CARDIOLOGY II:

- Cardiac Valvular Disease
- Ischemic Heart Disease
- Congenital Heart Disease (Adult presentations)
- Vascular Disease

Cardiac Valvular Disease

- Aortic – Stenosis and Regurgitation
- Pulmonic
- Mitral
- Tricuspid

Cardiac Valvular Disease

- Historically in US – Rheumatic in origin
- Still true in developing countries
- Now, atherosclerosis involved
- ? Genetic markers with AS ?
- Many patients are s/p surgical intervention
- ECHO remains best diagnostic tool

Valvular Dz Practice Case

A 22 y/o waitress presents c/o generalized, sub-sternal chest pain that is worsened with exertion. She appears anxious; she denies ETOH, tobacco, and illicit drug use. You auscultate her heart and diagnose MVP. What did you hear to make this diagnosis?

Choices:

A. A diastolic rumble
B. A holo-systolic murmur
C. A midsystolic click
D. An opening snap

Answer:

A. A diastolic rumble
B. A holo-systolic murmur
c. A midsystolic click
D. An opening snap

Valvular Disease Basics

Four Valves:

- Aortic
- Mitral
- Tricuspid
- Pulmonic

Two main conditions:

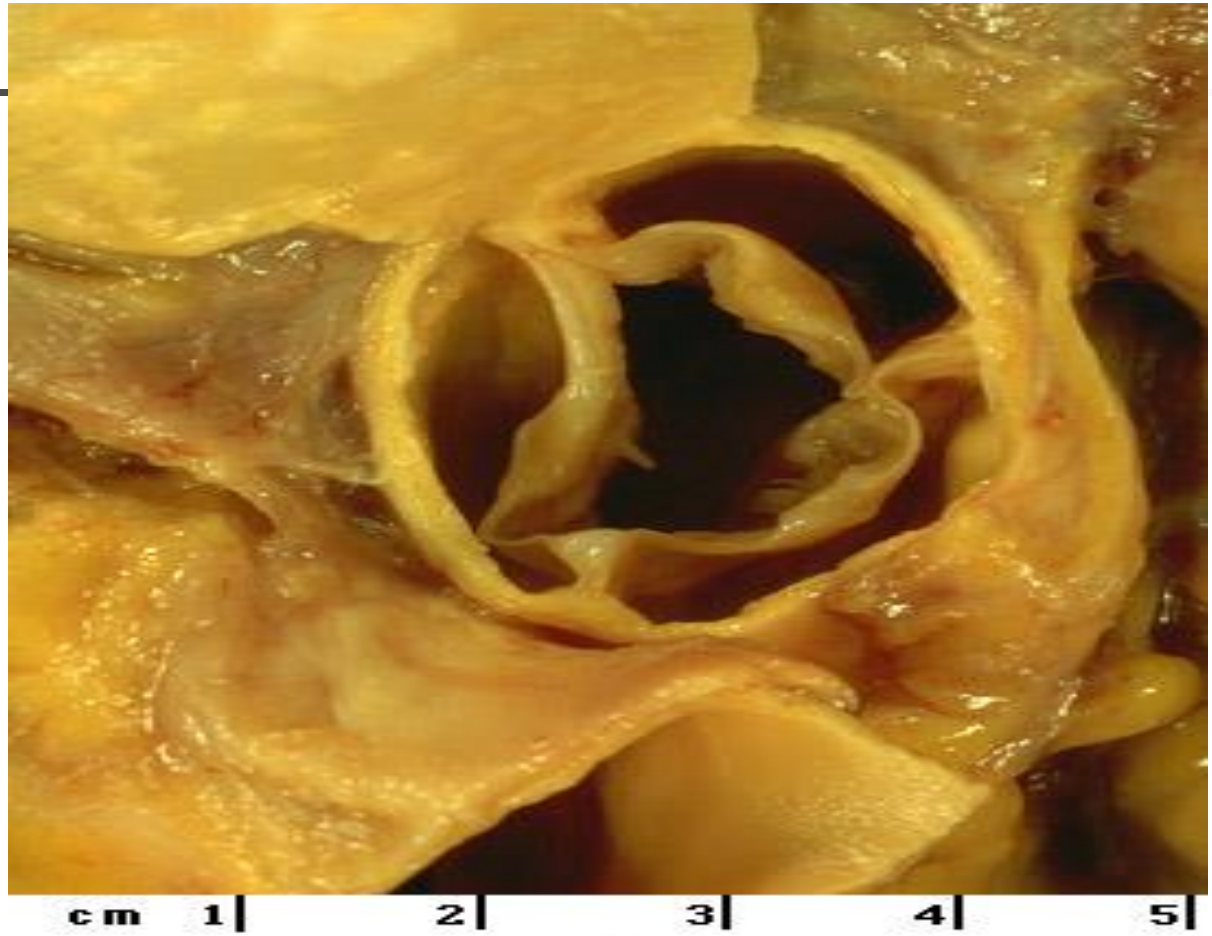
- Stenosis
- Regurgitation or insufficiency

Valvular Disease - localization

- Aortic area: 2nd R interspace
- Pulmonic area: 2nd L interspace
- Tricuspid area: LLSB
- Mitral area: Apex

(Think <u>APT.</u> <u>M</u>ent — going from right to left along patient's chest)

The Aortic Valve



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Aortic Stenosis (AS)

2 'routes of entry'/causes possible:

- Uni/bicuspid aortic valve (congenital)
  - often presents at 50-65 y/o
- Degenerative or calcific aortic valve
  - Results from calcium deposits 2º to atherosclerosis
- (Genetic markers associated: "*Notch 1*")
- So . . AS is the most common surgical valve lesion; most pts. are elderly

Aortic Stenosis – s/s

- dyspnea, angina, syncope w/ exertion
- LV failure common in severe AS
- LVH – displaced, powerful PMI
- Systolic ejection murmur, often harsh and loud w/ thrill
- Heard best (leaning forward w/expiration) over aorta, radiates to neck, apex, LSB

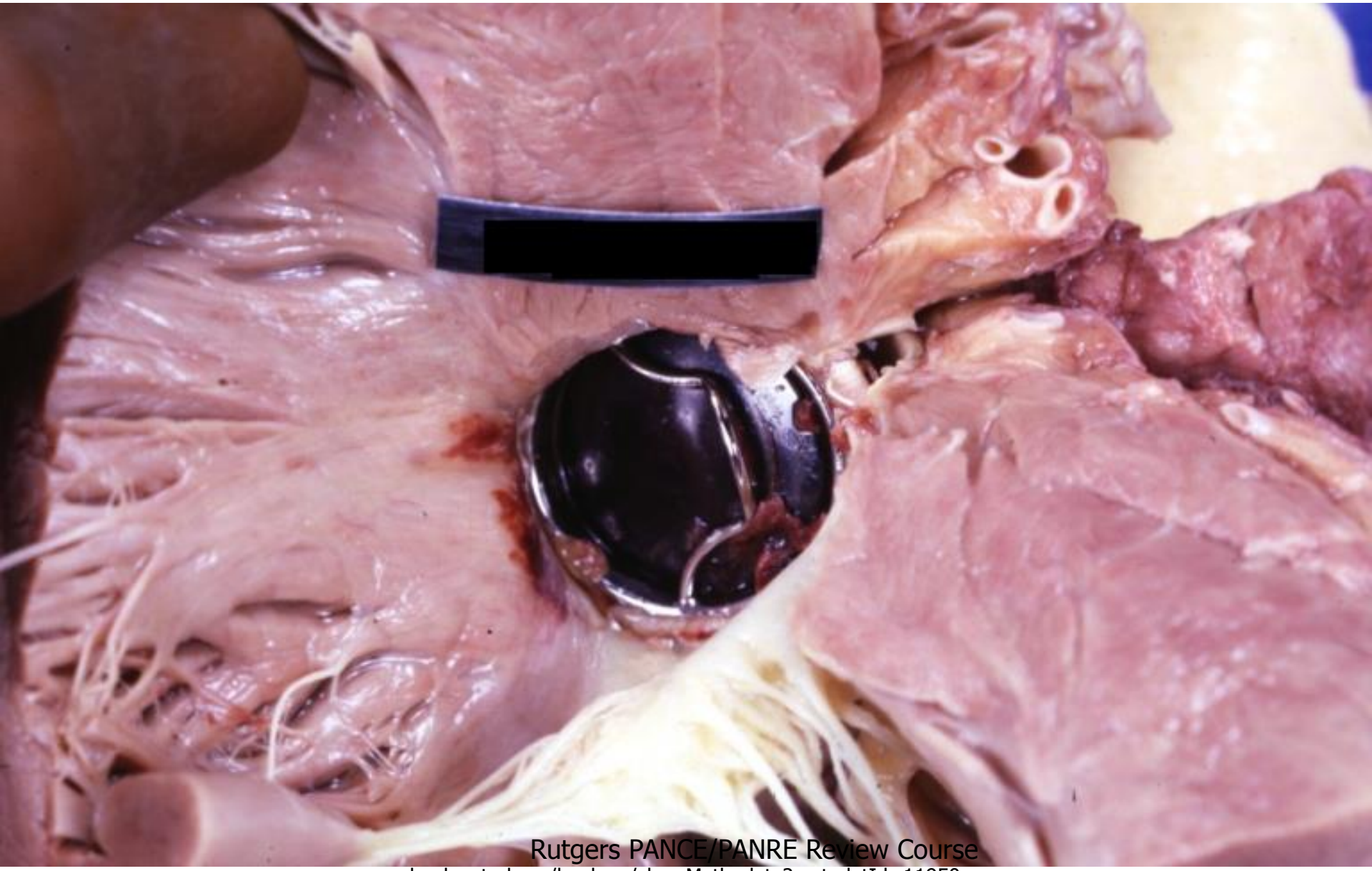
Aortic Stenosis - Diagnosis

- EKG: normal or LVH
- CXR: cardiomegaly, calcified valve and prominent ascending aorta
- Doppler ECHO: very good for anatomy and valve gradient
- Cardiac Cath: best for surgical clearance
- NEW: BNP >550 @ poor surgical result

Aortic Stenosis - Prognosis

- After triad of HF, Angina, Syncope – prognosis *without* surgery is poor
- Valve replacement has great results
  - Ross procedure in young (pulm valve)
  - Mechanical valve replacement (anticoag); now TAVR (transcatheter aortic valve replacement) surgery possible (not 'open heart')
  - Bioprosthetic valve (bovine/porcine) in very elderly has 10-15 year life
  - After surgery, EF may improve significantly

Mechanical Heart Valve replacement



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Aortic Regurgitation (AR)

- Rheumatic AR decreasing
- Causes include:
  - Congenital bicuspid aortic valve
  - Infective endocarditis
  - HTN
  - Aortic root disease e.g. Marfan's
  - Inflammatory disease e.g. Reiter's syndrome (rare)

Aortic Regurgitation – s/s

- Usually asymptomatic early-on; then DOE
- Wide pulse pressure, "water-hammer" pulse/Corrigan's pulse in chronic AR
- Hyperactive, enlarged LV; hyperdynamic and displaced PMI
- Soft, high pitched, blowing diastolic murmur along LSB, with pt. sitting, leaning forward after exhaling

Aortic Regurge - diagnosis

- EKG: moderate to severe LVH
- CXR: cardiomegaly esp. LV
- ECHO: best used serially with color doppler to estimate severity of regurgitation and need for surgery
- Cardiac Cath: useful pre-op to assess for CAD

Aortic Regurge - Prognosis

- Acute regurge (after infective endocarditis) results in acute LV failure and requires immediate surgery
- Chronic regurge
  - ACE-I and ARBs reduce symptoms
  - Valve replacement surgery as in AS
  - Some improvement in LV function and EF

Mitral Stenosis (MS)

- Classically, rheumatic in origin
- Can result from congenital disease
- Findings include thickened leaflets, fusion of the chordae, and calcium deposits in the valve
- Initial symptoms often with A-fib or pregnancy

Mitral Stenosis – s/s

- History of rheumatic fever; may have orthopnea, PND, exertional dyspnea
- Localized, mid-diastolic, low-pitched rumble heard at apex with bell while pt. is in L lateral position (louder w/ exer.)
- Opening snap heard after S2
- 50-80% develop atrial fibrillation

Mitral Stenosis - diagnosis

- EKG: LAE/RAE; +/- Atrial fibrillation
- ECHO: thick, stiff mitral valve; "hockey stick" shape to anterior leaflet
- Cardiac Cath: used to assess CAD, prior to surgery

Using echocardiography to diagnose valvular disorders

www.commons.wikimedia.org/wiki/Echocardiogram

Mitral Stenosis - Prognosis

- Often long asymptomatic period
  - Pregnancy can precipitate symptoms
  - Treat a-fib if present (anticoagulate)
- Always prescribe abx for prophylaxis w/valve replacement/surgery
- Percutaneous balloon valvuloplasty and surgical replacement have low mortality and are definitive treatment options

Mitral Regurgitation (MR)

- Anatomical findings differ with similar end results
- Initially increased pre-load and reduced after-load
- Eventually LV enlarges, weakens and EF drops

Mitral Regurgitation – s/s

- Gradually progressing dyspnea and fatigue over many years
- LAE and LVH with atrial fibrillation
- Harsh, blowing pansystolic murmur at the apex, radiates to L axilla
- Associated apical S3
- PMI has increased amplitude and duration, and possible thrill palpable

Mitral Regurge - diagnosis

- EKG: LAD, LVH, LAE and +/- atrial fib;
- BNP may help identify LV failure
- ECHO and TEE are best diagnostic tools
- Cardiac Cath – as previously stated

Mitral Regurge - Prognosis

- Vasodilators, ACE-Is, or intra-aortic balloon counterpulsation may "buy time" for acute MR
- Symptoms or reduced EF (<60%) are indication for surgery
- "Stay-tuned" for percutaneous and mitral clip devices as surgical alternates

Mitral Valve Prolapse (MVP)

- Thin, young females with pectus or scoliosis (~10% of healthy females)
- AKA "floppy" or myxomatous mitral valve
- Associated with hyperadrenergic syndrome esp. in young females
- Often attenuates with age

MVP – s/s

- Usually asymptomatic; non-specific CP, dyspnea, palpitations

- Mid-systolic click(s)
- Pan/late systolic murmur, expanding with severity of valve disease
- Click and murmur increase with standing or Valsalva

MVP – diagnosis & prognosis

- Clinically diagnosed; ECHO confirms
- B-blockers may be used w/ hyperadrenergic state
- Surgical repair favored over valve replacement
- Abx prophylaxis no longer recommended (regardless of +/- regurgitation)

Tricuspid Stenosis (TS)

- Uncommon valvular disease
- Females with Rheumatic disease
- Often a result of tricuspid valve repair or carcinoid syndrome (malignant neoplasms) in US

Tricuspid Stenosis – s/s

- Right-heart failure
  - Hepatomegaly
  - Ascites
  - Dependent edema
- Elevated JVP with giant *a* wave
- Diastolic, rumbling murmur heard at LLSB that increases with inspiration; use bell
- Pulsating hepatomegaly possible

TS – Diagnosis & Prognosis

- EKG: RAE, RVH, +/- atrial fib.
- ECHO & Cath show pressure gradient

- Diuretics may decrease RHF symptoms
  - Torsemide better than furosemide in presence of bowel edema
- Bioprosthetic valve is treatment of choice

Tricuspid Regurgitation (TR)

- Commonly occurs with RV dilation 2º pulmonary HTN or cardiomyopathy
- May result from pacemaker lead placement (iatrogenic cause now increasing)
- May be primarily caused by TV prolapse, carcinoid plaque, collagen inflammatory disease or tricuspid endocarditis

Tricuspid Regurgitation – s/s

- RV Failure signs and symptoms
- High JVP; large "*v*" wave
- Blowing, medium pitched, holosystolic murmur heard at LLSB, +/- S3; louder with inspiration
- Cyanosis may be present

TR – Diagnosis & Prognosis

- EKG: non-specific; a-fib, RAE, RVH
- ECHO & Cath: confirm regurgitance

- Minor regurge is well-tolerated: diuretics may be helpful
- Eliminate causes of TR, then surgical definitive intervention is best, using a bioprosthetic valve

Pulmonary Stenosis (PS)

- Congenital disorders > 95% of cases
- Due to valve fibrosis and thickening
- Acquired form occurs with carcinoid heart disease
- Frequently asymptomatic

Pulmonic Stenosis – s/s

- Frequently asymptomatic
- Gradually increasing DOE, CP, syncope

- Prominent JVP and "*a*" wave
- Harsh, loud, medium-pitched systolic murmur heard best at $2^{nd}/3^{rd}$ L interspace; may decrease w/ inspiration
- Widely split S2; +/- S4 R-sided

PS – Diagnosis & Prognosis

- EKG: RAD, RAE, RVH
- ECHO: accurate diagnosis; TEE useful with suspected endocarditis of PV

- Treat predisposing conditions
- Balloon Valvuloplasty in symptomatic pt
- Valve replacement surgery

Pulmonic Regurgitation (PR)

- Most cases 2º pulmonary HTN (high pressures)
- Low pressure cases seen with carcinoid plaques, IE vegetations, or s/p surgery for ToF repair
- Trivial PR seen on routine ECHO is a normal variant

Pulmonic Regurge – s/s

- High JVP with prominent "*a*" wave
- Loud, split S2 with RV S3 and S4
- Low-pitched, cres-decres, diastolic murmur heard near $3^{rd}/4^{th}$ L interspace
- With pulm HTN – Graham-Steell murmur, which increases with inspiring and diminishes with Valsalva

PR – Diagnosis & Prognosis

- ECHO: colorflow doppler shows regurge
- MRI & CT: useful for complete imaging information; cath is confirmative

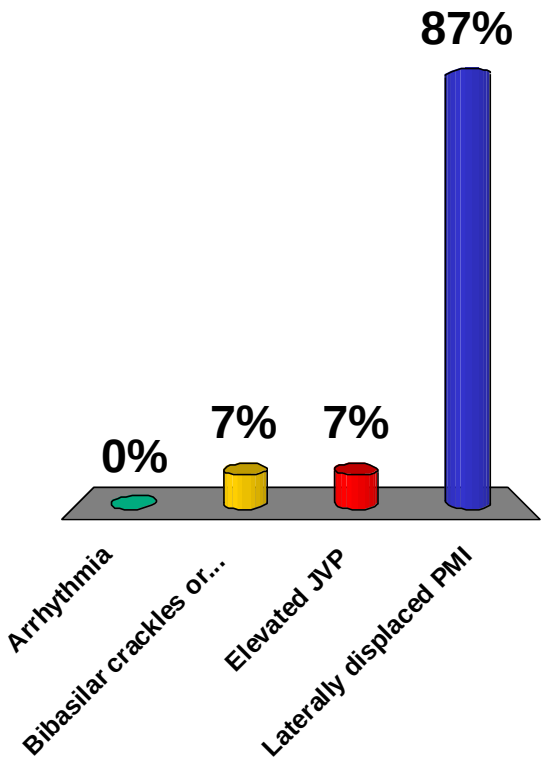
- First, treat primary cause (Pulm. HTN)
- Pulmonary valve surgical replacement is the definitive treatment

Management of Prosthetic Valves

- All mechanical valves require anticoagulation with warfarin/coumadin (NOT new agents)
- INR should be maintained at 2-3 (Mechanical mitral valves at 2.5-3.5, and add ASA 81 mg)
- Stop coumadin 5 days prior to elective surgeries and restart within 24 hours after surgery
- UF or LMW heparin may be used pre-operatively before surgery and post-op (after 48-72 hours) until INR > 2.

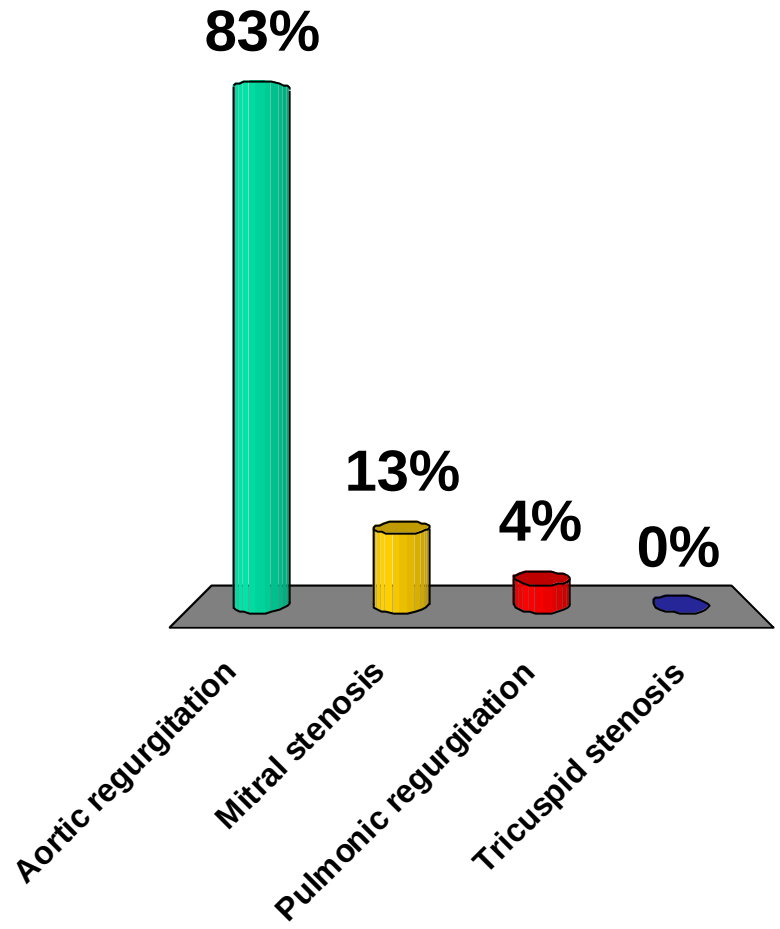
A 75 y/o male presents with chest pain, dyspnea and syncope. A harsh, IV/VI systolic murmur is heard over the right second interspace. What additional finding would you expect?

1. Arrhythmia
2. Bibasilar crackles or rales
3. Elevated JVP
4. Laterally displaced PMI



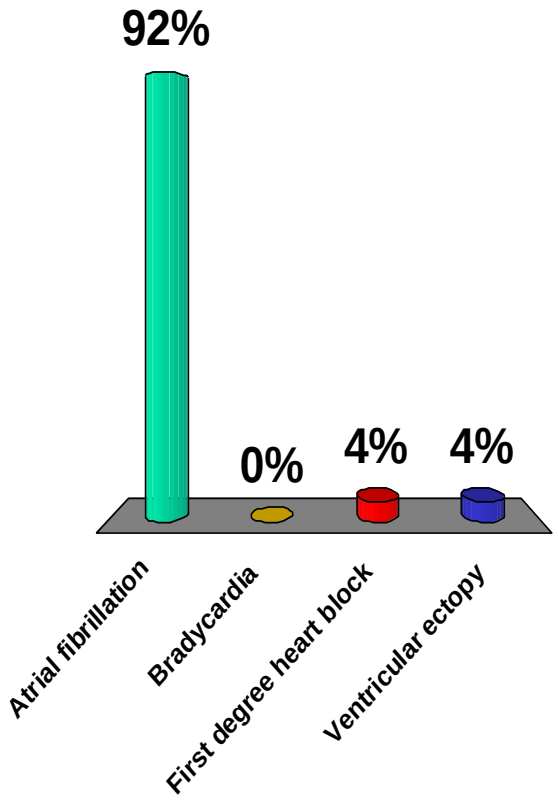
What valvular disorder is most commonly associated with Marfan's Syndrome?

1. Aortic regurgitation
2. Mitral stenosis
3. Pulmonic regurgitation
4. Tricuspid stenosis



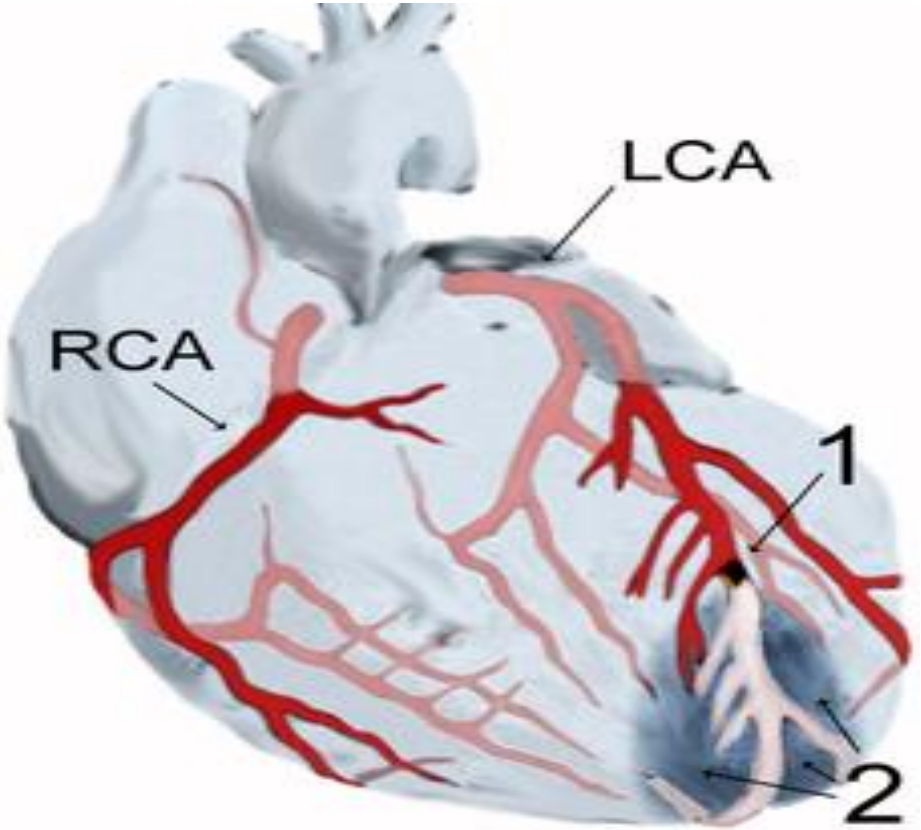
An 82 y/o female with a h/o rheumatic fever is found to have an opening snap on cardiac auscultation. Which of the following arrhythmias is most often associated?

1. Atrial fibrillation ✔
2. Bradycardia
3. First degree heart block
4. Ventricular ectopy



Let's change topics now!

CARDIOLOGY II: Ischemic Heart Disease



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Ischemic Heart Disease

- Angina (stable, chronic)
- ACS (Acute Coronary Syndromes)
  - Unstable Angina
  - Prinzmetal's Angina
- Acute MI
  - STEMI/non-STEMI
  - Q-wave/non Q-wave

IHD – Risk Factors: modifiable and most critical

- Tobacco - #1 preventable worldwide
- Diabetes Mellitus
- Hypertension
- Hyperlipidemia: high LDL, low HDL

IHD – Risk Factors: secondary, but may be very important, too.

- Increasing age
- Sedentary lifestyle
- Obesity (BMI > 30)/high abdominal girth (waist >40″M / 35″F)
- High stress/psychosocial factors
- Family history of early CAD
- Gender (male > female)
- Too few fruits/veggies
- ETOH excess

IHD – Risk Factors (other)

- Homocysteine
- Serum lipoprotein (a)
- Triglycerides
- C-reactive protein (hs CRP)
- Interleukin-6
- CD-40 ligand
- Myelopyroxidase

Stable Angina/Angina Pectoris

CHEST PAIN:

- Precipitated by stress/exertion
- Relieved rapidly by rest/nitrates

Stable Angina: History = dx

- Character or quality
- Location
- Radiation
- Duration
- Precipitating or relieving factors
- Effects of nitroglycerin
- Dyspnea = anginal equivalent

www.commons.wikimedia.org/wiki/Myocardial _Ischemia

Stable Angina – Physical Exam

- Commonly normal or non-specific
- May find: ↑ BP, S_3, arrhythmias
- DM-associated findings (retinopathy or neuropathy)
- Hyperlipidemia-associated findings (xanthomas, xanthelasma)
- HTN, hyperthyroid or PAD signs

Stable Angina – Diff Dx:

- MSS: bone, muscle, tissue injury/pain, (Tietze syndrome: costo-chondritis)
- Neuro: intercostal neuritis: zoster, DM
- GI: GERD, PUD, esophageal spasm
- Respiratory: pneumothorax
- Other Cardiac: pericarditis, MVP, MI, aortic dissection

Stable Angina - Evaluation

Labs

- CBC: anemia
- Lytes: glucose (DM); arrhythmias
- Lipids: cardiac risk
- Cardiac Markers: C(P)K-MB, Troponins
- PT/INR: prepare for anticoagulation
- CMP/LFTs: assess renal/hepatic function

Stable Angina - Evaluation

EKG:

- ~25% of resting EKGs are normal
- Classic: ST segment horizontal or down-sloping depression which resolves after pain subsides
- T-wave flattening or inversion *may* occur
- *Rarely* ST elevation due to coronary artery spasm (aka Prinzmetal's)

Stable Angina - Evaluation

Exercise Stress Test: most useful, non-invasive procedure

- Contraindication: Aortic Stenosis, rest pain
- Stop with: drop in BP, arrhythmia, increasing angina, >2-3mm ST-seg depression

Pharmacologic Stress Test: (when ambulation is difficult/impossible) use adenosine, dipyridamole, dobutamine (now Lexiscan/regadenoson) to simulate exercise and/or vasodilate vessels

Stable Angina - Evaluation

Myocardial perfusion scintigraphy (aka) Nuclear stress test:

- Thallium, sestamibi or tetrafosmin to show perfusion defects: scar or ischemia
- Add Stress ECHO to show wall motion defects, LV global and regional function (dobutamine w/o exercise)
- SPECT or PET for questionable results
- CT, MRI, EBCT used for specific cases

Stable Angina – Evaluation

CT: Ultrafast CT/EBCT: used to detect calcified plaques (aka calcium scoring); beware radiation and contrast

MRI: High resolution images *without* radiation. Uses gadolinium. Still slow, currently used for some non-urgent testing, but stay tuned . . .

Stable Angina - Evaluation

- Holter Monitor: 24 hour ambulatory EKG—arrhythmias and ST changes
- Event Recorder: long-term patient-activated ambulatory EKG
- Either/both useful in patients with "silent ischemia" (eg diabetics)

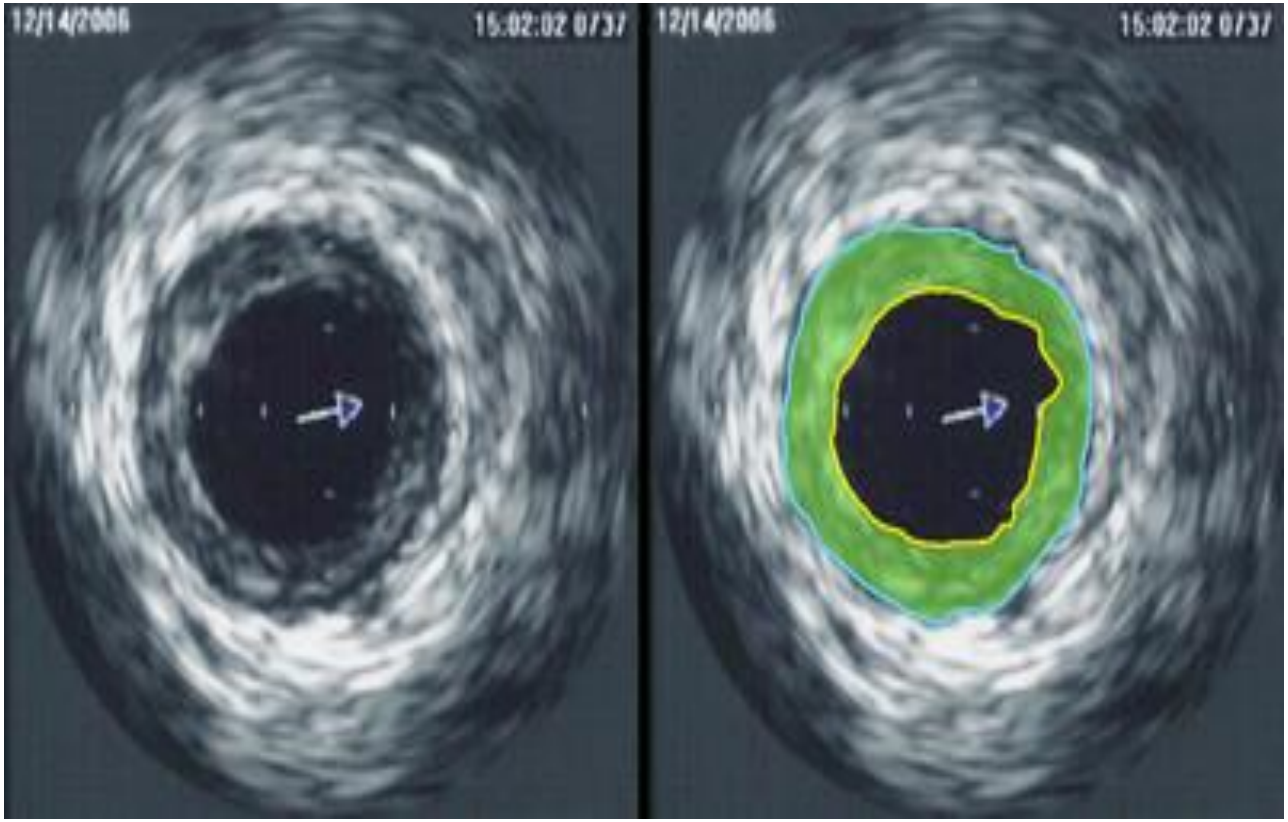
Stable Angina - Evaluation

Coronary Angiography: definitive diagnostic test for CAD; can be diagnostic and curative during one admission

- Invasive and expensive
- Used for CAD/Angina patients who have failed medical treatments to prepare for intervention

Intravascular ultrasound (IVUS) helpful for Left Main vessel lesions and coronary dissections

Intravascular Ultrasound (IVUS)



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Stable Angina - Treatment

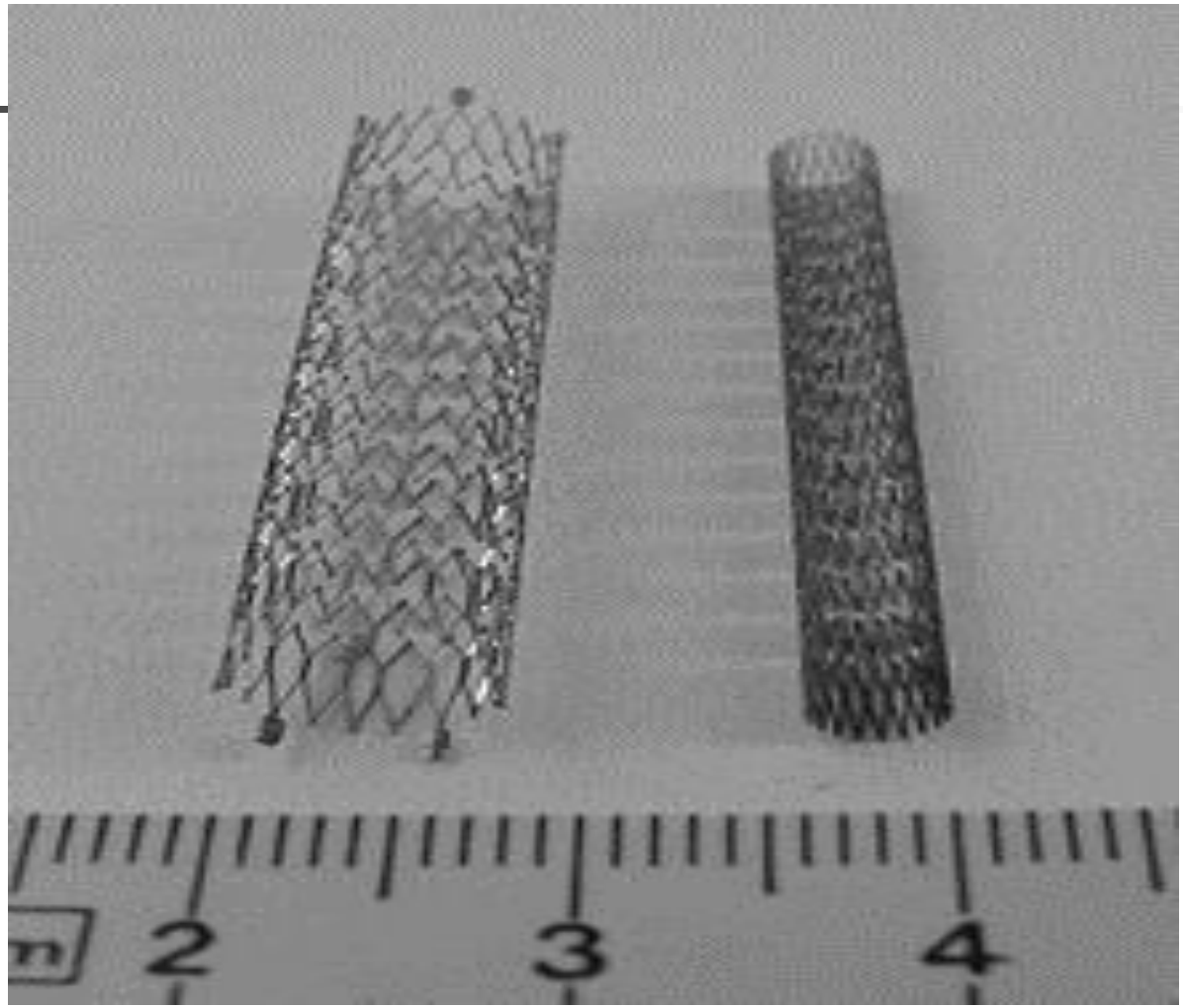
Nitroglycerin: drug of choice

- Sublingual acts in 1-2 min (spray ok)
- Decreases vascular tone, pre-load and after-load, and O2 demand
- Long-acting (for prevention of Angina): isosorbide di/mononitrates and transdermal patch; beware tolerance; remove night patch
- Side effects: headache, nausea, BP ↓

Angina – Treatments, cont.

- Revascularization:
- PCI: PTCA (percutaneous transluminal coronary angioplasty, aka "balloon angioplasty"), stents: bare metal or DES (drug-eluting stents)
  - Major limitation: restenosis
  - Less invasive
  - Faster recovery
  - Clopidogrel and ASA for 1 yr post-stenting

Two types of coronary artery stents



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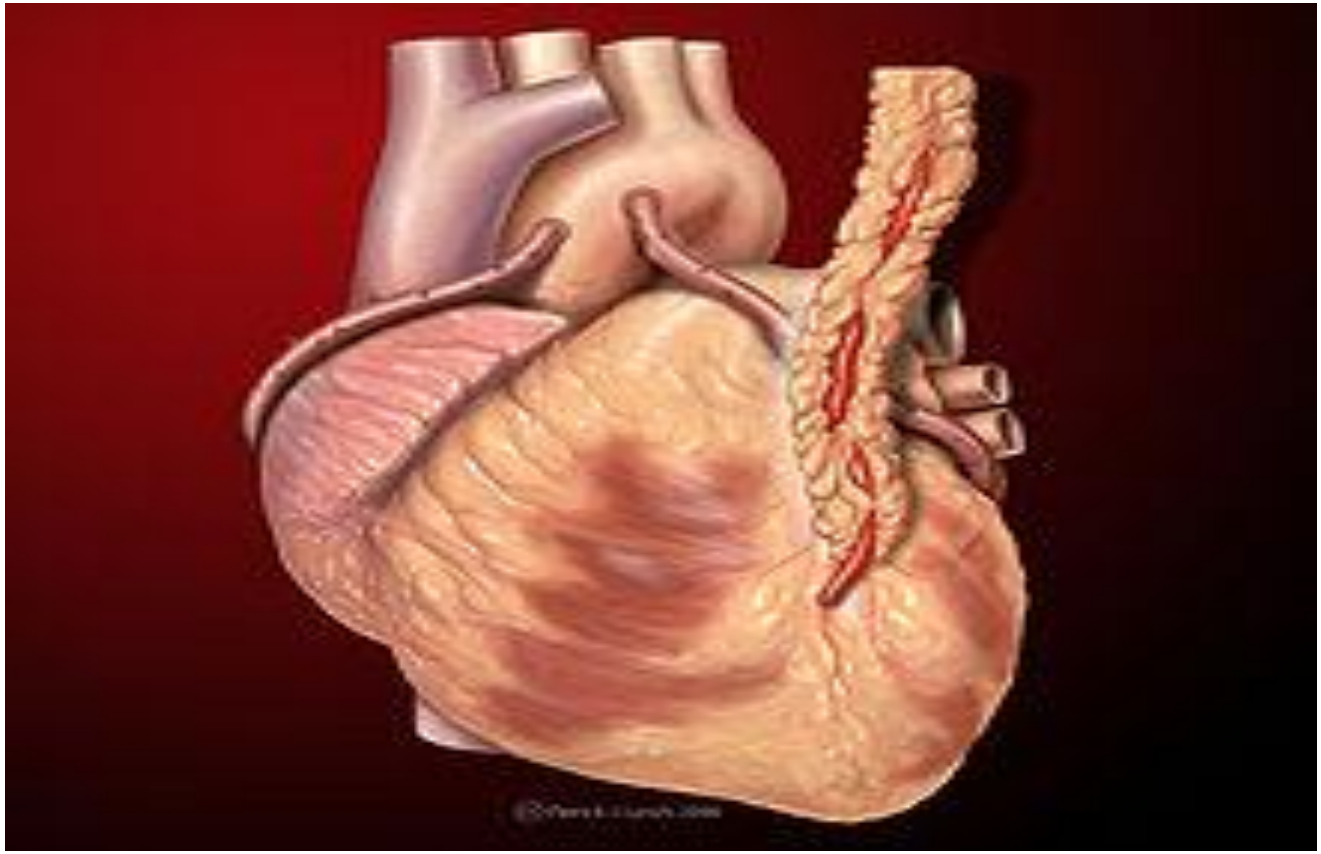
Angina Treatment – cont.

- Surgery: CABG (coronary artery bypass graft)
  - Best results in DM
  - Better for multivessel disease
  - Often used in large Left Main occlusion
  - Internal mammary arteries best grafts (saphenous vein and radial artery also)
  - LV function determines operative mortality

CABG utilizing saphenous vein graft



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Stable Angina - Prevention

Avoid provocative factors – cold, stress

Long Acting Nitrates – as discussed

Beta blockers – Proven to prolong life in post MI patients

CCBs – Both act to decrease O2 demand and allow for vasodilation

Ranolazine – New; no effect on HR, BP

ASA or clopidogrel – antiplatelet drugs

Risk reduction !!

Coronary vasospasm and Prinzmetal's/Variant Angina

- Chest pain occurring *without* usual precipitating factors: often AM, F>M, associated with arrhythmias
- EKG may show ST segment elevation
- Results from coronary vasospasm w/or w/out obstructive coronary disease
- May be induced by cocaine

Unstable Angina

- Now frequently grouped with "Acute Coronary Syndromes"
- Presents as ST- elevation (STEMI) vs. Non ST- elevation (Non-STEMI)
- Results of CK-MB and Troponins help determine if acute MI is present or not
- If negative, XST and discharge pt.

Acute Coronary Syndromes

General Measures

- Hospitalization
- Bed rest/limited activity x 24 hours
- Telemetry monitoring
- Supplemental O2 and sedation prn

Acute Coronary Syndrome: Non-STEMI

Thrombosis treatment: (anticoagulation and antiplatelet therapy; but NOT thrombolysis)

- ASA (81-325 mg) and heparin (UF or LMWH) STAT
- NTG and B-blockers
- Clopidogrel, prasugrel, ticagrelor, Glycoprotein IIb/IIIa inhibitors (eptifibatide or abciximab) then PCI

Acute Coronary Syndrome

Nitroglycerin

- First-line anti-ischemic therapy
- May use morphine if BP drops too low

B-blockers

- Oral or IV therapy; at discharge

Statins

- Start within hours/day(s) of ACS

Acute Coronary Syndrome

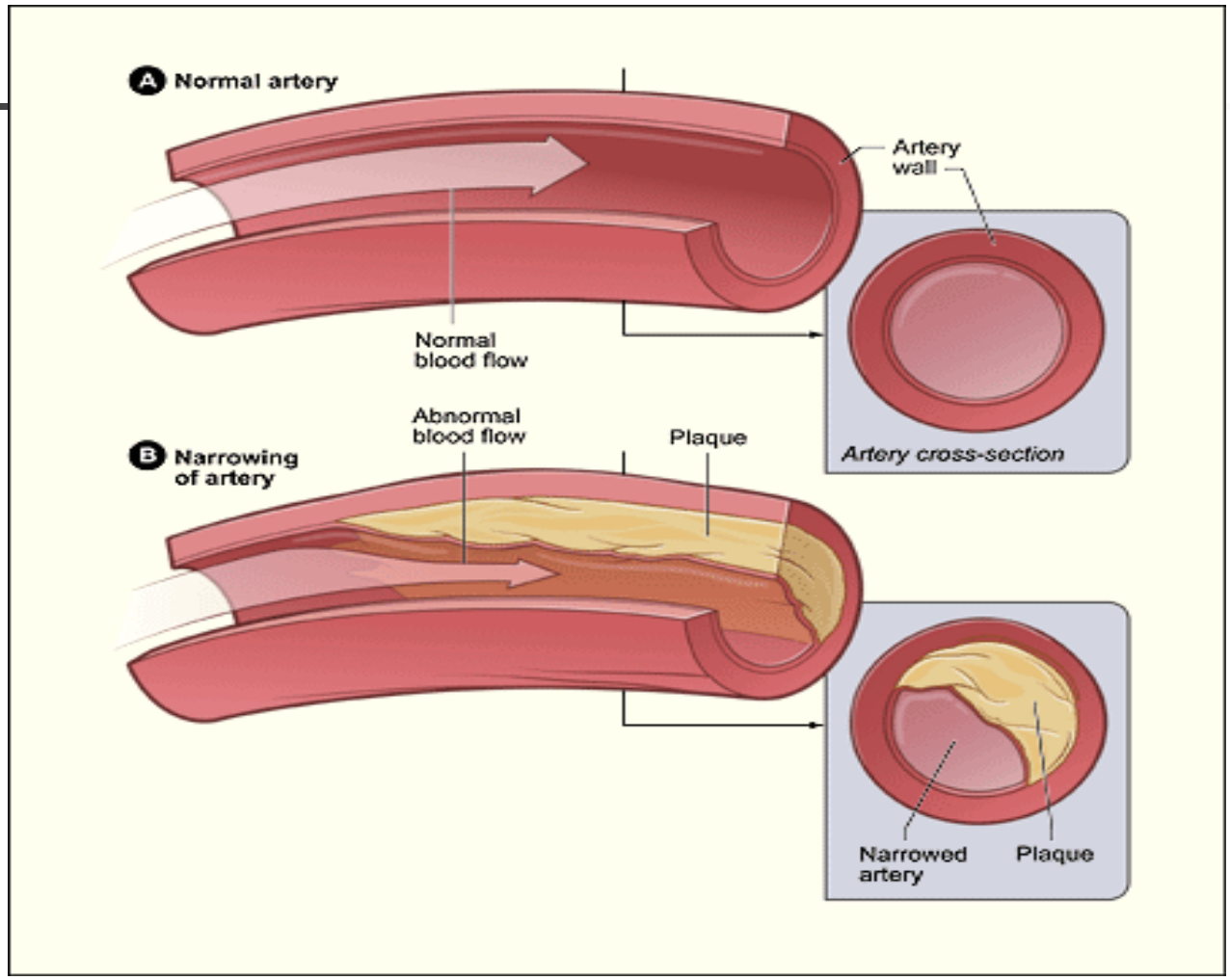
Catheterization and PCI for high-risk:

- Recurrent angina at rest
- Elevated troponin
- Low EF
- Hemodynamic instability
- Sustained VT
- Recent PCI or prior CABG

Acute Myocardial Infarction/MI--STEMI

- Sudden chest pain > 30 min.
- EKG shows ST elevation (STEMI) or (new) LBBB, and +/- evolving Qs
- Elevated CK-MB and Troponins
- Segmental wall motion abnormalities via ECHO at ED bedside
- Treat with immediate reperfusion (either PCI or t-PA w/in 3 hours)

A Common Cause of acute MI



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Acute STEMI - Symptoms

- Chest pain: worsening angina, early AM, at rest, NTG ineffective
- Dyspnea, diaphoresis, n/v, lightheaded
- Painless (1/3 of all MIs): women, elderly, diabetics
- Sudden death (50% occur before hospitalization): due to V-Fib

Acute STEMI - Signs

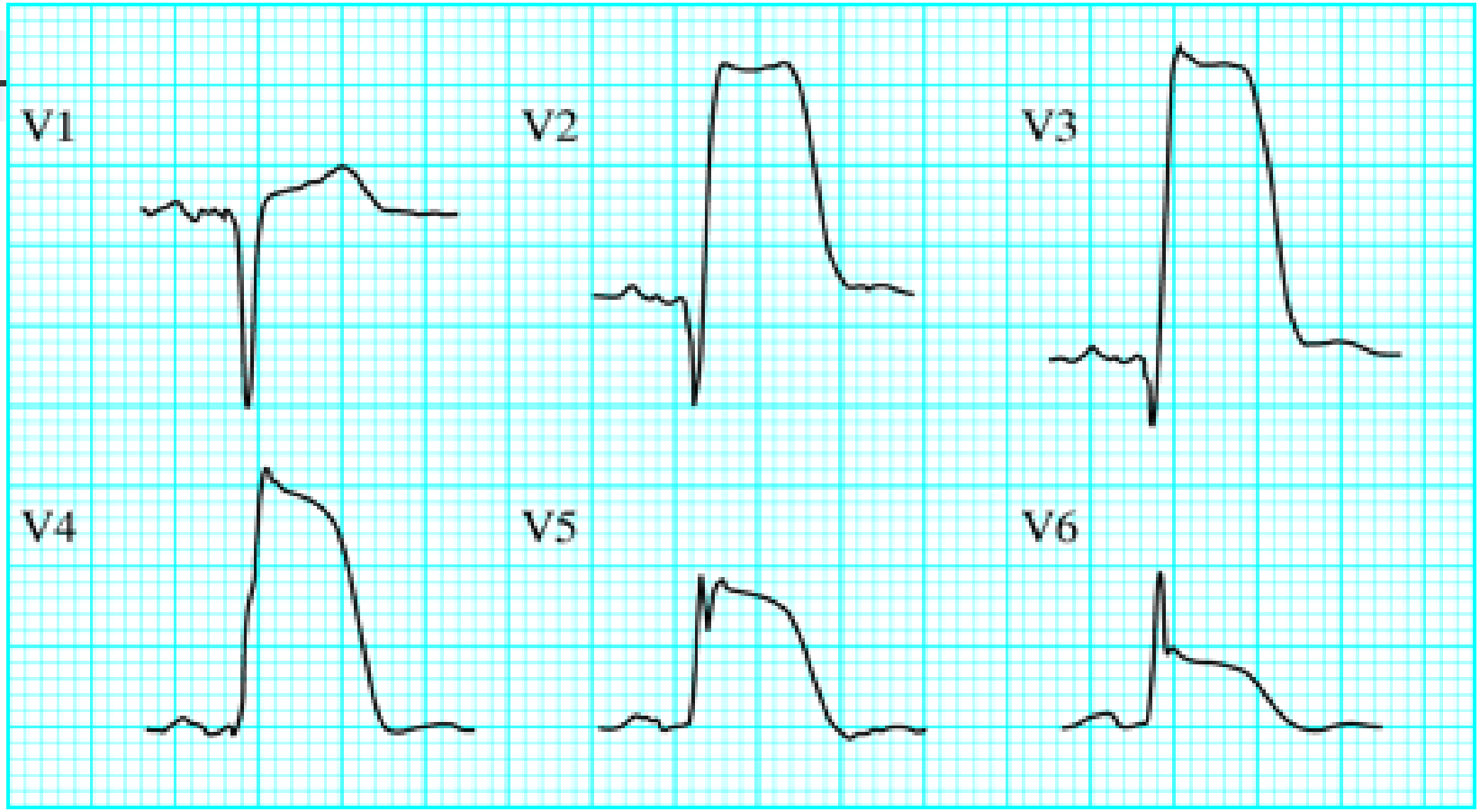
- General: anxiety and diaphoresis, possible low grade fever after 12 hours
  (with elevated WBC on CBC)
- Lungs: Tachypnea, rales
- Heart: displaced PMI, JVD, S4/atrial gallop, tachy/brady, hyper or hypotensive
- Extremities: possible cool/cyanotic indicating low cardiac output; no edema early

Acute MI - diagnostics

- Labs: CK-MB, Troponin I and T

  -both positive in 4-6 hours; CK-MB good for re-infarction dx.; Troponins stay elevated 5-7 days; elevated WBCs
- EKG: hyperacute T wave to ST-elev. to Q wave to T wave inversion; *new* LBBB
- CXR: CHF findings later; R/O aortic dissection via mediastinal widening

Classic "tombstoning" ST-elevations



Sadley, personal file

Acute MI - diagnostics

- ECHO: look for wall motion abnormalities (hypokinetic areas)

- Scintigraphic Studies: MRI w/ gadolinium; technetium, thallium, radionuclide injection of traceable radioactive substances (use these tests AFTER revascularization as they are 'time-intensive')

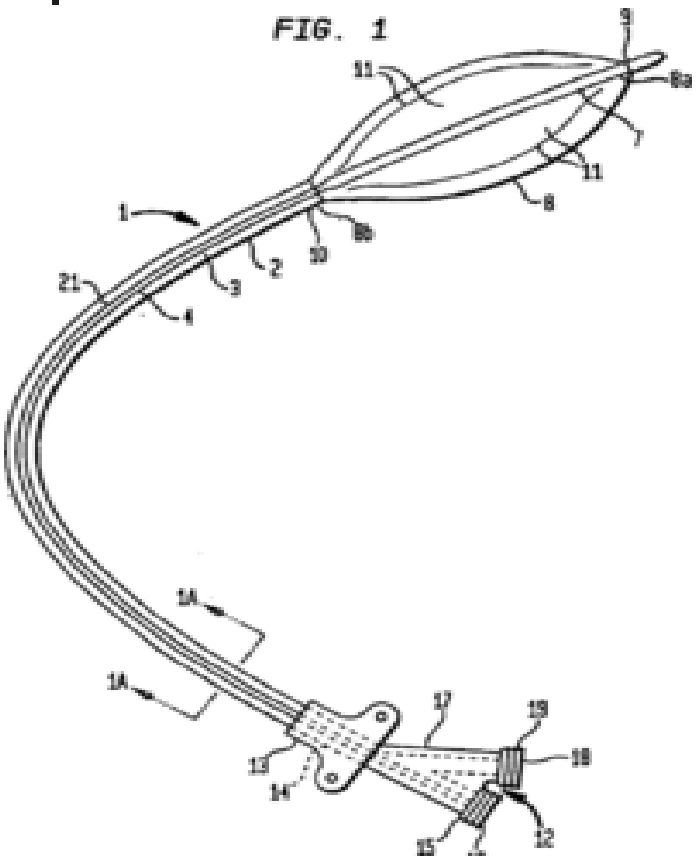
Acute STEMI - treatment

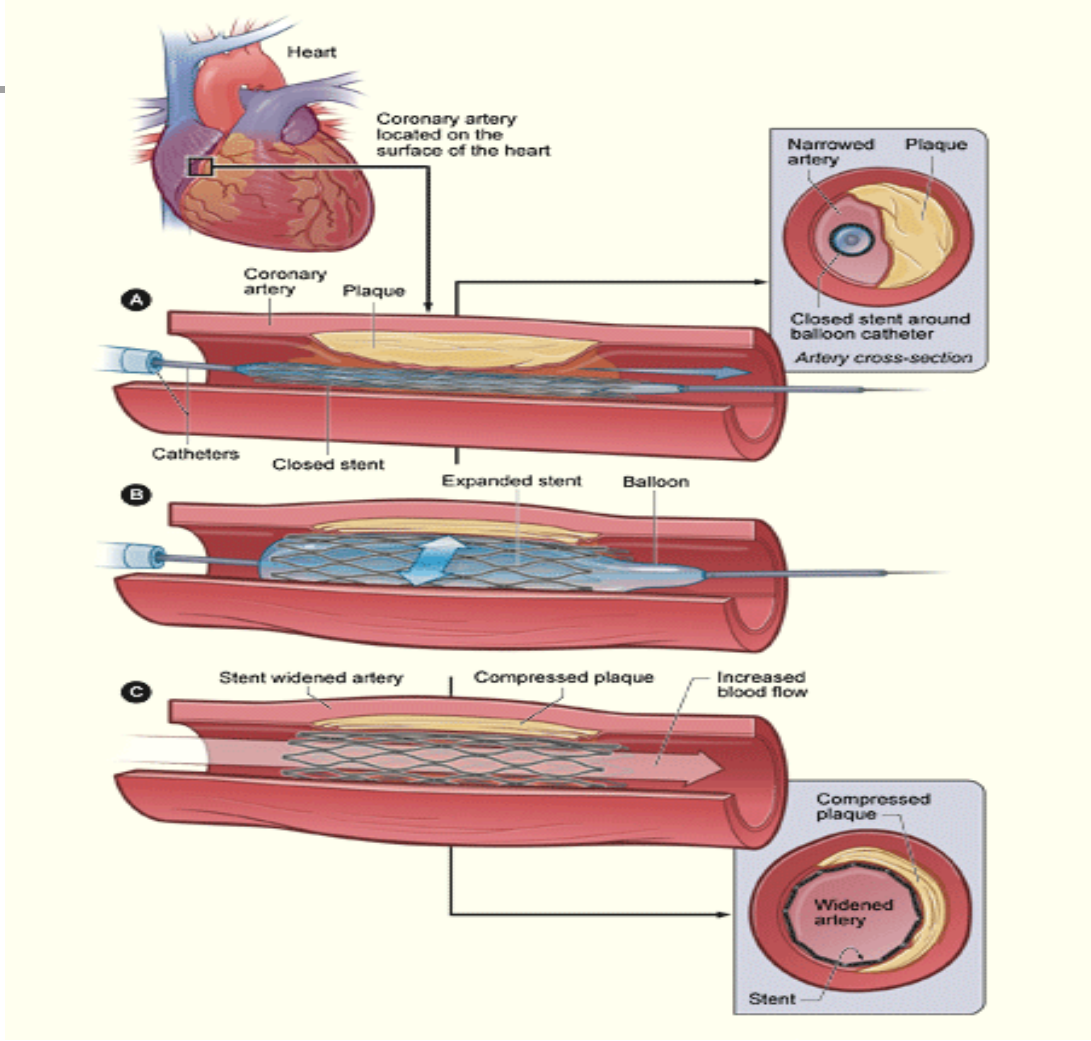
- ASA – (1/2 or whole) 325 mg chewed
- Clopidogrel (if ASA allergic) now for all
- Thrombolytics: best used within 3-12 hrs of STEMI or with LBBB (eg t-PA/ alteplase, reteplase, etc.)(danger: bleeding)
- t-PA NOT recommended for non-STEMIs
- Heparin (UF or LMWH)

Acute STEMI - Treatment

- Immediate coronary angiography w/ Percutaneous Coronary Intervention
- #1 today = PTCA and stenting (to maintain patency of vessel) w/ heparin and GP IIb/IIIa inhibitors
- CABG in some cases/problems; internal mammary art., saphenous vein or radial artery are grafts

Percutaneous Transluminal Coronary Angioplasty = PTCA





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Acute MI - hospitalization

- CCU/telemetry with O2 24-72 hours
- Pain relief with NTG or morphine
- Beta blockade (not with HF)
- ACE-I especially with low EF and CHF
- Antiarrhythmics: only with sustained VT
- d/c with ASA, B-blocker, clopidogrel, +/- statin

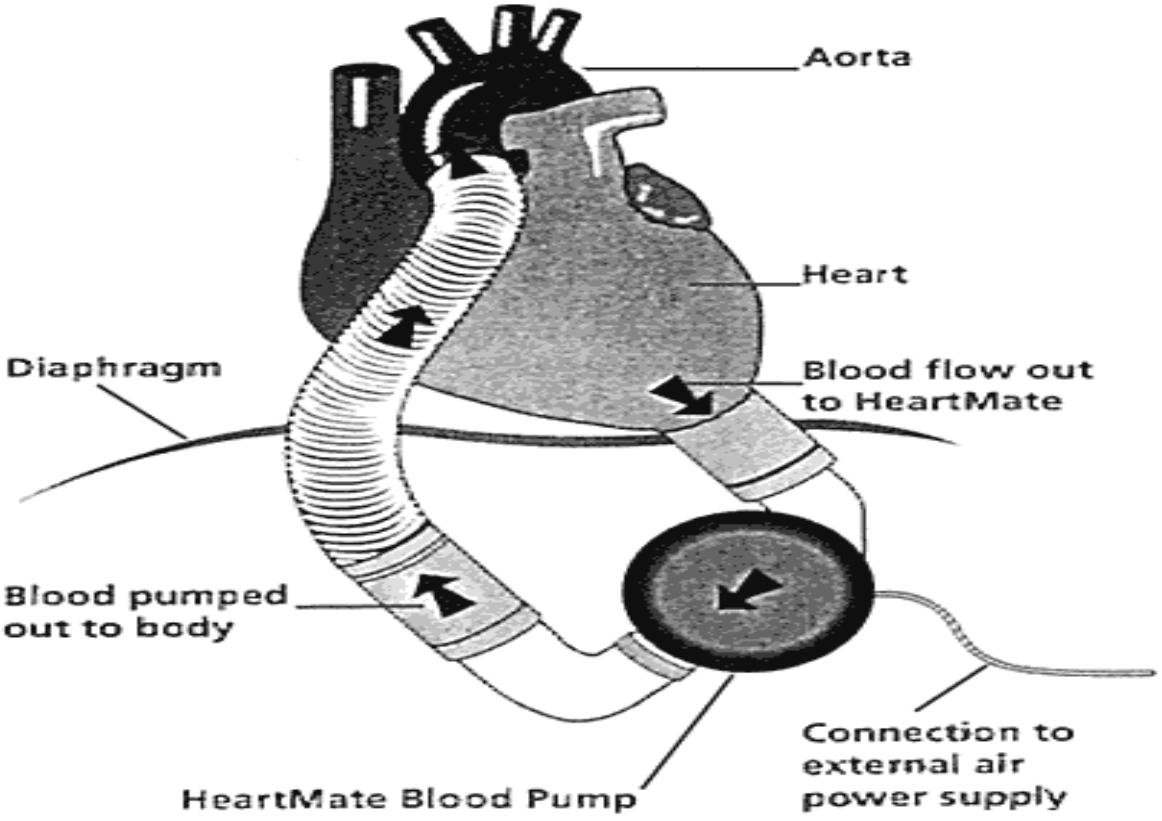
Acute MI - complications

- Ischemia: medical tx then cath/PCI
- Arrhythmias: medical therapy or pacemakers for conduction blocks (IWMI most often @ with arrhythmias)
- LV Failure (mild): O2, diuretics, morphine, NTG; monitor patient
- Pericarditis (Dressler syndrome) – autoimmune; 1-12 weeks post MI

AMI – severe complications

- Hypotension/shock: fluids, hemodynamic monitoring via PAC; may try IABP (intra-aortic balloon pump)
- Dopamine – best pressor agent
- Surgically implanted LVAD (LV assist device)

Left ventricular assist device (LVAD)



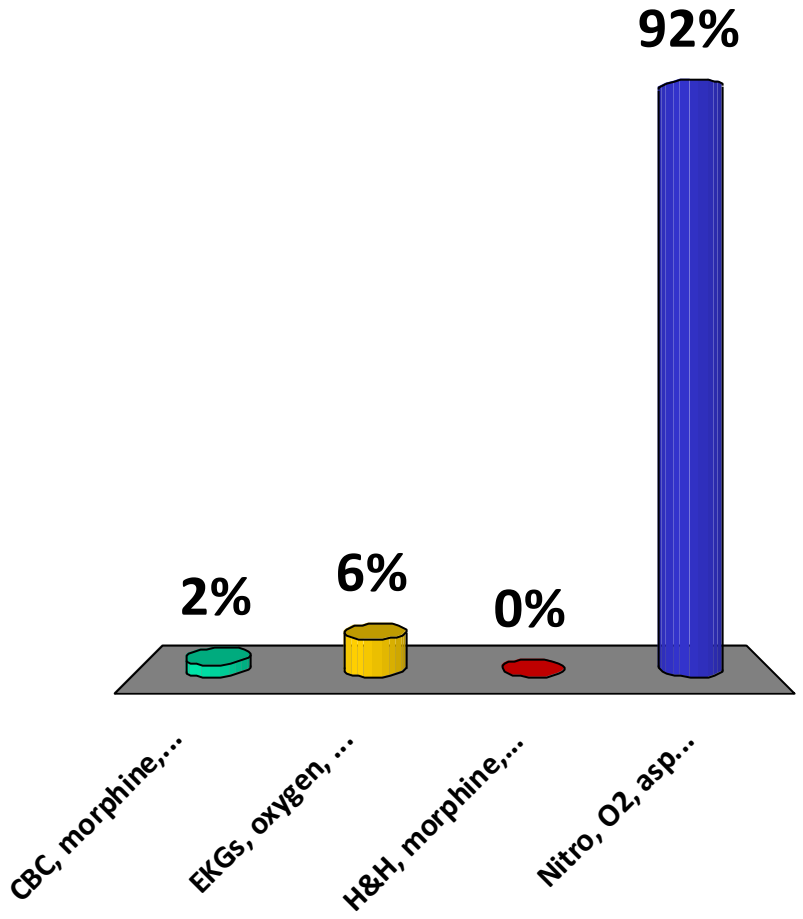
www.commons.wikimedia.org/wiki/Left_ventricular_assist _device

AMI – severe complications

- Papillary muscle rupture: AMI or IWMI, 3-7 days post MI; new systolic murmur
- Myocardial rupture: anterior wall, older females; 2-7 days post MI = death
- LV aneurysm: ST elevations persisting beyond 4-8 weeks post MI

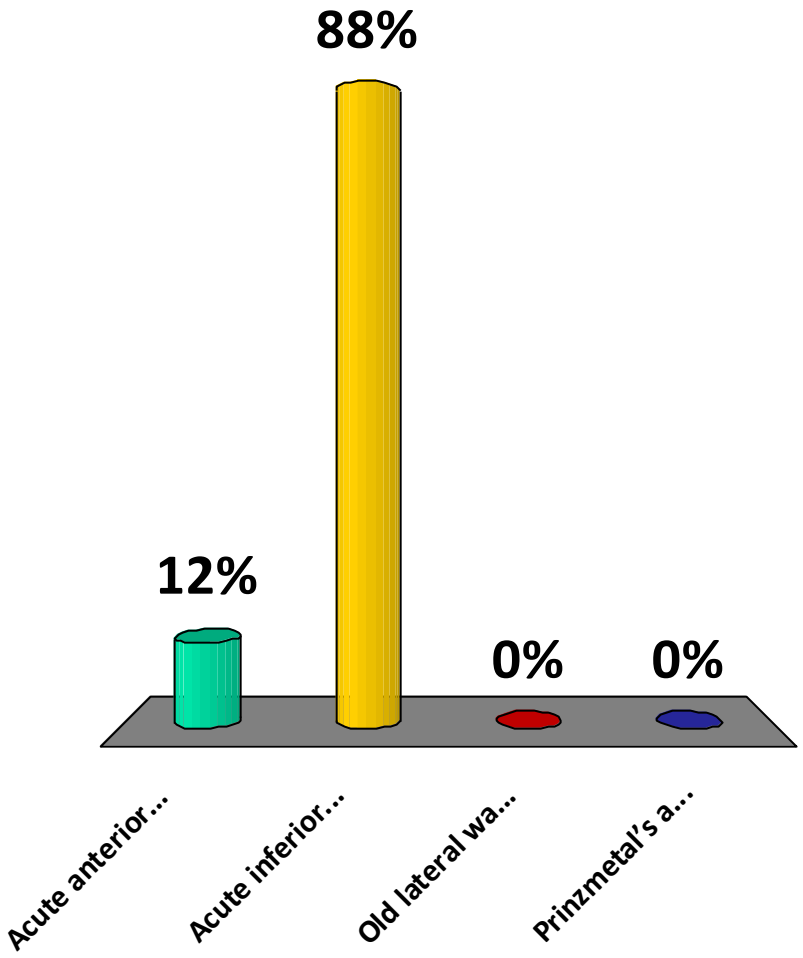
A 49 y/o male presents c\o substernal chest pain, radiating to his neck/jaw. Which of the following would be ordered first?

1. CBC, morphine, nitro
2. EKGs, oxygen, LFTs
3. H&H, morphine, aspirin
4. ✓ Nitro, O2, aspirin



A 62 y/o female with a h/o CAD presents with vague discomfort in her chest and back. An EKG reveals 2 mm ST-segment elevations in II, III, and AVF. What is the most likely diagnosis?

1. Acute anterior wall MI
2. ✓ Acute inferior wall MI
3. Old lateral wall MI
4. Prinzmetal's angina



Okay, time to change topics!

CARDIOLOGY II: Congenital Heart Disease

Adult Presentations

Congenital Heart Disease Case Study

- A 35 y/o female with a h/o "heart murmur as a child", presents as a new pt. for a complete H&P. She offers no complaints and PE reveals only a III/VI holosystolic, harsh murmur heard best at the 3rd & 4th interspaces of the LSB. What will her ECHO most likely show?

Choices:

A. LAE with thickened mitral leaflets
B. Left to right shunt with small VSD
C. LVH with calcified aortic valve
D. MVP with leaflet vegetations present

Answer:

A. LAE with thickened mitral leaflets (MS)
B. **Left to right shunt with small VSD**
C. LVH with calcified aortic valve (AS)
D. MVP with leaflet vegetations present (MR)

Congenital HD Overview

- Only 2% of total adult HD
- 85% of infants reach adulthood
- Total number of pts surviving to adulthood is increasing
- 1 million adults surviving with Congenital HD in US

Categories of Congenital Heart Disease in Adults:

- Patients without surgical corrections
- Patients with *curative* surgery or non-surgical interventions
- Patients with *palliative* surgery or non-surgical interventions

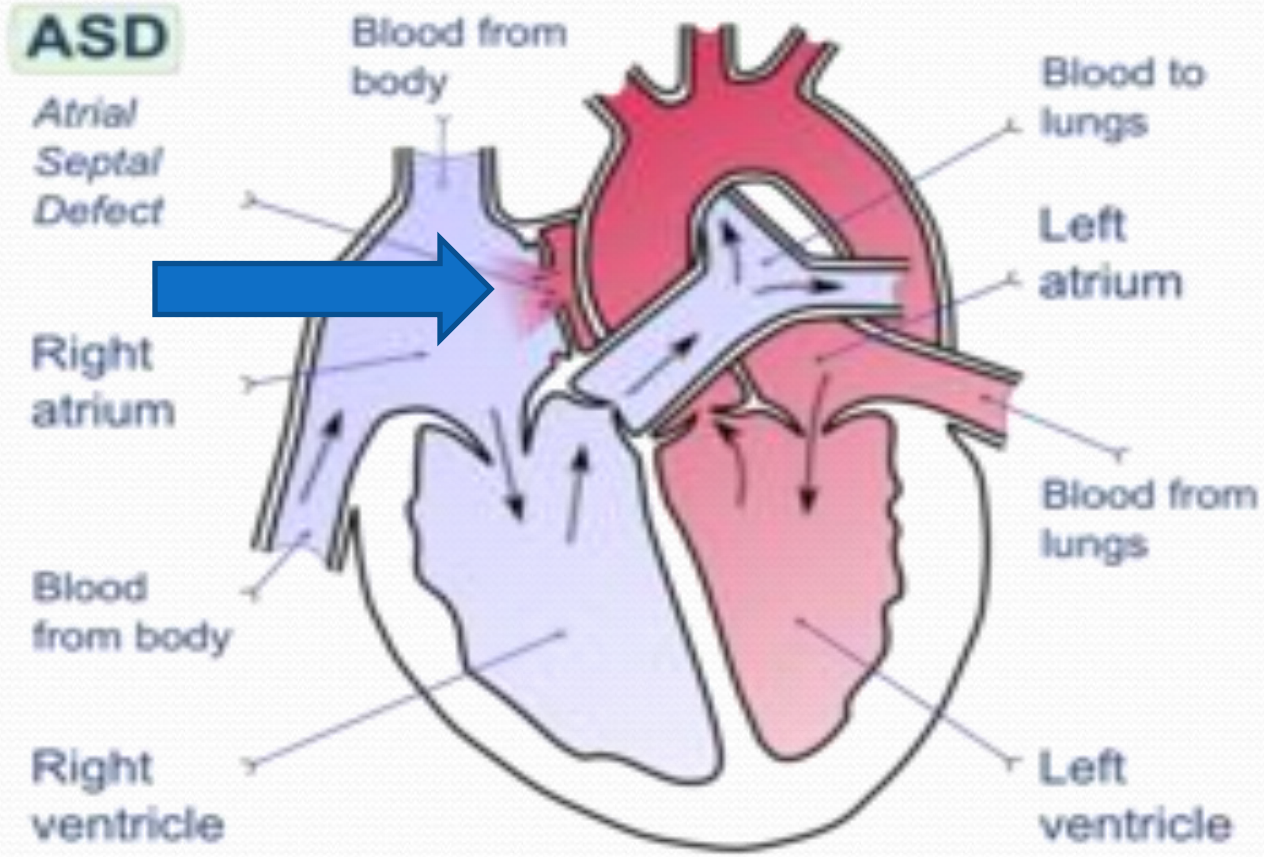
5 Important Congenital Heart Disease diagnoses in Adults:

- ASD – Atrial septal defect
- Coarctation of the aorta
- PDA - Patent ductus arteriosus
- Tetralogy of Fallot
- VSD - Ventricular septal defect

Atrial Septal Defect (ASD)

- 10% of CHD, size varies, PFO (patent foramen ovale) most common (80%); persistent ostium secundum
- Hx: asymptomatic with small/medium shunt
  - Over 30 y/o: dyspnea and CP
  - Over 50 y/o: atrial arrhythmias especially atrial fibrillation; RVF

Atrial Septal Defect (ASD)



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Atrial Septal Defect

Physical Exam:

- RV lift
- Widely split and fixed S2 into A2,P2
- II-III/VI systolic ejection murmur at L 2nd or 3rd interspace (aka pulmonic area)

Atrial Septal Defect

EKG:

- RBBB, RAD, RVH

CXR:

- Dilated pulmonary arteries
- Increased pulmonary vascularity
- Enlarged RA and RV

Atrial Septal Defect

ECHO:

- Definitive diagnosis w/ Doppler shows RV dilation and L to R atrial shunt (new: saline bubble contrast ECHO shows reverse shunt better)

Prognosis & treatment: All ASDs that show evidence of RV overload should be closed surgically.

Small shunts: no treatment/observe/normal life

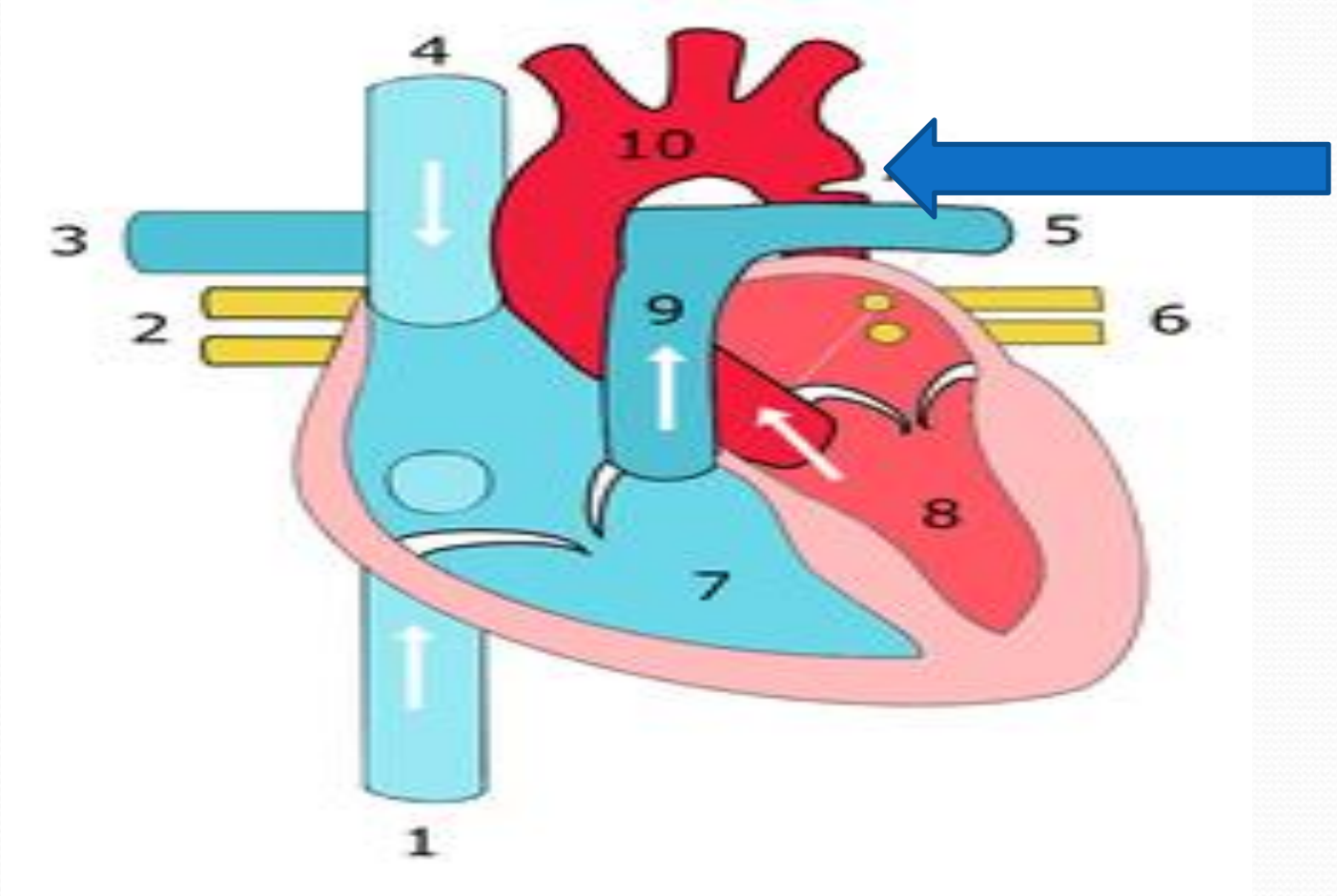
Large shunts: surgical repair, patches, percutaneous closure devices

Beware: paradoxical emboli leading to stroke/TIA!

Coarctation of the Aorta

- Localized narrowing of the aortic arch just distal to L subclavian artery
- A cause of secondary HTN in the young
- Bicuspid aortic valve in 50% of pts, and increased risk of cerebral berry aneurysm

Coarctation of the Aorta



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Coarctation of the Aorta

Hx: asymptomatic until sequellae of HTN appear as LV failure, CVA

PE: **absent or weak femoral pulses** with delay of palpable femoral pulse

- **HTN in arms but normal or low in LE**, which is exaggerated with exercise
- Late systolic ejection murmur-posterior or continuous murmur if collateral flow

Coarctation of the Aorta

EKG:

- LVH

CXR:

- Scalloping/notching of ribs
- Dilated L subclavian artery
- Poststenotic aortic dilation
- Aortic shadow shows "3" sign (notch in 3 = area of coarctation)

Coarctation of the Aorta

ECHO:

- confirmatory test; cardiac cath provides gradient info before surgery

Prognosis: Cardiac failure in older adults

- surgery is curative: endovascular stent good
- untreated adults die by 50 y/o due to aortic rupture, CVA, aortic dissection

Patent Ductus Arteriosus (PDA)

- Failure in closure of embryonic ductus
- Results in persistent shunt, connecting L pulmonary artery to aorta
- PDA treated/closed in neonates with indomethacin administration (**rare in adults**)
- Large shunts cause pulmonary HTN (aka Eisenmenger's physiology)

Patent Ductus Arteriosus

Hx: Asymptomatic unless HF/Pulm HTN

PE:

- Widened pulse pressure w/ low DBP
- Hyperdynamic PMI
- Harsh, continuous, ***machinery-like murmur*, +/- thrill, at L 2nd ICS**
- In large shunts, toes may be cyanotic/clubbed

Patent Ductus Arteriosus

EKG: normal w/ possible LVH

CXR: normal or LVH, LAE; pulmonary artery, aorta, and LA are prominent

ECHO/Doppler: helpful, but MRI, CT, and Cardiac Cath provide details

Patent Ductus Arteriosus

Prognosis:

- Large shunts cause mortality early
- Small shunts can be complicated by CHF
- Infective endocarditis risk requires abx prophylaxis
- Surgical ligation is curative; percutaneous approach preferred

Tetralogy of Fallot

Classic features:

- Ventricular-septal defect (VSD)
- Right Ventricular Hypertrophy (RVH)
- RV Outflow obstruction (aka PS)
- Overriding/dilated aorta (<50%)
- Right-sided aortic arch is common (25%)

Tetralogy of Fallot

- Most adults have prior surgical history consisting of outflow patch and VSD closure (Blalock shunt initially)
- Patients are generally asymptomatic but require abx prophylaxis for endocarditis

Tetralogy of Fallot

PE: possible decreased UE pulse on side of Blalock surgery

- Look for JVP increase, *a wave* increase
- May have S3 gallop (right-sided)
- Residual VSD may be present
- Blalock shunt may cause a continuous murmur

Tetralogy of Fallot

EKG: RVH, RAD; after repair: RBBB

- Check width of QRS annually via EKG to reduce risk of SCD

CXR: **Classic "*boot-shaped heart*"**

- Prominence of RV
- Concavity in the RV outflow tract
- Enlarged, right-sided aorta

Tetralogy of Fallot

ECHO: establishes the diagnosis

Prognosis: most adults have had sx repair

- 20 yrs post-op, 10-15% need reoperation, usually for severe PR
- All patients need abx prophylaxis
- Arrhythmias common after age 45

Ventricular Septal Defect (VSD)

- Most VSDs have closed in childhood
- In adults most VSDs are in the membranous septum
- Results in L to R shunt unless associated with RV hypertension
- Presentation depends on size of VSD

Ventricular Septal Defect

Hx: Asymptomatic; large shunts – CHF

PE: Small shunts result in greater gradient

- Loud, harsh, holosystolic murmur along L sternal border (3rd/4th ICS)
- Systolic thrill is common (IV-VI/VI)
- Cyanosis may occur in late stages

Ventricular Septal Defect

EKG: normal or Ventricular Hypertrophy: (R/L/bi)

CXR: normal or enlarged pulmonary arteries and increased pulmonary vasculature with large shunts

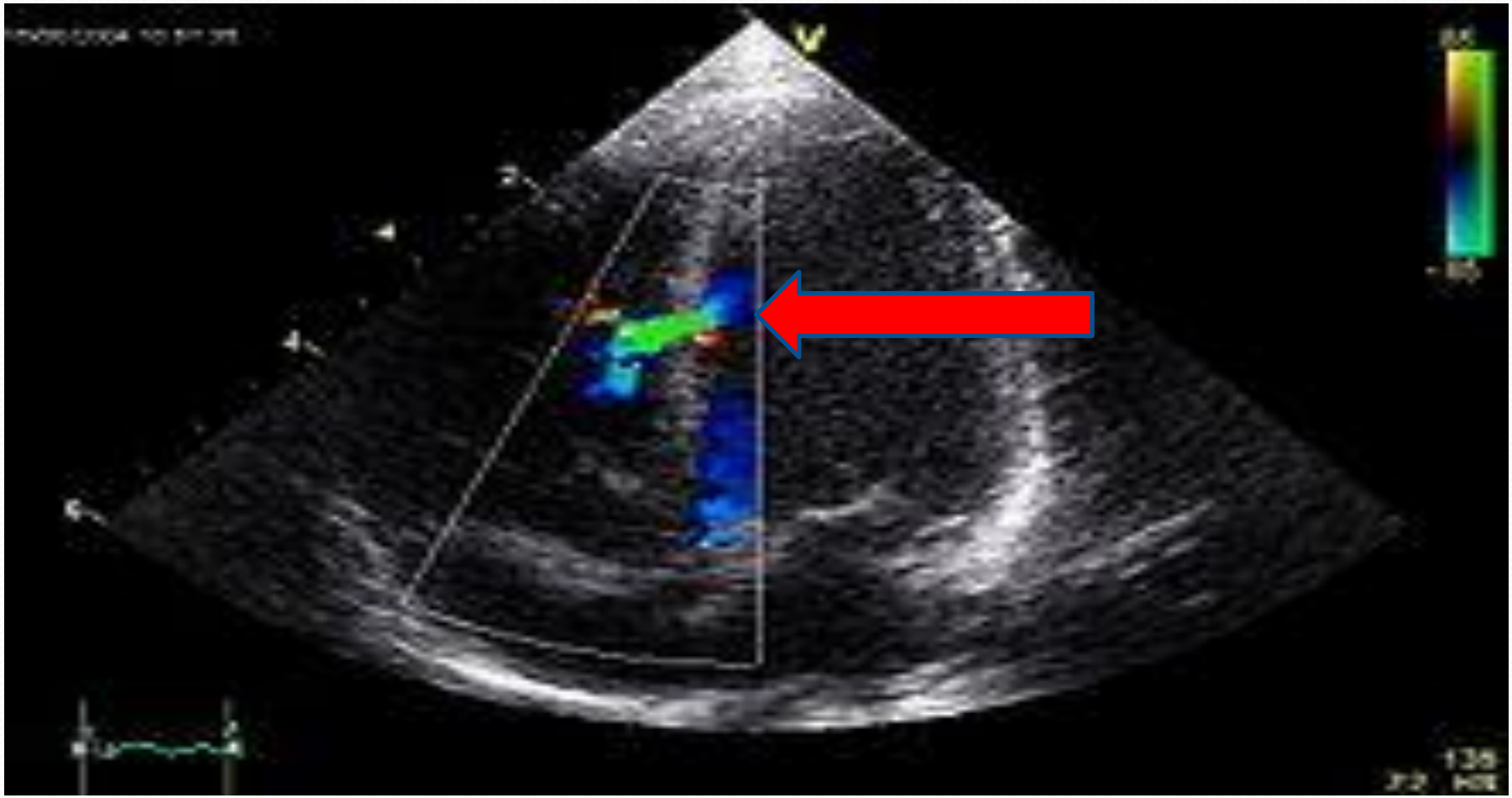
Ventricular Septal Defect

ECHO: demonstrates chamber enlargement and defect anatomy

Prognosis: small defect results in normal life expectancy; abx prophylaxis is mandatory

- Large shunts – CHF; survival < 40 y/o

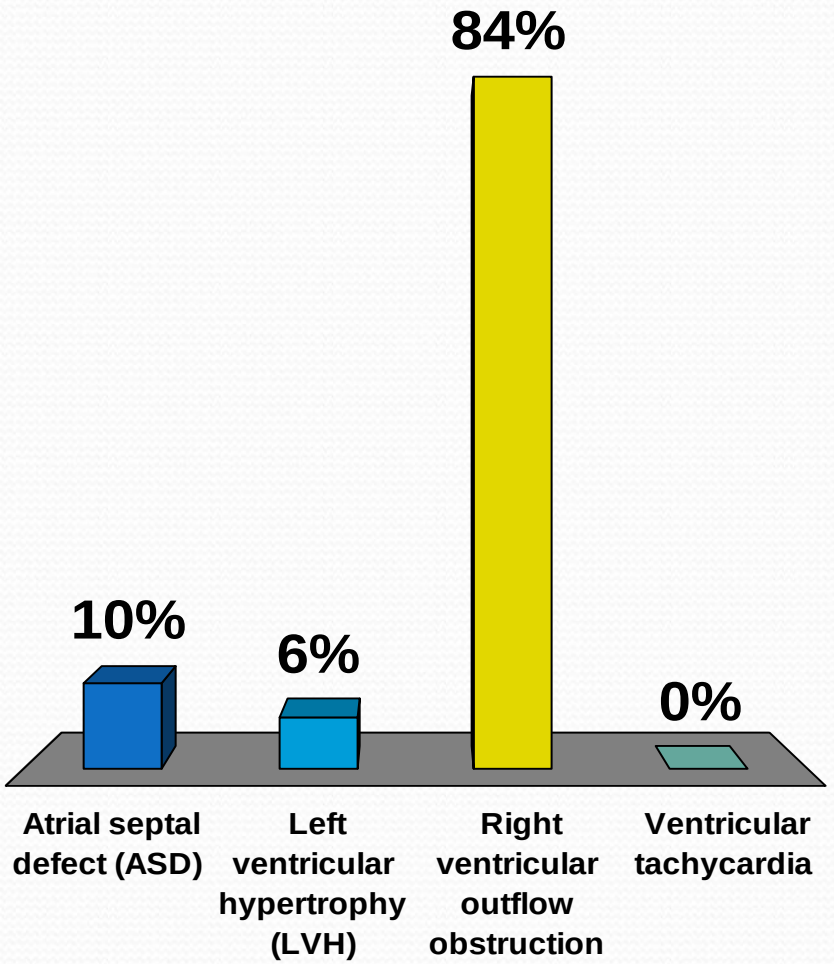
VSD ECHO showing L to R shunt



www.commons.wikimedia.org/wiki/Echocardiogram

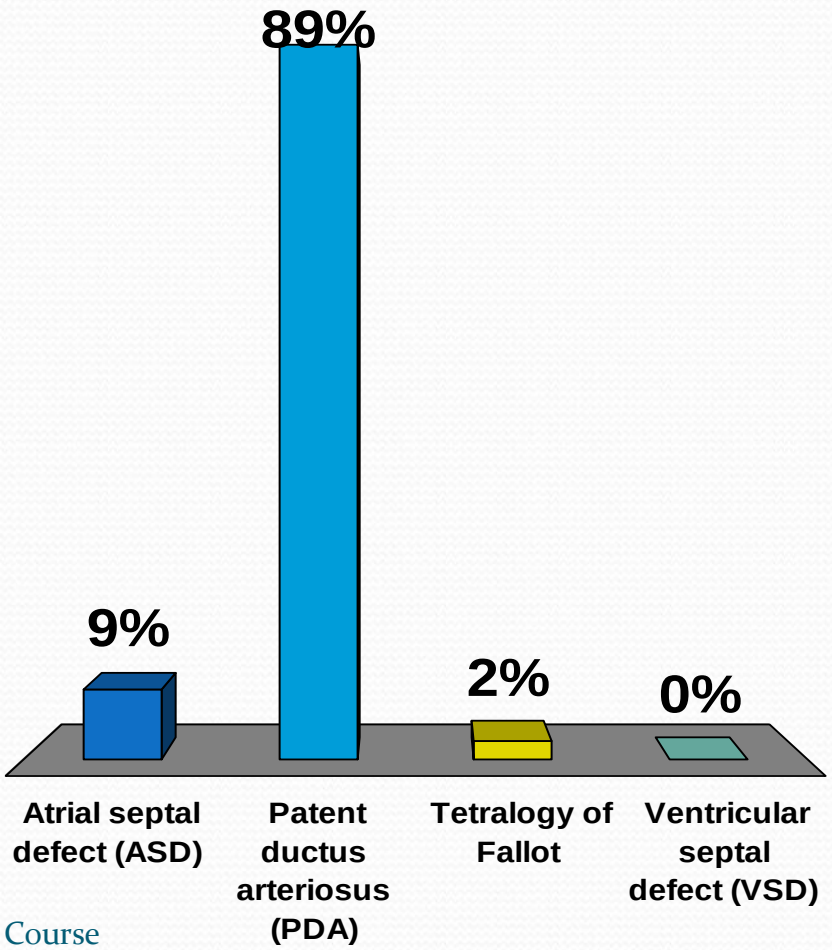
Which of the following is one of the four classic features of Tetralogy of Fallot?

1. Atrial septal defect (ASD)
2. Left ventricular hypertrophy (LVH)
3. ✓ Right ventricular outflow obstruction
4. Ventricular tachycardia

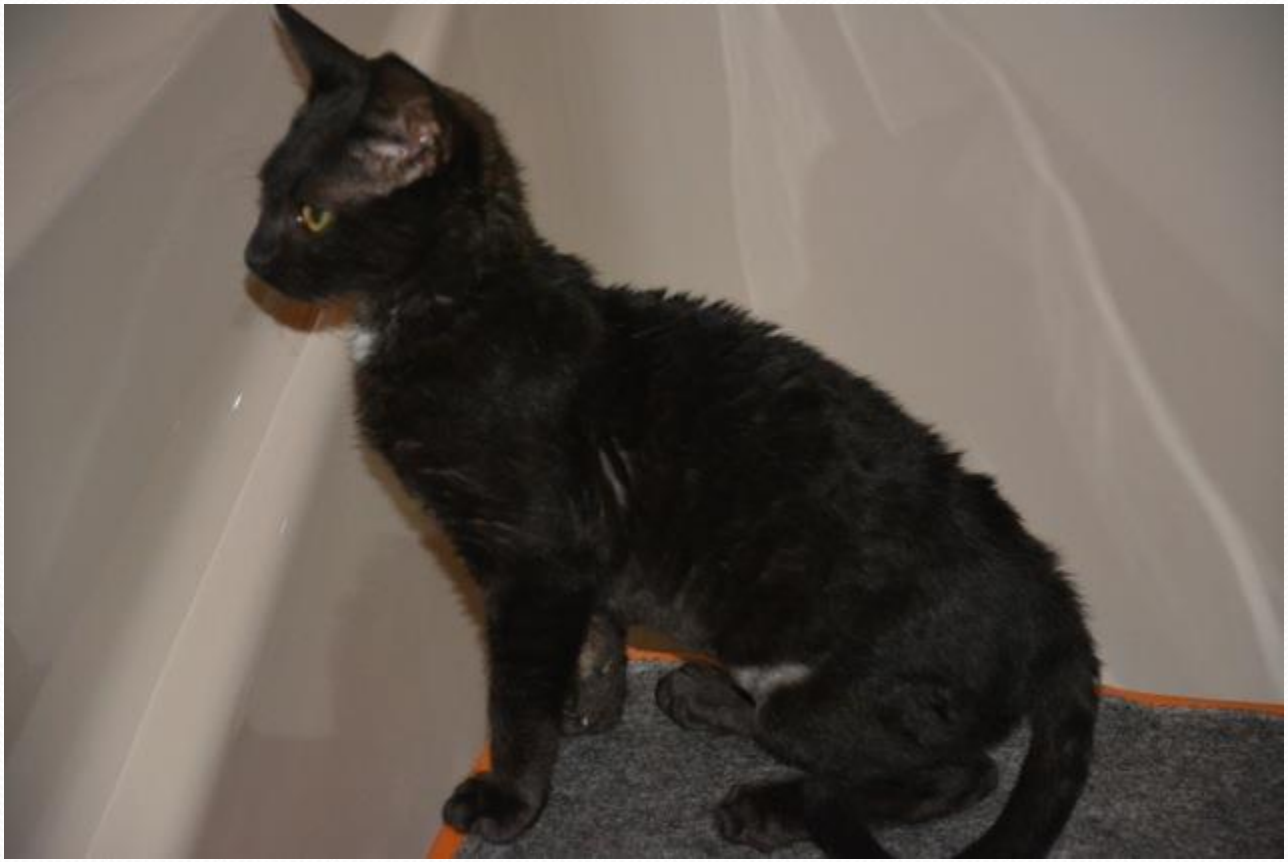


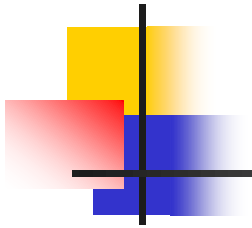
Cardiac auscultation of a 34 y/o male reveals a continuous, rough, “machinery-like” murmur, heard best in the first and second interspaces of the LSB. What is the diagnosis?

1. Atrial septal defect (ASD)
2. ✓ Patent ductus arteriosus (PDA)
3. Tetralogy of Fallot
4. Ventricular septal defect (VSD)



One more topic change in Cardio





CARDIOLOGY II: Vascular Disease

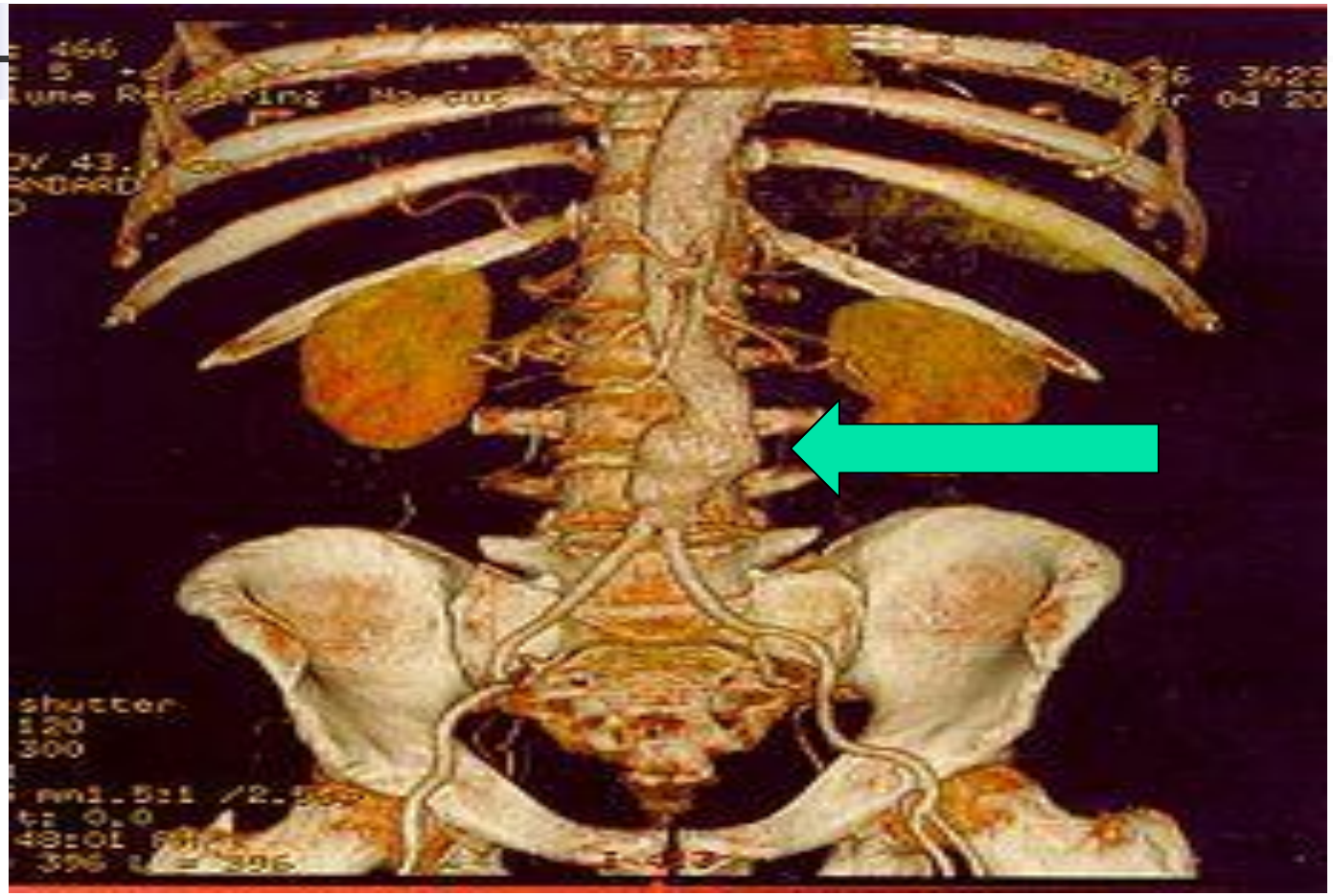
Vascular Disease

- Aortic aneurysm/dissection
- Arterial embolism or thrombosis
- Chronic/acute arterial occlusion
- Giant cell arteritis and Polymalgia Rheumatica
- Peripheral vascular disease
- Phlebitis/thrombo-phlebitis
- Venous thrombosis
- Varicose veins

Abdominal Aortic Aneurysm

- Abdominal AA most common (90%)
- 90% of these originate below the renal arteries
- Aortic diameter >3 cm (normal = 2cm)
- Aneurysms rarely rupture until >5cm
- Most aneurysms are asymptomatic
- Male : female = 4:1 ratio

Abdominal Aortic Aneurysm (infrarenal)



www.commons.wikimedia.org/wiki/Abdominal _aortic_aneurysm

AAA – signs/symptoms

- Asymptomatic – routine PE/incidentally (CT)
- 80% of 5cm aneurysms palpable on PE
- Often associated with LE aneurysms or LE occlusive disease (25%)
- Severe abd/low back pain, pulsatile mass & hypotension = rupture

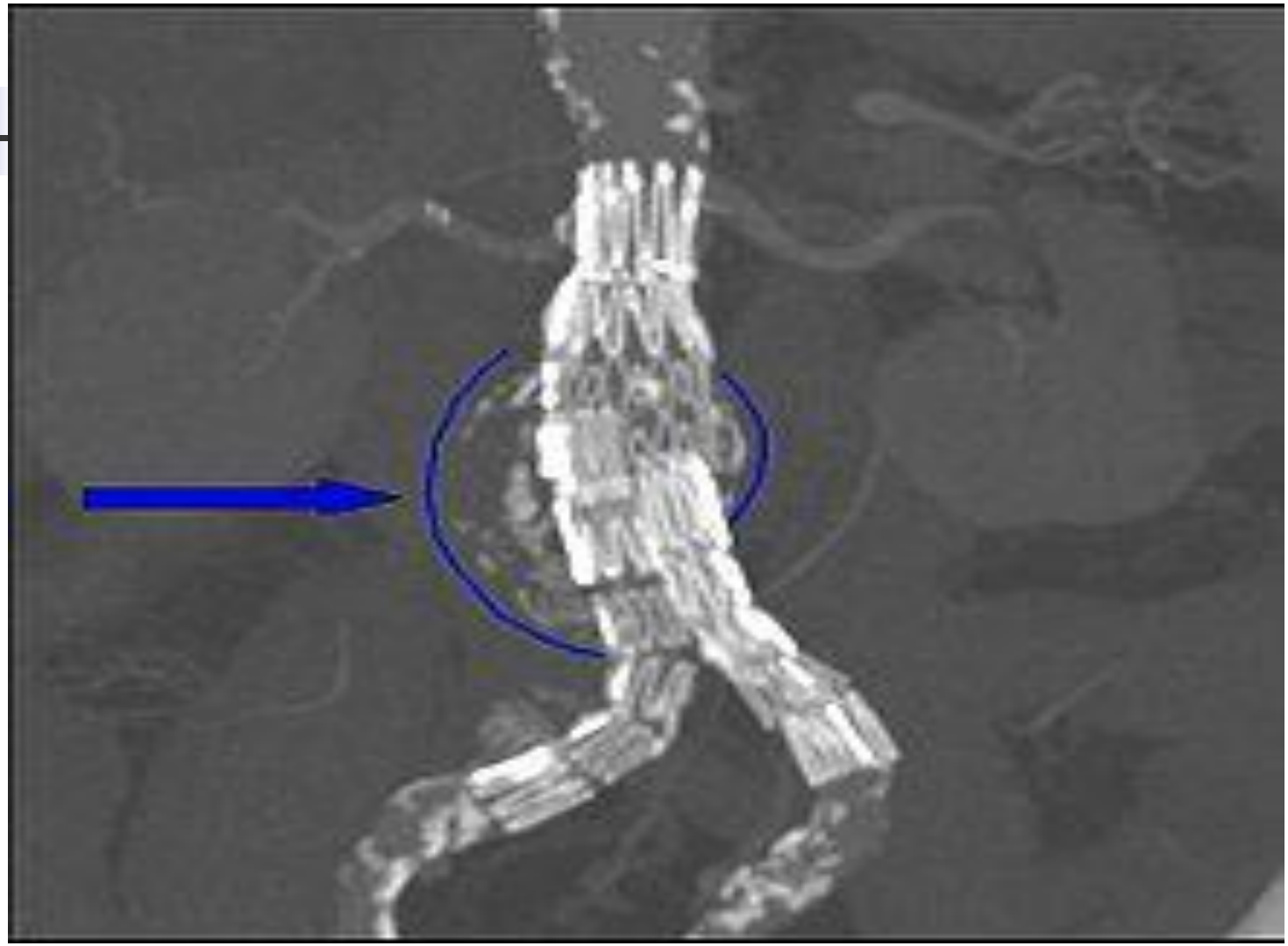
AAA - diagnosis

- Labs: EKG, creatinine, H&H, X-match
- Imaging: Abd US = initial screening study of choice
- Annual US for aneurysms > 3.5 cm
- Contrast-enhanced CT best prior to sx or when near 5 cm in size
- May use MRI if contrast is prohibited

AAA – treatment & prognosis

- B-blockade pre-op to reduce cardio complications
- Elective sx > 5.5 cm; poor risk > 6 cm
- Endovascular repair with "stent grafts" is good surgical procedure
- 1-5% mortality post-op

Endovascular repair of AAA



www.commons.wikimedia.org/wiki/File:Abdominal_aortic_aneurysm

Aortic Dissection

- Most common aortic catastrophe!
- Cause = intimal tear ⇨ false lumen between media and adventitia
- Commonly occur in asc/dec. thoracic aorta due to torque of heart beat
- Risks = HTN (80%), Marfan's, pregnancy, bicuspid aortic valve
- 90% mortality at 3 months

Aortic Dissection – s/s

- Sudden, excruciating, searing or ripping pain in the chest or upper back (85%)
- Pain may radiate to neck, abd, groin
- HTN at presentation
- PE: peripheral pulses and BP may be diminished or unequal
- AR diastolic murmur possible

Aortic Dissection: diagnosis

- EKG: normal or LVH
- CXR: widened mediastinum
- Mutiplanar CT scan of chest and abdomen = best test
- TEE, Angiography, MRI all okay, but often slower and not readily available

Aortic Dissection -- treatment

- STAT, aggressive BP control: Beta blockade (labetalol) to reduce LV ejection force; (esmolol if asthmatic)
- IV nitroprusside to lower BP if needed
- Pain relief with morphine
- SURGERY ! ! ! But still high mortality!

Arterial Embolism/Thrombosis

- Acute limb (LE) ischemia: embolic, thrombotic, or traumatic
- Most emboli arise from the heart (e.g. A-fib.)
- S/s related to location, duration of ischemia, and collateral flow present
- PE should focus on pulses, motor, and sensory systems

Arterial Embolism – s/s

- Six "Ps" of acute ischemia:
  - Pain (early)
  - Paresthesias (early)
  - Pallor
  - Pulselessness
  - Poikilothermia (aka varying temperature)
  - Paralysis

Labs and Studies

- Doppler of effected area reveals little or no blood flow distal to blockage
- Angiography, MRA, CTA first in cases with mild symptoms; otherwise STAT to OR

Arterial Embolism – acute treatment

- Heparin (unfractionated)
- t-PA via catheter < 3 hours(w/no neuro deficit)
- Emergent embolectomy via balloon catheter

<u>Complications:</u>

- Foot drop (due to peroneal nerve ischemia)
- Compartment syndrome

Arterial Thrombosis

- Most commonly results from chronic, atherosclerotic occlusive disease
- Smoking, polycythemia, dehydration, hypercoagulable states all increase risk of thrombus formation
- Most common s/s = intermittent claudication (cramp-like pain with exercise)

Arterial Thrombosis - labs

- Doppler ankle-brachial index (ABI): <0.9 (<0.5 suggest severe dz; 1.0-1.2 = Normal)

- CT/MR Angiography used to determine anatomic location of disease prior to surgery

Arterial Thrombosis - tx

- If mild, risk factor reduction, cilostazol (Pletal) (an antiplatelet drug) first
- Endovascular: angioplasty and stents
- Surgical interventions: bypass grafting
- Thromboendarterectomy: removal of plaque (in common femoral artery)

Acute Arterial Occlusion

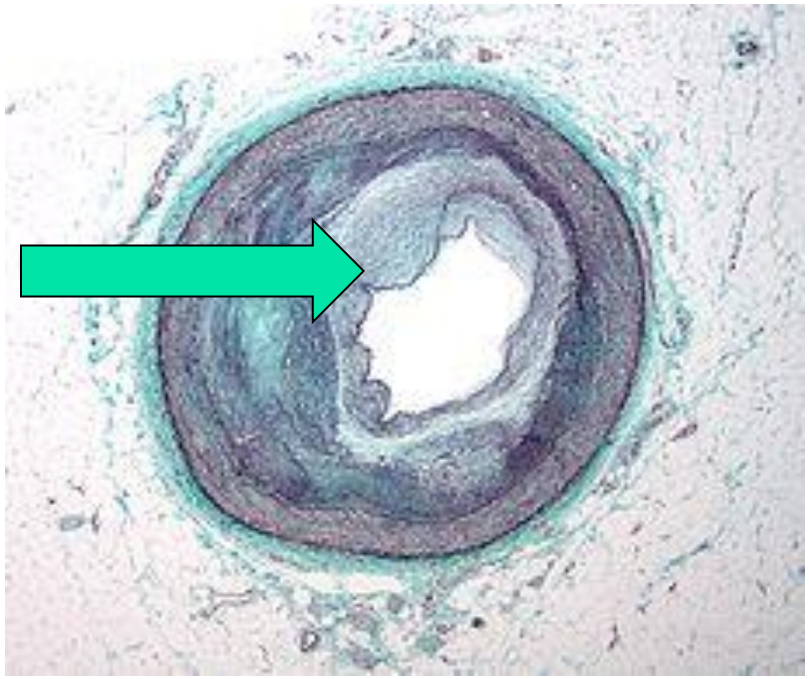
- Most common cause = atherosclerosis
- Systemic disease commonly found in arteries with turbulent flow and low sheer stress
  - Carotid bifurcation
  - Infrarenal aortic
  - Iliac, superficial femoral, tibial in LE

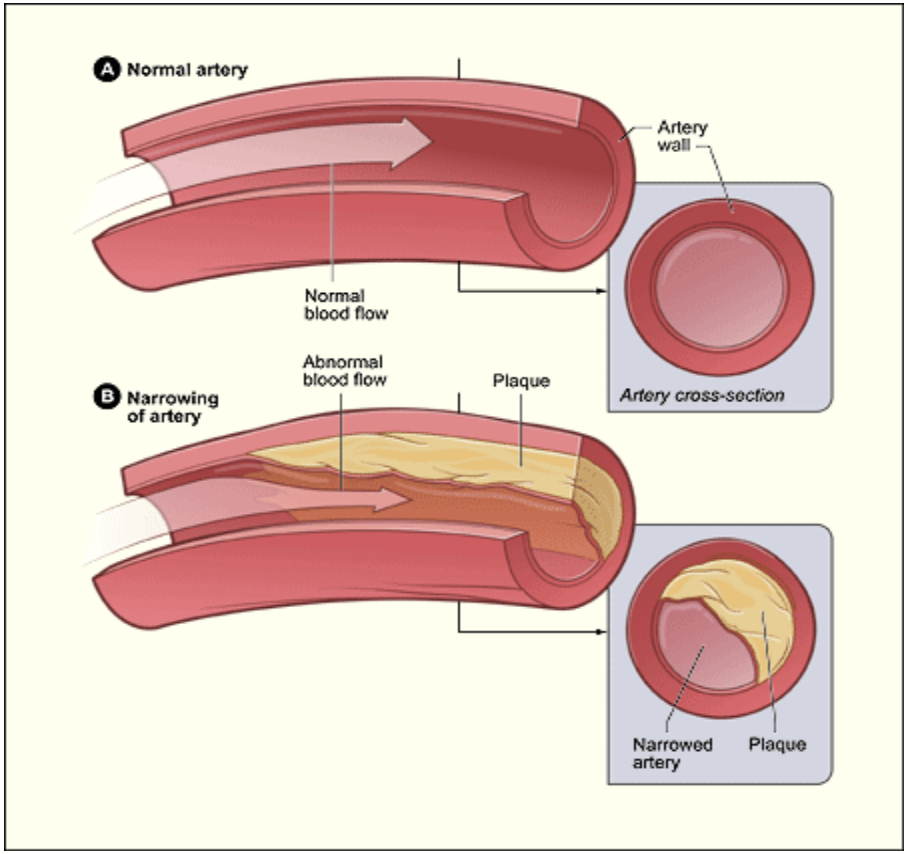
Arterial Occlusion - Carotid

- Carotid stenosis = 25% of strokes
- TIA: complete neuro resolution < 24hr
- S/S: sudden weakness, aphasia, vision loss (emboli to retinal artery resulting in unilateral blindness "amaurosis fugax"); mid-cervical bruit (not reliable)
- Dx: Duplex US; MRA/CTA if details needed
- Tx: medical: ASA and clopidogrel

  surgical: heparin and CEA or angioplasty/stenting via percutaneous route

Carotid Endarterectomy





www.commons.wikimedia.org/wiki/atherosclerosis

Carotid Artery Dissection

- Carotid artery dissection (classic triad):
  - CVA or TIA
  - Unilateral neck pain or severe h/a
  - Horner's syndrome (miosis & ptosis only)
- Tx: drug therapy (coumadin) then sx

Arterial Occlusion - Other

- Chronic/Acute Intestinal Ischemia
- Ischemic Colitis
- Renal Artery Stenosis
- Acute UE Limb Ischemia
- Mesenteric Vein Occlusion

Other Arteriopathies

- Buerger dz/(thromboangiitis obliterans): men, < 40, smokers; extremity vessels (toes)
- Pulseless Disease (Takayasu): Asian women, < 40; great vessel disease/MI
- Raynaud's: digital color change (w/b/r)
- Reflex Sympathetic Dystrophy/Complex Regional Pain syndrome: burning or aching pain disproportionate to cause after trauma

Classic red, white, blue of Raynaud's

www.commons.wikimedia.org/wiki/Raynaud%27s_disease

Peripheral Arterial Disease

- Lower extremities affected by atherosclerotic disease
- Risks include: male, increasing age, DM, HTN, smoking
- Highly associated with cerebrovascular and CAD

PAD – signs and symptoms

- Erectile dysfunction (iliac arteries)
- Claudication: fatigue/pain/weakness w/ walking & relieved by rest
- Ischemic rest pain: nocturnal foot pain
- Gangrene: implies impending limb loss
- Leriche's syndrome: b/l hip & buttock claudication, ED, and absent femoral pulses

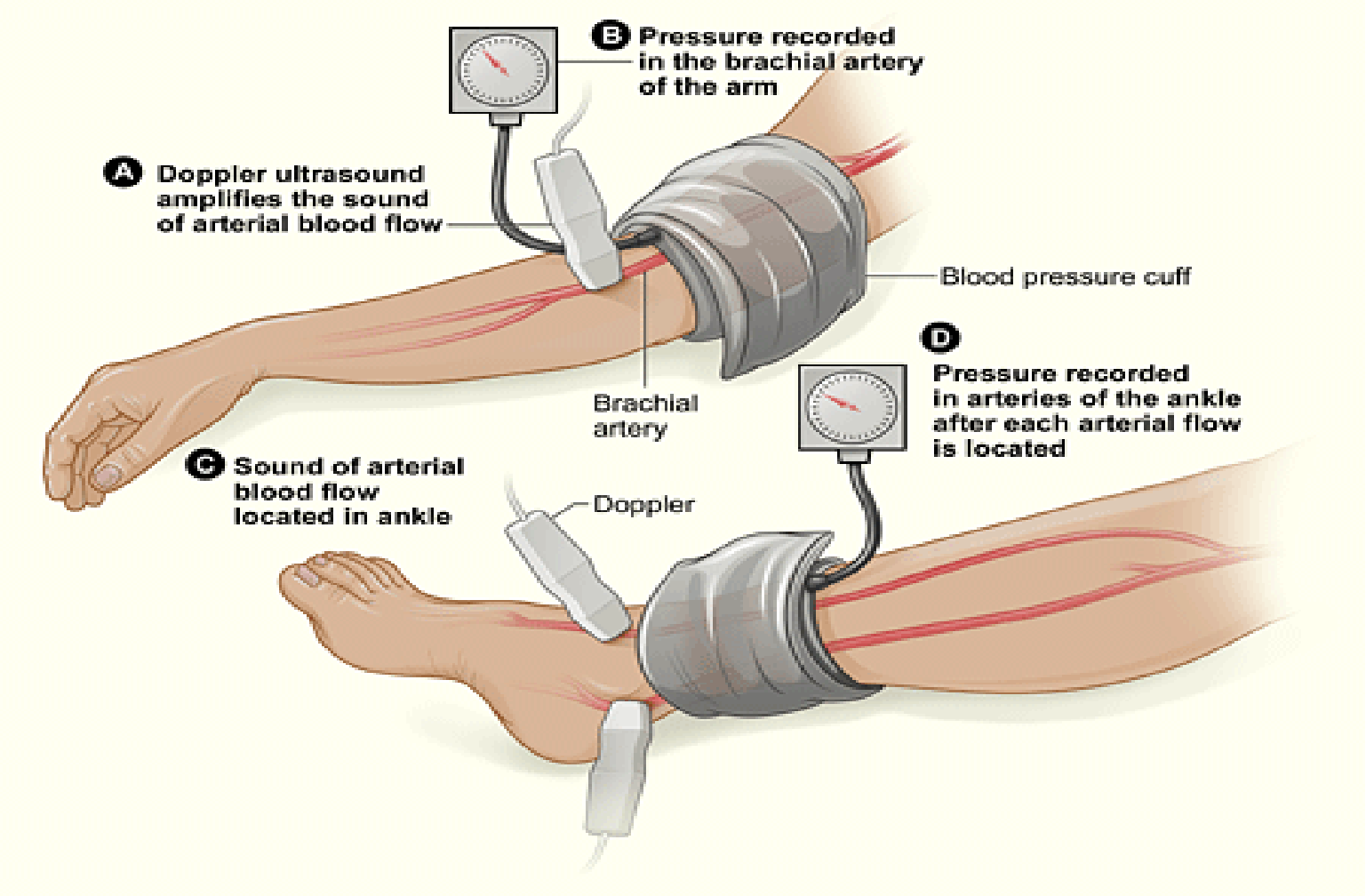
PAD – P.E. and Imaging

- Pulses, +/- bruits
- Atrophy of skin, coolness, hair loss, ulcers; dependent rubor, pale w/ elevation
- Ankle-brachial index: (1= normal; <0.8 = claudication)
- Use "waveform analysis" of ABI
- Image via angiography, CTA, or MRA prior to surgery or percutaneous treatment

Peripheral arterial disease

www.commons.wikimedia.org/wiki/Peripheral_artery_occlusive_disease

Ankle-brachial index



Sadley, personal file

PAD -- Treatment

- Identify and control risk factors: exercise, smoking cessation, lipid lowering
- Cilostazol/Pletal or ASA may be helpful
- Endovascular techniques
- Open bypass grafting
- Amputation

Giant Cell (temporal) Arteritis

- Affects medium and large vessels
- Associated with polymyalgia rheumatica (now thought to represent a spectrum of one disease—above the neck, GCA, and below the neck, Polymyalgia Rheumatica /PMR)
- Age > 50 (mean age at onset: 79 years)

Giant Cell Arteritis – s/s

- H/A, jaw claudication, scalp tenderness, visual symptoms (amaurosis fugax or diplopia)
- Blindness may result (ophthalmic artery affected)
- UE asymmetric pulses; AR murmur; subclavian bruit
- (Elderly) Fever with normal WBCs

Giant Cell Arteritis – Dx & Tx

- ESR > 50 mm/h; often > 100 (CRP, Interleukin-6)
- Biopsy of temporal artery makes dx
- Urgent tx to reduce blindness

- Prednisone 60 mg/d po X 1 month, then taper (high dose steroid)
- ? ASA 81 mg (may reduce visual loss)

Polymyalgia Rheumatica

- Pain & stiffness of shoulders/pelvis
- Frequently associated with fever, malaise and weight loss
- Often with anemia and elevated ESR
- Tx with prednisone 10-20 mg/d po (low dose steroid)
- If no improvement in 72 hours reconsider diagnosis

Phlebitis/Thrombophlebitis

- Superficial veins involved (long saphenous most common)
- IVs and PICC lines are very common
- Risks include: varicosities, pregnancy or postpartum, Behcet's (vasculitis) syndrome, trauma, abdominal cancer (Trousseau's synd.)
- Assoc. with occult DVT in 20% cases

Phlebitis – signs/symptoms

- Dull pain
- Redness, induration, tenderness in linear distribution (a firm cord)
- No edema (deep vein involvement)
- Chills/fever suggest septic cause (eg IV)
- Differentiate from cellulitis by linear distribution pattern (vs. round)

Phlebitis – Treatment

- NSAIDs, heat, elevation x 7-10 days
- Encourage ambulation
- Vein excision with complications
- Septic causes (S. aureus) require heparin and abx, such as vancomycin

Deep Vein Thrombophlebitis (DVT)

- Virchow triad (stasis, vascular injury, hypercoagulability) = cause
- Risks: CHF, recent surgery or trauma, neoplasia, OC use, sedentary eg. bedrest or long travel, factor V Leiden (inherited), protein C or S dysfxn
- 50% of patients are asymptomatic!
- Popliteal and ileofemoral veins most likely sites
- Main/serious complication is Pulmonary Embolism (50-60% of clots will migrate to lung)

DVT – signs and symptoms

- "Heavy legs", dull ache, tightness, calf/leg pain especially with walking
- Slight edema, palpable cord
- Low grade fever
- Tachycardia
- Homan's sign = only 50% positive

DVT – Diagnosis & Prevention

- Venous US is diagnostic!
- May use MR venography (gadolinium) or impedance plethysmography
- Venography w/contrast for complex presentations
- Early ambulation, SCDs, foot board
- Anticoagulation: LMWH, heparin, warfarin or prophylactic vena caval filter

Chronic Venous Insufficiency

- History of phlebitis, DVT, or leg injury
- Chronic elevation in venous pressure
- Ankle edema is earliest sign
- Late signs: itching, stasis pigmentation, dermatitis, induration, varicosities, ulceration (slow to heal)
- Ulcers: painless, large, irregular (stasis ulcers)

Chronic Venous Insufficiency ulcer

www.commons.wikimedia.org/wiki/venous_ulcer

CVI -- Management

- Leg elevation, graduated compression stockings, ambulatory exercise
- Wet saline compresses for weeping dermatitis
- Unna boot, Ace wrap, wet-to-dry saline dressings for ulcer tx; pneumatic compression devices
- Abx and antifungals when indicated
- Refer to wound center as needed

Varicose Veins

- Dilated, tortuous, superficial veins in LE
- Incompetent venous valves
- Seen in 15% of adults
- Risk factors: female, pregnancy, fam. hx., standing, h/o phlebitis
- Greater saphenous vein most common

Varicose Veins

www.commons.wikimedia.org/wiki/Varicose_veins

Varicose Veins – s/s

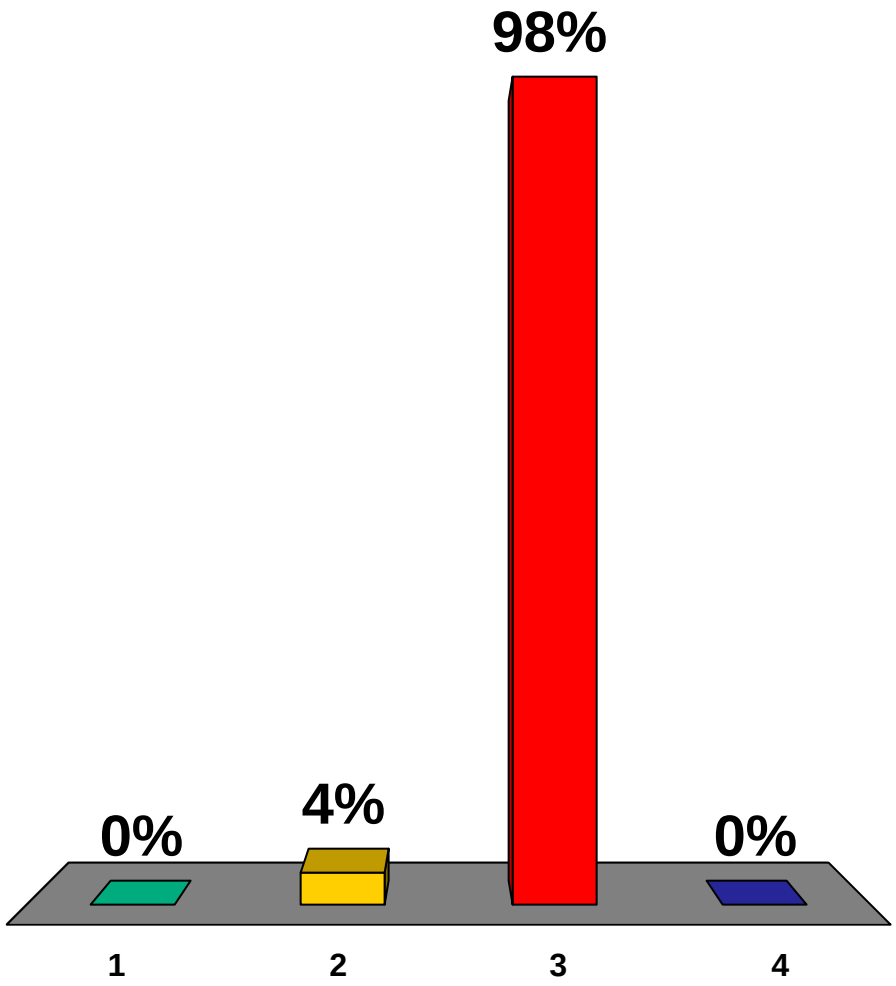
- Dull, achy, heaviness, fatigue in LE
- Dilated, tortuous, elongated veins
- Smaller, flat, blue/green veins, and spider veins provide evidence
- Signs of chronic venous insufficiency
- Test valve competence with Trendelenburg test

Varicose Veins - Treatment

- Non-sx = daytime stockings, exercise, elevate legs frequently when possible
- Surgery after Doppler US; endovenous ablation using radio frequency or laser
- Sclerotherapy useful for spiders, telangiectasias, and small varicosities

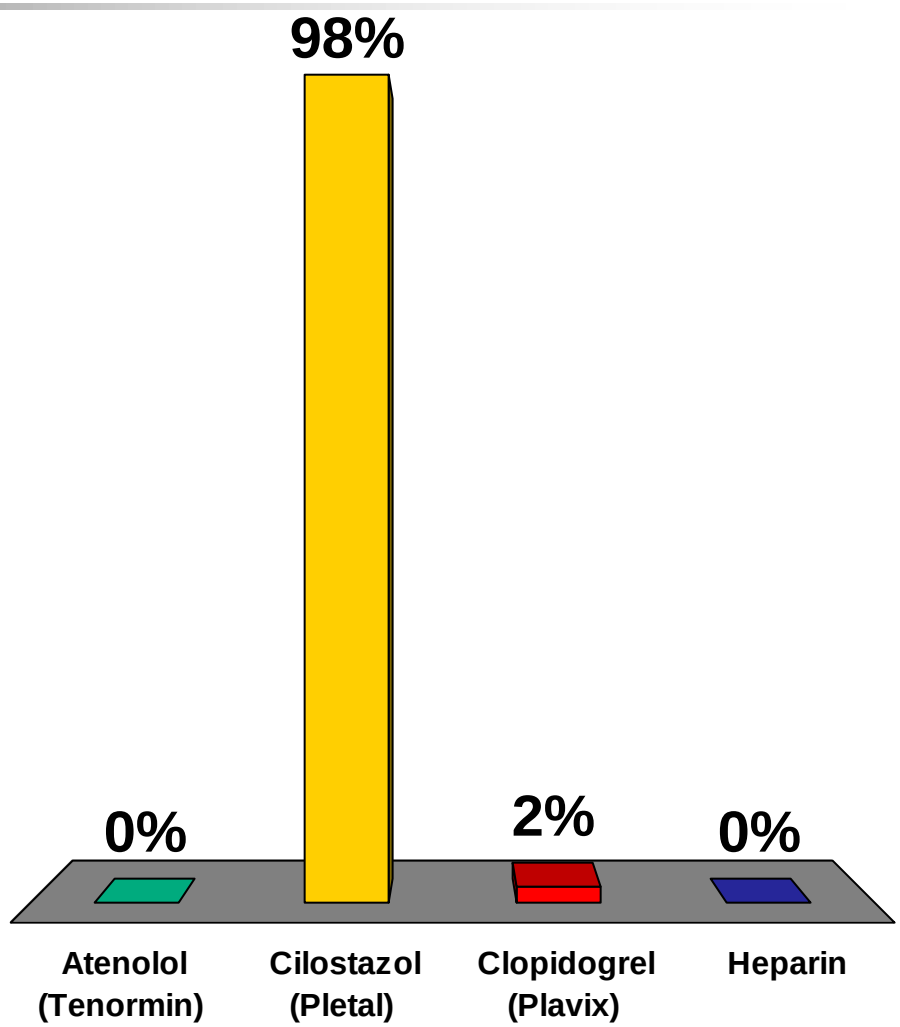
A 64 y/o male presents c/o headache pain that seems to worsen while eating for the past 2 days. PE reveals scalp tenderness and fever. What treatment is recommended?

1. Broad spectrum antibiotics
2. Cilostazol/Pletal
3. Corticosteroids ✓
4. Lipid-lowering agent



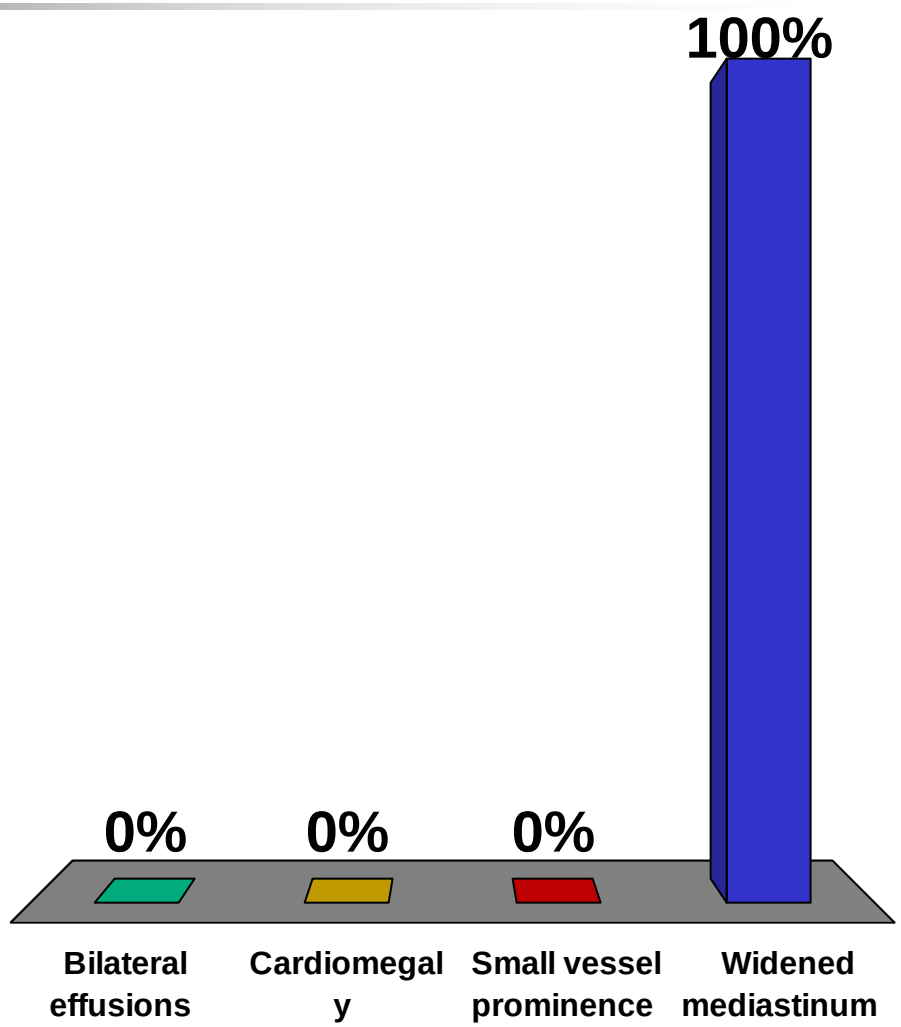
A 62 y/o male smoker presents for evaluation of lower extremity claudication. His ankle-brachial index is reduced to 0.8. Which medication should be used?

1. Atenolol (Tenormin)
2. ✓ Cilostazol (Pletal)
3. Clopidogrel (Plavix)
4. Heparin



A 78 y/o male presents to the ED c/o a sudden onset of searing chest pain, radiating to his back and neck. BP is noted at 220/108 mmHg. What would you expect on CXR?

1. Bilateral effusions
2. Cardiomegaly
3. Small vessel prominence
4. Widened mediastinum ✓



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Thanks and enjoy the remaining Conference!