm Hg[8,9] **(Flowchart 1)**.

Hypertension-mediated Organ Damage

Hypertension complicated with the presence of subclinical manifestations in various organs that indicate high risk for subsequent clinical events, is known as hypertension-mediated organ damage (HMOD).[1] **Figure 2** summarizes the consequences of HMOD.

INVESTIGATIONS

Routine tests—tests to detect increased CVD risk and associated comorbidities such as dyslipidemia and diabetes **(Table 6A)**.

Optional tests—are considered with the main rationale to improve CVD risk stratification and HMOD **(Table 6B)**.

RESISTANT HYPERTENSION

Resistant hypertension is defined as a BP of >140/90 mm Hg despite maximally tolerated doses of first-line anti-hypertensives including diuretic (thiazide or thiazide-like), a RAAS blocker, and a calcium channel blocker,[1] and after excluding pseudo-resistance. The causes of resistant and pseudo-resistant hypertension are summarized in **Table 7**.

MANAGEMENT OF HYPERTENSION

Non-pharmacological Treatment

Table 8 summarizes the non-pharmacological interventions for hypertension.

Pharmacological Treatment

Drugs lowering blood pressure: First-line BP lowering drugs and other drugs with BP lowering effects are enlisted in **Table 9A and B**.

Algorithm to initiate and continue pharmacological therapy for lowering BP is mentioned in **Flowchart 2 and Table 10**.

COMBINATION THERAPIES

Upfront low-dose combination therapy (LDC) in the form of single-pill is the latest treatment recommendation in patients with hypertension. LDCs have an advantages of fewer side effects, rapid and long-lasting BP control and better compliance.[10-12] LDC with appropriate dosing leads to simultaneous effects on multiple

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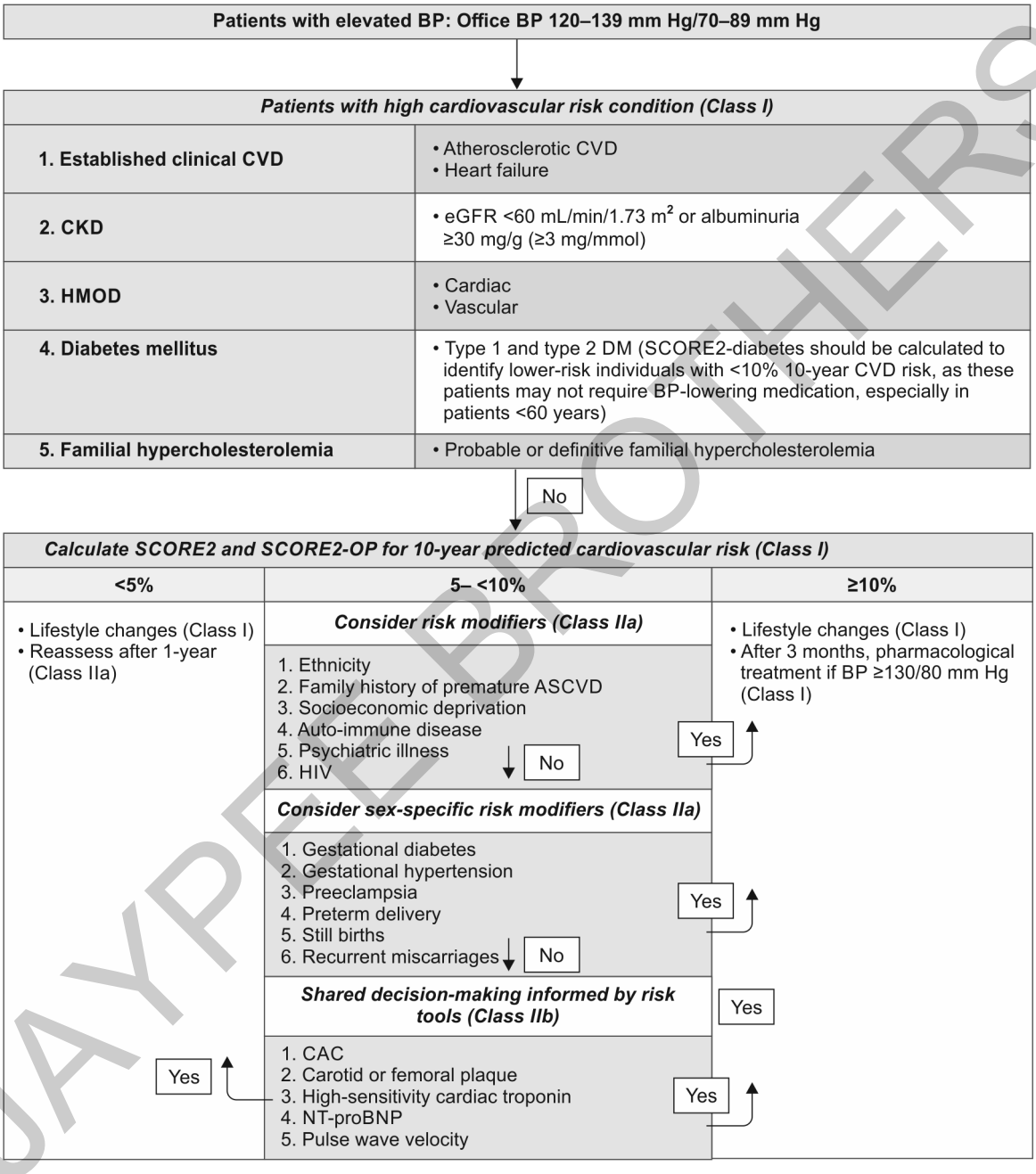
Flowchart 1: Cardiovascular disease risk stratification.



[ASCVD: atherosclerotic cardiovascular risk; BP: blood pressure; CKD: chronic kidney disease; CVD: cardiovascular disease; DM: diabetes mellitus; HIV: human immunodeficiency virus; HMOD: hypertension mediated organ damage; NT-proBNP: N-terminal pro-brain natriuretic peptide; SCORE2: systematic coronary risk evaluation 2 (40–69 years); SCORE2-OP: systematic coronary risk evaluation 2-older persons (≥70 years)]

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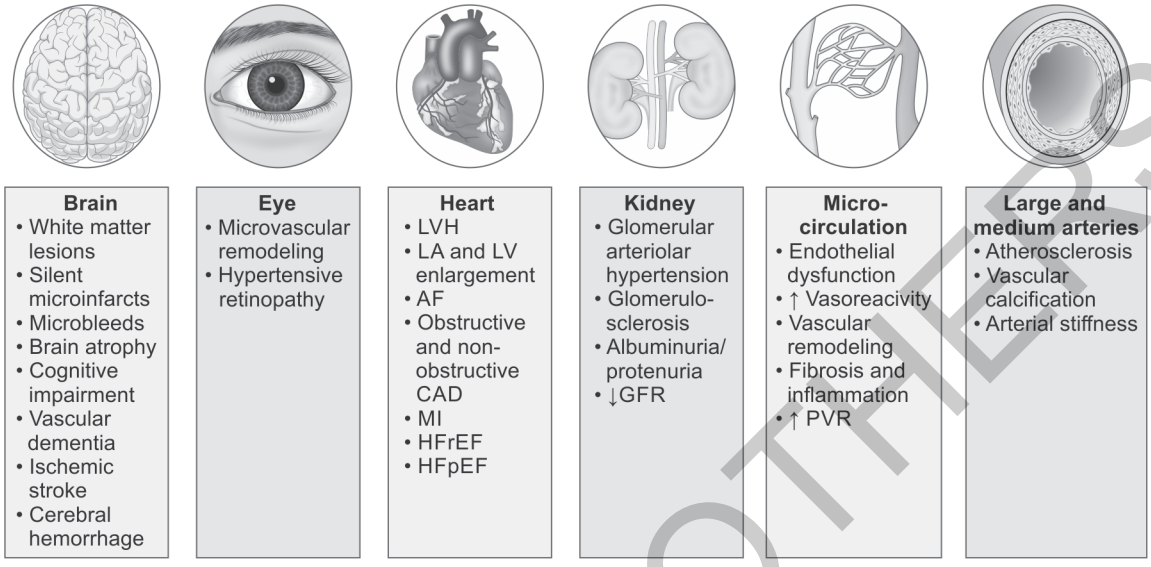

Fig. 2: Hypertension-mediated organ damage. (AF: atrial fibrillation; CAD: coronary artery disease; GFR: glomerular filtration rate; HFpEF: heart failure with preserved ejection fraction; HFrEF: heart failure with reduced ejection fraction; LA: left atrium; LV: left ventricle; LVH: left ventricular hypertrophy; MI: myocardial infarction; PVR: peripheral vascular resistance)

TABLE 6A: Routine tests for a patient with hypertension.

Routine test	*Clinical utility*
FBS, PPBS, and HbA1c	CVD risk assessment
Lipid profile	CVD risk assessment
Serum electrolytes	Screening secondary hypertension (endocrine causes)
TSH	Screening secondary hypertension (hyperthyroidism)
Hemoglobin/hematocrit	Screening secondary hypertension (polycythemia)
Blood creatinine and eGFR; urinalysis and urinary albumin-to-creatinine ratio	• CVD risk assessment and HMOD • Guiding antihypertensive drugs • Screening secondary hypertension (renal causes)
12-lead ECG	HMOD assessment (left atrial enlargement, left ventricular hypertrophy), rhythm abnormalities (AF), and previous acute myocardial infarction

(AF: atrial fibrillation; CVD: cardiovascular disease; ECG: electrocardiogram; eGFR: estimated glomerular filtration rate; HbA1c: glycated hemoglobin; HMOD: hypertension-mediated organ damage; PPBS: post-prandial blood sugar; TSH: thyroid-stimulating hormone)

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TABLE 6B: Optional tests for a patient with hypertension.

Optional test	*Clinical relevance*
Echocardiography	• HMOD (hypertensive heart disease) • Established CVD
CAC	HMOD (atherosclerotic plaque)
Large artery stiffness (carotid-femoral or brachial-ankle PWV)	HMOD (arterial stiffness)
High-sensitivity cardiac troponin and/or NT-proBNP	HMOD
Ankle-brachial index	Established CVD (lower-extremity arterial disease)
Abdominal ultrasound	Established CVD (abdominal aneurysm)
Fundoscopy	• HMOD (hypertensive retinopathy) • Diagnosing hypertensive emergency/malignant hypertension (hemorrhages and exudates, papilledema)

(CAC: coronary artery calcium; CT: computed tomography; CVD: cardiovascular disease; HMOD: hypertension-mediated organ damage; NT-proBNP: N-terminal pro-brain natriuretic peptide; PWV: pulse wave velocity)

TABLE 7: Causes of resistant and pseudo-resistant hypertension.

Causes of pseudo-resistant hypertension	*Causes of resistant hypertension*
Poor compliance	Behavioral factors
White-coat hypertension	Overweight/obesity
Poor blood pressure (BP) measurement method	Physical inactivity
Marked brachial artery calcification (Osler phenomenon)	Excess daily dietary sodium
Clinical inertia (inadequate doses, inappropriate combinations of BP-lowering drugs)	Excess habitual alcohol consumption
Munchausen syndrome (rare)	

TABLE 8: Non-pharmacological measures for hypertension management.

Exercise	Aerobic exercise training at least 150 minutes/week of moderate-intensity or at least 75 minutes/week vigorous intensity, brisk walking, jogging, cycling, and swimming
Dietary sodium intake	Dietary sodium ≤2 g/day Dietary salt ~5 g/day or ~1 teaspoon of salt/day
Dietary potassium intake	Dietary potassium ~3.5 g/day and <2.4 g/day in advanced CKD
Diet	DASH diet Mediterranean diet
Weight reduction	Weight loss of 5 kg is associated with an average systolic and diastolic BP reduction of 4.4 and 3.6 mm Hg, respectively
Alcohol and soft drinks	≤100 g/week of pure alcohol Sugar-sweetened beverages up to 10% of energy intake
Smoking	Cessation of smoking

(CKD: chronic kidney disease; DASH: dietary approach to stop hypertension)

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TABLE 9A: First line BP-lowering drugs.

ACEIs	• Captopril • Enalapril • Lisinopril • Perindopril • Ramipril
ARBs	• Telmisartan • Olmesartan • Valsartan • Candesartan • Irbesartan • Losartan • Azilsartan
CCBs (DHP)	• Amlodipine • Felodipine • Nifedipine • Manidipine • Lercanidipine
Diuretics	*Thiazide and thiazide-like diuretics:* • Hydrochlorothiazide • Chlorthalidone • Indapamide *Potassium-sparing diuretics:* • Spironolactone • Epleronone • Amiloride
Beta-blockers	• Atenolol • Bisoprolol • Carvedilol • Labetalol • Metoprolol succinate • Metoprolol tartrate • Nebivolol • Propranolol

(ACE: angiotensin-converting enzyme; ARB: angiotensin receptor blocker; CCBs: calcium channel blockers; DHPs: dihydropyridine)

TABLE 9B: Other drugs lowering BP.

Alpha-blockers	• Doxazocin • Prazosin • Terazosin
Diuretics (loop diuretics)	• Bumetanide • Torsemide • Furosemide
CCBs (NDHPs)	• Diltiazem • Verapamil
Vasodilators	• Hydralazine • Minoxidil
CNS antihypertensives	• Clonidine • Methyldopa • Moxonidine

(CCBs: calcium channel blockers; CNS: central nervous system; NDHPs: non-dihydropyridines)

mechanisms, thus achieving therapeutic levels regardless of baseline blood pressure, cardiovascular risks, and comorbidities. Two to five times greater antihypertensive effect can be achieved by combining two drugs with different mechanisms of action, than that obtained by monotherapy alone.[13,14] Increasing the dose of monotherapy reduced coronary events by 29% and cerebrovascular events by 40%, while combining two antihypertensive agents with different mechanisms of action reduced coronary events by 40% and cerebrovascular events by 54%.[15]

Evidence Supporting Combination Therapies Over Monotherapy

Randomized controlled studies show that ≥2 antihypertensive drugs are needed to reach liberal BP targets in the majority of hypertensive patients.[16]

STRATHE (Strategies of Treatment in Hypertension: Evaluation)[17] was a French randomized controlled trial which has shown that a LDC (62%) has shown a higher rate of success in achieving target BP at 3, 6, and 9 months follow-up, in previously untreated hypertensive patients compared to a sequential monotherapy regimen (49%, $p = 0.02$).

A meta-analysis of 42 trials with 11,000 participants has shown that combining drugs with different mechanisms of action is more efficient to reduce BP than doubling the dose of the same drug which has a very little additive effect.[13]

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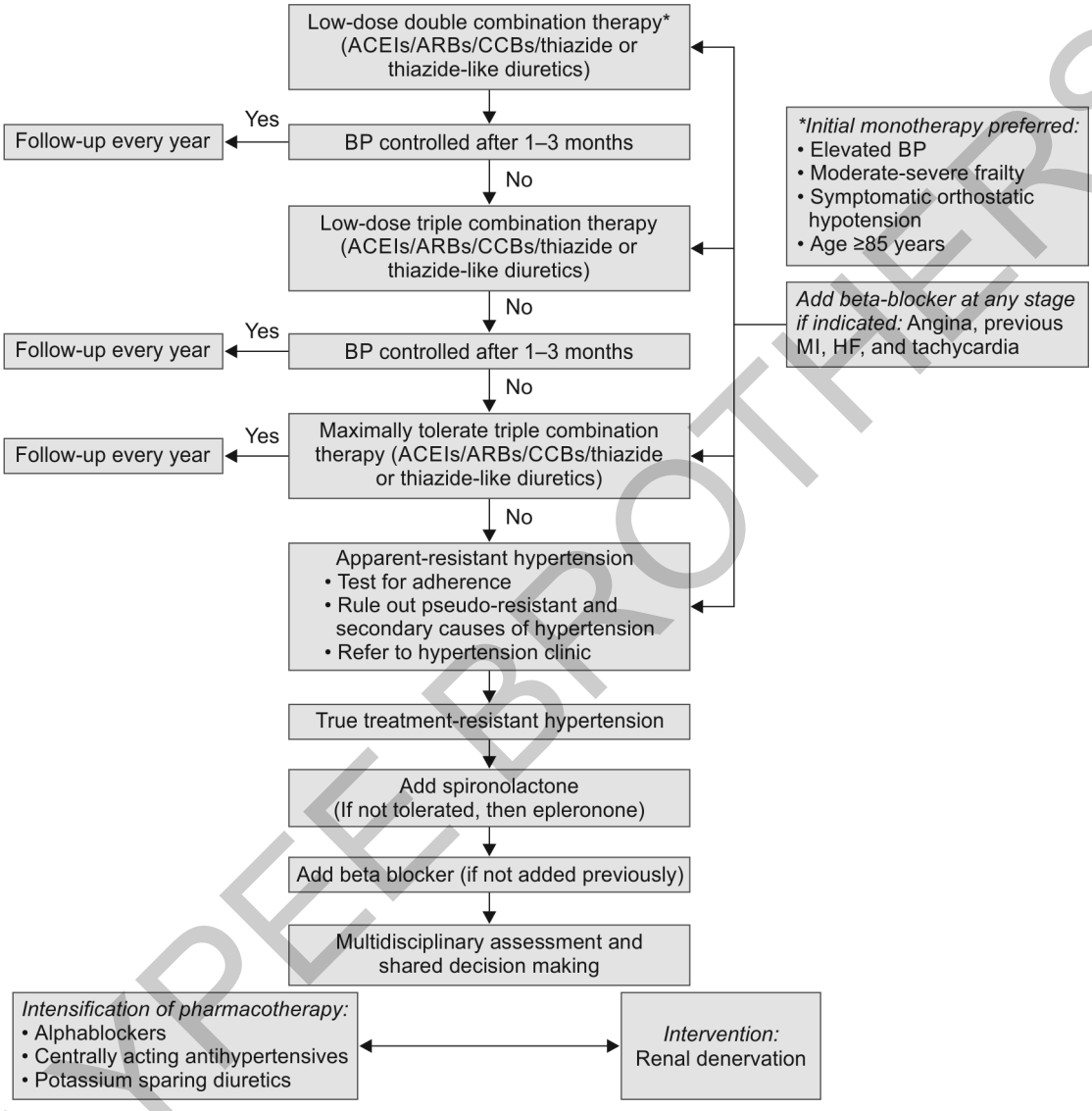
Flowchart 2: Pharmacological therapy for hypertension.



(ACE: angiotensin-converting enzyme; ARB: angiotensin receptor blocker; BP: blood pressure; CCBs: calcium channel blockers; HF: heart failure)

In a retrospective analysis including 1,700 patients, patients treated with LDC initially, achieved a significantly shorter (9.7 vs. 11.9 months, $p = 0.004$) median time for BP control as compared to those who were shifted to a combination after an initial monotherapy.[18]

BLOOD PRESSURE TARGETS

- *Target range:* 120–129/70–79 mm Hg
- *Target SBP:* As low as tolerable preferably <140 mm Hg if:
  - Target range is not tolerated
  - Orthostatic hypotension

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TABLE 10: Choice of BP-lowering drugs based on comorbidities.

Comorbidity	*Initial drug class*
• Diabetes • Metabolic syndrome	ACEI/ARB CCB
• Chronic kidney disease • Proteinuria/albuminuria	ACEI/ARB Diuretic CCB SGLT2 inhibitor
Postmyocardial infarction	Betablocker ACEI ARB MRA
Atrial fibrillation	Betablocker ACEI ARB
Heart failure	ACEI ARB MRA Betablocker SGLT2 inhibitor Diuretic

(ACEI: angiotensin-converting enzyme inhibitor; ARB: angiotensin receptor blocker; CCB: calcium channel blockers; MRA: mineralocorticoid receptor antagonist; SGLT2: sodium glucose cotransporter 2)

- Elderly especially, age ≥85 years
- Moderate-severe frailty

ACUTE AND SORT-TERM LOWERING OF BLOOD PRESSURE

Hypertensive emergency: BP of ≥180/110 mm Hg associated with symptomatic HMOD

Hypertensive urgency: BP of ≥180/110 mm Hg without clinical evidence of HMOD. These patients are managed with up titrating their oral medications.

Diagnostic work-up and management of patients presenting in hypertensive emergency is summarized in **Box 3 and Table 11**.

BOX 3: Work-up of patients presenting with hypertensive emergency.

Common tests:
- Fundoscopy
- 12-lead ECG
- Complete blood count and coagulation profile
- Kidney function tests, LDH, and haptoglobin
- UACR and urine routine microscopy
- Urine pregnancy test for women in reproductive age-group

Specific tests:
- Cardiac enzymes and NT-ProBNP
- Chest X-ray
- Echocardiography
- CT angiography of thorax and/or abdomen to rule out aortic dissection
- CT or MRI brain to rule out central nervous system involvement
- Renal ultrasound to rule out renal impairment or suspected renal artery stenosis
- Urine drug screen to rule out methamphetamine or cocaine use

(CT: computed tomography; ECG: electrocardiogram; eGFR: estimated glomerular filtration rate; LDH: lactate dehydrogenase; MRI: magnetic resonance imaging; NT-ProBNP: N terminal pro brain natriuretic peptide; UACR: urine albumin creatinine ratio)

TABLE 11: Drugs used in the treatment of hypertensive emergencies.

Drug	*Dosage*
Esmolol	0.5–1 mg/kg as i.v. bolus; 50–300 µg/kg/min as i.v. infusion
Metoprolol	2.5–5 mg i.v. bolus over 2 minutes—may be repeated every 5 minutes to a maximum dose of 15 mg
Labetalol	0.25–0.5 mg/kg i.v. bolus; 2–4 mg/min infusion until target BP is achieved, thereafter 5–20 mg/h
Fenoldopam	0.1 µg/kg/min i.v. infusion, increase every 5 min by 0.05–0.1 µg/kg/min until target BP is achieved
Clevidipine	2 mg/h i.v. infusion, increase every 2 minutes by 2 mg/h until target BP is achieved

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Drug	*Dosage*
Nicardipine	Start at 5 mg/h i.v. infusion, increase every 15–30 minutes by 2.5 mg until target BP is achieved, after which decrease to 3 mg/h
Nitroglycerin	5–200 µg/min i.v. infusion, 5 µg/min increase every 5 minutes
Nitroprusside	0.3–10 µg/kg/min i.v. infusion, increase by 0.5 µg/kg/min every 5 minutes until goal BP
Enalaprilat	0.625–1.25 mg i.v. bolus
Urapidil	12.5–25 mg i.v. bolus followed by 5–40 mg/h infusion
Clonidine	150–300 µg i.v. bolus over 5–10 minutes
Phentolamine	0.5–1 mg/kg i.v. bolus OR 50–300 µg/kg/min as i.v. infusion

CONCLUSION

Given the demographic transition and aging population, the number of people with elevated BP and hypertension is constantly increasing. Despite the growing number of hypertension guidelines, the rates of diagnosis, optimal treatment, and adequate control of BP remain suboptimal. A major factor underlying this is poor implementation of evidence-based guidelines in real-world clinical practice. Fixed dose combinations comprising of two or three antihypertensive drugs with different mechanisms of action have become Class I recommendation for treatment of patients with hypertension.

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