VENTRICULAR TACHYARRHYTHMIAS

Ventricular tachyarrhythmia can be considered under the following headings:

- ***Ventricular tachycardia (VT):*** this subdivided to sustained and non-sustained VT
- ***Ventricular fibrillation (VF)***
- ***Torsades de pointes***
- ***Ventricular premature beats (PVC)***

VENTRICULAR TACHYCARDIA

VT most often occurs in patients with coronary heart disease or cardiomyopathies. In these settings it is serious because it may cause hemodynamic compromise or degenerate into ventricular fibrillation. There are 2 type of VT:

(1) Sustained VT

(2) Non-sustained VT

Non-sustained ventricular tachycardia (NSVT):

Is defined as ventricular tachycardia that is ≥ 3 consecutive beats at a rate of ≥ 120 beat per minute (b.p.m.) but lasts < 30 s.

Sustained ventricular tachycardia:

Sustained ventricular tachycardia (> 30 s) often results in pre-syncope (dizziness), syncope, hypotension and cardiac arrest, although it may be remarkably well tolerated in some patients. Examination reveals a pulse rate typically between 120 and 220 b.p.m. The common causes of ventricular tachycardia include acute myocardial infarction, cardiomyopathy and chronic ischemic heart disease, particularly when it is associated with a ventricular aneurysm or poor left ventricular function.

The ECG shows a rapid ventricular rhythm with broad (often 0.14 s or more), abnormal QRS complexes. AV dissociation may result in visible P waves which appear to march through the tachycardia, capture beats (intermittent narrow QRS complex owing to normal ventricular activation via the AV node and conducting system) and fusion beats (intermediate between ventricular tachycardia beat and capture beat). VT may be difficult to distinguish from supraventricular tachycardia with bundle branch block (SVT with aberrancy) or pre-excitation (WPW syndrome). Features in favour of a diagnosis of VT are listed in table below:

Features in favour of VT in the differential diagnosis of broad complex tachycardia

- **A history of myocardial infarction**
- **AV dissociation (pathognomonic)**
- **Capture/fusion beats (pathognomonic)**
- **Extreme left axis deviation**
- **Very broad QRS complexes (> 140 ms)**
- **No response to carotid sinus massage or i.v. adenosine**

Management

Rapid action to restore sinus rhythm is required and in most cases should be followed by prophylactic therapy. 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 an intravenous infusion, intravenous lidocaine can be used. Hypokalemia, hypomagnesaemia, acidosis and hypoxemia can aggravate the situation and must be corrected.

Beta-blockers may be effective at suppressing VT. Amiodarone can be added if additional control is needed. In patients considered at high risk of arrhythmic death (e.g. those with poor left ventricular function, or in whom VT is associated with hemodynamic compromise), the use of an implantable cardiac defibrillator is recommended. Surgery or catheter ablation can be used to interrupt the arrhythmia focus or circuit.

VENTRICULAR FIBRILLATION

This is very rapid and irregular ventricular activation with no mechanical effect. The patient is pulseless and becomes rapidly unconscious, and respiration ceases (cardiac arrest). The ECG shows shapeless, rapid oscillations and there is no hint of organized complexes. It is usually provoked by a ventricular ectopic beat. Ventricular fibrillation rarely reverses spontaneously. The only effective treatment is electrical defibrillation. Basic and advanced cardiac life support is needed. If the attack of ventricular fibrillation occurs during the first day or two of an acute myocardial infarction, it is probable that prophylactic therapy will be unnecessary. Survivors of these ventricular tachyarrhythmia's are, in the absence of an identifiable reversible cause (e.g. acute myocardial infarction, severe metabolic disturbance), at high risk of sudden death. Implantable cardioverter–defibrillators (ICDs) are first-line therapy in the management of these patients.

TORSADES DE POINTES

This form of polymorphic ventricular tachycardia is a complication of prolonged ventricular repolarization (prolonged QT interval). The ECG shows rapid irregular complexes that oscillate from an upright to an inverted position and 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. Some of the common causes are listed in table below:

CAUSES OF LONG QT INTERVAL AND TORSADES DE POINTES

Bradycardia

Electrolyte disturbance

- Hypokalemia
- Hypomagnesaemia

- Hypocalcaemia

Drugs

- Disopyramide (and other class la anti-arrhythmic drugs)
- Sotalol, amiodarone (and other class III anti-arrhythmic drugs)
- Amitriptyline (and other tricyclic antidepressants)
- Chlorpromazine (and other phenothiazines)
- Erythromycin (and other macrolides)

Congenital syndromes

- Romano-Ward syndrome (autosomal dominant)
- Jervell and Lange-Nielson syndrome (autosomal recessive, associated with congenital deafness)

Treatment should be directed at the underlying cause. Intravenous magnesium should be given in all cases. Long-term therapy may not be necessary if the underlying cause can be removed. Beta-blockers may be of value in patients with a congenital long QT syndrome. An implantable cardiac defibrillator is often advisable.

Brugada syndrome:

The Brugada syndrome is a related genetic disorder that may present with polymorphic ventricular tachycardia or sudden death; it is characterized by a defect in sodium channel function, and an abnormal ECG (right bundle branch block and ST elevation in V1 and V2, but there is no prolongation of the QT interval).

VENTRICULAR PREMATURE BEATS (PVC)

Frequent PVCs often occur during acute MI but need no treatment. Other than β-blockers, anti-arrhythmic drugs do not improve and prognosis. PVCs are common in patients with heart failure. PVCs are also a feature of digoxin toxicity, and may occur as *'escape beats'* in the presence of an

underlying bradycardia. Treatment should always be directed at the underlying condition.

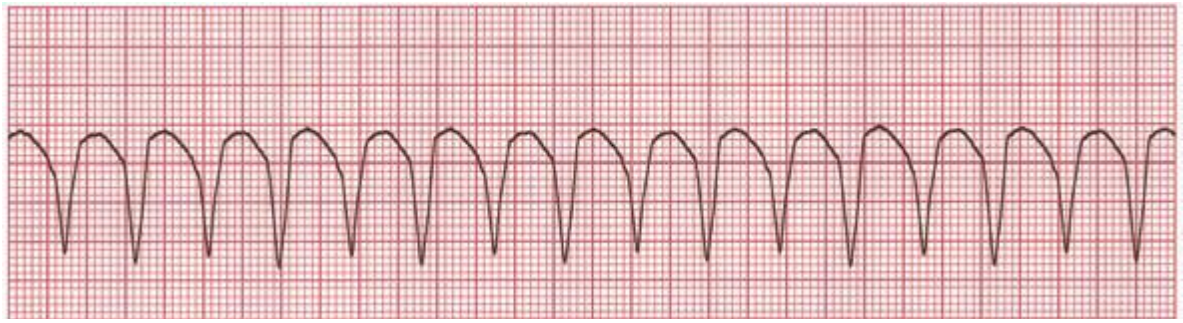
SINOATRIAL DISEASE (SICK SINUS SYNDROME)

Sinoatrial disease can occur at any age, but is most common in the elderly. The underlying pathology is not understood but may involve fibrosis, degenerative changes and/or ischemia of the sinoatrial (sinus) node. The condition is characterized by a variety of arrhythmias and may present with palpitation, dizzy spells or syncope, due to intermittent tachycardia, bradycardia, or pauses with no atrial or ventricular activity (sinoatrial block or sinus arrest).

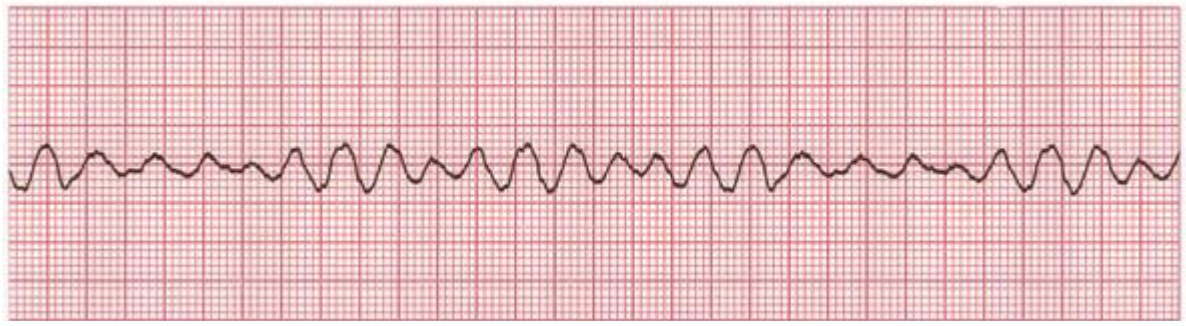
A permanent pacemaker may benefit patients with troublesome symptoms due to spontaneous bradycardias, or those with symptomatic bradycardias induced by drugs required to prevent tachyarrhythmia. Permanent pacing does not improve prognosis and is not indicated in patients who are asymptomatic.

COMMON FEATURES OF SINOATRIAL DISEASE

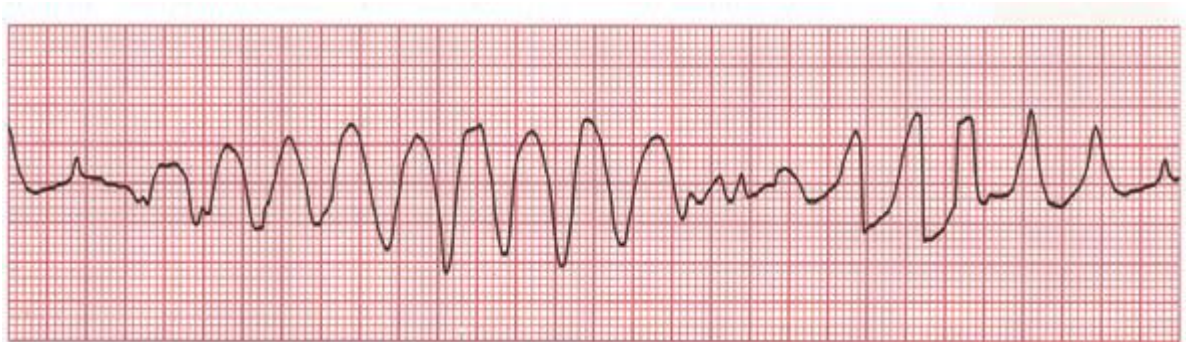
- *Sinus bradycardia*
- *Sinoatrial block (sinus arrest)*
- *Paroxysmal supraventricular tachycardia*
- *Paroxysmal atrial fibrillation*
- *Atrioventricular block*



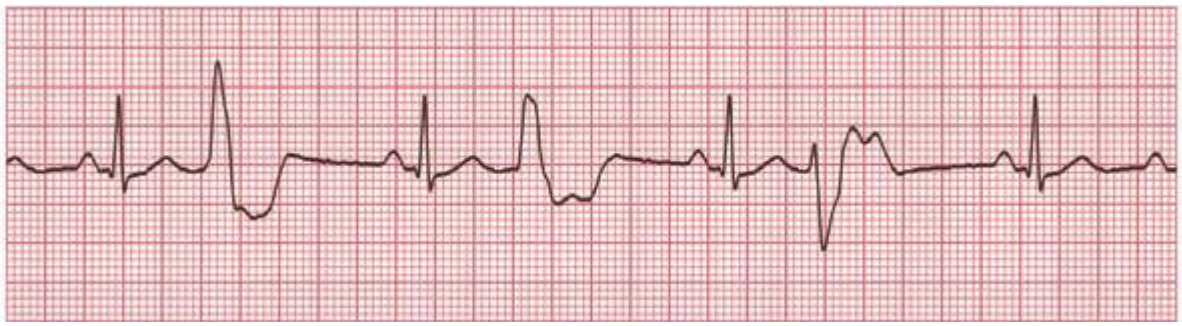
VT



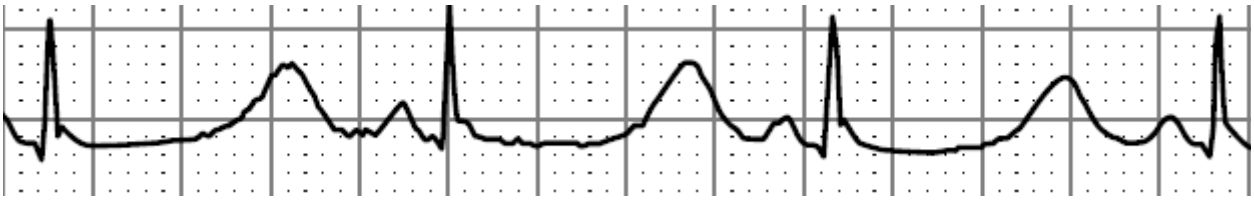
VF



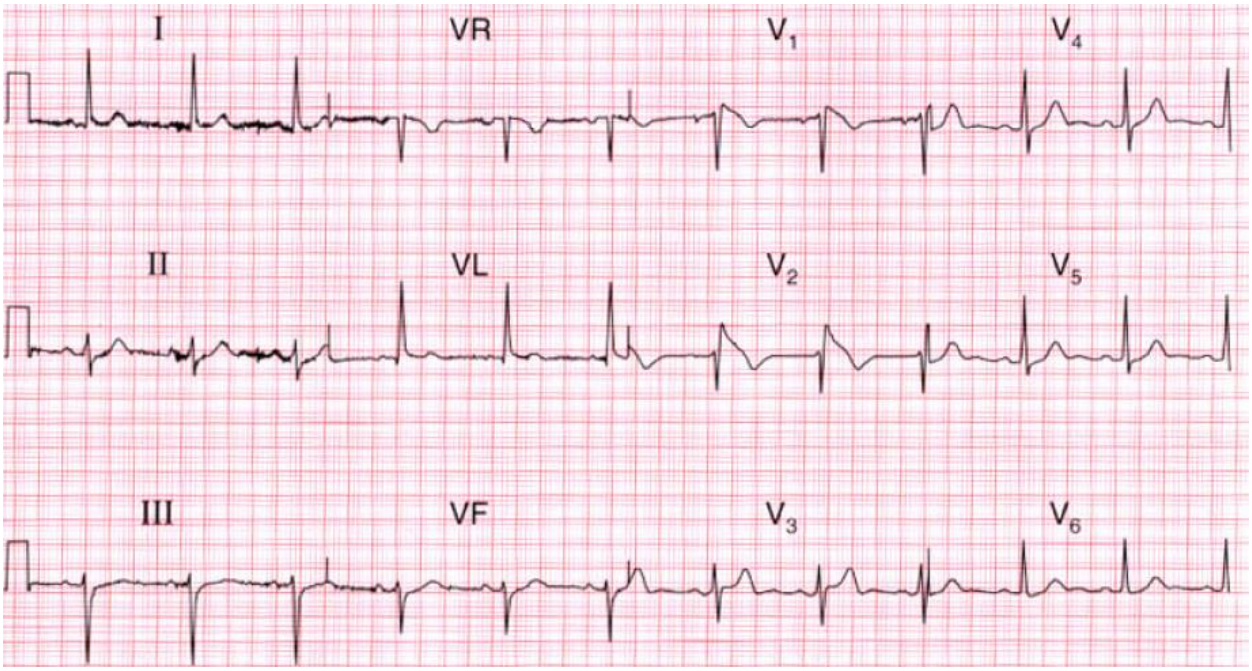
TORSADES DE POINTES



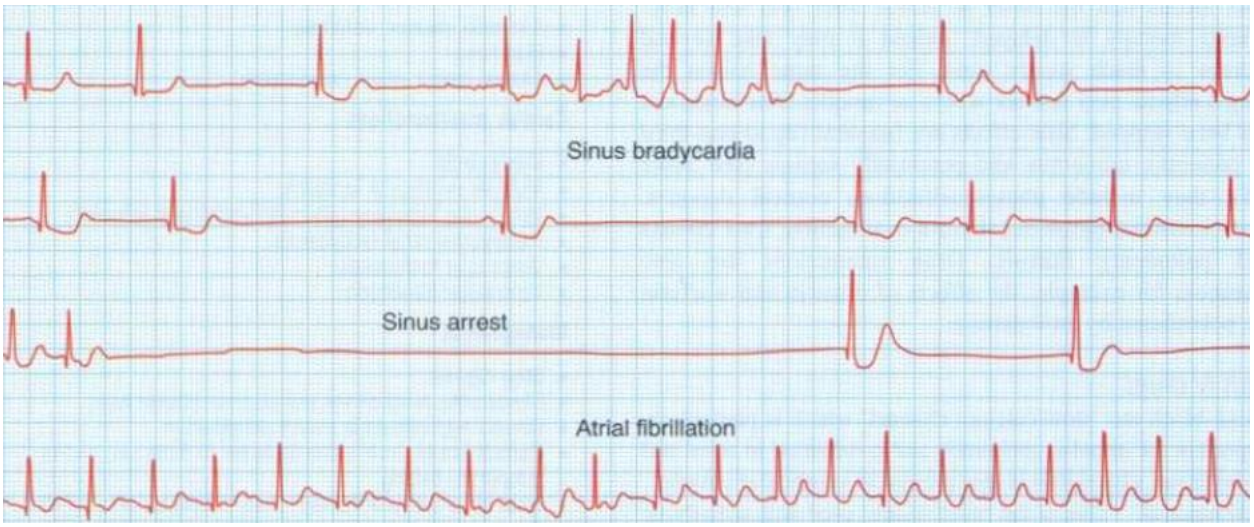
Ventricular Bigeminy (PVC every other beat)



LONG QT



BRUGADA SYNDROME



SICK SINUS SYNDROME