12 Lead ECG – Basics and Beyond

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September 2018

Objectives

1. Identify ECG changes associated with myocardial ischemia, injury, and infarction.
2. Associate lead views with the correlating area of the heart.
3. Identify abnormal ECG findings associated with various pathologies.
4. Discuss the management and therapies for identified pathologies.
5. Review the clinical practice guidelines for the acute myocardial infarction patient, including anti-platelet, beta blocker, and statin therapies.

Bipolar Limb Leads

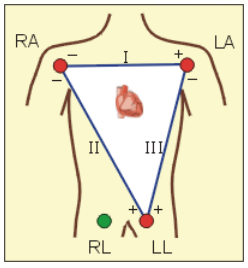
Einthoven's triangle

Lead I
- Measures electrical potential between right arm (-) and left arm (+).

Lead II
- Measures electrical potential between right arm (-) and left leg (+).

Lead III
- Measures electrical potential between left arm (-) and left leg (+).

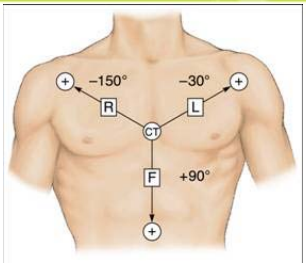
RL	Anywhere above the ankle and below the torso - right
RA	Anywhere between the shoulder and elbow – right
LL	Anywhere above the ankle and below the torso – left
LA	Anywhere between the shoulder and the elbow – left



Unipolar Limb Leads

avR – right arm (+)
avL – left arm (+)
avF – left foot (+)

Right foot is a ground lead.



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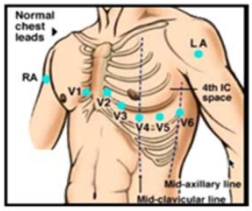
Precordial or Chest Leads



V1	4th intercostal space to the right of the sternum
V2	4th intercostal space to the left of the sternum
V3	Midway between V2 and V4
V4	5th intercostal space at the midclavicular line
V5	Anterior axillary line at the same level as V4
V6	Midaxillary line at the same level as V4 and V5

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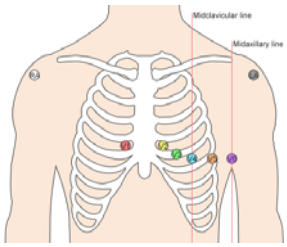
Lead Placement Matters

- Up to 50% of cases have the V1 and V2 electrodes above the 4th intercostal location, which can mimic an anterior MI and cause T wave inversion.
- Up to 33% of cases have the precordial electrodes misplaced, which can alter the amplitude and lead to a misdiagnosis.



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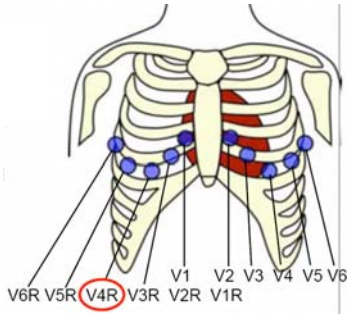
Right Sided ECG



May be useful in the diagnosis of a right ventricular infarct.

- 19-51% of inferior MIs

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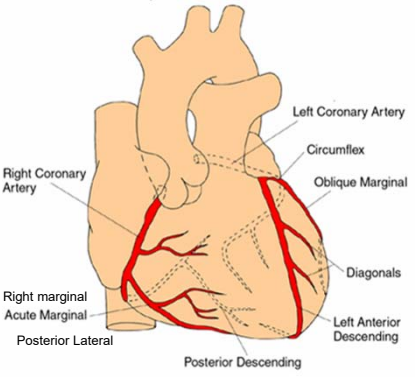
Coronary Arteries of the Heart



or Left Main

or Obtuse Marginal

Some people have an additional coronary artery off the left main called the ramus or intermediate artery.

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Right Coronary Artery (RCA)	Circumflex (Cx)	Left Coronary Artery (LCA,LAD)
SA node – 55% people **AV node, bundle of His** – 90% people **Right atrium** **Inferior left ventricle** Lower 1/3 of septum Major portion anterior right ventricle and posterior right ventricle Posterior left ventricle papillary muscles Posterior division left bundle branch	SA node – 45% people AV node – 10% people **Lateral and posterior left ventricle** Posterior left bundle branch **Left atrium**	Anterior 2/3rds of septum, bundle branches **Left ventricle – anterior, apex, posterior)** Minor portion of right ventricle

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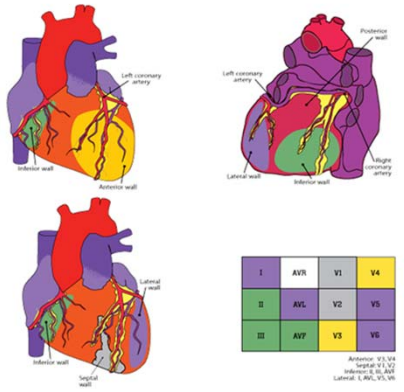
Wall	Leads	Coronary Artery	Reciprocal changes
Anterior	V1, V2, V3, V4	LAD branch of LCA	II, III, aVF
Inferior	II, III, aVF	RCA	I, aVL
Lateral	I, aVL, V5, V6	Circumflex branch of LCA	V1, V3
Posterior	V1, V2 (ST depression, tall R waves)	RCA, Circumflex	
Apical	V3, V4, V5, V6	LAD, RCA	
Anteriolateral	I, aVL, V1, V2, V3, V4, V5, V6	LAD, Circumflex	II, III, aVF
Septal	V1, V2	LAD	



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Steps to Interpreting the ECG

Basic rhythm steps

Rhythm
Rate
P Waves
PR Interval
QRS
QT Interval

Additional 12 Lead steps

Wall of the heart
3 I's of a MI
Axis Deviation
Bundle Branch Blocks
What's not normal
Ugly vs. Dangerous

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Review of electrocardiography

ECG is nothing more than a voltmeter and a stopwatch.

- Timing
- Voltage
  - Scars decrease the voltage.
  - Thick muscle increases the voltage.

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Information at the top of the 12 Lead

Last name, First name			ID: #########		Date and Time
Date of Birth		Vent rate BPM		66	Sinus rhythm with marked sinus arrhythmia
Gender	Race	PR interval	200	ms	ST elevation consider inferior injury or acute infarct
		QRS duration	102	ms	****ACUTE MI / STEMI *****
Location		QT/QTc	394/413	ms	Consider right ventricular involvement in acute inferior infarct
		P-R-T axes	61	52 97	Abnormal ECG
					When compared with ECG of 17-MAY-2006
					ST elevation now present in Inferior leads
					ST now depressed in Anterolateral leads
					T wave inversion now evident in Anterolateral leads

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QT Interval Prolongation

Normal is considered less than half of the R-R
(when the heart rate is ~70).

Conditions Predisposing for Long QT > Torsades

- Baseline long QT
  - **>450 ms, esp > 500 ms**
- Female gender
- Electrolyte disorder
  - Especially low K+ and Mg++
- Bradycardia < 50
- Structural heart disease
- Significant renal or hepatic dysfunction

Common causes:

- Medications
- Electrolyte imbalance
  - Hypokalemia
    - ST flattening, depression, develop U waves
  - Hypomagnesemia
    - Like hypokalemia
  - Hypocalcemia
    - Normal T wave after prolonged QT interval
- CNS catastrophes
  - Stroke, seizure, coma, intra-cerebral or brainstem bleeding
  - Can produce bizarre ST-T waves and some of the longest QT intervals

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Medications that prolong QT interval

Generic name	Brand name
sotalol	Betapace
quinidine	Quiniglute
*amiodarone	Cardarone, Pacerone
*procainamide	Procan, Pronestyl
*disopyramide	Norpace
nicardipine	Cardene
*ibutilide	Corvert
*dofetilide	Tikosyn
trimethoprim-sulfa	Bactrim
*clarithromycin	Biaxin
*erythromycin	EES, Erythrocin
ciprofloxacin	Cipro
levofloxacin	Levaquin
azithromycin	Zithromax
ampicillin	Omnipen
fluconazole	Diflucan
ketoconazole	Nizoral
foscarnet	Foscavir
cocaine	Cocaine
*methadone	Methadone, Dolophine
pseudoephedrine	Sudafed
tacrolimus	Prograf

Generic name	Brand name
albuterol	Ventolin, Proventil
levalbuterol	Xopenex
Salmeterol	Serevent
amitriptyline	Elavil
thioridazine	Mellaril
*haloperidol	Haldol
*mesoridazine	Serentil
risperidone	Risperdal
*chlorpromazine	Thorazine
fluxetine	Prozac
seraline	Zoloft
methylphenidate	Ritalin
chloral hydrate	Noctec
epinephrine	Primatene
norepinephrine	Levophed
phenylephrine	Neosynephrine
ondansetron	Zofran
dobutamine	Dobutrex
dopamine	Intropin
phenylpropanolamine	Dexatrim, Acutrim

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QTc by Bazett's Formula

Step 1
- Find the square root of the R-R interval
- Measure the R-R interval (# of squares x 0.04) then press the sign on a calculator.

Step 2
- Measure the QT interval
- Change the QT interval from seconds to milliseconds (QT .44 secs = 440 ms)

Step 3
- Divide the QT interval in ms by the square root of the R-R interval to calculate the QTc.

Example:

Step 1

R-R is 19 squares x 0.04 = 0.76

Press the square root button

The square root of 0.76 is 0.87

Step 2

QT interval is .48 sec or 480 ms

Step 3

480 ÷ 0.87 = QTc of 552 (551.7) ms

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12 Lead Format

I	AVR	V1	V4
II	AVL	V2	V5
III	AVF	V3	V6

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3 I's of a MI

Injury
- ST elevation on the affected side

Infarction
- Significant Q waves

Ischemia
- Inverted T waves

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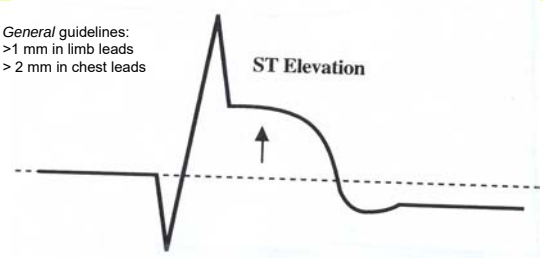
Injury ST Elevation

General guidelines:
>1 mm in limb leads
> 2 mm in chest leads



Acute injury is occurring. Heart attack is happening now.

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Causes of ST Elevation

Acute MI
Injury pattern
Left BBB
Angina with coronary artery spasm
Early repolarization
Left Ventricular hypertrophy
Hyperkalemia
Tako Tsubo cardiomyopathy
Intracranial bleeds
Acute corpulmonale
Myocarditis
Pericarditis
Cholecystitis
Myocardial tumors
Acute pancreatitis
Hypothermia

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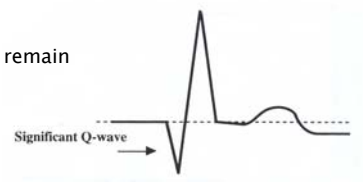
Infarction Significant Q Waves

May or not develop. If they do – Q waves develop over 4 to 24 hours and remain for life.



Significant Q waves are 25-33% of the R wave.
Q > 0.038 seconds

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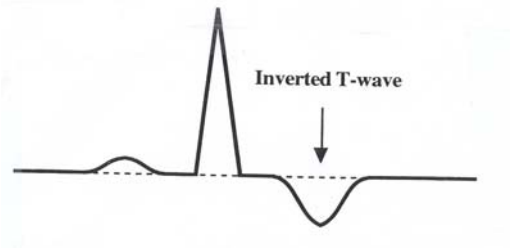
Ischemia Inverted T waves



Supply and Demand problem.

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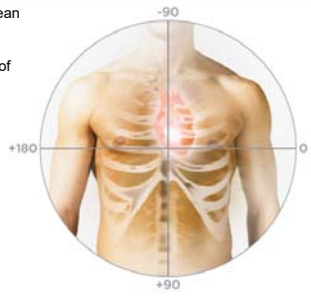
Determining Axis

Direction of the mean electrical vector.

Average direction of current flow.



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Axis

Axis	Lead I	Lead II	Lead III	Comments
Normal 0-90				aVF positive
Physiologic Left Axis 0--40				aVF negative
Pathological Left Axis -40 to -90				Anterior Hemiblock
Right Axis 90-180				aVF positive Posterior Hemiblock
Extreme Right Axis No Man's Land				aVF negative Ventricular in origin

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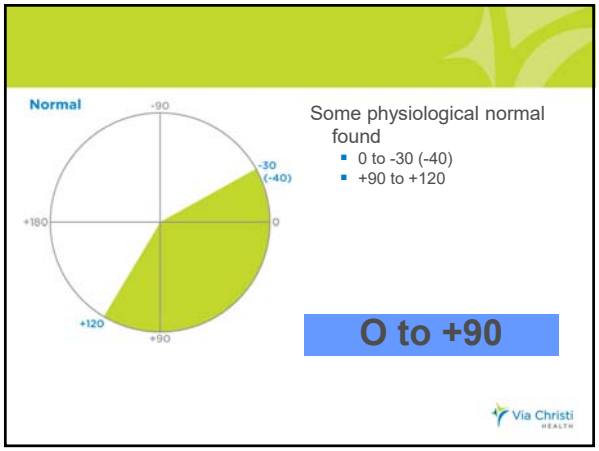
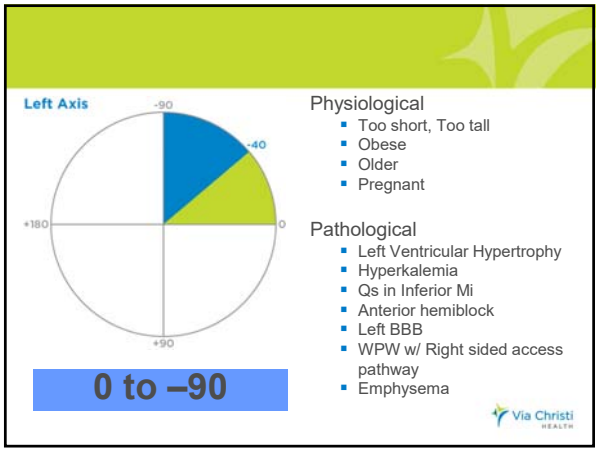
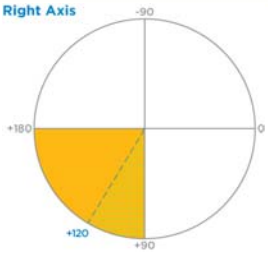




Right Axis



+90 to +180

Physiological
- Normal in children and thin adults

Pathological
- Right Ventricular Hypertrophy
- Anterior-Lateral MI
- Posterior hemiblock
- Right BBB
- COPD w/o Pulmonary HTN
- Pulmonary Emboli
- WPW w/ Left sided accessory pathway
- ASD, VSD

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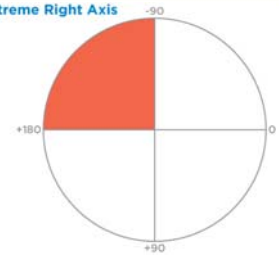
Extreme Right Axis



No Man's Land

Ventricular Tachycardia
Ventricular Pacing
Hyperkalemia

+180 to -90

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Signs and Symptoms of Acute Coronary Syndrome

Classic or usual
- Chest discomfort described as pain, pressure, ache, squeezing, burning or fullness.
- Discomfort or pain in one or both arms
- Shortness of breath with or before chest discomfort
- Diaphoresis - sweating
- Anxiety

Atypical or not usual
- Back, abdominal, neck or jaw pain
- Weakness or fatigue
- Indigestion
- Nausea or vomiting
- Dizziness or lightheadedness

Prodromal symptoms or pre-heart attack symptoms can occur one to six weeks before include:
- Chest pain
- Pain in one shoulder blade or upper back
- Indigestion
- Unusual fatigue
- Anxiety
- Sleep disturbances
- Shortness of breath, especially if no previous awareness of heart disease

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Acute Coronary Syndrome

- ST Elevated Myocardial Infarction- STEMI
  - ST segment is elevated above the isoelectric baseline
  - Classic presentation with elevated cardiac biomarkers
  - New LBBB – *not equivalent since 2013 updated position*
- Non ST Elevated Myocardial Infarction - NSTEMI
  - ST and T-wave changes with elevated cardiac biomarkers
    - Depressed ST, inverted T wave
  - Classical or atypical presentation
- Angina, Unstable angina

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Types of MI

Type 1

- Spontaneous MI related to ischemia due to a primary coronary event such as plaque erosion and/or rupture, fissuring, or dissection.
- Non ST Elevation MI or ST Elevation MI

Type 2

- MI secondary to ischemia due to either increased oxygen demand or decreased supply.
  - Coronary artery spasm, coronary embolism, anemia, arrhythmias, hypertension, or hypotension
  - Respiratory distress, renal failure, sepsis

Type 3

- Sudden unexpected cardiac death, including cardiac arrest, often with symptoms suggestive of MI.
- Accompanied by presumably new ST elevation or new LBBB
- Evidence of fresh thrombus in the coronary artery by angiography

Type 4

- MI associated with coronary angioplasty or stent.

Type 5

- MI associated with coronary artery bypass grafting (CABG)

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Pathological Types

Transmural AMI

- Infarct extends through the whole thickness of the heart muscle, usually resulting in complete occlusion of the area's blood supply.
- Associated with atherosclerosis involving a major coronary artery.
- Subclassified into anterior, posterior, inferior, lateral, or septal.
- ST elevation, and Q-waves

Subendocardial AMI

- Involves a small area in the subendocardial wall of the left ventricle, ventricular septum, or papillary muscles.
- Susceptible to ischemia.
- ST depression, T-wave changes

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AMI Clinical Practice Guidelines (CPGs)

During hospitalization

- Reperfusion strategies
- Aspirin within 24 hours before or after arrival
- Smoking (tobacco) cessation advice/counseling

At Discharge

- Aspirin
- Beta-Blocker
- Statin
- ACE-I or ARB therapy for left ventricular systolic dysfunction, EF (ejection fraction) $\leq$ 40%

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STEMI Reperfusion Strategy

Door-to-needle goal of 30 minutes Thrombolytic (fibrinolysis) therapy

- **TNKase (tenecteplase)**
- Activase (t-PA, alteplase)
- Retavase (r-PA, reteplase)
- Streptokinase (Streptase)

Door-to-Balloon (D2B) within 90 minutes

- Angioplasty
  - PTCA – Percutaneous Transluminal Coronary Angioplasty
- Coronary artery stents
- Atherectomy Percutaneous Coronary Intervention

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Percutaneous Coronary Intervention - PCI

- Atherectomy
- Angioplasty
- Coronary stenting
  - Bare metal (BMS)
  - Drug eluting (DES)
  - Bioresorbable (BVS)

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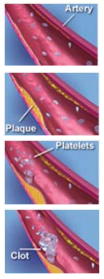
Antiplatelet Therapy

Aspirin

- 162 to 325 mg initially
- 81 (75-100 mg) daily



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Duration of Dual Antiplatelet Therapy

New 2016 guidelines

- http://circ.ahajournals.org/content/134/10/e123

- Generally
  - ASA indefinitely
  - Dual platelet therapy for 12 months
    - Less if high risk of bleeding

Recommendations for Duration of DAPT in Patients With ACS Treated With PCI

COR	LOE	Recommendations
I	B-R	In patients with ACS (NSTE-ACS or STEMI) treated with DAPT after BMS or DES implantation, $P2Y_{12}$ inhibitor therapy (clopidogrel, prasugrel, or ticagrelor) should be given for at least 12 months.
I	B-NR	In patients treated with DAPT, a daily aspirin dose of 81 mg (range, 75 mg to 100 mg) is recommended.
IIa	B-R	In patients with ACS (NSTE-ACS or STEMI) treated with DAPT after coronary stent implantation, it is reasonable to use ticagrelor in preference to clopidogrel for maintenance $P2Y_{12}$ inhibitor therapy.
IIa	B-R	In patients with ACS (NSTE-ACS or STEMI) treated with DAPT after coronary stent implantation who are not at high risk for bleeding complications and who do not have a history of stroke or TIA, it is reasonable to choose prasugrel over clopidogrel for maintenance $P2Y_{12}$ inhibitor therapy.
IIb	A[SR]	In patients with ACS (NSTE-ACS or STEMI) treated with coronary stent implantation who have tolerated DAPT without a bleeding complication and who are not at high bleeding risk (eg, prior bleeding on DAPT, coagulopathy, oral anticoagulant use), continuation of DAPT (clopidogrel, prasugrel, or ticagrelor) for longer than 12 months may be reasonable.
IIb	C-LD	In patients with ACS treated with DAPT after DES implantation who develop a high risk of bleeding (eg, treatment with oral anticoagulant therapy), are at high risk of severe bleeding complication (eg, major intracranial surgery), or develop significant overt bleeding, discontinuation of $P2Y_{12}$ inhibitor therapy after 6 months may be reasonable.
III: Harm	B-R	Prasugrel should not be administered to patients with a prior history of stroke or TIA.

SR indicates systematic review.

Antiplatelet Options

Name	Classification	Dosing – Std Concentration	Comments
Aspirin	Anti-platelet, attaches to TXA_2 site	162-325 mg loading, then 81 (75-100) mg PO per day	
Plavix (clopidogrel)	Anti-platelet, attaches to ADP $P2Y_{12}$ site	300-600 mg PO loading, then 75 mg daily	Do not take with PPI, especially Prilosec
Effient (prasugrel)	Anti-platelet, attaches to ADP $P2Y_{12}$ site	60 mg PO loading, then 10 mg daily	Caution in patients > 75 years old, < 60 kg. Box warning not to give if history of stroke or TIA.
Brilinta (ticagrelor)	Anti-platelet, attaches to ADP $P2Y_{12}$ site	180 mg PO loading, then 90 mg twice a day	ASA to be limited to 75-100 mg/day
Integrilin (eptifibatide)	Anti-platelet, attaches to GP IIb IIIa	2 mcg/kg/min infusion 12 to 24 hours after PCI -Decrease to 1 mcg/kg/min for renal impairment	Reversible in 2.5-4 hours. Don't get patients OOB until 2-2.5 hours after infusion is shut off.
ReoPro (abciximab)	Anti-platelet, attaches to GP 11a IIb	0.25 mg/kg bolus, then 10 mcg/min infusion x 18-24 hours or stop 1 hour after PCI	No renal dosing. 4 hour effect with half-life of 30 minutes

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Beta Blockers

- Reduce catecholamine levels
- Decrease myocardial ischemia and limit infarct size
- Reduce myocardial workload and oxygen demand
- Reduce heart rate and blood pressure
- Reduce supraventricular and malignant ventricular arrhythmias

Metoprolol – Lopressor, Toprol XL
Carvedilol – Coreg
Bisoprolol - Zebeta
Atenolol – Tenormin
Sotalol – Betapace
Betaxolol – Kerlone
Propranolol – Inderol
Esmolol – Brevibloc (IV)
Labetalol – Normodyne (IV)

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Common Beta Blockers

Beta blockers

Drug	Initial Daily Dose	Maximum Dose
carvedilol (Coreg)	3.125 mg BID	50 mg BID
Carvedilol extended release (Coreg CR)	10 mg daily	80 mg daily
metoprolol succinate extended release (Toprol XL, generic)	12.5-25 mg daily	200 mg daily
bisoprolol (Zebeta)	1.25 mg daily	10 mg daily
Atenolol (Tenormin)	50 mg daily	100 mg (200) daily

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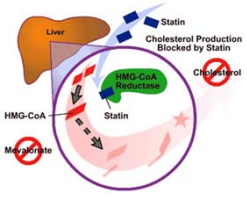
HMG-CoA Reductase



Cholesterol is synthesized in the smooth endoplasmic reticulum by a series of chemical reactions.

The first way to block cholesterol synthesis is to interrupt the conversion of HMG CoA to mevalonate.

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HMG-CoA Reductase Inhibitors or Statins

2013 guideline update

- Lifestyle modification
  - Diet, exercise, lose weight
- Assess ASCVD risk
- Four Benefit Groups
  - Individuals with clinical ASCVD
  - Individuals with primary elevations of LDL-C ≥190 mg/dL
  - Individuals age 40-75 with diabetes and LDL-C of 70-189 mg/dL without clinical ASCVD
  - Individuals without clinical ASCVD or diabetes who are age 40-75 with LDL-C of 70-189 mg/dL and estimated 10 year ASCVD risk of ≥7.5%

http://content.onlinejacc.org/article.aspx?articleid=1879710

Atorvastatin – Lipitor
Rosuvastatin – Crestor
Simvastatin – Zocor
Pravastatin – Pravachol
Lovastatin – Mevacor

Guidelines level to high or moderate intensity dosing.

Adverse effects – muscle aching, increase in liver enzymes

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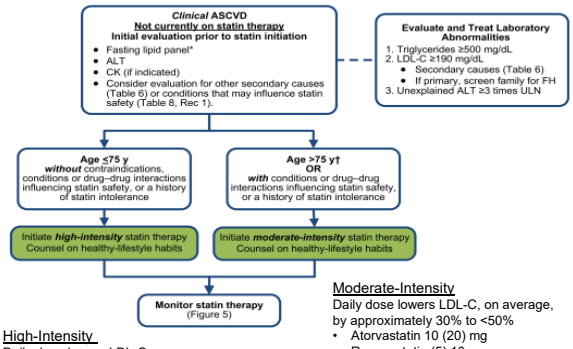

High-Intensity
Daily dose lowers LDL-C, on average, by approximately ≥50%
- Atorvastatin (40) – 80 mg
- Rosuvastatin 20 – (40) mg

Moderate-Intensity
Daily dose lowers LDL-C, on average, by approximately 30% to <50%
- Atorvastatin 10 (20) mg
- Rosuvastatin (5) 10 mg
- Simvastatin 20-40 mg
- Pravastatin 40 (80) mg
- Lovastatin 40 mg

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PCSK9 Inhibitors

By blocking PCSK9's ability to work, more receptors are available to get rid of LDL cholesterol from the blood and, as a result, lower LDL cholesterol levels

Alirucumab (Praluent)

- 75 mg or 150 mg SQ every 2 weeks

Evolocumab (Repatha)

- 140 mg every 2 weeks or 420 mg once monthly
- 420 mg dose - Single use body infusor over 9 minutes or 3 injections within 30 minutes

These are additions to statin therapy.

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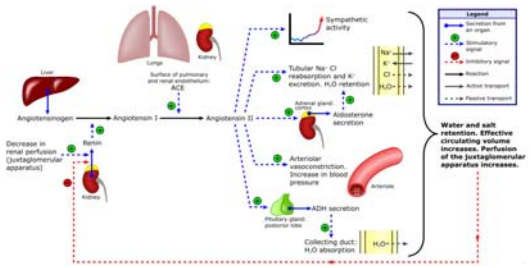
Renin-Angiotensin-Aldosterone System



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ACE-I & ARBs

ACE-I

Lisinopril – Prinivil, Zestril
Captopril – Capoten
Ramipril - Altace
Enalapril – Vasotec
Fosinopril – Monopril

Adverse effect – cough, angioedema, hyperkalemia
Watch renal function.

ARB

Losartan – Cozaar
Valsartan - Diovan
Candesartan - Atacand

Tend not to have as many adverse effects. Cough not really seen.

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ACE-I and ARBs

ACE Inhibitors

Drug	Initial Daily Dose	Maximum Dose
Captopril	6.25 mg TID	50 mg TID
Enalapril	2.5 mg BID	10-20 BID
Fosinopril	5-10 mg daily	40 mg daily
Lisinopril	2.5-5 mg daily	20-40 mg daily
Ramipril	1.25-2.5 daily	10 mg daily

ARBs

Drug	Initial Daily Dose	Maximum Dose
Losartan	25-50 mg daily	50-150 mg daily
Valsartan	20-40 mg BID	160 mg BID
Candesartan	4-8 mg daily	32 mg daily

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Patient Safety Indicators

AMI Quality Measures

Quality Measure	Definition	Weight in Composite	Collection
30-Day Mortality	30-day, all cause, risk-standardized mortality rate following a hospitalization for AMI	50%	Claims-based per IQR (NQF #0230)
AMI Excess Days	Excess days in acute care, including emergency department, observation, and inpatient readmission days following a hospitalization for AMI	20%	Claims-based per IQR
HCAHPS Survey	Patient experience composite measure (akin to star rating measure) not specific to DRGs. Reflects elements of care such as communication, pain management, discharge/transition information, cleanliness, and quietness.	20%	Patient Survey (NQF #0166)
Hybrid AMI Mortality Voluntary Data	30-day, risk-standardized AMI mortality rate, using a combination of claims data and EHR data submitted by hospitals	10%	Voluntary submission (NQF #2473)

September 2017 AHA / ACA New Performance Measures for MI

1. Immediate angiography for resuscitated out-of-hospital cardiac arrest in STEMI patients
2. Noninvasive stress testing before discharge in conservatively treated patients
3. Early cardiac troponin measurement, within 6 hours of arrival
4. Participation in a regional or national acute-MI registry

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September 2017 AHA / ACA New Quality Measures for MI

1. Risk-score stratification for NSTEMI patients
2. Early invasive strategy, within 24 hours, in high-risk NSTEMI patients
3. Therapeutic hypothermia for comatose STEMI patients with out-of-hospital cardiac arrest
4. Aldosterone antagonist at discharge
5. Inappropriate in-hospital use of NSAIDS
6. Inappropriate prescription of prasugrel at discharge in patients with a history of prior stroke or TIA
7. Inappropriate prescription of high-dose aspirin with ticagrelor at discharge

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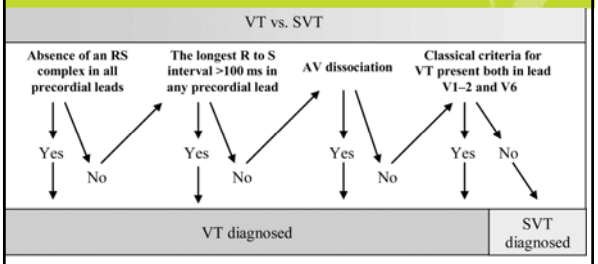
Brugada Criteria for Ventricular Tach



1. Is there extreme right axis?
2. Is there an RS complex?
3. Is the QRS wide?
4. Is there AV Dissociation?
5. Is there concordance in V1-V6?

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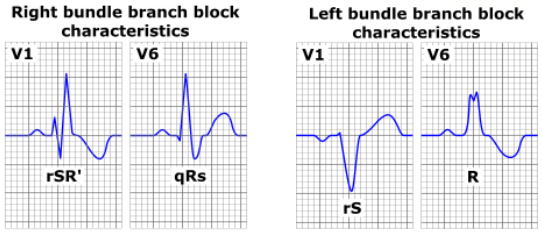
Right versus Left BBB



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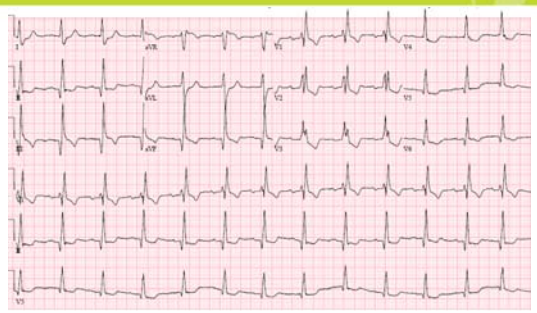
RBBB - Right Bundle Branch Block



54 QRS 162 ms

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RBBB

Look at V1 lead

- **QRS is ≥ 0.12 seconds**
  - An incomplete BBB measures < 0.12 sec.

Right BBB is blocked.

- Electrical impulse is going Left > Right
- Right ventricle conducts later than left ventricle

Physiological

- Athletes
  - Increased muscle mass

Pathological

- CAD
- Pulmonary HTN
- Inflammatory disease
- Lesions of the septum
- New RBBB after bypass surgery is a + periop MI

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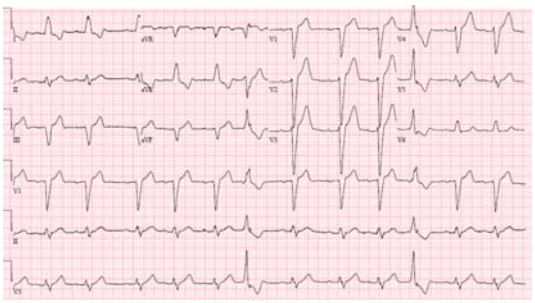
LBBB – Left Bundle Branch Block



56 QRS 144 ms

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LBBB

Look at V1 lead

- **QRS is ≥ 0.12 seconds**
  - An incomplete BBB measures < 0.12 sec.

Left BBB is blocked

- Electrical impulse is going Right > Left
- Left ventricle conducts later than right ventricle

Left bundle of HIS has 3 fascicles (fascicular block)

- Anterior (superior)
- Posterior (inferior)
- Midseptum

Pathological

- CAD
- Hypertension
- Dilated cardiomyopathy
- Calcified aortic valve, stenosis
- Aortic root dilation and aortic regurgitation
- Degenerative heart disease

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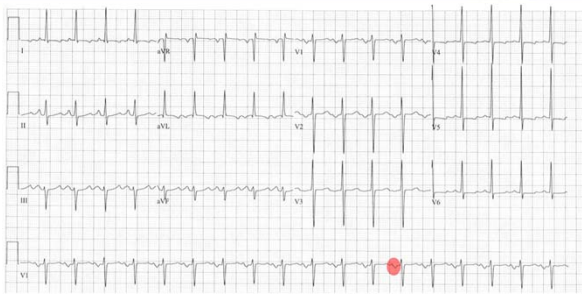
Left Atrial Hypertrophy



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Left Atrial Hypertrophy

Hypertension

Valvular Heart Disease

- Mitral stenosis
- Mitral regurgitation
- Aortic stenosis

Heart Failure

Ventricular Septal Defect

Cardiac myoma

Causes

Broad or notched P–waves

Prolonged P wave

- V1 broad trough
- I, II, & V4-V6 notched

What will see

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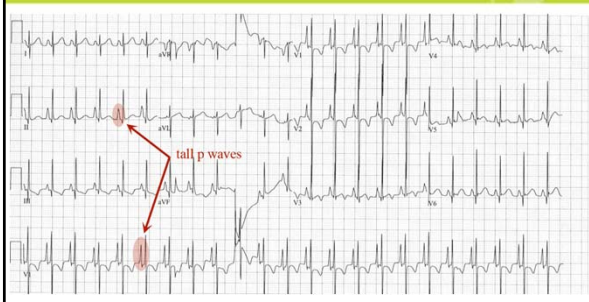
Right Atrial Hypertrophy



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Right Atrial Hypertrophy

Lung disease
- COPD

Pulmonary Embolus

Pulmonary Hypertension

Right ventricular failure

Tricuspid regurgitation or stenosis

Atrial Septal Defects

Causes

Tall, peaked P-waves
- II, III, aVF
- ≥ 2.5 mm tall in the inferior leads

What will see

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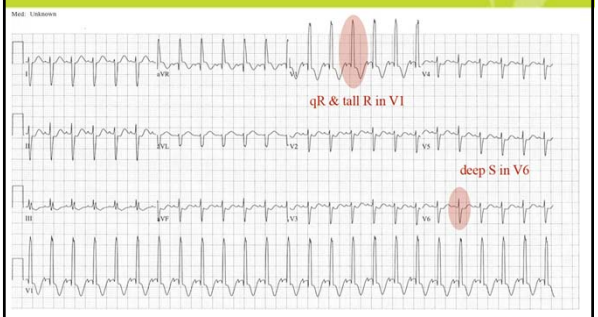
Right Ventricular Hypertrophy



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Right Ventricular Hypertrophy

Increased right ventricular mass
- Pulmonary stenosis or regurgitation
- Primary pulmonary hypertension
- Pulmonary embolus
- Diastolic overload
- Atrial septal defect
- Congenital heart disease

Causes

Right axis deviation

Tall "R" waves in right precordial leads
- V1 most sensitive

Deep "S" waves in left precordial leads
- V6

What will see

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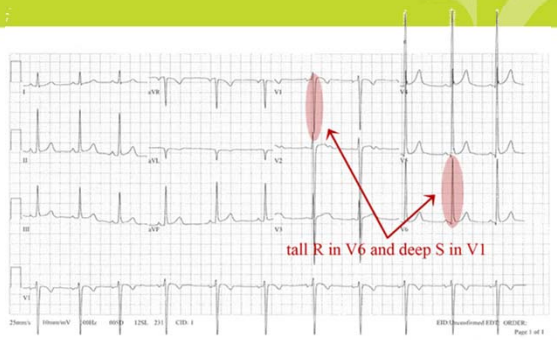
Left Ventricular Hypertrophy



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Left Ventricular Hypertrophy

Increased LV muscle mass

- **Hypertension**
- Cardiomegaly
- Cardiomyopathy
- Aortic stenosis and regurgitation
- Mitral regurgitation

Left axis deviation

Measure
V1 or V2 Deepest "S" wave
PLUS
V5 or V6 Tallest "R" wave

#mm add up > 35 mm

Causes | **What will see**

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Aortic Stenosis

Left Ventricular strain pattern

- Left Ventricular Hypertrophy
- Left atrial enlargement
- Left axis deviation
- Conduction defects
  - LBBB, RBBB, AV blocks

Testing sequence

- History and physical, Lab
- Chest x-ray and 12 Lead ECG
- Echocardiography and Doppler
- Cardiac catheterization*
  - 50% with critical AS have CAD

ECG not diagnostic, may see

- ST depression and T-wave inversion in anterior and lateral leads
- LV hypertrophy
  - Absence does not preclude AS
- Sub-endocardial ischemia

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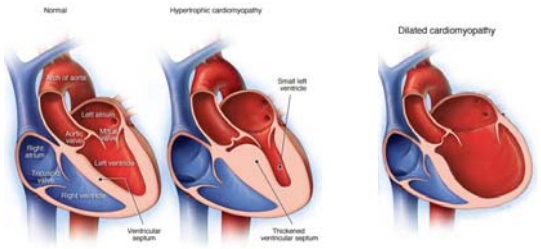
Hypertrophic Cardiomyopathy



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Hypertrophic Cardiomyopathy

Previously known as
- Hypertrophic obstructive cardiomyopathy – HCOM
- Idiopathic hypertrophic subaortic stenosis – IHSS

Number one cause of sudden cardiac death in young athletes (1-2%).

Inheritance is primarily autosomal dominant.

ECG changes
- Left ventricular hypertrophy pattern
  - Tall R waves
  - Large precordial voltages
- Deep, narrow "dagger-like" Q waves in lateral and inferior leads
- Giant T-wave inversion in apical HCM
- Left atrial enlargement
- Atrial fibrillation and SVTs are common

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12 Lead ECG as a Screening Test

United States does not require
- Italy and Israel do

Issues
- Placement of leads
- 30% false positives

Ethical issues
- Consent for screening
- Who receives results
- Who makes the determination of risk with participation in activities

Read more, including recommended 14 element screening at

http://circ.ahajournals.org/content/130/15/1303

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Treatment and Management

Medical

- No highly strenuous activity
- Control blood pressure
  - Beta blockers
  - Calcium channel blockers
- Amiodarone
- Norpace (disopyramide)
- Cautious with diuretics
- Avoid inotropes, nitrates, sympathomimetic amines

Surgical

- Surgical septal myectomy
- Alcohol septal ablation
- Heart transplant

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Wolff-Parkinson White

Sinus impulses bypass the AV node via an accessory pathway (AP) conduction.

- Uncommon - ~2 per 1,000 in the general population
- Can be right-sided, left-sided, anterior, or posterior – and sometimes more than a single AP.
- A very fast atrial fibrillation (250-300) – think WPW.

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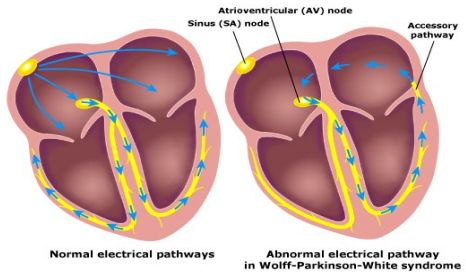
Accessary Pathway



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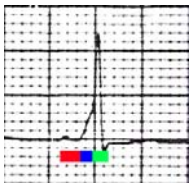
WPW

Three key signs:

- Delta wave which may be positive or negative
- QRS widening
- Short PR interval



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Treatment and Management

Acutely	Long Term
Adenosine	Catheter ablation
Consult cardiology	Flecainide (Tambocor) Sotalol (Betapace)

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Pulmonary Embolus

Look at the sum of all in context with the clinical history.

- ECG is *not* diagnostic.
- Can strongly suggest before the V/Q or CT scan.

Old – S_I-Q_{III}-T_{III} "classic" finding is neither sensitive nor specific.

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Suspect PE?

New onset dyspnea, pleuretic

Typically tachycardic

- Most common, seen in 44% of cases

- RBBB
  - Complete or incomplete
- **Right Ventricular strain pattern**
  - **T wave inversion in V1, V2, V3, also V4**
  - **T Wave inversion II, III, aVF**
- Right axis deviation
  - Extreme right axis may occur between 0 and -90, giving appearance of left axis (pseudo left axis)
- Dominant R wave in V1
  - Manifestation of acute right ventricular dilation
- RA enlargement
  - Peaked P waves in lead II
- Wide S in lead I, subtle S in V6
- ST elevation in aVR

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12 Lead ECG- Basics and Beyond

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Disclaimer: The overview is not all inclusive and I recommend reviewing the ACC/AHA guidelines.

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