FROM FIRST VISIT TO EXPERT MANAGEMENT

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Lifestyle Disease Management

Hypertension · Diabetes · Dyslipidaemia · Obesity

Definition, classification, basic and advanced investigations, lifestyle therapy, drugs with class and dose, step-up and combination therapy, the complete insulin chart, secondary causes and complications — based on current international and Indian guidelines

Hypertension | Diabetes | Insulin chart | Dyslipidaemia | Obesity | Lifestyle therapy | Drug doses | Step-up & combinations | Secondary causes | Complications | Emergencies | International guidelines | 16 cases



By

Dr. Gireesh Kumar K.P.

For MBBS students, interns, residents, general practitioners, physicians, endocrinologists, cardiologists, nurses and diabetes educators

Lifestyle Disease Management

Hypertension, Diabetes, Dyslipidaemia and Obesity — Diagnosis to Expert Therapy

Cardiovascular risk · Lifestyle therapy · Hypertension · Resistant and secondary hypertension · Emergencies

Diabetes · Oral and injectable drugs · Insulin chart and regimens · DKA · Complications

Dyslipidaemia · Statins and beyond · Obesity · Anti-obesity drugs · Metabolic surgery



Dr. Gireesh Kumar K.P.

Academy for Emergency, Trauma & Critical care Medicine (AETCM)

First Edition · October 2026

Disclaimer. This book is a learning and quick-reference guide for medical students and registered practitioners. Drug doses, targets and protocols follow international and Indian guidelines and landmark trials current at the time of writing (October 2026), but guidelines, approvals and drug availability change, and every patient is different. Always check current product information, adjust doses for renal and hepatic function, pregnancy and interactions, follow your institution's protocols, and seek specialist help when in doubt. Brand names are examples only and do not imply endorsement. Figures are schematic illustrations. The author and publisher accept no liability for any consequence arising from the use of this information.

Preface

Every day, in every outpatient clinic in India, a doctor meets a patient with high blood pressure, high sugar, high cholesterol and a waistline that has quietly grown over the years. Often the patient has all four. The consultation is short, the queue is long, and the guidelines — American, European, international and Indian — seem to say slightly different things. This book is written for that doctor.

Hypertension, diabetes, dyslipidaemia and obesity are not four separate diseases. They grow from the same soil, damage the same organs and respond to the same lifestyle changes — and increasingly to the same drugs. The book therefore begins with the foundations common to all four: the epidemic in India, cardiovascular risk assessment and lifestyle therapy prescribed with the precision of a drug.

Each disease is then covered in the same bedside sequence: definition and classification, basic investigations, initial management, lifestyle changes, drugs by class with doses and cautions, step-up and combination therapy, advanced investigations, and the secondary causes and complications that must not be missed. The diabetes section includes a complete insulin chart — every insulin from rapid-acting analogues to the new once-weekly basal insulin — with practical regimens for starting, titrating and switching, and protocols for hypoglycaemia, DKA, the hospital ward and the operating theatre.

Recommendations are drawn from the latest international guidelines — the 2025 AHA/ACC and 2024 ESC hypertension guidelines, the ADA Standards of Care 2026, the 2025 ESC/EAS and 2026 ACC/AHA lipid guidelines, the 2025 definitions of obesity — and from Indian guidance from ICMR, RSSDI, CSI, LAI and the Ministry of Health, with an eye on what is available and affordable in India. Tables and flowcharts are used generously because busy clinicians read them faster than paragraphs. The book closes with sixteen case scenarios and pocket reference cards.

Lifestyle diseases are, above all, diseases of time. Small, persistent improvements — a few kilograms, a few millimetres of mercury, a few points of HbA1c — sustained over years, prevent heart attacks, strokes, dialysis and amputations. I hope this book helps you make those improvements, one patient and one visit at a time.



Dr. Gireesh Kumar K.P.
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October 2026

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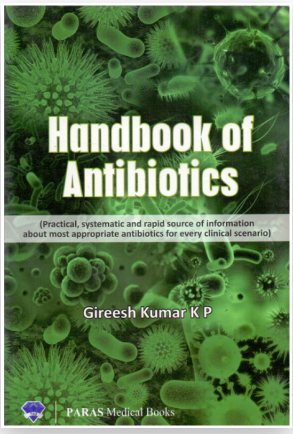
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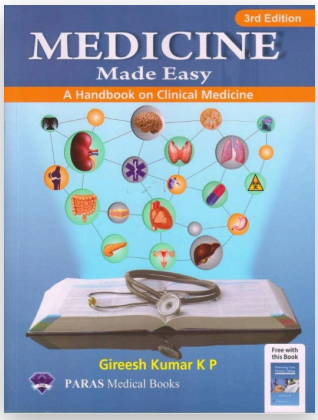
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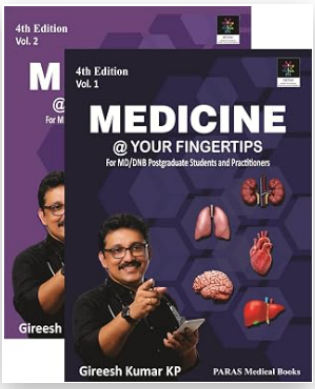
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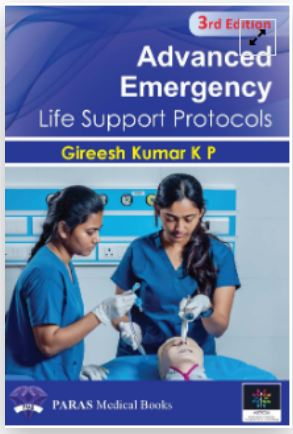
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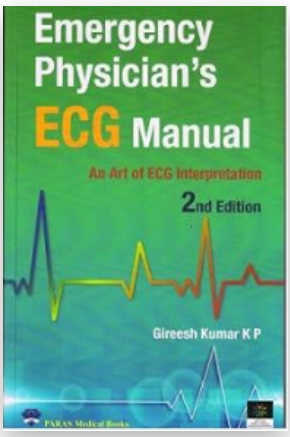
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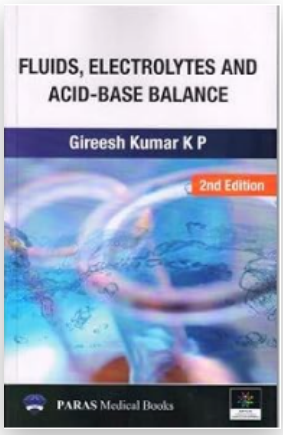
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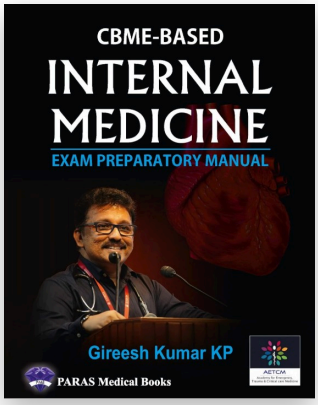
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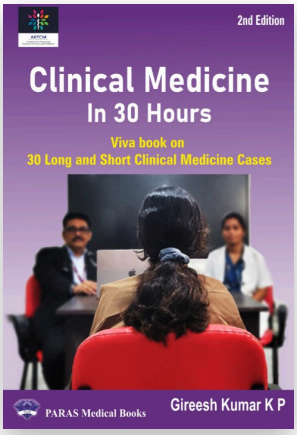
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CHAPTER 1

Lifestyle Diseases — The Cardiometabolic Epidemic

Why hypertension, diabetes, dyslipidaemia and obesity must be managed together

Four diseases, one soil

Hypertension, type 2 diabetes, dyslipidaemia and obesity are called **lifestyle diseases** (non-communicable diseases, NCDs) because they grow from the same soil: energy-dense diet, excess salt and refined carbohydrate, physical inactivity, tobacco, alcohol, poor sleep and chronic stress, acting on a genetically susceptible person. They rarely travel alone. A patient with one of them usually has, or will soon have, another — and together they multiply the risk of heart attack, stroke, heart failure, kidney failure, blindness and amputation.

This book treats them as **one cardiometabolic disease with four faces**. Every chapter follows the same bedside sequence: definition → classification → basic investigations → initial management → lifestyle therapy → drugs (class, drug, dose) → step-up and combination therapy → advanced investigations → secondary causes and complications.

The burden in India

The ICMR–INDIAB national study (2023; 1.1 lakh adults, all states) gives the clearest picture of the Indian epidemic. More than one in ten adults has diabetes, one in three has hypertension and four in five have at least one lipid abnormality. Kerala, Goa, Punjab, Tamil Nadu and urban India carry the highest prevalence, but rural rates are rising fastest.

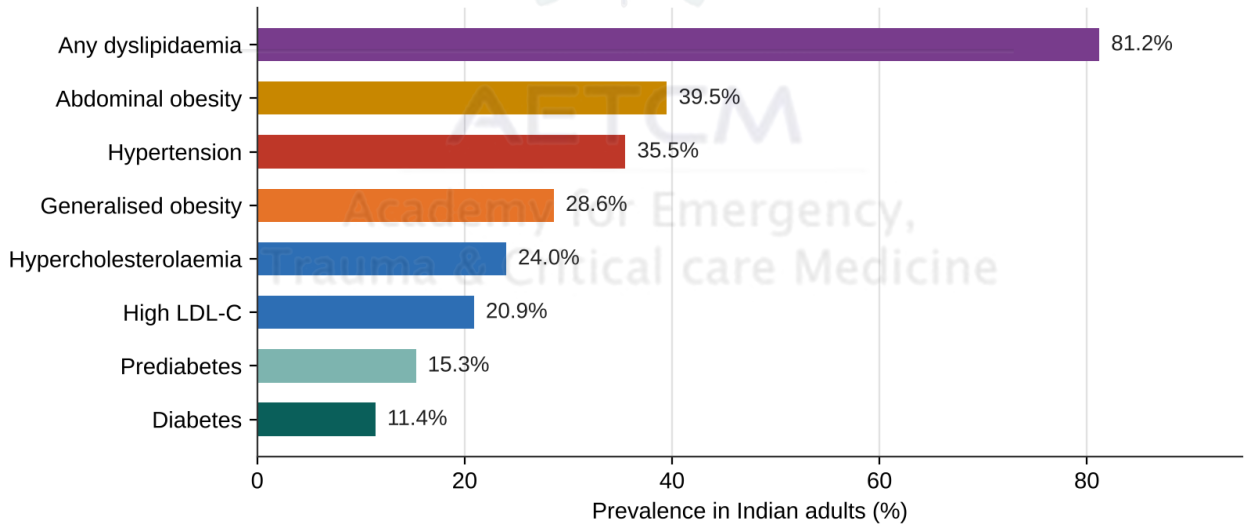


Figure 1.1. Prevalence of cardiometabolic risk factors in Indian adults (ICMR–INDIAB, Lancet Diabetes Endocrinol 2023). Asian Indian cut-offs used for obesity (BMI ≥25 kg/m²; abdominal obesity by waist circumference).

Condition	Estimated adults in India	Key point
Diabetes	≈101 million	Half are undiagnosed; onset a decade earlier than in Europeans
Prediabetes	≈136 million	Highest-yield group for prevention — lifestyle cuts progression by ≈30–58%

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Condition	Estimated adults in India	Key point
Hypertension	≈315 million	Only about one in four is treated and one in ten is controlled
Generalised obesity	≈254 million	Abdominal obesity is commoner than generalised obesity
Hypercholesterolaemia / high LDL-C	≈213 / 185 million	Low HDL-C and high triglycerides dominate the Indian pattern

The Asian Indian phenotype — 'thin-fat' Indians

At any given BMI, Asian Indians have more body fat, more visceral and liver fat, less muscle, more insulin resistance and a higher risk of diabetes and coronary disease than Europeans. That is why **lower cut-offs** are used for Indians throughout this book.

Feature	Asian Indian pattern	Practical consequence
BMI	More fat at the same BMI	Overweight ≥23, obesity ≥25 kg/m² (2025 revised definition: obesity >23)
Waist	Central (abdominal) adiposity common even with normal BMI	Waist ≥90 cm men, ≥80 cm women is abnormal
Diabetes	Earlier onset, lower BMI, early β-cell failure	Screen from age 30–35, or earlier if overweight or risk factors
Lipids	High TG, low HDL-C, small dense LDL, high Lp(a)	Use non-HDL-C and apoB; measure Lp(a) once
CAD	Premature, diffuse, multivessel	Treat risk factors earlier and to lower targets
Liver	High prevalence of MASLD (fatty liver)	Check ALT and FIB-4 in every metabolic patient

Metabolic syndrome — the cluster

Metabolic syndrome is the clustering of central obesity, raised BP, dysglycaemia and atherogenic dyslipidaemia. It doubles cardiovascular risk and increases diabetes risk five-fold. The **harmonised definition (IDF/AHA/NHLBI 2009)** with South Asian waist cut-offs is preferred.

Component (any 3 of 5)	Cut-off (South Asian)
Waist circumference	≥90 cm men, ≥80 cm women
Triglycerides	≥150 mg/dl, or on drug treatment
HDL-C	<40 mg/dl men, <50 mg/dl women, or on drug treatment
Blood pressure	≥130/85 mmHg, or on antihypertensive treatment
Fasting glucose	≥100 mg/dl, or on glucose-lowering treatment

KEY POINT The IDF 2005 definition makes central obesity mandatory plus two other components. The harmonised definition does not — useful in the lean Indian with high TG, low HDL-C and impaired fasting glucose.

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Cardiovascular–kidney–metabolic (CKM) syndrome

The American Heart Association (2023) recast these conditions as **CKM syndrome**: a continuum in which excess and dysfunctional fat drives metabolic risk factors, which damage the heart and kidneys. Staging helps decide how aggressively to treat.

CKM stage	Description	Focus of care
Stage 0	No CKM risk factors	Primordial prevention: healthy diet, activity, sleep, no tobacco
Stage 1	Excess or dysfunctional adiposity (overweight/obesity, abdominal obesity, prediabetes)	Weight reduction ≥5%; lifestyle; treat prediabetes
Stage 2	Metabolic risk factors (hypertension, diabetes, hypertriglyceridaemia, metabolic syndrome) or CKD	Treat each factor to target; organ-protective drugs (SGLT2i, GLP-1 RA, RAS blockers)
Stage 3	Subclinical CVD (raised CAC, LVH, raised NT-proBNP/troponin) or high predicted risk / very high-risk CKD	Intensify: statin to lower LDL-C, BP to <130/80, cardiorenal drugs
Stage 4	Clinical CVD (CAD, HF, stroke, PAD, AF) ± kidney failure	Secondary prevention; disease-specific therapy

How the four diseases damage the body

Target organ	Hypertension	Diabetes	Dyslipidaemia	Obesity
Heart	LVH, HFpEF, AF, CAD	CAD, HF, cardiomyopathy	Atherosclerotic CAD	HFpEF, AF, CAD
Brain	Stroke (haemorrhagic & ischaemic), dementia	Ischaemic stroke, cognitive decline	Ischaemic stroke	Stroke, depression
Kidney	Hypertensive nephrosclerosis	Diabetic kidney disease (commonest cause of ESKD)	Accelerates CKD	Obesity-related glomerulopathy
Eye	Hypertensive retinopathy	Diabetic retinopathy, cataract	Lipaemia retinalis, xanthelasma	Idiopathic intracranial hypertension
Vessels	Aortic dissection, aneurysm, PAD	PAD, microangiopathy	PAD, carotid disease	Venous thromboembolism
Other	—	Neuropathy, foot ulcer, infections	Pancreatitis (TG >880 mg/dl)	OSA, MASLD, OA, PCOS, cancers

KEY POINT Never treat one number in isolation. A patient referred for ‘sugar’ must leave with BP, lipids, weight, kidney function, eyes, feet and tobacco status checked and addressed — that is where most of the mortality benefit lies.

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CHAPTER 2

Cardiovascular Risk Assessment

Estimating 10-year risk to decide how hard and how early to treat

Why calculate risk?

The benefit of any preventive drug depends on the patient's **absolute risk**. A 45-year-old non-smoker with BP 142/92 and a 65-year-old diabetic smoker with the same BP do not need the same intensity of treatment. Risk calculators combine age, sex, BP, cholesterol, diabetes, smoking and kidney function into a 10-year probability of a cardiovascular event. Patients with established disease are already at high or very high risk and **do not need a calculator**.

Risk calculators in current use

Tool (source)	Population / outcome	Inputs	Notes for India
PREVENT (AHA 2023)	Age 30–79; 10- and 30-year total CVD, ASCVD and HF	Age, sex, TC, HDL-C, SBP, BP treatment, diabetes, smoking, eGFR, statin; optional UACR, HbA1c, social deprivation	Replaces the Pooled Cohort Equations in the 2025 ACC/AHA BP and 2026 ACC/AHA lipid guidelines; no race term
SCORE2 / SCORE2-OP (ESC 2021)	Age 40–69 / ≥70; 10-year fatal + non-fatal CVD	Age, sex, SBP, non-HDL-C, smoking; region-calibrated	Use the 'high-risk region' chart for South Asians; SCORE2-Diabetes for diabetics
WHO/ISH risk charts (2019)	Age 40–74; 21 world regions incl. South Asia	Lab-based (cholesterol, diabetes) or non-lab (BMI)	Recommended by WHO HEARTS and India's NCD programme (NP-NCD) for primary care
QRISK3 (UK)	Age 25–84; 10-year CVD	Includes ethnicity (Indian, Pakistani, Bangladeshi), SLE, migraine, antipsychotics	Captures South Asian excess risk; validated in UK Indians
Pooled Cohort Equations (2013)	Age 40–79; 10-year ASCVD	Age, sex, race, TC, HDL-C, SBP, diabetes, smoking	Underestimates risk in Indians — now superseded

Risk categories — what the numbers mean

Category	ESC/EAS (SCORE2, 10-yr fatal + non-fatal CVD)	ACC/AHA (PREVENT-ASCVD, 10-yr)	Typical action
Low	<2% (age <50: <2.5%)	<3%	Lifestyle; drugs only if a single factor is very high
Moderate / borderline	2 to <10% (age-dependent)	3 to <5%	Lifestyle; consider statin with risk enhancers
High / intermediate	10 to <20%	5 to <10%	Statin; BP drugs at ≥130/80
Very high / high	≥20%	≥10%	Intensive LDL-C and BP lowering; cardiorenal drugs

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Who is automatically high or very high risk?

Very high risk (no calculator needed)	High risk
Documented ASCVD — MI, ACS, coronary or other revascularisation, ischaemic stroke/TIA, PAD, unequivocal plaque on imaging	Markedly raised single factor: TC >310 mg/dl, LDL-C >190 mg/dl or BP ≥180/110
Diabetes with target-organ damage, ≥3 major risk factors, or long-standing type 1 diabetes (>20 years)	Familial hypercholesterolaemia without other major risk factors
Severe CKD (eGFR <30 ml/min/1.73 m²)	Diabetes without target-organ damage, duration ≥10 years or with another risk factor
Familial hypercholesterolaemia with ASCVD or another major risk factor	Moderate CKD (eGFR 30–59)

Risk enhancers — moving a patient up a category

When the calculated risk is borderline, these factors justify more intensive therapy. Most guidelines list **South Asian ancestry** itself as a risk enhancer.

Clinical	Laboratory / imaging
Family history of premature ASCVD (male <55, female <65 years)	LDL-C 160–189 mg/dl; non-HDL-C ≥190 mg/dl
South Asian ethnicity	Lp(a) ≥50 mg/dl (≥105–125 nmol/l)
Metabolic syndrome; MASLD; prediabetes	apoB ≥130 mg/dl
CKD; albuminuria (UACR ≥30 mg/g)	Persistent TG ≥175 mg/dl
Chronic inflammatory disease (RA, psoriasis, SLE, HIV)	hs-CRP ≥2 mg/l
Premature menopause; pre-eclampsia, gestational diabetes	Ankle–brachial index <0.9
Psychosocial stress, deprivation, severe mental illness, OSA	Coronary artery calcium (CAC) >0 (>100 = treat as high risk)

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Coronary artery calcium (CAC) score

CAC (Agatston units)	Meaning	Action in a borderline-risk patient
0	No calcified plaque; 10-year event rate very low	Statin may be deferred (re-scan in 5–10 years) unless diabetes, smoker, FH or strong family history
1–99	Mild plaque	Statin favoured, especially age ≥55
≥100 or ≥75th percentile	Moderate–severe plaque	Statin indicated; treat as high risk (aspirin may be considered if bleeding risk low)
≥300 / ≥1000	Extensive plaque	Manage like secondary prevention (very high risk)

KEY POINT Lifetime risk matters in young Indians. A 35-year-old with BP 150/95, LDL-C 170 and prediabetes may have a 10-year risk below 5% but a 30-year risk above 30%. Use PREVENT 30-year risk or ‘risk age’ to motivate early treatment.

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CLINICAL PEARL Recalculate risk after the first set of results and whenever a new risk factor appears. Never use a calculator in a patient who already has ASCVD, FH, CKD stage 4 or diabetes with organ damage — they are high or very high risk by definition.

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CHAPTER 3

Lifestyle Therapy — The Common Foundation

Diet, salt, activity, sleep, tobacco and alcohol — prescribed like a drug

Lifestyle therapy is not ‘advice’; it is the first and permanent line of treatment for all four diseases, and it should be **prescribed with the same precision as a drug** — what, how much, how often, and when to review. Every patient, at every visit, on every drug, continues lifestyle therapy.

Effect size of lifestyle measures

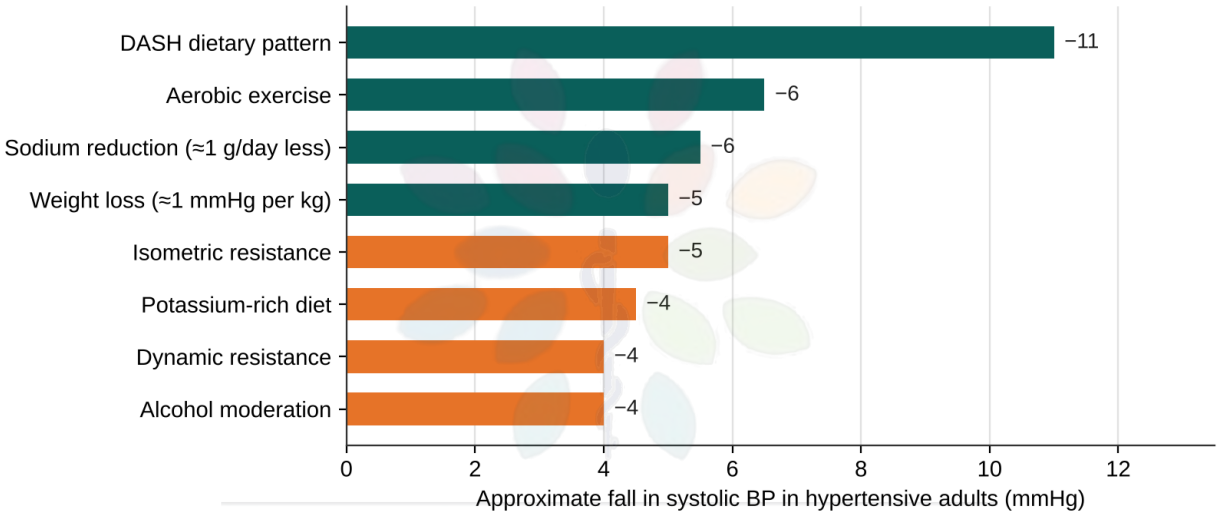


Figure 3.1. Expected fall in systolic BP with each lifestyle measure in hypertensive adults (ACC/AHA). Effects are roughly additive — combining three or four measures can match one or two drugs.

Measure	BP (systolic)	HbA1c	LDL-C / TG	Weight
5–10% weight loss	−5 to −10 mmHg	−0.5 to −1.0%	TG −20%; HDL-C ↑	—
≥15% weight loss	−10 to −15 mmHg	Remission in ≈50–60% of recent-onset T2D	TG −30–40%	—
DASH / Mediterranean diet	−8 to −11 mmHg	−0.3 to −0.5%	LDL-C −10%	Modest
Salt <5 g/day	−5 to −6 mmHg	—	—	—
150 min/week aerobic + resistance	−5 to −8 mmHg	−0.5 to −0.7%	TG −10–20%; HDL-C ↑	Prevents regain
Soluble fibre / plant sterols	—	Small	LDL-C −5 to −10%	—
Stop smoking	Acute rise abolished	Improves insulin sensitivity	HDL-C ↑	Small gain (1–4 kg)

Diet — what to eat

No single diet suits everyone; adherence matters more than the label. The **DASH** and **Mediterranean** patterns have the strongest outcome evidence. For Indians, the **ICMR–NIN Dietary Guidelines (2024)** ‘My Plate for the Day’ translates these into local foods.

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Food group	Recommendation	Indian examples
Vegetables & fruits	Half the plate; ≥400–500 g/day; prefer whole fruit to juice	Leafy greens, beans, gourds, guava, papaya, orange
Whole grains / millets	Quarter of the plate; replace refined rice and maida	Brown/hand-pounded rice, ragi, jowar, bajra, broken wheat, oats
Protein	Quarter of the plate; pulses daily; fish 2×/week	Dal, chana, rajma, curd, paneer (low-fat), egg, fish, chicken
Nuts & seeds	20–30 g/day	Almonds, walnuts, groundnuts, flax seeds
Fats & oils	Total visible oil ≈20–25 g (4–5 tsp)/day; rotate oils; avoid trans fat	Mustard, groundnut, rice bran, olive; limit ghee, coconut oil, vanaspati
Dairy	Low-fat milk/curd 300 ml/day	Buttermilk, curd
Sugar	Free sugars <5–10% of energy (<25 g/day ideal)	Avoid sweets, sugary tea/coffee, soft drinks, packaged juices
Salt	<5 g/day (one level teaspoon) — includes hidden salt	Avoid pickles, papad, chips, namkeen, sauces, processed food

INDIA NOTE The Indian plate is typically 60–70% carbohydrate, mostly refined rice or wheat. Simply **reducing the cereal portion by one-third and replacing it with dal, vegetables, curd or egg** lowers post-meal glucose, triglycerides and weight.

The DASH diet at a glance

Component	Daily target (2000 kcal)
Vegetables / fruits	4–5 servings each
Whole grains	6–8 servings
Low-fat dairy	2–3 servings
Lean meat, fish, poultry	≤6 servings (≈30 g each)
Nuts, seeds, legumes	4–5 servings per week
Potassium	3.5–5 g/day from food (avoid supplements in CKD or on RAS blockers/MRA)
Sodium	<2 g sodium (= 5 g salt); ideal 1.5 g

Physical activity — the exercise prescription (FITT)

Element	Aerobic	Resistance	Flexibility / balance
Frequency	≥5 days/week; no more than 2 consecutive days off	2–3 non-consecutive days/week	2–3 days/week (older adults)
Intensity	Moderate (can talk, not sing; 50–70% HRmax) or vigorous	Moderate–vigorous; 8–12 repetitions to near fatigue	Stretch to mild tension
Time	150–300 min/week moderate or 75–150 min vigorous; ≥10-min bouts count	8–10 exercises, major muscle groups, 1–3 sets	10–30 s per stretch
Type	Brisk walking, cycling, swimming, dancing, yoga (dynamic)	Body weight, bands, free weights, machines	Yoga, tai chi

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- **Break up sitting**: stand or walk for 3–5 minutes every 30 minutes — lowers post-meal glucose.
- A **10–15-minute walk after each main meal** blunts post-prandial glucose spikes.
- **Weight loss** needs 200–300 min/week; **weight maintenance** after loss needs ≥200 min/week.
- Isometric handgrip or wall-squat training (4 × 2 min, 3 days/week) lowers BP by ≈5 mmHg.

> **CAUTION** Before vigorous exercise, screen for symptoms of CAD, uncontrolled BP (>180/110), proliferative retinopathy (avoid heavy lifting, head-down positions), neuropathy (foot care, non-weight-bearing exercise if ulcer) and hypoglycaemia risk on insulin or sulfonylureas (check glucose; carry 15 g glucose).

Alcohol

No level of alcohol is protective. Advise **abstinence** where possible; if drinking, limit to ≤1 standard drink (10 g ethanol) per day for women and ≤2 for men, with alcohol-free days. Alcohol raises BP, triglycerides and weight, causes hypoglycaemia with insulin or sulfonylureas, and interacts with metformin.

Tobacco cessation — the single most effective preventive measure

Use the **5 A's** (Ask, Advise, Assess, Assist, Arrange) at every visit. Combining counselling with drug therapy doubles or triples quit rates. India's quit-line: **1800-11-2356** (National Tobacco Quit Line).

Drug	Dose	Duration	Cautions
Nicotine patch	>10 cigarettes/day: 21 mg/24 h; ≤10/day: 14 mg; step down every 2–4 weeks	8–12 weeks	Skin irritation, vivid dreams (remove at night)
Nicotine gum / lozenge	2 mg (<25/day) or 4 mg (≥25/day); 1 piece every 1–2 h, max 24/day	12 weeks	'Chew and park'; jaw pain, dyspepsia
Varenicline	0.5 mg OD × 3 days → 0.5 mg BD × 4 days → 1 mg BD; quit day 1–2 weeks after start	12 weeks (+12 to prevent relapse)	Nausea (take with food), sleep disturbance; halve dose if eGFR <30
Bupropion SR	150 mg OD × 3 days → 150 mg BD	7–12 weeks	Contraindicated in seizures, eating disorders, MAOI use; insomnia
Combination (patch + short-acting NRT, or varenicline + NRT)	As above	12 weeks	Most effective for heavy smokers

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Sleep, stress and mental health

- **Sleep 7–9 hours**; short sleep and shift work worsen insulin resistance, appetite and BP.
- Screen for **obstructive sleep apnoea** (STOP-BANG ≥3) in every obese or resistant-hypertensive patient.
- Screen for **depression** (PHQ-9) and **diabetes distress** — both predict poor adherence.
- Yoga, pranayama, mindfulness and regular routine lower BP by 3–5 mmHg and improve glycaemia modestly.

Making change happen — behavioural tools

Technique	How to use it in a 10-minute visit
SMART goals	Specific, measurable, achievable, relevant, time-bound: 'walk 30 min after dinner, 5 days a week, for 4 weeks'
Self-monitoring	Food diary or app, step counter (target 7000–10 000 steps), home BP and glucose log, weekly weight

Technique	How to use it in a 10-minute visit
Motivational interviewing	Ask, listen, reflect; let the patient state reasons for change
Family involvement	Cook for the household, not the patient alone; involve the person who prepares meals
Follow-up	Review within 4–12 weeks; praise progress; adjust one goal at a time

KEY POINT Give the lifestyle prescription in writing, like a drug prescription. Record weight, waist, BP and the agreed goal at every visit — what gets measured gets done.

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CHAPTER 4

Hypertension — Definition, Measurement and Classification

Getting the number right before treating it

Definition

Hypertension is a **persistent elevation of arterial blood pressure** to a level at which treatment reduces cardiovascular events. Because the risk of stroke and coronary disease rises continuously from 115/75 mmHg (each 20/10 mmHg rise doubles cardiovascular mortality), any cut-off is a practical threshold, not a biological one. International guidelines therefore differ slightly.

Classification — the major guidelines compared

Guideline	Normal	Intermediate	Hypertension
ACC/AHA 2025 (USA)	<120/<80	Elevated: 120–129/<80	Stage 1: 130–139 or 80–89 Stage 2: ≥140 or ≥90 Severe: ≥180 and/or ≥120
ESC 2024 (Europe)	Non-elevated: <120/<70	Elevated BP: 120–139/70–89	Hypertension: ≥140/≥90
ISH 2020 (International)	<130/<85	High-normal: 130–139/85–89	Grade 1: 140–159/90–99 Grade 2: ≥160/≥100
Indian Guidelines (IGH-IV, 2019) / NP-NCD	<130/<85 (optimal <120/80)	High-normal: 130–139/85–89	Stage 1: 140–159/90–99 Stage 2: ≥160/≥100 Isolated systolic: ≥140/<90

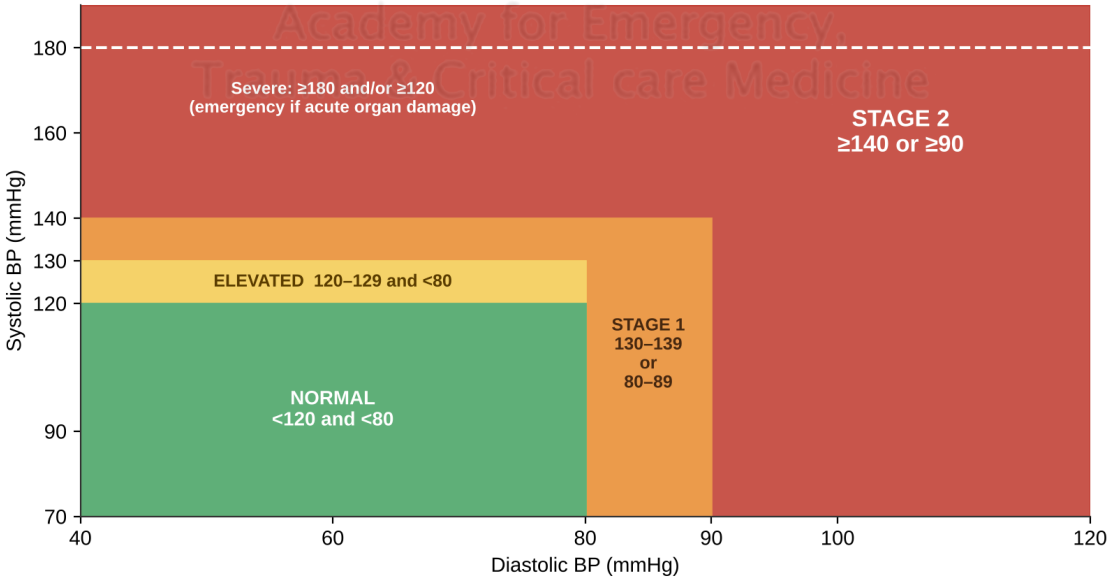


Figure 4.1. ACC/AHA 2025 BP categories. When systolic and diastolic values fall in different categories, the higher category applies.

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GUIDELINE Whatever the label, all guidelines agree on three things: confirm the diagnosis with out-of-office readings, start drugs at ≥140/90 in everyone, and start drugs at ≥130/80 in high-risk patients (CVD, diabetes, CKD or high calculated risk) if lifestyle therapy for 3–6 months has not succeeded.

Measuring BP correctly — the most neglected skill

Step	Correct technique	Error if ignored
Prepare	No caffeine, exercise or smoking for 30 min; empty bladder; sit quietly 5 min	Falsely high by 5–15 mmHg
Position	Seated, back supported, feet flat, legs uncrossed, arm supported at heart level	Unsupported back/arm: +5–10 mmHg
Cuff	Bladder encircles 80% of arm; adult large cuff if arm >32 cm; bare arm	Small cuff: +2–10 mmHg
Talking	Neither patient nor observer talks	+10–15 mmHg
Readings	2–3 readings 1 min apart; average the last two	Single reading misclassifies
Both arms	At first visit; use the higher arm thereafter (difference >10 mmHg is abnormal)	Misses subclavian stenosis
Standing	At 1 and 3 min in elderly, diabetics, those with dizziness	Misses orthostatic hypotension (fall ≥20/10)
Device	Validated automated upper-arm oscillometric device (validatebp.org, STRIDE BP)	Wrist and unvalidated devices are inaccurate

Out-of-office BP — confirming the diagnosis

Office BP alone misclassifies up to one-third of patients. Confirm with **home BP monitoring (HBPM)** or **24-hour ambulatory BP monitoring (ABPM)** before lifelong treatment, unless BP is ≥180/110 or there is target-organ damage.

Method	Protocol	Threshold — ACC/AHA	Threshold — ESC/ISH
Office	Average of ≥2 readings on ≥2 occasions	≥130/80	≥140/90
Home (HBPM)	Duplicate readings morning and evening for 4–7 days; discard day 1	≥130/80	≥135/85
ABPM — daytime (awake)	Readings every 15–30 min	≥130/80	≥135/85
ABPM — night-time (asleep)	Readings every 30–60 min	≥110/65	≥120/70
ABPM — 24-hour mean	≥70% valid readings	≥125/75	≥130/80

BP phenotypes

Phenotype	Office BP	Out-of-office BP	Significance and action
Sustained hypertension	High	High	Treat
White-coat hypertension	High	Normal	15–30% of ‘hypertensives’; risk modestly ↑; lifestyle, annual HBPM/ABPM; no drugs unless high risk
Masked hypertension	Normal	High	Common in smokers, diabetics, CKD, young men; risk as high as sustained HTN — treat

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Phenotype	Office BP	Out-of-office BP	Significance and action
White-coat effect / masked uncontrolled	High / normal on treatment	Normal / high	Avoid over- or under-treatment; titrate to home BP
Isolated systolic hypertension	SBP ≥140, DBP <90	—	Elderly (stiff arteries) — treat; young (spurious, tall men) — check central BP
Isolated diastolic hypertension	SBP <140, DBP ≥90	—	Young adults; lower risk; lifestyle ± drugs
Nocturnal hypertension / non-dipper	Any	Night fall <10% (reverse dipper: night > day)	Strong predictor of stroke; look for OSA, CKD, diabetes, autonomic failure
Morning surge	Any	Large early-morning rise	Associated with stroke; long-acting drugs

Etiological classification

Type	Proportion	Features
Primary (essential)	85–90%	Polygenic, lifestyle-driven; gradual onset age 30–60; family history
Secondary	10–15% (up to 30% in resistant HTN)	Identifiable cause: renal parenchymal, renovascular, primary aldosteronism, OSA, phaeochromocytoma, Cushing's, thyroid, coarctation, drugs — see Chapter 8

CLINICAL PEARL Hypertension is diagnosed on the **average of repeated, properly measured readings** — never on one reading in a busy OPD. A patient who has 'high BP only in the clinic' deserves a home monitor before a lifelong prescription.

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CHAPTER 5

Initial Evaluation and Basic Investigations in Hypertension

History, examination, baseline tests and hypertension-mediated organ damage

The initial evaluation has four aims: **confirm** hypertension, **assess total cardiovascular risk**, detect **hypertension-mediated organ damage (HMOD)**, and **screen for secondary causes**.

History

Ask about	Why
Duration, previous readings, treatment and side effects	Course, adherence, drug intolerance
Symptoms of target-organ damage: chest pain, dyspnoea, palpitations, claudication, TIA, visual loss, nocturia, oedema	HMOD and comorbidity
Clues to secondary causes: paroxysms of headache–sweating–palpitation, muscle weakness/cramps, snoring/daytime sleepiness, weight gain with striae, flash pulmonary oedema	Phaeochromocytoma, hyperaldosteronism, OSA, Cushing's, renovascular disease
Drugs and substances: NSAIDs, steroids, oral contraceptives, decongestants, erythropoietin, cyclosporine, VEGF inhibitors, licorice, alcohol, cocaine, energy drinks	Drug-induced or aggravated HTN
Risk factors: smoking, diabetes, lipids, family history of premature CVD/HTN, salt and alcohol intake, activity, sleep, stress	Total risk and lifestyle targets
Women: pre-eclampsia, gestational diabetes, contraception, pregnancy plans	Drug choice (avoid ACEi/ARB if pregnancy possible)

Physical examination

- BP in both arms (and legs if young — coarctation), standing BP, heart rate and rhythm (AF).
- Weight, height, BMI, waist circumference.
- Signs of HMOD: displaced apex, S4, carotid/renal/femoral bruits, absent pedal pulses, oedema.
- Signs of secondary causes: Cushingoid features, thyroid swelling, radio-femoral delay, palpable kidneys (polycystic), neurofibromas/café-au-lait spots.
- Fundoscopy (mandatory in grade 2/stage 2 and in emergencies): haemorrhages, exudates, papilloedema.

Basic investigations — for every patient

Test	Purpose	Interpretation
Serum creatinine with eGFR (CKD-EPI)	Renal function; drug dosing	eGFR <60 = CKD; baseline before ACEi/ARB/diuretics
Serum sodium and potassium	Baseline; clue to aldosteronism	Low K^+ (<3.5) unprovoked or with low-dose diuretic → screen for primary aldosteronism
Fasting plasma glucose and/or HbA1c	Diabetes / prediabetes	See Chapter 10
Fasting or non-fasting lipid profile	Risk estimation	See Chapter 23

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Test	Purpose	Interpretation
Urine dipstick (protein, blood) and urine albumin–creatinine ratio (UACR)	Kidney damage	UACR ≥30 mg/g = albuminuria (HMOD, higher CV risk)
12-lead ECG	LVH, ischaemia, AF, conduction disease	Sokolow-Lyon SV1+RV5/6 >35 mm; Cornell RaVL+SV3 >28 mm (men) / >20 mm (women)
Haemoglobin / CBC	Anaemia (CKD), polycythaemia	—
Serum uric acid	Gout risk with diuretics	Prefer losartan if gout
TSH (selected)	Hypo- or hyperthyroidism	If symptoms or resistant HTN

Assessing hypertension-mediated organ damage (HMOD)

Organ	Basic test	Advanced / recommended test	Abnormal value
Heart	ECG	Echocardiography (LV mass, LA size, diastolic function), NT-proBNP	LV mass index >115 g/m² (men), >95 g/m² (women); LA volume index >34 ml/m²
Kidney	eGFR, UACR	Renal ultrasound with Doppler (if indicated)	eGFR <60; UACR ≥30 mg/g
Eyes	Fundoscopy	Fundus photography / OCT	Haemorrhages, exudates, papilloedema (grade 3–4)
Arteries	Pulse examination, ABI	Carotid ultrasound (plaque), pulse-wave velocity (PWV), CAC score	ABI <0.9; carotid-femoral PWV >10 m/s; carotid plaque
Brain	Cognitive screen (MoCA) in elderly	MRI (white-matter lesions, lacunes, microbleeds) if symptoms	—

Fundoscopic grading (Keith–Wagener–Barker, modified)

Grade	Findings	Meaning
1	Generalised arteriolar narrowing	Mild; common with age
2	Focal narrowing, arteriovenous nipping	Chronic hypertension
3	Flame haemorrhages, cotton-wool spots, hard exudates	Accelerated hypertension — urgent treatment
4	Grade 3 + papilloedema	Malignant hypertension — hypertensive emergency

Red flags — when to search for a secondary cause

- Onset <30 years (especially without obesity or family history), or new onset after 60 years.
- Severe (≥180/110) or resistant hypertension; sudden worsening of previously controlled BP.
- Spontaneous or diuretic-induced hypokalaemia; adrenal incidentaloma.
- Rise in creatinine >30% after starting ACEi/ARB; flash pulmonary oedema; renal size asymmetry >1.5 cm; abdominal bruit.
- Paroxysmal symptoms; features of Cushing's, thyroid disease, acromegaly; snoring with daytime sleepiness.
- Disproportionate HMOD for the BP level; malignant hypertension.

KEY POINT Every newly diagnosed hypertensive needs only **six inexpensive tests**: creatinine/eGFR, Na^+/K^+, glucose/HbA1c, lipids, urine albumin (or dipstick) and ECG. Add echocardiography and fundoscopy when available — they often change the BP target or drug choice.

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CHAPTER 6

Antihypertensive Drugs — Class, Drug and Dose

First-line, add-on and newer agents with doses, cautions and monitoring

Four classes are **first-line** in all major guidelines: **ACE inhibitors (A), angiotensin receptor blockers (A), calcium-channel blockers (C)** and **thiazide/thiazide-like diuretics (D)**. **β-blockers** are first-line when there is a compelling indication (angina, post-MI, HFrEF, AF rate control, pregnancy planning). Doses below are adult oral doses; **start low and titrate every 2–4 weeks**.

First-line drugs

Class / drug	Usual dose	Frequency	Key notes
ACE INHIBITORS (ACEi)			
Enalapril	2.5–20 mg	OD–BD	Cheap; cough 5–20%
Ramipril	2.5–10 mg	OD	HOPE trial — vascular protection
Perindopril	4–8 mg (erbumine) / 5–10 mg (arginine)	OD	ASCOT, EUROPA; with indapamide/amlodipine SPCs
Lisinopril	5–40 mg	OD	Renal excretion — reduce in CKD
Captopril	12.5–50 mg	BD–TDS	Short-acting; used in renin tests
ANGIOTENSIN RECEPTOR BLOCKERS (ARB)			
Telmisartan	20–80 mg	OD	Longest half-life (24 h); widely used in India
Losartan	25–100 mg	OD–BD	Uricosuric (good in gout); weakest ARB
Olmesartan	10–40 mg	OD	Rare sprue-like enteropathy
Valsartan	80–320 mg	OD	HF evidence
Irbesartan	150–300 mg	OD	Diabetic nephropathy (IDNT)
Candesartan	8–32 mg	OD	HF evidence (CHARM)
Azilsartan medoxomil	40–80 mg	OD	Most potent ARB
DIHYDROPYRIDINE CALCIUM-CHANNEL BLOCKERS (CCB)			
Amlodipine	2.5–10 mg	OD	Half-life 35–50 h; ankle oedema dose-related
Cilnidipine	5–20 mg	OD	L/N-type; less oedema and reflex tachycardia (India, Japan)
Nifedipine ER / GITS	30–90 mg	OD	Never short-acting nifedipine for HTN
Felodipine ER	2.5–10 mg	OD	—
Lercanidipine	10–20 mg	OD	Less oedema
Benidipine	4–8 mg	OD	L/T/N-type (India, Japan)
THIAZIDE AND THIAZIDE-LIKE DIURETICS			
Chlorthalidone	12.5–25 mg	OD (morning)	Preferred: long-acting, outcome data; effective down to eGFR 15–30 (CLICK)

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Class / drug	Usual dose	Frequency	Key notes
Indapamide SR	1.5 mg (IR 1.25–2.5 mg)	OD	Outcome data in elderly (HYVET) and stroke (PROGRESS)
Hydrochlorothiazide	12.5–50 mg	OD	Shorter-acting; mainly in SPCs

Second-line and add-on drugs

Class / drug	Usual dose	Frequency	Key notes
MINERALOCORTICOID RECEPTOR ANTAGONISTS (MRA)			
Spironolactone	12.5–50 mg	OD	Best 4th drug in resistant HTN (PATHWAY-2); gynaecomastia
Eplerenone	25–50 mg	OD–BD	Selective; fewer sexual side effects
Finerenone (non-steroidal)	10–20 mg	OD	Indicated for T2D with CKD/albuminuria; modest BP effect
β-BLOCKERS			
Metoprolol succinate (ER)	25–200 mg	OD	HFrEF evidence; cardioselective
Bisoprolol	2.5–10 mg	OD	Cardioselective; HFrEF evidence
Nebivolol	2.5–10 mg	OD	Vasodilating (NO); fewer metabolic effects
Carvedilol	6.25–25 mg	BD	α + β blocker; HFrEF; useful in diabetes
Labetalol	100–400 mg (max 2400 mg/day)	BD–TDS	Pregnancy; hypertensive emergencies (IV)
Atenolol	25–100 mg	OD	Inferior stroke protection — not preferred
LOOP DIURETICS (for CKD eGFR <30 or volume overload)			
Furosemide	20–80 mg	BD	Short-acting — poor for BP alone
Torsemide	5–20 mg	OD	Longer-acting; preferred loop for HTN
OTHER POTASSIUM-SPARING DIURETIC			
Amiloride	5–10 mg (max 20)	OD	Alternative to spironolactone; Liddle syndrome
α1-BLOCKERS			
Prazosin GITS / XL	2.5–20 mg	OD	Phaeochromocytoma (pre-op), BPH; orthostatic hypotension
Doxazosin	1–8 mg (XL 4–8 mg)	OD	First-dose hypotension — give at bedtime
CENTRAL SYMPATHOLYTICS			
Clonidine	0.1–0.3 mg (max 0.6 mg/day)	BD–TDS	Rebound hypertension on abrupt withdrawal; sedation
Methyldopa	250–1000 mg (max 3 g/day)	BD–TDS	Pregnancy; hepatitis, haemolytic anaemia
Moxonidine	0.2–0.6 mg	OD–BD	Imidazoline agonist; avoid in HF
DIRECT VASODILATORS			
Hydralazine	25–100 mg (max 200 mg/day)	BD–TDS	With β-blocker + diuretic; lupus-like syndrome
Minoxidil	2.5–40 mg	OD–BD	Last resort; fluid retention, hirsutism, pericardial effusion

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Class / drug	Usual dose	Frequency	Key notes
OTHERS			
Sacubitril–valsartan (ARNI)	50–200 mg	BD	HFrEF/HFpEF; potent BP lowering (approved for HTN in some countries)
Aliskiren (renin inhibitor)	150–300 mg	OD	Never with ACEi/ARB

Newer agents for resistant hypertension

Drug	Mechanism	Dose	Status / notes
Aprocitentan	Dual endothelin receptor antagonist	12.5 mg OD	FDA 2024 (PRECISION); fluid retention, anaemia; teratogenic
Baxdrostat	Aldosterone synthase inhibitor	1–2 mg OD (per label)	FDA May 2026 (BaxHTN: −9 to −10 mmHg vs placebo on 2–5 drugs); hyperkalaemia, hyponatraemia
Lorundrostat	Aldosterone synthase inhibitor	Investigational	Phase 3 positive
Zilebesiran	siRNA against hepatic angiotensinogen	Twice-yearly SC (investigational)	Phase 3 outcome trial ongoing
Renal denervation	Catheter ablation of renal sympathetic nerves	Procedure	≈5–10 mmHg fall; for resistant HTN after expert review (ESC 2024, ACC/AHA 2025)

Compelling indications — matching drug to patient

Condition	Preferred	Avoid / caution
Diabetes	ACEi/ARB (especially with albuminuria) + CCB or thiazide; SGLT2i adds 3–5 mmHg	High-dose thiazide + β-blocker (metabolic effects)
CKD with albuminuria	ACEi or ARB at maximum tolerated dose; + SGLT2i; + finerenone (T2D)	ACEi + ARB together; NSAIDs
Coronary artery disease / post-MI	β-blocker + ACEi/ARB; CCB for angina	Short-acting nifedipine
Heart failure with reduced EF	ARNI/ACEi/ARB + evidence-based β-blocker + MRA + SGLT2i	Non-DHP CCB (verapamil, diltiazem), moxonidine, α-blockers
Heart failure with preserved EF	SGLT2i, diuretic, MRA/finerenone, ARNI; GLP-1 RA/tirzepatide if obese	—
Atrial fibrillation	β-blocker or non-DHP CCB (rate), ACEi/ARB	—
Previous stroke	ACEi + thiazide-like (indapamide), or ARB/CCB	—
Elderly / isolated systolic HTN	CCB, thiazide-like, ACEi/ARB	α-blockers, central agents (falls)
Pregnancy or planning pregnancy	Labetalol, nifedipine ER, methyldopa	ACEi, ARB, MRA, aliskiren, atenolol — contraindicated
Gout	Losartan, CCB	Thiazides and loops
Asthma / COPD	CCB, ACEi/ARB	Non-selective β-blockers
BPH	α-blocker (as add-on only)	—
Migraine / essential tremor / thyrotoxicosis	Propranolol	—
Osteoporosis	Thiazide (reduces calcium loss)	Loop diuretics

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Contraindications and adverse effects

Class	Absolute contraindications	Important adverse effects	Monitoring
ACEi	Pregnancy, angioedema (any cause), bilateral renal artery stenosis, K^+ >5.5	Dry cough, hyperkalaemia, angioedema (more in South Asians and Africans), first-dose hypotension	Creatinine and K^+ at 1–2 weeks; accept creatinine rise ≤30%
ARB	Pregnancy, bilateral RAS, K^+ >5.5	Hyperkalaemia, dizziness (no cough)	As ACEi
DHP-CCB	Severe aortic stenosis (relative), cardiogenic shock	Ankle oedema (not diuretic-responsive — add ACEi/ARB), flushing, headache, gum hypertrophy	Clinical
Non-DHP CCB	HFrEF, 2nd/3rd-degree AV block, sick sinus, with β-blocker	Bradycardia, constipation (verapamil)	Heart rate, ECG
Thiazide	Gout (relative), severe hyponatraemia	Hypokalaemia, hyponatraemia (elderly women), hyperuricaemia, hyperglycaemia, photosensitivity	Na^+/K^+ at 2–4 weeks, then 6–12 monthly
MRA	K^+ >5.0, eGFR <30 (relative)	Hyperkalaemia, gynaecomastia (spironolactone)	K^+ and creatinine at 1 week, 4 weeks, then 3-monthly
β-blocker	Asthma (non-selective), 2nd/3rd-degree AV block, HR <50	Fatigue, bradycardia, erectile dysfunction, masking hypoglycaemia, weight gain	Heart rate
α-blocker	Orthostatic hypotension, HF (as monotherapy)	Postural hypotension, first-dose syncope	Standing BP

CAUTION **Never combine an ACEi with an ARB** (or either with aliskiren): no extra benefit, more hyperkalaemia and acute kidney injury (ONTARGET, VA NEPHRON-D). A rise in creatinine of up to 30% after starting an ACEi/ARB is expected and is not a reason to stop — a rise >30% or K^+ >5.5 mmol/l is.

CLINICAL PEARL **Sick-day rule:** during vomiting, diarrhoea or dehydration, temporarily hold ACEi/ARB, diuretics, MRA, SGLT2 inhibitors, metformin and NSAIDs; restart 24–48 h after eating and drinking normally.

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CHAPTER 7

Step-Up Treatment and Combination Therapy in Hypertension

Thresholds, targets, the treatment algorithm and special populations

When to start drugs and what to aim for

Guideline	Start drugs	Target (office BP)
ACC/AHA 2025	≥140/90 in all; ≥130/80 if CVD, diabetes, CKD, or PREVENT 10-year CVD risk ≥7.5%; 130–139/80–89 with low risk after 3–6 months of lifestyle failure	<130/80 for all adults (encourage SBP <120 where tolerated)
ESC 2024	≥140/90 in all; 130–139/80–89 if high risk (CVD, diabetes, CKD, HMOD, SCORE2 ≥10%) after 3 months of lifestyle	SBP 120–129 if tolerated; otherwise 'as low as reasonably achievable'; <140/80 in frail/≥85 years
ISH 2020	≥140/90; ≥130/80 high risk	<130/80 (<140/90 in ≥65 years)
ADA 2026 (diabetes)	≥130/80	<130/80; SBP <120 in high CV/renal risk if safely achievable
KDIGO 2021 (CKD)	—	SBP <120 (standardised measurement) when tolerated

GUIDELINE Evidence from SPRINT, STEP and ESPRIT shows that an SBP target near 120 mmHg prevents more strokes, heart failure and deaths than 140 mmHg, including in people over 70 — provided BP is measured properly and orthostatic hypotension, falls and AKI are watched for.

Why combination therapy first?

Doubling the dose of one drug adds only ≈20% more BP lowering but many more side effects; **adding a second class is about five times more effective**. Most patients need two or three drugs. Starting with a **single-pill combination (SPC)** controls BP faster, improves adherence and reduces events.

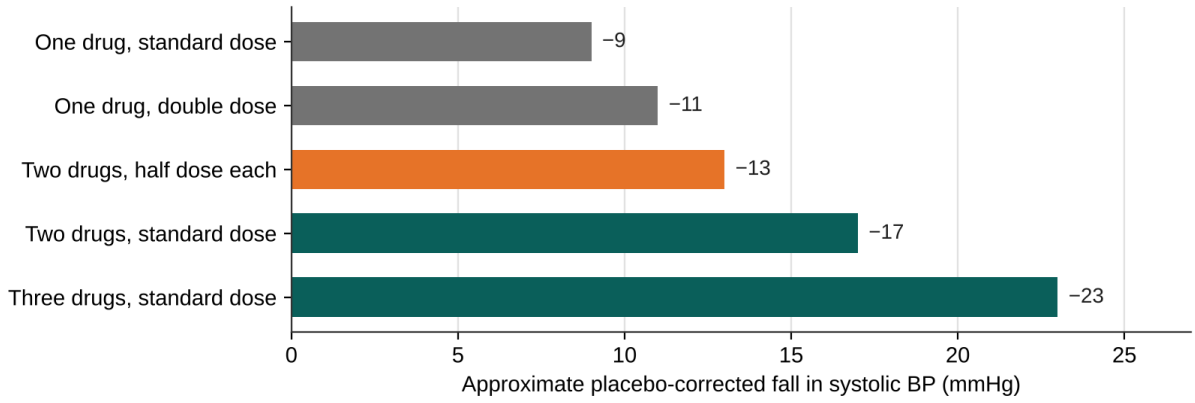


Figure 7.1. Approximate BP lowering with dose escalation versus combination (meta-analyses of Law and Wald). Adding a drug beats doubling a dose.

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The treatment algorithm (uncomplicated hypertension)

Hypertension treatment pathway (ESC 2024 / ACC/AHA 2025 / ISH 2020)

STEP 0 — Lifestyle for everyone

Diet, salt <5 g, weight loss, activity, alcohol and tobacco. In stage 1 / low risk, lifestyle alone for 3–6 months.

▼

STEP 1 — Dual combination, preferably as a single pill

ACEi or ARB + CCB, or **ACEi or ARB + thiazide-like diuretic**, low dose. Monotherapy only in: low-risk stage 1, age ≥80–85 or frail, orthostatic hypotension, BP close to target.

▼

STEP 2 — Up-titrate dual combination to full dose

e.g. telmisartan 80 + amlodipine 10, or telmisartan 80 + chlorthalidone 12.5–25. Review every 2–4 weeks; aim for control within 3 months.

▼

STEP 3 — Triple combination (A + C + D)

ACEi/ARB + CCB + thiazide-like diuretic, preferably as a single pill.

▼

STEP 4 — Resistant hypertension

Confirm adherence and home/ABPM; exclude secondary causes. **Add spironolactone 12.5–50 mg** (if K^+ <4.5, eGFR >45). Alternatives: eplerenone, amiloride, β-blocker, α-blocker, clonidine. Refer to a specialist.

CLINICAL PEARL β-blockers can be added at any step when there is a compelling indication (angina, post-MI, HFrEF, AF, heart-rate control).

Preferred, acceptable and unwise combinations

Combination	Verdict	Comment
ACEi/ARB + DHP-CCB	Preferred	ACCOMPLISH: superior to ACEi + HCTZ; CCB oedema reduced by RAS blockade
ACEi/ARB + thiazide-like	Preferred	Diuretic-induced hypokalaemia offset by RAS blockade
DHP-CCB + thiazide-like	Acceptable	Good in elderly, low-renin patients
β-blocker + DHP-CCB	Acceptable	Angina; reflex tachycardia offset
β-blocker + thiazide	Acceptable with caution	Increases new-onset diabetes — avoid in metabolic syndrome
ACEi + ARB / either + aliskiren	Avoid	Hyperkalaemia, AKI, no benefit
β-blocker + non-DHP CCB (verapamil/diltiazem)	Avoid	Bradycardia, heart block, HF
Two drugs of the same class	Avoid	Irrational

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Common single-pill combinations available in India

Combination	Common strengths (mg)
Telmisartan + amlodipine	40/5, 80/5, 40/10
Telmisartan + chlorthalidone	40/6.25, 40/12.5, 80/12.5
Telmisartan + hydrochlorothiazide	40/12.5, 80/12.5
Telmisartan + amlodipine + chlorthalidone (or HCTZ)	40/5/12.5, 80/5/12.5
Olmesartan + amlodipine (± HCTZ)	20/5, 40/5, 40/10; 20/5/12.5, 40/5/12.5
Losartan + amlodipine / + HCTZ	50/5; 50/12.5
Perindopril + amlodipine (± indapamide)	4/5, 8/5; 4/1.25/5
Ramipril + amlodipine	5/5
Amlodipine + atenolol / metoprolol / bisoprolol	5/50; 5/25–50; 5/2.5–5
Polypill (statin + aspirin + BP drugs)	Secondary prevention (TIPS-3, SECURE trials)

INDIA NOTE Public-sector protocols in India (India Hypertension Control Initiative, NP-NCD) use a simple, drug-specific step ladder — for example amlodipine 5 → amlodipine 10 → add telmisartan 40 → telmisartan 80 → add chlorthalidone 12.5 — with free drugs and nurse-led follow-up. Simple protocols improve population control rates.

Special populations

Population	Target	Preferred approach
Diabetes	<130/80 (SBP <120 if high risk)	ACEi/ARB-based combination; add SGLT2i/GLP-1 RA for organ protection
CKD without albuminuria	<130/80 (SBP <120 if tolerated)	Any first-line; thiazide-like (chlorthalidone) works to eGFR 15–30; loop if overloaded
CKD with albuminuria	As above	ACEi or ARB at maximal tolerated dose first; then SGLT2i, finerenone (T2D)
CAD	<130/80	β-blocker + ACEi/ARB ± CCB; avoid DBP <60–70 with LVH/CAD
Acute stroke	See Chapter 9	—
Previous stroke/TIA	<130/80	Start within days of stable stroke; ACEi/ARB + thiazide-like or CCB
Elderly 65–79	<130/80 if fit	Start dual low-dose combination; check standing BP
Age ≥80 or frail	<140/80 (ESC); individualise	Monotherapy start; avoid orthostatic hypotension; deprescribe if falls
Young women	<130/80	Avoid ACEi/ARB without reliable contraception; CCB, labetalol
Pregnancy	<140/90 (treat ≥140/90)	See Chapter 9
OSA	<130/80	CPAP plus drugs; spironolactone if resistant

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Follow-up and maintenance

Stage	Frequency	What to check
After starting/changing drugs	Every 2–4 weeks	Office and home BP, side effects, K^+/creatinine (ACEi/ARB/diuretic/MRA)
Once controlled	Every 3–6 months	BP, adherence, weight, lifestyle
Annually	Yearly	Creatinine, Na^+/K^+, glucose/HbA1c, lipids, UACR, ECG; repeat CV risk

- Use **once-daily, long-acting drugs and SPCs**; link dosing to a daily routine.
- Encourage **home BP monitoring** and team-based care (nurse, pharmacist) — improves control by 10–20%.
- Bedtime dosing offers no outcome advantage over morning dosing (TIME trial) — choose the time the patient remembers.
- **Deprescribe** carefully in frailty, recurrent falls, orthostatic hypotension, or SBP persistently <110.

KEY POINT The commonest cause of 'uncontrolled hypertension' is not a resistant disease but **clinical inertia** — failure to step up at a visit where BP is above target. If BP is not at target, change something at every visit.

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CHAPTER 8

Resistant and Secondary Hypertension — Advanced Evaluation

Pseudo-resistance, primary aldosteronism, renovascular disease, phaeochromocytoma and more

Definitions

Term	Definition
Resistant hypertension	BP above target despite ≥3 drugs of different classes at optimal doses, including a diuretic — or BP controlled only with ≥4 drugs — confirmed by out-of-office BP and adherence
Apparent resistant	Resistance not yet confirmed (adherence, measurement, white-coat effect not excluded)
Refractory hypertension	Uncontrolled on ≥5 drugs including chlorthalidone and an MRA
Pseudo-resistance	Apparent resistance due to poor technique, white-coat effect, non-adherence or inadequate regimen

Step 1 — exclude pseudo-resistance

Cause	How to detect	Fix
Poor measurement technique	Re-measure correctly with proper cuff	Train staff; validated device
White-coat effect	Home BP or ABPM	Titrate to out-of-office BP
Non-adherence (up to 50%)	Pill count, pharmacy record, direct questioning, urine/blood drug screening	SPC, once daily, reduce pill burden, counsel
Inadequate regimen	Low doses, no diuretic, short-acting drugs	Optimise A + C + D at full dose; chlorthalidone instead of HCTZ
Interfering substances	Drug history: NSAIDs, steroids, OCP, decongestants, licorice, alcohol, cocaine, calcineurin inhibitors, VEGF inhibitors, erythropoietin, antidepressants (SNRI, MAOI)	Stop or substitute
Volume expansion	High salt intake (24-h urine Na^+ >100 mmol), CKD	Salt restriction, loop diuretic if eGFR <30

Step 2 — screen for secondary causes

Cause	Clues	Screening test	Confirmation / localisation	Treatment
Primary aldosteronism (5–15% of all HTN; 20% of resistant)	Resistant HTN, hypokalaemia (only 30%), adrenal incidentaloma, AF, OSA, family history of early stroke	Aldosterone–renin ratio (ARR): aldosterone ≥10 ng/dl with ARR >20 (ng/dl per ng/ml/h); renin suppressed	Saline infusion (2 l over 4 h) or captopril / oral salt test; adrenal CT; adrenal venous sampling (AVS) before surgery	Unilateral: laparoscopic adrenalectomy; bilateral: spironolactone 12.5–100 mg or eplerenone 25–50 mg BD
Renal parenchymal disease (2–5%)	Raised creatinine, albuminuria, haematuria, oedema	eGFR, UACR, urine microscopy, renal ultrasound	Renal biopsy as indicated	RAS blockade, SGLT2i, BP <120–130 systolic

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Cause	Clues	Screening test	Confirmation / localisation	Treatment
Renovascular — atherosclerotic	Age >55, smoker, PAD/CAD, flash pulmonary oedema, creatinine rise >30% on ACEi/ARB, renal asymmetry, abdominal bruit	Renal artery Doppler	CT or MR angiography	Medical therapy (ACEi/ARB, statin, antiplatelet); stenting only for recurrent flash oedema or rapidly declining function
Fibromuscular dysplasia	Young woman, sudden severe HTN	Doppler, CTA	CT angiography ('string of beads')	Balloon angioplasty without stent
Obstructive sleep apnoea (25–50% of resistant HTN)	Obesity, snoring, witnessed apnoeas, daytime sleepiness, nocturnal HTN	STOP-BANG ≥3	Polysomnography (AHI ≥15)	CPAP, weight loss; tirzepatide improves OSA (SURMOUNT-OSA)
Phaeochromocytoma / paraganglioma (0.2–0.6%)	Paroxysms of headache, sweating, palpitation; labile BP; incidentaloma; MEN2, VHL, NF1	Plasma free metanephrines (supine) or 24-h urine fractionated metanephrines; >3–4× ULN is diagnostic	CT/MRI abdomen; ^{68}Ga-DOTATATE PET or MIBG; genetic testing	α-blockade first (phenoxybenzamine or doxazosin/prazosin 10–14 days), then β-blocker, high salt/fluid; surgery
Cushing's syndrome	Central obesity, purple striae, proximal myopathy, bruising, hypokalaemia, diabetes	1 mg overnight dexamethasone suppression (cortisol >1.8 µg/dl abnormal), late-night salivary cortisol, 24-h urinary free cortisol	ACTH, high-dose DST, MRI pituitary / CT adrenal, IPSS	Surgery; steroid-synthesis inhibitors
Thyroid disease	Hyperthyroid: systolic HTN, tachycardia; hypothyroid: diastolic HTN	TSH, FT4	—	Treat thyroid disease
Primary hyperparathyroidism	Hypercalcaemia, stones	Serum calcium, PTH	Sestamibi scan	Parathyroidectomy
Coarctation of aorta	Young, arm > leg BP, radio-femoral delay, rib notching	Arm–leg BP (gradient >20 mmHg)	Echo, CT/MR angiography	Stent or surgery
Monogenic (Liddle, GRA, AME)	Early onset, low renin, family history	Renin, aldosterone, K^+	Genetic testing	Amiloride (Liddle), dexamethasone (GRA)

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GUIDELINE The **Endocrine Society 2025** guideline suggests screening **all** people with hypertension for primary aldosteronism, and the ESC 2024 guideline recommends it for all with resistant or early-onset hypertension, hypokalaemia or adrenal nodules. Initial testing can be done **without stopping most drugs** (correct hypokalaemia first). A positive screen on an ACEi/ARB is strongly suggestive; β-blockers and clonidine suppress renin and can give false positives — false negatives occur with MRAs and diuretics.

Advanced investigations — summary

Investigation	Indication	What it adds
24-hour ABPM	White-coat/masked HTN, resistant HTN, nocturnal HTN, hypotension symptoms	True BP burden, dipping status
Echocardiography	Stage 2 HTN, ECG abnormality, symptoms	LVH, diastolic dysfunction, EF, aortic root

Investigation	Indication	What it adds
Carotid ultrasound / CAC	Risk reclassification	Subclinical atherosclerosis
Pulse-wave velocity	Specialist centres	Arterial stiffness
Renal Doppler / CTA / MRA	Suspected renovascular HTN	Stenosis, FMD
Plasma aldosterone & renin	Suspected primary aldosteronism	ARR
Adrenal CT; adrenal venous sampling	Confirmed PA, surgical candidate	Lateralisation
Plasma/urine metanephrines; ^{68}Ga-DOTATATE PET	Suspected phaeochromocytoma	Diagnosis and localisation
Polysomnography	Suspected OSA	AHI, oxygen desaturation
Genetic testing	Familial/early-onset syndromes, phaeochromocytoma	Cascade screening

Managing confirmed resistant hypertension

Optimise

Lifestyle (salt <5 g!), stop interfering drugs, A + C + D at maximum tolerated doses; switch HCTZ → chlorthalidone (or indapamide).

▼

4th drug

Spironolactone 25–50 mg (PATHWAY-2: −8.7 mmHg vs placebo). If not tolerated: eplerenone 50–100 mg, or **amiloride 10–20 mg** (equally effective). Check K^+ and creatinine at 1 and 4 weeks.

▼

5th drug

β-blocker (bisoprolol, nebivolol) if HR >70, or α-blocker (doxazosin), or clonidine. Loop diuretic if eGFR <30.

▼

Specialist options

Hydralazine, minoxidil; newer agents (aprocitentan, baxdrostat); **renal denervation** in selected patients with uncontrolled BP after expert review.

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CLINICAL PEARL Before labelling a patient as resistant, check three things: **Is the BP really high (home/ABPM)? Is the patient really taking the drugs? Is one of them a diuretic at an adequate dose?** — these explain most cases.

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CHAPTER 9

Hypertensive Emergencies and Hypertension in Pregnancy

Severe hypertension, organ-specific targets, IV drugs and pregnancy protocols

Severe hypertension — urgency versus emergency

Category	Definition	Management
Severe hypertension without acute organ damage ('urgency')	BP ≥180/110–120 without new or worsening organ damage	Rest, re-measure; restart/intensify oral drugs (amlodipine, ARB, labetalol, clonidine); lower over 24–48 h; review in 1 week. **No IV drugs, no sublingual nifedipine.**
Hypertensive emergency	Severe BP elevation with **acute** target-organ damage	ICU/HDU; arterial line; IV titratable drugs; condition-specific targets
Malignant hypertension	Severe HTN with bilateral flame haemorrhages, cotton-wool spots or papilloedema ± thrombotic microangiopathy, AKI	Emergency — reduce MAP by 20–25% over several hours

Target-organ damage that defines an emergency

- **Brain:** hypertensive encephalopathy (headache, confusion, seizures, PRES), intracerebral haemorrhage, acute ischaemic stroke.
- **Heart:** acute coronary syndrome, acute pulmonary oedema / acute HF.
- **Aorta:** acute aortic dissection.
- **Kidney:** AKI, thrombotic microangiopathy (haemolysis, low platelets).
- **Eye:** grade 3–4 retinopathy (malignant hypertension).
- **Pregnancy:** severe pre-eclampsia, eclampsia, HELLP.
- **Catecholamine crisis:** phaeochromocytoma, cocaine/amphetamine toxicity, clonidine withdrawal.

Condition-specific BP targets

Condition	Timeline and target	First-line drug(s)
Acute aortic dissection	SBP <120 and HR <60 within 20 min	Esmolol or labetalol FIRST (rate), then nitroprusside/nicardipine/NTG
Acute pulmonary oedema	SBP <140 immediately	IV nitroglycerin (± nitroprusside) + loop diuretic; NIV
Acute coronary syndrome	SBP <140 immediately	Nitroglycerin, labetalol/esmolol
Hypertensive encephalopathy / malignant HTN	MAP −20–25% over 1–4 h, then gradually	Labetalol, nicardipine, clevidipine
Acute ischaemic stroke — for thrombolysis	<185/110 before, <180/105 for 24 h after	Labetalol, nicardipine
Acute ischaemic stroke — no thrombolysis	Treat only if >220/120; lower by ≈15% in 24 h	Labetalol, nicardipine
Intracerebral haemorrhage (SBP 150–220)	SBP ≈140 (range 130–150) within 1 h; avoid <130 and large rapid falls	Nicardipine, labetalol, clevidipine

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Condition	Timeline and target	First-line drug(s)
Severe pre-eclampsia / eclampsia	<160/110 within 30–60 min; then 140–150/90–100	Labetalol IV, oral nifedipine, hydralazine IV + $MgSO_4$
Phaeochromocytoma / sympathomimetic crisis	MAP −20–25% in first hour	Phentolamine; benzodiazepines (cocaine); **no β-blocker first**
All other emergencies	MAP ↓ ≤25% in 1st hour; then 160/100 over 2–6 h; normal over 24–48 h	Labetalol, nicardipine

Intravenous antihypertensive drugs

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 HF
Nicardipine	5 mg/h, ↑ 2.5 mg/h every 5–15 min, max 15 mg/h	5–15 min / 4–6 h	Reflex tachycardia; avoid in severe AS
Clevidipine	1–2 mg/h, double every 90 s to max 32 mg/h	2–4 min / 5–15 min	Lipid emulsion — soy/egg allergy
Esmolol	Load 500 µg/kg over 1 min, then 50–300 µg/kg/min	1–2 min / 10–30 min	Bradycardia, HF, asthma
Nitroglycerin	5 µg/min, ↑ 5–10 µg/min every 3–5 min, max 200 µg/min	2–5 min / 5–10 min	Headache; venodilator — best for pulmonary oedema/ACS; PDE5 inhibitors
Sodium nitroprusside	0.25–0.5 µg/kg/min, max 10 µg/kg/min (keep ≤2 µg/kg/min if possible)	Seconds / 1–2 min	Cyanide/thiocyanate toxicity, light-sensitive, raises ICP; arterial line mandatory
Hydralazine	10–20 mg IV slowly (pregnancy 5–10 mg), repeat every 20–30 min	10–20 min / 2–4 h	Unpredictable; reflex tachycardia — avoid in ACS, dissection
Phentolamine	1–5 mg IV bolus (repeat every 5–15 min), max 15 mg	1–2 min / 10–30 min	Catecholamine crisis; tachyarrhythmia
Enalaprilat	1.25 mg IV over 5 min every 6 h (max 5 mg)	15–30 min / 6–12 h	Unpredictable; avoid in pregnancy, AKI
Urapidil (where available)	12.5–25 mg IV bolus, then 5–40 mg/h	3–5 min / 4–6 h	Used in Europe
Fenoldopam	0.1–1.6 µg/kg/min	5 min / 30 min	Glaucoma

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CAUTION **Do not lower BP too fast.** Chronically hypertensive patients have shifted cerebral and renal autoregulation; abrupt falls can cause stroke, MI, blindness and AKI. Sublingual nifedipine is dangerous and obsolete.

Hypertension in pregnancy — classification

Type	Definition
Chronic hypertension	BP ≥140/90 before pregnancy or before 20 weeks, or persisting >12 weeks postpartum
Gestational hypertension	New BP ≥140/90 after 20 weeks without proteinuria or organ dysfunction
Pre-eclampsia	Gestational HTN + proteinuria (≥300 mg/24 h or PCR ≥0.3) or maternal organ dysfunction (creatinine >1.1, transaminases ×2, platelets <1 lakh, neurological signs, pulmonary oedema) or fetal growth restriction

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Type	Definition
Pre-eclampsia with severe features	BP ≥160/110, or HELLP, eclampsia, pulmonary oedema, persistent headache/visual symptoms
Chronic HTN with superimposed pre-eclampsia	Chronic HTN plus new features of pre-eclampsia

Management of hypertension in pregnancy

- **Treat at ≥140/90** (CHAP trial) to a target of about **<140/90 (DBP ≈85)**; treat ≥160/110 urgently (within 30–60 min).
- Stop ACEi, ARB, MRA, aliskiren, atenolol, SGLT2i and statins before conception or as soon as pregnancy is detected.
- **Aspirin 75–150 mg at night** from 12–16 weeks until 36 weeks in high-risk women (previous pre-eclampsia, chronic HTN, diabetes, CKD, autoimmune disease, multiple pregnancy) — ASPRE trial.
- **Calcium 1–2 g/day** if dietary intake is low (most Indian women) — reduces pre-eclampsia.

Drug (oral)	Dose	Notes
Labetalol	100 mg BD → max 2400 mg/day (in 2–4 doses)	First-line; avoid in asthma
Nifedipine ER / retard	20–30 mg OD–BD → max 120 mg/day	First-line; avoid short-acting capsule
Methyldopa	250 mg BD–TDS → max 3 g/day	Long safety record; sedation, depression — switch postpartum
Hydralazine	25–50 mg TDS–QID	Add-on
Metoprolol	25–100 mg BD	Alternative β-blocker

Severe pre-eclampsia and eclampsia — acute protocol

Component	Regimen
Acute BP control (≥160/110)	**Labetalol IV** 20 mg → 40 mg → 80 mg every 10 min (max 300 mg); or **oral nifedipine** 10 mg (immediate-release, swallowed) repeat after 20–30 min; or **hydralazine IV** 5–10 mg every 20 min (max 30 mg)
Magnesium sulphate — Pritchard (IM) regimen	Loading: 4 g IV (20% solution) over 5–10 min + 10 g IM (5 g deep IM each buttock, 50%); maintenance 5 g IM every 4 h in alternate buttocks for 24 h after delivery/last fit
Magnesium sulphate — Zuspan (IV) regimen	Loading 4 g IV over 15–20 min; maintenance 1 g/h IV infusion for 24 h
Before each $MgSO_4$ dose	Knee jerk present, RR ≥16/min, urine output ≥30 ml/h (100 ml/4 h). Antidote: **calcium gluconate 10% 10 ml IV over 10 min**
Definitive treatment	Delivery (timing by gestation and severity)

Postpartum and breastfeeding

BP often peaks 3–6 days postpartum. Continue treatment and monitor. Compatible with breastfeeding: **enalapril, captopril, nifedipine, amlodipine, labetalol, metoprolol**. Avoid methyldopa (postpartum depression). Women with pre-eclampsia or gestational hypertension have a 2-fold lifelong cardiovascular risk — screen annually for BP, glucose and lipids.

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CHAPTER 10

Diabetes — Definition, Classification and Diagnosis

Diagnostic criteria, types of diabetes and who to screen

Definition

Diabetes mellitus is a group of metabolic disorders characterised by **chronic hyperglycaemia** resulting from defects in insulin secretion, insulin action or both, causing microvascular (retina, kidney, nerve) and macrovascular (coronary, cerebral, peripheral) complications. The diagnostic thresholds are the glucose levels above which the risk of diabetic retinopathy rises sharply.

Diagnostic criteria (ADA 2026 / WHO)

Test	Normal	Prediabetes	Diabetes
Fasting plasma glucose (≥8 h fast)	<100 mg/dl	100–125 mg/dl (IFG; WHO: 110–125)	≥126 mg/dl
2-h plasma glucose after 75 g OGTT	<140 mg/dl	140–199 mg/dl (IGT)	≥200 mg/dl
HbA1c (NGSP-certified, standardised lab)	<5.7%	5.7–6.4% (WHO/IEC: 6.0–6.4%)	≥6.5%
Random plasma glucose	—	—	≥200 mg/dl **with** classic symptoms or hyperglycaemic crisis

GUIDELINE In the absence of unequivocal hyperglycaemia, diagnosis needs **two abnormal results** — either the same test on two occasions or two different tests from the same sample. Capillary glucometer readings are not diagnostic.

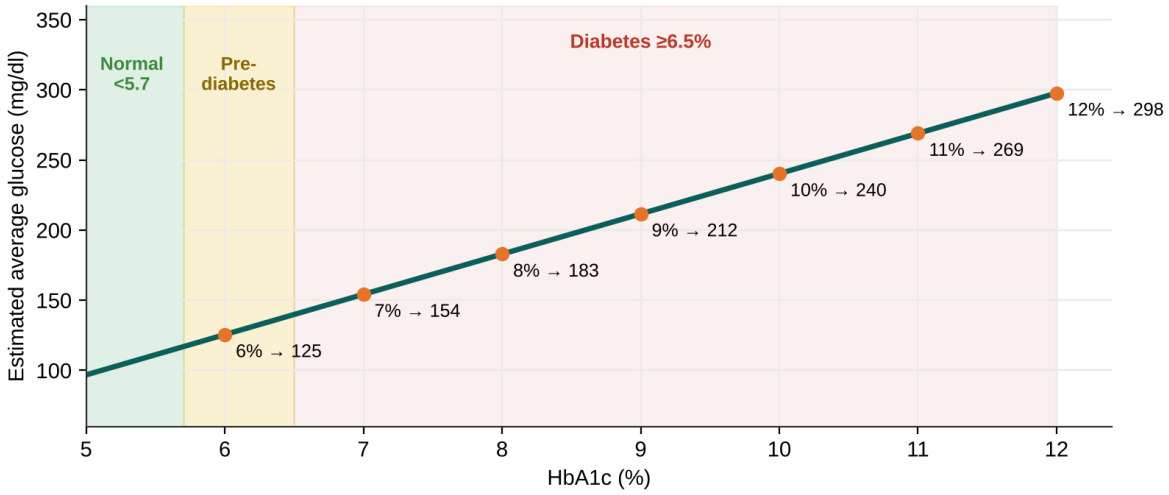


Figure 10.1. Relation between HbA1c and estimated average glucose (eAG = 28.7 × HbA1c − 46.7; ADAG study).

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When HbA1c misleads

Falsely LOW HbA1c	Falsely HIGH HbA1c
Haemolysis, blood loss, recent transfusion	Iron, B12 or folate deficiency anaemia (common in India)
Pregnancy (2nd–3rd trimester)	Splenectomy, aplastic anaemia
Treatment of iron deficiency, erythropoietin	Uraemia (carbamylated Hb), alcoholism, high-dose aspirin
CKD stage 4–5 / dialysis, liver cirrhosis	Some haemoglobin variants (assay-dependent)
HbS, HbC, HbE, thalassaemia (assay-dependent), HIV (on ART)	Hypertriglyceridaemia, hyperbilirubinaemia (some assays)

In these situations use plasma glucose, OGTT, fructosamine or CGM metrics (GMI).

Classification of diabetes

Type	Mechanism	Typical features
Type 1 diabetes	Autoimmune β-cell destruction (rarely idiopathic) → absolute insulin deficiency	Any age (peak childhood), lean, ketosis-prone; GAD65, IA-2, ZnT8, insulin autoantibodies; low C-peptide
Type 2 diabetes (90–95%)	Insulin resistance + progressive β-cell dysfunction	Adults (increasingly young Indians), overweight/central obesity, family history, acanthosis; metabolic syndrome
LADA (latent autoimmune diabetes in adults)	Slowly progressive autoimmune diabetes	Age >30, initially non-insulin-requiring, GAD-positive; insulin needed within months–years
MODY (monogenic)	Single-gene defects of β-cell (HNF1A, HNF4A, GCK, HNF1B…)	Onset <25–35 y, autosomal dominant, antibody-negative, C-peptide preserved
Neonatal diabetes	KCNJ11, ABCC8 etc.	Onset <6 months — genetic testing; many respond to sulfonylureas
Pancreatogenic (type 3c)	Chronic pancreatitis, **fibrocalculous pancreatic diabetes (FCPD)**, pancreatectomy, cystic fibrosis, haemochromatosis, cancer	Steatorrhoea, calcification on X-ray/CT; brittle; low glucagon → hypoglycaemia-prone
Drug- or chemical-induced	Glucocorticoids, antipsychotics, thiazides, calcineurin/mTOR inhibitors, checkpoint inhibitors, PI3K inhibitors, protease inhibitors	Post-transplant diabetes (PTDM); immune-checkpoint diabetes may present in DKA
Endocrinopathies	Cushing's, acromegaly, phaeochromocytoma, glucagonoma, hyperthyroidism	Features of the primary disease
Gestational diabetes (GDM)	Diabetes first diagnosed in 2nd/3rd trimester not clearly overt	See Chapter 21

INDIA NOTE **Fibrocalculous pancreatic diabetes** — a form of tropical chronic pancreatitis first described from Kerala — presents in young, lean people with abdominal pain, steatorrhoea and pancreatic calculi. It needs insulin and pancreatic enzyme replacement, rarely causes ketosis, and carries a risk of pancreatic cancer.

Staging of type 1 diabetes

Stage	Autoantibodies	Glucose	Symptoms	Action
Stage 1	≥2 positive	Normal	None	Monitor; trials/teplizumab programmes

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Stage	Autoantibodies	Glucose	Symptoms	Action
Stage 2	≥2 positive	Dysglycaemia	None	Teplizumab (FDA) can delay stage 3 by ≈2–3 years
Stage 3	Positive	Diabetes	Present	Insulin

Distinguishing the common types

Feature	Type 1	Type 2	LADA	MODY
Age of onset	Usually <30	Usually >30 (Indians often 25–40)	>30	<25–35
Body habitus	Lean	Overweight/central obesity	Lean/normal	Normal
Ketosis	Common	Rare (stress)	Late	Rare
C-peptide	Low/absent	Normal/high	Low–normal, declining	Normal
Autoantibodies	Positive	Negative	GAD positive	Negative
Family history	Uncommon	Common	Variable	Three generations (autosomal dominant)
Treatment	Insulin	Lifestyle + drugs	Insulin early; avoid SU	HNF1A/4A: low-dose SU; GCK: none

Who should be screened?

Group	Recommendation
All adults	Begin at age **35** (ADA); in India consider from **30** (NP-NCD screens everyone ≥30 annually with random capillary glucose)
Overweight adults (BMI ≥23 kg/m² in Asians) at any age with ≥1 risk factor	First-degree relative with diabetes, hypertension, dyslipidaemia, CVD, PCOS, physical inactivity, acanthosis nigricans, high-risk ethnicity (South Asian)
Prediabetes	Yearly
Previous GDM	At least every 1–3 years for life
HIV, chronic pancreatitis, antipsychotic or long-term steroid use	At baseline and periodically
Children/adolescents with overweight + risk factor	From puberty or age 10, every 3 years

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Indian Diabetes Risk Score (IDRS, Mohan 2005)

Parameter	Category	Score
Age (years)	<35 / 35–49 / ≥50	0 / 20 / 30
Waist (cm) — women	<80 / 80–89 / ≥90	0 / 10 / 20
Waist (cm) — men	<90 / 90–99 / ≥100	0 / 10 / 20
Physical activity	Vigorous exercise or strenuous work / moderate / mild / sedentary	0 / 10 / 20 / 30
Family history	None / one parent / both parents	0 / 10 / 20

Interpretation: <30 low risk; 30–50 moderate; **≥60 high risk — perform OGTT or HbA1c.**

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Newer data-driven subgroups

Cluster analyses (Ahlqvist, Sweden; validated in Indian cohorts by Anjana and colleagues) describe subgroups such as severe insulin-deficient (SIDD), severe insulin-resistant (SIRD), mild obesity-related (MOD) and mild age-related (MARD) diabetes. Indian patients show more insulin-deficient and insulin-resistant obese phenotypes. These concepts explain why ‘type 2 diabetes’ behaves differently between patients, though they do not yet change routine treatment.

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CHAPTER 11

Initial Evaluation, Investigations and Targets in Diabetes

The first visit, baseline tests, advanced tests and individualised goals

The comprehensive first visit

Domain	What to assess
History	Symptoms (polyuria, weight loss, infections), onset, family history, previous GDM, diet, activity, sleep, tobacco, alcohol, drugs (steroids, antipsychotics), hypoglycaemia, DKA admissions
Complications	Visual symptoms, numbness/burning feet, foot ulcers, chest pain, claudication, erectile dysfunction, gastroparesis, urinary symptoms
Comorbidities	Hypertension, dyslipidaemia, obesity, MASLD, OSA, thyroid disease, depression, TB, dental disease
Examination	BMI, waist, BP (lying/standing), pulse, acanthosis, thyroid, injection sites, **feet** (monofilament, vibration, pulses, deformities), oral cavity, fundus
Psychosocial	Diabetes distress, depression (PHQ-9), cost and access, literacy, family support

Basic investigations

Test	Purpose / interpretation	Frequency
HbA1c	Glycaemic control over 3 months	Every 3 months if above target or therapy changed; 6-monthly if stable
Fasting and 2-h post-meal plasma glucose	Pattern of hyperglycaemia; when HbA1c unreliable	Each visit
Lipid profile	LDL-C, non-HDL-C, TG	At diagnosis, then yearly
Serum creatinine + eGFR	CKD staging, drug dosing (metformin, SGLT2i, DPP-4i)	Yearly (more often if CKD)
Urine albumin–creatinine ratio (UACR, spot)	≥30 mg/g = albuminuria; confirm 2 of 3 samples in 3–6 months	Yearly
Liver enzymes (ALT, AST), platelets → FIB-4	MASLD and fibrosis risk	At diagnosis, then 1–2 yearly
Urine routine/microscopy	Infection, haematuria, ketones	At diagnosis
CBC	Anaemia (affects HbA1c)	At diagnosis
Serum potassium	On ACEi/ARB, MRA, diuretics	Periodically
Vitamin B12	Long-term metformin (neuropathy, anaemia)	Periodically (every 1–2 years)
TSH	Type 1 diabetes (autoimmune thyroid), women, dyslipidaemia	At diagnosis; T1D every 1–2 years
ECG	Silent ischaemia, LVH, QT	At diagnosis, then yearly in adults >40
Dilated fundus examination / retinal photography	Retinopathy	T2D at diagnosis; T1D within 5 years; then yearly (2-yearly if normal and well controlled)

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Advanced and selective investigations

Test	When to order
Fasting C-peptide (with simultaneous glucose)	Doubt about type; lean young patient; long-standing T2D to decide on insulin; >0.6 ng/ml suggests reserve
Islet autoantibodies (GAD65, IA-2, ZnT8, IAA)	Suspected T1D or LADA (lean, ketosis, rapid progression, poor response to oral drugs)
Genetic testing (MODY panel)	Onset <35, antibody-negative, family history over generations, mild stable fasting hyperglycaemia (GCK)
Continuous glucose monitoring (CGM)	Insulin users, hypoglycaemia, high variability, pregnancy
Fructosamine	When HbA1c unreliable (anaemia, haemoglobinopathy, pregnancy)
Ankle–brachial index / arterial Doppler	Absent pulses, claudication, age >50, diabetes >10 years
Nerve conduction studies, autonomic tests	Atypical neuropathy; suspected autonomic neuropathy
Echocardiography, NT-proBNP	Suspected heart failure (ADA recommends NT-proBNP screening for stage B HF)
Stress test / CT coronary angiography, CAC	Typical/atypical symptoms or abnormal ECG — not routine screening in asymptomatic
Transient elastography (FibroScan)	FIB-4 ≥1.3 — liver stiffness for advanced fibrosis
Ultrasound abdomen, faecal elastase, CT pancreas	Suspected pancreatogenic diabetes

Individualised targets (ADA 2026)

Parameter	General target	More stringent	Less stringent
HbA1c	<7.0%	<6.5% (young, short duration, no CVD, no hypoglycaemia, on low-risk drugs)	<8.0% (frail, limited life expectancy, severe hypoglycaemia history, advanced complications)
Pre-meal capillary glucose	80–130 mg/dl	—	90–150 mg/dl
Peak post-meal (1–2 h)	<180 mg/dl	<140 mg/dl	—
Bedtime	90–150 mg/dl	—	100–180 mg/dl
CGM: time in range 70–180	>70%	—	>50% (older/high-risk)
CGM: time below 70 / below 54	<4% / <1%	—	<1% below 70
Blood pressure	<130/80	SBP <120 in high CV/renal risk	<140/90 in poor health
LDL-C	<100 (no ASCVD, age 40–75); <70 if 1+ risk factor	<55 mg/dl with ASCVD	Statin per life expectancy
Weight	5–7% loss (prediabetes); ≥5–15% in T2D with obesity	≥15% for remission	Avoid weight loss in frail

Factors that decide how tight the target should be

Factor	Tighter (towards 6.5%)	Looser (towards 8%)
Hypoglycaemia risk / drug-related harm	Low	High
Disease duration	Newly diagnosed	Long-standing

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Factor	Tighter (towards 6.5%)	Looser (towards 8%)
Life expectancy	Long	Short
Comorbidities and complications	Absent / few	Severe
Patient preference, resources, support	Motivated, good support	Limited

Schedule of care — the diabetes checklist

Every visit	Every 3–6 months	Every year
Weight, BMI, waist, BP; glucose log/CGM review; hypoglycaemia; injection sites; adherence; lifestyle goal; tobacco	HbA1c; medication review and intensification	Lipids, creatinine/eGFR, UACR, ALT/FIB-4, B12 (metformin), dilated eye exam, comprehensive foot exam, ECG, dental check, vaccinations (influenza, pneumococcal, hepatitis B, zoster), depression/distress screen

CLINICAL PEARL A well-run diabetes visit is a **checklist**, not a glucose discussion. Use a printed or electronic flow-sheet — it is the simplest proven way to improve outcomes in busy clinics.

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CHAPTER 12

Lifestyle and Medical Nutrition Therapy in Diabetes

Eating, moving, fasting and remission — practical Indian guidance

Goals of medical nutrition therapy (MNT)

- Achieve glucose, BP, lipid and weight targets without hypoglycaemia.
- Match carbohydrate to insulin and activity; maintain the pleasure of eating and cultural food habits.
- Ensure adequate protein (≥0.8 g/kg/day; more in older adults and during weight loss) and micronutrients.

Macronutrients — what the evidence says

Nutrient	Recommendation	Practical Indian translation
Carbohydrate	No ideal %; reduce refined carbohydrate; emphasise high-fibre, low-GI sources. ICMR–INDIAB data suggest lowering carbohydrate to ≈50–55% of energy with more protein	Smaller rice/chapati portion; millets, brown rice, whole wheat; avoid maida, white bread, sugar
Protein	15–20% of energy (1.0–1.2 g/kg during weight loss); limit to 0.8 g/kg in non-dialysis CKD	Dal, legumes, curd, paneer, egg, fish, chicken, soy
Fat	Quality > quantity; <7–10% saturated; avoid trans fat; ↑ MUFA/PUFA	Rotate oils; nuts; limit ghee, butter, coconut oil, fried snacks
Fibre	≥14 g/1000 kcal (25–40 g/day)	Whole grains, vegetables, legumes, fenugreek (methi) seeds, guar gum, oats
Sodium	<2.3 g/day (<1.5 g if hypertensive)	Avoid pickles, papad, namkeen
Sweeteners	Non-nutritive sweeteners acceptable short-term as a bridge; water is best	Avoid sugar, jaggery, honey (all raise glucose equally)

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Glycaemic index (GI) of common Indian foods

Low GI (≤55)	Medium GI (56–69)	High GI (≥70)
Most dals and legumes (rajma, chana, moong), milk, curd, apple, guava, orange, barley, oats (steel-cut), nuts	Whole-wheat chapati, brown rice, basmati rice, ragi (in some forms), banana, papaya, mango	White (polished) rice, idli, dosa, upma (rava), white bread, cornflakes, potato, watermelon, glucose, sugar

CLINICAL PEARL GI is modified by the meal. Eating **vegetables and protein first and carbohydrate last**, cooling and reheating rice (resistant starch), adding dal and curd, and a short walk after meals all lower the post-meal glucose rise.

Carbohydrate counting for insulin users

- One carbohydrate exchange = **15 g carbohydrate** ≈ 1 small chapati (30 g atta), ⅓ cup cooked rice, 1 medium idli, 1 small fruit, 1 cup milk.
- **Insulin-to-carbohydrate ratio (ICR)** = 500 ÷ total daily insulin dose (e.g. TDD 50 U → 1 U per 10 g carbohydrate).

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- **Insulin sensitivity (correction) factor (ISF)** = 1800 ÷ TDD for rapid-acting analogues (1500 for regular insulin) = mg/dl fall per unit.
- Fixed-carbohydrate meal plans suit patients on premixed insulin or fixed prandial doses.

Physical activity in diabetes

Situation	Advice
General	150 min/week moderate aerobic + resistance 2–3 days/week; break sitting every 30 min; walk 10–15 min after meals
On insulin/SU — before exercise	Check glucose: <90 mg/dl → take 15–30 g carbohydrate first; 90–250 → exercise; >250 with ketones → avoid vigorous exercise
Insulin adjustment	Reduce pre-exercise bolus by 25–50% for planned activity; evening snack or basal reduction 10–20% after prolonged exercise (delayed hypoglycaemia)
Proliferative retinopathy	Avoid heavy lifting, Valsalva, high-impact and head-down exercise
Peripheral neuropathy / foot ulcer	Proper footwear, daily foot check; non-weight-bearing exercise (cycling, swimming) if active ulcer
Autonomic neuropathy	Cardiac evaluation before starting; watch for postural hypotension

Weight loss and diabetes remission

Remission is defined (ADA/EASD/Diabetes UK consensus 2021) as **HbA1c <6.5% for at least 3 months after stopping glucose-lowering drugs**. In DiRECT, a low-calorie meal-replacement programme (825–853 kcal/day for 12–20 weeks, then food reintroduction) produced remission in 46% at 1 year; remission correlated with weight loss (86% with ≥15 kg loss). Indian data (DIABETES REMISSION studies, intensive lifestyle programmes) show similar results in people with recent-onset diabetes.

Predictors of remission	Approach
Diabetes duration <6 years, not on insulin, C-peptide preserved, weight loss ≥10–15%	Total diet replacement or structured low-calorie diet with supervision; or GLP-1/GIP-based drugs; or metabolic surgery

CAUTION Continue to check HbA1c every 3–6 months, eyes, feet, kidneys and BP in people in remission — the 'legacy' of past hyperglycaemia persists and diabetes may return.

Fasting — Ramadan, Navaratri, Ekadashi and others

IDF-DAR risk category	Examples	Advice
Very high risk	Recent DKA/severe hypo, T1D with poor control, pregnancy, eGFR <30, advanced complications	Advise against fasting
Moderate–high risk	T2D on insulin/SU with HbA1c 7.5–9%, CKD 3	Fast only with careful plan and SMBG
Low risk	Well-controlled T2D on lifestyle/metformin/DPP-4i/SGLT2i/GLP-1 RA	Usually safe; adjust timing

- **Metformin:** take ⅔ of the dose at the main (evening/iftar) meal.
- **Sulfonylureas:** prefer gliclazide; take at the evening meal; reduce dose if twice daily.
- **Basal insulin:** reduce by 15–30%, give at the evening meal.
- **Premixed insulin:** usual morning dose at the evening meal; reduce the second (pre-dawn) dose by 25–50%.
- **SGLT2 inhibitors:** take with the evening meal, maintain hydration.
- **Break the fast** if glucose <70 mg/dl, or >300 mg/dl, or symptoms of hypoglycaemia or illness.

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Diabetes self-management education and support (DSMES)

Every person with diabetes should receive structured education at diagnosis, annually, when complications arise and at transitions of care: glucose monitoring, insulin technique, hypoglycaemia and sick-day rules, foot care, diet and activity. DSMES lowers HbA1c by ≈0.5–0.6% — as much as an extra drug.

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CHAPTER 13

Non-Insulin Glucose-Lowering Drugs — Class, Drug and Dose

Oral agents, GLP-1 receptor agonists and dual agonists with doses and cautions

Overview of drug classes

Class	HbA1c fall	Weight	Hypo risk	CV benefit	Renal benefit	Cost (India)
Metformin	1.0–1.5%	Neutral/↓	No	Possible	Neutral	Very low
Sulfonylureas	1.0–1.5%	↑	Yes	Neutral	Neutral	Very low
DPP-4 inhibitors	0.5–0.8%	Neutral	No	Neutral	Neutral	Low
SGLT2 inhibitors	0.5–0.8%	↓ (2–3 kg)	No	**HF** ↓↓, MACE ↓ (empa, cana)	↓↓ **CKD progression**	Low (generics)
Pioglitazone	1.0–1.5%	↑	No	Possible (stroke)	Neutral	Very low
α-Glucosidase inhibitors	0.5–0.8%	Neutral	No	Neutral	Neutral	Low
Meglitinides	0.5–1.0%	↑	Yes	Neutral	Neutral	Low
GLP-1 RA	1.0–1.8%	↓↓ (4–15%)	No	**MACE** ↓ (lira, dula, sema)	↓ (sema — FLOW)	Moderate (generic semaglutide)
GIP/GLP-1 RA (tirzepatide)	1.8–2.4%	↓↓↓ (15–22%)	No	MACE non-inferior to dulaglutide (SURPASS-CVOT); HFpEF ↓	Probable	High
Insulin	Unlimited	↑	Yes	Neutral	Neutral	Low (human) / moderate (analogues)

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Biguanide — metformin

Drug	Dose	Renal dosing	Cautions
Metformin IR	500 mg OD–BD with meals → 1000 mg BD (max 2550 mg/day; usual effective 2000)	eGFR ≥45: full dose; 30–44: max 1000 mg/day, do not start; <30: stop	GI upset (start low, with food, use XR); B12 deficiency; lactic acidosis (rare — sepsis, shock, hypoxia, liver failure)
Metformin XR (SR)	500 mg OD with evening meal → 2000 mg OD	As above	Better GI tolerance

CAUTION Withhold metformin on the day of **iodinated contrast** in patients with eGFR <60, liver disease, alcoholism or heart failure (or intra-arterial contrast); restart after 48 h if renal function is stable. Also hold during acute illness with dehydration, shock or hypoxia.

Insulin secretagogues — sulfonylureas and meglitinides

Drug	Dose	Notes
Gliclazide	40–160 mg BD (IR, max 320 mg/day); MR 30–120 mg OD	Lowest hypoglycaemia and CV risk among SUs — preferred
Glimepiride	0.5–4 mg OD before breakfast (max 8 mg; >4 mg adds little)	Widely used in India; once daily
Glipizide	2.5–20 mg OD–BD (ER 5–20 mg OD)	Short-acting; usable in CKD
Glibenclamide (glyburide)	2.5–15 mg/day	**Avoid** in elderly and CKD — prolonged hypoglycaemia
Repaglinide	0.5–4 mg before each meal (max 16 mg/day)	Skip dose if meal skipped; usable in CKD
Nateglinide	60–120 mg before meals	Short action

Cautions: hypoglycaemia (especially elderly, CKD, irregular meals, alcohol, with insulin), weight gain. Reduce or stop SU when adding insulin, GLP-1 RA or tirzepatide if HbA1c is near target.

DPP-4 inhibitors (gliptins)

Drug	Standard dose	eGFR 30–49	eGFR <30 / dialysis
Sitagliptin	100 mg OD	50 mg OD (eGFR 30–44)	25 mg OD
Vildagliptin	50 mg BD	50 mg OD	50 mg OD
Linagliptin	5 mg OD	No adjustment	No adjustment (hepatic excretion)
Saxagliptin	5 mg OD	2.5 mg OD	2.5 mg OD
Teneligliptin (India)	20 mg OD (max 40)	No adjustment	Caution
Alogliptin	25 mg OD	12.5 mg OD	6.25 mg OD

Cautions: pancreatitis (rare), bullous pemphigoid, arthralgia; saxagliptin (and alogliptin) — heart-failure hospitalisation signal. Do not combine with GLP-1 RA (no additive benefit).

SGLT2 inhibitors (gliflozins)

Drug	Dose	eGFR for initiation	Evidence
Empagliflozin	10 mg OD (25 mg for extra glucose lowering)	≥20 (cardiorenal); glucose effect falls below 45	EMPA-REG, EMPEROR-Reduced/Preserved, EMPA-KIDNEY
Dapagliflozin	10 mg OD (5 mg start for glycaemia in some)	≥25 (≥20 per guidelines)	DECLARE, DAPA-HF, DELIVER, DAPA-CKD
Canagliflozin	100 mg OD (300 mg if eGFR ≥60)	≥30	CANVAS, CREDENCE
Ertugliflozin	5–15 mg OD	≥45 for glycaemia	VERTIS-CV (neutral MACE)
Remogliflozin (India)	100 mg BD	≥45	Glycaemic data only
Sotagliflozin (SGLT1/2)	200–400 mg OD	≥25	SCORED, SOLOIST (HF)

SGLT2i caution	Prevention / action
Euglycaemic DKA (glucose may be <250 mg/dl)	Hold during acute illness, fasting, surgery (3 days before; 4 days for ertugliflozin), very-low-carb diets; check ketones if unwell; avoid in T1D
Genital mycotic infection	Genital hygiene; topical antifungal; more common in women and uncircumcised men

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SGLT2i caution	Prevention / action
Volume depletion / hypotension	Reduce diuretic dose; caution in elderly
Initial eGFR dip (3–5 ml/min)	Expected and haemodynamic — do not stop
Fournier's gangrene (rare)	Urgent assessment of perineal pain or swelling
Amputation (canagliflozin signal)	Caution in active foot ulcer/PAD

Thiazolidinedione — pioglitazone

Dose: 7.5–15 mg OD → max 45 mg OD. Effective insulin sensitiser; durable effect; **improves MASH/fibrosis** and reduces recurrent stroke (IRIS). **Cautions:** weight gain, oedema, **contraindicated in heart failure (NYHA III–IV)**, fractures (women), macular oedema; avoid in active bladder cancer. Low doses (7.5–15 mg) give most benefit with fewer side effects.

α-Glucosidase inhibitors and other oral agents

Drug	Dose	Notes
Acarbose	25 mg TDS with first bite → 50–100 mg TDS	Flatulence; useful for high-carb Indian diets; treat hypoglycaemia with glucose (not sucrose)
Voglibose	0.2–0.3 mg TDS before meals	Commonly used in India
Miglitol	25–100 mg TDS	—
Saroglitazar (India)	4 mg OD	Dual PPAR-α/γ agonist; diabetic dyslipidaemia (↓TG) and approved for MASLD/MASH in India
Imeglimin	1000 mg BD	Mitochondrial modulator (Japan, India); limited long-term data
Bromocriptine QR	0.8–4.8 mg in the morning	Modest effect; nausea
Colesevelam	3.75 g/day	Lowers LDL-C and HbA1c modestly

GLP-1 receptor agonists and GIP/GLP-1 dual agonist

Drug	Start	Escalation (≥4 weeks per step unless stated)	Maintenance / max
Liraglutide (SC, daily)	0.6 mg OD × 1 week	→ 1.2 mg	1.2–1.8 mg OD (3.0 mg for obesity)
Dulaglutide (SC, weekly)	0.75–1.5 mg weekly	→ 3 mg → 4.5 mg	1.5–4.5 mg weekly
Semaglutide (SC, weekly)	0.25 mg weekly × 4 weeks	→ 0.5 mg → 1 mg → 2 mg	0.5–2 mg weekly (2.4 mg for obesity)
Oral semaglutide (daily)	3 mg OD × 30 days	→ 7 mg → 14 mg	7–14 mg OD; take on waking with ≤120 ml water, 30 min before food/drugs
Exenatide (SC)	5 µg BD × 1 month	→ 10 µg BD	10 µg BD (ER 2 mg weekly)
Lixisenatide (SC)	10 µg OD × 14 days	→ 20 µg	20 µg OD
Tirzepatide (SC, weekly; GIP/GLP-1)	2.5 mg weekly × 4 weeks	+2.5 mg every 4 weeks	5–15 mg weekly

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INDIA NOTE Generic **semaglutide** became available in India after patent expiry in March 2026, substantially lowering cost; **tirzepatide** has been marketed in India since 2025. **Orforglipron** (oral non-peptide GLP-1 RA, FDA-approved for obesity April 2026) and **oral semaglutide 25 mg** for obesity are newer options; check local availability.

GLP-1 RA / tirzepatide caution	Advice
Nausea, vomiting, diarrhoea, constipation (20–40%)	Slow titration; small meals; stop eating when full; avoid fatty food; may hold escalation
Personal/family history of medullary thyroid carcinoma or MEN2	Contraindicated
Pancreatitis (history)	Avoid; stop if suspected
Gallbladder disease	Cholelithiasis risk with rapid weight loss
Diabetic retinopathy	Rapid HbA1c fall may transiently worsen retinopathy (semaglutide) — ensure eye exam
Peri-procedural aspiration	Inform anaesthetist; liquid diet 24 h before in high-risk; consider holding (see Chapter 17)
Hypoglycaemia	Only with insulin/SU — reduce their dose
Muscle loss	Ensure protein intake and resistance exercise
Pregnancy	Stop at least 2 months before planned conception (semaglutide)

Drugs with proven cardiovascular, renal and liver benefit

Outcome	Drugs with proven benefit
Major adverse CV events (ASCVD)	GLP-1 RA: liraglutide (LEADER), semaglutide SC (SUSTAIN-6, SELECT, SOUL — oral), dulaglutide (REWIND); SGLT2i: empagliflozin, canagliflozin; pioglitazone (stroke)
Heart failure hospitalisation	SGLT2i (all); tirzepatide and semaglutide in obese HFpEF (SUMMIT, STEP-HFpEF); finerenone (FINEARTS-HF)
CKD progression	SGLT2i (CREDENCE, DAPA-CKD, EMPA-KIDNEY); semaglutide (FLOW); finerenone (FIDELIO/FIGARO)
MASH / liver fibrosis	Pioglitazone, semaglutide (ESSENCE), tirzepatide (SYNERGY-NASH phase 2); saroglitazar (India)

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CHAPTER 14

Step-Up and Combination Therapy in Type 2 Diabetes

The ADA 2026 approach, Indian practice and rational combinations

Two parallel goals

Modern guidelines (ADA Standards of Care 2026, ADA/EASD consensus, RSSDI 2022) treat type 2 diabetes with **two parallel goals**: (1) **reduce cardiovascular and kidney risk** using agents with proven organ protection, **independent of HbA1c**; and (2) **achieve and maintain glycaemic and weight goals**. Metformin is no longer mandatory before an SGLT2 inhibitor or GLP-1 RA when there is a compelling indication.

Choosing glucose-lowering therapy in type 2 diabetes (ADA 2026)

At diagnosis — everyone

Lifestyle + DSMES. Assess ASCVD, HF, CKD, MASLD, obesity, hypoglycaemia risk, cost. Statin and BP control.

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ASCVD or high CV risk

GLP-1 RA (or tirzepatide) and/or **SGLT2i** with proven benefit

Heart failure

SGLT2i; in obese HFpEF add semaglutide or tirzepatide; finerenone (HFmrEF/HFpEF)

CKD (eGFR 20–60 or UACR ≥30 mg/g)

SGLT2i + max RAS blocker; **GLP-1 RA** (semaglutide); **finerenone** if albuminuria

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Glycaemia — very high efficacy

Semaglutide, tirzepatide, insulin, combinations

Glycaemia — high efficacy

Metformin, SGLT2i, dulaglutide, liraglutide, pioglitazone, sulfonylurea

Weight — very high efficacy

Semaglutide, tirzepatide; consider metabolic surgery

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Intensify every 3 months if not at target

Add an agent from a different class; avoid clinical inertia. If HbA1c >10% or glucose ≥300 mg/dl, catabolic symptoms or ketosis → **start insulin** (± GLP-1 RA).

Practical step-up ladder (cost-conscious Indian practice)

Step	HbA1c at presentation / situation	Therapy
1	HbA1c <7.5%, no complications	Lifestyle + metformin (XR 500 → 2000 mg)
2	HbA1c 7.5–9% or ≥1.5% above target	**Early dual combination**: metformin + SGLT2i, or + DPP-4i, or + SU (gliclazide/glimepiride); GLP-1 RA if obese/ASCVD (VERIFY trial: early combination more durable)
3	Not at target after 3 months on two drugs	Triple therapy: metformin + SGLT2i + DPP-4i (or SU, or pioglitazone); or replace DPP-4i with GLP-1 RA/tirzepatide
4	Not at target on three oral agents	Add **basal insulin** or GLP-1 RA/tirzepatide (if not already); continue metformin ± SGLT2i; reduce/stop SU when insulin is intensified
5	Not at target on basal insulin (HbA1c above target with FPG at target)	Add GLP-1 RA (or fixed-ratio combination) → basal-plus → basal-bolus or premix (Chapter 16)

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Step	HbA1c at presentation / situation	Therapy
Any time	HbA1c >10%, glucose ≥300, symptoms, weight loss, ketosis	Insulin (basal or basal-bolus) ± metformin; reassess and de-escalate once glucotoxicity resolves

Rational and irrational combinations

Combination	Verdict	Reason
Metformin + any other class	Rational	Complementary mechanisms
SGLT2i + GLP-1 RA	Rational — highly effective	Additive cardiorenal and weight benefits
SGLT2i + DPP-4i	Rational	DPP-4i offsets SGLT2i-induced glucagon rise; FDCs available
Basal insulin + GLP-1 RA	Rational	Less weight gain and hypoglycaemia than basal-bolus; FRCs (IDegLira, iGlarLixi)
Pioglitazone + SGLT2i	Rational	SGLT2i offsets pioglitazone fluid retention
DPP-4i + GLP-1 RA / tirzepatide	**Irrational**	Same pathway — no added benefit
Two sulfonylureas, or SU + meglitinide	**Irrational**	Same mechanism; hypoglycaemia
SU + prandial insulin (basal-bolus)	Avoid	Hypoglycaemia — stop SU
Pioglitazone + insulin in HF	Avoid	Fluid retention

Common fixed-dose combinations in India

Combination	Examples (mg)
Metformin + glimepiride	500/1, 500/2, 1000/1, 1000/2 (SR)
Metformin + gliclazide	500/80, 500/40 (SR 500/30 MR)
Metformin + sitagliptin / vildagliptin / teneligliptin / linagliptin	500/50 or 1000/50 BD; 500/50 or 1000/50 BD; 500/20; 500/2.5 BD
Metformin + dapagliflozin / empagliflozin	1000/10 XR OD; 500/12.5 BD (or 1000/10, 1000/25 XR)
Dapagliflozin + sitagliptin; empagliflozin + linagliptin	10/100; 10/5, 25/5
Metformin + glimepiride + voglibose / pioglitazone	500/1/0.2, 500/2/0.3; 500/2/15
Metformin + dapagliflozin + sitagliptin	1000/10/100 (XR)

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CLINICAL PEARL FDCs improve adherence but make dose adjustment difficult. Start with separate drugs when titrating, then switch to an FDC once doses are stable. Always check the renal dose of each component.

When to de-intensify

- Older or frail patients with HbA1c <6.5% on insulin or sulfonylureas — risk of hypoglycaemia outweighs benefit.
- Recurrent hypoglycaemia, weight loss, declining renal function, reduced food intake, cognitive decline.
- Approach: stop SU first; simplify insulin to once-daily basal; switch to agents with low hypoglycaemia risk (DPP-4i, SGLT2i, GLP-1 RA).

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Monitoring response

Parameter	When
HbA1c	3 months after any change
SMBG/CGM pattern	Fasting high → basal problem; post-meal high → prandial problem; nocturnal low → reduce basal/SU
Weight, BP, creatinine/eGFR, K^+	After SGLT2i, RAS blockers, finerenone
GI tolerance	During GLP-1 RA/tirzepatide titration

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CHAPTER 15

Insulin — Types, Profiles and the Complete Insulin Chart

Every insulin you will prescribe — onset, peak, duration, brands, devices and storage

Insulins are classified by their **time-action profile**: rapid-acting and short-acting (prandial/bolus), intermediate- and long-acting (basal), premixed and co-formulated combinations, and fixed-ratio combinations with GLP-1 RAs. Human insulins are cheaper; analogues give more predictable profiles and less hypoglycaemia.

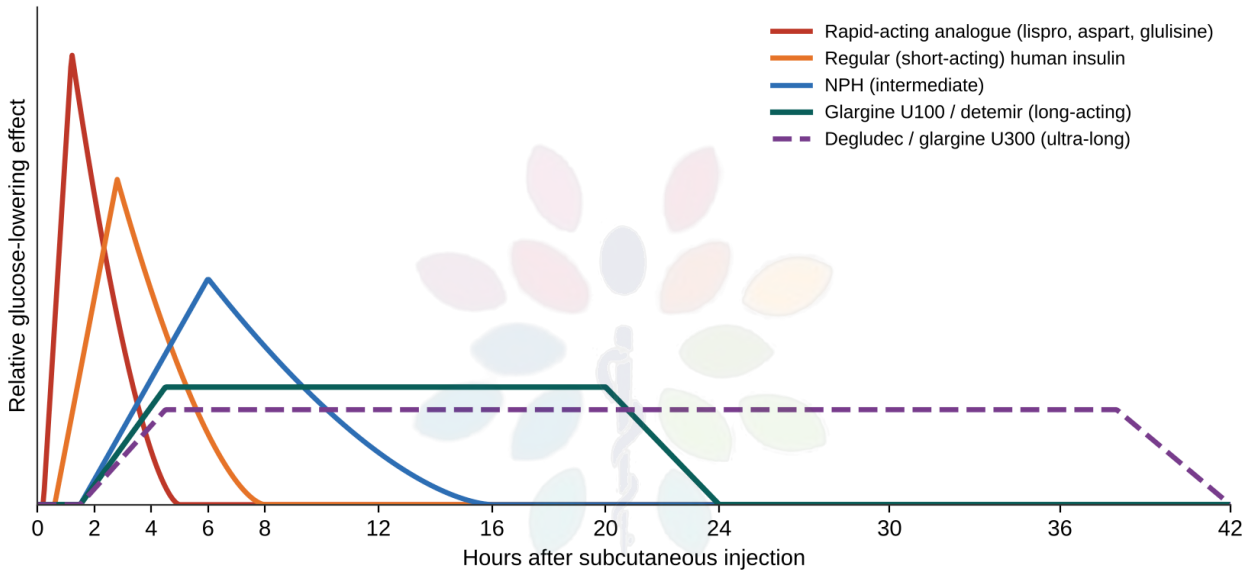


Figure 15.1. Schematic time-action profiles of subcutaneous insulins. Actual onset, peak and duration vary with dose, site, exercise and between individuals.

The complete insulin chart

Type	Insulin (examples of brands in India)	Onset	Peak	Duration	When to inject
RAPID-ACTING ANALOGUES (BOLUS)					
Ultra-rapid	Faster aspart (Fiasp); ultra-rapid lispro (Lyumjev)	2–5 min	1–2 h	3–5 h	0–2 min before meal (up to 20 min after)
Rapid	Insulin lispro (Humalog, biosimilars)	10–15 min	1–2 h	3–5 h	0–15 min before meal
Rapid	Insulin aspart (NovoRapid, biosimilars)	10–15 min	1–2 h	3–5 h	0–15 min before meal
Rapid	Insulin glulisine (Apidra)	10–15 min	1–1.5 h	3–4 h	0–15 min before meal
Inhaled	Technosphere insulin (Afrezza — not in India)	10–15 min	30–60 min	2–3 h	At start of meal
SHORT-ACTING (BOLUS)					
Regular human	Actrapid, Huminsulin R, Insugen R, Wosulin R	30–60 min	2–4 h	6–8 h	30 min before meal; IV use

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Type	Insulin (examples of brands in India)	Onset	Peak	Duration	When to inject
INTERMEDIATE-ACTING (BASAL)					
NPH (isophane)	Insulatard, Huminsulin N, Insugen N, Wosulin N	1–2 h	4–8 h	12–16 h	Bedtime or twice daily; cloudy — roll 10–20 times
LONG-ACTING ANALOGUES (BASAL)					
Glargine U100	Lantus; biosimilars Basalog, Glaritus, Basaglar, others	1–2 h	Minimal	≈24 h	Once daily, same time
Detemir	Levemir (being withdrawn in many markets)	1–2 h	6–8 h (mild)	12–20 h	Once or twice daily
ULTRA-LONG-ACTING ANALOGUES (BASAL)					
Glargine U300	Toujeo	1–6 h	Flat	≈30–36 h	Once daily (±3 h flexibility)
Degludec U100/U200	Tresiba	1 h	Flat	>42 h	Once daily, any time (≥8 h between doses)
Icodec (weekly)	Awiqli (launched in India July 2026; 700 U/ml)	—	Flat	≈1 week (t½ ≈196 h)	Once weekly, same day
PREMIXED HUMAN INSULINS					
Regular/NPH 30/70	Mixtard 30, Huminsulin 30/70, Insugen 30/70	30–60 min	Dual	12–16 h	30 min before breakfast and dinner
Regular/NPH 50/50	Huminsulin 50/50, Mixtard 50	30–60 min	Dual	12–16 h	30 min before meals
PREMIXED ANALOGUES					
Biphasic aspart 30/70	NovoMix 30	10–20 min	Dual	≈24 h	0–10 min before breakfast and dinner (or TDS)
Lispro mix 25/75 and 50/50	Humalog Mix 25, Humalog Mix 50	15 min	Dual	≈24 h	0–15 min before meals
CO-FORMULATION					
Degludec + aspart 70/30 (IDegAsp)	Ryzodeg	10–20 min	Aspart peak	Degludec >24 h	With the largest meal (OD) or two main meals (BD)
FIXED-RATIO BASAL INSULIN + GLP-1 RA					
Degludec + liraglutide	Xultophy (IDegLira) — where available	—	—	24 h	Once daily (dose in ‘dose steps’)
Glargine + lixisenatide	Soliqua (iGlarLixi) — where available	—	—	24 h	Within 1 h before first meal

INDIA NOTE Brand names are examples only — several biosimilars and new brands are marketed in India. Always prescribe by **generic name, concentration (U-40/U-100) and device**.

Concentrations and devices

Concentration	Products	Key safety rule
U-40 (40 U/ml)	Human insulin vials (India)	Use only **U-40 syringes** (red cap). A U-100 syringe with U-40 insulin delivers 60% less

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Concentration	Products	Key safety rule
U-100 (100 U/ml)	Most vials, cartridges, prefilled pens	Use U-100 syringes (orange cap) or pens. U-40 syringe with U-100 insulin gives 2.5× the dose — **dangerous**
U-200	Degludec, lispro pens	Pen dial shows actual units — no conversion
U-300	Glargine (Toujeo)	Pen only; may need 10–18% more units than U100
U-500	Regular insulin (severe insulin resistance)	Specialist use; dedicated syringe/pen
700 U/ml	Icodec (Awiqli) pen	Weekly dosing; pen dials in 10-unit steps

CAUTION Syringe–vial mismatch (U-40 vs U-100) is one of the commonest preventable insulin errors in India. Match the syringe to the vial every time, and never draw insulin from a pen cartridge with a syringe.

Storage and stability

Insulin	Unopened	In-use at room temperature (≤30 °C)
Human regular, NPH, premix (vials/pens)	2–8 °C until expiry	28 days (pens: NPH/premix 14 days for some products)
Rapid analogues (lispro, aspart, glulisine)	2–8 °C	28 days
Glargine U100	2–8 °C	28 days
Glargine U300, degludec	2–8 °C	56 days
Detemir	2–8 °C	42 days
Icodec	2–8 °C	12 weeks

- Never freeze; discard if frozen, clumped, discoloured or cloudy (clear insulins).
- Keep in-use insulin away from direct sunlight and heat; in hot climates use a clay pot/evaporative cooler or insulated pouch.
- Do not keep in the freezer compartment or car dashboard; during travel carry in hand luggage.

Injection technique

Step	Recommendation
Needle	**4 mm pen needle** (or 6 mm syringe) for all adults and children; inject at 90° (pinch up only if very thin or using 6–8 mm)
Sites	Abdomen (fastest absorption, preferred for prandial), thighs (slowest, good for NPH/basal), upper buttocks, back of arms
Rotation	Rotate systematically within a site (quadrants), ≥1 cm from the previous injection; avoid 5 cm around the umbilicus
Hold	Keep needle in for **10 seconds** after pressing the plunger (pens)
Mixing	Roll cloudy insulins (NPH, premix) 10 times and invert 10 times until uniformly milky
Needle re-use	Single use — re-use causes pain, bent tips and lipohypertrophy
Lipohypertrophy	Inspect and palpate sites every visit; injecting into lumps causes erratic absorption and hypoglycaemia when switching to healthy skin (reduce dose by 10–20%)
Sharps disposal	Puncture-proof container

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CHAPTER 16

Insulin Regimens, Initiation and Titration

Starting basal insulin, intensifying, switching and calculating doses

When to start insulin in type 2 diabetes

- HbA1c >10% or glucose ≥300 mg/dl, or symptomatic hyperglycaemia/weight loss (catabolism).
- Not at target despite two or three non-insulin agents (including GLP-1 RA when affordable).
- Ketosis, DKA or HHS; acute illness, surgery, hospitalisation.
- Pregnancy (GDM or pre-existing diabetes not controlled by diet ± metformin).
- Advanced CKD or liver failure where oral drugs are contraindicated; suspected LADA or T1D.

GUIDELINE Before starting insulin, consider a **GLP-1 RA or tirzepatide** — they give similar or greater HbA1c reduction with weight loss and no hypoglycaemia. Insulin is still essential when there is catabolism or ketosis.

Starting basal insulin

Item	Recommendation
Choice	Glargine U100/U300 or degludec (less nocturnal hypoglycaemia) or NPH at bedtime (cheapest)
Starting dose	**10 units/day or 0.1–0.2 U/kg/day** (0.2–0.3 U/kg if HbA1c very high)
Timing	Glargine/degludec — any fixed time daily; NPH — at bedtime (≈10 pm)
Continue	Metformin, SGLT2i, GLP-1 RA, DPP-4i; reduce SU dose (or stop) if hypoglycaemia
Target	Fasting glucose 80–130 mg/dl (individualise)

Basal insulin titration — choose one simple algorithm

Algorithm	Rule
Patient-led '2-every-3'	Increase by **2 units every 3 days** until fasting glucose is in target; if any glucose <70, reduce by 4 units or 10–20%
Daily 1-unit (INSIGHT-style)	Increase by 1 unit every day until fasting <100 mg/dl
Clinician-led (weekly)	Mean fasting ≥180: +8 U; 140–179: +6 U; 120–139: +4 U; 100–119: +2 U (Riddle treat-to-target)

Overbasalisation — stop increasing basal insulin when

- Basal dose exceeds **0.5 U/kg/day**.
- Bedtime-to-morning glucose difference ≥50 mg/dl, or post-meal values high while fasting is at target.
- Hypoglycaemia (especially nocturnal) or high glucose variability.
- → Add prandial coverage or a GLP-1 RA instead of more basal insulin.

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Intensification options

Option	How	Suits
Add GLP-1 RA (or switch to fixed-ratio combination)	Continue basal; reduce basal 10–20% if HbA1c ≤8%	Obese, hypoglycaemia-prone; first choice in ADA
Basal-plus	Add **4 units or 10% of basal** of rapid-acting insulin before the largest meal; titrate by 1–2 U (10–15%) twice weekly to 2-h post-meal <180	Stepwise approach
Basal-bolus	Total daily dose (TDD) ≈ 0.4–0.5 U/kg (T2D) — 50% basal, 50% bolus split across 3 meals	Flexible meals, motivated, T1D
Premixed twice daily	TDD 0.2–0.3 U/kg (or convert basal dose): **2/3 before breakfast, 1/3 before dinner** (or 50:50); titrate each by 1–2 U every 3 days using the next pre-meal reading	Fixed meal pattern, fewer injections, cost
Premixed thrice daily (analogue)	Add a lunch dose (e.g. 3–6 U), reduce breakfast dose accordingly	When twice-daily premix fails
Co-formulation (IDegAsp)	Once with largest meal, or twice daily	Fewer injections

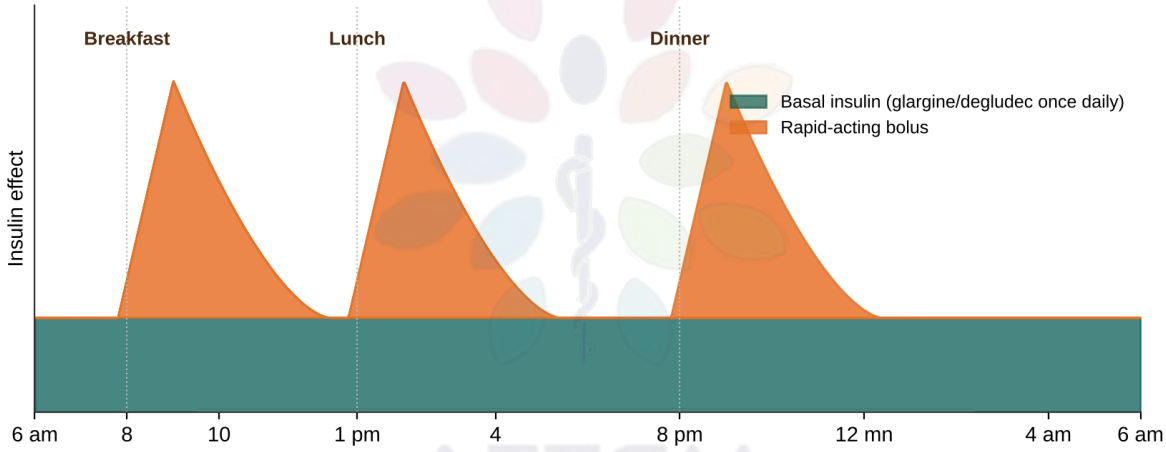


Figure 16.1. Basal-bolus regimen: one daily long-acting basal insulin plus a rapid-acting analogue before each main meal mimics normal physiology.

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Classic split-mixed regimen (NPH + regular)

Still widely used where analogues are unaffordable. **TDD 0.5–0.6 U/kg**; give **2/3 in the morning (2/3 NPH, 1/3 regular)** before breakfast and **1/3 in the evening (1/2 NPH, 1/2 regular)** before dinner. Moving the evening NPH to bedtime reduces nocturnal hypoglycaemia and fasting hyperglycaemia.

Type 1 diabetes — starting doses

Item	Calculation (example: 60 kg adult)
Total daily dose	0.4–0.6 U/kg/day initially (0.5 × 60 = 30 U); usual range 0.5–1.0 U/kg (higher in puberty, DKA recovery)
Basal	40–50% of TDD = 12–15 U glargine/degludec once daily
Bolus	50–60% of TDD split before meals (e.g. 5 U breakfast, 5 U lunch, 5 U dinner), or by carbohydrate counting
Insulin-to-carb ratio	500 ÷ TDD = 500 ÷ 30 ≈ **1 U per 15–17 g carbohydrate**
Correction factor	1800 ÷ TDD = 1800 ÷ 30 = **1 U lowers glucose by ≈60 mg/dl**
Correction dose	(Current glucose − target 120) ÷ correction factor; e.g. glucose 300 → (300 − 120) ÷ 60 = 3 U extra

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CAUTION In type 1 diabetes **basal insulin must never be stopped**, even if not eating — omitting it causes DKA within hours. ADA 2026 recommends offering CGM and automated insulin delivery (AID, 'hybrid closed loop') to all people with T1D and to adults with T2D on insulin where available.

Switching between insulins

From → To	Conversion
NPH once daily → glargine U100 / degludec	1:1 (same units)
NPH twice daily → glargine U100 / degludec once daily	Total NPH dose × 0.8 (reduce by 20%)
Glargine U100 → glargine U300	1:1; expect to need 10–18% more
Glargine U300 → glargine U100 / degludec	Reduce by ≈20%
Detemir/glargine → degludec	1:1 (degludec; detemir BD → total ×1)
Daily basal → icodec weekly	Weekly dose = daily dose × 7; for the **first dose only**, add 50% (T2D) — e.g. 20 U/day → first dose 210 U, then 140 U weekly. Insulin-naive T2D: 70 U weekly
Premix twice daily → basal-bolus	Total premix dose × 0.8 = new TDD; 50% basal, 50% bolus divided over meals
Human regular → rapid analogue	1:1; inject closer to meals
IV insulin infusion → SC	Total SC dose ≈ 60–80% of the previous 24-h IV requirement (when eating); give SC basal **2 h before** stopping the infusion

Interpreting glucose patterns on insulin

Pattern	Likely cause	Action
High fasting, normal rest of day	Insufficient basal; dawn phenomenon; bedtime snack	Increase basal; check 3 am glucose
High fasting with 3 am hypoglycaemia (Somogyi)	Too much evening NPH/premix	Reduce evening dose; move NPH to bedtime; switch to analogue basal
High after a particular meal	Inadequate bolus for that meal	Increase that bolus or reduce carbohydrate
Pre-lunch hypoglycaemia on premix	Morning dose too high / late lunch	Reduce morning dose; mid-morning snack
Erratic glucose	Lipohypertrophy, poor technique, variable meals, gastroparesis, alcohol	Inspect sites, re-educate, CGM

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CHAPTER 17

Inpatient, Perioperative and Sick-Day Diabetes Management

Correction scales, steroid hyperglycaemia, IV insulin, surgery and sick-day rules

Hospital glucose targets (ADA 2026)

Setting	Target	Notes
Most non-critically ill and critically ill patients	**140–180 mg/dl**	Start insulin if persistently ≥180 mg/dl
Selected patients (cardiac surgery, stable, low hypo risk)	110–140 mg/dl	If achievable without hypoglycaemia
Terminally ill / limited life expectancy	<250–300 mg/dl	Avoid symptoms; minimise testing

GUIDELINE **Sliding-scale insulin alone is discouraged** — it treats hyperglycaemia after it occurs and causes swings. Use **basal-plus-correction** (poor oral intake) or **basal-bolus-plus-correction** (eating) regimens. Check HbA1c on admission if not done in the last 3 months.

Weight-based subcutaneous insulin in hospital

Patient type	Starting total daily dose (TDD)
Elderly (>70), eGFR <45, low weight, insulin-naive	0.1–0.25 U/kg/day
Most adults with T2D, eating	0.3–0.5 U/kg/day (50% basal, 50% prandial)
Obese, on steroids, very high glucose	0.5–0.6 U/kg/day or more
Poor oral intake / NPO	Basal 0.1–0.15 U/kg/day + correction only
Already on home insulin	Usually 70–80% of home dose if intake reduced; continue basal

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Correction (supplemental) insulin scale — rapid-acting analogue or regular, before meals

Pre-meal glucose (mg/dl)	Insulin-sensitive (thin, elderly, CKD)	Usual	Insulin-resistant (obese, steroids, infection)
141–180	1 U	2 U	3–4 U
181–220	2 U	3–4 U	5–6 U
221–260	3 U	5–6 U	7–8 U
261–300	4 U	7–8 U	9–10 U
301–350	5 U	9–10 U	11–12 U
>350	6 U + call doctor	11–12 U + call doctor	13–14 U + call doctor

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At bedtime use half the scale (or no correction unless >250 mg/dl). If correction is needed repeatedly, increase the scheduled insulin by the total correction dose given the previous day.

Glucocorticoid-induced hyperglycaemia

Prednisolone given once in the morning raises glucose from **late morning to evening**, with near-normal fasting values. Match it with **morning NPH insulin**, which peaks in the afternoon.

Prednisolone dose (once daily, morning)	Morning NPH (with prednisolone)
10 mg/day	0.1 U/kg
20 mg/day	0.2 U/kg
30 mg/day	0.3 U/kg
≥40 mg/day	0.4 U/kg (max)

- Dexamethasone (long-acting) or divided-dose steroids → basal-bolus or glargine plus prandial insulin.
- Taper insulin as steroids are tapered — watch for hypoglycaemia.
- Non-insulin options for mild hyperglycaemia: SU (gliclazide in the morning), DPP-4i; metformin as prophylaxis in high-dose steroid regimens (ADA 2026).

Intravenous insulin infusion (ICU, perioperative, DKA/HHS excluded)

Prepare **50 units regular insulin in 50 ml 0.9% saline (1 U/ml)**. Check capillary glucose hourly. Provide a glucose source (5–10% dextrose or feed) when the patient is not eating.

Glucose (mg/dl)	Insulin rate (U/h) — standard	Insulin-resistant
<70	Stop; give 25% dextrose 50–100 ml; recheck in 15 min	Same
70–110	0.5	1
111–140	1	2
141–180	1.5–2	3
181–240	2–3	4
241–300	3–4	6
>300	4–6 and review	8 and review

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Perioperative management of glucose-lowering drugs

Drug	Day before surgery	Day of surgery (NPO)
Metformin	Take usual dose	Hold; restart when eating (check eGFR after contrast)
Sulfonylureas, meglitinides	Take usual dose	Hold
DPP-4 inhibitors	Take	May continue
SGLT2 inhibitors	**Stop 3 days before** (ertugliflozin 4 days)	Hold; restart when eating and hydrated
Pioglitazone	Take	Hold

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Drug	Day before surgery	Day of surgery (NPO)
GLP-1 RA / tirzepatide	Multisociety guidance (2024): most may continue; for high GI-risk patients (escalation phase, symptoms) consider holding (weekly: 1 week; daily: day of surgery) and a **24-h clear liquid diet** pre-op	Inform anaesthetist; consider gastric ultrasound or RSI if symptomatic
Basal insulin (glargine/degludec)	**75–80%** of usual dose the evening before (or morning dose)	Continue 75–80% if taken in morning
NPH	Usual evening dose (75% if evening hypoglycaemia risk)	**50%** of morning dose
Premix	Usual evening dose (reduce 20% if NPO later)	Half of morning dose as NPH, or switch to basal + correction / IV infusion
Prandial insulin	Usual	Hold while NPO

- Target perioperative glucose **100–180 mg/dl** (ADA) within 4 h before surgery.
- Major surgery, long fasting or T1D → IV insulin infusion with dextrose (variable-rate intravenous insulin infusion, VRIII).
- Postpone elective surgery if HbA1c is markedly high (>8.5–9%) where safely possible.

Sick-day rules for patients at home

Rule	Detail
Never stop basal insulin	May need **more** insulin during infection; reduce only if glucose <100 and not eating
Monitor	Glucose every 3–4 h; **blood or urine ketones** if glucose >250 mg/dl or vomiting (T1D, SGLT2i users)
Hydrate	100–200 ml fluid every hour; take carbohydrate as liquids (juice, kanji, ORS) if unable to eat
SADMANS — temporarily stop	**S**ulfonylureas, **A**CEi, **D**iuretics, **M**etformin, **A**RBs, **N**SAIDs, **S**GLT2 inhibitors (and GLP-1 RA if vomiting)
Extra rapid-acting insulin	Glucose >250 with ketones: give 10–20% of TDD as rapid-acting every 2–4 h
Seek help	Persistent vomiting, ketones moderate/large (blood β-OHB >1.5–3.0), glucose >300 despite extra insulin, drowsiness, breathlessness, abdominal pain

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CHAPTER 18

Glucose Monitoring and Hypoglycaemia

SMBG, CGM metrics and the recognition and treatment of low glucose

Self-monitoring of blood glucose (SMBG)

Regimen	Suggested SMBG
Lifestyle, metformin, DPP-4i, SGLT2i, GLP-1 RA	Not routinely needed; occasional paired pre/post-meal checks for education
Sulfonylurea	Several times a week and whenever symptoms suggest hypoglycaemia
Basal insulin	Daily fasting glucose during titration; then 3–4 times/week plus occasional pre-dinner
Premixed insulin	Pre-breakfast and pre-dinner daily during titration
Basal-bolus / pump / T1D	4–7 times daily (pre-meal, bedtime, occasionally 3 am, before driving/exercise) — or CGM
Pregnancy	Fasting and 1-h or 2-h post-meal daily

Continuous glucose monitoring (CGM)

CGM sensors measure interstitial glucose every 1–15 minutes for 10–15 days. **Real-time CGM** sends values and alarms continuously; **intermittently scanned CGM (isCGM, e.g. FreeStyle Libre)** shows values when scanned and is widely used in India. ADA 2026 recommends CGM **from diagnosis** for people on insulin and for those on other drugs that cause hypoglycaemia, and it should be considered for anyone in whom it aids management. Interstitial glucose lags blood glucose by ≈5–15 minutes — confirm with a fingerstick when rapidly changing or symptoms don't match.

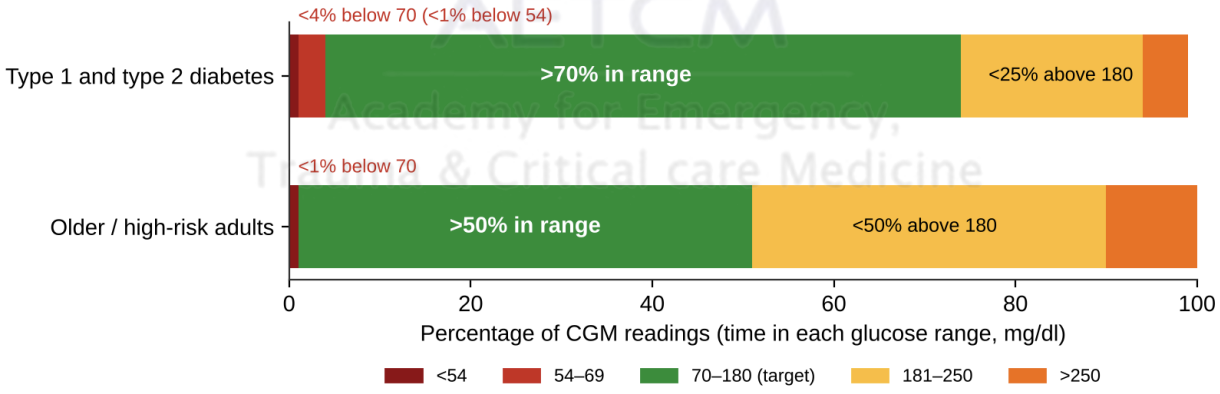


Figure 18.1. International consensus CGM targets (time in ranges). Each 10% increase in time in range ≈ 0.5–0.8% fall in HbA1c.

CGM metric	Target (most adults with T1D/T2D)	Older / high-risk
Time in range (TIR) 70–180 mg/dl	>70% (≈16 h 48 min/day)	>50%
Time below range <70 mg/dl	<4% (<1 h/day)	<1%
Time below range <54 mg/dl	<1% (<15 min/day)	—
Time above range >180 mg/dl	<25%	<50%
Time above range >250 mg/dl	<5%	<10%

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CGM metric	Target (most adults with T1D/T2D)	Older / high-risk
Glucose management indicator (GMI)	<7%	<8%
Coefficient of variation (CV)	≤36%	≤36%
Data sufficiency	≥14 days with ≥70% sensor wear	—

Hypoglycaemia — classification

Level	Definition	Meaning
Level 1	Glucose <70 mg/dl and ≥54 mg/dl	Alert value — treat with carbohydrate; review therapy
Level 2	Glucose <54 mg/dl	Clinically significant — immediate action
Level 3 (severe)	Altered mental or physical status requiring assistance from another person, regardless of glucose value	Medical emergency; review regimen urgently

Symptoms

Autonomic (glucose ≈ 55–70)	Neuroglycopenic (glucose <50–55)
Sweating, tremor, palpitations, anxiety, hunger, pallor, tingling of lips	Confusion, abnormal behaviour, slurred speech, blurred/double vision, drowsiness, seizures, coma, focal deficits (mimic stroke)

Causes and risk factors

- Drugs: **insulin and sulfonylureas** (glibenclamide > glimepiride > gliclazide), meglitinides; alcohol; quinolones, β-blockers (mask symptoms), pentamidine.
- Missed or delayed meals, fasting, unaccustomed exercise, vomiting/diarrhoea.
- **CKD** (reduced insulin and SU clearance), liver failure, adrenal insufficiency, hypothyroidism, gastroparesis.
- Old age, cognitive impairment, long duration, **hypoglycaemia unawareness**, previous severe hypoglycaemia.
- Injection errors (U-40/U-100 mismatch), lipohypertrophy, weight loss without dose reduction.

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Treatment of hypoglycaemia

Conscious and able to swallow — the 'rule of 15'

Give **15–20 g fast-acting glucose**: 3–4 glucose tablets, 150–200 ml fruit juice or regular soft drink, or 3 teaspoons sugar/glucose powder in water. Recheck in **15 minutes**; repeat if still <70 mg/dl.

▼

Once glucose >70 mg/dl

Give a snack or meal with complex carbohydrate and protein (e.g. 2 biscuits, chapati, milk) to prevent recurrence.

▼

Unconscious / unable to swallow (Level 3)

IV dextrose: 25% dextrose 50–100 ml (or 50% 25–50 ml into a large vein, or 10% 150–250 ml), then 10% dextrose infusion. **No IV access: glucagon 1 mg IM/SC** (0.5 mg if <25 kg) or **nasal glucagon 3 mg**. Recovery position.

▼

After recovery

Find the cause; adjust drugs; educate; prescribe glucagon for home if on insulin. **SU-induced hypoglycaemia may recur for 24–72 h** — admit, infuse dextrose, consider **octreotide 50–100 µg SC 6–12-hourly**.

CAUTION Do not use chocolate, ice cream or other fatty foods to treat hypoglycaemia — fat delays glucose absorption. In patients on **acarbose/voglibose**, use pure glucose (dextrose), not table sugar.

Hypoglycaemia unawareness

Repeated hypoglycaemia lowers the glucose threshold at which warning symptoms occur. Management: raise glycaemic targets for 2–3 weeks, **strictly avoid hypoglycaemia** (restores awareness), switch to analogues, CGM with alarms, structured education, and consider AID systems.

Driving and hypoglycaemia

- Check glucose before driving and every 2 hours on long drives; do not drive if <90 mg/dl.
- If hypoglycaemia occurs while driving: stop safely, switch off engine, leave the driver's seat, treat, wait 45 minutes after recovery.

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CHAPTER 19

Hyperglycaemic Emergencies — DKA and HHS

Diagnosis and protocol-based management (2024 international consensus)

Diagnostic criteria (ADA/EASD/JBDS/AACE/DTS consensus 2024)

Feature	Diabetic ketoacidosis (DKA)	Hyperglycaemic hyperosmolar state (HHS)
Glucose	≥200 mg/dl or known diabetes (euglycaemic DKA allowed)	≥600 mg/dl
Ketones	β-hydroxybutyrate **≥3.0 mmol/l** or urine ketones ≥2+	β-OHB <3.0 mmol/l (absent/mild)
Acidosis	pH <7.3 **and/or** bicarbonate <18 mmol/l	pH ≥7.3, bicarbonate ≥15 mmol/l
Osmolality	Variable	Calculated effective osmolality >300 mOsm/kg (or total >320)
Onset	Hours–1 day	Days–weeks
Typical patient	T1D, ketosis-prone T2D, SGLT2i users, checkpoint inhibitor diabetes	Elderly T2D, often undiagnosed; infection, stroke
Mortality	<1–2%	5–20%

DKA severity	pH	Bicarbonate (mmol/l)	Mental status
Mild	7.25–7.30	15–18	Alert
Moderate	7.00–7.24	10–14	Alert/drowsy
Severe	<7.00	<10	Stupor/coma

Mixed DKA–HHS occurs in up to one-third. Useful formulas: **anion gap** = $Na^+ - (Cl^- + HCO_3^-)$ (normal 10–12); **effective osmolality** = $2 \times Na^+$ + glucose/18; **corrected Na^+** = measured $Na^+ + 1.6 \times (\text{glucose} - 100)/100$.

Precipitating factors

Infection (commonest), missed insulin, new-onset T1D, MI, stroke, pancreatitis, alcohol, drugs (SGLT2i, steroids, antipsychotics, checkpoint inhibitors), pregnancy, cocaine, insulin pump failure.

Initial investigations

Capillary and plasma glucose, blood β-OHB (or urine ketones), venous blood gas, electrolytes (Na^+, K^+, Cl^-, HCO_3^-), urea/creatinine, serum osmolality, phosphate, magnesium, CBC, urine routine and culture, blood cultures, ECG, chest X-ray, lipase/amylase, HbA1c, troponin as indicated, pregnancy test.

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DKA management protocol

Adult DKA — first 24 hours

1. Fluids (first priority)

0.9% saline **or balanced crystalloid (Ringer's lactate/Plasma-Lyte — faster resolution)**: **1–1.5 l in the first hour** (15–20 ml/kg), then 250–500 ml/h guided by volume status. Shock → bolus. Cautious in elderly, HF, CKD.

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2. Potassium (before insulin)

K^+ <3.5: hold insulin, give 10–20 mmol/h KCl until ≥3.5. **K^+ 3.5–5.0: add 20–40 mmol KCl per litre** to keep K^+ 4–5. **K^+ >5.0: no K^+**, recheck every 2 h. Monitor ECG.

▼

3. Insulin

Fixed-rate IV regular insulin **0.1 U/kg/h** (optional 0.1 U/kg IV bolus), or 0.14 U/kg/h without bolus. Target glucose fall 50–75 mg/dl/h. Mild–moderate DKA in ward: SC rapid-acting analogue 0.1–0.2 U/kg every 1–2 h is acceptable. **Continue long-acting basal insulin** (or start 0.15–0.3 U/kg SC) from the outset.

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4. Add dextrose

When glucose <250 mg/dl: add **5–10% dextrose** at 150–250 ml/h and continue insulin (0.05 U/kg/h or adjust) until ketoacidosis resolves; keep glucose 150–200 (250 in HHS).

▼

5. Bicarbonate / phosphate

Bicarbonate only if **pH <7.0** (100 mmol in 400 ml water + 20 mmol KCl over 2 h, repeat until pH ≥7.0). Phosphate only if <1.0 mg/dl, cardiac/respiratory compromise or haemolysis.

▼

6. Resolution and transition

Resolved when **β-OHB <0.6 mmol/l and pH ≥7.3 (or HCO_3^- ≥18)**, patient eating. Give SC basal insulin **2 h before** stopping IV insulin; start basal-bolus (0.5–0.6 U/kg/day). Treat the precipitant; educate.

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Monitoring

Parameter	Frequency
Capillary glucose	Hourly
Blood ketones (β-OHB)	Every 1–2 h (should fall ≥0.5 mmol/l/h)
Venous pH, HCO_3^-, K^+	Every 2–4 h
Na^+, urea, creatinine, osmolality	Every 4 h
Fluid balance, GCS, vitals	Hourly

HHS — key differences

- Fluid deficit larger (100–200 ml/kg); replace over 24–48 h. Aim for **osmolality fall of 3–8 mOsm/kg/h**; avoid rapid sodium shifts (≤10 mmol/l per 24 h).
- **Do not start insulin until glucose stops falling with fluids alone** (unless ketonaemia) — then **0.05 U/kg/h**.
- Target glucose 200–300 mg/dl in the first 24 h.

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- High risk of thrombosis — **prophylactic LMWH**; look for MI, stroke, sepsis.
- Many patients can later be managed without insulin.

Complications of treatment

Complication	Prevention / treatment
Hypoglycaemia	Add dextrose when glucose <250; hourly checks
Hypokalaemia (commonest cause of death)	Replace K^+; hold insulin if K^+ <3.5
Cerebral oedema (children, young adults)	Avoid rapid osmolality/glucose fall; headache, bradycardia, confusion → mannitol 0.5–1 g/kg or 3% saline
Hyperchloraemic acidosis	Use balanced crystalloids; self-limiting
Fluid overload / ARDS	Careful fluids in elderly, cardiac and renal patients
Recurrence	Overlap SC basal before stopping IV insulin; continue basal from the start

CAUTION **SGLT2 inhibitor-associated euglycaemic DKA**: the patient is unwell with ketosis and acidosis but glucose may be normal or only mildly raised. Always measure ketones and blood gas in sick patients on SGLT2i; treat with fluids, insulin **plus dextrose from the start**; stop the SGLT2i.

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CHAPTER 20

Chronic Complications of Diabetes — Screening and Management

Retinopathy, kidney disease, neuropathy, the diabetic foot, heart and liver

Screening summary

Complication	Screening test	Start	Frequency
Retinopathy	Dilated fundoscopy or retinal photography (AI-assisted acceptable)	T2D at diagnosis; T1D within 5 years	Yearly (1–2-yearly if no retinopathy and good control); pregnancy each trimester
Kidney disease	UACR + eGFR	T2D at diagnosis; T1D after 5 years	Yearly (1–4 times/year if CKD)
Peripheral neuropathy	10-g monofilament + one of: 128-Hz tuning fork, pinprick, temperature, ankle reflex	T2D at diagnosis; T1D after 5 years	Yearly
Foot	Inspection, pulses, deformity, footwear; ABI if pulses reduced	At diagnosis	Every visit if high risk; yearly otherwise
Autonomic neuropathy	Resting tachycardia, orthostatic BP, gastroparesis, ED symptoms	At diagnosis (T2D)	Yearly
ASCVD / heart failure	Risk factors yearly; ECG; NT-proBNP for stage B HF	At diagnosis	Yearly
MASLD	ALT, FIB-4 → elastography if FIB-4 ≥1.3	At diagnosis	1–2-yearly

Diabetic retinopathy

Stage	Findings	Action
No DR	Normal	Annual screening
Mild NPDR	Microaneurysms only	Annual review; optimise glucose, BP, lipids
Moderate NPDR	Haemorrhages, hard exudates, cotton-wool spots	Ophthalmologist 3–6-monthly
Severe NPDR	4-2-1 rule: haemorrhages in 4 quadrants, venous beading in 2, IRMA in 1	Urgent ophthalmology; consider PRP
Proliferative DR	New vessels, vitreous haemorrhage	PRP laser and/or anti-VEGF; vitrectomy
Diabetic macular oedema	Thickening/exudates near fovea (OCT)	Intravitreal anti-VEGF (first-line), steroids, focal laser

Fenofibrate slows retinopathy progression (FIELD, ACCORD-Eye, LENS). Ensure an eye examination before or soon after starting intensive therapy (rapid HbA1c reduction, semaglutide, insulin) in patients with existing retinopathy.

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Diabetic kidney disease (DKD)

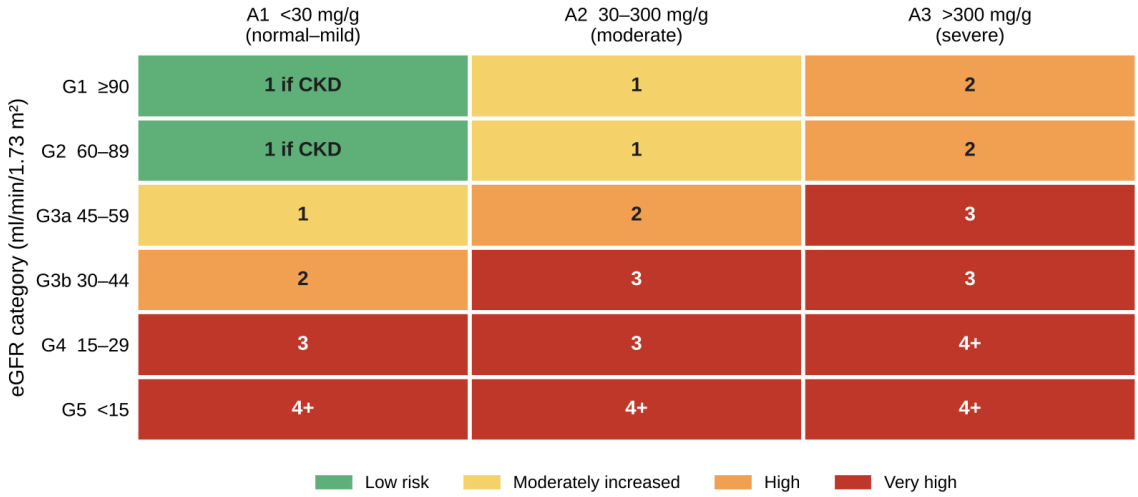


Figure 20.1. KDIGO heat map: CKD prognosis by eGFR and albuminuria category; numbers show recommended monitoring frequency per year.

Pillar	Therapy	Notes
1. RAS blockade	ACEi or ARB titrated to maximum tolerated dose	For hypertension or UACR ≥30 mg/g; check K^+/creatinine in 1–2 weeks
2. SGLT2 inhibitor	Dapagliflozin 10 mg, empagliflozin 10 mg, canagliflozin 100 mg	Start if eGFR ≥20; continue until dialysis/transplant
3. Non-steroidal MRA	**Finerenone** 10 mg (eGFR 25–60) or 20 mg (eGFR ≥60); titrate to 20 mg at 4 weeks if K^+ ≤4.8	For UACR ≥30 mg/g with K^+ ≤5.0; ADA 2026: can start with SGLT2i simultaneously if UACR ≥100 mg/g
4. GLP-1 RA	Semaglutide 1 mg weekly (FLOW)	Reduces kidney failure and CV death
Supportive	BP <130/80 (SBP <120 if tolerated), statin, protein 0.8 g/kg/day, salt <5 g, avoid NSAIDs, smoking cessation	Refer to nephrology if eGFR <30, rapid decline, UACR >300 with uncertainty, or non-diabetic features (haematuria, sudden onset, no retinopathy)

Drug	eGFR 45–59	eGFR 30–44	eGFR 15–29	Dialysis
Metformin	Full	Max 1000 mg; don't start	Stop	Stop
SGLT2i	Full (cardiorenal)	Full	Continue (start ≥20)	Stop
DPP-4i	Usual	Reduce (except linagliptin)	Reduce	Reduce
GLP-1 RA (sema, dula, lira)	Full	Full	Full (caution)	Limited data
Gliclazide / glipizide	Usual	Caution	Low dose	Avoid
Glimepiride	Caution	1 mg	Avoid	Avoid
Pioglitazone	Full	Full	Full (fluid retention)	Caution
Insulin	Usual	Reduce ≈25%	Reduce 25–50%	Reduce

Diabetic neuropathy

Type	Features	Management
Distal symmetric polyneuropathy (commonest)	Glove-and-stocking numbness, burning, loss of protective sensation	Glucose control (prevents in T1D), foot care, B12 if on metformin
Painful neuropathy	Burning, shooting pain worse at night	Drug table below

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Type	Features	Management
Autonomic — cardiovascular	Resting tachycardia, orthostatic hypotension, silent MI	Fludrocortisone, midodrine; slow positional change
Autonomic — gastrointestinal	Gastroparesis, diarrhoea, constipation	Small low-fat meals; metoclopramide 5–10 mg TDS (≤12 weeks); domperidone; erythromycin
Autonomic — genitourinary	Erectile dysfunction, neurogenic bladder	PDE5 inhibitors (not with nitrates); timed voiding
Mononeuropathy / amyotrophy / CIDP	Cranial nerve III/VI palsy, carpal tunnel, proximal thigh pain/wasting	Supportive; most recover; exclude other causes

Drug for painful neuropathy	Dose	Cautions
Pregabalin	75 mg BD → 150–300 mg BD (max 600 mg/day)	Dizziness, sedation, oedema, weight gain; reduce in CKD
Duloxetine	30 mg OD → 60 mg OD (max 120)	Nausea; avoid eGFR <30, liver disease
Gabapentin	300 mg at night → 900–3600 mg/day in 3 doses	Sedation; reduce in CKD
Amitriptyline / nortriptyline	10 mg at night → 25–75 mg	Anticholinergic; avoid in elderly, CAD, glaucoma
Tapentadol ER	50 mg BD → max 250 mg BD	Opioid; second-line
Capsaicin 8% patch	Up to 4 patches for 30 min every 3 months	Local burning

The diabetic foot

IWGDF risk category (2023)	Features	Foot screening interval
0 — very low	No LOPS, no PAD	Yearly
1 — low	LOPS or PAD	Every 6–12 months
2 — moderate	LOPS + PAD, or LOPS/PAD + deformity	Every 3–6 months
3 — high	LOPS or PAD + history of ulcer, amputation or ESKD	Every 1–3 months

Diabetic foot ulcer — principle	Action
Classify	Wagner (0–5), SINBAD or WIfI (wound, ischaemia, foot infection)
Off-load	Total contact cast or non-removable knee-high walker for plantar ulcers
Revascularise	ABI <0.9, toe pressure <30 mmHg or $TcPO_2$ <25 → urgent vascular imaging
Treat infection	Mild: oral amoxicillin-clavulanate or cephalexin (1–2 weeks). Moderate–severe: IV piperacillin-tazobactam or as per culture (deep tissue, not swab); X-ray/MRI for osteomyelitis (probe-to-bone)
Debride and dress	Sharp debridement; moist wound healing
Charcot foot	Warm, swollen, red foot with intact skin — immobilise in cast immediately
Prevent	Daily self-inspection, proper footwear, never walk barefoot, nail care, treat callus

Cardiovascular protection in diabetes

- **Statin** for all with diabetes aged 40–75 (high-intensity if ASCVD or additional risk factors); see Part IV.
- **BP <130/80** with ACEi/ARB-based therapy.

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- **Aspirin 75–100 mg**: for ASCVD (secondary prevention); for primary prevention only in high-risk patients after discussing bleeding risk.
- **SGLT2i and/or GLP-1 RA** with proven benefit for ASCVD, HF or CKD (Chapter 14).
- Screen for **stage B heart failure** with NT-proBNP; treat HF per guidelines.

MASLD (metabolic dysfunction-associated steatotic liver disease)

FIB-4	Interpretation	Next step
<1.3	Low risk of advanced fibrosis	Repeat in 1–3 years; lifestyle
1.3–2.67	Indeterminate	Transient elastography (liver stiffness <8 kPa low; ≥12 kPa high)
>2.67	High risk	Hepatology referral

Treat with weight loss (≥10% improves fibrosis), GLP-1 RA (semaglutide), tirzepatide or pioglitazone; **resmetirom** is FDA-approved for MASH with F2–F3 fibrosis; saroglitazar is approved in India.

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CHAPTER 21

Diabetes in Special Situations

Pregnancy, older adults, young-onset diabetes, pancreatic diabetes, TB and others

Gestational diabetes mellitus (GDM)

Approach	Test	Diagnostic threshold
Government of India (MoHFW 2018) / DIPSI — single step, non-fasting	75 g glucose irrespective of last meal; 2-h venous plasma glucose	**≥140 mg/dl** = GDM
IADPSG / WHO 2013 / ADA one-step	Fasting 75 g OGTT	Any one: fasting ≥92, 1-h ≥180, 2-h ≥153 mg/dl
Carpenter–Coustan two-step (ACOG)	50 g challenge (≥130–140) → 100 g 3-h OGTT	≥2 of: 95, 180, 155, 140 mg/dl

- Test at **first antenatal visit** (detect pre-existing diabetes) and again at **24–28 weeks** (India: repeat at 24–28 weeks if first test negative).
- **Glucose targets in pregnancy:** fasting <95 mg/dl, 1-h post-meal <140, 2-h post-meal <120; HbA1c <6% if achievable without hypoglycaemia.
- MNT and activity first; if targets not met in 1–2 weeks start **insulin** (NPH, regular, aspart, lispro, detemir; glargine/degludec acceptable).
- **Metformin** is permitted in Indian MoHFW guidance and widely used (crosses placenta; ADA prefers insulin). Glibenclamide is not preferred.
- **Postpartum:** stop treatment; OGTT at 4–12 weeks; lifelong screening every 1–3 years (≈50% develop T2D within 10 years); breastfeeding reduces risk.

Pre-existing diabetes and pregnancy

Preconception	During pregnancy
HbA1c <6.5% before conception; effective contraception until then	Insulin is the mainstay; requirements rise sharply after 20 weeks and fall abruptly after delivery
Folic acid 400 µg–5 mg daily (higher dose for obesity/diabetes per local protocols)	Aspirin 75–150 mg from 12–16 weeks to reduce pre-eclampsia
Stop statins, ACEi/ARB, SGLT2i, GLP-1 RA (≥2 months before), finerenone	Eye exam each trimester; BP and renal function; fetal growth scans
Retinal and renal assessment; thyroid function	Reduce insulin by ≈50% immediately after delivery (T1D); stop in GDM

Older adults (ADA 2026)

Health status	HbA1c	Fasting/pre-meal	BP	Lipids
Healthy (few comorbidities, intact cognition and function)	<7.0–7.5%	80–130	<130/80	Statin
Complex/intermediate (multiple illnesses, mild–moderate cognitive impairment, 2+ ADL limitations)	<8.0%	90–150	<130/80 (if safe)	Statin

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Health status	HbA1c	Fasting/pre-meal	BP	Lipids
Very complex/poor health (long-term care, end-stage illness, moderate–severe dementia)	Avoid reliance on HbA1c; avoid hypoglycaemia and symptomatic hyperglycaemia	100–180	<140/90	Consider benefit of statin

- Prefer drugs with low hypoglycaemia risk; avoid glibenclamide; simplify insulin to once-daily basal.
- Protein ≥0.8 g/kg/day; resistance exercise to prevent sarcopenia; screen for geriatric syndromes (falls, cognition, depression, polypharmacy).

Young-onset type 2 diabetes (<40 years)

Increasingly common in India; more aggressive course with earlier β-cell failure and complications. Treat intensively: lifestyle, early combination, GLP-1 RA/tirzepatide for weight, statin and BP control, contraception counselling, and screen for MODY/type 1 in lean patients.

Other special situations

Situation	Key points
Pancreatogenic diabetes (FCPD, chronic pancreatitis)	Insulin usually needed; pancreatic enzyme replacement (lipase 25 000–50 000 units with meals); vitamin D; avoid hypoglycaemia (low glucagon); screen for pancreatic cancer
MODY	HNF1A/HNF4A: very sensitive to **low-dose SU** (gliclazide 20–40 mg); GCK: mild stable hyperglycaemia — no treatment (except pregnancy); HNF1B: renal cysts, insulin
LADA	Avoid SU; early insulin if C-peptide low; DPP-4i/GLP-1 RA if C-peptide preserved
Diabetes and tuberculosis	Bidirectional screening (India's NTEP): screen all TB patients for diabetes and diabetics with cough >2 weeks for TB; rifampicin lowers SU/DPP-4i/pioglitazone levels — insulin often needed during intensive phase
HIV	Screen before ART and periodically; integrase inhibitors and protease inhibitors cause weight gain/hyperglycaemia; check drug interactions (dolutegravir raises metformin levels — max 1000 mg/day)
Post-transplant diabetes	Tacrolimus/steroids; insulin early; DPP-4i and SGLT2i (with care) later
Antipsychotic-induced	Olanzapine, clozapine highest risk; monitor weight, glucose, lipids at baseline, 12 weeks, yearly; metformin/GLP-1 RA
Checkpoint-inhibitor diabetes	Abrupt insulin-deficient diabetes, often DKA — insulin immediately
Cystic fibrosis-related diabetes	Insulin; high-calorie diet

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CHAPTER 22

Dyslipidaemia — Lipoproteins, Definition and Classification

From lipoprotein physiology to familial hypercholesterolaemia

Lipoproteins — what we measure and why

Lipoprotein	Main lipid	Main apolipoprotein	Role / atherogenicity
Chylomicrons	Dietary triglyceride	B-48, C-II, E	Carry dietary fat; very high levels cause pancreatitis
VLDL	Hepatic triglyceride	B-100, C-II, E	Remnants are atherogenic
IDL / remnants	TG + cholesterol	B-100, E	Highly atherogenic; raised in type III
LDL	Cholesterol	B-100 (one per particle)	**Causal** in atherosclerosis; small dense LDL more atherogenic
HDL	Cholesterol, phospholipid	A-I	Reverse cholesterol transport; low HDL is a risk marker, raising it with drugs does not help
Lipoprotein(a)	Cholesterol	B-100 + apo(a)	Genetically determined; atherogenic and pro-thrombotic; aortic stenosis

KEY POINT Every atherogenic particle (VLDL, IDL, LDL, Lp(a)) carries one **apoB** molecule. **Non-HDL-C** (total cholesterol − HDL-C) and **apoB** therefore capture total atherogenic burden better than LDL-C — especially when TG is high, in diabetes, obesity and in Indians.

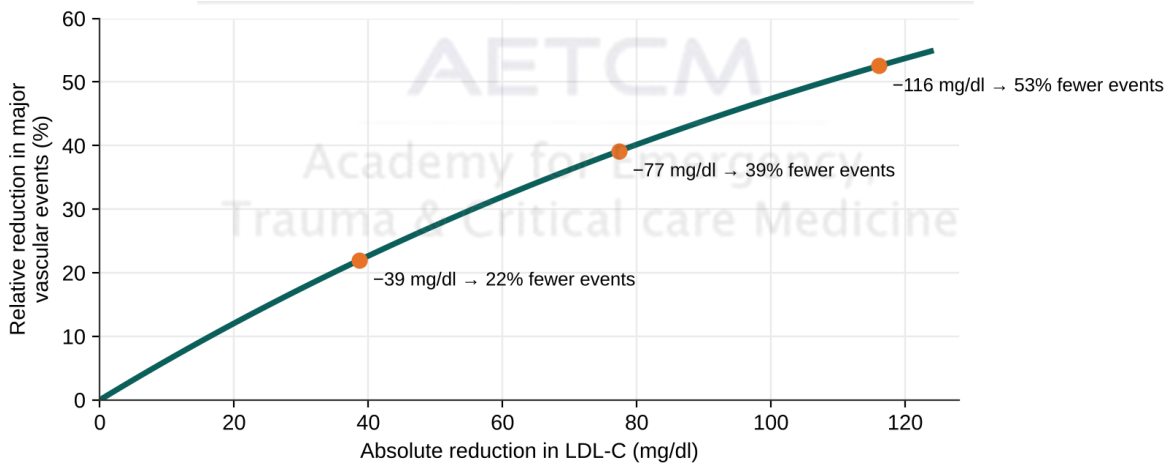


Figure 22.1. Cholesterol Treatment Trialists' meta-analysis: each 1 mmol/l (38.7 mg/dl) reduction in LDL-C lowers major vascular events by ≈22%, regardless of the starting LDL-C — 'lower is better'.

Definitions — lipid levels in adults (mg/dl)

Lipid	Desirable	Borderline	High	Very high
Total cholesterol	<200	200–239	≥240	—
LDL-C	<100 (optimal)	100–129 near-optimal; 130–159 borderline	160–189	≥190 (suspect FH)

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Lipid	Desirable	Borderline	High	Very high
Non-HDL-C	<130	130–159	160–189	≥190
Triglycerides (fasting)	<150	150–199	200–499	≥500 (severe ≥880–1000)
HDL-C	≥40 men, ≥50 women (low if below)	—	—	—
Lipoprotein(a)	<30 mg/dl (<75 nmol/l)	30–50 mg/dl	>50 mg/dl (>105–125 nmol/l)	>180 mg/dl (≈ FH-equivalent risk)
ApoB	<90 mg/dl	—	≥130 mg/dl (risk enhancer)	—

Conversion: cholesterol mg/dl ÷ 38.7 = mmol/l; triglyceride mg/dl ÷ 88.5 = mmol/l.

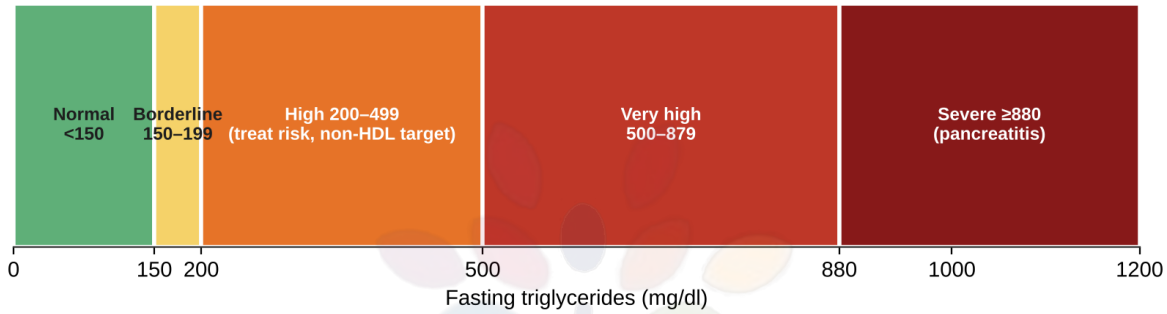


Figure 22.2. Triglyceride categories (mg/dl). Above 500 mg/dl the priority shifts to preventing pancreatitis; above 880–1000 mg/dl the risk of acute pancreatitis is high.

Phenotypic (Fredrickson) classification

Type	Lipoprotein raised	Lipids	Cause / clinical features
I	Chylomicrons	TG ↑↑↑	Familial chylomicronaemia syndrome (LPL, apoC-II deficiency); eruptive xanthomas, pancreatitis, lipaemia retinalis
IIa	LDL	TC ↑, LDL-C ↑	Familial hypercholesterolaemia, polygenic; tendon xanthomas, arcus
IIb	LDL + VLDL	TC ↑, TG ↑	Familial combined hyperlipidaemia — commonest familial form, premature CAD
III	IDL (remnants)	TC ↑, TG ↑ (similar)	Dysbetalipoproteinaemia (apoE2/E2); palmar and tubero-eruptive xanthomas
IV	VLDL	TG ↑	Familial hypertriglyceridaemia; obesity, diabetes, alcohol
V	VLDL + chylomicrons	TG ↑↑↑	Genetic + secondary factors (diabetes, alcohol, drugs); pancreatitis

Secondary causes — always exclude

Raised LDL-C	Raised triglycerides	Low HDL-C
Hypothyroidism, nephrotic syndrome, cholestasis (primary biliary cholangitis), anorexia nervosa, pregnancy; drugs — steroids, ciclosporin, thiazides (mild), progestins, anabolic steroids, amiodarone, isotretinoin	Obesity, uncontrolled diabetes, alcohol, CKD, hypothyroidism, pregnancy, MASLD, HIV; drugs — oral oestrogen, tamoxifen, β-blockers (non-selective), thiazides, steroids, retinoids, atypical antipsychotics, protease inhibitors, sirolimus, L-asparaginase	Smoking, obesity, inactivity, insulin resistance, very-low-fat diet, anabolic steroids, β-blockers

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CLINICAL PEARL Check **TSH, fasting glucose/HbA1c, creatinine, liver enzymes and urine protein** in every new dyslipidaemia — treating hypothyroidism or uncontrolled diabetes often corrects the lipids.

Familial hypercholesterolaemia (FH)

Autosomal dominant (LDLR, APOB, PCSK9 genes); heterozygous FH affects ≈1 in 250 people. Untreated men develop CAD by 50 and women by 60. Suspect FH if LDL-C ≥190 mg/dl in an adult (≥160 in a child), tendon xanthomas, or premature CAD in the patient or family.

Dutch Lipid Clinic Network criteria	Points
First-degree relative with premature ASCVD or LDL-C >95th percentile	1
First-degree relative with tendon xanthoma/arcus, or child <18 with LDL-C >95th percentile	2
Patient with premature CAD (men <55, women <60)	2
Patient with premature cerebral or peripheral vascular disease	1
Tendon xanthomas	6
Arcus cornealis before age 45	4
LDL-C ≥330 mg/dl / 250–329 / 190–249 / 155–189	8 / 5 / 3 / 1
Functional mutation in LDLR, APOB or PCSK9	8

Score: >8 definite FH; 6–8 probable; 3–5 possible; <3 unlikely. Treat as high risk from diagnosis (very high with ASCVD); target ≥50% reduction and LDL-C <70 (or <55); **cascade screening** of first-degree relatives.

The Indian dyslipidaemia pattern

- Total and LDL-C often only modestly raised, but **high TG and low HDL-C are very common** (atherogenic dyslipidaemia).
- Higher small dense LDL particles and **elevated Lp(a)** (≈25% of Indians vs 15–20% of Europeans).
- Therefore use **non-HDL-C** as a co-primary target and **measure Lp(a) at least once** (CSI 2024, LAI, ESC/EAS 2025, ACC/AHA 2026).

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CHAPTER 23

Lipid Evaluation, Risk Categories and Targets

Measuring lipids correctly and setting LDL-C, non-HDL-C and apoB goals

Basic lipid evaluation

Test	Notes
Standard lipid profile (TC, TG, HDL-C, calculated LDL-C, non-HDL-C)	**Non-fasting sample acceptable** for screening and follow-up; fast 8–12 h if TG >400 mg/dl or for diagnosis of hypertriglyceridaemia
LDL-C calculation	Friedewald (LDL = TC − HDL − TG/5) invalid if TG >400 and inaccurate when LDL <70; prefer **Martin–Hopkins** or **NIH (Sampson)** equations, or direct LDL-C
Non-HDL-C	TC − HDL-C; no extra cost; valid non-fasting
Secondary-cause screen	TSH, glucose/HbA1c, creatinine, urine protein, ALT/AST, (CK if planning statin with symptoms)

Advanced lipid tests

Test	When
Lipoprotein(a)	**Once in every adult** (ESC/EAS 2025, ACC/AHA 2026, CSI); especially premature ASCVD, family history, FH, recurrent events on therapy
Apolipoprotein B	High TG, diabetes, obesity, metabolic syndrome, very low LDL-C; residual risk on therapy
Genetic testing	Suspected FH (Dutch score ≥6), familial chylomicronaemia
hs-CRP	Risk enhancer (≥2 mg/l)
CAC score / carotid ultrasound	Borderline–intermediate risk with uncertainty about statin

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Risk categories and LDL-C targets — guidelines compared

Risk category	ESC/EAS 2019/2025	ACC/AHA 2026	India (CSI 2024 / LAI)
Very high (ASCVD, diabetes with TOD, eGFR <30, FH + ASCVD, SCORE2 ≥20%)	LDL-C <55 and ≥50% ↓; <40 if 2nd event within 2 years	Very high-risk ASCVD: <55; other ASCVD: <70	<55 (LAI extreme risk: <50, optional ≤30)
High (single severe factor, FH, diabetes without TOD, eGFR 30–59, SCORE2 10–19%)	<70 and ≥50% ↓	PREVENT ≥10%: <70	<70
Moderate (SCORE2 2–9%; young diabetics)	<100	PREVENT 3–<10%: <100	<100
Low	<116	PREVENT <3%: lifestyle	<100

Secondary targets	Very high	High	Moderate
Non-HDL-C (mg/dl)	<85	<100	<130
ApoB (mg/dl)	<65	<80	<100

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Secondary targets	Very high	High	Moderate
Triglycerides (mg/dl)	<150 (no formal target; indicates lower risk)	<150	<150

GUIDELINE Targets are **dual**: reach the absolute LDL-C goal **and** achieve at least a **50% reduction** from baseline in high and very high-risk patients. Lp(a) >50 mg/dl moves a patient up one risk category.

Statin benefit groups (ACC/AHA framework, adapted)

Group	Recommendation
Clinical ASCVD	High-intensity statin; add ezetimibe → PCSK9 mAb/inclisiran/bempedoic acid to reach <55–70
LDL-C ≥190 mg/dl (age 20–75)	High-intensity statin without risk calculation; screen for FH
Diabetes age 40–75	At least moderate-intensity statin; high-intensity if ≥1 risk factor/TOD/age ≥50; LDL-C <70 (<55 if ASCVD)
Primary prevention age 40–75, LDL-C 70–189	By risk: high (≥10%) → statin; intermediate → statin, use risk enhancers/CAC; borderline → discuss
CKD stage 3–4 (not on dialysis)	Statin or statin + ezetimibe
Age >75	Continue if tolerated; start based on risk and life expectancy
HIV age ≥40	Statin regardless of risk (REPRIEVE; pitavastatin)

Follow-up after starting therapy

When	What to check
4–12 weeks after starting or changing	Lipid profile (response and adherence); ALT if symptoms or baseline abnormal; CK only if muscle symptoms
Every 3–12 months	Lipids until at goal, then yearly
Each visit	Adherence, muscle symptoms, diet, weight

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CHAPTER 24

Lipid-Lowering Drugs and Step-Up Therapy

Statins, ezetimibe, bempedoic acid, PCSK9 inhibitors, inclisiran and triglyceride therapies

Statin intensity

Intensity (LDL-C reduction)	Atorvastatin	Rosuvastatin	Others
High (≥50%)	40–80 mg	20–40 mg	—
Moderate (30–49%)	10–20 mg	5–10 mg	Simvastatin 20–40; pravastatin 40–80; pitavastatin 2–4; fluvastatin XL 80
Low (<30%)	—	—	Simvastatin 10; pravastatin 10–20; pitavastatin 1

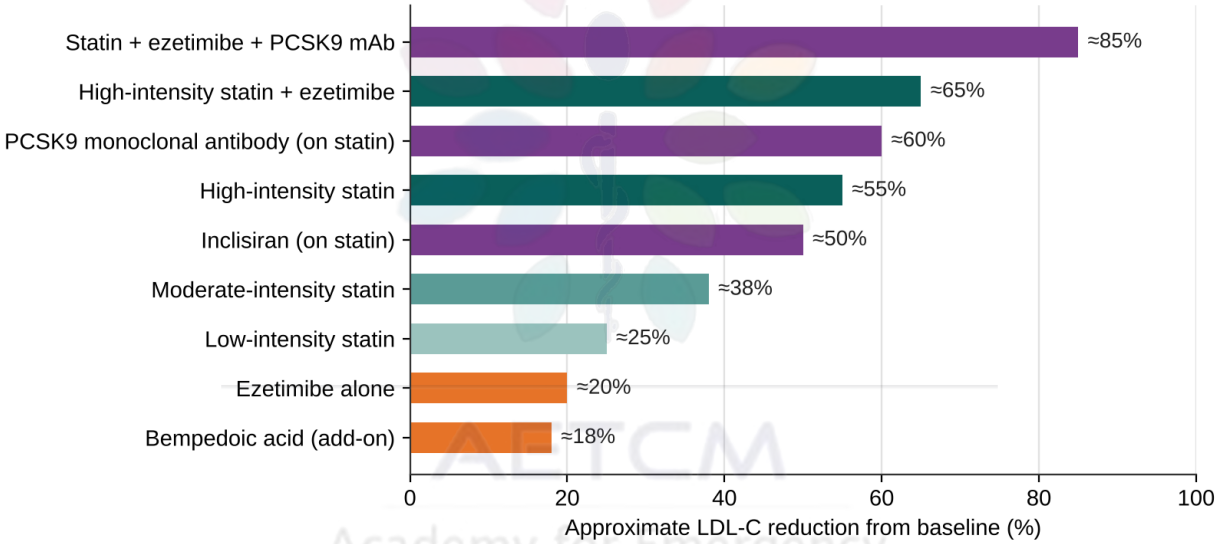


Figure 24.1. Approximate LDL-C lowering with each strategy. Combination therapy reaches very low targets that statin monotherapy cannot.

Non-statin LDL-lowering drugs

Drug	Dose	LDL-C ↓	Outcome evidence	Cautions
Ezetimibe	10 mg OD	20–25% (alone); additional 20–25% with statin	IMPROVE-IT (post-ACS)	Well tolerated; cheap; FDCs with statins
Bempedoic acid	180 mg OD (± ezetimibe 10 mg FDC)	17–25%	CLEAR Outcomes (statin-intolerant): MACE ↓13%	Hyperuricaemia/gout, tendon rupture (rare); no myalgia (not active in muscle)
Evolocumab (PCSK9 mAb)	140 mg SC every 2 weeks or 420 mg monthly	50–60%	FOURIER: MACE ↓15–20%	Injection-site reactions; cost
Alirocumab (PCSK9 mAb)	75–150 mg SC every 2 weeks or 300 mg monthly	50–60%	ODYSSEY OUTCOMES: MACE ↓15%, mortality ↓	As above

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Drug	Dose	LDL-C ↓	Outcome evidence	Cautions
Inclisiran (siRNA)	284 mg SC at 0, 3 months, then every 6 months	≈50%	Outcome trials (ORION-4, VICTORION-2P) ongoing/reporting	Given by health professional; twice-yearly — adherence
Bile acid sequestrants (colesevelam, cholestyramine)	Colesevelam 3.75 g/day; cholestyramine 4–24 g/day	15–25%	LRC-CPPT (older)	GI upset, raise TG; bind other drugs
Evinacumab (ANGPTL3 mAb)	15 mg/kg IV monthly	≈50% in HoFH	—	Homozygous FH (specialist)
Lomitapide (MTP inhibitor)	5–60 mg OD	≈40–50%	—	HoFH only; hepatic steatosis

Step-up therapy for LDL-C

LDL-C lowering ladder (ESC/EAS 2025, ACC/AHA 2026, CSI 2024)

Step 1

Lifestyle + **statin at the intensity matched to risk** (high-intensity for very high/high risk). Check lipids in 4–12 weeks.

▼

Step 2

Not at goal → **add ezetimibe 10 mg** (statin + ezetimibe single pill). In very high risk with LDL-C far above goal (e.g. post-ACS), start statin + ezetimibe together.

▼

Step 3

Still not at goal → **add PCSK9 monoclonal antibody or inclisiran** (≈50–60% further reduction); or **bempedoic acid** if injectables unaffordable or for modest gaps.

▼

Statin intolerance

Re-challenge with a different statin, low or alternate-day dosing (rosuvastatin 5 mg twice weekly) → ezetimibe → bempedoic acid → PCSK9 mAb/inclisiran.

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CLINICAL PEARL Estimate the **percentage reduction needed** = (baseline LDL − goal) ÷ baseline × 100. If >50% is needed, a statin alone will not get there — plan combination therapy from the start. Example: LDL-C 160 → goal 55 needs 66%: high-intensity statin + ezetimibe.

Statin-associated muscle symptoms (SAMS) and safety

Issue	Facts and action
Myalgia	Placebo-controlled trials show ≈90% of reported symptoms are not caused by the statin (nocebo; SAMSON). Check CK, TSH, vitamin D; stop for 2–4 weeks; re-challenge with another statin or lower dose
Myopathy / rhabdomyolysis	CK >10× ULN with symptoms — stop. Risk ↑ with age, hypothyroidism, CKD, gemfibrozil, macrolides, azoles, ciclosporin, amiodarone/verapamil (simvastatin), grapefruit
Liver	Transaminase rise <3× ULN — continue. Statins are safe and beneficial in MASLD and compensated cirrhosis; avoid in decompensated cirrhosis/acute liver failure
New-onset diabetes	Small increase (≈1 per 1000 per year), mainly in prediabetes; CV benefit far greater — do not stop

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Issue	Facts and action
Pregnancy and breastfeeding	Stop statins (may continue in very high-risk women after specialist discussion)
Drug interactions	Rosuvastatin and pravastatin have fewest CYP interactions; Asians need lower rosuvastatin doses (higher blood levels)

Management of hypertriglyceridaemia

TG level (mg/dl)	Priority	Management
150–499	ASCVD risk (non-HDL-C target)	Lifestyle (weight loss, alcohol, sugar), control glucose, review drugs; **statin** per risk; if high/very high risk and TG 135–499 on statin → **icosapent ethyl 2 g BD** (REDUCE-IT)
500–879	Risk + prevent progression	As above + fenofibrate and/or omega-3 (prescription 2–4 g/day EPA+DHA); saroglitazar 4 mg (India) in diabetic dyslipidaemia
≥880–1000	**Prevent pancreatitis**	Very-low-fat diet (<10–15% energy, ≈20 g/day), **total alcohol abstinence**, fenofibrate 145–160 mg, omega-3 3–4 g, insulin for diabetes; volanesorsen/olezarsen/plozasiran for familial chylomicronaemia syndrome
Acute pancreatitis with TG >1000	Emergency	NPO, IV fluids, **IV insulin + dextrose infusion** (lowers TG within days); heparin not recommended; plasmapheresis in selected severe cases

Triglyceride drug	Dose	Effect	Cautions
Fenofibrate	145–160 mg OD (micronised 200 mg)	TG ↓30–50%, HDL ↑	Raises creatinine (reversible); gallstones; myopathy with statins (safer than gemfibrozil); reduce in CKD, avoid eGFR <30
Gemfibrozil	600 mg BD	TG ↓	**Never with statins** (rhabdomyolysis)
Icosapent ethyl (pure EPA)	2 g BD with meals	TG ↓20–30%; MACE ↓25% (REDUCE-IT)	AF, bleeding; mixed EPA/DHA products did not show CV benefit
Omega-3 (EPA + DHA)	2–4 g/day	TG ↓20–45%	Prescription strength; not OTC fish-oil capsules
Saroglitazar (India)	4 mg OD	TG ↓45%, also HbA1c ↓	Diabetic dyslipidaemia; MASLD
Niacin	Not recommended	—	No outcome benefit (AIM-HIGH, HPS2-THRIVE); flushing, hyperglycaemia

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GUIDELINE Fibrates, niacin and omega-3 supplements added to statins **do not reduce cardiovascular events** (ACCORD-Lipid, PROMINENT, ADA 2026) — use fibrates mainly to prevent pancreatitis, and icosapent ethyl for residual risk.

Lipoprotein(a)

- No approved Lp(a)-specific therapy yet; **pelacarsen, olpasiran, lepodisiran, zerlasiran and muvalaplin** are in phase 3 outcome trials (Lp(a)HORIZON and others).
- Manage by **intensifying LDL-C lowering** and controlling all other risk factors; PCSK9 mAbs lower Lp(a) by ≈25%.
- Lipoprotein apheresis in progressive ASCVD with very high Lp(a) (specialist centres).

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Special populations

Group	Approach
Acute coronary syndrome	High-intensity statin before discharge; add ezetimibe if LDL-C unlikely to reach <55 (start together in lipid-naïve with high LDL-C); recheck at 4–6 weeks; PCSK9 mAb early if not at goal
CKD on dialysis	Do not start statins (no benefit — AURORA, 4D); continue if already taking
Children with FH	Statins from age 8–10
Elderly >75	Continue statin in secondary prevention; primary prevention by shared decision
Women planning pregnancy	Stop statins 1–3 months before conception
HIV	Pitavastatin or rosuvastatin (fewer interactions)

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CHAPTER 25

Obesity — Definition, Classification and Staging

Beyond BMI — adiposity, complications and the new clinical definitions

Definition

Obesity is a **chronic, relapsing, progressive disease** of excess or abnormally distributed body fat that impairs health. It is not a lifestyle choice: appetite and body weight are regulated by genes, the gut–brain axis (GLP-1, GIP, leptin, ghrelin), environment, sleep, stress and drugs. After weight loss, biological adaptations (lower energy expenditure, higher hunger) drive regain — which is why long-term treatment is needed.

BMI classification

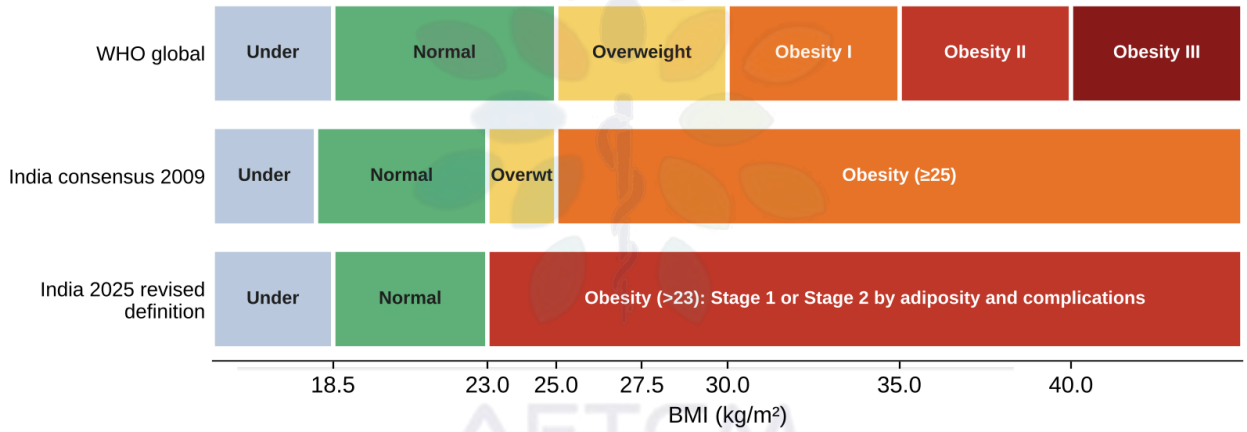


Figure 25.1. BMI categories: WHO global, Indian consensus 2009 (Misra et al.) and the 2025 revised Indian definition, which removes the 'overweight' category and stages obesity by adiposity and complications.

Category	WHO (kg/m²)	Asian Indian (2009 consensus)
Underweight	<18.5	<18.0
Normal	18.5–24.9	18.0–22.9
Overweight	25.0–29.9	23.0–24.9
Obesity class I	30.0–34.9	≥25
Obesity class II	35.0–39.9	—
Obesity class III (severe)	≥40	≥32.5 is often used for surgical thresholds in Asians

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Measures of adiposity

Measure	Abnormal (Asian Indian)	How
Waist circumference (WC)	≥90 cm men; ≥80 cm women	Midpoint between lowest rib and iliac crest, end of normal expiration
Waist-to-height ratio	>0.5	'Keep your waist to less than half your height'
Waist-to-hip ratio	>0.90 men; >0.85 women	—
Body fat % (DEXA, BIA)	>25% men; >30–35% women (2025 Indian: >25.5% men, >38% women)	DEXA is reference; BIA for clinics
Neck circumference	>40 cm (OSA risk)	—

The new definitions (2025)

BMI alone misclassifies many people — especially muscular individuals and 'thin-fat' Indians. Two influential frameworks published in 2025 define obesity by **confirmed excess adiposity plus its effects on health**.

Framework	Diagnosis	Stages
Lancet Diabetes & Endocrinology Commission (Rubino et al., 2025)	Excess adiposity confirmed by BMI **plus** ≥1 anthropometric measure (WC, WHR, WHtR) or direct body-fat measurement (BMI >40 alone suffices)	**Pre-clinical obesity**: excess adiposity with preserved organ function. **Clinical obesity**: excess adiposity with organ dysfunction (18 adult criteria — e.g. breathlessness, HFpEF, OSA, MASLD, knee/hip pain limiting mobility, lymphoedema) or limitation of daily activities
Revised definition for Asian Indians (Misra et al., 2025)	BMI **>23 kg/m²** (or high body fat with BMI ≤23)	**Stage 1**: BMI >23 without abdominal adiposity-related dysfunction or comorbidity. **Stage 2**: BMI >23 **plus** abdominal adiposity (WC ≥90/80 cm or WHtR >0.5) **plus** functional limitation or ≥1 obesity-related comorbidity

Obesity-related comorbidities (stage 2 / clinical obesity)	
Metabolic	Prediabetes, type 2 diabetes, dyslipidaemia, metabolic syndrome, MASLD, gout
Cardiovascular	Hypertension, CAD, HFpEF, AF, stroke, venous thromboembolism, lower-limb oedema
Respiratory	Obstructive sleep apnoea, obesity hypoventilation syndrome, asthma
Musculoskeletal	Knee/hip osteoarthritis, back pain
Reproductive	PCOS, infertility, male hypogonadism, pregnancy complications
Other	GERD, gallstones, idiopathic intracranial hypertension, CKD, urinary incontinence, depression, cancers (endometrium, breast, colon, kidney, liver, oesophagus)

Edmonton Obesity Staging System (EOSS)

Stage	Description	Implication
0	No risk factors, symptoms or limitations	Prevent further gain
1	Subclinical risk factors (borderline BP/glucose), mild symptoms	Lifestyle; monitor
2	Established comorbidity (HTN, T2D, OSA, OA), moderate limitation	Treat obesity actively — drugs/surgery

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Stage	Description	Implication
3	End-organ damage (MI, HF, stroke, severe disability)	Aggressive treatment
4	Severe, potentially end-stage disability	Palliative / specialist

KEY POINT Use BMI to **screen**, waist and body composition to **confirm**, and complications to **stage**. Treatment intensity should follow stage, not BMI alone.

Sarcopenic obesity

Low muscle mass and strength together with excess fat — common in the elderly and in Indians. Screen with grip strength (<27 kg men, <16 kg women) and chair-stand test. During weight loss preserve muscle with **protein 1.0–1.5 g/kg/day and resistance training**.

Childhood obesity — brief note

Use age- and sex-specific BMI charts (IAP 2015 for Indian children): overweight and obesity are defined by the centiles corresponding to adult BMI 23 and 27 kg/m². Family-based lifestyle therapy is the cornerstone; GLP-1 RA (liraglutide, semaglutide) are approved from age 12 in several countries.

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CHAPTER 26

Evaluation of the Patient with Obesity

History, causes, complications, basic and advanced investigations

History — the ‘weight story’

Ask about	Detail
Weight trajectory	Age of onset, maximum and minimum adult weight, triggers (pregnancy, job change, quitting smoking, drugs, menopause), previous attempts and results
Eating pattern	Meals, snacking, portion sizes, eating out, sugary drinks, night eating, binge eating, emotional eating
Activity and sleep	Steps, sitting time, exercise, shift work, sleep hours, snoring
Medications that cause weight gain	Insulin, sulfonylureas, pioglitazone, steroids, antipsychotics (olanzapine, clozapine), antidepressants (mirtazapine, paroxetine, TCAs), valproate, gabapentin/pregabalin, β-blockers (older), progestins (DMPA)
Symptoms of complications	Breathlessness, snoring/apnoeas, joint pain, reflux, menstrual irregularity, infertility, urinary leakage, low mood
Endocrine clues	Cold intolerance (hypothyroid), striae/proximal weakness (Cushing's), hirsutism (PCOS), headache/visual change (hypothalamic lesion)
Readiness to change	Goals, barriers, family support, finances

Examination

- Weight, height, BMI, waist, (hip), neck circumference, BP with appropriate cuff.
- Acanthosis nigricans and skin tags (insulin resistance); hirsutism, acne; striae (wide violaceous — Cushing's); goitre.
- Signs of complications: hepatomegaly, oedema, intertrigo, joint deformities, venous disease.
- Dysmorphism, short stature, developmental delay in childhood-onset obesity (genetic syndromes).

Secondary causes of obesity

Cause	Clues	Test
Hypothyroidism	Fatigue, cold intolerance, constipation	TSH
Cushing's syndrome	Central obesity, wide purple striae, proximal myopathy, easy bruising, hypertension, diabetes	1 mg overnight DST, late-night salivary cortisol, 24-h UFC
PCOS	Oligomenorrhoea, hirsutism, acne	Testosterone, LH/FSH, pelvic ultrasound
Male hypogonadism	Low libido, ED	Morning total testosterone, LH
Hypothalamic obesity	After tumour/surgery/radiation; rapid weight gain	MRI brain
Drug-induced	Temporal relation	Medication review
Monogenic (MC4R, leptin, POMC) and syndromic (Prader–Willi, Bardet–Biedl)	Severe early-onset (<5 years) obesity, hyperphagia, developmental delay	Genetic testing; setmelanotide for specific defects

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Basic investigations

Test	Purpose
Fasting glucose and HbA1c	Prediabetes/diabetes
Lipid profile	Dyslipidaemia
ALT, AST, platelets (FIB-4); ultrasound abdomen	MASLD and fibrosis; gallstones
Creatinine/eGFR, UACR	Kidney disease; drug dosing
TSH	Hypothyroidism
Uric acid	Gout risk
CBC, vitamin D, B12, ferritin	Baseline nutrition (essential before surgery)
ECG	Baseline; LVH, AF

Advanced investigations

Test	Indication
Body composition (DEXA, BIA)	Confirm adiposity; sarcopenia; monitor muscle during weight loss
Polysomnography	STOP-BANG ≥3, daytime sleepiness, resistant HTN, before bariatric surgery
Arterial blood gas, bicarbonate	Suspected obesity hypoventilation (BMI >30 with daytime hypercapnia, $PaCO_2$ >45)
Echocardiography, NT-proBNP (lower in obesity)	Breathlessness, oedema — HFpEF
Transient elastography	FIB-4 ≥1.3
Endocrine work-up (cortisol, testosterone, LH/FSH, prolactin)	Clinical suspicion
Upper GI endoscopy	Before bariatric surgery; reflux symptoms
Psychological assessment	Binge-eating disorder, depression, before surgery
Resting metabolic rate (indirect calorimetry)	Research/specialist; plateau

Who should be treated, and how intensively

Indian 2025 stage / BMI	Treatment
Stage 1 (BMI >23, no complications)	Lifestyle therapy. Drugs may be considered if BMI ≥27.5, weight gain >10% despite lifestyle, or high risk of progression (e.g. strong family history)
Stage 2 (abdominal adiposity + complication or limitation)	Intensive lifestyle **plus early pharmacotherapy**; metabolic surgery if criteria met
BMI ≥27.5 with comorbidities not controlled by drugs; ≥30 with comorbidities; ≥35 regardless	Consider metabolic–bariatric surgery

CLINICAL PEARL Ask permission before discussing weight, use person-first language ('person with obesity'), weigh privately, and use large cuffs, wide chairs and appropriate examination couches. **Weight stigma worsens outcomes**.

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CHAPTER 27

Lifestyle Therapy and Anti-Obesity Medications

Calorie deficit, activity, behaviour and drugs — class, dose and escalation

Setting goals

Weight loss	Expected benefits
3–5%	Lower TG, glucose, HbA1c; reduced risk of developing T2D (prediabetes)
5–10%	BP, lipids, HbA1c improve; less MASLD, knee pain, urinary incontinence; better fertility in PCOS
10–15%	MASH resolution, fewer OSA events, diabetes remission possible, CV events reduced (SELECT trial: semaglutide −20% MACE)
>15%	Diabetes remission in many, OSA resolution, HFpEF improvement, reduced mortality

Dietary therapy

Approach	Details
Calorie deficit	**500–750 kcal/day** deficit (≈1200–1500 kcal/day women, 1500–1800 kcal/day men) → 0.5–1 kg/week
Diet pattern	Any pattern the person can sustain: Mediterranean, low-carbohydrate, high-protein, DASH, Indian plate with reduced cereal portions
Meal replacement / low-calorie diets	800–1200 kcal/day for 8–16 weeks under supervision (DiRECT approach), then structured food reintroduction
Time-restricted eating / intermittent fasting	Eating window 8–10 h, or 5:2 — similar results to daily calorie restriction; helps some people adhere
Protein	1.0–1.5 g/kg (ideal body weight) to preserve muscle
Avoid	Sugar-sweetened beverages, fruit juices, fried snacks, bakery products, alcohol, large portions

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Physical activity and behavioural therapy

- **Weight loss:** 150 min/week initially, building to **200–300 min/week** moderate activity; resistance training 2–3 days/week.
- **Weight maintenance:** ≥200–300 min/week; daily self-weighing; continued contact.
- Behavioural package: self-monitoring (food, steps, weight), goal setting, stimulus control, problem solving, relapse prevention, stress and sleep management.
- High-intensity programmes (≥14 sessions in 6 months) achieve 5–10% weight loss (Look AHEAD, DPP).

Indications for anti-obesity medication (AOM)

Guideline	Indication
International (FDA/EMA labels; AACE, Obesity Society)	BMI ≥30, or ≥27 with ≥1 weight-related comorbidity, as adjunct to lifestyle

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Guideline	Indication
Asian / Indian practice	BMI ≥25 (obesity), or ≥23 with comorbidity; 2025 Indian definition — early drugs in stage 2
WHO 2025 guideline	Conditional recommendation for GLP-1-based therapies (semaglutide, tirzepatide, liraglutide) in adults with obesity, with lifestyle support

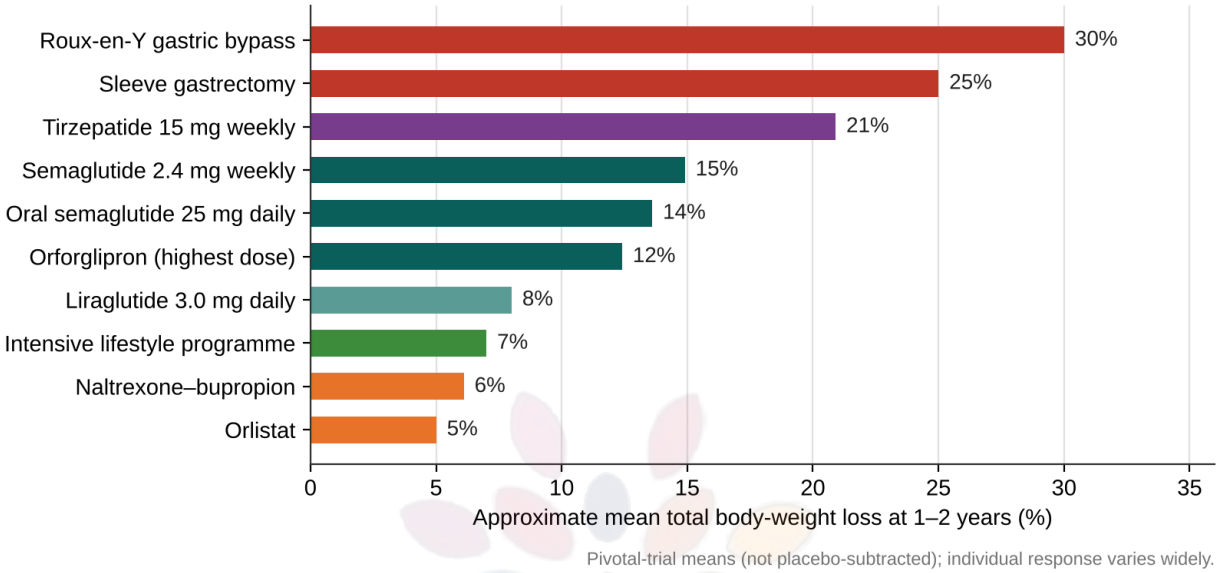


Figure 27.1. Approximate mean weight loss with lifestyle, anti-obesity drugs and metabolic surgery in pivotal trials.

Anti-obesity medications — class, drug and dose

Drug	Mechanism	Dose and escalation	Mean loss	Key cautions
Tirzepatide	GIP/GLP-1 dual agonist	2.5 mg SC weekly × 4 weeks → +2.5 mg every 4 weeks → 5, 10 or 15 mg weekly	≈15–21%	GI effects; pancreatitis/gallbladder; MTC/MEN2 contraindication; reduces oral contraceptive absorption during escalation (use barrier method 4 weeks after each step)
Semaglutide (SC)	GLP-1 RA	0.25 mg weekly → 0.5 → 1.0 → 1.7 → **2.4 mg** weekly (4-weekly steps)	≈15%	As GLP-1 RA; retinopathy in diabetes; muscle loss
Semaglutide (oral, obesity)	GLP-1 RA	Escalate to **25 mg** daily (oral Wegovy formulation), fasting with small sip of water	≈14%	As above; dosing conditions
Orforglipron	Oral small-molecule GLP-1 RA	Once daily, no food/water restrictions; escalated every 4 weeks to maintenance (per label)	≈11–12%	FDA-approved April 2026; India availability pending
Liraglutide 3.0 mg	GLP-1 RA	0.6 mg daily → increase 0.6 mg weekly → 3.0 mg daily	≈8%	Daily injection; GI effects
Orlistat	Pancreatic lipase inhibitor	120 mg TDS with fat-containing meals (OTC 60 mg)	≈3–5%	Oily stools, faecal urgency; fat-soluble vitamin deficiency (give multivitamin at bedtime); oxalate nephropathy
Naltrexone–bupropion ER	Opioid antagonist + NDRI	8/90 mg: 1 tab OD → 2 tabs BD by week 4	≈5–6%	Seizures, uncontrolled HTN, opioid use, MAOI — contraindicated; suicidality warning
Phentermine–topiramate ER	Sympathomimetic + anticonvulsant	3.75/23 mg → 7.5/46 mg (max 15/92 mg)	≈9–10%	Teratogenic; CVD, glaucoma, hyperthyroidism; not available in India

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Drug	Mechanism	Dose and escalation	Mean loss	Key cautions
Setmelanotide	MC4R agonist	1–3 mg SC daily	Variable	Only for POMC, PCSK1, LEPR deficiency or Bardet–Biedl syndrome

KEY POINT Evaluate response at **12–16 weeks on the maintenance dose**: if weight loss is <5%, change or stop the drug. Obesity drugs work only while they are taken — stopping typically leads to regain of two-thirds of the lost weight within a year (STEP-1 extension, SURMOUNT-4). Plan long-term therapy, as for hypertension. The optimal dose may be below the maximum (ADA 2026).

Choosing a drug

Patient profile	Preferred
Obesity + T2D	Tirzepatide or semaglutide (dual glycaemic and weight benefit); SGLT2i adds modest loss
Obesity + ASCVD	Semaglutide 2.4 mg (SELECT: MACE ↓20%)
Obesity + HFpEF	Semaglutide or tirzepatide (STEP-HFpEF, SUMMIT)
Obesity + OSA	Tirzepatide (FDA-approved for moderate–severe OSA in obesity)
Obesity + MASLD/MASH	Semaglutide, tirzepatide
Obesity + CKD	Semaglutide (FLOW); SGLT2i
Obesity + knee OA	Semaglutide (STEP-9 — pain improvement)
Cost-limited / needle-averse	Generic semaglutide (India), orlistat, oral options
Binge eating, smoking cessation, depression	Naltrexone–bupropion (where available)

Drugs to avoid or substitute in people with obesity

Instead of	Prefer
Sulfonylurea, pioglitazone, high-dose insulin	Metformin, SGLT2i, GLP-1 RA, tirzepatide
Atenolol/metoprolol (older β-blockers)	ACEi/ARB, CCB; carvedilol/nebivolol if β-blocker needed
Olanzapine, clozapine, mirtazapine, paroxetine	Aripiprazole, ziprasidone, bupropion, fluoxetine/sertraline (with psychiatrist)
Gabapentin/pregabalin (for pain)	Topical agents, duloxetine
Medroxyprogesterone injection	IUD, other contraceptives

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CHAPTER 28

Metabolic–Bariatric Surgery and Long-Term Obesity Care

Step-up beyond drugs, endoscopic options, post-operative care and weight maintenance

The treatment ladder

Stepped care of obesity

Level 1 — Lifestyle therapy for all

Diet, activity, behaviour, sleep; treat complications. Expected 3–7%.

▼

Level 2 — Lifestyle + anti-obesity medication

Stage 2 obesity or not reaching goal: GLP-1 RA, tirzepatide, orlistat. Expected 10–20%.

▼

Level 3 — Endoscopic therapy

Intragastric balloon (6–12 months, 10–12%) or endoscopic sleeve gastroplasty (15–18%) for those unwilling or unfit for surgery.

▼

Level 4 — Metabolic–bariatric surgery

Sleeve gastrectomy, Roux-en-Y gastric bypass, one-anastomosis gastric bypass. Expected 25–35% and durable; remission of diabetes, OSA, hypertension.

Indications for metabolic–bariatric surgery

Guideline	Indications
ASMBS/IFSO 2022	BMI **≥35** regardless of comorbidity; BMI **30–34.9** with metabolic disease; **Asians: BMI ≥27.5** may be considered; age is not an absolute limit (adolescents and >65 selected)
Revised Indian definition 2025	BMI ≥35 regardless of comorbidities; ≥30 with comorbidities not controlled by lifestyle + drugs; **≥27.5 with comorbidities** (especially uncontrolled T2D); preferred age 18–65
Diabetes surgery summit / ADA 2026	Metabolic surgery recommended in T2D with BMI ≥30 (≥27.5 in Asians) when hyperglycaemia is inadequately controlled despite optimal medical therapy

Contraindications

Uncontrolled psychiatric illness, active substance or alcohol misuse, untreated binge-eating disorder, inability to comply with lifelong supplementation and follow-up, prohibitive anaesthetic risk, active cancer, pregnancy (planned within 12–18 months)

Common procedures

Procedure	Mechanism	Weight loss at 5 years	Advantages	Disadvantages
Sleeve gastrectomy (SG)	Restrictive + hormonal (↓ghrelin, ↑GLP-1)	≈20–25%	Technically simpler; no anastomosis; commonest in India	GERD (new or worse), Barrett's; irreversible

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Procedure	Mechanism	Weight loss at 5 years	Advantages	Disadvantages
Roux-en-Y gastric bypass (RYGB)	Restrictive + mild malabsorption + hormonal	≈25–30%	Best for GERD and diabetes; durable	Dumping, marginal ulcer, internal hernia, micronutrient deficiencies, alcohol sensitivity
One-anastomosis gastric bypass (OAGB)	Restrictive + malabsorptive	≈25–30%	Shorter operation; strong diabetes effect	Bile reflux, protein–calorie malnutrition
Biliopancreatic diversion / SADI-S	Mainly malabsorptive	≈30–35%	BMI >50	Malnutrition, diarrhoea; specialist only

Pre-operative evaluation

- Multidisciplinary review: surgeon, physician/endocrinologist, dietitian, psychologist, anaesthetist.
- Labs: CBC, glucose/HbA1c, lipids, LFT, renal function, TSH, ferritin, B12, folate, vitamin D, calcium, albumin; pregnancy test.
- Polysomnography (start CPAP if OSA); upper GI endoscopy; cardiac evaluation as indicated.
- Optimise glucose (HbA1c <8% where possible), stop smoking ≥6 weeks before, pre-operative weight loss (liver shrinkage diet 2 weeks).

Post-operative care — lifelong

Item	Recommendation
Diet progression	Clear liquids (days 1–2) → full liquids (2 weeks) → pureed (2 weeks) → soft → regular by 6–8 weeks; protein **60–80 g/day** (≥1.2 g/kg); small meals, chew well, no drinking with meals
Supplements (after RYGB/SG)	Multivitamin-mineral 1–2 daily; **calcium citrate 1200–1500 mg/day**; **vitamin D 3000 IU/day** (titrate to >30 ng/ml); **B12** 350–1000 µg oral daily or 1000 µg IM monthly; **iron** 45–60 mg elemental (menstruating women); folate 400–800 µg; thiamine (if vomiting)
Diabetes drugs	Stop SU and prandial insulin at surgery; reduce basal insulin by 30–50%; continue metformin if needed; monitor glucose closely
BP drugs	Reduce as weight falls; watch for hypotension
Avoid	NSAIDs (marginal ulcers after RYGB), alcohol; extended-release tablets may be poorly absorbed
Pregnancy	Avoid for 12–18 months after surgery; reliable contraception (oral contraceptives may be less effective after malabsorptive procedures)
Monitoring	Labs at 3, 6 and 12 months, then yearly: CBC, ferritin, B12, folate, vitamin D, calcium, PTH, albumin, LFT; bone density as indicated

Late complication	Clue	Action
Dumping syndrome	Early (within 30 min): cramps, diarrhoea, palpitations; late (1–3 h): hypoglycaemia	Avoid simple sugars; small protein-rich meals; acarbose for late dumping
Post-bariatric hypoglycaemia	Neuroglycopenia 1–3 h after meals, months–years after RYGB	Diet modification, acarbose; specialist
Nutritional deficiencies	Anaemia, neuropathy (B12, copper), Wernicke's (thiamine), bone disease	Supplement; IV thiamine before glucose if vomiting
Weight regain	≥15–20% regain of lost weight	Reassess diet/behaviour; add GLP-1 RA/tirzepatide; revisional surgery
Gallstones	Rapid weight loss	Ursodeoxycholic acid 300 mg BD for 6 months post-op
Alcohol use disorder	Faster absorption after RYGB	Screen; counsel

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Weight maintenance — the long game

- Treat obesity as a **chronic disease**: regular follow-up (every 1–3 months in the first year, then 3–6-monthly).
- Continue effective anti-obesity medication long-term; dose may be reduced to the lowest effective dose.
- High activity (200–300 min/week), high protein, regular self-weighing, consistent eating pattern.
- Watch for and treat complications, mood disorders and weight-promoting drugs.

CLINICAL PEARL The best predictor of success is **continuity of care**. A patient who returns every three months to be weighed, encouraged and adjusted does far better than one given a diet sheet once.

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CHAPTER 29

Integrated Cardiometabolic Care — Putting It All Together

One patient, four diseases — prioritising, combining and following up

The integrated approach

Most patients in a lifestyle-disease clinic have two or more of the four conditions. Instead of four separate prescriptions, build **one plan** around shared lifestyle therapy and drugs with multiple benefits.

Drug	BP	Glucose	Lipids	Weight	Heart	Kidney	Liver
Semaglutide / tirzepatide	↓	↓↓	TG ↓	↓↓↓	MACE ↓, HFpEF ↓	↓ (sema)	MASH ↓
SGLT2 inhibitor	↓ 3–5	↓	—	↓	HF ↓↓	↓↓	—
ACEi / ARB	↓↓	—	—	—	↓	↓ (albuminuria)	—
Finerenone	↓ (small)	—	—	—	HF ↓	↓	—
Metformin	—	↓↓	—	↓ (small)	Possible	—	—
Pioglitazone	—	↓↓	TG ↓, HDL ↑	↑	Stroke ↓; HF ↑	—	MASH ↓
Statin	—	Slight ↑	↓↓ LDL	—	MACE ↓↓	—	Safe in MASLD
Saroglitazar (India)	—	↓	TG ↓↓	—	—	—	↓ ALT

A practical order of priorities for a new multi-morbid patient

1. **Stop tobacco** and start lifestyle therapy — the shared foundation.
2. **Treat dangerous numbers first**: BP ≥180/110, glucose ≥300 or ketosis, TG ≥1000.
3. Start **organ-protective drugs** where indicated: statin, ACEi/ARB, SGLT2i, GLP-1 RA/tirzepatide.
4. Titrate to **targets**: BP <130/80, HbA1c <7%, LDL-C per risk, weight −10–15%.
5. Screen and treat **complications**: eyes, kidneys, feet, heart, liver, OSA, depression.
6. Simplify with **single-pill combinations** and once-weekly injectables; review every 1–3 months until stable.

Common targets — one-page summary

Parameter	General target	High / very high risk
BP (office)	<130/80	SBP 120–129 (or <120 if tolerated)
HbA1c	<7.0%	<6.5% if safe; <8% if frail
Fasting glucose	80–130 mg/dl	—
LDL-C	<100 mg/dl	<70 (high) / <55 (very high)
Non-HDL-C	<130 mg/dl	<100 / <85
Triglycerides	<150 mg/dl	—

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Parameter	General target	High / very high risk
Weight	≥5–10% loss	≥15% (remission, OSA, HFpEF)
Waist	<90 cm men, <80 cm women	—
UACR	<30 mg/g	≥30% reduction if albuminuric
Physical activity	≥150 min/week	200–300 min/week for weight loss
Salt	<5 g/day	—
Tobacco	None	—

Aspirin, vaccines and other preventive measures

Measure	Recommendation
Aspirin 75–100 mg	All with ASCVD; primary prevention only in selected high-risk (e.g. diabetes + multiple risk factors) with low bleeding risk; not routinely >70 years
Influenza vaccine	Yearly — reduces CV events after MI and in diabetes
Pneumococcal vaccine	PCV20 (or PCV15 → PPSV23) in diabetes, CKD, HF, age ≥50–65
Hepatitis B	Unvaccinated adults with diabetes (<60 years; ≥60 by shared decision)
Herpes zoster (recombinant)	Age ≥50 (diabetes increases risk)
COVID-19 / RSV	As per current national recommendations; RSV for ≥60 with CV/metabolic disease
Dental care	Periodontitis worsens glycaemia — 6-monthly check

Polypill and fixed-dose combinations

Combining a statin, BP-lowering drugs (± aspirin) in one pill improves adherence and reduces events in primary (TIPS-3, PolyIran) and secondary prevention (SECURE). In India, many combinations exist: statin + aspirin + ramipril + atenolol/metoprolol; atorvastatin + ezetimibe; telmisartan + amlodipine + chlorthalidone; metformin + DPP-4i + SGLT2i. Use them once doses are stable.

Important drug interactions across the four diseases

Combination	Risk	Action
ACEi/ARB + MRA/finerenone + K^+ supplements or NSAIDs	Hyperkalaemia, AKI	Check K^+ and creatinine; avoid NSAIDs
SGLT2i + loop/thiazide diuretic	Volume depletion	Reduce diuretic dose
Insulin/SU + GLP-1 RA/tirzepatide	Hypoglycaemia	Reduce insulin 10–20% or SU by 50% when starting
β-blocker + insulin/SU	Masks hypoglycaemia (except sweating)	Prefer cardioselective; educate
Thiazide + β-blocker (high doses)	New-onset diabetes	Prefer ACEi/ARB + CCB in metabolic syndrome
Simvastatin/atorvastatin + clarithromycin, azoles, amlodipine (simva >20 mg), diltiazem/verapamil	Myopathy	Limit simvastatin to 20 mg with amlodipine; use rosuvastatin/pravastatin
Gemfibrozil + any statin	Rhabdomyolysis	Use fenofibrate
Metformin + iodinated contrast (eGFR <30–60)	Lactic acidosis	Hold 48 h

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Combination	Risk	Action
Tirzepatide + oral contraceptives	Reduced absorption during dose escalation	Add barrier method for 4 weeks after start and each increase
Orlistat + levothyroxine, ciclosporin, amiodarone, fat-soluble vitamins	Reduced absorption	Separate dosing by ≥4 h
Rifampicin + SU, DPP-4i, pioglitazone, statins, CCB	Lower drug levels	Monitor; use insulin during TB therapy if needed

Improving adherence

- Simplify: once-daily drugs, SPCs/FDCs, weekly injectables; align with daily routines.
- Explain the purpose of each drug; ask about side effects and costs at every visit.
- Use generics and government schemes (Jan Aushadhi, NP-NCD free drugs) to reduce cost.
- Home BP and glucose monitoring; phone/SMS reminders; involve family and community health workers (ASHA).

Follow-up schedule for the combined clinic

Interval	Assess
Every visit (1–3 months until stable)	Weight, waist, BP, home BP/glucose logs, hypoglycaemia, side effects, adherence, lifestyle goal, feet (high risk), tobacco
Every 3 months (until at goal), then 6-monthly	HbA1c; lipids (until at goal); K^+/creatinine (on RAS blockers, MRA, diuretics)
Yearly	Lipids, eGFR, UACR, ALT/FIB-4, B12, TSH (selected), ECG, eye exam, comprehensive foot exam, CV risk recalculation, vaccination review, depression screen

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CHAPTER 30

Case-Based Learning — 16 Lifestyle Disease Scenarios

From the first OPD visit to the emergency department and back

Case 1 — Newly detected hypertension

A 42-year-old bank manager has clinic BP 148/94 and 150/96 on two visits. BMI 27, waist 96 cm, non-smoker. ECG, creatinine, K^+ normal; LDL-C 142, HbA1c 5.9%.

Question	Answer
Confirm	Home BP 7 days: mean 142/90 — sustained hypertension (masked/white-coat excluded)
Risk	Stage 2 (ACC/AHA) / hypertension ≥140/90 (ESC 2024); prediabetes, central obesity, borderline LDL — moderate–high risk
Plan	Lifestyle prescription (salt <5 g, DASH, 150 min walk, 5–7% weight loss); start **telmisartan 40 + amlodipine 5 mg** single pill
Review	2–4 weeks: home BP 128/82 → continue; recheck K^+/creatinine; statin discussion (PREVENT risk, Lp(a))

Case 2 — Resistant hypertension with low potassium

A 48-year-old woman on telmisartan 80, amlodipine 10, chlorthalidone 25 mg has home BP 152/96. K^+ 3.2 mmol/l. Adherence confirmed.

Question	Answer
Suspect	Primary aldosteronism (resistant HTN + hypokalaemia)
Test	Correct K^+; aldosterone 24 ng/dl, PRA 0.2 ng/ml/h → ARR 120 (positive)
Next	Confirmatory saline infusion test; adrenal CT (1.6-cm left adenoma); adrenal venous sampling → left lateralisation
Treatment	Laparoscopic left adrenalectomy (spironolactone 25–50 mg meanwhile); BP and K^+ normalised post-op

Case 3 — Hypertensive emergency

A 55-year-old man presents with headache, confusion and BP 230/140. Fundus: bilateral flame haemorrhages and papilloedema. Creatinine 2.1 mg/dl.

Question	Answer
Diagnosis	Malignant hypertension with encephalopathy and AKI — hypertensive emergency
Target	Reduce MAP by 20–25% over the first few hours (to ≈160/100), then normalise over 24–48 h
Drug	IV labetalol 20 mg then infusion 1–2 mg/min, or nicardipine 5 mg/h titrated; arterial line, ICU

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Question	Answer
Then	Switch to oral agents; screen for secondary causes (renovascular, phaeochromocytoma, renal disease)

Case 4 — Severe hypertension in pregnancy

A 28-year-old primigravida at 34 weeks has BP 168/112, headache, 2+ proteinuria, platelets 1.2 lakh.

Question	Answer
Diagnosis	Pre-eclampsia with severe features
BP	IV labetalol 20 mg → 40 → 80 mg every 10 min (or oral nifedipine 10 mg) to <160/110 within 1 h
Seizure prophylaxis	$MgSO_4$ Pritchard: 4 g IV + 10 g IM (5 g each buttock), then 5 g IM every 4 h — monitor reflexes, RR, urine output
Definitive	Plan delivery after stabilisation; postpartum BP monitoring; nifedipine/enalapril if breastfeeding

Case 5 — New type 2 diabetes with obesity and ASCVD

A 52-year-old man with MI 2 years ago has HbA1c 8.4%, BMI 31, eGFR 78, UACR 15, LDL-C 92 on atorvastatin 40.

Question	Answer
Cardiorenal priority	**GLP-1 RA** (semaglutide 0.25 → 1 mg weekly) — MACE and weight benefit; add **SGLT2i** (empagliflozin 10 mg)
Glycaemia	Metformin XR 500 → 2000 mg as tolerated; target HbA1c <7%
Lipids	LDL-C goal <55: increase to atorvastatin 80 + ezetimibe 10 mg
Other	BP <130/80, aspirin 75 mg continues, diet and cardiac rehabilitation, eye and foot screening

Case 6 — HbA1c 11.8% with weight loss

A 38-year-old woman has polyuria, 6-kg weight loss, glucose 386 mg/dl, HbA1c 11.8%, urine ketones trace, pH normal. BMI 22.

Question	Answer
Interpretation	Symptomatic catabolic hyperglycaemia — insulin indicated; lean young patient — exclude LADA/T1D
Tests	GAD antibodies, C-peptide (fasting 0.4 ng/ml, GAD positive → LADA)
Treatment	Basal-bolus: TDD 0.4 U/kg ≈ 22 U (glargine 11 U + aspart 3–4 U with meals); CGM; education; avoid SU
Follow-up	Titrate every 3 days; sick-day rules; never stop basal insulin

Case 7 — Starting and titrating basal insulin

A 60-year-old man on metformin 2 g, glimepiride 2 mg, sitagliptin 100 mg has HbA1c 9.2%, fasting glucose 190. Weight 70 kg.

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Question	Answer
Start	Glargine 10 U at bedtime (0.14 U/kg); continue metformin and sitagliptin; reduce glimepiride to 1 mg
Titrate	Increase 2 U every 3 days until fasting 80–130 mg/dl
At 6 weeks	Glargine 28 U (0.4 U/kg), fasting 118 but HbA1c 8.0% and post-dinner 240 → overbasalisation risk
Intensify	Replace sitagliptin with GLP-1 RA (or add aspart 4 U before dinner — basal-plus); stop glimepiride

Case 8 — Severe hypoglycaemia in an elderly patient

A 76-year-old woman with CKD (eGFR 28) on glibenclamide 10 mg is found drowsy; glucose 38 mg/dl.

Question	Answer
Immediate	25% dextrose 100 ml IV, then 10% dextrose infusion; recheck every 15–30 min
Why	Long-acting SU accumulating in CKD — hypoglycaemia may recur for 24–72 h
Additional	Octreotide 50 µg SC 8-hourly if recurrent; admit and monitor
Prevent	Stop glibenclamide; HbA1c target <8%; linagliptin 5 mg ± low-dose basal insulin; CGM if possible

Case 9 — Diabetic ketoacidosis

A 19-year-old with T1D missed insulin for 2 days. Glucose 480, pH 7.08, HCO_3^- 8, β-OHB 6.2 mmol/l, K^+ 5.6, Na^+ 131. Weight 55 kg.

Question	Answer
Severity	Severe DKA (pH <7.1, HCO_3^- <10)
Fluids	Ringer's lactate/0.9% saline 1 l in first hour, then 500 ml/h, reassess
Potassium	K^+ 5.6 → no K^+ initially; recheck in 1–2 h; add 20–40 mmol/l once <5.0
Insulin	Regular insulin 0.1 U/kg/h = 5.5 U/h; continue glargine; add 10% dextrose when glucose <250
Resolution	β-OHB <0.6, pH ≥7.3 at 14 h → SC basal-bolus, overlap 2 h; education on sick-day rules

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Case 10 — Euglycaemic DKA after surgery

A 58-year-old on dapagliflozin is vomiting on post-operative day 2. Glucose 198, pH 7.18, HCO_3^- 11, β-OHB 4.8.

Question	Answer
Diagnosis	SGLT2 inhibitor-associated euglycaemic DKA (drug not stopped 3 days pre-op)
Treatment	IV fluids, **insulin infusion with 10% dextrose from the start**, potassium as needed
Lesson	Stop SGLT2i 3 days before surgery; check ketones in unwell patients on SGLT2i even if glucose is normal

Case 11 — Diabetic kidney disease

A 63-year-old with T2D has eGFR 48, UACR 640 mg/g, BP 146/88 on amlodipine 5. HbA1c 7.6%, K^+ 4.6.

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Question	Answer
Pillar 1	Add telmisartan 40 → 80 mg; check K^+/creatinine in 1–2 weeks
Pillar 2	Dapagliflozin 10 mg (expect small eGFR dip)
Pillar 3	Finerenone 10 mg (eGFR 25–60, K^+ ≤4.8) → 20 mg at 4 weeks if K^+ ≤4.8
Pillar 4 + other	Semaglutide weekly; statin; continue metformin (reduce to ≤1000 mg/day if eGFR falls below 45); BP <130/80; refer nephrology

Case 12 — Statin-intolerant patient after MI

A 64-year-old woman post-MI had myalgia on atorvastatin 40 and rosuvastatin 20. LDL-C 128 mg/dl.

Question	Answer
Check	CK, TSH, vitamin D; drug interactions
Re-challenge	Rosuvastatin 5 mg on alternate days — tolerated
Add	Ezetimibe 10 mg → LDL-C 88; then bempedoic acid 180 mg (or PCSK9 mAb/inclisiran)
Goal	LDL-C <55 mg/dl and ≥50% reduction; recheck in 4–12 weeks

Case 13 — Familial hypercholesterolaemia

A 32-year-old man has LDL-C 268 mg/dl, Achilles tendon xanthomas; father had MI at 44.

Question	Answer
Dutch score	LDL 250–329 (5) + xanthoma (6) + family history (1) = 12 → definite FH
Treatment	Rosuvastatin 20–40 mg + ezetimibe 10 mg; PCSK9 mAb if LDL-C remains >70
Family	Cascade screening of siblings and children (lipid profile; genetic test if available)
Also	Lp(a), lifestyle, no smoking

Case 14 — Severe hypertriglyceridaemia

A 40-year-old man with uncontrolled diabetes (HbA1c 10.5%) and daily alcohol has TG 2100 mg/dl and epigastric pain; lipase 4× ULN.

Question	Answer
Diagnosis	Hypertriglyceridaemia-induced acute pancreatitis
Acute	NPO, IV fluids, IV insulin infusion with dextrose — TG fell to 600 in 72 h
Long-term	Alcohol abstinence, very-low-fat diet, insulin-based glycaemic control, fenofibrate 160 mg, omega-3 4 g; statin for CV risk once TG <500
Look for	Secondary causes (hypothyroidism, drugs), familial chylomicronaemia if recurrent

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Case 15 — Obesity with prediabetes and OSA

A 35-year-old software engineer, BMI 34, waist 108 cm, HbA1c 6.1%, BP 138/88, ALT 72, snores; AHI 28 on polysomnography.

Question	Answer
Stage	Stage 2 obesity (India 2025) / clinical obesity (OSA, MASLD, prediabetes, elevated BP)
Lifestyle	600 kcal/day deficit, high protein, 200–300 min/week activity, sleep hygiene; CPAP
Drug	Tirzepatide 2.5 → 10–15 mg weekly (or semaglutide 2.4 mg); expected 15–20% loss — OSA and ALT improve
If inadequate	BMI ≥32.5/≥30 with comorbidity after 6–12 months → discuss sleeve gastrectomy or RYGB

Case 16 — Weight regain after stopping semaglutide

A 45-year-old woman lost 16 kg on semaglutide 2.4 mg; stopped due to cost and regained 10 kg in 9 months.

Question	Answer
Explanation	Obesity is chronic; drug effect stops on withdrawal (STEP-1 extension)
Options	Restart generic semaglutide (lower cost in India since 2026) at lowest effective dose; structured lifestyle; orlistat if drugs unaffordable
Long-term	Plan indefinite therapy as for hypertension; consider metabolic surgery if criteria met

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APPENDIX A

Formulas, Normal Values and Conversions

Calculations used at the bedside

Formulas

Formula	Calculation	Use
Body mass index	Weight (kg) ÷ height (m)²	Obesity classification
Waist-to-height ratio	Waist (cm) ÷ height (cm)	>0.5 = abdominal adiposity
Ideal body weight (Devine)	Men 50 kg + 0.91 × (height cm − 152.4); women 45.5 kg + 0.91 × (height − 152.4)	Protein and drug dosing
Mean arterial pressure	DBP + (SBP − DBP) ÷ 3	Emergency BP targets
eGFR (CKD-EPI 2021)	Race-free creatinine equation (use lab/app)	CKD staging, drug dosing
Estimated average glucose	28.7 × HbA1c − 46.7 (mg/dl)	Explaining HbA1c
Glucose management indicator	3.31 + 0.02392 × mean CGM glucose (mg/dl)	CGM-derived HbA1c estimate
Insulin-to-carbohydrate ratio	500 ÷ total daily insulin	Grams of carbohydrate covered by 1 U
Correction (sensitivity) factor	1800 ÷ TDD (rapid analogue); 1500 ÷ TDD (regular)	mg/dl fall per unit
Anion gap	$Na^+ - (Cl^- + HCO_3^-)$	DKA (normal 10–12)
Effective serum osmolality	2 × Na^+ + glucose (mg/dl) ÷ 18	HHS (>300)
Corrected sodium	Na^+ + 1.6 × (glucose − 100) ÷ 100	Hyperglycaemia
Friedewald LDL-C	TC − HDL-C − TG ÷ 5 (invalid if TG >400)	Calculated LDL
Non-HDL-C	TC − HDL-C	Atherogenic cholesterol
FIB-4	(Age × AST) ÷ (platelets [10^9/l] × √ALT)	Liver fibrosis risk
HOMA-IR	Fasting insulin (µU/ml) × fasting glucose (mg/dl) ÷ 405	Insulin resistance (research)
Aldosterone–renin ratio	Plasma aldosterone (ng/dl) ÷ PRA (ng/ml/h)	Primary aldosteronism screen (>20)

Normal values and conversions

Analyte	Normal / reference	Conversion
Fasting plasma glucose	70–99 mg/dl	mg/dl ÷ 18 = mmol/l
HbA1c	<5.7%	IFCC (mmol/mol) = (HbA1c% − 2.15) × 10.929
Total cholesterol	<200 mg/dl	mg/dl ÷ 38.7 = mmol/l
Triglycerides	<150 mg/dl	mg/dl ÷ 88.5 = mmol/l
Lp(a)	<30 mg/dl	mg/dl × ≈2.15–2.5 ≈ nmol/l (assay-dependent)
Serum creatinine	0.6–1.2 mg/dl	mg/dl × 88.4 = µmol/l
UACR	<30 mg/g	mg/g ÷ 8.84 ≈ mg/mmol (30 mg/g ≈ 3 mg/mmol)
Serum potassium	3.5–5.0 mmol/l	—

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Analyte	Normal / reference	Conversion
Uric acid	Men 3.5–7.2; women 2.6–6.0 mg/dl	mg/dl × 59.5 = µmol/l
Fasting C-peptide	0.8–3.1 ng/ml	ng/ml × 0.33 = nmol/l
β-Hydroxybutyrate	<0.6 mmol/l	0.6–1.5 ketosis; 1.5–3.0 risk of DKA; >3.0 DKA
Vitamin B12	200–900 pg/ml	—
25-OH vitamin D	30–100 ng/ml	ng/ml × 2.5 = nmol/l

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APPENDIX B

Quick-Reference Cards

Pocket summaries for the clinic and the emergency department

Card 1 — Hypertension in 60 seconds

Step	Action
Measure	Correct technique; confirm with home BP/ABPM
Tests	Creatinine, Na^+/K^+, glucose/HbA1c, lipids, UACR, ECG
Start	ACEi/ARB + CCB or thiazide-like as single pill (monotherapy in low-risk, frail, ≥80)
Step up	Full dose → A + C + D → spironolactone → specialist
Target	<130/80 (SBP 120–129 if tolerated)
Emergency	Organ damage → IV labetalol/nicardipine; MAP ↓ ≤25% in 1st hour

Card 2 — Type 2 diabetes in 60 seconds

Step	Action
Diagnose	FPG ≥126, 2-h ≥200, HbA1c ≥6.5% (confirm), random ≥200 + symptoms
Organ protection	ASCVD → GLP-1 RA/SGLT2i; HF → SGLT2i; CKD → SGLT2i + RASi + finerenone + semaglutide
Glycaemia	Metformin + early combination; intensify every 3 months
Insulin	HbA1c >10%, glucose ≥300, catabolic → basal 10 U (0.1–0.2 U/kg), +2 U every 3 days
Targets	HbA1c <7%, TIR >70%, BP <130/80, LDL-C <70/<55
Annual	Eyes, feet, UACR/eGFR, lipids, ALT/FIB-4, B12, vaccines

Card 3 — Insulin at a glance

Insulin	Onset	Peak	Duration	Use
Lispro / aspart / glulisine	10–15 min	1–2 h	3–5 h	Meal bolus, correction
Regular	30–60 min	2–4 h	6–8 h	Meal bolus, IV infusion
NPH	1–2 h	4–8 h	12–16 h	Basal (bedtime/BD), steroid cover
Glargine U100	1–2 h	Flat	24 h	Basal once daily
Glargine U300 / degludec	1–6 h	Flat	36–42 h	Basal, less hypoglycaemia
Icodec	—	Flat	1 week	Weekly basal
Premix 30/70	Per component	Dual	12–24 h	BD before breakfast and dinner

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Card 4 — Hypoglycaemia and DKA

Emergency	Action
Hypoglycaemia, conscious	15–20 g glucose → recheck 15 min → snack
Hypoglycaemia, unconscious	25% dextrose 100 ml IV or glucagon 1 mg IM; 10% dextrose infusion
DKA	Fluids 1–1.5 l/h → K^+ (hold insulin if <3.5) → insulin 0.1 U/kg/h → dextrose at <250 → resolve (β-OHB <0.6, pH ≥7.3) → SC basal 2 h overlap
HHS	Slow fluids; insulin 0.05 U/kg/h only once glucose stops falling; LMWH

Card 5 — Lipids in 60 seconds

Step	Action
Measure	Non-fasting lipid profile; Lp(a) once; apoB if TG high
Risk → goal	Very high <55; high <70; moderate <100 (and ≥50% ↓ in high/very high)
Ladder	High-intensity statin → + ezetimibe → + PCSK9 mAb / inclisiran / bempedoic acid
TG ≥500	Lifestyle, alcohol stop, glucose control, fenofibrate, omega-3
TG 150–499 on statin, high risk	Icosapent ethyl 2 g BD

Card 6 — Obesity in 60 seconds

Step	Action
Diagnose	BMI >23 (Indians) + waist ≥90/80 or WHtR >0.5; stage by complications
Lifestyle	500–750 kcal deficit, protein 1–1.5 g/kg, 200–300 min/week activity
Drugs	Tirzepatide 2.5 → 15 mg weekly; semaglutide 0.25 → 2.4 mg weekly; liraglutide 3 mg; orlistat 120 mg TDS
Review	<5% loss at 12–16 weeks on maintenance dose → change strategy
Surgery	BMI ≥35; ≥30 (≥27.5 Asians) with uncontrolled comorbidity

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APPENDIX C

Abbreviations, Guidelines and Key Trials

Terms used in this book and the evidence behind the practice

Abbreviations

Abbreviation	Meaning	Abbreviation	Meaning
ABPM	Ambulatory BP monitoring	HHS	Hyperglycaemic hyperosmolar state
ACEi	Angiotensin-converting enzyme inhibitor	HMOD	Hypertension-mediated organ damage
AID	Automated insulin delivery	ICR	Insulin-to-carbohydrate ratio
AOM	Anti-obesity medication	IFG / IGT	Impaired fasting glucose / glucose tolerance
apoB	Apolipoprotein B	LADA	Latent autoimmune diabetes in adults
ARB	Angiotensin receptor blocker	Lp(a)	Lipoprotein(a)
ARNI	Angiotensin receptor–neprilysin inhibitor	MASLD / MASH	Metabolic dysfunction-associated steatotic liver disease / steatohepatitis
ARR	Aldosterone–renin ratio	MNT	Medical nutrition therapy
ASCVD	Atherosclerotic cardiovascular disease	MODY	Maturity-onset diabetes of the young
β-OHB	β-hydroxybutyrate	MRA	Mineralocorticoid receptor antagonist
CAC	Coronary artery calcium	NPH	Neutral protamine Hagedorn insulin
CCB	Calcium-channel blocker	OGTT	Oral glucose tolerance test
CGM	Continuous glucose monitoring	OSA	Obstructive sleep apnoea
CKM	Cardiovascular–kidney–metabolic	PCSK9	Proprotein convertase subtilisin/kexin 9
DKA	Diabetic ketoacidosis	RAS	Renin–angiotensin system
DPP-4i	Dipeptidyl peptidase-4 inhibitor	SGLT2i	Sodium–glucose cotransporter-2 inhibitor
DSMES	Diabetes self-management education and support	SMBG	Self-monitoring of blood glucose
eGFR	Estimated glomerular filtration rate	SPC / FDC	Single-pill / fixed-dose combination
FCPD	Fibrocalculous pancreatic diabetes	SU	Sulfonylurea
FH	Familial hypercholesterolaemia	TDD	Total daily (insulin) dose
GDM	Gestational diabetes mellitus	TIR	Time in range
GLP-1 RA	Glucagon-like peptide-1 receptor agonist	UACR	Urine albumin–creatinine ratio
HBPM	Home BP monitoring	WHtR	Waist-to-height ratio

International and Indian guidelines cited

Area	Guideline / source
Hypertension	2025 AHA/ACC/multisociety Guideline for the Prevention, Detection, Evaluation and Management of High Blood Pressure; 2024 ESC Guidelines for Elevated BP and Hypertension; ISH 2020 Global Hypertension Practice Guidelines; Indian Guidelines on Hypertension (IGH-IV, 2019); India Hypertension Control Initiative / NP-NCD protocols; Endocrine Society 2025 Primary Aldosteronism Guideline; ACOG and ISSHP guidance on hypertension in pregnancy; MoHFW (India) guidance on $MgSO_4$

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Area	Guideline / source
Diabetes	ADA Standards of Care in Diabetes 2026; ADA/EASD consensus on management of hyperglycaemia in T2D; ADA/EASD/JBDS/AACE/DTS 2024 consensus on hyperglycaemic crises; RSSDI Clinical Practice Recommendations 2022; ICMR Guidelines for Management of T2D; MoHFW (India) GDM guidelines 2018 (DIPSI); IADPSG/WHO 2013; International Consensus on Time in Range; IWGDF 2023 diabetic foot guidelines; KDIGO 2022 Diabetes in CKD; IDF-DAR Ramadan guidelines
Dyslipidaemia	2019 ESC/EAS Dyslipidaemia Guidelines and 2025 Focused Update; 2026 ACC/AHA/multisociety Dyslipidemia Guideline; Cardiological Society of India Dyslipidaemia Guidelines 2024; Lipid Association of India 2023 consensus; NLA scientific statements
Obesity	Lancet Diabetes & Endocrinology Commission on Clinical Obesity (2025); Revised definition of obesity in Asian Indians (Lancet Diabetes Endocrinol 2025); WHO 2025 guideline on GLP-1 therapies for obesity; AACE and Obesity Society guidance; ASMBS/IFSO 2022 indications for metabolic–bariatric surgery; EASO framework
Prevention	AHA PREVENT equations (2023) and CKM syndrome advisory; ESC SCORE2/SCORE2-OP/SCORE2-Diabetes; WHO/ISH risk charts (2019); ICMR–NIN Dietary Guidelines for Indians (2024); WHO salt and sugar guidance

Landmark trials

Trial (year)	Finding
SPRINT (2015)	SBP target <120 vs <140: CV events ↓25%, mortality ↓27%
STEP (2021), ESPRIT (2024)	Intensive SBP targets beneficial in elderly Chinese and high-risk patients
PATHWAY-2 (2015)	Spironolactone best 4th drug in resistant hypertension
ACCOMPLISH (2008)	ACEi + amlodipine superior to ACEi + HCTZ
CHAP (2022)	Treating mild chronic hypertension in pregnancy (≥140/90) improves outcomes
UKPDS (1998) / legacy effect	Early intensive glycaemic control reduces microvascular and long-term macrovascular events
EMPA-REG, CANVAS, DECLARE (2015–19)	SGLT2i reduce HF hospitalisation; MACE ↓ (empa, cana)
DAPA-CKD, EMPA-KIDNEY, CREDENCE	SGLT2i slow CKD progression
LEADER, SUSTAIN-6, REWIND (2016–19)	GLP-1 RA reduce MACE
FLOW (2024)	Semaglutide reduced kidney outcomes in T2D with CKD
SELECT (2023)	Semaglutide 2.4 mg: MACE ↓20% in obesity without diabetes
SURMOUNT-1 (2022)	Tirzepatide: ≈21% weight loss at 72 weeks
FIDELIO/FIGARO-DKD (2020–21)	Finerenone reduced kidney and CV events in T2D with CKD
DiRECT (2018)	Weight-loss programme: diabetes remission in 46% at 1 year
VERIFY (2019)	Early metformin + vildagliptin more durable than sequential therapy
CTT meta-analysis (2010)	Each 1 mmol/l LDL-C ↓ → 22% fewer major vascular events
IMPROVE-IT (2015)	Ezetimibe + simvastatin reduced events after ACS
FOURIER (2017), ODYSSEY OUTCOMES (2018)	PCSK9 mAbs reduced MACE
CLEAR Outcomes (2023)	Bempedoic acid reduced MACE in statin-intolerant patients
REDUCE-IT (2019)	Icosapent ethyl reduced MACE in high-TG patients on statins
Look AHEAD (2013)	Intensive lifestyle: weight loss and many benefits, no CV event reduction
SOS study, STAMPEDE	Bariatric surgery reduced mortality and induced diabetes remission

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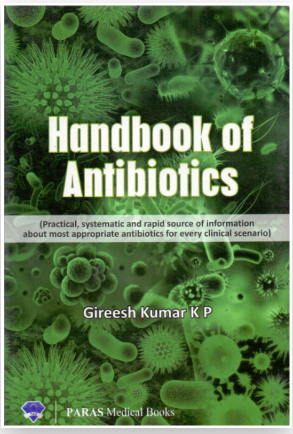
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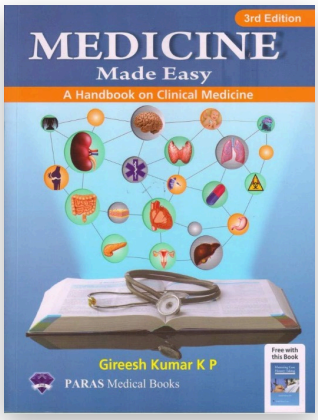
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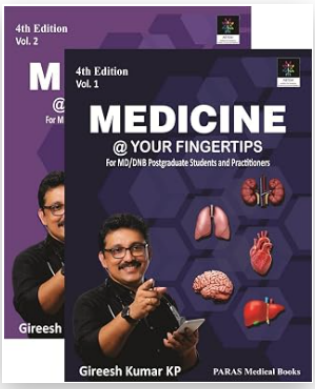
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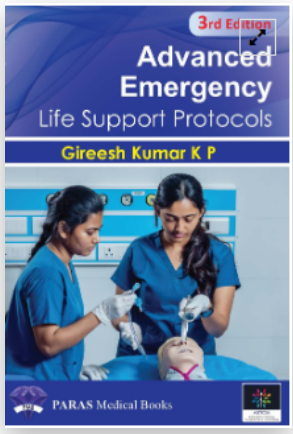
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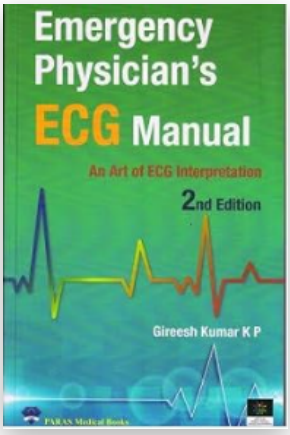
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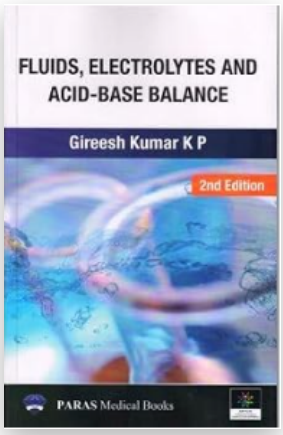
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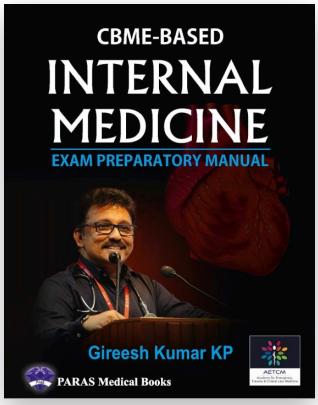
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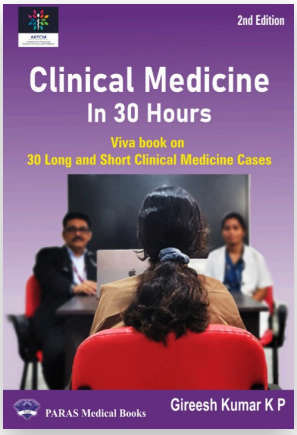
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