Medicine TEAM 437

MED437 KING SAUD UNIVERSITY

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

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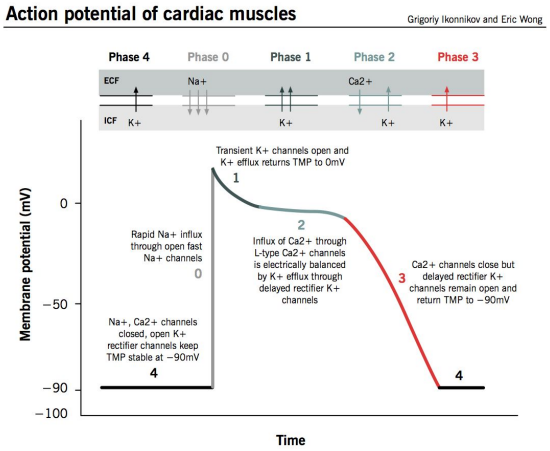
436 slides

Basic Sciences

Okay, so before we get into the arrhythmias, let's refresh our memories with some physiology:



So this is a cardiac muscle action potential:

Phase 0: Depolarization occurs through fast Na+ channels.

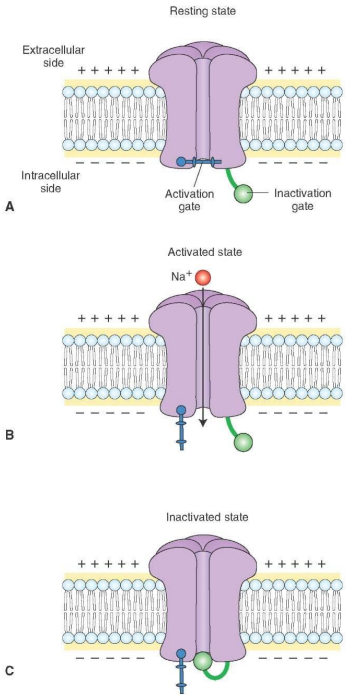
Phase 1: Na+ channels close and K+ starts leaving the cell, causing slight repolarization.

Phase 2: or better known as the "plateau phase", Ca+2 gets in the cell and K+ leaves the cell

Phase 3: K+ leaves the cell > restores resting membrane potential RMP

Phase 4: maintained by Na+/K+ ATPase channel.

As you can see Na+ channels are really important, so we would like to add a bit more information about them. Na+ channels actually have two gates (upper gate and lower gate) as shown in the picture below.



There are three states (shapes) of Na+ channels:

A. Closed state (during RMP), upper gate is closed
B. Open state (during Na+ flow), both gates are open
C. Inactive state (when depolarization continues the lower gate closes slowly)

So what's the significance of knowing this?

Closed gate is affected by class 1c antiarrhythmics ,

Open gate is affected by class 1a antiarrhythmics,

Inactive gate is affected by class 1b antiarrhythmics.

Knowing the fact that after an MI there is a risk of arrhythmia. What class of the mentioned antiarrhythmics would you use?

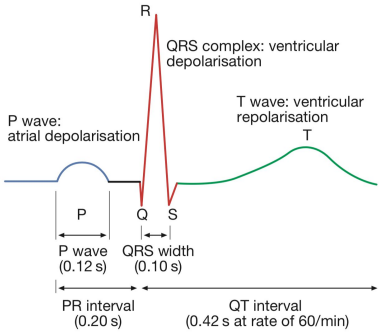
Since MI is due to hypoxia---> hypoxia leads to low activity of electron transport chain ETC---> low activity of ETC leads to low ATP---> which leads to a reduction in Na+/K+ channel---> which leads to accumulation of Na+ inside the cell> cell keeps on depolarizing---> thus increasing the amount of inactive Na+ channels (because as we've said as depolarization goes on Na+ channels turn into this state)---> So use class 1b to affect those channels and prevent the arrhythmia.(Lidocaine)

Let us remind you with what the waves on an EKG represent?

The P wave represents : atrial depolarization

The QRS complex represents : ventricular depolarization

The T wave represents: ventricular repolarization



Rhythm in the heart is controlled by the ANS by:

1. M2 receptor causes bradycardia
2. B1 receptor causes tachycardia

So 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.

So an athlete comes to the clinic. He has no symptoms, but he wants to have an annual check up. When the nurse took hit vitals. It was found that his heart rate was 55 beats per minute.

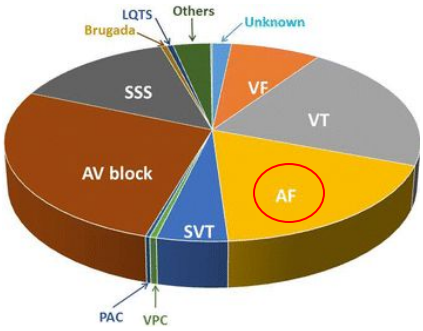
What are you going to do?

- He is asymptomatic -> do nothing.
- Athletes usually have sinus bradycardia, which is absolutely normal.
- If he says he has an underlying cause like: hypothyroidism, or takes some drugs(like B blockers),etc.. ---> then treat the underlying cause.
- If he was symptomatic give him Atropine,most initial management,(antimuscarinic > B1 receptor is unopposed---> increases sympathetic stimulation ---> increase HR)
- If atropine did not work ---> go for a pacemaker,most effective management.

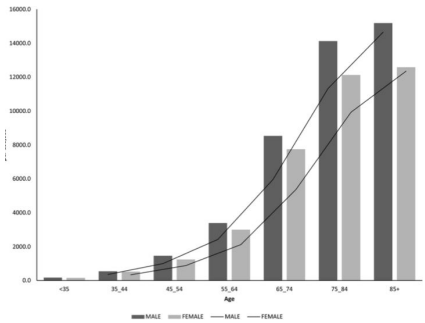
What would cause sinus tachycardia(>100 bpm) then?

usually represents a physiologic response to **fever, hypotension, volume depletion, anxiety, and pain.** Other causes include **thyrotoxicosis, anemia,** and some **drugs** >Treat the underlying cause.

Introduction



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

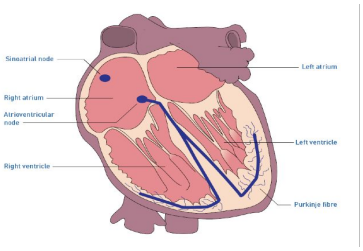
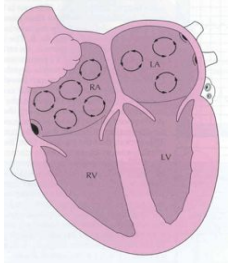


Note that it increases with age and females have less tendency.

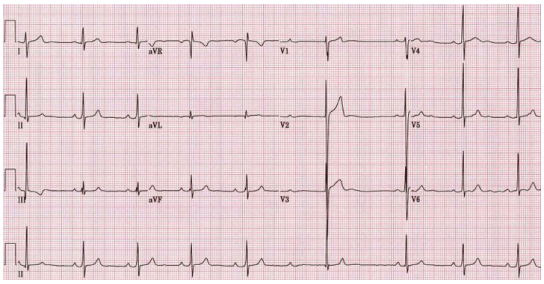
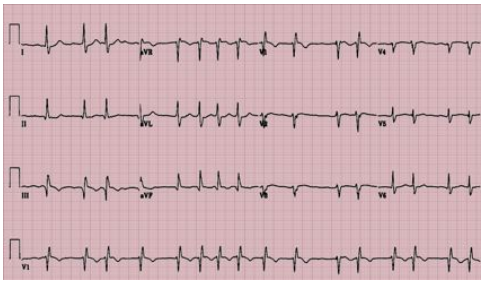
AF is mostly associated with the elderly, it's More common in male than female

Pathophysiology of Atrial Fibrillation and associated Stroke

In the pathophysiology, the doctor mentioned that irregular Electrical activity called microcircuit , While in atrial Flutter it's called macro-circuits (Watch video)

Normal heart	Atrial Fibrillation **Most common arrhythmia**
	
Normal regulation of heart rate and rhythm: Contraction is controlled by the sinoatrial (SA) node	In AF there will be microcircuit, very chaotic, disorganised flickers

EKG Changes

Normal EKG	AF EKG
	
Every P wave is followed By QRS	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). Why? bc signals are coming from different places through AV node to the ventricle so the AV node gets really confused.
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 Not all the 350-600 beats will be delivered by the AV node, it has a feature that will automatically block its conduction once it senses it is over conducting (usually at maximum 100 bpm). If it conducted more than 100 beats the patient would die. This feature is called **conduction decremental**. It will conduct, conduct and then it will stop to fill more.	Normal heart rhythm is disrupted in AF: 1. AF is characterized by: ○ **Rapid** (350–600 beats/min) and **irregular atrial rhythm**. R-R Is irregular ○ **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 ألن 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 هذي الخاصية اسمها conduction decremental. It will conduct conduct then it will stop to fill more. 3. Contraction of the ventricles can be: ○ **Fast AF**: Irregular and rapid (110–180 beats/min; tachycardia)Ventricular rate can never go faster than 180. ○ **Slow AF**: Irregular and slow (<50 beats/min; bradycardia) seen in elderly ○ **Controlled AF**: Normal 4. **Cardiac output** can be **reduced** (Due to loss of atrial contraction which contributes 20% of the cardiac output)

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:

Important to know to predict patient's response to treatment

Over time become harder to treat due to electrical and structural remodeling

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. Due to collagen deposit
- 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.

Consequences of AF

Death	Increased mortality, especially cardiovascular mortality due to sudden death, heart failure or stroke.
Stroke Important to prevent	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.

*Due to microemboli

Diagnosis of Atrial Fibrillation:

As anything else you need to be systematic.

1.Take history

2.Do physical examination

3. Do some tests.

Clinical Evaluation of Patients with AF

ALL patients:

- History
- Physical examination
- Electrocardiogram (ECG)
- Trans**thoracic** echocardiogram (T**T**E)
- Blood tests To check the thyroid.
- Holter monitor
- Chest x-ray

SELECTED patients:

- Transo**esophageal** echocardiogram (T**E**E)

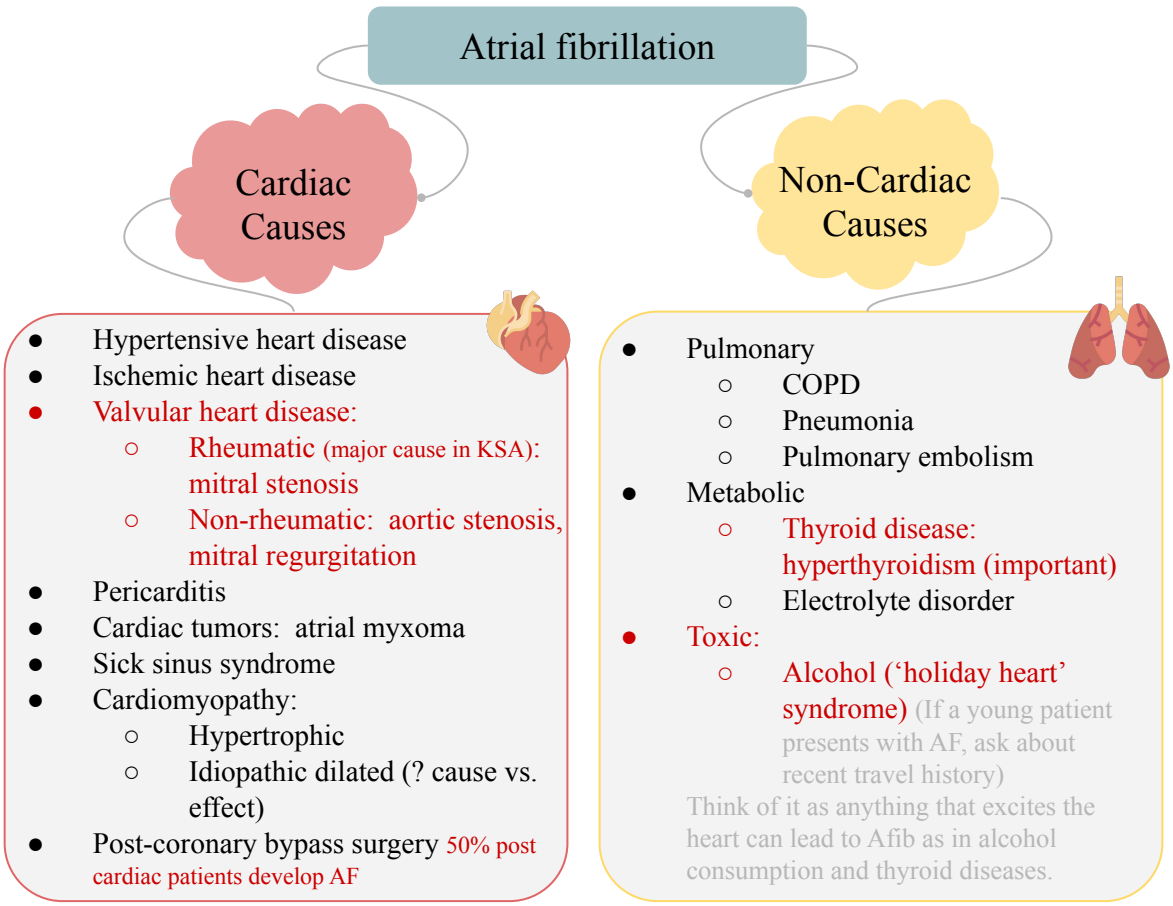
Who exactly? those presenting within 48 hours of onset of AF

History and Physical examination

- **Clinical conditions associated with AF**
  - **Underlying heart condition**s (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** dig in about detail of episode
  - **First episode, paroxysmal, persistent, permanent** more details below
  - **Triggers** – e.g. emotional stress, alcohol, physical exercise, gastroesophageal disease
  - **Specific symptoms**
  - **Response to** any **treatments** administered

Diagnosis of Atrial Fibrillation

In history you need to ask about the possible causes



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
  - Comorbidities

For each symptomatic episode, there are 12 asymptomatic episodes.

Signs and symptoms

Causes:

Irregular heart beat

Sign/Symptom

- Irregularly irregular pulse
- Palpitations Most of them

Cardiac ischaemia

Sign/Symptom

- Chest pain (angina)

Due to mismatch between decreased O2 supply (lower cardiac output), and increased demand (e.g. during exercise)

Hypotension

Sign/Symptom

- Dizziness and fainting (syncope)

Increased risk of clot formation

Sign/Symptom

- Thromboembolic TIA, stroke

Sometimes this will be the 1st presentation and th pt come to the ER with a stroke

Decreased cardiac output

Sign/Symptom

- Fatigue
- Diminished exercise capacity
- Breathlessness (dyspnoea)
- Weakness (asthenia)

Electrocardiogram

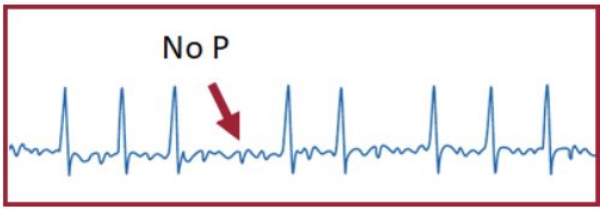
What does it do?

→ Assesses the electrical activity of the heart

→ Essential for all patients with suspected AF, to identify:

1. **Abnormal heart rhythm (verify AF)**
2. **Left ventricular hypertrophy**
3. **Pre-excitation**
4. **Bundle-branch block**
5. **Prior MI**
6. **Differential diagnosis of other atrial arrhythmias**

- Impulse from sinoatrial (SA) node stimulates myocardium to contract

Normal sinus rhythm	AF
	
• Normal heart rate • Regular rhythm • P Waves • Steady baseline	• Heart rate increased (tachyarrhythmia) • Irregular rhythm • No P wave • Irregular baseline (f waves)

Dr: AF episode must last at least 30 seconds

Transthoracic echocardiography (TTE)

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

Left atrial size:
- Enlarged means chronic AF
- Not enlarged new / acute AF

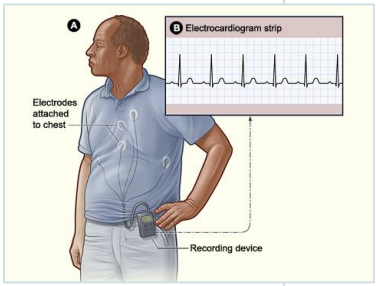
Laboratory tests

- Routine blood tests should be carried out at least once in patients with AF
- Important parameters to assess include:
  - Thyroid function (TSH)
  - Renal function
  - Hepatic function
  - Serum electrolytes
  - Complete blood count
- You need a baseline to monitor ADRs of treatment. (Eg: hypo/hyperthyroidism by TSH level with amiodarone use.)

Holter monitor:

We use the holter monitor to diagnose and follow up with the patient to see if the treatment is effective.

- Portable ECG device
- Continuous monitoring for a short period of time (typically 24-48 h)
- Useful for
    - Detecting asymptomatic AF
    - Evaluating patients with paroxysmal AF
    - Associating symptoms with heart rhythm disturbance
    - Assessing response to treatment



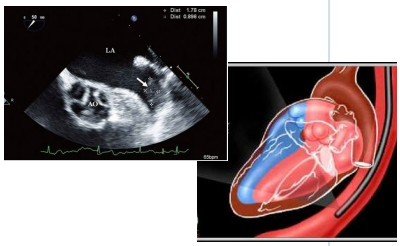
(which is a small device attached to the patient and tracks his ECG for 24 hours), in paroxysmal atrial fibrillation and it is the most accurate test. Dr's note: it's important to document AF before diagnosis because you will commit the patient for life long treatment

Transoesophageal echocardiogram (TEE)

(the left atrial appendage, which is a small ear-shaped trabeculation sac in the muscle wall of the left atrium, is most common site of thrombi) Only way to see appendage and most accurate to assess the risk of stroke or thombi (important)

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**
- 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 the heart loses it atrial kick, which contributes 20% to the cardiac output. Also there is blood stasis and this causes the blood to coagulate and form a thrombus that can empolise and cause a stroke.

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

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

Important to know to guide treatment

TABLE 4 Definitions of AF: A Simplified Scheme

Term	Definition
Paroxysmal AF	• AF that terminates spontaneously or with intervention within 7 d of onset. • Episodes may recur with variable frequency.
Persistent AF	• Continuous AF that is sustained >7 d.
Long-standing persistent AF	• Continuous AF >12 mo in duration.
Permanent AF Not used anymore since 2019	• The term "permanent AF" is used when the patient and clinician make a joint decision to stop further attempts to restore and/or maintain sinus rhythm. • Acceptance of AF represents a therapeutic attitude on the part of the patient and clinician rather than an inherent pathophysiological attribute of AF. • Acceptance of AF may change as symptoms, efficacy of therapeutic interventions, and patient and clinician preferences evolve.
Nonvalvular AF	• AF in the absence of rheumatic mitral stenosis, a mechanical or bioprosthetic heart valve, or mitral valve repair.

Treatment of Atrial Fibrillation

- If patient presents with acute AF—> patient is not hemodynamically stable
    - (I.e.Unstable: Hypotension, altered mental status, develops congestive heart failure)
    - Do synchronised electrical cardioversion.
- If the patient is not present with acute AF —> patient is hemodynamically stable

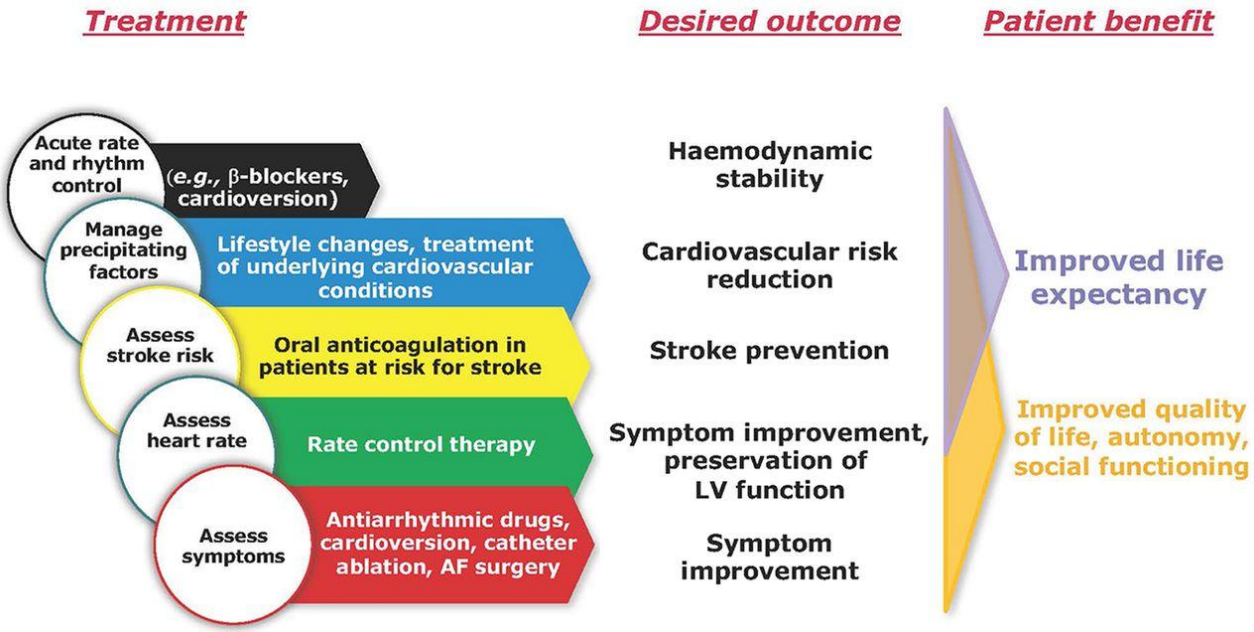
  —> need to achieve the following 3 domains:

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

You need address these questions before starting treatment:

- How to prevent stroke?
- Should i go for rate control?
- Do I need to maintain or restore sinus rhythm?

The Five Domains of Integrated AF Management



I don't have to do all 3 in every patient, it is considered therapy maintenance and beforehand we have already done cardioversion

Treatment of Atrial Fibrillation

A — Stroke Prevention

- Warfarin has food-drug interaction , mostly food that contains vitamin K . مثل الكراث
- Dabigatran
- Apixaban
- Rivaroxaban
- Edoxaban

Those 4 underlined drugs are the new oral anticoagulants NOAC, which has less drug-drug and food-drug interactions. 1st line therapy

- Removal/isolation of left atrial appendage, e.g. WATCHMAN® device or surgery

B — Rate Control

- Ca2+-channel blockers
- beta-blockers
- Digoxin
- Ablate/pace

Target heart rate for AF patient:
<110 for daily activity, <80 during rest

C — Maintenance of sinus rhythm

- Antiarrhythmic drugs
  - Class IA (procainamide)
  - Class IC (flecainide)
  - Class III: e.g. amiodarone (Best effective, but not first line due to complications. It can be given to elderly), dronedarone
- Ablation
- Surgery (MAZE)

1st thing we do is to restore sinus rhythm
المريض AF فالزم 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 (ablation)

A. **Stroke prevention**: The NOACs are better than warfarin in term of :
   - No need to monitoring
   - No Drug food interaction
   - Minimum Drug-Drug interaction
   - Their Efficacy as good as warfarin or better in some cases

B. **Rate control**: 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 your patient is a known case of heart failure —> digoxin or beta blockers are the best choice.
   - What if the patient has IHD? We give beta blockers.
   - The list of diseases go on , so assess your patient's history well and choose the most suitable drug.

Prevention of Thromboembolism: (You need to know how to calculate it!)

8.7 CHA_2DS_2-VAS_c stroke risk score for non-valvular atrial fibrillation

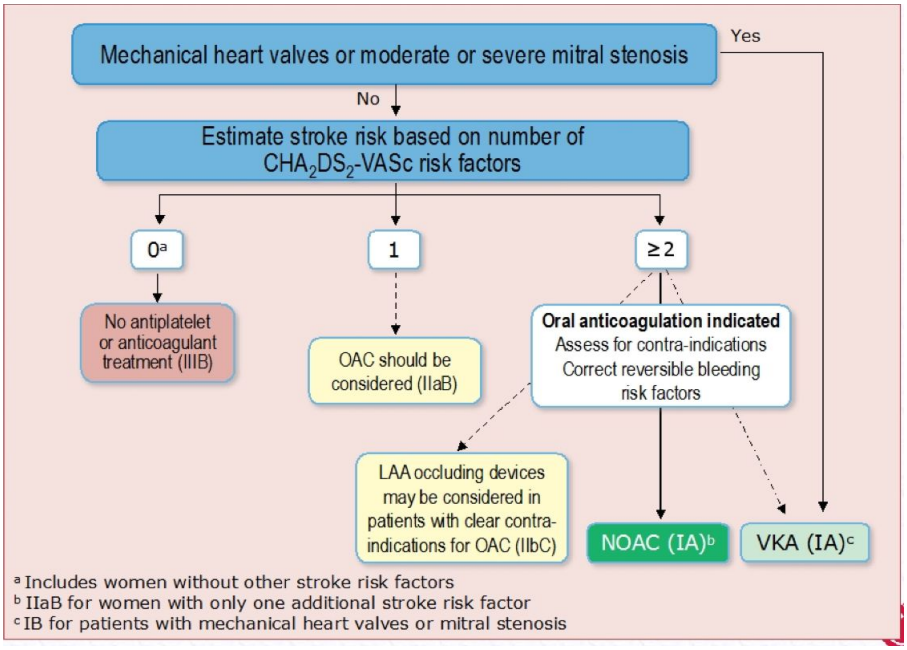
	Parameter	Score
C	Congestive heart failure	1 point
H	Hypertension history	1 point
A_2	Age ≥75 yrs	2 points
D	Diabetes mellitus	1 point
S_2	Previous stroke or transient ischaemic attack (TIA)	2 points
V	Vascular disease	1 point
A	Age 65–74 yrs	1 point
S_c	Sex category female	1 point
	Maximum total score	9 points

Annual stroke risk
0 points = 0% (no prophylaxis required)
1 point = 1.3% (oral anticoagulant recommended)
2+ points = >2.2% (oral anticoagulant recommended)

If the patient has a score of:

- 0, no anticoagulant is needed.
- 1, Aspirin or a NOAC can be considered.
- 2 or higher, must be on warfarin, or NOAC.

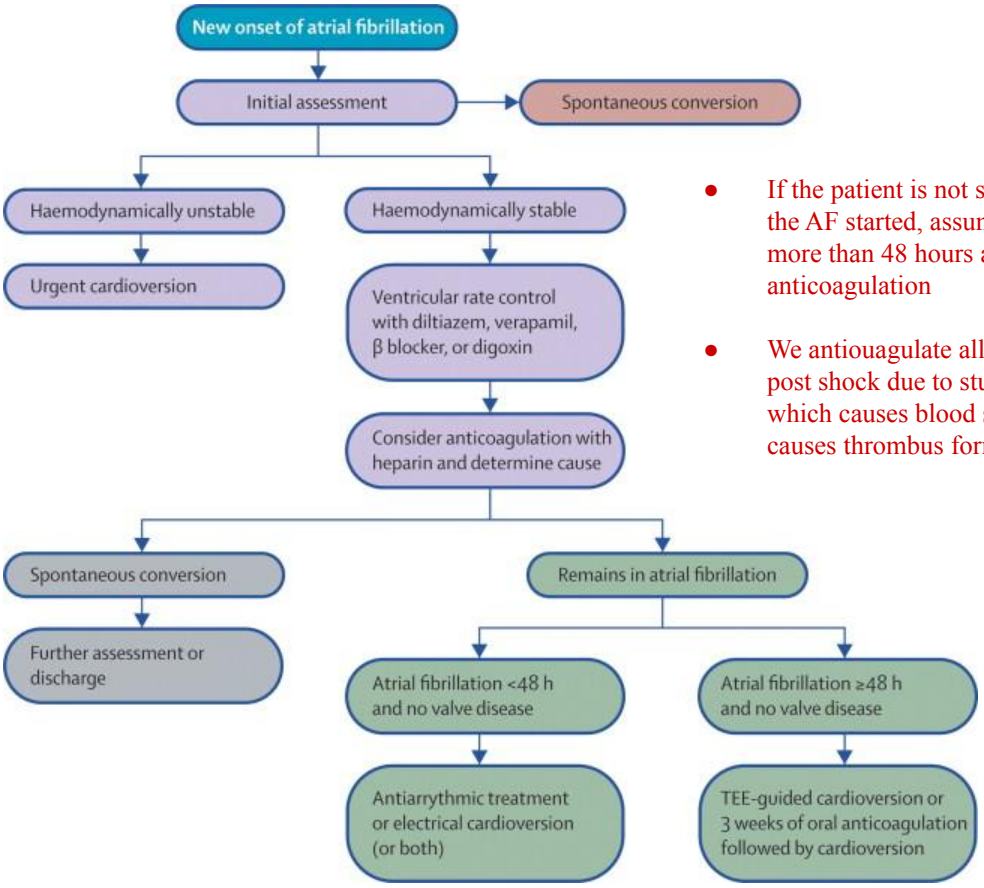
When do you give the patient heparin instead of warfarin?
A - when there is an actual clot.
B- when the patient has a mechanical heart valve.
If a patient has a mechanical valve or (moderate or severe) mitral stenosis. Skip the CHA2DS2 VASc and anticoagulation with **WARFARIN.**



$CHADS_2$ score	Patients (n = 1733)	Adjusted stroke rate (%/year)[a] (95% confidence interval)
0	120	1.9 (1.2–3.0)
1	463	2.8 (2.0–3.8)
2	523	4.0 (3.1–5.1)
3	337	5.9 (4.6–7.3)
4	220	8.5 (6.3–11.1)
5	65	12.5 (8.2–17.5)
6	5	18.2 (10.5–27.4)

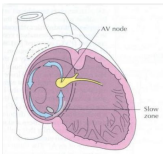
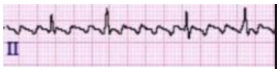
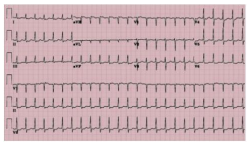
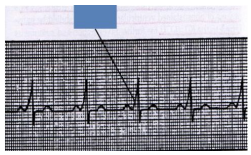
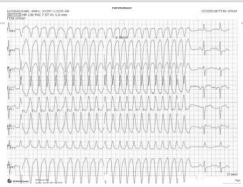
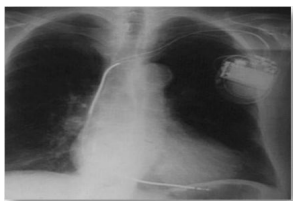
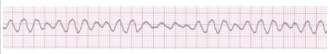
Rhythm-control therapies

- 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) It has a 98% success rate.
- Direct-current cardioversion is generally more effective than pharmacotherapy
- Likelihood of successful cardioversion decreases with the increase 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
- Rhythm control is prefered in young patients.
- When patient is hemodynamically unstable(which means he has: chest pain , dyspnea , hypotension , confusion) > Shock the patient (electric cardioversion).



- If the patient is not sure when the AF started, assume it is more than 48 hours and start anticoagulation
- We antiouagulate all patients post shock due to stunned atria which causes blood stasis and causes thrombus formation

Other arrhythmias

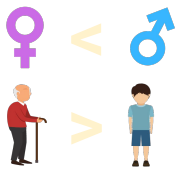
Type of Arrhythmia	Notes	ECG	Treatment
Atrial Flutter	 so basically there is a point in the atria that fire abruptly flutter wave macro circuit , unlike Afib which is micro circuits.	 2 or 3 atrial flutter waves and one QRS complex. Classic Sawtooth baseline Fixed R-R interval. HR: depends on the number of flutter waves; E.g 2:1 > 150 bpm 3:1 > 100 bpm	•***Unstable*** pt: •Synchronized cardioversion as per ACLS •***Stable*** pt: •Rate control :just like atrial fibrillation AF •Elective cardioversion :just like AF •Anti-coagulation :just like AF •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
Supra-ventricular Tachycardia **SVT are usually Benign and paroxysmal.**	Arrhythmias of supraventricular origin using a re-entrant mechanism with abrupt onset & termination - AVNRT (60%) - AVRT (30%) ex: Wolff-Parkinson-White (WPW) Syndrome Arrhythmia 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 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%)	 Wolff-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 + short P-R interval	-Medical therapy -Radiofrequency Ablation Mainly to improve quality of life (for ablation) AV blocking agents are contraindicated in patients with WPW (beta blockers and calcium channel blockers) as it will direct all the atrial firings to the ventricles and causes VT. 
Ventricular Tachycardia	This condition is lethal In IHD pt and familial genelopathy. Treatment: - Address the underlying cause and treat it - Shock Lethal in young patients		-Treat the underlying cause -Automatic Implantable defibrillators 
Ventricular Fibrillation	Low amplitudes no obvious QRS SO treat with shock immediately The patient will collapse as there is no coordinated ventricular contraction thus there is no cardiac output.		

SUMMARY: Atrial Fibrillation

Irregularly irregular rhythm with absent P-wave.

Most common arrhythmia (⅓)

90% of patients are asymptomatic



Types According to Termination

- **Paroxysmal:** Spontaneous or <7 days
- **Persistent:** >7 days
- **Long-Standing:** >12 months
- **Permanent:** No termination

Causes

Cardiac

- **Valvular Heart Disease**
- Ischemic Heart Disease
- Hypertensive Heart Disease
- Cardiomyopathies

Non-Cardiac

- **Pulmonary:** COPD, Pneumonia, PE
- **Metabolic:** Hyperthyroidism
- **Toxic**: Alcohol
- Cardiothoracic surgery

Diagnosis

Hx & Ex:

- Clinical type & 1st symptomatic attack
- Underlying heart disease
- Hyperthyroidism & Alcoholism

ECG:

- Irregular Rhythm
- Absent P-wave
- ↑ Heart Rate

Holter Monitor (Most Accurate):

- Detecting asymptomatic AF
- Evaluating paroxysmal AF
- Assessing response to treatment

Echo Type	Routinely Used	Structural Abnormalities	Functional Abnormalities	Sensitivity to LA Thrombus	Stroke Risk Assessment
TTE					
TEE					

Management

Acute AF + Hemodynamically Unstable → Electrical Cardioversion

AF + Hemodynamically Stable → Achieve the Following:

Rhythm Control

- **Pharmacotherapy:** Class 1A, 1C, and 3
- **Cardioversion:** Electrical shocks

Rate Control

- Ca+ Channel Blockers.
- β-Blockers.
- Digoxin.

Stroke Prevention

- **Antiplatelet:** Aspirin
- **Anticoagulants:** Warfarin or NOACs
- If the patient has severe mitral stenosis or a mechanical valve then use warfarin only.

Criterion	Score
CHF or LVEF ≤ 35%	1
Hypertension	1
Age >75	2
DM	1
Stroke | TIA | Systemic Embolism	2
Vascular Disease	1
Age 65-74	1
Female	1

CHA2DS2VASc	Treatment
0	Nothing or Antiplatelets
1	Aspirin or NOACs
≥ 2	NOACs or Warfarin

Questions:

1 - Which of the following characteristics is indicative of persistent AF?

A- Persisted for longer than 1 year because cardioversion has not been attempted

B- Persisted for longer than 1 year because cardioversion has failed

C- Persisted for longer than 7 days and may require either pharmacologic or electrical intervention to terminate

D- Persisted for 3-5 days and which terminated spontaneously

2- Which of the following is recognized as a risk factor for the development of AF?

A- Endocrine disorders

B- Hyperthermia

C- Female gender

D- African American heritage

3- Which of the following statements is accurate regarding the presentation of AF?

A- Patients presenting with syncope or near syncope require immediate direct current cardioversion

B- Most cases of AF are asymptomatic

C- Most patients present with bradycardia

D- The presence of ascites suggests left ventricular failure

4- A 76-year-old woman is evaluated in the emergency department for dizziness, shortness of breath, and palpitations that began acutely one hour ago. She has a history of hypertension and heart failure with preserved ejection fraction. Medications are hydrochlorothiazide, lisinopril, and aspirin. Electrocardiogram demonstrates atrial fibrillation with a rapid ventricular rate. Which of the following is the most appropriate acute treatment?

A- Adenosine

B- Amiodarone

C- Cardioversion

D- Diltiazem

Answers:

1. C
2. A
3. B
4. C