Saudi Journal of Medicine and Public Health

https://saudijmph.com/index.php/pub

https://doi.org/10.64483/202522527

Advanced Perioperative Nursing Care and Multidisciplinary Coordination in the Management of Patients With Congenital Heart Disease

Amal Ayyadah S. Alhazmi [(1)] , Khloud Mazki Alenazi [(2)] , Ahmed Ali Ahmed Eshwi [(3)] , Shaqra Mohammed Mohammed Awaji [(4)] , Ibrahim Abdullah Al-Zubni [(5)] , Ahmed Hassan Alrashidi [(6)] , Rahma Serihan Alharbi [(7)] , Areej Ali Saleh Shook [(8)] , Mohammed Hamdan Abdulhadi Alosaimi [(9)] , Sakinah Naif Alnakhli [(10)] , Sara Mohammed Mahzari [(8)] , Sohair Yahya Awam [(8)]

(1) Amayalhazmi@moh.gov.sa, Saudi Arabia,
(2) Women and Maternity Hospital, Ministry of Health, Saudi Arabia,
(3) King Fahd Central Hospital – Jazan, Ministry of Health, Saudi Arabia,
(4) Al-Twal General Hospital – Jazan, KSA, Ministry of Health, Saudi Arabia,
(5) Hail – Ambulance Transport, Saudi Arabia,
(6) Aradah Mental Health Complex – Riyadh, Ministry of Health, Saudi Arabia,
(7) Maternity and Children Hospital – King Salman Medical City, Ministry of Health, Saudi Arabia,
(8) King Fahad Hospital, Ministry of Health, Saudi Arabia,
(9) Dawadmi General Hospital – Dawadmi, Riyadh Third Health Cluster, Saudi Arabia,
(10) Al Salam Hospital, Ministry of Health, Saudi Arabia

Abstract

Background: Congenital heart disease (CHD) encompasses diverse structural and functional cardiac anomalies that significantly impact neonatal and pediatric morbidity and mortality.

Aim: To explore advanced perioperative nursing care and multidisciplinary coordination in managing CHD patients undergoing surgical interventions.

Methods: A comprehensive review of perioperative strategies, including preoperative evaluation, intraoperative monitoring, and postoperative management, was conducted, emphasizing individualized care and team collaboration.

Results: Advances in diagnostic imaging, cardiopulmonary bypass, and minimally invasive techniques have improved surgical outcomes. However, success relies on tailored perioperative planning, vigilant monitoring, and effective interdisciplinary communication. Key challenges include hemodynamic instability, airway management, and postoperative complications such as arrhythmias and pulmonary issues.

Conclusion: Optimal outcomes in CHD surgery require meticulous nursing care, adaptive strategies, and coordinated teamwork to ensure safety and enhance survival.

Keywords: Congenital heart disease, perioperative care, pediatric cardiac surgery, nursing, multidisciplinary coordination.

Introduction

Congenital heart disease represents a heterogeneous group of structural and functional abnormalities affecting the heart and great vessels that arise during fetal development. These conditions vary widely in anatomical complexity, physiological impact, and clinical presentation, ranging from mild defects with minimal functional consequences to severe malformations that threaten survival early in life. Although congenital heart disease is generally considered uncommon, its clinical and public health significance is substantial. Epidemiological data indicate that approximately 1 percent of live births in the United States are affected annually. Despite advances in diagnosis and treatment, congenital heart disease remained a leading cause of mortality during early life between 1999 and 2006, accounting for 27,960 deaths, with nearly half occurring in infants. This high mortality burden reflects the vulnerability of this population, particularly in the context of complex surgical interventions and prolonged admissions to intensive care units, where physiological instability and postoperative complications are common [1]. Accurately determining the true incidence of congenital heart disease remains challenging. A proportion of affected fetuses do not survive to term due to defects that are incompatible with life, resulting in fetal loss that is often underreported. In addition, certain cardiac anomalies, such as bicuspid aortic valve or patent ductus arteriosus, may remain clinically silent during infancy and early childhood, delaying diagnosis until later stages of life. These factors contribute to underestimation of prevalence and complicate population-level surveillance.

*Corresponding author e-mail: Amayalhazmi@moh.gov.sa (Amal Ayyadah S. Alhazmi).

Receive Date: 06 December 2025, Revise Date: 28 December 2025, Accept Date: 31 December 2025

Furthermore, the distribution and pattern of congenital heart disease vary across populations, influenced by genetic, environmental, and perinatal factors. Evidence demonstrates a higher prevalence of congenital anomalies, including cardiac defects, among preterm infants and those who are small for gestational age when compared with full-term neonates, underscoring the interaction between fetal growth, developmental vulnerability, and cardiac morphogenesis [2].

The perioperative management of children with congenital heart disease poses distinctive clinical challenges for healthcare professionals involved in surgical care. The presence of altered cardiovascular physiology limited physiological reserve, and the frequent coexistence of pulmonary, neurological, or metabolic comorbidities demands a high level of expertise and coordination. Over recent decades, significant progress has been achieved in diagnostic imaging, perioperative monitoring technologies, cardiopulmonary bypass systems, and cardiac catheterization techniques. These advances have expanded surgical eligibility to increasingly complex and critically ill pediatric patients. In parallel, the development of minimally invasive and hybrid procedures has reduced surgical trauma and improved recovery profiles for selected children with congenital cardiac lesions [3]. Despite these technological and procedural improvements, successful perioperative care depends heavily on individualized clinical planning. Each child with congenital heart disease presents a unique combination of anatomical features, hemodynamic consequences, and developmental considerations. As a result, standardized or rigid approaches are insufficient and may compromise patient safety. Instead, perioperative strategies must be tailored to the specific pathophysiology of the defect, the planned surgical intervention, and the child's overall clinical condition. This individualized approach requires adaptability, critical thinking, and continuous reassessment throughout the perioperative period, particularly during periods of physiological stress such as induction of anesthesia, cardiopulmonary bypass, and postoperative stabilization.

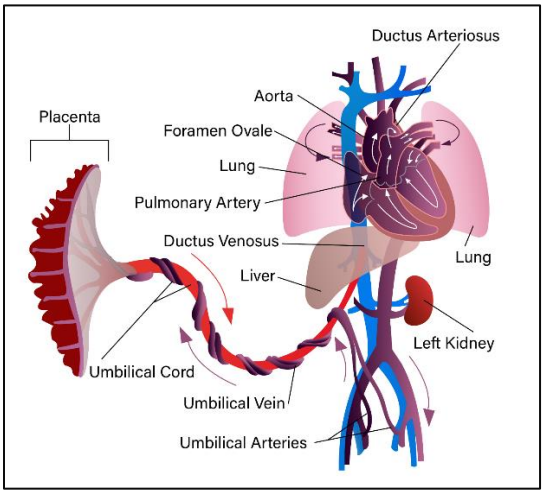

Fig. 1: Fetal blood circulation.

Equally important is the role of interdisciplinary collaboration in optimizing outcomes for this vulnerable population. Effective communication and coordinated teamwork among nurses, anesthesiologists, surgeons, perfusionists, and intensive care specialists are fundamental to the success of pediatric cardiac surgical programs. Early identification and timely surgical correction of congenital heart defects, when feasible, can prevent progression to congestive heart failure, irreversible pulmonary vascular disease, and other long-term complications. In cases where conventional surgical repair is unsuccessful or contraindicated, cardiac transplantation may represent a viable therapeutic option for selected patients [4]. Regardless of the chosen intervention, meticulous perioperative planning, vigilant monitoring, and individualized nursing and medical care remain essential to achieving favorable outcomes and improving survival and quality of life for children living with congenital heart disease [1][2][3][4].

Function

Congenital heart defects alter normal cardiac function by disrupting the structural integrity and coordinated performance of the heart chambers, valves, and major vessels. Abnormal development of cardiac architecture interferes with the normal separation of the cardiac segments, impairs venous and arterial blood flow, and compromises the functional competence of the valvular apparatus [5]. These structural disturbances result in altered hemodynamics that place an increased workload on the myocardium and reduce the efficiency of systemic and pulmonary circulation. The physiological consequences depend on the type, size, and location of the defect, as well as the degree to which normal blood flow patterns are disturbed. From a functional perspective, the American Heart Association categorizes congenital cardiac defects into three broad groups: septal defects, obstructive lesions, and cyanotic malformations. Septal defects involve abnormal communications between the right and left sides of the heart. Under normal postnatal conditions,

the septum provides complete separation between systemic and pulmonary circulations. Persistence of an opening after birth leads to pathological shunting of blood. Common examples include patent foramen ovale, atrial septal defect, ventricular septal defect, and complete atrioventricular septal defect. These conditions often result in left-to-right shunting, increased pulmonary blood flow, and progressive volume overload of the affected cardiac chambers. Over time, untreated septal defects may lead to pulmonary hypertension, ventricular dysfunction, and heart failure. Obstructive congenital heart anomalies primarily affect forward blood flow by narrowing or blocking outflow tracts. Conditions such as aortic stenosis, pulmonary stenosis, and coarctation of the aorta impose resistance to ventricular ejection. This resistance increases intracardiac pressures and leads to compensatory myocardial hypertrophy. Although cardiac output may initially be preserved, prolonged obstruction compromises ventricular performance and systemic perfusion. The severity of functional impairment depends on the degree of obstruction and the ability of the heart to compensate during growth and development [4][5].

Cyanotic congenital heart defects are characterized by inadequate oxygenation of arterial blood, resulting in clinically evident cyanosis. These conditions typically involve right-to-left shunting or parallel circulation that prevents effective pulmonary oxygen exchange. Disorders such as tetralogy of Fallot, transposition of the great arteries, tricuspid atresia, pulmonary atresia, truncus arteriosus, total anomalous pulmonary venous connection, and hypoplastic left heart syndrome represent some of the most physiologically complex congenital anomalies [6]. In these defects, systemic circulation receives partially or completely deoxygenated blood, leading to chronic hypoxemia. The functional consequences include impaired tissue oxygen delivery, metabolic acidosis, growth failure, and increased risk of neurological and multisystem complications. Functionally, cyanotic and non-cyanotic defects differ not only in oxygenation status but also in their long-term impact on cardiac performance and systemic health. Non-cyanotic lesions often present with signs of volume or pressure overload, whereas cyanotic lesions present with hypoxia-driven compensatory mechanisms such as polycythemia. Understanding these functional distinctions is essential for clinical assessment, perioperative planning, and long-term management, as the physiological burden imposed by each defect directly influences therapeutic strategies and patient outcomes [6].

Issues of Concern

Physiology of Fetal Circulation

A detailed understanding of the physiological differences between fetal and postnatal circulation is essential for interpreting the clinical behavior of congenital heart disease and for planning safe anesthetic and perioperative management. Fetal circulation is uniquely designed to support intrauterine growth and development in an environment where the lungs are nonfunctional, and oxygen exchange occurs through the placenta. This system allows adequate oxygen delivery to vital organs despite the absence of pulmonary respiration and remains effective even in the presence of significant structural cardiac abnormalities. The fetal cardiovascular system operates as a parallel circuit, in contrast to the series circulation observed after birth, and this distinction has major implications for the pathophysiology of congenital heart disease. In fetal life, specialized vascular channels divert blood away from the lungs and partially bypass the liver, optimizing delivery of oxygen to the brain and myocardium. Three essential shunts enable this process: the ductus venosus, the foramen ovale, and the ductus arteriosus. Oxygenated blood from the placenta enters the fetus through the umbilical vein with a partial pressure of oxygen of approximately 33 mmHg. Upon reaching the liver, nearly half of this blood bypasses hepatic circulation through the ductus venosus and flows directly into the inferior vena cava. This preferential streaming ensures that relatively well-oxygenated blood reaches the heart and upper body structures. Within the right atrium, blood flow is strategically directed. Approximately one-third of the blood entering the right atrium from the inferior vena cava crosses the foramen ovale into the left atrium, from where it enters the left ventricle and is pumped into the ascending aorta. This pathway supplies the coronary arteries, brain, and upper extremities with blood that has the highest available oxygen content. In contrast, blood returning from the superior vena cava is poorly oxygenated and predominantly enters the right ventricle. Only a small fraction, estimated at 2 to 3 percent, crosses the foramen ovale. The right ventricle ejects blood into the pulmonary artery, but due to the high pulmonary vascular resistance present in utero, most of this blood bypasses the lungs through the ductus arteriosus and enters the descending aorta [6].

This anatomical and physiological arrangement results in differential oxygen delivery, with the upper body receiving relatively oxygen-rich blood and the lower body being perfused with blood of lower oxygen content. Such selective distribution is critical for protecting organs with high metabolic demands during fetal development. In this context, pulmonary and systemic circulations function parallel, allowing both ventricles to contribute to systemic output. This is fundamentally different from postnatal circulation, where pulmonary and systemic circuits operate in series, and the entire cardiac output must pass sequentially through the lungs and then the systemic circulation [7]. At birth, the cardiovascular system undergoes a rapid and complex transition that enables survival outside the uterine environment. This transition is driven by the initiation of breathing,

clamping of the umbilical cord, and dramatic changes in vascular resistance. Although this process is essential, it is also inherently unstable and represents a critical period of vulnerability. The duration of transitional circulation varies widely, lasting from a few hours to several weeks, depending on physiological maturity and environmental stresses. Factors such as prematurity, hypoxia, hypothermia, hypercarbia, metabolic acidosis, sepsis, and underlying congenital heart defects can delay or disrupt this transition, prolonging fetal circulatory patterns [7]. One of the most significant changes after birth is the functional closure of the ductus arteriosus. This vessel typically constricts within hours in response to increased oxygen tension and reduced circulating prostaglandins, although complete anatomical closure may take several weeks. In infants with certain forms of cyanotic congenital heart disease, ductal patency becomes essential for survival, as it may provide the only source of pulmonary or systemic blood flow until definitive or palliative surgical intervention is performed. Pharmacologic maintenance of ductal patency is therefore a critical component of early management in these cases [7].

The foramen ovale also undergoes functional closure shortly after birth as left atrial pressure rises above right atrial pressure due to increased pulmonary venous return. While functional closure usually occurs within the first few breaths, anatomical fusion of the septa may take months to complete. Notably, probe patency of the foramen ovale persists in approximately 30 percent of adults, highlighting the incomplete nature of anatomical closure in a substantial portion of the population [9]. The umbilical arteries and vein close immediately after birth, followed by closure of the ductus venosus, further reinforcing separation of fetal and postnatal circulatory pathways. Pulmonary vascular resistance plays a central role during this transitional phase. In utero, pulmonary resistance is high, but it falls rapidly after birth as the lungs expand and oxygenation improves. Within the first 24 hours, pulmonary vascular resistance decreases below systemic levels and continues to decline over the following weeks, with further gradual reduction over the next two to three years. During this period, the pulmonary vasculature remains highly reactive. Hypoxemia, acidosis, bronchospasm, or inadequate ventilation can trigger pulmonary vasoconstriction, leading to elevated pulmonary artery pressures. Such changes may promote right-to-left shunting across the foramen ovale or ductus arteriosus, reestablishing fetal circulatory patterns and compromising systemic oxygen delivery [7]. Understanding the physiology of fetal and transitional circulation is therefore fundamental to recognizing the clinical manifestations of congenital heart disease and anticipating hemodynamic instability. This knowledge is especially critical in perioperative and anesthetic settings, where alterations in oxygenation, ventilation, and vascular tone can profoundly influence shunt dynamics and cardiac output in neonates and infants with congenital cardiac anomalies [7][8][9].

Preoperative Evaluation

Preoperative evaluation of patients undergoing surgical repair of congenital heart disease is a critical process that requires careful integration of clinical, physiological, and diagnostic information. The population presenting for surgery spans a wide age range, most commonly including neonates, infants, and children, and represents a highly heterogeneous group. Variations in cardiac anatomy, hemodynamic physiology, body size, and developmental maturity necessitate an individualized approach to assessment. A thorough preoperative evaluation allows accurate risk stratification, optimization of the patient's condition before surgery, and formulation of an appropriate perioperative management plan. Clinical history forms the foundation of preoperative assessment and provides essential insight into disease severity and functional status. Symptoms such as palpitations, syncope, feeding intolerance, or poor weight gain may indicate significant hemodynamic compromise or arrhythmias. A detailed review of signs and symptoms suggestive of congestive cardiac failure, including tachypnea, diaphoresis, fatigue, or recurrent respiratory infections, is essential. The presence of associated congenital abnormalities involving the airway, genitourinary system, musculoskeletal structures, or genetic syndromes must be identified, as these conditions may influence anesthetic management and surgical planning. Documentation of recent hospital admissions, current medications such as diuretics, inotropes, or anticoagulants, and prior surgical or interventional procedures provides important context for perioperative decision-making. The most recent cardiology follow-up should be reviewed to confirm current cardiac status and progression of disease. A comprehensive physical examination complements the clinical history and helps define the physiological impact of the cardiac lesion. Cardiac auscultation may reveal murmurs, thrills, bruits, or irregular rhythms that suggest valvular pathology, septal defects, or arrhythmias. Respiratory assessment focuses on identifying tachypnea, rales, or increased breathing work, which may reflect pulmonary congestion or elevated pulmonary blood flow. Evaluation of peripheral perfusion is particularly important and includes assessment of pulse quality, capillary refill time, and the presence of bounding or diminished pulses. Findings such as cold extremities, excessive sweating, skin mottling, hepatomegaly, splenomegaly, or dependent edema are indicative of compromised cardiac output and systemic venous congestion [9].

Laboratory investigations provide objective data that support clinical findings and guide perioperative management. A complete blood count is essential to identify erythrocytosis in cyanotic heart disease or anemia that may impair oxygen delivery. Serum electrolyte analysis detects abnormalities such

as hypokalemia or hyponatremia, which are common in patients receiving diuretics and may increase perioperative risk. Assessment of coagulation parameters is particularly important in children with liver dysfunction, chronic hypoxemia, or those receiving anticoagulant therapy. Imaging and additional diagnostic studies play a central role in defining cardiac structure and function. Chest radiography may demonstrate cardiomegaly, pulmonary edema, or parenchymal infiltrates. Electrocardiography provides information regarding cardiac rhythm and evidence of atrial or ventricular hypertrophy. Echocardiography remains the cornerstone of preoperative cardiac evaluation, offering detailed assessment of anatomy, ventricular function, valvular competence, and intracardiac shunts. In selected patients, advanced studies such as cardiac catheterization are required to evaluate pressure gradients, oxygen saturations, shunt magnitude, and vascular resistances. Cardiac magnetic resonance imaging may be used to further delineate anatomy, quantify ventricular function, and assess pulmonary blood flow. Preoperative preparation focuses on maintaining physiological stability while minimizing perioperative risk. Fasting guidelines are applied according to established recommendations, with specific intervals for clear liquids, breast milk, formula, and solid foods in neonates, infants, and children. Intravenous maintenance fluids are initiated when appropriate to prevent dehydration and electrolyte imbalance. Premedication is considered on an individual basis, balancing anxiolysis and comfort against the risk of respiratory depression or hemodynamic instability. Through meticulous evaluation and preparation, the perioperative team can optimize outcomes for children undergoing surgery for congenital heart disease [9].

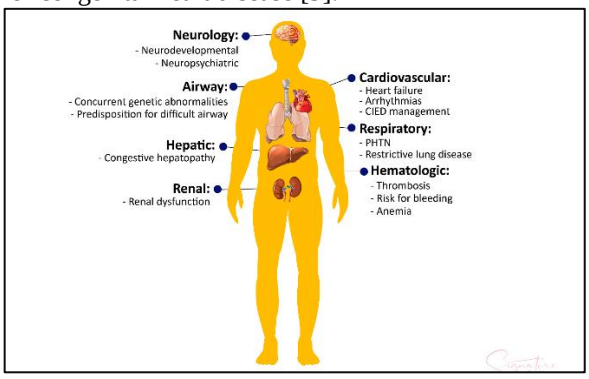

Fig. 2: Effects of congenital heart disorders.

Clinical History

Pediatric patients constitute the majority of individuals presenting for the correction of congenital heart defects, and their evaluation requires an age-specific and developmentally appropriate approach. Pediatric age groups are conventionally classified as neonates from birth to 28 days, infants from one month to one year, toddlers from one to four years, and children from four to thirteen years [7]. Each group demonstrates distinct physiological characteristics, compensatory mechanisms, and vulnerability to hemodynamic stress, which must be considered during preoperative assessment. A detailed and structured clinical history is therefore essential to establish baseline function, identify risk factors, and anticipate perioperative challenges. All pediatric patients scheduled for congenital cardiac surgery undergo a comprehensive preoperative assessment. This process includes a detailed review of current medications, known drug or food allergies, and previous hospital admissions, particularly those related to pulmonary infections or cardiac decompensation. Documentation of prior surgical procedures and anesthetic exposure is crucial, as it may reveal a history of difficult airway management, adverse anesthetic reactions, or complications related to cardiopulmonary bypass. Evaluation of cardiac functional reserve relies heavily on age-appropriate activity assessment. In infants and young children, feeding tolerance, weight gain, and activity during play provide indirect but reliable indicators of cardiac performance. Parental observations are of particular clinical value, as caregivers often recognize subtle changes such as worsening fatigue, poor feeding, excessive sweating, rapid breathing, inability to keep pace with siblings, or failure to thrive. These findings frequently reflect progressive cardiac failure and reduced physiological reserve [7].

Physical Examination

A thorough physical examination is indispensable for correlating historical findings with objective clinical signs and for identifying factors that may delay or contraindicate surgery. Assessment begins with measurement of age-appropriate vital signs and comparison of weight, height, and head circumference with standardized growth charts. Deviations from expected growth patterns may indicate chronic hypoxemia or prolonged heart failure. Key elements of the physical examination include evaluation for central or peripheral cyanosis, digital clubbing, signs of congestive cardiac failure, and assessment of potential monitoring and vascular access sites. Careful inspection of peripheral pulses and perfusion provides insight into cardiac output and systemic circulation. Approximately 8 percent of children with congenital heart disease have associated congenital anomalies, making a systematic examination of other organ systems essential [10]. Airway abnormalities are particularly relevant, especially in premature infants and children weighing less than 10 kilograms, as they may complicate airway management during anesthesia. Certain genetic syndromes have a strong association with congenital cardiac defects. For example, Trisomy 21 is associated with a 50 percent incidence of congenital heart disease, emphasizing the importance of physical examination in identifying syndromic features and

anticipating both cardiac and non-cardiac complications [11].

Laboratory Data

Laboratory investigations must be interpreted in close correlation with clinical findings. Children with congestive cardiac failure may present with iron deficiency anemia, which can further impair oxygen delivery. In contrast, cyanotic patients often exhibit erythrocytosis as a compensatory response to chronic hypoxemia, resulting in elevated hematocrit levels. Excessive erythrocytosis increases blood viscosity and may predispose to neurological complications, necessitating preoperative interventions such as exchange transfusion. Coagulation abnormalities, including prolonged prothrombin time and partial thromboplastin time, are also commonly observed in cyanotic heart disease and require careful evaluation. Close coordination between the anesthesiologist and surgeon is essential to ensure availability of blood and blood products, particularly in re-operative cases or procedures involving extensive dissection. Renal function assessment is a critical component of preoperative evaluation, as children with congenital heart disease are at increased risk of acute kidney injury during the perioperative period. Baseline serum creatinine and estimated glomerular filtration rate provide reference values for postoperative monitoring. Renal ultrasonography assists in excluding congenital anatomical abnormalities and supports early planning for renal replacement therapy if postoperative dialysis becomes necessary [12].

A chest radiograph remains a valuable screening tool, offering both anatomical and functional information. A systematic approach to interpretation includes assessment of imaging technique, cardiac position and situs, chamber size, pericardial contours, great vessel anatomy, and pulmonary vascularity. Although chest radiography demonstrates reproducibility in identifying abnormal pulmonary vascular patterns, its sensitivity for detecting pulmonary vascular disease ranges from 24 to 68 percent, limiting its diagnostic accuracy when used in isolation [13]. Electrocardiographic evaluation using a standard 12-lead ECG is fundamental for identifying arrhythmias, confirming sinus rhythm, and detecting evidence of chamber hypertrophy. More advanced diagnostic modalities are often required for comprehensive assessment. Cardiac catheterization and echocardiography provide essential data for understanding pathophysiology and guiding both surgical and anesthetic planning [14][15]. Cardiac catheterization yields detailed anatomical information, oxygen saturation measurements for calculating pulmonary-to-systemic flow ratios, pressure data for assessing gradients and shunts, and angiographic visualization of ventricular function and great vessel flow. It also allows evaluation of prior surgical interventions and assessment of reversible pulmonary hypertension [16]. Echocardiography serves as a cornerstone non-invasive modality in pediatric congenital heart disease. It defines both structural and functional characteristics, including cardiac position, atrial situs, atrioventricular and ventriculoarterial connections, and the spatial relationships of the great arteries [17]. Increasingly, cardiac magnetic resonance imaging complements these assessments by quantifying ventricular volumes, valve regurgitation, and blood flow while providing superior visualization of extracardiac vascular anatomy and postoperative scar tissue [18][19].

Preoperative Preparation

Preoperative preparation for patients undergoing surgery for congenital heart disease follows many of the same principles applied in adult practice; however, the complexity of pediatric physiology and the variability of cardiac pathology require a higher level of precision and anticipation. Meticulous preparation is essential to maintain hemodynamic stability, ensure patient safety, and support optimal surgical and anesthetic outcomes. Particular attention must be given to equipment readiness, medication preparation, fluid management, and psychological support, as these patients often present with limited physiological reserve. Complete and age-appropriate anesthesia equipment must be available before patient arrival in the operating room. This includes airway devices in multiple sizes, such as face masks, endotracheal tubes, laryngoscope blades, and supraglottic airway devices, to accommodate variations in patient size and anatomy. Emergency medications should be prepared in advance, with dosages calculated according to the patient's weight to prevent dosing errors during critical events. For critically ill children, anesthetist technicians or nurses often prepare selected infusions, such as inotropes or vasodilators, prior to induction to allow rapid initiation if hemodynamic compromise occurs. The use of appropriately sized intravenous catheters is essential, and air-trap filters are recommended to minimize the risk of air embolism, particularly in patients with intracardiac shunts. Fluid management and fasting protocols represent a critical component of preoperative preparation. Routine preoperative intravenous fluids and nil-per-os instructions are guided by age-specific requirements. The widely accepted 2,4,6,8 rule is applied to neonates, infants, and children with congenital heart disease, allowing clear liquids for up to two hours, breast milk up to four hours, formula milk up to six hours, and solid food up to eight hours before anesthesia [20]. Strict adherence to these guidelines reduces the risk of aspiration while minimizing unnecessary fasting that may predispose dehydration and hemodynamic instability.

Avoidance of dehydration is particularly important in children with congenital heart defects. Reduced intravascular volume can adversely affect cardiac output and exacerbate blood viscosity in patients with erythrocytosis. Neonates and infants require careful glucose supplementation to prevent hypoglycemia, which may negatively influence

neurological outcomes, especially during periods of deep hypothermic circulatory arrest. Perioperative glucose is commonly administered as part of maintenance fluid therapy, with 10 percent dextrose given at a rate of approximately 2 mL/kg/hour [21]. At the same time, close monitoring of blood glucose levels is essential, as patients receiving total parenteral nutrition or intralipid therapy are at increased risk of hyperglycemia. Psychological preparation and anxiolysis are also important aspects of preoperative care. Children with congenital heart disease often experience significant psychosocial stress due to repeated hospitalizations, multiple surgical procedures, and prolonged exposure to critical care environments. Premedication before induction of anesthesia can reduce anxiety, facilitate separation from parents, and allow smoother placement of intravenous access. In older children and adults without prior cardiac surgery, benzodiazepines such as midazolam may be effective when administered at appropriate doses [22]. However, patients with previous surgical experiences may exhibit heightened anxiety or inadequate response to benzodiazepines alone. In such cases, adjunctive agents such as oral or intranasal ketamine or intranasal dexmedetomidine are frequently employed to enhance sedation and anxiolysis while preserving spontaneous ventilation [23][24]. Premedication regimens must be carefully individualized to balance therapeutic benefit against the risk of adverse effects, including respiratory depression and hemodynamic instability. Administration and assessment of premedication should occur under the direct supervision of an anesthesiologist to ensure patient safety. Medication management on the day of surgery requires careful consideration. Many patients with congenital heart disease receive chronic therapy with diuretics, digoxin, or angiotensin-converting enzyme inhibitors. These medications are commonly withheld on the day of surgery to prevent exaggerated hemodynamic responses when combined with anesthetic agents. In contrast, essential infusions such as prostaglandins and inotropes are continued intraoperatively to maintain ductal patency and cardiac output. Through comprehensive and individualized preoperative preparation, the perioperative team can reduce complications and improve outcomes for patients undergoing surgery for congenital heart disease [21][22][23].

Intraoperative Monitoring:

Intraoperative monitoring is a cornerstone of anesthetic management in pediatric patients with congenital heart disease, yet the application of multiple monitors can complicate induction, particularly in minimally sedated neonates and infants. Monitoring is typically divided into pre- and post-induction phases. Pre-induction evaluation includes non-invasive measurements such as automated blood pressure, electrocardiography, pulse oximetry, and end-tidal carbon dioxide assessment. Following induction, invasive monitoring is established, most commonly through arterial catheterization of the radial or femoral arteries. In neonates, umbilical arterial access may be used temporarily before transitioning to peripheral arterial lines. Continuous invasive blood pressure monitoring, often at both upper and lower extremities, is essential for detecting residual aortic obstruction, including coarctation or arch abnormalities. Prior surgical interventions, such as Blalock-Taussig shunts or subclavian artery repairs, may alter local perfusion, requiring careful consideration for ipsilateral arterial monitoring. Central venous access is essential for nearly all pediatric cardiac procedures [25]. This access allows measurement of central venous pressure, administration of vasoactive medications and inotropes, delivery of blood products, and assessment of mixed venous oxygen saturation to estimate cardiac output and pulmonary-to-systemic flow ratios during cardiopulmonary bypass. Pulmonary artery catheters may occasionally be required for postoperative monitoring but are rarely indicated preoperatively. Temperature monitoring is conducted using rectal, esophageal, or nasopharyngeal probes, particularly during bypass, to guide thermal management [26]. Advanced monitoring modalities, including near-infrared spectroscopy, transcranial Doppler, and intraoperative transesophageal echocardiography (TEE), provide additional safety and functional assessment. Near-infrared spectroscopy offers continuous cerebral and somatic oxygenation monitoring [27], while transcranial Doppler facilitates assessment of cerebral blood flow and detection of microembolic events, refining post-bypass de-airing strategies [28]. TEE has transformed intraoperative decision-making by enabling real-time assessment of complex valvular repairs, left ventricular outflow reconstructions, and residual septal defects [29].

Airway management is fundamental in patients with congenital heart disease due to the impact of ventilatory parameters, oxygenation, and carbon dioxide tension on pulmonary vascular resistance. Poor lung compliance and high airway pressures are common in patients with pulmonary edema [30]. Mask ventilation must be performed cautiously to avoid gastric insufflation, often assisted by an oral airway to maintain patency. Nasal intubation is frequently preferred in children under three years to stabilize the airway and accommodate TEE, but caution is necessary in patients with elevated venous pressures, such as those with Glenn or Fontan physiology. Preoxygenation remains a priority even in patients receiving supplemental oxygen to maintain adequate vascular resistance and tissue oxygenation. Anesthetic induction is tailored to patient-specific factors including age, cardiac function, cyanosis, and emotional state. Intravenous induction is preferred in patients with severe ventricular dysfunction, often

employing opioids and muscle relaxants or etomidate. Ketamine is favored for maintaining systemic vascular resistance and minimizing pulmonary hypertension [32]. Volatile anesthetics such as sevoflurane are suitable for mask induction in patients with less severe compromise, whereas agents like thiopental and propofol are reserved for stable shunt lesions due to myocardial depression [33]. Maintenance of anesthesia combines opioid infusions with volatile agents, with careful titration during cardiopulmonary bypass to avoid hemodynamic instability. Cardiopulmonary bypass provides cardiac isolation and optimal surgical exposure while ensuring systemic perfusion, oxygenation, and carbon dioxide clearance [34]. Implementation of safety protocols and checklists is critical for preventing complications. Postoperative management requires infection prophylaxis due to immunosuppression from bypass, commonly using first- or second-generation cephalosporins for 24 to 72 hours [35][36]. Multidisciplinary care is essential postoperatively, including continuous monitoring of cardiac and end-organ function, early recognition of hemodynamic deviations, and prompt intervention to optimize recovery and surgical outcomes.

Postoperative Problems and Their Causes

Postoperative complications following congenital heart surgery are common and multifactorial, necessitating careful monitoring and prompt intervention. Hemodynamic instability represents a frequent concern. Hypertension may arise from anxiety, pain, hypervolemia, abrupt discontinuation of medications such as beta-blockers or ACE inhibitors, repair of aortic coarctation, seizures, or hypoglycemia. Conversely, hypotension is often associated with hypovolemia, low cardiac output due to myocardial dysfunction, pericardial effusion, arrhythmias, excessive blood loss, vasodilator therapy, diuresis, pneumothorax, sepsis, or shock. Central venous pressure (CVP) abnormalities provide additional insight into cardiac function. Elevated CVP may reflect inadequate sedation, right ventricular dysfunction, tamponade, or pulmonary complications, whereas low CVP generally indicates hypovolemia from fluid deficits, hemorrhage, or excessive drainage. Arterial oxygen saturation must also be interpreted within the context of the specific cardiac lesion. In univentricular hearts, saturation above 85% can signify an imbalance between pulmonary and systemic perfusion, while reductions may indicate basal atelectasis, hypoventilation, tube obstruction, pneumothorax, pleural effusion, pneumonia, pulmonary edema, hemorrhage, secretions, or right-to-left shunts. Tachyarrhythmias, including sinus or supraventricular tachycardia, and bradyarrhythmias such as atrioventricular block, frequently complicate the postoperative course. Elevated lactate levels may indicate systemic hypoperfusion, seizures, or mesenteric ischemia, highlighting the need for vigilant metabolic monitoring. Specific cardiac lesions carry unique postoperative risks and anesthetic considerations. Patent ductus arteriosus (PDA) typically requires conservative management initially, including fluid restriction and positive end-expiratory pressure, with indomethacin employed pharmacologically if necessary [37]. Surgical closure may be performed via thoracotomy or video-assisted thoracoscopic surgery, or percutaneous device closure under general anesthesia. Unrepaired PDA can result in pulmonary hypertension and ventricular dysfunction [38]. Atrial septal defects (ASD), particularly complete atrioventricular septal defects, result in substantial left-to-right shunting and potential heart failure as pulmonary vascular resistance falls [39]. Corrective surgery is typically performed between four and six months of age, with anesthetic management focusing on maintaining heart rate, contractility, preload, and a balanced pulmonary-to-systemic vascular resistance ratio.

Ventricular septal defects (VSD) are the most prevalent congenital heart defect, varying anatomically by location and size, which dictates the magnitude of shunting and timing of intervention [40][41]. Truncus arteriosus presents with early heart failure due to excessive pulmonary blood flow, requiring careful restriction of oxygen therapy and use of inotropes and afterload reducers [42][43]. Total anomalous pulmonary venous return constitutes a surgical emergency, often necessitating interventional stabilization prior to definitive repair [44][45]. Left ventricular outflow tract obstruction, coarctation of the aorta, Tetralogy of Fallot, transposition of the great arteries, tricuspid atresia, and Ebstein anomaly each require lesion-specific management strategies, including maintenance of ductal patency, careful ventilatory control, targeted inotropic support, and hemodynamic optimization [46–60]. Adults with congenital heart disease who have survived to adulthood pose additional challenges, particularly when presenting for noncardiac surgery. These patients may exhibit residual lesions, complex physiology, or prior palliative repairs, necessitating individualized anesthetic planning, often in adult congenital heart centers [61–66]. Anesthetic goals include optimizing systemic and pulmonary blood flow, maintaining appropriate vascular resistance, preventing air embolism, and ensuring adequate tissue oxygen delivery. Induction agents such as etomidate and ketamine are preferred in specific scenarios to preserve ventricular function and systemic vascular resistance [67][68]. Postoperative management in specialized cardiac intensive care units is critical for monitoring hemodynamics, arrhythmias, bleeding, and thromboembolic complications, ensuring timely intervention and favorable outcomes.

Conclusion:

Congenital heart disease presents complex physiological and surgical challenges that demand a highly individualized and multidisciplinary approach. Despite significant technological advancements,

patient safety and favorable outcomes hinge on comprehensive perioperative planning, vigilant monitoring, and seamless collaboration among healthcare professionals. Nurses play a pivotal role in ensuring stability during critical phases such as induction, cardiopulmonary bypass, and postoperative recovery. Early identification of complications, proactive management of hemodynamic changes, and adherence to evidence-based protocols are essential to minimize morbidity and mortality. Furthermore, psychological support for patients and families, combined with precise medication and fluid management, enhances overall care quality. As pediatric cardiac surgery evolves, continuous education, skill development, and integration of innovative monitoring techniques remain vital. Ultimately, individualized nursing care and interdisciplinary teamwork form the cornerstone of successful management, improving survival rates and quality of life for children with congenital heart disease.

References:

1. Hoffman JI, Kaplan S. The incidence of congenital heart disease. Journal of the American College of Cardiology. 2002 Jun 19:39(12):1890
2. Liu Y, Chen S, Zühlke L, Black GC, Choy MK, Li N, Keavney BD. Global birth prevalence of congenital heart defects 1970-2017: updated systematic review and meta-analysis of 260 studies. International journal of epidemiology. 2019 Apr 1:48(2):455-463. doi: 10.1093/ije/dyz009.
3. Divekar AA, Arar YM, Clark S, Tandon A, Zellers TM, Veeram Reddy SR. Transcatheter Device Therapy and the Integration of Advanced Imaging in Congenital Heart Disease. Children (Basel, Switzerland). 2022 Apr 2:9(4):. doi: 10.3390/children9040497.
4. Attenhofer Jost CH, Schmidt D, Huebler M, Balmer C, Noll G, Caduff R, Greutmann M. Heart transplantation in congenital heart disease: in whom to consider and when? Journal of transplantation. 2013:2013():376027. doi: 10.1155/2013/376027.
5. Thiene G, Frescura C. Anatomical and pathophysiological classification of congenital heart disease. Cardiovascular pathology : the official journal of the Society for Cardiovascular Pathology. 2010 Sep-Oct:19(5):259-74. doi: 10.1016/j.carpath.2010.02.006.
6. Donofrio MT, Moon-Grady AJ, Hornberger LK, Copel JA, Sklansky MS, Abuhamad A, Cuneo BF, Huhta JC, Jonas RA, Krishnan A, Lacey S, Lee W, Michelfelder EC Sr, Rempel GR, Silverman NH, Spray TL, Strasburger JF, Tworetzky W, Rychik J, American Heart Association Adults With Congenital Heart Disease Joint Committee of the Council on Cardiovascular Disease in the Young and Council on Clinical Cardiology, Council on Cardiovascular Surgery and Anesthesia, and Council on Cardiovascular and Stroke Nursing. Diagnosis and treatment of fetal cardiac disease: a scientific statement from the American Heart Association. Circulation. 2014 May 27:129(21):2183-242. doi: 10.1161/01.cir.0000437597.44550.5d.
7. Finnemore A, Groves A. Physiology of the fetal and transitional circulation. Seminars in fetal & neonatal medicine. 2015 Aug:20(4):210-6. doi: 10.1016/j.siny.2015.04.003.
8. Morton SU, Brodsky D. Fetal Physiology and the Transition to Extrauterine Life. Clinics in perinatology. 2016 Sep:43(3):395-407. doi: 10.1016/j.clp.2016.04.001.
9. Mojadidi MK, Ruiz JC, Chertoff J, Zaman MO, Elgendy IY, Mahmoud AN, Al-Ani M, Elgendy AY, Patel NK, Shantha G, Tobis JM, Meier B. Patent Foramen Ovale and Hypoxemia. Cardiology in review. 2019 Jan/Feb:27(1):34-40. doi: 10.1097/CRD.0000000000000205.
10. Foz C, Peyton J, Staffa SJ, Kovatsis P, Park R, DiNardo JA, Nasr VG. Airway Abnormalities in Patients With Congenital Heart Disease: Incidence and Associated Factors. Journal of cardiothoracic and vascular anesthesia. 2021 Jan:35(1):139-144. doi: 10.1053/j.jvca.2020.07.086.
11. Benhaourech S, Drighil A, Hammiri AE. Congenital heart disease and Down syndrome: various aspects of a confirmed association. Cardiovascular journal of Africa. 2016 Sep/Oct:27(5):287-290. doi: 10.5830/CVJA-2016-019.
12. Li S, Krawczeski CD, Zappitelli M, Devarajan P, Thiessen-Philbrook H, Coca SG, Kim RW, Parikh CR, TRIBE-AKI Consortium. Incidence, risk factors, and outcomes of acute kidney injury after pediatric cardiac surgery: a prospective multicenter study. Critical care medicine. 2011 Jun:39(6):1493-9. doi: 10.1097/CCM.0b013e31821201d3.
13. Tumkosit M, Yingyong N, Mahayosnond A, Choo KS, Goo HW. Accuracy of chest radiography for evaluating significantly abnormal pulmonary vascularity in children with congenital heart disease. The international journal of cardiovascular imaging. 2012 Jun:28 Suppl 1():69-75. doi: 10.1007/s10554-012-0073-x.
14. Godart F, Houeijeh A. [Interventional cardiac catheterization in congenital heart disease].

Presse medicale (Paris, France : 1983). 2017 May:46(5):497-508. doi: 10.1016/j.lpm.2016.11.013.
15. Finnerty DT, Griffin M. Recent Developments in Cardiology Procedures for Adult Congenital Heart Disease: The Anesthesiologist's Perspective. Journal of cardiothoracic and vascular anesthesia. 2021 Mar:35(3):741-751. doi: 10.1053/j.jvca.2020.07.037. Epub 2020 Jul 15
16. Kojima T, Imamura T, Osada Y, Muraji S, Nakano M, Oyanagi T, Yoshiba S, Kobayashi T, Sumitomo N. Efficacy of catheter interventions in the early and very early postoperative period after CHD operation. Cardiology in the young. 2018 Dec:28(12):1426-1430. doi: 10.1017/S1047951118001452.
17. Pacileo G, Di Salvo G, Limongelli G, Miele T, Calabrò R. Echocardiography in congenital heart disease: usefulness, limits and new techniques. Journal of cardiovascular medicine (Hagerstown, Md.). 2007 Jan:8(1):17-22
18. Fogel MA, Anwar S, Broberg C, Browne L, Chung T, Johnson T, Muthurangu V, Taylor M, Valsangiacomo-Buechel E, Wilhelm C. Society for Cardiovascular Magnetic Resonance/European Society of Cardiovascular Imaging/American Society of Echocardiography/Society for Pediatric Radiology/North American Society for Cardiovascular Imaging Guidelines for the Use of Cardiac Magnetic Resonance in Pediatric Congenital and Acquired Heart Disease: Endorsed by The American Heart Association. Circulation. Cardiovascular imaging. 2022 Jun:15(6):e014415. doi: 10.1161/CIRCIMAGING.122.014415.
19. Sachdeva S, Gupta SK. Imaging Modalities in Congenital Heart Disease. Indian journal of pediatrics. 2020 May:87(5):385-397. doi: 10.1007/s12098-020-03209-y.
20. Kafrouni H, Ojaimi RE. Preoperative Fasting Guidelines in Children: Should They Be Revised? Case reports in anesthesiology. 2018:2018():8278603. doi: 10.1155/2018/8278603.
21. Datta PK, Aravindan A. Glucose for Children during Surgery: Pros, Cons, and Protocols: A Postgraduate Educational Review. Anesthesia, essays and researches. 2017 Jul-Sep:11(3):539-543. doi: 10.4103/aer.AER_39_17.
22. Levine MF, Hartley EJ, Macpherson BA, Burrows FA, Lerman J. Oral midazolam premedication for children with congenital cyanotic heart disease undergoing cardiac surgery: a comparative study. Canadian journal of anaesthesia = Journal canadien d'anesthesie. 1993 Oct:40(10):934-8
23. Qiu L, Cao L, Lang Z, Li X, Lin H, Fan T. Preoperative sedation in children with congenital heart disease: 50% and 95% effective doses, hemodynamic effects, and safety of intranasal dexmedetomidine. Journal of clinical anesthesia. 2022 Oct:81():110908. doi: 10.1016/j.jclinane.2022.110908.
24. Stewart KG, Rowbottom SJ, Aitken AW, Rajendram S, Sudhaman DA. Oral ketamine premedication for paediatric cardiac surgery--a comparison with intramuscular morphine (both after oral trimeprazine). Anaesthesia and intensive care. 1990 Feb:18(1):11-4
25. Aiyagari R, Cooper DS, Jacobs JP. Vascular Access in Children With Congenital Heart Defects. Pediatrics. 2020 Jun:145(Suppl 3):S294-S295. doi: 10.1542/peds.2019-3474N.
26. Morettini E, Turchini F, Tofani L, Villa G, Ricci Z, Romagnoli S. Intraoperative core temperature monitoring: accuracy and precision of zero-heat flux heated controlled servo sensor compared with esophageal temperature during major surgery; the ESOSPOT study. Journal of clinical monitoring and computing. 2020 Oct:34(5):1111-1119. doi: 10.1007/s10877-019-00410-z.
27. Zaleski KL, Kussman BD. Near-Infrared Spectroscopy in Pediatric Congenital Heart Disease. Journal of cardiothoracic and vascular anesthesia. 2020 Feb:34(2):489-500. doi: 10.1053/j.jvca.2019.08.048.
28. Neunhoeffer F, Sandner K, Wiest M, Haller C, Renk H, Kumpf M, Schlensak C, Hofbeck M. Non-invasive assessment of cerebral oxygen metabolism following surgery of congenital heart disease. Interactive cardiovascular and thoracic surgery. 2017 Jul 1:25(1):96-102. doi: 10.1093/icvts/ivx080.
29. Friedberg MK. Imaging Right-Left Ventricular Interactions. JACC. Cardiovascular imaging. 2018 May:11(5):755-771. doi: 10.1016/j.jcmg.2018.01.028.
30. Apostolopoulou SC. The respiratory system in pediatric chronic heart disease. Pediatric pulmonology. 2017 Dec:52(12):1628-1635. doi: 10.1002/ppul.23900. E
31. Singh P, Chauhan S, Jain G, Talwar S, Makhija N, Kiran U. Comparison of cardioprotective effects of volatile anesthetics in children undergoing ventricular septal defect closure. World journal for pediatric & congenital heart surgery. 2013 Jan:4(1):24-9. doi: 10.1177/2150135112457580.

32. Baehner T, Kiefer N, Ghamari S, Graeff I, Huett C, Pflugradt S, Sendzik B, Heinze I, Mueller M, Schindler E, Duerr GD, Ellerkmann R, Velten M. A National Survey: Current Clinical Practice in Pediatric Anesthesia for Congenital Heart Surgery. World journal for pediatric & congenital heart surgery. 2020 May:11(3):257-264. doi: 10.1177/2150135120902122.
33. Joshi VS, Kollu SS, Sharma RM. Comparison of dexmedetomidine and ketamine versus propofol and ketamine for procedural sedation in children undergoing minor cardiac procedures in cardiac catheterization laboratory. Annals of cardiac anaesthesia. 2017 Oct-Dec:20(4):422-426. doi: 10.4103/aca.ACA_16_17.
34. Kumar TKS. Practical conduct of open heart procedures for congenital heart lesions. Journal of thoracic disease. 2020 Mar:12(3):1235-1259. doi: 10.21037/jtd.2019.11.80.
35. Yu X, Chen M, Liu X, Chen Y, Hao Z, Zhang H, Wang W. Risk factors of nosocomial infection after cardiac surgery in children with congenital heart disease. BMC infectious diseases. 2020 Jan 21:20(1):64. doi: 10.1186/s12879-020-4769-6.
36. Jaworski R, Dzierzanowska-Fangrat K, Czajkowska A, Maruszewski B, Kansy A. Cefazolin prophylaxis in children undergoing cardiac surgery with the use of cardiopulmonary bypass-is the dosing correct? European journal of cardio-thoracic surgery : official journal of the European Association for Cardio-thoracic Surgery. 2021 Dec 27:61(1):27-33. doi: 10.1093/ejcts/ezab251.
37. Gillam-Krakauer M, Reese J. Diagnosis and Management of Patent Ductus Arteriosus. NeoReviews. 2018 Jul:19(7):e394-e402. doi: 10.1542/neo.19-7-e394.
38. Conrad C, Newberry D. Understanding the Pathophysiology, Implications, and Treatment Options of Patent Ductus Arteriosus in the Neonatal Population. Advances in neonatal care : official journal of the National Association of Neonatal Nurses. 2019 Jun:19(3):179-187. doi: 10.1097/ANC.0000000000000590.
39. Bradley EA, Zaidi AN. Atrial Septal Defect. Cardiology clinics. 2020 Aug:38(3):317-324. doi: 10.1016/j.ccl.2020.04.001.
40. Rao PS, Harris AD. Recent advances in managing septal defects: ventricular septal defects and atrioventricular septal defects. F1000Research. 2018:7():. pii: F1000 Faculty Rev-498. doi: 10.12688/f1000research.14102.1.
41. Palladino-Davis AG, Davis CS. Outcomes of infants and children undergoing surgical repair of ventricular septal defect: a review of the literature and implications for research with an emphasis on pulmonary artery hypertension. Cardiology in the young. 2020 Jun:30(6):799-806. doi: 10.1017/S1047951120001146.
42. Taksande A, Jameel PZ. Critical Congenital Heart Disease in Neonates: A Review Article. Current pediatric reviews. 2021:17(2):120-126. doi: 10.2174/1573396317666210219162515.
43. Naimo PS, Konstantinov IE. Surgery for Truncus Arteriosus: Contemporary Practice. The Annals of thoracic surgery. 2021 May:111(5):1442-1450. doi: 10.1016/j.athoracsur.2020.06.036.
44. Files MD, Morray B. Total Anomalous Pulmonary Venous Connection: Preoperative Anatomy, Physiology, Imaging, and Interventional Management of Postoperative Pulmonary Venous Obstruction. Seminars in cardiothoracic and vascular anesthesia. 2017 Jun:21(2):123-131. doi: 10.1177/1089253216672442.
45. Gupta A, Mishra A, Shrivastava Y. Repair of intracardiac total anomalous pulmonary venous return. Multimedia manual of cardiothoracic surgery : MMCTS. 2021 Mar 8:2021():. doi: 10.1510/mmcts.2021.015.
46. Sun J, Brizard C, Winlaw D, Alphonso N, d'Udekem Y, Eastaugh L, Marathe S, Bell D, Ayer J. Biventricular repair versus Fontan completion for patients with d- or l-transposition of the great arteries with ventricular septal defect and left ventricular outflow tract obstruction. The Journal of thoracic and cardiovascular surgery. 2019 Oct:158(4):1158-1167.e1. doi: 10.1016/j.jtcvs.2019.05.061.
47. Meijs TA, van de Sande DAJP, Peek J, Limburg AS, Voskuil M. [Coarctation of the aorta as a cause of difficult-to-control hypertension]. Nederlands tijdschrift voor geneeskunde. 2021 Dec 9:165():. pii: D5417.
48. Holzer RJ, Gauvreau K, McEnaney K, Watanabe H, Ringel R. Long-Term Outcomes of the Coarctation of the Aorta Stent Trials. Circulation. Cardiovascular interventions. 2021 Jun:14(6):e010308. doi: 10.1161/CIRCINTERVENTIONS.120.010308.
49. Wise-Faberowski L, Asija R, McElhinney DB. Tetralogy of Fallot: Everything you wanted to know but were afraid to ask. Paediatric anaesthesia. 2019 May:29(5):475-482. doi: 10.1111/pan.13569.

50. Fanous E, Mogyorósy G. Does the prophylactic and therapeutic use of beta-blockers in preoperative patients with tetralogy of Fallot significantly prevent and treat the occurrence of cyanotic spells? Interactive cardiovascular and thoracic surgery. 2017 Oct 1:25(4):647-650. doi: 10.1093/icvts/ivx135.
51. Lee CH, Kwak JG, Lee C. Primary repair of symptomatic neonates with tetralogy of Fallot with or without pulmonary atresia. Korean journal of pediatrics. 2014 Jan:57(1):19-25. doi: 10.3345/kjp.2014.57.1.19.
52. Bauser-Heaton H, Ma M, Wise-Faberowski L, Asija R, Shek J, Zhang Y, Peng LF, Sidell DR, Hanley FL, McElhinney DB. Outcomes After Initial Unifocalization to a Shunt in Complex Tetralogy of Fallot With MAPCAs. The Annals of thoracic surgery. 2019 Jun:107(6):1807-1815. doi: 10.1016/j.athoracsur.2019.01.030.
53. Derrick G, Cullen S. Transposition of the Great Arteries. Current treatment options in cardiovascular medicine. 2000 Dec:2(6):499-506
54. Słodki M, Axt-Fliedner R, Zych-Krekora K, Wolter A, Kawecki A, Enzensberger C, Gulczyńska E, Respondek-Liberska M, International Prenatal Cardiology Collaboration Group. New method to predict need for Rashkind procedure in fetuses with dextro-transposition of the great arteries. Ultrasound in obstetrics & gynecology : the official journal of the International Society of Ultrasound in Obstetrics and Gynecology. 2018 Apr:51(4):531-536. doi: 10.1002/uog.17469.
55. Casanova J. The arterial switch operation. Revista portuguesa de cirurgia cardio-toracica e vascular : orgao oficial da Sociedade Portuguesa de Cirurgia Cardio-Toracica e Vascular. 2020 Jul-Sep:27(3):157
56. Sumal AS, Kyriacou H, Mostafa AMHAM. Tricuspid atresia: Where are we now? Journal of cardiac surgery. 2020 Jul:35(7):1609-1617. doi: 10.1111/jocs.14673.
57. Tutarel O. The Fontan operation: Possible anywhere? International journal of cardiology. 2021 Apr 1:328():96. doi: 10.1016/j.ijcard.2020.12.001.
58. Luedtke SA, Kuhn RJ, McCaffrey FM. Pharmacologic management of supraventricular tachycardias in children. Part 1: Wolff-Parkinson-White and atrioventricular nodal reentry. The Annals of pharmacotherapy. 1997 Oct:31(10):1227-43
59. Kron IL, Roeser ME. Management of Ebstein's anomaly. Annals of cardiothoracic surgery. 2017 May:6(3):266-269. doi: 10.21037/acs.2017.05.03.
60. Rao PS. Single Ventricle-A Comprehensive Review. Children (Basel, Switzerland). 2021 May 24:8(6):. doi: 10.3390/children8060441.
61. Mitchell SC, Korones SB, Berendes HW. Congenital heart disease in 56,109 births. Incidence and natural history. Circulation. 1971 Mar:43(3):323-32
62. Bajwa F, Jafri SM, Ananthasubramaniam K. A Review of Selected Adult Congenital Heart Diseases Encountered in Daily Practice. Current cardiology reviews. 2021:17(3):260-278. doi: 10.2174/1573403X16666200810104615.
63. Landzberg MJ, Murphy DJ Jr, Davidson WR Jr, Jarcho JA, Krumholz HM, Mayer JE Jr, Mee RB, Sahn DJ, Van Hare GF, Webb GD, Williams RG. Task force 4: organization of delivery systems for adults with congenital heart disease. Journal of the American College of Cardiology. 2001 Apr:37(5):1187-93
64. Wilson W, Taubert KA, Gewitz M, Lockhart PB, Baddour LM, Levison M, Bolger A, Cabell CH, Takahashi M, Baltimore RS, Newburger JW, Strom BL, Tani LY, Gerber M, Bonow RO, Pallasch T, Shulman ST, Rowley AH, Burns JC, Ferrieri P, Gardner T, Goff D, Durack DT, American Heart Association Rheumatic Fever, Endocarditis, and Kawasaki Disease Committee, American Heart Association Council on Cardiovascular Disease in the Young, American Heart Association Council on Clinical Cardiology, American Heart Association Council on Cardiovascular Surgery and Anesthesia, Quality of Care and Outcomes Research Interdisciplinary Working Group. Prevention of infective endocarditis: guidelines from the American Heart Association: a guideline from the American Heart Association Rheumatic Fever, Endocarditis, and Kawasaki Disease Committee, Council on Cardiovascular Disease in the Young, and the Council on Clinical Cardiology, Council on Cardiovascular Surgery and Anesthesia, and the Quality of Care and Outcomes Research Interdisciplinary Working Group. Circulation. 2007 Oct 9:116(15):1736-54
65. Williams T, Lluri G, Boyd EK, Kratzert WB. Perioperative Echocardiography in the Adult With Congenital Heart Disease. Journal of cardiothoracic and vascular anesthesia. 2020 May:34(5):1292-1308. doi: 10.1053/j.jvca.2019.11.036.
66. Brickner ME, Hillis LD, Lange RA. Congenital heart disease in adults. First of

two parts. The New England journal of medicine. 2000 Jan 27:342(4):256-63

67. Andropoulos DB, Stayer SA, Skjonsby BS, East DL, McKenzie ED, Fraser CD. Anesthetic and perioperative outcome of teenagers and adults with congenital heart disease. Journal of cardiothoracic and vascular anesthesia. 2002 Dec:16(6):731-6
68. Williams GD, Philip BM, Chu LF, Boltz MG, Kamra K, Terwey H, Hammer GB, Perry SB, Feinstein JA, Ramamoorthy C. Ketamine does not increase pulmonary vascular resistance in children with pulmonary hypertension undergoing sevoflurane anesthesia and spontaneous ventilation. Anesthesia and analgesia. 2007 Dec:105(6):1578-84