12 LEAD ECG VARIANTS, MIMICS AND ADVANCED SKILLS

CYNTHIA WEBNER DNP, RN, CCNS, CCRN-CMC
KAREN MARZLIN DNP, RN, CCNS, CCRN-CMC

ECG CONSIDERATIONS WITH BUNDLE BRANCH BLOCKS
CONDUCTION SYSTEM REVIEW

- Left Bundle Branch
  - Left anterior fascicle
  - Left posterior fascicle
- Right Bundle Branch
- Purkinje Network
- Purkinje Fibers

NORMAL DEPOLARIZATION

QRS .06-.10 sec
BUNDLE BRANCH BLOCK

- QRS complex is 0.12 sec or greater
- Incomplete BBB measures from 0.10 to 0.11

RIGHT BUNDLE BRANCH BLOCK CAUSES

- CAD
- Disease of right side of the heart
- Cor pulmonale
- Cardiomyopathy
- Congenital lesions
- A-S Defects
- Pulmonic Stenosis
- Pulmonary Embolism
RIGHT BUNDLE BRANCH BLOCK

- V1
  - Triphasic complex rsR' pattern - positive
  - Or an M shaped R wave with right peak taller
  - Or a qR pattern

- V6
  - Triphasic complex
  - qRs with wide S waves
  - Positive

V1 = rsR’
V6 = qRS
QRS = .12 sec or more
LEFT BUNDLE BRANCH BLOCK

CAUSES

• Left Ventricular Hypertrophy
• MI
• CAD
• Aortic Stenosis
• Cardiomyopathy
• Hypertensive cardiomyopathy
LEFT BUNDLE BRANCH BLOCK

$V_1 = QS$
$V_1 = rS$

QRS = .12 sec or more

$V_6 = wide R$

V1 = rS

LEFT BUNDLE BRANCH BLOCK

- $V_1$
  - Wide QS or rS complex - negative
  - Slick downstroke
  - Nadir <0.06 sec

- $V_6$
  - Wide R wave with no initial septal q wave - - positive

$V_6$
LEFT ANTERIOR HEMIBLOCK

Causes
• Ischemia
• Valvular disease
• Cardiomyopathy
• Congenital heart disease
• Rarely normal

• Blood supply received from septal branch of LAD (or AV nodal artery of RCA)
LEFT ANTERIOR HEMIBLOCK

- Block of anterior – superior fascicle of the LBB
- Left axis deviation
  - -30° to –75°
  - Become suspicious at -30°
  - Definitive at – 40 to 45°
  - Common at -60°

Key for recognizing -60° Axis
- aVR most equiphasic limb lead

- Commonly seen in anterior wall MI
- Low mortality if isolated
- Left anterior hemiblock in association with RBBB during AMI
  - Associated with left main occlusion and high mortality

LEFT ANTERIOR HEMIBLOCK

- Lead 2, Lead 3 and aVF
  - rS pattern
  - Small r waves
  - Slightly wide / deep S waves
  - Increased limb lead voltage
- Lead 1 and aVL
  - qR pattern
- Normal QRS duration
ST CHANGES WITH RBBB

- Normal direction of T wave (discordant)
  - In V₁ – V₃
- T wave inverted in leads with rSR’ pattern
- T wave upright in leads with S wave
- ST elevation is usually due to injury
- Measure QRS in easiest identified lead
- Determine the end of the QRS in any lead
ST CHANGES WITH LBBB

- Discordant ST and T waves in precordial leads
- LBBB typically manifest ST elevation certain leads in the absence of injury so assessment of STEMI is challenging

- Left BBB is common reason for delayed or withheld reperfusion
- New LBBB and clinical signs of AMI are indication for reperfusion therapy
- Old LBBB with increased ST elevation or specific indictors should also receive reperfusion
DIAGNOSTIC STRATEGIES FOR AMI WITH LBBB

- √ Concordant ST elevation > 1 mm in leads where QRS is predominantly positive
  - V5, V6, I, aVL, II
- √ Concordant ST depression > 1 mm in one or more leads where QRS is predominantly negative
  - V1 – V4
- √ Discordant ST elevation > 5 mm and disproportionate with the QRS voltage.
NORMAL VARIANTS
**EARLY REPOLARIZATION**

- Precordial ST elevation in most adults
  - Up to 90%
- Early repolarization most common normal variant
  - African American men < 50 years of age

? Increased Risk of Sudden Death

---

**ST ELEVATION OF EARLY REPOLORIZATION**

- ST segments highest in V2-V3
- ST elevation up to 5 mm
- J point elevation
- Concavity, not convexity
- Tall peaked asymmetric T waves
- T waves are tall (not too tall) but not wide
- ST elevation < 0.5 m in lead V5 and V6
- T wave amplitude in V6 > V1
- No reciprocal ST depression
- Less common after age 55
ST ELEVATION OF EARLY REPOLARIZATION

- Rarely seen in limb leads alone
- Right precordial early repolarization ("humpback")
- J wave (fishhook)

ECGs over time with early repolarization.

May be confused with or concurrent with LVH.
PERSISTENT JUVENILE T WAVES

- Negative (inverted) T waves in V1-V3 (right precordial leads)
- Not “deep” inversions
- Usually in young, healthy women
- More common in African Americans

Note: Infants > 48 hours through childhood have inverted right precordial T waves. Progressive change to upright through late childhood.
RSR’ PATTERN

- V1 and V2
- r’ amplitude < r
- QRS interval < 0.11 seconds

- When r’ changes to R’ consider incomplete RBBB

? Fragmented QRS and non STEMI

ECG MIMICS
LVH

- Common reason for false positive ST elevation
- Anatomic LVH may be present in absence of ECG criteria
  - To ascribe ST elevation to LVH the ECG must meet the voltage criteria

LEFT VENTRICULAR HYPERTROPHY

- V1 and V2
  - Deeper than normal S waves
  - Small r waves
- V5 and V6
  - Taller than normal R waves
  - Small S waves
LVH VOLTAGE CRITERIA

• One or more voltage criteria
• Only applicable if QRS is < 120 ms

• Precordial lead voltage criteria
  • R-wave in V5 or V6 > 26 mm
  • R-wave in V5 or V6 + S-wave in V1 > 35 mm
  • Largest R-wave + largest S-wave in precordial leads > 45 mm

ST – T WAVE CHANGES SECONDARY TO LVH

• ST elevation is generally discordant
  • ST elevation in V2 -V3
  • ST elevation in lead III
  • ST depression in V4-V6
    • Previously called strain pattern
    • Down sloping – not horizontal

Not due to LVH
• ST elevation in lateral leads
• ST depression in V2-V3
RIGHT VENTRICULAR HYPERTROPHY

- RV Hypertrophy
  - Right Axis deviation is one of earliest signs
  - Reverse R wave progression
  - Dominant R wave in V1 and V2
  - Deep S wave in V5 and V6

DIGITALIS EFFECT

- Sagging depression of ST segment in leads with positive QRS
- Reduced T wave amplitude
- Increased U wave amplitude
- Difficult to evaluate if hypertrophy or BBB
PERICARDITIS: ECG FINDINGS

- Mimics: anteroinferior; inferolateral; antero-inferolateral MI
- ST Elevation
  - ST elevation typically greatest in II and V5 (also I and V6)
  - ST elevation may also be in V1–V4; aVF, III and aVL (least)
  - Upwardly concave ST segments
  - ST elevation usually ≤ 5 mm
PERICARDITIS: ECG FINDINGS

- Other ST changes
  - ST depression in aVR
  - Minimal depression V1, III, aVL may exist

- PR Segment depression
  - PR depression most common in II, aVF and V4 – V6
  - PR elevation > 0.5 mm in aVR

- Electrical Alternans
  - Voltage changes with pericardial effusion or tamponade

STAGES OF PERICARDITIS

- **Stage I**
  - ST elevation
  - More concave
  - Lasts up to 2 weeks

- **Stage II**
  - ST to baseline
  - Decrease T wave amplitude
  - Lasts from days to several weeks

- **Stage III**
  - T wave inversion
  - Starts at end of second to third week

- **Stage IV**
  - Gradual resolution
  - T wave may stay inverted up to 3 months
PERICARDITIS

- Diffuse Pericarditis
  - Easiest to differentiate with both pain and ECG assessment
- Localized Pericarditis
  - May have reciprocal changes

Perimyocarditis
- Troponin
- Wall motion abnormalities

CLASSIC PERICARDITIS
PERICARDITIS
ACUTE COR PULMONALE

- Transient ECG changes
- ST or atrial tachycardia (or fib / flutter)
- T wave inversion (or other ECG signs of ischemia, injury, infarction) in both inferior and anteroseptal leads
- Elevated ST segments aVR and V1-V2
- Prominent S waves in I and aVL
- RAD or incomplete or complete RBBB
- Widespread S waves
- Prominent P waves in inferior leads
  *(right atrial strain)*
ACUTE COR PULMONALE

PULMONARY EMBOLISM

- Obstruction of blood flow to one or more arteries of the lung by a thrombus (other emboli – fat, air, amniotic fluid) lodged in a pulmonary vessel
- Lower lobes frequently affected due to increased perfusion
RISK FACTORS FOR PE

- Stasis of blood
- Prolonged immobilization after surgical procedures
- Plaster casts
- Venous obstruction
- Heart failure / Shock / Hypovolemia
- Varicose veins
- Obesity
- Hypercoagulability
  - Polycythemia vera
  - Sickle cell disease
  - Malignancy
  - Pregnancy
  - Recent trauma
  - Oral contraceptives
- Injury to the vascular endothelium
  - Central venous and arterial catheters
  - Phlebitis

PULMONARY EMBOLISM: PATHOPHYSIOLOGY

- > 90% of thrombus develop in deep veins of iliofemoral system
  - Can also originate in the right side of the heart, pelvic veins, and axillary or subclavian veins.
  - Another source is around indwelling catheters.
- Thrombus formation leads to platelet adhesiveness and release of serotonin (vasoconstrictor).
- Dislodgement of thrombus
  - Intravascular pressure changes (standing, massaging legs, fluid challenge, valsalva maneuver).
  - Natural clot dissolution (7-10 days after development).
PULMONARY EMBOLISM:
PATHOPHYSIOLOGY
• Clot lodges in pulmonary vessels
• Ventilation continues but perfusion decreases
  • Increase in alveolar dead space
  • Alveolar CO2 decreases
• Overperfusion of uninvolved lung results in a decreased V/Q ratio
  • Hypoxemia can occur due to ventilation / perfusion mismatching.
• Decreased blood flow damages type II pneumocytes, which results in a decrease in surfactant production. (atelectasis)
  • Pulmonary edema can develop as secondary complication
• Increased PVR can lead to pulmonary hypertension and potential acute cor pulmonale.
• Cardiogenic shock can occur as the result of right-ventricular failure.

PULMONARY EMBOLUS:
CLINICAL PRESENTATION
• Large to massive when 50% of pulmonary artery bed is occluded
  • Impending doom
  • Hypoxemia
  • Syncope
  • Sign and symptoms of right heart strain or right-ventricular failure
  • Signs of right-ventricular strain on ECG.
  • Sudden shock
  • Pulseless electrical activity
• Medium-sized emboli
  • Dyspnea
  • Substernal chest discomfort/pleuritic chest pain
  • Many non-specific signs
  • Tachypnea
  • Tachycardia
  • Rales
  • Accentuated 2nd heart sound
PULMONARY INFARCTION

• Pulmonary infarction is infrequent
• More common
  • Large embolus
  • Pre-existing lung disease
• Results in alveoli filling with RBCs and inflammatory cells
• Complicated by infection
  • Abscess

• Signs and Symptoms
  • Pleuritic chest pain
  • Dyspnea
  • Hemoptysis
  • Cough
  • Pleural friction rub

---

57

---

58
PULMONARY EMBOLUS: TREATMENT

- Prevent thrombus formation
  - Compression stockings that provide a 30-40 mm Hg or higher gradient
  - Low molecular weight heparin
  - Heparin is the treatment of choice for reducing mortality in PE
    - Initiated prior to a confirmed diagnosis
    - Slows or prevents clot progression and decreases risk of further emboli

- Fibrinolytic therapy
  - Indicated in patients with hypotension (even if resolved), hypoxemia, or evidence of right-ventricular strain
  - Troponin levels can also be used to guide decision-making in patients with sub-massive PE

- Pulmonary embolectomy is a surgical option when fibrinolytic therapy is contraindicated.
PULMONARY EMBOLUS: TREATMENT

- Oxygen is indicated, even in the absence of hypoxemia
- Pulmonary vasodilators to help reduce pulmonary vascular resistance
- Treat right-ventricular failure with fluids and inotropes
- Obstructive Shock

- Warfarin
  - 3 to 6 months if there is identifiable reversible risk factor
  - Minimum of 6 six months if there is no identifiable risk factor
  - Long term with recurrent PE or in patients with ongoing risk factors
- Surgical interruption of inferior vena cava with a filter
  - Patients with contraindication to anticoagulants.
  - Recurrent thromboembolism despite anticoagulant.
  - Survivor of massive PE

ADDITIONAL MIMICS WITH DEEP SYMMETRICAL T WAVE INVERSION

- Adams – Stokes attack
- Hypertrophic cardiomyopathy
- Central nervous system disease
- Post extrasystolic T wave change
ADDITIONAL MIMICS OF ST ELEVATION

- Hyperkalemia
- Intracranial bleed
  - Prolonged QT
  - Prominent U wave

NON MI CAUSES OF Q WAVES

- LVH
- RVH
- Cor Pulmonale
- Cardiomyopathy
- LBBB
- LAHB
- WPW
- Pulmonary Embolism
PAIN MIMICS

PATHOPHYSIOLOGY

- Intimal tear
- False channel
- Risk of rupture: outer wall
- Dissection – hematoma – occlusion of vessels

- Ascending aorta
- Descending aorta
- Abdominal aorta
RISK FOR DISSECTION

- Hypertension is present in most patients with dissections
- Congenital disorders affecting connective tissue such as Marfan Syndrome or Ehlers-Danlos syndrome; also presence of bicuspid aortic valve
- 3rd trimester of pregnancy
- After procedures where aorta or the aortic branches have been cannulated.
CLASSIFICATION OF DISSECTIONS

- Acute or chronic
- **Type A Dissections:** Dissections involving the ascending aorta.
- **Type B Dissections:** Dissections involving the descending thoracic aorta. These dissections begin distal to the left subclavian artery.

COMPLICATIONS OF DISSECTION

- Aortic regurgitation from retrograde dissection involving aortic valve or from aortic dilatation.
- MI from retrograde coronary artery dissection.
- Cardiac tamponade from ascending aorta or aortic arch rupture.
- Intrapleural rupture from descending aortic dissection ruptures into intrapleural space – most commonly left sided.
- Retroperitoneal bleed from rupture of abdominal aorta dissection.
- Stroke from brachial artery compromise.
- Paraplegia, reduced blood flow to kidneys, bowels, and lower extremities from compromise of arterial branches.
CLINICAL PRESENTATION

Chest or back pain with variation in upper extremity blood pressure is key assessment finding in aortic dissection. Recurrent chest or back pain can indicate extension or rupture. The presence of aortic regurgitation in the setting of chest pain is also suspicious for aortic dissection.

DIAGNOSIS

- **Chest Radiograph:** An abnormality of the aorta can usually be seen as a widening of the aorta and mediastinum. Calcium present in the intima is separated from the outer border of the false channel.
- **Transthoracic echocardiography and transesophageal echocardiography:** A visible intimal flap separates the true and false lumens. Echocardiography is most helpful with type A dissections. Transesophageal echocardiography is very valuable and can be performed bedside.
- **CT:** CT is particularly helpful in evaluating dissections of the thoracic aorta.
- **MRI:** MRI is an excellent diagnostic tool but has limitation because it cannot be used emergently or in critically ill patients.
- **Angiography:** Angiography is a definitive diagnostic tool and is required for patients having elective repair of the thoracic aorta.
TREATMENT OF DISSECTIONS

• Stop dissection  
  • Control of hypertension  
  • Lowest possible pressure without compromising perfusion  
  • Velocity of ejection  
  • Rate of rise  

• Beta-blocker in combination with sodium nitroprusside

TREATMENT OF DISSECTIONS

• Type A  
  • Surgery required  
  • Deep hypothermic circulatory arrest  
  • Distal false channel not completely eliminated  
  • Risk for bleeding  
  • Replacement of aortic valve / reimplantation of coronary arteries  
  • Hypertension management  
  • Surgical considerations similar for ascending aortic aneurysms  
  • Surgical mortality 15-20%
TREATMENT OF DISSECTIONS

• Type B
  • Typically treated medically
  • Higher surgical risk candidates
  • Surgical considerations similar to those for descending thoracic aneurysms
  • Surgery limited to complicated dissections:
    • Persistent pain
    • Uncontrolled hypertension
    • Evidence of expansion or rupture
    • Circulatory compromise to visceral, renal, or lower extremity vessels and resultant organ ischemia.
  • 6 to 6.5 cm diameter in chronic dissections
ADDITIONAL ADVANCED ECG SKILLS

Advanced Concepts in:

INJURY AND ISCHEMIA
ST SEGMENT DEVIATION VECTOR

- RCA occlusion: ST deviation vector points to lead III
  - More ST elevation in lead III than lead II
  - ST depression in lead I
- Circumflex occlusion: ST deviation vector points to lead II
  - More ST elevation in lead II than lead III
  - Isoelectric or depressed ST in lead I
ST SEGMENT EVALUATION IN V4R

- ST elevation in V4R
  - Proximal occlusion of RCA prior to marginal branch
  - Significant risk for advanced AV nodal block
- Isoelectric ST in V4R with upright T wave
- Isoelectric or depressed ST in V4R with negative T wave
ADDITIONAL ST / T WAVE CHANGES WITH INFERIOR MI

- Extension into posterior wall
  - ST segment depression in the precordial leads
    - ST deviation vector points to posterior wall

- Extension into lateral wall
  - ST elevation in leads V5, V6 (may also be leads I, and aVL)
    - ST deviation vector points to lateral wall
  - Usually caused by circumflex occlusion

ANTERIOR WALL MI

- Precordial ST elevation
- Location of ST segment deviation vector in limb leads helps predict the location of occlusion within the LAD
ST SEGMENT DEVIATION
VECTOR IN ANTERIOR WALL MI

• Occlusion proximal to first septal and first diagonal branch
  • Primary area of injury is basal part of LV
  • ST segment deviation vector points to base (to lead aVR and aVL) and away from apex (inferior leads)

• Occlusion between first septal and first diagonal branch
  • Dominant area of injury in high anterolateral area of LV
  • ST segment deviation vector points toward aVL and away from lead II

Septal branch perfuses sub AV nodal conduction system

LAD OCCLUSION PROXIMAL TO FIRST SEPTAL AND FIRST DIAGONAL BRANCH
Apart from ST elevation in the precordial leads V₁ to V₆, the frontal ST vector points toward the base of the heart.
• ST elevation in aVR, aVL, and V₁
• ST depression in II, III, aVF, V₅, and V₆
• Acquired right bundle branch block (qR in lead V₁) with or without hemiblock indicates a proximal LAD occlusion.
ST SEGMENT DEVIATION VECTOR IN ANTERIOR WALL MI

- Occlusion distal to the diagonal branch
- ST segment deviation vector points to apex
- ST elevation in inferior leads (II > III)

- Left Main Coronary Artery Occlusion
- ST segment deviation vector points to lead aVR
- ST elevation in aVR
- Less ST elevation in V1
- ST depression in leads V2 – V6 (posterior wall injury)
  - circumflex occlusion
The ECG in Anterior Wall MI

DISTAL LAD OCCLUSION
Apart from ST elevation in V_2 to V_6, a frontal ST deviation vector points in an apical direction.
- ST elevation in the inferior leads with lead II greater than lead III

The ECG in Left Main Occlusion

The combination of acute ischemia in the LAD and CX region results in a frontal ST deviation vector pointing toward aVR and a horizontal ST deviation vector directed away from the posterior wall.
LIMITATIONS OF ST SEGMENT DEVIATION VECTOR ANALYSIS

- Previous MI / preexisting ST / T wave abnormalities
- Multi vessel disease
- Altered ventricular activation
- Dominant or small coronary arteries
- Coronary artery anomalies

ST DEVIATION SCORE

- Score > 12 mm: substantial amount of myocardium to benefit from reperfusion
- ST segment deviation may be better than ST segment deviation score
- Limb leads provide more global information
- Precordial leads provide more local information
RISK STRATIFICATION

- Severity of ischemia
  - Grade 1
    - Peaked symmetrical T waves (hyperacute)
  - Grade 2
    - ST elevation / no distortion of terminal part of QRS
  - Grade 3
    - ST elevation with distortion of terminal part of QRS
    - J Point / R wave ratio > .5

- Grades of ischemia correlate with infarct size, LV function, and mortality (in hospital and late)

ADDITIONAL RISK CONSIDERATIONS

- Significance of 2nd degree or more AV nodal block
- Significance of conduction disturbances in or below bundle of His
  - Existing versus acquired RBBB
  - RBBB with LAHB
## High Risk Features in UA / NSTEMI

- Recurrent angina / ischemia
  - Rest or low level activity with medical treatment
- Troponin +
- New or presumed new ST depression
- S&S HF or worsening mitral regurgitation
- High risk findings on noninvasive testing
  - EF < 35%, large anterior perfusion defect, multiple perfusion defects
- Hemodynamic instability
- Sustained VT
- PCI within 6 months
- Prior CABG
- Reduced LV Function
- High risk TIMI or GRACE Score

## Risk Assessment in UA / NSTEMI

- **TIMI Risk Score**
  - Age > 65
  - 3 or > risk factors for CAD
  - Prior 50% or > stenosis
  - ST deviation on ECG
  - 2 or > anginal events in previous 24 hours
  - Use of ASA in prior 7 days
  - Elevated cardiac biomarkers

- **GRACE**
  - Older age
  - Killip class
  - Systolic BP
  - Cardiac arrest during presentation
  - Serum creatinine
  - Positive initial cardiac markers
  - HR
REPERFUSION ASSESSMENT

• ST segment resolution
• Terminal T wave inversion versus late symmetrical T wave inversion
• AIVR versus sustained VT
  • Specificity for reperfusion

VENTRICULAR ANEURYSM

• Persistent ST elevation after AMI (anterior)
• Anatomic LV aneurysm
  • Myocardial thinning and bulging
• Use of echo in the reperfusion decision
• Risk of fibrinolytics with ventricular aneurysm
  • Embolization of thrombus
ST ELEVATION OF VENTRICULAR ANEURYSM

- Most common in V1-V3
- Usually less than 3 mm elevation
- Relatively unchanged from previous ECGs
- Q waves are deep and well formed
  - QS pattern in V1-V3 or very minimal r
  - QR pattern common in inferior aneurysm / RBBB
MYOCARDIAL RUPTURE

- Incidence
  - 10% MI deaths
- Definition
  - Myocardial leakage – hemipericardium – tamponade
  - Perceived sudden; often slow tear
- Associated Factors
  - Late fibrinolytics
  - Delayed hospital admission
  - Septal involvement = VSD

MYOCARDIAL RUPTURE

- Post-infarction regional pericarditis precedes rupture
- Confirmation of rupture

-T Wave Patterns in Post-infarction Regional Pericarditis

- Persistently positive T waves 48 hours after an MI
- ST segment reelevation
- Premature reversal of T wave inversion to positive
Final Thought:

Mastery is not something that strikes in an instant, like a thunderbolt, but a gathering power that moves steadily through time, like weather.

- John Champlain Gardner Jr. (1933-1982)