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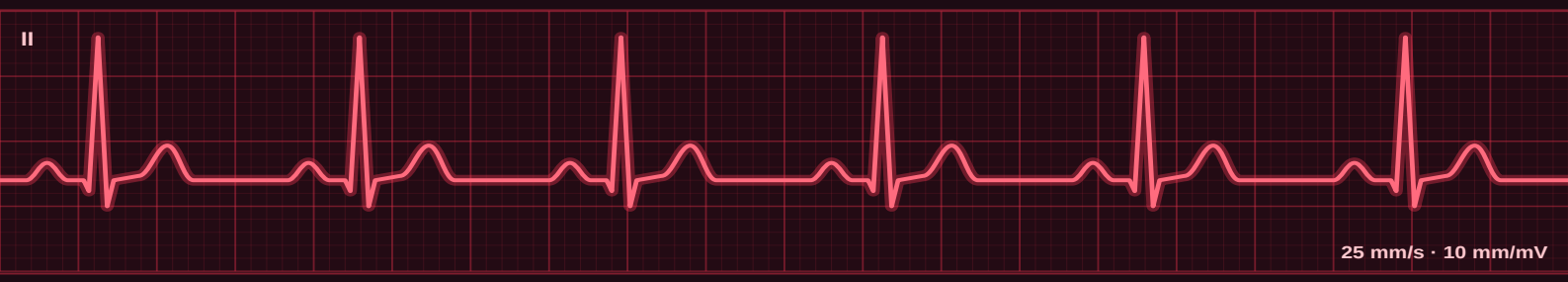
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Hypertension Management

Definitions, Diagnosis & Drug Therapy — for India

How to measure BP correctly, classify and evaluate, choose and step up drugs with full dosing tables, and manage complications and associated diseases — diabetes, heart disease, proteinuria, CKD, stroke and retinal damage

BP measurement technique · ACC/AHA 2025 · ESC 2024 · ESH 2023 · Primary & secondary hypertension · Initial evaluation · Lifestyle modification · First-line drugs & step-up · Master drug table · IHCI protocol · Diabetes · Cardiac · CKD · Proteinuria · Retinopathy · Hypertensive emergencies · Pregnancy & special groups



AETCM
Academy for Emergency, Trauma & Critical care Medicine

By

Dr. Gireesh Kumar K.P.

For general practitioners, physicians, emergency doctors, postgraduates, interns, nurses and MBBS students

Hypertension Management

Definitions, Diagnosis and Drug Therapy — A Practical Guide for India

Measurement · Classification · Primary and secondary hypertension · Initial evaluation
Lifestyle modification · First-line drugs and step-up therapy · Complete drug dosing tables
Complications · Associated diseases · Emergencies · Special populations



Dr. Gireesh Kumar K.P.
Academy for Emergency, Trauma & Critical care Medicine (AETCM)
First Edition · October 2026

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Preface

Hypertension is the most common condition seen in adult practice in India and the most important preventable cause of stroke, heart attack, heart failure and kidney failure. Yet only about one in seven Indians with high blood pressure has it under control. The reasons are familiar to every practising doctor: the disease is silent, measurement is often hurried, treatment is started late or at too low an intensity, and patients stop their tablets when they feel well.

The science of hypertension has moved quickly. Within the last two years we have new guidelines from the American College of Cardiology and American Heart Association (2025), the European Society of Cardiology (2024) and the European Society of Hypertension (2023). Large trials such as SPRINT, STEP, ESPRIT and BPROAD have shown the benefit of lower targets; single-pill combinations have become the standard way to start treatment; and SGLT2 inhibitors, finerenone and renal denervation have changed the care of patients with diabetes, kidney disease and resistant hypertension. At the same time, the India Hypertension Control Initiative has shown that a simple, standard protocol delivered by a team can control blood pressure for millions of people in primary care.

This book brings these developments together in a practical, page-by-page form for the doctor at the bedside and in the outpatient clinic. It begins with the physiology and the definitions, explains **exactly how to measure blood pressure**, how to classify it and how to evaluate a new patient. It then sets out **lifestyle advice** that works for Indian patients, the **first-line drugs**, a **step-by-step guide to adding the next drug**, and a **complete table of every antihypertensive drug with initial and maximum doses, adverse effects and cautions**. Separate chapters deal with the complications of hypertension and with its management in patients with diabetes, heart disease, proteinuria, chronic kidney disease, stroke and retinal damage, followed by hypertensive emergencies, resistant hypertension, pregnancy, children and the elderly.

Tables, flow charts, key points and clinical pearls are used throughout so that the answer can be found in seconds. Drugs are listed by generic name, and doses are those used in adult practice in India. A patient handout and home BP log are included for photocopying.

I hope this book helps general practitioners, physicians, emergency doctors, postgraduates, interns, nurses and students to diagnose hypertension earlier, treat it more confidently and keep it controlled for life.

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October 2026

How to use this book

If you need to…	Go to
Measure BP correctly, choose a cuff, use home or ambulatory BP	Chapter 7
Classify a reading or compare guidelines	Chapter 4
Decide which tests to order for a new patient	Chapter 9
Decide when to start drugs and how low to go	Chapter 13
Choose the first drug and add the next	Chapters 15–17
Look up a dose, maximum dose or side-effect	Chapter 18 (master table) and Appendix A
Treat hypertension with diabetes, heart disease, CKD, proteinuria or retinopathy	Chapter 19
Manage a hypertensive emergency	Chapter 20
Treat pregnant women, children or the elderly	Chapter 22

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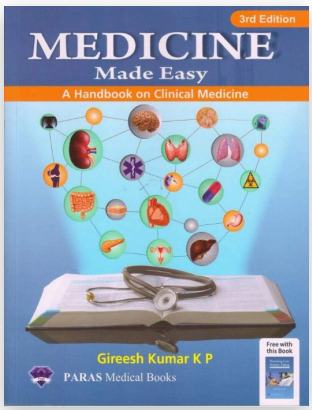
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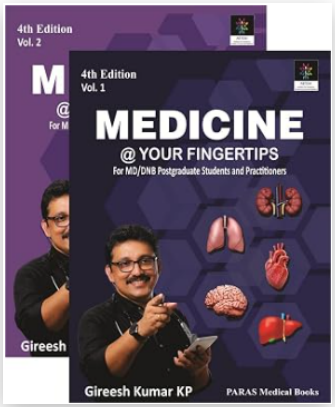
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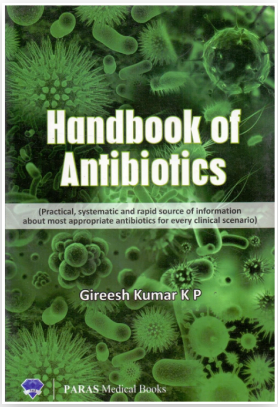
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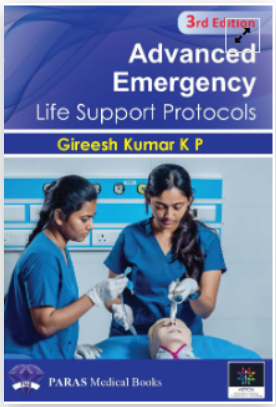
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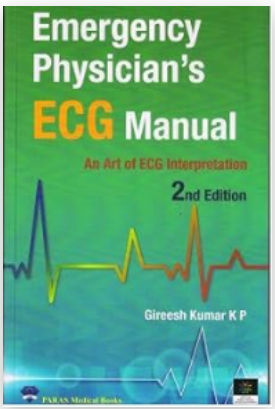
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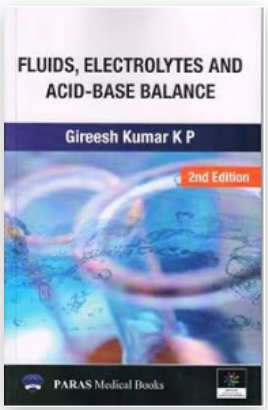
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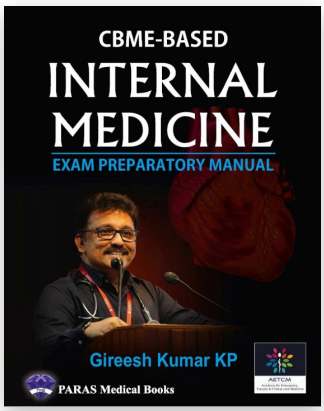
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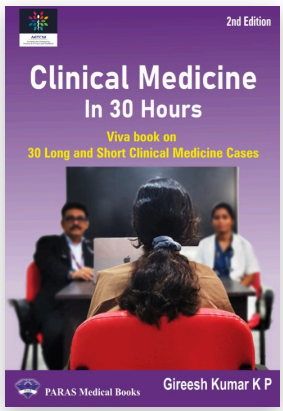
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Hypertension at a Glance

The whole book on one page — for the busy clinic.

1 · Define

- Office BP ≥140/90 on ≥2 visits (ACC/AHA: ≥130/80)
- Home BP ≥135/85 · 24-h ABPM ≥130/80
- Grade 1: 140–159/90–99 · Grade 2: 160–179/100–109 · Grade 3: ≥180/≥110

2 · Measure correctly

- 5 min rest, seated, back and arm supported, feet flat, no talking
- Correct cuff size; cuff at heart level; bare arm
- 2–3 readings 1 min apart; both arms at first visit; standing BP in elderly
- Confirm with home BP or ABPM

3 · Evaluate

- Urine dipstick, UACR, creatinine/eGFR, Na^+, K^+, glucose/HbA1c, lipids, Hb, ECG
- Fundus in grade 2–3/diabetes; echo if indicated
- Look for secondary causes if young, severe, resistant, low K^+
- Stratify total CV risk

4 · Lifestyle for all

- Salt <5 g/day (1 teaspoon); more fruit and vegetables
- Weight loss; 30 min brisk walk 5 days/week
- Stop tobacco; limit alcohol; sleep 7–8 h

5 · Drugs — step up every 2–4 weeks

- **Step 1:** ACEi/ARB + CCB (or + thiazide-like) — single pill
- **Step 2:** ACEi/ARB + CCB + thiazide-like
- **Step 3:** + spironolactone; refer
- β-blocker at any step if indicated
- Never ACEi + ARB; no ACEi/ARB in pregnancy

6 · Targets

- Most: <130/80 (SBP 120–129 if tolerated)
- ≥80 years: SBP 140–150 (130–139 if fit)
- Pregnancy: <140/90
- Reach target within 3 months

7 · Compelling indications

- Diabetes/CKD/proteinuria: ACEi/ARB + SGLT2i
- HFrEF: ARNI/ACEi, β-blocker, MRA, SGLT2i
- Angina/post-MI: β-blocker ± CCB + ACEi
- Stroke: ACEi + indapamide; gout: losartan

8 · Emergencies

- BP ≥180/110–120 + acute organ damage → admit, IV labetalol/nicardipine
- Lower MAP ≤25% in first hour (dissection: SBP <120 in 20 min)
- No sublingual nifedipine
- Without organ damage: oral drugs, review in days

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PART I · CHAPTER 1

History of Hypertension and Landmark Trials

From Hales' glass tube to SPRINT and ESPRIT — how we learnt that lowering blood pressure saves lives

Evolution of blood pressure measurement

Year	Milestone	Significance
1733	**Stephen Hales**	First direct (invasive) measurement of arterial pressure — a glass tube inserted into the carotid artery of a horse; blood rose about 9 feet.
1881	**Samuel von Basch**	First non-invasive sphygmomanometer using a water-filled bag and a mercury manometer.
1896	**Scipione Riva-Rocci**	Inflatable arm cuff with mercury manometer — the basis of today's instrument (systolic by palpation).
1905	**Nikolai Korotkoff**	Described the auscultatory sounds — allowed measurement of both systolic and diastolic pressure.
1960s–70s	Ambulatory BP monitoring	Portable recorders revealed circadian rhythm, nocturnal dipping, white-coat and masked hypertension.
1980s–2000s	Oscillometric automated devices	Validated upper-arm devices replaced mercury in clinics and homes; Minamata Convention (2013) phases out mercury devices.
2017–2025	Out-of-office BP and AOBP	Guidelines require home (HBPM) or ambulatory (ABPM) confirmation of diagnosis; cuffless wearables are not yet recommended for diagnosis.

From "essential" to treatable disease

In the first half of the 20th century, high blood pressure was widely regarded as "essential" — a compensatory rise needed to perfuse stiff arteries. President Franklin D. Roosevelt died in 1945 of a cerebral haemorrhage with untreated BP of about 300/190 mmHg, a reminder of the natural history of the disease. **Malignant hypertension** carried a one-year mortality of nearly 80% before effective drugs. Early therapies were crude — rice-fruit (Kempner) diet, sympathectomy, ganglion blockers and reserpine. The arrival of **chlorothiazide (1957)**, followed by beta-blockers (propranolol, 1960s), ACE inhibitors (captopril, 1981), calcium channel blockers and ARBs (losartan, 1995), made safe long-term control possible.

Milestone studies every clinician should know

Study (year)	Population / design	Key message
Framingham Heart Study (1948–)	Community cohort, Massachusetts	Coined "risk factor"; BP (especially systolic) is a continuous, graded predictor of stroke, CAD and heart failure.
VA Cooperative Study (1967, 1970)	Men with diastolic BP 90–129 mmHg	First RCT proving that drug treatment prevents strokes, heart failure and death.
SHEP (1991)	Isolated systolic hypertension, ≥60 y	Chlorthalidone-based therapy reduced stroke by 36% — systolic BP matters in the elderly.
UKPDS 38 (1998)	Type 2 diabetes with hypertension	Tight BP control reduced microvascular and macrovascular complications more than tight glucose control did.
HOT (1998)	Diastolic targets ≤90/≤85/≤80	Benefit of lower diastolic target in diabetes; low-dose aspirin data.

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Study (year)	Population / design	Key message
HOPE (2000)	High vascular risk	Ramipril reduced MI, stroke and death — RAAS blockade beyond BP lowering.
ALLHAT (2002)	>33,000 hypertensives ≥55 y	Chlorthalidone as good as amlodipine and lisinopril for CAD; superior for heart failure; doxazosin arm stopped early.
ASCOT-BPLA (2005)	Hypertension + ≥3 risk factors	Amlodipine ± perindopril beat atenolol ± thiazide — atenolol lost first-line status.
HYVET (2008)	Age ≥80 y	Indapamide ± perindopril to <150/80 cut mortality by 21% — treat the very elderly.
ACCOMPLISH (2008)	High-risk hypertensives	Benazepril + amlodipine superior to benazepril + hydrochlorothiazide.
ONTARGET (2008)	Telmisartan vs ramipril vs both	ARB = ACEi; dual RAAS blockade caused more renal harm — never combine.
ACCORD-BP (2010)	Type 2 diabetes	SBP <120 vs <140 did not reduce the composite outcome but reduced stroke.
SPRINT (2015)	Non-diabetic high-risk, ≥50 y	SBP target <120 reduced CV events by 25% and all-cause death by 27% versus <140; also reduced MCI (SPRINT-MIND).
PATHWAY-2 (2015)	Resistant hypertension	Spironolactone was the most effective fourth drug.
STEP (2021)	Chinese adults 60–80 y	SBP target 110–130 reduced CV events versus 130–150.
CHAP (2022)	Mild chronic hypertension in pregnancy	Treating to <140/90 reduced pre-eclampsia with severe features and preterm birth without fetal harm.
ESPRIT (2024) / **BPROAD** (2024)	High-risk Chinese adults; type 2 diabetes	Intensive SBP <120 reduced major CV events, with and without diabetes.
SPYRAL / RADIANCE (2018–2023)	Uncontrolled hypertension	Renal denervation lowers BP by about 5–10 mmHg (sham-controlled) — led to regulatory approval.

KEY POINT

Every 10 mmHg fall in systolic BP reduces major cardiovascular events by about 20%, stroke by 27%, heart failure by 28% and all-cause death by 13% (Ettehad meta-analysis, Lancet 2016) — regardless of the starting BP and of the drug class used.

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PART I · CHAPTER 2

Normal Physiology and Haemodynamics

How the body sets and defends arterial pressure — the basis of every drug we prescribe

The basic equation

EQUATION

Mean arterial pressure (MAP) = Cardiac output (CO) × Total peripheral resistance (TPR)

CO = Heart rate × Stroke volume · MAP ≈ DBP + ⅓ (SBP − DBP)

Any rise in BP must come from an increase in cardiac output (volume, heart rate, contractility) or in peripheral resistance (arteriolar tone and structure). In early hypertension, especially in young people, CO is often high ("hyperkinetic" state); in established hypertension, CO normalises and **raised peripheral resistance** sustains the BP. In older people, **large-artery stiffness** raises pulse pressure and produces isolated systolic hypertension.

Poiseuille's law shows that resistance is inversely proportional to the **fourth power of the radius** — a small narrowing of arterioles causes a large rise in resistance. This is why arteriolar remodelling is so important in sustained hypertension.

Determinants of blood pressure and the drugs that act on them

Determinant	Physiology	Drugs acting here
Blood volume / preload	Renal sodium and water handling; pressure natriuresis	Thiazide and loop diuretics, MRAs, SGLT2 inhibitors (modest)
Heart rate and contractility	Sympathetic β1 stimulation	Beta-blockers, non-DHP CCBs, ivabradine (rate only)
Arteriolar tone	Angiotensin II, noradrenaline (α1), endothelin, calcium entry, NO	ACEi, ARBs, DHP CCBs, α-blockers, hydralazine, minoxidil, nitrates
Central sympathetic outflow	Vasomotor centre in medulla	Clonidine, methyldopa, moxonidine
Renin release	Juxtaglomerular cells (β1, low perfusion, low NaCl at macula densa)	Beta-blockers, aliskiren (direct renin inhibitor)
Aldosterone	Distal Na^+ reabsorption, K^+ loss, fibrosis	Spironolactone, eplerenone, finerenone; amiloride (ENaC)

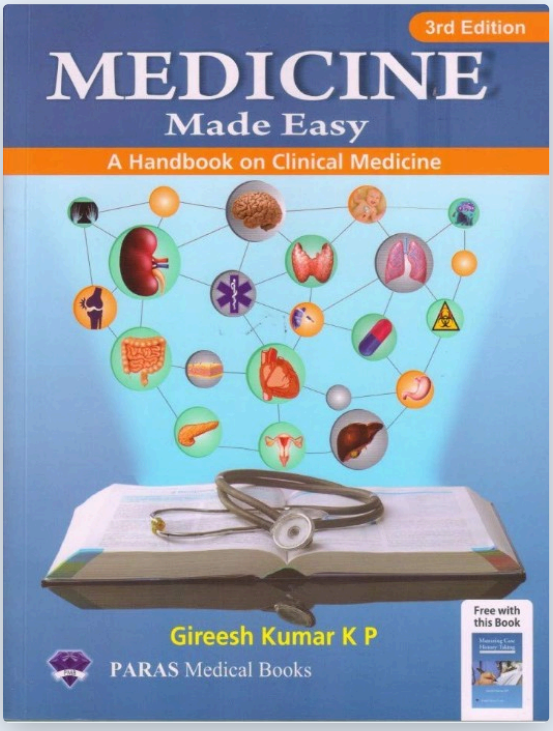
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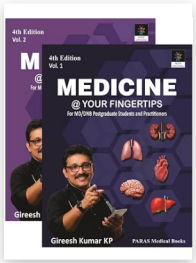
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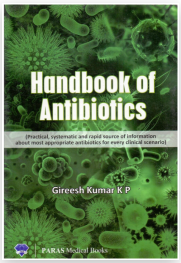
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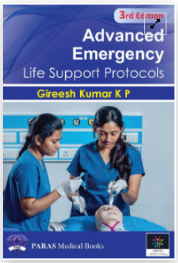
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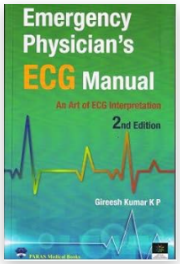
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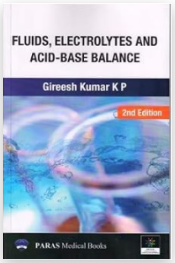
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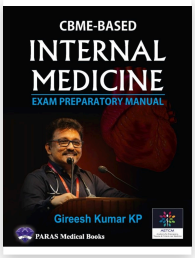
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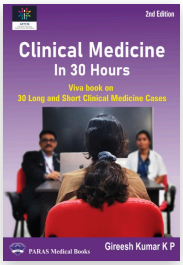
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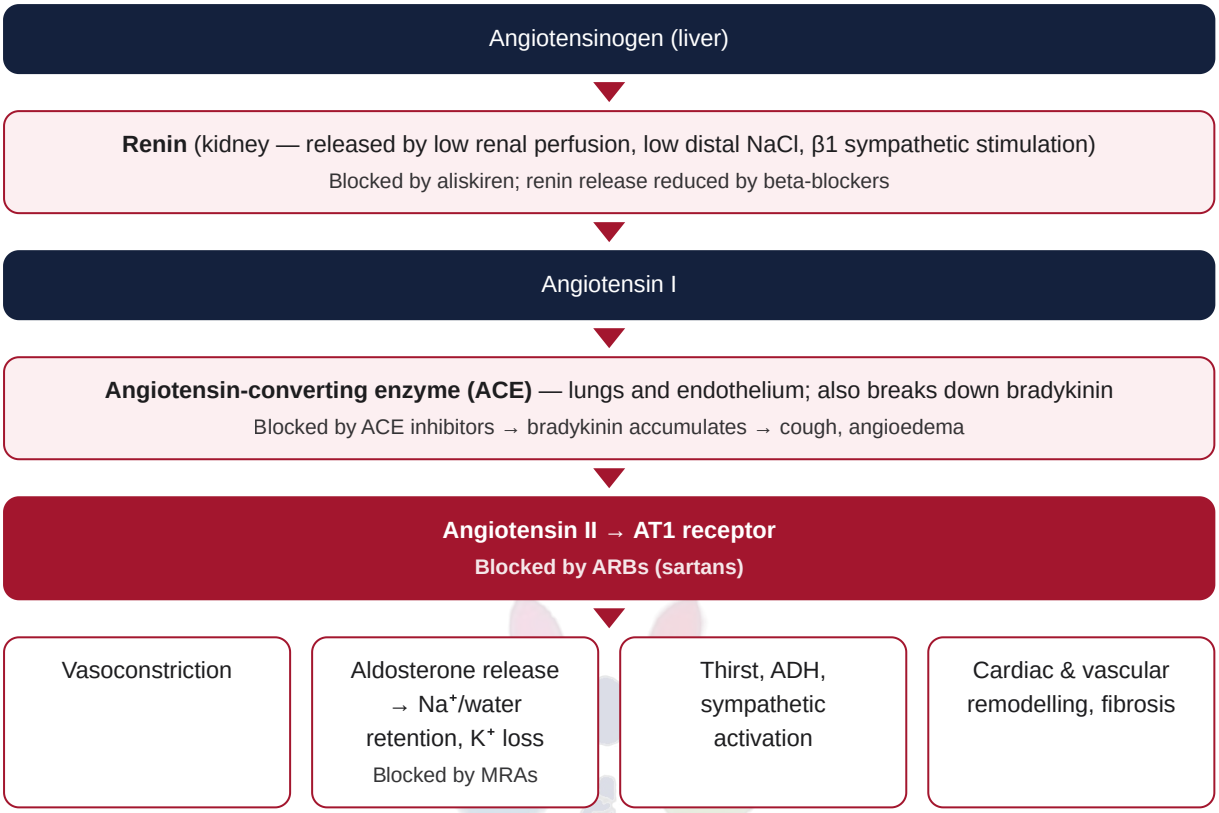


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The renin–angiotensin–aldosterone system (RAAS)



Autonomic regulation and baroreceptors

- **Arterial baroreceptors** in the carotid sinus (afferent via glossopharyngeal nerve) and aortic arch (vagus) sense stretch. A rise in BP increases firing → nucleus tractus solitarius → reduced sympathetic outflow and increased vagal tone → fall in heart rate and vasodilatation.
- Baroreceptors **reset** to a higher level within days of sustained hypertension — they buffer short-term fluctuations but do not set long-term BP. Baroreflex sensitivity falls with age, diabetes and hypertension → orthostatic hypotension and BP variability.
- **Long-term BP** is set by the kidney through **pressure natriuresis** (Guyton): as BP rises, the kidney excretes more sodium and water until volume and BP return to normal. In hypertension the pressure–natriuresis curve is shifted to the right — a higher BP is needed to excrete the same salt load.
- **Sympathetic overactivity** is prominent in obesity, obstructive sleep apnoea, CKD, stress and young hypertensives.

Other regulators

System	Effect on BP
Natriuretic peptides (ANP, BNP)	Released by atrial/ventricular stretch; natriuresis, vasodilatation, inhibit renin and aldosterone. Neprilysin breaks them down — sacubitril inhibits neprilysin.
Endothelium	Nitric oxide and prostacyclin dilate; endothelin-1 constricts. Endothelial dysfunction is an early feature of hypertension, diabetes and smoking. Aprocitentan blocks endothelin receptors.
Antidiuretic hormone	Water retention and V1 vasoconstriction; important in shock rather than in chronic hypertension.
Prostaglandins	Renal vasodilatation and natriuresis — NSAIDs block them and raise BP and blunt most antihypertensives.
Insulin	Hyperinsulinaemia increases renal sodium reabsorption and sympathetic tone — link between obesity, insulin resistance and hypertension.

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Circadian variation

BP is highest in the morning (morning surge 6–10 am, when MI and stroke peak), falls during the day's rest and drops 10–20% during sleep ("dipping"). **Non-dippers** (<10% fall) and **reverse dippers** (night BP higher than day) have higher cardiovascular risk; this pattern is common in CKD, diabetes, OSA, autonomic failure and secondary hypertension.

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PART I · CHAPTER 3

Epidemiology and Public Health Burden

The silent killer — common, under-diagnosed and poorly controlled, in India and worldwide

Global picture

- The **WHO Global Report on Hypertension (2023)** estimates that about **1.3 billion adults aged 30–79 years** have hypertension — roughly one in three adults. Nearly half do not know they have it, and only about **one in five** has the BP controlled.
- High BP is the **leading modifiable risk factor for death worldwide** (Global Burden of Disease), responsible for about 10 million deaths a year, mainly through stroke, ischaemic heart disease, heart failure and CKD.
- Most people with hypertension now live in low- and middle-income countries, where awareness, treatment and control rates are lowest.

Hypertension in India

Indicator	Estimate / comment
Prevalence in adults	About 30% of adults (WHO 2023 estimates ~188 million Indian adults aged 30–79 years). ICMR-INDIAB (2023) reported a prevalence of 35.5%; NFHS-5 (2019–21) found raised BP in about 24% of men and 21% of women aged ≥15 years.
Awareness, treatment, control	Only about one-third are diagnosed, under one-third treated and roughly **15% controlled** (<140/90) — the "rule of halves" is worse than a half in India.
Regional pattern	Higher prevalence in southern states (Kerala, Tamil Nadu, Andhra Pradesh, Telangana), Punjab and the north-east; urban > rural, but the rural gap is closing.
Younger onset	Hypertension and its complications appear about a decade earlier in South Asians; stroke and MI under 50 years are common.
Contribution to deaths	Raised BP is linked to more than half of stroke deaths and about a quarter of coronary deaths in India.

Risk factors driving the epidemic in India

- **High salt intake** — average 8–11 g/day (WHO target <5 g/day); pickles, papad, dried fish, salted snacks, bakery items and restaurant food.
- **Overweight and central obesity** — South Asians develop metabolic risk at a lower BMI (overweight ≥23 kg/m², obesity ≥25 kg/m²; waist ≥90 cm men, ≥80 cm women).
- **Physical inactivity** and sedentary urban work.
- **Low intake** of fruits and vegetables (low potassium).
- **Tobacco** — smoking, beedi and smokeless tobacco.
- **Alcohol** — rising consumption, especially among men.
- **Diabetes** — India has over 100 million people with diabetes; hypertension coexists in more than half.
- **Psychosocial stress**, poor sleep, air pollution and ageing of the population.

Social determinants and economic impact

- Lower education, low income, urban slums and rural areas with poor access to care have lower awareness and control.

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- Out-of-pocket expenditure on chronic medicines and complications (stroke rehabilitation, dialysis, cardiac procedures) pushes families into poverty.
- Long-term adherence is limited by cost, pill burden, belief that drugs are "needed only when symptomatic", and fear of "lifelong medicine".

National programmes

Programme	What it does
NP-NCD (formerly NPCDCS)	National Programme for Prevention and Control of Non-Communicable Diseases — population-based opportunistic screening of all adults ≥30 years at Ayushman Arogya Mandirs (Health and Wellness Centres).
India Hypertension Control Initiative (IHCI)	Started 2017 by MoHFW, ICMR, state governments and WHO — simple standard drug protocol, assured drug supply, team-based care with task sharing, patient-centred follow-up and digital tracking. Protocol in Chapter 17.
"75/25" initiative (2023)	Target of placing 75 million people with hypertension or diabetes on standard care by 2025.
FSSAI "Eat Right India"	Salt, sugar and trans-fat reduction in processed food; front-of-pack labelling efforts.

KEY POINT

Measure BP of **every adult at every visit**, whatever the complaint. Opportunistic screening is the single most effective step a general practitioner can take to close India's awareness gap.

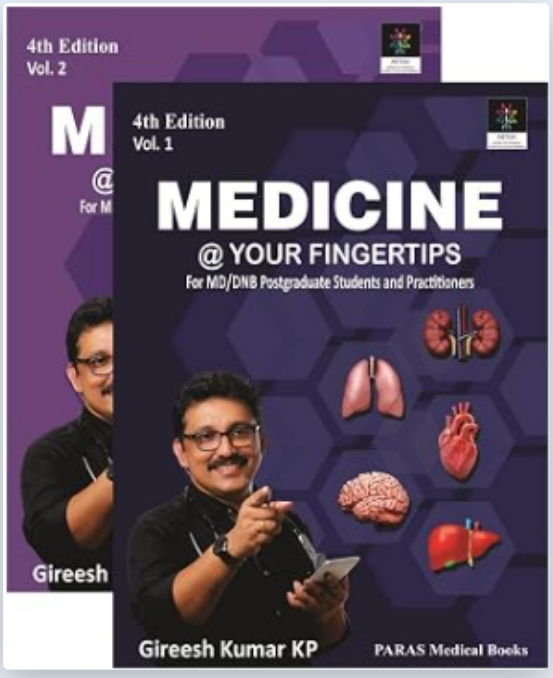
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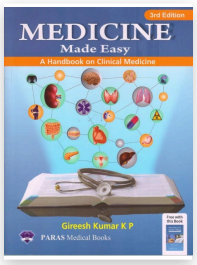
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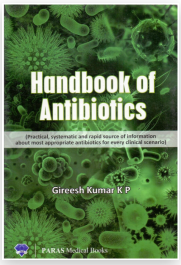
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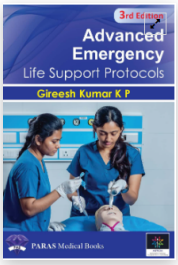
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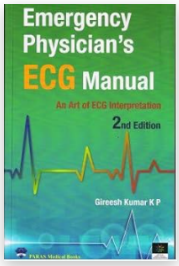
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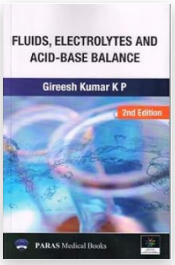
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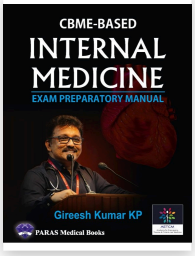
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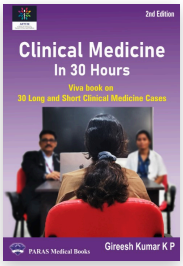
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PART II · CHAPTER 4

Definitions and Classification

What counts as hypertension, how guidelines differ, and the clinical phenotypes you must recognise

Definition

Hypertension is a persistent elevation of arterial blood pressure to a level at which the benefits of treatment (lifestyle and/or drugs) clearly outweigh the risks. Because the relation between BP and cardiovascular risk is continuous from as low as 115/75 mmHg, any cut-off is a practical threshold rather than a biological boundary.

WORKING DEFINITION USED IN THIS BOOK

Office BP **≥140/90 mmHg** on at least two visits (or ≥130/80 mmHg using the ACC/AHA definition), **confirmed by out-of-office readings** (home BP ≥135/85 or 24-hour ambulatory mean ≥130/80), or anyone already on antihypertensive treatment. A single office reading ≥180/110 with target-organ damage is diagnostic straight away.

Classification of office blood pressure — major guidelines compared

Guideline	Categories (mmHg)	Hypertension threshold
ACC/AHA 2017, retained in 2025	Normal <120 and <80 · Elevated 120–129 and <80 · **Stage 1** 130–139 or 80–89 · **Stage 2** ≥140 or ≥90	≥130/80
ESC 2024	Non-elevated <120/70 · **Elevated BP** 120–139/70–89 · **Hypertension** ≥140/90	≥140/90
ESH 2023	Optimal <120/80 · Normal 120–129/80–84 · High-normal 130–139/85–89 · **Grade 1** 140–159/90–99 · **Grade 2** 160–179/100–109 · **Grade 3** ≥180/≥110 · Isolated systolic ≥140/<90	≥140/90
ISH 2020	Normal <130/85 · High-normal 130–139/85–89 · **Grade 1** 140–159/90–99 · **Grade 2** ≥160/≥100	≥140/90
Indian Guidelines (IGH-IV, 2019) and NP-NCD	Follow ESH-type grading; IHCI uses ≥140/90 for diagnosis and <140/90 as the programme target	≥140/90
WHO 2021	Treat if ≥140/90; consider drugs at SBP 130–139 with CVD, diabetes, CKD or high CV risk	≥140/90

When systolic and diastolic readings fall in different categories, the higher category applies.

Practical classification for the clinic (ESH grading)

Category	Systolic (mmHg)		Diastolic (mmHg)	Usual action
Optimal	<120	and	<80	Recheck every 3–5 years
Normal	120–129	and/or	80–84	Recheck every 3 years; lifestyle
High-normal	130–139	and/or	85–89	Yearly; lifestyle; drugs only if very high risk/CVD
Grade 1 hypertension	140–159	and/or	90–99	Confirm out-of-office; lifestyle ± drugs
Grade 2 hypertension	160–179	and/or	100–109	Start drugs (usually two-drug combination)
Grade 3 hypertension	≥180	and/or	≥110	Start drugs immediately; look for target-organ damage

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Category	Systolic (mmHg)		Diastolic (mmHg)	Usual action
Isolated systolic hypertension	≥140	and	<90	Grade by systolic level
Isolated diastolic hypertension	<140	and	≥90	Common in young adults

Thresholds for out-of-office measurements

Method	ESH / ESC / ISH (equivalent to office 140/90)	ACC/AHA (equivalent to office 130/80)
Office BP	≥140/90	≥130/80
Home BP (HBPM) mean	≥135/85	≥130/80
ABPM daytime (awake) mean	≥135/85	≥130/80
ABPM night-time (asleep) mean	≥120/70	≥110/65
ABPM 24-hour mean	≥130/80	≥125/75

Clinical phenotypes of hypertension

Phenotype	Definition	Clinical significance
Sustained hypertension	High office BP and high out-of-office BP	True hypertension — treat.
White-coat hypertension	Office ≥140/90 but home/ambulatory BP normal (untreated patient)	15–30% of raised office readings; risk intermediate; lifestyle and annual out-of-office monitoring; avoid over-treatment. In a treated patient this is called **white-coat uncontrolled** hypertension.
Masked hypertension	Office BP normal but home/ambulatory BP high	Risk similar to sustained hypertension; suspect when office BP is high-normal, with TOD, diabetes, CKD, smokers, high stress. Treated equivalent: **masked uncontrolled**.
Nocturnal hypertension	Night-time ABPM ≥120/70	Seen in CKD, diabetes, OSA, secondary hypertension; predicts events better than daytime BP.
Isolated systolic hypertension (elderly)	SBP ≥140, DBP <90, age >60	Due to aortic stiffness; strong predictor of stroke; treat.
Isolated systolic hypertension of youth	Young, tall, athletic men with high brachial SBP	Often normal central aortic pressure ("spurious"); measure out-of-office and follow up.
Resistant hypertension	Uncontrolled on ≥3 drugs at optimal doses including a diuretic (after excluding pseudo-resistance)	See Chapter 21.
Hypertensive emergency	Severe BP (usually ≥180/120) with acute target-organ damage	Admit; IV therapy — Chapter 20.
Orthostatic hypertension	Rise in SBP ≥20 mmHg on standing	Elderly, autonomic dysfunction; increased risk.

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PART II · CHAPTER 5

Primary (Essential) Hypertension

90–95% of all hypertension — a polygenic, lifestyle-driven disease with no single identifiable cause

Definition

Primary hypertension is raised BP for which no single reversible cause can be identified. It accounts for about **90–95%** of adults with hypertension. It usually appears after 30 years of age, often runs in families, rises gradually and is commonly associated with overweight, high salt intake and the metabolic syndrome.

Risk factors

Non-modifiable	Modifiable
• Age — BP (especially systolic) rises with age • Family history — risk doubles if one parent, quadruples if both parents are hypertensive • Male sex (before 55 years); women catch up after menopause • Ethnicity — South Asians and people of African descent • Low birth weight (Barker hypothesis — fewer nephrons)	• **High sodium intake** (>5 g salt/day) and low potassium intake • **Overweight and obesity**, especially central (each 1 kg weight gain raises BP ~1 mmHg) • **Physical inactivity** • **Alcohol** — more than 1–2 drinks per day • Tobacco (acute pressor effect; accelerates arterial damage) • Diet low in fruit, vegetables and dairy; high in processed food • Psychosocial stress, shift work, short or poor-quality sleep • Diabetes, dyslipidaemia, insulin resistance • Air and noise pollution

Pathogenesis — the mosaic theory

Page's "mosaic theory" describes hypertension as the result of many interacting factors. The main mechanisms are:

Mechanism	What happens
Genetic predisposition	Polygenic — more than 1,000 gene variants each add a small effect on BP; rare monogenic forms (Liddle syndrome, glucocorticoid-remediable aldosteronism, Gordon syndrome) cause salt-sensitive hypertension at a young age.
Renal sodium retention	Impaired pressure natriuresis; salt sensitivity (common in elderly, obese, diabetic, CKD and people of African and South Asian origin).
Sympathetic overactivity	Raised heart rate and cardiac output in young hypertensives; driven by obesity, stress, OSA and insulin resistance.
RAAS activation	Inappropriately normal or high renin for the sodium status; angiotensin II causes vasoconstriction, sodium retention and vascular remodelling.
Endothelial dysfunction	Reduced nitric oxide, increased endothelin and oxidative stress.
Vascular remodelling and arterial stiffness	Small arteries: inward eutrophic remodelling (raised resistance). Large arteries: elastin loss and collagen deposition → raised pulse wave velocity → early reflected waves → isolated systolic hypertension in the elderly.
Obesity-related mechanisms	Leptin-driven sympathetic activation, adipose tissue RAAS, hyperinsulinaemia, perirenal fat compressing the kidney, OSA.
Immune and inflammatory factors	T-cell activation and inflammation in kidney and vessel wall perpetuate hypertension.

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Natural history

- Young adults: often isolated diastolic or combined systolic–diastolic hypertension with high cardiac output.
- Middle age: combined hypertension with raised peripheral resistance.
- Older age: diastolic BP falls after 55–60 years and **isolated systolic hypertension** with wide pulse pressure dominates.
- Untreated, hypertension leads to LVH, heart failure, coronary disease, stroke, CKD and retinopathy over 10–20 years.

Clues that it is primary and not secondary

CLINICAL PEARL

- Onset between 30 and 55 years, gradual rise, strong family history, overweight, no clinical or biochemical clues (normal potassium, creatinine and urine).
- BP responds to one or two standard drugs.
- Any atypical feature — onset <30 years, sudden worsening, resistant BP, hypokalaemia, paroxysms — should prompt a search for a secondary cause (Chapter 6).

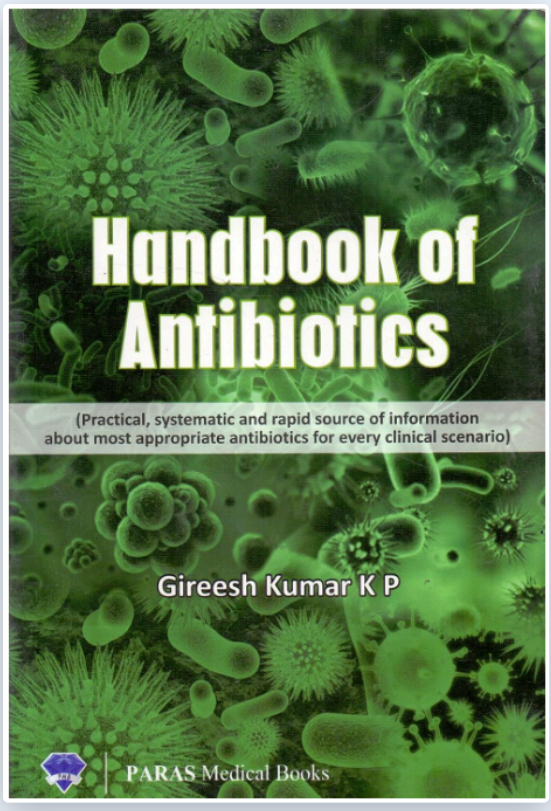
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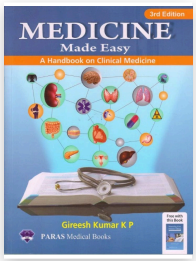
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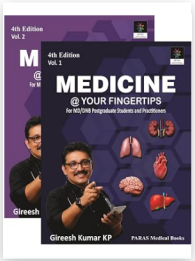
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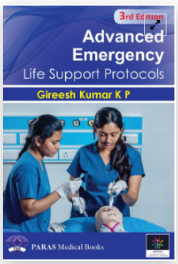
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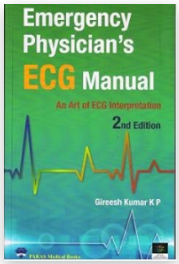
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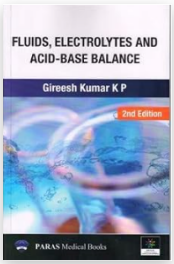
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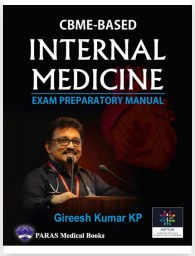
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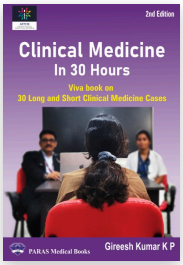
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PART II · CHAPTER 6

Secondary Hypertension

5–10% of adults (up to 30% of young or resistant cases) — identifiable and often curable causes

When to suspect secondary hypertension

- Onset **before 30 years** (especially without obesity or family history), or new onset after 60 years
- **Severe** (grade 3) or **resistant** hypertension
- **Sudden worsening** of previously controlled BP
- Hypertensive emergency or malignant hypertension
- Disproportionate target-organ damage
- **Spontaneous or diuretic-induced hypokalaemia**
- Rise in creatinine >30% after starting ACEi/ARB
- **Paroxysms** of headache, sweating, palpitations
- Abdominal bruit, absent/delayed femoral pulses
- Snoring with daytime sleepiness
- Cushingoid features, adrenal incidentaloma

Causes of secondary hypertension — clues, screening and treatment

Cause	Clinical clues	Screening test	Confirmation	Treatment
Renal				
Renal parenchymal disease (most common)	Oedema, nocturia, haematuria, proteinuria, diabetes, past GN, polycystic kidneys	Creatinine/eGFR, urine routine, UACR, renal ultrasound	Renal biopsy as needed	ACEi/ARB, BP <130/80, SGLT2i, treat cause
Renovascular — atherosclerotic (elderly) or fibromuscular dysplasia (young women)	Abdominal bruit, flash pulmonary oedema, rise in creatinine >30% on ACEi/ARB, asymmetric kidneys (>1.5 cm), widespread atherosclerosis	Renal Doppler ultrasound	CT or MR angiography; DSA	FMD: angioplasty (often curative). Atherosclerotic: medical therapy (statin, antiplatelet, BP control); stent only for selected cases
Endocrine				
Primary aldosteronism (5–10% of hypertensives; up to 20% of resistant)	Resistant BP, hypokalaemia (only 30–50% have it), adrenal nodule, OSA, AF, family history of early stroke	**Aldosterone–renin ratio** (ARR) — morning, seated; correct potassium first	Saline infusion or oral salt loading test; adrenal CT; adrenal vein sampling	Unilateral adenoma: laparoscopic adrenalectomy. Bilateral hyperplasia: spironolactone/eplerenone
Phaeochromocytoma / paraganglioma	Paroxysms of headache, palpitations, sweating ("the triad"), pallor, labile BP, orthostatic fall, adrenal mass, MEN2, NF1	Plasma free metanephrines or 24-h urine fractionated metanephrines	CT/MRI adrenal; MIBG or Ga-68 DOTATATE PET	α-blockade first (phenoxybenzamine/prazosin), then β-blocker, then surgery. **Never start a β-blocker alone**
Cushing's syndrome	Central obesity, moon face, purple striae, proximal myopathy, diabetes, bruising	Overnight 1 mg dexamethasone suppression, 24-h urinary free cortisol, late-night salivary cortisol	ACTH, high-dose DST, MRI pituitary / CT adrenal	Surgery of cause; MRAs control BP
Thyroid disease	Hyperthyroid: tachycardia, systolic HTN. Hypothyroid: diastolic HTN	TSH, free T4	—	Treat thyroid; β-blocker for hyperthyroid
Primary hyperparathyroidism	Hypercalcaemia, renal stones, bone pain	Serum calcium, PTH	Sestamibi scan	Parathyroidectomy
Acromegaly	Coarse features, large hands and feet	IGF-1	GH suppression test, MRI	Surgery

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Cause	Clinical clues	Screening test	Confirmation	Treatment
Others				
Obstructive sleep apnoea	Snoring, witnessed apnoeas, daytime sleepiness, obesity, neck >40 cm, nocturnal/resistant HTN	STOP-BANG questionnaire	Polysomnography	CPAP, weight loss
Coarctation of aorta	Young patient, radio-femoral delay, arm BP > leg BP by ≥20 mmHg, scapular collaterals, ejection murmur at back	Echocardiography, chest X-ray (rib notching, "3" sign)	CT/MR aortography	Balloon/stent or surgery
Takayasu arteritis (important in India)	Young woman, absent/unequal pulses, BP difference between arms, bruits, renal artery stenosis	ESR/CRP, Doppler	CT/MR angiography	Steroids, immunosuppression, revascularisation
Drug- or substance-induced	See table below	Detailed drug history	BP falls after withdrawal	Stop or substitute
Pregnancy-related	New hypertension after 20 weeks	Urine protein, platelets, LFT, creatinine	—	Chapter 22
Monogenic (Liddle, GRA, Gordon, AME)	Young onset, family history, hypokalaemia (or hyperkalaemia in Gordon), low renin	Renin, aldosterone	Genetic testing	Amiloride (Liddle), dexamethasone (GRA), thiazide (Gordon)

Drugs and substances that raise blood pressure

Drug / substance	Comment and alternative
NSAIDs (including COX-2 inhibitors)	Most common cause in practice; sodium retention, blunt diuretics/ACEi/ARB; prefer paracetamol, topical NSAIDs
Oral contraceptives (oestrogen-containing)	Small rise in most women, occasionally severe; switch to progestogen-only pill, IUCD
Corticosteroids, fludrocortisone	Dose-related; use lowest effective dose
Decongestants (pseudoephedrine, phenylephrine), weight-loss pills, stimulants (amphetamines, methylphenidate)	Common in cough and cold combinations — check OTC use
Antidepressants (venlafaxine, desvenlafaxine, MAOIs, TCAs), bupropion	Monitor BP; consider SSRI
Calcineurin inhibitors (ciclosporin, tacrolimus), erythropoietin	Common in transplant and CKD patients; CCBs preferred
VEGF inhibitors and TKIs (bevacizumab, sunitinib, sorafenib)	Monitor weekly at start; ACEi/ARB or DHP-CCB
Liquorice, carbenoxolone	Apparent mineralocorticoid excess — hypokalaemia
Alcohol, cocaine, caffeine (high dose), energy drinks	Avoid β-blockers alone in cocaine toxicity
Anabolic steroids, testosterone; some herbal products (ephedra, ginseng, yohimbine)	Ask specifically about gym supplements

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CLINICAL PEARL — ALDOSTERONE–RENIN RATIO

Correct hypokalaemia and liberalise salt before the test. Spironolactone, eplerenone and amiloride interfere most and should ideally be stopped for 4 weeks. ACEi, ARBs and diuretics can give false negatives; β-blockers and central agents give false positives. Verapamil, hydralazine and doxazosin interfere least and can be used to control BP during testing. Many centres now test without stopping drugs and interpret the result in context.

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PART III · CHAPTER 7

How to Measure Blood Pressure Correctly

Office technique step by step, cuff sizes, Korotkoff sounds, common errors, home and ambulatory monitoring

WHY TECHNIQUE MATTERS

A careless reading can be 10–20 mmHg off — enough to label a normal person hypertensive or to miss uncontrolled BP. Diagnosis and every change in treatment depend on **accurate, repeated, standardised** measurement.

Choosing the device

- Use a **validated automated upper-arm oscillometric device** (check validation lists such as STRIDE BP or validatebp.org) or a calibrated aneroid sphygmomanometer with a stethoscope. Mercury devices are being phased out but remain a reference where available.
- Wrist devices are less accurate (use only when upper-arm cuffs cannot fit). Finger devices and cuffless wearables (watches, rings) are **not recommended** for diagnosis or treatment decisions.
- Calibrate aneroid devices every 6–12 months — the needle must rest at zero.

Selecting the correct cuff size

The **bladder length should be 75–100% and the width about 40%** of the mid-arm circumference. Measure the arm circumference at the midpoint between acromion and olecranon.

Mid-arm circumference	Cuff	Bladder size (width × length)
22–26 cm	Small adult	12 × 22 cm
27–34 cm	Standard adult	16 × 30 cm
35–44 cm	Large adult	16 × 36 cm
45–52 cm	Adult thigh cuff	16 × 42 cm

A cuff that is too small over-reads BP (common in obese patients); a cuff that is too large slightly under-reads.

Office measurement — step by step

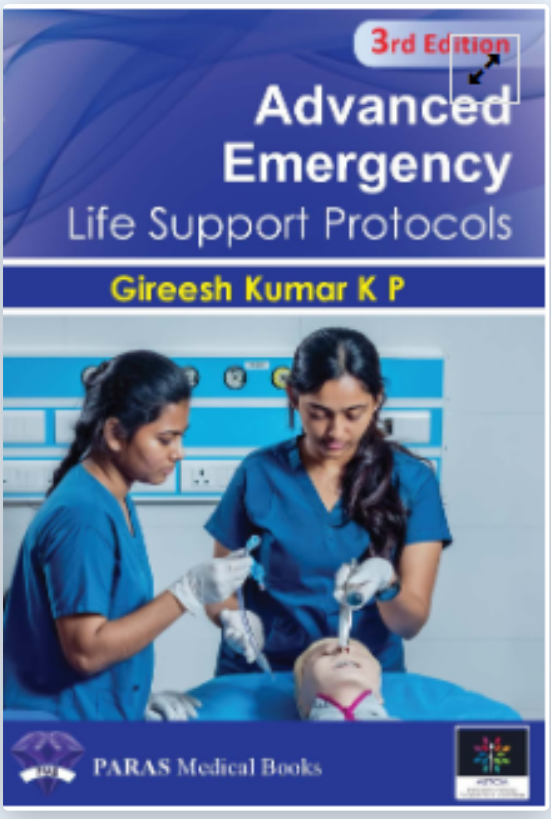
1. **Prepare the patient.** No smoking, caffeine or exercise for 30 minutes; empty the bladder. Patient sits quietly for **3–5 minutes** in a quiet room. Neither patient nor observer talks during the measurement.
2. **Position correctly.** Seated, back supported, feet flat on the floor, legs uncrossed. Bare arm (no rolled-up tight sleeve), supported on a table, with the cuff at **heart level** (mid-sternum).
3. **Apply the cuff.** Correct size; lower edge 2–3 cm above the antecubital fossa; bladder centre over the brachial artery (artery marker); snug enough to admit two fingers.
4. **Estimate systolic BP by palpation** (manual method). Palpate the radial pulse, inflate until it disappears, note the pressure, deflate. This avoids missing an **auscultatory gap**.

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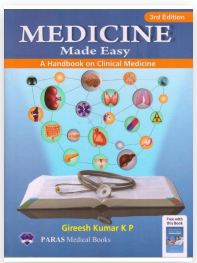
Step-by-step emergency life-support protocols for the first doctor at the scene.

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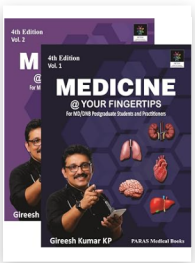
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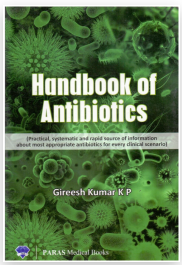
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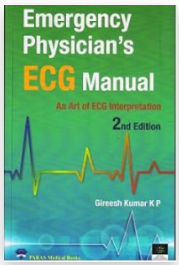
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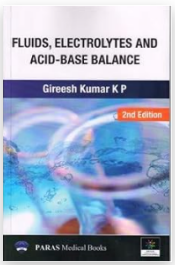
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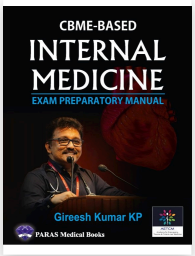
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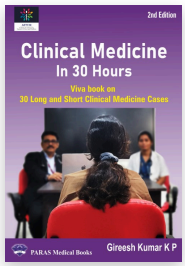
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5 **Auscultate.** Place the stethoscope diaphragm (or bell) lightly over the brachial artery, not under the cuff. Inflate to 20–30 mmHg above the palpated systolic pressure. Deflate at **2 mmHg per second**.

6 **Record.** Systolic = first appearance of clear tapping sounds (Korotkoff phase I). Diastolic = disappearance of sounds (phase V). Record to the nearest 2 mmHg, avoiding rounding to zero (terminal digit preference).

7 **Repeat.** Take **2–3 readings 1 minute apart** and use the average of the last two. If the first two readings differ by >10 mmHg, take more.

8 **Both arms at the first visit.** Use the arm with the higher reading for all future readings. A difference of **>10 mmHg** is associated with higher CV risk; >15–20 mmHg suggests subclavian stenosis, Takayasu arteritis or coarctation.

9 **Standing BP.** At the first visit and in the elderly, diabetic or patients with dizziness: measure after 1 and 3 minutes of standing. A fall of ≥20 mmHg systolic or ≥10 mmHg diastolic = **orthostatic hypotension**.

10 **Record heart rate** (palpate for 30 s) and inform the patient of the reading in writing.

Korotkoff sounds

Phase	Sound	Meaning
I	First clear, repetitive tapping sounds	**Systolic BP**
II	Softer swishing or murmur	May disappear briefly — the **auscultatory gap** (common in elderly and arterial stiffness)
III	Crisper, louder tapping	—
IV	Abrupt muffling	Use as diastolic only if sounds continue to zero (severe aortic regurgitation, hyperdynamic states, some children)
V	Disappearance of sounds	**Diastolic BP** in adults, including pregnancy

Common errors and their effect

Error	Effect on reading
Cuff too small for the arm	Over-reads by 2–10 mmHg (more in obesity)
Cuff over clothing	Over-reads by 5–50 mmHg
Talking or active listening	Over-reads by 10–15 mmHg
Full bladder	Over-reads by 10–15 mmHg
Arm unsupported	Over-reads by about 10 mmHg
Back unsupported, legs crossed	Over-reads by 2–10 mmHg
Arm below heart level / above heart level	Over-reads / under-reads (≈2 mmHg per 2.5 cm)
No rest period, recent smoking or coffee	Over-reads by 5–20 mmHg
Deflating too fast	Under-reads systolic, over-reads diastolic
Missing the auscultatory gap	Gross under-reading of systolic BP
Single reading only	Over-reads (first reading usually highest)

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Special situations

Situation	Technique
Atrial fibrillation	Take at least 3 readings; auscultatory method preferred; many automated devices are less accurate with irregular rhythm.
Obesity	Large or thigh cuff; conical (tapered) cuffs for cone-shaped arms; forearm measurement with a validated device as last resort.
Elderly and diabetics	Check standing BP at every visit; beware the auscultatory gap and pseudohypertension (Osler sign).
Pregnancy	Seated or left lateral, cuff at heart level; Korotkoff V for diastolic; use devices validated for pregnancy and pre-eclampsia.
Children	Cuff bladder width 40% and length 80–100% of arm circumference; compare with age/sex/height percentile tables.
Leg BP (suspected coarctation)	Prone, thigh cuff over the popliteal artery; normally leg SBP is 10–20 mmHg higher than arm; lower in coarctation.
Unattended automated office BP (AOBP)	Device takes 3–5 readings with patient alone in a room; reduces white-coat effect; values are about 5–10 mmHg lower than routine office BP.

Home blood pressure monitoring (HBPM)

STANDARD HOME PROTOCOL

- Validated upper-arm device; teach the patient the same preparation and posture as in the clinic.
- Measure for **7 days** (at least 3) before each clinic visit: **morning** (within 1 hour of waking, after urinating, before drugs and breakfast) and **evening** (before dinner).
- Take **2 readings 1 minute apart** on each occasion and write all of them down (or use device memory).
- Discard day 1; the **average of the remaining readings** is the home BP. Hypertension if mean ≥135/85 (ESH) or ≥130/80 (ACC/AHA).
- For long-term follow-up, 1–2 days a week is enough. Discourage obsessive repeated readings and self-adjustment of doses unless agreed.

Ambulatory blood pressure monitoring (ABPM)

- Readings every 15–30 minutes by day and 30–60 minutes at night for 24 hours, during normal activities, with a diary of sleep and symptoms.
- A valid study needs ≥70% successful readings (at least 20 daytime and 7 night-time).
- Gives day, night and 24-hour means, dipping status, morning surge and BP variability — the **best predictor of outcome**.
- **Indications:** suspected white-coat or masked hypertension, resistant hypertension, nocturnal hypertension (CKD, diabetes, OSA), discrepancy between office and home BP, symptoms of hypotension on treatment, autonomic dysfunction, evaluation in pregnancy.

Dipping pattern (night fall in SBP)	Category
10–20%	Normal dipper
>20%	Extreme dipper
0–10%	Non-dipper
<0% (night BP higher than day)	Reverse dipper (riser) — highest risk

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PART III · CHAPTER 8

Symptoms, History and Physical Examination

The silent disease — what the patient may tell you and what you must look for

Symptoms

Most people with hypertension have **no symptoms**; the first presentation may be a stroke, MI, heart failure or kidney failure. That is why hypertension is called the "silent killer". When symptoms occur, they fall into three groups:

Group	Symptoms
Due to high BP itself (usually only when severe)	Early-morning occipital headache, dizziness, tinnitus, blurred vision, epistaxis, palpitations, fatigue, nocturia. Headache and "tension" are often blamed on BP but are poorly related to the level of BP.
Due to target-organ damage	**Heart** — exertional breathlessness, orthopnoea, paroxysmal nocturnal dyspnoea, chest pain, palpitations, ankle swelling. **Brain** — transient weakness or numbness, speech disturbance, visual loss (TIA/stroke), memory decline. **Kidney** — nocturia, polyuria, oedema, frothy urine, haematuria. **Eyes** — blurring, field defects. **Arteries** — intermittent claudication; tearing chest/back pain (dissection).
Due to a secondary cause	Paroxysmal headache, sweating and palpitations (phaeochromocytoma); muscle weakness, cramps, polyuria (hypokalaemia — aldosteronism); snoring and daytime sleepiness (OSA); weight gain, striae, bruising (Cushing's); heat intolerance, tremor (thyrotoxicosis); leg fatigue (coarctation).
Danger symptoms (hypertensive emergency)	Severe headache with vomiting, confusion, seizures, visual loss, chest pain, acute breathlessness, focal neurological deficit, oliguria — **refer/admit immediately**.

History — checklist

About the hypertension

- When first detected; previous readings; highest BP
- Previous drugs — effect, side-effects, reasons for stopping
- Adherence; home BP readings
- Symptoms of target-organ damage and of secondary causes

Cardiovascular risk factors

- Diabetes, dyslipidaemia, CKD, smoking/tobacco, alcohol
- Family history of hypertension, premature CVD (men <55, women <65 years), sudden death, kidney disease
- Diet (salt, processed food), physical activity, sleep, stress, weight change

Past and drug history

- Stroke/TIA, CAD, heart failure, AF, PAD, kidney disease, gout, asthma/COPD, depression
- Drugs that raise BP: NSAIDs, steroids, OCPs, decongestants, antidepressants, herbal remedies, erythropoietin, calcineurin inhibitors

Women

- Hypertension or pre-eclampsia in pregnancy, gestational diabetes, PCOS, contraception, menopause, plans for pregnancy

Social

- Occupation, shift work, income, access to medicines and follow-up

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Physical examination

System	What to look for	Significance
General	Height, weight, **BMI**, **waist circumference**; pallor; oedema	Obesity; anaemia of CKD; fluid overload
	Cushingoid habitus, purple striae, acanthosis nigricans	Cushing's; insulin resistance
	Neurofibromas, café-au-lait spots	Neurofibromatosis — phaeochromocytoma, renal artery stenosis
	Goitre, tremor, eye signs; acromegalic features; xanthelasma	Thyroid; acromegaly; dyslipidaemia
Pulses	Rate, rhythm (AF), all peripheral pulses, **radio-femoral delay**, inequality of pulses and arm BP	Coarctation, Takayasu arteritis, PAD
Neck	Carotid bruit; JVP	Carotid atherosclerosis; heart failure
Heart	Apex beat — heaving (LVH) or displaced (dilatation); loud A2; S4 (stiff LV) or S3 (failure); murmurs — aortic regurgitation, ejection murmur at back (coarctation)	Hypertensive heart disease
Chest	Basal crackles, pleural effusion	Heart failure
Abdomen	Renal bruit (epigastrium, flanks) — systolic-diastolic; ballotable kidneys; pulsatile mass	Renovascular disease; polycystic kidneys; aortic aneurysm
Fundus	Arteriolar narrowing, AV nicking, haemorrhages, exudates, papilloedema	Hypertensive retinopathy — grade 3–4 = emergency
Nervous system	Focal deficits, cognition (MMSE/MoCA)	Stroke, vascular cognitive impairment
Legs	Pedal oedema, absent foot pulses; ankle–brachial index	CCB oedema, heart/kidney failure; PAD

CLINICAL PEARL

Always palpate the **radial and femoral pulses together** in every young hypertensive — coarctation of the aorta is easily missed and easily cured. Check BP in **both arms** in young women — Takayasu arteritis is not rare in India.

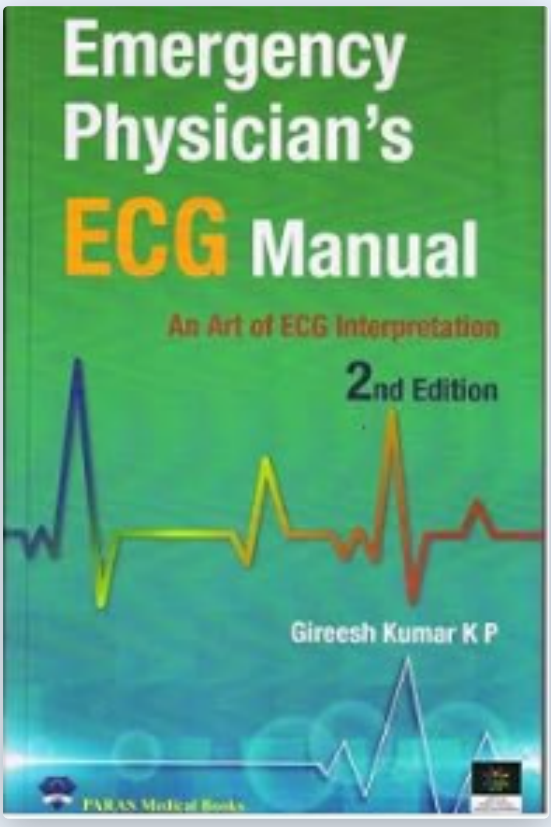
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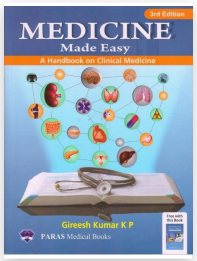
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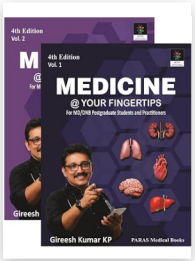
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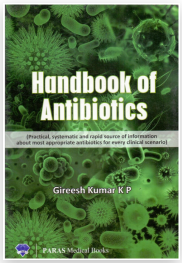
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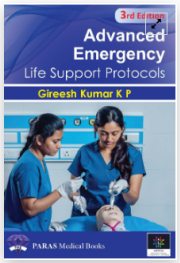
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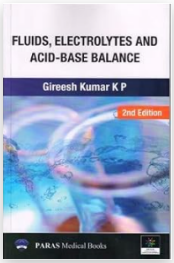
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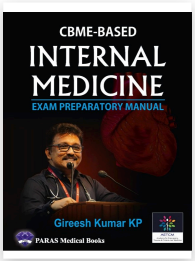
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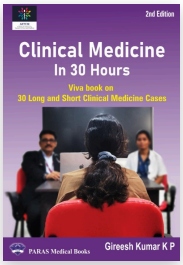
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PART III · CHAPTER 9

Initial Evaluation and Investigations

Confirm the diagnosis, detect target-organ damage, find secondary causes and stratify cardiovascular risk

The four goals of the initial evaluation

1. Confirm hypertension
repeat office + home/ABPM

2. Assess target-organ damage
heart, kidney, eye, brain, vessels

3. Look for secondary causes
when clues present

4. Estimate total CV risk
guides thresholds and targets

Diagnostic pathway

Raised office BP (screening or opportunistic)

≥180/110
With symptoms/TOD → emergency pathway (Ch. 20). Without → start treatment the same day and review within 1 week

140–179 / 90–109
Confirm with HBPM or ABPM (or repeat office visits) within 1 month; initial investigations meanwhile

130–139 / 85–89
High-normal: lifestyle; recheck yearly; look for masked HTN if TOD/diabetes

Hypertension confirmed → basic tests for all, risk stratification, treatment plan

Basic investigations for every patient

Test	Purpose
Urine dipstick and microscopy	Protein, blood, casts — renal disease
Urine albumin–creatinine ratio (UACR)	Earliest marker of renal and vascular damage; ≥30 mg/g (≥3 mg/mmol) is abnormal
Serum creatinine and eGFR	CKD staging; baseline before ACEi/ARB/diuretics
Serum sodium and potassium	Low K^+ → aldosteronism, diuretic; high K^+ → CKD, drugs. Baseline for therapy
Fasting glucose and/or HbA1c	Diabetes and prediabetes
Lipid profile	CV risk; statin decision
Haemoglobin / CBC	Anaemia (CKD), polycythaemia
Serum uric acid	Gout risk with diuretics; CV risk marker
12-lead ECG	LVH, strain pattern, AF, old MI, conduction defects
TSH, calcium, LFT	When clinically indicated; LFT before statins

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Additional tests (when indicated)

Test	Indication / finding
Echocardiography	ECG abnormality, murmur, symptoms of heart failure, suspected LVH; LV mass, diastolic function, EF, LA size, aortic root
Fundoscopy (dilated)	Grade 2/3 hypertension, diabetes, visual symptoms; mandatory in hypertensive emergency
Renal ultrasound ± Doppler	Abnormal renal function, haematuria, suspected renovascular or polycystic disease
Carotid ultrasound	Bruit, TIA/stroke; plaque = high risk
Ankle–brachial index	Claudication, absent pulses; ABI <0.9 = PAD
Pulse wave velocity	Arterial stiffness (carotid–femoral PWV >10 m/s)
Chest X-ray	Cardiomegaly, pulmonary congestion, coarctation signs, aortic widening
Holter / ABPM	Palpitations, suspected AF; BP phenotype
Brain CT/MRI; cognitive testing	Neurological symptoms, cognitive decline
Secondary hypertension tests	ARR, metanephrines, cortisol tests, sleep study — see Chapter 6

Criteria for hypertension-mediated organ damage (HMOD)

Organ	Criterion
Heart — ECG LVH	Sokolow–Lyon: SV1 + RV5 or RV6 **>35 mm**; RaVL ≥11 mm; Cornell voltage: RaVL + SV3 **>28 mm (men), >20 mm (women)**; Cornell product >2440 mm·ms
Heart — echo LVH	LV mass index **>115 g/m² (men), >95 g/m² (women)** (or >50 / >47 g/m$^{2.7}$); relative wall thickness >0.42 = concentric; LA volume index >34 ml/m²; E/e′ >14
Kidney	eGFR **<60 ml/min/1.73 m²**; UACR **≥30 mg/g** (moderately increased 30–300, severely increased >300)
Arteries	Carotid plaque; carotid–femoral PWV >10 m/s; ABI <0.9; coronary artery calcium
Eye	Haemorrhages, exudates, cotton-wool spots, papilloedema
Brain	Silent infarcts, lacunes, white-matter hyperintensities, microbleeds on MRI; cognitive impairment

Cardiovascular risk stratification (ESH 2023)

Risk factors to count: age (men >55, women >65 years), male sex, smoking/tobacco, dyslipidaemia, family history of premature CVD, obesity (especially central), sedentary lifestyle, raised uric acid, heart rate >80/min, early menopause, psychosocial factors, pulse pressure ≥60 mmHg in the elderly. Hypertension disease stage: **Stage 1** uncomplicated; **Stage 2** HMOD, CKD stage 3 or diabetes without organ damage; **Stage 3** established CVD, CKD stage 4–5 or diabetes with organ damage.

Disease stage	Other risk factors, HMOD or disease	High-normal 130–139/85–89	Grade 1 140–159/90–99	Grade 2 160–179/100–109	Grade 3 ≥180/≥110
Stage 1 (uncomplicated)	No other risk factors	Low	Low	Moderate	High
	1 or 2 risk factors	Low	Moderate	Moderate–High	High
	≥3 risk factors	Low–Moderate	Moderate–High	High	High
Stage 2	HMOD, CKD 3 or diabetes without organ damage	Moderate–High	High	High	High–Very high

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Disease stage	Other risk factors, HMOD or disease	High-normal 130–139/85–89	Grade 1 140–159/90–99	Grade 2 160–179/100–109	Grade 3 ≥180/≥110
Stage 3	Established CVD, CKD 4–5, diabetes with organ damage	Very high	Very high	Very high	Very high

For numerical 10-year risk in India use the WHO/ISH 2019 CVD risk charts (South Asia region, lab or non-lab version). The 2025 ACC/AHA guideline uses the PREVENT calculator (≥7.5% 10-year CVD risk = high risk); these US equations have not been validated in Indians and may underestimate risk.

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PART IV · CHAPTER 10

Complications of Hypertension — Overview and Cardiac Complications

Hypertension-mediated organ damage of the heart, coronaries and aorta

Complications at a glance

Organ	Complication	Mechanism
Heart	LVH, diastolic dysfunction, HFpEF, HFrEF, coronary artery disease, MI, atrial fibrillation, ventricular arrhythmias, sudden death	Pressure overload, accelerated atherosclerosis, reduced coronary reserve, fibrosis
Brain	Ischaemic stroke, intracerebral haemorrhage, subarachnoid haemorrhage, lacunar infarcts, vascular dementia, hypertensive encephalopathy, PRES	Atherosclerosis, lipohyalinosis, Charcot–Bouchard microaneurysms, loss of autoregulation
Kidney	Albuminuria, hypertensive nephrosclerosis, CKD, ESKD, malignant nephrosclerosis	Arteriolosclerosis, glomerular hyperfiltration and ischaemia
Eye	Hypertensive retinopathy, choroidopathy, optic neuropathy, retinal vein/artery occlusion	Arteriolar narrowing, ischaemia, breakdown of blood–retinal barrier
Blood vessels	Aortic dissection, abdominal aortic aneurysm, peripheral arterial disease, carotid stenosis	Medial degeneration, atherosclerosis
Others	Erectile dysfunction, increased risk of complications in pregnancy	Endothelial dysfunction

Hypertensive heart disease

Left ventricular hypertrophy (LVH)

- Adaptive thickening of the LV wall in response to raised afterload; present on echo in up to 30–40% of hypertensives (less often detected on ECG).
- **Patterns:** concentric remodelling (normal mass, increased wall thickness), concentric hypertrophy (commonest in hypertension), eccentric hypertrophy (volume overload, obesity).
- LVH is an independent predictor of heart failure, AF, ventricular arrhythmias, sudden death and stroke.
- **Regression** of LVH with treatment reduces events. Most effective: ACEi/ARB and CCBs, then diuretics; β-blockers least effective (atenolol).

Heart failure

- Hypertension is the commonest risk factor for heart failure. The usual sequence: LVH → diastolic dysfunction → **HFpEF** (EF ≥50%) → in some, LV dilatation or MI → **HFrEF** (EF ≤40%).
- Acute hypertensive heart failure (flash pulmonary oedema with very high BP) is a hypertensive emergency — IV nitrates and loop diuretic (Chapter 20).
- Management of hypertension with heart failure — Chapter 19.

Coronary artery disease

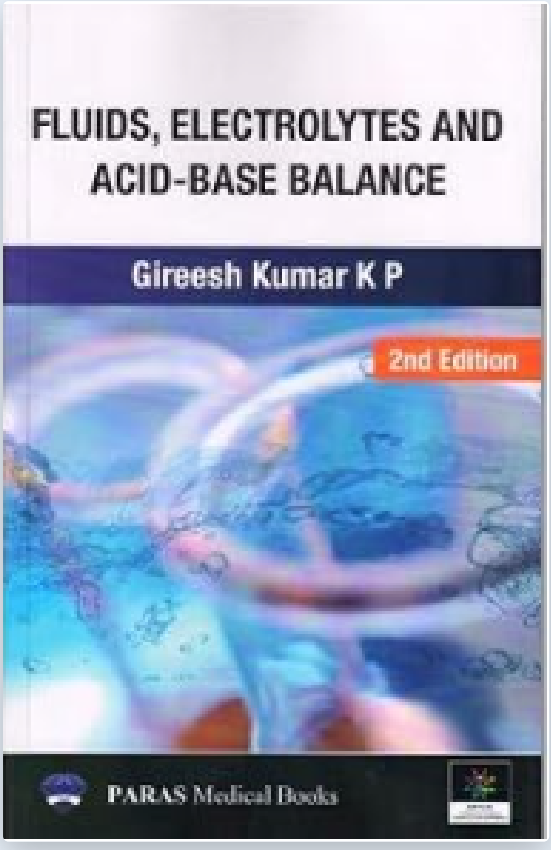
- Hypertension accelerates atherosclerosis and, with LVH, reduces coronary flow reserve — angina can occur even with normal coronaries.

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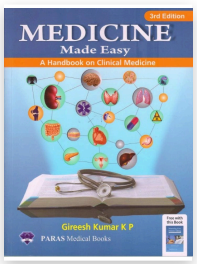
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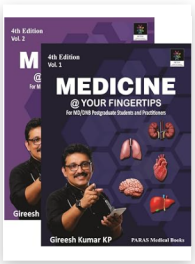
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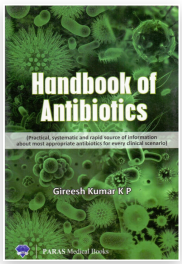
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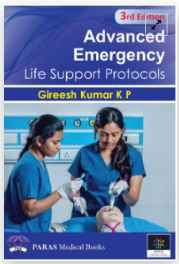
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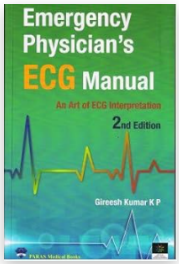
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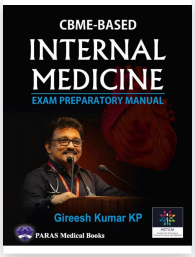
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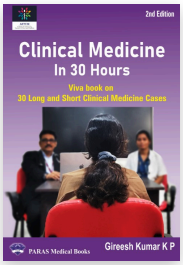
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- Risk of MI rises progressively with SBP; the "J-curve" concern applies mainly to diastolic BP <60–70 mmHg in patients with CAD.

Atrial fibrillation

Hypertension is the most common risk factor for AF (LA enlargement, fibrosis). Hypertension also adds 1 point to the CHA_2DS_2-VASc score — most hypertensive patients with AF need anticoagulation.

Aortic and vascular complications

Complication	Features	Key management point
Acute aortic dissection	Sudden tearing chest/back pain, pulse or BP difference between arms, AR murmur, wide mediastinum	Emergency: IV β-blocker (esmolol/labetalol) first to HR <60, then vasodilator; target SBP **100–120 mmHg within 20 minutes**; urgent CT and surgical opinion
Abdominal aortic aneurysm	Pulsatile abdominal mass, often asymptomatic; smokers, men >65	Ultrasound screening; strict BP control; surgery if ≥5.5 cm or rapidly enlarging
Peripheral arterial disease	Claudication, absent pulses, ABI <0.9	Target <130/80; ACEi/ARB; statin; antiplatelet; β-blockers are not contraindicated
Carotid disease	Bruit, TIA, amaurosis fugax	Statin, antiplatelet, BP control; endarterectomy for symptomatic stenosis ≥70%

KEY POINT

Look for HMOD at diagnosis and once a year: **ECG** and **UACR** are cheap, available everywhere in India and change management. Finding LVH or albuminuria moves the patient into a higher risk group and calls for earlier and more intensive treatment.

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PART IV · CHAPTER 11

Cerebrovascular and Neurological Complications

Stroke, intracerebral haemorrhage, hypertensive encephalopathy and vascular cognitive impairment

Stroke

- Hypertension is the **most important modifiable risk factor for stroke**; it accounts for more than half of all strokes. South Asians have a high proportion of haemorrhagic and lacunar strokes.
- **Ischaemic stroke** — large-artery atherosclerosis, cardio-embolism (AF), and small-vessel (lacunar) disease due to lipohyalinosis of penetrating arteries.
- **Intracerebral haemorrhage (ICH)** — rupture of small penetrating arteries (Charcot–Bouchard microaneurysms); typical sites are the putamen, thalamus, pons and cerebellum.
- Each 10 mmHg reduction in SBP lowers stroke risk by about one-quarter to one-third.

BP management in acute stroke

Situation	BP recommendation
Acute ischaemic stroke — eligible for thrombolysis	Lower BP to **<185/110** before thrombolysis and keep **<180/105** for 24 hours after (IV labetalol or nicardipine)
Mechanical thrombectomy	Keep ≤180/105 during and for 24 h after; avoid aggressive lowering below ~140 after successful recanalisation
Acute ischaemic stroke — not thrombolysed	Do not lower unless BP **≥220/120**; then reduce by about **15% in the first 24 hours**. Restart oral drugs after 24–72 h when neurologically stable
Intracerebral haemorrhage	If SBP 150–220: lower to a target of about **140 mmHg** (range 130–150) within 1 hour, smoothly; avoid falls >70 mmHg and SBP <130
Subarachnoid haemorrhage	Keep SBP **<160** until the aneurysm is secured; nimodipine 60 mg 4-hourly for 21 days (for vasospasm)
Secondary prevention after stroke/TIA	Target **<130/80**; ACEi/ARB + thiazide-like diuretic (PROGRESS: perindopril + indapamide) or CCB; plus statin, antiplatelet or anticoagulant

Hypertensive encephalopathy and PRES

- Sudden severe rise in BP overwhelms cerebral autoregulation → hyperperfusion, cerebral oedema (especially posterior white matter).
- **Features:** severe headache, vomiting, confusion, visual disturbance, seizures, coma; papilloedema may be present. MRI shows vasogenic oedema in parieto-occipital regions — posterior reversible encephalopathy syndrome (PRES).
- **Causes:** malignant hypertension, eclampsia, renal disease, immunosuppressants (tacrolimus), chemotherapy, sudden withdrawal of clonidine.
- **Treatment:** reduce MAP by 20–25% in the first hour with IV labetalol or nicardipine (Chapter 20). Symptoms usually reverse within days.

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Vascular cognitive impairment and dementia

- Midlife hypertension increases the risk of vascular dementia and Alzheimer's disease in later life. Small-vessel disease (white-matter hyperintensities, lacunes, microbleeds) is the main substrate.
- Intensive BP control reduced mild cognitive impairment in SPRINT-MIND. Treating hypertension is one of the most effective dementia-prevention strategies available.
- In patients with established dementia, avoid orthostatic hypotension and falls; simplify regimens; involve caregivers.

CLINICAL PEARL

A patient who presents with "headache and high BP" is far more often in pain (headache causing BP rise) than in hypertensive encephalopathy. Look for altered sensorium, papilloedema and focal signs before labelling an emergency — and treat the pain first.

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PART IV · CHAPTER 12

Renal and Ocular Complications

Hypertensive nephrosclerosis, CKD progression and hypertensive retinopathy

Hypertension and the kidney — a vicious cycle

Hypertension damages the kidney, and kidney disease raises BP. CKD is both the commonest secondary cause and a major complication of hypertension. In India, diabetes and hypertension together cause more than 60% of end-stage kidney disease.

Benign (hypertensive) nephrosclerosis

- Hyaline arteriolosclerosis of afferent arterioles → glomerular ischaemia and sclerosis, tubular atrophy; small, smooth, symmetrical kidneys.
- Slowly rising creatinine, mild proteinuria (usually <1 g/day), bland urine sediment. Albuminuria appears early and is a marker of systemic vascular damage.

Malignant nephrosclerosis

- Fibrinoid necrosis and "onion-skin" hyperplastic arteriolosclerosis with severe hypertension; rapidly progressive kidney failure, haematuria, proteinuria and microangiopathic haemolysis.
- Renal function may partly recover with careful BP control; some patients need temporary dialysis.

Staging CKD (KDIGO) — GFR and albuminuria

G stage	eGFR (ml/min/1.73 m²)
G1	≥90 (with markers of damage)
G2	60–89
G3a	45–59
G3b	30–44
G4	15–29
G5	<15 or dialysis

A stage	UACR	Description
A1	<30 mg/g (<3 mg/mmol)	Normal to mildly increased
A2	30–300 mg/g (3–30 mg/mmol)	Moderately increased (old "microalbuminuria")
A3	>300 mg/g (>30 mg/mmol)	Severely increased (old "macroalbuminuria")

Management of hypertension in CKD and proteinuria is detailed in Chapter 19.

Hypertensive retinopathy

The retina is the only place where small arteries can be seen directly. Fundoscopy should be performed in every patient with grade 2–3 hypertension, diabetes, visual symptoms or suspected hypertensive emergency.

Keith–Wagener–Barker (KWB) classification (1939)

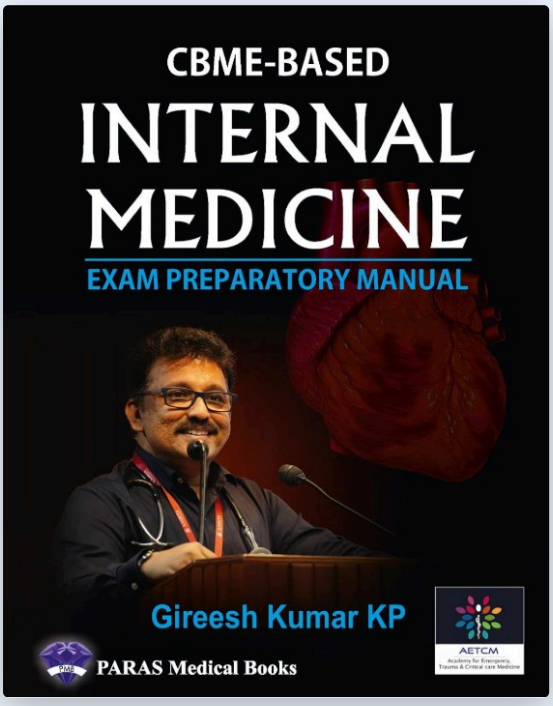
Grade	Fundus findings	Significance
I	Mild generalised arteriolar narrowing; increased light reflex ("silver/copper wiring")	Chronic hypertension
II	More marked narrowing, focal constriction and **arteriovenous nicking**	Chronic hypertension; higher CV risk

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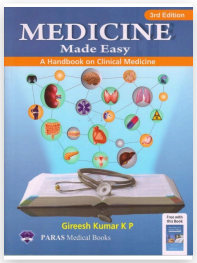
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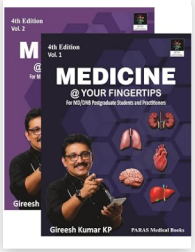
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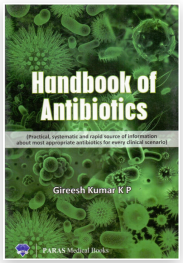
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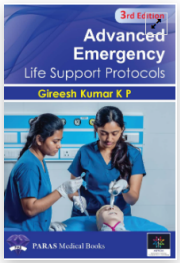
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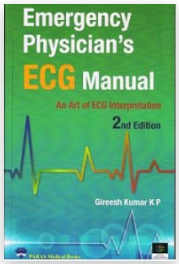
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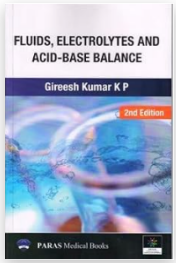
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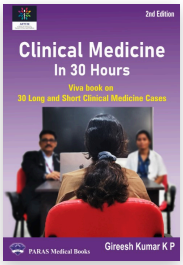
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Grade	Fundus findings	Significance
III	Grade II plus **flame-shaped haemorrhages, cotton-wool spots, hard exudates** (macular star)	Severe/accelerated hypertension
IV	Grade III plus **papilloedema** (optic disc swelling)	Malignant hypertension — emergency

Mitchell–Wong (simplified) classification (2004)

Grade	Signs	Association
None	No detectable signs	—
Mild	Generalised and focal arteriolar narrowing, AV nicking, opacity of arteriolar wall	Modest increase in risk of stroke, CAD and death
Moderate	Haemorrhages (blot, dot, flame), microaneurysms, cotton-wool spots, hard exudates	Strong association with stroke, cognitive decline and CV death
Malignant	Moderate retinopathy plus optic disc swelling	Strong association with death — hypertensive emergency

Other ocular complications

- **Hypertensive choroidopathy** — Elschnig spots (choroidal infarcts), Siegrist streaks, serous retinal detachment (pre-eclampsia, malignant hypertension).
- **Retinal vein occlusion** and **retinal artery occlusion** — hypertension is the main risk factor.
- **Non-arteritic anterior ischaemic optic neuropathy** — sudden painless visual loss; avoid overly aggressive night-time BP lowering.
- Hypertension accelerates **diabetic retinopathy** and macular oedema.

Management of retinal damage

KEY POINTS

- Mild–moderate retinopathy: control BP to target with standard therapy; regular fundus review (yearly). Changes of grade I–II regress slowly or not at all; haemorrhages and exudates usually resolve within weeks of control.
- Grade III–IV (malignant) retinopathy with severe BP: admit; **lower MAP by no more than 20–25% over several hours** with IV labetalol or nicardipine — sudden falls can cause optic nerve ischaemia and blindness.
- Refer to ophthalmology for vein/artery occlusion, macular oedema, visual loss or unexplained findings. Anti-VEGF or laser therapy may be needed.
- Control associated diabetes, lipids and smoking; check for secondary hypertension when retinopathy is severe at a young age.

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PART V · CHAPTER 13

When to Treat and How Low to Go

Thresholds for starting drugs and BP targets by patient group

Principles

- The decision to start drugs depends on **BP level** and **total cardiovascular risk** (Chapter 9), not on BP alone.
- **Lifestyle measures are for everyone** — from high-normal BP onwards, and alongside drugs, never instead of indicated drugs.
- Aim to reach the target within **3 months** of starting treatment.
- Targets are reached in two steps: first below 140/90 in all, then lower (usually <130/80) if well tolerated.

Thresholds for starting drug treatment

BP / patient group	ESH 2023 / ISH 2020 / Indian practice	ACC/AHA 2025
Grade 2–3 (≥160/100)	Start drugs immediately (usually two-drug SPC) with lifestyle	Stage 2 (≥140/90): start drugs — two first-line drugs, preferably SPC
Grade 1 (140–159/90–99), high risk, HMOD, diabetes, CKD or CVD	Start drugs immediately	Start drugs
Grade 1, low–moderate risk, no HMOD	Lifestyle for 3–6 months; start drugs if still ≥140/90	Stage 1 (130–139/80–89) with CVD, diabetes, CKD or PREVENT ≥7.5%: drugs. Others: lifestyle 3–6 months, then drugs if ≥130/80
High-normal (130–139/85–89)	Lifestyle; drugs only with very high risk due to established CVD (especially CAD)	As stage 1 above
Age ≥80 years	Start drugs if SBP ≥160 (consider 140–159 if fit)	Same as younger adults if fit; individualise if frail
Frail / limited life expectancy	Individualise; consider tolerance, orthostatic BP, falls	Shared decision
Pregnancy	Start drugs at ≥140/90 (Chapter 22)	≥140/90

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Blood pressure targets

Group	Office BP target	Comment
Most adults 18–64 years	**<130/80** (SBP 120–129 if tolerated)	ESC 2024 and ACC/AHA 2025: SBP 120–129; ACC/AHA encourages <120 when appropriate
65–79 years	<140/80 first, then **<130/80** if tolerated	Check standing BP; avoid orthostatic hypotension
≥80 years	SBP **140–150**; 130–139 if well tolerated; DBP <80	HYVET target <150/80
Diabetes	**<130/80** (not below 120/70)	ADA, ESH; BPROAD supports SBP <120 in selected patients
CKD (with or without proteinuria)	**<130/80**; KDIGO 2021: SBP <120 if tolerated (standardised measurement)	Monitor eGFR and K^+

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Group	Office BP target	Comment
CAD	**<130/80**; avoid DBP <60–70	J-curve for diastolic BP in CAD
After stroke/TIA	**<130/80**	Start after the acute phase
Heart failure	<130/80 (guideline-directed therapy titrated to tolerance)	BP often low in HFrEF on full therapy
Pregnancy	**<140/90**; DBP ~85	CHAP trial
Programme target (IHCI, NP-NCD)	<140/90	Simplicity for primary care; lower in high-risk groups

Home BP targets are about 5 mmHg lower than office targets for the ESH system (home <135/85 corresponds to office <140/90) and equal to office values in the ACC/AHA system (130/80).

CAUTION — DO NOT OVER-TREAT

- Avoid SBP <120 in the frail elderly, those with orthostatic hypotension, recurrent falls, severe carotid stenosis or limited life expectancy.
- In patients with CAD, avoid DBP <60–70 mmHg (coronary perfusion occurs in diastole).
- Symptoms of over-treatment: dizziness, fatigue, falls, syncope, rising creatinine. Reduce the dose if symptomatic.

Follow-up schedule

Situation	Review interval
BP ≥180/110 without emergency features (treatment started)	Within 1 week
After starting or changing a drug	2–4 weeks (check K^+/creatinine 1–2 weeks after ACEi/ARB/diuretic/MRA)
Lifestyle only (grade 1, low risk)	3 months, then 6 months
Controlled on stable treatment	Every 3–6 months; yearly UACR, creatinine, K^+, glucose, lipids, ECG
High-normal BP	Every year

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PART V · CHAPTER 14

Lifestyle Modifications

Non-drug treatment for every patient — salt, weight, diet, exercise, alcohol, tobacco, sleep and stress

Lifestyle changes prevent hypertension, lower BP in people with hypertension, reduce the number of drugs needed and lower overall cardiovascular risk. Each measure lowers SBP by about 4–11 mmHg; combined measures have additive effects. They should be advised **at every visit**, in simple, specific and culturally appropriate language.

Lifestyle measures and expected BP reduction

Intervention	Recommendation	Approximate SBP fall in hypertensives
Weight loss	Aim for ideal body weight (BMI 18.5–22.9 kg/m² for Indians); waist <90 cm (men), <80 cm (women). Even 5% loss helps	~1 mmHg per kg lost (≈5 mmHg)
Healthy diet (DASH-type)	Rich in vegetables, fruits, whole grains, pulses, low-fat dairy, nuts; low in saturated fat, red/processed meat, sugar and refined flour	~11 mmHg
Salt reduction	Salt <5 g/day (sodium <2 g/day); ideal sodium <1.5 g/day; at least reduce by 1 g sodium/day	~5–6 mmHg
Increase potassium	3.5–5 g/day from food (fruits, vegetables, coconut water, pulses) or potassium-enriched salt substitute — **not** in CKD or with hyperkalaemia-causing drugs without monitoring	~4–5 mmHg
Aerobic exercise	Brisk walking, cycling, swimming 30 minutes, 5–7 days a week (150 min/week moderate)	~5–8 mmHg
Dynamic resistance exercise	2–3 days a week (6 exercises, 3 sets, 10 repetitions)	~4 mmHg
Isometric exercise	Handgrip or wall-sit: 4 × 2 minutes, 1-minute rest, 3 sessions a week	~5 mmHg
Alcohol	Best avoided. If drinking: ≤2 standard drinks/day (men), ≤1 (women); avoid binge drinking	~4 mmHg
Stop tobacco	Smoking, beedi and smokeless tobacco — complete cessation; offer counselling and nicotine replacement/varenicline	Large reduction in CV risk
Stress, yoga, breathing	Yoga, meditation, slow breathing exercises 15–30 min/day	~3–5 mmHg
Sleep	7–9 hours; treat sleep apnoea (CPAP)	Variable

Salt — the most important single change for Indians

Know the numbers

- 1 level teaspoon of salt ≈ 5 g salt ≈ 2 g sodium — this is the **whole day's** allowance, including salt already present in food.
- Average Indian intake is 8–11 g/day, much of it added during cooking and from "hidden" sources.
- Salt preference falls within 6–8 weeks of reduction — food tastes normal again.

High-salt foods to limit

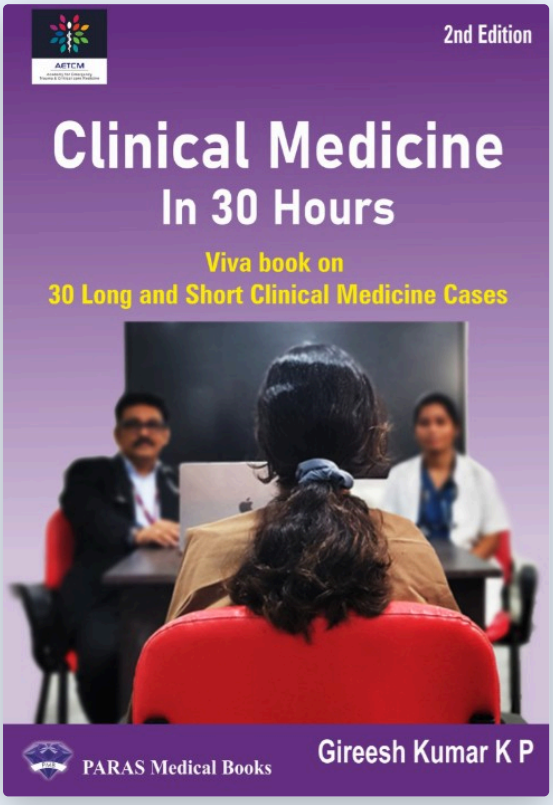
Food group	Examples
Preserved	Pickles (achar), papad, chutney powders, dried and salted fish
Snacks	Chips, namkeen, mixture, salted nuts, bhujia
Bakery	Bread, biscuits, puffs, cakes (baking soda = sodium)

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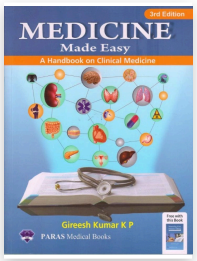
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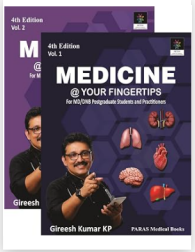
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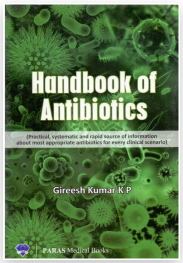
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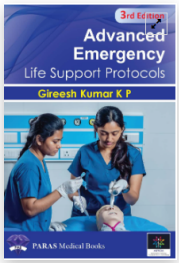
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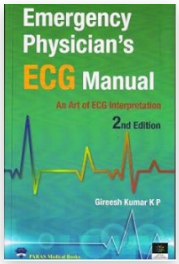
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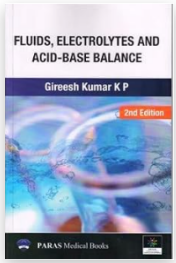
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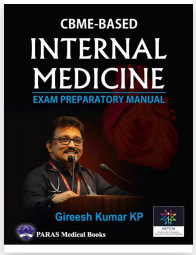
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Practical tips

- Use a measured spoon in the kitchen; reduce salt gradually.
- No salt shaker on the table; avoid extra salt on fruits, salads and curd.
- Flavour with lemon, herbs, garlic, ginger, pepper, tamarind, curry leaves.
- Read labels: sodium >600 mg/100 g = high.

Food group	Examples
Processed	Instant noodles, soups, sauces, ketchup, soy sauce, processed cheese, sausages
Restaurant/fast food	Biryani, fried rice, pizza, burgers, chaat
Hidden sodium	Ajinomoto (MSG), baking powder, some antacids and effervescent tablets

Potassium-rich Indian foods

Banana, orange, sweet lime (mosambi), papaya, guava, tender coconut water, tomato, potato and sweet potato (boiled), spinach and other green leafy vegetables, beans, dals and pulses, curd, dates and dry fruits. **Caution:** restrict potassium in CKD stage 4–5 and when serum K^+ is high, especially with ACEi, ARB, MRA or potassium-sparing diuretics. Potassium-enriched salt substitutes (e.g., 75% NaCl/25% KCl) reduced stroke and death in the SSaSS trial but should be avoided in advanced CKD.

Exercise prescription (FITT)

Element	Aerobic	Resistance
Frequency	5–7 days/week	2–3 days/week (non-consecutive)
Intensity	Moderate — can talk but not sing; 50–70% of maximum heart rate	Moderate load, 8–12 repetitions
Time	30–60 min/day (can be split into 10-minute bouts)	20–30 min
Type	Brisk walking, cycling, swimming, dancing, jogging	Bands, weights, body-weight exercises; isometric handgrip

CAUTION

Patients with BP ≥180/110 should start drugs and get BP under better control before vigorous or heavy resistance exercise. Patients with CAD or symptoms need assessment first. Avoid breath-holding (Valsalva) during lifting.

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Making it stick — counselling tips

- Agree on one or two specific changes per visit ("salt spoon", "30-minute evening walk").
- Involve the person who cooks at home; family-based advice works better.
- Use home BP records to show the benefit — this motivates continuing.
- Reassure that lifestyle and medicines work together; lifestyle may allow fewer or lower-dose drugs later.

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PART V · CHAPTER 15

First-Line Drugs

ACE inhibitors, ARBs, calcium channel blockers and thiazide/thiazide-like diuretics — the "A, C and D" of hypertension

Four classes have proven, similar reductions in cardiovascular events and form the backbone of treatment: **A** = ACE inhibitors or angiotensin receptor blockers (never both), **C** = dihydropyridine calcium channel blockers, **D** = thiazide or thiazide-like diuretics. **β-blockers** are added at any step when there is a specific indication (Chapter 16). Most patients need two or three of these drugs.

ACE inhibitors (ACEi)

Mechanism

Block conversion of angiotensin I to angiotensin II → vasodilatation, lower aldosterone, reduced intraglomerular pressure; bradykinin accumulates (cough, angioedema).

Preferred in

- Diabetes, albuminuria/proteinuria, CKD
- HFrEF, post-MI LV dysfunction, CAD
- Stroke prevention (with indapamide)
- LVH, metabolic syndrome

Adverse effects

- **Dry cough** (5–20%; switch to ARB)
- **Angioedema** (rare, can be fatal; never re-challenge)
- Hyperkalaemia, rise in creatinine (accept up to 30%)
- First-dose hypotension in volume-depleted patients
- Rash, taste disturbance (captopril)

Contraindications

- **Pregnancy** (teratogenic) — stop at conception
- History of angioedema; bilateral renal artery stenosis
- Combination with ARB or aliskiren; sacubitril within 36 h

Angiotensin II receptor blockers (ARBs)

Mechanism

Block the AT1 receptor; same benefits as ACEi without bradykinin effects. Very well tolerated — the best adherence of all classes.

Preferred in

- Same indications as ACEi; ACEi-induced cough
- Gout (**losartan** is uricosuric)
- Telmisartan has a long half-life (24 h) — good morning coverage; widely used in India

Adverse effects

- Hyperkalaemia, rise in creatinine
- Dizziness; angioedema (much rarer than ACEi)
- Olmesartan — rare sprue-like enteropathy (chronic diarrhoea, weight loss)

Contraindications

- **Pregnancy**; bilateral renal artery stenosis
- Combination with ACEi or aliskiren
- Telmisartan: caution in biliary obstruction/severe hepatic disease

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Calcium channel blockers (CCBs)

	Dihydropyridines (DHP)	Non-dihydropyridines (non-DHP)
Drugs	Amlodipine, S-amlodipine, nifedipine (extended release), felodipine, cilnidipine, lercanidipine, benidipine	Verapamil, diltiazem
Main action	Arterial vasodilatation (little effect on heart)	Slow heart rate and AV conduction; reduce contractility; mild vasodilatation
Preferred in	Elderly, isolated systolic hypertension, angina, PAD, carotid atherosclerosis, pregnancy (nifedipine), CKD (with ACEi/ARB), transplant	Angina, rate control in AF, supraventricular tachycardia, when β-blocker contraindicated (asthma); additional antiproteinuric effect
Adverse effects	**Ankle oedema** (dose-related; less with cilnidipine, lercanidipine, S-amlodipine and when combined with ACEi/ARB), flushing, headache, palpitations, gum hypertrophy	Bradycardia, AV block, **constipation** (verapamil), oedema (diltiazem), worsening heart failure
Avoid	Short-acting (immediate-release) nifedipine — reflex tachycardia, ischaemia, stroke	**HFrEF**, 2nd/3rd-degree AV block, sick sinus syndrome, combination with β-blockers; many CYP3A4 interactions

Thiazide and thiazide-like diuretics

Drugs

- **Thiazide-like** (preferred — longer acting, stronger outcome data): **chlorthalidone**, **indapamide**
- **Thiazide-type**: hydrochlorothiazide
- Metolazone — for resistant oedema and advanced CKD

Mechanism

Inhibit the Na^+–Cl^- co-transporter in the distal convoluted tubule; long-term effect is reduced peripheral resistance.

Preferred in

- Elderly, isolated systolic hypertension, heart failure, stroke prevention, osteoporosis (calcium retention), salt-sensitive patients
- Chlorthalidone remains effective in CKD stage 4 (CLICK trial)

Adverse effects (dose-related)

- **Hypokalaemia**, hypomagnesaemia — check K^+ at 2–4 weeks
- **Hyponatraemia** — especially elderly women; can be severe
- **Hyperuricaemia and gout**
- Hyperglycaemia, raised lipids (small at low doses)
- Hypercalcaemia, photosensitivity, erectile dysfunction

Contraindications / cautions

- Gout, symptomatic hyponatraemia, hypokalaemia
- Use lowest effective dose (chlorthalidone 12.5 mg; HCTZ 12.5–25 mg)
- Combine with ACEi/ARB to reduce K^+ loss

Quick comparison of first-line classes

Feature	ACEi / ARB	DHP CCB	Thiazide-like diuretic
Best evidence in	Diabetes, CKD, HF, post-MI	Elderly, stroke prevention, angina	Elderly, ISH, HF prevention
Main metabolic effect	K^+ ↑	Neutral	K^+ ↓, Na^+ ↓, uric acid ↑, glucose ↑
Lab monitoring	Creatinine and K^+ at 1–2 weeks	None routine	Na^+, K^+ at 2–4 weeks
Pregnancy	Contraindicated	Nifedipine safe	Generally avoided

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Feature	ACEi / ARB	DHP CCB	Thiazide-like diuretic
Cost / availability in India	Low; widely available (telmisartan, losartan, enalapril, ramipril)	Very low (amlodipine)	Low (chlorthalidone, indapamide, HCTZ)

Doses for every drug are in the master table, Chapter 18.

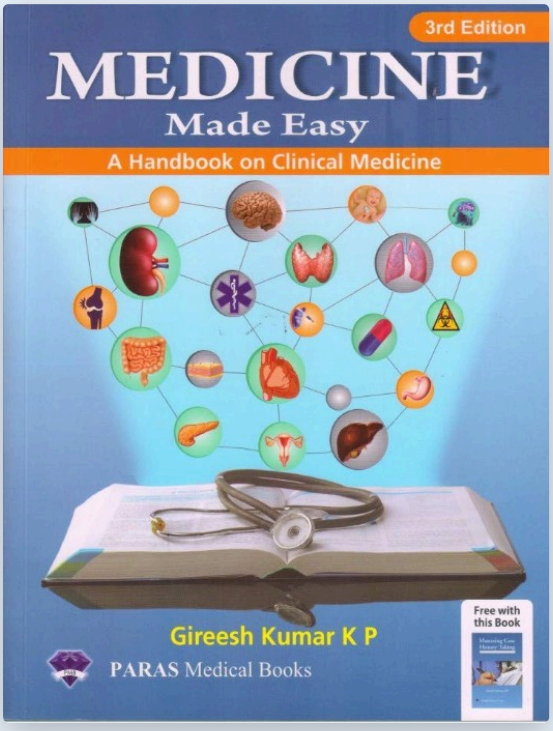
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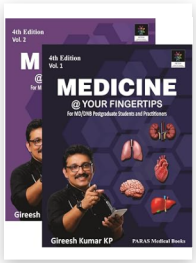
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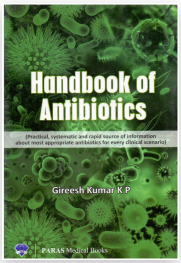
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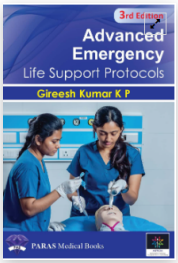
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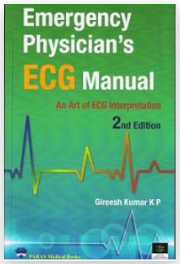
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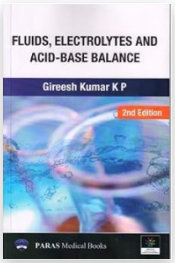
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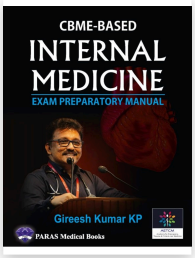
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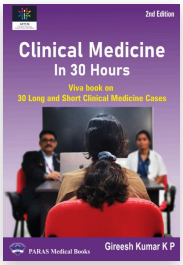
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PART V · CHAPTER 16

Second- and Third-Line Drugs

β-blockers, mineralocorticoid receptor antagonists, loop and potassium-sparing diuretics, α-blockers, central agents, vasodilators and newer agents

Beta-blockers

Not preferred as initial therapy for uncomplicated hypertension (less stroke protection than other classes, especially **atenolol**), but useful **at any step** when there is a specific indication. Prefer vasodilating or cardioselective agents: **nebivolol, bisoprolol, metoprolol succinate, carvedilol**.

Specific indications

- Angina, post-MI, HFrEF (bisoprolol, carvedilol, metoprolol succinate, nebivolol)
- AF rate control, tachyarrhythmias
- Young patients with hyperadrenergic hypertension, anxiety, essential tremor, migraine (propranolol)
- Women planning pregnancy (labetalol), aortic dissection, aortic aneurysm, hyperthyroidism

Adverse effects

- Fatigue, bradycardia, heart block, cold extremities
- Bronchospasm (non-selective), masking of hypoglycaemia
- Weight gain, glucose intolerance (especially with thiazides)
- Erectile dysfunction, sleep disturbance, depression
- Rebound tachycardia/angina on abrupt withdrawal — taper

Contraindications

- Asthma (non-selective agents), 2nd/3rd-degree AV block, sick sinus, severe bradycardia
- Untreated phaeochromocytoma; cocaine toxicity
- Combination with verapamil/diltiazem

Mineralocorticoid receptor antagonists (MRAs)

- **Spironolactone** — the preferred **fourth drug** for resistant hypertension (PATHWAY-2) and drug of choice for bilateral adrenal hyperplasia. Also indicated in HFrEF.
- **Eplerenone** — more selective, no gynaecomastia; shorter-acting and less potent for BP. **Finerenone** — non-steroidal; reduces kidney and CV events in type 2 diabetes with albuminuria (modest BP effect).
- **Adverse effects:** hyperkalaemia, rise in creatinine; gynaecomastia, breast pain, menstrual irregularity, erectile dysfunction (spironolactone).
- **Use with care** if eGFR <45 or K^+ >4.5 mmol/l; avoid if K^+ >5.0 or eGFR <30. Check K^+ and creatinine at 1 week, 4 weeks, then every 3–6 months.

Other diuretics

Class	Role	Cautions
Loop diuretics — furosemide, torsemide	Replace thiazides when eGFR <30 (except chlorthalidone, which still works), in heart failure and fluid overload. Torsemide is once-daily and better absorbed. Furosemide is short-acting — give twice daily for BP	Hypokalaemia, hyponatraemia, dehydration, ototoxicity (high IV doses), gout
Amiloride	Alternative fourth drug in resistant hypertension; Liddle syndrome; combined with thiazides to prevent hypokalaemia	Hyperkalaemia, especially with ACEi/ARB and CKD

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Alpha-1 blockers

- **Prazosin, doxazosin, terazosin** — peripheral vasodilatation; useful in **benign prostatic hyperplasia**, resistant hypertension, phaeochromocytoma (pre-operative).
- Not for monotherapy (ALLHAT: more heart failure with doxazosin).
- **Adverse effects:** first-dose syncope (give first dose at bedtime), orthostatic hypotension (elderly), dizziness, nasal congestion, floppy-iris syndrome during cataract surgery, stress incontinence in women.

Centrally acting agents

Drug	Use	Adverse effects / cautions
Methyldopa	Pregnancy (long safety record)	Sedation, depression, dry mouth, hepatitis, Coombs-positive haemolytic anaemia; avoid postpartum (depression)
Clonidine	Resistant hypertension, adjunct; opioid/alcohol withdrawal	Sedation, dry mouth, bradycardia; **severe rebound hypertension** if stopped suddenly (especially with β-blockers) — taper
Moxonidine	Adjunct in resistant hypertension, metabolic syndrome	Dry mouth, drowsiness; avoid in heart failure (MOXCON: increased mortality)

Direct vasodilators

- **Hydralazine** — pregnancy (oral and IV), resistant hypertension, HFrEF (with isosorbide dinitrate). Causes reflex tachycardia and fluid retention — combine with a β-blocker and diuretic. Drug-induced lupus at doses >200 mg/day, especially in slow acetylators.
- **Minoxidil** — very potent; reserved for severe resistant hypertension and advanced CKD. Must be given with a β-blocker and loop diuretic. Hirsutism, marked fluid retention, pericardial effusion.

Other and newer agents

Agent	Mechanism / status	Comment
Aliskiren	Direct renin inhibitor	Lowers BP but no outcome benefit; never combine with ACEi/ARB in diabetes/CKD (ALTITUDE)
Sacubitril–valsartan (ARNI)	Neprilysin inhibitor + ARB	First choice in HFrEF; also lowers BP strongly (approved for hypertension in some countries). 36-hour gap after ACEi
SGLT2 inhibitors	Glycosuria, natriuresis	Lower SBP 3–5 mmHg; strong kidney and heart failure benefit — add in diabetes, CKD, HF
GLP-1 receptor agonists / tirzepatide	Weight loss	Lower SBP 3–7 mmHg in obese patients
Aprocitentan	Dual endothelin receptor antagonist (approved in USA, 2024)	Resistant hypertension; teratogenic; oedema, anaemia. Not yet available in India
Aldosterone synthase inhibitors (baxdrostat, lorundrostat)	Block aldosterone production	Positive phase 3 trials in uncontrolled/resistant hypertension; investigational
Zilebesiran	siRNA against hepatic angiotensinogen; injection every 3–6 months	Investigational

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Agent	Mechanism / status	Comment
Renal denervation	Catheter-based ablation of renal sympathetic nerves	Lowers SBP ~5–10 mmHg; option for resistant or drug-intolerant patients in expert centres

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PART V · CHAPTER 17

Treatment Strategy — Starting and Stepping Up

Initial combination therapy, single-pill combinations, how to add the next drug, and the IHCI protocol

Core principles of drug treatment

- **Start with two drugs** (low-dose combination) in most patients — faster control, fewer side-effects than high-dose monotherapy, and better adherence. Adding a second drug lowers BP about **five times more** than doubling the dose of the first.
- **Prefer single-pill combinations (SPC)** — fewer tablets, better adherence and control.
- Use **long-acting once-daily** drugs. Morning or bedtime dosing gives similar outcomes (TIME trial) — choose the time the patient will remember.
- Review every **2–4 weeks** and step up until the target is reached — aim for control within **3 months**.
- Before stepping up, always check **adherence**, measurement technique, salt intake and interfering drugs (NSAIDs).

The standard treatment algorithm (ESH 2023 / ISH 2020 / ACC/AHA 2025)

Lifestyle advice for all + identify compelling indications (Chapter 19)

↓

STEP 1 — Two-drug combination, low dose, preferably single pill

ACEi or ARB + DHP-CCB or **ACEi or ARB + thiazide-like diuretic**

Monotherapy instead if: grade 1 low-risk with BP <150/95, high-normal BP with very high risk, age ≥80 years, or frailty

↓

Not controlled after 2–4 weeks → increase to full dose of the two drugs (or go straight to Step 2)

↓

STEP 2 — Three-drug combination, preferably single pill

ACEi or ARB + DHP-CCB + thiazide-like diuretic

↓

Not controlled on full-dose triple therapy = resistant hypertension → confirm adherence, out-of-office BP, exclude secondary causes

↓

STEP 3 — Add spironolactone 12.5–50 mg

If not tolerated or K^+ high: amiloride, eplerenone, β-blocker, α-blocker, clonidine/moxonidine, hydralazine; refer to specialist

β-blocker can be added at any step for angina, post-MI, heart failure, AF rate control, or women planning pregnancy

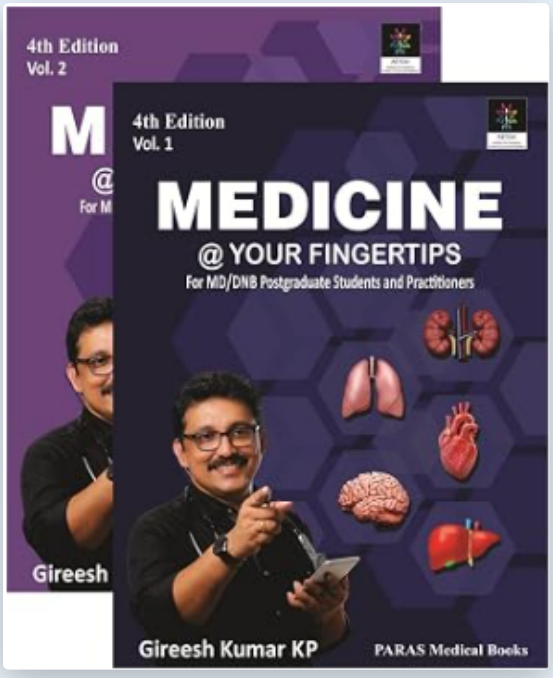
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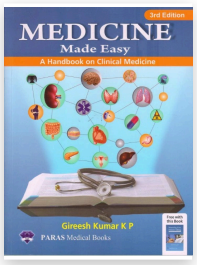
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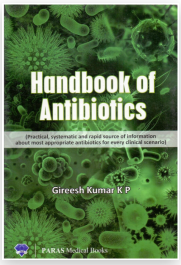
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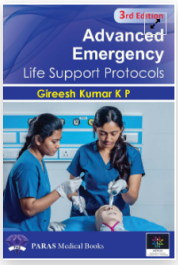
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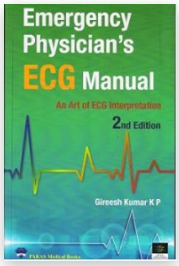
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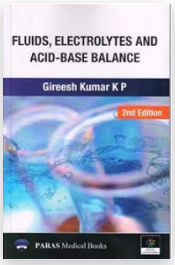
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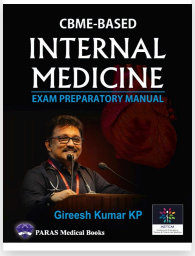
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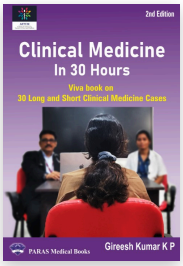
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How to add the next drug

Currently on (and uncontrolled)	Next step	Comment
ACEi or ARB alone	Add **amlodipine** (or chlorthalidone/indapamide)	Use SPC, e.g., telmisartan + amlodipine
CCB alone (e.g., amlodipine)	Add **ARB or ACEi**	Also reduces CCB ankle oedema
Thiazide alone	Add **ARB or ACEi**	Corrects diuretic hypokalaemia
β-blocker alone (no compelling indication)	Add **DHP-CCB** or switch to A + C	Avoid verapamil/diltiazem with β-blocker
A + C	Add **thiazide-like diuretic**	Triple SPC available
A + D	Add **DHP-CCB**	—
A + C + D (full dose)	Add **spironolactone** (K^+ ≤4.5, eGFR ≥45)	Check K^+ at 1 and 4 weeks
A + C + D + spironolactone	Add β-blocker, α-blocker or central agent; refer	Consider renal denervation in expert centres
eGFR <30 and on thiazide	Switch to **loop diuretic** (or keep chlorthalidone)	Torsemide once daily

Rational and irrational combinations

Category	Combinations
Preferred	ACEi/ARB + DHP-CCB; ACEi/ARB + thiazide-like diuretic; ACEi/ARB + CCB + diuretic
Acceptable	DHP-CCB + thiazide; β-blocker + DHP-CCB; β-blocker + diuretic (watch glucose); any + spironolactone (resistant); α-blocker add-on
Avoid	**ACEi + ARB**; ACEi/ARB + aliskiren; **β-blocker + verapamil/diltiazem** (bradycardia, heart block); β-blocker + clonidine (rebound on withdrawal); two drugs of the same class; ACEi/ARB + K^+-sparing diuretic + K^+ supplements without monitoring

Single-pill combinations commonly available in India (generic names)

Combination	Usual strengths (mg)
ARB/ACEi + CCB	
Telmisartan + amlodipine	40/5, 80/5, 40/10, 80/10
Olmesartan + amlodipine	20/5, 40/5, 40/10
Losartan + amlodipine	50/5
Telmisartan + cilnidipine	40/10
Perindopril + amlodipine	4/5, 8/5, 8/10 (perindopril erbumine) or 5/5, 10/10 (arginine)
Ramipril + amlodipine	2.5/5, 5/5
ARB/ACEi + diuretic	
Telmisartan + chlorthalidone	40/6.25, 40/12.5, 80/12.5
Telmisartan + hydrochlorothiazide	40/12.5, 80/12.5
Losartan + hydrochlorothiazide	50/12.5, 100/25
Olmesartan + hydrochlorothiazide	20/12.5, 40/12.5
Perindopril + indapamide	4/1.25, 8/2.5

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Combination	Usual strengths (mg)
Triple combinations	
Telmisartan + amlodipine + hydrochlorothiazide	40/5/12.5, 80/5/12.5
Telmisartan + amlodipine + chlorthalidone	40/5/6.25, 40/5/12.5
Olmesartan + amlodipine + hydrochlorothiazide	20/5/12.5, 40/5/12.5, 40/10/12.5
With β-blocker	
Amlodipine + metoprolol succinate	5/25, 5/50
Telmisartan + metoprolol succinate	40/25, 40/50
Amlodipine + atenolol	5/50 (not preferred)

Strengths vary by manufacturer — check the product available locally.

India Hypertension Control Initiative (IHCI) — standard protocol for primary care

Step	Protocol 1 ("AATTCC")	Protocol 2 ("ATTACC")
1	Amlodipine 5 mg	Amlodipine 5 mg
2	Amlodipine 10 mg	Amlodipine 5 mg + telmisartan 40 mg
3	Amlodipine 10 mg + telmisartan 40 mg	Amlodipine 5 mg + telmisartan 80 mg
4	Amlodipine 10 mg + telmisartan 80 mg	Amlodipine 10 mg + telmisartan 80 mg
5	+ Chlorthalidone 12.5 mg	+ Chlorthalidone 12.5 mg
6	Chlorthalidone 25 mg / refer to physician	Chlorthalidone 25 mg / refer to physician

Review monthly; step up if BP ≥140/90 (patient adherent). Women of child-bearing age: avoid telmisartan (use amlodipine, then add a β-blocker or refer). Refer at any step for pregnancy, BP ≥180/110 with symptoms, suspected secondary cause or side-effects. State protocols may differ slightly.

CLINICAL PEARL — BEFORE YOU ADD A FOURTH DRUG

Ask: Is the patient actually taking the tablets (count them; ask the family)? Is the BP reading right (cuff size, home BP)? Is the patient eating too much salt, taking NSAIDs or drinking heavily? Is the diuretic dose adequate for the kidney function? Correcting these is more effective than any new drug.

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PART V · CHAPTER 18

Master Table of Antihypertensive Drugs

Class, drug, initial dose, maximum dose, frequency, adverse effects and cautions — oral adult doses

Doses are usual adult oral doses for hypertension. Start at the lower dose in the elderly, in CKD, with volume depletion or when combined with other drugs. Maximum = usual maximum for hypertension (higher doses in some labels or other indications). Check local product information.

Drug	Initial dose	Maximum dose	Frequency	Adverse effects / cautions
THIAZIDE-LIKE AND THIAZIDE-TYPE DIURETICS — first line				
Chlorthalidone	12.5 mg	25 mg	OD	Hypokalaemia (more than HCTZ), hyponatraemia, hyperuricaemia/gout, hyperglycaemia; long-acting; effective even at eGFR 15–30
Indapamide	1.5 mg SR (or 1.25 mg IR)	1.5 mg SR (2.5 mg IR)	OD	Fewer metabolic effects; hyponatraemia, hypokalaemia; QT prolongation (rare)
Hydrochlorothiazide	12.5 mg	25 mg (50 mg)	OD	As above; photosensitivity; possible skin cancer risk with long-term use; weak when eGFR <30
Metolazone	2.5 mg	10 mg	OD	Reserved for resistant oedema/CKD with loop diuretic; profound hypokalaemia and volume depletion
ACE INHIBITORS — first line				
Enalapril	5 mg (2.5 mg elderly/diuretic)	40 mg/day	OD–BD	Dry cough, angioedema, hyperkalaemia, rise in creatinine (accept ≤30%), first-dose hypotension. **Contraindicated in pregnancy**, angioedema, bilateral renal artery stenosis; never combine with ARB/aliskiren; 36 h gap with sacubitril. Check K^+/creatinine at 1–2 weeks. Reduce dose in CKD (except fosinopril)
Ramipril	2.5 mg	10 mg (20 mg)	OD–BD	
Lisinopril	5–10 mg	40 mg	OD	
Perindopril	4 mg erbumine (5 mg arginine)	8 mg erbumine (10 mg arginine)	OD	
Captopril	12.5–25 mg	50 mg TDS (150 mg/day)	BD–TDS	
Trandolapril	1–2 mg	4 mg	OD	
ANGIOTENSIN RECEPTOR BLOCKERS — first line				
Telmisartan	40 mg (20 mg)	80 mg	OD	Longest half-life (~24 h); mainly biliary excretion — no renal dose change; caution in cholestasis/severe liver disease
Losartan	50 mg (25 mg if volume depleted/hepatic)	100 mg	OD–BD	Uricosuric — useful in gout; shorter acting
Olmesartan	20 mg	40 mg	OD	Rare sprue-like enteropathy (chronic diarrhoea)
Valsartan	80 mg	320 mg	OD	Also in heart failure (40–160 mg BD)
Irbesartan	150 mg	300 mg	OD	Diabetic nephropathy evidence (IDNT)
Candesartan	8 mg (4 mg)	32 mg	OD	Heart failure (CHARM); migraine prophylaxis
Azilsartan medoxomil	40 mg	80 mg	OD	Potent; diarrhoea

*All ARBs: hyperkalaemia, rise in creatinine, dizziness; **contraindicated in pregnancy**; do not combine with ACEi or aliskiren.*

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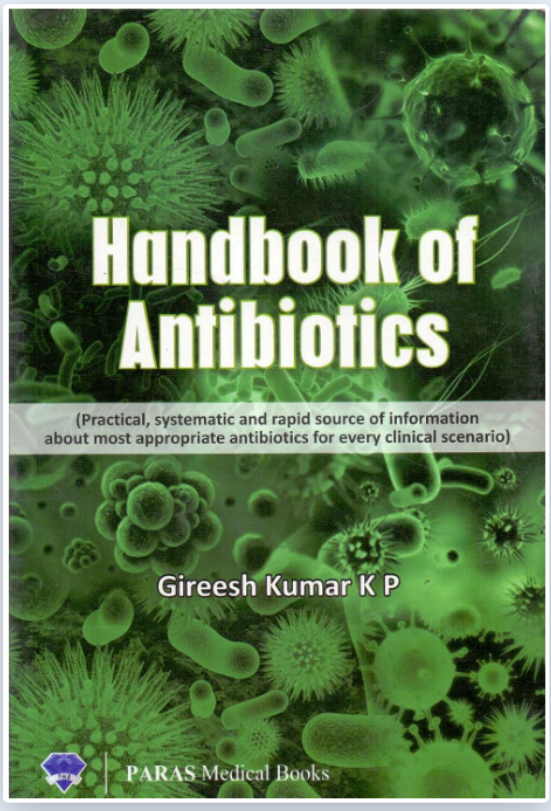
Drug	Initial dose	Maximum dose	Frequency	Adverse effects / cautions
DIHYDROPYRIDINE CALCIUM CHANNEL BLOCKERS — first line				
Amlodipine	2.5–5 mg	10 mg	OD	Ankle oedema (dose-related; not helped by diuretics, reduced by ACEi/ARB), flushing, headache, palpitations, gum hyperplasia. Very long half-life — forgiving of missed doses
S-amlodipine	2.5 mg	5 mg	OD	
Nifedipine (extended release / GITS / retard)	20–30 mg ER OD (retard 10–20 mg BD)	90 mg/day ER (retard 40 mg BD)	OD–BD	Oedema, headache, flushing; safe in pregnancy. **Never use immediate-release capsules sublingually**
Cilnidipine	5–10 mg	20 mg	OD	L/N-type blocker; less oedema and reflex tachycardia
Lercanidipine	10 mg	20 mg	OD	Take 15 min before food; less oedema
Felodipine ER	5 mg (2.5 mg elderly)	10 mg	OD	Grapefruit and CYP3A4 inhibitors raise levels
Benidipine	4 mg	8 mg	OD	Oedema, palpitations
NON-DIHYDROPYRIDINE CALCIUM CHANNEL BLOCKERS				
Diltiazem SR/CD	90 mg BD SR or 120–180 mg CD OD	360 mg/day	OD–BD	Bradycardia, AV block, worsening HFrEF (contraindicated), oedema; **avoid with β-blockers**; CYP3A4 interactions (statins, ticagrelor, DOACs, ciclosporin)
Verapamil SR	120–240 mg	480 mg/day	OD–BD	
Verapamil — constipation is common; raises digoxin levels.				
BETA-BLOCKERS				
Metoprolol succinate ER	25–50 mg	200 mg	OD	Cardioselective; HFrEF (start 12.5–25 mg)
Metoprolol tartrate	25–50 mg BD	100 mg BD	BD	Short-acting
Bisoprolol	2.5–5 mg	10 mg (20 mg)	OD	Highly cardioselective; HFrEF (start 1.25 mg)
Nebivolol	2.5–5 mg	20 mg (up to 40 mg)	OD	Vasodilating (NO-mediated); fewer metabolic and sexual side-effects
Carvedilol	6.25 mg BD (3.125 mg BD in HF)	25 mg BD	BD	α + β blocker; orthostatic hypotension; take with food
Labetalol	100 mg BD	2400 mg/day (in 2–4 doses)	BD–QID	First choice in pregnancy; hepatotoxicity (rare), scalp tingling, orthostatic hypotension
Atenolol	25–50 mg	100 mg	OD	Not preferred for uncomplicated hypertension; renal excretion — reduce dose if eGFR <35
Propranolol	40 mg BD (LA 80 mg OD)	320 mg/day	BD–TDS	Non-selective — avoid in asthma; useful in tremor, migraine, anxiety, thyrotoxicosis, portal hypertension
All β-blockers: bradycardia, AV block, fatigue, cold extremities, bronchospasm, masking of hypoglycaemia, erectile dysfunction; taper — do not stop abruptly.				
MINERALOCORTICOID RECEPTOR ANTAGONISTS AND POTASSIUM-SPARING DIURETICS				
Spironolactone	12.5–25 mg	50 mg (100 mg)	OD	Hyperkalaemia, gynaecomastia, menstrual disturbance; avoid if K^+ >5.0 or eGFR <30; check K^+ at 1 and 4 weeks
Eplerenone	25–50 mg	50 mg BD	OD–BD	Hyperkalaemia; no gynaecomastia; avoid strong CYP3A4 inhibitors
Amiloride	5 mg	10 mg (20 mg)	OD	Hyperkalaemia; useful in Liddle syndrome and resistant hypertension
LOOP DIURETICS				
Furosemide	20–40 mg	80 mg BD (higher in CKD/HF)	BD	Short-acting; hypokalaemia, hyponatraemia, dehydration, gout, ototoxicity

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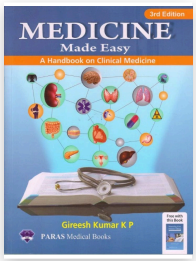
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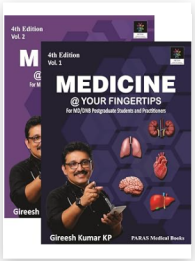
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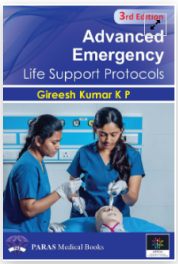
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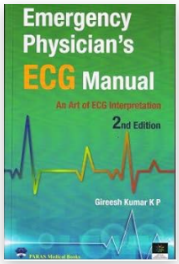
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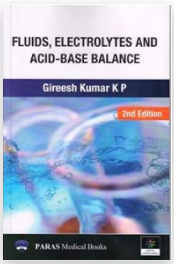
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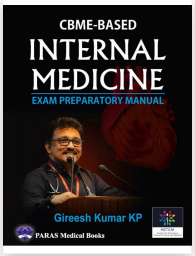
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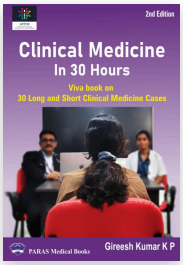
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Drug	Initial dose	Maximum dose	Frequency	Adverse effects / cautions
Torsemide	5–10 mg	20 mg (higher in CKD/HF)	OD	Longer acting, more reliable absorption; preferred for BP when eGFR <30
ALPHA-1 BLOCKERS				
Prazosin (IR / XL)	0.5–1 mg at bedtime (XL 2.5 mg)	20 mg/day in divided doses (XL 10 mg)	BD–TDS (XL OD)	First-dose syncope (give at bedtime), orthostatic hypotension, dizziness, nasal stuffiness, floppy-iris syndrome; useful with BPH; not for monotherapy
Doxazosin	1 mg (XL 4 mg)	16 mg (XL 8 mg)	OD	
Terazosin	1 mg at bedtime	20 mg	OD–BD	
CENTRALLY ACTING AGENTS				
Clonidine	0.1 mg BD	0.3 mg BD (usual); 2.4 mg/day absolute	BD–TDS	Sedation, dry mouth, bradycardia; **rebound hypertension** on sudden withdrawal — taper over 1–2 weeks
Methyldopa	250 mg BD–TDS	3 g/day (in 2–4 doses)	BD–QID	Pregnancy; sedation, depression, hepatitis, haemolytic anaemia
Moxonidine	0.2 mg	0.6 mg/day (0.3 mg BD)	OD–BD	Dry mouth, drowsiness; avoid in heart failure and eGFR <30
DIRECT VASODILATORS				
Hydralazine	10–25 mg TDS–QID	200 mg/day (HF up to 300 mg/day with nitrate)	TDS–QID	Reflex tachycardia, fluid retention, headache; drug-induced lupus (>200 mg/day); give with β-blocker + diuretic
Minoxidil	2.5–5 mg	40 mg/day (100 mg absolute)	OD–BD	Severe fluid retention, pericardial effusion, hirsutism; must combine with β-blocker + loop diuretic
OTHERS				
Aliskiren	150 mg	300 mg	OD	Diarrhoea, hyperkalaemia; contraindicated with ACEi/ARB in diabetes or eGFR <60; pregnancy
Sacubitril–valsartan	24/26–49/51 mg BD	97/103 mg BD	BD	Hypotension, hyperkalaemia, angioedema; 36 h after last ACEi dose; mainly for HFrEF
Aprocitentan (not in India)	12.5 mg	12.5 mg	OD	Resistant hypertension; teratogenic (pregnancy testing), oedema, anaemia, liver enzymes

OD once daily · BD twice daily · TDS three times daily · QID four times daily · ER/SR/XL/CD extended/sustained/controlled release · IR immediate release.

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PART V · CHAPTER 19

Hypertension with Associated Diseases

Compelling indications — choosing drugs for diabetes, heart disease, proteinuria, CKD, stroke, retinopathy and other comorbidities

Quick guide — preferred and avoided drugs

Condition	BP target	Preferred drugs	Avoid / use with caution
Diabetes mellitus	<130/80	ACEi/ARB (essential if albuminuria) + CCB or thiazide-like; add SGLT2i/GLP-1 RA for organ protection	High-dose thiazide + β-blocker (glucose); ACEi + ARB
Stable angina / CAD	<130/80 (DBP ≥70)	β-blocker and/or CCB; ACEi/ARB	Short-acting nifedipine; hydralazine/minoxidil alone (reflex tachycardia)
After myocardial infarction	<130/80	β-blocker + ACEi (or ARB); MRA if EF ≤40% with HF/diabetes	Non-DHP CCB if LV dysfunction
HFrEF (EF ≤40%)	<130/80	ARNI (or ACEi/ARB) + β-blocker (bisoprolol, carvedilol, metoprolol succinate, nebivolol) + MRA + SGLT2i; loop diuretic for congestion; amlodipine if BP still high	**Verapamil, diltiazem**, moxonidine, α-blockers, NSAIDs, pioglitazone
HFpEF	<130/80	Diuretic for congestion, SGLT2i; ARB/ARNI and MRA (spironolactone); any first-line drug to reach target	Excess diuresis
Atrial fibrillation	<130/80	β-blocker or non-DHP CCB (rate control); ACEi/ARB; anticoagulation per CHA_2DS_2-VASc	Non-DHP CCB if HFrEF
LVH	<130/80	ACEi/ARB + CCB or diuretic	Hydralazine, minoxidil (do not regress LVH)
CKD without albuminuria	<130/80	Any first-line; ACEi/ARB + CCB; loop diuretic if eGFR <30 (or chlorthalidone)	NSAIDs; MRAs if eGFR <30 or K^+ high
CKD with albuminuria / proteinuria	<130/80 (SBP <120 if tolerated)	**ACEi or ARB at maximum tolerated dose** + SGLT2i; finerenone (type 2 diabetes); non-DHP CCB adds antiproteinuric effect	ACEi + ARB; DHP-CCB alone without RAAS blocker
Stroke / TIA (after acute phase)	<130/80	ACEi/ARB + thiazide-like (perindopril + indapamide) or + CCB	Hypotension in carotid stenosis
Hypertensive retinopathy	<130/80 (gradual)	Any first-line combination; ACEi/ARB in diabetes	Rapid falls in malignant hypertension
Peripheral arterial disease	<130/80	ACEi/ARB, CCB; β-blockers acceptable	—
Aortic aneurysm / dissection	<130/80 (lower in acute dissection)	β-blocker first; ARB (Marfan)	Vasodilators without β-blocker
Asthma	<130/80	ACEi/ARB, CCB, diuretic	**Non-selective β-blockers**; avoid most β-blockers
COPD	<130/80	ACEi/ARB, CCB, diuretic; cardioselective β-blocker if indicated	Non-selective β-blockers
Gout / hyperuricaemia	<130/80	**Losartan**, CCB	Thiazide and loop diuretics
Benign prostatic hyperplasia	<130/80	Add α-blocker (doxazosin, terazosin, prazosin) to standard therapy	α-blocker alone for BP
Osteoporosis	<130/80	Thiazide (reduces calcium loss)	Loop diuretics; drugs causing falls
Migraine	<130/80	Propranolol, metoprolol, candesartan	—

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Condition	BP target	Preferred drugs	Avoid / use with caution
Essential tremor / anxiety	<130/80	Propranolol	—
Hyperthyroidism	—	Propranolol (symptom control)	—
Liver cirrhosis	Often low BP	Non-selective β-blocker (carvedilol, propranolol) for portal hypertension; CCB	ACEi/ARB with ascites; diuretics carefully
Obesity / metabolic syndrome	<130/80	ACEi/ARB, CCB; weight loss; GLP-1 RA	High-dose thiazide + β-blocker
Obstructive sleep apnoea	<130/80	CPAP, weight loss; spironolactone in resistant cases	Sedating central agents
Erectile dysfunction	<130/80	ARB, ACEi, CCB, nebivolol	Thiazides, older β-blockers; PDE-5 inhibitors with nitrates; caution with α-blockers
Young women / pregnancy planned	<130/80 (pregnancy <140/90)	Nifedipine ER, labetalol, methyldopa	**ACEi, ARB, aliskiren, MRA**; atenolol
Elderly / frail	Individualised	CCB, thiazide-like, ACEi/ARB at low dose	α-blockers, central agents (falls, orthostasis)

Hypertension with diabetes mellitus

- More than half of people with type 2 diabetes have hypertension; together they multiply the risk of CAD, stroke, CKD and retinopathy. BP control is as important as glucose control (UKPDS).
- **Threshold:** start drugs at ≥130/80 (ACC/AHA, ADA) or ≥140/90 (ESH, start at 130–139 if HMOD). **Target:** <130/80 if tolerated; not below 120/70.
- **Drug choice:** ACEi or ARB is first choice, especially with albuminuria; combine with amlodipine or a thiazide-like diuretic. β-blockers when indicated (CAD, HF) — prefer nebivolol, carvedilol, bisoprolol.
- **Organ-protective glucose drugs:** SGLT2 inhibitors (empagliflozin, dapagliflozin) lower SBP by 3–5 mmHg and protect kidney and heart; GLP-1 RAs (semaglutide) reduce weight, BP and CV events; **finerenone** in type 2 diabetes with albuminuria on maximal ACEi/ARB.
- Check **orthostatic BP** (autonomic neuropathy) and consider ABPM for nocturnal hypertension.
- Monitor UACR and eGFR yearly; fundoscopy yearly; statin for most diabetics with hypertension.

Hypertension with cardiac disease

Coronary artery disease and angina

- β-blockers and CCBs relieve angina and lower BP; ACEi/ARB reduce events in high-risk patients (HOPE, EUROPA).
- If angina persists on a β-blocker, add a **DHP-CCB** (amlodipine) — not verapamil/diltiazem. Avoid DBP below 60–70 mmHg.
- Add statin, antiplatelet; nitrates are for symptom relief.

After acute coronary syndrome

- β-blocker (especially if EF reduced), ACEi (start within 24 h if no hypotension) or ARB, MRA (eplerenone/spironolactone) when EF ≤40% with HF or diabetes; target <130/80.

Heart failure with reduced EF

- The four pillars — **ARNI/ACEi/ARB, evidence-based β-blocker, MRA, SGLT2i** — lower both BP and mortality. Titrate each to target dose.
- If BP remains high: add amlodipine or felodipine (neutral in HF), hydralazine + isosorbide dinitrate, or a thiazide with loop diuretic.
- Avoid verapamil, diltiazem, moxonidine, α-blockers and NSAIDs.

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Heart failure with preserved EF

- Control BP and volume: diuretics for congestion, SGLT2i (EMPEROR-Preserved, DELIVER); spironolactone and ARNI may reduce hospitalisation; treat AF and ischaemia.

Atrial fibrillation

- Rate control with β-blocker or diltiazem/verapamil (if EF normal); ACEi/ARB may reduce recurrence; anticoagulate (hypertension scores 1 point in CHA_2DS_2-VASc). Uncontrolled BP increases bleeding risk on anticoagulants — aim <130/80.

Hypertension with proteinuria and chronic kidney disease

STEP-BY-STEP APPROACH

1. **Measure** eGFR and UACR (or urine protein–creatinine ratio). Use standardised office BP or home BP.
2. **Target** <130/80 (KDIGO 2021: SBP <120 if tolerated, using standardised measurement). Restrict salt to <5 g/day (sodium <2 g).
3. **Start ACEi or ARB** in all with albuminuria ≥30 mg/g (A2–A3) — and titrate to the **maximum tolerated dose**. Aim to reduce proteinuria by ≥30% (ideally <0.5–1 g/day).
4. **Check creatinine and K^+ at 1–2 weeks.** A rise in creatinine up to 30% is expected and acceptable (haemodynamic); if >30%, look for volume depletion, NSAIDs or renal artery stenosis and reduce the dose.
5. **Add an SGLT2 inhibitor** (dapagliflozin 10 mg or empagliflozin 10 mg) if eGFR ≥20 — with or without diabetes.
6. **Add a second BP drug:** DHP-CCB or diuretic (thiazide-like if eGFR ≥30; chlorthalidone works down to eGFR 15; loop diuretic for oedema/eGFR <30).
7. **Type 2 diabetes with albuminuria:** add finerenone 10–20 mg (if eGFR ≥25, K^+ ≤5.0).
8. **Manage hyperkalaemia** rather than stopping the RAAS blocker: low-potassium diet, diuretic, correct acidosis (sodium bicarbonate), potassium binders (sodium zirconium cyclosilicate, patiromer) where available. Reduce/stop ACEi/ARB if K^+ >5.5–6.0 mmol/l.

- **Never combine ACEi + ARB** (ONTARGET, VA NEPHRON-D — more hyperkalaemia and AKI).
- Non-DHP CCBs (diltiazem, verapamil) have additional antiproteinuric effect; DHP-CCB alone may increase proteinuria — always use with ACEi/ARB.
- **"Sick-day rules":** temporarily stop ACEi/ARB, diuretics, MRA, SGLT2i and metformin during vomiting, diarrhoea or poor intake; restart when eating and drinking normally.
- **Dialysis patients:** volume control (dry weight, salt and fluid restriction) is the main treatment; prefer drugs not removed by dialysis (amlodipine, ARBs, carvedilol); give doses after dialysis where relevant.
- **Renal transplant:** CCBs (counteract calcineurin vasoconstriction) and ARBs/ACEi (proteinuria) are preferred; target <130/80.
- **Renovascular disease:** ACEi/ARB can be used with careful monitoring in unilateral stenosis; avoid in bilateral stenosis or single kidney with stenosis.

Hypertension with retinal damage

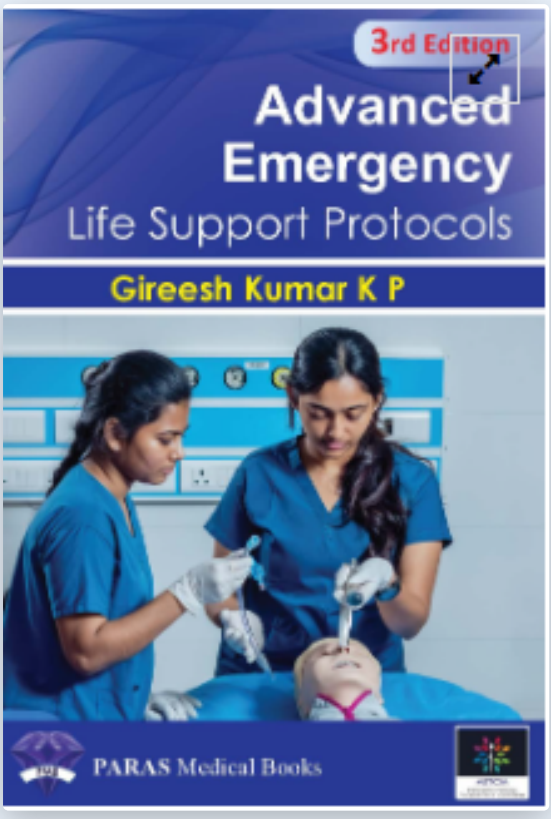
- **Grade I–II (mild):** standard treatment to target; yearly fundus review.
- **Grade III (moderate):** indicates severe/accelerated hypertension — intensify treatment, look for other HMOD and secondary causes; reassess fundus in 4–6 weeks.
- **Grade IV / malignant (papilloedema):** hypertensive emergency — admit; lower MAP by 20–25% over several hours (IV labetalol or nicardipine), then gradually to target over 24–48 h and weeks (Chapter 20).
- With **diabetic retinopathy**: tight BP control (<130/80) slows progression; ACEi/ARB preferred; refer to ophthalmology for maculopathy.
- **Retinal vein occlusion:** screen for and control hypertension, diabetes and lipids; ophthalmology for anti-VEGF/laser.

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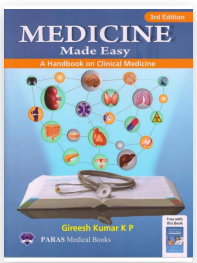
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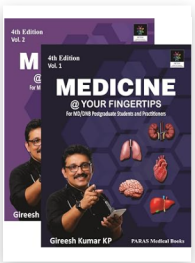
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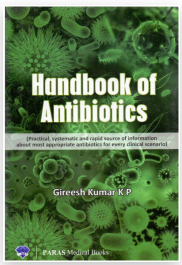
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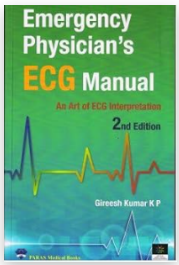
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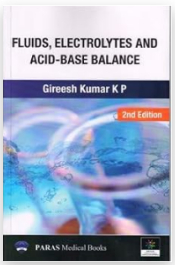
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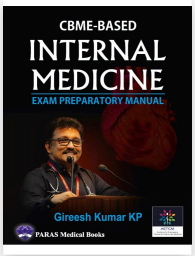
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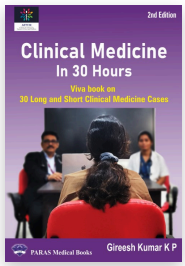
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Hypertension with stroke and cognitive impairment

- After the acute phase (usually after 72 h, once stable), start or restart drugs; target <130/80. ACEi + indapamide (PROGRESS) or CCB-based regimens are effective.
- In severe bilateral carotid stenosis or haemodynamic TIAs, lower BP more slowly.
- Combine with statin and antiplatelet (or anticoagulant for AF).

Hypertension with dyslipidaemia

- Most hypertensives at moderate or higher CV risk benefit from a statin (target LDL by risk category — e.g., <70 mg/dl in high risk; <55 mg/dl (ESC) or <50 mg/dl (Lipid Association of India) in very high risk).
- Prefer metabolically neutral drugs (ACEi/ARB, CCB); low-dose thiazides are acceptable.

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PART VI · CHAPTER 20

Hypertensive Emergencies and Severe Hypertension

Telling an emergency from severe asymptomatic hypertension, condition-specific targets and IV drugs

Definitions

Term	Definition	Setting
Hypertensive emergency	Severe BP elevation (usually **≥180/110–120 mmHg**) with **new or worsening acute target-organ damage** — brain, heart, aorta, kidney, retina, or pregnancy	ICU/HDU; IV drugs; controlled reduction
Malignant hypertension	Severe hypertension with grade III–IV retinopathy (flame haemorrhages, cotton-wool spots, papilloedema), often with AKI and thrombotic microangiopathy	Emergency
Severe hypertension without acute organ damage (formerly "hypertensive urgency")	BP ≥180/110–120 without acute HMOD — often non-adherence, pain, anxiety	Outpatient/ED; **oral** drugs; review in days

Recognising acute target-organ damage

- **Brain:** encephalopathy (headache, vomiting, confusion, seizures), stroke, ICH, SAH
- **Heart:** acute coronary syndrome, acute pulmonary oedema
- **Aorta:** acute dissection
- **Kidney:** AKI, haematuria, proteinuria, thrombotic microangiopathy
- **Eye:** retinal haemorrhages, exudates, papilloedema
- **Pregnancy:** severe pre-eclampsia, eclampsia, HELLP
- **Catecholamine crisis:** phaeochromocytoma, cocaine, amphetamines, MAOI interactions

Initial tests: fundoscopy, ECG, troponin, creatinine, electrolytes, urine for protein and blood, CBC with smear and platelets, LDH, haptoglobin (TMA), chest X-ray; CT/MRI brain, CT aortogram or echocardiography as indicated; pregnancy test in women of child-bearing age.

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General rules for lowering BP in an emergency

THE "25% RULE"

- Most emergencies: reduce **MAP by no more than 20–25% in the first hour**; then, if stable, to about **160/100–110 over the next 2–6 hours**; then cautiously to normal over the next 24–48 hours.
- **Exceptions needing faster lowering:** acute aortic dissection (SBP <120 within 20 min), severe pre-eclampsia/eclampsia (<160/105 promptly), acute pulmonary oedema and ACS (SBP <140 promptly), phaeochromocytoma crisis.
- **Exception needing slower lowering:** acute ischaemic stroke (see Chapter 11).
- Use titratable **IV infusions** with close (ideally intra-arterial) monitoring. Avoid sublingual nifedipine, sublingual captopril and IM injections — unpredictable falls cause stroke and MI.
- Many patients are volume depleted (pressure natriuresis) — avoid diuretics unless fluid overloaded.

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Condition-specific targets and drugs

Condition	Timeline and target	First-line	Alternative / note
Malignant hypertension (± TMA/AKI)	Several hours; MAP −20–25%	Labetalol, nicardipine	Nitroprusside, urapidil; RAAS blockers useful once stable
Hypertensive encephalopathy	Immediately; MAP −20–25%	Labetalol, nicardipine	Nitroprusside
Acute ischaemic stroke, BP >220/120	1 h; MAP −15%	Labetalol, nicardipine	Do not lower if <220/120 and no thrombolysis
Ischaemic stroke for thrombolysis, BP >185/110	1 h; <185/110 before, <180/105 for 24 h	Labetalol, nicardipine	—
Intracerebral haemorrhage, SBP >150–220	Within 1 h; SBP ~140 (130–150)	Labetalol, nicardipine	Avoid SBP <130 and abrupt large falls
Acute coronary syndrome	Immediately; SBP <140	IV nitroglycerin, labetalol/esmolol	Avoid hydralazine (reflex tachycardia)
Acute cardiogenic pulmonary oedema	Immediately; SBP <140	IV nitroglycerin (or nitroprusside) + IV furosemide	Urapidil; NIV. Avoid β-blockers in acute decompensation
Acute aortic dissection	Immediately; **SBP <120 and HR <60** within 20 min	IV esmolol (or labetalol) first, then nitroprusside/nitroglycerin/nicardipine	Never give vasodilator before β-blockade; urgent CT and surgery
Severe pre-eclampsia / eclampsia / HELLP	Immediately; SBP <160, DBP <105 (aim ~140–150/90–100)	IV labetalol or oral nifedipine (IR or ER) + **magnesium sulphate**	IV hydralazine; plan delivery. Avoid nitroprusside (fetal cyanide)
Phaeochromocytoma crisis	Immediately; MAP −20–25%	Phentolamine	Nitroprusside, urapidil; β-blocker only after α-blockade
Cocaine / amphetamine toxicity	Immediately	Benzodiazepines, then phentolamine or nitroglycerin/nicardipine	Avoid pure β-blockers
Post-operative / bleeding risk	Immediately	Labetalol, esmolol, nicardipine	Treat pain, urinary retention, anxiety

Intravenous drugs for hypertensive emergencies

Drug	Dose	Onset / duration	Cautions
Labetalol	20 mg IV over 2 min, then 20–80 mg every 10 min (max 300 mg); or infusion 0.5–2 mg/min	5–10 min / 3–6 h	Asthma, bradycardia, heart block, acute decompensated HF
Nicardipine	Infusion 5 mg/h; increase by 2.5 mg/h every 5–15 min; max 15 mg/h	5–15 min / 30–40 min (hours after long infusion)	Reflex tachycardia, headache; caution in severe aortic stenosis and acute HF
Esmolol	Load 0.5–1 mg/kg over 1 min, then 50–300 µg/kg/min	1–2 min / 10–30 min	Bradycardia, heart block, HF; ideal in dissection and perioperative
Glyceryl trinitrate (nitroglycerin)	5 µg/min; increase by 5 µg/min every 3–5 min; max 200 µg/min	1–5 min / 3–5 min	Headache, tolerance; contraindicated after PDE-5 inhibitors (24–48 h) and in RV infarction
Sodium nitroprusside	0.3–0.5 µg/kg/min; titrate every 5 min; usual ≤2 µg/kg/min; max 10 µg/kg/min for ≤10 min	Immediate / 1–2 min	Cyanide and thiocyanate toxicity (prolonged use, renal/hepatic failure); protect from light; intra-arterial monitoring; avoid in pregnancy and raised ICP
Hydralazine	5–10 mg IV over 2 min; repeat every 20–30 min (pregnancy); max 20 mg	10–20 min / 2–6 h	Unpredictable, prolonged fall; reflex tachycardia; avoid in ACS and dissection

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Drug	Dose	Onset / duration	Cautions
Urapidil (where available)	12.5–25 mg IV bolus, then infusion 5–40 mg/h	3–5 min / 4–6 h	Hypotension; useful in pulmonary oedema and perioperative
Phentolamine	1–5 mg IV bolus (up to 15 mg), repeat; or infusion	1–2 min / 10–30 min	Tachycardia, arrhythmia; for catecholamine excess
Clevidipine (not widely available in India)	1–2 mg/h, double every 90 s; usual max 21–32 mg/h	2–4 min / 5–15 min	Soy/egg allergy; lipid load
Magnesium sulphate (eclampsia)	Pritchard: 4 g IV over 15–20 min + 10 g IM (5 g each buttock), then 5 g IM 4-hourly; or Zuspan: 4 g IV then 1 g/h infusion — for 24 h	—	Seizure prophylaxis, not an antihypertensive. Monitor knee jerks, respiration (≥12/min), urine (≥30 ml/h); antidote calcium gluconate 1 g IV

Severe hypertension without acute organ damage

1. **Confirm** with correct technique after 30 minutes' rest in a quiet room; treat pain, anxiety, urinary retention.
2. **Exclude acute organ damage** — history, neurological exam, fundus, ECG, urine, creatinine.
3. **Start or intensify oral therapy** — aim for gradual reduction over 24–48 h to <160/100, then to target over weeks. Options: restart missed drugs; amlodipine 5–10 mg; telmisartan/amlodipine SPC; labetalol 100–200 mg; captopril 12.5–25 mg (onset 15–30 min); clonidine 0.1–0.2 mg.
4. **Do not** use IV drugs or sublingual nifedipine; do not aim to normalise BP in the ED.
5. **Follow-up** within 24–72 hours (high risk) or within 1 week; address adherence and secondary causes.

After stabilisation

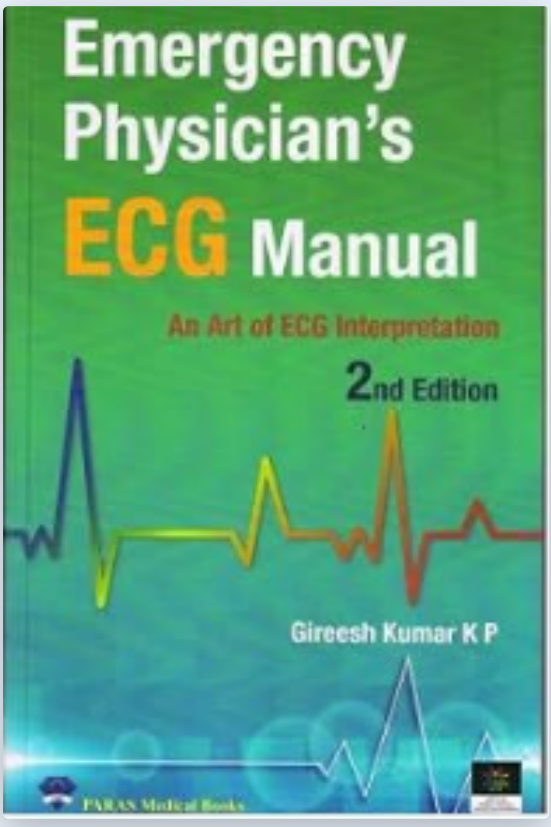
- Switch to oral therapy over 12–24 hours, overlapping with tapering of infusions.
- Investigate for secondary hypertension — it is present in up to one-third of emergencies (renal parenchymal disease, renovascular disease, aldosteronism, phaeochromocytoma, drugs).
- Close follow-up — survivors have high long-term cardiovascular and renal risk.

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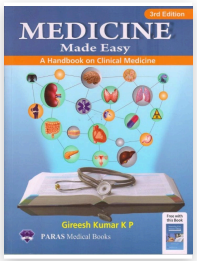
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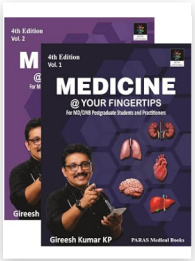
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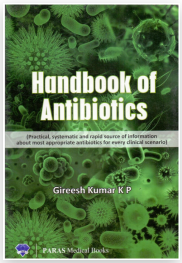
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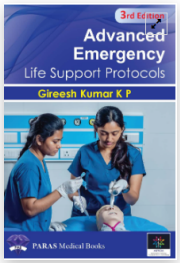
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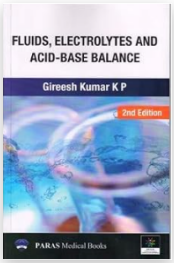
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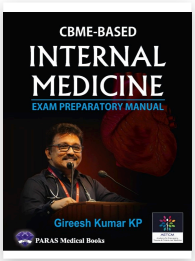
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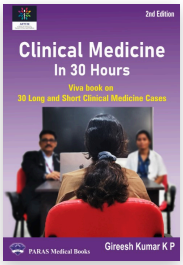
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PART VI · CHAPTER 21

Resistant and Refractory Hypertension

Confirming true resistance, excluding pseudo-resistance and secondary causes, and the fourth and fifth drug

Definitions

Term	Definition
Resistant hypertension	BP above target despite **≥3 drugs** of different classes — usually ACEi/ARB + CCB + diuretic — at maximum or maximally tolerated doses, **with adherence confirmed and uncontrolled BP confirmed by home or ambulatory BP**. ACC/AHA also includes patients controlled only on ≥4 drugs ("controlled resistant").
Apparent resistant hypertension	Uncontrolled on ≥3 drugs but adherence, technique or out-of-office BP not yet confirmed.
Refractory hypertension	Uncontrolled on **≥5 drugs** including chlorthalidone (or other long-acting diuretic) and an MRA.

True resistant hypertension affects about 10% of treated hypertensives and carries high risk of HMOD, CKD and CV events.

Exclude pseudo-resistance first

Cause	How to detect and correct
Poor adherence (most common — up to 50%)	Non-judgemental questioning, pill counts, pharmacy refill records, directly observed therapy; drug-level testing where available. Simplify to once-daily SPC
White-coat effect	Home BP or ABPM
Incorrect measurement	Cuff size (obesity), technique, rest period
Sub-optimal regimen	Inadequate doses; irrational combinations; no diuretic; hydrochlorothiazide instead of chlorthalidone; thiazide when eGFR <30; once-daily furosemide
Interfering substances	NSAIDs, steroids, decongestants, OCPs, alcohol, liquorice, stimulants, erythropoietin, calcineurin inhibitors, VEGF inhibitors
Lifestyle factors	High salt intake (24-h urine sodium >100 mmol suggests excess), obesity, inactivity, heavy alcohol
Pseudohypertension	Calcified, non-compressible arteries in the elderly — suspect if no HMOD despite very high readings and symptoms of hypotension on treatment

Evaluation of true resistant hypertension

- **Screen all for primary aldosteronism** (aldosterone–renin ratio) — found in about 20%.
- Screen for **obstructive sleep apnoea** (very common), CKD (eGFR, UACR), renovascular disease, and — when suggested — phaeochromocytoma, Cushing's syndrome, coarctation, thyroid disease.
- Assess HMOD: ECG/echo, UACR, fundus.
- Refer to a hypertension specialist/centre.

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Treatment

1. **Reinforce lifestyle** — salt <5 g/day (largest effect in resistant hypertension), weight loss, exercise, alcohol limit; treat OSA (CPAP).
2. **Optimise the triple regimen** — full doses of ACEi/ARB + DHP-CCB + long-acting thiazide-like diuretic (chlorthalidone 12.5–25 mg or indapamide); switch to loop diuretic (torsemide) if eGFR <30 or fluid overload. Use SPCs.
3. **Add spironolactone 12.5–25 mg, increase to 50 mg** if K^+ ≤4.5 mmol/l and eGFR ≥45 (caution 30–45). Check K^+ and creatinine at 1 and 4 weeks. Most effective fourth drug (PATHWAY-2).
4. **If spironolactone is not tolerated or contraindicated:** amiloride 10–20 mg, eplerenone 50–100 mg/day, a β-blocker (bisoprolol, nebivolol), an α-blocker (doxazosin), or a central agent (clonidine/moxonidine).
5. **Further options:** hydralazine or minoxidil (with β-blocker and loop diuretic); in heart failure or CKD consider ARNI instead of ACEi/ARB. Where available: aprocitentan; clinical trials of aldosterone synthase inhibitors.
6. **Device therapy: renal denervation** (radiofrequency or ultrasound) in expert centres for uncontrolled resistant hypertension with eGFR >40 ml/min, or in patients unable to tolerate drugs, after shared decision-making.

CLINICAL PEARL

In a patient on "five drugs" whose BP never falls, the commonest explanation is that the tablets are not being taken. Ask the patient to bring all medicines to the clinic and observe a supervised dose with BP measured 2–4 hours later — a large fall confirms non-adherence and avoids needless investigation.

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PART VI · CHAPTER 22

Hypertension in Special Populations

Pregnancy and lactation, children and adolescents, the elderly, women, perioperative care and other groups

Hypertension in pregnancy

Classification

Type	Definition
Chronic hypertension	BP ≥140/90 before pregnancy or before 20 weeks (persists >12 weeks postpartum)
Gestational hypertension	New BP ≥140/90 after 20 weeks without proteinuria or organ dysfunction
Pre-eclampsia	Hypertension after 20 weeks with proteinuria (≥300 mg/24 h, PCR ≥0.3 mg/mg, dipstick ≥2+) **or** maternal organ dysfunction (platelets <100,000, creatinine >1.1 mg/dl or doubling, raised liver enzymes, pulmonary oedema, cerebral or visual symptoms) or uteroplacental dysfunction (fetal growth restriction)
Pre-eclampsia with severe features	BP ≥160/110, or any of the organ-dysfunction features above
Superimposed pre-eclampsia	Chronic hypertension with new proteinuria or organ dysfunction
Eclampsia	Generalised seizures in a woman with pre-eclampsia
HELLP syndrome	Haemolysis, Elevated Liver enzymes, Low Platelets

Management principles

- **Treat at ≥140/90** (CHAP trial); target **<140/90** (DBP about 85). Treat **≥160/110 urgently** — within 30–60 minutes (risk of stroke).
- **Stop ACEi, ARBs, aliskiren, MRAs and statins** before conception or as soon as pregnancy is confirmed; avoid atenolol (fetal growth restriction). Diuretics are generally avoided (reduced plasma volume).
- **Aspirin prophylaxis** for women at high or moderate risk of pre-eclampsia (previous pre-eclampsia, chronic hypertension, diabetes, CKD, autoimmune disease, multiple pregnancy, first pregnancy with other risk factors): **75–150 mg at bedtime from 12 weeks (before 16 weeks) until 36 weeks**.
- **Calcium** 1–1.5 g/day in women with low dietary calcium intake (common in India).
- Monitor for pre-eclampsia: BP, urine protein, platelets, creatinine, liver enzymes, fetal growth.
- Delivery: gestational hypertension and pre-eclampsia without severe features at **37 weeks**; severe pre-eclampsia at 34 weeks or earlier if maternal/fetal compromise.

Drugs in pregnancy

Drug	Dose	Comment
Labetalol	100 mg BD–TDS; up to 2400 mg/day in divided doses	First line; avoid in asthma
Nifedipine ER	20–30 mg OD–BD ER (or retard 10–20 mg BD); max 120 mg/day	First line; headache, oedema
Methyldopa	250 mg BD–TDS; max 3 g/day	Long safety record; sedation, depression — switch after delivery
Hydralazine	Oral 10–25 mg TDS–QID; IV 5–10 mg for acute severe BP	Second line; with labetalol or methyldopa

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Drug	Dose	Comment
Acute severe BP (≥160/110)	IV labetalol 20 mg → 40 mg → 80 mg every 10 min (max 300 mg); or oral nifedipine IR 10–20 mg, repeat in 20 min (max 50 mg in 1 h); or IV hydralazine 5–10 mg	Plus magnesium sulphate for severe pre-eclampsia/eclampsia (Chapter 20)

Postpartum and breastfeeding

- BP often peaks 3–6 days after delivery — monitor for at least 72 hours and again at 1–2 weeks. Postpartum pre-eclampsia can present up to 6 weeks.
- **Compatible with breastfeeding:** enalapril, captopril, nifedipine, amlodipine, labetalol, metoprolol. Switch methyldopa to another agent (postnatal depression).
- Women with hypertensive pregnancy have a 2–4-fold higher lifetime risk of hypertension and CVD — yearly BP check for life.

Children and adolescents

Category (AAP 2017)	Age 1–12 years	Age ≥13 years
Normal	<90th percentile	<120/80
Elevated BP	≥90th to <95th percentile (or 120/80 to <95th, whichever lower)	120–129/<80
Stage 1 hypertension	≥95th percentile to <95th + 12 mmHg (or 130/80–139/89)	130/80–139/89
Stage 2 hypertension	≥95th percentile + 12 mmHg (or ≥140/90)	≥140/90

- Measure BP yearly from age 3 (earlier in high-risk children — prematurity, congenital heart or kidney disease). Use percentile tables for age, sex and height.
- Younger children and severe hypertension → look hard for **secondary causes** (renal parenchymal disease, renal artery stenosis, coarctation, endocrine). Obesity-related primary hypertension is now common in adolescents.
- Treatment: lifestyle first; drugs for stage 2, symptomatic, HMOD, CKD or diabetes, or persistent stage 1. ACEi, ARB, long-acting CCB or thiazide (weight-based doses). Counsel adolescent girls about ACEi/ARB in pregnancy. Target <90th percentile or <130/80 in adolescents.

Older adults (≥65 years) and the very elderly (≥80 years)

- Treatment is highly beneficial even after 80 years (HYVET) — age alone is not a reason to withhold treatment.
- Start low, go slow; prefer CCB, thiazide-like diuretic and ACEi/ARB. Monotherapy is acceptable initially in the very elderly and frail.
- Check **standing BP** at every visit; watch for orthostatic hypotension, falls, hyponatraemia (thiazides) and AKI. Avoid α-blockers and central agents where possible.
- Consider de-prescribing in frail patients with low BP, falls or limited life expectancy.
- **Orthostatic hypotension with supine hypertension** (autonomic failure): avoid lying flat (head-up tilt 10–20°), short-acting drug at bedtime (e.g., losartan, captopril, nitrate patch), avoid daytime antihypertensives; compression stockings.

Women

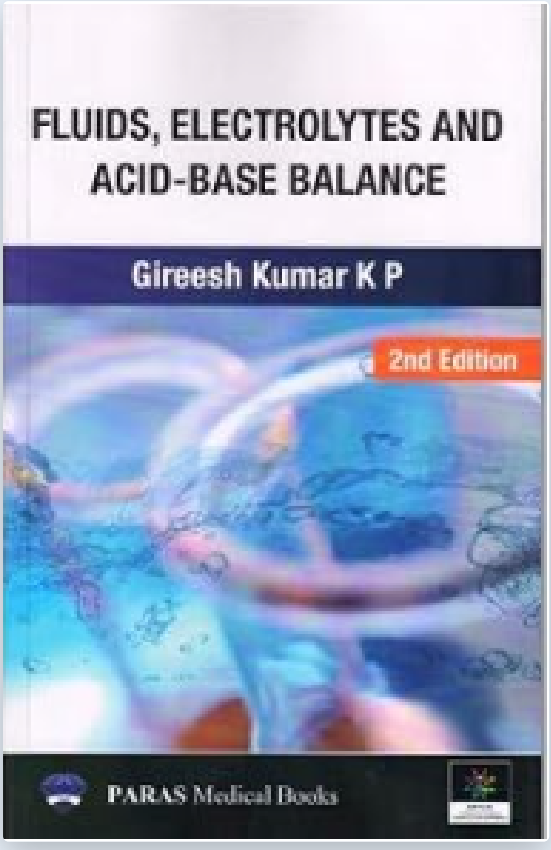
- **Oral contraceptives:** combined pills can raise BP — check BP before and every 6 months; if hypertensive, prefer progestogen-only methods or IUCD. Avoid combined pills in uncontrolled hypertension, age >35 with smoking, or hypertension with vascular disease.
- **Menopausal hormone therapy** is not contraindicated in controlled hypertension; transdermal oestrogen is preferred.

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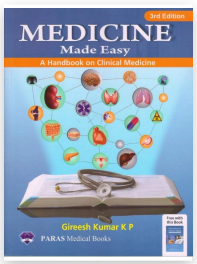
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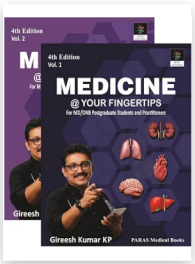
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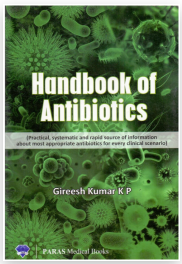
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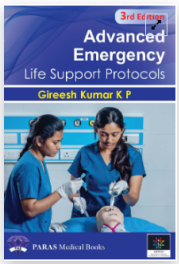
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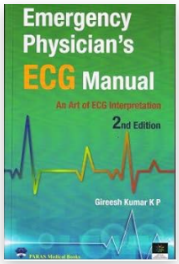
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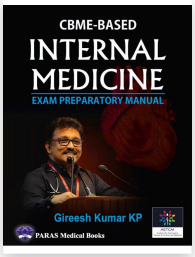
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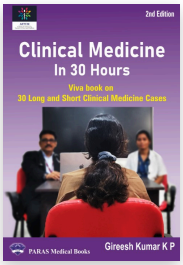
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- Women of child-bearing age on ACEi/ARB must be counselled about stopping on conception and should use effective contraception.

Perioperative hypertension

- Elective surgery need not be postponed for BP <180/110 without HMOD; postpone for BP ≥180/110 to allow control (unless urgent).
- **Continue** β-blockers, CCBs and most other drugs on the day of surgery; do not start a high-dose β-blocker just before surgery. Many anaesthetists withhold ACEi/ARB and diuretics on the morning of surgery (risk of intra-operative hypotension) — restart within 48 h when stable.
- Avoid abrupt withdrawal of clonidine and β-blockers. Post-operative high BP: treat pain, urinary retention, hypoxia, anxiety first.

Other groups

Group	Key points
Young adults (<40 years)	Screen for secondary causes; lifetime risk is high even if 10-year risk is low — treat confirmed hypertension; ACEi/ARB + CCB; counsel women on pregnancy
Athletes	Avoid β-blockers (reduce performance; banned in some sports) and diuretics (banned, dehydration); ACEi/ARB or CCB preferred; screen for stimulant and steroid use
Cancer patients	VEGF inhibitors, TKIs, steroids raise BP; ACEi/ARB or DHP-CCB; avoid non-DHP CCBs with CYP3A4-metabolised drugs
People living with HIV	Higher CV risk; watch interactions of CCBs with protease inhibitors and cobicistat (reduce amlodipine dose)
Chronic alcohol use	Reduction lowers BP within weeks; watch for withdrawal hypertension

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PART VI · CHAPTER 23

Long-Term Follow-Up, Adherence and Team-Based Care

Keeping BP controlled for life — the most neglected part of hypertension care

What to do at each follow-up visit

Area	Checklist
BP	Office BP (correct technique), review home BP diary; standing BP in elderly/diabetics
Adherence	Ask how many doses missed in the last week; check refill dates; ask about side-effects and cost
Side-effects	Ankle oedema, cough, dizziness, erectile dysfunction, fatigue, gout attacks
Lifestyle	Weight, waist, salt, exercise, tobacco, alcohol
Labs	Creatinine and K^+ after RAAS blockers/diuretics/MRA; yearly UACR, glucose/HbA1c, lipids, Na^+
Organ damage	Yearly ECG; fundus every 1–2 years (yearly in diabetes); echo if indicated
Overall risk	Statin, aspirin only for secondary prevention, diabetes control, vaccination (influenza, pneumococcal) in high-risk patients

Why patients stop their medicines

- No symptoms — "I feel fine, so I don't need it"
- Belief that BP drugs damage the kidneys or are "addictive"
- Fear of lifelong medicine; preference for traditional remedies
- Side-effects (oedema, cough, impotence, frequent urination)
- Cost and long distance to the clinic or pharmacy
- Complex regimens — many tablets, multiple times a day
- Forgetfulness, depression, cognitive impairment
- Poor communication and short consultations

Strategies that improve adherence and control

- **Single-pill combinations**, once-daily dosing, the fewest tablets possible; choose low-cost generic drugs (Jan Aushadhi, government free-drug schemes).
- Explain clearly: hypertension is lifelong and silent; medicines protect the brain, heart and kidneys; stopping when BP is normal causes BP to rise again.
- **Home BP monitoring** with feedback — patients who see their own readings are more adherent.
- Link tablets to a daily habit (brushing teeth, morning tea); pill boxes; phone reminders; involve family members.
- Dispense 30–90 days of medicine; synchronise refills; follow up missed visits by phone.
- **Team-based care and task sharing** — nurses, pharmacists, community health officers and ASHAs can measure BP, educate and dispense refills under a standard protocol (IHCI model). Digital tools (e.g., the government NCD portal and apps used in IHCI) track control rates.
- Ask about adherence **without blame**: "Many people find it hard to take tablets every day — how often do you miss yours?"

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Can treatment be reduced or stopped?

- Rarely possible in established hypertension. Consider step-down only in patients with sustained control (≥1 year) on low-dose therapy, low risk and major lifestyle change (e.g., large weight loss after bariatric surgery) — with continued home monitoring.
- Reduce doses when BP is low with symptoms, in frailty, after major weight loss, or with intercurrent illness (sick-day rules).

Quality indicators for a clinic

Indicator	Target
Adults ≥30 years with BP recorded in the past year	>80%
Registered hypertensives with BP <140/90 at last visit	>60–70%
Patients on single-pill combination when on ≥2 drugs	As high as possible
Patients with no visit in the past 3 months ("lost to follow-up")	<20%
Hypertensive diabetics on ACEi/ARB with UACR done yearly	>80%

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APPENDIX A

Quick Reference — Renal, Hepatic, Pregnancy and Lactation

Dose adjustments and safety of common antihypertensives at a glance

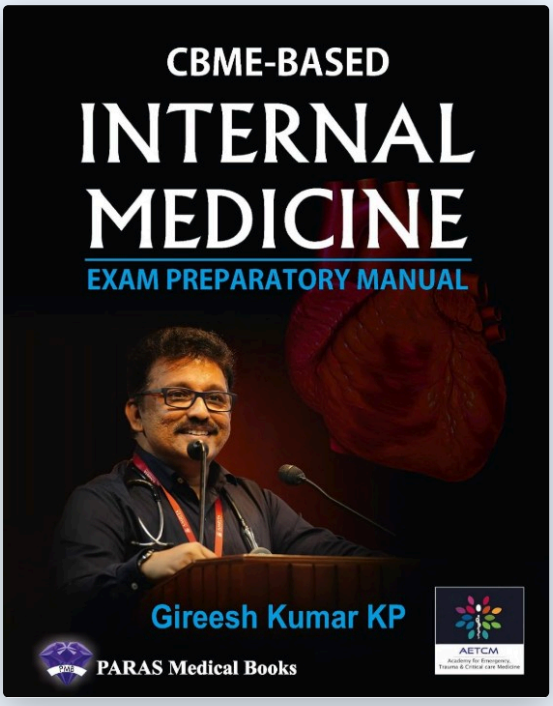
Drug	CKD / low eGFR	Liver disease	Pregnancy	Breastfeeding
Diuretics				
Chlorthalidone	Effective to eGFR ~15 (CLICK); watch volume, K^+	Caution — hypokalaemia may precipitate encephalopathy	Avoid (unless HF)	Low doses acceptable; may suppress lactation
Indapamide / HCTZ	Less effective <30; switch to loop	Caution	Avoid	Low doses acceptable
Furosemide / torsemide	Higher doses needed in CKD	Caution (encephalopathy, hypokalaemia)	Avoid unless pulmonary oedema	Acceptable
Spironolactone	Avoid if eGFR <30 or K^+ >5.0; caution 30–45	Drug of choice for ascites	Avoid (anti-androgenic)	Acceptable
RAAS blockers				
Enalapril	Start 2.5 mg if eGFR <30	Prodrug — caution	**Contraindicated**	**Preferred ACEi**
Ramipril	Max 5 mg if eGFR <30	Caution	**Contraindicated**	Limited data — prefer enalapril
Perindopril	Reduce dose (2 mg) if eGFR 30–60; alternate days if 15–30	Caution	**Contraindicated**	Limited data
Captopril	Reduce dose	—	**Contraindicated**	Compatible
Telmisartan	No change; caution on dialysis	Max 40 mg; avoid in cholestasis/severe disease	**Contraindicated**	Limited data
Losartan	No change	Start 25 mg	**Contraindicated**	Limited data
Olmesartan / valsartan	Caution if eGFR <20–30	Caution / max 80 mg (valsartan)	**Contraindicated**	Limited data
Calcium channel blockers				
Amlodipine	No change; not dialysed	Start 2.5 mg	Acceptable (limited data)	Compatible
Nifedipine ER	No change	Reduce dose	**First line**	Compatible
Cilnidipine / lercanidipine	Caution in severe CKD (lercanidipine avoid <30)	Avoid in severe disease	Avoid (no data)	Avoid (no data)
Diltiazem / verapamil	No change	Reduce dose	Avoid (limited data)	Compatible
β-blockers and others				
Labetalol	No change	Reduce; rare hepatotoxicity	**First line**	Compatible
Metoprolol	No change	Reduce dose	Acceptable	Compatible
Bisoprolol	Max 10 mg if eGFR <20	Max 10 mg in severe disease	Acceptable if needed	Limited data
Atenolol	25–50 mg if eGFR 15–35; 25 mg (or 50 mg alternate days) if <15	No change	Avoid (growth restriction)	Avoid (accumulates in infant)

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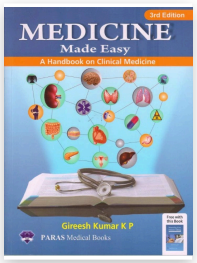
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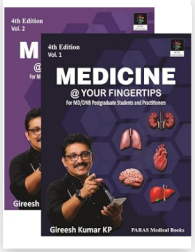
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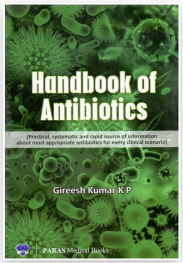
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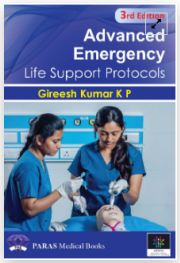
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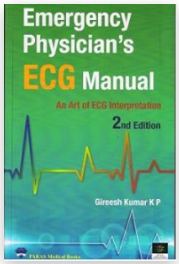
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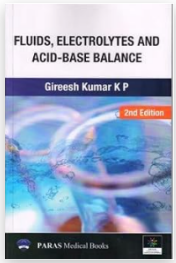
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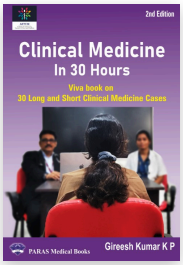
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Drug	CKD / low eGFR	Liver disease	Pregnancy	Breastfeeding
Carvedilol / propranolol	No change	Reduce; useful in portal hypertension	Acceptable if needed	Compatible
Methyldopa	Increase dosing interval	Avoid in active liver disease	**First line**	Compatible, but depression risk
Clonidine	Reduce dose	—	Acceptable	Avoid (infant sedation, hypotension)
Hydralazine	Increase interval	Caution	Second line	Compatible
Prazosin / doxazosin	Start low	Start low	Acceptable (phaeochromocytoma)	Limited data
Moxonidine	Max 0.2 mg (eGFR 30–60); avoid <30	—	Avoid	Avoid

Drug interactions to remember

Combination	Problem
ACEi/ARB/MRA + K^+ supplements, K^+ salt substitutes, trimethoprim, NSAIDs	Hyperkalaemia, AKI ("triple whammy" with diuretic + NSAID)
Thiazide/loop + lithium; ACEi/ARB + lithium	Lithium toxicity
Verapamil/diltiazem + β-blocker, digoxin, ivabradine	Bradycardia, heart block
Verapamil/diltiazem + simvastatin, atorvastatin (high dose), ciclosporin, tacrolimus, DOACs	Raised drug levels (CYP3A4/P-gp)
Nitrates/α-blockers + sildenafil, tadalafil	Severe hypotension
Clonidine + β-blocker (sudden withdrawal)	Rebound hypertensive crisis
Diuretics + corticosteroids, amphotericin, salbutamol	Hypokalaemia
Any antihypertensive + NSAIDs, steroids, decongestants	Reduced BP control

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APPENDIX B

Patient Education Handout and BP Log

Photocopy and give to patients — simple advice in plain language

Living well with high blood pressure

What is high blood pressure?

- It is the force of blood on the walls of your arteries being too high for too long.
- Usually it causes **no symptoms** — you can feel well and still be at risk.
- It can damage your brain (stroke), heart, kidneys and eyes over the years.
- It can be controlled, but usually not cured — treatment is lifelong.

Your medicines

- Take them **every day**, at the same time, even when you feel well.
- Do not stop them when your BP becomes normal — the medicine is what keeps it normal.
- Do not take painkillers like diclofenac or ibuprofen without asking your doctor.
- Tell the doctor about side-effects — there are many alternatives.
- If you are planning pregnancy, tell your doctor first.

Less salt

- No more than **1 level teaspoon (5 g) of salt a day** in total.
- Avoid pickles, papad, dried fish, chips, namkeen, instant noodles and bakery items.
- Do not add salt at the table, or on fruit and curd.
- Use lemon, herbs and spices for flavour.

Healthy food and weight

- Plenty of vegetables, fruits, dal, whole grains.
- Less oil, ghee, fried food, sweets and red meat.
- Keep your waist below 90 cm (men) or 80 cm (women).

Move more

- Brisk walk for **30 minutes, at least 5 days a week**.
- Yoga, cycling, swimming, dancing are all good.
- Sit less — stand up and walk every hour.

Stop tobacco, limit alcohol

- Stop cigarettes, beedi, gutka and pan masala completely.
- Alcohol is best avoided; never drink heavily.
- Sleep 7–8 hours; manage stress with yoga or breathing exercises.

GO TO HOSPITAL IMMEDIATELY IF YOU HAVE

Severe headache with vomiting · chest pain · sudden breathlessness · weakness or numbness of the face, arm or leg · difficulty speaking · sudden loss of vision · fits or confusion · BP above 180/110 with any of these symptoms.

How to check your BP at home

1. Use an upper-arm automatic machine with the right cuff size.
2. No tea, coffee, smoking or exercise for 30 minutes. Empty your bladder.
3. Sit quietly for 5 minutes, back supported, feet flat, arm resting on a table at heart level. Do not talk.
4. Take 2 readings, 1 minute apart, in the **morning** (before medicines and breakfast) and in the **evening** (before dinner).
5. Do this for 7 days before your clinic visit and write down every reading below.

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Home BP log

Day / date	Morning reading 1	Morning reading 2	Evening reading 1	Evening reading 2	Notes (missed dose, symptoms)
Day 1					
Day 2					
Day 3					
Day 4					
Day 5					
Day 6					
Day 7					

For the doctor: discard day 1 and average the remaining readings. Home BP ≥135/85 mmHg = hypertension (ESH); treatment target usually <130/80 at home if tolerated.

My medicines

Medicine and strength	Morning	Night	Purpose / notes

My target BP: _____ Next visit: _____ Clinic phone: _____

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APPENDIX C

Abbreviations and Key Sources

Terms used in this book and the guidelines it is based on

Abbreviations

Abbreviation	Meaning
ABI	Ankle–brachial index
ABPM	Ambulatory blood pressure monitoring
ACC/AHA	American College of Cardiology / American Heart Association
ACEi	Angiotensin-converting enzyme inhibitor
ACS	Acute coronary syndrome
AF	Atrial fibrillation
AKI	Acute kidney injury
AOBP	Automated office blood pressure
ARB	Angiotensin II receptor blocker
ARNI	Angiotensin receptor–neprilysin inhibitor
ARR	Aldosterone–renin ratio
BD	Twice daily
CAD	Coronary artery disease
CCB	Calcium channel blocker
CKD	Chronic kidney disease
CO	Cardiac output
CPAP	Continuous positive airway pressure
CVD	Cardiovascular disease
DASH	Dietary Approaches to Stop Hypertension
DBP	Diastolic blood pressure
DHP	Dihydropyridine
eGFR	Estimated glomerular filtration rate
ER / SR / XL	Extended / sustained / extra-long release
ESC / ESH	European Society of Cardiology / of Hypertension

Abbreviation	Meaning
HBPM	Home blood pressure monitoring
HCTZ	Hydrochlorothiazide
HFpEF / HFrEF	Heart failure with preserved / reduced ejection fraction
HMOD	Hypertension-mediated organ damage
ICH	Intracerebral haemorrhage
IHCI	India Hypertension Control Initiative
ISH	International Society of Hypertension; also isolated systolic hypertension
KDIGO	Kidney Disease: Improving Global Outcomes
LVH	Left ventricular hypertrophy
MAP	Mean arterial pressure
MRA	Mineralocorticoid receptor antagonist
NP-NCD	National Programme for Prevention and Control of NCDs
NSAID	Non-steroidal anti-inflammatory drug
OD	Once daily
OSA	Obstructive sleep apnoea
PRES	Posterior reversible encephalopathy syndrome
PWV	Pulse wave velocity
RAAS	Renin–angiotensin–aldosterone system
SBP	Systolic blood pressure
SGLT2i	Sodium–glucose co-transporter-2 inhibitor
SPC	Single-pill combination
TDS / QID	Three / four times daily
TMA	Thrombotic microangiopathy
TOD	Target-organ damage
TPR	Total peripheral resistance
UACR	Urine albumin–creatinine ratio

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Key guidelines and sources

Source	Topic
Jones DW, et al. 2025 AHA/ACC/multisociety Guideline for the Prevention, Detection, Evaluation and Management of High Blood Pressure in Adults. *Hypertension* 2025	Definitions, thresholds, targets, PREVENT risk, initial SPC therapy
McEvoy JW, et al. 2024 ESC Guidelines for the management of elevated blood pressure and hypertension. *Eur Heart J* 2024	Elevated BP category, SBP target 120–129
Mancia G, et al. 2023 ESH Guidelines for the management of arterial hypertension. *J Hypertens* 2023	Grading, risk stratification, treatment algorithm, emergencies
Unger T, et al. 2020 ISH Global Hypertension Practice Guidelines. *Hypertension* 2020	Essential and optimal standards for low-resource settings
Indian Guidelines on Hypertension-IV (2019); India Hypertension Control Initiative protocols (MoHFW/ICMR/WHO)	Indian practice and programme protocols
WHO Guideline for the pharmacological treatment of hypertension in adults (2021); WHO Global Report on Hypertension (2023); WHO HEARTS technical package	Global thresholds, epidemiology, standard protocols
KDIGO 2021 Clinical Practice Guideline for BP management in CKD; KDIGO 2024 CKD guideline	BP in CKD, RAAS blockade, SGLT2i
ADA Standards of Care in Diabetes (current edition)	Hypertension in diabetes
ACOG Practice Bulletins (2019–2020, updated 2022 after CHAP); ISSHP 2021; FOGSI guidance	Hypertension in pregnancy
Flynn JT, et al. AAP Clinical Practice Guideline (2017)	Paediatric hypertension
Greenberg SM, et al. AHA/ASA 2022 ICH guideline; Powers WJ, et al. AHA/ASA 2019 acute ischaemic stroke guideline	BP in acute stroke
Muntner P, et al. AHA Scientific Statement on BP measurement (2019)	Measurement technique and cuff sizes

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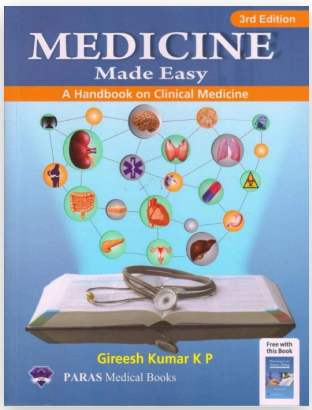
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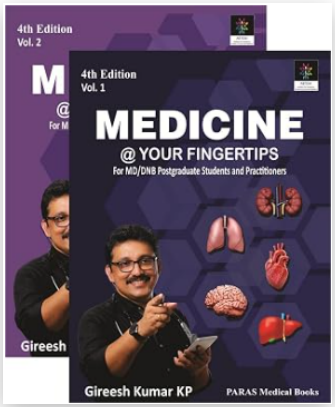
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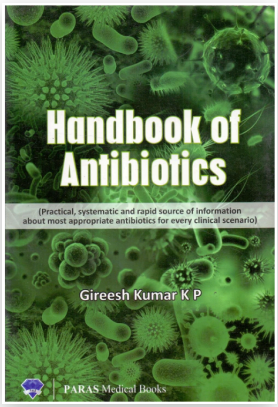
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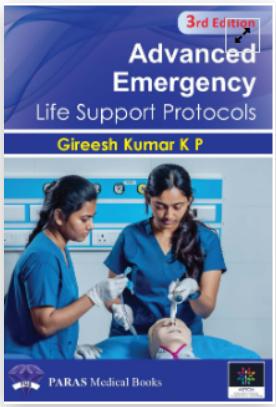
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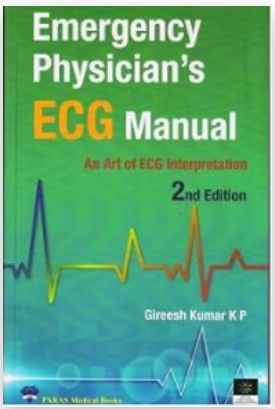
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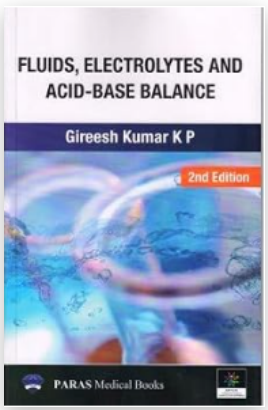
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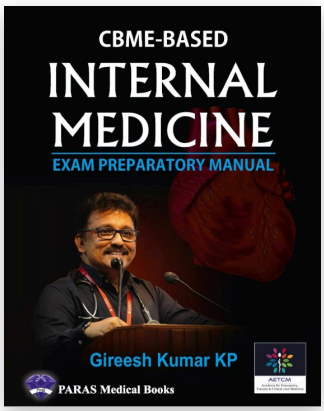
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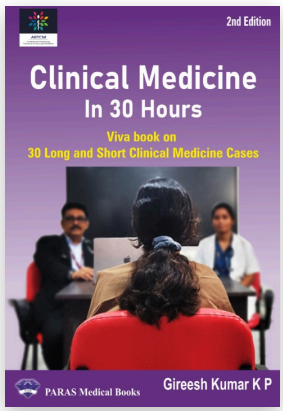
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