Cardiac arrhythmias

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Cardiac arrhythmias

- cardiac arrhythmia is defined as a disturbance of the electrical rhythm of the heart.
- Arrhythmias are often a manifestation of structural heart disease but may also occur because of abnormal conduction or depolarization in an otherwise healthy heart.
- There are many types of cardiac arrhythmia,. however, a heart rate of more than 100/min is a tachycardia, and a heart rate of less than 60/min is a bradycardia

Pathogenesis

- Arrhythmias usually occur as the result of pathology affecting the conduction system of the heart.
- The cardiac cycle is normally initiated by spontaneous depolarisation in the SA node
- The atria and ventricles then activate sequentially as the depolarisation wave passes through specialised conducting tissues

• There are three main mechanisms of tachycardia:

- 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.
- Re-entry. The tachycardia is initiated by an ectopic beat and sustained by a re-entry circuit. Most tachyarrhythmias are caused by re-entry.
- Triggered activity. This can cause ventricular arrhythmias in patients with coronary artery disease. It is a form of secondary depolarisation

• Arrhythmias may be supraventricular (sinus, atrial or junctional) or ventricular in origin

Clinical features

- Many arrhythmias are asymptomatic but sustained tachycardias typically present with rapid palpitation. Dizziness, chest discomfort or breathlessness may also occur. Extreme tachycardias can also cause syncope
- because the heart is unable to fill properly at extreme rates.
 Bradycardias tend to cause symptoms of low cardiac output, including fatigue, lightheadedness and syncope. Extreme bradycardias or tachycardias can precipitate sudden death or cardiac arrest

Symptoms of Cardiac Arrhythmia

- Palpitations: Feeling of a rapid, fluttering, or pounding heartbeat.
- Fatigue: Feeling unusually tired or weak.
- Chest Pain: Possible tightness or discomfort in the chest.
- Shortness of Breath: Difficulty breathing or rapid breathing.
- Fainting or Dizziness: Sudden loss of consciousness or feeling lightheaded.

Sinus arrhythmia

- This is 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 heart rate variation with breathing or with changes in posture may be a feature of diabetic neuropathy autonomic neuropathy or increased sympathetic drive.
- Sinus arrhythmia does not require treatment

Sinus bradycardia

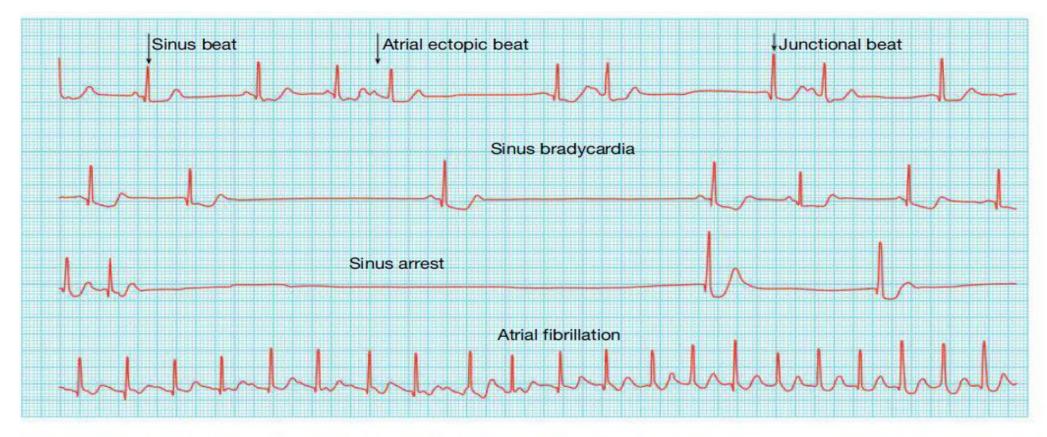
- This may occur in healthy people at rest and is a common finding in athletes.
- If sinus bradycardia is asymptomatic, then no treatment is required. Symptomatic sinus bradycardia may occur acutely during an MI and can be treated with intravenous atropine (0.6–1.2 mg).
- Patients with recurrent or persistent symptomatic sinus bradycardia should be considered for permanent pacemaker implantation

Sinus tachycardia

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

Sinoatrial disease

- Sinoatrial disease or 'sick sinus syndrome' can occur at any age but is
- most common in older people.
- It is caused by degenerative changes in the SA node and is characterised by a variety of arrhythmias.
- The typical presentation is with palpitation, dizzy spells or syncope, due to intermittent tachycardia, bradycardia, or pauses in sinus rhythm with no atrial or ventricular activity



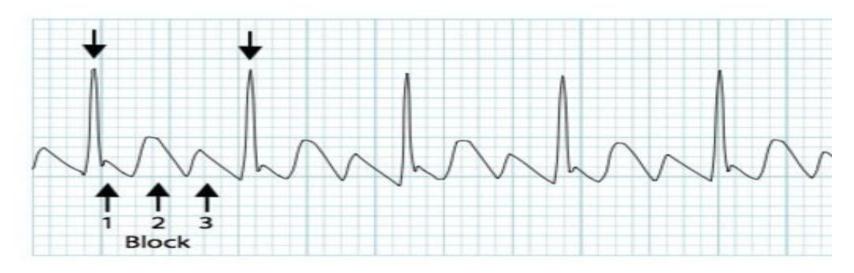
O Sinoatrial disease (sick sinus syndrome). A continuous rhythm strip from a 24-hour ECG tape recording illustrating periods of sinus rhythm, atrial ectopics, beats, sinus bradycardia, sinus arrest and paroxysmal atrial fibrillation.

Atrial tachycardia

- Atrial tachycardia may be a manifestation of increased atrial automaticity, sinoatrial disease or digoxin toxicity.
- It produces a narrow-complex tachycardia with abnormal P-wave morphology, sometimes associated with AV block if the atrial rate is rapid.
- It may respond to β -blockers, which reduce automaticity, or class I or III anti-arrhythmic drugs

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).
- Atrial flutter should be suspected when there is a narrow-complex
- tachycardia of 150/min



Atrial flutter with a 3:1 block

Atrial fibrillation

- Atrial fibrillation (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.
- 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

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 because of loss of atrial function and heart rate control.
- A fall in BP may cause lightheadedness, and chest pain may occur with underlying coronary artery disease, sometimes accompanied by ST segment and T-wave abnormalities on the ECG, and troponin elevation.

- In older patients, AF may not be associated with a rapid ventricular rate and may be asymptomatic, only to be discovered as a result of a routine examination or an ECG.
- Asymptomatic AF may also present with systemic embolism and is a major cause of stroke in older people

Supraventricular tachycardia

- The term supraventricular tachycardia (SVT) describes a group of regular tachycardias that have a similar appearance on ECG.
- These are usually narrow-complex tachycardias and are characterised by a re-entry circuit or automatic focus involving the atria.
- The three principal types are atrioventricular nodal re-entrant tachycardia (AVNRT), atrioventricular re-entrant tachycardia (AVRT) and atrial tachycardia. The term SVT is notstrictly accurate as, in many cases, the ventricles also form part of the re-entry circuit.

Ventricular premature beats

- 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

Ventricular tachycardia

- Ventricular 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 Patients may experience palpitation, dyspnoea, lightheadedness and syncope.
- The ECG shows tachycardia and broad, abnormal QRS complexes
- with a rate of more than 120/min

Principles of management of cardiac arrhythmias

- 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

Thank you