CARDIOLOGY

APPROVED
By Zurab Azmaiparashvili at 2:38 am, Jan 24, 2009

Ischemic Heart Disease/Coronary Artery Disease (IHD/CAD)

- **Atherosclerosis** is the **major cause** of CAD
- **1/3** of **all deaths** secondary to CAD

Risk Factors for CAD

Major Modifiable	**Major Non-modifiable**	**Minor**
Smoking	Age	Obesity
Diabetes Mellitus	Gender	Sedentary Lifestyle
Hypercholesterolemia/ Hyperlipidemia	Family History of Premature CAD	Hyperhomocysteinemia
Hypertension		Stress/depression
		OCP use
		Hypertriglyceridemia
		"Type A" Personality
		↑ Lipoprotein (a)
		↑ C-reactive Protein
		Heavy Alcohol Intake

- **Cigarette Smoking →**
  1. **2x incidence** of CAD compared to **non-smokers**
  2. **Cessation** of smoking → **risk decreases** to **control** values within **2-3 years**
- **Gender →**
  1. **Male**/Female ratio **2:1** (all age groups)
  2. **M/F** ratio **1:1 →** age **> 70**
  3. **Males** develop **CAD** at an **earlier age** than females
- **Age →** > **45** in **males** and/or > **55** in **females**
- **Premature CAD → age < 55** in **males** and/or **age < 65** in **females**
- **OCP use →** women **> 35** years of age who **smoke**
- **↑ C-reactive Protein → 2-3x incidence** of **MI** in the absence of other risk factors
- **Obesity →** contributing mechanisms include:
  1. **Insulin resistance**/Type 2 DM
  2. **↑ total** and/or **LDL cholesterol**
  3. **↓ HDL** cholesterol
  4. **↑ BP**
- **DM →** considered a **"CAD-equivalent"** (*see below*)

*** Protective** factors **→ ↑ estrogen** (pre-menopause), **moderate alcohol** consumption, **weight loss**, **exercise, HDL** cholesterol > **60 mg/dL** (cancels one risk factor)

Hypercholesterolemia

- **Fasting total** cholesterol **> 200 mg/dL**, **LDL** cholesterol **> 130 mg/dL** and/or **HDL** cholesterol < **35 mg/dL**
- May be **hereditary** (e.g. Type II hyperlipidemia) or **acquired** (more common)
- Acquired causes of hypercholesterolemia include:
    1. **Diet** rich in **fat** and/or **cholesterol**
    2. **Smoking**
    3. Excessive **alcohol** intake
    4. **Obesity**
    5. **DM**
    6. Nephrotic syndrome
    7. Liver disease
    8. **Hypothyroidism**
    9. **Drugs** (e.g. **OCPs**, thiazide/loop **diuretics**)
- Clinical features → **asymptomatic** to **xanthelasmas**, tendon **xanthomas** and/or lipemia retinalis (**more common** in **hereditary** forms of disease)
- **Screening** for hyperlipidemia → age > **35** in **men** and age > **45** in **women** (if normal, repeat in ~ 5 years)
- Management →

Patient Category	Indication for lifestyle modification	Indication for medical therapy	Target LDL
0-1 CAD **risk factor**	**LDL > 160 mg/dL**	**LDL > 190 mg/dL**	**< 160 mg/dL**
≥ 2 CAD **risk factors**	**LDL > 130 mg/dL**	**LDL > 160 mg/dL**	**< 130 mg/dL**
CAD or **CAD equivalents**	**LDL > 100 mg/dL**	**LDL > 130 mg/dL**	**< 100 mg/dL**

* **CAD equivalents → DM**, **Peripheral** artery, **Carotid** and/or **Aortic** disease

* **Target LDL < 70 mg/dL →**
    1. **CAD + DM**
    2. **CAD** + cigarette **smoking**
    3. **CAD + metabolic syndrome** (syndrome X)
    4. **ACS** or acute coronary syndrome (UA, NSTEMI and/or STEMI)

* **Lifestyle modification →**
    1. **High-fiber diet** low in **fat** and **cholesterol**
    2. **Exercise**
    3. **Weight loss**
    4. Smoking cessation, ↓ alcohol intake

- **No**/minimal **response** to **3-month** lifestyle modification → **lipid-lowering** agents
- **Drug of choice** for ↑ **LDL** cholesterol → **statins** or HMG-CoA reductase inhibitors (e.g. simvastatin)
- **Drug of choice** for ↑ **triglycerides** → **fibrates** (e.g. gemfibrozil)
- **Drug of choice** for ↓ **HDL** cholesterol → **niacin**
- **Failure** to respond to **statins** → **addition** of **second-line** agents

* Clues to hereditary hyperlipidemia → **young** age, ↑↑ **lipid levels**, ± family history of premature CAD

Anti-hyperlipidemic Medications			
Drug Category	*Mechanism of Action*	*Net Effect*	*Side Effects*
HMG-CoA Reductase Inhibitors (e.g. simvastatin, lovastatin, etc.)	**Inhibition** of **rate-limiting** step in **cholesterol biosynthesis**	↓↓ **LDL**, ↑ HDL ↓ triglycerides,	GI distress, ↑ **LFTs**, myalgias, **myositis** with possible **rhabdomyolysis**/acute renal failure
Niacin/nicotinic acid	↓ **hepatic VLDL** synthesis ↑ **lipoprotein lipase** activity ↑ **t-PA** activity	↓ **LDL**, ↑↑ **HDL** ↓ triglycerides ↓ fibrinogen	**Prostaglandin-**mediated **flushing**/**pruritus** (self-limited; prevent with **aspirin**), **hyperuricemia**/gout, **hyperglycemia**, ↑ LFTs, **PUD**
Fibrates (e.g. gemfibrozil, fenofibrate, etc.)	↑↑ **lipoprotein lipase** activity	↓ LDL, ↑ HDL ↓↓ **triglycerides**	GI distress, cholesterol **gallstones**, myalgias/**myositis**, hypokalemia, ↑ **LFTs**
Bile-acid binding resins (e.g. cholestyramine, colestipol, colesevelam)	↓ **bile salt** absorption → ↑ **de novo** bile salt synthesis → ↓ liver cholesterol → ↑ hepatic **LDL uptake**	↓ **LDL** ↑ **triglycerides**	**GI distress**, ↓ **absorption** of concomitantly administered drugs (e.g. digoxin, warfarin, tetracycline)
Cholesterol absorption inhibitors (e.g. ezetimibe)	↓ GI **cholesterol absorption**	↓ **LDL**	**GI distress**

* **Most commonly** used **second-line** medication for ↑ **LDL** (in conjugation with statins) → **ezetimibe**

* Try to **avoid** combining **statins** with **fibrates** → ↑↑ risk of **rhabdomyolysis** (watch for extremely elevated CK, oliguria/anuria, dark-urine**,** etc.)

* **Do not** use **bile-acid sequestrants** in case of **hypertriglyceridemia**

* Note → patients with **hypertriglyceridemia** present with **eruptive xanthomas** and/or attacks of **acute pancreatitis** with **no**/minimal risk of **CAD** (e.g. Type I and V hyperlipidemias)

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By Zurab Azmaiparashvili at 2:39 am, Jan 24, 2009

Angina	Chest **"tightness"**, **"pressure**", discomfort **Substernal** location Radiation to the **neck, jaw**, **left arm** **Exertional** Relieved by **rest** and/or **nitroglycerin** Duration **5-15 minutes**
Myocardial Infarction	Similar to angina, but: 1. **more severe** 2. **longer duration** (>20-30 minutes) 3. **not relieved** by rest/nitroglycerin 4. accompanied by **nausea, vomiting, diaphoresis** and/or **shortness of breath**
Aortic Stenosis	Similar to angina, but **accompanied** by **characteristic murmur** of aortic stenosis
Aortic Dissection	**Sudden** onset **Sharp "tearing"** pain Radiation to the **back** **Unequal** arm **BP/pulses** Possible murmur of **aortic regurgitation**
Acute Pericarditis	**Sharp** "**pleuritic**" pain (worse with inspiration) Pain **worse** when **lying down** and/or swallowing Pain **improves** with **leaning forward** Possible pericardial **friction rub**
Pulmonary Embolism	**Sudden** onset **"Pleuritic**" chest pain Accompanied by **tachycardia, tachypnea**/dyspnea, **cough**/hemoptysis
Pulmonary Hypertension	Similar to angina, but **accompanied** by **loud S2** and/or symptoms/signs of **right ventricular failure**
Pneumonia	**Sharp, "pleuritic"** chest pain **accompanied** by **cough**, ↑ **fever**, dyspnea and/or **signs** of **consolidation**
Pneumothorax	**Sudden** onset **Sharp,** "**pleuritc"** chest pain and/or **dyspnea** accompanied by **hyperresonance** to percussion
Costochondritis	**Sharp** chest pain **worse** with **movement** Reproduced with **palpation**
GERD	**"Burning"** sensation **Worse** when **lying down, leaning forward** and/or wearing **tight clothing** **Worsens** with **nitroglycerin** **Improves** with **antacids**
Diffuse Esophageal Spasm	Similar to angina, but with **sudden** onset; usually brought on by drinking **cold beverages**

* Routine diagnostic tests to order in patients with chest pain and/or shortness of breath → **EKG** and **Chest X-ray**

Chronic Stable Angina

- > **60%** atherosclerotic **narrowing** of the **coronary arteries** → **adequate resting** perfusion + **inability** to **increase blood flow** with ↑ **demand**
- > **90-95% narrowing** → **resting ischemia**
- Definition → **long-lasting** chest pain of the **same frequency** and **intensity**, **reproducible** by the **same** amount of **exertion**
- Clinical features →
    1. Chest pain **precipitated** by **physical** and/or **emotional stress**
    2. Pain described as **"tightness", "pressure"** and/or **"squeezing"** sensation
    3. **Substernal** location (may be precordial, epigastric, etc.)
    4. **Radiation** to the **neck, lower jaw, left shoulder/arm** (right-sided radiation also a possibility)
    5. Possible **nausea, vomiting**, **dyspnea** and/or **diaphoresis** (more common with MI)
    6. **Brief duration** (<15-20 minutes)
    7. **Relieved** by **rest** and/or **nitroglycerin**
    8. New-onset **S4** heart sound (diastolic dysfunction) and/or **holosystolic murmur** of **mitral regurgitation** (papillary muscle dysfunction)
- Common **precipitants** of **angina pectoris** →
    1. Physical **exertion**
    2. **Emotional** upset
    3. Walking in **cold weather**
    4. Heavy **meal**
    5. **Early morning** hours
- **EKG** during the **episode** of **pain** → **ST-segment depression** and/or **T wave inversion** (indicating **subendocardial ischemia**)

* **Isolated dyspnea** and/or **fatigue** may be a manifestation of **ischemia**, especially in **women, elderly** and/or patients with **DM**
* **Silent ischemia** → pretty **common** in patients with known **stable angina** (~ 5:1 ratio); also common in the **elderly**, patients with **DM** and/or **post-MI**; **asymptomatic** + **ST** segment **depression** on **EKG**

- **Best initial** diagnostic test → **resting** (baseline) **EKG** to **rule out** ongoing **ischemia** and/or presence of **EKG changes** that may **preclude** performing a **standard exercise stress testing**

* Not for Test Qs → all patients should undergo a **2-D echocardiography** to assess **left ventricular function**, which is the **most important prognostic** indicator

- **Low probability** of **stable angina** (e.g. young, healthy female with atypical chest pain) → **look for another etiology** of chest pain; **do not** order **stress testing** (↑ **false-positive** results)
- **High probability** of **stable angina** (e.g. older male, with multiple CAD risk factors presenting with typical chest pain) → determine disease **severity**/**need for revascularization** and/or proceed to **medical treatment**; **do not** order **stress testing** for **diagnostic purposes** (↑ **false-negative** results)

- **Intermediate probability** of **stable angina → proceed** to **stress testing**

* You may still order **stress testing** in patients with **high-probability angina** for **risk-stratification** but not diagnostic **purposes**

- **Indications** for **stress testing**:
    1. Evaluation of **chest pain**
    2. Assessment of **treatment efficacy**
    3. **Risk stratification**/need for revascularization
    4. Assessment of **myocardial viability**
- **Contraindications** to **stress testing**:
    1. **Acute MI** (first 2 days)
    2. **Unstable** angina
    3. Uncontrolled **hypertension**
    4. Uncontrolled **tachy-** and/or **bradyarrhythmias**
    5. Aortic **dissection**
    6. Severe **aortic stenosis**
    7. Uncontrolled **heart failure**
    8. **Hypertrophic** cardiomyopathy
    9. Acute **myocarditis, pericarditis** and/or **endocarditis**

- **Positive** findings →
    1. **ST-**segment **depression** (horizontal or down-sloping) ≥ **1 mm**
    2. Significant **hypotension**
    3. **Chest pain**
- Patient **able** to **exercise** + **normal** baseline **EKG → regular exercise** stress testing (e.g. treadmill, bicycle)
- Patient **able** to **exercise** + **uninterpretable EKG →** exercise stress testing with **thalium imaging** (nuclear stress testing)
- Patient **able** to **exercise** + **uninterpretable EKG** + **obesity** and/or **female** gender → exercise stress testing with **Tc-99 imaging** (**sestamibi** scan)

* **Uninterpretable EKG → LBBB**, left ventricular **hypertrophy**, baseline **ST depression** > 1 mm, **pacemaker**, **digoxin** therapy, **Wolff-Parkinson-White** syndrome

- Patient **unable** to **exercise → pharmacologic** stress testing:
    1. **Persantine** (dipyridamole) thallium (most common test Q answer)
    2. Adenosine thallium
    3. **Dobutamine** with **echo** (patients with **asthma**, **COPD**)

* **Nuclear** imaging **→ reversible "cold spots"** indicate areas of **ischemia**/↓ perfusion; **persistent** changes – **previous MI**

* **ECHO → decreased**/abnormal **wall motion** indicates areas of **ischemia**/↓ perfusion

- **High-risk** findings during **exercise stress** testing:
    1. **ST**-segment **depression** > **2 mm**
    2. **ST**-segment **depression** > **6 min.** in duration
    3. Any degree of **ST**-segment **elevation**
    4. Significant **hypotension**
    5. Significantly **decreased LV contractility**

- **Most accurate** diagnostic test → **coronary angiography**

* **Indications** for coronary **angiography**:
    1. **High-risk** patient (*see above*)
    2. **Contraindications** to **stress testing**
    3. Stable angina **refractory** to **medical therapy**
    4. **STEMI**
    5. High-risk **UA/NSTEMI**
    6. **Non-diagnostic** stress testing
    7. **Post-infarction** chest pain

- Management:
    1. **Short-**acting **nitrates** (sublingual nitroglycerin) for **acute relief** of chest pain
    2. **β-blockers** → **first-line** agents for **chronic** management of **stable angina**
    3. **No**/minimal **response** to β-blockers → **add long**-acting **nitrates** (e.g. isosorbide dinitrate)
    4. **Avoid short-**acting **Ca-channel** blockers → ↑ **mortality**
    5. Daily **aspirin** ± statins (*see above*)
    6. **Correction** of CAD **risk factors**
    7. **No**/minimal **response** to **medical** therapy and/or **high-risk** patient → consider coronary **revascularization** (PCI vs. CABG)

* **Indications** for **CABG** (coronary artery bypass grafting) →
    1. **Left main coronary** artery disease
    2. **Three-vessel** disease
    3. **Proximal left anterior descending** artery involvement
    4. **Diffuse** disease (unable to perform PTCA)
    5. CAD + **DM**
    6. CAD + ↓ **LV function**

* **PCI** (percutaneous coronary intervention) → **PTCA** (percutaneous transluminal coronary angioplasty) ± **stent** placement →
    1. **Shorter** hospital stay
    2. **Lower cost**
    3. ↑ **need** for **repeat** revascularization
    4. Complications → intimal **dissection** (requiring urgent CABG), **restenosis** (major complication; 30-40% at 6 months; lower incidence with stent placement)

* Patients undergoing **PTCA + stent** placement should take **aspirin** (indefinitely), **clopidogrel** (at least 1 month) and **GP IIb/IIIa inhibitors** (before procedure) → *see below*

Prinzmetal's Angina (Variant Angina; Coronary Artery Spasm)

- **Females** > Males
- **Young** age (~ 20-40 years of age)
- Risk factors → cigarette **smoking**, **cocaine** abuse
- Associated conditions → **migraine** headaches, **Raynaud's** phenomenon
- Clinical features → **nocturnal** and/or **early morning** chest **pain** (usually awakening the patient from sleep) + **ST**-segment **elevation** on **EKG** (as opposed to MI, EKG returns to normal in patients with variant angina)
- Stress testing and/or coronary angiography → **normal** (although coronary spasm most commonly occurs at sites of atherosclerosis)
- **Most accurate** diagnostic test → **angiographic** demonstration of coronary **spasm** induced by **intracoronary injection** of **ergonovine** (ergonovine challenge test) and/or **acetylcholine**
- Management → **Ca-channel blockers** (**drugs of choice**) ± long-acting **nitrates**

Acute Coronary Syndrome (ACS)

- **ACS** consists of **UA** (unstable angina), **NSTEMI** (non-ST elevation myocardial infarction) and **STEMI** (ST elevation myocardial infarction)
- Most cases of **STEMI** progress to **QwMI** (Q-wave myocardial infarction)
- Most cases of **NSTEMI** progress to **NQMI** (non-Q wave myocardial infarction)
- Pathophysiology: atherosclerotic **plaque disruption** (rupture, fissuring, etc.) with **superimposed thrombosis** resulting in **complete** (STEMI) or **non-complete**/flow-limiting (UA/NSTEMI) coronary artery **occlusion**

UA/NSTEMI

- Definition of **unstable angina** →
    1. **new-onset** angina
    2. angina occurring at **rest**
    3. **"crescendo**" angina (↑ frequency, duration and/or severity)
    4. **post-MI** angina
    5. **post-CABG** angina
    6. **post-angiography/PCI** angina
- **NSTEMI** is similar to **UA** with regard to **pathophysiology** and/or **clinical features**
- **Differentiation** between UA and NSTEMI → order **cardiac troponins** (Troponin T or Troponin I) and/or **CK-MB** determination, which should be **normal** with **UA** and **elevated** with **NSTEMI**
- **Best initial** diagnostic test in **suspected** UA/NSTEMI → **EKG** showing **normal** tracing or **dynamic** changes (e.g. **ST** segment **depression** and/or **T wave inversion**)
- **Best next** step (even if EKG is normal) → **serial** measurements of **cardiac enzymes** (on admission + every 8 hours)

- All patients with **UA/NSTEMI** should receive →
  1. **Aspirin** ± clopidogrel
  2. **β-blockers**
  3. **Nitroglycerin**
  4. Supplemental **oxygen**
  5. **Morphine** (if pain non-responsive to nitroglycerin)
  6. **Heparin**
  7. **Statins**
- **High-risk** patients should also be started on **GP IIb/IIIa inhibitors** (e.g. abciximab, tirofiban, eptifibitide)

* **High-Risk UA/NSTEMI →**

1. **> 20 min.** ongoing **rest pain**
2. **recurrent** ischemic **chest pain**
3. **↓ BP**
4. **↑** cardiac **enzymes**
5. **pulmonary edema**
6. **↓ LV function** (EF < 40%)
7. **Dynamic EKG** changes
8. presence of **DM** and/or **kidney failure**
9. Severe **arrhythmia** (e.g. ventricular tachycardia)
10. Age > **75**

- **High-risk** patients should undergo **early** (within **48 hours**) **coronary angiography** with possible **revascularization** (PTCA or CABG)
- **Stabilized, low-**/intermediate-**risk** patients should undergo **pre-discharge** evaluation of **LV function** and **stress testing**:
  1. **EF < 40%** and/or **high-risk** findings on **stress testing →** proceed to **coronary angiography** with possible **revascularization**

* **DO NOT** use **thrombolytic** agents in patients with **UA/NSTEMI → ↑ mortality**

* **DO NOT FORGET** to give **heparin** to patients with **UA/NSTEMI** (unless contraindicated) → continuous intravenous **UFH** or subcutaneous **LMWH**

* **Contraindications** to **β-blockers →** substitute with **verapamil** or **diltiazem** (non-dihydropyridine Ca-channel blockers)

* Add **ACE-inhibitors** in patients with **↓ LV function**

- Chronic management → **similar** to chronic **stable angina**
- **50%** of cases of **UA** progress to **MI without** treatment

STEMI

- History → **severe, prolonged** (>20 min) **retrosternal** chest **pain** with **radiation** to the **neck,** lower **jaw**, left **shoulder/arm** + **nausea**/vomiting, **dyspnea** and/or **diaphoresis**; extreme fear/**apprehension** common
- Physical Exam → **tachycardia** (possible bradycardia with inferior wall/right ventricular infarctions), **hypertension**/hypotension, new-onset **S4 heart sound** and/or **murmur** of **mitral regurgitation**
- **Best initial** diagnostic test → **EKG** showing ≥ **1 mm ST** segment **elevation** in ≥ **2 contiguous** leads with **ST** segment **depression** in **reciprocal** leads and/or **new-onset LBBB**
- **Confirmation** of diagnosis → ↑ cardiac **troponins** and/or **CK-MB** detected during **serial determinations** of cardiac enzymes (may be normal at presentation)
- **Evolution** of **EKG changes**:
    1. **hyperacute T** waves
    2. **ST** segment **elevation**
    3. **development** of **Q** waves/**T** wave **inversion**
    4. **normalization** of **ST** segment
    5. **normalization** of **T** waves

* Cardiac Enzymes:
1. **CK-MB → peak** at **12-24** hours **post-MI**; **detectable** for ~ **3 days**
2. **Troponins → detectable** for ~ **7-10 days**; **most specific** MI marker
3. **Myoglobin → appears first** (~ 30 min post-MI); **negative** result **does not rule out** MI (lacks specificity)

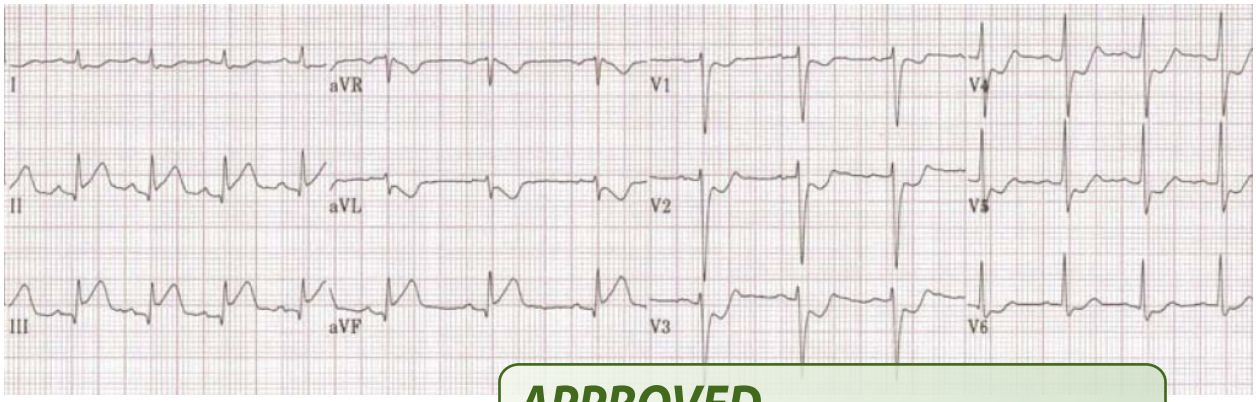
* **Suspected Re-infarction** → order **CK-MB**

* **Suspected recent** infarction → order cardiac **troponins**

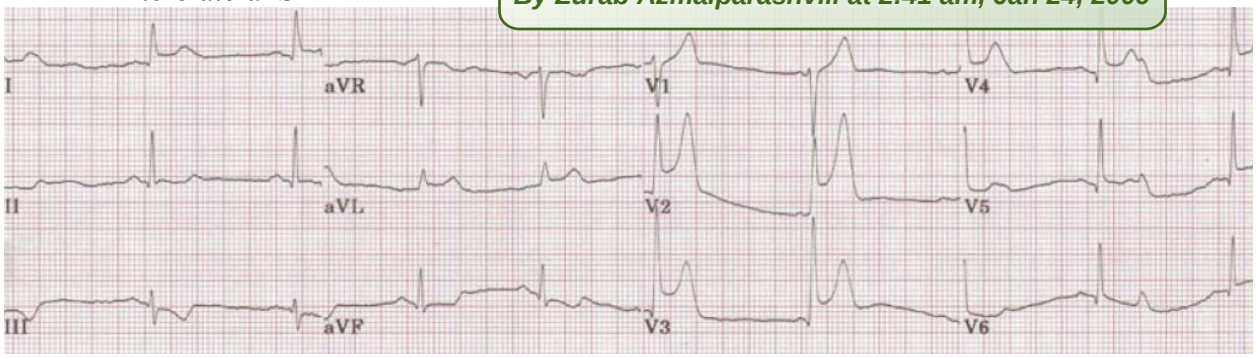
- **Localization** of **STEMI**:
    1. **Inferior** wall → **II, III** and **aVF**
    2. **Lateral** wall → **I, aVL** and **V_5 – V_6**
    3. **Anterior** wall → **V_3 – V_4**
    4. **Septal** → **V_1 – V_2**
    5. **Posterior** wall → **reciprocal** changes in **V_1 – V_2** (e.g. **ST** segment **depression, peaked T** waves)
- **Vessels affected** depending on MI location:
    1. **Right coronary** artery → **right ventricle, inferior** wall, **posterior** wall, **SA** and/or **AV nodes**
    2. **Left anterior descending** artery → **anterior** wall, **septal** wall
    3. **Left circumflex artery** → **lateral** wall (possible inferior/posterior walls)

* Remember → **most** individuals have **right coronary dominance** (artery that gives rise to posterior descending artery supplying the posterior/inferior walls of LV)

- Inferior wall STEMI



- Anterolateral STEMI



- Patients with **STEMI** should be started on:
    1. Supplemental **oxygen**
    2. **Aspirin**
    3. **Nitroglycerin**
    4. **β –blockers**
    5. **Morphine**
    6. **Heparin**
    7. **Statins**
- Consider **ACE inhibitors** (in the absence on contraindications and/or hypotension), especially if **EF < 40%**, pulmonary **edema** and/or **anterior MI**
- **Add clopidogrel** if the patients is to undergo **reperfusion** therapy (fibrinolysis vs. PCI)
- **Add** GP **IIb/IIIa inhibitors** before undergoing **PCI**

* **DO NOT** give **clopidogrel** if **CABG** is **anticipated**

* **Avoid** using **Ca-channel blockers**, especially short-acting **nifedipine**

- < **12 hours** since **onset** of **chest pain** → consider **reperfusion therapy**
- **PCI** better than **fibrinolysis** if performed within **90 minutes** of patient encounter
- Anticipated **delay > 90 min** → proceed to **fibrinolysis** (unless contraindicated)
- **Best results** with **fibrinolysis** if performed within **30 minutes** of patient encounter

- **Indications** for **thrombolysis** (fibrinolysis) →
  1. **Chest pain** > **30 min** in duration *plus*
  2. **< 12 hours** since onset *plus*
  3. **> 1 mm ST** segment **elevation** and/or **new-onset LBBB**
- **Indication** for **PCI** →
  1. **as above** *plus* < **90 min "**door-to-balloon" time *or*
  2. **contraindications** to **fibrinolysis**

* **Rescue PCI → continued ischemia** after thrombolytic therapy (or **cardiogenic shock, pulmonary edema** and/or **EF < 40%** post-fibrinolysis)

* **No response** to **PCI** → proceed to **CABG** (also consider CABG in cases of mechanical complications of MI requiring surgical intervention)

Contraindications to Thrombolysis	
Absolute *Contraindications*	***Relative*** *Contraindications*
Active bleeding, excluding menses	**Uncontrolled hypertension** (SBP > 180 mmHg and/or DBP > 110 mmHg)
Bleeding diathesis	Ischemic **stroke > 3months**
Suspected **aortic dissection**	**Traumatic/prolonged** (>10 min) **CPR**
Known **cerebral neoplasm**	Major **surgery within 3 weeks**
Known cerebral **vascular lesion**	Internal **bleeding within 2-4 weeks**
Previous **intracranial hemorrhage**	**Non-compressible** vascular **punctures**
Ischemic **stroke within 3 months**, excluding acute ischemic stroke < 3 hours duration	**Active PUD**
Significant **head/facial trauma within 3 months**	**Pregnancy**
Proliferative retinopathy	**Prior exposure**/allergic reaction (for **streptokinase**/anistreplase only)

- **Chronic Management** (drugs patients should be taking at discharge and thereafter) →
  1. **Aspirin**
  2. **β-blockers**
  3. **Statins**
  4. **ACE-inhibitors**, in patients with **anterior MI** and/or **EF < 40%**
  5. Short-acting **nitrates** for **acute relief**
  6. **Clopidogrel** (at least **1 month** post-**fibrinolysis**; ~ **12 months** post-**PCI**)
  7. **Warfarin** for **large, anterior MI** ± **intramural thrombus** (3-6 months)

* Everyone needs **modification** of **risk factors** (e.g. control of hypertension, smoking cessation, regular exercise, etc.)

* **Drug** that have shown to ↓ **mortality** include → **aspirin, β-blockers, ACE-inhibitors, statins** and **warfarin**

* **EF < 30%** 1 month post-MI → consider **implantable cardioverter-defibrillator**

* **Diltiazem** has shown to ↓ **mortality** in patients with **NQMI**

- Perform **sub-maximal** exercise and/or nuclear **stress testing 4-6 days** post-MI
- Perform **standard stress testing 3-6 weeks** post-MI
- Stress testing **positive for ischemia** → proceed to **angiography**

Selected Complications of MI

Complication	*Key Features*	*Management*
Right Ventricular Infarction	↑ **JVP**, **hypotension**, **absence** of pulmonary edema Associated with **inferior**/posterior **MI** **ST** segment **elevation** in **V_4R** Echo: **RV hypokinesis** Heart Catheterization:↑ **right**-sided **pressures** + ↓ **PCWP**	Step 1 → **IV fluids** + **stop nitroglycerin** infusion Step 2 → **dobutamine**, if above measures fail
Ventricular **Free Wall Rupture**	**Females** > Males **3-5 days** post-MI Symptoms/signs of **pericardial tamponade** (*see below*) **Electromechanical dissociation** ± sudden death Echo: large pericardial **effusion** Heart Catheterization: **equalization** of **diastolic** pressures	**Pericardiocentesis** (if tamponade) + **emergent surgical** repair
Papillary Muscle **Rupture**	**3-5 days** post-MI Associated with **inferior MI** **Sudden** onset of **hypotension** and **pulmonary edema** **Pansystolic murmur** of mitral regurgitation Echo: severe **mitral insufficiency** Heart Catheterization: **prominent V wave**	**Emergent surgical** repair (consider vasodilators and/or **IABP** as temporary measures before surgery)
Ventricular **Septal Rupture**	**3-5 days** post-MI **Sudden** onset of **hypotension** and **pulmonary edema** Harsh**, pansystolic murmur** + **palpable thrill** Echo: **septal** defect Heart Catheterization: **Step-up** in O_2 **saturation** at **RV** (from RA to PA)	**Emergent surgical** repair (consider vasodilators and/or **IABP** as temporary measures before surgery)
Acute **Pericarditis**	**"Pleuritic"** chest **pain** (radiating to the **trapezius**) ± **friction rub** EKG: **diffuse ST** segment **elevation** ± **PR** segment **depression**	**Aspirin** (or other NSAIDs) ± steroids Consider **stopping anticoagulation** (may progress to hemorrhagic pericarditis)
Dressler's Syndrome	Possibly **autoimmune** Several **weeks post-MI** Symptoms/signs of **pericarditis** + ↑ **ESR**	**Aspirin** (or other NSAIDs) ± **steroids**

Ventricular **True Aneurysm**	Associated with **anterior MI** Usually **apical location** **Several weeks** post-MI Symptoms/signs of **CHF**, **systemic embolism** and/or ventricular **arrhythmias** **Double**/diffuse apical **impulse** EKG: **persistent ST** segment **elevation** Echo: demonstration of the **aneurysm** (paradoxical wall motion) ± mural thrombus	**No specific** therapy (manage associated complications)
Ventricular **False Aneurysm**	Ventricular **free wall rupture** with **containment** by **pericardium** ↑ **risk** of **rupture**	**Surgical** repair
Systemic (Arterial) **Embolism**	Associated with **anterior MI** ± **LV dysfunction** Echo: **mural thrombus**	**Warfarin** anticoagulation for a period of **3-6 months** (**INR 2.0 – 3.0**)
Post-MI angina	Consider infarct **extension** and/or **re-infarction**	**Emergent angiography** + **CABG**
Congestive Heart Failure	Symptoms/signs of **CHF** (*see below*) **Pulmonary congestion** and/or peripheral **hypoperfusion** (↑ PCWP and/or ↓ CI**)**	If **PCWP < 15** mmHg, consider **IV fluid** resuscitation If **PCWP > 15** mmHg, give **diuretics** ± **inotropic** agents (e.g. digoxin, dobutamine) Long-term management → **ACE-inhibitors** and/or β-**blockers** (↓ mortality)
Arrhythmias	Any **tachy-** or **bradyarrhythmias** possible Usually occur **within** several **hours** or > **48 hours** post-**MI**	**Prophylactic** treatment **not indicated** (no survival benefit or ↑ mortality) Treatment depends on the type of arrhythmia and the hemodynamic status of the patient (*see below*)

* ↓ **CI** (Cardiac Index) → **< 2.2** L/min/m²

* **Premature Ventricular Contractions** (PVCs) → give **β-blockers**; correct **electrolyte** abnormalities (if applicable)

* Although **lidocaine** is **effective** in **preventing** post-MI **VF** (ventricular fibrillation), its prophylactic use is **discouraged** because of **no survival benefit** (↑ incidence of **asystole**)

* Sinus bradycardia and/or **AV block + inferior**/right ventricular **MI** → give **atropine**

* **AV block** + **anterior MI** → **pacemaker** (also used for **bradyarrhythmias unresponsive** to **atropine**)

* **Most common** cause of **sudden death** post-MI → **VF**

Sudden Death

- May be the **initial manifestation** of **CAD** in ~ 20-30% of cases
- **Most common** cause of **death** related to **CAD**
- Secondary to **VF** (most common), **Vtach** and/or ventricular **asystole**
- **Better prognosis** when associated with **MI** (↓ risk of **recurrence** after successful **resuscitation**)
- Etiology → **MI**/ischemia, **severe LV dysfunction** (e.g. ventricular aneurysm), **hypertrophic cardiomyopathy**, **long-QT** syndromes
- Acute Management → **CPR,** electrical **defibrillation** ± anti-arrhythmic medications
- Long-term Management:
    1. **Not associated** with MI → **electrophysiologic** study + **ICD** (implantable cardioverter-defibrillator) placement
    2. **Associated** with **MI** → management depends on **LV function 1 month** post-MI:
        - **EF < 30%** → **ICD** placement
        - **EF 30-40%** → **electrophysiologic** study ± **ICD** (in patients with inducible VF/Vtach)
        - **EF > 40%** → no further management required

Non-Atherosclerotic CAD

- Coronary **embolism** → infective **endocarditis**, **mural thrombus** (e.g. post-MI, atrial fibrillation), cardiac **myxoma**
- Coronary **vasculitis** → **SLE, Kawasaki's** disease
- Coronary artery **spasm** → **cocaine** abuse
- **Anomalous origin** of coronary arteries → **young** patient with **MI + no cocaine**
- **Hypercoagulability** → **Factor V** mutation, **anti-phospholipid** antibody syndrome
- Coronary artery **dissection** → **primary** and/or associated with **aortic dissection**
- Post-**radiotherapy**
- Post-cardiac **transplantation** → **major limiting** factor for successful transplantation

* Remember → **withhold β-blockers** for a period of **12-24 hours** before **stress testing** (digoxin should also be discontinued, if possible)

* Remember → **nitroglycerin** (or any other nitrate) is **contraindicated** for **24 hours** since the **last dose** of **sildenafil**/Viagra (↑ risk of **severe hypotension**)

Drugs used in the management of CAD

Anti-Anginal Medications		
Nitrates (nitroglycerin, isosorbide dinitrate)	↓ **preload** ↓ coronary **spasm** ↓ platelet **aggregation** ↓ **afterload**	Side effects → **headache**, flushing, **hypotension**, **tachycardia**, fluid retention, **methemoglobinemia** Contraindications → **sildenafil** (within 24 hours), **BP < 90** mmHg**,** suspected **RV infarction** Development of **tolerance** (**tachyphylaxis**) with **long-term** use (manage with **drug-free** periods of ≥ 12 hours)
β-blockers (metoprolol, carvedilol, atenolol, propranolol, etc.)	↓ cardiac **contractility** ↓ **HR** (heart rate) ↓ **afterload**	Side effects → **insomnia**, **depression**, **sexual** dysfunction, **bronchospasm**, exacerbation of **peripheral artery disease** (PAD), **hypotension**, ↓ AV conduction, **"masking"** of hypoglycemia, exacerbation of **CHF, ↑ LDL** cholesterol Contraindications → severe **CHF**, **AV block**, **BP < 90** mmHg**, HR < 60,** history of **asthma**, COPD, PAD
Ca-channel blockers (verapamil, diltiazem, dihydropyridines → amlodipine, nicardipine, etc.)	↓ **afterload** ↓ coronary **spasm** ↓ HR and contractility (with verapamil and diltiazem)	Side effects → flushing, **constipation**, peripheral **edema**, exacerbation of **CHF**, ↓ AV conduction (with verapamil, diltiazem) Contraindications → **BP < 90** mmHg, **CHF**, **AV block**

* **Idiosyncratic** reaction to **nitroglycerin**, manifested as **sudden** onset of **severe hypotension** → treat with **atropine**

Anti-Platelet Medications		
Aspirin (ASA)	↓ **cycloxygenase** activity → ↓ **thromboxane A2** synthesis ↓ **platelet aggregation**	Side effects → **PUD**/GI **bleeding**, "**salicylism**" (tinnitus, ↓ hearing, vertigo), exacerbation of **asthma**, ↑ **anion gap metabolic acidosis** **Avoid** in patients with **nasal polyps** ± chronic **rhinitis** (hypersensitivity reaction → **severe bronchospasm**)
Clopidogrel, Ticlopidine	Blockade of **ADP receptors** → ↓ platelet **aggregation**	Side effects → **bleeding** **Neutropenia** and/or **TTP** (↑ risk with **ticlopidine**)
GP IIb/IIIa Inhibitors (abciximab, tirofiban, eptifibatide, lamifiban)	↓ **vWF** and **fibrinogen** binding → ↓ platelet **aggregation**	Side effects → **bleeding**, thrombocytopenia, coronary artery **dissection**

* **Most potent** anti-platelet agents → **GP IIb/IIIa inhibitors**

* **Thrombolytic** agents (**alteplase** (t-PA), **streptokinase**, anistreplase, **reteplase, tenecteplase**) → side effects include:

1. **bleeding**
2. **intracranial hemorrhage**
3. **allergic** reactions (streptokinase, anistreplase)
4. **hypotension**
5. ↑ risk of **myocardial rupture** (if performed > 12 hours post-MI)

* **Antidote** for **bleeding** secondary to **thrombolytic** agent **toxicity** → **ε-aminocaproic acid** (or any other anti-fibrinolytic agent)

Valvular Heart Disease

Aortic Stenosis

APPROVED
By Zurab Azmaiparashvili at 2:42 am, Jan 24, 2009

- Etiology:
  1. **Senile calcific aortic stenosis** (degenerative aortic valve disease) → **most common**; age > **55; males** > females
  2. **Bicuspid** aortic **valve** → age < **55**; associated with **coarctation** of the **aorta**
  3. **Rheumatic** heart disease → ~ **15 years** after **rheumatic fever**; **universally** associated with **mitral** valve disease (stenosis and/or regurgitation)
  4. **Congenital** aortic stenosis
- Pathophysiology: **pressure overload** of the **LV / pressure gradient** across the **aortic valve** (LV systolic pressure > aortic systolic pressure) → **concentric** LV **hypertrophy** (compensatory reaction maintaining normal cardiac output) → **diastolic dysfucntion** (↑ stiffness) → **systolic dysfunction** (final stage)
- Symptoms:
  1. **Angina** → secondary to ↑ **O_2 demand** (from LV hypertrophy) + **subendocardial ischemia** (↑ LV filling pressures → ↓ diastolic coronary blood flow) ± coexistent CAD; **prognosis → 50% 5-year survival**
  2. Exertional **syncope** → secondary to **reflexive vasodilation** (induced by exertion) + **fixed cardiac output** (inability to ↑ CO with exercise), leading to ↓ **BP**; possible ventricular/atrial arrhythmias; **prognosis → 50% 3-year survival**
  3. **Dyspnea** (symptoms/signs of CHF) → **prognosis 50% 1-year survival**
- Signs:
  1. **Pulsus parvus et tardus** (reduced and delayed carotid upstroke)
  2. **Carotid thrill**
  3. Sustained/forceful apical impulse (usually not displaced)
  4. **Soft** and/or **absent A_2** sound (possible **paradoxical splitting** of **S_2**)
  5. **S_4** sound (may be palpable)
  6. **Ejection click** (shortly after **S_1**) with **bicuspid** valve
  7. Harsh, **crescendo-decrescendo systolic murmur** (best heard at the right 2nd intercostal space) **radiating** to the **carotids** and/or the **apex** (Gallavardin phenomenon)
  8. ↑ **murmur** with **amyl nitrite** (↓ afterload) and/or **leg raising** (↑ preload)
  9. ↓ **murmur** with **Valsalva** maneuver (↓ preload) and/or **handgrip** (↑ afterload)
- EKG → LV **hypertrophy** ± **LBBB**
- Chest X-ray → post-stenotic **dilation** of aortic **root**, **cardiomegaly** ± aortic valve **calcification**
- **Best initial** diagnostic test → **echocardiography** showing ↓ **excursion** of aortic valve **leaflets**, ↓ aortic valve **surface area**, **LV hypertrophy** and/or **pressure gradient** across the aortic valve
- **Most accurate** diagnostic test → **left-heart catheterization**
- Classification of aortic stenosis:
  1. **normal → 2.5 - 3.5** cm²
  2. **mild →** 1.5 – 2.0 cm²
  3. **moderate →** 1.0 – 1.5 cm²
  4. **severe → 0.75 – 1.0** cm²
  5. **critical →** < **0.75** cm²

- Management:
    1. **Asymptomatic** aortic stenosis → **serial echocardiograms** (every 6-12 months with severe stenosis) + patient **education** (↓ exercise, nature of symptoms, etc.); **avoid vasodilators** (e.g. ACE inhibitors, hydralazine, nitroglycerin)
    2. **Symptomatic** aortic stenosis → **aortic valve replacement**
    3. **Critically ill** patient + **indications** for treatment → aortic valve **balloon valvuloplasty**

* **Indications** for **aortic valve replacement** →
    1. **symptomatic** disease
    2. **critical** stenosis
    3. **severe stenosis** + **LV dysfunction**
    4. ≥ **moderate** stenosis + **CABG**

* Remember → there is **no medical treatment** for **aortic stenosis**

* According to 2007 guidelines **endocarditis prophylaxis** is **no longer recommended** for **acquired valvular disease**

- Differential Diagnosis →
    1. Hypertrophic cardiomyopathy → ↑ **murmur** with **Valsalva** maneuver and/or **amyl nitrate**; ↓ **murmur** with **handgrip, squatting** and/or **leg raising**
    2. Mitral regurgitation → **holosystolic** murmur **radiating** to the **axilla** (**never** to the **carotids**)
    3. Pulmonary stenosis → ↑ **murmur** with **inspiration**

Aortic Insufficiency/Regurgitation

- Etiology:
    1. **Valvular** → bicuspid valve, **infective endocarditis**, **rheumatic fever** (associated with aortic stenosis ± mitral valve disease), connective tissue disorders (SLE, RA)
    2. **Aortic root dilatation** (supravalvular) → **hypertension** (most common cause; ↑ incidence with age), **Marfan** syndrome, **syphilis, ankylosing spondylitis,** aortic **dissection**

* Etiology of **acute** aortic insufficiency → infective **endocarditis**, aortic **dissection**, aortic **trauma**

- **Men** > Women
- Pathophysiology: **volume overload** of the **LV** → **eccentric** LV **hypertrophy** → ↑ chamber size/↑ **end-diastolic volume** → ↑ **stroke volume** → ↑ **pulse pressure** with ↑ **systolic** pressure + ↓ **diastolic** pressure (secondary to regurgitation + ↓ vascular resistance) → ↓ **LV systolic** function → **CHF**

* Pathophysiology of **acute** aortic insufficiency: **sudden** ↑ in **end-diastolic pressure** → pressure **transmitted** to **pulmonary vasculature** → acute **pulmonary edema**

- Symptoms:
    1. Fatigue
    2. **Palpitations** (secondary to arrhythmias and/or hyperdynamic circulation)
    3. **Dyspnea**, orthopnea (secondary to CHF)
    4. Exertional **angina** (secondary to ↓ **diastolic BP** leading to ↓ **coronary perfusion** ± LV hypertrophy)
    5. Possible **syncope** (secondary to ↓ **mean BP** leading to ↓ **cerebral perfusion**)

* Symptoms of **acute** aortic insufficiency → **dyspnea**, hemodynamic instability ± manifestations of **underlying disease** (e.g. chest pain from aortic dissection)

- Signs:
    1. **Corrigan pulse** → rapid, full upstroke with **rapid collapse**
    2. **De Musset** sign → **head bobbing** with each systole
    3. **Duroziez** sign → **"to-and-fro"** (both systolic and diastolic) **murmur**/bruit over **femoral** arteries
    4. **Quincke** sign → **pulsations** in the **nail beds** (e.g. diastolic blanching)
    5. **Traube** sign → **"pistol-shot"** sounds over **femoral** arteries
    6. **Muller** sign → **pulsations** in the **uvula**
    7. **Hill** sing → **20mmHg** difference in femoral/brachial **systolic BP** (↑ femoral)
    8. Pulsus **bisferiens** (with combined stenosis and regurgitation)
    9. Hyperdynamic, **displaced apical impulse**
    10. **Decrescendo, diastolic murmur** best heard at the left (valvular) or right (supravalvular) **sternal border** (duration correlates with severity: ↓ **duration** - ↑ **severity**)
    11. **Murmur** of aortic regurgitation **improves** with ↓ **preload** (e.g. Valsalva maneuver) and/or ↓ **afterload** (e.g. amyl nitrite)
    12. **Systolic murmur** (physiologic, flow murmur) best heard in the 2nd right intercostal space
    13. **Austin-Flint murmur** → **mid-diastolic** murmur resembling **mitral stenosis**
    14. **Soft S_1** sound (secondary to **early mitral** valve **closure**) → correlates with ↑ **severity**
    15. **Soft**/absent **S_2** sound
    16. Possible **S_3** sound → indicates **CHF**

* Signs of **acute** aortic insufficiency → **soft S_1,** soft/absent S_2 ± **short, diastolic murmur**

- EKG → **LV hypertrophy** ± LA enlargement
- Chest X-ray → **LV enlargement** ± LA enlargement and/or aortic root dilatation
- **Best initial** diagnostic test → **echocardiography** showing aortic **regurgitation**, **fluttering** of **anterior mitral leaflet**, LV hypertrophy, aortic root dilatation

* Remember to order **blood cultures** in cases of **acute** aortic insufficiency, if **infective endocarditis** is suspected

- Management:
    1. **Asymptomatic** with **preserved LV** function → **serial echocardiograms** + **salt-restriction** and **diuretics** (preload reduction) + **ACE inhibitors** or nifedipine (afterload reduction; ↓ disease progression)
    2. **Indications** for **aortic valve replacement:**
        - **Symptomatic** aortic regurgitation
        - **EF < 50-55%**
        - **End-systolic LV size > 55 mm**

* Management of **acute** aortic insufficiency → **emergent surgical** intervention (may use **sodium nitroprusside** ± **digitalis** as a **bridge** to surgery)

* **IABP** is **contraindicated** in aortic insufficiency

* **Marfan** syndrome with aortic **root diameter > 5-5.5 cm → aortic root replacement**

Mitral Stenosis

- Almost always secondary to **rheumatic heart disease** (~ 10 years post-infection)
- **Females** > Males
- **Most common** valvular abnormality caused by **rheumatic fever**
- Pathophysiology: **pressure gradient** across the **mitral valve → LA hypertrophy** + ↑ LA pressure → pressure transmitted to the **pulmonary vasculature** → pulmonary **congestion → pulmonary hypertension → RV hypertrophy → cor pulmonale**

* **Cardiac output** becomes more **dependent** on **atrial contraction** → any condition that **impairs**/shortens **ventricular filling** (e.g. atrial fibrillation, tachycardia) leads to clinical **deterioration**

- Symptoms (~ 20 years post-infection):
    1. **dyspnea**, **orthopnea**, paroxysmal nocturnal dyspnea
    2. **hemoptysis**
    3. **palpitations** (secondary to atrial fibrillation)
    4. extreme **weight loss**
    5. manifestations of **systemic embolism** (stagnation of blood ± atrial fibrillation)
    6. **hoarseness** (recurrent laryngeal nerve compression from LA enlargement)
    7. manifestations of **RV failure** (e.g. ascites, peripheral edema)
- Signs:
    1. **loud S_1**
    2. **opening snap/OS** (following S_2 sound) → **S_2 – OS interval** correlated with disease **severity** (↓ interval - ↑ severity)
    3. **mid-diastolic rumble** best heard at the **apex** ± **pre-systolic accentuation** (lost with atrial fibrillation)
    4. **loud P_2** and/or **widely-split S_2** (indicates pulmonary hypertension)
    5. **sternal lift**/right parasternal heave (secondary to RV enlargement)
    6. **irregularly irregular pulse** (with atrial fibrillation)
    7. manifestations of **RV failure** (e.g. hepatomegaly, JVD)
    8. pulmonary **rales**

- EKG → **LA enlargement** ("**P mitrale**") ± RV hypertrophy; **atrial fibrillation**
- Chest X-ray:
    1. **LA enlargement → straight left** heart **border**, **"double density" right** heart **border**, **posterior displacement** of the **esophagus**, **elevation** of **left main bronchus**, splaying of the **carina**
    2. Pulmonary **congestion** → ↑ vascular **markings**, **Kerley-B** lines
    3. Pulmonary **hypertension** → ↑ prominence of **pulmonary arteries**
    4. **RV enlargement** → loss of **retrosternal airspace**
- **Best initial** diagnostic test → **echocardiography** showing ↓ **excursion** of mitral valve **leaflets,** valvular **thickening**, **fusion** of mitral valve **leaflets**, ↓ valvular **surface area**, **LA enlargement**
- Management:
    1. **Avoid** conditions that lead to ↓ **diastolic filling** of the LV (e.g. tachycardia, fever, exercise)
    2. **Asymptomatic** patients → **serial echocardiograms**
    3. **Symptomatic** patients → **salt-restriction, diuretics** (↓ preload); consider β-blockers and/or Ca-channel antagonists (↓ HR)
    4. **Atrial fibrillation** → **anticoagulation** with **warfarin** + **digitalis** for ventricular rate control (although β-blockers and/or Ca-channel antagonists can also be used for the same purpose, **digoxin** is the most common correct answer)
    5. **Indications** for **surgical** therapy:
        - **Symptomatic, severe** mitral stenosis (surface area < 1 cm^2)
        - **Failure** of **medical** therapy
        - **Worsening** pulmonary **hypertension**
        - Recurrent **embolization**
    6. Surgical **procedure of choice** for **young** patients with **non-calcified, pliable** valves → **balloon valvuloplasty** (or mitral **commissurotomy**)
    7. **heavily calcified** valves ± coexistent **mitral regurgitation** → mitral valve **replacement**

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Mitral Insufficiency/Regurgitation

- Etiology:

Acute Mitral Regurgitation	Chronic Mitral Regurgitation
Infective endocarditis **Papillary** muscle **rupture**/dysfunction (secondary to ischemia/MI) **Rupture** of **chordae tenidinae** (spontaneous or associated with trauma and/or myxomatous degeneration) **Prosthetic valve dysfunction**	Mitral valve **prolapse** (most common cause of mitral regurgitation; *see below*) **Rheumatic fever** **Dilated** cardiomyopathy (or any other cause of severe LV dilatation) **Hypertrophic** cardiomyopathy **Congenital** Mitral valve annulus calcification Connective tissue disorders **Drug-**induced (e.g. anorexigenics) Any cause of acute mitral regurgitation

- Pathophysiology: **volume overload** of the **LA** and **LV** → progressive **LA enlargement** and **eccentric LV hypertrophy** → eventual **LV** ± RV **systolic dysfunction** → **CHF**

* Acute mitral regurgitation → **sudden increase** in blood **volume** in a **non-compliant LA** → ↑ **LA pressure** → pressure transmitted to **pulmonary vasculature** → acute **pulmonary edema** ± RV dysfunction

* Vicious cycle: mitral **regurgitation** → **LV dilation** → ↑ **regurgitation. . .**

- Symptoms:
  1. **dyspnea, orthopnea**, paroxysmal nocturnal dyspnea
  2. fatigue
  3. manifestations of **systemic embolism**
  4. manifestations of **pulmonary hypertension**/**RV failure**
- Signs:
  1. **soft**/absent **S_1** sound
  2. **widely-split S_2** sound
  3. **S_3** sound
  4. **holosystolic** murmur **best heard** at the **apex** and **radiating** to the **axilla** and/or **back**
  5. ↑ **murmur** with ↑ **preload** (e.g. leg raising) and/or ↑ **afterload** (e.g. handgrip)
  6. possible **early diastolic** rumble (secondary to ↑ flow across the mitral valve)
  7. **pulsus parvus** (but not tardus)
  8. hyperdynamic, **displaced apical impulse**
  9. manifestations of **RV failure**

* **Severe** and/or **acute** mitral **regurgitation** → ↓ **EF**, **S_3** and/or **diastolic rumble**

- EKG → **LA enlargement** ± **LV hypertrophy**
- Chest X-ray → **cardiomegaly** (both LA and LV enlargement) ± ↑ pulmonary **vascular markings**
- **Best initial** diagnostic test → **echocardiography** showing both **LA** and **LV enlargement**, **mitral regurgitation** ± clues to etiology (e.g. vegetations with infective endocarditis)

- **Most accurate** diagnostic test → cardiac **catheterization**:
    1. **right heart** catheterization → **prominent V waves** in PCWP tracing
    2. **left heart** catheterization → **demonstration** of mitral regurgitation, **severity assessment** ± evaluation of **coronary anatomy**
- Management:
    1. **Asymptomatic** + **normal**/supranormal **EF** → **serial echocardiograms**
    2. **Symptomatic** + **normal**/supranormal **EF** → **ACE inhibitors,** salt-restriction, **diuretics** (add **warfarin** and **digoxin** if **atrial fibrillation**)
    3. Indications for **surgical therapy** (mitral **valve repair** or replacement) →
        - **Failure** of **medical therapy**
        - **Acute** mitral **regurgitation**
        - **EF < 60%**
        - **LV end-diastolic size > 45 mm**

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Mitral Valve Prolapse (MVP)

- Synonyms → **"click-murmur"** syndrome, **Barlow** syndrome
- **Most common valvular abnormality**
- **Most common** cause of **mitral regurgitation** in the USA
- **Females** > Males; **Young age**
- **Systolic bulging** of ≥ 1 **mitral leaflets** into the **LA** ± mitral regurgitation
- Secondary to **myxomatous degeneration** of the **leaflets** and/or **chordae tenidinae**
- Etiology:
    1. **idiopathic**
    2. **connective tissue** disorders (e.g. **Marfan** syndrome, Ehlers-Danlos syndrome)
- Commonly associated with **musculoskeletal** abnormalities (e.g. pectus excavatum)
- Clinical features → **asymptomatic** to atypical **chest pain, dyspnea,** anxiety, **palpitations**, near-**syncope** ± manifestations of **mitral regurgitation**
- Physical examination →
    1. **mid-systolic click** followed by **late systolic murmur** of mitral regurgitation
    2. **click** moves **closer to S_1**/↑ murmur with **Valsalva** maneuver, **standing**, **amyl nitrite**
    3. **click** moves **closer to S_2**/↓ murmur with **squatting** and/or **handgrip**
- **Complications** of MVP
    1. **arrhythmias**/sudden death
    2. **systemic embolism** (especially TIA/stroke)
    3. **rupture** of **chordae tenidinae** (with sudden decompensation)
    4. infective **endocarditis**
    5. progressive **mitral regurgitation**
- EKG (especially 24-hour Holter monitoring) → **arrhythmias** (premature ventricular contractions, atrial fibrillation, supraventricular tachycardia, etc.)
- Chest X-ray → associated **musculoskeletal** abnormalities
- **Best diagnostic** test → **echocardiography** demonstrating ≥ **2mm systolic displacement** of mitral leaflets into the LA ± associated **mitral regurgitation**
- Management:
    1. **Asymptomatic** + **no regurgitation** → **reassurance**; follow-up in 3-5 years
    2. **Symptomatic** → **β-blockers**
    3. Presence of mitral regurgitation → *see above*

Tricuspid Insufficiency/Regurgitation

- Etiology:
  1. **RV dilatation** (**most common**; secondary to **pressure**/volume **overload**)
  2. Infective **endocarditis** (**most commonly** affected valve in **IV drug users**)
  3. **Congenital** (e.g. Ebstein anomaly)
  4. **Rheumatic fever** (accompanied by mitral ± aortic valve disease; primary involvement of the tricuspid valve less common)
  5. **Carcinoid** syndrome
  6. Tricuspid valve **prolapse** (secondary to connective tissue disorders)
- Clinical features → manifestations of **RV failure** (ascites, peripheral edema, hepatomegaly ± RUQ pain, jaundice)
- Physical examination:
  1. **JVD** with **large V waves**
  2. **Hepatojugular reflux**/pulsatile liver
  3. **Sternal lift**/right parasternal heave
  4. **Holosystolic** murmur that **increases** with **inspiration** (left sternal border)
  5. **S_3** sound **best heard** at **lower left sternal border**
- EKG and Chest X-ray → **RV** ± RA **enlargement**
- **Best initial** diagnostic test → **echocardiography**
- Management:
  1. Salt restriction**, diuretics**
  2. **Annuloplasty** for **normal-appearing** tricuspid valve
  3. **Valve replacement**, if **pulmonary hypertension** and/or **valve damage**

* Tricuspid Stenosis → almost always secondary to **rheumatic fever**; **JVD** with **prominent "a" waves; diastolic rumble** that **increases** with **inspiration**; EKG and Chest X-ray → **RA enlargement**

Prosthetic Valves

Bioprosthetic (heterograft, homograft)	***Mechanical***
↓ durability	↑ durability
Anticoagulation not required (only aspirin)	Anticoagulation required (INR 2.5 – 3.5)
Endocarditis prophylaxis required	Endocarditis prophylaxis required
Contraindicated in children	Contraindicated in pregnancy

Diseases of the Myocardium

Dilated Cardiomyopathy (DCM)

APPROVED
By Zurab Azmaiparashvili at 2:42 am, Jan 24, 2009

- **Most common** cardiomyopathy
- **Most common** reason for **heart transplantation**
- Characterized by **biventricular dilatation** and ↓ **EF** in the absence of **pressure** and/or **volume overload** or **significant CAD** (although **CAD** is the **most common** cause **secondary** dilated cardiomyopathy)
- Etiology:
    1. **Idiopathic → most common** cause of DCM**; African-American males** most commonly affected; possible **family history** (genetic component)
    2. **Post-viral →** usually **following** viral **myocarditis** (e.g. Coxsackie virus)
    3. **Alcohol** abuse **→ most common reversible** cause of DCM
    4. **Drug-**induced **→ doxorubicin**, **trastuzumab**, other chemotherapeutic agents
    5. **Metabolic → thiamine** (vitamin B_1) deficiency (**"wet beriberi"**), hypophosphatemia, hypocalcemia, uremia, **carnithine** deficiency, **selenium** deficiency
    6. **Endocrine → thyroid** disorders, **pheochromocytoma**
    7. **Connective tissue** disorders (e.g. SLE, RA)
    8. **Postpartum** (or peripartum) cardiomyopathy
    9. **Prolonged tachycardia**
    10. Selected causes of **myocardial infiltration** (e.g. **hemochromatosis**)
    11. **Toxins → cocaine**, **lead** and/or **mercury** poisoning
    12. **Neuromuscular** disorders (e.g. **Duchenne** muscular dystrophy)
- Clinical features → manifestations of **LV failure** ± **RV failure** (*see congestive heart failure*), atypical **chest pain**, both **atrial** and **ventricular arrhythmias**, manifestations of **systemic embolism**
- Physical examination:
    1. **Jugular venous distention** (JVD) ± parasternal lift
    2. **Displaced apical impulse**
    3. **S_3** sound
    4. **Murmur** of **mitral regurgitation**
- EKG **→ LV hypertrophy**, **LBBB** and/or RBBB, **non-specific ST-T** wave abnormalities ± atrial and/or ventricular **arrhythmias**
- Chest X-ray **→ cardiomegaly** ± pulmonary **congestion**
- **Best initial** diagnostic test **→ echocardiography** demonstrating **biventricular** (possible **four-chamber**) **dilatation** and ↓ **EF** ± mitral and/or tricuspid **regurgitation**
- **Most accurate** diagnostic test for **heart failure → MUGA scan** (multi gated acquisition scan):
    1. **More accurate** than echocardiography
    2. **Use** in patients on **doxorubicin** and/or trastuzumab therapy for **EF assessment**
- Management:
    1. **Treatment** of **reversible** causes of DCM (e.g. **alcohol abstinence**)
    2. Routine **CHF therapy** (e.g. diuretics, ACE inhibitors, β-blockers) **→** *see CHF*
    3. **Anticoagulation**, if atrial **fibrillation**, **mural thrombus**, systemic **embolism** and/or ↓↓ **EF**
    4. Management of **arrhythmias** (consider ICD placement)
    5. **Definite therapy →** cardiac **transplantation**
- **Leading cause** of **mortality → sudden death** (secondary to arrhythmias)

Hypertrophic Cardiomyopathy (HCM)

- Synonyms → hypertrophic obstructive cardiomyopathy (**HOCM**), idiopathic hypertrophic subaortic stenosis (**IHSS**), asymmetric septal hypertrophy (**ASH**)
- Characterized by marked **LV hypertrophy** (unexplained by hypertension and/or aortic stenosis), **asymmetric septal hypertrophy, diastolic LV dysfunction** ± LV outflow tract obstruction
- Etiology:
    1. **Autosomal dominant** mutations involving the **sarcomere** (~50% of cases)
    2. **Sporadic**

* **Secondary** hypertrophic cardiomyopathy → **hypertension**, **aortic stenosis**, myocardial **infiltration**

- **Most common** cause of **sudden death** in otherwise **healthy, young athletes**
- Pathophysiology:
    1. ↓ **compliance** + abnormal relaxation → **diastolic dysfunction** → ↑ **end-diastolic pressure** → manifestations of **angina** and **dyspnea**
    2. **Asymmetric septal** hypertrophy → **narrowing** of the **LVOT** (LV outflow tract) → **systolic anterior motion** of the **mitral** valve (Bernoulli effect) → **dynamic** LVOT **obstruction** ± mitral **regurgitation**
    3. ↑ **arrhythmogenic** potential of the **myocardium** → manifestations of **syncope** ± **sudden death**

* **HCM** is associated with ↑ **EF** (secondary to ↑ systolic function)
* A **small proportion** of patients with **HCM progress** to **systolic dysfunction** and **DCM**

- Symptoms (especially common in **older patients**) → **triad** of **chest pain**, **syncope** (usually post-exercise) and **dyspnea**

* **Younger** patients **most commonly** present with **syncope** and/or **sudden death**

- Signs:
    1. **Pulsis bisferiens**
    2. **Prominent** jugular venous **"a" waves**
    3. "Double" or "**triple**" **apical impulse**
    4. **S4** sound
    5. Loud, **systolic ejection murmur** (no radiation to the carotids) ± murmur of **mitral regurgitation**
- **Factors** that **influence** dynamic **LVOT obstruction** (and change murmur intensity):

↑ **LVOT obstruction** (↑ murmur intensity)	↓ **LVOT obstruction** (↓ murmur intensity)
Decreased afterload: Amyl nitrite Vasodilators (e.g. nitroglycerin)	**Increased afterload:** Handgrip Phenylephrine Squatting
Decreased preload: Valsalva maneuver Dehydration Diuretics Nitroglycerin Tachycardia	**Increased preload**: Blood volume expansion Bradycardia

Increased contractility:	**Decreased contractility:**
Digoxin, dobutamine, dopamine Post-PVC	β-blockers Ca-channel antagonists (e.g. verapamil) Disopyramide Sedation/Anesthesia

- EKG → **LV hypertrophy**, **non-specific ST-T** wave changes, "**pseudo-Q**" waves in **precordial leads**; possible **ventricular arrhythmias** (more commonly detected during 48-72 h EKG monitoring)
- **Best initial** diagnostic test → **echocardiography** showing marked **LV enlargement** ± **asymmetric septal hypertrophy**, **systolic anterior motion** of the **mitral valve** and/or **early closure** of the **aortic valve**

* **Risk factors** for **sudden death** in patients with HCM →

1. **family history** of **sudden death**
2. **personal history** of **sudden death**
3. **young** age; **male** gender
4. history of **syncope**
5. ↑↑ **LV hypertrophy**
6. **ventricular tachycardia**

- Management:
  1. **Asymptomatic** + no/minimal risk for sudden death → ↓ **exercise, avoid factors** that lead to ↑ **LVOT obstruction**; **yearly follow-up**
  2. **Asymptomatic** + **high risk** for **sudden death** → as above + **ICD placement** or **amiodarone** therapy
  3. **Symptomatic** + no/minimal risk for sudden death → **high-dose β-blockers**
  4. **Symptomatic** + **high risk** for **sudden death** → as above + **ICD placement** or **amiodarone** therapy
  5. **Symptomatic** + **contraindications** to/**failure** of **β-blockers** → consider **verapamil** and/or **disopyramide**
  6. **Symptomatic despite** optimal **medical therapy** → **surgical myectomy** or **alcohol**-induced septal **ablation**; consider **dual-chamber pacing** as an alternative
  7. **Progression** to **DCM** → consider **heart transplantation**

* Don't forget to **screen 1st degree relatives** (physical exam, EKG, echocardiography)

* Although HCM is the leading cause of sudden death, **echocardiography** is **not** to be **used** as a **screening** tool in **asymptomatic** young athletes with **normal physical** exam

Restrictive Cardiomyopathy (RCM)

- A group of disorders characterized by **stiff, non-compliant myocardium** resulting in **diastolic dysfunction**
- Etiology:
    1. Idiopathic
    2. Myocardial **infiltration → amyloidosis**, **hemochromatosis**, **sarcoidosis**, **glycogen storage** disorders
    3. **Carcinoid** syndrome
    4. **Hyper-eosinophilic** syndromes (e.g. Loeffler's endocarditis)/**endomyocardial fibrosis** (primary disorder or terminal stage of hyper-eosinophilic syndromes)
    5. **Scleroderma**
    6. **Radiation** exposure

* **Endomyocardial fibrosis →** seen in **tropical Africa** (**Loeffler's** endocarditis has **no geographic predilection**; otherwise quite similar, at least for the USMLE)

* Clues to **Loeffler's** endocarditis **→ young male**, **small**/normal **ventricles**, **thrombosis** involving the **apex** and/or the **inflow tract** (under the mitral valve) of the **LV** causing **systemic embolism** ± **mitral regurgitation;** eventual **fibrosis** (secondary endomyocardial fibrosis) with progression to **RCM**

- Pathophysiology: ↓ **compliance → diastolic dysfunction →** ↑ **filling pressures** (with manifestations of systemic/pulmonary congestion) ± ↓ **cardiac output** (despite **normal EF**)
- Clinical features → similar to **constrictive pericarditis** (major differential diagnosis) with **manifestations** of **RV** and **LV failure** (edema, ascites, dyspnea, fatigue, etc.)
- Physical exam:
    1. **JVD** with **rapid X** and **Y** descents
    2. Possible **Kussmaul sign** (↑ JVP with inspiration)
    3. **Soft** heart sounds
    4. Possible **S_3** and/or **S_4**
- EKG **→ low voltage**, "pseudo-Q" waves, **conduction abnormalities**
- Chest X-ray → pulmonary **congestion** ± bilateral **pleural effusion** + **normal** heart size
- **Best initial** diagnostic test **→ echocardiography** showing **thickening** of the **myocardium**, **normal** ventricular **cavity** size, **atrial enlargement** and **normal EF**

* **Low-voltage EKG + thick myocardium = RCM**

* **Echocardiographic** findings suggestive of **amyloidosis →** myocardial "**speckling"** (granular, sparkling appearance)**, pericardial effusion**

* **Echocardiographic** findings suggestive of **endomyocardial fibrosis → apical thrombosis**, **thick endocardium** under the **mitral** valve, mitral **regurgitation**

- **Most accurate** diagnostic test → cardiac **catheterization** showing the **"square-root" sign** or **"dip and plateau" pattern** (rapid rise in diastolic pressure with subsequent plateau); **elevated** (but **not equal**) **diastolic pressures**
- **Most accurate** diagnostic test for **etiology determination → endomyocardial biopsy**

- Management:
    1. **treatment**/correction of any **underlying abnormality**
    2. **no effective** medical therapy for **idiopathic RCM**
    3. **symptomatic** treatment (e.g. diuretics for CHF)
    4. consider **heart transplantation**

Myocarditis

- **Inflammation** of the **myocardium** with ↑ risk of **progression** to **DCM**
- Etiology:
    1. **Idiopathic** (**most common**; presumed to be viral)
    2. **Infectious:**
        - **Viral** (e.g. **Coxsackie virus**, **adenovirus**, HIV, EBV) → **most common known** etiology in the **USA**
        - **Bacterial** (e.g. S. *aureus*, **diphtheria**)
        - **Lyme** disease (Borrelia *burgdorferi*)
        - **Chagas** disease (Trypanosoma *cruzi*) → **most common** cause in **Central** and **South America**
    3. **Drug**/Toxin-related → **doxorubicin**, **cocaine**
    4. Scorpion/Snake **venom**
    5. **Connective tissue** disorders → SLE, RA, dermatomyositis
    6. **Granulomatous** disorders → **sarcoidosis**
    7. Acute **rheumatic fever**
- Clinical features → **asymptomatic** to **chest pain**, manifestations of **CHF**, **palpitations** (secondary to arrhythmias), ↑ **temperature**; possible **sudden death** (**antecedent viral infection** common)
- Lab findings → ↑ **ESR**, ↑ WBC count, ↑ **CK-MB** and/or cardiac **troponins**, possible ↑ **antibody titers** against suspected viruses
- EKG → **non-specific ST-T** wave changes ± **conduction** abnormalities
- Chest X-ray → **cardiomegaly** ± pulmonary **congestion**
- Echocardiography → **diffuse**/local **wall motion abnormalities,** ↓ **EF** ± chamber **dilatation**
- **Most accurate** diagnostic test → **endomyocardial biopsy**
- Management → **supportive**; consider heart **transplantation** in **fulminant** myocarditis

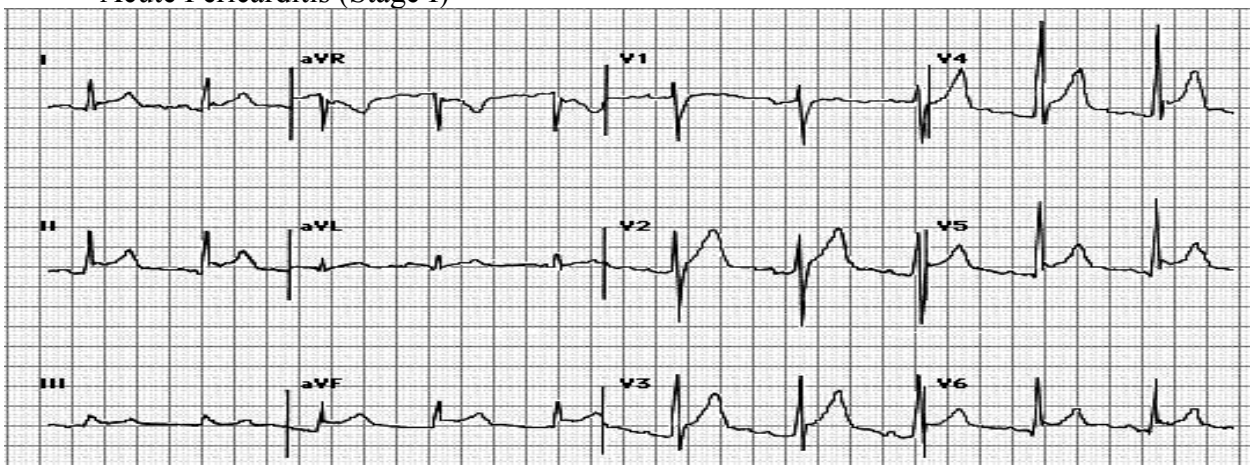
Diseases of the Pericardium

Acute Pericarditis

APPROVED
By Zurab Azmaiparashvili at 2:43 am, Jan 24, 2009

- Etiology:
  1. **Idiopathic** (presumed to be viral)
  2. **Infectious**:
     - **Viral** (e.g. Coxsackie virus, adenovirus, HIV) → **most common** (after idiopathic pericarditis)
     - **Bacterial** (e.g. S. *aureus*, S. *pyogenes*)
     - **TB**
  3. **Post-MI:**
     - Local **irritation** (following transmural MI) → **within 1 week**
     - **Dressler** syndrome (autoimmune) → **after 1 week**
  4. **Connective tissue** disorders (e.g. SLE, RA, scleroderma)
  5. **Uremia**
  6. **Hypothyroidism**
  7. **Radiation** exposure
  8. **Post-cardiotomy** syndrome → **within** several **weeks** of **open heart** surgery
  9. **Malignancy** (e.g. **lymphomas**, breast and lung carcinomas, melanoma)
  10. **Drug**-induced → **procainamide, hydralazine** (drugs that cause lupus-like syndrome)
- Clinical features → possible **antecedent URI** followed by **"pleuritic" chest pain**, ↑ **temperature**, myalgias, headache, fatigue ± **friction rub** (**scratchy sound** with **3 components** → atrial systole, ventricular systole and ventricular diastole)
- Characteristics of **chest pain** in **acute pericarditis**:
  1. **substernal** location
  2. **radiation** to the **trapezius**
  3. **worse** with **deep breathing**, **swallowing** and/or **lying down**
  4. **improves** with **sitting up** and/or **leaning forward**
- Chest X-ray → **normal** to ↑ cardiac silhouette (*see pericardial effusion*)
- **Best initial** diagnostic test → **EKG:**
  1. Stage I → **diffuse, concave ST** segment **elevation** (except aVR and V_1) ± **PR** segment **depression** + **upright T** waves
  2. Stage II → **normal ST** segment ± **PR** segment **depression** + **upright T** waves
  3. Stage III → **normal ST** and **PR** segments + **inverted T** waves
  4. Stage IV → **normalization** of **all** EKG changes
- Order **echocardiography** if you suspect:
  1. **Myocarditis**
  2. Pericardial **effusion**
- Management:
  1. **Treatment** of any **underlying** disease (e.g. **dialysis** for **uremia; drainage** + **antibiotics** for **bacterial** pericarditis)
  2. **NSAIDs** (e.g. aspirin, ibuprofen) → **symptomatic** relief (consider adding **colchicine** to prevent **recurrent** pericarditis)
  3. **Steroids** → **intractable** cases
  4. Avoid **anticoagulation** → ↑ risk of progression to **hemorrhagic** pericarditis
- Complications → pericardial **effusion**, cardiac **tamponade**, **constrictive** pericarditis, **recurrent** pericarditis

- Acute Pericarditis (Stage I)



Pericardial Effusion

APPROVED
By Zurab Azmaiparashvili at 2:43 am, Jan 24, 2009

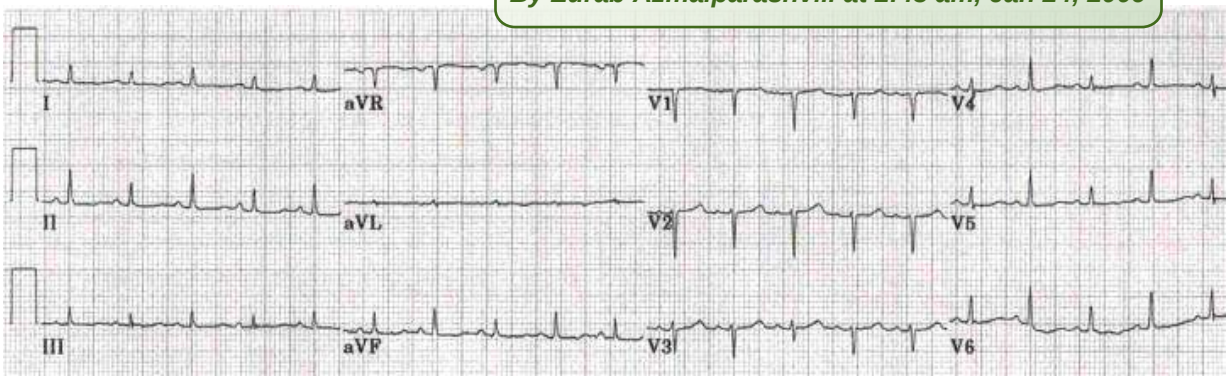
- Virtually **any cause** of acute pericarditis can result in **exudative** pericardial **effusion:**
    1. **Serosanguinous** pericardial effusion → consider **TB, malignant** effusion, **uremia**
    2. **Frank blood** (**hemopericardium**) → consider myocardial **free wall rupture**, **coagulopathy**, aortic **dissection**, **trauma**
- **Transudative** pericardial effusion → consider **CHF**, **cirrhosis**, **nephrotic** syndrome
- Clinical features → **similar** to acute pericarditis, except:
    1. **distant**/soft heart **sounds**
    2. **non-palpable** apical impulse
- Chest X-ray → ↑ **cardiac silhouette** ("water-bottle" appearance) + **no congestion**
- EKG → **low voltage** ± **electrical alternans**
- **Best initial** diagnostic test → **echocardiography** ("echo free" space)
- **Most accurate** diagnostic test → **pericardiocentesis**
- Management → **similar** to acute pericarditis + **serial echocardiograms** (consider **fluid aspiration**)
- Complications → cardiac **tamponade**

Cardiac Tamponade

- Syndrome characterized by **rapid accumulation** of and/or **large volume** pericardial **effusion** leading to **compression** of the heart
- Etiology → virtually **any cause** of acute pericarditis can result in cardiac **tamponade**; **most common**/notable examples include:
    1. **malignancies** (most common)
    2. **idiopathic** pericarditis
    3. **uremia**
    4. **infectious** pericarditis → bacterial, **TB**
    5. **trauma**
    6. aortic **dissection**
    7. ventricular **free wall rupture**

- Pathophysiology: ↑ **intrapericardial** pressure → ↑ **diastolic pressure** → ↓ **venous return** → ↓ **CO** → cardiogenic **shock**
- Clinical features:
    1. **Beck's triad** → hypotension + JVD + "muffled" (distant/soft) heart sounds
    2. **Tachycardia**, tachypnea
    3. **Pulsus paradoxus** → > 10 mmHg fall in BP with inspiration (↓/absent pulse with inspiration)
    4. **JVD** with **prominent X** but **absent Y** descent
    5. **Narrow pulse pressure**
- Chest X-ray → ↑ **cardiac silhouette** ("water-bottle" appearance)
- EKG → **low voltage, electrical alternans**
- **Best initial step** in patient management → **emergent pericardiocentesis**
- **Best initial** diagnostic test → **echocardiography** showing **large** pericardial **effusion** + ↓ **diastolic filling**
- Cardiac catheterization → **elevated** and **equal diastolic pressures** (**equilibration** of diastolic pressures)
- Management → **emergent pericardiocentesis** (consider "pericardial window" or pericardiotomy as an alternative) ± **IV fluids** and/or **pressor** agents

- Electrical Alternans:



Constrictive Pericarditis

- Syndrome characterized by **diffusely thickened, calcified, non-compliant pericardium** resulting in ↓ **ventricular filling**
- May follow virtually **any cause** of **acute pericarditis**
- **Most commonly** follows:
    1. **TB**
    2. **Radiation** exposure
    3. Open heart **surgery**
    4. **Recurrent** pericarditis
- Clinical features → manifestations of **biventricular** heart **failure** (with symptoms/signs of **RV failure predominating**):
    1. Peripheral **edema, ascites, RUQ tenderness**
    2. **Dyspnea** on exertion ± orthopnea
    3. **Hepatomegaly** ± jaundice
    4. **JVD** with **prominent X** and **Y descents**

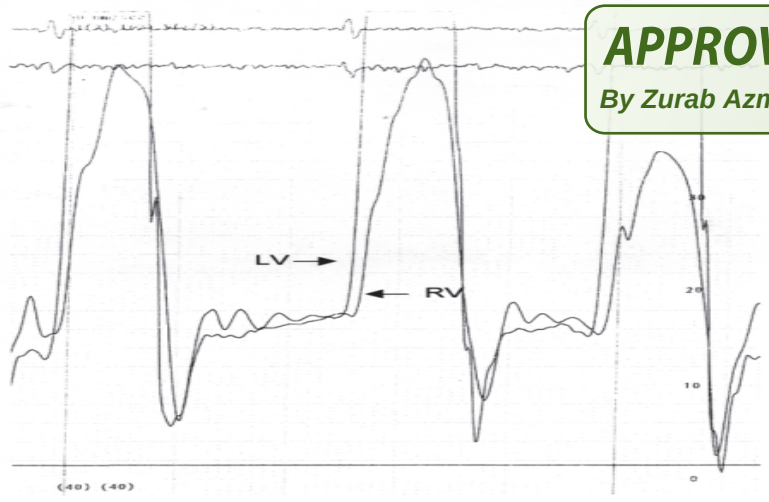
5. **JVD** with **inspiration** → **Kussmaul** sign
6. **Distant**/soft heart **sounds** ± **pericardial knock** (early diastolic sound; higher pitched than S_3)

- Chest X-ray → pericardial **calcifications**
- EKG → **low voltage** ± atrial **fibrillation**
- Echocardiography → ↑ early diastolic **mitral flow rate** (↓ flow with **inspiration**) ± **thick pericardium**
- **Most accurate** diagnostic test → chest **CT** or **MRI** showing **thickened, calcified** pericardium
- Cardiac catheterization → **elevated** and **equal diastolic pressures**, **"square-root" sign** or **"dip and plateau"** pattern, **normal CO**
- Management:
  1. **Salt** restriction, **diuretics**
  2. Definite treatment → **pericardiectomy** or "pericardial stripping")

Differential Diagnosis of RCM, Cardiac Tamponade and Constrictive Pericarditis

	Restrictive Cardiomyopathy	***Cardiac Tamponade***	***Constrictive Pericarditis***
Pulsus Paradoxus	-	+	-
Kussmaul Sign	±	-	+
Pericardial Knock	-	-	+
Prominent Y descent	±	-	+
Low Voltage	+	+	+
Electrical Alternans	-	+	-
Thick Myocardium	+	-	-
Thick Pericardium	-	-	+
Pericardial Effusion	-	+	-
"Square-Root" Sign	+	-	+
Equilibration of Diastolic Pressures	-	+	+

- "Square-Root" Sign with Equilibration of Diastolic Pressures:



Bradyarrhythmias/Conduction Abnormalities

APPROVED
By Zurab Azmaiparashvili at 2:43 am, Jan 24, 2009

Sinus Bradycardia

- **Sinus rhythm** with **rate < 60 bpm**
- Etiology:
    1. ↑ **vagal tone** → cardiovascular **conditioning** (e.g. athletes), **inferior MI**, **vomiting**, ↑ ICP (intracranial pressure), **carotid sinus** syndrome, **straining** (e.g. Valsalva maneuver)
    2. **Drug**-induced → β-**blockers**, **Ca**-channel **antagonists**, cardiac glycosides
    3. **Hypothyroidism**
    4. Hypothermia
    5. **SA node** dysfunction
- Clinical features → **asymptomatic** to **palpitations** and/or **hemodynamic instability** (e.g. chest pain, confusion, syncope, dyspnea/CHF, hypotension)
- EKG → **normal P** waves **preceding** each **QRS** complex; **rate < 60 bpm**
- Management:
    1. **Asymptomatic** → treatment **not indicated**
    2. **Acutely** symptomatic → **atropine**
    3. **Acutely** symptomatic **despite** atropine → **temporary pacemaker**
    4. **Symptomatic** (chronic) → permanent **pacemaker** (definite therapy)
    5. **Drug**-induced → **removal** of any offending agent ± specific **antidotes** (e.g. **glucagon** for **β-blockers**)
    6. Secondary to **hypothyroidism** → **L-thyroxine** replacement

* Sinus Arrhythmia → **normal** variant; ↑ HR with **inspiration** and ↓ HR with **expiration**

* SA Node Dysfunction → secondary to **structural** abnormalities (e.g. amyloidosis, sarcoidosis, age-related fibrosis, post-MI scarring); may result in sinus **bradycardia**, sinus **pause**/**arrest** and/or **tachycardia-bradycardia** syndrome; management → permanent **pacemaker** usually required

* Tachycardia-Bradycardia Syndrome (Sick Sinus Syndrome) → alternation of **bradyarrhythmias** with **tachyarrhythmias** (especially atrial **fibrillation** and/or **flutter**); management → permanent **pacemaker** (for bradyarrhythmias) + medications that **decrease HR** (for associated tachyarrhythmias)

* Carotid Sinus Syndrome (Carotid Sinus Hypersensitivity) → **carotid massage** induced **sinus pause > 3 sec** in duration and/or **> 50 mmHg decrease** in BP; management → permanent **pacemaker** (avoid diagnostic carotid massage in patients with audible carotid bruits and/or history of cerebrovascular disease)

* **EKG Classification** of Atrioventricular (AV) Block:
    1. 1st degree AV block
    2. 2nd degree AV block:
        - Mobitz type I (Wenckebach)
        - Mobitz type II
    3. 3rd degree (complete) AV block

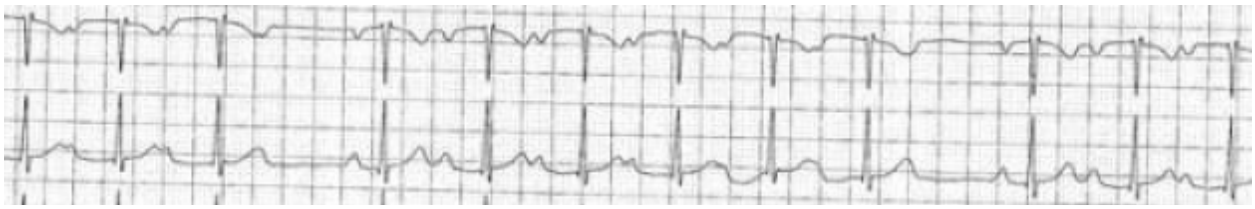
First Degree AV Block

APPROVED
By Zurab Azmaiparashvili at 2:43 am, Jan 24, 2009

- Etiology:
    1. ↑ **vagal tone**
    2. Acute **inferior**/right ventricular **MI**
    3. **Myocarditis** (e.g. Lyme disease)
    4. **Infiltrative** disorders (e.g. amyloidosis)
    5. Acute **rheumatic fever**
    6. **Drug**-induced (e.g. β-blockers, Ca-channel antagonists, glycosides)
    7. Age-related **fibrosis**
- Usually **asymptomatic**
- EKG → **PR interval > 0.2 s**
- Management → usually **not indicated** (unless symptomatic)

Mobitz Type I (Wenckebach) Second Degree AV Block

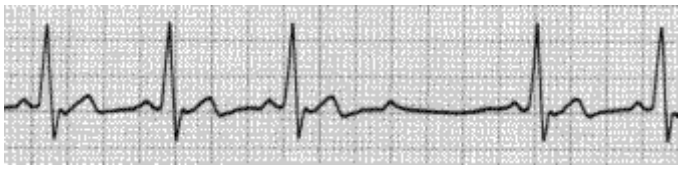
- Etiology: *see 1st degree AV block*
- Usually **asymptomatic**
- EKG (*see image below*) →
    1. **Progressive PR** interval **prolongation** followed by
    2. **Non-conducted P** wave (dropped QRS complex)
    3. **PR** interval **before** the non-conducted P wave **longer** than **PR** interval **after** the non-conducted P wave
    4. **RR** interval **encompassing** the non-conducted P wave **shorter** than **2 RR** intervals **preceding** the non-conducted P wave



- Management:
    1. **Asymptomatic** → treatment **not indicated**
    2. **Acutely** symptomatic → **atropine**
    3. **Acutely** symptomatic **despite** atropine → **temporary pacemaker**
    4. **Symptomatic** (chronic) → permanent **pacemaker** (definite therapy)

Mobitz Type II Second Degree AV Block

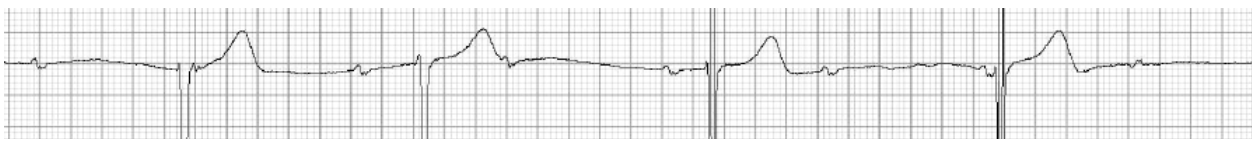
- Etiology (usually structural):
    1. **Anterior MI**
    2. **Age**-related **fibrosis**
    3. **Infiltrative**/inflammatory disorders
    4. **Drug**-induced (e.g. **digitalis**)
- Clinical features → asymptomatic to **hemodynamic instability**
- ↑↑ **risk** of **progression** to **3rd degree** AV block
- EKG (*see image below*) →
    1. **Sudden failure** of **P** wave **conduction**
    2. **No** progressive PR interval prolongation
    3. **PR** interval of **fixed duration** (normal or prolonged)



- Management:
    1. **Symptomatic**/asymptomatic → permanent **pacemaker**
    2. **Acutely** symptomatic → **temporary** (transvenous or transcutaneous) **pacemaker** (consider **isoproterenol** or **epinephrine** if pacing not immediately available)

Third Degree (Complete) AV Block

- Etiology:
    1. **Age**-related **fibrosis** (most common)
    2. **Scarring** from **previous MI**
    3. **Infiltrative**/inflammatory disorders
    4. **Drug**-induced (e.g. digitalis)
    5. **Seronegative spondyloarthropathies** (e.g. ankylosing spondylitis)
- Clinical features → asymptomatic to **hemodynamic instability;** manifestations of **AV dissociation**:
    1. "**Cannon**" **a waves** (giant a waves)
    2. **Variable intensity** of **S_1** sound
    3. **Intermittent** S_3 and/or S_4
- Frequent **Adams-Stoke** attacks → **syncope** secondary to **transient asystole**
- EKG (*see image below*) →
    1. **No relationship** between **P** waves and **QRS** complexes
    2. **Faster** atrial rhythm
    3. **Junctional** escape rhythm → **narrow QRS**; rate **40-60 bpm**
    4. **Ventricular** escape rhythm → **wide QRS**; rate < **40 bpm**



- Management → Permanent **pacemaker** (*see Mobitz type II AV block*)

Comparison between Mobitz type I and Mobitz type II AV block

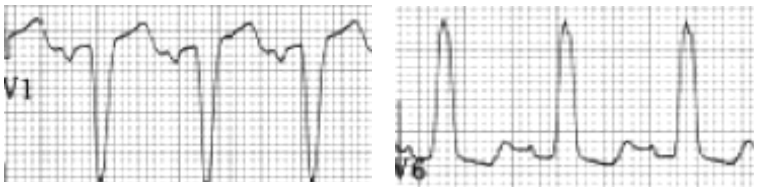
	Mobitz Type I	**Mobitz Type II**
Location of Block	**AV Node**	**His-Purkinje** System
Associated MI	**Inferior**/Right Ventricle	**Anterior**
Associated with ↑ vagal tone	+	-
QRS complex	Usually **narrow**	Usually **wide** (infra-Hisian)
Progression to 3rd degree AV Block	± (escape rhythm usually **junctional**)	+ (escape rhythm usually **ventricular**)
Adams-Stoke syndrome	-	+
Effect of Atropine	↓ **PR** interval (↑ AV **conduction**)	Usually **none**

Bundle Branch Block

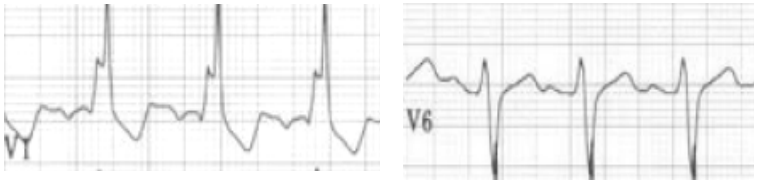
APPROVED
By Zurab Azmaiparashvili at 2:43 am, Jan 24, 2009

	Left Bundle Branch Block (**LBBB**)	Right Bundle Branch Block (**RBBB**)
Etiology	**CAD**/acute MI **Hypertension** **Aortic valve** disease **Cardiomyopathies**	Frequently seen in **normal individuals** Consider **PE** if **acute onset** **Surgical repair** of **VSD** **Congenital** (e.g. associated with ASD or Ebstein anomaly)
Physical Exam	**Paradoxically split S_2**	**Widely split S_2**
EKG	QRS > 0.12 s **QS** pattern in **V_1-V_2** **Notched, tall R** waves with **inverted T** waves in **V_6, I**	QRS > 0.12 s **rSR** pattern with **inverted T** waves in **V_1-V_2** **Wide, deep S** waves in **V_6**, **I**
EKG Mnemonic	**WiLLiaM**	**MaRRoW**

- LBBB:



- RBBB:

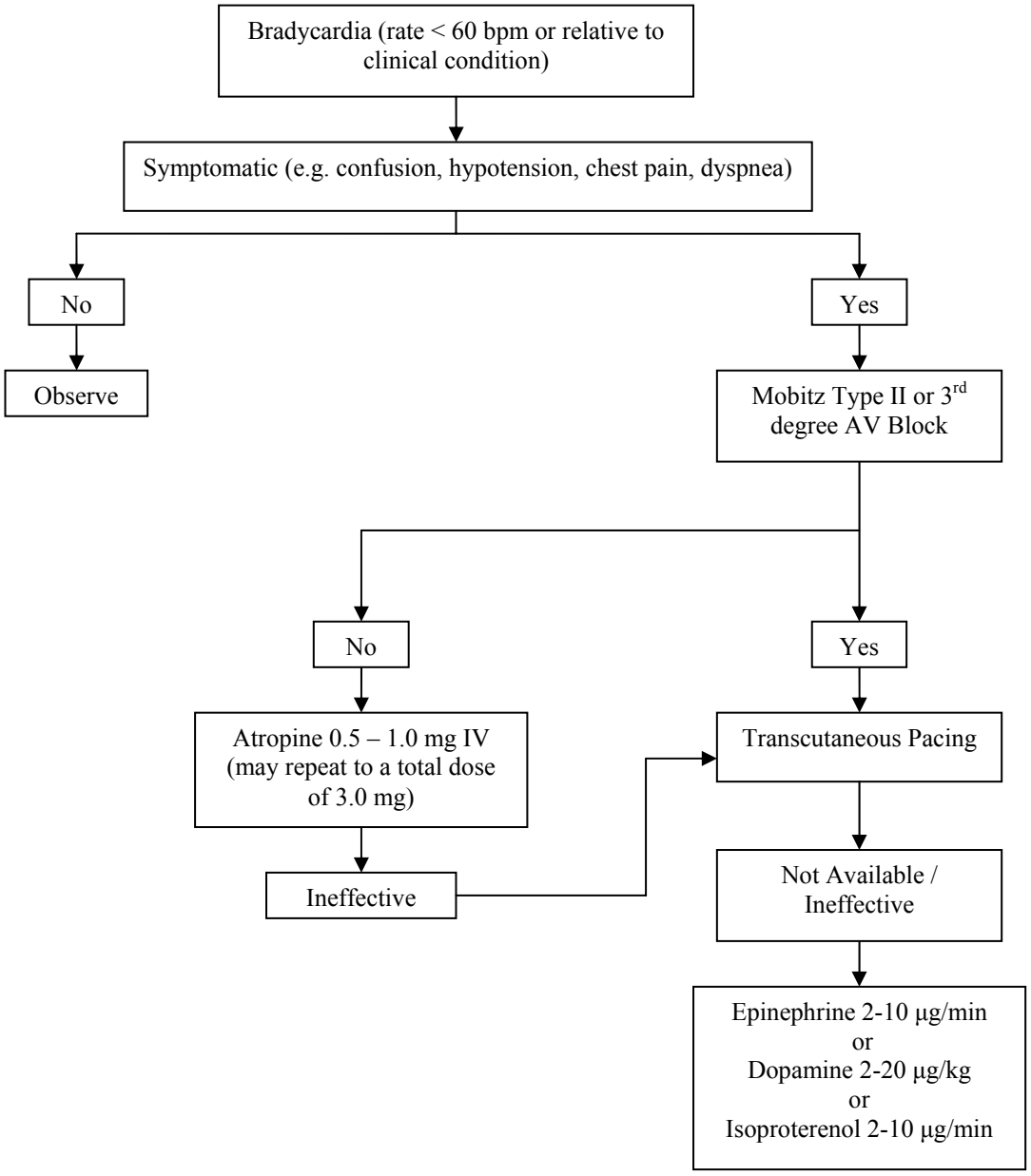


Indications for Permanent Pacing

APPROVED
By Zurab Azmaiparashvili at 2:44 am, Jan 24, 2009

- **Carotid sinus** hypersensitivity
- **Symptomatic bradycardia** secondary to **SA** node **dysfunction**
- **Symptomatic bradycardia** secondary to **AV block**
- **Postoperative** AV block
- **Asymptomatic 3rd degree** AV block (especially if rate < 40 bpm)
- **Asymptomatic Mobitz type II** 2nd degree AV block (especially if infra-Hisian)
- **Asystole > 3 sec**

* **Algorithm** – Bradycardia



Tachyarrhythmias

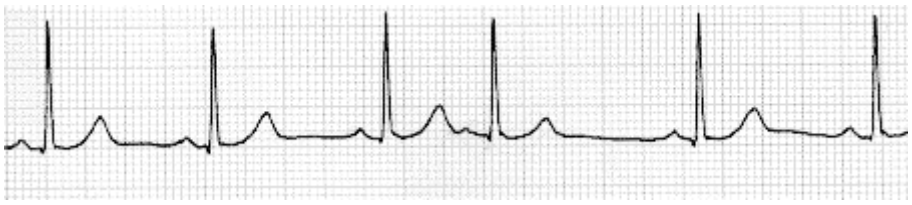
APPROVED
By Zurab Azmaiparashvili at 2:44 am, Jan 24, 2009

Sinus Tachycardia

- **Sinus rhythm** with **rate > 100 bpm** (usually < 200 bpm)
- Etiology:
    1. ↑ **temperature**
    2. **Volume depletion**/hypotension
    3. **Anxiety**/Pain
    4. Exercise
    5. **Hyperthyroidism**
    6. **Anemia**
    7. Congestive heart failure
    8. Pulmonary embolism
    9. **Drug**-induced → caffeine, ethanol, atropine, sympathomimetics
    10. **Withdrawal** from **β-blockers**
- Clinical features → **asymptomatic** to **palpitations** ± manifestations of **hemodynamic instability** (very rare; only in patients with underlying disease)
- EKG → **normal P** waves **preceding** each **QRS** complex; **rate > 100 bpm**
- Management:
    1. **Correction** of any **underlying** abnormality
    2. Overtly **symptomatic** and/or patient with **CAD** → consider **β-blockers** and/or Ca-channel antagonists

Premature Atrial Contractions (PACs)

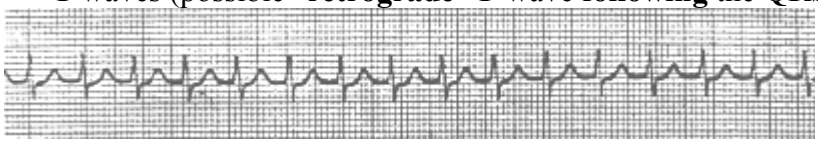
- > **60%** of **adults**
- Common **triggers** → **caffeine, alcohol** and/or **nicotine**
- May be associated with **CAD, COPD** or **electrolyte** disturbances
- Clinical features → **asymptomatic** to **palpitations**
- EKG →
    1. **Early P** wave with a **different morphology**
    2. **Normal QRS**
    3. **PAC** followed by a **less than fully compensatory pause**



- May degenerate into **atrial fibrillation** and/or **flutter** (especially in patients with CAD)
- Management → usually **not indicated** (except for **healthier lifestyle**); consider **β-blockers** in patients with CAD

Paroxysmal Supraventricular Tachycardia (PSVT)

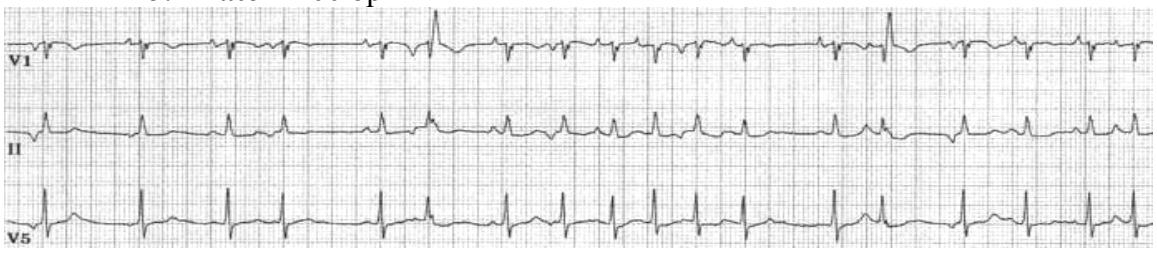
- Characterized by:
  1. **Abrupt onset** and **termination**
  2. **Regular** rhythm; **rate ~ 150-250 bpm**
  3. **Narrow QRS** complexes
- Usually **initiated** by a **PAC**
- Secondary to **reentrant circuit within** the **AV node** (~80% of cases) → **AV nodal reentrant tachycardia** (AVNRT)
- Frequently found in **otherwise healthy** individuals
- Clinical features → asymptomatic to **palpitations;** manifestations of **hemodynamic instability**
- EKG → **no discernible P** waves ("buried in the QRS") with **normal-**appearing **QRS** and **T** waves (possible "**retrograde" P** wave **following** the **QRS** complex with **AVNRT**)



- Management:
  1. **Best initial** step → **vagal maneuvers** (e.g. carotid massage, Valsalva maneuver, head immersion in cold water)
  2. **No response** to **vagal** stimulation → **IV adenosine** (drug of choice); **repeat 2x** at a **higher dose** if unsuccessful
  3. **No response** to **adenosine** → consider IV **verapamil, β-blockers** or **digoxin**
  4. **Hemodynamic instability** ± EF < 40% → DC **cardioversion**
  5. **Definite** (curative) treatment → **radiofrequency**/catheter **ablation**
  6. **Chronic** management if catheter **ablation not** possible → **β-blockers** or Ca-channel antagonists

Multifocal Atrial Tachycardia (MAT)

- Characterized by:
  1. **Irregular** rhythm; **rate ~ 100-200 bpm**
  2. **Narrow QRS** complexes
- Seen in patients with **severe lung disease** and **hypoxia**; possible **electrolyte** and/or **acid-base** abnormalities
- Clinical features → **asymptomatic** to **palpitations**; possible manifestations of hemodynamic instability
- EKG →
  1. ≥ **3 different P** wave morphologies
  2. ≥ **3 different PR** intervals
  3. **Irregular RR** intervals
  4. **Normal-**appearing **QRS**
  5. **Rate > 100** bpm



- Management → **treatment**/correction of any **underlying** disorder ± **Ca-channel** antagonists and/or **β-blockers** (digoxin makes MAT worse)

* Criteria of **MAT** but **rate < 100** bpm → **Wandering Atrial Pacemaker**

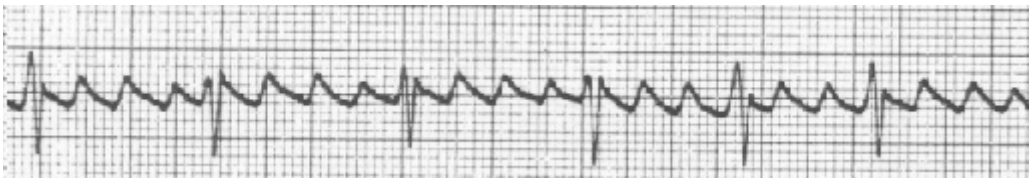
Atrial Flutter (AF)

- **Unstable** rhythm that usually **progresses** to **atrial fibrillation** or **converts** to **sinus rhythm**
- Characterized by:
  1. **Regular** rhythm
  2. **Atrial rate** ~ **300**
  3. **Ventricular rate** ~ **150** (physiologic **2:1 AV block**)
  4. **Narrow QRS** complexes (unless aberrant conduction)
- **Risk factors** → **CAD**, hypertension, **hyperthyroidism**, **mitral valve** disease, pericarditis, cardiomyopathies, **COPD**, **PE**, **alcohol** abuse

* AF with **higher degrees** of **AV block** (e.g. 3:1, 4:1) → **drug-**induced and/or **intrinsic AV** node **dysfunction**

- Clinical features → **asymptomatic** to **palpitations** ± manifestations of **hemodynamic instability**
- EKG → **"sawtooth"** pattern of **atrial activity** (so called "**flutter" waves**) best seen in **inferior leads** (at 2:1 AV block one of the "flutter" waves is buried in the QRS complex making it difficult to differentiate AF from PSVT)

* Maneuvers that ↓ **AV conduction** (e.g. Valsalva, carotid massage) make the **"flutter**" waves more **apparent** → helpful in establishing the diagnosis



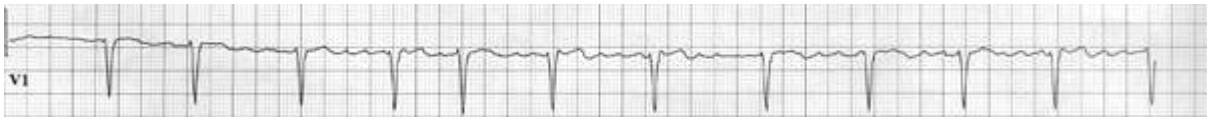
- Management *(see atrial fibrillation)*:
  1. Hemodynamic **instability** → synchronized **cardioversion** (start at 50-100 J) followed by anticoagulation
  2. Hemodynamically **stable** → **rate control** + anticoagulation**:**
     - **β-blockers** (especially in patients with **CAD**)
     - **Ca-channel** antagonists (**avoid** in patients with **CHF**)
     - **Digoxin** (drug of choice in patients with **mitral valve** disease and/or significant **CHF**)
  3. **Definite** therapy → **radiofrequency**/catheter **ablation**

Atrial Fibrillation (Afib)

- **Most common** arrhythmia, especially in the **elderly**
- Characterized by **irregularly irregular** rhythm
- Risk factors → **CAD, hypertension, COPD, PE, mitral valve** disease, cardiomyopathies, pericarditis, atrial **myxomas**, **alcohol** abuse ("holiday heart"), **sepsis**

* **Lone Atrial Fibrillation** → Afib **without** structural heart disease

- Clinical features → asymptomatic to **palpitations,** fatigue, **exacerbation** of **CHF** ± manifestations of **hemodynamic instability** (e.g. shortness of breath, hypotension, chest pain, confusion, syncope); possible manifestations of **systemic embolism** (e.g. ischemic stroke)
- Physical exam → **irregularly irregular,** frequently **rapid pulse** + **absence** of **"a" waves**
- EKG →
    1. **No discernible P waves**
    2. **Wavy**, undulating **baseline** ("fibrillatory" or "F" waves)
    3. **Irregularly irregular RR** intervals
    4. **Narrow QRS** complexes (unless with aberrant conduction)



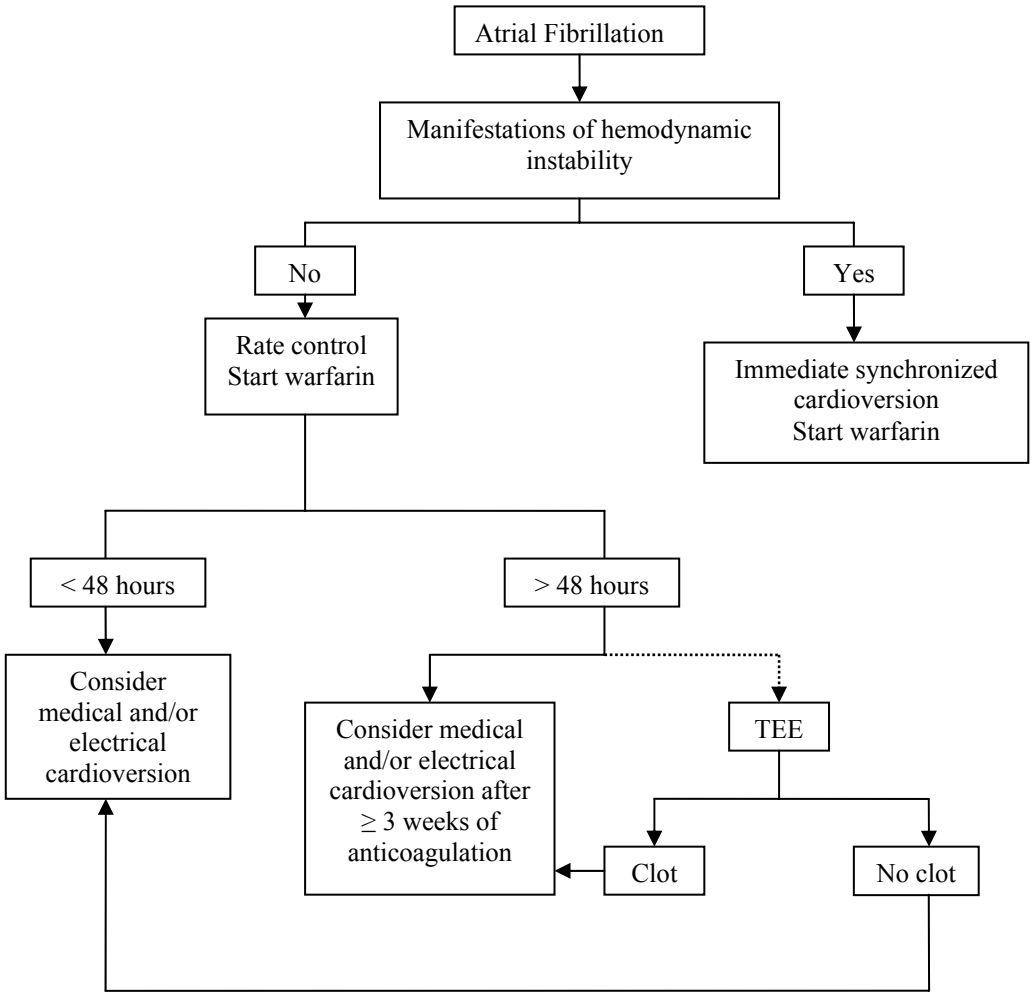
- Management:
    1. Hemodynamic **instability** → synchronized **cardioversion** followed by **warfarin** anticoagulation
    2. Hemodynamically **stable** → **rate control**
        - **β-blockers** (especially in patients with **CAD**, **hyperthyroidism** and/or chronic **CHF**)
        - **Ca-channel** antagonists (**avoid** in patients with **CHF**)
        - **Digoxin** (drug of choice in patients with **mitral valve** disease and/or significant **CHF**)
    3. All patients with **Afib** (or AF) need **warfarin anticoagulation** (except those with **lone Afib** → use **aspirin**) with the **target INR** of **2.0 – 3.0**
    4. **Rhythm control** (medical or electrical cardioversion), if:
        - < **48 hours** since onset
        - > **48 hours** + **TEE** (transesophageal echocardiogram) shows **no clot**
        - ≥ **3 weeks** on **anticoagulation**

* Use **amiodarone** for medical cardioversion **post-MI** or if ↓ **EF**; Use **Class IC** drugs (e.g. flecainide, propafenone) if **normal EF**

* **Refractory Afib** (symptomatic only) → **AV** node **ablation** + permanent **pacemaker**

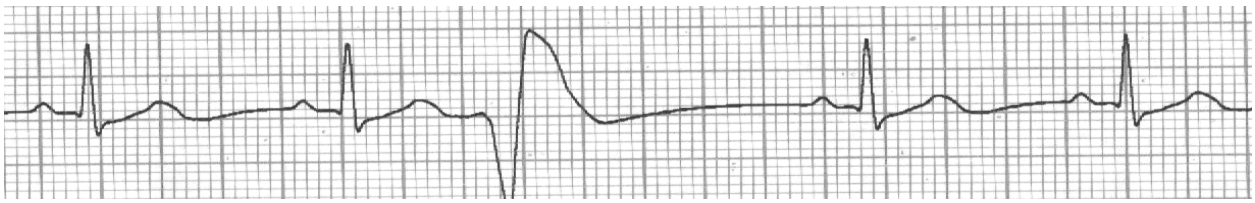
- Algorithm – Atrial Fibrillation

APPROVED
By Zurab Azmaiparashvili at 2:44 am, Jan 24, 2009



Premature Ventricular Contractions (PVCs)

- Usually **benign**; found in ~ 60% of **adult males**
- ↑ **incidence** → **post-MI, CAD**, electrolyte abnormalities, hyperthyroidism
- ↑ **mortality** if:
    1. **Frequent** (>10/hour)
    2. **Complex** (e.g. couplets)
    3. **Polymorphic**
    4. **Early** (PVC occurring during previous T wave)
- **Early PVCs** tend to degenerate into **ventricular tachycardia** and/or **fibrillation**
- **Early PVCs** more common in patients with **long QT syndrome**
- Clinical features → **asymptomatic** to **palpitations**
- EKG →
    1. **Wide** (>0.12s), **bizarre QRS complexes**
    2. **No preceding P** waves
    3. Followed by a **fully compensatory pause**



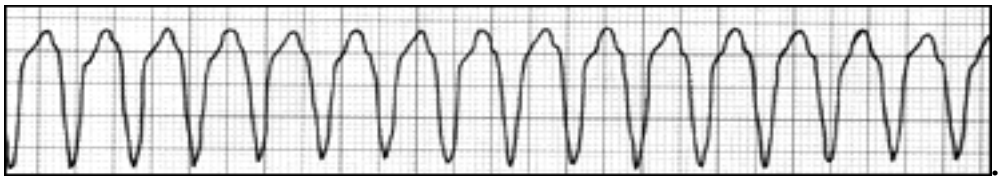
- Management:
    1. **Asymptomatic + no** cardiac disease → treatment **not indicated**
    2. **Symptomatic + no** cardiac disease → **β-blockers**
    3. **Cardiac disease** → **β-blockers**
    4. **Frequent/complex** PVCs → **β-blockers**

* **Bigeminy** → every sinus beat followed by a VPC
* **Trigeminy** → every second sinus beat followed by a VPC
* **Couplets** → two consecutive VPCs
* **Triplets**/ventricular tachycardia → three consecutive VPCs ± rate > 100 bpm.

Ventricular Tachycardia (VT)

APPROVED
By Zurab Azmaiparashvili at 2:44 am, Jan 24, 2009

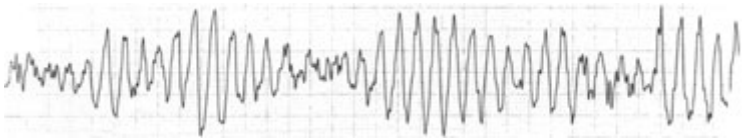
- Definition → **≥ 3 consecutive PVCs** at a **rate > 100 bpm**
- Risk factors → **CAD, MI** (most common), **electrolyte** abnormalities (e.g. hypokalemia, hypomagnesemia), **drug-**induced (e.g. **digitalis** toxicity), **long QT syndrome**, cardiomyopathies, **mitral valve prolapse**
- Significant if **sustained** (> 30 sec) or associated with **ventricular fibrillation**
- Clinical features:
    1. **Non-sustained VT** → usually **asymptomatic**; possible palpitations
    2. **Sustained VT** → manifestations of **hemodynamic instability**
- Physical exam → signs of **AV dissociation** (e.g. "cannon" a waves, variable intensity of S_1 sound)
- EKG:
    1. **Monomorphic VT** → *see PVCs*
    2. **Polymorphic VT** → *see Torsades de Pointes*



- Management:
    1. **Pulseless VT** → *see ventricular fibrillation*
    2. Hemodynamic **instability** → synchronized **cardioversion** (100J → 200J → 300J → 360J)
    3. Hemodynamically **stable** → IV **amiodarone** (drug of choice) or IV **lidocaine** (amiodarone not effective/not available)
    4. **Definite** therapy in **sudden death survivors** → **ICD** placement
    5. Consider **chronic** therapy with **β-blockers**

Torsades de Pointes (Polymorphic VT)

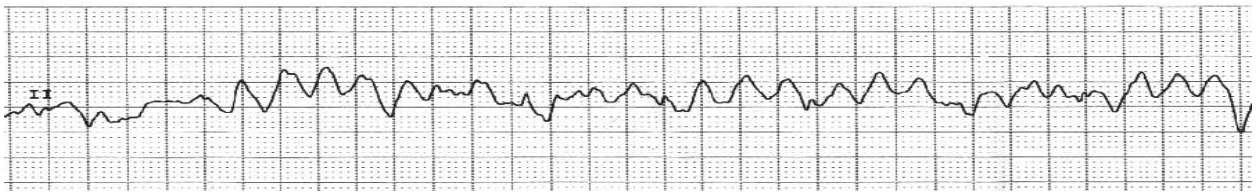
- Risk factors:
    1. **Congenital long QT** syndromes → **Romano-Ward** syndrome (isolated long QT interval), **Jervell** and **Lange-Nielsen** syndrome (long QT + **hearing loss**)
    2. **Drug**-induced **long QT** syndrome → **Class IA** or **III** anti-arrhythmics (e.g. quinidine, procainamide, sotalol), **phenothiazine** antipsychotics, **TCAs**
    3. **Electrolyte** disturbances → **hypomagnesemia**, hypokalemia
    4. **Post-MI** bradycardia
- Clinical features → **recurrent** episodes of dizziness/**syncope**; **sudden auditory stimuli** may precipitate ***torsades de pointes*** in some forms of **congenital long QT** syndrome
- EKG → **waxing** and **waning amplitude** of the **QRS** complexes ("twisting of the points")



- Management:
    1. Hemodynamic **instability** → **cardioversion**
    2. **Treatment**/correction of any **underlying** abnormality
    3. Consider → **magnesium** supplementation, **isoproterenol** infusion and/or **overdrive pacing**
    4. **Congenital long QT** syndrome → **β-blockers** (even if asymptomatic)

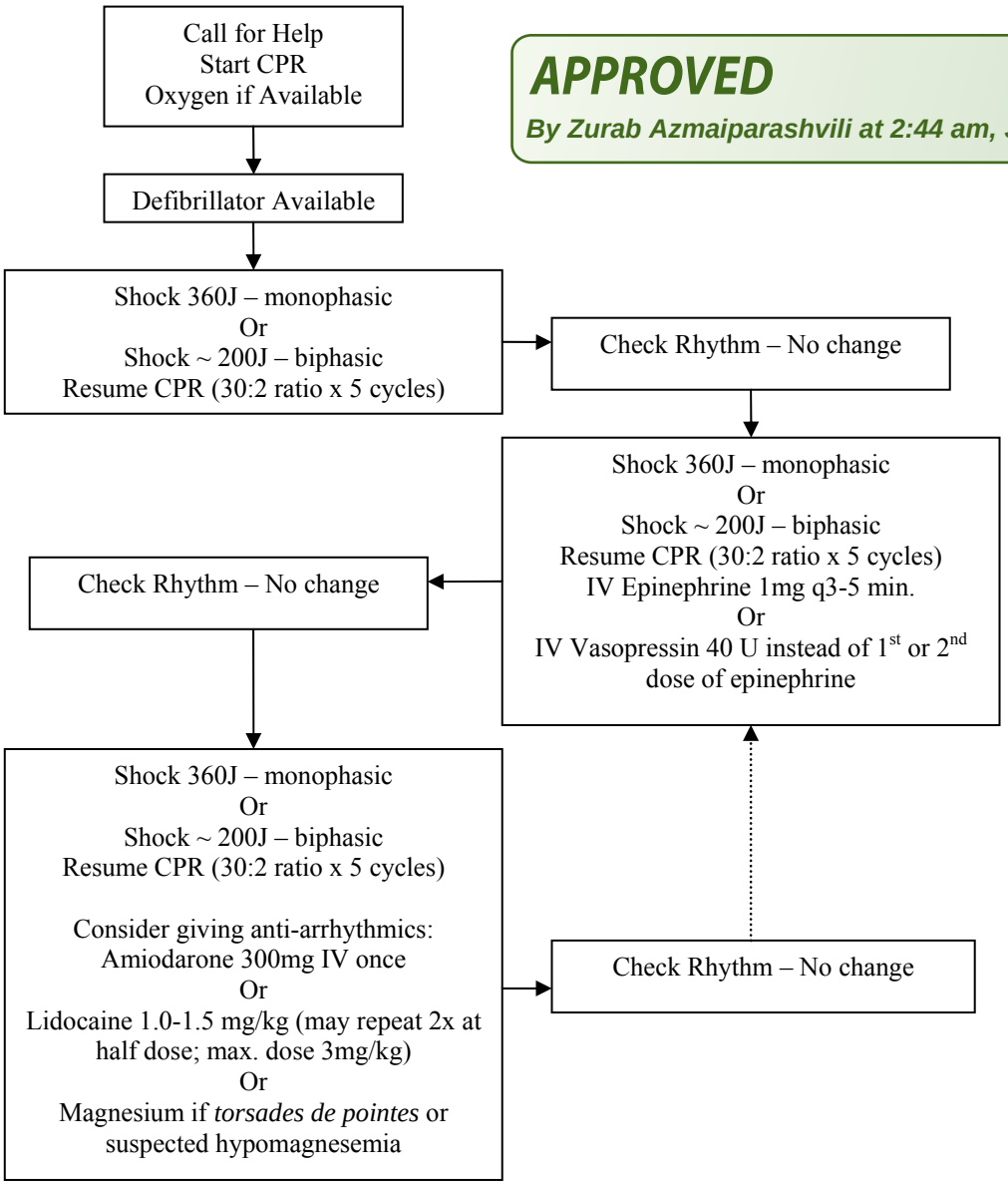
Ventricular Fibrillation (VF)

- **Most common** cause of **sudden death**
- Risk factors → **CAD, MI, electrolyte** disturbances, **hypothermia**, **drug** toxicity
- Clinical features → **syncope + no pulses** (virtually dead)
- EKG → **oscillation from baseline** with no discernible waves



- Management → immediate **asynchronized cardioversion** (*see algorithm)*

- Algorithm – VF/Pulseless VT



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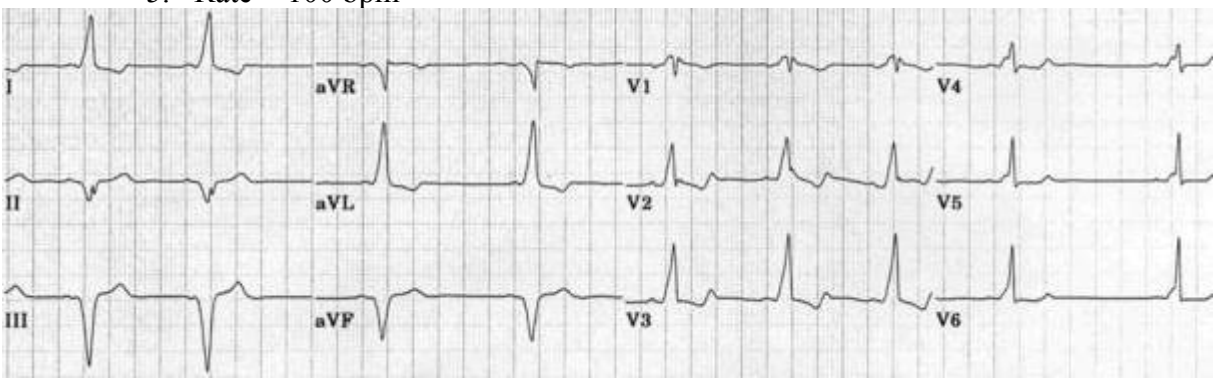
By Zurab Azmaiparashvili at 2:44 am, Jan 24, 2009

Wolff-Parkinson-White Syndrome (WPW syndrome)

- Secondary to the presence of an **accessory pathway** (Bundle of Kent) **connecting** the **atria** and the **ventricles** (bypassing the normal conduction system)
- Clinical features → **asymptomatic** to **palpitations** (due to tachycardia)
- Complications → **PSVT** and **Afib; ↑ risk** of **sudden death** in symptomatic patients
- EKG →
    1. **Short PR** interval (<0.12 s)
    2. **Delta wave**
    3. **Wide QRS** complex
    4. **Normal PJ** interval
    5. Rate > 100 bpm



* Note → in **WPW** syndrome **EKG** becomes **uninterpretable** for the presence of **ischemia/MI** and/or ventricular **hypertrophy**

- Management → **radiofrequency**/catheter **ablation** of the accessory pathway (only after **electrophysiologic** demonstration of **cause-effect relationship**):
    1. **Afib** + hemodynamic **instability** → synchronized **cardioversion**
    2. **Afib** + hemodynamically **stable** → **procainamide** (**1st choice**) and/or amiodarone
    3. **Avoid drugs**/maneuvers that ↓ **AV conduction** (e.g. digoxin, β-blockers, Ca-channel antagonists, adenosine, carotid massage) → may **precipitate** hemodynamic **instability** secondary to ↑ **ventricular rate**/VF

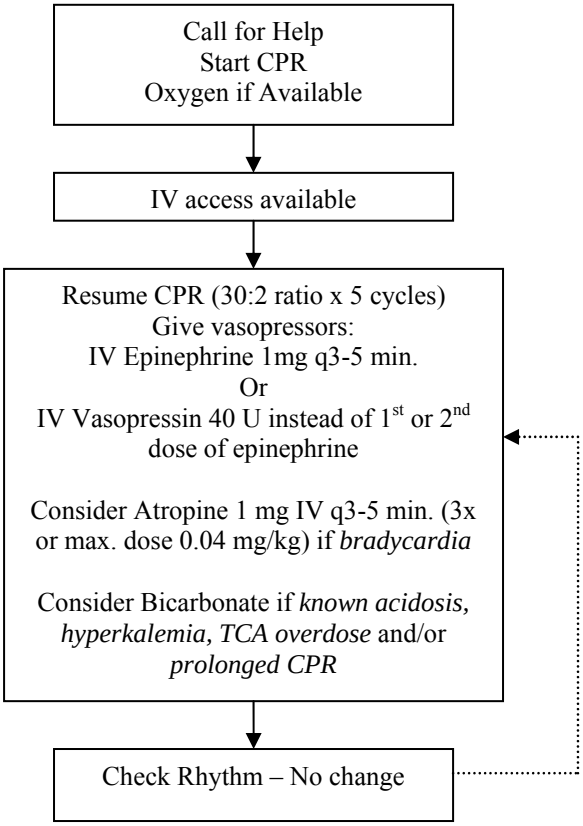
Lown-Ganong-Levine Syndrome

- **Aberrant pathway** that **joins** the **His-Purkinje** system
- **Pre-excitation** syndrome (similar to WPW)
- EKG characteristics →
    1. **Short PR** interval
    2. **No** delta wave
    3. **Narrow QRS** complex
    4. **Short PJ** interval

Pulseless Electrical Activity (PEA)

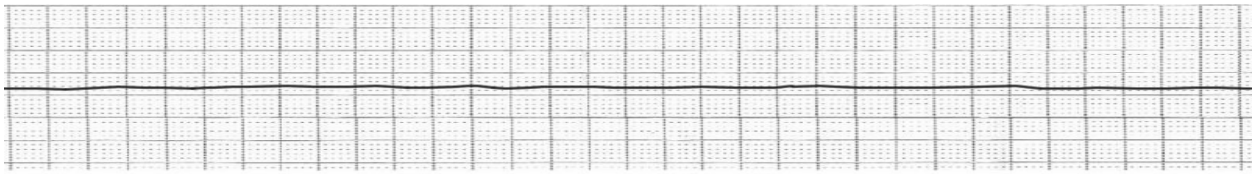
- **Synonyms** → electromechanical dissociation
- **Loss** of peripheral **pulse** in the **presence** of **significant EKG activity**
- Etiology (should **guide therapy** when known):
    1. **Tension pneumothorax**
    2. Cardiac **tamponade**
    3. Massive **PE**
    4. Extensive **MI**
    5. **6Hs** → *hypoxia, hypovolemia, hyper-/hypokalemia, hypothermia, ↑ hydrogen ions*
    6. **Drug-**induced (e.g. TCA toxicity)
- **Only EKG** can **differentiate** between **PEA**, ventricular **asystole** and **VF**/Pulseless VT
- Management → **etiology**-dependent; if **unknown** → *see algorithm*

- Algorithm – PEA/Asystole



Ventricular Asystole *(see algorithm)*

- Consider **transcutaneous pacing** if *very slow bradycardia*
- EKG →.nearly **flat line**



Anti-Arrhythmic Medications, Classification

- **Class I** – Blockade of **Na Channels**:
    1. **Class IA** - ↑ AP duration (*quinidine, procainamide, disopyramide*)
    2. **Class IB** - ↓ AP duration (*lidocaine, phenytoin, mexiletine, tocainide*)
    3. **Class IC** – no change in AP duration (*flecainide, encainide, propafenone*)
- **Class II** – Blockade of **β-**adrenergic **receptors** *(see anti-anginal medications)*
- **Class III –** Blockade of **K Channels** (*amiodarone, sotalol, bretylium, dofetilide*)
- **Class IV –** Blockade of **Ca Channels** *(see anti-anginal medications)*

Anti-Arrhythmic Medications, Selected Examples

- **Quinidine →**
    1. **Class IA** medication
    2. Side effects: **GI distress**, immune-mediated **thrombocytopenia**, **cinchonism** (dizziness, **tinnitus**, hearing loss), **hypotension,** hypoglycemia, ↑ **QT** interval ± *torsades de pointes* and/or **syncope**
    3. Important drug interactions: ↑ **digoxin toxicity**

- **Procainamide →**
    1. **Class IA** medication
    2. Side effects: ↑ **ANAs** ± **lupus**-like syndrome, **GI distress,** ↑ **QT** interval ± *torsades de pointes* and/or **syncope**, **hypersensitivity** reactions (e.g. fever, rash, **agranulocytosis**), **hallucinations**

- **Disopyramide →**
    1. **Class IA** medication
    2. Side effects: ↑↑ **anti-cholinergic** activity (e.g. blurred vision, constipation, dry mouth, urinary retention), **CHF**, severe **hypotension**

- **Lidocaine →**
    1. **Class IB** medication
    2. Side effects: **CNS** toxicity (e.g. confusion, **tremor, seizures,** drowsiness, **coma**) ± **cardiovascular** toxicity (e.g. hypotension, **bradycardia, asystole**)

- **Amiodarone →**
    1. **Class III** medication (also Class **IA, II** and **IV**)
    2. Side effects: **photosensitivity**, blue-gray discoloration of the skin, **corneal deposits** ± blue halos, **hypo-/hyperthyroidism**, **pulmonary fibrosis** (interstitial lung disease), ↑ **LFTs** ± hepatic necrosis, **neuropathy** (e.g. paresthesias, ataxia, tremor), ↑ **QT** interval (**no** *torsades de pointes*)
    3. Important drug interactions: ↑ **digoxin** and **warfarin toxicity**

- **Adenosine →**
    1. **Unclassified**
    2. Side effects: **flushing,** chest **tightness**, **dyspnea**, **diaphoresis**, apprehension, metallic taste, paresthesias, possible asystole

Infections of the Heart

Infective Endocarditis (IE)

- Infection of the **endocardium** characterized by formation of **friable vegetations** (fibrin + platelets + bacteria) ± **valvular destruction**
- **Most commonly** affected valve → **mitral** (followed by aortic valve; **Left** > Right)
- **Most commonly** affected valve in **IV drug users** → **tricuspid** (followed by pulmonary valve; **Right** > Left)
- Risk factors → **IV drug** use, **indwelling catheters** (e.g. pulmonary artery catheters, central venous catheters, hyper-alimentation catheters)

High Risk	***Intermediate Risk***	***Low Risk (No Risk)***
Prosthetic heart valves **Previous IE** **Surgically** constructed systemic and/or pulmonary **shunts/conduits** Complex **cyanotic congenital** heart disease	**Acquired** valvular dysfunction (e.g. **rheumatic fever**) **Most** cases of **congenital** heart disease **HCM** **MVP** with **regurgitation** and/or **thick** leaflets	Isolated **secundum ASD** **Surgical repair** of ASD, VSD or PDA (> 6 months) **CABG** **MVP without** regurgitation **Innocent** heart **murmur** **Rheumatic fever without** valvular dysfunction Cardiac **pacemakers** **ICD**

- **Most common** etiology:
  1. Previously **damaged, native** valves → Streptococcus ***viridans***
  2. **Prosthetic** valves **< 2 months** → Staphylococcus ***epidermidis***
  3. **Prosthetic** valves > **2 months** → Streptococcus ***viridans***
  4. **IV drug** users → Staphylococcus ***aureus***

Acute bacterial endocarditis	***Subacute bacterial endocarditis***
Previously **normal valves**	Previously **damaged** and/or **prosthetic** valves
S. aureus	**S. viridans**
IV drug users	
Large vegetations	**Small** vegetations
Rapid onset and progression	**Less fulminant** course
Right > Left (**tricuspid** valve)	**Left** > Right (**mitral** valve)
Pulmonary septic **emboli**	**Systemic** septic **emboli**

* IE secondary to **S. *bovis*** → order **colonoscopy** (to rule out associated colon cancer)

* **Culture-negative** IE and/or **FUO** (fever of unknown origin) → consider **HACEK** organisms (**H**aemophilus, **A**ctinobacillus, **C**ardiobacterium, **E**ikenella and **K**ingella)

* **Fungal** endocarditis (e.g. **Candida** albicans) → **IV drug** users, indwelling **catheters**

* **Gram-negative** (e.g. Pseudomonal) endocarditis → **IV drug** users

- Clinical features:
    1. **Constitutional** symptoms → **fever/chills**, fatigue, malaise, anorexia, weight loss
    2. **New** and/or **changing** heart **murmur** (**regurgitation** murmur)
    3. Manifestations of **CHF** (e.g. dyspnea on exertion, orthopnea)
    4. **Peripheral manifestations** of IE (secondary to **vasculitis**):
        - **Petechiae** (conjunctive and/or oral mucosa)
        - **Splinter hemorrhages** (liner hemorrhages affecting the proximal nail bed)
        - **Osler's nodes** (small painful nodules on the finger pads)
        - **Roth spots** (white/pale retinal lesions surrounded by hemorrhage)
    5. **Peripheral manifestations** of IE (secondary to **embolism**):
        - **Janeway lesions** (red, painless lesions on the palms and soles)
    6. Manifestations of **systemic** septic **embolism** (with left-sided lesions) → **CNS** (e.g. stroke), **renal** (e.g. hematuria, flank pain), **splenic** (e.g. LUQ pain), **coronary** (e.g. acute MI), **mesenteric** (e.g. abdominal pain, lower GI bleed)
    7. Manifestations of **pulmonary** septic **embolism** (with right-sided lesions) → **chest pain, cough** ± hemoptysis
    8. Possible **splenomegaly**

*** IV drug abuser** + acute onset of **fever/chills** ± heart **murmur** = **IE**

- Complications (other than listed above) → **aneurysm** formation (e.g. mycotic aneurysms), immune-mediated **glomerulonephritis**, **abscess** formation with associated **conduction abnormalities**
- **Most common** cause of **mortality** → **CHF**
- Lab findings → **anemia**, **leukocytosis**, ↑ **ESR**, ± RF positivity, ± active renal sediment
- Chest X-ray → multiple **nodules ± cavitation** (in cases of pulmonary septic embolism)
- **Best initial** diagnostic test → trans-thoracic **echocardiography** (**TTE**)
- **Most accurate** diagnostic test(s) → blood **culture** (≥ 3 samples in 24 hours) and/or trans-esophageal **echocardiography** (**TEE**)
- **Duke** diagnostic **criteria**:

Major Criteria	**Bacteremia** with typical organisms (**≥ 2 positive** blood cultures) **Echocardiographic** evidence of **IE** (e.g. vegetations, new regurgitation, abscess)
Minor Criteria	**Risk factors** for IE **Temperature** > **38 C** **Vascular** phenomena (embolism, mycotic aneurysms, Janeway lesions, hemorrhages) **Immunologic** phenomena (glomerulonephritis, Osler nodes, Roth spots, ↑ RF) **Echocardiographic** findings consistent with IE but **do not meet** a **major criterion** **Microbiologic** evidence **not meeting** a **major criterion** (e.g. one positive blood culture)
Diagnosis	**2 major** criteria *or* **1 major** + **3 minor** criteria *or* **5 minor** criteria

- **Empiric** antibiotic **therapy** (until culture results become available) → **vancomycin** + **gentamicin**
- **Pathogen-specific** antibiotic **therapy**:

Pathogen/Situation	***Regimen***	***Duration***
Penicillin-Sensitive Streptococci + native valve endocarditis	Penicillin G or Ceftriaxone Or Penicillin G or Ceftriaxone + Gentamicin	4 weeks 2 weeks
As above + penicillin-allergy	Vancomycin	4 weeks
Penicillin-Resistant Streptococci + native valve endocarditis	Penicillin G + Gentamicin (*for 2 weeks*)	4 weeks
As above + penicillin-allergy	Vancomycin	4 weeks
Enterococcal endocarditis	Penicillin G or Ampicillin + Gentamicin	4-6 weeks
As above + penicillin allergy	Vancomycin + Gentamicin	4-6 weeks
Methicillin-Sensitive Staphylococcus + native valve endocarditis	Oxacillin (or nafcillin, etc.) or Cefazolin + Gentamicin (*for 3-5 days*)	4-6 weeks
As above + penicillin-allergy	Vancomycin + Gentamicin (*for 3-5 days*)	4-6 weeks
Methicillin-Resistant Staphylococcus + native valve endocarditis	Vancomycin + Gentamicin (*for 3-5 days*)	4-6 weeks
Methicillin-Sensitive Staphylococcus + prosthetic valve endocarditis	Oxacillin (or nafcillin, etc.) + Gentamicin (*for 2 weeks*)+ Rifampin	≥ 6 weeks
Methicillin-Resistant Staphylococcus + prosthetic valve endocarditis	Vancomycin + Gentamicin (*for 2 weeks*) + Rifampin	≥ 6 weeks
HACEK endocarditis	Ceftriaxone Or Ampicillin + Gentamicin	4 weeks 4 weeks

- **Indications** for **surgical intervention**:
  1. **Acute AR** with **early S1**
  2. **Acute AR** or **MR** with **CHF**, **NYHA III–IV**
  3. **Refractory CHF**
  4. **Fungal** endocarditis
  5. **Recurrent** systemic **emboli**
  6. **Prosthetic** valve **dysfunction**
  7. **Early** (< 2 months) **prosthetic** valve **endocarditis**
  8. Evidence of **ring abscess** formation
  9. Evidence of **aneurysm** formation
  10. **Persistent infection** after **7-10 days** of appropriate antibiotic therapy
  11. **Recurrent** infection
  12. **Large** (>10 mm), mobile **vegetations**
  13. **Gram-negative** (or antibiotic-resistant) endocarditis + **valve dysfunction**

- IE **prophylaxis** (only if both present):
    1. **High-risk** cardiac conditions *(see above)*
    2. **Procedures** associated with transient **bacteremia:**
        - **Dental** procedures that involve **manipulation** of the **gingival tissue** and/or **perforation** of the **oral mucosa** (**does not include** anesthetic injections, placement/adjustment of orthodontic appliances, shedding of deciduous teeth and trauma to the lips and/or oral mucosa)
        - **Invasive Respiratory** tract procedures, such as **tonsillectomy** and/or **adenoidectomy** (**does not include** bronchoscopy, rigid or flexible)
        - **Manipulations** involving the **infected skin** and/or **musculoskeletal** tissue

* IE prophylaxis **no longer recommended** for **GI** and/or **GU** procedures

* IE prophylaxis **no longer recommended** for **intermediate risk** cardiac conditions

- **Antibiotic regimens** for IE **prophylaxis**:

Able *to take* ***oral*** *medications*	**Amoxicillin**
Unable *to take* ***oral*** *medications*	**Ampicillin** Or Cephalosporins (e.g. cefazolin, ceftriaxone)
Penicillin allergy + ***Able*** *to take* ***oral*** *medications*	**Clindamycin** Or Macrolides (e.g. azithromycin, clarithromycin) Or Cephalosporins (e.g. cephalexin)
Penicillin allergy + ***Unable*** *to take* ***oral*** *medications*	**Clindamycin** Or Cephalosporins (e.g. cefazolin, ceftriaxone)

Acute Rheumatic Fever (ARF)

- Immunologic **complication** of **Streptococcal pharyngitis** (note: ARF does not follow impetigo and/or other streptococcal skin infections)
- Children of **school age** (5-15 years of age)
- Incubation period → **2-3 weeks**
- Clinical features:
    1. **Migratory Polyarthritis:**
        - **Most common** manifestation
        - Usually effects the **ankles, knees, elbows** and **wrists**
        - Usually **resolves within 2 weeks**
        - **No residual** deformities
    2. **Sydenham's Chorea** (St. Vitus' dance)
    3. **Carditis:**
        - **Murmurs** of **mitral** and/or **aortic regurgitation** are the most common cardiac manifestations (secondary to **endocarditis**)
        - **Pericarditis** *(see diseases of the pericardium)*

- **Myocarditis** (*see diseases of the myocardium*)
- **CHF**
- **Carditis** is the **most serious manifestation** of ARF

4. **Subcutaneous nodules:**
   - Usually occur on the **extensor surfaces** of **large joints**
   - Usually **painless** and **transient**
5. **Erythema marginatum:**
   - **Flat, non-scarring, painless** rash
   - **Transitory**, sometimes lasting less than 1 day
6. Other manifestations include:
   - abdominal pain and anorexia
   - malaise, lethargy, fatigue
   - fever

- Diagnosis (**Jones Criteria**):
  1. **2 major criteria plus evidence of streptococcal infection** or
  2. **1 major and 2 minor criteria plus evidence of streptococcal infection**
  3. Evidence of Infection:
     - **Positive** throat **culture**
     - **Rising** Antistreptolysin-O (**ASO**) **titer**

Major Criteria	***Minor Criteria***
Carditis	Arthralgias
Polyarthritis	Fever
Chorea	Elevated ESR
Erythema marginatum	Prolonged PR interval
Subcutaneous nodules	

- Management →
  1. **Aspirin** and/or other **NSAIDs** for arthritis and/or carditis
  2. **1 dose** of IM **benzathrine Penicillin G** (acceptable **alternative → 10-day** course of **oral Penicillin** V)
  3. Penicillin **allergy → Erythromycin** or other macrolides
  4. **Severe** and/or **refractory carditis → Steroids**
- Secondary prevention → **benzathrine Penicillin G q3-4 weeks** for ≥ **10 years** and/or **until age 40** (alternatives → daily oral penicillin or erythromycin in penicillin-allergic patients)

Tumors of the Heart

Cardiac Myxomas

- **Most common primary** cardiac tumor
- **Females** > Males
- Almost always **benign**
- Usually **single** (~95% of cases)
- Most common location → **left atrium** (~85% of cases), followed by **right atrium**
- Gross appearance → **gelatinous, friable, pedunculated** mass attached to the **interatrial septum**
- Clinical features:
    1. **Obstruction** to **blood flow** → symptoms/signs of **mitral stenosis** (e.g. dyspnea, orthopnea, hemoptysis, pulmonary edema, dizziness/syncope, diastolic rumble, loud S_1) that **changes with body position** ± **early diastolic** sound – "**tumor plop**"
    2. **Systemic embolization** → **CNS** and **extremities** being **most common**
    3. **Systemic** manifestations (secondary to ↑ IL-6 production) → fever, weight loss, fatigue, arthralgias, Raynaud phenomenon
- Lab findings → ↑ ESR, ↑ WBC count, ↓ Hb, hypergammaglobulinemia
- **Best initial** diagnostic test → trans-thoracic **echocardiography**
- Management → **surgical** removal

* Cardiac **Rhabdomyoma** → **most common primary** cardiac tumor in **infants** and/or **young children**; associated with **tuberous sclerosis; spontaneous regression** common

* Cardiac **metastases** → **most common** cardiac neoplasm (far exceeding primary tumors); **pericardium** most commonly involved; **lymphomas**/leukemias, **breast** and **lung** carcinomas, malignant **melanomas**

Congestive Heart Failure (CHF)

Definition

- **Syndrome** characterized by **inadequate cardiac output** necessary to meet the metabolic demands of the body, **manifested** as end-organ **hypoperfusion** and/or **vascular congestion**

Classification

- **Acute** *(see acute heart failure)* vs. **Chronic**
- **Systolic** vs. **Diastolic** *(see Table 1)*
- **Left-**sided vs. **Right-**sided *(see Table 2)*
- **Low-output** vs. **High-output** *(see Table 3)*
- **Forward** vs. **Backward** *(hypoperfusion vs. congestion)*
- **NYHA** classification *(see Table 4)*
- **ACC/AHA** classification *(see Table 5)*

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* Note → **Most** cases of **CHF** are of **mixed type** (e.g. biventricular, both systolic and diastolic, both forward and backward)

Table 1	
Systolic Heart Failure	***Diastolic Heart Failure***
↓ ventricular **contractility**	↓ **compliance** and/or **impaired relaxation**
↓ **EF**	↔ **EF**
S3 sound	**S4** sound
↑ **heart size**	↔ **heart size**
Etiology → **MI**/ischemic cardiomyopathy, **valvular** heart disease, **DCM**	Etiology → **acute ischemia**, **hypertension**, **HCM, RCM**, **AS**

Table 2	
Left-Sided Heart Failure	***Right-Sided Heart Failure***
Etiology → **IHD**, **valvular** heart disease, **hypertension**, **cardiomyopathies**, etc.	Etiology → **left-**sided **CHF** (most common), **COPD, pulmonary hypertension**, **restrictive** lung disease, massive **PE**
Manifestations of **pulmonary congestion** (e.g. pulmonary edema)	Manifestations of **systemic congestion** (e.g. peripheral edema, hepatic congestion)

Table 3	
Low-Output Heart Failure	***High-Output Heart Failure***
Disorders leading to **ventricular dysfunction** (systolic and/or diastolic)	Severe **anemia**, "wet **beriberi**" (vitamin B1/ thiamine deficiency), **AVM**s, **AV fistulas**, **hyperthyroidism**, **Paget's** disease of bone

Table 4	
NYHA** (New-York Heart Association) **Classification of Heart Failure	
NYHA **Class 1**	**No limitation** of physical activity
NYHA **Class 2**	Comfortable at rest + **mild limitation** of physical activity
NYHA **Class 3**	Comfortable at rest + **marked limitation** of physical activity
NYHA **Class 4**	**Uncomfortable at rest +** marked limitation of physical activity

Table 5	
ACC/AHA** (American College of Cardiology/American Heart Association) **Classification of Heart Failure	
Stage A	Patients at **high risk** for developing CHF in the future but **no functional** or **structural** heart disorder
Stage B	**Structural** heart disorder but **no symptoms** of CHF
Stage C	**Structural** heart disorder with **current** and/or **prior symptoms** of CHF
Stage D	**Refractory CHF** despite medical therapy

Compensation/Pathophysiology

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Compensatory Mechanism	***Pros***	***Cons***
Renin-Angiotensin-Aldosterone System Activation	↑ BP ↑ SV & CO (↑ preload)	Volume overload (congestion) ↑ peripheral resistance Cardiac Remodeling
Sympathetic Nervous System Activation	↑ BP ↑ HR ↑ SV & CO (↑ contractility)	↑ peripheral resistance ↑ O_2 demand Cardiac Remodeling
Frank-Starling mechanism	↑ SV & CO (↑ preload)	↑ filling pressure (congestion)
Ventricular **Hypertrophy**	↑ SV & CO (↑ contractility)	↑ O_2 demand

BP – blood pressure, SV – stroke volume, CO – cardiac output, HR – heart rate

Etiology

- **CAD** (most common; ~ 70% of cases)
- **Hypertension**
- **Valvular** Heart Disease
- **Cardiomyopathies**/myocarditis
- **Pericardial** Disease (e.g. constrictive pericarditis)
- Idiopathic

Precipitating Factors

- Usually **reversible**
- **Sudden decompensation** (e.g. 1st episode of CHF and/or clinical deterioration)
- Important to **differentiate** from **disease progression**
- Routine **tests** to order → **Chest X-ray** and **EKG** ± cardiac **enzymes** (to rule out pneumonia, arrhythmias and/or ischemia/infarction)
- Common precipitants include:
    1. **Life-style** changes, e.g. ↑ **salt** intake, ↑ **fluid** intake, excessive **alcohol** consumption (most common)
    2. **Non-compliance** with treatment
    3. Uncontrolled **hypertension**
    4. **Ischemia**/infarction
    5. **Infections**, especially pneumonia
    6. **Arrhythmias**, especially **Afib**
    7. **Anemia**
    8. **Hyperthyroidism**
    9. Other → PE, renal failure, sleep apnea, NSAIDs

Clinical Features

- Symptoms →
    1. **Dyspnea** on exertion, **orthopnea, paroxysmal nocturnal dyspnea,** chronic/**nocturnal cough** ± wheezing, **nocturia**
    2. **Fatigue, weakness**
    3. **RUQ** discomfort/pain ± nausea
- Signs →
    1. ↑ HR
    2. **Bilateral** pulmonary **rales** (especially **basal**), ± **dullness** to percussion, **displaced** apical impulse, **S3** (**most reliable** sign of CHF) and/or **S4** sound
    3. Peripheral **edema**, **JVD**, **hepatomegaly** ± hepatojugular reflux, **ascites**

Diagnosis

- Chest X-ray → pulmonary **congestion** (e.g. **Kerley B** lines, **cephalization** of pulmonary vessels, **interstitial edema**), ± **pleural**/pericardial **effusion**, ± **cardiomegaly**
- EKG → possible **ischemic** changes and/or **Afib** ± ventricular **hypertrophy**
- **Best initial** diagnostic test → **echocardiography**
- **Most accurate** diagnostic test (for **EF estimation**) → **MUGA** scan

* Order **BNP** (brain natriuretic peptide) to **differentiate** between **CHF** (↑ levels) and **dyspnea** of **pulmonary** origin (↔ levels)

Management

*ACC/AHA **Stage A***	**Life-style** modification (e.g. ↓ alcohol intake, smoking cessation) **Correction** of any **underlying** abnormalities (e.g. hypertension, hyperlipidemia, hyperglycemia) ± **ACE** inhibitors for **DM**
*ACC/AHA **Stage B***	As for **Stage A** + **ACE inhibitors** ± **β-blockers** (in appropriate patients)
*ACC/AHA **Stage C***	As for **Stage B** + **salt restriction**, **diuretics** ± **aldosterone** antagonists (e.g. spironolactone), **inotropic** agents (e.g. digoxin), **vasodilators** (e.g. hydralazine, nitrates) and/or **ICD** placement (e.g. EF < 30%)
*ACC/AHA **Stage D***	As for **stage C +** heart **transplantation**, chronic **inotropic** and/or **mechanical** support; consider **experimental** therapies

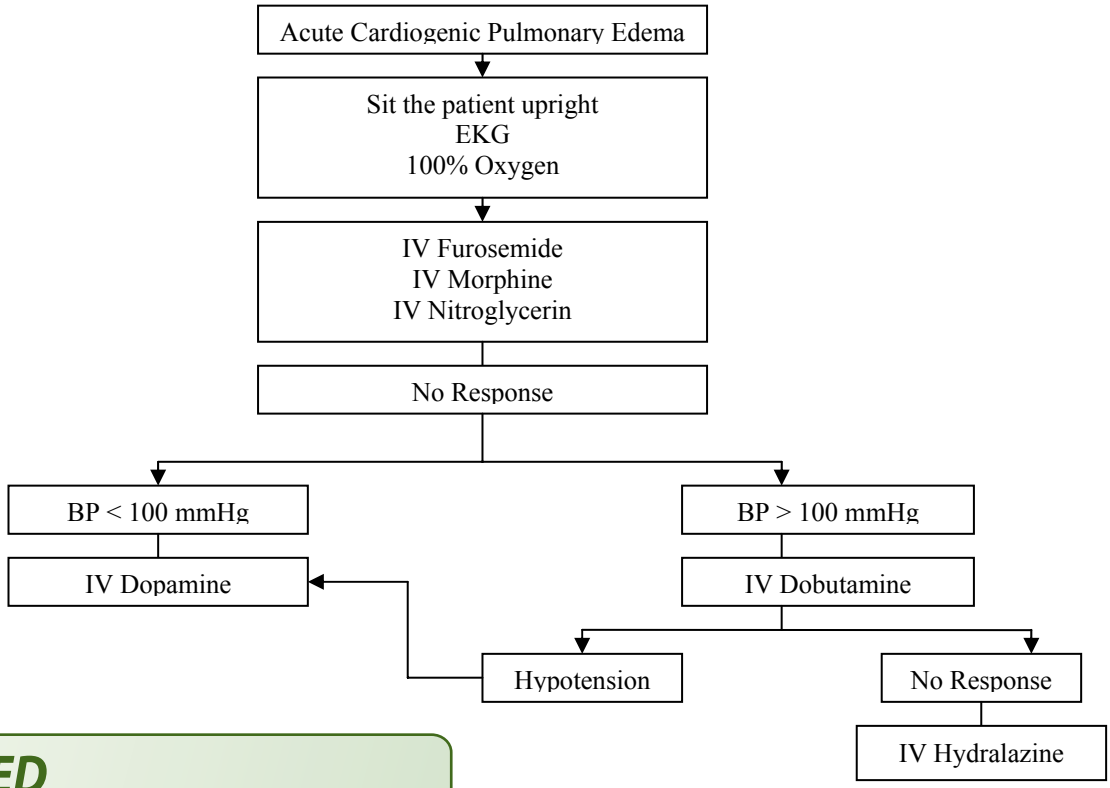
- **Indications** for **ACE inhibitor** therapy:
  1. **Symptomatic** CHF
  2. **Asymptomatic** CHF + **DM**
  3. **Asymptomatic** CHF + **EF** < **35-40%**
  4. **Post-MI** (especially **anterior MI**) if **EF < 40%** and/or manifestations of **CHF**
- Manifestations (clinical and/or radiographic) of **systemic** and/or **pulmonary congestion** → add **diuretics**
- **Do not** use **β-blockers** in → **decompensated CHF** (first stabilize with ACE inhibitors and diuretics) and/or **NYHA Class IV** CHF
- **Symptomatic** despite **standard therapy** (ACE inhibitors + diuretics ± β-blockers) → add **spironolactone** (especially beneficial in **NYHA class III** and **IV** patients)
- **Intolerance** to **ACE** inhibitors (e.g. chronic cough) → **ARBs**
- **Intolerance** and/or **contraindications** to **ACE** inhibitors and/or **ARBs** → **hydralazine** + high-dose **nitrates** (especially beneficial in **African-American** patients)
- **CHF** + **Afib** → start **digoxin** (although **β-blockers** can also be used)
- **Diastolic** dysfunction → **β-blockers** and/or **Ca-channel** antagonists (e.g. verapamil, diltiazem) ± diuretics
- **Severely symptomatic** despite **all** available therapy → 48-hour **infusion** of **inotropic** agents (e.g. dobutamine, milrinone); may ↑ **mortality**

* **Drug** that have shown to ↓ **mortality in CHF:**

1. **ACE** inhibitors
2. **β-blockers** (only **carvedilol, metoprolol** and **bisoprolol**)
3. **Aldosterone** antagonists
4. **ARBs**
5. Combination of **hydralazine** and **nitrates**

Acute Cardiogenic Pulmonary Edema

- **Sudden increase** in **LV filling pressure** → **pulmonary** venous **congestion** → acute pulmonary **edema** → possible **respiratory failure**
- Common precipitants → extensive **MI**, severe **arrhythmias**
- Clinical features → **tachypnea, dyspnea, cough** ± hemoptysis, **tachycardia,** diaphoresis, pulmonary **rales** ± **wheezing**
- Chest X-ray → evidence of pulmonary **congestion** (e.g. prominent vascular markings)
- **Best initial step** in patient management → **EKG** to rule out associated **ischemia/infarction** and/or serious **arrhythmias**
- **Best initial therapy** → 100% **oxygen** (remember the **ABCs**)
- **Best next** step → **furosemide** + **morphine** + **nitroglycerin** (avoid if BP < 90 mmHg)
- *See algorithm*



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Digoxin and other cardiac glycosides

- Mechanism of Action: **Inhibition** of **Na/K –ATPase** → ↑ intracellular Na → ↓ activity of Na-Ca – exchanger → ↑ intracellular Ca → ↑ **contractility**
- Other actions → **parasympathetic** nervous system **activation**
- Clinical Uses → **CHF, Afib** and/or **AF**
- Factors that **increase** digoxin **toxicity** ± levels:
    1. **hypokalemia**
    2. **hypercalcemia**
    3. **hypomagnesemia**
    4. **renal** insufficiency
    5. ↑ **age**
    6. thiazide and/or loop **diuretics** (hypokalemia)
    7. **spironolactone** (↓ renal excretion)
    8. **quinidine** (↓ renal excretion)
    9. **verapamil** (↓ renal excretion)
    10. **amiodarone** (multiple effects, including ↓ renal excretion, ↓ protein binding)
    11. **NSAIDs** (e.g. indomethacin)
    12. **erythromycin** (↓ intestinal flora → ↓ intestinal metabolism)
- Factors that **decrease** digoxin **toxicity** ± levels:
    1. **hyperkalemia**
    2. **antacids** (↓ bioavailability)
    3. **cholestyramine** and other bile-acid binding resins (↓ enterohepatic circulation)
- Side effects:
    1. **GI distress** → anorexia, nausea, vomiting, diarrhea
    2. **Visual disturbances → yellow-green halos**, blurry vision
    3. **CNS** effects → confusion, drowsiness, dizziness ± psychosis/delirium
    4. **Arrhythmias** → virtually any type of brady- and/or tachyarrhythmia; "**PAT with block**" (**pathognomic** for digoxin toxicity)
    5. **Hyperkalemia**
    6. **Gynecomastia**
- Management of digoxin toxicity:
    1. Supportive measures
    2. **Correction** of **electrolyte** abnormalities
    3. Ventricular arrhythmias → **lidocaine** and/or **phenytoin**
    4. Supraventricular arrhythmias → **β-blockers**
    5. **Indications** for **Digibind** (digoxin-fab fragments) administration:
        - Hemodynamic **instability**
        - **Hyperkalemia**
        - Altered **LOC**
        - Accidental/intentional **overdose** (>10 mg)

Hypertension (HTN)

Definition/Classification

- **Systolic BP** ≥ **140** mmHg and/or **diastolic BP** ≥ **90** mmHg measured on **≥ 2** occasions
- Divided into **essential/primary** (**90-95%** of cases) and **secondary** forms of disease

Stage	***Systolic BP***	***Diastolic BP***
Normal	< 120 mmHg	< 80 mmHg
Pre-hypertension	120 – 139 mmHg	80 – 89 mmHg
Stage 1 hypertension	140 – 159 mmHg	90 – 99 mmHg
Stage 2 hypertension	≥ 160 mmHg	≥ 100 mmHg

Essential (Primary) Hypertension

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- **Most common** form of HTN
- **No identifiable** causative factors
- Risk factors → ↑ **age**, **male** gender, **African-American** race, **family history** of HTN, **obesity**/sedentary lifestyle, ↑ **sodium** diet, **excessive alcohol** intake, **metabolic syndrome**, **pre-hypertension**
- Clinical features → usually **asymptomatic**; possible manifestations of **end-organ damage** and/or **hypertensive emergency** *(see below)*

Target Organ	***Manifestations***
Heart	**LV hypertrophy** (e.g. S_4 sound, ↑ intensity of A_2, prominent apical impulse) **CHF** (e.g. S_3 sound, peripheral/pulmonary congestion) **CAD** (e.g. angina, MI)
Blood vessels	**Peripheral Artery** Disease (e.g. intermittent claudication, ↓ pulses) Aortic **dissection** Aortic **aneurysms**
Brain	**TIAs/stroke** Dementia
Kidneys	**Chronic renal failure** Proteinuria, hematuria
Eyes	Hypertensive **retinopathy** *(see below)*

- Diagnosis → *see definition*

* Keith-Wagener-Barker classification of **hypertensive retinopathy:**

1. Grade 1 – arteriolar **narrowing**
2. Grade 2 – arteriovenous **nicking**, "**copper-wiring**"
3. Grade 3 – flame-shaped **hemorrhages**, "**cotton-wool**" spots, hard **exudates**
4. Grade 4 – as above + **papilledema**

* **"White-Coat"** hypertension → **HTN** when measured **in the clinic**/office *plus* **no evidence** of end-organ damage; confirmation → **normal BP** when self-measured **at home**
* "**Masked"** hypertension → **HTN** when measure **outside the clinic**/office ± evidence of **end-organ** damage; confirmation → **24h** ambulatory **BP monitoring**

* **"Pseudo-**hypertension**"** → **HTN** when measured by the **cuff method** secondary to **stiffness** of the vascular tree *plus* **no evidence** of end-organ damage; confirmation → **intra-arterial** BP measurement

- Follow-up:
    1. **Normal BP** → ~ **2 years**
    2. **Pre-hypertension** → ~ **1 year**
    3. **Stage 1** HTN → ≤ **2 months**
    4. **Stage 2** HTN → ≤ **1 month**
    5. **Stage 3** HTN (BP > 180/110 mmHg) → ≤ **1 week** and/or **immediate** treatment

* **Routine evaluation** (to rule out complications and/or secondary HTN):
    1. CBC
    2. Urinalysis
    3. Serum creatinine/BUN
    4. Serum electrolytes
    5. Blood glucose
    6. Lipid profile
    7. EKG

- Findings suggestive of essential HTN →
    1. **Age 30-50**
    2. **Family history** of HTN
    3. **Mild** HTN
    4. **Easily controlled** HTN
    5. **No evidence** of end-organ damage
    6. **Normal** lab findings
- **BP goal** in **uncomplicated** HTN → **< 140/90** mmHg
- **BP goal** in HTN + **DM, kidney** disease, **CAD** (or CAD-equivalents) → **< 130/80** mmHg
- **BP goal** in HTN + **CAD** + ↓ **EF** → **< 120/80** mmHg
- Management:

Pre-hypertension	**Life-style** modification
Stage 1 hypertension	**Life-style** modification for ~ **1 year**
Stage 1 hypertension + **risk factors** for **CAD**	**Life-style** modification for ~ **3-6 months**
Stage 1 hypertension + **DM**, **end-organ** damage and/or **cardiovascular** disease	**Life-style** modification + **pharmacological mono-**therapy
Stage 2 hypertension	**Life-style** modification + **pharmacological combination** therapy

- **Life-style** modification:
    1. **DASH** (dietary approaches to stop hypertension) **eating plan** → diet rich in **potassium** (fresh fruits & vegetables) and **calcium** (low-fat dairy products); **reduced** amount of **fats**
    2. **Sodium restriction**
    3. Regular **exercise**
    4. **Weight loss** (~ 0.5-2.0 mmHg drop in BP for every kg weight loss)
    5. ↓ **alcohol** consumption
- **Drug of choice** for **uncomplicated** HTN in the **absence** of **specific indications** *(see below)* → **thiazide** diuretics (e.g. hydrochlorothiazide)
- HTN **not controlled** with **mono-therapy** → two-drug **combinations** (add diuretics, if not already in use)
- Specific indications:

DM ± proteinuria	**ACE** inhibitors
CHF	**ACE** inhibitors, **diuretics** and/or **β-blockers**
CAD	**β-blockers**, **ACE** inhibitors
GFR < 30 mL/min	**Loop**-diuretics
Supraventricular arrhythmias	**β-blockers, Ca-channel** antagonists
Raynaud phenomenon	**Ca-channel** antagonists
Peripheral artery disease	**Ca-channel** antagonists
Osteoporosis	**Thiazide** diuretics
BPH (benign prostatic hypertrophy)	**α_1-antagonists**
Migraine	**β-blockers, Ca-channel** antagonists
Recurrent **nephrolithiasis**	**Thiazide** diuretics
Isolated systolic HTN	**Thiazide** diuretics
Pregnancy	**α-methyldopa, hydralazine, labetalol, Ca-channel** antagonists
Hyperlipidemia	**α_1-antagonists**
Elderly, African-Americans	**Thiazide** diuretics, **Ca-channel** antagonists

Hypertensive Crises

- Hypertensive **urgency** → ↑↑ **BP** (**diastolic BP** ≥ **130** mmHg) **without** evidence of end-organ damage
- Hypertensive **emergency** → ↑↑ **BP** *plus* **acute,** progressive **end-organ damage**; examples include **malignant** HTN, **ACS**, aortic **dissection**, **encephalopathy**, acute **pulmonary edema** and/or **acute renal failure**
- **Malignant** HTN → ↑↑ **BP** *plus* **encephalopathy,** progressive **renal failure** and **papilledema**
- **Accelerated** HTN → ↑↑ **BP** *plus* retinal **hemorrhages/exudates** but **no papilledema**
- Risk factors → **untreated** HTN, **sudden discontinuation** of anti-hypertensive therapy (especially clonidine and/or β-blockers), **scleroderma**, **renovascular** disease
- Clinical features → hypertensive **encephalopathy** (headache, confusion, focal neurologic signs, seizures, papilledema, visual loss, stupor, coma), **nausea/vomiting**, **oliguria**/anuria ± **hematuria**, **chest pain**, **dyspnea**, acute **pulmonary edema**
- Diagnostic evaluation (in addition to focused history, physical and fundoscopic examinations) → **hemoglobin**/**blood smear** (look for **schistocytes**), urinalysis, creatinine/BUN, serum electrolytes, blood glucose, **chest X-ray**, **EKG**, ± **head CT**

- Management → Parenteral **therapy** *(see below)*
- **Goal diastolic BP** → ~ **100-110** mmHg within **1-2 hours** (~ **20-25%** decrease in mean BP); **further reduction** may lead to → **cerebral/myocardial ischemia,** mesenteric ischemia

* Note → hypertensive urgency does not require hospitalization and/or parenteral therapy

Medication	***Most Useful***	***Best avoided***
Nitroprusside		↑ **ICP**
Labetalol	Aortic **dissection** **Pregnancy**	**Acute heart failure**
Nitroglycerin	**Acute heart failure** **ACS**	
Nicardipine		**Acute heart failure**
Enalaprilat	**Acute heart failure**	**Pregnancy**
Phentolamine	↑ **catecholamines** (e.g. pheochromocytoma, clonidine withdrawal, cocaine abuse)	
Hydralazine	**Pregnancy**	Aortic **dissection** Myocardial **ischemia**
Esmolol	Aortic **dissection** Myocardial **ischemia**	**Acute heart failure**
Fenoldopam	↓ **renal function**	

Selected Causes of Secondary Hypertension

Renovascular Hypertension (Renal Artery Stenosis)

- **Most common** cause of **curable secondary HTN**
- Secondary to:
  1. **fibromuscular dysplasia** → **females** > males; **age < 30**
  2. **atherosclerosis** → **age > 50**
- Usually **unilateral** (**bilateral** disease suggested by **sudden** ↓ in **renal function** with the introduction of **ACE** inhibitors and/or **ARBs**)
- Clues:
  1. **new-onset** HTN before **age < 30** or after **age > 50**
  2. **sudden worsening** of **essential HTN**
  3. **severe** HTN + severe **peripheral artery disease**
  4. **severe** HTN + severe hypertensive **retinopathy**
  5. **disparity** in renal **size**/function
- Key features → upper **abdominal bruit; refractory** HTN
- **Best initial** diagnostic tests → renal artery **duplex ultrasonography**, **MRA** or **CT angiography**
- **Most accurate** diagnostic test → renal **angiography**

* Other diagnostic tests to consider → **captopril renal scan** (positive result → decreased radionuclide uptake after captopril administration) and **renal vein renin** studies (lateralization of renin levels)

- **Best initial therapy** → percutaneous transluminal **angioplasty** ± **stenting**
- **No response** to angioplasty → **surgical** intervention
- **No response** to either intervention → **medical** management (consider **ACE inhibitors** and/or ARBs for **unilateral disease**)

Coarctation of the Aorta

- **Males** > Females
- Common associations → **Turner** syndrome, **bicuspid** aortic valve
- Key features → **headache**/nosebleeds + cold feet/**claudication**, ↑ **BP** in the **upper extremities** with ↓/**unobtainable BP** in the **lower extremities**; ↓ **femoral pulses**, visible **chest wall**/neck **pulsations**, **murmurs** heard over the **chest** and **back**
- Chest X-ray → **rib notching**, **figure "3"** appearance of the **aorta**
- **Best initial** diagnostic test → **echocardiography**
- **Most accurate** diagnostic test → **angiography**
- Management → **angioplasty ± stenting** (treatment of choice) or **surgical** intervention

Other Causes of Secondary HTN

- **Pheochromocytoma** *(see Endocrinology)*
- **Primary hyperaldosteronism** *(see Endocrinology)*
- **Cushing's syndrome** *(see Endocrinology)*
- Hyperthyroidism *(see Endocrinology)*
- Acromegaly *(see Endocrinology)*
- Hyperparathyroidism *(see Endocrinology)*
- Congenital adrenal hyperplasia *(see Endocrinology)*
- **Renal parenchymal** disease → **most common** form of **secondary HTN**, treat with **salt restriction, diuretics** (usually loop diuretics) ± **ACE** inhibitors (if **proteinuria**)
- **OCPs** → **discontinue** OCP use and/or **switch** to **progestin-only** contraception

Drugs that Interfere with the Treatment of HTN

- **OCPs**
- **NSAIDs**
- Excessive **alcohol**
- OTC **allergy** and/or **cold medications**

Anti-Hypertensive Medications

Diuretics

APPROVED
By Zurab Azmaiparashvili at 2:46 am, Jan 24, 2009

Carbonic Anhydrase Inhibitors

- Agents: **acetazolamide,** dorzolamide
- Mechanism of Action: **inhibition** of **carbonic anhydrase** → ↑ **$NaHCO_3$** excretion
- Site of Action: **proximal** convoluted tubule
- Side Effects: sulfa **allergy**, hyperchloremic **metabolic acidosis**, **hypokalemia**, precipitation of **kidney stones**, **paresthesias**
- Relative Contraindications: ↓ **liver** function (secondary to ↑ **NH_3 absorption**)

Thiazide Diuretics

- Agents: **hydrochlorothiazide**, **indapamide** (also a vasodilator), **metolazone**
- Mechanism of Action: **inhibition** of **Na/Cl cotransporter**
- Site of Action: **distal** convoluted tubule
- Side Effects: **hypokalemia**, metabolic **alkalosis**, **hyponatremia**, **hypercalcemia**, **hyperglycemia**, **hyperuricemia, hyperlipidemia,** sulfa **allergy**
- Relative Contraindications: **DM, gout, dyslipidemia**

Loop Diuretics

- Agents: **furosemide, ethacrynic** acid, **bumetanide,** torsemide
- Mechanism of Action: **inhibition** of **Na/K/2Cl cotransporter**
- Site of Action: **thick ascending** limb of **loop of Henle**
- Side Effects: **hypokalemia,** metabolic **alkalosis**, dehydration, **hypocalcemia,** hypomagnesemia**, hyperglycemia, hyperuricemia, hyperlipidemia,** sulfa **allergy, ototoxicity**
- Relative Contraindications: **DM, gout, dyslipidemia**

K-sparing Diuretics

- Agents: **spironolactone, triamterene, amiloride**
- Mechanism of Action: **aldosterone antagonism** (spironolactone), **Na** channel **blockade** (triamterene, amiloride)
- Site of Action: **collecting** tubules/ducts
- Side Effects: **hyperkalemia,** metabolic **acidosis**, **gynecomastia** (spironolactone), **nephrolithiasis** (triamterene)

Osmotic Diuretics

- Agents: **mannitol**
- Mechanism of Action: ↑ urine **osmolarity** → ↑ urine flow rate
- Site of Action: **proximal** convoluted tubule
- Side Effects: **GI distress**, dehydration, **pulmonary edema,** hypo-/hypernatremia
- Relative Contraindications: **CHF**

Sympathoplegics

Centrally-Acting α-2 Receptor Agonists

Medication	*Major Side Effects*	*Other uses*
α-methyldopa	**Sedation** Dry mouth Depression Sexual dysfunction **Positive Coombs** test/**hemolytic** anemia **Hepatitis** **Lupus**-like syndrome	
Clonidine (guanabenz, guanfacine)	Sedation **Dry mouth** Depression Sexual dysfunction **Rebound hypertension**	**Opiate withdrawal**

Miscellaneous Agents

Medication	*Mechanism of Action*	*Major Side Effects*
Trimethaphan *Hexamethonium*	**Blockade** of **autonomic ganglia**	**Blurry vision** **Constipation** **Urinary retention** **Sexual** dysfunction **Orthostatic** hypotension
Reserpine	**Depletion** of norepinephrine **stores** from nerve terminals	**Depression** **GI distress**
Guanethidine	**Blockade** of norepinephrine **release** from nerve terminals	**Orthostatic** hypotension **Sexual** dysfunction

α-1 receptor antagonists

- Agents: **prazosin**, terazosin, doxazosin, **tamsulosin**
- Major Side Effects: **1st-dose hypotension**/syncope, **worsening** of urinary **incontinence**
- Other Uses: **BPH,** acute treatment of **renal colic**

β-blockers *(see anti-anginal medications)*

Vasodilators

Hydralazine

- Mechanism of Action: **NO** release
- Site of Action: **arterioles**
- Major Side Effects: **lupus-**like syndrome, **marked compensatory** responses (fluid retention, reflex tachycardia)

Minoxidil

- Mechanism of Action: **opening** of **K channels**
- Site of Action: **arterioles**
- Major Side Effects: **hirsutism, pericardial** effusion, **marked compensatory** responses
- Other Uses: **alopecia** (topically)

Diazoxide

- Mechanism of Action: **opening** of **K channels**
- Site of Action: **arterioles**
- Major Side Effects: **hyperglycemia** (↓ insulin secretion), **marked compensatory** responses
- Other Uses: **insulinomas**

Nitroprusside

- Mechanism of Action: ↑ activity of **guanylyl cyclase → NO** release
- Site of Action: **venules**, **arterioles**
- Major Side Effects: **methemoglobinemia** (antidote: **methylene blue**), **cyanide** toxicity (antidotes: **amyl nitrite**, sodium **nitrite** and sodium **thiosulfate**), **thiocyanide** toxicity (in patients with ↓ **renal function**)

Ca-channel Antagonists *(see anti-anginal medications)*

ACE Inhibitors

- Agents: **captopril. enalaprili, lisinopril,** etc.
- Mechanism of Action: ↓ activity of **angiotensin-converting enzyme** → ↓ production of **angiotensin II** and ↓ degradation of **bradykinin**
- Major Side Effects: chronic **cough**, angioedema, **hyperkalemia, renal** impairment (especially in patients with **renovascular hypertension**), **neutropenia, rash** (especially with **captopril**), **taste** disturbances (especially with **captopril**)
- Absolute Contraindications: **bilateral renal artery stenosis, pregnancy** (**renal** malformations, fetal **hypotension**, etc.)

Angiotensin-Receptor Blockers (ARBs)

- Agents: **losartan,** valsartan, irbesartan, etc.
- Similar to **ACE inhibitors**, but:
  1. **no effect** on **bradykinin** metabolism, hence
  2. ↓ **incidence** of dry **cough**
- Major Use: **intolerance** to **ACE inhibitors**, secondary to chronic **cough**

Diseases of the Aorta

APPROVED

By Zurab Azmaiparashvili at 2:46 am, Jan 24, 2009

Aortic Dissection

- **Tear** of the **intimal layer** of the aorta with **secondary dissection** into the **media** (less frequently, medial hemorrhage with secondary intimal disruption)
- **Stanford** classification:
  1. **Type A** → involving the **ascending aorta** (proximal dissection)
  2. **Type B** → **limited** to the **descending aorta** (distal dissection)
- **DeBakey** classification:
  1. **Type I** → involving both the **ascending** and the **descending aorta**
  2. **Type II** → **limited** to the **ascending aorta**
  3. **Type III** → **limited** to the **descending aorta**
- Risk factors:
  1. **Hypertension** (**most common** underlying abnormality)
  2. **Cystic medial necrosis,** secondary to **connective tissue** disorders (e.g. **Marfan** syndrome, **Ehlers-Danlos** syndrome)
  3. **Coarctation** of the aorta
  4. **Bicuspid** aortic valve
  5. **Pregnancy**
- Clinical features:
  1. **Sudden** onset of **severe, "tearing", "ripping" chest pain,** often involving the **interscapular** region
  2. Significant **BP difference** between the **right** and the **left arms**
  3. **Left**-sided **pleural effusion**
  4. **Acute AR** → acute onset of **CHF**, new-onset **diastolic murmur**, **early S1** sound
  5. Cardiac **tamponade**

6. Acute **MI**
7. **Stroke** (e.g. hemiplegia)
8. **Spinal cord** ischemia (e.g. paraplegia)
9. **Mesenteric** ischemia
10. **Compression** of **adjacent** structures → **dyspnea** (trachea)**, dysphagia** (esophagus)**, hoarseness** (recurrent laryngeal nerve)**, SVC** syndrome (superior vena cava), **Horner's** syndrome (superior cervical ganglia)

- EKG → helpful in **ruling out** acute **MI** (unless aortic dissection involves the coronary ostia)
- Chest X-ray → **widening** of the **mediastinum, left-**sided **pleural effusion**, **blunting** of the **aortic knob, "calcium"** or "**ring"** sign, **tracheal deviation**
- **Best initial** diagnostic test → chest **CT scan** with **I/V contrast** (some test Qs may offer **trans-esophageal echocardiography** as an alternative, especially in **hemodynamically unstable** patients)
- **Most accurate** diagnostic test → **angiography** (e.g. when chest X-ray shows wide mediastinum and CT scan is non-diagnostic)

* Although MRA can also be used as an initial diagnostic test, it's not uniformly available, requires more time and costs more

- Management:
  1. **β-blockers** (even in patients with normal BP) → ↓ dissection **propagation**
  2. Add **nitroprusside** for **optimal BP control** (consider **labetalol** as a valuable alternative)
  3. **Goal HR → 60-80** bpm
  4. **Goal systolic BP →** ≤ **120** mmHg
  5. **Surgical** intervention →
     - **All Type A** dissections (emergently)
     - **Complicated Type B** dissections (e.g. rupture, propagation, vascular compromise)
     - **Dissections** in patients with **Marfan** syndrome

*** Note → direct vasodilators** (without the prior use of β-blockers) are **contraindicated** in aortic **dissection** (↑ risk of propagation)