Supraventricular Arrhythmias

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Trauma, Acute Care Surgery, & Surgical Critical Care Fellow
SCC Lecture Series
January 2015
Objectives:

- Review anatomy and physiology of cardiac conduction system.
- Define supraventricular arrhythmia.
- Describe the initial approach to a patient with an arrhythmia.
- Discuss diagnosis and treatment options for patients with atrial fibrillation, atrial flutter, and supraventricular tachycardia.
Cardiac arrhythmias

- Caused by a derangement in electrical impulse initiation, conduction, or both
- Classified as:
  - Brady (<60 bpm) or tachy (>100 bpm)
- Tachyarrhythmias categorized by location of origin of irregular impulse:
  - Above the AV node (supraventricular)
  - Below the AV node (ventricular)
Cardiac Conduction System.

- SA node = pacemaker
  - Generates cardiac impulse, automaticity
  - Jxn SVC and RA
  - RCA in 60%
Cardiac Conduction System.

- SA node -> atria
- Atrioventricular node
- AV node controls atrial impulse tx to ventricles, thus regulating speed of atrial and ventricular contractions
Cardiac Conduction System.

- AV node to intraventricular septum via bundle of His
- R, L BB
- Purkinje fibers
  - Activate ventricles
Cardiac Conduction System.

- Heavy innervation from sympathetic and parasympathetic nervous system determines heart rate and speed of contraction.

**Sympathetic:** Epinephrine, NE act on adrenergic receptors. Faster conduction, Increased impulse generation.

**Parasympathetic:** Vagus nerve releases acetylcholine, which acts on muscarinic receptors. Slows sinus node impulse generation and conduction thru AV node.
Factors promoting arrhythmias in surgical pts:

**Iatrogenic Factors:**
- volume overload
- direct manipulation of the heart
- intravascular catheters
- drugs
- cardiopulmonary bypass

**Patient Factors:**
- Underlying structural abnormalities
- CHF
- CAD

**Other:**
- Electrolyte imbalances
- Excess sympathetic tone
What is supraventricular arrhythmia?

- Abnormal impulse arises above bundle of His
- Require atrial or AV nodal tissue to initiate & maintain
Supraventricular arrhythmias

- Atrial tachycardia
- Atrial fibrillation
- Atrial flutter
- Supraventricular tachycardia
  - AV nodal re-entrant (60%)
  - Atrioventricular reentry, accessory pathway (30%)
Initial evaluation

- Vital Signs: stable vs. unstable
- History & Physical Exam
- EKG
- Other diagnostic modalities
Stable vs. Unstable

- The urgency of therapy depends on hemodynamic stability.
- If unstable... address ABC’s first
  - and brady: call for external pacers
  - and wide QRS: call for defibrillator
- All patients:
  - Consider underlying ischemia or heart failure
  - Telemetry and pulse oximetry
Stable vs. Unstable

- The hemodynamic impact of an arrhythmia depends on:
  - the ventricular response
  - preservation of cardiac output
  - degree of underlying structural or ischemic disease
History

- Family or personal history of arrhythmia, ischemic disease, valvular disease
- Assess recent medications
- ROS:
  - Chest pain, SOB, palpitations, presyncope, syncope
  - Above may occur w/ any arrhythmia
Physical Exam

- Airway
- Oxygenation
- Pulses, IVs
- Mentation
- Regular or irregular? Murmur?
- Crackles?
- JVD?
Look at the monitor.

- Supraventricular arrhythmia:
  - Rapid, narrow QRS complex (<120 msec) with P waves

- IF SVA: attempt vagal maneuvers
  - Breath-holding or Valsalva
  - Carotid massage
Diagnosis

- Progress from simple to invasive testing.
- EKG is the first line in diagnosis.
- Early in evaluation, address underlying abnormalities which may be triggers:
  - Ischemia (EKG)
  - Hypercarbia (ABG)
  - Proarrhythmic drugs
  - Electrolytes (BMP)
  - Malpositioned catheter (CXR)
Electrocardiogram

Conduction velocity Through AV Node
EKG and Supraventricular Arrhythmia

- Assess QRS: wide vs. narrow
  - Wide: ventricular arrhythmia, but also SVA w/ bundle block or accessory pathway
- Look for presence of P waves
  - No p waves- suspect A fib.
- Rate
  - 300 bpm suggests atrial flutter
- More P waves than QRS: AV block
  - SA node firing but signal not conducting
1. Atrial Fibrillation

2. Supraventricular Tachycardia

3. Atrial Flutter
Other diagnostic modalities

- ECHO
  - Evaluates for functional and structural abnormalities
- Electrolytes
- Thyroid function tests
- EP Studies:

Table 2. Conditions Warranting Referral to an Electrophysiologist.

- Tachycardia with a wide QRS complex
- Supraventricular tachycardia
  - In a patient with syncope or severe symptoms
  - In a patient with drug resistance or intolerance
  - In a patient who prefers to be free of drug therapy
- Preexcitation syndrome (with or without supraventricular tachycardia)
Supraventricular arrhythmias

- Atrial tachycardia
- Atrial fibrillation
- Atrial flutter
- SVT
  - AV nodal re-entrant (60%)
  - Atrioventricular reentry, accessory pathway (30%)
SVA: Atrial Fibrillation

- MC post-op arrhythmia
- Impulse above bundle of His -> disorganized atrial activity, dyssynchrony of contraction between atrium and ventricle
- Loss of atrial kick and reduced CO
- No reserve = unstable
- Stasis leads to thromboembolic events
Afib: Risk Factors

**Patient Factors**
- Age >60 years old**
- Male gender
- CHF
- Valvular disease

**Surgeries with high risk AF**
- Esophagectomy
- Pulmonary resection
- Intra-abdominal surgery
- Vascular surgery
Risk factors associated with atrial fibrillation after noncardiac thoracic surgery: Analysis of 2588 patients

Ara A. Vaporgiyan, MD
Arlene M. Correa, PhD
David C. Rice, MD
Jack A. Roth, MD
W. R. Smythe, MD

- Prospective database
- 2588 pts undergoing major non-cardiac thoracic surgery at a single institution, 1998 to 2002
- What are the risk factors associated with atrial fibrillation after noncardiac thoracic surgery?

Results:

- Rate of fib = 12.3%
- Development of fib significantly increased mortality rates (from 2.0% to 7.5%), length of stay, and cost of stay.

TABLE 4. Results of multivariate analysis to identify variables associated with AF after major thoracic surgery

<table>
<thead>
<tr>
<th>Variable</th>
<th>Relative risk</th>
<th>Confidence interval</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y (reference: age &lt;50 y)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>50-59</td>
<td>1.70</td>
<td>1.01-2.88</td>
<td>.05</td>
</tr>
<tr>
<td>60-69</td>
<td>4.49</td>
<td>2.79-7.22</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>≥70</td>
<td>5.30</td>
<td>3.28-8.59</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>Sex</td>
<td>1.72</td>
<td>1.29-2.28</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>Congestive heart failure</td>
<td>2.51</td>
<td>1.01-6.24</td>
<td>.05</td>
</tr>
<tr>
<td>Arrhythmias</td>
<td>1.92</td>
<td>1.22-3.02</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>Peripheral vascular disease</td>
<td>1.65</td>
<td>0.93-2.92</td>
<td>.09</td>
</tr>
<tr>
<td>Procedure performed (reference: single wedge)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lobectomy</td>
<td>3.89</td>
<td>2.19-6.91</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>Multiple wedges</td>
<td>0.87</td>
<td>0.41-1.85</td>
<td>.72</td>
</tr>
<tr>
<td>Esophagectomy</td>
<td>2.95</td>
<td>1.55-5.62</td>
<td>.01</td>
</tr>
<tr>
<td>Chest wall or sternum</td>
<td>0.92</td>
<td>0.34-2.47</td>
<td>.86</td>
</tr>
<tr>
<td>Pneumonectomy</td>
<td>8.91</td>
<td>4.59-17.28</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>Mediastinal tumor resection or thymectomy</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Segmentectomy</td>
<td>1.57</td>
<td>0.57-4.29</td>
<td>.38</td>
</tr>
<tr>
<td>Bilobectomy</td>
<td>7.16</td>
<td>3.02-16.96</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>Any intraoperative transfusion</td>
<td>1.39</td>
<td>0.98-1.98</td>
<td>.07</td>
</tr>
</tbody>
</table>

Incidence and risk factors of atrial fibrillation in a surgical intensive care unit

Philippe Seguin, MD; Thomas Signouret, MD; Bruno Laviolle, MD; Bernard Branger, MD; Yannick Mallédant, MD

- Prospective
- Observational
- n = 460 pts
- AF in 5.3%

Table 5. Multivariate predictors of atrial fibrillation

<table>
<thead>
<tr>
<th>Predictor</th>
<th>Odds Ratio (95% Confidence Interval)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>1.04 (1.01–1.07)</td>
<td>.009</td>
</tr>
<tr>
<td>Blunt thoracic trauma</td>
<td>16.84 (4.00–71.20)</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td>Shock</td>
<td>6.77 (2.17–21.12)</td>
<td>.01</td>
</tr>
<tr>
<td>Pulmonary artery catheter</td>
<td>5.46 (1.84–16.21)</td>
<td>.002</td>
</tr>
<tr>
<td>Previous treatment by calcium-channel blockers</td>
<td>3.87 (1.18–12.74)</td>
<td>.026</td>
</tr>
</tbody>
</table>

Table 6. Severity, length of stay, and mortality in patients with atrial fibrillation vs. control patients

<table>
<thead>
<tr>
<th></th>
<th>No Atrial Fibrillation</th>
<th>Atrial Fibrillation</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n = 429</td>
<td>n = 24</td>
<td></td>
</tr>
<tr>
<td>SAPS II</td>
<td>31 ± 17</td>
<td>45 ± 20</td>
<td>.0001</td>
</tr>
<tr>
<td>OMEGA</td>
<td>90 ± 140</td>
<td>262 ± 260</td>
<td>.0001</td>
</tr>
<tr>
<td>ICU length of stay, days</td>
<td>7 ± 9</td>
<td>16 ± 14</td>
<td>.0001</td>
</tr>
<tr>
<td>Hospital length of stay, days</td>
<td>22 ± 21</td>
<td>34 ± 30</td>
<td>.009</td>
</tr>
<tr>
<td>Mortality, %</td>
<td>17.5</td>
<td>37.5</td>
<td>.025</td>
</tr>
</tbody>
</table>

Values are given as mean ± sd unless otherwise noted.
Atrial Fibrillation: Treatment

- **Who to treat?**
  - Pts with heart failure
  - Afib >48h
  - Uncontrolled ventricular rates
  - Prior history of stroke

- **How to treat?**
  - Rate control
    - Slows HR and allows ventricular filling
  - Rhythm control
    - Resynchronizes to NSR
  - Anticoagulation?
Afib: Treatment

- Rate control
  - Slows ventricular response, allows ventricular and coronary filling, increased CO
  - Treatment options:
    - Beta-blockers
    - Calcium Channel Blockers
    - Amiodarone
    - Digoxin
Afib: Rate control

- **Beta-blockers**
  - 1st line for rate control
  - Direct anti-arrhythmic activity on conduction cells
  - Counteract hyperadrenergic post-op state
  - Shown to accelerate conversion to sinus rhythm vs. CCBs
  - Agents:
    - Esmolol, Metoprolol
  - Contraindications: Hypotension, bradycardia, heart block, decompensated heart failure, asthma
Afib: Rate control

- **Calcium Channel Blockers (CCBs)**
  - 2nd-line therapy for rate control, or 1st line for those intolerant of B-Bl
  - Block the calcium channel in AV node, leads to slower impulse conduction
  - Agents:
    - Verapamil, diltiazem
  - May result in hypotension
**Afib: Rate control**

**Amiodarone**
- Good choice for heart failure, HD instability
- Monitor for ADRs:
  - Sinus brady
  - AV block
  - Respiratory dysfunction
  - Hypotension

**Digoxin**
- Increases parasympathetic stimulation to heart
- Good choice in heart failure
Afib: Rhythm control

- Re-synchronizes atrium with ventricle
- Pharmacologic or electrical (DC)
Afib: Rhythm control

- Pharmacological:
  - Single dose flecanide or propafenone
    - Risk of VT, sinus brady; contraindicated in CAD
    - Prolongs QT
  - Ibutilide
    - Use in unstable hemodynamics, adr: nausea
    - Prolongs QT, don’t use in hypokalemia
  - Amiodarone
    - Good choice in heart failure, structural heart dz
  - ADR: thyroid, optic, pulm toxicity
Afib: Rhythm control

- Electrical: Direct Current cardioversion
  - Use for:
    - ongoing stable Afib >48h
    - refractory Afib, unstable/ischemic
  - Don’t use: asymptomatic arrhythmia
  - 120-200 joule biphasic or 200 joule monophasic shock in synchrony w QRS complex
  - Exclude intracardiac thrombus w/ TEE
- Maintain w amiodarone, sotalol
Prospective study

Primary success rate of DC cardioversion in postop ICU pts w new-onset supraventricular tachyarrhythmias

N= 37 pts

NSR restored in 35% after 1 shock, with 100% converted after 4 shocks

At 48 hours, only 13.5% remained in sinus rhythm

?different pathophysiologic mechanisms in surgical pts, making them less responsive to DC cardioversion

Crit Care Med 2003; 31:401-405
Multicenter RCT
4060 pts w Afib: rate control vs. rhythm control
Primary endpoint: mortality
Inclusion
- age>65
- At least 1 risk factor for stroke or death (LA enlargement, HTN, DM, CHF, prior TIA, LV dysfunction)
Results:

- rhythm control offers no mortality benefit over rate control
- Potential benefits in rate control in less toxic drugs
Treatment of new-onset atrial fibrillation in noncardiac intensive care unit patients: A systematic review of randomized controlled trials*

Salmaan Kanji, PharmD; Robert Stewart, MD; Dean A. Fergusson, MHA, PhD; Lauralyn McIntyre, MD, MSc, FRCPC; Alexis F. Turgeon, MD, MSc, FRCPC; Paul C. Hébert, MD, MSc, FRCPC

- Systematic review, 1966-2006
- 4 trials, 143 pts w/ supraventricular arrhythmia

<table>
<thead>
<tr>
<th>Trial Design</th>
<th>Blinding</th>
<th>Patients</th>
<th>N</th>
<th>Intervention</th>
<th>Patients with AF</th>
<th>Definition of Cardioversion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chapman (1993)</td>
<td>No</td>
<td>Med/surg</td>
<td>24</td>
<td>Amiodarone (3 mg/kg bolus then 10 mg/kg over 24 hrs) vs. procainamide (10 mg/kg bolus then 4 mg/min × 2 hrs, then 3 mg/kg × 2 hrs then 2 mg/kg × 20 hrs)</td>
<td>16/24</td>
<td>Within 12 hrs</td>
</tr>
<tr>
<td>Moran (1995)</td>
<td>No</td>
<td>Med/surg</td>
<td>42</td>
<td>Amiodarone (5 mg/kg bolus then 10 mg/kg over 24 hrs) vs. magnesium (37 mg/kg bolus, then 25 mg/kg/hr × 24 hrs)</td>
<td>18/34</td>
<td>Within 24 hrs</td>
</tr>
<tr>
<td>Borrando (1994)</td>
<td>No</td>
<td>Med</td>
<td>30</td>
<td>Flecaïnide (2 mg/kg bolus then 1.5 mg/kg over 1 hr) vs. verapamil (0.15 mg/kg bolus then 0.005 mg/kg/min × 1 hr)</td>
<td>11/30</td>
<td>Within 1 hr</td>
</tr>
<tr>
<td>Balzer (1998)</td>
<td>No</td>
<td>Surg</td>
<td>55</td>
<td>Esmolol (12.5–50 mg repeated bolus until HR &lt;110 beats/min then 50–100 µg/min vs. diltiazem (20 mg bolus then 10–20 mg/hr)</td>
<td>44/55</td>
<td>Within 12 hrs</td>
</tr>
</tbody>
</table>

Total 143

HR, heart rate; AF, atrial fibrillation.
“Using published literature, we cannot recommend a standard treatment for atrial fibrillation in non-cardiac ICU patients”
Atrial Fibrillation: Anticoagulation

- When to anticoagulate?
Nonvalvular: CHA2DS2-VaSc
- Anticoag for CHADS2 2 or higher, prior stroke
  - Bridge depending on risk:benefit
Valvular: warfarin, bridge prn
<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Congestive heart failure/LV dysfunction</td>
<td>1</td>
</tr>
<tr>
<td>Hypertension</td>
<td>1</td>
</tr>
<tr>
<td>Age ≥ 75 y</td>
<td>2</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>1</td>
</tr>
<tr>
<td>Stroke/TIA/TE</td>
<td>2</td>
</tr>
<tr>
<td>Vascular disease (prior myocardial infarction, peripheral artery disease, or aortic plaque)</td>
<td>1</td>
</tr>
<tr>
<td>Age 65-74 y</td>
<td>1</td>
</tr>
<tr>
<td>Sex category (ie female gender)</td>
<td>1</td>
</tr>
</tbody>
</table>

LV = left ventricular; TE = thromboembolism. See Table 1 for expansion of other abbreviations.
SVA: Atrial Flutter (AF)

- Reentrant arrhythmia
  - Alternate circuit rotates around tricuspid valve annulus
  - Saw-tooth pattern of P waves
  - Usual rate 240-320 bpm
  - A rate of 150 bpm could be AF with 2:1 AV block
SVA: Atrial Flutter

- Rate control
  - Diltiazem, verapamil, beta-blockade
  - Digoxin for CHF

- Rhythm control
  - Ibutilide, dofetilide, sotalol to terminate rhythm
  - May prolong QT and lead to torsades
  - DC cardioversion: 50-100 joule biphasic shock
  - If recurrent: EP for ablation
Supraventricular Tachycardia

- Narrow complex <120 msec
  - P wave may be buried in QRS
  - May see wide QRS if present with BBB or accessory pathway
- Sudden onset and termination
- Common subtypes include:
  - AV nodal reentrant
  - AV reciprocating
  - Focal atrial tachycardia
SVT: treatment

- Vagal maneuvers
  - Carotid massage, Valsalva
  - Stimulate baroreceptors, increase vagal activity, slows impulse conduction through AV node
SVT: Treatment

- Pharmacotherapy
  - Adenosine
    - AV nodal blocking agent, t1/2 = 10 seconds
    - 1st line tx SVT
    - Diagnostic and tx for wide-complex SVT
    - RCTs: 60-80% termination with 6mg adenosine, 90-95% after 12mg.
      - Use under cardiac monitoring with defibrillation pads in place (asystole or VF may result)
  - Don’t use in heart transplant
SVT: treatment

- Pharmacotherapy
  - Verapamil or diltiazem
    - Use for recurrent SVT after adenosine
    - May cause vasodilation, bradycardia, heart block
  - Esmolol
    - Short half-life
    - Preferred for use in pts at risk for B-bl complications
Multicenter, retrospective, observational study

197 pts w wide-complex tachycardias

Response to adenosine: 90% with SVT and 2% with v-tach

No adverse events in either groups

Response to adenosine increased odds of SVT by 36x, and nonresponse increased odds of ventricular tachycardia by 9x

Adenosine is safe in wide-complex tachycardia as both diagnostic and therapeutic measure

SVT: treatment

- If SVT still refractory to above therapies:
  - Antiarrhythmics (watch for torsades)
    - Procainamide
    - Ibutilide

- For unstable SVT:
  - R-wave synchronous DC cardioversion with 100-200 joules
Objectives:

- Review anatomy and physiology of cardiac conduction system.
- Define supraventricular arrhythmia.
- Describe the initial approach to a patient with an arrhythmia.
- Discuss diagnosis and treatment options for patients with atrial fibrillation, atrial flutter, and supraventricular tachycardia.
SUMMARY

- Supraventricular arrhythmia: any arrhythmia initiated above the atrioventricular node
- Afib, a flutter, SVT
- Stable vs. Unstable... if unstable, ABCs, consider cardioversion
- If stable, obtain a 12 lead ECG...
  - A Fib, A Flutter: rate vs. rhythm control
  - SVT: vagal maneuvers, adenosine
- Remember risk factors and precipitating conditions in surgical pts
- Long term therapy depends on mechanism and can be conservative, pharmacologic or invasive
Bibliography

Bibliography
