# APPROACH TO TACHYARRYTHMIAS

**PROF.DR.MD.ZAKIR HOSSAIN**

PROFESSOR AND HEAD

DEPARTMENT OF MEDICINE

SZMCH

# TACHYARRYTHMIA

- **Cardiac arrythmia is a disturbance of electrical rhythm of heart.**

- **Cardac arrythmia with rate above 100/min is called tachyarrythmia.**

# Mechanism of tachyarrythmia

A. Increased automaticity.

B. Triggered activity.

C. Rentry.

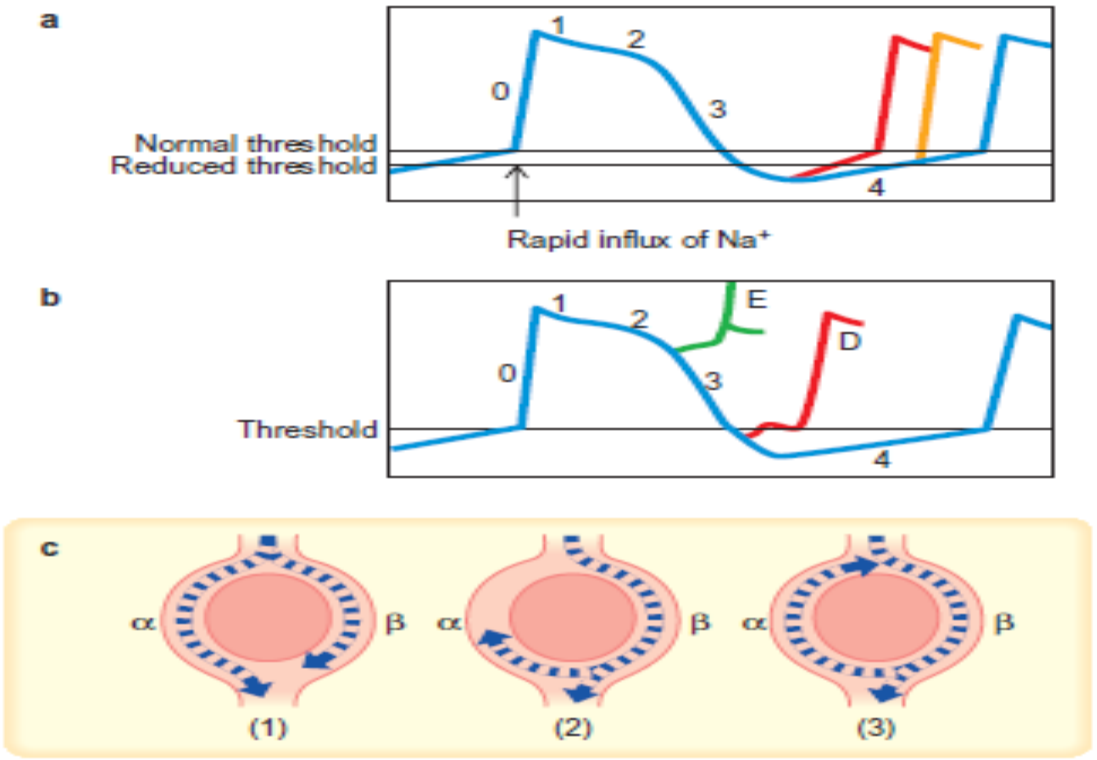
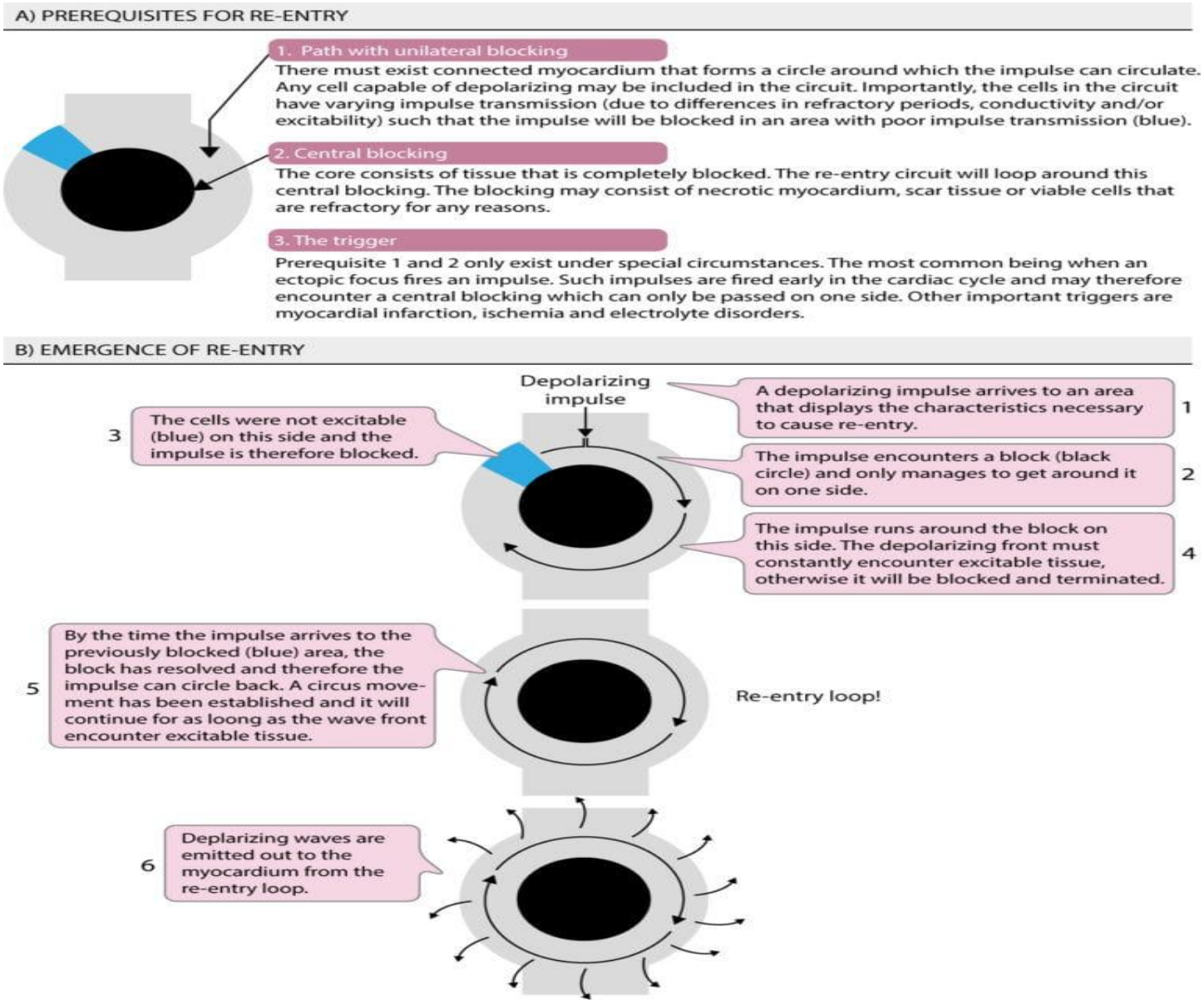



**Figure 14.37 Mechanisms of arrhythmogenesis. (a,b)** Action potentials (i.e. the potential difference between intracellular and extracellular fluid) of ventricular myocardium after stimulation. **(a)** Increased (accelerated) automaticity due to reduced threshold potential or an increased slope of phase 4 depolarization (see p. 680). **(b)** Triggered activity due to early (E) or delayed (D) 'after depolarizations' reaching threshold potential. **(c)** Mechanism of circus movement or re-entry. In panel (1) the impulse passes down both limbs of the potential tachycardia circuit. In panel (2) the impulse is blocked in one pathway (α) but proceeds slowly down pathway β, returning along pathway α until it collides with refractory tissue. In panel (3) the impulse travels so slowly along pathway β that it can return along pathway α and complete the re-entry circuit, producing a circus movement tachycardia.

# MECHANISM OF REENTRY



# Evaluation of tachyarrythmia

**At the beginning of evaluation important points are**

- **Whether patient is haemodynamically stable or not.**
- **Rapid ECG evaluation to decide any emergency intervention like cardioversion or defibrillation.**
- **Detailed evaluation can be done later on when patient is stable**

# HISTORY

- Heart beat regular/irregular
- What is the approximate heart rate
- Continuous /intermittent
- Number and frequency of episodes
- Onset gradual/abrupt
- How an attack terminates

- **Any associated symptoms:**

  **Chest pain , dyspnea ,lightheadedness, polyuria.**

- **Any history of triggers:**

  **Exercise,alcohol,medication,heart failure, infection**

- **Any history of structural heart disease CAD,Vulvular disease.**

- **Family history of arrhythmia or sudden death**

# Physical examination

- **Physical examination often unrevealing when tachycardia is episodic and not ongoing at the time of examination.**

- **Detailed cardiac examination**

| | |
|---|---|
| pulse | Rate,rhythm,volume,character |
| BP | HTN,Hypotension |
| JVP | |
| precordium | Apex location and character, heart sound and murmur |
| | |

**Other relevant systemic examination including thyroid.**

# INVESTIGATION

Resting 12 lead ECG even if patient dosent have tachycardia at the time of ECG. May reveal:

- Evidence of prior MI
- long QT interval
- Ischemia
- Atrial enlargement/hypertrophy
- Ventricular enlargement/hypertrophy
- Evicence of preexcitation

ECG is particularly diagnostic if patient has tachycardia at the time of recording.

- **Other ECG recording**

| HOLTER MONITOR | Several episodes/day |
|---|---|
| EVENT MONITOR | Less frequent episodes |
| IMPLANTABLE LOOP RECORDER | <2 episodes /month |

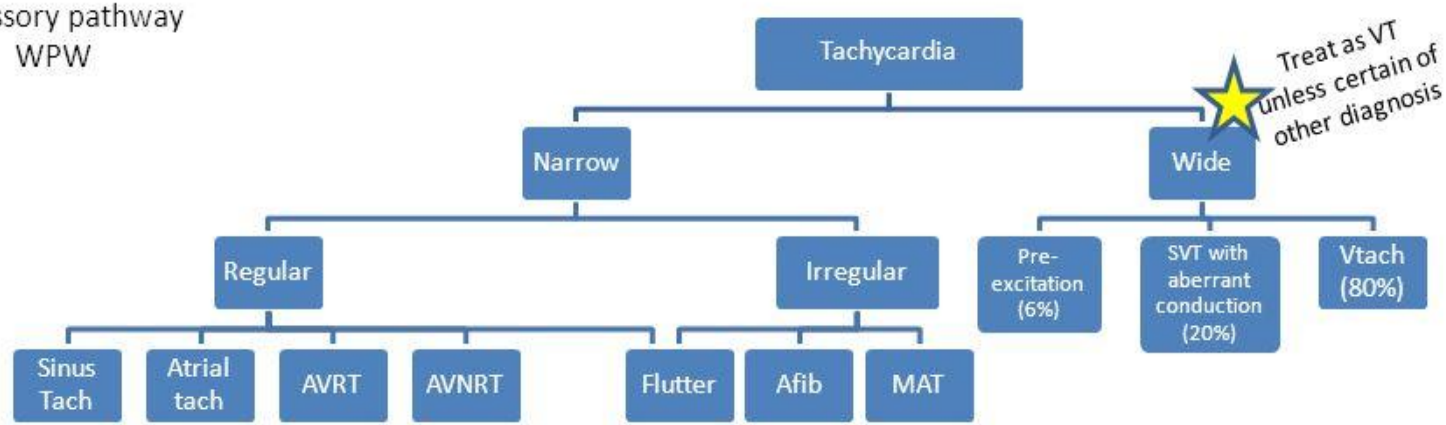
- **Echocardiograpy**
- **Exercise test**
- **Electrophysiology which is sometimes associated with therapeutic RFA.**

# OTHER TESTS

- Serum electrolyte Ca, Mg , K, Na.
- CBC
- Thyroid function test
- Cardiac biomarkers
- Drug level in suspected drug toxicity
- Toxin screen

# Tachycardia

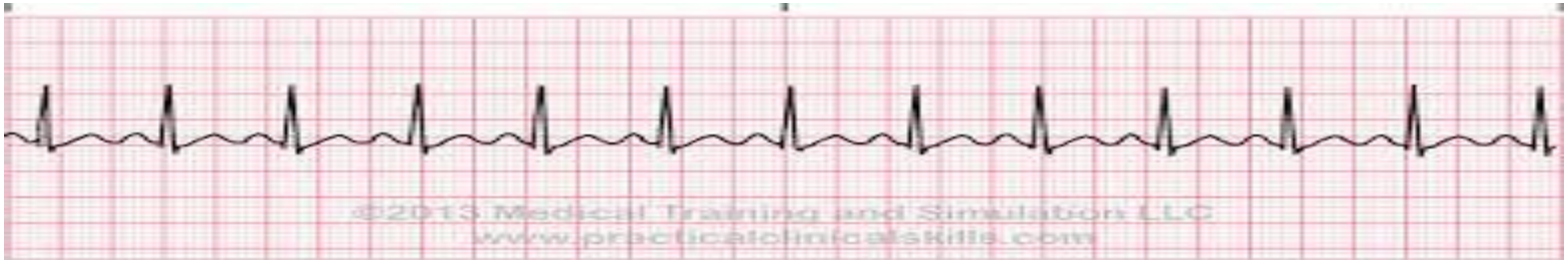
- AVNRT (60% of SVT) – reentry circuit within AV node
- AVRT (30% of SVT) – accessory pathway
  - WPW




- If any of the above are unstable - need synchronized cardioversion
- If stable: vagal maneuver or adenosine (if AV node part of reentry circuit)
  - ***Do NOT use AV nodal blockers if AVRT!!!
- After arrhythmia terminated, then check:
  - EKG: looking for prior MI, LVH, long QT
  - Echo: looking for underlying structural disease
  - Labs: electrolytes, TFTs (K, Ca, Mg – disturbances may precipitate arrhythmias)

# Tachycardia with regular narrow QRS complex

# Sinus tachycardia




## CAUSES OF SINUS TACHYCARDIA

- **Physiological**

  Exertion, anxiety, pain

- **Pathological**

  Fever, anaemia, hypovolaemia, hypoxia, heart failure

- **Endocrine**

  Thyrotoxicosis, pregnancy, pheochromocytoma

- **Pharmacological**

  Adrenaline as a result of phaeochromocytoma; salbutamol; alcohol, caffeine

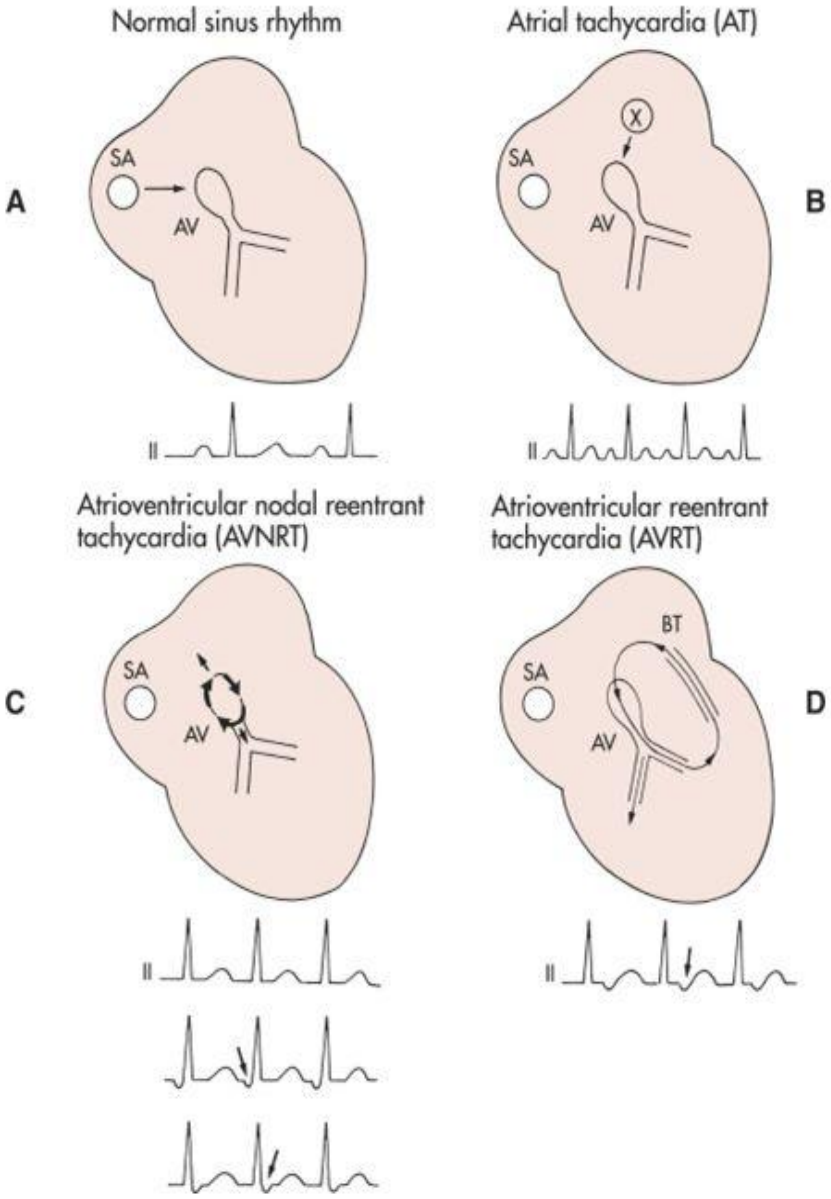
# PSVT

*A Normal*

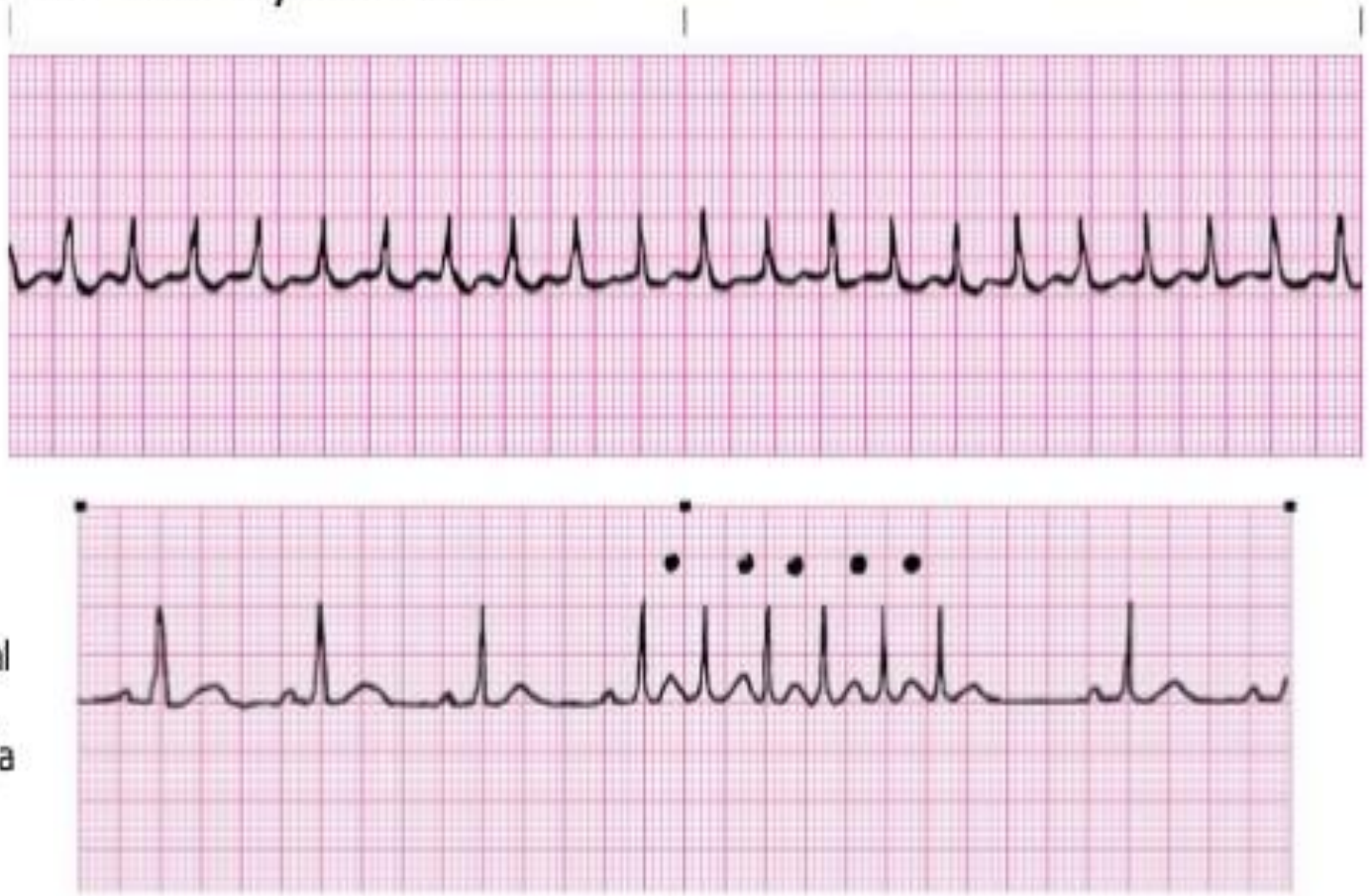
*B Atrial tachycardia* (MAT)

C *atrioventricular nodal reentrant tachycardia* (AVNRT)

D *AV reentrant tachycardia* (AVRT) involving a bypass tract of the type seen in the Wolff-Parkinson-White (WPW) syndrome

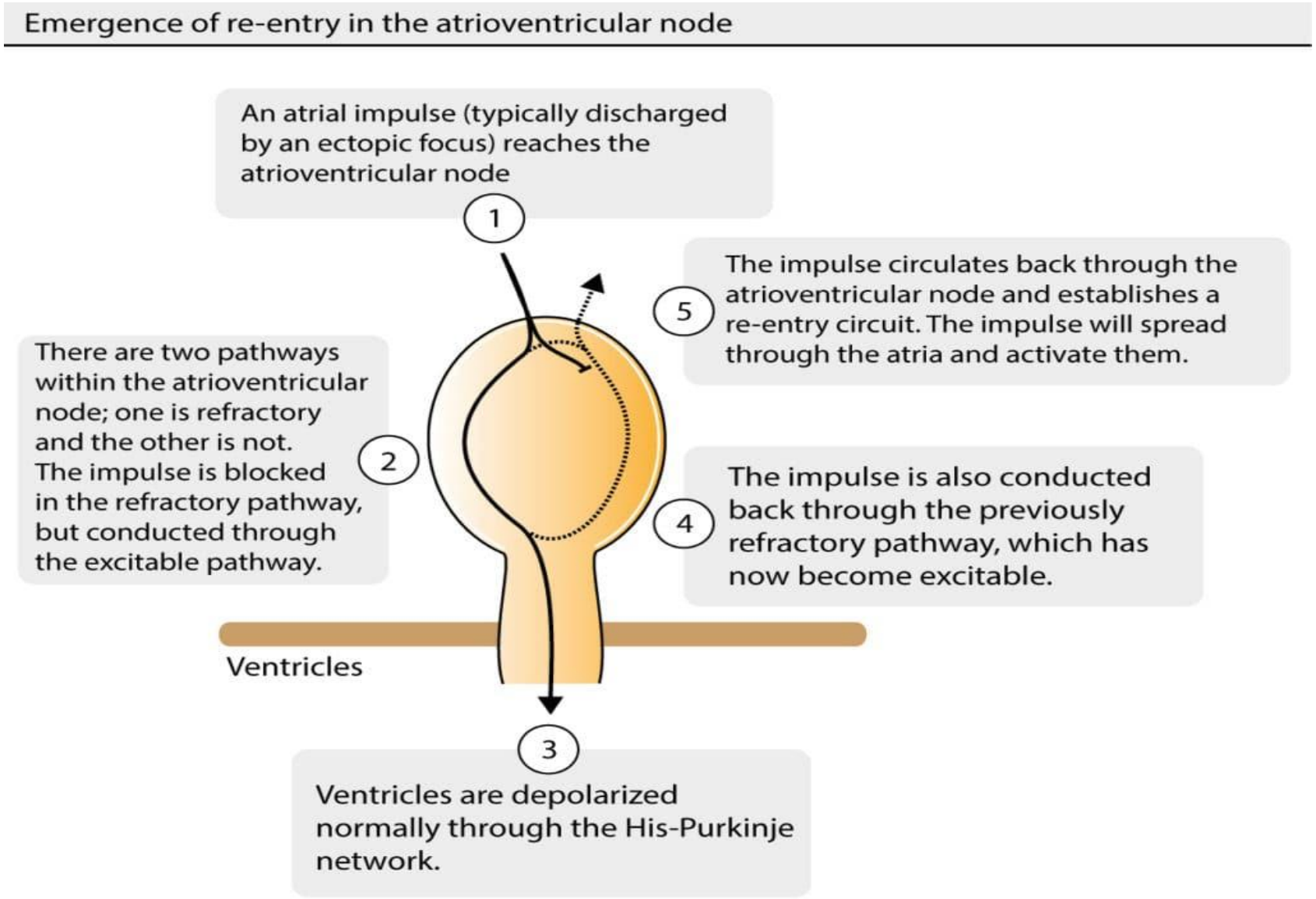


# Atrial Tachycardia

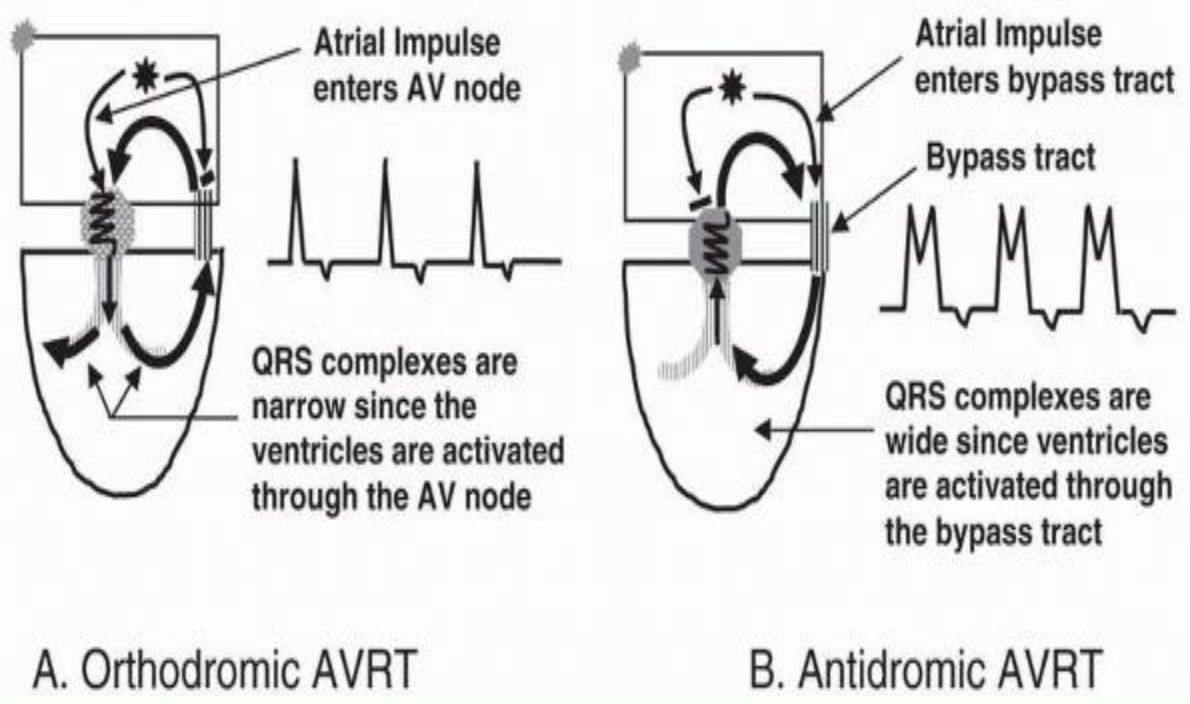


Paroxysmal Atrial Tachycardia

# AVNRT mechanism



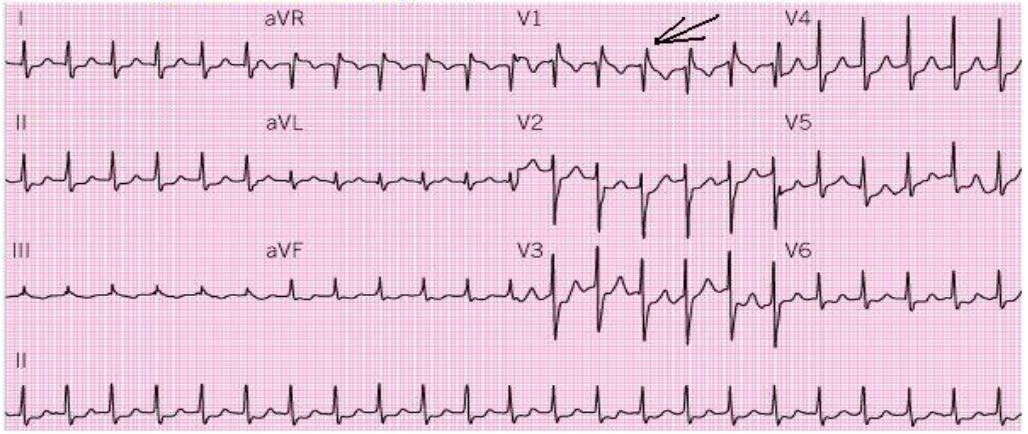
# AVRT mechanism

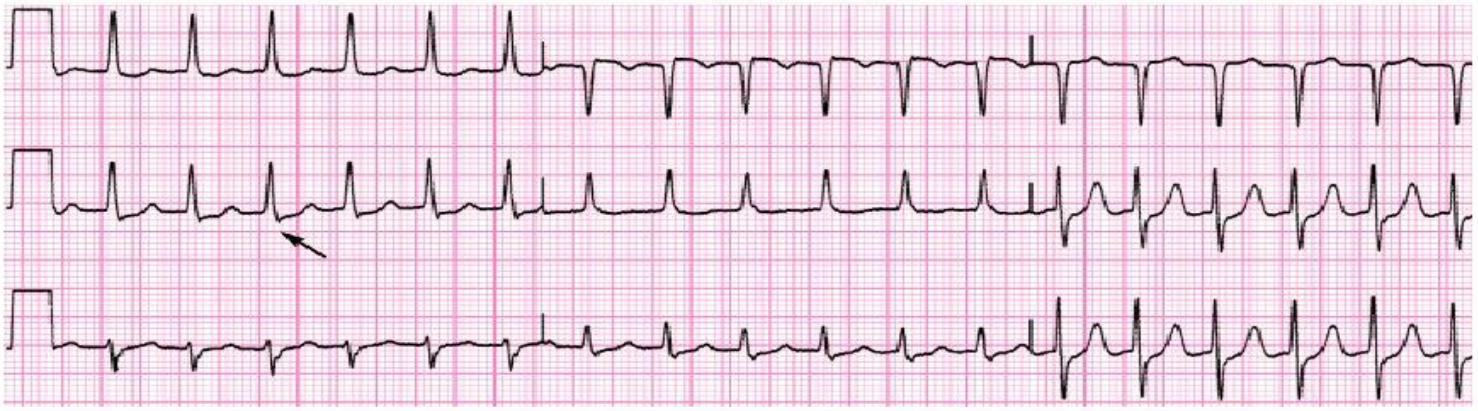


# ECG in AV Nodal Reentrant Tachycardia (AVNRT)

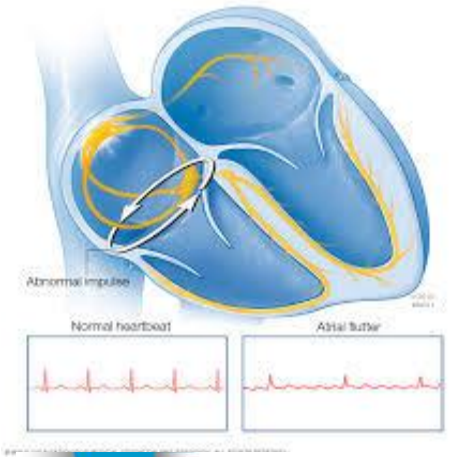
**QRS is**

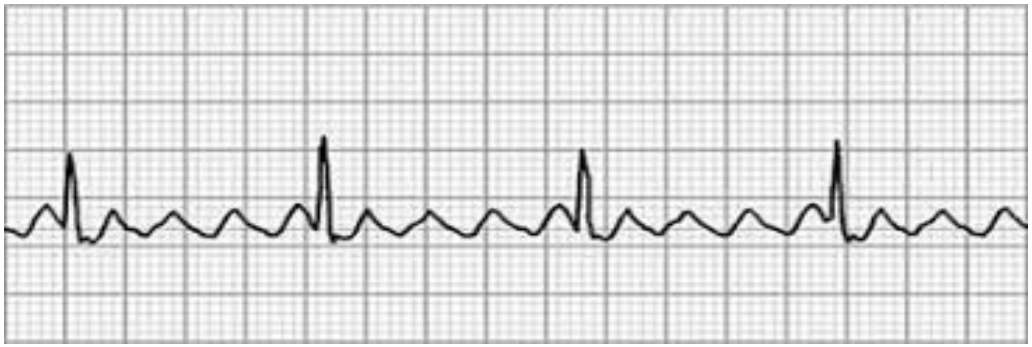
- **Regular (180-200/min)**
- **Narrow (<120ms),**
- **No distinct P wave or retrograde P just after QRS**

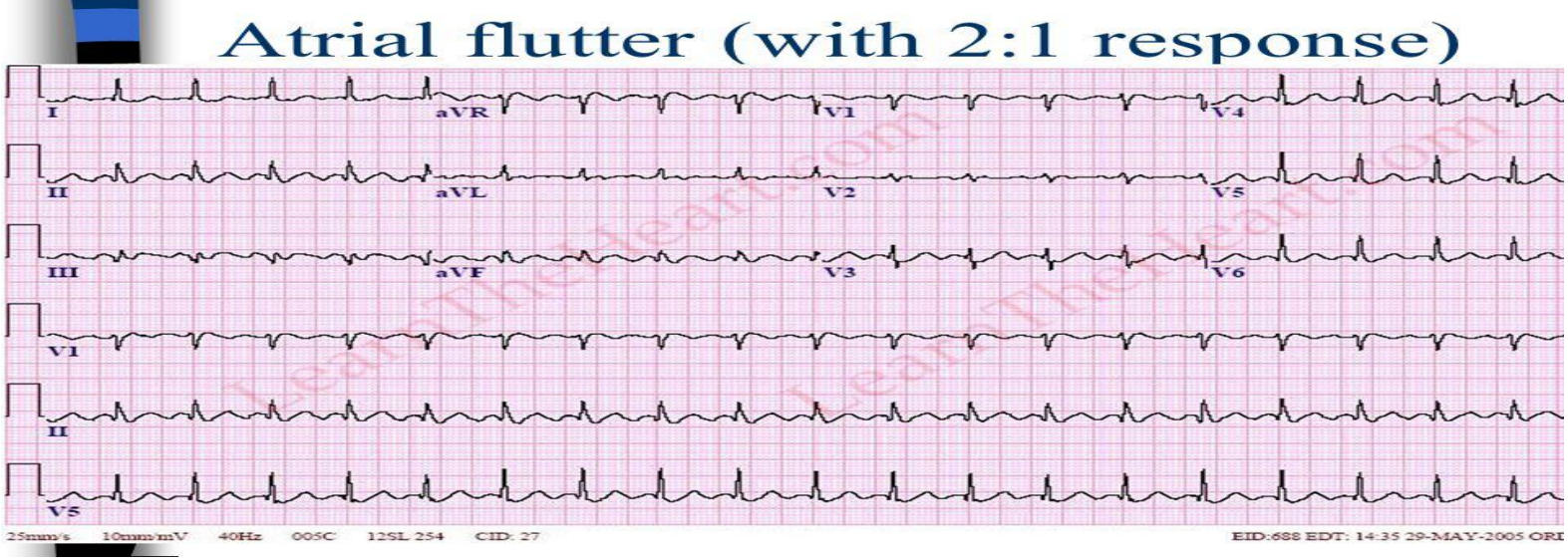


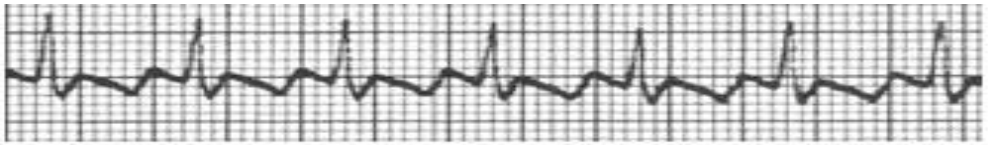
# ATRIAL FLUTTER



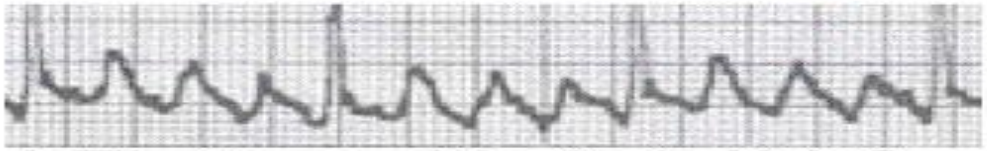





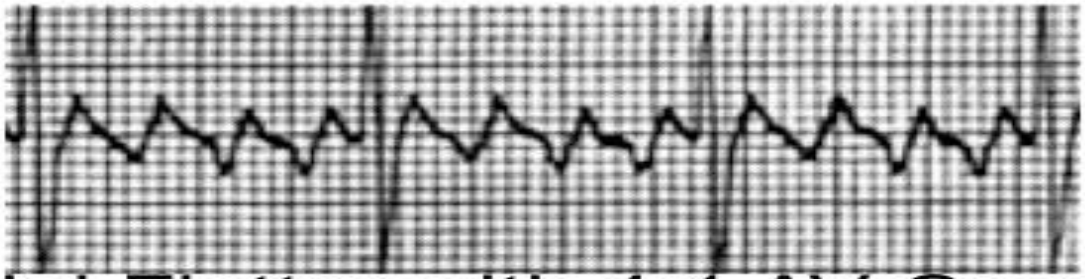
# TYPES OF ATRIAL FLUTTER



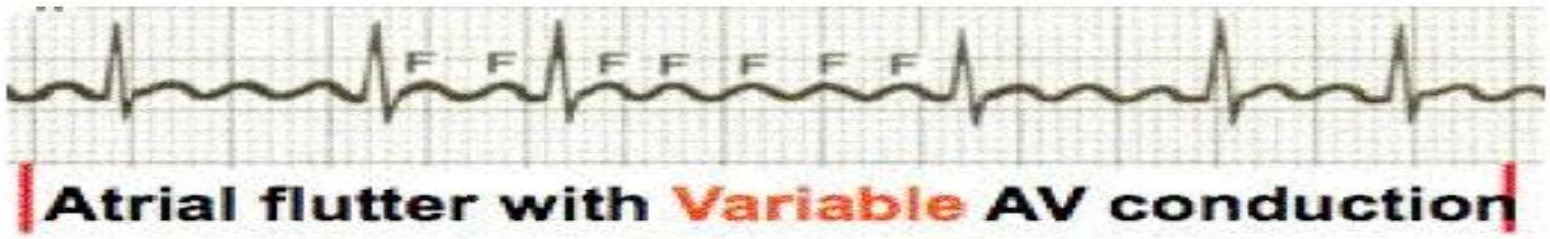
Atrial Flutter with 2:1 AV Conduction



Atrial Flutter with 3:1 AV Conduction

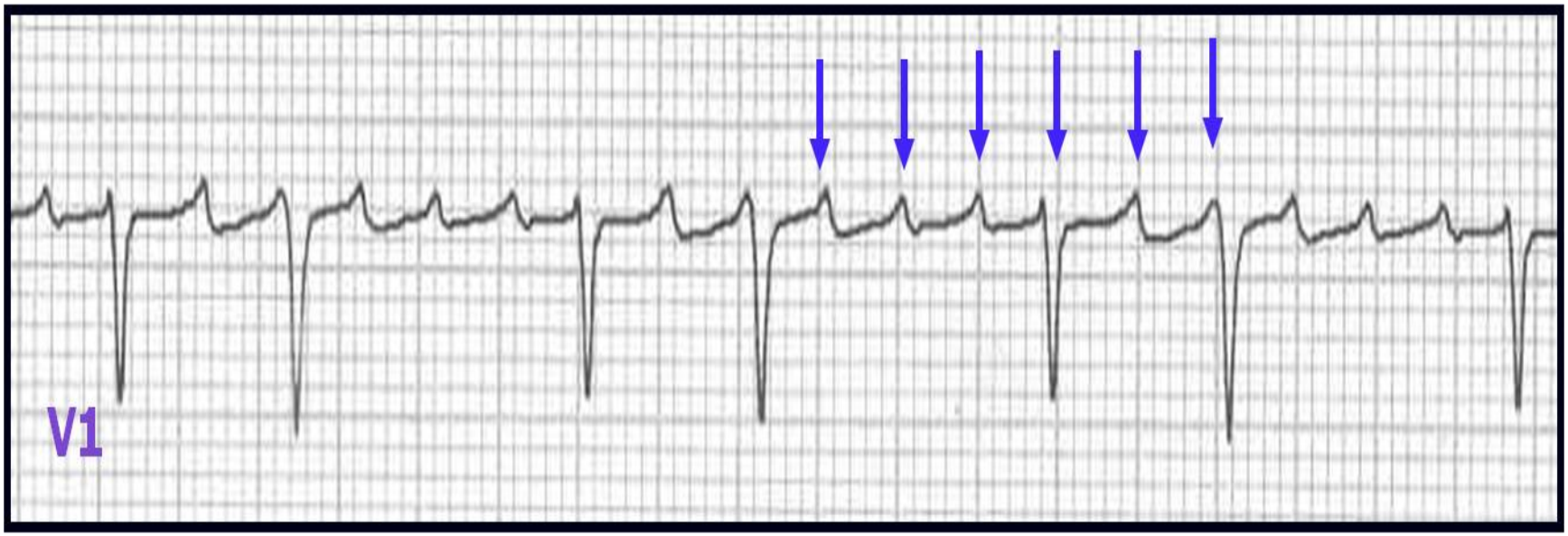


Atrial Flutter with 4:1 AV Conduction

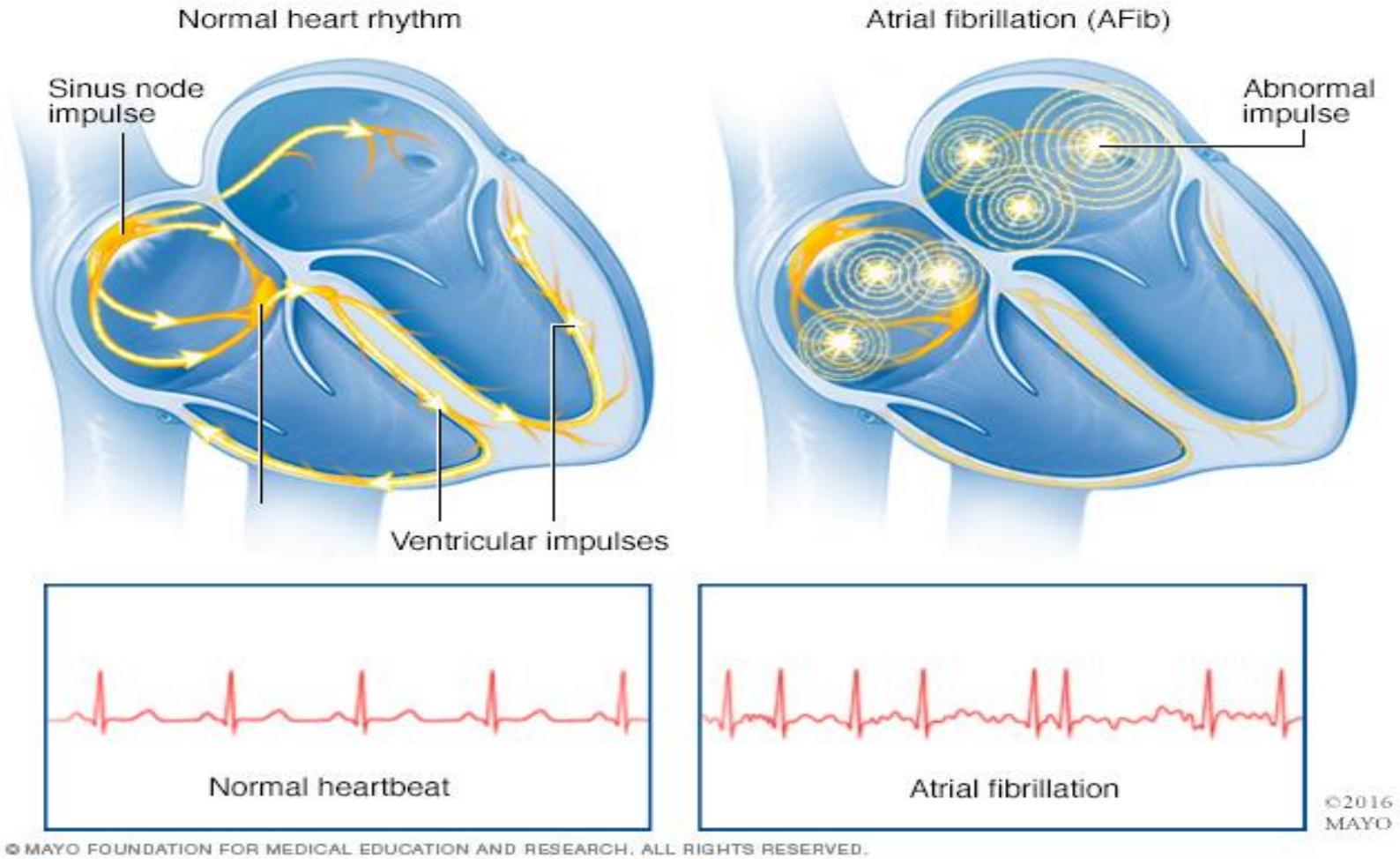


# TACHYARRYTHMIA WITH NARROW IRREGULAR QRS COMPLEX

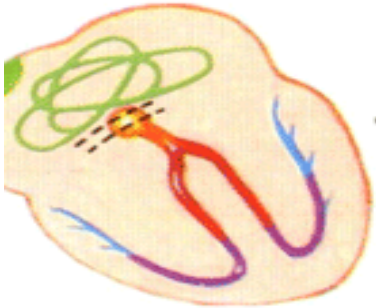
# Atrial flutter with variable block

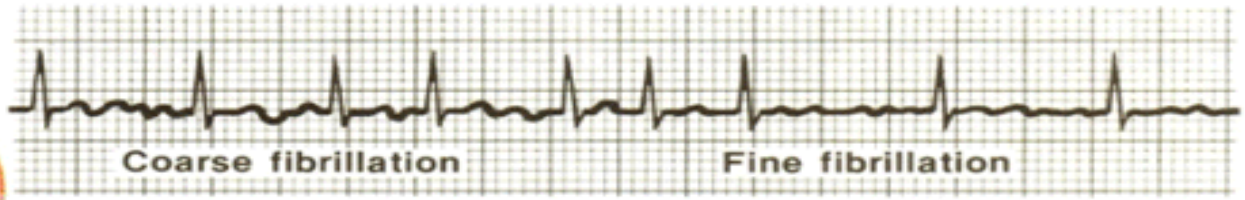


# ATRIAL FIBRILLATION



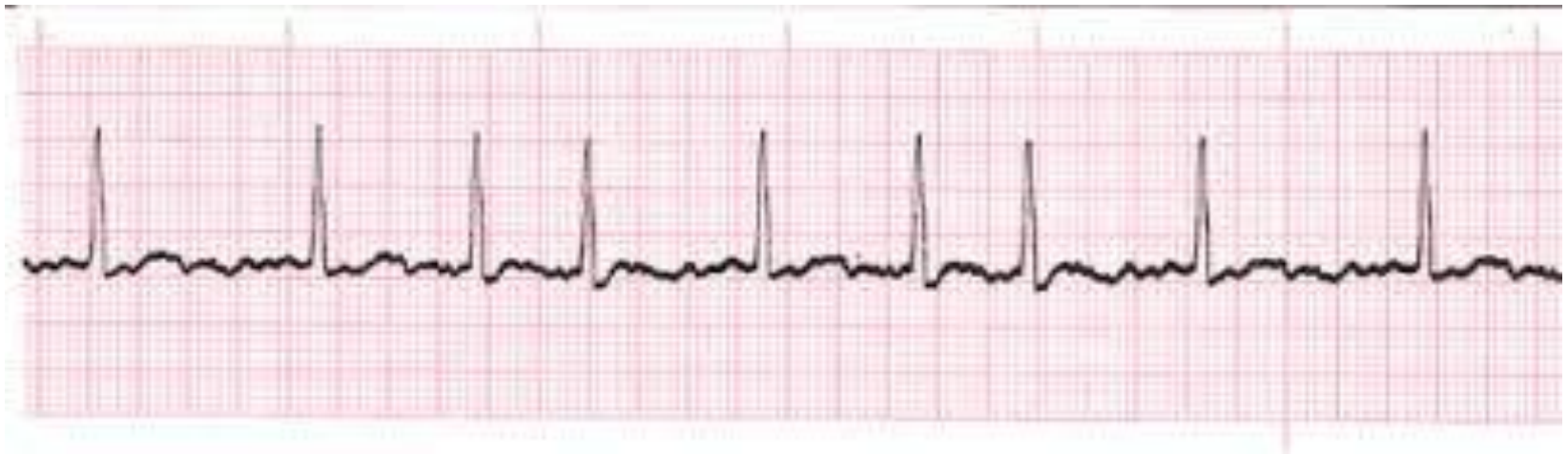
# Atrial fibrillation

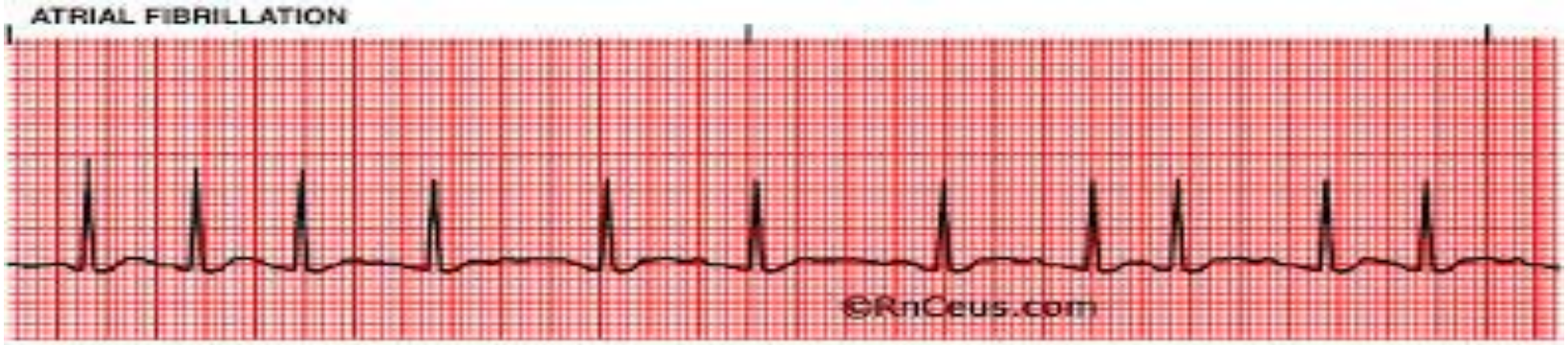





Baseline coarsely or finely irregular; P waves absent.
Ventricular response (QRS) irregular, slow or rapid

- No discernable p-waves
- Multiple foci rapidly discharging
- No organized electrical activity in atria
- Rhythm is irregular
- "Atrial fibrillation Controlled " = rate ≤ 100 bpm
- "Atrial fibrillation Uncontrolled " = rate > 100 bpm




ATRIAL FIBRILLATION
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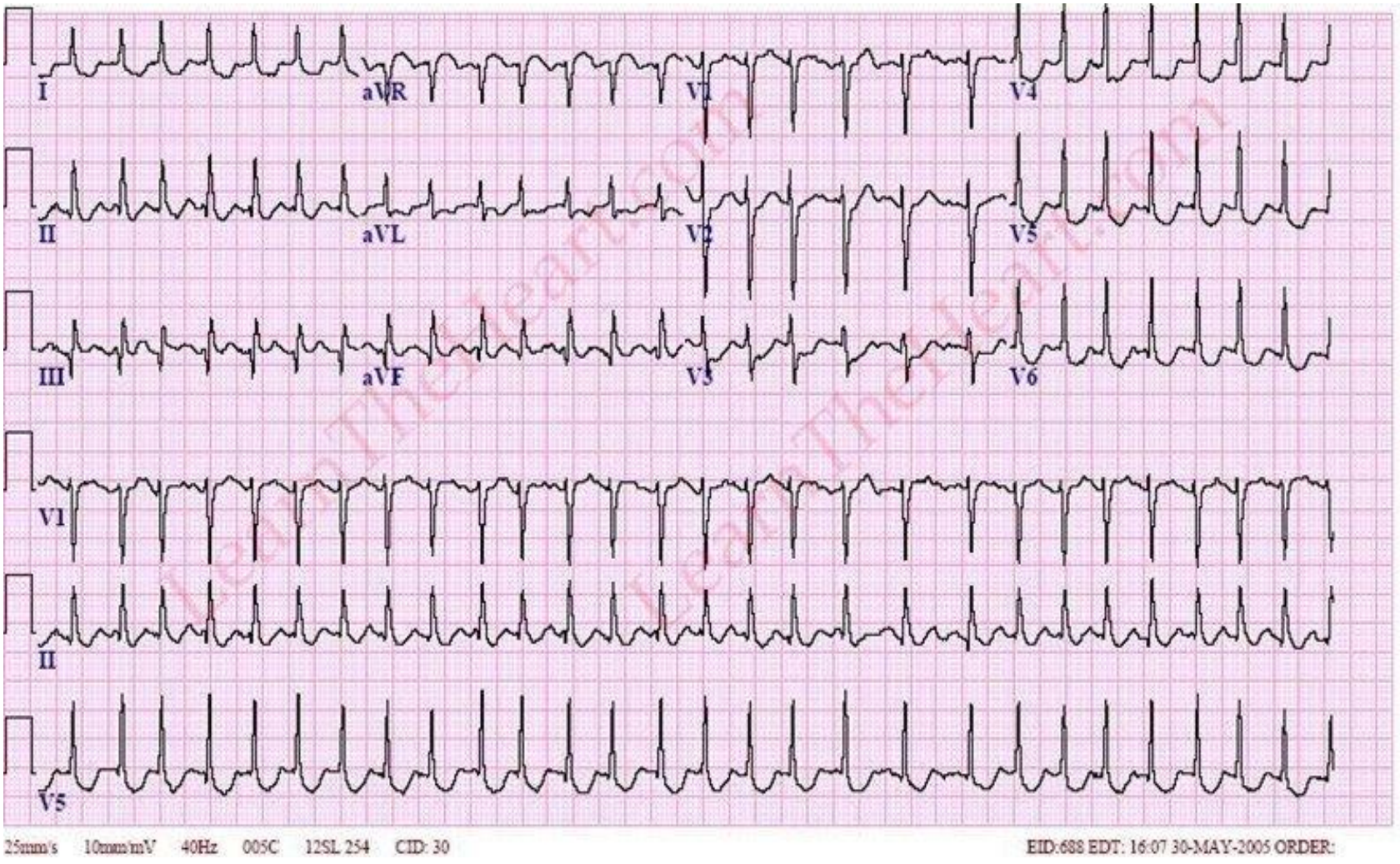
# AF with Digoxin effect



# AF classification

## Paroxysmal

AF that terminates spontaneously or with intervention within 7 days of onset though recurrent paroxysm may occur.

## Persistent

AF that persist for >7 days but < 12 months. sinus rhythm can be restored and maintained.

## Permanent

Sinus rhythm cant be restored or maintained.

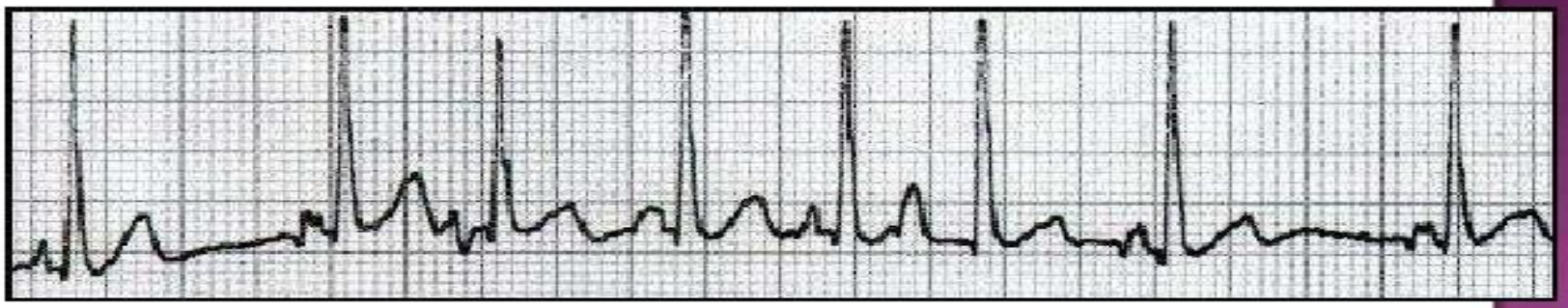
# Causes of atrial fibrillation

Table 1. Etiology Of Atrial Fibrillation.

| Cardiac | Non-cardiac |
| --- | --- |
| • Ischemic heart disease | • Pulmonary embolism |
| • Valvular disease | • Idiopathic |
| • Hypertension | • Medication noncompliance |
| • Congestive heart failure | • Thyroid disease |
| • Sick sinus syndrome | • Holiday heart syndrome |
| • Pericarditis | • Medication use |
| • Infiltrative heart disease | • Electrocution |
| • Cardiomyopathy | • Other pulmonary disease |
| • Cardiac surgery | • Chest trauma |
| • Myocarditis | • Hypokalemia |
| • Congenital heart disease | • Hypomagnesemia |
| | • Hypothermia |

# Multifocal atrial tachycardia

## MULTIFOCAL ATRIAL TACHYCARDIA

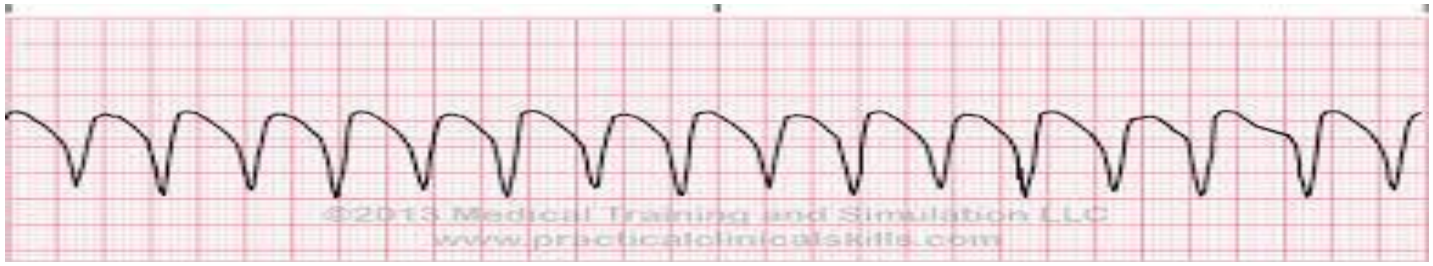


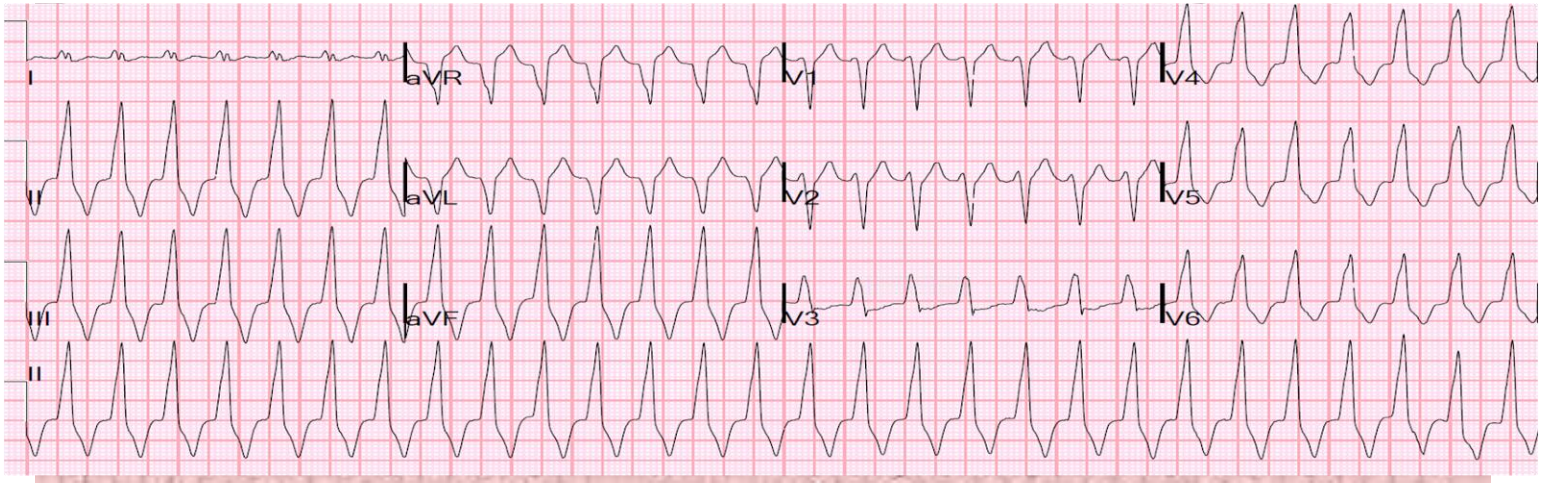
ECG Characteristics: Discrete P waves with at least 3different morphologies.
Absence of one dominant atrial pacemaker
Atrial rate > 100 bpm.

The PP, PR, and RR intervals all vary.

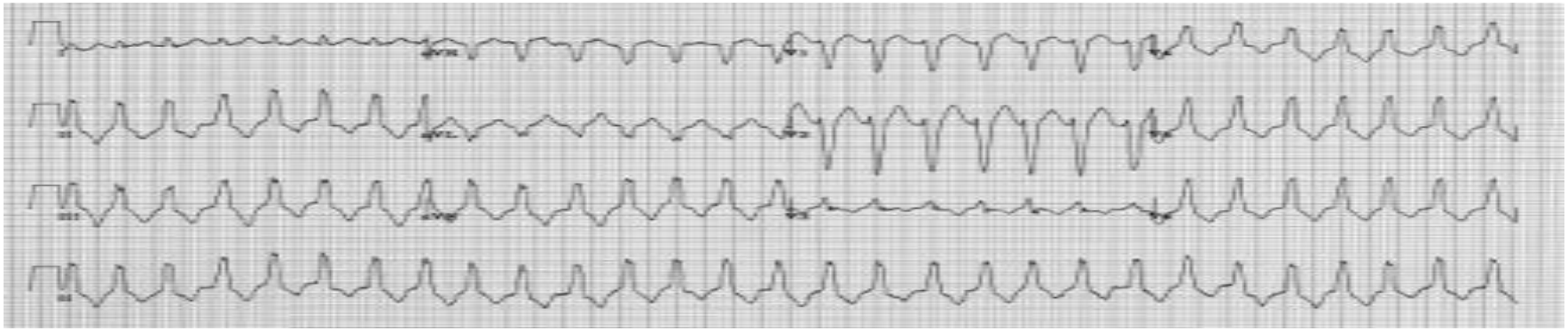
# Tachyarrythmia with regular wide QRS complex

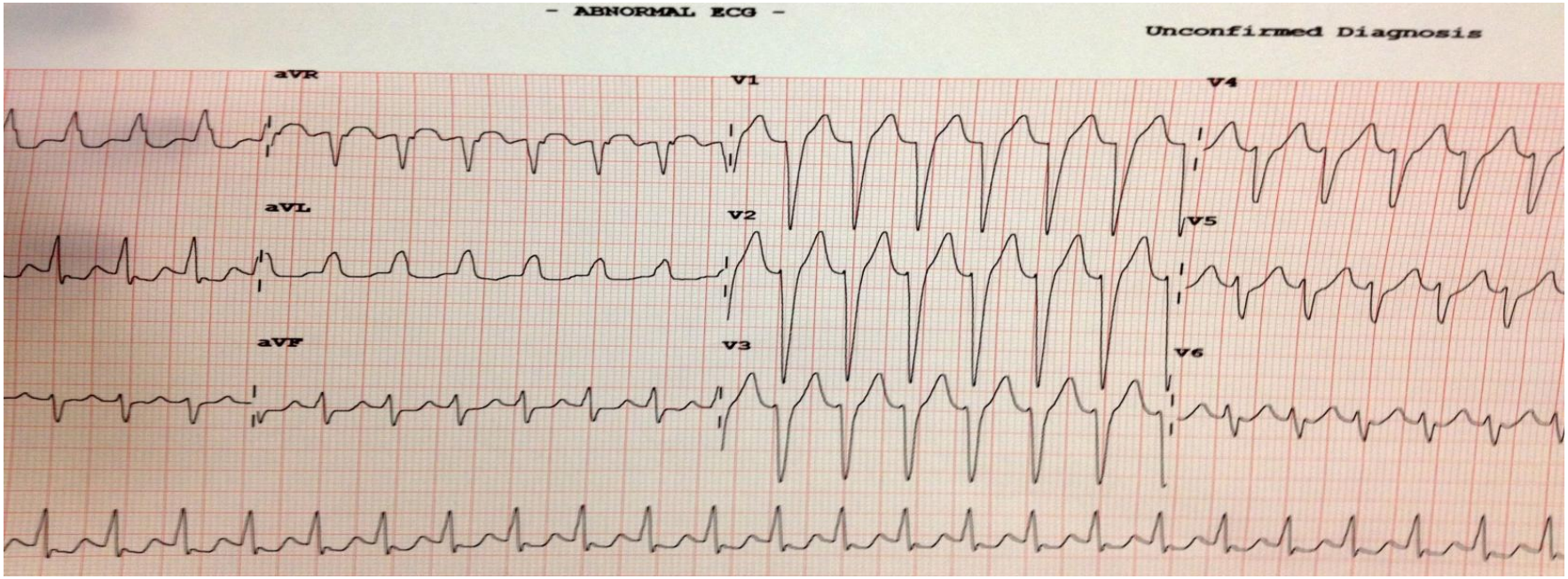
# Ventricular tachycardia



# SVT with aberrant conduction



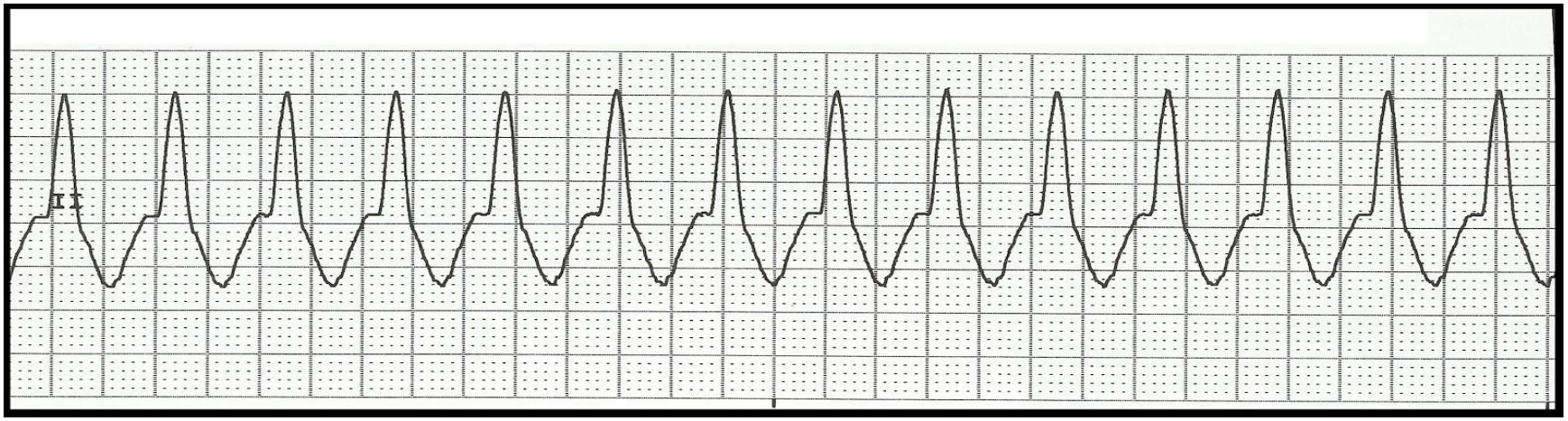


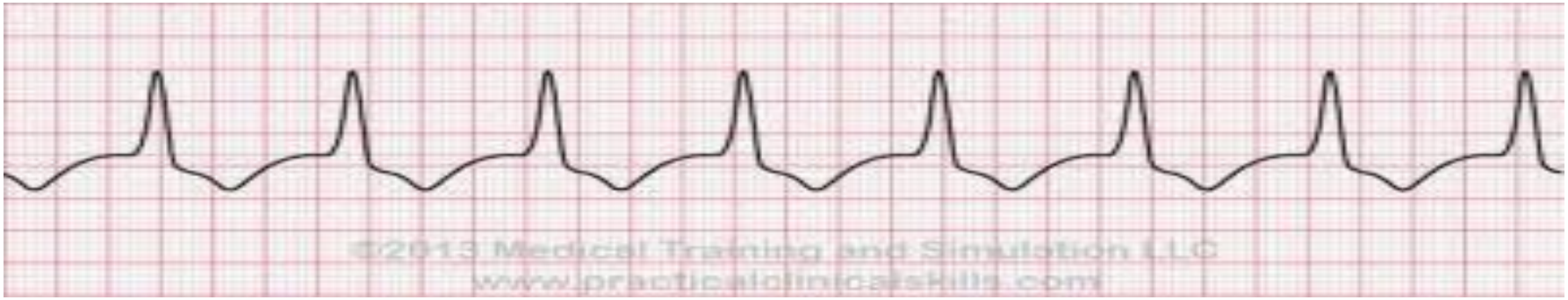
## Table 14.11 ECG distinction between supraventricular tachycardia (SVT) with bundle branch block and ventricular tachycardia (VT)

VT is more likely than SVT with bundle branch block where there is:

- a very broad QRS (>0.14 s)
- atrioventricular dissociation
- a bifid, upright QRS with a taller first peak in $V_1$
- a deep S wave in $V_6$
- a concordant (same polarity) QRS direction in all chest leads ($V_1$–$V_6$)

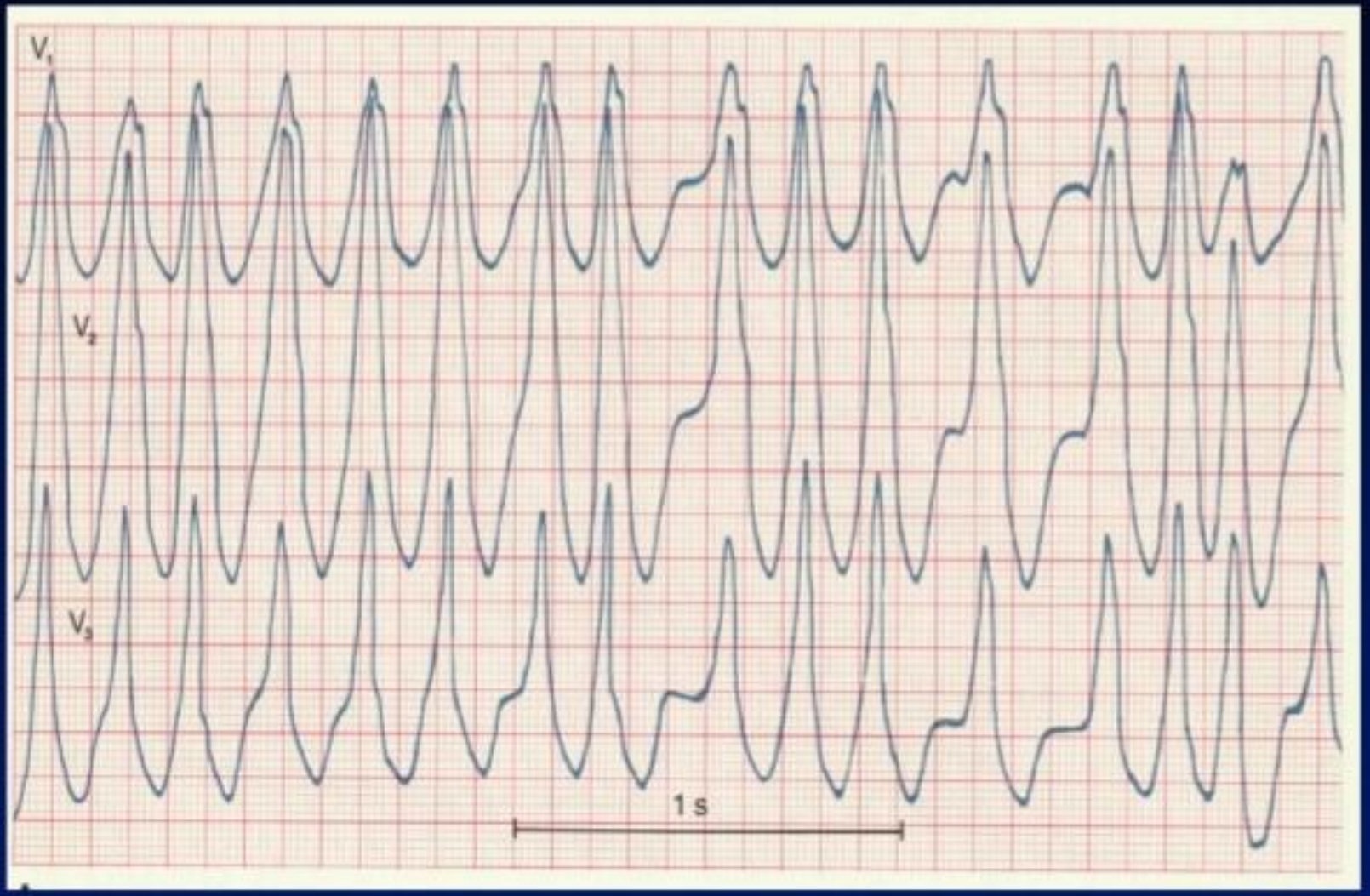
# ACCELERATED IDIOVENTRICULAR RYTHM



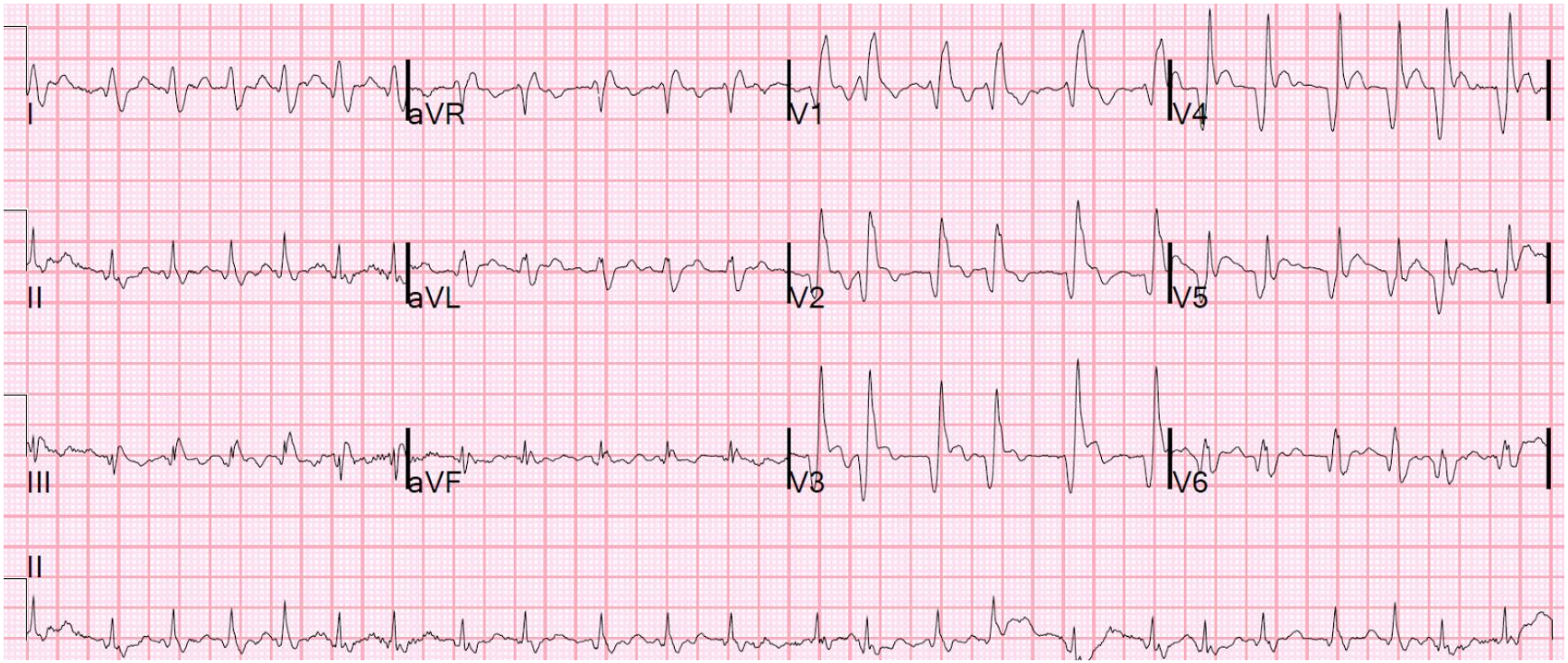
# Tachyarrythmia with irregular wide QRS complex

# *WPW with Atrial Fibrillation*




*Y.B. Liu*

# ATRIAL FIBRILLATION WITH RBBB



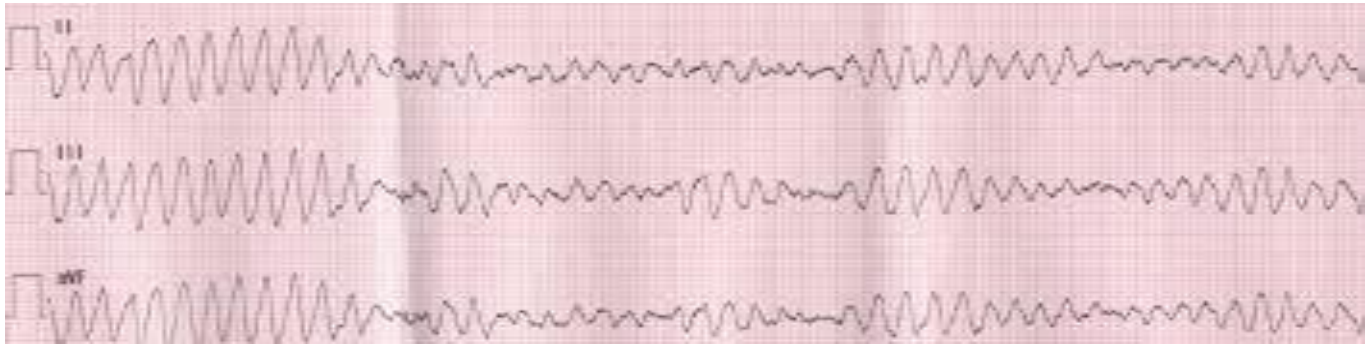
# Irregular rhythm with variable QRS duration

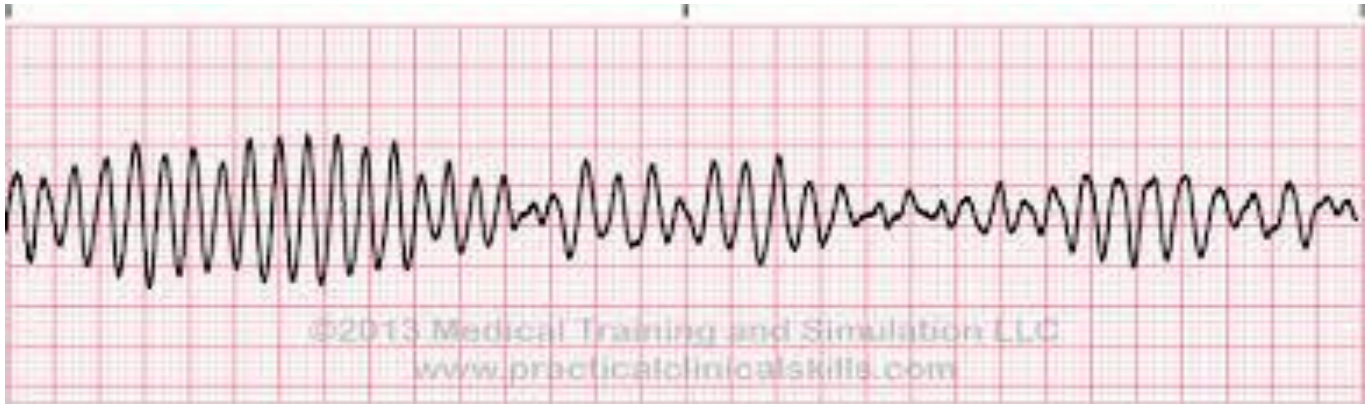
# POLYMORPHIC VT

## Two types

- **Polymorphic VT with normal QT interval eg; post MI.**
- **Polymorphic VT with long QT either congenital or acquired called “torsades de pointes”.**

# Torsades de pointes





# Polymorphic VT - Torsades de pointes

- Torsades de pointes is a polymorphic VT following a long resting long QT interval (>0.44 seconds). The ECG waves are rapid, and irregular (i.e. polymorphic) and continuously change from an upright to inverted position.

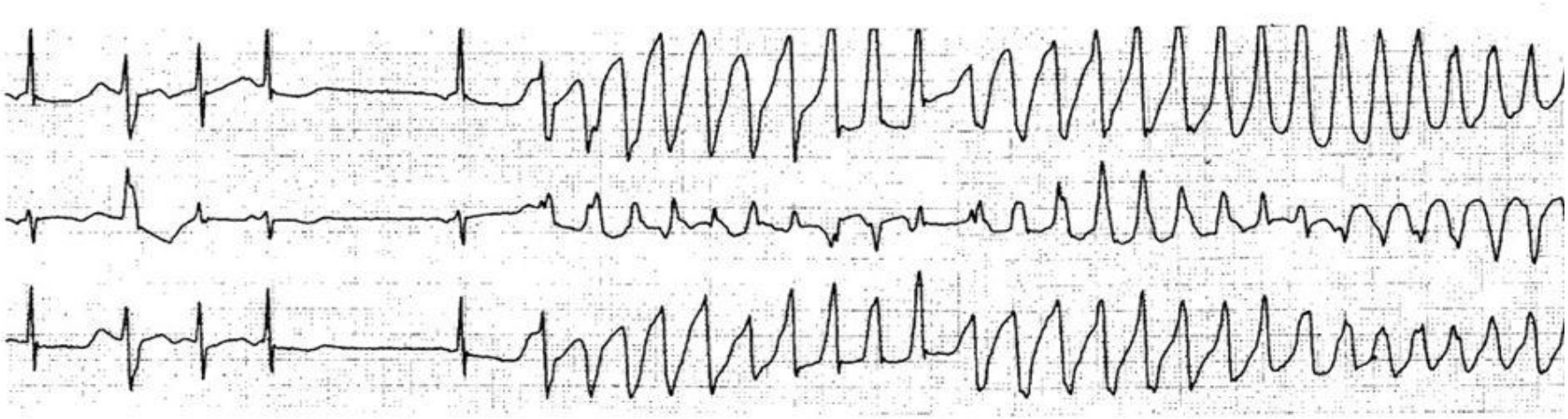
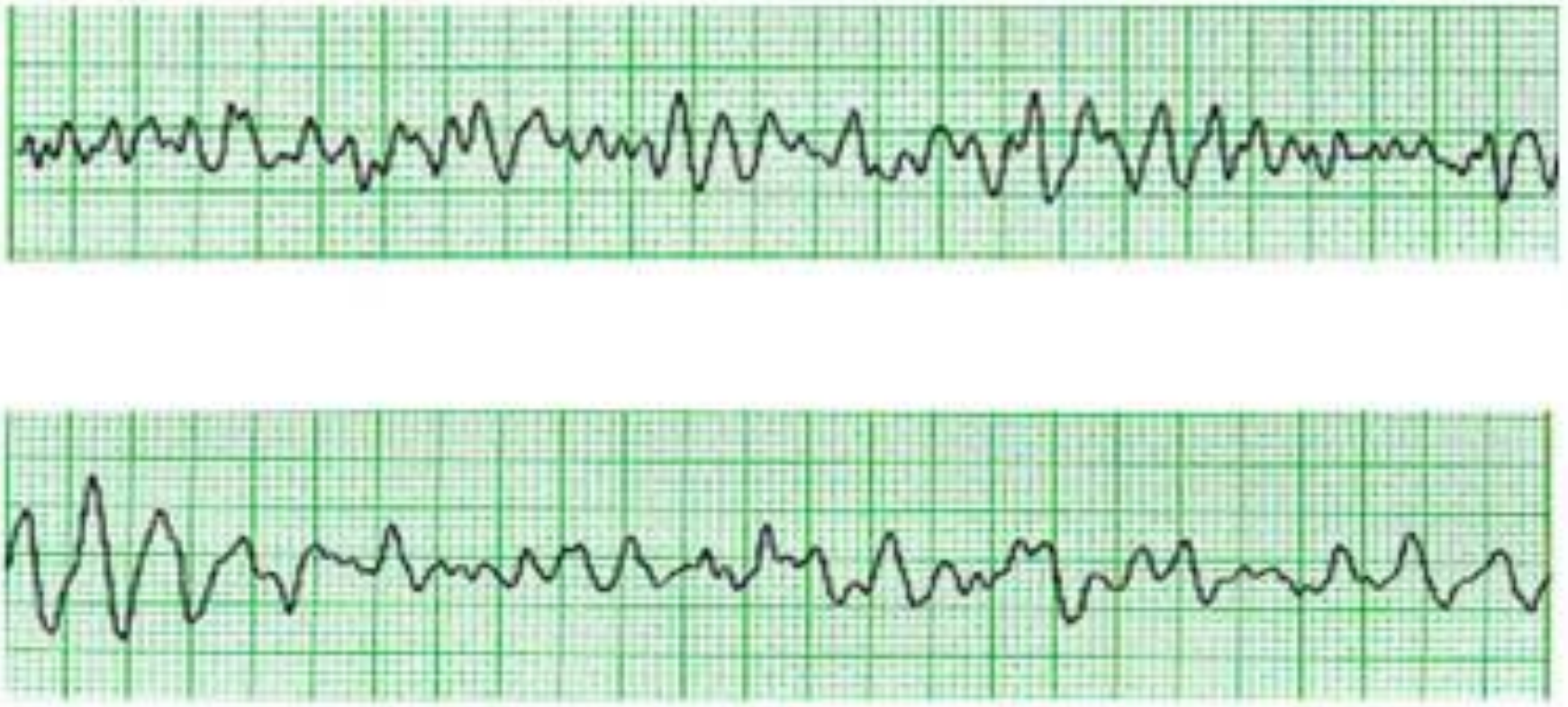


**Table 14.12 Causes of long QT syndrome**

| Congenital | Acquired |
|---|---|
| Jervell–Lange–Nielsen (autosomal recessive)<br>Romano–Ward (autosomal dominant) | **Electrolyte abnormalities**<br>Hypokalaemia<br>Hypomagnesaemia<br>Hypocalcaemia<br>**Drugs**<br>Quinidine, disopyramide<br>Sotalol, amiodarone<br>Tricyclic antidepressants, e.g. amitriptyline<br>Phenothiazine drugs, e.g. chlorpromazine<br>Antipsychotics, e.g. haloperidol, olanzapine<br>Macrolides, e.g. erythromycin<br>Quinolones, e.g. ciprofloxacin<br>Methadone<br>**Poisons**<br>Organophosphate insecticides<br>**Miscellaneous**<br>Bradycardia<br>Mitral valve prolapse<br>Acute myocardial infarction<br>Diabetes<br>Prolonged fasting and liquid protein diets (long-term)<br>Central nervous system diseases, e.g. dystrophia myotonica |

# Ventricular Fibrillation



# Management of SVT

## If patient is haemodynmically stable:

- Vagal menoeuvre
- IV adenosine (6 mg IV push followed by 12 mg if needed.)
- Alternative IV verapamil ( 5-10n mg IV over 5-10 min)

## If patient is haemodynamically unstable – Emergency cardioversion

## Long term management

- EPS and RFA of reentry circuit.
- Drugs

  **Amiodarone, flecainide,propafenone,verapamil diltiazem,beta blocker.**

## Valsalva manoeuvre

- Valsalva manoeuvre is an abrupt voluntary increase in intrathoracic and intra-abdominal pressures by straining.
- Provide continuous electrocardiographic monitoring.
- Patient is in supine position.
- Patient should not take deep inspiration before straining.
- Ideally, the patient blows into the mouthpiece of a manometer against a pressure of 30–40 mmHg for 15 s.
- Alternatively, the patient strains for 15 s while breath-holding.
- Transient acceleration of tachycardia usually occurs during the strain phase as a result of sympathetic excess.
- On release of strain, the rate of tachycardia slows because of the compensatory increase in vagal tone (baroreceptor reflex), and it may be terminated in about 50% of patients.
- Termination of tachycardia may be followed by pauses and transient ventricular ectopics.

## Carotid sinus massage

- Ensure there is no significant carotid artery disease (carotid bruits).
- Provide continuous electrocardiographic monitoring.
- Patient is in supine position with the head slightly extended.
- Start with right carotid sinus massage.
- Apply firm rotary pressure to the carotid artery at the level of the third cervical vertebra for 5 s.
- Alternatively, steady pressure can be applied.
- If no response, massage left carotid sinus.
- Generally, right carotid sinus massage decreases the sinus node discharge, and left carotid sinus massage slows atrioventricular conduction.
- Do not massage both carotid sinuses at the same time.
- Single application of carotid sinus pressure may be effective in about 20–30% of patients with paroxysmal supraventricular tachycardias; multiple applications can terminate tachycardia in about 50% of patients.
- Asystole is a potential but rare complication.

# Management of atrial fibrillation

**If AF occurs as a complication of acute illness like pneumonia or pulmonary embolism treatment of primary disorder will restore sinus rhythm.**

## Paroxysmal AF

**Aim is to prevent attack**

### Drugs

**Beta blocker , flecainide or propafenone if no CAD or LV dysfunction, amiodarone if CAD or LV dysfunction present.**

### RFA

**Isolation of pulmonary veins from left atrium**

# Persistent and permanent AF

- Rhythm control if possible.
- Rate control accepting that restoration to sinus rhythm is not possible.
- Anticoagulation by assessing risk of thromboembolism.

**Restoration to sinus rhythm is possible**

**< 3 Months duration**

**Patient is young**

**No structural heart disease**

**Cardioversion**

**If AF duration is of <48 hours**

**Immediate cardioversion after IV heparin by IV flecainide in patient with no structural heart disease or IV amiodarone in patient with structural heat disease or by DC cardioversion if pharmacological cardioversion failed.**

**If AF duration >48 hours**

**At first patient must be anticoagulated by warfarin for at least for 4 weeks before cardioversion and continue anticoagulation for further 3 months.**

**In case of urgent cardioversion TOE should be done to exclude LA thrombus.**

## Rate control

If sinus rhythm cant be restored, rate control is the goal by drugs or by pace and ablation therapy.

## Anticoagulation

With oral anticoagulants by risk stratification with $CHA_2DS_2VASc$ scoring. If score $\geq 2$, oral anticoagulants like warfarin,dabigatran or rivaroxaban is used. If score $\leq 1$ aspirin can be used. if score is O ,no need of anticoagulation.

# Atrial flutter

- Acute paroxysm if haemodynamically unstable by electrical cardioversion
- Pharmacological cardioversion by amiodarone or flecainide if duration <48 hours.
- If >48 hours treatment strategy is similar to AF.
- Recurrent paroxysm may be prevented by drugs like amiodarone or by RFA of reentry circuit.

# Ventricular tachycardia

- Prompt restoration to sinus rhythm is the goal
- Synchronized DC cardioversion if systolic BP is <90 mm of Hg.
- If well tolerated and haemodynamically stable , IV amiodarone or IV lignocaine.

**Prevention of attack**

Drugs- amiodarone,beta blocker
ICD if poor LV function
RFA of scar tissue.

# Torsades de pointes

- Treatment of underlying cause
- IV magnesium
- Atrial pacing
- IV isoprenaline
- Beta blocker in congenital long QT
- ICD

# VENTRICULAR FIBRILLATION

**Immediate defibrillation**

**In survivors**

**Prophylactic ICD Implantation.**

# Simplified management options

| Cardiac arrest due to VF or pulseless VT | CPR and urgent Defibrillation |
|---|---|
| AF, Atrial tachycardia, SVT or any tachycardia causing haemodynamic instability | Urgent cardioversion |
| Regular narrow complex tachycardia with haemodynamic stability | Vagal menoeuvre or IV Adenosine<br>May terminate AVNRT or unmask Atrial tachycardia |
| New onset AF/A flutter<48 hours<br>Persistent /permanent AF or flutter<br>AVNRT,AVRT | Electric or chemical cardiovesion<br>Rate control,anticoagulation,catheter ablation<br>Antiarrythmic, catheter ablation |
| SVT with aberrancy | If confusion treat as VT<br>Adenosinre,verapamil/electrical cardioversion |
| Atrial fibrillation with aberrancy | Immediate chemical or electrical cardioversion as chance of degeneration into VF |
| Polymorphic VT (torsades de pointes) | Mg sulphate, electrical cardioversionoverdrivre pacing.<br>Prevent and correct precipitating cause. |
| | |

# Antiarrythmic drugs

| Table 14.15 | Vaughan Williams' classification of antiarrhythmic drugs |
|---|---|
| Class I | Membrane-depressant drugs (sodium-channel blockers) |
| Ia | Disopyramide |
| Ib | Lidocaine, mexiletine |
| Ic | Flecainide, propafenone |
| Class II | β-Adrenoceptor blocking drugs, e.g. atenolol, propranolol, esmolol |
| Class III | Prolong action potential, e.g. amiodarone, dronedarone, sotalol |
| Class IV | Calcium-channel blockers, e.g. verapamil, diltiazem |
| (Other | Adenosine, digoxin) |

# Catheter ablation can be done in following tachyarrythmia:

AV nodal re-entry tachycardia (AVNRT)

AV re-entry tachycardia (AVRT) with an accessory pathway including WPW syndrome

Normal heart VT

Atrial flutter

Atrial tachycardia

Atrial fibrillation

# ICD

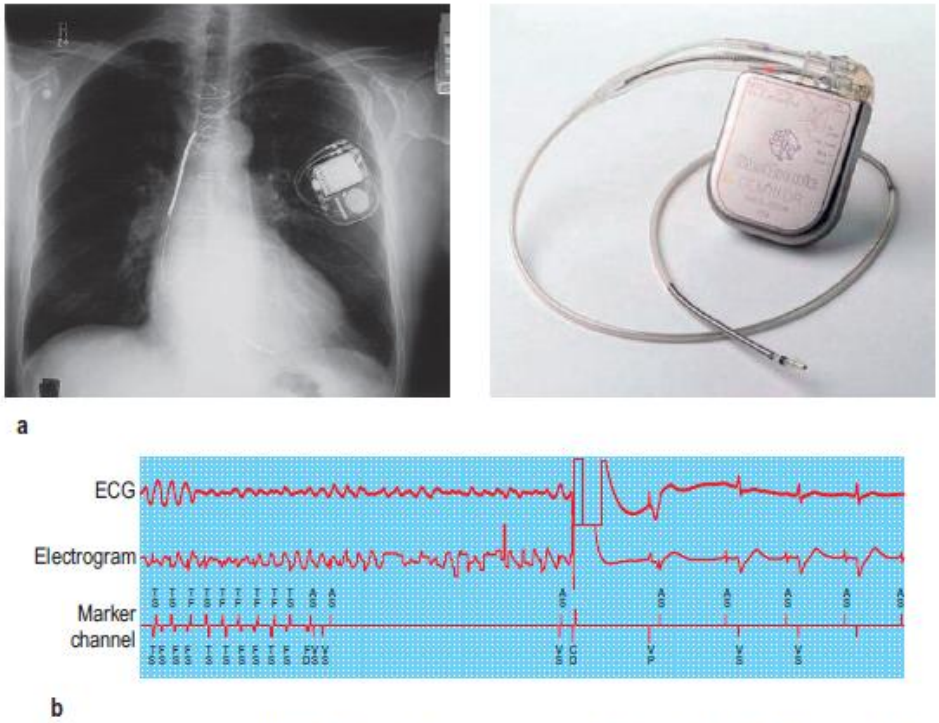
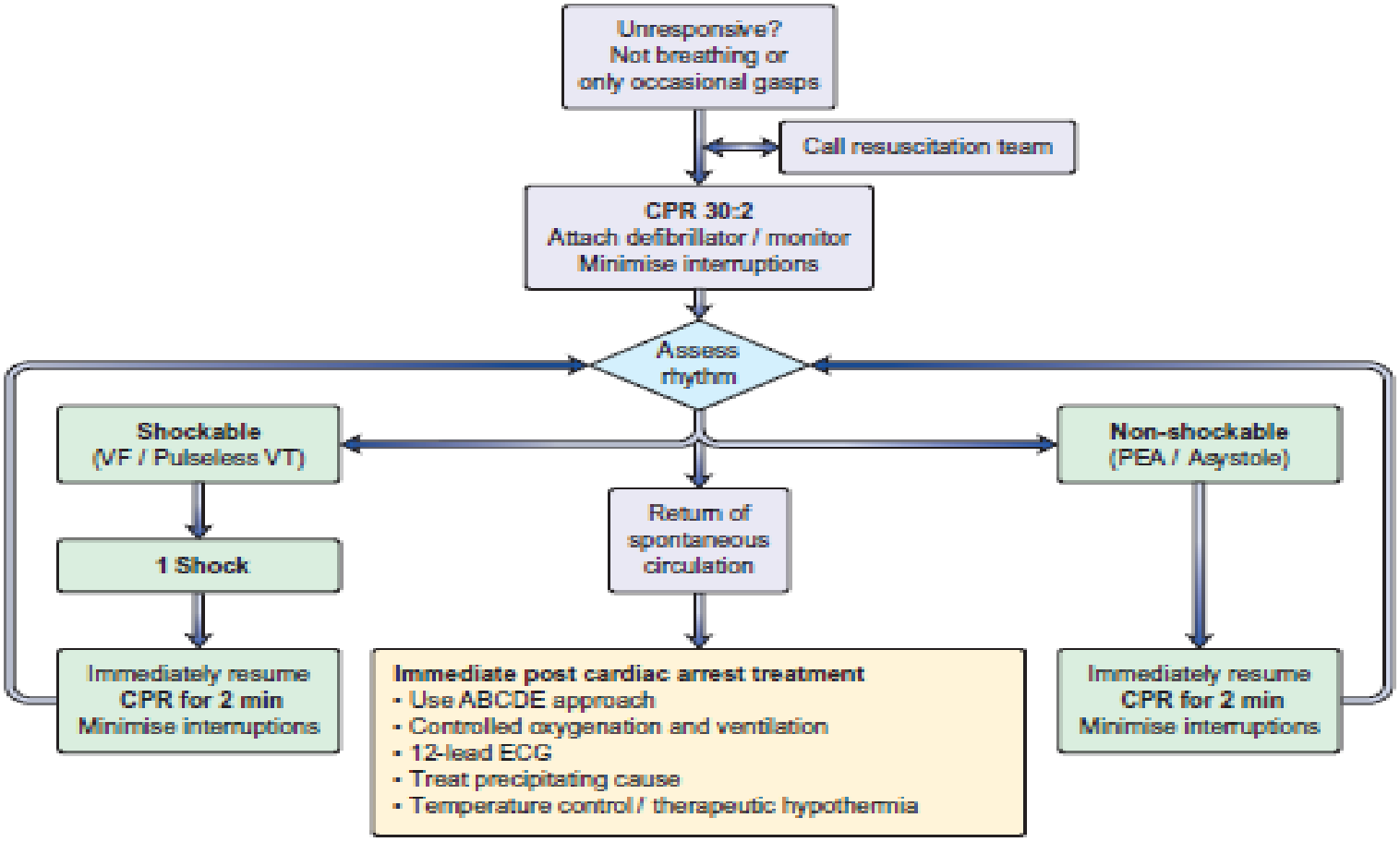



**Figure 14.50 Implantable cardioverter-defibrillator. (a)** X-ray of a 'dual-chamber' defibrillator in a left pectoral position with atrial and ventricular leads. **(b)** ECG showing the termination of ventricular fibrillation by the direct current shock at 20 J. Electrogram (EG) recorded internally from the ventricular lead of an ICD reveals chaotic ventricular activity consistent with ventricular fibrillation. This is confirmed by an electrocardiogram (lead $V_2$). The marker channel (MC) demonstrates that the device detects ventricular fibrillation correctly (FS, fibrillation sensing, lower line) and delivers an appropriate shock (CD) that terminates the arrhythmia and restores normal sinus rhythm (VS, ventricular sensing, lower line). Incidentally, atrial fibrillation is also detected before shock delivery (upper line on the marker channel).

# Adult Advanced Life Support




**During CPR**
- Ensure high-quality CPR: rate, depth, recoil
- Plan actions before interrupting CPR
- Give oxygen
- Consider advanced airway and capnography
- Continuous chest compressions when advanced airway in place
- Vascular access (intravenous, intraosseous)
- Give adrenaline every 3-5 min
- Correct reversible causes

**Reversible Causes**
- Hypoxia
- Hypovolaemia
- Hypo- / hyperkalaemia/metabolic
- Hypothermia

- Thrombosis - coronary or pulmonary
- Tamponade - cardiac
- Toxins
- Tension pneumothorax

# TAHNK YOU