12 LEAD ECG INTERPRETATION in
Acute Coronary Syndromes & Sudden Arrhythmia Death Syndromes (ACS & SADS)

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Wayne W Ruppert
Electrophysiology Lab Case Studies

EP Catheters within the heart used for obtaining the Electrogram (the “internal ECG”) Tracing and for Pace-mapping, an integral component of an EP study

Author Wayne Ruppert conducting Pace-mapping during EP study at the St Joseph’s Hospital Heart Institute, Pediatric Electrophysiology Program, Tampa, FL in 2004
Observation Medicine ECG Course

BASIS:

- Current ACC/AHA Guidelines and Recommendations
- Multiple additional recent Evidence-Based Publications
- ECGs from case files of the author, Wayne Ruppert
- Graphic art / images from published textbooks authored by Wayne Ruppert

www.TriGenPress.com
www.ECGtraining.org
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The EKG in PERSPECTIVE

1. Much development in the 1950s and 60s, and at that time, EKGs were the primary diagnostic tool.

2. Today we have better diagnostic tools (e.g. ECHO, CARDIAC CATH, EP STUDIES) that sometimes conflict with traditional EKG-made diagnoses.

3. Some EKG findings are more accurate and reliable than others.

AND...
Sometimes, ECGs LIE to us!

ECGs and USED CAR SALESMEN often have MUCH in common!
The EKG in PERSPECTIVE

PROBLEMS WITH EKGs...

↓ SENSITIVITY
   (FALSE NEGATIVES)

↓ SPECIFICITY
   (FALSE POSITIVES)

AND...

PROBLEMS WITH SPECIFICITY . . .

S-T SEGMENT ELEVATION - COMMON ETIOLOGIES:

<table>
<thead>
<tr>
<th>CONDITION</th>
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<tbody>
<tr>
<td>ACUTE INfarction</td>
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<tr>
<td>HYPERkALEMIA</td>
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<td>BRUGADA SYNDROME</td>
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<td>PULMONARY EMBOLus</td>
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<td>INTRACRANIAL BLEEd</td>
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<td>MYOCARDITis / PERICARDITis</td>
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<td>L. VENT. HYPERTrophy</td>
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<td>PRINZMETAL'S ANGINA</td>
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<tr>
<td>L. BUNDLE BRANCH BLOCK</td>
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<tr>
<td>PACED RHYTHM</td>
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<tr>
<td>EARLY REPOLARIZATION &amp; &quot;MALE PATTERN&quot; S-T ELEV.</td>
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</table>
Patient:
• Asymptomatic
• Troponin normal
• Cardiac Cath angiography = “no obstructive CAD.”
• Discharge diagnosis:

EARLY REPOLARIZATION. This degree of ST Elevation in early repolarization is VERY RARE: The only such ECG I have seen in approximately 13,000 cardiac catheterizations.
EKGs in PERSPECTIVE, con't:
One of the MOST MISLEADING scenarios of all is when the EKG APPEARS PERFECTLY NORMAL . . .

. . .but MASKS serious, LIFE - THREATENING CONDITIONS.
that is why YOU must do a THOROUGH PATIENT EVALUATION . . . and have a HIGH INDEX OF SUSPICION !!!
NORMAL ECG.

But . . . .

PROBLEMS WITH SENSITIVITY . . .

LETHAL TRIPLE VESSEL DISEASE
Despite the ECG’s problematic issues with Lack of Sensitivity & Lack of Specificity, the 12 Lead ECG remains one of our QUICKEST, most cost-efficient front-line Triage Tools that we have today.
- We utilize ACS Risk Stratification to compensate for the ECG’s lack of sensitivity and specificity, to aid us in clinical decision-making and to improve our diagnostic accuracy.
RISK FACTORS for the development of CORONARY ARTERY DISEASE:
- HEREDITY
- ↑ LDL and ↓ HDL CHOLESTEROL PROFILES
- SMOKING
- DIABETES MELLITUS
- OBESITY
- PHYSICAL INACTIVITY
- HYPERTENSION
- AGE - OVER 65
- MALE
- HIGH STRESS

per the AMERICAN HEART ASSOCIATION
Heart Score Reliability

PROBLEMS WITH SENSITIVITY . . .

NORMAL ECG.

But . . . . .

LETHAL TRIPLE VESSEL DISEASE
HEART Score: 5

Outcome:
Successful Emergency Bypass Surgery
2 patients with the above ECG.
- Patient 1 HEART Score of “0”
- Patient 2 HEART Score of “7”

Should they get the same care ??
TYPICAL SYMPTOMS of ACUTE CORONARY SYNDROME:

- **CHEST PAIN** - DESCRIBED AS...
  - "HEAVINESS, PRESSURE, DULL PAIN, TIGHTNESS"
  - CENTERED IN CHEST, SUBSTERNAL
  - MAY RADIATE TO SHOULDERS, JAW, NECK, LEFT or RIGHT ARM
  - NOT EFFECTED by:
    * MOVEMENT
    * POSITION
    * DEEP INSPIRATION

- **SHORTNESS OF BREATH**
  - MAY or MAY NOT BE PRESENT

- **NAUSEA / VOMITING**
  - MAY or MAY NOT BE PRESENT

---

INFARCTION

--- "Classic Symptoms" ---

QUICK ASSESSMENT "SHORT FORM"

- **SUBSTERNAL CHEST PAIN** (HAVE PATIENT POINT TO WORST PAIN)
- **DESCRIBED AS "DULL PAIN," "PRESSURE," or "HEAVINESS"
- **DOES NOT CHANGE WITH DEEP BREATH**
BEWARE of the patient with “INTERMITTENT CHEST PAIN” . . .

ATYPICAL SYMPTOMS of ACS

???
Acute MI patients who present without chest pain are SHREWD:

- Stroke (previous history of)
- Heart failure (previous history of)
- Race (non-white)
- Elderly (age 75+)
- Women
- Diabetes mellitus

*The information listed in the table to the immediate left resulted from a study conducted by John G. Canto, MD, MSPH, et al., of the University of Alabama. The study consisted of 434,877 patients diagnosed with AMI between 1994 and 1998 in 1,674 US hospitals. Study results were published in the Journal of the American Medical Association (JAMA) on June 28, 2000, Vol. 283, No. 24, pages 3223-3229.

Common atypical complaints associated with AMI without chest pain include:

- Malaise (weakness)
- Indigestion
- Nausea
- Dizziness
- Syncope
- Fatigue
- Abdominal pain
- Cold sweats
- Elevated heart rate
- Dyspnea

Effect of Having Multiple Risk Factors for AMI Without Chest Pain

<table>
<thead>
<tr>
<th>% of Patients with Acute MI Presenting to the Emergency Department Without Chest Pain</th>
<th>Number of Risk Factors Present</th>
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<tr>
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OBTAINING THE 12 LEAD ECG

And have it interpreted by a physician or mid-level provider ...within 10 minutes!
Obtaining the 12 Lead ECG

- Limb leads should be on the limbs.
Obtaining the 12 Lead ECG

- Limb leads should be on the limbs.
- When emergency circumstances dictate that limb leads be placed on patient's torso, the words "LIMB LEADS ON PATIENT'S TORSO" should be noted on the ECG.

Obtaining the 12 Lead ECG

Recent AHA/ACC/HRS literature indicates QRS AMPLITUDE, Q WAVE DURATION, AXIS and WAVEFORM DEFLECTION can be altered when limb leads are placed on the patient's torso (Mason-Likar lead placement).

*Therefore every effort should be made to place limb leads on the limbs.*
Recommendations for the Standardization and Interpretation of the Electrocardiogram
Part I: The Electrocardiogram and Its Technology

... affected by monitoring lead placement; however, tracings that use torso electrodes differ in important ways from the standard 12-lead ECG. In addition to body position differences that affect the ECG, monitoring electrodes placed on the trunk do not provide standard limb leads, and distortion of the central terminal alters the augmented limb leads and the precordial leads. Tracings with Mason-Likar and other alternative lead placement may affect QRS morphology more than repolarization compared with the standard ECG; these differences can include false-negative and false-positive infarction criteria. Motion artifact of the limbs is a particular problem for dating scoring in acute infarct...
Leads V1 & V2 on 12 Lead ECG:

• Proper lead placement of precordial Leads V1 and V2 are 4th intercostal space on opposite sides of the sternum.

• Incorrect placement of Leads V1 and V2 will result in: reduction of R wave amplitude (resulting in poor R wave progression) leading to misdiagnosis of previous anterior / septal infarction.
Initial 12 Lead ECG

- Obtain and interpret within 10 minutes of patient presentation

Initial 12 Lead ECG

- Obtain and interpret within 10 minutes of patient presentation
- Interpreted by physician / advanced practitioner
Initial 12 Lead ECG

- Obtain and interpret within 10 minutes of patient presentation
- Interpreted by physician / advanced practitioner
- **Determine presence of STEMI and/or other imminent life-threatening condition**

Initial 12 Lead ECG

- Obtain and interpret within 10 minutes of patient presentation
- Interpreted by physician / advanced practitioner
- Determines presence of STEMI and/or other imminent life-threatening condition
- **Should be compared to any previously recorded ECGs in the patient’s medical records**
Initial 12 Lead ECG, continued:

• Additional Serial ECGs should be compared to the BASELINE ECG for determining the presence of Dynamic J Point, ST-Segment and T Wave Changes

Initial 12 Lead ECG, continued:

• Additional Serial ECGs should be compared to the BASELINE ECG for determining the presence of Dynamic J Point, ST-Segment and T Wave Changes

• Serves as “footprint” for determining ECG lead(s) to be used during Continuous ECG Monitoring
  – Ischemia
  – QT interval
Normal ECG – No ACS Indicators

Defining NORMAL – QRS <120ms:
HEART Score criteria is J Point should be less than 0.5mm
THE S-T SEGMENT

SHOULD HAVE A "SLIGHT POSITIVE" INCLINATION

THE S-T SEGMENT

SHOULD BE "CONCAVE" IN SHAPE . . .
Evaluating the ECG for ACS:
A TWO-STEP process:

THE S-T SEGMENT

AS OPPOSED TO "CONVEX" IN SHAPE

SHOULD BE "CONCAVE" IN SHAPE ...
Evaluating the ECG for ACS:
A TWO-STEP process:

STEP 1: Evaluate QRS Width

STEP 2: Evaluate J Points, ST-Segment and T waves in EVERY Lead
STEP 1 – evaluate QRS width:

• QRS is ABNORMALLY WIDE (>120 ms),
  – indicates DEPOLARIZATION ABNORMALITY (e.g. “bundle branch block, Wolff-Parkinson-White Syndrome, etc).

STEP 1 – evaluate QRS width:

• QRS is ABNORMALLY WIDE (>120 ms),
  – indicates DEPOLARIZATION ABNORMALITY (e.g. “bundle branch block, Wolff-Parkinson-White Syndrome, etc).
  – DEPOLARIZATION ABNORMALITIES in turn cause REPOLARIZATION ABNORMALITIES, which alters the: \( J \) Points, \( ST \)-Segments and/or \( T \) Waves.
Evaluating the ECG for ACS:
Wide QRS present: QRSd > 120ms

• Determine RIGHT vs. LEFT Bundle Branch Block Pattern

Simple “Turn Signal Method” . . .
“Terminal Phase of QRS Method”...

**Diagnosing Bundle Branch Block**

- **L.B.B.B.**
  - Using Lead V1
  - QRS WIDER THAN 120 ms
  - BEAT IS SUPRAVENTRICULAR IN ORIGIN
  - TERMINAL PHASE OF QRS COMPLEX (LAST DEFLECTION)

- **R.B.B.B.**
  - NEGATIVE = LEFT BUNDLE BRANCH BLOCK
  - POSITIVE = RIGHT BUNDLE BRANCH BLOCK

**Diagnosing LBBB in Lead V1:**

- QRS GREATER THAN 120 ms (.12)
- EVIDENCE THAT THIS IS NOT VENTRICULAR BEAT
- TERMINAL PHASE (LAST PART) OF QRS COMPLEX IS NEGATIVE DEFLECTION
- S-T SECTIONS ARE NORMALLY ALWAYS ELEVATED!
DIAGNOSING RBBB IN LEAD V1:

- WIDER THAN 120 ms (.12)
  (or 3 little boxes)
- TERMINAL PHASE
  (LAST PART) OF
  QRS COMPLEX
  IS POSITIVE
  DEFLECTION

DIAGNOSING BUNDLE BRANCH BLOCK

USING LEADS V1, V2, and V5, V6:

LOCATING RsR' or RR' COMPLEXES:

<table>
<thead>
<tr>
<th>V1</th>
<th>V2</th>
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<tr>
<td>RIGHT BUNDLE BRANCH BLOCK</td>
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<table>
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<tr>
<th>V5</th>
<th>V6</th>
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<tbody>
<tr>
<td>LEFT BUNDLE BRANCH BLOCK</td>
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</table>
Terminal phase of QRS is positive

= Right bundle branch block
TERMINAL PHASE OF QRS IS NEGATIVE

= LEFT BUNDLE BRANCH BLOCK
Wide QRS present: (QRSd > 120ms)

• When RIGHT Bundle Branch Block pattern is present:
  – Precordial Leads typically demonstrate ST Depression and T wave Inversion
Wide QRS present: (QRSd > 120ms)

• When RIGHT Bundle Branch Block pattern is present:
  – Precordial Leads typically demonstrate ST Depression and T wave Inversion
  – DOES NOT MASK STEMI; when ST Elevation is noted, CONSIDER STEMI !!
Wide QRS present: (QRSd > 120ms)

• When LBBB QRS pattern is present:
  – ST-Segment Elevation is typically noted in Precordial Leads
Wide QRS present:  
(QRSd > 120ms)

- When LBBB QRS pattern is present:  
  - ST-Segment Elevation is typically noted in Precordial Leads  
  - *Can cause up to 5mm of J Point Elevation in normally calibrated ECG (1mm=10mv)*
Diagnosis of STEMI with LBBB pattern:

2013 ACC/AHA Guideline for Management of STEMI

- ST Elevation of 0.1mv (1mm) or more in leads with Positive Deflection QRS complexes

- ST Elevation of 0.5mv (5mm) or more in leads with Negative Deflection QRS complexes
Diagnosis of STEMI with LBBB pattern:

2013 ACC/AHA Guideline for Management of STEMI

- ST Elevation of 0.1mv (1mm) or more in leads with Positive Deflection QRS complexes
- ST Elevation of 0.5mv (5mm) or more in leads with Negative Deflection QRS complexes
- ST Segment Changes as compared with those of older ECGs with LBBB

• Convex ST Segment
A.H.A. ACLS GUIDELINES

1. If patient has a CONFIRMED HISTORY of LBBB, rely on:
   - CARDIAC MARKERS
   - SYMPTOMS
   - RISK FACTOR PROFILE
   - HIGH INDEX OF SUSPICION
   for diagnosis of STEMI

2. If patient has:
   a) previously NORMAL ECGs (no LBBB)  
      -- or --
   b) no old ECGs available for comparison
   consider diagnosis as STEMI until proven otherwise.
LBBB with CHEST PAIN - CASE 1: PRESENTING EKG

DIAGNOSIS: STEMI - INFERIOR-POSTERIOR WALL
CATH LAB FINDINGS: TOTAL OCCLUSION DISTAL RCA (PDA / PLV)

OLD ECG  NEW ECG
V3
2 mm ST elev.  1.5 mm ST depr.
2 + 1.5 = 3.5mm CHANGE

EKG RECORDED 7 MONTHS AGO
Evaluating the ECG for ACS:

Step 1 - Evaluate Width of QRS:

- Normal (≤ 120 ms)
- Wide (> 120 ms)

Determine QRS Morphology

Evaluate for ST Depression
Evaluate for ST Elevation
Do Not Rely on ST Depression

Use Caution - ST Elevation is routinely seen in wide QRS complexes. Rhythms with LBBB pattern. Follow AHA criteria (page 198) for diagnosis of STEMI in presence of LBBB.
Evaluating the ECG for ACS:

*Patients with Normal Width QRS (QRSd < 120ms)*

Defining NORMAL – QRS <120ms:
When QRS duration is NORMAL (< 120 ms):

**NORMAL ST - T WAVES**

- WHEN QRS WIDTH IS NORMAL (< 120 ms)

**ASSESS:**
- J POINT: ISOELECTRIC (or < 1 mm dev.)
- ST SEG: SLIGHT, POSITIVE INCLINATION
- T WAVE: UPRIGHT, POSITIVE

*in EVERY LEAD EXCEPT aVR !!*

**THE J POINT SHOULD BE . . .**

WITHIN
1 mm
ABOVE
OR
BELOW
the
ISOELECTRIC LINE
...the “flat line” between ECG complexes, when there is no detectable electrical activity...
Use the P-Q junction as a reference point for measuring the J Point and ST-Segment when "iso-electric line is not iso-electric!"
Defining NORMAL:

THE J POINT SHOULD BE:

- WITHIN 1 mm ABOVE
- OR
- BELOW THE P-Q JUNCTION

THE S-T SEGMENT

SHOULD HAVE A "SLIGHT POSITIVE" INCLINATION
THE S-T SEGMENT

SHOULD BE "CONCAVE" IN SHAPE . . .

THE S-T SEGMENT

AS OPPOSED TO "CONVEX" IN SHAPE

SHOULD BE "CONCAVE" IN SHAPE . . .
**THE T WAVE**

- SHOULD BE A "NICE," ROUNDED, CONVEX SHAPE
- SHOULD BE SYMMETRICAL

**THE T WAVE**

- SHOULD BE A "NICE," ROUNDED, CONVEX SHAPE
- SHOULD BE SYMMETRICAL
- SHOULD BE UPRIGHT IN ALL LEADS, EXCEPT AVR
THE T WAVE

LEAD AVR

- REMEMBER, IN LEAD AVR EVERYTHING IS "UPSIDE-DOWN"

Normal Variants: T Wave Inversion

Leads where the T WAVE may be INVERTED:
When QRS duration is NORMAL (<120 ms):

**THE T WAVE**

**AMPLITUDE GUIDELINES:**

- IN THE LIMB LEADS, SHOULD BE LESS THAN 1.0 mv (10 mm)
- IN THE PRECORDIAL LEADS, SHOULD BE LESS THAN 0.5 mv (5 mm)
- SHOULD NOT BE TALLER THAN R WAVE IN 2 OR MORE LEADS.

**NORMAL ST - T WAVES**

- J POINT: ISOELECTRIC (or <1 mm dev.)
- ST SEG: SLIGHT, POSITIVE INCLINATION
- T WAVE: UPRIGHT, POSITIVE

*in EVERY LEAD EXCEPT aVR !!*
ECG Indicators of ACS in Patients with Normal Width QRS Complexes (QRS duration < 120 ms)

Multiple patterns of ABNORMAL:
- J Point
- ST-Segment
- T Wave

configurations may indicate ACS.

Remember, “IF IT’S NOT NORMAL, it’s ABNORMAL!”

BOOK PAGE: 83
<table>
<thead>
<tr>
<th>J POINT, ST SEGMENT, and T WAVE ABNORMALITIES</th>
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- ACUTE MI (EARLY PHASE)                        - ACUTE MI (EARLY PHASE)
- ACUTE MI (EARLY PHASE)                        - ACUTE MI (EARLY PHASE)
- ACUTE (NON-Q WAVE) MI                         - ACUTE (NON-Q WAVE) MI
- ACUTE MI (RECI PROCAL CHANGES)                - ACUTE MI (RECI PROCAL CHANGES)
- ISCHEMIA                                      - ISCHEMIA
ECG Patterns associated with “EARLY PHASE MI:”

- J-T Apex abnormalities
- Hyper-Acute T Waves
- ST-T Wave Changes
WHEN EVALUATING for ST SEGMENT ELEVATION ............

From:
AMERICAN HEART ASSOCIATION
ACLS 2005 REVISIONS

PATTERNS of EARLY INFARCTION
-- FLAT and CONVEX J-T APEX SEGMENTS
LEAD II

41 y/o FEMALE
In ER C/O CHEST PAIN
x 30 minutes.
* FLAT J-T APEX SEGMENT
* NO ST ELEVATION at
  J POINT

1839 hrs

STEMI - INFERIOR WALL
11 MINUTES LATER, S-T
ELEVATION at the J POINT
IS NOTED
* CATH LAB FINDINGS:
  TOTAL OCCLUSION of the
  RIGHT CORONARY ARTERY
CASE STUDY: ABNORMAL J-T Apex Segments

CHIEF COMPLAINT and SIGNIFICANT HISTORY:

56 y/o MALE presents to ED with complaint of "INTERMITTENT SUBSTERNAL & SUB-EPIGASTRIC PRESSURE" x 3 HOURS. PMHx of ESOPHAGEAL REFUX. NO other significant past medical history.

RISK FACTOR PROFILE:

● FAMILY HISTORY - father died of MI at age 62
● PREVIOUS CIGARETTE SMOKER - quit 15 years ago.
● CHOLESTEROL - DOES NOT KNOW; "never had it checked."
● OBESITY

PHYSICAL EXAM: Patient supine on exam table, mildly anxious, currently complaining of "mild indigestion," skin is warm, pale, dry; REST OF EXAM is UNREMARKABLE.

VITAL SIGNS: BP 142/94, P 80, R 20, SAO2 98%

LABS: JUST OBTAINED, RESULTS NOT AVAILABLE YET.

ECG COMPUTER DOES NOT NOTICE THE CONVEX J-T APEX SEGMENTS!
S-T elevation at J point = 0.5 mm

ACUTE MI = S-T elev. > 1.0 mm

measurement of S-T elevation

measurement of S-T elevation by "J point + .04" method

S-T elevation at J point = 0.5 mm

S-T elevation at J + .04 = 2.0 mm

ACUTE MI = S-T elev. > 1.0 mm
CASE STUDY: 56 y/o male with INTERMITTENT "CHEST HEAVINESS"

TREATMENT PLAN: EMERGENCY CORONARY ARTERY BYPASS SURGERY (4 VESSEL)

L.A.B. SUBTOTAL PROXIMAL OCCLUSION WITH THROMBUS
RAMUS ARTERY w/ SUBTOTAL OCCLUSION
G.M.T w/ SUBTOTAL OCCLUSION

PATTERNS of ACS & ISCHEMIA

- J POINT, ST SEGMENT, and T WAVE ABNORMALITIES -

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<td>ACUTE MI - (RECIPIROCAL CHANGES)</td>
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<td>ISCHEMIA</td>
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9/3/2018
T waves should not be HYPERACUTE

HYPERACUTE T Waves may indicate:

• Early phase Acute MI
• Transmural ischemia (usually seen in one region of the ECG)
• Hyperkalemia (seen globally across ECG)
• Hypertrophy
Helpful Clue: Hyper-Acute T Waves

- GLOBAL Hyper-acute T Waves (in leads viewing multiple myocardial regions / arterial distributions) favors HYPERKALEMIA
Helpful Clue: Hyper-Acute T Waves

- **GLOBAL** Hyper-acute T Waves (in leads viewing multiple myocardial regions / arterial distributions) favors HYPERKALEMIA

- **Hyper-acute T Wave noted in ONE ARTERIAL DISTRIBUTION** (Anterior / Lateral / Inferior) favors TRANSMURAL ISCHEMIA / Early Phase Acute MI
CASE STUDY: HYPERACUTE T WAVES

CHIEF COMPLAINT and SIGNIFICANT HISTORY:
30 y/o male presents to ER via EMS, c/o sudden onset of dull chest pain x 40 min. Pain level varies, not effected by position, movement or deep inspiration. No associated symptoms.

RISK FACTOR PROFILE: NONE. CHOLESTEROL UNKNOWN.

PHYSICAL EXAM: Patient is supine on exam table, CAO x 4, anxious, restless, skin pale, cool, dry. Patient c/o chest pressure, "7" on 1 - 10 scale, uneffected by position, movement, deep inspiration. Lungs clear. HS: NL S1, S2, no rubs, murmurs, gallops

VITAL SIGNS: BP 138/88  P 90  R 20  SAO2 98%

DIAGNOSTIC TESTING: 1st TROPONIN I - ultra: <0.07
Cath Lab findings:
Dynamic ST-T Wave Changes:

- Other than HEART RATE related variations (which affect intervals), J Points, ST-Segments and T Waves SHOULD NOT CHANGE.

- When changes to J Points, ST-Segments and/or T waves are NOTED, consider EVOLVING MYOCARDIAL ISCHEMIA and/or EARLY PHASE MI, until proven otherwise.
46 year old male

• Exertional dyspnea X “several weeks”
• Intermittent chest pressure X last 3 hours. Currently pain free.

46 year old male: ECG 1

• Chest pressure has returned, “5” on 1-10 scale. 2nd ECG obtained due to “change in symptoms”: 
ECG #1
- Increased T wave in Lead II
- New Q waves in Leads II, III, and aVF

ECG #2
- Increased T wave in Lead II
- New Q waves in Leads II, III, and aVF

7:59 am
- Increase of 2.5 mm in lead V1

8:08 am
- Increase of 3.0 mm in lead V2
- Increase of 4.0 mm in lead V3
ST-Segment Depression

7:59 am  8:08 am

Cath Lab Angiography:

Proximal subtotally occluded Left Anterior Descending Artery (LAD)
ISCHEMIA

*BI-PHASIC T WAVE*

- SUB-TOTAL OCCLUSION of LEFT ANTERIOR DESCENDING ARTERY (when noted in V1-V4)
- LEFT VENTRICULAR HYPERTROPHY
- COCAINE INDUCED VASOSPASM

BI-PHASIC T WAVES

58 y/o MALE WITH SUB-TOTAL OCCLUSIONS OF THE LEFT ANTERIOR DESCENDING ARTERY
Classic "Wellen’s Syndrome:"

- Characteristic T wave changes
  - Biphasic T waves
  - Inverted T waves
- History of anginal chest pain
- Normal or minimally elevated cardiac markers
- ECG without Q waves, without significant ST-segment elevation, and with normal precordial R-wave progression
Wellen’s Syndrome ETIOLOGY:
• Critical Lesion, Proximal LAD
• Coronary Artery Vasospasm
• Cocaine use (vasospasm)
• Increased myocardial oxygen demand
• Generalized Hypoxia / anemia / low H&H

Wellen’s Syndrome EPIDEMIOLOGY & PROGNOSIS:
• Present in 14-18% of patients admitted with unstable angina
• 75% patients not treated developed extensive Anterior MI within 3 weeks.
• Median Average time from presentation to Acute Myocardial Infarction – 8 days

Sources: H Wellens et. Al, Am Heart J 1982; v103(4) 730-736
Wellen’s Syndrome Case Study

- 33 y/o male
- Chief complaint “sharp, pleuritic quality chest pain, intermittent, recent history lower respiratory infection with productive cough.”
- ED physician attributed the ST elevation in precordial leads to “early repolarization,” due to patient age, gender, race (African American) and concave nature of ST-segments.
Wellen’s Syndrome Case Study

**DYNAMIC ST-T Wave Changes ARE PRESENT !!**

**NOW**

is the time for the **STAT CALL**

to the **CARDIOLOGIST !!!**
Wellen’s Syndrome Case Study

Wellen’s Syndrome Case Study
Wellen’s Syndrome Case Study

SUB-TOTAL OCCLUSION OF LEFT ANTERIOR DESCENDING ARTERY

STENT DEPLOYMENT, LEFT ANTERIOR DESCENDING ARTERY, 33 y/o male

SUB-TOTAL OCCLUSION OF LEFT ANTERIOR DESCENDING ARTERY

POST PCI - LAD
Additional Resources:

- Wellen’s Syndrome, NEJM case study

Recommendations
1. For men 40 years of age and older, the threshold value for abnormal J-point elevation should be 0.2 mV (2 mm) in leads V2 and V3, and 0.1 mV (1 mm) in all other leads.
2. For men less than 40 years of age, the threshold values for abnormal J-point elevation in leads V2 and V3 should be 0.25 mV (2.5 mm).
3. For women, the threshold value for abnormal J-point elevation should be 0.15 mV (1.5 mm) in leads V2 and V3, and greater than 0.1 mV (1 mm) in all other leads.
4. For men and women, the threshold for abnormal J-point elevation in V1, V2, and V3 should be 0.05 mV (0.5 mm), except for males less than 30 years of age, for whom 0.1 mV (1 mm) is more appropriate.
5. For men and women, the threshold value for abnormal J-point elevation in V1 through V4 should be 0.05 mV (0.5 mm).
6. For men and women of all ages, the threshold value for abnormal J-point depression should be −0.05 mV (−0.5 mm) in leads V2 and V3, and −0.1 mV (−1 mm) in all other leads.
ST SEGMENT ELEVATION:

S-T SEGMENTS ELEVATE WITHIN SECONDS OF CORONARY ARTERY OCCLUSION:

IN THIS CASE, a normal response to balloon occlusion of the RIGHT CORONARY ARTERY during PTCA in the CARDIAC CATH LAB

3 COMMON PATTERNS of ST SEGMENT ELEVATION From ACUTE MI:
Reciprocal S-T Segment Depression *may or may not* be present during STEMI.

The presence of S-T Depression on an EKG which exhibits significant S-T elevation is a fairly reliable indicator that STEMI is the diagnosis.
Reciprocal S-T Segment Depression *may or may not* be present during STEMI.

The presence of S-T Depression on an EKG which exhibits significant S-T elevation is a fairly reliable indicator that STEMI is the diagnosis.

However the lack of *Reciprocal S-T Depression* DOES NOT rule out STEMI.
STEMI

- Correlation of ECG Leads with Coronary Arterial Anatomy and the STRUCTURES SERVED by the OCCLUDED ARTERY... will serve as a “crystal ball,” allowing you to ANTICIPATE complications of STEMI...
STEMI

- Correlation of ECG Leads with Coronary Arterial Anatomy and the STRUCTURES SERVED by the OCCLUDED ARTERY . . . .
  . . . . Will serve as a “crystal ball,” allowing you to ANTICIPATE complications of STEMI . . . .
  . . . . . BEFORE they occur!!

"Having knowledge of common coronary artery anatomy is the . . . .

to understanding the PHYSIOLOGICAL CHANGES that occur during ACUTE MI."

“an INVALUABLE ASSET for ALL MEDICAL PROFESSIONALS who
provide direct care to STEMI patients!”
INTERPRET THE EKG, THEN:

- IDENTIFY THE AREA OF THE HEART WITH A PROBLEM . . .
- RECALL THE ARTERY WHICH SERVES THAT REGION . . .
- RECALL OTHER STRUCTURES SERVED BY THAT ARTERY . . .
- ANTICIPATE FAILURE OF THOSE STRUCTURES . . .
- **INTERVENE APPROPRIATELY!**

3 STEMI Case Studies, excerpts from “12 Lead ECG Interpretation in ACS with Case Studies from the Cardiac Cath Lab.”
CASE STUDY 1 - STEMI

CHIEF COMPLAINT and SIGNIFICANT HISTORY:
72 y/o male, c/o CHEST "HEAVINESS," started 20 minutes before calling 911. Pain is "8" on 1-10 scale, also c/o mild shortness of breath. Has had same pain "intermittently" x 2 weeks.

RISK FACTOR PROFILE:
- FAMILY HISTORY - father died of MI at age 77
- FORMER CIGARETTE SMOKER - smoked for 30 year - quit 27 years ago
- DIABETES - oral meds and diet controlled
- HIGH CHOLESTEROL - controlled with STATIN meds
- AGE: OVER 65

PHYSICAL EXAM: Patient calm, alert, oriented X 4, skin cool, dry, pale.
No JVD. Lungs clear bilaterally. Heart sounds normal S1, S2. No peripheral edema.

VITAL SIGNS: BP: 100/64, P: 75, R: 20, SAO2: 94%

LABS: FIRST TROPONIN: 6.4
Note: There is NO Reciprocal ST Depression on this STEMI ECG!
OCCLUSION of MID - LEFT ANTERIOR DESCENDING ARTERY

LEFT MAIN CORONARY ARTERY
AV NODE
LBB
CIRUMFLEX ARTERY
LEFT ANTERIOR DESCENDING ARTERY
AREA OF INFARCT

ANTERIOR VIEW

LAD DISTRIBUTION
35 - 45 % of LV MUSCLE MASS

A BLOCKAGE OF THE LAD CAN RESULT IN LV PUMP FAILURE --
CARDIOGENIC SHOCK
PULMONARY EDEMA

LV FUNCTION
LEFT ANTERIOR DESCENDING ARTERY (LAD)

- ANTERIOR WALL OF LEFT VENTRICLE

⚠️ 35 - 45% OF LEFT VENTRICLE MUSCLE MASS

- SEPTUM, ANTERIOR 2/3

⚠️ BUNDLE BRANCHES

- ANTERIOR-MEDIAL PAPILLARY MUSCLE
# Anticipated Complications of Anterior-Septal Wall STEMI & Possible Indicated Interventions

<table>
<thead>
<tr>
<th>Complication</th>
<th>Interventions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac Arrest</td>
<td>BCLS / ACLS</td>
</tr>
<tr>
<td>Cardiac Dysrhythmias (VT / VF)</td>
<td>ACLS (antiarrhythmics)</td>
</tr>
<tr>
<td>Pump Failure with Cardiogenic Shock</td>
<td><strong>Inotrope Therapy:</strong>&lt;br&gt;- Dopamine / Dobutamine / Levophed&lt;br&gt;- Intra-Aortic Balloon Pump (use caution with fluid challenges due to Pulmonary Edema)</td>
</tr>
<tr>
<td>Pulmonary Edema</td>
<td>CPAP&lt;br&gt;- ET intubation (use caution with diuretics due to pump failure and hypotension)</td>
</tr>
<tr>
<td>3rd Degree Heart Block - Not Responsive to Atropine</td>
<td>Transcutaneous or Transvenous Pacing</td>
</tr>
</tbody>
</table>
CASE STUDY 2: STEMI

CHIEF COMPLAINT and SIGNIFICANT HISTORY:
46 y/o Female walks into ED TRIAGE, with chief complaint of EPIGASTRIC PAIN, NAUSEA and WEAKNESS. Symptoms have been intermittent for last two days. She was awakened early this morning with the above symptoms, which are now PERSISTENT.

RISK FACTOR PROFILE:
6° FAMILY HISTORY - father died of CAD, older brother had CABG, age 39
6° DIABETES - diet controlled
6° HYPERTENSION

PHYSICAL EXAM: Pt. CAOx4, anxious, SKIN cold, clammy, diaphoretic. No JVD.
Lungs: clear, bilaterally. Heart Sounds: Normal S1, S2.

VITAL SIGNS: BP: 168/98, P: 110, R: 24, SAO2: 97% on O2 4 LPM via nasal canula

LABS: TROPONIN ultra = 2.8
CASE PROGRESSION: As the patient was being prepared for transport to the Cardiac Cath Lab, she experienced an episode of Ventricular Fibrillation.
CASE STUDY 3: STEMI

CHIEF COMPLAINT and SIGNIFICANT HISTORY:
29 y/o male presents to the ER c/o “HEAVY CHEST PRESSURE” x 30 minutes. The patient states he was playing football with friends after eating a large meal. Pt. also c/o nausea. Denies DIB.

RISK FACTOR PROFILE:
- FAMILY HISTORY - father died of MI age 46
- CURRENT CIGARETTE SMOKER
- "MILD" HYPERTENSION - untreated
- CHOLESTEROL - unknown - “never had it checked.”

PHYSICAL EXAM: Patient alert, oriented X 4, skin cool, dry, pale. Patient restless. No JVD. Lungs clear bilaterally. Heart sounds normal S1, S2. No peripheral edema.

VITAL SIGNS: BP: 104/78, P: 76, R: 20, SAO2: 96%

LABS: INITIAL CARDIAC MARKERS - NEGATIVE
- Reciprocal ST Depression is NOW PRESENT
- Additional ST Elevation is present in Leads I, AVL
\[\text{Occlusion of Diagonal Artery} + \text{Occlusion of Mid-Left Anterior Descending Artery} = \text{Occlusion of Proximal Left Anterior Descending Artery}\]
ANTICIPATED COMPLICATIONS of ANTERIOR-SEPTAL WALL STEMI 
& POSSIBLE INDICATED INTERVENTIONS:

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WHILE AWAITING THE CATH TEAM, THE PATIENT BEGAN VOMITING. SKIN BECAME ASHEN & DIAPHORETIC. REPEAT BP = 50/30. 
- WHAT THERAPEUTIC INTERVENTIONS SHOULD BE IMPLEMENTED AT THIS POINT?
CASE STUDY 4: CRITICAL DECISIONS SCENARIO

As per current AHA recommendations, your hospital’s policy is to send every STEMI patient to the Cardiac Catheterization Lab for emergency PCI.

You are the ranking medical officer on duty in the ED when two acute STEMI patients arrive, ten minutes apart. The Cath Lab has one lab open, and can take ONE patient immediately. Both patients duration of symptoms and state of hemodynamic stability are similar.

WHO SHOULD GO TO THE CATH LAB FIRST?

And . . .

WHAT WOULD YOU DO WITH THE PATIENT WHO DID NOT GO TO THE CATH LAB?
ECG Clues... for identifying x-ray causes by LEFT MAIN CORONARY ARTERY occlusion:

1. ST ELEVATION in anterior leads (V1-V3) and lateral leads (V5 & V6).
2. ST DEPRESSION or INELECTRIC P POINTS may be seen in some leads, typically V2 and/or V3.

**Note:** It is very unusual to see ST DEPRESSION in V1 during left main.

3. ANTEGRADE WALL motion caused by included LAD.

4. ST ELEVATION in AVR is GREATER THAN 0.1 mm.

5. ST ELEVATION in leads I and aVL. Caused by NOFLOW vs. DIAGONAL (OBSTRIE MARGINAL BRANCHES).**

6. ST ELEVATION in leads II, III, and aVF. In cases of LMCA occlusion of dominant coronary, leads II, III, and aVF may show ST ELEVATION or INELECTRIC P POINTS.***

**Note:** Presence of new nails or$ LEFT ANTERIOR DESCULAR BLOCK.**

---

Lead AVR Views the BASILAR SEPTUM (region of the Bundle of His):
In STEMI with ST-Segment Elevation in Lead AVR, This is indicative of Left Main Coronary Artery Occlusion . . .
Despite the dismal mortality rate associated with STEMI from total LMCA occlusion, this patient survived and was later discharged. His EF is estimated at approximately 30%. He received an ICD, and is currently stable.
**CASE STUDY 4: CRITICAL DECISIONS SCENARIO**

**CONCLUSIONS:**

**QUESTION 1: WHICH PATIENT SHOULD BE TAKEN FIRST FOR IMMEDIATE CARDIAC CATHETERIZATION for EMERGENCY PCI ?**

**ANSWER:** PATIENT B was taken emergently to the Cardiac Cath Lab - both the ED physician and the Interventional Cardiologist correctly identified the EKG patterns of LMCA occlusion.

**QUESTION 2: WHAT COURSE OF ACTION SHOULD BE TAKEN WITH THE PATIENT NOT CHOSEN TO BE SENT TO THE CATH LAB FIRST?**

**ANSWER:** PATIENT A received thrombolytic therapy in the ED. It was determined that THROMBOLYTIC THERAPY would achieve the FASTEST ROUTE to REPERFUSION -- by at least 60 minutes.

---

**ECG Clues... for IDENTIFYING STEMI CAUSED BY LEFT MAIN CORONARY ARTERY occlusion:**

- **ST ELEVATION in ANTERIOR LEADS (V1 - V4) and LATERAL LEADS (V5 & V6)**
- **ST DEPRESSION or ISOELECTRIC J POINTS may be seen in V LEADS, mainly V2 and/or V3 caused by COMPETING FORCES of ANTERIOR vs. POSTERIOR WALL MI**
  - **NOTE:** it is very unusual to see ST DEPRESSION in V LEADS with isolated ANTERIOR WALL MI when caused by occluded LAD.
- **ST ELEVATION in AVR is GREATER THAN ST ELEVATION in V1**
- **ST ELEVATION in AVR GREATER THAN 0.5 mm**
- **ST ELEVATION in LEAD I and AVL (caused by NO FLOW to DIAGONAL / OBSTUSE MARGINAL BRANCHES)**
- **ST DEPRESSION in LEADS II, III, and AVL. (in cases of LMCA occlusion of DOMINANT CIRCUMFLEX, leads II, III, and AVL may show ST ELEVATION or ISOELECTRIC J POINTS)**
- **NEW / PRESUMABLY NEW RBHE, and/or LEFT ANTERIOR FASCULAR BLOCK**

---

Yamaji et al, JACC vol 38, No 5, 2001: 1348-54

In patients without STEMI, ST Elevation in AVR, when seen with global indications of ischemia (ST Depression in 8 leads or more), is indicative of advanced multi-vessel disease or significant Left Main Coronary Artery stenosis.

“In patients with:
- Angina at rest
- ST Elevation in AVR and ST Depression in 8 or more ECG leads (global ischemia), it is reported with a 75% predictive accuracy of 3-vessel or left main coronary artery stenosis” . . .

- Wagner et al, 2009 ACC/AHA Standardization and Interpretation of the ECG, Part VI, ACS.
Critical Triple Vessel Disease = *STAT* Coronary Artery Bypass Surgery

ANTICIPATED COMPLICATIONS of GLOBAL ISCHEMIA with POSSIBLE NSTEMI -- INTERVENTIONS to be CONSIDERED:

**Patients with CHEST PAIN at REST and this ECG presentation have a 75% incidence of severe LMCA STENOSIS and/or TRIPLE - VESSEL DISEASE -- in such cases Coronary Artery Bypass Surgery (CABG) is frequently indicated.**

**PREHOSPITAL:** if patient has no hospital preference consider transport to Chest Pain Center WITH Open Heart Surgery capabilities IF nearby.

**HOSPITAL:** consider use of SHORT-ACTING intravenous GP IIb/IIIa receptor agonists

- ACTIVE CHEST PAIN
- ISCHEMIA - CONSIDER DYSRHYTHMIAS
- INCREASED PROBABILITY of IMMINENT MYOCARDIAL INFARCTION

1. AGGRESSIVE SERIAL TROPONIN and SERIAL ECG PROTOCOLS (2014 AHA /ACC / NSTEMI-ACS Guidelines)
2. Positive TROPONIN: consider STAT / early Cardiac Catheterization

Excerpt from *STEMI Assistant*
CASE STUDY 7 - STEMI

CHIEF COMPLAINT and SIGNIFICANT HISTORY:
46 yr. old MALE arrives in ER, C/O SUDDEN ONSET OF CHEST PRESSURE 45 MINUTES AGO. PAIN IS CONSTANT, PRESSURE-LIKE, AND NOT EFFECTED BY POSITION, MOVEMENT or DEEP INSPIRATION. ALSO C/O D.I.B.

RISK FACTOR PROFILE:
6 CURRENT CIGARETTE SMOKER x 18 YEARS
6 HYPERTENSION
6 HIGH LDL CHOLESTEROL

PHYSICAL EXAM: Patient is alert & oriented x 4, skin warm, dry, color normal. Non-anxious
Lungs clear, normal S1, S2. No JVD, No ankle edema.

VITAL SIGNS: BP: 136/88  P: 88  R: 20  SAO2: 100% on 4 LPM O2

LABS: TROPOIN: < .04
RIGHT DOMINANT

75 - 80% POPULATION

POSTERIOR VIEW

RIGHT CORONARY ARTERY (RCA)

- RIGHT ATRIUM
- SINUS NODE (55% of the population)
- RIGHT VENTRICLE - 100% of muscle mass
- LEFT VENTRICLE: 15 - 25% of muscle mass
  - INFERIOR WALL
  - approx. 1/2 of POSTERIOR WALL
- AV NODE
A standard 12 LEAD EKG Does NOT show the RIGHT VENTRICLE
To see the RIGHT VENTRICLE...

...such as in cases of INFERIOR WALL M.I.

☞ You must do a RIGHT - SIDED EKG!!
ANTICIPATED COMPLICATIONS of INFERIOR WALL STEMI secondary to RCA Occlusion & POSSIBLE INDICATED INTERVENTIONS:

- CARDIAC ARREST  BCLS / ACLS
- CARDIAC DYSRHYTHMIAS (VT / VF)  ACLS (antiarrhythmics)
- SINUS BRADYCARDIA  ATROPINE 0.5mg, REPEAT as needed UP TO 3mg. (follow ACLS and/or UNIT protocols)
- HEART BLOCKS (1st, 2nd & 3rd Degree HB)  ATROPINE 0.5mg, REPEAT as needed UP TO 3mg, Transcutaneous Pacing, (follow ACLS and/or UNIT protocols)
- RIGHT VENTRICULAR MYOCARDIAL INFARCTION
  - The standard 12 Lead ECG does NOT view the Right Ventricle.
  - You must do a RIGHT-SIDED ECG to see if RV MI is present.
  - Do NOT give any Inferior Wall STEMI patient NITRATES or DIURETICS until RV MI has been RULED OUT.

- POSTERIOR WALL INFARCTION
  - POSTERIOR WALL MI presents on the 12 Lead ECG as ST DEPRESSION in Leads V1 - V3.
  - POSTERIOR WALL MI is NOT PRESENT ON THIS ECG.
If this patient becomes HYPOTENSIVE . . . .
In every case of inferior wall STEMI, you must first rule out right ventricular MI before giving any:

- Nitroglycerin
- Diuretics
Nitroglycerin & Diuretics are CLASS III CONTRINDICATED in RIGHT VENTRICULAR MI ! !*

They precipitate SEVERE HYPOTENSION

* A.H.A. ACLS 2010 / 2015
LEFT DOMINANT

10 - 15% POPULATION

POSTERIOR VIEW

CASE STUDY 9 - STEMI

CHIEF COMPLAINT and SIGNIFICANT HISTORY:
42 y/o MALE arrived via EMS, c/o "HEAVY CHEST PRESSURE," SHORTNESS of BREATH X 40 min.
He has experienced V-FIB and been DEFIBRILLATED multiple times

RISK FACTOR PROFILE:
- CIGARETTE SMOKER
- HYPERTENSION
- HIGH LDL CHOLESTEROL

PHYSICAL EXAM:
Patient is alert & oriented x 4, ANXIOUS, with COOL, PALE, DIAPHORETIC
SKIN. C/O NAUSEA, and is VOMITING. LUNG SOUNDS: COARSE CRACKLES, BASES, bilaterally

VITAL SIGNS:
BP: 80/40  P: 70  R: 32  SAO2: 92% on 15 LPM O2

LABS:
TROBNIN: <.04
**SHOCK ASSESSMENT**

<table>
<thead>
<tr>
<th>LOC:</th>
<th>ANXIOUS</th>
<th>RESTLESS</th>
<th>LETHARGIC</th>
<th>UNCONSCIOUS</th>
<th>AWAKE</th>
<th>ALERT &amp; ORIENTED</th>
</tr>
</thead>
<tbody>
<tr>
<td>SKIN:</td>
<td>PALE / ASHEN</td>
<td>CYANOTIC</td>
<td>COOL</td>
<td>DIAPHORETIC</td>
<td>NORMAL HUE</td>
<td>WARM</td>
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<tr>
<td>BREATHING:</td>
<td>TACHYPNEA</td>
<td></td>
<td></td>
<td></td>
<td>NORMAL</td>
<td></td>
</tr>
<tr>
<td>PULSE:</td>
<td>WEAK / THREADY</td>
<td>TOO FAST or SLOW</td>
<td></td>
<td></td>
<td>STRONG</td>
<td></td>
</tr>
<tr>
<td>STATUS:</td>
<td><strong>SHOCK</strong></td>
<td></td>
<td></td>
<td></td>
<td><strong>NORMAL</strong></td>
<td></td>
</tr>
</tbody>
</table>

**CASE STUDY QUESTIONS:**

- Evaluate EKG for indicators of ACS:
  - ST SEGMENT ELEVATION / DEPRESSION
  - HYPERACUTE T WAVES
  - CONVEX ST SECTIONS
  - OTHER ST SEGMENT / T WAVE ABNORMALITIES

- Note leads with ST ELEVATION:
- Note leads with ST DEPRESSION:
- What is the suspected diagnosis?
- What is the "culprit artery" if applicable?
- List any critical structures compromised:
- List any potential complications:
LEADS II, III, and aVF VIEW
INFERIOR WALL of the LEFT VENTRICLE

FED by the RCA (75 - 80 % pop) or the CIRCUMFLEX (10 - 15 %)
LEADS V1 - V3 view the POSTERIOR WALL via RECIPROCAL CHANGES.
V5 - V6 VIEW THE LATERAL WALL
of the LEFT VENTRICLE
THE 12 LEAD ECG HAS TWO MAJOR BLIND SPOTS...

CHEST LEADS V1 - V6
WHAT EACH LEAD "SEES"...

THE 18 LEAD ECG COVERS THE ENTIRE HEART...

CHEST LEADS V1 - V6 PLUS V4R, V5R, V6R, and V7, V8, V9
WHAT EACH LEAD "SEES"...
INDICATIONS for 18 Lead ECG include:

- INFERIOR WALL MI
- ST Depression in LEADS V1-V4

To do 18 Lead ECG with 12 Lead machine – after you obtain 12 Lead, reposition CHEST LEADS to this configuration, then print!
Posterior wall STEMI – ST Elevation
V7 – V9
CLICK HERE to download “A SHORT Course in LONG QT Syndrome,” a focused excerpt from:
Brief, focused ECG excerpts from the “19th Congress, American College of Cardiology Accreditation Services” national conference, Miami, 2016………

Prevalence
SADS Foundation Stats:

• Each year in the United States, 350,000 Americans die suddenly and unexpectedly due to cardiac arrhythmias. Almost 4,000 of them are young people under age 35. (CDC 2002)
• In 30%–50% of sudden cardiac deaths, it is the first clinically identified expression of heart disease
• 10-12% of Sudden Infant Death Syndrome (SIDS) cases are due to Long QT Syndrome.
• LQTS is now known to be 3 times more common in the US than childhood leukemia.
• 1 in 200,000 high school athletes in the US will die suddenly, most without any prior symptoms—JAMA 1996; 276
The SADS Conditions:

- **Hypertrophic Cardiomyopathy** (HCM)
- **Long QT Syndrome** (LQTS)
- **Short QT Syndrome** (SQTS)
- **Brugada Syndrome** (BrS)
- **Arrhythmogenic Right Ventricular Dysplasia** (ARVD)
- **Catecholaminergic Polymorphic Ventricular Tachycardia** (CPVT)
- **Wolff-Parkinson-White** (WPW) Syndrome
- **Commotio Cordis**
- Less-common conditions (e.g. **Marfans**, **Ehlers-Danlos**, **Loeys-Dietz Syndromes**)

Estimated SADS Prevalence in US Population:

- HCM: 1/500  
  *J Am Coll Cardiol. 2014;64*
- BrS: 1/2,500  
  SADS Foundation
- LQTS: 1/2,500  
  Lenhart,SE 2007 AHA Circ
- ARVD: 1/10,000  
  SADS Foundation
- CPVT: 1/10,000  
  US Nat’l Library of Medicine
- WPW: 1/1,000  
  Circulation.2011; 124: 746-757
Prevalence
Sudden Deaths in Young Competitive Athletes
B Maron et al; AHA Circulation.2009; 119: 1085-1092

Analysis, causes of 1866 Deaths in the US, 1980 –2006:

- Cardiovascular: 56%
- Traumatic: 22%
- Commotio Cordis: 3%
- Heat Stroke: 2%
- Other: 17%

Prevalence
Adverse Drug Reactions: Torsades de Pointes secondary to QT prolonging medications:

- Occur in and out of hospital
- Underreported
- Medical community undereducated
- 7,000 in-hospital ADRs / year (all cause)
- Major issue with pharmaceutical industry, many drugs removed from market due to high incidence of TdP and TdP associated mortality
Compared to sudden death from CAD, SADS mortality prevalence is low, HOWEVER . . . .

• Nearly EVERY SADS death is a NEEDLESS TRAGEDY that could have been AVOIDED with appropriate screening and management.
• Many SADS victims are infants, children and young adults who are otherwise healthy.
• Sudden death is often the first symptom of SADS
• Diagnosed and managed properly, SADS patients can live long, productive and happy lives

Leave the detailed ECG diagnosis to the cardiologist.
Leave the detailed ECG diagnosis to the cardiologist.

However every critical care nurse, paramedic or other professional who reads an ECG should be aware of some important clues . . .
DETERMINING Q-T INTERVAL LIMITS
THE "QUICK PEEK" METHOD

Relatively accurate method to quickly identify patients with abnormal QT Intervals.
- Applies to patients with normal heart rates (60-100) and narrow QRS (QRSd <120ms)

The Q - T Interval should be LESS THAN 1/2 the R - R Interval
Determining the QT / QTc

Method 1 – 12 Lead ECG Report:

Standard 12 Lead ECG printout...

- Rate: 83, Sinus rhythm, Borderline
- PR: 183
- QRS: 88
- QT: 357
- QTC: 420

Heart Rate = 83
QT Interval = 357
QTc = 420

The QT Interval should be LESS THAN 1/2 the R-R Interval

OK  TOO LONG

DANGER!
WHEN THE “QUICK PEEK” METHOD for QT INTERVAL EVALUATION IS APPLIED TO THE ABOVE ECG, WHAT IS THE RESULT?

Dysrhythmia Associated with Mortality, Triggered by LQTS: *Torsades de Pointes*

Torsades de Pointes (TdP) – HEMODYNAMICS:
- Decreased – to – NO Cardiac Output
- Often patient PULSELESS during episode
- Patients often report SYNCOPE when TdP self-terminates.
- May DETERIORATE into VENTRICULAR FIBRILLATION and CARDIAC ARREST. (“Sudden Death”)

ECG Characteristics of TdP: The QRS Pattern of *Torsades de Pointes*

* a piece of Twisted Ribbon! *
**Etiology of Long QT Syndromes:**

**Congenital** (14 known subtypes)
Genetic mutation results in abnormalities of cellular ion channels

**Acquired**
- Drug Induced
- Metabolic/electrolyte induced
- Very low energy diets / anorexia
- CNS & Autonomic nervous system disorders

**Miscellaneous**
- Coronary Artery Disease
- Mitral Valve Prolapse

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**Prolonged Q-T Interval**

**Think:**
- Check K+ and Mag levels
- Possibility of Torsades
- Question meds that prolong Q-T
Avoidance of Meds that are known to prolong the QT interval. Click here for current list from CREDIBLEMEDS.ORG

Commonly used QT prolonging meds include:
- Amiodarone
- Procainamide
- Levaquin
- Erythromycin
- Norpace
- Tequin
- Benadryl
- Ritalin
- Pseudephedrine
- Haloperidol
- Thorazine
- Propulcid
- Zofran
- Ibutilide

and MANY more!

PATIENT 1: NORMAL

PATIENT 2: Genetic susceptibility; sensitivity to QT prolonging drugs:

Click here for link to paper by Kannankeril et al (2010 Pharmacological Reviews) that describes genetic susceptibility described above.
ECG Indicators: Hypertrophic Cardiomyopathy

- ECG may be normal
- Deep, narrow (dagger-like) Q waves
ECG Indicators: Hypertrophic Cardiomyopathy

- ECG may be normal
- Deep, narrow (dagger-like) Q waves
- Inverted T waves in multiple regions
- Left Ventricular and possibly Left Atrial Hypertrophy

Hypertrophic Cardiomyopathy (HCM)

12 Lead ECG Traits:
- QRS Height -- exceeds normal size, “spearing through QRS” in other leads
- Inverted T waves appear in multiple regions (ANTERIOR, LATERAL)
- BiPHASIC T waves in Inferior Leads.
- T WAVES are SYMMETRICAL.
ECG Indicators: Brugada Syndrome

1. RBBB PATTERN
2. J POINT ELEVATION V1, V2 and possibly V3
3. DOWNWARD SLOPING S-T SEGMENT
4. INVERTED T WAVE
5. GIVES S-T SEGMENT A "TRIANGULAR" APPEARANCE
IS THERE ANYTHING ABNORMAL WITH THIS EKG?

This patient exhibits a "classic" Type I Brugada Syndrome ECG pattern:
- Elevated J points in V1, V2
- Downsloping "coved" ST segment
- Inverted T wave.

Never forget the "triangular" shape!
PATTERNS of S-T ELEVATION:

**BEWARE of the**

"TRIANGULAR" SHAPED S-T SEGMENT IN V1, V2, and sometimes also in V3. . . . THINK - -

**BRUGADA SYNDROME**

Trigger for Torsades de Pointes – ECTOPIC BEAT during The “ELECTRICAL GRADIENT” phase shown above.
Torsades de Pointes:
- Decreased – to – NO Cardiac Output
- Often patient PULSELESS during episode
- Causes SYNCOPE
- Often DETERIORATES into VENTRICULAR FIBRILLATION and CARDIAC ARREST.

Brugada / Long QT Syndromes cause:
ECG abnormality diagnostic or suspected of Brugada syndrome.

**BRUGADA SYNDROME**

- **SEVERAL VARIATIONS** of this disorder are known to exist.
- **CONCEALED** and **NON-CONCEALED**.
- The **NON-CONCEALED** version **HAS THE V1-V3 abnormality VISIBLE** at all times.
- The **CONCEALED** version - pt. has a NORMAL EKG at most times - a DRUG STUDY, an EP STUDY, and/or GENETIC TESTING must be done to rule out or confirm diagnosis.
Arrhythmogenic Right Ventricular Dysplasia

- A genetically acquired myocardial disease associated with paroxysmal ventricular arrhythmias and sudden cardiac death.
- Characterized pathologically by fibro-fatty replacement of the right ventricular myocardium.
- The second most common cause of sudden cardiac death in young people (after HOCM), causing up to 20% of sudden cardiac deaths in patients < 35 yrs of age.
- Typically inherited as an autosomal dominant trait, with variable penetrance and expression (there is an autosomal recessive form called Naxos Disease, which is associated with woolly hair and skin changes).
- More common in men than women (3:1) and in people of Italian or Greek descent.
- Estimated to affect approximately 1 in 5,000 people overall.

Arrhythmogenic Right Ventricular (RV) Cardiomyopathy and/or Dysplasia:

In 1 autopsy study examining a series of 200 cases of sudden death associated with arrhythmogenic RV cardiomyopathy and/or dysplasia, death occurred in 9.5% of cases during the perioperative period. This emphasizes the importance of close perioperative evaluation and monitoring of these patients for ventricular arrhythmia. Most of these patients require cardiac electrophysiologist involvement and consideration for an implantable cardioverter-defibrillator (ICD) for long-term management.

ARVD – 12 Lead ECG Indicators

1. "Incomplete RBBB" Pattern
2. V1, V2 Rs pattern
3. Inverted T waves, symmetrical, - Global

ARVD ECG 1

1. Epsilon's waves
1. "Incomplete RBBB" Pattern
2. V1, V2 Rs pattern
3. Inverted T waves, symmetrical, - Global
4. Epsilon’s waves
9/3/2018

CLICK HERE to download "A SHORT Course in LONG QT Syndrome," a focused excerpt from:

Evidence Based Reference Sources

- **2016 ACC Interassociation Consensus Statement on Cardiovascular Care of College Student-Athletes**
- **2014 AHA/ACC Scientific Statement**: Assessment of the 12-Lead ECG as a Screening Test for Detection of Cardiovascular Disease in Healthy General Populations of Young People (12–25 Years of Age)
- **AHA Circulation: Inherited Arrhythmias; Basic Science for Clinicians**
- **AHA ACC Scientific Statement Prevention of Torsade de Pointes in Hospital Settings**
- **AHA ACC QTc Behavior During Exercise and Genetic Testing for the Long-QT Syndrome**
- **Pharmacology Review: Drug Induced Long QT Syndromes**
Evidence Based Reference Sources, cont’

• HRS/EHRA/APHRS Expert Consensus Statement on the Diagnosis and Management of Patients with Inherited Primary Arrhythmia Syndromes
• Genetic Determinants of Sudden Cardiac Death: AHA Circulation.2008; 118: 1854-1863
• AHA/ACCF/HRS Recommendations for the Standardization and Interpretation of the Electrocardiogram: Part III: Intraventricular Conduction Disturbances
• AHA/ACCF/HRS Recommendations for the Standardization and Interpretation of the Electrocardiogram : Part V: Electrocardiogram Changes Associated With Cardiac Chamber Hypertrophy
• Arrhythmogenic Disorders of Genetic Origin; Brugada Syndrome: Circulation: Arrhythmia and Electrophysiology.2012; 5: 606-616

Other Reference Sources:

www.JACC.org

http://circ.ahajournals.org/

www.SADS.org
My top two reasons for giving everything in life the best I have to offer.