EKG Interpretation

Epidemiology of dysrhythmia

In 2003, dysrhythmias caused or contributed to 479,000 deaths. The conduction system is susceptible to damage by heart disease. Ischemia can cause tissues of the conduction system to be irritable or excitable (extra beats). Ischemia can also cause tissues to block electrical impulses. This effects hemodynamics and the hearts ability to perfuse the tissues. Note that some dysrhythmias are benign, but we're focusing today on the lethal ones.

Basic Electrophysiology

Electrophysiology is the study of the electrical properties of the heart. There is a pattern of electrical impulses through the conduction system of the heart, whereby electrical signals become mechanical events. However, sometimes the mechanical event does not happen leading to a dysrhythmia or an arrhythmia.

- dysrhythmia = change in rhythm
- arrhythmia = no rhythm

The SA node sends the impulse down the intranodal pathway to the AV node to the Bundle of His through the bundle branches to the Perkinje fibers. Think of the AV node as a "relay station", and the branches as a "freeway system." On the EKG we are watching the timing of the electrical impulses to see if it follows a pathway (freeway), or if it got off the freeway and took a "side street". If it took a side street, the timing is going to be slooowwwer...just like in real life!

Nursing Role

- Monitoring and identifying dysrhythmias
- Patient symptoms
  - Chest pain
  - Shortness of Breath
  - Hypotension
  - Altered mental status
- Consider the cause of the dysrhythmia
- Intervene appropriately for life threatening dysrhythmias

Intrinsic Rates of the Heart
SA Node: 60-100 beats per minute
AV Node: 40-60 beats per minute
Purkinje: 20-40 beats per minute

The 4 Properties of Cardiac Cells

1. Automaticity - this is the pacing function of the heart and is the role of the SA Node (preferred), the AV Node and the Purkinje fibers.
   The AV node is a relay station and it can block the SA node if it is going too fast, or block the SA completely if the AV is ischemic. The Purkinje fibers will take over if nothing else is working well...this is pretty bad news for your patient.
2. Excitability - this is the ability to respond to an electrical impulse and explains why the impulse can get off the freeway and take the side streets. All cardiac cells are excitable!
3. Conductivity - this is the ability to transmit the electrical impulse
4. Contractility - this is the ability of cardiac cells to shorten in response to an electrical stimulus. This is the mechanical event we mentioned earlier!

Some meds will increase contractility of the heart such as Digoxin, Dopamine, Dobutamine.

Cardiac Action Potential

Polarization = cell is at rest and ready for an impulse
Depolarization = reversal of electrical charge across cell membrane (this is the Na and K changing places)
Repolarization = recovery of the cell to its original polarized state (Na and K returning to their original positions). The cell is refractory during this time period. If a cell receives an impulse during this period, it gets irritable leading to a lethal rhythm and sudden cardiac death. No bueno! More on refractory periods below.

Refractory Periods...3 Stages

- Absolute refractory period: Cardiac cells will NOT respond to a stimulus AT ALL! No way Jose!
- Relative refractory period: This is a vulnerable period. Some cells have repolarized and the tissue may respond with a strong impulse.
- Supernormal period: Weaker than normal stimulus could cause depolarization. The cell is "hyper" during this time and it doesn't take much to set it off. Stimulation at this time often results in very fast, dangerous rhythms.

Cardiac Conduction System

- SA Node is the pacemaker for the heart. It is located on the upper posterior wall of the RA. It generates a stimuli at regular intervals (60-100 bpm), and it corresponds with the P WAVE on the ECG.
- Intranodal pathways (between SA and AV nodes)
- AV Node slows the impulse coming from the SA Node (recall that the AV is the "relay station"). The slowing of the SA node allows the atria time to contract and the ventricles to fill. The ventricles have to be nice and full in order to have optimal cardiac output! The AV can spontaneously generate an impulse btwn 40-60 bpm and this is usually an "escape mechanism" or "rescue mechanism". The AV node's impulse is portrayed as the PR interval on the EKG. We want to keep an eye on the length of this line!
- The Bundle of His is the "freeway system" of the heart. It bifurcates into the left and right branches and travels through the ventricles.
- The Purkinje fibers allow for rapid depolarization of the ventricle. It is seen as the QRS on the ECG. If left to its own devices, Purkinje would fire at 20-40 bpm...not very good!

ECG Waveforms

The full cardiac cycle consists of a P wave, a PR interval, a QRS complex, an ST segment, a T wave and an isoelectric line.

- **P wave** is the start of the cardiac cycle. It results from the electrical firing of the SA node/atrial depolarization. It is rounded and smooth in appearance, and has a positive deflection (it points up like a little hill). All P waves on the EKG should look the same. If they don't something is going on with the AV node.
- **The PR Interval** relates to the depolarization of the right and left atria, and the impulse delay through the AV junction (which is your relay station). This is a period of electrical silence and it establishes the isoelectric line. Your ST segment needs to be at the same level as this line!
  - It is measured from the beginning of the P wave to the beginning of the QRS complex. It's normal length is 0.12 to 0.20 seconds (3-5 little boxes).
  - If the PR interval is long this means there is a blockage in the AV node (most likely d/t ischemia)
- **The QRS Complex** is made up of 3 waveforms and these can vary dramatically depending on the view you are utilizing. It represents the depolarization of the ventricles.
  - Q wave is the FIRST downward deflection after the P wave...it can be pathological indicating damage to the heart muscle. The Q wave is usually not there unless the pt has had an acute MI in the past. Tiny ones are OK though.
  - R wave is the FIRST upward deflection in the QRS. This is always a good thing! On a 12-lead view, we are looking for R-wave progression...it should get taller and taller as time goes on. If pt has had an acute MI, the R wave will not progress normally b/c the Q gets in the way.
  - S wave is the downward deflection AFTER the R wave.
  - The QRS should be no wider than 3 little boxes (0.12 seconds) If it is wider, this means there is a blockage along the bundle and the impulse got off the freeway and took the side street.
- ST segment is an isoelectric line between the QRS and the T-wave. It should be on the same plane as the PR interval.
  - If it is elevated or depressed this is indicative of injury in the myocardium.
  - It begins at the end of the QRS and ends at the beginning of the T wave.
  - It represents early repolarization of the ventricle
- T Wave follows the ST segment and represents ventricular repolarization
  - It is usually rounded and deflected in the same direction as the QRS.
  - A negative T-wave following a positive QRS is suggestive of ischemia

The Boxes

Each little box signifies TIME
Each LITTLE box = 0.04 seconds
Each BIG box = 0.20 seconds

- Tall pointy (tented) T waves can mean hyperkalemia
- An electrical impulse during this time can lead to an “R on T” phenomenon, causing a lethal dysrhythmia.

How does the electrocardiogram work?

The ECG provides a graphic picture of the electrical waves of depolarization and repolarization. These waves are transmitted to the body surface and picked up by conductive gel within the electrode pads. It is transmitted to the ECG monitor via the leads.

- The Leads provide views of the heart, and the standard ECG has 12 leads (12 views). The lead is a graphic picture of electrical current flowing between a positive and negative electrode.
- Monitoring is usually in two leads.
  - Limb leads or precordial leads (V1-V6) These precordial leads go around the chest to view R waves
  - Lead I, II, III (bipolar leads look at the negative to positive. When you look at the upper right to the lower left (lead II), you will have a positive QRS deflection. We like lead II the best!
  - Modified chest lead
- Lead Names
  - Limb leads: Bipolar (I, II, III)
  - Precordial leads: Unipolar (V1-V6)
  - Augmented leads: Bipolar (aVf, aVI, aVr)
- Einthoven’s Triangle is defined as an equilateral triangle whose vertices lie at the left and right shoulders and the pubic region, and whose center corresponds to the vector sum of all electric activity occurring in the heart at any given moment, allowing for the determination of the electrical axis. It is approximated by the triangle formed by the axes of the ECG limb leads I, II and III. The center of the triangle offers a reference point for the unipolar ECG leads.
- Lead Placement
  - White on right upper chest
  - Black on left upper chest
  - Brown at 5th ICS, right sternal border
  - Green at right hip
  - Red at left hip
  - An easy way to remember this is: White on right, smoke over fire, poop over grass.
  - Lead wires are often labeled RA, LA, MCL, LL, and RL.
  - Note that you will get a better picture over flat bone.

ECG Paper

Graph paper with little boxes and big boxes.

- Small squares = 0.04 seconds or 1 mm
- Large squares = 0.20 seconds or 5 mm
- 5 large squares = 1 second
- Horizontal axis measures time
- Verticle axis measures amplitude
- ECG monitors all record at a standard speed of 25 mm/sec
- You can speed up the flow of the paper through the machine if you are looking at a really fast rhythm...this will spread the rhythm out a bit so you can see the detail.

Interpreting ECG Strips

- Step 1: Look for regularity or irregularity.
- Step 2: Determine the heart rate
- Step 3: Look for P waves
- Step 4: Measure the PR interval
- Step 5: Examine QRS complex
- Step 6: Check out the T wave

Step 1: Look for regularity or irregularity

- Are the P-R intervals the same?

- Are things generally happening in a consistent manner?
- The faster the rhythm the more difficult to tell if it is irregular

Step 2: Determine the heart rate

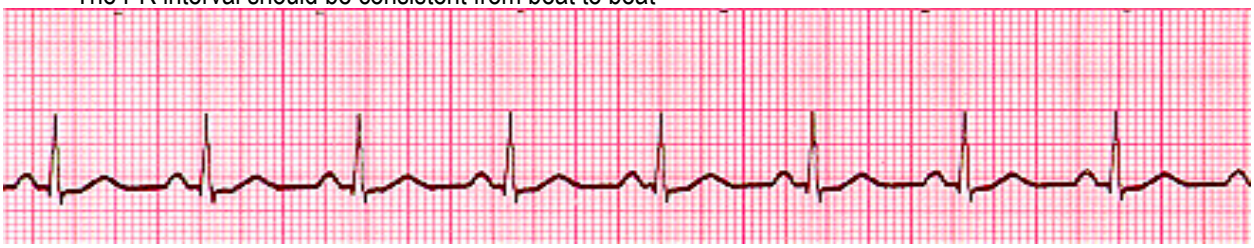
- Easy way: count the complexes on a 6-second strip and multiply by 10. This is your heart rate.
- Math way: count the boxes between two consecutive R waves
  - Divide 300 by the number of boxes between R waves (i.e. 300 ÷ 6 = 50 bpm)

Step 3: Look for the P waves

- Are there P waves present?
- Are they consistent...do they fall in the same place relative to the QRS?
- Is there a P wave preceding every QRS?

Step 4: Measure the PR interval

- Normal is 0.12 - 0.20 seconds
- A PR interval of > 2.0 seconds indicates a delay across the AV node
- A PR interval of < 0.12 is considered questionable for conduction (not enough time for impulse to go from SA to AV)
- The PR interval should be consistent from beat to beat

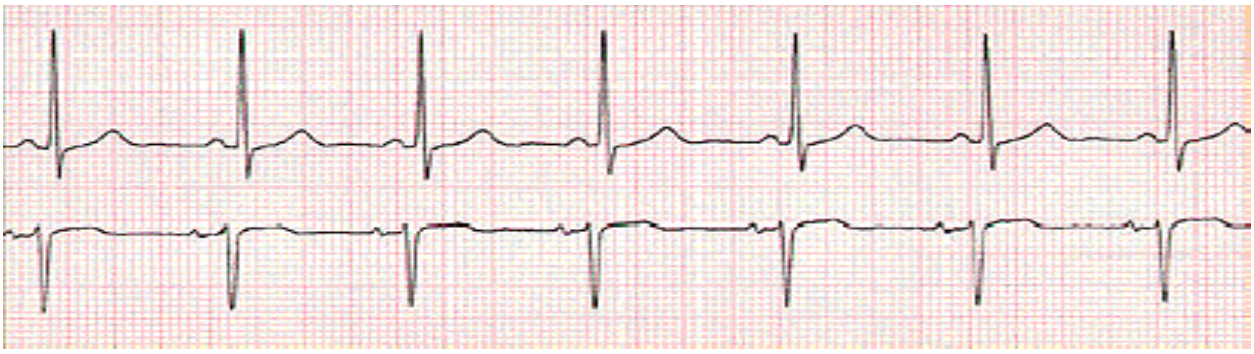


In the above EKG strip, it is REGULAR, the P waves are CONSISTENT, there is a QRS after every P. This is a normal sinus rhythm.

Step 5: Examine the QRS complex

- Determine the polarity (up or down). Both can be normal depending on the lead used.
- Is it a QRS, or just an RS?
- What is the width of the QRS? Should be , 0.12 or 3 little boxes. If it is wider this means there is a block in the Bundle of HIS or bundle branches
- Is it rSR' (RSR prime). This indicates a block somewhere along the bundle branches (looks kind of like an M)
- Is there a QRS following every P wave?

In the above example, the P waves are regular, the QRS are not coordinated, indicating that it is the Purkinje fibers firing. The SA node and the Purkinje fibers are each doing their own thing. The QRS is wide b/c the impulse has taken the side streets. This is called a 3rd Degree Block.



In the above graph, the top line is Lead II, the bottom line is MCL (brown lead). Notice that the QRS points down, this is normal for the MCL lead. See the QRS and that there is no Q (recall that the Q is the first downward deflection after the P wave. There is no downward deflection after the P, so no Q is present. This is OK! If that little upward deflection was not there, then the big downward deflection would actually be the FIRST downward deflection after the P, meaning it would be a GINORMOUS Q wave, which would be super-duper pathological. When looking at the MCL lead, the R is still shown as an upward deflection. Got it? :-)

Step 6: Check out the T-Wave

- The T-wave should be rounded and have a positive deflection
- Downward deflection indicates ischemia (inverted T wave)
- Tall and pointy = hyperkalemia
- Flattened = hypokalemia
- There's lots more than can be determined by the T wave, but we'll just start with that.

Ex: Right bundle branch block (see diagram to right). Remember the rSR' we mentioned earlier? See how it looks kind of like an M?
Also notice how the T wave is inverted? This is ischemia!

What is funky about the above strip? Well, check out the P waves....there isn't one here! Oops! The QRS is also very wide b/c the impulse is coming from the ventricle.

<u>Types of Rhythms</u>
Sinus rhythms are any rhythms that originate from the SA node.

- Rhythm is regular
- PR interval is normal (0.12 to 0.20 seconds)
- P wave precedes each QRS
- QRS follows each P wave
- QRS width is 0.08 to 0.12 seconds.

Sinus Tachycardia is a rate > 100 bpm. It is important to determine the reason for the tachycardia

- Fever
- Dehydration
- Anxiety or pain
- Hyperthyroid
- Hypoxia (keep this top of mind!)
- CHF
- Shock

Sinus Bradycardia is a rate < 60 bpm. It may or may not be symptomatic (an athlete could have a normal HR of 45)

- Assess these 4 things when you see a change in rhythm
  - Hypotension
  - Altered mental status
  - SOB, tachypnea
  - Chest pain

Sinus Arrhythmia is a variation of a NSR that is also called respiratory sinus arrhythmia.

- This is a normal finding in the young (20 year old)
- Occurs with respiration
- HR increases during inhalation and decreases during expiration
- It is cyclical with "regular irregularity" and is repetitive
- Strip to the right is one strip

Sick Sinus Syndrome is an abnormality of the sinus node (it's a little ischemic)

- Persistent sinus bradycardia
- Sinus arrests or pauses
- Combinations of SA and AV nodal conduction disturbances
- Alternating paroxysms of rapid atrial tachycardias
- Fast, slow, fast, slow
- Shown at right

<u>Sino-Atrial Dysrhythmias</u>

These are problems with transmission from the Sinus node to the AV node. This can occur b/c the impulse from the SA Node is not generated, or the impulse from the SA Node is not conducted. The causes are:

- Conditions that increase vagal tone (vaso-vagal episode: pt starts to pass out, but once they are no longer upright, it corrects itself...when you pass out, you fall down and this makes it "self limiting).
- Coronary artery disease
- Acute MI
- Digitalis and calcium channel blocker toxicity
- Hypertensive disease
- Tissue hypoxia
- Scarring of intra-atrial pathways
- Electrolyte imbalances
- Ischemia

<u>Atrial Dysrhythmias</u>

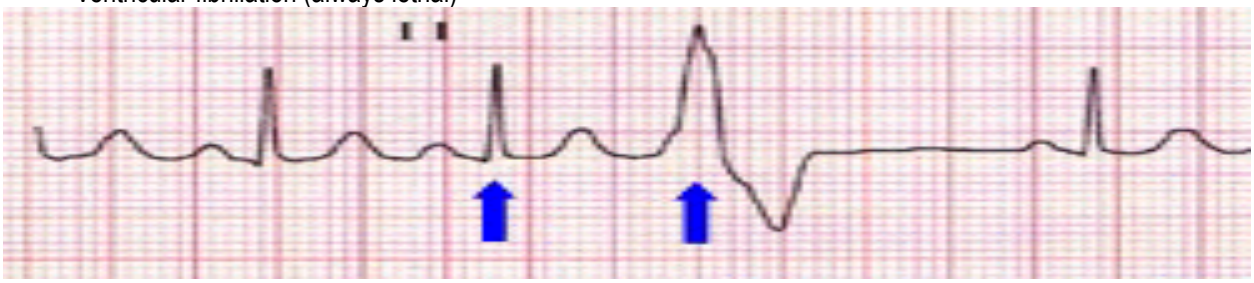
- Premature atrial contractions: causing an irregularity in rhythm, pretty benign and pretty common
- Paroxysmal atrial tachycardia (PAT): starts and stops abruptly, difficult to see P waves, common in mitral valve prolapse and people experiencing anxiety
- Atrial fibrillation: most common, there are no P waves, atrial rate is 350-700, ventricular rate varies (ventricular rate > 100 is "rapid ventricular response" aka "rapid atrial fib", affects 1-2% of general population, irregular irregularity
- Atrial flutter: saw tooth pattern

Blocks Across the AV Node

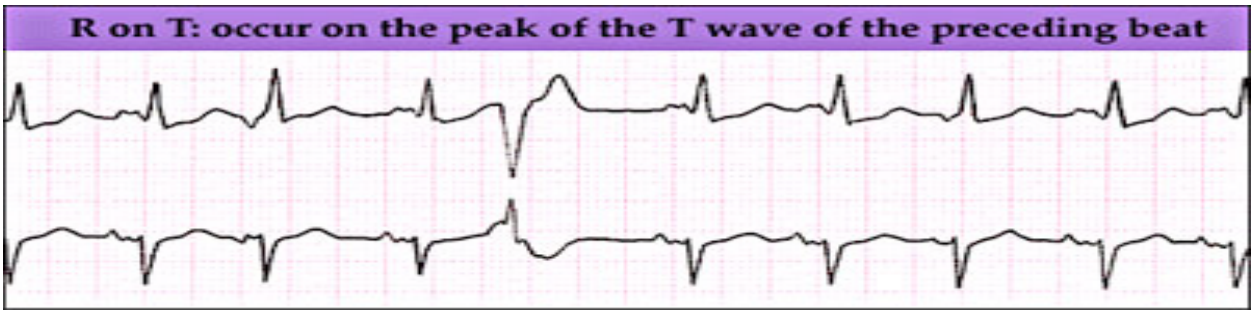
- First degree block has a prolonged PR interval (slows down SA conduction)
- Second degree block will block SOME impulses from SA).
  - The PR interval is inconsistent
  - No QRS following a P wave
- Third degree block will block ALL impulses and the ventricle does its own thing
  - No connection between P wave and QRS
  - Lethal rhythm

Ventricular Rhythms

- These are often VERY lethal
- Usually a disturbance in automaticity and excitability
- Premature ventricular contractions. These are more worrisome than AVC, especially if >6 x minute or 'R on T'. These may be prodromal (I suspect we'll learn what AVC is at some point?)
- Ventricular tachycardia (can be lethal)
- Ventricular fibrillation (always lethal)



In this strip, you see that there is no P wave, the QRS is wide and "odd looking". This QRS with no P is a premature ventricular contraction...if you feel the pulse as this rhythm is going on, you won't feel this one...does not perfuse.



The rhythm directly below is V-Tach...will probably lead to V-Fib. V-tach usually does not generate a blood pressure. Pt may lose consciousness and can be VERY symptomatic VERy quickly!
The rhythm directly above is V-Fib...pt will be non responsive with no pulse, cold and pale. This is LETHAL!

To end on a somber note...Sudden Cardiac Death occurs from an abrupt loss of heart function and pt may not even have coronary artery disease...more than 163,000 deaths annually!

Black, Joyce M., and Jane Hokanson Hawks. *Medical-Surgical Nursing: Clinical Management for Positive Outcomes - Single Volume (Medical Surgical Nursing- 1 Vol (Black/Luckmann))*. St. Louis: Saunders, 2009. Print.

Kelly, K. (2009, September). EKG Intepretation. Advanced Med/Surg. Lecture conducted from CSU Sacramento, Sacramento.