



CHEST PAIN

The five that kill · exclude them first, then risk-stratify with the right score

ECG IN 10 MINUTES
before anything else

THE FIVE THAT KILL *exclude these before you reach for a differential*

ACS

Pressure, exertional, radiating to jaw or arm, diaphoresis. **ECG within 10 minutes** and serial troponin. **A normal first ECG does not exclude it** — repeat it if the pain continues or changes.

PULMONARY EMBOLISM

Pleuritic pain, dyspnea, tachycardia, hypoxia. **Wells or Geneva first, then D-dimer only if probability is low or intermediate**; CTPA if the probability is high or the D-dimer is positive.

AORTIC DISSECTION

Abrupt, maximal at onset, tearing, radiating to the back. Pulse or blood-pressure differential, new aortic regurgitation, focal deficit. **CT angiography** of the aorta.

TAMPONADE

Beck's triad (hypotension, raised JVP, muffled sounds), **pulsus paradoxus over 10 mmHg**, electrical alternans. **Bedside echo makes the diagnosis**; pericardiocentesis treats it.

TENSION PNEUMOTHORAX

Sudden pain, severe dyspnea, absent breath sounds, tracheal shift, shock. **A clinical diagnosis — do not wait for the film.** Needle decompression, then a chest drain.

THE FIRST TEN MINUTES *the order that catches the time-critical causes*

- 1 ECG within 10 minutes of arrival**, and repeat at 15–30 minutes if pain persists or the picture changes. **Compare with an old ECG if one exists.** Look specifically for **ST elevation, new left bundle branch block, and the posterior MI pattern** (tall R and ST depression in V1–V3, which needs posterior leads V7–V9). **Right-sided leads in inferior MI** before you give any nitrate.
- 2 Vital signs in both arms.** A **systolic differential, a pulse deficit, or a new diastolic murmur** moves aortic dissection to the front of the queue and changes everything downstream, because anticoagulating a dissection is catastrophic.
- 3 High-sensitivity troponin at 0 and 1–2 hours.** Use your laboratory's assay-specific thresholds; **the change between samples matters more than a single absolute value.** Troponin marks myocardial injury, not infarction — PE, sepsis, heart failure, myocarditis, tachyarrhythmia, chronic kidney disease and dissection all raise it.
- 4 Bedside ultrasound and a chest film.** Echo for effusion, right ventricular strain and regional wall-motion abnormality; lung ultrasound for pneumothorax. **Widened mediastinum on the film supports dissection but its absence does not exclude it.**
- 5 Then, and only then, risk-stratify.** Scores are for patients in whom you have already excluded the immediately lethal causes — **they are not a substitute for that exclusion.**

RIGHT SCORE, RIGHT DECISION *each score answers one question only*

Score	Population	Bands	The one decision it answers
HEART	Undifferentiated chest pain in the emergency department	0–3 low (MACE roughly 2%) · 4–6 moderate (roughly 15–20%) · 7–10 high (roughly 50%)	Disposition. Low with negative serial troponins can go home with follow-up. It does not tell you when to take an admitted patient to the catheter laboratory — that is the commonest misuse of this score.
GRACE	Confirmed NSTE-ACS, already admitted	≤ 108 low · 109–139 intermediate · ≥ 140 high	Timing of the invasive strategy. GRACE ≥ 140 is the guideline trigger for an early invasive approach, within 24 hours. Intermediate risk is managed invasively during the admission. This is the score for the cath-timing question, not HEART.
TIMI	Unstable angina or NSTEMI	Seven equally weighted variables; 0–1 low, 6–7 high (14-day event rate roughly 5% rising to over 40%)	Prognosis, and who gains most from an invasive strategy and more intensive antithrombotics. Simpler than GRACE but less discriminating, so where guidelines specify a threshold for invasive timing they use GRACE, not TIMI.
Wells / PERC	Suspected pulmonary embolism	Wells sets pre-test probability; PERC applies only once probability is already low	Whether to test at all. PERC is a rule-out for low-probability patients, not a probability score. D-dimer belongs in low or intermediate probability, never as a screen in high probability — there, image regardless.
ADD-RS	Suspected aortic dissection	One point each for high-risk condition, pain feature, and examination finding	Whether to image now. Two or three features means immediate CT angiography. Zero or one with a negative D-dimer makes dissection unlikely.

The trap: these scores were derived in different populations to answer different questions, and their numbers are not interchangeable. **Applying a HEART band to a cath-timing decision, or a GRACE band to an emergency-department discharge, is the classic error.** Check that the score named actually matches the decision in front of you.

DON'T FORGET THESE

ESOPHAGEAL RUPTURE

Boerhaave: forceful vomiting, then severe pain, then subcutaneous emphysema. Mortality climbs steeply with delay. **CT with oral contrast**; it is a surgical emergency and is missed because it mimics ACS.

THE COMMON, NON-LETHAL CAUSES

Musculoskeletal, reflux, anxiety, herpes zoster (pain can precede the rash by days), pneumonia and pericarditis. **Reproducible tenderness lowers but does not exclude ACS** — a meaningful minority of infarcts have a reproducible chest wall.

WHO PRESENTS ATYPICALLY

Women, people with diabetes, the elderly, and postoperative patients present with dyspnea, fatigue, nausea or confusion rather than pain. **Keep the threshold for an ECG and a troponin low in these groups**; this is where infarcts are missed.

PEARLS & PITFALLS

- ECG within 10 minutes, and repeat it.** A single normal tracing in ongoing pain is not reassurance, it is an incomplete test.
- Check the blood pressure in both arms before you anticoagulate.** Treating a dissection as ACS is the catastrophic version of this miss.
- Troponin means myocardial injury, not infarction.** Interpret the delta and the clinical context, never a lone number.
- HEART is for disposition; GRACE is for cath timing.** Swapping them is the classic score error on this presentation.
- Tension pneumothorax is diagnosed at the bedside.** If you are waiting on the chest film, you have already waited too long.
- Reproducible chest wall tenderness does not exclude ACS,** and neither does relief with a gastrointestinal cocktail.





Acute Decompensated Heart Failure

Phenotype before you prescribe · decongest properly · find the precipitant · start the pillars

Wet or dry
warm or cold

PHENOTYPE FIRST *congestion (wet vs dry) and perfusion (warm vs cold); the quadrant dictates the drug*

Quadrant	What it is	Treatment
Warm & wet ~2/3 of admissions	Congested, perfusing normally	IV loop diuretic . Add a vasodilator (nitroglycerin) if hypertensive. No inotrope , which only adds arrhythmia and oxygen demand.
Cold & wet	Congested and hypoperfused: this is cardiogenic shock	Restore perfusion first : norepinephrine for pressure, then an inotrope (dobutamine or milrinone) for output. Diurese only once perfused, and involve the shock team early.
Cold & dry	Hypoperfused, not congested	Cautious fluid challenge (250 mL), then an inotrope if output stays low. The rarest and most easily over-diuresed quadrant.
Warm & dry	Compensated, euvolemic	Not an acute problem . Optimize oral GDMT and work out why they decompensated.

HOW TO CALL THE QUADRANT AT THE BEDSIDE

Wet: raised JVP (the single most useful sign), orthopnea, **B-lines on lung ultrasound**, a plethoric non-collapsing IVC, edema. **Cold**: narrow pulse pressure (under 25% of systolic), cool mottled peripheries, rising lactate, confusion, worsening renal function. **Natriuretic peptides rule out rather than rule in**: a **BNP under 100** or **NT-proBNP under 300 pg/mL** makes acute HF very unlikely, while raised values also occur in AF, PE, sepsis and renal failure. **Obesity lowers them; sacubitril-valsartan raises BNP but not NT-proBNP**.

FIND THE PRECIPITANT *CHAMPIT: three of these change the whole plan, and diuresis alone will fail*

Letter	What to do about it
C · Acute C oronary syndrome	ECG and troponin in everyone . Shock plus ischemia means the catheter lab, not more furosemide.
H · Hypertension emergency	Drives SCAPE (below). The treatment is vasodilation, not volume removal.
A · Arrhythmia	Atrial fibrillation with a rapid rate is a classic trigger. Rate or rhythm control; a fast irregular rate can be both cause and consequence.
M · Mechanical cause	Papillary muscle rupture, VSD, acute valve failure, tamponade . A new murmur with abrupt decompensation is surgical. Echo .
P · Pulmonary embolism	Hypoxia out of proportion with a clear chest. CTPA if the picture does not fit.
I · Infection · T · Tamponade	Plus the everyday ones: non-adherence to drugs or salt, NSAIDs, anemia, thyroid disease, alcohol . Non-adherence is the commonest precipitant of all , so ask about it directly rather than assuming disease progression.

DECONGEST, AND CHECK THAT IT IS WORKING *the commonest failure is an inadequate dose left unreviewed for a day*

- Dose off their home dose, not off a protocol. IV furosemide at 1 to 2.5 times the total daily oral dose**, given IV because gut wall edema makes oral absorption unreliable. **Diuretic-naïve: 20 to 40 mg IV**. DOSE, NEJM 2011 found **no difference between bolus and infusion**, so either is acceptable; the high-dose strategy relieved symptoms better at the cost of a transient creatinine rise.
- Check the response early, not tomorrow. A spot urine sodium at 2 h below 50 to 70 mEq/L, or urine output under 100 to 150 mL/h at 6 h, means the dose is too low. Double it** rather than waiting a day to discover the weights have not moved. Track daily weight, strict input and output, and the JVP.
- If still resistant, block a second nephron site. Acetazolamide 500 mg IV daily** blocks proximal reabsorption: ADVOR, NEJM 2022 (n = 519) improved successful decongestion at 3 days to **42.2% from 30.5%** (RR 1.46, P < 0.001) and shortened stay, **though without a mortality or rehospitalization benefit**. A **thiazide (metolazone or IV chlorothiazide)** blocks the distal tubule and works similarly, with more electrolyte and renal derangement. **Ultrafiltration** is for true diuretic resistance.
- Do not stop for a modest creatinine rise**. A rise during effective decongestion usually reflects **relief of renal venous congestion and hemoconcentration, not injury**, and patients who decongest fully do better despite it. **Keep going if the JVP is falling and the patient feels better**; stop and reassess if they are hypotensive, oliguric or clearly dry. **Replace potassium and magnesium aggressively**.

TWO PRESENTATIONS THAT ARE NOT ORDINARY CONGESTION

SCAPE · FLASH PULMONARY EDEMA

Sympathetic crashing acute pulmonary edema: severe hypertension, sudden onset, distressed and drowning. **The problem is afterload and redistribution, not total body water**, so these patients are frequently **euvolemic or even dry**. **High-dose IV nitroglycerin is the key drug**, at doses far above the anginal range, with **early CPAP or BiPAP**. **Heavy diuresis is the wrong reflex** and leaves them hypotensive once the pressure breaks.

LOW-OUTPUT AND CARDIOGENIC SHOCK

Norepinephrine first to restore a perfusing pressure, then **dobutamine or milrinone** for output; **milrinone if they are beta-blocked**, since it works downstream of the receptor. **Escalate at SCAI stage C, not at stage E**. Mechanical support and advanced-HF or transplant referral belong in the conversation early, not after multi-organ failure.

BEFORE THEY LEAVE *the admission is the opportunity, and discharge is where the readmission is decided*

START AND UPTITRATE THE FOUR PILLARS

For HFrEF: **ARNI (or ACE inhibitor / ARB) · beta-blocker · MRA · SGLT2 inhibitor**. **Start them in hospital and do not stop chronic beta-blockade unless the patient is in shock**; withdrawal worsens outcomes. Begin or uptitrate the beta-blocker **once euvolemic**. **SGLT2 inhibitors can be started during the admission**.

UPTITRATE FAST, WITH FOLLOW-UP

STRONG-HF, Lancet 2022 (n = 1078) uptitrated GDMT rapidly before discharge with close review afterwards and cut **180-day death or HF readmission to 15.2% from 23.3%** (P = 0.021). **Review within 1 to 2 weeks**, because the readmission risk is front-loaded.

DISCHARGE CRITERIA

Do not discharge a congested patient. Require: **euvolemia with a normal JVP**, transition to oral diuretic for at least 24 h with stable weight, stable renal function and potassium, **ambulating without dyspnea**, and a documented weight, daily-weight plan and follow-up appointment.

PEARLS & PITFALLS

- Phenotype before you prescribe**. Warm-and-wet needs diuresis; cold-and-wet needs perfusion restored first.
- Check the response at 2 to 6 h and double the dose if it is inadequate**, rather than discovering it tomorrow.
- SCAPE is a vasodilator problem**. High-dose nitroglycerin and NIV, not aggressive diuresis.
- A creatinine bump during effective decongestion is usually not injury**. Keep going if the JVP is falling.

References: ACC / AHA / HFSA guideline for the management of heart failure · ESC guidelines for acute and chronic heart failure · Felker et al, DOSE, NEJM 2011 ·

Mullens et al, ADVOR, NEJM 2022 · Mebazaa et al, STRONG-HF, Lancet 2022 · HFA position statement on diuretic use in heart failure.

Educational aid; verify doses against local protocol, renal function, and patient factors.





AF with RVR

Atrial fibrillation with rapid ventricular response · treat the trigger, not just the number

< 110

lenient rate target

UNSTABLE? Cardiovert NOW

Hypotension or shock, ischemic chest pain, acute pulmonary edema, or altered mentation caused by the rate means **immediate synchronized DC cardioversion at 120 to 200 J biphasic**, with sedation if time allows. **The judgment is whether the rate is causing the instability or merely accompanying it:** a septic patient at 140 is usually tachycardic because of the sepsis, and cardioverting them treats the wrong problem and rarely holds.

TREAT THE TRIGGER FIRST *new AF in an acutely ill patient is usually a symptom, and it often reverts when the cause is fixed*

FIND THE DRIVER

Sepsis, hypovolemia, pain, PE, hyperthyroidism, alcohol, post-operative state, and myocardial ischemia. Rate-controlling a patient whose driver is untreated means escalating doses against a physiologic tachycardia, and the drugs then cause the hypotension you were trying to avoid.

CORRECT POTASSIUM AND MAGNESIUM

Low magnesium sustains RVR and blunts every rate-control drug you give. Replace it empirically in the acutely ill: **magnesium 2 g IV over 15 min** both slows AV conduction directly and improves the response to everything else.

WORK IT UP

12-lead ECG (not just the monitor), **electrolytes**, **TSH**, **troponin if ischemia is plausible**, and **an echo**. The ECG is also how you catch the one presentation that changes every drug decision, below.

RATE CONTROL *lenient is as good as strict, and the choice of agent is decided by the ejection fraction*

Drug	Dose	When to use it, and when not to
Metoprolol <i>first-line</i>	2.5 to 5 mg IV over 2 min, repeat q5 min to a max of 15 mg , then convert to oral	Preferred when there is no decompensated heart failure , and the better choice when the driver is adrenergic (post-operative, thyrotoxic, ischemic). Avoid in acute decompensated HF, severe asthma, and bradycardia .
Diltiazem	0.25 mg/kg IV over 2 min , may repeat at 0.35 mg/kg , then infusion 5 to 15 mg/h	Equally effective and often faster. Contraindicated in HFrEF , because its negative inotropy can precipitate cardiogenic shock, and in pre-excitation .
Digoxin	0.25 to 0.5 mg IV load , then 0.25 mg q6h , total ≤ 1.5 mg in 24 h	The add-on for heart failure or hypotension , since it does not drop the blood pressure. Slow onset over hours , so it is never the answer to an urgent rate. It controls the rate at rest but poorly with exertion, and toxicity rises in renal impairment.
Amiodarone	150 mg IV over 10 min , then 1 mg/min for 6 h , then 0.5 mg/min	Useful when heart failure or hypotension rules out the others, and gives rate and rhythm control together . Give by central line where possible (phlebitis). Treat it as chemical cardioversion : it may restore sinus rhythm, so the same anticoagulation rules apply.

LENIENT BEATS STRICT

Target a resting rate below 110. RACE II, NEJM 2010 found lenient control non-inferior to a strict target of under 80, with **fewer drugs, fewer visits and fewer side effects**. Chase a tighter number only if the patient remains symptomatic or the ventricle is failing from the rate.

RHYTHM CONTROL AND ANTICOAGULATION *the 48-hour rule governs when you may cardiovert safely*

- Under 48 h of clearly documented onset:** cardioversion is reasonable without prior anticoagulation, electrically or chemically. Use **flecainide or propafenone only if there is no structural heart disease**, and **amiodarone** if there is. **Anticoagulate at the time of cardioversion regardless**, since the atrium is stunned afterwards and the stroke risk is highest in the days that follow.
- Over 48 h or unknown duration:** either **3 weeks of therapeutic anticoagulation before** elective cardioversion, or **a TEE to exclude left atrial appendage thrombus** and cardiovert immediately. Either way, **anticoagulate for at least 4 weeks afterwards**, because mechanical atrial function recovers long after the rhythm looks normal.
- Consider early rhythm control in newly diagnosed AF.** EAST-AFNET 4, NEJM 2020 found that starting rhythm control **within a year of diagnosis** reduced cardiovascular death, stroke and hospitalization. In critical illness the opposite applies: **treat the cause and rate-control**, because most of these patients revert on their own.

STROKE PREVENTION *decided by the score, never by the rhythm strategy*

CHA₂DS₂-VASc

C heart failure 1 · **H** hypertension 1 · **A**₂ age ≥ 75 2 · **D** diabetes 1 · **S**₂ prior stroke, TIA or thromboembolism 2 · **V** vascular disease 1 · **A** age 65-74 1 · **Sc** female 1.

Anticoagulate at ≥ 2 in men and ≥ 3 in women; at 1 and 2 respectively it is a discussion. **The score does not change because you restored sinus rhythm.**

DOAC DOSING

Apixaban 5 mg BID, reduced to **2.5 mg BID** if **2 of 3:** age ≥ 80, weight ≤ 60 kg, creatinine ≥ 1.5. **Rivaroxaban 20 mg daily with the evening meal** (15 mg if CrCl 15-50). **Edoxaban 60 mg daily**, 30 mg if CrCl 15-50 or weight ≤ 60 kg, and **avoid if CrCl > 95**. **Dabigatran 150 mg BID** (75 mg if CrCl 15-30).

WHEN A DOAC IS WRONG

Moderate-to-severe mitral stenosis or a mechanical valve: warfarin only. DOACs were harmful in the mechanical-valve trial. **Antiphospholipid syndrome: warfarin.** In severe renal impairment use apixaban or warfarin, and **never combine a DOAC with an antiplatelet** without a specific, time-limited indication.

THE TRAP: PRE-EXCITED AF *the one situation where standard rate control can kill the patient*

RECOGNIZE IT

An irregularly irregular, broad-complex tachycardia, often very fast (above 200) with beat-to-beat variation in QRS width. This is AF conducting down an accessory pathway in **WPW**. Look for a delta wave on any previous ECG.

WHY AV-NODAL BLOCKERS KILL

Blocking the AV node removes the only competing route and pushes all conduction down the accessory pathway, which has no decremental properties. The ventricular rate accelerates and degenerates into **ventricular fibrillation**. This applies to **adenosine, beta-blockers, diltiazem, verapamil and digoxin**.

WHAT TO GIVE INSTEAD

Procainamide, or **synchronized DC cardioversion**, which is the safest choice in anyone unstable or when you are unsure. Amiodarone is **not** a safe default here. Refer for accessory pathway ablation afterwards.

PEARLS & PITFALLS

- New AF in critical illness is usually the symptom.** Treat sepsis, pain, hypovolemia and the PE, and the rate often follows.
- Replace magnesium early.** It aids rate control and is nearly always low in these patients.
- A rate above 150 that is perfectly regular is probably not AF.** Reconsider flutter with 2:1 block or SVT.
- Irregular and broad means no AV-nodal blocker.** Procainamide or shock.
- Diltiazem is the wrong drug in HFrEF**, where digoxin or amiodarone is safer.
- Anticoagulation follows CHA₂DS₂-VASc, not the rhythm.** Restoring sinus rhythm does not remove the stroke risk.





NSTEMI & Unstable Angina

NSTE-ACS · Risk stratify → time the cath → treat for life

RISK DRIVES TIMING
not the troponin alone

DIAGNOSE *ischemia without persistent ST elevation*

THE DEFINITION

Rise and/or fall of troponin with at least one value above the 99th percentile, **plus** evidence of ischemia (symptoms, ECG change, imaging). **Unstable angina** = same picture with **normal troponin**.

ECG — SUPPORTIVE, NOT REQUIRED

ST depression, transient elevation, or **deep symmetric T inversion**. A normal ECG never excludes NSTE-ACS. Repeat at **15–30 min** if pain persists, and check posterior leads.

TYPE 1 vs TYPE 2 — ASK FIRST

Type 1 = plaque rupture → this pathway. **Type 2** = supply/demand mismatch (sepsis, anemia, tachyarrhythmia, hypoxia) → **treat the trigger**, not the cath lab.

RISK-STRATIFY, THEN TIME THE ANGIOGRAM *this is the central decision*

Strategy	Who	Why
Immediate < 2 hours	Refractory angina, hemodynamic or electrical instability, mechanical complication , acute HF from ischemia	These patients behave like a STEMI. Do not wait for troponin trends or risk scores.
Early < 24 hours	GRACE score > 140 , or other high-risk features including dynamic ST change and rising troponin	Early invasive reduces recurrent ischemia and MACE in the genuinely high-risk group.
During admission	Intermediate risk, or low risk after further stratification	Invasive with intent to revascularize is recommended in intermediate/high risk to reduce MACE. Low risk may take a selective approach.

USE THE RIGHT SCORE FOR THE RIGHT DECISION

GRACE guides invasive timing (> 140 = early). **TIMI** estimates 14-day risk. **HEART** is an **ED disposition** tool for undifferentiated chest pain — it does **not** decide cath timing in an admitted NSTEMI. Applying one score's cutoff to another's decision is a common and dangerous error.

ANTITHROMBOTICS *load, anticoagulate, then 12 months of DAPT or ticagrelor alone from 1 month*

Agent	Dose	Notes
Aspirin	162–325 mg chewed	Then 81 mg daily indefinitely.
Ticagrelor	180 mg load, 90 mg BID	Preferred over clopidogrel in NSTE-ACS undergoing PCI. Dyspnea via adenosine (ENT1 inhibition).
Prasugrel	60 mg load, 10 mg daily	Also preferred over clopidogrel. Contraindicated with prior stroke/TIA ; avoid ≥ 75 y or < 60 kg. Usually given after anatomy is known.
Anticoagulation	Enoxaparin 1 mg/kg SC BID or UFH 60 U/kg bolus	Continue until PCI or for the hospital stay. Reduce enoxaparin to daily if CrCl < 30.

UPSTREAM P2Y12 — ONLY IF THE CATH IS DELAYED

If angiography is planned **> 24 hours away**, upstream clopidogrel or ticagrelor may be considered to reduce MACE. If the patient is going early, many centers wait for the anatomy, since a surgical lesion means CABG and a loaded P2Y12 forces a delay.

MEDICAL THERAPY *started in hospital, continued for life*

Drug	When	Why / when to hold
Beta-blocker	Oral within 24 h , low dose	Reduces ischemia, reinfarction, arrhythmia. Hold in cardiogenic shock, decompensated HF , bradycardia, or high-grade block.
High-intensity statin	Before discharge, everyone	Atorvastatin 80 mg. Plaque stabilization; do not wait for a lipid panel.
ACE inhibitor / ARB	First 24 h if BP allows	Indicated if LVEF ≤ 40% , hypertension , diabetes , or CKD; reasonable otherwise.
MRA	After ACEi/BB established	Add if LVEF ≤ 40% with HF or diabetes . Hold if K ⁺ > 5.0 or significant renal impairment.
Nitrates	Ongoing ischemic pain	Symptom relief only, no mortality benefit. Avoid with recent PDE5 inhibitor or suspected RV infarct.

TYPE 2 MI *a different disease*

Troponin rises because of **supply–demand mismatch**, not plaque rupture: sepsis, tachyarrhythmia, anemia, hypoxemia, hypotension, hypertensive crisis. **Treatment is the underlying cause**. DAPT and urgent angiography are usually **not** indicated and add bleeding risk. Say which type you mean in the note.

BEFORE DISCHARGE *secondary prevention*

DAPT **12 months**, or **ticagrelor alone from 1 month** after PCI if DAPT is tolerated (both Class 1) · high-intensity statin, adding ezetimibe ± PCSK9i if **LDL ≥ 70** on it (Class 1), reasonable at **55–69** (2a) · beta-blocker · ACEi/ARB · **cardiac rehab referral for every patient** · smoking cessation · BP and diabetes control, consider an **SGLT2 inhibitor**.

BALANCE ISCHEMIA AGAINST BLEEDING *the other half of the decision*

WHAT GRACE USES

Age, heart rate, systolic BP, **creatinine**, Killip class, cardiac arrest at presentation, **ST deviation**, and elevated markers. It estimates in-hospital and 6-month mortality — which is why it, not a chest-pain triage score, drives invasive timing.

HIGH BLEEDING RISK

Older age, low body weight, **CKD**, anemia, prior bleeding or stroke, and need for oral anticoagulation. Consider **radial access**, weight- and renally-adjusted anticoagulation, a PPI with DAPT (Class 1 when GI bleeding risk is high), and a single antiplatelet after 1 month (2b).

IF THEY NEED ANTICOAGULATION TOO

With atrial fibrillation or a mechanical valve, avoid indefinite triple therapy. Usual approach is a short period of triple therapy, then **DOAC plus a single P2Y12 inhibitor** (typically clopidogrel), dropping aspirin early.

PEARLS & PITFALLS

- Do not use a **HEART score to time the cath**. It is an ED disposition tool; GRACE > 140 is the early-invasive threshold.
- A normal ECG does not exclude NSTE-ACS. Repeat it, and get posterior leads if the story fits.
- Persistent ST elevation is a STEMI, not an NSTEMI — stop stratifying and activate the lab.
- Always ask type 1 or type 2** before committing to DAPT and angiography.
- Loading prasugrel before you know the anatomy can delay urgent CABG.
- Rising troponin in a septic patient is usually demand ischemia. Treat the sepsis.

References: 2025 ACC/AHA/ACEP/NAEMSP/SCAI Guideline for the Management of Patients With Acute Coronary Syndromes (JACC / Circulation, 2025) · Fourth Universal Definition of Myocardial Infarction.

Educational aid; verify doses against local protocol and patient factors.





Chronic HFrEF · GDMT

Four pillars · Start all four early, titrate to target, then reassess the EF

ALL FOUR, LOW DOSE
beats two at target

THE FOUR PILLARS *every patient with HFrEF, unless contraindicated*

Pillar	Start	Target	Why
1 • ARNI sacubitril/valsartan or ACEi / ARB	24/26 or 49/51 mg BID	97/103 mg BID	Preferred over ACEi/ARB. 36-hour washout from an ACEi before starting — angio-edema risk. Monitor K ⁺ and creatinine.
2 • Beta-blocker carvedilol, metoprolol succinate, bisoprolol	Carvedilol 3.125 mg BID	Carvedilol 25 mg BID Metoprolol succ. 200 mg Bisoprolol 10 mg	Only these three have mortality evidence in HFrEF — they are not interchangeable with other beta-blockers. Start when euvolemic, not during decompensation.
3 • MRA spironolactone or eplerenone	12.5–25 mg daily	25–50 mg daily	Avoid if K ⁺ > 5.0 or eGFR < 30. Check K ⁺ and creatinine at 1 week, 4 weeks, then periodically.
4 • SGLT2 inhibitor dapagliflozin or empagliflozin	10 mg daily — no titration needed		Reduces hospitalization and cardiovascular death regardless of diabetes . Easiest pillar to start and the one most often forgotten.

HOW TO SEQUENCE *the modern approach is breadth before depth*

- 1 Get all four classes on board at low dose within weeks, rather than maximizing one drug before adding the next. Benefit appears early and is largely independent between classes.
- 2 Start with the two that need no titration and are best tolerated — the SGLT2 inhibitor and a low-dose beta-blocker or ARNI, guided by blood pressure and volume status.
- 3 Titrate every 2 weeks as blood pressure, heart rate, potassium, and renal function allow. Aim for target doses, or the maximally tolerated dose.
- 4 Accept an asymptomatic creatinine rise of up to about 30% and mild asymptomatic hypotension. These are not reasons to abandon therapy that reduces mortality.
- 5 Do not stop GDMT during a hospitalization unless there is shock, symptomatic hypotension, or worsening renal failure. Continuing therapy through admission improves outcomes; drugs stopped in hospital are often never restarted.

ADD-ONS & DEVICES *after the four pillars are optimized*

ADDITIONAL DRUGS

Loop diuretic for congestion — symptom control, not mortality. **Hydralazine plus isosorbide dinitrate** in self-identified Black patients with persistent NYHA III–IV symptoms on optimal therapy. **Ivabradine** if sinus rhythm and heart rate ≥ 70 despite a maximally tolerated beta-blocker. **Vericiguat** after a recent worsening event. **Digoxin** for symptoms and admission reduction only. **IV iron** for symptomatic iron deficiency.

DEVICES — REASSESS AFTER 3 MONTHS OF GDMT

ICD for primary prevention if EF ≤ 35%, NYHA II–III, on optimal therapy for ≥ 3 months, with expected survival > 1 year. **CRT** if EF ≤ 35%, sinus rhythm, LBBB with QRS ≥ 150 ms. **Do not implant early** — a meaningful proportion recover EF above 35% on GDMT and avoid a device altogether.

MONITORING & FOLLOW-UP *the why behind each check*

Check	Interval and reason
Potassium & creatinine	1–2 weeks after every uptitration of ARNI/ACEi or MRA, then periodically. Catching hyperkalemia is what keeps the MRA on board long term.
Blood pressure & heart rate	Every visit — they define the room you have to titrate. Resting HR ≥ 70 in sinus rhythm on a maximal beta-blocker raises the ivabradine question.
Weight & volume status	Daily weights at home with a diuretic action plan. A 2–3 kg rise over days is congestion, not diet.
Repeat echo	After 3 months of optimized therapy — this is the decision point for ICD or CRT, and many EFs improve.
NT-proBNP	Prognostic and useful for trajectory. On sacubitril/valsartan trend NT-proBNP, not BNP — neprilysin inhibition raises BNP by design.

TROUBLESHOOTING *the four reasons GDMT gets abandoned, and what to do instead*

Problem	Do this before stopping anything
Hyperkalemia K ⁺ 5.0–5.5	Stop potassium supplements, NSAIDs, and trimethoprim, and consider a potassium binder to keep the MRA on. Halve the dose before abandoning it. Hold at K⁺ > 5.5.
Hypotension asymptomatic low BP	Asymptomatic systolic in the 90s is acceptable. Separate dosing times, cut the loop diuretic if euvolemic, and stop non-GDMT antihypertensives before touching a pillar.
Rising creatinine	Accept up to about a 30% rise that plateaus. Check volume status — over-diuresis is commoner than the ACEi/ARNI. Recheck in 1–2 weeks rather than stopping.
Fatigue on the beta-blocker	Usually transient over 2–4 weeks . Warn in advance, slow the titration, and do not withdraw early — benefit builds over months.

HFmrEF & HFpEF *brief, because the questions come up together*

HFmrEF (EF 41–49%)

SGLT2 inhibitor is recommended. ARNI/ACEi/ARB, beta-blocker, and MRA **may be considered**, particularly toward the lower end of the range. Treat as HFrEF-like when the EF is trending down.

HFpEF (EF ≥ 50%)

SGLT2 inhibitor is the cornerstone; add an **MRA**, consider ARB/ARNI in selected patients. **Diuretics for congestion**, and treat the drivers: **hypertension, AF, obesity, sleep apnea**. Screen for **amyloidosis** if the phenotype fits.

PEARLS & PITFALLS

- All four pillars at low dose beats two at target dose. Breadth first, then titrate.
- Only carvedilol, metoprolol succinate, and bisoprolol have mortality benefit. Atenolol and metoprolol tartrate are not substitutes.
- A 36-hour washout is mandatory switching from an ACEi to sacubitril/valsartan, or you risk angio-edema.
- On an ARNI, trend NT-proBNP rather than BNP. Neprilysin inhibition raises BNP by design.
- Wait 3 months before implanting a device. Recovery of EF on GDMT is common and spares the patient a device.

References: 2022 AHA/ACC/HFSA Guideline for the Management of Heart Failure (Circulation / JACC, 2022) · 2024 ACC Expert Consensus Decision Pathway for HFrEF ·

PARADIGM-HF, DAPA-HF, EMPEROR-Reduced.

Educational aid; verify doses against local protocol, renal function, and patient factors.

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Syncope

Transient loss of consciousness · Reflex, orthostatic, or cardiac?

HISTORY + EXAM + ECG

the whole foundation

THREE BUCKETS *sorting into one of these drives every subsequent decision*

REFLEX (VASOVAGAL) — commonest, benign

Prodrome: warmth, nausea, diaphoresis, tunnel vision. Provoked by standing, pain, emotion, micturition, cough. Rapid full recovery. **Long-term outcomes match matched controls without syncope.**

ORTHOSTATIC

On standing, within 3 minutes. **Drop of ≥ 20 mmHg systolic or ≥ 10 mmHg diastolic.** Causes: volume depletion, **drugs** (antihypertensives, alpha-blockers, diuretics, nitrates), autonomic failure (diabetes, Parkinson disease).

CARDIAC — the one that kills

Arrhythmic (brady, VT, SVT) or **structural/obstructive** (severe AS, HCM, PE, tamponade, dissection, pulmonary hypertension). Little or no prodrome. Highest mortality — find these.

INITIAL EVALUATION *history, physical exam, and a 12-lead ECG guide everything after*

- 1 **Was it truly syncope?** Transient loss of consciousness with **global cerebral hypoperfusion**, rapid onset, short duration, spontaneous complete recovery. Distinguish from **seizure** (postictal confusion, tongue biting, prolonged), hypoglycemia, and psychogenic pseudosyncope.
- 2 **Get the story from a witness.** Position, trigger, prodrome, duration, color, movements, and how fast they came round. This is worth more than any test.
- 3 **Orthostatic vitals** and a full cardiac exam — murmur, carotid upstroke, signs of heart failure.
- 4 **12-lead ECG in everyone.** Look for conduction disease, ischemia, LVH, delta wave, **long or short QT**, Brugada pattern, epsilon waves, and pathological Q waves.
- 5 **Targeted labs only.** Routine broad panels and head CT have low yield. Image the head only with focal deficit or head trauma.

RED FLAGS FOR CARDIAC SYNCOPE *any of these changes the disposition*

THE EVENT

Syncope during exertion or while **supine** · **no prodrome** at all · preceded by palpitations · injury from the fall · recurrent unexplained episodes.

THE PATIENT

Known structural heart disease or reduced EF · older age · **family history of sudden death < 50** or inherited channelopathy · abnormal ECG · heart failure or a significant murmur.

DISPOSITION *driven by serious conditions, not by a single risk factor*

Decision	Basis
Admit	Presence of ≥ 1 serious medical condition identified in the ED is the key determinant of in-hospital management, rather than any individual risk factor in isolation. This includes arrhythmia, ischemia, severe AS, PE, dissection, GI bleeding, and significant volume loss.
Discharge	Presumptive vasovagal syncope should be discharged from the ED. Reassurance and counseling, not admission.
Structured outpatient	Suspected cardiac syncope is seldom admitted purely for diagnostics in the outpatient world, and it is reasonable to extend that approach from the ED once initial evaluation is complete — provided reliable follow-up and monitoring are arranged.

TARGETED TESTING & TREATMENT *choose the test the history points to*

TESTS

Echo if structural disease is suspected. **Ambulatory monitoring matched to frequency:** Holter for daily symptoms, patch or external monitor for weekly, **implantable loop recorder for infrequent unexplained** episodes. Exercise testing for exertional syncope. Tilt table for suspected reflex when the diagnosis is unclear.

TREATMENT

Reflex: education, avoid triggers, **fluid and salt, counter-pressure maneuvers**, consider midodrine or fludrocortisone if recurrent. **Orthostatic: stop the offending drug first**, compression stockings, slow position change. **Cardiac:** treat the cause — pacemaker, ICD, ablation, or valve intervention.

IS IT ACTUALLY SYNCOPE *the three mimics that get mislabelled*

Condition	Discriminating features	Why it matters
Seizure	Postictal confusion lasting minutes, lateral tongue biting, incontinence, prolonged event, head turning, cyanosis. Brief myoclonic jerks alone favor syncope.	Completely different workup and driving restrictions. Postictal confusion is the single best discriminator.
Psychogenic pseudosyncope	Frequent events, prolonged apparent unconsciousness with normal vitals and normal color, eyes closed, resistance to eye opening.	Avoids an endless negative cardiac workup. Needs psychiatric input, not a loop recorder.
Metabolic / hypoglycemia	Gradual onset, no rapid spontaneous recovery, sweating and confusion that persists until treated.	By definition not syncope, since recovery is not spontaneous. Check a glucose in everyone.

BEFORE THEY LEAVE *the counseling that prevents the next event*

COUNSEL EVERY PATIENT

Teach **recognition of the prodrome** and to **lie down immediately** rather than fight it. **Counter-pressure maneuvers:** leg crossing with tensing, handgrip, arm tensing. Increase fluid and salt unless contraindicated. Rise slowly. Avoid prolonged standing, heat, and alcohol.

DRIVING & OCCUPATION

Ask about driving and the patient's job — and document the advice. Restrictions vary by jurisdiction and by cause, and are longer for untreated cardiac syncope than for a single vasovagal faint. Occupational risk matters for drivers, pilots, and those working at height or with machinery.

PEARLS & PITFALLS

- **Exertional syncope is an emergency workup**, not reassurance — severe AS, HCM, anomalous coronary, or exercise-induced arrhythmia.
- **Syncope with no prodrome, or while supine**, is arrhythmic until proven otherwise. A prodrome is protective information.
- Brief myoclonic jerks are common in vasovagal syncope and do **not** make it a seizure. Postictal confusion is the discriminator.
- **Review the medication list before anything else** in the older patient. Polypharmacy is the commonest reversible cause.
- **Head CT and routine bloodwork are low yield.** The ECG and the history are where the answer is.
- Ask about **family sudden death under 50** — it reframes an otherwise benign-sounding faint.





STEMI

ST-Elevation MI · Recognize → Reperfuse → Protect the myocardium

TIME IS MUSCLE
every minute counts

DIAGNOSE *the ECG decides — never wait for troponin*

ST-ELEVATION CRITERIA

New STE at the J point in **≥ 2 contiguous leads. ≥ 1 mm** in all leads except V2–V3, where it is **≥ 2 mm** (men ≥ 40 y), **≥ 2.5 mm** (men < 40 y), **≥ 1.5 mm** (women).

EQUIVALENTS — DO NOT MISS

Posterior: depression V1–V3 → get V7–V9. **New LBBB** + ischemic symptoms (apply Sgarbossa). **de Winter T** waves. **aVR elevation** + diffuse depression = left main.

TERRITORY → VESSEL

II, III, aVF inferior (RCA) — **always add V3R/V4R**. **V1–V4** anterior (LAD). **I, aVL, V5–V6** lateral (LCx). **V1–V3 depression** posterior.

REPERFUSE *the one decision that saves myocardium*

- 1 Activate the cath lab now.** Primary PCI is preferred. Target **first-medical-contact-to-device ≤ 90 min** at a PCI center, **≤ 120 min** if transferring. **Radial access preferred over femoral** — less bleeding, fewer vascular complications, lower mortality.
- 2 PCI not achievable within 120 min?** Give a fibrinolytic **within 30 min of arrival**, then transfer for angiography regardless of response. Fibrinolysis has **no role in NSTEMI-ACS**.
- 3 Multivessel disease:** revascularize significant non-culprit lesions, favoring **a single procedure** over staged in the stable patient.
- 4 Comatose after arrest with ROSC + STEMI** → EMS routes directly to a primary-PCI center.

FIBRINOLYSIS — ABSOLUTE CONTRAINDICATIONS

Any prior **intracranial hemorrhage** · ischemic stroke < 3 months · known intracranial neoplasm or AVM · suspected **aortic dissection** · active bleeding or bleeding diathesis · significant closed head/facial trauma < 3 months. *Tenecteplase single weight-based bolus is the usual agent. Reperfusion suggested by ≥ 50% ST resolution at 60–90 min.*

ANTITHROMBOTICS *load early · 12 months of DAPT, or ticagrelor alone from 1 month*

Agent	Dose	Why / caution
Aspirin	162–325 mg chewed	Then 81 mg daily indefinitely. Chewed for faster absorption.
Ticagrelor	180 mg load, 90 mg BID	Preferred over clopidogrel. Dyspnea via adenosine (ENT1 inhibition). Avoid with prior ICH.
Prasugrel	60 mg load, 10 mg daily	Also preferred over clopidogrel. Contraindicated with prior stroke/TIA ; avoid if ≥ 75 y or < 60 kg.
Clopidogrel	600 mg load, 75 mg daily	Reserve for high bleeding risk, need for anticoagulation, or after fibrinolysis.
Heparin	60 U/kg bolus (max 4000)	12 U/kg/h infusion; titrate to ACT in the lab. Bivalirudin an alternative.

DAPT DURATION

Aspirin + P2Y12 for **at least 12 months** when bleeding risk is not high (Class 1). **Equally Class 1 after PCI:** if ticagrelor DAPT is tolerated, stop aspirin at **≥ 1 month** and continue **ticagrelor alone** (TWILIGHT: 44% less bleeding, no excess ischemia). **High bleeding risk:** a single antiplatelet after 1 month may be reasonable (2b). Never stop all antiplatelets after a stent without cardiology: stent thrombosis is often fatal.

IN-HOSPITAL MEDICAL THERAPY *start before discharge — this is where mortality is won*

Drug class	When to start	Why / when to hold
Beta-blocker metoprolol tartrate 12.5–25 mg PO	Oral within 24 h , low dose, escalate slowly	Reduces ventricular arrhythmias and reinfarction. Hold or stop if cardiogenic shock, new or worsening HF , bradycardia, or heart block. Avoid IV in unstable patients.
High-intensity statin atorvastatin 80 mg	Before discharge, in everyone	Plaque stabilization and event reduction independent of baseline LDL. Do not wait for a lipid panel.
ACE inhibitor / ARB lisinopril 2.5–5 mg	First 24 h once BP tolerates	Indicated if LVEF ≤ 40%, hypertension, diabetes, or anterior STEMI ; reasonable in everyone else. Watch Cr and K ⁺ .
MRA eplerenone 25 mg	Post-MI, after ACEi/BB established	Add if LVEF ≤ 40% with HF or diabetes . Contraindicated if K ⁺ > 5.0 or significant renal impairment.
Nitrates / morphine	Symptom control only	No mortality benefit. Nitrates contraindicated in RV infarct and with recent PDE5 inhibitor. Morphine blunts P2Y12 absorption — refractory pain only.
Oxygen	Only if SpO ₂ < 90%	Routine oxygen in the normoxic patient may increase infarct size.

CARDIOGENIC SHOCK *7–10% of ACS*

Emergency revascularization of the culprit vessel is the intervention that changes survival — do not delay it. **Do not routinely PCI non-culprit vessels** in shock. A **microaxial flow pump** is reasonable in selected patients to reduce mortality, accepting more bleeding, limb ischemia, and renal failure. **Stop beta-blockers.** Norepinephrine is the usual first-line pressor.

COMPLICATIONS *think days 2–7*

Mechanical (rarer with timely reperfusion): **VSR** — new harsh murmur + thrill, O₂ step-up RA→RV. **Papillary muscle rupture** — acute severe MR and flash edema, usually inferior MI. **Free wall rupture** — tamponade, PEA arrest. All need **emergency surgery**. **RV infarct:** hypotension + clear lungs + raised JVP → **fluids**, avoid nitrates and diuretics.

BEFORE DISCHARGE *secondary prevention is where the next 5 years are decided*

MEDICATIONS

DAPT · high-intensity statin, then ezetimibe ± PCSK9i if **LDL ≥ 70** (Class 1) or **55–69** (2a) · beta-blocker · ACEi/ARB · MRA if **EF ≤ 40%** with HF or DM.

REASSESS THE EF

Repeat echo. If **EF ≤ 35% at ≥ 40 days** post-MI (or ≥ 90 days post-revascularization) on optimal therapy → **refer for primary-prevention ICD**.

CARDIAC REHAB

Refer **every** patient. Reduces cardiovascular mortality and readmission.

RISK FACTORS

Smoking cessation is the single highest-yield action. Control BP and diabetes, consider an **SGLT2 inhibitor** if diabetic or EF reduced.

PEARLS & PITFALLS

- **Do not delay the cath lab to "stabilize":** reperfusion is the stabilization.
- A normal first troponin never excludes STEMI; the ECG decides.
- Isolated ST depression V1–V3 = posterior STEMI until posterior leads prove otherwise.
- **Nitrates in RV infarct cause profound hypotension** — the RV is preload-dependent. Give fluids.
- Beta-blockers early are good; beta-blockers in shock are harmful. Assess perfusion first.





Valvular Heart Disease

AS · MR · AR · MS · Grade the lesion, then time the intervention

SYMPTOMS CHANGE EVERYTHING

and so does the EF

AORTIC STENOSIS *the commonest valve lesion you will admit*

Stage	Echo	What to do
Severe (C/D)	Vmax ≥ 4.0 m/s, mean gradient ≥ 40 mmHg, AVA ≤ 1.0 cm ²	Symptomatic → replace. Angina, dyspnea, or syncope on exertion is the trigger.
Very severe	Vmax ≥ 5.0 m/s or mean gradient ≥ 60 mmHg	Intervene even if asymptomatic (Class 2a), as are rapid progression, abnormal exercise test, and elevated BNP.
Asymptomatic severe	With LVEF < 50%	Replace — a falling EF in severe AS is decompensation, not incidental.
Low-flow, low-gradient	AVA ≤ 1.0 cm ² but mean gradient < 40 with reduced EF	Dobutamine stress echo to separate true severe AS from pseudo-severe. This changes the operation.

TAVR OR SAVR — LARGELY AN AGE AND LIFE-EXPECTANCY DECISION

< 65 y, or life expectancy > 20 y → **SAVR preferred** (durability). 65–80 y → **either**, by shared decision making. > 80 y, or life expectancy < 10 y → **transfemoral TAVR preferred**. SAVR is also favored if vascular anatomy precludes transfemoral access, or when intervening on an asymptomatic patient.

MITRAL REGURGITATION *primary and secondary are different diseases*

PRIMARY (degenerative) — fix the valve

Severe = EROA ≥ 0.4 cm², regurgitant volume ≥ 60 mL, regurgitant fraction ≥ 50%. **Operate if symptomatic**, or asymptomatic with LVEF ≤ 60% or LVESD ≥ 40 mm. **Repair beats replacement** when feasible. Waiting for a low EF is waiting too long.

SECONDARY (functional) — treat the ventricle

The valve is structurally normal; the **ventricle is the problem**. **Optimize GDMT first**, plus CRT if indicated. Transcatheter edge-to-edge repair is for persistent severe MR with symptoms **despite maximally tolerated GDMT** and suitable anatomy. Surgery alone does not fix the ventricle.

AORTIC REGURGITATION & MITRAL STENOSIS *the other two*

Lesion	Intervene when	Notes
Chronic severe AR	Symptomatic , or asymptomatic with LVEF ≤ 55% or LVESD > 50 mm	Track LV size and EF on serial echo — AR is a slow, silent dilator. Vasodilators do not delay surgery in asymptomatic disease.
Acute severe AR	Emergency surgery	Think endocarditis or aortic dissection . Sudden pulmonary edema with a soft murmur and no time to dilate. Avoid beta-blockade , which removes the compensatory tachycardia.
Severe MS	MVA ≤ 1.5 cm ² with symptoms	Almost always rheumatic . Percutaneous balloon commissurotomy if favorable morphology, no left atrial thrombus , and no more than mild MR. Otherwise surgery.

THE MURMUR, AND WHAT ELSE TO DO *bedside first*

LOCALIZE IT

AS: crescendo-decrescendo, right upper sternal border, radiates to carotids, **soft S2 and delayed carotid upstroke = severe**. **MR**: holosystolic, apex, radiates to axilla. **AR**: early diastolic decrescendo, wide pulse pressure. **MS**: opening snap plus diastolic rumble.

ALONGSIDE THE VALVE

Refer to a multidisciplinary heart valve team for severe disease. Treat comorbid HF and atrial fibrillation. **Endocarditis prophylaxis** only for prosthetic valves or prior IE before dental work. Serial echo for asymptomatic severe disease. Prosthetic valve: **warfarin for mechanical valves — DOACs are contraindicated**.

PROSTHETIC VALVES *the questions you will actually be asked on call*

Question	Answer
Mechanical or bioprosthetic?	Mechanical lasts longer but requires lifelong warfarin — favored in younger patients. Bioprosthetic avoids long-term anticoagulation but structurally degenerates — favored in older patients or when anticoagulation is unsafe.
Which anticoagulant?	Warfarin only for mechanical valves — DOACs are contraindicated and were associated with harm. Target INR depends on valve type and position: roughly 2.5–3.5 for mechanical mitral , 2.0–3.0 for mechanical aortic . Add low-dose aspirin in selected patients.
Needs a procedure?	Interrupt warfarin for major surgery and bridge with heparin for high thrombotic risk (mechanical mitral, older prostheses, prior thromboembolism). Minor dental work often needs no interruption.
Fever with a prosthesis?	Three sets of blood cultures and a TEE . Prosthetic valve endocarditis has high mortality and a low threshold for surgery. A new murmur or conduction delay suggests dehiscence or abscess.
New dyspnea years later?	Think structural valve degeneration , prosthetic stenosis, pannus, thrombus, or paravalvular leak. Get an echo and compare with the baseline post-operative study.

SURVEILLANCE IN ASYMPTOMATIC DISEASE *how often to re-image*

ECHO INTERVALS

Severe asymptomatic disease: every **6–12 months**. **Moderate**: every **1–2 years**. **Mild**: every **3–5 years**. Re-image sooner for any change in symptoms or exam — the interval is a floor, not a rule.

WHAT PROMPTS EARLIER ACTION

New symptoms on careful questioning — patients self-limit activity and call it ageing, so ask what they have stopped doing. Also a falling EF, rising pulmonary pressures, new atrial fibrillation, or rapid hemodynamic progression.

PEARLS & PITFALLS

- Nitrates and other vasodilators are dangerous in severe AS**. A fixed obstruction cannot raise output when you drop afterload, so pressure falls and coronary filling suffers.
- Do not wait for a low EF**. In severe AS or primary MR, an EF that has started to fall means the ventricle is already injured.
- A soft, late-peaking murmur with a delayed carotid upstroke is **more severe**, not less — loudness is unreliable.
- DOACs are contraindicated with mechanical valves**. Warfarin only.
- Low-gradient AS with a poor EF needs a **dobutamine stress echo** before you conclude the valve is fine.
- In secondary MR, **optimize GDMT before** considering the valve. The problem is the ventricle.



DIAGNOSE *ESC: any 2 of 4*

1 • PERICARDITIC CHEST PAIN

Sharp and **pleuritic, worse lying flat, relieved sitting forward**. Present in the large majority of cases and usually the strongest clue.

2 • FRICTION RUB

Scratchy, triphasic, best at the left sternal border with the patient leaning forward. **Highly specific but evanescent** — listen repeatedly.

3 • ECG | 4 • EFFUSION

Diffuse concave ST elevation with PR depression (and PR elevation in aVR). Plus a **new or worsening pericardial effusion** on echo.

THE CRITICAL DISTINCTION — PERICARDITIS vs STEMI

Pericarditis: ST elevation is **diffuse and does not respect a coronary territory**, concave upward, with **PR depression** and **no reciprocal change** (except aVR/V1). **STEMI:** regional, convex, with **reciprocal depression** and evolving Q waves. When genuinely unsure, treat as STEMI and get the angiogram — the cost of missing an occlusion is far higher.

TREAT *anti-inflammatory plus colchicine — both, not either*

Drug	Dose	Why
Aspirin first-line, and preferred post-MI	750–1000 mg every 8 h for 1–2 weeks, then taper	Choose aspirin over other NSAIDs when pericarditis follows an MI, since NSAIDs may impair infarct healing.
Ibuprofen alternative first-line	600 mg every 8 h for 1–2 weeks, then taper	Equivalent efficacy. Duration guided by symptoms and CRP , generally 1–2 weeks in uncomplicated disease.
Colchicine add to every case	0.5 mg daily if < 70 kg 0.5 mg twice daily if ≥ 70 kg for 3 months	First-line adjunct , not an alternative. Substantially reduces recurrence. Tapering is not mandatory but may be considered. Reduce dose in renal impairment; expect diarrhea.
Gastric protection	PPI alongside high-dose NSAID	These are large anti-inflammatory doses given for weeks.
Corticosteroids avoid routinely	Low-to-moderate dose only if truly needed	Associated with more recurrences and steroid dependence. Reserve for NSAID/colchicine failure, contraindication, or a specific indication such as autoimmune or uremic pericarditis.

FOLLOW-UP & RECURRENCE *CRP is the taper guide*

MONITOR

Track CRP to guide duration — taper only after symptoms resolve **and** CRP normalizes. Restrict strenuous exercise until both settle. Recurrence affects roughly **15–30%**, and is higher if colchicine was omitted.

RECURRENT DISEASE

Aspirin or an NSAID until symptoms resolve, **plus colchicine for 6 months**. Some advocate longer, up to 12–24 months after the last recurrence with gradual tapering, since recurrences follow abrupt discontinuation.

RED FLAGS — ADMIT AND INVESTIGATE *most cases are viral and outpatient; these are not*

ADMIT IF

Fever > 38°C · subacute onset · **large effusion or tamponade** · failure of NSAID after 7 days · **immunosuppression** · oral anticoagulation · trauma · elevated troponin (myopericarditis).

COMPLICATIONS

Tamponade — hypotension, raised JVP, muffled sounds, pulsus paradoxus > 10 mmHg → urgent **pericardiocentesis**. **Constrictive pericarditis** — late, with right-heart failure and a pericardial knock. **Myopericarditis** — troponin rise; restrict exercise, avoid high-dose NSAIDs.

FIND THE CAUSE *most are viral or idiopathic — but look for the ones that change treatment*

Cause	Clue	What changes
Viral / idiopathic the large majority	Preceding viral illness, otherwise well, self-limiting	No extensive workup needed. NSAID + colchicine and follow CRP.
Post-MI	Early peri-infarction, or late Dressler weeks after	Use aspirin , not other NSAIDs, which may impair infarct healing. Avoid steroids.
Uremic	Advanced renal failure, often no ECG changes	Treated with intensified dialysis , not NSAIDs. A dialysis indication in its own right.
Tuberculous	Endemic exposure, HIV, subacute with weight loss and fever	Needs pericardial fluid or tissue for diagnosis. High risk of constriction . Antituberculous therapy.
Malignant	Known cancer, large or recurrent bloody effusion	Cytology and drainage. Consider a pericardial window for recurrence.
Autoimmune	Lupus, rheumatoid disease, other connective tissue disease	One of the few settings where corticosteroids are appropriate first-line.
Post-cardiac injury	After surgery, ablation, or device implantation	Colchicine has a role in prevention after cardiac surgery.

TAMPONADE — RECOGNIZE AND DECOMPRESS *a clinical diagnosis, not an echo one*

THE PICTURE

Beck triad: hypotension, raised JVP, muffled heart sounds. Plus **pulsus paradoxus > 10 mmHg**, tachycardia, and electrical alternans or low voltage on ECG. Echo shows **diastolic right-heart collapse** and respiratory variation across the valves.

WHAT TO DO

Urgent pericardiocentesis, echo-guided. **Give fluids** to support preload while preparing. **Avoid intubation and positive-pressure ventilation if you can** — it drops preload further and can precipitate arrest. **Never delay drainage for a trial of diuretics**.

PEARLS & PITFALLS

- **Colchicine is not optional.** Adding it to an NSAID is the single most effective step for preventing recurrence, and it is the most commonly omitted part of the prescription.
 - **Steroids feel effective and cause relapse.** They are a last resort, not a shortcut.
 - Diffuse ST elevation with PR depression and no reciprocal change points away from occlusion — but stay humble.
- **Taper by CRP, not by the calendar.** Stopping while CRP is still elevated invites recurrence.
 - **Post-MI, use aspirin** rather than another NSAID.
 - A rising troponin means myocardial involvement — reclassify as myopericarditis and change the exercise advice.



Bradycardia & Heart Block

AV conduction disease · Is it symptomatic? Is it reversible? Is it infranodal?

SITE DECIDES FATE
nodal vs infranodal

CLASSIFY *where the block sits predicts whether it will progress*

Degree	ECG	Site & behavior
First degree	PR > 200 ms, every P conducts	Usually nodal . Benign in isolation. Marked delay (> 300 ms) can cause pseudo-pacemaker symptoms.
Mobitz I (Wenckebach)	Progressive PR lengthening, then a dropped beat; grouped beating	Nodal (AV node) . Often vagal or drug-related, frequently benign, rarely progresses. Improves with exercise or atropine.
Mobitz II	Constant PR , then a sudden dropped beat; often wide QRS	Infranodal (His-Purkinje) . Unstable, progresses to complete block without warning. Escape rhythm is slow and unreliable .
2:1 block	Every other P conducts — cannot classify by PR pattern alone	Ambiguous. Narrow QRS + PR long favors nodal; wide QRS favors infranodal. Exercise or atropine improves nodal, worsens infranodal .
Third degree (complete)	AV dissociation, atrial rate > ventricular, regular P-P and R-R	Escape site sets the risk: junctional (narrow, 40–60) is more stable than ventricular (wide, 20–40), which is unreliable.

ACUTE MANAGEMENT *treat the patient, not the number*

- 1 **Is the patient unstable?** Hypotension, altered mentation, ischemic chest pain, or acute heart failure. A rate of 38 in a well athlete needs nothing; a rate of 48 with shock needs everything.
- 2 **Atropine 1 mg IV**, repeat every 3–5 min to a maximum of 3 mg. Works on the **AV node** — so it helps nodal block but is **often ineffective in Mobitz II or complete block**, and by speeding the atria it can **worsen** infranodal block.
- 3 **Transcutaneous pacing** if atropine fails — sedate, set rate 60–80, increase output until **electrical and mechanical capture** (confirm with a pulse, not the monitor).
- 4 **Chronotropic infusion** as a bridge: dopamine 5–20 mcg/kg/min or epinephrine 2–10 mcg/min.
- 5 **Transvenous pacing** for persistent unstable bradycardia, then decide on a permanent device once reversible causes are excluded.

FIND THE REVERSIBLE CAUSE *before anyone implants a device*

DRUGS — check the chart first
Beta-blockers, non-dihydropyridine **calcium channel blockers**, digoxin, amiodarone, clonidine, acetylcholinesterase inhibitors. **Glucagon** for beta-blocker toxicity, **IV calcium** for CCB toxicity, **digoxin Fab** for digoxin.

METABOLIC & ISCHEMIC
Hyperkalemia (widening QRS, peaked T) · **hypothyroidism** · hypothermia · **inferior MI** (RCA supplies the AV node; often transient and atropine-responsive) · **anterior MI** with block, which signals extensive infarction and is ominous.

INFILTRATIVE & INFECTIOUS
Lyme carditis — young patient, endemic exposure, high-grade block that usually **resolves with antibiotics**, so do not implant early. Also sarcoidosis, amyloidosis, endocarditis with **root abscess**, myocarditis, post-TAVR or post-surgical.

PERMANENT PACING *symptoms that match, or a block that will not recover*

Indication	Detail
Symptomatic bradycardia	Pace when symptoms correlate temporally with the bradycardia and no reversible cause is found. Correlation is the whole game.
Mobitz II, high-grade, or complete block	Permanent pacing when not attributable to a reversible cause — regardless of symptoms, because progression is unpredictable.
Do not pace	Sleep-related sinus bradycardia or transient sinus pauses during sleep should not be paced unless another indication exists. Asymptomatic Mobitz I generally needs no device.
Choose the right pacing mode	If LVEF 36–50% with AV block and > 40% ventricular pacing expected, use more physiologic activation (CRT or His-bundle pacing) rather than RV pacing, to prevent pacing-induced heart failure.

BLOCK IN ACUTE MI *the territory tells you the prognosis*

INFERIOR MI — usually benign
The **RCA supplies the AV node** in most people. Block is **nodal**, with a narrow escape, often **atropine-responsive**, and typically **resolves within days** of reperfusion. Temporary pacing may bridge; permanent devices are usually unnecessary.

ANTERIOR MI — ominous
Block reflects **extensive septal infarction** destroying the His-Purkinje system. **Infranodal**, wide escape, **atropine will not help**, and it signals a large infarct with high mortality. Prepare for pacing early and expect a permanent device.

AFTER THE DEVICE *what to check and what can go wrong*

Issue	Recognize it
Failure to capture	Pacing spike with no following complex . Causes: lead displacement, rising threshold, hyperkalemia, ischemia, or metabolic derangement.
Failure to sense	Spikes falling where they should not, risking R-on-T . Undersensing means the device is blind to intrinsic beats.
Oversensing	The device inhibits inappropriately (myopotentials, electromagnetic interference) and the patient becomes symptomatically bradycardic.
Early complications	Pneumothorax , hematoma, lead perforation with tamponade , and infection. Get a post-procedure chest X-ray; fever with a device demands blood cultures and an echo.
Pacing-induced cardiomyopathy	Falling EF with a high RV pacing burden — the reason physiologic pacing is preferred when heavy pacing is anticipated.

PEARLS & PITFALLS

- **Atropine can worsen infranodal block**. It speeds the atria while the diseased His-Purkinje system cannot follow, dropping the ventricular rate. Have pads on before you push it.
- **Wide QRS escape = infranodal = unstable**. Narrow junctional escape is comparatively reassuring.
- Always check **potassium and TSH**, and review every rate-slowing drug, before calling it primary conduction disease.
- **Lyme carditis usually recovers** — ask about travel and rash in the young patient with high-grade block before implanting.
- Confirm pacing **capture by palpating a pulse**. Electrical spikes on the monitor without mechanical capture is a trap.
- Block with an **anterior MI** implies a large infarct and carries far worse prognosis than block with an inferior MI.



DEFINE IT AND STAGE IT *hypotension alone is not shock; hypoperfusion is*

Element	What it means, and why
Definition	Sustained hypotension (SBP < 90 for ≥ 30 min, or vasopressors needed to maintain it) plus end-organ hypoperfusion (rising lactate, oliguria, cool or mottled skin, altered mentation) from a cardiac cause . Normotensive cardiogenic shock exists: a patient maintaining pressure by intense vasoconstriction while the lactate climbs is already in shock, and waiting for the number to fall wastes the window.
SCAI stages A to E	A at risk · B beginning (hypotension or tachycardia, no hypoperfusion) · C classic (hypoperfusion, needs pressor, inotrope or support) · D deteriorating despite that · E extremis (arrest, refractory collapse). Stage tracks mortality and dictates tempo: stage C is the point at which delay begins to cost lives, and staging is the language the shock team expects.
Hemodynamics	Low cardiac index with high filling pressures . Serial lactate , ScvO₂ , urine output and mentation are the bedside surrogates. A PA catheter is reasonable when the phenotype is unclear or before committing to mechanical support, because it distinguishes a failing left ventricle from a right ventricle or from unrecognized sepsis.

FIND THE CAUSE *acute MI until proven otherwise, and the mechanical complications are the ones that kill in hours*

ACUTE MI · MOST CASES

Left ventricular pump failure is the commonest mechanism. **ECG and troponin immediately, echo early**. The treatment is **revascularization**, and nothing else on this sheet substitutes for opening the artery.

MECHANICAL · SURGICAL EMERGENCIES

Papillary muscle rupture (acute severe MR), **ventricular septal rupture**, **free-wall rupture** and **tamponade**. Suspect on a **new harsh murmur, sudden deterioration days after an MI, or shock out of proportion to the infarct**. **Echo is the test** and the answer is urgent surgery, not more inotrope.

RIGHT VENTRICLE AND OTHER

RV infarct: hypotension with **clear lungs** and a **raised JVP**; confirm with **right-sided leads (V4R)**. Also **myocarditis**, **arrhythmia**, **tamponade**, **decompensated chronic HF**, **valve failure**, **drug toxicity**, and **takotsubo**.

REVASCULARIZE, AND ONLY THE CULPRIT *the one intervention with a clear mortality benefit*

CULPRIT LESION ONLY

Emergency PCI of the culprit artery is the priority. CULPRIT-SHOCK, NEJM 2017 (n = 706) compared culprit-only PCI against immediate multivessel PCI: **30-day death or renal replacement therapy 45.9% vs 55.4%** (RR 0.83, 95% CI 0.71 to 0.96, P = 0.01). **Doing the non-culprit lesions in the same sitting causes harm**, through contrast load, longer procedures and ischemia in already marginal myocardium. Stage them later if needed.

IF PCI IS NOT AVAILABLE

Transfer for primary PCI rather than settling for lysis where feasible, since fibrinolysis works poorly in a low-flow state. CABG is the answer for **left main or complex multivessel disease**, and is mandatory alongside repair of a **mechanical complication**.

DRUGS AND DEVICES *supportive only: they buy time to fix the cause, and every one carries a cost*

Option	Use	Evidence, and the catch
Norepinephrine <i>first-line pressor</i>	Restore perfusion pressure while you arrange revascularization	Preferred over dopamine, which caused more arrhythmias and, in the cardiogenic shock subgroup of a large randomized trial, higher mortality. Preferred over epinephrine, which produced more refractory shock and lactic acidosis in a dedicated trial stopped early for safety.
Dobutamine or milrinone	Added for low output once pressure is adequate	Both increase output at the price of arrhythmia , vasodilation and myocardial oxygen demand . Milrinone is renally cleared and lasts longer, so it is harder to undo. Neither improves survival; use the lowest dose that restores perfusion.
Fluids	A cautious 250 mL challenge only if underfilled	Most of these patients are congested, not dry. The exception is RV infarct , which is preload dependent.
Microaxial flow pump (Impella CP)	STEMI-related shock , alongside PCI	DanGer Shock, NEJM 2024 (n = 355) is the first device trial to show a mortality benefit: 180-day death 45.8% vs 58.5% (HR 0.74, 95% CI 0.55 to 0.99, P = 0.04, NNT 8). The cost is real: more bleeding, limb ischemia and renal replacement therapy. Now a class 2a recommendation.
VA-ECMO	Refractory shock, arrest, or as a bridge	ECLS-SHOCK, NEJM 2023 (n = 420) found no 30-day mortality benefit in infarct-related shock (47.8% vs 49.0%) with more bleeding and vascular complications . Do not deploy it routinely ; reserve it for selected patients with a defined exit strategy.
Intra-aortic balloon pump	Largely historical in this setting	No mortality benefit in infarct-related shock. Retains a role in mechanical complications such as acute MR or VSD, where afterload reduction genuinely helps.

TAILOR TO THE MECHANISM

RV INFARCT

Preload dependent: give fluid, and avoid nitrates, morphine and diuretics, which drop preload and can cause abrupt collapse. **Maintain AV synchrony**, so cardiovert atrial fibrillation and pace AV sequentially rather than ventricular only.

ARRHYTHMIA

Synchronized cardioversion for any unstable tachyarrhythmia rather than a drug trial. Pace symptomatic bradycardia, and **correct potassium and magnesium**, since shock and catecholamines make the myocardium irritable.

CALL THE SHOCK TEAM EARLY

Escalate at stage C, not at stage E. Deciding on mechanical support after cardiac arrest or multi-organ failure is deciding too late, and the decision needs cardiology, cardiac surgery and intensive care in the room together.

PEARLS & PITFALLS

- **Open the culprit and nothing else**. Same-sitting multivessel PCI increases death or dialysis.
- **A normal blood pressure does not exclude shock**. Follow the lactate and the urine output.
- **A new murmur with sudden deterioration is a mechanical complication** until echo says otherwise. It is surgical.
- **Devices support, they do not treat**. Only DanGer Shock has shown a survival benefit, and it carries real harms.



Aortic Dissection

Acute aortic syndrome · Rate first, then pressure, then the surgeon

1% MORTALITY / HOUR
untreated type A

SUSPECT IT *the great mimic — missed in up to a third of cases*

THE STORY

Abrupt, maximal-at-onset tearing or ripping chest or back pain, often migrating. **Severity at onset** is the clue — MI pain crescendos, dissection pain peaks instantly.

EXAMINE FOR

Pulse or BP differential between arms (> 20 mmHg) · new **aortic regurgitation** murmur · **focal neuro deficit** or syncope · limb or mesenteric ischemia · tamponade.

CONFIRM

CT angiography of chest/abdomen/pelvis is the test of choice. **TEE** if too unstable to travel. **A normal D-dimer does not exclude it** — never rule out on D-dimer alone.

CLASSIFY — THIS DECIDES EVERYTHING *Stanford*

TYPE A — involves the ascending aorta

Surgical emergency. Call cardiothoracic surgery immediately, in parallel with medical therapy. Mortality rises roughly **1% per hour** untreated. **Immediate operative repair** is indicated, including when there is renal, mesenteric, or lower-extremity malperfusion.

TYPE B — descending only, distal to left subclavian

Medical therapy is first-line for uncomplicated disease. **Intervene** for rupture, branch occlusion with malperfusion, dissection extension, progressive aortic enlargement, **intractable pain, or uncontrolled hypertension**. TEVAR is preferred over open repair with suitable anatomy.

MEDICAL MANAGEMENT *the order of drugs matters more than the drugs*

- 1 **Beta-blocker FIRST** — IV esmolol, labetalol, or metoprolol. Target **heart rate 60–80/min**. Lowering rate lowers dp/dt, the shear force propagating the dissection.
- 2 **Then add a vasodilator** if pressure remains high — nicardipine or nitroprusside. Target **SBP < 120 mmHg**, or the lowest pressure that still perfuses brain, kidneys, and gut.
- 3 **Never give a vasodilator before rate control.** Unopposed vasodilation causes reflex tachycardia, raises dp/dt, and can extend the dissection.
- 4 **Analgesia** — IV opioid. Pain drives catecholamines, tachycardia, and hypertension, so treating it is part of the hemodynamic plan.
- 5 **If beta-blockade is contraindicated** (severe asthma, high-grade block), use a non-dihydropyridine calcium channel blocker such as diltiazem or verapamil for rate control.

WHEN NOT TO LOWER THE PRESSURE

If the patient is **hypotensive or in shock**, think **tamponade, aortic rupture, or severe AR** — not overtreatment. Resuscitate, get an emergent echo, and go to the operating room. **Do not perform pericardiocentesis** for tamponade from type A dissection unless in extremis; draining it can worsen bleeding. The fix is surgical.

LONG-TERM *this is a lifelong aortic disease*

Element	Detail
Beta-blockade for life	Long-term beta-blockers are recommended after the initial treatment of an acute aortic syndrome. Target strict BP control.
Surveillance imaging	CT or MR at intervals after discharge, then lifelong, watching for aneurysmal degeneration of the false lumen.
Screen the family	First-degree relatives of patients with heritable thoracic aortic disease should be screened. Ask about Marfan, Loeys-Dietz, vascular Ehlers-Danlos, and bicuspid aortic valve.
Activity	Avoid heavy isometric lifting and competitive/contact sport, which spike aortic wall stress.

MALPERFUSION — WHICH BRANCH IS THE FLAP OCCLUDING *this is how dissection kills organs*

Territory	How it presents	What it changes
Coronary	Inferior STEMI pattern — the flap most often involves the RCA ostium	Do not thrombolyse or fully anticoagulate. This is the single most lethal misdiagnosis in dissection.
Cerebral / carotid	Stroke, syncope, or depressed consciousness at onset	Chest pain with a focal deficit means CT angiography of the aorta, not a lone stroke pathway.
Spinal	Paraplegia or sensory level from artery of Adamkiewicz involvement	Painless leg weakness after chest pain is dissection until excluded.
Renal	AKI, oliguria, severe renin-mediated hypertension	Pressure that will not come down despite adequate therapy may be renal malperfusion, not undertreatment.
Mesenteric	Pain out of proportion to exam, rising lactate	High mortality. An indication for urgent intervention in type B disease.
Limb	Cold, pulseless, painful extremity	Document pulses in all four limbs on arrival, and recheck — the flap can move.

WHILE YOU WAIT FOR THE SURGEON *parallel tasks, not sequential*

RESUSCITATION SETUP

Two large-bore IVs, arterial line (use the arm with the **higher** pressure), continuous ECG. **Type and crossmatch 4–6 units**. Keep the patient NPO. Send CBC, chemistry, lactate, troponin, and coagulation studies.

DO NOT

Do not give **thrombolytics** or therapeutic anticoagulation. Do not delay CT angiography for a D-dimer. Do not let a normal ECG or chest X-ray reassure you. Do not start a vasodilator before rate control.

PEARLS & PITFALLS

- **Dissection can cause a STEMI** when the flap occludes a coronary, usually the RCA giving an inferior pattern. **Thrombolysis or full anticoagulation here is catastrophic.**
- Never exclude dissection on a normal D-dimer or a normal chest X-ray. A widened mediastinum is neither sensitive nor specific.
- Pain that migrates from chest to back to abdomen is dissection until proven otherwise.
- **Rate before pressure, always.** Nitroprusside first is a classic and harmful error.
- Check BP in **both arms** and document it. A differential can be the only physical clue.
- Syncope, stroke, or acute limb ischemia with chest pain should trigger CT angiography, not a stroke pathway alone.

