ECG Interpretation

Introduction to Cardiac Telemetry
Michael Peters, RN, CCRN, CFRN – CALSTAR Air Medical Services
Disclosures

• Nothing to disclose
Objectives

• Describe the electrical conduction pathway in the heart
• Interpret a rhythm strip and identify life threatening dysrhythmias
• Anticipate common dysrhythmia treatment plans
• Identify presence of a pacemaker and its proper operation
• Apply skills toward the introduction of 12-lead EKG analysis
Cardiac Anatomy & Physiology

[Image: Cartoon of a heart saying "Imposter!"
A cartoon heart is speaking with another heart character who is surprised and angry. The text "Imposter!" is shown in bold, uppercase letters, conveying a humorous or exaggerated message about hearts, possibly referencing the complexity or misconception about cardiac anatomy and physiology.]
Cardiac Anatomy & Physiology

**Conducting System, a series of Specialized Cardiac Muscle Cells**

1. **The sinoatrial (SA) node (pacemaker)** generates 70-80 impulses per minute; atria contract.

2. The impulses pause (0.1 sec) at the AV node so ventricles have time to fill.

3. **The atrioventricular (AV) bundle** connects the atria to the ventricles.

4. The AV bundle branches conduct the impulses through the interventricular septum.

5. **The Purkinje fibers** stimulate the contractile cells of both ventricles, starting at apex and moving superiorly.
Einthoven’s Triangle

- Lead I
  - Right arm (RA)
  - Left arm (LA)
  - Lead II
  - Left leg (LL)
  - Lead III

**Lead** | **Views** | **Heart Chambers**
--- | --- | ---
Lead I | Lateral | Left ventricle, left atrium
Lead II | Inferior | Left and right ventricle,
Lead III | Inferior | Right and left ventricles
Precordial Leads

- Frontal plane
- Horizontal plane

- Lead locations:
  - V1, V2, V3, V4, V5, V6
  - aVR, aVL, aVF

- Heart regions:
  - RV, LV

- Body orientation:
  - Anterior, Posterior, Lateral
ECG monitor lead placement

“White on Right”

“Smoke over Fire”

“Snow over Trees”

“Chocolate lies close to the heart”
Anatomy of the ECG

**P wave**
- Precedes QRS
- Usually rounded and upright
- 2-3mm amplitude
- 0.06 - 0.12 second duration

**Abnormalities**
- Notched, peaked, enlarged – atrial hypertrophy
- Inverted – retrograde (junctional) conduction
- Varying – wandering pacemaker rhythm
Anatomy of the ECG

**PR Interval**
- Measured from the beginning of the P-wave to the beginning of the QRS.
- Tracks atrial impulse from SA node through AV node, bundle branches.
- 0.12 – 0.20 second duration
Anatomy of the ECG

**QRS Complex**
- Depolarization of the ventricles
- 5 – 30mm amplitude
- 0.06 – 0.10 second duration
- May not see all 3 waves
- + or – deflection depending on lead

Wide QRS (>0.12 sec) may signify ventricular conduction delay or origin.

- Notched R wave – BBB
- Deep Q wave – prior MI
Anatomy of the ECG

**ST Segment**
- Beginning of ventricular repolarization
- S to beginning of T
- Usually isoelectric

**ST depression**
- >0.5mm below baseline
- Myocardial ischemia, electrolyte imbalance

**ST elevation**
- >1mm above baseline
- Myocardial injury (STEMI)
Anatomy of the ECG

**T wave**
- Ventricular repolarization
- Follows S wave
- 0.5-10mm amplitude
- Round and smooth
- Upright I, II, V3-V6, inverted aVR
Anatomy of the ECG

**T wave**

- Tall, peaked or tented – myocardial injury or hyperkalemia
- Inverted (I, II, V3-V6) – myocardial ischemia
- Notched or pointed (adult) - pericarditis
Anatomy of the ECG

**QT interval**
- Represents ventricular polarization/depolarization
- Beginning of QRS to end T
- Varies with age, gender, heart rate (0.36 – 0.44 sec)

**Prolonged QT**
- Congenital defect
- Caused by certain medications
- Risk for torsades de pointes
# QT Prolongation Drugs

## DRUGS TO BE AVOIDED BY CONGENITAL LONG QT PATIENTS

<table>
<thead>
<tr>
<th>Generic Name</th>
<th>Brand Name</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atracurium (PR)</td>
<td>Keepl</td>
</tr>
<tr>
<td>Alcuronium (PR)</td>
<td>Edaravone</td>
</tr>
<tr>
<td>Axonanotide (PR)</td>
<td>Rapsodol</td>
</tr>
<tr>
<td>Bembricine (PR)</td>
<td>Yoxapin</td>
</tr>
<tr>
<td>Breiprolin (PR)</td>
<td>Idoxx</td>
</tr>
<tr>
<td>Breiprolin (PR)</td>
<td>Rubus and others</td>
</tr>
<tr>
<td>Breiprolin (PR)</td>
<td>Sennat and others</td>
</tr>
<tr>
<td>Breiprolin (PR)</td>
<td>Streptococcus and others</td>
</tr>
<tr>
<td>Breiprolin (PR)</td>
<td>Tramadol and others</td>
</tr>
<tr>
<td>Breiprolin (PR)</td>
<td>Lortrax</td>
</tr>
<tr>
<td>Breiprolin (PR)</td>
<td>Zenafide and others</td>
</tr>
<tr>
<td>Breiprolin (PR)</td>
<td>Dentine and others</td>
</tr>
<tr>
<td>Breiprolin (PR)</td>
<td>Paracetamol and others</td>
</tr>
<tr>
<td>Breiprolin (PR)</td>
<td>Disodium and others</td>
</tr>
<tr>
<td>Breiprolin (PR)</td>
<td>Ixekin and others</td>
</tr>
<tr>
<td>Breiprolin (PR)</td>
<td>Zosozide and others</td>
</tr>
<tr>
<td>Breiprolin (PR)</td>
<td>Oxigen and others</td>
</tr>
<tr>
<td>Breiprolin (PR)</td>
<td>Degrin (discontinued) and others</td>
</tr>
<tr>
<td>Breiprolin (PR)</td>
<td>Thermoproteasome and others</td>
</tr>
<tr>
<td>Breiprolin (PR)</td>
<td>Septin and others</td>
</tr>
<tr>
<td>Breiprolin (PR)</td>
<td>Tararon and others</td>
</tr>
<tr>
<td>Breiprolin (PR)</td>
<td>Valrenen and others</td>
</tr>
<tr>
<td>Breiprolin (PR)</td>
<td>Vendaril and others</td>
</tr>
<tr>
<td>Breiprolin (PR)</td>
<td>Zetax and others</td>
</tr>
<tr>
<td>Breiprolin (PR)</td>
<td>Zosozide and others</td>
</tr>
</tbody>
</table>

**Note:** Medicines on this list are reviewed on an ongoing basis to assure that the available evidence supports their continued placement on this list. The list changes regularly and we recommend checking the website at credibledrs.org for the most up-to-date information. There may be many additional brand names that are not listed on this form.

**Disclaimer and Waiver:** Information presented is intended solely for the purpose of providing general information about health-related matters. It is not intended for any other purpose, including but not limited to medical advice and/or treatment, nor is it intended to substitute for the users' relationships with their own healthcare providers. To that extent, by use of this website and the information it contains, the user affirms the understanding of the purpose and releases AZCERT, Inc. from any claims arising out of its use of the website and its lists. The absence of drugs from these lists should not be considered an indication that they are free of risk of QT prolongation or torsades de pointes. Many medicines have not been tested for this risk in patients, especially those with congenital long QT syndrome.

**Generated:** September 17, 2018.  **List last revised:** September 12, 2018.
Analyzing a Rhythm Strip

Analyzing a Rhythm Strip

Heart Rate – Methods of calculation

1. Six second count

7 QRS complexes in 6 seconds \times 10 = 70 QRS complexes/minute

Analyzing a Rhythm Strip

Heart Rate – Methods of calculation

2. Countdown Method

Analyzing a Rhythm Strip

Heart Rate – Methods of calculation

3. Caliper Method (1,500 Method)
   – Use calipers to measure the number of 1mm (small) boxes between R-R interval. Divide 1500 by this #.

   Example:
   1500 / 19 QRS complexes per minute
   Heart Rate = 79 beats per minute
Artifact and Wandering Baseline
Interpreting a Rhythm Strip

1. Rate – Too fast or slow?
2. Rhythm – Regular or Irregular?
3. QRS wide or narrow?
4. P-wave for every QRS?
5. ST segment/T-wave abnormalities, ectopy, pacemaker spikes, etc.
Normal Sinus Rhythm

Rate: 60-100 bpm

Rhythm: Regular

QRS: Narrow (<0.12 sec)

P wave for every QRS

T wave rounded, upright

No ectopic beats (PVC/PAC)
Sinus Bradycardia

Rate: Less than 60 bpm
Rhythm: Regular
QRS: Narrow (<0.12 sec)

P wave for every QRS
T wave rounded, upright
No Ectopic beats
Sinus Tachycardia

Rate: **Greater than 100bpm**
P wave for every QRS

Rhythm: Regular
T wave rounded, upright

QRS: Narrow (<0.12 sec)
No Ectopic beats
Sinus Arrhythmia

Usually regular sinus rate (60-100) with irregular rhythm that corresponds with respirations. Common in pediatrics.
Sinus Arrest/Pause

SA node fails to discharge and then resumes.
Atrial Arrhythmias

• Premature Atrial Contraction (PAC)
  – Conducted (QRS complex following) or non-conducted.
  – P-P interval shorter than prior beats.
  – P wave may be hidden in prior T wave.
Atrial Arrhythmias

- Premature Atrial Contraction (PAC)
  - Conducted (QRS complex following) or non-conducted.
  - P-P interval shorter than prior beats.
  - P wave may be hidden in prior T wave.
Atrial Arrhythmias

- Multifocal Atrial Tachycardia (MAT)
  - Irregular rhythm, rate >100, varying P waves
Atrial Arrhythmias

- Wandering Atrial Pacemaker
  - Irregular rhythm resulting from multiple pacemaker sites initiating beats.

http://www.ekgstripsearch.com/WAP.htm
Atrial Arrhythmias

• Paroxysmal Atrial Tachycardia (PAT)
  – Brief periods of tachycardia that alternate with periods of normal sinus rhythm
SUPRAVENTRICULAR TACHYCARDIA (SVT)

Rate: 150–250 bpm
P Waves: Frequently buried in preceding T waves and difficult to see
PR Interval: Usually not possible to measure
Rhythm: Regular
QRS: Normal (0.06–0.10 sec) but may be wide if abnormally conducted through ventricles
Atrial Fibrillation

Chaotic, asynchronous, electrical activity in atrial tissue.

- Leads to loss of atrial kick (30% of cardiac output)
- Absence of P waves and irregular ventricular response (Irregularly irregular)
Atrial Flutter

- Atrial rate approx. 300 bpm with characteristic “sawtooth” pattern (flutter waves).
- Ventricular rate dependent on conduction ratio.
- Symptoms dependent on cardiac output.
Atrial Flutter

- Atrial rate approx. 300 bpm with characteristic “sawtooth” pattern (flutter waves).
- Ventricular rate dependent on conduction ratio.
- Symptoms dependent on cardiac output.
Junctional Rhythms
Junctional Rhythms

- Originates from AV junction if SA node fails
- Atria depolarize but impulse is retrograde (reverse)
- Junctional escape rate 40-60bpm
Junctional Rhythms

Accelerated Junctional (60-100 bpm)

Junctional Tachycardia (>100 bpm)
First Degree AV Block

*Characterized by PR interval > 0.2 sec*

Rate: Normal (not affected)  
P wave for every QRS

Rhythm: Regular  
Narrow QRS, normal T
2nd Degree AV Block Type I

*AKA Wenckebach or Mobitz I*

Rate: Normal (A>V)  P wave for every QRS but
Rhythm: Atrial – regular  QRS drops
Ventricular – Irregular  Gradual PR increase

Successive prolongation of the PR interval until a P-wave is completely blocked
2nd Degree AV Block Type II

*AKA Mobitz II*

Rate: Normal (A>V)  
P wave for every QRS but

Rhythm: Atrial – regular  
QRS drops SUDDENLY

Ventricular – Irregular  
PR remains constant
3rd Degree AV Block (Complete)

*Potential LIFE THREATENING Rhythm*

Rate: Slow (20-60) QRS: normal or wide
Rhythm: Atrial – regular; Ventricular – regular
Atria and ventricles operating independently, no relation between P and QRS.
3rd Degree AV Block (Complete)

3° AV Block
Atria and Ventriles are depolarizing independently
No association between atria & ventricles

![Diagram showing 3rd Degree AV Block](https://www.unm.edu/~lkravitz/EKG/avblocks.html)
Bundle Branch Block
Bundle Branch Blocks

- QRS > 0.12
- Assess V1 for QRS morphology
- Patients with a prolonged QRS (> 0.15) may require a pacemaker
Practice with Heart Blocks
More Practice...
Ventricular Dysrhythmias

- QRS > 0.12
- Wide and Bizarre
- Hidden P waves
- 20-40 BPM
Premature Ventricular Contraction (PVC)
Premature Ventricular Contraction (PVC)

• Patient Assessment?
• Treatment?
Ventricular Tachycardia

- Life-threatening dysrhythmia!
- Wide and bizarre
- Rate of 101-250 impulses/min
- Pulses vs. pulseless
Torsades De Pointes

- Lethal dysrhythmia!
- Wide and bizarre
- Often > 150 impulses/min
- Pulses vs. pulseless
Torsades De Pointes

• Lethal dysrhythmia!
• Wide and bizarre
• Often > 150 impulses/min
• Pulses vs. pulseless
Ventricular Fibrillation

Pulseless!!! Lethal dysrhythmia
EMS

WHERE FIBBERS RARELY MAKE IT TO HEAVEN.
Idioventricular/Agonal Dysrhythmia

- Lethal! Last attempt!
Asystole

Lethal!
Immediate Treatment Required!
Pulseless Electrical Activity (PEA)
Pulseless Electrical Activity (PEA)

FREQUENTLY LISTED CAUSES OF PEA (Hs & Ts)

- Hypovolemia
- Hypoxia
- Hydrogen ion
  - acidosis
- Hyperkalemia
- Hypokalemia
- Hypothermia
- (Hypoglycemia)
- Toxins
- Tamponade
- Tension PTX
- Thrombosis
  - coronary
- Thrombosis
  - pulmonary
- (Trauma)
Pacemakers

- Power source generates an impulse, which is transmitted to the heart tissue, causing depolarization.

Pacemakers

Types of temporary pacing

- **Epicardial** – wires attached directly to the heart wall during cardiac surgery. (A/V)
Pacemakers

Types of temporary pacing

- **Transvenous** – Placed percutaneously and advanced to the ventricle via the IJ or subclavian vein. (V)

Pacemakers

Types of temporary pacing

- Transcutaneous – Pads placed on the patient’s bare skin.
Pacemakers
Failure to Capture

• Check connections, patient position, increase mA.
Pacemaker Troubleshooting

Failure to Pace

• Check connections, change battery, change pulse generator
• Over sensing?? – mistakes other impulses or muscle activity for intrinsic activity
Pacemaker Troubleshooting

Failure to Sense
- Increase sensitivity
- Decrease demand rate
Recognizing Cardiac Tissue Damage

ECTOPOOPY:
WHEN YOU LOOK AT YOUR PATIENTS’ ECG AND SAY ‘OH S**T’!
Recognizing Cardiac Tissue Damage

More than just ST-elevation

**Ischemia**

– Interrupted oxygen (blood) supply.
– Tissue is still viable
– Repolarization temporarily impaired (inverted T-waves)
Recognizing Cardiac Tissue Damage

More than just ST-elevation

**Injury**
- Prolonged lack of oxygen (blood) supply.
- Tissue is still viable (for now)
- Cells do not fully repolarize because of deficient blood supply (ST elevation)
Recognizing Cardiac Tissue Damage

More than just ST-elevation

**Infarction**
- Areas of necrosis (dead tissue).
- Cells do not depolarize, causing new pathological Q-waves to appear.
- Tissue is eventually replaced by scar tissue.
Zones of Injury

Ischemia: T-wave inversion

Injury: ST-elevation

Infarction (necrosis): pathological Q-wave

Reciprocal Changes (opposite side of heart)
A Look at 12-Leads
Where is the MI?
Where is the MI?
Location Matters

Anterior, Septal, Lateral – Involve Left Anterior Descending and Circumflex

• Left sided Heart Failure (Pump problem)
  – Dyspnea (w/wo exertion)
  – Pulomanry Edema (rales, crackles)
  – Orthopnea
  – Cool extremities, weak pulses, cyanosis
  – Lethargy, fatigue, confusion
  – Restlessness

• Treatment – ASA, O2, analgesia, NTG, inotrope
Location Matters

Inferior, RV, Posterior – Right Coronary Artery (possibly L Circumflex)

• RV failure (Preload problem)
  – JVD, Edema, Swelling
  – Anorexia, Nausea, Ascites
  – Confusion, Lethargy
  – Hypotension
  – Heart Block (be prepared to PACE!)

• Treatment – ASA, O2, analgesia, VOLUME
Location Matters

• R sided MI: Why not NTG??
  – Nitro vasodilates, causing further loss of preload (i.e. blood, O2), worsening the problem.

You're gonna do WHAT?
R sided MI after NTG
Putting It All Together

- Always *assess* your patient first!
  - Treat the patient not the monitor!
- Determine the situation
- Investigate the cause
- Intervene/Treatment
- Reassess
- Get to definitive care
References


Practice Strips
Practice Strips
Practice Strips
Practice Strips
• **ECG simulator**