Cardiac arrhythmia

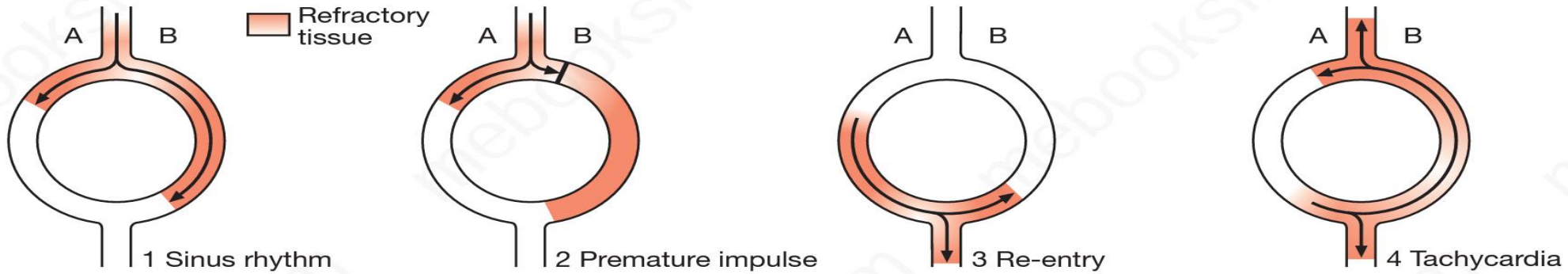
A cardiac arrhythmia is defined as a disturbance of the electrical rhythm of the heart. Cardiac arrhythmias are often a manifestation of structural heart disease but may also occur because of abnormal conduction or depolarisation in an otherwise healthy heart.

Pathogenesis

There are three main mechanisms of tachycardia:

1. Increased automaticity. The tachycardia is produced by spontaneous depolarisation of an ectopic focus in the atria, atrioventricular junction or ventricles, often in response to catecholamines. Single depolarisations lead to atrial, junctional or ventricular premature (ectopic) beats. Repeated depolarisation leads to atrial, junctional or ventricular tachycardia.

2. Re-entry. The tachycardia is initiated by an ectopic beat and sustained by a re-entry circuit (Fig. 16.31). Most tachyarrhythmias are caused by 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. Arrhythmias may be supraventricular (sinus, atrial , junctional) or ventricular.

Supraventricular rhythms usually produce narrow QRS complexes because the ventricles are depolarised in their normal sequence via the AV node, the bundle of His and associated Purkinje fibres.

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

Occasionally, supraventricular tachycardia can mimic ventricular tachycardia and present as a broad-complex tachycardia due to coexisting bundle branch block or the presence of an additional atrioventricular connection (accessory pathway

If the sinus rate becomes unduly slow, another, more distal part of the conducting system may assume the role of pacemaker. This is known as an escape rhythm and may arise in the AV node or His bundle (junctional rhythm) or in the ventricles (idioventricular rhythm).

Clinical features

Asymptomatic, sustained tachycardia might cause

Palpitation, Dizziness, Chest discomfort, Breathlessness, Syncope

Bradycardias tend to cause symptoms that reflect low cardiac output, including fatigue, lightheadedness and syncope. Extreme bradycardias or tachycardias can precipitate sudden death or cardiac arrest

Investigation

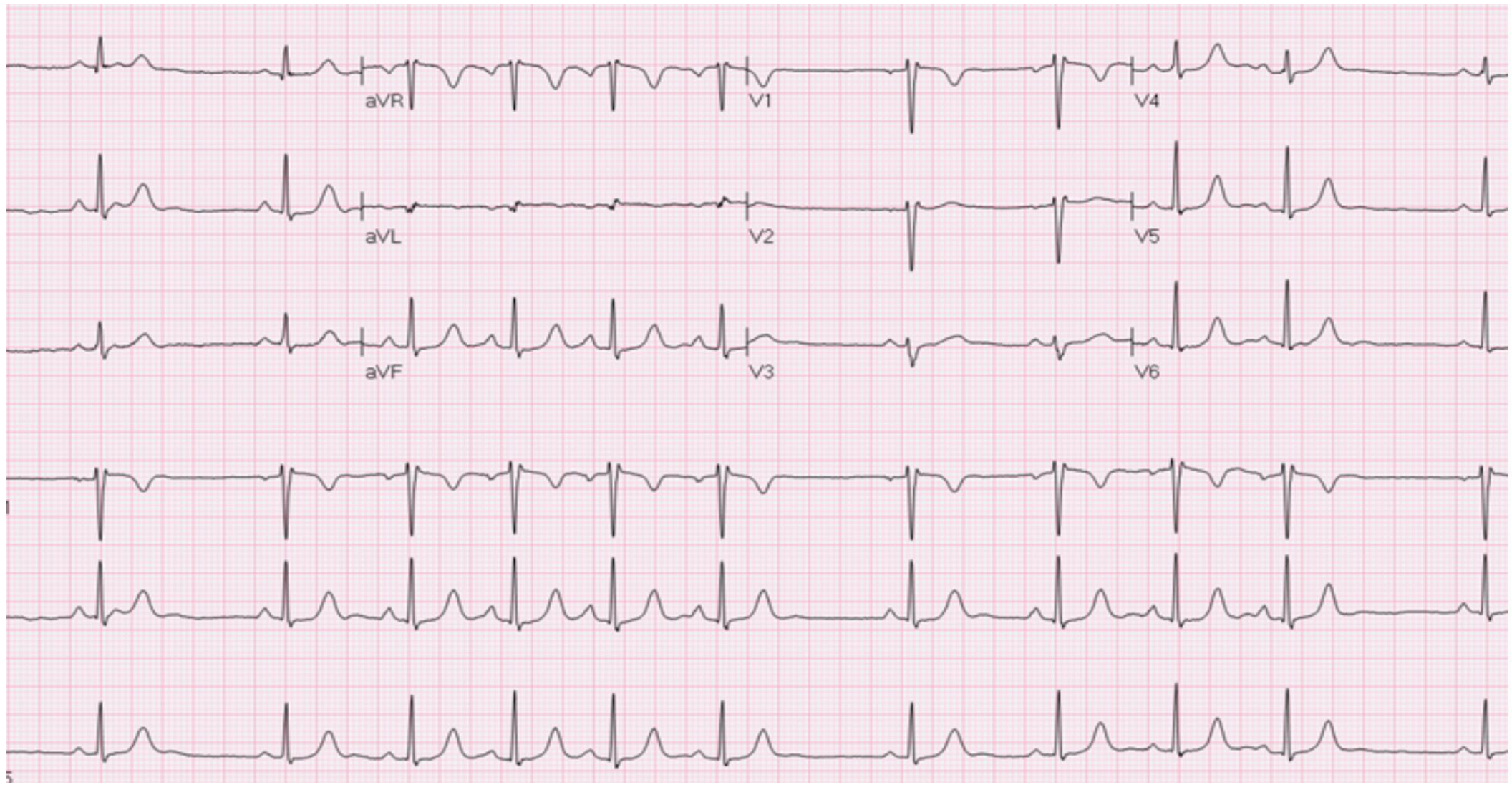
- ❑ *Standard 12 leads ECG*
- ❑ *Ambulatory ECG*
- ❑ *Pateint activated loop recorder*

Sinus arrhythmia

defined as a cyclical alteration of the heart rate during respiration, with an increase during inspiration and a decrease during expiration. Sinus arrhythmia is a normal phenomenon and can be quite pronounced in children.

Absence of this normal variation in heart rate with breathing or with changes in posture may be a feature of diabetic neuropathy, autonomic involvement in patients with diseases of peripheral nerves or increase sympathetic drive.

Need no treatment.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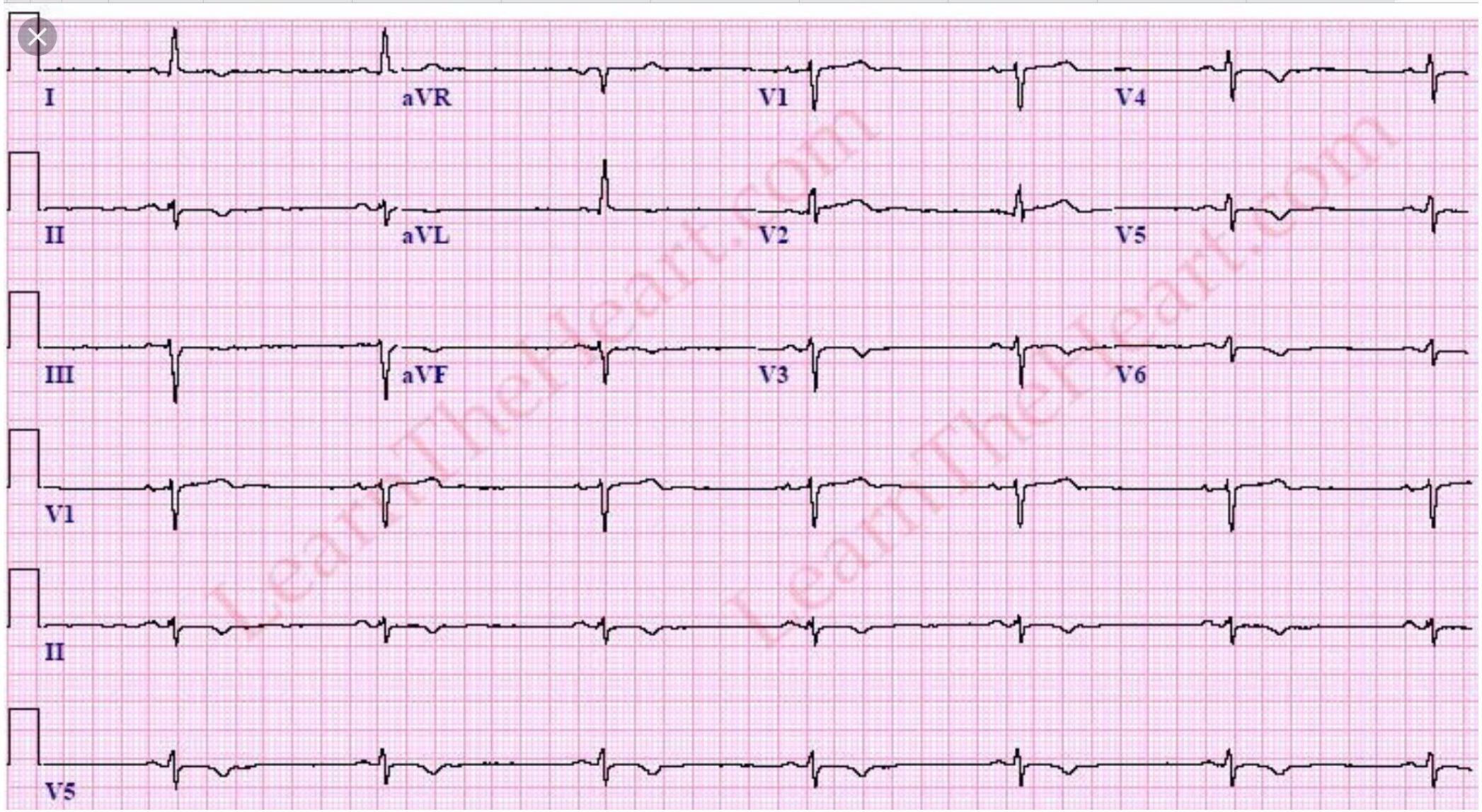
V3
V6

Sinus bradycardia

HR less than 60 beat per minute. May occur in normal people, a common finding in athlets.

Asymptomatic bradycardia need no treatment

Treatment: atropine , pacing (temporary or perminant)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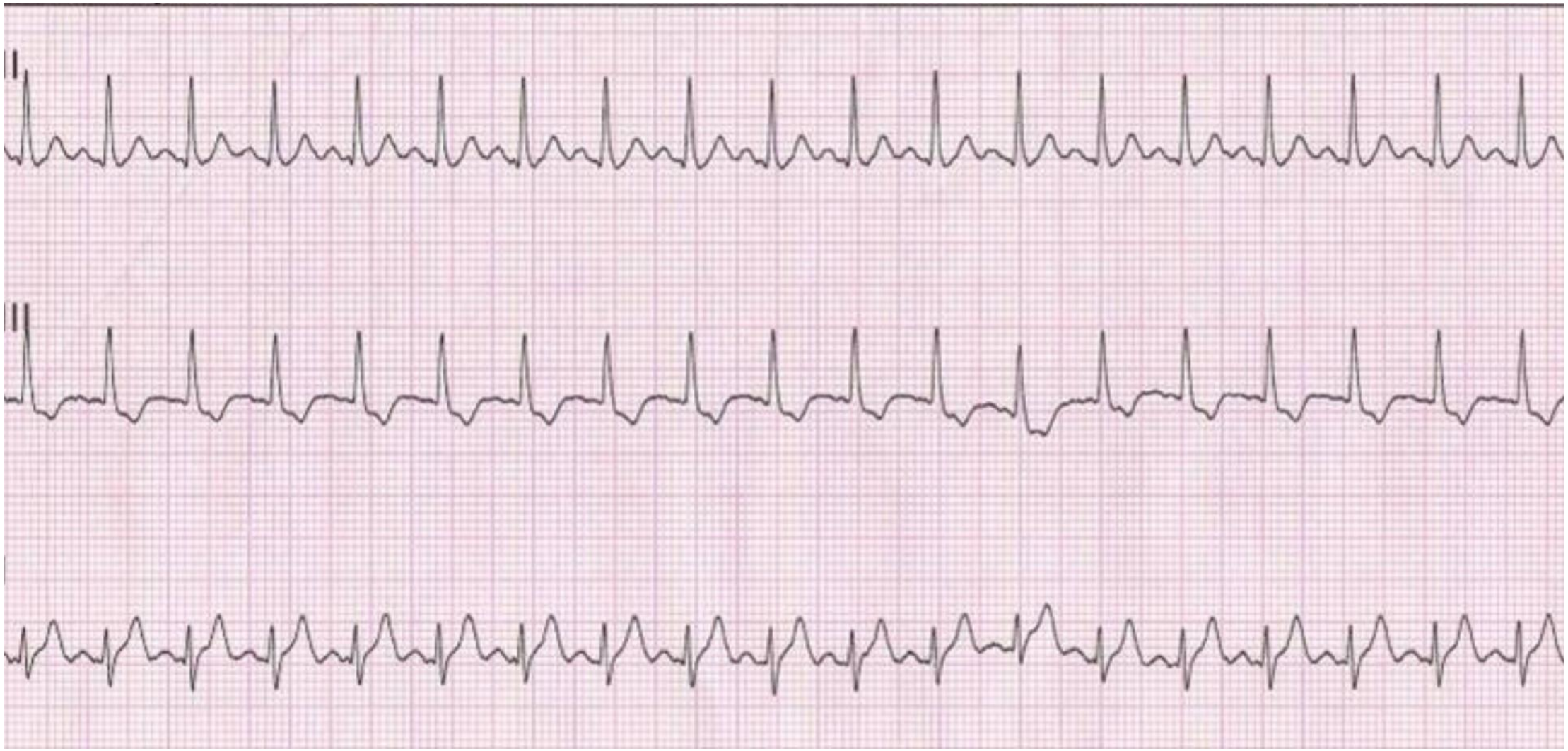
Sinus tachycardia

Sinus tachycardia is usually due to an increase in sympathetic activity associated with exercise, emotion and pregnancy. Healthy young adults can produce a rapid sinus rate, up to 200/min, during intense exercise. Sinus tachycardia does not require treatment but sometimes may reflect an underlying disease.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16.19 Some pathological causes of sinus bradycardia and tachycardia

Sinus bradycardia

- Myocardial infarction
- Sinus node disease (sick sinus syndrome)
- Hypothermia
- Hypothyroidism
- Cholestatic jaundice
- Raised intracranial pressure
- Drugs (β-blockers, digoxin, verapamil)

Sinus tachycardia

- Anxiety
- Fever
- Anaemia
- Heart failure
- Thyrotoxicosis
- Phaeochromocytoma
- Drugs (β-agonists)

Sinus node dysfunction

Caused by ischemia, fibrosis and degenerative changes of SA node.

Typical presentation is with palpitation, dizzy spells or syncope, due to intermittent tachycardia, bradycardia, or pauses with no atrial or ventricular activity (SA block or sinus arrest)

A permanent pacemaker may benefit patients with troublesome symptoms due to spontaneous bradycardias, or those with symptomatic bradycardias induced by drug required to prevent tachyarrhythmias. Atrial pacing may prevent episodes of atrial fibrillation. Pacing improves symptoms but not prognosis, and is not indicated in patients who are asymptomatic.

16.20 Common features of sinoatrial disease

- Sinus bradycardia
- Sinoatrial block (sinus arrest)
- Paroxysmal atrial fibrillation
- Paroxysmal atrial tachycardia
- Atrioventricular block

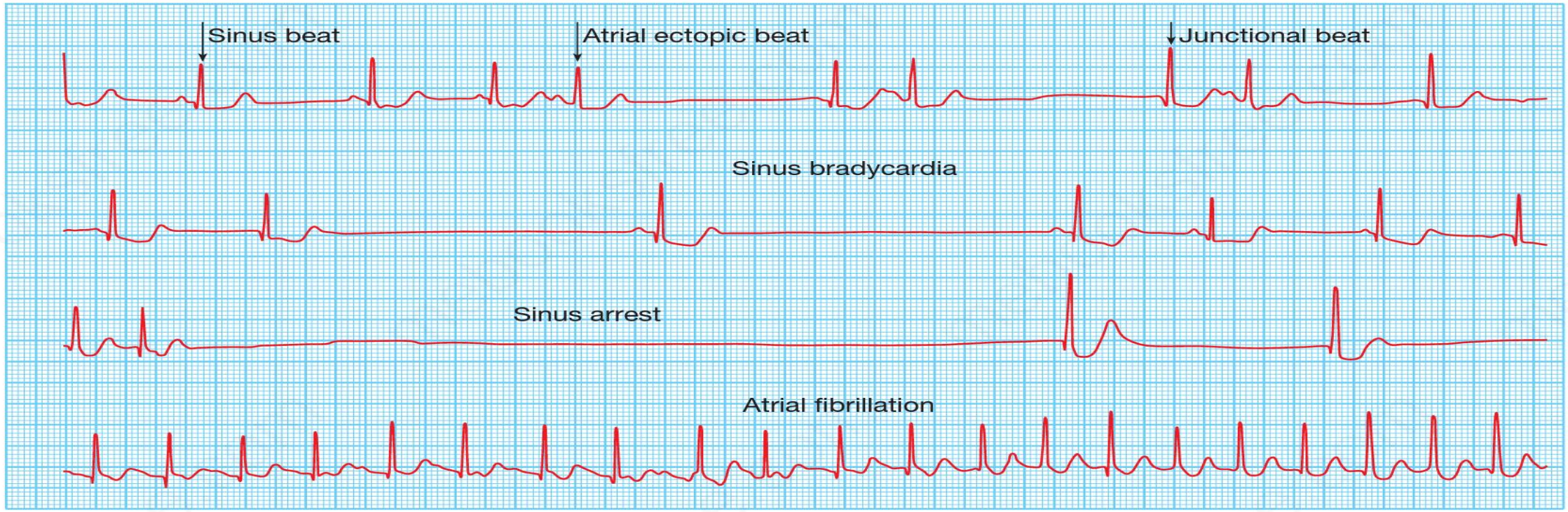


Fig. 16.32 Sinoatrial disease (sick sinus syndrome). A continuous rhythm strip from a 24-hour ECG tape recording illustrating periods of sinus rhythm, atrial ectopics, junctional beats, sinus bradycardia, sinus arrest and paroxysmal atrial fibrillation.

Premature atrial beat (atrial ectopic)

- ❑ Usually asymptomatic, or may give sensation of missed beat or abnormally strong beat
- ❑ ECG show premature but otherwise normal QRS complex , p wave if visible has abnormal morphology because the atria activate from abnormal site.
- ❑ Usually need no treatment, however frequent atrial e topics may herald the onset of atrial fibrillation
- ❑ Beta blocker can be used if symptoms is significant.

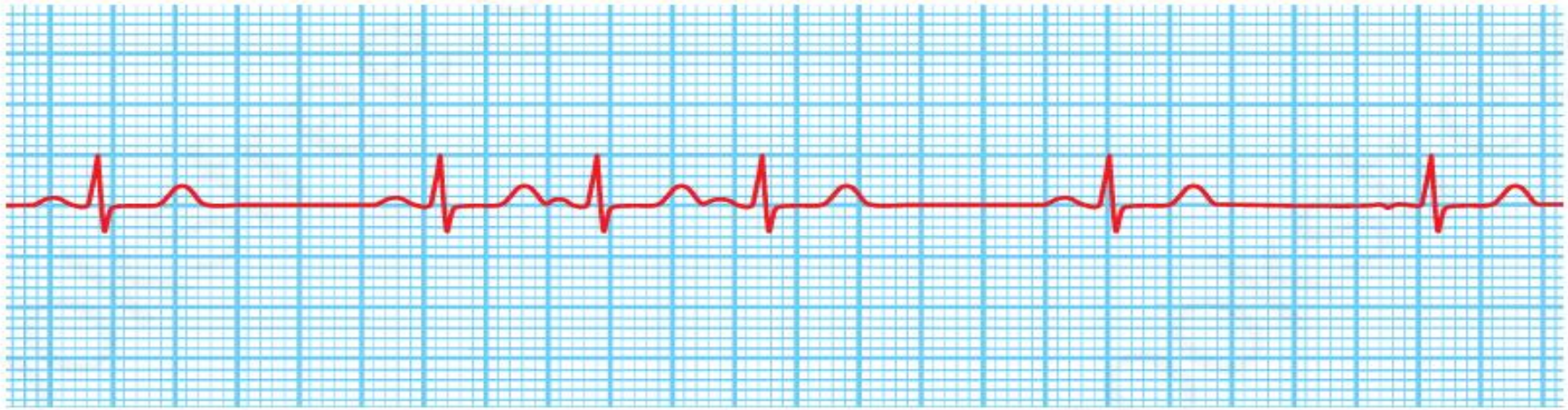


Fig. 16.33 Atrial ectopic beats. The first, second and fifth complexes are normal sinus beats. The third, fourth and sixth complexes are atrial ectopic beats with identical QRS complexes and abnormal (sometimes barely visible) P waves.

Atrial fibrillation

Atrial fibrillation (AF) is the most common sustained cardiac arrhythmia, with an overall prevalence of 0.5% in the adult population of the UK.

The prevalence rises with age, affecting 1% of those aged 60–64 years, increasing to 9% of those aged over 80 years. It is associated with significant morbidity and a twofold increase in mortality. This is mainly because of its association with underlying heart disease but also because of its association with systemic embolism and stroke

Classification

- ❑Paraxysmal: intermittent episode of AF that self terminated in 7 days
- ❑Persistent: prolong episode that can be terminated by electrical or pharmacological cardioversion.
- ❑ Perminant.

Other classifcation is:

- ❑ valvular AF in prosthetc valves, mitral stenosis, mitral annuloplasty surgery.
- ❑Non valvular , other causes

- ❑ Valvular AF has 10 fold risk of thromboembolism than non valvular AF

Pathogenesis

AF is a complex arrhythmia characterised by both abnormal automatic firing and the presence of multiple interacting re-entry circuits looping around the atria. Episodes of AF are initiated by rapid bursts of ectopic beats arising from conducting tissue in the pulmonary veins or from diseased atrial tissue. It becomes sustained because of re-entrant conduction within the atria or sometimes because of continuous ectopic firing.

LA size correlate with the risk of AF

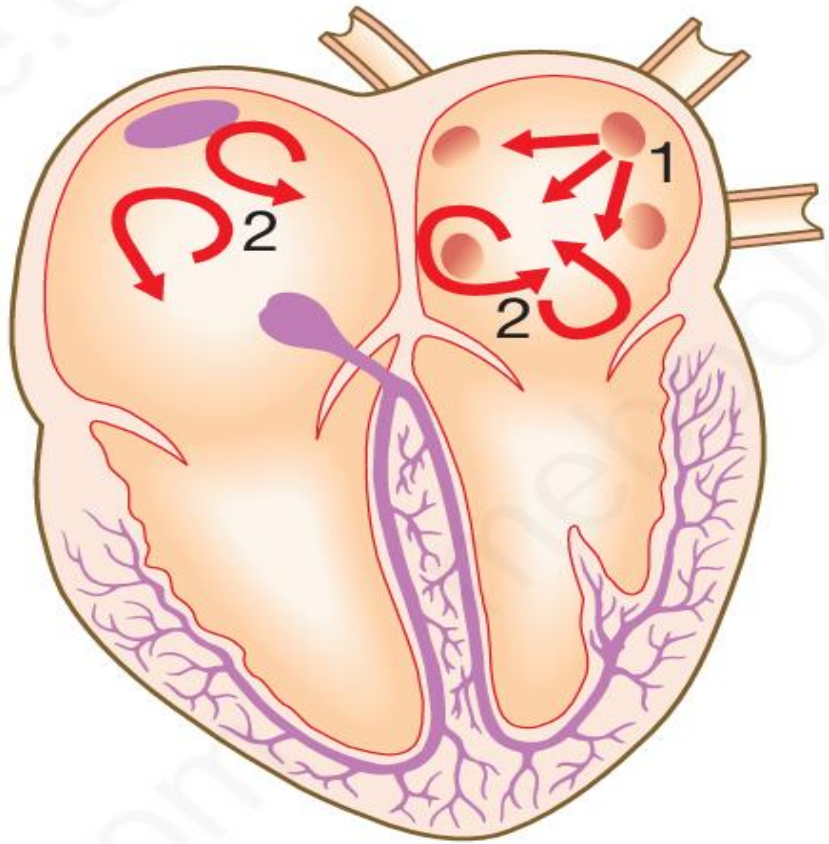


Fig. 16.36 Mechanisms initiating atrial fibrillation. (1) Ectopic beats, often arising from the pulmonary veins, trigger atrial fibrillation. (2) Re-entry within the atria maintains atrial fibrillation, with multiple interacting re-entry circuits operating simultaneously.

During episodes of AF, the atria beat rapidly but in an uncoordinated and ineffective manner. The ventricles are activated irregularly at a rate determined by conduction through the AV node. This produces the characteristic 'irregularly irregular' pulse. The ECG shows normal but irregular QRS complexes; there are no P waves but the baseline may show irregular fibrillation waves.

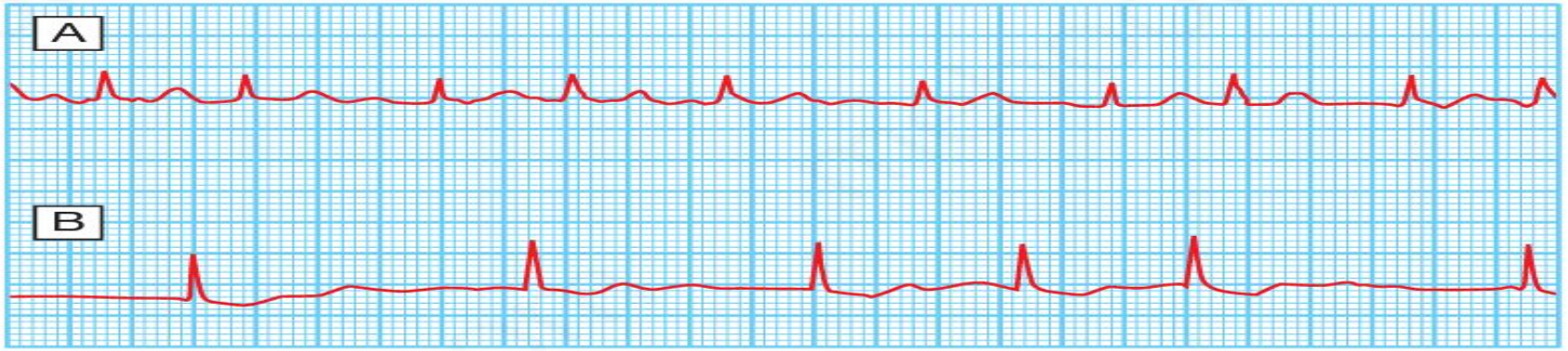


Fig. 16.37 Two examples of atrial fibrillation. The QRS complexes are irregular and there are no P waves. **A** There is usually a fast ventricular rate, between 120 and 160/min, at the onset of atrial fibrillation. **B** In chronic atrial fibrillation, the ventricular rate may be much slower, due to the effects of medication and AV nodal fatigue.

16.21 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)

Alcohol excess, hyperthyroidism and chronic lung disease are also common causes of AF, although multiple predisposing factors may coexist, such as the combination of alcohol, hypertension and coronary artery disease.

*About 50% of all patients with paroxysmal AF and 20% of patients with persistent or permanent AF have structurally normal hearts; this is known as '**lone** atrial fibrillation'.*

Clinical features

The typical presentation is with palpitation, breathlessness and fatigue.

In patients with poor ventricular function or valve disease, AF may precipitate or aggravate cardiac failure due to loss of atrial contraction and the rapid ventricular response.

In elderly AF may be asymptomatic and discovered incidently.

Investigation

- ❑ *12 lead ECG*
- ❑ *Echocardiography*
- ❑ *TFT*
- ❑ *TMT & coronary angio if IHD is a possible cause*

Managment

- *Rate control*
- *Rhythm control*
- *Prevention of recurrence*
- *Prevention of thromboembolism*

Rate control

- ✓ *Beta blocker*
- ✓ *Calcium channel blocker*
- ✓ *Digoxin*
- ✓ *AV node ablation and perminant pacemaker implantation (pace and ablate strategy)*

- ✓ *Beta-blockers and rate-limiting calcium antagonists are more effective than digoxin at controlling the heart rate during exercise and have additional benefits in patients with hypertension or structural heart disease*

Rhythm control

- ✓ *Cardioversion is initially successful in most patients but relapse is frequent (25–50% at 1 month and 70–90% at 1 year). Attempts to restore and maintain sinus rhythm are most successful if AF has been present for less than 3 months, the patient is young and there is no important structural heart disease.*

- ✓ *Cardioversion is either pharmacological or electrical.*
- ✓ *Electrical cardioversion if there is hemodynamic instability or no response to pharmacological cardioversion*

- ✓ *If onset of AF less than 48 hours, cardioversion can be given and intravenous heparin should be given*

- ✓ *If onset is unknown or more than 48 hours, TEE should be done to R/O LAA thrombus, or anticoagulation with warfarin or NOAC for 4 weeks should be given before cardioversion.*

- ✓ *In stable patients with no history of structural heart disease, intravenous flecainide (2 mg/kg over 30 mins, maximum dose 150 mg) can be used for pharmacological cardioversion and will restore sinus rhythm in 75% of patients within 8 hours.*

- ✓ *In patients with structural heart disease intravenous amiodarone can be given.*

- ✓ *Pills in pocket regimen: flecainide 200 to 300 mg orally can be used to restore sinus rhythm in young patient without structural heart disease if proved to be safe and effective in hosoital to restore sinus rhythm.*

AF catheter ablation

- ✓ Pulmonary vein isolation

Prevention of recurrence

- Class Ic drugs such as propafenone or flecainide, are also effective at preventing episodes but should not be given to patients with coronary artery disease or left ventricular dysfunction.

- Flecainide is seldom used alone, since it can precipitate atrial flutter, and is usually prescribed with a rate-limiting β-blocker.

- Class III drugs can also be used; amiodarone is the most effective agent for preventing AF but side-effects restrict its use to when other measures fail.

- Dronedarone is an effective alternative but is contraindicated in patients with heart failure or significant left ventricular impairment.

Prevention of thrmboemblosim

- *Valvular Af , anticoagulation with warfarin is indicated*
- *Non valvular AF, CHADS VASC score is applied*

- *patients with intermittent AF, stroke risk is similar to that in patients with persistent AF when adjusted for CHA2DS2-VASc score. The risk of embolism is only weakly related to the frequency and duration of AF episodes, so stroke prevention guidelines do not distinguish between those with paroxysmal, persistent and permanent AF*

- The factor Xa inhibitors rivaroxaban, apixaban and edoxaban, and the direct thrombin inhibitor dabigatran (collectively reffeted to as directly acting oral anticoagulants, or DOACs), can be used as an alternative to warfarin in non valvular AF.

- They are at least as effective as warfarin at preventing thrombotic stroke and are associated with a lower risk of intracranial haemorrhage. Other advantages include the lack of requirement for monitoring and the fact that they have fewer drug interactions.

- Agents that reverse the effects of DOACs have been developed. These include **idarucizumab**, which binds to dabigatran and allows acute bleeding complications to be managed more effectively.

- Aspirin should not be used since it has little or no effect on embolic stroke and is associated with significant bleeding risk.

16.23 CHA_2DS_2-VASc stroke risk scoring system for non-valvular atrial fibrillation

	Parameter	Score
C	Congestive heart failure	1 point
H	Hypertension history	1 point
A_2	Age ≥75 years	2 points
D	Diabetes mellitus	1 point
S_2	Previous stroke or transient ischemic attack (TIA)	2 points
V	Vascular disease	1 point
A	Age 65–74 years	1 point
Sc	Sex category female	1 point
	Maximum total score	9 points

Annual stroke risk

0 points = 0% (no prophylaxis required)

1 point = 1.3% (oral anticoagulant recommended in males only)

2+ points = > 2.2% (oral anticoagulant recommended)

From European Society of Cardiology Clinical practice guidelines: atrial fibrillation (management of) 2010 and focused update (2012). Eur Heart J 2012; 33:2719–2747.

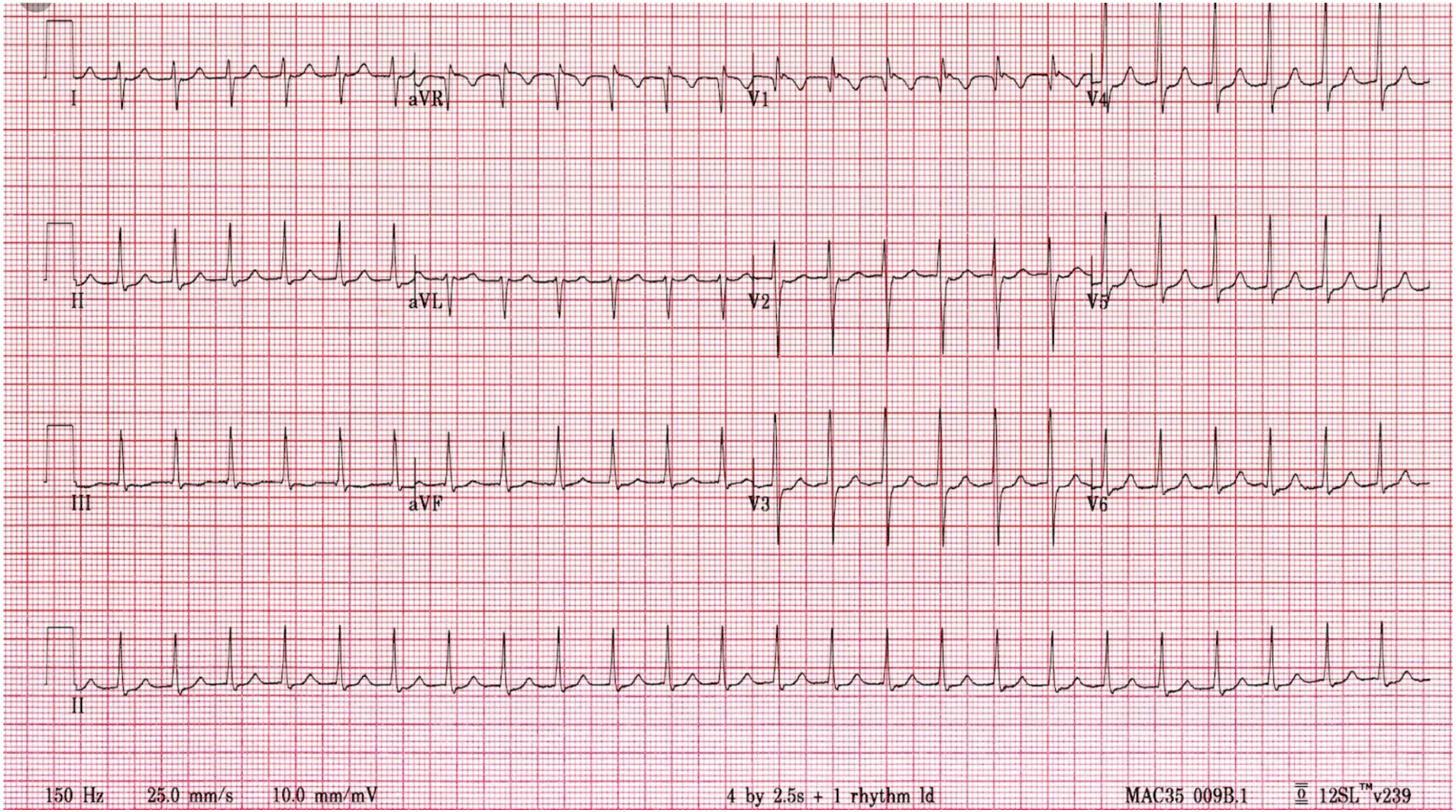
- LA appendage occluder (watchman occluder)

Supraventricular tachycardia

- *The three principal types are*

- *atrioventricular nodal re-entrant tachycardia (AVNRT)*
- *atrioventricular re-entrant tachycardia (AVRT)*
- *atrial tachycardia.*

- *The term SVT is technically incorrect as, in many cases, the ventricles also form part of the re-entry circuit.*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25.0 mm/s
10.0 mm/mV
4 by 2.5s + 1 rhythm ld
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Atrioventricular nodal re-entrant tachycardia

- *AVNRT is a type of SVT caused by re-entry in a circuit involving the AV node and its two right atrial input pathways: a superior 'fast' pathway and an inferior 'slow' pathway.*

- *Normal heart arrythmia, occur in Patients with structurally normal heart.*

- *ECG : narrow complex, rate 120 to 240 bpm, rate dependent BBB may occur*

- *Present with palpitation lasting few seconds to hours*

Management of AVNRT

- For acute attack

- Carotid sinus massage
- Valsalva maneuver
- Intravenous adenosine
- Intrvenous verapamil
- Intrvenous beta blocker , intravenous flecainide or amiodarone

- For recurrent AVNRT, catheter ablation is indicated with success rate of more than 90 %

Atrioventricular re entry tavhycardia

- In this condition there is an abnormal band of conducting tissue that connects the atria and ventricles.

- This so-called accessory pathway comprises rapidly conducting fibres that resemble Purkinje tissue, in that they conduct very rapidly and are rich in sodium channels.

- In about 50% of cases, this pathway conducts only in the retrograde direction (from ventricles to atria) and thus does not alter the appearance of the ECG in sinus rhythm. This is known as a concealed accessory pathway.
- In the rest, the pathway also conducts in an antegrade direction (from atria to ventricles), so AV conduction in sinus rhythm is mediated via both the AV node and the accessory pathway, distorting the QRS complex.

- Premature ventricular activation through the accessory pathway shorten the PR interval and result in initial slurring of QRS called delta wave, this is called manifested pathway.

- Wpw pattern, WPW syndrome ?

- Accessory pathway and the AV node has different refractory period and conduction velocity lead to re entry circuit

Managment

- Narrow complex arrhythmia, retrograde conduction the accessiry pathway , i.v adenosine and carotid sinus massage

- Wide complex arrhythmia, antegrade conduction through AV node , class I c antiarrhythmic with beta blocker

- If AF occurs, it may produce a dangerously rapid ventricular rate because the accessory pathway lacks the rate-limiting properties of the AV node. This is known as pre-excited atrial fibrillation and may cause collapse, syncope and even death. It should be treated as an emergency, usually with DC cardioversion.

- Catheter ablation sucessful in majority of cases and it is curative treatment.

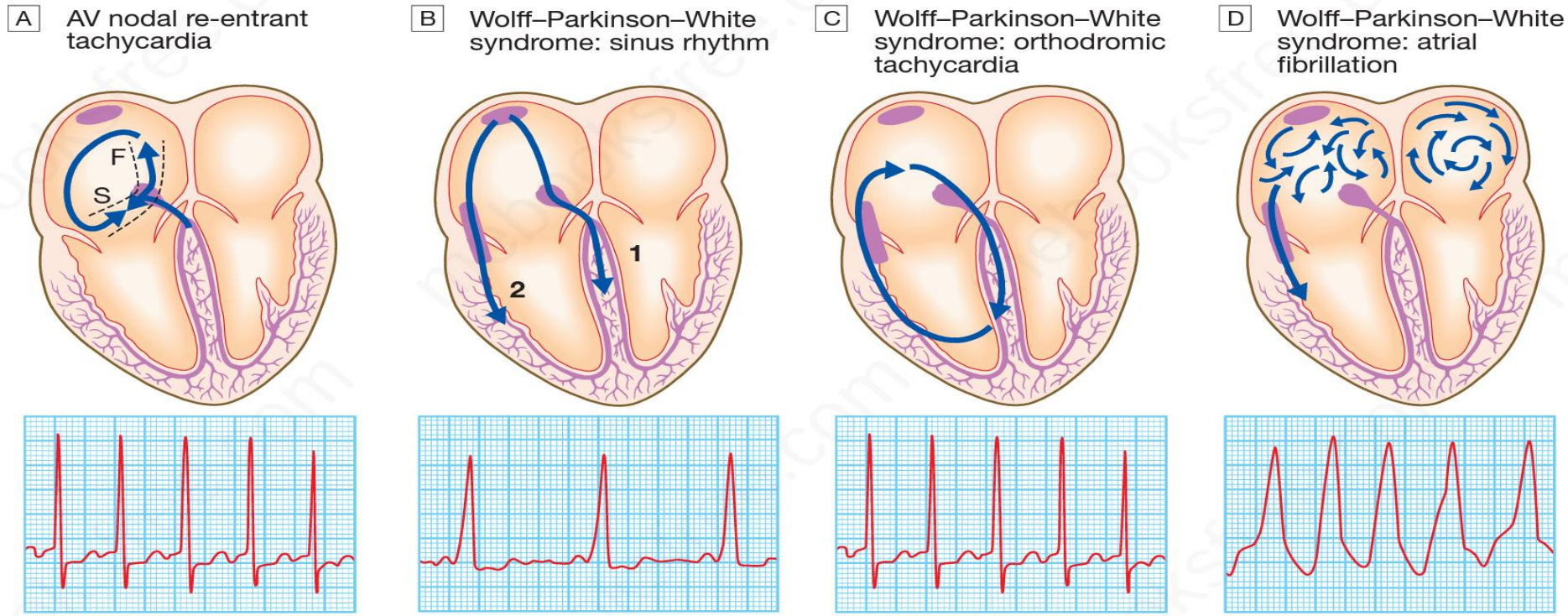
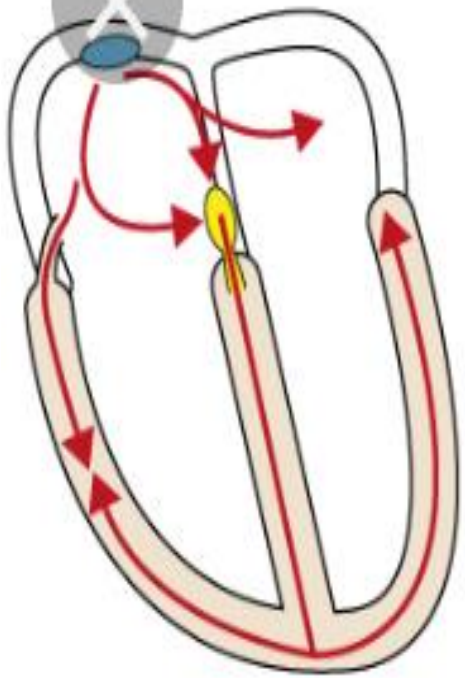


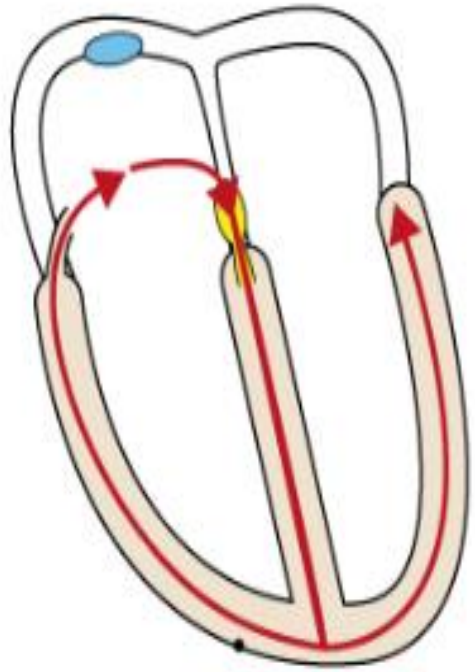
Fig. 16.39 Atrioventricular nodal re-entrant tachycardia (AVNRT) and Wolff–Parkinson–White (WPW) syndrome. **A** AV node re-entrant tachycardia. The mechanism of AVNRT occurs via two right atrial AV nodal input pathways: the slow (S) and fast (F) pathways. Antegrade conduction occurs via the slow pathway; the wavefront enters the AV node and passes into the ventricles, at the same time re-entering the atria via the fast pathway. In WPW syndrome, there is a strip of accessory conducting tissue that allows electricity to bypass the AV node and spread from the atria to the ventricles rapidly and without delay. When the ventricles are depolarised through the AV node the ECG is normal, but when the ventricles are depolarised through the accessory conducting tissue the ECG shows a very short PR interval and a broad QRS complex. **B** 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. The degree of pre-excitation (the proportion of activation passing down the accessory pathway) and therefore the ECG appearances may vary a lot, and at times the ECG can look normal. **C** 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 supraventricular tachycardia. **D** Atrial fibrillation. In this rhythm, the ventricles are largely depolarised through the accessory pathway, producing an irregular broad-complex tachycardia that is often more rapid than the example shown.

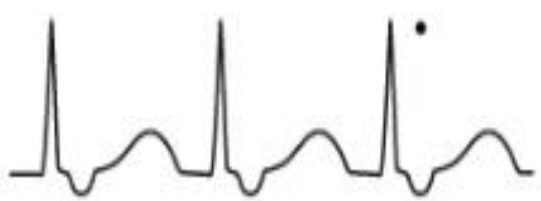
Preexcitation (WPW)
För tidig aktivering av kamrar



Kort PQ-tid (<0,12 s). Deltavåg.
I detta fall avbryts P-vågen av deltavågen.

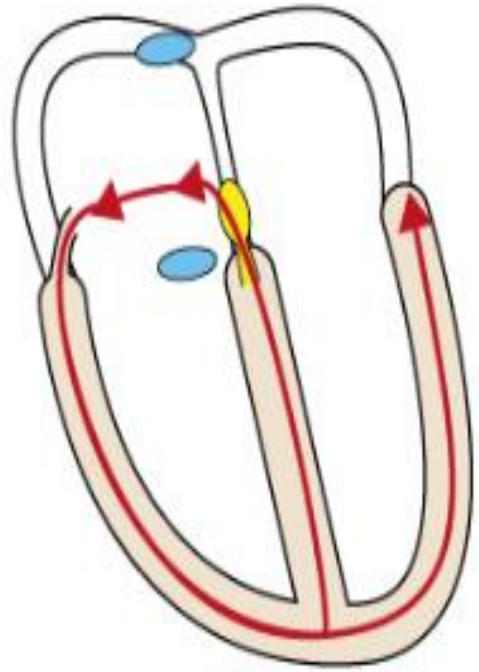
Ortodrom AVRT
Antegrad riktning genom AV-nod





Normal QRS
Retrograd P strax efter QRS

Antidrom AVRT
Retrograd riktning genom AV-nod



Abnormal QRS med deltavåg
Sällan ses P-våg men den är då retrograd och infaller innan QRS

Atrial flutter

- *Atrial flutter is characterised by a large (macro) re-entry circuit, usually within the right atrium encircling the tricuspid annulus.*
- *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).*
- *Rarely, in young patients, every flutter wave is conducted, producing a rate of 300/min and, potentially, haemodynamic compromise.*
- *The ECG shows saw-tooth flutter waves. When there is regular 2 : 1 AV block, it may be difficult to identify flutter waves that are buried in QRS complexes and T waves.*
- *Atrial flutter should always be suspected when there is a narrow-complex tachycardia of 150/min. Carotid sinus pressure or intravenous adenosine may help to establish the diagnosis by temporarily increasing the degree of AV block and revealing flutter waves.*

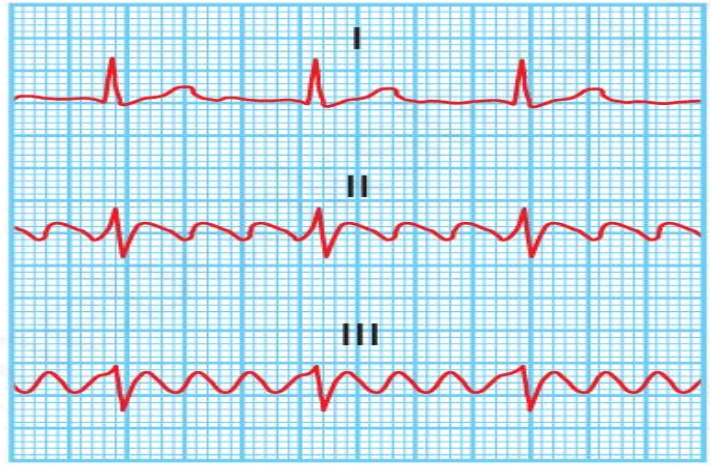


Fig. 16.34 Atrial flutter. Simultaneous recording showing atrial flutter with 4 : 1 atrioventricular block. Flutter waves are visible only in leads II and III.

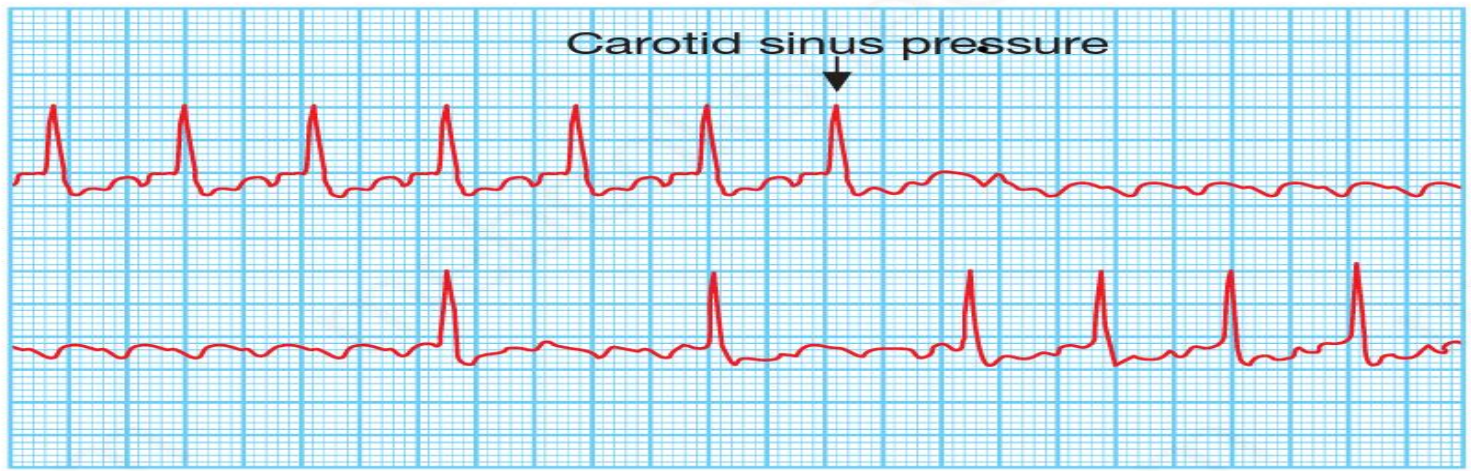
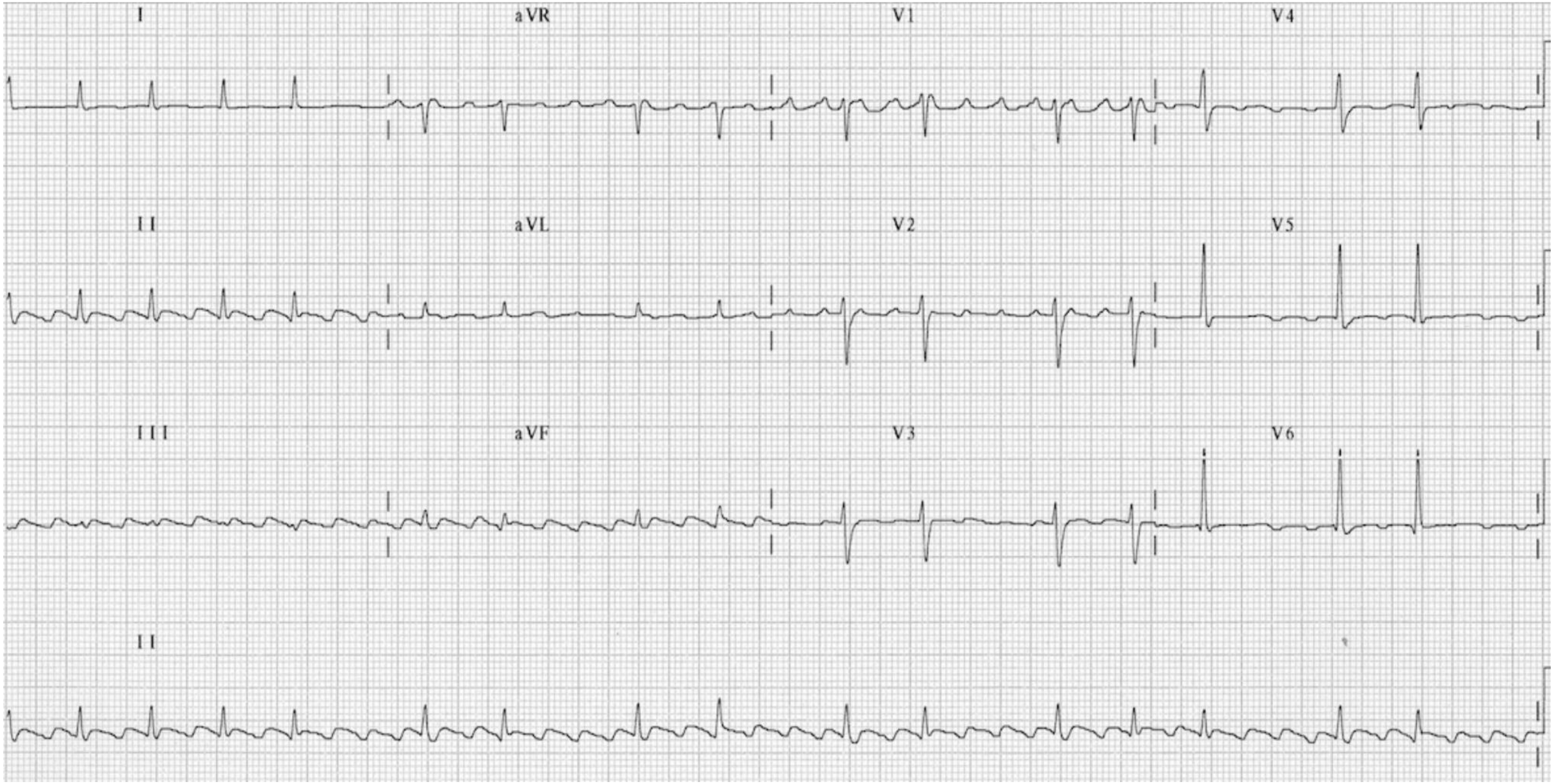


Fig. 16.35 Carotid sinus pressure in atrial flutter: continuous trace. The diagnosis of atrial flutter with 2 : 1 block was established when carotid sinus pressure produced temporary atrioventricular block, revealing the flutter waves.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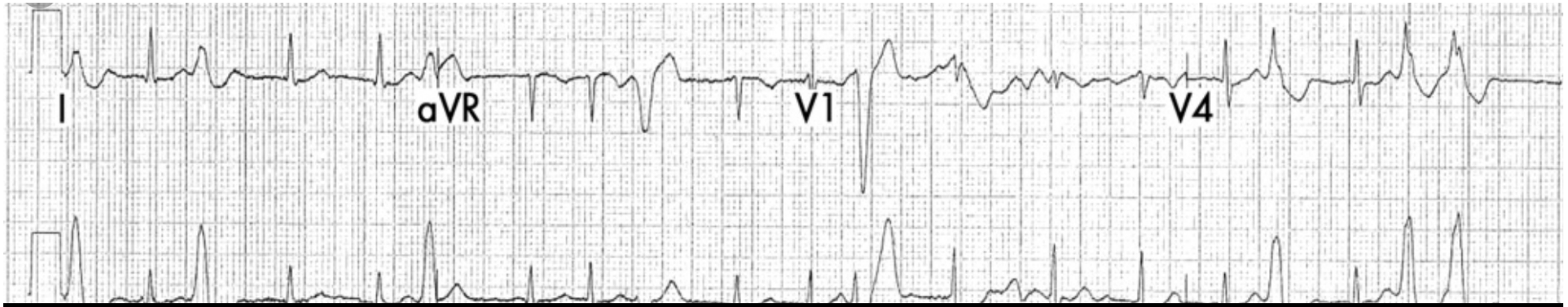
V3
V6
II

Managment

- ✓ *Medical management with digoxin, β-blockers or verapamil is often successful in controlling the ventricular rate.*
- ✓ *In many cases, however, it may be preferable to try to restore sinus rhythm by direct current (DC) cardioversion.*
- ✓ *Following cardioversion, β-blockers or amiodarone can be used to prevent recurrent episodes of atrial flutter.*
- ✓ ***Class Ic anti-arrhythmic drugs such as flecainide are contraindicated because there is a risk of slowing the flutter circuit, facilitating 1 : 1 AV nodal conduction and producing a paradoxical extreme tachycardia and haemodynamic compromise**.*
- ✓ *Catheter ablation is a highly effective treatment, offering a greater than 90% chance of complete cure, and is the treatment of choice for patients with persistent symptoms. Anticoagulant management in patients with atrial flutter, including management around cardioversion, is identical to that of patients with atrial fibrillation.*

Premature ventricular ectopics

- *Ventricular premature beats (VPBs) are frequently found in healthy people and their prevalence increases with age.*
- *Ectopic beats in patients with otherwise normal hearts are more prominent at rest and disappear with exercise.*
- *Sometimes VPBs are a manifestation of subclinical coronary artery disease or cardiomyopathy but also may occur in patients with established heart disease following an MI. Most patients with VPBs are asymptomatic but some present with an irregular heart beat, missed beats or abnormally strong beats, due to increased cardiac output of the post-ectopic sinus beat.*
- *The ECG shows broad and bizarre complexes because the ventricles are activated sequentially rather than simultaneously.*
- *The complexes may be **unifocal** (identical beats arising from a single ectopic focus) or multifocal (varying morphology with multiple foci, Fig. 16.40). '**Couplet**' and '**triplet**' are the terms used to describe two or three successive ectopic beats. A run of alternating sinus and ventricular ectopics called **bigeminy**.*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V1
V4

Management of PVE

- *In symptomatic patients , b blocker helpful to control symptoms.*
- *In refractory cases , catheter ablation can be useful.*
- *Frequent PVCs are common in patients with structural heart disease, like MI, cardiomyopathy, and associated with worse prognosis, treatment should be directed toward underling heart disease, unfortunately antiarrythmic medication not improve prognosis except beta blocker in the setting of MI and heart failure.*

➢Ventricular tachycardia

- ➢vetricular tachycardia (VT) occurs most commonly in the settings of acute MI, chronic coronary artery disease and cardiomyopathy , It is associated with extensive ventricular disease, impaired left ventricular function and ventricular aneurysm.
- ➢ In these settings, VT may cause haemodynamic compromise or degenerate into ventricular fibrillation.
- ➢ VT is caused by abnormal automaticity or triggered activity in ischaemic tissue, or by re-entry within scarred ventricular tissue. Patients may complain of palpitation or symptoms of low cardiac output, including dyspnoea, lightheadedness and syncope.
- ➢ The ECG shows tachycardia and broad, abnormal QRS complexes with a rate of more than 120/min.
- ➢ It may be difficult to distinguish VT from SVT with bundle branch block or pre-excitation (WPW syndrome) on ECG but features in favour of VT are listed in Box 16.25. A 12-lead ECG or electrophysiology study (p. 454) may help establish the diagnosis.

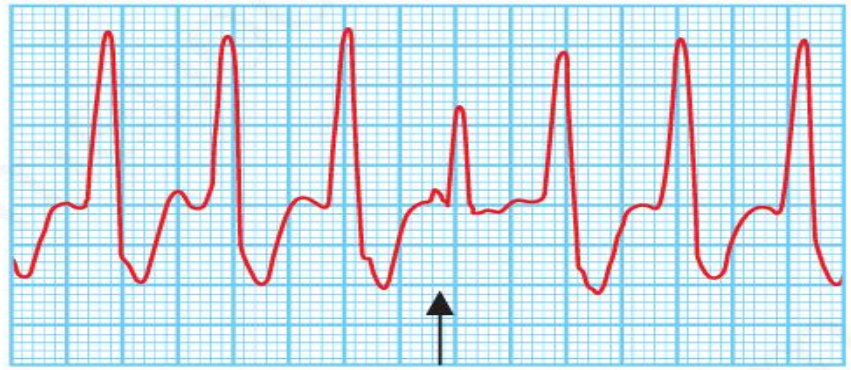
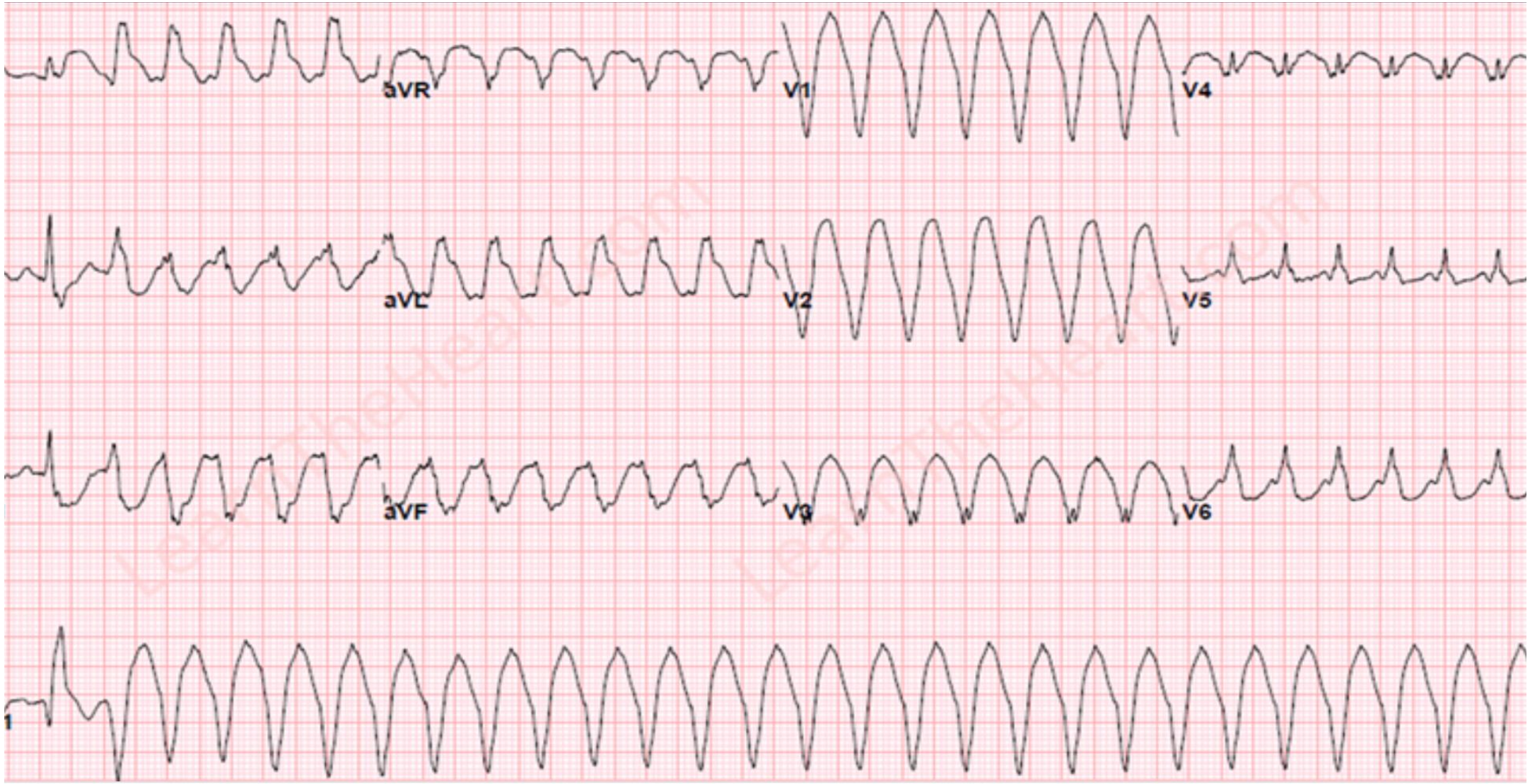


Fig. 16.41 Ventricular tachycardia: fusion beat (arrow). In ventricular tachycardia, there is independent atrial and ventricular activity. Occasionally, a P wave is conducted to the ventricles through the AV node, producing a normal sinus beat in the middle of the tachycardia (a capture beat); more commonly, however, the conducted impulse fuses with an impulse from the tachycardia (a fusion beat). This can occur only when there is atrioventricular dissociation and is therefore diagnostic of ventricular tachycardia.

16.25 Features more in keeping with ventricular tachycardia

- History of myocardial infarction
- Atrioventricular dissociation (pathognomonic)
- Capture/fusion beats (pathognomonic; see Fig. 16.41)
- Extreme left axis deviation
- Very broad QRS complexes (>140 msecs)
- No response to carotid sinus massage or intravenous adenosine

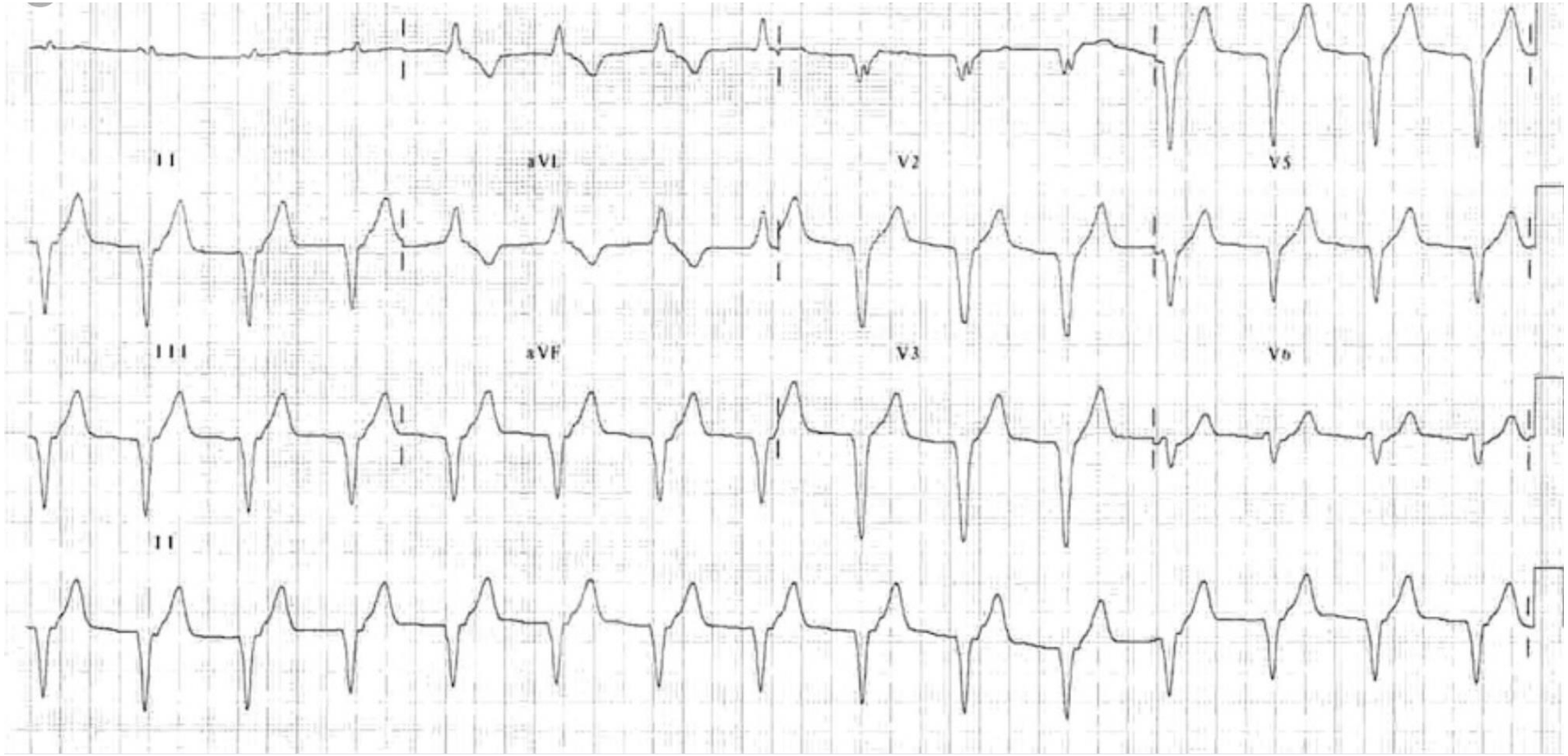

aVR
V1
V4
aVL
V2
V5
aVF
V3
V6
I

- *Patients recovering from MI sometimes have periods of idioventricular rhythm ('slow' VT) at a rate only slightly above the preceding sinus rate and below 120/min.*

- *These episodes often reflect reperfusion of the infarct territory and may be a good sign. They are usually self-limiting and asymptomatic, and do not require treatment.*

- *Occasionally, VT occurs in patients with otherwise healthy hearts ('normal heart VT'), usually because of abnormal automaticity in the right ventricular outflow tract or one of the fascicles of the left bundle branch*

Idioventricular rhythm



Managment

- ✓ *If VT associated with hemodynamic instability, require DC cardioversion (chest pain, dizziness, raised JVP, bibasal crackles)*

- ✓ *If VT is hemodynamically stable, intravenous amiodarone bolus and maintenance . Intravenous lidocaine can be used for chemical cardioversion of VT , but might cause hypotension, convulsion, myocardial depression.*

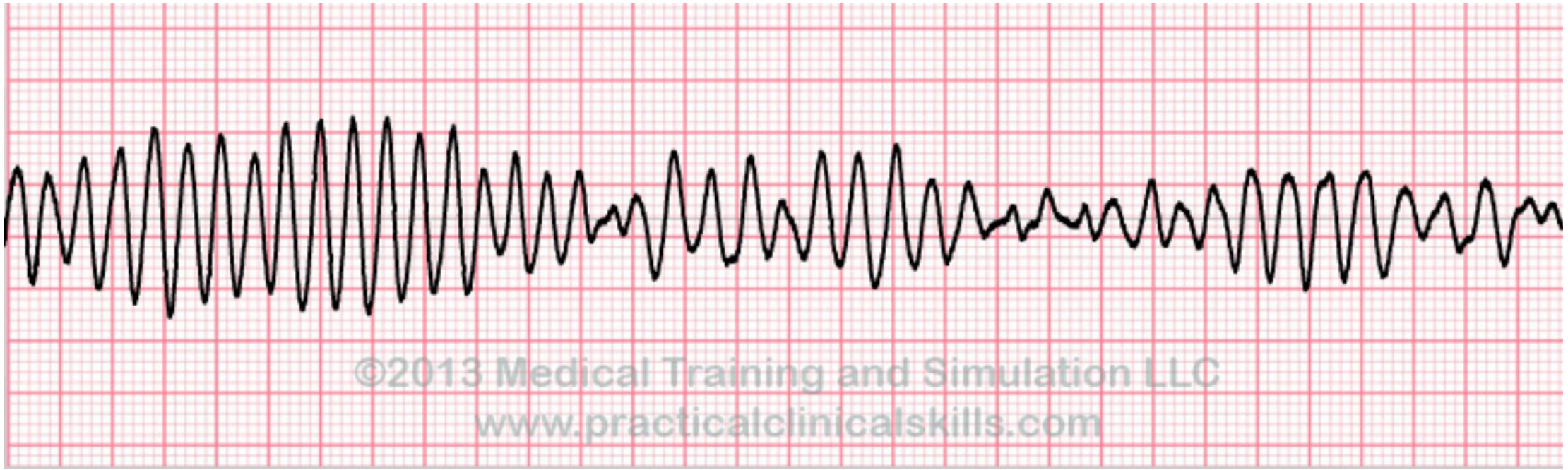
- ✓ *VT can be prevented by beta blocker and amiodarone, class IC antiarrythmic is contraindicated in patient with structural heart disease like CAD, cardiomyopathy.*
- ✓ *Consider ICD implantation*

Torsade de pointes

- *This form of polymorphic VT is a complication of prolonged ventricular repolarisation (prolonged QT interval).*

- *The ECG shows rapid irregular complexes that seem to twist around the baseline as the mean QRS axis changes.*
- *The arrhythmia is usually non-sustained and repetitive, but may degenerate into ventricular fibrillation.*
- *During periods of sinus rhythm, the ECG will usually show a prolonged QT interval (> 0.44 sec in men, > 0.46 sec in women when corrected to a heart rate of 60 bear per minutes)*

Torsade de pointes



- *The arrhythmia is more common in women and is often triggered by a combination of factors, such as administration of QT-prolonging medications and hypokalaemia.*

- *The congenital long QT syndromes are a family of genetic disorders that are characterised by mutations in genes that code for cardiac sodium or potassium channels.*
- *Adrenergic stimulation through vigorous exercise is a common trigger in long QT type 1, and a sudden noise may trigger arrhythmias in long QT type 2. Arrhythmias are more common during sleep in type 3*

16.26 Causes of long QT interval and torsades de pointes

Bradycardia

- Bradycardia compounds other factors that cause torsades de pointes

Electrolyte disturbance

- Hypokalaemia
- Hypomagnesaemia
- Hypocalcaemia

Drugs*

- Disopyramide, flecainide and other class Ia, Ic anti-arrhythmic drugs (p. 479)
- Sotalol, amiodarone and other class III anti-arrhythmic drugs
- Amitriptyline and other tricyclic antidepressants
- Chlorpromazine and other phenothiazines
- Erythromycin and other macrolides

Congenital syndromes

- Long QT1: gene affected *KCNQI*: K^+ channel, 30–35%
- Long QT2: gene affected *HERG*: K^+ channel, 25–30%
- Long QT3: gene affected *SCNSA*: Na^+ channel, 5–10%
- Long QT4–12: rare; various genes implicated

*Many other drugs that are not shown can be associated with prolongation of the QT interval. See www.crediblemeds.org for a complete list.

Managment

- ✓ Intravenous magnesium 8 mmol over 15 mins, 72 mmol over 24 hours should be given in all patients with torsade de pointes.

- ✓Overdrive atrial pacing useful suppress arrhythmia through rate dependent shortening of QT interval.

- ✓ Intravenous isoprenaline is alternative to atrial pacing , but contraindicated in long QT syndrome.

- ✓ *Beta-blockers are effective at preventing syncope in patients with congenital long QT syndrome.*

- ✓ *Some patients, particularly those with extreme QT interval prolongation (> 500 msecs) or certain high-risk genotypes, should be considered for an implantable defibrillator.*

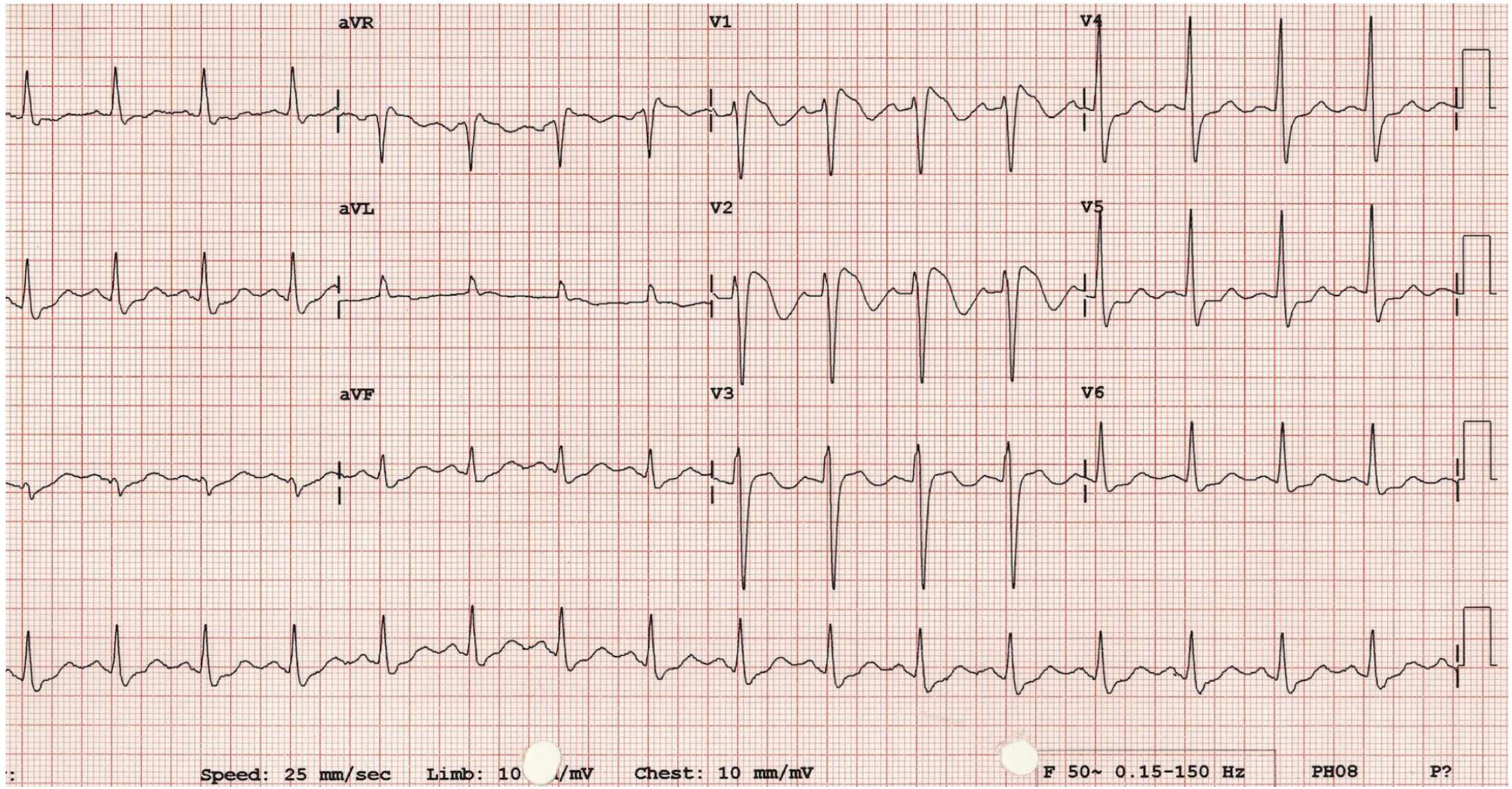
- ✓ *Left stellate ganglion block may be of value in patients with resistant arrhythmias.*

Brugada syndrome

- *Brugada syndrome is a related genetic disorder that may present with polymorphic VT or sudden death.*

- *It is characterised by a defect in sodium channel function and an abnormal ECG (right bundle branch block and ST elevation in V1 and V2 but not usually prolongation of the QT interval).*

- *The only known effective treatment is an implantable defibrillaton*


aVR
V1
V4
aVL
V2
V5
aVF
V3
V6
Speed: 25 mm/sec
Limb: 10
/mV
Chest: 10 mm/mV
F 50~ 0.15-150 Hz
PH08
P?

Atrioventricular block

- First-degree atrioventricular block .In this condition, AV conduction is delayed and so the PR interval is prolonged (> 0.20 sec. It rarely causes symptoms and does not usually require treatment.

- Second-degree atrioventricular block Here dropped beats occur because some impulses from the atria fail to conduct to the ventricles. Two subtypes are recognized :

- In Mobitz type I second-degree AV block, there is progressive lengthening of successive PR intervals, culminating in a dropped beat.
- The cycle then repeats itself. This is known as the Wenckebach phenomenon and is usually due to impaired conduction in the AV node itself. The phenomenon may be physiological and is sometimes observed at rest or during sleep in athletic young adults with high vagal tone.
- In Mobitz type II second-degree AV block, the PR interval of the conducted impulses remains constant but some P waves are not conducted. This is usually caused by disease of the His–Purkinje system and carries a risk of asystole.

- Third degree atrioventricular block :
- In third-degree AV block, conduction fails completely and the atria and ventricles beat independently. This is known as AV dissociation, Ventricular activity is maintained by an escape rhythm arising in the AV node or bundle of His (narrow QRS complexes) or the distal Purkinje tissues (broad QRS complexes).

- Distal escape rhythms tend to be slower and less reliable. Complete AV block (Box 16.27) produces a slow (25–50/min), regular pulse that does not vary with exercise, except in the case of congenital complete AV block. There is usually a compensatory increase in stroke volume, producing a large-volume pulse. Cannon waves may be visible in the neck and the intensity of the first heart sound varies due to the loss of AV synchrony.

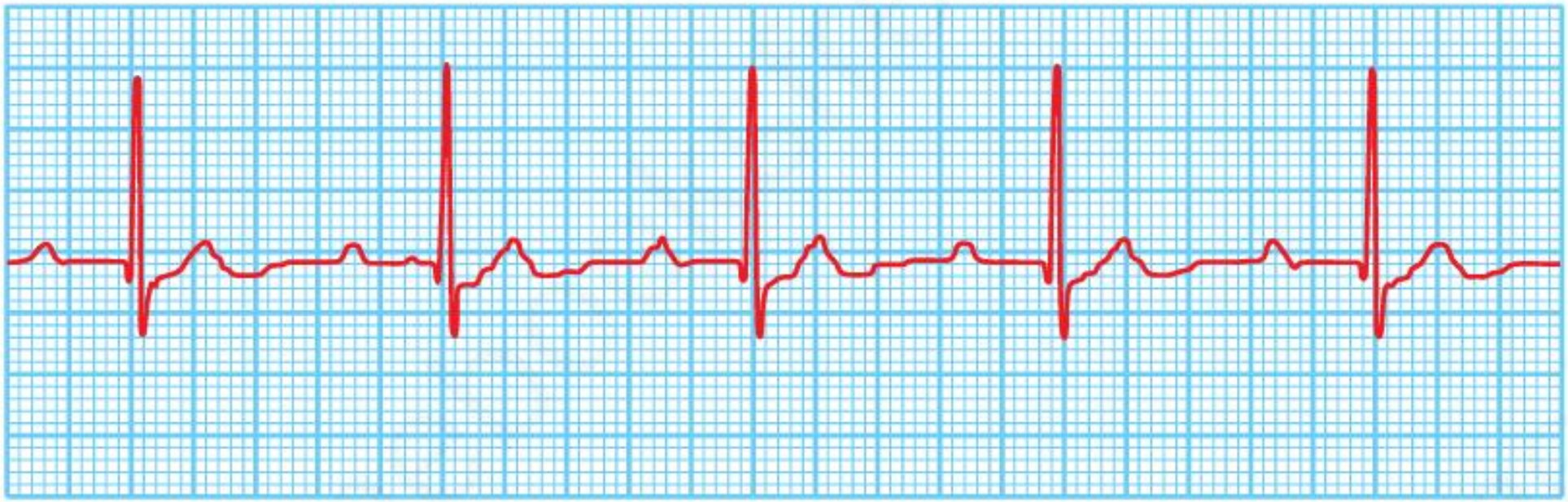


Fig. 16.44 First-degree atrioventricular block. The PR interval is prolonged and measures 0.26 sec.

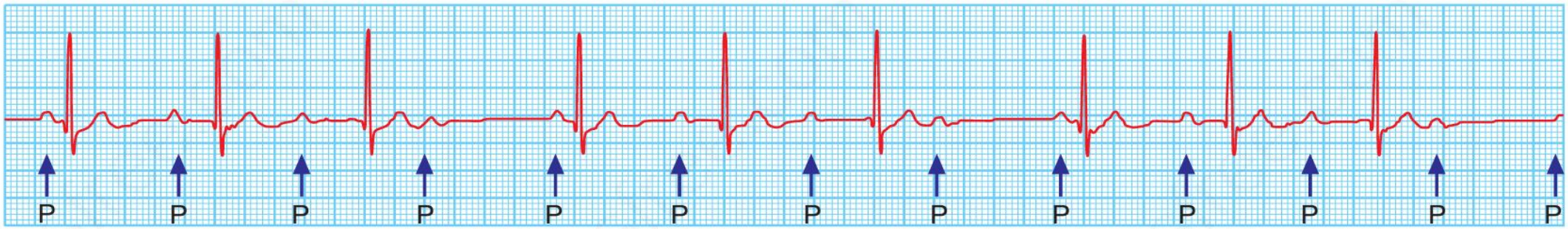


Fig. 16.45 **Second-degree atrioventricular block (Mobitz type I – the Wenckebach phenomenon).** The PR interval progressively increases until a P wave is not conducted. The cycle then repeats itself. In this example, conduction is at a ratio of 4:3, leading to groupings of three ventricular complexes in a row.

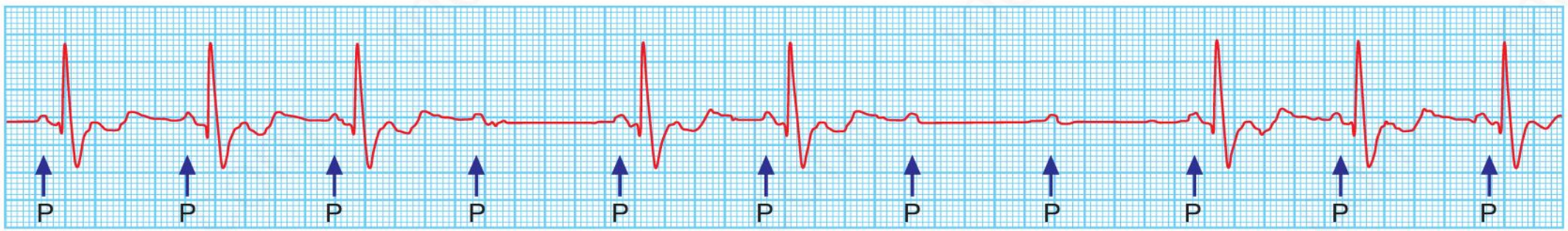
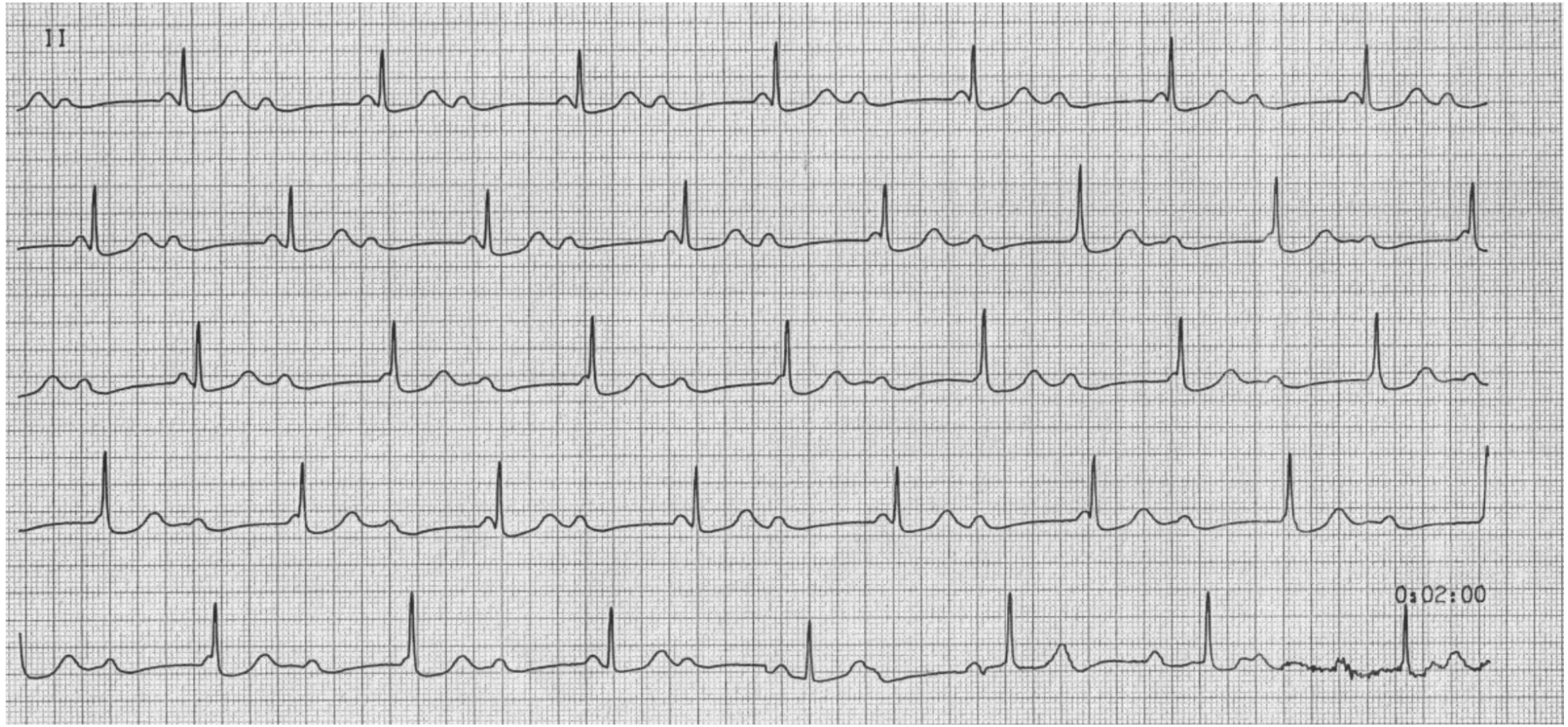


Fig. 16.46 **Second-degree atrioventricular block (Mobitz type II).** The PR interval of conducted beats is normal but some P waves are not conducted. The constant PR interval distinguishes this from the Wenckebach phenomenon.

CHB



16.27 Causes of complete atrioventricular block

Congenital

Acquired

- Idiopathic fibrosis
- Myocardial infarction/ischaemia
- Inflammation:
  - Infective endocarditis
  - Sarcoidosis
  - Chagas' disease
- Trauma
- Drugs:
  - Digoxin
  - β-blockers
  - Calcium antagonists

Clinical features

- Syncope
- Adam stoke attacks is a sudden loss of consciousness that occur without warning and result in collapse. Brief anoxic seizure may developed if there is a prolong asystole. There is pallor and death like appearance during the attack , and when the heart beat again there is characteristic flushing.

Treatment of heart block

- ✓ 1st and mobitz type 1 second degree AV block, usually asymptomatic
- ✓Mobitz type 2 second degree AV block , 3rd degree AV block; Acute inferior MI is often complicated by transient AV block because the right coronary artery (RCA) supplies the AV node. There is usually a reliable escape rhythm and, if the patient remains well, no treatment is required. Symptomatic second- or third-degree AV block may respond to atropine (0.6 mg IV, repeated as necessary) or, if this fails, a temporary pacemaker. In most cases, the AV block will resolve within 7–10 days

- ✓Second- or third-degree AV heart block complicating acute anterior MI indicates extensive ventricular damage involving both bundle branches and carries a poor prognosis.

- ✓ Mobitz type 2 or third degree AV block , even a symptomatic , if not due to reversible cause is an indication for pacing.

Lines in the management of arrythmias

- ✓ Drugs
- ✓ DC shock (cardioversion and defibrillation)
- ✓ Implantable device like pacemaker and ICD
- ✓ Catheter ablation

Antiarrythmic medication

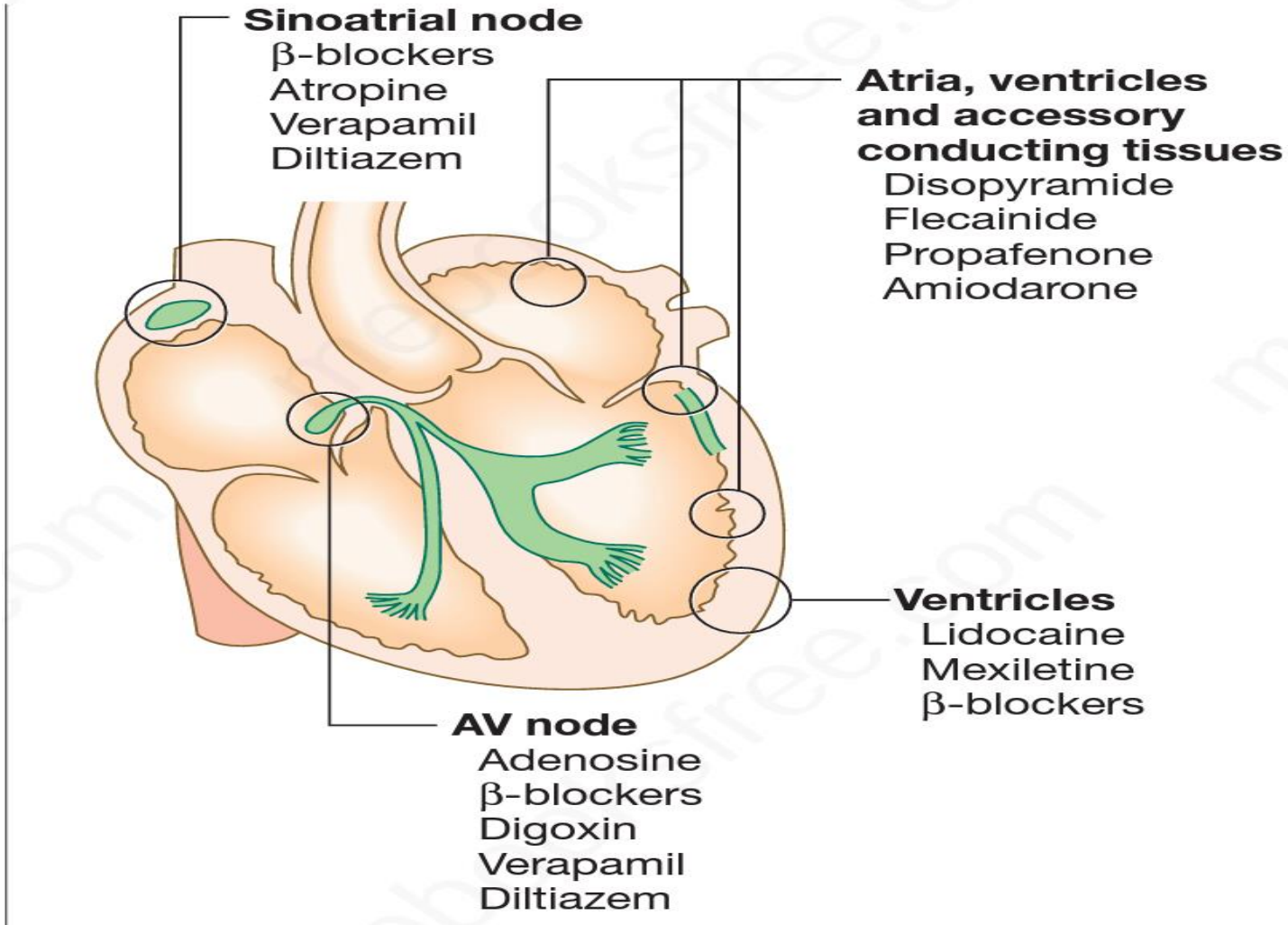


Fig. 16.51 Classification of anti-arrhythmic drugs by site of action.

Fig. 16.51 Classification of anti-arrhythmic drugs by site of action.

16.29 Classification of anti-arrhythmic drugs by effect on the intracellular action potential*

Class I: membrane-stabilising agents (sodium channel blockers)

(a) Block Na^+ channel and prolong action potential

- Quinidine, disopyramide

(b) Block Na^+ channel and shorten action potential

- Lidocaine, mexiletine

(c) Block Na^+ channel with no effect on action potential

- Flecainide, propafenone

Class II: β-adrenoceptor antagonists (β-blockers)

- Atenolol, bisoprolol, metoprolol

Class III: drugs whose main effect is to prolong the action potential

- Amiodarone, dronedarone, sotalol

Class IV: slow calcium channel blockers

- Verapamil, diltiazem

*Some drugs such as digoxin, ivabradine and adenosine have no place in this classification, while others such as amiodarone have properties in more than one class.