Tachyarrhythmias in the ICU

ACNP/PA Critical Care Boot Camp
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Ariel Kappa
RN, MSN, ACNP-BC
• “Acute Arrhythmias are the gremlins of the ICU because they pop up unexpectedly, create some havoc, and are often gone in a flash...”

  -Paul Marino
Objectives

• Review
  – EKG characteristics
  – Initial Considerations
  – Pharmacology

• Interactive Case Studies
Stable or Unstable (Shock ?)

Regularity? Appreciable P waves?

Morphology of QRS

Blocks? Infarction?
Initial considerations

• Stable?
  – Hypotension, AMS, signs of shock, CP
  – 12-lead EKG

• Pharmacological management

• Non-Pharmacological management
  – Synchronized cardioversion (50-200 J)
    • Oxygen, airway, monitor, sedation
<table>
<thead>
<tr>
<th>Class</th>
<th>Common Examples</th>
<th>Mechanism</th>
<th>Clinical Use</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ia</td>
<td>Quinidine, Procainamide</td>
<td>Na+ Channel blocker; Fast (effects QRS)</td>
<td>Pre-excited afib, Stable monomorophic VT</td>
</tr>
<tr>
<td>Ib</td>
<td>Lidocaine, Mexilitine, Phenytoin</td>
<td>Na+ Channel blocker; No effect on conduction; may prolong APD</td>
<td>VT</td>
</tr>
<tr>
<td>Ic</td>
<td>Fleicanide, Propafenone</td>
<td>Na+ Channel blocker; (no effect on QRS)</td>
<td>SVT, PVCs, WPW</td>
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<tr>
<td>II</td>
<td>Metoprolol, Esmolol, Propranalol</td>
<td>Block beta-adrenergic receptors</td>
<td>Afib rate control, Narrow Stable SVT</td>
</tr>
<tr>
<td>III</td>
<td>Amiodarone*, Sotalol*, Ibitulide</td>
<td>K+ channel blocker</td>
<td>SVT (Regular and Irregular)</td>
</tr>
<tr>
<td>IV</td>
<td>Verapamil, Diltiazem</td>
<td>Non-dihydropyridine Calcium channel blocker, vasodilate, negative inotrope</td>
<td>Rate control SVT *Avoid in CHF/VT Pre-excited Afib</td>
</tr>
<tr>
<td>Misc.</td>
<td>Digoxin, Adenosine Magnesium</td>
<td>Dig- parasympathetic ↓ AV, + Inotrope Adenosine- ↓ AV Mag- effect Na/K transport</td>
<td>Dig- SVT, Afib in HF, Adenosine- SVT, stable WCT Mg- Polymorphic VT/Torsades</td>
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Case 1

- 73 yo female with history of HTN, Aortic Stenosis, DMII, COPD

- POD 1 s/p AVRt with EF of 60% c/o SOB.

- Vitals: HR: 162, SBP: 131/71, RR: 26
Case 1

- What does this EKG show?
- 1. Atrial tachycardia
- 2. AV nodal reentrant tachycardia
- 3. Atrial fibrillation
- 4. Atrial flutter
What is the best next step?

• 1. Prepare for synchronized cardioversion of atrial fibrillation.
• 2. Initiate IV amiodarone bolus and infusion.
• 3. Assess timing, electrolytes and consider IV diltiazem bolus and infusion.
• 4. Administer 5mg IV metoprolol.
Rate vs. Rhythm control

• **AFFIRM TRIAL**
  • True or False:
    • Rhythm control is more important than rate control
  • 1. True
  • 2. False

• **RACE II**
  • True or False:
    • HR less than 110 is non-inferior to HR less than 80 in morbidity/mortality outcomes
  • 1. True
  • 2. False

(AFFIRM, NEJM, 2002), (VanGelder et al., NEJM, 2010)
Atrial Fibrillation

EKG Characteristics: Irregularly Irregular.

Etiology: Numerous waves of depolarization spreading throughout the atria, leading to an absence of coordinated atrial contraction.

Treatment: Class II, Class IV, Class III, Digoxin: HF (inotropic support). Unstable: Low voltage DCCV

AFFIRM trial (NEJM, 2000): Rate control not rhythm control

Race II Trial: (NEJM, 2010): Lenient rate control
Case 2

• 68 yo male with COPD exacerbation admitted to MICU.

• HR: 151, SBP: 121/73, RR: 28
Case 2

What does this EKG show?

1. Atrial tachycardia
2. Sinus tachycardia
3. Atrial fibrillation
4. Atrial flutter
Atrial Flutter

EKG Characteristics: ‘Sawtooth’ pattern: atrial rate ~300, ventricular rate 75-150 bpm (unless irregular). Usually with 2:1/4:1 block at AV node. Rhythm constant amplitude, morphology, duration. **May be variable and irregular

Etiology: Reentrant circuit in the wall of the atrium

Treatment: Class III (Ibutilide, sotalol, amiodarone): prolong refractory period (not slowing conduction) *Small risk for torsades. Ventricular rate control can be difficult, AV nodal blockers prevent 1:1 conduction. Unmasking of flutter waves with adenosine.

*Nonpharmacological: Rapid pacing or low voltage DC cardioversion is effective.

(Blomström-Lundqvist, et al., 2003)
Case 3

- 24 yo female admitted to Trauma ICU with pelvic fracture s/p MVC complaining of palpitations.

- Vitals: HR: 148, SBP 118/58, RR: 22
Case 3

What does this EKG show?

1. Sinus Tachycardia
2. AV nodal reentrant tachycardia
3. Atrial fibrillation
4. Atrial flutter
AV Nodal Reentrant Tachycardia (AVNRT)

- **EKG characteristics**: Rate 140-280, *P* wave undetected (activation atria/ventricle simultaneously),
- **Etiology**: Triggered by PACs, “Micro” Reentry at AV node (atrial stretch, inflammation, irritability- catecholamines)
- **Treatment**: Vagal maneuvers, Adenosine 6-12 mg IV push, Ca++ channel blockers, Digoxin, Beta blockers

(Blomström-Lundqvist, et al., 2003)
Multifocal Atrial Tachycardia (MAT)

• **EKG characteristics**: at least 3 P wave morphologies, variable intervals P-P, R-R, P-R, look for isoelectric baseline

• **Etiology**: No single dominant pacemaker, multiple atrial foci fire independently. COPD/CHF

• **Treatment**: Treat underlying cause (electrolyte derangement, hypoxemia). Rate control- Class IV- CCBs, Class II- Beta-blockers

• **PACs**
Atrioventricular Reentrant Tachycardia (AVRT)

- **Etiology:** Macroreentry through normal conduction system with accessory AV pathway; Delayed activation of atria = visible P wave
- **Treatment:** Similar to treatment of AVNRT, AV nodal blockers, eventual ablation
**Sinus Tachycardia**

EKG characteristics:
- Constant PR interval
- Varies with stimulation/respiration
- Normal Rate 220 bpm – age (yrs)

Etiology: Physiologic

Treatment: Fix underlying physiologic insult
- (Fever, anxiety, thyrotoxicosis, exogenous catecholamines, anticholinergic, LV dysfunction - MI etc.)

**Atrial Tachycardia**

EKG characteristics:
- Atrial rate 150-250
- Distinct P wave morphology

Etiology: CHF, HTN, electrolyte abnormalities

Treatment: Adenosine, Class II, Class III, *Dig toxicity?
Case 4

- 38 yo male with PMH: Appendectomy
- Presented to ED with SBO s/t incarcerated groin hernia now POD 3 s/p SBO repair.
- Vitals: HR 148, SBP 100/58, RR: 20
- BMP:

<table>
<thead>
<tr>
<th>Value</th>
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<tbody>
<tr>
<td>148</td>
<td>19</td>
<td>41</td>
</tr>
<tr>
<td>2.1</td>
<td>21</td>
<td>1.6</td>
</tr>
</tbody>
</table>
Case 4
What is your next course of action?

• 1. Correct hypokalemia and prepare amiodarone for administration.
• 2. Grab the crash cart and prepare for emergent cardioversion.
• 3. Correct hypokalemia and prepare lidocaine for bolus/infusion.
• 4. Grab the crash cart and ask the nurse to give sotalol.
Monomorphic VT

EKG characteristics: 3 consecutive beats >100 bpm, QRS >120ms
Brugada criteria: Precordial leads- No RS complex or RS >100ms, AV dissociation, QRS morphology- Fusion beats
Etiology: CAD, CM, Ischemia
Treatment: Correct aggravating conditions (hypokalemia, ischemia)

(Zipes et al., 2006)
**Wide Complex SVT**

- **EKG characteristics:** Regular, Wide complex, Fails Brugada’s Criteria (from previous slide)
- **Etiology:** BBB/IVCD, Pre-excitation, Presence of PPM/ICD
- **Treatment:** WPW- Procainamide (*Avoid AV nodal blockers)
- Vagal maneuvers, Adenosine, Class III, Cardioversion

![EKG Image]
Which of these below is **not** a treatment option?

- 1. DC Cardioversion
- 2. Metoprolol
- 3. Amiodarone
- 4. Procainamide
Case 5

• Lets go back to the 38 yo. male with VT

• Unfortunately you look up at the monitor and see this...
What do you do next?

1. Give magnesium
2. Defibrillate
3. Stop all QT prolonging medications
4. All of the above.
Irregular, Wide Complex

- **Polymorphic VT**
  - Etiology: Ischemia, Catecholamines
  - Treatment: Defibrillation

- **Ventricular fibrillation, Torsades de pointes**
  - Etiology- QT prolongation, Class I, III- prolong refractory period
General Tips

• Narrow complex tachyarrhythmias can be diagnosed or possibly terminated in most cases with **IV adenosine**.

• If the origin of arrhythmia is unclear on a symptomatic patient, it is generally consider **safe to treat as ventricular** in origin.
References


• Dubin, Dale. (2000). Rapid Interpretation of EKGs: Dr. Dubin’s classic simplified methodology for understanding EKGs, 6th Ed. Location. C.o.v.e.r.


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