Methodology

- Rhythm Interpretation
- Patient Information
- Physiology and Pathophysiology
Blocks and Bradys
SA Block (SA Exit Block)

- Type I and Type II
  - Signs of Wenckebach
  - Fixed P to P
- Dropped P waves
- Typically transient
  - Healthy young people
  - Trained athletes
  - Digitalis toxicity
  - Other antiarrhythmics
  - Infarction / myocarditis
  - Part of SSS

- Quality of sinus node cells
- Sinus discharge versus atrial activation
Sinus Arrest or Sinus Pause

- Failure of impulse formation
- Impossible definitive diagnosis on surface ECG
- Clue: PP intervals of cycle cannot be walked out across the pause and end on P wave

Sick Sinus Syndrome

- Disorders of impulse generation and conduction
- Failure of escape pacemakers
- Susceptibility to atrial tachyarrhythmias
- Bradycardia / tachycardia syndrome
  - Long pause after tachycardia (overdrive suppression)
  - Syncope

40% SSS: coronary atherosclerosis
5-10% SSS: ideopathic cardiomyopathy
Heart Blocks – AV Blocks

- Classification
  - 1\textsuperscript{st} Degree
  - 2\textsuperscript{nd} Degree
    - Type I (Wenckebach)
    - Type II
  - High Grade
  - Third Degree

2nd Degree AV Blocks

- One P Wave at a time fails to conduct to ventricle
- Type I (Wenckebach)
  - Conduction fails in AV node
- Type II
  - Conduction fails below the AV node and usually involves both bundles
Wenckebach (2\textsuperscript{nd} Degree Type I)

- Sinus node fires regularly
- Disease in AV node
- Group beating is noted
- First P-R of group of often longer than normal with progressive lengthening of the P-R until a beat is not conducted
- In absence of BBB QRS is normal

- Conduction ratios may be 2:1, 3:2, 4:3 etc.
- May develop 2:1 conduction if sinus rate increases
  - Verify the block is still type I
  - P-R longer than normal
  - Absence of prolonged QRS
- Treatment: Often none
  - Acutely with symptoms: Atropine or TTVP
2\textsuperscript{nd} Degree Type (Wenckebach)

2\textsuperscript{nd} Degree AV Block Type II

- Similar to Type I however no progressive lengthening of P-R interval
- Disease within or below bundle of His
- P-R interval is fixed with normally conducted beats
- QRS: wide
- If 2:1 conduction look for:
  - Normal P-R interval with conducted beats
  - Wide QRS complex
- Treatment: Usually requires permanent pacing
Heart Blocks - High Grade AV Block

- Two or more consecutive atrial impulses are blocked.
- P waves: Regular, but 2 or > fail to conduct to the ventricles
- QRS: Narrow in type I & wide in type II
- Ventricular Rate: Slow, often symptomatic
- Treatment: Atropine for Type I
  Pacing for Type II - Usually

Third Degree AV Block – Complete

- No atrial impulses are conducted to the ventricles
- One form of AV dissociation
- Ventricular Rate: Maintained by junctional escape (narrow QRS) or ventricular escape (wide QRS)
- Symptomatic if develops acutely
- May be well tolerated if develops overtime
- Treatment: Perm. Pacer
Third Degree (Complete) Heart Block

Junctional Escape and Rhythm

- HR 35-60 beats per minute
- P' waves may or may not be associated with QRS complexes
- QRS complexes same as sinus beats
Ventricular Escape Beats
Idioventricular Ventricular Rhythm

Supraventricular Arrhythmias (SVA)
Supraventricular Tachycardia (SVT) – *in the broadest sense*

**Covers:**

- All tachycardias originating above the ventricles

Supraventricular Arrhythmias (SVA) in ACC/AHA Guidelines excludes Atrial Fibrillation

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**Classification of SVT**

- Sinus Tachycardia
  - Physiological
  - Inappropriate
  - SANRT
- Atrial Tachycardia
  - Focal
  - Intraatrial reentrant
  - Multi-focal
- Atrial Flutter
- Atrial Fibrillation
- Junctional Tachycardia
  - Nonparoxysmal JT
  - Junctional ectopic tachycardia
- AV Nodal Reentrant Tachycardia
- AV Reentrant Tachycardia
  - Orthodromic
  - Antidromic *(always wide)*

SVTs are narrow unless conducted aberrantly except antidromic tachycardia
Supraventricular Tachycardias

- Factors determining seriousness of any tachycardia
  - Absolute ventricular rate
  - Size of heart
  - Relationship between atrial and ventricular contraction
  - Presence of additional complications
  - Amount of time in tachycardia

Tachyarrhythmias

- Most common mechanism is re-entry

Re-entry is mechanism of action in:
  - AVNRT
  - AVRT
  - Atrial Flutter
  - Atrial Fibrillation (often)
Specific Tachycardias

Paroxysmal Atrial Tachycardia (PAT) with Block
Multi Focal Paroxysmal Atrial Tachycardia

Atrial Flutter

- Rapid, regular flutter waves at 250-350 / min
- Saw tooth 2, 3 aVf
- AV conduction is variable
  - 1:1 WPW with conduction over accessory pathway (wide QRS)
  - 2:1 untreated; physiologic block
  - 4:1 Treated
  - Higher degrees of block: AV nodal disease, drugs, increased vagal tone
- QRS usually normal
  - Can have aberration after long - short cycle
Atrial Flutter

- Macreentrant circuit (up the septum and down the right atrial free wall – typical)
- Treated with ablation
- Long term results
Atrial Fibrillation

- Rapid, irregular fib waves
- Atrial rate > 350
- Fib wave seen best in V1
- Irregularly irregular ventricular rate
- QRS usually normal
- If QRS is wide and rate > 200 then consider WPW with conduction over accessory pathway
AV Nodal Re-entrant Tachycardia (AVNRT)

- A PAC initiates atrial depolarizations which travel via the slow AV nodal pathway. (the fast pathway is refractory (blocked) due to previous SA node depolarization.)
- At the AV node exit depolarizations travel antegrade to depolarize the ventricles and retrograde up the fast pathway to depolarize the atria.
- This cycle repeats.
AV Nodal Re-entrant Tachycardia

[Diagram of cardiac conduction system]

AV Nodal Re-entrant Tachycardia

[Diagram of cardiac conduction system]
AV Nodal Re-entrant Tachycardia

• Most common supraventricular tachycardia
• Least likely to be life threatening
• **Narrow QRS has no visible P waves**
  • Simultaneous depolarization
• Or, P waves are so close to QRS they look like part of it (pseudo R waves in V1 and pseudo R waves in inferior leads)
Pseudo R Wave

Treatment for AVNRT

- Vagal (teach patient)
  - Valsalva
  - Carotid massage
  - Facial cold water immersion
- Adenosine or non-dihydropyridine calcium channel blockers (stable)
  - Adenosine preferred
  - Contraindications
  - Benefits of long acting agents
- DC Cardioversion (unstable)
Atrioventricular Reciprocating Tachycardias (AVRT)

- Requires the presence of a bypass tract or accessory pathway

- Most common: Kent bundles in “Wolf Parkinson White” Syndrome

- Left lateral free wall, right lateral free wall, and posterior septum
Concept of Pre-excitation

- Termed Pre-excitation because some conduction occurs via the Kent bundles in addition to the normal pathway; because conduction via the Kent bundles is faster than via the AV node the ventricles are pre-excited.

- This produces a “delta wave” on the EKG
- Fusion beat
  - Short PR
  - Wider than normal QRS

Delta Wave of Pre-excitation Syndrome

✓ 60 to 70% of WPW shows evidence in SR

Left sided accessory pathway:
Positive delta wave in V1

Right sided accessory pathway:
Negative delta wave in V1
Arrhythmias of WPW (AVRT or CMT)

Orthodromic SVT

Antidromic SVT

Atrial fibrillation
Atrioventricular Reentrant Tachycardia (AVRT)

- **Orthodromic**
  - Traveling down the AV junction and up an accessory pathway
  - Sequential depolarization
  - Narrow because travel via the AV node
  - More common than antidromic tachycardia

- **Antidromic**
  - Activation of the ventricles is initiated by impulses descending via an accessory pathway
  - Ventricular depolarization begins at an ectopic site in the myocardium and returns via the AV node

Presence of pre-excitation on 12 lead and paroxysmal palpitations.

Orthodromic Tachycardia

Orthodromic tachycardia occurs when the wave of electrical activation enters the ventricle normally through the AV node and returns to the atrium (retrograde) via the accessory pathway.

This allows the electrical impulse to re-enter the AV node and stimulate the ventricles once again.

Triggered by PAC conducted down AV node only (no prolonged AV interval) , leaves AP non refractory and ready for retrograde conduction.
Orthodromic Tachycardia

Negative P’ in lead 1 = left sided accessory pathway
Positive P’ in lead 1 = right sided accessory pathway

Orthodromic AVRT or AVNRT

• AVNRT
  • Simultaneous depolarization
  • P’ waves buried
  • Initial P’-R interval prolonged (.38 second)
  • Aberrancy Uncommon

• Orthodromic AVRT
  • Sequential Depolarization
  • Distinct P’ waves
  • Initial P’-R interval normal
  • Faster rate
  • Accessory pathway required
  • Persistent QRS alternans
  • Aberrancy Common
Antidromic Tachycardia

- The less common form of atrioventricular reentrant tachycardia
- The path of tachycardia passes from the atrium to the ventricle via the accessory pathway (Kent bundles) and returns to the atrium via the AV node
- The QRS complex is wide because antegrade conduction bypasses the AV node
- Antidromic tachycardia is very difficult to distinguish from ventricular tachycardia because ventricular depolarization begins where the accessory pathway enters the ventricle
  - Negative concordance will not be antidromic tachycardia

Antidromic Tachycardia

- Antidromic Tachycardia
- Antidromic Tachycardia

Antidromic SVT

AVN

AP

HB

V1
WPW and Atrial Fibrillation

• Mechanism of Action
  – Development of Atrial Fibrillation in WPW
    • 10-32% of patients
  – Refractory period of accessory pathway

• Danger

Example of WPW Atrial Fib
(antegrade conduction via accessory pathway)
Example of WPW Atrial Fib
(antegrade conduction via accessory pathway)

Treatment for WPW Tachycardias

- AV Reentrant (orthodromic)
- AV Reentrant (antidromic)
- Atrial Fib with antegrade conduction over accessory pathway

Slow conduction over accessory pathway:
- Amiodarone
- Procainamide
- Flecainide
- Sotalol
- Propafenone
Wide Complex Tachycardias

- Ventricular Tachycardia
- Torsades de Pointes
- Atrial Fib with antegrade conduction in WPW
- SVT with BBB Aberration
- AV Reentrant Tachycardia in WPW
Torsades De Pointes

- Recognition of this life-threatening arrhythmia is important because it is not treated like other VTs
- Two groups: Acquired and congenital
- Acquired
  - Drugs prolonging repolarization (class 1a, 1c, and sotolol)
  - Electrolyte abnormalities
  - Severe bradycardias
- Congenital
  - Brugada Syndrome
  - Hereditary long QT syndromes
Acquired Torsades De Pointes

- **Warning Signs:**
  - QT prolongation
    - Usually greater than 0.5 sec
    - Rate adjustment QTc
  - T Wave aberration (T wave alternans)
  - Prominent U waves
  - Pause dependent (couplet of PVCs)

- Short bursts: QRS peaks first appear to be up and then to be down (Can degenerate into V fib)
Torsades de Pointes

• Class I
  – Discontinue offending drugs
    • Note: Class IA drug induced TdP usually appears soon after the initial administration of the drug
    • www.qtdrugs.org
    • www.torsades.org
  – Correct electrolytes
    • Magnesium
    • Potassium
  – Temporary or permanent pacing
    • Symptomatic bradycardia
    • Heart block

Congenital Long QT: Torsades de Pointes

• Occurs with increase in sympathetic tone
• Usually terminates spontaneously (can degenerate into V fib)
• Treat:
  – Beta blockers
  – Permanent pacemakers
  – ICD
Polymorphic VT with normal QT:

- Seen frequently in ischemic conditions
- Treated like monomorphic VT

Brugada Syndrome

- Disorder of cardiac sodium channel
- ST elevation in anterior precordial leads
  - Intermittent
  - Present with provocative maneuver (Procainamide infusion)
- ECG can be dynamic
- Syncope
  - 2 year risk of SCD approximately 30%
- ICD recommended
Mechanisms of VT

- Disorder of impulse initiation
  - Abnormal automaticity
  - Triggered mechanism: disturbance in recovery or repolarization (less common)

- Disorder of impulse conduction
  - Reentrant Circuit (most common)
    - Reentrant Circuit within Myocardium
    - Bundle Branch Reentrant

Abnormal automaticity

- Phase 4
- Polymorphic VT/VF associated with acute conditions
  - Acute MI or ischemia
  - Electrolyte or acid/base disturbance; hypoxia
  - Markedly increased sympathetic tone
  - Benign PVCs, idiopathic VT

Triggered arrhythmias

- Early after-depolarization
- Congenital or acquired long QT syndrome
- Antiarrhythmic drug causing proarhythmia
- Ischemia
- Exercise-induced VT
- Delayed after-depolarization
- Digitalis toxicity

Reentrant ventricular arrhythmias

- Monomorphic VT in chronic, healed MI
- VF in healed MI
- Bundle branch reentry in dilated cardiomyopathy
Better Understanding Reentry

[Diagram of heart with infarcted areas and ECG tracings labeled I, II, III, aVR, aVL, aVF, V1, V2, V3, V4, V5, V6]
Bedside Cardiac Monitoring

- V1 and V6 are gold standard monitoring leads for ectopy versus aberrancy
- Bundle branch block patterns and ventricle ectopy can be differentiated by using the morphology of these leads.

DON'T rely on Lead II!!

Differentiating RBBB from Left Ventricular Ectopy

- Right Bundle Branch Aberration
- Left Ventricular Ectopy
Differentiating LBBB from Right Ventricular Ectopy

- **Left Bundle Branch Aberration**
- **Right Ventricular Like Ectopy**

Patient / History Assessment

- Suspect VT in patients with ischemia and / or reduced left ventricular function
- Don’t assume VT cannot be well tolerated!
- The rate, size of the heart and presence of additional complications are often more important than the source of the tachycardia

- Check the patient for responsiveness
- Check for BP for hemodynamic instability
- Check the 12 Lead to diagnose the rhythm
AV Dissociation

- Independent atrial and ventricular activity (AV dissociation) is diagnostic for ventricular ectopy
  
  Only seen in 30% VTs

- Ventricular tachycardia may also have retrograde P waves (retrograde P waves do not confirm VT)

Extreme Axis in VT
Methodology for Differentiation Using ECG

Step 1: Axis
Step 2: V1 (morphology)
Step 3: V6 (morphology)
Step 4: Back to V1 (confirm morphology)

Note: Bedside monitoring starts with step 2

Practice EKG
Practice EKG

Practice EKG
Practice ECG

QUESTIONS??

Thanks for Attending Cardiovascular Boot Camp

You may contact us at www.cardionursing.com
Dream