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CARDIOLOGY

Examination Paper

for DM (Cardiology) Students



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Foreword
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LONG-ESSAY QUESTIONS AND ANSWERS

Q 1. Discuss embryology, pathophysiology of conotruncal abnormalities, and the principles of their management.
(Refer 2014/2019 MUHS, 2009 MGR, 2018 Safdarjung)

Ans. Conotruncal abnormalities encompass a group of congenital heart defects that involve malformations of the outflow tracts of the heart during embryological development. Understanding the embryology, pathophysiology, and principles of management of conotruncal abnormalities is essential for effective clinical management.

- *Embryology:* During embryonic development, the heart forms from the fusion and differentiation of the bilateral cardiogenic plates. The heart tube undergoes looping and septation to form the four-chambered heart with the great vessels. The outflow tracts of the heart, which include the conus arteriosus and truncus arteriosus, give rise to the aorta and pulmonary artery, respectively.
- *Pathophysiology:* Conotruncal abnormalities arise from disruptions in the development of the outflow tracts and are often associated with defects in septation and alignment. Common conotruncal abnormalities include:
  - *Tetralogy of Fallot (TOF):* TOF is characterized by four main features: (1) A ventricular septal defect (VSD), (2) pulmonary stenosis (obstruction of the right ventricular outflow tract), (3) overriding aorta (aorta that straddles the VSD), and (4) right ventricular hypertrophy. These abnormalities result in mixing of oxygenated and deoxygenated blood, leading to cyanosis.
  - *Transposition of the great arteries (TGA):* In TGA, the aorta arises from the right ventricle, and the pulmonary artery arises from the left ventricle, leading to parallel circulation and inadequate oxygenation of blood. This defect typically results in severe cyanosis soon after birth.
  - *Persistent truncus arteriosus:* In this defect, there is failure of septation between the aorta and pulmonary artery, resulting in a single arterial trunk arising from both ventricles. This leads to mixing of oxygenated and deoxygenated blood and subsequent cyanosis.
- *Principles of management*: The management of conotruncal abnormalities typically involves a multidisciplinary approach and may include medical

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management, surgical intervention, and long-term follow-up. Principles of management include:

- *Medical stabilization*: Initial management focuses on stabilizing the patient and managing symptoms such as cyanosis, respiratory distress, and heart failure. This may involve supplemental oxygen, intravenous (IV) fluids, and medications to improve cardiac function and reduce pulmonary congestion.
- *Surgical repair*: Definitive treatment for conotruncal abnormalities often requires surgical intervention. The specific surgical procedure depends on the type and severity of the defect but may include repair of the VSD, relief of pulmonary stenosis, arterial switch operation in TGA, or repair of the truncus arteriosus.
- *Timely intervention*: Surgical repair is typically performed in early infancy to optimize outcomes and prevent complications associated with cyanotic heart disease, such as polycythemia, thromboembolism, and developmental delays.
- *Long-term follow-up*: Patients with conotruncal abnormalities require lifelong follow-up with a cardiologist to monitor cardiac function, assess for complications, and optimize medical management. Regular echocardiograms, electrocardiograms, and cardiac imaging may be necessary to monitor for residual defects, valve function, and cardiac function.
- *Genetic counseling*: In cases of syndromic conotruncal abnormalities or family history of congenital heart disease, genetic counseling may be recommended to assess the risk of recurrence in future pregnancies and provide information about inheritance patterns and genetic testing options.

In summary, conotruncal abnormalities result from disruptions in the development of the outflow tracts of the heart during embryological development. Management requires a multidisciplinary approach involving medical stabilization, surgical intervention, and long-term follow-up to optimize outcomes and quality of life for affected individuals. Early diagnosis, timely intervention, and comprehensive care are essential for improving outcomes in patients with conotruncal abnormalities.

Q 2. Discuss accessory pathways of conduction and treatment of related arrhythmias. ***(Refer 2019 MUHS)***

Ans. Accessory pathways of conduction, also known as bypass tracts, are abnormal electrical connections between the atria and ventricles of the heart. These pathways bypass the normal conduction system, allowing for rapid conduction of electrical impulses, and predisposing individuals to various types of arrhythmias, most notably Wolff–Parkinson–White (WPW)

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syndrome. Understanding the anatomy, physiology, and treatment of accessory pathways is crucial for managing arrhythmias associated with these pathways effectively.

- *Anatomy and physiology:* Accessory pathways can be classified based on their location and connection to the atrioventricular (AV) node:
  - *Kent bundles:* These pathways connect the atria and ventricles directly, bypassing the AV node. Kent bundles are characteristic of WPW syndrome.
  - *Mahaim fibers:* These pathways connect the atria and ventricles at the level of the bundle of His or bundle branches. Mahaim fibers are less common than Kent bundles but can also contribute to preexcitation syndromes.

  Accessory pathways allow for rapid conduction of electrical impulses from the atria to the ventricles, bypassing the normal delay at the AV node. This can result in premature activation of the ventricles and predispose individuals to supraventricular tachyarrhythmias, including atrioventricular reentrant tachycardia (AVRT) and atrial fibrillation with rapid ventricular response.
- *Clinical presentation:* Patients with accessory pathways may be asymptomatic or present with symptoms related to arrhythmias, such as palpitations, chest pain, syncope, or sudden cardiac arrest. The presence of characteristic ECG findings, including a short PR interval, delta waves (slurred upstroke of the QRS complex), and widened QRS complexes during tachycardia, can help diagnose WPW syndrome.
- *Treatment of arrhythmias related to accessory pathways:* The treatment of arrhythmias associated with accessory pathways depends on the type of arrhythmia, symptom severity, and underlying cardiac status. Treatment options include:
  - *Observation:* Asymptomatic individuals with WPW pattern on ECG may not require specific treatment but should be monitored regularly for arrhythmias and symptom development.
  - *Acute management of symptomatic arrhythmias:*
    - Vagal maneuvers: Valsalva maneuver or carotid sinus massage can sometimes terminate re-entrant tachyarrhythmias such as AVRT.
    - Adenosine: IV adenosine can be used to terminate paroxysmal supraventricular tachycardia (PSVT) by blocking conduction through the AV node.
    - Electrical cardioversion: Synchronized cardioversion may be necessary for hemodynamically unstable patients with refractory arrhythmias.
  - *Long-term management:*
    - Catheter ablation: Radiofrequency catheter ablation is the treatment of choice for symptomatic accessory pathways. During

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the procedure, the abnormal pathway is identified and selectively ablated, interrupting conduction through the pathway.

- Antiarrhythmic medications: Medications such as beta-blockers, calcium channel blockers, or antiarrhythmic drugs (e.g., flecainide and propafenone) may be used to suppress arrhythmias or reduce symptom frequency in patients who are not candidates for or decline catheter ablation.
- Risk assessment and anticoagulation: Patients with WPW syndrome and a history of atrial fibrillation may require anticoagulation to reduce the risk of stroke.

- *Prognosis:* The prognosis for patients with accessory pathways depends on various factors, including the type and location of the pathway, the presence of associated cardiac abnormalities, and the effectiveness of treatment. With appropriate management, including catheter ablation, the majority of patients can achieve resolution of symptoms and normalization of cardiac conduction.

In summary, accessory pathways of conduction are abnormal electrical connections between the atria and ventricles that can predispose individuals to supraventricular tachyarrhythmias. Treatment options include observation, acute management of symptomatic arrhythmias, and long-term management strategies such as catheter ablation and antiarrhythmic medications. Timely intervention and individualized treatment plans are essential for optimizing outcomes in patients with accessory pathway-related arrhythmias.

SHORT QUESTIONS AND ANSWERS

Q 1. Cyanotic spell. ***(Refer 2012/2019 MUHS)***

Ans. Cyanotic spell, also known as cyanotic episode or "tet spell," are acute exacerbations of cyanosis (bluish discoloration of the skin and mucous membranes) in infants and young children with congenital heart defects, particularly those with TOF. TOF is a complex congenital heart defect characterized by four main features:

1. *Ventricular septal defect*: A hole in the wall (septum) that separates the right and left ventricles.
2. *Pulmonary stenosis*: Narrowing of the pulmonary valve or pulmonary artery, which restricts blood flow to the lungs.
3. *Overriding aorta*: The aorta is positioned directly over the VSD, allowing oxygen-poor blood from the right ventricle to flow into the aorta and mix with oxygen-rich blood from the left ventricle.

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4. *Right ventricular hypertrophy*: Enlargement of the right ventricle due to increased workload.

During a cyanotic spell, several factors contribute to a sudden decrease in systemic arterial oxygen saturation, leading to worsening cyanosis and potentially life-threatening hypoxemia. The exact mechanisms underlying cyanotic spells are not fully understood, but they are believed to involve a combination of increased right-to-left shunting of deoxygenated blood, decreased pulmonary blood flow, and increased systemic vascular resistance.

The typical triggers for cyanotic spells in infants and young children with TOF include:

- *Crying or agitation:* Increased sympathetic tone and increased oxygen demand can worsen right-to-left shunting and exacerbate cyanosis.
- *Feeding:* Increased oxygen demand during feeding can lead to decreased pulmonary blood flow and worsening cyanosis.
- *Exercise or exertion*: Physical activity can increase oxygen demand and worsen cyanosis.

Clinical manifestations of cyanotic spells may include:

- *Cyanosis:* Bluish discoloration of the skin, lips, and mucous membranes due to decreased oxygen saturation.
- *Respiratory distress:* Tachypnea (rapid breathing), dyspnea (shortness of breath), and respiratory distress may occur due to hypoxemia.
- *Irritability or agitation:* Infants may become fussy or irritable during cyanotic spells.
- *Syncope or loss of consciousness:* Severe hypoxemia can lead to loss of consciousness in some cases.

Management of cyanotic spells typically involves addressing the underlying cause (e.g., correcting dehydration and relieving airway obstruction) and providing supplemental oxygen to alleviate hypoxemia. In cases of severe cyanotic spells, interventions aimed at increasing systemic vascular resistance and reducing right-to-left shunting may be necessary. These interventions may include:

- *Knee-to-chest position:* Placing the child in a knee-to-chest position can increase systemic vascular resistance and improve pulmonary blood flow.
- *Oxygen therapy:* Administration of supplemental oxygen can alleviate hypoxemia and improve oxygen delivery to tissues.
- *IV fluids:* Administering IV fluids can help correct dehydration and increase intravascular volume, which may improve cardiac output and systemic perfusion.
- *Sedation*: Administering sedatives or analgesics may help calm the child and reduce agitation, thereby decreasing oxygen demand.

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In severe cases refractory to medical management, emergency interventions such as IV morphine administration or even emergent surgical interventions to relieve obstruction (e.g., balloon valvuloplasty for pulmonary stenosis) may be necessary to stabilize the child and prevent further deterioration.

Overall, cyanotic spells are critical events in infants and children with congenital heart defects, particularly TOF, and require prompt recognition and appropriate management to prevent complications and optimize outcomes. Close monitoring and follow-up with a pediatric cardiologist are essential for infants and children with TOF to detect and manage cyanotic spells effectively.

Q 2. Endothelial receptor antagonists. ***(Refer 2010/2019 MUHS)***

Ans. Endothelial receptor antagonists are a class of medications that target receptors on the endothelial cells lining blood vessels. These receptors play crucial roles in regulating vascular tone, endothelial function, and cardiovascular homeostasis. By blocking these receptors, endothelial receptor antagonists exert various effects on the cardiovascular system, making them valuable therapeutic agents for several cardiovascular conditions. Two prominent types of endothelial receptor antagonists are—(1) endothelin receptor antagonists (ERAs) and (2) prostaglandin receptor antagonists.

Endothelin Receptor Antagonists

Endothelin is a potent vasoconstrictor peptide produced by endothelial cells, and dysregulation of the endothelin system is implicated in various cardiovascular diseases. ERAs selectively block endothelin receptors, primarily endothelin receptor type A (ETA) and type B (ETB), thereby inhibiting the vasoconstrictive and profibrotic effects of endothelin.

Examples of ERAs:

- Bosentan
- Ambrisentan
- Macitentan

Clinical uses:

- *Pulmonary arterial hypertension (PAH)*: ERAs are approved for the treatment of PAH, where they help dilate pulmonary arteries, reduce pulmonary vascular resistance, and improve exercise capacity and quality of life in affected individuals.
- *Digital ulcers in systemic sclerosis:* Bosentan is approved for the treatment of digital ulcers in patients with systemic sclerosis (scleroderma).

Adverse effects: Common adverse effects of ERAs include liver enzyme abnormalities, peripheral edema, headache, nasal congestion, and anemia.

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