ELECTROCARDIOGRAPHY: A USEFUL TOOL AT THE BEDSIDE

For the Family Medicine Refresher Course
March 6, 2019
The content and many of the images are taken from ACCSAP 2019 of the American College of Cardiology
Disorders of Impulse Generation and Propagation
Sinus Node Dysfunction

- Sinus arrest
- Sinus bradycardia
- Sinoatrial exit block
- Chronotropic incompetence
Sinus arrest (sinus pause)

A short sinus arrest terminated by a junctional escape beat and restoration of NSR
Post-conversion arrest (post-conversion pause)

Paroxysmal atrial fibrillation with a 3.8 second post-conversion pause
Defines the brady-tachy syndrome
The normal corrected sinus node recovery time in the EP lab is ≤ 550 m sec (a pause of 0.55 sec)
Normal sinus node impulse generation with intermittent exit block from the perinodal tissue resulting in intermittent dropped p-waves on the surface ECG.

Type 1 block-There is an incremental increase in the time between the sinus node depolarization and the p-wave on ECG. Can be shown in EP lab with intracardiac electrical recording or inferred on the surface ECG by a progressive shortening of the p-p interval before the dropped beat.

Type 2 block-The sudden failure of the sinus node impulse to conduct a p-wave.

Complete sinoatrial block is asystole.
Disorders of AV Conduction
AV Nodal Block

- First Degree AV Block
- 2:1 AV nodal block
- Mobitz Type 1 Second Degree AV block
- Mobitz Type 2 Second degree AV Block
- High-Grade Second Degree AV Block
- Third Degree AV Block (Complete Heart Block)
First Degree AV Block

PR interval is greater than 200 msec
Failure of every other to be conducted. While it is a second degree AV block, you can’t distinguish Mobitz 1 from Mobitz 2. Since the QRSs are narrow, this is likely to be AV block at the AV nodal level.
Mobitz Type 1 Second Degree AV Block

Progressive prolongation of PR interval until the QRS is blocked
The RR interval shortens until the QRS is blocked
Mobitz Type 2 Second Degree Block

Sudden failure of the P-wave to be conducted
Complete Heart Block

Complete AV Dissociation with a Junctional Escape Rhythm
Advanced Second Degree AV Block

The sudden failure of two or more consecutive beats to conduct.
Supraventricular Tachycardia
Supraventricular Tachycardia

- AV Nodal Re-entrant Tachycardia
- Atrio-Ventricular Re-entrant Tachycardia (Pathway-Mediated tachycardia)
  - Orthodromic
  - Antidromic
- Atrial Tachycardia

- SVT is usually a narrow-complex tachycardias
- They may be wide-complex tachycardias, however
  - Bundle Branch aberrancy
  - Pre-excitation
Mechanism of AVNRT: Micro-reentry

A-Dual pathways. Fast pathway has faster conduction velocity, but longer ERP
B-Sinus beat
C-PAC
D-AV nodal reentry circus movement tachycardia (AVNRT)
AVNRT

Note the pseudo s-waves in V2
AV Reentrant Tachycardia

1. They are pathway dependent

2. Require an accessory pathway

3. Orthodromic (A) down-going limb is through AVN-His-Purkinje system and retrograde along accessory pathway narrow complex

4. Antidromic (B) Down the accessory pathway and retrograde through AVN-His-Purkinje system. Wide complex caused by Delta wave
AVRT

Note the pseudo s-waves with longer RP interval than AVNRT
Pre-excited Atrial Fibrillation with Ventricular Rate ≈ 300 bpm
Wolf-Parkinson-White Pattern and Syndrome

Short PR interval caused by the delta wave which is a fusion of pre-excited and normally conducted wavefront. The accessory pathway is infero-septal.
Atrial Tachycardia

Initiation of Atrial Tachycardia

Common Sites for Focal Atrial Tachycardia in the Right Atrium
Atrial Tachycardia

Note the effect of adenosine on the arrhythmia.
Ventricular Arrhythmias in Patients with Structurally Abnormal Hearts
Ventricular Arrhythmias

- PVC’s
- Non-sustained VT
- Sustained VT
Premature Ventricular Contractions

- If they are prevalent to the degree of ≥ 15% of the QRS complexes in a 24 hour period (e.g. Holter monitor) they may cause a dilated cardiomyopathy

- One can assess their locus of origin by their morphology in multiple leads

- Exercise-induced PVC’s, particularly those with RBBB morphology in V1 (LV origin) are associated with future sudden cardiac death than those with LBBB morphology (RV origin)
Nonsustained Ventricular Tachycardia

Definition: 3 or more ectopic beats in a row at a rate of > 100 bpm and lasting no more than 30 sec.

A-Monomorphic VT

B-Polymorphic VT initiated by a PVC (R on T phenomenon). Common during ischemia

C-Polymorphic VT in patient with long QTc. Usually caused by meds or inherited channelopathy
Sustained Monomorphic VT

**Twelve-Lead Electrocardiograms of Two Different Sustained Monomorphic Ventricular Tachycardias**

**Electrocardiogram Markers of Ventricular Tachycardia**

<table>
<thead>
<tr>
<th>Criteria</th>
<th>Sensitivity</th>
<th>Specificity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atrioventricular dissociation</td>
<td>10%</td>
<td>100%</td>
</tr>
<tr>
<td>AVR: Initial R</td>
<td>40%</td>
<td>98%</td>
</tr>
<tr>
<td>AVR: Initial q or r &gt;40 ms</td>
<td>30%</td>
<td>92%</td>
</tr>
<tr>
<td>AVR: Notched q or s wave</td>
<td>20%</td>
<td>95%</td>
</tr>
<tr>
<td>Absence of RS in all precordial leads V1-V6</td>
<td>22%</td>
<td>95%</td>
</tr>
</tbody>
</table>
Ventricular Tachycardia in Patients With Normal Hearts
Frequent Sites of Idiopathic Ventricular Tachycardia

- Outflow Tract VT
  - LV outflow tract
  - RV outflow tract
- Malignant form of Idiopathic VT
- Left ventricular tachycardia
  - Originates most commonly in the left posterior fascicile of LV conduction system and less commonly in the left anterior fascicile)
RVOT Tachycardia

*Most common idiopathic VT
*Mean age of occurrence  50 ± 15 years
*SCD is rare. It does cause palpitations and dizziness
*May be associated with frequent enough PVC’s to cause tachycardia-mediated cardiomyopathy
*Features:
  - The V1 pattern is that of “Left Bundle” VT
  - The QRS is upright in the inferior leads
  - Late QRS transition V3-V4
*Caused by triggered automaticity
*Amenable to ablation
Indications for Treatment of Idiopathic VT

- Symptom control (most common)
- Sustained VT
- PVC-related cardiomyopathy
- Malignant form of idiopathic VT (rare)
RVOT Tachycardia Must be Distinguished from Arrhythmogenic RV Cardiomyopathy/Dysplasia

- Clinical Features
  - Palpitations, dizziness, syncope and SCD
  - Eventual RV failure
  - Family history of SCD, autosomal dominant
  - Usually diagnosed between 18 and 50 years of age
- Pathology: fibrofatty replacement of the RV free wall
- ECG Features
  - Wide QRS Complex and T-wave inversion V1-V3
  - Epsilon waves in V1
  - Delayed S-wave upstroke (55msec) in V1-V3
LVOT Tachycardia

*May originate in sinuses of Valsalva or aortic cusps
*Features
  “Left Bundle” pattern in V1
  Positive QRS complexes in the inferior leads
  Early QRS transition (V1-V2)
PVC-Associated Cardiomyopathy

- Requires over 15% of beats are PVC’s or >20,000 PVC’s in 24 hours
- Beta blockers, amiodarone, and sotalol may decrease PVC frequency and reduce symptoms, however, to avoid dilated cardiomyopathy they must be eliminated
- Amenable to ablation
Malignant Form of Idiopathic VT

- This is a fast, polymorphic ventricular tachycardia
- It is triggered by early PVC’s (R on T phenomenon)
- It may be triggered by PVC’s originating from an outflow tract tachycardia
- Presents as syncope or a survived episode of SCD
- Fortunately, it is very rare and the triggering focus is amenable to ablation
*Idiopathic Left Ventricular Tachycardia*

- Occurs between 15 and 40 years of age with 60% male predominance
- Associated with emotions and exercise
- May be paroxysmal or incessant and result in cardiomyopathy
- Note the VT pattern is a RBBB VT based on V1 and left anterior hemiblock

*Respnds to*

*Note AV dissociation*
Atrial Fibrillation
Atrial Fibrillation

- Classification
- Triggers and Substrate
- Pharmacologic Rhythm Control
- Pulmonary Vein Isolation (Ablation)
- Cardioversion
- Pharmacologic Rate Control
- Pacemaker Implant and AV Node Ablation
The ECG Tracing of Atrial Fibrillation

*Absent P-waves
* Irregularly irregular
To Be Distinguished from Multifocal Atrial Tachycardia

* Discreet P-waves
* Three or more P-wave morphologies
* Rate greater than 100 beats per minute (Rate less than 100 bpm is called wandering atrial pacemaker)
* Irregularly irregular
* Frequentely seen in decompensated COPD
* No direct therapy. Treat the underlying illness
Classification of Atrial Fibrillation

- Paroxysmal AF: Terminates spontaneously or with intervention within 7 days
- Persistent: Continuous AF that is maintained for > 7 days
- Longstanding persistent AF: Sustained AF of > 12 months duration
- Permanent: Term used when there has been a joint decision by the patient and clinician to cease further attempts to restore or maintain sinus rhythm
Triggers and Substrate

- **Triggers**
  - PAC’s originating, most commonly from the region around the Pulmonary veins.
  - The triggering PAC’s are caused by abnormal calcium metabolism

- **Substrate**
  - Fibrosis, inflammation, reduced cell coupling, slow conduction and short effective refractory periods
  - Maintenance of Afib thought due to multiple wavelets or to rotors (small established reentrant circuits)
  - Persistence of AF results in electrophysiologic and structural remodeling of the LA which ultimately prevent restoration of NSR
Primary rhythm control depends upon symptoms, age, duration of the arrhythmia, and presence of tachycardia associated CM.

First treat underlying diseases associated with AF such as thyrotoxicosis, pericarditis, OSA, hypertension, and obesity.

Overview of drugs:
- Amiodarone is the most effective drug in restoring and maintaining NSR.
- Propafenone and flecanide are effective, but can’t be used in presence of structural heart disease (CAD, HF, LVH >1.5 cm on TTE). They must be used in combination with a beta blocker because they can facilitate atrial flutter with 1:1 coduction.
- IV dofetilide is effective and must be initiated in hospital. It causes significant QTc prolongation.
- Sotalol and dronedarone cannot be used in HF.

Several RCT’s have shown no significant long-term survival advantage or freedom from cardiac events when rhythm control is compared to rate control.
Guideline-based Strategy

Strategies for Rhythm Control in Patients With Paroxysmal and Persistent AF

No Structural Heart Disease

- Dofetilide
- Droperidol
- Prazosin
- Propafenone
- Sotalol
- Amiodarone

3. Catheter ablation is only recommended as first-line therapy for patients with paroxysmal AF (Class IIIa recommendation).
4. Drugs are listed alphabetically.
5. Depending on patient preference when performed in experienced centers.
6. Not recommended with severe LV wall thickness >1.5 cm).
7. Should be used with caution in patients at risk for ventricular de points ventricular tachycardia.
8. Should be combined with AV nodal blocking agents.

Structural Heart Disease

- CAD
- HF

3. Catheter ablation
- Dofetilide
- Droperidol
- Prazosin
- Propafenone
- Sotalol
- Amiodarone

- Amiodarone
## Selection of Agents

### Dosage and Safety Considerations for Maintenance of Sinus Rhythm in Atrial Fibrillation (1 of 3)

<table>
<thead>
<tr>
<th>Drug</th>
<th>Usual Dose</th>
<th>Exclude/Use With Caution</th>
<th>Major Pharmacokinetic Drug Interactions</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Vaughan Williams class IA</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Disopyramide</td>
<td>Immediate release: 100-200 mg once every 6 hours • Extended release: 200-600 mg once every 12 hours</td>
<td>HF</td>
<td>Metabolized by CYP3A4; caution with inhibitors (e.g., verapamil, diltiazem, ketoconazole, macrolide antibiotics, protease inhibitors, grapefruit juice) and inducers (e.g., rifampin, phenobarbital, phenytoin)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Prolonged QT interval</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>Propranolol, propranolol, lurasidone, esmolol, celecoxib, irbesartan, valsartan, and losartan into the systemic circulation.</td>
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<tr>
<td></td>
<td></td>
<td>Atorvastatin</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>Metabolism</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>Metabolized by CYP3A4; caution with inhibitors (e.g., verapamil, diltiazem, ketoconazole, macrolide antibiotics, protease inhibitors, grapefruit juice) and inducers (e.g., rifampin, phenobarbital, phenytoin)</td>
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<tr>
<td><strong>Quinidine</strong></td>
<td>324-648 mg every 8 hours</td>
<td>Prolonged QT interval</td>
<td>Inhibits CYP2D6; 9 concentrations of tricyclic antidepressants, benzodiazepines, antipsychotics, morphine, anticoagulant, and digoxin.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Quinidine</td>
<td>Inhibits P-glycoprotein; 6 digoxin concentration.</td>
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<tr>
<td><strong>Vaughan Williams class IC</strong></td>
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</tbody>
</table>

### Dosage and Safety Considerations for Maintenance of Sinus Rhythm in Atrial Fibrillation (2 of 3)

<table>
<thead>
<tr>
<th>Drug</th>
<th>Usual Dose</th>
<th>Exclude/Use With Caution</th>
<th>Major Pharmacokinetic Drug Interactions</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Amiodarone</strong></td>
<td>Oral: 400-600 mg daily in divided doses for 2-4 weeks; maintenance typically 200-250 mg daily</td>
<td>AF</td>
<td>Inhibits most CYPs to cause drug interaction: 9 concentrations of warfarin (HR 0.8-2.00), statins, metoprolol, and other drugs</td>
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<tr>
<td></td>
<td></td>
<td>Prolonged QT interval</td>
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<td></td>
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<td></td>
</tr>
<tr>
<td><strong>Dofetilide</strong></td>
<td>125-500 mg once every 12 hours</td>
<td>Propranolol, digitalis, lurasidone, and losartan into the systemic circulation.</td>
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</tr>
<tr>
<td><strong>Dronedarone</strong></td>
<td>400 mg once every 12 hours</td>
<td>Propranolol, digitalis, lurasidone, and losartan into the systemic circulation.</td>
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### Notes
- The medications listed are for the management of atrial fibrillation.
- Dosages and precautions vary; consult a healthcare provider for specific recommendations.
- Interactions with other medications can be significant; consider ongoing monitoring.
- Caution is advised with the use of these agents, especially in patients with pre-existing cardiac conditions.
- Always review the most current labels and guidelines for the medications mentioned.
### Dosage and Safety Considerations
for Maintenance of Sinus Rhythm in Atrial Fibrillation (3 of 3)

<table>
<thead>
<tr>
<th>Drug</th>
<th>Usual Doses</th>
<th>Exclude/Use With Caution</th>
<th>Major Pharmacokinetic Drug Interactions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vaughan Williams class III</td>
<td></td>
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</tr>
<tr>
<td>Sotalol</td>
<td>40-600 mg once every 12 hours</td>
<td>Prolonged QT interval</td>
<td>None (renal excretion)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Renal disease</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>Hypokalemia</td>
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<tr>
<td></td>
<td></td>
<td>Hypomagnesaemia</td>
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<tr>
<td></td>
<td></td>
<td>Diuretic therapy</td>
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<tr>
<td></td>
<td></td>
<td>Avoid other QT prolonging</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>Antibiotics</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>No AF or AV nodal dysfunction</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>age</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Ejection fraction</td>
<td></td>
</tr>
</tbody>
</table>
Pulmonary Vein Isolation

- Two meta analyses suggest freedom from arrhythmia is superior with ablation compared with pharmacologic therapy
- An RCT looking at risk of survival or stroke showed no difference between ablation and pharmacologic therapy

Techniques
- Catheter-based
- Open surgical (Cox MAZE) for patients having open heart surgery for other reasons

Indications
- Class I: For symptomatic paroxysmal AF refractory to at least one pharmacologic agent
- Class IIa: For symptomatic persistent AF refractory to at least one pharmacologic agent
- Class IIb: For symptomatic longstanding persistent AF refractory to at least one pharmacologic agent
Cardioversion

- Pharmacologic cardioversion
  - Oral flecanide or propafenone
  - IV ibutilide or dofetilide
  - Pill in the pocket
  - All of these presuppose that the patient is anticoagulated

- Direct current cardioversion (DCCV)
  - Heparin and DCCV if arrhythmia began less than 48 hours earlier
  - If > than 48 hours or you are unsure, treat with Vitamin K antagonist or DOAC for 3 weeks before and for 4 weeks after DCCV if CHA2DS2-VASc is <2 or permanently for score of ≥ 2
Pharmacologic Rate Control

- The target for pharmacologic rate control was studied in RCT (RACE II). It compared lenient rate control (<110 bpm) with strict rate control (80 bpm) and found no difference in outcomes at 3 years between them.
The Choice of Agents for Pharmacologic Rate Control

<table>
<thead>
<tr>
<th>Common Medication Dosage for Rate Control of AF</th>
<th>Intravenous Administration</th>
<th>Usual Oral Maintenance Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Beta-Blockers</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Metoprolol tartrate</td>
<td>2.5-5.0 mg IV bolus over 2 minutes; up to 3 doses</td>
<td>25-100 mg bid</td>
</tr>
<tr>
<td>Metoprolol sodium succinate</td>
<td>N/A</td>
<td>50-400 mg qd</td>
</tr>
<tr>
<td>Alprenolol</td>
<td>N/A</td>
<td>25-100 mg qd</td>
</tr>
<tr>
<td>Enalapril</td>
<td>500 mg/kg IV bolus over 1 minute, then 50-300 mcg/kg/minute IV</td>
<td>N/A</td>
</tr>
<tr>
<td>Propranolol</td>
<td>1 mg IV over 1 minute, up to 3 doses at 2 minute intervals</td>
<td>10-40 mg bid or qd</td>
</tr>
<tr>
<td>Nadolol</td>
<td>N/A</td>
<td>10-240 mg qd</td>
</tr>
<tr>
<td>Carvedilol</td>
<td>N/A</td>
<td>3.125-25 mg bid</td>
</tr>
<tr>
<td>Bisoprolol</td>
<td>N/A</td>
<td>2.5-10 mg qd</td>
</tr>
<tr>
<td><strong>Non-Hydropyridine Calcium Channel Antagonists</strong></td>
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</tr>
<tr>
<td>Verapamil</td>
<td>0.075-0.15 mg/kg IV bolus over 2 minutes, may give an additional 10.0 mg after 30 minutes if no response, then 0.005 mg/kg/minute infusion</td>
<td>180-480 mg qd (ER)</td>
</tr>
<tr>
<td>Diltiazem</td>
<td>0.25 mg/kg IV bolus over 2 minutes, then 0.25 mg/8 hours</td>
<td>120-360 mg qd (ER)</td>
</tr>
<tr>
<td><strong>Digitalis Glycerides</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Digoxin</td>
<td>0.25 mg IV with repeat dosing to a maximum of 1.5 mg over 24 hours</td>
<td>0.125-0.25 mg qd</td>
</tr>
<tr>
<td><strong>Others</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Amiodarone</td>
<td>300 mg IV over 1 hour, then 10-50 mg/hour over 24 hours</td>
<td>100-200 mg qd</td>
</tr>
</tbody>
</table>
Catheter Ablation of the AV Junction

- Requires initial placement of a permanent pacemaker and renders the patient pace-dependent
- Carries the risk of causing a cardiomyopathy as a result of RV apical pacing and the associated LV dys-synchrony
  - Biventricular pacing should be used for all patients with HF and LVEF < 35%
  - An RCT, the BLOCK HF Trial, demonstrated the benefit of using biventricular pacing in all patients with LVEF <50% at baseline
  - Any patient who develops dyssynchrony-related cardiomyopathy should be upgraded to a biventricular device
- The lower pacing rate is set initially at 90-100 beats per minute and tapered over the next several months to prevent torsade de pointes or VF immediately after ablation
Atrial Flutter
Atrial Flutter

- Classification
  - Isthmus-dependent atrial flutter
    - Counter-clockwise (typical)
    - Clockwise (atypical)
  - Non-isthmus-dependent atrial flutter
  - Coarse atrial fibrillation versus atypical flutter
- Treatment
  - Pharmacologic rate control
  - Non-pharmacologic rate control
  - Pharmacologic rhythm control
  - Direct current cardioversion
- Anticoagulation in atrial flutter
Classification

- Isthmus-dependent
  - Cavo-tricuspid isthmus is a narrow channel between the IVC and the tricuspid valve that is part of the macro-re-entry circuit

- Non-Isthmus dependent
  - The reentrant circuit does not involve this region
Isthmus-Dependent Atrial Flutter

*Counter-clockwise loop is typical form of atrial flutter
*Counter-clockwise loop may occur
*They both include the cavo-tricuspid isthmus
*The wave front enters the LA at three levels
The ECG in Isthmus-dependent flutter
Non-Isthmus-Dependent Atrial Flutter

- May be seen late after cardiac surgery (closure of ASD, MV replacement) or after pulmonary vein ablation.
- Re-entry circuits develop around scars, or in the case of prior ablation, at gaps in the ablation lines.
The ECG in Non-isthmus-dependent Atrial Flutter

*Absence of a true sawtooth pattern
Coarse Atrial Fibrillation versus Atypical Atrial Flutter

- High amplitude waves which could reflect an atypical pattern of flutter
- Wave to wave variation speaks to coarse atrial fib rather than atrial fibrillation
Rate Control

- Pharmacologic Rate control
  - Similar approach as in AF. Single or combined AV nodal blocking drugs
  - The target rate range is the same
  - AV block is more difficult to achieve than in AF. In AF, the irregular impingement of the AVN allows the AV node to exert its intrinsic conduction delay. Not so in flutter.
  - High grade AV block may be induced suddenly

- Nonpharmacologic Rate control
  - Placing a permanent pacer to allow high doses of AV node blocking drugs
Rhythm Control

- Pharmacologic rhythm control
  - Flecanide, propafenone, sotalol, amiodarone may be used
  - Pharmacologic cardioversion with IV dofetilide or ibutilide is possible

- Ablation
  - Isthmus-dependent flutter has 95% success rate
  - Non-isthmus dependent atrial flutter has less success. The arrhythmia has to be inducible and must be mapped before an ablation strategy can be attempted

- Direct current cardioversion
  - Cardioversion can usually be achieved with 100 J rather than the 200+ J required to convert AF
Anticoagulation in Atrial Flutter

- Exactly the same approach as with AF
Sudden Cardiac Death Syndromes in Patients with Structurally Normal Hearts

- Long QT Syndromes
- Brugada Syndrome
- Catecholaminergic Polymorphic Ventricular Tachycardia
- Short QT Syndrome
Sudden Cardiac Death Syndromes in Patients with Structurally Normal Hearts
Long QT Syndrome
Long QT Syndrome Characteristics

- Inherited disorder characterized by long QTc and often T-wave abnormalities.
- Diagnosis: QTc > 480 msec, syncope or sudden death often manifesting before age 20, and a family history of the same.
- There are three ECG phenotypes and 15 genes identified that code for cell membrane proteins that form the ion channels that maintain the resting membrane potential and action potential propagation.
- There are two clinical syndromes with sensorineural deafness and skeletal abnormalities (Jervell and Lange-Nielsen syndrome).
Long QT Syndrome Treatment

- Avoid QT prolonging drugs (www.crediblemeds.org)

- Beta blocker: Nadolol (1-2.5 mg/kg) or Metoprolol (2-4 Mg/kg) for patients with asthma
  - For all patients with the clinical diagnosis (Class I)
  - For silent mutation carriers (Class IIb)

- Avoid competitive sports

- ICD
Brugada Syndrome
Overview of Brugada Syndrome

- Arrhythmogenic syndrome with incomplete or complete RBBB, j-point elevation, and a descending ST segment
- Prevalence 1: 1000-1:5000 in US and higher in Far East Countries
- Autosomal dominant with variable penetrance and 8:1 male:female
- Mean age of occurrence: 40
- The ECG pattern can be exposed by a challenge with ajmaline or flecaïnide, both Class Ic drugs
- 21 genes coding for ion channel proteins are associated with it
Treatment of Brugada Syndrome

- Avoid specific drugs including antiarrhythmics psychotropic drugs, anesthetics (www.brugadadrugs.org)
- Treat fever
- Avoid competitive sports
- ICD may be helpful in symptomatic patients, there are multiple therapies delivered and the ICD must be programmed to withhold therapy sufficiently long for a VT episode to spontaneously resolve. Risk stratification is difficult
Catecholaminergic Polymorphic VT
Characteristics of Catecholaminergic Polymorphic Ventricular Tachycardia

- Patients experience episodes of bi-directional polymorphic ventricular tachycardia (also seen in digitalis intoxication)
- Triggered by exercise or emotions. Each patient has a particular HR between 100 and 120 at which the arrhythmia reproducibly appears
- Mean age of onset 12-15 years of age
- Prevalence 1:7000 to 1:10,000
- Several genes coding for the sarcoplasmic reticulum have been identified
- The pathophysiology of the arrhythmias is calcium overload of the cytosol from the sarcoplasmic reticulum causing delayed after-potentials
Treatment of Catecholaminergic Polymorphic Ventricular Tachycardia

- Beta blockers for symptomatic patients and asymptomatic gene carriers (Class I). Also asymptomatic gene carriers who are young
  - Nadolol (1.5-2.5 mg/kg) and propranolol (2-4.4 mg/kg) are preferred and flecainide (100-200 mg bid) may be added
- Avoidance of physical exertion
- ICD for patients symptomatic despite drugs and for those with SCD
  - Precautions in programming needed to prevent excessive therapies
Characterization of the Short QT syndrome

- An arrhythmic disorder causing sudden cardiac death and crib death that is identified by a QTc < 360 msec
- Prevalence < 1:10,000
- There is overlap with healthy patients who have low normal QTc
- Bi modal occurrence of cardiac arrest:
  - 4% risk in the first year of life
  - 1.3% between 20 and 40 years
  - 41% occurrence of cardiac arrest by age 40.
- Ventricular arrhythmias are inducible in 90% of these patients and don’t correlate with likelihood of clinical events
- No single gene accounts for > 10% of cases. The involved genes code for ion channel proteins
- Treatment is ICD for secondary prevention. No drugs are effective for primary prevention
Acquired Long QT Syndrome

- 1-10% of patients who take a QT prolonging drug experience polymorphic VT. [www.crediblemeds.org](http://www.crediblemeds.org)
- The drugs reduce the function of one of the potassium channels in cardiac myocytes resulting in delayed egress of potassium from the cell, delaying repolarization
- 36% of patients who experience polymorphic VT as a result of drug induced QT prolongation are found to have a mutation in one of the potassium channel proteins and so may have had diminished repolarization reserve to begin with.

**Treatment:**
- Discontinue the offending drug
- Magnesium sulfate infusion at 2 gm/hour
- Overdrive cardiac pacing
- Isoproterenol infusion
Vaughan Williams classification of antiarrhythmic drugs

- **Class I:** block sodium channels
  - Ia (quinidine, procainamide, disopyramide) ↑AP
  - Ib (lignocaine) ↓AP
  - Ic (flecainide) ↔AP
- **Class II:** β-adrenoceptor antagonists (atenolol, sotalol)
- **Class III:** prolong action potential and prolong refractory period (suppress re-entrant rhythms) (amiodarone, sotalol)
- **Class IV:** Calcium channel antagonists. Impair impulse propagation in nodal and damaged areas (verapamil)