L3: Arrhythmias

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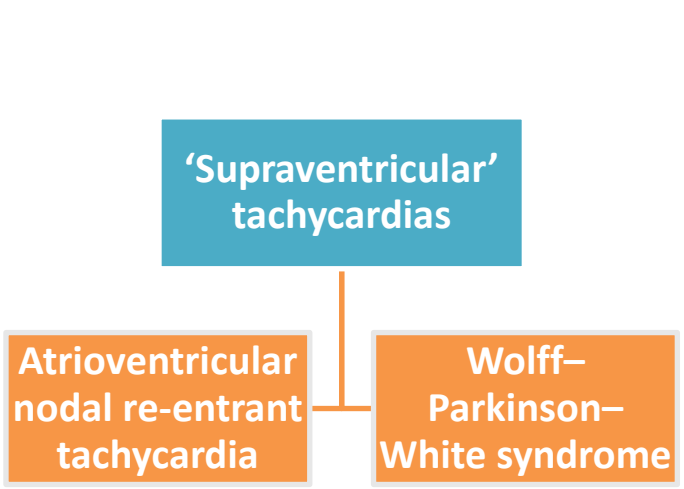
MEDICINE 433

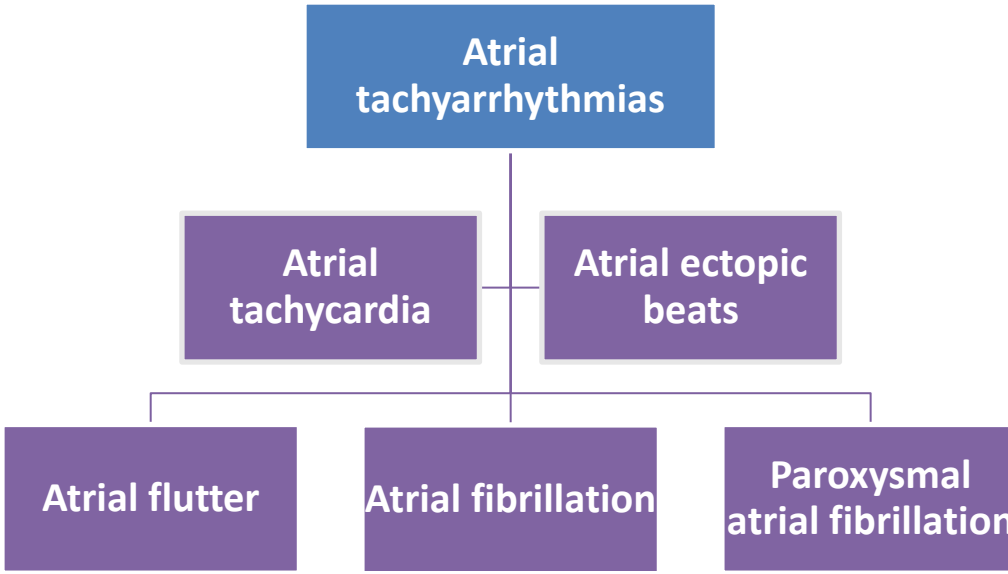
objectives

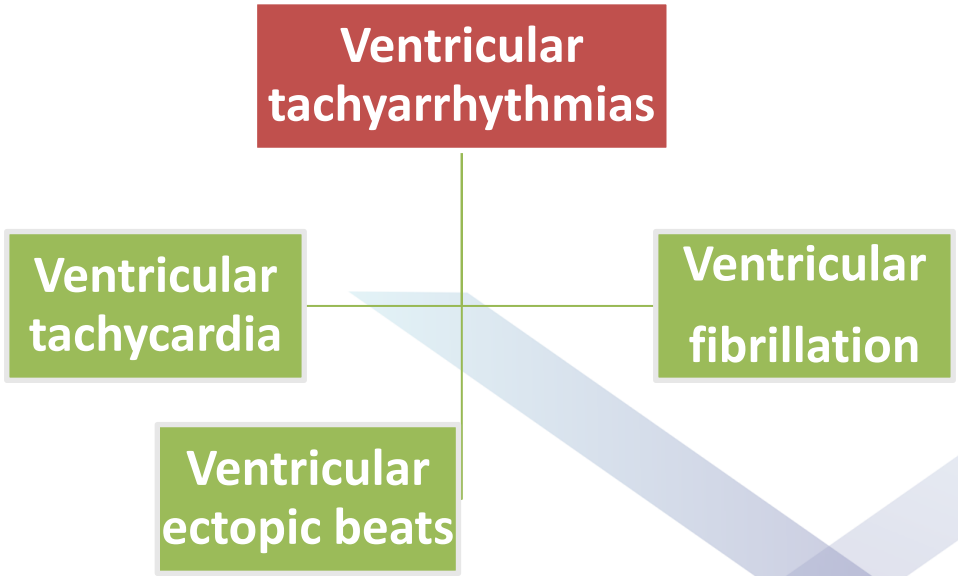
1. Identify the common arrhythmias.
2. Know the differential diagnosis of heart rhythm disorder.
3. Learn the treatment modalities and diagnosis of atrial fibrillation.
4. Know the risk stratification of atrial fibrillation patient.

Color index: Step up to medicine , slide , Female's note , Male's note , Davidson

Mind Map


'Supraventricular' tachycardias
Atrioventricular nodal re-entrant tachycardia
Wolff–Parkinson–White syndrome


Atrial tachyarrhythmias
Atrial tachycardia
Atrial ectopic beats
Atrial flutter
Atrial fibrillation
Paroxysmal atrial fibrillation


Ventricular tachyarrhythmias
Ventricular tachycardia
Ventricular fibrillation
Ventricular ectopic beats

Atrial Tachyarrhythmias

Atrial tachycardia

-may be a manifestation of increased atrial automaticity, sinoatrial disease or digoxin toxicity
-It produces a narrow-complex tachycardia with abnormal P-wave morphology
-It may respond to β-blockers or class I or III anti-arrhythmic drugs
-Catheter ablation can be used to target the ectopic site and should be offered as an alternative to anti-arrhythmic drugs in patients with recurrent atrial tachycardia.

Atrial ectopic beats

-These usually cause no symptoms but can give the sensation of a missed beat or an abnormally strong beat.
-The ECG shows a premature but otherwise normal QRS complex.
-atrial ectopic beats may herald the onset of atrial fibrillation.
-β-blockers can be used if symptoms are intrusive.

Paroxysmal atrial fibrillation

-Occasional attacks that are well tolerated do not necessarily require treatment.
-Beta-blockers are normally used as first-line therapy if symptoms are troublesome.
-propafenone or flecainide, are also effective at preventing episodes but should not be given to patients with coronary artery disease or left ventricular dysfunction.
-Flecainide is usually prescribed along with a rate limiting β-blocker because it occasionally precipitates atrial flutter.
-amiodarone is the most effective agent for preventing AF but its side-effects restrict its use to patients in whom other measures fail.
-Dronedarone is an effective alternative, but is contraindicated in patients with heart failure or significant left ventricular impairment.
-Ablation for AF is an attractive treatment for patients in whom drugs are ineffective or poorly tolerated.

Atrial Fibrillation

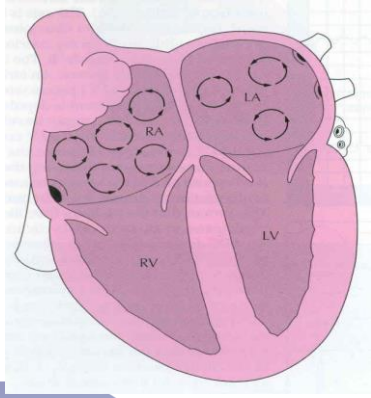
- Accounts for 1/3 of all patient discharges with arrhythmia as principal diagnosis.
- 2.3 million people in north america
- 4.5 million in EU
- In the 20 year AF admission have increased by 66%.
- $ 15.7 billion annually in EU
- Estimated prevalence of AF is 0.4% to 1% in the general pop. 8% in pt. >80 years

History and physical examination

- **Clinical conditions associated with AF**
  - Underlying heart conditions (e.g. valvular heart disease, heart failure, coronary artery disease, hypertension)
  - Other reversible conditions
- **Family history**
  - Familial AF (lone AF in a family)
  - AF secondary to other genetic conditions (familial cardiomyopathies)
- **Type of AF**
  - First episode, paroxysmal, persistent, permanent
  - Triggers – e.g. emotional stress, alcohol, physical exercise, gastroesophageal disease
  - Specific symptoms
  - Response to any treatments administered

Pathophysiology of Atrial Fibrillation

- Multiple foci in the atria fire continuously in a chaotic pattern, causing a totally **irregular, rapid ventricular rate.** Instead of intermittently contracting, the atria quiver continuously. Many episodes resolve spontaneously.
- Atrial rate is over 400 bpm (350–600 beats/min) , but most impulses are blocked at the AV node so ventricular rate ranges between 75 and 175 (Reduced filling) tends to become persistent or permanent.
- Contraction of the ventricles can be:
  - Irregular and rapid (110–180 beats/min; tachycardia)
  - Irregular and slow (<50 beats/min; bradycardia)
  - Normal
- AF causes remodelling:
  - **Electrical**: shortening of refractory period
  - **Structural**: enlargement of atrial cavities
- Patients with AFib and underlying heart disease are at a markedly increased risk for adverse events, such as thromboembolism and hemodynamic compromise.



Risk Factors

Cardiac Causes

- Hypertensive heart disease
- Ischemic heart disease
- Valvular heart disease
  - Rheumatic: mitral stenosis
  - Non-rheumatic: aortic stenosis, mitral regurgitation
- Pericarditis
- Cardiac tumors: atrial myxoma
- Sick sinus syndrome
- Cardiomyopathy
  - Hypertrophic
  - Idiopathic dilated (lone atrial fibrillation)
- Post-coronary bypass surgery

Non-Cardiac Causes

- Pulmonary
  - COPD
  - Pneumonia
  - Pulmonary embolism
- Metabolic
  - Thyroid disease: hyperthyroidism, Hypothyroidism.
  - Electrolyte disorder
- Toxic: alcohol ('holiday heart' syndrome)
- Systemic illness (e.g., sepsis, malignancy, DM)
- Stress (e.g., postoperative)
- Pheochromocytoma

Signs & Symptoms

Cause	Sign/symptom
• Irregular heart beat	• Irregularly irregular pulse • Palpitations
• Decreased cardiac output	• Fatigue • Diminished exercise capacity • Breathlessness (dyspnoea) • Weakness (asthenia)
• Hypotension	• Dizziness and fainting (syncope)
• Cardiac ischaemia	• Chest pain (angina)
• Increased risk of clot formation	• Thromboembolic TIA, stroke

Atrial Fibrillation

Clinical Presentation

- With or without detectable heart disease
- **Episodic**
  - Symptoms may be absent or intermittent
  - Up to 90% of episodes may not cause symptoms
- **Symptoms vary according to**
  - Irregularity and rate of ventricular response
  - Functional status
  - AF duration
  - Patient factors
  - Co-morbidities

Clinical evaluation

- **All patients**
  - History
  - Physical examination
  - Electrocardiogram (ECG)
  - Transthoracic echocardiogram (TTE)
  - Blood tests
  - Holter monitor
  - Chest x-ray
- **Selected patients**
  - Transesophageal echocardiogram (TEE)

Classification of AF: joint guidelines of the ACC, AHA and ESC (1)

Classification	Definition
First-detected	First recognised episode of AF
Recurrent - Paroxysmal - Persistent	≥2 episodes of arrhythmia AF that terminates spontaneously AF than persists for >7 days but can be converted with cardioversion
Permanent	AF that cannot be terminated by cardioversion, and long-standing AF (>1 year) where cardioversion not indicated/not attempted

Classification of AF: joint guidelines of the ACC, AHA and ESC (2)

Classification	Definition
Lone or primary	AF without clinical/ECG evidence of cardiopulmonary disease
Secondary	AF associated with cardiopulmonary disease (e.g. myocardial infarction or pneumonia)
Non-valvular	AF that is not associated with damage to the heart valves (e.g. rheumatic mitral valve disease, prosthetic heart valve or mitral valve repair)

How To Diagnose

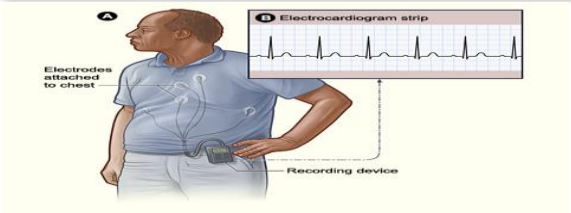
1. Holter monitoring

Portable ECG device

Continuous monitoring for a short period of time (typically 24 hours)

Useful for:

- Detecting asymptomatic AF
- Evaluating patients with paroxysmal AF
- Associating symptoms with heart rhythm disturbance
- Assessing response to treatment



2. Transoesophageal echocardiography

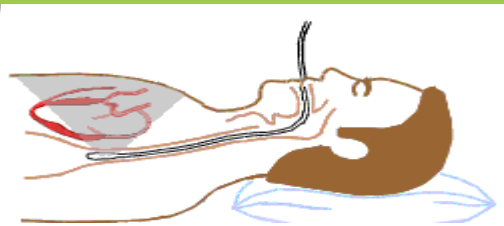
Ultrasound transducer positioned close to the heart using an endoscope-like device

High quality images of cardiac structure and function

- Particularly the left atrial appendage, the most common site of thrombi in patients with AF

Not routinely used but useful for:

- Accurate assessment of risk of stroke
- Detection of low flow velocity ('smoke' effect)
- Sensitive detection of atrial thrombi



3. Transthoracic echocardiography

Non-invasive

Used to identify

Size and functioning of atria and ventricles

Ventricle hypertrophy

Pericardial disease

Valvular heart disease

4.Chest radiography

When clinical findings suggest an abnormality chest radiography may be used to

Evaluate pulmonary pathology and vasculature

Detect congestive heart failure

Assess enlargement of the cardiac chambers

5. Laboratory tests

Routine blood tests should be carried out at least once in patients with AF

Important parameters to assess include:

- Thyroid function
- Renal function
- Hepatic function
- Serum electrolytes
- Complete blood count

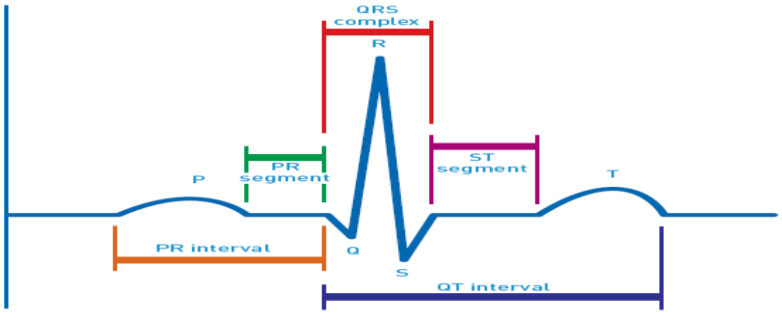
6. Signs and symptom

7. Exercise testing

8.Electrocardiogram

ECG

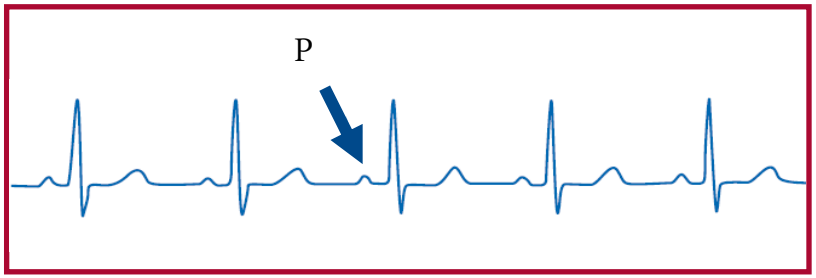
- **Assesses the electrical activity of the heart**
- Essential for all patients with suspected AF, to identify
  - Abnormal heart rhythm (verify AF)
  - Left ventricular hypertrophy
  - Pre-excitation
  - Bundle-branch block
  - Prior MI
  - Differential diagnosis of other atrial arrhythmias
- Impulse from sinoatrial (SA) node stimulates myocardium to contract
- P-wave: atrial depolarization
- QRS complex: ventricular depolarization
- T-wave: ventricular repolarization



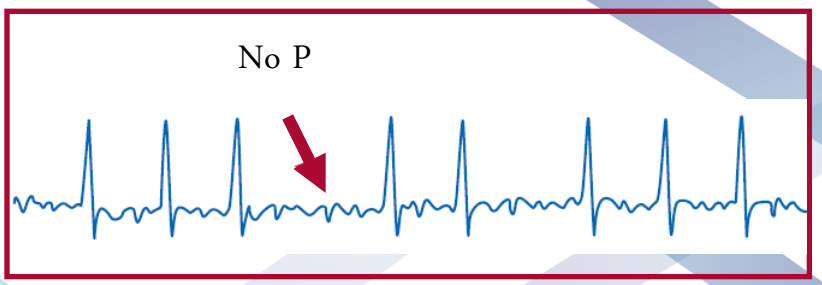
ECG

- ECG findings: Irregularly irregular rhythm (irregular RR intervals and excessively rapid series of tiny, erratic spikes on ECG with a wavy baseline and no identifiable P waves)

- **Normal sinus rhythm**
  - Normal heart rate
  - Regular rhythm
  - P Waves
  - Steady baseline



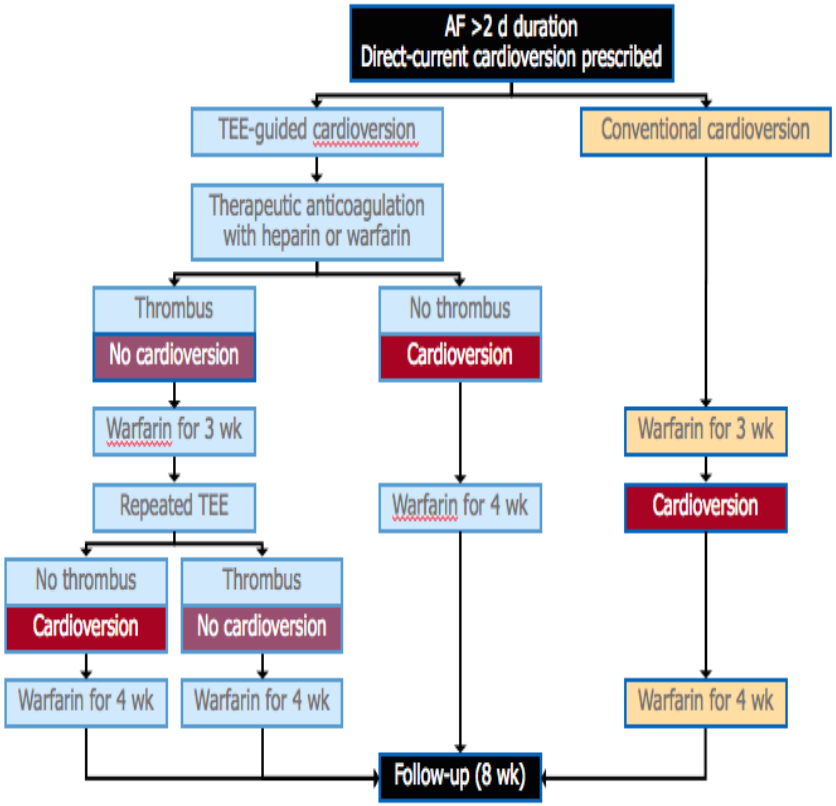
- **AF**
  - Heart rate increased (tachyarrhythmia)*
  - Irregular rhythm
  - No P wave
  - Irregular baseline



Rhythm-control therapies

- The objective of rhythm-control therapy is to restore (cardioversion) and maintain normal sinus rhythm
- Cardioversion can be achieved by:
  - Pharmacotherapy with antiarrhythmic agents
  - Electrical shocks (direct-current cardioversion)
- Direct-current cardioversion is generally more effective than pharmacotherapy
- Likelihood of successful cardioversion decreases with the duration of AF
  - Pharmacological cardioversion is most effective when initiated within 7 days of AF onset
- Cardioversion can dislodge thrombi in the atria, increasing the risk of stroke
  - Thromboprophylaxis is recommended for ≥3 wk before and for at least 4 wks after cardioversion in patients with AF that has persisted for ≥48 h

TEE-guided cardioversion: ACUTE study design



DC = direct-current; TEE = transoesophageal echocardiography

Treatment

- **Chronic Afib**

a. Rate control with a β-blocker or calcium channel blocker

b. Anticoagulation

  – Patients with “lone” AFib (i.e., AFib in the absence of underlying heart disease or other cardiovascular risk factors) **under age 60** do not require anticoagulation because they are at low risk for embolization (aspirin may be appropriate).

  – Treat all other patients with chronic anticoagulation (warfarin).

- **Acute Afib** in a hemodynamically ***unstable**** patient:

Immediate electrical cardiover-sion to sinus rhythm

*means
Chest pain
Shortance of breath
Hypotension
confusion

Treatment

-Digoxin is not recommended for hypotensive patients and for post-opretive patients
-Amiodarone is recommended for elderly not for adult.

- **Acute AFib** in a hemodynamically ***stable*** patient:

a. Rate control

- Determine the pulse in a patient with AFib. If it is too rapid, it must be treated. Target rate is 60 to 100 bpm.
- β-blockers are preferred. Calcium channel blockers are an alternative.
- If left ventricular systolic dysfunction is present, consider digoxin or amiodarone (useful for rhythm control).
- Non pharmacological: Ablate/pace

b. Cardioversion to sinus rhythm (after rate control is achieved)

- Candidates for cardioversion include those who are hemodynamically unstable, those with worsening symptoms, and those who are having their first ever case of AFib.
- Electrical cardioversion is preferred over pharmacologic cardioversion. Attempts should be made to control ventricular rate before attempting DC cardioversion.
- Use pharmacologic cardioversion only if electrical cardioversion fails or is not feasible: Parenteral ibutilide, procainamide, flecainide*, sotalol, dronedarone
- or amiodarone are choices.
- Non pharmacological: Ablation, Surgery (MAZE)

c. Anticoagulation to prevent embolic cerebrovascular accident (CVA)

- If AFib present >48 hours (or unknown period of time), risk of embolization during cardioversion is significant (2% to 5%). Anticoagulate patients for 3 weeks before and 4 weeks after cardioversion.
- **An INR of 2 to 3 is the anticoagulation goal range.**
- To avoid waiting 3 weeks for anticoagulation, obtain a transesophageal echo-
- cardiogram (TEE) to image the left atrium. If no thrombus is present, start IV heparin and perform cardioversion within 24 hours. Patients still require
  4 weeks of anticoagulation after cardioversion.
- Removal/isolation of left atrial appendage, e.g. WATCHMAN® device or surgery

Prevention of Thromboembolism

- The $CHADS_2$ Index
- Stroke Risk Score for Atrial Fibrillation

	Score (points)	Prevalence (%)*
Congestive Heart failure	1	32
Hypertension	1	65
Age >75 years	1	28
Diabetes mellitus	1	18
Stroke or TIA	2	10
Moderate-High risk	>2	50-60
Low risk	0-1	40-50

- The CHA_2DS_2VASc Index
- Stroke Risk Score for Atrial Fibrillation

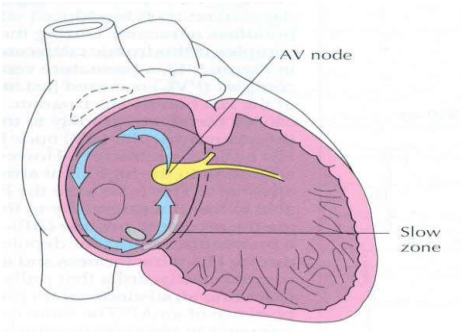
	Weight (points)
Congestive heart failure or LVEF < 35%	1
Hypertension	1
Age >75 years	2
Diabetes mellitus	1
Stroke/TIA/systemic embolism	2
Vascular Disease (MI/PAD/Aortic plaque)	1
Age 65-74 years	1
Sex category (female)	1
Moderate-High risk	> 2
Low risk	0-1

Complications

- Formation of blood clots (thrombosis) on the walls of the atria that can dislodge (embolize), leading to stroke and systemic embolism
- Reduction in cardiac output can precipitate heart failure leading to:
  - Peripheral oedema
  - Pulmonary oedema

-The most common site to find embolism is the Appendage in the atrium.

Atrial Flutter



- **Pathophysiology**

A. One irritable automaticity focus in the atria fires at about 250 to 350 bpm (typically very close to 300 bpm) , giving rise to regular atrial contractions.

B. Atrial rate between around 300 bpm. Ventricular rate is one-half to one-third of the atrial rate. The long refractory period in the AV node allows only one out of every two or three flutter waves to conduct to the ventricles.

- **Causes**

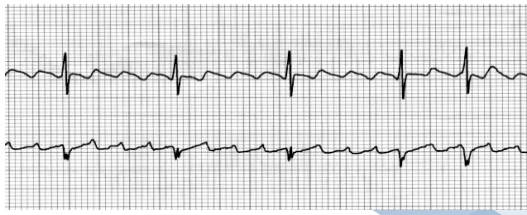
Heart disease: Heart failure (most common association), rheumatic heart disease, CAD

A. COPD

B. Atrial septal defect (ASD)

- **Diagnosis**

ECG provides a saw-tooth baseline, with a QRS complex appearing after every second or third "tooth" (P wave). Saw-tooth flutter waves are best seen in the inferior leads (II, III, aVF).



- **Treatment**

Similar to treatment for Afib

- ***Unstable*** pt (i.e. low BP / CP / AMS):
  - Synchronized cardioversion as per ACLS
  - 50J → 100J → 200J → 300J → 360J
- ***Stable*** pt:
  - Rate control - just like atrial fibrillation (AFib)
  - Elective cardioversion - just like AFib
  - Anti-coagulation – just like AFib
  - Refer for Ablation

-Atrial flutter is characterised by a large (macro) re-entry circuit.
-Carotid sinus pressure or intravenous adenosine may help to establish the diagnosis.

Supraventricular Tachycardia

- The term ‘supraventricular tachycardia’ (SVT) is commonly used to describe regular tachycardias that have a similar appearance on ECG. These are usually associated with a narrow QRS complex and are characterised by a re-entry circuit or automatic focus involving the atria. The term SVT is misleading, as, in many cases, the ventricles also form part of the re-entry circuit, such as in patients with AV re-entrant tachycardia.

- Arrhythmias of supraventricular origin using a re-entrant mechanism with abrupt onset & termination

- AVNRT (60%)

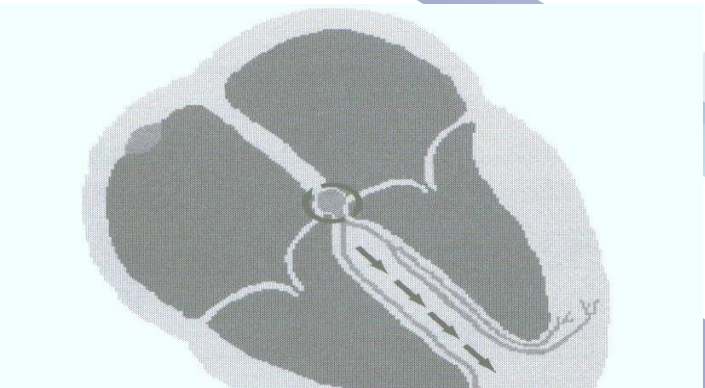
- AVRT (30%)

- Atrial tachycardia (10%)

Atrioventricular Nodal Re-entrant Tachycardia (AVNRT)

- AVNRT is due to re-entry in a circuit involving the AV node and its two right atrial input pathways one is fast and superior the other is slow and inferior this produces regular tachycardia with a rate of 120-240\min.Tends to occur in the absence of any structural disease.

- Episode may last from seconds to hours and the patient may experience:
- ✓ Rapid and very forceful beats
- ✓ Lightheadedness
- ✓ Breathlessness
- ✓ Polyuria (due to ANP release).
- ECG: tachycardia with normal QRS.
- Management: treatment not always necessary as these episode can be terminated easily by simple maneuvers (as mentioned before) in some resistant cases pharmacotherapy is indicated (refer to previous page for the specific agents)



Wolff–Parkinson–White syndrome + Atrioventricular Re-entrant Tachycardia (AVRT)

- An accessory conduction pathway from atria to ventricles through the bundle of Kent causes premature ventricular excitation because it lacks the delay seen in the AV node.
- May lead to a paroxysmal tachycardia, which can be produced by two possible mechanisms:

a**. Orthodromic reciprocating tachycardia**

  - The impulse travels through the AV node (anterograde limb) and depolarizes the ventricles. Then it travels back through the accessory pathway (the retro- grade limb) and redepolarizes the atria, creating a reentry loop.
  - No delta waves because conduction occurs retrograde over the accessory pathway.

b. Supraventricular tachycardias (AFib or atrial flutter)

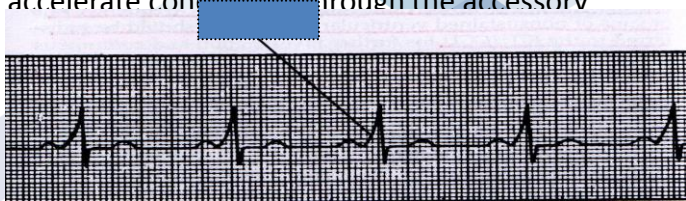
Usually, AV node only allows certain impulses to get to ventricles. With an accessory pathway, all or most of the impulses may pass to the ventricles. A fast ventricular rate may occur and cause hemodynamic compromise.

- **Diagnosis**

  - ECG: Narrow complex tachycardia, a short P-R interval, and a delta wave (upward deflection seen before the QRS complex)

- **Treatment**

- Radiofrequency catheter ablation of one arm of the reentrant loop (i.e., of the accessory pathway) is an effective treatment. Medical options include procain- amide or quinidine.
- Avoid drugs active on the AV node (e.g., digoxin, verapamil) because they may accelerate conduction through the accessory pathway. Type IA or IC antiarrhyth- mics are better choices.



Paroxysmal supraventricular tachycardia

- **Pathophysiology (most often due to reentry)**

  a. **AV nodal reentrant tachycardia**

  – Two pathways (one fast and the other slow) **within the AV node,** so the reentrant circuit is within the AV node
  – Most common cause of supraventricular tachyarrhythmia (SVT)
  – Initiated or terminated by PACs
  – ECG: **Narrow QRS complexes** with no discernible P waves (P waves are buried
  – within the QRS complex). This is because the circuit is short and conduction
  – is rapid, so impulses exit to activate atria and ventricles simultaneously.

  b. **Orthodromic AV reentrant tachycardia**

  - An accessory pathway between the atria and ventricles that conducts retrogradely
  - Called a "concealed bypass tract," and is a common cause of SVTs
  - Initiated or terminated by PACs or PVCs
  - ECG: **Narrow QRS complexes** with P waves which may or may not be dis-
  - cernible, depending on the rate. This is because the accessory pathway is at some distance from the AV node (reentrant circuit is longer), and there is a difference in the timing of activation of the atria and ventricles.

- **Causes**

  – Ischemic heart disease
  – Digoxin toxicity—paroxysmal atrial tachycardia with 2:1 block is the most
  – common arrhythmia associated with digoxin toxicity.
  – AV node reentry
  – Atrial flutter with rapid ventricular response
  – AV reciprocating tachycardia (accessory pathway)
  – Excessive caffeine or alcohol consumption

Paroxysmal supraventricular tachycardia

- **Treatment**
  - Maneuvers that stimulate the vagus delay AV conduction and thus block the reen- try mechanism: The Valsalva maneuver, carotid sinus massage, breath holding, and head immersion in cold water (or placing an ice bag to the face)
  - Acute treatment
    a. Pharmacologic therapy
  - • **IV adenosine—agent of choice** due to short duration of action and effective- ness in terminating SVTs; works by decreasing sinoatrial and AV nodal activity
- IV verapamil (calcium channel blocker) and IV esmolol (β-blocker) or digoxin are alternatives in patients with preserved left ventricular function.
- • DC cardioversion if drugs are not effective or if unstable; almost always successful

- **Prevention**

  a. **Pharmacologic therapy**
- Digoxin is usually the drug of choice.
- Verapamil or β-blockers are alternatives.

  b. **Radiofrequency catheter ablation of either the AV node or the accessory tract**
- (depending on which is the accessory pathway) is preferred if episodes are recurrent and symptomatic.

Ventricular tachyarrhythmias

Ventricular Tachycardia

-Defined as rapid and repetitive firing of three or more PVCs in a row, at a rate of between 100 and 250 bpm

Sustained versus nonsustained VT

a. Sustained VT (persists in the absence of intervention)

b. Nonsustained VT : Brief, self-limited runs of VT

Prognosis depends on the presence of heart disease and on whether VT is sustained or nonsustained.

-Clinical Features

Palpitations, dyspnea, lightheadedness, angina, impaired consciousness (syncope or near-syncope)

-Diagnosis

1.ECG: Wide and bizarre QRS complexes

2.QRS complexes may be monomorphic or polymorphic.

a. In monomorphic VT, all QRS complexes are identical.

b. In polymorphic VT, the QRS complexes are different.

3.Unlike PSVT, VT does not respond to vagal maneuvers or adenosine.

-Treatment

- ✓ If bp<90 mmhg → DC cardioversion
- ✓ If well tolerated → IV amiodarone
- ✓ Treat the underlying cause
- ✓ Automatic Implantable defibrillators
- ✓ Hypokalaemia, hypomagnesaemia, acidosis and hypoxaemia should be corrected.

Ventricular tachyarrhythmias

Ventricular ectopic beats

-QRS complexes in sinus rhythm are normally narrow because the ventricles are activated rapidly broad and bizarre because the ventricles are activated sequentially rather than simultaneously.
-Ectopic beats produce a low stroke volume because left ventricular contraction occurs before filling is complete.
-The pulse is therefore irregular, with weak or missed beats.
-Patients are usually asymptomatic.
-The significance of VEBs depends on the presence or absence of underlying heart disease.

- **Ventricular ectopic beats in otherwise healthy subjects**

-found in healthy people and their prevalence increases with age.
-are more prominent at rest and disappear with exercise.
-Treatment is not necessary, unless the patient is highly symptomatic, in which case β-blockers or, in some situations, catheter ablation can be used.

- **Ventricular ectopic beats associated with heart disease**

-Frequent VEBs often occur during acute MI but need no treatment.
-VEBs are common in patients with heart failure of any cause, including cardiomyopathy. While they are associated with an adverse prognosis.
-VEBs are also a feature of digoxin toxicity, and may occur as ‘escape beats’ in patients with bradycardia. Treatment is that of the underlying condition.

Ventricular tachyarrhythmias

Ventricular Fibrillation

- Multiple foci in the ventricles fire rapidly, leading to a chaotic quivering of the ventricles and no cardiac output.

-Clinical Features

Cannot measure BP; absent heart sounds and pulse

-Diagnosis

1. ECG: No atrial P waves can be identified.
2. No QRS complexes can be identified
3. In sum, no waves can be identified; there is a very irregular rhythm.

-Treatment

unsynchronized DC cardioversion→ three sequential shocks (assess the rhythm between each) →persists CPR, start epinephrine defibrillate 30-60 sec after

Most survivors have implantable defibrillator installed

MCQs

1. What is a third degree block ?

A. Atrial impulses fail to reach the ventricles as a result of complete AV node Block
B. Dissociation of atrial and ventricular activity as a result of complete block at the AV node
C. PR interval < 200 milliseconds as a result of complete block at the AV node
D. PR interval > 200 milliseconds as a result of complete block at the AV node

2. What is the best question to ask to determine whether a patient has an SVT with aberrancy or VT?

A. Have you had a heart attack?" If the answer is "yes," then the rhythm is a SVT; if the answer is "no," consider VT
B. Have you had a heart attack?" If the answer is "yes," then the rhythm is a VT; if the answer is "no," consider SVT
C. Have you had a AFib?" If the answer is "yes," then the rhythm is a VT; if the answer is "no," consider SVT
D. Have you had a AFib?" If the answer is "yes," then the rhythm is a SVT; if the answer is "no," consider VT

MCQs

3. How do you treat AF in a patient with hypotension and dyspnea?

A. Calcium-channel blockers, Beta-adrenergic blockers
B. Only amiodarone
C. Amiodarone Beta-adrenergic blockers
D. Direct electrical cardioversion

4. A 58 years old woman is admitted to the hospital with an acute myocardial infraction. On the second hospital day she develops sustained ventricular tachycardia even though she is on aspirin, heparin, lisinopril, and metoprolol.

What is the most appropriate next step in management?

A. Increase the dose of metoprolol
B. Add diltiazem
C. Angiography for angioplasty or bypass
D. Implantable defibrillator

MCQs

5. Which of the following tests would you do for this patient to determine a risk of recurrence ?

A. EP studies
B. Ecocardiography
C. MUGA scan
D. Ventilation/perfusion scan

6. 73 years old man has his third syncopal episode in the last 6 months. An EKG done in the filed shows ventricular tachycardia. His stress test is normal.

What is the most appropriate next step in management this patient ?

A. Metoprolol
B. Diltiazme
C. Angiography
D. Implantable defibrillator

MCQs

7. A 46 years old man has intermittent episode of palpitations, lightheadedness, and near-syncope. His EKG is normal. The echo shows an ejection fraction of 42% Holter monitor shows several runs of wide complex tachycardia lasting 5 to 10 seconds. Which of the following is most likely to benefit this patient ?

A. Pacemaker placement
B. Digoxin
C. Warfarin
D. EP studies

8. What medication used to treat atrial fibrillation should be avoided in patients with WPW syndrome?

A. Beta blockers
B. Calcium-channel blockers
C. Digoxin
D. B+C

Answers: 1-B 2-B 3-D 4-C 5-B 6-D 7-D 8-D

DONE BY

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Medicine is a science of uncertainty and an art of probability