Normal Cardiac Conduction

Conduction Pathway

- Sino atrial (SA) node (in RA) fires
- Signal travels across atria to AV node
- Slight pause, then signal travels down Bundle of His
- His Bundle branches into Right & Left bundles (and further into fasicles)
- Ending at the Purkinje fibers
- Slight pause, then repolarization occurs
EKG Nomenclature

Normal 12-lead EKG

Heart Rate determination
- Start with a QRS on a heavy black line
- Use sequence to label rate at subsequent lines
Six Second Strip (aka 30 big blocks)

Calculate the rate:

62 is correct (or just a little faster than 60)

Rate of Impulse Formation

- S-A Node = 60-100 bpm
- A-V Node = 40-60 bpm
- Ventricle = 20-40 bpm
Normal Sinus Rhythm

<table>
<thead>
<tr>
<th>Heart Rate</th>
<th>Rhythm</th>
<th>P Wave</th>
<th>PR interval</th>
<th>QRS complex</th>
</tr>
</thead>
<tbody>
<tr>
<td>60-100 bpm</td>
<td>Regular</td>
<td>Before each QRS</td>
<td>.12 to .20</td>
<td>&lt;.12</td>
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Sinus Bradycardia

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<tr>
<td>&lt;60 bpm</td>
<td>Regular</td>
<td>Before each QRS</td>
<td>.12 to .20</td>
<td>&lt;.12</td>
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</tbody>
</table>

Sinus Bradycardia

- NSR criteria, but heart rate is < 60
- Can be normal especially in well-trained athletes or people taking B-blockers
- Most common rhythm disturbance seen in early stages of AMI
- If symptomatic, treat with Atropine, consider pacing (TransCutaneous Pacing/TCP)
Sick Sinus Syndrome

- Associated with sinus arrest, s-a exit block, or persistent sinus bradycardia < 45 bpm
- Seen in elderly, with atrial fibrillation, but often asymptomatic
- Patchy fibrosis of SA node and conduction
- May be caused by drug therapy, sarcoid, amyloidosis, cardiomyopathies
- Treat with pacemaker if symptomatic

Sinus Tachycardia

- NSR criteria, but HR is >100<180
- Normal under many conditions: exercise, fever, hyperthyroid, CHF, COPD, ETOH
- Treat cause first
- If unstable/pt. symptomatic, immediate synchronized cardioversion
- If stable, try vagal maneuvers,
  - B-blockers, +/- RFA used definitively
Sinus Arrhythmia

- Normal physiological mechanism 2nd vagal influence (young and/or old)
- Barely detectable rate changes corresponding to the phases of respiration
- Slight increase in rate during inspiration
- Slight decrease in rate during expiration
Premature Atrial Contractions

- Arrive earlier than expected next beat
- Different shape to p wave (because it comes from a different and irritable focus in the atria)
- Normal/narrow QRS complex
- Followed by a compensatory pause
- Usually benign, provide reassurance

(P)SVT/Supraventricular Tachycardia

- Rate 140-240 and regular, w/ narrow QRS
- Sudden/abrupt start/stop
- AKA “a-v nodal reentry tachycardia” (AVNRT)
- Responds (slows or terminates) to vagal maneuvers, carotid massage (R then L: not both!)
- Pharmacologic agents: (IV) adenosine - 6 mg bolus or verapamil - 2.5 mg bolus
- +/- Cardioversion (100 j) if symptomatic
(P)Supraventricular Tachycardia

Wandering Atrial Pacemaker

- Somewhat irregular rate of < 100
- Impulses originate from three or more different foci in the atria, so we see at least 3 different p wave morphologies
- Followed by narrow QRS complex
- Often not treated
Multifocal Atrial Tachycardia

- Same as WAP, but rate is 100-140
- Impulses originate from three or more different foci in the atria, so we see at least 3 different p wave morphologies
- Followed by narrow QRS complex
- **Often associated with COPD**
- Treated with verapamil to slow conduction

Atrial Flutter

- **sawtooth pattern w/ narrow QRS**
- common in COPD
- 2:1 block most common, but 3:1, 4:1, and variable block exist
- Treated with antiarrhythmic (ibutilide) or electrical cardioversion if unstable
- catheter ablation is definitive therapy
Atrial Flutter with 2:1 block; ventricular rate is ~150 bpm

Atrial Flutter with variable block

Atrial Fibrillation
- No common/distinct/visible p waves
- Irregularly irregular w/ narrow QRS
- New onset: check thyroid function
- Treated with b-blocker, Ca blocker, cardioversion or ?permanently with radio frequency ablation (RFA)
Atrial Fibrillation

Common causes: HTN, CHD, myopathy
- r/o thyrotoxicosis and ETOH use/abuse
- Danger due to potential for blood to coagulate in RA sending clot to brain: CVA
- First goal in AF = rate control
- Second goal = Cardioversion or ablation
- Third goal in chronic AF = anticoagulation

Chronic Atrial Fibrillation Tx

- Anticoagulation most commonly treated with Coumadin (warfarin), Pradaxa (dabigatran) or Xarelto (rivaroxaban)
  - Levels must be monitored regularly (PT, INR) for coumadin-not Pradaxa/Xarelto
- On occasion, aspirin and/or Plavix (clopidogrel) is used for this purpose if ASA allergic
Normal Cardiac Pacemakers

- Atrial: SA Node – rate = 60-100
- Junctional: AV Node – rate = 40-60
- Ventricular: ventricle – rate = 20-40

Junctional Rhythm

- Regular rhythm (so NOT a-fib)
- Normal/narrow QRS (wide possible)
- Absent, retrograde, or inverted p waves
- Rate usually 40-60 bpm
- Often seen with digitalis toxicity
Premature Junctional Contraction (PJC)
- PJC is early, narrow QRS, followed by compensatory pause, and with absent, inverted, or retrograde p wave

Accelerated Junctional Rhythm
- Regular rhythm with narrow/normal QRS
- Rate > 60 but < 100
- P wave is absent, inverted, or retrograde

Junctional Tachycardia
- Regular rhythm
- Rate > 100
- P waves are absent, inverted, retrograde
Normal Cardiac Pacemakers

- **Atrial**: SA Node – rate = 60-100
- **Junctional**: AV Node – rate = 40-60
- **Ventricular**: ventricle – rate = 20-40

---

Idioventricular Rhythm – treat with Pacemaker

<table>
<thead>
<tr>
<th>Heart Rate</th>
<th>Rhythm</th>
<th>P Wave</th>
<th>PR interval (ms)</th>
<th>QRS (ms)</th>
</tr>
</thead>
<tbody>
<tr>
<td>20-40</td>
<td>Regular</td>
<td>Absent or not related</td>
<td>N/A</td>
<td>&gt;12</td>
</tr>
</tbody>
</table>

---

Accelerated IVR (AIVR): common after MI: do not treat

<table>
<thead>
<tr>
<th>Heart Rate</th>
<th>Rhythm</th>
<th>P wave</th>
<th>PR interval (ms)</th>
<th>QRS (ms)</th>
</tr>
</thead>
<tbody>
<tr>
<td>40-100</td>
<td>Regular</td>
<td>Absent or not related</td>
<td>N/A</td>
<td>&gt;12</td>
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</table>
PVCs (Premature Ventricular Contraction)

- Most common ventricular arrhythmia
- Beat is early, wide, bizarre
- Sticks out like a “sore thumb”
- Followed by a compensatory pause
- Rare/occasional PVCs may be normal, especially if they resolve with activity
- Check electrolytes, thyroid, occult heart dz
- If symptomatic, look for cause and treat with b-blockers first then consider ablation

Premature Ventricular Contractions

Unifocal PVC

Unifocal PVCs: identical shapes
Note: A single PVC is labeled isolated
Multifocal PVCs

Ventricular Bigeminy

Ventricular Couplet
“Salvo” or 3-beat Run of VT

Triplet PVCs: occur in groups of three

“R-on-T” phenomenon

- Danger: may initiate Torsade/VT/VF
- Find cause; Tx with lidocaine or amiodarone then cardiovert (100-360 J)

R on T: occur on the peak of the T wave of the preceding beat

Ventricular Tachycardia

- A run of > 3 PVCs in a row
- Rate regular and > 100 (160-240)
- May be mono or polymorphic
- Common cause = hypo- K+ or hypo-magnesemia
- Non-sustained: <30 sec., spont. terminate
Ventricular Tachycardia
- Monomorphic: more commonly associated with a healed infarction
- If symptomatic, tx with cardioversion; drugs = lidocaine or amiodarone; OD pace if recurrent; definitive therapy = ICD

Torsades de Pointes
- Polymorphic VT

Polymorphic VT
- Torsades de pointes means “twisting of the points”
- Often associated with Long QT intervals
- May be congenital, but often results from electrolyte imbalance (K, Mg, Ca)
- Treat with B-blockers or temporary pacing (if pulse is present)
- Do NOT treat with antiarrhythmics as they prolong the QT interval.
Ventricular Fibrillation

**Ventricular Fibrillation**

<table>
<thead>
<tr>
<th>Heart Rate</th>
<th>Rhythm</th>
<th>P Wave</th>
<th>PR interval (in seconds)</th>
<th>QRS (in seconds)</th>
</tr>
</thead>
<tbody>
<tr>
<td>300-600</td>
<td>Extremely irregular</td>
<td>Absent</td>
<td>N/A</td>
<td>Fibrillatory baseline</td>
</tr>
</tbody>
</table>
AV Conduction Blocks

- An AV Conduction Block is any obstruction or delay of the normal conduction between the SA node and the Purkinje fibers.
- Most commonly occurs between AV node and His bundle.
- 3 varieties of AV Block: 1st, 2nd, 3rd degree.

First Degree AV Block

- All normal except PR interval > .2 sec (1 big block); often seen in athletes and with bradycardia; no treatment required.
Second degree AV Block
Wenckebach (aka Mobitz type I)

- Gradually lengthening PR interval until a QRS complex is dropped.
- Look for “grouped beating” in the EKG
- No treatment required; +/- EP studies

![3:2 2nd Degree AV block (Type I)](www.healcentral.org/content/collections/ECG/ecg_2A_V_block.gif)

Second Degree AV Block – Mobitz II

- PR interval remains constant
- Intermittent dropping of QRS
- Usually @ with organic heart disease
- Pacemaker usually indicated

![2:1 2nd Degree AV block (Type II)](www.healcentral.org/content/collections/ECG/ecg_2B_V_block.gif)

Third Degree (aka) Complete Heart Block

<table>
<thead>
<tr>
<th>P Wave</th>
<th>PR Interval (in seconds)</th>
<th>QRS (in seconds)</th>
<th>Characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal but not related to QRS</td>
<td>None</td>
<td>N/A</td>
<td>No relationship between P &amp; QRS</td>
</tr>
</tbody>
</table>

![Third Degree (complete) AV Block](www.healcentral.org/content/collections/ECG/ecg_chb.gif)
Third Degree/Complete Heart Block

- No relationship between p waves and QRS (p waves “marching through”)
- More p waves than QRS complexes
- Ventricular response often slow (IVR)
- Treat with permanent pacemaker

Third Degree A-V Block with Pacemaker Insertion Treatment

Pacemakers

- A power source connected to electrodes
- Most popular type = dual chamber multiple programmable units
Single Chamber Pacemaker

Electronic Pacemaker Spikes

Artificially induces electronic stimulus that paces the patient's rhythm causing a blip or spike on the ECG waveform.

Pacemaker Wire Placement

Ventricular Pacing
The pacing lead is inserted into the ventricle to cause ventricular depolarization.

Atrial Pacing
The pacing lead is inserted into the atrium to cause atrial depolarization.

A-V Sequential Pacing
The pacing leads are inserted into both the atrium and ventricle to produce atrial and ventricular capture.

Dual Chamber Pacing (aka AV sequential pacing)

AV Sequential Pacemaker (dual chamber)

One spike followed by an abnormal P (atrial capture) followed by a Second spike producing a wide QRS (ventricular capture).
Pre-excitation Syndromes

- Accessory AV conduction pathways
- Result in “short circuits” or shortcuts from SA node to AV node
- Usually diagnosed on 12-lead EKG
- 2 types: Wolff-Parkinson-White Syndrome and Lown-Ganong-Levine Syndrome

Wolff-Parkinson-White Syndrome

- Accessory pathway via “bundle of Kent”
- Appears as short PR interval (<.08)
- Presence of delta wave
- Predisposes to tachyarrhythmias (A-fib—VF)
- Treated with amiodarone or sotalol; if unstable cardiovert, then RFA definitively
Lown-Ganong-Levine Syndrome

- Accessory pathway via “James bundle”
- Short PR interval
- No delta wave

- Beware rapid arrhythmias
- Treat as with WPW

LGL – short PR, no delta wave
Mean QRS Vector and Axis

- Down and to pt.'s left

EKG Axis Determination
(Who’s triangle?) Dr. Einthoven!

Normal 12-lead EKG
12 Lead EKG Arrangement

- I  AVR  V1  V4
- II  AVL  V2  V5
- III  AVF  V3  V6

- Rhythm strip (1-3 leads)

Axis determination short cut

- Look at the QRS deflection
- Using leads I and AVF: (up = +; down = -)
  - Up in I and Up in AVF = Normal Axis
  - Up in I and Down in AVF = Left Axis Deviation
  - Down in I and Up in AVF = Right Axis Dev.
  - Down in I and Down in AVF = Extreme RAD

Left Axis Deviation (LAD)
**Right Axis Deviation (RAD)**

**Bundle Branch Block (BBB)**

**LBBB Diagnosis Criteria**

- **Wide (>12 sec) QRS**
- **RSR' in V5 or V6** (aka “dog ears”)
- Associated with ST depression in I, AVL, V5, and V6
- Often associated with CAD
- New LBBB (@ w/ CP) = “STEMI equivalent”

**BEWARE:** Difficult to diagnose MI via EKG in presence of LBBB.
Left Bundle Branch Block

Criteria to Diagnose RBBB

- Wide QRS (> .12 sec)
- RSR’ in V1 or V2 (aka “rabbit ears”)
- May have associated ST depression in V1, V2, +/- V3
- Can be found in a “normal” EKG (i.e., usually not associated with CAD)
Right BBB with A-fib

Hemiblocks
- Blocks occurring in either the Anterior or Posterior Divisions (fascicles) of the Left Bundle Branch (just distal to the AV node)
- Often result from diminished blood supply, i.e. MI or ischemia
- QRS may be normal or wide
- Major effect on EKG is axis deviation

Hypertrophy and Enlargement
- Hypertrophy = ventricles; enlargement = atria
- Specific EKG leads show evidence of these findings
- ALL EKG findings of hypertrophy and enlargement must be verified with ECHOCARDIOGRAM ! ! !
Examples of LVH & RVH

Left Atrial Enlargement

- Look for biphasic p wave in V1 with large terminal portion
- May be associated with wide p waves elsewhere
- May result from mitral stenosis, HTN

Left Atrial Enlargement
Right Atrial Enlargement

- Look for tall (>2.5 mm) peaked p waves in II, III, AVF
- Results from tricuspid stenosis, pulmonary HTN, severe lung dz

Left Ventricular Hypertrophy

- S in V1/2 + R in V5/6 > 35 mm
- R in I + S in III > 25 mm
- R in AVL > 13 mm
- Often caused by HTN and valvular dz
- Often associated with LAD & depressed ST seg (aka “ventricular strain” pattern)
- Criteria not totally applicable < 35 y/o or thin
Left Ventricular Hypertrophy: look for deep S in V1/2 plus tall R in V5/6 > 35 mm

Right Ventricular Hypertrophy
- Tall R wave in V1 (R>S)
- Deep S wave in V6 (S>R)
- Must be accompanied by RAD
- Commonly caused by pulmonary disease and congenital heart disease

RVH with RAE, causing RAD
Myocardial Infarction

Diagnosing an MI

- History and Physical Exam
- Cardiac Markers (CK-MB, Troponin)
- EKG

Diagnosing an MI: EKG findings

- T wave peaking (rarely seen on EKG; occurs within minutes of MI) followed by inversion of T wave (hours to days later)
- ST segment elevation (STEMI) (within hours)
- Appearance of new Q waves (hours to days)
Evolving EKG changes of MI

- Normal
- Prolonged T wave
- Degrees of ST segment elevation
- Q wave formation and loss of R wave
- T wave inversion

Evolution of Acute MI ST-changes

Localizing an MI

- Anterior Wall MI: Supplied by Left Anterior Descending Artery
- Seen as EKG changes in I, V1-V4
- Often a very deadly MI
- Often associated with “poor R-wave progression”

- V1 & V2 are mainly considered “septal” leads, so many MIs are “antero-septal.”
Leads I, V1-V4 = Anterior MI

Acute Antero-septal wall STEMI

Inferior Wall MI
- Inferior or diaphragmatic portion of the heart is supplied mostly by the Right Coronary Artery
- Can be associated with Right ventricular infarction
- EKG changes are seen in II, III, AVF
II, III, AVF (± V6 ST elevations) = Inferior MI

Inferior Wall MI

Lateral Wall MI

- Lateral portion of the heart is supplied by the Left Circumflex Artery.
- EKG changes seen in I, AVL, V5 & V6
I, AVL, V5, V6 = Lateral MI

- Often a result of left circumflex artery occlusion

Acute LWMI w/ reciprocal changes

- Posterior cardiac muscle is mainly supplied by the Right Coronary Artery
- Frequently associated with arrhythmias
- Since we have no EKG leads there, we look for ST segment depression and tall R waves in V1/V2 to diagnose a PWMI
- (Or you may invert tracing and use “mirror test” to see ST elevation in V1/V2)
Look in V1 for “mirror image”:
Tall R wave in V1, +/- ST dep

Reciprocal Changes
- Sometimes, the dramatic EKG changes in the infarct area produce opposing changes in distant leads (or those opposite the infarct location).
- In an Acute MI with ST elevations, ST depression may appear in a distant lead.
- These ST depressions are called “reciprocal changes”

Non-Q Wave or Non-STEMI MIs
- Not all MIs produce Q waves or ST elevations
- On EKG, a non-Q Wave or non-STEMI only shows: T wave inversion and ST depression
- Must use History and Cardiac Markers to make diagnosis
Non STEMI vs. Ischemia: note ST dep. and flipped T waves in I, II, V3-V6 (lateral wall area)

EKG findings of “old” MI
- ST segments and T wave changes usually return to “normal” after several days.
- Q waves often remain indefinitely
- Look for residual Q waves in specific areas to localize old MIs

Remember this?
- Normal
- Maximal T wave
- Depression of ST segment elevation
- Q wave formation and loss of R wave
- T wave inversion
Old IWMI: note sig. Qs only present

Ischemia/Angina Pectoris
- Angina is the diagnosis given to the typical chest pain associated with CAD
- Classic finding is precordial chest pain precipitated by stress or exertion and rapidly relieved by rest or nitrates (NTG)
- EKG shows classic ST depressions

Types of ST Segment Depression
- Downsloping ST
- Upsloping ST
- Horizontal ST

ST Segment Depression

The J point occurs at the end of the QRS complex.
The ST segment begins at the J point and extends to a user-defined interval.
Ischemia: EKG w/ ST depression

- ST depression > 1 mm = significant
- < 1 mm – “non-specific ST segment changes”

Antero(septal) wall Ischemia

Inferolateral wall ischemia (XST)
Benign ST changes: Early repolarization (see diffuse ST elevations)

Prinzmetal’s (Variant) Angina
- Angina-like chest pain often a result of coronary artery spasm
- Associated with ST elevation on EKG
- Thought to be a reversible injury, with ST segments returning to baseline after treatment with nitroglycerin

Miscellaneous EKG Diagnoses
- Certain effects may be recognized by their characteristic appearance on EKG
- The EKG “alerts us” to the diagnosis—it does NOT make the diagnosis
- It can act as another “clue” in making a diagnosis
Brugada Syndrome

- Familial condition predisposing to sudden cardiac death
- Young Asian Males more commonly affected
- Classic EKG: RBBB w/ ST elevation in V1, V2, V3
- Treat with b-blocker and ICD

Pulmonary Embolus (PE)

- Classic trio: S I, Q III, Inverted T in III
- Most common EKG finding in PE = ST
- May show T wave inversion in V1-V4
- RBBB (complete or incomplete)
Hyperkalemia (high potassium)

- The potassium ion is critical in the cardiac conduction cycle at the cellular level
- The range of potassium is very narrow
- Classic finding is tall, peaked T waves
- As the levels increase, the p wave flattens while the QRS widens – beware!
Hyperkalemia: (early stage); note tall, peaked T waves in V1-V4

Note wide QRS, absent p throughout EKG in advanced hyperkalemia

Hypokalemia: Classic findings: flattened T wave and new U wave
Hypokalemia: u wave in V2, V3

Hypocalcemia
- Levels of calcium drop below normal
- QT lengthens—DANGER!

Hypocalcemia with long QT seen in all leads here
Long QT Syndrome

- May be an inherited syndrome (Romano-Ward syndrome)
- May be due to a variety of drugs (quinidine, sotalol, abx such as the quinolones, and antidepressants)
- May lead to “R-on-T” or Torsades
- Treat with b-blocker, Mg, pacer, ICD

Hypothermia

- J/Osborne wave (usually with bradycardia)

Digitalis: effect and toxicity

- Digitalis effect causes “scooped” ST seg and is normal
- Digitalis excess and toxicity can cause AV blocks and ventricular rhythms
Pericarditis: inflammation of the pericardial sac – acute or chronic

Acute pericarditis: diffuse ST elevations

Chronic pericarditis: diffuse ST depressions
Electrical Alternans

- Associated with a large pericardial effusion
- Electrical axis of the heart varies with each beat due to the heart “floating in fluid-filled sac”
- Results in varying amplitude (alternating large and small) of EKG beats (and pulse on PE = pulses alternans)

Hypertrophic Cardiomyopathy

- Previously known as IHSS, may occur as obstructive (HOCM), dilated, or restrictive
- Causes SCD in some instances (young)
- EKG may show Q waves in many leads, LVH, LAD, and some deeply inverted T waves.
- Treated (if diagnosed) by placement of ICD
Hypertrophic Cardiomyopathy

Implantable Cardioverter Defibrillator (ICD)

References:

- www.healcentral.org
- www.commons.wikimedia.org
This 54 y/o female presents c/o new onset of intermittent palpitations and SOB x 2 mos. What is the rhythm? Atrial fibrillation

If she maintains this rhythm, despite treatment, what medication should she be prescribed and why?
Coumadin, Pradaxa, or Xarelto for anticoagulation.

Your pt. is c/o chest pain, SOB, and is diaphoretic. He has a pulse.
- What is this rhythm?
  - VT (sustained ventricular tachy/monomorphic)
- What is the recommended treatment?
  - W/pulse, cardioversion; if no pulse, defibrillation

This rhythm strip is taken from a 62 y/o man. He is unresponsive.
- What should you do first?
  - Initiate ABCs of BLS/CPR or call a code
- What is his definitive treatment?
  - Defibrillation
This is a 44 y/o man who presents c/o mild CP and nausea x 2 hours.

44 y/o man with CP & nausea

--Red flags/findings?
  - ST elevations in II, III, AVF, V5, V6

--Other findings?
  Reciprocal changes in I, AVL, V1

--Rate, rhythm, axis?
  110, ST, normal

This is a 55 y/o female with a h/o DM who presents c/o dyspnea x 2 hours.
55 y/o female diabetic w/ dyspnea

- Red flags?
- ST elevations in I, AVL, (?)V5, V6
- Tall R wave and ST depression in V1

Findings/Interpretation?
- RSR' in V1/V2 = RBBB
- Acute postero-lateral wall MI
- Reciprocal changes in II, III, AVF

This EKG is taken on a 25 y/o male who presents for a pre-employment physical.

What is the most likely diagnosis here? -- Why?
Brugada Syndrome (note classic ST shape in V1-V3)
Remember this T-shirt?

I LOVE CARDIOLOGY

Thanks for your attention and GOOD LUCK!

UMDNJ PANCE/PANRE Review Course