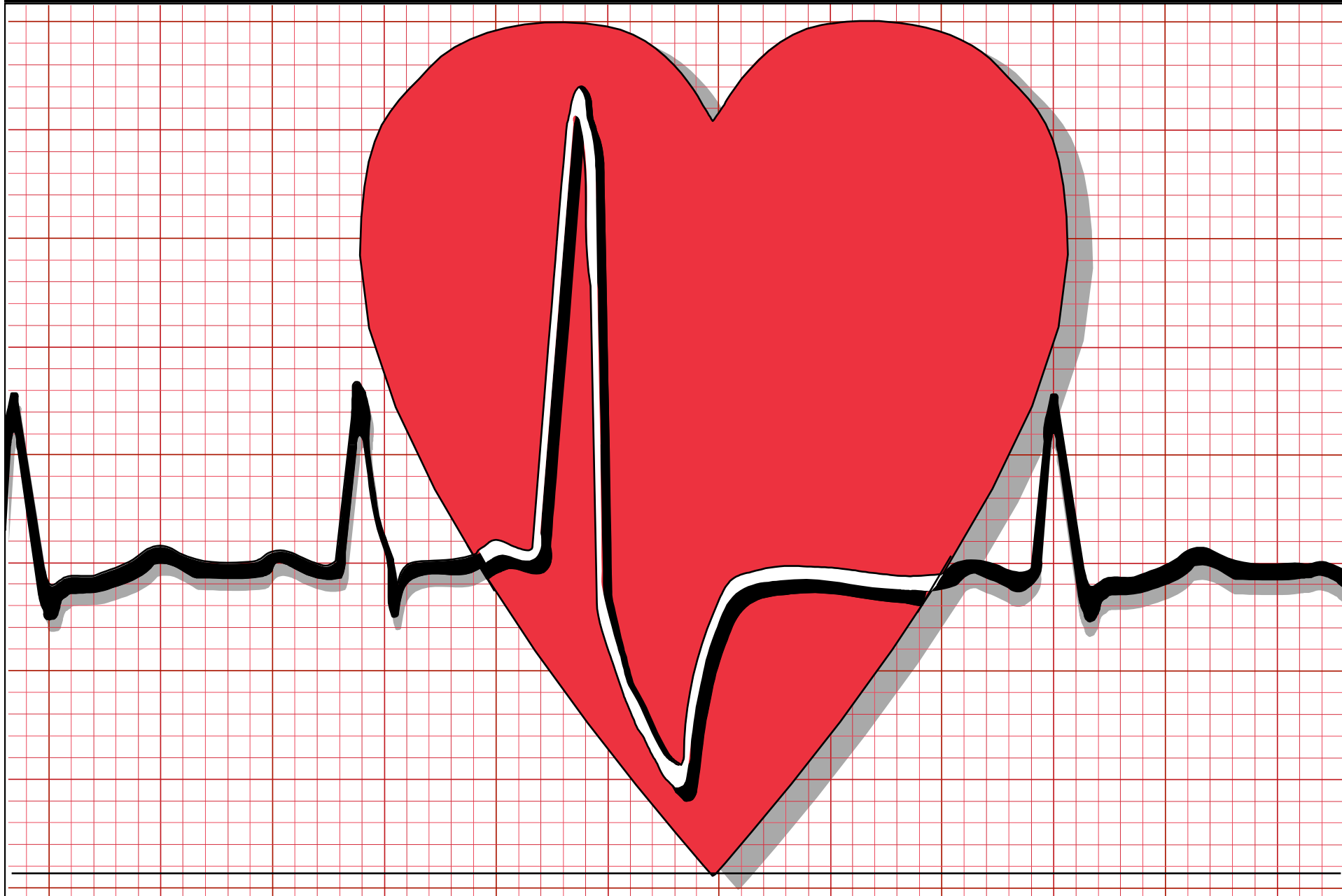


Cardiology



A few words first...

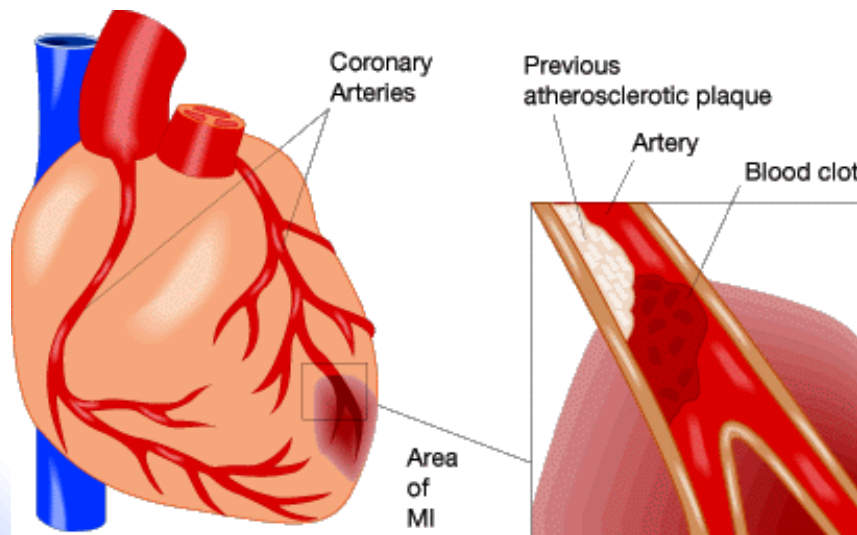
- Review, review, review
- Handout more comprehensive
- I'll focus on key points
- Special attention to red underlined text
- Special attention to “most common cause of...”
- Special attention to triads, quadrads
- Know ACLS 2010
- Always consider deadly causes first

Acute Coronary Syndrome (ACS)

- Spectrum of disease due to myocardial ischemia
 - Unstable angina to acute MI
- Ischemic heart disease is the most common cause of death in the US
- 2 million MI and unstable angina patients each year

Causes of ACS

- Usually due to atherosclerosis
 - Fixed lesion, critical stenosis
 - Plaque disruption, platelet aggregation, thrombus formation
- Results in oxygen supply/demand imbalance leading to cardiac muscle damage



Non-Atherosclerotic Causes of ACS

- Trauma
- Connective tissue diseases (vasculitis)
- Metabolic diseases (thickening of vessels)
- Congenital anomalies
- Thrombus (DIC, TTP)
- Emboli (bacterial, non-bacterial)
- Thoracic aortic dissection
- Drugs (cocaine)
- Infectious diseases



Cocaine-Associated Chest Pain

Acad Emerg Med 2000

- How often is chest pain associated with cocaine use reflective of an AMI?
- Only 6% had an AMI (consistent with other studies)
- Acute use of cocaine markedly increases the risk of acute MI
- Chronic use of cocaine causes premature atherosclerosis (recent literature — controversial)

ACS

ACS: Unstable angina —→ Non-STEMI —→ STEMI

- Unstable angina
 - Resting
 - New onset
 - Increasing severity
 - High risk of MI or death
 - Duration >20 minutes, hypotension, CHF, dynamic ST changes

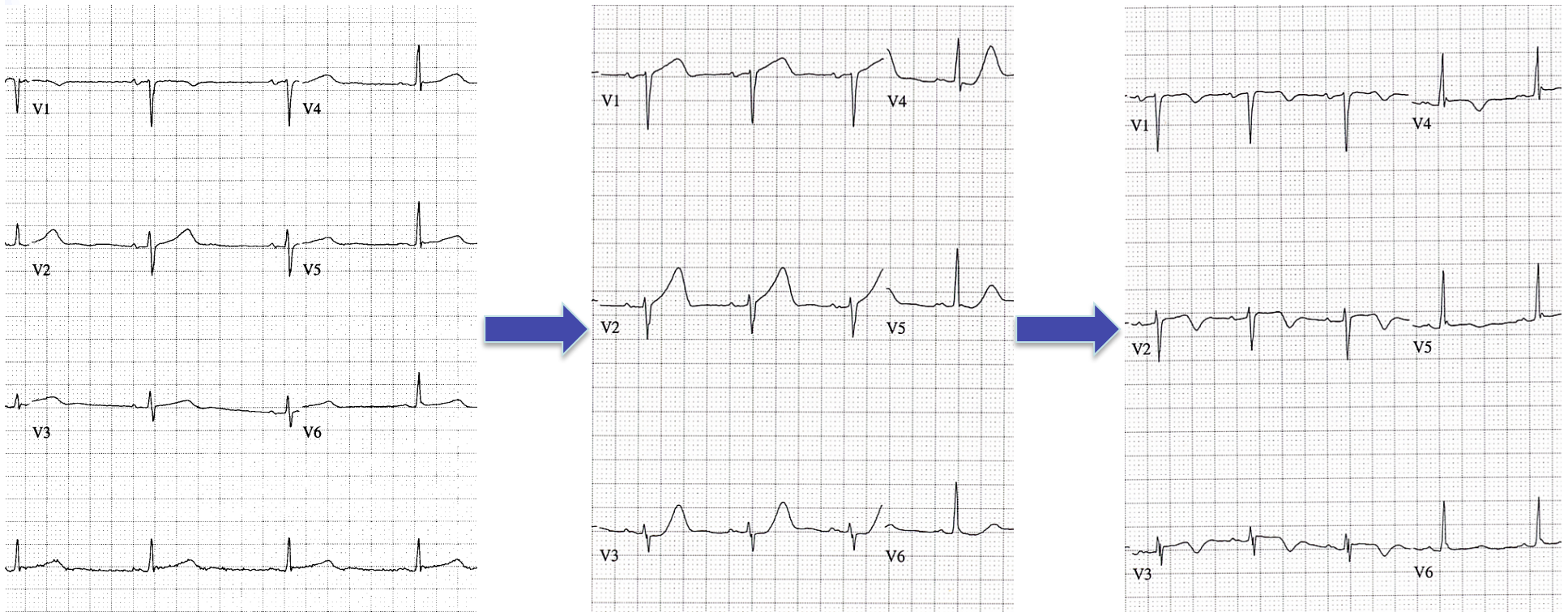
Prinzmetal's angina (variant angina):
Coronary artery spasm, occurs at rest,
ST elevation (not depression), may not have CAD

Acute Myocardial Infarction (AMI)

- Classification of AMI
 - **STEMI/Q wave**: transmural infarction involving full thickness of myocardium
 - **Non-STEMI/Non-Q wave**: subendocardial infarction involving partial wall thickness. Has lower initial mortality and morbidity, but long term prognosis is similar

AMI EKG Changes

- 4% of AMIs have normal EKG
- Hyperacute T waves (early)



AMI EKG Changes

- ST segment changes
 - 1mm of elevation in two anatomically contiguous leads
 - Elevation usually corresponds to areas of involvement
 - Exception: AMI with LBBB
 - Depression over areas opposite injury (reciprocal changes). Predictor of larger MI, increased mortality
- T wave inversion: within 4 hours is good Px sign
- Significant Q waves: 1 square wide, 1/3 of height of R wave

The EKG in AMI (1)

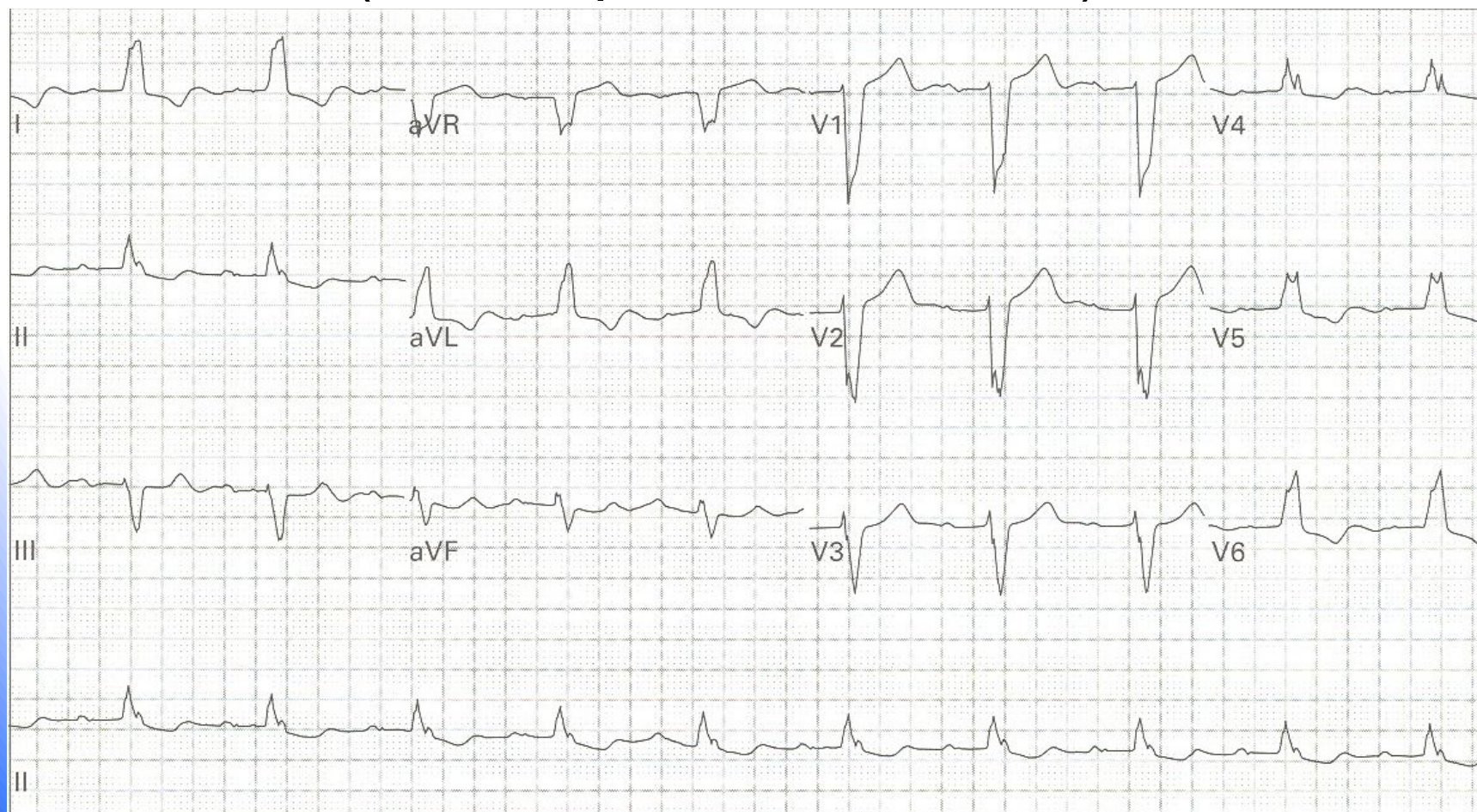
- The EKG can guide therapy in AMI by helping:
 - Identify who needs emergent reperfusion
 - Identify the infarct-related artery
 - Predict the amount of myocardium at risk
 - Reflect reperfusion
 - Identify new dysrhythmias / conduction issues

The EKG in AMI (2)

- EKG indications for emergent reperfusion
 - STE ≥ 1 mm in 2 contiguous leads (incl. posterior)
 - New left bundle branch block
 - Old left bundle branch block with Sgarbossa criteria
 - ST-segment elevation measuring ≥ 1 mm concordant with the QRS in any lead.
 - ST-segment depression measuring ≥ 1 mm in any of the V1 through V3 leads.
 - Discordant ST-segment elevation measuring ≥ 5 mm (lower utility)

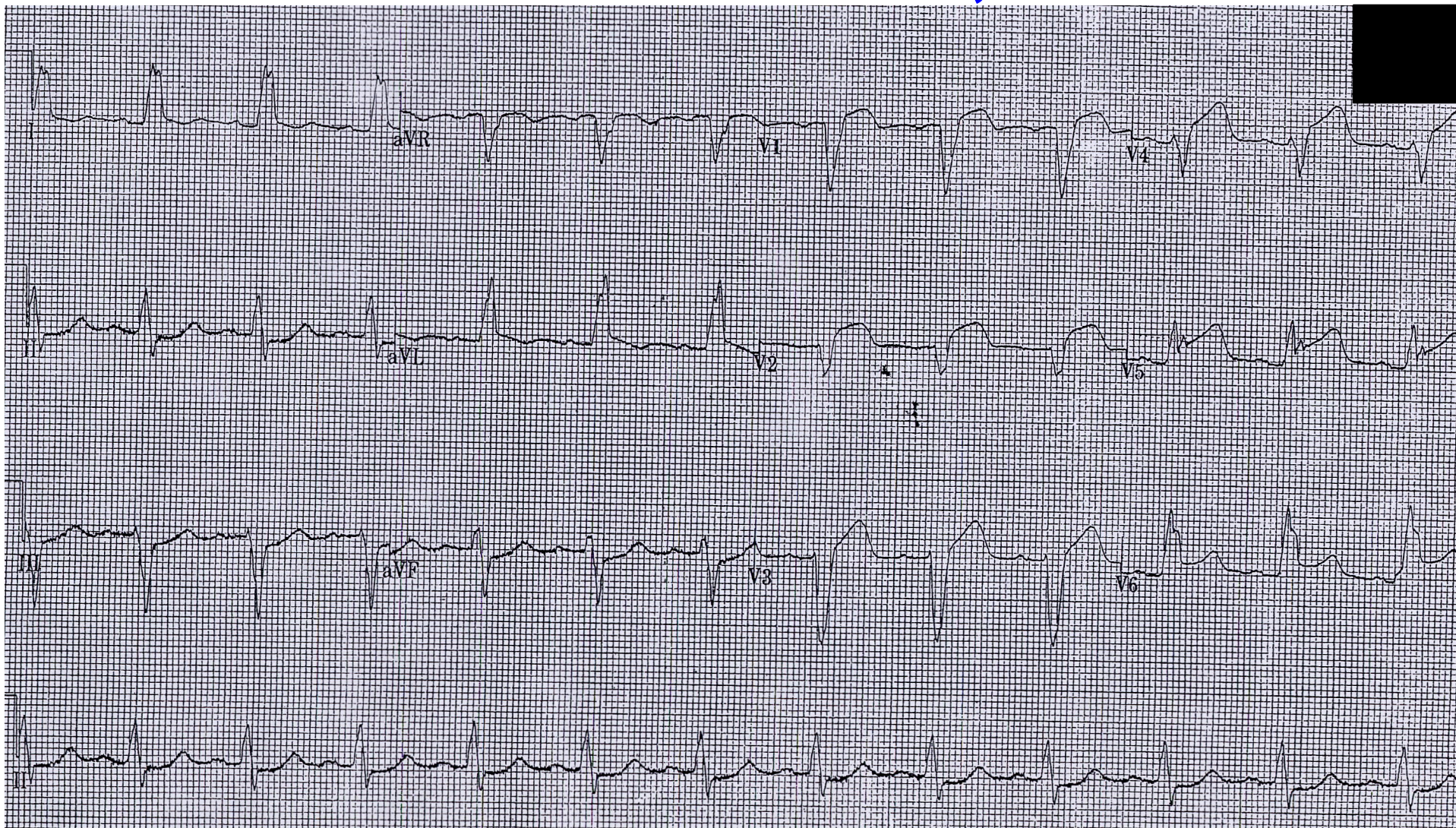
Normal LBBB

Rule of appropriate discordance
(true for pacemakers also)



The EKG in AMI (2)

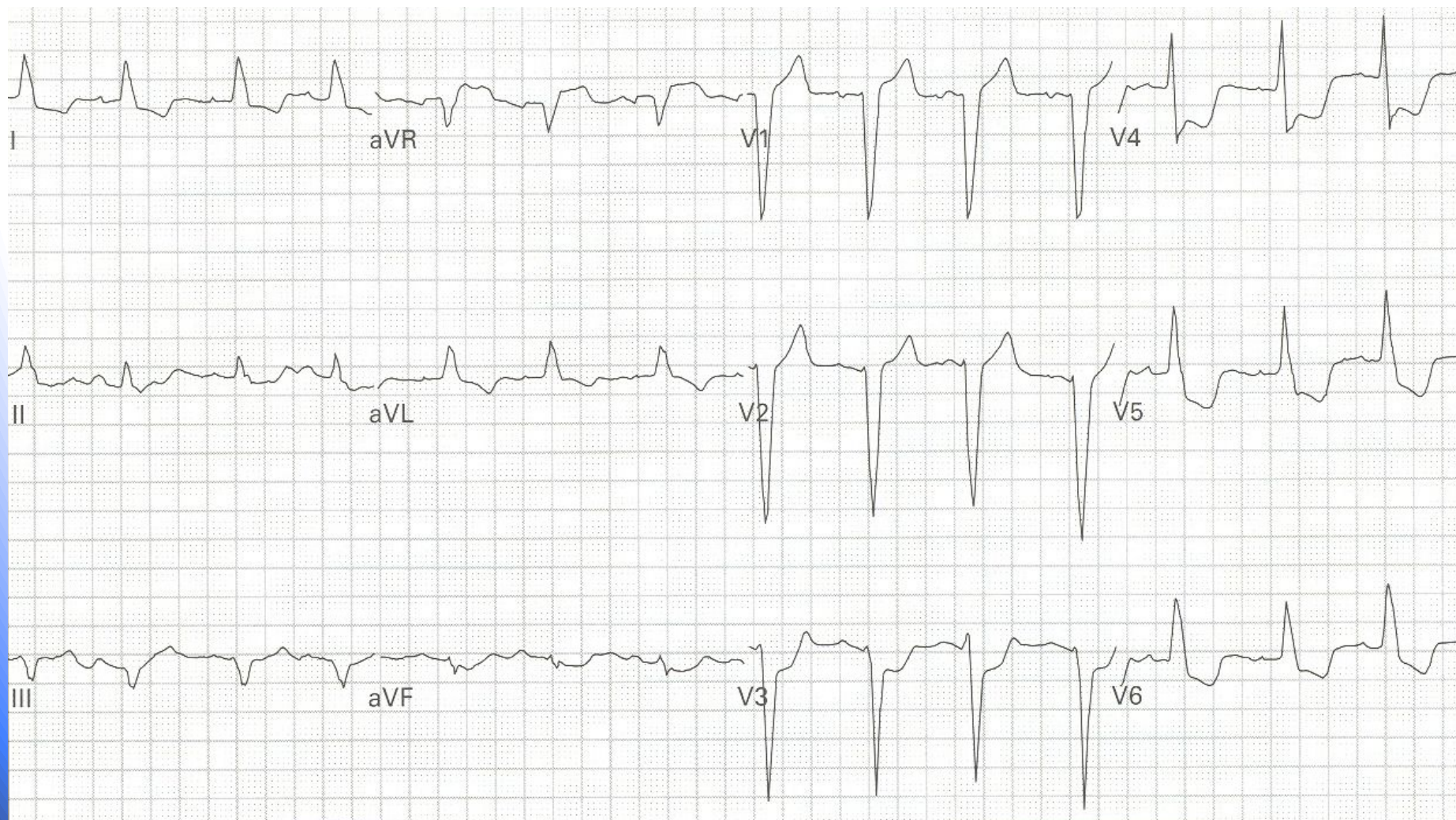
Concern in leads aVL, V5-6



From *ECGs for the Emergency Physician Vol 2* (Mattu, Brady; Blackwell 2008)

The EKG in AMI (2)

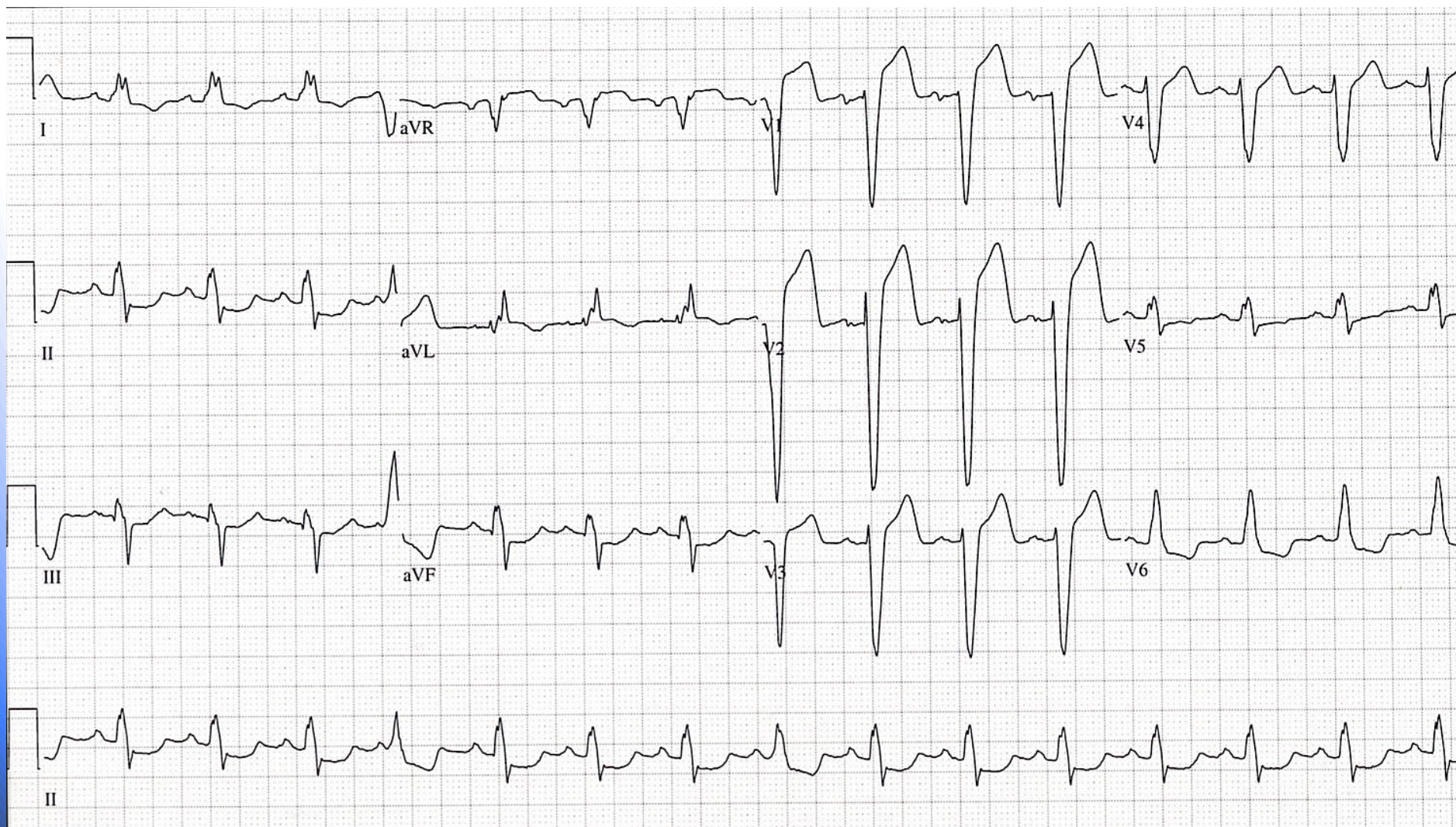
Concern in lead V3



From *ECGs for the Emergency Physician Vol 1* (Mattu, Brady; Blackwell 2003)

The EKG in AMI (2)

Concern in leads V1-2

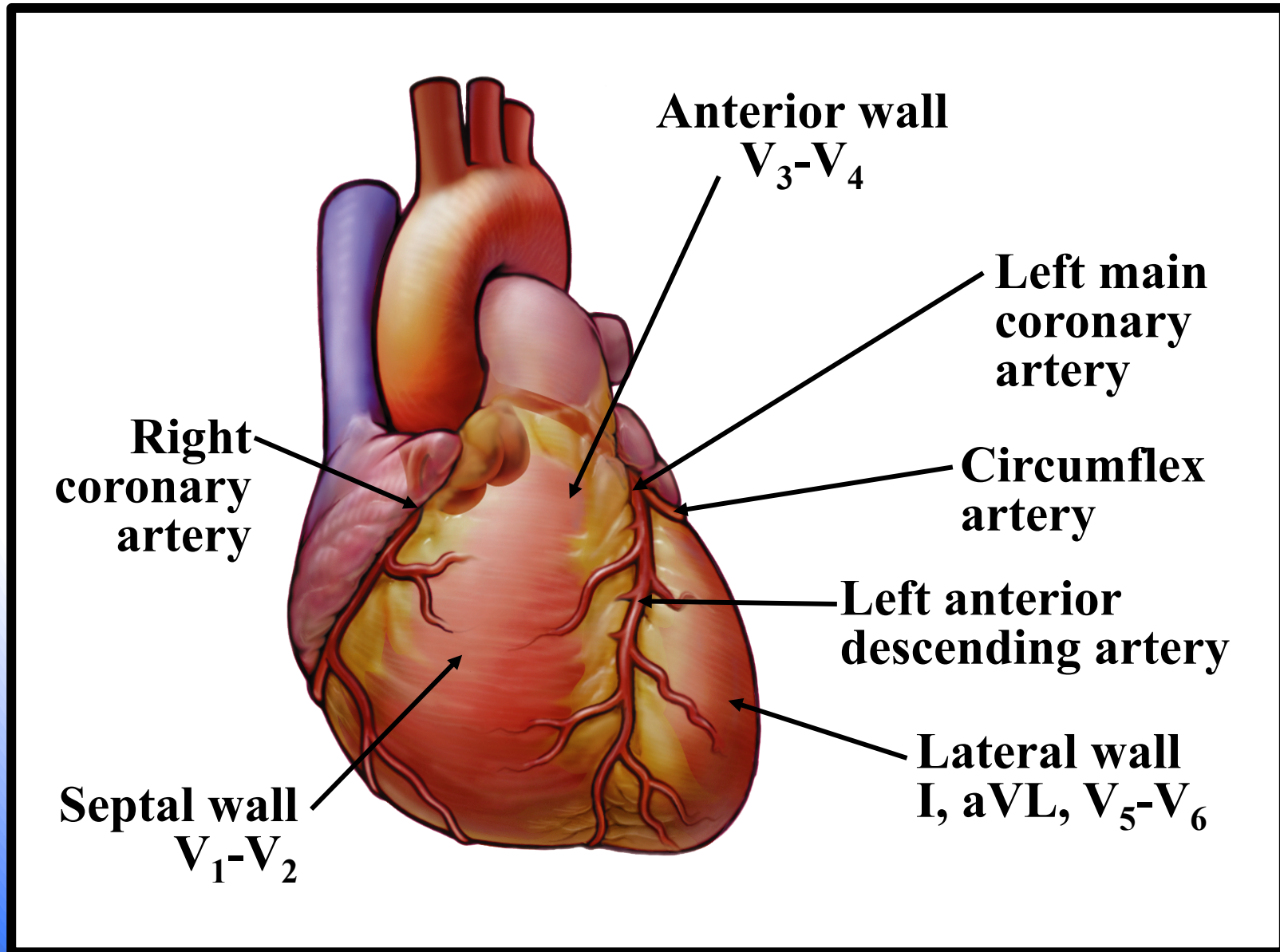


From *ECGs for the Emergency Physician Vol 2* (Mattu, Brady; Blackwell 2008)

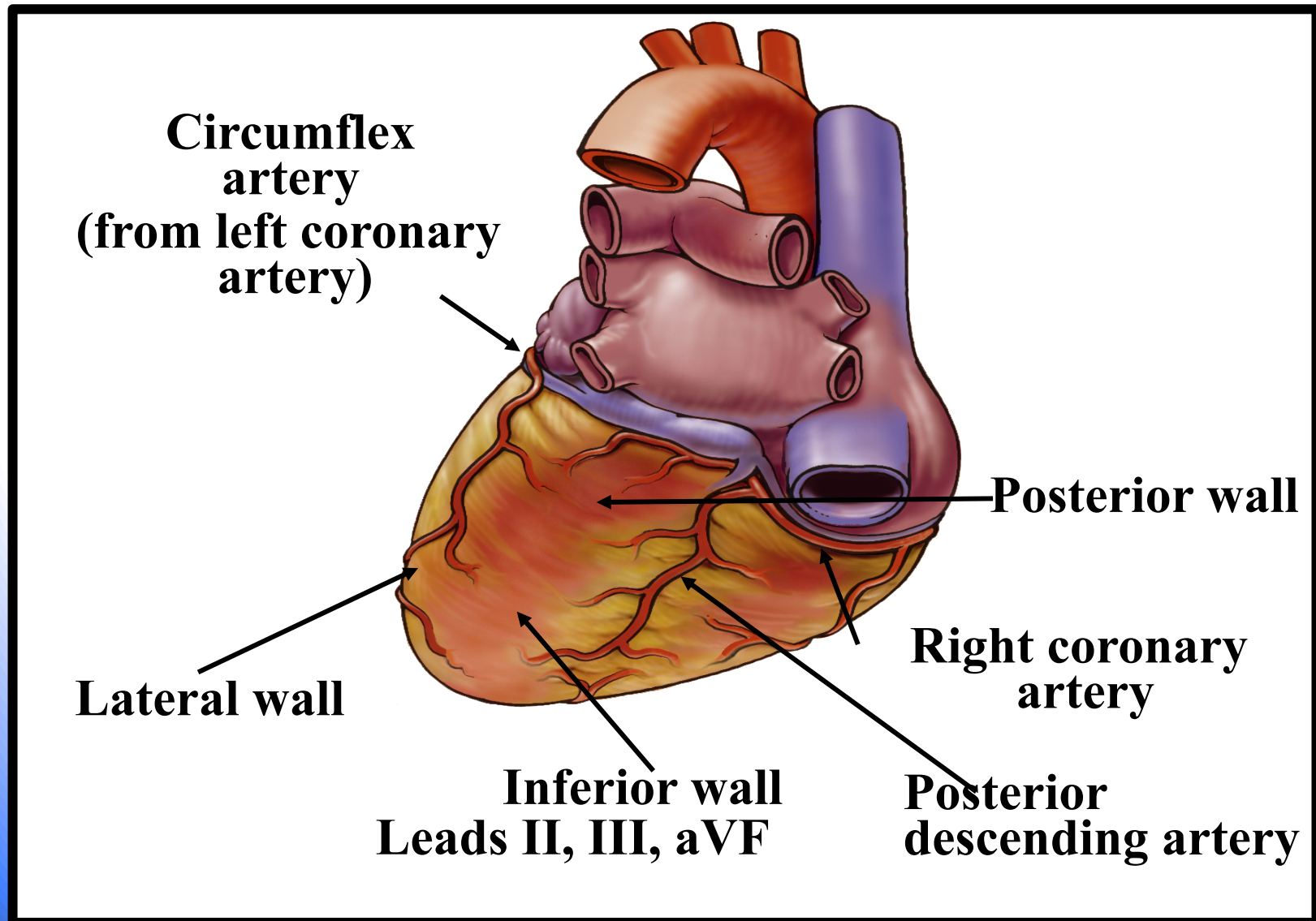
The EKG in AMI (2)

- Predictors of reperfusion
 - Normalization of ischemia-related ST elevation
 - Failure to normalize may indicate the need for “rescue PCI”
 - Early T wave inversions can be highly specific markers of reperfusion
 - An accelerated idioventricular rhythm (60-120) is also highly specific for reperfusion / benign, don't suppress it

EKG Anatomy (1)



EKG Anatomy (2)



EKG Anatomy (3)

I lateral	aVR	V₁ septal	V₄ anterior
II inferior	aVL lateral	V₂ septal	V₅ lateral
III inferior	aVF inferior	V₃ anterior	V₆ lateral

- RV infarct: inferior injury pattern + ST elevation V4R
- **True posterior infarct**: R>S waves V1 and V2, upright Ts, ST depression in V1-2

The EKG in AMI (1)

- Inferior MI (ST elevation II, III, aVF)
 - 80% right coronary, 20% circumflex
- Right ventricular MI (ST elev. V1 and II, III, aVF)
 - Always right coronary artery
- Anterior MI (ST elevation in V1, V2, V3)
 - Left anterior descending coronary artery (LAD)

The EKG in AMI (3)

- Bradycardias or conduction defects associated early with inferior AMIs tend to resolve spontaneously and are atropine-sensitive (tend to reflect self-resolving edema)
- Conduction abnormalities associated with anterior AMIs carry a worse prognosis because they reflect necrosis of the conduction tissue (early pacing if any new or partial BBB develops)

The EKG in AMI (4)

- Tachycardias
 - Causes = reperfusion, altered autonomic tone or hemodynamic instability
 - New atrial fib = increased vagal tone, increase left atrial pressure, atrial infarction or pericarditis (is a marker of a worse prognosis no matter the cause)
 - Sustained V. tach over 150 = very large infarction
 - Ventricular fibrillation - early = ischemia / late (3 weeks) = progressive pump failure
 - Ventricular fibrillation in early AMI indicates lack of reperfusion = PCI
 - PVCs don't predict sustained ventricular dysrhythmias / don't routinely suppress

Cardiac Markers (1)

- Myoglobin
 - 1-2 hours (rises)
 - 4-6 hours (peaks)
 - 24 hrs (normalizes)
- Troponin
 - 3-6 hours (rises)
 - 12-24 hours (peaks)
 - 7 days (normalizes)
- CK-MB
 - 3-4 hours (rises)
 - 12-24 hours (peaks)
 - 1-2 days (normalizes)

Cardiac Markers (2)

- Myoglobin
 - Advantage: early detection
 - Disadvantage: poor specificity, especially in trauma, renal failure, hemolytic syndromes
- Troponin: more specific for AMI than CK-MB
 - High values predict complications and mortality
- CK-MB
 - Elevation without infarction (skeletal disease, muscle exertion, cocaine, renal failure)
 - Comparing MB to total CK improves specificity
 - 30-40% false negatives in UA = NSTEMI

Therapy of AMI (1)

- Oxygen
- Nitrates (sublingual, topical, IV)
 - Contraindicated if sildanefil (Viagra) etc. within 24 hours or if hypotensive
 - Caution/avoid in RV MI
 - Vasodilatation
- ASA (162-325 mg)
 - ASA alone reduces mortality 23%
 - Combined with thrombolytics, ASA reduces mortality 42%
- Morphine for persistent pain

Therapy of AMI (2)

- Heparin (Unfractionated or LMWH)
 - 60 u/kg IVP, followed by 12u/kg/hr IVPB
 - 30 mg IVP, followed by 1 mg/kg SQ q 12 hrs
- Beta blocker (metoprolol) early IV discouraged
 - Give within 24 hours orally but no rush
 - Contraindications (asthma, CHF, bradycardia, hypotension; caution in RV MI)
- Platelet inhibitors
 - GP IIb/IIIa receptor antags IV
 - Clopidogrel: 300-600 mg oral load or
 - Prasugrel 60 mg oral load at cath; avoid if history of TIA or stroke

Therapy of AMI (3)

- Thrombolysis vs. percutaneous coronary intervention
 - Outcomes have shown consistent benefit with PCI over thrombolytics
 - Guidelines recommend PCI if balloon inflation can be performed **within 90 minutes**
 - Window of benefit extended if chest pain > 6 hours or if cardiogenic shock

Thrombolytic Therapy Indications

- Chest pain greater than 30 minutes but less than 12 hours, not relieved by nitroglycerin
- Acute change in EKG with any of the following
 - At least 1 mm ST elevation in two contiguous leads
 - New LBBB or Sgarbossa criteria
- PCI delayed greater than 90 minutes

A RBBB should not obscure the diagnosis of an acute MI

Contraindications to Thrombolytic Therapy

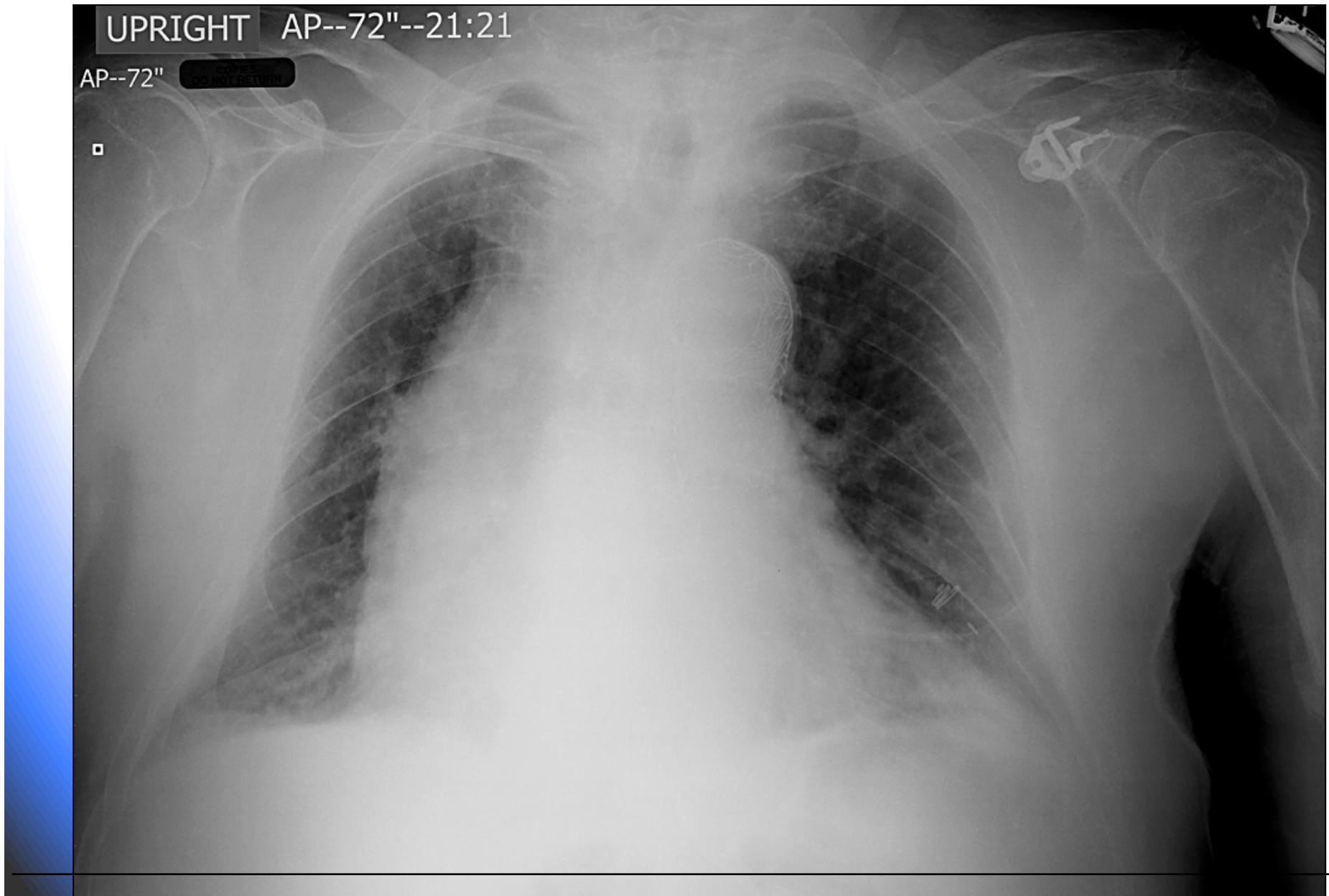
Absolute

- (PCI immediately available)
- Active bleeding from any site
- CVA within 6 months or hemorrhagic CVA at any time in the past
- Intracranial or intraspinal surgery or trauma within 2 months
- Intracranial or intraspinal neoplasm, aneurysm or AV malformation
- Suspected aortic dissection

Relative

- History of GI bleed
- Prolonged CPR
- Surgical or invasive procedure within 3 weeks
- Severe bleeding diathesis or thrombocytopenia
- Uncontrolled hypertension (diastolic >120 after treatment)
- Significant trauma within 4 weeks
- Pregnancy or ≤ 10 days post-partum
- Active cavitory lung disease
- Known allergy to agent

No Thrombolytics



Complications of Thrombolytic Therapy

- Bleeding (send blood for type and screen)
- Serious bleeding in 5%
- Intracranial hemorrhage: approximately 0.5%.
Higher risk with uncontrolled BP, age > 65, low body weight

Evidence of Reperfusion

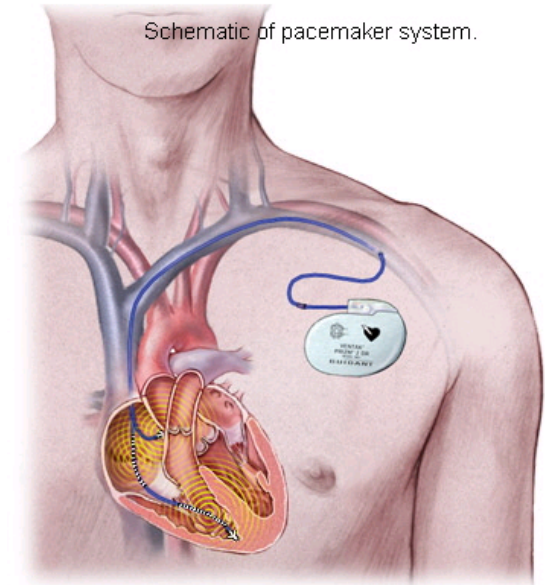
- Chest pain resolved
- ST elevation resolved
- Reperfusion dysrhythmias

Early Complications of AMI (1)

- Blood supply to pacemaker and conduction tissue
 - Sinus node (right coronary, 60% / circumflex, 40%)
 - AV node (right coronary, 90% / circumflex, 10%)
 - Septal conduction (left anterior descending)
- Good prognosis
 - Bradycardia (without hypotension)
 - 2° Mobitz type 1 AV block (Wenckebach)
 - 3° AV block with inferior MI (resolves)
 - Narrow QRS complexes
 - PVCs, PACs
 - Early non-sustained V tach

Early Complications of AMI (2)

- Poor prognosis
 - 2° Mobitz II (progress to 3°)
 - 3° AV block from anterior MI
 - Persistent sinus tach, SVT, A-fib
 - New BBB, bifascicular block (RBBB + hemiblock)
 - Left posterior hemiblock (large infarct size)
 - Increased risk of pump failure, mortality



High-grade blocks (i.e. Mobitz II, 3° AV block) seen in anterior MI due to structural loss of conduction tissue → need pacemaker

Early Complications of AMI (3)

- Cardiogenic shock: usually $>40\%$ of LV muscle necrosed
 - High mortality
 - Treatment: fluids, inotropes, IABP (intra-aortic balloon pump) to increase coronary blood flow
- Papillary muscle dysfunction
- Acute mitral regurgitation (usually due to ischemic dysfunction of papillary muscles)
- Recurrent chest pain
- Ischemia vs reinfarction (may require PTCA or CABG)

Early Complications of AMI (4)

- Right ventricular infarction
 - Associated with inferior MI
 - Do right sided chest leads (especially V4R) looking for ST elevation

Triad of hypotension, JVD and clear lungs

- RV loses function, acts as conduit only (not pump)
- Heart becomes very preload-dependent
- Use NTG and MS with caution (can drop BP precipitously)
- Treat as any other MI but use fluids liberally to augment preload

Late Complications of AMI (1)

- Recurrent chest pain
- Embolism (from mural thrombus)
- Pericarditis
 - Post-MI (seen 1-7 days after transmural infarct)
 - Treatment: NSAIDs
- Dysrhythmias

Dressler's Syndrome: pericarditis 2-8 weeks post-MI
(probably a continuum with earlier pericarditis)
Fever, leukocytosis, friction rub, pericardial and
pleural effusions. Treatment: NSAIDs & steroids

Late Complications of AMI (2)

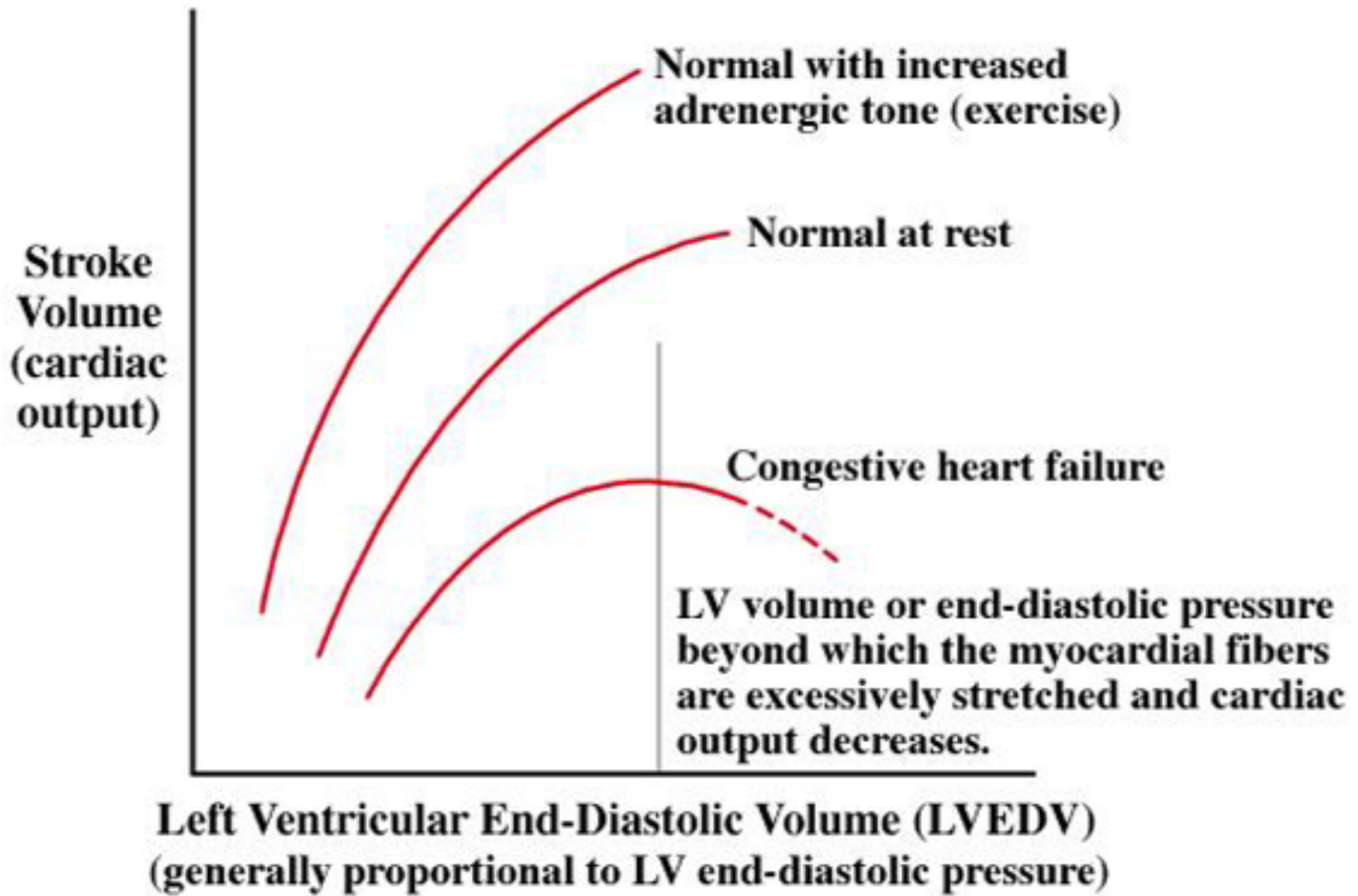
- Myocardial rupture
 - LV free wall: often results in acute tamponade, hypotension and death within two weeks
- Papillary muscle rupture
 - Results in acute MR and acute onset CHF
- Septal wall rupture
 - Results in acute VSD with acute onset CHF
 - Anterior or inferior MI
 - Treatment: afterload reduction, aortic balloon, surgery

All seen 1-7 days post-MI and treated surgically

Heart Failure

- Heart failure is the heart's inability to maintain sufficient perfusion for adequate organ function
- Congestive heart failure is the inability to clear venous return
- Stroke volume of heart depends on
 - Preload
 - Afterload
 - Contractility
- Frank-Starling curve shows the relationship between these

The Frank-Starling Curve

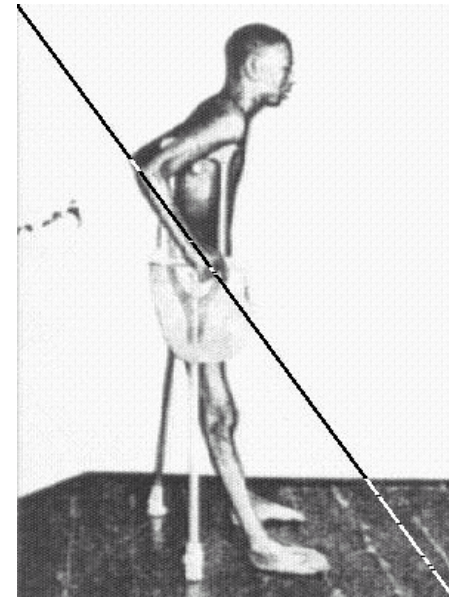


Etiology of Heart Failure

- Primary myocardial diseases (low output failure)
 - Hypertension (most common cause)
 - Coronary artery disease, MI
 - Valvular heart disease
 - Cardiomyopathy (i.e. ischemic)

Etiology of Heart Failure

- Increased workload on heart (high output failure)
 - Thyrotoxicosis
 - Anemia
 - A-V fistula
 - Beriberi
 - Paget's disease of the bone



Classification of Heart Failure

- Left-sided or right-sided systolic dysfunction
 - Impaired contractility → low ejection fraction, low cardiac output (e.g. MI, dilated cardiomyopathy) → high renin and angiotensin levels, high afterload

Classification of Heart Failure

- Diastolic dysfunction
 - Impaired relaxation of heart in diastole leads to decreased LV filling and pulmonary congestion
 - May eventually lead to systolic dysfunction
 - Causes: ischemia, hypertrophy, amyloidosis

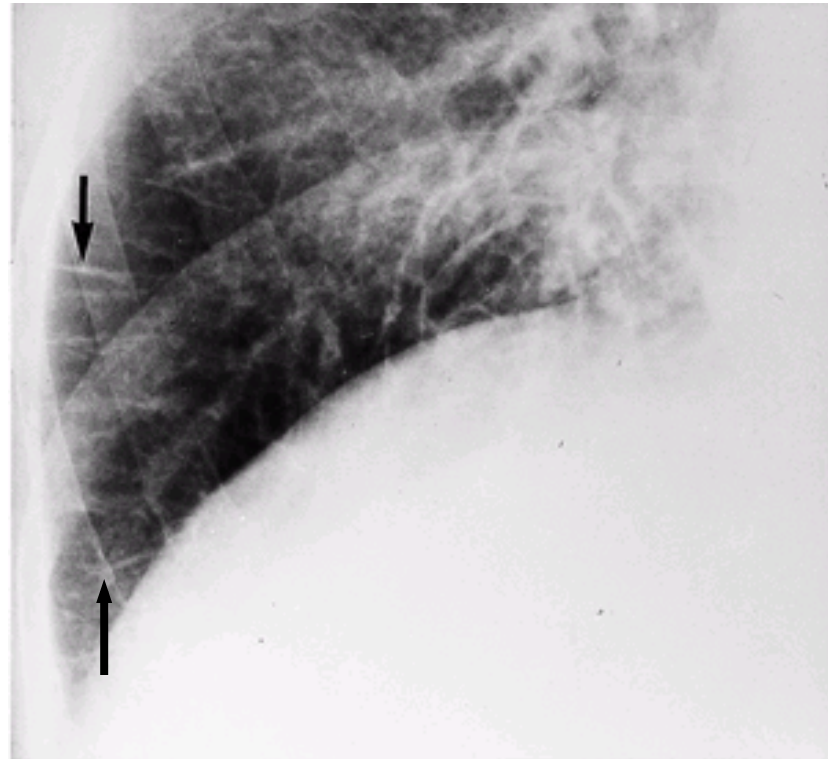
Presentation of Heart Failure (1)

- Left sided failure

- Dyspnea
- Orthopnea
- Tachycardia
- S3 gallop

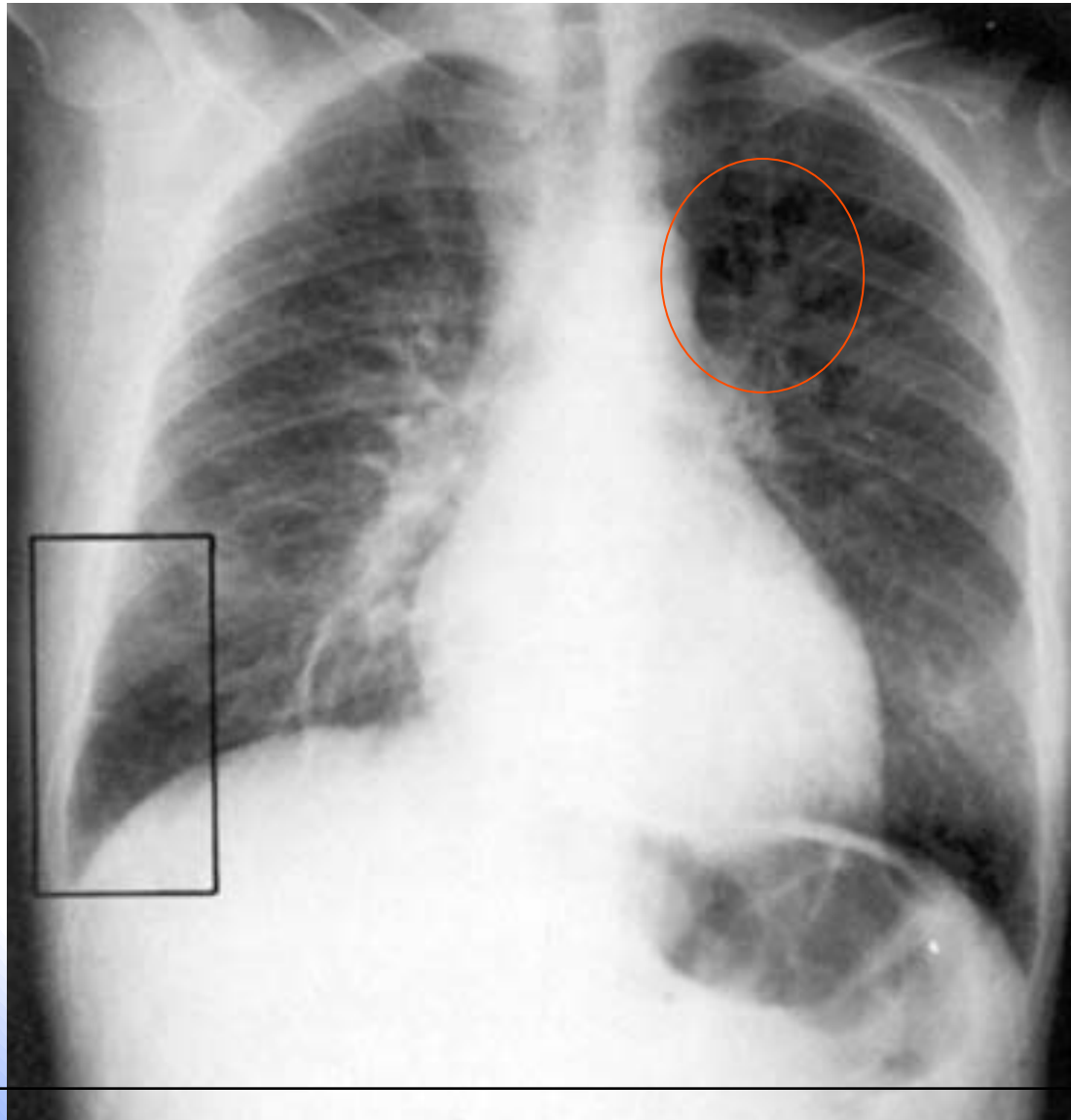
Pulmonary edema

- Redistribution
- Kerley B lines (Interstitial edema / lymphatic engorgement)
- Alveolar edema



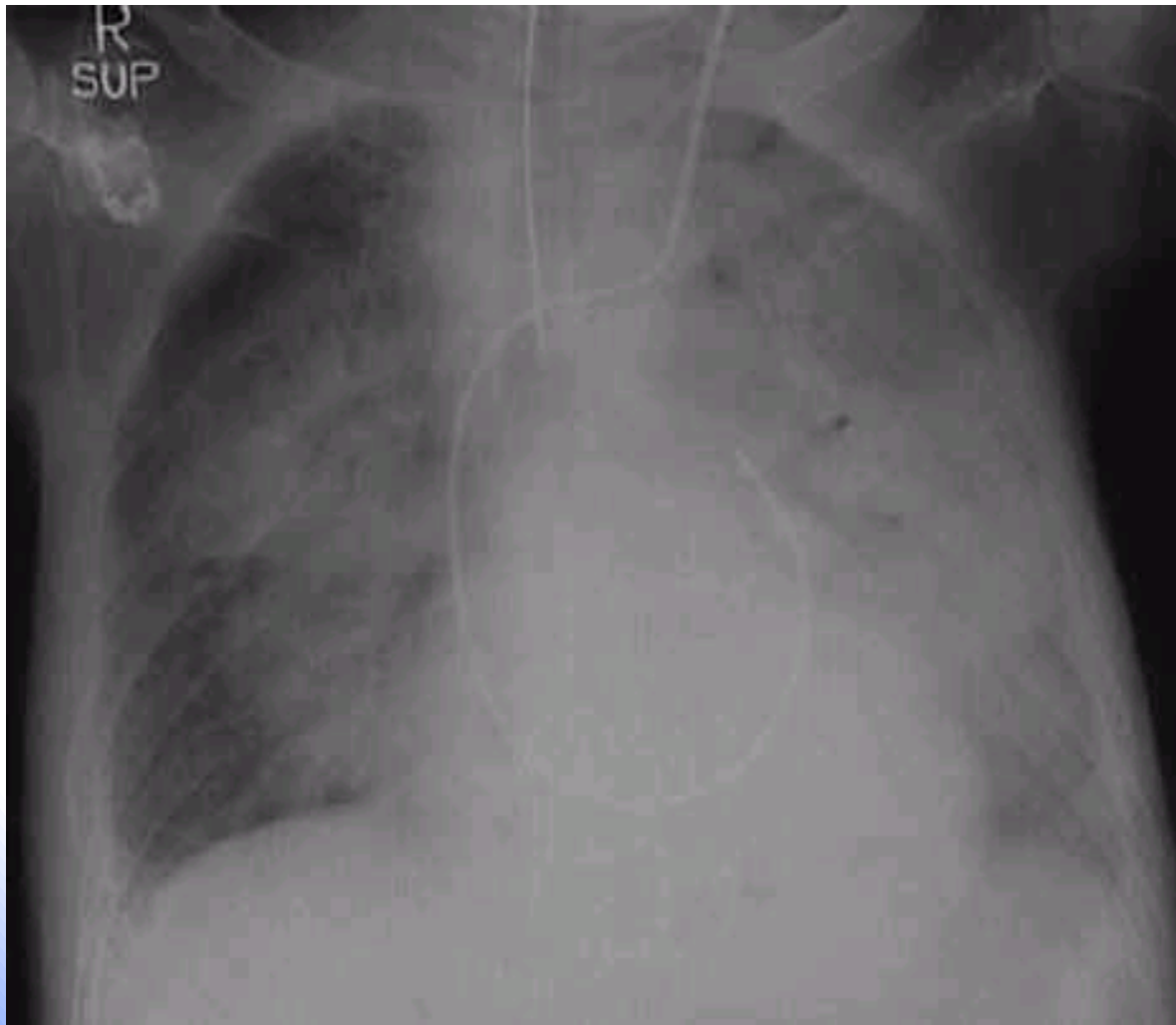
Congestive Heart Failure

Interstitial edema



Congestive Heart Failure

Alveolar edema



Presentation of Heart Failure (2)

- Causes of acute right sided failure (rare)
 - Pulmonary embolism
 - RV infarction
- Signs and symptoms
 - Peripheral edema
- Right upper quadrant pain (liver engorgement)
 - JVD
 - Pulsatile, enlarged liver
- The most common cause of right sided failure is left sided failure
- Longstanding heart failure is usually due to dysfunction of both ventricles

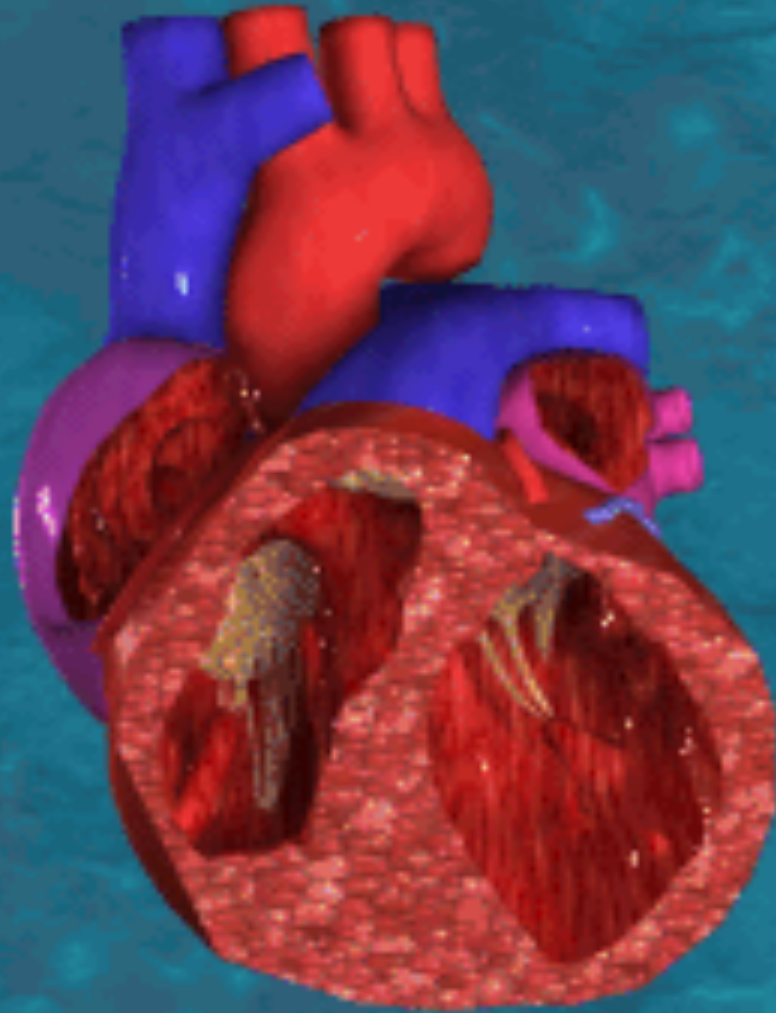
Treatment of Acute Heart Failure (1)

- Refractory heart failure (5 year mortality is 50%)
- Antidysrhythmics do not lower fatal arrhythmias
 - AICDs commonly used now chronically
- ACE inhibitors prolong survival
- Treat underlying cause (remember ischemia!)
- Symptomatic treatment with O₂, CPAP, BiPAP
- Afterload reduction with
 - IV nitroglycerin
 - ACE inhibitors
 - Nitroprusside

Treatment of Acute Heart Failure (2)

- Preload reduction with
 - Nitrates
 - Diuretics (after afterload reduction)
 - Nesiritide (??)
 - Morphine (??)
 - Phlebotomy (decrease circulatory volume, best with renal failure)
- Inotropes
 - Dopamine
 - Dobutamine

Valvular Heart Disease



Infective Endocarditis (1)

- Risk factors for infective endocarditis (IE)
 - Rheumatic or congenital heart disease
 - Prosthetic valves
 - IVDA
 - Acquired valvular disorders (e.g. AS)
 - Mitral valve prolapse (small risk)
 - Cardiac pacemakers
 - Prior history of endocarditis
- Median age is increasing
 - More prosthetic heart valve survivors

Infective Endocarditis (2)

- Valvular involvement (MATP)
 - Mitral > aortic > tricuspid (IVDA) > pulmonic
- Native valve endocarditis: age > 50, M > F
 - Predisposing factors: rheumatic heart disease (<20%), calcifications (50%, usually elderly, aortic valve) congenital heart disease (15%),
- IVDA
 - Most have normal valves (75%)
 - Tricuspid valve most common (50%)
 - Staph. aureus is the most common pathogen
- Prosthetic valves
 - Staph. aureus

Infective Endocarditis (3)

- Pathogenesis of IE
 - GU, GI, major dental procedures, IVDA
- Abnormal turbulent flow
- Platelet aggregation and fibrin deposition (sterile vegetations)
- Bacteria colonize sterile vegetations

Infective Endocarditis (4)

Types

- Acute IE
 - Younger, normal valves in half
 - Virulent strains
 - Higher morbidity and mortality
 - Staph. aureus
- Subacute IE
 - Older, abnormal valves
 - Anemia of chronic disease
 - Strep. viridans (50-60%)



Infective Endocarditis (5)

Types

- Left sided IE
 - S. viridans, S. aureus
 - Gram negatives (IVDA or contaminated catheters)
 - Cause of death is heart failure
 - Emboli: CNS and systemic infarction
- Right sided IE
 - IVDA, indwelling catheters
 - S. aureus, S. pneumoniae, gram negatives
 - Emboli: pulmonary infarction & infection
 - Less heart failure, mortality rate lower

Infective Endocarditis (6)

Types

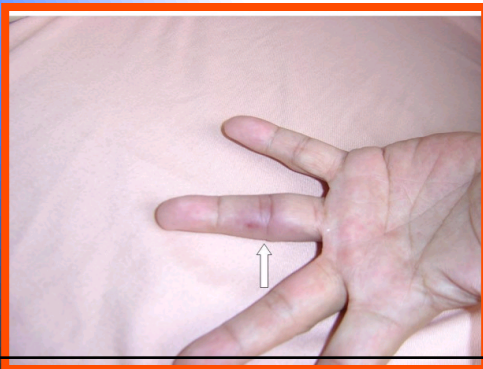
- Prosthetic valve IE
 - Most common during first two months post-op
 - S. epidermidis, S. aureus
 - Late causes similar to native valve endocarditis
 - S. viridans, Serratia, Pseudomonas

Infective Endocarditis (7)

- Etiology
 - Streptococci (various species) are the most common cause with native heart valves
 - Staph aureus most common in IV drug users (IVDA), prosthetic valves (also Staph epidermidis), catheter-related sepsis
 - Gram negatives & fungi (IVDA)
- Findings of IE
 - Fever, chills, "flu-like" illness, back pain
 - Heart murmur
 - Valvular incompetence (the most common cause of acute AR)

Infective Endocarditis (8)

- Embolic and vasculitic components
 - Osler nodes: tender nodules on the tips of the fingers and toes (Osler = Ow!)
 - Janeway lesions: nontender, hemorrhagic plaques on the palms and soles
 - Roth spots: retinal hemorrhages with central clearing
 - Petechiae and splinter hemorrhages



Septic Emboli



Janeway Lesion



Osler Nodes



Splinter Hemorrhages



Roth Spots



Infective Endocarditis (9)

- Laboratory
 - Two blood cultures: > 90% sensitivity if bacteremic
 - Anemia and elevated ESR
- Diagnosis: ultrasound (TEE) for vegetations
- Treatment: penicillins, vancomycin, aminoglycosides

Infective Endocarditis (9)

- Surgical indications
 - Refractory CHF
 - Valve ring abscess
 - Multiple major emboli
 - Medical failure
 - Mycotic aneurysm

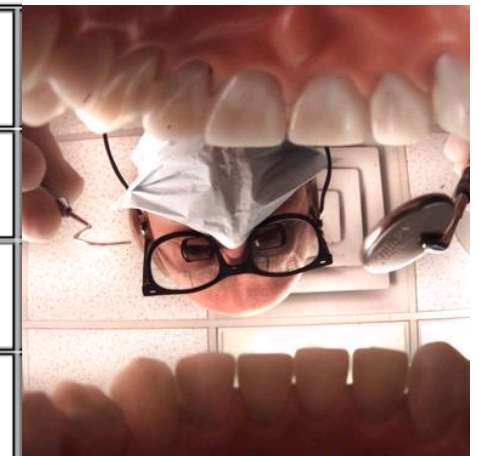
Infective Endocarditis (10)

- **Prophylaxis**
- **High-risk cardiac conditions**
 - Prosthetic cardiac valve
 - History of infective endocarditis
 - Congenital heart disease (CHD)
 - Cardiac transplantation recipients with cardiac valvular disease

Infective Endocarditis (11)

- Major dental and invasive respiratory only
- GU/GI: No prophylaxis

Description	Drug	Adult Dose [*]
Standard general prophylaxis	Amoxicillin	2 g PO
Unable to take oral medication	Ampicillin	2 g IV/IM
Allergic to penicillin	Clindamycin	600 mg PO
	Cephalexin [†] or other first- or second-generation oral cephalosporin in equivalent dose	2 g PO
	Azithromycin or clarithromycin	500 mg PO
Allergic to penicillin and unable to take oral medication	Clindamycin	600 mg IV
	Cefazolin or ceftriaxone [†]	1 g IV/IM



Rheumatic Heart Disease (1)

- Diagnosis of rheumatic fever: Jones criteria
 - 2 major criteria or
 - 1 major and 2 minor
 - Plus evidence of preceding Strep infection (scarlet fever, positive group A strep throat culture, elevated ASO titer)

Degenerative valvular disease is the most common cause of valvular disease overall

Rheumatic Heart Disease (2)

- Jones major criteria (CASES)

Carditis

Arthritis

Sydenham's chorea

Erythema marginatum

Subcutaneous nodules

- Jones minor criteria
 - Previous history of RF or RHD
 - Arthralgia
 - Elevated ESR
 - Prolonged PR interval
 - Fever

Aortic Stenosis (1)

- Etiology
 - Usually congenital bicuspid valve, or acquired through RHD
- Pathophysiology
 - Small valve orifice → LV outflow obstruction → fixed output → unable to meet higher demands

**Often presents with exertional dyspnea,
chest pain and syncope
(exertional syncope = think aortic stenosis)**

Aortic Stenosis (2)

- Physical exam
 - Carotid pulse is weak, with a slow rate of rise
 - Systolic murmur radiating into neck
 - Hypotension is a late finding
- EKG
 - LVH with strain pattern
 - LBBB
- CXR
 - LVH
 - LAE

Aortic Stenosis (3)

- Natural history
 - LVH leads to heart failure
 - Sudden death from dysrhythmias and acute LV failure
- Medical management
 - Avoid strenuous exertion
 - Treat CHF
 - Avoid nitrates and vasodilators (may drop BP precipitously)
 - Endocarditis prophylaxis (s/p replacement)
- Surgical management
 - Valve replacement

Aortic Regurgitation (1)

- Etiology
 - Acute: endocarditis, dissection
 - Chronic: RHD; also seen with Marfan's syndrome
- Pathophysiology of AR
 - Incompetent valve → run-off into LV → volume overload → LV dilation → CHF (usually no symptoms until CHF develops)

Acute MI + acute AR: rule out dissection

Aortic Regurgitation (2)

- Physical exam
 - Diastolic decrescendo murmur at left lower sternal border
 - “Water hammer” (bounding) pulse
 - Duroziez's murmur (to-and-fro femoral murmur)
 - Quincke's sign (pulsatile blushing of nail beds)
 - Austin-Flint murmur (diastolic murmur 2° to AR with regurgitant jet hitting mitral valve)
- EKG: LVH, LAE
- CXR: LV and LA enlargement

Aortic Regurgitation (3)

- Natural history
 - Symptoms late in disease course
 - Prognosis much worse after symptoms develop
- Complications include
 - CHF
 - Dysrhythmias
 - Infective endocarditis
- Medical management
 - Afterload reduction
 - Inotropes (digoxin)
 - Endocarditis prophylaxis (s/p replacement)

Mitral Stenosis (1)

- Pathophysiology
 - Small valve orifice → LV inlet obstruction → LAE → increased LA pressure → pulmonary edema → increased right sided pressures and RV failure → loss of atrial kick with A-fib may cause acute decompensation

RHD is the most common cause of mitral stenosis

Mitral Stenosis (2)

- Symptoms
 - DOE (or dyspnea in pregnancy, anemia, infection)
 - Orthopnea, PND, hemoptysis
- Physical exam
 - Diastolic rumble in mitral area
 - Loud S1, opening snap
- EKG: LAE, A-fib
- CXR: LAE, CHF

Suspect MS if murmur and pulmonary edema are present in pregnancy

Mitral Stenosis (3)

- Natural history
 - Gradual onset of CHF
- Complications include
 - Systemic emboli
 - CHF
 - A-fib
 - Endocarditis
- Medical management
 - Treat A-fib with rate control and anticoagulation
 - Endocarditis prophylaxis (s/p replacement)
- Surgical management
 - Commissurotomy
 - Valve replacement

Mitral Regurgitation (1)

- Etiology of acute MR
 - Chordae tendineae rupture (trauma, IE, MI)
 - Papillary muscle dysfunction (ischemia, MI)
- Etiology of chronic MR
 - MVP
 - Dilated cardiomyopathy
 - RHD
 - Mitral annulus calcification

Mitral Regurgitation (2)

- Pathophysiology of acute MR
 - Sudden mitral valve incompetence → sudden increase LA pressure → pulmonary edema → RV pressure overload and right sided failure
- Pathophysiology of chronic MR
 - Gradual valve incompetence → gradual LA pressure increase → LAE compensation → gradual CHF → late right sided failure

Mitral Regurgitation (3)

- Symptoms
 - Acute: dyspnea, CHF, right-sided failure
 - Chronic: CHF (compensated)
- Physical exam
 - Acute: loud holosystolic murmur (may have no murmur if chordae rupture, resulting in wide-open valve)
 - Palpable LV heave and thrill
 - EKG: LAE, LVH
 - CXR: cardiomegaly, CHF

Mitral Regurgitation (4)

- Acute complications
 - Acute pulmonary edema
 - Cardiogenic shock
- Chronic complications
 - CHF
 - A-fib

Mitral Regurgitation (5)

- Medical management of acute MR
 - Afterload reduction
 - IABP to temporize until surgery
- Medical management of chronic MR
 - Symptomatic treatment of CHF
 - Treatment of A-fib
 - Endocarditis prophylaxis (s/p replacement)
- Surgical management
 - Valve repair or replacement

Mitral Valve Prolapse (1)

- Idiopathic myxomatous degeneration of leaflets (leaflets become too thick to appose well)
- Symptoms
 - Chest pain (atypical)
 - Dyspnea
 - Anxiety
 - Palpitations
- Mostly young women

Mitral Valve Prolapse (2)

- Physical exam
 - Mid-systolic click, late systolic murmur
- EKG
 - Nonspecific ST-T wave changes
 - Ectopy and PSVT common
- CXR
 - Not helpful
- Echocardiogram
 - Diagnostic
 - Regurgitation uncommon (requires prolapse of both leaflets)

Mitral Valve Prolapse (3)

- Natural history
 - Very common. The vast majority never have a problem
 - Increased incidence of arrhythmias
 - Thromboembolic disease
 - Endocarditis rarely
- Management
 - No longer requires endocarditis prophylaxis unless full-blown regurgitation develops
 - Anticoagulation if embolic disease is present
 - Beta blockers helpful for chest pain

Cardiomyopathy

- Group of diseases directly altering cardiac structure, impairing myocardial function
- Three types
 - Dilated cardiomyopathy
 - Hypertrophic cardiomyopathy
 - Restrictive cardiomyopathy

Dilated Cardiomyopathy (1)

- Dilated cardiomyopathy
 - Idiopathic (most common)
 - Alcohol
 - Peripartum
 - Viral (myocarditis)
 - End-stage CAD
 - Sarcoidosis
 - Amyloidosis
 - Hypothyroidism

Dilated Cardiomyopathy (2)

- Pathophysiology
 - Decreased contractility → dilatation of all chambers → decreased output
- Clinical presentation
 - CHF (biventricular failure)
 - Emboli
 - Dysrhythmias
 - Sudden death
- CXR
 - Globular heart
 - CHF
- EKG
 - LVH
 - LAE
 - Conduction defects
 - A-fib

Dilated Cardiomyopathy (3)

- Treatment
 - Largely supportive
 - Diuretics
 - Afterload reduction
 - Anticoagulation
 - Antidysrhythmics
 - Transplantation

Hypertrophic Cardiomyopathy (1)

- Often familial autosomal dominant
- Asymmetric thickening of septum causing two problems
 - Noncompliant ventricle with decreased diastolic filling
 - Dynamic obstruction of LV outflow (with mitral valve leaflets blocking outflow tract)

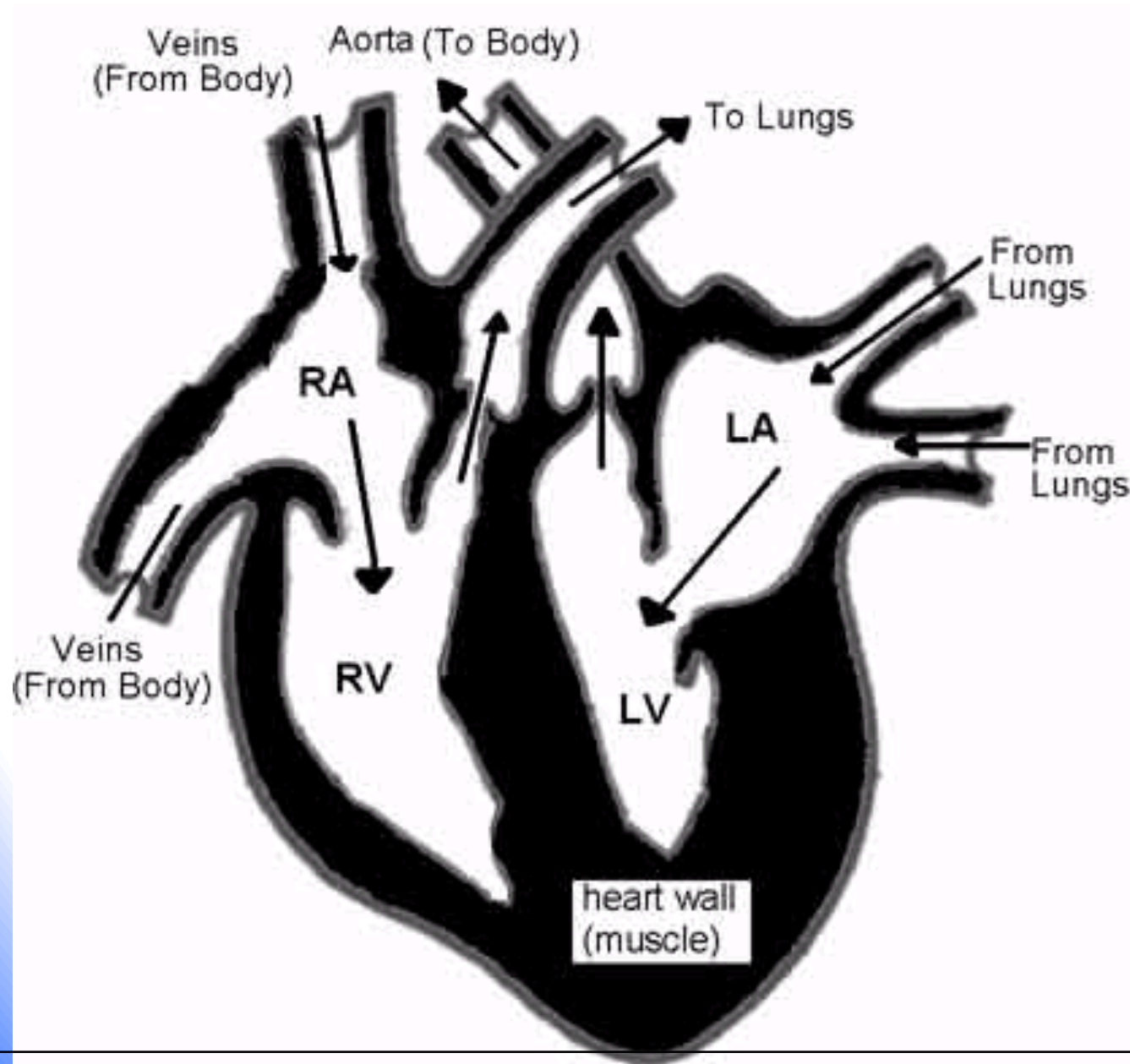
Hypertrophic Cardiomyopathy (1)

- Clinical manifestations
 - Exertional syncope
 - Sudden death
 - Cardiac ischemia
 - Dysrhythmias

Hypertrophic Cardiomyopathy (2)

- Physical exam
 - Harsh, mid-systolic murmur at LLSB
 - Murmur louder with decreased preload (hypovolemia, standing, Valsalva, amyl nitrite, beta agonists)
 - Murmur decreased with increased afterload (squatting, Trendelenburg, hand grip, volume expansion, alpha agonists)

Hypertrophic Cardiomyopathy (3)



Hypertrophic Cardiomyopathy (4)

- EKG: hypertrophy in septal leads
- CXR: nondiagnostic
- Treatment of hypertrophic cardiomyopathy
 - Avoid exertion
(worsens obstruction and leads to arrhythmias)
 - Negative inotropes (beta blockers, calcium channel blockers) to decrease obstruction
 - Never use digoxin or positive inotropes
(increased obstruction)
 - Surgical myomectomy

Pericarditis (1)

- Etiology
 - Infectious
 - Viral (most common)
 - Bacterial
 - TB
 - Fungal
 - Acute MI (Dressler's syndrome)
 - Connective tissue disease
 - Neoplasm
 - Uremia
 - Radiation

Pericarditis (2)

- Clinical manifestations
 - Chest pain (may radiate to trapezius)
 - Chest pain increases with inspiration and swallowing
 - Dysphagia
 - Relief on sitting up, bending forward (and may hear the rub better in this position)
 - Fever, malaise

Pericarditis (3)

- Physical exam
 - Rub (increased by leaning forward)
 - Tachycardia, pulsus paradoxus (an exaggerated BP response to breathing – BP goes down on inspiration and up on expiration)

Pericarditis (3)

- EKG: four stages
 - Stage 1: Diffuse ST elevation (does not correspond to coronary artery distribution) & PR segment depression
 - Stage 2: ST-segments and PR return to baseline
 - Stage 3: T wave inversions
 - Stage 4: Normalization of EKG

Acute Pericarditis



**ST elevation diffusely
PR segment depression**

Acute Pericarditis



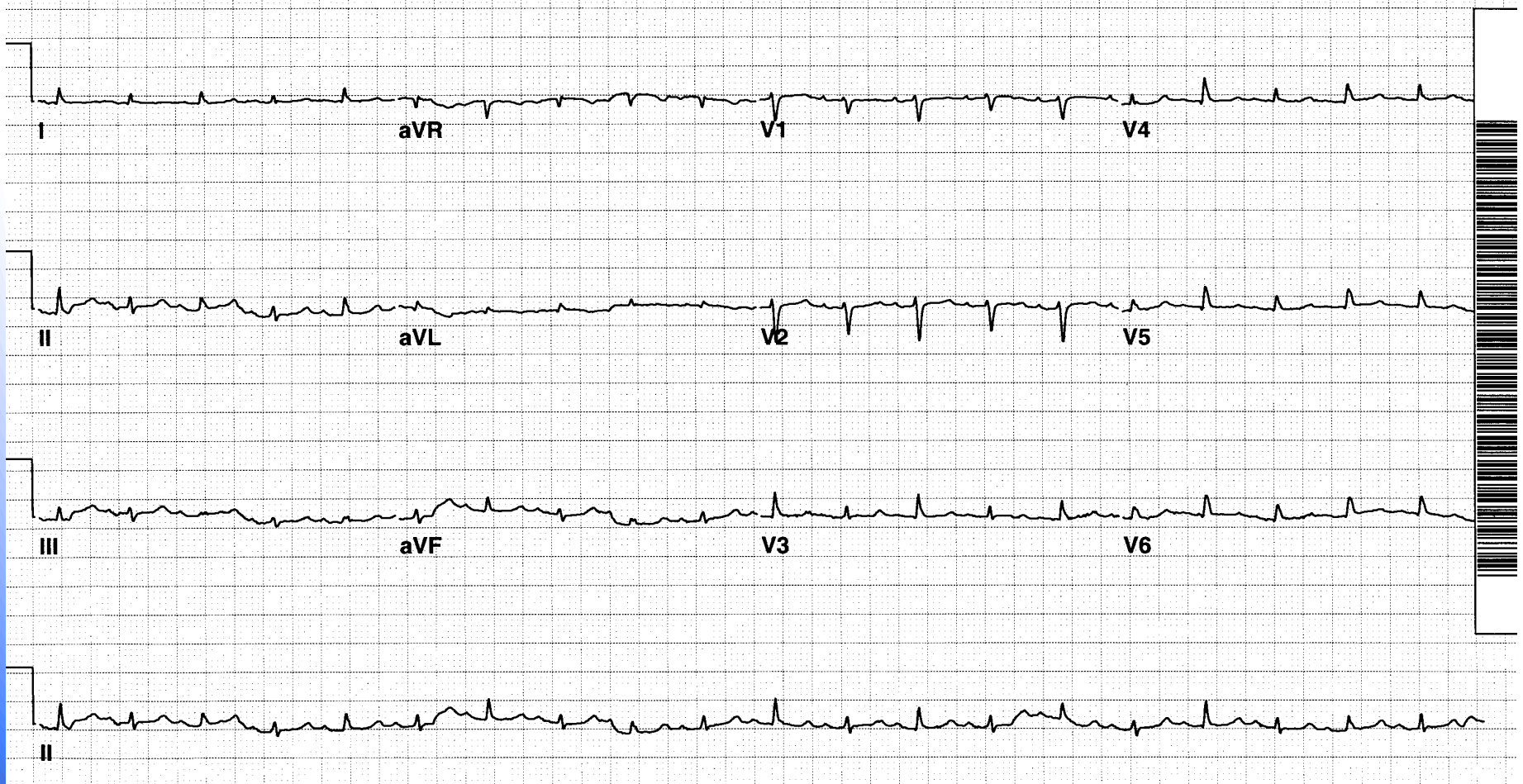
Pericarditis (4)

- CXR: usually normal
- Echocardiogram: pericardial effusion
- Complications
 - Dysrhythmias (atrial)
 - Large pericardial effusions, tamponade
 - Residual pericardial constriction
- Treatment
 - Treat underlying cause if possible
 - ASA, NSAIDs, colchicine; steroids controversial

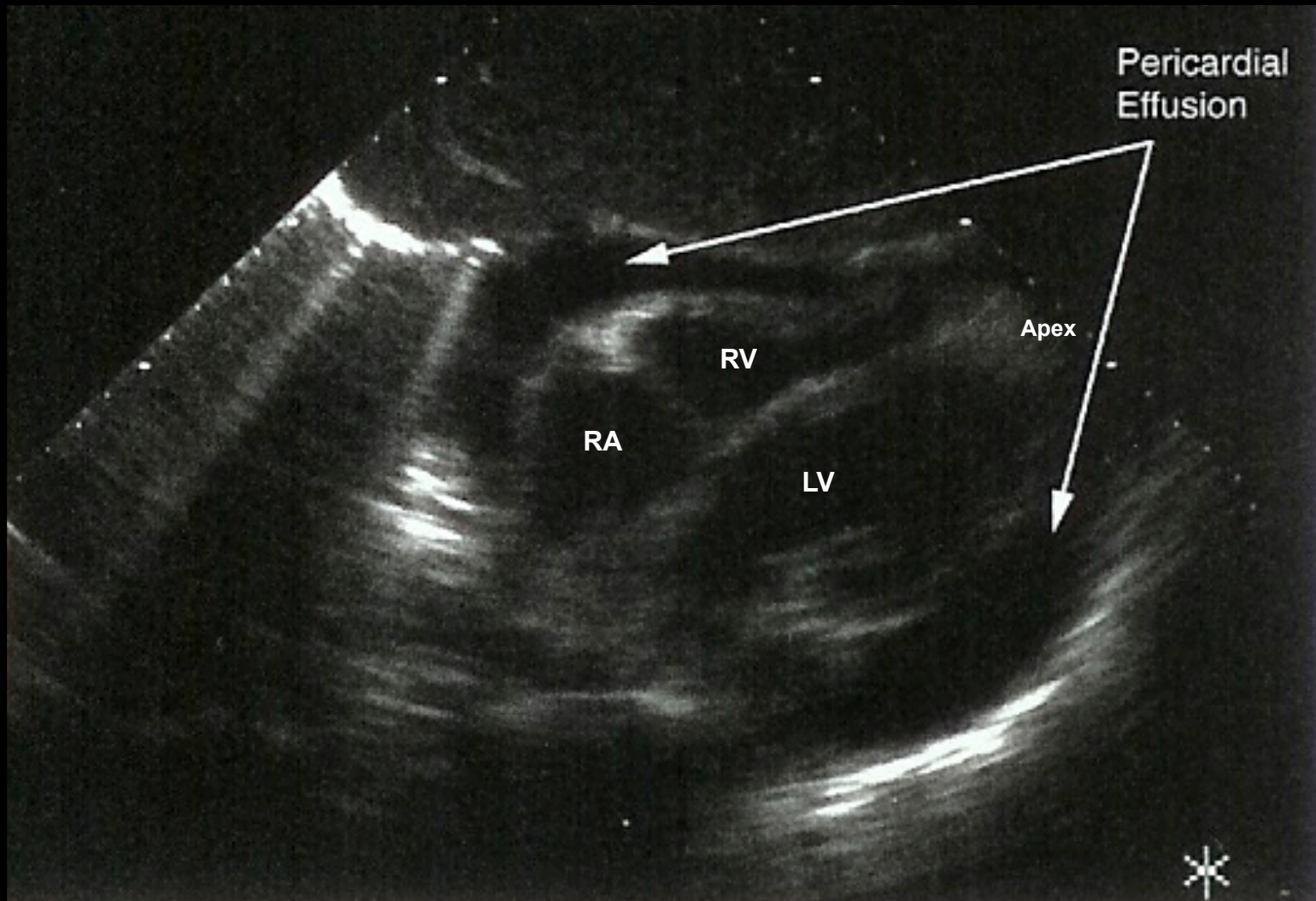
Pericardial Tamponade

- Causes
 - Trauma, uremia, anticoagulation, neoplasm
- Clinical signs
 - Hypotension, JVD, muffled heart sounds, pulsus paradoxus
- EKG
 - Electrical alternans (beat to beat alteration in the amplitude of the QRS complex), low voltage
- ECHO findings
 - Effusion
 - RV diastolic collapse (specific for tamponade)

Pericardial Effusion



Pericardial Effusion



Myocarditis (1)

- Inflammation of the myocardium
 - Often associated with pericarditis
- Etiology
 - Idiopathic
 - Infectious (usually viral, especially coxsackie B virus)
 - Chemotherapy
 - Connective tissue disease

Myocarditis (2)

- Clinical manifestations
 - “Flu-like” illness
 - Fever, sinus tach (out of proportion to exam)
 - Symptoms of CHF
 - Dysrhythmias
 - Emboli
 - Sudden death
- EKG: nonspecific changes, conduction blocks
- CXR: possibly enlarged heart
- Lab: elevated biomarkers

Myocarditis (3)

- Natural history
 - Most recover
 - May have sudden death
 - May have dilated cardiomyopathy
- Treatment
 - Supportive care

Hypertension (1)

- Definition (JNC VII)
 - Normal: Systolic lower than 120, diastolic lower than 80
 - Prehypertension: Systolic 120-139, diastolic 80-89
 - Stage 1: Systolic 140-159, diastolic 90-99
 - Stage 2: Systolic equal to or more than 160, diastolic equal to or more than 100
 - Cardiovascular risk: BP > 120/80

Hypertension (2)

Definition / Classification

- Hypertensive urgency (DBP > 110)
 - No signs or symptoms of organ damage
 - Acute intervention may be harmful
 - Behavior modification, initiation of therapy
 - Close follow up, e.g. in several days

Hypertension (3)

Definition / Classification

- Hypertensive emergency
 - Malignant hypertension
 - End-organ and CNS signs
 - Immediate BP reduction

Hypertensive Emergencies (1)

- Severe elevation of BP with associated end-organ damage

No specific BP level

- End-organs include
 - Heart (ACS, CHF, dissection)
 - Brain (encephalopathy, hemorrhage)
 - Eyes (hemorrhages, exudates)
 - Kidneys (failure, preeclampsia)

Hypertensive Emergencies (2)

- Types
 - Encephalopathy
 - Stroke syndromes
 - Malignant hypertension (eyes)
 - Acute pulmonary edema
 - Acute coronary syndromes
 - Aortic dissection
 - Acute renal failure
 - Preeclampsia, eclampsia
 - Catecholamine crises

Hypertensive Emergencies (2)

- Pediatric hypertension emergencies
 - Blood pressure $> 95^{\text{th}}$ percentile
 - Common causes: renovascular, nephritis, pheochromocytoma

Hypertensive Emergencies (3)

- Malignant hypertension
 - Hypertension with end-organ damage and papilledema
 - Not defined by absolute blood pressure reading
 - Goal is to reduce mean arterial pressure by 30% in 30 minutes

$$\text{MAP} = \text{DBP} + (\text{SBP} - \text{DBP}) / 3$$

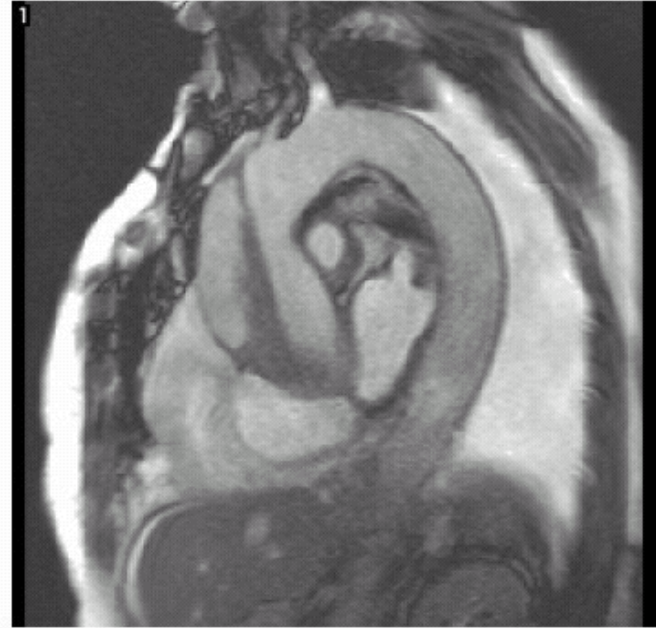
Treatment is nitroprusside and/or labetalol

Hypertensive Emergencies (4)

- CNS: encephalopathy, hemorrhagic CVA
 - Nitroprusside
 - Labetalol
- Ischemic stroke
 - Hypertension usually resolves within hours
 - Transient and cerebroprotective
 - Treatment: observe, labetalol, nicardipine
- Pregnancy-induced hypertension, eclampsia
 - Hydralazine
 - Labetalol
 - Magnesium sulfate for seizures

Hypertensive Emergencies (5)

- Cardiac: angina, CHF
 - IV nitroglycerin
 - Nitroprusside
- Aortic dissection
 - Need to decrease rate of rise of BP (dP/dT) to decrease shear forces on aorta
 - Beta blockers (esmolol, labetalol, propranolol), then nitroprusside



Hypertensive Emergencies (6)

- Catecholamine surge (pheochromocytoma, MAOI crisis, cocaine overdose)
 - Alpha plus beta blocker is best
 - Do not use beta blocker alone
(avoid unopposed alpha effect)
 - Labetalol plus phentolamine

Hypertensive Emergency Medications (1)

- Sodium nitroprusside
 - Mode of action: arterial and venous dilatation
 - Onset of action: 1-2 minutes
 - Half life: 3-4 minutes
 - Metabolized to thiocyanate (cyanide)
 - Ideal medication for hypertensive emergencies (rapid onset, potent, short half life)
 - Can cause reflex tachycardia, therefore use with beta blocker

Hypertensive Emergency Medications (2)

- Labetalol
 - Alpha and beta blocker (primarily beta)
 - Onset of action: 5-10 minutes
 - Half life: 5.5 hours
 - No reflex tachycardia
 - Low doses may lead to paradoxical hypertension due to predominant beta effect (unopposed alpha)

Contraindicated in bronchospasm, CHF,
AV-blocks

Hypertensive Emergency Medications (3)

- Nitroglycerin
 - Arteriolar and venous dilatation
 - Onset: immediate
 - Half life: 4 minutes
 - Ideal for cardiac emergencies such as CHF, MI
 - Side effects: headache and tachycardia

Hypertensive Emergency Medications (4)

- Hydralazine
 - Direct arteriolar vasodilator
 - Onset: 10 min (IV)
 - Half life: 2-4 hours
 - Indicated in pregnancy-related hypertension, pediatric nephritis

Side effects include
reflex tachycardia (limits use in CAD, dissection)
Chronic use associated with “lupus-like” syndrome

Syncope (1)

- Defn. = Sudden, brief LOC and postural tone with spontaneous recovery due to a decrease in cerebral blood flow
- Causes:
 - Neurally mediated (reflex-mediated HR or vascular tone changes):
 - Vasovagal (18%), situational (5%), carotid sinus (1%)
 - Psychiatric causes (2%)
 - Panic attacks, anxiety, somatization
 - Orthostatic hypotension (8%)

Syncope (2)

- Medications (3%)
- Neurologic disease (10%)
- Cardiovascular causes:
 - Organic heart disease (4%), arrhythmia (14%)
 - Vascular – subclavian steal (syncope associated with arm exercise)
- Unknown (34%)
 - Tilt table tests suggest that most of these are neurally mediated

Aortic Dissection (1)

- Dissection of intima from media
- Depends on the rate of rise in blood pressure
- Bimodal age distribution
 - Young with predisposing factors
 - Collagen vascular disorders such as Marfan's
 - Pregnancy (especially third trimester)
 - Chest trauma, iatrogenic (cardiac catheterization)
 - Bicuspid aortic valve
 - Aortic coarctation
 - Elderly males with chronic hypertension
 - Atherosclerotic risk factors (smoking, hypertension, cholesterol, diabetes)

Aortic Dissection (2)

- Clinical presentation
 - Tearing chest pain with radiation to back
 - Migrating, dynamic pain pattern
 - Sudden onset of severe pain
 - Aortic insufficiency
 - Pulse deficits
 - Syncope, acute paralysis
- Physical exam
 - Hypertension, normal BP or hypotension
 - Asymmetric pulses, asymmetric BP
 - Acute aortic regurgitation
 - Tamponade

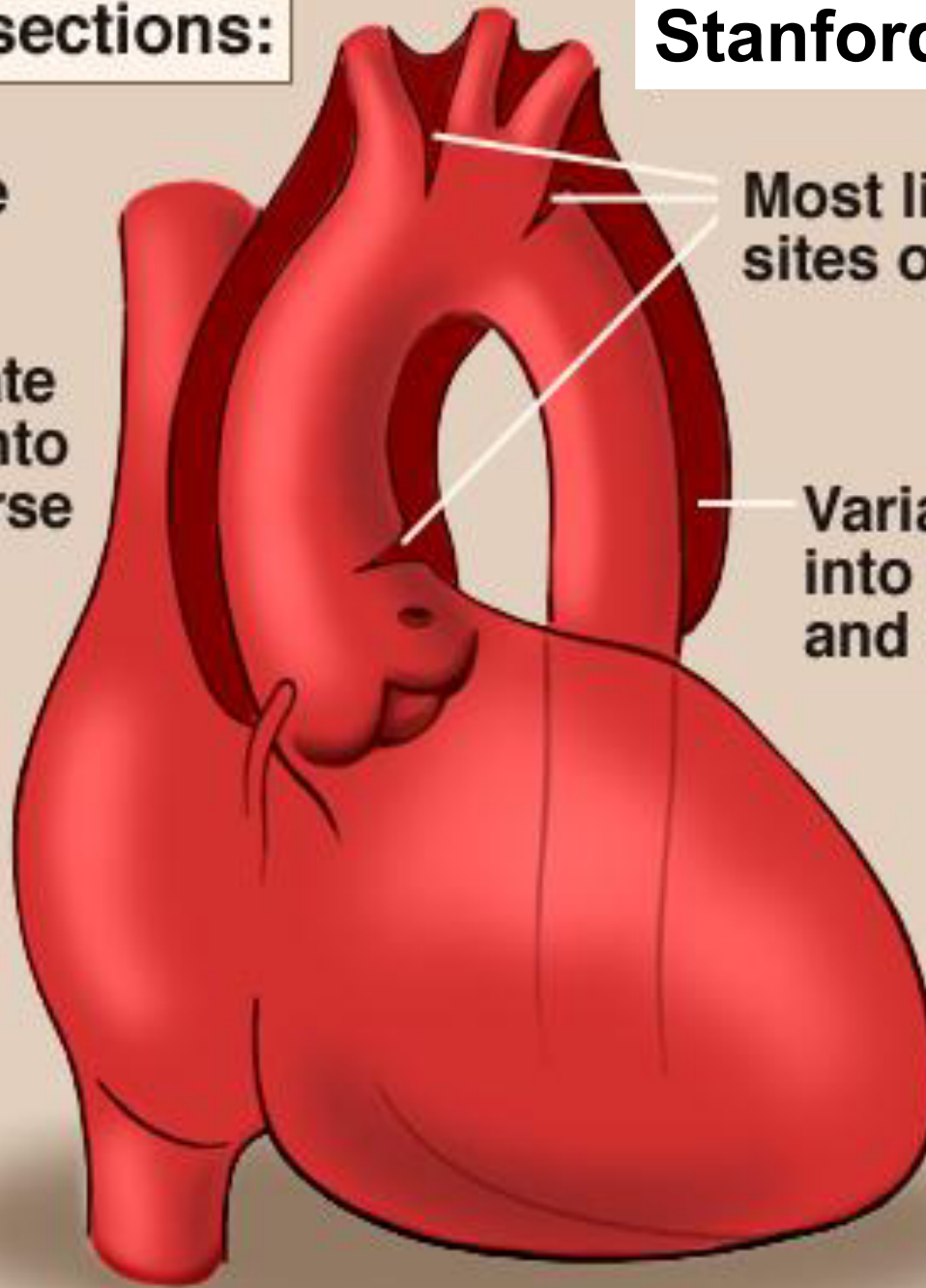
Aortic Dissection (3)

- Stanford classification
 - Type A: any dissection which involves ascending aorta (surgical treatment)
 - Type B: descending aorta only (primarily medical management)

Aortic Dissections:

Stanford Type A

Involves the ascending aorta with indeterminate extension into the transverse and descending aorta.



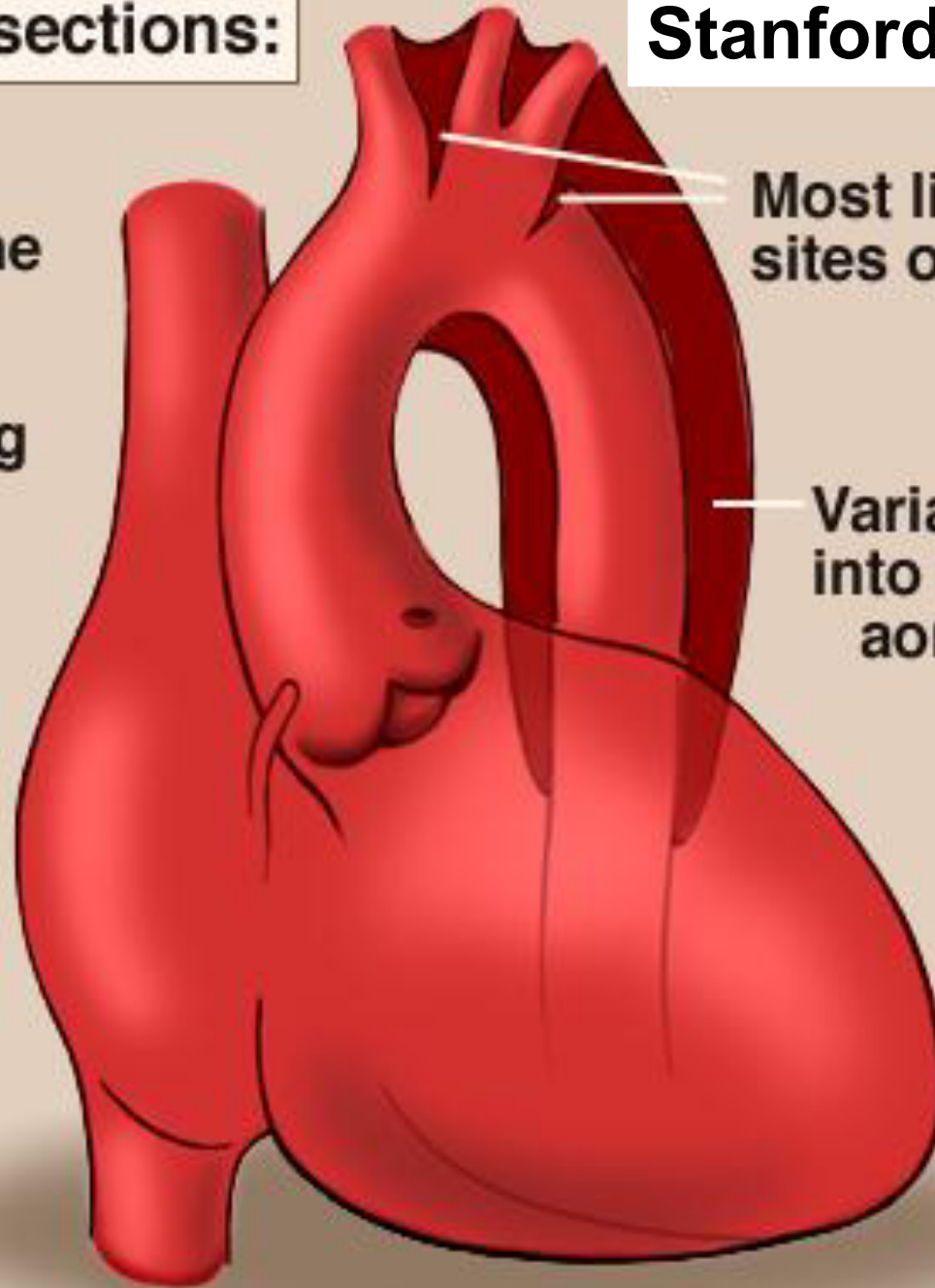
Most likely sites of origin

Variable extension into the transverse and descending aorta

Aortic Dissections:

Stanford Type B

Involves the transverse and/or descending aorta only.

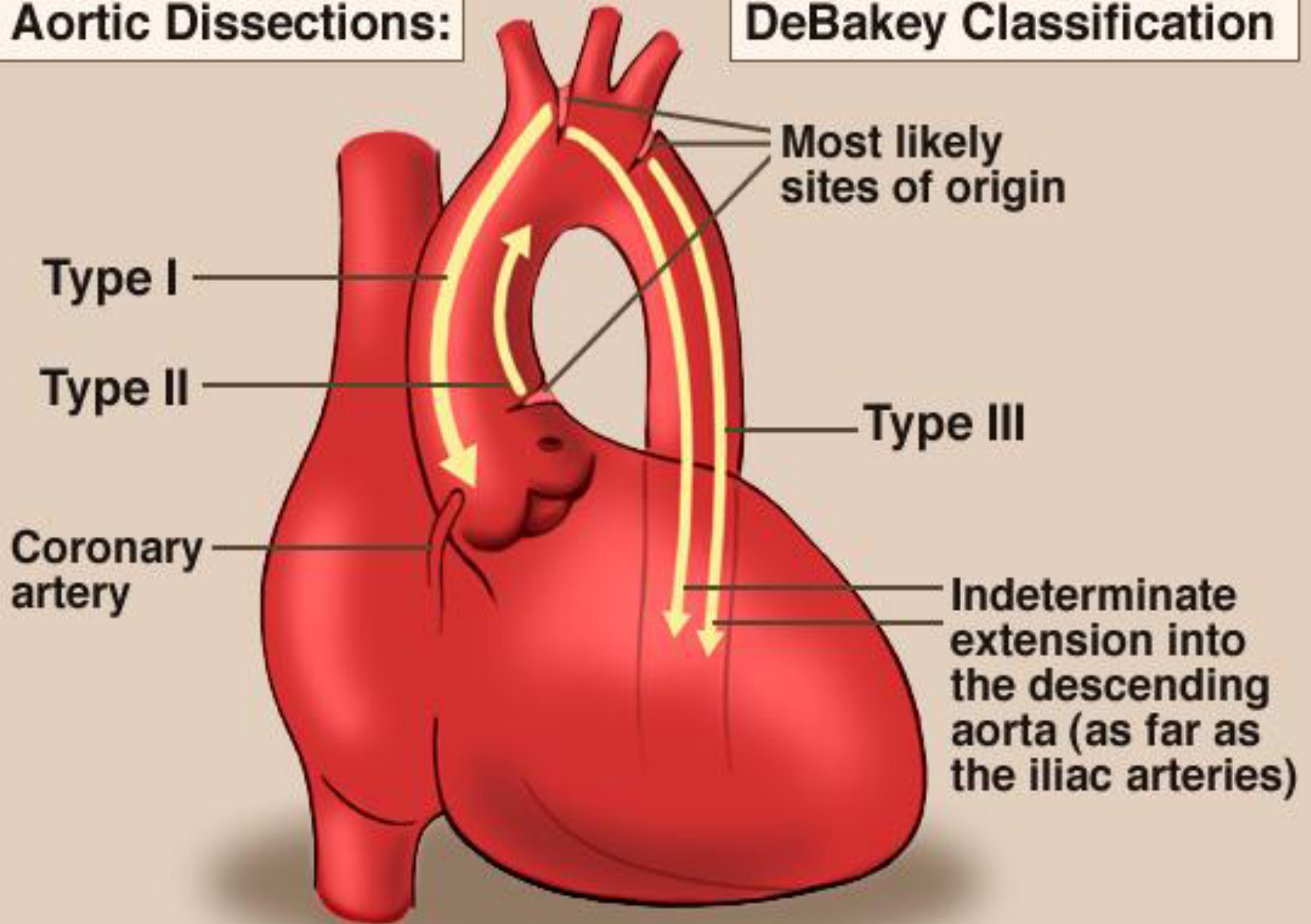


Most likely sites of origin

Variable extension into the descending aorta

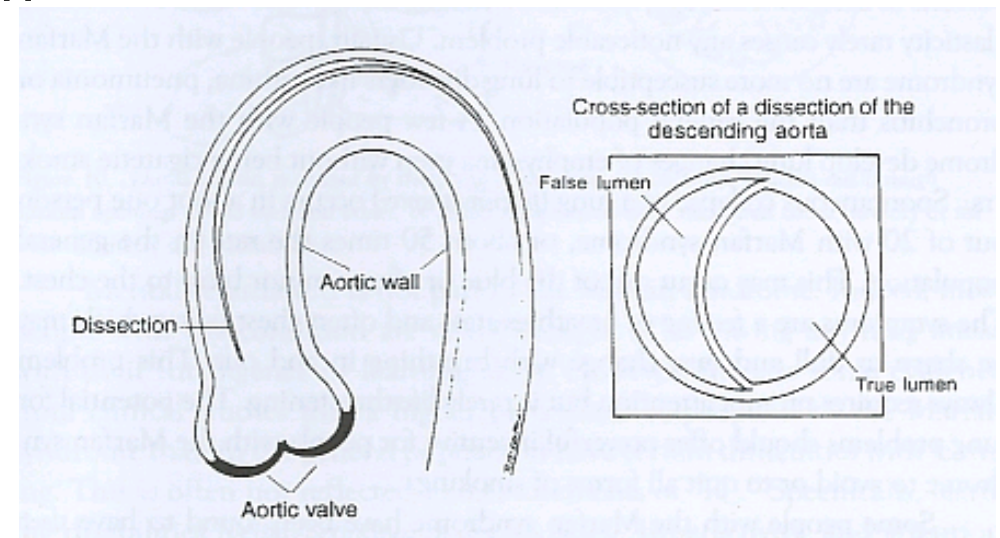
Aortic Dissections:

DeBakey Classification



Aortic Dissection (4)

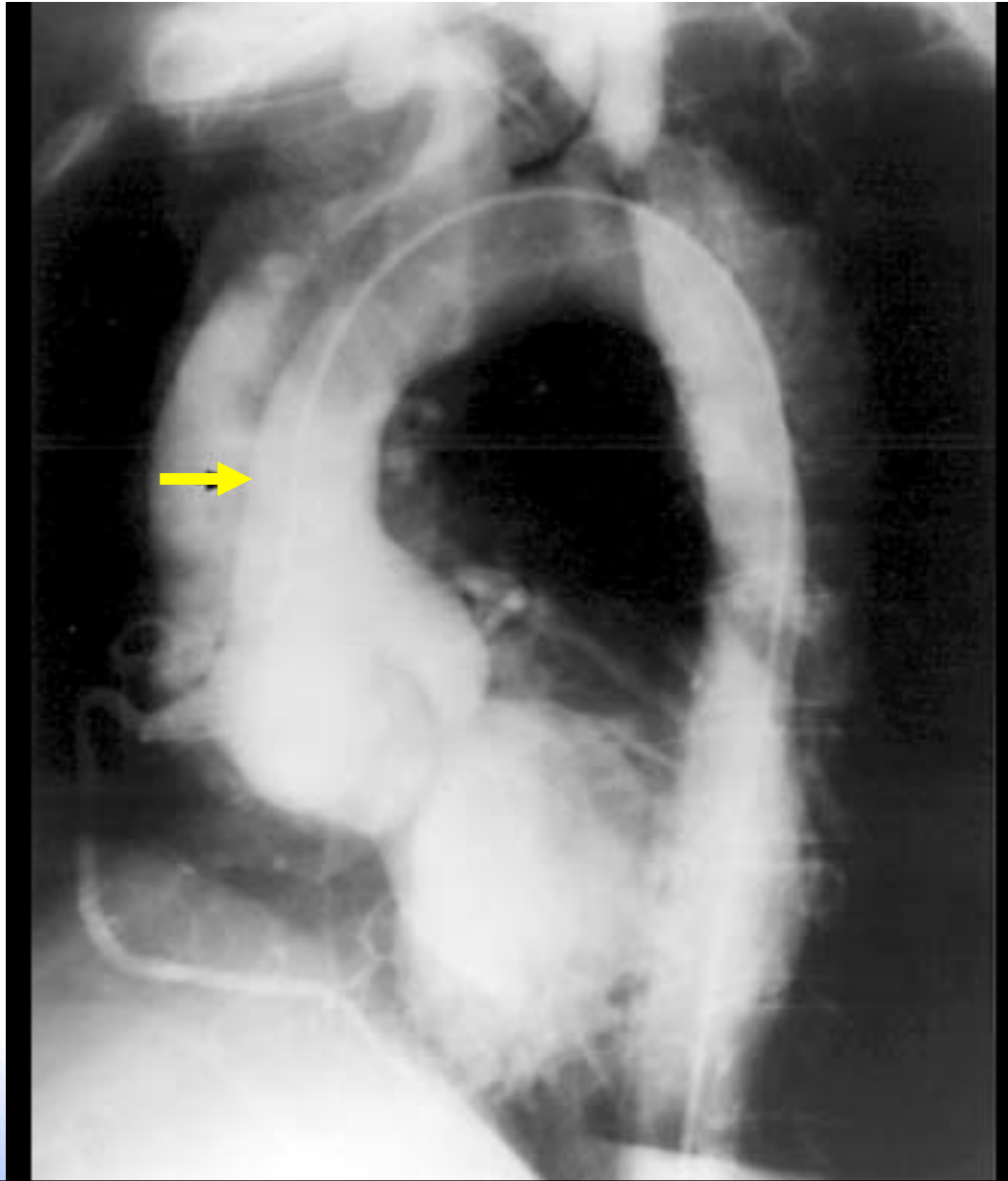
- EKG
 - May show acute MI if dissection is proximal and involves coronary ostia
 - Up to 8% with Type A will have ST elevations
- CXR
 - Widened mediastinum in majority
 - Intimal calcium separation
 - Left pleural effusion



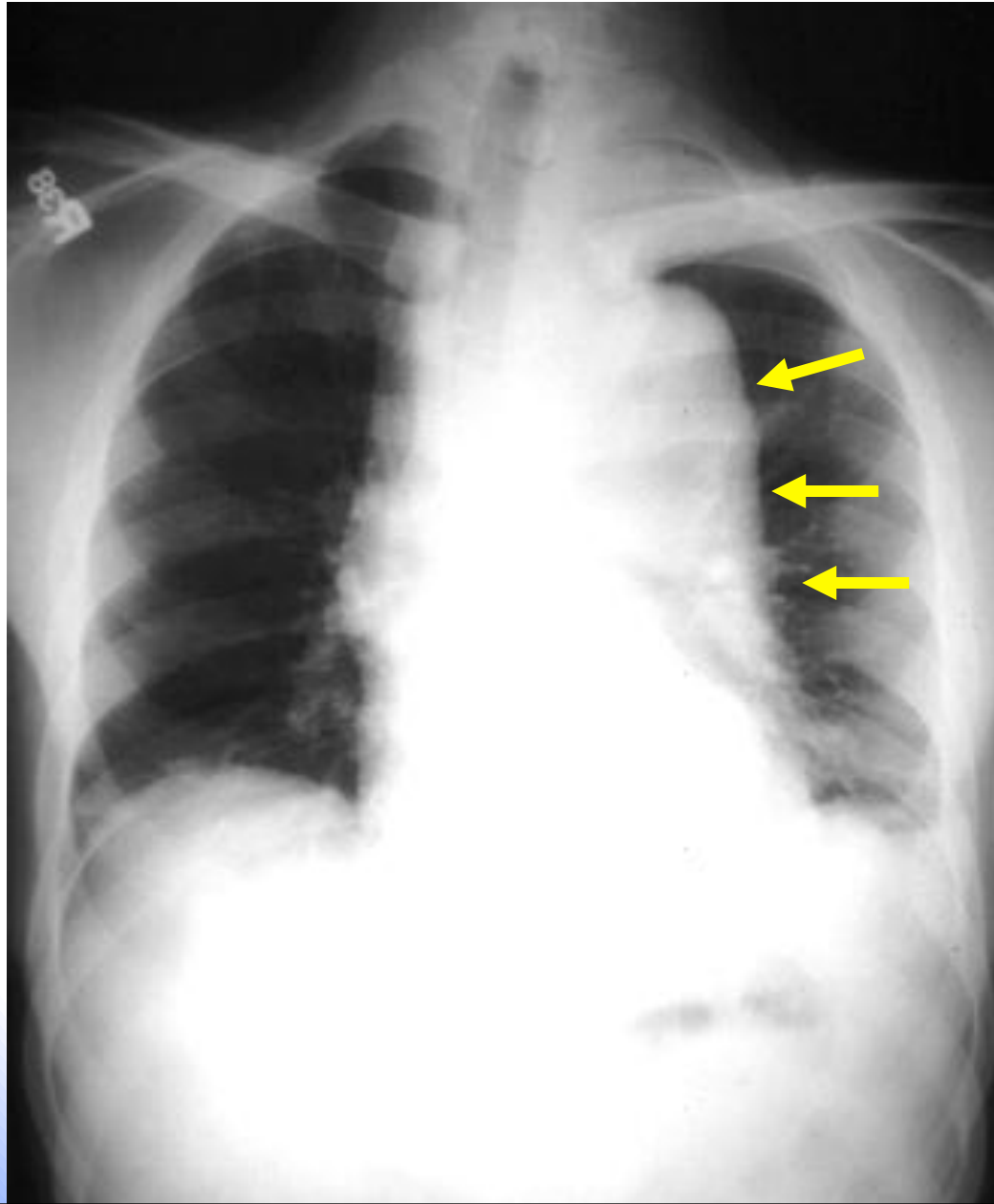
Aortic Dissection (5)

- MRI
 - Very sensitive and specific
 - Usually impractical due to inability to monitor patient and time constraints
- CT with contrast
 - Helpful if negative (high sensitivity) but does not provide details needed for OR
 - Does not show valve competence
 - Shows thrombosed false lumen
 - Requires dye load
- TEE (transesophageal echo)
 - Very sensitive and specific → best test if available
 - Done in ED (safer for patient)

Dissecting Aneurysm



Aortic Dissection



Aortic Dissection (6)

- Aortography
 - Higher risk, logistically difficult and expensive
 - Provides anatomy necessary for OR, including coronary involvement
 - False negatives possible (thrombosed false lumen)
- Treatment
 - Beta blockers, then nitroprusside for both types
 - Stanford A usually requires surgery also
 - Stanford B usually doesn't require surgery

**All types need control of blood pressure
and decreased contractility (decrease dP/dT)**

Abdominal Aortic Aneurysm (1)

- Pathophysiology
 - Increase in diameter $>50\%$ over normal artery
 - Due to medial degeneration (usually atherosclerosis)
 - Majority are infrarenal
- Risk factors
 - Elderly male with atherosclerosis, hypertension
 - Connective tissue disease

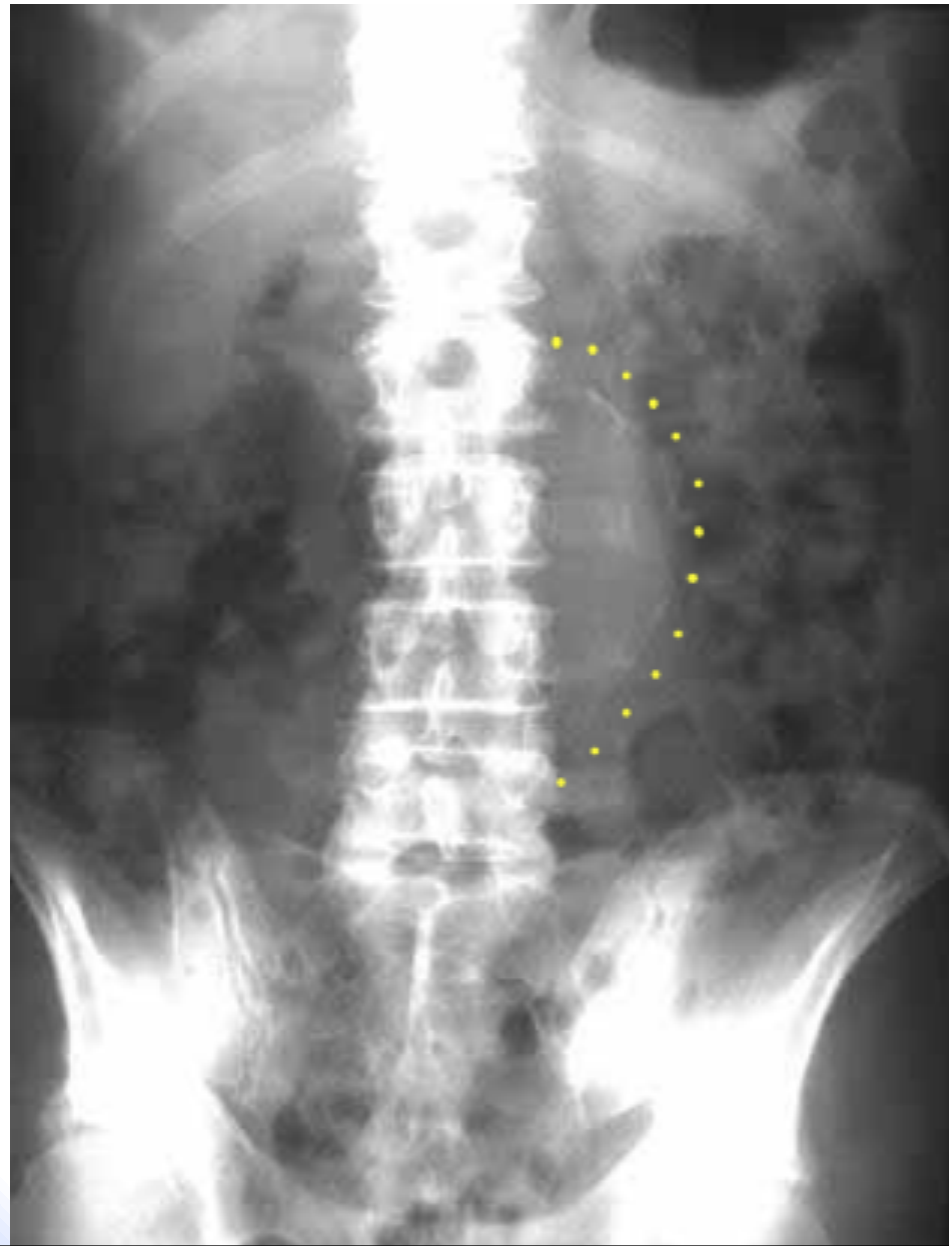
Abdominal Aortic Aneurysm (2)

- Clinical Presentation
 - Asymptomatic until bleed or rupture
 - Abdominal, flank or back pain
 - Most common misdiagnosis: renal colic
 - Syncope
- More unusual presentations
 - Erosion into duodenum → aortoenteric fistula with massive GI bleed
 - Partial bowel obstruction
 - Erosion into IVC → aortocaval fistula with embolization distally

Abdominal Aortic Aneurysm (3)

- Physical exam
 - Pulsatile mass is found in <50%
 - Abdominal or femoral bruits
 - Decreased femoral pulses
- Diagnosis
 - Plain films: rule out calcified aneurysm
 - Abdominal cross table lateral
 - Lateral L-spine

Abdominal Aortic Aneurysm

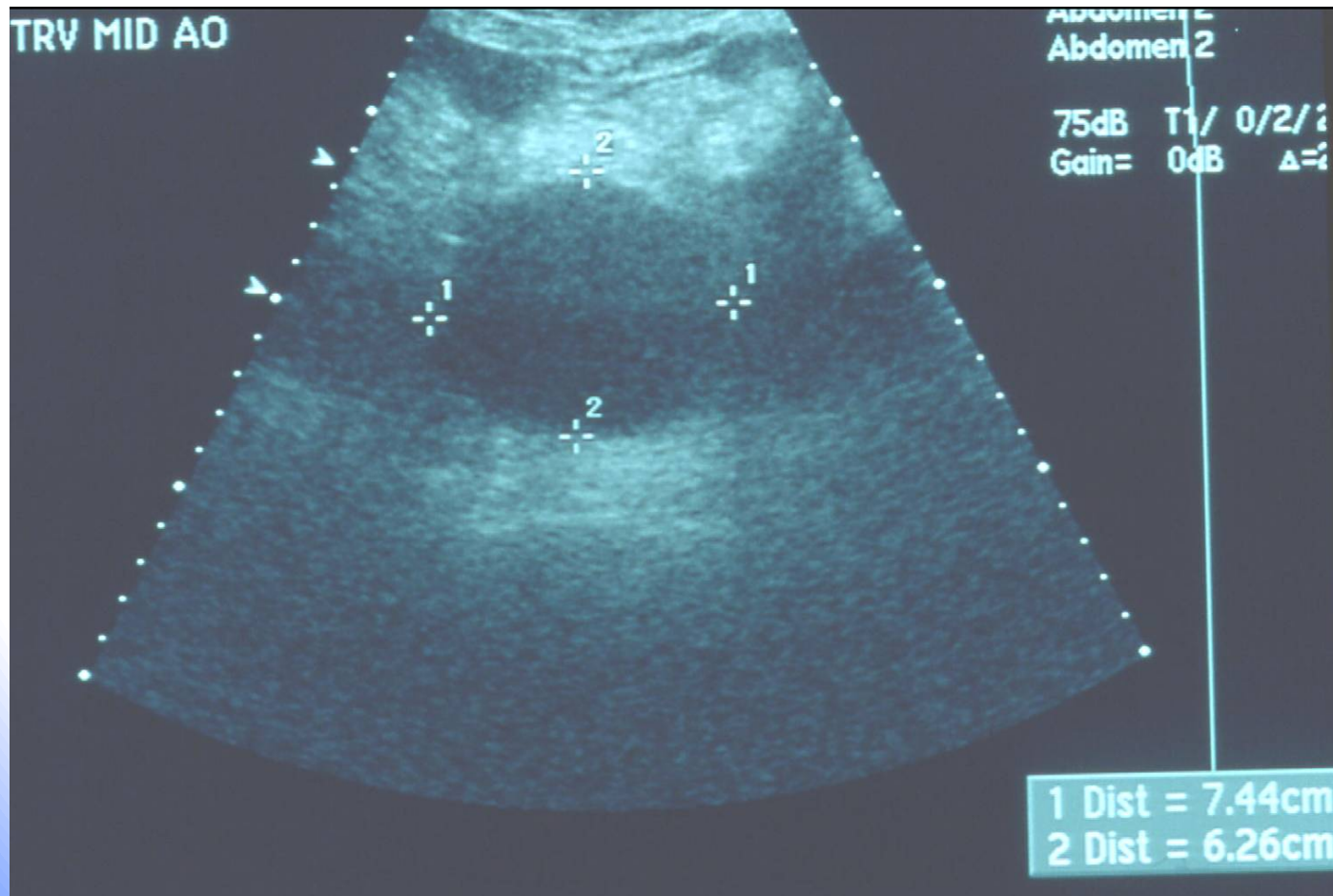


Abdominal Aortic Aneurysm (4)

- Ultrasound
 - Excellent bedside screening tool
 - Very sensitive
 - Unable to determine leakage (unless free fluid)
 - May help to differentiate kidney stone (hydronephrosis, dilated ureter) from aneurysm

Abdominal Aortic Aneurysm (4)

- Ultrasound



Abdominal Aortic Aneurysm (4)

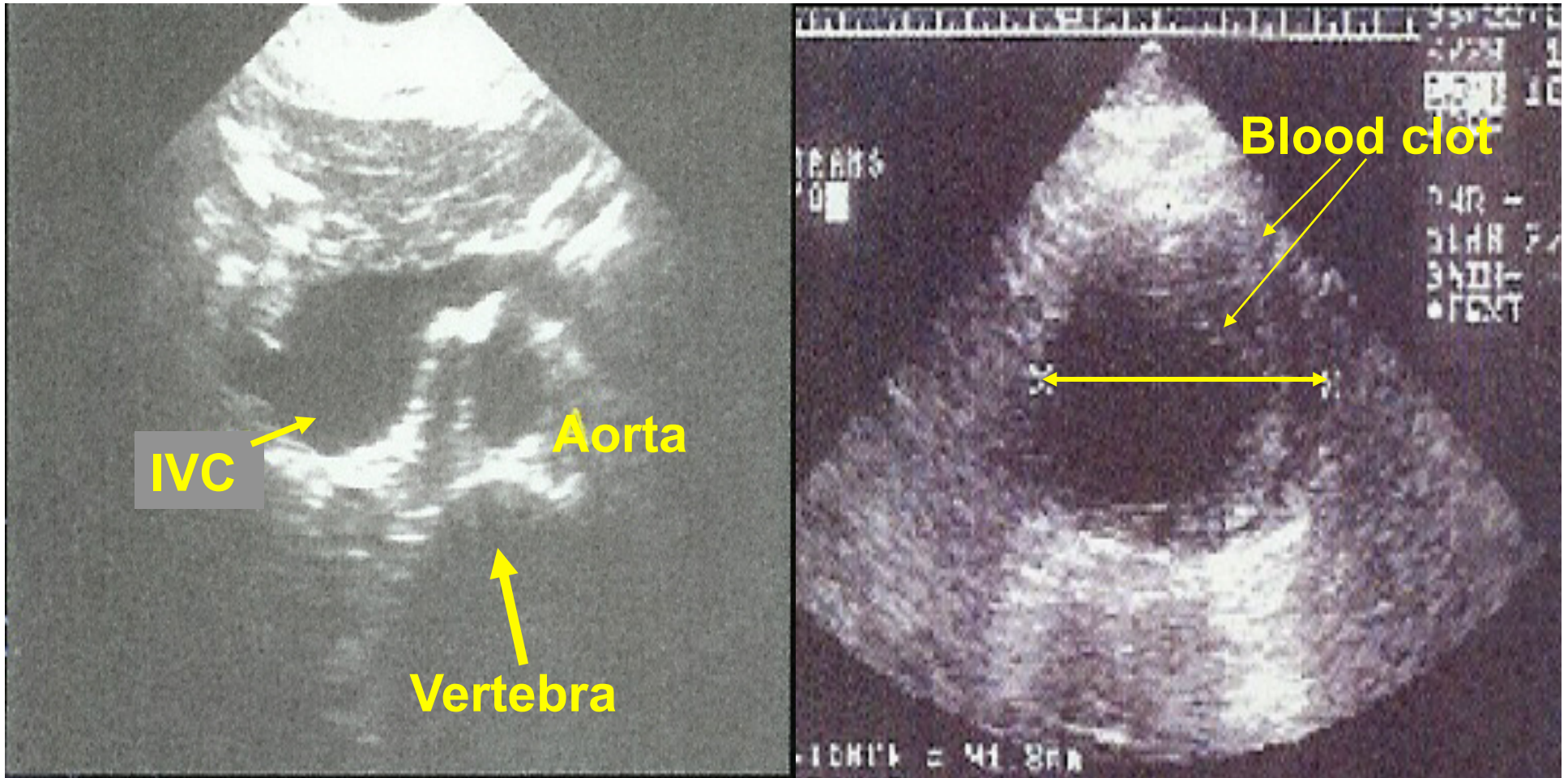
- CT scan
 - Useful in stable patients
 - Able to visualize leakage into retroperitoneal space
 - Risky because patient is out of department

Abdominal Aortic Aneurysm (5)

- Management
 - Hypotensive or unstable
 - Priority is resuscitation and OR (multiple large-bore IVs, type and cross 10 units)
 - Leaking: Emergent operation
 - Asymptomatic: elective repair if >5 cm

Any back or abdominal pain in a patient known to have an aneurysm must be presumed to be leaking or ruptured AAA until proven otherwise

Abdominal Aortic Aneurysm (6)



Normal Aorta < 3 cm

AAA > 3 cm





Acute Limb Ischemia (1)

- Emboli
 - Usually cardiac
 - Mural thrombus from MI
 - A-fib
 - Endocarditis
- Arterial source
 - Aneurysm
 - Dissection
 - Atherosclerotic disease
- Paradoxical embolus
 - From venous emboli through septal defect

Acute Limb Ischemia (2)

- Embolus
 - Sudden onset
 - No previous arterial insufficiency
- Thrombus
 - Etiology: atherosclerosis (most common)
 - Low-flow states
 - Develops slowly
 - Past history of claudication
 - Chronic arterial insufficiency



Acute Limb Ischemia (3)

- Signs of acute ischemia (6 P's)
 - Pain
 - Pallor
 - Paresthesias
 - Paralysis
 - Pulselessness (late finding)
 - Poikilothermia (polar, cold)
- Treatment of acute limb ischemia
 - Heparin
 - Embolectomy, thrombolytics
 - Bypass for atherosclerotic disease

Consider aortic
dissection
in all these cases

Deep Venous Thrombosis (1)

- Pathogenesis (Virchow's triad)
 - Stasis
 - Hypercoagulability
 - Endothelial damage
- Risk factors
 - Immobilization
 - Pregnancy
 - Estrogen use
 - Neoplasm
 - Trauma

Deep Venous Thrombosis (2)

- Presentation
 - Pain and swelling (unilateral)
 - Erythema
 - Low-grade fever
 - Physical exam (e.g. Homans' sign) is insensitive

Deep Venous Thrombosis (2)

- Diagnosis
 - Duplex ultrasonography: very sensitive and specific for proximal thrombi
 - Venography: "gold standard" but invasive and has phlebitis as complication
 - D-dimer (if negative, rules out DVT in low risk patients)
 - CT venography

Deep Venous Thrombosis (3)

- Treatment
 - Heparin therapy initially
 - Then oral warfarin for 3-6 months
 - LWMH allows early outpatient treatment
 - Caval filter when there is a contraindication to, or failure of, long-term anticoagulation
 - Recurrent DVT
 - Emboli

Deep Venous Thrombosis (4)

- Phlegmasia cerulea dolens and phlegmasia alba dolens
 - Uncommon, severe presentation of DVT
 - Massive iliofemoral DVT
 - Acute, severe, massive swelling
 - Cyanotic, congested extremity (cerulea)
 - Pale (alba) if arterial spasm causes “milk leg”
 - Increased compartment pressure, ischemia
 - May require surgery for compartment syndrome

Deep Venous Thrombosis (5)

Phlegmasia cerulea dolens

Phlegmasia alba dolens



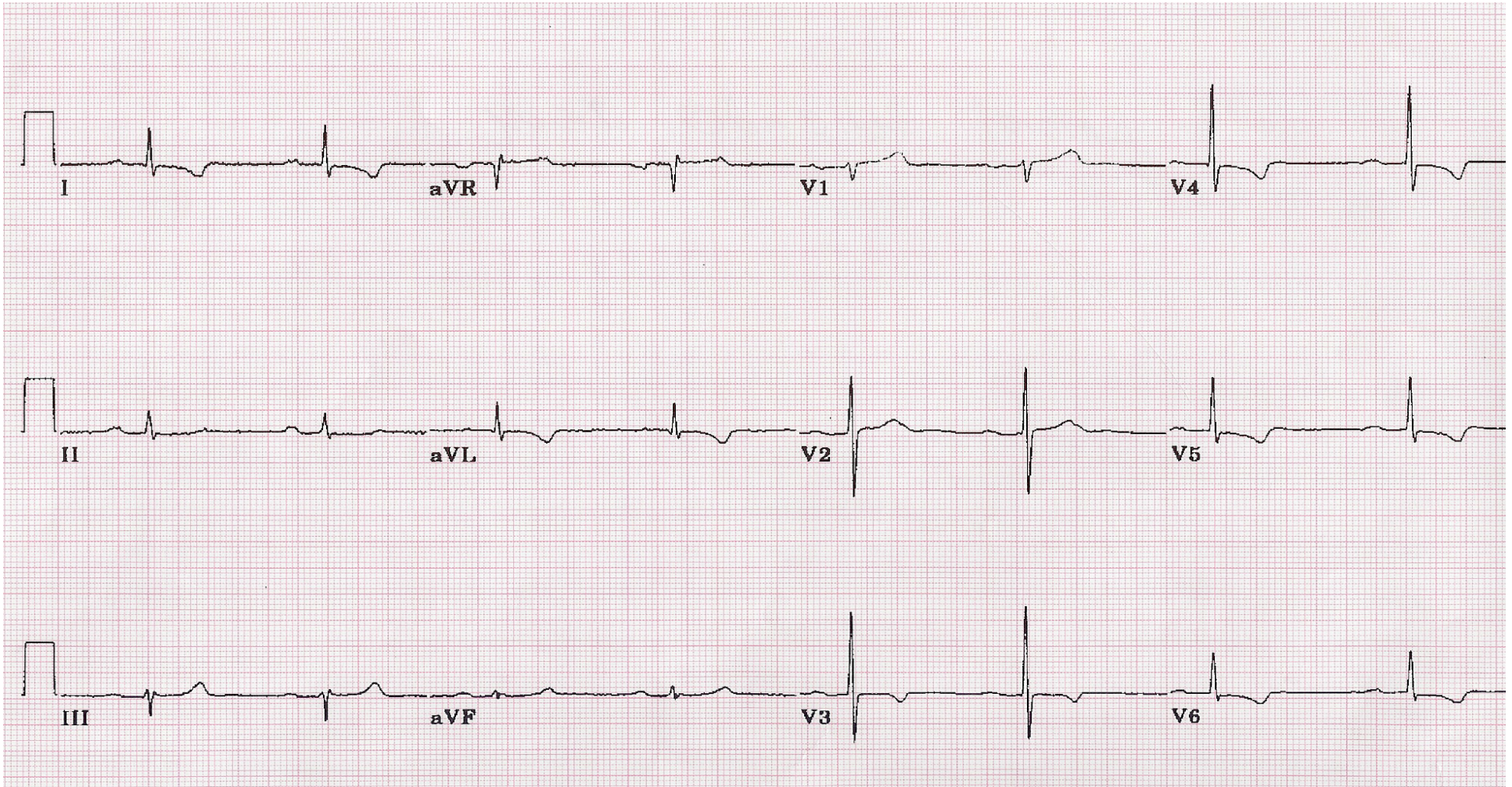
EKGs



EKG - AMI (1)

- Acute Q wave MI (transmural)
 - ST elevation
 - Q waves
 - T wave inversion
- Non-Q wave MI (subendocardial)
 - ST depression with ischemia
 - T wave inversion
- Left ventricle divided into 4 regions
 - Anterior (precordial V leads)
 - Posterior (suggested from V1-V3 or post. leads)
 - Lateral (I, aVL, V5-V6)
 - Inferior (II, III, aVF)

Lateral Ischemia



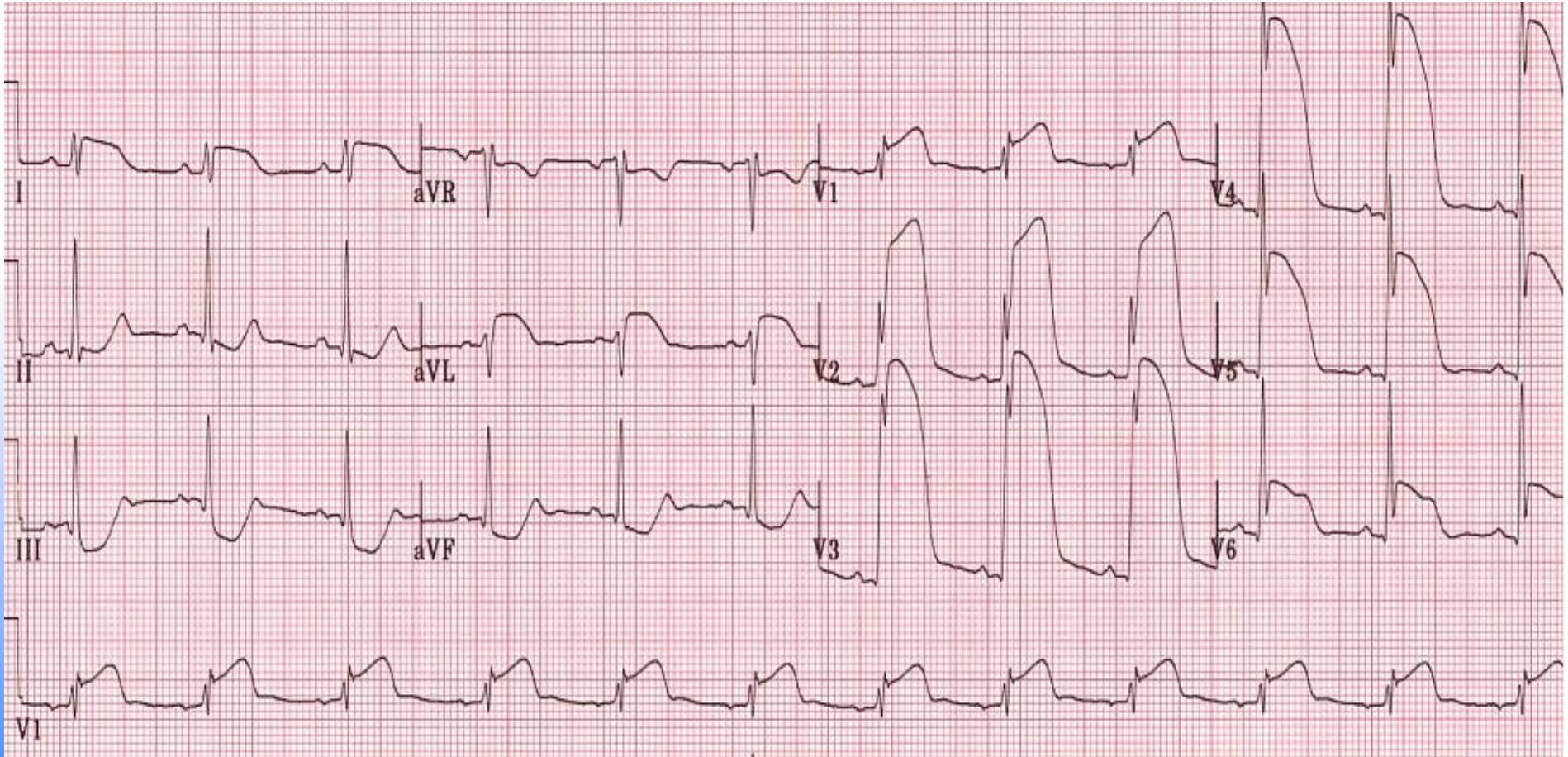
Classic findings:

- ST depression / T wave ↓ in I, aVL, V5, V6

EKG - AMI (2)

- Anterior
 - LAD occlusion
 - ST elevation in any of V1-V4
 - Reciprocal changes inferiorly

Anterior MI



Classic findings:

- ST elevation and poor R wave progression in V1-V4
- Reciprocal changes inferiorly

EKG - AMI (3)

- Anteroseptal
 - Septal branches of LAD
 - ST elevations V1-V2
- Anterolateral
 - LAD or left circumflex
 - ST elevations V5, V6, I and AVL
 - Reciprocal changes inferiorly
- Inferior MI
 - RCA occlusion
 - ST elevations II, III, AVF
 - Reciprocal changes anteriorly

**All potentially
include pathologic
Q waves**

Septal MI



Classic findings:

- ST elevation V1-V2
- Q waves V1-V2

Anterolateral MI



Classic findings:

- ST elevation V1 - V6 +/- I and AVL
- Reciprocal changes inferiorly

Inferior (and posterior?) MI



Classic findings:

- ST elevation in II, III, AVF
- Reciprocal ST changes I and AVL

EKG - AMI (4)

- Lateral MI
 - Circumflex occlusion
 - ST elevations I, AVL, V5, V6
 - Reciprocal changes inferiorly

- Posterior MI
 - Usually accompanies inferior MI due to RCA
 - 4% will be isolated posterior MI
 - Large R waves with ST depressions in V1, V2
 - $R:S > 1$
 - Mirror image of septal MI

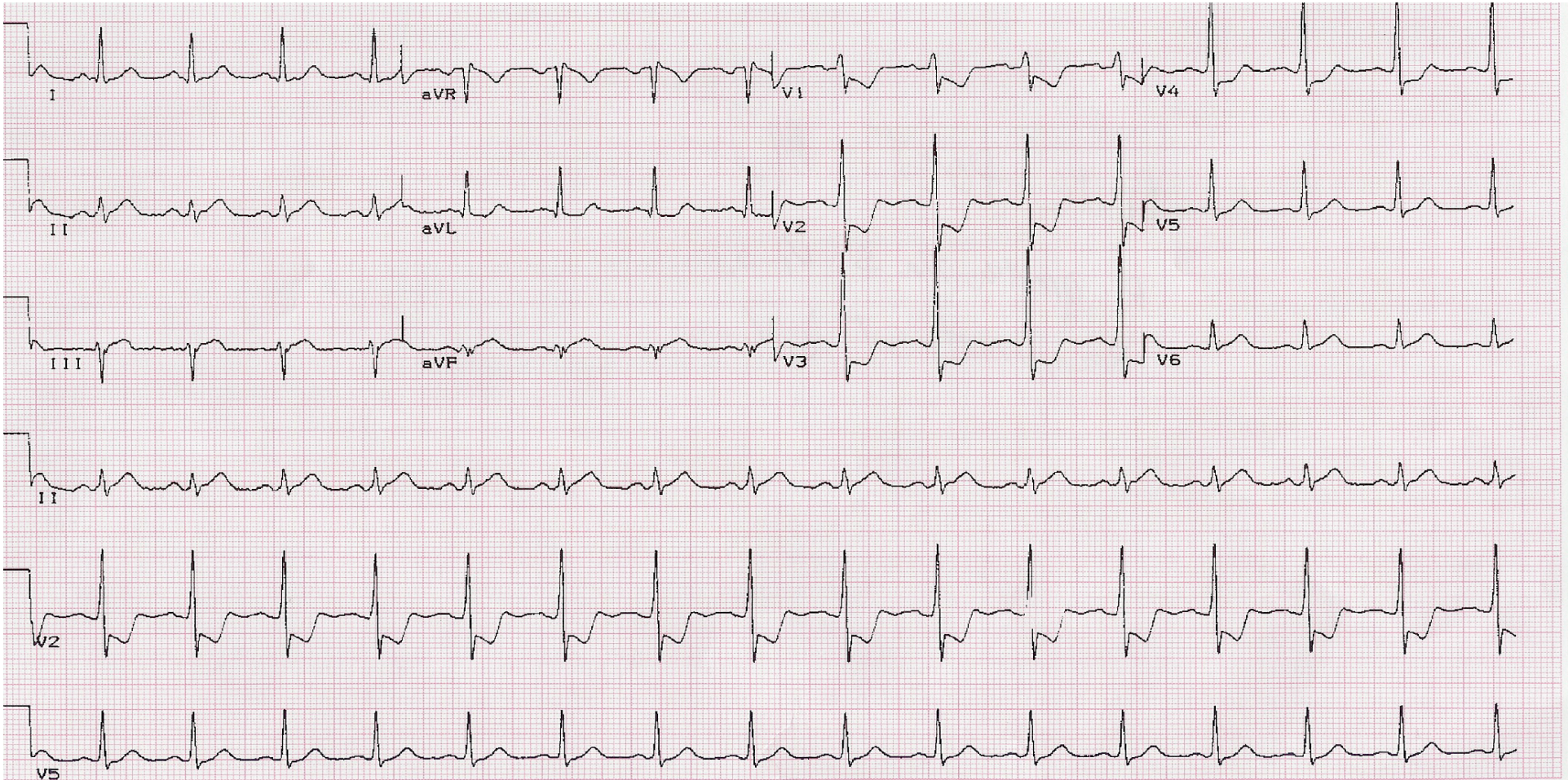
Inferior-Posterior MI



Classic findings:

- ST elevation II, III, AVF
- Large R waves with ST depression in V1 – V3
- Mirror image of septal MI

Isolated posterior MI



Classic findings:

- Large R waves with ST depression in V1, V2
- Mirror image of septal MI

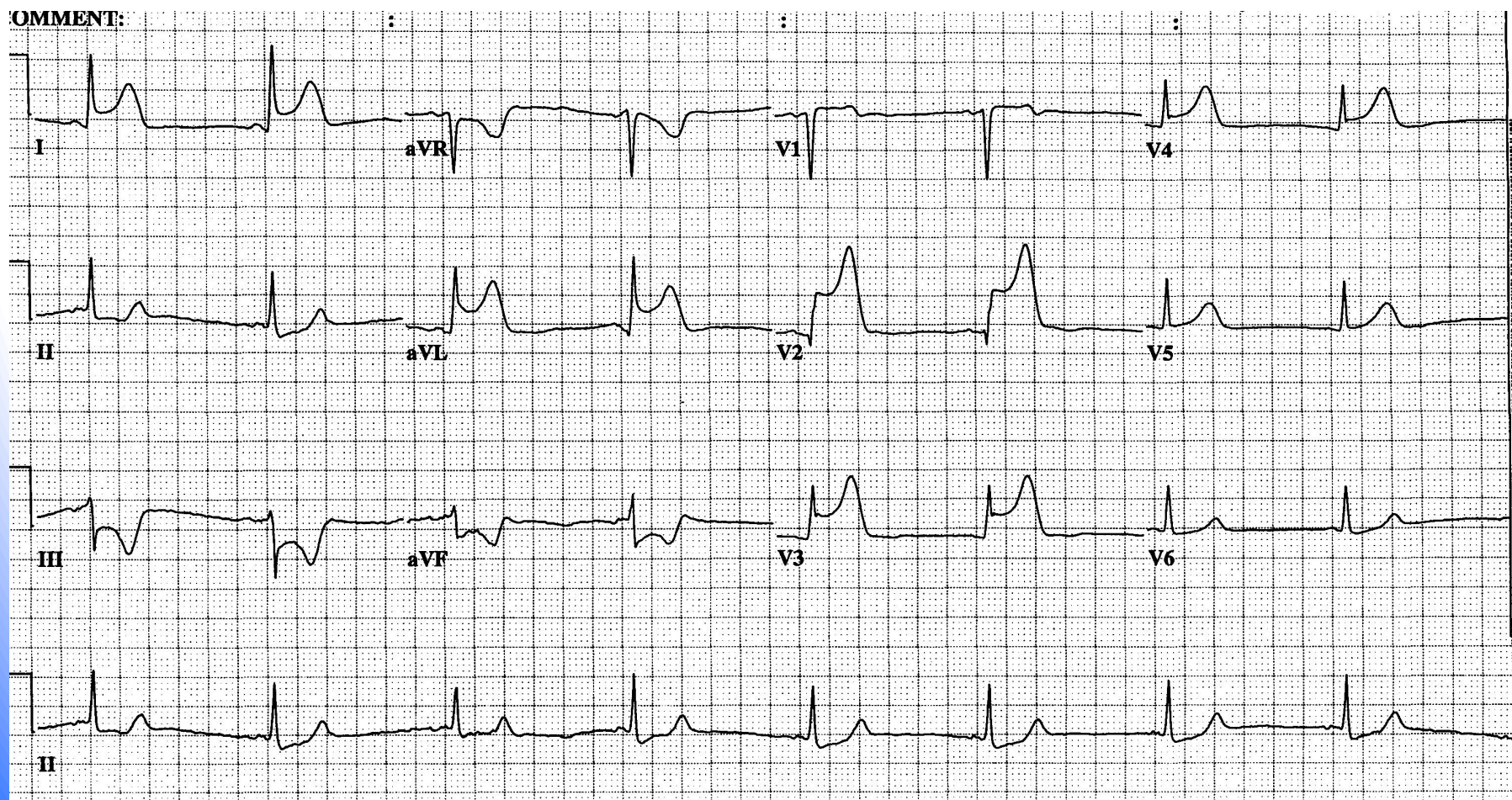
EKG - ST Elevation

- Differential diagnosis of ST segment elevation
 - Acute MI
 - Prinzmetal's angina (vasospasm)
 - Pericarditis
 - Ventricular wall aneurysm
 - Benign early repolarization
 - Bundle branch block

ST Segment Elevation Predictors of MI

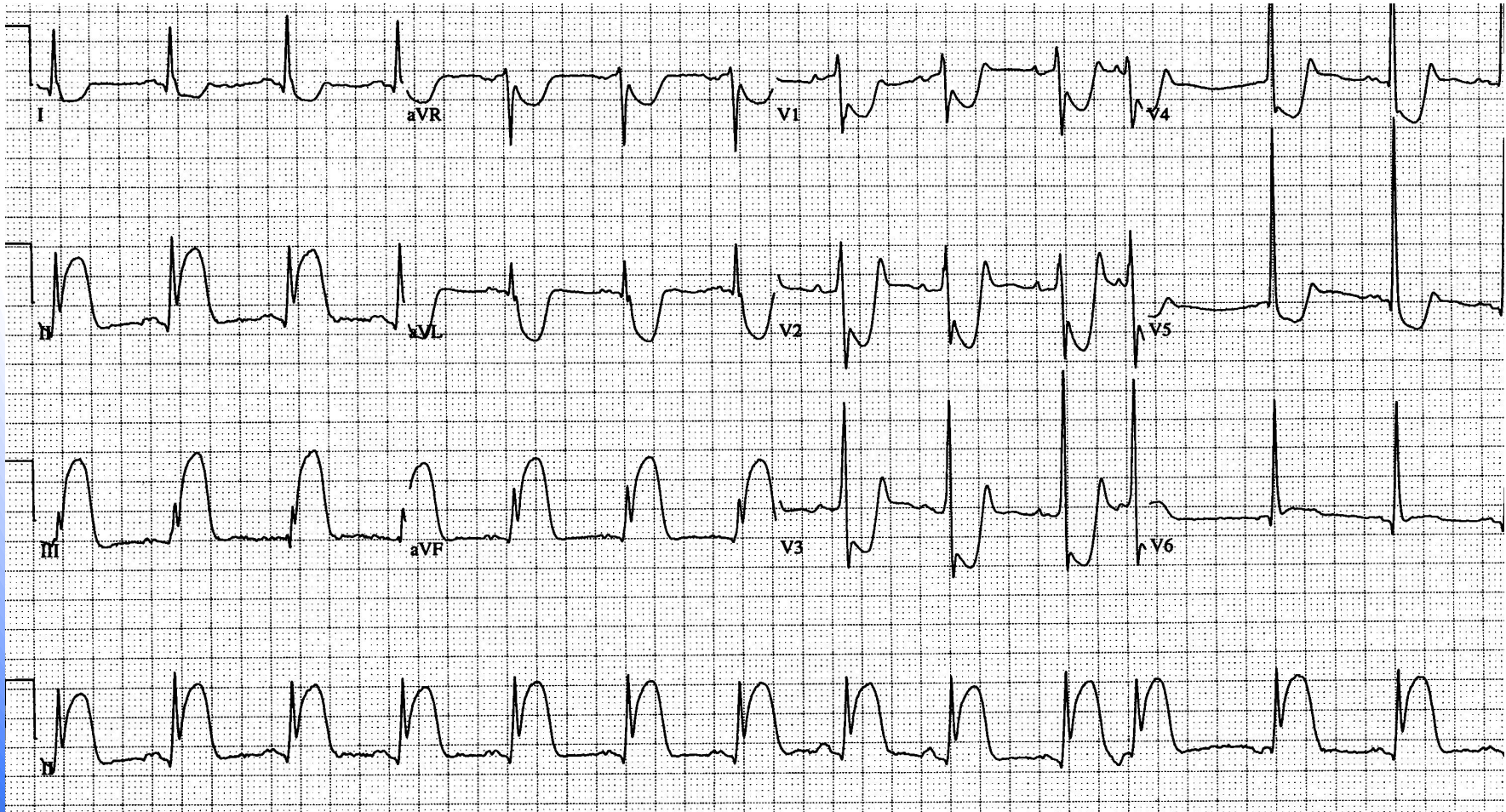
- Factors that increase the specificity
 - ST elevation (horizontal or convex upwards)
 - Follows coronary anatomy
 - Reciprocal ST depression
 - Changes over time (minutes to hours)
 - May see hyperacute T waves (early)
 - Q waves usually only after hours to days

Anterolateral MI: Horizontal ST segments



From *ECGs for the Emergency Physician Vol 1* (Mattu, Brady; Blackwell 2003)

Inferior MI: Convex upward ST segments



From *ECGs for the Emergency Physician Vol 1* (Mattu, Brady; Blackwell 2003)

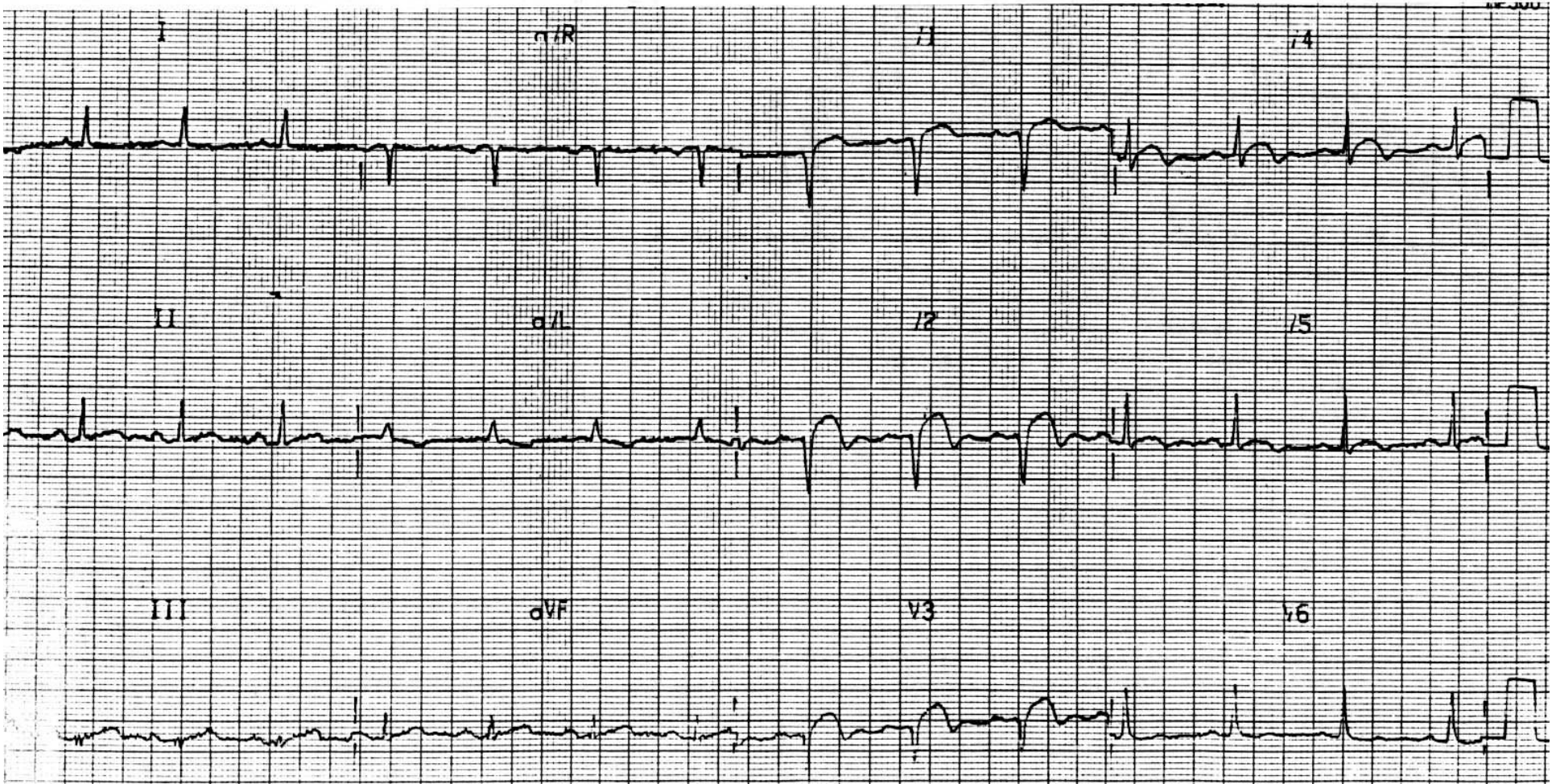
Causes of ST Segment Elevation: Prinzmetal's angina/spasm

- Convex or concave upwards morphology
- Transient over minutes
- More common in women
- Difficult to distinguish from true STEMI

Causes of ST Segment Elevation: Ventricular Aneurysm

- Convex or concave upwards morphology
- Persistent ST elevation
- Q waves usually present
- Usually in anterior leads
- Easily seen on echo
- No reciprocal depression
- Look for old ECGs — no change
- No serial changes

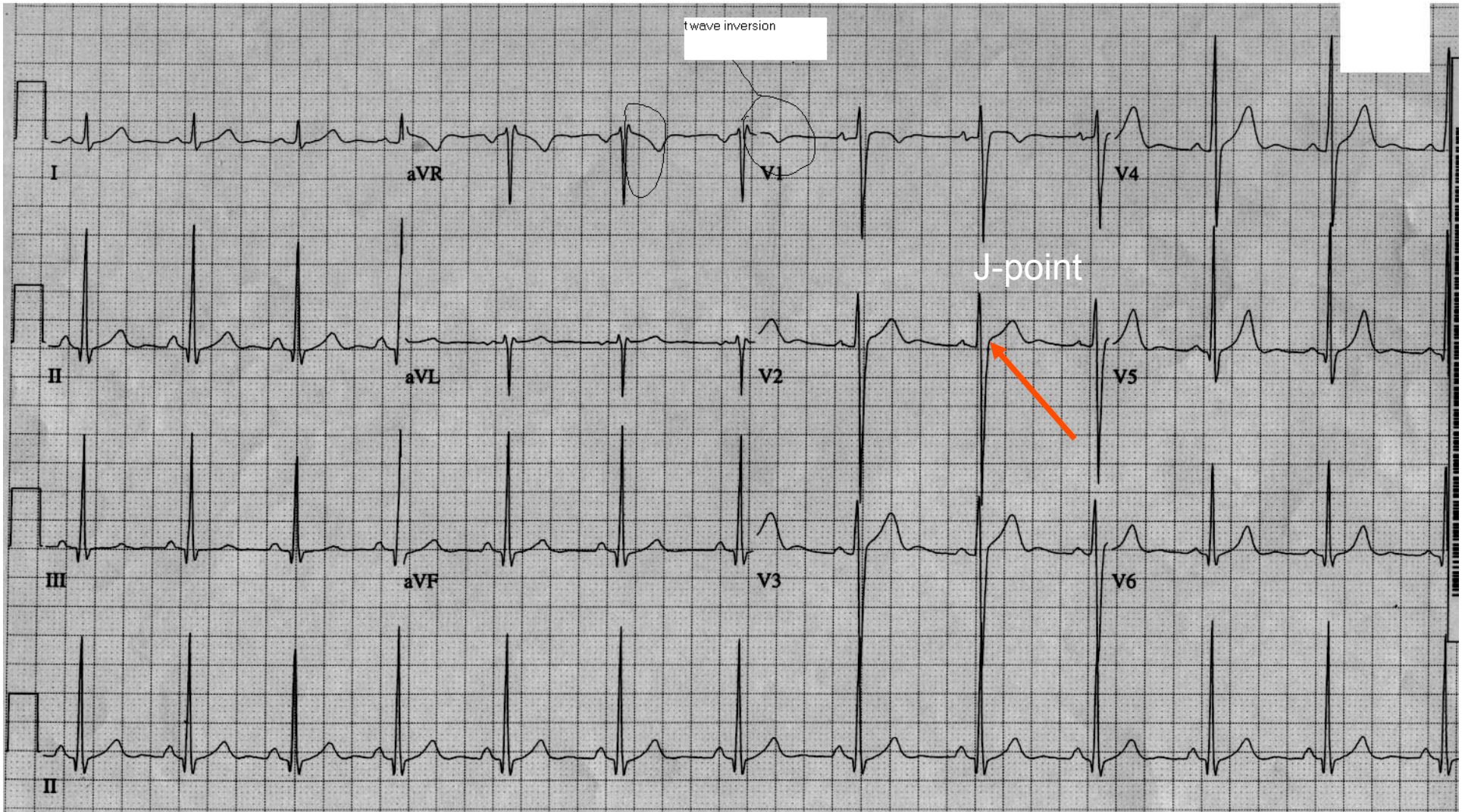
VENTRICULAR ANEURYSM



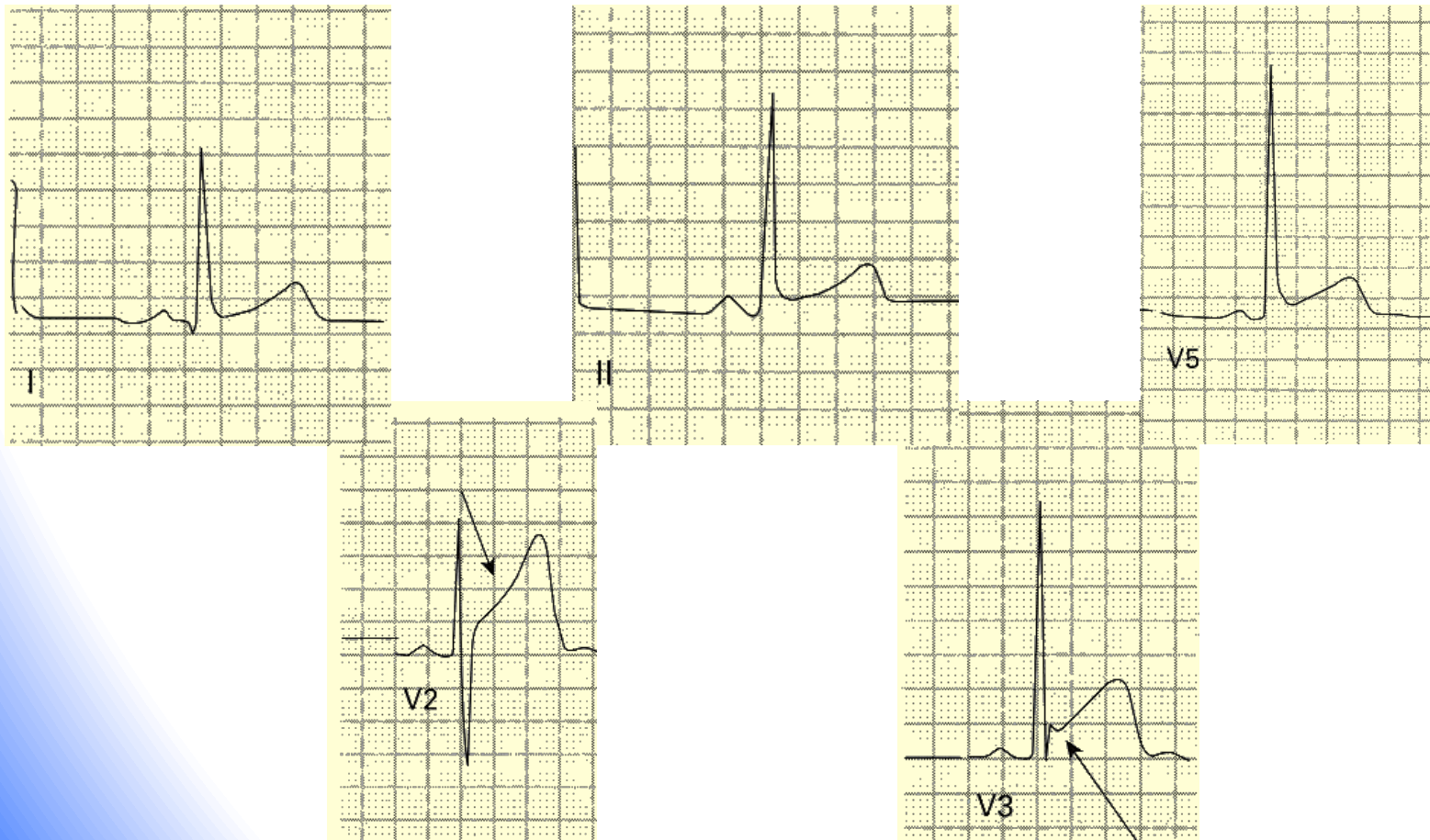
Causes of ST Segment Elevation: “Early Repolarization”

- Concave upwards morphology
- In many leads, maximal in mid-precordial leads with “fishhook” J-point
- Doesn't change over time
- No reciprocal depression
- Men > women, young > old

EARLY REPOLARIZATION



EARLY REPOLARIZATION



From *ECGs for the Emergency Physician Vol 1* (Mattu, Brady; Blackwell 2003)

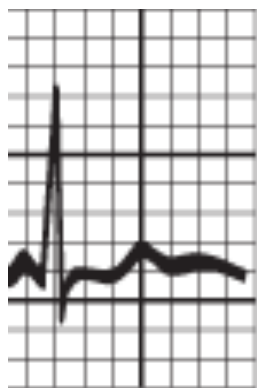
EKG - Hyperkalemia

- Hyperkalemia
 - Flat P waves
 - Peaked T waves
 - Wide QRS
 - Prolonged QT (due to hypoCa) and PR
 - Bradycardias and AV blocks
 - Eventual sine wave and Vfib

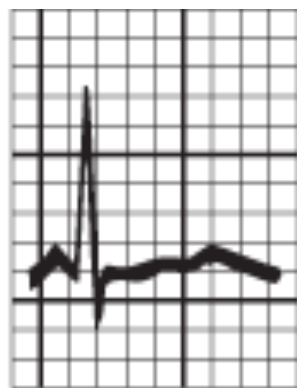
EKG – Hypokalemia

- Hypokalemia
 - PVCs
 - U waves
 - Prolonged QT
 - ST segment depression

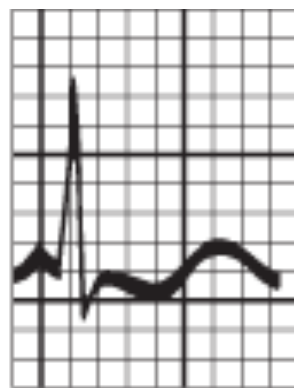
Hypokalemia / Hyperkalemia



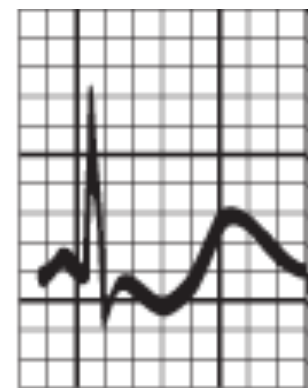
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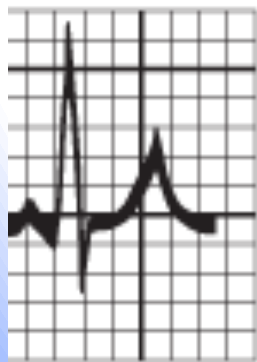


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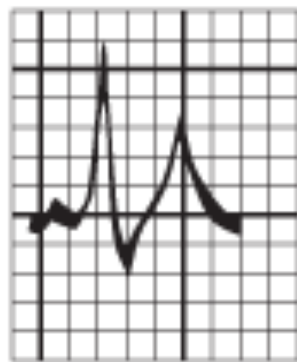


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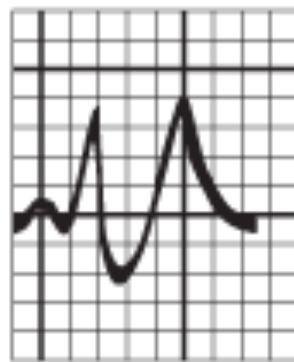
Hypokalemia



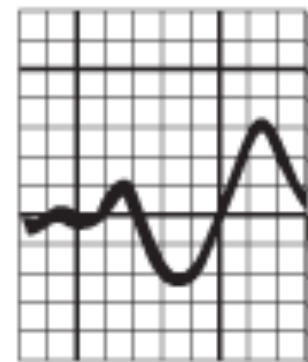
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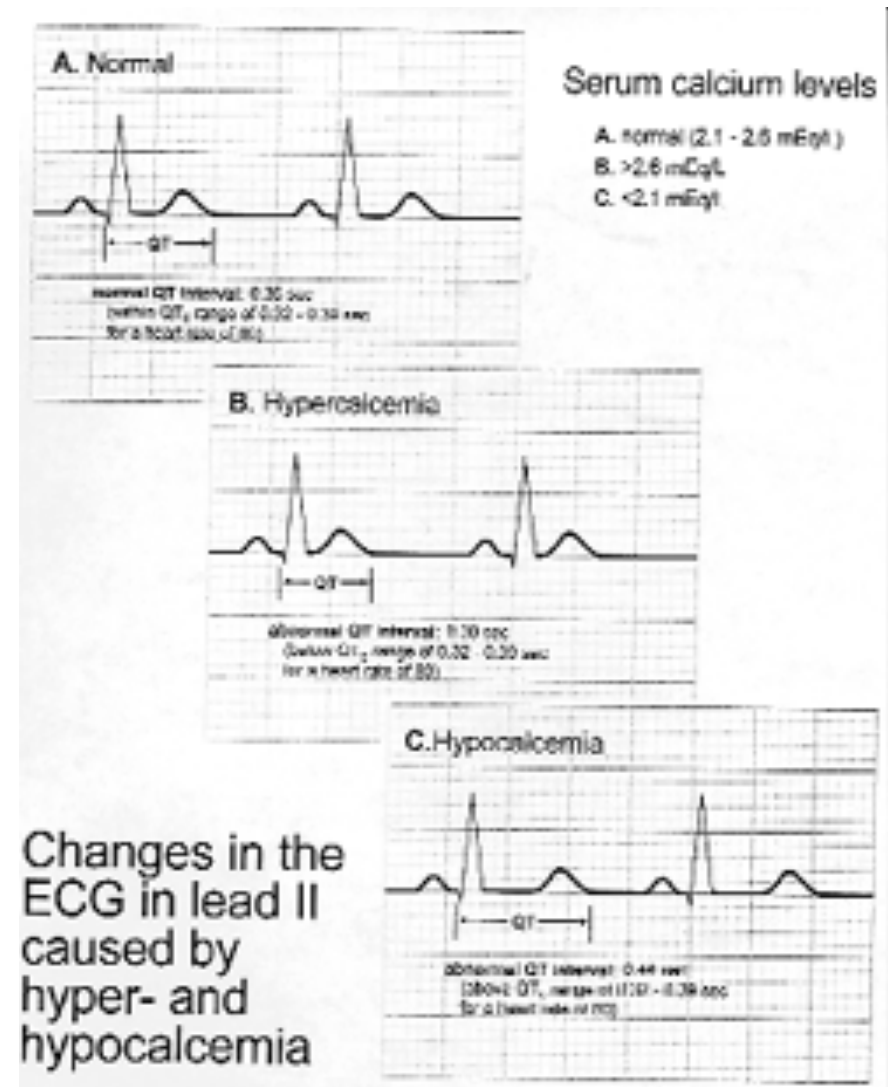


9.0

Hyperkalemia

EKG - Hypercalcemia / Hypocalcemia

- Hypercalcemia
 - Shortened QT interval
 - ST segment depression
- Hypocalcemia
 - Prolonged QT interval



EKG - Electrolyte-Induced QT Prolongation

- Electrolyte abnormalities that **prolong QT** interval
 - Hypokalemia
 - Hypocalcemia
 - Hypomagnesemia
 - Hyperphosphatemia
- Prolonged QT leads to ventricular tachydysrhythmias (torsade de pointes, Vfib)

EKG - Torsade De Pointes

- Torsade de Pointes
 - Polymorphic Vtach (“twisting of the points”)
 - Associated with prolonged QT interval
- Causes
 - Quinidine, procainamide
 - TCAs
 - Phenothiazines
 - Electrolyte abnormalities
 - Low Mg^{+} , low K^{+} , low Ca^{++}

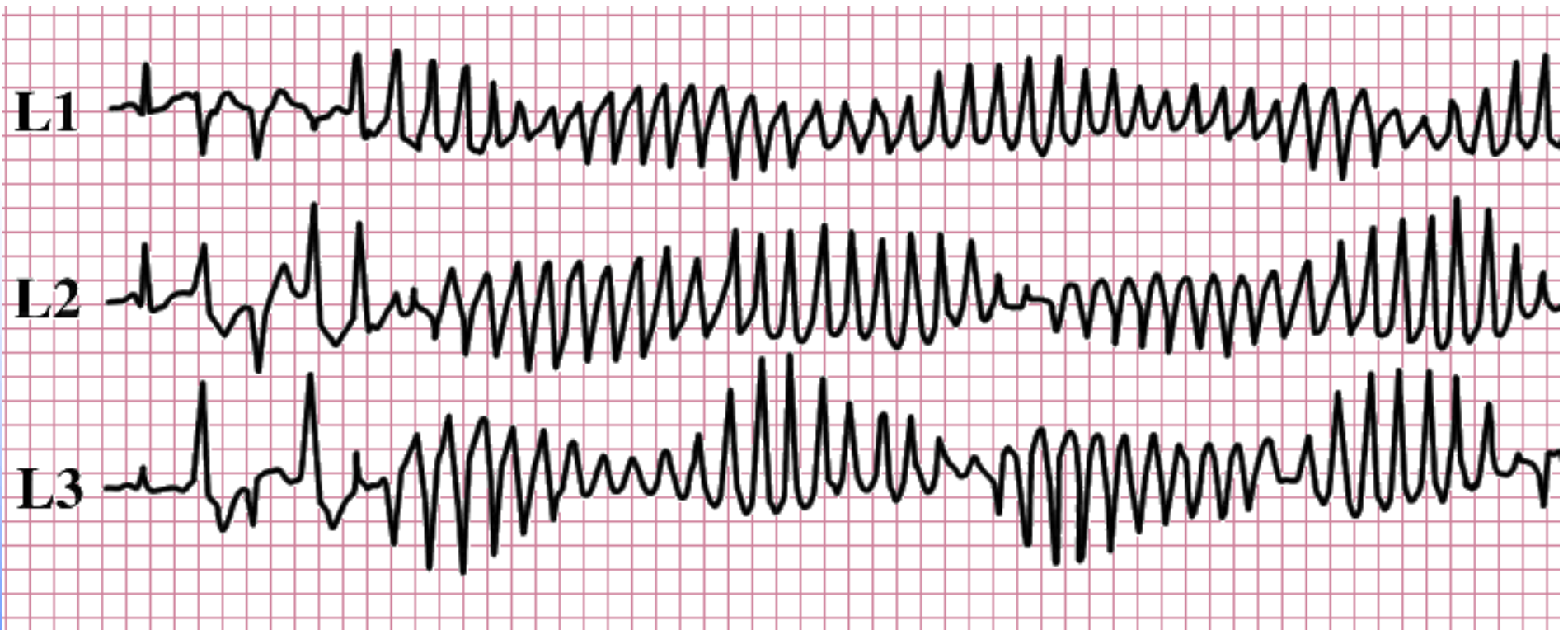


**No Procainamide
or amiodarone**

EKG - Torsade De Pointes

- Treatment
 - Magnesium (will shorten QT interval)
 - Overdrive pacing
 - Isoproterenol
 - Cardioversion/defibrillation
 - Magnesium infusion for prophylaxis after conversion

Torsade de Pointes



Classic findings:

- Polymorphic V. Tach - "a twisting of the points"
- Associated with increased QT interval

EKG - Hypothermia

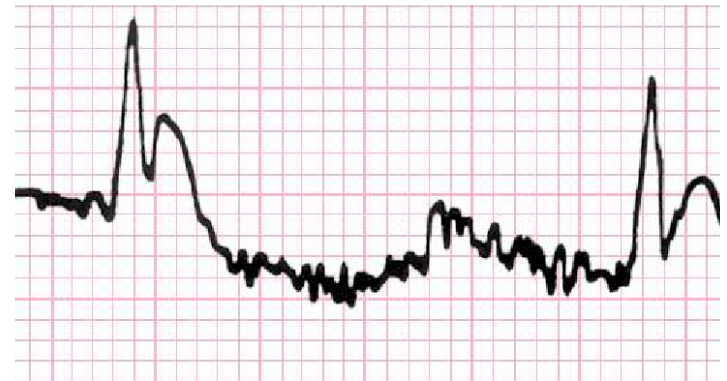
- Hypothermia ($< 30^{\circ}\text{C}$)
 - Prolonged intervals
 - Sinus bradycardia
 - Muscle tremor artifact
 - Slow A-fib
 - Osborne J waves
 - Vfib, asystole

Hypothermia



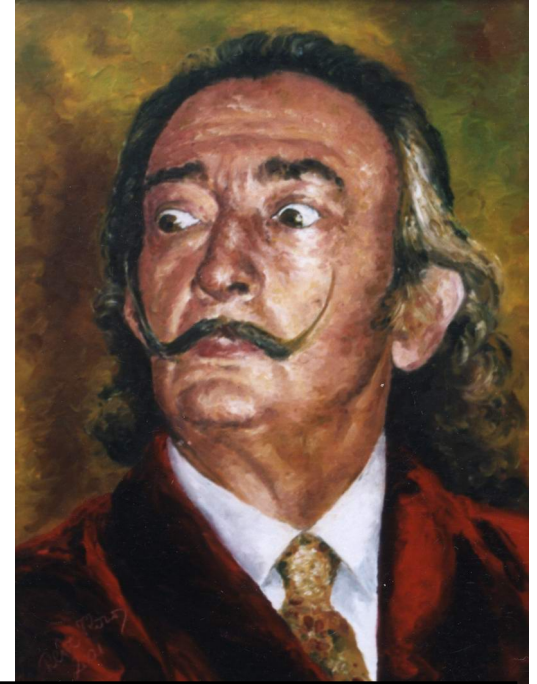
Classic findings:

- Prolonged intervals
- Sinus bradycardia
- +/- muscle tremor artifact
- Slow a. fib
- Osborne (J) wave
- V. fib / asystole



EKG - Digoxin

- Causes increased automaticity with conduction block (PAT with block)
- Therapeutic levels may cause
 - T wave depression
 - ST downsloping (Salvador Dali moustache)
 - QT shortened
- Toxic levels
 - PVCs (most common dysrhythmia)
 - Sinus and AV node blocks
 - AV dissociation
 - SVT (especially with blocks)
 - Sinus bradycardia



Digitalis Effects



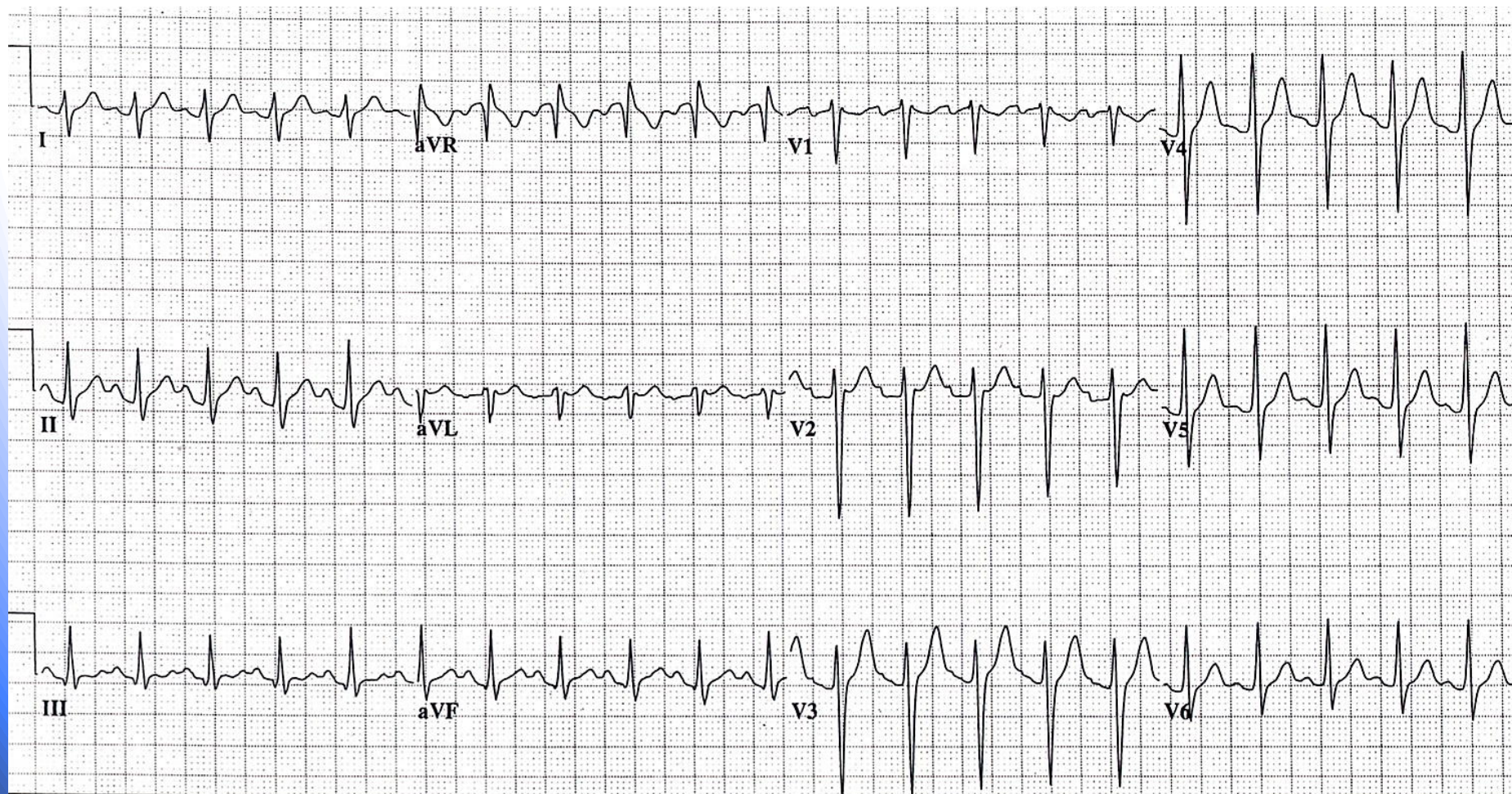
Classic findings:

- T wave depression
- ST down sloping (Salvador Dali moustache)
- QT shortened

EKG – Cyclic Antidepressants

- Sinus tachycardia (most common)
- Prolonged QT
- Right axis deviation
- QRS width > 100 ms
- Conduction blocks

EKG – Cyclic Antidepressants

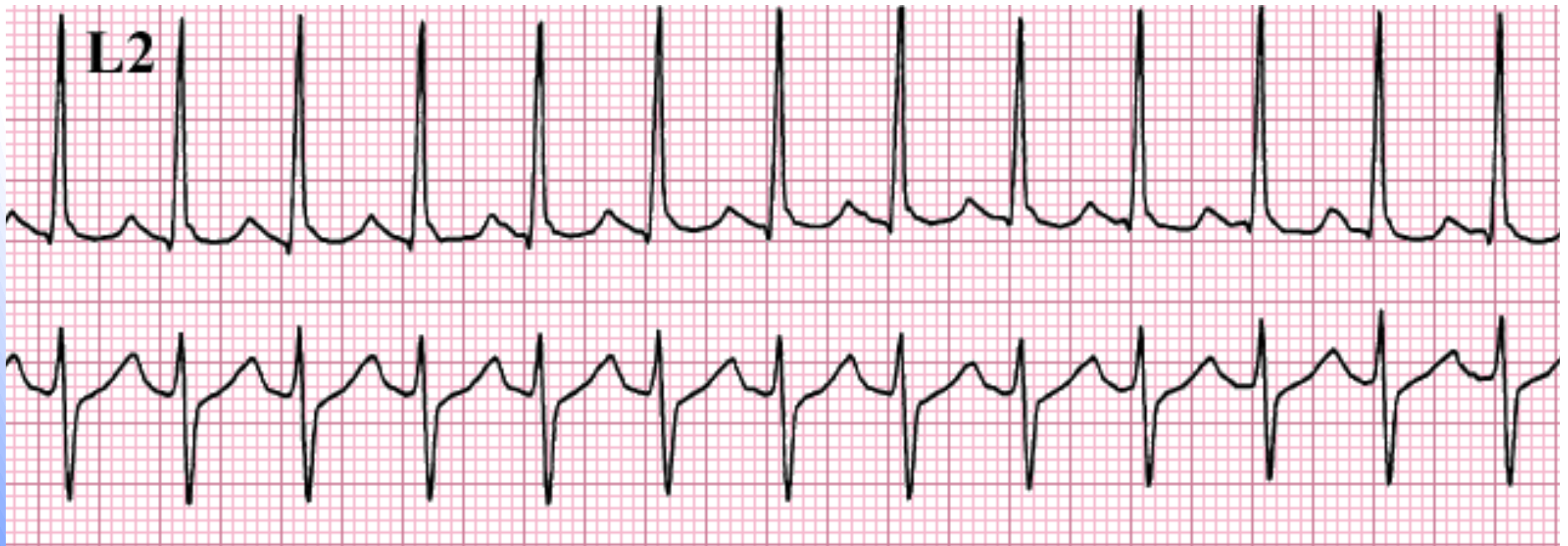


From *ECGs for the Emergency Physician Vol 1* (Mattu, Brady; Blackwell 2003)

EKG - Supraventricular Tachycardia

- Supraventricular tachycardia
 - Usually re-entrant mechanism
 - Regular, narrow complex tachycardia
 - Rate 150-200
 - Absent or retrograde P waves
 - Treatment: adenosine, calcium channel blockers (diltiazem, verapamil)
 - Don't use calcium channel blockers if a long acting beta blocker was recently administered

Supraventricular Tachycardia



Classic findings:

- Regular, narrow complex tachycardia
- Rate 150-200
- Absent P waves or retrograde

EKG - Atrial Fib / Flutter (1)

- Etiologies
 - AMI, HTN, RHD
 - Thyrotoxicosis
 - Digoxin toxicity
 - Chronic obstructive pulmonary disease
 - Pericarditis
 - PE, hypoxia
 - WPW
 - Electrolyte abnormalities

EKG - Atrial Fib / Flutter (2)

- Atrial flutter
 - Regular, narrow complex with atrial rate 250-350
 - Ventricular rate usually blocked (2:1, 3:1, 4:1)
 - Sawtooth baseline (flutter waves)
- Atrial fibrillation
 - Irregularly irregular rhythm with undulating baseline (associated with thyrotoxicosis, CAD, CHF, PE, LAE, alcohol)

EKG - Atrial Fib / Flutter (3)

- Treatment: rate control (chemical, electrical)
 - Stable: diltiazem, beta blockers, amiodarone, digoxin
 - Unstable
 - Atrial flutter: synchronized cardioversion (start at 50 J)
 - A-fib: synchronized cardioversion (start at 200 J)
 - Anticoagulation for long term A-fib

Atrial Flutter



Classic findings:

- Regular, narrow complex with atrial rate = 250-350
- Ventricular rate usually blocked - 2:1, 3:1, 4:1
- Sawtooth baseline (flutter waves)

Atrial Fibrillation



Classic findings:

- Irregularly, irregular rhythm

EKG - Multifocal Atrial Tachycardia

- Multifocal atrial tachycardia
 - Irregularly irregular narrow complexes, rate >100
 - At least 3 different P wave morphologies
 - Variable PR intervals
 - Associated with
 - Hypoxia
 - COPD
 - Theophylline toxicity
 - Treat underlying condition
 - MgSO_4 may be helpful; no shocks!

Multifocal Atrial Tachycardia



Classic findings:

- Irregularly irregular with narrow complex, rate >100
- At least 3 different P wave morphologies, Variable PR

EKG - Wolff-Parkinson-White Syndrome (1)

- Bypass tract joining atria to ventricles with no conduction delay (short PR interval)
- A-fib with 1:1 conduction of atrial rate → death
- Delta wave when conduction is through bypass tract during NSR
 - Delta wave is not reliably seen in tachyarrhythmias

EKG - Wolff-Parkinson-White Syndrome (1)

- Avoid digoxin and calcium channel blockers (verapamil, diltiazem) during A-fib
 - These drugs block the AV node but not the bypass tract. All impulses then go through the bypass tract (not slowed down at AV node)
 - Procainamide or electrical cardioversion is safe
 - Adenosine and other AV nodal blockers are contraindicated during A-fib

EKG - Wolff-Parkinson-White Syndrome (2)

- Treatment is based on QRS width
 - Narrow-complex tachycardia
 - Treat like SVT
 - Wide-complex tachycardia, no history of WPW
 - Treat like Vtach
 - A-fib with known history of WPW
 - Procainamide, cardioversion
 - Do not use digoxin, verapamil, diltiazem, adenosine, ?amiodarone (AVN blocker)

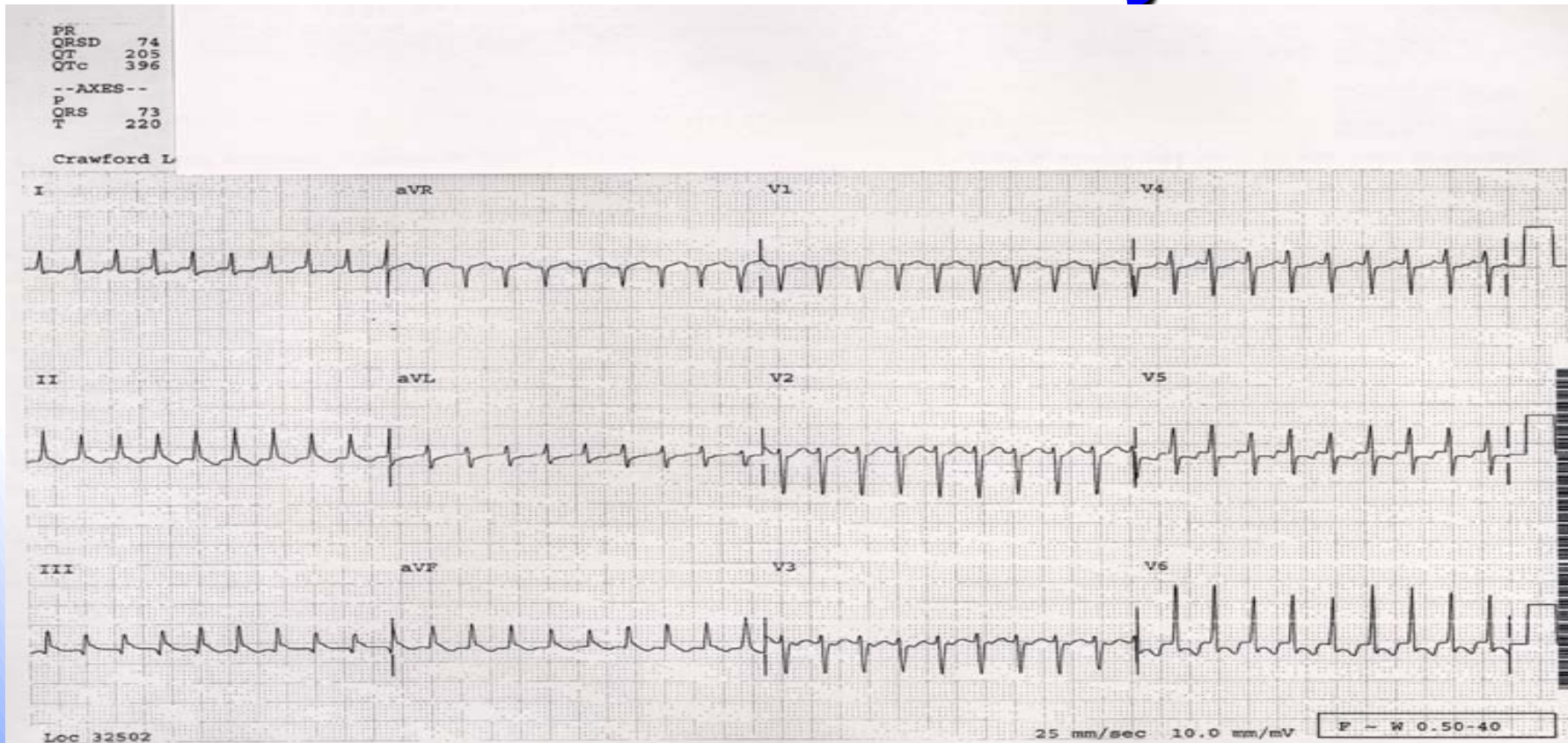
Wolff-Parkinson-White Syndrome



Classic findings:

- Wide complex seen with delta wave when conduction through bypass tract

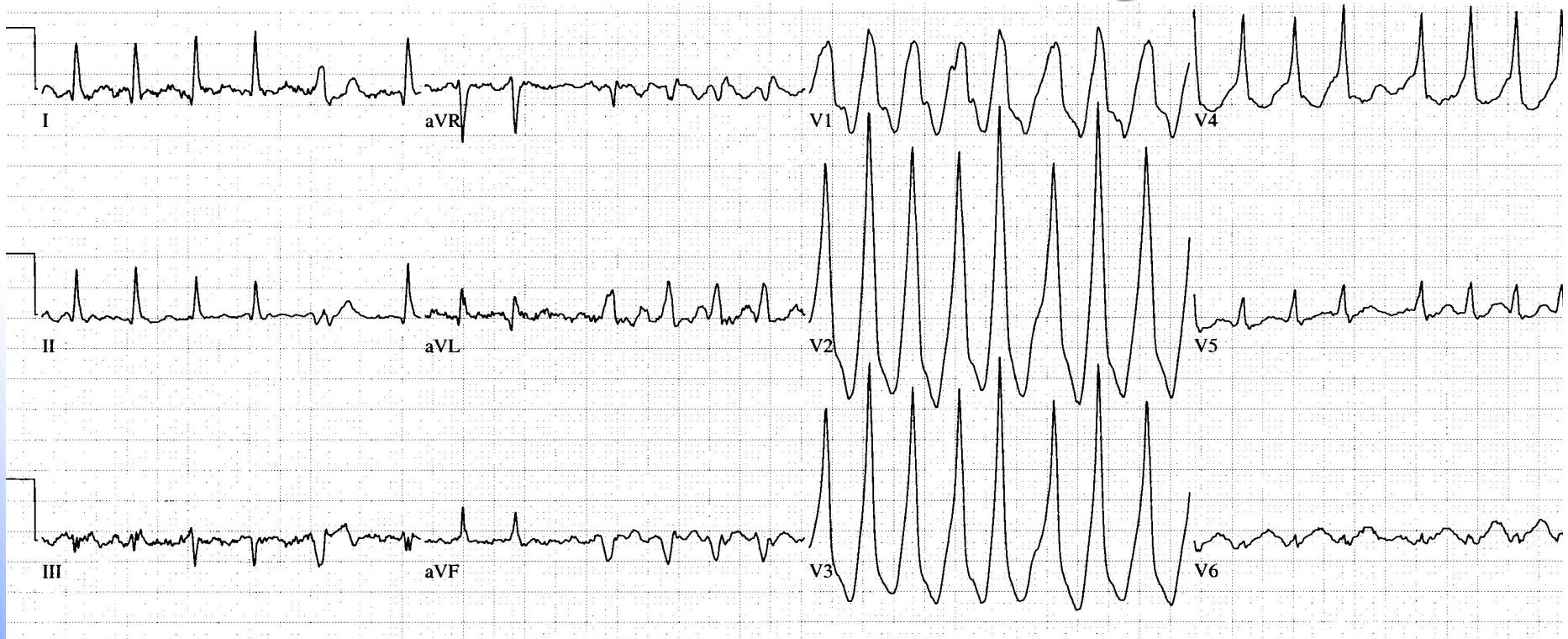
Wolff-Parkinson-White Syndrome



SVT classic findings:

- Regular tachycardia
- Treat as any other regular NCTs or WCTs

Wolff-Parkinson-White Syndrome



From *ECGs for the Emergency Physician Vol 1* (Mattu, Brady; Blackwell 2003)

A-fib classic findings:

- Irregularly irregular tachycardia
- QRS morphologies change
- Treat with procainamide or electricity

EKG - Ventricular Tachycardia (1)

- Ventricular tachycardia
 - Regular, wide-complex, rate >120 (usually >150)
 - Associated with
 - Underlying heart disease (CAD, cardiomyopathy, MVP)
 - Hypokalemia
 - Electrolyte disturbances
 - Toxic ingestions
 - If stable: procainamide > amiodarone > lidocaine,
 - If unstable: sync cardioversion

Ventricular Tachycardia



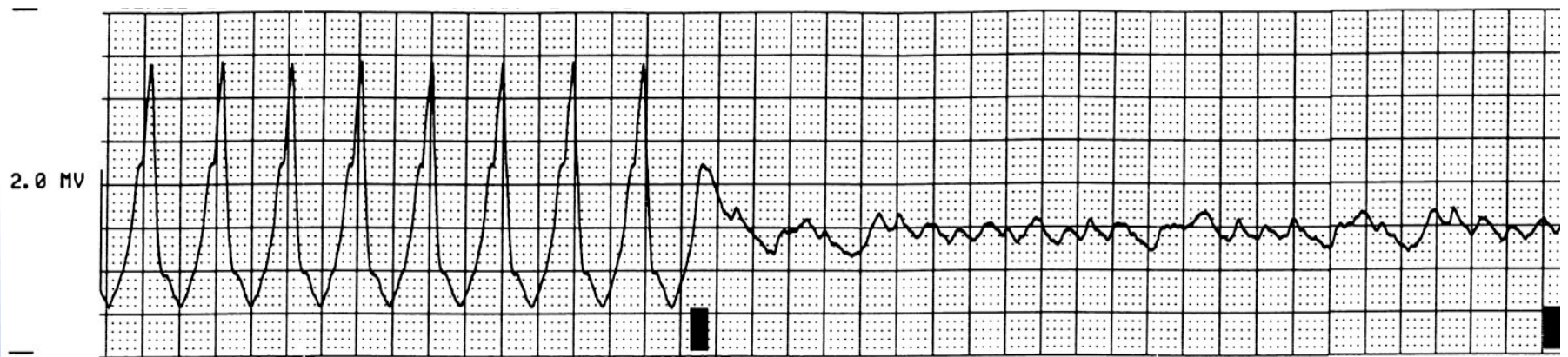
Classic findings:

- Regular wide complex with rate > 120 (usually >150)

EKG - Ventricular Fibrillation

- Ventricular fibrillation
 - Primary: no preceding hemodynamic compromise
 - Secondary: prolonged LV dysfunction, shock
 - Structural heart disease, ischemia
 - Totally disorganized, non-perfusing rhythm
 - Associated with CAD, MI, toxic ingestions, electrolyte disturbances
 - Treatment: defibrillation (ACLS protocols)

Ventricular Fibrillation



Classic findings:

- Totally disorganized, non-perfusing rhythm

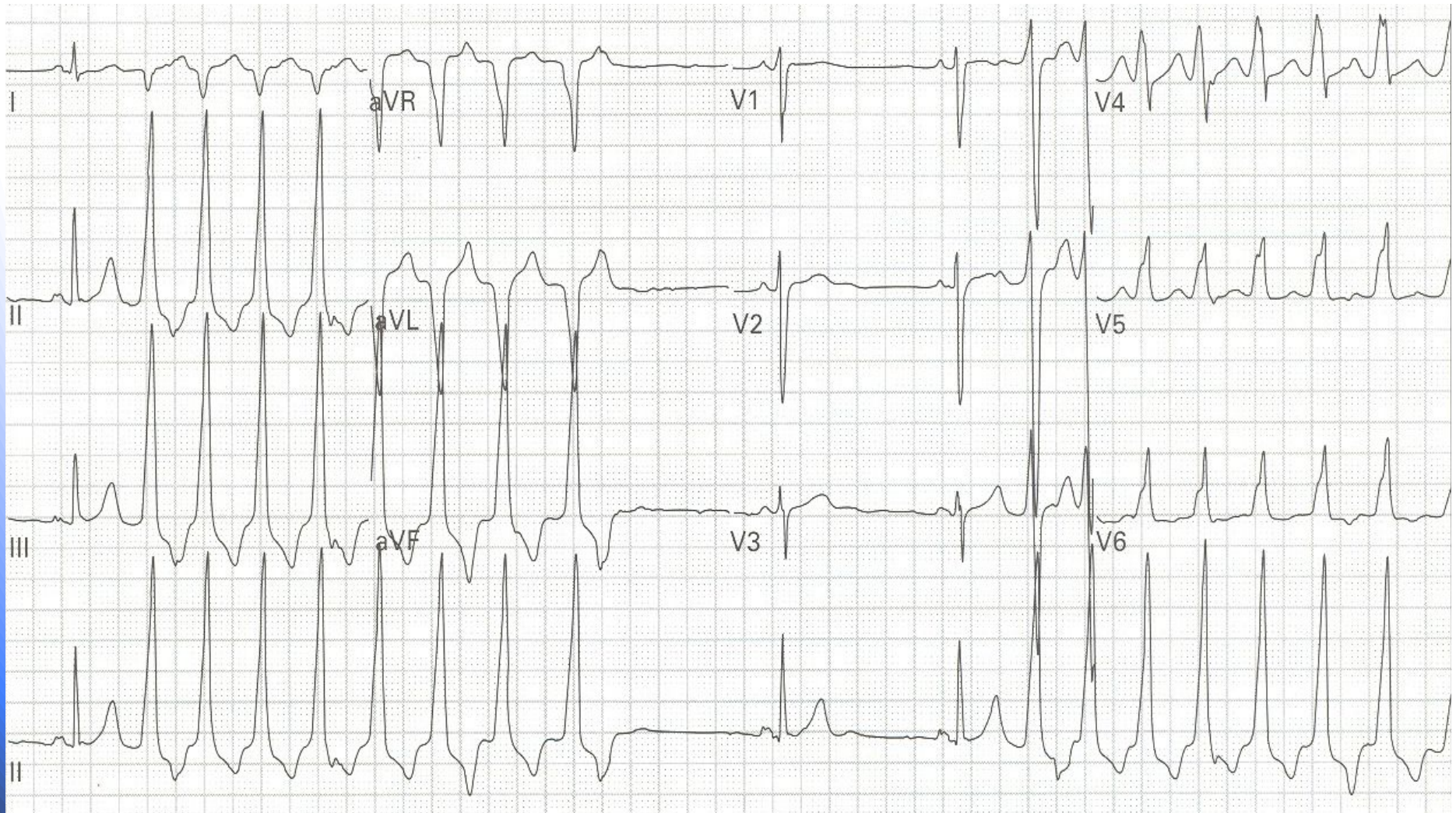
EKG - Premature Ventricular Contractions (1)

- Premature ventricular contractions (PVCs)
 - Premature, wide complex, no preceding P wave (compensatory pause)
 - Unifocal or multifocal
- Associated with
 - Normal heart, alkalosis, CHF, MI, hypokalemia, hypoxia, cardiomyopathy, drugs, digoxin toxicity
- PVCs in acute MI
 - Indicate electrical instability
 - Prophylactic treatment not proven to decrease mortality

EKG - Premature Ventricular Contractions (2)

- Treatment
 - Treat underlying cause (e.g. electrolytes, ischemia, hypoxia)
 - Consider magnesium, beta blockers
 - Treat sustained VT (≥ 30 seconds or if hemodynamic instability develops)

EKG - Premature Ventricular Contractions (2)



From *ECGs for the Emergency Physician Vol 1* (Mattu, Brady; Blackwell 2003)

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EKG - Accelerated Idioventricular Tachycardia

- Accelerated idioventricular rhythm (“slow Vtach”)
 - Wide complex regular rhythm with rate 40-120
 - Runs may last few minutes
 - Associated with acute MI and reperfusion
 - Treatment: observe!
 - Is self-terminating, NOT destabilizing
 - If hypotensive, consider atrial pacing

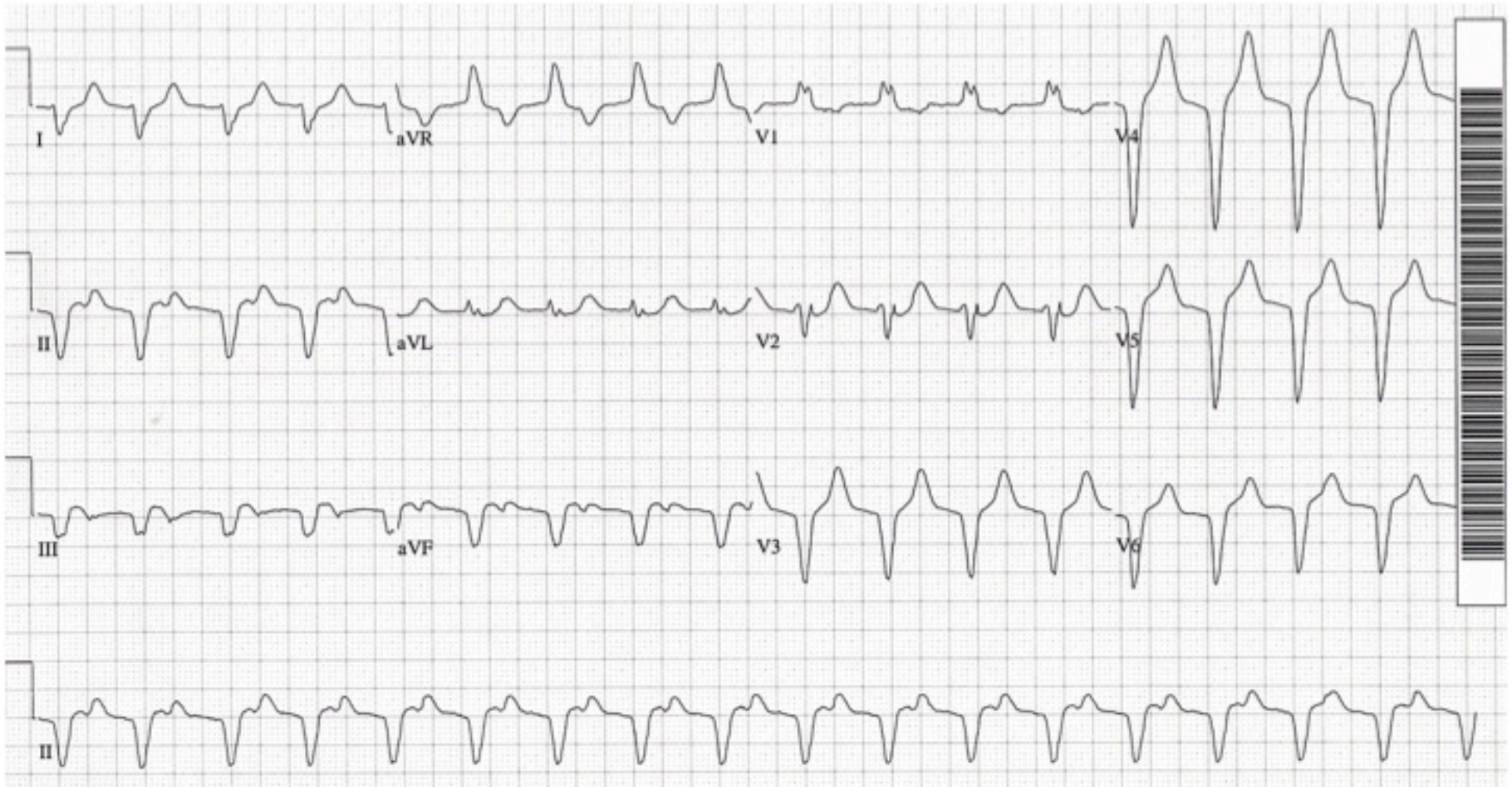
Accelerated Idioventricular Rhythm



Classic findings:

- Wide complex rhythm with rate = 40-120

Accelerated Idioventricular Rhythm (rate < 120)



From *ECGs for the Emergency Physician Vol 1* (Mattu, Brady; Blackwell 2003)

EKG - Junctional Rhythm

- Regular escape beats, rate 40-60, no P waves, usually narrow-complex
- Abnormal automaticity (accelerated junctional rhythm): rate 60-100, retrograde P waves
- Associated with
 - CHF, AMI
 - Myocarditis
 - Hypokalemia
 - Digoxin toxicity
- Treatment
 - Usually none
 - Underlying condition
 - Atropine only if needed to speed rate

Junctional Rhythm



Classic findings:

- Regular usually narrow complex with rate = 40-100
- No P wave or retrograde P wave

EKG - First Degree Heart Block

- First degree AV block
 - PR interval > 200 ms
- No treatment necessary, but look for underlying cause
- Causes: increased vagal tone, cardiac drugs, myocarditis, endocarditis, AMI (inferior), hypothermia, hyperkalemia, age-related degeneration (common in elderly)

First Degree AV Block

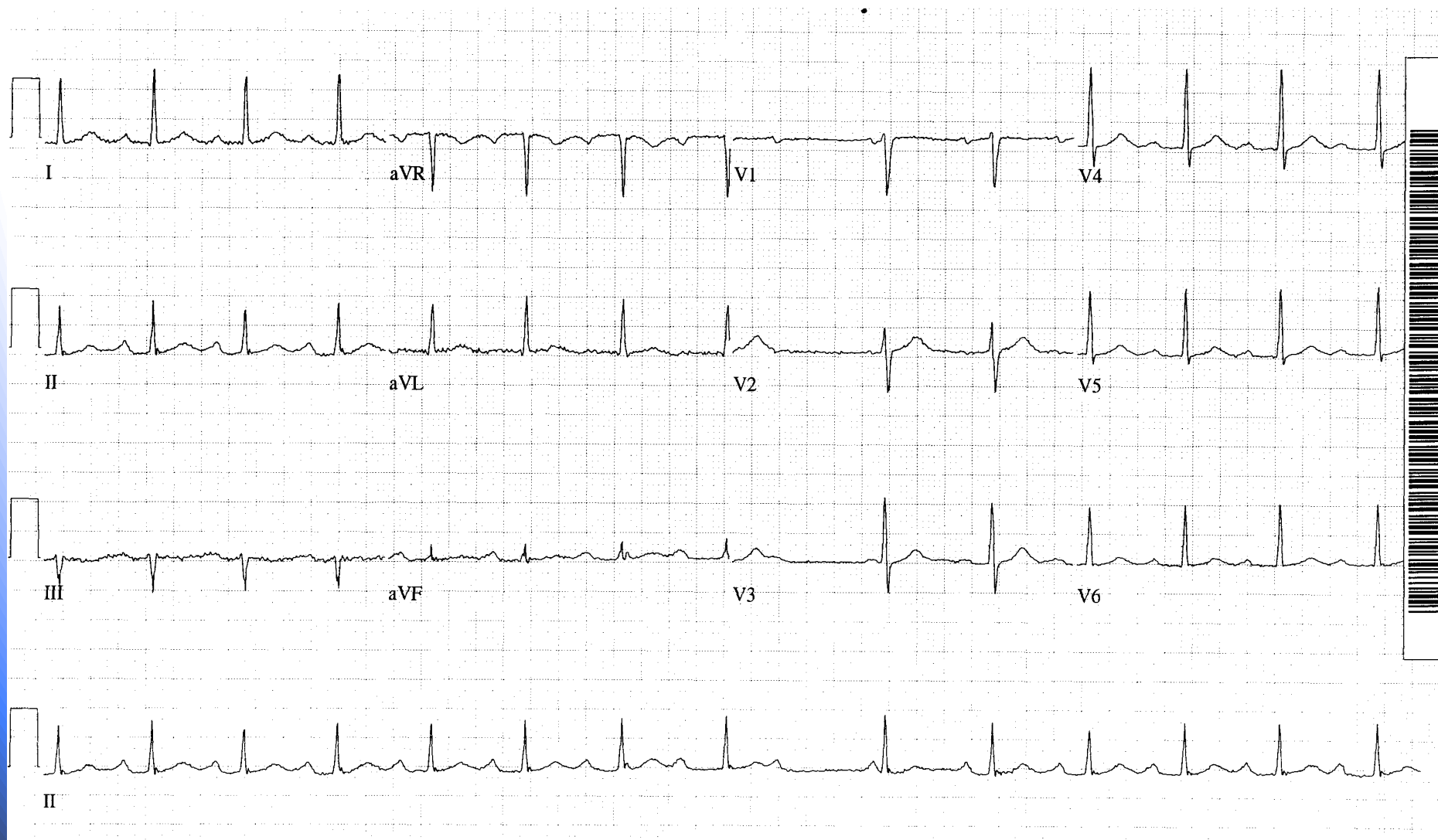


Classic findings:
- PR interval > 200 ms

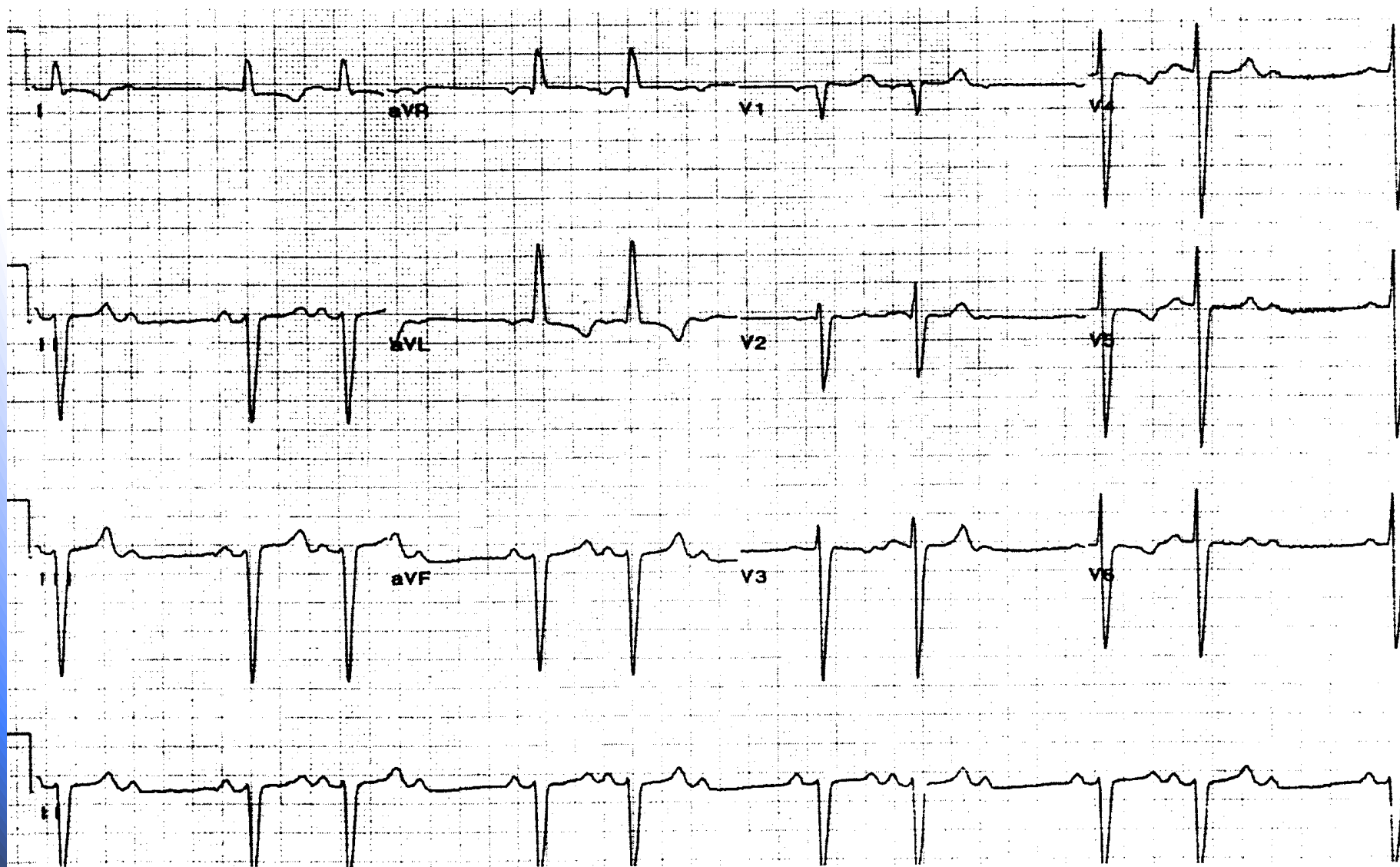
EKG - Second Degree Heart Block

- Mobitz I (Wenckebach)
 - Progressively longer PR and shorter RR until a beat is dropped, then starts over
 - Causes: same as 1st degree block, common with inferior MI, often transient; treatment if hemodynamic instability
 - Atropine or transcutaneous pacemaker usually sufficient
- Mobitz II
 - Constant PR interval and dropped beats
 - Associated with anterior MI and destruction of conduction tissue
 - Usually associated with bundle branch block
 - May progress to complete heart block
 - Temporary pacer often needed in the setting of AMI

Second Degree AV Block Type I (Mobitz I, Wenkebach)



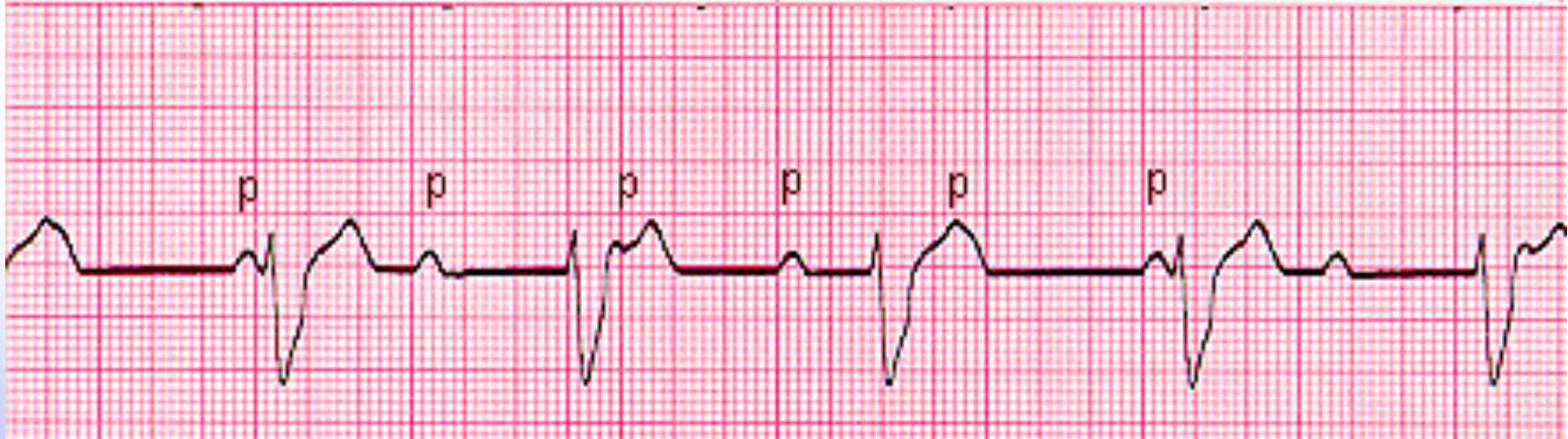
Second Degree AV Block Type II (Mobitz II)



EKG - Third Degree Heart Block

- Third degree AV block (complete heart block)
 - AV dissociation: no relation between P and QRS → PR interval changes randomly
 - Junctional (narrow QRS's) or ventricular (wide QRS's) escape beats
 - Associated with anterior MI and destruction of conduction tissue
 - Narrow complex: may be temporary due to vagal tone
 - Wide complex: usually requires transvenous pacer

Third Degree AV Block



Classic findings:

- AV dissociation → no relation between P and QRS
- PR interval changes randomly

AV Dissociation

- Atria and ventricles beat independently
- Atrial rate > ventricular rate
 - Sinus dysfunction, bradycardia and block (3^o block is a form of AV dissociation) leads to ventricular escape rhythms
- Causes
 - Ischemia (inferior MI)
 - Myocarditis (acute rheumatic fever)
 - Drug toxicity (digoxin)
 - Vagal reflex
- Fusion beats are common

Premature Ventricular Contractions (PVCs)



Classic findings:

- Premature, wide complex with no preceding P wave
- Unifocal or multifocal

Brugada Syndrome

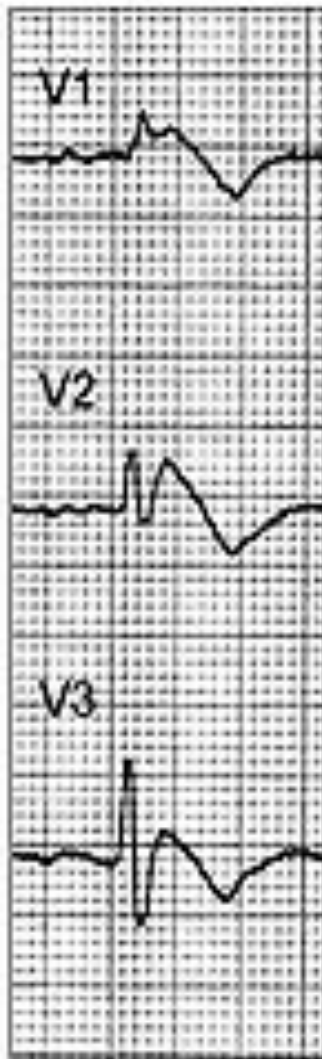
- Syncope or sudden death in young patients with a structurally normal heart resulting from ventricular fibrillation
- Most common cause of sudden death in young males without heart disease
- Familial autosomal dominant
- Particularly common in SE Asian males
- Pseudo-RBBB pattern
- ST elevations in V1 and V2
- Implantable defibrillator is treatment

Brugada Syndrome

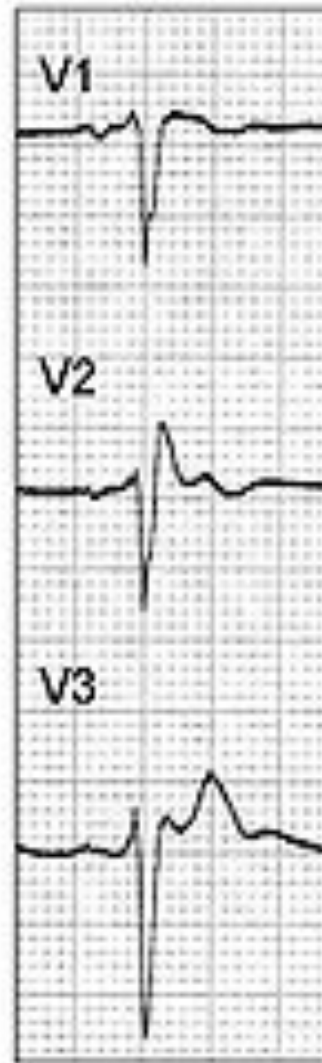
- Precipitants of Dysrhythmias
 - Beta blockers
 - Sodium channel blockers (TCAs, Type I antiarrhythmics, cocaine)
 - Ethanol
 - Fever, hot ambient temperature
 - Electrolyte abnormalities (high K and Ca, low K)
 - Increased alpha adrenergic tone
 - Pacing
 - Vagal maneuvers
 - Combinations of glucose and insulin

Brugada Syndrome

Type 1



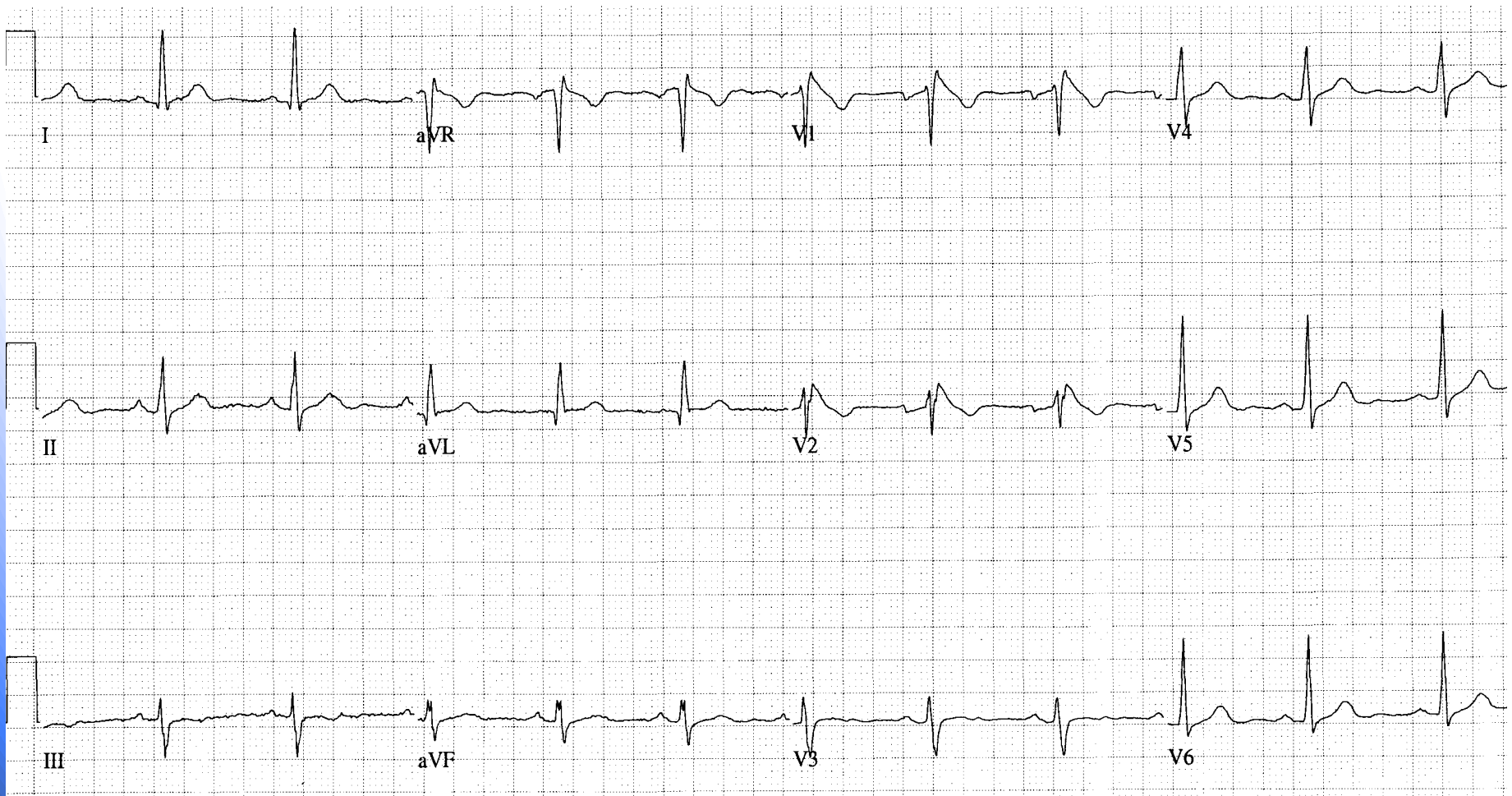
Type 2



Type 3



Brugada Syndrome



From *ECGs for the Emergency Physician Vol 1* (Mattu, Brady; Blackwell 2003)

Ventricular Tachycardia vs. SVT

- VTach findings
 - AV dissociation (“cannon” A waves in neck, variable intensity S1, variable pulse amplitude beat-to-beat)
 - Fusion beats
 - QRS >14 ms
 - Elderly patients
 - History of CAD or MI
 - Concordance
- SVT findings
 - QRS in same direction as normal QRS

Ventricular Tachycardia vs. SVT

- VTach findings
 - AV dissociation (“cannon” A waves in neck, variable intensity S1, variable pulse amplitude beat-to-beat)
 - Fusion beats
 - QRS >14 ms
 - Elderly patients
 - History of CAD or MI
 - Concordance
- SVT findings
 - QRS in same direction as normal QRS

Ventricular Tachycardia vs. SVT

- VTach findings
 - AV dissociation (“cannon” A waves in neck,

Always assume a regular WCT on the boards is Vtach

- Concordance
- SVT findings
 - QRS in same direction as normal QRS

CARDIOLOGY QUESTIONS

Which of the following factors is consistent with Prinzmetal's (variant) angina?

- A. It is associated with ST depression rather than elevation
- B. It usually comes on during exertion rather than rest
- C. Most patients have some mild evidence of coronary artery disease on angiography
- D. Never results in myocardial infarction
- E. Patients undergoing cardiac cath are rarely diagnosed with variant angina

Which of the following statements is true regarding aortic regurgitation?

- A. Results in a decrescendo systolic murmur at the left sternal border
- B. Is responsible for producing a “water hammer” pulse and Austin-Flint murmur
- C. Endocarditis prophylaxis and afterload reduction are generally not indicated
- D. Is never associated with Marfan’s syndrome
- E. ECG reveals RVH

A patient had a myocardial infarction three weeks prior and today he presents with chest discomfort (made worse on swallowing), fever, and pleural effusion. The preferred treatment for the most likely diagnosis is:

- A. Angiography
- B. NSAIDs
- C. Nitrates
- D. Antibiotics
- E. Thrombolytics

A patient presents with a regular pulse at a rate of ~ 220/min. The patient's BP is 135/80. A "delta wave" is noted on one of the patient's old ECGs. Which of the following is the most appropriate for this situation?

- A. Digoxin
- B. Verapamil
- C. Procainamide
- D. Adenosine
- E. All of the above

A patient presents with an irregularly irregular rhythm with a rapid ventricular response (120/min), which later increases to 220/min. He has a known history of WPW. The BP is 100/60. Which of the following is the most appropriate treatment?

- A. Amiodarone
- B. Diltiazem
- C. Procainamide
- D. Synchronized cardioversion (zap!)
- E. Adenosine

Classic ECG finding of hypothermia is:

- A. Delta wave
- B. Prolonged intervals
- C. J wave
- D. Osgood wave
- E. Asystole

**In non-embolic limb ischemia,
which of the following is the last
sign to develop?**

- A. Pain
- B. Pulselessness
- C. Pallor
- D. Paresthesias
- E. Coolness

A patient presents with a blood pressure of 260/150, dyspnea and chest pain. Which of the following agents is most likely to be successful for reducing this patient's blood pressure?

- A. Diltiazem
- B. Nitroprusside
- C. Clonidine
- D. Nifedipine
- E. Nitroglycerin

Which of the following statements is true regarding endocarditis?

- A. The pulmonic valve is the most commonly infected
- B. A murmur is auscultated in less than 5% of cases
- C. Optimal antibiotic therapy consists of first or second generation cephalosporins
- D. TEE is considered the “gold standard” for detecting valvular vegetations
- E. Roth spots are noted on the palms and soles

Which of the following are causes of myocardial infarction?

- A. Aortic dissection
- B. Atherosclerosis
- C. Cocaine
- D. Emboli
- E. All of the above

Which of the following is consistent with unstable angina?

- A. Positive cardiac markers
- B. Consistent chest pain relieved by rest and nitroglycerin
- C. Chest pain with acute ST-segment elevations
- D. Chest pain at rest
- E. Unchanged frequency and severity

65 y/o diabetic patient presents to the ED with new onset C.P. Troponin I and CPK MB are normal, ECG = SR without ischemic changes. Which plan is the most appropriate?

- A. Discharge the patient as his enzymes and ECG are normal
- B. Repeat a second set of enzymes and discharge if normal
- C. Admit the patient to rule out ACS
- D. Administer thrombolytics
- E. Perform a CT to rule out PE

**A 72 y/o male patient presents with an MI
ECG = 3rd degree AV block and ST
elevation in leads 2, 3 and AVF. The
patient experiences a brief non-sustained
run of V-tach. Which of the following is
true?**

- A. These are reperfusion dysrhythmias
- B. This patient needs a transvenous pacemaker placed immediately
- C. 3rd degree AV block is associated with poor prognosis in anterior wall MIs
- D. Thrombolytics will not be effective in this case
- E. The patient should be treated with intravenous lidocaine

Which is an absolute contraindication for thrombolytic administration?

- A. CVA within 6 months
- B. Squamous cell carcinoma
- C. Menstruation
- D. Spinal surgery 6 months ago
- E. Coumadin

A 45 y/o patient presents with C.P. The ECG = SR with ST elevations in leads 2, 3 and AVF. BP = 90/60. PE: Lungs clear +JVD. What is the most appropriate next step, based on the most likely diagnosis?

- A. Oxygen, IV nitroglycerin
- B. ASA PO and IV NS bolus 500cc
- C. Sublingual nitroglycerin x3 and morphine 2 mg IV
- D. Oxygen, IV nitroglycerin and IV dopamine
- E. Call for Intra-aortic balloon pump placement

Which of the following is not one of Jones major criteria?

- A. Subcutaneous nodules
- B. Sydenham's chorea
- C. Arthritis
- D. Carditis
- E. Erythema migrans

Exertional syncope, dysrhythmias, cardiac ischemia and sudden death are all associated with which of the following?

- A. Pericarditis
- B. Restrictive cardiomyopathy
- C. Hypertrophic cardiomyopathy
- D. Dilated cardiomyopathy
- E. Amyloidosis

A 56 y/o patient's BP = 190/115. The patient has no complaints, excluding mild frontal headache. PE: Normal. Which option is appropriate?

- A. Reassurance and defined follow-up
- B. Nifedipine 10 mg PO
- C. Clonidine 0.1 mg PO
- D. Labetalol 20 mg IVP
- E. Follow-up as needed (prn)

A 64 y/o patient presents with C.P. & SOB. PE: JVD; rales; Dyspnea; Grade 5/6 holosystolic murmur radiating into the axilla. ECG: Acute anterior wall MI. CXR: Pulmonary edema. What is this patient's complication?

- A. Dressler's syndrome
- B. Aortic dissection
- C. Atrial septal defect
- D. Chordae tendonae rupture
- E. Pericardial effusion

A 74 y/o male patient presents with an acute onset of mid-abdominal pain that radiates to the right flank. PMHx: CAD BP 80/40. What is the most appropriate next step for the most likely diagnosis?

- A. Order a CT of the abdomen with triple contrast
- B. Order a selective mesenteric angiogram
- C. Consult surgery for emergent exploratory laparotomy
- D. Order an abdominal ultrasound
- E. Order a renal protocol CT

Cardiology Answer Key

1. C

2. B

3. B

4. E

5. D

6. C

7. B

8. B

9. D

10. E

11. D

12. C

13. C

14. A

15. B

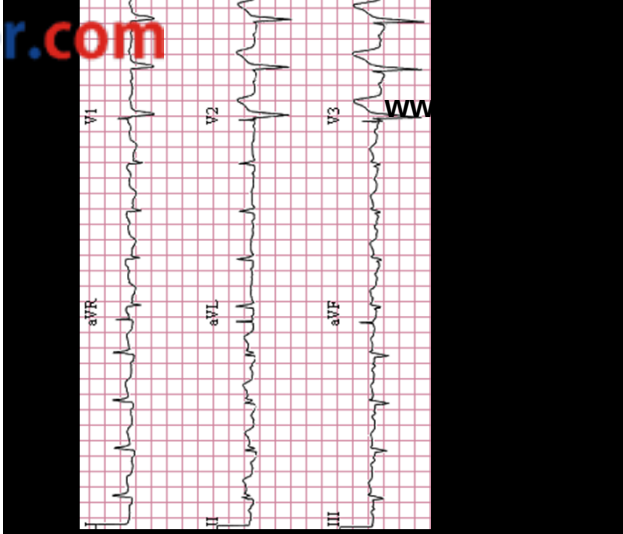
16. E

17. C

18. A

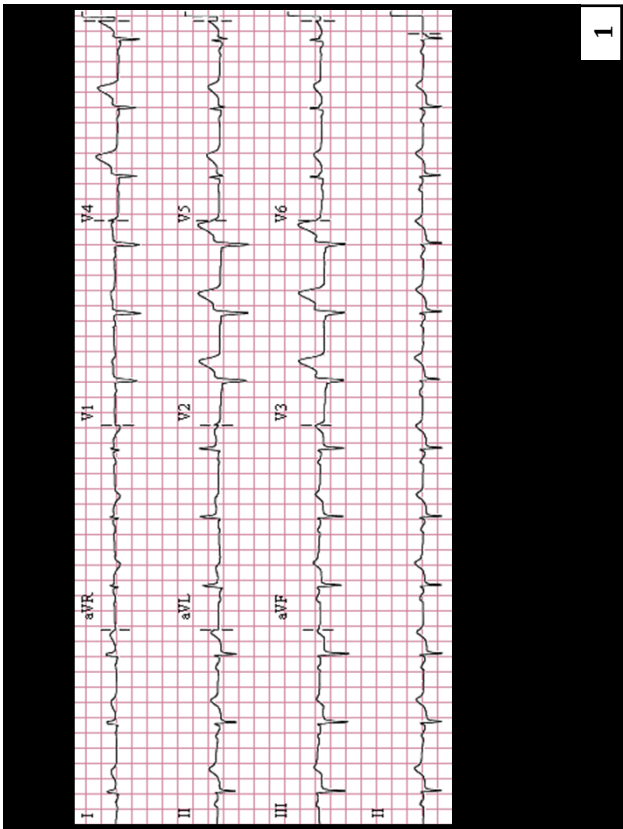
19. D

20. C

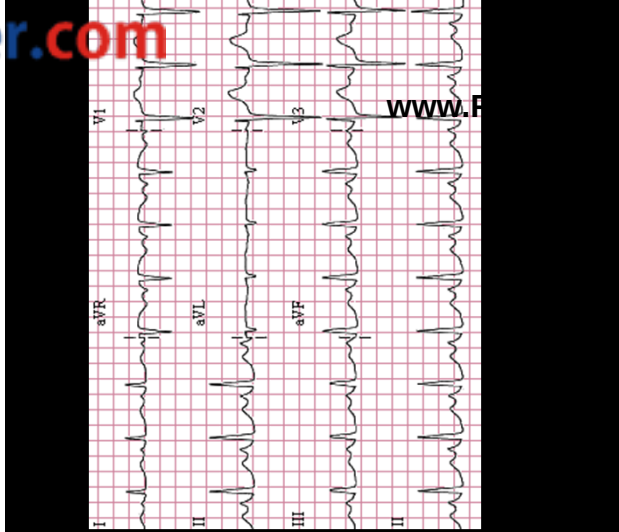


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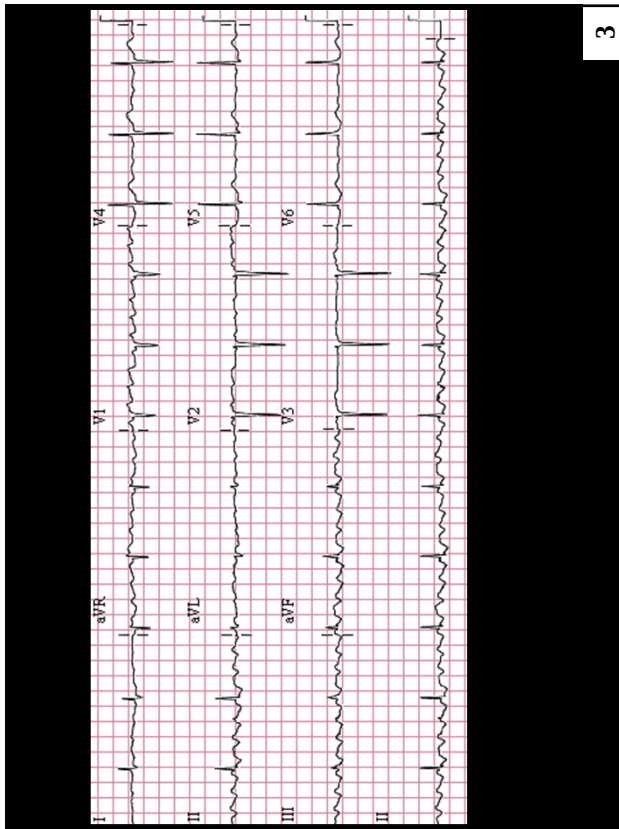


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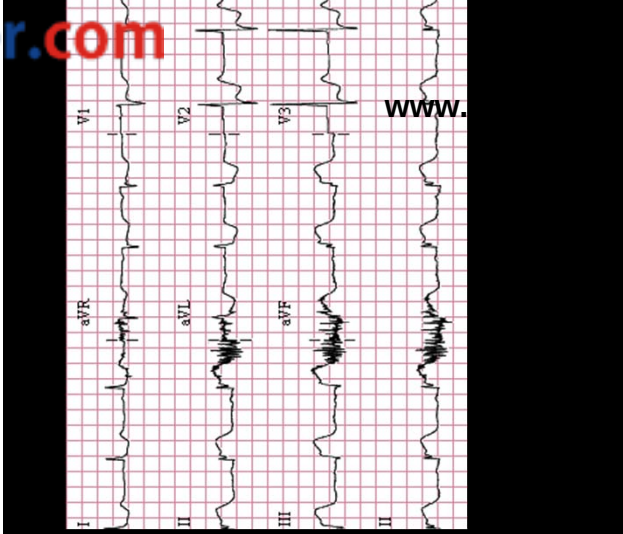


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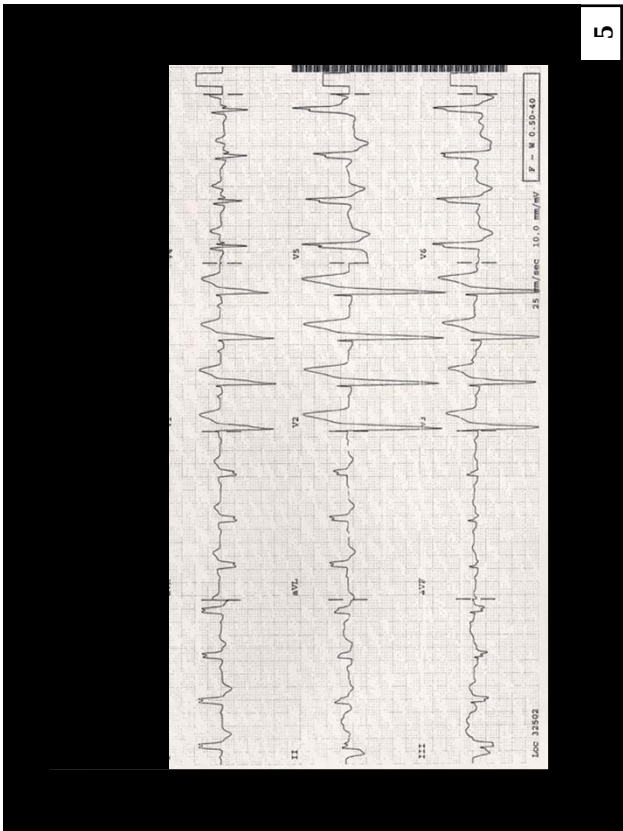


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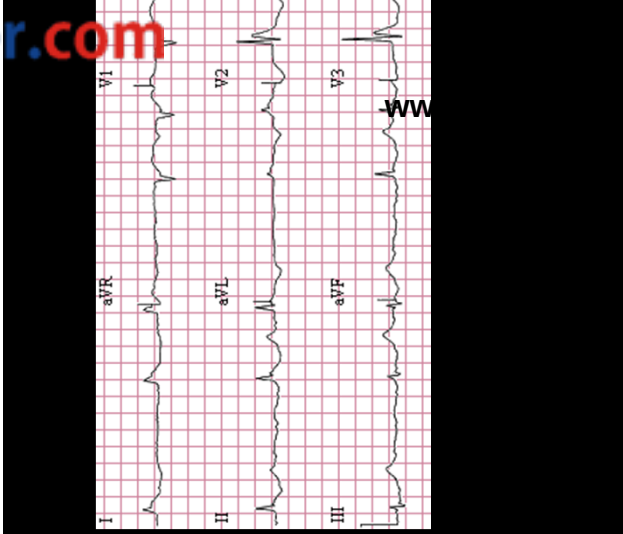


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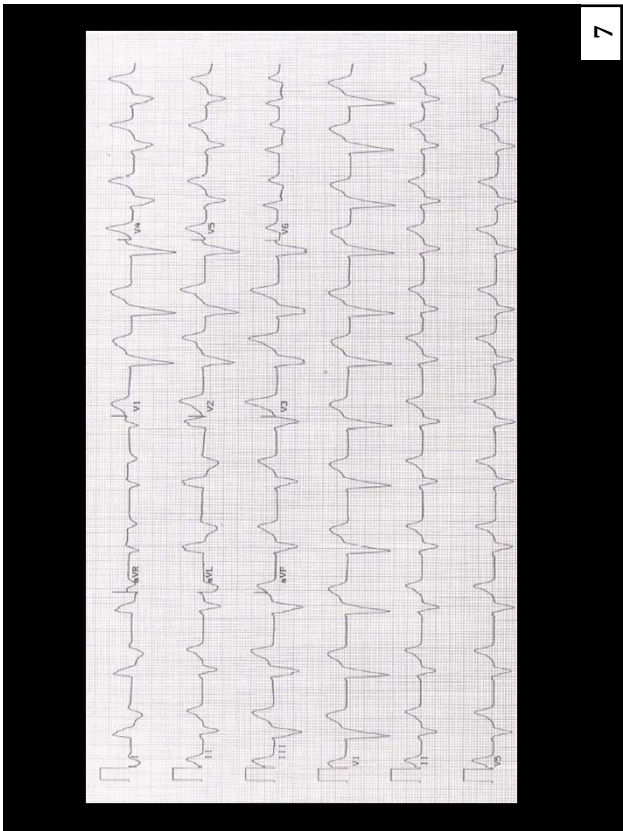
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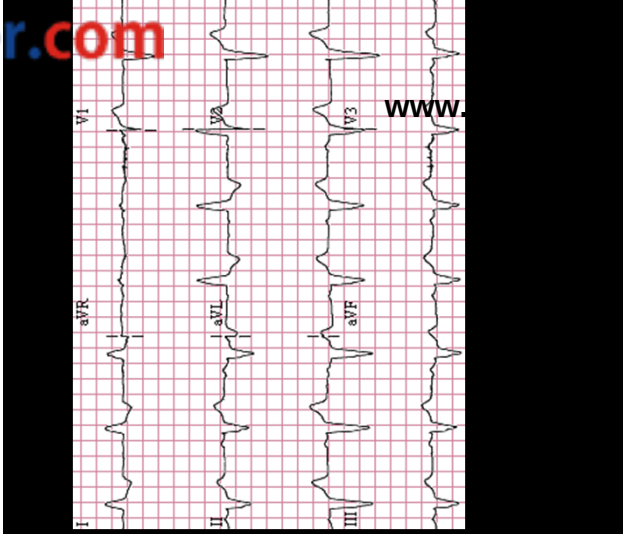
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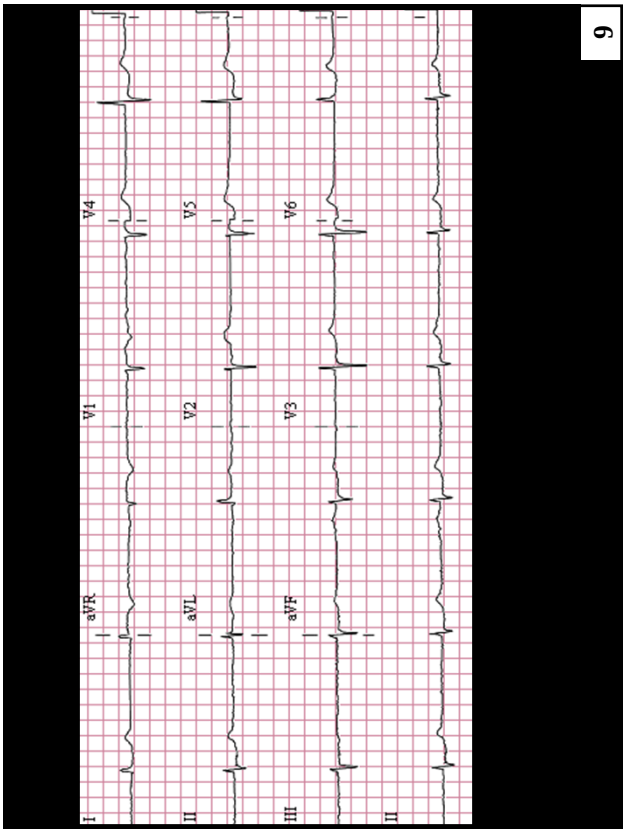


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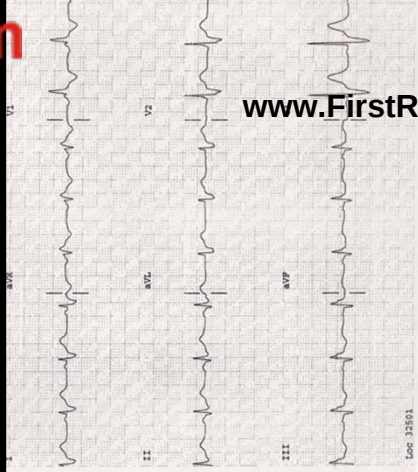


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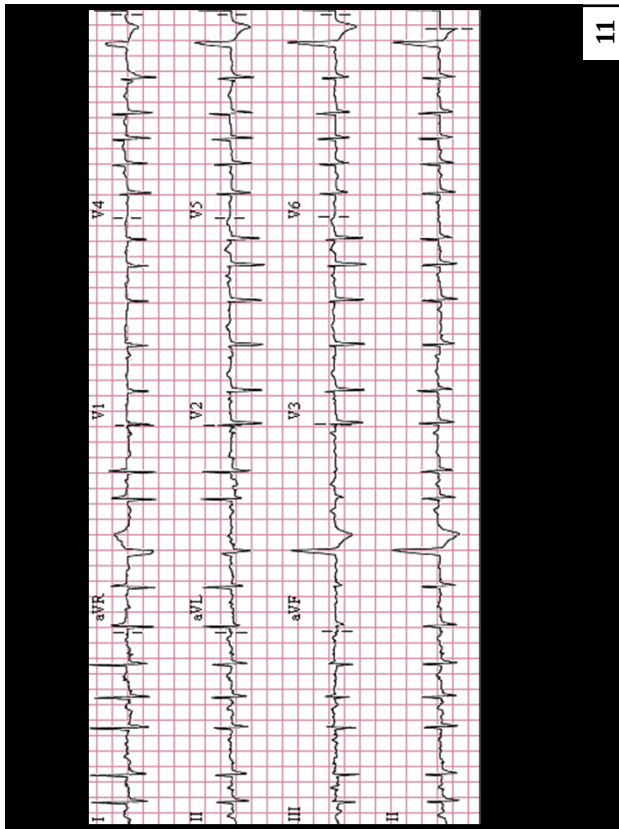


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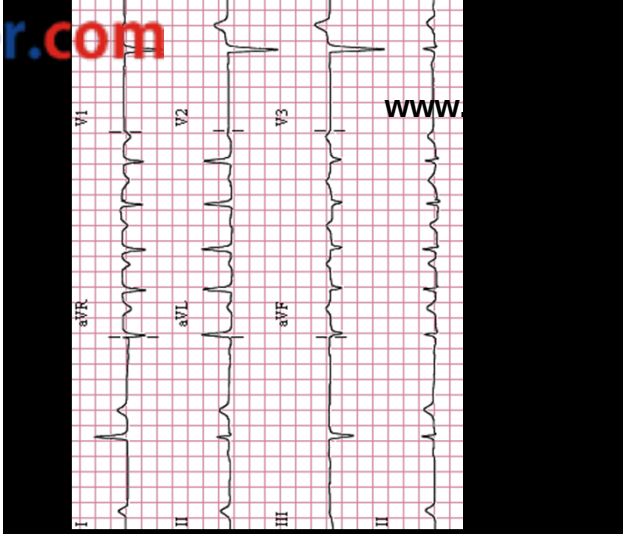


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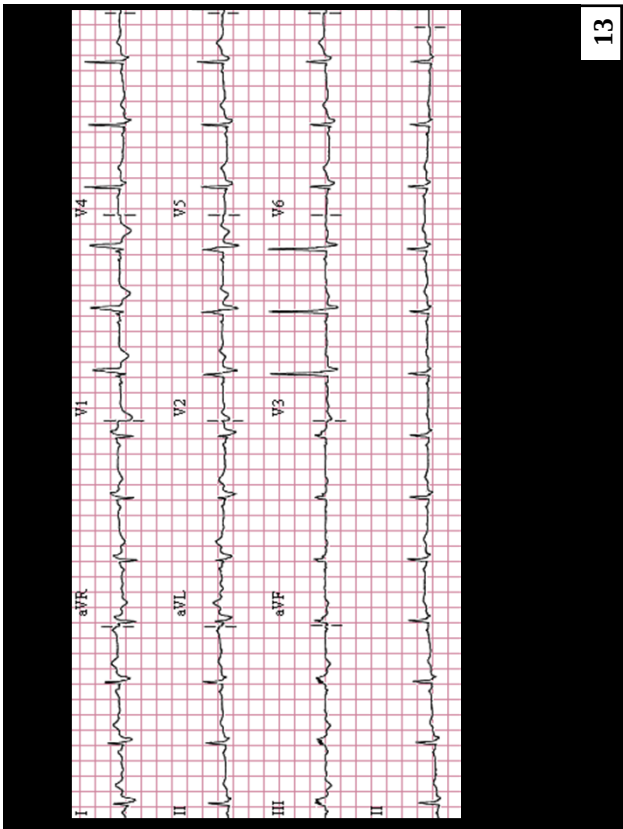


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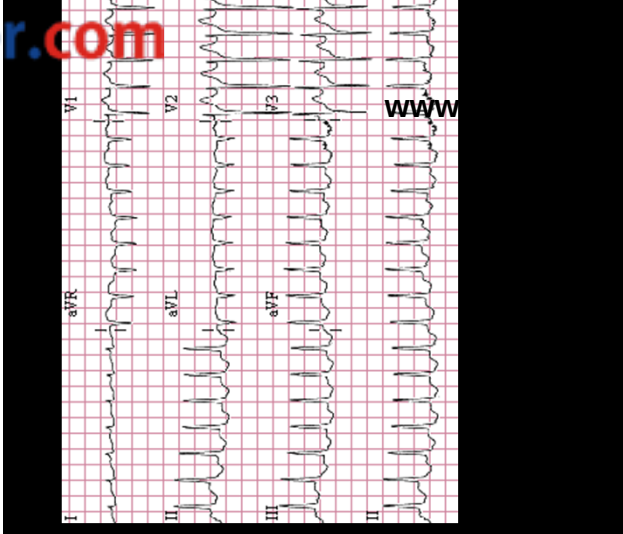


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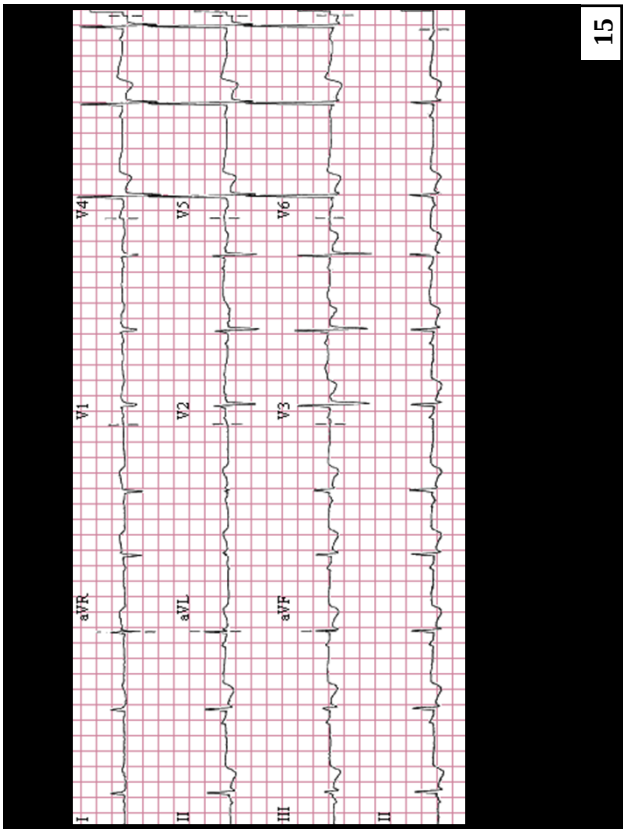
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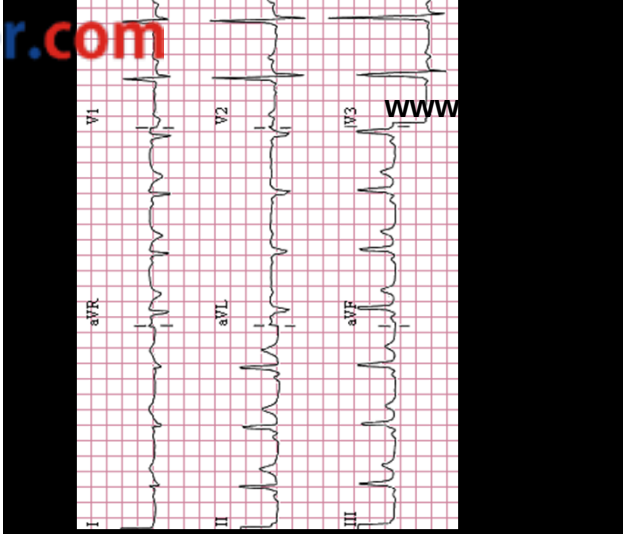
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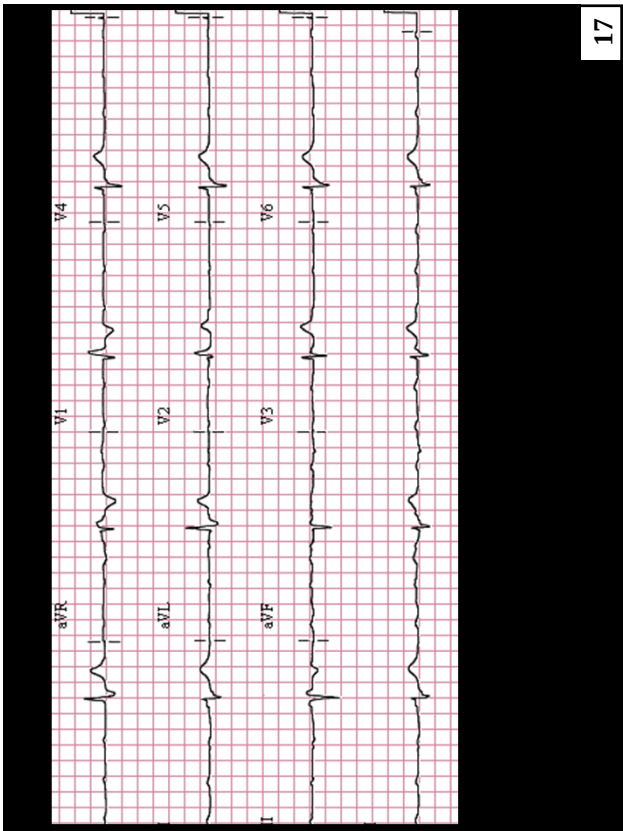
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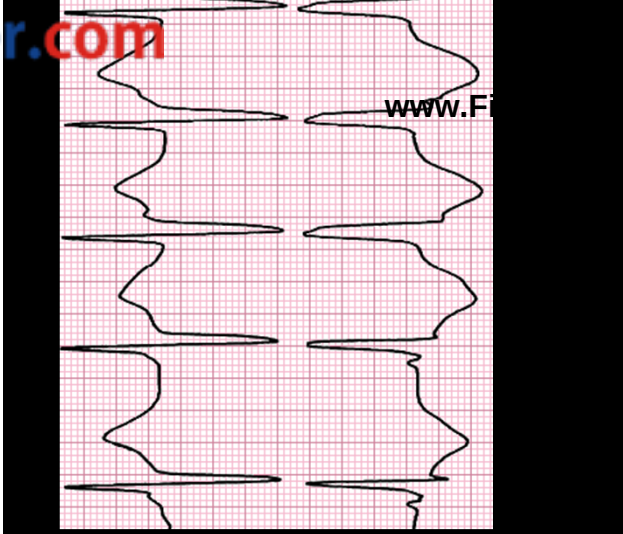
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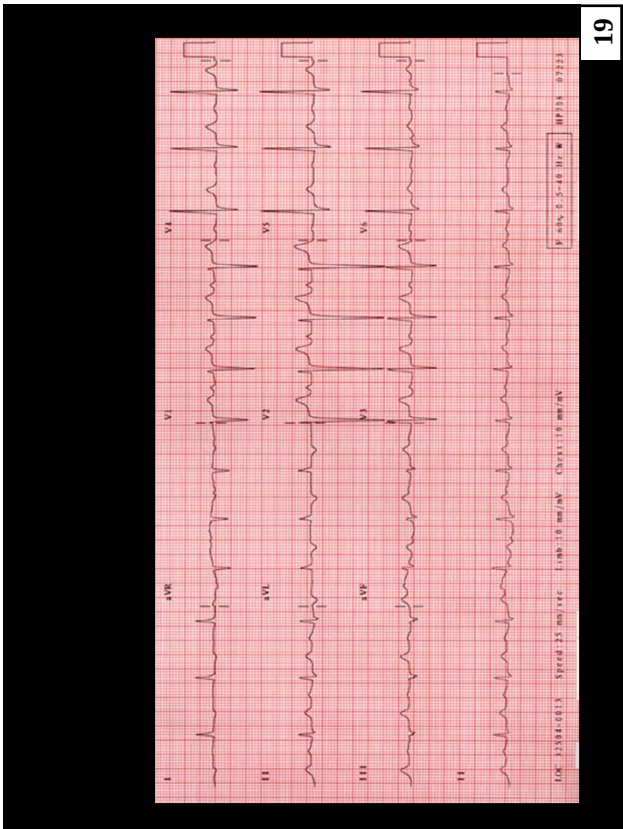


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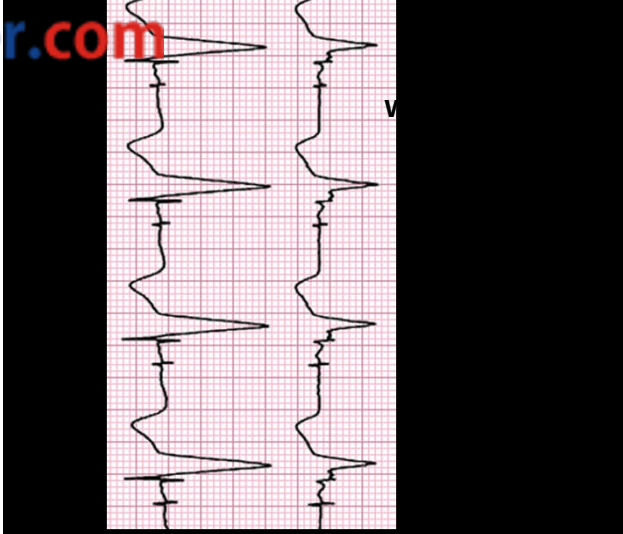


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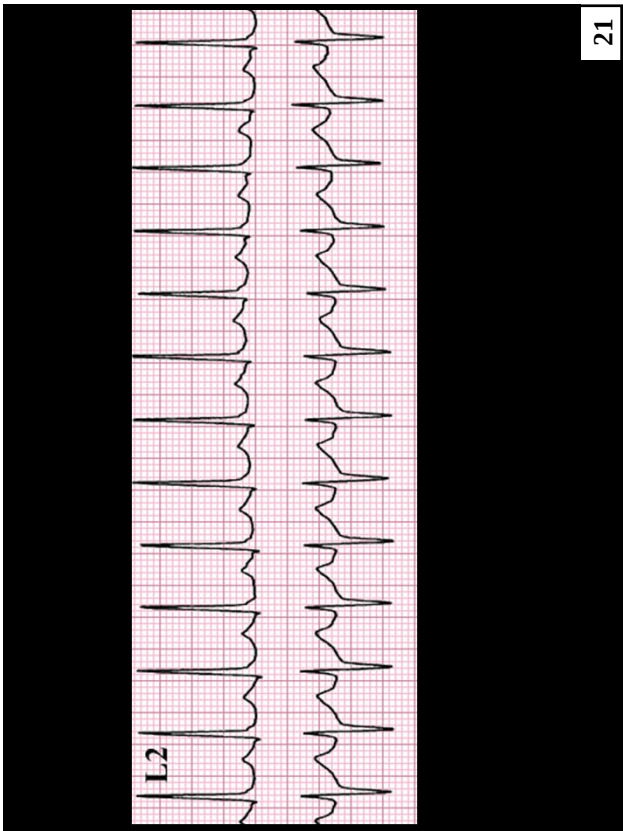


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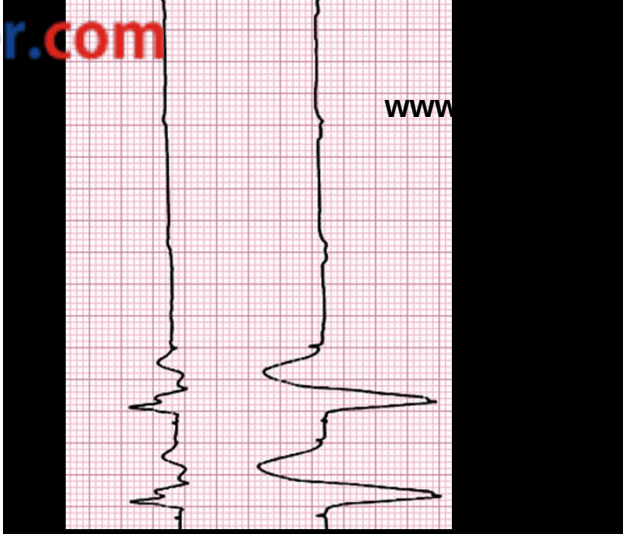


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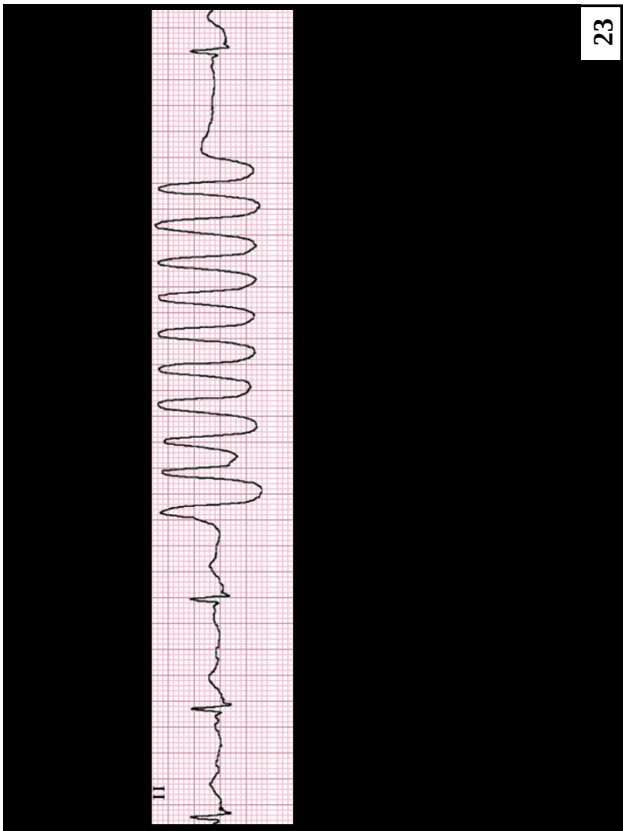


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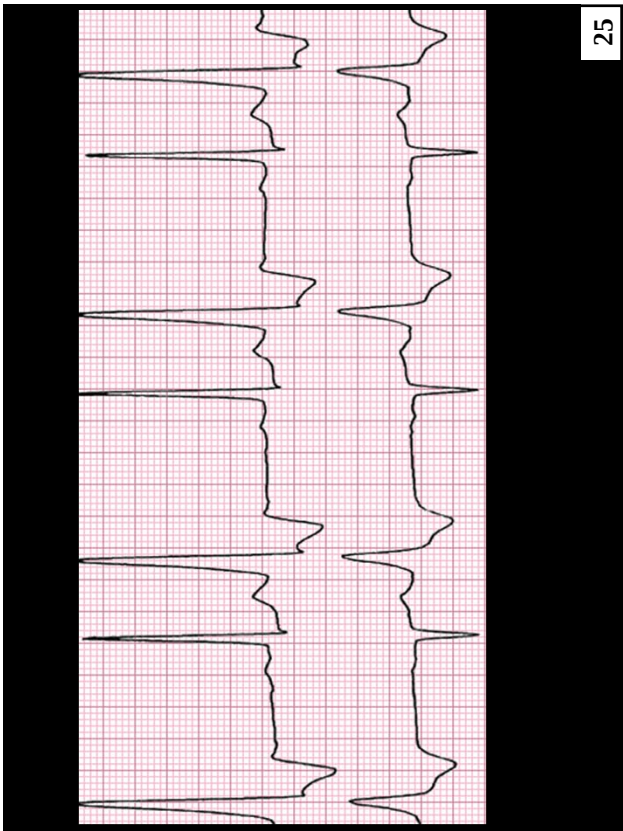


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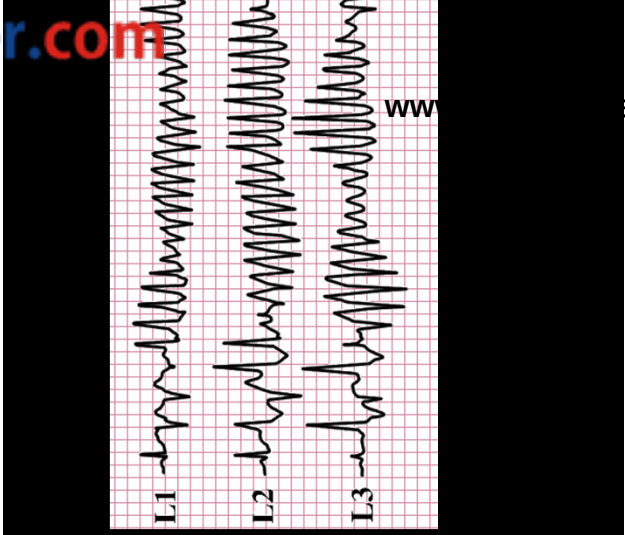


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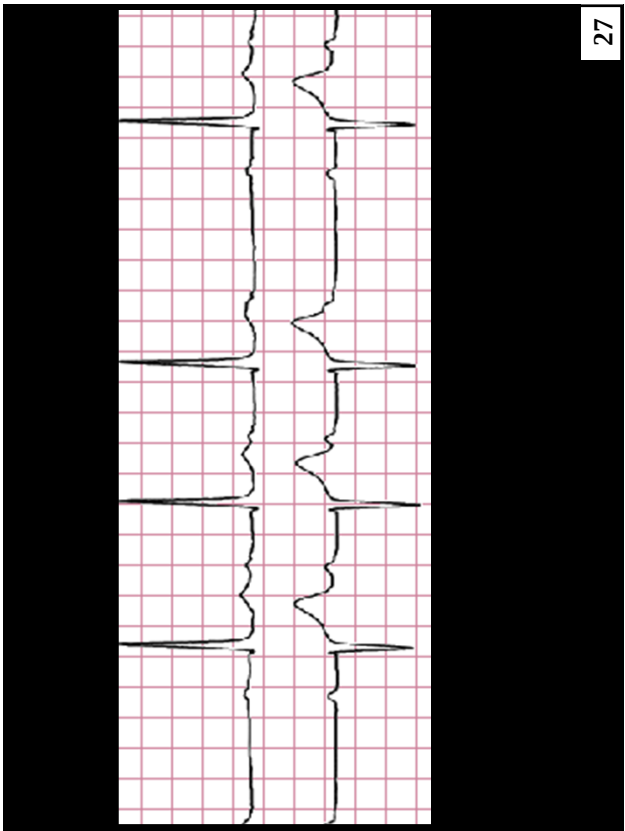


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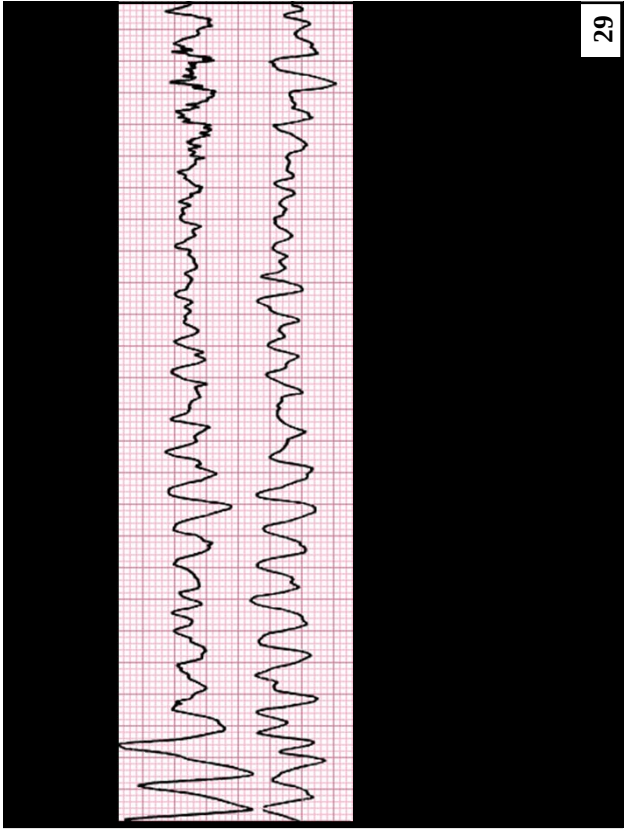
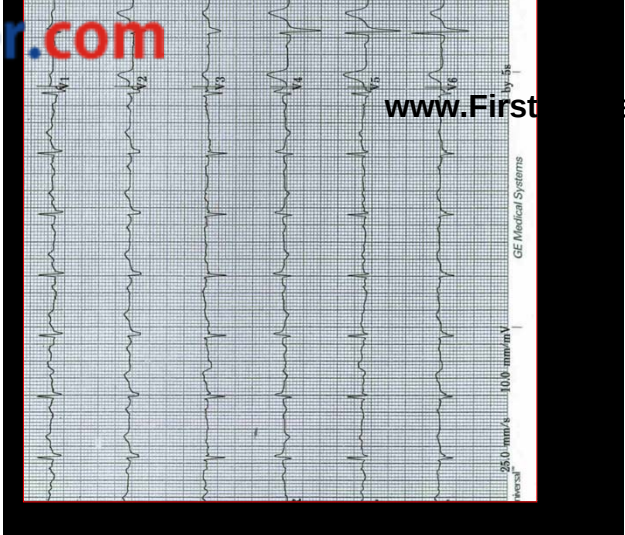


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ECG REVIEW – ECG ANSWER KEY

SLIDE #1

The ECG of this 71-year-old man shows:

- Normal sinus rhythm, rate 70
- Left anterior fascicular block
- Anterior infarct, probably acute with increased ST segment elevation since earlier this date
- Reciprocal inferior ST segment depression

SLIDE #2

ECG: Acute Pericarditis: findings of acute pericarditis:

- ST segment elevations in all limb leads except aVR
- ST segment elevations in all precordial leads except V1
- PR segment depression in multiple leads

NOTE: As is typical for pericarditis, the ST segments are concave upward and the T waves have a return of the ST segments to near baseline. PR segment depression is characteristic and helps confirm pericarditis from ischemia or infarction and early repolarization. In stage three, the T-waves involution normalization in stage four.

SLIDE #3

The ECG of this 64-year-old woman shows:

- Atrial flutter with 4:1 conduction, A-rate 272
- Diffuse ST-T abnormalities
- Poor R wave progression in leads VI

SLIDE #4

The serum calcium must be less than 8 mg/100 mL for recognizable EKG changes to occur. Changes include:

- The QT interval is consistently prolonged.
- The PR interval may also be prolonged.
- Terminal negativity of the T waves in the precordial leads may occur.

SLIDE #5

Sinus rhythm with borderline 1° AVB and Left bundle branch block:

- ST segment elevation in leads V1-V3
- Acute anterior wall myocardial infarction

- Signs of acute injury in the context of LBBB include ST elevation of ≥ 1 mm in leads V1-V4 and segment elevation ≥ 5 mm in leads with negative QRS deflections and ST depression ≥ 1 mm in leads with positive QRS deflections.

SLIDE #6

None of the standard EKG leads overlie the posterior aspect of the heart, so Q waves and ST depression with posterior infarction. Posterior infarction is indicated by reciprocal large R waves and ST depression in the anterior leads, with non-inverted T-waves. It is usually accompanied by inferior infarction as well.

SLIDE #7

EKG findings in this patient with hyperkalemia include:

- Absent P waves: "sinoventricular rhythm" of hyperkalemia
- Prominent narrow based T waves, suggestive of hyperkalemia
- Widening of the QRS is a late finding of hyperkalemia

NOTE: that marked hyperkalemia can depress or suppress activation of the atrial myocardium, resulting in absent P waves, while conduction through the atrial internodal tracts is preserved (sinoventricular rhythm).

SLIDE #8

The ECG of this hypothermic woman shows:

- Bradycardia with an idioventricular or junctional escape rhythm
- Prominent J waves in the anterior leads

This 80-year-old woman was found lying down in her backyard. Her rectal temperature was 80°F (26.7°C). She was unresponsive. Her heart rate was 45 beats per minute. Her blood pressure was 80/50 mmHg. She shows an idioventricular or junctional (more likely) escape rhythm at a rate of less than 100 beats per minute. The axis is normal. There are diffuse ST abnormalities. The positive deflection at the end of the QRS complex in the anterior leads, is known as the Osborn or J wave that is characteristic, but not pathognomonic for hypothermia.

SLIDE #9

The ECG of this 96-year-old woman shows:

- Absent P waves and narrow QRS complexes
- Consider atrial fibrillation
- Regular rate and rhythm with bradycardia, ventricular rate 36
- Junctional escape rhythm
- Non-specific ST-T abnormalities

SLIDE #10

The ECG of this 84-year-old man shows:

- Normal sinus rhythm, rate 65
- Left bundle branch block
- Left axis deviation

SLIDE #11

The ECG of this 85-year-old woman shows:

- Multifocal atrial tachycardia
- Ventricular premature complexes
- Left axis deviation
- Late transition
- Diffuse non-specific T wave abnormalities
- This rhythm may be drug related
- Rare aberrant complexes
- No change from 10/3/94

SLIDE #12

This patient with a pulmonary embolus has a typical "S1, Q3, T3" pattern of acute right ventricular deviation. An S wave develops in aVL or V1 and a Q wave in aVF and L3. The overall pattern is RBBB plus LPFB. In some cases, the T wave becomes negative in the inferior leads, resulting in a pattern. ECG findings include:

- S wave in L1 with
- Q waves in aVF and L3, suggesting
- Acute right ventricular strain

SLIDE #13

This 72-year-old woman's ECG shows:

- Normal sinus rhythm, rate 78
- Right axis deviation
- Right bundle branch block
- Inferior T wave abnormality

SLIDE #14

The ECG in this 96-year-old man with tachy-brady syndrome and syncope shows:

- Sinus bradycardia, rate 47
- A four beat run of supraventricular tachycardia
- Left axis deviation
- Possible inferior infarct
- Old anterior infarct

SLIDE #15

This 75-year-old woman's ECG shows findings of:

- Sinus rhythm, rate 62
- Multiple atrial premature complexes
- Left ventricular hypertrophy by voltage criteria
- QT interval prolongation

- Diffuse ST-T abnormalities, consistent with
- Ischemia and/or subendocardial infarction

SLIDE #16

The ECG of this 45-year-old woman shows:

- Supraventricular tachycardia, rate 189
- Right axis deviation
- Diffuse ST-T abnormalities

There appear to be P wave deflections on the initial part of the ST segment. These are particularly prominent in leads II, III, and aVF. These suggest a supraventricular tachycardia of the short RP interval category, most likely a reentrant tachycardia (AVNRT) or orthodromic AV reentrant tachycardia (AVRT).

SLIDE #17

The ECG of this 59-year-old man shows:

- Regular P waves at a rate of approximately 125/minute
- Complete (third degree) AV block
- Ventricular or junctional escape, rate 29
- Superior QRS axis

SLIDE #18

These EKGs are from a patient with WPW type A in NSR and in tachycardia with antegrade conduction accessory pathway and subsequent slurred QRSs, rapid ventricular response, and hypotension. *NOTE:* In most cases of WPW, antegrade conduction follows the normal pathway during tachycardia, conduction up the accessory path. In those cases, the rate and QRS morphology are the same as in NSR and the delta waves are detectable only during NSR.

SLIDE #19

The ECG shows:

- Sinus rhythm with 1° AVB
- T-wave inversions in I and AVL, indicative of lateral wall ischemia

SLIDE #20

The third and succeeding QRSs on this rhythm strip are wide with retrogradely conducted P waves with a ventricular origin or a junctional origin with Intraventricular conduction delay. The rate is more usual for ventricular escape. Findings summary:

- Wide QRS complexes
- Intermittent retrograde conduction of P waves
- Accelerated idioventricular rhythm or junctional rhythm with intraventricular conduction delay

Retrograde P waves immediately follow the trailing edge of the last four QRS complexes. narrower than the others and are preceded by P waves.

SLIDE #21

This patient has:

- Tachycardia with narrow QRS complexes
- Strongly consider atrial flutter with a 2:1 conduction ratio.

The heart rate is fast and regular at 150. An abnormal P wave precedes each QRS, but may be a rapid rate.

SLIDE #22

This rhythm strip shows findings of:

- Regular rhythm with wide QRS complexes
- Dual pacemaker spikes preceding each QRS complex (AV sequential pacemaker)

NOTE: one spike followed by an abnormal P wave (atrial capture) followed by a second spike preceded by a narrow QRS complex (ventricular capture). Pacing the atrium and ventricle sequentially improves cardiac output.

SLIDE #23

Findings on this ECG rhythm strip include:

- Normal sinus rhythm, with
- Brief run of ventricular tachycardia

SLIDE #24

This rhythm strip shows findings of:

- Regular P waves
- Complete (third degree) AV block with prolonged ventricular asystole
- Ventricular or junctional escape rhythm
- Artificial pacemaker with failure to discharge and failure to capture, earlier in the rhythm strip

NOTE: The missing pacemaker spikes and secondary asystole. Discharge or output failure can be caused by the pacemaker, a weak battery, a cracked or broken wire, electrode displacement, or fibrosis abutting the electrode.

SLIDE #25

Findings on this rhythm strip include:

- Sinus rhythm
- Frequent PVCs in a bigeminy pattern
- Every other complex is a PVC. This is called ventricular bigeminy.

SLIDE #26

Findings on this ECG rhythm strip include:

- Classic "J" wave of hypothermia
- Baseline artifact

SLIDE #27

This ECG rhythm strip shows:

- Mobitz I second degree AV block (Wenckebach)

NOTE: the gradually lengthening PR interval prior to each dropped beat.

SLIDE #28

Simultaneous leads from a patient with hypokalemia showing the torsade de pointes type of ventricular tachycardia (VT). The last three sinus beats, at the beginning of the strip, show an u tachycardia of about 140 beats per minute. The run of VT with a gradually rotating axis begins (adapted from D.M. Krickler and P.V.L. Curry, Br. Heart J. 38:117, 1976)

SLIDE #29

This ECG rhythm strip shows:

- Coarse ventricular fibrillation
- The rhythm is extremely rapid and irregular.

SLIDE #30

Sinus Rhythm with Brugada Syndrome:

- Pseudo RBBB pattern
- ST elevations in V1 and V2
- TX: AICD