Team Medicine

Arrhythmia

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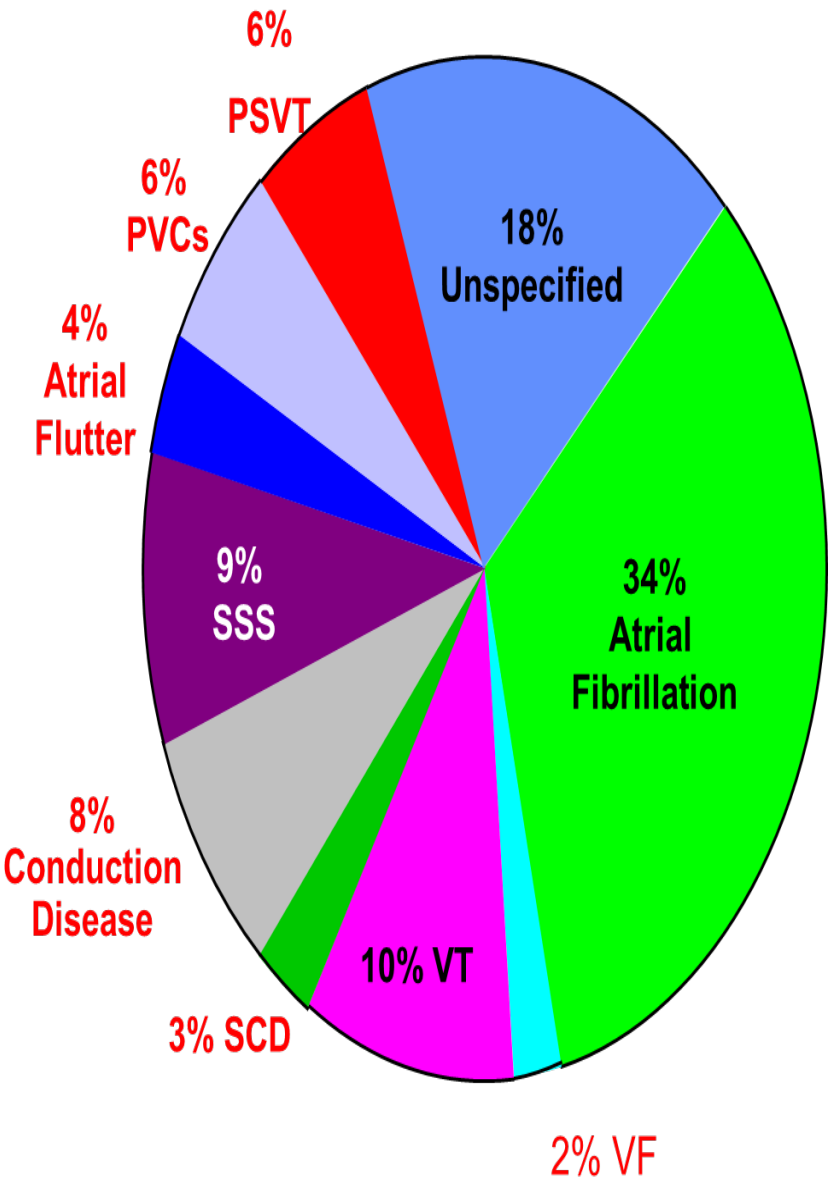
■ Important notes ■ doctor notes

Objectives:

- Identify mechanism of AF (Atrial Fibrillation)
- Recognize EKG of AF
- Discuss treatment options of AF
- Identify other forms of Arrhythmia

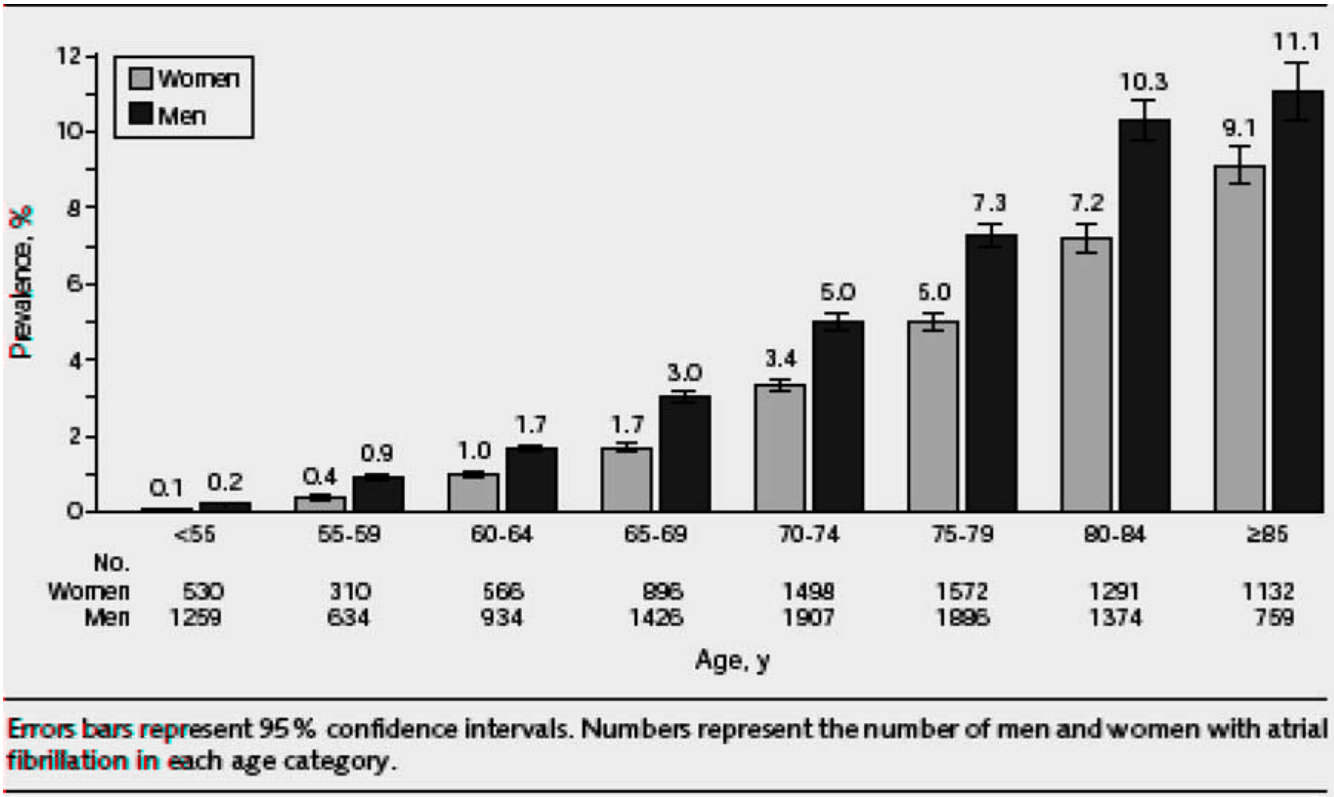
Atrial fibrillation accounts for 1/3 of all patient discharges with arrhythmia as principal diagnosis. **So Atrial Fibrillation is the most common arrhythmia**.

VF = Ventricular Fibrillation
VT = Ventricular Tachycardia
SCD = Sudden Cardiac Death
SSS = Sick Sinus Syndrome (there is a problem in the SA node like degenerative issues. As a result of that the SA node is not firing as it should)
PVCs = Premature Ventricular Contraction
PSVT = Paroxysmal Supra Ventricular tachycardia



Epidemiology:

- 2.3 million people in North America
- 4.5 million in EU
- In the 20 year AF admission have increased by 66%.
- $15.7 billion annually in EU
- Estimated prevalence of AF is 0.4 %to 1 %in the general pop. 8 % in pt. <80 years

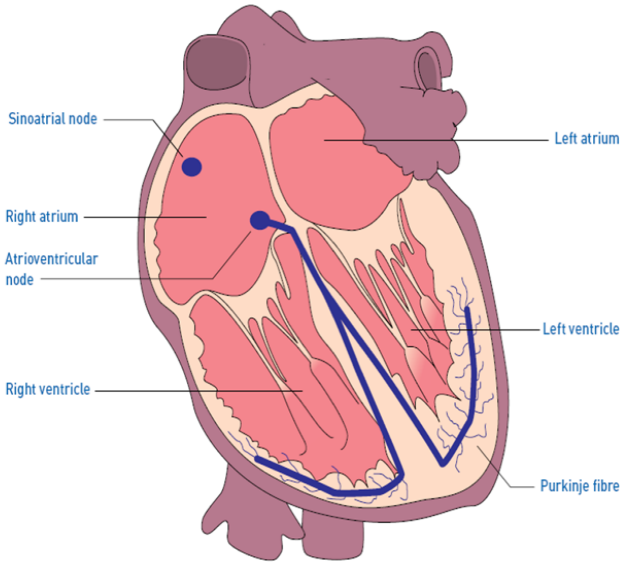


Errors bars represent 95% confidence intervals. Numbers represent the number of men and women with atrial fibrillation in each age category.

As the people get old the risk of AF increases dramatically

Pathophysiology of Atrial Fibrillation and associated Stroke:

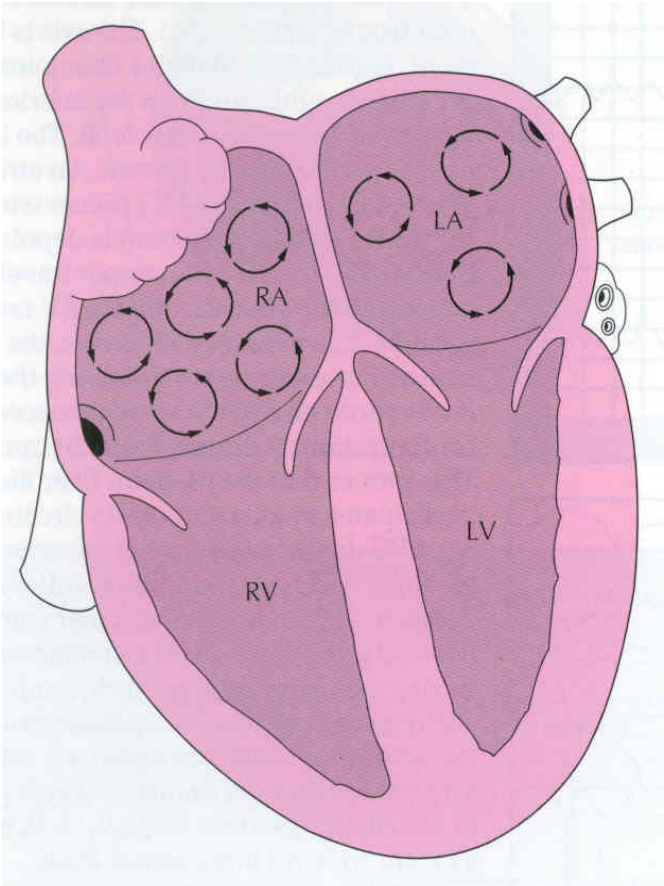
Physiologically: in the heart there is a system called conduction system (includes the myocardial cells and the nodes). Its function is to initiate and conduct the electrical impulses and contraction. SinoAtrial (SA) node is the natural pacemaker of the heart, SA node generate impulse – Action potential - automatically by spontaneous depolarization, then the impulses travel to the whole atria (this feature is only in the heart because it contains syncytium tissue) and then contraction takes place. The AtrioVentricular node subsequently receives the impulse and transmits it to the whole ventricle through the bundle of His and Purkinje Fibers and then contraction takes place.

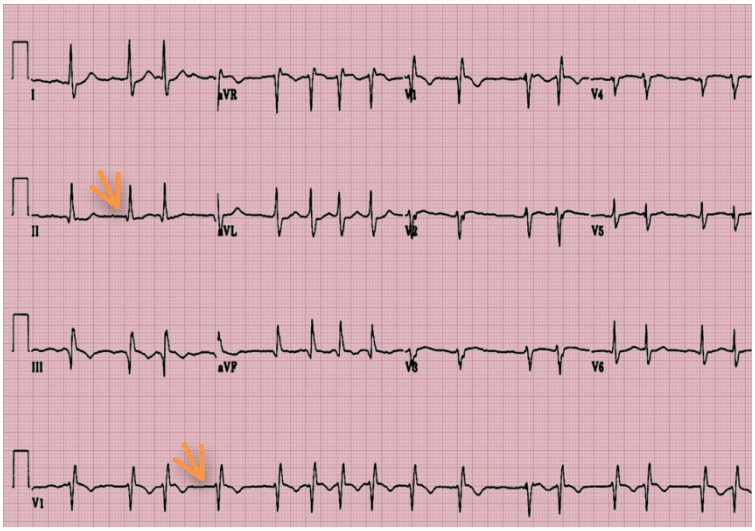


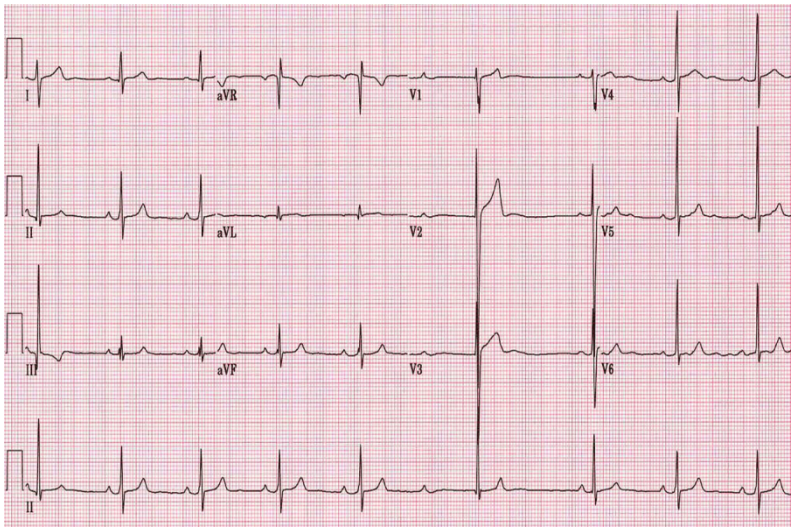
Every cardiac cell has the ability to generate electrical impulse by its own, but the Almighty Allah has created them to follow and listen to the SA node, so there would be no problem or dissociation AF Pathology : Multiple foci in the atria (located predominately within the pulmonary veins) fire continuously in a chaotic pattern and that will cause the atria to beat 400-600 beats /minute , causing irregular and rapid ventricular rate . Ventricular rate ranges between 75 – 175BPM that is because the smartest piece in the heart which is the AV node which will block the most coming impulses from the atria

What if there is no AV block?

The ventricles will fibrillate and by that time the patient will be dead







ECG of AF : we can see clearly two hallmarks:

1 – Absence of P waves(green arrows)

2- Irregularly irregular rhythm (Irregular RR interval)

NORMAL ECG

Normal heart rhythm is disrupted in AF :

- AF is characterized by:
  - Rapid (350-600 beats/min) and irregular atrial rhythm
  - Reduced filling of the left and right ventricles
- Conduction of most impulses from the atria to ventricles is blocked at the AV node
- Contraction of the ventricles can be:
  - Irregular and rapid (110-180 beats/min ;tachycardia)
  - Irregular and slow (50 > beats/min ;bradycardia)
  - Normal
- Cardiac output can be reduced

Because of the reduction in the filling of the ventricle and that will lead to decrease in the output and the patient will be fatigued and may cause pulmonary edema

AF begets AF: begets means generates

- AF causes remodeling:
  - Electrical :shortening of refractory period
  - Structural :enlargement of atrial cavities
- Many episodes of AF resolve spontaneously
- Over time AF tends to become persistent or permanent.

Consequences of AF:

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

VERY IMPORTANT:
Patient with AF in the presence of underlying heart disease have an especially high risk of embolization and hemodynamic compromise

Causes of Atrial Fibrillation:

1-Cardiac causes:

Hypertensive heart disease + heart failure are the most common causes
Ischemic heart disease
Valvular heart disease
Rheumatic: mitral stenosis
Non-rheumatic: aortic stenosis, mitral regurgitation
Pericarditis
Cardiac tumors: atrial myxoma
Sick sinus syndrome
Cardiomyopathy
Hypertrophic
Idiopathic dilated (cause vs. effect)
Post-coronary bypass surgery

2- Non-Cardiac causes:

Pulmonary:

COPD

Pneumonia

Pulmonary embolism

Metabolic:

Thyroid disease: hyperthyroidism (Doctors should do thyroid function test, especially in young patients)

Example that the doctor mentioned (young Saudi male comes to the clinic and you suspect AF. You order Echocardiography and it reveals that there is no abnormality in the heart valves. What is your next step?

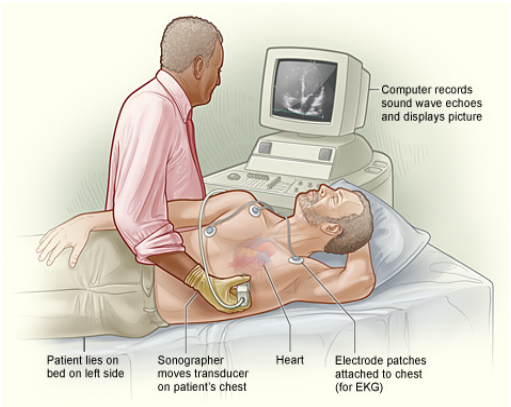
You should do Thyroid function Test and Alcohol test

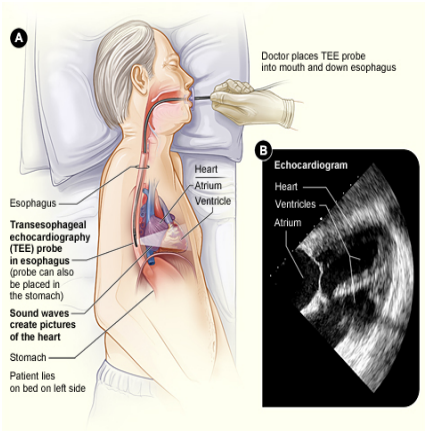
Electrolyte disorder

Toxic: excessive alcohol intake ('holiday heart' syndrome)

Diagnosis of Atrial Fibrillation:

- Signs and symptoms
- Electrocardiography (ultrasound)
- Transthoracic echocardiography (this test allows you to see the valve and to assess the status of heart walls)
- Laboratory tests
- Holter monitoring
- **Transoesophageal echocardiography (this is very excellent test. It helps to assess the left atrial appendage – auricle – which is the most common site where the blood gets stagnated there and form clot which may go up and cause STROKE)**
- Exercise testing
- Chest radiography





Heterogeneous 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

Sign and Symptoms of AF:

Cause	Sign/symptom
Irregular heart beat	Irregularly irregular pulse Palpitations
Decreased cardiac output	Fatigue(the pumped blood is not enough) Diminished exercise capacity Breathlessness (dyspnea) Weakness (asthenia)
Hypotension	Dizziness and fainting (syncope)
Cardiac ischaemia	Chest pain (angina)
Increased risk of clot formation	Thromboembolic TIA, stroke

Clinical evaluation of patients with AF:

- **All patients**
  - History
  - Physical examination
  - Electrocardiogram(ECG)
  - Transthoracic echocardiogram (TTE)
  - Blood tests
  - Holter monitor
  - Chest x-ray (To rule out pulmonary causes)
- **Selected patients:** (For patient who are at high risk of getting stroke ,and also interfere with plan of management)
  - **Transesophageal echocardiogram (TEE)**.

History and physical examination:

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

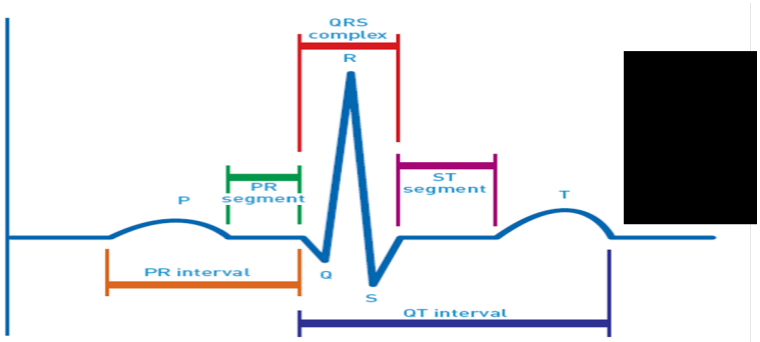
In some patients no cause can be found, and this group is called ' Lone ' or ' Idiopathic ' AF. The pathogenesis of Lone AF is unknown but genetic predisposition of arrhythmia has been proposed.

Electrocardiogram (ECG)

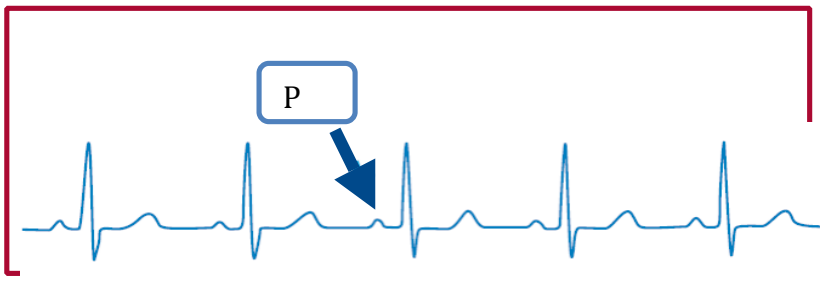
- Assesses the electrical activity of the heart
- Essential for all patients with suspected AF, to identify:
  - Abnormal heart rhythm (verify AF)
  - Left ventricular hypertrophy
  - Pre-excitation
  - Bundle-branch block
  - Prior MI
  - Differential diagnosis of other atrial arrhythmias

Normal Sinus Rythym

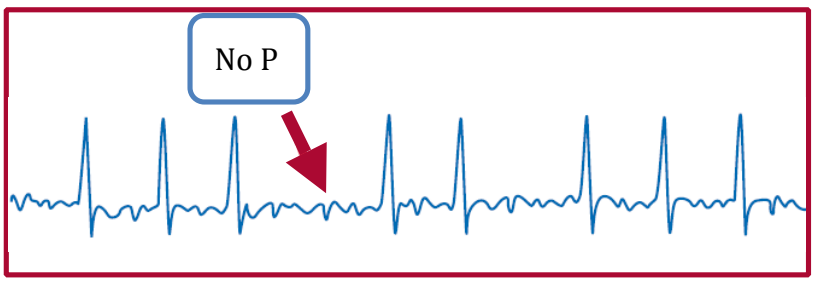
- Impulse from sinoatrial (SA) node stimulates myocardium to contract
- P-wave: atrial depolarization
- QRS complex: ventricular depolarization
- T-wave : ventricular repolarization



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



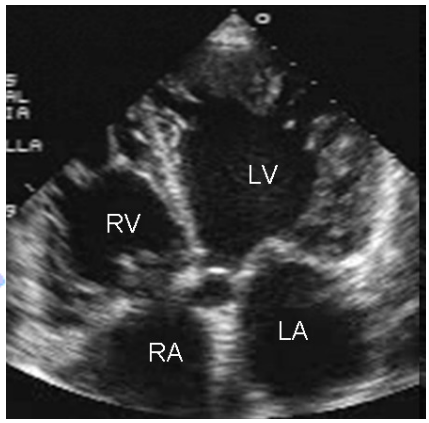
- AF
  - **Heart rate increased (tachyarrhythmia) - bradyarrhythmia may happen also**
  - **Irregular rhythm**
  - **No P wave**
  - **Irregular baseline**



Transthoracic Echocardiography: TTE

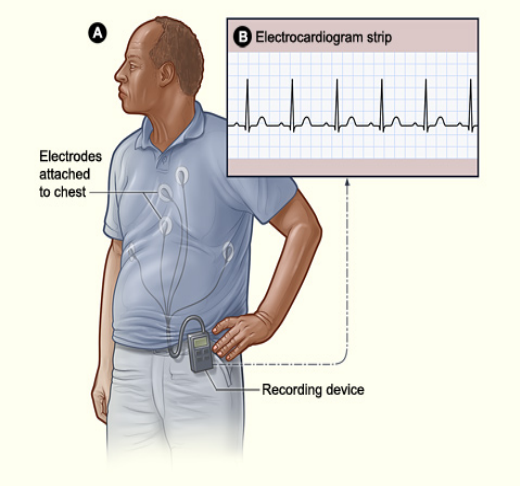
TTE is NOT good to assess the status of left atrial appendage

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



Laboratory Test:

- 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



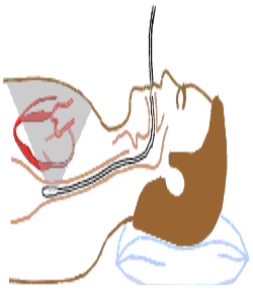
Holter Monitor:

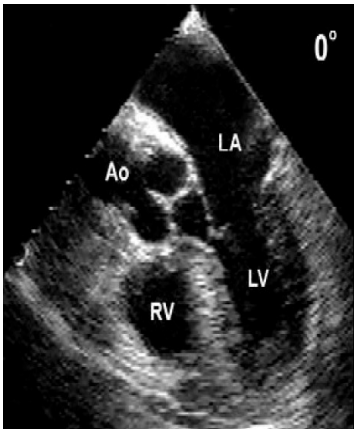
- Portable ECG device
- Continuous monitoring for a short period of time (typically 24 hours)
- Useful for
  - Detecting asymptomatic AF
  - **Evaluating patients with paroxysmal AF**
  - Associating symptoms with heart rhythm disturbance
  - Assessing response to treatment

Notebook is given along with the device. The patient is asked to write any activity he/she has done to assess the ECG correctly

Transoesophageal echocardiogram (TEE)

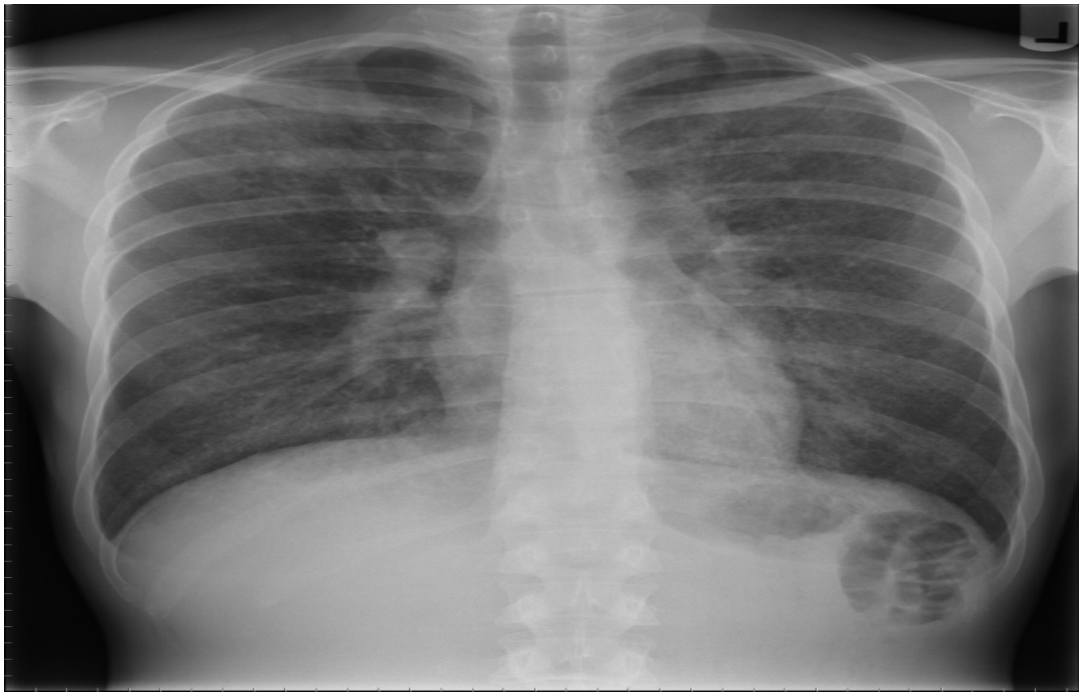
- Ultrasound transducer positioned close to the heart using an endoscope-like device
- **High quality images of cardiac structure and function**
  - Particularly the left atrial appendage, the most common site of thrombi in patients with AF
- Not routinely used but useful for:
  - **Accurate assessment of risk of stroke**
  - Detection of low flow velocity ('smoke' effect)
  - **Sensitive detection of atrial thrombi**





Chest Radiology:

- 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



Classification of Atrial Fibrillation

1-Classification of AF: joint guidelines of the ACC, AHA and ESC (1)
depends on the duration

Classification	Definition
First-detected	**First recognized episode AF**
- Recurrent - Paroxysmal - Persistent	**Episodes of arrhythmia ≤2** **AF that terminates spontaneously** **within 24 hours** **AF that persists for > 7 days but can be converted by electrical conversion**
- Permanent	**AF that cannot be terminated by cardioversion. And long-standing AF (for > 1 year) where cardioversion not indicated/not attempted**

2-Classification of AF: joint guidelines of the ACC, AHA and ESC (2)
depends on the causes

Classification	Definition
Lone or Primary	**AF without clinical/ECG evidence of cardiopulmonary causes**
Secondary	**AF associated with cardiopulmonary diseases (e.g: MI, Pneumonia)**
Non-valvular	**AF that is not associated with damage to heart valves (e.g: Rheumatic mitral valve disease, mitral valve repair, Prosthetic heart valve.**

Treatment of Atrial Fibrillation

3 STRATEGIES:

1. Prevention of Thromboembolism
2. Rate Control
3. Restoration and Maintenace of sinus rhythm

	Pharmacological	Non-Pharmacological
Stroke prevention	Anticoagulants: 1- Warfarin 2- Aspirin **3- Dabigatran** **4- Apixaban** **5- Rivaroxaban**	**Removal/Isolation of left atrial appendage (e.g: WATCHMAN device or surgery)**
Control of heart rate (AV nodal slowing agents)	**1- Ca++ Channel blockers :** Verapamil, diltiazem **2- B-blockers** : Metoprolol, propranolol 3- Digoxin (Digitalis)	**Ablation** **Pacemaker**
Maintenance of sinus rhythm	Antiarrhythmic drugs : 1- Class IA 2- Class IC: Flecainide 3- Class III **: Amiodarone**, Dronedarone	**Ablation** **Surgery (MAZE)**

Notes:

1- Warfarin has many disadvantages, first one, is the checking of INR* weekly in the hospital because of the low therapeutic index. Second one is the risk of hemorrhagic shock because of the low therapeutic window (INR becomes more than 3 where it should be maintained between 2-3). Third one is food-drug interaction (Green leaves)

2- Watchman device is a Left Atrial Appendage closure surgery. It is a device that the surgeon implants in the Left Atrial Appendage to close it and then the endocardial tissue will grow over the device. The time required to be completely overlapped with endocardial tissue is about 4 weeks (so the patient will be anticoagulated for 4 weeks). This procedure done in the cath lab. (It is a reduction risk of thromboembolism surgery)

3- MAZE procedure is a catheter ablation technique aiming to form scar tissues in the atria (Especially around the pulmonary veins) (Many studies suggest that the most common area where the chaotic impulses generated is from the pulmonary veins). This technique is indicated in patients who don't respond to antiarrhythmic drugs.

4- Amiodarone has some side effects like hepatotoxicity.

Prevention of thromboembolism

Stroke Risk Score for Atrial Fibrillation:

1-The **CHADS2** score is a clinical prediction rule for estimating the risk of stroke

	Clinical condition	Score	Prevalence
C	Congestive heart failure	1	**32%**
H	Hypertension	1	**65%**
A	Age >75	1	**28%**
D	Diabetes Mellitus	1	**18%**
S2	Stroke or TIA (Transient Ischemic Attack)	2	**10%**
Moderate-high risk		≤2	**50-60%**
Low risk		0-1	**40-50%**

2- The **CHA2DS2-VASc** score is a refinement of CHADS2 score

	Clinical Condition	Score (Points)
C	Congestive heart failure	**1**
H	Hypertension	**1**
A2	Age> 75	**2**
D	Diabetes Mellitus	**1**
S2	Stroke or TIA / systemic embolism	**2**
V	Vascular diseases (Aortic plaque, Peripheral Artery Diseases, MI)	**1**
A	Age (65-74)	**1**
Sc	Sex category (Female)	**1**
	Moderate-high risk	**≤2**
	Low risk	**0-1**

To assess the risk of thromboembolism, you have to calculate the points (e.g.: If it is 2 and above that means the patient is at high risk to develop stroke)
Example: patient has been diagnosed with AF. The patient is hypertensive and diabetic and has had prior stroke. How risky is the stroke to develop?
The patient is at high risk (the score is 4)

Restoration of sinus rhythm (Rhythm-Control therapies)

- The objective of rhythm-control therapy is to restore (cardioversion) and maintain normal sinus rhythm
- Cardioversion can be achieved by:

1) Pharmacotherapy with antiarrhythmic agents
2) Electrical shocks (direct-current cardioversion)
   - Direct-current cardioversion is generally more effective than pharmacotherapy
   - Likelihood of successful cardioversion decreases with the duration of AF:
   1. Pharmacological cardioversion is most effective when initiated within 7 days of AF onset
   2. Cardioversion can dislodge thrombi in the atria, increasing the risk of stroke
   3. Thromboprophylaxis is recommended for ≥3 weeks before and for at least 4 weeks after cardioversion in patients with AF that has persisted for ≥48 h

1- Electrical cardioversion ' Shock ' is the excellent choice to restore sinus rhythm and its success rate is 98%.

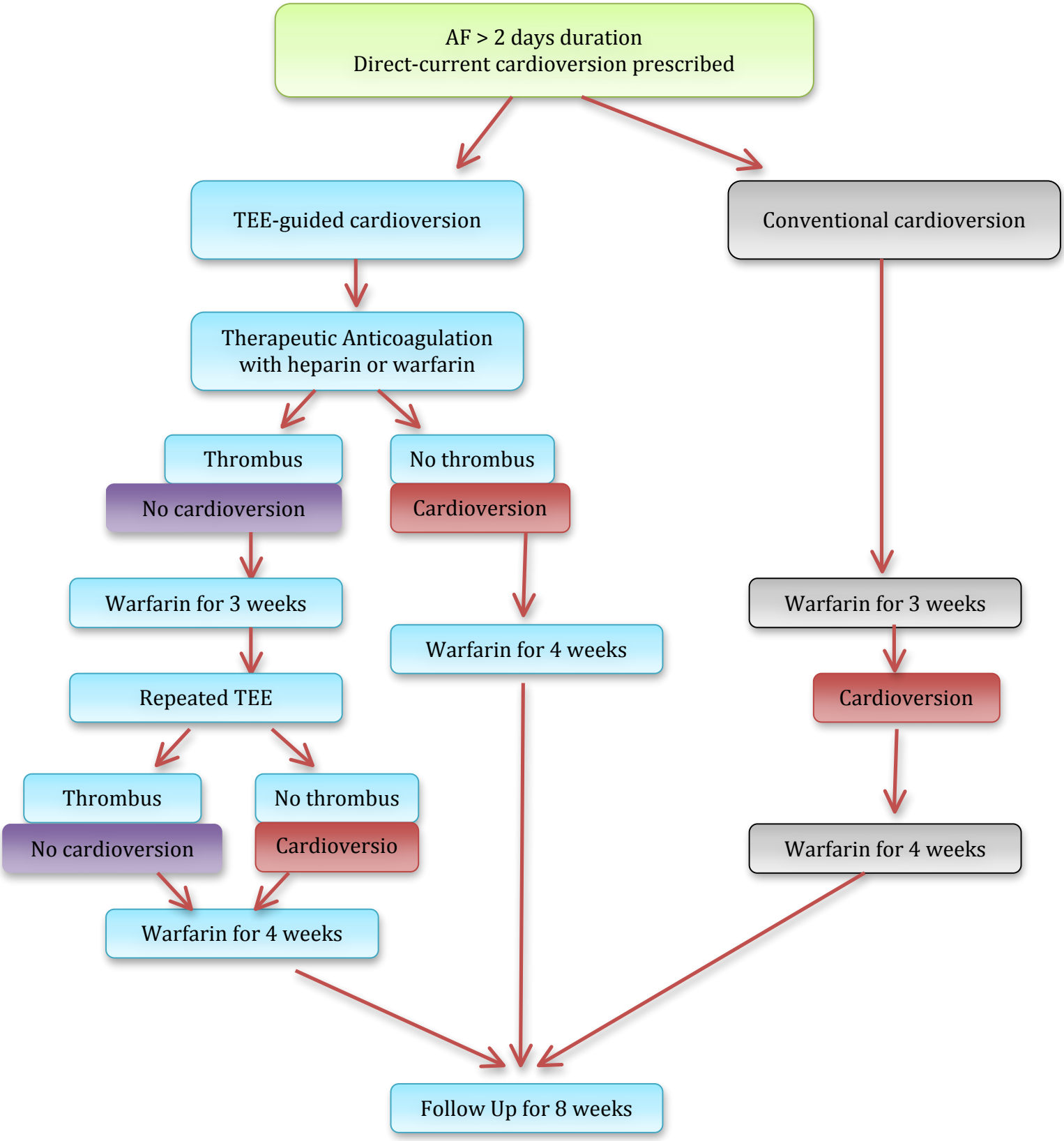
2- In some situations, the cardioversion may become critical choice and may itself cause stroke. For instance, patient came to the ER with history of AF for one week and the doctor shocked the patient. After that the patient developed stroke. WHY?? Because the patient has AF for one week (more than 48 hours ' 2 days') and the shock dislodge the clot and the clot moved up and caused stroke…. The good management here is to anticoagulate the patient for at least 3 weeks and then shock him. After that the patient should be anticaoagulated for at least 4 weeks

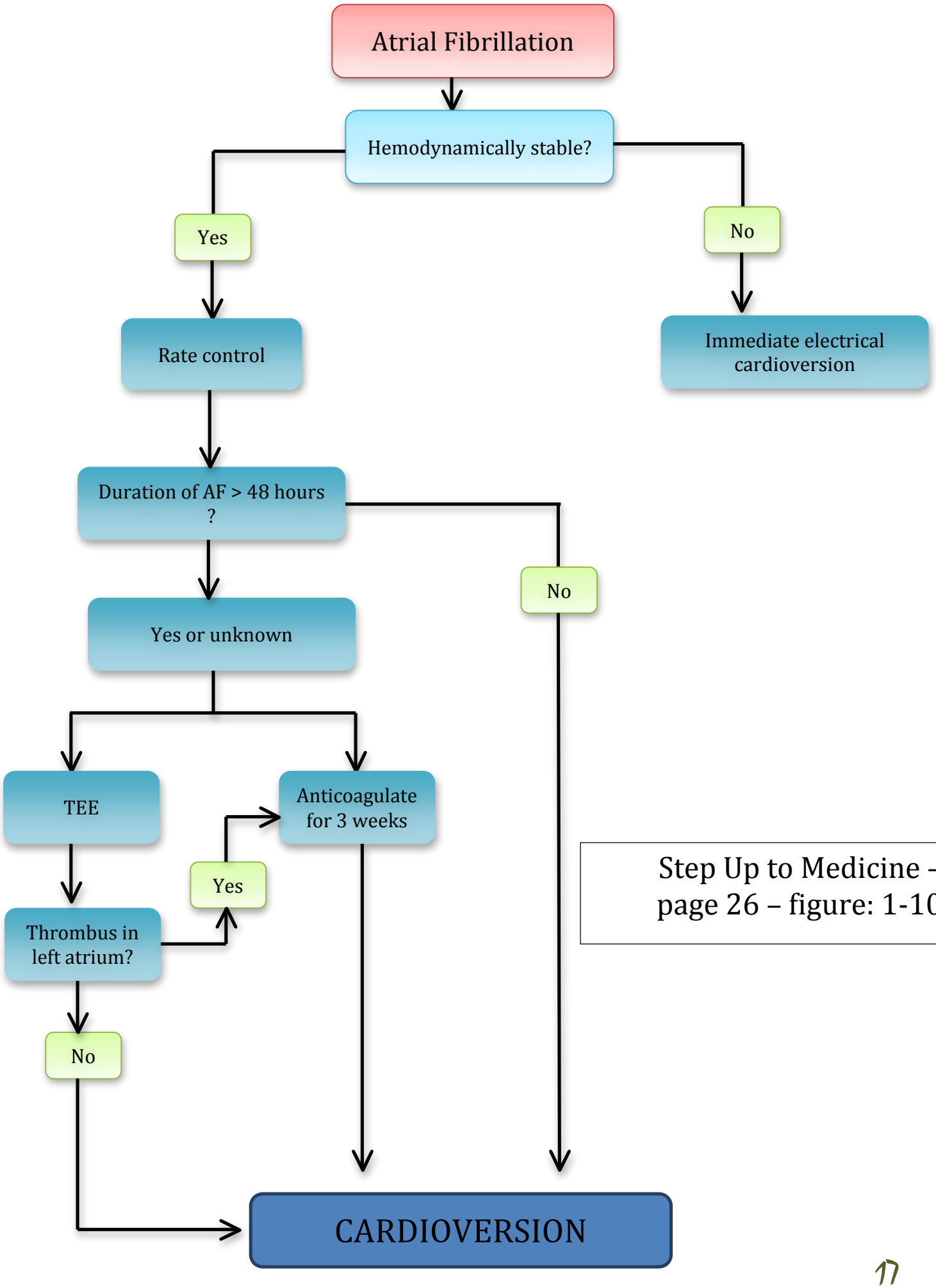
3- The clot in patients with AF needs only 2 days to get almost formed in the atria (especially the left atrial appendage). So if a patient came with AF for less than 2 days, the doctor can shock him (but to be more sure the doctor should do Transesophageal Echocardiogram (TEE) to estimate the status of the clot)

4- If a patient is confused and he doesn't know when the AF started >> doctor should do TEE before shock .

5- Patient came to the ER suffering from the AF for one day. The doctor shocked him and then he has been discharged. After one week the patient came back with STROKE .WHY?? Because they did not put him on anticoagulants after the shock.

TEE-guided cardioversion: ACUTE study design





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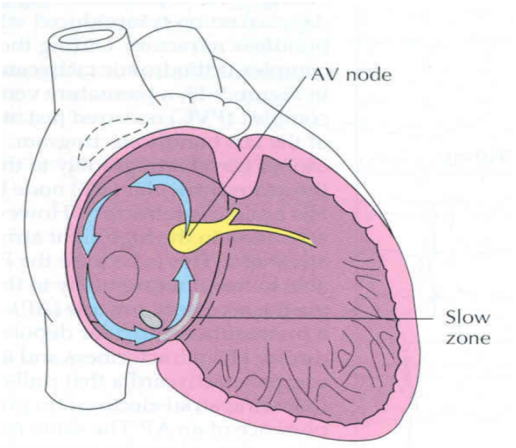
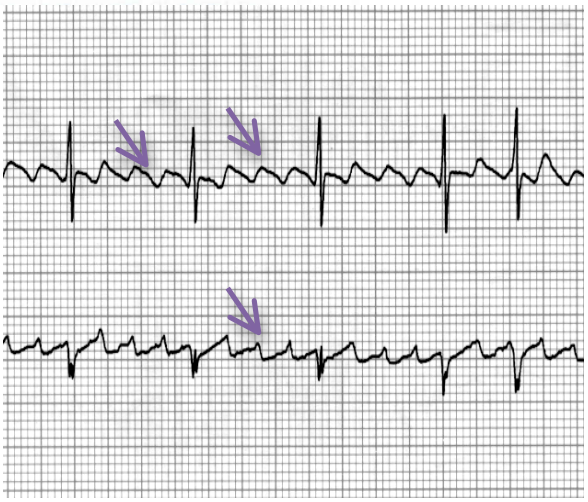
Atrial Flutter

Atrial flutter is often associated with Atrial Fibrillation and often requires a similar initial therapeutic approach.
Atrial flutter pathophysiology: one macro re-entrant right atrial circuit. The atria beats about 250-350 bpm in regular contraction.



ECG of Atrial Flutter shows saw-tooth baseline – purple arrows- , with a QRS complex appearing after every second to third ' tooth ' (P waves) in regular pattern.



Rx (management) of Atrial Flutter

- ***Unstable*** Patient (i.e. low BP / CP / AMS) :

  1- Synchronized cardioversion as per ACLS (Advanced Cardiovascular Life Support)
  2- 50J → 100J → 200J → 300J → 360J

  Stable Patient:
    - Rate control - just like atrial fibrillation (AFib)
    - Elective cardioversion - just like AFib
    - Anti-coagulation – just like AFib
    - Refer for ablation

SupraVentricular Tachycardia (SVT)

So, what is actually meant by SVT?

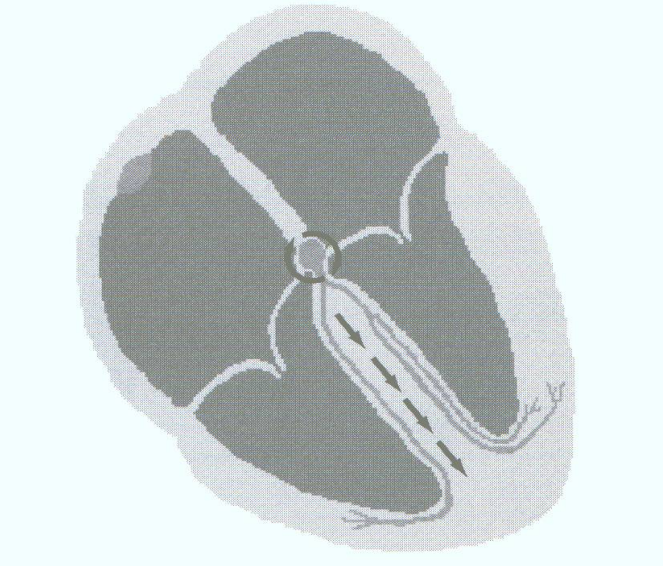
- Arrhythmias of supraventricular origin (Atrial origin) using a re-entrant mechanism with abrupt onset & termination

- AVNRT (60 %)
- AVRT (30 %)
- Atrial tachycardia (10%)

Adenosine is the drug of choice (it is called one-disease drug)

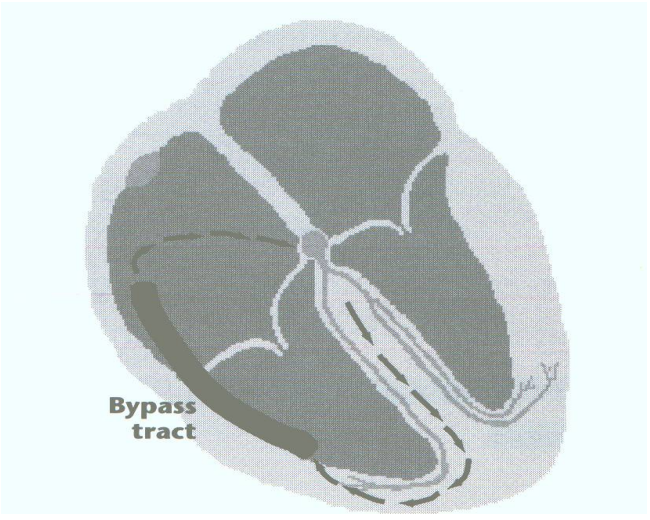
Atrio-Ventricular Nodal Reentrant Tachycardia (AVNRT):

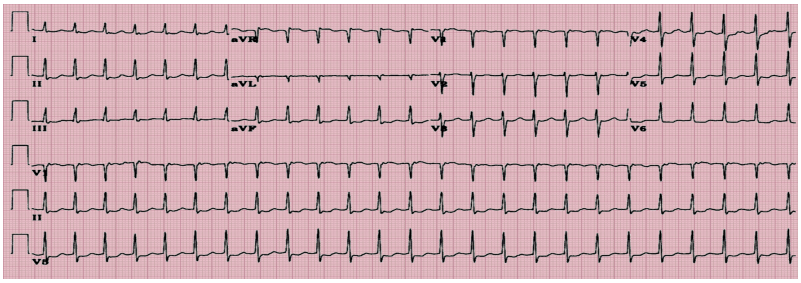
1- Two pathways (one is fast and the other is slow) Within AV node
2- It is the most common cause of SVT.
3- ECG**: Narrow QRS complex** with no discernible P waves (P waves are buried within the QRS complex) the venticles depolarization takes over the atrial depolarization (as they depolarize simultaneously)



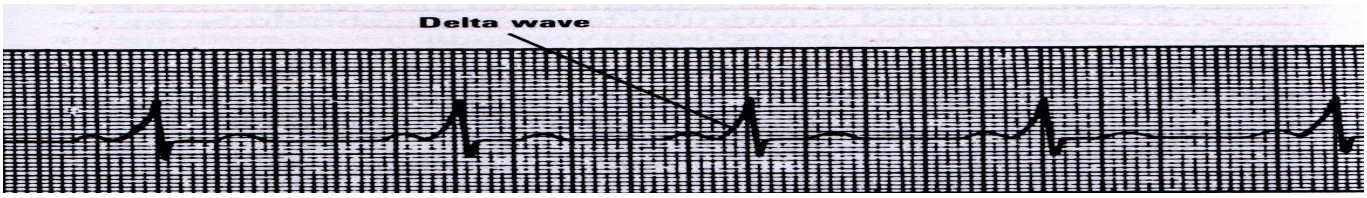
Atrio-Ventricula Reentrant Tachycardia (AVRT) :

1- An accessory pathway between the atria and ventricles that conducts retrogradely
2- This pathway is called Bypass tract (as they bypass the AV node)





Wolff-Parkinson-White Syndrome



ECG of WPW syndrome: Delta waves (Upward deflection seen before QRS complex)

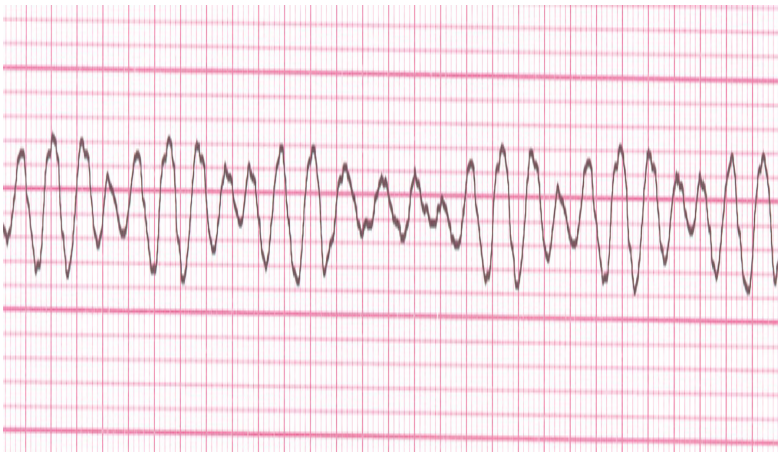
Pathophysiology of WPW syndrome: It is an accessory conduction pathway (extra abnormal band of tissue resembling purkinje) from atria to ventricles through the bundle of Kent causes premature ventricular excitation because it lacks the delay seen in the AV node.

* Treatment options:

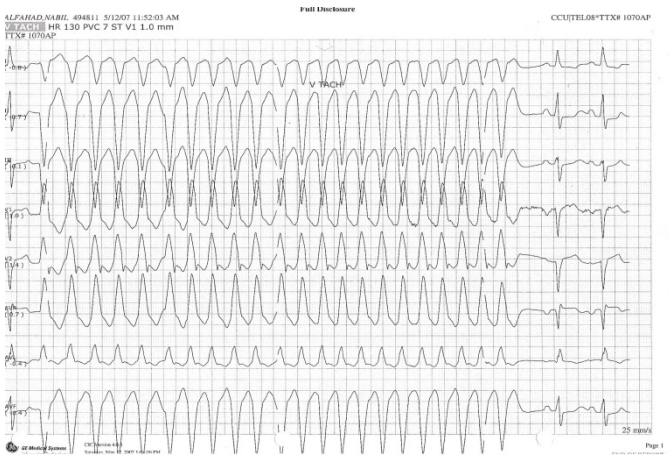
1- Medical therapy

2- Radio-frequency ablation

Other Arrhythmias:



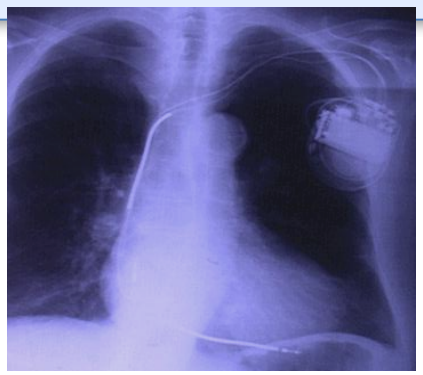
Ventricular Fibrillation:
ECG: No waves can be identified, very irregular rhythm.



Ventricular Tachycardia:
ECG: Wide and bizzare QRS complex

Treatment options:

1- Treat the underlying cause
2- Automatic implantable Defibrillators: it is a device that is surgically placed. It delivers an electric shock to defibrillate. It highly indicated in VFib



Summary:

1- Arrhythmia means absence of rhythm.
2- Arrhythmias are common and often benign, but may reflect underlying heart diseases
3- The presenting symptoms of arrhythmia are palpitation, chest pain, fatigue, syncope, and sometimes it is asymptomatic.
4- The most common arrhythmia is Atrial Fibrillation.
5- Absence of P waves and irregular QRS complex are hallmarks of AF.
6- The most important goal in management of AF is prevention of thromboembolism.
7- TEE is a good method to determine the presence or absence of thrombus.
8- The common complication of AF is stroke.
9- HTN and congestive heart failure are the commonest cause of AF.