STEM-S

Last Minute Revision (LMR)

CARDIOLOGY

ISCHEMIC HEART DISEASE

STABLE ANGINA - PATHOPHYSIOLOGY & MANAGEMENT

UNSTABLE ANGINA / NSTEMI – MANAGEMENT ALGORITHM

Dual Antiplatelet Therapy (DAPT)

- Aspirin: Lifelong (irreversible COX-1 inhibition).
- P2Y12 inhibitors:

1)Clopidogrel: Prodrug, CYP2C19-dependent (loss-of-function alleles efficacy).
2)Prasugrel: Contraindicated in prior stroke/TIA; avoid if age >75 or weight <60 kg.
3)Ticagrelor: Reversible, faster onset; dyspnea via adenosine potentiation.

Pathophysiology

- Plaque rupture + non-occlusive thrombus (vs total occlusion in STEMI).
- Troponin positive → NSTEMI, negative → UA.

Anticoagulation

- Preferred: Enoxaparin or fondaparinux (lower bleeding risk).
- Fondaparinux contraindicated in PCI → add UFH bolus to avoid catheter thrombosis.
- Bivalirudin: Alternative in heparin-induced thrombocytopenia.

Risk Stratification

- TIMI score: Age ≥65, ≥3 risk factors, prior CAD, ST deviation, ≥2 anginal episodes/24 h, aspirin use, ↑ biomarkers.
- GRACE score: Age, HR, BP, creatinine, ST deviation, cardiac arrest, Killip class, troponin → guides early invasive approach if >140.

Common Mixed Acid–Base Disorders

Condition	Mixed Disorder
Salicylate poisoning	High AG metabolic acidosis + respiratory alkalosis
COPD with diarrhea	Respiratory acidosis + metabolic acidosis
Vomiting + COPD	Metabolic alkalosis + respiratory acidosis
DKA with vomiting	High AG metabolic acidosis + metabolic alkalosis
Sepsis	Lactic acidosis + respiratory alkalosis

Finerenone:

- A non-steroidal mineralocorticoid receptor antagonist (MRA).
- More selective + less hyperkalemia than spironolactone.

Benefits

↓ Risk of kidney failure.
↓ Albuminuria significantly.
↓ CV events (especially HF hospitalization).

Mechanism

- Blocks mineralocorticoid receptor–mediated inflammation and fibrosis.
- Acts in kidney, heart, and vasculature.

Indications (Evidence-Based)

From FIDELIO-DKD and FIGARO-DKD:

- Type 2 diabetes with CKD (albuminuria) despite maximally tolerated ACEI/ARB.
- Slows CKD progression.
- Reduces cardiovascular events (HF hospitalization, CV death).

Dosing

Based on eGFR:
- eGFR ≥60 → 20 mg daily
- eGFR 25–60 → 10 mg daily, titrate to 20 mg
- Not used in eGFR <25.

Monitoring

- Potassium (major concern though less than spironolactone). (4 monthly)
- Serum creatinine/eGFR.

Contraindications

- Hyperkalemia at baseline.
- Concurrent strong CYP3A4 inhibitors.
- eGFR <25 mL/min/1.73 m².

STEMI – DIAGNOSIS & REPERFUSION STRATEGY

Diagnosis

- ST elevation: ≥1 mm in limb leads or ≥2 mm in precordial (V3, V4) leads (men <40 yrs – 2.5 mm; women – 1.5 mm).
- Posterior MI: ST depression in V1–V3 with tall R waves (mirror image).
- STEMI equivalents:

1)Wellens' syndrome: Biphasic or deep symmetric T in V2–V3 (critical LAD).
2)de Winter pattern: Up-sloping ST depression + peaked T in precordial leads.
3)LBBB with Sgarbossa criteria.

Post-MI Management

- Beta-blockers: Start after stabilization; avoid in HR <50, hypotension, heart block, shock.
- ACE inhibitors/ARBs: Within 24 h, especially if EF <40% or anterior MI; avoid if Cr >2.5 mg/dL or K□ > 5.5.
- Aldosterone antagonist: Indicated if EF <40% + HF or DM (avoid if Cr >2.5 or K□ >5).
- Statins: High-intensity (rosuvastatin/atorvastatin).
- Nitrates: Only for persistent pain/HTN/pulmonary congestion.

Reperfusion

- PCI preferred if <120 min delay from first medical contact.
- Fibrinolysis if PCI unavailable within 120 min and <12 h symptom onset.
  Absolute contraindications: Prior ICH, intracranial lesion, active bleeding, aortic dissection.
- Post-lysis rescue PCI: If <50% ST resolution in 60-90 min

KILLIP CLASSIFICATION (For prognosis associated with post-MI heart failure)

Class	Description	Mortality
I	No HF	<5%
II	S3 gallop/rales/JVP ↑	10–15%
III	Pulmonary edema	20–30%
IV	Cardiogenic shock	40–70%

CARDIAC BIOMARKERS – KINETICS

Marker	Rise	Peak	Normalizes	Note
Troponin I/T	3–6 h	12–24 h	7–10 d	Highest sensitivity/specificity
CK-MB	3–6 h	24 h	48–72 h	Detects reinfarction
Myoglobin	1–2 h	6–8 h	24 h	Earliest but nonspecific

COMPLICATIONS OF MI

Mechanical

- Papillary rupture: 2–7 days, severe MR, new murmur, pulmonary edema.
- Septal rupture: Harsh holosystolic murmur, shock; O_2 step-up in RV.
- Free wall rupture: Sudden tamponade, PEA arrest.
- LV aneurysm: Persistent ST elevation >2 weeks; risk of emboli/VT.

ARRHYTHMIC

- Most common cause of early death: VT/VF within 24 h.
- Accelerated idioventricular rhythm: Reperfusion marker, benign.

PERICARDITIS

Early: 1–3 days (fibrinous).
Dressler's: Autoimmune, 2–6 weeks post-MI.

SILENT ISCHEMIA & MICROVASCULAR ANGINA

- **Silent ischemia: Common in diabetics due to autonomic neuropathy; detected on Holter/ST-segment monitoring.**
- **Microvascular angina (Cardiac Syndrome X): Normal coronaries with impaired small-vessel dilation; ST depression on stress test; Rx – beta-blockers, nitrates often less effective, consider ranolazine.**

Causes of Troponin I Elevation Other than MI

Cardiac Causes:

- **Myocarditis**
- **Heart failure (acute/chronic)**
- **Takotsubo (stress) cardiomyopathy**
- **Tachyarrhythmias (AF with RVR, SVT)**
- **Hypertensive emergency**
- **Myocardial contusion or trauma (post-CPR, defibrillation, biopsy, cardiac surgery)**
- **Post–cardiac procedures (PCI, CABG, ablation, pacing)**
- **Infiltrative cardiomyopathy (amyloidosis, sarcoidosis)**

Systemic / Non-Cardiac Causes:

- Pulmonary embolism
- Pulmonary hypertension
- Sepsis
- Shock (hypovolemic, septic, cardiogenic)
- Chronic kidney disease / acute renal failure
- Stroke or subarachnoid hemorrhage
- Pheochromocytoma
- Thyrotoxicosis
- Diabetic ketoacidosis
- Severe anemia
- Cocaine or amphetamine toxicity
- Chemotherapy (anthracyclines, 5-FU)
- Extreme physical exertion or seizures
- Electrical injury or burns

HEART FAILURE:

Classification

HFrEF (Reduced EF)
- LVEF <40%

HFmrEF (Mildly reduced)
- LVEF 41–49%

HFpEF (Preserved EF)
- LVEF ≥50% + ↑ LV filling pressures (by echo or natriuretic peptides)

Diagnostic Criteria for HFpEF

Symptoms/signs of HF
LVEF ≥50%
Elevated natriuretic peptides (BNP >35 pg/mL or NT-proBNP >125 pg/mL)
Structural heart disease (LVH, LA enlargement) or diastolic dysfunction (E/e' >14)

Pharmacologic Therapy (HFrEF)

- ARNI (sacubitril/valsartan): preferred over ACEi; washout 36h after ACEi.
- ACEi/ARB: for symptom and mortality reduction.
- β-blockers (carvedilol, metoprolol succinate, bisoprolol): initiate when euvolemic.
- MRA (spironolactone, eplerenone): add if EF ≤35%.
- SGLT2 inhibitors (dapagliflozin, empagliflozin): mortality benefit independent of diabetes.
- Hydralazine + nitrates: in African Americans or ACEi/ARB intolerance.
- Ivabradine: if sinus rhythm, HR ≥ 70, on maximized β-blocker.
- Vericiguat / Omecamtiv mecarbil: advanced HFrEF options.

CRT & ICD – Indications

- CRT:

LVEF ≤35%, sinus rhythm, NYHA II–IV despite GDMT, QRS ≥150 ms (LBBB).

- ICD:

Secondary prevention – prior VT/VF.
Primary prevention – ischemic or nonischemic cardiomyopathy, LVEF ≤35%, ≥40 days post-MI, NYHA II–III on GDMT.

Diuretic Resistance

- Causes: ↓ renal perfusion, neurohormonal activation, distal nephron adaptation.
- Overcome by:

1)Sequential nephron blockade (add thiazide/metolazone).
2)IV infusion vs bolus loop diuretic.
3)Sodium restriction.
4)Ultrafiltration if persistent congestion.

ACC/AHA & ESC Staging (Flowchart)

Stage (ACC/AHA)	Description	Treatment Focus
A	At risk (HTN, DM, obesity, CAD)	Risk factor modification
B	Structural disease, no symptoms	ACEi/ARB, β-blocker
C	Structural disease + symptoms	GDMT (ACEi/ARB/ARNI + β B + MRA + SGLT2i)
D	Refractory HF	Advanced therapies (LVAD, transplant, palliative)

Biomarkers

Marker	Utility
BNP / NT-proBNP	Diagnosis, prognosis, and therapy response
BNP <100 pg/mL = unlikely HF	NT-proBNP <300 pg/mL = unlikely HF
BNP elevated in renal failure, pulmonary HTN, sepsis	Lower in obesity

Restrictive vs Constrictive Physiology (Echo/Hemodynamics)

Feature	Restrictive (Myocardial)	Constrictive (Pericardial)
LV thickness	↑ (amyloid, fibrosis)	Normal
Pericardium	Normal	Thickened / calcified
Resp variation in mitral inflow	Minimal	>25%
Annulus reversus (TDI e' lateral < septal)	Absent	Present
Ventricular interdependence	Minimal	Marked
BNP	High	Mildly ↑
Kussmaul's sign	May be present	Typically present

Cardiomyopathy Subtypes

- Dilated: Systolic dysfunction, LV dilation; causes – idiopathic, post-viral, toxins, peripartum.
- Hypertrophic: Asymmetric septal hypertrophy, SAM of mitral valve, diastolic dysfunction.
- Restrictive: Rigid ventricles, preserved EF, causes – amyloidosis, sarcoidosis, hemochromatosis.
- Takotsubo: Stress-induced, apical ballooning, mimics STEMI, normal coronaries, reversible.

Cardiac cachexia:

Diagnosis requires ≥5% unintentional weight loss over 6–12 months and at least 3 of the following 5 criteria:

- Decreased muscle strength
- Fatigue or reduced exercise tolerance
- Anorexia (loss of appetite)
- Low fat-free mass index (lean body mass reduction)
- Abnormal biochemistry, including any of:

1) ↑ Inflammatory markers (CRP >5 mg/L, IL-6 >4 pg/mL)
2)Anemia (Hb <12 g/dL)
3)Hypoalbuminemia (<3.2 g/dL)

Mechanical circulatory support devices:

Type	Examples	Duration / Purpose
Short-term (Temporary)	Intra-aortic balloon pump (IABP), Impella, TandemHeart, VA-ECMO	Hours to days; used for cardiogenic shock, bridge to decision or recovery
Intermediate-term	CentriMag, paracorporeal VADs	Days to weeks
Long-term (Durable)	LVAD (HeartMate 3, HeartWare HVAD)	Months to years; bridge to transplant or destination therapy

Indications

A. Temporary / Short-Term

- Cardiogenic shock unresponsive to inotropes/vasopressors
- Post-MI shock (Killip IV)
- Fulminant myocarditis / Takotsubo with shock
- Post-cardiotomy LV failure
- Bridge to recovery / bridge to decision / bridge to transplant

B. Long-Term / Durable

- End-stage (NYHA IV) heart failure refractory to GDMT
- Bridge to transplant – awaiting donor heart
- Destination therapy – not a transplant candidate but life expectancy >1 yr
- Bridge to recovery – reversible causes (myocarditis, peripartum)
- Bridge to candidacy – to allow recovery of organ function pre-transplant

Contraindications

Absolute:

Irreversible end-organ failure (Cr >3 mg/dL, bilirubin >3 mg/dL)
Severe, irreversible neurological injury
Active systemic infection / sepsis
Advanced malignancy or limited life expectancy (<2 yrs)
Severe bleeding diathesis / coagulopathy

Relative:

Severe peripheral vascular disease
Nonadherence / psychosocial limitations
RV failure (for isolated LVAD)
Severe pulmonary hypertension (fixed >5 Wood units)

PROSTHETIC VALVES:

Feature	Mechanical	Bioprosthetic (Tissue)
Material	Pyrolytic carbon, titanium	Porcine/bovine pericardial
Durability	20–30 yrs	8–15 yrs
Anticoagulation	Lifelong (INR 2.5–3.5)	3 months only
Preferred in	Young (<50 yr), already anticoagulated	Elderly (>65 yr), contraindication to anticoagulation
Thrombogenicity	High	Low
Click sound	Present	Absent

VALVULAR HEART DISEASES

Feature	Mitral Stenosis (MS)	Mitral Regurgitation (MR)	Aortic Stenosis (AS)	Aortic Regurgitation (AR)
Normal Valve Area (cm^2)	4–6	4–6	3–4	3–4
Severe Lesion (Valve Area)	≤1.0 cm^2	—	<1.0 cm^2	—
Severe Lesion (Gradient / Velocity)	Mean gradient >10 mmHg	Regurgitant fraction >50%	Mean gradient >40 mmHg, velocity >4 m/s	Regurgitant volume >60 mL, fraction >50%
Key Murmur	Mid-diastolic rumble with presystolic accentuation (best at apex in LLDP)	Pansystolic at apex → axilla	Harsh ejection systolic → carotids	High-pitched early diastolic → LLSB
Special Murmur/Sign	Opening snap after S2 (shorter S2–OS = severe)	—	Gallavardin phenomenon (AS murmur radiating to apex)	Austin Flint murmur – mid-diastolic rumble at apex
Severity Assessment Clue	Short S2–OS interval, loud S1, pulmonary HTN	V wave on PCWP ↑, systolic flow reversal in pulmonary veins	High gradient or jet velocity >4 m/s	LVEDD >65 mm / LVESD >50 mm ("55/50 rule")
AF Occurrence	Very common (↑ LA pressure & dilation)	Late (chronic LA overload)	Rare	Rare
Pulmonary HTN	Very common; poor prognosis	May occur in chronic severe MR	Late stage	Late stage
Surgery Indications	Severe MS (area ≤1.5 cm^2) + symptoms or pulmonary HTN	Symptomatic severe MR or asymptomatic with EF ≤60% / LVESD ≥40 mm	Symptomatic severe AS or EF <50%	Symptomatic severe AR or EF ≤55% / LVESD ≥50 mm
Preferred Intervention	PMBV if pliable, noncalcified valve	Valve repair > replacement if feasible	AVR / TAVR	AVR (surgery of choice)
Hemodynamic Load Type	Pressure overload (LA)	Volume overload (LA & LV)	Pressure overload (LV)	Volume overload (LV)
Echo Key Sign	Diastolic doming of leaflets ("hockey stick")	Regurgitant jet on color Doppler	High peak jet velocity (>4 m/s)	Diastolic flow reversal in descending aorta
Common Misconception (Exam Trap)	Gradient depends on HR — ↑ HR exaggerates severity	Chronic MR may have normal LA pressure initially	Normal EF may mask LV dysfunction (AS)	Bounding pulses (Corrigan) not specific; use LV dimensions for surgery
Unique Sign / Pulse	—	—	Parvus et tardus pulse	Corrigan (water-hammer), Quincke, Duroziez, Hill sign
Drug to Avoid	Tachycardia (β-agonists worsen gradient)	Afterload ↑ agents worsen regurgitation	Vasodilators contraindicated (severe AS)	β-blockers worsen regurgitation (prolong diastole)

ENDOCARDITIS PROPHYLAXIS (AHA/ESC Guidelines)

Indicated only in high-risk patients before high-risk procedures.

High-Risk Cardiac Conditions:

- Prosthetic valves (mechanical or bioprosthetic)
- Previous infective endocarditis
- Certain congenital heart diseases:

1)Unrepaired cyanotic CHD
2)Repaired CHD with prosthetic material (<6 months)
3)Residual defects at/adjacent to prosthetic patch

- Cardiac transplant recipients with valvulopathy

High-Risk Procedures:

- Dental procedures involving gingival tissue, periapical region, or mucosal perforation.
- Not indicated for GI, GU, respiratory, or TEE procedures.

Recommended Regimen:

- Amoxicillin 2 g PO 30–60 min before procedure
- (If allergic) Clindamycin 600 mg PO/IV before procedure.

ARRHYTHMIAS

Type	ECG Findings	Mechanism	Acute Management	Definitive / Chronic
AVNRT	Narrow QRS, P waves buried in/after QRS (“pseudo r’ in V1”)	Dual AV nodal pathway reentry	Vagal → Adenosine	β-blocker / verapamil / RF ablation (curative)
AVRT (Orthodromic)	Narrow QRS, retrograde P after QRS (short RP)	Accessory pathway (WPW)	Adenosine	Ablation of accessory pathway
AVRT (Antidromic)	Wide QRS (delta-like), difficult to distinguish from VT	Antegrade via pathway, retrograde AVN	Procainamide (avoid AV nodal blockers)	Ablation
Atrial Flutter	Sawtooth F waves, atrial rate ~300/min, 2:1 block → HR 150	Reentry in RA	Rate control (βB/CCB), DC cardioversion	Catheter ablation (cavotricuspid isthmus)
Atrial Fibrillation	Irregularly irregular rhythm, no P waves	Multiple reentrant foci in atria	Rate control + anticoagulation, cardiovert if unstable	Long-term anticoagulation, ablation (PV isolation)
Multifocal Atrial Tachycardia	≥3 P-wave morphologies, irregular RR	Seen in COPD, theophylline	Treat underlying cause, Mg^{2+}	—

Broad QRS complex tachycardia: VT or SVT with aberrancy- How to differentiate on ECG?

Clue	Ventricular Tachycardia	SVT with Aberrancy
AV dissociation	Present (P independent of QRS)	Absent
Capture / Fusion beats	Present	Absent
QRS width	>140 ms	Usually <140 ms
Axis	Extreme (“northwest”)	Normal
Concordance (precordial)	All positive or all negative	Absent
RS interval in V1–V6	>100 ms	<100 ms
Morphology in V1	QS or monophasic R	rSR' (RBBB pattern)

Long QT, Brugada and WPW syndromes:

Syndrome	ECG Findings	Risk	Management
Long QT	QTc >460 ms (F), >450 ms (M); T-U fusion	Torsades de pointes	$MgSO_4$ IV, stop offending drugs, isoproterenol/pacing if bradycardic
Brugada	Coved ST ↑ in V1–V3, RBBB-like QRS	VF/sudden death (esp. males, Asians)	ICD (definitive), avoid Na^+ blockers (e.g., flecainide)
WPW (Pre-excitation)	Short PR <120 ms, delta wave, wide QRS	AF → VF risk	Avoid AV nodal blockers, use procainamide or ablation (curative)

ATRIAL FIBRILLATION (AF)

Stroke Risk – CHA_2DS_2-VASc

| C | Congestive HF | 1 || H | Hypertension | 1 || A_2 | Age ≥75 | 2 || D | Diabetes | 1 || S_2 | Stroke/TIA | 2 || V | Vascular disease | 1 || A | Age 65–74 | 1 || Sc | Sex (female) | 1 |

- Score ≥2 (M) / ≥3 (F): Oral anticoagulation indicated.
- Score 1 (M): Consider OAC.

Bleeding Risk – HAS-BLED

| Hypertension, Abnormal renal/liver, Stroke, Bleeding history, Labile INR (like patient on warfarin), Elderly >65, Drugs/alcohol. |

- Score ≥3 □ high bleeding risk; caution with OAC.

ANTICOAGULATION

- NOACs preferred: Apixaban, Rivaroxaban, Dabigatran, Edoxaban.
- Warfarin: Target INR 2–3 (if prosthetic valve or cost issue).
- Bridging: Heparin until INR therapeutic for warfarin.

Heart blocks:

Type	ECG Findings	Site	Prognosis / Treatment
Mobitz I (Wenckebach)	Progressive PR prolongation → dropped QRS	AV node	Benign; pacemaker rarely needed
Mobitz II	Constant PR with dropped beats	His–Purkinje	Risk of complete block → Pacemaker indicated
Complete (3rd-degree)	P–QRS dissociation	Below AV node	Permanent pacemaker mandatory
1° AV block	PR >200 ms	AV node	Observe unless symptomatic

Permanent pacemaker is indicated in Symptomatic bradycardia, SSS, Mobitz II / complete heart block, post-AV node ablation in AF

ANTIARRHYTHMIC DRUGS – Vaughan Williams Classification

Class	Mechanism	Drugs	Notes / Uses
I (Na^+ blockers)	↓ depolarization	IA – Quinidine, Procainamide; IB – Lidocaine; IC – Flecainide	IA – prolong QT; IB – post-MI VT; IC – contraindicated in structural heart disease
II	β-blockers	Metoprolol, Esmolol	Rate control in SVT/AF
III	K^+ channel blockers	Amiodarone, Sotalol, Dofetilide	Prolong QT; risk of Torsades (esp. Sotalol)
Others	Adenosine, Digoxin, $MgSO_4$, Ivabradine	Adenosine – AVNRT; Mg – torsades; Ivabradine – sinus tachycardia	SVT termination, rate control

DRUG-INDUCED ARRHYTHMIAS (QT PROLONGATION)

Drugs Causing Torsades / Long QT:

- Antiarrhythmics: Quinidine, Procainamide, Disopyramide, Sotalol, Amiodarone (less)
- Antibiotics: Macrolides, Fluoroquinolones
- Antifungals: Azoles
- Antipsychotics: Haloperidol, Ziprasidone, Thioridazine
- Antidepressants: TCAs, SSRIs (Citalopram)
- Others: Methadone, Ondansetron, Cisapride

Management: Stop drug → IV Magnesium sulfate, pacing if bradycardic.

HYPERTENSION:

DEFINITION (ACC/AHA 2017 & ESC/ESH 2023)

Category	ACC/AHA (mmHg)	ESC/ESH (mmHg)
Normal	<120 / <80	<130 / <85
Elevated / High-Normal	120–129 / <80	130–139 / 85–89
Stage 1 HTN	130–139 / 80–89	140–159 / 90–99
Stage 2 HTN	≥140 / ≥90	≥160 / ≥100
Isolated systolic HTN	SBP ≥140 & DBP <90	Same

Diagnosis

- Based on average of ≥2 readings on ≥2 separate occasions.
- Home BP: ≥135/85 mmHg = hypertension.
- Ambulatory (ABPM): ≥ 130/80 (daytime) or ≥ 120/70 (nighttime) = hypertension.

HYPERTENSIVE URGENCY vs EMERGENCY

Feature	Urgency	Emergency
BP	≥180/≥120 mmHg	≥180/≥120 mmHg
Target Organ Damage	Absent	Present (CNS, CV, renal, retinal)
Examples	Asymptomatic severe HTN	Encephalopathy, stroke, MI, pulmonary edema, aortic dissection, eclampsia
Goal of Reduction	Gradual ↓ over 24–48 h (PO drugs)	Immediate (reduce MAP by ≤25% in 1st hr, then to 160/100 in 2–6 h)
Agents	Oral captopril, clonidine, labetalol	IV labetalol, nitroglycerin, nitroprusside, nicardipine, esmolol (avoid rapid drop)

Special Situations:

- Aortic dissection: Reduce SBP to 100–120 within 20 min (IV esmolol ± nitroprusside)
- Eclampsia: Labetalol / hydralazine + magnesium sulfate
- Pheochromocytoma crisis: Phentolamine (α-blocker first)
- Acute pulmonary edema: IV nitroglycerin / nitroprusside + loop diuretic

RESISTANT HYPERTENSION

Definition:BP ≥130/80 despite 3-drug regimen (including diuretic) or controlled BP with ≥4 drugs.

Causes:

- Pseudoresistance: White coat, poor adherence, wrong cuff, measurement error.
- Secondary HTN: CKD, OSA, primary aldosteronism, renal artery stenosis, pheochromocytoma.
- Drug-related: NSAIDs, OCPs, steroids, cyclosporine, erythropoietin, decongestants, licorice.

Work-up:

- Confirm adherence & technique → evaluate for secondary causes → check volume overload, drug interference, OSA.
- Investigations:

1)Serum creatinine, electrolytes
2)Plasma aldosterone/renin ratio
3)Urinalysis, renal Doppler
4)Sleep study
5)24-h urinary metanephrines

BP TARGETS:

Population	Target BP (mmHg)
General population <60 yrs	<130/80
Diabetes / CKD	<130/80
Elderly (≥65 yrs, fit)	SBP 130–139; avoid <120
Post-stroke	<130/80
CAD / heart failure	<130/80

HYPERTENSIVE EMERGENCIES DRUG CHOICES:

Drug	Preferred Situations	Avoid In
Labetalol	Stroke, encephalopathy, pregnancy	Heart failure
Esmolol	Aortic dissection	Heart failure
Nicardipine / Clevidipine	Most emergencies	HF (caution)
Nitroglycerin	MI, pulmonary edema	None significant
Nitroprusside	Refractory HTN	CKD/liver disease (cyanide toxicity)
Hydralazine	Eclampsia	CAD (tachycardia)
Phentolamine	Pheochromocytoma	—

PERICARDIAL DISEASES:

Condition	Key Findings / Mechanisms / Cut-offs
Acute Pericarditis	□ Causes → Viral (Coxsackie, Echo), Post-MI (Dressler), Uremic, TB, Malignancy, CTD □ ECG 4 Stages: 1 – Diffuse ST ↑ (concave) + PR ↓ → 2 – ST normalization → 3 – T inversion → 4 – Normal □ Diagnostic Triad: Pleuritic CP ↑ on lying flat ↓ on sitting forward + Pericardial rub + Typical ECG □ Treatment → NSAIDs + Colchicine (0.5 mg BID × 3 mo); avoid steroids unless recurrent / CTD
Cardiac Tamponade	□ Beck's Triad → ↓BP + ↑JVP + Muffled HS □ Pulsus paradoxus >10 mmHg (SBP ↓ in inspiration) □ Echo signs → RA collapse (systolic), RV collapse (diastolic), IVC plethoric non-collapsing □ Equalization of diastolic pressures (RA ≈ RV ≈ PCWP within 5 mmHg) □ Treatment → Urgent Pericardiocentesis (USG-guided subxiphoid)
Constrictive vs Restrictive Physiology	□ Constrictive Pericarditis: Thick calcified pericardium → diastolic plateau (square root sign) → Equal RV/LV EDP <5 mmHg diff □ Restrictive CM: Myocardial fibrosis → ↑ LV EDP > RV EDP > 5 mmHg; BNP ↑↑ □ Echo Distinctions: Annulus reversus (TDI e' lat < sept), Resp variation mitral E >25% → Constrictive
Pericardial Effusion	□ Causes → Malignancy, Uremia, Hypothyroid, Post-MI, Autoimmune, TB □ ECG → Low voltage QRS ± electrical alternans □ Echo → >10 mm separation in diastole = moderate; >20 mm = large □ Tx → Observe if small; Pericardiocentesis if symptomatic or tamponade
Dressler's Syndrome	□ Autoimmune post-MI pericarditis (weeks 2–6) → fever, pleuritic CP, ↑ESR, pericardial/pleural effusion □ Tx → NSAIDs ± Colchicine (Avoid steroids early post-MI)

PULMONARY HYPERTENSION & COR PULMONALE

Aspect	High-Yield Facts
Definition (RHC)	mPAP ≥ 20 mmHg (at rest) + PAWP ≤ 15 mmHg + PVR ≥ 3 WU
WHO Classification	I – PAH (idiopathic, heritable, CTD, HIV, drug) → II – LH disease → III – Lung/OSA → IV – CTEPH → V – Misc
CTEPH	Chronic PE → persistent obstruction □ Diagnostic test = V/Q scan (best screen) □ Tx = Pulmonary endarterectomy / Riociguat
PAH Management Algorithm	Step 1: Exclude secondary causes → Confirm RHC → Vasoreactivity test (↓ mPAP ≥10 to ≤40 = positive) → CCB trial if positive Step 2: If negative → Endothelin antagonist (Ambrisentan, Bosentan) ± PDE5 inh (Sildenafil, Tadalafil) ± Prostacyclin analog (Epoprostenol, Iloprost) Step 3: Advanced → Selexipag / Riociguat / Lung Tx
Cor Pulmonale	RV dilation due to chronic lung disease → JVP ↑, loud P_2, pedal edema → Tx = O_2 therapy + diuretics + treat lung disease

| Acute PE Diagnosis | Wells Score > 4 = likely → CTPA (+ve = treat) ; If unlikely → D-dimer → CTPA if +ve || PE Treatment | LMWH → Warfarin (INR 2-3) or DOAC 3-6 mo Massive PE + shock → tPA Contra to lysis → Catheter / Surgical embolectomy |

AORTA & PERIPHERAL ARTERY DISEASE

Condition	Essentials / Cut-offs / Treatment
Aortic Dissection	Stanford: A = ascending ± descending B = descending only DeBakey: I (asc + desc) II (asc only) III (desc only) Imaging: CTA best (TEE bedside) Tx: Type A → Surgery Type B → IV β-blocker (Esmolol) ± Nitroprusside → BP 100-120 SBP
Aortic Aneurysm (AAA)	RF: Smoking > Age > M > HTN > Atherosclerosis Repair if >5.5 cm (men) / >5.0 cm (women) or growth >0.5 cm/6 mo or symptomatic
Takayasu Arteritis	Young female + absent pulse ESR ↑ Angiogram: Skip lesions Tx = Steroids ± Tocilizumab
Peripheral Arterial Disease (PAD)	ABI = Ankle/Arm SBP: Normal 0.9-1.3 Mild 0.7-0.9 Mod 0.4-0.7 Severe <0.4 Fontaine: I asymptomatic II claudication III rest pain IV ulcer/gangrene Tx: Smoking ↓ Exercise Statin Antiplatelet Cilostazol Revascularize if lifestyle limiting
Acute Limb Ischemia	6 Ps → Pain, Pallor, Pulselessness, Paresthesia, Paralysis, Poikilothermia Tx = IV Heparin → Embolectomy / Thrombolysis + Fasciotomy if reperfusion
Carotid Artery Disease	>70% stenosis or symptomatic → Endarterectomy 50-69% → Case-based Antiplatelet + Statin BP <130/80 Smoking cessation

MISCELLANEOUS / NEW CONCEPTS

Topic	High-Yield Points
Cardiac Amyloidosis	□ Echo = “Sparkling” myocardium + ↑ wall thickness + small LV cavity + restrictive pattern □ MRI = Global subendocardial LGE □ Low QRS voltage vs ↑LV mass = diagnostic clue
Myocarditis (Lake Louise MRI)	≥2 of 3 → T2 ↑ (edema) + T1 ↑ (non-ischemic LGE) + T2 mapping ↑ □ Causes = Viral > Autoimmune > Drugs
Cardiac Tumors	Myxoma – LA > RA Ball-valve obstruction + “tumor plop” Embolic + constitutional symptoms Tx = Surgery
COVID Myocarditis / MIS-C	↑ Troponin, ↑ BNP LV dysfunction Shock Tx = IVIG + Steroids + Supportive
Inherited Channelopathies	□ Brugada → V1-V3 coved ST ↑ → ICD □ Long QT → β-blockers QTc >470 F >450 M Avoid QT-prolonging drugs □ CPVT → Bidirectional VT → β-blockers + Flecainide ICD if refractory
Cardio-Oncology	Anthracyclines → dose-dependent HF >400 mg/m^2 = risk Prevent → Dexrazoxane Monitor EF by Echo/MRI
Cardiac MRI / CT Applications	MRI → Tissue characterization (myocarditis, amyloid, scar) CT → Coronary calcification / TAVR planning / Aortic disease
Biomarkers	HF: BNP, NT-proBNP ACS: High-sensitivity Troponin (T/I) – rise ≥5–10 ng/L = acute ST2, Galectin-3 – fibrosis markers (prognostic in HF)
New Antiplatelet / Anticoagulant Updates	□ Antiplatelets → Ticagrelor > Clopidogrel Prasugrel CI in CVA/TIA □ Anticoagulants → DOACs (first-line non-valvular AF) Reversal → Idarucizumab (dabigatran) / Andexanet alfa (FXa inh)

Cardiorenal Syndrome (CRS)

Type	Mechanism / Key Associations	Examples / Management Focus
Type 1 – Acute Cardiorenal	↓ CO → renal hypoperfusion + venous congestion → ↑ creatinine within 48 h	ADHF → optimize preload & afterload + avoid nephrotoxins
Type 2 – Chronic Cardiorenal	Chronic HF → RAAS ↑ → progressive CKD	RAAS blockade (ACEi/ARB/ARNI + MRA + SGLT2i)
Type 3 – Acute Renocardiac	AKI → volume overload → LV dysfunction	Diuretics / UF / dialysis
Type 4 – Chronic Renocardiac	CKD → LVH + fibrosis via anemia, Ca-P load	Treat anemia (ESA + Fe) + BP < 130/80
Type 5 – Secondary/Systemic	Sepsis, SLE, Amyloidosis → both organ injury	Treat underlying systemic cause
Clue: 20–30 % of ADHF → AKI; best marker = ↑ Cystatin C.		

Endocrine Hypertension

Disorder	Mechanism / Diagnostic Keys	Key Cut-offs & Management
Primary Aldosteronism	↑ Aldo / ↓ Renin → Na^+ retention, K^+ loss	Aldo:Renin > 20 + Aldo > 15 ng/dL → confirm (saline suppression) → Adrenal CT → Unilateral = surgery ; Bilateral = MRA (Spironolactone /eplerenone)
Cushing's Syndrome	Cortisol excess → ↑ BP (via mineralocorticoid action)	Dx = ↑ 24 h urine cortisol / ↑ midnight cortisol / no suppression with 1 mg DST → Tx cause (adrenal/pituitary)
Pheochromocytoma	Catecholamine surge → episodic HTN + H/A + sweating + palpitation	Plasma metanephrines ↑ > 3×ULN → CT/MRI abdomen ± MIBG → Pre-op α-block (phenoxybenzamine) then β-block → Surgery
Acromegaly / Thyroid	GH ↑ or T_3↑ → SV ↑ / TPR ↓ → systolic HTN	Control primary endocrinopathy

Thyrotoxic Heart Disease

Mechanism	Findings / Numbers	Management
↑ T_3 → ↑ β-receptor sensitivity → ↑ HR, CO → high-output HF	Wide PP, sinus tachy or AF (10–15 %), TR murmur	β-blocker (propranolol) + antithyroid (PTU/Methimazole) + treat AF (rate control + OAC if CHA_2DS_2-VASc ≥ 2)
Thyrotoxic cardiomyopathy	Chronic T_3 excess → dilated LV, low EF (5–10 %)	Reversible with euthyroid state (~3 mo)

Rheumatic Heart Disease (RHD) & Secondary Prevention

Aspect	High-Yield Points
Pathogenesis	Post-Strep A pharyngitis → cross-reactive M-protein → Aschoff body (pancarditis)
Jones Criteria (2023 mod)	Major – Carditis / Arthritis / Chorea / Erythema marginatum / Subcutaneous nodules Minor – Fever / ↑ ESR or CRP / Prolonged PR
Secondary Prophylaxis	Benzathine Pen G 1.2 M U IM q3 wk (alt q4 wk if low risk) Duration → • No carditis = 5 y or age 21 • Carditis no residual = 10 y or age 40 • Carditis + valve = lifelong / ≥10 y post last attack
Surgery indications	Severe MS / MR / AR per echo criteria ; Balloon valvotomy if pliable valve + no thrombus + no MR > mild

Cardiac Manifestations of Systemic Diseases

Systemic Disease	Cardiac Involvement	Diagnostic Clues / Imaging
SLE	Libman–Sacks (sterile verrucous MV/AV vegetations), Pericarditis (MC), Myocarditis rare	Echo shows valvular nodules; aPL ↑ → thrombosis
Sarcoidosis	Non-caseating granulomas → AV block, VT, HF (patchy fibrosis)	Cardiac MRI – mid-myocardial LGE (basal septum); FDG-PET ↑ uptake; Tx = Steroids + ICD if VT
Amyloidosis	AL / ATTR → restrictive CM + low voltage ECG + "sparkling LV"	MRI – global subendocardial LGE ; Bone-scan grade ≥ 2 = ATTR; Tx = Tafamidis (ATTR) / Chemo (AL)
Scleroderma	Fibrosis → restrictive CM + PAH	Echo PA press ↑ ; avoid βB ; use PDE5i / ERAs
Hemochromatosis	Iron → dilated / restrictive CM + arrhythmia	T2* MRI ↓ (<20 ms) ; Tx = Phlebotomy / Deferasirox

Trials in cardiology:

Trial	Intervention / Comparator	Primary Finding	
1. EMPEROR-Preserved	Empagliflozin vs placebo	↓ Composite CV death/HF hospitalization → First SGLT2i proven in HFpEF	
2. DELIVER	Dapagliflozin vs placebo	↓ CV death/HF hosp similar to EMPEROR → Class effect confirmed	
3. DAPA-HF	Dapagliflozin vs placebo	↓ CV death/HF hosp even without diabetes → SGLT2i = Core HFrEF drug	
4. EMPULSE	Empagliflozin started early in hospital	Improved clinical benefit composite → Safe to start SGLT2i during admission	
5. PARADIGM-HF	Sacubitril/valsartan vs enalapril	↓ CV death/HF hosp 20% → ARNI replaces ACEi in HFrEF	
6. VICTORIA	Vericiguat vs placebo	↓ CV death/HF hosp modestly → Use post-ADHF if persistent symptoms	
7. GALACTIC-HF	Omecamtiv mecarbil vs placebo	↓ HF events, not mortality → Myosin activator – new class	
8. EMPACT-MI / DAPA-MI	SGLT2i early post-MI	↓ HF hospitalization trend → Expanding SGLT2i role beyond HF/DM	
9. REVIVED-BCIS2	2022 – Ischemic HFrEF (EF ≤35%)	PCI + OMT vs OMT alone	No survival benefit → Revascularization doesn't improve mortality in stable ischemic HFrEF
10. ISCHEMIA Trial	2020 – Stable CAD with mod-severe ischemia	Early invasive vs OMT	No mortality benefit; OMT equally effective → Guideline-changer for stable CAD

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