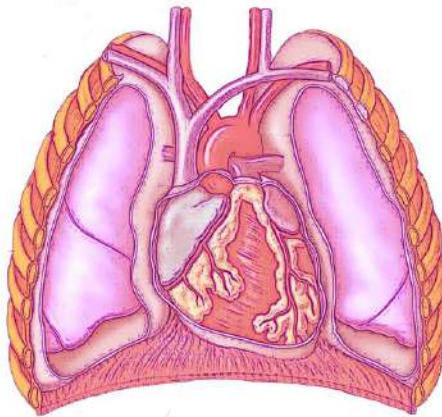




Internal Medicine



Topic : Tachyarrhythmia Notes
Done by : Johainah Taha

ECG waves:

* The more muscle you have → Stronger voltage.

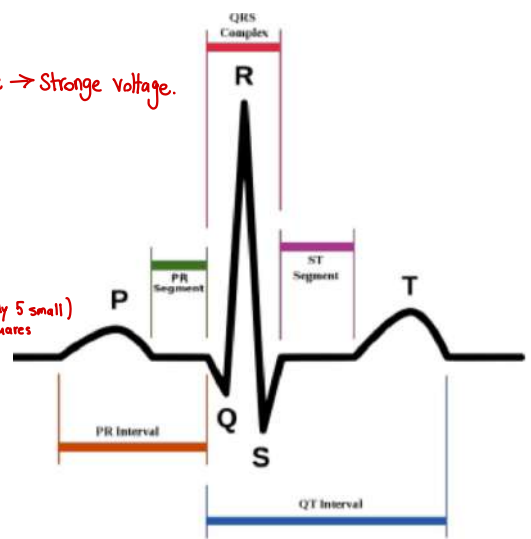
- **P wave: Atrial depolarization**
- **PR interval:** time for impulse to travel

from SA node to myocardium → to know the conduction between Atrium + vent.

- **QRS: Ventricular depolarization** (normally 5 small Squares)

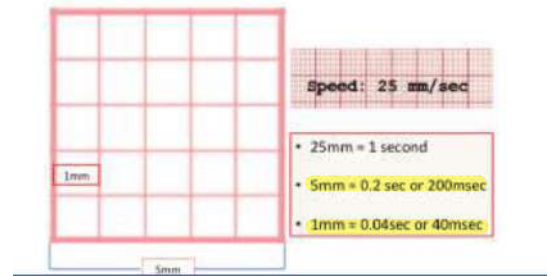
- **ST segment:** isoelectric period before repolarization

- **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) → example BBB.
- Short duration = fast conduction (via nerves) → example arrhythmia

Wave height: muscle hypertrophy

Purkinje fibers: anatomy and physiology:

SV node

- Automatic pacing

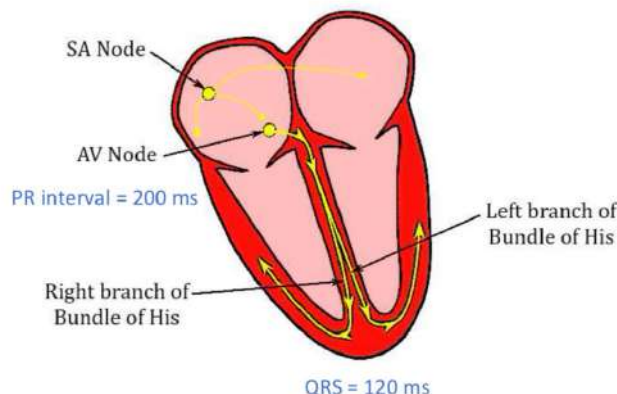
AV node

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

برضه يساعد في طاية ال Arrhythmia في Atrium من الانتقال ال Ventricle

Buddle of his and branches

- Fast depolarization of the ventricles
- ↳ + synchronised



!! Transmission via nerves is faster than transmission though muscles

لهو صاير block حتمتيل بين Muscle
 ↳ ابطأ ع
 ↳ Wide QRS Complex

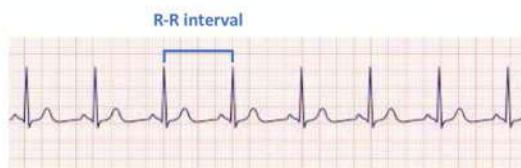
Approach to ECG:

1. Patient profile (name, date of birth)
2. ECG quality (calibration, speed)
25mm/sec
3. Rate? له مستطيل طوله 2 large squares → Check it when having tachy or brady.
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 waves

- Normal Heart rate: 60 – 100 Beats/minute



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

→ 5 large squares



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

((مهم نشوف الرقم))

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?

Check the long strip.

→ The most common arrhythmia.
Atrial fibrillation
Absent p waves
Irregular QRS: irregularly irregular.

Narrow QRS → Atrium		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

Arhythmia هارت فوف Ventricles ولا توصل ال AVnode و

QRS يعني الونج طوي فينخل ال QRS
→ NARROW !!

هون عنا اجماليات
Ventricular
SVT with heart block
↓
عنا مشكلت هون
① SVT came above ventricles
+ aberrancy (heart block)
نملا ال wave توصل على Bundle of his
في جزء مائل block ال activation حيزون
wide ← muscles
QRS

Sinus tachycardia



- Sinus rate $>100\text{bpm}$ 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.

Cause of tachycardia

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

Avoid Beta blockers except in hyperthyroidism.

→ we can use it but not as acute treatment.
→ in decompensated HF; we tend to start B-blockers

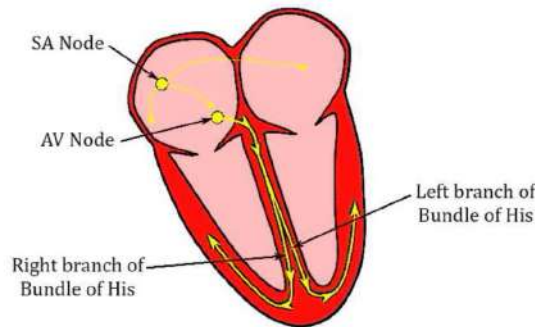
Mechanisms of wide QRS:

1. Ventricular arrhythmias → wide QRS

- 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 (wide + Slow QRS)**
conduction through an accessory pathway (a position away from Purkinje fibers)



!! Transmission via nerves is faster than transmission through muscles

Tachyarrhythmias → it differs from tachycardia.

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

Types of tachyarrhythmias

Supraventricular tachycardias (SVTs)

- Focal Atrial tachycardia
- Multifocal atrial tachycardia
- Atrial fibrillation
- Atrial flutter
- AV Nodal reentrant tachycardia (AVNRT)
- AV reentrant tachycardia (AVRT)

AVRT = accessory = WPW

Junctional arrhythmias

- Junctional tachycardia

Ventricular tachyarrhythmias

- Ventricular fibrillation
- Ventricular tachycardia

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 (irregular pulse)	Feeling heart beats
Shortness of breath	Pulmonary edema	Impaired heart contractility
Chest pain (not common)	Active pain on exam	cardia ischemia ↳ ↑HR → ↑demand O ₂

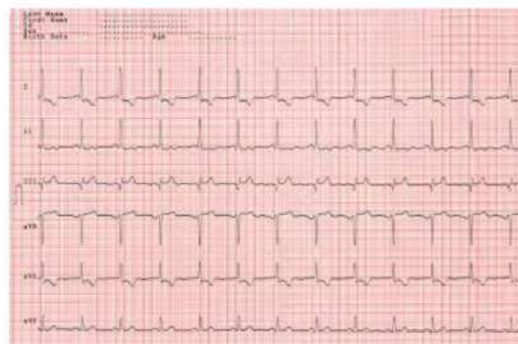
Arrhythmias: Diagnostic approach

- * **Hemodynamic assessment**

Blood pressure, heart rate, mental confusion
↳ with symptoms

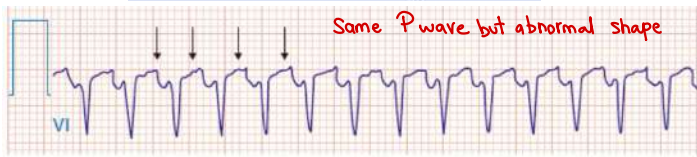
- **Stable patient → 12 lead Electrocardiogram (ECG)**

- **Unstable patient → Cardiac monitor** ← Semi-Con.



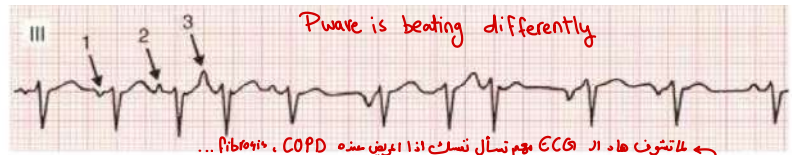
Supraventricular tachyarrhythmias (SVTs)

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**.

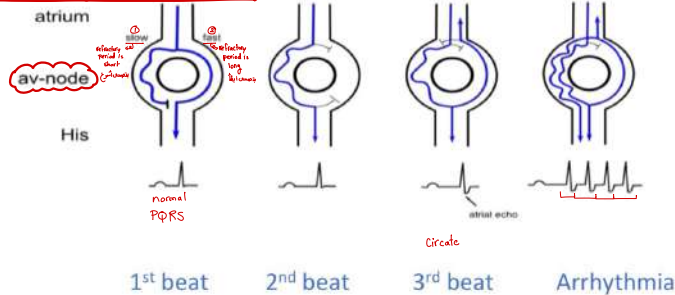
Multifocal atrial tachycardia



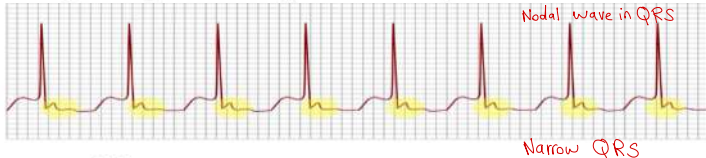
- Usually **secondary to underlying illness** (usually **pulmonary**)
- Treatment is based on **oxygen therapy** and **supportive care**
- Treat the underlying cause
- Medication maybe used as an alternative therapy if the above fails

AV Nodal reentrant tachycardia [AVNRT]

Apathophysiology of how beat start:



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

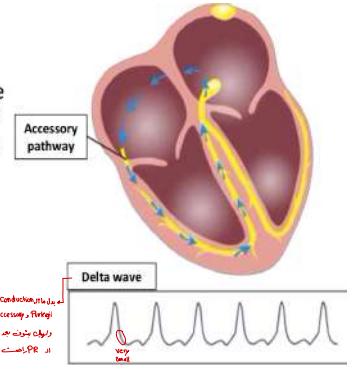


- Nodal reentry**
- Activates both atria and ventricles at the same time**



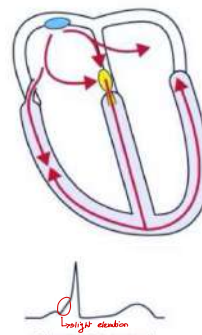
AV reentrant tachycardia (AVRT)

- 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:**
 - Asymptomatic with ECG changes
 - Regular Tachycardia and palpitation
 - Atrial Fibrillation

Pre-excitation



Orthodromic AVRT

Antegrade conduction through atrioventricular node



Antidromic AVRT

Retrograde conduction through atrioventricular node



- Short PR interval**
- In this case the PR segment cannot be seen.**
- Normal QRS duration**
- No delta wave due to no delay**
- Retrograde P-wave after QRS**
- Wide QRS complex with delta wave**
- P-wave rarely seen**
- If P-wave visible, it is retrograde and occurs just before the QRS**

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

Management

Unstable SVT → DC cardioversion

Stable SVT

- Vagal maneuvers slow: deep breath, put ice, spit can work.
- IV adenosine a short acting beta blocker - not blocks arrhythmia
- IV calcium channel blocker or IV beta blocker

Unstable WPW → DC cardioversion

Stable WPW

- Vagal maneuvers
- Procainamide** are available for use in resistant cases for short-term management
- Involve cardiology consultant

Long-term management: **Radiofrequency ablation** of the accessory pathway كوبين

! Avoid use of calcium channel blocker or IV beta blocker they block arrhythmia

Wolff Parkinson white syndrome

can lead to AF

delta

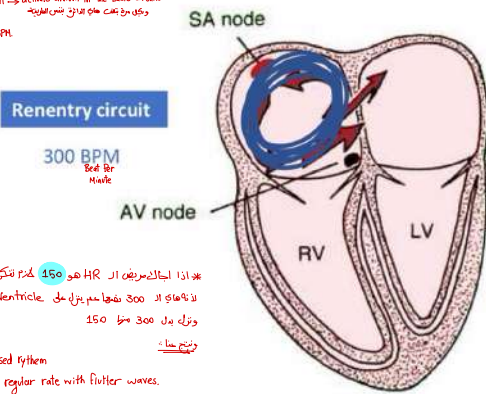
wide irregular tachy

مراجعة 1 و 2 لوني Symptoms

Supraventricular tachyarrhythmias (SVTs)

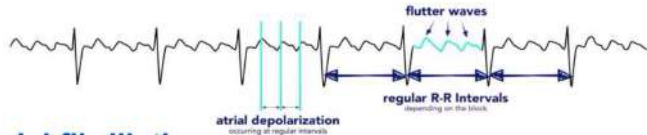
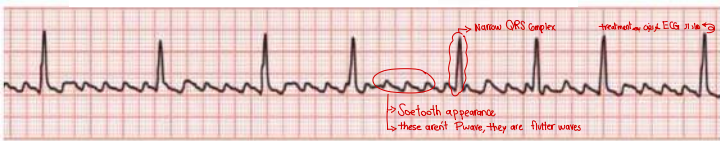
Atrial flutter

* Note organised.
* Reentry circuit → activate atrium in the same circuit
* The rate = 300 BPM



Flutter HR هو 150 bpm
لأنه معدل الـ 300 bpm يتم تقسيمه على 2:1
وتكون الـ 150 bpm
وهذا إذا كان معدل الـ HR هو 150 bpm
وهذا إذا كان معدل الـ HR هو 150 bpm

- Very organised rhythm
- beating at regular rate with flutter waves.



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 is similar to management of the need for cardioversion.

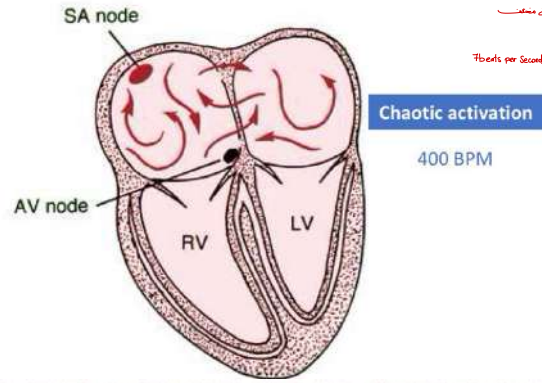
• Ventricular rate slowing therapy, and anticoagulation therapy.

Our goals:
1. heart rate control
2. Anticoagulant therapy
3. Rhythm control (according to case)

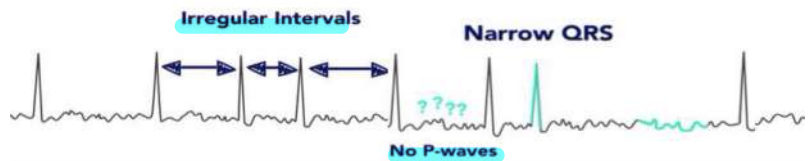
Atrial flutter with variable AV block



Atrial fibrillation



بشكل غير منظم و لكن بمعدل 400 bpm = Rate *
فibrils per second



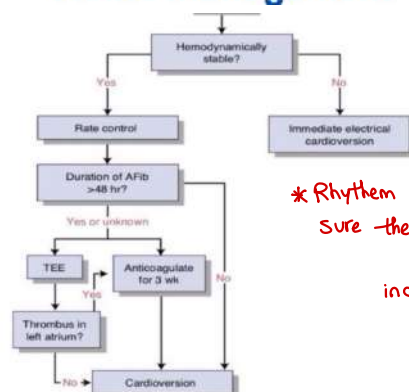
Risk factors of atrial fibrillation

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



* Rhythm control but first make sure there is no thrombus
↓
increased stroke risk

Atrial fibrillation

Acute management:

* Important principles :-

1. it's recontrolled by AV blocking agents by chemical or electrical agents But before that

you have to rule out the present of thrombus

Stroke = Cerebral artery = Left ventricle = Left atrium = Atrial fibrillation = Stroke

↳ So these pt. are of ↑ risk to have a stroke.

A.Fibb increase the risk of thrombus formation

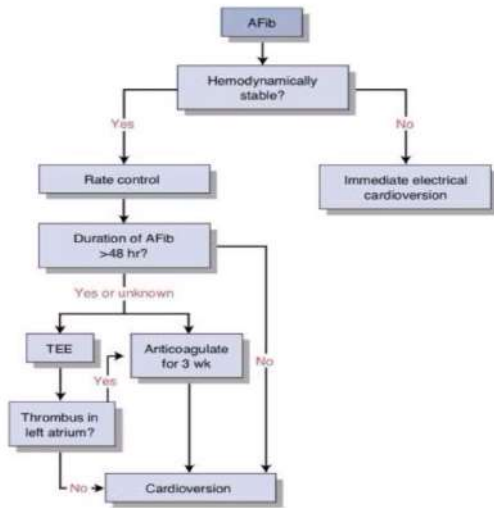
2. Studies found that if AFib is <48h → The risk of Clot formation is very low

↳ I can do Cardioversion DC . (Cardioversion DC is a safe procedure)

if it was AFib >48h → Do a transesophageal echocardiogram (to check if the atria has clot or not) ← مؤمن الوقت أكثر

Or give anti-coagulants for 3 weeks then do DC Cardioversion

1. Give anti-coagulants for 3 weeks
2. Do a DC cardioversion



There is a risk of stroke when performing chemical/electrical cardioversion

Pharmacological cardioversion

التعامل مع الرجفة

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.

Types of anticoagulants:

A) **warfarin**: anticoagulant of choice in patients with valvular disease, mechanical valves, or ventricular assist devices.

B) **direct oral anticoagulants (DOACs)**: factor Xa inhibitors (apixaban), and direct thrombin inhibitors (Dabigatran)

what is DC cardioversion ?

- a medical procedure used to restore normal heart rate rhythm in pts with arrhythmia.
- Electrical shock → to synchronize heart's electrical cycle

Defibrillator → used in VF and Pulseless VT

أهم شي نشيلو لو المريض عنده thrombus أولاً
دعوية Anti-Coag

CHADS-VASC → A very important score.

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

Extra- Notes :

Male 0
Female 1 → no needed anti-coagulants

Male 1
Female 2 → give anti-coagulants

Male ≥ 2
Female ≥ 3 → Anti-coagulants are recommended*

Example :

a 76 year old female with A-Fib, dm, previous stroke, HTN

CHADS₂VASC Score ⇒ Age : 2 / Female : 1 / Stroke : 2 / HTN : 1 total = 6

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.

* it uses heat to destroy abnormal tissue.

- 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
↳ Liver failure, lung fibrosis, thyrotoxicosis

* لا Amiodarone لا يحسن AF

Ventricular tachyarrhythmias

Ventricular fibrillation



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

Management

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

the most imp. thing in CPR
Unstable patients: Defibrillation improves mortality



Healthcare setting:
Cardioversion → *in hospital*

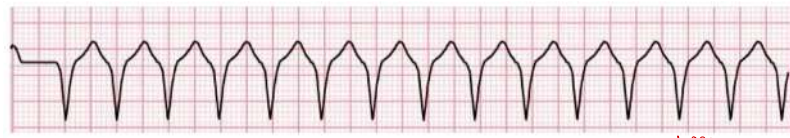


Ambulatory setting:
automated External Defibrillator (AED)

Immediate Synchronized cardioversion + medications

1. Start Chest compression
2. Do ventilation support (30:2)
3. Monitor → to see if he had Shockable rhythm
↓
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

Dealing with future V.tach

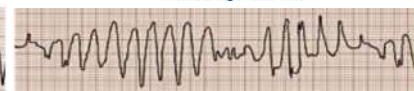


- Ideally all patient with sustained VT should undergo placement of Implantable **cardioverter-defibrillator (ICD)**
- It has longer benefits

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 abnormalities 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

For sustained stable monomorphic VT:

- Give IV amiodarone.

Treat according to diagnosis:

- **VT specifically with acute MI**: Most use amiodarone first. IV lidocaine can be useful.

Do not use CCBs for wide-complex tachycardias. (fear of hemodynamic collapse, hypotension and cardiac arrest)

- **RVOT VT** can be terminated acutely with adenosine. Beta-blockers/calcium channel blockers (CCBs) can be used for long-term management.
- **Patients with NSVT without structural heart disease** have good prognosis, and they do not require further management.

*ACLS = advance cardiac life support