

# CARDIOVASCULAR PATHOLOGY

## ATRIAL FIBRILLATION

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Atrial Fibrillation

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### OUTLINE

- I) ATRIAL FIBRILLATION
- II) RISK FACTORS FOR ATRIAL FIBRILLATION
- III) PATHOPHYSIOLOGY
- IV) CLINICAL MANIFESTATIONS
- V) DIAGNOSIS
- VI) TREATMENT
- VII) REVIEW QUESTIONS
- VIII) REFERENCES

### I) ATRIAL FIBRILLATION

- Atrial Fibrillation (A-fib) is a supraventricular arrhythmia that originates above the ventricles
- Causes irregularly irregular rhythm and many other complications
- There is **no absolute identifiable cause** for A-fib, but there are risk factors which will be referred to as "cardiac causes" and "non-cardiac causes" in this lecture

#### (A) MECHANISMS

##### (1) Stretching of the myocardium

- Causes myocardium to dilate and hypertrophy

##### (2) Ischemia: Decreased oxygen

- To myocardium of atria

##### (3) Inflammation

- Of the atria itself or anywhere near the atria

##### (4) Increase in sympathetic nervous system (SNS) stimulation on the atria

- Causes a lot of calcium loading and agitation to the atria

##### (5) Electrolyte Imbalance

- Particularly  $\text{Ca}^{2+}$  and  $\text{K}^+$

### II) RISK FACTORS FOR ATRIAL FIBRILLATION

#### (A) CARDIAC CAUSES

##### (1) Congestive Heart Failure (CHF)

- Mechanism: **Increased Stretch**
- Condition wherein the heart's muscles are floppy and flabby causing the stretching and distention of the myocardium of both the ventricles and the atria
- ↑stretch of myocardium → atrial dilation → atrial hypertrophy
- Changes the morphology of the atria

### (2) Dilated cardiomyopathy

- Mechanism: **Increased Stretch**
- The ventricles get thin, floppy, and ballooned, affecting the atria

### (3) Valvular Diseases

- Mechanism: **Increased Stretch**
- Conditions wherein the mitral valve isn't working very well
- With a normal mitral valve, the atria contracts to push blood into the ventricles

#### (i) Mitral Stenosis

- Stenotic mitral valve (valvular narrowing) allows less blood to flow from the atria into the ventricles  
→ some volume is retained in the atria → atria will balloon up to accommodate the extra volume

#### (ii) Mitral Regurgitation

- Can have the same effect as mitral stenosis, but is less common

### (4) Coronary Artery Disease or Myocardial Infarction

- Mechanism: **Ischemia** (decreased  $\text{O}_2$ )
  - Ischemia can trigger atria to develop ectopic foci and reentry circuits nearby, causing an atrial arrhythmia.
- Severe coronary artery disease or history of myocardial infarction
- If there's an ischemic or infarcted area, tissue in the atria near the area must undergo remodeling

### (5) Rheumatic Heart Disease

- Mechanism: **Inflammation**
  - The valves, atria, endocardium, or myocardium may be inflamed
- May be due to Rheumatic fever
- May lead to Mitral Stenosis, ultimately leading to A-fib
- May also generate enough inflammation and atrial agitation to directly cause the A-fib

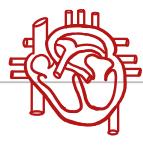
**Note:** There is a separate lecture on Rheumatic Fever: Etiology, Pathophysiology, & Diagnosis.

### (6) Hypertension

- Mechanism: **Increased Stretch**
- Due to high blood pressure, left ventricle contracts more  
→ left ventricular hypertrophy
- Overtime, there is enough hypertrophy to cause atrial hypertrophy or atrial remodeling
- Cause the atria to generate abnormal electrical activities



CHF  
- STRETCH



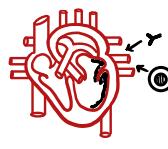
DILATED CMP  
- STRETCH



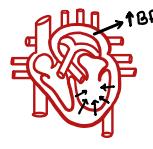
VALVULAR D/o's  
- MITRAL STENOSIS  
↑ DILATION  
- MITRAL REGURGITATION  
(L.R.)



C.A.D / M.I  
- ISCHEMIA ( $\downarrow \text{O}_2$ )



RHEUMATIC HEART Ds.  
- INFLAMMATION



HYPERTENSION  
- L.V.H.  
- ATRIAL HYPERTROPHY

Figure 1 Cardiac causes of Atrial fibrillation



## (B) NON-CARDIAC CAUSES

### (1) Lung Pathologies

- Mechanism: Ischemia**

- $\downarrow O_2 \rightarrow$  Hypoxemia  $\rightarrow$  May trigger atria to be irritable  
→ abnormal foci or reentrance circuits  $\rightarrow$  Ischemia
- In a normal lung: takes in  $O_2$ , bring it into the alveoli, push it into the blood, and get rid of  $CO_2$
- In a pathological process of the lung parenchyma,
  - COPD and pneumonia: Ventilation Problem
    - Decreased ability to get  $O_2$  to the blood
  - Acute onset pulmonary embolism: Perfusion Problem
    - A clot in the vessel may prevent  $O_2$  from getting pass the occlusion
  - Either mechanisms will lead to low  $O_2$

### (2) Thyrotoxicosis or Hyperthyroidism

- Mechanism: Increased SNS activity**

- Releases plenty of FT3 and FT4
  - T3 and T4 increases the  $\beta$ -adrenergic stimulation
  - Increased sensitivity of the  $\beta_1$ -adrenergic receptors to epinephrine and norepinephrine

### (3) Holiday Heart Syndrome

- Mechanisms: Inflammation, Electrolyte Imbalance, and Increased SNS activity**

- In **excessive alcohol intake** (e.g., *acute alcohol binge-drinking*), ethanol causes a cytotoxic effect on the body
  - $\uparrow$  ROS (reactive oxygen species) formation  $\rightarrow$  inflammation
  - $\uparrow$  SNS effects on the heart
  - Can alter the electrolytes  $\rightarrow$  electrolyte abnormalities
    - e.g.,  $K^+$  and  $Mg^{2+}$ : important cations in the electrical activities of the heart
    - May be due to alcohol-induced vomiting or the alcohol content itself not having enough micronutrients we need

### (4) Pheochromocytoma

- Mechanism: Increased SNS activity**

- Tumor in adrenal medulla
- Releases catecholamines (epinephrine & norepinephrine) that bind to  $\beta_1$ -adrenergic receptors  $\rightarrow$   $\uparrow Ca^{2+}$  influx and cation loading  $\rightarrow$  1SNS activity  $\rightarrow$  agitates atria  $\rightarrow$  A-fib

### (5) Drugs (Cocaine and Methamphetamines)

- Mechanism: Increased SNS activity**

- Act as sympathomimetics
- Bind to the same receptors that epinephrine and norepinephrine bind to  $\rightarrow$  1SNS activity  $\rightarrow$  increased cation loading  $\rightarrow$  irritation of the atria  $\rightarrow$  atrial fibrillation
- Can be an acute onset

### (6) Sepsis

- Mechanism: Increased SNS activity**

- Can also be an acute onset
- When in sepsis, the blood pressure drops (may lead to a hypotensive state), patient becomes febrile, and has other body complications
- In a septic response, there's vasodilation, capillary permeability, fever
- Since sepsis is a stressful event, the body's protective response is to go into a **"Fight or Flight" mode by hyperactivating the SNS**.
  - This increases the heart rate and blood pressure, counteracting the negative effects of sepsis

### (7) Surgical Procedures

- An invasive surgical procedure can cause **significant post op stress** on the body  $\rightarrow$  trigger a catecholamine response  $\rightarrow$  hyperactive SNS  $\rightarrow$  atrial fibrillation

### (8) Electrolyte Abnormalities

- Mechanism: Electrolyte Imbalance**

- Hypokalemia ( $\downarrow [K^+]$ ) and hypomagnesemia ( $\downarrow [Mg^{2+}]$ ) in the body can alter the heart's electrical activity, causing the body to undergo an atrial fibrillation or supraventricular tachyarrhythmia.

### NON-CARDIAC CAUSES

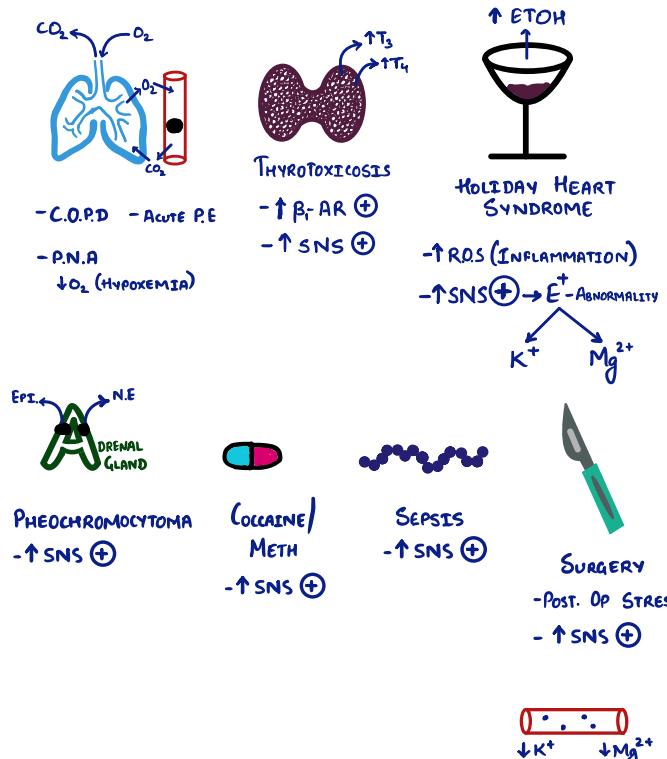


Figure 2 Non-Cardiac Causes of Atrial Fibrillation



### III) PATHOPHYSIOLOGY

- How do these risk factors lead to the pathophysiological and clinical features & complications associated with atrial fibrillation?

#### (A) RECALL: WHAT ARE THE FIVE MECHANISMS BY WHICH ATRIAL FIBRILLATION OCCURS?

##### (1) Increased stretch on the heart

- CHF,
- dilated cardiomyopathy,
- mitral stenosis,

##### (2) Inflammation of the myocardium

- Rheumatic heart disease,
- Holiday heart syndrome,
- increased ROS production

##### (3) Low Oxygen Delivery to the Myocardium

- Coronary artery disease,
- myocardial infarction,
- COPD,
- pneumonia,
- acute pulmonary embolism,

##### (4) Increased sympathetic activity

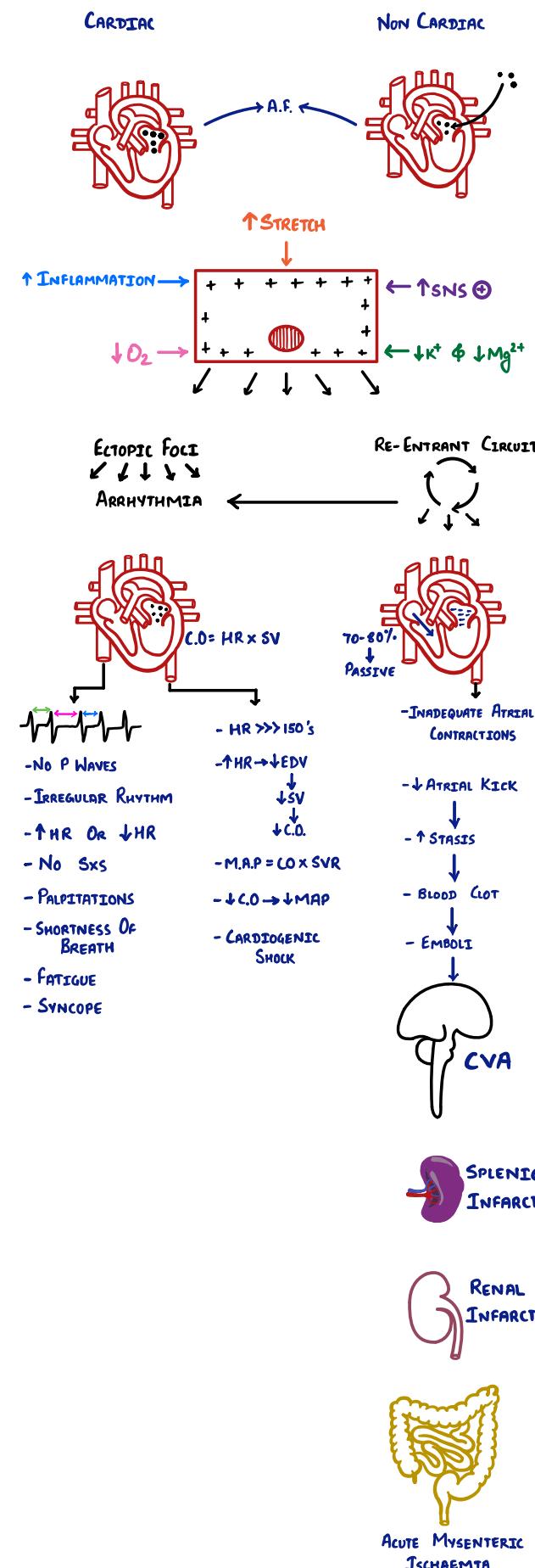
- Thyrotoxicosis,
- Pheochromocytoma,
- excessive alcohol intake,
- cocaine & methamphetamine consumption,
- septic reaction,
- post-op

##### (5) Electrolyte imbalance,

- particularly hypokalemia and hypomagnesemia

- If there's significant agitation to the myocardium to the point that it needs to undergo remodeling, it can start generating abnormal electrical activity via 2 ways
  - Formation of ectopic foci** in multiple areas in myocardium of the atria
  - Development of re-entrant circuits**  
Alpha and beta pathways (check out overview of arrhythmia lecture)

### PATHOPHYSIOLOGY



#### IV) CLINICAL MANIFESTATIONS

##### ● SA Node

- Pacemaker of the heart
- Generates sinus rhythm
- Send electrical activities to the AV node to be sent to the ventricles via the bundles of His, bundle branches, and Purkinje fibers

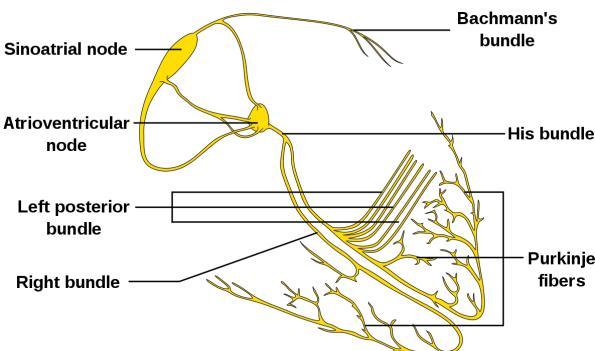


Figure 3. Bundles of His

##### (1) Heart Rhythm

- If there are areas other than the SA node that are actively firing due to the previously mentioned triggers, they're going to interfere with the normal sinus pathway
- For example, if there are different ectopic foci firing and they get to the AV node before the SA node, they will trigger QRS complexes with noted rhythm variations.

##### (i) Observable Changes from normal rhythm

- No visible p waves
- Irregular rhythm
  - Variations in R-R intervals due to different ectopic foci sending signals to the AV node → ventricles
- Variations in HR
  - Can be fast ( $\uparrow$ HR) or slow ( $\downarrow$ HR)
  - It's also possible to be asymptomatic for atrial fibrillation

##### (ii) Common Symptoms

- Palpitations
- Shortness of breath
- Fatigue
- Episodes of syncope

##### (iii) Asymptomatic for atrial fibrillation

- It's also possible for a patient to be completely asymptomatic for atrial fibrillation.

##### (2) Cardiogenic Shock

- Summary of Mechanism:  $\uparrow\uparrow$ HR  $\rightarrow$   $\downarrow$ ventricular filling time  $\rightarrow$   $\downarrow$ end-diastolic volume  $\rightarrow$   $\downarrow$ SV  $\rightarrow$   $\downarrow$ CO  $\rightarrow$   $\downarrow$ MAP  $\rightarrow$  hypotension  $\rightarrow$  cardiogenic shock
- If there is tachycardia: HR  $>>>$  150
  - Recall: CO = HR X SV
    - Technically, increase in HR should increase CO
  - But in this case, the HR is so fast  $\rightarrow$  there's not enough time for the ventricles to fill  $\rightarrow$  lowers end-diastolic volume  $\rightarrow$  lowers SV  $\rightarrow$  lowers CO
  - MAP = CO x SVR
    - MAP: Mean Arterial Pressure
    - SVR: Systemic Vascular Resistance
    - $\downarrow$ CO  $\rightarrow$   $\downarrow$ MAP  $\rightarrow$  hypotension
    - Since it's coming from the heart, it can lead to cardiogenic shock

##### (3) Emboli

- Sometimes, patients may not have any symptoms
- If there's an ectopic foci or re-entrant circuits causing depolarization and contractions, the abnormal electrical activity produces inadequate atrial contractions

##### ● Recall: Cardiac Cycle

- 70-80% of the blood from the atria passively flows down to the ventricles
- 20-30% blood volume comes from the **atrial kick**
  - Phenomenon where the atria contracts end-diastole to squeeze the remaining volume out

##### (i) Summary of Mechanism:

- Atrial fibrillation  $\rightarrow$  atrial quivering  $\rightarrow$  inadequate electrical activity  $\rightarrow$  inadequate atrial contraction  $\rightarrow$   $\downarrow$ atrial kick  $\rightarrow$  1blood volume stays in the atria and doesn't flow to the ventricle  $\rightarrow$   $\uparrow$ Stasis  $\rightarrow$  clot
- **Stasis:** blood stays in an area longer than it should
- **Virchow's Triad:** Stasis, endothelial injury, and hypercoagulability increases the risk of a clot

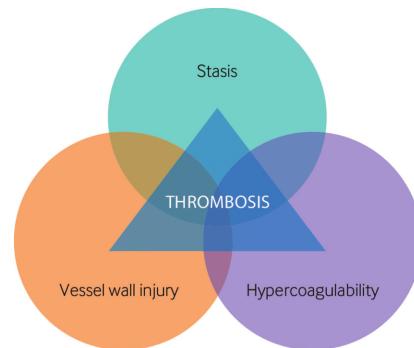


Figure 4. Virchow's Triad [Khan, et al.]

##### (ii) What happens to the clot?

- When a clot forms on the atria or the valve
  - A patient may undergo cardioversion or the patient's heart may naturally convert
    - **Cardioversion:** medical procedure used to return an abnormal heartbeat to normal.
- **Clot is removed from the atria or valve due to:**
  - The atria contracting strong enough
  - An increased stressful event causes the heart to contract more
- **The removed clot may flow through the systemic circulation** up to the brain, down to the descending aorta, and **embolize in multiple different organs**

##### (iii) Where can the emboli go?

###### (a) Brain

- One of the first signs is a cerebrovascular accident (CVA)
- Develops an ischemic stroke
- May present as neurologic deficits

###### (b) Spleen

- May develop a splenic infarct
- May present as pain in LUQ and show issues or abnormalities in the CBC

###### (c) Kidney

- May develop a renal infarct
- May cause increase in BUN and creatinine

###### (d) Gut

- May develop an acute mesenteric ischemia
- May present as abdominal pain



## V) DIAGNOSIS

### (A) CLASSIFICATION OF ATRIAL FIBRILLATION

#### (1) Hemodynamics

##### (i) Hemodynamically unstable Atrial Fibrillation

- Patient presents with any of the following:
  - ↓BP (hypotensive)
  - Pulmonary edema
    - Especially in a state of cardiogenic shock, wherein the Ejection Volume Fraction is not adequate enough → blood backs up to the pulmonary circulation
  - Altered Mental Status
  - Severe Chest Pain

##### (ii) Hemodynamically Stable Atrial Fibrillation

- Simply put, patient does not present with any of the symptoms listed above.
- Patient has:
  - Normal BP
  - No pulmonary edema
  - Normal mentation
  - No obvious significant refractory chest pain

#### (2) Ventricular Rate

##### (i) Atrial Fibrillation with Rapid Ventricular Rate

- HR > 100 bpm
- HR, particularly the ventricular rate, is really fast
- Ectopic foci are firing really fast; hitting the AV nodes, and going down to the ventricles

##### (ii) Atrial Fibrillation with Slow Ventricular Rate

- HR < 60 bpm
- Re-entrant circuits or ectopic foci are not firing as fast or as frequent, causing the AV nodes to send conduction down to the ventricles
  - This slows down the ventricular rate

#### (3) Onset and Duration

##### (i) New Onset Atrial Fibrillation

- A-fib developed less than 48-72 hours ago.

##### (ii) Paroxysmal Atrial Fibrillation

- A-fib developed less than 7 days ago

##### (iii) Persistent Atrial Fibrillation

- A-fib has been present for more than 7 days

##### (iv) Long-Standing Atrial Fibrillation

- A-fib has been present for more than a year

##### (v) Permanent Atrial Fibrillation

- Persistent A-fib without any attempt to cardiovert the patient to normal sinus rhythm
- A-fib > 7 days without cardioversion attempt

#### (4) Mitral Valve Involvement

##### (i) Valvular Atrial Fibrillation

- Presence of mitral stenosis or a mechanical valve
  - This can determine what type of anti-coagulant the patient needs

##### (ii) Nonvalvular Atrial Fibrillation

- No involvement of mitral valve or mechanical valve
  - This can determine what type of anti-coagulant the patient needs

## DIAGNOSIS

### CLASSIFICATION

#### HEMODYNAMICS

##### HD UNSTABLE -

■ ↓BP ■ PULMONARY EDEMA ■ AMS ■ CHEST PAIN

##### HD STABLE -

NONE OF THE ABOVE ISSUES

#### VENTRICULAR RATE

RAPID VENTRICULAR RATE: AF & RVR  
HR >> 100 BPM

SLOW VENTRICULAR RATE: AF & SVR  
HR < 60 BPM

#### ONSET AND DURATION

NEW: <48-72 HRS LONG STANDING: >1YR

PAROXYSMAL: <7 DAYS PERSISTANT: >7 DAYS

PERMANENT: >7 DAYS WITHOUT ATTEMPTS TO  
CARDIOVERT → NORMAL SINUS RHYTHM

#### MITRAL VALUE INVOLVEMENT

VALVULAR A.F.: MITRAL STENOSIS OR  
MECHANICAL VALVE

NONVALVULAR A.F.: NO MITRAL STENOSIS OR  
MECHANICAL VALVE



## (B) DIAGNOSTIC TESTS

### (1) EKG

- The best test that can be used: quick, cheapest, and the most non-invasive procedure
- **Focus on Lead V<sub>1</sub>**
  - Shows atrial activity
  - If there's no evidence on V<sub>1</sub>, look at V<sub>2</sub>, V<sub>3</sub>, and aVF

#### (i) What can be seen on ECG?

- (a) Presence or absence of A-Fib
- (b) Wolff-Parkinson-White (WPW) syndrome
  - Tachycardia (close to 200 bpm)
  - Irregular rhythm
- (c) Potential causes of A-fib
  - E.g., recent MI, coronary artery disease, hypertension
- (d) Rule out ST segment elevation/depression
- (e) Left Ventricular Hypertrophy

**Note:** A patient may not be in A-fib at the exact moment the ECG is done. A normal ECG does not automatically mean that the patient is fine.

#### (ii) What can be done other than the ECG?

##### (a) Holter Monitoring

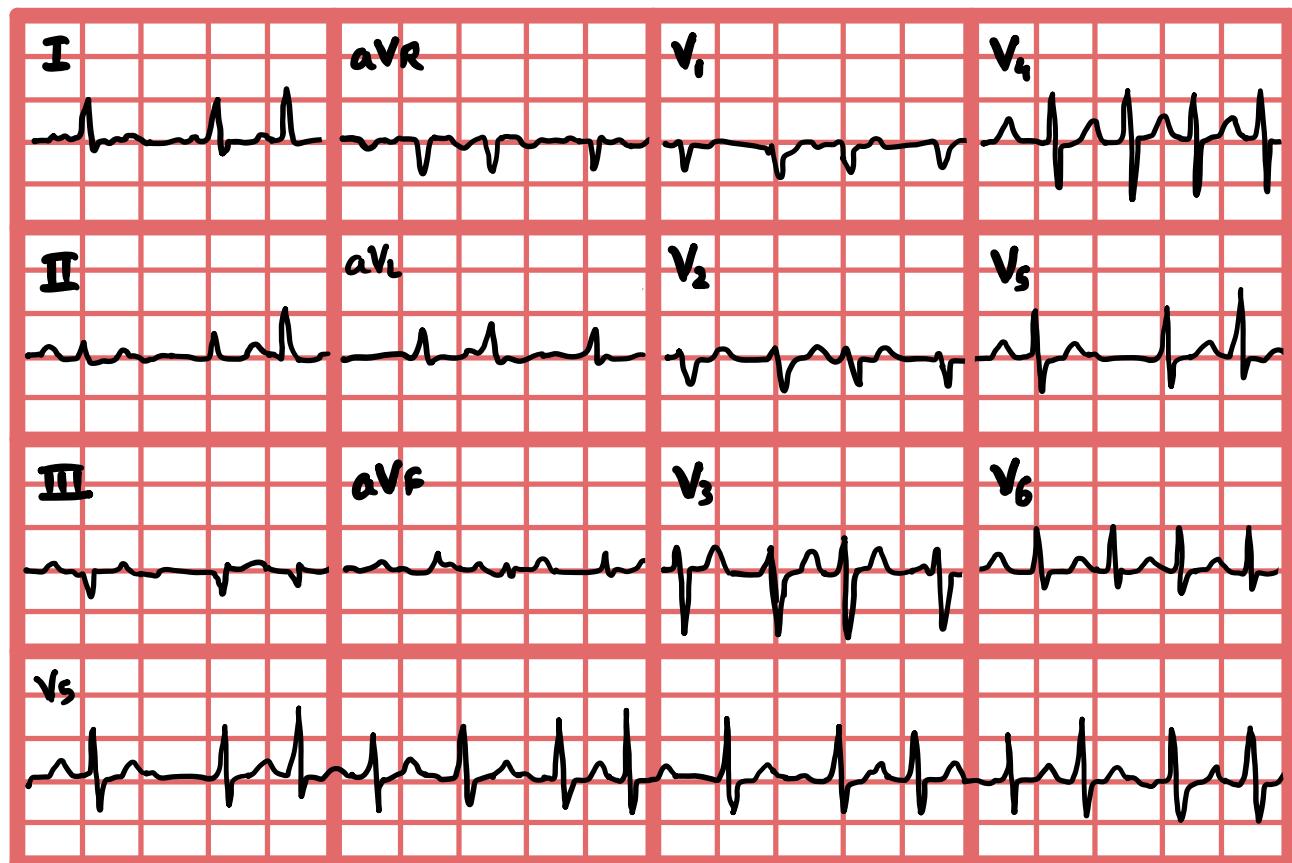
- Gold standard
- Addresses problem with standard ECG wherein electrical activity is only assessed at a particular moment in time
- Can track a patient's electrical activity over a certain amount of time
- It's essentially ECG for outpatient monitoring

##### (b) Loop Recorder

- An implantable heart recording device may also be used to monitor outpatient's electrical activity
- More invasive than Holter monitoring

##### (c) Telemetry

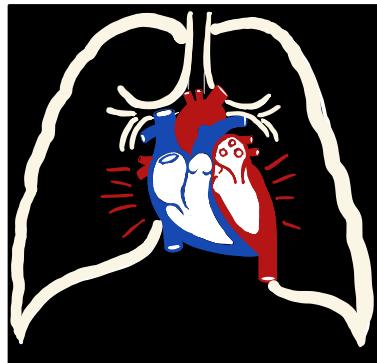
- In-patient continuous monitoring of heart's electrical activity



### (2) Chest X-Ray (CXR)

- A key test in determining the trigger/s for A-fib
  - **Pneumonia**
  - **COPD**
    - You may see hyperinflated lungs, hyperlucid lungs, and flattened diaphragm
    - These aren't diagnostic of COPD, but if patient also has a history of tobacco abuse, then COPD is a possible rule in.
  - **Cardiomegaly**
    - May indicate CHF, dilated cardiomyopathy, and other cardiac conditions
- Can also be used to **rule out other conditions** to make sure that the diagnosis is really A-fib

CXR



### (3) Ultrasound: Echocardiogram

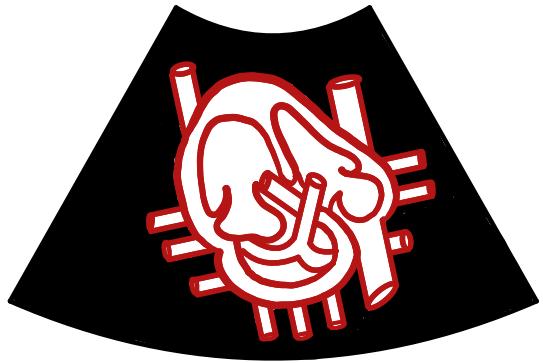
#### (i) Types of Echocardiograms

- (a) Trans-Esophageal Echocardiogram (TEE)
  - Gold standard
  - May be a little bit better than the TTE at picking up something in the atria
  - Invasive type of procedure
- (b) Trans-Thoracic Echocardiogram (TTE)
  - Non-invasive procedure
  - Most used echocardiogram

#### (ii) What to look for in an echocardiogram

- (a) Atrial thrombus
  - Indicates presence of a clot
    - Needs urgent treatment (anti-coagulant)
  - Recall: If patient goes back into normal sinus rhythm, clot may embolize into other organs
- (b) Mitral Valve Issues
  - Mitral valve stenosis, mitral regurgitation, or mechanical valve that may not have been indicated on patient's history (*i.e., patient doesn't know*)
- (c) Left Ventricular Ejection Fraction (LVEF)
  - Useful in determining if patient has heart failure with reduced ejection fraction
- (d) Left Ventricular Hypertrophy
  - Associated with hypertension
- (e) Atrial Dilation
  - An enlarged atrial size may be diagnostic for atrial fibrillation, even if it doesn't show on their ECG, Holter monitor, or in-patient Telemetry.

T.E.E Or T.T.E



- ATRIAL THROMBUS +
- MITRAL VALVE D/o +
- LVEF
- LVH
- ATRIAL DILATION +

### (4) Laboratory Tests

- Not necessarily enough to diagnose A-fib, but can help determine what is triggering or causing the A-fib.

#### (i) Brain natriuretic peptide (BNP)

- Elevated BNP may point to CHF
- Result can be correlated with ECG reading
  - Check for signs of LVEF abnormalities and hypertrophic ventricles

#### (ii) Troponin Levels

- If elevated, may point to myocardial ischemia
- Correlate with the ST segment on ECG

#### (iii) Complete Blood Count (CBC)

- Elevated WBC count may indicate pneumonia
- Pro-calcitonin levels may also indicate an infection
- Results can be correlated with CXR

#### (iv) TSH with Reflex to FT<sub>4</sub>

- If TSH is low, thyroid hormone may be high
- Useful in screening for hyperthyroidism
- May also be used for the differential diagnosis of primary, secondary, and tertiary hypothyroidism

#### (v) Blood alcohol level (etOH)

- Recall: Holiday Heart Syndrome

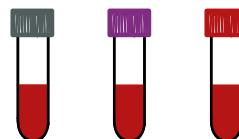
#### (vi) Urine Metanephrine Test

- To detect or rule out pheochromocytoma
  - Additionally, an Adrenal CT can be done to check for a mass within the adrenal gland

#### (vii) Urine Toxicology Test ("Tox Screen")

To detect the presence of metabolites of cocaine, methamphetamine, or urinary alcohol

LABS



- BNP ↑↑↑ → CHF

- TROPONINS → M.I.

- CBC → ↑WBC

- TSH & REFLEX → ↑T<sub>3</sub> + T<sub>4</sub>

- BLOOD ETOH +

- URINE METANEPHRINES +

- URINE TOX +



## VI) TREATMENT

### (A) TREATMENT ALGORITHM

#### **(1) Determine Patient's Hemodynamic Status**

##### **(i) Perform Direct Current Cardioversion**

- If patient is **hemodynamically unstable**

##### **(ii) Perform Rate Control**

- If patient is **hemodynamically stable**
- Rate control primarily regulates the rapid A-fib.
- The two preferred rate control agents are beta blockers and calcium channel blockers

#### **(2) Determine patient's need for anti-coagulation**

- This is **done after performing rate control** and ideally reaching a target HR of less than 100-110 bpm
- Goal: **to reduce the risk of embolization**
  - Patients getting admitted are usually started on unfractionated or low molecular weight heparin
  - Oral anti-coagulants can also be used
- 50-70% of the time, patients will undergo spontaneous cardioversion within 24 hours of onset
  - A-fib (especially acute onset A-fib) will convert into normal sinus rhythm without intervention with electricity or chemical induction process

#### **(3) Step 3: Consider Cardioversion**

- For patients who did not undergo spontaneous cardioversion, determine if it's necessary to cardiovert the patient back to normal sinus rhythm.

##### **(i) Perform Cardioversion**

- For patients who are on A-fib for less than 48 hours and have a low risk of ischemic stroke
  - Determine risk via the **CHA<sub>2</sub>DS<sub>2</sub>-VASc system**
- Can be done electrically or chemically
- Based on their CHADS-VASc score and other associated risk factors, decide if patient needs to be put on oral anticoagulants for four weeks after cardioversion.

##### **(ii) Perform TEE and Give Oral Anti-Coagulants**

- For patients who have been on A-fib for more than 48 hours or for an unknown period, and have an unknown period or for more than 48 hours

###### **(a) Option 1**

- **Perform TEE** to check if they have a clot within their atria at that moment
  - (+) Left Atrial Thrombus
    - Oral anti-coagulation for 3 weeks
    - Recheck for clot via TEE
  - (-) Atrial Thrombus
- **Perform cardioversion**
- **Give oral anticoagulants for 4 weeks**
  - It's also possible for these patients to be on long-term anti-coagulants due to their increased risk

###### **(b) Option 2**

- Give **oral anticoagulant** for 3 weeks
- **Perform cardioversion**
- Give **oral anticoagulant** for 4 weeks or more

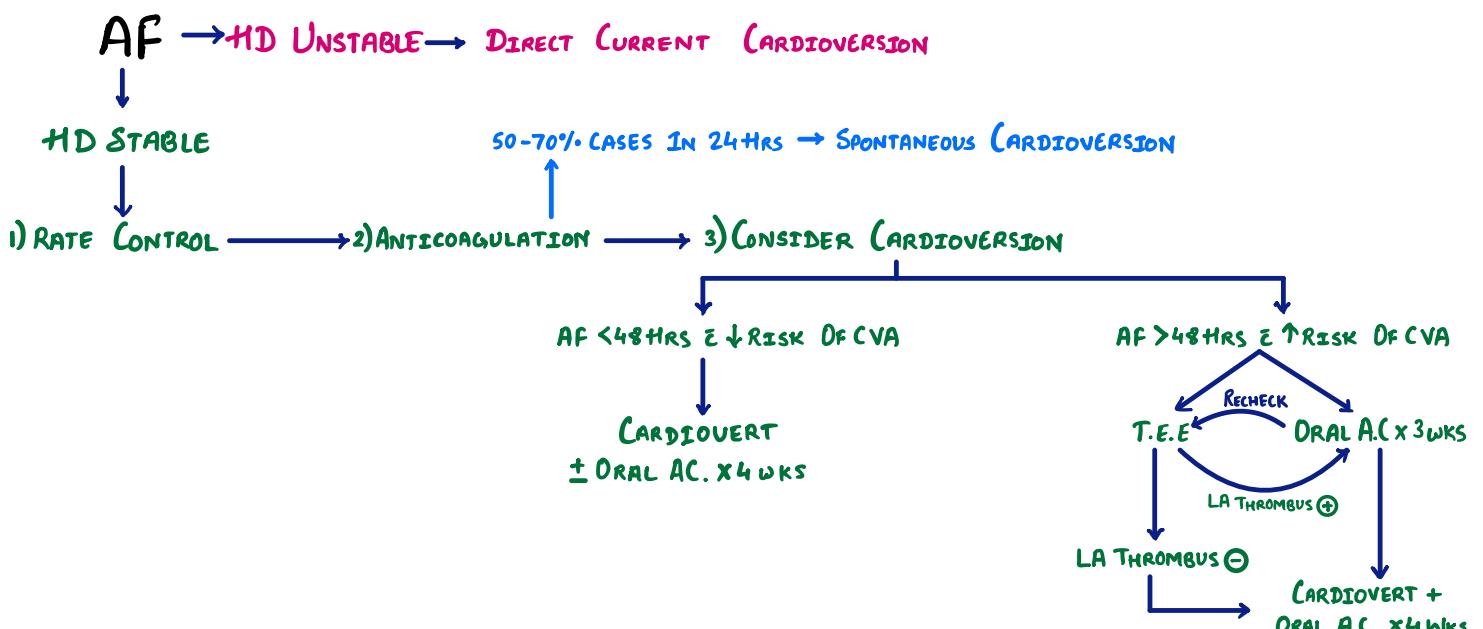


Figure 5 Overview of Atrial Fibrillation Treatment Algorithm



## (B) TREATMENT MECHANISMS

### (1) Rate Control

- Conduction system
- AV Node

#### (i) Rate control agents

- **block the electrical activity** coming from the ectopic foci and re-entrant circuits from moving through the AV node and down into the Bundles of His
- Sifts through the excess electrical activity and only allows for certain things to go down to the ventricles
- Slows down the ventricular rate

##### (a) Beta blockers

- Metoprolol: commonly utilized drug
- Contraindication: reactive airway disease, underlying COPD
  - $\beta$ -2 receptor in the lungs may exacerbate their condition

##### (b) Calcium Channel Blockers

- Drugs that block calcium channels in AV cells
  - block calcium entry → slows down electrical activity being transmitted through AV node
- Contraindication: patients with decompensated heart failure
- Drugs: Diltiazem (most common), Verapamil

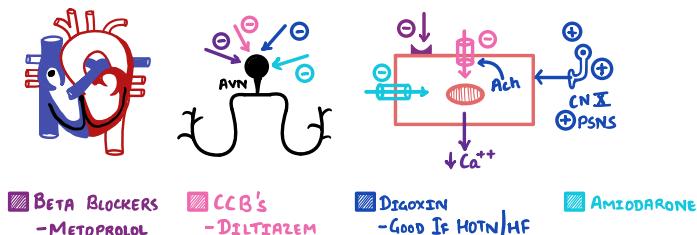
##### (c) Digoxin

- Takes a bit longer for its effects to kick in
- Drug good for hypotension or heart failure
- Targets sodium-potassium-ATPase
  - Alters calcium-sodium exchangers
  - Effect: increases contractility
- AV Node: Stimulates Vagus Nerve (CN X) → stimulates parasympathetic nervous system
  - increases acetylcholine release → more  $K^+$  leave the cell → slows down HR

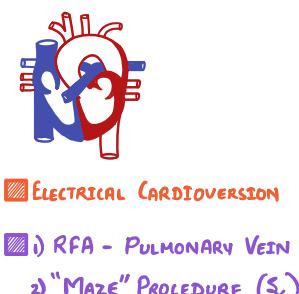
##### (d) Amiodarone

- Third-line agent
- Has effects on both rate and rhythm control
  - Blocks entry of sodium and calcium, and has a little bit of beta blocker effect

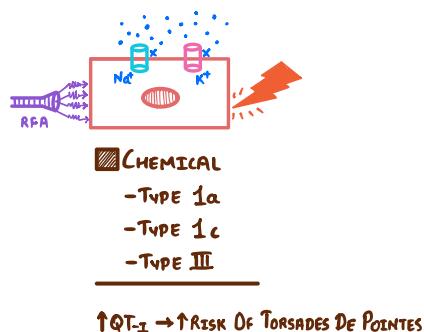
#### RATE CONTROL:



#### RHYTHM CONTROL:



#### DRUGS



### (2) Three Ways to Control Rhythm

#### (i) Chemical Cardioversion: Utilize drugs

##### (a) Examples of drugs:

- Type 1a (e.g., procainamide)
- Type 1c (e.g., Flecainide, Propafenone)
- Type III (e.g., Ibutilide, Amiodarone)

**Note:** There will be a separate lecture on the different classes of antiarrhythmic drugs.

##### (b) Mechanism:

- Alters activity of different type of channels (e.g.,  $Na^+$  channels,  $K^+$  channels) → regulates electrical activity within the myocardial cells of the atria → regulates rhythm

##### (c) Contraindication:

- May potentially affect QT interval → increases risk for Torsades de pointes (TdP)

#### (ii) Electrical Cardioversion

- Shock the heart to reset all the electrical activity of the myocardium so that it can get back into a normal sinus rhythm

#### (iii) Radiofrequency Ablation (RFA)

- Utilization of radiofrequency waves to burn and destroy ectopic areas
  - They are usually near the entrances of the pulmonary veins and atria

#### (iv) Maze procedure

- Surgical procedure
- RFA would still be the first line or option before the maze procedure



### (3) Anticoagulation

- Patients with A-fib are at high risk for forming clots and embolizing them to different areas in the body

#### (i) CHA<sub>2</sub>DS<sub>2</sub>-VASc Score

- Way to risk stratify and determine patient's need for anticoagulation
- Compared with patient's **HAS-BLED score**  
→ Determines the risk of bleeding

Criteria	Poss. Point
<b>C</b> ongestive heart failure Signs/symptoms of heart failure confirmed with objective evidence of cardiac dysfunction	+1
<b>H</b> ypertension Resting BP > 140/90 mmHg on at least 2 occasions <u>or</u> current antihypertensive pharmacologic treatment	+1
<b>A</b> ge 75 years or older	+2
<b>D</b> iabetes mellitus Fasting glucose > 125 mg/dL or treatment with oral hypoglycemic agent and/or insulin	+1
<b>S</b> troke, TIA, or TE Includes any history of cerebral ischemia	+2
<b>V</b> ascular disease Prior MI, peripheral arterial disease, or aortic plaque	+1
<b>A</b> ge 65 to 74 years	+1
<b>S</b> ex Category (female) Female gender confers higher risk	+1

Figure 6. CHA<sub>2</sub>DS<sub>2</sub>-VASc Criteria for Atrial Fibrillation

#### (a) Score Assessment

- 0 → Anticoagulation is not needed
- 1 → Anticoagulation possibly needed
  - Compute for HAS-BLED score for benefit-risk assessment
  - Also check patient's risk factors and other underlying medical conditions
- ≥ 2 → Anticoagulation is needed
  - If their HAS-BLED score is high, use a **Watchman device**
    - Used to prevent patient from forming clots, if they can't be on anticoagulants
    - The left atrial appendage is occluded so that clots cannot be formed

### (ii) Anticoagulant Drugs

#### (a) Warfarin

- Drug of choice**, especially for patients with the following conditions:

- Valvular A-fib
- Chronic Kidney disease
- Liver Disease

#### • Monitor PT/INR level

- PT/INR: prothrombin time international normalized ratio
- Level must be kept at a range of 2-3
  - < 2 → risk of clots
  - > 3 → risk of bleeding

#### (b) Direct Oral Anticoagulants (DOACs)

- Generally good for patients with non-valvular A-fib
- Lower risk of bleeding compared to Warfarin

#### (i) Factor Xa inhibitors

- Rivaroxaban
- Apixaban
- Edoxaban

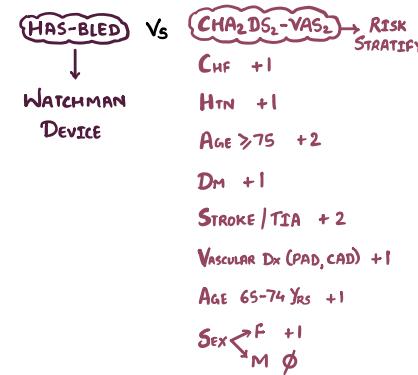
#### (ii) Factor IIa inhibitors

- Dabigatran

#### (c) Heparin

- Unfractionated heparin or Low molecular weight heparin
- Easily titratable
- Monitored via the PTT
  - Too high PTT → decrease the dose
  - Too low PTT → increase the dose
- Can be used as a 'bridge'
  - To bridge a patient into DOAC or Warfarin
  - E.g., useful for patient on Warfarin (to be hypercoagulable for a small period of time)

### ANTICOAGULATION %



WARFARIN:  
-VALVULAR AF  
-CKD OR LIVER Dx  
-INR = 2.3

DOAC's  
Xa-I  
-RIVAROXABAN  
-APIXABAN  
IIa-I  
-DABIGATRAN  
-NON VALVULAR AF

HEPARIN  
-UFH OR LMWH  
-HOSPITAL  
-PTT  
-BRIDGE



## VII) REVIEW QUESTIONS

1) What type of Atrial Fibrillation developed less than 7 days ago?

- a) New Onset Atrial Fibrillation
- b) Paroxysmal Atrial Fibrillation
- c) Persistent Atrial Fibrillation
- d) Long-Standing Atrial Fibrillation

2) Which test/s is/are considered gold standard in diagnosing Atrial Fibrillation?

- a) Chest X-Ray
- b) Complete Blood Count
- c) Electrocardiogram
- d) Urine Metanephrite Test

3) Which disease is known to be associated with excessive alcohol intake?

- a) Torsades de pointes
- b) Holiday Heart Syndrome
- c) Myocardial Infarction
- d) Acute onset pulmonary embolism

## VIII) REFERENCES

- Khan, F., Vaillancourt, C., & Bourjeily, G. (2017). Diagnosis and management of deep vein thrombosis in pregnancy. *BMJ Clinical Research*, 357. <https://doi.org/10.1136/bmj.j2344>

