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
Rate of Impulse Formation

- S-A Node = 60-100 bpm
- A-V Node = 40-60 bpm
- Ventricle = 20-40 bpm
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
Supraventricular Tachycardia (SVT)

Wandering Atrial Pacemaker (n.o.b.b.)*

- 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

(*not on NCCPA Board Blueprint)
Multifocal Atrial Tachycardia (n.o.b.b.)

- 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
- 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 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

- 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)

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 CHADS2 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

<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>40-60 bpm</td>
<td>Regular</td>
<td>Inverted, absent or after QRS</td>
<td>&lt;.12</td>
<td>&lt;.12</td>
</tr>
</tbody>
</table>
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

Accelerated Junctional Rhythm (n.o.b.b.)

- Regular rhythm with narrow/normal QRS
- Rate > 60 but < 100
- P wave is absent, inverted, or retrograde
Junctional Tachycardia (n.o.b.b.)

- 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
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
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 β-blockers first then consider ablation
3-beat Run of VT (aka “Salvo”)

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**

- 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

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**Ventricular Rhythms: compared**

- Torsade des Pointes
- Asystole
- Ventricular Fibrillation
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 –

Mobitz type II

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

Third Degree A-V Block with Pacemaker Insertion Treatment

Rutgers, The State University of New Jersey
Rutgers, The State University of New Jersey

**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 to set specified intervals.
Dual Chamber Pacing (aka AV sequential pacing)

AV Sequential Pacemaker (dual chamber)

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

WPW and LGL: shortcuts in conduction
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)
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

Lown-Ganong-Levine Syndrome

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

• Beware rapid arrhythmias
• Treat as with WPW

Mean QRS Vector and Axis

• Down and to pt.’s left

![Mean QRS Vector and Axis Diagram](https://commons.wikimedia.org/wiki/File:ECG_Eomtjpvem_vect0.svg)
EKG Axis Determination
(Who’s triangle?) Dr. Einthoven!

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

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)
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.

**Bundle Branch Block (BBB)**

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RBBB: RSR’ in V1/V2

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- Often associated with CAD
- New LBBB (@ w/ CP) = “STEMI equivalent”
- BEWARE: Difficult to diagnose MI via EKG in presence of LBBB.

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

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

www.commons.wikimedia.org/wiki/File:Heart_Left_ventricular_hypertrophy_sa.jpg

www.commons.wikimedia.org/wiki/File:Heart/kabulki_syndrome.jpg

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

LAE:
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

- 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

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
Myocardial Infarction

Diagnosing an MI

- History and Physical Exam
- Cardiac Markers (CK-MB, Troponin I & T)
- 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
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
• 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

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”
Acute LWMI w/ reciprocal changes

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."

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

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

Rutgers, The State University of New Jersey
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
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

- Downsliding ST
- Upsliding ST
- Horizontal ST
Antero(septal) wall Ischemia

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
**Brugada Syndrome**

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**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 (n.o.b.b.)

- 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)

Electrical Alternans; seen in various leads here

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)

Question 1 of 1:

A 37 yr female with a long history of "racing heart". Here is a rhythm strip taken at presentation. Which of the following is her rhythm?

- Atrial fibrillation
- Atrial flutter
- Supraventricular tachycardia
- Ventricular tachycardia
Thanks for your attention!

References:

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