



Hypertension — Outpatient

2025 ACC/AHA · Risk now decides who gets a drug, not the blood pressure number alone

TARGET < 130/80
for essentially all adults

WHAT CHANGED IN 2025 *the thresholds held; the decision rule moved*

PREVENT REPLACES THE POOLED COHORT EQUATIONS

Risk is now estimated with **PREVENT**, which removes **race as an input** and instead incorporates **kidney and metabolic measures** alongside the cardiovascular ones. It estimates **10-year total cardiovascular risk**, and the number it produces now feeds directly into the decision to start a drug.

RISK-BASED INITIATION

A **10-year PREVENT risk of 7.5% or more** pushes a patient with stage 1 hypertension straight to drug therapy rather than to lifestyle alone. Two people with identical readings can now correctly get different plans.

ALSO NEW

Systolic below 130 carries a Class 1 recommendation to reduce cognitive impairment and dementia — a genuinely new argument for control. Plus a stronger push for **single-pill combinations, home monitoring, team-based care**, and expanded guidance in **pregnancy, chronic kidney disease and resistant hypertension**.

MEASURE IT PROPERLY, THEN CLASSIFY *bad technique manufactures hypertension*

Step	Detail
Technique	Seated and rested 5 minutes, back supported, feet flat, arm at heart level, correct cuff size, no talking. A cuff that is too small, an unsupported arm or a hurried reading each add clinically meaningful millimetres. Average two or more readings on two or more occasions before labelling anyone.
Confirm out of office	Home or ambulatory readings should confirm the diagnosis. This identifies white-coat hypertension , which does not need drugs, and masked hypertension , which is normal in clinic yet raised at home and carries real risk — the one you will otherwise miss entirely.
The categories	Normal below 120/80 · Elevated 120–129 systolic with diastolic below 80 · Stage 1: 130–139 systolic or 80–89 diastolic · Stage 2: 140 or above systolic, or 90 or above diastolic. The category is set by whichever number is higher.
Baseline workup	Electrolytes and creatinine, urine albumin-to-creatinine ratio, lipids, HbA1c, thyroid function, complete blood count and an ECG. Look for end-organ damage, which raises the urgency independently of the reading.

WHO GETS A DRUG, AND WHEN *the 2025 decision tree in four steps*

- Everybody gets lifestyle therapy**, at every stage, including those already on drugs. **Sodium below 2,300 mg a day and ideally below 1,500**, a DASH-style diet, **potassium-based salt substitutes** where renal function permits, weight loss, regular aerobic activity, and **alcohol reduced to at most 2 drinks a day for men and 1 for women, or abstinence**.
- Stage 2 (140/90 or above): start drug therapy now**, alongside lifestyle change. **Begin with a single-pill combination of two classes** rather than sequential single agents — it controls pressure faster and improves adherence.
- Stage 1 (130–139/80–89): the risk decides. Start a drug if there is established cardiovascular disease, diabetes, chronic kidney disease, or a 10-year PREVENT risk of 7.5% or more.** Otherwise **trial lifestyle change for 3 to 6 months**, then start a drug if the pressure remains at or above 130/80.
- Choose from the first-line classes: a thiazide-type diuretic, an ACE inhibitor or ARB, or a dihydropyridine calcium channel blocker. Never combine an ACE inhibitor with an ARB.** Beta-blockers are **not first-line** without a separate indication such as ischemic heart disease, heart failure or rate control. **Reassess monthly until controlled**, then at longer intervals.

WHEN CONTROL FAILS *most apparent resistance is not resistant hypertension*

Check	Detail
Define it first	Resistant hypertension is blood pressure above goal despite three agents of different classes including a diuretic at optimal doses, or controlled pressure requiring four or more. Confirm with out-of-office readings before accepting the label.
Non-adherence	The single commonest reason a regimen appears to fail. Ask non-judgementally about missed doses, cost and side effects, and simplify to once-daily single-pill combinations . Poor technique and an undersized cuff are the next commonest.
Interfering substances	NSAIDs, decongestants, oral contraceptives, corticosteroids, cyclosporine, erythropoietin, stimulants, excess alcohol and licorice. Review the full list including anything bought over the counter.
Add spironolactone	The preferred fourth agent , supported by PATHWAY-2, where it outperformed the alternatives added to triple therapy. Monitor potassium and creatinine within 1 to 2 weeks , and be cautious in chronic kidney disease.
Look for a secondary cause	Primary aldosteronism is the commonest and is markedly underdiagnosed — screen with an aldosterone-to-renin ratio in resistant hypertension, unexplained hypokalemia, an adrenal incidentaloma, or hypertension with sleep apnea or atrial fibrillation. Also consider obstructive sleep apnea, renal artery stenosis, renal parenchymal disease, pheochromocytoma, Cushing's syndrome, coarctation and thyroid disease .

SPECIAL SITUATIONS *where the standard regimen is wrong*

PREGNANCY

ACE inhibitors, ARBs and direct renin inhibitors are contraindicated because of fetal renal and skeletal harm. **Use labetalol, nifedipine or methyldopa**, and stop the offending agent as soon as pregnancy is recognized or planned. **Discuss contraception and pregnancy intention before prescribing** to anyone who could conceive — this conversation is routinely skipped.

CKD AND DIABETES

Use an ACE inhibitor or ARB when there is albuminuria — the benefit is on kidney outcomes beyond the pressure itself. **Expect and tolerate a creatinine rise of up to about 30%** after starting; a larger rise or hyperkalemia prompts review rather than reflexive discontinuation. **Check renal function and potassium 1 to 2 weeks after every initiation or uptitration.**

PEARLS & PITFALLS

- Confirm with out-of-office readings before committing to lifelong treatment** — and to catch masked hypertension, which clinic readings hide.
- Run PREVENT before deciding on stage 1.** Under the 2025 guideline the risk score, not the reading, is what triggers drug therapy.
- Start stage 2 on two drugs in one pill.** Sequential single agents control pressure more slowly and adherence is worse.
- Non-adherence, not true resistance, is the leading cause of uncontrolled pressure.** Ask before you add a fourth drug.
- Screen for primary aldosteronism** in resistant hypertension or unexplained hypokalemia — it is common and specifically treatable.
- Stop ACE inhibitors and ARBs before or as soon as pregnancy occurs**, and raise the issue proactively rather than at the first positive test.





Outpatient Type 2 Diabetes

ADA Standards of Care 2026 · targets, drug selection driven by comorbidity, and the annual screening set

Organ protection
is not glycemia

TARGETS *individualized, and the number is not the same for everyone*

GLYCEMIA

A1c < 7% for most non-pregnant adults. **< 8%** is the safer target with limited life expectancy, advanced complications, extensive comorbidity or hypoglycemia unawareness. **A tighter number in a frail patient causes harm**, it does not earn credit.

BLOOD PRESSURE

< 130/80 on treatment. **SBP < 120 is encouraged when cardiovascular or kidney risk is high**, if it can be reached safely. **< 140/90** for older adults in poor health or with limited life expectancy.

LIPIDS

Statin intensity by risk, not by LDL alone. **Do not add a fibrate, niacin or fish oil to a statin for cardiovascular risk reduction**: they add no further benefit. Fibrate remains appropriate for **triglycerides above 500** to prevent pancreatitis.

DIAGNOSIS, AND WHEN THE A1c LIES *confirm with a repeat test unless the patient is unequivocally hyperglycemic*

DIAGNOSTIC THRESHOLDS

A1c ≥ 6.5% · **fasting glucose ≥ 126** (8 h fast) · **2-h glucose ≥ 200** on 75 g OGTT · **random ≥ 200 with classic symptoms** or hyperglycemic crisis.

PREDIABETES

A1c 5.7 to 6.4% · fasting **100 to 125** · 2-h **140 to 199**. Not a benign label: intensive lifestyle change, and **metformin is reasonable** particularly under age 60, with BMI ≥ 35, or after gestational diabetes.

WHEN NOT TO TRUST THE A1c

It assumes a normal red cell lifespan. **Hemoglobinopathies, recent transfusion or blood loss, hemolysis, iron deficiency, erythropoietin, advanced CKD, dialysis and pregnancy all distort it**. Use fasting glucose, OGTT or CGM instead.

DRUG SELECTION *the single most important idea on this page*

Situation	What to use	Why
Everyone at diagnosis	Lifestyle plus metformin . Metformin remains first-line for glycemic control	Effective, cheap, weight-neutral, no hypoglycemia alone. ADA 2026 did not remove it , despite what you may hear
ASCVD, or high cardiovascular risk	GLP-1 RA with proven benefit, <i>or</i> SGLT2i with proven benefit	These are indication-driven, not A1c-driven . Start them for the organ indication independent of the A1c and independent of whether the patient is on metformin . ADA 2026 pushes them earlier, considered from the point of diagnosis in high-risk patients, because they are disease-modifying rather than glucose-lowering
Heart failure, any ejection fraction	SGLT2i	
CKD (reduced eGFR or albuminuria)	SGLT2i preferred; GLP-1 RA if SGLT2i not tolerated. Add finerenone for residual albuminuria	
Obesity is the dominant problem	GLP-1 RA or dual GIP/GLP-1	Greatest weight effect, and weight loss drives blood pressure, lipids and hepatic steatosis at the same time
Cost is the barrier	Metformin, sulfonylurea , pioglitazone, human insulin	A cheap regimen taken beats an ideal regimen unfilled. Counsel on hypoglycemia with sulfonylureas and on weight gain and fluid retention with pioglitazone
A1c far above goal , or catabolic (weight loss, ketones)	Insulin , plus evaluate for type 1 or pancreatic diabetes	Do not titrate oral agents slowly in a patient who is losing weight and spilling ketones

Reconciling two 2026 guidelines that sound contradictory. ADA keeps metformin first-line for glycemia. The 2026 CKM guideline, in a staged CKM population with PREVENT 10-year risk at or above 7.5%, makes the cardioprotective agent the Class 1 step and positions metformin as the Class 2a add-on for glycemic control. **Both are saying the same thing from different starting points**: the organ-protective drug is chosen for the organ, and metformin is chosen for the glucose.

THE ANNUAL SET *what gets missed when the visit is only about the A1c*

Check	How often	What it is for
eGFR and UACR	At least annually , both	UACR is the one most often skipped . Albuminuria appears before eGFR falls and independently predicts kidney and cardiovascular events. ACEi or ARB is strongly recommended at UACR ≥ 300 , and suggested for hypertension with CAD or UACR 30 to 299
Dilated eye exam	Annually. Every 1 to 2 years if no retinopathy and glycemia at goal	Retinopathy is asymptomatic until it is not. In T2DM, start screening at diagnosis , because the disease predates the diagnosis
Comprehensive foot exam	At least annually , more often if high risk	Identifies the neuropathy, deformity and vascular disease that precede ulceration and amputation. Inspect footwear at the same visit
Lipids, blood pressure, weight	Every visit or annually	The cardiovascular risk is what most of these patients die of, not the hyperglycemia itself
Vaccines, mental health, MASLD	Annually	Influenza, pneumococcal, hepatitis B, COVID-19 and RSV per schedule. Screen for depression and diabetes distress , and calculate FIB-4 for liver fibrosis

Starting insulin: basal **10 units daily**, or **0.1 to 0.2 units/kg/day**, titrated against the **fasting** glucose every few days. Add prandial insulin only once basal is optimized and the fasting number is at goal, otherwise you are treating the wrong meal. **On CGM, aim for time in range above 70% with under 4% of readings below 70 mg/dL**; a good time in range with frequent lows is not good control.

PITFALLS & SAFETY

- SGLT2i: euglycemic DKA is the one to know**. Ketoacidosis with a near-normal glucose, so it is missed unless ketones are checked. **Hold during acute illness, prolonged fasting and before surgery**. Also counsel on genital mycotic infections and volume depletion.
- Expect an early eGFR dip after starting an SGLT2i and do not stop for it**. It is hemodynamic and precedes the long-term kidney benefit. Stopping the drug at the dip is the commonest way this benefit is lost.
- Metformin is a lactic acidosis question, not a kidney question**. Continue to **eGFR 30**, do not start below 45, and hold around iodinated contrast and acute illness. Check **B12** on long-term use.
- Do not stack incretins**. A GLP-1 RA plus a DPP-4 inhibitor adds no benefit; stop the DPP-4 inhibitor when starting a GLP-1. Reduce sulfonylurea or insulin when adding a GLP-1 or SGLT2i to avoid hypoglycemia.





Outpatient Lipid Management

2026 ACC/AHA Multisociety Dyslipidemia Guideline · PREVENT replaces the pooled cohort equations

Earlier & lower
for longer

STEP 1 · THE CPR FRAMEWORK *the guideline's own model for primary prevention*

C — CALCULATE

PREVENT-ASCVD, ages 30-79, LDL 70-189. Bands: **low** < 3% · **borderline** 3-<5% · **intermediate** 5-<10% · **high** ≥ 10%. Race dropped as an input; CKD, HbA1c and BMI added; reports **10- and 30-year** risk.

P — PERSONALIZE

Apply **risk enhancers** PREVENT misses: family history of premature ASCVD, LDL ≥ 160, metabolic syndrome, CKD, elevated Lp(a), South Asian ancestry, **inflammatory disease** (RA, lupus, psoriasis, HIV) and **reproductive history** (preeclampsia, early menopause, gestational diabetes).

R — RECLASSIFY & REASSESS

When the decision is still uncertain, **CAC scoring** reclassifies. Then reassess the treatment plan. **Age 30-59, low risk, LDL < 160 and 30-year risk < 10%: lifestyle counseling alone** is the Class 1 answer.

STEP 2 · GOALS ARE BACK *LDL and non-HDL-C, plus a percent reduction*

Risk category	LDL-C goal	non-HDL-C goal	Also
Very-high-risk ASCVD Class 1	< 55	< 85	High-intensity statin, ≥ 50% LDL reduction. Most patients with ASCVD land here.
ASCVD, not very high risk	< 70	< 100	A minority of ASCVD patients.
Primary prevention, high (≥ 10%)	< 70	< 100	High-intensity: atorvastatin 40-80, rosuvastatin 20-40 (≥ 50% reduction).
Borderline 3-<5% or intermediate 5-<10%	< 100	< 130	Moderate: atorvastatin 10-20, rosuvastatin 5-10 (30-49%). Intermediate: should be considered; borderline: may be, after discussion. Use CAC if undecided.

Very high risk = multiple major ASCVD events, or one major event plus multiple high-risk conditions (age > 65, prior revascularization, smoking, diabetes, heart failure, hypertension, LDL > 100 on maximal statin + ezetimibe).

STEP 3 · WHO GETS THERAPY *and at what intensity*

Indication	Intensity and notes
Clinical ASCVD	High-intensity. Add ezetimibe, then a PCSK9i, to reach goal. Combination is the expectation, not the exception.
LDL ≥ 190	High-intensity. Treat as familial hypercholesterolemia until proven otherwise; no risk calculation needed.
Diabetes, CKD stage 3-4, or HIV age 40-75	Recommended regardless of LDL level. After 75, therapy <i>can be considered</i> alongside lifestyle.
Early intervention NEW	HeFH at diagnosis (including children 8-10), young adults with LDL ≥ 160 , or strong family history of premature ASCVD. Exists because 10-year risk is always low in the young and systematically under-treats them.

CAC · NOW A CLASS 1 TOOL *upgraded from 2a in 2018*

WHO TO SCAN

Men ≥ 40, women ≥ 45, at **intermediate** risk or **selected borderline** risk, when the treatment decision remains uncertain. Both the **absolute score and the age/sex/race percentile** carry prognostic weight.

HOW IT CHANGES THE PLAN

CAC 0 supports withholding or postponing therapy in a low- or intermediate-risk patient with no other comorbidity. **Any calcium argues for treating**, and a higher score argues for treating harder. **Incidental calcium on a non-gated CT now counts**, so do not ignore “coronary calcifications” on a chest CT report.

ORDER ONCE, AND ONCE ONLY *Lp(a), plus apoB when LDL misleads*

Test	When, and what the number means
Lp(a) Class 1, all adults	Measure at least once in every adult. Genetically fixed, so one result settles it. ≥ 125 nmol/L (50 mg/dL) is a risk enhancer, roughly 1.4× risk; ≥ 250 nmol/L (100 mg/dL) is ≥ 2×. It has no drug of its own yet: it intensifies LDL lowering and risk-factor control. Cascade test first-degree relatives. In ASCVD with high Lp(a) not at goal on maximal statin, add a PCSK9 mAb (Class 1).
apoB	Useful once LDL and non-HDL goals are met , and especially when TG ≥ 150 , in diabetes , or when achieved LDL < 70 . In those settings LDL-C understates particle number, so a patient “at goal” can still carry residual risk.

SPECIAL SITUATIONS *the ones that change the drug*

Situation	What to do, and why
TG ≥ 500	Fenofibrate to prevent pancreatitis. Never gemfibrozil with a statin (rhabdomyolysis). At ≥ 1000 the urgency is pancreatitis, not cardiovascular risk. Statin remains the foundation for ASCVD risk in high TG.
ASCVD, TG 150-499	Icosapent ethyl (REDUCE-IT). Pure EPA, not interchangeable with OTC fish oil. PROMINENT closed the door on fibrates for event reduction.
“Statin intolerant”	Mostly placebo: SAMSON found identical symptoms on placebo. Rechallenge with a different agent or alternate-day dosing before accepting the label , which otherwise costs decades of protection. True intolerance: bempedoic acid, ezetimibe, PCSK9i.
Pregnancy	Stop statins, ezetimibe, fibrates, niacin and PCSK9 inhibitors. Bile acid sequestrants only, as they are not absorbed. Discuss contraception before starting a statin.
Dose caps	eGFR < 30: rosuvastatin max 10 mg. Asian ancestry: start rosuvastatin 5 mg. HIV: pitavastatin , minimal antiretroviral interaction.

MONITORING & PITFALLS

- **Recheck lipids 4-12 weeks after starting or changing**, then every 3-12 months. Not at goal is the trigger to add a second agent, not to wait another year.
- **Do not monitor LFTs or CK routinely.** Baseline ALT, then check CK **only if muscle symptoms** appear.
- **Exclude secondary causes before labeling primary dyslipidemia:** hypothyroidism, nephrotic syndrome, cholestasis, uncontrolled diabetes, alcohol, and drugs (thiazides, steroids, retinoids, antipsychotics).
- **Intensity is percent reduction, not milligrams.** Each dose doubling adds only about 6%.





Preventive Care & Screening

USPSTF adult screening · the ages that changed recently, and the ones people still quote wrong

Ages moved
check yours

CANCER SCREENING *three of these ages have changed, so check what you were taught*

Cancer	Who and how often	Notes
Colorectal	Start at 45, continue to 75. Colonoscopy every 10 years, or annual FIT, or other approved strategies	CHANGED Starting age moved from 50 to 45. Ages 45 to 49 is a Grade B recommendation and 50 to 75 is Grade A. 76 to 85 is selective , based on health, prior screening and preference. The best test is the one that gets done: a completed FIT beats a declined colonoscopy, but any positive non-colonoscopy test needs a colonoscopy
Breast	Biennial mammography from 40 to 74	CHANGED 2024 The starting age moved from 50 to 40 , and it is now a straightforward Grade B rather than an individualized decision in the 40s. Evidence is insufficient at 75 and older , and also insufficient on supplemental ultrasound or MRI for dense breasts on an otherwise negative mammogram
Cervical	21 to 29: cytology every 3 years. 30 to 65: HPV primary screening every 5 years , clinician- or patient-collected	NEW Self-collection is now included for HPV primary screening in the 30 to 65 group. Alternatives in that group remain cytology every 3 years or co-testing every 5 years. Do not start before 21 regardless of sexual history
Lung	Annual low-dose CT, ages 50 to 80 , with a 20 pack-year history, who currently smoke or quit within the past 15 years	CHANGED Age dropped from 55 to 50 and the threshold from 30 to 20 pack-years. Stop screening once 15 years have passed since quitting , or if health would not permit curative treatment. Pair it with cessation support, which is where most of the benefit sits
Prostate	Shared decision making, ages 55 to 69. Do not screen routinely at 70 and over	An individual decision rather than a default. Discuss overdiagnosis and the consequences of biopsy and treatment before ordering the PSA

CARDIOVASCULAR, METABOLIC & BONE *the ones most often skipped in a busy clinic*

What	Who
Blood pressure	All adults 18 and over, with out-of-office confirmation before diagnosing hypertension
Lipids and cardiovascular risk	Risk-based, using a validated calculator. Statins for primary prevention in adults with risk factors and calculated risk above the treatment threshold
Diabetes and prediabetes	Screen adults 35 to 70 who are overweight or obese , and repeat at least every 3 years if normal
Abdominal aortic aneurysm	One-time ultrasound in men 65 to 75 who have ever smoked. Easy to forget because it is once only and needs the smoking history
Osteoporosis	Women 65 and over , and younger postmenopausal women at increased risk by a formal tool. Men are not routinely screened , though those on long-term steroids or with prior fragility fracture warrant assessment
Obesity and unhealthy alcohol and drug use	Screen all adults. Offer behavioral intervention for obesity, and brief intervention for unhealthy alcohol use

INFECTION SCREENING *the three universal ones people still treat as risk-based*

HIV: 15 TO 65, EVERYONE Screen every adult at least once, not only those with risk factors, plus younger and older patients at increased risk, and all pregnant patients . Risk-based screening alone misses a large share of infections, which is exactly why the recommendation is universal.	HEPATITIS C: ALL ADULTS 18 TO 79 At least once in every adult, with periodic testing for ongoing risk. Curative therapy is short and highly effective, so finding it is the hard part , not treating it.	HEPATITIS B AND STIs Screen all adults at least once for hepatitis B, and all pregnant patients. Screen for syphilis, gonorrhea and chlamydia per risk and age, and latent tuberculosis in higher-risk populations. Offer PrEP to anyone at increased HIV risk.
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ADULT IMMUNIZATION *the preventive care most often left off the problem list*

Vaccine	Who
Influenza	Annually, everyone 6 months and older. Higher-dose or adjuvanted formulations are preferred at 65 and over
COVID-19	Per the current season's schedule, with additional doses for older and immunocompromised adults
Pneumococcal	All adults 50 and over , and younger adults with risk conditions including chronic heart, lung, liver or kidney disease, diabetes, smoking, immunocompromise, cochlear implant or CSF leak. Asplenia needs a specific schedule , so check it rather than assuming
Recombinant zoster	Two doses for all adults 50 and over , and for immunocompromised adults from 19 . Non-live, so it is safe in immunosuppression
RSV	Adults 75 and over , and 50 to 74 with risk factors , per the current schedule
Tdap and HPV	Td or Tdap booster every 10 years , with Tdap in every pregnancy. HPV routinely through 26 , and shared decision making from 27 to 45

Before any immunosuppressant or biologic, bring vaccines up to date and give live vaccines first, since they are contraindicated afterward. **Asplenia, transplant and complement inhibitor therapy each carry their own schedules**, and these are the patients in whom a missed vaccine does the most harm.

PEARLS & PITFALLS

- **Recommendations change, and the retired numbers are the ones still being quoted.** Colorectal at 50, breast at 50, and lung at 55 with 30 pack-years are all out of date. **Check the current USPSTF statement rather than trusting memory or an old note template**, since the ages move more often than the diseases do.
- **Screening is not automatically good.** Grades matter: **A and B are recommended, C is selective and individualized, D means recommend against, and I means insufficient evidence.** A Grade D such as PSA in men 70 and over is an instruction not to screen, not an invitation to shared decision making.
- **Stop screening when it can no longer help.** Consider **life expectancy and whether the patient would accept treatment** before ordering a test, because screening only benefits someone who will live long enough to reach the outcome it prevents. Continuing mammography or colonoscopy in advanced frailty causes harm without benefit.
- **Do not forget the non-test items:** immunizations per schedule, **tobacco cessation**, which outperforms almost everything else on this sheet, intimate partner violence screening, depression and anxiety screening, and falls prevention in older adults. **Also check that a positive screen was actually acted upon**, since the commonest failure is a result nobody followed up.





Obesity Management & GLP-1 Agents

Who qualifies, how to titrate, what to monitor · and the two guideline reversals residents still get wrong

Treat the disease
not the number

WHO QUALIFIES *and what each tier actually buys*

LIFESTYLE, FOR EVERYONE

Target **5% to 10%** of baseline weight. That range is where blood pressure, glycemia, triglycerides and hepatic steatosis reliably improve, **even if the patient never leaves the obese BMI range**. Counsel on it at least annually.

PHARMACOTHERAPY

BMI ≥ 30, or **BMI ≥ 27 with a weight-related comorbidity** (T2DM, hypertension, dyslipidemia, OSA, MASLD). Add to lifestyle, never instead of it.

METABOLIC / BARIATRIC SURGERY

BMI > 35 regardless of comorbidity, and **consider at BMI 30 to 34.9 with metabolic disease** (ASMBS/IFSO 2022). **This replaced the NIH 1991 criteria** of BMI 40, or 35 with a comorbidity, which are still widely quoted.

BEFORE YOU PRESCRIBE *three things that change the plan, all easy to skip*

EXCLUDE SECONDARY CAUSES

Hypothyroidism (TSH), **Cushing's syndrome** (proximal weakness, striae, easy bruising, new diabetes and hypertension together), **hypothalamic injury**, and **PCOS**. Rare as a whole, but each is treatable and none responds to a GLP-1.

SWAP THE OBESOGENIC DRUG FIRST

Review the list before adding one: **antipsychotics** (olanzapine, clozapine, quetiapine), **insulin and sulfonylureas**, **glucocorticoids**, **mirtazapine** and some **SSRIs**, **gabapentin** and **pregabalin**, **beta-blockers**. **Changing the culprit is cheaper and faster than treating the consequence.**

SCREEN THE TRAVELLING COMPANIONS

OSA (STOP-BANG), **MASLD** (FIB-4), **T2DM** or **prediabetes**, lipids, blood pressure, and **depression**. These are the conditions the weight is driving, and treating them is much of the benefit.

THE AGENTS *titration is slow on purpose: it is how you keep the patient on the drug*

Drug	Dosing and titration	Mean weight loss	Notes
Tirzepatide (Zepbound) GIP + GLP-1	2.5 mg SC weekly , then +2.5 mg every 4 weeks : 5, 7.5, 10, 12.5, 15 mg	~22.5% at 72 wk SURMOUNT-1	Most effective agent available. Maintenance is 10 or 15 mg, or the maximum tolerated
Semaglutide (Wegovy) GLP-1	0.25 mg SC weekly , then every 4 weeks: 0.5, 1.0, 1.7, 2.4 mg . Tablet (Dec 2025): 1.5 mg daily , then every 30 days: 4, 9, 25 mg	~15% at 68 wk STEP 1 ~14% tablet, 64 wk OASIS 4	The only agent with a cardiovascular outcome benefit in obesity without diabetes (see below). The tablet needs an empty stomach, 4 oz water or less and a 30-minute wait or it is barely absorbed; it was not studied in T2DM , where levels run lower
Liraglutide (Saxenda)	0.6 mg SC daily , uptitrate weekly to 3.0 mg	~8%	Daily injection, now largely superseded, but useful where weekly agents are unavailable
Phentermine / topiramate Naltrexone / bupropion Orlistat	Oral options	~5 to 10%	Consider when GLP-1-based therapy is not accessible or not tolerated. Phentermine/topiramate is teratogenic : pregnancy test and contraception. Naltrexone/bupropion is contraindicated with seizure disorder or opioid use

BEYOND WEIGHT *why these are cardiometabolic drugs, not cosmetic ones*

Evidence	What it showed, and what it changes
SELECT, 2023 semaglutide 2.4 mg	17,604 adults with established CVD and overweight or obesity, without diabetes. 20% reduction in MACE (CV death, nonfatal MI, stroke). The benefit was largely independent of how much weight was lost , which is the argument that these are disease-modifying rather than weight drugs. Practical effect: in a patient with ASCVD and obesity but no diabetes, this is now a cardiovascular indication
CKM syndrome framing 2026 AHA/ACC/ADA/ASN	Obesity is Stage 1 , the first actionable stage, and weight loss is the main lever for stage regression . Obesity pharmacotherapy and surgery are added as needed to reach the 5% to 10% target rather than being held in reserve

MONITORING, HARMS & STOPPING

Issue	What to do about it
GI intolerance the usual reason for stopping	Nausea, vomiting, constipation, diarrhea. Nearly always dose-related and worst after each escalation. Slow the titration or hold at the current dose rather than abandoning the drug. Small meals, stop eating at satiety
Muscle mass loss	A meaningful fraction of weight lost is lean mass. Resistance training and adequate protein are part of the prescription , not optional advice, and this matters most in older and frail patients
Contraindications	Personal or family history of medullary thyroid carcinoma or MEN2 (class boxed warning). Stop in pregnancy and discuss contraception before starting. Caution with prior pancreatitis and with gastroparesis
Gallbladder disease	Rapid weight loss raises gallstone and cholecystitis risk. Consider it in anyone on therapy presenting with right upper quadrant pain
Non-response at 12 weeks	A 12-week checkpoint at the maintenance dose appears in the labeling for several agents: if weight loss is under 5% , do not simply continue. Check adherence and injection technique first, then switch agent or refer for surgery rather than waiting another year on a drug that is not working
Stopping the drug	Weight is regained after discontinuation , and a large share of it. Frame treatment as chronic disease management from the first visit, the way you would antihypertensives, so that stopping is a decision rather than a default

Do not prescribe two of the same molecule. Semaglutide is **Wegovy** for obesity (to 2.4 mg) and **Ozempic** for T2DM (to 2.0 mg); tirzepatide is **Zepbound** for obesity and **Mounjaro** for T2DM. Semaglutide also comes as **Rybelsus** tablets for T2DM, and Rybelsus and Wegovy tablets share the 1.5, 4 and 9 mg strengths. The brand differs by indication, route and maximum dose, not by drug, so a patient can arrive on two and be double-dosed.

TWO REVERSALS RESIDENTS STILL GET WRONG

- **Perioperative: the blanket hold is out of date.** The 2023 ASA guidance advised holding weekly agents for a week before a procedure. **The December 2024 multi-society guideline replaced that: most patients may continue their GLP-1**, with individualized risk assessment rather than an automatic stop. The underlying concern is delayed gastric emptying and residual gastric contents, so assess GI symptoms and recent dose escalation, and escalate airway precautions if concerned rather than reflexively cancelling.
- **Bariatric surgery thresholds moved in 2022.** BMI > 35 alone is sufficient, and 30 to 34.9 with metabolic disease should be considered. Quoting the NIH 1991 numbers (40, or 35 with a comorbidity) **under-refers patients who now qualify**, and surgery still outperforms medication for durable weight loss and diabetes remission.

References: SURMOUNT-1 · STEP 1 · SELECT (NEJM 2023) · 2022 ASMBS/IFSO indications for metabolic and bariatric surgery · 2024 multi-society perioperative GLP-1

guidance · 2026 AHA/ACC/ADA/ASN CKM guideline · FDA prescribing information.

Educational aid; verify against current guidance, local formulary and patient factors.

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Depression & Anxiety

Score it, exclude bipolar and the mimics, pick the drug by its side effects · and give every trial an adequate dose for an adequate time

Ask about suicide
directly, every time

SCREEN, SCORE, AND TWO QUESTIONS FIRST *the number guides treatment; two questions prevent disasters*

SCREENING AND SCORING

USPSTF 2023: screen all adults for depression (including pregnancy/postpartum) and adults up to 64 for anxiety. PHQ-2 gates to PHQ-9: bands at 5/10/15/20, ≥ 10 is the usual treatment trigger, remission < 5 , response = 50% drop. GAD-7 bands 5/10/15. **Re-score at every visit:** treating without trending is titrating insulin without glucose values.

⚠ BIPOLAR SCREEN FIRST

Antidepressant monotherapy in unrecognized bipolar disorder can precipitate mania, and bipolar illness usually debuts as a depressive episode: the patient asking for help with depression is exactly the one who might be bipolar. Ask about **decreased need for sleep with increased energy, racing thoughts, pressured speech, impulsive spending or sexual behavior**, plus family history (or use the MDQ). Positive = mood-stabilizer pathway with psychiatry, not an SSRI.

⚠ SUICIDE: ASK DIRECTLY

"Are you having thoughts of killing yourself?" **does not plant the idea:** that fear is disproven and is the main reason clinicians under-ask. Any PHQ-9 item-9 positive gets the same-day conversation: **ideation, plan, intent, means, protective factors. Ask about firearms by name:** means restriction is one of the few interventions proven to prevent deaths. Active ideation with plan and intent is an emergency, not a referral.

CHOOSE THE DRUG BY ITS SIDE EFFECTS *efficacy is comparable, so the profile is the selection tool*

Agent	Dose and the reason to pick (or avoid) it
Sertraline / escitalopram <i>the defaults</i>	Sertraline 25-50 → 50-200 mg; escitalopram 5-10 → 10-20 mg. Best tolerated, safe in cardiac disease; sertraline is the usual choice in pregnancy and lactation. Class effects: early GI upset and activation, sexual dysfunction, hyponatremia in the elderly, bleeding with NSAIDs/anticoagulants
⚠ Citalopram	FDA QT cap: max 40 mg/day, 20 mg if elderly or hepatic impairment. ECG with cardiac disease or other QT drugs. "Failed citalopram 40" means switch agents, not push the dose
Duloxetine	30 → 60 mg. SNRI that also treats neuropathic pain and fibromyalgia: the rational pick when depression and chronic pain coexist. Avoid in heavy alcohol use and hepatic disease
Venlafaxine XR	37.5-75 → 75-225 mg. Effective in depression and anxiety; dose-dependent hypertension (check BP at titration) and one of the harshest discontinuation syndromes: always taper
Bupropion	150 → 300-450 mg XL. No sexual dysfunction, no weight gain , activating; the switch or add-on when SSRIs cause either, and it aids smoking cessation. Contraindicated in seizure disorders and active eating disorders; not for prominent anxiety
Mirtazapine	7.5-15 → 15-45 mg qHS. Sedation + appetite stimulation ARE the therapy in the depressed elderly patient with insomnia and weight loss; more sedating at LOWER doses; minimal sexual dysfunction
Fluoxetine / paroxetine	Fluoxetine's long half-life self-tapers (best for dose-missers, useful to bridge hard tapers); activating. Paroxetine: most weight gain, most anticholinergic, worst discontinuation syndrome , avoided in pregnancy: rarely the answer. Low-dose trazodone is a sleep aid, not an adequate antidepressant

RUN THE TRIAL PROPERLY *most "failures" were underdosed, under-timed, or not taken*

Rule	Detail
Adequate trial	Therapeutic dose for 4-6 weeks minimum (full effect 8-12) before judging. Warn about the first two weeks (GI upset, jitteriness before benefit): patients who expect it stay on the drug. Under-25s: boxed warning for early emergent suicidality , so see or contact them within 1-2 weeks of starting
Ask what they will not volunteer	Sexual dysfunction is the leading silent adherence killer: ask at follow-up. Bupropion (switch or add-on) is the usual answer. Prior response to a specific agent is the best predictor of response now: ask what worked before
Inadequate response	Optimize dose → switch (within class, or SNRI/bupropion/mirtazapine) or augment (bupropion, mirtazapine, or an atypical antipsychotic). Two adequate failed trials = treatment-resistant: refer , where esketamine and ECT live. ECT remains the most effective treatment for severe, psychotic or catatonic depression
The calendar	Continue 6-12 months after remission for a first episode (stopping at "I feel better" roughly doubles relapse); maintenance after two episodes. Taper on the way out: discontinuation syndrome (days: dizziness, "brain zaps," flu-like) is not relapse (weeks: the original symptoms), and misreading one for the other re-anchors the drug
Mimics and severity	TSH, CBC, B12 where the picture fits; quantify alcohol, screen OSA . Mild disease (PHQ-9 5-9): CBT, behavioral activation and exercise before medication. Moderate-severe: combination beats either alone . Psychosis, catatonia, or refusing intake: psychiatry now

ANXIETY: SAME DRUGS, GENTLER START *and the benzodiazepine trap*

GAD AND PANIC DISORDER

SSRIs/SNRIs first-line for both, started at **HALF the usual dose** (sertraline 12.5-25, escitalopram 5): anxious patients are exquisitely sensitive to early activation and a bad first week ends treatment. Say the timeline out loud: benefit at 2-4 weeks, mature at 8-12. **CBT equals medication with more durable benefit.** Buspirone adjuncts GAD (not panic; needs 2-4 weeks, BID-TID). Propranolol 10-40 mg covers performance situations only. For panic, name the physiology: **the attack peaks and passes within minutes and cannot hurt you:** that sentence alone reduces attack frequency.

⚠ BENZODIAZEPINES: THE IMMEDIACY IS THE TRAP

They work instantly, **which is how they entrench:** a PRN dose during a panic attack teaches the patient the pill ended an event that would have self-terminated anyway, avoidance is reinforced, and tolerance and dependence develop within weeks. Elderly patients add falls and cognitive harm. **Bridge use only (2-4 weeks while the SSRI takes hold), with the end date written into the first prescription.** Never with opioids (boxed warning: fatal respiratory depression) or substance-use history. Long-term users taper slowly: withdrawal seizures are real.

PEARLS & PITFALLS

- **Anhedonia counts even when sadness is denied.** "Nothing is enjoyable anymore" satisfies the cardinal criterion in the patient who insists they are not depressed.
- **Alcohol quietly defeats the antidepressant.** Nightly drinking is both depressogenic and the cause of the 3 AM awakenings: quantify it and name it as part of the prescription.
- **A first "panic attack" in an older patient gets the organic workup:** hyperthyroidism, arrhythmia, PE and hypoglycemia all masquerade as anxiety, and the mislabel runs in both directions.
- **Discontinuation symptoms arrive in days with brain zaps; relapse arrives in weeks with the original symptoms.** Teaching the difference prevents both unnecessary restarts and false "treatment failures."





Low Back Pain

90-95% is nonspecific and self-limited · the skill is finding the few percent that is not · and not imaging the rest

Retention first
cauda equina is a same-hour problem

⚠ RED FLAGS *ask these every visit; they are the reason the history exists*

Cause	Red flags	Action
⚠ Cauda equina <i>the emergency</i>	New urinary retention is the most consistent early finding ; saddle anesthesia, bilateral leg weakness or sciatica, lost anal sphincter tone, overflow incontinence are classic but later	Post-void residual is the fastest bedside test. Elevated PVR with back pain and leg symptoms = urgent MRI and immediate surgical consult , not a referral. Delay converts a recoverable syndrome into permanent bladder, bowel and sexual dysfunction
Malignancy	History of cancer (strongest single predictor) , unexplained weight loss, age over 50, pain worse at night or at rest, no improvement after 4-6 weeks	ESR/CRP plus imaging; MRI if suspicion is meaningful. Breast, prostate, lung, kidney and thyroid go to bone
⚠ Spinal infection	Fever, injection drug use, recent bacteremia or endocarditis, indwelling lines or catheters, immunosuppression, recent spinal procedure, unremitting night pain, focal tenderness	MRI with contrast + blood cultures + ESR/CRP. Hold antibiotics for cultures unless septic. Epidural abscess can progress to paralysis over hours
Vertebral fracture	Age over 70, significant trauma or minor trauma with osteoporosis , prolonged glucocorticoids, known osteoporosis, sudden severe focal pain	Plain films first; CT or MRI if negative and suspicion persists. A fragility fracture is itself an osteoporosis treatment indication
Axial spondyloarthritis	Under 45, insidious onset, morning stiffness over 30 min, BETTER with exercise and WORSE with rest , second-half-of-night pain, alternating buttock pain, psoriasis, IBD, uveitis	ESR/CRP, HLA-B27, MRI of the sacroiliac joints (sacroiliitis shows years before plain films), rheumatology. The inverted pattern is the tell, and it is missed for years
Not the spine at all	AAA (older vasculopath, tearing pain, pulsatile mass -a rupture presenting as "back pain" is a classic fatal miss), renal colic, pyelonephritis, pancreatitis, dissection, zoster before the rash	Examine the abdomen and flanks, not only the spine

DO NOT IMAGE THE FIRST SIX WEEKS *and understand that the harm is not just cost*

THE RULE

No imaging for nonspecific acute low back pain without red flags (ACP and Choosing Wisely both lead with this). Imaging does not improve pain, function or outcomes in this group.

⚠ WHY IT ACTIVELY HARMS

Degenerative changes, disc bulges and facet arthropathy are present in a large majority of **ASYMPTOMATIC** adults and increase with age, so a scan nearly always finds something. Reported without framing, it **anchors the patient to a structural explanation, increases fear-avoidance, and is associated with more procedures, more surgery and worse function.**

WHEN TO IMAGE, AND WITH WHAT

Any red flag; persistent pain beyond 4-6 weeks of conservative care; progressive or severe deficit; or a procedure genuinely being considered. MRI for suspected infection, malignancy, cauda equina and radiculopathy; **plain films first** for suspected fracture. **Frame incidental findings as age-expected in the same breath as the result**, or they become the patient's diagnosis for years.

LOCALIZE THE ROOT (the three that matter)

L4: medial shin and knee sensory loss, **weak knee extension**, reduced patellar reflex.
L5: dorsal foot and great toe, **weak dorsiflexion and great-toe extension (foot drop)**, reflexes preserved. **S1:** lateral foot and sole, **weak plantarflexion, reduced Achilles reflex**. L5 and S1 account for the large majority of lumbar radiculopathies.

THE THREE CATEGORIES

Nonspecific mechanical pain: 90-95%, no structural cause, most improve within 4-6 weeks. **Radicular pain:** a small minority, pain **below the knee** in a dermatomal pattern. **Serious pathology:** a few percent, and the reason the red-flag questions exist. **Straight-leg raise reproducing pain below the knee at 30-70 degrees** supports radiculopathy; a crossed positive SLR is more specific.

MANAGEMENT *the effective interventions are mostly not drugs*

Phase	Detail
Acute / subacute	Reassure with a specific prognosis and stay active: bed rest DELAYS recovery. First-line is nonpharmacologic (superficial heat, massage, acupuncture, spinal manipulation). If drugs are needed, NSAIDs first at lowest effective dose; muscle relaxants help briefly but sedate, especially in older adults
⚠ What does not work	Acetaminophen is ineffective for acute low back pain in the better trials, despite being the reflex first choice. Gabapentinoids do not work for nonspecific back pain or sciatica. Systemic glucocorticoids do not help. Opioids are not routine: severe refractory pain only, lowest dose, shortest course, explicit stop plan
Chronic (> 12 weeks)	Exercise therapy is the backbone and the sustainable modality wins. Add multidisciplinary rehabilitation, CBT, mindfulness, tai chi or yoga. Duloxetine has evidence; NSAIDs remain first-line pharmacotherapy
Yellow flags	Fear-avoidance, catastrophizing, depression, job dissatisfaction and pending litigation predict disability better than any imaging finding - and unlike the imaging, they are modifiable. Address them explicitly rather than treating them as background noise
Radiculopathy	Most disc herniations improve without surgery , often with radiographic regression, so 6 weeks of conservative care is the default absent progressive deficits. Early surgery for sciatica speeds relief but does not improve 1-2 year outcomes , making it a preference conversation. Epidural steroids give short-term radicular relief without changing the long game
Stenosis and fusion	Neurogenic claudication: bilateral leg heaviness on walking, relieved by sitting or leaning forward (shopping-cart sign; contrast vascular claudication, relieved by standing still). PT first; decompression for persistent function-limiting symptoms. Fusion for nonspecific back pain has poor evidence
Safety-netting	Review at 4-6 weeks if not improving , with return precautions the patient can repeat back: new leg weakness, saddle numbness, or difficulty passing urine. Cauda equina and infection can develop after a normal first assessment

PEARLS & PITFALLS

- **Pain below the knee in a dermatomal pattern** suggests radiculopathy; pain confined to the back and buttocks usually does not.
 - **Walking uphill is easier with neurogenic claudication** (more flexion opens the canal) and harder with vascular claudication (more demand). Cycling tolerance beats walking tolerance in stenosis.
- **"Nonspecific low back pain" is a positive diagnosis, not a failure to find something.** Saying so changes how the patient hears the plan.
 - **The job-security question is a clinical question.** Work worry predicts chronicity more strongly than anything on the exam, and it is asked far less often than the red flags are.

STAGE IT ON TWO AXES *albuminuria is not optional — it drives prognosis and therapy***G STAGE — eGFR (mL/min/1.73 m²)**

G1 ≥ 90 · **G2** 60–89 · **G3a** 45–59 · **G3b** 30–44 · **G4** 15–29 · **G5** < 15. CKD requires abnormality present for **> 3 months**. A single low eGFR is not CKD.

A STAGE — urine albumin-to-creatinine ratio (mg/g)

A1 < 30 · **A2** 30–300 · **A3** > 300. A patient with a normal eGFR and A3 albuminuria has CKD and substantial risk. **Check a UACR in everyone you stage.**

THE PILLARS OF KIDNEY PROTECTION *this is where progression is actually slowed*

Therapy	Who qualifies	Notes
RAS inhibitor ACEi or ARB	CKD with albuminuria , especially A2–A3, with or without diabetes	Titrate to the maximum tolerated dose. Do not stop for a stable creatinine rise of up to ~30% — that reflects intraglomerular pressure reduction, which is the point.
SGLT2 inhibitor	eGFR ≥ 20 with UACR ≥ 200 mg/g ; or T2D + CKD with eGFR ≥ 20 ; or heart failure at any albuminuria	Roughly 37% reduction in kidney disease progression and 23% reduction in AKI, irrespective of diabetes status . Expect a small initial eGFR dip — continue through it.
Finerenone non-steroidal MRA	T2D, eGFR > 25, normal potassium , and albuminuria > 30 mg/g despite a maximally tolerated RAS inhibitor	Requires careful monitoring of potassium and eGFR , with dose reduction or discontinuation if needed. Adds benefit on top of RASi and SGLT2i.
Blood pressure	Most adults with CKD	Target office BP < 130/80 mmHg , using standardised measurement technique.
Statin	Most adults with CKD	CKD is a cardiovascular risk state. Most patients with CKD die of cardiovascular disease, not of dialysis.

MANAGE THE COMPLICATIONS *each has a threshold worth remembering***ANEMIA**

Replete iron first — target ferritin and transferrin saturation before starting an erythropoiesis-stimulating agent. **Do not target a normal hemoglobin**; overcorrection increases stroke and thrombosis. Investigate other causes before attributing it to CKD.

MINERAL & BONE

Rising **phosphate** and **PTH** with falling **calcitriol**. Dietary phosphate restriction, then binders — **prefer non-calcium-based**. Treat vitamin D deficiency. Calcimimetics for persistent hyperparathyroidism on dialysis.

ACIDOSIS & POTASSIUM

Oral bicarbonate for serum bicarbonate persistently < 18–22 — slows progression and protects muscle and bone. For **hyperkalemia**, use a potassium binder to **keep the RASi and MRA on board** rather than stopping them.

WHEN TO REFER, AND WHAT TO AVOID *plan the transition long before you need it***REFER TO NEPHROLOGY**

eGFR < 30 (G4–G5) · **UACR > 300** · rapid progression · persistent hematuria with proteinuria · refractory hypertension on 4 agents · recurrent stones · suspected genetic or glomerular disease · persistent electrolyte abnormality. **Early referral allows planned access and modality choice** rather than a crash start on a temporary line.

AVOID / ADJUST

NSAIDs · iodinated contrast where avoidable · nephrotoxic antibiotic combinations such as **vancomycin plus piperacillin-tazobactam** · **metformin below eGFR 30** · renally cleared drugs at unadjusted doses. **Give a sick-day plan** to hold RASi, SGLT2i, diuretics, and metformin during intercurrent illness.

PLANNING FOR KIDNEY FAILURE *start the conversation at G4, not at G5*

Element	What to do, and when
Modality education	Discuss hemodialysis, peritoneal dialysis, transplantation, and conservative care while the patient is still well enough to choose. Conservative management is a legitimate option, particularly in the frail elderly, where dialysis may not extend meaningful life.
Vascular access	Refer for an arteriovenous fistula several months before anticipated need — fistulas take time to mature. Protect the non-dominant arm : no PICC lines, no routine phlebotomy or cannulation.
Transplant workup	Refer early — assessment takes months, and pre-emptive transplantation gives better outcomes than transplanting after years of dialysis. Living donor options should be raised explicitly.
Vaccination	Hepatitis B (higher doses and more doses may be needed in CKD), influenza, pneumococcal, and COVID-19. Do it before immunosuppression or dialysis.
Advance care planning	Document goals and resuscitation preferences. Dialysis withdrawal is a common terminal event and is far easier discussed early than in a crisis.

WHEN THE eGFR DROPS SUDDENLY *AKI on CKD is not just progression***WORK THROUGH THE REVERSIBLE CAUSES**

Volume depletion from over-diuresis or poor intake · **obstruction** — scan the bladder · new **NSAID**, contrast, or nephrotoxic antibiotic · sepsis or hypotension · decompensated heart failure or cirrhosis · rapidly progressive glomerular disease with an active sediment.

THE COMMON MISSTEP

Attributing every rise to "CKD progression" and doing nothing. True CKD progression is gradual over months. A jump over days is an **acute insult** with something reversible behind it — check a urine dipstick, a bladder scan, the drug chart, and the volume status before accepting a new baseline.

PEARLS & PITFALLS

- **A normal eGFR does not exclude CKD.** Isolated A3 albuminuria carries high renal and cardiovascular risk.
- **Do not stop the ACEi for a 25% creatinine rise.** Recheck, look for volume depletion, and preserve the drug that is protecting the kidney.
- **Expect an eGFR dip when starting an SGLT2 inhibitor.** It is hemodynamic and it precedes long-term benefit.
- **SGLT2 inhibitors help regardless of diabetes.** Do not reserve them for diabetic patients.
- **Use a potassium binder to keep the RASi**, rather than sacrificing a mortality-modifying drug.
- **Refer early.** Late referral means a tunnelled line and an emergency start, which carries worse outcomes than a planned fistula.





GERD, PUD & *H. pylori*

Alarm features decide the first step · PPI timing before escalation · and the first-line eradication regimen changed in 2024

Clarithromycin triple
is no longer first-line

FIRST DECISION: SCOPE OR TREAT *alarm features are the whole triage*

⚠ ENDOSCOPY, NOT A PPI TRIAL

Dysphagia or odynophagia (structural until proven otherwise), unintentional weight loss, GI bleeding, iron deficiency anemia, persistent vomiting, mass or lymphadenopathy, family history of upper GI cancer, and new dyspepsia at age 60 or older, where malignancy risk justifies scoping rather than empiric therapy.

NO ALARM FEATURES, UNDER 60

Test-and-treat for *H. pylori* (urea breath or stool antigen): more effective and cheaper than empiric PPI where prevalence is meaningful, and it removes a curable cause instead of masking it. If negative or symptoms persist, **8-week empiric PPI, then reassess**, not renew forever. Typical GERD needs no up-front endoscopy.

WHEN IT WILL NOT SETTLE

pH or pH-impedance monitoring confirms pathologic reflux when endoscopy is normal and PPI response is poor, and is **required before anti-reflux surgery**. **Manometry** excludes achalasia masquerading as refractory reflux. **Think eosinophilic esophagitis** in younger patients with dysphagia or food impaction: biopsy even if the mucosa looks normal.

GERD MANAGEMENT *the fix is usually timing, not dose*

Step	Detail
⚠ PPI timing	30-60 minutes BEFORE the first meal . PPIs irreversibly bind only <i>actively secreting</i> pumps, and the meal is what recruits them; bedtime dosing on an empty stomach wastes most of the effect. Wrong timing is the commonest cause of apparent PPI failure , so confirm it before doubling the dose or doubting the diagnosis
Initial course	Standard-dose PPI once daily for 8 weeks for symptomatic GERD or erosive esophagitis, then step down to lowest effective dose, on-demand, or stop. H2 blockers for breakthrough nocturnal symptoms (tachyphylaxis with scheduled use)
Lifestyle that works	Weight loss (strongest evidence: intra-abdominal pressure drives reflux), head-of-bed elevation and no eating within 3 h of lying down for nocturnal symptoms, targeted trigger avoidance rather than blanket elimination diets, smoking and alcohol reduction (both lower sphincter tone)
Long-term PPI: real indications	Barrett esophagus, LA grade C/D esophagitis, peptic stricture, hypersecretory states, and high-risk NSAID users (prior ulcer/bleed, age 65+, concurrent anticoagulant, antiplatelet or steroid). Everyone else gets a deprescribing attempt
⚠ Stopping a PPI	Taper, never stop abruptly . Chronic suppression raises gastrin and expands parietal cell mass, so withdrawal produces rebound hypersecretion worse than baseline for weeks , which is routinely misread as relapse and re-anchors the prescription. Bridge with an H2 blocker or alternate-day dosing, and warn the patient the first 2 weeks are hardest
Surgery	Fundoplication or magnetic sphincter augmentation for objectively confirmed reflux with incomplete PPI response or large hiatal hernia, only after pH study and manometry . In obesity, gastric bypass treats both while sleeve gastrectomy can worsen reflux

H. PYLORI: THE 2024 CHANGE *the regimen most residents memorized is no longer first-line*

Regimen (all 14 days)	Components and when
Bismuth quadruple <i>preferred empiric first-line</i>	PPI BID + bismuth QID + tetracycline QID + metronidazole TID-QID . The empiric choice when susceptibility is unknown, and the regimen of choice with penicillin allergy or prior macrolide exposure. Black stools and tongue are harmless; no alcohol with metronidazole. Pill burden is the adherence threat, so say so out loud
Vonoprazan dual	Vonoprazan BID + high-dose amoxicillin. A potassium-competitive acid blocker gives faster, more sustained suppression; non-inferior to bismuth quadruple with fewer adverse events and a simpler regimen. Not with penicillin allergy
Rifabutin triple	Omeprazole + amoxicillin + rifabutin. Empiric alternative or salvage; rifabutin resistance remains rare. Watch myelosuppression and rifamycin drug interactions
⚠ Clarithromycin triple	RETIRED as empiric first-line (ACG 2024) because US clarithromycin resistance crossed the threshold of usefulness. Use only with documented susceptibility , and never after prior macrolide exposure. Levofloxacin regimens carry the same caveat
Failure and salvage	Never reuse an antibiotic the patient already failed : resistance is the likeliest reason, so repeating it repeats the failure. Bismuth quadruple after failed clarithromycin; rifabutin or vonoprazan-based after failed bismuth quadruple. Susceptibility testing after two failures
Who to test	Active or prior peptic ulcer (eradication converts a relapsing disease into a cured one), gastric MALT lymphoma (eradication alone can induce remission in early disease), uninvestigated dyspepsia under 60, functional dyspepsia, early gastric cancer after resection and first-degree relatives of gastric cancer patients , long-term NSAID/aspirin users with ulcer history, unexplained iron deficiency anemia, and ITP

TESTING AND ULCER CARE *the false negative you will cause, and the ulcer you must re-scope*

⚠ HOW TO GET A FALSE NEGATIVE

Hold PPIs 2 weeks and antibiotics or bismuth 4 weeks before testing. Suppressed bacterial load is the commonest reason an infected patient is told they are clear. **Serology cannot confirm cure** (IgG persists for life after eradication), so use urea breath or stool antigen. **Test-of-cure at 4+ weeks after therapy is mandatory**: eradication failure is common enough that assuming success is unsafe.

ULCER CARE BEYOND ERADICATION

Gastric ulcers are biopsied at diagnosis and re-scoped at 8-12 weeks, because a malignant ulcer can look benign and can *partially heal* on acid suppression, which is exactly the false reassurance that delays a cancer diagnosis. Duodenal ulcers carry negligible malignancy risk. **Stop the NSAID if possible**; if not, co-prescribe a PPI. **An ulcer negative for both *H. pylori* and NSAIDs** needs a second look: Zollinger-Ellison (fasting gastrin off PPI), Crohn disease, malignancy, or unreported over-the-counter analgesics.

PEARLS & PITFALLS

- Duodenal ulcer pain is relieved by food; gastric ulcer pain is worsened by it, which is why gastric ulcer patients avoid eating and lose weight, adding an alarm feature to a disease that already needs biopsy.
- Ask about NSAIDs explicitly, including over-the-counter. Patients do not volunteer "just ibuprofen," and *H. pylori* plus NSAIDs frequently coexist: finding one does not excuse you from the other.
- Extraesophageal reflux (cough, hoarseness, asthma) is rarely the sole cause of those symptoms, and treating them with a PPI alone often fails: this indication is where years of unnecessary acid suppression begin.
- If you test for *H. pylori* you must be ready to treat and to confirm cure. There is no role for finding it and leaving it.
- Barrett esophagus is intestinal metaplasia, the precursor to adenocarcinoma: surveillance interval follows dysplasia grade, and dysplasia is treated endoscopically (ablation or resection), not merely watched.





Smoking Cessation

The highest-value intervention in general medicine · combine a drug with behavioral support, and treat relapse as data

Both together
beat either alone

THE FRAMEWORK *brief, repeatable, and effective even in a few minutes*

ASK AND ADVISE

Ask every patient at every visit and record it. **Advise clearly and personally:** "the most important thing you can do for your health is stop smoking", linked to *their* current problem. **Brief advice from a clinician measurably increases quit rates**, so it is worth doing even when time is short.

ASSESS AND ASSIST

Ask whether they are willing to try **now**. If yes, set a quit date, **offer pharmacotherapy** and arrange behavioral support. If not, **do not lecture**: state your availability, address the specific barrier, and ask again next time. Readiness changes.

ARRANGE FOLLOW-UP

Contact in the first week and again in the first month, which is when most relapse happens. Refer to a **quitline** or counseling: combining medication with behavioral support is **substantially more effective than either alone**, and this combination is the single most important message on this sheet.

PHARMACOTHERAPY *offer it to everyone who is willing, not just heavy smokers*

Option	How to use it	Notes
Varenicline most effective single agent	Start 1 week before the quit date and titrate up over the first week, continuing for at least 12 weeks. A flexible quit date within the first month is also acceptable	Nausea is the commonest side effect: take it with food and a full glass of water , and it usually settles. Also vivid dreams and insomnia. The neuropsychiatric boxed warning was removed after a large safety trial found no significant excess of serious neuropsychiatric events, so do not withhold it on that basis
Combination NRT preferred over single NRT	A patch for the steady background PLUS a short-acting form (gum, lozenge, inhaler or nasal spray) for cravings. Match the patch strength to how much they smoke	Combination beats a patch alone . Common failure: under-dosing and incorrect technique . Gum is chewed then parked against the cheek, not chewed continuously, and acidic drinks reduce absorption , so avoid coffee and soda just before
Bupropion SR	Start 1 to 2 weeks before the quit date, continuing for 7 to 12 weeks	Contraindicated with a seizure disorder, an eating disorder, or abrupt alcohol or benzodiazepine withdrawal , and with MAO inhibitors. Useful when depression coexists, and it is less likely to cause weight gain , which matters to many patients
Combinations	Varenicline plus NRT, or bupropion plus NRT	Reasonable in heavy dependence or after a previous failure on monotherapy
E-cigarettes	Not an FDA-approved cessation treatment	Some evidence of benefit versus NRT in trials, but they are not a licensed therapy, long-term safety is not established, and dual use is common . Offer approved treatments first, and if a patient has already switched completely, the goal becomes stopping the e-cigarette too

SPECIAL SITUATIONS *where the usual answer changes*

Situation	What to do
Hospitalized patients	The single best teachable moment there is , and the environment is already smoke-free. Offer NRT for withdrawal on admission rather than leaving the patient agitated and assumed to be delirious. Cessation support started in hospital and continued for at least a month after discharge is what works , so arrange the follow-up before they leave
Before surgery	Quitting reduces wound infection and respiratory complications. Longer is better, but even short-term cessation helps , so there is no point at which it is too late to raise it
Pregnancy	Behavioral intervention is first-line . Pharmacotherapy data are more limited, so it is an individualized discussion weighing continued smoking against drug exposure. Provide intensive support and follow up closely
Psychiatric illness and other substance use	Smoking prevalence is far higher and these patients want to quit at similar rates. Do not defer cessation on the assumption it will destabilize them : treat concurrently, and note that quitting can raise levels of drugs metabolized by CYP1A2 , including clozapine, olanzapine and theophylline , since it is the tobacco smoke rather than the nicotine that induces the enzyme. Doses may need reducing after quitting
Also screen for lung cancer	Annual low-dose CT for eligible patients aged 50 to 80 with a 20 pack-year history who smoke now or quit within 15 years. Screening and cessation belong in the same conversation , since cessation delivers more benefit than the scan

WHAT TO TELL THE PATIENT *concrete benefits beat general warnings*

THE BENEFITS START FAST

Circulation and lung function begin improving within **weeks**, cough and breathlessness improve over months, and **cardiovascular risk falls substantially within the first year or two**. **Excess lung cancer risk falls steadily over years but never returns fully to baseline**, which is the honest framing and the argument for quitting sooner.

IT HELPS AT ANY AGE

Quitting benefits patients even in their 60s, 70s and beyond, and even after a diagnosis of cancer or heart disease, where it improves treatment outcomes and survival. **"It is too late for me" is the belief to challenge directly**, because it is both common and wrong.

WITHDRAWAL IS TIME-LIMITED

Irritability, poor concentration, restlessness, low mood and increased appetite peak in the first week and largely settle within 2 to 4 weeks. Telling patients this in advance makes them far more likely to push through, and **adequate pharmacotherapy blunts it**.

PEARLS & PITFALLS

- **Relapse is the norm, not failure**. Most people need several attempts. **Frame a relapse as information**: what triggered it, what was the medication dose, was there behavioral support. Then re-treat, because a previous failed attempt predicts nothing about the next one.
- **Under-treatment is the commonest prescribing error**. A single low-dose patch in a heavy smoker will fail. **Match the dose to the dependence**, and ask how soon after waking they smoke, since within 30 minutes indicates high dependence.
- **Address weight gain before it becomes the reason for relapse**. Some gain is usual and is **vastly outweighed** by the benefit of quitting. Raise it proactively, offer activity and dietary support, and note bupropion and NRT blunt it somewhat.
- **Cutting down is a legitimate step, not a failure to commit**, and reduction with NRT support increases the chance of eventually quitting. **Do not dismiss the patient who will not set a quit date today**, since the strongest predictor of future success is continuing to be asked.





Osteoporosis

A fragility fracture IS the diagnosis · screen by age and risk, treat by risk tier, and never stop denosumab without a plan

Treat the disease
not just the fracture

DIAGNOSE & SCREEN *three doors into the diagnosis, and the 2025 screening rules*

THREE WAYS TO MAKE THE DIAGNOSIS

T-score ≤ -2.5 at femoral neck, total hip or lumbar spine (lowest site governs). **OR a hip or vertebral fragility fracture at ANY T-score:** bone that breaks from standing height has proven it fails under physiologic load, and a normal DEXA does not un-diagnose it. **OR osteopenia plus FRAX above threshold** (10-yr hip $\geq 3\%$ or major osteoporotic $\geq 20\%$).

WHO GETS A DEXA (USPSTF JAN 2025)

All women 65 and older (Grade B). Postmenopausal women under 65 with a risk factor: run a risk tool and screen if elevated (Grade B). **Men: no USPSTF mandate**, but fracture, glucocorticoids, ADT and hypogonadism justify scanning, and male post-hip-fracture mortality exceeds female. **Repeat on the SAME machine:** inter-machine error can fake a change.

T VS Z, AND THE SECONDARY HUNT

Z-score ≤ -2.0 in premenopausal women or men under 50 means bone below expected for age: hunt causes. Check **25-OH vitamin D, calcium, creatinine, TSH** in everyone; targeted PTH, celiac serologies, SPEP/light chains, testosterone, 24-h urine calcium. **Review the drug list first:** steroids, PPIs, aromatase inhibitors, ADT, anticonvulsants.

TREAT BY RISK TIER *foundation for everyone, then match the drug to the risk*

Tier	What and why
Everyone	Calcium 1,000-1,200 mg/day, food first (supplement only the shortfall); vitamin D 800-1,000 IU to 25-OH ≥ 30 , repleted BEFORE antiresorptives or you provoke hypocalcemia; resistance plus balance exercise (the balance half prevents the fall that breaks the hip); deprescribe sedatives and orthostatic agents; stop smoking, alcohol under 2/day
Who gets drugs	T ≤ -2.5 , OR hip/vertebral fragility fracture at any T-score, OR osteopenia with FRAX $\geq 3\%$ hip / $\geq 20\%$ major. FRAX under-reads recent fractures, multiple vertebral fractures, steroids and falls , so do not let it overrule a fracturing skeleton
High risk (most)	Oral bisphosphonate first-line: alendronate 70 mg or risedronate 35 mg weekly. Decades of fracture data, generic, rare harms. Zoledronic acid 5 mg IV yearly if GI intolerance or adherence fails
⚠ Very high risk T ≤ -3.0 with fracture, multiple or recent vertebral fractures, fracture on therapy	Anabolic FIRST, antiresorptive second. Teriparatide or abaloparatide 18-24 months, or romosozumab 12 months (boxed warning: not within 1 year of MI/stroke), then lock gains with bisphosphonate or denosumab. Why the order matters: antiresorptive-first blunts the anabolic, and unconsolidated anabolic gains drain away. Build first, then lock
Steroid users	≥ 2.5 mg prednisone ≥ 3 months: assess everyone; most on ≥ 7.5 mg at moderate+ risk get an oral bisphosphonate (ACR). Steroid bone loss is fastest in the first 3-6 months, so waiting for a bad DEXA wastes the window
Doses at a glance	Alendronate 70 mg PO weekly · risedronate 35 mg PO weekly · zoledronic acid 5 mg IV yearly · denosumab 60 mg SC q6 months · teriparatide 20 mcg SC daily · abaloparatide 80 mcg SC daily · romosozumab 210 mg SC monthly · raloxifene 60 mg PO daily
Niche agents	Raloxifene: vertebral protection only, raises VTE, lowers ER+ breast cancer, occasionally the right trade in a younger postmenopausal woman. Calcitonin: short-term analgesic bridge for acute vertebral fracture only, not a long-term drug. HRT: prevention in younger postmenopausal women who need it for vasomotor symptoms anyway
Monitoring	Repeat DEXA in 1-2 years on the same machine. Fracture or falling BMD on therapy: first ask adherence and administration technique (half stop orals by 1 year), then re-hunt secondary causes, then escalate to anabolic-first. A suppressed CTX confirms the bisphosphonate is actually on board when adherence is in doubt

THE RULES THAT PREVENT THE COMMON FAILURES *administration, holidays, and the one drug you must not stop*

Rule	Detail
Oral bisphosphonate technique	Empty stomach, full glass of water, upright 30 min. Absorption is under 1% and food abolishes it, so "taking it with breakfast" mimics treatment failure; upright prevents pill esophagitis. Avoid below CrCl 30-35
Drug holiday (bisphosphonates ONLY)	After 5 years oral / 3 years IV , reassess: low-moderate risk can pause because the drug stays bound in bone and keeps protecting, while duration-linked risks recede. High risk continues to 10 / 6 years
⚠ NEVER just stop denosumab	RANKL inhibition vanishes in ~ 7 months and the osteoclast rebound causes multiple spontaneous vertebral fractures . A lapsed refill is a patient-safety event. Stopping requires a bisphosphonate transition ; there is no denosumab holiday. Flag it on the med list like an anticoagulant
Keep rare harms in proportion	ONJ ~ 1 per 10,000-100,000 patient-years and atypical femoral fracture similarly rare, versus fractures prevented in 1 of every 10-20 treated. New thigh or groin ache on long-term therapy gets an X-ray (AFF prodrome), but stopping therapy over headlines trades a rare risk for a common one
CKD routing	CrCl < 30 -35 bars bisphosphonates; denosumab is not renally cleared and becomes the antiresorptive of choice, with hypocalcemia surveillance (replete vitamin D, check calcium after dosing)

THE HIP-FRACTURE ADMISSION *where the treatment gap closes, or becomes permanent*

ZOLEDRONIC ACID BEFORE DISCHARGE

5 mg IV within 90 days of repair: new clinical fractures down $\sim 35\%$ and all-cause mortality down 28% (HORIZON-RFT). Few ward drugs carry a mortality benefit this clean. Replete vitamin D first, confirm CrCl ≥ 35 , and warn about the first-infusion flu-like reaction (1-3 days, acetaminophen blunts it). **"Too acute to treat" becomes "never treated":** fewer than 25% of hip-fracture patients ever start therapy.

CLOSE THE LOOP

Name the diagnosis in the chart: "mechanical fall with fracture" buries the disease that caused it. Lateral spine imaging if height loss > 1.5 in (silent vertebral fractures reclassify to very-high-risk). Ortho-geriatric comanagement, PT with balance work, fall-maker deprescribing, calcium and vitamin D, and a discharge line naming who repeats the DEXA and when. **Acute vertebral fracture: mobilize early**, bed rest accelerates bone loss.

PEARLS & PITFALLS

- The fracture is the diagnosis.** A hip or vertebral fragility fracture with a T-score of -1.8 is osteoporosis, and waiting for -2.5 is how the treatment gap happens. Prior fracture is also the strongest predictor of the next one.
- Same hormone, opposite outcome:** continuous PTH (hyperparathyroidism) destroys bone; intermittent daily pulses (teriparatide) build it. The exposure pattern, not the molecule, decides.
- Half of oral bisphosphonate patients stop by one year.** Before calling any regimen a failure, ask about adherence and administration technique, then re-hunt secondary causes, then escalate.
- The drug protects the bone, not from the floor.** Balance exercise and deprescribing sedatives and orthostatic agents prevent the fall itself, and exercise is the strongest single non-drug intervention.





Cardiovascular-Kidney-Metabolic (CKM) Syndrome

2026 AHA/ACC/ADA/ASN Guideline (JACC, June 2026) · retires the 2013 AHA/ACC/TOS obesity guideline

Stage decides what & how often

THE FIVE STAGES *bidirectional: the point is regression, not just prevention*

Stage	Definition	What it changes for you
0	No CKM risk factors. Normal weight, glucose, BP, lipids and kidney function, no subclinical or clinical CVD	Goal is keeping them here. Widest screening interval; lifestyle counseling is the whole intervention
1	Excess or dysfunctional adiposity: overweight, obesity, or abdominal obesity, may include prediabetes. Without Stage 2 metabolic risk factors and without subclinical CVD	First actionable stage and the most often ignored, because the patient feels well . Weight loss here can drive true stage regression
2	Metabolic risk factors or moderate-to-high-risk CKD: hypertension, T2DM, hypertriglyceridemia, metabolic syndrome, or CKD in moderate-to-high-risk KDIGO categories	Screening tightens sharply (annual lipids, glycemia, BP, and eGFR <i>and</i> UACR). Entry point for cardioprotective drug therapy
3	Subclinical CVD or risk equivalent: CAC > 100, pre-HF (NT-proBNP ≥ 125 pg/mL with echo dysfunction, or elevated hs-troponin), very high-risk CKD , or PREVENT 10-yr CVD risk ≥ 20%	Last stop before clinical disease. In pre-HF Stage 3, intensive lifestyle plus intensive risk factor control is Class 1 , because clinical HF is still preventable
4	Clinical CVD (CHD, HF, stroke, PAD, atrial fibrillation) overlapping CKM risk factors. 4a without kidney failure, 4b with kidney failure	Treating the coronary disease while ignoring obesity, diabetes and albuminuria leaves most of the modifiable risk on the table

Stage youths under 18 as well as adults. The stages are explicitly bidirectional, which is the practical difference from a risk calculator: a score gives a probability and implies nothing can be undone, while the stage tells you what to try to reverse.

THE FOUR CKD THRESHOLDS *written in mg/g and mL/min, and easy to mix up*

Agent	Who qualifies	Why / what it prevents
RAS inhibitor (ACEi or ARB) Class 1	CKD with T2DM , or without T2DM but UACR ≥ 30 , and eGFR ≥ 30	Lowers intraglomerular pressure and slows GFR loss. Maximum tolerated dose is the recommendation , not a token dose, and that is the step most often left undone
SGLT2 inhibitor Class 1	CKD with T2DM , or without T2DM but UACR ≥ 200 , and eGFR ≥ 20	Slows GFR loss, cuts HF hospitalization and CV mortality (DAPA-CKD, EMPA-KIDNEY). Expect an early eGFR dip and do not stop for it: it is hemodynamic and precedes the benefit
SGLT2 inhibitor Class 2a	CKD without T2DM , UACR 30 to 199	Fills the gap below the Class 1 albuminuria threshold, where benefit is likely but the trial evidence is less direct
Finerenone (nonsteroidal MRA) Class 1	CKD + T2DM + UACR ≥ 30 despite ACEi/ARB and SGLT2i as tolerated, with eGFR ≥ 25	Targets the residual albuminuria that persists on standard therapy (FIDELIO-DKD). Added <i>despite</i> the other two, not instead of them. Monitor potassium

You cannot apply any of these without a UACR, because every threshold is written in mg/g. eGFR alone misses early kidney disease: albuminuria appears first and independently predicts both kidney and cardiovascular events.

TYPE 2 DIABETES *drug choice follows risk, not A1c*

Recommendation	Threshold	Why
SGLT2i or GLP-1-based therapy with proven benefit Class 1	Stage 2 to 3 with T2DM and PREVENT 10-yr CVD ≥ 7.5%	Benefit is largely independent of glucose lowering , which is why the trigger is cardiovascular risk rather than A1c
Intensified multifactorial treatment Class 1	Stage 2 to 3 with T2DM	Treating glycemia, BP, lipids and albuminuria as a bundle beats sequential single-target care for CVD, mortality and kidney outcomes
Metformin, added to a cardioprotective agent Class 2a	A1c 0.5% to 1% above the individualized goal	Note the reversal: metformin is now what you add for glycemic control <i>after</i> the cardioprotective drug, not the mandatory first step before it
GLP-1 plus SGLT2i together Class 2b	Increased CVD risk or multiple CKM risk factors	May add benefit beyond either alone; the weak class reflects thin dedicated combination outcome data

SCREENING, SET BY STAGE *and the two numbers that replace BMI alone*

EVERY ADULT BMI and waist circumference, both. Obesity at BMI ≥ 30; abdominal obesity at ≥ 88 cm in women, ≥ 102 cm in men . BMI alone misclassifies: it misses the normal-BMI patient with central adiposity and overcalls the muscular one.	FROM STAGE 2, ANNUALLY Lipids, glycemia, blood pressure, and eGFR with UACR , all Class 1. PREVENT risk in Stages 0 to 3: ≥ 20% 10-year risk is itself a Stage 3 criterion , and ≥ 7.5% prioritizes starting cardioprotective therapy.	DO NOT FORGET FIB-4 every 1 to 2 years with diabetes or ≥ 2 risk factors: MASLD travels with this syndrome, is silent, and FIB-4 costs nothing extra. Pre-HF biomarkers (BNP/NT-proBNP, hs-troponin) if PREVENT-HF ≥ 5%, Class 2a. Screen for OSA .
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WHAT CHANGED, AND WHAT GETS MISSED

- Obesity is the entry point, and the conversation is the intervention. Lifestyle first-line targeting **5% to 10%** of baseline weight, and counsel about that benefit **at least annually** (Class 1). That range is where blood pressure, glycemia, triglycerides and hepatic steatosis reliably improve **even if the patient stays in the obese BMI range**.
 - Two reversals worth knowing: metformin has moved from mandatory first step to a Class 2a glycemic add-on, and **BMI alone is no longer adequate** without a waist circumference.
- Social determinants are clinical data, not paperwork. Food insecurity, housing instability and cost barriers predict both the development of CKM syndrome and the failure of the plan you are writing. **A GLP-1 the patient cannot afford is a failed prescription.**
 - Add finerenone despite the other two, not instead. The commonest error is treating the four thresholds as a ladder where each replaces the last, when RAS inhibitor, SGLT2i and finerenone are meant to stack.