CARDIAC ARRHYTHMIAS **Learning objectives 1-Normal rhythm generation ,pathway and its normal variations

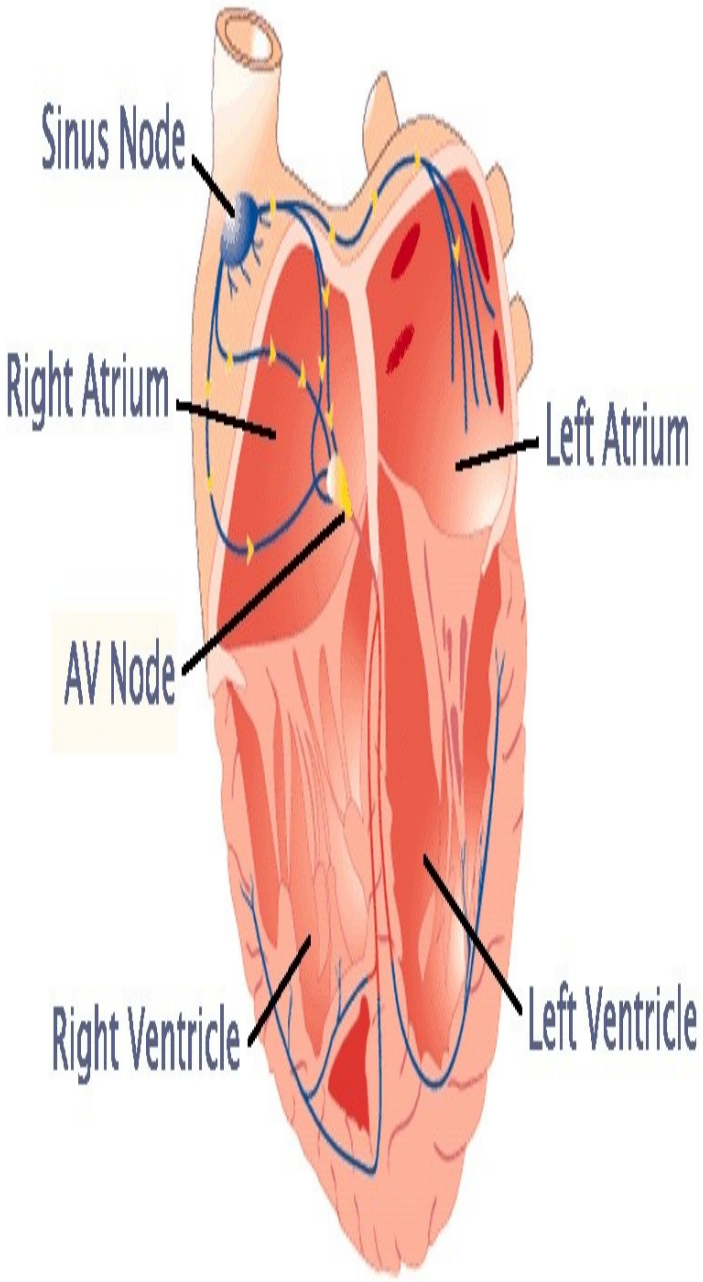
2-Mechanism and types of arrythmias 3-Management of arrythmias

4Risk factors for thromboembolism in atrial fibrillation

Sinus (Sinoatrial nodal) rhythm

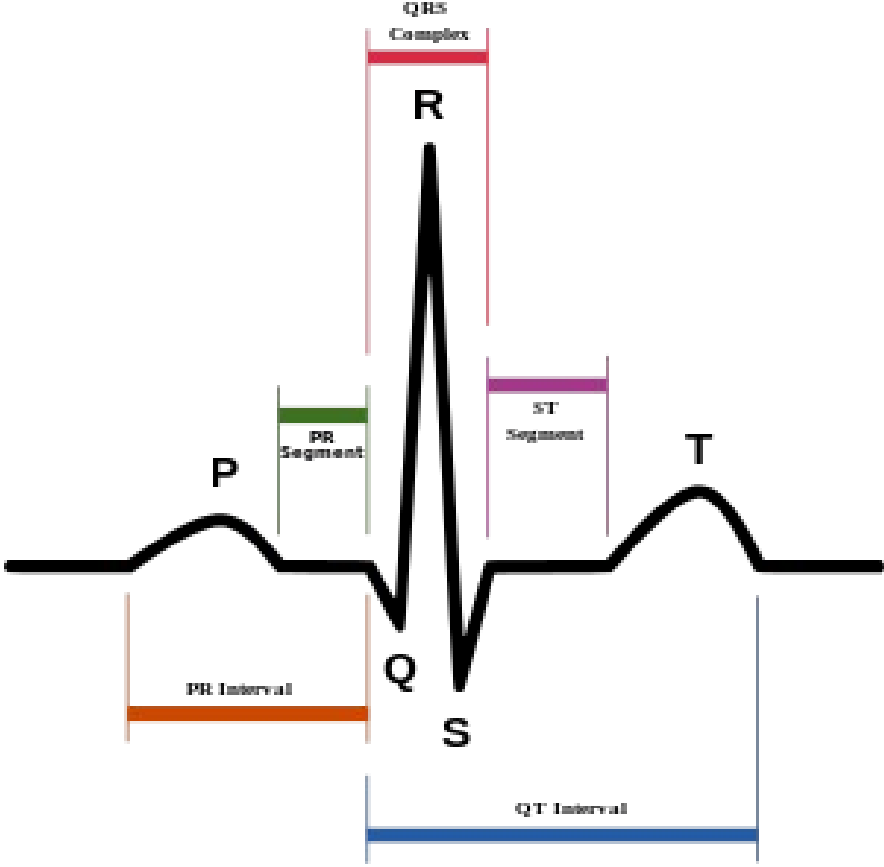
Sinus Rhythm: is the name given to the normal rhythm of the heart where electrical stimuli are initiated in the sinoatrial (SA) node, and are then conducted through the atrioventricular (AV) node and bundle of His, bundle branches and Purkinje fibres.

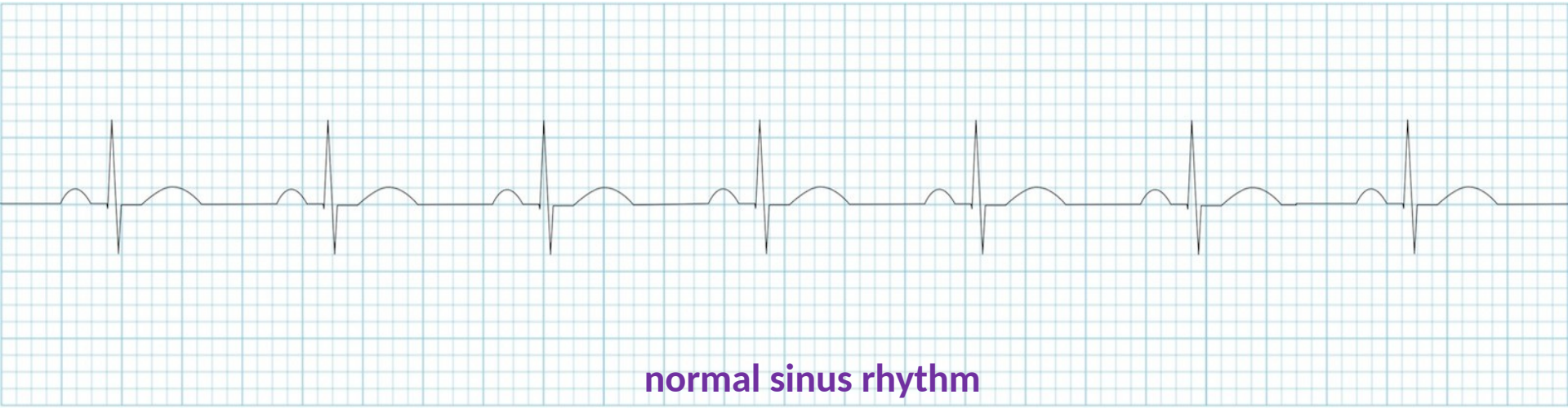
The sinus node acts as a pacemaker and its intrinsic rate is regulated by the autonomic nervous system; vagal activity decreases the heart rate, and sympathetic activity increases it via cardiac sympathetic nerves and circulating catecholamines.



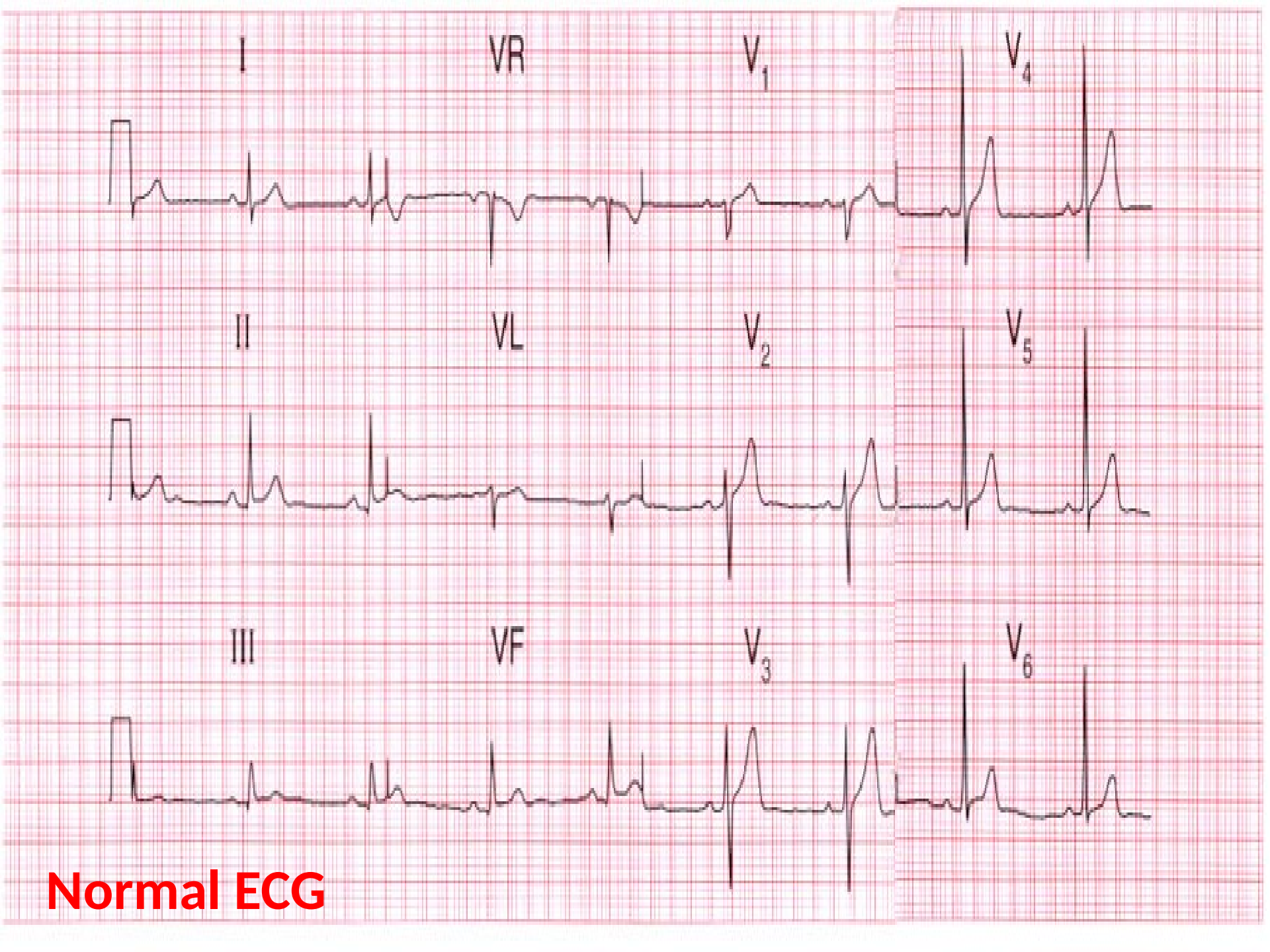
Characteristics of normal sinus rhythm:

- Regular rhythm at a rate of 60-100 bpm for an adult.
- Each QRS complex is preceded by a normal P wave (Smooth contour).
- Normal P wave axis: P waves should be upright in leads I and II, inverted in aVR.





normal sinus rhythm



Normal ECG

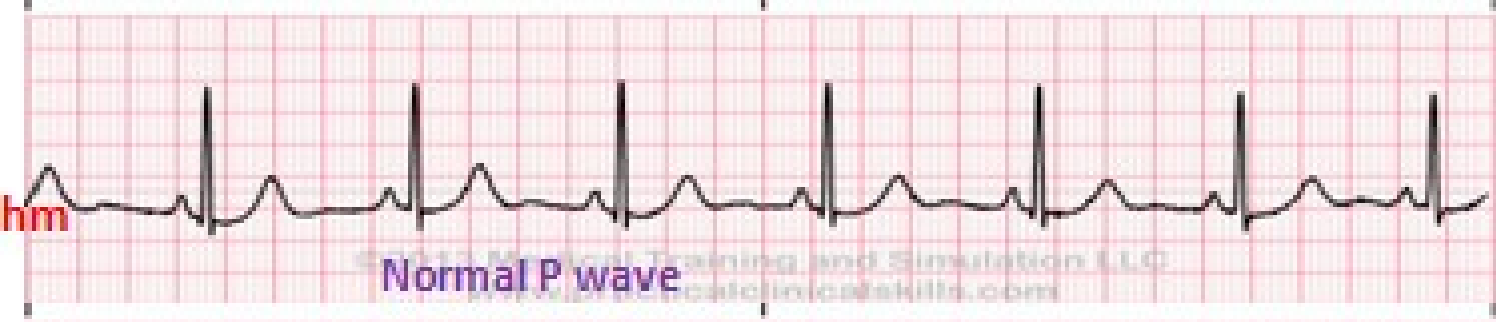
Variations on sinus rhythm

Sinus tachycardia: A sinus rate of more than 100/min, and is usually due to an increase in sympathetic activity associated with exercise, emotion, pregnancy or pathology (Anxiety, Fever, Anaemia, Heart failure, Thyrotoxicosis, Phaeochromocytoma, Drugs, e.g. β-agonists (bronchodilators))

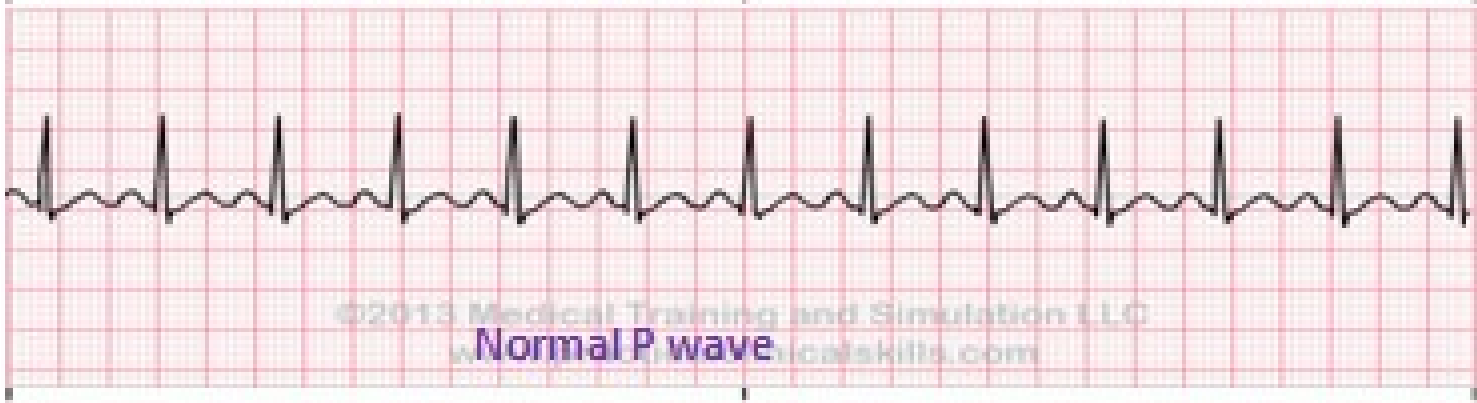
Sinus bradycardia: A sinus rate of less than 60/min may occur in healthy people at rest and is a common finding in athletes. Some pathological causes (Inferior myocardial infarction, Sinus node disease (sick sinus syndrome), Hypothermia, Hypothyroidism, Cholestatic jaundice, Raised intracranial pressure, Drugs (e.g. β-blockers, digoxin, verapamil))

Sinus arrhythmia: Phasic alteration of the heart rate during respiration (the sinus rate increases during inspiration and slows during expiration) is a consequence of normal parasympathetic nervous system activity and can be pronounced in children. Absence of this normal variation is an indication of autonomic neuropathy.

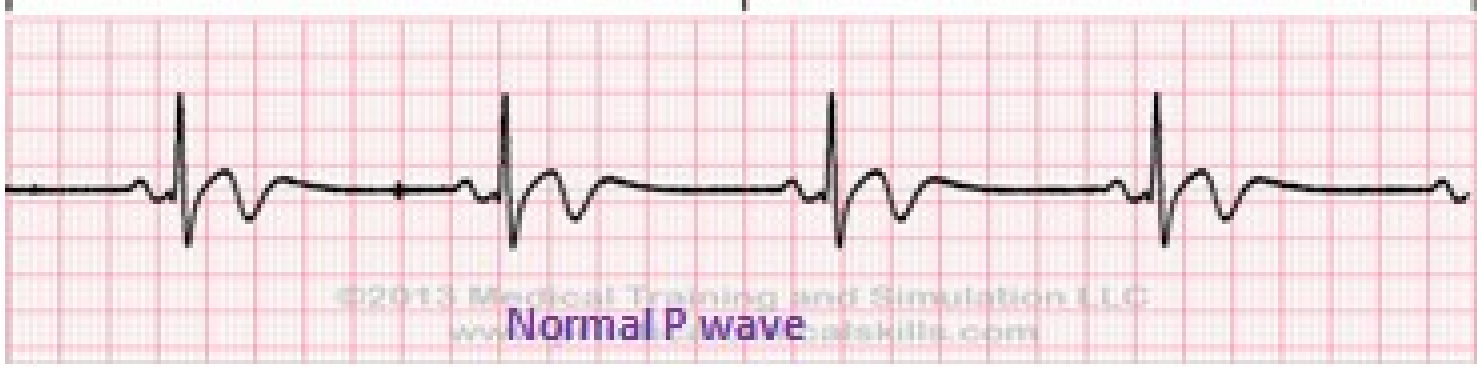
Normal Sinus Rhythm



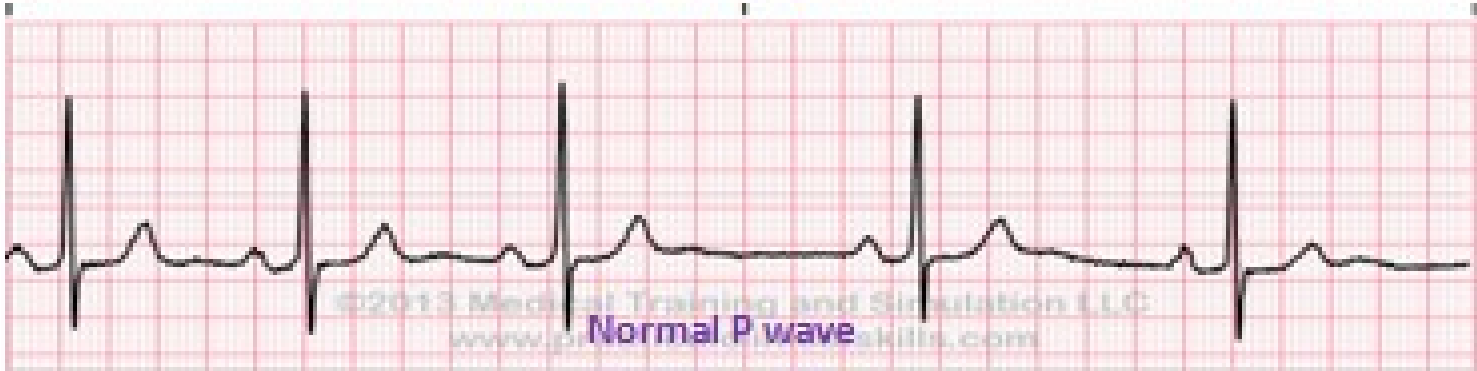
Sinus Tachycardia



Sinus Bradycardia



Sinus Arrhythmia

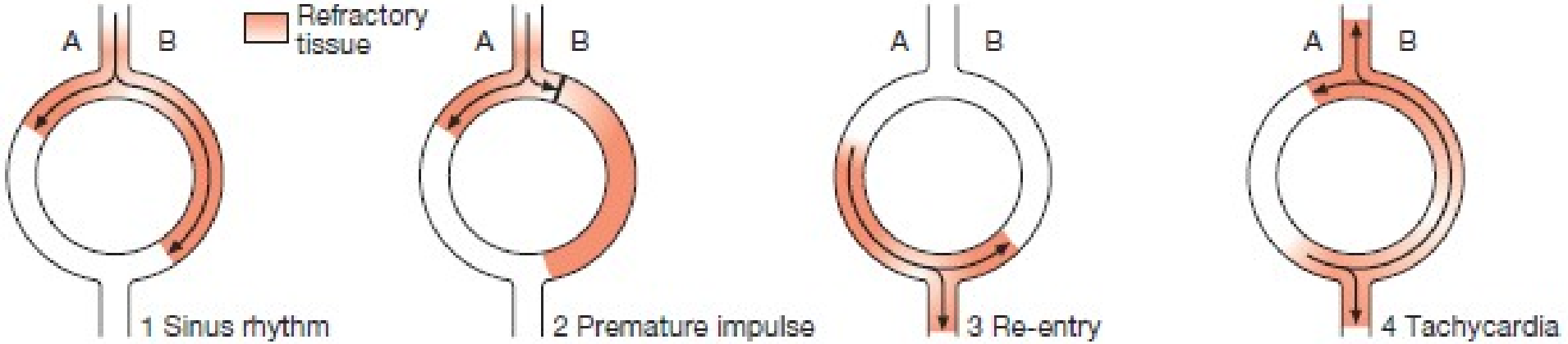


Arrhythmias

Arrhythmias: are disturbances of the electrical rhythm of the heart, which are a manifestation of structural heart disease or due to abnormal conduction or depolarisation in healthy heart.

There are **three main mechanisms** of tachycardia:

1. ***Increased automaticity:*** *The tachycardia is produced* by repeated spontaneous depolarisation of an ectopic focus, often in response to catecholamines.
2. ***Re-entry:*** *The tachycardia is initiated by an ectopic* beat and sustained by a re-entry circuit. Most tachyarrhythmias are due to re-entry.
3. ***Triggered activity:*** *This can cause ventricular* arrhythmias in patients with coronary artery disease. It is a form of secondary depolarisation arising from an incompletely repolarised cell membrane.



The mechanism of re-entry. Re-entry can occur when there are two alternative pathways with different conducting properties (e.g. the AV node and an accessory pathway, or an area of normal and an area of ischaemic tissue). Here, pathway A conducts slowly and recovers quickly, while pathway B conducts rapidly and recovers slowly.

.(1) In sinus rhythm, each impulse passes down both pathways before entering a common distal pathway.

(2) As the pathways recover at different rates, a premature impulse may find pathway A open and B closed.

(3) Pathway B may recover while the premature impulse is travelling selectively down pathway A. The impulse can then travel retrogradely up pathway B, setting up a closed loop or re-entry circuit.

(4) This may initiate a tachycardia that continues until the circuit is interrupted by a change in conduction rates or electrical depolarisation.

Classification of arrhythmias

Arrhythmias can be classified into supraventricular and ventricular in type.

Supraventricular arrhythmias usually produce narrow QRS complexes because the ventricles are depolarised in their normal sequence via the AV node and bundle of His.

In contrast, ventricular arrhythmias produce broad, bizarre QRS complexes because the ventricles are activated in an abnormal sequence.

Supraventricular arrhythmias

- Atrial ectopic beats (extrasystoles, Premature Atrial Complex (PAC))
- Supraventricular tachycardia (Atrioventricular nodal re-entrant tachycardia)
- Supraventricular tachycardias with Wolff–Parkinson–White syndrome
- Atrial flutter
- Atrial fibrillation

Ventricular arrhythmias

- Ventricular ectopic beats (extrasystoles, Premature ventricular complex (PVC))
- Ventricular tachycardia
- Ventricular fibrillation

Atrial ectopic beat (extrasystole, Premature Atrial Complex (PAC))

A PAC arising from an ectopic focus within the atria

ECG features: An abnormal or ectopic (non-sinus) P wave is followed by a QRS complex. The ectopic P has a different morphology, inverted or may be hidden in the preceding T wave. PAC arriving early in the cardiac cycle followed by post-extrasystolic pause.

PAC may be either:

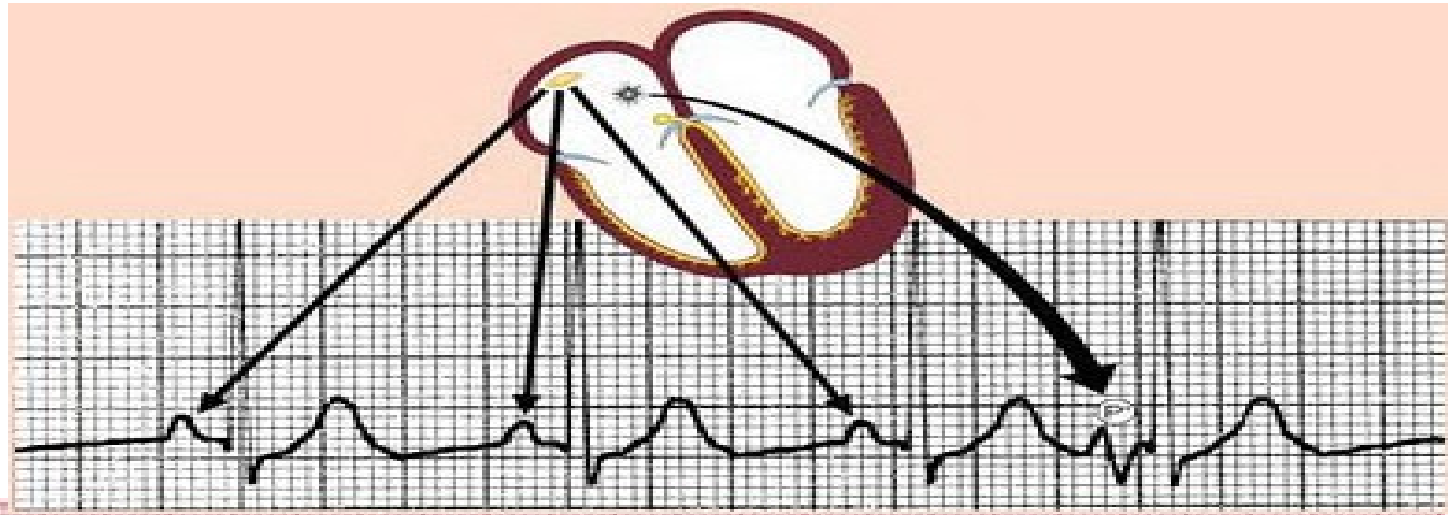
Unifocal: Arising from a single ectopic focus, each PAC is identical.

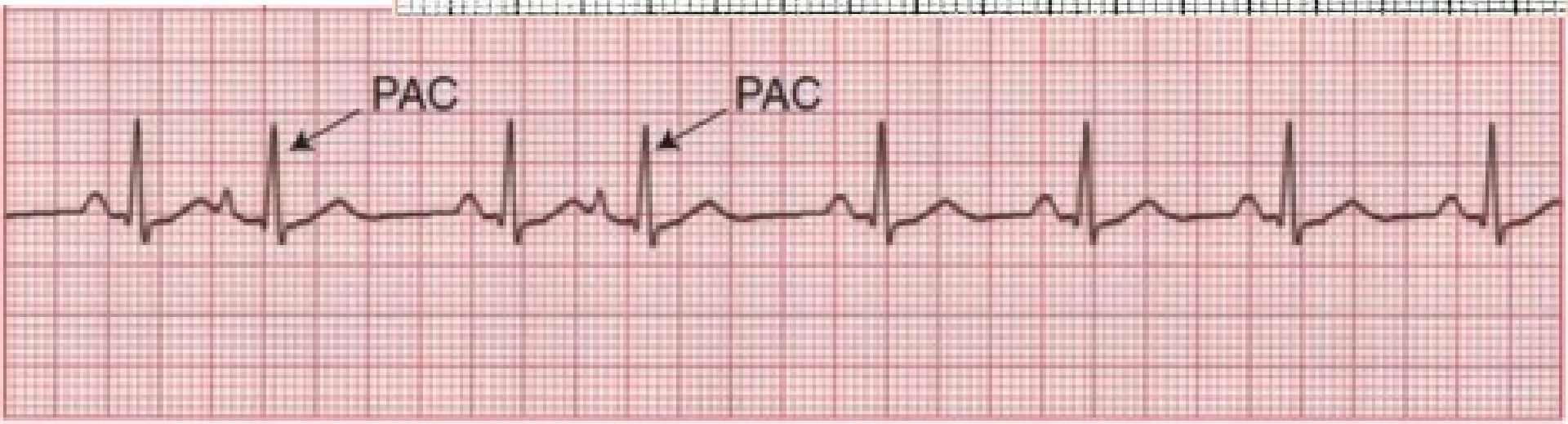
Multifocal: Arising from two or more ectopic foci, multiple P-wave morphologies.

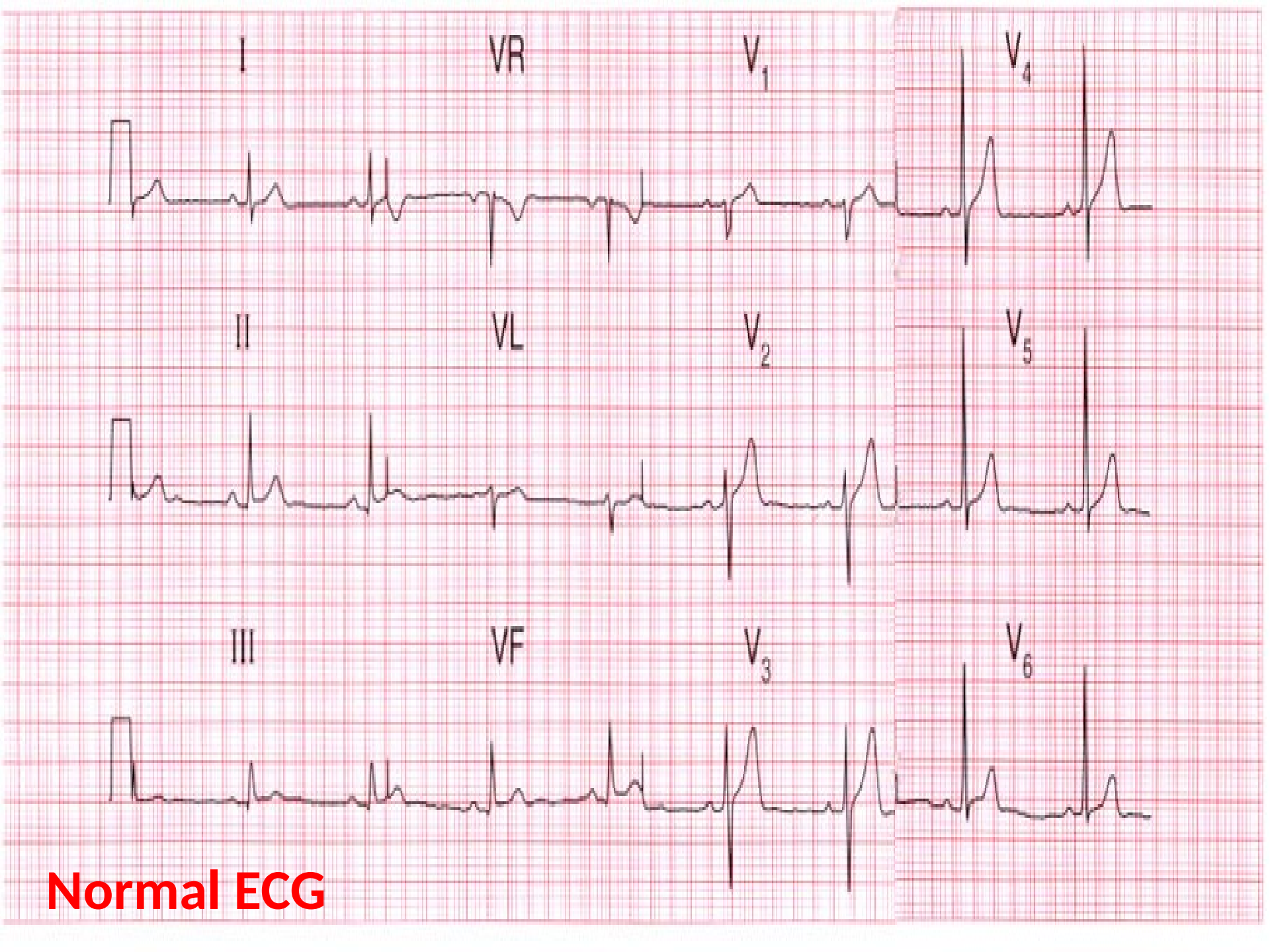
The most common symptoms include palpitations, or the sensation of skipped beats.

Treatment is rarely necessary but β-blockers can be used for highly symptomatic patients.

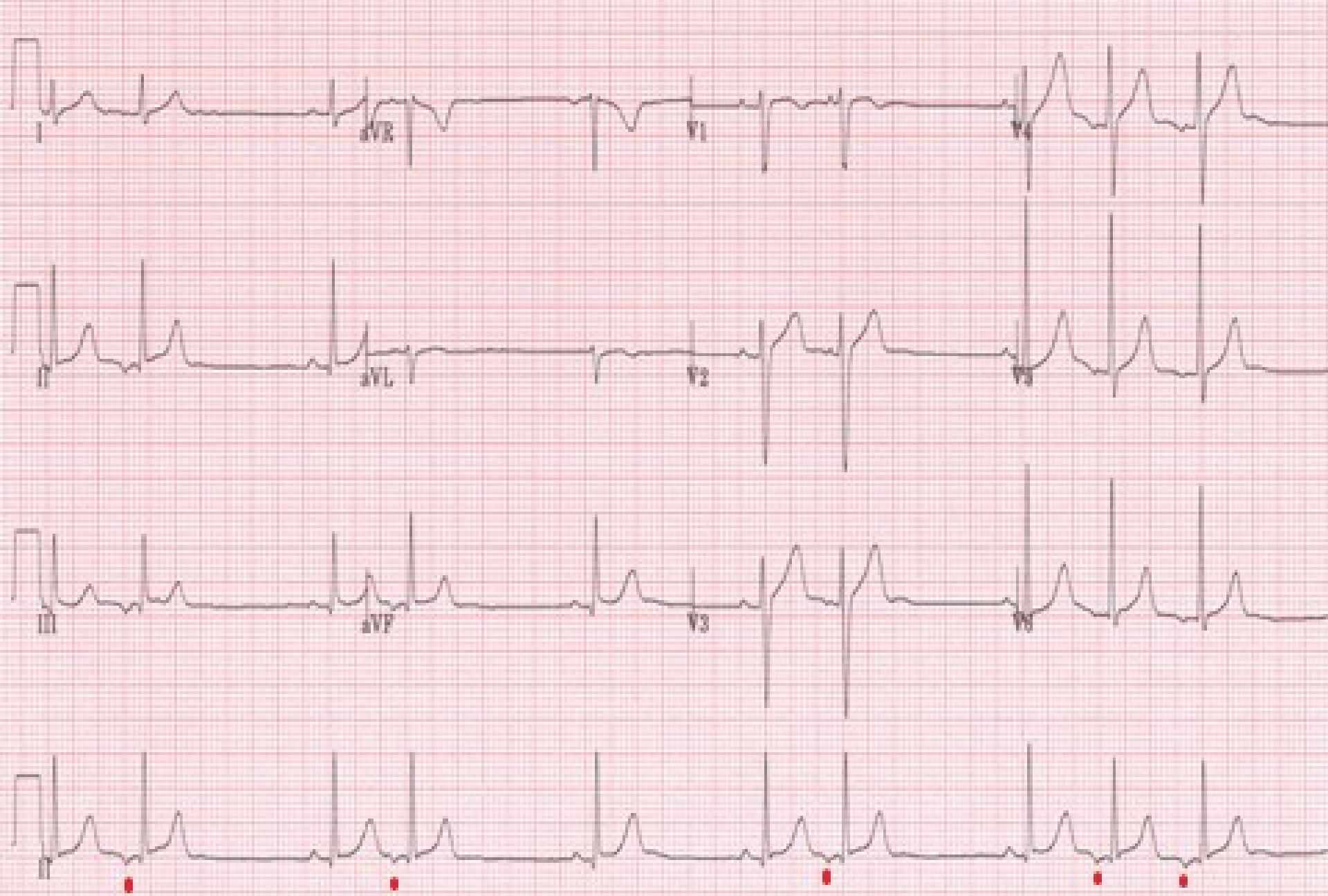
Premature atrial complexe occurs earlier than the next expected sinus complexes







Normal ECG



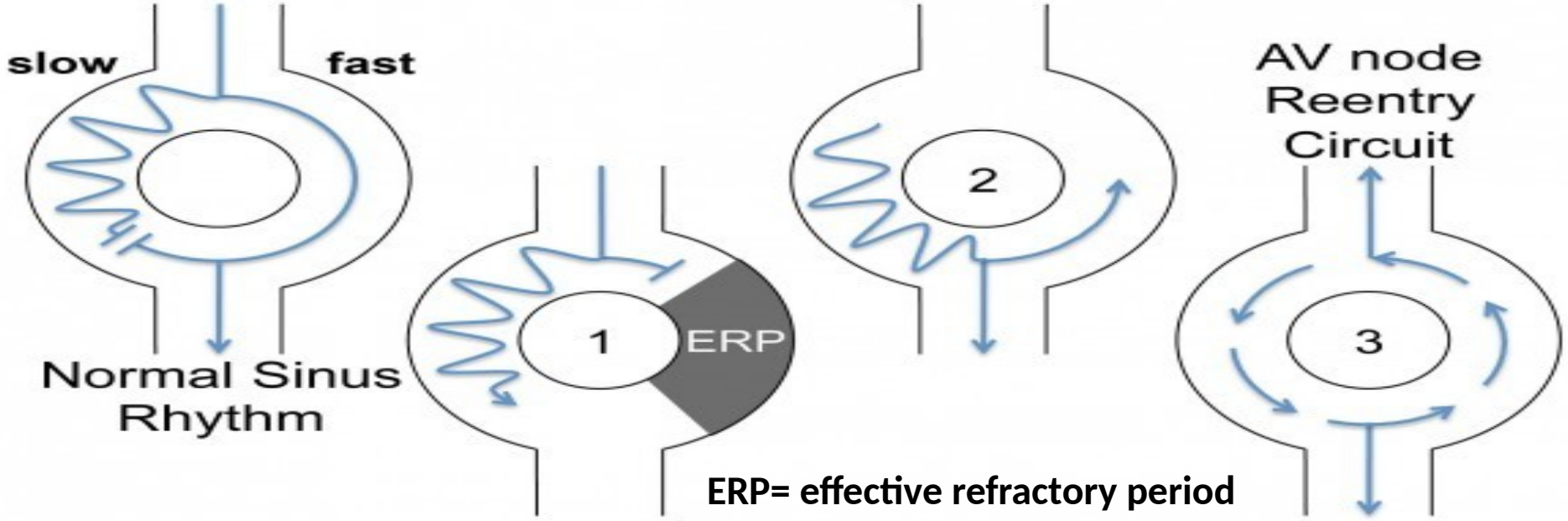
Frequent PAC, referred by red dots below it.

Supraventricular Tachycardia (SVT)

Supraventricular tachycardia (SVT): is referred to describe the sudden onset and termination of ectopic, regular, narrow QRS complex tachycardia on ECG, due to presence of re-entry circuit or automatic focus involving the atria.

ECG Features: Regular tachycardia >150-280 bpm. P waves are absent, or present as inverted small waves inside the QRS complexes or T waves. QRS complexes usually narrow (< 110 ms).

The patient is usually aware of a rapid, very forceful, regular heart beat and may experience chest discomfort, lightheadedness or breathlessness. Polyuria, mainly due to the release of atrial natriuretic peptide, is sometimes a feature.

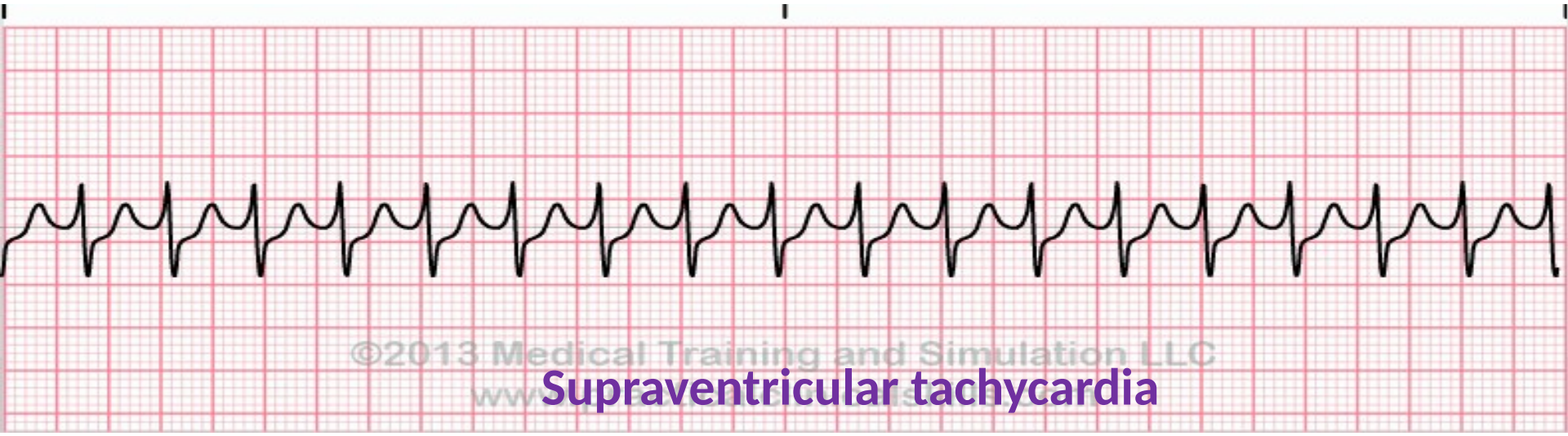


Mechanism of re-entry in SVT:

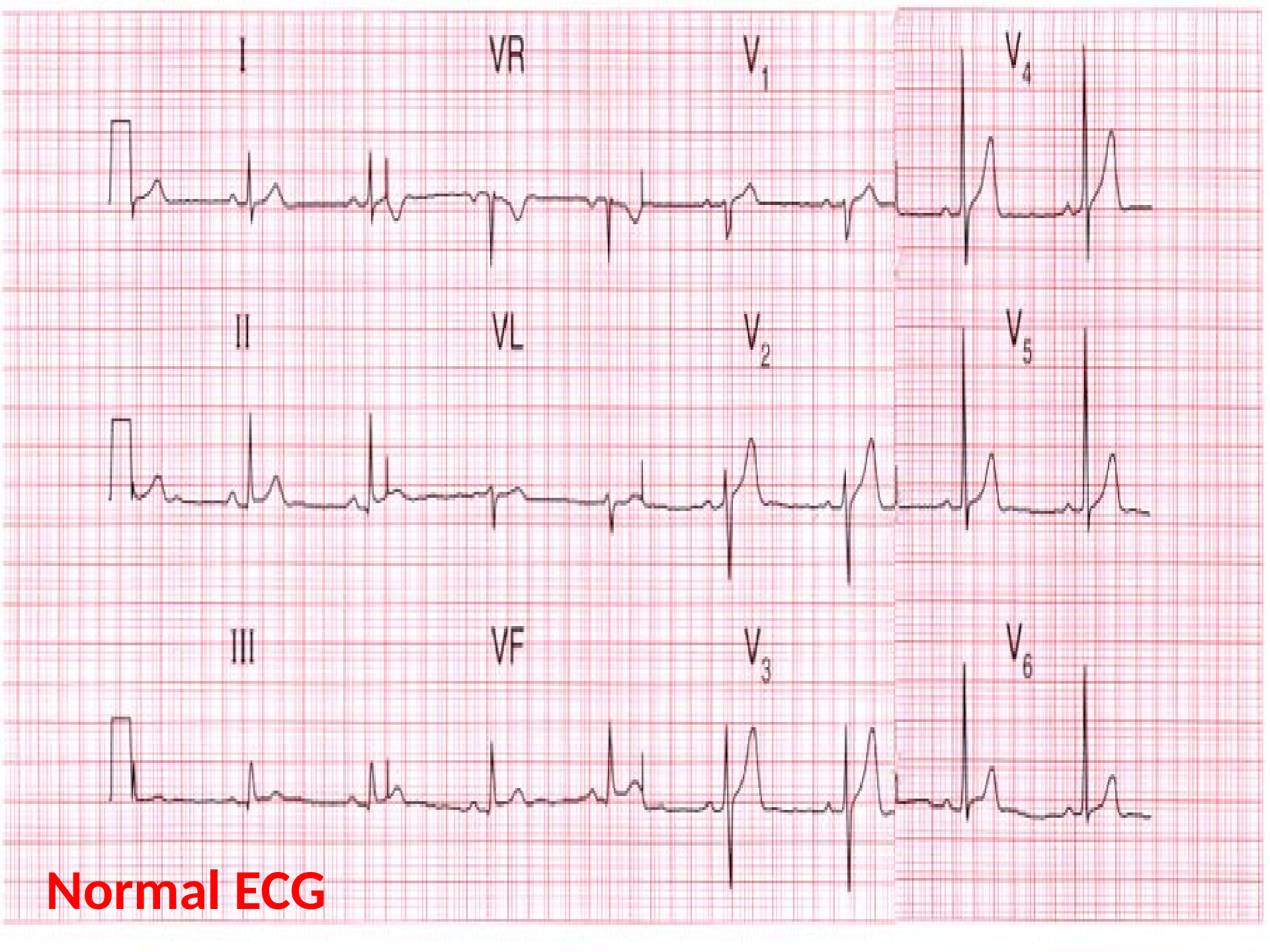
- During sinus rhythm, electrical impulses travel down both pathways simultaneously. The impulse transmitted down the fast pathway enters the distal end of the slow pathway and the two impulses cancel each other out.
- However, if a PAC arrives while the fast pathway is still refractory, the electrical impulse will be directed solely down the slow pathway (1).
- By the time the premature impulse reaches the end of the slow pathway, the fast pathway is no longer refractory (2). Hence the impulse is permitted to recycle retrogradely up the fast pathway.
- This creates a circus movement whereby the impulse continually cycles around the two pathways, which is responsible for the rapid heart rate.

Treatment is not always necessary. However, an episode may be terminated by carotid sinus pressure or by the Valsalva manoeuvre. Intravenous adenosine or verapamil will restore sinus rhythm in most cases.

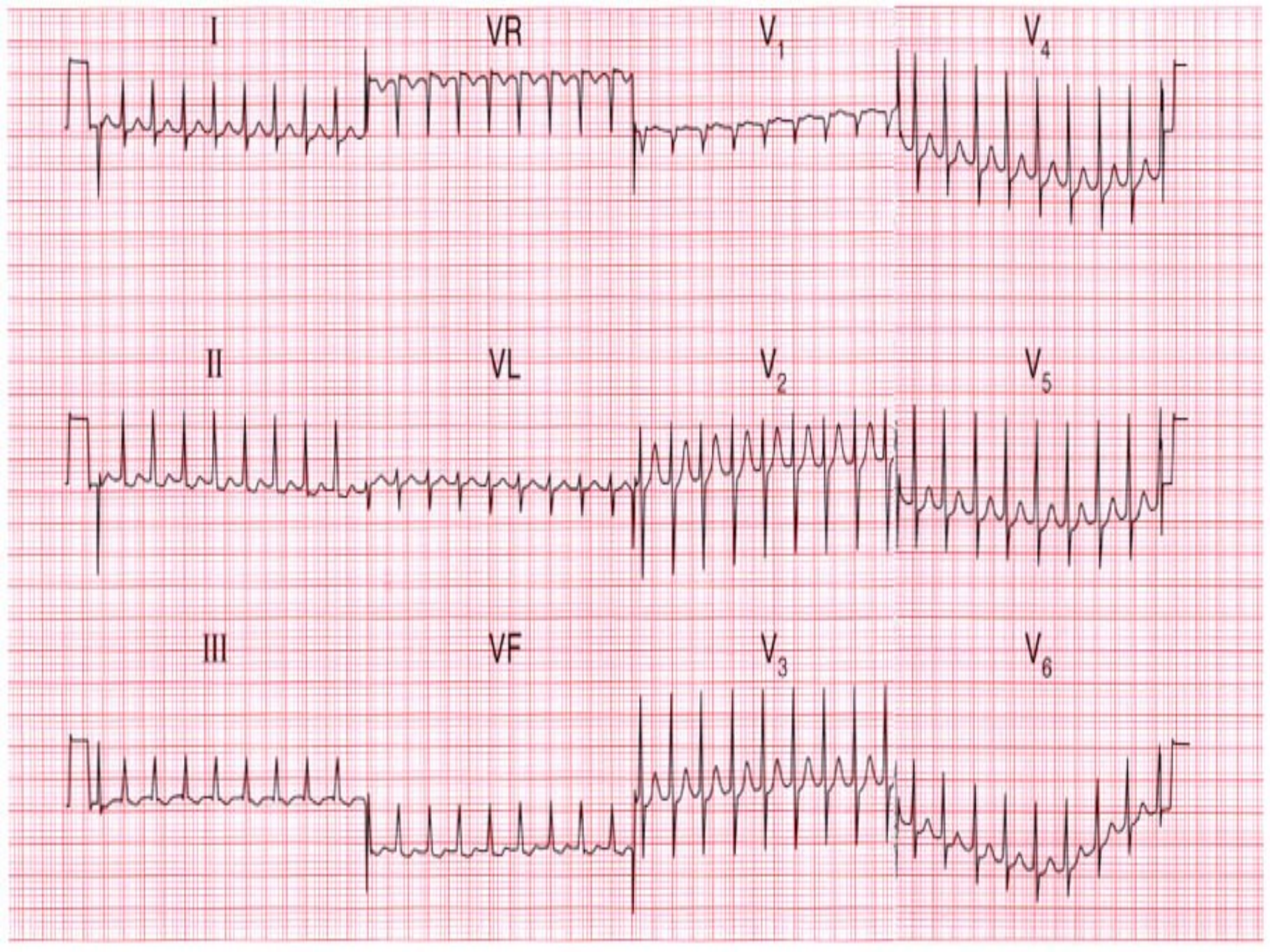
In patients with recurrent SVT, catheter ablation is the most effective therapy. Alternatively, prophylaxis with oral β-blocker, verapamil or flecainide may be used.



Supraventricular tachycardia



Normal ECG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V3
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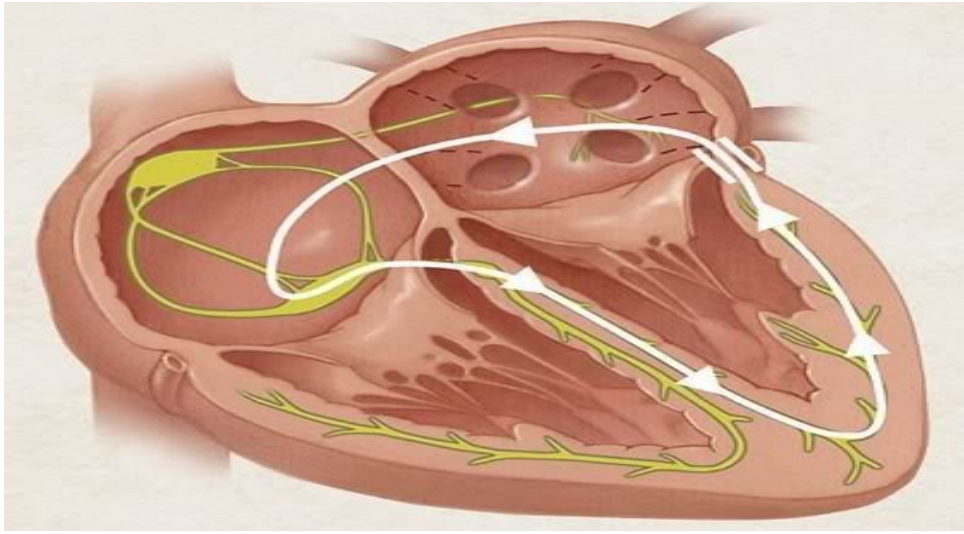
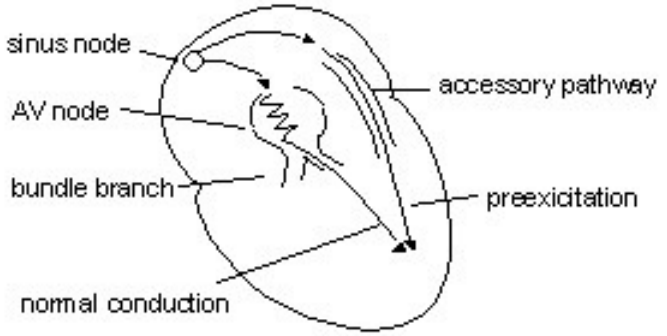
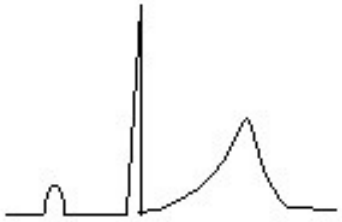
Wolff–Parkinson–White syndrome

In WPW the accessory pathway is often referred to as the Bundle of Kent (an abnormal band of conducting tissue connects the atria and ventricles). which conduct impulses either anterograde, towards the ventricle, retrograde, away from the ventricle, or in both directions. The direction of conduction affects the appearance of the ECG in sinus rhythm and during tachyarrhythmias.

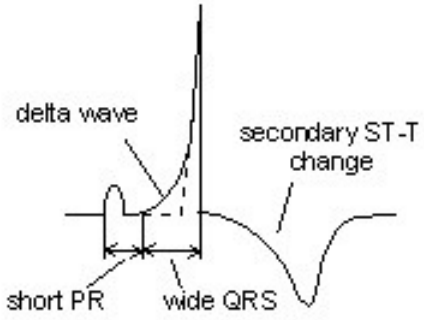




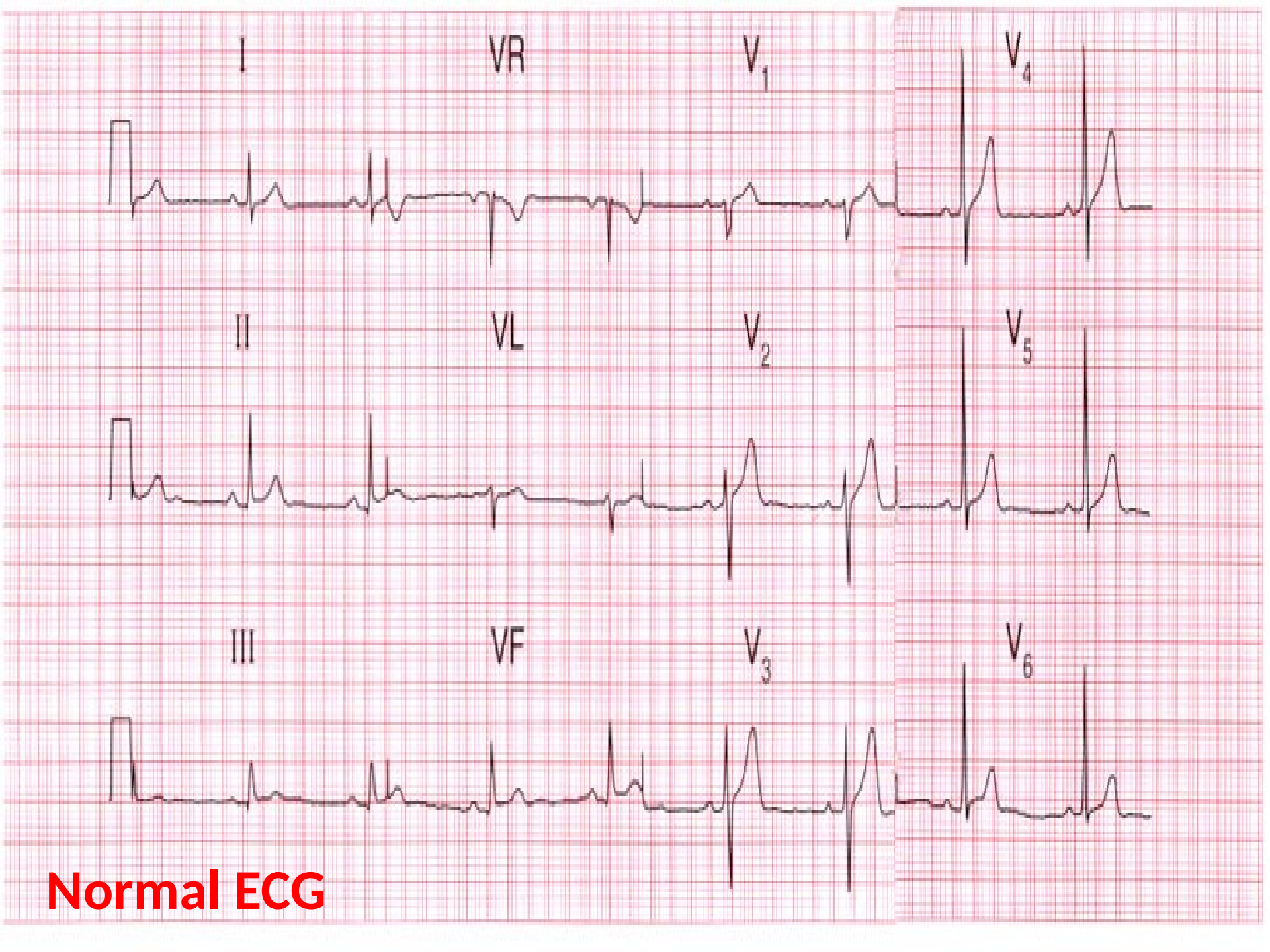
ECG features of WPW in sinus rhythm are: short PR interval (<120ms). Delta wave- slurring slow rise of initial portion of the QRS. QRS prolongation >110ms.



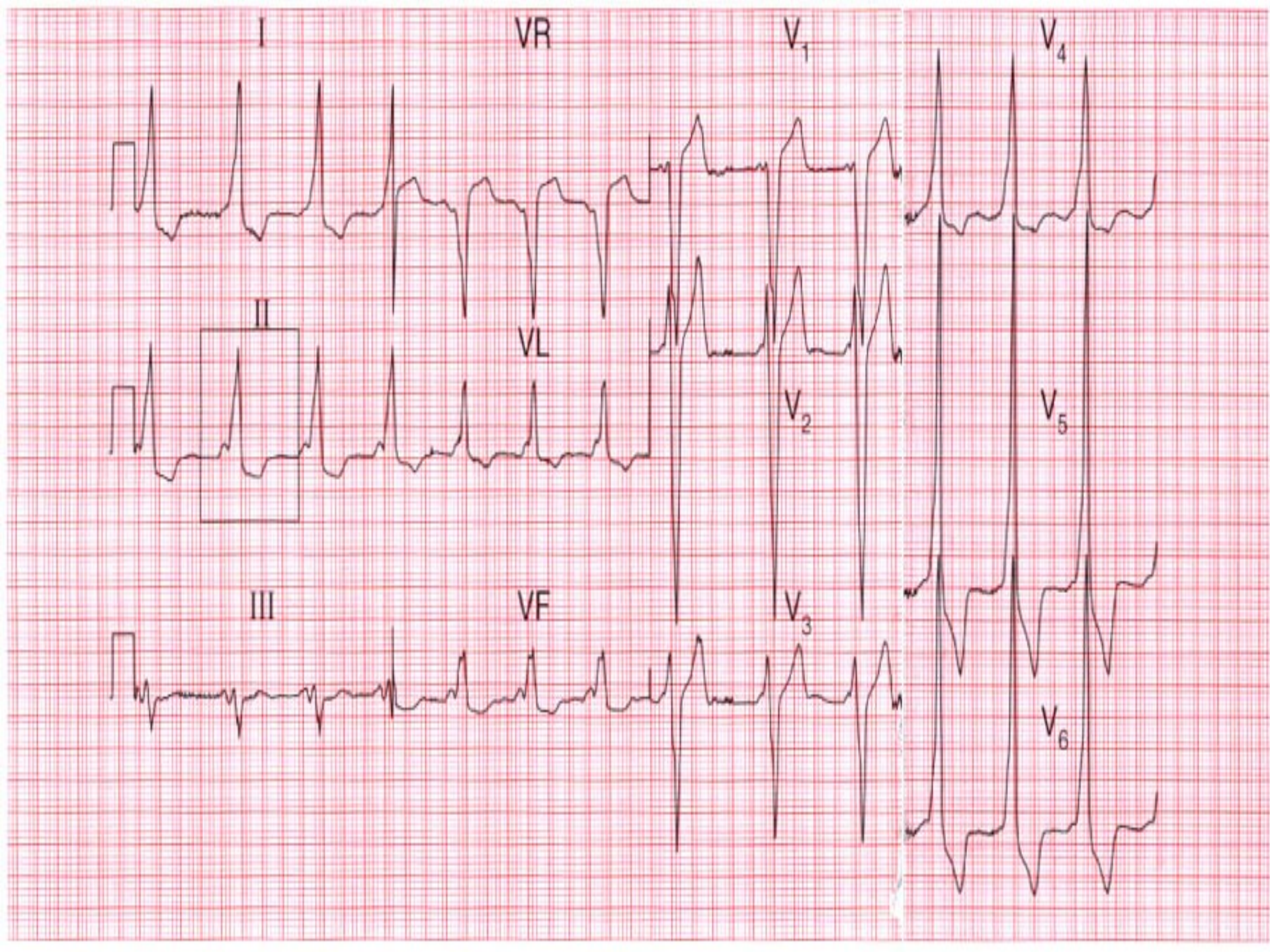
Normal conduction



Preexcitation



Normal ECG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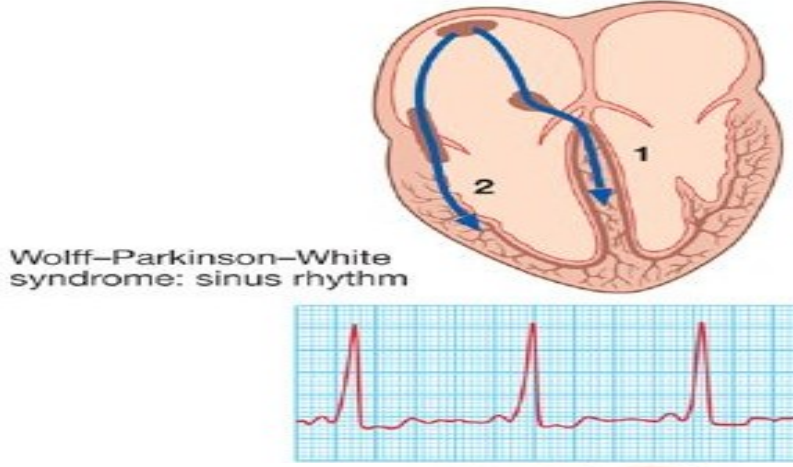
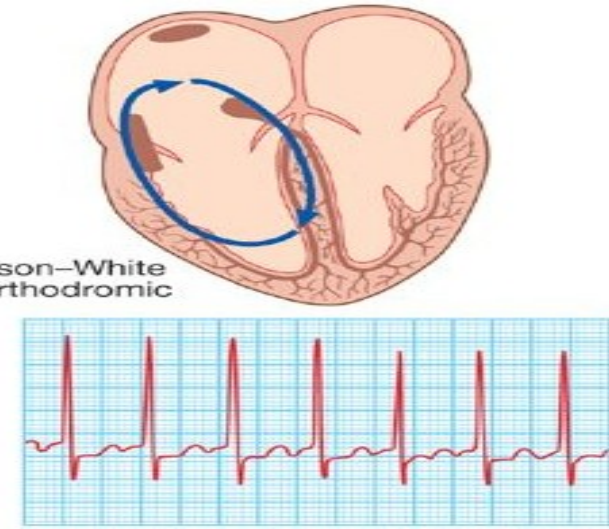
V3
V6

Sinus rhythm. In sinus rhythm the ventricles are depolarised through (1) the AV node and (2) the accessory pathway, producing an ECG with a short PR interval and broadened QRS complexes; the characteristic slurring of the upstroke of the QRS complex is known as a delta wave.



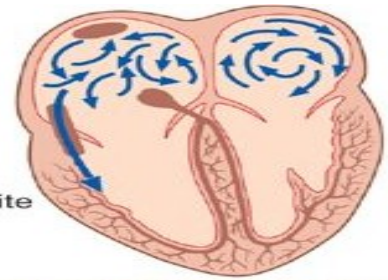
Wolff–Parkinson–White syndrome: sinus rhythm

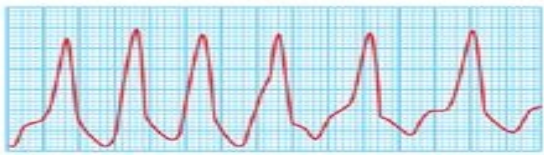


Orthodromic tachycardia. This is the most common form of tachycardia in WPW. The re-entry circuit passes antegradely through the AV node and retrogradely through the accessory pathway. The ventricles are therefore depolarised in the normal way, producing a narrow-complex tachycardia that is indistinguishable from other forms of SVT.

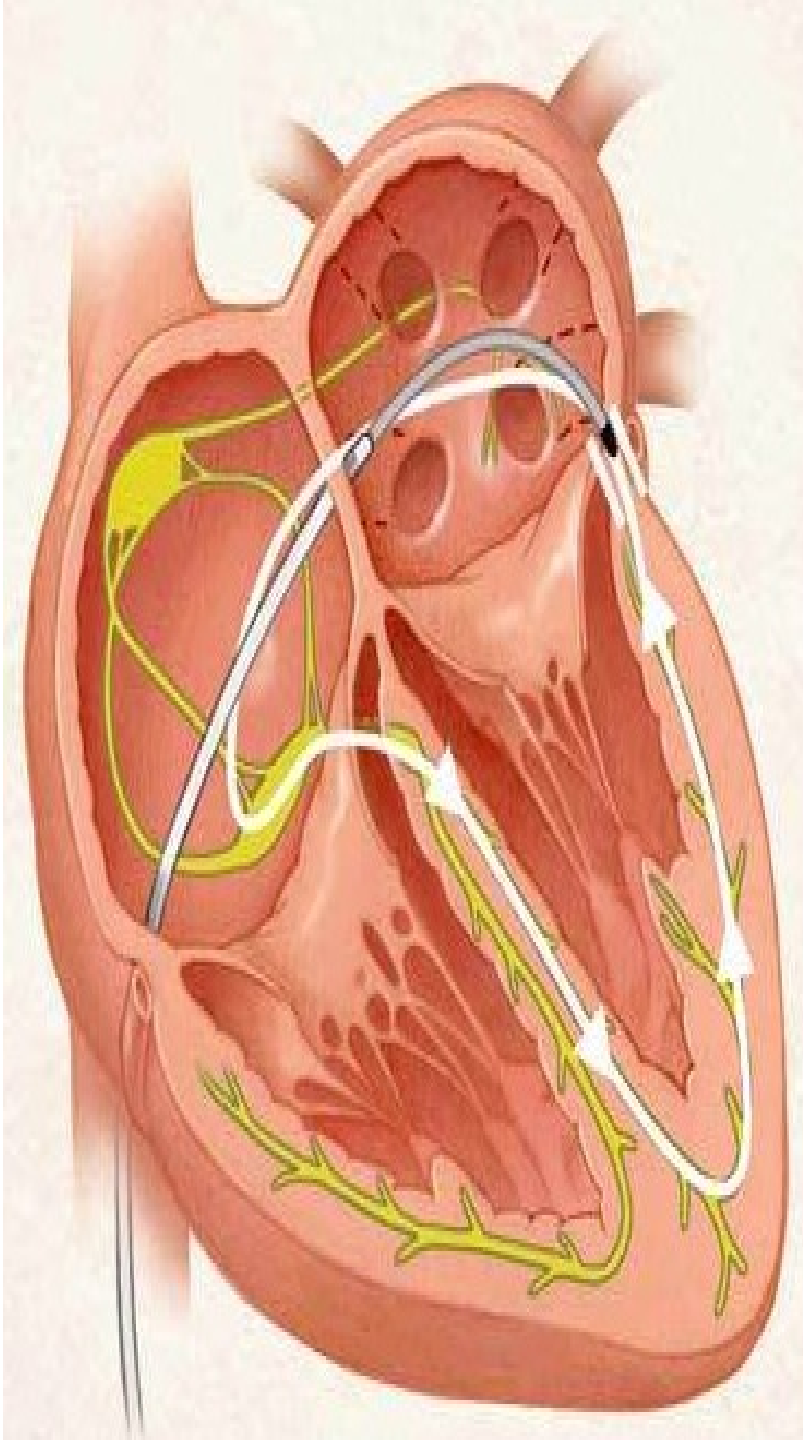
Wolff–Parkinson–White syndrome: orthodromic tachycardia

Atrial fibrillation. In this rhythm the ventricles are largely depolarised through the accessory pathway, producing an irregular broad-complex tachycardia, which is often more rapid than the example shown


Wolff–Parkinson–White syndrome: atrial fibrillation

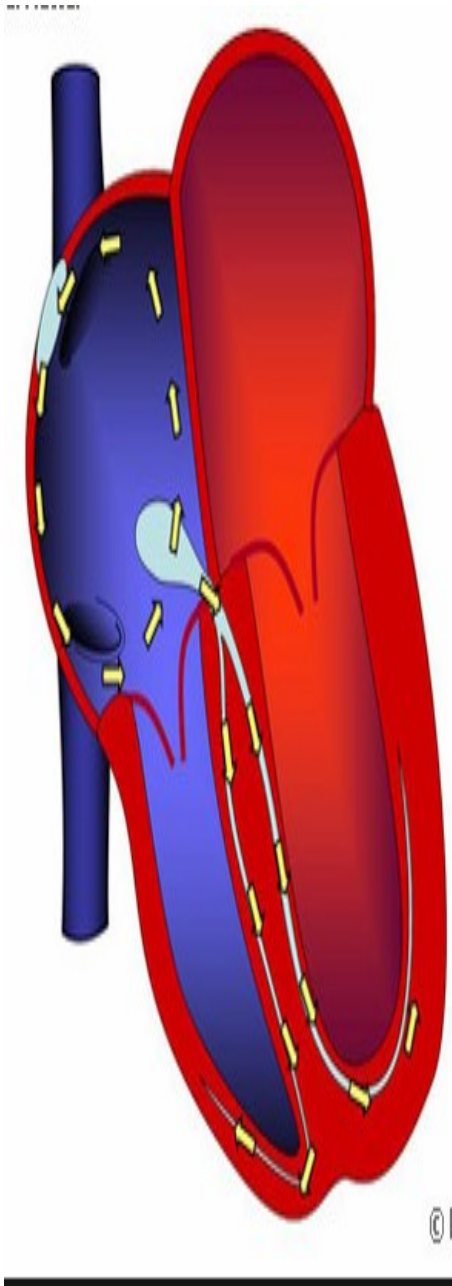


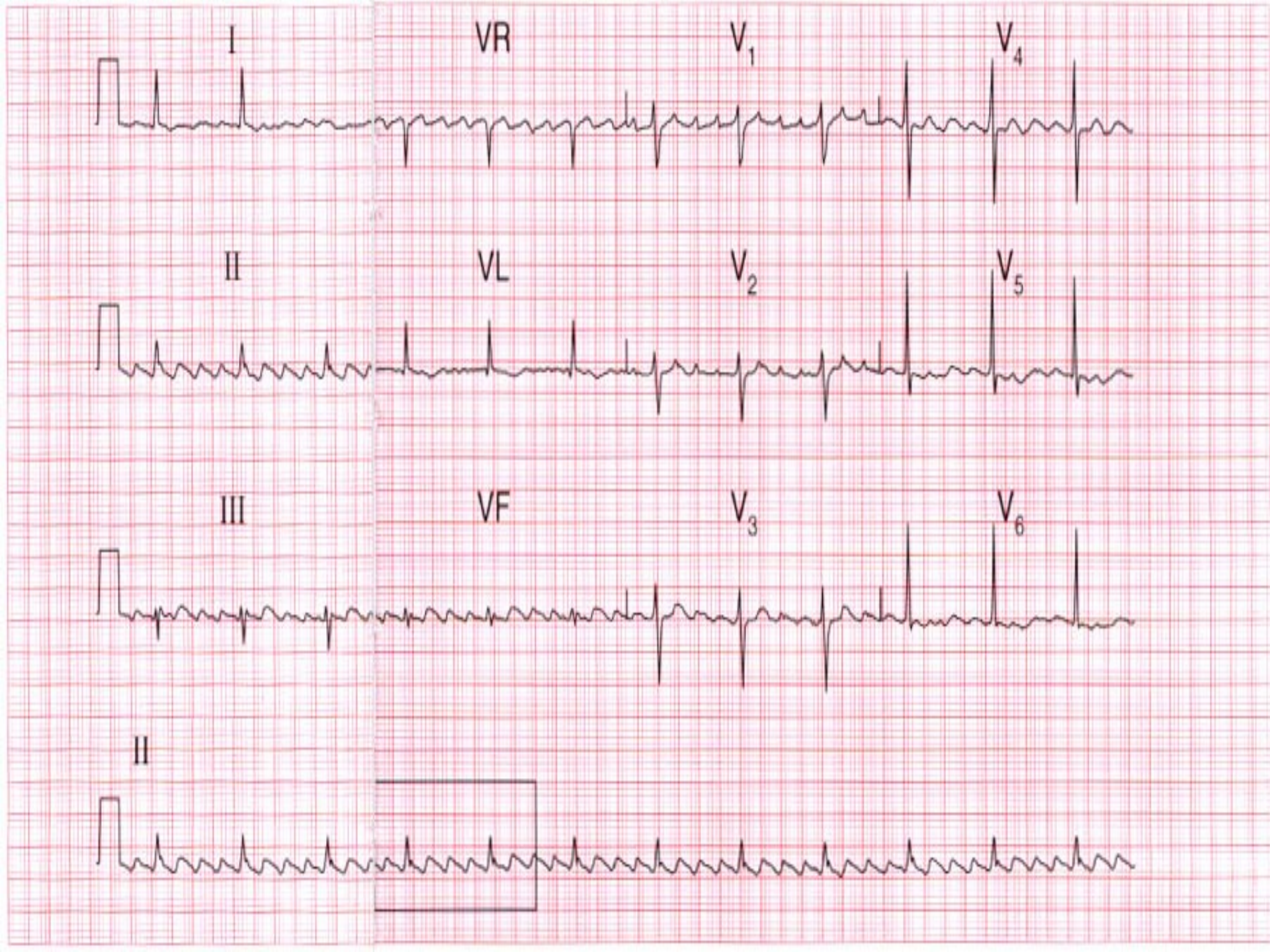
Catheter ablation is first-line treatment in symptomatic patients and is nearly always curative. Alternatively, prophylactic anti-arrhythmic drugs, such as flecainide or propafenone, can be used to slow conduction in, and prolong the refractory period of, the accessory pathway.



Atrial flutter

- Atrial flutter is characterised by a large (macro) re-entry circuit, usually within the right atrium encircling the tricuspid annulus.
- ECG features: Narrow complex tachycardia. Regular atrial activity, the atrial rate is approximately 300/min, and is usually associated with 2 : 1, 3 : 1 or 4 : 1 AV block (with corresponding heart rates of 150, 100 or 75/min). Flutter waves ("saw-tooth" pattern) best seen in leads II, III, aVF. Loss of the isoelectric baseline.
- When there is regular 2 : 1 AV block, it may be difficult to identify flutter waves. Carotid sinus pressure or intravenous adenosine may help to establish the diagnosis by temporarily increasing the degree of AV block and revealing flutter waves.

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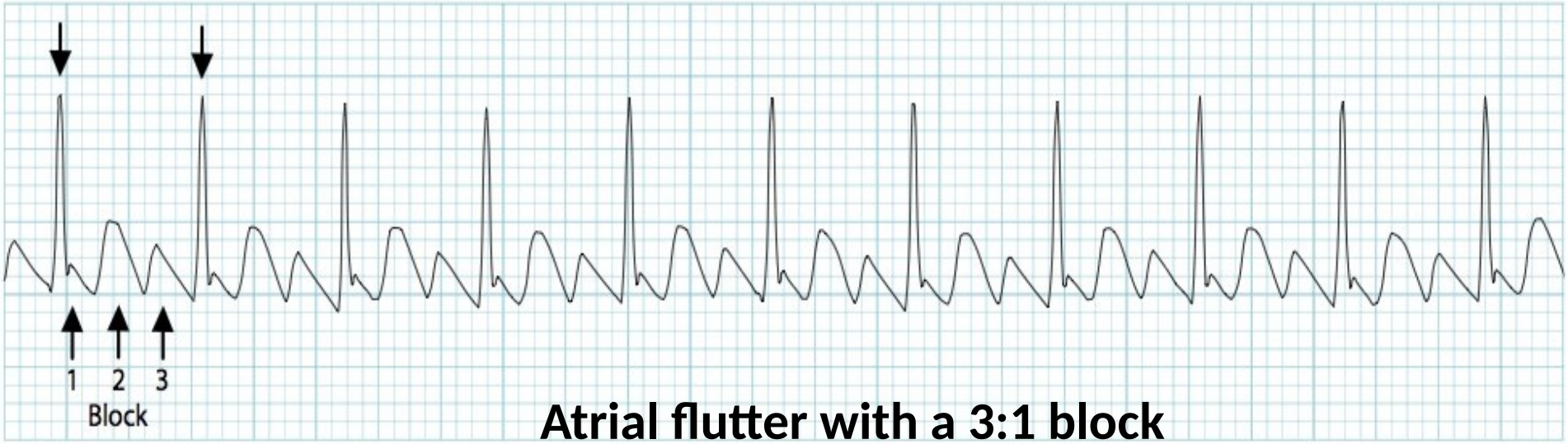
Management

Control the ventricular rate by digoxin, β-blockers or verapamil.

Restore sinus rhythm by direct current (DC) cardioversion or by using intravenous amiodarone.

Catheter ablation offers a 90% chance of complete cure and is the treatment of choice for patients with persistent symptoms.



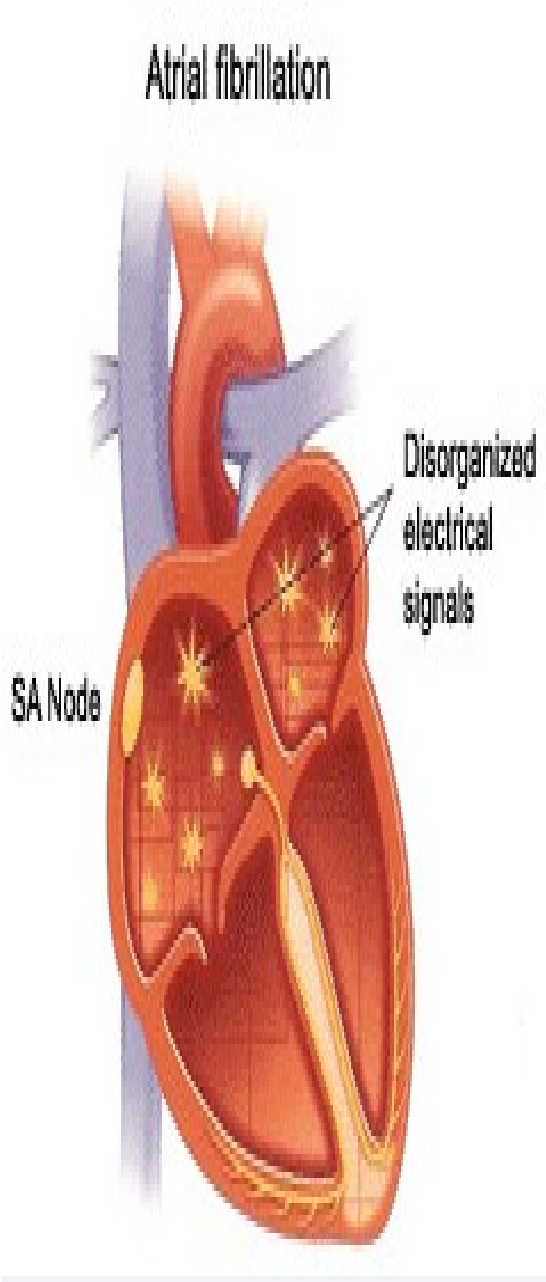
Atrial flutter with a 3:1 block

Atrial fibrillation (AF)

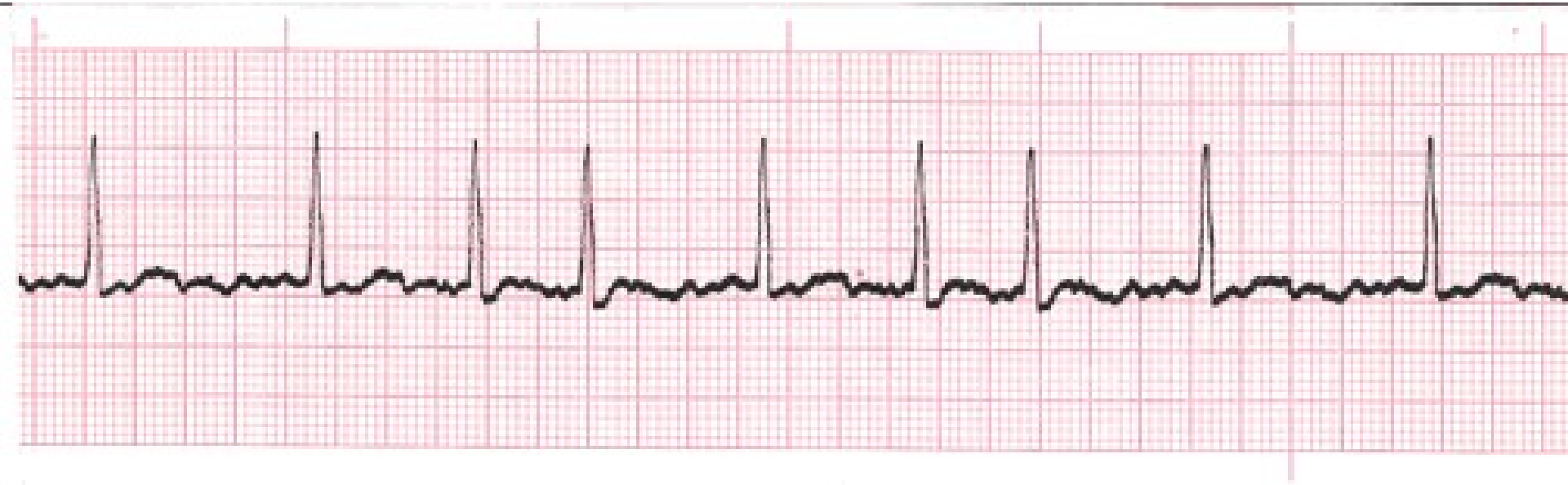
AF is the most common sustained cardiac arrhythmia. The prevalence rises with age, affecting 1% of those aged 60–64 years, increasing to 9% of those aged over 80 years.

AF is a complex arrhythmia characterised by both abnormal automatic firing and the presence of multiple interacting re-entry circuits looping around the atria.

AF can be classified as paroxysmal (intermittent episodes which self-terminate within 7 days), persistent (prolonged episodes that can be terminated by electrical or chemical cardioversion) or permanent.

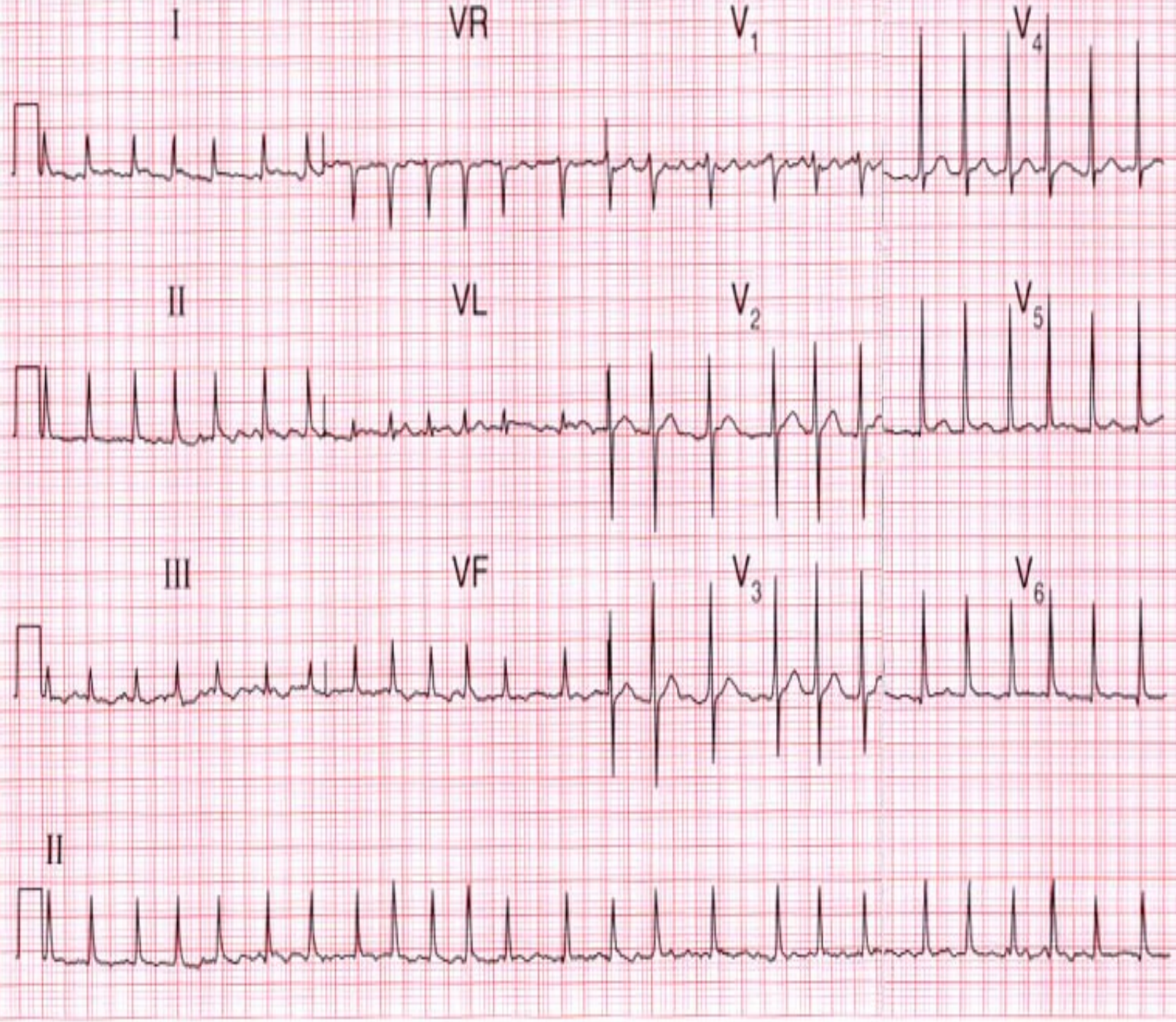


ECG Features of AF: Irregularly irregular ectopic rhythm. No P waves. Absence of an isoelectric baseline, small fibrillatory waves may be present .Variable ventricular rate. Narrow QRS complexes.



AF is associated with significant increase in morbidity and mortality because of :

1. **Its association with other underlying heart disease**
2. **Its association with stroke and systemic embolism**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Common causes of atrial fibrillation

- Coronary artery disease (including acute MI)
- Valvular heart disease, especially rheumatic mitral valve disease
- Hypertension
- Sinoatrial disease
- Hyperthyroidism
- Alcohol
- Cardiomyopathy
- Congenital heart disease
- Chest infection
- Pulmonary embolism
- Pericardial disease
- Idiopathic (lone atrial fibrillation)

AF can cause palpitation, breathlessness, fatigue, lightheadedness, and chest pain. Patient may be asymptomatic, discovered on routine examination or ECG.

In patients with poor ventricular function or valve disease, it may precipitate or aggravate cardiac failure because of loss of atrial function and heart rate control.

Management

Management of an acute episode

Objectives are to optimise heart rate during AF, restore sinus rhythm as soon as possible, prevent thromboembolism and identify/treat precipitating factors.

- Within 48 hrs of AF onset, heparinisation and cardioversion with a synchronised DC shock or drug therapy (flecainide or amiodarone) can be attempted.
- Beyond 48 hrs, the ventricular rate should be controlled and cardioversion deferred until anticoagulation with warfarin (INR 2–3) has been established for at least 3 wks.
- When AF complicates an acute illness (e.g. chest infection), treatment of the primary disorder usually restores sinus rhythm.
- All patients with haemodynamic compromise (e.g. hypotension) should receive immediate i.v. heparin and DC cardioversion.

Long-term management

The aim is to maintain sinus rhythm, or achieving an appropriate heart rate.

Rhythm control- prevention of recurrent episodes: ***Treatment with*** **amiodarone or β-blockers may reduce risk of recurrence following successful cardioversion.**

Rate control: **β-blockers and rate-limiting calcium antagonists (e.g. verapamil) are often more effective than digoxin at controlling the heart rate during exercise.**

Prevention of thromboembolism: **Warfarin**

(target INR 2.0–3.0) is indicated for patients with AF and specific risk factors for stroke, as shown in the next slide:

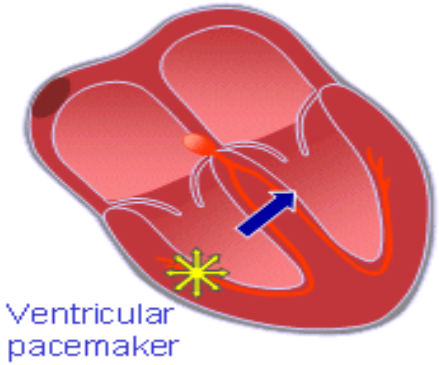
Risk factors for thromboembolism in AF

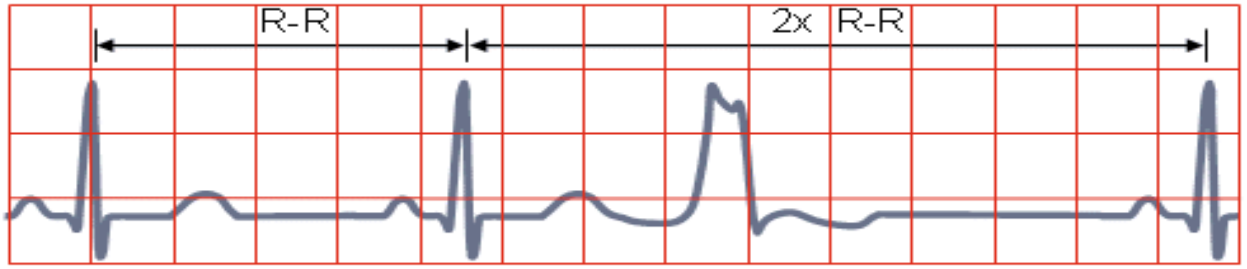
- Previous ischaemic stroke or TIA
- Mitral valve disease
- Age >65 yrs
- Hypertension
- Diabetes mellitus
- Heart failure
- Echocardiographic features of LV dysfunction, left atrial enlargement or mitral annular calcification

Ventricular ectopic beats (Extrasystoles, Premature ventricular complex (PVC))

Premature ventricular complex (PVC): A premature beat arising from an ectopic focus within the ventricles, firing of this focus within the ventricles bypasses the His-Purkinje system and depolarizes the ventricles directly.

ECG Features: Broad QRS complex (≥ 120 ms) with abnormal morphology (bizarre shape). Premature: i.e. occurs earlier than would be expected for the next sinus impulse. Usually followed by a compensatory pause. The ST segment and T wave are directed opposite to the main vector of the QRS complex





PVCs may be either:

Unifocal: arising from a single ectopic focus; each PVC is identical.

Multifocal: arising from two or more ectopic foci; multiple QRS morphologies.

PVCs often occur in repeating patterns:

Bigeminy: every other beat is a PVC.

Trigeminy: every third beat is a PVC.

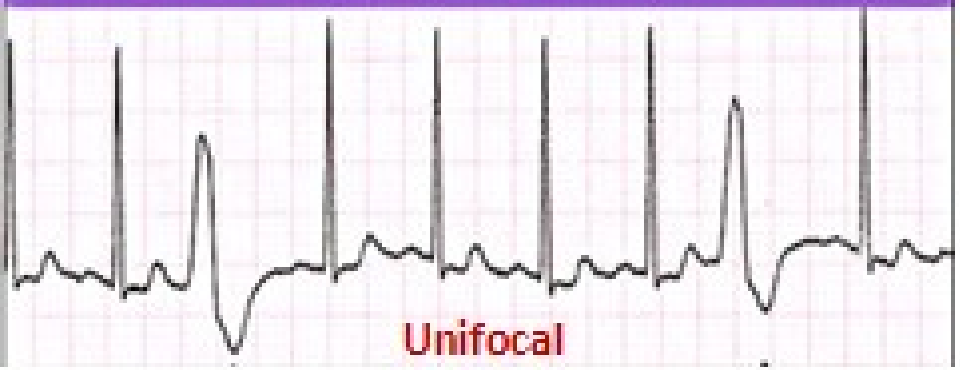
Quadrigeminy: every fourth beat is a PVC.

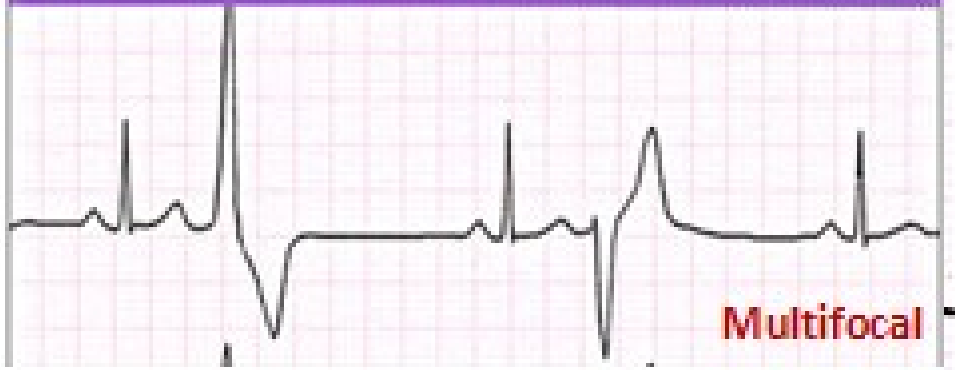
Couplet: two consecutive PVCs.

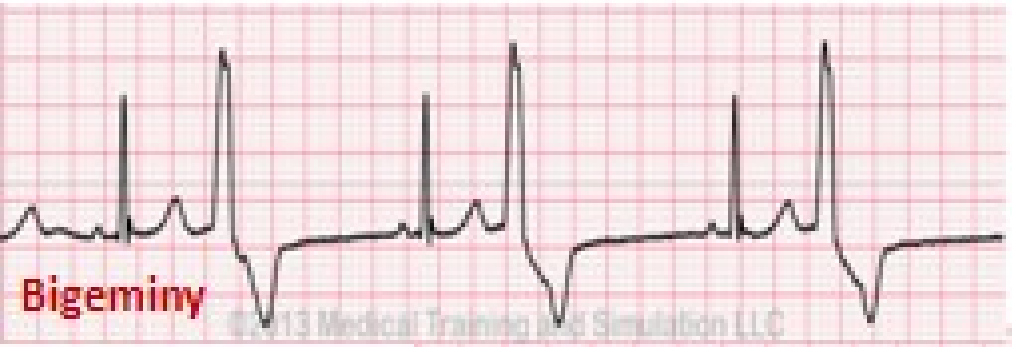
Triplet: three consecutive PVCs.

Patients are usually asymptomatic but may complain of an irregular heart beat, missed beats or abnormally strong beats.

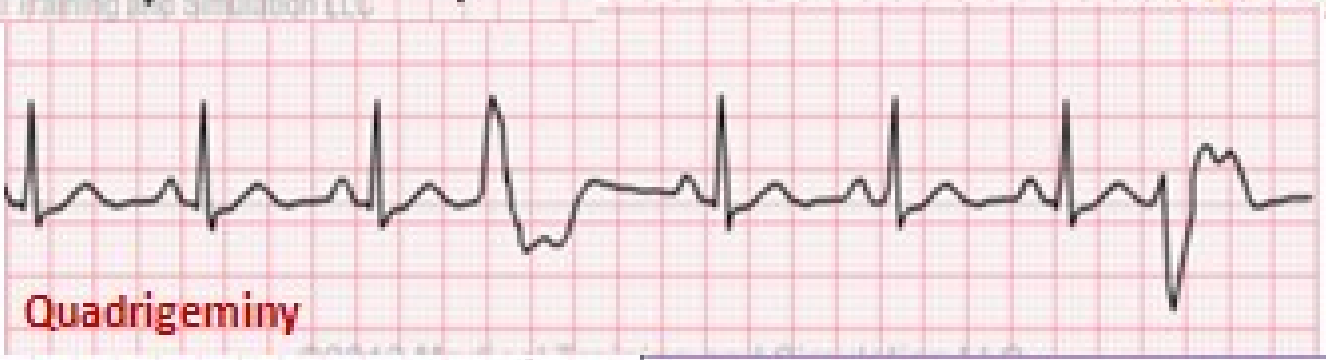
The significance of VEBs depends on the presence or absence of underlying heart disease.

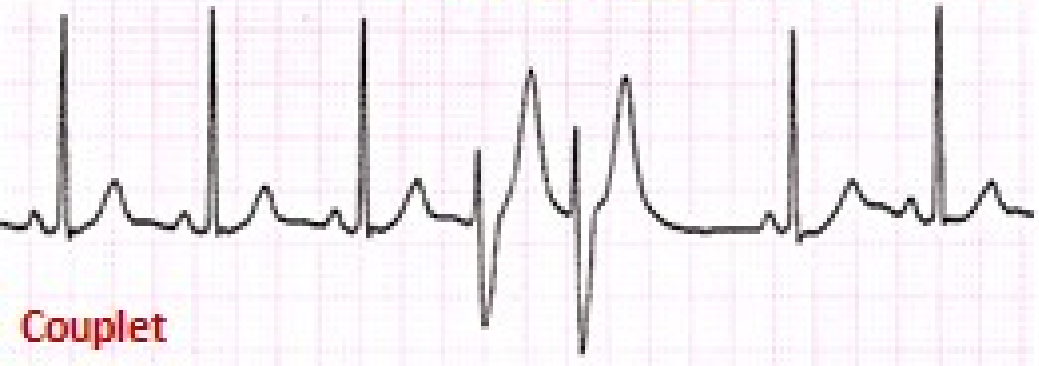

Unifocal

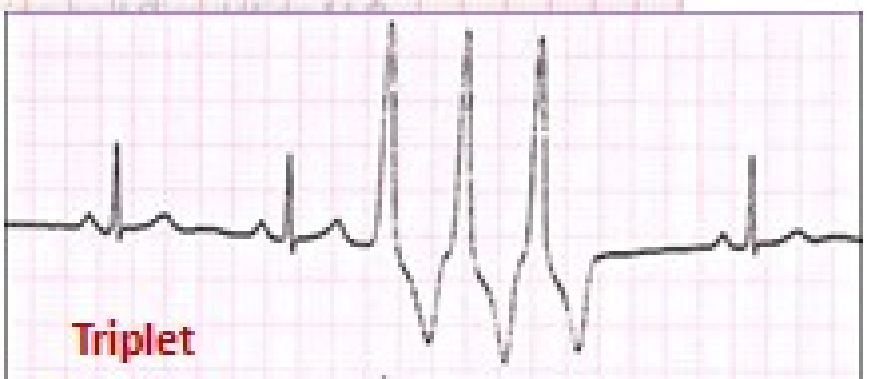

Multifocal

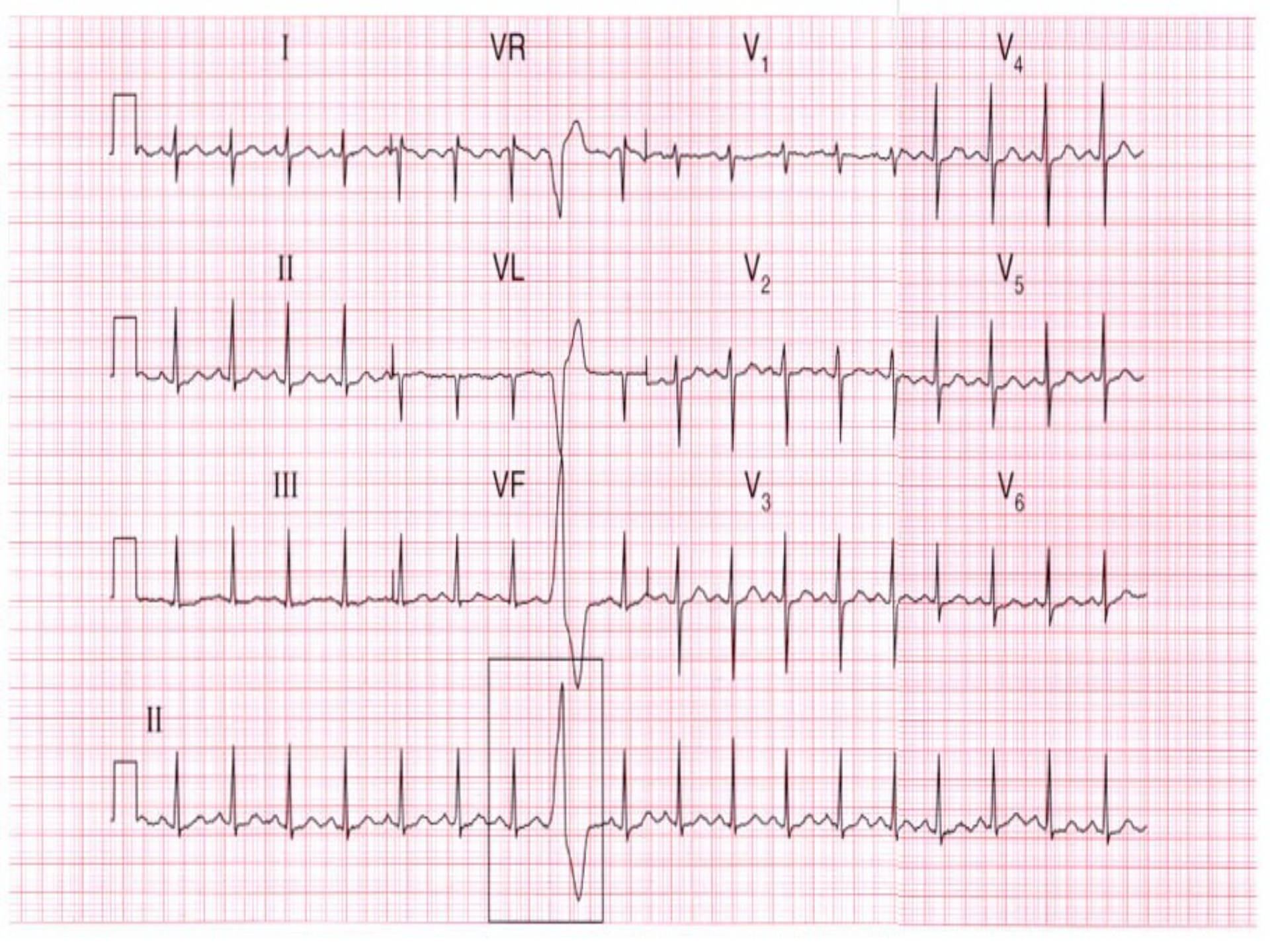

Bigeminy


Trigeminy


Quadrigeminy


Couplet


Triplet


I
VR
V1
V4
II
VL
V2
V5
III
VF
V3
V6
II

- PVC in normal heart treatment is not necessary, unless the patient is highly symptomatic, in which case β-blockers or, in some situations, catheter ablation can be used.
- Effective treatment of underlying conditions such as heart failure, coronary artery disease, cardiomyopathy or digoxin toxicity may suppress the ectopic beats.
- PVC with heart disease: Other than β-blockers, anti-arrhythmic drugs do not improve and may even worsen prognosis.

Ventricular tachycardia (VT)

Ventricular Tachycardia (VT): is defined as three or more consecutive ventricular depolarizations occurring at a rate greater than 100 beats/minute, it is a broad complex tachycardia originating in the ventricles.

VT duration

- **Sustained= that lasts for more than 30 seconds or requires termination because of hemodynamic instability.**
- **Non-sustained= that lasts less than 30 seconds and is hemodynamically stable and terminating spontaneously.**

Mechanism of VT: is due to re-entry within scarred ventricular tissue (the commonest mechanism) or abnormal automaticity or triggered activity in ischaemic tissue

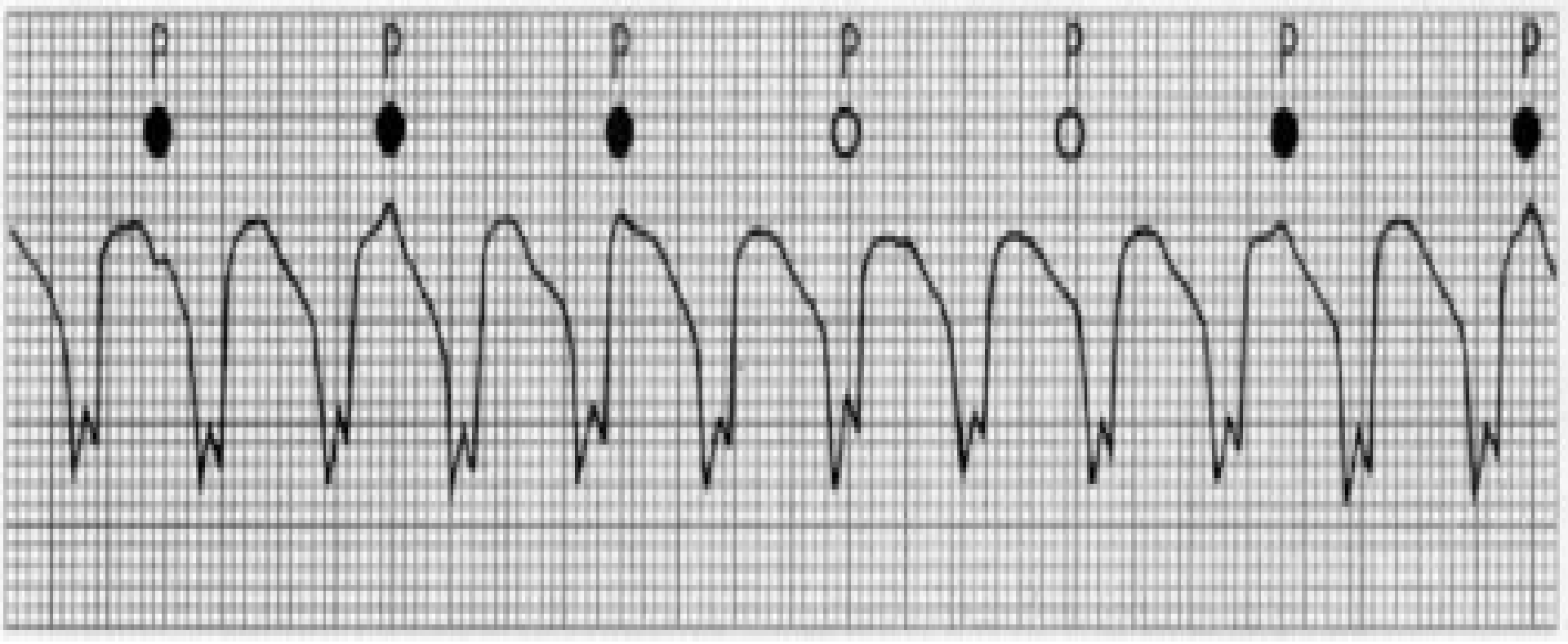
Causes of VT: acute ischemia, prior infarction with scar formation, congestive cardiomyopathy, right ventricular dysplasia, and hypertrophic heart disease. Metabolic abnormalities, such hypokalaemia, hypomagnesaemia, acidosis and hypoxaemia, and medications such as digoxin.

Clinical features: Patients may complain of palpitation or symptoms of low cardiac output, e.g. dizziness, dyspnoea or syncope. So patients clinically present as:

- Haemodynamically stable.
- Haemodynamically unstable — e.g hypotension, chest pain, cardiac failure, decreased conscious level.

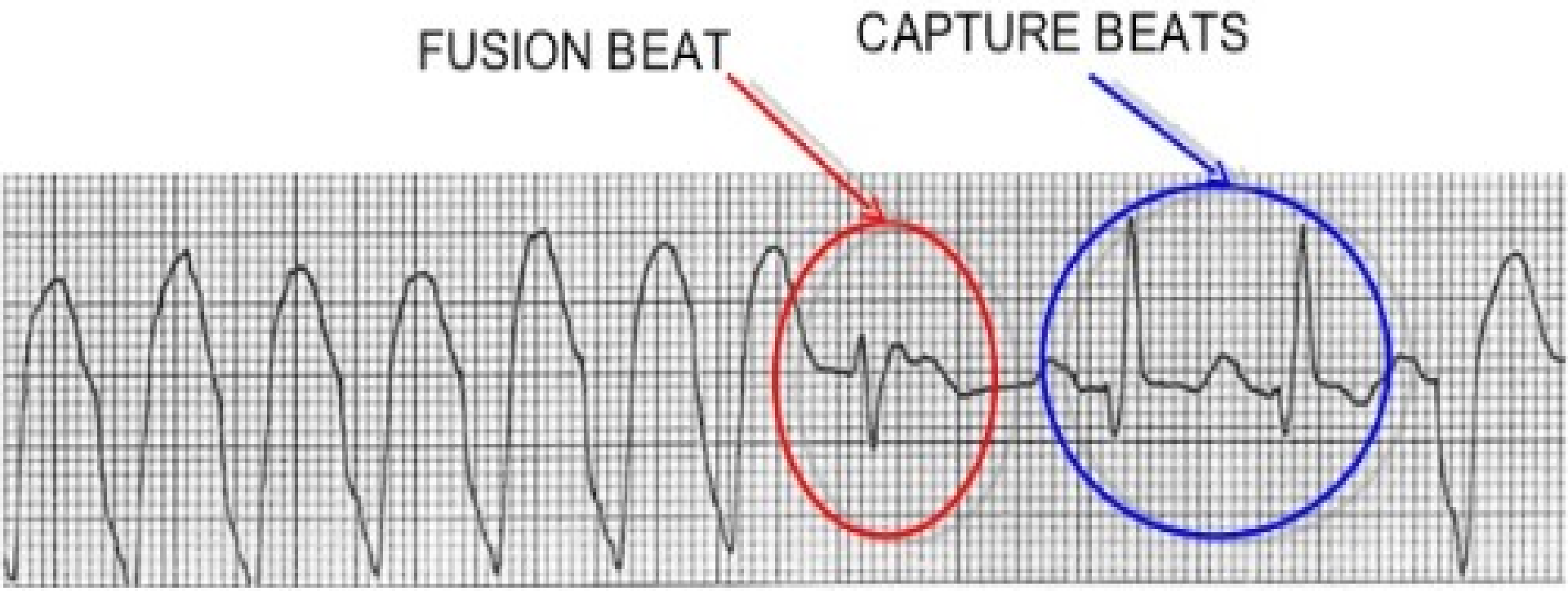
Features of ventricular tachycardia

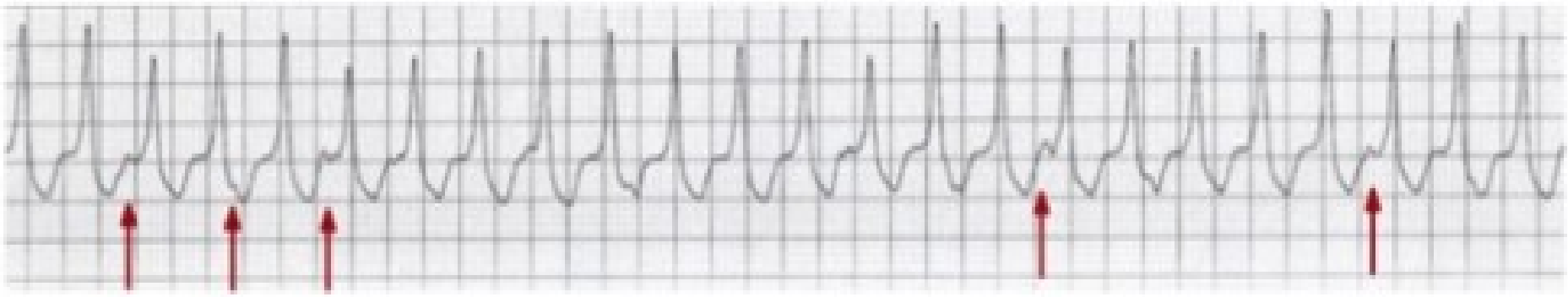
1. History of myocardial infarction
2. Very broad QRS complexes (> 140 ms), regular tachycardia (> 100 bpm).
3. AV dissociation: independent atrial and ventricular activity. P and QRS complexes at different rates (pathognomonic).
4. Capture beat: occur when the sinoatrial node transiently 'captures' the ventricles, which produce a normal QRS complex inside the run of VT (pathognomonic).
5. Fusion beat: occur when a sinus and ventricular beat coincide to produce a hybrid complex of intermediate morphology (pathognomonic).
6. Extreme axis deviation
7. No response to carotid sinus massage or IV adenosine



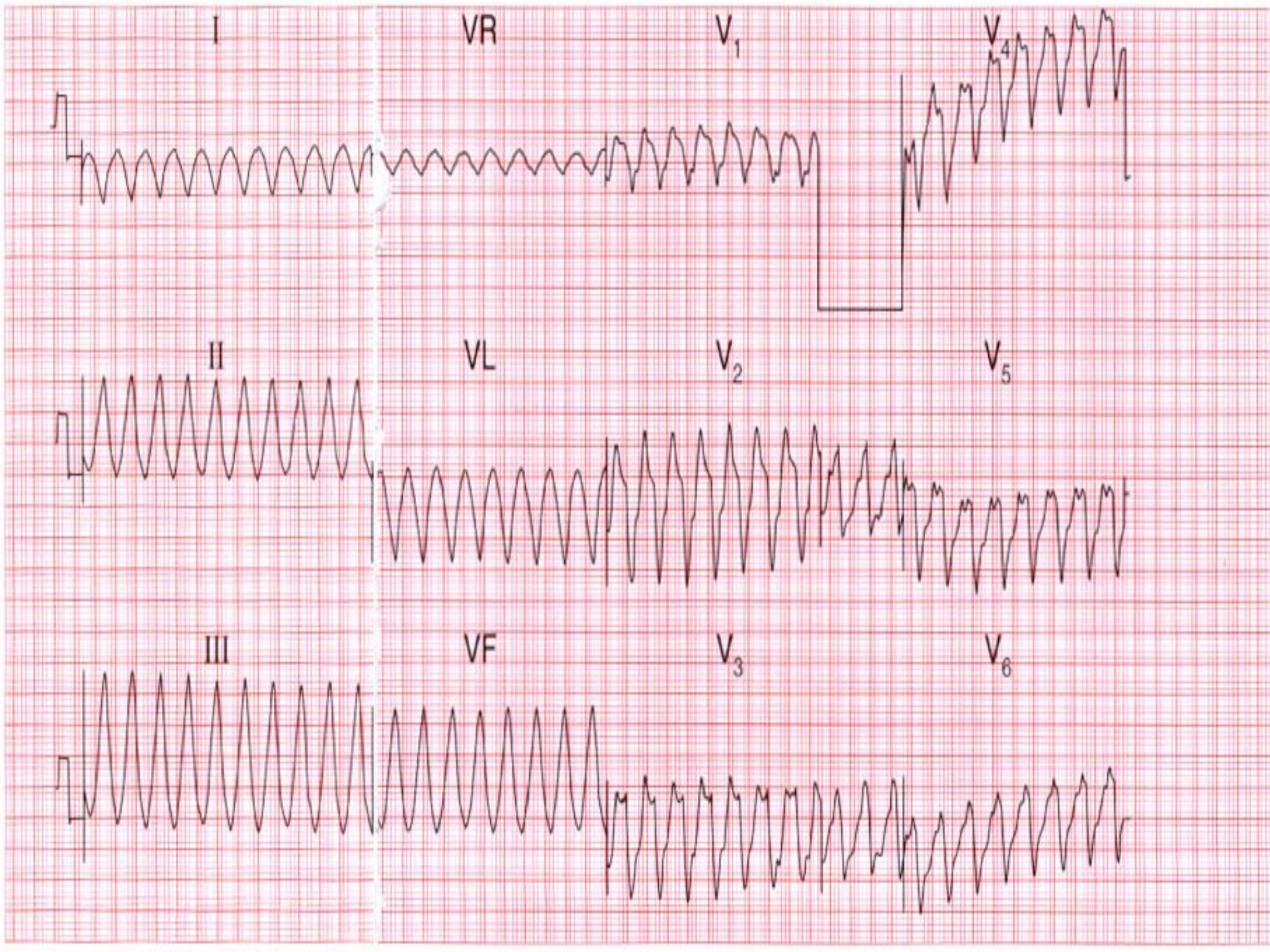
VT: There are typically very broad QRS complexes. There is also AV dissociation; some P waves are visible and others are buried in the QRS complexes

ECG Findings Suggestive of VT





AV DISSOCIATION


I
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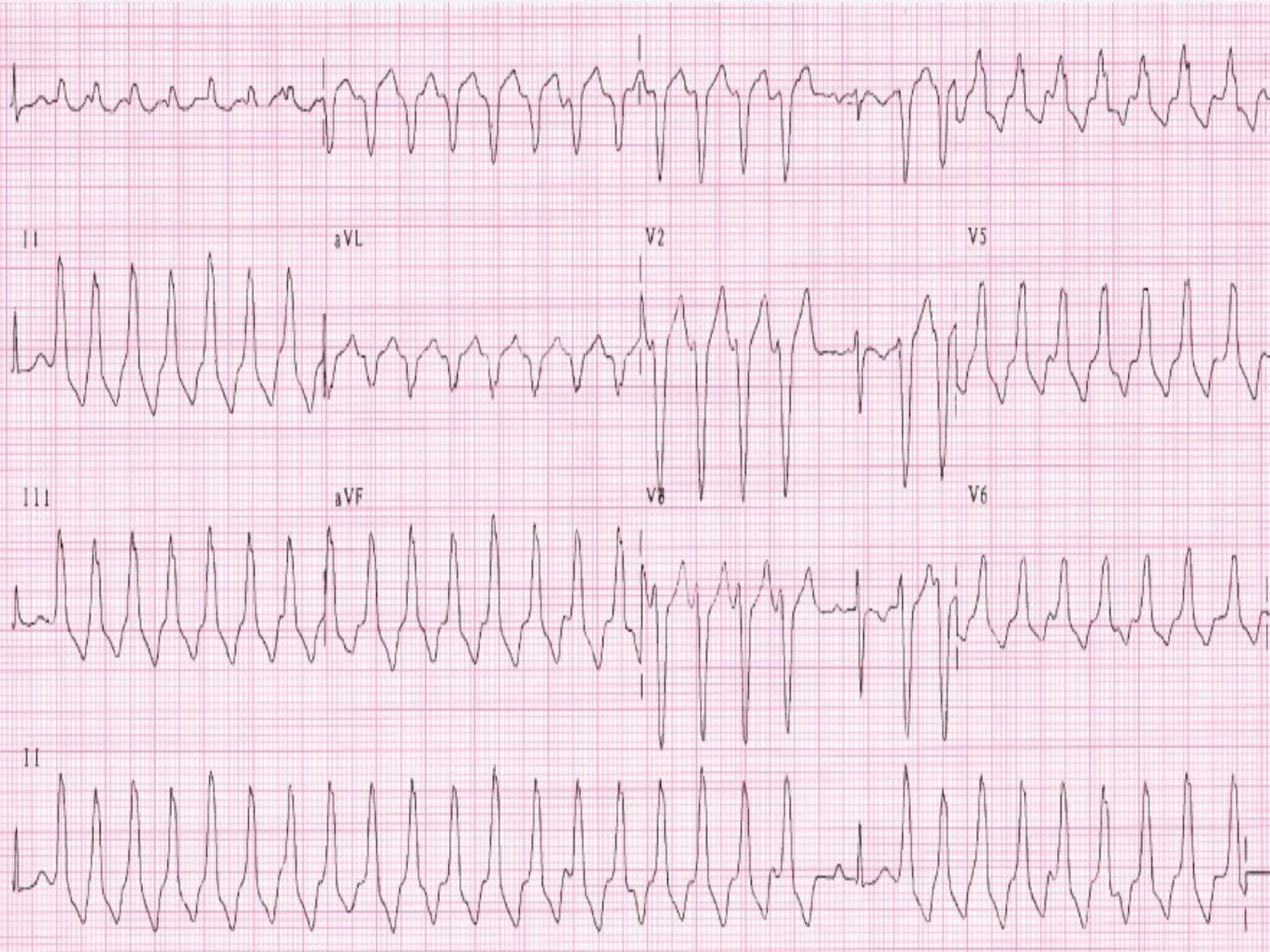

I
II
aVL
V2
V5
III
aVF
V3
V6
II

Management:

Synchronised DC cardioversion is the treatment of choice if systolic BP is less than 90 mmHg.

If the arrhythmia is well tolerated, intravenous amiodarone may be given as a bolus, followed by a continuous infusion. Intravenous lidocaine can be used but may depress left ventricular function, causing hypotension or acute heart failure.

Hypokalaemia, hypomagnesaemia, acidosis and hypoxaemia should be corrected.

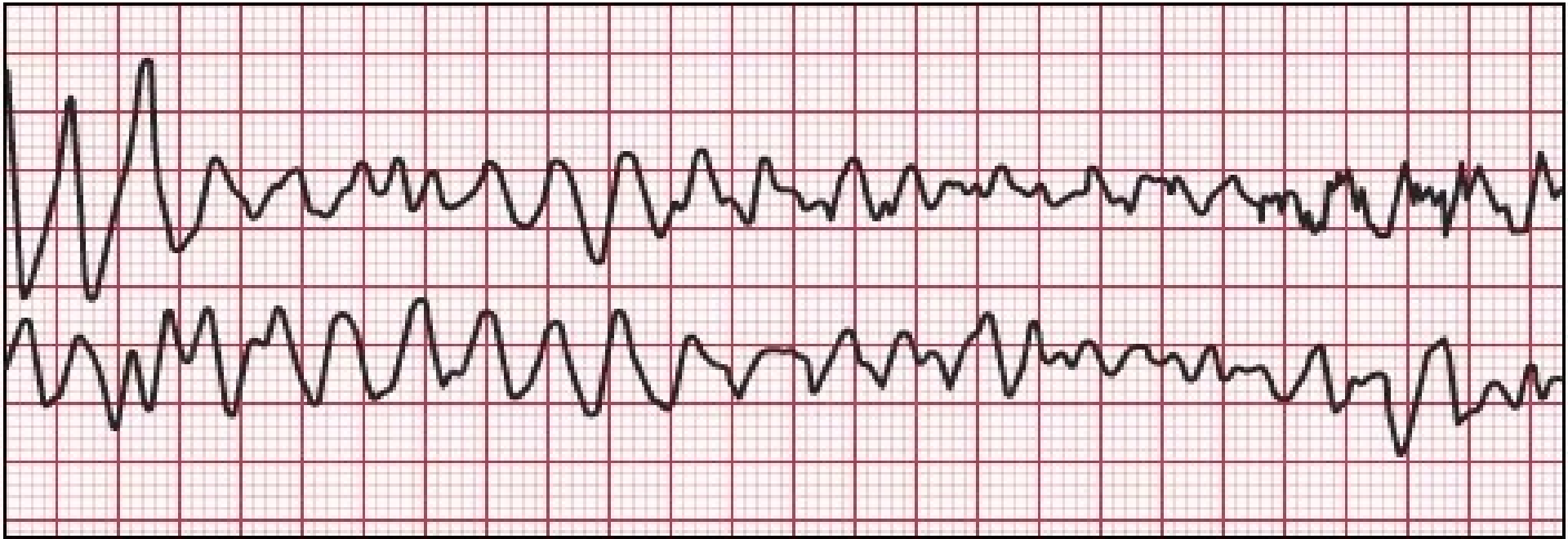
Prevention of VT by: Beta-blockers, amiodarone or implantable cardiac defibrillator (high risk of arrhythmic death).

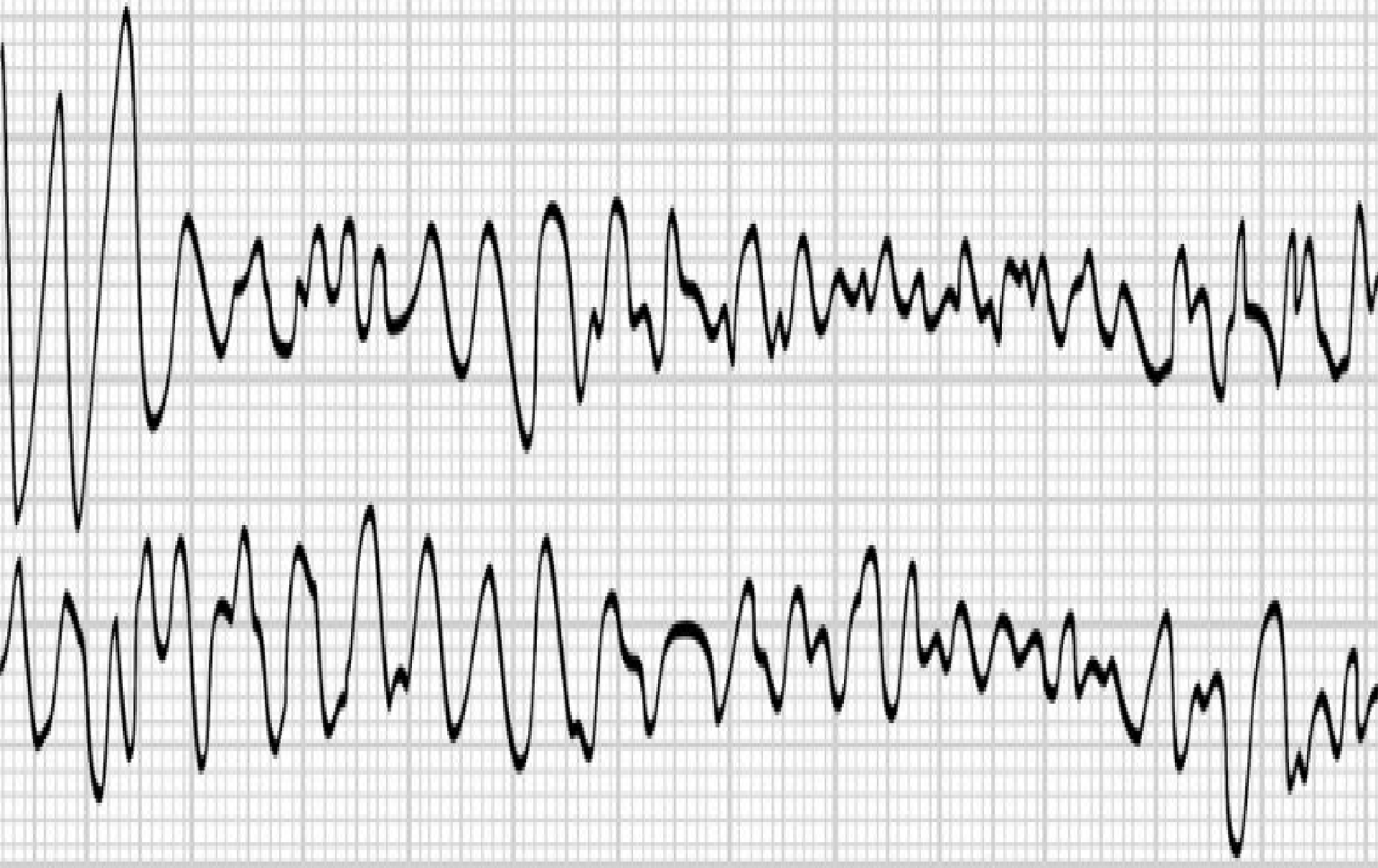
Surgery: rarely indicated e.g. aneurysm resection or catheter ablation of focus or circuit.

Ventricular fibrillation (VF)

VF: is the sudden, rapid and uncoordinated movement of the ventricles, resulting in immediate loss of cardiac output. Which is the most common and most easily treatable cause of cardiac arrest.

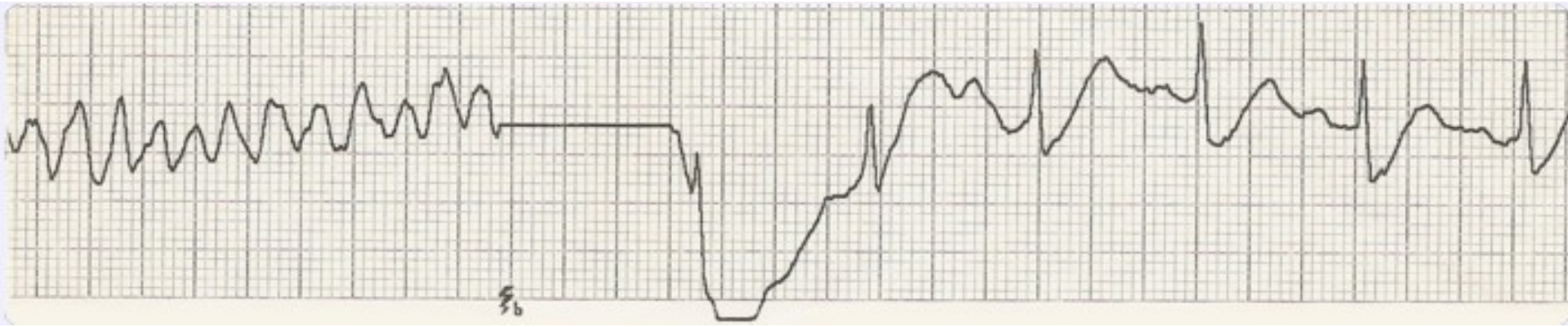
ECG Findings: Rapid, chaotic, irregular deflections of varying amplitude. No identifiable P waves, QRS complexes, or T waves.





Causes: Coronary artery disease (CAD) is the single most common etiologic factor predisposing patients to VF. Other causes are cardiomyopathy, valvular lesions like aortic stenosis, congenital heart disease like TOF, long QT syndrome and idiopathic VF.

Unsynchronized electrical (DC) shock with at least 200 to 300 J, implemented as rapidly as possible, this will restore cardiac output in more than 80% of patients, if delivered immediately.



cardiac arrest due to ventricular fibrillation which resuscitated by emergency DC shock

Thanks