EKG Workshop
Texas Academy of Physician Assistants
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David J. Klocko is receiving a stipend from TAPA to do this workshop.

Objectives
- Review basic electrophysiology.
- Recognize common cardiac dysrhythmias.
- Recognize common conductive disorders.
- Review axis, bundle branch block, chamber enlargement and hypertrophy.
- Recognize common ECG changes associated with myocardial ischemia and infarction.
### Systematic Evaluation of the 12-lead EKG – Our ultimate goal!

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### Intervals & Segments

- **PR interval (PRI):** start of atrial depolarization to start of ventricular depolarization
  - **Normal:** 0.12 – 0.20 sec

- **QRS complex:** depolarization of ventricles
  - **Normal:** 0.04 – 0.11 sec
  - When QRS >0.12 sec, think about an interventricular conduction delay
Intervals & Segments

- **ST segment**: end ventricular depolarization to start of repolarization

- **QT interval (QTI)**: both ventricular depolarization & repolarization
  - Normal: 0.3 – 0.4 sec; varies with heartrate
  - Faster heartrates….shorter QTIs; slower heartrates ….longer QTIs

Heart Rate

- For regular rhythms, measure the interval between complexes in large boxes
  - 300-150-100-75-60-50-43-37-33

- Alternate method – count # R waves in 6 second strip x 10

Sinus Rhythm

- “Normal” rhythm of the heart
- SA node → AV node → Bundle of His → Purkinje fibers
- P-QRS = 1:1, regular
- Normal sinus rhythm (NSR): 60-100
  - Sinus bradycardia: < 60
  - Sinus tachycardia: >100
  - Sinus arrhythmia:
    - P-QRS = 1:1, irregular
Sinus Arrhythmia

- **Normal variant**: changes w/ respiration; seen in children & young adults
  - ↑HR with inspiration; slight ↓ with expiration
- Nonrespiratory is rare; seen w/ heart disease
- P:QRS = 1:1; rate irregular

Premature Atrial Contractions (PACs)

- Early beat from ectopic atrial pacemaker
  - Different P wave & QRS morphology
- P:QRS = 1:1
- PRI usually normal
- May be uniform, multiform, pairs or short runs
- May be normal; also seen with stress, ↑ caffeine/tobacco/alcohol, hypoxemia, valvular heart disease & CAD

Premature Atrial Contractions
Supraventricular Tachycardia

- Generic term for any tachyarrhythmic rhythm generated above the ventricles
- May include:
  - Atrial tachycardia
  - Multifocal atrial tachycardia
  - Atrial flutter
  - Atrial fibrillation
- May also see designated as paroxysmal
  - For example – PSVT

Supraventricular Tachycardia (SVT)

- Ectopic atrial focus overrides the SA node
- Rhythm is fast but regular (150-250)
- P waves have different configuration, often hidden
  - While P:QRS is 1:1, often hard to appreciate
- Same etiologies as PACs
Atrial Tachycardia

- Able to identify P waves… and determine PRI and P:QRS
  - P waves have the same morphology
- Rate 150-250; regular

Multifocal Atrial Tachycardia (MAT)

- Rate 150-250; irregular
- P waves will have different morphology
- PRIs may be variable

Atrial Flutter

- Atrial rate between 250-350/minute
- Flutter waves seen, “sawtooth” pattern
  - P:QRS and PRI – not measurable
- Variable ventricular response:
  - 2:1, 3:1, 4:1, variable
- Rare to see in normal hearts
Atrial Fibrillation

- Atrial activity > 350/minute
- Chronic asynchronous firing from multiple areas causes suppression of SA node
- No discernible P wave activity; wavy baseline
- AV node will block some impulses; ventricular response with vary (irregularly irregular)
Junctional Rhythm

- Absent or inverted P waves in Lead II
  - If P wave present, can determine PRI & P:QRS ratio

- Origin at AV node: normal escape rhythm (40 – 60)
  - If rate 60-100…Accelerated Junctional Rhythm
  - If rate 100-180…Junctional Tachycardia

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- If P wave present, can determine PRI & P:QRS ratio
Premature Ventricular Contractions (PVCs)

- Ectopic beat from ventricular focus
  - Wide, bizarre QRS complex
  - No P waves seen prior to QRS complex
- Sign of myocardial irritability
  - Watch for ↑ frequency, pairs, runs, “R on T” phenomenon
- May be uniform, multiform, couplets, triplets, bi-/tri-/quadrigeminy

Unifocal v. Multifocal PVCs

Ventricular Bigeminy & Trigeminy
Ventricular Tachycardia (VT)

- Serious dysrhythmia – associated with underlying cardiac disease
- Usually regular, may be irregular
- Rate: 100-250
- No P waves….PRI or P:QRS
- QRS complexes widened
Torsades des Pointes

- Polymorphic ventricular tachycardia
- May be associated with prolonged QT interval
  - May be drug-induced or associated with electrolyte abnormalities

Ventricular Fibrillation

- Sign of extreme myocardial irritability
- Multiple irritable foci in the ventricles
- Chaotic rhythm; irregular
- Rate indeterminate

Idioventricular Rhythm

- Ventricular escape rhythm – last back-up system
- Usually regular (20 – 40)
  - If rate 40 – 100...accelerated idioventricular rhythm v. “slow V-tach”
  - No P waves; no PRI or P:QRS ratio
  - QRS complex is wide & bizarre
Idioventricular Rhythms

- Wolff Parkinson White (WPW)
  - AV node normally holds up conduction 0.1 sec for full atrial depolarization
  - In WPW – an alternate pathway (Bundle of Kent) bypasses the delay at the AV node and allows shorter conduction and prematurely activates the ventricles
    - Looks like a short PRI <0.12

Wolff Parkinson White

- Early ventricular depolarization reflected in delta wave
- Widened QRS (>0.10) reflective of fusion beat
  - Depolarization from normal & abnormal pathways
- WPW can predispose to tachydysrhythmias (Paroxysmal SVT & Afib)
  - Afib may potentiate VF since alternate pathway bypasses AV node which usually blocks atrial impulses
Wolff Parkinson White

First Degree AV Block
- Impulse from SA node delayed conducting to ventricles at AV node.
- PR interval on ECG >0.20 seconds
- Every P wave followed by normal ventricular impulse (QRS complex)
  - Rhythm is regular; P:QRS = 1:1
  - Seen with inferior MI, also drug effect
  - Usually asymptomatic; no treatment

First Degree Heart Block
Second Degree AV Block

- Failure of some sinus impulses to be conducted to the ventricles
  - More P waves than QRS complexes
- Sinus impulses are conducted normally… just see increased difficulty getting through the conduction system
- Level of block (High Grade AV block)
  - 2° AVB, Type I – AV node
  - 2° AVB, Type II – bundle branches

Second Degree AV Block

- 2nd Degree Heart Block, Wenckebach (aka Type I, Mobitz I)
  - Regularly Irregular
    - P-P interval is regular; ventricular response irregular (variable R-R interval)
    - More P waves than QRS complexes
  - Progressively prolonged PRI followed by nonconducted P wave (cyclic pattern)

2° Heart Block, Type I (Wenckebach)
2° Heart Block, Type II (Mobitz II)

- PRI remains constant, but periodic non-conducted P wave
  - PRI may be normal or prolonged
- P:P will be regular; ventricular response (R-R) regular unless conduction ratio varies
  - More P waves than QRS complexes
- More serious than Wenckebach; may progress to third degree AV block

Second Degree Heart Block:
2:1 Conduction
Third Degree Heart Block

- Complete dissociation between the atria & ventricles
  - Atria responsive to SA node
    - P:P interval will be constant but the PRI varies
    - More P waves than QRS complexes

- Ventricles responsive to ectopic pacer
  - If back-up pacer is junctional:
    - Rate is 40-60; QRS < 0.12
  - If back-up pacer is ventricular:
    - Rate is 20-40; QRS > 0.12
Systematic Evaluation of the 12-lead EKG

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**Red flags:**
- Lots of jiggly bits.
- A lumpy bit on the jiggly bit.
- Big tall lumps.
- No humps, lumps or jiggles.

**AND**
- Squiggly stuff you have no idea about but the patient looks bad.
Axis Determination

- Direction of mean electrical vector
  - Represents average direction of current flow
- Quick assessment: look to see whether QRS complex positive or negative in Leads I & AVF

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<tr>
<td>Normal</td>
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<tr>
<td>LAD</td>
<td>Positive</td>
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![Diagram of electrocardiogram axis determination]
Left Axis Deviation ***

I- UP
AvF- Down

Normal Axis

Lead I- Up
Lead AvF- Up

Right Axis Deviation

Lead I- Down
Lead AvF- Up
Right Bundle Branch Block

- Interruption of cardiac impulse at right bundle branch causing left ventricle to be depolarized slightly before right ventricle
- Widened QRS (>0.12 sec.) with RR configuration seen in V1 and V2
- Biphasic QRS with broad S wave also seen in lead I

Bundle Branch Block

- Rule- ST Pattern of a Left Bundle Branch block may obscure or mimic ST changes assoc. with ischemia or infarct!

- Example- 75 yo male c/o vague chest pressure for 2 hours, EKG shows a L BBB. Your EKG is considered non-diagnostic if it is not showing acute changes. Treat based on history!
Left Bundle Branch Block

- QRS > 0.12 sec
- Broad/notched R waves in V5 and 6, lead I and AVL with ST segment depression and T wave inversions
- May see LAD

Atrial Enlargement

- Normal P Waves (use leads II and V1)
  - Amplitude: 0.5 – 2.5 mm
  - Duration: 0.06 – 0.10 sec
  - Morphology: Usually rounded and upright
    - May be biphasic in V1
- Atrial Enlargement—the Basics:
  - RAE: Amplitude > 2.5 mm (2 1/2 blocks)
  - LAE: Duration > 0.10 sec (2 1/2 blocks)
  - Biatrial Enlargement: increased amplitude and duration.
Right Atrial Enlargement

- Peaked 2.5mm

Left Atrial Enlargement

- Broad P
- Biphasic

Right Ventricular Hypertrophy

- Often associated with pulmonary HTN or pulmonic stenosis
- May also see RAD
- Criteria:
  - Right axis deviation (> +90 degrees)
  - R wave > S wave in V1
    - R wave is usually > 7 mm tall
  - S wave > R wave in V6
Right Ventricular Hypertrophy

- Common causes are hypertension and valvular heart disease
- Basic criteria:
  - Sum of the deepest S in V1 or V2 plus the tallest R in V5 or V6 is > 35mm
  - R in AVL > 11mm
  - Statler’s criteria – QRS off the paper

Left Ventricular Hypertrophy

- Common causes are hypertension and valvular heart disease
- Basic criteria:
  - Sum of the deepest S in V1 or V2 plus the tallest R in V5 or V6 is > 35mm
  - R in AVL > 11mm
  - Statler’s criteria – QRS off the paper
LVH Rule of 35

- If deepest S wave in V1 or V2 + tallest R wave in V5 or V6 = 35 mm
- AND
- The patient is over 35 yo
- You meet electrical criteria for LVH

Evolution of MI

- **Ischemia** – ST segment & T wave changes
  - T wave inversion due to delayed repolarization
  - ST segment depression
- **Injury** - ST segment changes
  - ST Elevation in leads facing injury, due to incomplete depolarization
  - Look for ST elevation >1-2mm in 2 or more contiguous leads
- **Infarct** – pathologic Q waves
  - Enlarging or new in appearance
  - Infarcted tissue is electrically silent

Evolution of an MI

- Reversible
- Irreversible
- Ischemia  Injury  Infarct
Localizing MIs

- **Anterior MI**: Changes in precordial leads (V1-V4); reciprocal changes in inferior leads
  - Septal (V1-2)
- **Lateral MI**: Changes in lead I, aVL, V5 & 6; reciprocal changes in inferior leads
- **Inferior MI**: Changes in leads II, III, and aVF; reciprocal changes in anterolateral leads
- **Posterior MI**: Reciprocal changes in V1 & 2, tall R waves with ST depression in these leads

“RIP” & “LAL”

RCA = Inferior + Posterior
LCA = Anterior + Lateral

Key Cardiac Regions on ECG
Anterior Injury

ST Elevation V1-V4 with Q wave development

Anterolateral Injury

Look in V1-V4
Posterior Injury (often with Inferior wall)

Look in V1, V2, V3

Horizontal ST depression
Tall, broad R waves (>30ms)

Acute Inferoposterior Injury

Posterior Chest Leads
Posterior Leads (left scapula)

NSTEMI -- Non-Q Wave MI (Ant/Lat ST-Changes)

Wellen’s T waves
**Wellen’s T-waves**

- Prior history of chest pain
- During chest pain: EKG is normal or with mild ST elevation or depression, or with terminal negative deflection of the T wave in V1 and V2
- Cardiac enzymes are normal or mildly elevated
- No pathologic precordial Q-waves
- No loss of precordial R-waves
- **Deeply inverted or biphasic T-waves in V2 and V3, possibly V1, V4, V5 and/or V6 when pain free**

High grade stenosis of the proximal Left Anterior Descending. **Anterior MI Possible very soon !!!**

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**Electrolyte Disturbances**

- **Hyperkalemia:**
  - As potassium levels rise... see **tall, peaked T waves** across the entire 12-lead
  - Use your clinical picture to help rule out an acute MI
    - Helpful hint – MI changes are focal; hyperkalemia changes are diffuse
  - With **increasing** potassium levels... the PRI prolongs, P waves flatten and then disappear
  - Eventually the QRS widens until it merges with the T wave forming a sine pattern

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**Hyperkalemia**
Hypokalemia

- ECG changes:
  - Prolongation of QT interval
  - ST segment depression
  - Flattening or inversion of the T wave
  - Increased prominence of a U wave

Prolongation of QT Interval

- Drugs that can prolong the QT interval and put patient at risk for torsades include:
  - Anti-arrhythmics:
    - Quinidine, procainamide (Procanbid), disopyramide (Norpace), amiodarone (Cordarone), & sotalol (Betapace)
  - TCAs
  - Phenothiazines
  - Erythromycin

Prolonged QT Scan

- QT interval greater the ½ the RR Interval
Prolonged QT

Initially see ST elevation associated with upright T waves
- ST changes are more diffuse…as opposed to localized changes with a MI

No Q waves seen

Pericarditis
Let's Practice !!!

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Resources

All images and EKGs were obtained with use of the Creative Commons license from these websites:
ECG Learning Center
https://ecg.utah.edu/img_index

Life in the Fastlane EKG
https://lifeinthefastlane.com