

Medicine Team Notes

Arrhythmias

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427 Medicine Notes

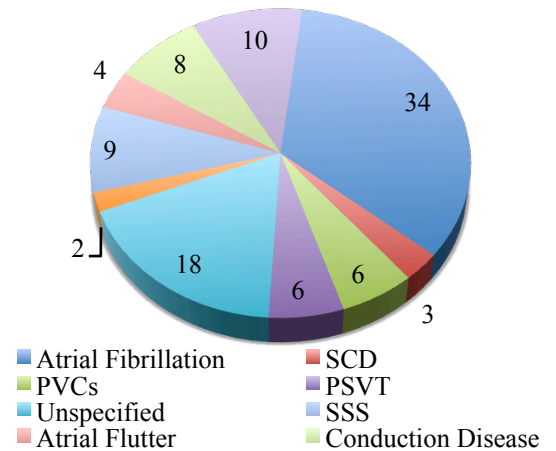
ARRHYTHMIA: the absence of normal rhythm

ATRIAL FIBRILLATION

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

EPIDEMIOLOGY:

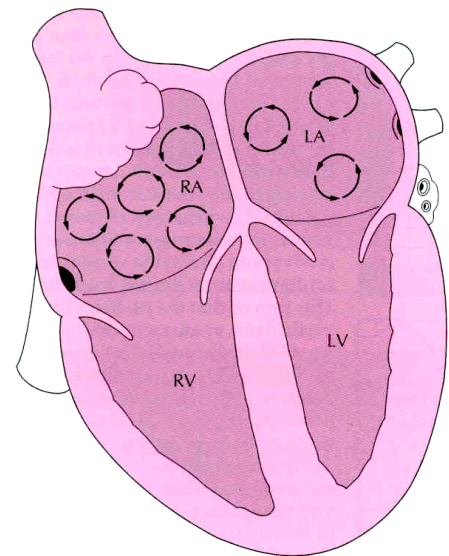
- 2.3 million people in North America
- 4.5 million in EU
- In the 20-year AF admission have increased by 66%.
- Increases with age
- \$ 15.7 billion annually in EU
- Estimated prevalence of AF is 0.4% to 1% in the general pop. 8% in pt. >80 years
- Common and age-dependent 2 - 4% over age 60.
- Significant risk of stroke 4% per year (Framingham Study) as a result of stagnation of the blood in the Left atrial appendage.
- Annual Incidence 0.1% Per Year



So remember as you grow up the incidence increases + men are more prone to developing atrial fibrillation

PATHOPHYSIOLOGY:

- Contraction is controlled by the sinoatrial (SA) node in normal conditions
- Impulse is initiated in the SA node > both atria contract/ then the ventricle receives the impulse from AV node then it contracts.
- Any cell in the heart can give you an impulse, but they all listen to the master the SA node.
- In Atrial Fibrillation you lose this autonomous organized electric activity and the cells in the atria start to fire erratically in a chaotic manner.
- And then the AV node is confused and bombarded by many activities that it cannot conduct all of them.
 - Normally, the AV node is responsible for slowing down the ventricular beats to protect it.
- So you get atrial fibrillation which is a type of micro re-entry



In atrial fibrillation the normal heart rhythm is disrupted and you get:

1. AF is characterized by:

- a. Rapid (350–600 beats/min) and irregular atrial rhythm
- b. Reduced filling of the left and right ventricles

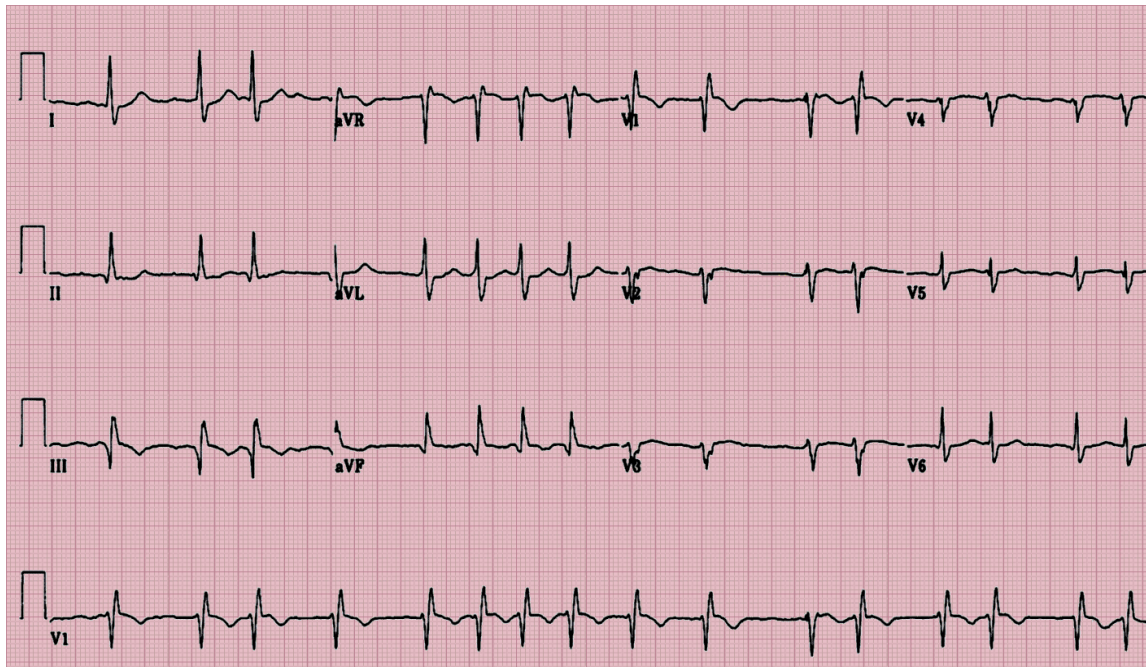
2. Conduction of most impulses from the atria to ventricles is blocked at the AV node

3. Contraction of the ventricles can be:

- a. Irregular and rapid (110–180 beats/min; tachycardia)
- b. Irregular and slow (<50 beats/min; bradycardia)
- c. Normal

4. Cardiac output can be reduced

ECG of Atrial Fibrillation:



Two Important points about the ECG that are the hallmarks for atrial fibrillation:

1. Absent P wave

- The P wave is: mechanically an atrial contraction and electrically from the SA node atrial depolarization, so in AF it disappears because the impulse is missing

2. Irregularly irregular rhythm

- Its irregularity arises from the fact that the impulse is getting to the AV node from different locations in the Atria and not the SA node

A CONCEPT: AF BEGETS AF

- The longer the heart is in atrial fibrillation the longer the chances are of atrial fibrillation to occur again, and that happens because the atrial fibrillation causes:
 - Structural changes: enlargement of atrial cavities
 - Electrical conductivity changes: shortening of refractory period
- That's why returning the patient's sinus rhythm (i.e. By cardioversion) is crucial in treatment of AFEB
- Keep in mind that Many episodes of AF resolve spontaneously
- Over time AF tends to become persistent or permanent.

CONSEQUENCES OF ATRIAL FIBRILLATION:

1. Formation of blood clots (thrombosis) on the walls of the atria that can dislodge (embolize), leading to stroke and systemic embolism.
 - a. The stagnation of blood in the atria causes thrombus formation
 - b. The presence of the appendage in the left atrium makes it more prone to forming clots in cases of fibrillation than the right atrium
 - c. The most devastating complication of Atrial Fibrillation is stroke
2. Low cardiac output due to decreased filling (no atrial contraction "kick")
 - a. Causing heart failure that precipitates
 - i. Peripheral oedema
 - ii. Pulmonary oedema
3. Rate-related atrial myopathy and dilatation

CAUSES:

Cardiac

- Hypertensive heart disease
- Ischemic heart disease
- Valvular heart disease
 - Valvular heart disease is the most common cause in this region
 - 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

Non-Cardiac

- Pulmonary :
 - COPD.
 - Pneumonia.
 - Pulmonary embolism.
- Metabolic :
 - Thyroid disease: hyperthyroidism.
 - Electrolyte disorder like hypokalemia.
- Toxic: alcohol ('holiday heart' syndrome).
- In young patients with atrial fibrillation think of alcohol use or thyroid problems

DIAGNOSIS:

I. Clinical presentation:

- a. With or without detectable heart disease
- b. Episodic
 - i. Symptoms may be absent or intermittent
 - ii. Up to 90% of episodes may not cause symptoms
- c. Symptoms vary according to
 - i. Irregularity and rate of ventricular response
 - ii. Functional status
 - iii. AF duration
 - iv. Patient factors
 - v. Co-morbidities

2. Signs & Symptoms

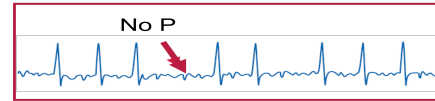
Cause	Sign/Symptom
Irregular heart beat	- Irregularly irregular pulse - Palpitations
Decreased cardiac output	- Fatigue - Diminished exercise capacity - Breathlessness (dyspnoea) - Weakness (asthenia)
Hypotension	Dizziness and fainting (syncope)
Cardiac ischaemia	Chest pain (angina)
Increased risk of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

3. History

- a. Clinical conditions associated with AF
 - i. Underlying heart conditions (e.g. valvular heart disease, heart failure, coronary artery disease, hypertension)
 - ii. Other reversible conditions
- b. Family history
 - i. Familial AF (lone AF in a family)
 - I. Lone AF: (less than 40) with atrial fibrillation and no reason underlying it, idiopathic.
 - ii. AF secondary to other genetic conditions (familial cardiomyopathies)
- c. Type of AF
 - i. First episode, paroxysmal, persistent, permanent
 - ii. Triggers – e.g. emotional stress, alcohol, physical exercise, gastroesophageal disease
 - iii. Specific symptoms
 - I. Diastolic rumbling murmur
 - iv. Response to any treatments administered

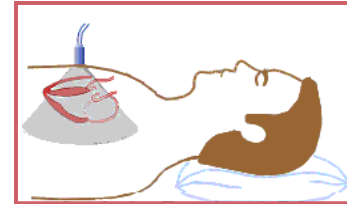
4. ECG

- a. Heart rate increased (tachyarrhythmia)*
- b. Irregular rhythm
- c. No P wave
- d. Irregular baseline



5. TTE (Transthoracic Echocardiography)

- a. Non invasive method
- b. Used to identify
 - i. Size and functioning of atria and ventricles
 - ii. Ventricle hypertrophy
 - iii. Pericardial disease
 - iv. Valvular heart disease



6. Lab tests: at least once

7. Holter Monitor

- a. Used to see if treatment is effective in 24 hours
- b. Portable ECG device

8. TEE (Transoesophageal Echocardiogram)

- a. Only in selected patients
- b. TEE is better when a clot is suspected, TTE is not sensitive
- c. Endoscope-like device
- d. High quality images of cardiac structure and function
 - i. Particularly the left atrial appendage, the most common site of thrombi in patients with AF
- e. Useful for:
 - i. Accurate assessment of risk of stroke
 - ii. Detection of low flow velocity ('smoke' effect)
 - iii. Sensitive detection of atrial thrombi

9. CXR (Chest X-Ray): helps in evaluation of lung pathology or underlying causes

CLASSIFICATION

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

Classification	Definition
Lone or primary	AF without clinical/ECG evidence of cardiopulmonary 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

There are three main strategies:

1. Prevention of thromboembolism
 - a. Prevention of stroke is the most important management strategy
2. Rate control
3. Restoration and maintenance of sinus rhythm

FIRST: PREVENTION OF THROMBOEMBOLISM

- We use aspirin with low risk group
- And use warfarin with high risk group
 - Lots of adverse effects and dietary restrictions
 - Doctor said that 2011 guidelines recommend Dabigatran as the drug of choice
 - Dabigatran: is as good as warfarin, does not need monitoring, no drug-drug interaction or drug food interaction, given to high risk patients
- We determinate these group by **CHA₂DS₂VASc index**
 - This index is only used in patients with non-valvular disease! Patients with valvular diseases should be treated immediately because they are at risk!

Weight (points)

Congestive heart failure or LVEF \leq 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

EXAMPLE: 70-year-old female, history of stroke and hypertension develops AF. The drug of choice for prevention of thromboembolism would be?

Answer: First check the index, in this case 5, so she should be treated with warfarin or dabigatran.

EXAMPLE 2: 20-year-old male with history of rheumatic valve disease develops AF, what is the index for prevention of thromboembolism in this patient?

Answer: No index, patient is already at high risk due to valvular disease.

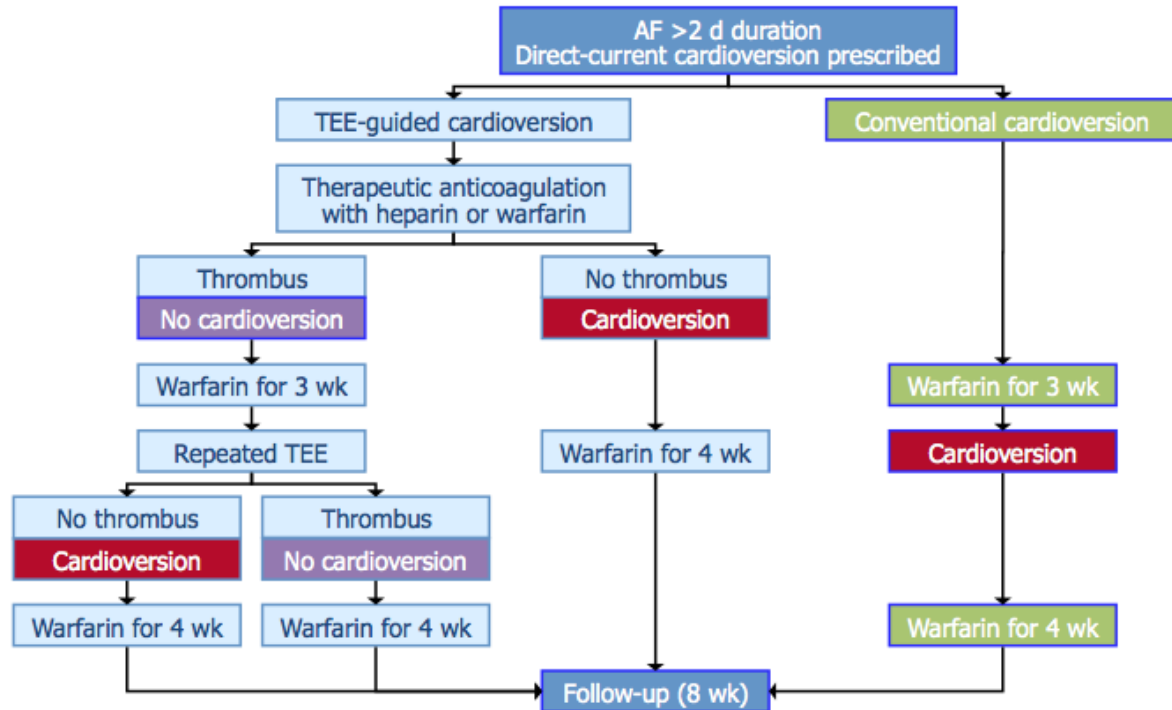
So remember: To treat thromboembolism: 2 things, categorize and choose a drug

SECOND: RATE CONTROL WITH AV BLOCKERS

THIRD: RESTORATION OF SINUS RHYTHM

- The objective of rhythm-control therapy is to restore (cardioversion) and maintain normal sinus rhythm
 - Acute: cardioversion
 - Long run (maintaining): anti arrhythmic medication
 - Best is amiodarone, but it's like using a big gun to kill a fly
 - So it's given to old patients, young patients will develop effects in 10 years so you give them class IC: **Flecainide, Propafenone**
- 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
 - When you shock a patient the atrias are stunned so you must use anticoagulant before and after

TEE-GUIDED CARIOVERSION: ACUTE STUDY DESIGN



DC = direct-current; TEE = transoesophageal echocardiography

Klein AL et al. N Engl J Med 2001;344:1411–20

NOTE: the warfarin is used before and after cardioversion

SUMMARY:

TREATMENT OPTIONS FOR AF

STROKE PREVENTION	CONTROL OF HEART RATE	MAINTENANCE OF SINUS RHYTHM
PHARMACOLOGIC <ul style="list-style-type: none"> Warfarin Aspirin Dabigatran (Drug of choice) Apixaban Rivaroxaban 	PHARMACOLOGIC <ul style="list-style-type: none"> Ca²⁺-channel blockers β-blockers Digoxin 	PHARMACOLOGIC <ul style="list-style-type: none"> Antiarrhythmic drugs <ul style="list-style-type: none"> – Class IA – Class IC – Class III: e.g. amiodarone, dronedarone
NON-PHARMACOLOGIC <ul style="list-style-type: none"> Removal/isolation of left atrial appendage, e.g. WATCHMAN® device (like an umbrella inserted via catheter used to cover up the left atrial appendage and prevent embolism) or surgery 	NON-PHARMACOLOGIC <ul style="list-style-type: none"> Ablate/pace 	NON-PHARMACOLOGIC <ul style="list-style-type: none"> Ablation (done to the pulmonary vein because it's been proven that many of AF arise from the pulmonary vein) Surgery (MAZE)

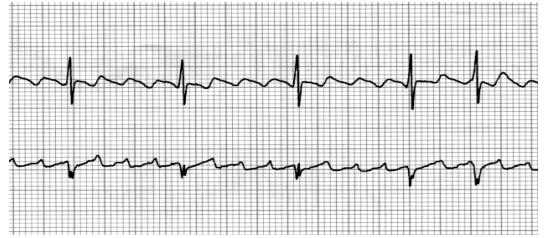
•ACE = angiotensin-converting enzyme

•Rate control are AV node blockers

•Calcium blockers like: Verapamil, Diltiazem

ATRIAL FLUTTER

- A type of macro re-entry
- Saw tooth appearance on ECG
- RX:
 - Unstable → cardioversion
 - Stable → Refer for Ablation



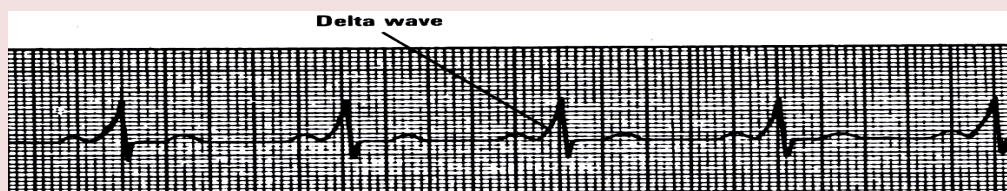
SUPRAVENTRICULAR TACHYCARDIA (SVT)

- Arrhythmias of supraventricular origin using a re-entrant mechanism with abrupt onset & termination
- AVNRT (60%)
 - Atrioventricular nodal re-entry tachycardia
 - All of the circuit is in the AV node
 - Rx is ablation of part of the AV node
- AVRT (30%)
 - AV re-entrant tachycardia
 - There is an extra bundle
 - Rx is by ablation
- Atrial tachycardia

WOLF-PARKINSON-WHITE (WPW) SYNDROME:

- It's a type of AVRT
- Short PR
- Presence of Delta waves on ECG
- Rx:
 - 1st choice – procainamide 20mg/min IV (max 17mg/kg).
 - § Drugs to avoid – AV nodal blocking agents!

Radio-frequency ablation curative > 95% cases



OTHER ARRHYTHMIAS:

- VT: ventricular tachycardia: wide complex tachycardia
- VF: Ventricular fibrillation: chaotic activity of the ventricle

