Conduction and Rhythm Disorders: Dancing to the beat of a different rhythm

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Disorders Covered

- Supraventricular Rhythms/Arrhythmias
  - Sinus Arrhythmias, Bradycardia, Tachycardia
  - Paroxysmal Supraventricular Tachycardia
  - Atrial Fibrillation & Flutter
- Conduction Disturbances
  - Blocks
    - 1st Degree, 2nd Degree (Mobitz I and II), & 3rd Degree Block
    - LBBB, RBBB, IRBBB, RIVRBBB
- Ventricular Dysrhythmias
  - Premature Ventricular Contractions
  - Ventricular Tachycardia & Fibrillation
  - Torsade de Points
- Asystole / Pulseless Electrical Arrhythmia

Normal Sinus Rhythm

- Rate: Normal (60-100 bpm)
- Rhythm: Regular
- P-Wave: Normal (upright and uniform)
- PR Interval: Normal (0.12-0.20 sec)
- QRS: Normal (0.08-0.10 sec)
Normal Sinus Rhythm

• **Mechanism**
  - Due to normal discharging of the SA node
  - SA node is sensitive to stimuli from the autonomic nervous system
  - Sympathetic (adrenergic) stimuli serve to increase the rate
  - Parasympathetic (cholinergic) stimuli serve to slow the rate

• **Management**
  - None

Sinus Bradycardia

• **Rate:** Slow (<60 bpm)
• **Rhythm:** Regular
• **P Waves:** Normal (upright and uniform)
• **PR Interval:** Normal (0.12-0.20 sec)
• **QRS:** Normal (0.06-0.10 sec)

• **Mechanism**
  - Suppression of the SA node causing it to discharge at less than 60 times per minute

• **Etiology**
  - May be an incidental finding in otherwise healthy individuals
  - Acute MI, athletic heart, sleep, aging, right CAD, myxedema, increased intracranial pressure, valsalva, fibrosis of SA node
  - Hypothermia, hypoglycemia, sleep apnea
  - Medications: digitalis, beta blockers, procainamide, calcium channel blockers, reserpine, phenothiazines
Sinus Bradycardia

• Signs & Symptoms
  • Usually asymptomatic
  • Possible hypotension, altered mental status, syncope, and ectopic activity

• Treatment
  • If symptomatic
    • IV access, supplemental oxygen, cardiac monitoring
    • Atropine
    • Transcutaneous pacing (rare)

Sinus Tachycardia

• Rate: Fast (>100 bpm)
• Rhythm: Regular
• P Waves: Normal (upright and uniform)
• PR Interval: Normal (0.12-0.20 sec)
• QRS: Normal (0.06-0.10 sec)

Sinus Tachycardia

• Mechanism
  • SA node discharges at rates of 100-150
  • Usually begins and ends gradually

• Etiology
  • Exercise, pain, fever, dehydration, hypotension, anxiety, thyrotoxicosis, stimulants (coffee, etc), anemia, pulmonary embolism, MI, hypoxia, CHF, COPD, malignant hyperthermia
  • Medications: atropine, epinephrine, isoproterenol
Sinus Tachycardia

- Signs & Symptoms
  - Possible palpitations
  - In patients with underlying heart disease, possible angina or signs of failure

- Treatment
  - None usually or treat underlying cause
  - Beta blockers for acute myocardial infarction

Supraventricular Tachycardia (SVT)

- Mechanism
  - ANY tachycardic rhythm originating above the ventricular tissue with a narrow QRS complex

- Types
  - Sinus Tachycardia
  - Atrial Tachycardia
  - Junctional Tachycardia
  - Paroxysmal supraventricular tachycardia (PSVT)
  - Atrial Fibrillation with rapid ventricular response
  - Atrial Flutter with rapid ventricular response

Atrial Tachycardia

- Rate: 150-250 bpm
- Rhythm: Regular
- P Waves: Normal (upright & uniform) but differ in shape from sinus P waves
- PR Interval: May be short (<0.12 sec) in rapid rates
- QRS: Normal (0.06-0.10 sec) but can be aberrant at times
Atrial Tachycardia

- Types
  - Paroxysmal Atrial Tachycardia (PAT)
  - Multifocal Atrial Tachycardia (MAT)

- Mechanism
  - Firing of an ectopic atrial pacemaker sustains a rapid rhythm usually between 150-250 bpm
  - Most cases are believed due to a sustained re-entry of impulses through the atria causing rapid, repeated atrial depolarization

- Etiology
  - CAD, MI, stimulants (coffee, etc), thyrotoxicosis, hypoxia (COPD, CHF), WPW

- Medications
  - Amphetamines
  - Aminophylline

- Signs & Symptoms
  - Depends on rate and severity of any underlying heart disease
  - Palpitations, angina, syncope, or failure may result

- Treatment
  - Treat underlying condition
  - Beta blockers, calcium channel blockers

Paroxysmal Supraventricular Tachycardia (PSVT)

- Rate: 150-250 bpm
- Rhythm: Irregular
- P Waves: Frequently buried in preceding T waves and difficult to see
- PR interval: Usually not possible to measure
- QRS: Normal (0.06-0.10 sec) but may be wide if abnormally conducted through the ventricles
Paroxysmal Supraventricular Tachycardia (PSVT)

**Mechanism**
- Episodic abnormalities in impulse formation and conduction pathways
- The most common mechanism identified is reentry

**Etiology**
- Premature atrial or ventricular ectopic beats
- Hyperthyroidism
- Stimulants

**Signs & Symptoms**
- SOB, CP, pounding heart, dizziness, LOC

**Treatment**
- Maneuvers
  - Valsalva maneuver
  - Coughing
  - Carotid massage
- Medications
  - Adenosine
  - Diltiazem, verapamil
  - Metoprolol
  - Sotalol or amiodarone
- Cardioversion

Atrial Fibrillation

- Rate:
  - Atrial: chaotic, 350-600 bpm (F waves)
  - Ventricular: variable (usually 100-160 bpm)
- Rhythm: Irregularly irregular
- P Waves: Mitrail or white, chaotic atrial activity, best seen in leads II, III, or V1
- RR Interval: None
- QRS: Normal (0.06-0.10 sec)
Atrial Fibrillation

• Mechanism
  • Rapid firing of different atrial foci, or sustained reentry of impulses through the atria
  • Result is totally ineffective atrial contraction which causes a 25-33% drop in cardiac output
  • AV node conducts the chaotic atrial impulses irregularly, resulting in irregular R-R intervals

• Classification
  • First detected: only one diagnosed episode
  • Paroxysmal: recurrent episodes that self-terminate in less than 7 days
  • Persistent: recurrent episodes that last more than 7 days
  • Permanent: an ongoing long-term episode

Atrial Fibrillation

• Etiology
  • Hypoxia (COPD, PE), CAD, MI, hyperthyroidism, WPW, pericarditis, mitral stenosis, stress, hypothermia, hypertension, hypertrophic cardiomyopathy, excessive alcohol consumption
  • Medications: epinephrine, atropine, isoproterenol

• Signs & Symptoms
  • Some patients are asymptomatic
  • Others may have: palpitations, angina, syncope, diaphoresis, nausea/vomiting, dyspnea
  • 30% of patients will develop pulmonary or systemic emboli secondary to thrombosis development in the non-contracting atria
  • A pulse deficit may be detected clinically

Atrial Fibrillation

• Treatment
  • Anticoagulation
    • Aspirin, heparin, warfarin & dabigatran
  • Rate control (reduce HR to 60-100 bpm)
    • Beta blockers, calcium channel blockers, digoxin
  • Rhythm control
    • amiodarone, procainamide, ibutilide, propafenone or flecaïnide
  • Electrical Cardioversion
  • Surgical Ablation: Cox MAZE, radiofrequency ablation, cryomaze ablation
  • Electrophysiology Ablation
Atrial Flutter

- **Rate**
  - Atrial: 250-350 bpm
  - Ventricular: variable, 2:1 conduction is common
- **Rhythm**
  - Atrial: regular; Ventricular: variable
- **P Waves**
  - Saw-toothed flutter waves, may be buried
- **PR Interval**
  - Variable
- **QRS**
  - Usually normal (0.06-0.10 sec)

**Mechanism**

- Repetitive firing of the atrial due to a single irritable focus, or more commonly, due to sustained re-entry of a stimulus back through the atria
- Usually conducts to the ventricles in a 2:1 ratio resulting in a ventricular rate of usually 150/min

**Etiology**

- Hypoxia (COPD, PE), MI, thyrotoxicosis, mitral stenosis, pericarditis, cardiac trauma, WPW
- Drugs: epinephrine, atropine, isoproterenol

**Signs & Symptoms**

- Same as atrial fibrillation

**Treatment**

- Same as atrial fibrillation
- Atrial flutter is more sensitive to electrical direct-current cardioversion than atrial fibrillation
First-Degree AV Block

- Rate: depends on rate of underlying rhythm
- Rhythm: regular
- P Waves: normal (upright and uniform)
- PR Interval: prolonged (>0.20 sec)
- QRS: Normal (0.06-0.10 sec)

First-Degree AV Block

- Mechanism
  - Delay in conduction which produces a prolonged PR interval of greater than 0.21 seconds
  - Every atrial impulse, although delayed, is conducted to the ventricles, producing a regular ventricular rhythm

- Etiology
  - Rheumatic myocarditis, right CAD, MI, fibrosis of AV node, systemic infections, vagal stimulation, cardiomyopathies, tertiary syphilis, collagen diseases, myxedema, hyperkalemia
  - Drugs: digitalis, quinidine, procainamide, BB, CCB

First-Degree AV Block

- Signs & Symptoms
  - Usually none

- Treatment
  - Usually none
  - Correct possible electrolyte imbalances
  - Withhold possible offending medications
Second-Degree AV Block (Mobitz I or Wenckebach)

- Rate: Depends on rate of underlying rhythm
- Rhythm: Atrial: regular; ventricular: irregular
- P Waves: Normal (upright & uniform), more P waves than ARS complexes
- PR Interval: Progressively longer until one P wave is blocked & a QRS is dropped
- QRS: Usually Normal (0.06-0.10 sec)

Mechanism
- More atrial stimuli are blocked at the AV node compared to first degree AV block
- Bundle branch blocks may be frequently seen

Etiology
- Same as first degree AV block
- More frequently associated with chronic heart conditions

Signs & Symptoms
- Forceful beats are often felt by patients
- Wide pulse pressure is common

Treatment
- Usually none, same as first degree AV block

Second-Degree AV Block (Mobitz II)

- Rate: Atrial: usually 60-100 bpm; Ventricular: slower than atrial rate
- Rhythm: Atrial: regular; Ventricular: regular or irregular
- P Waves: Normal (upright & uniform), more P waves than ARS complexes
- PR Interval: Normal or prolonged but tend to be constant
- QRS: May be normal, but usually wide (>0.15 sec) if bundle branches are involved

Mechanism

Etiology

Signs & Symptoms

Treatment
Second-Degree AV Block (Mobitz II)

- Mechanism
  - More atrial stimuli are being blocked at the AV node compared to Mobitz I
  - Bundle branch blocks are frequently seen

- Etiology
  - Same as first degree AV block
  - Frequently associated with chronic heart conditions

Second-Degree AV Block (Mobitz II)

- Signs & Symptoms
  - Forceful beats may be felt by patients
  - Wide pulse pressure common

- Treatment
  - Because Mobitz II heart block can rapidly progress to complete heart block, patients are usually implanted with a pacemaker to prevent the possibility of cardiac arrest or sudden cardiac death

Third-Degree AV Block (Complete Heart Block)

- Rate: Atrial: usually 60-100 bpm; Ventricular: 20-60 bpm
- Rhythm: Usually regular, but atria & ventricles act independently
- P Waves: Normal (upright & uniform); may be superimposed on QRS complexes or T waves
- PR Interval: Irregular, varies greatly
- QRS: Normal if ventricles are activated by junctional escape focus; wide if escape focus is ventricular
Third-Degree AV Block (Complete Heart Block)

- **Mechanism**
  - Complete AV block does not allow any atrial impulses to reach the ventricles
  - Atrial pacemaker is totally independent of the ventricular pacemaker
  - The closer the pacemaker controlling the ventricles is to the Bundle of His, the more normal the QRS complex will appear

- **Etiology**
  - MI / Coronary ischemia: most common
  - Digitalis toxicity, degeneration of the conduction system, congenital if mother has lupus

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Third-Degree AV Block (Complete Heart Block)

- **Signs & Symptoms**
  - Possible forceful beats, angina, syncope, cannon “A” waves, PVC’s, & wide pulse pressure

- **Treatment**
  - Dual-chamber artificial pacemaker
  - Atropine usually does not help but may be attempted if waiting for setup of temporary pacing

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Bundle Branch Block

- **Left Bundle Branch Block**
  1. Complete LBBB
  2. Incomplete LBBB

- **Right Bundle Branch Block**
  1. Complete RBBB
  2. Incomplete RBBB
Left Bundle Branch Block
Electrocardiographic Criteria

1. The QRS duration is >/- 120 ms
2. Leads V5, V6 and AVL show broad and notched or slurred R waves
3. With the possible exception of lead AVL, the Q wave is absent in left-sided leads
4. Reciprocal changes in V1 and V2
5. Left axis deviation may be present

Causes Of LBBB

- Hypertrophy, dilatation or fibrosis of the left ventricular myocardium
- Ischemic heart disease
- Cardiomyopathies
- Advanced valvular heart disease
  - Toxic, inflammatory changes
  - Hyperkalemia
  - Digitalis toxicity
  - Degenerative disease of the conducting system (Lenegre disease)
Prevalence Of LBBB

At age 50 is 0.4%, and at age 80 it is 6.7%

In most subjects with LBBB, regional wall motion abnormalities (akineti c or dyskinetic segments in the septum, anterior wall or at the apex) are present even in the absence of CAD or cardiomyopathy.

Incomplete Left Bundle Branch Block

• Criteria for incomplete LBBB include

1. QRS duration > 100 ms but < 120 ms

2. Absence of a Q wave in leads V5, V6 and I

Right Bundle Branch Block

• The diagnostic criteria include

1. QRS duration is >/= 120 ms

2. An rsr’, rsR’ or rSR’ pattern in lead V1 or V2 and occasionally a wide and notched R wave.

3. Reciprocal changes in V5, V6, I and AVL.
Causes of RBBB

1. After repair of the VSD
2. After right ventriculotomy
3. Right ventricular hypertrophy
4. Increase incidence of RBBB among population at high altitude
5. Ebstein's anomaly
6. Large ASD (secundum type) or AV cushion defect
7. Brugada Syndrome

RBBB in the General Population

- The incidence increased with age
  1. Below age 30 the incidence is 1.3 per 1000
  2. Between 30 and 44 it ranges from 2.0 to 2.9 per 1000
Incomplete RBBB

- Criteria for incomplete RBBB are the same as for complete RBBB except that the QRS duration is < 120 ms

Causes of Incomplete RBBB

1. Atrial septal defect (RAD in secundum or sinus venosus type, LAD with ostium primum type)
2. Ebstein's anomaly
3. Right ventricular dysplasia
4. Congenital absence or atrophy of the bundle branch
5. After CABG and in transplanted hearts
6. Brugada Syndrome

Premature Ventricular Contraction (PVC)

- Rate: Depends on rate of underlying rhythm
- Rhythm: Irregular whenever a PVC occurs
- P-Wave: None associated with the PVC
- PR Interval: None associated with the PVC
- QRS: (>0.10 sec), bizarre appearance
Premature Ventricular Contraction (PVC)

- **Mechanism**
  - Result from an irritable ventricular focus
  - Can be uniform or multiformal (different forms)
  - PVC is usually followed by a compensatory pause
  - Numerous PVC’s may lead to ventricular tachycardia

- **Etiology**
  - Ischemia, myocarditis, cardiomyopathy, COPD, MI, stimulants, exercise, post-prandial, cocaine, thyroid dysfunction, stress, lack of sleep
  - Medications: digitalis (increased heart contraction)

Premature Ventricular Contraction (PVC)

- **Signs & Symptoms**
  - Palpitations, irregular pulse, forceful post-ectopic beat
  - Runs of PVC’s may cause angina, hypotension or syncope

- **Treatment**
  - Electrolyte replacement
  - Beta blockers, calcium channel blockers
  - Ablation

Ventricular Tachycardia (VT)

- Rate: 100-250 bpm
- Rhythm: Regular
- P-Waves: None or not associated with the QRS
- PR Interval: None
- QRS: Wide (>0.10 sec), bizarre appearance
Ventricular Tachycardia (VT)

- Types
  - Monomorphic ventricular tachycardia
    - Right ventricular outflow tract (RVOT) tachycardia
  - Polymorphic ventricular tachycardia

- Mechanism
  - Another example of AV dissociation where rapid firing of a ventricular focus at rates 100-250/min occur independently of the SA node

Ventricular Tachycardia (VT)

- May have associated
  - Fusion beats: ventricles depolarized simultaneously by supraventricular and ventricular pacemakers resulting in a QRS complex which is nearly normal in configuration but slightly aberrant. A P wave may precede the QRS
  - Capture beats: represent momentary recapture of the ventricles by a supraventricular pacemaker causing a perfectly normal PQRST complexes to appear in the midst of the ventricular tachycardia

Ventricular Tachycardia (VT)

- Etiology
  - CAD (most common cause), MI, myocarditis, 3rd degree AV block, mechanical irritation (catheterization), R on T phenomenon
  - Medications: digitalis, quinidine, procainamide, epinephrine, isoproterenol

- Signs & Symptoms
  - Depends on ventricular rate and severity of underlying heart disease
  - Angina / Failure
  - Syncope
  - Palpitations
  - Cannon “A” waves if A dissociation present
  - Possible pulselessness
Ventricular Tachycardia (VT)

- Treatment
  - Pulseless
    - Defibrillation per ACLS protocol
      - Monophasic defibrillator: 360J
      - Biphasic defibrillator: 200J
      - Chest compressions (BLS)
  - Pulse present
    - Synchronized Electrical Cardioversion
    - Antiarrhythmic medications
      - Amiodarone, procainamide, beta blockers, lidocaine
    - Cardiac ablation
    - ICD implantation

Ventricular Fibrillation (VF)

- Rate: Indeterminant
- Rhythm: Chaotic
- P Waves: None
- PR Interval: None
- QRS: None

Ventricular Fibrillation (VF)

- Mechanism
  - Rapid firing of multiple ventricular foci resulting in various sites depolarizing and repolarizing simultaneously
  - This produces entirely chaotic ventricular activity without any coordinated contractions

- Etiology
  - MI / CAD – most frequent
  - 3rd degree AV block, hypoxia, R on T phenomenon, hyper/ hypokalemia, hypercalcemia, mechanical irritation (i.e., catheter), electrical shock, renal failure, hepatic failure, acid/base imbalance
  - Medications: digitalis, procainamide, quinidine
**Ventricular Fibrillation (VF)**

- **Signs/Symptoms**
  - Pulselessness, apnea

- **Treatment**
  - Defibrillation per ACLS protocol
  - Chest compressions (BLS)
  - Antiarrhythmic medications
    - Amiodarone, procainamide, beta blockers, lidocaine
  - ICD implantation

**Torsade de Points**

(twisting of the points)

- Rate: 200-250 bpm
- Rhythm: Irregular
- P Waves: None
- PR Interval: None
- QRS: (>0.10 sec), bizarre appearance

**Mechanism**

- Twisting of QRS complex around the isoelectric baseline
- Often associated with prolonged QT syndrome
- May degenerate into ventricular fibrillation

**Etiology**

- Diarrhea, hypomagnesemia, hypokalemia, malnourished individuals, chronic alcoholics, hypokalemia, hypocalcemia, acidosis, heart failure, hyperthermia, subarachnoid hemorrhage
- Class IA and class III antiarrhythmics
Torsade de Points

- Treatment
  - Magnesium sulfate 2 grams IV push (first line)
  - Antiarrhythmic drugs
  - Defibrillation as needed
  - Chest compressions

Asystole

- Rate: None
- Rhythm: None
- P Waves: None
- PR Interval: None
- QRS: None

Asystole

- Mechanism
  - Complete absence of electrical activity producing no atrial or ventricular contractions

- Etiology
  - Most frequently the end stage of untreated ventricular fibrillation
  - H's & T's
    - Hypovolemia, hypoxia, hydrogen ions (acidosis), hypothermia, hyper/hypokalemia, hypoglycemia
    - Tablets/toxins (drug overdose), cardiac tamponade, tension pneumothorax, thrombosis, trauma
Asystole

- Signs/Symptoms
  - Pulselessness, apnea

- Treatment
  - Chest compressions
  - Epinephrine
  - Atropine

Pulseless Electrical Activity (PEA)

- AKA: Electromechanical Dissociation

- Mechanism
  - ANY organized electrical activity without effective contractions

- Etiology
  - H's & T's
    - Hypovolemia, hypoxia, hydrogen ions (acidosis), hypothermia, hyper/hypokalemia, hypoglycemia
    - Tablets/toxins (drug overdose), cardiac tamponade, tension pneumothorax, thrombosis, trauma

- Treatment
  - Chest compressions
  - Treat underlying cause
  - Epinephrine 1 mg every 3-5 minutes
  - Atropine (recently withdrawn in 2010 by the AHA)
  - Sodium bicarbonate may be considered
Thank You

Any Questions?