INDIAN SOCIETY OF HYPERTENSION

Indian Society of Hypertension (InSH) Consensus Guideline for the Management of Hypertension, 2025

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SECTION 1 & 2 - Introduction & Methodology

Dr. VV Muthusamy, Dr. R B Singh, Dr. Jalees Fatima, Dr. Narsingh Verma

The Global Burden of Disease Study 2021 estimates that elevated systolic blood pressure (SBP) was responsible for approximately 10.9 million deaths worldwide.[1] The ICMR-INDIAB 2023 study reported a hypertension prevalence of 35.5% in India, with rates of 38.7% among men and 32.6% among women. Prevalence rises steeply with age, with over 66% affected above 60 years.[2] The growing burden is attributed to lifestyle factors (unhealthy diet, tobacco and alcohol consumption, obesity, and sedentary behavior), socioeconomic differences, and increased life expectancy. NFHS-5 observed an overall hypertension prevalence of about 22.6% in the 15–49 years age group, showing an increasing trend from the previous survey (NFHS-4).[3] Its multifaceted etiology includes genetic, lifestyle, and secondary factors, while intertwined risk factors like obesity, diabetes, pregnancy-related disorders, and more, leading to a need for comprehensive management.[4,5] Undiagnosed hypertension also poses a significant challenge, particularly in rural areas, underscoring the importance of accurate measurement and awareness initiatives.[6,7] This trend underscores the urgent need for effective hypertension management strategies in India and similar contexts.

The growing burden of hypertension may be due to behavioral risk factors for cardiovascular diseases (CVD) such as unhealthy diet, physical inactivity, tobacco use, and harmful alcohol consumption.[1,2,8] Since alcohol in moderation, in particular wine, may be protective, due to the adverse effects of alcohol. Policies should be implemented, such as an increase in alcohol taxation, a decrease in availability, and marketing restrictions to reduce alcohol-related harms.[9] The thrifty genotype hypothesis suggests that it arises from a genetic adaptation during a period of food scarcity, in which "thrifty" genes become efficient at promoting energy storage, which becomes disadvantageous in a modern environment with increased food availability. The mechanism is that "thrifty" metabolism prioritizes keeping glucose for the brain and liver and converts excess glucose to fat for later use, leading to fatty liver, central obesity, insulin resistance, and metabolic syndrome, in which hypertension is a major component.[10]

The 75/25 Initiative, launched by India's Ministry of Health and Family Welfare on World Hypertension Day in May 2023, the "75/25" initiative targets providing standardized care for 75 million people with hypertension and diabetes by December 2025. The program emphasizes universal screening of individuals aged 30 years and above at the primary care level, including Ayushman Arogya Mandirs and other health centers. As of early 2025, the initiative has reached over 42 million people with hypertension and more than 25 million with diabetes, achieving close to 90% of the ambitious care coverage target.[11]

With a mission of taking the forefront in the campaign to control elevated blood pressure and its associated complications across the Indian subcontinent, the Indian Society of Hypertension (InSH) took the responsibility of creating a clinical guideline to better adapt the knowledge, best practices, and recommendations to the Indian context. The silent yet pervasive issues of high prevalence with substantial undiagnosed cases of hypertension are compounded by limited patient awareness of hypertension's often symptomless nature. The country observes an alarming rise in mean SBP and hypertension rates, particularly in rural areas, accompanied by geographical disparities favoring the more developed regions. Premature hypertension onset among the young and increasing mortality from hypertension-related conditions are sources of major concern. Patient awareness, treatment compliance, and control of hypertension remain suboptimal, with insufficient focus on strategies to minimize hypertension-mediated organ damage. The unequal allocation of resources at the country and state levels further compounds the challenge. India's distinct socio-economic, geographical, and cultural diversity requires tailored strategies to avert the impending non-communicable diseases (NCD) pandemic, with hypertension and obesity at the forefront, particularly among the youth.

The 2025 InSH guidelines aim to summarize the best available evidence for all aspects of hypertension management for people living in India. The guidelines were developed by a Special Committee of 63 experts from India, representing the areas of internal medicine, cardiology, nephrology, endocrinology, pediatrics, obstetrics and gynecology, general medicine, geriatrics, pharmacology, and epidemiology. The core committee comprised 5 clinical experts, 2 advisors, and section-specific committees of 3-6 experts each, totaling 14 sections. Each topic was assigned to a group of members responsible for reviewing and summarizing the available evidence within that topic. All recommendations were first raised by the respective section-level group and subsequently reviewed by members of other sections. All panel members reviewed the recommendations, and a Delphi Consensus was conducted to finalize the recommendations across all sections.

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SECTION 3 - Hypertension Definition

Dr. S N Narasingan, Dr. Sanjay Agarwal, Dr. Anubha Srivastava

Recommendations

1	Classification • Normal: <120 (systolic) and <80 (diastolic) mmHg • Raised Blood pressure: 120-129 (systolic) and <80 (diastolic) mmHg • Stage 1 Hypertension: 130-139 (systolic) and/or 80-89 (diastolic) mmHg • Stage 2 Hypertension: ≥140 (systolic) and/or ≥90 (diastolic) mmHg
2	Persons with raised blood pressure or Stage 1 in the first office visit should be advised 7 days out of Office Blood Pressure Monitoring OBPM (HBPM or ABPM) and rechecked at the office after 1 week, and then advised lifestyle modifications for at least 3 months and followed up after 3 months.
3	Patients with raised blood pressure category should be advised to monitor their blood pressure at least twice daily with 2 readings every time and noting the average of these two values, while continuing the lifestyle modifications. Ambulatory blood pressure monitoring (ABPM) may be advised once a month or more frequently, depending on available resources. If blood pressure increases to Stage 1 hypertension despite ongoing lifestyle modifications, the patient should be advised to consult a physician at the earliest.
4	Persons with Stage 1 or Stage 2 Hypertension should be referred to a qualified physician or started on pharmacological or non-pharmacological interventions while continuing with lifestyle modifications, based on individual risk factors.
5	Persons with systolic blood pressure (SBP) greater than 180 mmHg and or diastolic blood pressure (DBP) greater than 110 mmHg should be immediately referred to a hypertension expert for further investigation to exclude any target organ damage and for initiation of treatment, which is required on an urgent basis for gradual reduction of blood pressure over hours or days according to the target organ damage.

3.1 Definition of Hypertension

Hypertension, also known as high blood pressure, is characterized by an unusually elevated arterial blood pressure. American College of Cardiology (ACC)/American Heart Association (AHA) guidelines recommend that hypertension may be diagnosed when a person's systolic blood pressure (SBP) in the office is ≥ 130 mmHg and/ or their diastolic blood pressure (DBP) is ≥ 80 mmHg following repeated examinations.[1]

3.2 Classification of Hypertension

- Normal: <120 (systolic) and <80 (diastolic) mmHg
- Raised Blood pressure: 120-129 (systolic) and <80 (diastolic) mmHg
- Stage 1 Hypertension: 130-139 (systolic) and/or 80-89 (diastolic) mmHg
- Stage 2 Hypertension: ≥140 (systolic) and/or ≥90 (diastolic) mmHg

A 2023 systematic review and meta-analysis that combined data from over 1.5 million participants found that cardiovascular risk and all-cause mortality were significantly increased in people with systolic blood pressure 130–139 mmHg compared to those with lower pressures.[2] Intensive blood pressure lowering with targets below 130/80 mmHg significantly reduces major cardiovascular events in people with hypertension. A meta-analysis of 19 randomized trials (55,529 participants) found that intensive blood pressure-lowering strategies (often to <130/80 mmHg) lead to fewer heart attacks, strokes, and major cardiovascular events, with no significant increase in deaths, renal events, or adverse effects compared to less intensive control.[3] Recent systematic reviews and meta-analyses by Saad *et al.*[4] and Abe *et al.*[5] highlight that intensive lowering of both SBP and DBP is crucial for reducing cardiovascular events.

The recommended blood pressure target for most adults on treatment is below 130/80 mmHg. For individuals with comorbidities such as diabetes, chronic kidney disease (CKD), or cardiovascular disease (CVD), a more optimal range of 120–129/70–79 mmHg is advised. Emphasis should be placed on risk stratification in individuals with comorbidities to determine the appropriate timing and need for initiating antihypertensive

pharmacotherapy, ensuring treatment is tailored to their overall risk profile. Start antihypertensive treatment at lower blood pressure thresholds in individuals at high risk, such as those with diabetes, CKD, established CVD, or elevated calculated cardiovascular risk scores. Targets should be individualized in older adults, those with frailty, or patients experiencing symptomatic orthostatic hypotension. Treatment strategies should prioritize improvements in cardiovascular outcomes over focusing solely on blood pressure reduction.

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SECTION 4 - Blood Pressure Measurement And Monitoring

Dr. Narsingh Verma, Dr. Bijay Patni, Dr. Arvind Gupta

Recommendations

1	Screening for hypertension should be done by a trained healthcare professional using an automated, validated electronic blood pressure instrument following a standardized BP measurement procedure.
2	Mercury sphygmomanometers, aneroid blood pressure devices, should not be used to evaluate or manage hypertension in clinical practice. Cuffless automated devices are a newer technology and are not yet widely accepted for clinical diagnosis or management of hypertension. They may be used only for screening. These devices are under testing and validation to demonstrate accuracy comparable to validated cuff-based devices before being used in clinical practice.
3	Validated upper-arm automated oscillometric devices are recommended for office and out-of-office blood pressure measurements (home and ambulatory).
4	Hybrid manual auscultatory devices with LCD or LED display, or digital countdown, or shock-resistant aneroid devices can be used for office blood pressure monitoring (OBPM) if automated devices are not available.
5	OBPM is required for making a diagnosis of hypertension with at least two separate OBPMs, a few weeks (3-4) apart. The diagnosis of hypertension should be based on an OBP reading of >130/80 mm Hg.
6	OBPM should be performed in standardized conditions, with an average of two of the triplicate measurements.
7	Blood pressure should be measured at least 5 minutes after the patient enters the office.
8	The patient should be in a relaxed state, seated with the arm at the level of the heart, legs uncrossed, flat on the floor. The cuff should have a bladder whose length is about 80% and whose breadth is about 40% of the arm circumference.
9	Blood pressure should be measured in both arms at the first office visit. If there is a consistent difference between arms >10 mmHg in repeated measurements, use the arm with the higher blood pressure. If the difference is >20 mmHg, consider further investigation and refer the patient to experts.
10	Measure standing blood pressure after 1 minute and again after 3 minutes when there are symptoms suggesting postural hypotension, and at the first visit in all elderly and people with diabetes.
11	For Home blood pressure monitoring (HBPM), three readings should be obtained at 1-minute intervals, and the average of the last 2 readings after discarding the 1st reading should be considered as the blood pressure reading. This procedure should be repeated for at least 3 days, preferably 7 consecutive days, and the mean of these readings is used for clinical evaluation. Readings from the first day should be excluded when calculating the average.
12	HBPM is recommended for long-term follow-up of treated hypertension because it improves BP control, especially when combined with education and counseling.
13	Ambulatory blood pressure monitoring (ABPM) is recommended to identify white-coat hypertension, masked hypertension, and nocturnal BP phenotypes. Repeated ABPM may be necessary because these phenotypes have a limited reproducibility.
14	When ABPM is unavailable, HBPM can be done to diagnose white-coat and masked hypertension.

4.1 Mercury Devices

Measuring accurate blood pressure in patients is critical for therapeutic decisions. There are mainly two types of

sphygmomanometers – manual and digital. Mercury-based manual sphygmomanometers have been instrumental in ensuring the uniformity of blood pressure measurements in initial studies conducted to identify cardiovascular risks associated with elevated blood pressure.[1,2] In 2013–2014, international hypertension societies ultimately concluded that the risk of toxicity superseded any potential benefit of using mercury-based blood pressure devices. Electronic, non-mercury blood pressure monitoring devices have replaced the use of traditional mercury instruments in many settings.

4.2 Automated electronic devices

Automated electronic devices have been introduced that eliminate the need for additional monitoring with a stethoscope. The upper-arm cuff automated blood pressure measurement has been used and recommended for office, home, and other ambulatory settings.[3] The cuff-blood pressure method, however, has two major limitations. First, it provides only intermittent blood pressure measurements in static conditions and cannot detect rapid and dynamic blood pressure fluctuations. Secondly, using a cuff introduces errors related to its size, shape, and positioning, and the compression of the limb during inflation may alert the user and induce anxiety and discomfort during daily activities and sleep (ambulatory electronic devices with a cuff).[4,5] Several approaches have recently been developed that use cuffless technologies to estimate blood pressure based on sensors, signal processing, and algorithms embedded in wearable devices, smartphones, pocket devices, or other types of devices.[6,7] While promising cuffless devices are yet to prove their accuracy in real-world settings and amongst Indian patients.[8]

Oscillometric blood pressure monitoring devices are highly user-friendly, requiring minimal training or expertise to operate, which makes them ideal for both clinical and home settings. Their automated nature helps eliminate observer bias and reduces errors associated with manual auscultation, allowing for accurate, reproducible measurements even in noisy environments or among patients with feeble Korotkoff sounds, such as those who are obese.[9,10]

4.3 Office Blood Pressure Monitoring (OBPM)

Office blood pressure monitoring (OBPM) is often the first step in initiating a properly calibrated treatment plan. OBPM should commence at least 5 minutes after the time of the office visit, with the patient comfortably seated in a chair with back support, arms bared without constricting clothing, and supported at heart level with their feet flat on the floor. Patients should not engage in conversations during the measurement process. The measurement arm should be supported on a flat surface with an appropriately sized cuff at the mid-sternal level.[11] Preferably, blood pressure should be measured in both arms simultaneously. A validated electronic device should be used to measure blood pressure for at least three readings, at least 1 minute apart. The first reading should be ignored, and the average of the last two readings should be charted and used for considering classification and treatment strategy. Such an exercise should be repeated on 3 separate occasions in the office to confirm the diagnosis.[3,12] (Figure 1)

Assessment for the postural drop can be performed by measuring blood pressure in both supine and standing positions. Additionally, blood pressure measurements between both arms may be used to assess the presence of coarctation. If blood pressure is found to be elevated, four extremity readings should be obtained to evaluate for vascular anomalies, in particular, aortic coarctation, which the presence of a heart murmur might further suggest. Lower-extremity blood pressure is typically equal to or higher than upper-extremity blood pressure. A right upper-extremity blood pressure greater than 10 mmHg above that of lower-extremity blood pressure is a suggestive indication of aortic coarctation.[13]

4.4 Home Blood Pressure Monitoring (HBPM)

An alternative approach widely adopted for monitoring blood pressure outside the office setting is the use of HBPM. Patients are responsible for performing their own blood pressure

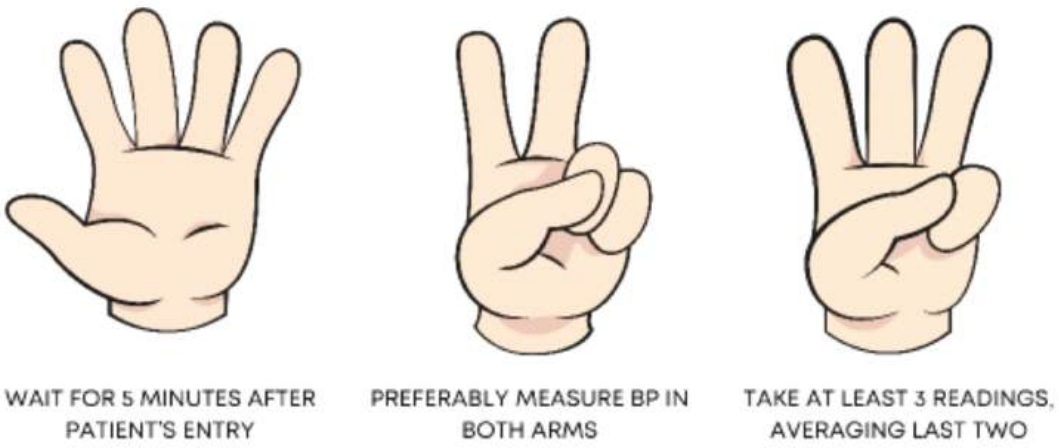

Figure 1: Blood pressure measurement

measurements, maintaining a log, and reporting data back to their physician's office. Despite various concerns related to operator error, measurement techniques, health literacy, and routine compliance, home blood pressure reading exercises have shown an association with fewer antihypertensive medications over time. Patients must be encouraged not to respond to daily fluctuations in their blood pressure values by dose adjustments; rather, they should note all the blood pressure readings in the logbook and share them with their physicians. (Figure 2)

Evidence from the J-HOP cohort indicates that keeping home systolic blood pressure (SBP) below 125 mmHg markedly lowers stroke risk and may reduce overall cardiovascular events over 10 years.[14] Similarly, a meta-analysis by Satoh *et al.*[15] (65 studies, n=21,053) showed that HBPM, especially when paired with telemonitoring or enhanced clinical support using upper-arm devices, achieved greater blood pressure reductions than standard care.

4.5 Ambulatory Blood Pressure Monitoring (ABPM)

Current guidelines for the diagnosis and management of hypertension unanimously recommend using (where possible) 24-hour ambulatory blood pressure monitoring (ABPM) for assessing blood pressure variations over the whole day, including sleeping hours. ABPM has also been recommended as a prerequisite for individualizing hypertension management, especially in patients with additional risk factors and erratic office measurements in many countries.[16] ABPM also allows for assessing the responsiveness of blood pressure to physical and mental stressors. In clinical practice, ABPM is used to determine the presence of white-coat hypertension (high office blood pressure without high out-of-office blood pressure among those not taking antihypertensive medication) and masked hypertension (high out-of-office blood pressure without high office blood pressure among those not taking antihypertensive medication), nocturnal hypertension (high night-time blood pressure), and blood pressure dipping patterns (i.e., dipping, nondipping, extreme dipping, and reverse dipping).[17,18] (Figure 2)

In an ABPM registry, especially night-time SBP, showed much stronger associations with all-cause and cardiovascular mortality than clinic blood pressure. While clinic SBP had only modest predictive value, 24-hour and night-time blood pressure remained strongly linked to mortality risk even after adjusting for clinic readings. ABPM also better identified high-risk phenotypes such as masked and sustained hypertension, whereas white-coat hypertension was not associated with excess risk.[19]

Table 1 shows the corresponding blood pressure values of ABPM and HBPM for office measurements.

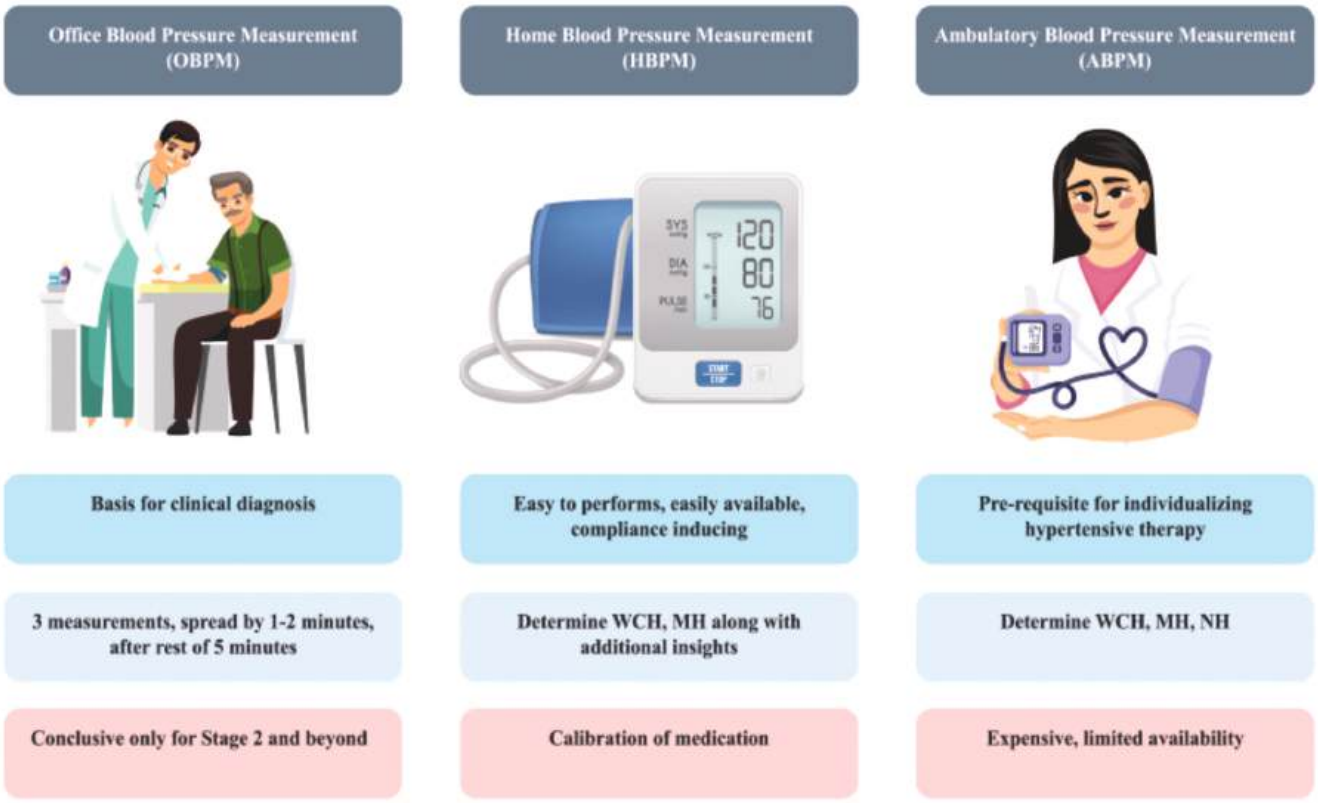

Figure 2: Office, home, ambulatory blood pressure measurement

Table 1: Corresponding SBP/DBP values for ABPM and HBPM compared with office measurements[11]

Office (mmHg)	HBPM (mmHg)	Daytime ABPM (mmHg)	Nighttime ABPM (mmHg)	24-hour ABPM (mmHg)
120/80	120/80	120/80	100/65	115/75
130/80	130/80	130/80	110/65	125/75
140/90	135/85	135/85	120/70	130/80
160/100	145/90	145/90	140/85	145/90

4.6 Special situations

4.6.1 White Coat Hypertension (WCH)

White coat hypertension (WCH) refers to a blood pressure phenotype observed in individuals with untreated raised clinic blood pressure but normal out-of-office blood pressure values.[20] WCH is an important phenomenon to understand because it is a proposed risk factor for the development of sustained hypertension, target organ damage, and possibly the occurrence of cardiovascular events.[21] Furthermore, WCH can impair myocardial function; compared with normotensive patients, there is an increased risk of carotid atherosclerosis.[22] The diagnosis is confirmed when the office blood pressure presents a clinically significant elevation [>20 mmHg for SBP and >10 mmHg for diastolic blood pressure (DBP)] compared to OBPM, ABPM, or HBPM measurements. This condition should be well evaluated, as it may give the false impression of uncontrolled hypertension.[11] (Figure 3)

According to a single-centre study, ABPM is recommended for all newly diagnosed hypertensive patients, particularly those without a family history of hypertension and with DBP <95 mmHg, to exclude WCH.[23] A study of 383 outpatients with elevated office blood pressure developed and validated a prediction model to distinguish WCH from sustained hypertension (SH). Six clinical predictors—office SBP, body mass index (BMI), sex, total cholesterol, homocysteine, and heart rate—were identified. The model showed good discrimination (AUC 0.79 in training, 0.69 in validation) and may aid clinicians in identifying WCH.[24]

4.6.2 Masked Hypertension (MH)

Masked hypertension (MH) might be suspected in persons with an increased office blood pressure at some time, in young persons with normal or high normal office blood pressure who have left ventricular hypertrophy, in persons who have a family history of hypertension in both parents, in diabetics, in persons with multiple cardiovascular risk factors, and in obese individuals. According to a systematic review by Thakkar *et al.*, the overall weighted-mean prevalence was 10% for MH and 13% for masked uncontrolled hypertension.[25] It may be reasonable to screen for MH with ABPM or with HBPM if the SBP is 120 to 129 mm Hg or if the office DBP is 75 to 79 mm Hg.[26] (Figure 3) A systematic review of 136 studies (n=28,612; ages 4–25 years) found MH in ~10% of the

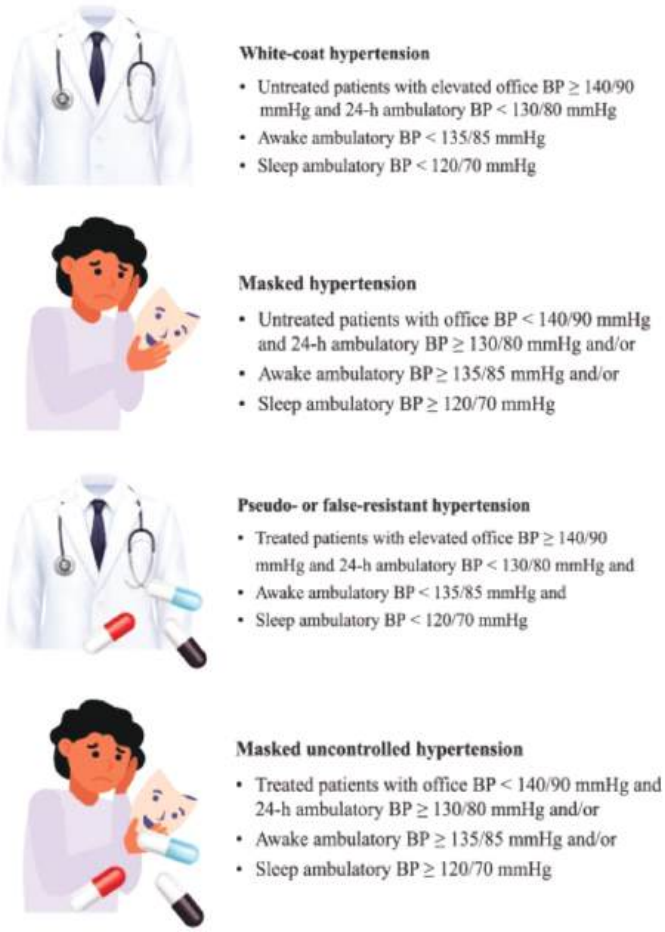

Figure 3: Criteria for the diagnosis of WCH and MH in clinical practice[38]

general pediatric population, with higher prevalence in those with coarctation of the aorta, chronic kidney disease (CKD), transplants, and sickle cell disease. Compared with normotensive peers, MH was linked to subclinical cardiovascular damage, including greater left ventricular mass index, increased risk of LVH (OR 2.44), and higher pulse wave velocity.[27]

Accurate diagnosis of hypertension in resource-limited settings remains a major public health challenge in India. Limited access to healthcare facilities, shortage of trained personnel, and inadequate availability of calibrated devices often result in underdiagnosis and late detection. In such contexts, opportunistic screening during routine clinic visits, outpatient consultations, or community health programs, Telemedicine, and simplified diagnostic algorithms offer a practical approach for early identification. In resource-constrained environments, the diagnosis should rely on standardized office blood pressure measurements, taken on at least two separate occasions. Community-based screening and follow-up by accredited social health activists (ASHAs) or auxiliary nurse midwives (ANMs) can serve as practical alternatives for confirming persistent elevation.

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SECTION 5 - Diagnosis of Hypertension, Essential & Additional Investigations

Dr. Anuj Maheshwari, Dr. Rishi Sethi, Dr. Lily Rodrigues, Dr. Dinesh Agarwal, Dr. Shailendra Vajpayee, Dr. Vinayak Hiremath

Recommendations

1	We recommend an ongoing initiative to increase awareness of hypertension, a widely prevalent but asymptomatic disease associated with many adverse outcomes like heart attacks, strokes, and kidney failure.
2	All adults aged 18 and above should undergo opportunistic screening by healthcare providers at all points of care in India.

3	Individuals with blood pressure >130/80 mmHg should be considered as having stage 1 hypertension. Early diagnosis and timely initiation of lifestyle modification and pharmacotherapy are essential to prevent progression and reduce cardiovascular risk.
4	Targeted screening at the community level is suggested for high-risk groups like the elderly (>60 years), obese, current smokers, those with diabetes, and existing cardiovascular disease or their family history.
5	History and physical examination for risk factors (smoking, obesity-diet, and exercise patterns, family history of premature CVD, angina, heart failure, stroke) should be done in all patients.
6	The presence of proteinuria doubles the risk of morbidity and mortality at a certain blood pressure level and is an independent predictor of all-cause mortality in hypertension.

5.1 Medical History

A detailed medical history is essential in hypertension for risk stratification, identification of secondary causes, and prediction of target-organ damage and cardiovascular events.[1,2] It should include hypertension duration, past blood pressure records, and history of coronary artery disease, heart failure, stroke, renal disease, peripheral arterial disease, dyslipidemia, diabetes, gout, and sleep disorders (including obstructive sleep apnea and snoring).[3]

- Family and Personal History: Assessing the family history of hypertension, premature cardiovascular disease, and the age of onset provides important insights into risk.[4] Certain occupations associated with high stress, long working hours, or sedentary lifestyles are linked to an increased risk of developing hypertension.
- Target Organ Damage and Risk Factors: Note previous cardiovascular, renal, or cerebrovascular events, as well as modifiable factors such as smoking, alcohol use, obesity, physical inactivity, stress, and sleep disturbances, which may influence adherence and outcomes.[5]
- Medication history should include the current and past use of antihypertensive agents as well as non-antihypertensive drugs that may elevate blood pressure or interfere with control, such as nonsteroidal anti-inflammatory drugs (NSAIDs), corticosteroids, oral contraceptives, and sympathomimetic agents.
- The dietary aspect of the history focuses on salt and stimulant intake, encompassing substances such as tea, coffee, caffeine-containing soft drinks, and energy beverages.

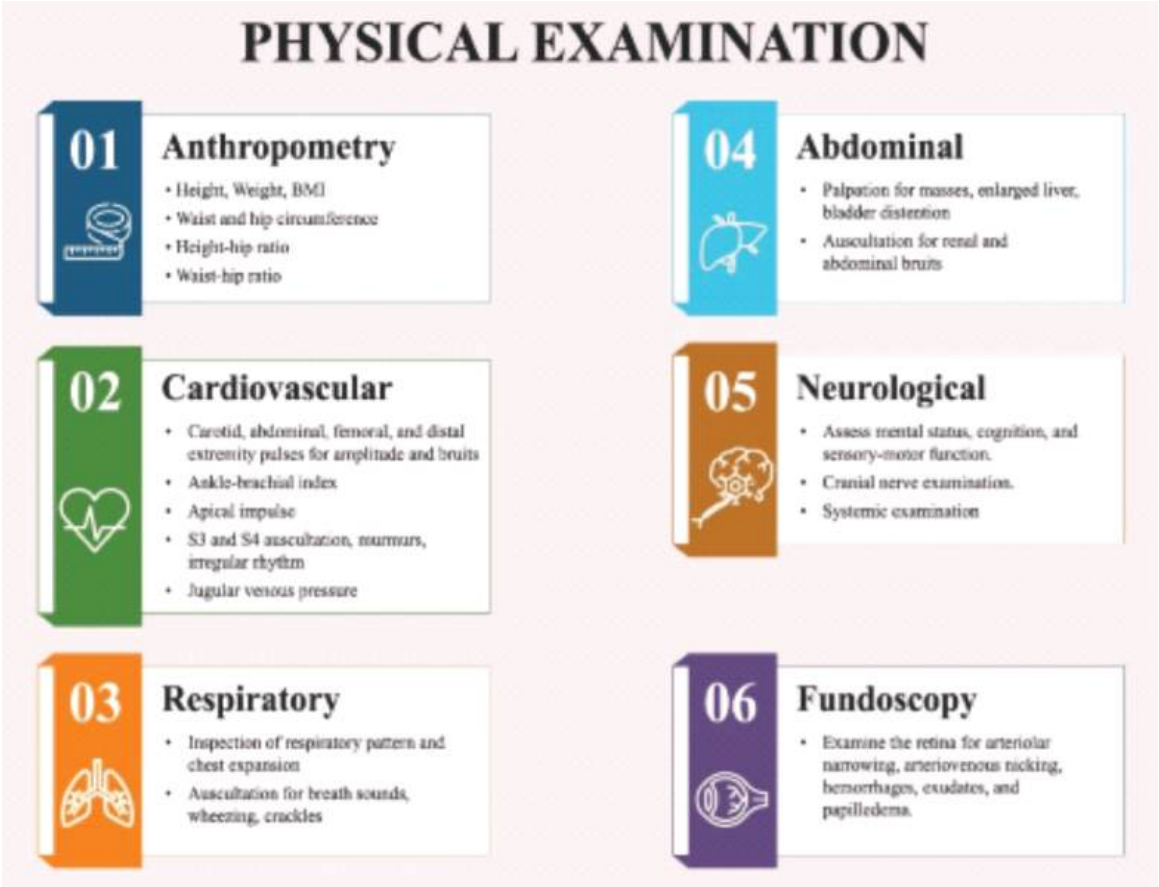

Figure 4: Physical examination

5.2 Physical examination

A comprehensive physical examination is recommended for all patients with suspected or confirmed hypertension. Physical examination and history also provide important clues about secondary hypertension, cardiovascular risk, and target organ damage.[6] (Figure 4)

5.3 Routine clinical chemistry investigation

It is important to note that laboratory testing should be performed in conjunction with other components of the physical examination, medical history, and imaging studies to obtain a comprehensive assessment of the patient's health and identify any underlying conditions contributing to hypertension. The principal goals of laboratory testing for hypertension are to assist in global cardiovascular risk assessment (e.g., detection of end-organ damage and other cardiovascular risk factors); to identify remediable forms of hypertension (e.g., consideration of hyperaldosteronism with hypokalemia); and to help guide drug selection and monitoring (e.g., avoidance of thiazide diuretics in a patient with hyponatremia).[1,2] (Table 2)

5.4 Electrocardiogram

Electrocardiography helps detect subclinical cardiovascular disease such as left ventricular hypertrophy (LVH), while cardiac biomarkers, echocardiography, and coronary calcium scoring offer more precise evaluation of cardiovascular risk and disease burden.[7] Additionally, an ECG can identify arrhythmias, such as atrial fibrillation, which can be linked to hypertension and increase the risk of stroke.[8] However, some studies have shown that LVH diagnosis by ECHO is a better predictor of cardiovascular events compared to LVH diagnosis by ECG, as the sensitivity of ECG for LVH is low (only detects advanced hypertrophy).[9]

5.5 Kidney ultrasound

Renal ultrasound is an invaluable diagnostic tool, capable of providing immediate insights at the bedside concerning potential underlying factors and subsequent implications related to hypertension. Doppler ultrasound can evaluate blood flow in renal arteries/veins, and can detect renal artery stenosis that contributes to hypertension.[10] A kidney ultrasound can help evaluate renal parenchymal changes and hemodynamic parameters like the resistive index (RI) in patients with hypertension to identify atrophic changes or arterial damage.[11] It is important to note that while the RI serves as a useful surrogate marker of intrarenal hemodynamics, it can be influenced by age, heart rate, and systemic vascular resistance, which should be considered when interpreting results.[12] Serial RI measurements may provide additional clinical value by helping to track the progression of hypertensive nephropathy or assess response to antihypertensive therapy.[13] In cases where ultrasound is inconclusive or if complex cysts/masses are suspected, Computed Tomography (CT) or Magnetic Resonance Imaging (MRI) scanning may be considered for more definitive structural evaluation.

Table 2: Investigations for individuals with raised blood pressure

Essential investigations	Additional investigations
1. Complete Blood Count with hemoglobin 2. Electrocardiogram (ECG) 3. Urinalysis with microscopy, Urine albumin creatinine ratio (UACR) 4. Kidney Function Tests, serum creatinine, Blood Urea Nitrogen (BUN), Serum electrolytes 5. Thyroid-stimulating hormone (TSH) 6. Fasting blood glucose, Glycated hemoglobin 7. Lipid profile	1. Echocardiogram (ECHO) 2. Abdominal ultrasound 3. Ankle–brachial index 4. Fundoscopy 5. Complement levels and autoimmune profile in suspected cases of autoimmune diseases affecting the renal vasculature. 6. High sensitivity C-reactive protein (hsCRP), Lipoprotein a (Lp(a)), homocysteine, uric acid, transaminases, and alkaline phosphatase. ***Endocrinal examination*** 7. 24-hour urinary free cortisol or low-dose dexamethasone suppression test to rule out Cushing's syndrome. 8. Plasma-free metanephrines or 24-hour urinary fractionated metanephrines and normetanephrine to rule out pheochromocytoma. 9. Plasma renin-aldosterone ratio to rule out hyperaldosteronism. 10. Renal arteriography

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SECTION 6 - Cardiovascular Risk Factors

Dr. M K Das, Dr. Aditya Kapoor, Dr. Sajid Ansari

Recommendations

1	Cardiovascular risk can be established by taking different risk factors into account: age (>65 years), sex (male > female), heart rate (>80 beats/min), increased body weight, diabetes, high low-density lipoprotein-cholesterol (LDL-C)/triglyceride, sleep disorders (obstructive sleep apnea), family history of hypertension, early onset of menopause, smoking habits, and psychosocial or other socioeconomic factors.
2	To lower the cardiovascular risk, the therapeutic approach must emphasize blood pressure control, lifestyle adjustments, and the efficient treatment of the other risk factors.
3	Elevated serum uric acid (s-UA) should be addressed with diet, urate-lowering medications (allopurinol or febuxostat), along with anti-hypertensive medications (losartan, fibrates, atorvastatin) that have urate-lowering as a secondary effect. Initiate urate-lowering medications in symptomatic patients (gout with s-UA >6 mg/dl [0.357 mmol/L]).
4	Hypertension and Coronary artery disease: Blood pressure to be lowered if ≥130/80 mmHg and treated to a target 120-129/70-79 mmHg (<140/80 in elderly patients).
5	Hypertension and previous stroke: Blood pressure should be lowered if ≥130/80 mmHg and treated to a target 120-129/70-79 mmHg (<140/80 in elderly patients).
6	Hypertension and Heart failure: Blood pressure to be lowered if ≥130/80 mmHg and treated to a target 120-129/70-79 mmHg.
7	Hypertension and chronic kidney disease: Blood pressure should be lowered if ≥130/80 mmHg and treated to a target 120-129/70-79 mmHg.
8	Hypertension and chronic obstructive pulmonary disease: Blood pressure should be lowered if ≥140/90 mmHg and treated to a target of <130/80 mmHg (<140/80 in elderly patients).

Hypertension is a leading driver of cardiovascular morbidity and mortality in India, where it frequently coexists with diabetes, dyslipidemia, obesity, and lifestyle-related risk factors. Comprehensive assessment of cardiovascular risk is therefore essential to guide treatment intensity, prioritize preventive strategies, and allocate resources effectively. The identification and understanding of risk factors play a pivotal role in preventing and managing cardiovascular diseases.

6.1 Diagnostic Approach

Cardiovascular risk assessment is essential for preventing cardiovascular disorders (CVDs), particularly in high-risk individuals. Angiography is considered the gold standard for identifying Coronary Artery Disease (CAD). Doppler and echocardiography can be combined to assist in diagnosing valvular stenosis and/or regurgitation by measuring blood flow across the valves. Priority may be given to transthoracic echocardiography (TTE), with a focus on assessing ventricular function and identifying regional wall motion abnormalities, to diagnose acute coronary syndrome (ACS) and rule out other potential etiologies.[1,2] (Table 3)

6.2 Cardiovascular Risk Factors

In addition to classical risk factors such as dyslipidemia, diabetes, obesity, smoking, and sedentary lifestyle, recent evidence highlights the importance of social determinants of health and environmental exposures, including noise and air pollution, which significantly contribute to cardiovascular risk in Indian populations.[3] Lipoprotein(a) [Lp(a)], a genetically determined pro-atherogenic lipoprotein, is present at elevated levels in nearly one-fourth of Indians and has been consistently linked to premature atherosclerosis and residual CV risk.[4] Similarly, coronary artery calcium (CAC) scoring has shown strong prognostic value in South Asians and Indians, offering incremental risk discrimination beyond traditional factors, especially in borderline or intermediate-risk adults.[5] In developing countries, in addition to the existing modifiable risk factors, low level of education, low vegetable and fruit intake, and alcohol abuse rank first on the list of risk factors. An increase in cardiovascular risk must be considered in patients with hypertension and chronic inflammatory diseases, chronic obstructive pulmonary disease (COPD), obstructive sleep apnea, psychiatric disorders, and psychosocial stressors, where effective blood pressure control is warranted.[6]

NFHS-5 (2019–21) identifies increasing age as the strongest predictor of hypertension, with prevalence approaching 50% in those over 60 years. Men have higher overall rates, but older and obese women carry a greater risk than their male counterparts. Urban residence, higher socioeconomic status, and certain religious or tribal affiliations are associated with socio-demographic risk. Key modifiable factors include overweight, obesity, central adiposity, elevated glucose, and alcohol use, while higher education and being unmarried were associated with a lower likelihood of hypertension.[7]

6.3 Cardiovascular Risk Scores

Accurate risk estimation is central to guiding the intensity of antihypertensive and preventive therapies. Earlier consensus

Table 3: Investigative modalities

Electrocardiography	Sum of the R-wave in aVL and the S-wave in lead V3 exceeding 2.8 mm in men and 2.0 mm in women (Cornell voltage criteria). R wave in aVL≥11 mm; Rv5 + S v1 >35 mm (Sokolow-Lyon criteria). Left atrial (LA) assessment in terms of LA hypertrophy or abnormal PQ segment depolarization-repolarization may offer advanced information on LV diastolic dysfunction.
Echocardiography Increased left ventricular (LV) mass	Increased LV mass by ASE criteria: LV mass index to body surface area (BSA), i.e., LV mass/BSA g/m^2 >115 (men) and >95 (women), E/e > 14, and Left Atrial Index volume >34, suggestive of high end filling pressure. A height-indexed LV mass exceeds 47 in women or 50 in men. A patient deemed to be high-risk based on cardiac risk stratification should be advised to undergo strain imaging so that patients prone to developing atrial arrhythmias can be diagnosed early.
Carotid ultrasound Increased carotid artery intima-media thickness (IMT)	Composite measure averaging standardized measures of the maximal common carotid artery IMT and maximal internal carotid artery IMT; a standardized carotid IMT that met or exceeded the sex-specific Common carotid IMT ≥1.0 mm.
Peripheral arterial disease Reduced ankle-brachial index (ABI)	ABI is the ratio of the average systolic blood pressure at the ankle of each leg divided by the average systolic blood pressure in the arm with the highest blood pressure. An ABI <0.9 in either leg is abnormal.
Reduced Kidney function and glomerular endothelial dysfunction	Microalbuminuria - A urine albumin to urine creatinine ratio ≥30 µg/mg. Reduced kidney function - Estimated glomerular filtration rate (eGFR) <60 mL/min per 1.73 m^2.
Arterial stiffness: High pulse wave velocity	A carotid-femoral pulse wave velocity (CFPWV) >10 m/sec.
Brain Imaging: Subclinical brain injury on magnetic resonance imaging	Lacunar infarcts, white matter hyperintensities, microbleed

documents highlighted legacy scores, such as Framingham, PROCAM, systemic coronary risk evaluation, Reynolds risk score, QRISK3 score, and JBS3; however, recent evidence suggests that these tools may under- or overestimate risk in South Asian populations.[8]

The SCORE2 Asia-Pacific model (2025) is now the preferred tool for the region, offering a recalibrated 10-year risk prediction for both fatal and non-fatal cardiovascular events among individuals without prior CVD or diabetes. Countries are categorized into low, moderate, high, or very high risk categories based on WHO standardized mortality data. The model was recalibrated using aggregate data from >8 million individuals and validated in 9.5 million participants, with a pooled C-index of 0.71 (95% CI: 0.68–0.74). Risk prediction uses standard variables (age, smoking status, systolic blood pressure, and cholesterol).[9] In resource-limited Indian settings, the updated WHO risk charts (both laboratory and non-laboratory versions) remain useful; however, local validation studies have demonstrated calibration challenges.[10] When laboratory values are available, the Pooled Cohort Equations (AHA/ACC) continue to be used in research and clinical practice, though they are not region-specific.[11]

Analysis from the Longitudinal Ageing Study in India (LASI) estimated 10-year CVD risk in adults ≥45 years using the WHO non-laboratory chart for South Asia. About 69% had a risk <10% and 2.8% had ≥20%, with both diagnosed and undiagnosed hypertension showing strong associations—linear for self-reported cases and sigmoid for actual hypertensives—emphasizing early detection. Higher risk was linked to diabetes, low education, and rural poverty, while urban residence, higher income, exercise, and being unmarried were associated with lower odds of being in a higher CVD risk score.[12] A second national survey of adults 40–69 years to assess 10-year CVD risk found 85% at low risk, 14.4% moderate, and <1% high to very high risk, with raised glucose and unemployment as key predictors.[13]

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SECTION 7- Hypertension-Mediated Organ Damage (HMOD)

Dr. Kamlakar Tripathi, Dr. Muruganathan, Dr. Shivam Verma

Recommendations

1	To optimize cardiovascular and renal risk, evaluation for hypertension-mediated organ damage (HMOD) should be performed promptly after confirming hypertension. The goal is to identify subclinical damage to guide risk stratification and treatment decisions, including the initiation or intensification of therapy. Basic HMOD screening is recommended for all hypertensive patients, with a detailed assessment when clinically relevant. However, limited resources and costs may constrain its implementation in some settings.
2	A comprehensive clinical evaluation should be performed in all patients with hypertension to determine the presence and extent of HMOD. ***Clinical assessment*** Obtain a detailed medical history covering: • Duration and severity of hypertension • Treatment adherence • Comorbidities (diabetes, dyslipidemia, chronic kidney disease)

	• Symptoms of target organ involvement (chest pain, dyspnea, pedal edema, neurological deficits, visual changes) Conduct a physical examination for: • Heart failure, carotid bruits • Peripheral arterial disease and hypertensive retinopathy Evaluate and exclude secondary hypertension due to: • Renal parenchymal or renovascular disease • Endocrine causes (primary aldosteronism, pheochromocytoma, Cushing's, thyroid disorders) • Drug- or lifestyle-induced hypertension • Use appropriate clinical, laboratory, and imaging modalities as indicated ***Routine investigations to detect HMOD*** • Serum creatinine and estimated glomerular filtration rate (eGFR) • 12-lead electrocardiogram (ECG) for left ventricular hypertrophy (LVH) • Urine Albumin-Creatinine Ratio (UACR), or alternatively, urine protein-creatinine ratio/urine albumin • Fundus examination for hypertensive retinopathy • Clinical vascular examination (peripheral pulses, carotid/femoral auscultation)
	Extended evaluation (high-resource or tertiary settings) • Echocardiography for LV mass index (gender-adjusted), geometry, LA volume index, and diastolic indices (e', E/e') • Cystatin C for early detection of nephropathy • Carotid ultrasound for plaque and intima–media thickness (cIMT) • Pulse wave velocity (PWV) and central aortic systolic BP (cSBP) via pulse wave analysis • Coronary artery calcium scoring • Ankle–brachial index (ABI) • Neuroimaging (MRI/CT) for silent cerebrovascular damage (white matter hyperintensities, lacunar infarcts) • Biomarkers: Natriuretic peptides (BNP, NT-proBNP) and high-sensitivity troponin for detecting subclinical cardiac stress/injury and for prognostic assessment.
3	Screen all patients, both hypertensive and those at risk for substances that may elevate blood pressure or diminish the effectiveness of antihypertensive therapy. Review and, where feasible, reduce or discontinue medications known to raise blood pressure. Achieve target blood pressure levels even when such medications are necessary or preferred.

7.1 Definition and Role of HMOD in Hypertension Management

Sustained elevated blood pressure leads to detrimental effects on various organs due to structural and functional changes in arteries or end organs. Hypertension-mediated organ damage (HMOD), refers to measurable structural or functional alterations in organs caused by chronically elevated blood pressure. It includes changes in the heart [left ventricular hypertrophy (LVH), diastolic dysfunction], large and small arteries (arterial stiffness, atherosclerosis, increased carotid intima–media thickness), kidneys (albuminuria, reduced glomerular filtration), brain (silent lacunar infarcts, white matter hyperintensities), and the retina (hypertensive retinopathy). HMOD can be subclinical but is prognostically important. HMOD may be present in people with less severe hypertension, including asymptomatic patients with increased blood pressure, as well as patients with severe or long-term hypertension.[1] Moreover, subclinical HMOD can already be present at the time of hypertension diagnosis. (Figure 5)

7.2 Assessment to Detect HMOD

A thorough clinical evaluation remains fundamental in the assessment of hypertension and its related organ damage. Beyond blood pressure measurement, the medical history, family history, physical examination, and basic laboratory evaluation together form the cornerstone for detecting HMOD and identifying potential secondary causes.[2] Physical examination should be systematic, beginning with general appearance, anthropometric measurements (height, weight, body mass index, waist, and neck circumference), and a detailed cardiovascular, renal, neurological, and ophthalmic assessment.[3] (Table 4)

7.3 Treatment optimization and prevention of HMOD

Lifestyle modifications, including salt restriction, weight reduction, regular physical activity, a heart-healthy diet, and avoiding alcohol and smoking, are essential for preventing and managing HMOD. Renin-angiotensin-aldosterone system (RAAS) receptor blockade is an essential part of the treatment of early end-organ damage. Mineralocorticoid receptor antagonists (MRAs), glucose-lowering agents, endothelin receptor antagonists, and statins/fibrates are useful.[5] The novel MRA finerenone shows promise in reducing albuminuria and organ damage, despite potential hyperkalemia.[6] Spironolactone is recommended as a fourth-line therapy for resistant hypertension. Sodium-glucose co-transporter-2 (SGLT2) inhibitors like canagliflozin and dapagliflozin demonstrate benefits in reducing nephropathy and albuminuria. Glucagon-like peptide-1 (GLP-1) agonists offer cardiovascular improvements and decreased albuminuria.[7] Endothelin receptor antagonists have mixed effects on albuminuria and blood pressure. Atrasentan shows promise in reducing albuminuria without significant fluid retention.[8] (refer to section 9 on management of hypertension)

7.4 Evidence Gaps, Research Priorities, and Controversies

- The optimal monitoring frequency and cost-effectiveness of broad HMOD screening in all hypertensive patients remain debated; more data are needed to balance benefit vs. resource use.

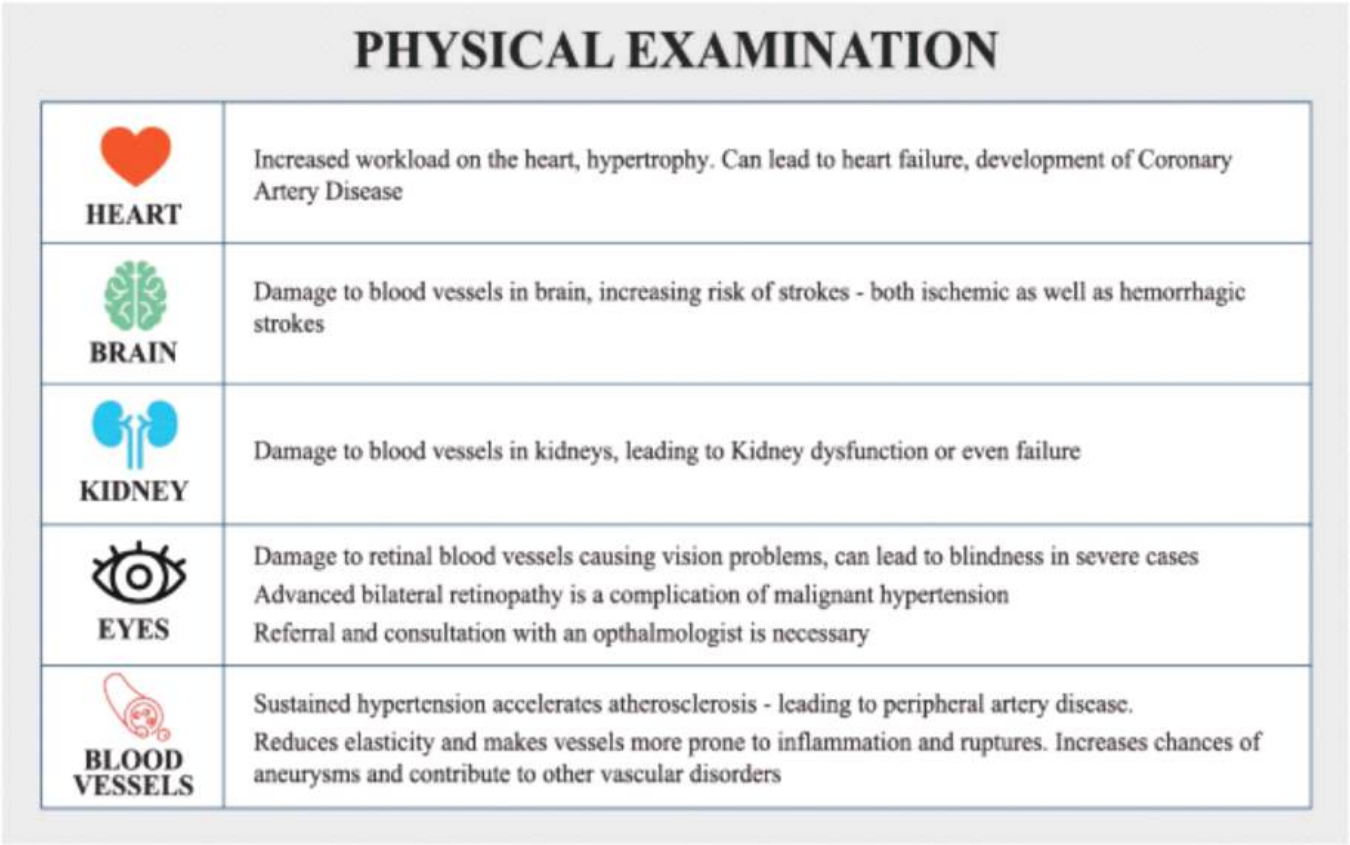

Figure 5: HMOD

Table 4: Essential and Additional Investigations for HMOD[2,4]

Organ	Essential investigations	Additional/ extended investigations	Purpose
Heart	12-lead ECG (Sokolow–Lyon, Cornell criteria for LVH)	• Echocardiography (2D/3D) for LV mass, geometry, LA volume index, diastolic indices (e′, E/e′) • Cardiac MRI (CMR) for structural and tissue characterization (fibrosis via T1 mapping / LGE) • Coronary CT angiography for suspected coronary artery disease (CAD). • Biomarkers: Natriuretic peptides (BNP, NT-proBNP) and high-sensitivity troponin	ECG is the baseline tool; echo is preferred when available. LVH on either test predicts arrhythmia, heart failure, stroke, and mortality. Biomarkers can indicate subclinical cardiac stress/injury and may provide prognostic information
Kidney	Serum creatinine + eGFR (CKD-EPI) Urine albumin-to-creatinine ratio (UACR)	• Renal ultrasound ± Doppler for morphology, resistive index (RRI > 0.7 = parenchymal disease) • CT/MR angiography for renal artery stenosis • Contrast-enhanced ultrasound (CE-US) for vascular assessment	Detects early nephropathy, renovascular disease, and chronic kidney disease (CKD) progression.
Vascular	Clinical vascular exam (peripheral pulses, carotid/femoral bruits, radio-femoral delay)	• Carotid intima–media thickness (cIMT) by ultrasonography • Pulse wave velocity (PWV) for arterial stiffness • Pulse wave analysis (PWA) for central systolic blood pressure (cSBP) and augmentation index	Evaluates arterial stiffness and subclinical atherosclerosis, useful for refining cardiovascular risk and guiding therapy.

Brain	Clinical neurological exam Cognitive screening (MMSE, MoCA > 65 years)	MRI/CT brain imaging for white-matter lesions, microbleeds, infarcts, or atrophy	Identifies cerebrovascular injury and cognitive impairment; MRI preferred for subclinical changes.
Eye	Fundoscopy for hypertensive retinopathy (hemorrhages, papilledema, microaneurysms)	• Digital fundus imaging for reproducibility • Optical coherence tomography angiography (OCTA) / fluorescein angiography (FFA) for retinal microvasculature • Scanning laser Doppler flowmetry (SLDF) • Micro-myography	Detects microvascular damage; retinal changes mirror systemic vascular injury

- Biomarker incorporation: the role of routine NT-proBNP or high-sensitivity troponin for general hypertensive population screening is promising but not universally standardized.
- Interventions beyond blood pressure lowering: pleiotropic cardioprotective therapies (SGLT2 inhibitors and GLP-1 receptor agonists show organ-protective effects in specific populations, but their routine use solely for HMOD prevention requires more targeted trials and guideline reconciliation.
- Standardising imaging cutoffs (e.g., for PWV, LA volume index) across populations and modalities to harmonise HMOD definitions internationally remains a work in progress.

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SECTION 8 - Exacerbators and Inducers of Hypertension

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Recommendations

1	Routinely screen all adults with uncontrolled or resistant hypertension for blood pressure-raising drugs, over-the-counter products, supplements, and high-sodium consumables; review at every visit.
2	Initiate/adjust antihypertensive therapy promptly when blood pressure-raising agents cannot be avoided (e.g., in oncology or transplant settings), and monitor home blood pressure during the first 2–4 weeks after initiation or dose changes.
3	Reduce sodium intake to ~2 g/day (about 5 g of salt/day) for most adults with hypertension. For people with diabetes or chronic kidney disease (CKD), aim closer to 1.5–2.0 g/day where feasible and safe.
4	Minimize intake of packaged and processed foods, encourage freshly prepared meals, and read food labels to identify high-sodium products. Minimize energy drinks with a high concentration of caffeine.
5	Nonsteroidal anti-inflammatory drugs and steroids are the two most significant prescription medications that impact blood pressure.
6	Each person's response will differ greatly, with greater increases seen in older people, those with higher baseline blood pressure, people on antihypertensive medication, and those with kidney disease.
7	We must be careful about which medications cause or worsen hypertension when they are taken for other conditions. Therefore, it is imperative to test all patients (both those with hypertension and those at risk for developing it) for substances that could raise their blood pressure or interfere with the blood pressure-lowering effects of antihypertensive drugs.

8.1 Sodium Intake

Long-term high sodium intake increases the risk of hypertension. Several health organizations advocate reducing salt intake, suggesting limits below 2 g/day of sodium (ideal limit is < 1.5 g/day) or 5 g/day of salt, on the premise that modest reductions in sodium intake will significantly decrease the incidence of cardiovascular disease in many hypertensive subjects and in resistant hypertension.[1,2] A recent systematic review and meta-analysis (36 cluster randomized trials, 66,803 participants) demonstrated that population-based sodium reduction interventions significantly lowered office systolic blood pressure by −2.6 mmHg (95% CI: −3.5 to −1.8 mmHg) compared with usual care. The benefit was greater among hypertensive and Asian populations, highlighting the high sensitivity of these groups to dietary sodium intake.[3]

8.2 Alcohol and other substances (caffeine, nicotine, and recreational drugs)

Individuals should be encouraged to avoid the use of alcohol and tobacco whenever possible, as both contribute to elevated blood pressure and a higher risk of cardiovascular disease. If alcohol is consumed, it should be limited to moderate amounts—not exceeding two standard drinks per day for men and up to one to one and a half drinks for women (one standard drink ≈ 10 g of pure alcohol).[2,4] Excessive consumption of caffeinated, sugary, or energy beverages should also be discouraged, as these can adversely influence blood pressure control and overall cardiovascular health. Recreational substances such as cannabis, amphetamine, etc, use increases the risk for acute cardiovascular events, including hypertensive crises and other life-threatening complications.[5]

Analysis of data from the National Family Health Survey (NFHS-5) indicates that tobacco use is significantly associated with an increased risk of hypertension. Women who consumed tobacco were 1.11 times more likely to have hypertension and 1.09 times more likely to exhibit elevated mean arterial pressure compared to non-users. In addition, the use of smokeless tobacco was linked to a higher likelihood of hypertension, with an odds ratio of 1.12.[6]

8.3 Nonsteroidal anti-inflammatory drugs (NSAIDs)

Non-steroidal anti-inflammatory drugs (NSAIDs) can raise blood pressure and attenuate the effect of antihypertensive therapy via renal sodium retention and neurohormonal effects.[1] India has substantial NSAID exposure through over-the-counter (OTC)/self-medication, amplifying the potential for drug-induced or resistant hypertension and acute kidney injury (AKI) when NSAIDs are combined with Angiotensin-Converting Enzyme inhibitors (ACEi)/Angiotensin Receptor Blockers (ARB) and diuretics ("triple whammy").[7,8]

Daily consumption of acetaminophen, an atypical NSAID devoid of any anti-inflammatory action, can raise the relative risk of hypertension. In a randomized, placebo-controlled crossover trial (PATH-BP), 4 g/day acetaminophen for two weeks increased systolic BP by ~5 mmHg vs placebo in patients with established hypertension.[9,10] Use the lowest effective dose for the shortest duration; reassess indication frequently, especially in patients with uncontrolled blood pressure, chronic kidney disease (CKD), or high cardiovascular risk.

8.4 Corticosteroids

Systemic corticosteroids (e.g., dexamethasone, methylprednisolone, prednisone/prednisolone) and mineralocorticoids (e.g., fludrocortisone) can raise blood pressure.[1,2] Active review, dose minimization, and monitoring, and shifting to inhaled or topical when feasible is advised. Recent reviews/meta-analyses link glucocorticoid exposure with higher risks of hypertension and adverse cardiovascular events.[11,12]

8.5 Calcineurin inhibitors

Calcineurin inhibitors (CNIs) are well-recognized causes of hypertension, affecting 50–80% of patients with long-term use.[13] Cyclosporine A (CsA) is associated with a higher incidence of hypertension compared to tacrolimus. The underlying mechanisms include afferent arteriolar vasoconstriction, activation of the renin–angiotensin–aldosterone system (RAAS), and sodium retention with volume expansion. Both CsA and tacrolimus promote vascular smooth muscle constriction, reduce nitric oxide synthesis, and stimulate endothelin-1 activity, leading to increased systemic vascular resistance. CsA additionally reduces glomerular filtration rate and enhances tubular sodium reabsorption, further elevating blood pressure.[14]

8.6 Menopausal hormone therapy and Oral contraceptive pills

Menopausal hormone therapy (MHT), including estrogen or estrogen–progestogen combinations, is frequently prescribed for vasomotor and urogenital symptoms in postmenopausal women. While MHT remains effective for symptom relief and early postmenopausal bone protection, its impact on blood pressure depends on formulation, route, and patient risk profile. Emphasize careful blood pressure monitoring before and after MHT initiation, especially in women with existing hypertension or high cardiovascular risk.[15] Systemic hormone therapy (HT) is generally contraindicated in women with established coronary heart disease (CHD), previous stroke or transient ischemic attack (TIA), venous thromboembolism (VTE), or a high 10-year atherosclerotic cardiovascular disease (ASCVD) risk (≥10%). In cases where severe menopausal symptoms persist despite non-hormonal options, treatment decisions should involve shared decision-making, with a preference for transdermal estrogen formulations when HT is deemed necessary. Systemic HT should not be initiated in women with uncontrolled blood pressure (≥180/110 mmHg); underlying risk factors should be evaluated and optimally managed before considering therapy.[16]

The risk of hypertension with oral contraceptive pills (OCPs) is influenced by estrogen dose, progestin type, and duration of use.

Higher estrogen doses and certain synthetic progestins are more likely to elevate blood pressure, and the risk increases with longer duration of therapy.[17] Women with prehypertension or additional cardiovascular risk factors such as obesity, a family history of hypertension, or metabolic syndrome are particularly susceptible. Before initiating OCPs, baseline blood pressure should be assessed, and combined estrogen-containing formulations should be avoided if blood pressure is ≥140/90 mmHg.[18] Blood pressure should be monitored at 3 and 6 months after initiation and annually thereafter. If hypertension develops, OCPs should be discontinued or switched to lower-dose or non-estrogen options, such as progestin-only pills, intrauterine devices, or barrier methods.[19]

8.1.9 Anti-cancer therapy

Anticancer therapy–related hypertension is common with VEGF/VEGFR inhibitors (mAbs and TKIs), Bruton's tyrosine kinase (BTK) inhibitors, proteasome inhibitors, and androgen-axis agents, and can be dose-limiting. Real-world and meta-analytic data reinforce high hypertension rates with VEGF-pathway drugs (up to 20–90%), substantial burdens with ibrutinib, carfilzomib, and abiraterone/enzalutamide, and show that early blood pressure control reduces downstream renal toxicity (e.g., proteinuria).[20,21]

70% patients on VEGF inhibitors develop grade 2 hypertension within one week of starting treatment, and nearly 50% develop new onset sub-nephrotic proteinuria within 12 weeks, highlighting early monitoring of blood pressure and proteinuria on initiation of VEGF inhibitors.[22] Before initiating VEGF inhibitors, blood pressure should be optimized to below 140/90 mmHg, and home blood pressure monitoring (HBPM) should be established to enable early detection of treatment-related hypertension. During therapy, anticancer treatment should be withheld if blood pressure is greater than 180/100 mmHg. Treatment may be restarted once blood pressure is controlled to below 160/100 mmHg. For antihypertensive therapy, amlodipine or another dihydropyridine calcium channel blocker (DHP-CCB) is recommended as first-line treatment. In patients with proteinuria, an ACE inhibitor or an ARB should be used preferentially.[23] Non-dihydropyridine calcium channel blockers such as diltiazem and verapamil should be avoided with most TKIs and BTK inhibitors, as these agents are metabolized by the cytochrome P450 3A4 (CYP3A4) pathway, creating a risk of significant drug–drug interactions.[24,25]

8.1.10 Other drugs

Several medications can elevate blood pressure through sympathetic activation or renal effects. Venlafaxine and other serotonin–norepinephrine reuptake inhibitors (SNRIs) modestly increase BP, while tricyclic antidepressants carry a higher hypertension risk.[26] In contrast, selective serotonin reuptake inhibitors (SSRIs) have minimal impact and are generally safe in patients with cardiovascular disease.[27] Sympathomimetic agents such as nasal decongestants, appetite suppressants, Attention-Deficit/Hyperactivity Disorder (ADHD) stimulants, and â$_2$-agonist bronchodilators can cause significant BP elevation, tachycardia, and palpitations, particularly in hypertensive or elderly patients.[28] Other agents raise blood pressure through hormonal and metabolic mechanisms. Calcitonin gene-related peptide (CGRP) inhibitors for migraine may increase blood pressure in susceptible individuals, requiring regular monitoring. Liquorice and carbenoxolone can induce hypertension and hypokalemia by inhibiting 11â-hydroxysteroid dehydrogenase, leading to increased local glucocorticoid activity and sodium retention.[29] These diverse mechanisms underscore the importance of reviewing medication, supplement, and herbal use in patients with resistant or unexplained hypertension.

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SECTION 9 - Treatment of Hypertension

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Recommendations

1	To maximize treatment benefits, blood pressure lowering should take precedence over choosing specific antihypertensive medication groups.
2	Lifestyle measures—including sodium restriction (with salt alternatives rich in potassium advised), a balanced diet rich in fruits, vegetables, and whole grains (such as the DASH diet), regular physical activity, weight control, and cessation of alcohol and tobacco use—significantly reduce blood pressure.
3	Controlled breathing techniques, mindfulness-based exercise, and meditation can all help to reduce stress.
4	Key medication classes in managing hypertension are Angiotensin-Converting Enzyme inhibitors (ACEis), Angiotensin Receptor Blockers (ARBs), Calcium channel blockers (CCBs), Thiazide/Thiazide-like diuretics, and Beta blockers (BBs), and the foundation of hypertension treatment plans should consist of these medications and their combinations.
5	Two-drug combination is advised for therapy initiation. A CCB or Thiazide/Thiazide-like diuretic should be used with a Renin-Angiotensin-Aldosterone System (RAAS) blocker (either an ACEi or an ARB).
6	Initiate pharmacological treatment in adults with confirmed office or out-of-office blood pressure ≥ 140/90 mmHg (average of multiple readings over ≥2 visits). In individuals with high cardiovascular risk, existing cardiovascular disorders (CVD), diabetes with organ damage, chronic kidney disease (CKD ≥G3), or with evidence of target-organ damage, consider starting treatment earlier, i.e., when blood pressure is >130/80 mmHg. In frail or elderly individuals, the decision should be individualized, and therapy should be cautious (e.g., starting at lower doses, monitoring more closely). In patients ≥80 years, the recommended office ~~systolic~~ threshold for initiation of drug treatment is ≥130/80 mmHg.
7	Treatment should be escalated to a three-drug combination, often a RAAS blocker, CCB, or a Thiazide/Thiazide-like diuretic, if blood pressure is not controlled with the initial two-drug combination after taking the maximum suggested and tolerable dose of each drug. Â-blockers are appropriate in cases of angina, post-myocardial infarction, heart failure (HFrEF or HFpEF with ischemia), or when heart rate control is needed.

8	Single pill combinations (SPCs) should be used wherever possible, including when starting treatment with a two-drug combination and at all other times.
9	It is not advised to combine two RAAS blockers because doing so increases the risk of side effects, including acute kidney injury (AKI).
10	In older individuals, diuretics or CCBs are favored as first-line medicines. The second stage involves combining one of the drugs from groups A (ACE inhibitor/ARB) or B (beta-blocker) with either C (calcium channel blocker) or D (thiazide diuretic).
11	Blood pressure should decline gradually. To begin treatment, start with low doses of antihypertensive medication. Age, concurrent risk factors, the presence of hypertension-mediated organ damage (HMOD), other coexisting conditions, Socioeconomic variables, the drug's availability, and the clinician's acumen all play a role in the decision to use an antihypertensive medication.
12	It is advised to continue treatment in accordance with the guidelines for resistant hypertension if blood pressure is not controlled with a three-drug combination when the maximum indicated and tolerated dose of each drug is used, and one of these drugs is a diuretic
13	Evaluate adherence to antihypertensive treatment as appropriate at each visit and prior to escalation of antihypertensive treatment. • Reducing polypharmacy – use of single pill combinations • Once-daily dosing over multiple times per day dosing • Linking adherence behavior with daily habits • Providing adherence feedback • Home blood pressure monitoring • Reminder packaging of medications • Empowerment-based counseling for self-management • Electronic adherence aids such as mobile phones or short message services • A multidisciplinary healthcare team approach (i.e., pharmacists) to improve monitoring for adherence

9.0.1 Lifestyle Modification

9.0.1.1 Yoga and mindfulness

The age-old Indian practice of yoga is known to help reduce stress and is likely to reduce blood pressure.[1]

Electroencephalogram (EEG) studies showed an initial increase in beta wave activity during practice, indicating that a calm state of mind was induced in the practitioners.[2] A meta-analysis by Ahuja *et al.*[3] (8 studies, n=482) reported that Yoga Nidra significantly lowered systolic blood pressure by 12.03 mmHg and diastolic blood pressure by 6.32 mmHg. Meditation practices can be classified into two broad categories - mindfulness meditation and concentrative meditation.[4,5] Regardless of the nuances between these types of modalities, typical outcomes for most meditative practices are mental calmness and focus. These practices can be recommended to patients to ensure a comprehensive lifestyle modification strategy.

9.0.1.2 Dietary Modification

The Dietary Approaches to Stop Hypertension (DASH) diet consists of fruits and vegetables combined with low-fat dairy products (with diminished saturated and total fat content), low levels of cholesterol, and whole grains, nuts, poultry, and fish. Usually, red meat, sweets, and sugar-containing beverages are exempted from the DASH diet and are enriched with potassium, magnesium, calcium, and fiber with excess antioxidants.[6,7] Additionally, a healthy gut flora can reduce inflammation and hence is expected to have a blood pressure-lowering effect.[8] This can be achieved through dietary measures such as increasing the intake of dietary fiber, fermented foods (e.g., yogurt, kefir, kimchi), and prebiotic or probiotic-rich foods, while reducing consumption of processed foods, excess salt, and saturated fats.

9.0.1.3 Additional lifestyle modifications

The American College of Cardiology and American Heart Association (ACC/AHA) recommend six types of additional non-pharmacological interventions: reducing alcohol intake, reducing salt intake, increasing potassium intake, engaging in physical activity, losing weight, and following heart-healthy diets.[9] The International Society of Hypertension guidelines also highlighted the importance of non-pharmacological interventions and recommended them to be used along with the antihypertensive medications for optimum control of hypertension.[7] Sodium intake should be restricted to less than 2 g per day, ideally below 1.5 g. Fast food and processed foods should be avoided. Weight reduction—preferably by at least 1 kg—through regular exercise, a low-sodium diet, and adherence to DASH or Mediterranean dietary patterns is advised, along with pharmacological therapy when indicated. Alcohol intake should ideally be avoided for individuals with hypertension; if consumed, it should be minimized (no more than two standard drinks per day for men and no more than one for women, with one standard drink containing 12–14 grams of alcohol). Smoking and energy drinks should be avoided. Engage in aerobic physical activity such as walking or jogging for 30 minutes on most days of the week (75–150 minutes weekly).[6,9]

9.0.2 Pharmacological Management of Hypertension

For decades, the blood pressure threshold of ≥140/90 mm Hg has been considered an indication for drug therapy in hypertensive individuals. ACC/AHA guidelines recommended a blood pressure of ≥130/80 mmHg as the cut-off for undergoing treatment in patients with high cardiovascular risks.[9] In view of the Indian population's high metabolic risk, it is more logical to initiate treatment at 130/80 mmHg.

A systematic review (21 studies, 63,000 participants) showed significant blood pressure reductions across all interventions, with pooled systolic and diastolic decreases of −6.95 mmHg and −4.12 mmHg, respectively. Pharmacological therapy achieved the highest target blood pressure control (67.8%) and lowest mortality (3.7%), outperforming lifestyle-only approaches, though combined strategies yielded the greatest overall BP and metabolic improvements.[10] Non-pharmacological management with regular follow-ups are generally suitable for patients with stage 1 hypertension and no associated comorbidities. When comorbid conditions exist or blood pressure remains uncontrolled with a single agent, combination therapy is advised. This approach offers several advantages, including stronger blood pressure reduction, the use of lower individual drug doses, fewer side effects, better organ protection, targeting of multiple regulatory mechanisms, improved adherence, and reduced treatment costs.[11] A meta-analysis by Wang *et al.*[12] (7 studies, n = 1,918) demonstrated that low-dose combination therapy achieved a greater mean systolic blood pressure reduction (−7.4 mmHg; 95% CI 4.3–10.5) and a higher rate of blood pressure control (<140/90 mmHg) compared with monotherapy.

Five major drug classes - Angiotensin-Converting Enzyme inhibitors (ACEIs), Angiotensin Receptor Blockers (ARBs), Calcium channel blockers (CCBs), Thiazide/Thiazide-like diuretics, and Beta blockers (BBs) form the basis of antihypertensive treatment strategies, where ACEi and ARBs belong to the Renin-Angiotensin-Aldosterone System (RAAS) blockers.

1. Patients with Stage~~Grade~~ 1 can be initiated with monotherapy (CCBs, ACE inhibitors, thiazide-like diuretics) in combination with lifestyle modifications.
2. Patients with Stage~~Grade~~ 2 hypertension or Stage 1 Hypertension with Cardiovascular risk or organ risk or uncontrolled on monotherapy, should be initiated, preferably with dual therapy as the first treatment, using a combination that is calibrated according to the patient's individual risk factors. Lifestyle modifications must equally support any combination therapy.
3. According to the European Society of Cardiology (ESC) 2024 and the ACC/AHA 2025, initiation of therapy with a two-drug combination is recommended for most hypertensive patients, where an RAAS blocker with a CCB or Thiazide is a preferred combination.[6,9]

To avoid complications, it is important to start treatment early, achieve the goals in the shortest time possible, and ensure treatment adherence.[13] For most patients with hypertension, initial therapy should consist of a single-pill combination (SPC) containing two complementary antihypertensive agents at low doses.[14] Low-dose SPC can be initiated in those with blood pressure ≥ 140/90 mmHg.[15] Dual therapy may also be started in patients with high cardiovascular risk, even at lower blood pressure. If blood pressure remains above target, a third drug can be added. Persistent hypertension despite a maximally tolerated triple regimen—typically including a RAAS, a CCB, and a diuretic—after confirming adherence and excluding secondary causes, is defined as resistant hypertension. Such cases warrant specialist referral, with the addition of spironolactone or another mineralocorticoid receptor antagonist (MRA) considered as the next therapeutic step.[6] Patients more likely to have hypertension mediated organ damage (HMOD) are recommended an initial treatment with a combination of two drugs, usually an ACEi or ARB in combination with CCB or thiazide-type diuretic, which would be the optimal treatment for all manifestations of HMOD.[16]

9.0.3 Adherence to Anti-hypertensive Treatment

Poor adherence to hypertension treatment increases the risk of cardiovascular events and mortality, and higher adherence lowers the risk of cardiovascular events; thus, improving medication adherence should be the priority in any patient with hypertension.[17] Burnier and Egan, in their review, highlighted five major categories for adherence: sociodemographic, health-care team/system, therapy-related, condition-related, and patient-related factors. More than one factor may be the reason for suboptimal adherence.[18] Multiple factors, such as type of treatment, comorbidities, cost, and physician-patient relationship, affect adherence. Various methods, such as drug prescription records, pill count, digital tools, increased physician-patient collaboration, and involvement of other healthcare professionals, can help improve adherence.[19] Besides medication adherence, there is a wide variation in adherence to lifestyle therapy (diet, physical activity, salt, alcohol restriction, etc.). A systematic review of 12 Indian studies (n = 3,164) using the Morisky Medication Adherence Scale reported a pooled adherence rate of only 15.8% (95% CI: 4.4–43.4). Factors linked to non-adherence included older age, complex medication regimens, low socioeconomic and educational status, comorbidities, and uncontrolled blood pressure.[20]

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SECTION 9.1- Drug Monitoring, Safety, and Follow-Up in Hypertension

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Recommendations

1	Dihydropyridine Calcium channel blockers (CCBs) may cause peripheral edema and reflex tachycardia; this can be mitigated with dose reduction or combining with angiotensin-converting enzyme inhibitors (ACEi)/ Angiotensin Receptor Blocker (ARB). Newer agents, such as cilnidipine, have a lower risk of edema and tachycardia. Special populations, like in chronic kidney disease (CKD), heart failure, and older age, may need closer monitoring. Diuretics may cause hypokalemia, hyperglycemia, or hyponatremia, particularly in the elderly or those with low body weight; these effects are less frequent with low doses or when combined with ACEi. ACEi can cause cough and hypotension, especially in patients on diuretics or low-sodium diets; a small rise in serum creatinine is usually reversible. An increase of up to 30% from baseline may be acceptable, helping clinicians decide when to continue or intervene. Hyperkalemia is more likely in patients with renal insufficiency, diabetes, high-dose ACEi therapy, or concurrent potassium-sparing agents. ARBs have fewer cases of cough but may cause hypotension. â-blockers require monitoring for bradycardia, heart block, hypotension, metabolic effects, and peripheral ischemia. Newer $â_1$-selective agents such as bisoprolol and nebivolol have a more favorable metabolic profile.
2	During the first three months after initiating therapy, follow-up visits, either in-person or virtual, should occur monthly or more frequently, depending on hypertension severity, cardiovascular risk, prior control, and treatment complexity. Patients who have not achieved target blood pressure may require follow-up every 1–2 weeks until control is achieved. Once blood pressure is stable, the frequency of visits should be based on disease severity, comorbidities, and the presence of target organ damage. All patients with hypertension should have an annual review to reassess blood pressure control, evaluate treatment response, review side effects, and check for target organ damage with a minimum suggested panel including urine albumin creatinine ratio (UACR), electrocardiogram (ECG) & serum creatinine.

9.1.1 Drug Monitoring and Safety

Before starting antihypertensive therapy, it is essential to perform a baseline assessment, including renal function [e.g., estimated glomerular filtration rate (eGFR) and creatinine], serum electrolytes (especially potassium), and orthostatic blood pressure readings (lying and standing) to identify hypotension risk. Orthostatic hypotension may be defined as a ≥20 mmHg

systolic or ≥10 mmHg diastolic fall, which helps ensure uniform interpretation in clinical practice.

It is also important to review co-morbidities [for example, chronic kidney disease (CKD) or heart failure] and to check for possible drug-drug interactions, especially in older adults or those on multiple medications.[1] Angiotensin-converting enzyme inhibitors (ACEi) may cause cough, angioedema, hyperkalaemia, and renal impairment, with a higher risk in patients who are dehydrated, have bilateral renal artery stenosis, or belong to populations more prone to angioedema. A modest creatinine rise (up to 30%) may be acceptable after initiation. Angiotensin receptor blockers (ARBs) share the risks of hyperkalaemia and renal dysfunction but are less likely to cause cough or angioedema. Caution is advised when using ACEi/ARBs during pregnancy and lactation, as they are contraindicated. Thiazide and thiazide-like diuretics commonly lead to hypokalaemia, hyponatraemia, hyperuricaemia, and mild increases in glucose and lipid levels, posing higher risks in individuals with gout, diabetes, or low-volume status. Calcium channel blockers (CCBs), especially the dihydropyridine type, may cause peripheral oedema, flushing, dizziness, or hypotension, effects that are more pronounced in older adults or when used with other vasodilators. Beta-blockers can cause bradycardia, fatigue, depression, and worsening of peripheral vascular disease, with caution needed in patients with asthma, chronic obstructive pulmonary disease (COPD), or those taking other heart-rate-lowering drugs.[1] However, newer â$_1$-selective agents such as bisoprolol and nebivolol have a more favorable metabolic profile, which is particularly relevant in patients with diabetes. Additionally, measure for postural (orthostatic) hypotension — especially in older patients or those taking multiple agents, by assessing blood pressure both lying down (or seated) and standing.[2]

Polypharmacy is a concern in hypertension management, especially in elderly patients with multiple conditions, increasing risks of drug interactions, adverse reactions, and non-adherence. Clinical Pharmacological Reconciliation, Review, and Feedback (CPRRF) offer a patient-centered approach, involving regular medication review, dose adjustment, and deprescribing. CPRRF promotes rational prescribing, improves affordability, and aligns therapy with patient needs, minimizing risks and improving outcomes.

9.1.2 Follow-up of Hypertension

In the first 3 months of antihypertensive treatment, patients should have follow-up visits, at least once a month, or more often depending on blood pressure severity, cardiovascular risk, prior control levels, and treatment complexity, either in person or via teleconsultation. After achieving stable control, the interval between visits can be adjusted based on disease severity, comorbidities, and evidence of target organ damage.[3] More frequent and regular follow-up visits, significantly improved systolic and diastolic blood pressure control. Shorter follow-up intervals enhanced outcomes further.[4] A quasi-experimental study from India reported that improving hypertension follow-up care, from 18% to 54%, was achieved by implementing community-based strategies such as home visits conducted by Accredited Social Health Activists (ASHAs).[5] Trained community health workers can help address undetected, untreated, and uncontrolled hypertension by improving screening, promoting lifestyle changes, monitoring treatment response, supporting adherence, and guiding individuals with raised blood pressure. A study from the Mumbai Hypertension Project found that reminder phone calls significantly improved follow-up and blood pressure control among hypertensive patients. Of those who received calls, 82% returned for follow-up within a month. Blood pressure control rates increased from 23.6% to 48.8% among those who answered calls, compared to 21.0% to 44.3% among non-responders.[6]

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SECTION 10 - Comorbidities and Complications of Hypertension and Their Management

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Recommendations

Cardiovascular disorders	
1	Drug therapy should begin in systolic blood pressure (SBP) > 130 and diastolic blood pressure (DBP) > 80 mmHg in adult patients, with coronary artery disease (CAD), along with lifestyle modifications.
2	In patients with hypertension and CAD, Angiotensin-Converting Enzyme inhibitors (ACEi) or Angiotensin Receptor Blockers (ARBs), along with beta-blockers, are preferred agents. BBs and both dihydropyridine (DHP) and non-dihydropyridine (non-DHP) calcium channel blockers (CCBs) are particularly beneficial when angina pectoris is present. The therapeutic goal is to maintain a heart rate between 60–80 beats per minute, achievable with beta-blockers or non-DHP CCBs. However, beta-blockers and non-DHP CCBs (such as diltiazem or verapamil) should not be used together, and these agents should be avoided in patients with bradycardia (heart rate ≤50 bpm).
3	Effective control of hypertension with any of the major antihypertensive drug classes—ACEi, ARBs, BBs, CCBs, or thiazide/thiazide-like diuretics—helps prevent heart failure. Blood pressure should be controlled to approximately 120-129/70-79 mmHg for optimal outcomes.
4	For patients with hypertension and heart failure with reduced ejection fraction (HFrEF), therapy should include agents with proven mortality and morbidity benefits: an ACE inhibitor or ARB [preferably replaced by an Angiotensin Receptor-Neprilysin Inhibitor (ARNI) such as sacubitril/valsartan], a beta-blocker, a mineralocorticoid receptor antagonist (MRA), and a Sodium-Glucose Cotransporter 2 (SGLT2) inhibitor if tolerated and not contraindicated. If blood pressure remains uncontrolled, along with these drug classes and adequate diuretic therapy, a DHP-CCB may be added. Non-DHP (verapamil, diltiazem) should be avoided because of their negative inotropic effects.
5	In heart failure with preserved ejection fraction (HFpEF), hypertension should be managed with standard antihypertensive agents—ACEi or ARBs, beta-blockers (used selectively for arrhythmia or ischemic heart disease), CCBs, ARNI, and thiazide-like diuretics. MRA (such as finerenone, spironolactone) are beneficial, especially in patients with lower-range HFpEF, and SGLT2 inhibitors and Glucagon-like peptide-1 receptor agonists (GLP-1 RA) are recommended to improve symptoms and outcomes, in addition to their modest BP-lowering effects.
6	In patients with atrial fibrillation (AF), blood pressure should be measured carefully, with at least three office readings by auscultation to account for beat-to-beat variability. Effective treatment of hypertension reduces the risk of stroke and other cardiovascular events in AF. The same blood pressure thresholds, targets, and antihypertensive strategies used for the general population apply to patients with AF. Beta-blockers are the preferred agents for rate control, aiming for a resting heart rate below 110 beats per minute, and ideally around 80 beats per minute in symptomatic individuals. Oral anticoagulation (preferably with DOAC) should be considered in all hypertensive patients with AF according to their thromboembolic risk.
7	In patients with aortic stenosis (AS), blood pressure thresholds, targets, and treatment strategies should follow those recommended for the general hypertensive population. However, in severe AS, blood pressure lowering should be done cautiously to avoid excessive reductions and recurrent syncope. In patients with aortic regurgitation (aortic incompetence), blood pressure thresholds and targets are the same as for the general hypertensive population. Afterload-reducing agents such as renin–angiotensin system (RAAS) blockers and CCBs (Nifedipine) are recommended to reduce left ventricular afterload, alleviate symptoms, and help prevent myocardial ischemia.
Ophthalmological Disorders	
8	All patients with hypertension should undergo a baseline fundus examination, with repeat evaluation every 1–2 years or earlier if needed. In hypertensive patients with glaucoma, ABPM and ophthalmologic examinations should be frequently done.
9	Presence of hypertensive retinopathy, retinal vein occlusion (RVO), or ischemic optic neuropathy indicates increased cardiovascular risk and should be evaluated for hypertension-mediated organ damage (HMOD).
10	BP thresholds and targets for patients with hypertensive retinopathy are the same as for the general hypertensive population (target <130/80 mmHg if tolerated).
11	All standard antihypertensive classes—ACE inhibitors, ARBs, CCBs, beta-blockers, and thiazide/thiazide-like diuretics—can be used to control blood pressure and prevent retinal vascular complications. Beta-blockers have been associated with lower intraocular pressure and decreased risk of primary open-angle glaucoma.
12	Avoid routine bedtime dosing of antihypertensive medications in patients with glaucoma or advanced optic neuropathy, as it may worsen nocturnal hypotension and optic nerve perfusion.

Cerebrovascular disorders	
13	*Acute Ischemic Stroke (AIS): Patients Eligible for Reperfusion Therapy (IV Thrombolysis or Mechanical Thrombectomy)* Before initiating intravenous thrombolysis (IV alteplase), lower blood pressure to <185/110 mmHg, and maintain ≤180/105 mmHg for the first 24 hours after treatment. For patients undergoing mechanical thrombectomy (MT), maintain blood pressure ≤185/110 mmHg before the procedure and ≤180/105 mmHg during and 24 hours after the intervention. Routine blood pressure reduction is not recommended if blood pressure is <185/105 mmHg during the first three days post-stroke.
14	*AIS: Patients Not Eligible for Reperfusion Therapy* If blood pressure ≥220/120 mmHg, cautious reduction by approximately 15% within the first 24 hours may be considered, using clinical judgment. If blood pressure is <220/120 mmHg and there are no comorbidities requiring urgent antihypertensive therapy, initiating or restarting treatment within the first 48–72 hours is not recommended. Avoid rapid or aggressive blood pressure lowering during the acute phase, as excessive reduction may worsen cerebral ischemia. In patients with comorbid conditions (e.g., acute coronary syndrome, heart failure, aortic dissection, post-fibrinolysis intracerebral hemorrhage, or pre-eclampsia/eclampsia), BP should be managed as clinically indicated.
15	In patients not receiving thrombolysis with SBP >220 mmHg or DBP >120 mmHg, reduce mean arterial pressure by no more than 15% in the first 24 hours. Avoid antihypertensive therapy within the first 72 hours of an ischemic stroke unless clinically indicated, as excessive blood pressure reduction can aggravate ischemia.
16	*Intracerebral Hemorrhage (ICH): Acute BP Management* In patients presenting within 6 hours of symptom onset with SBP between 150–220 mmHg, gradual lowering to a target of 140 mmHg, maintaining 130–150 mmHg, is advised to limit hematoma expansion. If SBP >220 mmHg, reduce blood pressure gradually using intravenous therapy to around 180 mmHg. Slow and moderate blood pressure reductions are preferred over aggressive lowering when SBP ≤220 mmHg. In large or severe ICH or those requiring surgical decompression, the safety and efficacy of intensive blood pressure lowering are not established. Excessive blood pressure reduction (<130 mmHg) in mild to moderate ICH is potentially harmful.
17	For long-term management and to prevent recurrence, maintain SBP around 130 mmHg and DBP around 80 mmHg. All major antihypertensive classes may be used for secondary prevention once the acute phase is over.
18	*Secondary Prevention After Stroke or Transient Ischaemic Attack (TIA)* All major antihypertensive classes are effective in preventing recurrent stroke when blood pressure is well controlled. Even in Stage 1 hypertension, start pharmacologic therapy in patients with prior stroke or TIA to reduce SBP to <140 mmHg. ACEi, ARBs, or thiazide/thiazide-like diuretics are preferred initial agents. For long-term prevention of recurrent stroke and vascular events, the target office blood pressure is 130/80 mmHg for most patients.
Kidney disorders	
19	Individuals with chronic kidney disease (CKD) often exhibit non-dipping or nocturnal hypertension. Ambulatory blood pressure monitoring (ABPM) or home blood pressure monitoring (HBPM) is recommended to assess blood pressure variations accurately.
20	Thresholds In CKD patients (diabetic and non-diabetic), lowering blood pressure helps slow kidney disease progression and reduce cardiovascular complications. Initiate lifestyle and pharmacologic treatment when SBP ≥130 mmHg or DBP ≥80 mmHg. The blood pressure goal for most CKD patients is <130/80 mmHg. A lower SBP target of <120 is targeted if well tolerated. A lower target of 120-129/70-79 mmHg is advisable and well tolerated in: Younger individuals, patients with albuminuria (UACR ≥300 mg/g), and in those at high cardiovascular risk. In kidney transplant recipients, the target blood pressure is <130/80 mmHg. Avoid excessive blood pressure lowering to <120/70 mmHg, as it may compromise renal perfusion.
21	Salt restriction is the cornerstone of optimal blood pressure control in CKD. However, low-sodium salts that are rich in potassium are not recommended due to the risk of hyperkalemia. Instead, limiting regular salt intake to no more than one teaspoon (≈5 grams) per day is appropriate.
22	ACEi or ARBs are recommended first-line agents, particularly in patients with moderate (UACR 30–300 mg/g) or severe albuminuria (>300 mg/g). Use the maximum tolerated dose of ACEi or ARB; monitor serum creatinine and potassium within the first week of initiation or dose adjustment. In patients with proteinuria or microalbuminuria, ACEi or ARB therapy is preferred for their antiproteinuric effect. If additional blood pressure control is required, use thiazide or thiazide-like diuretics; in volume overload, use loop diuretics (e.g., furosemide).
23	In both diabetic and non-diabetic CKD with hypertension, SGLT2 inhibitors are recommended when eGFR ≥20–25 mL/min/1.73 m^2 to slow CKD progression and reduce cardiovascular risk.

24	The non-steroidal MRA finerenone is recommended for patients with type 2 diabetes and CKD with albuminuria when eGFR ≥25 mL/min/1.73 m^2 and serum potassium <5.0 mmol/L. Finerenone offers cardiorenal protection and contributes modestly to blood pressure reduction. If hyperkalemia occurs, potassium-binding agents may be used to maintain serum potassium <5.5 mmol/L, enabling continued use of RAAS blockers or MRAs.
Diabetes Mellitus	
25	Antihypertensive therapy should be initiated when office SBP ≥130 mmHg or DBP ≥80 mmHg, along with appropriate lifestyle modifications. The treatment goal is to maintain blood pressure around 120-129/70-79 mmHg, if well tolerated. The aim is to prevent both macrovascular (e.g., coronary artery disease, stroke) and microvascular (e.g., nephropathy, retinopathy) complications.
26	To detect non-dipping or elevated nighttime blood pressure, ABPM and HBPM are recommended.
27	All classes of antihypertensive agents [RAAS inhibitors (more evidence), thiazide-type diuretics, and long-acting CCB] are useful. Combination therapy is preferred for initial management.
28	SGLT2 inhibitors are recommended for individuals with type 2 diabetes, as they reduce cardiovascular and kidney-related events and provide an additional blood pressure-lowering benefit. GLP-1 RAs and dual GLP-1 / GIP agonists are adjunctive therapies that may help lower blood pressure modestly, especially in patients with obesity or metabolic syndrome.
Obesity	
29	In adults with raised blood pressure who are overweight or obese, weight reduction is recommended to help lower blood pressure and improve overall cardiovascular outcomes.
30	Thiazide diuretics and beta-blockers may have unfavorable metabolic effects; however, achieving effective blood pressure control remains the primary treatment goal. In obese patients with diabetes and hypertension, anti-diabetic agents that help reduce both body weight and blood pressure are preferable when suitable.
31	Dual GIP/GLP-1 receptor agonists or GLP-1 receptor agonists alone should not be prescribed solely for blood pressure control in patients with obesity. Bariatric surgery should not be considered as a referral procedure for the primary purpose of controlling blood pressure.
Cancer	
32	Patients with cancer should follow the same definitions, thresholds, targets, lifestyle measures, and treatment strategies recommended for the general hypertensive population. Blood pressure and HMOD assessment is recommended before initiating anticancer therapy, regardless of prior hypertension history, as certain anticancer drugs (Tyrosine kinase inhibitors and proteasome inhibitors, corticosteroids, and calcineurin inhibitors) can cause sudden blood pressure elevations and hypertensive complications.
33	Thiazide or thiazide-like diuretics should be used only when necessary, such as in patients with fluid retention, and with caution due to risks of hypercalcemia (in bone metastasis), arrhythmias from hypokalemia, hyponatremia, and worsening dehydration. Non-DHP CCB should be avoided in patients receiving anticancer drugs that interact via CYP3A4 or P-glycoprotein pathways. Hypertension caused by VEGF inhibitors can be managed with ACEi, ARBs, or DHP CCBs.

Hypertension is a primary contributor to both mortality and morbidity of cardiovascular disorders (CVDs) like heart attacks and strokes. Logistic regression models showed hypertension associated with a six- to eight-fold increase in odds of ischemic heart disease (IHD) and stroke mortality, while diabetes was linked to double the odds of IHD and elevated odds of stroke mortality.

10.1 Cardiovascular Disorders

10.1.1 Coronary Artery Disease (CAD)

Analysis of blood pressure distribution indicates a higher prevalence of pre-hypertension and hypertension among individuals with coronary artery disease (CAD) and diabetes. Management involves the use of antihypertensive agents such as angiotensin-converting enzyme inhibitors (enalapril, lisinopril, ramipril), which promote vasodilation, lower blood pressure, and provide cardio-protection; angiotensin receptor blockers (losartan, valsartan, telmisartan), which counteract the effects of angiotensin II; beta-blockers (metoprolol, carvedilol, bisoprolol), which reduce heart rate and myocardial oxygen demand[1]; calcium channel blockers (amlodipine, verapamil), which lower vascular resistance and cardiac workload; and diuretics (thiazide-type and loop diuretics such as furosemide), which decrease fluid retention and circulating volume. Low-dose aspirin helps prevent thrombosis, while statins (atorvastatin, simvastatin, rosuvastatin) reduce cholesterol and overall cardiovascular risk.[2] Nitroglycerin improves coronary blood flow and relieves angina.[3] Optimal control of hypertension and CAD requires an integrated approach combining pharmacotherapy with lifestyle interventions, including a heart-healthy diet, regular physical activity, weight management, smoking cessation, and stress reduction.[4,5]

10.1.2 Heart Failure (HF)

In India, the estimated incidence of heart failure ranges from 0.5 to 1.7 cases per 1,000 person-years, translating to approximately 492,000 to 1.8 million new cases annually.[6] A recent study in India reported a high prevalence of stage B HF (52.4%).[7] Estimated

HF-related mortality ranges from 0.1 to 0.16 million individuals annually.[8]

Policy initiatives such as reducing salt and tobacco consumption can play a key role in the primordial prevention of heart failure. Alongside these, lifestyle measures—adopting a heart-healthy diet, regular exercise, weight control, smoking cessation, and stress management—are essential to lower heart failure risk in individuals with hypertension. Pharmacological interventions play a key role, with antihypertensive medications like ACEi, ARBs, beta-blockers, CCBs, and diuretics serving to control blood pressure and mitigate the risk of heart failure. The PARADIGM-HF trial reported that sacubitril/valsartan reduced cardiovascular deaths by 20% and HF-related hospitalizations by 21% compared to enalapril, albeit at a higher cost.[9] The EMPEROR-Preserved trial shows promising results with Sodium-Glucose Cotransporter 2 (SGLT2) inhibitors like empagliflozin in Heart failure with preserved ejection fraction (HFpEF) treatment, reducing hospitalizations regardless of diabetes status.[10] Finerenone provides significant cardiorenal benefits, including a reduced risk of cardiovascular death and hospitalization for heart failure.[11] The combination of sacubitril and valsartan [Angiotensin Receptor–Neprilysin Inhibitors (ARNis)] is effective for hypertension and resistant hypertension, in addition to its established role in managing chronic heart failure with reduced ejection fraction (HFrEF).[12]

10.1.3 Atrial Fibrillation (AF)

Hypertension is a common risk factor for nonvalvular AF. Hypertension is prevalent among Indian AF patients, but lower than in other regions. A meta-analysis of 11 randomized trials involving 42,892 patients showed that treatment with ACEi or ARBs reduced atrial fibrillation recurrence by 52% compared with CCBs and by 61% compared with beta-blockers, without affecting rates of cardiac death, myocardial infarction, or stroke.[13] Another meta-analysis of seven trials (1,495 patients) confirmed that renin–angiotensin inhibitors significantly lowered the risk of AF recurrence (OR 0.43) and persistent AF (OR 0.41) compared with CCBs.[14]

10.1.4 Valvular heart disease

Heart valve diseases contribute significantly to global cardiovascular morbidity and mortality, particularly in developing countries where rheumatic heart disease (RHD) prevails. Occurrence and patterns of valvular heart disease (VHD) can be studied using Echocardiography. Renin-angiotensin-aldosterone system (RAAS) inhibitors and CCBs are recommended to reduce left ventricular afterload and help prevent myocardial ischemia.[15,16]

10.1.5 Aortic dilatation, aneurysm, and dissection

In patients with aortic dilatation, blood pressure should be optimally controlled according to the standard hypertension management algorithm.[5] Aortic dissection is rare in childhood and demands a high level of suspicion for prompt treatment. It should be considered in children with Marfan syndrome who experience chest pain. Medications like beta-blockers, calcium channel blockers, and angiotensin inhibitors can slow aortic dilation and reduce the risk of dissection.[17,18]

10.2 Cerebrovascular Disorders

Cerebrovascular disorders, including acute ischemic stroke (AIS) and intracerebral hemorrhage (ICH), require precise blood pressure management to prevent secondary injury and optimize neurological recovery. In AIS patients who are eligible for reperfusion therapy, BP should be <185/110 mmHg before intravenous alteplase and ≤180/105 mmHg for 24 hours after treatment; similar levels apply to mechanical thrombectomy. Routine BP lowering is unnecessary if BP remains <185/105 mmHg in the initial 3 days. For AIS patients ineligible for reperfusion, treatment should be deferred unless BP is ≥220/120 mmHg, when cautious reduction by ~15% over 24 hours is warranted.[5,19] Evidence from the INTERACT-III trial underscores the value of goal-directed care bundles emphasizing BP modulation in early management of stroke in varied healthcare settings.[20] Similarly, the BOSS Study demonstrated a J-shaped relation between systolic blood pressure and recurrent events, reinforcing individualized BP control strategies.[21] The 2024 ATACH-II and MISTIE III analysis confirmed that overly aggressive systolic blood pressure (SBP) lowering increases adverse outcomes.[22] The PROGRESS trial showed that perindopril ± indapamide reduced recurrent stroke risk by 28%, confirmed by the SPS3 trial demonstrating that tighter control (<130 mmHg) reduced small-vessel stroke recurrence.[23,24] For ICH survivors, the RESTART trial validated the long-term safety of moderate blood pressure control and timely resumption of secondary prevention agents.[25] Collectively, these studies confirm that sustained BP optimization around 130/80 mmHg, using ACEi, ARBs, or thiazide-based regimens, is effective for reducing both recurrence and mortality in cerebrovascular disease.

10.3 Diabetes Mellitus

Recent guidelines emphasize an SBP target of <130 mmHg and a diastolic <80 mmHg for most adults with diabetes, particularly for those at high cardiovascular risk.[5,19] A 2025 meta-analysis of 21,169 diabetic hypertensive patients found intensive SBP lowering reduced stroke by 29%, heart failure by 31%, and cardiovascular death by 24%.[26] First-line treatment usually includes ACEi or ARBs, which not only reduce blood pressure but confer nephroprotection through attenuation of hyperfiltration and inhibition of intraglomerular hypertension. A meta-analysis by Hao *et al.*[27] (10 studies, 21,871 participants) reported that using ACEi/ARB in hypertensive individuals with type 2 diabetes reduced cardiovascular events by about 10% and cardiovascular mortality by 17%. Similarly, a systematic review by Peresuodei *et al.*[28] (10 studies, 1.6 million participants) found that both drug classes were equally effective in lowering blood pressure; however, ACEi provided greater reductions in cardiovascular events and all-cause mortality, while ARBs were better tolerated and associated with fewer adverse effects. SGLT2 inhibitors such as empagliflozin and canagliflozin have demonstrated modest blood pressure reductions (2–4 mmHg systolic) and additive renal-cardiovascular

protection, emerging as valuable adjuncts for diabetic hypertensives.[29] Finerenone demonstrated superior cardiovascular and renal outcomes compared to spironolactone and eplerenone, with a 47% reduction in MACE and 55% reduction in all-cause mortality among type 2 diabetes.[30] A 2025 meta-analysis of 30 RCTs (over 37,000 participants) reported a mean SBP reduction of –3.4 mmHg and a diastolic reduction of –1 mmHg by GLP-1 receptor agonists (GLP-1RAs).[31]

10.4 Kidney Disorders

KDIGO guidelines advocate for an SBP target of <130 mmHg (or <120 mmHg if tolerated) in CKD patients, with a DBP <80 mmHg to slow eGFR decline and lower albuminuria.[32] RAAS blockers (ACEi or ARBs), which effectively reduce proteinuria and delay chronic kidney disease (CKD) progression by lowering intraglomerular pressure and exerting anti-fibrotic effects, are recommended as first-line agents.[33] For patients with diabetic or proteinuric kidney disease, combination therapy with an ACE inhibitor or ARB and an SGLT2 inhibitor is recommended.[34] The addition of non-steroidal mineralocorticoid receptor antagonists (MRAs) like finerenone further reduces albuminuria and renal inflammation while minimizing hyperkalemia risk, thereby improving outcomes in diabetic CKD.35 Most patients require combination therapy that includes an ACEi (preferred) or an ARB, both effective in reducing albuminuria, together with a CCB or a diuretic. Cilnidipine, a CCB with both cardioprotective and renoprotective properties, demonstrates antihypertensive efficacy comparable to other first-line agents. In patients with CKD and hypertension, it effectively lowered SBP (−4.33 mmHg), reduced proteinuria (−0.61), reduced uric acid, and significantly decreased the urinary albumin-to-creatinine ratio (from 152.82 ± 82.39 to 39.55 ± 17.28).36–38 When the estimated glomerular filtration rate (eGFR) falls below 30 mL/min/1.73 m^2 (or <40 mL/min/1.73 m^2), a loop diuretic, titrated to an appropriate dose, is recommended for optimal volume control.[5,19]

10.5 Other Comorbidities and Their Management

10.5.1 Glaucoma in hypertension

Systemic hypertension may elevate intraocular pressure (IOP) by increasing aqueous humor production and episcleral venous pressure, contributing to optic nerve damage in susceptible individuals. Antihypertensive therapy itself has been shown to lower the risk of glaucoma development by approximately 43%.[39] All standard antihypertensive classes—ACE inhibitors, ARBs, CCBs, beta-blockers, and thiazide/thiazide-like diuretics—can be used to control blood pressure and prevent retinal vascular complications.

10.5.2 Hypertension oncology

Cancer therapy–related hypertension (CTRH) contributes significantly to cardiovascular morbidity and may necessitate modifications in oncologic treatment if uncontrolled. Current evidence supports initiating antihypertensive therapy at blood pressure ≥130/80 mmHg in patients with preexisting cardiovascular disease, diabetes, or high atherosclerotic risk, and at ≥140/90 mmHg in lower-risk populations.40 ACEi, ARBs, and CCBs are frontline agents due to their efficacy and cardiovascular protective effects. SGLT2 inhibitors have gained attention for their potential to mitigate treatment-induced hypertension by improving endothelial function and providing cardiorenal protection, though further oncologic-specific trials are needed.[41]

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SECTION 11 - Special Circumstances

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Recommendations

1	For resistant hypertension, confirm treatment adherence with home blood pressure monitoring (HBPM)/ ambulatory blood pressure monitoring (ABPM), exclude secondary causes, optimize angiotensin-converting enzyme inhibitor (ACEi)/angiotensin receptor blocker (ARB) + calcium channel blockers (CCB) + thiazide-like diuretic; add spironolactone if tolerated. If blood pressure remains uncontrolled, renal denervation (RDN) may be considered.
2	Patients with resistant hypertension should be referred to specialized hypertension centers for comprehensive evaluation and management.
3	Screen for secondary hypertension in patients with new-onset hypertension at a young age (<40 years), resistant hypertension (uncontrolled BP on ≥3 medications), abrupt worsening of previously well-controlled blood pressure, hypertensive crisis, presence of suggestive clinical features (e.g., hypokalemia, adrenal findings, renal bruit), or evidence of end-organ damage disproportionate to blood pressure level.
4	Use a combination of history, physical exam, targeted laboratory and imaging studies (metabolic panel, eGFR, urinalysis, urinary protein/creatinine ratio, renal ultrasound, aldosterone/renin ratio, plasma/urinary metanephrines, thyroid function) to identify common causes of secondary hypertension.
5	For patients with secondary hypertension, irrespective of etiology, the general target should be to achieve and maintain a blood pressure below 130/80 mmHg in most individuals, provided therapy is well tolerated and without adverse effects.

11.0.1 Resistant Hypertension

Resistant hypertension is a condition where blood pressure remains above the desired target (>140/90 mmHg) even though the patient is on a minimum of 3 medications with distinct mechanisms of action.[1,2] These medications should be prescribed at the highest doses the patient can tolerate, and it is recommended to include a prolonged-release calcium channel blocker (CCB), along with either an angiotensin-converting enzyme inhibitor (ACEi) or an angiotensin receptor blocker (ARB), as well as a diuretic. An alternative definition of resistant hypertension is when blood pressure is well-managed using a minimum of 4 antihypertensive medications.[3] A systematic review and meta-analysis (91 studies, n=3207911) conducted by Noubiap *et al.*[4] reported a global prevalence of 10.3% (true-resistant), 14.7% (apparent treatment-resistant), and 10.3% (pseudo-resistant hypertension). The Jaipur heart watch study by Gupta *et al.* reported a prevalence of 19.4% (uncontrolled BP > 140/90 mmHg, using any 3 drug classes) and 6.3% (uncontrolled BP, using 4 drugs).[5] (Figure 6) Lifestyle modification (weight loss and exercise) as well as enquiring for and addressing non-adherence to treatment is the initial approach to resistant hypertension followed by stepwise escalation in pharmacotherapy.

Guideline-endorsed multidrug regimens recommend initial therapy with an ACEi or ARB, a CCB, and a thiazide or thiazide-like diuretic, followed by addition of a mineralocorticoid receptor antagonist (MRA)—typically spironolactone or, in patients at risk for hyperkalemia or with chronic kidney disease (CKD), finerenone (lower risk of hyperkalemia).[6–8] Central sympatholytics may be added if blood pressure remains uncontrolled with a combination of CCBs, diuretics, and ACEi/ARBs. Direct vasodilators like hydralazine may be prescribed for severe resistant hypertension. For patients uncontrolled on four or more agents, sodium-glucose cotransporter 2 (SGLT2) inhibitors have emerged as a valuable adjunct, with a 2024 cohort showing mean systolic and diastolic pressure (SBP & DBP) reductions of 6.2/2.8 mmHg and improved target blood pressure attainment in apparent treatment-resistant hypertension and those with associated proteinuria. Guidelines now recognize renal sympathetic denervation and baroreflex activation therapy as options for selected, truly resistant cases.[9–11] Aldosterone synthase inhibitors, Baxdrostat and Lorundrostat, are being considered for use in resistant hypertension, and these may be a future novel treatment strategy.[12] In cases of refractory hypertension, an MRA, is the drug of choice, after ensuring that a long-acting thiazide-like diuretic (such as chlorthalidone) is already in use along with an ACEi or ARB and a CCB, and pseudoresistance has been excluded. (Figure 6)

11.0.2 Secondary hypertension

Patients with early onset of hypertension (<30 years of age), especially in the absence of hypertension risk factors (obesity, metabolic syndrome, familial history, etc.), should be promptly screened for causes of secondary hypertension. Additional indications for detailed assessment and screening for secondary hypertension include pediatric cases of Stage 2 hypertension, patients with malignant hypertension, patients with hypertension-mediated organ damage (HMOD), in cases with sudden deterioration of blood pressure, unprovoked or excessive hypokalemia, or metabolic alkalosis.[17] Ambulatory blood pressure monitoring (ABPM) can also be used for identifying causes of secondary hypertension, and non-dipping or reverse-dipping patterns can be identified in such observations.[18] Normally, blood pressure falls at night, creating a nocturnal "dip." The absence of this dip—or a "reverse dipping" pattern, where blood pressure rises at night—may indicate an underlying secondary cause of hypertension (Table 5,6).[19] For endocrine hypertension, refer to Section 11.2.

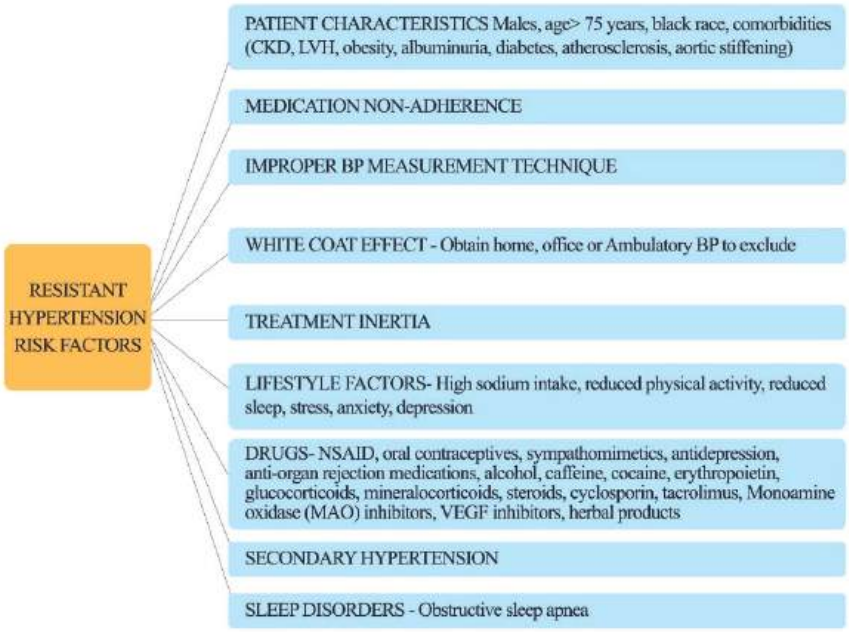

Figure 6: Resistant Hypertension - Risk factors[13,14]

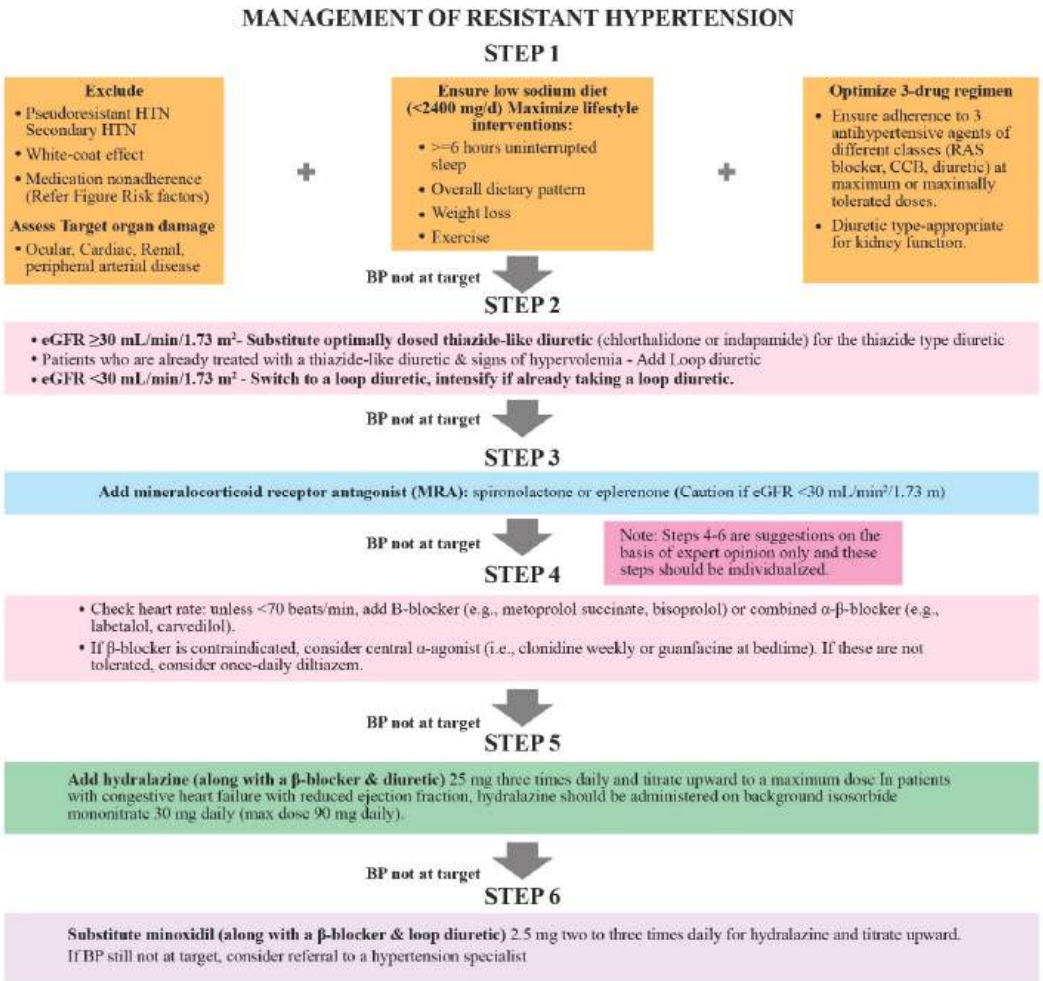

Figure 7: Algorithm for the management of Resistant hypertension[14–16]

Table 5: Classification of Secondary hypertension[20]

ENDOCRINE	Adrenal-dependent causes, Parathyroid-dependent causes, Pituitary-dependent causes, Secondary hyperaldosteronism, Thyroid-dependent causes, Vitamin D deficiency
RENAL PARENCHYMAL	Polycystic kidney disease, Chronic kidney disease, Urinary tract obstruction, Renin-producing tumor, Liddle syndrome, Chronic glomerulonephritis, Chronic interstitial nephritis, Analgesic nephropathy, Gout with renal failure
RENOVASCULAR	Renal artery stenosis, Fibromuscular dysplasia, or atherosclerosis
VASCULAR	Coarctation of aorta, Vasculitis, Collagen vascular disease
NEUROGENIC	Autonomic dysfunction, Sleep apnea, Intracranial hypertension
DRUGS & TOXINS	Alcohol, Cocaine, Cyclosporine, Tacrolimus, NSAIDs, Erythropoietin, Adrenergic medications, Decongestants containing ephedrine, Herbal remedies containing licorice or ephedrine, Nicotine

Table 6: Secondary hypertension - Non-endocrine causes

Clinical condition	Screening	Clinical Features	Diagnostic tests	Treatment
Renal parenchymal	Urinary tract infections, Hematuria, Urinary frequency and nocturia, Analgesic abuse, Family history of polycystic kidney disease, elevated serum creatinine, abnormal urinalysis.	Abdominal mass (polycystic kidney disease), skin pallor.	Proteinuria, hematuria, leukocyturia on a dipstick urine analysis, decreased estimated GFR, renal ultrasonography, or biopsy, and other tests to evaluate the cause of renal disease.	Reduced salt intake, physical activity, Renin-Angiotensin-Aldosterone System (RAAS) inhibitors, CCBs, and diuretics.
Renovascular	Resistant hypertension, hypertension of abrupt onset, worsening, or increasingly difficult to control, flash pulmonary edema (atherosclerotic), early-onset hypertension, especially in women (fibromuscular hyperplasia).	Abdominal or renal bruit, bruits over other arteries (i.e., carotid & femoral arteries), drop in estimated glomerular filtration rate (eGFR) >30% after exposure to ACEi/ ARBs.	Imaging of renal arteries (duplex ultrasound, abdominal CT, or magnetic resonance angiograms, bilateral selective renal intra-arterial angiography, decrease in eGFR.	RAAS inhibitors, CCBs, ACEi, ARBs, diuretics, and beta-blockers, percutaneous transluminal renal angioplasty (PTRA), Angioplasty, or surgical revascularization.
Obstructive Sleep Apnea (OSA)	Resistant hypertension, snoring, fitful sleep, breathing pauses during sleep, daytime sleepiness	Increased BMI, snoring, daytime sleepiness, gasping or choking at night, witnessed apneas during sleep, nocturia, Mallampati class III–IV.	STOP-Bang questionnaire, Berlin Questionnaire, Epworth Sleepiness Score, overnight Oximetry, Polysomnography.	Anti-hypertensive therapy, weight loss, oral appliances, continuous positive airway pressure (CPAP), surgery (Nasal & Bariatric)
Coarctation of aorta	Young patient with hypertension (<30 years of age)	Blood pressure is higher in the upper extremities than in lower extremities, absent femoral pulses, continuous murmur over patient's back, chest, or abdominal bruit, left thoracotomy scar (postoperative).	Echocardiogram, Computational Tomography Angiogram or Magnetic Resonance Angiogram	Surgery, ACEi, ARBs, beta-blockers

11.0.3 Hypertensive Crises (Emergencies & Urgencies)

In the Indian population, hypertensive crises have a prevalence ranging from 0.5% to 2%.[21–23] Noncompliance with antihypertensive therapy, chronic hypertension, secondary causes, antihypertensive drug withdrawal, preeclampsia and eclampsia, pseudohypertension, and some medications such as oral contraceptives, linezolid, non-steroidal anti-inflammatory drugs (NSAIDS), amphetamines, and Phencyclidine have been associated with hypertensive crisis (Tables 7 and 8).

The diagnostic evaluation of hypertensive emergencies and urgencies should include a comprehensive set of baseline tests such as fundoscopy, 12-lead ECG, complete blood counts (hemoglobin, platelets, fibrinogen, and peripheral smear), renal function tests [creatinine, eGFR, electrolytes, lactate dehydrogenase (LDH), haptoglobin], and urine analysis including urine albumin creatinine ratio (UACR) and microscopy for red blood cells, leukocytes, or casts. A pregnancy test is recommended for women of childbearing age. Depending on clinical presentation, additional investigations may include cardiac markers (troponin, NT-proBNP) in suspected acute coronary syndrome or heart failure; chest X-ray or ultrasound for pulmonary congestion; echocardiography for assessing heart failure, ischemia, or aortic dissection; CT angiography of the thorax or abdomen in suspected aortic disease; CT or MRI brain for neurological involvement; renal ultrasound for kidney impairment or renovascular disease; and urine toxicology screening in suspected stimulant (cocaine or methamphetamine) use.[14,24]

Table 7: Hypertensive urgency vs. Hypertensive emergency[14]

	Hypertensive urgency	Hypertensive emergency
Description	Blood pressure levels exceeding 180/120 mmHg, evidence of the target organ damage.	Extremely high blood pressure, exceeding 220/140 mmHg, Damage to vital organs. It may be associated with serious complications such as acute myocardial infarction, hypertensive encephalopathy, intracranial hemorrhage, dissecting aneurysm, acute renal failure, and pulmonary edema. Hypertensive emergency in pregnant women is identified when blood pressure reaches or surpasses 169/109 mmHg.
Signs and symptoms	Headache, Chest pain, Dyspnea, Epistaxis, faintness, Psychomotor agitation.	Neurologic deficits, Signs of heart failure (elevated jugular venous distention, rales on lung auscultation, or a gallop on heart auscultation), Papilledema.
Management	Patient admission and reduce Blood pressure to 160/100 mmHg over an hour, with gradual control over 12 to 48 hours. Blood pressure should not be lowered rapidly to their normal baseline. Low-dose, short-acting oral medications are preferred. Parenteral route & high dose to be avoided to protect against hypoperfusion of major arterial beds.	Admit the patient and reduce blood pressure to < 25% within 2 to 6 hours, then stabilize to 160/100 mmHg, and finally reduce to 25% within 48 hours. Admission to the ICU for prompt control. Once stabilized patient may be shifted to the general ward. Rapid-acting, parenteral agents with continuous monitoring of blood pressure, neurological status, and urine output. Aggressive reduction of blood pressure may aggravate hypoperfusion & worsening of end-organ damage. However, in patients with aortic dissection, rapid reduction of blood pressure to <120/80 mmHg should be achieved within 5–10 minutes initially with beta-blockers.
Pharmacotherapy	Captopril - 12.5–25 mg PO, Nifedipine (extended-release- 10–20 mg PO), Labetalol - 200–400 mg PO, Clonidine- 0.1–0.2 mg PO, Prazosin - 1–2 mg PO, Amlodipine - 5–10 mg.	Nicardipine - 5–15 mg/h IV infusion, starting dose 5 mg/h, increase every 15–30 min with 2.5 mg until goal, maximum 15 mg/h, Sodium Nitroprusside - 0.3–0.5 mg/kg/min IV infusion, increase by 0.5 mg/kg/min every 5 min until goal (maximum dose 10 mg/kg/min), Esmolol - 0.5–1 mg/kg IV bolus; 50–300 mg/ kg/min IV infusion Metoprolol - 2.5–5 mg IV bolus over 2 min; may repeat every 5 min to a maximum dose of 15mg, Labetalol - 10–20mg IV Bolus in 1 min incremental doses 20mg may be administered IV at 10-minute intervals (max 80 mg) or 1–3 mg/min IV infusion until goal blood pressure is reached, Fenoldopam - 0.1–0.3 mg/kg/ min IV infusion, increase every 15 min with 0.1 mg/kg/min increments until goal is reached. Nitroglycerine - 5–200 mg/min IV infusion, 5mg/min increase every 5min Enalaprilat - 0.62–1.25 mg IV bolus given over 5 min every 6 h, Clonidine - 0.2–0.5mg/kg/ min IV, Phentolamine - 1–5mg IV bolus or continuous IV infusion at a rate of 0.5–20mg/ kg/min, Hydralazine - 10–20 mg IV The bolus may be repeated every 30 minutes till the goal blood pressure is reached.

Table 8: Hypertensive emergencies requiring immediate BP-lowering with drug therapy[14]

Clinical presentation	Timing and BP target	First-line treatment	Alternative
Malignant hypertension with or without acute renal failure	Several hours Reduce Mean Arterial Pressure (MAP) by 20–25%	Labetalol Nicardipine	Nitroprusside Urapidil
Hypertensive encephalopathy	Immediately reduce MAP by 20–25%	Labetalol Nicardipine	Nitroprusside
Acute coronary event	Immediately reduce SBP to <140 mmHg	Nitroglycerine Labetalol	Urapidil
Acute cardiogenic pulmonary edema	Immediately reduce SBP to <140 mmHg	Nitroprusside or nitroglycerine (with loop diuretic)	Urapidil (with loop diuretic)
Acute aortic dissection	Immediately reduce SBP to <120 mmHg and heart rate to <60 bpm	Esmolol & nitroprusside or nitroglycerine or nicardipine	Labetalol or metoprolol
Eclampsia and severe preeclampsia/ HELLP	Immediately reduce SBP to <160 mmHg & DBP to <105 mmHg	Labetalol or nicardipine and magnesium sulphate	Consider delivery

11.0.4 Hypertension in Asthma and Chronic Obstructive Pulmonary Disease (COPD)

Elevated blood pressure is linked to worsened asthma severity, decreased lung function, and reduced forced expiratory volume in 1 second (FEV1), which serves as a marker for cardiovascular mortality. COPD fosters harmful vascular modifications such as endothelial dysfunction, atherosclerosis, and damage to the heart and kidneys (HMOD).[25] Adopting lifestyle adjustments that are relevant to both conditions is imperative. Pharmacotherapy of hypertension in patients with COPD may be considered as follows (Table 9).

11.0.5 Hypertension in the Elderly

Isolated systolic hypertension (ISH), defined by SBP greater than 140 mmHg and DBP below 90 mmHg, becomes the prevailing form of hypertension after the age of 50 years, and it is prevalent in people 70 years or older. The Longitudinal Ageing Study of India reports that nearly one in three older adults has hypertension, with 42.3% undiagnosed, 6% untreated, and 18.7% undertreated. Undiagnosed and untreated hypertension are more common in rural populations (by 12.4% and 1.7%, respectively), whereas undertreatment is higher in urban areas (by 7.2%).[26,27] Data from the National Family Health Survey (NFHS) indicate the prevalence of ISH at 1.2% and isolated diastolic hypertension (IDH) at 5.7%.[28] In the elderly, particularly the frail elderly, treatment of hypertension is initiated with monotherapy rather than a single pill combination to minimise the incidence of adverse effects. Blood pressure targets are set lower, with an office SBP of 125–135 mmHg being recommended. The initial antihypertensive dose is around half the standard dose, and gradual blood pressure control over 1–2 months is recommended. Dihydropyridine CCBs and ACEi/ ARBs are the preferred drugs for managing ISH. ACEi and ARBs should be prioritized as primary treatment options when compelling indications such as heart failure, coronary artery disease, chronic kidney disease, metabolic syndrome, or diabetes are present.[29]

11.0.6 Cardiac Autonomic Neuropathy (CAN) & Orthostatic Hypotension (OH)

Orthostatic hypotension refers to a significant drop in blood pressure (a decrease of >20 mmHg systolic or >10 mmHg diastolic) within 3 minutes of standing up, often accompanied by symptoms like lightheadedness, weakness, dizziness, palpitations, blurred vision, and even nausea and fainting.[30] Guidelines recommend screening all patients with diabetes, CKD, neurodegenerative diseases, and autonomic failure for orthostatic blood pressure changes, as coexistent OH and hypertension are independently associated with increased cardiovascular and mortality risk.[31]

Blood pressure is measured after the patient has been in the supine position for at least 5 minutes. This is followed by measurement of standing blood pressure within 3 minutes of standing.

Management

- Prescription review: Drugs known to cause or exacerbate OH must be withdrawn – tricyclic antidepressants, trazodone, tamsulosin, tizanidine, and sildenafil.

Table 9: Asthma, COPD Pharmacology

	Safe	Use with caution	Avoid
Asthma	CCBs, ARBs	Diuretics, Clonidine	Beta blockers, ACEi
COPD	CCBs, ARBs	Cardio-selective Beta blockers, Diuretics	ACEi

- Antihypertensives which abolish the sympathetic response to hypotension must be reconsidered – alpha blockers, beta blockers, and central sympatholytics must either be represcribed in a lower dose or withdrawn.
- Non-pharmacological interventions such as sleeping with the head of the bed elevated, continuous positive airway pressure (CPAP), increased fluid and salt intake (in the absence of contraindications), and use of compression stockings or abdominal binders form the first-line management to reduce OH without worsening supine hypertension.[32] Long-acting agents with a low risk for orthostatic symptoms, such as low-dose ARBs or CCBs are preferred at bedtime.[33] Treating CAN is a multifaceted endeavor encompassing lifestyle changes, improving insulin resistance, tight glycemic control, addressing dyslipidemia, and utilizing antioxidants like á-lipoic acid (ALA), aldose reductase inhibitors, acetyl-L-carnitine, and specific vitamins such as fat-soluble vitamin B1.[34]
- Pharmacological interventions: Midodrine, fludrocortisone, and droxidopa may be considered. Drugs that can treat nocturnal hypertension without resulting in early morning OH include nitroglycerine patch, nebivolol, eplerenone, losartan, and nifedipine.

11.0.7 Hypertension & Human Immunodeficiency Virus (HIV)

Management of hypertension in people living with HIV requires integrated approaches that address antiretroviral therapy (ART) interaction risks, comorbidity control, and close monitoring of drug tolerability.[35] Those who are on ART, especially protease inhibitors, should be screened frequently for new onset diabetes, hypertension and metabolic disorders. WHO guidelines recommend routine blood pressure screening at ART centers with blood pressure targets of <140/90 mmHg, while acknowledging lower targets (<130/80 mmHg) in those with diabetes or nephropathy.[36] First-line antihypertensive agents—including CCB (amlodipine), ACEi, ARBs, and thiazide diuretics—are preferred, with minimal risk of clinically meaningful drug–drug interactions for most combinations.[37]

11.0.8 Hypertension in Collagen Vascular Diseases

11.0.8.1 Rheumatoid Arthritis (RA)

Individuals diagnosed with RA experience higher cardiovascular morbidity and mortality rates. Hypertension emerges as a conventional cardiovascular risk factor with an increased prevalence and is also influenced by the presence of RA.[38] ACEi and ARBS are used as first-line antihypertensives, especially in the presence of albuminuria or insulin resistance.[39]

11.0.8.2 Progressive Systemic Sclerosis (PSS)

Progressive Systemic Sclerosis (PSS), which may be precipitated by high-dose corticosteroids, is a severe and life-threatening complication of scleroderma, which manifests with sudden and severe hypertension, rapidly progressing renal failure, hypertensive encephalopathy, congestive heart failure, and/or microangiopathic hemolytic anemia.[40] Typically, over 90% of SRC patients present with blood pressure readings exceeding 150/90 mmHg and a substantial decrease in kidney function (a reduction of ≥30% in estimated glomerular filtration rate or eGFR).[41] The rapid increase in blood pressure damages renal blood vessels, initiating a damaging cycle that ultimately culminates in malignant hypertension.[42] ACEi are the preferred initial treatment for SRC. If blood pressure remains uncontrolled even after reaching the maximum ACEi dose, CCBs can be introduced as an additional therapeutic option to enhance blood pressure management.[43]

11.0.8.3 Systemic Lupus Erythematosus (SLE)

Systemic inflammation and the therapies commonly administered to manage SLE can contribute to heightened cardiovascular risk in these patients. Lupus nephritis classes IV and V are typically associated with hypertension, primarily due to decreased glomerular filtration rates resulting from vasoconstriction.[44] Antihypertensive drug therapy should be considered when blood pressure levels reach or exceed 140/90 mmHg in patients without any hypertensive-related organ involvement. The initiation of antihypertensive drug therapy typically begins with first-line agents, which include thiazide diuretics, CCBs, as well as ACEi or ARBs.[45]

11.0.9 Sickle cell anemia

Patients with Sickle cell disease (SCD), a hereditary autosomal recessive condition resulting from mutations in the â-globin gene (HBB), face an elevated risk of developing high blood pressure. Notably, relative systemic hypertension in sickle cell disease patients is linked to heightened susceptibility to pulmonary hypertension and renal dysfunction.[46] Though not approved yet by the FDA for the treatment of systemic hypertension in those with SCD, recent trials of riociguat, a nitric oxide pathway stimulator, show safety and efficacy in reducing BP without serious adverse effects.[47]

11.0.10 Psychiatric disorders

In individuals with psychiatric disorders, depression, stress, and anxiety have all been linked to a higher prevalence of hypertension, cardiovascular morbidity, and mortality, underscoring the importance of blood pressure management in these cases.[48] For blood pressure control in patients with psychiatric conditions, using RAAS inhibitors and diuretics is preferable, as they tend to have fewer pharmacological interactions with antidepressants.

Caution should be exercised when considering CCBs and alpha1-blockers, especially in patients on treatment with medications that might cause orthostatic hypotension (e.g., serotonin reuptake inhibitors or SRIs). Beta-blockers (excluding metoprolol) may be appropriate in the presence of drug-induced tachycardia, often associated with the use of antidepressant and antipsychotic drugs.[49,50]

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SECTION 11.1 - Hypertension In Women

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Recommendations

1	It is advised to screen premenopausal women for essential and secondary hypertension.
2	When prescribing a combination of oral contraceptives, monitoring their use and effects throughout the therapy is essential.
3	For women experiencing hypertensive disorders during pregnancy, it is advisable to commence or enhance drug therapy when the systolic blood pressure (SBP) reaches or exceeds 140 mmHg and/or the diastolic blood pressure (DBP) reaches or exceeds 90 mmHg.
4	In cases of pre-existing hypertension, whether with or without superimposed pre-eclampsia, gestational hypertension, or preeclampsia (PE), the goal is to maintain blood pressure below 140/90 mmHg.
5	It is important to exercise caution in women with hypertensive disorders during pregnancy to avoid overly aggressive lowering of blood pressure. Specifically, lowering the on-treatment DBP below 80 mmHg is not recommended.
6	During pregnancy, avoiding angiotensin-converting enzyme inhibitors (ACEi), angiotensin receptor blockers (ARBs), or direct renin inhibitors is advisable. In case of planned pregnancy, it is advised to stop ACEi, ARBs, and direct renin inhibitors before conception. Labetalol (alpha and beta adrenergic antagonist), nifedipine [Dihydropyridine calcium channel blockers (CCBs)] are preferable.
7	Women with hypertension who are planning to conceive or are already pregnant should be advised to take low-dose aspirin (75–150 mg daily), as it can help reduce the risk of developing PE and related complications.

Globally, there is a prevailing notion that women exhibit lower susceptibility to cardiovascular diseases (CVD), including hypertension.[1] Unfortunately, this belief has contributed to medical professionals conducting fewer tests and administering less treatment to females for conditions like hypertension and other cardiovascular ailments.[2] According to the National Family Health Survey (NFHS-5), the prevalence of hypertension in India is 24% among men and 21% among women.[3] Analysis of data from the NFHS, Study of Ageing and Health Wave 2 datasets (NFHS-SAGE), and the Longitudinal Ageing Study of India (LASI) dataset revealed a distinct trend: males demonstrated a higher prevalence of hypertension until the age of 50, after which females exhibited higher rates.[4] Hypertension in women arises from interconnected social, metabolic, reproductive, and vascular factors. Social issues like inequality and financial insecurity, metabolic risks such as obesity and inflammation, reproductive conditions including Polycystic ovary syndrome (PCOS), adverse pregnancy outcomes, and menopause, and vascular changes like endothelial dysfunction and arterial stiffness collectively increase susceptibility, underscoring the need for a comprehensive, sex-specific approach to prevention and management.[5] Based on the WHO-SAGE study, hypertension was more prevalent in tribals, the poor, Sikhs, and older women.[6] Child marriage, adolescent pregnancy, early menarche, oral contraceptive use, hysterectomy, and exposure to domestic violence have all been linked to an increased risk of developing hypertension later in life.[7–9]

11.1.1 Premenopausal Women

Before entering menopause, women tend to exhibit slightly lower blood pressure levels and a reduced likelihood of developing hypertension. Estrogen, in particular, is believed to yield favorable effects on the cardiovascular system, potentially safeguarding women of reproductive age by amplifying nitric oxide (NO) mediated vasodilation and modulating the potent vasoconstrictor actions of endothelin-1.[10] Estrogen is also known to elevate angiotensinogen levels while simultaneously reducing renin levels, angiotensin converting enzyme (ACE) activity, angiotensin AT-1 receptor density, and aldosterone production.[11] Noteworthy contributors to secondary hypertension among younger women include obesity, polycystic ovarian syndrome, obstructive sleep apnea, aortic coarctation, Turner syndrome, autoimmune disorders, endocrine irregularities (such as hyperaldosteronism, hypothyroidism, hyperthyroidism, hyperparathyroidism, pheochromocytoma, and diabetes mellitus), renal ailments, and the use of certain medications (e.g., corticosteroids, hormonal contraceptives, etc.).[12–14]

11.1.2 Postmenopausal Women

During menopause, women exhibit a heightened occurrence of left ventricular hypertrophy (LVH) and a greater vulnerability to the development of diastolic dysfunction when compared to younger adult women. The presence of isolated systolic hypertension among postmenopausal women is intricately linked to aortic stiffness.[15] A noticeable escalation in the concentration of the potent vasoconstrictor, endothelin, is observed in postmenopausal women, which normally is inhibited by estrogens.[5,16] The decline in progesterone levels, a vasoactive hormone, may be partially linked to the emergence of arterial hypertension in postmenopausal women.[17–19] Postmenopausal hypertension might also be associated with changes in the autonomic nervous system that occur with age.[20] Secondary data from the LASI reported a prevalence of 51.68% (postmenopausal hypertension), out of which 19.14% of women were unaware. Studies conducted in other parts of India (Delhi & Kerala) reported the prevalence of systolic (27.2–32%) and diastolic blood pressure (41.1–44%).[21–24] Hormone replacement therapy (HRT) in postmenopausal women has shown contrasting results. Women with hypertension on HRT should get their blood pressure measured regularly. A systematic review and meta-analysis found that type and route of estrogen play a crucial role in determining cardiovascular safety, and HRT should be individualized, avoiding conjugated equine estrogen combinations in women at risk for hypertension.[25]

11.1.3 Hypertension in Pregnancy

Hypertensive disorders during pregnancy encompass a range of conditions, starting from pre-existing chronic hypertension in the current pregnancy and extending to intricate multi-system disorders such as preeclampsia (PE). These conditions can give rise to various complications, including eclampsia, Hemolysis, Elevated Liver enzymes, and Low Platelets (HELLP) syndrome, acute renal failure, pulmonary edema, stroke, and left ventricular failure.[24] As the World Health Organization (WHO) outlined, the lethal trifecta of pregnancy are hemorrhage, hypertensive disorders of pregnancy (HDP), and infections. These elements play a substantial role in maternal mortality and morbidity, leading to the loss of at least one woman's life every 7 minutes.[26] A systematic review and meta-analysis conducted by Dhinwa *et al.* estimated a prevalence of 11% in India (1/11 women suffer from pregnancy-induced hypertension).[27] Refer to Table 10 for classification.

Investigations: After 20 weeks of gestation, blood pressure measurement should be performed with the woman in a seated position or lying on her left side, ensuring the cuff level is aligned with the heart for accurate readings. Proteinuria assessment can be done using a urinary dipstick (≥+2) or a spot urine protein-to-creatinine ratio (≥30 mg/mmol)[29,30] Laboratory evaluation should include a complete blood count, liver and kidney function tests, serum electrolytes, and a coagulation profile (especially if the platelet count is <100,000/mm^3); if haemolysis is suspected, perform lactate dehydrogenase and a peripheral blood film (for schistocytes). Imaging and further investigations, such as chest X-ray, maternal and fetal ultrasonography, fundoscopy, 2D echocardiography, and MRI, may be warranted based on clinical indication to assess maternal and fetal well-being.

Preeclampsia (PE)

Pregnant women should undergo a comprehensive screening for preterm PE during the early stages of pregnancy. Screening should be conducted as a one-step procedure, and the best combined

Table 10: Classification- Pregnancy hypertension[24,28]

Gestational hypertension	Blood pressure levels reaching or exceeding 140/90 mmHg, identified after the 20th week of gestation, which normalize by the 42nd day after childbirth and do not display any additional signs of PE or proteinuria.
Chronic hypertension	Hypertension identified before the 20th week of gestation in the absence of neoplastic trophoblastic disease or multiple pregnancies. Alternatively, if the individual is hypertensive and/or is undergoing antihypertensive treatment. It can be either primary or secondary.
Preeclampsia (PE) Non-severe, Severe Early and Late-onset	It presents as a multi-system inflammatory disorder occurring after the 20th week of pregnancy, featuring proteinuria (≥300 mg in 24 hours or ≥+1 on dipstick), edema, and marked by the de novo onset of hypertension (blood pressure reaching or exceeding 140/90 mmHg). Atypical variant (Severe PE) - neurological, hematological, hepatic, renal manifestations, or fetal growth restriction without proteinuria.
Eclampsia	Occurrence of seizures in association with PE. Pathogenesis: hypertensive encephalopathy, cerebral edema, infarction, hemorrhage, endothelial dysfunction, etc.
Superimposed PE	PE in women with chronic hypertension.
Other types	Resistant hypertension, Chronic hypertension, and Proteinuria, Unclassified hypertension, Whitecoat hypertension, Masked and Transient hypertension.

test includes maternal risk factors, assessment of proteinuria via urine tests, measurements of mean arterial pressure (MAP), serum placental growth factor (PLGF), and uterine artery pulsatility index (UTPI). MAP is calculated from systolic (SBP) and diastolic blood pressure (DBP) readings. (MAP = DBP + (SBP-DBP)/3). Women who develop PE exhibit notably lower maternal PLGF concentrations in the first trimester compared to those with pregnancies that progress normally.[31] Primary clinical assessment for preeclampsia screening can be objectively done using the simple, easy-to-use HDP-Gestosis score. The UTPI is a Doppler ultrasound measure of blood flow resistance in the uterine arteries, with higher UTPI values indicating increased resistance linked to preeclampsia risk (refer to Table 11 for risk factors).[32]

Table 11: Risk factors and complications.[28]

PE (Maternal Risk Factors)	Maternal Complications	Fetal Complications
Maternal age, weight, height, Past obstetric history - nulliparous, parous without prior PE, parous with prior PE, interpregnancy interval in years between the birth of the last child, gestational age at delivery, birthweight of previous pregnancy beyond 24 weeks, family history of PE in mother, method of conception, smoking, chronic hypertension, Diabetes (type 1, type 2, insulin intake), systemic lupus erythematosus, antiphospholipid syndrome.	Pulmonary edema, Congestive cardiac disorders, Acute left ventricular failure (ALVF), Venous thromboembolism, Posterior reversible encephalopathy syndrome (PRES), Visual disturbances such as temporary amaurosis, Cerebral edema, Hemorrhage, Stroke, End-stage renal disease (ESRD), Placental abruption, Postpartum hemorrhage, HELLP syndrome.	Fetal growth restriction (FGR), Premature birth (iatrogenic), Small for age, Oligohydramnios, Perinatal death

Management of hypertension in women is given in Table 12.

Table 12: Management.

Patient Education	Education regarding the associated risks with high blood pressure, Advantages of making lifestyle adjustments, Education regarding the necessity of long-term treatment adherence, and the importance of consistent monitoring and therapy. Chronic hypertension - Prenatal counseling, assessment of potential end-organ damage, medication adjustments when needed, suitable lifestyle changes, and exploration of secondary factors contributing to hypertension.
Lifestyle modifications	Lifestyle modifications include a proper diet (rich in vegetables and fruits while eliminating high-fat dairy products) and regular exercise. Salt intake <5 g (Sodium <2 g/day) and potassium intake (>3.5 g/day) Weight reduction

Target	<140/90 mmHg during pregnancy, <130/80 mmHg for hypertensive adults.
Monitoring	Blood pressure measurement should be performed at every antenatal visit. Home blood pressure monitoring (HBPM) is recommended for self-monitoring between clinic visits in both low- and high-risk pregnancies. Ambulatory blood pressure monitoring (ABPM) should be considered in women with suspected white-coat, masked, or nocturnal hypertension, in high-risk pregnancies, and postpartum after preeclampsia.
Pharmacological management	
• Avoid Angiotensin-converting enzyme inhibitors (ACEi), angiotensin receptor blockers (ARBs), atenolol, direct renin inhibitors, nitroprusside, or mineralocorticoid receptor antagonist (MRA) in individuals with hypertension who are planning a pregnancy or who become pregnant.[33,34] • Low-dose aspirin (75–150 mg/day) is recommended for individuals with hypertension who are planning a pregnancy or those who become pregnant to reduce the risk of preeclampsia. Aspirin for preeclampsia prevention should be started before 12 weeks, continued throughout pregnancy, and stopped 2 days before delivery or cesarean.[24] • Oral labetalol 200 mg orally (every 4 hours, maximum 1200 mg/day) or 40 mg IV over 2 minutes as a first-line agent till the blood pressure falls to <140/90 mmHg. Alternatives include nifedipine (20 mg orally, repeat at 20-minutes intervals up to a maximum of 120 mg) and methyldopa (unavailable in India). • In severe cases of gestational hypertension and preeclampsia (>160/110 mmHg), Labetolol can be administered as slow IV bolus injections of 10–20 mg, followed by 20–80 mg every 20–30 minutes if blood pressure remains above target, with a maximum total dose of 300 mg. Oral nifedipine at a recommended dose is 10–30 mg orally, which can be repeated every 30–45 minutes if blood pressure remains uncontrolled. The maximum total dose should not exceed 120 mg per day. Hydralazine at a dose of 5 mg IV or IM, followed by 5–10 mg every 20–40 minutes until the desired BP level is achieved. If blood pressure is not controlled after 20 mg IV or 30 mg IM, an alternative drug should be considered. Continuous IV infusion (0.5–10 mg/h) may be used when close monitoring is possible. Nicardipine, at a starting dose is 1.5 mg/h as an IV infusion, which can be gradually increased by 0.5 μg/kg/min every 15–30 minutes up to a maximum of 6 mg/h.[24,33] • Seizures (eclampsia/PE): Magnesium sulfate is the drug of choice for the prevention and treatment of eclamptic seizures. In the Pritchard regimen, an initial loading dose of 4 g IV is given slowly over 5 minutes, followed immediately by 10 g IM (5 g in each buttock). This is followed by a maintenance dose of 5 g IM every 4 hours in alternate buttocks, provided deep tendon reflexes are present, respiratory rate exceeds 16/min, and urine output is >100 mL in the last 4 hours.[35] In Zuspan regimen 4 g IV loading dose over 20 minutes, followed by a 1-g/hour IV infusion, with monitoring of respiration, reflexes, and urine output throughout therapy • Corticosteroids: single course of antenatal corticosteroids is recommended for women with HDPs who are <34 weeks and at risk of delivery within 7 days (to reduce neonatal death, respiratory distress), with one repeat course considered if ≥7–14 days have passed and preterm risk persists. Corticosteroids may also be given at 34+0 to 36+6 weeks in non-diabetic women with pre-eclampsia or gestational hypertension, with a singleton pregnancy, who have not previously received steroids.[36]	
Postpartum care	Closely monitor the first 48 hours for eclampsia, Postpartum hemorrhage (PPH), hemolysis, elevated liver enzymes, low platelets (HELLP), pulmonary edema, cardiovascular, cerebrovascular events, and thrombo-embolic complications. The patient requires a 2-week post-delivery follow-up. Antihypertensive medication may be needed for 2–6 weeks. If complications arise, readmission might be necessary. A final post-partum assessment at 6 weeks is vital.

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SECTION 11.2 - Endocrine Hypertension

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Recommendations

1	Reconsider screening for endocrine hypertension in • New onset or uncontrolled hypertension (moderate/severe hypertension (>150/100 mmHg on three separate occasions) • Resistant hypertension (>140/90 mmHg on three antihypertensive medications and anyone on four or more antihypertensives) or drug-induced. • Abrupt onset of hypertension and family history of early onset • Onset of hypertension in young persons (aged <30 years) • Exacerbation of previously controlled hypertension • Disproportionate target organ damage for the degree of hypertension • Accelerated or malignant hypertension • Onset of diastolic hypertension in older adults (aged ≥65 years) • Unprovoked or excessive hypokalemia
2	Screening for primary aldosteronism is recommended in patients with resistant hypertension, spontaneous or diuretic-induced hypokalemia, sustained moderate to severe hypertension (≥150/100 mmHg on three occasions), adrenal incidentaloma, early-onset hypertension (<40 years), family history of early-onset hypertension or sudden cardiovascular events, and in those with unexplained atrial fibrillation or target organ damage disproportionate to blood pressure levels.
3	Initial screening for endocrine hypertension should encompass a comprehensive evaluation of the patient's medical history, a thorough physical examination focusing on clinical indicators, and basic blood biochemistry tests.
4	Subsequent investigations aimed at diagnosing endocrine hypertension, including additional biochemistry, imaging studies, and other relevant tests, should be selected thoughtfully. This selection process should be guided by the information gained from the patient's medical history, findings from the physical examination, and the results of initial basic clinical assessments.
5	Should an adult with persistent hypertension exhibit indications of a particular type of endocrine-related hypertension during screening, it could be prudent to consider referring them to a specialist physician well-versed in managing that specific form of hypertension. This step aims to ensure accurate diagnostic confirmation and the initiation of appropriate treatment measures.
6	Achieve a target blood pressure of <130/80 mmHg for most secondary hypertension patients, with individualization for age, frailty, CKD, and target-organ damage

Primary hypertension, often referred to as essential or idiopathic hypertension, accounts for most cases of hypertension. However, a subset of around 10–15% is attributed to secondary hypertension, and this prevalence might be underestimated (Ref. to Tables 13 & 14 for more details).[1]

Table 13: Endocrine hypertension – Etiology.[1,2]

Adrenal-dependent causes	Pheochromocytoma and sympathetic paraganglioma, Primary aldosteronism, Hyperdeoxycorticosteronism - Congenital adrenal hyperplasia (11â-Hydroxylase deficiency, 17á-Hydroxylase deficiency). Deoxycorticosterone-producing tumor, Primary cortisol resistance, Cushing syndrome, Chrousos syndrome, Genetic, Acquired (Licorice or carbenoxolone ingestion, Cushing syndrome)
Parathyroid-dependent causes	Hyperparathyroidism
Pituitary-dependent causes	Acromegaly, Cushing syndrome
Secondary hyperaldosteronism	Renovascular hypertension, Renin-producing tumor, Renal artery stenosis, Edematous disorders like left ventricular heart failure, Pregnancy, Cor-pul-monale, Cirrhosis with ascites
Thyroid-dependent causes	Hypothyroidism Hyperthyroidism
Vitamin Deficiency	Vitamin B9, Vitamin D deficiency
Complex effects	Obstructive sleep apnea (OSA)

Table 14: Secondary hypertension - Endocrine[3–5]

CLINICAL CONDITION	WHO SHOULD BE SCREENED	CLINICAL FEATURES	DIAGNOSTIC TESTS	TREATMENT
Pheochromocytoma & Paragangliomas	• Spontaneous or provoked occurrence; • Cardiovascular incidents presenting with PPGL symptoms, including Takotsubo cardiomyopathy. • Elevated blood pressure variability. • Individuals under 50 years of age with type 2 diabetes, despite a BMI below 25 kg/m^2 • Adrenal incidentaloma with a density surpassing 10 HU, even in the absence of hypertension. • Adrenal mass that is > 4 cm is cystic or has hemorrhagic changes. • Presence of genetic disorders or mutations associated with heightened PPGL risk or a family history of PPGL. • Resistant or severe hypertension (grade 3). • Indications of cervical, abdominal, or pelvic mass syndrome.	• Menard Triad - Headache (60–90%), palpitations (50–70%), and sweating (55–75%). • Skin stigmata of neurofibromatosis (café-au-lait spots, neurofibromas) • Orthostatic hypotension • Non-specific headaches, dizziness, hyperhidrosis, anxiety. • Blood pressure (labile or paroxysmal).	• 24-h urinary fractionated metanephrines or plasma metanephrines under standard conditions (supine position with an indwelling IV cannula) • Contrast-enhanced CT or MRI • Genetic testing •	• Alpha-adrenergic blockade, followed by a â-adrenergic blockade. • Tyrosine kinase inhibitors (selected patients) • Surgical resection
Primary aldosteronism (PA)	• Resistant hypertension: SBP ≥ 140 mmHg or DBP ≥ 90 mmHg despite three medications including thiazides, renin-angiotensin system blockers (RASb), and calcium channel blockers (CCB). • Grade 3 hypertension: SBP ≥ 180 mmHg or DBP ≥ 110 mmHg. • Grade 2 hypertension, especially with poor treatment response, as prevalence increases with hypertension severity. • Hypertension at a young age (< 40 years). • Hypokalemia, regardless of diuretic use. (60% patients do not have hypokalemia)	• Symptoms of hypokalemia (muscle weakness, muscle cramps, tetany) • Hypertension and an incidentally discovered adrenal mass. • Arrhythmias, especially atrial fibrillation. • Polyuria •	• Plasma aldosterone (PA) and plasma renin activity (PRA) • Elevated PA/PRA ratio (ARR) under standard conditions (correction of hypokalemia and withdrawal of aldosterone antagonists for 4–6 wk) • Oral Salt Suppression Test • Intravenous Saline Infusion Test • Captopril Challenge Test • Fludrocortisone Suppression Test • Oral Salt Suppression Test	• Spironolactone • Unilateral PA - Laparoscopic unilateral adrenalectomy.

	• Presence of adrenal incidentaloma. • Family history of PA, although monogenic forms are rare. • Unexplained atrial fibrillation by structural heart disease or hyperthyroidism. • Early stroke or disproportionate renal/ cardiovascular complications relative to age or hypertension severity, as patients with PA tend to have higher cardiovascular and renal morbidity. • Hypertension and obstructive sleep apnea.		• Intravenous Saline Infusion Test • CT scan & adrenal vein sampling. • Genetic testing	
Cushing's syndrome	• Young individuals with atypical medical conditions, like osteoporosis and resistant hypertension. • Patients exhibiting classic manifestations such as easy bruising, weight gain, facial redness, and purple striae. • Children experiencing a decline in height percentile, coupled with rising weight. • Individuals with adrenal incidentalomas suggestive of adenomas. •	• Truncal obesity • Moon face • Hypertension • Skin atrophy and bruising • Purple striae • Diabetes or glucose intolerance • Gonadal dysfunction • Muscle weakness • Hirsutism, acne • Mood disorders, insomnia, depression • Osteoporosis • Fungal infections	• Overnight 1mg Dexamethasone suppression test. • 24-hour Urinary free cortisol • Midnight salivary/ plasma cortisol • Morning plasma Adrenocorticotropic hormone (ACTH) • ACTH stimulation by corticotropin-releasing hormone (CRH) or desmopressin • CT imaging	• Spironolactone or diuretics to treat hypokalemia (ectopic ACTH production). • Surgical resection.
Hyperthyroidism	• Warm, moist skin • Heat intolerance • Nervousness, Tremulousness • Insomnia • Weight loss • Diarrhea & proximal muscle weakness • Healthcare providers should perform screening for hyperthyroidism in individuals with hypertension if there are indications based on their clinical symptoms.	• Tremor • Tachycardia • Atrial fibrillation • Weight loss • Goiter • Ophthalmopathy • Pretibial myxedema.	• Thyroid-stimulating hormone • Free thyroxine • Radioactive iodine uptake and scan. • •	Antithyroid medications and Beta-blockers.
Hypothyroidism	• Dry skin • Cold intolerance • Constipation • Hoarseness • Weight gain	• Fatigue & Cold Intolerance • Weight gain • Non-pitting edema • Periorbital puffiness • Slow speech	• Thyroid-stimulating hormone • Free thyroxine	Levothyroxine

	• Healthcare providers should perform screening for hypothyroidism in individuals with hypertension if there are indications based on their clinical symptoms.	• Coarse voice and skin • Constipation & Enlarged tongue • Brittle hair & Delayed ankle reflex • Bradycardia		
Primary Hyperparathyroidism (PHTP)	Hypercalcemia	Bones, stones, abdominal groans, and psychic moans, polyuria, and polydipsia	PTH, calcium, phosphate, albumin, and vitamin D, as well as 24 h urinary calcium excretion	• Surgical excision of the gland. • Vitamin D supplements.
Growth Hormone (GH)	Patients with hypertension, incidentally discovered pituitary tumors, and patients with typical clinical manifestations of acromegaly	Acral features, large hands and feet, frontal bossing, progressive dental malocclusion, degenerative arthritis, low-pitched sonorous voice, excessive sweating, and oily skin, perineural hypertrophy leading to nerve entrapment, and cardiac dysfunction.	• Serum growth hormone ≥1 ng/mL during oral glucose load. • Elevated age- and sex-matched IGF-1 level • MRI scan of the pituitary	• Somatostatin analogs • GH receptor antagonist • Dopamine agonists • Surgical resection of tumor.
Congenital Adrenal Hyperplasia (CAH)	Children, adolescents, &young adults with hypertension, spontaneous hypokalemia, & low levels of aldosterone and renin. *11-beta-OH*- Girls with virilization and boys with pseudoprecocious puberty. *17-alpha-OH*- primary amenorrhea and pseudohermaphroditism	• *11-beta-OH* - Girls (hypertension, hypokalemia, acne, hirsutism, & virilization), Boys (hypertension, hypokalemia, and pseudoprecocious puberty) • *17-alpha-OH* - primary amenorrhea & pseudohermaphroditism	• Hypokalemia with low or normal aldosterone & renin. • *11-beta-OH*: elevated DOC, 11-deoxycortisol & androgens • *17-alpha-OH* - decreased androgens & estrogen; elevated DOC & corticosterone. • Germline mutation testing	• Glucocorticoid replacement • Bilateral adrenalectomy

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SECTION 11.3 - Obesity & Hypertension

Dr Brij Mohan Makkar, Dr Sanjay Singla, Dr Shubhashree Patil

Recommendations

1	In adults with obesity and hypertension, use home blood pressure monitoring (HBPM)/ ambulatory blood pressure monitoring (ABPM) to detect white-coat/masked patterns. Aim treated systolic blood pressure (SBP) 120–129 mmHg when tolerated.
2	For adults experiencing high normal blood pressure and being overweight, it is advisable to prioritize weight reduction as a means to lower blood pressure and enhance cardiovascular health.
3	Through integrating lifestyle modifications and weight reduction, a significant decrease in hypertension is observed in most instances.
4	In cases where obesity coexists with diabetes and hypertension, it is preferable to consider treatment strategies involving antidiabetic medications that have the dual benefit of reducing both body weight and blood pressure [Glucagon-like peptide-1 receptor agonists (GLP1RA), Sodium-Glucose Cotransporter 2 (SGLT2) inhibitors].

The rise of industrialization, sedentary habits, genetic predisposition, dietary patterns, lack of awareness/ education, and swift urbanization has led to a substantial increase in individuals grappling with overweight or obesity, along with elevated blood pressure and blood glucose levels.[1] According to the ICMR-INDIAB study, the distribution of obesity phenotypes in the Indian population was reported as follows: metabolically obese non-obese (MONO) – 43.3%, metabolically obese obese (MOO) – 28.3%, metabolically healthy non-obese (MHNO) – 26.6%, and metabolically healthy obese (MHO) – 1.8%. Among individuals with metabolic obesity, hypertension is highly prevalent (69.5%), highlighting its importance as a core component and primary treatment focus in obesity and metabolic syndrome management.[2] According to Vasudevan *et al.*, the prevalence of high blood pressure was 35.1% among children aged 10–12 years and 25.1% among those aged 13 years and above, particularly in those with overweight or obesity, who also exhibited associated cardiovascular risk factors.[3]

The Asian Indian phenotype is characterized by a substantial body fat percentage combined with a comparatively lower body mass index (BMI), reduced lean body mass- especially in the lower limbs, and an elevated BF/BMI ratio, indicating greater body fat relative to BMI.[4] This group tends to present a greater body fat percentage (BF%) than other ethnicities, even with lower BMI measurements, a phenomenon commonly known as the Yajnik and Yudkin (Y-Y) paradox.[5] The Asian Indian phenotype encompasses heightened insulin resistance and increased abdominal adiposity, as evident from elevated waist circumference and waist-to-hip ratio. (Table 15) The revised definition of obesity for Asian Indians classifies Stage 1 obesity as increased body fat with a BMI >23 kg/m^2, without any noticeable impairment in organ function or daily activities. Stage 2 obesity requires a BMI >23 kg/m^2, along with at least one of the following: elevated waist circumference or waist-to-height ratio, and evidence of functional limitation or obesity-associated comorbid disease.[6]

Possible pathways interlinking obesity, hypertension, and chronic kidney disease (CKD) are given in Figure 8.

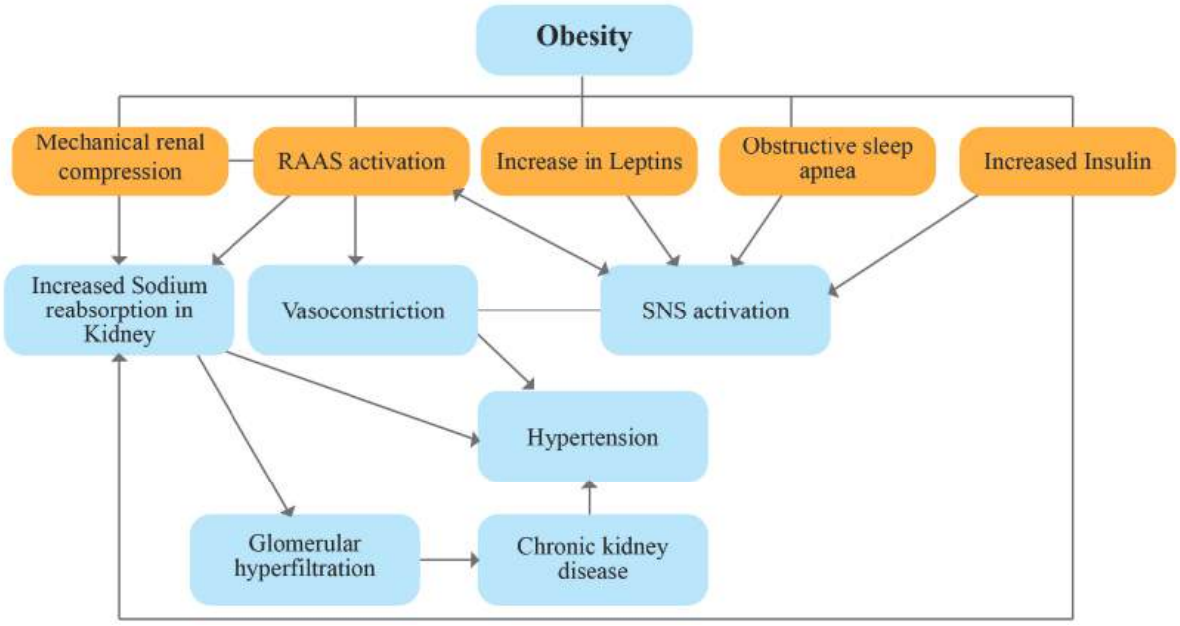

Figure 8: Possible pathways connecting obesity, hypertension, and chronic kidney disease[8-10] (RAAS-Renin-angiotensin-aldosterone system; SNS-sympathetic nervous system).

Table 15: Normal Cut-off values.[4,5,7]

Body Mass Index (BMI) (kg/m²)	**Consensus statement Asian Indians**	**WHO – Asian population**
	Normal BMI: 18.0–22.9	Underweight: <18.5
	Overweight: 23.0–24.9	Normal: 18.5–22.9
	Obesity: >25	Overweight: 23.0–24.9
		Pre-obese: 25.0–29.9
		Obese: ≥30.0
		Type 1 (obese): 30.0–40.0
		Type 2 (morbidly obese): 40.1–50.0
		Type 3 (super obese): >50.0
Waist Circumference (WC) (cm)	**Consensus statement Asian Indians**	**National Family Health Survey (NFHS)- 5**
	Men: >90	Men: >94
	Women: >80	Women: >80
Body Fat Percentage (%)	**Male**	**Females**
	Essential fat: 2–5	Essential fat: 10–13
	Athletes: 6–13	Athletes: 14–20
	Fitness: 14–17	Fitness: 21–24
	Acceptable: 18–24	Acceptable: 25–31
	Obese: ≥25	

Table 16: Obesity & hypertension - Management

PATIENT EDUCATION	Regarding the enduring financial consequences of various conditions associated with obesity and hypertension. Obesity is recognized as a complex, multifactorial, chronic, non-communicable, and relapsing disease.[13,14] Encourage weight loss, adopt a healthy diet, and start a moderate-intensity physical activity program.
LIFESTYLE AND DIETARY MODIFICATIONS	Sufficient consumption of fruits and vegetables, and reduced intake of unhealthy food. Yoga asanas should be encouraged. 60 minutes of physical activity daily or ≥ 300 minutes of weekly, moderate-intensity physical activity. Reduced calorie intake, increased consumption of complex carbohydrates, proteins, fruits, vegetables, berries, pulses, whole grain cereals, and nuts.
PHARMACOTHERAPY FOR WEIGHT LOSS	Indications: BMI ≥ 27 kg/m² without co-morbidities, ≥ 25 kg/m² with co-morbidities, WC is 10 cm above the upper limit of gender-specific normal values. Obese patients with diabetes – Metformin, Sodium-Glucose Cotransporter 2 (SGLT2) inhibitors, Glucagon-like peptide-1 receptor agonists (GLP1RA): (Semaglutide & Liraglutide), dual glucose-dependent insulinotropic polypeptide (GIP) and glucagon-like peptide-1 (GLP-1) receptor agonist: Tirzepatide, Pancreatic lipase inhibitor: Orlistat
PHARMACOTHERAPY FOR HYPERTENSION	Initiate antihypertensive therapy with ACEi, ARBs, or CCBs - they do not worsen insulin sensitivity or adiposity. Thiazide/Thiazide-like diuretics may be used in combination therapy (can worsen glucose and lipid metabolism at higher doses). Beta-blockers promote weight gain, have dyslipidemic effects, and may also increase the risk of type 2 diabetes, but in patients with obesity, hypertension associated with comorbidities such as postmyocardial infarction or Heart failure with reduced ejection fraction (HFrEF), beta-blockers are an indication.
BARIATRIC SURGERY	Indications: BMI of ≥ 32.5 kg/m² with co-morbidities, BMI ≥ 37.5 kg/m² without co-morbidities. Laparoscopic adjustable gastric banding (LAGB), Laparoscopic sleeve gastrectomy (LSG), Laparoscopic Roux-en-Y gastric bypass (LRYGB), Biliopancreatic diversion with duodenal switch (BPDS).

11.3.1 Management

The main objective in managing hypertension linked to obesity is achieving weight loss, as it effectively counteracts the underlying pathophysiological processes contributing to hypertension. The blood pressure-lowering benefits of weight loss exhibit a linear relationship, with an approximate reduction of 1-mmHg per kilogram of weight loss.[11] However, this effect might diminish over extended periods, revealing a decrease of approximately 6 mmHg for every 10 kilograms of weight loss.[12] Lifestyle modifications, pharmacotherapy, and metabolic surgeries are the next effective strategies (Table 16).

A systematic review and meta-analysis showed that semaglutide lowered systolic blood pressure by an average of 4.83 mmHg (95% CI: −5.65 to −4.02) and diastolic blood pressure by 2.45 mmHg (95% CI: −3.65 to −1.24).[15] In the SELECT trial, adults with obesity and established atherosclerotic cardiovascular disease (without diabetes) treated with semaglutide 2.4 mg weekly experienced a 20% reduction in major cardiovascular events, a 3.3 mmHg fall in systolic blood pressure (SBP), and approximately 10% weight loss, supporting its role as an adjunct to standard antihypertensive therapy.[16] Similarly, in a 72-week trial among overweight or obese adults without diabetes, tirzepatide at 5, 10, and 15 mg doses achieved weight reductions of 15%, 19.5%, and 20.9%, respectively, compared with 3.1% with placebo, and produced mean SBP and diastolic blood pressure (DBP) reductions of 6.8 mmHg and 4.2 mmHg, largely attributable to weight loss.[17]

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SECTION 12 – Technology

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Recommendations

1	Using digital devices for blood pressure measurement is encouraged.
2	Home blood pressure monitoring (HBPM) (preferable)/ Ambulatory Blood Pressure monitoring (ABPM) for diagnosis confirmation, titration, and long-term control. Prefer upper-arm, automated, clinically validated devices and standardized measurement protocols. Consider connected telemonitoring with team-based care where feasible.
3	We recommend regular usage of Electronic Health Records (EHRs) as they facilitate quick retrieval of patient records and augment the efficacy of existing treatments.

The future of hypertension management lies in digital solutions and wearable blood pressure monitoring technology. These innovations hold the promise of significantly reducing and possibly even eliminating cardiovascular events in patients with hypertension. Emerging technologies such as Wrist-based Oscillometric Measurement, Information/Communication Technology (ICT) multisensor blood pressure monitoring systems, application trigonometry method, and Photoplethysmography (PPG)

represent exciting prospects for the future of blood pressure monitoring.[1,2] Digital transformation has become a cornerstone in modern hypertension management, bridging the gap between clinical precision and population health delivery.

12.1 Digital Aids

12.1.1 Telemedicine

Telemedicine has the advantage of being fast, easy to access, saving time (no long-distance travel) and efforts of the patient, better documentation, and safety.[3] It can be used as a tool to interactively educate the young and old patients alike. Telemedicine, using trained non-physician health workers equipped with electronic tablets and point-of-care devices, improved blood pressure control in rural areas through remote consultations, benefiting known hypertensive patients. According to a randomized controlled trial, at six months, systolic blood pressure decreased significantly in both groups—from 148.2 ± 7.5 mmHg to 132.6 ± 6.3 mmHg in the telehealth group and from 147.9 ± 8.1 mmHg to 135.4 ± 7.1 mmHg in the in-person group (both $p < 0.01$). The telehealth group achieved a slightly greater reduction in systolic blood pressure, along with higher medication adherence (92% vs 87%, $p = 0.04$) and greater patient satisfaction (4.5 vs 3.9 on a 5-point scale).[4] Electronic health records (EHRs) can significantly improve hypertension control by providing quick access to patient data, optimizing treatment, enhancing patient-physician communication, and promoting preventive care.[5]

12.1.2 Mobile & Mobile Applications

Mobile applications for managing hypertension are becoming popular, offering features like data tracking, personalized guidance, and smartphone interactive communication. However, challenges include a lack of clinical validation, poor quality, data privacy concerns, and limited adoption among elderly populations.[1,6]

According to a quasi-experimental study involving 272 patients with hypertension, participants using a mobile health application demonstrated significantly better outcomes than controls, including improved blood pressure control and greater adherence to healthy lifestyle behaviors. Notably, adherence to the DASH diet was higher among app users compared to non-users (63.2% vs 29.4%, $p < 0.001$).[7]

In the mPower Heart Project in Himachal Pradesh, a nurse-facilitated smartphone or tablet-based clinical decision support system (DSS) significantly reduced systolic (-12.9 to -14.6 mmHg) and diastolic (-7.1 to -7.7 mmHg) blood pressure.[8] In the SIMCARD Trial, community health workers supported by physicians using mobile health technology achieved a 25.5% increase in antihypertensive medication use and a reduction in systolic blood pressure by -2.7 mmHg.[9] While mobile technologies offer clear advantages in hypertension management, emerging evidence also highlights potential adverse effects of excessive use. A study reported a six-fold higher risk of developing hypertension among individuals using mobile phones for over eight years and a four-fold increase among those using them for more than 60 minutes daily.[10]

The India Hypertension Control Initiative (IHCI), implemented across 26 districts in five states, reported an average clinic-level blood pressure control rate of 43% (ranging from 22% to 79%). The proportion of individuals with hypertension who achieved and recorded blood pressure control in public health facilities increased nearly threefold—from 1.4% to 5.0%—over the program period. The IHCI demonstrated that large-scale implementation of protocol-driven hypertension management.[11] "Simple," a quick and cost-free mobile app, was collaboratively developed with IHCI for physicians and officials to handle hypertension and diabetes patients. This Android application empowers doctors to store and oversee vital patient data, such as blood pressure, blood glucose levels, medication schedules, and follow-up appointments.[12,13]

12.2. Artificial Intelligence (AI) driven Approaches

Machine learning (ML) and Artificial Intelligence (AI) amalgamate computer science, statistics, and decision theory to identify intricate patterns within extensive datasets. In hypertension, these technologies enable early prediction, real-time monitoring, and personalized management by combining cardiovascular, multiomics, and sociodemographic data.[14] AI-driven risk stratification models, tailored to community-based screening programs, can improve hypertension detection and management in resource-limited settings. Advanced applications such as digital twin frameworks simulate disease progression to guide individualized therapy, while deep learning models using electrocardiogram (ECG) and PPG signals allow noninvasive blood pressure estimation with high accuracy.[15–17] It is also being applied to optimize antihypertensive drug selection and treatment response prediction.[18] Despite this promise, widespread adoption requires explainable, validated, and ethically governed models to ensure transparency, equity, and patient data protection. Despite this promise, a cautious approach is essential. AI systems must prioritize data security, maintain strict compliance with privacy regulations, and safeguard sensitive health information throughout the data lifecycle. Furthermore, widespread implementation requires models that are explainable, rigorously validated, and ethically governed to ensure transparency, fairness, and responsible use in clinical practice.

12.3. Digital Devices and Sphygmomanometer

Accurately measuring and controlling blood pressure prevents conditions like hypertension, cardiovascular diseases, and strokes.[19] Ensuring precise blood pressure readings, even with an error margin as small as 5 mmHg, holds immense significance, particularly amidst the increasing enigma of hypertensive disorders.[20] There has been a notable shift towards automated blood pressure devices like Aneroid and Digital sphygmomanometers. A key advantage of the aneroid instrument is its portability, while the digital counterpart excels in user-friendliness, reducing the need for the examiner's auscultatory skills.[21] The blood pressure

measured by mercury, aneroid, and digital sphygmomanometers are comparable, but aneroids are considered more sensitive and specific when compared to digital apparatus.[21] (Refer Section 6: Blood pressure measurement).

Cuffless wearable blood pressure devices—using sensors like PPG, tonometry, bioimpedance, or hybrid systems- offer the promise of continuous, noninvasive blood pressure monitoring during daily life without the discomfort of cuffs.[22] Despite progress, most wearable cuffless devices remain in development or early validation stages, with limited adoption in routine clinical practice due to the need for standardized validation protocols, user calibration, motion artifact correction, and regulatory oversight.[23]

12.4. Barriers to implementing digital technology interventions to improve hypertension management in the public healthcare system in India

Barriers to implementing digital technology interventions to improve hypertension diagnosis & management in India include a lack of healthcare infrastructure, a shortage of trained healthcare professionals, a lack of technology literacy, data privacy & security concerns, affordability, and resistance to change.[24,25] Addressing these barriers requires a multi-faceted approach involving collaboration between stakeholders, including state and national governments, healthcare institutes, and healthcare device manufacturers.

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SECTION 13 - Pediatric & Adolescent Hypertension

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Recommendations

1	Blood pressure levels should be evaluated in children from three years old.
2	Screening for blood pressure in children under the age of three is advised when there is Coarctation of the Aorta (COA) or other risk factors for elevated blood pressure, such as chronic kidney disorders (contracted kidney, nonfunctioning kidney), solid organ transplantation, use of blood pressure-raising medications, a history of preterm birth, and other relevant factors.
3	Blood pressure can be measured using the auscultatory method (aneroid sphygmomanometer) or with validated, regularly calibrated oscillometric devices. However, any elevated reading obtained by an oscillometric device should be confirmed using the auscultatory method (multiple measurements), as it provides greater accuracy and is more reliable for predicting target organ damage.
4	In children with obesity, diabetes, chronic kidney disease (CKD), a history of prematurity, or discordant clinic readings, 24-hour ambulatory blood pressure monitoring (ABPM) should be used to confirm the diagnosis and detect masked or white-coat hypertension. Contemporary pediatric data show that home blood pressure monitoring (HBPM) alone misses masked hypertension and may misclassify white-coat hypertension; ABPM remains the preferred diagnostic modality where available.

Neonatal hypertension occurs in approximately 0.2% of term newborn infants and can affect up to 3% of infants admitted to the Neonatal Intensive Care Unit (NICU).[1] Various systematic reviews and meta-analyses have consistently reported a prevalence ranging from 5.54% to 7.6% in children and adolescents below the age of 18 years in India.[2-4] A secondary analysis of data obtained from the Comprehensive National Nutrition Survey found that the prevalence of high blood pressure was 35.1% among children aged 10 to 12 years and 25.1% among those aged 13 years and older.[5] Another systematic review estimated the prevalence of hypertension among adolescents in South Asia to be 13.77%.[6] All the studies point towards a high prevalence of hypertension in apparently healthy children. Table 17 shows the definition and classification of pediatric and adolescent hypertension.

The Indian Academy of Pediatrics (https://pubmed.ncbi.nlm.nih.gov/17351301/) has given a screening tool to identify children and adolescents who need further blood pressure assessment through repeated measurements. It should not be solely relied upon for diagnosing elevated blood pressure or hypertension; accurate diagnosis requires reference to the specific cutoff values outlined in the comprehensive blood pressure classification tables.[7] This is imperative due to the potential variation in systolic blood pressure (SBP) and diastolic blood pressure (DBP) cutoffs, which can be elevated by up to 9 mmHg based on the individual's age and height.[7]

Neonates - Defining hypertension in neonates (average blood pressure is 64/41 mmHg) is notably challenging due to the widely recognized fluctuations in blood pressure that transpire during the initial weeks of life.[10] These fluctuations can be particularly pronounced in preterm infants, wherein blood pressure is influenced by many factors encompassing postmenstrual age, birth weight, and maternal health conditions.[11] Refer to Table 18 for Neonatal blood pressure values. Direct intra-arterial measurements using indwelling catheters and indirect measurements using the oscillometric technique (In-office method) are the two methods for measuring blood pressure in hospitalized neonates.[7]

Table 17: Definition and classification of Pediatric & Adolescent Hypertension[7–9]

Blood pressure/ Hypertension	Age 1 – <13 years	Age > 13 years
Normal Blood Pressure	<90th percentile	< 120/< 80 mmHg
Elevated Blood Pressure	≥90th percentile to <95th percentile or 120/80 mmHg to <95th percentile (which-ever is lower)	120/< 80–129/< 80 mmHg
Stage 1 Hypertension	≥95th percentile to <95th percentile + 12 mmHg or 130/80–139/89 mmHg (whichever is lower)	130/80–139/89 mmHg
Stage 2 Hypertension	≥95th percentile + 12 mmHg	≥140/90 mmHg
	or ≥ 140/90 mmHg (whichever is lower)	
Hypertensive Crisis		
Hypertensive Urgency	>95th centile + 30 mmHg without symptoms/signs of target end organ damage	>180/120 without symptoms/signs of target end organ damage.
Hypertensive emergency	>95th centile + 30 mmHg associated with encephalopathy (headache, vomiting, vision changes, and neurological symptoms (facial nerve palsy, lethargy, seizures, coma) +/- target-end organ damage).	>180/120 associated with encephalopathy (headache, vomiting, vision changes, and neurological symptoms (facial nerve palsy, lethargy, seizures, coma) +/- target-end organ damage)

Table 18: Reference values for Neonatal blood pressure values.[12]

Postmenstrual Age		50th Percentile	95th Percentile	99th Percentile
44 weeks	Systolic	88	105	110
	Diastolic	50	68	73
	Mean	63	80	85
42 weeks	Systolic	85	98	102
	Diastolic	50	65	70
	Mean	62	76	81
40 weeks	Systolic	80	95	100
	Diastolic	50	65	70
	Mean	60	75	80
38 weeks	Systolic	77	92	97
	Diastolic	50	65	70
	Mean	59	74	79
36 weeks	Systolic	72	87	92
	Diastolic	50	65	70
	Mean	57	72	71
34 weeks	Systolic	70	85	90
	Diastolic	40	55	60
	Mean	50	65	70
32 weeks	Systolic	68	83	88
	Diastolic	40	55	60
	Mean	48	62	69
30 weeks	Systolic	65	80	85
	Diastolic	40	55	60
	Mean	48	65	68
28 weeks	Systolic	60	75	80
	Diastolic	38	50	54
	Mean	45	58	63
26 weeks	Systolic	55	72	77
	Diastolic	30	50	56
	Mean	38	57	63

Essential or primary hypertension - where an underlying cause cannot be identified in children above ≥6 years.[13] Risk factors for primary hypertension include - overweight/obesity, suboptimal diet (ultra processed foods, high sodium diet, consumption of fewer fruits and vegetables), physical inactivity, poor sleep, environmental factors (air pollution, exposure to phthalates, compounds commonly found in plastics, etc.), hereditary, and low birth weight/intrauterine growth restriction, low socioeconomic status and food insecurity, tobacco use and males.[14–16]

Secondary hypertension is caused by an underlying disorder or the use of certain medications (Refer to sections 11.0.2 & 11.2). More prone to manifest at a younger age.[17]

13.1 Blood Pressure Measurement

Normative values for blood pressure are established through auscultatory sphygmomanometry, which remains the preferred technique for blood pressure assessment.[18,19] (Refer to Figure 9). Invasive intra-arterial blood pressure monitoring (direct arterial catheter) is the gold standard, providing the most reliable readings, especially in critically ill neonates. For most infants without invasive lines, non-invasive automated oscillometric devices (cuff width to arm ratio is 0.5) are commonly used.[20] Measurements should be taken on the right upper arm using an oscillometric device, ideally 15 minutes after cuff placement and at least 1.5 hours after feeding, while the infant is calm. Three readings should be recorded at two-minute intervals.[21]

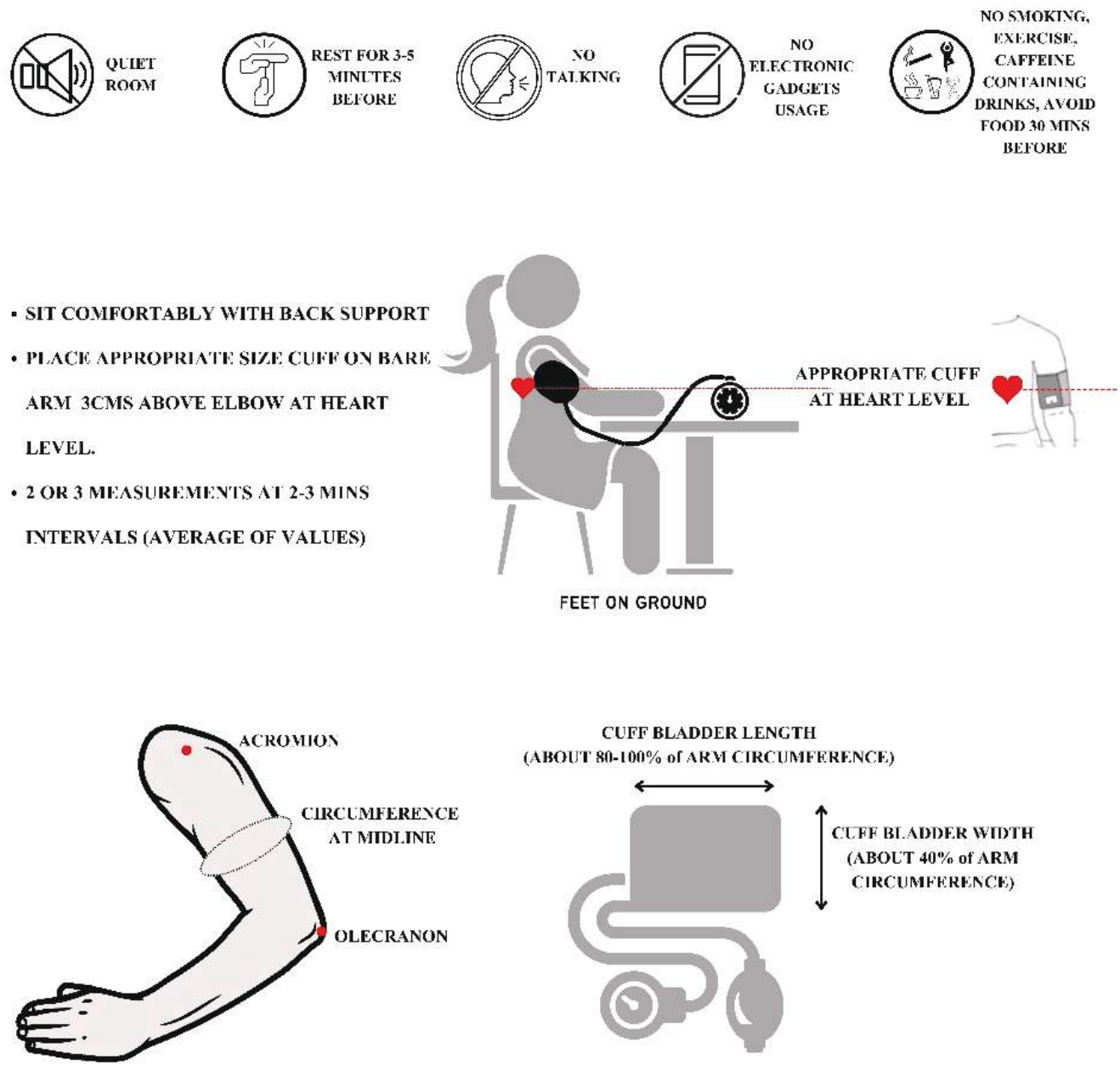

Figure 9: Blood Pressure measurement in children

Oscillometric devices tend to overestimate both systolic and diastolic blood pressure readings. Ambulatory blood pressure monitoring (ABPM) devices are being utilized more frequently as they capture blood pressure readings outside of the clinical setting over an extended period. Based on current data, ABPM is superior to office/clinic-measured blood pressure in diagnosing hypertension and is more predictive of future blood pressure trends. (Table 19)

13.2 Screening

Diagnosing hypertension in children often goes unnoticed due to the absence of symptoms or the inability to express discomfort. In cases with no identifiable underlying cause, blood pressure should be assessed annually starting from the age of 3.[23] However, when any risk factors are present—such as prematurity, <32 week's gestation, small for gestational age or low birth weight, history of umbilical artery line, congenital heart disease, recurrent urinary tract infections, or abnormal urinalysis, known renal disease or urologic malformations, family history of renal disease, history of solid-organ transplant, malignancy or bone marrow transplant, treatment with medications known to increase BP, a BMI ≥ 95th percentile, diabetes, aortic arch obstruction or coarctation—blood

Table 19: Phenotype classification based on ABPM and office blood pressure readings[22]

Category	Clinic SBP or DBP	Mean ambulatory SBP or DBP
Normal BP	<95th percentile 9 (<13 years) <130/80 (≥13 years)	95th percentile or adolescent cut points* (<13 years) <125/75 mmHg 24-h and <130/80 mmHg wake and <110/65 mmHg sleep (≥13 years)
White coat hypertension	≥95th percentile (<13 years) ≥130/80 (≥13 years)	
Masked hypertension	<95th percentile (<13 years) <130/80 (≥13 years)	≥95th percentile or adolescent cut points* (<13 years) ≥125/75 mmHg 24-h or ≥130/80 mmHg wake or ≥110/65 mmHg sleep (≥13 years)
Ambulatory hypertension	≥95th percentile (<13 years) ≥130/80 (≥13 years)	

pressure measurements should be taken during every clinic visit, regardless of the child's age.[13,17,19]

13.3 Examination

Medical history, conducting a thorough clinical examination (Table 20), performing relevant laboratory tests, and radiological evaluations are the key steps in diagnosing hypertension (Table 21). Initiating the clinical examination involves the measurement of fundamental parameters, including weight, height, and body mass index (BMI). These measurements provide essential baseline data for assessing the child's growth and overall health status.

Table 20: History and Clinical examination.[13,19,24]

History	*Birth or Antenatal history* - Maternal history of hypertension, low birth weight, gestational age, etc.
	Family history - Hypertension, ischemic heart disease, familial hyperlipidemia, diabetes, sudden cardiac death, hereditary renal or endocrine syndromes (<55 years).
	Family Structure - Nuclear/ joint/both parents working. May determine ease of following dietary or activity advice given.
Environmental factors	Smoking, alcohol consumption, drug/substance intake.
Diet	Daily intake of high-sodium, high-fat, ultra-processed junk food, caffeine-containing drinks, and gym formulations
Physical activity	Physical exercise/leisure time
Screen time	Time spent on mobile phone/ laptop/ tablet/television (Inversely proportional to physical & mental activity level)
Sleep	*Obstructive sleep apnea* - Sleeplessness, snoring, daytime sleepiness
Symptoms	Headache, epistaxis, vertigo, visual impairment, strokes, low school performance, attention defects, dyspnoea, chest pain, palpitations, and syncope.
Physical examination	*General* - Stunted growth, pallor, edema, obesity
	Endocrine - cushingoid features, dysmorphology
	Skin - Rash, vasculitis, axillary freckling, acne, acanthosis nigricans, xanthelasma, xanthomas, café au lait spots
	Coarctation of Aorta (COA), Aortoarteritis, hyperthyroidism - Pulse rate and volume in both upper and lower limbs
	Thyroid - Enlargement
	Cardiovascular - Apical heave, murmur, bruits (LVH), weak femoral pulses, tachycardia
	Genitalia - Virilization (Congenital adrenal hyperplasia)
	Renal - Bruit over flanks (renal artery), Renal mass
	Joints - Arthritis, in certain autoimmune causes of glomerulonephritis
	Abdomen - Check for masses, hepatosplenomegaly

Table 21: Investigations.[13,25,26]

Blood chemistry	Complete haemogram, Electrolytes (serum sodium, potassium, chloride levels), thyroid function tests, cortisol, and aldosterone. Plasma renin activity, plasma and urine catecholamines and metanephrines, serum 11-deoxycortisol and 11-deoxycorticosterone, urinary 17-hydroxysteroid and 11-deoxycorticosterone.
	Renal disease- serum blood urea nitrogen, serum creatinine, estimated glomerular filtration rate (eGFR).
	Cardiovascular disorders - Lipid profile (especially Cardiolipins).
	Obesity - fasting blood sugar, HbA1c, serum alanine aminotransferase, serum aspartate aminotransferase (AST)
Bilateral upper arm and single leg blood pressure measurement	To rule out COA and aortoarteritis
Drug screening	To rule out substances causing an increase in heart rate & blood pressure.
Urine analysis	Routine and microscopic examination
Echocardiography	Cardiac organ damage, left ventricular hypertrophy (LVH)
Retinal fundus examination	To rule out fundal edema and hemorrhages.
Renal Doppler and Ultrasound	To assess underlying renal disease.
Computed Tomography (CT) and Magnetic Resonance Angiography (MRA)	Mass, complete visualization of the aorta and branches, including renal vessels.
Polysomnography	Obstructive sleep apnea
Genetic testing	To test a few secondary causes (pheochromocytoma, etc.)

13.4 Management

The objectives for managing pediatric hypertension should encompass preventing damage to target organs and the development of adult hypertension. (Figure 10) The whole family has to be involved to adapt to the lifestyle changes.

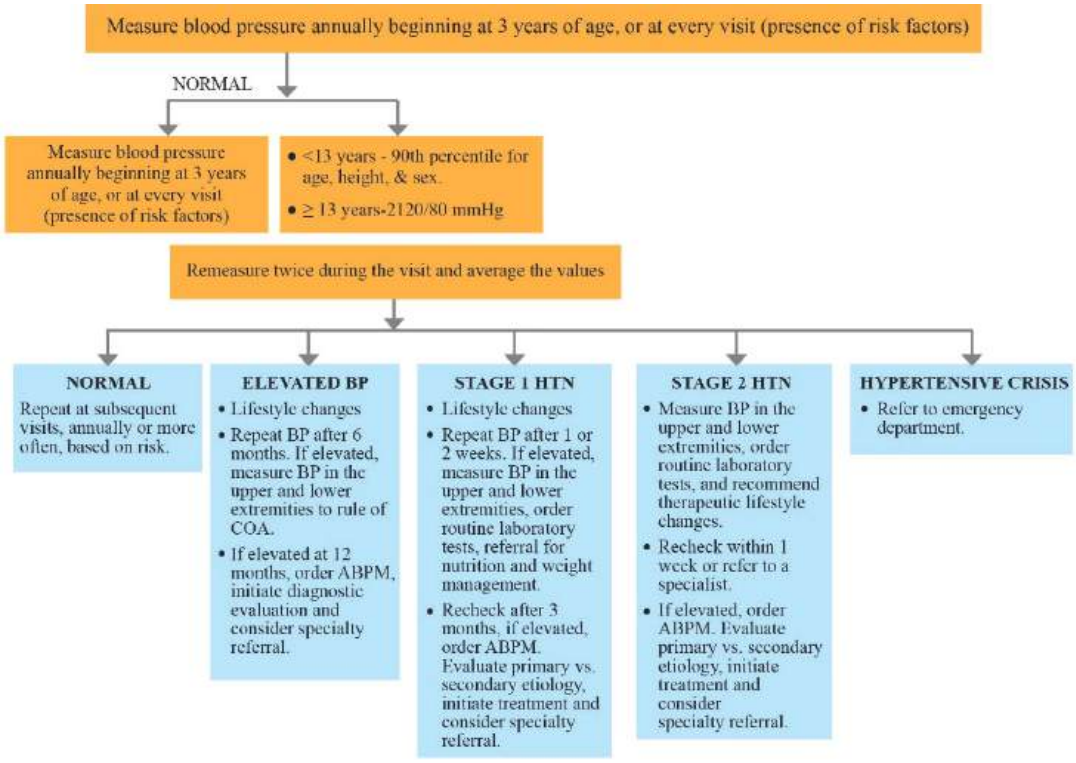

Figure 10: Algorithm for management of elevated BP in children and adolescents.

Table 22: Pharmacological management.[20,25,30,31]

DRUG	DOSE RANGE (INITIATING TO MAXIMUM)	DOSES PER DAY	REMARKS
FIRST LINE ORAL ANTI-HYPERTENSIVE FOR PEDIATRIC HTN			
Enalapril	0.08 to 0.6 mg/kg/dose	Twice	Age > 1 month (max dose range: 5 mg to 40 mg/day)
Lisinopril	0.07 to 0.6 mg/kg/dose	Once	Age > 6 years (max dose range: 5 mg to 40 mg/day)
Ramipril	1.6 to 6 mg/m^2/dose	Once	Max dose in adults: 2.5 mg to 20 mg/day. Has been used in children above 18 months of age.
Candesartan	0.2 mg/kg/dose	Once	Age 1-6 years (max dose range: 0.4 mg/kg up to 4 mg/day)
	< 50 kgs - 4-8 mg (max dose - 16 mg) > 50 kgs - 8-16 mg (max dose - 32 mg)	Once	Age 6 - 17 years
Olmesartan	10 to 20 mg/dose	Once	Age > 6 years, weight < 35 kg
	20 to 40 mg/dose	Once	Age > 6years, weight >35 kg
Losartan	0.7 to 1.4 mg/kg/dose	Once	Age > 6 years (max dose range: 50 to 100 mg/day)
Valsartan	0.4 to 3.4 mg/kg/dose	Once	Age: 1-5 years and weight > 8 kg Max dose: 40 mg (< 18 kg) to 80 mg (> 18 kg /day)
	1.3 to 2.7 mg/kg/dose	Once	Age: > 6 years Max dose range: 40 to 160 mg/day
Hydrochlorothiazide	0.5 to 1 mg/kg/dose	Twice	Max dose range: 25 to 75 mg/day
Chlorothiazide	5 to 10 mg/kg/dose	Twice	Max dose for age <2 years: 375 mg/day Max dose for 2-12 years: 1000 mg/day Max dose for > 12 years: 2000 mg/day
Amlodipine	0.1 to 0.6 mg/kg/dose	Once	Age 1 to 5 years. (max dose 5 mg/day)
	2.5 to 10 mg/dose	Once	Age > 6 years. (max dose 10 mg/day)
Nifedipine (extended-release)	0.2 to 0.5 mg/kg/dose	Once or Twice	Max dose: 3 mg/kg/day - 120 mg/day
SECOND-LINE ORAL ANTI-HYPERTENSIVE FOR PEDIATRIC HTN			
Atenolol	0.5 to 1 mg/kg/dose	Once or Twice	Max dose: 2 mg/kg/day up to 100 mg/day
Metoprolol	1 to 2 mg/kg/dose	Twice	Max dose: 6 mg/kg/day up to 200 mg/day
Metoprolol (extended-release)	1 to 2 mg/kg/dose	Once	Max dose range: 50 to 200 mg /day
Propranolol	1.0 mg/kg/dose	Twice or Thrice	Max dose: 8mg/kg/day up to 640 mg/day
Labetalol	2 to 3 mg/kg/dose	Twice	Max dose: 10 - 12 mg/kg/day up to 1.2 g/day
Prazosin	0.02 to 0.15 mg/kg/dose	Thrice	Max dose: 2 to 20 mg/day
Clonidine	5-10 mcg/kg/dose	Twice or Thrice	Max dose: 25 mcg/kg/day up to 0.9 mg/ day
HYPERTENSION EMERGENCIES & URGENCIES (PARENTERAL)			
DRUG	**DOSE**		**REMARKS**
Nicardipine infusion	0.5 to 4 mcg/kg/min		Reflex tachycardia is a side effect. It can be used even in infants.

Labetalol infusion	0.25 to 3 mg/kg/hour	Bolus or infusion is contraindicated in asthma and frank heart failure
Labetalol bolus	0.2 to 1 mg/kg/dose Max 40 mg/dose	Can be repeated every 10 minutes.
Sodium Nitroprusside infusion	0.5 to 3 mcg/kg/min Max dose 10 mcg/kg/min	Avoid in chronic renal disease
Hydralazine bolus	0.1–0.2 mg/kg/dose. Max 0.4 mg/kg/dose	The onset of action is slower. Can be repeated every 4 hours. It can be given intramuscularly as well. Tachycardia is a side effect. It can be used in infants.

13.4.1 Lifestyle modifications

- Children should follow the DASH (Dietary Approach to Stop Hypertension) diet, which includes minimally processed foods, whole grains, fresh legumes, vegetables, fruits, low-fat dairy, fish, and lean red meat. This diet reduces refined sugar and avoids excessive salt, sugar, and refined white wheat flour. The goal is to limit daily sodium intake (Age 1–3 years: < 1200 mg per day; Age 4–8 years: < 1500 mg per day; Age 9–13 years: < 1800 mg per day; Age 14–18 years: < 2300 mg per day).[27] A potassium-rich diet is recommended instead of high sodium intake. Coffee, gym formulations, caffeinated, carbonated, and energy drinks (including sugar-free drinks), and junk food should be avoided.

Table 23: Management of Neonatal hypertension.[10,21,33]

DRUG	DOSE	REMARKS
Amlodipine	PO 0.1–0.5 mg/kg/day once daily	Peripheral edema
Hydralazine	PO 0.25–1 mg/kg/ dose every 6-8 hours. Maximum 7.5 mg/kg/day	Tachycardia
Chlorothiazide	PO 20–40 mg/kg/ day every 12 hours	Monitor for hypercalcemia & electrolyte abnormalities
Spironolactone	PO 1–3 mg/kg/day every 12–24 hours	Monitor for hyperkalemia and renal failure
Labetalol	PO 0.5 to 1 mg/kg/dose BD/TDS	Use with caution in bronchospastic disease and congestive heart failure.
Propranolol	PO 0.25 mg/kg/dose every 6 to 8 hours. Maximum 5 mg/kg/day PO	Bradycardia, hyperkalemia, Use with caution in bronchospastic disease.
Captopril	PO <3 months: 0.01 to 0.5 mg/kg/dose and a maximum dose of 2 mg/kg/day, TDS/QID	Hyperkalemia
Lisinopril	PO 0.07–0.1 mg/kg/dose once daily. Maximum 0.5 mg/kg/day PO	Avoid in preterm infants Use with caution in decreased renal function.
INTRAVENOUS		
Nicardipine	0.5–2 mcg/kg/dose infusion	Edema & Tachycardia
Sodium Nitroprusside	Initial 0.2 mcg/kg/min infusion. Not exceeding 10 mcg/kg/min	Cyanide and thiocyanate toxicity, renal failure
Hydralazine	0.1–0.5 mg/kg/dose every 6–8 hours, not exceeding 2 mg/kg/dose	Monitor for tachycardia & edema.
Labetalol	0.1–1 mg/kg/dose every 4–6 hours or 0.25–3 mg/kg/hour infusion	Use with caution in bronchospastic disease and congestive heart failure.
Esmolol	Term infant 0–7 days of life: 50 mcg/kg/min. In 8–28 days old - Start with 75 mcg/kg/min. Do not exceed 500 mcg/kg/min	Hyperkalemia and bronchospasm
Enalaprilat	5–10 mcg/kg/dose every 8–24 hours	Do not use in preterm infants and if GFR < 30 mL/min. Monitor serum electrolytes and creatinine levels.
Propranolol	0.01 mg/kg/dose every 6-8 hours. Maximum 0.15 mg/kg/dose every 6–8 hours	Bradycardia

- Weight reduction
- Physical activity - Promote outdoor activities that involve moderate to vigorous physical activity for 40–60 minutes, at least 3 to 5 times per week. Decrease the time spent on sedentary video games, reduce screen time (< 2 years: avoid all forms of screen exposure, 2–<5 years: limited use, no more than 1 hour per day and under adult supervision, 5–10 years: less than 2 hours per day)[28], and ensure age-appropriate sleep duration.
- Stress reduction (Yoga & meditation), avoiding smoking and alcohol use.[13,29]

13.4.2 Pharmacological interventions

The decision to start pharmacological therapy is advised when signs or symptoms linked to hypertension, hypertensive organ damage (HMOD), stage 2 HTN, concurrent comorbidities, or resistance to lifestyle changes are present. The recommended first-line antihypertensive agents are angiotensin-converting enzyme inhibitors (ACEi) (but contraindicated in renal artery stenosis), angiotensin receptor blockers (ARB), calcium channel blockers (CCB), and thiazide diuretics (Beta blockers are avoided due to side effects). Initiation with a low dose is preferable; if ineffective, a single full dose or low-dose combination is considered. If needed, a full-dose combination is preferred (Table 22).[29] If a child remains consistently hypertensive despite being on three different antihypertensive medications (resistant hypertension), a trial of spironolactone may be considered.[7,29]

Neonates

In neonatal hypertension, addressing correctable causes usually resolves the condition. Blood pressure should be monitored in neonates with risk factors such as: prematurity, very low birth weight, bronchopulmonary dysplasia, renal/urinary tract abnormalities, umbilical arterial catheterization, congenital heart disease, and extracorporeal membrane oxygenation (ECMO).[10] Mild hypertension (>95th to <99th percentile) can be monitored, with treatment options if needed. For moderate hypertension (≥99th percentiles with no end-organ damage), CCBs, vasodilators, and diuretics. ACEi should be avoided in preterm infants below 40–42 weeks postmenstrual age because they may impair nephron development.[32] Severe hypertension (>99th percentile with evidence of end-organ damage) requires cautious IV drug infusion for treatment. Surgical intervention is considered in specific cases, such as coarctation of the aorta or renal artery obstruction, etc. (Table 23).[33]

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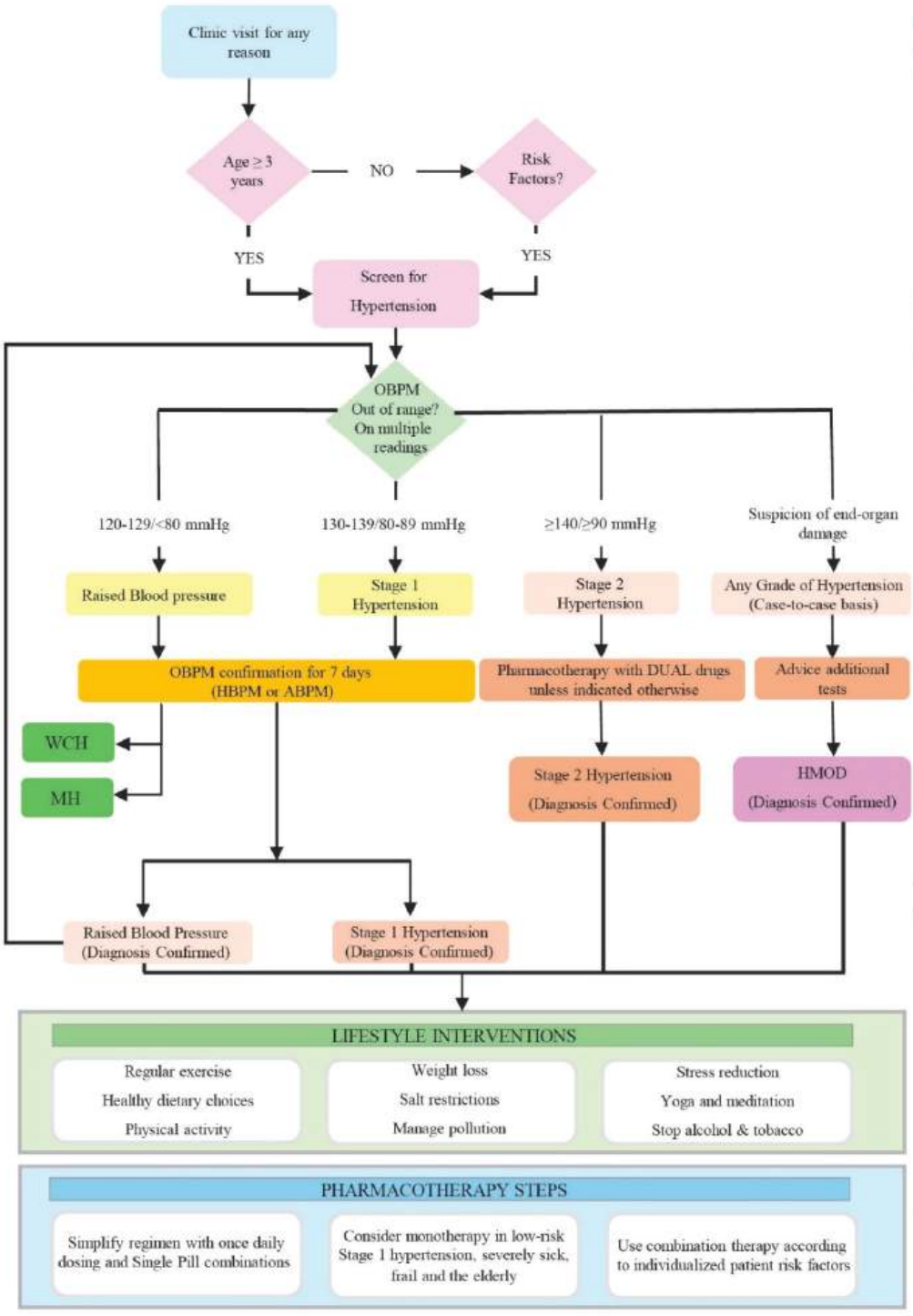
SECTION 14 - Hypertension Management at A Glance

Dr. A K Virmani, Dr. Ajoy Tewari, Dr. Amit Gupta



History and Physical Examination

- Chronic illnesses
- Ongoing treatments
- CV Risk factors
- Evaluate for organ damage
- Symptoms/signs of secondary hypertension
- Check adherence

Laboratory Tests

Essentials

Renal Tests

- Microscopic urinalysis
- Albumin excretion
- Serum BUN and/or creatinine

Endocrine Tests

- Serum sodium
- Serum potassium
- Serum calcium
- TSH

Metabolic Test

- HbA1c
- Total cholesterol
- HDL and LDL cholesterol

Additional

On a case-by-case basis

- Complement levels and autoimmune profile such as hsCRP, LP(a), 24 hour urinary fractionated metanephrines and non metanephrine
- 24-hour urinary free cortisol or low-dose dexamethasone suppression test
- Fundus examination

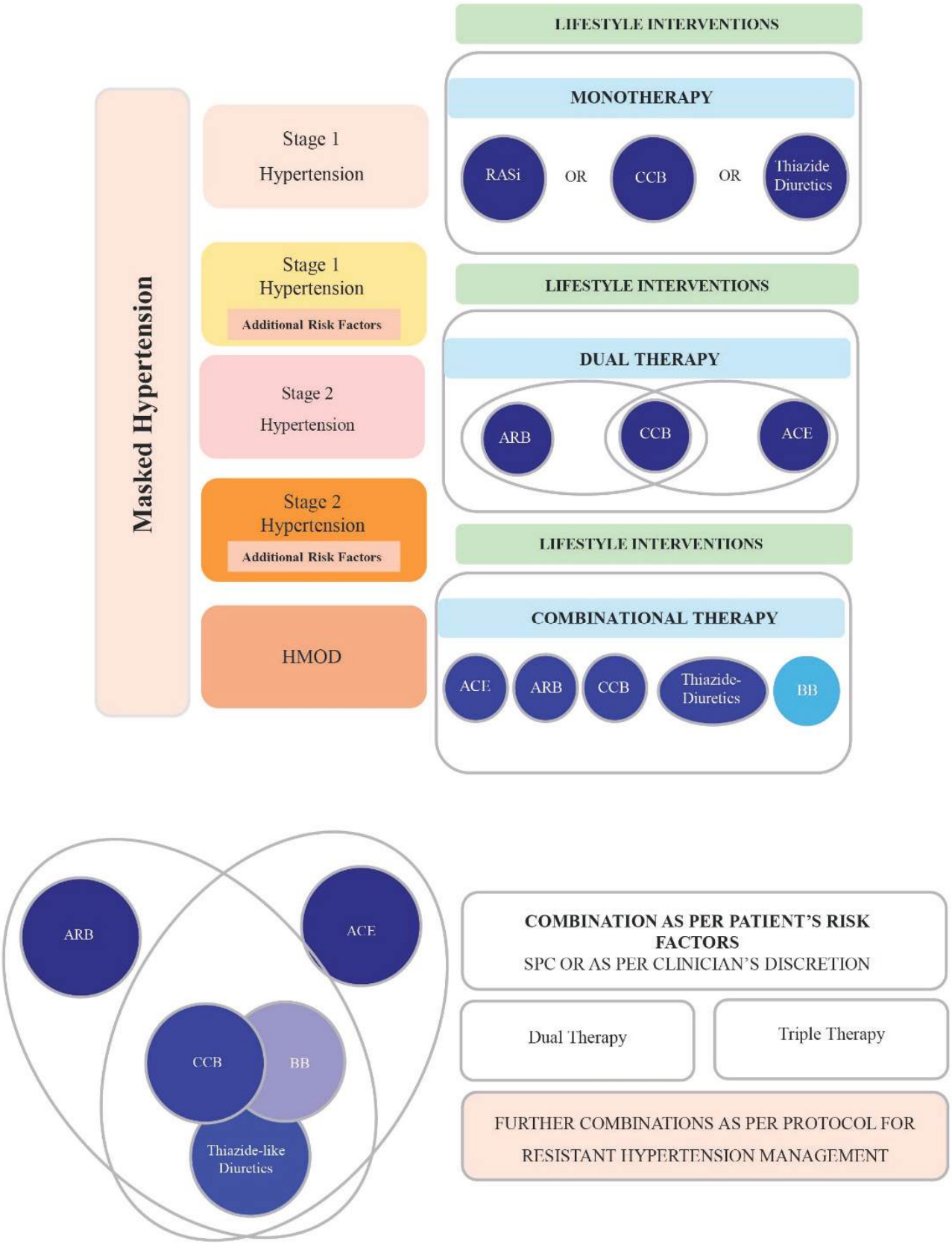


Figure 11: Hypertension management at a glance

Maheshwari A, Verma N, Narasingan SN, Tripathi K, Rajeev Gupta R, Joshi S, Manoria PC. Clinical Practice Recommendations Review Committee. Hypertens J. 2025;11(4):76-138