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

UMDNJ PANCE/PANRE Review Course (becoming Rutgers July 1, 2013)
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
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 (in seconds)</th>
<th>QRS (in seconds)</th>
</tr>
</thead>
<tbody>
<tr>
<td>60-100 bpm</td>
<td>Regular</td>
<td>Before each QRS, identical</td>
<td>.12 to .20</td>
<td>&lt;.12</td>
</tr>
</tbody>
</table>

Sinus Bradycardia

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

- 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 due to 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

**Premature Atrial Contraction• Isolated PAC's: Occur Single**

<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>N/A</td>
<td>Irregular</td>
<td>Premature &amp; abnormal or hidden</td>
<td>&lt;.20</td>
<td>&lt;.12</td>
</tr>
</tbody>
</table>

(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 patients
- 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

<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>A: 220-430 bpm Vc &lt;300 bpm</td>
<td>Regular or variable Sawtoothed appearance</td>
<td>N/A</td>
<td>&lt;.12</td>
<td></td>
</tr>
</tbody>
</table>

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: (details via CHADS$_2$ scoring system)

Chronic Atrial Fibrillation Tx

- Anticoagulation most commonly treated with Coumadin (warfarin), Pradaxa (dabigatran), or Xarelto (rivaroxaban)
- Levels must be monitored regularly (PT, INR) for warfarin only
- 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</th>
<th>QRS Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>20-40</td>
<td>Regular</td>
<td>Absent or not related</td>
<td>N/A</td>
<td>≥ .12</td>
</tr>
</tbody>
</table>

Accelerated IVR (AIVR): common after MI: do not treat
PVCs (Premature Ventricular Contraction)

Premature Ventricular Contractions

- 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

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 PVC’s: 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)

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 = amiodarone or lidocaine; 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**

- **(Coarse) Ventricular Fibrillation**
  - Most common cause of SCD (sudden cardiac death)
  - Chaotic, irregular rhythm; no true QRS
  - No pulse on PE; treat with CPR/defibrillation/ACLS protocols
  - With survival, treat with ICD

**Asystole**

- No electrical activity/no defib; look for cause and treat specific abnormality
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

1st degree AV block (PR = 280 ms)
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

Second Degree AV Block – Mobitz type II

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

Third Degree (aka) Complete Heart Block

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>PR Interval</th>
<th>QRS Interval</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal but not related to QRS</td>
<td>None</td>
<td>N/A</td>
</tr>
</tbody>
</table>

No relationship between PQRST
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.

AV Sequential Pacing: The pacing leads are inserted into both the atrium and ventricle stimulating at set intervals.

**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 tachycardias (A-fib—VF)
- Treated with amiodarone or sotalol; if unstable cardiovert, then RFA definitively
WPW (here, look in V4-V6, delta waves)

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

![QRS Vector Diagram](www.commons.wikimedia.org/wiki/File:ECG_Eomtjpvem_vect0.svg)

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

![Einthoven's Triangle Diagram](www.commons.wikimedia.org/wiki/File:ECG_Einthoven_vect.2.svg)

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 (>0.12 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)

RBBB: RSR’ in V1/V2
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

LAE:
Right Atrial Enlargement

- RAE:
  - 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 w/in minutes of MI) followed by inversion of T wave (hours to days later)
- ST segment elevation (STEMI) (w/in hours)
- Appearance of new Q waves (hours to days)
Evolving EKG changes of MI

- Normal
- Pulled T wave
- Degree 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 Wall MI

- 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?
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**

- Downslowing ST
- Upsloring ST
- Horizontal ST
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 β-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 progression

- Tall peaked T wave
- Loss of P wave
- Widened QRS with tall T wave
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

Stages of Pericarditis

Acute Pericarditis w/ diffuse ST elevation
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)

A 27 y/o female with a h/o anxiety presents c/o “a racing heart”. Here is a rate/rhythm strip taken at presentation. Which of the following is her rhythm?

1. Atrial fibrillation
2. Atrial flutter
3. Supraventricular tachycardia
4. Ventricular tachycardia

- Atrial flutter

- Supraventricular tachycardia

- Ventricular tachycardia
A 67 y/o female, 1 day post PM presents with severe SOB and dizziness. What is her rhythm via telemetry?

1. Atrial fibrillation
2. Supraventricular tachycardia
3. Ventricular fibrillation
4. Ventricular tachycardia

References:

- www.healcentral.org
- www.commons.wikimedia.org