Block O points

by Fatima Haider

- **Coronary artery disease (CAD):** ischemic heart disease due to narrowing or blockage of coronary arteries, most commonly due to atherosclerosis, resulting in a mismatch between myocardial oxygen supply and demand
- **Cardiac chest pain**: likely associated with cardiac ischemia based on symptoms (e.g., central, retrosternal, squeezing, exertional).
- **Levine's sign** is a clenched fist held over the chest to describe ischemic chest pain.
- Hypotension (inferior wall MI)
- **Possible cardiac chest pain**: may be associated with cardiac ischemia based on symptoms (e.g., stabbing, tearing, ripping, burning).
- **Noncardiac chest pain**: unlikely associated with cardiac ischemia based on symptoms (e.g., positional, fleeting).
- **Chronic ischemic heart disease** - progressive heart failure that occurs after many years of chronic ischemic damage to the myocardium
- Patients with CAD usually become symptomatic when the degree of coronary stenosis reaches ≥ 70%.
- Coronary CT angiography **(CCTA)**: can visualize anatomic CAD
- **Vasospastic angina** - Angina caused by transient coronary spasms (usually due to spasms occurring close to areas of coronary stenosis); Not affected by exertion (may also occur at rest); Typically occurs early in the morning
- **Myocardial infarction (MI)** refers to ischemic necrosis of myocardial tissue. The most common underlying cause is coronary artery disease.
- **Type 1** myocardial infarction occurs when an unstable plaque ruptures, leading to occlusion of a coronary artery.
- **Type 2** myocardial infarction occurs when there is a mismatch between oxygen supply and demand (due to, e.g., systemic hypotension, vasospasm).
- ”**Silent MI”** without chest pain is more common in patients with diabetes, as a result of polyneuropathy.
- Clinical triad in **right ventricular infarction**: hypotension, elevated jugular venous pressure, clear lung fields
- The **most commonly occluded coronary arteries** (in descending order): left anterior descending artery, right coronary artery, circumflex artery.
- **Revascularization**: all patients with suspected acute coronary syndrome should be considered for emergency percutaneous coronary intervention **(PCI)**
- **Cardiac arrhythmias** are accelerated, slowed, or irregular heart rates caused by abnormalities in the electrical impulses of the myocardium.
- **Bradyarrhythmias** include sinus node dysfunction and atrioventricular block, and are characterized by a resting heart rate < 60/minutes.
- **Tachyarrhythmias** (heart rates > 100/minute) are classified as supraventricular arrhythmias or ventricular arrhythmias.
- **Supraventricular arrhythmias**: Arrhythmias that originate in the sinoatrial node, atrial myocardium, or atrioventricular node (regular QRS complex)

- **Ventricular arrhythmias** originate below the atrioventricular node, on the ventricular level (wide QRS complex)
- **Atrial fibrillation** (Afib) is a common type of supraventricular tachyarrhythmia characterized by uncoordinated atrial activation that results in an irregular ventricular response
- The most common congenital accessory pathway (**bundle of Kent) is seen in Wolff-Parkinson-White (WPW) syndrome** and can cause ventricular preexcitation, in which supraventricular impulses bypass the AV node and are abnormally conducted to the ventricles, leading to a characteristic preexcitation pattern on ECG and a specific subset of preexcited tachyarrhythmias.
- **Paroxysmal tachycardia:** an arrhythmia with an abrupt onset and termination that can last from seconds to days (e.g., AVNRT)
- **Paroxysmal SVT (PSVT) -** Any SVT with a narrow QRS complex and an abrupt onset, Most commonly caused by AV nodal reentry
- Treat unstable SVTs (except for MAT and junctional tachycardia) with electrical cardioversion.
  If there is doubt about the supraventricular origin of a wide complex tachycardia, treat it as ventricular tachycardia.
- TTE is Initial imaging modality of choice in supraventricular tachyarrhythmia
- **Vagal maneuvers** - slows the conduction through the AV node (negative homotopy) and may terminate the arrhythmia
- **Catheter ablation** - Radiofrequency or cryothermal energy is applied via a cardiac catheter to eliminate aberrant pathways or impulses from arrhythmogenic foci.
- **Orthodromic AVRT**: Antegrade conduction (atrium → ventricle) through AV node (narrow QRS complex); retrograde conduction (ventricle → atrium) through accessory pathway
- **Antidromic AVRT:** Antegrade conduction (atrium → ventricle) through accessory pathway; retrograde conduction (ventricle → atrium) through AV node
- **Junctional tachycardia** - A tachyarrhythmia caused by abnormal automaticity of myocytes in the AV node and bundle of His
- **Nonsustained ventricular tachycardia (NSVT):** VT lasting < 30 seconds with spontaneous termination
- **Sustained ventricular tachycardia**: VT lasting ≥ 30 seconds or VT causing hemodynamic instability within 30 seconds
- **Monomorphic VT**: QRS morphology similar in all beats, indicating a single arrhythmogenic focus
- **Polymorphic VT**: QRS morphology varies in each beat, indicating multiple arrhythmogenic foci
- ≥ 3 consecutive wide QRS complexes at a frequency ≥ 100/minute and signs of AV dissociation confirm a diagnosis of VT
- **Bradycardia** is generally defined as a heart rate of < 60/min.
- **Transcutaneous pacing** - a temporizing treatment for bradyarrhythmias in which electrical impulses are delivered through pacing pads placed on the chest wall to stimulate cardiac contraction

- **Transvenous pacing** - The delivery of electrical impulses to stimulate cardiac contraction using an electrode placed via a central venous access site into the right ventricle; often used as a bridge to permanent pacemaker implantation
- **Cannon A waves**: physical examination finding seen in AV dissociation (e.g., in third-degree heart block)
- **Acute rheumatic fever** (ARF) is a sequela of streptococcal infection—typically following 2 to 4 weeks after group A streptococcal (strep. pyogenes) pharyngitis and has rheumatologic, cardiac, and neurologic manifestations.
  Antibodies produced against the streptococcal antigens cause inflammation in the endocardium, myocardium and pericardium, as well as the joints and skin, but the major effect on health is due to damage to heart valves
- Histological findings: **Aschoff bodies in myocardium** composed of: Central area of fibrinoid necrosis surrounded by characteristic multinucleated giant cells, mononuclear cells (Anitschkow cells) and other inflammatory cells(plasma cells, and T lymphocytes) due to a type IV hypersensitivity reaction
- **Treatment of Rheumatic fever without carditis**: 5 years or until the patient reaches 21 years of age. (whichever is longer)
- **Rheumatic fever with carditis**: 10 years or until the patient reaches 21 years of age. (whichever is longer)
- **Rheumatic fever with carditis and permanent valvular heart defects**: 10 years or until patient reaches 40 years of age.(whichever is longer, sometime life long prophylaxis)
- In **chronic rheumatic heart disease**, the mitral valve is affected in more than 70% of cases; the aortic valve is the next most frequently involved (40%) , followed by the tricuspid (10%) and then the pulmonary valve (2%).
- **Echocardiogram**: Best initial test for all VHDs (valvular heart diseases)
- **Ortner's syndrome**: which is hoarseness due to left recurrent laryngeal nerve compression by the dilated LA.
- **Mitral facies (Malar flush)** pink purple plaques on cheeks (due ↑ Systemic vasoconstriction)
- **Hockey stick appearance on echo: classic for Mitral stenosis**
- **Mitral valve prolapse click:** High frequency, Mid-systolic click (most common sign) Produced by the sudden prolapse of the valve and the tensing of the chordae tendineae that occurs during systole. best heard at the mitral region
- **Aortic Regurgitation (AR)** is the leaking of the aortic valve of the heart that causes blood to flow from Aorta to the LV during diastole.
- **When IE is suspected the best initial test is to obtain serial blood cultures**.
- **RBBB** can be either physiological or the result of damage to the right bundle branch. Causes of damage include underlying lung pathology (COPD, pulmonary emboli, cor pulmonale), primary heart muscle disease (ARVC), congenital heart disease (e.g. ASD), ischaemic heart disease and primary degeneration of the right bundle.
- **LBBB** is always pathological. Left bundle branch block may be due to conduction system degeneration or myocardial pathologies such as ischaemic heart disease, cardiomyopathy and valvular heart disease.

Respiratory points

- Bradypnea - Respiratory rate < 12/min in adults
- Tachypnea - Respiratory rate > 20/min, shallow breathing in adults
- Hyperpnea - respiratory rate > 20/min, deep breathing
- Inspiratory:expiratory ratio: The ratio of the inspiratory time to expiratory time during spontaneous breathing, which is normally 1:2.
- Cheyne-Stokes breathing: alternating periods of deep breathing followed by apnea
- Ataxic breathing: irregular breathing in rhythm and depth
- Obstructive breathing: prolonged exhalation
- Tripod position: patients with emphysema and respiratory distress will lean forward while sitting, resting with their hands on their knees.
- Cyanosis: bluish discoloration of the skin and mucosa (due to deoxygenated hemoglobin)
- Kyphosis or scoliosis may lead to decreased forced vital capacity, forced expiratory volume and overall respiratory function
- Tactile fremitus - Can be asymmetrically decreased in effusion, obstruction, or pneumothorax, among others. Can be asymmetrically increased in pneumonia
- Hyper-resonant percussion note - Sign of increased air inside the thoracic cavity: emphysema, bronchial asthma, pneumothorax
- Dull percussion note - Sign of fluid inside the thoracic cavity: pneumonia, pleural effusion
- Emphysema leads to expiratory airflow limitation and air trapping. The loss of lung elastic recoil results in an increase in TLC. Premature closure of airways limits expiratory flow while the loss of alveoli decreases capacity for gas transfer.
-