Knowing is not enough; we must apply. Willing is not enough; we must do.

Johann Wolfgang von Goethe
Methodology

Patient Information

Physiology and Pathophysiology

Rhythm Interpretation
Electrical Conduction Pathway

- SA Node
- Interatrial pathways
- AV Node
- Bundle of His
- AV Junction
- Right and Left Bundle Branches
- Anterior and Posterior Fascicles
- Purkinje Fibers
WAVES and COMPLEXES

- P wave: atrial depolarization
- QRS: ventricular depolarization
- T wave: ventricular repolarization
- PR interval: AV conduction time
- QRS width: intraventricular conduction time
- ST Segment: sustained ventricular depolarization
- QT interval: used to reflect ventricular repolarization time
Blocks and Bradys
Potential Causes of Bradycardias in Critical Care

- Propofol
- Cardiac disorders and medications
- Vasovagal
- CNS injury
- Hypothyroid
- Hypothermia
- Multiple other

Rule of thumb: Pace if cause cannot be reversed.
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
SA Block

Lead II  
1080 ms  2 x 1080 ms  
Sino-Atrial Exit Block (Type II)
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 tachyarrhythmia’s
- Bradycardia / tachycardia syndrome
  - Long pause after tachycardia (overdrive suppression)
  - Syncope

- 40% SSS: coronary atherosclerosis
- 5-10% SSS: idiopathic cardiomyopathy
Physiologically Based Question:

Why Can the P Wave Appear 3 Distinct Ways in Junctional Rhythm?
Physiological Critical Thinking

Question: Why can P wave appear 3 ways on ECG?

• P wave preceding QRS with short PR interval
  – Atria depolarizes from junctional impulse backwards
  – Atria depolarize before ventricles because the impulse fires before the AV node

• No P wave visible at all
  – Atria and ventricles depolarize at the same time because impulse fires very close to AV node

• P wave following QRS with short “R-P” interval
  – Impulse fires below AV node and ventricles depolarize first
  – Impulse has to travel retrograde through AV node before depolarizing atria
Junctional Escape Rhythm

- HR 35-60 beats per minute
- P waves may or may not be associated with QRS complexes
- QRS complexes same as sinus beats
Heart Blocks: AV Blocks

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

When the term heart block is used, clinicians are referring to block within, around, or below the AV node.
THE DEFINITION OF 2ND DEGREE AV BLOCK IS………………

One P Wave at a time fails to conduct to ventricle.
After you determine it is second degree heart block:

THEN YOU CAN ASK IF IT IS TYPE 1 OR TYPE 2.
What’s the difference between Type 1 and Type 2?

Not only is the rhythm strip criteria different. It also has to do with the most common physiological location of the block:

Type 1 block is usually within the AV node.
Type 2 block is below the AV node and usually involves both bundle branches.
PR Interval

• Reflects atrial depolarization (P wave) and time through the AV node (PR segment)

QRS Interval

• Reflects time through the His Purkinje system (His Bundle, Bundle Branches, and Purkinje Fibers)
Most often conduction fails within the AV node

• Therefore: There will be a problem with the PR interval.
• The QRS remains narrow (unless there is an existing BBB) because the block is in the AV node and does not involve the His Perkinje system.

Conduction fails below the AV node and usually involves both bundles

• There will be no problem with the PR interval (AV node not involved)
• QRS complex is usually wide (His Perkinje system involved)
  – Can be narrow if the block only involves the Bundle of His
Wenckebach
(2\textsuperscript{nd} Degree Type I)

- Sinus node fires regularly
- Disease is 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
  - PR problem because of physiological location of the block
- In absence of BBB QRS is normal
  - Normal QRS width because of physiological location of the block

- 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
  - Atropine will work because of physiological location
  - Atropine will only work where there are parasympathetic nervous system fibers. These fibers are present in the AV node.
Wenckebach (2\textsuperscript{nd} Degree Type I)

- Progressive lengthening of the PR interval (problem in the AV node)
- Normal QRS width (no problem in the His Perkinge System)
Wenckebach (2nd Degree Type I)

**Note:** This is a patient with a pre-existing RBBB who develops a 2nd degree Type I block in the bottom strip. Notice the lengthening PR interval (problem in the AV node) until there is a P wave that is not conducted. The QRS is wide in this patient due to the pre-existing RBBB, not due to the development of the 2nd degree heart block.
2nd Degree AV Block Type II

• No progressive lengthening of P-R interval
  – Physiological problem does not involve AV node
  – P-R interval is fixed with normally conducted beats

• Disease within the Bundle of His or below Bundle of His
  – QRS: wide – when below the Bundle of His
Both of these examples:
1. One P wave at a time fails to conduct
2. There is a fixed PR interval
3. There is a wider than normal QRS
2nd Degree Differentiation
HEART BLOCK WITH 2:1 CONDUCTION

When a second degree heart block conducts with a 2:1 conduction it is
difficult to determine if the block is within or below the AV node. The
reason is because you cannot see any consecutive lengthening or lack
there of regarding the PR intervals.

If a patient has been in a 2\textsuperscript{nd} degree Type I and develops a fixed 2:1 conduction
it is most likely still a 2\textsuperscript{nd} degree Type I. If the PR interval for the conducted beat
is prolonged and the QRS width is normal, then Type I is most likely.

If the patient is a 2\textsuperscript{nd} degree Type 2 with fixed 2:1 conduction look for:
- Normal P-R interval with conducted beats
- Wide QRS complex
Heart Blocks - High Grade AV Block

- Two or more consecutive atrial impulses are blocked.
- P waves: Regular, but 2 or > in a row fail to conduct to the ventricles.
- This is not 2\textsuperscript{nd} degree heart block by definition because more than one P wave in a row fails to conduct.
- In the strip below every third P wave is conducted. This differentiates High Grade AV Block from Complete Heart block which has no conducted P waves.
Third Degree AV Block – Complete

- No atrial impulses are conducted to the ventricles
- One form of AV dissociation
- Ventricular Rate: Maintained by ventricular escape (wide QRS) or by pacemaker coming from His bundle (narrow QRS – less common)
- Symptomatic if develops acutely
  - May be well tolerated if develops overtime
  - Treatment: Permanent Pacemaker
Remember – Patients in atrial fibrillation can develop complete heart block. The R to R interval becomes regular because the escape pacemaker is now in control.
Ventricular Escape Beats
Idioventricular Ventricular Rhythm

Ventricular escape rhythms are like having a ventricular pacemaker.
1st Question: What are the two reasons (physiological problems) that lead to a tachyarrhythmia?
Two Reasons Why a Patient Develops a Tachyarrhythmia

Ectopic Foci

• Focus other than sinus node takes over as pacemaker of heart.
• Enhanced or abnormal automaticity.

Conduction Disturbance

• An impulse does not travel normally down the conducting pathways but rather finds a way to re-enter the conducting pathways.
• Tachycardias with this etiology are called re-entrant tachycardias.
Understanding the Origin of Arrhythmias

- **Disorder of impulse initiation**
  - Abnormal automaticity
    - Enhanced
    - Abnormal
  - Triggered mechanism: disturbance in recovery or repolarization (less common)
    - Early or delayed after depolarizations

- **Disorder of impulse conduction**
  - Reentrant Circuit *(Most common)*
Two major categories of tachyarrhythmias by location:

Supra ventricular
Ventricular
Where (location) can an ectopic tachyarrhythmia start?
Difference Between Reentry and Abnormal Impulse Initiation

Ectopic focus in paroxysmal atrial fib (near pulmonary veins)

Ectopic focus in VT from ischemia or electrolyte abnormalities
Prerequisites for Re-entrant Tachycardias

• Available circuit
• Differing responsiveness in two limbs of circuit
  – Slow Pathway – conducts slow; recovers fast
  – Fast Pathway – conducts fast; recovers slowly
NEXT QUESTION?

What about reentry circuits: Where can they be?
Examples of Reentrant Tachycardias: Atrial
Examples of Reentrant Tachycardias: SVTs
Reentry in Monomorphomic VT
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
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
More on SVT

• Physiologically based question: If the origin of the tachycardia is above the AV node – will the QRS width be narrow or wide?
  • Narrow
  • Why?
  • Because conduction will enter AV node and travel normally through His Perkinje System

• What two circumstances would result in an exception to the rule?
  – Aberrant conduction due to a refractory BBB
  – Conduction over an accessory pathway
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
• Most common mechanism is re-entry

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

- **Focal ectopic atrial tachycardia**
  - Originate from a point source and activation spreads out from that source.
Atrial Tachycardia

- 1:1 P to QRS
- P wave different from sinus
- Rate up 250 minute
- Regular but may “warm up” at onset
- Normal QRS unless aberrant or bundle branch block
- P wave may be lost in preceding QRS or preceding T wave
- P not retrograde
Paroxysmal Atrial Tachycardia (PAT) with Block
Multi Focal Paroxysmal Atrial Tachycardia

- Wandering atrial pacemaker with increased rate
- P waves: three different shapes
- Absence of one dominant atrial pacemaker
- Rate > 100 per minute up to 250
- P-R interval and R-R interval vary
Multi Focal Paroxysmal Atrial Tachycardia
Organized Atrial Tachycardias: Reentrant Atrial Flutter

<table>
<thead>
<tr>
<th>Classifications</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Typical</strong></td>
</tr>
<tr>
<td>Confined to RA</td>
</tr>
<tr>
<td>Macro re-entrant circuit</td>
</tr>
<tr>
<td>Isthmus dependent</td>
</tr>
<tr>
<td>Counter-clock wise most common</td>
</tr>
<tr>
<td>May also be clockwise</td>
</tr>
</tbody>
</table>

60
The Isthmus

• Between tricuspid annulus – inferior vena cava
Typical Atrial Flutter: Clockwise Versus Counter Clockwise
Atrial Flutter

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

- Difficult to achieve rate and rhythm control with drugs

- Treatment with ablation
  - Approximately 20% to 25% of all ablations (more common than atrial fibrillation ablation)
  - Excellent short term success of up to 95%
  - At 5 years: Some reports of up to 70% recurrent either atypical atrial flutter or atrial fibrillation

Create bidirectional block through isthmus
Catheter Ablation of Atrial Flutter

• First line therapy for recurrent, typical isthmus dependent flutter
  — Excellent efficacy, low risk
• Subsequent atrial fibrillation can occur
• Non-isthmus dependent flutter is occasionally encountered, particularly in patients with prior atrial surgery
  — Efficacy is less predictable
Saw Tooth Pattern of Typical Atrial Flutter
Atrial Flutter 2:1 Conduction
It is important to recognize RBBB and LBBB morphology in lead V1 (and document) when patients are in SR. This skill allows you to better differentiate between VT and SVT with BBB (aberrancy) when the patient is in a wide complex tachycardia.

In the examples above the patient is in an atrial flutter. In the first strip the patient is conducting with a normal QRS width. The second strip the patient is now in a 2:1 atrial flutter with an increased ventricular rate resulting in the right bundle becoming refractory. Therefore the patient conducts with a RBBB.
The Lewis Lead

When P waves are not clearly seen in a rhythm strip (see lead 3 above), the Lewis lead can be very helpful in assessing for the presence of atrial activity.

As seen in the Lewis lead below, this patient is clearly in an atrial flutter. The atrial flutter is not as obvious in the lead III rhythm strip.
Lewis Lead
Lewis Lead

Telemetry Pack

Lead 1

Lead 1

SpO2

93

90
Atrial Lead: Atrial Pacing Wire
Additional Clinical Pearls with Atrial Flutter

COPD

- Patients on bronchodilators are at higher risk for developing 1:1 conduction

Left Ventricular Dysfunction

- Loss of AV synchrony and increased heart rate can lead to decompensation
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
PSVT

- Abrupt onset and termination
- Recurrent

Most commonly results from:

AVRT or AVNRT * (more common)

Termination with vagal maneuver: suggests re-entrant involving AV nodal tissue
AVNRT

- More prevalent in females
- Lone PSVT = no structural cardiac disease
- Most AVNRT is not associated with structural heart disease
- Rates are often 140-250 BPM
PSVT

• Symptoms
  – Palpitations
  – Dizziness
  – Neck pulsations
  – Fatigue
  – Lightheadedness
  – Chest Discomfort
  – Dyspnea
  – Presyncope
  – Rarely Syncope
AV Nodal Re-entrant Tachycardia (AVNRT) – Typical

- 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.
Typical AV Nodal Re-entrant Tachycardia
Typical AV Nodal Re-entrant Tachycardia
Typical AV Nodal Re-entrant Tachycardia
Typical AV Nodal Re-entrant Tachycardia
Electrophysiological Mechanism of AVNRT

A

Atrium

α
(slow pathway)

β
(fast pathway)

Ventricle

B

Atrium

α
(slow pathway)

β
(fast pathway)

Ventricle
AV Nodal Re-entrant Tachycardia

[Diagram showing the AV node and pathways, with labels for normal sinus rhythm and AV node reentry circuit.]
AV Nodal Reentrant Tachycardia (Typical)

• 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
Pseudo S Wave
SVT

Pseudo S

Pseudo S

Pseudo S

Pseudo R'

Sinus Rhythm

II

aVF

II

daVF

III

V1

III

V1
AVNRT
AV Nodal Reentrant Tachycardia

May also have fast – slow conduction
5 to 10%; long R-P
Treatment for AVNRT

• Vagal (teach patient)
  – Valsalva
  – Carotid massage
  – Facial cold water immersion
• Adenosine or non-dihydropyridine calcium channel blockers (stable)
  – Adenosine preferred
• DC Cardioversion (unstable)

Ablation: AVNRT is most common reason for cardiac ablation.
AVRT
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
**Concealed Accessory Pathway**

- **AVN**
- **AP**
- **HB**

- **Action**: Concealed AV connection (no antegrade conduction)
- **ECG**: Ventricles activated by normal H-P system (QRS narrow)
- **Comments**: Fusion of conduction over AV connection and dominant over AV node (QRS minimally wide)

**Manifest Accessory Pathway**

- **AVN**
- **AP**
- **HB**

- **Action**: Conduction through accessory pathway
- **ECG**: More pre-excited
- **Comments**: More of ventricle activated via the AV connection (QRS even wider)

**Concealed Accessory Pathway**

- **AVN**
- **AP**
- **HB**

- **Action**: Concealed AV connection (no antegrade conduction)
- **ECG**: Minimal pre-excited
- **Comments**: Fusion of conduction over AV connection and dominant over AV node (QRS minimally wide)

**Manifest Accessory Pathway**

- **AVN**
- **AP**
- **HB**

- **Action**: Conduction through accessory pathway
- **ECG**: Maximal pre-excitation
- **Comments**: Ventricle activated only by AV connection (resembles PVC)

**Electrophysiological Characteristics**

- **PR ≤ 120 ms**
- **Delta wave present**
- **QRS ≥ 120 ms**
- **Repolarization abnormal**

- **PR normal**
- **Delta wave absent**
- **QRS normal**
- **Repolarization normal**
Arrhythmias of WPW
(AVRT or CMT)
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)

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

Aberrancy Uncommon

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
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.
WPW and Atrial Fibrillation

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

• Danger

Danger!
AF in WPW

Accessory Pathway
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
- Propofenone
- Ibutelide
Catheter Ablation of Accessory Pathway in Wolff-Parkinson-White Syndrome
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