Acute Diagnosis and Treatment of Common Dysrhythmias

ACNP/PA Critical Care Boot Camp
Vanderbilt University Medical Center
September 9, 2014

Ariel Kappa
RN, MSN, ACNP-BC
Objectives

• Review
  – Cardiac conduction cycle
  – EKG

• Dysrhythmia Diagnosis and Treatment
  – Characteristics
  – Etiology
  – Pharmacological Management

• Case Studies
(Cardiac Muscle Cell)

+ 10 mV

Phase 0
Depolarization
Na⁺ Enters

- 90 mV

Phase 1

Phase 2

Phase 3
Repolarization
K⁺ is extruded

QRS

T
Blocks?
Infarction?

Rhythm (P waves, QRS)

Rate (Fast or Slow)

Stable or Unstable (Shock ?)

Blocks? Infarction?
Mechanism of Dysrhythmias

- Automaticity
- Ectopic foci / Escape beats
- Reentry / Conduction block
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 on QRS)</td>
<td>Pre-excited afib, Stable monomorphic 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>Flecanide, Propafenone</td>
<td>Na+ Channel blocker; (no effect on QRS)</td>
<td>SVT, PVCs, WPW</td>
</tr>
<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*, Ibutilide</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>
</tr>
</tbody>
</table>
Regular Narrow Complex

- Sinus Tachycardia
- Atrial Tachycardia
- Atrioventricular Nodal Tachycardia (AVNRT)
- Atrioventricular Tachycardia (AVRT, Orthodromic)
- Atrial flutter
**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?*
AV Nodal Reentrant Tachycardia (AVNRT)

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

- **Etiology**: Macroleentry 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
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.
Regular Wide Complex

- Ventricular Tachycardia (VT)
- Wide complex SVT
  - Bundle branch block
  - Antidromic- Wolf-Parkinson-White (WPW)
  - Pacemaker-mediated
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

**Etiology:** CAD, CM, Ischemia

**Treatment:** Correct aggravating conditions (hypokalemia, ischemia) *Wide QRS presumed VT if unclear (LOE: C). *DCCV unstable (LOE: C). Class Ia: Procainamide- careful in LV dysfunction, CHF, hypotension- prolong QT. Class III- Sotalol, Amiodarone (benefit IV to PO). Class IIb- IV lidocaine initial treatment associated with MI (LOE: C).
Wide Complex SVT

- EKG characteristics: Regular, Wide complex, Fails Brugada criteria
- Etiology: BBB/IVCD, Preexcitation, Presence of PPM/ICD
- Treatment: WPW- Procainamide (*Avoid AV nodal blockers)
- Vagal maneuvers, Adenosine, Class II, Class III, Cardioversion
Irregular Narrow Complex

• Atrial fibrillation

• MAT

• Sinus Tachycardia w/ Premature atrial contractions (PACs)

• SVT with block
Atrial Fibrillation

EKG Characteristics: Irregularly Irregular.

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

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
Irregular, Wide Complex

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

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

Regular

Sinus Bradycardia, Junctional Bradycardia, Idioventricular escape rhythm, 1\textsuperscript{st} Degree AV block, 3\textsuperscript{rd} Degree AV block

Irregular

2\textsuperscript{nd} Degree Type I AV block, 2\textsuperscript{nd} Degree Type II AV block
Initial considerations

• Stable?
  – Hypotension, AMS, Light-headedness, syncope, angina

• Pharmacologic
  – Atropine (selective muscarinic antagonist)
  – Epinephrine, Dopamine

• Non-pharmacologic
  – Transcutaneous pacing > Transvenous
  – Expert consultation, PPM
Sinus Bradycardia

- **EKG Characteristics**: <60 bpm, normal P wave with PR < .20 prior to every narrow QRS
- **Etiology**: Normal variant, hypoxemia, Increased ICP, SSS, OSA, hypothermia, MI, Drugs
- **Treatment**: Symptomatic?
- CCB and Beta-blocker overdose- IV calcium, glucagon with or without insulin
**Junctional Escape**

**EKG Characteristics:** 40-60 bpm (regular w/ narrow QRS), No P waves, >60bpm = ‘accelerated’

**Etiology:** Escape rhythm with focus from AV node

**Treatment:** Usually stable.

---

**Idioventricular Escape**

**EKG Characteristics:** Regular rate 30-45 bpm, QRS wide (below AV node), >60bpm = ‘accelerated’

**Etiology:** Focus in His-bundle branch system

**Treatment:** Pacing
1\textsuperscript{st} Degree AV block

EKG Characteristics: Prolongation of the PR interval, which is constant. All P waves are conducted.

Etiology: Usually benign

Treatment: None

3\textsuperscript{rd} Degree AV block

EKG Characteristics: No relationship between P waves and QRS, atrial rate faster than ventricular rate, PP intervals and RR intervals remain regular and constant.

Etiology: Inferior MI

Symptoms: Syncopal symptoms, angina, CHF

Treatment: Transcutaneous Pacemaker
**2\textsuperscript{nd} Degree AV Block** *(Mobitz I, Wenckebach)*

**EKG Characteristics:** Progressive prolongation of the PR interval until a P wave is not conducted. PR interval prolongs, the RR interval actually shortens, Narrow QRS.

**Etiology:** High AV nodal block. Usually benign unless associated with underlying pathology, i.e. Inferior MI, Toxicity (beta-blockers, CCBs).

---

**2\textsuperscript{nd} Degree AV Block** *(Mobitz II)*

**EKG Characteristics:** Constant PR interval with intermittent failure to conduct. Rhythm is dangerous as the block is lower in the conduction system.

**Etiology:** Infranodal His-Purkinje system conduction delay. Inferior/ Anterior MI-fibrotic disease of the conduction system.
**Left Bundle Branch Block**

- QS or rS complex in lead V1 and a monophasic or notched R wave in lead V6.

**Right Bundle Branch Block**

- R-wave (R’) in V1, and a slurred S-wave in V5 - V6.

**Etiology:**
- **Left Bundle Branch Block:** CAD, HTN, CM, Aortic valve disease
- **Right Bundle Branch Block:** Congenital, MI, PE
Case Study # 1

- 78 yo female POD 3 s/p AVRt.

- Patient c/o palpitations.

- Vitals HR: 160s, SBP: 119/65, SPO2: 95% 4 LNC
Case Study # 2

• 54 yo male POD 1 s/p TURP recovering on stepdown. RRT for Chest Pain.
• Pt c/o SOB. Diaphoretic
• HR: 41, SBP: 108/69, SPO2: 96% 2L NC
Case Study # 3

44 yo male admitted to observation unit for SOB, dyspnea...

HR 174, SBP 92/60, SPO2 94% NRB
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.

References Cont.


- Robert W. Neumar, Chair; Charles W. Otto; Mark S. Link; Steven L. Kronick; Michael Shuster; Clifton W. Callaway…Laurie J. Morrison. Part 8: Adult Advanced Cardiovascular Life Support 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. *Circulation 2010,* 122, S729-S767. DOI: 10.1161/CIR.0b013e31820ff511.