EKG Review and Cardiac Arrhythmias

Bob Wilmouth

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Depolarization and Repolarization

- **Depolarization**: Cell's membrane charge becomes positive in order to generate an action potential. Caused by positive sodium and calcium ions going into the cell.
- **Repolarization**: Cell's membrane charge returns to negative after depolarization. Caused by positive potassium ions moving out of the cell.
EKG Lingo

- P - wave
- Q - wave
- R - wave
- S - wave
- T - wave
- PR interval
- QRS interval
- ST segment

P Wave caused by atrial depolarization
norm is less than 0.12 secs

PR Interval
- Time from the beginning of the P wave to the beginning of the QRS complex (onset of ventricular depolarization) Normal range is from 0.12 sec - 0.20 sec
- Atrial contraction begins in the middle of the P wave and continues throughout the PR interval
- Corresponds to the delay necessary for the ventricles to fill after atrial contraction
- The atrial repolarization wave (electrical impulse) is usually hidden by the QRS complex

QRS Complex
Time it takes for depolarization of the ventricles
Norms - 0.04 to 0.12 sec measured from the initial deflection of the QRS from the isoelectric line to the end of the QRS complex.

R wave is the point when half of the ventricular myocardium has been depolarized
RS line is activation of the posteriobasal portion of the ventricles
Ventricular depolarization requires normal function of the right and left bundle branches.
Ventricular contraction begins half-way through the QRS complex and continues to the end of the T-wave.
Pumping of blood begins when ventricular pressure exceeds aortic pressure, causing the semi lunar valves to open. This is normally at the end of the QRS complex and start of ST segment.

ST Segment
Period from the end of ventricular depolarization to the beginning of ventricular repolarization. Although the ST segment is isoelectric, the ventricles are actually contracting. Norm 0.08 to 0.12 sec

QT Interval
- Normally 0.34 seconds to 0.43 second
- Measure from the beginning of the Q to the end of the T
- Represents the total duration of electrical activity of the ventricles.
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**T Wave**
- Corresponds to the rapid ventricular repolarization
- Normally rounded and positive
- May be positive, negative or biphasic

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**U wave**
- Repolarization of the purkinje fibers
- Not always seen
- Prominent U waves
  - hypokalemia, hypercalcemia, thyrotoxicosis, or exposure to digitalis, epinephrine
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**Precordial Leads**

- Six Precordial Electrode Placement:
  - V1 - fourth intercostal, right sternal border.
  - V2 - fourth intercostal, left sternal border.
  - V3 - equal distance between V2 and V4.
  - V4 - fifth intercostal, left mid clavicular line.
  - V5 - anterior axillary line, same level with V4.
  - V6 - mid axillary line, same level with V4 and V5

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**Precordial Leads**

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**12-lead EKG**

- 10 leads that are placed to look at heart from many different angles, producing 12 views of the heart.
- 3-D view of the heart.
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EKG Distributions

- Anteroseptal: V1, V2, V3, V4
- Anterior: V1–V4
- Anterolateral: V4–V6, I, aVL
- Lateral: I and aVL
- Inferior: II, III, and aVF
- Inferolateral: II, III, aVF, and V5 and V6

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ECG Interpretation

- Systematic approach to reading ECGs
  - Rate
  - Regularity
  - Rhythm
  - P waves
  - PR interval
  - R wave progression
  - QRS interval
  - ST segment
  - QT interval

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**The QRS Axis**
- Represents the overall direction of the heart's activity
- Axis of -30 to +90 degrees is normal

![Diagram of QRS Axis]

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**The Quadrant Approach**
- QRS up in I and up in aVF = Normal

<table>
<thead>
<tr>
<th>Lead/Phase</th>
<th>Positive</th>
<th>Normal Axis</th>
<th>LAD</th>
<th>Indeterminate Axis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Positive</td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Negative</td>
<td></td>
<td></td>
<td>LAD</td>
<td></td>
</tr>
</tbody>
</table>

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[ECG Lead Diagram]

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Determine regularity
- Look at the R-R distances
- Regular (are they equal distance apart)?
  Occasionally irregular? Regularly irregular? Irregularly irregular?

Assess the P waves
- Are there P waves?
- Do the P waves all look alike?
- Do the P waves occur at a regular rate?
- Is there one P wave before each QRS?

P wave abnormalities
- Lack of P waves caused by
  - Atrial fibrillation
  - Atrial flutter
- Biphasic P waves can be seen
  - Dysrhythmias
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Determine PR interval

- Normal: 0.12 - 0.20 seconds.
  (3 - 5 boxes)

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PR interval

- Prolonged PR interval is caused by:
  - 1st degree AV block
  - 2nd degree AV block Type I
  - 2nd degree AV block Type II
  - 3rd degree AV block

- Short PR interval is caused by:
  - WPW

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R wave progression

- The QRS complex should start out negative in lead V1.
- The QRS complex should end up positive in lead V6.
- The R wave will be tallest in lead V3 or V4 (can be dependent on lead placement).
- When the transition happens in lead V1 or V2 it is referred to as an early transition and can be indicative of a previous posterior wall MI.
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QRS duration

- Normal: 0.04 - 0.11 seconds.
  (1 - 3 boxes)
- Anything 0.12 or greater is considered a Bundle Branch Block or Interventricular conduction delay

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QRS Segments

- Axis
- Conduction delays
  - Bundle Branch Blocks
  - IVCD
- Left ventricular hypertrophy

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Conduction Delays

- If the QRS complex is wider than 0.12 seconds, this is caused by a delay in the conduction tissue of one of the bundle branches:
  - Left Bundle Branch Block (LBBB)
  - Right Bundle Branch Block (RBBB)
  - Intraventricular conduction delay
For RBBB the wide QRS complex assumes a unique, virtually diagnostic shape in those leads overlying the right ventricle (V₁ and V₂). "Rabbit Ears"

For LBBB the wide QRS complex assumes a characteristic change in shape in those leads opposite the left ventricle (right ventricular leads - V₁ and V₂). Broad, deep S waves
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IVCD – Intraventricular Conduction Delay
• QRS duration >0.10s indicating slowed conduction in the ventricles
• Criteria for specific bundle branch or fascicular blocks not met

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Left Ventricular Hypertrophy
• Criteria exists to diagnose LVH using a 12-lead ECG.
  – For example:
    • The R wave in V5 or V6 plus the S wave in V1 or V2 exceeds 35 mm.
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**Left Ventricular Hypertrophy**

Compare these two 12-lead ECGs.

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**Normal**

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**Left Ventricular Hypertrophy**

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**ST Segments**

- The normal ST segment has a slight upward concavity.
- Flat, downsloping, or depressed ST segments may indicate coronary ischemia.
- ST elevation may indicate myocardial infarction. An elevation of >1mm and longer than 80 milliseconds following the J-point. This measure has a false positive rate of 15-20% (which is slightly higher in women than men) and a false negative rate of 20-30%.
- ST depression may be associated with hypokalemia or digitalis toxicity.

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**ST Elevation and non-ST Elevation MIs**

- When myocardial blood supply is abruptly reduced or cut off to a region of the heart, a sequence of injurious events occur beginning with *ischemia* (inadequate tissue perfusion), followed by *necrosis* (infarction), and eventual *fibrosis* (scarring) if the blood supply isn’t restored in an appropriate period of time.
- The ECG changes over time with each of these events...
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**ECG Changes**

Ways the ECG can change include:
- ST elevation & depression
- Appearance of pathologic Q-waves
- T-waves: peaked, flattened, inverted

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**ECG Changes & the Evolving MI**

There are two distinct patterns of ECG change depending if the infarction is:
- ST Elevation (Transmural or Q-wave), or
- Non-ST Elevation (Subendocardial or non-Q-wave)

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**ST Elevation Infarction**

The ECG changes seen with a ST elevation infarction are:
- Before injury: Normal ECG
- Ischemia: ST depression, peaked T-waves, then T-wave inversion
- Infarction: ST elevation & appearance of Q-waves
- Fibrosis: ST segments and T-waves return to normal, but Q-waves may persist
**ST Elevation Infarction**

Here's a diagram depicting an evolving infarction:

A. Normal ECG prior to MI

B. Ischemia from coronary artery occlusion results in ST depression (not shown) and peaked T-waves

C. Infarction from ongoing ischemia results in marked ST elevation

D/E. Ongoing infarction with appearance of pathologic Q-waves and T-wave inversion

F. Fibrosis (months later) with persistent Q-waves, but normal ST segment and T-waves

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**EKG of an inferior MI:**

Look at the inferior leads (II, III, aVF).

What ECG changes do you see?
Non-ST Elevation Infarction

EKG of an inferior MI later in time:

ST elevation, Q-waves and T-wave inversion

Non-ST Elevation Infarction

The ECG changes seen with a non-ST elevation infarction are:

Before injury: Normal ECG
Ischemia: ST depression & T-wave inversion
Infarction: ST depression & T-wave inversion
Fibrosis: ST returns to baseline, but T-wave inversion persists

Non-ST Elevation Infarction

ECG of an evolving non-ST elevation MI:

Note the ST depression and T-wave inversion in leads V2-V6.

Non-ST Elevation Infarction

ECG of an evolving non-ST elevation MI:

Note the ST depression and T-wave inversion in leads V2-V6.
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**QT Interval**

- QT interval represents electrical depolarization and repolarization of the left and right ventricles.
- Lengthened QT interval is a biomarker for ventricular tachyarrhythmias like torsades de pointes and a risk factor for sudden death.
- QT interval is dependent on the heart rate (the faster the heart rate the shorter the QT interval) and may be adjusted to improve the detection of patients at increased risk of ventricular arrhythmia.
- Causes:
  - Drugs (Na channel blockers)
  - Hypocalcemia, hypomagnesemia, hypokalemia
  - Hypothermia
  - AMI
  - Congenital
  - Increased ICP

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**Normal Sinus Rhythm**

Implies normal sequence of conduction, originating in the sinus node and proceeding to the ventricles via the AV node and His-Purkinje system.

**EKG Characteristics:**
- Regular narrow-complex rhythm
- Rate 60-100 bpm
- Each QRS complex is preceded by a P wave
- P wave is upright in lead II & downgoing in lead aVR

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**Sinus Bradycardia**

- HR< 60 bpm; every QRS narrow, preceded by p wave
- Can be normal in well-conditioned athletes
- HR can be<50 bpm in children, young adults during sleep, with up to 2 sec pauses
Sinus bradycardia--etiologies

- Normal aging
- 15-25% Acute MI, esp. affecting inferior wall
- Hypothyroidism, infiltrative diseases (sarcoid, amyloid)
- Hypothermia, hypokalemia
- SLE, collagen vasc diseases
- Situational: micturation, coughing
- Drugs: beta-blockers, digitalis, calcium channel blockers, amiodarone, cimetidine, lithium

Sinus bradycardia--treatment

- No treatment if asymptomatic
- Sxs include chest pain (from coronary hypoperfusion), syncope, dizziness
- Office: Evaluate medicine regimen—stop any drugs
- Bradycardia associated with MI will often resolve as MI is resolving
- ER: Atropine if hemodynamic compromise, syncope, chest pain
- Pacing

Sinus tachycardia

- HR > 100 bpm, regular
- Often difficult to distinguish p and t waves
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Sinus tachycardia--etiologies

- Fever
- Hyperthyroidism
- Effective volume depletion
- Anxiety
- Pheochromocytoma
- Sepsis
- Anemia
- Exposure to stimulants (nicotine, caffeine) or illicit drugs
- Hypotension and shock
- Pulmonary embolism
- Acute coronary ischemia and myocardial infarction
- Heart failure
- Chronic pulmonary disease
- Hypoxia
- Fever
- Hyperthyroidism
- Effective volume depletion
- Anxiety
- Pheochromocytoma
- Sepsis
- Anemia
- Exposure to stimulants (nicotine, caffeine) or illicit drugs
- Hypotension and shock
- Pulmonary embolism
- Acute coronary ischemia and myocardial infarction
- Heart failure
- Chronic pulmonary disease
- Hypoxia

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Sinus Tachycardia--treatment

- Office: evaluate/treat potential etiology: check TSH, CBC, optimize CHF or COPD regimen, evaluate recent OTC drugs
- Verify it is sinus rhythm
- If no etiology is found and is bothersome to patients, can treat with beta-blocker

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Sick Sinus Syndrome

- Bradycardia
- Sinus bradycardia (rate of ~43 bpm) with a sinus pause
- Often result of tachy-brady syndrome: where a burst of atrial tachycardia (such as afib) is then followed by a long, symptomatic sinus pause/arrest, with no breakthrough junctional rhythm.
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Sick Sinus Syndrome—etiology

- Often due to sinus node fibrosis, arterial atherosclerosis (ischemia to node), inflammation (Rheumatic fever, amyloid, sarcoid)
- Occurs in congenital and acquired heart disease and after surgery
- Hypothyroidism, hypothermia
- Drugs: digitalis, lithium, cimetidine, methyldopa, reserpine, clonidine, amiodarone
- Most patients are elderly, may or may not have symptoms

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Sick sinus syndrome—treatment

- Address and treat cardiac conditions
- Review med list, TSH
- Pacemaker for most is required

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Paroxysmal Supraventricular Tachycardia

- Refers to supraventricular tachycardia other than afib, aflutter and MAT
- Occurs in 35 per 100,000 person-years
- Usually due to reentry—AVNRT or AVRT
Supraventricular Tachycardia

- Heart rate >150bpm
- Supraventricular means "above the ventricles," in other words, originating from the atria, the upper chambers of the heart.
- The heart rate is sped up by an abnormal electrical impulse starting in the atria.
- May also be a side effect of medications such as digitalis, asthma medications, or cold remedies.
- In some cases, the cause of supraventricular tachycardia is unknown.

Supraventricular Tachycardia

- Can be found in healthy young children, in adolescents, and in people with underlying heart disease.
- Most people who experience it live a normal life without restrictions.
- Often occurs in episodes with stretches of normal rhythm in between.
  - This is usually referred to as paroxysmal supraventricular tachycardia (often abbreviated PSVT).
- Supraventricular tachycardia also may be chronic (ongoing, long term).

PSVT

- Initial eval: Is the patient stable?
- Determine quickly if sinus rhythm
- If not sinus and unstable, cardioversion
- Unstable sinus tachycardia—IV beta-blocker, and treat cause

   - Sxs of instability would include: chest pain, decreased consciousness, short of breath, shock, hypotension—unstable sxs require shock
PSVT

- If stable, determine whether regular rhythm (sinus or PSVT) vs irregular (afib/flutter, MAT)?
- If regular, determine whether p waves are present, if can’t see—administer adenosine (6mg, can give 2 doses) or other vagal maneuvers.

PSVT

- CSM or adenosine commonly terminate the arrhythmia
- Can also use CCB or beta blockers to terminate
- Counsel to avoid triggers, caffeine, Etoh, pseudoephedrine, stress
Supraventricular Tachycardia

• Treatment:
  – Vagal Maneuvers:
    • Hold breath for a few seconds
    • Dip face in cold water
    • Cough
    • Tense stomach muscles as if bearing down to have a bowel movement

• Medications:
  – Adenosine (Adenocard)
    • a short-acting medication that decreases heart rate.
    • ½ life is less than 10 seconds
    • Given IV, 6mg fast push followed by NS flush.
    • May be repeated at 12mg.
    • Adenosine successfully stops paroxysmal supraventricular tachycardia (PSVT) in more than 90% of cases.

• If adenosine is unsuccessful:
  – Could consider cardioversion

• Medications:
  – To prevent recurrence of SVT
    • Beta Blockers (Metoprolol)
    • Calcium channel blockers (Diltiazem)
    • Digoxin
Atrial Fibrillation

- Micro reentrant circuit
- AV node is bombarded with rates greater than 400 bpm for atrial foci.
- AV works hard to block impulses
- Ventricular rate is irregularly irregular
  - Between 110-170 bpm
  - Can be a slow rate as well
  - Sign of significant underlying conduction disorder
- No distinguishable P waves on EKG
- Auscultated rate is more accurate.
- Not all ventricular responses produce a palpable pulse rate.
Atrial Fibrillation

- Can be precipitated by:
  - Valvular disease IHD and CHF
  - Pericarditis
  - Thyrotoxicosis
  - Pulmonary embolism
  - Pneumonia
  - Acute alcohol ingestion
  - Post op cardiac surgery
  - Post op thoracotomy
  - Sleep apnea

Atrial Fibrillation

- Results in:
  - Poor cardiac output
  - Heart failure
  - Embolic stroke due to pooling of blood in the atria.

Atrial Fibrillation

- 3 Treatment Goals:
  1. Prevention of thromboembolic complications
     - 5-6% risk of embolic stroke
     - Stasis of blood in atria
     - Warfarin (Coumadin)
       - INR 2.5 – 3.0
     - Pradaxa – new AC/ no INR’s or dietary interactions
  2. Control of ventricular rate
     - Outpatient management consists of rate control and restoration of sinus rhythm
       - Rate Control
         - Oral Diltiazem (Cardizem)
       - Beta Blockers
       - Digoxin
Atrial Fibrillation

• (3) Restoration of NSR
  – Class 1A antiarrhythmics
    – Pronestyl (Procainamide)
    – Quinidine (Cardioquin)
  – Class III antiarrhythmics
    – Sotalol (Betapace)
    – Ibutilide (Corvert)
    – Amiodarone
  – Class IC antiarrhythmics
    – These agents are used only in patients with structurally normal hearts (ie, absence of coronary artery disease or cardiomyopathy).
    – Propafenone (Rythmol)
    – Flecainide (Tambocor)

• Cardioversion
  – Less than 48-72 hours of a-fib
    – Safe to cardiovert without anticoags.
  – If duration unknown
    – Rate control, try to restore sinus rhythm
    – Anticoagulate x 3 weeks then cardiovert
    – Anticoagulants x 3 weeks after successful cardioversion
    – Can use TEE to see if clots exist

• If rate can’t be controlled:
  – Atrial fibrillation ablation
  – AV node ablation in extreme cases which would require permanent pacemaker placement
Atrial Flutter

- Macro reentrant circuit
- Atrial rate 250-350
- Ventricular rate 150
- AV node blocks at a 2:1, 3:1, 4:1 rate
- Can be a slower ventricular rate
- Regularly irregular
- Classic sawtooth pattern on EKG

Atrial Flutter

- Occurs in patient with or without structural heart disease.
- Atrial flutter almost always occurs in diseased hearts. It frequently precipitates CHF
- May be precipitated by:
  - Thyrotoxicosis
  - Pericarditis
  - Alcohol ingestion
- The treatment depends on the level of hemodynamic compromise.
Atrial Flutter

- Rate is harder to control
- Thrombolytic event risk is somewhat lower than a-fib

- Treatment:
  - Class 1A antiarrhythmics are used to convert to sinus rhythm
    - Pronestyl (Procainamide)
  - Ventricular rate controlled with:
    - Beta Blockers
    - Calcium channel blockers
    - Digoxin

AV node disturbances

- Junctional Escape Rhythm
- Accelerated Junctional Rhythm
- Junctional Tachycardia
- Wolfe-Parkinson-White Syndrome

Junctional Escape, Accelerated 
Junctional Rhythm

- Junctional escape rhythm
  - 40-60 bpm
- Accelerated Junctional Rhythm
  - 60-100 bpm
  - Common in patients with inferior MI
  - Digoxin Toxicity
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Junctional Escape, Accelerated Junctional Rhythm

- EKG shows:
  - Narrow complex QRS
  - Retrograde P wave
    - Inverted P with very short PR interval
    - Or P wave immediately after QRS
    - Sometimes there is no P wave
  - Specific treatment is usually not required

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Junctional Escape Rhythm

![EKG](image)

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Junctional Tachycardia

- Junctional tachycardia
  - 150 – 250 bpm
  - Occurs more commonly in women
  - May occur in absence of heart disease
  - Usually initiated by a PAC
Junctional Tachycardia Rhythms

- **Treatment:**
  - Vagal maneuvers
  - Adenosine
    - Drug of choice
    - Terminates 95% of the cases
  - Long term treatment
    - Beta Blockers
    - Calcium Channel Blockers
    - Class 1A, 1C and III antiarythmics for resistant cases

Wolfe-Parkinson White Syndrome

- Delta Wave
**Wolfe-Parkinson White Syndrome**

- Form of supraventricular tachycardia but involves an accessory pathway that bypasses the AV node.
- Can be referred to as an AV reciprocating arrhythmia.
- Along with the normal conduction pathway, there are extra pathways called accessory pathways. They look like normal heart muscle, but they may:
  - Conduct impulses faster than normal
  - Conduct impulses in both directions
- Impulses travel through the extra pathway (short cut) as well as the normal AV-HIS Purkinje system.
- Impulses can travel around the heart very quickly, in a circular pattern, causing the heart to beat unusually fast. This is called re-entry tachycardia.
- The greatest concern for people with WPW is the possibility of having atrial fibrillation with a fast ventricular response that worsens to ventricular fibrillation, a life-threatening arrhythmia.

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**Wolfe-Parkinson White Syndrome**

- Congenital defect
- Can occur at any age
- One of the most common causes of fast arrhythmia in infants and children.
- Highest incidence occurs between the ages of 30 and 40 years old
- Men have a higher incidence women do, and there is a higher incidence of multiple accessory pathways in men.

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**Wolfe-Parkinson White Syndrome**

- EKG characteristics are seen only after a rhythm conversion from PSVT to NSR.
- PR interval is shorter
- Upstroke of the QRS wave is slurred; this is known as a delta wave.
- 12 lead is essential as delta wave may not show up in all leads.
Wolfe-Parkinson White Syndrome

- Treatment depends on the type of arrhythmias, the frequency and the associated symptoms.
- Observation
  - If have no symptoms, treatment may not be required
  - Regular follow-up visits
- Medications
  - Beta blockers
  - Calcium Channel Blockers
  - Procainamide

Because everyone is different, it may take trials of several medications and doses to find the one that works best.

Ablation of the accessory pathway is treatment of choice for symptomatic patients.

Heart blocks

- 1st degree AV Block
- 2nd degree AV Block, Mobitz Type I (Wenkebach)
- 2nd degree AV Block, Mobitz Type II
- 3rd degree heart block (AV dissociation)
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1st degree AV block

- This is the most common conduction disturbance.
- Occurs in both healthy and diseased hearts.
- First degree AV block can be due to:
  - inferior MI
  - digitalis toxicity
  - hyperkalemia
  - increased vagal tone
  - acute rheumatic fever
  - myocarditis

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1st degree AV block

- Interventions include treating the underlying cause.
- Usually don’t need any other treatment.
- Observe for progression to a more advanced AV block.

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1st degree AV Block

- EKG –
  - PR interval greater than 0.20
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**2nd degree AV Block-Mobitz Type I (Wenckebach)**

- Second degree AV block type I occurs in the AV node above the Bundle of His.
- It is often transient and may be due to acute inferior MI or digitalis toxicity.
- Treatment is usually not indicated as this rhythm usually produces no symptoms.

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**2nd degree AV Block-Mobitz Type I (Wenckebach)**

- **EKG:**
  - Rate may be variable
  - PR interval gets progressively longer until a QRS is dropped (or blocked)
- Usually no treatment is needed
- Observe for progression to more severe block.

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**2nd degree AV Block-Mobitz Type I (Wenckebach)**

- EKG showing second degree AV block-Mobitz Type I.
2nd degree AV Block-Mobitz Type 11

• This block usually occurs below the Bundle of His and may progress into a higher degree block.

• Can occur after an acute anterior MI due to damage in the bifurcation or the bundle branches.

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2nd degree AV Block-Mobitz Type 11

• Rate: variable

• P-wave – normal

• QRS: usually widened because this is usually associated with a bundle branch block.

• PR: may be normal until dropped QRS

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2nd degree AV Block-Mobitz Type 11

• It is more serious than the type I block.

• Treatment is usually artificial pacing, via external pacer or temporary pacer wire insertion.

• May need permanent pacer.
2nd degree AV Block-Mobitz Type 11

3rd degree heart block
AV dissociation

- Complete block of the atrial impulses occurs at the A-V junction, common bundle or bilateral bundle branches.
- Another pacemaker distal to the block takes over in order to activate the ventricles or ventricular standstill will occur.
- Atrial and ventricular activities are unrelated due to the complete blocking of the atrial impulses to the ventricles

3rd degree heart block
AV dissociation

- May be caused by:
  - digitalis toxicity
  - acute infection
  - MI
  - degeneration of the conductive tissue
    - Due to MI

- Due to MI
3rd degree heart block
AV dissociation

- EKG:
  - Atrial rate is usually normal
  - Ventricular rate is usually less than 70/bpm
  - Atrial rate is always faster than the ventricular rate.
  - P waves: normal with constant P-P intervals, but not "married" to the QRS complexes.
  - QRS: may be normal or widened depending on where the escape pacemaker is located in the conduction system.

Treatment modalities include:

- external pacing and atropine for acute, symptomatic episodes
- permanent pacing for chronic complete heart block

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3rd degree heart block
AV dissociation

- EKG:
  - Atrial rate is usually normal
  - Ventricular rate is usually less than 70/bpm
  - Atrial rate is always faster than the ventricular rate.
  - P waves: normal with constant P-P intervals, but not "married" to the QRS complexes.
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- external pacing and atropine for acute, symptomatic episodes
- permanent pacing for chronic complete heart block

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3rd degree heart block
AV dissociation

- Treatment modalities include:
  - external pacing and atropine for acute, symptomatic episodes
  - permanent pacing for chronic complete heart block
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**Ventricular Dysrhythmias**

- Premature Ventricular Contractions
- Ventricular Tachycardia
- Ventricular Fibrillation
- Asystole
- Idioventricular Rhythm
- PEA

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**Premature Ventricular Contractions (PVC's)**

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**Premature Ventricular Contractions (PVC's)**

- **Causes:**
  - PVCs can occur in healthy hearts
  - Increasing circulating catecholamines
  - Occurs in diseased hearts
  - Hypokalemia
  - Low magnesium level
  - Digitalis toxicity
Premature Ventricular Contractions (PVC's)

- **EKG:**
  - Rate: variable
  - P wave – usually obscured by the QRS with the PVC.
  - QRS: Wide 0.12, morphology is bizarre.
  - The impulse originates below the branching portion of the Bundle of His.
  - Full compensatory pause is characteristic.

- **Rhythm:** looks irregular due to the premature beat.

- PVC's may occur in singles, couplets or triplets; or in bigeminy, trigeminy or quadrigeminy.

- **Bigeminal PVC’s**
Premature Ventricular Contractions

- Couplets

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Premature Ventricular Contractions

- Trigeminal

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Premature Ventricular Contractions

- Multifocal PVCs
Premature Ventricular Contractions

• R on T phenomenon

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Premature Ventricular Contractions

• Treatment is required if they are:
  – associated with an acute MI,
  – occur as couplets, bigeminy or trigeminy continuously
  – are multifocal
  – are frequent (>6 PVC's per minute)

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Premature Ventricular Contractions

• Treatment:
  – Lidocaine - Class 1B antiarrhythmic
  – Procainamide (Pronestyl) - Class 1A antiarrhythmic
  – Amiodarone (Cordorone) – Class III antiarrhythmic
  – Replace Magnesium
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**Ventricular Tachycardia**

- Triggers of VT include ischemia and electrolyte abnormalities.
- **Hypokalemia** is the most important arrhythmia trigger clinically, followed by hypomagnesemia.
- Hyperkalemia also may predispose to VT and VF, particularly in patients with structural heart disease.

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**Ventricular Tachycardia**

- **Causes**
  - MI
  - Irritable ventricle
  - Congenital Heart Defect
  - Dilated cardiomyopathy
  - Hypertrophic cardiomyopathy

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**Ventricular Tachycardia**

- Can have a pulse or be pulseless
- **Treatment:**
  - Pulse
    - Cardioversion
    - Antiarrhythmics to prevent recurrence.
      - Amiodarone
  - Pulseless
    - Defibrillation
    - Antiarrhythmics to prevent recurrence.
      - Amiodarone
• Short runs of V-Tach
Torsades de Pointes

- The term Torsades de Pointes means “twisting about the points.”
- Paroxysmal – starting and stopping suddenly
- Hallmark of this rhythm is the upward and downward deflection of the QRS complexes around the baseline.
- Consider it V-tach if it doesn’t respond to antiarythmic therapy or treatments

Torsades de Pointes

- Caused by:
  - drugs which lengthen the QT interval such as quinidine
  - electrolyte imbalances, particularly hypokalemia
  - myocardial ischemia

Torsades de Pointes

- Treatment:
  - Synchronized cardioversion is indicated when the patient is unstable.
  - IV magnesium
  - IV Potassium to correct an electrolyte imbalance
  - Overdrive pacing
Idioventricular Rhythm

• Absent P wave
  Widened QRS > 0.12 sec.
  Also called “dying heart” rhythm
  Pacemaker will most likely be needed to re-establish a normal heart rate.

• Causes:
  – Myocardial Infarction
  – Pacemaker Failure
  – Metabolic imbalance
  – Myocardial Ischemia
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**Idioventricular Rhythm**

- Treatment goals include measures to improve cardiac output and establish a normal rhythm and rate.
- Options include:
  - Atropine
  - Pacing
- Caution: Suppressing the ventricular rhythm is contraindicated because that rhythm protects the heart from complete standstill.

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**Idioventricular Rhythm**

- Asystole in the presence of acute MI and CAD is frequently fatal.
- Complete cessation of any electrical or mechanical activity.
- Interventions include:
  - CPR, 100% oxygen
  - IV
  - intubation
  - transcutaneous pacing
  - epinephrine 1.0 mg., IV push, q3-5 minutes
  - atropine

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**Asystole**

- Asystole in the presence of acute MI and CAD is frequently fatal.
- Complete cessation of any electrical or mechanical activity.
- Interventions include:
  - CPR, 100% oxygen
  - IV
  - intubation
  - transcutaneous pacing
  - epinephrine 1.0 mg., IV push, q3-5 minutes
  - atropine
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Asystole

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Pulseless Electrical Activity (PEA)
Electromechanical dissociation

• There is electrical activity, but no mechanical response.
• What is seen on the EKG is electrical activity.
• NO pulse

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Pulseless Electrical Activity (PEA)
Electromechanical dissociation

• Look for underlying causes:
  – MI
  – Hypoxia
  – Hypovolemia
  – Hypoglycemia
  – Acidosis
  – Hypothermia
  – Trauma
  – Hypo/Hyperkalemia
  – Cardiac Tamponade
  – Tension Pneumothorax
  – Toxins
Pulseless Electrical Activity (PEA)
Electromechanical dissociation

• Treatment:
  – Correct underlying cause
  – Epinephrine – 1:10,000
  – Atropine
  – CPR