Dysrhythmias

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Dysrhythmias

I. INTRODUCTION

Review basic dysrhythmias. Questions on PCCN exam will be related to rhythm identification, cause or appropriate treatment. ACLS is not listed on the blueprint but identifying and treating life-threatening dysrhythmias is the major reason to place a patient on telemetry.

II. CARDIAC ELECTROPHYSIOLOGY

Impulse Conduction & Pathways
SA Node → Internodal Pathways (atrial contraction) → AV Node (delay) → His-Purkinje System (ventricular contraction)

![Heart Diagram with Conduction Pathways]
III. DYSRHYTHMIAS

Common Causes

a. Decreased Coronary Perfusion (CAD)
b. Impaired Myocardial Oxygen Delivery (hypoxia)
c. Electrolyte Disturbances
d. Cardiac Muscle Injury
e. Ischemia or Infarction
f. Defects in the Heart Muscle or Electrical System
g. Cardiac Surgery
h. Electrical Stimulation to the Heart Muscle
i. Medications

Lead Selection for Monitoring

AACN Practice Alerts for ECG Monitoring
a. Dysrhythmia Monitoring (4/08)
b. ST Segment Monitoring (5/09)

AHA/ACCF/HRS Recommendations for Standardization & Interpretation of the Electrocardiogram
a. I ECG Technology ‘07
b. II ECG Diagnostic Tests ‘07
c. Pre-hospital ECG and ACS ‘08
d. III Intraventricular Conduction Disturbances ‘09
e. ST-Segment, T, and U Wave and QT Interval ‘09
f. Hypertrophy Evaluation ‘09
g. Acute Ischemia/Infarction ‘09
h. Prevention of Torsade de Points 2/8/10

<table>
<thead>
<tr>
<th>Monitoring Purpose</th>
<th>Lead Recommendation</th>
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<tbody>
<tr>
<td>Dysrhythmia Detection</td>
<td>• Aberrancy vs Ectopy: V₁ or V₆ (if V₁ not available)</td>
</tr>
<tr>
<td></td>
<td>• AFib/flutter: II, III, aVF : Monitoring of P wave Problems;</td>
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<tr>
<td></td>
<td>whichever lead allows best visualization of fib/flutter waves;</td>
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<td></td>
<td>consider atrial ECG for post-cardiac surgery patients if pacer wires in place,</td>
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<tr>
<td></td>
<td>could try Lewis Lead RA electrode at 4th ICS Rt Sternal boarder, LA on back at Rt</td>
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<tr>
<td></td>
<td>side of spine read Lead I</td>
</tr>
<tr>
<td></td>
<td>• Junctional Rhythms: Lead II</td>
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<tr>
<td></td>
<td>• Bundle Branch Blocks V₁ and/or V₆</td>
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<tr>
<td>ST Segment Monitoring</td>
<td>• Unknown Problem III or V₃</td>
</tr>
<tr>
<td></td>
<td>• Right coronary artery: III or aVF</td>
</tr>
<tr>
<td></td>
<td>• Left anterior descending/Circumflex: V₃</td>
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<tr>
<td></td>
<td>• Activity-induced ischemia (no specific vessel identified): V₅</td>
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<tr>
<td></td>
<td>• Pt “Finger Print” from Known Ischemic Changes</td>
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### Dysrhythmias

<table>
<thead>
<tr>
<th>Inferior Wall</th>
<th>II, III, AVF</th>
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<tbody>
<tr>
<td>Anterior Wall</td>
<td>V&lt;sub&gt;1&lt;/sub&gt;-V&lt;sub&gt;4&lt;/sub&gt;</td>
</tr>
<tr>
<td>Lateral Wall</td>
<td>V&lt;sub&gt;5&lt;/sub&gt;, V&lt;sub&gt;6&lt;/sub&gt;, I, aVL</td>
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<tr>
<td>QTc</td>
<td>Identify 12-lead with most well-defined T wave</td>
</tr>
<tr>
<td></td>
<td>V&lt;sub&gt;3&lt;/sub&gt;, V&lt;sub&gt;4&lt;/sub&gt;, II</td>
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#### Best Lead Combinations

<table>
<thead>
<tr>
<th>One Channel Recording</th>
<th>V&lt;sub&gt;1&lt;/sub&gt; or V&lt;sub&gt;6&lt;/sub&gt;</th>
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<tbody>
<tr>
<td>Two Channel Recording</td>
<td>Arrhythmia: V&lt;sub&gt;1&lt;/sub&gt; and III</td>
</tr>
<tr>
<td></td>
<td>ST Segment: V&lt;sub&gt;3&lt;/sub&gt; and III</td>
</tr>
<tr>
<td></td>
<td>Arrhythmia + ST Segment: V&lt;sub&gt;1&lt;/sub&gt; or V&lt;sub&gt;6&lt;/sub&gt; + aVF or III</td>
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</table>

Table originally developed by Bridges and published in CCN (2008) Rauen et al. Updated for this outline by Rauen 1/13

### Treatment Options

**Identify & Treat the Underlying Cause**

**Defibrillation/Cardioversion**

The passage of electrical current through the cardiac muscle (cells) causes a massive depolarization allowing the cells to ‘reset’ themselves and hopefully creating an environment where the SA node can ‘take back’ the pacemaker function.

**Pacing**

For bradycardic rhythms, electrical stimulation of the heart might be necessary with a transcutaneous, transvenous or permanent pacemaker.

**Pharmacology**

Drugs are the primary treatment if the dysrhythmia is NOT life-threatening. Meaning there is not a significant drop in blood pressure or level of consciousness. The Antidysrhythmic agents are classified using the VaughanWilliams classification system.

<table>
<thead>
<tr>
<th>Drug Classification</th>
<th>Primary Action</th>
<th>Drug Options/Indications</th>
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<tbody>
<tr>
<td><strong>CLASS I</strong></td>
<td><strong>Membrane-Stabilizing</strong></td>
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<tr>
<td>Sodium Channel</td>
<td>- Slow/Block influx of Na into cell</td>
<td>IA</td>
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<tr>
<td>Blockers</td>
<td>- Effect Stimulus Automaticity, Conduction &amp; Excitability</td>
<td>Quinidine (Quinaglute)</td>
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<td></td>
<td></td>
<td>Procainamide (Procan, Proestyl)</td>
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<tr>
<td></td>
<td></td>
<td>Disopyramide (Norpace)</td>
</tr>
<tr>
<td>IA</td>
<td>- Slows Conduction Velocity</td>
<td>IA</td>
</tr>
<tr>
<td></td>
<td>- Negative Inotrope</td>
<td>Quinidine (Quinaglute)</td>
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<tr>
<td></td>
<td>- Prolong Refractory Period (wide QRS &amp; QT)</td>
<td>Procainamide (Procan, Proestyl)</td>
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<td></td>
<td>- Suppress Ectopic &amp; Reentry Foci</td>
<td>Disopyramide (Norpace)</td>
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<td></td>
<td></td>
<td>Atrial Flutter, Atrial Fib, SVT, VT</td>
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Specific Dysrhythmias

Tachycardias

The major problem with the tachy dysrhythmias is that the heart chambers do not have enough time to completely fill or empty. This leads to a drop in stroke volume and subsequently cardiac output. Depending on the exact rhythm, there may also be loss of synchrony between atrial and ventricular contractions (A-fib, V-Tach), which causes a loss of atrial kick and up to 30% of cardiac output. Another potential problem is clot formation in a chamber that has incomplete emptying. In clinical terms tachycardic rhythms can cause anything from dizziness to heart failure and cardiac arrest.
Narrow QRS Complex Tachycardias (supraventricular)

a. Rhythms
   - Sinus Tachycardia (ST)
   - Atrial Fibrillation (A-Fib)
   - Atrial Flutter (AF)
   - Atrial Tachycardia (ectopic and reentrant) (AT)
   - Multifocal Atrial Tachycardia (MAT)
   - Junctional Tachycardia (JT)
   - Accessory Pathway-Mediated
     o Atrial tachycardia w/ accessory pathway
     o AV reentry tachycardia

b. Treatment (remember to evaluate ventricular function) Based on 2010 ACLS guidelines
   - Stable?
     o A-Fib or AF: Identify length of time in rhythm and consider WPW and LV impairment before determining treatment. Control Rate, Convert Rhythm, Provide Anticoagulation
     o Vagal Stimulation
     o Adenosine
     o PSVT: β-Blockers, Ca++ Channel Blockers, Dig, Antiarrhythmics and Cardioversion. If EF < 40% Start with Cardioversion
     o JT: β-blockers, Ca++ Channel Blockers, Amiodarone, NO Cardioversion
     o MAT: β-blockers, Ca++ Channel Blockers, Amiodarone, NO Cardioversion
   - Unstable? Immediate Cardioversion, Followed by Drugs

Wide QRS Complex Tachycardias

a. Criteria for Wide QRS
   - Rate > 120 bpm
   - Uniform QRS > 120 ms
   - No S&S or Δ in Consciousness

b. Rhythms
   - Ventricular Tachycardia (VT)
   - Ventricular Fibrillation (VF)
   - SVT with Aberrancy (identify and treat as SVT)

c. Treatment (remember to evaluate ventricular function) Based on 2010 ACLS guidelines
   - Ventricular Tachycardia Stable w/ Pulse
     o Monomorphic: Procainamide, Sotalol, Amiodarone, Lidocaine. Amiodarone 1st if ventricle impaired
     o Polymorphic: normal QT - β-blockers, Lidocaine, Amiodarone, Procainamide, Sotalol, Amiodarone 1st if ventricle impaired. Long QT – Mg⁺, overdrive pacing, Isoproterenol, Phenytoin, Lidocaine
   - Ventricular Tachycardia Unstable w/ Pulse: Immediate Cardioversion
   - Ventricular Tachycardia Without Pulse – Treat as VF
- Ventricular Fibrillation/Pulseless VT:
  - Assess ABCs
  - Basic Life Support
  - Defibrillation: 120-200J biphasic or 360J monophasic (one shock)
  - CPR
  - Defibrillation: 120-200J biphasic or 360J monophasic (one shock)
  - Vasopressin or Epinephrine
  - Defibrillation: 200J biphasic or 360J monophasic
  - Antiarrhythmics: Amiodarone, Lidocaine, Magnesium, Procainamide
  - Defibrillation: 200J biphasic or 360J monophasic

**Long QT Syndrome:**
The QT represents the repolarization of the ventricle. Repolarization is an electrically unstable time. VT is a likely outcome if the next R wave were to fall on the T wave. In situations where the QT interval is long, there is an increased likelihood of an R on T to occur. Conditions that can lead to this situation include:
- Congenital Long QT Syndrome (genetic)
- Exercise Induced QT Syndrome
- Drug Induced QT Syndrome (many drugs lengthen QT)
  - Antiarrhythmic Agents: Class IA, IB & III
  - Tricyclic Antidepressants
  - Phenothiazine
  - Antimicrobials (specifically Erythromycin)
  - Nicardipine (Cardene)
  - Cisapride (Propulsid)
  - Haloperidol (Haldol)
  - Tamoxifen (Nolvadex)

**Bradycardias**
The major problem with slow rhythms is a lack of stroke volume to sustain an adequate cardiac output. Treatment is dependent on rhythm and cause of slow rate. In the unstable patient with a slow rate:
- ABCs & BLS
- Atropine
- Transcutaneous Pacing
- Dopamine or Epinephrine
- If the rhythm is Type II 2nd Degree or 3rd Degree HB and the pt is unstable pace ASAP (transcutaneous → transvenous)
Conduction Defects

Normal Parameters:
PR = 0.12-0.20
QRS = < 0.12
QT rate dependent

First Degree AV Heart Block
a. Rate: 60 - 100 bpm
b. Rhythm: Regular
c. P Waves: One P for Every QRS with PRI > 0.20
d. QRS Complexes: Normal
e. Symptoms/Concerns: Symptoms will depend on HR Concern for reason this is occurring and will it progress to higher level block
f. Tx: Depends on Symptoms, tx rarely required. Rhythm very common in elderly

Second Degree AV Heart Block (two types)
a. Mobitz Type I, also known as Wenckebach
   • Rate: atrial rate 60 - 100 bpm, ventricular rate varies
   • Rhythm: Irregular with Pattern
   • P Waves: All QRSs are preceded by Ps
     o But not all Ps are followed by QRSs
     o The PRI progressively gets longer
     o Until there is a dropped beat (a P wave not followed by a QRS)
     o Pattern then starts over
   • QRS Complexes: Normal
   • Symptoms/Concerns: Symptoms will depend on Ventricular HR
   • Tx: Depends on Symptoms, tx rarely required
b. Mobitz Type II, also known as Classical
   • Rate: atrial rate 60 - 100 bpm, ventricular varies
   • Rhythm: P-P regular, R-R regular or irregular
   • P Waves: All QRSs are preceded by Ps
     o But not all Ps are followed by QRSs
     o The PRI consistent and typically > 0.20
     o More than one P wave for every QRS
     o Typically a consistent pattern ex. 2Ps:1QRS
   • QRS Complexes: Normal
   • Symptoms/Concerns: Symptoms will depend on Ventricular HR
Dysrhythmias

- Tx: Depends on Symptoms, typically treated
  - Consider External Pacemaker
  - Consider Cause
  - Stop Digoxin
  - Atropine or Epinephrine

Third-degree AV Heart Block/Complete Heart Block aka AV Dissociation
a. Rate: < 60 bpm
b. Rhythm: P-P regular, R-R regular
c. P Waves: P waves “march” out regular but have no discernible relationship to the QRS
d. QRS Complexes: Slow, Wide > 0.12, “march” out, regular
e. Symptoms/Concerns: Symptoms will depend on Ventricular HR and LOC
f. Tx: Depends on Symptoms, tx typically required
   - External pacemaker
   - Atropine (not typically helpful because it will increase sinus node firing (P waves) but not ventricular conduction
   - Epinephrine

Bundle Branch Blocks
QRS Complex
a. Represents: Ventricular Depolarization
b. Shape:
   - Q First Negative Deflection
   - R First Positive Deflection
   - S Second Negative Deflection
c. Duration (time):
   - QRS > 0.12sec (3mm)
   - Q < 0.03sec (< 1mm)

When there is an electrical block in the normal conduction pathway for ventricular depolarization it is a called a bundle branch block. This block can be permanent or intermittent and has a variety of causes. Depolarization occurs because of the principle of conductivity. This depolarization takes longer (QRS duration > 0.12sec) and the configuration is slightly different than the normal QRS pattern. Bundle Branch Block patterns are best evaluated in precordial leads V₁ and V₆.
Dysrhythmias

Right Bundle Branch Block

V1 rsR' > 0.12sec
V6 qRs > 0.12sec

Left Bundle Branch Block

V1 rS > 0.12sec
V6 R > 0.12sec