Arrhythmias and conduction disturbances

Supraventricular Tachyarrhythmias
Alterations in Normal Rhythm

- **Bradycardia**
  - “conventional”: < 60 beats/min
  - More useful: < 50 beats/min

- **Tachycardia**
  - “conventional” > 100 beats/min
  - More useful: > 90 beats/min
Rates

- Bradyarrhythmias
  - Sinus Bradycardia
  - Sick Sinus Syndrome

- AV Nodal Blockade
  - First Degree
  - Second Degree
    - Mobitz I
    - Mobitz II
  - Third Degree
    - Complete Heart Block
Rates

- **Tachyarrhythmias**
  - **Supraventricular**
    - Originate from foci above or within the atrioventricular node
    - All the favorites
      - AV nodal reentrant tachycardia (SVT)
      - Atrial flutter
      - Atrial fibrillation
Location

- Supraventricular Arrhythmias
  - Originate from foci above or within the atrioventricular node

- Ventricular Arrhythmias
  - Non-sustained ventricular tachycardia
  - Sustained ventricular tachycardia
    - Stable
    - Unstable
      - ACLS
  - Ventricular fibrillation
    - Never a stable rhythm
    - Immediate ACLS
Sinus Bradycardia

- Sinus rhythm with a resting heart rate of 60 beats/minute or less
- Commonly, sinus bradycardia is an incidental finding in otherwise healthy individuals, particularly in young adults or sleeping patients
Sinus Bradycardia

- Other causes of sinus bradycardia are related to increased vagal tone.
  - Physiologic causes include the bradycardia seen in athletes.
  - Pathologic causes include:
    - Inferior wall myocardial infarction
    - Toxic or environmental exposure
    - Electrolyte disorders
    - Infection
    - Sleep apnea
    - Drug effects
    - Hypoglycemia
    - Hypothyroidism
    - Increased intracranial pressure.
Sinus Bradycardia

- **History:**
  - Symptoms may include the following:
    - Syncope
    - Dizziness
    - Lightheadedness
    - Chest pain
    - Shortness of breath
  - Elements of the history include:
    - Previous cardiac history (e.g., myocardial infarction, congestive heart failure, valvular failure)
    - Medications
    - Toxic exposures
    - Prior illnesses
Sinus Bradycardia

- **Physical:**
  - Cardiac auscultation and palpation of peripheral pulses reveal a slow, regular heart rate.
  - The physical examination is generally nonspecific, although it may reveal the following signs:
    - Decreased level of consciousness
    - Cyanosis
    - Peripheral edema
    - Pulmonary vascular congestion
    - Dyspnea
    - Poor perfusion
    - Syncope
Sinus Bradycardia

- **Causes:**
  - One of the most common pathologic causes of symptomatic sinus bradycardia is the **sick sinus syndrome**.
  - The most common medications responsible include therapeutic and supratherapeutic doses of:
    - digitalis glycosides
    - beta-blockers
    - calcium channel-blocking agents.
  - Other cardiac drugs less commonly implicated include class I antiarrhythmic agents and amiodarone.
Sinus Bradycardia

- **Causes:**
  - other drugs and toxins - *lithium, paclitaxel, toluene, dimethyl sulfoxide (DMSO), topical ophthalmic acetylcholine, fentanyl, alfentanil, sufentanil, reserpine, and clonidine.*
  - Sinus bradycardia may also be seen in *hypothermia, hypoglycemia, hypothyroidism and sleep apnea.*
  - Less commonly, the sinus node may be affected as a result of *diphtheria, rheumatic fever, or viral myocarditis.*
Sinus Bradycardia

- **Lab Studies:**
  - Reasonable screening studies, especially if the patient is symptomatic and this is the initial presentation, include the following:
    - Electrolytes
    - Glucose
    - Calcium
    - Magnesium
    - Thyroid function tests
    - Toxicologic screen
Sinus Bradycardia

- Imaging Studies:
  - Routine imaging studies are rarely of value in the absence of specific indications.

- Other Tests:
  - 12-lead ECG may be performed to confirm the diagnosis.
Sinus Bradycardia

- **Treatment**
  - **Asymptomatic**
    - No treatment required
  - **Symptomatic**
    - Treatment aimed at restoring normal sinus rate
      - Specific to etiology of bradycardia
        - If patient is on rate controlling medications-stop them.
        - If patient is hypokalemic-replace it.
        - If the patient is hypothyroid-replace it (you get the idea)
    - **Permanent pacemaker** if the patient has continued symptoms with no improvement from intervention or with no identifiable cause.
AV Block

- Atrioventricular Block
  - Not truly part of the bradyarrhythmias, but usually slow.
  - Varying degrees
    - Think of them as burns…the higher the degree, the worse they are.
First-degree AV block

- **Definition:**
  - Prolongation of the PR interval on the ECG to more than 200 msec.

- **Pathophysiology:**
  - Every atrial impulse is transmitted to the ventricles, resulting in a regular ventricular rate.
  - Can arise from delays in the conduction system in the AV node itself (most common), the His-Purkinje system, or a combination of both.
First-degree AV block

- **Mortality/Morbidity:**
  - In and of itself, first-degree AV block is a benign condition, with no associated increase in morbidity or mortality.

- **Treatment**
  - If underlying condition suspected (drug overdose, acute MI, myocarditis, etc) treat that condition.
  - No treatment indicated if asymptomatic.
Second-degree AV Block

- Refers to a disorder of the cardiac conduction system in which some atrial impulses are not conducted to the ventricles.
- Electrocardiographically, some P waves are not followed by a QRS complex
  - composed of 2 types: Mobitz I or Wenckebach block, and Mobitz II.
Second-degree AV Block

- **Mobitz I second-degree AV block**
  - Characterized by a progressive prolongation of the PR interval, which results in a progressive shortening of the R-R interval.
  - Ultimately, the atrial impulse fails to conduct, a QRS complex is not generated, and there is no ventricular contraction.
Second-degree AV Block

- Mobitz II second-degree AV block
  - Characterized by an unexpected nonconducted atrial impulse.
  - Thus, the PR and R-R intervals between conducted beats are constant.
Second-degree AV Block

- **Pathophysiology:**
  - **Mobitz type I block**
    - Caused by conduction delay in the AV node in 72% of patients and by conduction delay in the His-Purkinje system in the remaining 28%.
  - **Mobitz type II block**
    - Conduction delay occurs infranodally. The QRS complex is likely to be wide, except in patients where the delay is localized to the bundle of His.
Second-degree AV Block

- **History:**
  - Mobitz I (Wenckebach) block
    - Most patients are asymptomatic.
    - Patients may experience light-headedness, dizziness, or syncope, but these symptoms are uncommon.
    - Patients may have chest pain if the heart block is related to myocarditis or ischemia.
    - Patients may have a history of structural heart disease.
  - Mobitz II block
    - Unlike Mobitz I block, patients with type II block are more likely to experience light-headedness, dizziness, or syncope, although they may be asymptomatic as well.
    - Patients may have chest pain if the heart block is related to myocarditis or ischemia.
Second-degree AV Block

- **Physical:**
  - Patients often have a regularly irregular heartbeat.
  - Bradycardia may be present.
  - Symptomatic patients may have signs of hypoperfusion, including hypotension.
Second-degree AV Block

- **Causes:**
  - **Mobitz I block**
    - individuals with high vagal tone, such as athletes or young children.
    - infants and young children with structural heart disease (eg, tetralogy of Fallot)
    - individuals of any age following valvular surgery (especially mitral valve).
    - myocardial infarction (especially inferior wall)
    - drug induced (including beta-blockers, calcium channel blockers, amiodarone, digoxin, and possibly pentamidine).
  - **Mobitz II block**
    - acute myocardial infarction (anterior or inferior).
    - Drug-induced etiologies can also occur.
Second-degree AV Block

- **Lab Studies:**
  - Serum electrolytes, calcium, and magnesium
  - A digoxin level should be obtained for patients on digoxin.
  - Cardiac enzymes tests are indicated for any patient with suspected myocardial ischemia.
  - Myocarditis-related laboratory studies (eg, Lyme titers, HIV serologies, enterovirus polymerase chain reaction [PCR], adenovirus PCR, Chagas titers), if clinically relevant.

- **Imaging Studies:**
  - Routine imaging studies are not required.
  - Follow-up ECGs and cardiac monitoring are appropriate.
Third-degree AV Block

- Disorder of the cardiac conduction system where there is no conduction through the AV node.
- Complete disassociation of the atrial and ventricular activity exists.
- Ventricular escape mechanism can occur anywhere from the AV node to the bundle-branch Purkinje system.
- not all patients with AV dissociation have complete heart block.
  - For example, patients with accelerated junctional rhythms have AV dissociation, but not complete heart block, if the escape rate is faster than the intrinsic sinus rate.
- ECG - complete heart block is represented by QRS complexes being conducted at their own rate and totally independent of the P waves.
Third-degree Block

- **Mortality/Morbidity:**
  - Frequently hemodynamically unstable
  - The patient may experience syncope, cardiovascular collapse, or death.

- **History:**
  - Patients occasionally are asymptomatic or have only minimal symptoms related to hypoperfusion. In these situations, symptoms include:
    - Fatigue
    - Dizziness
    - Impaired exercise tolerance
    - Chest pain
Third-degree Block

- Patients who concurrently experience an MI can have associated symptoms from the MI, including chest pain, dyspnea, nausea or vomiting, and diaphoresis.
- Patients who have a history of cardiac disease may be on medications that affect the conduction system through the AV node, including the following:
  - Beta-blockers
  - Calcium channel blockers
  - Digitalis cardioglycosides
Third-degree Block

- **Physical:**
  - Bradycardia.
  - Signs of congestive heart failure as a result of decreased cardiac output may be present and include the following:
    - Tachypnea or respiratory distress
    - Rales
    - Jugular venous distention
  - Signs of hypoperfusion, including:
    - Altered mental status
    - Hypotension
    - Lethargy
  - In patients with concomitant myocardial ischemia or infarction, corresponding signs may be evident:
    - Signs of anxiety such as agitation or unease
    - Diaphoresis
    - Pale or pasty complexion
    - Tachypnea
Third-degree AV Block

- **Causes:**
  - Can be either congenital or acquired.

- **Congenital form**
  - Usually occurs at the level of the AV node
  - Patients are relatively asymptomatic at rest but later develop symptoms because the fixed heart rate is not able to adjust for exertion.
Acquired complete heart block

- Can develop from isolated, single-agent overdose, or often from combined or iatrogenic coadministration of AV-nodal, beta-adrenergic, and calcium channel blocking agents.
- Drugs or toxins associated with heart block include the following:
  - Class Ia antiarrhythmics (eg, quinidine, procainamide, disopyramide)
  - Class Ic antiarrhythmics (eg, flecainide, encainide, propafenone)
  - Class II antiarrhythmics (beta-blockers)
  - Class III antiarrhythmics (eg, amiodarone, sotalol, dofetilide, ibutilide)
  - Class IV antiarrhythmics (calcium channel blockers)
  - Digoxin or other cardiac glycosides
Third-degree AV Block

- **Acquired complete heart block**
  - Infectious causes include the following:
    - Cardiomyopathy, eg, Lyme carditis and acute rheumatic fever
    - Metabolic disturbances, eg, severe hyperkalemia
    - Ischemia
    - MI - Anterior wall MI can be associated with an infranodal AV block.
    - Complete heart block develops in slightly less than 10% of cases of acute inferior MI and often resolves within hours to a few days.
Third-degree AV Block

- **Treatment**
  - For all symptomatic high degree heart block
    - ACLS as indicated
    - Identification of etiology based on clinical presentation
    - Transcutaneous pacing for unstable patients
    - Permanent pacemaker when indicated
# Indications for Pacing for AV Block

<table>
<thead>
<tr>
<th>Degree</th>
<th>Pacemaker necessary</th>
<th>Pacemaker probably necessary</th>
<th>Pacemaker not necessary</th>
</tr>
</thead>
</table>
| **Third** | Symptomatic congenital complete heart block  
Aquired symptomatic complete heart block  
Atrial fibrillation with complete heart block  
Acquired asymptomatic complete heart block | | |
| **Second** | Symptomatic type I  
Symptomatic type II | Asymptomatic type II  
Asymptomatic type I at intra-His or infra-His levels | Asymptomatic type I at supra-His (AV nodal) block |
| **First** | | | Asymptomatic or symptomatic |
## Indications for Pacing for Sinus Node Dysfunction

<table>
<thead>
<tr>
<th>Pacemaker</th>
<th>Pacemaker probably necessary</th>
<th>Pacemaker not necessary</th>
</tr>
</thead>
<tbody>
<tr>
<td>Symptomatic bradycardia</td>
<td>Symptomatic patients with sinus node dysfunction with documented rates of &lt;40 bpm without a clear-cut association between significant symptoms and the bradycardia</td>
<td>Asymptomatic sinus node dysfunction</td>
</tr>
<tr>
<td>Symptomatic sinus bradycardia due to long-term drug therapy of a type and dose for which there is no accepted alternative</td>
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</tbody>
</table>
Bradycardia

**Primary ABCD Survey**
- Assess ABCs
- Secure airway noninvasively
- Ensure monitor/defibrillator is available

**Secondary ABCD Survey**
- Assess secondary ABCs (invasive airway management needed?)
- Oxygen–IV access–monitor–fluids
- Vital signs, pulse oximeter, monitor BP
- Obtain and review 12-lead ECG
- Obtain and review portable chest x-ray
- Problem-focused history
- Problem-focused physical examination
- Consider causes (differential diagnoses)
Serious signs or symptoms? Due to bradycardia?

- No
  - Type II second-degree AV block or Third-degree AV block?
    - No
      - Observe
    - Yes
      - Intervention sequence
        - Atropine 0.5 to 1.0 mg
        - Transcutaneous pacing if available
        - Dopamine 5 to 20 µg/kg per minute
        - Epinephrine 2 to 10 µg/min
        - Isoproterenol 2 to 10 µg/min

- Yes
  - Prepare for transvenous pacer
  - If symptoms develop, use transcutaneous pacemaker until transvenous pacer placed
Atropine Sulfate

- Indications *(When & Why?)*
  - First drug for symptomatic bradycardia
    - Increases heart rate by blocking the parasympathetic nervous system

*Bradycardias*
Atropine Sulfate

- Dosing *(How?)*
  - 0.5 to 1.0 mg IV every 3 to 5 minutes as needed
  - May give via ET tube (2 to 2.5 mg) *diluted in 10 mL* of NS
  - Maximum Dose: 0.04 mg/kg

*Bradycardias*
Dopamine

- **Indications** *(When & Why?)*
  - Second drug for symptomatic bradycardia (after atropine)
  - Use for hypotension (systolic BP 70 to 100 mm Hg) with S/S of shock

*Bradycardias*
Dopamine

- **Dosing** *(How?)*
  - IV Infusions (Titrate to Effect)
    - 400 mg / 250 mL of D5W = 1600 mcg/mL
    - 800 mg/ 250 mL of D5W = 3200 mcg/mL

_Bradycardias_
Dopamine

- **Dosing (How?)**
  - IV Infusions (Titrate to Effect)
    - Low Dose **“Renal Dose”**
      - 1 to 5 µg/kg per minute
    - Moderate Dose **“Cardiac Dose”**
      - 5 to 10 µg/kg per minute
    - High Dose **“Vasopressor Dose”**
      - 10 to 20 µg/kg per minute

*Bradycardias*
Epinephrine

- **Indications** *(When & Why?)*
  - Symptomatic bradycardia: After atropine, dopamine, and transcutaneous pacing (Class IIb)
Epinephrine

- **Dosing** *(How?)*
  - Profound Bradycardia
    - 2 to 10 µg/min infusion (add 1 mg of 1:1000 to 500 mL normal saline; infuse at 1 to 5 mL/min)

Bradycardias
Isoproterenol

- **Indications** *(When & Why?)*
  - Temporary control of bradycardia in heart transplant patients
  - Class IIb at low doses for symptomatic bradycardia
  - *Heart Transplant Patients!*

*Bradycardias*
Isoproterenol

- **Dosing (How?)**
  - Infuse at 2 to 10 µg/min
  - Titrate to adequate heart rate
# Tachyarrhythmias

Ectopic rate nomenclature:

<table>
<thead>
<tr>
<th>Rate Range</th>
<th>Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>[150-250]</td>
<td>Paroxysmal tachycardia</td>
</tr>
<tr>
<td>[250-350]</td>
<td>Flutter</td>
</tr>
<tr>
<td>[350+]</td>
<td>Fibrillation</td>
</tr>
</tbody>
</table>
Tachyarrhythmias

- **Frequency:**
  - AVNRT occurs in 60% of patients (with a female predominance) presenting with paroxysmal SVT.
  - The prevalence of SVT in the general population is likely several cases per thousand persons.

- **Mortality/Morbidity:**
  - AVNRT is usually well tolerated; it often occurs in patients with no structural heart disease.
  - In patients with coronary artery disease, AVNRT may cause angina or myocardial infarction.
  - Prognosis for patients without heart disease is usually good.

- **Age:** AVNRT may occur in persons of any age. It is common in young adults.
AVNRT is characterized by an abrupt onset and termination of episodes. Episodes may last from seconds to minutes to days. In the absence of structural heart disease, it is usually well tolerated. Common symptoms include palpitations, nervousness, anxiety, lightheadedness, neck and chest discomfort, and dyspnea. Polyuria can occur after termination of the episode (due to the release of atrial natriuretic factor).
Tachyarrhythmias

- History:
  - AVNRT may cause or worsen heart failure in patients with poor left ventricular function.
  - It may cause angina or myocardial infarction in patients with coronary artery disease.
  - Syncope may occur in patients with a rapid ventricular rate or prolonged tachycardia due to poor ventricular filling, decreased cardiac output, hypotension, and reduced cerebral circulation. Syncope may also occur because of transient asystole when the tachycardia terminates, owing to tachycardia-induced depression of the sinus node.
Tachyarrhythmias

- Physical:
  - The heart rate - ranging from 150-250 beats per minute (bpm).
    - It is usually 180-200 bpm in adults and, in children, may exceed 250 bpm.
  - Hypotension may occur initially or with rapid ventricular rates and prolonged episodes.
  - Signs of left heart failure may develop or worsen in patients with poor left ventricular function.
Mechanisms of Arrhythmia

- Abnormal automaticity
  - automatic impulse generation from unusual site or overtakes sinus node
- Triggered activity
  - secondary depolarization during or after repolarization
  - Dig toxicity, Torsades de Pointes
- Reentry
  - 90% of arrhythmias
Regular SVT in adults

- 90% reentrant 10 % not reentrant
- 60% AV nodal reentrant tachycardia (AVNRT)
  - The most common type of reentrant supraventricular tachycardia (SVT).
  - Because of the abrupt onset and termination of the reentrant SVT, the nonspecific term paroxysmal SVT has been used to describe these tachyarrhythmias
- 30% orthodromic reciprocating tachycardia (ORT)
- 10% Atrial tachycardia
- 2 to 5% involve WPW syndrome
Supraventricular Tachycardias

**Diagnosis**

- ECG is cornerstone
- Observe zones of transition for clues as to mechanism:
  - onset
  - termination
  - slowing, AV nodal block
  - bundle branch block
Tachyarrhythmias

- Imaging Studies:
  - Echocardiogram - To evaluate for the presence of structural heart disease
  - Electrophysiology study - To induce and map the reentrant circuit.
    - performed if ablation of the reentrant circuit is planned
## ECG Distinction of VT from SVT with Aberrancy

<table>
<thead>
<tr>
<th></th>
<th>Favors VT</th>
<th>Favors SVT</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Duration</strong></td>
<td>RBBB: QRS &gt; 0.14 sec.</td>
<td>&lt; 0.14 sec.</td>
</tr>
<tr>
<td></td>
<td>LBBB: QRS &gt; 0.16 sec.</td>
<td>&lt; 0.16 sec.</td>
</tr>
<tr>
<td><strong>Axis</strong></td>
<td>QRS axis -90° to ±180°</td>
<td>Normal</td>
</tr>
</tbody>
</table>
ECG Distinction of VT from SVT with Aberrancy

Favors VT  Favors SVT

with Aberrancy

Morphology  Precordial concordance

If LBBB:  

\( V_1 \) duration > 30 ms
S wave > 70 ms
S wave notched or slurred
\( V_6 \): qR or QR
R wave

If RBBB:  

\( V_1 \): monophasic R wave
qR
If triphasic, \( R > R^1 \)
\( R < R^1 \)
\( V_6 \): R < S
Differential Dx of Regular SVT

- Short RP tachycardia
  - AV nodal reentrant tachycardia
  - ORT (Orthodromic reciprocating tachycardia)
  - atrial tachycardia when associated with slow AV nodal conduction
AV Nodal Reentrant Tachycardia

- Responds to vagal maneuvers in 1/3 cases
- Very responsive to AV nodal blocking agents such as beta blockers, Ca-channel blockers, adenosine.
- Recurrences are the norm on medical therapy
- Catheter ablation 95% successful with 1% major complication rate
Medical Care:

- Rest, reassurance, and sedation may terminate the attack.
- Vagal maneuvers (e.g., carotid sinus massage, exposure of the face to ice water, Valsalva maneuver) before initiating drug treatment.
- These maneuvers could also be tried after each pharmacological approach.
- Vagal maneuvers are unlikely to work and should not be tried if hypotension is present. Sometimes, putting the patient in the Trendelenburg position facilitates termination with a vagal maneuver.
- ACLS algorithm if no response to these measures.
Tachyarrhythmias

- **Preventive therapy**
  - Needed for frequent, prolonged, or highly symptomatic episodes that do not terminate spontaneously or those that cannot be easily terminated by the patient.

- **Drugs**
  - Include long-acting beta-blockers, calcium channel blockers, and digitalis.

- **Radiofrequency catheter ablation**
  - Should be considered in patients with frequent symptomatic episodes who do not want drug therapy, who cannot tolerate the drugs, or in whom drug therapy fails.
Atrial Tachycardia

- Atrial rate between 150 and 250 bpm
- Does not require AV nodal or infranodal conduction
- P wave morphology different than sinus
- P-R interval ≥ 120 msec differentiating from junctional tachycardia
- Origin inferred from P wave morphology.
Atrial tachycardia

- P wave upright lead V1 and negative in aVL consistent with left atrial focus.
- P wave negative in V1 and upright in aVL consistent with right atrial focus.
- Adenosine may help with diagnosis if AV block occurs and continued arrhythmia likely atrial tachycardia
- 70-80% will also terminate with adenosine.
Atrial Tachycardia

- Most are due to abnormal automaticity and have right atrial focus.
- May be reentry particularly in patients with previous atriotomy scar, such as CABG or congenital repair patients.
Atrial Tachycardia Therapy

- Frequently treated with antiarrhythmics
  - **Class 1 agents** - procainamide, quinidine, flecainide may be used in patients without structural heart disease.
  - **Class III agents** - sotalol, amiodarone, dofetilide may be used with caution according to specific side effects
- AV Nodal blocking agents for rate control.
- Catheter ablation effective in 70-80%
Sinus Tachycardia

- Sinus node is still the pacemaker, but the rate is accelerated for some physiologic reason:
  - Rhythm is regular
  - Rate > 100 beats/minute
  - P wave, PR interval, and QRS complex are all normal
- Can look like Sinus Node Reentry – paroxysmal and less than 160 BPM; incidence of ~10% of all PSVT’s
Sinus Tachycardia

- **Treatment:**
  - Alleviate the underlying cause - anemia, pheo, hyperthyroid…
  - Could be inappropriate ST - a type of autonomic dysfunction with HR consistently above 120
Adult tachycardia algorithm (with pulse)

**Synchronised DC Shock**
- Up to 3 attempts
- Amiodarone 300 mg IV over 10-20 min and repeat shock; followed by:
  - Amiodarone 900 mg over 24 h

**Is patient stable?**
- Signs of instability include:
  1. Reduced conscious level
  2. Chest pain
  3. Systolic BP < 90 mmHg
  4. Heart failure
- Rate-related symptoms uncommon at less than 150 beats min⁻¹

**Is QRS narrow (< 0.12 sec)?**

**Wide QRS**
- Is QRS regular?
  - Irregular: Seek expert help
    - Possibilities include:
      - AF with bundle branch block: treat as for narrow complex
      - Pre-excited AF: consider amiodarone
      - Polymorphic VT (e.g. torsade de pointes - give magnesium 2 g over 10 min)

  - Regular: If Ventricular Tachycardia (or with bundle branch block):
    - Amiodarone 300 mg IV over 20-60 min; then 900 mg over 24 h
    - If previously confirmed SVT with bundle branch block:
      - Give adenosine as for regular narrow complex tachycardia

**Narrow QRS**
- Is rhythm regular?
  - Regular: Use vagal manoeuvres
    - Adenosine 6 mg rapid IV bolus; if unsuccessful give 12 mg; if unsuccessful give further 12 mg.
    - Monitor ECG continuously
  - Irregular: Probable atrial fibrillation
    - Probable re-entry PSVT:
      - Record 12-lead ECG in sinus rhythm
      - If recurs, give adenosine again & consider choice of anti-arrhythmic prophylaxis
  - Normal sinus rhythm restored?
    - Yes: Seek expert help
    - No: Possible atrial flutter
      - Control rate (e.g. β-Blocker)
Atrial flutter

Relatively common atrial tachyarrhythmia.

- Has traditionally been characterized as a macroreentrant arrhythmia with atrial rates between 240-400 beats per minute.
- Defined by the presence of stable, uniform atrial activation (flutter waves).
- Can impede cardiac output and lead to atrial thrombus formation, with risk of systemic embolization.
- Commonly includes some form of A-V block.
  - Most commonly atrial depolarization is conducted at a 2:1 ratio, though it can also be conducted at a 4:1 ratio, and less commonly at a 3:1 or 5:1 ratio.
Atrial flutter

- **Pathophysiology:**
  - **Type I is the most common form**
    - Also referred to as typical, common, or counter-clockwise isthmus-dependent atrial flutter and involves a re-entrant circuit that encircles the tricuspid annulus of the right atrium.
    - Traditionally been distinguished by a rate of 240-340 beats, and the ability to be entrained by atrial pacing
  - **Type II atrial flutter**
    - Also known as atypical aflutter, is still poorly characterized, but may result from an intraatrial reentrant circuit operating at a faster rate.
    - Type II has a rate greater than 340 beats.
  - Atrial flutter is associated in patients with heart failure, valvular disease, COPD, hyperthyroidism, pericarditis, pulmonary embolism, and a history of open heart surgery.
Atrial flutter

- **Mortality/Morbidity:**
  - Due to complications of rate (ie, syncope, congestive heart failure [CHF]). The risk of embolic occurrences approaches that of atrial fibrillation.

- **Sex:**
  - Men are affected more often than women, with a 2:1 male-to-female ratio.

- **Age:**
  - The prevalence of atrial flutter increases with age and varies from 1 case out of 200 persons for people younger than 60 years, to almost 9 cases out of 100 persons for people over 80 years.
Atrial flutter

- **History:**
  - Symptomatic atrial flutter is typically a manifestation of the rapid ventricular rate that decreases cardiac output.
    - Palpitations
    - Fatigue or poor exercise tolerance
    - Mild dyspnea
    - Presyncope
    - Less common symptoms include angina, profound dyspnea, or syncope.
    - Symptomatic embolic events are rare, but must be considered.
Atrial flutter

Physical:

- Tachycardia may or may not be present, depending on the degree of AV block associated with the atrial flutter activity.
  - Cardiac rate, often approximately 150 beats per minute because of a 2:1 AV block
- Regular or slightly irregular heartbeat
- Hypotension is possible, but normal blood pressure is observed more commonly.
- Peripheral embolization may occur, if associated with AF.
- CHF may be found, usually caused by LV dysfunction.
Atrial flutter

- Causes:
  - Patients at highest risk include those with long-standing hypertension, valvular heart disease (rheumatic), left ventricular hypertrophy, coronary artery disease with or without depressed left ventricular function, pericarditis, pulmonary embolism, hyperthyroidism, and diabetes.
  - Additionally, CHF for any reason is a noted contributor to this disorder.
  - Additional causes include the following:
    - Postoperative revascularization
    - Digitalis toxicity
    - Rare causes
      - Myotonic dystrophy in childhood (case report by Suda K, Matsumura M, Hayashi Y)
Atrial flutter

- Imaging Studies:
  - Chest radiographic findings are usually normal.
  - Look for radiographic evidence of pulmonary edema in subacute cases.
  - thyroid function studies.
  - serum electrolyte and digoxin levels if appropriate.
  - CBCs if anemia is suspected or a history of recent or current blood loss is associated with presenting symptoms.
  - blood gases in patients with hypoxia, or carbon monoxide intoxication.
  - Seek a history of stimulant drug usage (eg, ginseng, cocaine, ephedra, methamphetamine).
Atrial flutter

- **Electrocardiography (ECG)**
- **Transthoracic echocardiogram**
  - Can be performed to evaluate right and left atrial size, as well as the size and function of the right and left ventricles, which assists in diagnosing valvular heart disease, LVH, and pericardial disease.
  - Has low sensitivity for intra-atrial thrombi, and is the preferred modality for testing atrial flutter.
- **Exercise Testing**
  - Can be utilized to identify exercise-induced atrial fibrillation and to evaluate ischemic heart disease.
- **Holter Monitoring**
  - Can be used to help identify arrhythmias in patients with non-specific symptoms, identify triggers, and detect associated atrial arrhythmias.
Atrial flutter

- **Treatment:**
  - Hemodynamically unstable – appropriate ACLS
    - **Synchronous direct-current (DC) cardioversion is commonly the initial treatment of choice.**
      - Cardioversion often requires low energies (<50 J).
    - If the electrical shock results in AF, a second shock at a higher energy level is used to restore normal sinus rhythm (NSR).
Atrial flutter

- Treatment:
  - Hemodynamically Stable
    - Slowing the ventricular response with verapamil or diltiazem may be the appropriate initial treatment.
    - Adenosine produces transient AV block and can be used to reveal flutter waves.
    - These drugs generally do not convert atrial flutter to NSR.
Atrial flutter

Treatment

- If the flutter cannot be cardioverted, terminated by pacing, or slowed by the drugs mentioned above, **digoxin** can be administered alone or with either a **calcium antagonist or beta-blocker**.

- **IV amiodarone** has been shown to slow the ventricular rate and is considered as effective as digoxin.
Atrial flutter

Treatment

- Rate control is the goal of medication in atrial flutter or AF.
- Beta-adrenergic blockers are effective in the presence of thyrotoxicosis and increased sympathetic tone.
- Antiarrhythmic drugs alone control atrial flutter in only 50-60% of patients.
- Radiofrequency catheter ablation has been used to interrupt the re-entrant circuit in the right atrium and prevent recurrences of atrial flutter.
  - is immediately successful in more than 90% of cases and avoids the long-term toxicity observed with antiarrhythmic drugs.
  - "electrical cardioversion is the preferred modality in the patient whose condition is unstable."
Atrial Fibrillation

- Most commonly encountered arrhythmia in clinical practice.
- Defined by the absence of coordinated atrial systole.
- Results from multiple reentrant electrical wavelets that move randomly around the atria.
- P waves are replaced by irregular, chaotic fibrillatory waves, often with a concomitant irregular ventricular response.
Atrial Fibrillation

- When ventricular rate increases to tachycardic levels, a situation of atrial fibrillation with rapid ventricular response (AF with RVR) ensues.
- The incidence of atrial fibrillation increases significantly with advancing age.
- AF may increase mortality up to 2-fold, primarily due to embolic stroke.
Atrial Fibrillation

- Occurs in 3 distinct clinical circumstances:
  - As a primary arrhythmia in the absence of identifiable structural heart disease
  - As a secondary arrhythmia in the presence of a systemic abnormality that predisposes the individual to the arrhythmia
  - As a secondary arrhythmia associated with cardiac disease that affects the atria
Conditions predisposing to, or encouraging progression of AF

- Hypertension
- Symptomatic heart failure (NYHA II - IV) including tachycardiomyopathy
- Valvular heart disease
- Cardiomyopathies including primary electrical cardiac disease
- Atrial septal defect and other congenital heart defects
- Coronary artery disease
- Thyroid dysfunction and possibly subclinical thyroid dysfunction
- Obesity
- Diabetes mellitus
- Chronic obstructive pulmonary disease (COPD) and sleep apnoea
- Chronic renal disease
Types of Atrial Fibrillation

First diagnosed episode of atrial fibrillation

- Paroxysmal (usually ≤ 48 h)
- Persistent (requires CV)
- Long-standing Persistent (> 1 year)
- Permanent (accepted)
Atrial Fibrillation

The 3 primary ways AF affects hemodynamic function include the following:

- Loss of atrial kick (synchronized atrial mechanical activity)
- Irregularity of ventricular response
- Inappropriately rapid heart rate
Atrial Fibrillation

- **History:**
  - Palpitations
  - Fatigue or poor exercise tolerance
  - Dyspnea
  - Chest pain (true angina)
  - Presyncope or syncope
  - Generalized weakness
Atrial Fibrillation

- Noncardiovascular causes of atrial fibrillation:
  - Hyperthyroidism
  - Low levels of potassium, magnesium, or calcium
  - Pheochromocytoma
  - Sympathomimetic drugs, alcohol, electrocution

- Respiratory causes include:
  - Pulmonary embolism
  - Pneumonia
  - Lung cancer
  - Idiopathic: Lone AF is idiopathic and defined as the absence of any known etiologic factors plus normal ventricular function by echocardiography. Most patients with lone AF are younger than 65 years, although age is not used to define lone AF.
  - Hypothermia
Natural time course of AF

'Upstream' therapy of concomitant conditions

Anticoagulation

Rate control

Antiarrhythmic drugs

Ablation

cardioversion

first documented

silent
paroxysmal
persistent
long-standing persistent
permanent

AF = atrial fibrillation
Management of Atrial Fibrillation

- Aimed at symptom relief by rate and rhythm control
- Aimed at reducing risk of thromboembolism by anticoagulation
- Preventing tachycardia mediated cardiomyopathy (a progressive, reversible rate-induced form of LV dysfunction)
The management cascade for patients with AF

Atrial fibrillation → Record 12-lead ECG

- Presentation
- EHRA score
- Associated disease
- Initial assessment

Anticoagulation issues → Assess TE Risk

- Oral anticoagulant
- Aspirin
- None

Rate and rhythm control → AF type Symptoms

- Rate control
- ± Rhythm control
- Antiarrhythmic drugs
- Ablation

Treatment of underlying disease ‘Upstream’ therapy → Consider referral

- ACEIs/ARBs
- Statins/PUFAs
- Others

ACEI = angiotensin-converting enzyme inhibitor; AF = atrial fibrillation; ARB = angiotensin receptor blocker; PUFA = polyunsaturated fatty acid; TE = thrombo-embolism.
### Risk factors for stroke and thrombo-embolism in non-valvular AF

<table>
<thead>
<tr>
<th>Major risk factors</th>
<th>Clinically relevant non-major risk factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Previous stroke</td>
<td>CHF or moderate to severe LV systolic dysfunction [e.g. LV EF ≤ 40%]</td>
</tr>
<tr>
<td>TIA or systemic embolism</td>
<td>Hypertension</td>
</tr>
<tr>
<td>Age ≥ 75 years</td>
<td>Diabetes mellitus</td>
</tr>
<tr>
<td></td>
<td>Age 65-74 years</td>
</tr>
<tr>
<td></td>
<td>Female sex</td>
</tr>
<tr>
<td></td>
<td>Vascular disease</td>
</tr>
</tbody>
</table>

AF = atrial fibrillation; EF = ejection fraction (as documented by echocardiography, radionuclide ventriculography, cardiac catheterization, cardiac magnetic resonance imaging, etc.); LV = left ventricular; TIA = transient ischaemic attack.
## Risk factor-based point-based scoring system - CHA$_2$DS$_2$-VASc

<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 $\geq$ 75 ans</td>
<td>2</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>1</td>
</tr>
<tr>
<td>Stroke/TIA/thrombo-embolism</td>
<td>2</td>
</tr>
<tr>
<td>Vascular disease*</td>
<td>1</td>
</tr>
<tr>
<td>Age 65-74</td>
<td>1</td>
</tr>
<tr>
<td>Sex category [i.e. femal sex]</td>
<td>1</td>
</tr>
<tr>
<td><strong>Maximum score</strong></td>
<td><strong>9</strong></td>
</tr>
</tbody>
</table>

*Prior myocardial infarction, peripheral artery disease, aortic plaque. Actual rates of stroke in contemporary cohorts may vary from these estimates.*
Use of oral anticoagulation for stroke prevention in AF

CHADS₂ score ≥ 2†

No

Yes

Consider other risk factors* No

Age ≥ 75 years

< 2 others risk factors*

No

OAC

Yes

≥ 2 others risk factors*

No

1 other risk factor*

No

OAC (or aspirin)

Yes

Nothing (or aspirin)

*Congestive heart failure, hypertension. Age 75 years. Diabetes. Stroke/TIA/thromboembolism (doubled)

*Other clinically relevant non-major risk factors: age 65-74, female sex, vascular disease.

AF = atrial fibrillation; OAC = oral anticoagulant; TIA = transient ischaemic attack.
## Approach to thromboprophylaxis in AF

<table>
<thead>
<tr>
<th>Risk category</th>
<th>CHA$_2$DS$_2$-VASc score</th>
<th>Recommended antithrombotic therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>One ‘major’ risk factor or ≥ 2 ‘clinically relevant non-major’ risk factors</td>
<td>≥ 2</td>
<td>OAC</td>
</tr>
<tr>
<td>One ‘clinically relevant non-major’ risk factor</td>
<td>1</td>
<td>Either OAC or aspirin 75-325 mg daily. Preferred: OAC rather than aspirin.</td>
</tr>
<tr>
<td>No risk factors</td>
<td>0</td>
<td>Either aspirin 75-325 mg daily or no antithrombotic therapy. Preferred: no antithrombotic therapy rather than aspirin.</td>
</tr>
</tbody>
</table>

AF = atrial fibrillation; CHA$_2$DS$_2$-VASc = cardiac failure, hypertension, age ≥ 75 (doubled), diabetes, stroke (doubled)-vascular disease, age 65–74 and sex category (female); INR = international normalized ratio; OAC = oral anticoagulation, such as a vitamin K antagonist (VKA) adjusted to an intensity range of INR 2.0–3.0 (target 2.5).
# The HAS-BLED bleeding risk score

<table>
<thead>
<tr>
<th>Letter</th>
<th>Clinical characteristic*</th>
<th>Points awarded</th>
</tr>
</thead>
<tbody>
<tr>
<td>H</td>
<td>Hypertension</td>
<td>1</td>
</tr>
<tr>
<td>A</td>
<td>Abnormal renal and liver function (1 point each)</td>
<td>1 or 2</td>
</tr>
<tr>
<td>S</td>
<td>Stroke</td>
<td>1</td>
</tr>
<tr>
<td>B</td>
<td>Bleeding</td>
<td>1</td>
</tr>
<tr>
<td>L</td>
<td>Labile INRs</td>
<td>1</td>
</tr>
<tr>
<td>E</td>
<td>Elderly (e.g. age &gt; 65 years)</td>
<td>1</td>
</tr>
<tr>
<td>D</td>
<td>Drugs or alcohol (1 point each)</td>
<td>1 or 2</td>
</tr>
</tbody>
</table>

*Maximum 9 points*

*Hypertension is defined as systolic blood pressure > 160 mmHg. INR = international normalized ratio.*
Cardioversion, TOE and anticoagulation

AF = atrial fibrillation; DCC = direct current cardioversion; LA = left atrium; LAA = left atrial appendage; OAC = oral anticoagulant; SR = sinus rhythm; TOE = transoesophageal echocardiography.

*Anticoagulation should normally be continued for 4 weeks after a cardioversion attempt except when AF is recent onset and no risk factors are present.
†Long-term OAC if stroke risk factors and/or risk of AF recurrence/presence of thrombus.
## Drugs and doses for pharmacological conversion of (recent-onset) AF

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose</th>
<th>Follow-up dose</th>
<th>Risks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amiodarone</td>
<td>5 mg/kg i.v. over 1 h</td>
<td>50 mg/h</td>
<td>Phlebitis, hypotention. Will slow the ventricular rate. Delayed AF conversion to sinus rhythm.</td>
</tr>
<tr>
<td>Flecainide</td>
<td>2 mg/kg i.v. over 10 min, or 200-300 mg p.o.</td>
<td>N/A</td>
<td>Not suitable for patients with market structural heart disease; may prolong QRS duration, and hence the QT interval; and may inadvertently increase the ventricular rate due to conversion to atrial flutter and 1:1 conduction to the ventricles.</td>
</tr>
<tr>
<td>Ibutilide</td>
<td>1 mg i.v. over 10 min</td>
<td>1 mg i.v. over 10 min after waiting for 10 min</td>
<td>Can cause prolongation of the QT interval and torsades de pointes; watch for abnormal T-U waves or QT prolongation. Will slow the ventricular rate.</td>
</tr>
<tr>
<td>Propafenone</td>
<td>2 mg/kg i.v. over 10 min, or 450-600 mg p.o.</td>
<td></td>
<td>Not suitable for patients with market structural heart disease; may prolong QRS duration; will slightly slow the ventricular rate, but may inadvertently increase the ventricular rate due to conversion to atrial flutter and 1:1 conduction to the ventricles.</td>
</tr>
<tr>
<td>Vernakalant</td>
<td>3 mg/kg i.v. over 10 min</td>
<td>Second infusion of 2 mg/kg i.v. over 10 min after 15 min rest</td>
<td>So far only evaluated in clinical trials; recently approved.</td>
</tr>
</tbody>
</table>

ACS = acute coronary syndrome; AF = atrial fibrillation; DCC = direct current cardioversion; i.v. = intravenous; N/A = not applicable; NYHA, New York Heart Association; p.o. = per os; QRS = QRS duration; QT = QT interval; T-U = abnormal repolarization (T-U) waves.
DCC and pharmacological conversion
recent-onset AF

Recent-onset AF (< 48 h)

- Haemodynamic instability
  - Yes: Electrical cardioversion
  - No: Structural heart disease
    - Yes: i.v. amiodarone
    - No: i.v. flecainide or i.v. propafenone or i.v. ibutilide

AF = atrial fibrillation; i.v. = intravenous.
Optimal level of heart rate control

Rate control

- No or tolerable symptoms
  - Accept lenient rate control

- Symptoms
  - More strict rate control
    - Exercise test if excessive heart rate is anticipated during exercise
    - 24 h ECG for safety
The choice of drugs depends on life style and underlying disease.
### Drugs for rate control

<table>
<thead>
<tr>
<th></th>
<th>Intravenous administration</th>
<th>Usual oral maintenance dose</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>β-Blockers</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Metoprolol CR/XL</td>
<td>2.5–5 mg iv bolus over 2 min; up to 3 doses</td>
<td>100–200 mg o.d. (ER)</td>
</tr>
<tr>
<td>Bisoprolol</td>
<td>N/A</td>
<td>2.5–10 mg o.d.</td>
</tr>
<tr>
<td>Atenolol</td>
<td>N/A</td>
<td>25–100 mg o.d.</td>
</tr>
<tr>
<td>Esmolol</td>
<td>50–200 µg/kg/min iv</td>
<td>N/A</td>
</tr>
<tr>
<td>Propranolol</td>
<td>0.15 mg/kg iv over 1 min</td>
<td>10–40 mg t.i.d.</td>
</tr>
<tr>
<td>Carvedilol</td>
<td>N/A</td>
<td>3.125–25 mg b.i.d.</td>
</tr>
<tr>
<td><strong>Non-dihydropyridine calcium channel antagonists</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Verapamil</td>
<td>0.0375–0.15 mg/kg iv over 2 min</td>
<td>40 mg b.i.d. to 360 mg (ER) o.d.</td>
</tr>
<tr>
<td>Diltiazem</td>
<td>N/A</td>
<td>60 mg t.i.d. to 360 mg (ER) o.d.</td>
</tr>
<tr>
<td><strong>Digitalis glycosides</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Digoxin</td>
<td>0.5–1 mg</td>
<td>0.125 mg–0.5 mg o.d.</td>
</tr>
<tr>
<td>Digitalis</td>
<td>0.4–0.6 mg</td>
<td>0.05 mg–0.1 mg o.d.</td>
</tr>
<tr>
<td><strong>Others</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Amiodarone</td>
<td>5 mg/kg in 1 h, and 50 mg/h maintenance</td>
<td>100 mg–200 mg o.d.</td>
</tr>
<tr>
<td>Dronedarone&lt;sup&gt;a&lt;/sup&gt;</td>
<td>N/A</td>
<td>400 mg b.i.d.</td>
</tr>
</tbody>
</table>

ER = extended release formulations; N/A = not applicable. ‡Only in patients with non-permanent atrial fibrillation.
## AV node ablation in AF patients

<table>
<thead>
<tr>
<th>Recommendations</th>
<th>Class&lt;sup&gt;a&lt;/sup&gt;</th>
<th>Level&lt;sup&gt;b&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ablation of the AV node to control heart rate should be considered when the rate cannot be controlled with pharmacological agents and when AF cannot be prevented by antiarrhythmic therapy or is associated with intolerable side-effects, and direct catheter-based or surgical ablation of AF is not indicated, has failed or is rejected.</td>
<td>IIa</td>
<td>B</td>
</tr>
<tr>
<td>Ablation of the AV node should be considered for patients with permanent AF and an indication for CRT (NYHA functional class III or ambulatory class IV symptoms despite optimal medical therapy, LVEF ≤ 35%, QRS width ≥ 130 ms).</td>
<td>IIa</td>
<td>B</td>
</tr>
<tr>
<td>Ablation of the AV node should be considered for CRT non-responders in whom AF prevents effective biventricular stimulation and amiodarone is ineffective or contraindicated.</td>
<td>IIa</td>
<td>C</td>
</tr>
</tbody>
</table>

<sup>a</sup>Class of recommendation.  
<sup>b</sup>Level of evidence.

AF = atrial fibrillation; AV = atrioventricular; CRT = cardiac resynchronization therapy; LV = left ventricular; LVEF = left ventricular ejection fraction; NYHA = New York Heart Association.
### AV node ablation in AF patients

<table>
<thead>
<tr>
<th>Recommendations</th>
<th>Class&lt;sup&gt;a&lt;/sup&gt;</th>
<th>Level&lt;sup&gt;b&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>In patients with any type of AF and severely depressed LV function (LVEF ≤ 35%) and severe heart failure symptoms (NYHA III or IV), biventricular stimulation should be considered after AV node ablation.</td>
<td>IIa</td>
<td>C</td>
</tr>
<tr>
<td>Ablation of the AV node to control heart rate may be considered when tachycardia-mediated cardiomyopathy is suspected and the rate cannot be controlled with pharmacological agents, and direct ablation of AF is not indicated, has failed or is rejected.</td>
<td>IIb</td>
<td>C</td>
</tr>
<tr>
<td>Ablation of the AV node with consecutive implantation of a CRT device may be considered in patients with permanent AF, LVEF ≤ 35% and NYHA functional class I or II symptoms on optimal medical therapy to control heart rate when pharmacological therapy is insufficient or associated with side-effects.</td>
<td>IIb</td>
<td>C</td>
</tr>
<tr>
<td>Catheter ablation of the AV node should not be attempted without a prior trial of medication, or catheter ablation for AF, to control the AF and/or ventricular rate in patients with AF.</td>
<td>III</td>
<td>C</td>
</tr>
</tbody>
</table>

<sup>a</sup>Class of recommendation.  
<sup>b</sup>Level of evidence.  
AF = atrial fibrillation; AV = atrioventricular; CRT = cardiac resynchronization therapy; LV = left ventricular; LVEF = left ventricular ejection fraction; NYHA = New York Heart Association.
<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose</th>
<th>Main contraindications and precautions</th>
<th>ECG features prompting lower dose or discontinuation</th>
<th>AV nodal slowing</th>
</tr>
</thead>
<tbody>
<tr>
<td>Disopyramide</td>
<td>100-250 mg t.i.d.</td>
<td>Contraindicated in systolic heart failure. Caution when using concomitant medication with QT-prolonging drugs.</td>
<td>QT interval &gt; 500 ms</td>
<td>None</td>
</tr>
<tr>
<td>Flecaïnide</td>
<td>100-200 mg b.i.d.</td>
<td>Contraindicated if creatinine clearance &lt; 50 mg/mL, in coronary artery disease, reduced LV ejection fraction.</td>
<td>QRS duration increase &gt; 25% above baseline</td>
<td>None</td>
</tr>
<tr>
<td>Flecaïnide XL</td>
<td>200 mg o.d.</td>
<td>Caution in the presence of conduction system disease.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Propafenone</td>
<td>150-300 mg t.i.d.</td>
<td>Contraindicated in coronary artery disease, reduced LV ejection fraction.</td>
<td>QRS duration increase &gt; 25% above baseline</td>
<td>Slight</td>
</tr>
<tr>
<td>Propafenone SR</td>
<td>225-425 mg b.i.d.</td>
<td>Caution in the presence of conduction system disease and renal impairment.</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

AF = atrial fibrillation; AV = atrioventricular; bpm = beats per minute; CYP = cytochrome P; ECG = electrocardiogram; LV = left ventricular; NYHA = New York Heart Association.
### Suggested doses and main caveats for commonly used antiarrhythmic drugs (Contd)

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose</th>
<th>Main contraindications and precautions</th>
<th>ECG features prompting lower dose or discontinuation</th>
<th>AV nodal slowing</th>
</tr>
</thead>
<tbody>
<tr>
<td>d,l-Sotalol</td>
<td>80-160 mg b.i.d.</td>
<td>Contraindicated in the presence of significant LV hypertrophy, systolic heart failure, pre-existing QT prolongation, hypokalaemia. Creatinine clearance &lt; 50 mg/mL. Moderate renal dysfunction requires careful adaptation of dose.</td>
<td>QT interval &gt; 500 ms</td>
<td>Similar to high-dose β-blockers</td>
</tr>
<tr>
<td>Amiodarone</td>
<td>600 mg o.d. for 4 weeks, 400 mg o.d. for 4 weeks then 200 mg o.d.</td>
<td>Caution when using concomitant medication with QT-prolonging drugs, heart failure. Dose of vitamin K antagonists and of digitoxin/digoxin should be reduced.</td>
<td>QT interval &gt;500 ms</td>
<td>10–12 bpm in AF</td>
</tr>
</tbody>
</table>

AF = atrial fibrillation; AV = atrioventricular; bpm = beats per minute; CYP = cytochrome P; ECG = electrocardiogram; LV = left ventricular; NYHA = New York Heart Association.
<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose</th>
<th>Main contraindications and precautions</th>
<th>ECG features prompting lowerdose or discontinuation</th>
<th>AV nodal slowing</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dronedarone</td>
<td>400 mg b.i.d.</td>
<td>Contraindicated in NYHA class III–IV or unstable heart failure, during concomitant medication with QT-prolonging drugs, powerful CYP 3A4 inhibitors, if creatinine clearance &lt; 30 mg/mL. Dose of digitoxin/digoxin should be reduced. Elevations in serum creatinine of 0.1–0.2 mg/dL are common and do not reflect reduced renal function.</td>
<td>QT interval &gt; 500 ms</td>
<td>10–12 bpm in AF</td>
</tr>
</tbody>
</table>

AF = atrial fibrillation; AV = atrioventricular; bpm = beats per minute; CYP = cytochrome P; ECG = electrocardiogram; LV = left ventricular; NYHA = New York Heart Association.
Choice of antiarrhythmic for the patient with no or minimal structural heart disease

No or minimal structural heart disease

Adrenergically mediated
- β-blockers
  - Sotalol
  - Dronedarone

Undetermined
- Dronedarone
- Flecainide
- Propafenone
- Sotalol

Vagally mediated
- Disopyramide

Amiodarone
Choice of antiarrhythmic drug according to underlying pathology

**Minimal or no heart disease**

- ? Prevention of remodeling
  - ACE/ARB/statin
  - β-blockade where appropriate
  
- Dronedarone / Flecainide / Propafenone / Sotalol
  - Amiodarone

**Significant underlying heart disease**

- Treatment of underlying condition and ? Prevention/reversal of remodelling - ACEI/ARB/statin. β-blockade where appropriate

- HT
  - No LVH
    - Dronedarone
    - Amiodarone
  - LVH
    - Dronedarone
    - Amiodarone
  - Dronedarone Sotalol
    - Amiodarone

- CAD
  - CHF
    - Stable NYHA I/II
    - NYHA III/IV or ‘unstable’ NYHA II

**Notes:**

ACEI = angiotensin-converting enzyme inhibitor; ARB = angiotensin receptor blocker; CAD = coronary artery disease; CHF = congestive heart failure; HT = hypertension; LVH = left ventricular hypertrophy; NYHA = New York Heart Association; unstable = cardiac decompensation within the prior 4 weeks. Antiarrhythmic agents are listed in alphabetical order within each treatment box. ? = evidence for ‘upstream’ therapy for prevention of atrial remodelling still remains controversial.
Choice between ablation and antiarrhythmic drug therapy for patients with and without structural heart disease

Relevant underlying heart disease

CHF

NYHA III/IV
or unstable NYHA II

NYHA III/IV
or unstable NYHA II

CAD

Stable NYHA III

Hypertension with LVH

Dronedarone

Sotalol

Dronedarone

Amiodarone

Catheter ablation for AF

No or minimal heart disease (including HT without LVH)

Paroxysmal AF

Persistent AF

Dronedarone

Flecainide

Propafenone

Sotalol

Catheter ablation for AF

Amiodarone

The restriction of the use of dronedarone has been recommended by the European Medicines Agency (EMA).

†More extensive LA ablation may be needed; *usually PVI is appropriate.
AF = atrial fibrillation; CAD = coronary artery disease; CHF = congestive heart failure; HT = hypertension; LVH = left ventricular hypertrophy; NYHA = New York Heart Association; PVI = pulmonary vein isolation. Antiarrhythmic agents are listed in alphabetical order within each treatment box.
# Surgical ablation of AF

<table>
<thead>
<tr>
<th>Recommendations</th>
<th>Class&lt;sup&gt;a&lt;/sup&gt;</th>
<th>Level&lt;sup&gt;b&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>Surgical ablation of AF should be considered in patients with symptomatic AF undergoing cardiac surgery.</td>
<td>IIa</td>
<td>A</td>
</tr>
<tr>
<td>Surgical ablation of AF may be performed in patients with asymptomatic AF undergoing cardiac surgery if feasible with minimal risk.</td>
<td>IIb</td>
<td>C</td>
</tr>
<tr>
<td>Minimally invasive surgical ablation of AF without concomitant cardiac surgery is feasible and may be performed in patients with symptomatic AF after failure of catheter ablation.</td>
<td>IIb</td>
<td>C</td>
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</tbody>
</table>

<sup>a</sup>Class of recommendation.

<sup>b</sup>Level of evidence.

AF = atrial fibrillation.
Nonpharmacologic Treatment of Atrial Fibrillation

- **Maze Procedure**
  - 90% freedom from atrial fibrillation
  - 2% mortality required thoracotomy

- **Catheter ablation procedure**
  - only moderate success
  - long procedures, difficult
  - selecting population
  - 60% to 80% effective
  - Pulmonary vein stenosis, cava perforation,
  - esophageal fistula
Nonpharmacologic Treatment of Atrial Fibrillation

- AV node ablation with pacemaker implant
  - recently shown to have no effect on mortality
  - effective at reducing symptoms
  - Does not alter need for anticoagulation
  - Pace at 90 BPM 1 month after procedure to avoid Torsades des Pointes
Drugs for Stable Tachycardias
Diltiazem

- Indications *(When & Why?)*
  - To control ventricular rate in atrial fibrillation and atrial flutter
  - Use after adenosine to treat refractory PSVT in patients with narrow QRS complex and adequate blood pressure
  - As an alternative, use verapamil

*Stable Tachycardias*
Diltiazem

- **Dosing** *(How?)*
  - **Acute Rate Control**
    - 15 to 20 mg (0.25 mg/kg) IV over 2 minutes
    - May repeat in 15 minutes at 20 to 25 mg (0.35 mg/kg) over 2 minutes
  - **Maintenance Infusion**
    - 5 to 15 mg/hour, titrated to heart rate

*Stable Tachycardias*
Diltiazem

- Precautions *(Watch Out!)*
  - Do not use calcium channel blockers for tachycardias of uncertain origin
  - Avoid calcium channel blockers in patients with Wolff-Parkinson-White syndrome, in patients with sick sinus syndrome, or in patients with AV block without a pacemaker
  - Expect blood pressure drop resulting from peripheral vasodilation
  - Concurrent IV administration with IV β-blockers can cause severe hypotension

*Stable Tachycardias*
Verapamil

- **Indications (When & Why?)**
  - Used as an alternative to diltiazem for ventricular rate control in atrial fibrillation and atrial flutter
  - Drug of second choice (after adenosine) to terminate PSVT with narrow QRS complex and adequate blood pressure

*Stable Tachycardias*
Verapamil

- Dosing *(How?)*
  - 2.5 to 5.0 mg IV bolus over 1 to 2 minutes
  - Second dose: 5 to 10 mg, if needed, in 15 to 30 minutes. Maximum dose: 30 mg
  - Older patients: Administer over 3 minutes

*Stable Tachycardias*
Verapamil

- **Precautions** *(Watch Out!)*
  - Do not use calcium channel blockers for wide-QRS tachycardias of uncertain origin
  - Avoid calcium channel blockers in patients with Wolff-Parkinson-White syndrome and atrial fibrillation, sick sinus syndrome, or second- or third-degree AV block without pacemaker

*Stable Tachycardias*
Verapamil

- Precautions *(Watch Out!)*
  - Expect blood pressure drop caused by peripheral vasodilation
  - IV calcium can restore blood pressure, and some experts recommend prophylactic calcium before giving calcium channel blockers
  - Concurrent IV administration with IV β-blockers may produce severe hypotension

*Stable Tachycardias*
Indications (When & Why?)

- First drug for narrow-complex PSVT
- May be used diagnostically (after lidocaine) in wide-complex tachycardias of uncertain type

Stable Tachycardias
Adenosine

- **Dose** *(How?)*
  - IV Rapid Push
  - Initial bolus of 6 mg given rapidly over 1 to 3 seconds followed by normal saline bolus of 20 mL; then elevate the extremity
  - Repeat dose of 12 mg in 1 to 2 minutes if needed
  - A third dose of 12 mg may be given in 1 to 2 minutes if needed

*Stable Tachycardias*
Adenosine

- Precautions *(Watch Out!)*
  - Transient side effects include:
    - Facial Flushing
    - Chest pain
    - Brief periods of asystole or bradycardia
  - Less effective in patients taking theophyllines

*Stable Tachycardias*
Beta Blockers

- Indications *(When & Why?)*
  - To convert to normal sinus rhythm or to slow ventricular response (or both) in supraventricular tachyarrhythmias (PSVT, atrial fibrillation, or atrial flutter)
  - β-Blockers are second-line agents after adenosine, diltiazem, or digoxin

Stable Tachycardias
Beta Blockers

- **Dosing (How?)**
  - **Esmolol**
    - 0.5 mg/kg over 1 minute, followed by continuous infusion at 0.05 mg/kg/min
    - Titrate to effect, Esmolol has a short half-life (<10 minutes)
  - **Labetalol**
    - 10 mg labetalol IV push over 1 to 2 minutes
    - May repeat or double labetalol every 10 minutes to a maximum dose of 150 mg, or give initial dose as a bolus, then start labetalol infusion 2 to 8 µg/min

*Stable Tachycardias*
Beta Blockers

- Dosing *(How?)*
  - Metoprolol
    - 5 mg slow IV at 5-minute intervals to a total of 15 mg
  - Propranolol
    - 1 to 3 mg slow IV. Do not exceed 1 mg/min
    - Repeat after 2 minutes if necessary

*Stable Tachycardias*
Beta Blockers

- Precautions *(Watch Out!)*
  - Concurrent IV administration with IV calcium channel blocking agents like verapamil or diltiazem can cause severe hypotension
  - Avoid in bronchospastic diseases, cardiac failure, or severe abnormalities in cardiac conduction
  - Monitor cardiac and pulmonary status during administration
  - May cause myocardial depression

*Stable Tachycardias*
Indications *(When & Why?)*

- To slow ventricular response in atrial fibrillation or atrial flutter
- Third-line choice for PSVT

Stable Tachycardias
Digoxin

- **Dosing (How?)**
  - IV Infusion
    - Loading doses of 10 to 15 µg/kg provide therapeutic effect with minimum risk of toxic effects
    - Maintenance dose is affected by body size and renal function

*Stable Tachycardias*
Digoxin

- Precautions (*Watch Out!*)
  - Toxic effects are common and are frequently associated with serious arrhythmias
  - Avoid electrical cardioversion unless condition is life threatening
    - Use lower current settings (10 to 20 Joules)

*Stable Tachycardias*