

Arrhythmia Diagnosis and Management

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Overview of Arrhythmias

- Diagnostic testing
- Review of bradycardias
- Review of tachycardias
- Antiarrhythmic agents
- Cardiac devices
- Questions!

Diagnostic testing

- Resting EKG
- Continuous ambulatory EKG (24-48hrs)
- Event recorders: triggered by patient
 - Looping recorder
 - Postsymptom recorder
- Implantable loop recorder (up to 3 years battery life)

- Others to consider:
 - TTE to evaluate for structure heart disease
 - Ischemia evaluation if life-threatening arrhythmia
 - EP testing to provoke arrhythmia and potentially ablate

Overview of Bradycardia

- 2/2 disorders of impulse **generation** (impaired automaticity) or impulse **conduction** (heart block)
- Reversible causes include Lyme disease, drugs, hyperkalemia and thyroid disease
- Symptoms: **syncope**, fatigue, dyspnea, exercise-intolerance, ventricular arrhythmias
- Evaluation includes documentation of bradycardia with symptoms
- Management includes atropine/transcutaneous pacing if unstable, treatment of reversible causes, PPM

Impaired automaticity

- **Sinus brady not necessarily pathologic (i.e. athletes)**
- **Causes of pathologic sinus bradycardia**
 - Sick sinus syndrome/SA node dysfunction
 - Infarction or cardiac surgery
 - Infiltrative processes (amyloidosis, sarcoidosis)
 - Increased vagal tone (Valsalva, vomiting)
 - Medications (ie BB, CCB)
 - Genetic diseases
- **Pacemaker** is indicated for symptomatic sinus bradycardia

Heart Block

- 1st degree and 2nd degree Mobitz 1 block usually 2/2 disease within the *AV node*
- 2nd degree Mobitz 2 and 3rd degree block usually *His bundle or below*
- 1st degree block associated with increased risk afib, pacemaker implantation and all-cause mortality
- “Advanced” or “high-grade” 2nd degree heart block if 2+ nonconducted P’s before each QRS
- Pacemaker indicated for advanced 2nd degree or 3rd degree AV block (CHB)
- Reversible causes of CHB include medications and Lyme disease

Overview of tachycardia

- **Narrow complex tachycardias**

- Atrial fibrillation/atrial flutter
- Supraventricular tachycardias

- **Wide complex tachycardias**

- SVT with aberrancy
- Ventricular arrhythmias

Acute management of afib

- Immediate synchronized cardioversion if **hypotension, angina or heart failure**
- If HD stable, goal HR 60-110/min with BB, CCB or digoxin
- **Consider elective cardioversion:**
 - If duration afib < 48hrs, can proceed with DCCV
 - If duration > 48hrs or unknown: anticoagulate for 3+ weeks prior to DCCV, or anticoagulate then TEE to rule out intracardiac thrombus prior to DCCV
 - Anticoagulation must continue after cardioversion

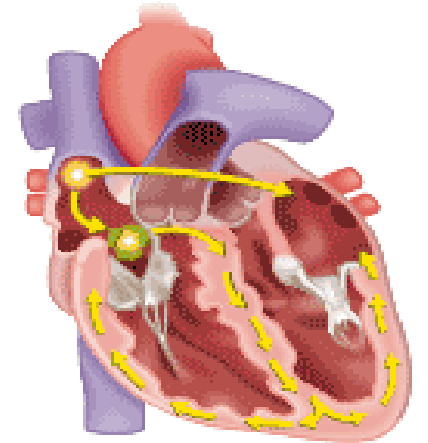
Long-term management of afib

- Increased **risk of embolic stroke**, heart failure and all-cause mortality
- Anticoagulation based on risk factors (ie CHADS2, rheumatic MS, or mechanical heart valve)
 - **Warfarin** > aspirin + plavix > aspirin alone
 - Direct thrombin inhibitors (ie apixaban)
 - Rivaroxaban (oral factor Xa inhibitor)
- **Rate control**
- **Rhythm control if symptoms despite adequate rate control**
 - Consider “pill-in-the-pocket” approach with flecainide or propafenone for symptomatic paroxysmal afib (remember to rate control as well, i.e., diltiazem!)
- Ablation, esp if aflutter
- AV nodal ablation
- Maze surgery

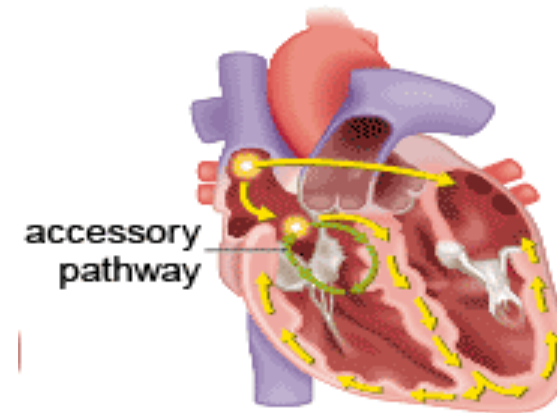
Supraventricular tachycardias

- **AVNRT** – fast and slow pathways *within* AV node
- **AVRT** – circuit includes AV node and bypass tract
 - Anterograde conduction leads to preexcitation: short PR interval, delta wave (WPW)
 - Retrograde conduction -> concealed bypass tract
- **Atrial tachycardia** – ectopic focus or area of micro-reentry
- **Management includes AV nodal blocking agents, antiarrhythmics or catheter ablation**

heart with AVNRT



heart with AVRT



Ventricular Arrhythmias

- ***Premature ventricular contractions***
 - Present in up to 75% of healthy persons
 - More common in persons with HTN or structural heart disease
 - Treat symptoms with B-blocker or CCB
 - Antiarrhythmics or ablation if refractory symptoms
- ***Ventricular tachycardia***
 - Increased risk of SCD in patients with structural heart disease
 - Idiopathic VT carries better prognosis
 - B-blockers for patients with HF or ischemic heart disease
 - ICD for primary or secondary prevention
 - Antiarrhythmics may reduce frequency of shocks but not mortality
- ***Inherited arrhythmia syndromes***
 - Long QT syndrome -> torsades
 - Short QT syndrome -> afib, VT/VF, SCD
 - Brugada syndrome -> VF, SCD

Antiarrhythmic Medications

Vaughan-Williams Classification	Mechanism of Action	Examples	Effect	Use
Class IA	Na-channel blockade, some K-channel blockade	Quinidine, procainamide, disopyramide	Slows conduction, prolongs repolarization	Preexcited afib, SVT, ventricular arrhythmias
Class IB	Na-channel blockade	Lidocaine, mexiletine, phenytoin	Slows conduction in diseased tissues, shortens repolarization	Ventricular arrhythmias
Class IC	Na-channel blockade	Flecainide, propafenone	Markedly slows conduction, slightly prolongs repol	Afib, aflutter, SVT, ventricular arrhythmias
Class II	Beta blockade	Metoprolol, propranolol,	Suppresses automaticity and slows AV nodal conduction	Rate control of arrhythmias, SVT, ventricular arrhythmias
Class III	Potassium channel blockade	Sotalol, amio, dofetilide, dronedarone	Prolongs action potential duration	Afib, aflutter, ventricular arrhythmias
Class IV	Calcium channel blockade	Verapamil, diltiazem	Slows SA node automaticity and AV nodal conduction	SVT, rate control of atrial arrhythmias, triggered arrhythmias
	A1 receptor agonist	Adenosine	Slows or blocks SA node automaticity and AV node conduction	Termination of SVT
	Increasing vagal activity	Digoxin	Slows AV nodal conduction	Rate control of arrhythmias



Indications for PPM

- Symptomatic bradycardia (HR<40/min) or sinus pauses
- Symptomatic CHB or 2nd degree heart block (type 1 or 2)
- Asymptomatic CHB or advanced 2nd degree HB
- Afib with pauses \geq 5 seconds
- Alternating bundle branch block
- After catheter AV nodal ablation

Overview of ICD

- Indicated for primary and secondary prevention
- Management perioperatively: shock function should be turned off during procedures using electrocautery
- Management of device infection: removal of entire system

Question:

56yo M is evaluated in the hospital for paroxysmal afib. The patient develops increasing SOB during these episodes. 5 days ago, he was admitted for an acute MI and cardiogenic shock and received a DES in the LAD. Medications are lisinopril, digoxin, furosemide, aspirin, clopidogrel, eplerenone, simvastatin, and unfractionated heparin.

On exam, the patient is afebrile, blood 92/65, pulse 75. O2 sat is 95% with 3L NC. Cardiac exam reveals estimated CVP of 12cm H2O. Heart sounds are distant and regular. There is a grade 2/6 holosystolic murmur at the cardiac apex. A summation gallop is present. Crackles are auscultated bilaterally in the lower lung fields.

Transthoracic echocardiogram shows LVEF 32%.

Which of the following is the most appropriate treatment for this pt's afib?

- (A) Amiodarone
- (B) Disopyramide
- (C) Dronedarone
- (D) Flecainide
- (E) Sotalol

Answer: amiodarone

Amiodarone is the best option for managing symptomatic afib in the setting of heart failure. Patients with heart failure and MI are at increased risk for development of afib. Although amio has many extracardiac side effects, it is the most effective agent for preventing afib recurrences, and it is one of the few agents **proved safe in patients with heart failure, LVH, CAD, or previous MI.** In addition, amio has B-blocking properties that can help with rate control.

Disopyramide has negative inotropic effects, which can be detrimental to someone with reduced LVEF and heart failure.

Dronedarone increases mortality in pts with NYHA class IV or class II-III HF with recent decompensation.

Flecainide is contraindicated after MI because of increased risk polymorphic VT.

Like amio, sotalol is class III antiarrhythmic but because of more potent B-blocking effects, it should not be used in acute heart failure.