ANESTHESIA MANAGEMENT FOR CONGENITAL HEART DISEASE

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ABSTRACT:

The anesthetic management of pediatric patients with congenital heart disease (CHD) requires a thorough understanding of the patient's unique anatomical variations, the planned surgical intervention, and associated physiological changes. CHD is broadly categorized as either cyanotic or a cyanotic. Cyanotic CHD results from the admixture of oxygenated and deoxygenated blood within the systemic circulation. Examples of cyanotic CHD include Tetralogy of Fallot, Double Outlet Right Ventricle (DORV), Total Anomalous Pulmonary Venous Return (TAPVR), Tricuspid Atresia, Critical Pulmonary Stenosis, Pulmonary Atresia, and Hypoplastic Left Heart Syndrome. A cyanotic CHD encompasses conditions such as Atrial Septal Defect, Ventricular Septal Defect, and Patent Ductus Arteriosus.

Pre-anesthetic evaluation is essential for determining the patient's pre-operative physical status, assessing the specific congenital anomaly, and evaluating the severity of the cardiac defect. This information allows for meticulous perioperative planning. Intraoperative management must consider the dynamics of shunt flow in CHD, which depends upon the shunt's anatomical characteristics, specifically its size and configuration.

While inhalational agents are frequently employed for induction and maintenance of anesthesia in pediatric cardiac cases, patients exhibiting compromised ventricular function or dependence on systemic vascular resistance (SVR) and/or pulmonary vascular resistance (PVR) necessitate pre-induction intravenous access. In these patients, anesthetic agents that could further impair hemodynamic stability should be avoided or used judiciously. Post-induction anesthetic choices are guided by ventricular function (including the presence or absence of congestive heart failure), the anticipated need for cardiopulmonary bypass, and the probability of requiring mechanical ventilation or tracheal extubating post-operatively.

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KEYWORDS: Congenital Heart Disease, Cyanotic, A cyanotic, Shunt, Eisenmenger Syndrome, Pulmonary Hypertension, PaO_2, SaO_2.

INTRODUCTION

Anesthetic management in children with congenital heart disease (CHD) necessitates a comprehensive understanding of anatomical abnormalities, planned surgical procedures, and physiological alterations. The overall incidence of CHD varies between 4 and 12 per 1,000 live births.[1]

CHD can be classified as cyanotic or a cyanotic. Cyanotic conditions in CHD arise from a mixture of oxygenated and deoxygenated blood in the systemic circulation. It is essential to differentiate between cyanotic lesions (where the abnormality itself causes a right-to-left shunt) and cyanotic conditions resulting from a cyanotic lesion (such as Eisenmenger syndrome).[2]

Anesthetic management of patients with CHD is tailored to the underlying abnormality and its severity (e.g., Eisenmenger syndrome, and pulmonary hypertension). Oxygen delivery depends on cardiac output and oxygen content. Low PaO_2 and SaO_2 levels can significantly compromise oxygen content in cyanotic CHD patients. Chronic hypoxemia often leads to compensatory increases in hemoglobin concentration to maintain adequate oxygen delivery.[3]

DISCUSSION

1. Anesthesia in Cyanotic Congenital Heart Disease

The underlying cause of cyanosis in congenital heart disease (CHD) is a mixture of oxygenated and deoxygenated blood within the systemic circulation. Types of cyanotic CHD include:

1. Tetralogy of Fallot
2. Double outlet right ventricle (DORV)
3. Total anomalous pulmonary venous return (TAPVR)
4. Tricuspid atresia
5. Critical pulmonary stenosis

6. Pulmonary atresia
7. Hypoplastic left heart syndrome

A comprehensive medical history and physical examination are essential to establish the cardiac diagnosis and assess implications for anesthetic management. A detailed, written cardiac evaluation by a cardiologist is necessary for patients with complex heart defects.[1,4]

Premedication

The primary objective of premedication is to mitigate both physical and psychological stress experienced before and during anesthetic induction. In compensated pediatric patients with congenital heart disease (CHD), standard dosages of medications such as oral midazolam (0.5 mg/kg) can be administered. However sedatives generally induce vasodilation. Consequently, supplemental oxygen should be readily available to maintain adequate oxygen saturation, if necessary.[5,6]

Prophylactic Antibiotics

Bacterial endocarditis is a serious complication of CHD. Patients undergoing surgical procedures involving mucosal surfaces or contaminated tissues are at risk of transient bacteremia, which can lead to endocarditis. Cyanotic CHD patients are at particularly high risk and should receive prophylactic antibiotics.[7]

Intraoperative Management

The primary objectives of intraoperative management in CHD patients, whether palliated or undergoing non-cardiac surgery, are to maintain adequate oxygenation and ventilation, and to manage arrhythmias and fluid overload. Oxygenation is generally a more critical concern in patients with right-to-left shunts, while fluid overload and heart failure are more prominent challenges in patients with obstructive lesions. In patients with low pulmonary blood flow and right-to-left shunts, one of the goals of anesthesia is to reduce or at least not exacerbate the shunt. It is also important to avoid factors that increase systemic oxygen demand.[5,6]

Anesthetic Agents

The selection of anesthetic agents for pediatric patients with CHD depends on the type of surgery, anticipated duration of surgery, anesthetic considerations, and the patient's cardiovascular status. The rate of inhalation anesthetic induction is determined by the inspired anesthetic concentration and the transfer of the anesthetic from the lungs to the blood. [1,8]

In pediatric CHD patients with low pulmonary blood flow, inhalation induction in patients with right-to-left shunts may be prolonged. Higher concentrations of dissolved anesthetic, combined with ventilation, may be used to overcome this effect. Although all volatile agents are myocardial depressants, their effects on cardiac output and arterial blood pressure are modified by other factors, such as their effects on systemic vascular resistance. [1,4]

The decrease in blood pressure caused by halothane is due to a decrease in cardiac output, while the decrease in isoflurane levels is due to a decrease in systemic vascular resistance. Sevoflurane is considered superior to halothane in terms of causing bradycardia, dysrhythmias, and decreased cardiac output. Remifentanil can be used in children undergoing short surgical procedures without prolonging recovery time. Ketamine can be administered intramuscularly for induction of anesthesia (4-8 mg/kg) or sedation (2-3 mg/kg) in uncooperative pediatric CHD patients. The dose of thiopental (4-6 mg/kg IV) should be reduced in children with poor circulatory function. [1,4]

The selection of muscle relaxants in pediatric CHD patients is typically based on their cardiovascular effects and duration of action. Succinylcholine can cause bradycardia or cardiac arrest in children. Its use should be preceded by or combined with atropine. Mivacurium, atracurium, vecuronium, and rocuronium can be used with minimal hemodynamic effects. [1,4]

Table 1: Desired Hemodynamic Parameter Changes in Congenital Heart Disease (CHD)

	Preload	PVR	SVR	Heart Rate	Contractility
Pulmonary stenosis	↑	N	N	* ↓	* ↓
Infundibular	↑	↑	↓	N	N
Pulmonary stenosis	↑	N	↓	N	N

Postoperative Management and Analgesia

Children with cyanotic CHD often require hospitalization to ensure adequate intravenous hydration, even after minor surgical procedures. Prevention of postoperative nausea and vomiting is essential to ensure adequate oral intake and hydration. Patients who have undergone extensive surgery and/or have decompensated cardiac status may require intensive care postoperatively. [1,4]

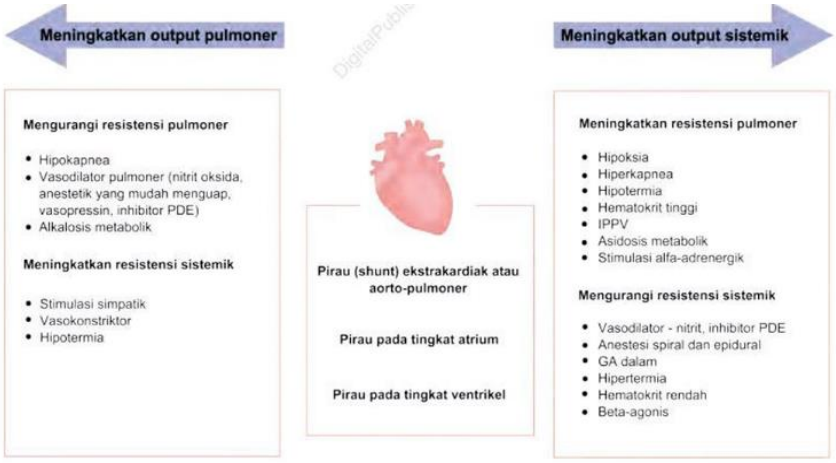
Acetaminophen is the most commonly used mild analgesic in pediatric patients. For younger children, the initial dose is usually administered rectally (up to 45 mg/kg) before awakening from anesthesia. Supplemental doses are given (10-20 mg/kg every 4-6 hours) to maintain adequate blood levels and provide effective analgesia. NSAIDs, such as ketorolac, have been shown to reduce postoperative pain in children effectively. Early administration, immediately after induction, appears to provide optimal postoperative analgesia. Fentanyl, up to a dose of 2.0 µg/kg, is the drug of choice for intravenous use. Codeine (1.0-1.5 mg/kg) can be administered intramuscularly if intravenous access is not available and may reduce vomiting compared to other opioids, especially morphine. [1,2]

Regional anesthesia can be combined with light general anesthesia to reduce postoperative pain. Regional block techniques performed before surgery begin to reduce the need for general anesthesia, resulting in better hemodynamic stability, faster recovery, earlier discharge, and reduced nausea and vomiting. The types of blocks that can be safely used in CHD patients are limited by the anesthesiologist's skills and preferences. Simple infiltration of the surgical site with a local anesthetic reduces postoperative pain after procedures such as tooth extraction and excision of superficial lesions. [1,2]

2. Anesthesia in A cyanotic Congenital Heart Disease

The pre-anesthetic evaluation aims to determine the patient's physical status before surgery, the condition of preoperative abnormalities, and the severity of the heart defect. The severity of the disease is correlated with post-anesthesia mortality. With this knowledge, perioperative planning is expected to be considered more carefully. [1,2]

The shunt flow in CHD depends on the anatomical shape of the shunt, namely the size of the shunt and the channel. The smaller the size and the absence of a channel (for example, in a small VSD), the smaller the pressure difference (PVR and SVR) between the two sides has a small effect on the degree and direction of the shunt. In very large sizes, there is no pressure difference between the chambers and the flow that occurs is a bidirectional flow that is highly dependent on the ratio of pulmonary to systemic pressure. [1,4]



Increased pulmonary blood flow can increase the workload on the pulmonary circulation, leading to adverse effects such as pulmonary edema or pulmonary hypertension, which can cause desaturation and decreased oxygen delivery to tissues. Conversely, increased systemic flow, which has the effect of decreasing pulmonary flow, will have the opposite effect. [1,4]

Propofol has the effect of reducing systemic vascular resistance (SVR) and mean arterial pressure (MAP), which can lead to increased shunt flow. Conversely, ketamine has a minimal effect on changes in SVR, pulmonary vascular resistance (PVR), and MAP, making it a preferred choice for patients who cannot tolerate a decrease in SVR or for patients with pulmonary

hypertension. Inhalation anesthetics, such as isoflurane and sevoflurane, are often used in cases of CHD due to their minimal effects on myocardial contractility and shunt fraction. The administration of 100% oxygen and hyperventilation should be avoided as they can cause vasodilation and worsen pulmonary congestion. [1,4]

Pulmonary Hypertension

The definition of pulmonary hypertension is a mean pulmonary arterial pressure > 25 mmHg at rest and > 30 mmHg during activity. Management of patients with pulmonary hypertension includes.[9,10]:

1. Oxygenation with FiO_2 0.6-1.0.
2. Moderate hyperventilation (target: $PaCO_2$ 30-35 mmHg).
3. Avoid metabolic acidosis (pH > 7.4).
4. Recruitment maneuvers to avoid V/Q mismatch.
5. Ventilation with low tidal volume to avoid alveolar overinflation (target 6-8 ml/kg body weight).
6. Maintain body temperature between 36-37°C. Optimal fluid administration (goal-directed fluid & volume therapy).

1. Atrial Septal Defect (ASD)

There is a left-to-right shunt at the atrial level with right ventricular volume overload. In early life, symptoms are not severe because the shunt is small, the right ventricle is not yet hypertrophy, and pulmonary artery pressure is relatively normal. If the defect is not corrected while the patient is young, shunt flow can become bidirectional due to right ventricular hypertrophy and increased pulmonary artery pressure. If left uncorrected, Eisenmenger syndrome occurs with persistently higher pulmonary than systemic arterial pressure and subsequent severe desaturation. [1,4]

2. Ventricular Septal Defect (VSD)

A left-to-right shunt causes increased pulmonary blood flow, increased volume overload in both ventricles, and increased workload on both ventricles. High pulmonary blood flow decreases lung compliance, increases work of breathing, and can lead to respiratory failure. Premedication depends on the severity of ventricular dysfunction. Induction, if left ventricular function is good,

can use inhalation induction. If left ventricular function is poor, ketamine/opioid is a better choice. In large and long ventricular septal defects, ventilation needs to be adjusted to maintain normocapnia and limit FiO_2 to prevent a decrease in pulmonary vascular resistance and further increase in pulmonary blood flow (pulmonary steal). [1,4]

3. Patent Ductus Arteriosus (PDA)

PDA occurs due to the failure of the ductus arteriosus to close. There is a bidirectional shunt between the main pulmonary artery and the descending aorta while pulmonary vascular resistance is high. The left-to-right shunt will dominate when pulmonary vascular resistance decreases. Increased pulmonary blood flow causes pulmonary hypertension, increased pulmonary fluid, and increased respiratory work. Corrective therapy includes fluid restriction, oxygen supplementation, and indomethacin. Maintain SaO_2 at 85-90% to minimize the risk of retrolental fibroplasia. Moore and Nicholson have created a heart table for common congenital heart diseases regarding hemodynamic changes in each abnormality. [1,4]

CONCLUSIONS

Anesthetic management of patients with CHD is tailored to the underlying abnormality and its severity (e.g., Eisenmenger syndrome, and pulmonary hypertension). Oxygen delivery depends on cardiac output and oxygen content. Low PaO_2 and SaO_2 levels can significantly compromise oxygen content in cyanotic CHD patients. Chronic hypoxemia often leads to compensatory increases in hemoglobin concentration to maintain adequate oxygen delivery.

Further research on Anesthetic management in children with congenital heart disease (CHD) could enhance patient care by better understanding its benefits and risks.

CONFLICT OF INTEREST

This review article does not contain any conflicts of interest

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