Tachyarrhythmias

Ahmad Sheyyab MD, FASN
Assistant professor of medicine

Interpretation of ECG

Dr.Ahmad Sheyyab MD, FASN

ECG waves:

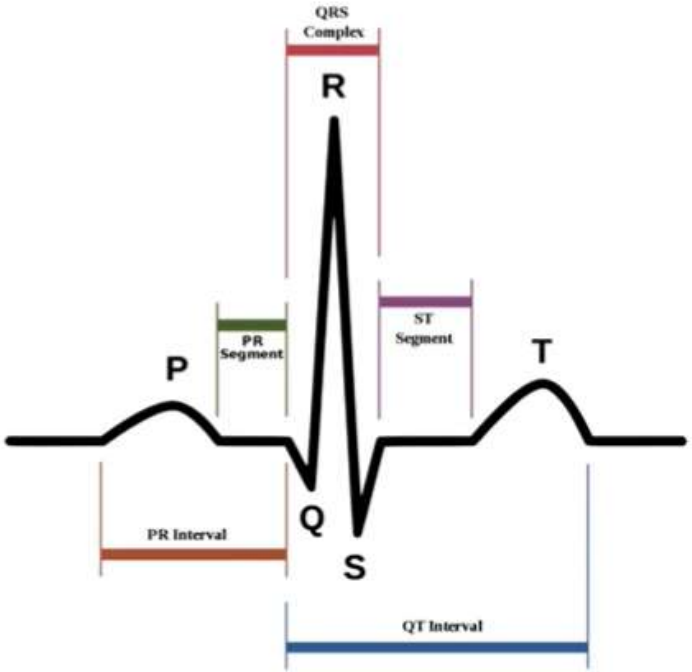
- **P wave: Atrial depolarization**

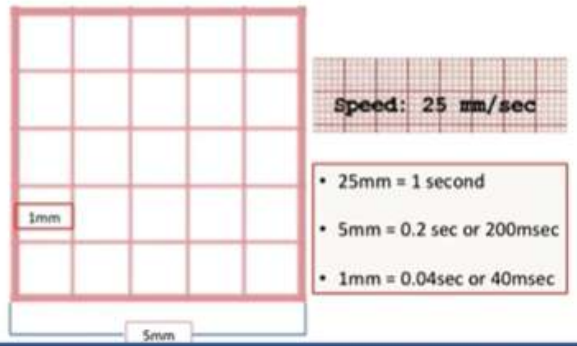
- PR interval: time for impulse to travel from SA node to myocardium

- **QRS: Ventricular depolarization**

- ST segment: isoelectric period before repolarization

- **T wave: Ventricular repolarization**





ECG waves:

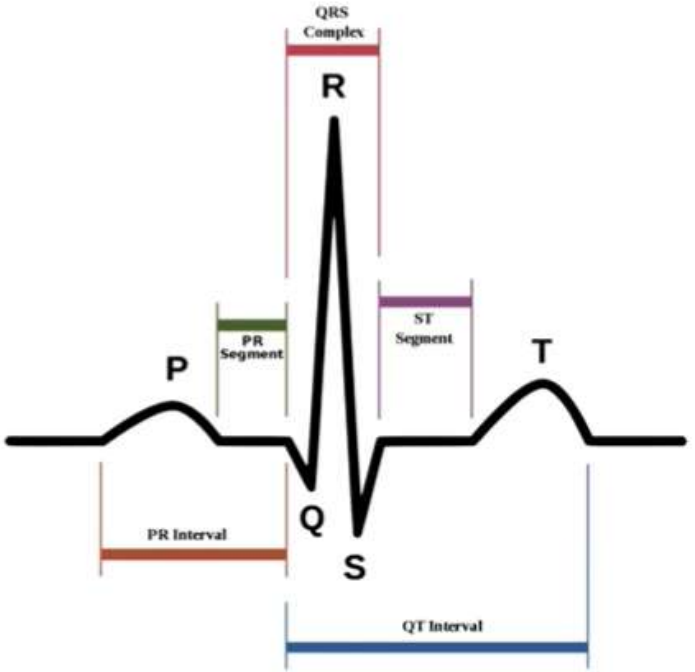
- **P wave: Atrial depolarization**

- PR interval: time for impulse to travel from SA node to myocardium

- **QRS: Ventricular depolarization**

- ST segment: isoelectric period before repolarization

- **T wave: Ventricular repolarization**



ECG segment:	Duration:	Squares number:
P	< 80 milliseconds	2 small
PR*	< 200 millisecond	5 small (1 large)
QRS	< 120 millisecond	3 small

*PR is measured from the beginning of p wave till the R peak

ECG principles:

Wave correlation:

- P wave = atrial depolarization
- QRS = ventricular depolarization
- T = ventricular repolarization

Wave width (duration):

- absent wave = no conduction
- Long duration = slow conduction (via muscles)
- Short duration = fast conduction (via nerves)

Wave height: muscle hypertrophy

Purkinje fibers: anatomy and physiology:

SV node

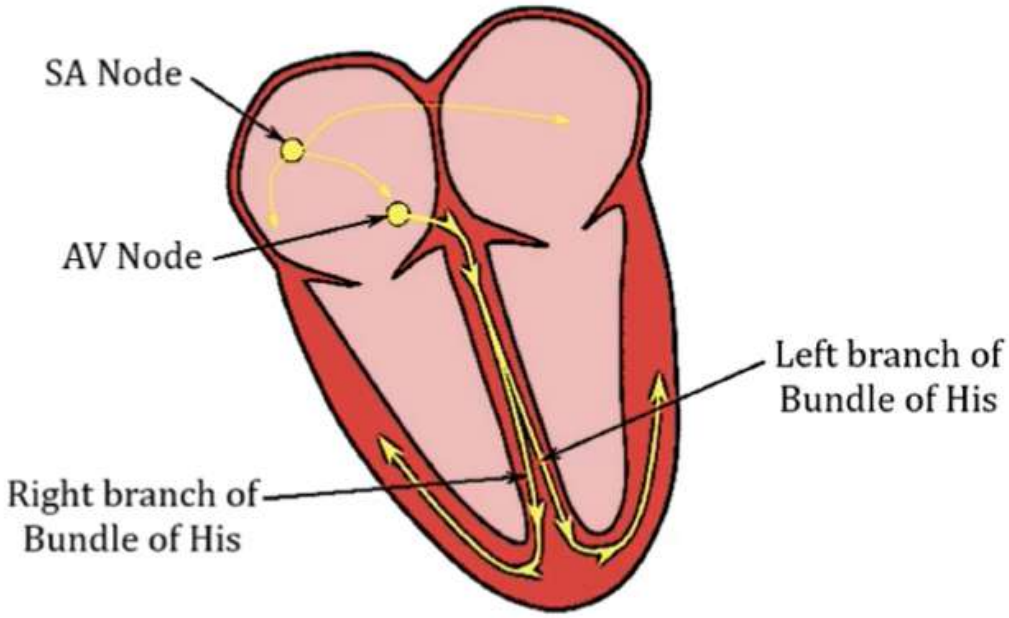
- Automatic pacing

AV node

- Delay between contractions of atria and ventricles
- to allow for ventricular filling)

Buddle of his and branches

- Fast depolarization of the ventricles

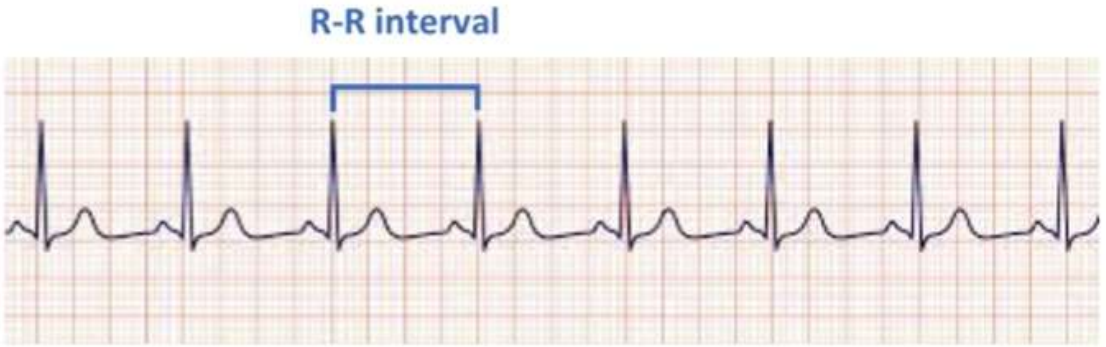


Approach to ECG:

1. Patient profile *(name, date of birth)*
2. ECG quality *(calibration, speed)*
3. Rate?
4. Rhythm?
5. PQRST interpretation
   - P wave
   - PR interval
   - QRS segment
   - ST segment
   - T wave

Heart Rate:

Heart rate (regular) = 300 / number of big squares between R raves



4 squares → HR = 300 / 4 squares
= 75 Beats / minutes

Heart rate (irregular) = Number of R waves over a 10 second period x 6

Heart Rate:

Heart rate (irregular) = Number of R waves over a 10 second period x 6

Beats = 14 beasts in 10 seconds → Heart rate = 14 X 6 = 84 beats/min

Heart rate interpretation:

Slow <60 beat / min	Fast > 100 beats /min
Bradycardia	Tachycardia
Heart blocks	Arrhythmias

Types of bradycardia	Types of tachyarrhythmias
Sinus bradycardia 50 bpm	Sinus tachycardia usually < 130
Complete heart block 30 bpm	Atrial fibrillation Variable ventricle response
	Atrial flutter = 150 bpm
	Supraventricular tachycardia > 150 bpm
	Ventricular tachycardia Variable

Heart rhythm:

1. P-wave? *(lead II , III, aVF)*
2. Regularity? *(QRS)*
3. QRS rate?
4. QRS narrow or wide?

Atrial fibrillation
Absent p waves
Irregular QRS

Narrow QRS		Wide QRS	
Regular	Irregular	Regular	Irregular
- Sinus tachycardia - SVT	Atrial fibrillation (AF)	- SVT with aberrancy - Ventricular tachycardia (monomorphic)	- AF with aberrancy - Ventricular tachycardia (polymorphic)
- Junctional tachycardia	- MAT	- Antidromic AVRT	

SVT = Supraventricular tachyarrhythmias, AF = Atrial fibrillation, VT = ventricular tachycardia, MAT = multifocal atrial tachycardia
AAVRT = Atrioventricular reentrant tachycardia

Sinus tachycardia

- Sinus rate >100bpm but wave are generated from SA node
- Sinus tachycardia is a physiological response to a stimulus
- Treatment is aimed at correction of the underlying cause.

Avoid Beta blockers except in hyperthyroidism.

Cause of tachycardia
Exercise & excitement
pain
anemia
hypovolemia
thyrotoxicosis
acute pulmonary embolism
drugs
heart failure

Mechanisms of wide QRS:

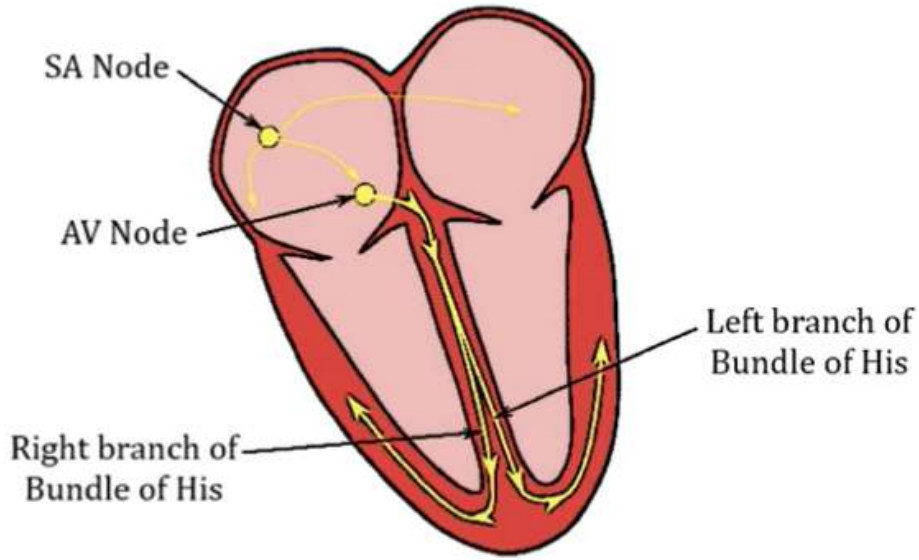
1. Ventricular arrhythmias

- Fast depolarization of the ventricles

2. Atrial arrhythmias

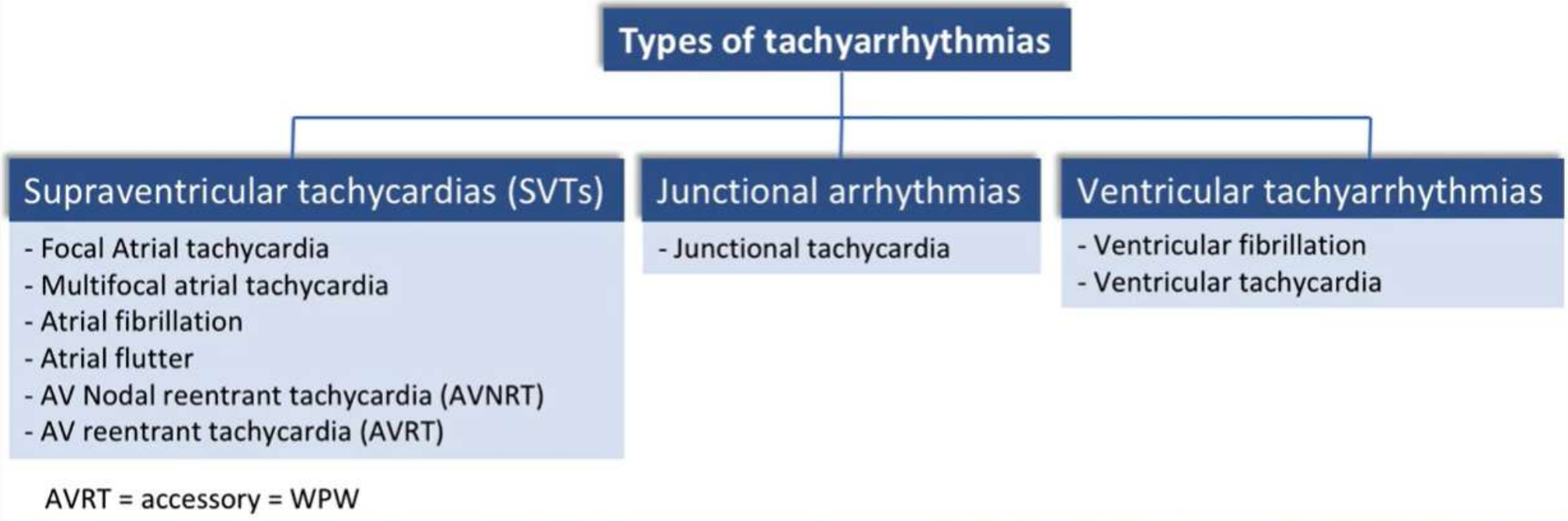
- **Aberrancy or Aberrant conduction**
  Conduction through the atrioventricular node with delay or block (LBBB or RBBB)

- **Accessory pathway**
  conduction through an accessory pathway (a position away from Purkinje fibers)



Tachyarrhythmias

Definition: An abnormal heart rhythms with a ventricular rate of 100 bpm or more



AVRT = accessory = WPW

Mechanism of Tachyarrhythmias

- **Increased automaticity.**

  repeated spontaneous depolarization of an ectopic focus, often in response to catecholamines

- **Re-entry**

  The tachycardia is initiated by an ectopic beat and sustained by a re-entry circuit.

- **Triggered activity**

  It is a form of secondary depolarization arising from an incompletely repolarized cell membrane (acute coronary syndrome).

Signs and symptoms:

Symptoms:	Signs:	Mechanism:
dizziness or lightheadedness Fainting decreased level of consciousness	Hypotension	Cerebral hypoperfusion
Palpitations or fluttering	Tachycardia	Feeling heart beats
Shortness of breath	Pulmonary edema	Impaired heart contractility
Chest pain	Active pain on exam	cardia ischemia

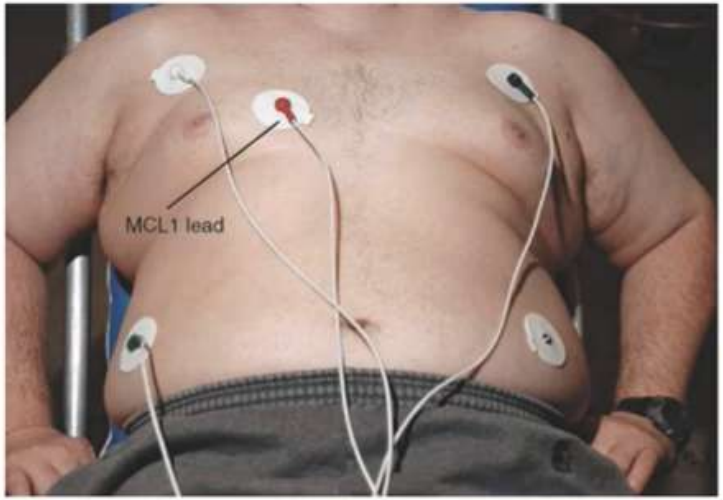
Arrhythmias: Diagnostic approach

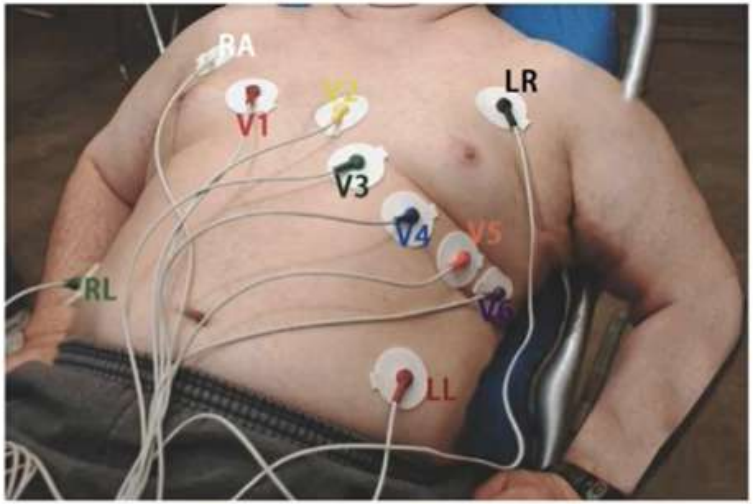
- **Hemodynamic assessment**

  Blood pressure, heart rate, mental confusion

- **Stable patient → 12 lead Electrocardiogram (ECG)**

- **Unstable patient → Cardiac monitor**





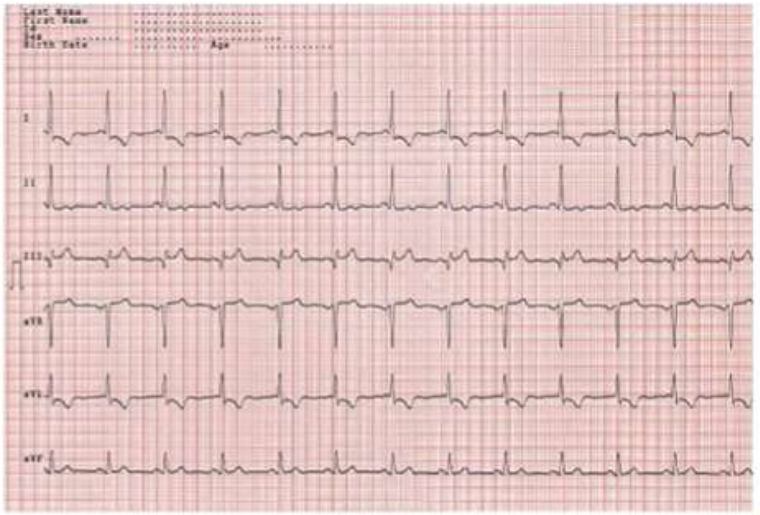
Arrhythmias: Diagnostic approach

- **Hemodynamic assessment**

  Blood pressure, heart rate, mental confusion

- **Stable patient → 12 lead Electrocardiogram (ECG)**

- **Unstable patient → Cardiac monitor**



Focal Atrial tachycardia

- originating from a single ectopic focus within the atria but outside of the sinus node.
- these are usually paroxysmal and self-limited.

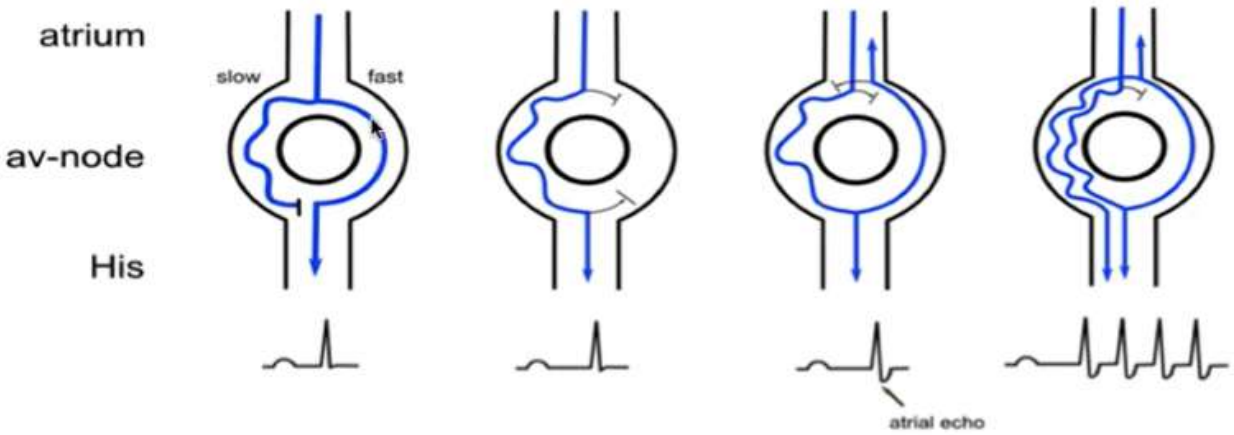
Multifocal atrial tachycardia

- Usually secondary to underlying illness (usually pulmonary)
- Treatment is based on **oxygen therapy** and supportive care
- Treat the underlying cause
- Medication maybe used as an alternative therapy if the above fails

Focal Atrial tachycardia

Multifocal atrial tachycardia

Atrioventricular nodal reentrant tachycardia (AVNRT)

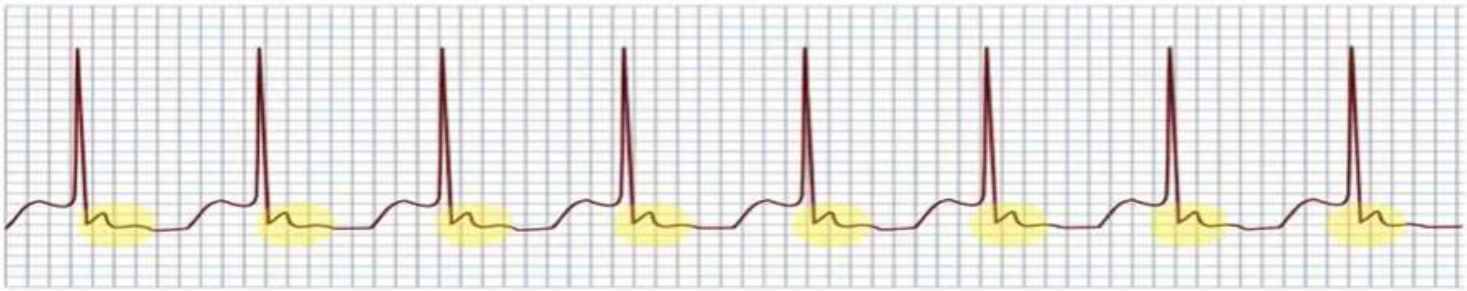


1st beat 2nd beat 3rd beat Arrhythmia

Atrioventricular nodal reentrant tachycardia (AVNRT)

- **P waves** are **either not visible or are seen immediately before or after the QRS complex.**
- **QRS complex** is usually of normal shape.

Atrioventricular nodal reentrant tachycardia (AVNRT)



- **P waves** are **either not visible or are seen immediately before or after the QRS complex.**
- **QRS complex** is usually of normal shape.

Atrioventricular reentrant tachycardia (AVRT)

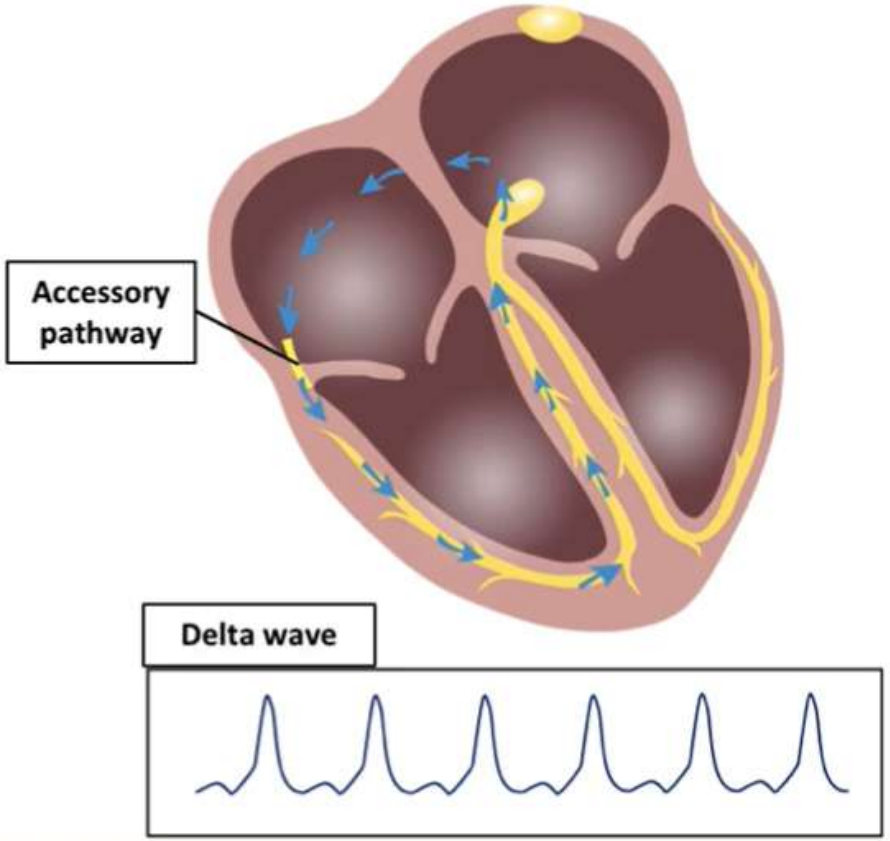
- occurs due to the presence of **an accessory pathway** that connects the atria and ventricles and is capable of antegrade or retrograde conduction, or both.

- **Bypass consequence:**

1- Asymptomatic with ECG changes

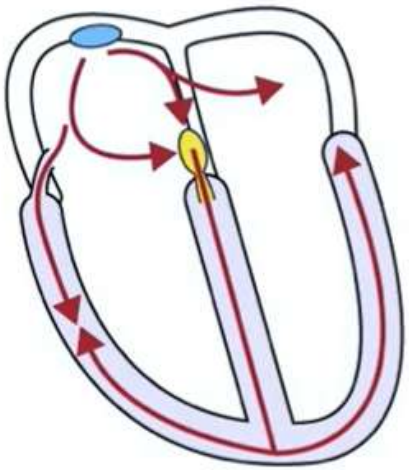
2- Regular Tachycardia and palpitation

3- Atrial Fibrillation



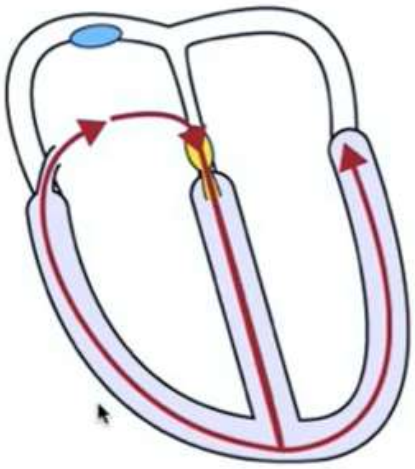
Atrioventricular reentrant tachycardia

Pre-excitation



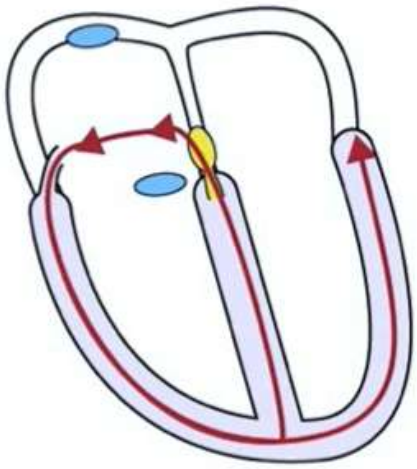
- Short PR interval
- In this case the PR segment cannot be seen.

Orthodromic AVRT
Antegrade conduction through atrioventricular node



- Normal QRS duration
- No delta wave
- Retrograde P-wave after QRS

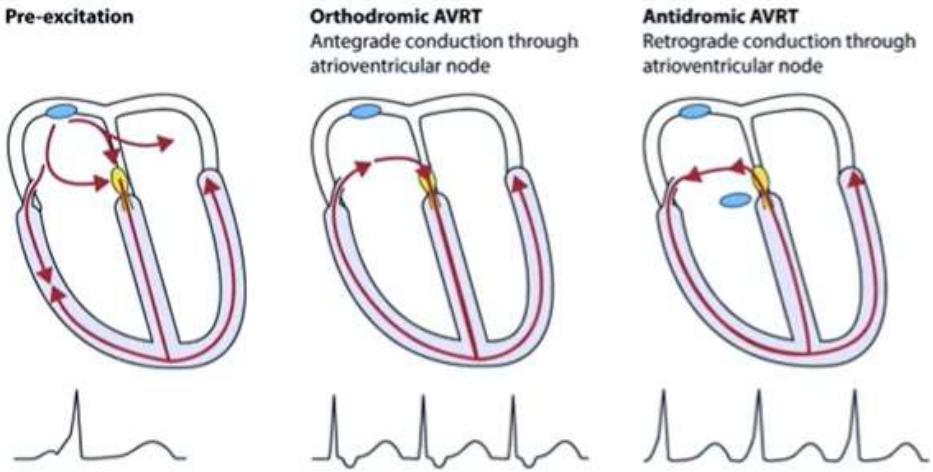
Antidromic AVRT
Retrograde conduction through atrioventricular node



- Wide QRS complex with delta wave
- P-wave rarely seen
- If P-wave visible, it is retrograde and occurs just before the QRS

Atrioventricular reentrant tachycardia (AVRT)

- Accessory pathway
- Accessory pathways alters the activation of atria and ventricle



Atrioventricular nodal reentrant tachycardia (AVNRT)

- Nodal reentry
- Activates both atria and ventricles at the same time

Atrioventricular reentrant tachycardia (AVRT)

Management:

Unstable WPW → DC cardioversion

Stable WPW

1. Vagal maneuvers
2. Procainamide are available for use in resistant cases
3. Involve cardiology consultant

Long-term management: Radiofrequency ablation of the accessory pathway

! Avoid use of calcium channel blocker or IV beta blocker

Atrioventricular nodal reentrant tachycardia (AVNRT)

Management:

Unstable SVT → DC cardioversion

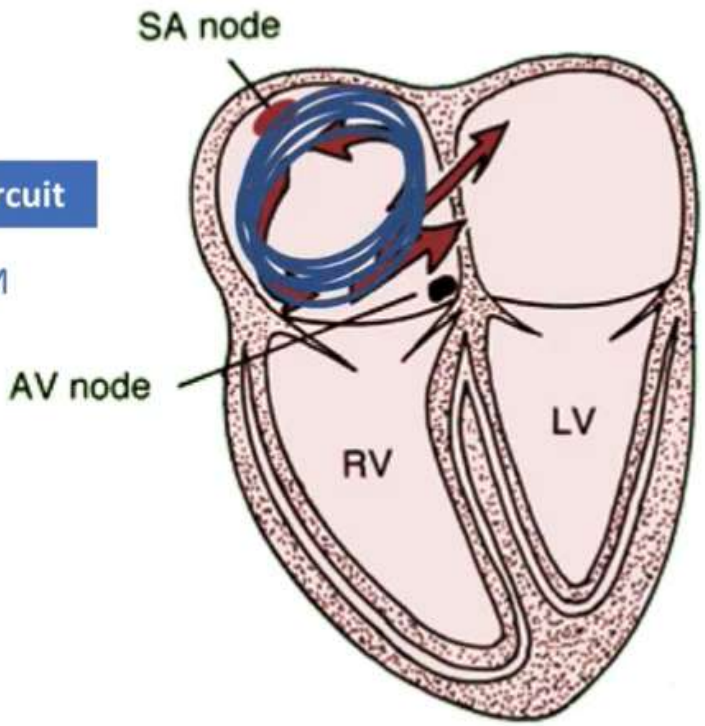
Stable SVT

1. Vagal maneuvers
2. IV adenosine
3. IV calcium channel blocker or IV beta blocker

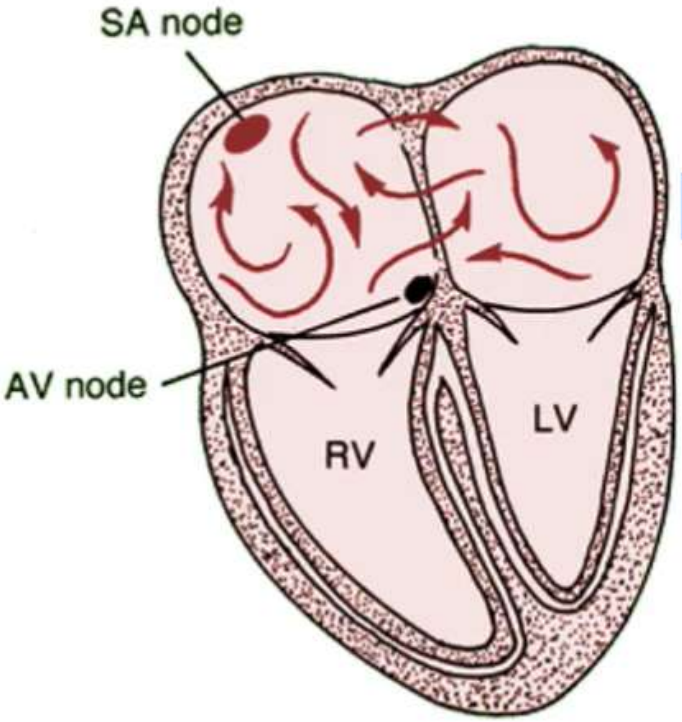
Atrial flutter

Renentry circuit

300 BPM



Atrial fibrillation



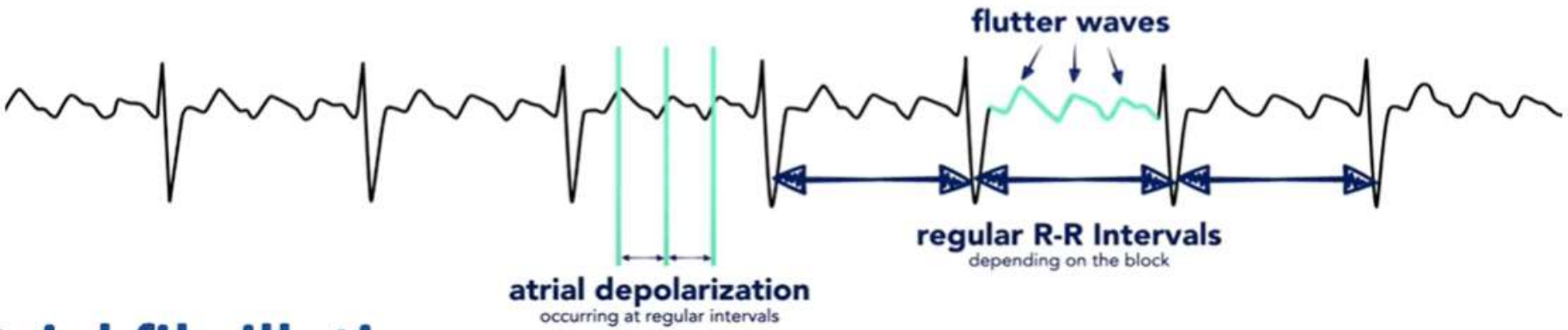
Chaotic activation

400 BPM

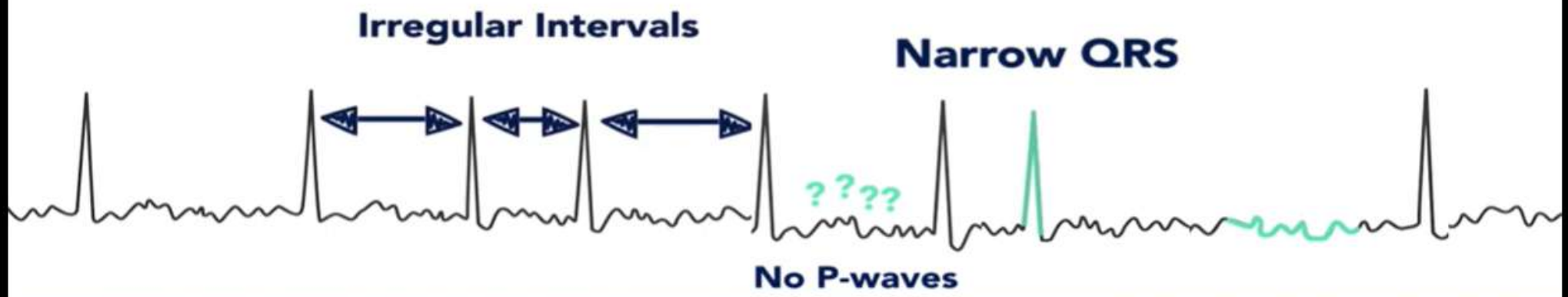
Atrial flutter

Atrial fibrillation

Atrial flutter



Atrial fibrillation



Atrial flutter management

Atrial fibrillation management

- Our initial approach to the management of patients with atrial flutter is the **same as our approach for atrial fibrillation.**
- Management of atrial flutter involves an assessment of the need for cardioversion.
- Ventricular rate slowing therapy, and anticoagulation therapy.

Risk factors of atrial fibrillation

1. **Hypertension**
2. **Age (risk factors)**
3. Coronary artery disease (including acute MI)
4. Valvular heart disease, especially rheumatic mitral valve disease
5. Sinoatrial disease
6. Hyperthyroidism
7. Alcohol
8. Cardiomyopathy
9. Congenital heart disease
10. Pulmonary embolism ,Chest infection
11. Idiopathic (lone atrial fibrillation)

Types of Atrial fibrillation

- **First diagnosed:**
  Not diagnosed previously, irrespective of duration or presence of AF-related symptoms
- **Paroxysmal AF = intermittent**
  characterized as sporadic episodes. Self-terminating, in most cases within 48 hours, but could last up to 7 days.
- **Persistent AF:**
  Lasts longer than 7 days and unlikely to resolve without treatment
- **Permanent:**
  Can not be reverted to normal sinus rhythm despite treatment

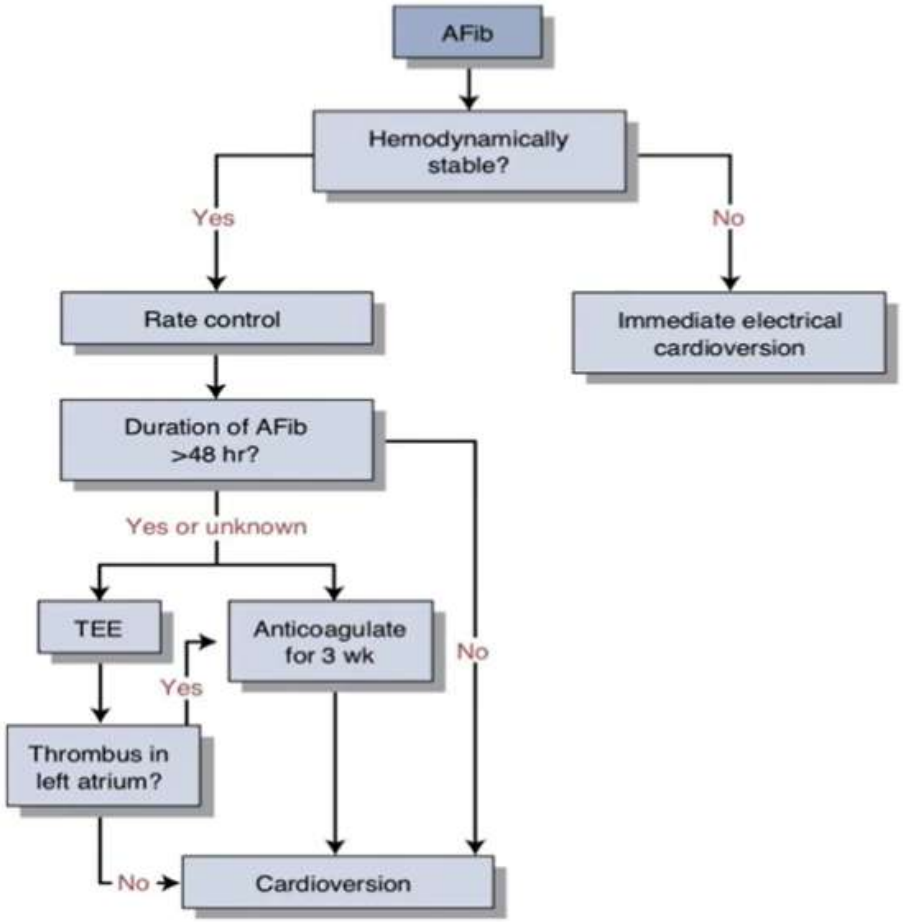
Acute management:

Rate Control:
- target heart rate is <110 bpm, B blockers (preferred agents), CCBs (diltiazem), or digoxin.

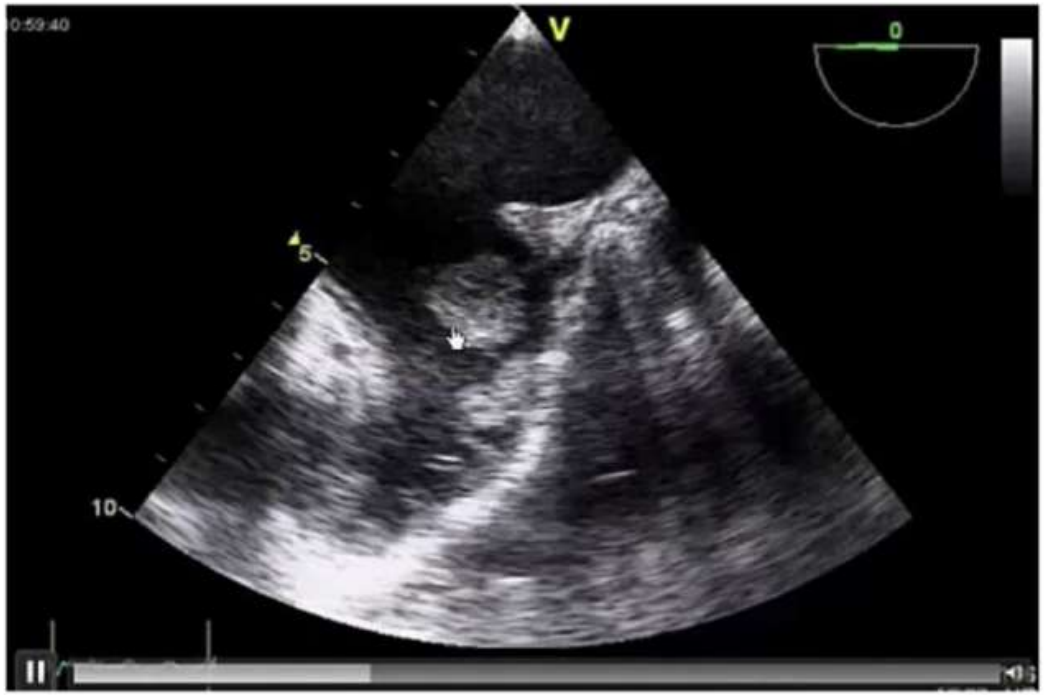
Rhythm control:
--DC cardioversion (Synchronus) is the most effective method to restore sinus rhythm. Chemical cardioversion rates of success are lower and depend on the antiarrhythmic drug used and clinical scenario. (Drug toxicity)

- If it has been > 48 hours since the onset of Afib (or you are unsure of the onset/duration) and the patient is stable, you must achieve adequate anticoagulation x3 weeks before you attempt cardioversion. As an alternative to preceding anticoagulation, it is reasonable to perform Transesophageal echocardiogram, and if there is no identifiable thrombus, perform a cardioversion. If it has been< 48 hours since the onset of Afib, cardiovert most patients without any preceding anticoagulation.

Acute management:



A.Fibb increase the risk of thrombus formation



Pharmacological cardioversion

When attempting pharmacological cardioversion, use is based on duration of symptoms.

For A-fib> 7 days:

- 1st line: dofetilide (class III antiarrhythmic agent)
- 2nd line: amiodarone or ibutilide

For A-fib< 7 days:

- 1st line: dofetilide, ibutilide,flecainide or propafenone (previously, dronedarone*)
- 2nd line: amiodarone (Exception: If< 48 hours and poor cardiac function, amiodarone is 1st line.)

Chronic A.fib Management

1) Rate control:

- with a B blocker or Calcium channel blocker

2) Anticoagulants:

to prevent cardioembolic cerebrovascular accident (CVA), risk stratify patients with CHA2DS2-VASc score.

- For patients with a score >1 anticoagulation is generally indicated unless high bleeding risk.

CHADS-VASC

Letter	Risk factor	Score
C	Congestive heart failure/LV dysfunction	1
H	Hypertension	1
A_2	Age ≥75	2
D	Diabetes mellitus	1
S_2	Stroke/TIA/thrombo-embolism	2
V	Vascular disease*	1
A	Age 65–74	1
S	Sex category (i.e., female sex)	1
	Maximum score	9

Congestive heart failure/LV dysfunction means LV ejection fraction ≤40%. Hypertension includes the patients with current antihypertensive medication. *Prior myocardial infarction, peripheral artery disease, aortic plaque. LV: left ventricular, TIA: transient ischemic attack

Radiofrequency Ablation

- **Radiofrequency ablation of the AV node with subsequent permanent pacing**

is a treatment for patients with refractory Afib and for those who cannot tolerate the meds needed for rate or rhythm control, HFrEF, & high Afib burden.

- **Radiofrequency ablation, or isolation of the pulmonary veins**

Is a procedure becoming increasingly popular in treating recurrent, drug refractory, symptomatic A-fib, although it is not yet established as 1st line therapy

Postoperative A.Fib

- **Is usually has good prognosis and is short-lived**
- For patients undergoing cardiac surgery, give an oral beta-blocker to prevent postoperative Afib (unless contraindicated).
- For those who develop postoperative Afib, achieve rate control with AV nodal blocking drugs (beta-blockers, calcium channel blockers, or digoxin).
- Routine postoperative amiodarone is not indicated for the prevention of atrial fibrillation

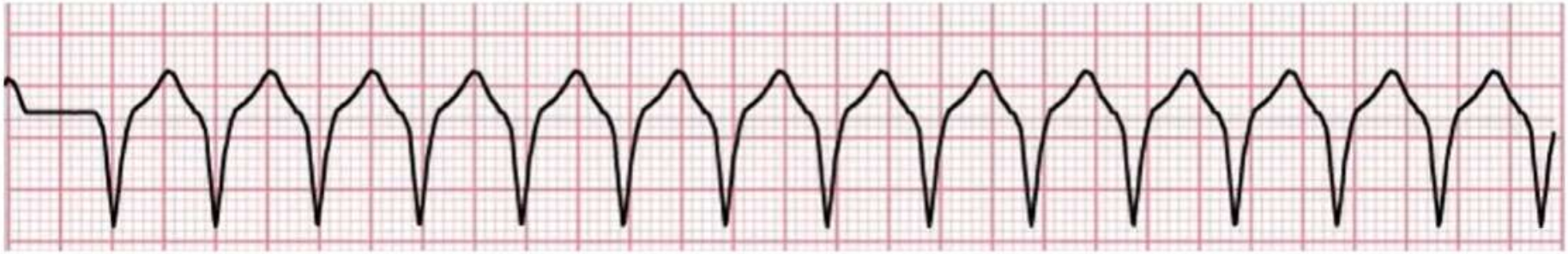
Ventricular tachyarrhythmias

- Ventricular fibrillation
- Ventricular tachycardia

Ventricular Fibrillation

- Rhythm: chaotic & disorganized waves
- Patient is unconscious (lethal)
- It is an emergency situation, requires CPR
- Shockable rhythm – requires defibrillation (unsynchronized DC shock)

Ventricular tachycardia



- Types: monomorphic Vs polymorphic (poor prognosis)
- Rhythm: wide complex tachycardia
- Patient stability varies (stable, unstable, unconscious)
- Management varies depending on clinical stability

Management of ventricular fibrillation:

Immediate assessment of patient stability takes precedence over any further diagnostic evaluation.

- **Unresponsive or pulseless**: cardioversion (without sedation) + epinephrine +/- amiodarone (ACLS*)
- **Unstable but conscious**: immediate synchronized cardioversion (with sedation)
- **Stable patient:** a focused diagnostic evaluation may proceed to determine the etiology of the arrhythmia and guide specific therapy.

*ACLS = advance cardiac life support

Unstable patients: Defibrillation improves mortality

Healthcare setting:

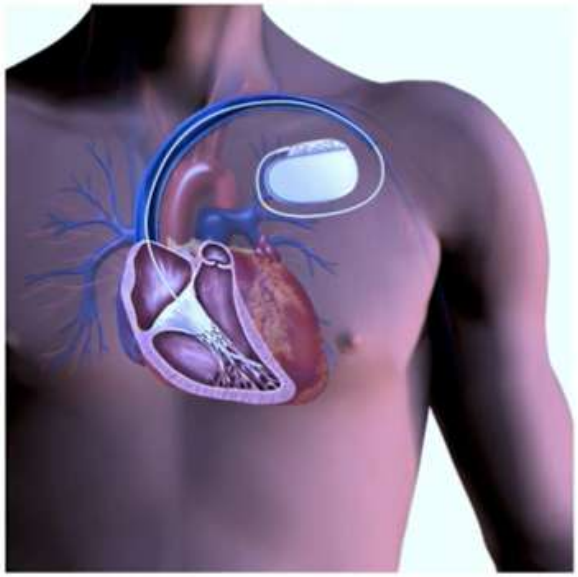
Cardioversion

Immediate Synchronized cardioversion + medications

Ambulatory setting:

automated Eternal Defibrillator (AED)

Dealing with future V.tach



- Ideally all patient with sustained VT should undergo placement of Implantable **Implantable cardioverter-defibrillator (ICD)**
- It has longer benefits

Monomorphic ventricular tachycardia

Polymorphic ventricular tachycardia

Treatment of Polymorphic ventricular tachycardia

In addition to immediate defibrillation, further therapy is intended to treat underlying disorders and to prevent recurrences.

- **QT prolongation + V.tach = Torsades de pointes.**
  - Intravenous magnesium sulfate is first-line therapy, as it is highly effective for both treatment and prevention of recurrence .
  - Correct metabolic abnormailities including electrolytes
  - Other treatments: ATP pacing, beta-blocker (congenital)
- **V.tach + no QT prolongation = the most likely cause is myocardial ischemia**
  - Betablocker (if BP tolerates)
  - Amiodarone to prevent recurrence
  - Angiography and revascularization
  - Intravenous magnesium sulfate is ineffective