MEDICINE

432 Team

1 Arrhythmias

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COLOR GUIDE: • Females' Notes • Males' Notes • Important • Additional

Objectives

- Identify mechanism of AF
- Recognize EKG of AF
- Discuss treatment options of AF
- Identify other forms of Arrhythmia

Helpful Video:
http://www.youtube.com/watch?v=eTd0oMqauIY

Atrial Fibrillation (AF):

Atrial Fibrillation is a disorder in which the two upper chambers of the heart beat fast and erratically. It is a condition that can come and go, but for many people it becomes a constant heart rhythm for the rest of their lives. It accounts for 1/3 of all patient discharges with arrhythmia as principal diagnosis. (Most common arrhythmia)

Epidemiology:

- 2.3 million people in North America
- 4.5 million in EU
- In the 20 year AF admission have increased by 66%.
- Costs $ 15.7 billion annually in EU
- Estimated prevalence of AF is 0.4% to 1% in the general pop. 8% in pt. >80 years (increases with age).
- Incidence and prevalence is more common in Male than Female

Pathophysiology of Atrial Fibrillation and associated Stroke

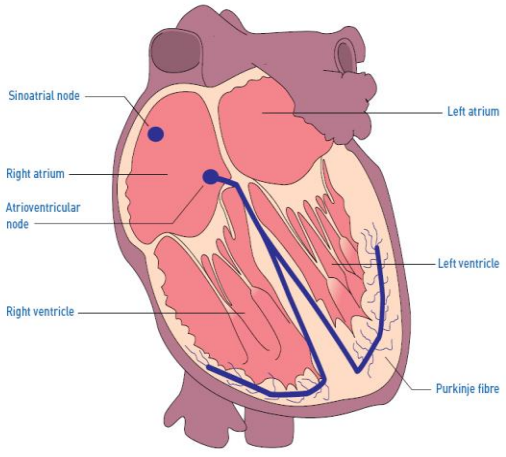
Every single cell in the heart can initiate and form an action potential, but all cells obey the signal generated by the SA node. In atrial fibrillation, multiple micro electrical circuits in left and right atrium that are fibrillatory or chaotic cause fast and inefficient rhythm (arrhythmia) (it is between 400-600 bpm!).

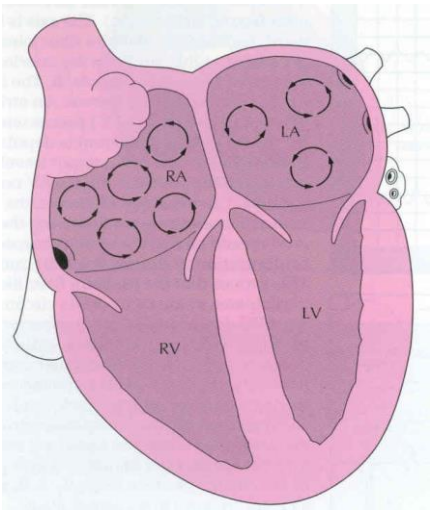
Note(s): *from Davidson page 564-565*

- AF is a complex arrhythmia characterized by both abnormal automatic firing and the presence of multiple interacting re-entry circuits looping around the atria.
- Episodes of AF are initiated by rapid bursts of ectopic beats or from diseased atrial tissue.

Normal regulation of heart rate and rhythm

- **Contraction is controlled by the sinoatrial (SA) node.**

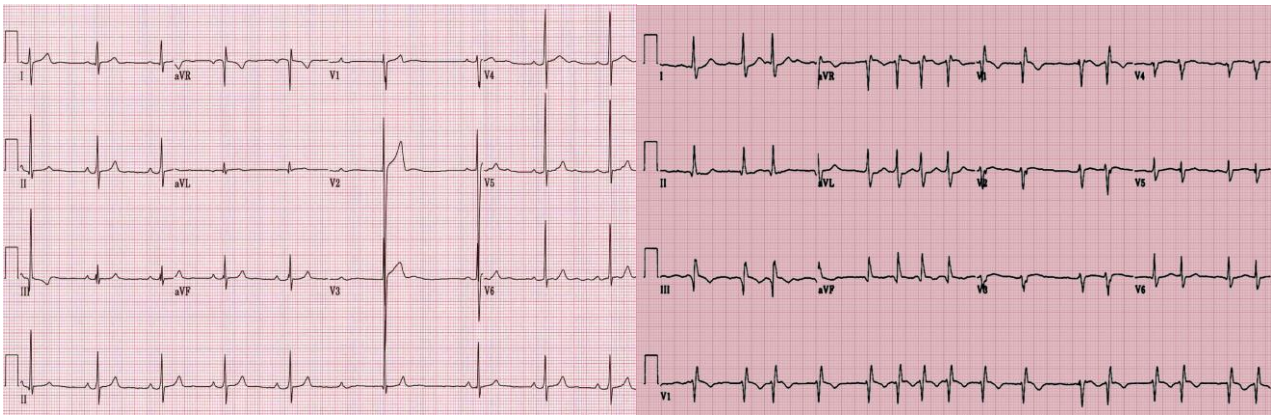




Note(s):

- *SA node is at the junction between the superior vena cava and right atrium.*
- *SA node is the pacemaker of the heart.*
- *All myocardial cells have the ability to generate electrical impulses (action potential) that trigger cardiac contraction; the SA node normally initiates it (Simply because SA node generates impulses slightly faster than the other areas with pacemaker potential).*

Normal EKG AF



Normal heart rhythm is disrupted in AF

- Absence of p wave (Normally p wave represents atrial contraction).
- Irregularity of RR intervals, irregularly irregular rhythm (doesn't follow a pattern).

AF is characterized by:

- Rapid (350–600 beats/min) and irregular atrial rhythm
- Reduced filling of the left and right ventricles (AF→ reduced filling → decreased cardiac output → fatigue, palpitations, shortness of breath).

- 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

AF begets AF

- AF causes remodelling:
  - **Electrical**: shortening of refractory period. (Refractory period is the amount of time it takes for an excitable membrane to be ready for a second stimulus once it returns to its resting state following excitation).
  - **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) (due to abnormal blood flow) on the walls of the atria that can dislodge (embolize), leading to stroke and systemic embolism (stroke is the worst complication of AF)
- Reduction in cardiac output can precipitate heart failure leading to:
  - Peripheral oedema.
  - Pulmonary oedema.

Causes of Atrial Fibrillation:

Cardiac Causes:

- Heart disease (hypertensive, ischemic, valvular)
- Pericarditis
- Cardiac tumors: Atrial Myxoma
- Sick sinus syndrome (sinus node doesn't work properly)
- Cardiomyopathy (Hypertrophic, idiopathic dilated)
- Post-coronary bypass surgery

Note(s):

Valvular heart disease:

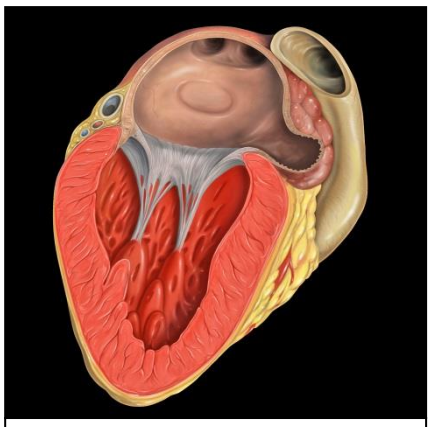
- Rheumatic: mitral stenosis (commonest)
- Non-rheumatic: aortic stenosis, mitral regurgitation

Non-Cardiac Causes (usually the cause in younger patients)

- Pulmonary (COPD, Pneumonia, Pulmonary embolism)
- Metabolic (Thyroid disease: hyperthyroidism, electrolyte disorder)
- Toxic: alcohol ('holiday heart' syndrome)

Diagnosis of AF

- Signs and symptoms (palpitation, shortness of breath, fatigue, dizziness, syncope, chest pain or may present as stroke!)
- Electrocardiography
- Laboratory tests (e.g. Thyroid hormone tests)
- Holter monitoring (Portable ECG digital recorder that used in the investigation of patients with suspected arrhythmias and it can record for 24h or 3 weeks " or 1-7 days as mentioned in Davidson" used for diagnosis as well as to guide therapy)
- Transoesophageal echocardiography (TEE) (the only way to rule out clots in left atrium)
- Exercise testing
- Chest radiography (mainly to rule out pulmonary causes of AF)



Left atrial appendage shown at upper right

Note(s):

Where might a clot form in left atrium?
In the left atrial appendage (LAA), because of the presence of trabeculae while the rest of the left atrium is smooth.

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

Signs and symptoms

Causes	Sign/Symptoms
• Irregular heart beat	• Irregularly irregular pulse • Palpitations
• Decreased cardiac output	• Fatigue. • Diminished exercise capacity. • Breathlessness (dyspnea) • Weakness (asthenia)
• Hypotension	• Dizziness and fainting (syncope)
• Cardiac ischemia	• 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

Selected patients: (patients with suspected clot).

- Transesophageal echocardiogram (TEE)

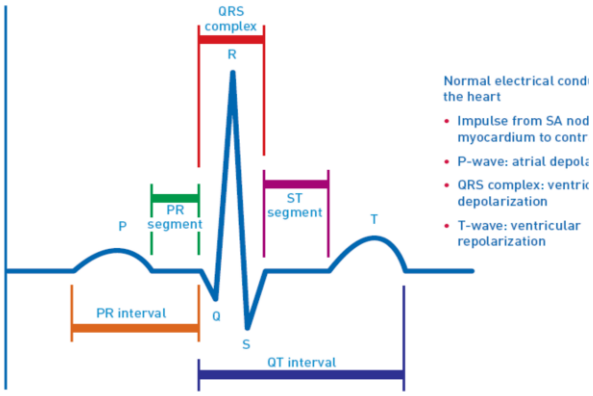
DR. DIDN'T MENTION SLIDE 20

Electrocardiogram

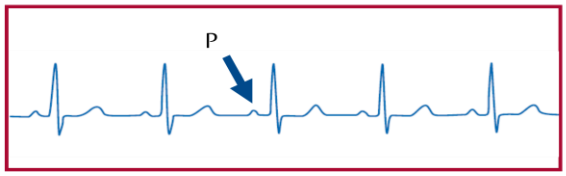
- Assesses the electrical activity of the heart
- Essential for all patients with suspected AF, to identify
  - Abnormal heart rhythm (verify AF)
  - Left ventricular hypertrophy
  - Pre-excitation
  - Bundle-branch block
  - Prior MI
  - Differential diagnosis of other atrial arrhythmias

Eletrocardiogram: Normal sinus rhythm

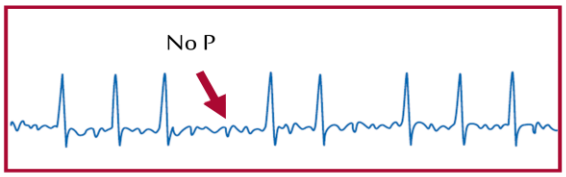
- Impulse from sinoatrial (SA) node stimulates myocardium to contract
- **P-wave:**
  atrial depolarization
- **QRS complex:**
  ventricular depolarization
- **T-wave:**
  ventricular repolarization



Electrocardiogram: loss of P wave in AF



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

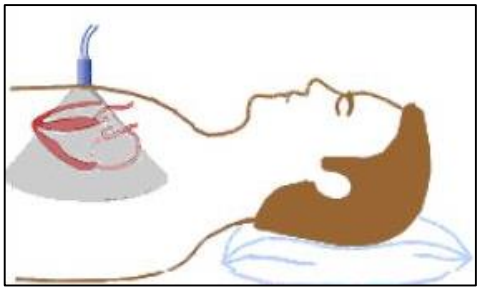


- AF
  - Heart rate increased (tachyarrhythmia)*
  - Irregular rhythm
  - No P wave
  - Irregular baseline

* Reduced heart rate (bradyarrythmia) may also be observed

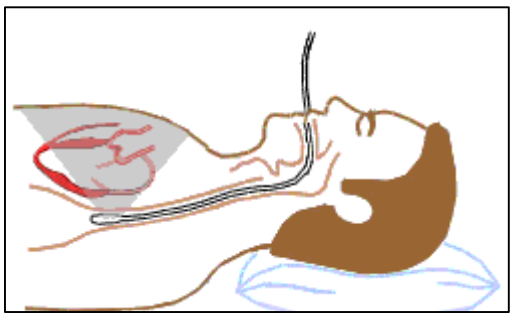
Transthoracic echocardiography (TTE)

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



Transesophageal 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

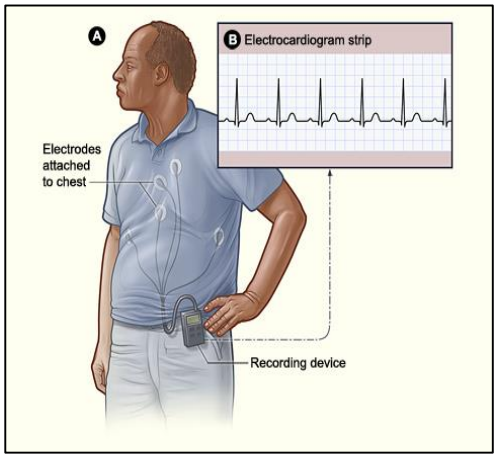


Laboratory tests

- 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)

Holter monitor

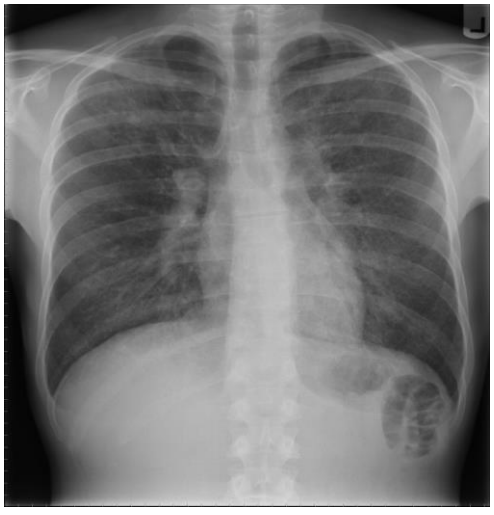
- Portable ECG device
- Continuous monitoring for a short period of time (typically 24 hours)
- Useful for:
  - Detecting asymptomatic AF
  - Evaluating patients with paroxysmal AF (ends by itself)
  - Associating symptoms with heart rhythm disturbance
  - Assessing response to treatment



Chest Radiography

When clinical findings suggest 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:

Classification	Definition
1- First detected	First recognized episode of AF
2- Recurrent -Paroxysmal -Persistent	≥2 episodes of arrhythmia AF that terminates spontaneously (lasts less than 24 hours) AF than persists for >7 days but can be converted with cardioversion (electrical shock)
3-Permanent	AF that cannot be terminated by cardioversion, and long-standing AF (>1 year) where cardioversion not indicated/not attempted

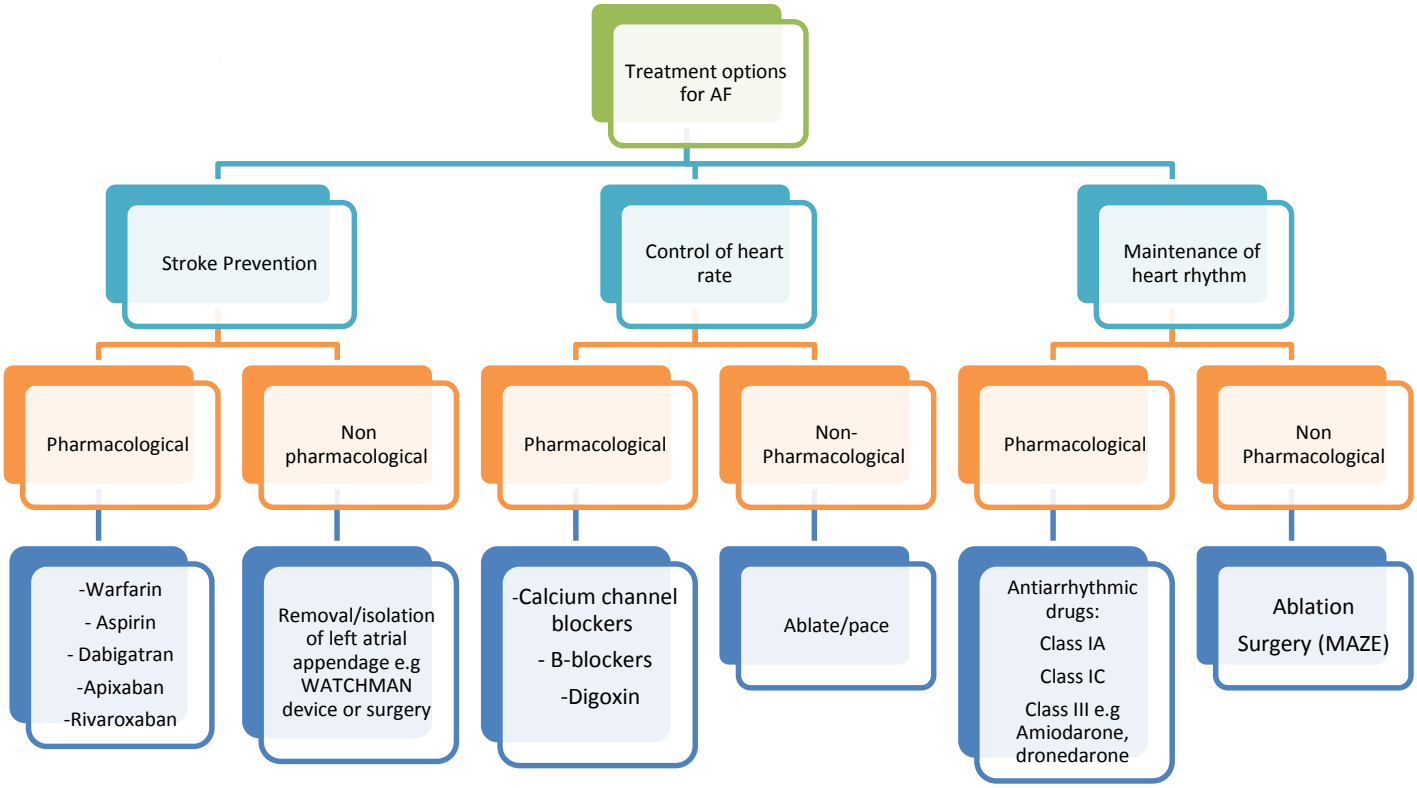
Classification of AF: joint guidelines of the ACC, AHA and ESC (2)

Classification	Definition
Lone or primary	AF without clinical/ECG evidence of cardiopulmonary disease (no underlying disease)
Secondary	AF associated with cardiopulmonary disease (e.g. myocardial infarction or pneumonia)
Non-valvular	AF that is not associated with damage to the heart valves (e.g. rheumatic mitral valve disease, prosthetic heart valve or mitral valve repair)

Treatment of Atrial Fibrillation (3 strategies):

- Prevention of thromboembolism
- Rate control
- Restoration and maintenance of sinus rhythm

Treatment options for AF (Rate control)



Note(s):

- Efficacy of Warfarin → reduces risk of stroke by 68%
- Efficacy of Aspirin → reduces risk of stroke by 20%

-Warfarin has drug- drug and food- drug interaction (e.g. food containing Vitamin K)
-Warfarin must be controlled. If INR > 3 → bleeding, if INR < 2→ clot formation (An INR of 2 to 3 is the goal range)

-The drugs Dabigatran, Apixaban, Rivaroxaban (the "ban" family) have neither drug- drug interaction, nor food-drug interaction so they can replace Warfarin.

-Watchman device: The device is meant to capture blood clots and prevent them from migrating into the circulation and causing a stroke. Watchman would provide an alternative to warfarin and other blood thinners for preventing stroke and systemic embolism.

-B blockers are contraindicated in patients with Asthma so Calcium channel blockers are used instead and these two drugs have the same efficacy while Digoxin is the least efficient.

-Cardiac Ablation (MAZE): Cardiac ablation works by scarring or destroying tissue in your heart that triggers an abnormal heart rhythm.

- Amiodarone is the most powerful with unpleasant side effects but they take a long time (years) to appear so can be given to elderly patients.

Prevention of Thromboembolism:

The $CHADS_2$ Index: Stroke Risk Score for Atrial Fibrillation (a clinical prediction rule for estimating the risk of stroke in patients with non-rheumatic atrial fibrillation)

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

The CHA_2DS_2VASc Index is more accurate than $CHADS_2$

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

Note(s):

-If patient was at a high risk according to CHADS score then he must take Warfarin or one of "ban" family drugs.

-If patient was at low risk then Aspirin is given

-CHADS score is not used for patients with Valvular heart disease. Why? Because they already are at a high risk thus the CHADS score doesn't apply to them.

-If CHADS score applies on a patient then he/she must take anticoagulants for long term but if patient is not under CHADS score (e.g. valvular heart disease) then he/she must take anticoagulants but for short term after shock.

-If palpitation persists for 2 days or more than a clot is probably forming.

Restoration of Sinus Rhythm (after rate control is achieved)

"Rhythm-control therapies"

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

Note(s):

- Cardioversion can be done using an electric shock or drugs.

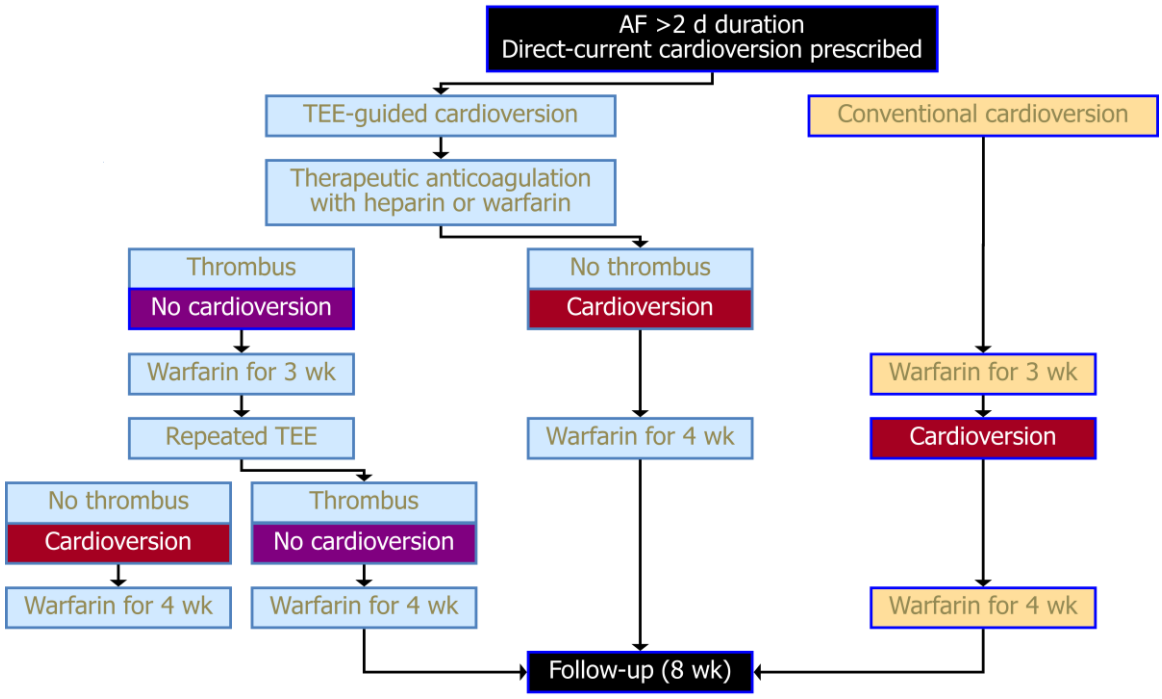
- Candidates for cardioversion include those who are hemodynamically unstable, those with worsening symptoms, those who are having their first ever case of AF.

- After shocking the patient, anticoagulants must be given for 4 weeks because patient might develop a clot. DO NOT DISMISS A PATIENT AFTER SHOCK

-Use pharmacological cardioversion only if electrical cardioversion fails or is not feasible. (e.g. Amiodarone, parental ibutilide, procainamide,…)

-To avoid waiting 3 weeks for anticoagulants, obtain TEE to image the left atrium. If no thrombus is present → start IV Heparin and perform cardioversion within 24 hours. Patients still require 4 weeks of anticoagulation after cardioversion.

TEE-guided cardioversion: ACUTE study design



Atrial Flutter

Pathophysiology: From Step Up page 25

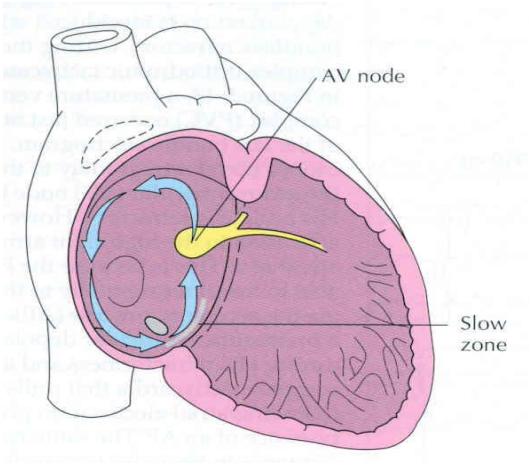
One irritable automaticity focus in the atria fired at about 250 to 350 bpm, giving rise to regular atrial contractions.

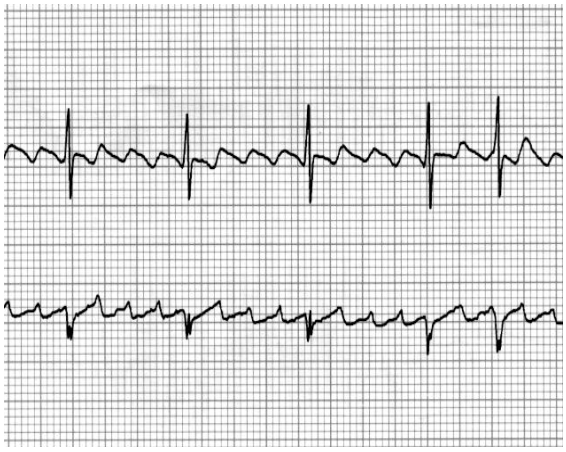
Atrial rate around 300 bpm. Ventricular rate is one half to one third of atrial rate. The long refractory period in the AV node allows only one out of every two or 3 flutter waves to conduct to the ventricles.

Note(s):

Atrial Flutter is different from atrial fibrillation in that the electrical signal travels as a single large wave always in one direction around and around the atrial muscle mass.

(Atrial flutter → one irritable automaticity focus, while Atrial Fibrillation → multiple foci in the atria in a chaotic pattern)





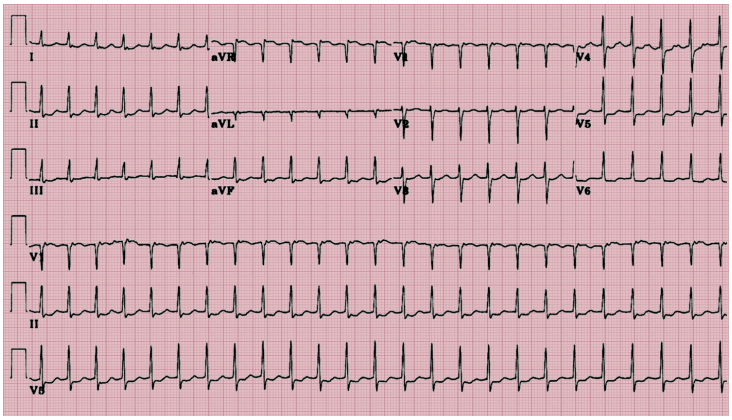
ECG provides a saw-tooth baseline, with a QRS complex appearing after every second or third "tooth" (P wave). Saw-tooth flutter waves are best seen in the inferior leads (II, III, aVF).

Rx of Atrial Flutter: (the treatment of atrial fibrillation and Atrial Flutter are similar)

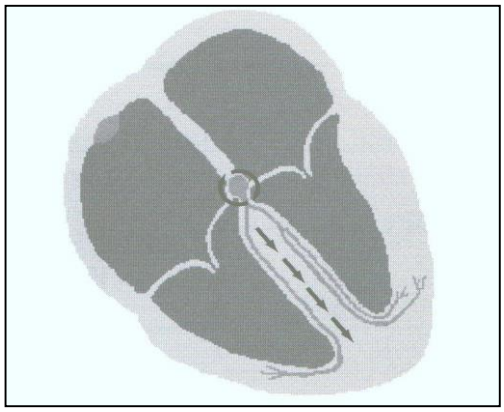
- Unstable pt (i.e. low BP / CP / AMS):
  - Synchronized cardioversion as per ACLS
  - 50J → 100J → 200J → 300J → 360J
- Stable pt:
  - Rate control - just like atrial fibrillation (AFib)
  - Elective cardioversion - just like AFib
  - Anti-coagulation – just like AFib
  - Refer for Ablation

SVT (Supraventricular Tachycardia):

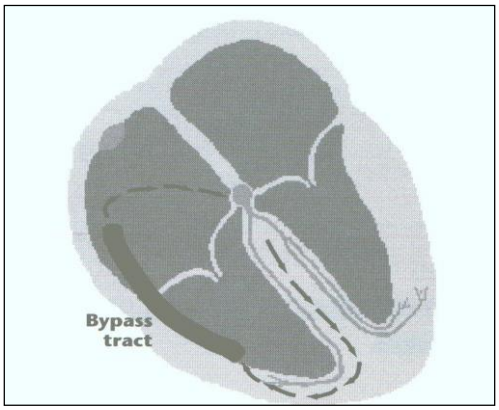
- Arrhythmias of supraventricular origin using a re-entrant mechanism with abrupt onset & termination
- AVNRT (60%)
- AVRT (30%)
- Atrial tachycardia (10%)



AVRT-Narrow complex

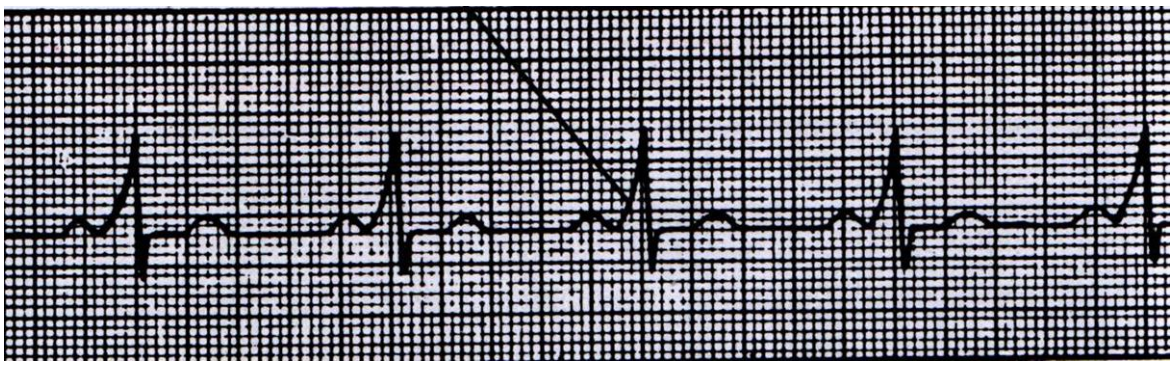


Atrioventricular Nodal Re-entrant Tachycardia (AVNRT)



Atrioventricular Re-entrant Tachycardia (AVRT)

Wolf-Parkinson-White (WPW) Syndrome: presence of an accessory conduction pathway from atria to ventricles through the bundle of Kent→ causes premature ventricular excitation because it lacks the delay seen in the AV node. (Step-up to Medicine, page 29)



ECG: narrow complex tachycardia, short PR interval and a delta wave (upward deflection seen before the QRS complex).

Medical treatment:

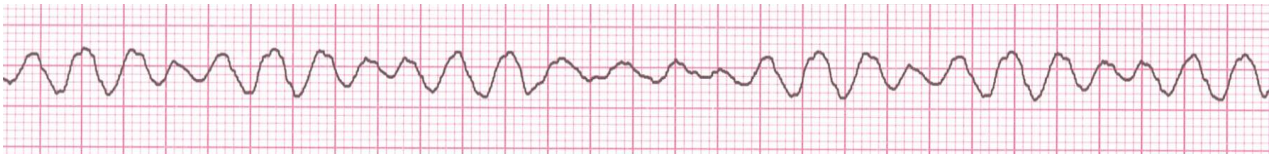
- Medical therapy. (Avoid drugs active on the AV node e.g. Digoxan, verapamil because they may accelerate conduction through the accessory pathway. Type IA or IC antiarrhythmics are better choices)
- Radio Frequency Ablation.

Other arrhythmias

- Ventricular Tachycardia (defined as rapid and respective firing of three or more PVC "premature ventricular contraction" in a row at a rate of between 100 and 250 Bpm)
- Ventricular Fibrillation

Ventricular Fibrillation

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

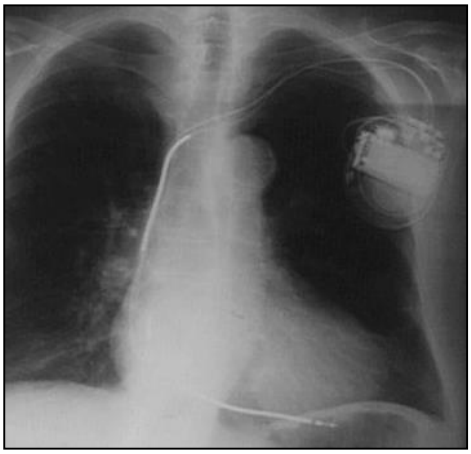


Treatment options

- Treat the underlying cause
- Automatic Implantable defibrillators

This is a medical emergency!! Immediate defilation and CPR indicated.

Drugs cannot convert VF by themselves defibrillation is the key.



ECG (Step-up to Medicine, page31)

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

SUMMARY

Arrhythmia

An abnormality of cardiac rhythm is called a cardiac arrhythmia. Arrhythmia may cause sudden death, syncope, dizziness, palpitations or no symptoms at all. Paroxysmal arrhythmias may not be detected on a single ECG recording. Twenty-four-hour ambulatory ECG monitoring and event recorders are often used to detect arrhythmias causing intermittent symptoms. There are two main types of arrhythmia:

Bradycardia: the heart rate is slow (< 60 beats/min). Slower heart rates are more likely to cause symptomatic arrhythmias. *Tachycardia:* the heart rate is fast (> 100 beats/min). Tachycardias are more likely to be symptomatic when the arrhythmia is fast and sustained.They are subdivided into *supraventricular tachycardias* (SVTs), which arise from the atrium or the atrioventricular junction, and *ventricular tachycardias*, which arise from the ventricles.

Atrial fibrillation (AF)

This is the most common arrhythmia and occurs in 5–10% of patients over 65 years of age. It also occurs, particularly in a paroxysmal form, in younger patients. Atrial activity is chaotic and mechanically ineffective. The AV node conducts a proportion of the atrial impulses to produce an irregular ventricular response – giving rise to an irregularly irregular pulse. In some patients it is an incidental finding; in others symptoms range from palpitations and fatigue to acute heart failure. AF is associated with a five-fold increased risk of stroke, primarily as a result of embolism of a thrombus that has formed in the atrium. There are no clear P waves on the ECG, only a fine oscillation of the baseline (so-called fibrillation or f waves).

Questions

1) Which one of the following are the hallmark ECG changes in AF?
   a. Presence of QRS complex with regular RR intervals.
   b. Absence of QRS complex with regular RR intervals.
   c. Presence of p wave with Irregularity of RR intervals.
   d. Absence of p wave with Irregularity of RR intervals.

2) Which one of the following used to rule out left atrium clots?
   a. Transthoracic echocardiography (TTE).
   b. Transoesophageal echocardiography (TEE).
   c. Holter monitor.
   d. WATCHMAN device.

3) In a 62 years-old diabetic female patient who is a known case of hypertension. What is her CHA_2DS_2VASc score?
   a. 2 points.
   b. 3 points.
   c. 4 points.
   d. 5 points.

432 Medicine Team Leaders

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Answers:
1st Questions: D
2nd Questions: B
3rd Questions: B