An Introduction to Tachyarrhythmias
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Things to keep in mind when analyzing arrhythmias:
- Electrical activity recorded in 12 and 15 leads
- Examine lead with max information
- One lead is NEVER enough
- Analysis requires systematic approach →
  - Rate
  - Rhythm
  - P wave → atrial depolarization
  - PR interval → time for impulse to reach ventricles
  - QRS interval/complex → time for impulse to travel through ventricles
  - ST segment
  - T wave → repolarization of ventricles
  - U wave → papillary repolarization/ventricular recovery
  - QT duration → ventricular systole
- There can be ‘issues’ at every level
  - SA node
  - Atria
  - AV node
  - His Bundle
  - Bundle Branches

Supraventricular vs. Ventricular
- General rule of thumb
  - Generated from above the AV node → Narrow Complex
    → Supraventricular
  - Generated from below the AV node → Wide Complex → Ventricular
- EXCEPT if there is disease at or below the AV node

Two mechanisms:
1. Ectopic – enhanced automaticity or triggered
   - ie. atrial or AV junctional
2. Reentry – requires an ‘arena’ → Available circuit, Differing responsiveness,
   Varying conduction
   - ie. SA nodal, atrial, AV junctional/nodal, AV junctional/nodal bypass,
     Wolff-Parkinson-White
Supraventricular Arrhythmias

1. SA nodal – circuit travels through SA node and atria
2. Atrial – circuit travels through atria (detailed below)
3. AV junctional/nodal – circuit travels within AV node and travels back to atria and down AV node to ventricle
4. AV junctional/nodal bypass – circuit uses bypass tract and travels either antegrade or retrograde through AV node
5. Wolff-Parkinson-White – circuit uses Kent bypass tract and travels retrograde through AV node (could be either antegrade or retrograde through AV node giving either narrow or wide complex QRS morphology, respectively)

Irregular Narrow Complex Arrhythmias

1. Atrial Fibrillation
   - Characterized by chaotic atrial activity → fibrillatory waves
   - Irregular, narrow QRS, unless (bundle disease or extreme rates)
   - Preceded by SV ectopic beats, PSVT, paroxysmal atrial fibrillation
   - Associated with left and right atrial enlargement usually, unless focus is in pulmonary vein – atrial junction
   - Classified based on:
     1. Duration – Acute – 24 - 48 hours
        - Chronic – persistent, paroxysmal, permanent
     2. Lone – no associated structural heart disease or risk factors
     3. Rate – Uncontrolled vs. Controlled

2. Atrial Flutter
   - Usually paroxysmal, unusual to be chronic
   - Macro-reentry circuit in right atria
   - Ventricular rate – 140-150 depending on AV conduction (2:1, 4:1, 6:1)
   - Atrial rate – 240 - 340 (modified by size of circuit and drugs)

   ❖ Ventricular rate for fibrillation and flutter can be regular if there is disease in the AV node

Management of Atrial Fibrillation/Flutter

IF SYMPTOMATIC OR HEMODYNAMICALLY UNSTABLE (Chest pain, hypotension, shortness of breath, pulmonary edema) → DIRECT CURRENT CARDIOVERSION

Anticoagulation

Stratify risk of stroke and vascular events and bleeding risk

<table>
<thead>
<tr>
<th>High-Risk Factors</th>
<th>Moderate-Risk Factors</th>
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<tbody>
<tr>
<td>History of stroke/TIA</td>
<td>Age 65-75 years</td>
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<tr>
<td>HTN</td>
<td>Diabetes</td>
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<tr>
<td>Reduced LV function</td>
<td>CAD without LV dysfunction</td>
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<tr>
<td>Age &gt; 75 years</td>
<td>-</td>
</tr>
<tr>
<td>Mitral Stenosis</td>
<td>-</td>
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<tr>
<td>Prosthetic heart valve</td>
<td>-</td>
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</tbody>
</table>
Any high-risk factors or > one moderate-risk factor → Warfarin
One moderate-risk factor → ASA 75-325 mg/day or Warfarin
No high or moderate-risk factors → ASA 75-325 mg/day, unless excessive bleeding risk

Pharmacological Options
Rhythm Control:
- Structurally normal hearts: Propafenone, Sotalol, Flecainide
- CAD with normal EF: Sotalol
- LV Dysfunction and/or serious structural heart disease: Amiodarone
**Cautioned use of Sotalol in females who are > 65 years taking diuretics or renal insufficiency → Increased incidence of Torsades de Pointes

Rate Control:
- Active and young patients: Non-dihydropyridine calcium channel blockers or b-Blockers
- Heart Failure: b-Blockers + Digoxin
- Pre-excitation over an accessory pathway: Procainamide (acutely)
- Consider pacemaker and AV nodal ablation for patients with persistent symptoms due to rapid or irregular ventricular rate where drug therapy poorly tolerated or ineffective

Regular Narrow Complex Arrhythmias

1. Sinus tachycardia
   - P wave morphology upright in I and II
   - Physiologic response to stress or increase sympathetic tone
   - Treatment of arrhythmia involves treatment of the underlying cause
2. SA nodal
   - Circuit travels through SA node (slow arm of reentry loop) and atria
   - Thought to present on surface EKG as PSVT; usually rate < 150 bpm with wide fluctuations in rate, but most do not last longer than 10 to 20 beats
   - Bursts repetitive and sensitive to autonomic tone
   - P waves appear to be sinus, but not always identical
   - Often difficult to differentiate from sinus arrhythmia, sinus tachycardia, atrial tachycardia
   - Acute setting, if symptomatic, should respond to vagal maneuvers and adenosine
   - Chronic setting the use of radiofrequency ablation to alter circuit (uncommonly required)
3. AV junctional/nodal
   - Circuit travels within AV node and travels back to atria and down AV node to ventricle
   - Heart rate usually between 170 – 250 bpm, can be regular or irregular
- P waves buried in QRS and negative in II, III, AVF and positive in V1
- Usually self-limiting
- Easily terminated with vagal maneuvers, if not use of adenosine to break reentry
- If these do not work the use of pharmacology to slow conduction through the AV node, ie. Calcium channel blockers, B-Blockers, Procainamide, or electrical cardioversion

4. AV junctional/nodal bypass
   - Circuit travels antegrade through AV node and retrograde through bypass tract
   - Usually initiated by premature atrial beat
   - Heart rate usually between 170 – 250 bpm, can be regular or irregular
   - P waves are always separate from the QRS with a 1:1 conduction; of P wave negative in I is diagnostic of accessory pathway
   - May lead to life-threatening atrial fibrillation when rates > 200 bpm
   - Treatment is similar to AV junctional/nodal tachycardias

5. Atrial tachycardia
   - P wave morphology can be inverted/biphasic in II and III with a rate >100 bpm
   - 6 different classifications/mechanisms of which MAT is the most easily diagnosed
   - Multifocal atrial tachycardia
     i. Three or more P wave morphologies with a rate >100 bpm
     ii. Often seen in patients with pulmonary disease (Pneumonia, COPD)
     iii. Treat underlying pathology

Wide Complex Supraventricular Tachycardias

1. Atrial Fibrillation with a bundle branch block vs. accessory pathway
   - Differentiated based on rate and QRS pattern

2. Antidromic Circus Movement Tachycardia
   - Uses accessory pathway in anterograde direction
   - Difficult to differentiate from VT from surface EKG
**Case Example #1:**

73 year young male with known ischemic heart disease presented with this wide complex tachycardia. Atrial Fibrillation with RBBB. Tachycardia appears regular early in EKG tracing. Note the break in the tachycardia at the end of the tracing with no notable P waves.

**Case Example #2:**

70 year old presented with palpitations, normotensive, & mild SOB. Wide complex tachycardia with a rate between 150 - 170
Given intravenous Verapamil and this was the EKG to follow. Ventricular rate increased significantly and became hypotensive.

Subsequently, ventricular rate increased to ~300 bpm with drop in pressure; Required immediate cardioversion.
When in Doubt with a Wide Complex Tachycardia??

- Never use vagal maneuvers or adenosine, these maneuvers are acceptable if Atrial Fibrillation can be ruled out definitively.
- Never use Calcium Channel Blocker
- Never use Digitalis with Calcium Channel Blocker when rate > 200 beats/min and irregular

*These maneuvers/medications block the AV node and allow for the rapid conduction down the accessory pathway which increases the chances of the rhythm degenerating into ventricular fibrillation, if the underlying rhythm is atrial fibrillation with use of an accessory pathway!!!*

**When in Doubt??**

Systematically, evaluate

**History**

- Ischemic heart disease → Assume arrhythmia to be ventricular in origin until proven otherwise

**Physical signs of AV dissociation**

- Sensitivity/specificity low
- Irregular cannon ‘a’ waves
- Varying intensity of 1st heart sound
- Beat to beat changes in systolic blood pressure
EKG CRITERIA → Suggestive of Ventricular Origin

AV dissociation

QRS > 140 msec in + V1 and > 160 msec in − V1

Notched or slurring downslope on S or QS in V1 or V2

Ventricular-Atrial conduction – uncommon > 200/min

Capture/Fusion Beats

Extreme axis

Left peak taller in V1 RBBB

Monophasic or diphasic in V1, not triphasic

Negative V1 and V6

> 70 msec from onset of ventricular complex to nadir – caution

QRS pattern differs from BBB pattern

Ventricular Arrhythmias

1. Monomorphic Ventricular Tachycardia

   I. Accelerated Idioventricular Rhythm

   • Results more than likely to an enhanced-automaticity

   • Self-terminating, usually asymptomatic, requiring no interventions

   • Seen post-infarction and is thought to be a sign of reperfusion
II. Ventricular Tachycardia  
   a. Monomorphous
      • Focus is close to or within conduction system or use the intraventricular conduction system for a reentry circuit
      • Seen with ischemic heart disease or serious structural heart disease including nonischemic cardiomyopathies
      • Seen during ischemia
      • Often seen in idiopathic VT, usually occurs at rest, and is characterized by frequent PVB
   b. Dimorphous
      • Bidirectional – does not imply mechanism, but describes alternating polarity of wide ventricular complexes, some are truly ventricular in origin, but often is a SVT with a RBBB and alternating hemiblock, seen often with digitalis toxicity
      • Alternating – alternating amplitude
   c. Polymorphus (Please see handout entitled ‘Drug-Induced Long QT Syndrome’)
      • A disorder of myocardial repolarization
      • QRS complexes are variable

Note the widening of the QRS complex on the 5th beat and loss of P waves on the 6th beat. The AIVR usurps the sinus rhythm and a rate slightly faster. The P waves appear to be buried in the R deflection of the widened QRS.
• Often self-terminating, but if prolonged will degenerate into fibrillation
• More ominous prognosis than sustained monomorphic VT
• Can have normal and prolonged QT interval
• Long QT interval – Acquired or Congenital
• Known as torsade de pointes (TdP), ‘Twisting of the points’, term is used when polymorphic VT is associated with prolonged QT interval
• Usually associated with bradycardia
• Short-long cycles 2° VPBs \(\Rightarrow\) The Short and Long of It. . . . .

Commonest causes
• Medications (dosage, infusion rate, concurrent)
• Electrolyte disorders

Others
• Structural heart disease
• Stroke + brain injury
• HIV
• Eating disorders

Management
• Revascularization for non-Torsades polymorphic Ventricular Tachycardia
• Correction of Electrolytes
• Remove offending agents
• Overdrive pacing/Isoproterenol/Magnesium
III. Ventricular Flutter/Fibrillation

*Management for Monomorphic and Ventricular Flutter/Fibrillation*

- Cardioversion/Defibrillation – if symptomatic
- Amiodarone/Lidocaine/Procainamide/bBlocker