

ATRIAL FIBRILLATION

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- C. ANTICOAGULATION

00:36

I. PATHOPHYSIOLOGY & CAUSES

- Atrial fibrillation is a prevalent form of arrhythmia, classified specifically as a type of supraventricular tachycardia (SVT)

A. CARDIAC CAUSES

01:18

1. ↑ Left Atrial Pressure (LAP)

- **Pathophysiology:**
 - ↑ Left atrial pressure → Atrial dilation and remodeling → Disrupts electrical pathways → Creating re-entrant circuits that can send electrical impulses to the ventricles → Results in an irregular and at times, fast heartbeat

Causes:

a) Mitral Stenosis

- **Pathophysiology:**
 - Mitral valve dysfunction can obstruct the flow of blood from LA → LV which results in ↑ Left atrial pressure
- Hints: Assess for a **Diastolic Murmur** at Apex

Valvular AF: Interfering with the normal flow of blood due to valvular abnormality

b) Congestive Heart Failure

- **Pathophysiology:**
 - Impaired ventricular filling and impaired contractility → ↑ LV end-diastolic pressures (LVEDP) → ↑ LAP
- Hints: Listen for **Crackles/Rales** on Lung auscultation

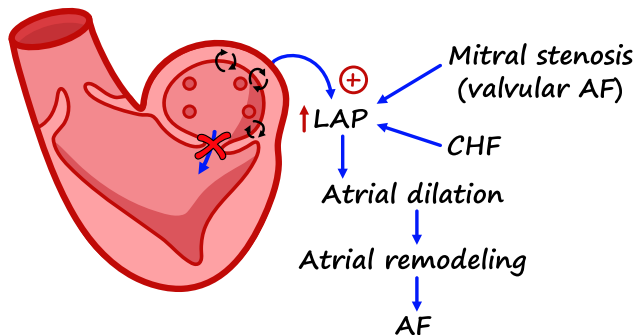
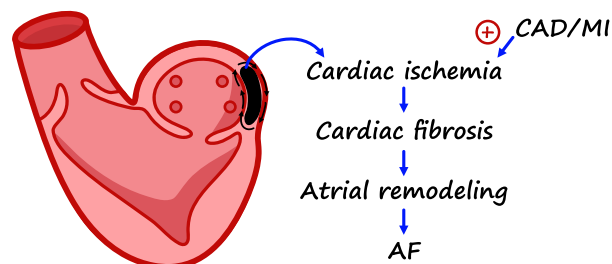
2. Cardiac Ischemia

- **Pathophysiology:**
 - Ischemic cardiac tissue → Cardiac fibrosis → Atrial remodeling → Produces **Re-entrant circuits** → Causing an **Irregular rhythm**

Causes:

c) CAD or MI

- **Pathophysiology:**
 - MI → Cardiac tissue injury → Fibrotic scar → Electrical remodeling → Re-entrant circuits
- Hints: Look for **anginal chest pain**



1. Pulmonary Disease

- **Pathophysiology:**
 - Hypoxia → Atrial cells in the vicinity of the pulmonary veins generate ectopic activity → Primary stimulus for AF

Causes of Hypoxia:

Pneumonia

- Alveolar filling → V/Q mismatch → Hypoxia
 - More of an acute presentation

COPD

- Alveolar hypoventilation → V/Q mismatch → Hypoxia
 - More of a chronic presentation

Pulmonary Embolism

- Pulmonary artery occlusion → V/Q mismatch → Hypoxia
 - More of an acute presentation

2. ↑ Catecholamines (NE, Epi)

- **Pathophysiology:**
 - Stimulates the β_1 receptors in the atrial cells → **Ectopy** → Primary stimulus for AF

Causes of ↑ Catecholamines:

Sepsis

- Infection → Systemic vasodilation → ↑ Catecholamine response

Post-operative

- Tissue damage produces a stress response → ↑ Catecholamine response

Pheochromocytoma

- Adrenomedullary tumor → ↑ Catecholamine secretion

Thyrotoxicosis

- ↑ T3 and T4 → ↑ Sensitivity of the β_1 receptors to catecholamines

Sympathomimetics

- Cocaine, Methamphetamine, PCP → ↑ Catecholamine-like response

3. Electrolyte Disturbances

- **Pathophysiology:**
 - Hypokalemia and Hypomagnesemia → Alter electrical activity in the atrial cells → Trigger **ectopy** → Primary stimulus for AF

Holiday Heart Syndrome

- Refers to the development of AF, typically after episodes of heavy drinking, often occurring during holidays or weekends causing severe electrolyte disturbances

Mnemonic: PIRATE

Pulmonary Diseases

(COPD, Pulmonary embolism, Pneumonia)

Infections/stress response

(↑ Catecholamines)

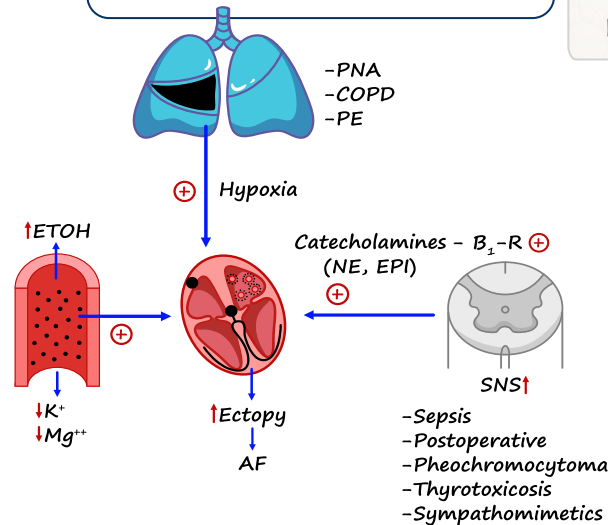
Remodeling of atria

(cardiac diseases)

Alcohol

Thyrotoxicosis

Electrolyte abnormalities (K/Mg)



C. PROGRESSION OF ATRIAL FIBRILLATION

- Based on the duration that a patient has experienced atrial fibrillation and the chances of them converting to sinus rhythm

Paroxysmal Atrial Fibrillation

- Duration: AF for < 7 days
- Chances of Conversion:
 - Remodeling has not yet occurred thus given the chance will revert to a normal sinus rhythm

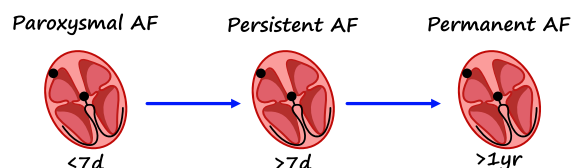
Persistent Atrial Fibrillation

- Duration: AF for > 7 days
- Chances of Conversion:
 - Cardiac remodeling due to persistent AF has begun to occur thus making it much more difficult, but possible to convert to normal sinus rhythm

Permanent Atrial Fibrillation

- Duration: AF for > 1 year
- Chances of Conversion:
 - Extensive cardiac remodeling has occurred, and it is unlikely the heart will be able to return to its native normal sinus rhythm

Progression of atrial fibrillation



II. COMPLICATIONS

A. THROMBOEMBOLI

14:09

● Pathophysiology:

- Ineffective atrial contractions → Suboptimal blood flow into the ventricles → Blood accumulates in the left atrium → **Stasis** of blood → ↑ Risk of a **thrombus**, usually on the left atrial appendage → ↑ Risk of an embolus breaking away from thrombus and obstructing flow to various organs

1. Effects of Embolus Obstruction

TIA or CVA

- Pathophysiology:
 - Embolus blocks cerebral blood flow → Cerebral ischemia/infarct
- Clinical Presentation:
 - Acute Neurological deficits based on vascular territory occluded

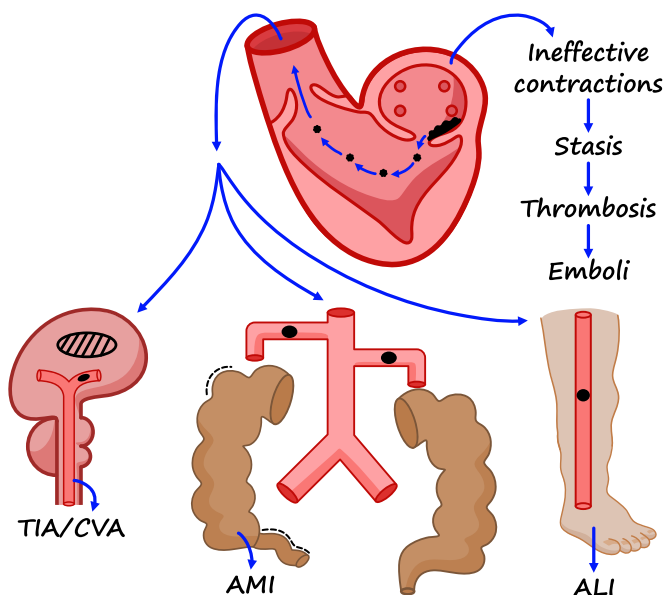
Acute Mesenteric Ischemia

- Pathophysiology:
 - Embolus blocks mesenteric blood flow → Mesenteric ischemia/infarct
- Clinical Presentation:
 - Abdominal pain out of proportion
 - ↑ Lactate levels

Acute Limb Ischemia

- Pathophysiology:
 - Embolus blocks lower limb blood flow → Acute limb ischemia/infarct
- Clinical Presentation:
 - ↓ Pulses and sensations of limbs
 - Weakness of limbs
 - Pallor of limbs

Thromboemboli



B. ACUTE HEART FAILURE

17:57

1. Shock

● Pathophysiology:

- AF with rapid ventricular rate (RVR) → Insufficient ventricular filling time → ↓ End-diastolic volume (EDV) → ↓ SV → ↓ CO → ↓ BP → **Shock**

● Clinical Presentation:

- ↓ BP (SBP < 90 mmHg)
- ↑ HR (> 150 bpm)

2. Pulmonary Edema

● Pathophysiology:

- AF with RVR → Insufficient ventricular filling time → ↓ EDV → ↓ SV → ↓ CO leading to pulmonary venous backflow → ↑ Pulmonary capillary wedge pressure → **Pulmonary edema**

● Clinical Presentation:

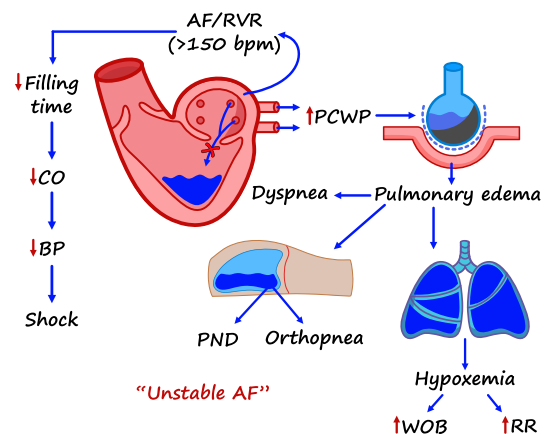
- Dyspnea (at rest or with exertion)
- PND or Orthopnea
- If severe enough → Pulmonary edema may present with hypoxia, ↑ RR, and ↑ WOB → **Acute Respiratory Failure**

Unstable Atrial Fibrillation

- Clinical Presentation when the HR > 150 bpm, sustained:

- ↓ BP (SBP < 90 mmHg)
- Dyspnea or Hypoxia

Acute heart failure



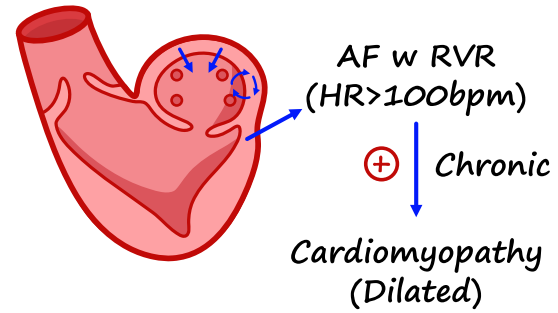
1. Dilated Cardiomyopathy

- **Pathophysiology:**
 - Chronic AF with RVR (HR > 150 bpm) → Develop Dilated Cardiomyopathy as compensation for ↓filling time and chronic tachycardia
- **Clinical Presentation:**
 - Biventricular HF findings (JVD, ascites, pitting, and pulmonary edema)
 - S3 heart sound

Please note:

- Patients may exhibit a normal or reduced heart rate in the context of AF, referred to as "AF with controlled ventricular response" and "AF with bradycardia," respectively

Tachycardia



III. DIAGNOSTIC APPROACH

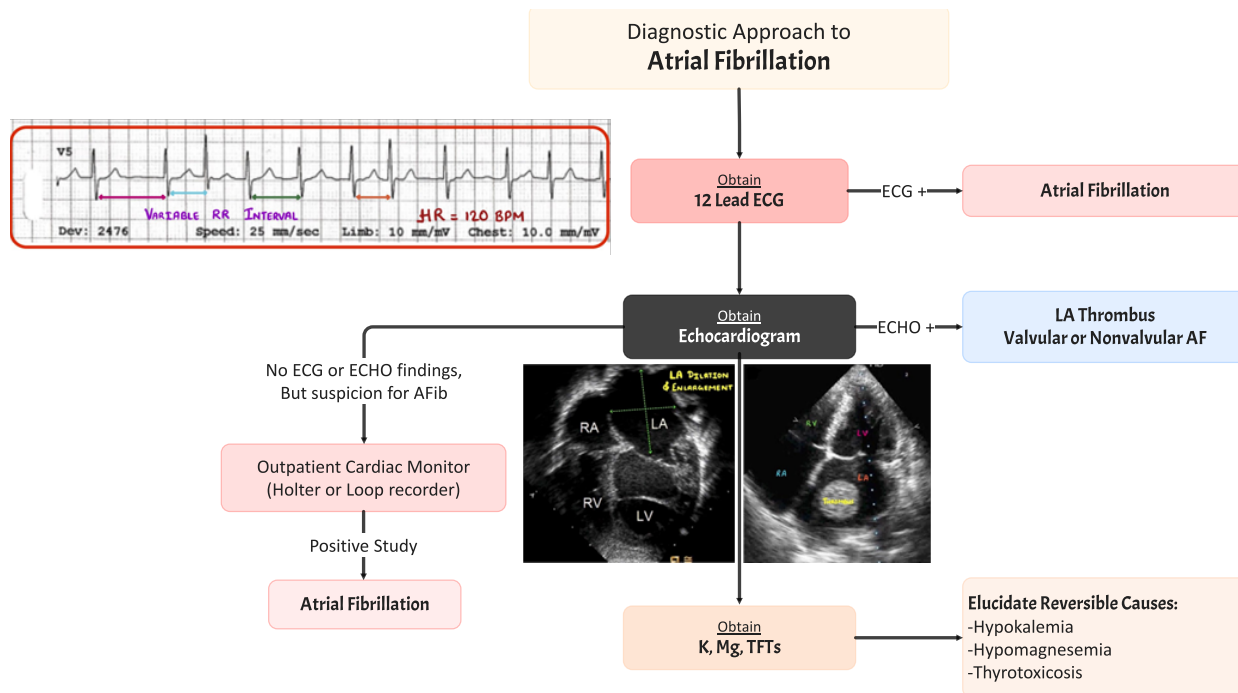


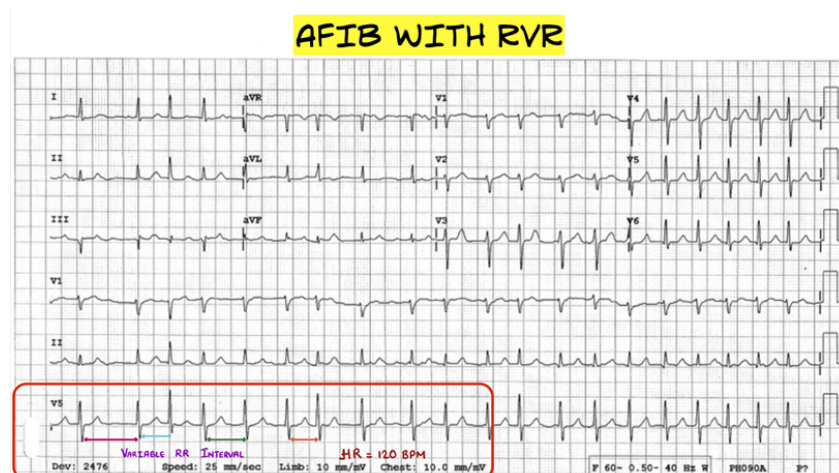
FIGURE 1. DIAGNOSTIC APPROACH TO ATRIAL FIBRILLATION.

A. 12-LEAD ECG

- **Primary Purpose:**
 - Diagnostic of current AF but may miss occult AF

Atrial Fibrillation with RVR ECG Findings:

- **Rhythm:** variable R to R interval (**irregular rhythm**) and absent p-waves
- **Rate:** Can vary in rate depending on the patient



B. ECHOCARDIOGRAM

- **Primary Purpose:**

- May identify a cardiac cause (Mitral stenosis, CHF) as the cause of AF based on ↑LAP and LA dilation
- May identify if a Thrombus is present thus identifying risk of embolic complications → Identifies need for anticoagulation and risks of cardioversion

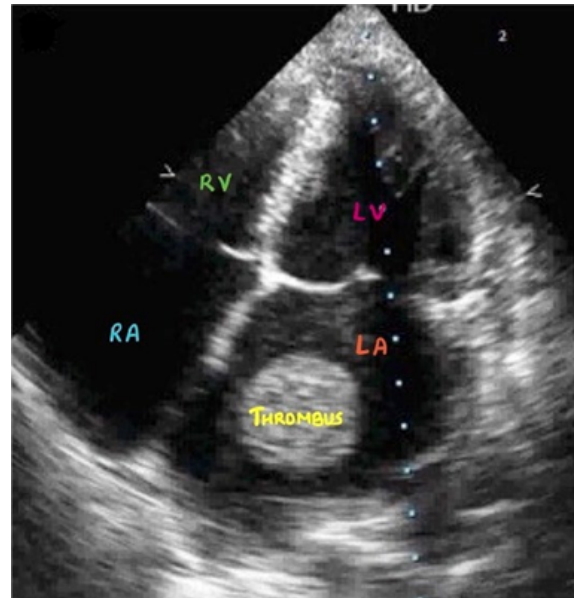
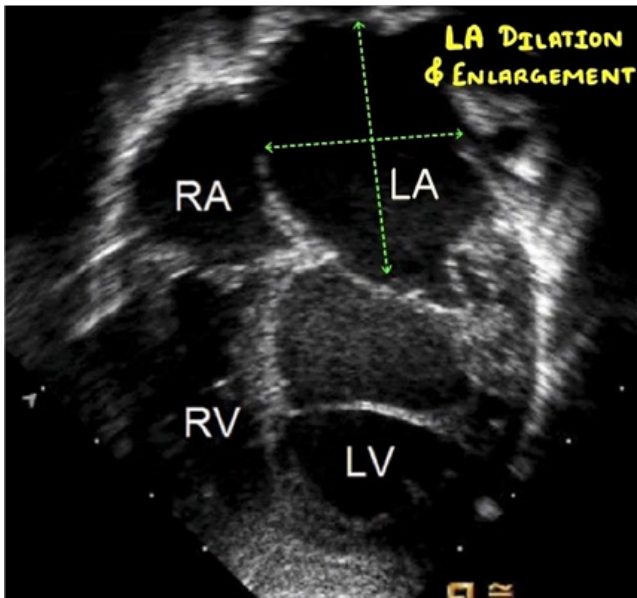


FIGURE 2. ECHOCARDIOGRAM FINDINGS SHOWING LA DILATION (LEFT) AND THROMBUS FORMATION (RIGHT).

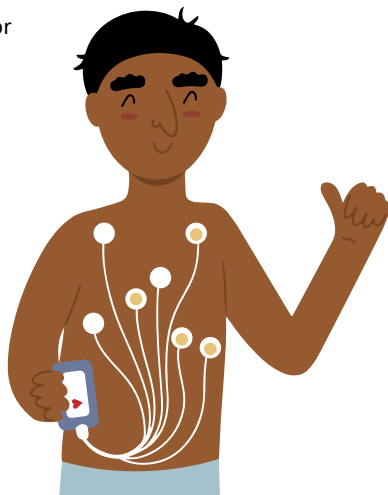
C. OUTPATIENT CARDIAC MONITOR

- **Primary Purpose:**

- Patients with no ECG or echocardiographic evidence to support AF and suspicion for AF remains high → Obtain continuous recording and analysis of cardiac electrical activity for a duration of 24 hours or longer, facilitating the detection of occult AF

- **Types of Outpatient cardiac monitors:**

- Loop recorder
- Holter monitor



D. SERUM CHEMISTRY AND TFT'S

- **Primary Purpose:**

- Identify reversible and easily treatable causes of AF such as hypokalemia, hypomagnesemia, and hyperthyroidism

1. Reversible Causes of Atrial Fibrillation

- **Hypokalemia**

- Administer IV K

- **Hypomagnesemia**

- Administer IV magnesium

- **Thyrotoxicosis**

- Administer Propranolol and PTU



IV. TREATMENT

Treatment Goals:

- Rate control
- Rhythm control
- Anticoagulation

A. RATE CONTROL

Primary Purpose of Rate Control:

- Aims to manage the ventricular response, preventing complications like dilated cardiomyopathy, acute heart failure, and further electrical remodeling, often making it the preferred initial treatment approach
- Goal of rate control: **HR < 110 bpm**

Rate Control Group	Drugs	Additional Comments
Beta Blockers	Metoprolol	Ø in Acute Heart Failure Ø in Bradycardia and COPD
Calcium Channel Blockers	Diltiazem Verapamil	Ø in Acute Heart Failure Ø in Bradycardia
Cardiac Glycoside	Digoxin	✓ in Heart Failure (↓EF)

1. Beta Blockers

Agents: Metoprolol

- **MOA:** Inhibits atrioventricular (AV) nodal conduction by antagonizing the β -1 adrenergic receptors
- **Avoided in:**
 - Acute Heart Failure
 - Bradycardia
 - COPD

2. Calcium-Channel Blockers

Agents: Diltiazem, Verapamil

- **MOA:** Inhibits atrioventricular (AV) nodal conduction by blocking calcium influx and thus depolarization
- **Avoided in:**
 - Acute heart failure
 - Bradycardia

3. Cardiac Glycoside

Agents: Digoxin

- **MOA:** Inhibits atrioventricular (AV) nodal conduction via vagal effect on AV node
- **Indications:**
 - Heart Failure (↓EF) → Inotropic agent

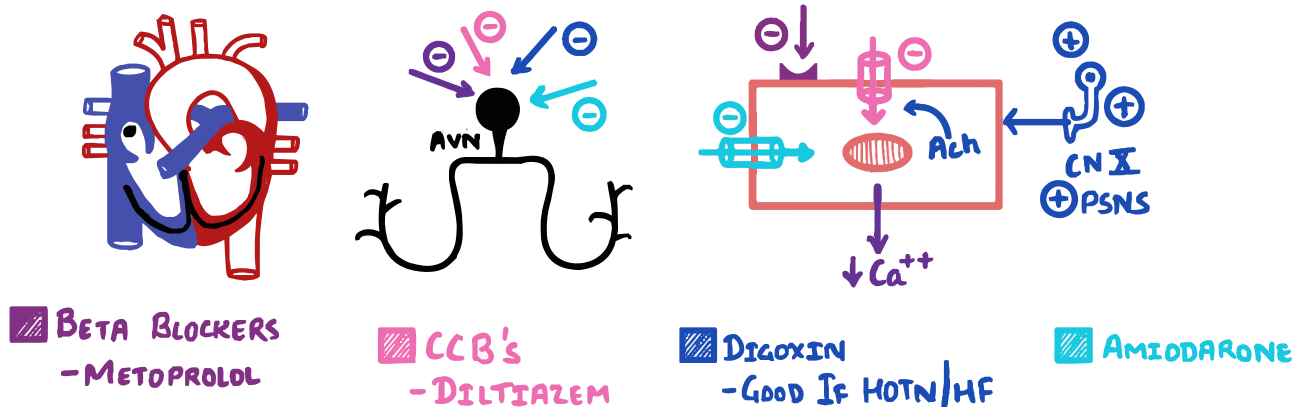


FIGURE 3. DRUGS USED AS RATE CONTROL IN ATRIAL FIBRILLATION.



Primary Purpose:

- Rhythm control strategies, which focus on restoring and maintaining sinus rhythm, may be considered when rate control is insufficient, symptomatic improvement is inadequate, or the patient is hemodynamically unstable

Type of Therapy	Indications	Additional Comments
Direct Current Cardioversion (Old sparky) ⚡	1. HD instability 2. AF < 48 hrs 3. Anticoagulated x 3-4 weeks	DCC > Pharmacological
Pharmacological Cardioversion (Amiodarone, Flecainide)	+ TEE shows no LA thrombus	↑ of Torsades de Pointes (Via ↑ QT-interval)
Surgical Intervention (RFA, Maze procedure)	Refractory Atrial Fibrillation	

1. Direct Current Cardioversion

Cardioversion Indications

- Hemodynamical Instability**
 - ↓ BP, angina, pulmonary edema, acute HF, altered mental status
- AF < 48 hrs**
 - Less likely to form emboli due to short time
- Anticoagulant x 3-4 weeks + TEE shows no LA thrombus**
- Main advantage:**
 - No risk of Torsade's de Pointes (TdP)
- Main disadvantage:**
 - Painful and requires analgesia

2. Pharmacological Cardioversion

Agents: Amiodarone, Flecainide, Lidocaine

- Same indications as Direct Current Cardioversion with different MOAs
- Main Disadvantage:
 - ↑ Risk of TdP from prolongation of the QT interval
- Drug of choice most often: **Amiodarone**

3. Surgical Intervention

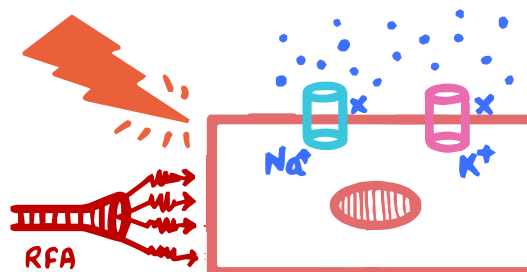
RFA, Maze Procedure

- Indicated for **refractory AF**
- Maze Procedure:** Best performed when undergoing cardiac surgery

Direct Current > Pharmacological Cardioversion

ELECTRICAL CARIOVERSION

- 1) RFA - PULMONARY VEIN
- 2) "MAZE" PROCEDURE (Sx)



CHEMICAL

- TYPE Ia
- TYPE Ic
- TYPE III

↑QT-I → ↑RISK OF TORSADES DE POINTES

FIGURE 4 RHYTHM CONTROL FOR ATRIAL FIBRILLATION



- Primary Purpose → ↓ Risk of thromboembolic events such as:
 - Stroke, AMI, ALI

1. CHA₂DS₂-VASc Score

Understand the criteria:

Criteria	Score
CHF (1pt)	≥ 2: Anticoagulate
HTN (1pt)	1: Clinical judgement
Age ≥ 75 (2 pts)	0: No anticoagulation, start Aspirin
DM (1pt)	
Stroke/TIA (2pts)	
Vascular Dx (1pt)	
Age 64-75 (1pt)	
Sex, female (1pt)	
	Anticoagulant choice
	DOAC → nonvalvular AF (Rivaroxaban, Apixaban)
	Warfarin → Valvular or Nonvalvular AF (monitor INR)
	Heparin → Bridge to DOAC or Warfarin (good for hospitalized patients)

- Score ≥ 2:** Requires anticoagulation due to ↑ risk of stroke
 - Also, weigh the risk of bleeding via HAS-BLED score
- Score = 1:** Use clinical judgment
 - Do they have a GI bleed?
 - Advanced age?
 - High risk of bleeding?
 - Do they have a recent history of stroke?
- Score = 0:** No anticoagulation → Administer Aspirin

2. Anticoagulant Choice

a) DOAC

Rivaroxaban, Apixaban, Dabigatran

- Indicated For patients with **Nonvalvular AF**
- No monitoring necessary**

b) Warfarin

- Indicated:** Patients with **Valvular AF/Nonvalvular AF with CKD**
- Monitor INR:**
 - INR 2-3 for those WITH prosthetic valve
 - INR 2.5-3.5 for those WITHOUT prosthetic valve

c) Heparin

- Indicated in the inpatient setting due to its immediate anticoagulation effect
 - Used as a bridge to DOAC or warfarin in the outpatient setting
- Monitor PTT:**
 - Range depends on the institution and underlying anticoagulation goals

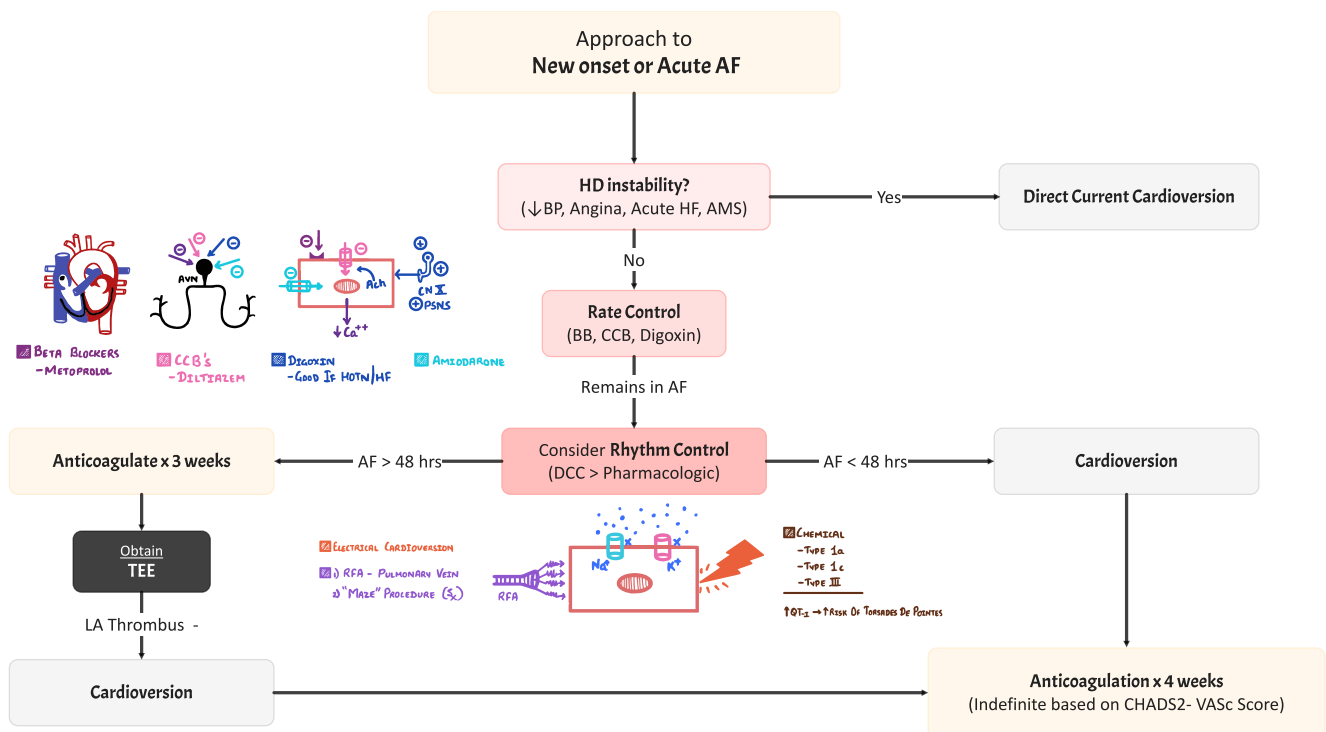


FIGURE 5. APPROACH TO TREATMENT AND DIAGNOSIS OF NEW-ONSET ATRIAL FIBRILLATION.

