I. **Brief review of physiological concepts:**

a. The heart contains specialized cells that exhibit automaticity, i.e. can generate rhythmic action potentials in the absence of external stimuli.

b. The heart requires well-synchronized repetitive electrical activity for optimal mechanical performance.

c. The sino-atrial node (SA) sets the pace (i.e. pace maker) for the electrical activity of the heart.

d. Abnormal heart tissue is usually depolarized, which reduces Na current, and decreases conduction velocity.

e. Recovery time of Na channels from excitation/depolarization contributes to the refractory period and is increased by many drugs.
**Schematic diagram of ion permeability**

**PHASE 0: FAST UPSTROKE**
- Na⁺ channels open ("fast channels") resulting in a fast inward current.
- Upstroke ends as Na⁺ channels are rapidly inactivated.
- Sodium current is blocked by anti-arrhythmics agents, such as quinidine.

**PHASE 1: PARTIAL REPOLARIZATION**
- The initial rapid phase of repolarization is due to:
  1) Inactivation of Na⁺ channels.
  2) K⁺ channels rapidly open and close causing a transient outward current.

**PHASE 2: PLATEAU**
- Voltage-sensitive Ca²⁺ channels open, resulting in slow inward (depolarizing) current that balances the slow (polarizing) outward leak of K⁺.

**PHASE 3: REPOLARIZATION**
- Ca²⁺ channels close.
- K⁺ channels open resulting in an outward current leading to membrane repolarization.
- The net result of the action to this point is a net gain of Na⁺ and loss of K⁺. This imbalance is corrected by Na⁺/K⁺ ATPase.

**PHASE 4: FORWARD CURRENT**
- Increasing depolarization results from gradual increase in sodium permeability.
- The spontaneous depolarisation automatically brings the cell to the threshold of the next action potential.
II. Characteristics of arrhythmias

A. Definitions:
1. normal sinus rhythm (60-90bpm), set by SA node pacemaker
2. arrhythmia; any abnormality of firing rate, regularity or site of origin of cardiac impulse or disturbance of conduction that alters normal sequence of activity of atria and ventricles.

B. Occurrence:
1. more than 80% of patients with acute myocardial infarctions
2. 50% of anaesthetized patients
3. less than 25% of patients on digitalis

C. Classification of arrhythmia characteristic or sites involved:
1. characteristics:
   a. flutter – very rapid but regular contractions
   b. tachycardia – increased rate
   c. bradycardia – decreased rate
   d. fibrillation – disorganized contractile activity

2. sites involved:
   a. ventricular
   b. atrial
   c. sinus
   d. AV node
   e. Supraventricular (atrial myocardium or AV node)

III. Mechanisms of arrhythmias

1. Abnormal impulse generation (abnormal automaticity)
   a. automaticity of normally automatic cells (SA, AV, His)
   b. generation of impulses in normally non-automatic cells
      i. phase 4 depolarization in normally non-automatic cells
      ii. ‘triggered activity’ due to afterdepolarizations
         - early afterdepolarization
         - delayed afterdepolarization
2. Abnormal impulse conduction (more common mechanism)
   - damaged tissue usually depolarized → ↓ conduction velocity
   a. AV block – ventricle free to start own pacemaker rhythm
   b. Re-entry: re-excitation around a conducting loop, which produces tachycardia
      i. unidirectional conduction block
      ii. establishment of new loop of excitation
      iii. conduction time that outlasts refractory period

IV. General strategy of antidysrhythmic drugs

Suppression of dysrhythmias

A. Alter automaticity
   i. decrease slope of Phase 4 depolarization
   ii. increase the threshold potential
   iii. decrease resting (maximum diastolic) potential

B. Alter conduction velocity
   i. mainly via decrease slope of Phase 0 upstroke
   ii. decrease rate of rise of Phase 4 depolarization
   ii. decrease membrane resting potential and responsiveness

C. Alter the refractory period
   i. increase Phase 2 plateau
   ii. increase Phase 3 repolarization
   iii. Increase action potential duration

IV. Classification of Antidysrhythmic Drugs

A. Vaughan-Williams classification (1970), subsequently modified by Harrison.
   1. based on the pattern of electrophysiological actions in normal tissue
   2. presumes a mechanism of action of antidysrhythmic drugs
   3. consists of four main classes and three subclasses
   4. does not include agents (ie. Adenosine, digoxin) that act at other sites
Table 1a. **Class I** directly block Na channels. All behave like local anaesthetics. Subclasses based on differing effects on AP duration (APD) and degree of Na channel block (Phase 0).

<table>
<thead>
<tr>
<th>Subclass</th>
<th>Mechanism</th>
<th>Examples</th>
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<tbody>
<tr>
<td>IA</td>
<td>Na channel blocker</td>
<td>Quinidine, Procainamide</td>
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<td>Mod.block Ph.0; slow conduction; increase APD</td>
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<tr>
<td>IB</td>
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<td>Lidocaine, Tocainide, Mexiletine</td>
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<tr>
<td></td>
<td>Min.block Ph 0; slow conduction; shorten phase 3 repolarization, decrease APD</td>
<td></td>
</tr>
<tr>
<td>IC</td>
<td>Na channel blocker</td>
<td>Flecainide, Encainide</td>
</tr>
<tr>
<td></td>
<td>Marked block Ph.0; slow conduction.; no change APD or repolarization. Increased suppression of Na channels</td>
<td></td>
</tr>
</tbody>
</table>

Class II
Beta blockers decrease adrenergic input. No effect APD, suppress phase 4 depolarization.

Class III
K channel blockade Prolong repolarization/refractory period other means than exclusively INa block (mainly).

Class IV
Ca channel blockers Slow conduction and increase effective refractory period in normal tissue (A-V node) and Ca-dependent slow responses of depolarized tissue (atria, ventricle, Purkinje).

Others Adenosine, Digoxin, Anticoagulants

Table 1b. **Effects Of Therapeutic Concentrations Of Antiarrhythmic Drugs On Electrophysiological Properties Of Specialized Cardiac Fibers**

<table>
<thead>
<tr>
<th>CLASS OF ANTIARRHYTHMIC DRUG</th>
<th>IA</th>
<th>IB</th>
<th>IC</th>
<th>II</th>
<th>III</th>
<th>IV</th>
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<tbody>
<tr>
<td><strong>Sinus node</strong></td>
<td></td>
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<tr>
<td>Automaticity</td>
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<td>↑</td>
<td>0</td>
<td>0</td>
<td>↓</td>
<td>↓</td>
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<tr>
<td><strong>AV node</strong></td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Effective refractory period (ERP)</td>
<td>↓</td>
<td>0</td>
<td>↑</td>
<td>0</td>
<td>↓</td>
<td>↑</td>
</tr>
<tr>
<td><strong>Purkinje fibers</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Action potential amplitude</td>
<td>↓</td>
<td>0</td>
<td>↓</td>
<td>↑</td>
<td>↓</td>
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<td>0</td>
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<tr>
<td>Action potential duration (APD)</td>
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<td>↓</td>
<td>↓</td>
<td>0</td>
<td>↑</td>
<td>0</td>
</tr>
<tr>
<td>Effective refractory period (ERP)</td>
<td>↑</td>
<td>↓</td>
<td>↓</td>
<td>0</td>
<td>↑</td>
<td>0</td>
</tr>
<tr>
<td>ERP/APD</td>
<td>↑</td>
<td>↑</td>
<td>↑</td>
<td>↑</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Membrane responsiveness</td>
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<td>0</td>
<td>↓</td>
<td>↓</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Automaticity</td>
<td>↓</td>
<td>0</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
<td>0</td>
</tr>
</tbody>
</table>

* Changes are indicated as follows: Ø, decreased; 0, no change; ≠, increased; where bidirectional arrows are shown, there is variability in the direction of change. Boldface arrows indicate effects of greater magnitude. † Bretyllium only; due to release of catecholamines on initial exposure to the drug. ‡ Due to a complex balance of direct and indirect autonomic effects.
V. **Specific examples** of drugs from each Vaughan-Williams class and their key aspects

A. **Quinidine** (Class IA prototype; *Quinaglute, Quinidex, Cardioquin*)

   others: Procainamide, Disopyrimide

1. General properties:
   a. use decreasing, significant side effects
   b. D-isomer active
   c. As with most of the Class I agents
      - moderate block of sodium channels
      - decreases automaticity of pacemaker cells
      - increases effective refractory period/AP duration

2. Cardiac effects of quinidine
   a. Decreases automaticity, conduction velocity and excitability
   b. Preferentially blocks open Na channels
   c. Recovery from block slow in depolarized tissue; lengthens refract. period
   d. All effects are potentiated in depolarized tissues
   e. Increases action potential duration (APD) and prolongs AP repolarization via block of K channels; decreases reentry
   f. Indirect action: anticholinergic effect (accelerates heart), which can speed A-V conduction. Better agents available for treating atrial tachycardia because of enhancement of A-V transmission to ventricle and significant side effects.

3. Extracardiac
   a. blocks alpha-adrenoreceptors to yield vasodilatation.
   b. blocks muscarinic receptors (ie. blurred vision, dilated pupils)
4. Toxicity
   a. Cardiac
      - "Quinidine syncope" (fainting)- due to disorganized ventricular tachycardia
      - associated with greatly lengthened Q-T interval; can lead to Toursades de Pointes (precursor to ventricular fibrillation)
      - negative inotropic action (decreases contractility)
   b. Extracardiac
      - GI - diarrhea, vomiting
      - CNS - headaches, nausea, dizziness, tinnitus (quinidine "Cinchonism")

5. Pharmacokinetics/therapeutics
   a. Oral, rapidly absorbed, 80% bound to membrane proteins
   b. Hydroxylated in liver; $T_{1/2} = 6-8$ h
   c. Drug interaction: displaces digoxin from binding sites; so avoid giving drugs together
   d. Some active metabolites of quinidine
   e. Effective in treatment of nearly all dysrhythmias, including:
      1) Premature atrial contractions
      2) Paroxysmal atrial fibrillation and flutter
      3) Intra-atrial and A-V nodal reentrant dysrhythmias
      4) Wolff-Parkinson-White tachycardias (short PR, long QRS)
   f. Useful in chronic dysrhythmias requiring outpatient treatment

B. Procainamide (*Procanbid, Procan; Class 1A*)
   1. Cardiac effects
      a. Very similar to quinidine in general but less blockade of muscarinic and adrenergic receptors
      b. Has negative inotropic action
   2. Extracardiac effects
      a. Ganglionic blocking reduces peripheral vascular resistance
   3. Toxicity
      a. Cardiac: Similar to quinidine; cardiac depression
      b. Noncardiac: Syndrome resembling lupus erythematosus
4. Pharmacokinetics/therapeutics
   a. Administered orally, I-V and intramuscularly
   b. Major liver metabolite is N-acetylprocainamide (NAPA), a weak Na+ channel blocker with class III activity. Bimodal distribution in population of rapid acetylators, accumulate high levels of NAPA.
   c. $T_{1/2} = 3-4$ hours; necessitates frequent dosing; kidney chief elimination path. NAPA has longer $T_{1/2}$ and accumulates readily.
   d. Usually used short-term. Common choice in CCUs for ventricular dysrhythmias associated with acute MIs (more effective than Lidocaine).

C. Lidocaine (*Xylocaine*; Class IB prototype)
   Other examples: Phenytoin (Dilantin), Tocainide (Tonocard), Mexiletine (Mexitil)
   - similar to Lidocaine but effective orally
   a. Use as antidysrhythmic agent in emergency care (AHA 2001) decreased
   b. Given I-V and I-M; commonly used in ICU-critical care units (old DOC).
   c. Very low toxicity, good therapeutic index
   d. A local anesthetic, works on nerve at higher doses.

2. Cardiac effects
   a. Generally decreases APD, hastens AP repolarization, decreases automaticity and increases refractory period in depolarized cells.
   b. Exclusively acts on Na channels in depolarized tissue by blocking open and inactivated Na channels
   c. Potent suppresser of abnormal activity
   d. Most Na channels of normal cells rapidly unblock from lidocaine during diastole; few electrophysiological effects in normal tissue

3. Toxicity:
   - least cardiotoxic, high dose can lead to hypotension
   - tremors, nausea, slurred speech, CNS stimulation & convulsions
4. Pharmacokinetics/therapy
   a. I-V, I-M since extensive first pass hepatic metabolism, never oral
   b. $T_{1/2} = 0.5-4$ hours
   c. Effective in suppressing dysrhythmia associated with depolarized tissue (ischemia; digitalis toxicity); ineffective against dysrhythmias in normal tissue (atrial flutter).
   d. Suppresses ventricular tachycardia; prevents fibrillation after acute MI; rarely used in supraventricular arrhythmias.

D. Phenytoin (*Dilantin*, Class IB)
   1. Non-sedative anticonvulsant used in treating epilepsy
   2. Limited efficacy as antidysrhythmic (second line antiarrythmic)
   3. Suppresses ectopic activation by blocking Na and Ca channels
   4. Especially effective against digitalis-induced dysrhythmias
   5. $T_{1/2} = 24$ hr - metabolized in liver
   6. Use associated with gingival hyperplasia

E. Flecainide (*Tambocor*, Class IC prototype)
   Other examples: Lorcaidine, Propafenone (Rythmol), Indecainide, Moricizine.
   Depress rate of rise of AP without change in refractoriness or APD in normally polarized cells
   1. Decreases APD, decreases automaticity, conduction in depolarized cells.
   2. Marked block of open Na channels ($\downarrow$ Ph. 0); no change repolarization.
   3. Used primarily for ventricular dysrhythmias but effective for atrial too
   4. No antimuscarinic action
   5. Suppresses premature ventricular contractions
   6. Associated with significant mortality; use limited to last resort
F. **Propranolol** (*Inderal*; Class II, beta adrenoreceptor blockers)
   Others: Metoprolol, Esmolol, Sotalol (also Class III), Acebutolol

1. General properties of all Class II agents:
   a. Slow A-V conduction
   b. Prolong A-V refractory period
   c. Suppress automaticity

2. Cardiac effects (of propranolol), a non-selective beta blocker
   a. Main mechanism of action is block of beta receptors; ↓ Ph 4 slope, which decreases automaticity under certain conditions
   b. Some direct local anesthetic effect by block of Na channels (membrane stabilization)-at higher doses
   c. Increases refractory period in depolarized tissues
   d. Increases K channel current
   e. Increases A-V nodal refractory period

3. Non-cardiac: Hypotension

4. Therapeutics
   a. Blocks abnormal pacemakers in cells receiving excess catecholamines (e.g. pheochromocytoma) or having up-regulated betas (thyroid disorder)
   b. Blocks A-V nodal reentrant tachycardias; inhibits ectopic foci
   c. Propranolol used to treat supraventricular tachydysrhythmias; objective to slow the ventricular rate by beta affects on A-V conduction rather than abolish dysrhythmias.
   d. Contraindicated in patients with ventricular failure; also can lead to A-V block.
   e. Oral (propranolol) or IV. Extensive metabolism in liver.

G. **Amiodarone** (*Cordarone*; Class III; other class III: *Ibutilide*, *Bretylium*, *Sotalol*)

General

a. new frontline agent for ventricular tachycardia (American Heart Association guidelines 2001), prolongs refractory period by blocking potassium channels, very long T1/2 (>25days).
   b. also member of Classes IA,II,III,IV since blocks Na, K, Ca channels and alpha and beta adrenergic receptors
   c. has significant side effects (cardiac depression, pulmonary fibrosis, corneal microdeposits, photosensitivity)
   d. effective against atrial, A-V and ventricular dysrhythmias
H. **Bretylium** (*Bretylol;* Class III, K channel blockers; others, *ibutilide*)

1. General: originally used as an antihypertensive agent, availability limited

2. Cardiac effects
   
a. Direct antidysrhythmic action
b. Increases ventricular APD and increases refractory period; decreases automaticity
c. Most pronounced action in ischemic cells having short APD
d. Initially stimulates and then blocks neuronal catecholamine release from adrenergic nerve terminals
e. Blocks cardiac K channels to increased APD.

3. Extracardiac effects: Hypotension (from block of NE release)

4. Pharmacokinetics/therapeutics
   
a. IV or intramuscular
b. Excreted mainly by the kidney
c. Usually emergency use only: ventricular fibrillation when lidocaine and cardioversion therapy fail. Increases threshold for fibrillation.
d. Decreases tachycardias and early extrasystoles by increasing effective refractory period
I. **Verapamil** *(Isoptin; Calan; Class IV, Ca channel blockers; other eg. diltiazem)*

1. **Cardiac**
   a. Blocks active and inactivated Ca channels, prevents Ca entry
   b. More effective on depolarized tissue, tissue firing frequently or areas where activity dependent on Ca channels (SA node; A-V node)
   c. Increases A-V conduction time and refractory period; directly slows SA and A-V node automaticity
   d. Suppresses oscillatory depolarizing after depolarizations due to digitalis

2. **Extracardiac**
   a. Peripheral vasodilatation via effect on smooth muscle
   b. Used as antianginal, antihypertensive
   c. Hypotension may increase HR reflexively.

3. **Toxicity**
   a. **Cardiac**
      1) Negative inotropic activity bad for damaged heart
      2) Can produce full A-V block
      3) Increase toxicity to cardiac glycosides
   b. **Extracardiac**
      Dizziness, constipation, nervousness, gingival hyperplasia
4. Pharmacokinetics/Therapeutics
   a. $T_{1/2} = 7$ h, metabolized by liver
   b. Oral administration; also available parenterally
   c. Caution for patients with liver disease
   d. Blocks reentrant supraventricular tachycardia (“A-V nodal reentrant tachycardia”), decreases atrial flutter and fibrillation
   e. Only moderately effective against ventricular arrhythmias

J. Others

1. **Adenosine**: i.v. ($T_{1/2} 15$ sec), activates P1 purinergic receptors (A1) coupled to K channels $\rightarrow \uparrow$ RP, $\downarrow$ CV

2. **Potassium ions (K+)**: Depress ectopic pacemakers, $\downarrow$ membrane potential

3. **Digoxin**: depressant action on A-V node (vagal stimulation), used for atrial flutter and fibrillation. Purkinje fibers $\rightarrow \uparrow$ RP, $\downarrow$ CV

4. **Autonomic agents**: Beta-agonists, anticholinergics, used to treat AV block

5. **Anticoagulant therapy**: adjunct, prevent formation of systemic emboli & stroke

6. **Magnesium ions**: unknown mechanism, useful when [Mg++] low

### Important considerations for treating Dysrrhythmias

1. **Acute vs chronic treatment**

2. **Ventricular vs supraventricular**

<table>
<thead>
<tr>
<th></th>
<th>Acute</th>
<th>Chronic</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Supraventricular</strong></td>
<td>Adenosine, Digoxin</td>
<td>Beta-blocker, Calcium antagonist</td>
</tr>
<tr>
<td><strong>Ventricular</strong></td>
<td>Amiodarone, Procainamide, Sotalol, Bretylium, Lidocaine</td>
<td>Amiodarone, Sotalol, Flecaainide</td>
</tr>
</tbody>
</table>
### Summary of Effects of Antiarrhythmic Drugs

<table>
<thead>
<tr>
<th>Drug</th>
<th>Class</th>
<th>Auto</th>
<th>CV</th>
<th>RP</th>
<th>APD</th>
<th>ANS effects</th>
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<tbody>
<tr>
<td>Quinidine</td>
<td>IA</td>
<td>↓</td>
<td>↓</td>
<td>↑</td>
<td>↑</td>
<td>vagal, α-block</td>
</tr>
<tr>
<td>Procainamide</td>
<td>IA</td>
<td>↓</td>
<td>↓</td>
<td>↑</td>
<td>↑</td>
<td>vagal, α-block</td>
</tr>
<tr>
<td>Disopyramide</td>
<td>IA</td>
<td>↓</td>
<td>↓</td>
<td>↑</td>
<td>↑</td>
<td>vagal, α-block</td>
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<td>β-block</td>
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<td>β-block</td>
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<tr>
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<td>↓↑</td>
<td>↑↑</td>
<td>↑↑</td>
<td>β-block</td>
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<tr>
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<td>↓</td>
<td>↑↑</td>
<td>↑↑</td>
<td>α-, β-block</td>
</tr>
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<td>↑↑</td>
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<td>vagal stimulation</td>
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<tr>
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### Summary of Pharmacokinetic Properties of Antiarrhythmic Drugs

<table>
<thead>
<tr>
<th>Drug</th>
<th>Class</th>
<th>Plasma Binding %</th>
<th>$T_{1/2}$ (hrs)</th>
<th>Drug Excretion Unchanged</th>
</tr>
</thead>
<tbody>
<tr>
<td>Quinidine</td>
<td>IA</td>
<td>60</td>
<td>6</td>
<td>20-40%</td>
</tr>
<tr>
<td>Procainamide</td>
<td>IA</td>
<td>15</td>
<td>4</td>
<td>60%</td>
</tr>
<tr>
<td>Disopyramide</td>
<td>IA</td>
<td>39-95</td>
<td>5</td>
<td>50-70%</td>
</tr>
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<td>Lidocaine</td>
<td>IB</td>
<td>40</td>
<td>2</td>
<td>&lt;10%</td>
</tr>
<tr>
<td>Tocainide</td>
<td>IB</td>
<td>10</td>
<td>14</td>
<td>40%</td>
</tr>
<tr>
<td>Mexiletine</td>
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<td>12</td>
<td>10%</td>
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<td>45</td>
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<td>40%</td>
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<tr>
<td>Propafenone</td>
<td>IC</td>
<td>85</td>
<td>5</td>
<td>&lt;1%</td>
</tr>
<tr>
<td>Propranolol</td>
<td>II</td>
<td>90</td>
<td>4</td>
<td>&lt;1%</td>
</tr>
<tr>
<td>Acebutolol</td>
<td>II</td>
<td>25</td>
<td>3</td>
<td>40%</td>
</tr>
<tr>
<td>Esmolol</td>
<td>II</td>
<td>(esterase)</td>
<td>9 min</td>
<td>&lt;1%</td>
</tr>
<tr>
<td>Sotalol</td>
<td>II/III</td>
<td></td>
<td>9</td>
<td>80%</td>
</tr>
<tr>
<td>Amiodarone</td>
<td>III</td>
<td>95</td>
<td>25 days</td>
<td>&lt;1%</td>
</tr>
<tr>
<td>Bretylium</td>
<td>III</td>
<td>5</td>
<td>9</td>
<td>80%</td>
</tr>
<tr>
<td>Verapamil</td>
<td>IV</td>
<td>90</td>
<td>5</td>
<td>2%</td>
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<tr>
<td>Adenosine</td>
<td></td>
<td></td>
<td>15 secs</td>
<td></td>
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K. Dysrhythmics treatment summary

This common arrhythmia involves multiple ectopic foci of atrial cells creating a chaotic movement of impulses through the atria. The ventricular response is rapid (100-150 beats per minute) and irregular.

NOTE: Amiodarone use for acute ventricular tachycardia preferred over Lidocaine

L. References