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A HANDS-ON MANUAL FOR IDENTIFYING AND TREATING HEART FAILURE IN CHILDREN

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Abstract

Context: *The incidence of heart failure among children constitutes a major cause of sickness and mortality during their initial years of existence. Whereas well-established guidelines are present for managing heart failure in adults, a corresponding consensus for the pediatric group is lacking. Within a healthcare setting, it's of paramount importance to establish a correct diagnosis and identify the root cause for the most effective intervention. Primary interventions include diuretics and angiotensin-converting enzyme inhibitors, however, the utilization of beta-blockers and cardiac therapy devices is less common in pediatric cases as compared to adults. For severe conditions, heart transplant remains the go-to therapeutic choice. Additionally, left ventricular assist devices can be employed as a temporary solution awaiting transplantation (due to scarcity of organ donors), recovery (in instances of myocarditis), or a definitive solution (for patients with systemic disease).*

Key Phrases: *Myocarditis, Congenital heart abnormalities, Pediatric heart transplantation, Pediatric heart failure.*

1. OVERVIEW

Heart failure in children, also known as Pediatric Heart Failure (PHF), is a significant contributor to illness and death during the early years of life. The causes and development of the disease differ between adults and children: in adults, it is primarily related to ischemia (60-70% of cases), whereas in children, it is usually due to congenital heart diseases (CHDs) or cardiomyopathies.[1] As a result, the management of PHF necessitates specialized knowledge and abilities. While there are comprehensive protocols for treating heart failure in adults,[2] a similar agreement for PHF is absent. This article provides a summary of the causes, diagnosis, and treatment of PHF, with a particular emphasis on practical aspects necessary for management.

2. Definition

In the 1950s, heart failure was defined as a clinical condition resulting from reduced cardiac output. However, our understanding of the disease's pathophysiology has grown over the years, leading to the discovery of neurohormonal and molecular pathways that influence the functioning of hearts in a state of failure. Today, heart failure is viewed as a clinical syndrome marked by distinct symptoms and signs, which are linked to particular circulatory, neurohormonal, and molecular irregularities.[3]

3. Causes

Heart failure in children mainly arises due to inborn heart abnormalities (CHDs) and heart muscle disorders (cardiomyopathies). At birth, heart failure can stem from fetal heart muscle disorders or non-cardiac issues such as widespread infection, low blood sugar, and insufficient calcium in the blood. During the initial week after birth, the chief trigger is CHDs that depend on the ductus arteriosus for overall circulation (like severe narrowing of the aortic valve/aorta and underdeveloped left heart syndrome), wherein the closure of the ductus arteriosus results in a substantial drop in blood supply to various organs. Within the first month after birth, prevalent causes of CHF are CHDs with a left-to-right blood flow anomaly (like holes in the ventricular septum, open ductus arteriosus, and aorto-pulmonary passages), where the blood flow to the lungs gradually rises as the resistance in the pulmonary vessels diminishes. Finally, heart failure in adolescents is rarely due to CHDs but more often linked with heart muscle disorders or inflammation of the heart muscle.[3]

4. The Fundamental Processes Behind Child Heart Failure

A root "triggering incident," irrespective of its source, instigates a reduction in the pumping ability of the heart muscle cells, setting the stage for heart failure. This primary harm results in a decline in heart function, which is then counteracted by two principal "balancing strategies". The first strategy initiates the sympathetic nervous system, leading to an elevated release and diminished absorption of norepinephrine, which in turn prompts peripheral blood vessel narrowing to safeguard the average blood pressure and blood flow to organs by enhancing resistance in the body's circulatory system. However, increased concentrations of these stress hormones can inflict additional harm on the heart muscle cells, disturb cell-to-cell communication, and eventually culminate in heart muscle cell death. The second notable "balancing" strategy is the instigation of the renin-angiotensin-aldosterone system, which involves an increase in circulating renin, angiotensin II, and aldosterone. Renin splits angiotensinogen into angiotensin I, which is then transformed into angiotensin II by the angiotensin-converting enzyme (ACE). Angiotensin II is a potent blood vessel narrowing agent that helps sustain blood flow to the organs. Aldosterone encourages the body to hold onto salt and water, leading to an increase in the volume of blood entering the heart, which subsequently boosts the heart's pumping output, in line with the Frank-Starling principle. However, increased amounts of both aldosterone and angiotensin II can lead to heart scarring and planned cell death. Although these strategies may initially help maintain the balance in the circulation, over time, they turn harmful and fuel the advancement of heart failure.[4]

5. Clinical Manifestations

The clinical symptoms of Pediatric Heart Failure (PHF) are closely tied to the age of the patient.[5]

Infants and Young Children: The typical symptoms include feeding difficulties, ranging from extended feeding times to outright intolerance. Other signs may include cyanosis, rapid breathing, sinus tachycardia, and excessive sweating.

Older Children and Adolescents: The primary symptoms in this age group are fatigue, shortness of breath, rapid breathing, and intolerance to physical activity. Abdominal pain, reduced urine output, and swelling in the legs due to fluid accumulation may also be observed. The severity of heart failure in children should be categorized according to the modified Ross classification4, which identifies four functional classes with increasing severity of clinical features from I to IV (Table 2).

Table 1: Pediatric heart failure classification according to the revised Ross system

Class I	•Asymptomatic
Class II	•Mild tachypnea or diaphoresis with feeding in infants •Dyspnea on exertion in older children
Class III	•Marked tachypnea or diaphoresis with feeding in infants. Prolonged feeding times with growth failure •Marked dyspnea on exertion in older children
Class IV	•Symptoms such as tachypnea, retractions, grunting, or diaphoresis at rest

6. Diagnostic Procedure
The initial step in diagnosing Pediatric Heart Failure (PHF) relies on non-invasive clinical examinations.

6.1 Electrocardiogram
In acute heart failure, sinus tachycardia is a common finding. In chronic heart failure, an abnormal electrocardiogram can indicate a higher likelihood of decompensated heart failure.[4]

6.2 Chest X-Ray
A chest X-ray is recommended for all children suspected of having heart failure to evaluate the size of the heart and to look for other signs of heart failure such as fluid in the lungs, septal lines (also known as Kerley B lines), and fluid around the lungs.[4]

6.3 Echocardiography
Echocardiography is the most useful, accessible, and cost-effective test for patients with PHF. It provides immediate information on the shape and structure of the heart, the size of the chambers, the thickness of the walls, the function of the ventricles during systole and diastole, and the pressure in the pulmonary arteries. This information is essential for making an accurate diagnosis and determining the most suitable treatment.[5]

6.4 Diagnostic Laboratory Procedures
The purpose of lab tests in the handling of heart failure is depicted in Table 3.

Table 2: Diagnostic lab procedures for heart failure.

Test	Rationale
Complete blood count	• Leukocytosis can be caused by stress or indicate an infection. • This examination is beneficial for identifying anemia, which can trigger or exacerbate heart failure.
Electrolytes	• Low sodium levels indicate expansion of the fluid volume outside cells in the presence of normal total body sodium. • Extended use of diuretics can lead to low potassium and chloride levels. • High potassium levels can be the result of inadequate kidney blood flow and significant reduction in the filtration rate of the kidneys, or the release of potassium from within cells due to deficient tissue blood flow.
Tests for kidney function	• Severe heart failure is indicated by heightened blood urea nitrogen (BUN) and the ratio of BUN to creatinine.
Liver function tests	• Liver enlargement due to congestion is often associated with compromised liver function, as shown by raised levels of AST, ALT, LDH, and other liver enzymes. • High levels of bilirubin (both direct and indirect) are associated with acute congestion of the liver's blood supply and are common in patients with severe right heart failure. • There is an increase in alkaline phosphatase and a prolongation of clotting time. • Low albumin levels are due to impaired liver synthesis in children with long-standing heart failure and poor nutrition.
CPK-MB, troponin I and T	• This test is beneficial if there's a possibility of an ischemic event or inflammation of the heart muscle.
Natriuretic peptides (NT-proBNP/BNP)	• The levels of these peptides have a strong correlation with the NYHA/Ross heart failure classification and ventricular filling pressures.
Thyroid function tests	• Severe hyperthyroidism or hypothyroidism can both lead to heart failure.
Lactate	• Elevated lactate levels are observed in patients with severe heart failure as a result of diminished tissue blood flow and/or reduced metabolism due to secondary liver dysfunction, and it may be a useful biomarker for monitoring the efficacy of treatment.
Arterial blood gas	• In patients with mild-to-moderate heart failure, this test typically shows slightly low oxygen levels. • Severe heart failure often results in very low oxygen levels, or even a lack of oxygen. • Low carbon dioxide levels develop in the early stages of fluid buildup in the lungs due to an imbalance between ventilation and perfusion, progressing to high carbon dioxide levels and respiratory acidosis, both of which can be attributed to reduced lung capacity and inadequate ventilation.

ALT:alanine aminotransferase. AST:aspartate aminotransferase. BNP:B-type natriuretic peptide. BUN:blood urea nitrogen. CPK-MB:creatine phosphokinase. LDH:lactic dehydrogenase. NT-proBNP:N-terminal proBNP. PTT:prothrombin time. V/Q:ventilation/perfusion.

6.5 Cardiac MRI Scanning
Cardiac MRI scans are suggested for the assessment of intricate congenital heart conditions or for analyzing tissue properties, which are key to diagnosing, risk evaluation, and patient management with specific kinds of heart muscle disorders.[6]

6.6 Heart Catheterization

Even with advancements in non-invasive diagnostic techniques, heart catheterization is currently advocated for[2]:

- Accurate measurement of pressure differences in patients with complex heart valve conditions
- Examination of circulatory parameters (such as resistance in the pulmonary and systemic vascular systems, heart function, and heart efficiency index) in patients with Fontan circulation or during pre-transplant screening

6.7 Endomyocardial Tissue Sampling

Endomyocardial tissue sampling is an invasive method with considerable risks and should only be undertaken to verify the clinical diagnosis of myocarditis and to identify the most appropriate treatment strategy[2] (for instance, in scenarios of giant cell myocarditis).[7]

7. Therapy Approach

The objective of the treatment for Child Heart Failure (CHF) is to:[2]

- Resolve the underlying causes of CHF
- Control the symptoms and decelerate the disease's advancement.

8. Addressing the Root Causes of Heart Failure

When possible, the triggers of heart failure should be remedied through various methods:

- Corrective therapy should be provided for inborn heart abnormalities[7]
- Systemic conditions (like widespread infections) or imbalances in body salts (such as low calcium levels) should be meticulously probed and remedied.

9. Addressing Symptoms and Decelerating Disease Evolution

9.1 Broad Strategies

In the case of infants, nutritional backing should secure a caloric consumption of around 150 kcal/kg/day. This can be accomplished through nutritional enhancements and small, recurrent meals that are more easily accepted.[8]
For older children and teenagers, current recommendations propose a goal of 25-30 kcal/kg/day for the majority of patients.[8]

Carbohydrate consumption should not surpass 6 g/kg/day, and fat consumption should not go over 2.5 g/kg/day. Essential amino acids are vital for critically ill patients, with research indicating a need for 1.2-1.5 g/kg/day of protein.[8]

Nutritional fortification is necessary in heart failure resulting from metabolic and mitochondrial conditions (like carnitine and ubiquinone deficiencies).[8]

For patients with non-cyanotic congenital heart disease or cardiomyopathies, oxygen therapy should commence when oxygen saturation (SaO2) drops below 90%.[8]

Conversely, for patients with cyanotic congenital heart disease, oxygen has minimal effect on raising SaO2 and is not advised.[8]

However, in some scenarios with chronic left-to-right shunting, irreversible lung vascular disease can develop, causing right-to-left shunting (Eisenmenger syndrome). In the early stages, the resulting pulmonary hypertension may respond to oxygen, making it a suggested treatment while the child awaits heart transplantation or palliative surgery. Salt consumption reduction is recommended for all patients with swelling and fluid retention. Fluid limitation is advised for patients with swelling that does not respond to diuretic treatment or for those with low sodium levels.[8]

9.2 Pharmacological Treatment

Pharmacological treatment for heart failure (Table 4) focuses on three main goals[2]:

- **Reducing lung wedge pressure**
- **Enhancing heart function and boosting organ perfusion**
- **Decelerating the disease's progression.**

Table 3: Medications utilized to manage pediatric heart failure.

Medication	Administration Route	Dosages
Captopril	Oral	0.3–2 mg/kg q8h
Carvedilol	Oral	0.05 mg/kg/d q12h
Digoxin	Oral	5–10 µg/kg/d
Dobutamine	Continuous Infusion	2.5–10 µg/kg/min
Enalapril	Oral	0.05–0.25 mg/kg q12h
Epinephrine	Continuous Infusion	0.01–0.1 µg/kg/min
Epinephrine	Intermittent bolus	0.01 µg/kg
Furosemide	Oral	1–2 mg/kg q6–12h
Furosemide	Intermittent bolus	0.5–2 mg/kg q6–12h

Furosemide	Continuous infusion	0.1–0.4 mg/kg/h
Hydralazine	Intermittent bolus	0.1–0.2 mg/kg every 4–6 h
Hydralazine	Oral	0.3–1 mg/kg/d in q8–12h
Levosimendan	Continuous Infusion	0.05–0.2 µg/kg/min
Losartan	Oral	0.5–1.5 mg/kg/d
Metoprolol	Oral	0.25 mg/kg/d q12h
Milrinone	Continuous Infusion	0.5–1 µg/kg/min
Nitroglycerin	Continuous infusion	0.5–10 µg/kg/min
Nitroprusside	Continuous infusion	0.5–4 µg/kg/min
Spironolactone	Oral	0.5–1.5 mg/kg q12h

9.3 Diuretics

Diuretics have a significant role in managing heart failure (HF) in pediatric cases. The advantages of this therapy are the alleviation of systemic, lung, and venous congestion. Spironolactone may provide added benefits by impeding the formation of myocardial fibrosis triggered by aldosterone and the release of catecholamines. Possible drawbacks of diuretic therapy involve electrolyte imbalances (low sodium, abnormal potassium levels, and deficiency in chloride) and metabolic alkalosis. Careful monitoring of electrolyte equilibrium is critical, particularly in intensive diuretic treatment, as a weakening heart muscle is more susceptible to arrhythmias induced by electrolyte disturbances.[2]

9.4 Inhibitors of Angiotensin-Converting Enzyme (ACE)

ACE inhibitors have the ability to halt, mitigate, or even reverse adverse cardiac remodeling. Moreover, they decrease afterload by antagonizing the renin-angiotensin-aldosterone system. The most recent guidelines for pediatric heart failure (HF) management by the International Society for Heart and Lung Transplantation advocate ACE inhibitors for all HF patients with compromised left ventricular systolic function.[8] The treatment should start with minimal doses, gradually escalating to the desired level, while closely monitoring blood pressure, renal function, and blood potassium levels.

9.5 β-adrenergic Blocking Agents

β blockers have now gained acceptance as a treatment in pediatric cases. They neutralize the detrimental effects of continuous activation of the heart's sympathetic system and can reverse left ventricular remodeling and boost systolic function. Emerging evidence proposes that the inclusion of β blockers in standard therapy could benefit patients with compromised left ventricular systolic function.[9] A recent analysis in the Cochrane Database of Systematic Reviews on β blockers for children with congestive HF, which incorporated seven studies involving 420 children, concluded that existing data implies β-blocker treatment could be advantageous for children with HF.[10] Therapy should commence with a low dosage in stable patients, gradually escalating to the targeted dosage.

9.6 Inotropic Agents

Digoxin is the most commonly prescribed oral inotropic drug in pediatric heart failure and is recommended for symptomatic individuals with left and/or right ventricular systolic dysfunction. Intravenous inotropes should be confined to patients experiencing a significant drop in cardiac output, leading to serious compromise in organ perfusion (hypotensive acute/decompensated HF). Though elevated inotropic activity can improve cardiac output and blood pressure, it can also lead to increased oxygen consumption and demand by the heart. The debilitated heart muscle has limited contractile reserve, and excessive inotropic support could induce hemodynamic collapse.[2]

9.7 Sympathomimetic Amines

Dopamine and dobutamine have shown efficacy as inotropic agents and vasopressors in newborns, infants, and children with circulatory failure. These medications amplify cardiac output and reduce both systemic and pulmonary vascular resistance. However, they can trigger tachycardia/tachyarrhythmia, leading to an imbalance between the oxygen supply and demand in the heart muscle. Hence, their use should be confined to patients with low cardiac output despite other treatments.[2]

9.8 Phosphodiesterase Type III Inhibitors

This drug class includes amrinone, enoximone, milrinone, and olprinone, with milrinone being the most potent, shortest acting, and best-controlled medication, often used in pediatric intensive care. Phosphodiesterase type III inhibitors possess vasodilatory and inotropic properties and improve diastolic ventricular relaxation. Despite the risk of inducing arrhythmias, milrinone is the preferred treatment for patients with moderate to severe ventricular dysfunction who display symptoms of inadequate blood flow.[2]

9.9 Calcium Sensitizer

Levosimendan has strong inotropic and vasodilatory properties, potentially superior to dobutamine, with a reduced risk of causing myocardial ischemia. Its lack of arrhythmia-inducing effects and its capacity to counteract the effects of β blockade make levosimendan a potentially preferred medication in postoperative low-cardiac output syndrome, rather than in acute HF in children.[11]

9.10 Vasodilators

Vasodilators, administered either intravenously (nitroglycerin and nitroprusside) or orally (hydralazine and nifedipine), are indicated solely in instances of hypertensive acute HF resistant to treatment (β blockers and ACE inhibitors), and severe valve regurgitations in patients who cannot tolerate ACE inhibitors.[2]

9.11 Innovative Treatment Approaches

Several new drugs are being studied as potential solutions for pediatric HF. A high resting heart rate is recognized as a risk factor for adult HF mortality. Ivabradine, which inhibits the If current in the sinoatrial node, is used in the treatment of chronic heart failure patients. Its use has been linked with a reduction in HF hospitalizations and deaths.[1] A recent large, randomized, controlled trial compared the combined use of a neprilysin inhibitor and valsartan to enalapril. Neprilysin, a neutral endopeptidase, breaks down vasoactive neurohormones like natriuretic peptides, bradykinin, and adrenomedullin. By inhibiting neprilysin, vasoconstriction, sodium retention, and remodeling can be decreased. The study was halted early due to significantly better survival rates, lower hospitalization risk, and improvement in symptoms in patients treated with the neprilysin inhibitor-valsartan combination. Further research with these drugs in children with HF is merited.[2]

10. Therapeutic Devices

Despite the significant improvements in lifespan and quality of life due to medication treatments in pediatric HF patients, a significant proportion of patients still experience poor outcomes due to disease progression or sudden cardiac death. These patients could potentially benefit from the use of device therapy. The implantable cardioverter defibrillator (ICD) and cardiac resynchronization therapy (CRT) devices are commonly used in heart failure cases. The ICD plays a pivotal role in preventing sudden cardiac death due to ventricular arrhythmias in HF patients. Based on observational studies, the accepted indications for ICD implantation in pediatric HF include[1]:

- Prophylaxis against repeated sudden cardiac deaths in patients who have survived a cardiac arrest or those with a history of ventricular tachycardia causing hemodynamic instability
- Unexplained syncope in postoperative patients of congenital heart diseases
- Patients with severe left ventricular systolic dysfunction (left ventricular ejection fraction < 35%).

Around 30% of adults with HF display a left bundle branch block (LBBB) with mechanical dyssynchrony. However, only 9% of pediatric HF patients exhibit LBBB and a QRS duration > 120 milliseconds, probably reflecting the different causes of HF in this population.[12]

Left ventricular dyssynchrony refers to a situation where the left ventricular function in failing hearts is influenced not only by weakened contractility of the heart muscle, abnormal loading conditions, or both, but also by a disruption in the synchrony of the heart muscle walls. Delayed activation of certain segments leads to a slower rise in systolic pressure, delayed left ventricular ejection, slower relaxation, and delayed left ventricular filling.[13] It is believed that CRT, through biventricular pacing, improves the contraction pattern of the left ventricle.[14] Despite the lack of randomized clinical trials, retrospective studies have demonstrated the utility of CRT in pediatric patients with[2]:

- Cardiomyopathy, complete LBBB, and a significant reduction in left ventricular systolic function (left ventricular ejection fraction < 35%)
- Third-degree heart block necessitating pacemaker implantation in DDD mode in patients with mild/moderate systolic dysfunction (left ventricular ejection fraction < 55%)
- Congenital heart diseases with a two-ventricle physiology involving a systemic left ventricle with substantial systolic function reduction.

The efficacy of CRT in patients with isolated right ventricle dysfunction is still under debate. Some studies suggest CRT may improve right ventricular ejection fraction and New York Heart Association/Ross class while reducing QRS duration. However, this response was significantly less robust than that seen in patients with left ventricle dysfunction.[12]
Mechanical circulatory support systems can be used in pediatric HF patients unresponsive to medication to alleviate the load on the failing ventricle and maintain end-organ perfusion. Patients with cardiogenic shock/acute HF not responding to medication should initially receive short-term support from non-durable life support systems such as extracorporeal life support and extracorporeal membrane oxygenation.[15]

For patients with chronic intractable HF despite medication, the consideration of a permanently implantable left ventricular assist device should be made as a bridge to transplantation, recovery, and in rare cases, as a last therapy.

11. Cardiac Transplantation

Even though no controlled studies have been conducted, there's a general agreement that heart transplantation substantially improves life expectancy, physical capacity, and overall quality of life. Table 5 provides the qualifying factors and limitations for pediatric heart transplants. The success rate of these transplants has seen an incremental rise in recent years. The International Society of Heart and Lung Transplantation's most recent statistics show the average survival period as 19.7 years for newborns, 16.8 years for children aged 1-5, 14.5 years for those aged 6-10, and 12.4 years for adolescents aged 11-17 at the time of the transplant.[16]

Table 5: Pediatric Heart Transplantation Eligibility and Limitations

Candidates for Consideration	• Advanced heart failure resulting in severe exercise and activity limitations. If these patients were measured, their peak oxygen uptake would only be 50% of the expected level for their age and sex. • End-stage heart failure in patients with cardiomyopathies or previously treated congenital heart diseases, associated with systemic ventricular dysfunction. • Advanced heart failure in patients with restrictive cardiomyopathy related to reactive pulmonary hypertension. • Advanced heart failure associated with reactive pulmonary hypertension, presenting a potential risk of developing permanent, irreversible increases in pulmonary vascular resistance, which could complicate future heart transplantation. • Advanced heart failure accompanied by fatal arrhythmias that cannot be managed with drug or device therapy.
Contraindications	• Infection that is active or recurring. • Recent or recurring malignancies. • Diseases that are genetic or metabolic in nature and have a poor long-term prognosis. • Systemic diseases that are significant. • Pharmacologically irreversible pulmonary hypertension (pulmonary vascular resistance over 6 μm2). • Renal or liver dysfunction, not explained by the underlying heart failure and believed to be permanent.

Following are the main complications post-transplant:

Rejection: After the transplant, rejection significantly affects the survival of the transplanted heart. According to the Paediatric Heart Transplant Study, 64% of patients did not experience rejection in the first year (meaning 36% did face rejection), and the rate of being rejection-free over five years was 52%.[16]

Infection: Owing to immunosuppression, the recipient becomes susceptible to opportunistic infections, contributing to approximately 12% of deaths within the first year post-transplant.[16]

Cardiac Allograft Vasculopathy: This is a leading cause of mortality post-pediatric transplantation and affects 34% of patients within ten years of their transplant.[17]

Malignancy: Tumors, particularly lymphoproliferative diseases, are relatively rare after a transplant. However, the incidence of cancer within the ISHTL registry, 5 and 10 years post-transplant, is reported to be 5% and 9.5% respectively.[16]

12. Adopted Strategy for Managing Pediatric Heart Failure

When faced with emergency conditions, immediate heart failure is addressed as a unique syndrome, regardless of the underpinning factors, at the onset. The lone exception involves the onset of acute heart failure originating from ductus-dependent circulation congenital heart illness or non-cardiac reasons, where the immediate commencement of Prostaglandin E1 or a specialized treatment is essential. For the remainder of the patients, the treatment path depends on the manifestation of fluid overload and insufficient perfusion. Given that fluid overload is a frequent occurrence, the early application of intravenous loop diuretics proves to be beneficial for virtually all patients dealing with acute or deteriorating heart failure. However, it's critical to be aware of the adverse impacts of diuretic usage, including the loss of sodium and potassium, potential hearing damage, and potential renal issues, thus warranting a cautious approach to diuretic administration. In situations of inadequate perfusion, the application of low to moderate quantities of inotropes is advised. Once stabilization is achieved, a comprehensive investigation of the root cause should be launched.

In cases involving cardiomyopathies and patients contending with moderate to severe left ventricular systolic dysfunction, the crux of the medical treatment strategy is built around ACE inhibitors and β blockers.

For individuals with congenital heart disorders, preparations for rectifying or palliative measures are required, and medical treatment is reserved for those exhibiting left ventricular systolic dysfunction. Finally, for patients in terminal stages, the consideration of a ventricular assist device implantation or cardiac transplantation should be made, with decisions about the timing and indications made on a case-by-case basis.

13. Final Words

Pediatric heart failure is a complex syndrome with a myriad of causes and manifestations. Unlike in adults, heart failure in children often originates from structural heart abnormalities and conditions that can be reversed, thereby lending themselves to decisive or potent short-term interventions. Even though the overarching management principles mirror those applied to adults, there is a glaring scarcity of randomized clinical trials and globally recognized guidelines specifically catered to pediatric heart failure. Striking a judicious balance between adopting adult heart failure guidelines and the generation of child-centric treatment data embodies a logical approach to enhance management in this intricate field.

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