Identification and Management of Common Cardiac Arrhythmias

Supraventricular Tachycardias

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
    - Origin from foci above or within the atrioventricular node
    - Main players in outpatient setting
    - All the favorites
      - AV nodal reentrant tachycardia (SVT)
      - Atrial flutter
      - Atrial fibrillation

Location

- Supraventricular Arrhythmias
  - Origin from foci above or within the atrioventricular node
- Ventricular Arrhythmias
  - Non-sustained ventricular tachycardia
  - Sustained ventricular tachycardia
    - Stable
    - Know the neighborhood
    - On-set form
    - Unstable
    - ACLS
  - Ventricular Fibrillation
    - Never a stable rhythm
    - Immediate ACLS

Bradyarrhythmias

- Sinus Bradycardia
  - Sinus rhythm with a resting heart rate of 60 beats/minute or less
  - Few patients actually become symptomatic until their heart rate drops to less than 50 beats/minute
  - Pathophysiology of sinus bradycardia is dependent upon the underlying cause
  - Commonly, sinus bradycardia is an incidental finding in otherwise healthy individuals, particularly in young adults or sleeping patients
Bradyarrhythmias

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

- **History:**
  - Sinus bradycardia is most often asymptomatic. However, symptoms may include the following:
    - Syncope
    - Dizziness
    - Lightheadedness
    - Chest pain
    - Shortness of breath
  - Pertinent elements of the history include the following:
    - Previous cardiac history (e.g., myocardial infarction, congestive heart failure, valvular failure)
    - Medications
    - Toxic exposures
    - Prior illnesses

- **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

- **Causes:**
  - A broad variety of other drugs and toxins have been reported to cause bradycardia, including
    - lidocaine
    - propranolol
    - ibuprofen
    - dimethyl sulfoxide (DMSO)
    - topical ophthalmic acetylcholine
    - fentanyl
    - alfentanil
    - sufentanil
    - reserpine
    - 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.
Bradyarrhythmias

- **Lab Studies:**
  - Laboratory studies may be helpful if the cause of the bradycardia is thought to be related to electrolytes, drug, or toxins.
  - 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

- **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

Bradyarrhythmias

- **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.

Bradyarrhythmias

- **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.

AV Block

- **First-degree heart block, or first-degree atrioventricular (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.

AV Block

- **First-degree heart block, or first-degree atrioventricular (AV) block**
  - **Frequency:**
    - The prevalence of first-degree AV block among young adults ranges from 0.65-1.6%.
    - Higher prevalence is reported in studies of trained athletes (8.7%) and medical students (8%).
    - It is more common among African Americans compared with Caucasian populations.
    - The prevalence of first-degree AV block increases with advancing age.
First-degree heart block, or first-degree atrioventricular (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 heart block, or second-degree atrioventricular (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.

- **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.

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 heart block, or second-degree atrioventricular (AV) block

- **Mobitz type II second-degree AV block**
  - Carries a risk of progressing to complete heart block.
  - Is associated with an increased risk of mortality.

Mobitz type I second-degree AV block localized to the AV node

- Not associated with any increased risk of morbidity or death, in the absence of organic heart disease.
- No risk of progression to a type II second-degree block or complete heart block exists.
- When a Mobitz type I block occurs during an acute myocardial infarction, mortality is increased.

Mobitz type II block

- Carries a risk of progressing to complete heart block.
- Is associated with an increased risk of mortality.
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.
  - 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.

- **Causes:**
  - Mobitz I block
    - Can occur in individuals with high vagal tone, such as athletes or young children.
    - Can occur in infants and young children with structural heart disease (e.g., tetralogy of Fallot) and in individuals of any age following valvular surgery (especially mitral valve).
    - Other causes of type I block include myocardial infarction (especially inferior wall), and drug-induced block (including beta blockers, calcium channel blockers, amiodarone, digoxin, and possibly pentamidine).
  - Mobitz II block
    - Most commonly is caused by an acute myocardial infarction (anterior or inferior).
    - Drug-induced etiologies can also occur.

- **Complete heart block, also referred to as third-degree heart block, or third-degree atrioventricular (AV) block**
  - Disorder of the cardiac conduction system where there is no conduction through the AV node.
  - Complete dissociation of the atrial and ventricular activity exists.
  - Ventricular escape mechanism can occur anywhere from the AV node to the bundle-branch Purkinje system.
  - It is important to realize, however, that 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.
  - Electrocardiographically, complete heart block is represented by QRS complexes being conducted at their own rate and totally independent of the P waves.

AV Block

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

- **Lab Studies:**
  - Serum electrolytes, calcium, and magnesium levels should be checked.
  - 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 (e.g., 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.

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

- **History:**
  - Complete heart block has a wide range of clinical presentations; most patients are asymptomatic.
  - Patients occasionally are asymptomatic or have only minimal symptoms related to hypoperfusion. In these situations, symptoms include the following:
    - Fatigue
    - Dyspnea
    - Impaired exercise tolerance
    - Chest pain
AV Block

- Because an acute myocardial infarction is one cause of complete heart 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 cardiodiglycosides

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.
  - In the absence of major structural abnormalities, congenital heart block is often associated with maternal antibodies to SS-A (Ro) and SS-B (La).

AV Block

- Acquired complete heart block
  - Infectious causes include the following:
    - Cardiomyopathy, eg, Lyme cardiitis 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.

AV Block

- Physical:
  - Notable for bradycardia, which can be quite severe.
  - Signs of congestive heart failure as a result of decreased cardiac output may be present and include the following:
    - Tachypnea or respiratory distress
    - Edema
    - Jugular venous distention
  - Patients may have signs of hypoperfusion, including the following:
    - Altered mental status
    - Hypotension
    - Leathery
  - In patients with concomitant myocardial ischemia or infarction, corresponding signs may be evident on examination:
    - Signs of anxiety such as agitation or uncase
    - Diaphoresis
    - Pale or pasty complexion
    - Tachypnea

AV Block

- 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

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>
<tbody>
<tr>
<td>Third</td>
<td>Symptomatic congenital complete heart block</td>
<td>Asymptomatic congenital complete heart block</td>
<td>Asymptomatic complete heart block</td>
</tr>
<tr>
<td>Second</td>
<td>Symptomatic type I</td>
<td>Asymptomatic type I</td>
<td>Asymptomatic type I at infra-His levels</td>
</tr>
<tr>
<td>First</td>
<td>Symptomatic type I</td>
<td>Asymptomatic type II</td>
<td>Asymptomatic type II at infra-His block</td>
</tr>
</tbody>
</table>

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>
</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)

Atropine Sulfate

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

- **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
**Atropine Sulfate**

- **Precautions *(Watch Out!)***
  - Use with caution in presence of myocardial ischemia and hypoxia
  - Increases myocardial oxygen demand
  - Seldom effective for:
    - Infranodal (type II) AV block
    - Third-degree block (Class IIb)

**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

**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

**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)

- **Precautions** *(Watch Out!)*
  - Raising blood pressure and increasing heart rate may cause myocardial ischemia, angina, and increased myocardial oxygen demand
  - Do not mix or give with alkaline solutions

**Isoproterenol**

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

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

- **Precautions** *(Watch Out!)*
  - Increases myocardial oxygen requirements, which may increase myocardial ischemia
  - *DO NOT* administer with poison/drug-induced shock
  - *Exception:* Beta Blocker Poisoning

**Tachyarrhythmias**

Ectopic rate nomenclature:

<table>
<thead>
<tr>
<th>Rate</th>
<th>Rhythm</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>
Mechanisms of Arrhythmia

- Abnormal automaticity
  - Automatic impulse generation from unusual site or overrides sinus node
- Triggered activity
  - Secondary depolarization during or after repolarization
  - Dig toxicity, Torsades de Pointes
- Reentry
  - 90% of arrhythmias

Reentry

- Most common mechanism
- Requires two separate paths of conduction
- Requires an area of slow conduction
- Requires unidirectional block

Supraventricular Tachycardias Diagnosis

- ECG is cornerstone
- Observe zones of transition for clues as to mechanism:
  - Onset
  - Termination
  - Slowing, AV nodal block
  - Bundle branch block

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

AV Nodal Reentrant Tachycardia

- 2 pathways within or limited to perinodal tissue
- Anterograde conduction down fast pathway blocks with conduction down slow pathway, with retrograde conduction up fast pathway.
- May have very short RP interval with retrograde P wave visible as an R' in lead V1 or pseudo-S wave in inferior leads in 1/3 of cases. No P wave seen in 2/3.

AVNRT: 12-Lead ECG

Retrograde P-waves in leads I, II, V1-V3. These findings are consistent with the pattern of atrial activation from the lower septum upwards as the circuit exits the fast pathway from the AV node. AVNRT typically presents retrograde P waves closely coupled after the QRS, though often the P wave is buried in the QRS itself.
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

Orthodromic Reciprocating Tachycardia (ORT)
- Anterograde over AV node and retrograde conduction of an accessory pathway.
- RP interval short but longer than AVNRT due to required conduction through ventricle prior to conduction up accessory pathway.
- Frequently presents in patients with WPW patients as narrow complex tachycardia

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

Differential Dx of Regular SVT
- Long RP tachycardia
  - Atrial tachycardia
  - Sinus node reentry
  - Sinus tachycardia
  - Atypical AV nodal reentrant tachycardia
  - Permanent form of junctional reciprocating tachycardia

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 I 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.
**Other Long RP tachycardias**

- Sinus node reentrant
  - abrupt onset and offset
  - P wave complex same as sinus
  - Amenable to calcium channel blockers, much less responsive to beta blockers
  - Amenable to catheter ablation
- Synd. of inappropriate sinus tachycardia
  - typical sinus tachycardia with lowest rate on Holter of 130 bpm
  - Treated with high dose beta blockers
  - Poor results with catheter ablation

**Tachyarrhythmias**

- **History:**
  - 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).

- **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 without 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.

- **Other Long RP tachycardias**
  - AVNRT may occur in persons of any age. It is common in young adults. AVNRT may cause or worsen heart failure in patients with coronary artery disease. AVNRT may cause angina or myocardial infarction.
  - 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**

- **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

- **Physical:**
  - The heart rate is usually rapid, 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.
  - Sometimes, initial hypotension evokes a sympathetic response that increases blood pressure and may terminate the tachycardia by an increase in vagal tone.
  - Signs of left heart failure may develop or worsen in patients with poor left ventricular function.
Tachyarrhythmias

Other Tests:
- ECG or ambulatory monitoring
  - Evaluation usually reveals a supraventricular origin of QRS complexes at rates of 150-250 bpm and a regular rhythm.
  - The QRS complex usually narrows unless a conduction abnormality is present or is functionally induced from the rapid heart rate.
  - P waves are not usually seen because they are buried within the QRS complex. A pseudo R prime may be seen in V1, or pseudo S waves may be seen in leads II, III, or aVF. The onset is abrupt with an atrial premature complex, which conducts with a prolonged PR interval.
  - The PR interval may shorten over the first few beats at onset, or it may lengthen during last few beats preceding termination of the tachycardia.
  - Abrupt termination occurs with a retrograde P wave, sometimes followed by a brief period of asystole or bradycardia.
- Other Tests: ECG or ambulatory monitoring
  - Evaluation usually reveals a supraventricular origin of QRS complexes at rates of 150-250 bpm and a regular rhythm.
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  - Evaluation usually reveals a supraventricular origin of QRS complexes at rates of 150-250 bpm and a regular rhythm.

ECG Distinction of VT from SVT with Aberrancy

Favors VT | Favors SVT with Aberrancy
---|---
Duration | RBBB: QRS > 0.14 sec. | < 0.14 sec.
LBBB: QRS > 0.16 sec. | < 0.16 sec.
Axis | QRS axis -90° to ±180° | Normal

Morphology

If LBBB:
- V1 duration > 30 ms
- S wave > 70 ms
- S wave notched or slurred
- V5: qR or QR R wave
If RBBB:
- V1: monophasic R wave qR
- If triphasic, R > R1 R < R1
- V6: R < S

ACLS algorithm if no response to these measures.

Medical Care:
- Management of an acute attack depends on the symptoms, the presence of underlying heart disease, and the natural history of previous episodes.
- Rest, reassurance, and sedation may terminate the attack.
- To terminate the tachycardia, try 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.

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 flutter

Relatively common atrial tachyarrhythmia.

- Atrial flutter is the most significant of the atrial tachyarythmias.
- 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

Frequency:

- Atrial flutter affects approximately 88 out of 100,000 new patients each year.
- This represents approximately 200,000 patients presenting with atrial flutter annually.

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

Pathophysiology:

- Multiple re-entrant or primarily generated (ectopic) atrial waveforms bombards the atrioventricular (AV) node.
- The two forms of atrial flutter are known as type I and type II.

Type I is the most common form

- Also referred to as typical, common, or counter-clockwise sinus-node-dependent atrial flutter and involves an 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 flutter, is still poorly characterized, but may result from an intra-atrial 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

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

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

Physical:

- Pertinent physical findings are limited to cardiovascular system.
- If embolization has occurred from intermittent AF, findings are related to brain and/or peripheral vascular involvement.
- 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 (This is dependent on the atrial firing rate, which may be influenced by medications as well as intrinsic cardiac factors).
- 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 left ventricle dysfunction.
Atrial flutter

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

- 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

- 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 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
  - Rate control is the goal of medication in atrial flutter or AF.
  - Beta-adrenergic blockers are especially 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.
  - Radiofrequency ablation is immediately successful in more than 90% of cases and avoids the long-term toxicity observed with antiarrhythmic drugs.
  - When considering drug therapy for atrial flutter/fibrillation, remember the treatment caveat: "electrical cardioversion is the preferred modality in the patient whose condition is unstable."
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

- Commonly broken down into acute versus chronic AF.
  - Paroxysmal - Duration less than 7 days, with spontaneous termination.
  - Persistent - Duration greater than 7 days and would last indefinitely unless cardioverted.
  - Permanent - Duration greater than 7 days, with restoration to sinus rhythm not possible.
  - Lone AF - Used to describe AF in individuals without structural or cardiac or pulmonary disease, with low risk for thromboembolism.
  - It has traditionally been applied to patients younger than 60 years.

Atrial Fibrillation

- Frequency:
  - Approximately 1% of the total population, currently have atrial fibrillation.
  - Atrial fibrillation can be considered a disease of aging, and with the projected increase in the elderly population, the prevalence is expected to more than double by the year 2050.

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 absence of structural heart disease but 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.

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

- Mortality/Morbidity:
  - Rate of ischemic stroke among patients with nonrheumatic AF averages 5%.
  - Between 2-7 times the rate of stroke in patients without AF.
  - Risk of stroke is not due solely to AF; it increases substantially in the presence of other cardiovascular disease.
  - The attributable risk of stroke from AF is estimated to be 1.5% for those aged 50-59 years, and it approaches 30% for those aged 80-89 years.
  - AF complicates acute myocardial infarction (AMI) in 5-10% of cases.
  - Patients who developed new-onset AF during the course of myocardial infarction (MI) are at higher risk than patients who presented with chronic AF.
  - Patients with AMI and AF tend to be older, be less healthy, and have poorer outcomes during hospitalization and after discharge than individuals without AF.
Atrial Fibrillation

- **History:** In addition to eliciting symptoms listed below, history taking of any patient presenting with suspected AF should include questions relevant to temporality, precipitating factors (including hydration status, recent infections, alcohol use), history of pharmacologic or electric interventions and responses, and presence of heart disease. Occasionally, a patient may have clear and strong belief about the onset of symptoms that may be helpful in determining a course of action.
  - Palpitations
  - Fatigue or poor exercise tolerance
  - Dyspnea
  - Chest pain (true angina)
  - Presyncope or syncope
  - Generalized weakness

- **Causes of atrial fibrillation can be divided into cardiovascular versus noncardiovascular causes.**
  - Important cardiovascular causes include the following:
    - Long-standing hypertension
    - Ischemic heart disease
    - CHF
    - Any form of carditis
    - Cardiomyopathy
    - Infiltrative heart disease of any type
    - Sick sinus syndrome
  - Noncardiovascular respiratory causes include the following:
    - Pulmonary embolism
    - Pneumonia
    - Pheochromocytoma
    - Hyperthyroidism
    - 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

- **Other Tests:**
  - ECG: Absent P waves, replaced by irregular, chaotic fibrillatory F waves, in the setting of irregular QRS complexes.
  - Other features to be looked for on the ECG include: LVH, preexcitation, bundle branch blocks, acute or prior MI, and intervals (R-R, QRS, QT).
  - Holter monitoring or event monitoring may be considered for those discharged from the ED (eg, in cases of paroxysmal AF not evident upon presentation).
  - Exercise testing might also be used in the outpatient setting to determine adequacy of rate-control, to reproduce exercise-induced EF, and to exclude ischemic pathology.
Atrial Fibrillation

- Outpatient Therapy
  - Long-term management of AF has most commonly centered around 1 of 2 strategies: rhythm control versus rate control.
  - Five significant randomized clinical trials have taken place in the past few years.
  - A review of these studies yielded the following conclusions in regards to rate versus rhythm control:
    - AFFIRM and RACE both failed to demonstrate a clear benefit with rhythm control strategy.
    - All 5 studies failed to show any significant difference in all-cause or cardiovascular mortality between the two strategies.
    - With respect to stroke, no difference was noted between the two strategies.
    - Warfarin lowers the risk of stroke in both strategies.
    - With respect to quality of life and functional status, all 5 trials failed to show any differences between rate control and rhythm control.

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)

Acute Management of Atrial Fibrillation

- Focuses on Rate control
- Patient with atrial fibrillation may undergo DC cardioversion or pharmacologic conversion if less than 48 hours duration or following TEE on Heparin without evidence of left atrial thrombus. Stroke rate .8%
- Following cardioversion the patient should be kept anticoagulated for 4 weeks with goal INR of 2 to 3 until atrial function normalizes.

Acute Management of Atrial Fibrillation

- 50% of patients with paroxysmal atrial fibrillation will spontaneously convert within 24 hours
- Digoxin used heavily in the past for prevention and conversion of atrial fibrillation is ineffective at either and may be profibrillatory as it decreases the atrial refractory period

Acute Management of Atrial Fibrillation

- Rate control may be attained with calcium channel blockers or beta blockers in patients with normal L.V. function.
- Calcium channel blockers may be used cautiously in patients with depressed LV function but are associated with increased mortality in the long term.
- Beta blockers should be avoided in acutely decompensated CHF patients with atrial fibrillation

Atrial Fibrillation and Depressed L.V. Function

- Digoxin and amiodarone may be of effective in patients with LV dysfunction and decompensated congestive heart failure to slow ventricular response.
- Digoxin alone is rarely effective when the patient is sympathetically driven
- Avoid high dose digoxin with amiodarone as digoxin levels increase 2-fold with amiodarone
Chronic Management of Atrial Fibrillation

- Patients with atrial fibrillation, paroxysmal or sustained should be anticoagulated if any of the following risk factors for stroke are present:
  - diabetes
  - valvular disease
  - hyperthyroidism
  - Prior CVA
  - hypertension
  - congestive heart failure
  - age greater than 65

**Rate control with**
- calcium channel blockers, beta blockers or combination with digoxin.

- Digoxin may be used in bed bound patients but is easily overcome with sympathetic stimulation.

- Maintenance of sinus is similar with class I and class III drugs approaching 50% recurrence at 1 year.

- Recurrence of atrial fibrillation 80% at 1 year without treatment.

Class III agents may have improved efficacy
- Dronedarone
- Amiodarone (pulmonary toxicity, thyroid, liver)
- Dofetilide (Torsades des Pointes, Safe in CHF and CAD, Limited due to side effect profile)

Class IC agents safe in absence of structural heart disease.
- Few side effects
- Need stress testing
- Can lead to 1 to 1 ventricular conduction of atrial flutter
- Use with beta blocker

Recent large trials reveal no benefit of rhythm control over rate control.
- Trend of increased mortality in rhythm arm likely due to proarrhythmia from drugs.
- Patients unable to tolerate atrial fibrillation due to symptoms were not enrolled in these studies and are increasingly undergoing ablation, catheter and surgical procedures.

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

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
Atrial Fibrillation

ACC/AHA Recommendations for Anticoagulation

- Class I
  - All patients with AF, except those with lone AF or contraindications
  - Selection of antithrombotic agent based upon the absolute risks of stroke and bleeding and the relative risk and benefit for a given patient.
  - For patients without mechanical heart valves at high risk of stroke, chronic warfarin therapy is recommended at a dose adjusted to achieve an INR of 2.0 to 3.0 unless contraindicated.

Factors associated with highest risk of stroke are prior thromboembolism (stroke, TIA, or systemic) and rheumatic mitral stenosis.

Anticoagulation with warfarin is recommended for patients with more than one moderate risk factor.

- Age > 75, hypertension, HF, impaired LV systolic function (EF 35% or less) and DM

INR should be determined at least weekly during initiation of therapy and monthly once stable.

Aspirin 81-325 mg daily, is recommended as an alternative to vitamin K in low-risk patients or in those with contraindications to vitamin K antagonist.

Patients with mechanical heart valves should have INR based on requirement of valve (at least 2.5).

Anticoagulate for Aflutter the same as for AF.

WPW syndrome

- Accelerated AV conduction PR <120 msec
- Prolonged QRS > 120 msec
- Abnormal slurred upstroke of QRS (delta wave)
- Abnormal depolarization and repolarization may lead to pseudoinfarction pattern

WPW pathophysiology

- Short AV conduction
- early excitation of ventricle at site of accessory pathway
- Bizarre upstroke of QRS
- abnormal initial site of depolarization
- Wide QRS
- early initiation of ventricular depolarization

The result is fusion of both normal and accessory conduction.

WPW epidemiology

- Present in 0.3% of the population
- Risk of sudden death 1 per 1000 patient-years
- Sudden death due to atrial fibrillation with rapid ventricular conduction
- Atrial fibrillation often induced from rapid ORT

ORT (orthodromic reciprocating tachycardia)
Atrial Fibrillation and WPW

- AV nodal blocking agents may paradoxically increase conduction over accessory pathway by removing concealed retrograde penetration into accessory pathway.

Concealed penetration into the pathway causes intermittent block of pathway conduction.

Management of Atrial Fibrillation with WPW

- Avoid AV nodal blockers
- IV procainamide to slow accessory pathway conduction
- Amiodarone if decreased LVEF
- DC cardioversion if symptomatic with hypotension

Management of Patients with WPW

- All patients with symptomatic AF & WPW should be evaluated with EPS
- Accessory pathways capable of conducting faster than 240 BPM should be ablated
- Patients with inducible arrhythmias involving pathway should be ablated
- WPW patients in high risk professions should be ablated.

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 ß-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

**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

**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

Adenosine

**Indications (When & Why?)**
- First drug for narrow-complex PSVT
- May be used diagnostically (after lidocaine) in wide-complex tachycardias of uncertain type

**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
Adenosine

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

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)
  - ß-Blockers are second-line agents after adenosine, diltiazem, or digoxin

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

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

- **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)

Amiodarone

- **Indications (When & Why?)**
  - Powerful antiarrhythmic with substantial toxicity, especially in the long term
  - Intravenous and oral behavior are quite different

- **Dosing (How?)**
  - **Stable Wide-Complex Tachycardias**
    - Rapid Infusion
      - 150 mg IV over 10 minutes (15 mg/min)
      - May repeat
    - Slow Infusion
      - 360 mg IV over 6 hours (1 mg/min)

- **Precautions (Watch Out!)**
  - May produce vasodilation & shock
  - May have negative inotropic effects
  - May prolong QT Interval
    - DO NOT administer with other drugs that may prolong QT Interval (Procainamide)
  - Terminal elimination
    - Half-life lasts up to 40 days
Amiodarone

- Precautions *(Watch Out!)*
  - Contraindicated in:
    - Second or third degree A-V block
    - Severe bradycardia
    - Pregnancy
    - CHF
    - Hypokalaemia
    - Liver dysfunction