Arrhythmia

Objectives:

- Epidemiology and Mechanisms of AF.
- Evaluation of AF patients.
- Classification of AF.
- Treatment and Risk stratification of AF.
- Identify other forms of Arrhythmia,

Team Members: Maha alghamdi , saqr altamimi , mshari alshbri , Laila mathkour

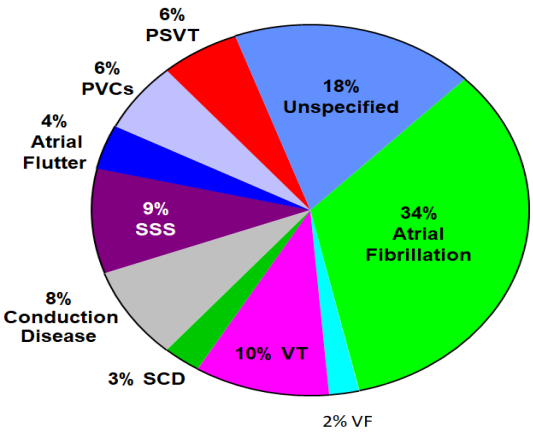
Team Leader: Haneen AlSubki

Revised By: Maha alghamdi

Resources: 435 team + Davidson + kumar + Recall questions step up to medicine.

- Editing file
- Feedback

Introduction



Atrial fibrillation accounts for **1/3** of all patient discharges with arrhythmia as principal diagnosis.

PSVT: Paroxysmal supraventricular tachycardia

PVCs: Premature ventricular contraction

SSS: Sick sinus syndrome

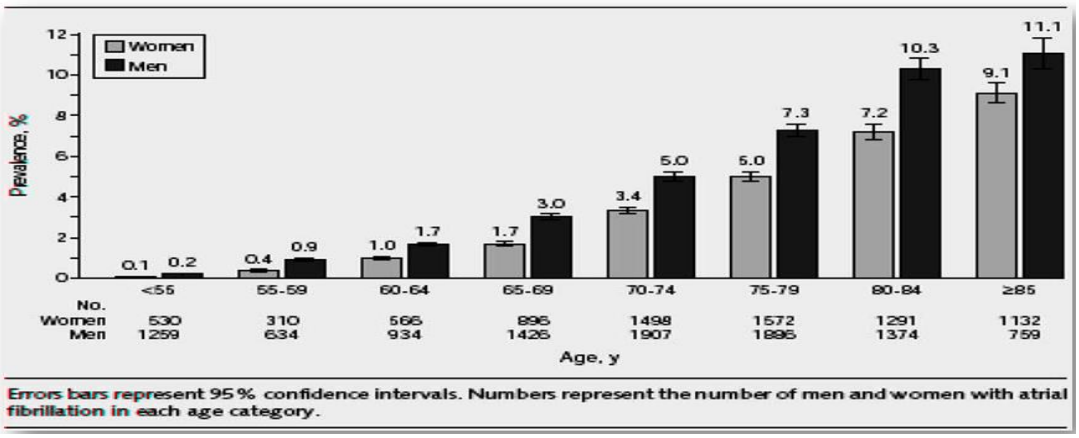
SCD: Sudden cardiac death

VT: Ventricular tachycardia

VF: ventricular fibrillation

AF Accounts for most of the arrhythmias so this lecture will focus more on AF.

AF Prevalence in US Population



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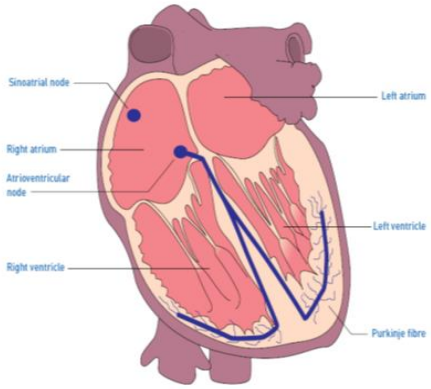
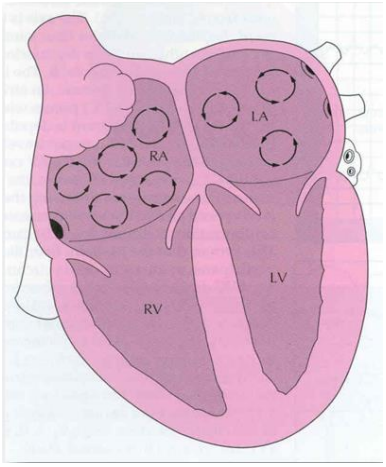
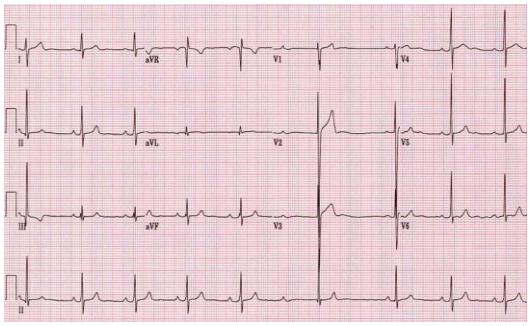
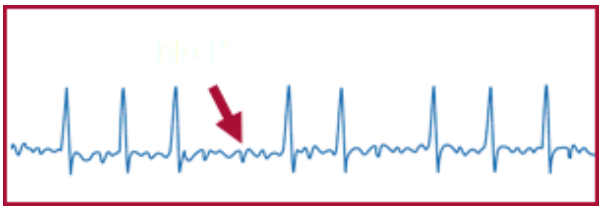
Note that it increases with age and females have less tendency .

- **Pathophysiology of Atrial Fibrillation and associated Stroke** [Watch Video!]

Normal Heart	AF
 **Normal regulation of heart rate and rhythm:** Contraction is controlled by the sinoatrial (SA) node	 In Af there will be a microcircuit, very chaotic, disorganised flickers.
 Every P (atrial contraction) wave is followed By QRS (ventricular contraction)	 2 Features that characterise AF: 1. Absent P wave. Disorganised, discrete waves. 2. The ventricular response is irregular meaning that QRS is irregularly irregular rhythm (chaotic and the distance between each QRS isn't equal) bc signals are coming from different places through AV node to the ventricle so the AV node gets really confused. 3. Absent a wave
we all know that the natural pacemaker of the heart is the SA node. What is the normal range of heart beat per minute? 60-100 beats per minute If it was lower than 60 it's called bradycardia. If it was over a 100 it's called tachycardia	**Normal heart rhythm is disrupted in AF:** 1. **AF is characterized by:** - Rapid (350–600 beats/min) and irregular atrial rhythm R-R Is fast. - Reduced filling of the left and right ventricles. 2. **Conduction of most impulses from the atria to ventricles is blocked at the AV node.** Not all the 350-600 beats will be delivered by AV node لأن عندها خاصية بحيث اذا حست ان فيه تو متش كندكشن رح يصير لها بلوك وتستقبل 100 بيت ماكسمم. لو استقبلت أكثر من كذا رح يموتوا المرضى **decremental conduction.** هذي الخاصية اسمها It will conduct conduct then it will stop to fill more.

	3. **Contraction of the ventricles can be:** - Irregular and rapid (110–180 beats/min; tachycardia) Ventricular rate can never go faster than 180. - Irregular and slow (<50 beats/min; bradycardia) - Normal 4. **Cardiac output can be reduced**.

- ## AF Begets (generates) AF

Atrial fibrillation gives rise to atrial fibrillation, How?

The longer you are in AF the more likely it becomes permanent.

It causes remodeling, both **electrical** and **structural**.

1. **Electrical remodeling** : Electrophysiological changes occur in the **atria** within a few hours of the onset of AF, that tend to maintain fibrillation. (By shortening the refractory period)
2. **Structural remodeling** : 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.

- ## Diagnosis of Atrial Fibrillation

As anything else you need to **be systematic.**

1.Take history 2.Do physical examination 4. Do some tests.

Clinical evaluation of patients with AF

- All patients
- History
- Physical examination
- **Electrocardiogram (ECG)**
- **Transthoracic echocardiogram (TTE)**
- **Blood tests** To check the thyroid.
- **Holter monitor**
- **Chest x-ray**

- Selected patients
  - Transesophageal echocardiogram (TEE)

1&2. History and physical examination: The doctor skipped this

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

In history you need to ask about the possible causes The doctor DID NOT skip this

Cardiac Causes	Non-Cardiac Causes

• Hypertensive heart disease
• Ischemic heart disease
• Valvular heart disease
  ○ Rheumatic: mitral stenosis
  ○ Non-rheumatic: aortic stenosis, mitralRegurgitation
• Pericarditis
• Cardiac tumors: atrial myxoma
• Sick sinus syndrome
• Cardiomyopathy
  ○ Hypertrophic
  ○ Idiopathic dilated (? cause vs. effect)
• Post-coronary bypass surgery
	• **Pulmonary** **Most common**
	  ○ COPD
	  ○ Pneumonia
	  ○ Pulmonary embolism
	• Metabolic
	  ○ Thyroid disease: hyperthyroidism
	  ○ Electrolyte disorder
	• Toxic: alcohol (‘holiday heart’ syndrome)
	
	Think of it as anything that excites the heart can lead to Afib as in alcohol consumption and thyroid diseases .

Heterogeneous “diverse“ clinical presentation of AF	

• 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.	

After knowing the causes we start asking about the **Signs and symptoms:** **The doctor DID NOT skip this**

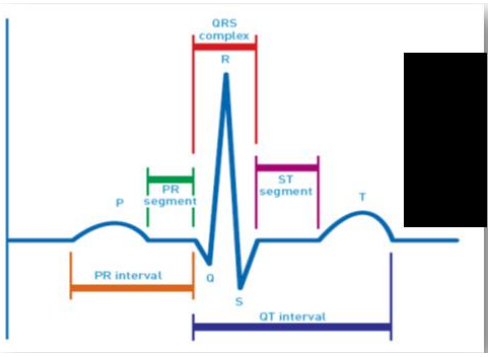
Cause	Signs and symptoms
• Irregular heart beat	- Irregularly irregular pulse - **Palpitations** Most of them.
• Decreased cardiac output	- **Fatigue** - Diminished exercise capacity - Breathlessness (dyspnoea) - Weakness (asthenia)
• Hypotension	- **Dizziness** and fainting (syncope)
• Cardiac ischaemia	- **Chest pain (angina)** Miss match and dec demsnd.
• Increased risk of clot formation	- **Thromboembolic TIA, stroke** Sometimes this will be the **1st** presentation and th pt come to the ER with a stroke

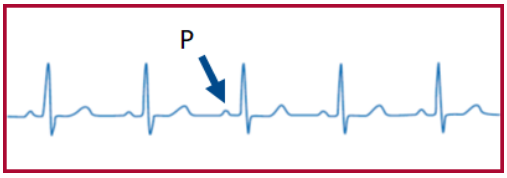
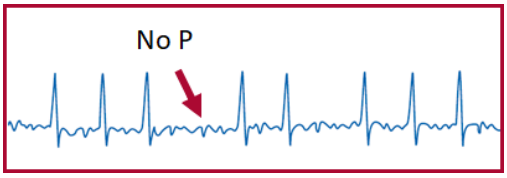
3. As for the tests which you do:

1. Electrocardiogram **The doctor skipped this test**

- 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



- **Normal sinus rhythm**	- **AF**
	
Normal heart rate Regular rhythm P Waves Steady baseline	Heart rate increased (tachyarrhythmia)* Irregular rhythm No P wave Irregular baseline

2. Transthoracic echocardiography (TTE)

- Non-invasive
- Used to identify
  - Size and functioning of atria and ventricles
  - Ventricle hypertrophy
  - Pericardial disease
  - Valvular heart disease

3. 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

4. Holter monitor

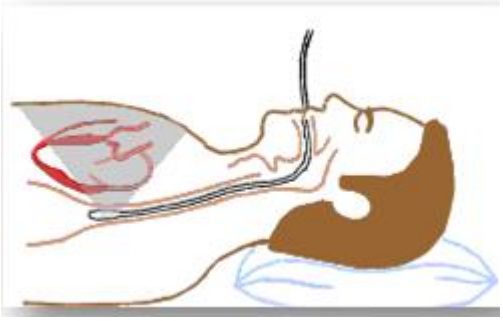
following up treatment and Diagnosis نركّب أسلاك على المريض ونوصلها بجهاز صغير ونقول له يروّح البيت فوائده

- 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

(which is a small device attached to the patient and tracks his ECG for 24 hours), in paroxysmal atrial fibrillation.**And it is the most accurate test.**

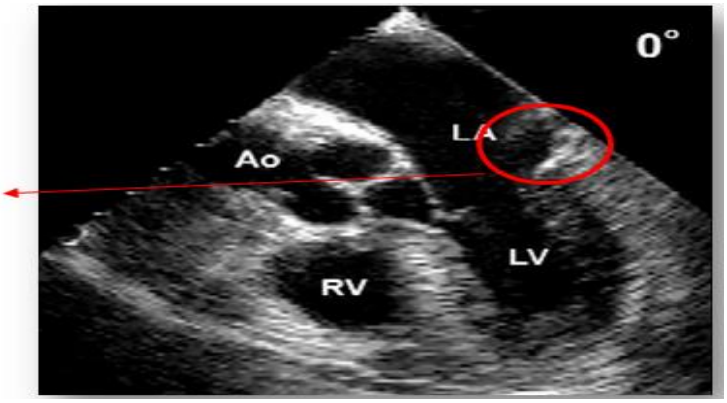
5. Transoesophageal echocardiogram (TEE): The only way to roll out stroke in the LA appendage!!

- 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 (By checking the heart from behind), the most common site of thrombi in patients with AF because it is trabeculated and the thrombi can easily form there .
- Not routinely used but useful for:
    - Accurate assessment of risk of stroke
    - Detection of low flow velocity ('smoke' effect)
    - Sensitive detection of atrial thrombi



When the atrium loses it contractility, it becomes insufficient. فالدم بيصير له ستاقنيشن ومارح يحصل complete emptying مما يؤدي الى تكون جلطات ممكن تروح لأي مكان آخر وتسبب جلطة (مثل الدماغ).

هذا النتوء موجود بال LA وهو اللي يحصل فيه الجلطة. اسمه auricle
It's a remnant of muscle trabeculated and not as smooth as the rest of the heart



6. 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

- **Summary for evaluation of AF patient**

APPENDIX 3. INITIAL CLINICAL EVALUATION IN PATIENTS WITH AF

Minimum Evaluation	
1. History and physical examination, to define	• Presence and nature of symptoms associated with AF • Clinical type of AF (paroxysmal, persistent, or permanent) • Onset of first symptomatic attack or date of discovery of AF • Frequency, duration, precipitating factors, and modes of initiation or termination of AF • Response to any pharmacological agents that have been administered • Presence of any underlying heart disease or reversible conditions (e.g., hyperthyroidism or alcohol consumption)
2. ECG, to identify	• Rhythm (verify AF) • LVH • P-wave duration and morphology or fibrillatory waves • Pre-excitation • Bundle-branch block • Prior MI • Other atrial arrhythmias • To measure and follow R-R, QRS, and QT intervals in conjunction with antiarrhythmic drug therapy
3. TTE, to identify	• VHD • LA and RA size • LV and RV size and function • Peak RV pressure (pulmonary hypertension) • LV hypertrophy • LA thrombus (low sensitivity) • Pericardial disease
4. Blood tests of thyroid, renal, and hepatic function	• For a first episode of AF • When ventricular rate is difficult to control
Additional Testing (1 or several tests may be necessary)	
1. 6-min walk test	• If adequacy of rate control is in question • If adequacy of rate control is in question
2. Exercise testing	• To reproduce exercise-induced AF • To exclude ischemia before treatment of selected patients with a type IC * antiarrhythmic drug
3. Holter or event monitoring	• If diagnosis of type of arrhythmia is in question • As a means of evaluating rate control
4. TEE	• To identify LA thrombus (in LAA) • To guide cardioversion
5. Electrophysiological study	• To clarify the mechanism of wide-QRS-complex tachycardia • To identify a predisposing arrhythmia such as atrial flutter or paroxysmal supraventricular tachycardia • To seek sites for curative AF ablation or AV conduction block/modification
6. Chest radiograph, to evaluate	• Lung parenchyma, when clinical findings suggest an abnormality • Pulmonary vasculature, when clinical findings suggest an abnormality

- **Classification of Atrial Fibrillation** (According to the duration)

First diagnosed	مازال عندنا أمل نشيل الAF			هنا نفقد الأمل والمريض يصير مصاب بشكل دائم
	Paroxysmal	**Persistent**	**Long-standing persistent AF**	**Permanent**
-Not been diagnosed before -Irrespective of severity and duration	-Most cases within 48 hours, may continue to 7 days. -Self terminating - Af that terminates spontaneously or with intervention within 7 D of onset.	-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. - When the pt and clinician make a joint decision to stop further attempts to restore and/or maintain sinus rh. - Acceptance of AF represent a

	- episodes may recure with variable frequency.	- Continuous AF that is sustained more than 7 D.	- Continuous AF more than 12 M.	therapeutic attitude on the part of the patient and clinician rather than inherent pathophysiological attribute of af. - Acceptance pt AF may change as symptoms, efficacy of the therapeutic interventions, and pt and clinician preference evolve. صعب ارجع الرذم حتى لو سوينا للمريض شوك رح يرجع الرذم حقه غلط.

- **classes of atrial fibrillation** (based on cause)

Lone OR Primary	Secondary	Non-valvular AF	Valvular AF
without clinical/ECG evidence of cardiopulmonary disease (which runs in families) يحصل في الناس اللي ماعندهم رسك ويكونوا بعمر صغير. So you have to role out 3 things: - Alcohol - Thyroid - Familial We treat it as transient attack.	AF associated with cardiopulmonary disease (e.g. myocardial infarction or pneumonia). Happens secondary to something else (stress) ex: Elderly pt with pneumonia who developed AF along with his condition. Or patient with thyrotoxicosis or Pt underwent a cholecystectomy.	AF that is not associated with damage to the heart valves (e.g. rheumatic mitral valve disease, prosthetic heart valve or mitral valve repair). Note that other Afibs which are caused by other valvular conditions (MR,AS,AR) are called non-valvular Afibs.	Any AF associated with **mitral stenosis and mechanical valve**. those pt have risk of stroke 10 times. Note that Mitral regurgitation pt are not considered valvular.

Treatment Atrial Fibrillation

- If patient presents with acute A fib---> patient is not hemodynamically stable---> electric cardioversion
- if the patient is not present with acute A fib--->patient is hemodynamically stable ---> need to achieve the following 3 domains:

  cardioversion وقبلها نكون مسوين maintenance therapy مب لازم اطبقها كلها على كل مريض وهذي تعتبر

1. Control heart rate
2. Maintain sinus rhythm
3. Prevent thromboembolism

	A. STROKE PREVENTION You need to assess the risk before - see next page-	**B. CONTROL OF HEART RATE**	**C. MAINTENANCE OF SINUS RHYTHM**
PHARMACOLOGIC	Warfarin has food-drug interaction , mostly food that contains vitamin K مثل الكراث . Aspirin Dabigatran Apixaban Rivaroxaban Those 3 underlined drugs are the new oral anticoagulants NOAC, which has less drug-drug and food-drug interactions. 1st line therapy	Ca2+-channel blockers B- blockers Digoxin	Antiarrhythmic drugs – Class IA – Class IC – Class III: e.g. amiodarone, dronedarone Best effective but it has so many side effects so It can be given to elderly only . because if you give it to a young man he will come after 10 years with tons of other PROBLEMS !
NON-PHARMACOLOGIC	Removal/isolation of left atrial appendage, e.g. WATCHMAN® device or surgery	Ablate/pace (نكوي المنطقة اللي مسوية المشكلة من الأساس) All three drugs have same effect, so how are you going to choose between them? Let's say your patient is also asthmatic > give calcium channels blockers (because beta blockers will exacerbate the asthma) What if he was hypotensive > give him digoxin (because calcium channel blockers exacerbate hypotension) And if you're patient is a known case of heart failure> digoxin or beta blockers are the best choice. And the list of diseases go on , so assess your patient's history well and choose the most suitable drug. And also give your patient blood thinners .	Ablation Surgery (MAZE) 1st thing we do is to restore sinus rhythm. المريض AF فلازم نرجع الرذم بعدين نعطيه هذي الدرقز عشان نحافظ على الرذم. If the pt is more than 60 years of age and AF we give him amiodarone but if the pt is young we give class 1 to avoid long term side effects and if it wasn't effective we do قسطرة كهربائية

A. Prevention of Thromboembolism

-The CHADS2 Index (OLD ONE NOT USED ANY MORE)

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	More than or equal 2	50-60
Low risk	0-1	40-50

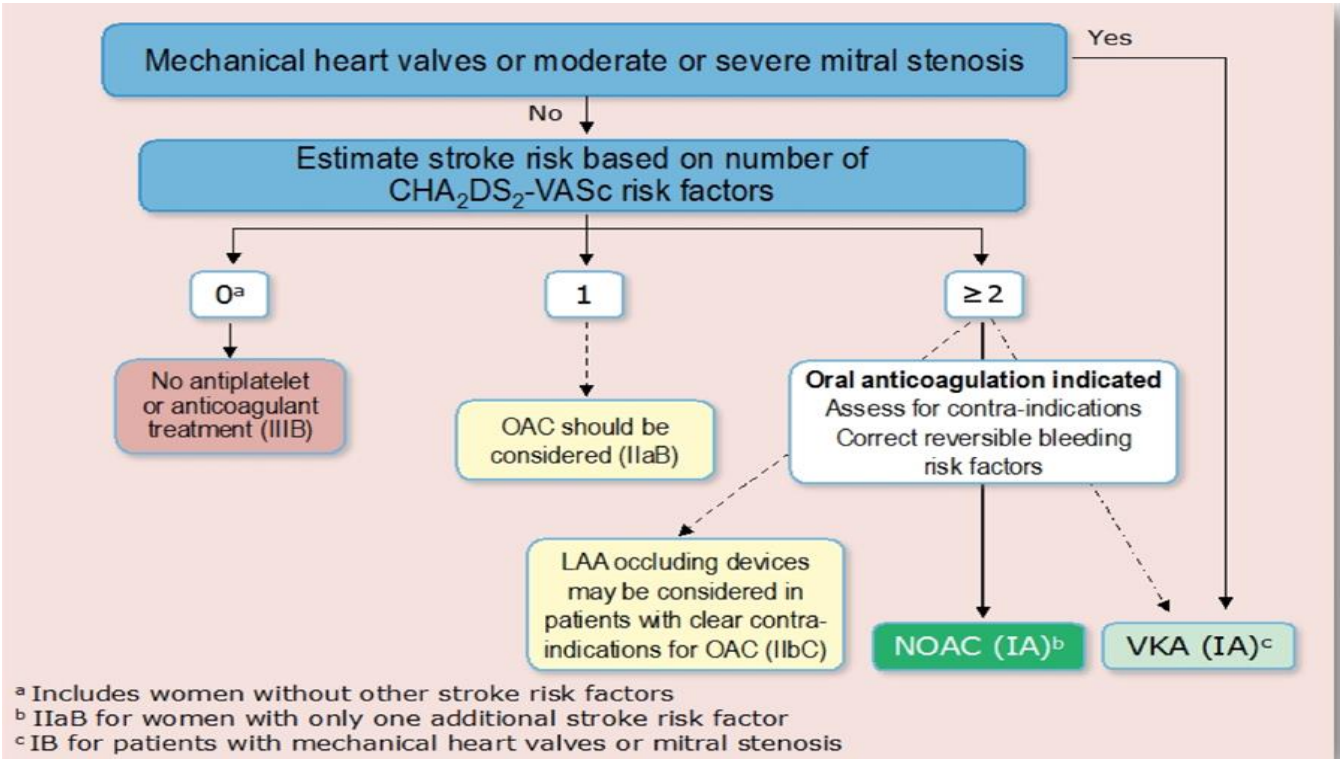
-The CHA2DS2VASc Index (NEW ONE)

Stroke Risk Score for Atrial Fibrillation

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

What is CHADVAS score for:

- Female + 55 Y + with DM = 2
- Male + 70 Y + DM +HTN = 3



If we get more than two points we give the patient anticoagulants, if less don't administer anticoagulants.

>>patients with score 0 or 1, can be given aspirin alone.

>>patients with score 2 or higher, must be on warfarin, or dabigatran, or rivoxaban.

When do you give the patient heparin instead of warfarin?

A - when there is an actual clot.

B- when the patient has a mechanical heart valve.

If a patient has a mechanical valve or (moderate or severe) mitral stenosis>

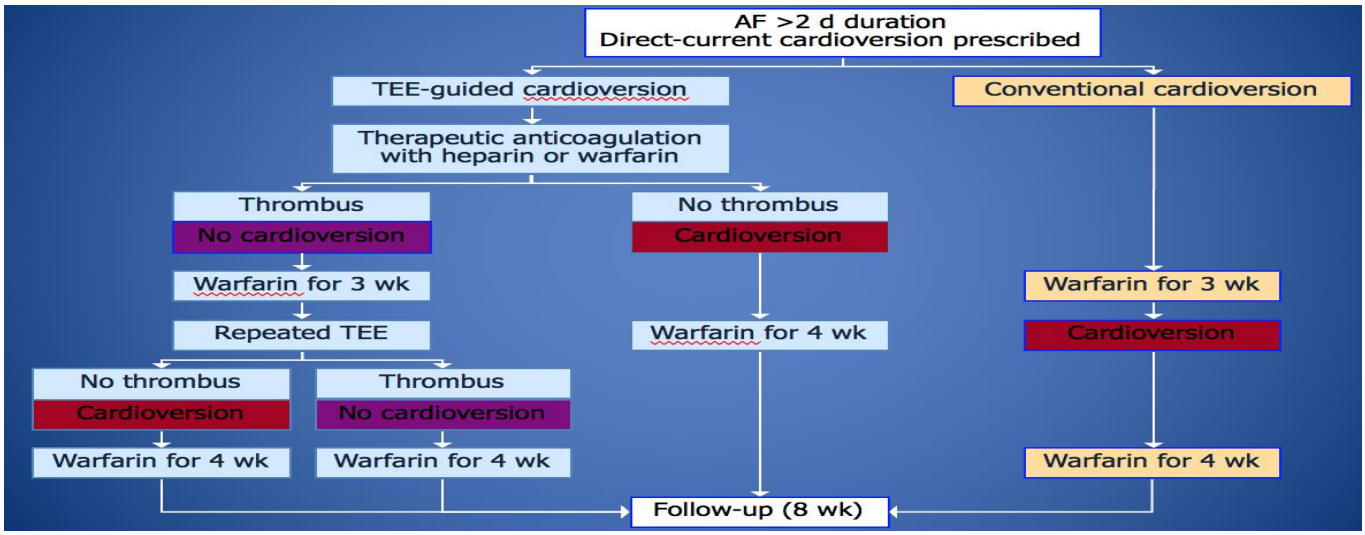
Skip the CHA2DS2 VASc score and go for therapy???? Because as we said they are at X10 risk to develop thrombi !!

B. Restoration of Sinus Rhythm

-with patient is hemodynamically unstable(which means he has: chest pain , dyspnea , hypotension , confusion) > Shock the patient (electric cardioversion)

The objective of rhythm-control therapy is to restore (cardioversion) and maintain normal sinus rhythm

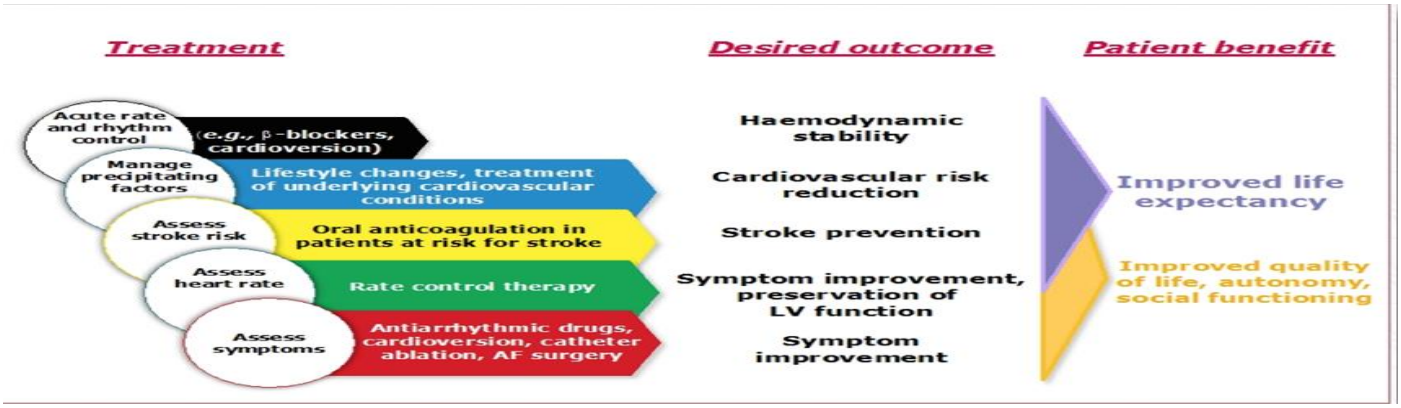
- Cardioversion can be achieved by:
    - Pharmacotherapy with antiarrhythmic agents Electrical shocks (direct-current cardioversion)
    - Direct-current cardioversion is generally more effective than pharmacotherapy
- Likelihood of successful cardioversion decreases with the duration of AF
    - Pharmacological 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 and after this 4 week you use the ChadsVas score to asses if the patient needs to continue on Anticoagulants or not !



أهم سؤال نسأله المريض (متى بدأ الخفقان) مع h cutoff 48 اذا كان أقل يعني نسوي شوك اما اذا اكثر من كذا بيكون فيه احتمالية تكوّن جلطة في LA واذا سويت شوك ممكن تتحرك الجلطة وتوصل الدماغ. Dont take the risk if the pt wasn't sure when it started with him/her لأنها وحدة من الأشياء اللي ما تقبل المخاطرة. او اقدر اسوي TEE عشان اتأكد, AND IF I FOUNd A CLOT we treat with anti-COAGULANT for 4 weeks then shock the pt.

After we shock the patient you have to put him on anti-coagulants irrespective to the Chads-vasc score !! And after this 4 weeks we assess the patient according to the Chads vasc score to decide if he has to stay on anti-coagulants or not .

- **Summary of treatment**

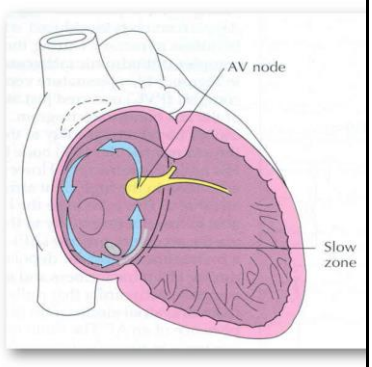


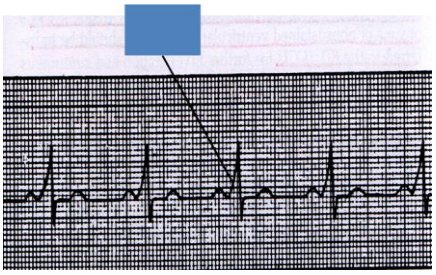
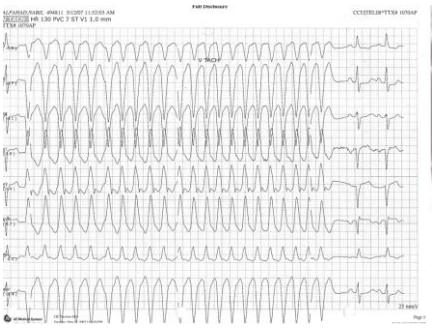
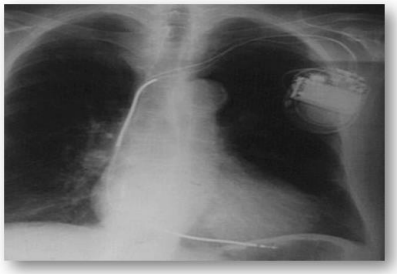
- **Consequences of AFib:**

Event	Association with AF
Death	Increased mortality, especially cardiovascular mortality due to sudden death, heart failure or stroke.
Stroke	20–30% of all strokes are due to AF. A growing number of patients with stroke are diagnosed with 'silent', paroxysmal AF.
Hospitalizations	10–40% of AF patients are hospitalized every year.
Quality of life	Quality of life is impaired in AF patients independent of other cardiovascular conditions.
Left ventricular dysfunction and heart failure	Left ventricular dysfunction is found in 20–30% of all AF patients. AF causes or aggravates LV dysfunction in many AF patients, while others have completely preserved LV function despite long-standing AF.
Cognitive decline and vascular dementia	Cognitive decline and vascular dementia can develop even in anticoagulated AF patients. Brain white matter lesions are more common in AF patients than in patients without AF.

Notes related to the scheduled: Stroke is the most devastating complication and the main corner stone treatment is how to prevent it.

Now we will talk about other types of arrhythmia which are:

Type of Arrhythmia	Notes	ECG	Treatment
Atrial Flutter	 so basically there is a point in the atria that fire abruptly flutter wave macro circuit , unlike Afib which is micro circuits. AV node will block some of the flutter waves ---> so on the ECG we will find can be caused by A- COPD B- atrial septal defect	**2 or 3 atrial flutter waves and one QRS complex.** **Classic Saw-tooth baseline** 	(just like A fib) -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 If patient is not hemodynamically unstable > synchronized cardioversion Hemodynamically stable patient > rate control, elective cardioversion, assess stroke risk (CHA2DS2 VASc score) **Ablation is the best treatment**
Supraventricular Tachycardia تسارع شديد يوصل 250-200 Watch video !	Arrhythmias of supraventricular origin using a re-entrant mechanism with abrupt onset & termination -**AVNRT** (60%):  -**AVRT** (30%):ex: Wolf-Parkinson-White (WPW) SyndromeArrhythmia caused by re-entry of the impulses with abrupt onset and termination, divided to 2 types: AV Nodal Reentrant Tachycardia 60%, most common. Two pathways within the AV node. (one fast the other slow) ECG : Narrow QRS + no discernible P waves	 -Wolf-Parkinson-White (WPW) 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. Observing Delta wave on ECG.	-Medical therapy - Radio Frequency Ablation

	Orthodromic AV Reentrant Tachycardia 30% An accessory pathway called a "concealed bypass tract" conducts impulses retrogradely from the ventricles to the atria. Causes: Ischemia / Digoxin toxicity On ECG : Narrow QRS + P wave which may be discernible or may not.  -Atrial tachycardia (10%)		
Ventricular Tachycardia	This condition is lethal In IHD pt and familial genelopathy. Treatment: - Address the underlying cause and treat it - Shock		-Treat the underlying cause -Automatic Implantable defibrillators 
Ventricular Fibrillation	Low amplitudes no avios QRS SO treat with shock immediately		

Great video: https://www.youtube.com/watch?v=36MT8hR-gDg

Summary

A cardiac arrhythmia is a disturbance of the electrical rhythm of the heart. Arrhythmias are often a manifestation of structural heart disease but may also occur because of abnormal conduction or depolarization in an otherwise healthy heart.

Arrhythmia (Based on origin)

1. Ventricular	Types:	Ventricular tachycardia	Ventricular Fibrillation	2. Supraventricular	Types:	(AVNRT)	(AVRT)
	Characteristics:	- Rapid and repetitive firing at rate. - Usually occurs in patients with CHD or Cardiomyopathies.	- Usually preceded by V tach. - Very rapid and irregular ventricular activation without mechanical effect. - Patient is pulseless, becomes unconscious, can get into cardiac arrest.		Pathophysiology:	- Two pathways within the AV node, so the reentrant circuit is within the AV node. - Most common cause of SVT. -Initiated or terminated by PACs.	- An accessory pathway between the atria and ventricles that conducts anterogradely or retrograde, called a (concealed bypass tract). - A common cause of SVTs. - Initiated or terminated by PACs or PVCs.
	Treatments:	In cases of emergency with a systolic BP less than 90 mmhg > DC cardioversion is required. But if the patient is stable give IV amiodarone.	- Immediate defibrillation and CPR, if persistent> IV epinephrine or vasopressin If fails > antiarrhythmic. -ICD when cardioversion is successful.		ECG:	Narrow QRS complexes with no discernible P Waves.	Narrow QRS complexes with P waves which may or may not be discernible , depending on the rate.
					Causes:	• Ischemic heart disease • Digoxin AV node reentry • Atrial flutter with rapid ventricular response • AV reciprocating tachycardia (accessory pathway) • Excessive caffeine or alcohol consumption	
					Prevention:	• Digoxin is usually the drug of choice. • Verapamil or β-blockers are alternatives. • Radiofrequency catheter ablation of either the AV node or the accessory tract	
					Treatment:	• IV adenosine: Only used in SVT and has a short duration of action. • CCB (IV Verapamil) and β-blocker (IV Esmolol) or Digoxin are appropriate in Patients with preserved Left Ventricular function • Direct Current Cardioversion	

Arrhythmia (Based on rate)

Atrial tachyarrhythmia's

1. Atrial fibrillation

Atrial fibrillation (AF) is the most common sustained cardiac arrhythmia with an overall prevalence of 0.5% in the adult population of the UK.

Characteristics:		1. abnormal automatic firing (350–600 beats/min) and irregular atrial rhythm 2. Reduced filling of the left and right ventricles 3. The presence of multiple interacting re-entry circuits looping around the atria.
Classification:	Based on time:	- First diagnosed: AF that has not been diagnosed before. - Paroxysmal: Intermittent episodes which self-terminate within 7 days. - Persistent: AF that lasts longer than 7 days including episodes that are terminated by cardioversion either by drugs or by direct current cardioversion. - Long-standing persistent AF: Continuous AF lasting for 1 year or more when it is decided to adopt a rhythm control strategy. - Permanent: More than one year and does not respond to cardioversion.
	Based on cause:	- Lone or primary: AF without clinical/ECG evidence of cardiopulmonary disease. - secondary: AF associated with cardiopulmonary disease. - Non-valvular: AF that is not associated with damage to the heart valves.
Causes:		- Cardiac & Non cardiac.
Diagnosis:		Sign & symptoms, ECG, TTE & TEE, Laboratory tests, Holter monitoring, Chest radiography.
Treatment and management:		1. Prevention of thromboembolism 2. Rate control 3. Restoration and maintenance of sinus rhythm

2. Atrial Flutter

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.

Pathophysiology:	- 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. - COPD. - ASD.
Diagnosis:	- ECG: ECG provides a saw-tooth baseline
Treatment:	Similar to treatment for AFib

3. Multifocal atrial tachycardia	Usually occurs in patients with severe pulmonary disease.	
	Diagnosis:	- ECG: Variable P wave morphology and variable PR and RR intervals. At least three different P wave morphologies are required to make an accurate diagnosis. - Can also be diagnosed by use of vagal maneuvers or adenosine to show AV block without disrupting the atrial tachycardia.
	Treatment:	Treatment involves improving oxygenation and ventilation (strong association between MAT and lung disease). If left ventricular function is preserved, acceptable treatments include calcium channel blockers, β-blockers, digoxin, amiodarone, IV flecainide, and IV propafenone. If LV function is not preserved, use digoxin, diltiazem, or amiodarone. Electrical cardioversion is ineffective and should not be used.

Arrhythmia (Based on rate)

Atrial Bradyarrhythmias

Sinus bradycardia	AV block				
1. Extrinsic factors that influence the normal SA node. 2. Intrinsic sinus node disease.	Degree:	First degree	Second degree		Third degree
			Mobitz type I	Mobitz type II	
	Characteristics:	- PR interval (>0.2ms). - rarely causes symptoms.	- Progressive PR prolongation and beat drop.	Constant PR prolongation and beat drop	Complete dissociation, ventricle and atria contract irrespective to each other.
	Treatments:	Nil		Pacemaker	

1.A 75 year old woman presents to her family physician with a 24-hour history of rapid, irregular palpitations accompanied by fatigue. In an elderly patient, what is the most likely cause of palpitations?

A. Atrial ectopic (premature) beats

B. Atrial fibrillation

C. Supraventricular tachycardia

D. Ventricular ectopic (premature) beats

2. A 72 year old hypertensive woman presents with a history of sudden-onset, rapid, irregular palpitation. She has had several episodes over the previous 3 months, which have resolved within 1 hour. She feels tired and slightly lightheaded during episodes. From this history, which of the following most likely explains her symptoms?

A. Atrial fibrillation

B. Sinus arrhythmia

C. Supraventricular tachycardia

D. Ventricular ectopic beats (extrasystoles)

3. Which of the following drugs is known to be effective in preventing stroke in patients with atrial fibrillation?

A. Amiodarone

B. Apixaban

C. Aspirin

D. blocker

4. A 69-year-old woman complains of intermittent palpitations, lasting several hours, which then stop spontaneously. She also suffers from asthma. Holter monitoring confirms paroxysmal atrial fibrillation. Which of the following statements is correct regarding the management of this patient?

A. Digoxin effectively prevents recurrence of the arrhythmia

B. Anticoagulation is not necessary

C. Sotalol may be effective

D. Flecainide orally may be an effective as-needed treatment to abort an attack

5. A sawtooth pattern on ECG suggests:

A. Atrial fibrillation

B. Atrial flutter

C. Tachycardia

D. WPW

7.Which of the following rhythms is NOT commonly associated with sick sinus syndrome (sinoatrial disease)?

A .Atrial fibrillation

B. Atrial tachycardia

C. Sinus bradycardia

D. Ventricular tachycardia

8. A 75 year old woman has a history of hypertension and diabetes. She presents with atrial fibrillation. What is her CHA2DS2-VASc score?

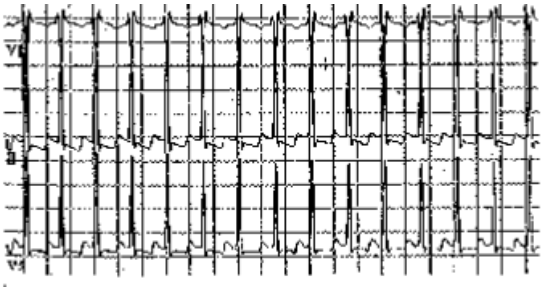
A. 2

B. 3

c. 4

D. 5

9. The EGG below shows a regular, narrow complex tachycardia in a patient presenting with sudden-onset, rapid palpitation. Which of the following should be used first in attempting to terminate this rhythm?



A. Direct current cardioversion

B. Intravenous B-blocker

C. Oral B-blocker

D.Vagal manoeuvres. e.g. Valsalva manoeuvre.

10. A 71 year old woman with a history of hypertension presents with fatigue and rapid, irregular palpitations. She normally takes enalapril for blood pressure control. Clinical examination reveals an irregularly irregular pulse, rate 125 beats/min, and BP 128/86 mmHg. Cardiovascular examination is otherwise normal. A 12-lead EGG is performed, which shows atrial fibrillation with poor ventricular rate control, but no other abnormality. Which of the following drugs is the most suitable agent to control heart rate in this patient?

A. Adenosine

B. Amiodarone

C. B-blocker

D. Flecainide

11. A 48 year old woman with no significant previous medical history collapses while running a marathon. Despite attempts at resuscitation, she does not survive. Postmortem exq.mination reveals asymmetric left ventricular hypertrophy with disproportionate thickening of the interventricular septum. A postmortem diagnosis of hypertrophic cardiomyopathy is made. What is the most likely cause of this patient's sudden collapse?

A. Atrial fibrillation

B. Left ventricular failure

G. Pulmonary embolism

D. Ventricular arrhythmia

Answers:

1. B 2. A 3. B 4. D 5. B 7.D 8.D 9.D 10.C 11.D