ISSN – 2349-0683

Asian Pacific Journal of Nursing

Journal homepage: www.mcmed.us/journal/apjn

THE MANAGEMENT OF CHD: COMPREHENSIVE CONTROLLING OF THE DISEASE PATHOPHYSIOLOGY TO THE LONG-TERM MANAGEMENT

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ABSTRACT

CHD is a heterogeneous combination of structural cardiac defects of embryonic origin related to cardiovascular maldevelopment during embryogenesis and is currently one of the largest causes of infant morbidity and mortality in the world. Improvements in prenatal diagnosis, diagnostic ultrasound, pharmacological therapy, interventional cardiology, and cardiac surgery have greatly enhanced survival and CHD has become a chronic disease to be managed lifelong. The pathophysiology is caused by the abnormal shunting, obstruction, or mixing of blood leading to the imbalance of hemodynamics, hypoxemia, and possible heart failure. Pulse oximetry, echocardiography, and high-quality imaging are necessary to produce an early diagnosis that can be used to intervene in time. It is managed through medical stabilization, catheterization procedures and corrective palliative surgery with extensive preoperative and postoperative care offered. Nutritional care, developmental observation as well as psychosocial intervention are essential in ensuring maximum improvement. The follow-up is required throughout the lifespan to identify complications, functional recovery, and the quality of life. Efforts to realize the best possible long-term prognosis and general health of a CHD patient should be centered on a multidisciplinary patient-centered care approach.

Key words: Congenital heart disease; Cardiac surgery; Echocardiography; Multidisciplinary management; Long-term follow-up.

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Article Info
Received 05/02/2026; Revised 28/02/2026
Accepted 03/03/2026

INTRODUCTION

CHD is a heterogeneous condition affecting the structure and functional defects of the heart and is found in the fetus. It is known to be one of the most prevalent forms of congenital anomalies in the world and one of the most prominent causes of infant morbidity and mortality. Congenital heart defects are a serious health concern to the populace as the prevalence of CHD is estimated to be about 810 per 1,000 live births according to the global estimates of epidemiological data. The development of prenatal diagnosis, neonatal care, cardiac surgery and long-term management has led to drastic increases in survival rates during the last decades. Consequently, more children with CHD are surviving into adolescence and adulthood and this has led to an ever-growing population that needs specialized cardiac care throughout life[1–5].

CHD is a broad term that includes the simple defects such as small defects of the septum which may close on its own, through to the complex ones that involve the malformation of many cardiac structures and may only be remedied through surgical operations in stages. Typical ones are atrial septal defects, ventricular septal defects, patent ductus arteriosus, tetralogy of Fallot, transposition of the great arteries and hypoplastic left heart syndrome. Such abnormalities may involve the chambers of the heart, the valves, septa, great vessels or a combination of the two or more resulting in distorted hemodynamics and lack of adequate oxygen supply to the body.

CHD etiology is multi-factorial and can be explained by a complex of genetic, environmental, and maternal factors. Congenital cardiac anomalies are usually

associated with genetic syndromes like the Down syndrome and Turner syndrome. Also, maternal diseases such as diabetes, pregnancy-related infections, exposure to teratogenic agents, and nutritional deficits can also be involved in abnormal cardiac development in embryogenesis. Although genetic research has improved, in most instances the exact cause is not known.

The pathophysiology of congenital heart disease is important to the management. Abnormalities of the structure can cause blood to be shunted between the chambers of the heart, that is, there may be obstruction to flow of blood or mixing of oxygenated and deoxygenated blood. The changes may culminate in cyanosis, heart failure, pulmonary hypertension, growth failure and developmental delays. Depending on the nature and the extent of the defect, the clinical presentation is diverse. Others come shortly after birth with respiratory distress or cyanosis and others can stay asymptomatic until later in childhood or even later in adulthood.

Congenital heart disease treatment has become one of the most specialized and multidisciplinary areas of care that include pediatric cardiologists, cardiothoracic surgeons, nurses, nutritionists, and developmental specialists among other healthcare providers. Prenatal ultrasound, and postnatal screening including pulse oximetry and echocardiography, have been found to improve the situation by allowing the prevention of consequences before they occur. The treatment plans can involve the medical treatment to control symptoms, interventions based on a catheter, and corrective or palliative surgeries. In most situations, the combination of these methods is needed throughout the life of a patient.

The preoperative and postoperative care is a major issue in order to maximize the outcome especially in infants subjected to complicated cardiac surgery. Surgical practice, cardiopulmonary bypass, and intensive care have advanced considerably to lower the rate of mortality during an operation. Nevertheless, there are still long-term problems, such as arrhythmias, residual defects, neurodevelopmental problems, and psychosocial consequences of a chronic condition.

Along with the ever-increasing survival rates, the outcome has moved beyond merely securing survival in the short-term viewpoint but on the optimal quality of life throughout the lifespan. Other important aspects of comprehensive management are long-term follow-up care, adult-to-congenital heart disease service transition, nutritional support, developmental follow-up, and psychosocial counseling. The combined methodology is expected to not only respond to the anatomical defect but also to the general physical, emotional, and developmental health of patients who live with congenital heart disease[6–8].

An overview of the congenital heart diseases

CHDs are abnormalities of the heart and great vessels which do not develop postnatally. These occur as a result of inadequate development of the cardiovascular system in embryonic development, which occurs in the first eight weeks of pregnancy when the heart is developing. CHDs are the most prevalent birth defect across the world and have diverse severity, clinical outcomes and prognosis outcomes. Certain defects are minor and can be corrected naturally, whereas others are complicated and life threatening thus emergency medical or surgery treatment can be necessary[9].

The CHDs are categorized into two major groups, including acyanotic and cyanotic defects. Acyanotic defects are aberration of blood flow where blood moves to the right instead of the left or the blockage to blood flows without substantial decreases in blood oxygen in the body. They can be atrial septal defects (ASD), ventricular septal defects (VSD) and patent ductus arteriosus (PDA). Under such circumstances, the left side of the heart supplies the oxygenated blood to the right side which is deoxygenated, reaching the lungs and increasing blood flow to the lungs and causing pulmonary hypertension and heart failure in the untreated case scenario.

Cyanotic defects, conversely, are characterized by the left-to-right shunting or the mixing of the oxygenated and the deoxygenated blood, which leads to a lack of oxygen saturation and cyanosis. Examples of notable ones are tetralogy of Fallot (TOF), transposition of the great arteries (TGA) and hypoplastic left heart syndrome (HLHS). To give an example, Tetralogy of Fallot is a combination of four structural defects that block the blood flow in the lungs and permit deoxygenated blood into the blood stream[10,11]. Likewise, there is Transposition of the Great Arteries whereby the major arteries are reversed and fail to follow a sequential circuit but instead parallel and this can be fatal unless timely action is taken.

Congenital heart diseases are etiologically multifactorial. Hereditary factors are also important, and most are associated with chromosomal aberrations and single-gene mutations. Genetic syndromes have often been linked to CHDs including Down syndrome and DiGeorge syndrome. The risk of abnormal cardiac development is also augmented by environmental factors which comprise maternal infections, inadequately controlled diabetes, alcoholism, some drugs, and prenatal exposure to teratogens.

CHDs can vary in their severity based on the presence of simple lesions which may accumulate over the years without symptoms, or the presence of critical defects which manifest during the neonatal years with respiratory distress, cyanosis, or even a circulatory collapse. The early identification is critical to the best results. The progress in screening the fetus through fetal echocardiography has enabled detection of many serious structural defects of the heart before birth. In the postnatal period, pulse oximetry and other screening tools have been used to identify potential critical congenital heart diseases early so that they can be referred and treated on time.

The CHDs are epidemiologically common at a prevalence of 1% of live births globally. Medical and surgical therapies have been enhanced, which has resulted in a huge improvement in survival, making CHD less of a life-threatening disease in infancy and a chronic illness that needs a lifetime repair. This leads to an increase in the number of adolescents and adults with congenital heart defects that are repaired or not.

Overall, CHD is a heterogeneous group of structural heart defects, which have different pathophysiology, clinical manifestations, and prognoses. The management of them involves early diagnosis, multidisciplinary care, and regular check-ups to not only deal with the short-term complications that arise but also deal with health issues in the long term.

Table 1: Overview of Congenital Heart Diseases: Classification, Pathophysiology, and Clinical Implications

Category	Definition / Description	Examples	Pathophysiology / Mechanism	Clinical Implications
Acyanotic CHDs	Defects with left-to-right shunting or obstruction without systemic hypoxemia	ASD, VSD, PDA	Oxygenated blood from left heart enters right heart → increased pulmonary blood flow → pulmonary hypertension and potential heart failure	May remain asymptomatic initially; risk of pulmonary overcirculation, growth delay, and heart failure if untreated
Cyanotic CHDs	Defects with right-to-left shunting or mixing of oxygenated and deoxygenated blood → systemic hypoxemia	Tetralogy of Fallot (TOF), Transposition of Great Arteries (TGA), Hypoplastic Left Heart Syndrome (HLHS)	Blood bypasses lungs or mixes, lowering oxygen saturation	Cyanosis, fatigue, poor growth, “tet spells” in TOF; requires timely intervention to prevent mortality
Etiology	Causes of CHD are multifactorial	Genetic syndromes: Down syndrome, DiGeorge syndrome; environmental: maternal infections, diabetes, teratogens, alcohol	Chromosomal abnormalities, single-gene mutations, teratogenic effects during early embryogenesis	Identifying risk factors can guide prenatal screening and early management
Severity	Varies by defect type and hemodynamic impact	Simple lesions vs. critical defects	Simple defects may remain asymptomatic; critical defects cause neonatal respiratory distress or circulatory collapse	Early detection via fetal echocardiography or postnatal pulse oximetry is crucial for timely intervention
Epidemiology	Global prevalence of CHD	–	–	~1% of live births; increasing number of adolescents and adults living with repaired or unrepaired CHDs
Management	Multidisciplinary approach	Medical therapy, catheter interventions, surgery	Addresses hemodynamic abnormalities, oxygenation, and complications	Lifelong follow-up needed to monitor growth, development, residual defects, and long-term complications

Table 2: Clinical Manifestations of Congenital Heart Disease Across Age Groups and Defect Types

Category / Defect Type	Age of Presentation	Key Clinical Features	Additional Notes / Signs
Cyanotic CHDs (e.g., Tetralogy of Fallot, Transposition of Great Arteries)	Neonates / Infants	Cyanosis (greyish/blue lips, tongue, nails), tachypnea, feeding difficulties, poor weight gain, excessive sweating	Clubbing of fingers/toes in chronic hypoxemia, squatting/tet spells in older children
Acyanotic CHDs with left-to-right shunt (e.g., VSD,	Infants / Young Children	Symptoms of heart failure: tachycardia, tachypnea,	Recurrent respiratory infections, exercise intolerance in older

PDA)		hepatomegaly, poor feeding, irritability, failure to thrive	children
Heart murmurs	Any age	Turbulent heart sounds detected during physical exam	Not all murmurs indicate CHD, but loud/persistent murmurs require evaluation
Obstructive lesions (e.g., Coarctation of the Aorta)	Infancy / Childhood	Weak or absent peripheral pulses, differences in blood pressure between upper and lower extremities	May cause hypertension, heart failure, or delayed growth if untreated
Mild / small defects (e.g., small ASDs)	Childhood / Adolescence / Adulthood	Often asymptomatic early; may develop arrhythmias, exercise intolerance, or pulmonary hypertension later	Adult presentation can include stroke, infective endocarditis, or progressive heart failure
Compensatory behaviors	Older children with cyanotic CHD	Squatting to relieve dyspnea, fatigue on exertion	Helps temporarily increase systemic vascular resistance and improve oxygenation

Figure 1: Comprehensive Diagnostic Evaluation Pathway for Congenital Heart Disease (CHD)

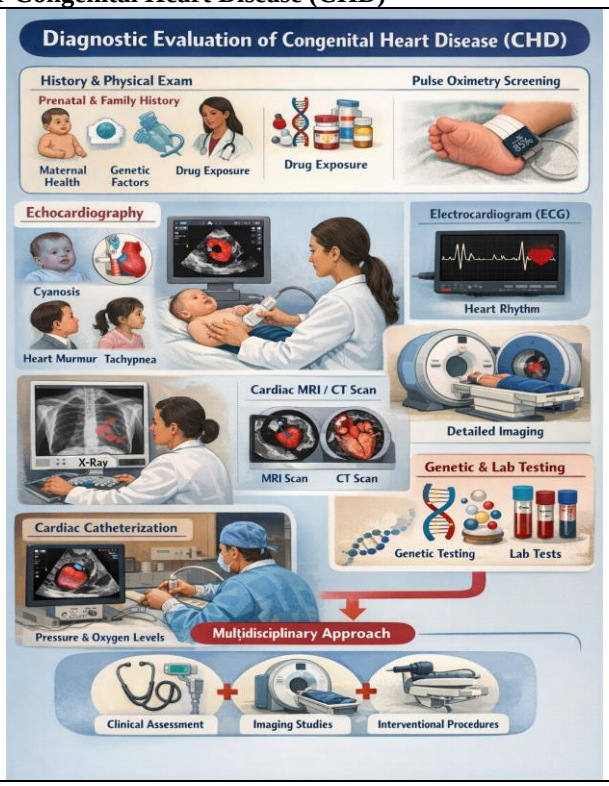
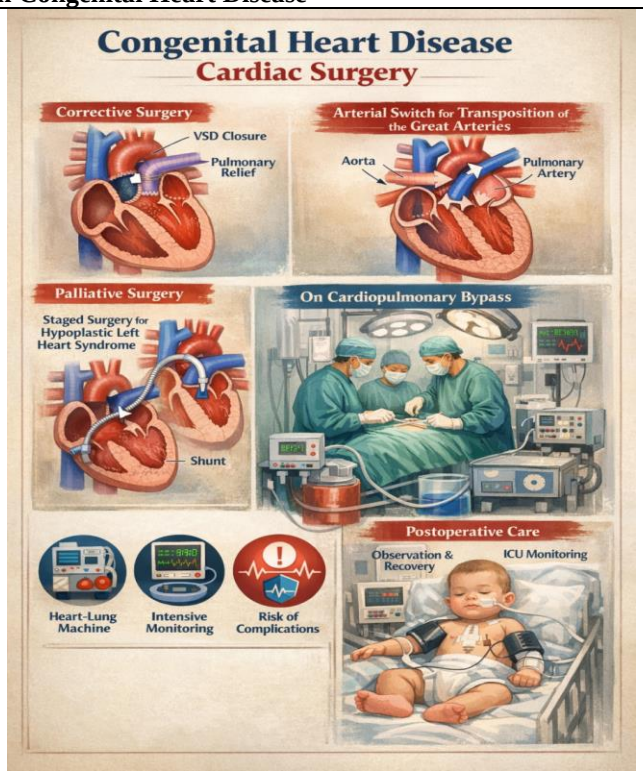


Figure 2: Surgical Management and Postoperative Care in Congenital Heart Disease



Pathophysiology

The pathophysiology of congestional heart disease (CHD) has its basis in maladaptation of cardiac development during embryonic development, which occurs normally between the third and eighth weeks of gestation. The primitive heart tube loops, separates and divides into four chambers with valves and great vessels in this critical period[12–14]. Any disturbance in these processes, e.g., genetic mutation, chromosome abnormalities, environmental exposures, and multifactorial factors, may lead to structural defects that cause a change in normal cardiac anatomy and hemodynamics.

The pathophysiology of CHD is really a combination of disruptions in the blood flow, pressure relationship, and oxygen supply. The nature, size, and position of the defect, to a great extent, determine these disturbances. There are three significant processes that usually result in hemodynamic outcome shunting of blood, blocking of blood flow, and mixing of bloodsheds (oxygenated and deoxygenated).

Some of the most frequent types of CHD are shunt lesions. In left-to-right shunts (like atrial septal defects (ASD) or ventricular septal defects (VSD), the oxygenated blood on the left-side of the heart is pumped

into the right-side of the heart because of increased systemic pressure. This causes there to be more blood flow and volume overloading of the right heart and pulmonary circulation[15]. Within a long period of time, continuous overcirculation can cause pulmonary vascular remodeling and pulmonary hypertension. Uncontrolled long-term pulmonary hypertension may cause a reversal of blood flow (turning right to left) (Eisenmenger syndrome), leading to hypoxemia and cyanosis throughout the body.

Another important pathophysiological category is the presence of the obstructive lesions. These malformations hinder the forward blood flow contributing to pressure load on the defective chamber. In Coarctation of the Aorta, as an example, aortic constriction raises the afterload of the left ventricle and that results in left ventricular hypertrophy. Likewise, the pulmonary stenosis leads to right ventricular hypertrophy because of the resistance to the blood flow into the pulmonary artery. With time, the ventricular functioning may be impaired by pressure overload and result in heart failure.

Cyanotic heart defects include right to left shunting or parallel circulation meaning that deoxygenated blood can pass by-passing the lungs into the systemic circulation. In some disorders like Tetralogy of Fallot, there is an obstruction of the pulmonary outflow and blockage of the septal defects, which allow the deoxygenated blood to be mixed with the oxygenated blood, lowering the saturation of arterial oxygen. The chronic hypoxemia activates the compensatory mechanisms that includes polycythemia (production of red blood cells) to be in a position to increase the oxygen-carrying capacity. Yet, these adaptations may elevate the blood viscosity and expose the individual to the risk of thrombotic complications.

In more complicated defects, the circulation is reversed so that the aorta and the pulmonary artery trade places making two parallel circulations. Systemic oxygenation is impossible without a shunt (e.g. ASD, VSD, or patent ductus arteriosus) to permit mixing of blood. This causes severe cyanosis immediately after birth and needs emergency medical procedures or surgeries.

The pathophysiology of CHD can also be caused by neurohormonal activation, especially in the heart failure situation. The sympathetic nervous system and renin angiotensin aldosterone system (RAAS) activation causes fluid retention, vasoconstriction and increased cardiac workload which worsens the ventricular dysfunction.

In general, the pathophysiology of congenital heart disease is characterized by complex interactions between the structure abnormality and the compensatory physiological process. Hemodynamic implications of the defect will vary based on the defect type and determine clinical manifestations, disease course, and treatment. These mechanisms make it necessary to understand how to diagnose, manage and prevent the development of long-term complications.[16–18]

Clinical Manifestations

The clinical presentations of CHD are diverse in relation to the nature, size and severity of the defect, and the age of the patient. There are those congenital heart defects which have been found to manifest themselves as life threatening signs soon after birth, others which may be asymptomatic over a number of years and yet others may be identified incidentally through routine medical check ups. The features and symptoms are mostly manifestations of hemodynamic imbalances, such as abnormal blood circulation, pressure overload, volume overload and poor oxygenation.

Severe CHDs in infants and neonates tend to manifest early. A cyanosis (greying lips, tongue and nail beds) is a characteristic of cyanotic congenital cardiac defects and is caused by low O2 saturation in the arteries. Veins including Tetralogy of Fallot and Transposition of the Great Arteries can easily show cyanosis soon after birth. The infants with such defects can also experience rapid breathing (tachypnea), feeding difficulties, excessive perspiration during feeding and poor weight gain. The result is the development of feeding intolerance and fatigue since the infant uses a lot of energy in his attempts to breathe and keep a sufficient circulation[19,20].

The other prevalent clinical presentation is heart failure especially in acyanotic defects with severe left-to-right shunting with ventricular septal defects or patent ductus arteriosus. Infants display symptoms of heart failure, which include tachycardia, tachypnea, hepatomegaly, inadequate feeding, irritable, and failure to thrive. Heart failure in older children can be diagnosed by intolerance to exercise, dyspnea, and peripheral edema. Recurring respiratory infections can also be a result of chronic volume overload caused by pulmonary congestion.

One of the most common signs of congenital heart disease that are most often detected are murmurs. They are induced by arterial turbulent flow over deformed cardiac valves and are frequently detected in regular physical examination. Although not every murmur signifies heart disease in the structure, positive or loud murmurs should be investigated. Other abnormalities can include the abnormal heart sounds, weak or absent peripheral pulses, and lack of blood pressure in the upper and lower extremities as is observed in Coarctation of the Aorta.

With chronic hypoxemia in cyanotic defects, the fingers and toes can be clubbed which is defined as a bulbous swelling of the distal digits. Squatting poses can also be accepted by older children with cyanotic CHD during dyspnea, and this position assists in raising systemic vascular resistance and temporarily enhances oxygenation. These are episodes that are widely known as tet spells and they are usually linked with Tetralogy of Fallot.

Not all congenital heart defects have symptoms until the teenage or adulthood. As an example, small atrial defects of the septum may have few signs and symptoms during early stages of life, but present with arrhythmias or exercise intolerance or evidence of pulmonary hypertension. In adults, CHD that has not been previously diagnosed can present as stroke, infective endocarditis or progressive heart failure.

The intensity and time of symptoms can be strongly related to the amount of hemodynamic decompensation and the compensatory capacity of the body. Appropriate early detection of warning symptoms, including cyanosis, feeding problems, poor development, frequent respiratory infections, etc., is very important in early diagnosis and treatment. With the increase in survival and the number of children with CHD surviving to adulthood, clinicians should be attentive to both early and late signs of congenital heart disease so as to achieve the best outcomes throughout the lifespan[18,21,22].

Diagnostic Evaluation

Diagnostic evaluation of congenital heart disease (CHD) is an organized set of clinical assessment, noninvasive imaging, and in some cases, invasive procedures that were successfully used to identify a type of defect and its severity. The diagnosis should be done early to avoid complications and start the timely management[23,24]. Assessment usually starts with an elaborate prenatal, perinatal and family history, including maternal diseases, drug exposure, and inherited diseases related to heart defects. A physical examination can provide the following essential results: cyanosis, heart murmurs, abnormal heart sounds, tachypnea, hepatomegaly, peripheral pulse and blood pressure discrepancies. Pulse oximetry screening in newborns has emerged as an important intervention to identify serious defects of the cardiac system of the body through low levels of oxygen saturation that might remain unidentified.

Echocardiography is the key to the diagnosis of CHD and gives a detailed description of the cardiac anatomy, chamber sizes, valve functioning, blood circulation of blood, and pressure gradient. It is also noninvasive, easily accessible, and highly sensitive and thus it is the leading imaging modality when screening structural abnormality. Fetal echocardiography is able to detect most significant defects prior to birth, which can result in plenty of planning of delivery and early intervention. In some complicated cases, sophisticated imaging, e.g. cardiac magnetic resonance imaging (MRI) scan or computed tomography (CT) scan, provides more anatomical information especially when great vessels and extracardiac structures are required to be studied.

ECG can be helpful in measuring cardiac rhythm disorders, enlargement of chambers as well as conduction disorders. The additional supportive information that can be obtained with the help of chest radiography is the size of the heart and pulmonary vascular markings that can indicate the increased or reduced blood flow in the lungs. Cardiac catheterization is conducted in specific patients in order to determine accurate measurements of intracardiac pressures, oxygen saturation and vascular resistance. Interventional procedures can also be performed through it like device closure of septal defects or balloon valvuloplasty.

In case when CHD is suspected to be a part of a larger syndrome, including Down syndrome or DiGeorge syndrome, genetic testing and consultation can be mentioned. Lab tests can also be helpful to assess the complications, such as polycythemia in the patient with cyanosis or heart failure biomarkers. On the whole, a multidisciplinary approach, which involves the integration of clinical knowledge and sophisticated technology, is needed in the diagnostic evaluation to determine the precise definition of the defect, make treatment decisions, and track long-term outcomes[4,25,26].

Health Medical and Surgery

Congenital heart disease (CHD) is medically and surgically treated in accordance with the type of defect, its severity, and the complications that arise, as well as the age and the overall health conditions of a patient. The main objectives of the management are to maximize cardiac performance, proper oxygenation and development, avoid complications, and enhance long-term survival and quality of life[4,18,27,28]. Initial treatment is often through medical management especially in patients with the heart failure symptoms or patients awaiting surgical correction. The pharmacological treatment can comprise of diuretics that should be used to alleviate the fluid overload, angiotensin-converting enzyme (ACE) inhibitors that should be used to reduce afterload and increase cardiac output, and beta-blockers that should be used to regulate the heart rate and decrease the myocardial workload. The use of prostaglandin E1 infusion can help to keep the ductus arteriosus pat within neonates with duct-dependent lesions and retain systemic or pulmonary blood flow until the appropriate definitive intervention can be undertaken. Components of supportive care are also oxygen therapy, nutrition, and close attention to fluid balance.

Although the defect minor defects can close on their own or leave a hemodynamically insignificant defect, several congenital heart defects necessitate correction with the help of interventional or surgical measures. Pediatric cardiology has seen increased innovations that result in catheter-based interventions that may be used to treat some defects without the need of an open-heart operation. As an illustration, atrial or ventricular septal defects repair through device closure or balloon valvuloplasty with stenotic valves can be undertaken frequently through cardiac catheterization, minimizing the amount of time and surgical risk. However, intricate structural defects usually require surgery. Depending on the condition, corrective or palliative cardiac surgery can either be done

in one operation or in stages. When the Tetralogy of Fallot occurs, ventricular septal defect is repaired and the right ventricle outflow obstruction is corrected. In the case of severe cases such as Transposition of the Great Arteries, surgical repair is very early in life to ensure that normal circulatory channels are instituted.

In intensive care units, postoperative management is a crucial factor contributing to hemodynamic stabilization, infection prevention, as well as complications treatment, including arrhythmias or residual defects. The follow-up may be lifelong because some of them need further intervention, replacement of the valves, or they may have to deal with the late complications. In conclusion, medical treatment, interventional cardiology, as well as surgical skills have greatly enhanced the survival and the long-term outcome of the people living with the congenital heart disease.

Pharmacological Therapy

The pharmacological treatment is one of the most significant aspects of the treatment of congenital heart disease (CHD), especially in regulating symptoms, hemodynamics, and complications prevention of CHD before or after surgery. The medication is based on the nature of the defect, the presence of heart failure, pulmonary hypertension, arrhythmias or cyanosis, age and clinical status of the patient. Medical treatment in children and infants with left-to-right shunt lesions, including ventricular septal defects, is usually focused on congestive heart failure, which is due to the overload of the heart. The more common ones are diuretic, furosemide, which is given to reduce retention of fluids, ease congestion of the lungs, and alleviate the symptoms of tachypnea and edema. Diuretics assist in reducing preload and enhancing respiratory conditions by stimulating sodium and water loss.

Angiotensin-converting enzyme (ACE) inhibitors, such like captopril and enalapril, are also common in order to decrease afterload and increase cardiac output. These drugs work by suppressing the reninangiotensinaldosterone system, and hence they decrease the myocardial workload and systemic vascular resistance. Beta-blockers can be used in selected patients to boost the ventricular performance and regulate the heart rate especially when it comes to cardiomyopathy or arrhythmias. In certain patients, digoxin can also be employed to enhance the contractility of the heart and control the symptoms of heart failure, but its application has become more selective with the emergence of modern therapy of heart failure.

The life-saving intervention in neonates who present with duct-dependent congenital heart defects is the prostaglandin E1 infusion. It also keeps the ductus arteriosus patent, so that the systemic or pulmonary flow of blood is adequate until the definitive operation can be carried out. E.g., where there is Transposition of the Great Arteries, ductal patency will be preserved so that the deoxygenated and oxygenated blood will be mixed, to stabilize the infant while a temporary fix is found.

Treatment of the CHD related pulmonary hypertension can involve sildenafil or endothelin receptor blocker to reduce pulmonary vascular resistance to enhance oxygen dissolution. Anticoagulants or antiplatelet drugs can be prescribed to some patients to lessen the chances of thromboembolism particularly in patients with cyanotic heart disease or prosthetic valves. Moreover, the antiarrhythmic drugs are prescribed in case of rhythm disorders that are frequent in unrepaired and surgically repaired CHD[29–31].

Structural heart defects are rarely treated with pharmacological therapy but is an invaluable supplement to surgical or interventional intervention. Strict dosage, observation of side effects and frequent follow up are essential especially in the case of infants and children whose drug absorption and physiological reactions are not similar to those in the adult. When applied in form of specific medical care, the symptoms are reduced, the complications are reduced, and the patients are stabilized, the final result will be more favorable outcomes and the quality of life of people with congenital heart disease.

Cardiac Surgery

In the treatment of congenital heart disease (CHD), cardiac surgery forms a pillar especially when the structural anomaly is moderate to severe and cannot be properly managed with medical therapy. Surgical intervention is meant to correct or palliate anatomical abnormalities, normalize hemodynamics, optimize oxygenation, alleviate symptoms, and increase survival in the long run[4,32,33]. The time and nature of the surgery will depend on the defect itself, its extent, the age of the patient and the general clinical state. In most instances, infants may require surgical intervention at an early age to avoid complication that may be irreversible including, pulmonary hypertension, heart failure, or poor growth and development.

Cardiac surgical interventions on CHD may be of corrective or palliative classification. Corrective surgeries are done to fix the defect in structure and create a situation of almost normal circulation. As an illustration, the entire treatment of Tetralogy of Fallot would entail the sealing of the ventricular septal defect and the decongestion of the right ventricular outflow to enhance the operations of the lung, and the distribution of oxygen to all parts of the body. In a similar case, during Transposition of the Great Arteries, an arterial switch surgery is undertaken at a young age to re-lay the great arteries and re-establish normal circulation systems. Such corrective measures have contributed a lot in terms of survival rates and long-term outcomes.

Palliative surgeries instead do not correct the defect totally, but are aimed at enhancing oxygenation and stabilizing the patient until some definitive repair can be done or to deal with sophisticated single-ventricle

physiology. A case in point is the staged surgery in hypoplastic left heart syndrome, where several surgeries are done to divert blood flow and aid in the systemic blood circulation. There are other instances where shunt surgeries are developed to enhance blood flow into the lungs in cyanotic children.

The majority of cardiac surgeries done on congenital cases are done with cardiopulmonary bypass, which temporarily replaces the functions of the heart and lungs in the process. The development of surgical methods, anesthesia, myocardial protection, and the intensive care of the postoperative period has significantly decreased the mortality and postoperative complications associated with operations. However, cardiac surgery is not without risks as such as bleeding, infection, arrhythmias, neurological complications, and residual defects that need reoperation.

Special cardiac intensive care units should provide postoperative care and facilities to observe the stability of hemodynamics, pain control, preventing infection, and the early detection of complications. The follow-up is vital in the long term since certain patients might need further interventions, valve replacement, or treatment of the late sequelae, including arrhythmias or ventricular dysfunction. All in all, the cardiac surgery has changed the prognosis of congenital heart disease where many of the victims have been able to survive to adulthood and live a much better life [4, 34–36].

CONCLUSION

The management of congenital heart disease requires a comprehensive, multidisciplinary, and lifelong patient-centered approach that extends from early diagnosis to long-term follow-up and transition into adulthood. Understanding the diverse pathophysiological mechanisms and ensuring timely diagnosis through advanced diagnostic tools enable appropriate therapeutic planning and intervention. While medical management provides supportive care, surgical and catheter-based interventions remain essential for correcting structural defects and improving survival outcomes. However, continued monitoring is crucial due to potential residual complications, functional limitations, and psychosocial challenges. Equally important are nutritional support, developmental care, and family education, which significantly influence recovery and quality of life. The transition to adult congenital heart disease care further ensures continuity and addresses evolving health needs. Ultimately, a holistic approach focusing on long-term well-being, functional capacity, and psychosocial support can significantly enhance the lifespan and overall quality of life of individuals living with congenital heart disease.

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