## All <br> is



## Is Not an MI

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## Lead Placement



## Bipolar Leads

## Limb leads MUST go on the limbs

## Precordial Leads



V1 $4^{\text {th }}$ ICS 1 " right of the sternum
V2
$4^{\text {th }}$ ICS 1 " left of the sternum
V3 Inline between V2 and V4
V4 $\quad 5^{\text {th }}$ ICS left mid clavicular line
V5 Inline between V4 and V6
V6 Left mid axillary line at same level as V4


Waveforms are created as a result of their direction of movement in relation to the positive electrode

## Leads

Leads "look" at the heart
Record the flow of electricity through the heart

Bi Polar Leads (limb leads)


Augmented Leads


## Contiguous Leads

| Leads | View |
| :--- | :--- |
| II, III, aVF | Inferior |
| V1, V2 | Septal |
| V3, V4 | Anterior |
| V5, V6, I, aVL | Lateral |

Precordial Leads (V Leads)


R wave progression:
V6 should have a greater positive deflection than V1

## Einthoven's Triangle



Normal 12 Lead EKG Appearance

| I | aVR | V 1 | V 4 |
| :---: | :---: | :---: | :---: |
| II |  | aVL | V 2 |
| V |  |  |  |
|  |  | V 3 | V 5 |
|  |  |  |  |

## ST Segment Elevation



## Indicative Changes



Normal


Possible Ischemia (No Cardiac Alert)


Injury (Cardiac Alert)


Injury (Cardiac Alert)

Injury (Cardiac Alert)


Old Infarction (No Cardiac Alert)

## Normal Ventricular Conduction

- From the bundle of His to the bundle branches
o Right
o Left
- Ventricular septum depolarizes from the left to the fight
- Left and right ventricles depolarize simultaneously

- QRS $\leq 0.10$ seconds
- rS complex in V1
- Most positive complex in V6



## Left Bundle Branch Block

## Ventricular Conduction in a Left Bundle Branch Block

- The $L$ bundle branch is blocked and impulses will not travel through it
- Impulse travels through the intact $R$ bundle branch causing depolarization of the $R$ ventricle
- The $L$ ventricle receives no direct depolarization

Depolarization of the Left Ventricle

- The $L$ ventricle is depolarized indirectly
- Impulses cross the intraventricular septum
 from the R ventricle causing depolarization
- This causes a delay in depolarization and results in a wide QRS complex


## EKG Findings in a Left Bundle Branch Block

Definitive characteristics

- Prolonged QRS; > 0.12 sec
- $\quad$ rS pattern in $\mathrm{V}_{1}$

Other changes you may see

- Wide R waves in $\mathrm{I}, \mathrm{a}_{\mathrm{L}}, \mathrm{V}_{5}-\mathrm{V}_{6}$
- $R$ waves may be notched
- Deep, wide $S$ waves in $\mathrm{V}_{1}-\mathrm{V}_{3}$



## Early Repolarization

- Early repolarization describes a pattern of localized or diffuse ST segment elevation. This is especially seen in leads with prominent $R$ wave
- Early repolarization is a cause of ST elevation. This innocent condition typically occurs in young healthy males. The T wave begins early, adding elevation to the ST segment
- Usually, early repolarization shows elevation of the J point (the junction between the end of the QRS and the ST segment) and a concave upward curve towards the T wave. ("Concave upward" means the hollow portion of the curve is on top.)
- Usually seen in the anterior precordial leads of the ECG, but can be seen in limb leads to a lesser degree.
- Early repolarization cannot always be differentiated from myocardial infarction. In the chest pain patient, it's safest to assume ST elevation to be infarction until proven otherwise by reviewing a previous ECG or by obtaining serial ECGs
- PR depression is absent, and reciprocal changes tend not to be found in lead=s aVR and V 1 . ST elevation due to early repolarization is said to be more common in young African-American males, and tends to be most apparent at slower heart rates.
- While the ECG can be misleading, the clinical history and physical examination frequently point to the correct diagnosis. The chest discomfort of pericarditis is sharp, pleuritic, and positional, unlike that of acute infarction. A multi component pericardial friction rub is frequently present on physical examination in patients with acute pericarditis.

EKG Findings of Early Repolarization

- ST segment elevation commonly seen in I, II, aVF, and V2 - V6
- ST depression may be seen in aVR
- Concave, up sloping ST segments
- J point is frequently elevated
- No reciprocal changes




## Hyperkalemia

- Caused by elevated serum potassium levels
- Electrolyte imbalances rare in patients with healthy kidneys and access to water
- May mimic an AMI pattern
- The PR interval may be normal or prolonged
- EKG changes are dependent upon potassium levels
$K^{+}$Level
$5.5-6.5 \mathrm{mEq} / \mathrm{L}$
$6.0 \mathrm{mEq} / \mathrm{L} \quad$ ST segments disappear
$6.0-6.5 \mathrm{mEq} / \mathrm{L}$
$6.5 \mathrm{mEq} / \mathrm{L}$
$7.0-9.0 \mathrm{mEq} / \mathrm{L}$
$10.0 \mathrm{mEq} / \mathrm{L}$


## EKG Change

T waves become peaked

QRS complex begins to widen
$P$ waves begin to flatten out
$P$ waves disappear
-Earliest T wave changes seen in II, III, and V2 - V6

QRS becomes very wide and slurred. They may merge with the
T waves causing the appearance of a sine wave


## Pericarditis

- Depressed PRI
- ST elevation all leads (could localize)
- No reciprocal depression
- Dry cough
- Positional chest pain
- Recent history of illness
- Presentation is similar to MI


Comparison of ECG Changes Associated with Acute Pericarditis, Myocardial Infarction and Early Repolarization

| ECG finding | Acute pericarditis | Myocardial infarction | Early repolarization |
| :--- | :--- | :--- | :--- |
| ST-segment shape | Concave upward | Convex upward | Concave upward |
| Q waves | Absent | Present | Absent |
| Reciprocal ST-segment changes | Absent | Present | Absent |
| Location of ST-segment <br> elevation | Limb and precordial leads | Area of involved artery | Precordial leads |
| Loss of R-wave voltage | Absent | Present | Absent |
| PR-segment depression | Present | Absent | Absent |

## Ventricular Hypertrophy

## Left Ventricular Hypertrophy

## Causes

- Systemic hypertension
- Aortic stenosis
- Hypertrophic cardiomyopathy


## EKG Findings

| Wave | I | III | aVL | V1 or V2 | V5 or V6 |
| :--- | :--- | :--- | :--- | :--- | :--- |
| - R Wave | $\geq 20 \mathrm{~mm}$ |  | $\geq 11 \mathrm{~mm}$ |  | $\geq 30 \mathrm{~mm}$ |
| - S Wave |  | $\geq 20 \mathrm{~mm}$ |  | $\geq 30 \mathrm{~mm}$ |  |

- Summation
$-R(I, I I$ or III) $+S(I, I I$ or III $)=\geq 20 \mathrm{~mm}$
$-R I=S$ III $=\geq 25 \mathrm{~mm}$
$-S(V 1$ or $V 2)+R(V 5$ or $V 6)=\geq 35 m m$
- Strain Pattern
$-1$
- aVL
- V5
- V6




## Causes

- Pulmonary hypertension
- COPD
- Mitral valve stenosis
- Pulmonary embolism


## EKG Findings

- Q waves may be present in II, III, and aVF
- Tall R waves in II, III, and V1
- R waves in $\mathrm{V} 1>7 \mathrm{~mm}$ and equal to or greater than the depth of the S wave
- Deeper than normal S waves in I and V4 - V6
- In V6 the depth of the $S$ wave may be greater than the $R$ wave
- Strain Pattern
- II
- III
- aVF
- V1




## Digitalis

## EKG Changes

- Prolonged PRI
- Flattened or inverted T waves
- Shortened QT intervals $(0.30 \mathrm{sec})$
- ST segments depressed $>1 \mathrm{~mm}$
o "dig scoop"


Interpretation:


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Interpretation:


