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Arrhythmias

★ 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.

★ Resources used for this lecture:

Slides, Davidson's, Kumar, Step-up, Kaplan CK, DR. HERSI CLASS NOTES

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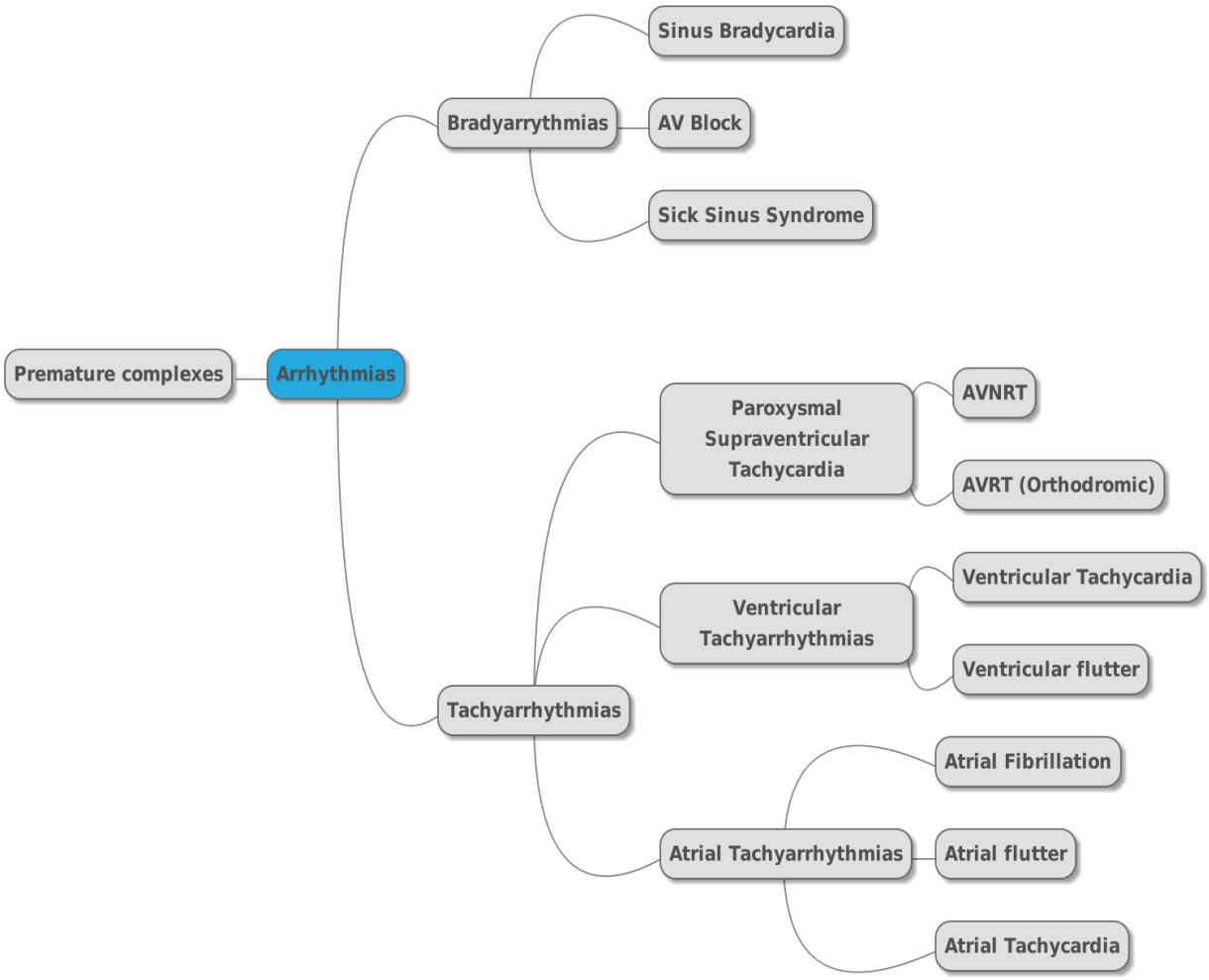
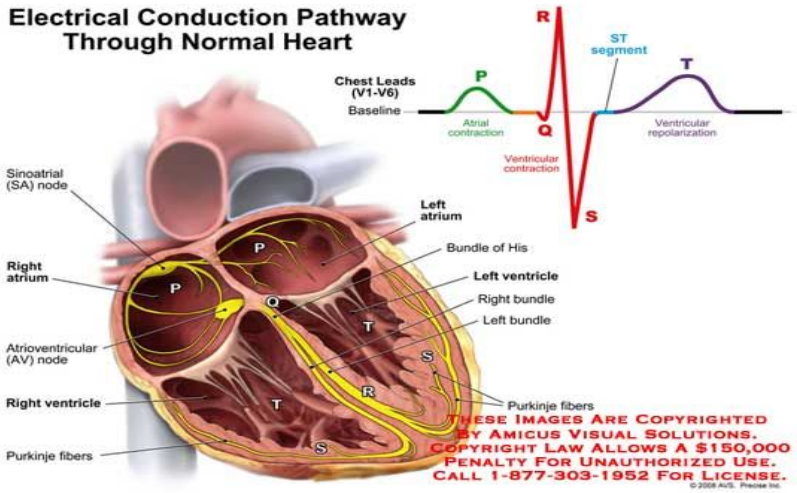
Our lecture is mainly about Atrial fibrillation, but in our teamwork we mentioned the other types for the sake of completion.. **so your main focus should be on Afib!**
If you don't have time just jump to page 8 where Afib is.



Introduction

Normal heartbeat is initiated by an electrical discharge from the Sinoatrial node (SA) passess through the atrium to the Atrioventricular node (AV) then to the ventricles . The Atria and ventricle depolarize sequentially as the electrical charge passes through them.

So Sinus node acts as the pacemaker of the heart.



Arrhythmias

A disturbance in the electrical rhythm of the heart is called Arrhythmia or Dysrhythmia "is the true terminology". Arrhythmias are often caused by structural heart disease, but may also occur because of abnormal conduction or depolarization in an otherwise healthy heart.

Arrhythmias are classified based on the **origin** and **rate**.

Origin based classifications are :

1- Supraventricular "above the ventricle":

A.Sinus : Electrical charges originate in the sinus node (SA) .

B.Atrial : Electrical charges begin in the atrium from foci other than the normal SA node.

Junctional : Electrical charges originate from the Atrioventricular area.

2- Ventricular : Electrical charges originate from the ventricles .

Rate based classifications are :

1- Bradycardia : heart rate less than 60 beats/min.

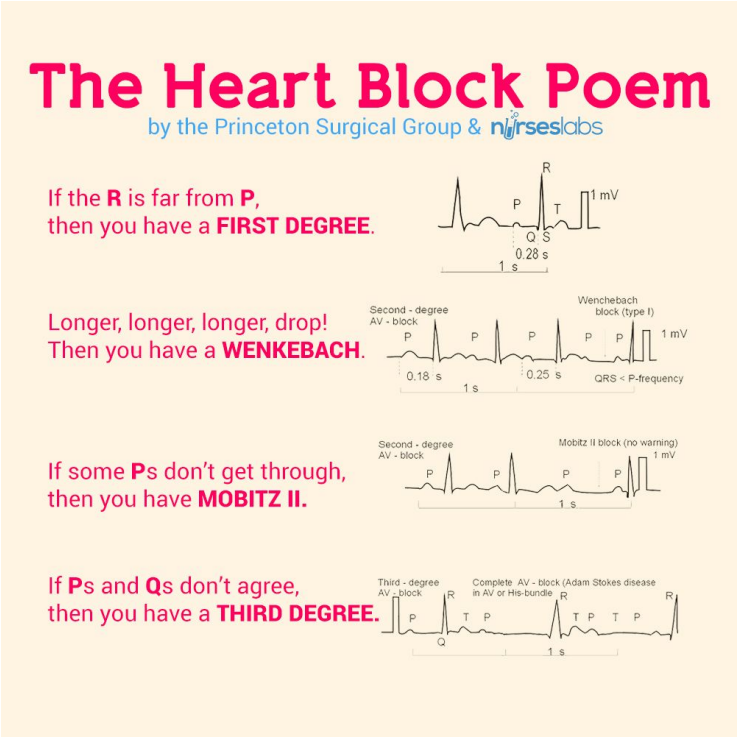
2- Tachycardia : heart rate more than 100 beats/min.

1- Bradyarrhythmias

Symptoms: -Could be asymptomatic-

Decrease of cardiac output leads to dizziness, cool extremities, shortness of breath, fainting, mental status alteration.

	Sinus Bradycardia	AV Block VIDEO			
		First degree	Second degree		Third degree
			Mobitz 1	Mobitz 2	
Characteristics	-Sinus rate *less than 60 bpm*. -Normal finding in *athletes*. -Side effect of antiarrhythmics.	Prolonged PR interval caused by **delay in the AV node.**	"Wenckebach" -PR interval prolongation till the **P wave fail to conduct.** -AV node block	1. P wave fail to conduct **without a preceding PR** prolongation. 2. **QRS suddenly drop.** -His-purkinje system block.	**-No correspondence** between P waves and QRS complex. -Complete block (AV dissociation)
Treatment	*Atropine* Blocking the vagal stimulation-	No treatment required.		Pacemaker.	



2- Tachyarrhythmias

Symptoms:

Decrease of cardiac output leads hypotension, syncope, blurred vision, dizziness shortness of breath.

- Chest pain/ Palpitations
- Anxiety / Nervousness

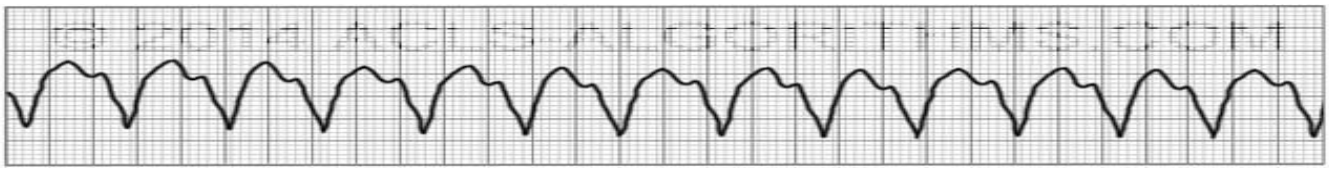
★ Ventricular Tachycardia

Rapid and repetitive firing of three or more ventricular contractions at rate of more than 120 bpm.

Less common than atrial but much more sever.

Causes: Commonly ischemic heart diseases.

ECG findings: (FAT & FAST) Wide and bizarre QRS complexes.



Treatment:

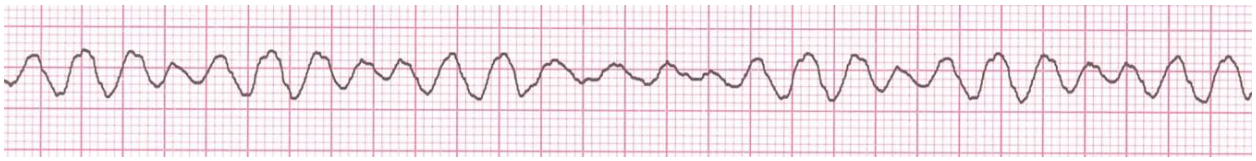
- ✓ **Stable patient** : IV Amiodarone, Lidocaine or Procainamide.
- ✓ **Unstable patient** "Chest pain, SOB, Hypotension and confusion": Synchronized cardioversion.

★ Ventricular Fibrillation

Multiple foci in the ventricles fire rapidly and usually preceded by ventricular tachycardia.

- BP unmeasurable / absent heart sounds
- Unconscious patient
- Serious case if not treated leads to sudden death.

ECG findings: No P waves identified + No QRS Identified



Treatment:

Immediate defibrillation and CPR, **if persisten**t IV epinephrine or vasopressin **If fails** antiarrhythmics.

Implantable defibrillator when cardioversion is successful.

Atrial Tachycardia: Rarely associated with hemodynamic[1] compromise because cardiac output is largely dependent upon ventricular output.

Look for the following finding in the history to suggest an atrial arrhythmias :

- ✓ Palpitation
- ✓ Exercise intolerance or dyspnea
- ✓ Embolic stroke

The most likely diagnosis even before EKG is Atrial fibrillation, since it's the most common.

Let's go briefly over the least common atrial tachycardias first:

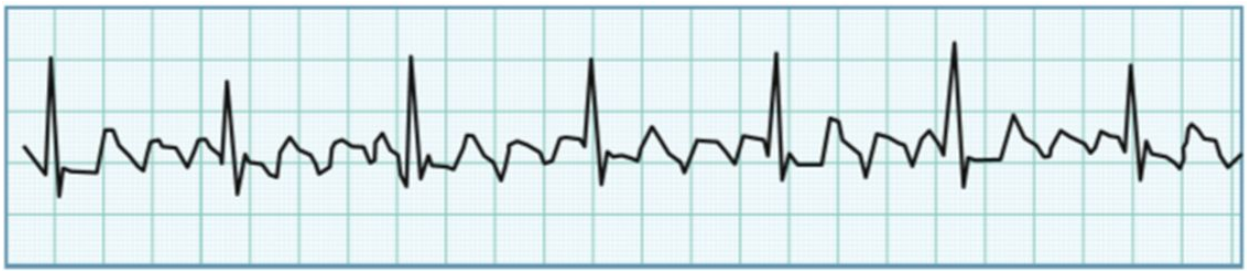
★ Atrial Flutter

One irritable automaticity focus in the atria fires regularly at about 250-350 bpm, and the long refractory period in the AV node allows only one out of every two or three atrial flutter waves to conduct to the ventricles, so the ventricular rate is 125-150 bpm.

Causes:

- ✓ Heart disease (heart failure commonly)
- ✓ COPD
- ✓ Atrial septal defect

ECG findings: Saw-tooth baseline (QRS complex after 2 or 3 p waves)



Treatment : Same as Atrial Fibrillation.

- ✓ **Hemodynamically unstable patient:** *Synchronized cardioversion* ***(shock)***
- ✓ **Hemodynamically stable patient**: Rate control - Elective cardioversion-, Anticoagulation (depending on CHAD VAS score)- Refer for Ablation[2].

[1] Relating to the flow of blood within the organs and tissues of the body.

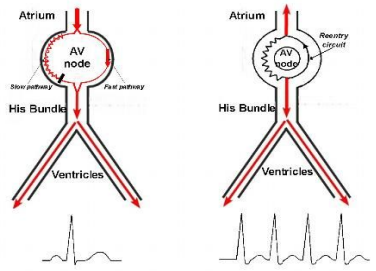
[2] During the procedure, small wires called electrodes are placed inside your heart to measure your heart's electrical activity. When the source of the problem is found, the tissue causing the problem is destroyed.To prevent the abnormal rhythms from moving through the heart.

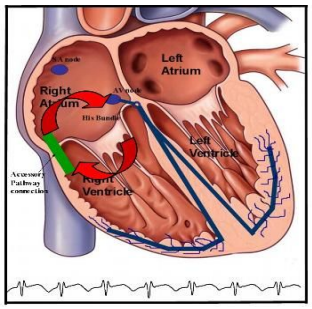
★ Paroxysmal Supraventricular Tachycardia

Arrhythmia caused by re-entry of the impulses with abrupt onset and termination, divided to two types:

	AV Nodal Reentrant Tachycardia	Orthodromic AV Reentrant Tachycardia
Characteristics	-60%, most common. -Two pathways within the AV node. (one fast the other slow)	-30% -An accessory pathway called a "concealed bypass tract" conducts impulses *retrogradely* from the ventricles to the atria. Causes**:** Ischemia / Digoxin toxicity.
ECG findings	Narrow QRS + no discernible P waves	Narrow QRS + P wave which may be discernible or may not
Treatment	-Stimulate vagus delay by carotid sinus massage (or breath holding / head immersion in cold water/ valsalva maneuver) followed by IV Adenosine (act by decreasing SA and AV nodal activity). -If episodes are recurrent and symptomatic → ablation is preferred	

VIDEO





★ Wolff-Parkinson-White Syndrome

An accessory pathway of conduction from atria to ventricles through the bundle of kent. It may lead to paroxysmal supraventricular tachycardia alternating with ventricular tachycardia or atrial flutter and fibrillation**. Three things can make you recognise it:**

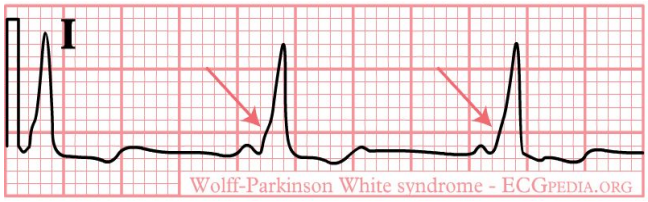
1- SVT alternating with ventricular tachycardia.

2- SVT get worse with digoxin or diltiazem.

3- Observing Delta wave on ECG.

ECG findings: Short PR interval **+ delta** waves (upward deflection before QRS) + wide QRS complex.

- Most accurate test is Electrophysiology studies (EP).

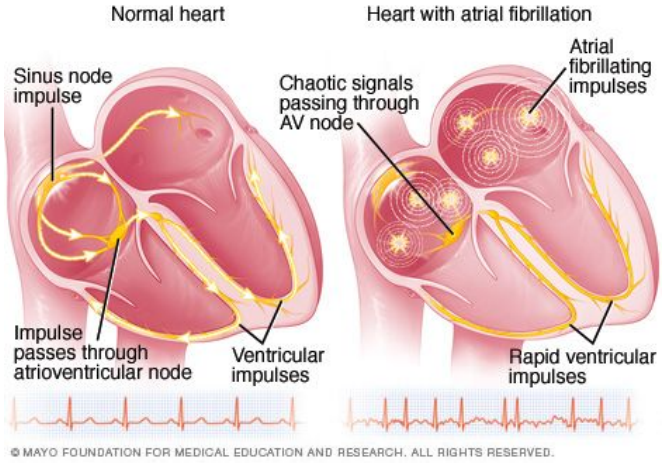


Treatment: Procainamide (Avoid digoxin / beta blockers / calcium channels blockers) or Radiofrequency catheter ablation.

★ Atrial fibrillation (AF):

Uncoordinated atrial activation with subsequent decline of atrial function.

- Very common ⅓ of cases of arrhythmias are AFib.
- Common in elderly above 50 years, always think of secondary causes "pulmonary or alcohol" if young.
- **AF** is associated with significant morbidity and a two-fold increase in mortality that are largely attributable to the effects of the *underlying heart disease and the risk of Thromboembolic events (most feared> cerebral embolism "stroke").*



Characterised by:

- Rapid (350-600 bpm) & irregular atrial rhythm.
- Reduced filling of the left and right ventricle.
- Cardiac output can be reduced. (causing unstable hemodynamic status)
- Conduction of most impulses from atria to ventricles is blocked at AV node.
- Contraction of ventricles can be: -either-

✓ Irregular and rapid (110-180 bpm) → tachycardia
✓ Irregular and slow (<50 bpm) → bradycardia
✓ Normal

Note: If the ventricles did response for 350-600 beats from atrium (SA) → no time for ventricular filling→ no Cardiac output→ no BP → collapse→ death. (That's why AV node is smart and always trying to compensate by blocking most impulses coming from the atrium)

The ventricular response to AF depends on to Atrial craziness depends on:
Electrophysiologic properties of AV node, the level of vagal and sympathetic tone, and the action of drugs.

So A fib is a supraventricular tachyarrhythmia characterized by uncoordinated atrial activation with subsequent decline of atrial function. On the **ECG**, there is replacement of consistent P waves by **fibrillatory waves** that vary in **size, shape, and timing** (350-600 beats/min), associated with an irregular, frequently rapid ventricular response **(irregularly,irregular)**

Mechanisms of the initiation of AF:

1. Ectopic beats, often arising from the pulmonary veins that triggers AF.
2. Re-entry within the atria maintains AF, with multiple interacting reentry circuits operating simultaneously.

AF causes remodelling: " AF begets AF" وحدة تولد وحدة	
Electrophysiological changes occur in the atria within a few hours of the onset of AF that tend to maintain fibrillation: *electrical remodelling*. (by shortening refractory period)	When AF persists for a period of months, *structural remodeling* occurs with atrial fibrosis and dilation that further **predispose** to AF.
• Many episodes of AF resolve spontaneously. • Over time AF tends to become persistent or permanent. *AF for a year is different than AF for a day, this dictates how to treat the patient.	

Common Causes of AF:

Cardiac Causes:	**Non-Cardiac Causes:**
• HTN • Ischemic heart disease • MI • Pericarditis • Pericardial trauma • Valvular heart disease: ○ Rheumatic: ✓ mitral stenosis ○ Non-rheumatic: ✓ Aortic stenosis ✓ Mitral regurgitation • Cardiomyopathy • Post-coronary bypass surgery	• **Pulmonary:** ○ COPD ○ Pneumonia ○ Pulmonary embolism • **Metabolic** • **Thyroid disease:** ○ Hyperthyroidism OR Hypothyroidism • **Electrolyte disorder** • **Toxic:** ○ **Alcohol** "Younger" • **Systemic illness:** ○ **Sepsis** ○ **Malignancy** ○ **DM**

Different classifications of AF

"Based on Time"				
First diagnosed	**Paroxysmal**	**Persistent**	**Long-standing persistent AF**	**Permanent**
-*Not* been diagnosed before -Irrespective of severity nor duration	-Most cases within 48 hours, may continue to 7 days. -*Self terminating*	-Prolonged episodes *>7 days* that can be terminated by electrical or chemical cardioversion.	-Continues AF lasting for *one year or more.* -When it's decided to adopt a rhythm strategy.	-More the one year and does not respond to cardioversion. -Paroxysmal AF will become permanent as the underlying disease process that disposes to AF **progress.**
	Note: About 50% of all patients with paroxysmal AF and 20% of patients with persistent or permanent AF have structurally normal hearts; this is known as **'lone atrial fibrillation'.**			

"Based on the Cause"		
Lone OR Primary	**Secondary**	**Non-valvular**
AF without clinical/ECG evidence of cardiopulmonary disease	Associated with cardiopulmonary disease.	Not associated with damage to the heart valves.

Patient that has valvular disease should automatically be given ⟶ Anticoagulants.

Diagnosis of AF:

- Signs and symptoms: (elderly with SOB, dizziness and palpitations consider AF)

Symptoms vary according to: irregularity / rate of ventricular response, functional status, AF duration, patient factors, and comorbidities..

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

- ECG findings:

Irregularly irregular rhythm, no identifiable P waves there is replacement of consistent P wave by fibrillatory waves that vary in size, shape, and timing (350-600 beats/min), associated with an irregular, frequently rapid ventricular response. "AF ECG may show-Left ventricular hypertrophy, Pre-excitation, Bundle branch block or Prior MI"

	 **Atrial Fibrillation**	 **Normal**
Heart rate	Tachyarrhythmia	Normal
Rhythm	Irregular	Regular
P wave	No P wave	P waves
Baseline	Irregular basline	Steady Base line

Investigations of AF:

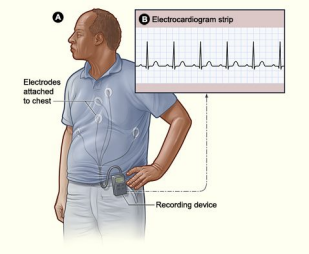
- **Transthoracic Echocardiography** (To rule out cardiac diseases)

Used to identify: Size and functioning of atria and ventricles,Ventricle hypertrophy, Pericardial disease and Valvular heart disease.

Note: it's the only way to be 99% sure that there is no clots in (LAA)

- **Laboratory tests** Important parameters to assess include:

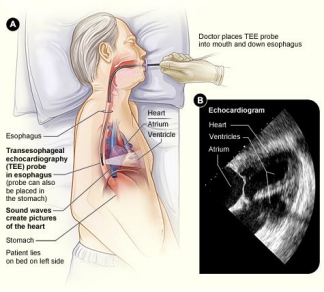
Thyroid function,Renal function, Hepatic function, Serum electrolytes and Complete blood coun.



- **Holter monitoring** (Portable ECG device "recorder"):

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 and Assessing response to treatment.



- **Transoesophageal echocardiography** (for Selected patients):

useful for: Accurate assessment of risk of stroke), Sensitive detection of atrial thrombi> (Particularly the *left atrial appendage*, as it is the most common site of thrombi in patients with AF)

- **Exercise testing.**
- **Chest radiography:** allows evaluation of the lung parenchyma and identifies coexisting lung disease.

Management of AF

Two general approaches are used for managing AF:

(1) Ventricular **rate control.** (considered alone for patients with little symptoms of arrhythmias)

(2) **Rhythm control.** (for patients who are experiencing consequences of arrhythmias.. ex.SOB,HF.)

\+ **Anticoagulation** to reduce the risk of stroke in patients with AF >>**The CHA2DS2VASc Index**

Treatment options for AF Depending on the condition of the patient we will treat:

1- Acute AF: A) Hemodynamically **unstable** patient → immediate electrical cardioversion to sinus rhythm.

Note: All patients who go for cardioversion "regardless of the case" should take anticoagulants, why? مو الواحد أول مايقوم من النوم يكون مفجوع، ويجلس فترة على ما يستوعب ويمارس نشاطه طبيعي Atrial is not mechanically functioning > Blood stasis > Thrombus formations.

B) Hemodynamically **stable** patient: Three things you have to achieve.

Prevention of Thromboembolism "Stroke" (Depending on CHADVASc ⋆)	**Control of Heart Rate**	**Maintenance of Sinus Rhythm** (after rate control is achieved)
AF present>48 hours, risk of embolization during cardioversion is significant. Anticoagulate patient for 3 weeks **before** and 4 weeks **after** cardioversion -To avoid waiting 3 weeks for anticoagulation, obtain a TEE to image the left atrium, if no thrombus is present, start IV heparin and perform cardioversion within 24 hours -An INR of 2 to 3 is the anticoagulation range. less than 2 indicate clot and more than 3 indicate bleeding	-Determine the pulse in a patient with AF. if its too rapid, it must be treated .(target rate is 60 to 100 bpm)	Use pharmacological cardioversion only if electrical cardioversion fails or is not feasible.
Pharmaceutical: •*Warfarin*[3] (INR must be checked regularly) •*Aspirin* (LEAST effective, but has no interaction, no INR) •*Dabigatran,Apixaban* (no interaction, no INR) •*Rivaroxaban* *Last 3 drugs are mostly used now..	**Pharmaceutical:** • *Ca^{2+}-channel blockers* In asthmatics •*B-blockers* In CAD •*Digoxin* (if BP is very low and cannot be tolerated at rest last choice)	**Pharmaceutical:** most effective when initiated (within 7 days of AF onset). •Antiarrhythmic drugs – Class IA / IC – Class III: e.g. *Amiodarone* Best effective, but not first line due to complications. It can be given to elderly •*Flecainide* •*Propafenone* Safer, less side effect, given to Youngers
Non-Pharmaceutical: •Removal/isolation of left atrial appendage e.g. WATCHMAN® device or surgery	**Non-Pharmaceutical:** •Ablate/Pacing.	**Non-Pharmaceutical:** •Ablation •Surgery (MAZE) **-Electrical shocks** Direct-current cardioversion

2- Chronic AF:

A) Rate control: With •*Beta-blockers* or • *Ca^{2+}-channel blockers* •*Digoxin* if BP is low.

B) Anticoagulation (use CHADSVASc score)

- Patient with 'lone' AF under age 60 > no need for anticoagulation therapy(low risk of emboli).
- Treat all other patients with chronic anticoagulation (warfarin).

[3] Warfarin inhibits the vitamin K-dependent synthesis of biologically active forms of the calcium-dependent clotting **factors II, VII, IX and X**, as well as the regulatory factors protein C, protein S, and protein Z. it is contraindicated for pregnancy and has a high food-drug interaction (green leaves كراث)

⋆ Prevention of Thromboembolism "Stroke" cont.

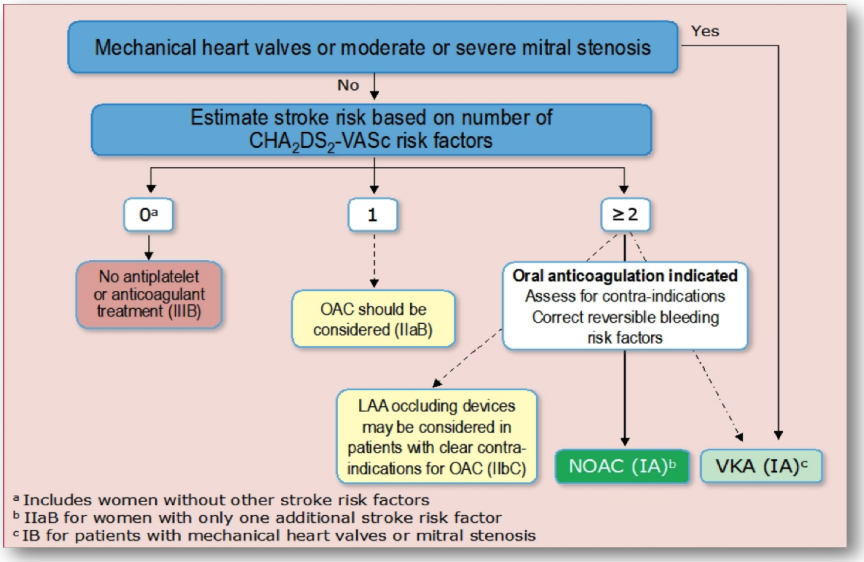
When to know your patient should or shouldn't take antithrombotics?

By using Stroke Risk Score for Atrial Fibrillation: **The CHA2DS2-VASc Index**

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 **c**ategory (female)	**1**

> 2 : Moderate-High risk "Give anticoagulant Give Warfarin"

0-1 : Low risk " No anticoagulant"



Example:

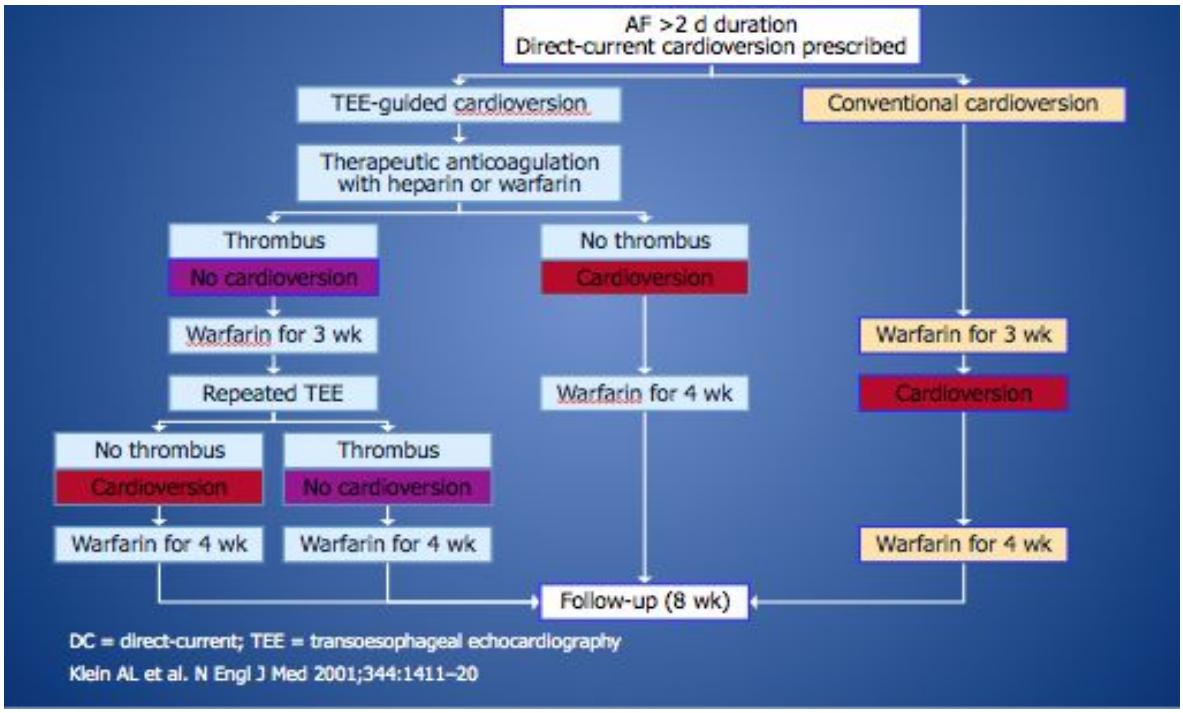
***78* year old *female* with history of *DM* and *HTN*.**

calculate her CHADSVASc score, and determine her mangement?

Age = 2 , Female = 1, DM= 1, HTN= 1 SO 2+1+1+1= **5**

Her score is 5, so she's high risk, we give Warfarin

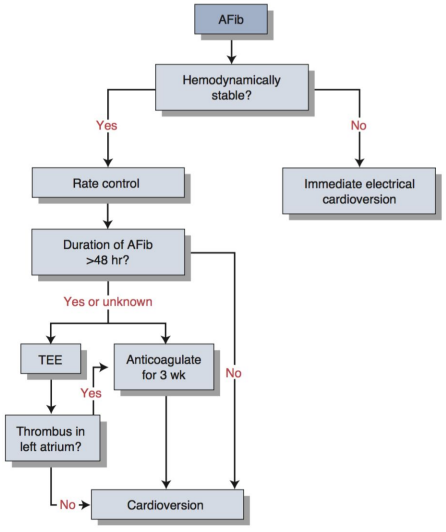
TEE-guided cardioversion: acute study design



Difference between Cardioversion and Defibrillation :

Cardioversion	Defibrillation
Delivery of a shock ***in synchrony*** with QRS complex to terminate dysrhythmias.	Delivery of a shock ***not in synchrony*** with QRS complex, to convert dysrhythmias to normal sinus rhythm.

Summary of the management plan in Acute AF:

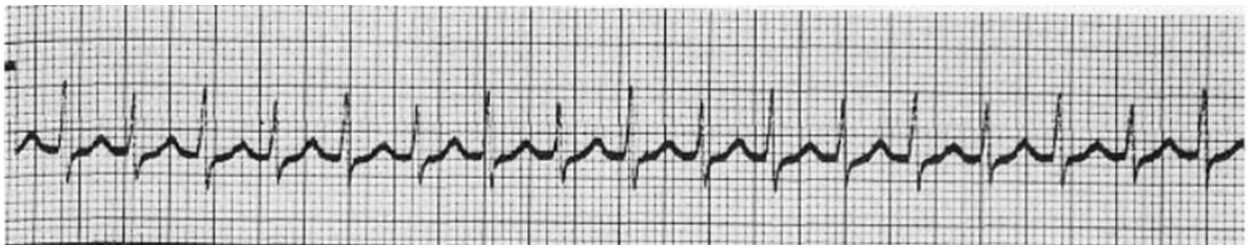


MCQs

1- An active 78-year-old female has been followed for hypertension
but presents with new onset of mild left hemiparesis and the finding of atrial fibrillation on ECG.
She had been in sinus rhythm six months earlier. Optimal treatment
by hospital discharge includes anti-hypertensives plus :

a. Close observation
b. Permanent pacemaker
c. Aspirin
d. Warfarin (Coumadin)
e. Subcutaneous heparin

2- A 36-year-old white female nurse comes to the ER due to a sensation
of fast heart rate, slight dizziness, and vague chest fullness. The following
rhythm strip is obtained which shows :

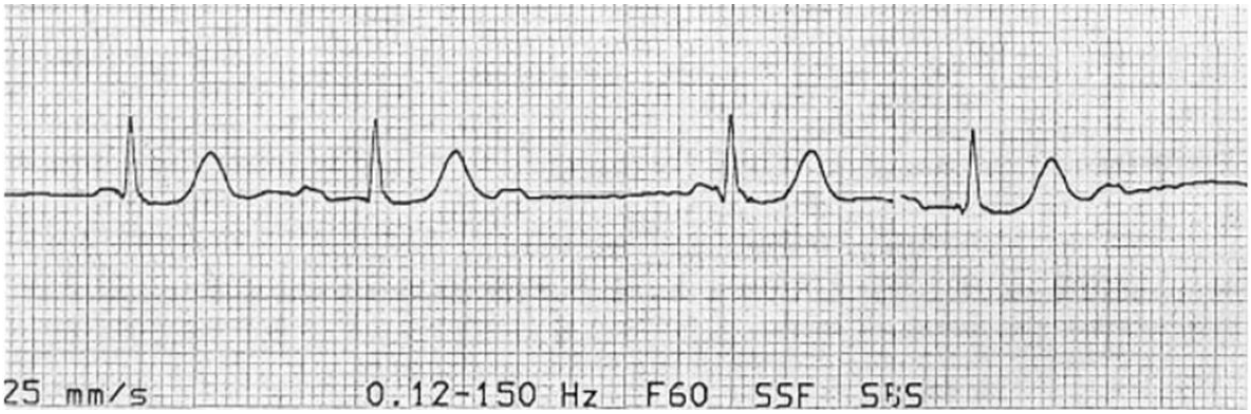


a. Atrial fibrillation
b. Atrial flutter
c. Supraventricular tachycardia
d. Ventricular tachycardia

3- The initial therapy of choice in this stable patient is :

a. Adenosine 6 mg rapid IV bolus
b. Verapamil 2.5 to 5 mg IV over 1 to 2 minutes
c. Diltiazem 0.25 mg/kg IV over 2 minutes
d. Digoxin 0.5 mg IV slowly
e. Lidocaine 1.5 mg/kg IV bolus
f. Electrical cardioversion at 50 joules

4- A 65-year-old man with diabetes, on an oral hypoglycemic, presents to the ER with a sports-related right shoulder injury. His heart rate was noted to be irregular and the following ECG obtained. The best immediate therapy is :



A. Atropine

B. Isoproterenol

C. Pacemaker

D. Electrical cardioversion

E. Digoxin

F. Diltiazem

G. Observation

5- In the ICU, a patient suddenly becomes unresponsive, pulseless, and hypotensive, with cardiac monitor indicating ventricular tachycardia. The crash cart is immediately available. The first therapeutic step should be

A. A precordial thump

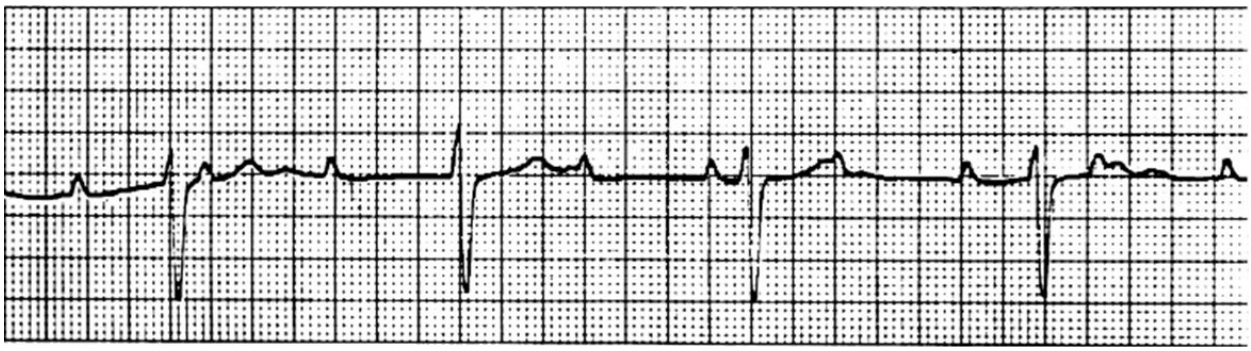
B. Lidocaine 1.5 mg/kg IV push

C. Epinephrine 1 mg IV push

D. Defibrillation with 200 joules

E. Defibrillation with 360 joules

6- A patient has been in the cardiac care unit with an acute anterior myocardial infarction. He develops the abnormal rhythm shown below. You would :



A. Give digoxin

B. Consult for pacemaker

C. Perform cardioversion

D. Obtain digoxin level

E. Give lidocaine

Q: What's the difference between Atrial flutter and fibrillation?

- Flutter is regular rhythm whereas fibrillation irregular.
- Flutter usually goes back into sinus rhythm or deteriorates into fibrillation

ANSWERS
1- D
2-C
3- A
4- G
5- D
6-B