
**“EARLY GROWTH TRENDS OF CHILDREN
UNDER 5 YEARS OF AGE WITH CONGENITAL
HEART DISEASE WHO HAVE UNDERGONE
CORRECTIVE SURGERY - A LONGITUDINAL
STUDY”**

By

REG NUMBER: BM0122007

Dissertation

Submitted to

*KLE Academy of Higher Education and Research,
Belagavi, Karnataka*

*In partial fulfilment
of the requirements for the degree of*

**M.D. (Doctor of Medicine) in
PEDIATRICS**

**DEPARTMENT OF PEDIATRICS,
JAWAHARLAL NEHRU MEDICAL COLLEGE,
BELAGAVI, KARNATAKA**

SEPTEMBER / OCTOBER – 2025

**KLE ACADEMY OF HIGHER EDUCATION AND
RESEARCH BELAGAVI, KARNATAKA**

**ENDORSEMENT BY THE HOD, PRINCIPAL/
HEAD OF THE INSTITUTION**

This is to certify that the dissertation entitled “**EARLY GROWTH TRENDS OF CHILDREN UNDER 5 YEARS OF AGE WITH CONGENITAL HEART DISEASE WHO HAVE UNDERGONE CORRECTIVE SURGERY- A LONGITUDINAL STUDY**” is a bonafide research work done by **REG NUMBER: BM0122007.**


Dr. DNYANESH D K M.D., L.L.B

Professor & Head

Department of Pediatrics

Dr. Dnyanesh D.K
PROFESSOR & HEAD
DEPARTMENT OF PEDIATRICS
J. N. Medical College, Belagavi
KAHER'S J. N. Medical College,
Nehru Nagar, Belagavi -590010

Date: 28/03/25

Place: Belagavi


Dr. (Mrs) N. S. MAHANTASHETTI M.D(Peds)

Professor and Principal,

KAHER's J. N. Medical College,

Nehru Nagar, Belagavi -590010

PRINCIPAL
Jawaharlal Nehru Medical College
BELAGAVI

Date: 28/03/25

Place: Belagavi

UNDERTAKING

I, **REG NO. BM0122007**, hereby declare that the information and the data mentioned in my dissertation entitled **“EARLY GROWTH TRENDS OF CHILDREN UNDER 5 YEARS OF AGE WITH CONGENITAL HEART DISEASE WHO HAVE UNDERGONE CORRECTIVE SURGERY - A LONGITUDINAL STUDY”** belongs to me and is original. I am aware of the definition of plagiarism as detailed below:

- An act or instance of using or closely imitating the language and thoughts of another author without authorization and the representation of that author's work as one's own, as by not crediting the original author.
- A piece of writing or other work reflecting such unauthorized use or imitation.
- The deliberate or reckless representation of another's words, thoughts or ideas as one's own without attribution in connection with submission of academic work, whether graded or otherwise.

I hereby declare that the dissertation prepared by me is original one and does not involve plagiarism anywhere. In case at a later stage, it is found that I have indulged in plagiarism, then I am solely responsible for the same and the institution is at liberty to take any disciplinary action against me including cancellation of dissertation or any other penalties imposed by the University.

Date:28-03-2025

Place: Belagavi



REG. NO: BM0122007

PLAGIARISM CERTIFICATE



JAWAHARLAL NEHRU MEDICAL COLLEGE

(A constituent unit of KLE Academy of Higher Education & Research Deemed-to-be-University)

(Recognized by National Medical Commission, New Delhi)



Accredited 'A+' Grade by NAAC (3rd Cycle)

Placed in Category 'A' by MoE (GoI)

Nehru Nagar, Belagavi- 590 010, Karnataka, INDIA

0831 - 2471350

0831 - 2470759

www.jnmc.edu

principal@jnmc.edu

Ref No: MDC/PG/


Date: 21-03-2025

"ACCEPTANCE LETTER"

The softcopy of thesis entitled: "EARLY GROWTH TRENDS OF CHILDREN UNDER 5 YEARS OF AGE WITH CONGENITAL HEART DISEASE WHO HAVE UNDERGONE CORRECTIVE SURGERY- A LONGITUDINAL STUDY" has been submitted for anti-plagiarism check through Turnitin software. The scan has been carried out and the scanned output reveals a match percentage of 10% which is within the acceptable limits of 10% as per the guidelines given by UGC.


Guide.




Dr. (Mrs.) N.S. Mahantashetti,
Chairperson-Antiplagiarism Committee &
Principal,
J. N. Medical College, Belagavi.

To,
Reg. No. BM0122007
Postgraduate Student,
2022-23 Batch,
Department of Paediatrics
J. N. Medical College, Belagavi.

ETHICAL CLEARANCE LETTER



K.L.E. ACADEMY OF HIGHER EDUCATION AND RESEARCH
(Deemed – to- be- University)

Accredited 'A+' Grade by NAAC In (3rd Cycle) Placed in Category 'A' by MHRD (GoI)

JNMC INSTITUTIONAL ETHICS COMMITTEE
JAWAHARLAL NEHRU MEDICAL COLLEGE,
NEHRU NAGAR, BELAGAVI-590010 (KARNATAKA-INDIA)

Website: <http://www.jnmc.edu>
E-Mail : dome@jnmc.edu

Phone: (+ 91-(0)831 Office : 2472550
Principal: 2471701
Fax No. +91 (0)831 – 2470759

Ref No.MDC/JNMCIEC/ 95

Date: 26/03/2023

To,
BM0122007
PG Student in Paediatrics
J. N. Medical College,
BELAGAVI.

Sub: Institutional Ethical Clearance for the study.

With reference to the above, we wish to inform you that your proposed research project titled "EARLY GROWTH TRENDS OF CHILDREN UNDER 5 YEARS OF AGE WITH CONGENITAL HEART DISEASE WHO HAVE UNDERGONE CORRECTIVE SURGERY- A LONGITUDINAL STUDY", is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee.

(Dr. Smita Sonoli)
Member Secretary
JNMC Institutional Ethics Committee
J.N.Medical College, Belagavi.

(Dr. Harsha Hegde)
Chairman,
JNMC Institutional Ethics Committee
J.N.Medical College, Belagav

LIST OF ABBREVIATIONS

1. **AP** – Aortopulmonary
2. **AS** – Aortic Stenosis
3. **ASD** – Atrial Septal Defect
4. **AV** – Atrioventricular
5. **AVSD** – Atrioventricular Septal Defect
6. **BMI** – Body Mass Index
7. **CHD** – Congenital Heart Disease
8. **CPB** – Cardiopulmonary Bypass
9. **CPD** – Cephalopelvic Disproportion
10. **CT** – Computed Tomography
11. **d-TGA** – d-Transposition of the Great Arteries
12. **ECHO** – Echocardiography
13. **ECG** – Electrocardiogram
14. **FTT** – Failure to Thrive
15. **GDM** – Gestational Diabetes Mellitus
16. **HAZ** – Height-for-Age Z-score
17. **HCZ** – Head Circumference-for-Age Z-score
18. **HLHS** – Hypoplastic Left Heart Syndrome
19. **IGF-1** – Insulin-Like Growth Factor 1
20. **LGA** – Large for Gestational Age
21. **LSCS** – Lower Segment Cesarean Section
22. **MAM** – Moderate Acute Malnutrition
23. **MRI** – Magnetic Resonance Imaging
24. **MSL** – Meconium-Stained Liquor

25. **NICU** – Neonatal Intensive Care Unit
26. **NPL** – Non-Progressive Labor
27. **NVD** – Normal Vaginal Delivery
28. **PAH** – Pulmonary Arterial Hypertension
29. **PDA** – Patent Ductus Arteriosus
30. **PIH** – Pregnancy-Induced Hypertension
31. **PROM** – Premature Rupture of Membranes
32. **Qp:Qs** – Pulmonary-to-Systemic Blood Flow Ratio
33. **SAM** – Severe Acute Malnutrition
34. **SPSS** – Statistical Package for the Social Sciences
35. **TAPVR** – Total Anomalous Pulmonary Venous Return
36. **TGA** – Transposition of the Great Arteries
37. **TOF** – Tetralogy of Fallot
38. **VSD** – Ventricular Septal Defect
39. **WAZ** – Weight-for-Age Z-score
40. **WHZ/WLZ** – Weight-for-Height/Length Z-score
41. **WHO** – World Health Organization

ABSTRACT

Background

Congenital heart diseases are the most common congenital anomaly found in newborn and nearly 9 to 11 in 1000 newborns are born with cardiac anomalies in India . Atrial septal defect, ventricular septal defect, patent ductus arteriosus are the most common congenital heart diseases. Malnutrition is a common cause of morbidity in such children. The extent to which the preoperative nutritional status affects the post op growth is unknown. There may be various factors such as ethnicity, socioeconomic status, nutritional practices, other cultural and environmental factors that may affect the nutritional status of the child with congenital heart disease. There are various studies showing that corrective surgery shows a significant improvement in growth parameters within 3 months of post surgery. However, studies have also shown that postoperative catch-up growth was less pronounced or even absent in 15%–50% of patients depending on the study. The factors affecting growth in a child with congenital heart disease may vary worldwide and hence there is a need to assess growth in these children in local conditions. The understanding and prevention of the effect of CHD on patients' growth and development status in developing countries still needs improvement. This study Is aimed at analysing the early growth trends of children pre and post operatively and factors affecting growth in this region of the country. This study also emphasises the importance of nutritional and growth assessment preoperatively so as to predict the early catchup growth post surgical correction.

Objectives

- To evaluate the early growth trend of children with congenital heart disease who have undergone corrective surgery under 5 years of age.
- To identify factors affecting growth in children with Congenital heart disease and analyse the short term effects of corrective surgery.

Methodology

A hospital based longitudinal observational study was conducted at the Department of Paediatrics, DR Prabhakar Kore Charitable Hospital, Belagavi, from August 2023 to July 2024. It included children under five years of age with congenital heart disease undergoing corrective surgery, with follow-up assessments at three months post-surgery. A sample size of 102 was determined, considering a 10% loss to follow-up. Preoperative and postoperative anthropometric measurements, including height, weight, head circumference, and mid-upper arm circumference, were recorded and compared using WHO growth standards. Additional factors such as hospital stay duration, sepsis, and pulmonary arterial hypertension severity were documented. Socioeconomic status was assessed using the modified Kuppuswamy scale. Data were analyzed using SPSS Version 16, with descriptive statistics, t-tests, and chi-square tests to determine malnutrition patterns and potential growth predictors. A p-value <0.05 was considered statistically significant.

Result

The study included 105 children undergoing corrective surgery for congenital heart disease, with a mean age of 2.31 ± 1.09 years. The majority (59%) were female, and 59% belonged to the lower middle class. Most children (96.2%) were born at term, and 70.5% had a hospital stay exceeding seven days. Atrial septal defect (29.5%) and

ventricular septal defect (28.6%) were the most common heart defects, with 88.6% having acyanotic CHD. Severe pulmonary arterial hypertension was present in 26.7% of cases. Preoperatively, 83.8% were underweight, 50.5% were stunted, 81% were wasted, and 32.4% had microcephaly. At three months post-surgery, significant improvements were observed: underweight prevalence dropped to 42.9%, stunting to 28.6%, and microcephaly to 17.1%. Severe wasting was eliminated, and normal nutritional status improved from 11.4% at baseline to 48.6%. The mean weight z-score improved from -3.16 to -2.11, height z-score from -1.97 to -1.71, and head circumference z-score from -1.55 to -1.16 ($p = 0.001$). Regression analysis showed that age and birth weight significantly influenced preoperative weight and height z-scores, while NICU admission, sepsis, and hospital stay had no major impact. Overall, the study demonstrated significant growth recovery in children post-surgery.

Conclusion

This study demonstrates that corrective surgery for congenital heart disease (CHD) in children under five significantly improves growth and nutritional status within three months. Pre-surgery, severe malnutrition and growth retardation were prevalent, largely influenced by age and birth weight. Post-surgery, weight, height, and head circumference z-scores improved, with severe acute malnutrition dropping from 41.9% to 2.9% and normal nutritional status rising from 11.4% to 48.6%. These findings highlight the critical role of early diagnosis, timely surgery, and post-operative care in enhancing growth and overall health in children with CHD.

CONTENTS

| SR. NO. | TOPIC | PAGE NO. |
|----------------|---|-----------------|
| 1. | INTRODUCTION | 1-4 |
| 2. | AIMS AND OBJECTIVES | 5 |
| 3. | REVIEW OF LITERATURE | 6-27 |
| 4. | MATERIALS AND METHODS | 28-31 |
| 5. | RESULTS | 32-61 |
| 6. | DISCUSSION | 62-72 |
| 7. | CONCLUSION | 73 |
| 8. | SUMMARY | 74-76 |
| 9. | BIBLIOGRAPHY | 77-84 |
| 10. | ANNEXURES | 85- |
| | ANNEXURE I – CONSENT FORM | 85-87 |
| | ANNEXURE II- DEFINITIONS | 88 |
| | ANNEXURE II – PROFORMA | 89-91 |
| | ANNEXURE IV – PHOTOGRAPHS | 92-93 |
| | ANNEXURE V – KEY TO MASTER CHART | 94-95 |
| | ANNEXURE VI –MASTER CHART | |

LIST OF TABLES

| TABLE NO. | DESCRIPTION | PAGE NO. |
|-----------------|---|------------|
| Table 1 | Relative frequency of the most common congenital cardiac lesions | 8-9 |
| Table 2 | Distribution of Antenatal History of Mothers of Children Undergoing Corrective Surgery | 34 |
| Table 3 | Indications for Cesarean Section in Children Undergoing Corrective Surgery | 37 |
| Table 4 | Type of Congenital Heart Disease (CHD) in Children Undergoing Corrective Surgery | 39 |
| Table 5 | Distribution of CHD based on Severity of Pulmonary Arterial Hypertension (PAH) | 40 |
| Table 6 | Incidence of Sepsis in Children Undergoing Corrective Surgery in immediate post-op period | 41 |
| Table 7 | Distribution of Children with Left to right shunt lesions Undergoing Corrective Surgery based on severity of shunt. | 43 |
| Table 8 | Percentage of Presence of Residual Lesion in Children with left to right shunt lesions after Corrective Surgery as determined by Post-op ECHO | 44 |
| Table 9 | Baseline Growth Parameters of Children Undergoing Corrective Surgery | 45 |
| Table 10 | Growth Parameters at 3-Month Follow-Up After Corrective Surgery | 46 |

| | | |
|-----------------|---|--------------|
| Table 11 | Comparison of Growth Parameters at Baseline and 3-Month Follow-Up | 47 |
| Table 12 | Improvement in Wasting Status at 3-Month Follow-Up After Corrective Surgery | 48 |
| Table 13 | Improvement in Underweight Status at 3-Month Follow-Up After Corrective Surgery | 49 |
| Table 14 | Improvement in stunting Status at 3-Month Follow-Up After Corrective Surgery | 50 |
| Table 15 | Improvement in Microcephaly Status at 3-Month Follow-Up After Corrective Surgery | 51 |
| Table 16 | Comparison of Type of Malnutrition from baseline to 3-Month Follow-Up After Corrective Surgery | 52 |
| Table 17 | Transition in Type of Malnutrition from Baseline to 3-Month Follow-Up After Corrective Surgery | 53-54 |
| Table 18 | Comparison of mean weight gain among children with different types of Acyanotic CHD with Left to right shunt. | 55 |
| Table 19 | Comparison of mean difference in height/length among children with different types of Acyanotic CHD with Left to right shunt. | 57 |
| Table 20 | Factors Influencing Pre-Operative Weight z-Score in Children with Congenital Heart Disease (CHD) | 58 |
| Table 21 | Factors Influencing Pre-Operative Height/Length z-Score in Children with Congenital Heart Disease (CHD) | 59 |
| Table 22 | Factors Influencing Pre-Operative Weight/Height z-Score in Children with Congenital Heart Disease (CHD) | 60 |

LIST OF FIGURES

| FIGURE NO. | DESCRIPTION | PAGE NO. |
|------------------|--|-------------|
| Figure 1 | A general algorithmic approach to the initial diagnosis of congenital heart disease, based on observation/pulse oximetry and chest x-ray | 15 |
| Figure 2 | Bar graph showing age wise Distribution of Children Undergoing Corrective Surgery | 32 |
| Figure 3 | Pie chart showing Gender Distribution of Children Undergoing Corrective Surgery | 33 |
| Figure 4 | Bar graph depicting distribution of study population based on socioeconomic status | 33 |
| Figure 5 | Donut pie chart showing distribution of birth weight of Children Undergoing Corrective Surgery | 35 |
| Figure 6 | Pie chart showing distribution of study population based on Gestational Age at Birth. | 35 |
| Figure 7 | Pie chart depicting distribution of study population based on Mode of Delivery of Children at birth | 36 |
| Figure 8 | Horizontal Bar chart showing Indications for Cesarean Section in Children Undergoing Corrective Surgery. | 38 |
| Figure 9 | Bar chart showing distribution of type of Congenital Heart Disease (CHD) in Children Undergoing Corrective Surgery | 39 |
| Figure 10 | Pie chart showing distribution of Congenital Heart Disease (CHD) Based on Cyanosis in Children Undergoing Corrective Surgery | 40 |

| | | |
|------------------|--|-----------|
| Figure 11 | Bar chart showing Distribution of CHD based on Severity of Pulmonary Arterial Hypertension (PAH) | 41 |
| Figure 12 | Pie chart showing Incidence of Sepsis in Children Undergoing Corrective Surgery in immediate post-op period. | 42 |
| Figure 13 | Pie chart depicting Duration of Hospitalization in Children Undergoing Corrective Surgery during the perioperative stay. | 42 |
| Figure 14 | Doughnut Pie chart showing Distribution of Children with Left to right shunt lesions Undergoing Corrective Surgery based on severity of shunt. | 43 |
| Figure 15 | Pie chart showing Distribution of study population based on Complexity of Lesion. | 44 |
| Figure 16 | Grouped Bar chart showing Comparison of Growth Parameters at Baseline and 3-Month Follow-Up | 48 |
| Figure 17 | Stacked Bar graph showing Overall Improvement in Nutritional Status at 3-Month Follow-Up After Corrective Surgery | 55 |
| Figure 18 | Line graph showing Mean weight gain of children with CHD with Left to right shunt. | 56 |
| Figure 19 | Line graph showing Mean increase in height in children with CHD with Left to right shunt | 57 |

LIST OF PHOTOGRAPHS

| FIGURE NO. | DESCRIPTION | PAGE NO. |
|-------------------|---|-----------------|
| Photo 1 | Stadiometer used for measuring height | 92 |
| Photo 2 | Infantometer used for measuring length | 92 |
| Photo 3 | Multipurpose digital weighing scale ranging from 50g to 120kgs with 5g division was used (meditive brand) | 93 |
| Photo 4 | IS Indosurgicals head circumference measuring tape calibrated upto 60cm and with 1mm graduation was used. | 93 |

INTRODUCTION

Congenital heart defects (CHDs) are one of the most frequently occurring birth anomalies, with approximately one-third requiring surgical treatment during infancy. Approximately 2,00,000 children are born with congenital heart disease (CHD) each year in India, with a birth prevalence of 8 per 1,000.¹ Out of these, nearly 50,000 children would require intervention during infancy. Prevalence of CHD does not differ much in developed and developing countries. In developed countries, most children with CHD are diagnosed early and undergo timely intervention. The Center for Disease Control and Prevention (CDC), USA reports 18-y survival rates of 95% and 69% in babies born with noncritical CHD and critical CHD, respectively.² In contrast, developing countries report far higher mortality rates.³

In neonates, infants, and children, the clinical symptoms of congenital heart disease (CHD) varies from one another. It is often easy to recognize the presence of CHD in older children. In infants and particularly in newborns, manifestations of heart disease can often be subtle. Now withstanding these difficulties, it is possible to identify the presence of heart disease in most infants and newborns after careful clinical evaluation. Clinical symptoms of CHD include forehead sweating during feeding, difficulty in breathing, chest indrawing, bluish discolouration of face and limbs, suck rest suck cycle, swelling of limbs, repeated chest infections etc. A thorough and systematic cardiovascular examination provides valuable clues to the presence of heart disease.⁴

Echocardiography is a non-invasive modality used to define both the morphological and functional findings in congenital heart disease including size of the lesion, direction of the shunt, morphology of the valves and chambers, pulmonary

artery pressures and pressures across the shunts. Paucity of pediatric cardiac surgical facilities and financial constraints often lead to delay in seeking health care and/or getting appropriate and timely interventions. This results in a large cohort of children with undiagnosed/untreated CHD. Since these children are prone to develop respiratory tract infections, congestive heart failure, and cyanotic spells, they need frequent hospital admissions. This further leads to high prevalence of pre-operative malnutrition in patients with CHD.⁴

Malnutrition being the most common cause of morbidity among the children with CHD has also been found to be associated with higher mortality after surgery even in developed nations⁵. The implications of preoperative malnutrition for future somatic growth are unknown and the role of different ethnic, socioeconomic and cultural factors (very common in India) on the nutritional status of patients with CHD have not been hitherto studied. It has been seen that most children with CHD have normal weight at birth in developed countries but start falling off their trajectory in the initial few months due to disproportionate needs. Studies have shown that an appropriate nutrition plan helps to reduce the prevalence of malnutrition in children waiting for surgery.⁶

It is important to note that the prevalence rates of malnutrition differ depending on the kind of malnutrition and the particular case of heart disease. According to a study conducted in North India, 72.5% of children diagnosed with cyanotic congenital heart disease (CHD) were malnourished, with 68.9% of those children being categorised as seriously malnourished.⁷ On the other hand, according to statistics collected from around the world, roughly 27.4% of children diagnosed with congenital heart disease are affected by preoperative malnutrition, with 24.4% exhibiting stunting and 24.8% wasting.⁸ Underweight, stunting, and wasting are the

three kinds of malnutrition that are most common among children in India who have congenital heart disease. These findings underscore the critical need for early nutritional assessment and intervention in children with CHD in India to enhance growth outcomes and overall health.⁴

Growth impairment is frequently observed in infants with congenital heart diseases (CHDs) monitored through the growth chart, which reflects height and weight in terms of absolute values and percentiles. Accurate plotting and following of the growth curve are essential parts of the initial and follow-up evaluations of a child with significant heart problems. Different patterns of growth impairment are seen in different types of CHD.⁹

1. Cyanotic patients often experience growth disturbances in both height and weight. Conditions such as TOF, TGA, tricuspid atresia, HLHS, and TAPVR can impair growth due to chronic hypoxia, increased metabolic demands, and inadequate oxygenation, leading to lower height and weight percentiles.

2. In acyanotic congenital heart defects (CHDs) with large left-to-right shunts, such as ventricular septal defect (VSD), atrial septal defect (ASD), and patent ductus arteriosus (PDA), weight gain is often more affected than height. This occurs due to increased pulmonary blood flow, leading to higher metabolic demands, respiratory distress, and inadequate calorie intake. The severity of growth impairment correlates with the size of the shunt, with larger defects causing more significant weight deficits.

3. Acyanotic patients with pressure overload lesions like aortic stenosis, pulmonary stenosis, and coarctation of the aorta typically grow normally, as they do not have intracardiac shunts affecting oxygenation or metabolism. Poor growth in a child with mild cardiac anomaly or failure of catch-up weight gain after repair of the defect may

be caused by failure to recognize certain syndromes, inadequate calorie intake, or the underlying genetic predisposition.⁹

It is well known that children with CHD are at risk of impaired somatic growth and neurodevelopment. Multiple cardiac and patient factors are considered to impact growth, including type and severity of the CHD, weight at birth, nutritional factors, and hemodynamic instabilities. For early childhood, several studies have shown some catch-up for weight and height after corrective surgery in children with CHD. Nonetheless, growth parameters may remain below the expected values for preschool aged children. Similarly, lower head circumference at birth is frequently reported in infants with CHD. In contrast, information on growth parameters at school age is scarce and studies mainly include univentricular defects. Further, there is little information on longitudinal growth. In contrast to the impaired growth in younger children with CHD, studies in teenagers and young adults with CHD report a risk of overweight and obesity comparable to the general population. Yet, little is known about BMI in prepubertal children.¹⁰

There is very little data available on the growth trends as it depends on various factors and need for advanced medical support during and post surgery. So the number of studies done on this aspect of congenital heart disease are less. Hence, this study was aimed at analysing the early growth trends of children pre and post operatively and factors affecting growth in this region of the country. This study also emphasises the importance of nutritional and growth assessment preoperatively so as to predict the early catchup growth post surgical correction.

AIMS AND OBJECTIVES

Primary objective:

- To evaluate the early growth trend of children with congenital heart disease who have undergone corrective surgery under 5 years of age.

Secondary objective:

- To identify factors affecting growth in children with Congenital heart disease and analyse the short term effects of corrective surgery.

REVIEW OF LITERATURE

Congenital heart disease (CHD) occurs in approximately 0.6–1.3% of live births. This overall incidence does not include mitral valve prolapse, PDA of preterm infants, and bicuspid aortic valve (present in 1–2% of adults). Congenital cardiac defects have a wide spectrum of severity in infants: approximately 2-3 in 1,000 newborn infants will be symptomatic with heart disease in the first year of life. The diagnosis is established by 1 week of age in 40–50% of patients with CHD and by 1 month of age in 50–60%. Approximately 13% of infants with CHD have associated extracardiac malformations. With advances in both corrective and palliative surgery, 90% of children with CHD survive to adulthood. Despite these advances, CHD remains the leading cause of death in children with congenital malformations. The risk of morbidity and mortality begins to rise for patients who have had congenital heart disease that has been corrected or palliated when they reach older adulthood.⁹

3.1 Fetal and Postnatal Hemodynamic Adaptations in Congenital Heart Defects

Most congenital defects are well tolerated in the fetus because of the parallel nature of the fetal circulation. Even the most severe cardiac defects, such as hypoplastic left heart syndrome (HLHS), can usually be well compensated for by the fetal circulation. In HLHS the entire fetal cardiac output is ejected by the right ventricle via the ductus arteriosus into both the descending and ascending aorta (the latter filling in a retrograde fashion), so that fetal organ blood flow is minimally perturbed. Because the placenta provides for gas exchange and the normal fetal circulation has mixing between more highly and more poorly oxygenated blood at the foramen ovale and ductus arteriosus, fetal organ oxygen delivery is also not dramatically affected. It is only after birth, when the fetal pathways begin to close and

the umbilical cord is cut, that the full hemodynamic impact of an anatomic abnormality becomes apparent. One notable exception is the case of severe regurgitant lesions, most frequently of the tricuspid valve. In these lesions, such as Ebstein anomaly of the tricuspid valve or severe right ventricular outflow obstruction, the parallel fetal circulation cannot compensate for the severe volume or pressure load imposed on the right side of the heart.

In utero heart failure, often with fetal pleural and pericardial effusions, and generalized ascites (nonimmune hydrops fetalis) may occur. Although the most significant transitions in the circulation occur in the immediate perinatal period, the circulation continues to undergo changes after birth, and these later changes may also have a hemodynamic impact on cardiac lesions and their apparent incidence. When pulmonary vascular resistance (PVR) falls in the first several weeks of life, left-to-right shunting through intracardiac defects increases and symptoms and signs become more apparent. Thus in patients with a ventricular septal defect (VSD), heart failure is often first noticed between 1 and 3 months of age and not in the immediate newborn period. The severity of various defects can also change dramatically with growth; some VSDs may become smaller and even close as the child ages. Alternatively, stenosis of the aortic or pulmonary valve, which may be only moderate in the newborn period, may become worse if valve orifice growth does not keep pace with patient growth. The physician should always be alert for associated noncardiac congenital malformations, which can adversely affect the patient's prognosis. Developmental delay of various degrees is also a concern in many patients with CHD and may have its origins in alterations in fetal blood flow patterns caused by the heart defect, postnatal hypoxemia, and the effects of cardiopulmonary bypass during open heart surgery.⁹

2.2 Etiology of CHD

The cause of most congenital heart defects is still unknown. Many cases of CHD are multifactorial and may result from a combination of genetic predisposition and an as-yet-to-be-determined environmental stimulus. Overall, variants in approximately 400 genes have been identified as potentially causative of CHD. Despite these advances, half of cases still lack a known genetic cause. A small percentage of congenital heart lesions are related to known chromosomal abnormalities, in particular, trisomies 21, 13, and 18 and Turner syndrome; heart disease is found in >90% of patients with trisomy 18, 50% of patients with trisomy 21, and 40% of those with Turner syndrome. Ethnic factors may have a role in CHD; certain types of VSDs (supracristal) are more common in Asian children. There are also male:female differences for many common forms of CHD. The risk of CHD increases if a first-degree relative (parent or sibling) is affected, again emphasizing the role of genetics, even if the individual genes have not yet been identified.⁹

Table 1: Relative frequency of the most common congenital cardiac lesions.⁹

| LESION | % OF ALL LESIONS |
|-----------------------------------|-------------------------|
| Ventricular septal defect | 35–30 |
| Atrial septal defect (secundum) | 6–8 |
| Patent ductus arteriosus | 6–8 |
| Coarctation of aorta | 5–7 |
| Tetralogy of Fallot | 5–7 |
| Pulmonary valve stenosis | 5–7 |
| Aortic valve stenosis | 4–7 |
| d-Transposition of great arteries | 3–5 |

| | |
|---|------|
| Hypoplastic left ventricle | 1–3 |
| Hypoplastic right ventricle | 1–3 |
| Truncus arteriosus | 1–2 |
| Total anomalous pulmonary venous return | 1–2 |
| Tricuspid atresia | 1–2 |
| Single ventricle | 1–2 |
| Double-outlet right ventricle | 1–2 |
| Others | 5–10 |

2.3 Classification of CHD

Congenital heart defects (CHD) are classified based on their physiological impact on circulation. Broadly, they are divided into acyanotic and cyanotic defects. Acyanotic CHD includes defects with left-to-right shunting (e.g., atrial septal defect, ventricular septal defect, patent ductus arteriosus) and obstructive lesions without a shunt (e.g., aortic stenosis, pulmonary stenosis, coarctation of the aorta). These conditions do not cause immediate oxygen deprivation but can lead to heart failure over time. Cyanotic CHD, on the other hand, includes defects that cause right-to-left shunting, leading to reduced oxygenation of blood and cyanosis. Examples include Tetralogy of Fallot, transposition of the great arteries, tricuspid atresia, and hypoplastic left heart syndrome.

ACYANOTIC CONGENITAL HEART LESIONS

Acyanotic congenital heart defects (CHDs) are structural anomalies present at birth that typically do not result in cyanosis, or a bluish tint to the skin due to low oxygen levels. These defects are often categorized based on the primary physiological

burden they impose on the heart: increased volume load or increased pressure load. The most common lesions are those that produce a volume load, and the most common of these are the left-to-right shunt lesions. Atrioventricular (AV) valve regurgitation and dilated cardiomyopathies are other causes of increased volume load. The second major class of lesions causes an increase in pressure load, most often secondary to ventricular outflow obstruction (pulmonic or aortic valve stenosis) or narrowing of a great vessel (branch pulmonary artery stenosis or coarctation of the aorta). The chest radiograph and ECG are useful tools for differentiating between these major classes of volume and pressure-overload lesions while awaiting confirmation with echocardiography.¹¹

Lesions Resulting in Increased Volume Load

The most prevalent acyanotic CHDs causing increased volume load are those that facilitate left-to-right shunting of blood. This group includes atrial septal defects (ASD), ventricular septal defects (VSD), atrioventricular septal defects (AVSD), and patent ductus arteriosus (PDA). In these conditions, oxygen-rich blood is abnormally redirected from the systemic circulation back to the pulmonary circulation, leading to excessive pulmonary blood flow. The magnitude of this shunt is often quantified by the ratio of pulmonary to systemic blood flow ($Q_p:Q_s$), with a higher ratio indicating a more significant shunt. The direction and extent of the shunt are influenced by factors such as the size of the defect, relative pressures and resistances in the pulmonary and systemic circuits, and the compliance of the cardiac chambers involved.

As a result of the increased pulmonary blood flow, patients may develop symptoms of heart failure, including tachypnea (rapid breathing), tachycardia (rapid heart rate), sweating, nasal flaring, and wheezing. Infants with large left-to-right

shunts often exhibit signs of heart failure between one and three months of age, corresponding with the natural decline in pulmonary vascular resistance during this period. Chronic exposure to elevated pulmonary pressures can eventually lead to irreversible pulmonary hypertension, a condition known as Eisenmenger syndrome, where the shunt reverses direction, resulting in cyanosis and significant clinical deterioration.

Other conditions that impose a volume load on the heart include valvular regurgitation and dilated cardiomyopathies. Regurgitant lesions, such as those involving the atrioventricular valves, can exacerbate volume overload, leading to earlier and more severe heart failure symptoms. Dilated cardiomyopathies, characterized by impaired myocardial contractility, result in increased atrial and ventricular filling pressures, pulmonary congestion, and reduced cardiac output.¹¹

Lesions Resulting in Increased Pressure Load

Acyanotic CHDs that cause increased pressure load typically involve obstruction to blood flow, leading to elevated pressure proximal to the site of obstruction. Common examples include valvular pulmonic stenosis, valvular aortic stenosis, and coarctation of the aorta. In these conditions, the affected ventricle must generate higher pressures to overcome the obstruction, resulting in ventricular hypertrophy (thickening of the ventricular wall) as a compensatory mechanism. Over time, if the obstruction is severe and left untreated, the ventricle may develop fibrosis, dilate, and eventually progress to ventricular failure.¹¹

In neonates with critical obstruction, such as severe aortic stenosis or coarctation of the aorta, clinical manifestations can be profound, including signs of heart failure, poor perfusion, and, in severe cases, circulatory collapse. Coarctation of

the aorta, particularly when severe, may present with diminished or absent pulses in the lower extremities and differential cyanosis, where the upper body remains well-oxygenated while the lower body is cyanotic. Early recognition and intervention are crucial to prevent long-term complications and to improve outcomes in these patients. Understanding the classification and pathophysiology of acyanotic CHDs is essential for accurate diagnosis, timely management, and optimization of long-term outcomes for affected individuals.¹¹

CYANOTIC CONGENITAL HEART LESIONS

Cyanotic congenital heart defects (CHDs) can be classified based on their underlying physiological mechanisms. The primary distinction is between lesions that reduce pulmonary blood flow due to right ventricular outflow or inflow obstruction (e.g., tetralogy of Fallot, pulmonary atresia with or without a ventricular septal defect, and tricuspid atresia) and lesions that result in increased pulmonary blood flow with mixing of oxygenated and deoxygenated blood (e.g., transposition of the great arteries, truncus arteriosus, total anomalous pulmonary venous return [TAPVR] without obstruction, and single ventricle defects). A third subset includes TAPVR with obstruction, where blood flow from the pulmonary veins is impeded, leading to pulmonary congestion. Chest radiography plays a critical role in distinguishing between these categories based on lung vascularity.¹¹

Cyanotic Lesions with Decreased Pulmonary Blood Flow

For a cyanotic presentation to occur, defects in this group must include both an obstruction to pulmonary blood flow, which can occur at the tricuspid or pulmonary valve level, and an alternate right-to-left shunting pathway allowing systemic venous blood to bypass the lungs and enter systemic circulation. This shunting typically

occurs via an atrial septal defect (ASD), ventricular septal defect (VSD), or a patent foramen ovale. The severity of cyanosis is directly correlated with the degree of obstruction to pulmonary blood flow.¹¹

Common conditions in this group include tricuspid atresia, tetralogy of Fallot, tetralogy of Fallot with pulmonary atresia, pulmonary atresia with an intact ventricular septum, and various single ventricle anomalies accompanied by pulmonary stenosis or atresia. If pulmonary outflow obstruction is mild, cyanosis may not be apparent at rest, but these patients may experience episodic hypercyanotic (tet) spells in response to stress. In contrast, severe obstruction can render pulmonary circulation entirely dependent on ductal patency. Once the ductus arteriosus closes within the first few days of life, profound hypoxemia can develop rapidly, necessitating urgent medical intervention.¹¹

Cyanotic Lesions with Increased Pulmonary Blood Flow

Unlike the previous group, these lesions do not involve pulmonary outflow obstruction. Instead, cyanosis results from either abnormal ventricular-arterial connections or a complete mixing of deoxygenated systemic venous blood with oxygenated pulmonary venous blood within the heart. The most common example of an abnormal ventricular-arterial connection is transposition of the great arteries (TGA). In TGA, the aorta arises from the right ventricle while the pulmonary artery originates from the left ventricle. This anatomical configuration leads to a parallel rather than a series circulation, where systemic venous blood bypasses the lungs and is recirculated to the body, while oxygenated pulmonary venous blood is sent back to the lungs. Temporary mixing via the patent foramen ovale or ductus arteriosus can sustain a limited oxygen supply in the immediate neonatal period, but as these fetal

shunts begin to close, systemic oxygen saturation drops dramatically, causing severe cyanosis.¹¹

Another subset of this category includes total mixing lesions, in which all venous blood—both systemic and pulmonary—combines at a common site in the heart. Examples include truncus arteriosus, TAPVR, and single ventricle physiology. In these conditions, oxygen saturation levels in both the pulmonary artery and aorta are identical due to the complete mixing of blood. If there is no obstruction to pulmonary blood flow, the combination of cyanosis and pulmonary overcirculation can lead to heart failure. However, if pulmonary stenosis is present, cyanosis may be the dominant clinical feature, resembling the presentation seen in tetralogy of Fallot.¹¹

2.4 Evaluation and Screening of the infant or child with CHD

The initial evaluation for suspected congenital heart disease (CHD) involves a systematic approach with two major components. First, congenital cardiac defects can be divided into two major groups based on the presence or absence of cyanosis, which can be determined by physical examination aided by pulse oximetry. Second, these groups can usually be further subdivided based on whether the chest radiograph shows evidence of increased, normal, or decreased pulmonary vascular markings. Next, the electrocardiogram (ECG) can be used to determine whether right, left, or biventricular hypertrophy exists. The character of the heart sounds and the presence and character of any murmurs further narrow the differential diagnosis. The final diagnosis is then confirmed by echocardiography, cardiac computed tomography (CT) or magnetic resonance imaging (MRI), and/or cardiac catheterization. In a cyanotic or otherwise sick newborn, echocardiographic exam, if available, should not be delayed while awaiting these other modalities.¹¹

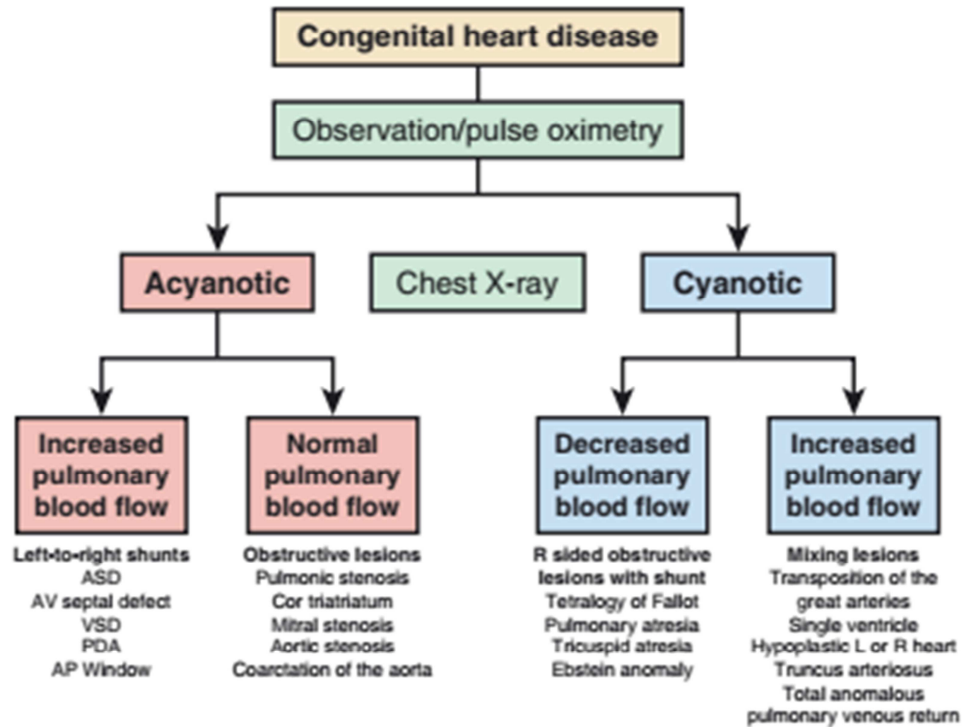


Fig 1: A general algorithmic approach to the initial diagnosis of congenital heart disease, based on observation/pulse oximetry and chest x-ray, to separate patients into four major physiologic subgroups of congenital heart disease. This schematic is a broad, but useful, diagnostic overview; only the most common forms of congenital heart disease are included. A patient’s initial presentation may sometimes straddle two of these physiologic groups and can evolve between them over the first week or two of life. For example, in a patient with a VSD, the pulmonary blood flow will be relatively normal in the newborn period and only increase as the pulmonary vascular resistance begins to drop. Similarly, a patient with a mixing lesion may not show pulmonary overcirculation at birth. ASD, Atrial septal defect; AV, atrioventricular; VSD, ventricular septal defect; PDA, patent ductus arteriosus, AP, aortopulmonary; L, left; R, right¹¹

Routine pulse oximetry screening is recommended for all newborns to detect unsuspected critical cyanotic CHD; lesions include hypoplastic left heart syndrome, pulmonary atresia, tetralogy of Fallot, total anomalous pulmonary venous return, transposition of the great arteries, tricuspid atresia, truncus arteriosus, neonatal coarctation of the aorta, and aortic arch hypoplasia/atresia. Many of these lesions are ductal dependent, and if the ductus arteriosus closes, severe cardiac decompensation will ensue. In addition, pulse oximetry may detect respiratory disorders and primary pulmonary hypertension. Screening is performed between 24 and 48 hours of life and before discharge in asymptomatic newborns. The neonate passes if the oxygen saturation is 95% or greater in the right hand or either foot and the difference is 3% or less between the right hand and foot. The screen fails if the saturation is less than 90% in either the right hand or foot. If it is between 90% and 94% OR there is more than a 3% difference between the right arm and foot, the screen should be repeated once in 1 hour. If the saturation is 90–94% OR there is a 3% difference after the third screen, urgent echocardiography is indicated. In addition, a careful reexamination of the pulses and blood pressure in the upper and lower extremity and cardiac auscultation are indicated in children with an initial positive screen.¹¹

2.5 Impact of CHD on growth

The development of a child is a dynamic process that is impacted by the interaction of numerous elements, including biological, psychological, and social components. It is directly related to a child's age and is influenced by interactions with individuals in their social surroundings¹² Research has highlighted the significance of evaluating environmental conditions and the stimuli provided by families, as these factors play a crucial role in shaping child development. Such assessments can offer valuable insights for designing preventive and health-promoting interventions¹³.

From a biological perspective, various chronic illnesses can impact pediatric development, with congenital heart disease (CHD) being a prominent example. CHD encompasses a broad spectrum of structural and functional cardiac abnormalities that can significantly affect a child's overall well-being^{14,15}. A number of symptoms, including dyspnoea, weariness, dizziness, low body weight, frequent respiratory infections, arrhythmias, and cyanosis, are frequently associated with the presence of congenital heart disease (CHD). In some cases, the severity of these symptoms might result in physical and motor restrictions, which in turn can have a direct impact on the development of both emotional and cognitive abilities¹⁶. The impact of CHD extends beyond physical health, as cultural and environmental factors also play a critical role in shaping neurodevelopment. Socioeconomic status, for instance, influences access to education, nutritional adequacy, medical care quality, and overall stimulation, all of which contribute to cognitive and emotional growth. Other influential determinants include perinatal risks, parental occupation, family dynamics, social engagement, and housing conditions¹³.

Children with CHD often exhibit delays in psychomotor development due to both pathophysiological and environmental factors. Physiological issues such as low birth weight, chronic hypoxia, and cyanosis contribute to developmental challenges. Additionally, the natural course of CHD can significantly impair growth due to chronic hypoxemia, inadequate oxygen delivery to tissues, and increased metabolic demands. Infants with CHD, particularly those with cyanotic heart defects, often experience failure to thrive due to increased caloric expenditure associated with tachypnea, labored breathing, and increased cardiac workload. Moreover, poor appetite and feeding difficulties are common due to early fatigue, gastroesophageal reflux, and fluid restriction in those with heart failure. Malabsorption resulting from

intestinal hypoperfusion further exacerbates nutritional deficiencies, leading to stunted linear growth and poor weight gain.^{17,18,19}

Frequent hospitalizations, recurrent infections, and the need for multiple surgical interventions disrupt normal growth patterns. Prolonged illness can also lead to endocrine disturbances, including alterations in growth hormone secretion and resistance to insulin-like growth factor 1 (IGF-1), both of which are critical for normal development. Furthermore, chronic inflammation and oxidative stress contribute to muscle wasting and delayed pubertal development in older children. Prolonged oxygen deprivation in cyanotic CHD cases may also affect brain growth and maturation, leading to neurodevelopmental impairments.¹⁹

Additionally, the chronic nature of the disease necessitates frequent medical interventions and physical restrictions, which can disrupt social participation and academic progress. Prolonged absences from school and reduced peer interactions may hinder social and emotional development, leading to lower adaptive functioning. Given this complex interplay of biological and environmental influences, children with CHD, particularly those with severe forms of the condition, often demonstrate significantly lower developmental scores compared to their healthy peers.¹⁹

2.6 Nutritional Challenges

Impaired growth has become a notable concern for children with CHD both before and after cardiac surgery, irrespective of the type of cardiac defect and the presence or absence of cyanosis.²⁰⁻²² Most children with CHD born at term gestational age have normal anthropometric indices, however soon after birth many begin to exhibit growth challenges including deficits in weight, height and head circumference that place them at high risk for malnutrition and failure to thrive²³⁻²⁵. The terms

malnutrition and undernutrition are often used interchangeably. Malnutrition encompasses three broad groups of conditions including undernutrition, overnutrition and micronutrient related malnutrition. Undernutrition refers to the insufficient intake of calories and nutrients and includes underweight (low weight-for-age), stunting (low height for-age) and wasting (low weight-for-height)²⁶. It is recognized that the cause of malnutrition, specifically undernutrition, in children with CHD is multifactorial. Causes include the underlying cardiac abnormality itself, hemodynamic factors, chronic hypoxemia, inadequate calorie intake, increased energy expenditure in relation to intake (hypermetabolism), malabsorption, co-occurring genetic comorbidity and feeding and swallowing difficulties (FSDs)²⁷⁻³⁰. Difficulties in coordinating breathing with swallowing due to shortness of breath resulting from congestive cardiac failure (CCF) or respiratory infection, neurobehavioral challenges, fatigue due to poor physical endurance, dysphagia, gastroesophageal reflux disease (GERD), cleft lip palate (CLP), oral aversion, delayed acquisition of feeding skills and vocal cord dysfunction caused by laryngeal nerve injury during cardiac surgery contribute to FSDs^{31,32}. FSDs result in inadequate calorie intake, making the early recognition and management thereof important to improve growth outcomes^{31,32}. Malnutrition prior to cardiac surgery contributes to delayed cardiac surgery, and poorer short and long term outcomes including longer hospital length of stay (HLOS), higher risk of infection, poorer clinical and neurodevelopmental outcomes, and poorer health related quality of life (HRQOL)^{21,22,33-36}. Furthermore, growth failure and FSDs contribute to increased levels of parenting stress^{35,37-41}. Similar to other children, children with CHD are also at risk for malnutrition secondary to social and economic factors^{42,43}.

The reported overall prevalence of moderate malnutrition in young children with CHD in high income countries (HICs) ranges from 21 to 29%^{20,22,29,33}. A recent systematic review and meta-analysis on the prevalence on malnutrition in children with CHD found that 27.4% of all children with CHD were underweight and 24.4% were stunted³³. This is significantly higher than the estimated prevalence of underweight of 6.1% and stunting of 7.6% reported for the global population of otherwise healthy same-aged children^{44,45}. Admittedly early intervention for CHD is shown to improve catch-up growth^{27,46}. Published data on malnutrition in children with CHD living in low-to-middle income countries (LMICs), though far from scanner, suggests the prevalence of growth failure is substantially higher and the extent of the malnutrition greater than for children living in HICs³³. Moderate malnutrition is said to occur in more than half of children with CHD living in LMICs, and severe malnutrition is noted to be as high as 60% in some cases^{30,46-49}. Overall rates of malnutrition as high as 90% have been reported in some studies^{30,47-55}. It is apparent that poverty, delayed diagnosis and later age at corrective surgery exacerbate the extent of the growth failure in children with CHD in LMICs^{27,56}.

2.7 Corrective surgery and Growth outcomes :^{4, 24, 30,57-59}

Corrective surgery for congenital heart disease (CHD) in children under five is pivotal in improving survival rates and enhancing growth outcomes. Preoperatively, children with CHD often experience growth impairments due to factors like malnutrition, feeding difficulties, and increased metabolic demands. Post-surgical interventions have demonstrated significant improvements in growth parameters, though challenges remain.

Preoperative Growth Challenges

Children with CHD frequently present with undernutrition and growth delays before surgery. A study conducted in South Africa highlighted that malnutrition is prevalent among young children with CHD, exacerbated by feeding difficulties, poverty, delayed diagnosis, and late corrective surgeries. This undernutrition is strongly associated with poorer motor development prior to surgical intervention.

Postoperative Growth Improvements

Following corrective surgery, many children exhibit notable improvements in growth metrics. Research indicates significant gains in weight and head circumference within six months post-surgery, although complete catch-up growth may not be achieved in this timeframe. This underscores the positive impact of surgical intervention on reversing growth delays associated with CHD.

Factors Influencing Post-Surgical Growth

Despite surgical advancements, several factors can influence postoperative growth outcomes: **Nutritional Status:** Persistent feeding challenges and inadequate nutritional support can hinder optimal growth recovery. Early referral for nutritional support and speech therapy is recommended to address these issues.

Severity of CHD: Complex heart defects may require multiple surgeries, potentially impacting the child's overall growth trajectory.

Socioeconomic Factors: Limited access to healthcare resources and socioeconomic constraints can affect both the timing of surgery and postoperative care, influencing growth outcomes.

FACTORS INFLUENCING GROWTH POST-CORRECTIVE SURGERY^{4, 24,}

30,57-59

Post-corrective surgery growth in children with congenital heart disease (CHD) is influenced by several interrelated factors:

- **Nutritional Intake and Feeding Challenges:** Postoperative feeding difficulties can persist, impacting caloric intake and growth. Increased metabolic demands due to residual cardiac issues may exacerbate this challenge. Addressing these issues is crucial for promoting optimal growth.
- **Severity and Type of Cardiac Lesion:** The specific nature of the CHD and the complexity of the surgical repair can affect recovery and subsequent growth. Children with more complex defects may experience different growth trajectories post-surgery.
- **Socioeconomic Status and Access to Healthcare:** Limited access to healthcare resources and socioeconomic constraints can delay interventions and affect postoperative care, influencing growth outcomes. Early diagnosis and timely surgical intervention are crucial for improving growth trajectories.
- **Genetic and Syndromic Associations:** Children with CHD may have associated genetic syndromes that inherently affect growth potential, independent of surgical outcomes. These genetic factors can influence both pre- and post-surgical growth patterns.
- **Postoperative Complications:** Complications such as infections or residual cardiac dysfunction can prolong recovery and negatively impact growth. Effective management of these complications is essential to support growth post-surgery.
- **Timing of Surgical Intervention:** Early corrective surgery is associated with better growth outcomes, as prolonged periods of cardiac insufficiency can lead

to more significant growth delays. Timely intervention helps in minimizing the duration of compromised cardiac function, thereby supporting better growth trajectories.

2.8 LITERATURE FROM PREVIOUS STUDIES:

In order to evaluate the prevalence of failure to thrive (FTT) and the factors that contribute to it, He et al. (2024) carried out a study that was a cross-sectional investigation including 13,256 children who were diagnosed with congenital heart disease (CHD). In the cohort, the prevalence of FTT, which is defined as stunting or underweight, was found to be 36.9%, with the prevalence increasing to 54.2% in patients of complicated congenital heart. Preoperative anaemia, left ventricular systolic dysfunction, younger age, lower birth weight, and hereditary abnormalities were all factors that contributed to the risk of the condition taking place. Particularly noteworthy is the fact that the CHD subtype that was shown to be most closely connected with severe FTT was interrupted aortic arch (IAA).⁵⁷

Murni et al. (2023) conducted a study in Indonesia to investigate the prevalence of undernutrition among children who were diagnosed with congenital heart disease (CHD) and the factors that contributed to it. A total of 1,149 children participated in this prospective cohort study. Of those children, 49 percent were underweight, 47.8 percent were stunted, and 31.4% were wasting. The presence of cyanotic congenital heart disease, delayed diagnosis, congestive heart failure, pulmonary hypertension, low birth weight, and young mother age were considered to be independent factors that were related with undernutrition. A substantial association was found between undernutrition and an increased risk of mortality, with children who were underweight exhibiting an odds ratio (OR) of 3.54 and children who were stunted exhibiting an OR of 3.31.⁵⁸

Congenital heart disease (CHD) patients up to the age of ten were the subjects of a longitudinal study that was carried out by Hapuoja et al. (2021). The purpose of the study was to investigate somatic growth patterns and related risk factors in these patients. For the purpose of the study, 135 children who had undergone cardiopulmonary bypass surgery were tracked. During the period of time between the ages of one and ten years, the authors noticed a significant catch-up increase in height, weight, and head circumference (HC). However, both weight and HC remained below the normative norms at the ten-year mark. This study found that weight and body mass index (BMI) trajectories were influenced by socioeconomic status and gestational age. Additionally, prolonged hospitalisation durations following surgery were related with decreased growth.¹⁰

In China, Li et al. (2021) conducted a study in which they examined the growth and developmental outcomes of 262 children under the age of five who were diagnosed with tetralogy of Fallot (TOF). The study compared the participants' height, weight, and body mass index (BMI) to world health organisation (WHO) growth norms. A considerable amount of growth retardation was discovered, with 21.4% of children exhibiting stunting, 14.5% being underweight, and 9.9% being wasting according to the findings. When compared to babies (0–12 months), older children (13–60 months) were more likely to have stunting, which is indicative of a gradual decline in growth that occurs with increasing age.⁵⁹

At a teaching hospital in Nigeria, Arodiwe et al. (2015) conducted an investigation into the prevalence of malnutrition and the factors that contribute to it among children who were diagnosed with congenital heart disease (CHD). In this descriptive cross-sectional investigation, fifty patients with congenital heart disease were assessed, and ninety-two percent of them had malnutrition. At sixty percent,

severe malnutrition was the prevalent condition. Among the factors that significantly contributed to malnutrition were pulmonary hypertension, inadequate food intake, and a delay in seeking medical attention. Individuals with cyanotic pulmonary hypertension were more likely to exhibit stunting, whereas individuals with acyanotic pulmonary hypertension were more likely to exhibit wasting.⁴⁸

A retrospective matched cohort study was carried out by Daymont et al. (2013) with the purpose of analysing growth trends in children who were diagnosed with congenital heart disease (CHD) in comparison to these of healthy controls. A total of 856 children with congenital heart disease were involved in the study. These children were divided into four groups: single ventricle (SV) physiology, complicated repair (CR), simple repair (SR), and no repair (NR). Early and simultaneous impairments in weight-for-age (WFA), length-for-age (LFA), and head circumference-for-age (HCFA) z-scores were observed within the first month of life, with deficits reaching their peak at four months and continuing through 24 to 36 months, depending on the severity of the impairments.²⁴

At the Liaquat University Hospital in Hyderabad, Pakistan, Mohammad et al. (2014) carried out a descriptive cross-sectional study with the purpose of analysing the spectrum of heart illnesses that are prevalent in children under the age of five. Among the 150 children who were suspected to have cardiac difficulties, 89.3 percent were diagnosed with congenital heart disease (CHD). The prevalence of acyanotic defects was significantly higher (74.6%) than the prevalence of cyanotic defects (23.9%). Ventricular septal defect was the most common acyanotic lesion, accounting for 29.9% of all cases, whereas tetralogy of Fallot was the most common cyanotic defect, accounting for 11.2% of all cases. The majority of acquired cardiac disorders were caused by myocarditis (94%). The majority of patients manifested themselves at

the infant stage with symptoms such as respiratory distress (80%) and difficulties with feeding (58%).⁶⁰

Okoromah et al. (2011) conducted a case-control observational study in Nigeria to evaluate the prevalence of malnutrition among children with congenital heart abnormalities (CHD), as well as the profile of malnutrition and the factors that make it more likely to occur. The prevalence of malnutrition was 90.4% among the 73 children who had symptomatic congenital heart disease, with 61.2% demonstrating severe manifestations. While stunting was more prevalent in cyanotic congenital heart disease (68.0%), wasting was more prevalent in acyanotic congenital heart disease (58.3%). A number of factors, including anaemia, congestive heart failure, poor fat intake, and chronic unoperated disease, were found to be significant predictors.³⁰

Researchers Vaidyanathan et al. (2008) investigated the factors that contribute to malnutrition in infants who were born with congenital heart disease (CHD) and investigated the effect that corrective intervention had on the nutritional outcomes of these children. A total of 476 children who were receiving corrective procedures were evaluated for their nutritional status both before and after surgery over the course of a period of three months. Malnutrition, which is defined as z-scores that are less than or equal to two, was found to be widespread among 59% (weight), 26.3% (height), and 55.9% (weight/height) of children initially. A number of characteristics, including congestive heart failure, age at correction, low birth weight, and socioeconomic determinants, were included in the list of candidates for prediction. After the intervention, statistically significant improvements in nutritional status were reported across the board for all metrics.⁴

In order to investigate the neurodevelopmental outcomes at one year of age in newborns with congenital heart disease (CHD) who had undergone corrective or palliative surgery, Dittrich et al. (2003) carried out a prospective cohort study. It was shown that the mean developmental quotient (DQ) of CHD patients (99) was considerably lower than that of controls (106.7) among the 90 newborns who were considered for the study. A mean DQ of 88 was found among infants who underwent palliative surgery, which was significantly lower than the DQ of 101.4 for those who underwent corrective surgery. In the case of congenital heart disease (CHD), palliative surgery newborns had greater rates of developmental delay (DQ < 93.5) compared to corrective surgery infants (19%). This was observed in 27% of the patients. In addition, the incidence of neurological abnormalities was much higher in cases of palliative surgery (69%) compared to cases of corrective surgery (24%).⁶¹

MATERIALS AND METHODS

4.1 Source of Data: Children under 5 years of age diagnosed with congenital heart disease and admitted in general paediatric ward and general cardiac ward of Dr. Prabhakar Kore Charitable Hospital, Belagavi for corrective surgery with follow up measurements taken at 3 months post surgery in paediatric cardiology OPD of Dr. Prabhakar Kore Charitable Hospital, Belagavi. From August 2023 to July 2024

4.2 Methods of collection of Data:

A. STUDY DESIGN: Longitudinal Observational Study

B. PLACE OF STUDY: The present study was carried out in the Department of Paediatrics, Dr Prabhakar Kore Charitable Hospital, Belagavi, Karnataka.

C. STUDY PERIOD: 1 Year (August 2023 to July 2024)

D. SAMPLE SIZE:

Formula used for sample size calculation is,

$$n = \frac{p(100 - p)Z^2}{E^2}$$

where n is the sample size required, p is the percentage occurrence of a state or condition (proportion or prevalence), E is the percentage maximum error required, Z is the value corresponding to level of confidence required.

The z-score of weight was observed to be less than -2 in 59% children aged <5 years undergoing surgical or catheter-based corrective intervention. Considering similar result at 95% confidence level and 10% maximum error, the sample size is given by,

$$n = \frac{59 \times (100 - 59) \times 1.96^2}{10^2}$$

$$n = 92.9283 \approx 93$$

Considering 10% loss to follow up, the minimum sample size required is 102.

As sample size increases, accuracy of result also increases.

E. INCLUSION CRITERIA:

- Children under 5 years of age with congenital heart disease fit for corrective surgery.
- Children under 5 years of age with failure to thrive with heart disease

F. EXCLUSION CRITERIA:

- Children under 5 years of age undergoing palliative surgery.
- Children under 5 years of age with congenital heart disease with other comorbidities like proven chromosomal abnormalities such as downs syndrome, diagnosed with metabolic disorders like galactosemia and hypothyroidism, congenital anomalies that restrict oral feeding like cleft palate and cleft lip.
- Children whose parents didn't give consent for the study

G. METHODOLOGY:

After obtaining informed consent from the parents, comprehensive demographic and clinical data were recorded, including the child's birth history, presenting complaints, parental educational qualifications, and socioeconomic status. Anthropometric measurements—height/length, weight, head circumference, and mid-upper arm circumference—were assessed preoperatively (baseline) and at three months postoperatively using standardized scales. These measurements were then

plotted on WHO growth charts based on the child's age and gender. Additionally, data on the duration of hospital stay, presence of sepsis at the time of admission, and severity of pulmonary arterial hypertension (as determined by 2D echocardiography) were documented.

F. MATERIALS USED:

- For weight measurement, multipurpose digital weighing scale ranging from 50g to 120kgs with 5g division was used (meditive brand)
- For height/length measurement, Indosurgicals infantometer which is calibrated till 90 cm with 1mm graduation was used for neonates and for children under 5 years IS Indosurgical height measuring scale measuring from 20 cm to 210cm with 1mm graduation was used.
- For measuring head circumference, IS Indosurgicals head circumference measuring tape calibrated upto 60cm and with 1mm graduation was used.
- For measuring mid upper arm circumference, a non stretchable measuring tape was used

H. DATA PROCESSING AND ANALYSIS:

With the collected data, growth parameters such as height for age(HAZ), weight for age(WAZ), weight for height/length(WHZ/WLZ), for age, head circumference for age (HCZ) was calculated and converted in terms of z scores , after which they were compared with normal growth parameters according to standard WHO growth parameters for respective age and gender. Pre operative and the postoperative growth parameters of each child were compared and malnutrition patterns such as stunting, wasting, underweight etc were analyzed. The

socioeconomic status of the parents was analysed using a modified Kuppaswamy scale. Using the history noted, the effect of various factors such as educational qualification of parents, duration of hospital stay etc on growth will be analysed for potential malnutrition predictors.

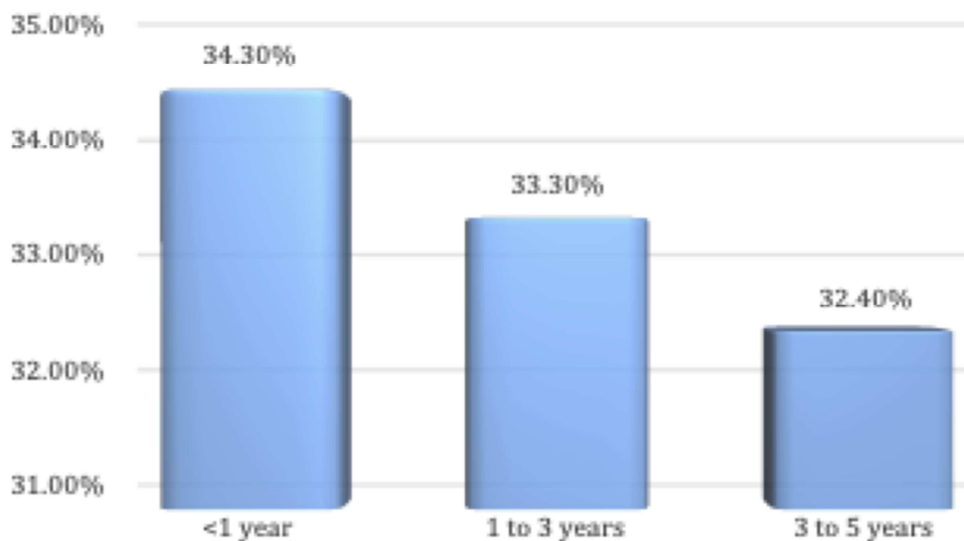
4.3 STATISTICAL ANALYSIS:

Data entry was done using M.S. Excel and statistically analysed using Statistical package for social sciences (SPSS Version 16) for M.S Windows. Descriptive statistical analysis was carried out to explore the distribution of several categorical and quantitative variables. Categorical variables were summarized with n (%), while quantitative variables were summarized by mean \pm S.D. All results were presented in tabular form and are also shown graphically using bar diagram or pie diagram as appropriate. The difference in the two groups was tested for Statistical Significance using Parametric tests such as t-test and categorical variables tested by chi square test. P-value <0.05 was considered statistically significant after assuming all the rules of statistical tests.

RESULTS

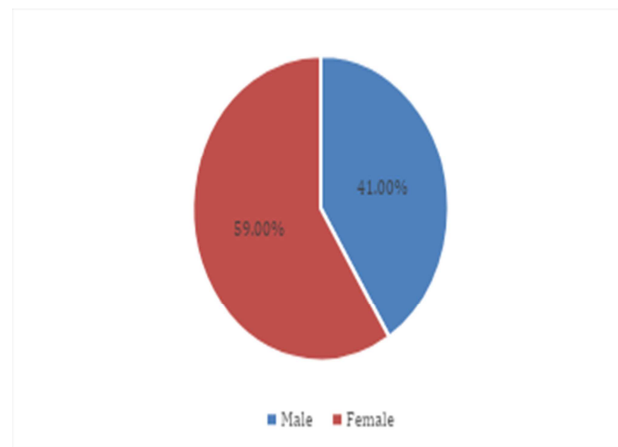
Of the initial 116 children diagnosed with CHD, 105 were included in our study after obtaining informed parental consent, following the exclusion of 2 patients who succumbed post-surgery and 9 patients who were lost to follow-up. Among these children, the largest proportion (34.3%) who underwent surgery were under 1 year of age, followed closely by those aged 1 to 3 years (33.3%) and 3 to 5 years (32.4%)(FIGURE 2). Mean age of the study population was found to be 2.31 ± 1.09 years.

Figure 2: Bar graph showing age wise Distribution of Children Undergoing Corrective Surgery



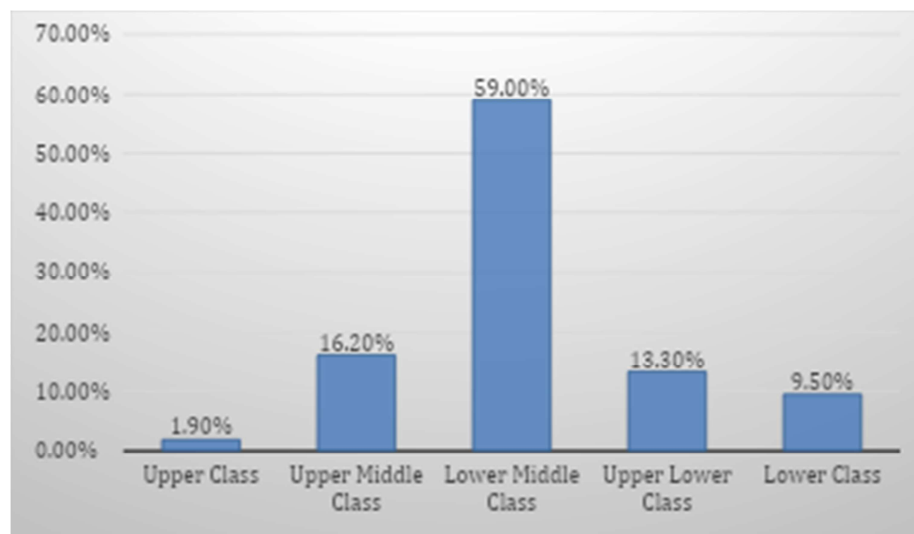
In our study population, 62 (59.0%) were females, and 43 (41.0%) were males. (Figure 3).

Figure 3: Pie chart showing Gender Distribution of Children Undergoing Corrective Surgery



Furthermore, the distribution of our study population based on socio-economic status, using the modified Kuppuswamy scale, showed that the majority (59.0%) belonged to the lower middle class, followed by 16.2% in the upper middle class, 13.3% in the upper lower class, and 9.5% in the lower class. Only 1.9% of the children belonged to the upper class. (Figure 4)

Figure 4: Bar graph depicting distribution of study population based on socioeconomic status



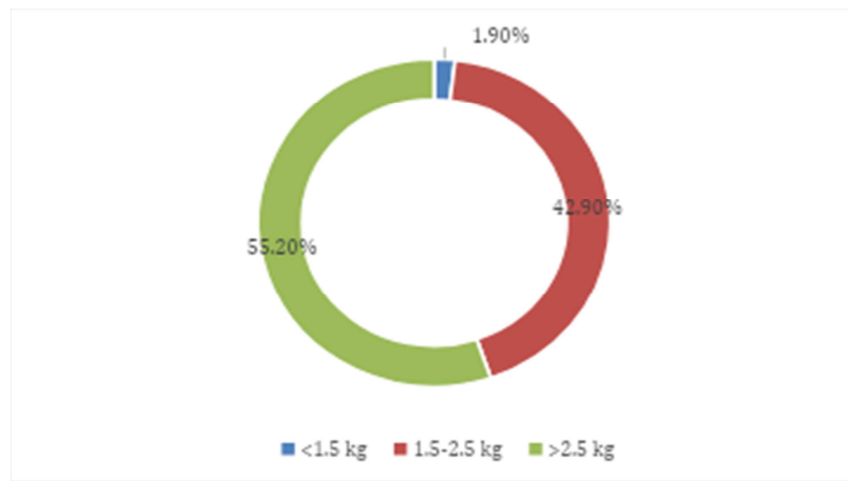
Out of the total 105 cases, 90.5% of mothers had no significant antenatal complications. However, 9.5% of mothers reported a history of antenatal complications, including gestational diabetes mellitus (GDM) (1.9%), hypothyroidism (1.9%), pregnancy-induced hypertension (PIH) (1.9%), anemia (1.0%), chronic hypertension (1.0%), hypotension (1.0%), and pre-eclampsia (1.0%). (Table 2)

Table 2: Distribution of Antenatal History of Mothers of Children Undergoing Corrective Surgery

| | | Frequency | Percent |
|----------------------|-----------------------|------------------|----------------|
| Antenatal H/O | Chronic HTN | 1 | 1.0% |
| | GDM | 2 | 1.9% |
| | Hypotension | 1 | 1.0% |
| | Hypothyroidism | 2 | 1.9% |
| | PIH | 2 | 1.9% |
| | Anemia | 1 | 1.0% |
| | Pre-Eclampsia | 1 | 1.0% |
| | Nil | 95 | 90.5% |
| | Total | 105 | 100.0% |

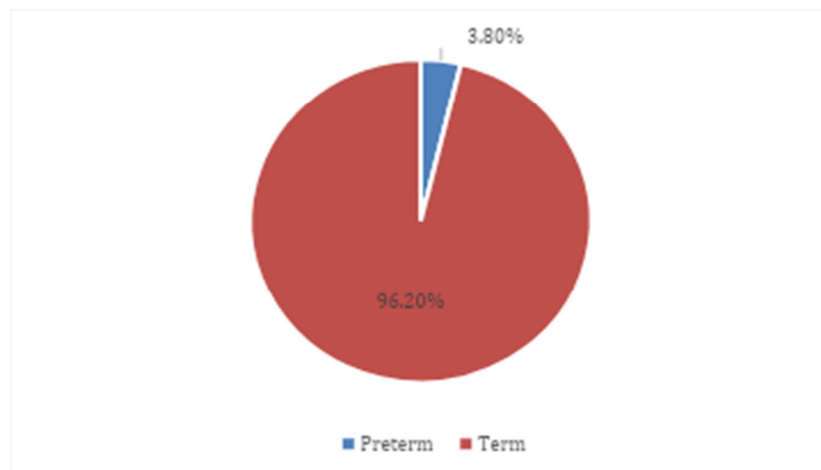
Further, 58 (55.2%) children out of the study population, had a birth weight greater than 2.5 kg, while 45 (42.9%) children had a birth weight between 1.5 and 2.5 kg. Only 2 children (1.9%) had a low birth weight of less than 1.5 kg. (Figure 5)

Figure 5: Donut pie chart showing distribution of birth weight of Children Undergoing Corrective Surgery



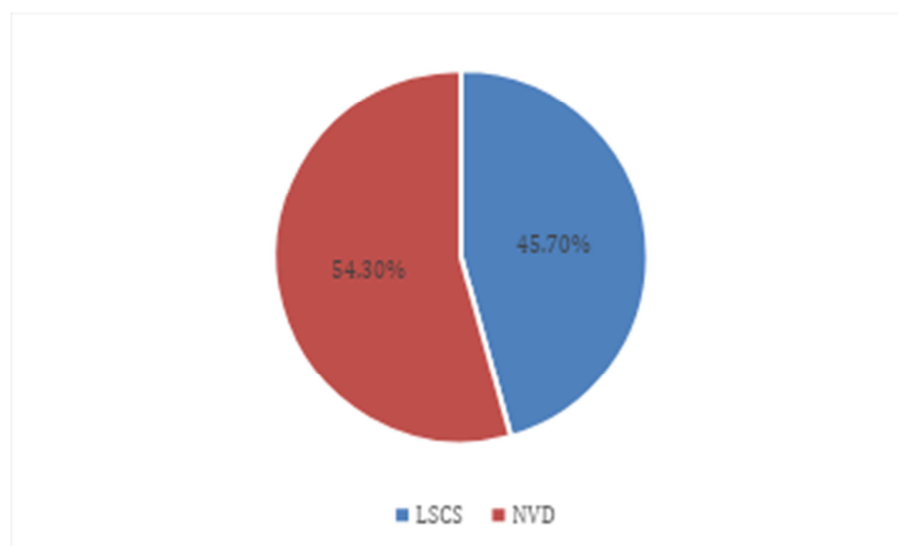
The distribution of the study population based on gestational age at birth revealed that the majority (96.2%) were born at term, while only four children (3.8%) were born preterm (Figure 6)

Figure 6: Pie chart showing distribution of study population based on Gestational Age at Birth.



Out of these children, 57 (54.3%) were delivered through normal vaginal delivery (NVD), while 48 (45.7%) were delivered via lower segment cesarean section (LSCS). (Figure 7)

Figure 7: Pie chart depicting distribution of study population based on Mode of Delivery of Children at birth

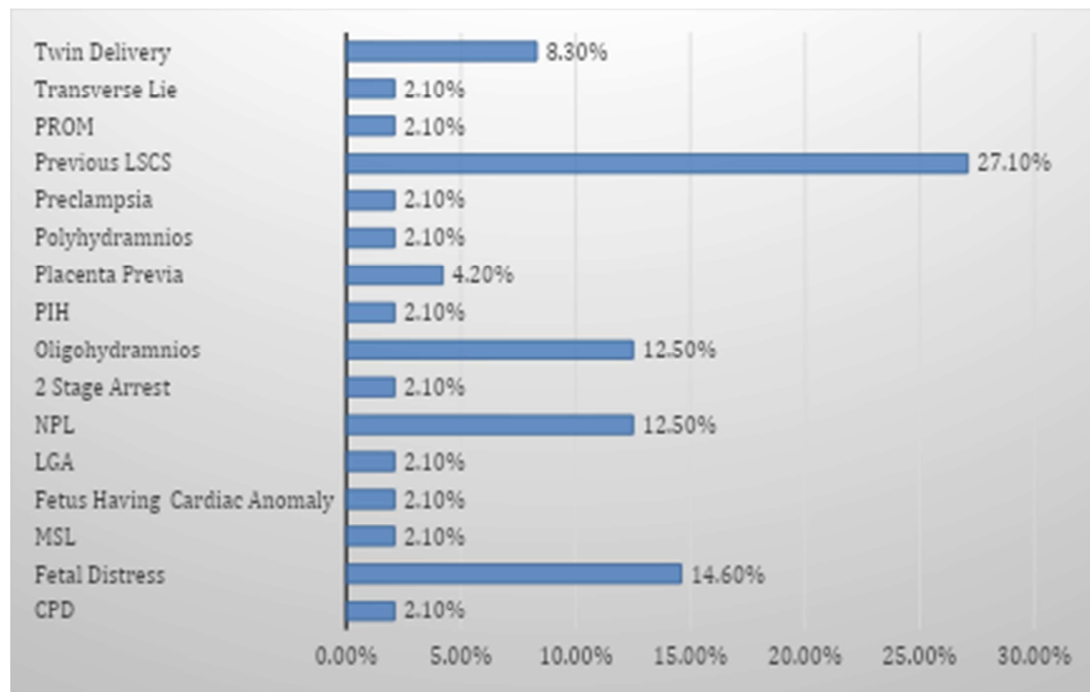


Among the 48 cesarean deliveries, the most common indication was a history of previous LSCS (27.1%), followed by fetal distress (14.6%), non-progressive labor (NPL) (12.5%), and oligohydramnios (12.5%). Other notable indications included twin delivery (8.3%), placenta previa (4.2%), and fetal cardiac anomaly (2.1%). Less frequent indications included cephalopelvic disproportion (CPD), meconium-stained liquor (MSL), preeclampsia, polyhydramnios, premature rupture of membranes (PROM), transverse lie, and large for gestational age (LGA), each accounting for 2.1%. Antenatal detection of fetal cardiac anomaly was found only in 1% of the total study population (n=1) at our centre. (Table 3)(Figure 8)

Table 3: Indications for Cesarean Section in Children Undergoing Corrective Surgery

| | Frequency | Percent | |
|------------|------------------------------|---------|-------|
| Indication | CPD | 1 | 2.1% |
| | Fetal Distress | 7 | 14.6% |
| | MSL | 1 | 2.1% |
| | Fetus Having Cardiac Anomaly | 1 | 2.1% |
| | LGA | 1 | 2.1% |
| | NPL | 6 | 12.5% |
| | 2 Stage Arrest | 1 | 2.1% |
| | Oligohydramnios | 6 | 12.5% |
| | PIH | 1 | 2.1% |
| | Placenta Previa | 2 | 4.2% |
| | Polyhydramnios | 1 | 2.1% |
| | Preclampsia | 1 | 2.1% |
| | Previous LSCS | 13 | 27.1% |
| | PROM | 1 | 2.1% |
| | Transverse Lie | 1 | 2.1% |
| | Twin Delivery | 4 | 8.3% |
| Total | 48 | 100.0% | |

Figure 8: Horizontal Bar chart showing Indications for Cesarean Section in Children Undergoing Corrective Surgery.



Atrial septal defect (ASD) was the most common cardiac defect found in our study, with 31 children (29.5%), followed closely by ventricular septal defect (VSD) in 30 children (28.6%) and patent ductus arteriosus (PDA) in 22 children (21.0%). More complex cases included tetralogy of Fallot (TOF) (4.8%), PDA with aortic stenosis (2.9%), and VSD with PDA (2.9%). Other less common defects accounted for 10.5% of the cases. (Table 4)(Figure 9)

Table 4: Type of Congenital Heart Disease (CHD) in Children Undergoing Corrective Surgery

| | | Frequency | Percent |
|-----|--------------|-----------|---------|
| CHD | ASD | 31 | 29.5 |
| | VSD | 30 | 28.6 |
| | PDA | 22 | 21.0 |
| | TOF | 5 | 4.8 |
| | PDA WITH AS | 3 | 2.9 |
| | VSD WITH PDA | 3 | 2.9 |
| | Others | 11 | 10.5 |
| | Total | 105 | 100.0 |

Figure 9: Bar chart showing distribution of type of Congenital Heart Disease (CHD) in Children Undergoing Corrective Surgery

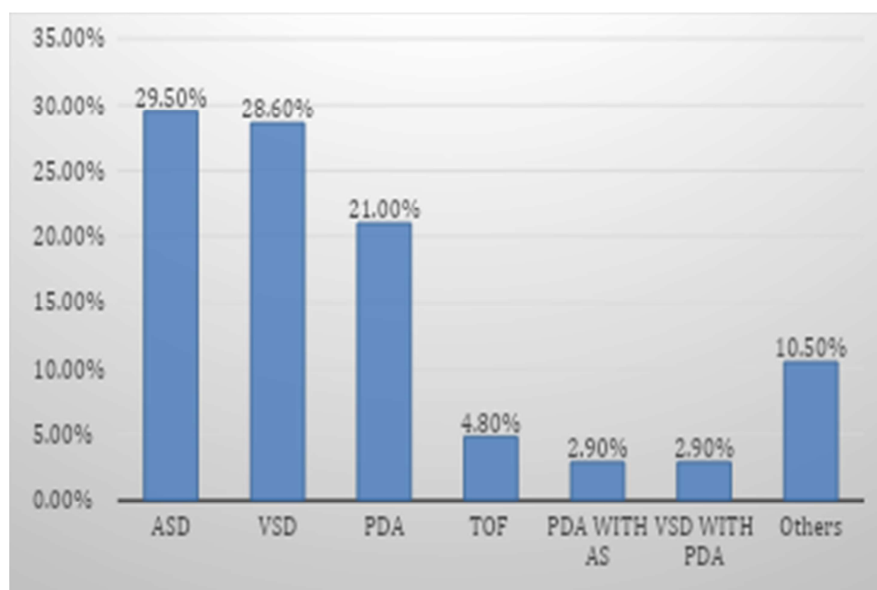
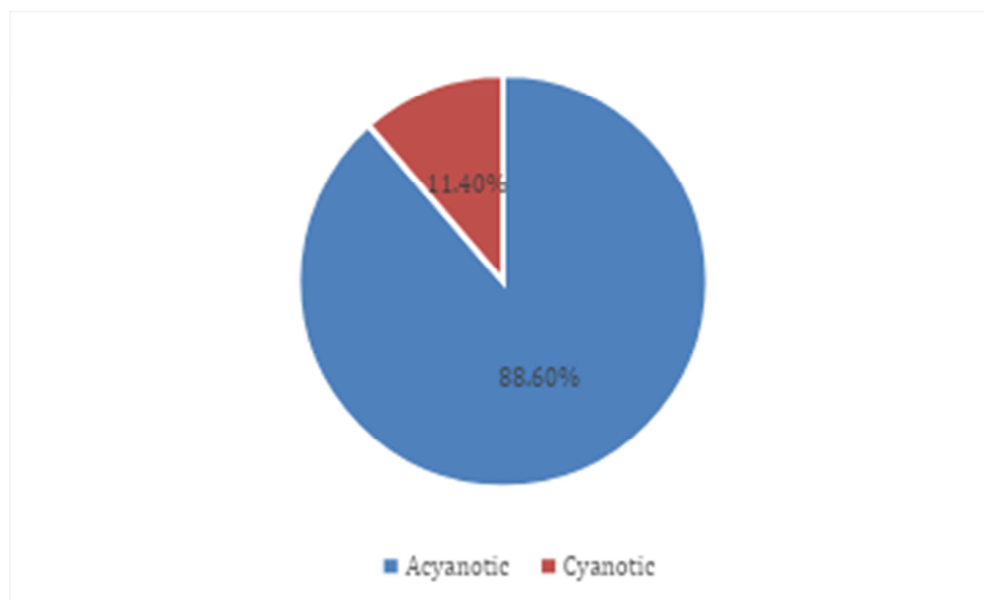


Figure 10 shows the distribution of CHD based on Cyanosis, 88.6% were acyanotic CHD, while only 12 children (11.4%) had cyanotic CHD.

Figure 10: Pie chart showing distribution of Congenital Heart Disease (CHD) Based on Cyanosis in Children Undergoing Corrective Surgery

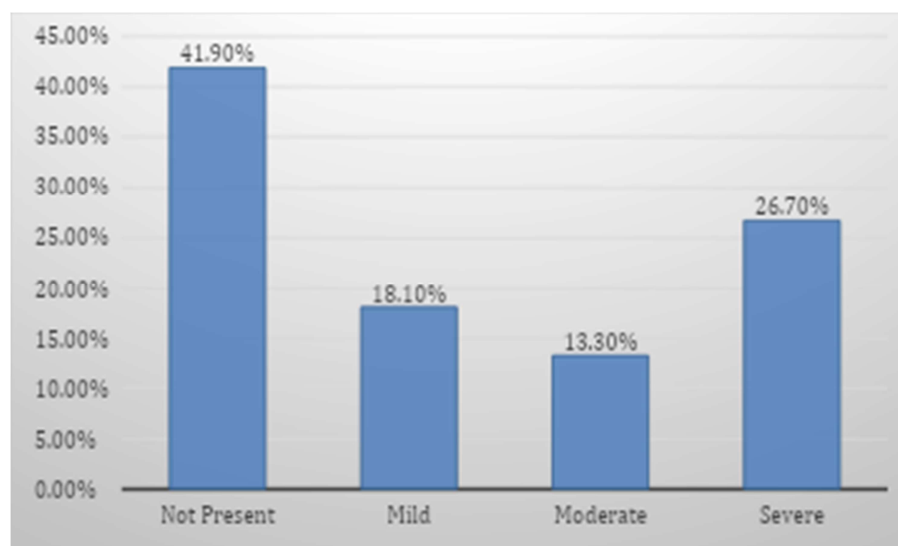


Based on the severity of PAH assessed by ECHO, 44 children (41.9%) had no PAH, while 19 children (18.1%) had mild PAH, 14 children (13.3%) had moderate PAH, and 28 children (26.7%) had severe PAH. (Table 5)(Figure 11)

Table 5: Distribution of CHD based on Severity of Pulmonary Arterial Hypertension (PAH)

| | | Frequency | Percent |
|-----------------|-------------|-----------|---------|
| Severity Of PAH | Not Present | 44 | 41.9 |
| | Mild | 19 | 18.1 |
| | Moderate | 14 | 13.3 |
| | Severe | 28 | 26.7 |
| | Total | 105 | 100.0 |

Figure 11: Bar chart showing Distribution of CHD based on Severity of Pulmonary Arterial Hypertension (PAH)

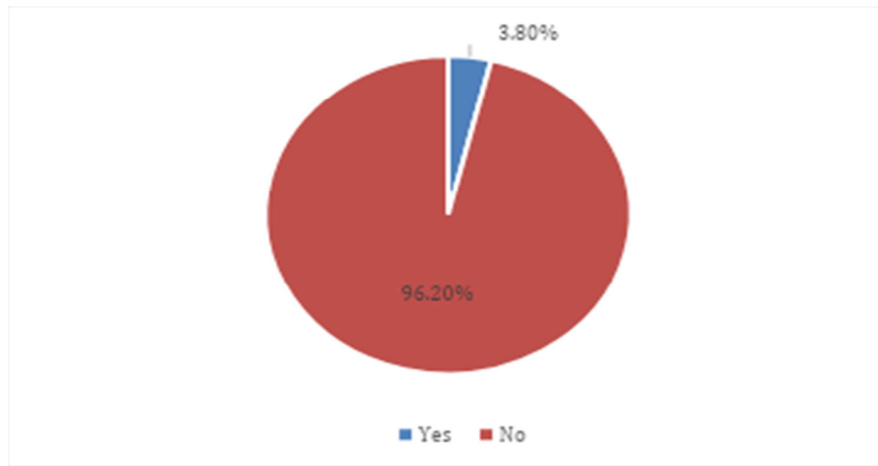


In our study, four children (3.8%) developed sepsis. Blood culture results showed *E. coli* sepsis in two children, while the other isolates were *Candida glabrata* and *Klebsiella pneumoniae*. (Table 6)(Figure 12)

Table 6: Incidence of Sepsis in Children Undergoing Corrective Surgery in immediate post-op period.

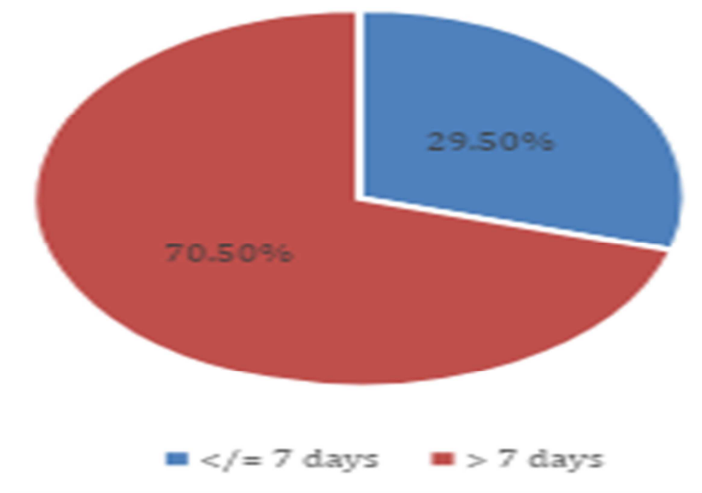
| | | Frequency | Percent |
|--------|-------|-----------|---------|
| Sepsis | Yes | 4 | 3.8 |
| | No | 101 | 96.2 |
| | Total | 105 | 100.0 |

Figure 12: Pie chart showing Incidence of Sepsis in Children Undergoing Corrective Surgery in immediate post-op period.



Of all the children undergoing cardiac surgery, 74 (70.5%) required hospitalization for more than seven days, while 31 (29.5%) were discharged within seven days. (Figure 13)

Figure 13: Pie chart depicting Duration of Hospitalization in Children Undergoing Corrective Surgery during the perioperative stay.

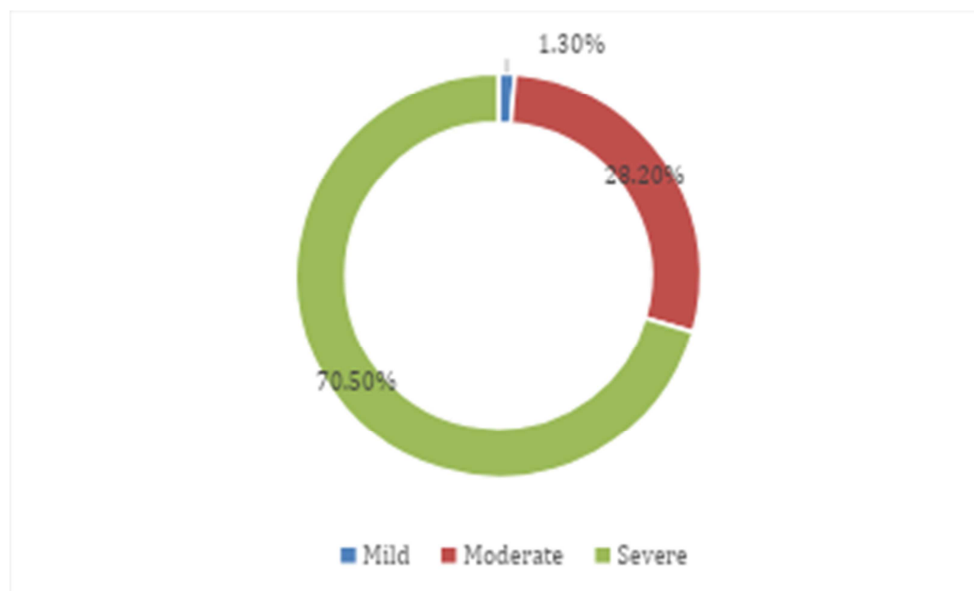


Among the 85 children with acyanotic CHD with a left-to-right shunt, the distribution based on shunt severity, as determined by ECHO, showed that 70.5% had a severe shunt, 24 children (28.2%) had a moderate shunt, and only one child (1.3%) had a mild shunt. (Table 7)(Figure 14)

Table 7: Distribution of Children with Left to right shunt lesions Undergoing Corrective Surgery based on severity of shunt.

| | | Frequency | Percent |
|-------------------|----------|-----------|---------|
| Severity of Shunt | Mild | 1 | 1.3 |
| | Moderate | 24 | 28.2 |
| | Severe | 60 | 70.5 |
| | Total | 85 | 100.0 |

Figure 14: Doughnut Pie chart showing Distribution of Children with Left to right shunt lesions Undergoing Corrective Surgery based on severity of shunt.



Furthermore, only two children (2.4%) had a residual lesion after corrective cardiac surgery among the 85 cases with left-to-right shunt lesions, including those with VSD, ASD, and PDA. The remaining 83 children (97.6%) had no residual lesions post-surgery. (Table 8)

Table 8: Percentage of Presence of Residual Lesion in Children with left to right shunt lesions after Corrective Surgery as determined by Post-op ECHO

| | | Frequency | Percent |
|-----------------------------|-------|-----------|---------|
| Presence of Residual Lesion | Yes | 2 | 2.4 |
| | No | 83 | 97.6 |
| | Total | 85 | 100.0 |

Based on the complexity of the lesion, children undergoing corrective surgery were classified into those with simple lesions, seen in 83 children (79.0%), and those with complex lesions, seen in 22 children (21.0%).(Figure 15)

Figure 15: Pie chart showing Distribution of study population based on Complexity of Lesion.

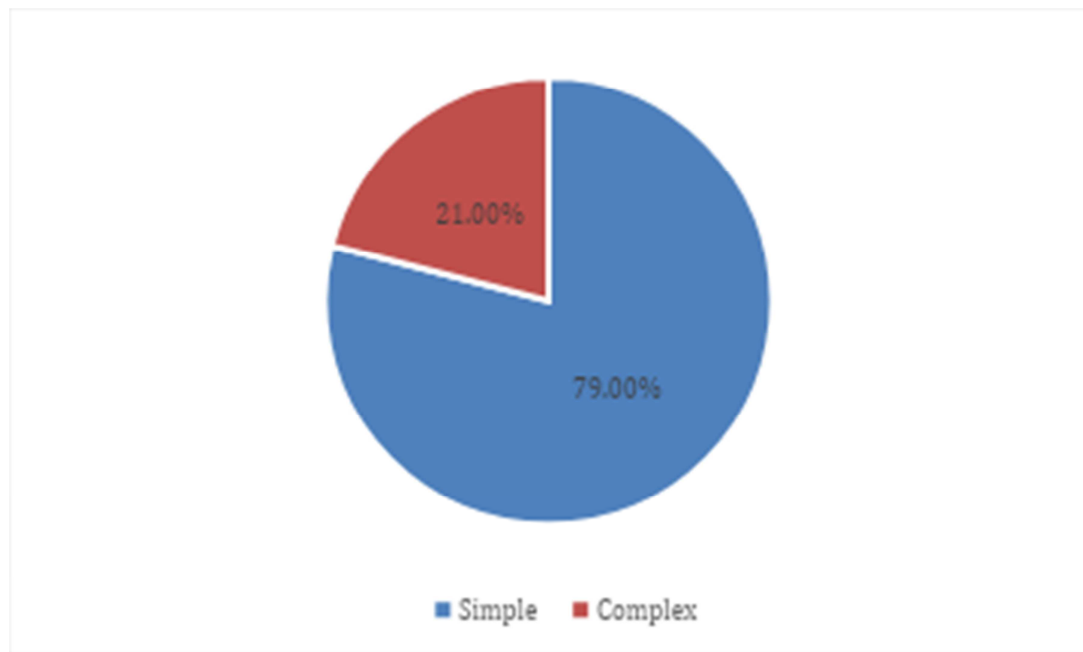


Table 9: Baseline Growth Parameters of Children Undergoing Corrective Surgery

| Baseline measurements | | Frequency | Percent |
|----------------------------|---------------------|-----------|---------|
| Weight z-score | <-3 (Severe) | 29 | 27.6% |
| | -2 to -3 (Moderate) | 59 | 56.2% |
| | >-2 (Normal) | 17 | 16.2% |
| Height/Length z-score | <-3 (Severe) | 21 | 20.0% |
| | -2 to -3 (Moderate) | 31 | 29.5% |
| | >-2 (Normal) | 53 | 50.5% |
| Head Circumference z-score | <-2 (Microcephaly) | 34 | 32.4% |
| | >-2 (Normal) | 71 | 67.6% |
| Weight/Height z-score | <-3 (Severe) | 17 | 16.2% |
| | -2 to -3 (Moderate) | 68 | 64.8% |
| | >-2 (Normal) | 20 | 19.0% |

In our study, analysing the Weight z-score, it was found that, at baseline, 29 children (27.6%) were severely underweight (z-score < -3), while 59 children (56.2%) were moderately underweight (z-score between -2 and -3). Only 17 children (16.2%) had a normal weight z-score. Further it was also found that 21 children (20.0%) were severely stunted, and 31 children (29.5%) were moderately stunted. More than half (50.5%) had a normal height/length z-score. Analysis of Head Circumference z-score revealed that 34 children (32.4%) had microcephaly and the majority (67.6%) had a normal head circumference. Severe wasting was present in 17 children (16.2%), moderate wasting was noted in 68 children (64.8%) and 20 children (19.0%) had a normal weight-for-height z-score. (Table 9)

Table 10: Growth Parameters at 3-Month Follow-Up After Corrective Surgery

| Follow up at 3 months | | Frequency | Percent |
|----------------------------|---------------------|-----------|---------|
| Weight z-score | <-3 (Severe) | 4 | 3.8% |
| | -2 to -3 (Moderate) | 45 | 42.9% |
| | >-2 (Normal) | 56 | 53.3% |
| Height/Length z-score | <-3 (Severe) | 3 | 2.9% |
| | -2 to -3 (Moderate) | 27 | 25.7% |
| | >-2 (Normal) | 75 | 71.4% |
| Head Circumference z-score | <-2 (Microcephaly) | 18 | 17.1% |
| | >-2 (Normal) | 87 | 82.9% |
| Weight/Height z-score | <-3 (Severe) | 0 | 0.0% |
| | -2 to -3 (Moderate) | 31 | 29.5% |
| | >-2 (Normal) | 74 | 70.5% |

Analysis of growth parameters at 3 months follow-up showed that 25 children improved significantly and 4 children (3.8%) remained severely underweight (z-score < -3), compared to 29 (27.6%) at baseline. Moderate underweight status was observed in 45 children (42.9%), while 56 children (53.3%) had achieved normal weight for age. Severe stunting was present in only 3 children (2.9%) at 3 months, compared to 21 children (20.0%) at baseline. Moderate stunting was reduced to 27 children (25.7%), and 75 children (71.4%) achieved normal height for age. Microcephaly was seen in 18 children (17.1%) at follow-up after 3 months of corrective surgery and 87 children (82.9%) had normal head circumference. Severe wasting was eliminated, with no cases recorded at 3 months follow up. Moderate wasting was observed in 31 children (29.5%), while 74 children (70.5%) had a normal weight-for-height z-score. (Table 10)

Table 11: Comparison of Growth Parameters at Baseline and 3-Month Follow-Up

| | Baseline | | Follow up at 3 months | | P Value |
|----------------------------|----------|------|-----------------------|------|---------|
| | Mean | SD | Mean | SD | |
| Weight z-score | -3.16 | 1.34 | -2.11 | 0.89 | 0.001 |
| Height/Length z-score | -1.97 | 1.25 | -1.71 | 1.04 | 0.001 |
| Head Circumference z-score | -1.55 | 1.20 | -1.16 | 0.98 | 0.001 |
| Weight/Height z-score | -2.80 | 1.40 | -1.56 | 0.89 | 0.001 |

Comparison of Growth Parameters at Baseline and 3-Month Follow-Up shown by Figure 16 and Table 11, which shows that the mean weight z-score of the study population improved from -3.16 ± 1.34 at baseline to -2.11 ± 0.89 at 3 months post-surgery which was statistically significant ($P = 0.001$). Similarly, the mean height/length z-score also increased from -1.97 ± 1.25 at baseline to -1.71 ± 1.04 at 3 months follow up ($P = 0.001$). Further, the mean head circumference z-score and weight/height z-score also improved from -1.55 ± 1.20 at baseline to -1.16 ± 0.98 at 3 months and -2.80 ± 1.40 at baseline to -1.56 ± 0.89 at 3 months respectively ($P = 0.001$).

Figure 16 : Grouped Bar chart showing Comparison of Growth Parameters at Baseline and 3-Month Follow-Up

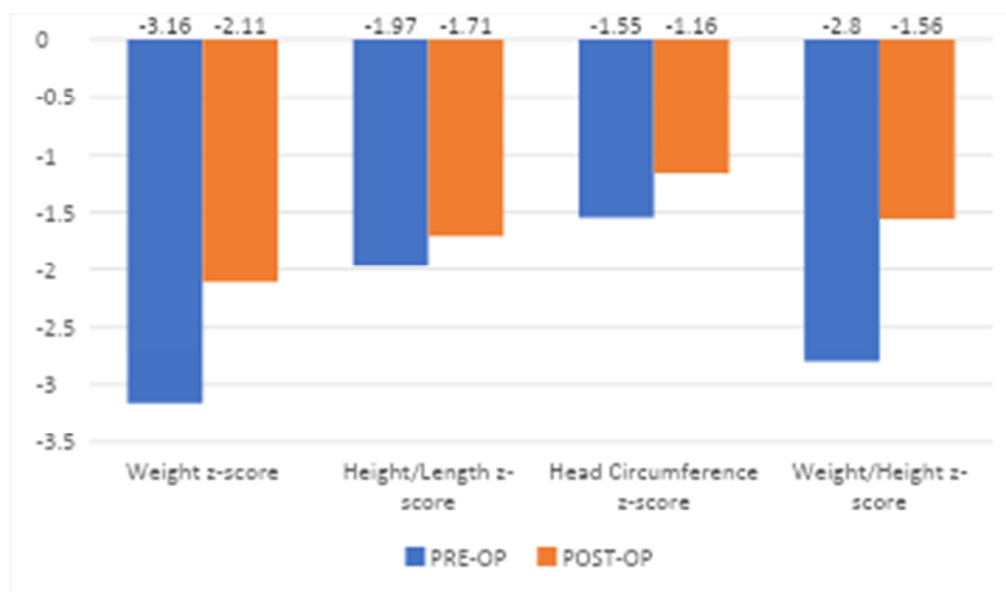


Table 12: Improvement in Wasting Status at 3-Month Follow-Up After Corrective Surgery

| | | | Follow up at 3 months -Wasting | | Total |
|--|---------|---|--------------------------------|--------|--------|
| | | | Present | Absent | |
| Baseline percentage of wasting noted pre surgery | Present | n | 31 | 54 | 85 |
| | | % | 100.0% | 73.0% | 81.0% |
| | Absent | n | 0 | 20 | 20 |
| | | % | 0.0% | 27.0% | 19.0% |
| Total | | n | 31 | 74 | 105 |
| | | % | 100.0% | 100.0% | 100.0% |

Chi-Square: 10.35, P value: 0.001, Statistically Significant

At baseline, 85 children (81.0%) were classified as wasted, with 31 children (100.0%) remaining wasted at follow-up, while 54 children (73.0%) showed recovery from wasting. Among the 20 children who were not wasted at baseline, all (100.0%) remained non-wasted at follow-up.(Table 12)

**Table 13: Improvement in Underweight Status at 3-Month Follow-Up After
Corrective Surgery**

| | | | Follow up at 3 months - Underweight | | Total |
|--|---------|---|-------------------------------------|--------|--------|
| | | | Present | Absent | |
| Baseline percentage of underweight noted pre surgery | Present | n | 47 | 41 | 88 |
| | | % | 100.0% | 70.7% | 83.8% |
| | Absent | n | 0 | 17 | 17 |
| | | % | 0.0% | 29.3% | 16.2% |
| Total | | n | 47 | 58 | 105 |
| | | % | 100.0% | 100.0% | 100.0% |

Chi-Square: 16.43, P value: 0.001, Statistically Significant

At baseline, 88 children (83.8%) were classified as underweight. Among them, 47 children (100.0%) remained underweight at follow-up, while 41 children (70.7%) showed recovery and achieved normal weight for age. Of the 17 children who were not underweight at baseline, all (100.0%) maintained their normal weight status at follow-up. (Table 13)

**Table 14: Improvement in stunting Status at 3-Month Follow-Up After
Corrective Surgery**

| | | | Follow up at 3 months - Stunting | | Total |
|---|---------|---|----------------------------------|--------|--------|
| | | | Present | Absent | |
| Baseline percentage of stunting noted pre surgery | Present | n | 31 | 22 | 53 |
| | | % | 100.0% | 29.7% | 50.5% |
| | Absent | n | 0 | 52 | 52 |
| | | % | 0.0% | 70.3% | 49.5% |
| Total | | n | 31 | 74 | 105 |
| | | % | 100.0% | 100.0% | 100.0% |

Chi-Square: 43.15, P value: 0.001, Statistically Significant

At baseline, 53 children (50.5%) were classified as stunted. Among them, 31 children (100.0%) remained stunted at the 3-month follow-up, while 22 children (29.7%) showed recovery from stunting. Of the 52 children who were not stunted at baseline, all (100.0%) remained non-stunted at follow-up. (Table 14)

Table 15: Improvement in Microcephaly Status at 3-Month Follow-Up After Corrective Surgery

| | | | Follow up at 3 months - Microcephaly | | Total |
|---|---------|---|--------------------------------------|--------|--------|
| | | | Present | Absent | |
| Baseline percentage of microcephaly noted pre surgery | Present | n | 18 | 16 | 34 |
| | | % | 100.0% | 18.4% | 32.4% |
| | Absent | n | 0 | 71 | 71 |
| | | % | 0.0% | 81.6% | 67.6% |
| Total | | n | 18 | 87 | 105 |
| | | % | 100.0% | 100.0% | 100.0% |

Chi-Square: 45.36, P value: 0.001, Statistically Significant

At baseline, 34 children (32.4%) were diagnosed with microcephaly. Among them, 18 children (100.0%) remained microcephalic at follow-up, while 16 children (18.4%) showed improvement in head circumference. Of the 71 children without microcephaly at baseline, all (100.0%) maintained normal head growth at follow-up. (Table 15)

Table 16: Comparison of Type of Malnutrition from baseline to 3-Month Follow-Up After Corrective Surgery

| | Type of Malnutrition | | | |
|---|----------------------|-------|-----------------------|-------|
| | Baseline | | Follow up at 3 months | |
| | n | % | n | % |
| MAM | 38 | 36.2 | 26 | 24.8 |
| Moderate Stunting | | | 1 | 1.0 |
| Moderate Underweight | 4 | 3.8 | 5 | 4.8 |
| Moderate Underweight With Stunting | | | 12 | 11.4 |
| Moderate Underweight With Moderate Stunting | 1 | 1.0 | 1 | 1.0 |
| Moderate Stunting | 1 | 1.0 | 1 | 1.0 |
| Moderate Wasting | 3 | 2.9 | 2 | 1.9 |
| Normal | 12 | 11.4 | 51 | 48.6 |
| SAM | 44 | 41.9 | 3 | 2.9 |
| Severe Stunting | 1 | 1.0 | 1 | 1.0 |
| Severe Underweight With Severe Stunting | 1 | 1.0 | 2 | 1.9 |
| Total | 105 | 100.0 | 105 | 100.0 |

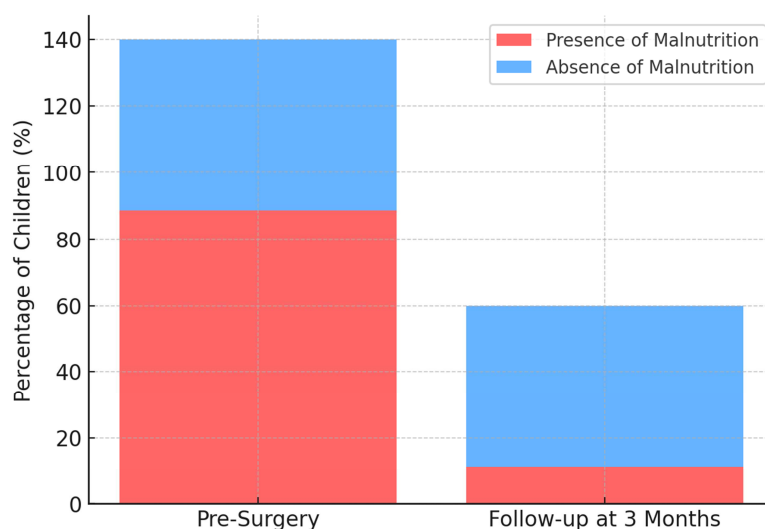
At baseline, 44 children (41.9%) were diagnosed with severe acute malnutrition (SAM), which significantly reduced to just 3 children (2.9%) at the 3-month follow-up. Similarly, the number of children with moderate acute malnutrition (MAM) decreased from 38 (36.2%) at baseline to 26 (24.8%) at follow-up. The percentage of children with normal nutritional status increased significantly from 11.4% at baseline to 48.6% at follow-up. Cases of moderate underweight increased slightly from 4 (3.8%) to 5 (4.8%), and there were 12 cases (11.4%) of moderate underweight with stunting identified at follow-up. The number of children with moderate wasting decreased from 3 (2.9%) to 2 (1.9%), indicating better nutritional balance post-surgery. Severe underweight with severe stunting increased slightly from 1 (1.0%) to 2 (1.9%).(Table 16).

Table 17: Transition in Type of Malnutrition from Baseline to 3-Month Follow-Up After Corrective Surgery

| | Follow up at 3 months -Type Of Malnutrition | | | | | | | | | | | Total | |
|--------------------------------------|---|-------------------|----------------------|------------------------------------|---|------------------|-------------------|--------|-----|---|-----------------|-------|----|
| | MAM | Moderate Stunting | Moderate Underweight | Moderate Underweight With Stunting | Moderate Underweight With Moderate Stunting | Moderate Wasting | Moderate Stunting | Normal | SAM | Severe Underweight With Severe Stunting | Severe Stunting | | |
| Baseline- Type of Malnutrition | MAM | | | 4 | 6 | | 1 | | 27 | | | | 38 |
| | Moderate Underweight | | | | | | | | 4 | | | | 4 |
| | Moderate Underweight With Moderate Stunting | | 1 | | | | | | | | | | 1 |
| | Moderate Stunting | | | | | | | | 1 | | | | 1 |
| | Moderate Wasting | | | | | | 1 | | 2 | | | | 3 |
| | Normal | | | | | | | | 12 | | | | 12 |
| | SAM | 26 | | 1 | 6 | 1 | | | 5 | 3 | 1 | | 44 |
| | Severe | | | | | | | | | | 1 | | 1 |

| | | | | | | | | | | | | | |
|-------|----------------------------------|---|---|----|---|---|---|----|---|---|---|-----|---|
| | Underweight With Severe Stunting | | | | | | | | | | | | |
| | Severe Stunting | | | | | | | | | | | 1 | 1 |
| Total | 26 | 1 | 5 | 12 | 1 | 2 | 1 | 51 | 3 | 2 | 1 | 105 | |

At baseline, 38 children were classified with moderate acute malnutrition (MAM). By the 3-month follow-up, 27 of these children (71.1%) had transitioned to normal nutritional status, 6 shifted to moderate underweight with stunting, and 4 remained in the MAM category. Among the 4 children initially categorized as having moderate underweight, all (100%) remained in the same category at follow-up. One child initially diagnosed with moderate underweight with moderate stunting remained in the same category at follow-up. Similarly, the child diagnosed with moderate stunting at baseline showed no change at follow-up. Of the 3 children with moderate wasting at baseline, 2 transitioned to normal nutritional status, while 1 remained in the moderate wasting category. Among the 44 children initially diagnosed with severe acute malnutrition (SAM) at baseline, 26 (59.1%) moved to MAM status, 6 transitioned to moderate underweight with stunting, 5 improved to normal nutritional status, and 3 remained in the SAM category. One child initially diagnosed with severe underweight with severe stunting remained in the same category, while another transitioned to severe underweight with severe stunting. Overall, the number of children with normal nutritional status increased from 12 (11.4%) at baseline to 51 (48.6%) at follow-up.(Table 17)

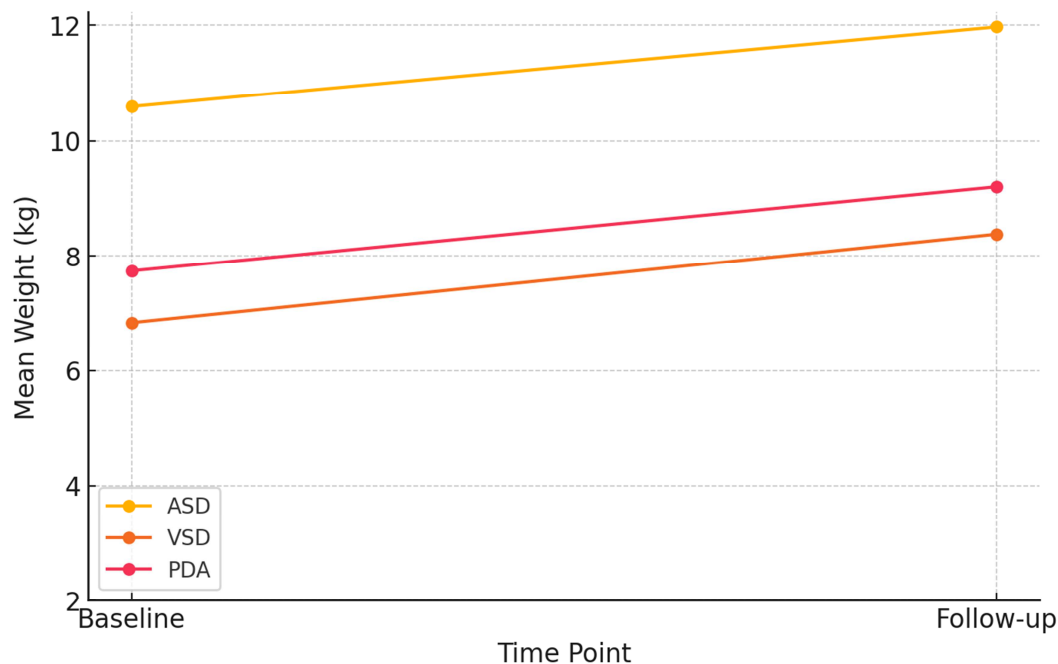
Figure 17: Stacked Bar graph showing Overall Improvement in Nutritional**Status at 3-Month Follow-Up After Corrective Surgery**

At baseline, only 12 children (11.4%) were classified as having a normal nutritional status, while 93 children (88.6%) were categorized as having some form of malnutrition. At the 3-month follow-up, the percentage of children with normal nutritional status increased significantly to 51 (48.6%), while the number of children with malnutrition status decreased to 54 (51.4%).(Figure 17)

Table 18: Comparison of mean weight gain among children with different types of Acyanotic CHD with Left to right shunt.

| | Age (years) | | Baseline-Weight(Kgs) | | Follow up at 3 months Weight(Kgs) | | Weight Gain (Kgs) | |
|-----|-------------|------|----------------------|------|-----------------------------------|------|-------------------|------|
| | Mean | SD | Mean | SD | Mean | SD | Mean | SD |
| ASD | 3.20 | 1.44 | 10.59 | 3.37 | 11.97 | 3.05 | 1.37 | 0.55 |
| VSD | 1.61 | 1.39 | 6.84 | 2.62 | 8.38 | 2.45 | 1.53 | 0.47 |
| PDA | 1.86 | 1.63 | 7.74 | 3.16 | 9.20 | 2.97 | 1.45 | 0.50 |

Figure 18-Line graph showing Mean weight gain of children with CHD with Left to right shunt.

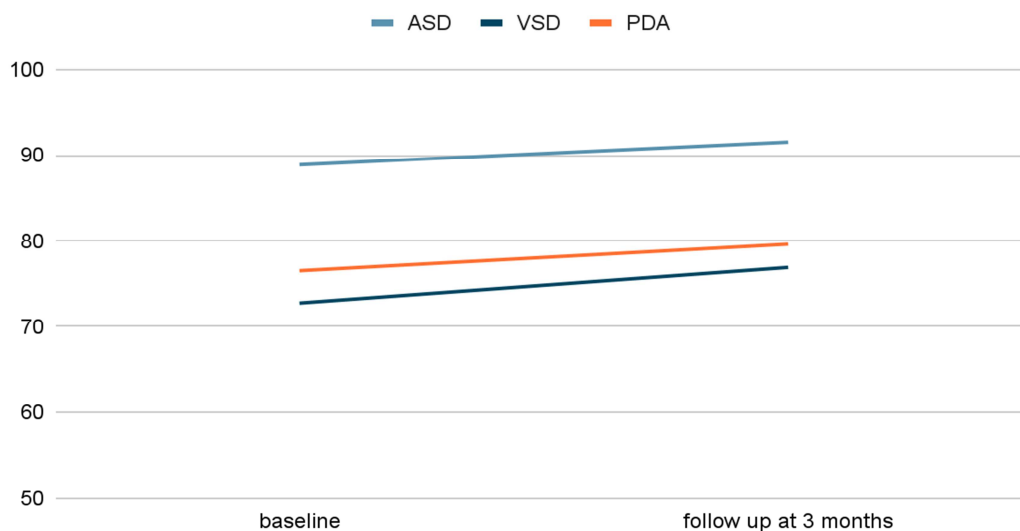


The mean weight at baseline of children with ASD was 10.59 ± 3.37 kg, which increased to 11.97 ± 3.05 kg at follow-up, with a significant mean weight gain of 1.37 ± 0.55 kg ($P = 0.001$). Further, among children with VSD, the mean weight at baseline was 6.84 ± 2.62 kg, which increased to 8.38 ± 2.45 kg at follow-up, with a mean weight gain of 1.53 ± 0.47 kg ($P = 0.001$). Similarly, the mean weight of children with PDA at baseline was 7.74 ± 3.16 kg, which increased to 9.2 ± 2.97 kg at follow-up, with a mean weight gain of 1.45 ± 0.50 kg ($P = 0.001$). It was found that children with VSD has maximum mean weight again compared to the other groups.(Table 18)(Figure 18)

Table 19: Comparison of mean difference in height/length among children with different types of Acyanotic CHD with Left to right shunt.

| | Baseline- Height/Length (Cms) | | Follow up at 3 months - Height/Length (Cms) | | Height/Length difference (Cms) | |
|-----|----------------------------------|-------|--|-------|-----------------------------------|------|
| | Mean | SD | Mean | SD | Mean | SD |
| ASD | 88.87 | 13.57 | 91.54 | 12.87 | 2.67 | 1.28 |
| VSD | 72.76 | 13.37 | 76.93 | 12.24 | 4.16 | 2.12 |
| PDA | 76.54 | 13.27 | 79.65 | 12.40 | 3.11 | 1.50 |

Figure 19- Line graph showing Mean increase in height in children with CHD with Left to right shunt



The mean height at baseline of children with ASD, VSD and PDA was 88.87 ± 13.57 cm, 72.76 ± 13.37 cm and 76.54 ± 13.27 respectively. At 3 months follow up, the mean height increased to 91.54 ± 12.87 cm, 76.93 ± 12.24 cm and 79.65 ± 12.40 cm respectively which was statistically significant ($P = 0.001$). Maximum increase in height was also seen in children with VSD compared to other groups (Table 19)(Figure 19)

Table 20: Factors Influencing Pre-Operative Weight z-Score in Children with Congenital Heart Disease (CHD)

| | | Unstandardized Coefficients | | P value |
|------------------------------|-------------------------------|-----------------------------|------------|---------|
| | | B | Std. Error | |
| Pre-OP Weight z- score | (Constant) | -4.93 | 2.39 | 0.04 |
| | Age Group | 0.56 | 0.15 | 0.001 |
| | Birth Weight(Kgs) | 0.84 | 0.23 | 0.001 |
| | NICU Admission | -0.23 | 0.29 | 0.42 |
| | Severity Of Pah | 0.12 | 0.11 | 0.25 |
| | Sepsis (+/-) | 0.16 | 0.63 | 0.79 |
| | No Of Days Of Hospitalisation | -0.18 | 0.26 | 0.49 |
| | Presence Of Residual Lesion | -0.73 | 0.88 | 0.40 |

Linear Regression Test Applied

The regression model showed that age group (B = 0.56, P = 0.001) and birth weight (kgs) (B = 0.84, P = 0.001) were significant positive predictors of pre-operative weight z-score. This indicates that older age and higher birth weight were associated with better preoperative weight status. NICU admission (B = -0.23, P = 0.42), severity of PAH (B = 0.12, P = 0.25), sepsis (B = 0.16, P = 0.79), number of hospital days (B = -0.18, P = 0.49), and presence of residual lesion (B = -0.73, P = 0.40) were not statistically significant predictors of pre-operative weight z-score, indicating that these factors did not significantly influence the child's weight status before surgery. The constant value of -4.93 (P = 0.04) suggests that the baseline weight z-score was low even after adjusting for the influencing factors. The findings indicate that age and

birth weight have a stronger impact on preoperative weight status, while postnatal complications such as NICU admission, sepsis, and PAH severity do not significantly affect weight before surgery.(Table 20)

Table 21: Factors Influencing Pre-Operative Height/Length z-Score in Children with Congenital Heart Disease (CHD)

| | | Unstandardized Coefficients | | P value |
|-------------------------------------|-------------------------------|-----------------------------|------------|---------|
| | | B | Std. Error | |
| Pre-OP Height/Length z- score | (Constant) | -1.59 | 2.30 | 0.49 |
| | Age Group | 0.13 | 0.14 | 0.37 |
| | Birth Weight(Kgs) | 0.92 | 0.22 | 0.001 |
| | NICU Admission | -0.42 | 0.28 | 0.13 |
| | Severity Of Pah | 0.07 | 0.10 | 0.46 |
| | Sepsis(+/-) | -0.20 | 0.61 | 0.74 |
| | No Of Days Of Hospitalisation | -0.38 | 0.25 | 0.14 |
| | Presence Of Residual Lesion | -0.72 | 0.85 | 0.39 |

Linear Regression Test Applied

Birth weight (B = 0.92, P = 0.001) was the only significant predictor of pre-operative height/length z-score, indicating that higher birth weight was associated with better preoperative height/length status. Age group (B = 0.13, P = 0.37), NICU admission (B = -0.42, P = 0.13), severity of PAH (B = 0.07, P = 0.46), sepsis (B = -0.20, P = 0.74), number of hospital days (B = -0.38, P = 0.14), and presence of residual lesion (B = -0.72, P = 0.39) were not statistically significant predictors of pre-operative height/length z-score, indicating that these factors did not have a strong influence on

the child's height status before surgery. The constant value of -1.59 (P = 0.49) was not statistically significant, indicating that other unexplored factors may contribute to variations in height status before surgery. The findings suggest that birth weight is a key determinant of pre-operative height/length status, while postnatal complications and other medical factors, such as PAH severity and sepsis, have minimal impact on height status before surgery.(Table 21)

Table 22: Factors Influencing Pre-Operative Weight/Height z-Score in Children with Congenital Heart Disease (CHD)

| | | Unstandardized Coefficients | | P value |
|------------------------------|-------------------------------|-----------------------------|------------|---------|
| | | B | Std. Error | |
| Pre-OP Weight/Height z-score | (Constant) | -4.80 | 2.63 | 0.07 |
| | Age Group | 0.53 | 0.17 | 0.001 |
| | Birth Weight (Kgs) | 0.33 | 0.25 | 0.19 |
| | Nicu Admission | 0.06 | 0.32 | 0.84 |
| | Severity Of Pah | 0.14 | 0.12 | 0.22 |
| | Sepsis (+/-) | 0.22 | 0.70 | 0.75 |
| | No Of Days Of Hospitalisation | 0.06 | 0.29 | 0.83 |
| | Presence Of Residual Lesion | -0.49 | 0.97 | 0.60 |

Linear Regression Test Applied

Age group (B = 0.53, P = 0.001) was the only significant predictor of pre-operative weight/height z-score(wasting), indicating that older age was associated with a better weight/height status before surgery. Birth weight (B = 0.33, P = 0.19), NICU admission (B = 0.06, P = 0.84), severity of PAH (B = 0.14, P = 0.22), sepsis (B =

0.22, $P = 0.75$), number of hospital days ($B = 0.06$, $P = 0.83$), and presence of residual lesion ($B = -0.49$, $P = 0.60$) were not statistically significant predictors of pre-operative weight/height z-score. The constant value of -4.80 ($P = 0.07$) approached statistical significance, suggesting that baseline weight/height status was generally low across the study population, but the difference was not large enough to reach statistical significance. The findings suggest that age is the most influential factor in determining pre-operative weight/height status, while other clinical factors such as birth weight, PAH severity, and NICU admission have minimal impact on weight-for-height status before surgery.(Table 22)

DISCUSSION

Children with congenital heart disease (CHD) often experience growth failure due to the increased metabolic demands, inadequate caloric intake, and compromised nutrient absorption associated with their underlying cardiac defects. Growth impairment in these children is influenced by factors such as birth weight, gestational age, presence of pulmonary hypertension, and the complexity of the heart defect. Corrective surgery is expected to improve hemodynamic stability and enhance nutrient utilization, thereby facilitating catch-up growth. This study aims to evaluate the early growth trends of children under 5 years with CHD following corrective surgery and identify the key factors affecting postoperative growth.

The largest proportion of children (34.3%) who underwent surgery were under 1 year of age, followed closely by those aged between 1 to 3 years (33.3%) and 3 to 5 years (32.4%). This relatively balanced distribution across the three age groups indicates that corrective surgeries are performed consistently at various early childhood stages and is also dependent on the age at presentation of individual congenital heart disease. Vaidyanathan et al. reported that 62.2% of the total children who underwent cardiac surgery were infants (≤ 1 year), 9.5% being neonates.⁴ Similarly, Hapuoja et al. and He et al. reported that the percentage of children undergoing their cardiac surgery within their first year of life to be 95% and 32.7% respectively.^{10,57} The higher percentage of surgeries in infants under 1 year suggests that early diagnosis and intervention for CHD are common in younger children, which may contribute to better post-operative outcomes and improved growth trajectories.

The mean age at correction in our study was found to be 2.31 ± 1.09 years. Vaidyanathan et al. reported a similar result that the mean age at corrective

intervention was 15.2 ± 16.2 months and the study also mentioned that the age at corrective intervention significantly influenced the weight and weight/height z-scores, with older children showing poorer growth parameters at presentation ($P < 0.001$).⁴ Okoromah et al. reported that the mean age of children with CHD was 40.0 ± 44.96 months (approximately 3.3 years). The age range was 3 to 192 months (3 months to 16 years), and 64.3% of children were aged under 5 years.³⁰ Arodiwe et al. reported that the mean age of children with CHD was 4.66 ± 5.02 years (range: 1 month to 16 years). Older age at presentation was significantly associated with worse nutritional status ($P = 0.01$).⁴⁸

In our study, among the total 105 children, 62 (59.0%) were female, and 43 (41.0%) were male with male to female ratio of 1.0:1.4. This indicates a higher proportion of females undergoing corrective surgery compared to males in our study. Other studies have shown a varied male to female ratio 1:1.04, 1.2:1, 1.6:1.1 etc.^{4,30,48}. Further, Murni et al. reported that among the 1,149 children with CHD, 530 (46.1%) were male and 619 (53.9%) were female.⁵⁸ Gender disparity may be due to various factors like sex ratio of the region, availability of resources, socioeconomic status etc.

Based on the modified Kuppuswamy scale, with occupation, paternal education and income as key indicators, our study showed that CHD cases requiring corrective surgery are more prevalent among children from lower middle (59%) and lower socioeconomic backgrounds (22.8%) which was similar to the outcomes of other studies.^{4,30,48} Vaidyanathan et al. also identified that lower socioeconomic status was associated with poorer nutritional outcomes in children with CHD which was in accordance with our study.⁴ Factors such as limited access to cardiac surgical facilities, poor nutritional status, and delayed diagnosis in lower-income families may contribute to the higher burden of CHD in these groups and also have a significant

impact on the growth of children with CHD. Only 9.5% of mothers of the study population, reported a history of antenatal complications, including gestational diabetes mellitus (GDM) (1.9%), hypothyroidism (1.9%), pregnancy-induced hypertension (PIH) (1.9%), anemia (1.0%), chronic hypertension (1.0%), hypotension (1.0%), and pre-eclampsia (1.0%). The low percentage of antenatal complications suggests that while maternal health issues may play a role in the development of CHD, the majority of CHD cases requiring corrective surgery are not strongly linked to maternal antenatal history.

Our study shows that the majority(55.2%) of children requiring corrective cardiac surgery had a normal birth weight, which suggests that low birth weight alone may not be a significant risk factor for CHD requiring early surgical intervention. But our study also shows that low birth weight (kgs) ($B = 0.84$, $P = 0.001$) was a significant positive predictor of pre-operative weight z-score and height/length z score. This finding is consistent with other studies like Vaidyanathan et al. where low birth weight (<2.5 kg) was a significant predictor of poor nutritional status ($OR = 20.24$, $P < 0.001$). and Hapuoja et al., where Lower birth weight was associated with lower weight and head circumference at 10 years ($P < 0.001$).^{4,10}. He et al. also reported that the majority of CHD patients (85.7%) had a normal birth weight (NBW) between 2500–4000 g. and, LBW and VLBW were found to be significant risk factors for failure to thrive (FTT) ($P < 0.001$).⁵⁷ Further, Murni et al. reported that low birth weight (<2,500 g) was independently associated with increased risk of underweight ($OR = 2.65$, 95% CI: 1.95–2.61; $P < 0.001$) and stunting ($OR = 2.91$, 95% CI: 2.17–3.62; $P < 0.001$) as determined in our study.⁵⁸

Majority of the children from our study population (96.2%) were born at term, while only 4 children (3.8%) were born preterm. This indicates that most CHD cases

requiring corrective surgery occur in children born at term, suggesting that prematurity alone may not be a significant risk factor for CHD requiring early surgical intervention. Small for gestational age (SGA) was associated with a higher risk of lower height and weight/height z-scores (OR = 6.51, P < 0.001).⁴ However, we could not assess the exact period of gestation at birth due to insufficient records. He et al. reported that 88.7% of the children were born at term (37–42 weeks), while 9.4% were preterm, and 0.1% were extremely preterm (<28 weeks). Post-term births accounted for 1.8%.⁵⁷ which was similar to our study.

Out of the 105 children, 57 (54.3%) were delivered through normal vaginal delivery (NVD), while 48 (45.7%) were delivered via lower segment cesarean section (LSCS). The nearly equal distribution of delivery methods suggests that the mode of delivery may not have a significant influence on the occurrence of CHD requiring corrective surgery. Out of the 48 cesarean deliveries, the most common indication was a history of previous LSCS (27.1%), followed by fetal distress (14.6%), non-progressive labor (NPL) (12.5%), and oligohydramnios (12.5%). The diversity of indications highlights the complex prenatal and intrapartum factors that may influence the need for surgical delivery in cases of fetus with CHD.

In our study, atrial septal defect (ASD) was the most common congenital heart defect (CHD) (29.5%), followed closely by ventricular septal defect (VSD) (28.6%) and patent ductus arteriosus (PDA) (21.0%). The majority of cases (88.6%) were acyanotic CHD, while only 11.4% were cyanotic CHD. This predominance of acyanotic CHD aligns with its higher overall incidence and the greater availability of corrective surgical options.

Comparing our findings with other studies, Vaidyanathan et al. reported that left-to-right shunts were the most prevalent CHD type (64.3%), with PDA (24.8%)

and VSD (23.5%) being the most common defects.⁴ Okoromah et al. found VSD (35.6%) to be the most frequent defect, followed by PDA (13.7%) and ASD (8.2%), while cyanotic CHD accounted for 15.1% of cases, primarily tetralogy of Fallot (TOF).³⁰ Murni et al. reported a similar trend, with 81.4% of cases being acyanotic CHD, predominantly VSD (27.2%), ASD (20.3%), and PDA (20.8%). Among cyanotic CHD, TOF (4.6%) and pulmonary atresia (5.3%) were the most common.⁵⁸ Arodiwe et al. also observed a higher prevalence of acyanotic CHD (72%), with VSD (24%), ASD (12%), and PDA (6%) being the most frequent, while TOF (16%) was the most common cyanotic CHD.⁴⁸ Overall, across studies, acyanotic CHD consistently accounted for the majority of cases, with VSD, ASD, and PDA being the most common defects.

In our study, 41.9% of children had no pulmonary arterial hypertension (PAH), while 18.1% had mild PAH, 13.3% had moderate PAH, and 26.7% had severe PAH. The higher proportion of children without PAH may be due to the predominance of children with ASD and PDA in our study population. However, the significant proportion of severe PAH cases highlights the considerable hemodynamic burden associated with certain congenital heart defects (CHDs), emphasizing the need for early diagnosis and surgical intervention.

Comparing our findings with other studies, Vaidyanathan et al. reported PAH in 40.8% of cases, with significant poor growth outcomes ($P < 0.05$).⁴ Arodiwe et al. found that 44% of acyanotic CHD cases had PAH, which was associated with worse nutritional status ($P = 0.01$).⁴⁸ Murni et al. further demonstrated a strong association between PAH and underweight (OR = 2.48, $P < 0.001$), stunting (OR = 1.55, $P = 0.001$), and wasting (OR = 1.53, $P < 0.001$).⁵⁸ Unlike these studies, we could not

establish PAH severity as an independent predictor of poor outcomes or its direct association with malnutrition indicators such as wasting, stunting, or underweight.

Further in our study 4 children (3.8%) developed sepsis, while the majority (96.2%) did not develop sepsis. The low incidence of sepsis suggests that effective perioperative care and infection control measures were largely successful in preventing postoperative infections. Blood culture results showed *E. coli* sepsis in two children, while the other isolates were *Candida glabrata* and *Klebsiella pneumoniae*.

Out of all the children who underwent cardiac surgery, 74 children (70.5%) required hospitalization for more than 7 days, while 31 children (29.5%) were discharged within 7 days. The high percentage of prolonged hospital stays reflects the complexity of surgical management and post-operative care associated with CHD. Longer hospital stays may be indicative of surgical complications, slower recovery, or the need for additional medical support post-surgery. Hapuoja et al. reported that the median length of hospital stay after the first CPB surgery was 23 days (IQR: 14–33 days) for biventricular CHD and 30.5 days (IQR: 17.5–67 days) for univentricular CHD. Longer hospital stay was associated with lower height and weight at 10 years ($P = 0.007$, $P = 0.021$).¹⁰ He et al. reported that the median length of hospital stay was 23 days (IQR: 14–33 days) for biventricular CHD and 30.5 days (IQR: 17.5–67 days) for univentricular CHD.⁵⁷

Among the 85 children with acyanotic CHD with left to right shunt, 70.5% had severe shunt, 24 children (28.2%) had moderate shunt, and only 1 child (1.3%) had mild shunt. The high proportion of severe shunt cases indicates that most children requiring corrective surgery had significant intracardiac shunting, leading to considerable hemodynamic compromise. Out of these patients, 83 children (97.6%) had no residual lesion post-surgery, while 2 children (2.4%) had residual lesions. The

low incidence of residual lesions reflects the effectiveness and accuracy of the corrective surgical procedures. However, the presence of even a small percentage of residual lesions highlights the need for ongoing post-operative monitoring and potential follow-up interventions to ensure complete recovery and optimal cardiac function.

Out of the study population 83 children (79.0%) had simple lesions, while 22 children (21.0%) had complex lesions. The higher proportion of simple lesions indicates that the majority of CHD cases requiring corrective surgery involved relatively straightforward anatomical defects. Hapuoja et al. reported that 21% of cases had univentricular CHD, which was not significantly associated with growth parameters at 10 years ($P > 0.05$).¹⁰ He et al. reported that complex CHD cases accounted for 15.6% of the cohort and were associated with higher failure to thrive rates (54.2%).⁵⁷ These findings were consistent with our study.

In our study, underweight(weight z score < -2) was prevalent before surgery, with 27.6% of children being severely underweight (z-score < -3) and 56.2% being moderately underweight (z-score between -2 and -3). Only 16.2% had a normal weight z-score. However, at 3 months post-surgery, there was a significant improvement in nutritional status—only 3.8% remained severely underweight, while 42.9% were moderately underweight, and 53.3% had achieved a normal weight z-score. The mean weight z-score improved from -3.16 ± 1.34 at baseline to -2.11 ± 0.89 post-surgery ($P = 0.001$), indicating substantial weight gain and better nutritional recovery. These findings align with other studies.

Vaidyanathan et al. reported that 59% of children had a weight z-score ≤ -2 at presentation, with significant improvement post-surgery ($P < 0.001$).⁴ Hapuoja et al. observed significantly lower birth weight z-scores in CHD patients ($P = 0.007$), which

improved at 10-year follow-up ($P = 0.001$).¹⁰ He et al. found that 29.3% of CHD patients were underweight, with complex CHD cases showing the highest prevalence (42.9%) ($P < 0.001$).⁵⁷ Similarly, Okoromah et al. and Arodiwe et al. reported significantly lower weight z-scores in CHD patients ($P = 0.0001$ and $P = 0.001$, respectively), with Arodiwe et al. noting that 60% had a weight z-score ≤ -2 .^{30,48} Murni et al. also found that 49% of their cohort was underweight.⁵⁸

In our study, 20.0% of children were severely stunted (height z-score < -3), and 29.5% were moderately stunted (height z-score between -2 and -3), while 50.5% had a normal height z-score. Although stunting was present, height was less affected than weight. At 3 months post-surgery, severe stunting had decreased to 2.9%, moderate stunting to 25.7%, and 71.4% of children had achieved a normal height-for-age. The mean height z-score improved significantly from -1.97 ± 1.25 at baseline to -1.71 ± 1.04 post-surgery ($P = 0.001$), indicating improved linear growth. These findings are consistent with previous studies. Vaidyanathan et al. reported that 26.3% of children had a height z-score ≤ -2 at baseline, with significant improvement post-surgery ($P = 0.04$).⁴ Hapuoja et al. found that height z-scores were significantly below the norm at birth ($P < 0.001$) but improved by the 10-year follow-up ($P = 0.001$).¹⁰ He et al. reported that 24% of CHD patients were stunted, with complex CHD cases showing the highest prevalence (41.1%) ($P < 0.001$).⁵⁷ Similarly, Okoromah et al. and Arodiwe et al. found that the mean height z-score in CHD patients was significantly lower than in controls ($P = 0.0001$ and $P = 0.001$, respectively), with 28.8% classified as stunted.^{30,48} Murni et al. reported that 47.8% of CHD patients were stunted, with cyanotic CHD, delayed diagnosis, and low birth weight being significant predictors ($P < 0.001$).⁵⁸

Analysis of Head Circumference z-score revealed that 34 children (32.4%) had microcephaly and the majority (67.6%) had a normal head circumference. Microcephaly was seen in 18 children (17.1%) at follow-up after 3 months of corrective surgery and 87 children (82.9%) had normal head circumference. The mean head circumference z-score improved from -1.55 ± 1.20 at baseline to -1.16 ± 0.98 at 3 months ($P = 0.001$), showing enhanced brain growth and improved nutritional status after surgery. There are rarely studies showing head circumference assessment in this study population. Hapuoja et al. reported that the median head circumference z-score was significantly below the norm at birth ($P < 0.001$) but showed significant improvement at 10 years ($P = 0.001$).¹⁰

Further, in our study, severe wasting was observed in 16.2% of children, and moderate wasting was present in 64.8%, with only 19.0% having a normal weight-for-height z-score, indicating a high prevalence of acute malnutrition before surgery. However, at 3 months post-surgery, severe wasting was completely eliminated, moderate wasting decreased to 29.5%, and 70.5% of children achieved a normal weight-for-height. The mean weight-for-height z-score improved significantly from -2.80 ± 1.40 at baseline to -1.56 ± 0.89 post-surgery ($P = 0.001$), demonstrating enhanced nutritional recovery and catch-up growth. These findings are in line with previous research. Vaidyanathan et al. reported that 55.9% of children had a weight-for-height z-score ≤ -2 at baseline, with significant improvement at 3 months ($P < 0.001$).⁴ Hapuoja et al. also observed significant improvement in weight-for-height z-scores over time ($P < 0.001$).¹⁰ Okoromah et al. found that CHD patients had significantly lower weight-for-height z-scores compared to controls ($P = 0.0001$), with 41.1% classified as wasted³⁰. Similarly, Arodiwe et al. reported significantly lower weight-for-height z-scores in CHD patients compared to the reference

population ($P = 0.001$), with 41.1% classified as wasted¹⁰⁵. Murni et al. found that 31.4% of CHD patients had wasting, with cyanotic CHD and delayed diagnosis being significant predictors ($P < 0.001$)⁵⁸.

At baseline, 41.9% of children had severe acute malnutrition (SAM), which significantly decreased to 2.9% at 3 months post-surgery. Moderate acute malnutrition (MAM) also declined from 36.2% to 24.8%, while the proportion of children with normal nutritional status increased from 11.4% to 48.6%, reflecting substantial catch-up growth. Among children with MAM, 71.1% transitioned to normal status, while 59.1% of those with SAM also reached normal weight-for-age. However, a small number remained malnourished, emphasizing the need for continued nutritional support. These findings are consistent with Vaidyanathan et al., who reported a significant increase in normal weight-for-age cases post-surgery ($P < 0.001$)⁴. Okoromah et al. found severe malnutrition in 61.2% of CHD cases, with wasting more common in acyanotic CHD and stunting in cyanotic CHD ($P = 0.0001$) (30.). Arodiwe et al. reported severe malnutrition in 60% of cases, with a similar pattern of wasting and stunting distribution ($P = 0.001$)⁴⁸

Significant improvements were also observed in weight, height, and weight-for-height z-scores, indicating the positive impact of corrective surgery on growth and nutritional recovery. Enhanced cardiac function, better nutrient absorption, and improved metabolic efficiency likely contributed to this progress. Vaidyanathan et al. found similar improvements in all nutritional indices at 3 months ($P < 0.001$)⁴, while Hapuoja et al. reported sustained growth improvements over 10 years ($P < 0.001$)¹⁰. He et al. identified failure to thrive in 36.9% of CHD cases, with complex CHD showing the highest rates, and risk factors including low birth weight and prematurity ($P < 0.001$)⁵⁷.

Weight gain was significant across all CHD types ($P = 0.001$), particularly in ASD, VSD, and PDA cases, though the difference in weight gain between CHD types was not statistically significant ($P = 0.65$). Height-for-age scores improved significantly ($P < 0.05$), with the highest mean height gain observed in VSD cases (4.16 cm), suggesting pronounced catch-up growth. Vaidyanathan et al. reported significant height improvements at follow-up ($P = 0.04$)⁴, and Hapuoja et al. found substantial height increases over 10 years ($P < 0.001$)¹⁰.

Corrective surgery significantly improves nutritional status, weight gain, and linear growth in children with CHD. The reduction in malnutrition rates and improved growth parameters emphasize the need for timely intervention. While most children experience positive outcomes, some remain at risk for undernutrition, highlighting the importance of post-surgical nutritional support and long-term follow-up.

CONCLUSION

This study demonstrates that corrective surgery for congenital heart disease (CHD) in children under 5 years of age leads to significant improvements in growth and nutritional status within three months post-surgery. The findings highlight that most children with CHD present with severe growth retardation and malnutrition before surgery, which are strongly influenced by age and birth weight. The substantial increase in weight, height, and head circumference z-scores post-surgery reflects enhanced metabolic efficiency and improved nutrient absorption following surgical correction. The reduction in severe acute malnutrition (SAM) from 41.9% to 2.9% and the increase in children with normal nutritional status from 11.4% to 48.6% underscore the positive impact of early surgical intervention and post-operative nutritional care. The results show the importance of timely diagnosis, early corrective surgery, and comprehensive post-operative management in improving the growth outcomes and overall health of children with CHD.

SUMMARY

A hospital-based observational study was conducted at KLES Dr. Prabhakar Kore Hospital, Belgaum, Karnataka, from August 2023 to July 2024, to evaluate early growth trends in children under five years of age with congenital heart disease (CHD) who underwent corrective surgery. The study included 105 children who met the inclusion and exclusion criteria, with informed consent obtained from their parents. The demographic analysis of the study revealed that the majority of children (34.3%) who underwent corrective surgery were under 1 year of age, with a balanced distribution among the other age groups. A higher proportion of females (59%) underwent surgery compared to males (41%). Most children (59%) belonged to the lower middle-class socioeconomic group, highlighting the socioeconomic disparity in accessing healthcare for CHD. Regarding maternal health, 90.5% of mothers reported no significant antenatal complications, while 9.5% had a history of chronic hypertension, gestational diabetes, or hypothyroidism. Birth weight data showed that 55.2% of children had a birth weight greater than 2.5 kg, and 96.2% were born at term, indicating that most CHD cases requiring surgical correction were not linked to low birth weight or prematurity, but our study also shows that low birth weight (kgs) ($B = 0.84$, $P = 0.001$) was a significant positive predictor of pre-operative weight z-score and height/length z score

In terms of CHD type and severity, acyanotic CHD (88.6%) was more common than cyanotic CHD (11.4%). Atrial septal defect (ASD) (29.5%), ventricular septal defect (VSD) (28.6%), and patent ductus arteriosus (PDA) (21%) were the most prevalent types of CHD. Severe pulmonary arterial hypertension (PAH) was observed in 26.7% of cases, and 70.5% of children had severe intracardiac shunting. This reflects the hemodynamic complexity of the cases that required surgical correction.

Preoperative nutritional status assessment revealed that 88.6% of children were malnourished before surgery. Severe wasting, underweight, and stunting were highly prevalent at baseline, with a mean baseline weight z-score of -3.16, height z-score of -1.97, head circumference z-score of -1.55, and weight/height z-score of -2.80. This indicates that most children had significant growth delays and nutritional deficits before surgery.

At the 3-month follow-up after undergoing corrective cardiac surgery, there was a significant improvement in all growth parameters. The mean weight z-score improved from -3.16 to -2.11 ($P = 0.001$), height z-score increased from -1.97 to -1.71 ($P = 0.001$), head circumference z-score improved from -1.55 to -1.16 ($P = 0.001$), and weight/height z-score increased from -2.80 to -1.56 ($P = 0.001$). These improvements reflect enhanced metabolic efficiency, better cardiac function, and improved nutrient absorption following surgery. The proportion of children with normal nutritional status increased significantly from 11.4% at baseline to 48.6% at follow-up. Severe acute malnutrition (SAM) reduced from 41.9% at baseline to 2.9% at follow-up, while moderate acute malnutrition (MAM) decreased from 36.2% to 24.8%. This highlights the significant role of surgical correction and post-operative nutritional support in promoting catch-up growth and recovery from malnutrition.

Regression analysis identified significant predictors of pre-operative growth status. Age ($B = 0.56$, $P = 0.001$) and birth weight ($B = 0.84$, $P = 0.001$) were significant positive predictors of pre-operative weight z-score, indicating that older children and those with higher birth weight had better weight status before surgery. Birth weight ($B = 0.92$, $P = 0.001$) was also the only significant predictor of height/length z-score before surgery. For weight/height z-score, age was the only significant predictor ($B = 0.53$, $P = 0.001$), suggesting that older children were more

likely to have better weight-for-height status before surgery. Factors such as NICU admission, PAH severity, sepsis, duration of hospital stay, and residual lesions did not significantly influence pre-operative growth status, indicating that these medical complications were less important than age and birth weight in determining baseline growth parameters.

Weight and height gain analysis by type of CHD showed significant improvement in all groups. In ASD cases, the mean weight increased from 3.20 ± 1.44 kg at baseline to 11.97 ± 3.05 kg at follow-up ($P = 0.001$), with a mean weight gain of 1.37 ± 0.55 kg. In VSD cases, the mean weight increased from 1.61 ± 1.39 kg to 8.38 ± 2.45 kg ($P = 0.001$), with a mean gain of 1.53 ± 0.47 kg. PDA cases showed a similar trend, with a mean weight increase from 1.86 ± 1.63 kg to 9.20 ± 2.97 kg ($P = 0.001$), with a mean gain of 1.45 ± 0.50 kg. Height gain was highest in VSD cases, increasing from 72.76 ± 13.37 cm at baseline to 76.93 ± 12.24 cm at follow-up ($P = 0.001$), with a mean gain of 4.16 ± 2.12 cm. ASD and PDA cases also showed significant height improvements, with gains of 2.67 ± 1.28 cm and 3.11 ± 1.50 cm, respectively

BIBLIOGRAPHY

1. Saxena A, Mehta A, Sharma M. Birth prevalence of congenital heart disease: a cross-sectional observational study from North India. *Ann Pediatr Cardiol.* 2016;9:205–9.
2. Centre for Disease Control and Prevention. Data and statistics on congenital heart defects [Internet]. 2024 [cited 2025 Jan 24]. Available from: <https://www.cdc.gov/ncbddd/heartdefects/data.html>.
3. Saxena A. Congenital heart disease in India: a status report. *Indian Pediatr.* 2018;55:1075–82.
4. Vaidyanathan B, Nair SB, Sundaram KR, Babu UK, Shivaprakasha K, Rao SG, Kumar RK. Malnutrition in children with congenital heart disease (CHD): determinants and short-term impact of corrective intervention. *Indian Pediatr.* 2008;45:541-546.
5. Salzer HR, Haschke F, Wimmer M, Heil M, Schilling R. Growth and nutritional intake of infants with congenital heart disease. *Pediatr Cardiol* 1989; 10:17-23.
6. Menon G, Poskitt EME. Why does congenital heart disease cause failure to thrive? *Arch Dis Child* 1985; 60:1134-1139
7. Mir AB, Shah JA, Mushtaq S, Jan M. Prevalence, profile of malnutrition and iron deficiency anemia in children with cyanotic congenital heart defects: a case control observational study in a tertiary care hospital in North India. *International Journal of Contemporary Pediatrics.*2019; 6(3):1303–1308.
8. Diao J, Chen L, Wei J. Prevalence of Malnutrition in Children with Congenital Heart Disease: A Systematic Review and Meta-Analysis. *J Pediatr.* 2022;242:39-47.e4.

9. Bernstein D. Epidemiology and genetic basis of congenital heart disease. In: Kliegman RM, St. Geme JW, Blum NJ, Shah SS, Tasker RC, Wilson KM, editors. *Nelson Textbook of Pediatrics*. 21st ed. Philadelphia: Elsevier; 2024. p. 2763-2773.
10. Hapuoja L, Kretschmar O, Rousson V, Dave H, Naef N, Latal B. Somatic growth in children with congenital heart disease at 10 years of age: Risk factors and longitudinal growth. *Early Hum Dev*. 2021;156:105349
11. Bernstein D. Evaluation and screening of the infant or child with congenital heart disease. In: Kliegman RM, St. Geme JW, Blum NJ, Shah SS, Tasker RC, Wilson KM, editors. *Nelson Textbook of Pediatrics*. 21st ed. Philadelphia: Elsevier; 2024. p. 2773-2775.
12. Martorell G. O desenvolvimento da criança: do nascimento à adolescência. Trad. Bueno D, Pizzato R. Porto Alegre: McGraw-Hill; 2014.
13. Martins MF, Costa JS, Saforcada ET, Cunha MD. Quality of the environmental and associated factors: a pediatric study in Pelotas, Rio Grande do Sul, Brazil. *Cad Saúde Pública*. 2004;20(3):710-8.
14. Gianotti A. Efeitos psicológicos das cardiopatias congênitas. São Paulo: Lemos Editorial; 1996.
15. Pinto Jr VC, Daher CV, Sallum FS, Jatene MB, Croti AU. The situation of congenital heart surgeries in Brazil. *Rev Bras Cir Cardiovasc*. 2004;19(2): III-VI.
16. Monteiro MC. Um coração para dois: a relação mãe-bebê cardiopata [Dissertação de Mestrado]. Rio de Janeiro: Pontifícia Universidade Católica do Rio de Janeiro; 2003.
17. Rufo-Campos M, Rojas-Pérez I, Gómez-de Terreros M, Grueso-Montero J, Álvarez-Madrid A, Gómez-de Terreros I, et al. Estado psiconeurológico de los

- recién nacidos afectados de cardiopatía congénita antes de Su intervención.
Rev Neurol. 2003;37(8):705-10.
18. Frankenburg WK, Dodds J, Archer P, Shapiro H, Bresnick B. The Denver II: a major revision and restandardization of the Denver Developmental Screening Test. *Pediatrics*. 1992;89(1):91-7
19. Mari MA, Cascudo MM, Alchieri JC. Congenital heart disease and impacts on child development. *Braz J Cardiovasc Surg*. 2016;31(1):31-7.
20. Marino LV, Johnson MJ, Davies NJ. Improving growth of infants with congenital heart disease using a consensus-based nutritional pathway. *Clin Nutr*. 2020;39:2455–62.
21. Marino LV, Magee A. A cross-sectional audit of the prevalence of stunting in children attending a regional paediatric cardiology service. *Cardiol Young*. 2016;26:787–9.
22. Costello CL, Gellatly M, Daniel J. Growth restriction in infants and young children with congenital heart disease. *Congenit Heart Dis*. 2015;10:447–56.
23. Herridge J, Tedesco-Bruce A, Gray S. Feeding the child with congenital heart disease: a narrative review. *Pediatric Medicine*. 2021;4
<https://doi.org/10.21037/PM-20-77>.
24. Daymont C, Neal A, Prosnitz A. Growth in children with congenital heart disease. *Pediatrics*. 2013;121:e236–42.
25. Wong JJM, Cheifetz IM, Ong C Nutrition support for children undergoing congenital heart surgeries: a narrative review. *World J Pediatr Congenit Heart Surg*. 2015;6:443–54.
26. World Health Organization. Malnutrition fact sheet [Internet]. 2021 [cited 2025 Mar 16]. Available from: <https://www.who.int>.

27. Argent AC, Balachandran R, Vaidyanathan B, et al. Management of undernutrition and failure to thrive in children with congenital heart disease in low- and middle-income countries. *Cardiol Young*. 2017;27:S22–30.
28. Knirsch W, Zingg W, Bernet V. Determinants of body weight gain and association with neurodevelopmental outcome in infants operated for congenital heart disease. *Interact Cardiovasc Thorac Surg*. 2010;10:377–82.
29. Medoff-cooper B, Irving SY, Hanlon AL. The association among feeding mode, growth, and developmental outcomes in infants with complex congenital heart disease at 6 and 12 months of age. *J Pediatr*. 2016;169:154–159.e1.
30. Okoromah CAN, Ekure EN, Lesi FEA. Prevalence, profile and predictors of malnutrition in children with congenital heart defects: a case-control observational study. *Arch Dis Child*. 2011;96:354–60.
31. Jones CE, Desai H, Fogel JL. Disruptions in the development of feeding for infants with congenital heart disease. *Cardiol Young*. 2021;31:589–96.
32. Norman V, Zühlke L, Murray K. Prevalence of feeding and swallowing disorders in congenital heart disease: a scoping review. *Front Pediatr*. 2022;10.
33. Diao J, Chen L, Wei J. Prevalence of malnutrition in children with congenital heart disease: a systematic review and Meta-analysis. *J Pediatr*. 2022;242:39–47.e4.
34. Ross FJ, Radman M, Jacobs ML. Associations between anthropometric indices and outcomes of congenital heart operations in infants and young children: an analysis of data from the Society of Thoracic Surgeons database. *Am Heart J*. 2020;224:85–97.
35. Sethasathien S, Silvilairat S, Sittiwangkul R. Prevalence and predictive factors of malnutrition in Thai children with congenital heart disease and short-term

- postoperative growth outcomes. *Nutr Health Epub* ahead of print. 2022; <https://doi.org/10.1177/02601060221082382>.
36. Lim JYJ, Wee RWB, Gandhi M. The Associations between preoperative anthropometry and postoperative outcomes in infants undergoing congenital heart surgery. *Front Cardiovasc Med.* 2022;9 <https://doi.org/10.3389/fcvm.2022.812680>.
37. Silverman AH, Erato G, Goday P. The relationship between chronic paediatric feeding disorders and caregiver stress. *Journal of Child Health Care.* 2021;25:69–80.
38. Ringle ML, Wernovsky G. Functional, quality of life, and neurodevelopmental outcomes after congenital cardiac surgery. *Semin Perinatol.* 2016; <https://doi.org/10.1053/j.semperi.2016.09.008>.
39. Tregay J, Brown K, Crowe S. “I was so worried about every drop of milk” – feeding problems at home are a significant concern for parents after major heart surgery in infancy. *Matern Child Nutr.* 2017;13:1–11.
40. Golfenshtein N, Hanlon AL, Deatrck JA. Parenting stress trajectories during infancy in infants with congenital heart disease: comparison of single-ventricle and biventricular heart physiology. *Congenit Heart Dis.* 2019;14:1113–22.
41. Qin C, Li Y, Wang D. Maternal factors and preoperative nutrition in children with mild cases of congenital heart disease. *Jpn J Nurs Sci.* 2019;16:37–46.
42. Larson-Nath C, St Clair N, Goday P. Hospitalization for failure to thrive: a prospective descriptive report. *Clin Pediatr (Phila).* 2018;57:212–9.
43. Larson-Nath C, Goday P. Malnutrition in children with chronic disease. *Nutr Clin Pract.* 2019;34:349–58.
44. World Health Organization. Underweight prevalence among children under 5 years of age (%) (JME). *The Global Health Observatory* [Internet]. 2020

[cited 2024 Jun 16]. Available from: <https://www.who.int/data/gho/data/indicators/indicator-details/GHO/ghojme-underweight-prevalence>.

45. World Health Organization. Stunting prevalence among children under 5 years of age (%) (JME). Geneva: World Health Organization; 2020. p. 1–4.
46. Vaidyanathan B, Nair SB, Sundaram KR, Babu UK, Shivapparkasha KRS, KRK. Malnutrition in children with congenital heart disease (CHD): determinants and short-term impact of corrective intervention. *Indian Paediatrics*. 2008;45:541–6.
47. Varan B, Yilmaz G. Malnutrition and growth failure in cyanotic and acyanotic congenital heart disease with and without pulmonary hypertension; 1999. p. 49–52.
48. Arodiwe I, Chinawa J, Ukoha M. Nutritional status of children with congenital heart disease (CHD) attending university of Nigeria teaching hospital ituku – ozalla. *Enugu Pak J Med Sci*. 2015;31:1140–5.
49. Assefa B, Tadele H. Severe acute malnutrition among Unoperated Ethiopian children with congenital heart disease: a wake-up call to reverse the situation, a retrospective cross-sectional study. *Ethiop J Health Sci*. 2020;30:707–14.
50. Batte A, Lwabi P, Lubega S. Wasting, underweight and stunting among children with congenital heart disease presenting at Mulago hospital, Uganda. *BMC Pediatr*. 2017;17:1–7.
51. Woldesenbet R, Murugan R, Mulugeta F. Nutritional status and associated factors among children with congenital heart disease in selected governmental hospitals and cardiac center, Addis Ababa Ethiopia. *BMC Pediatr*. 2021; 21:1–9.

52. Ismail SR, Mehmood A, Rabiah N. Impact of the nutritional status of children with congenital heart diseases on the early post-operative outcome. *Egyptian Pediatric Association Gazette*. 2021;69 <https://doi.org/10.1186/s43054-021-00077-9>.
53. Hassan BA, Albanna EA, Morsy SM. Nutritional status in children with un-operated congenital heart disease: an Egyptian center experience. *Front Pediatr*. 2015;3<https://doi.org/10.3389/fped.2015.00053>.
54. Ratanachu-Ek S, Pongdara A. Nutritional status of pediatric patients with congenital heart disease: pre- and post cardiac surgery. *J Med Assoc Thai*. 2011;94:S133–7.
55. Rubia B, Kher A. Anthropometric assessment in children with congenital heart disease. *Int J Contemp Pediatrics*. 2018;5:634.
56. Amakali K, Small LF. The plight of parents/caregivers of children with heart disease in the rural areas of Namibia: a problem of coping. *Global J Health Sci*. 2012;5:62–73
57. He Q, Lin X, Zhou Z, Shen H, Ma K, Dou Z, et al. Failure to thrive in pediatric patients with congenital heart disease: A cross-sectional study of 13,256 patients. *Lancet Reg Health West Pac*. 2024;44:101002.
58. Murni IK, Patmasari L, Wirawan MT, Arafuri N, Nurani N, Sativa ER, et al. Outcome and factors associated with undernutrition among children with congenital heart disease. *PLoS One*. 2023;18(2):e0281753.
59. Li X, Zhu J, An J, Wang Y, Wu Y, Li X. Growth and development of children under 5 years of age with tetralogy of Fallot in a Chinese population. *Sci Rep*. 2021;11(1):14255.

60. Mohammad N, Shaikh S, Memon S, Das H. Spectrum of heart disease in children under 5 years of age at Liaquat University Hospital, Hyderabad, Pakistan. *Indian Heart J.* 2014;66(2):145–9.
61. Dittrich H, Bühler C, Grimmer I, Dittrich S, Abdul-Khaliq H, Lange PE. Neurodevelopment at 1 year of age in infants with congenital heart disease. *Heart.* 2003;89(4):436–41.

ANNEXURE – I - INFORMED CONSENT FORM

**“EARLY GROWTH TRENDS OF CHILDREN UNDER 5 YEARS OF AGE
WITH CONGENITAL HEART DISEASE WHO HAVE UNDERGONE
CORRECTIVE SURGERY - A LONGITUDINAL STUDY”**

Name of Student/Principal Investigator: **Dr.**

Name of Guide/Co Investigators: **Dr.**

Introduction: To measure the growth parameters in children with congenital heart disease to look for malnutrition and compare with growth parameters post surgery. Also to analyse for improvement in parameters.

Explanation of procedure: Growth parameters such as height, weight, head circumference, upper mid arm circumference and triceps skin fold thickness will be taken pre and post operatively at 3 months.

Withdrawal from participation in the study: Participation in this study is voluntary. You will be free to decide whether to participate in this study or continue participation once enrolled. In case you decide to withdraw your participation, you are free to do so. However, please convey the decision to the principal investigator.

Possible benefits from participating in the study: Participation of your child will help us to assess the prognosis of the child’s heart disease and also helps us to screen a lot of medical conditions also including malnutrition. You will get benefits by participating in this study. The data gathered will help population at large.

Possible risks from participating in the study: There are no risks involved in participating in this study

Privacy and confidentiality: The information collected from you will be coded, to prevent any person to identify you. Your identity will never be revealed. The data collected from you will be kept confidential and only processed or aggregated data will be used for publication.

Use of photography/Identifying details: Any photography or identification details will be disclosed only with your permission.

Financial incentives: You will not receive any payment for participating in this study. Cost of investigations done during the course of study will be paid by the principal investigator / Participant.

Authorization for publication of aggregated data: Results obtained after processing of the aggregated data will be published for scientific purpose and or presented to scientific groups. However, your identity will never be revealed.

Questions: In case of any questions with regard to this study, you are free to contact:

If you have any question or complaints with regard to your right as study participant you may contact Dr Harsha Hegde, Chairperson, Ethical committee of JNMC, 0831-2473777 Extension 4052.

Legal rights: By signing this consent form, we are not waving any of your legal rights

CONSENT STATEMENT

I am making a voluntary decision to participate in the study EARLY GROWTH TRENDS OF CHILDREN UNDER 5 YEARS OF AGE WITH CONGENITAL HEART DISEASE WHO HAVE UNDERGONE CORRECTIVE SURGERY - A LONGITUDINAL STUDY. My signature below indicates that I have decided to participate and I have read the information provided above or the information provided above has been read to me in the language that I understand best. I was given the opportunity to ask questions and that they have been answered to my satisfaction.

Name of the participant:

Signature or left thumb impression of the participant:

Name of the witness:

Signature or left thumb impression of the witness:

Name of the investigator:

Signature of the investigator:

ANNEXURE II- DEFINITIONS

DEFINITIONS: (ACCORDING TO WHO):

MODERATE UNDERWEIGHT: It is defined as weight for age less than $-2z$ and $>3Z$ or Weight-for-age <-2 SD and ≥-3 SD of the median.

SEVERE UNDERWEIGHT: It is defined as weight for age less than $-3z$ or Weight-for-age <-3 SD of the median

MODERATE WASTING: It is defined as weight-for-height between -3 and -2 Z-scores of the WHO Child Growth Standards median

SEVERE WASTING : It is defined as weight-for-height less than $-3Z$ -scores of the WHO Child Growth Standards median

MODERATE STUNTING: It is defined as Length/height-for-age between $<-3z$ and $-2z$ or Length/height-for-age <-2 SD and ≥-3 SD of the median.

SEVERE STUNTING : It is defined as Length/height-for-age <-3 SD of the median or $<-3Z$.

SEVERE ACUTE MALNUTRITION : weight-for-length <-3 Z-scores of the WHO Child Growth Standards median or or clinical signs of bilateral pitting oedema, or a very low mid-upper arm circumference (MUAC < 11.5 cms)

MODERATE ACUTE MALNUTRITION : It is defined as moderate wasting (i.e. weight-for-height between -3 and -2 Z-scores of the WHO Child Growth Standards median) and/or mid-upper-arm circumference (MUAC) greater or equal to 115 mm and less than 125 mm.

ANNEXURE III- PROFORMA

PATIENT'S PROFORMA

NAME:

AGE:

SEX:

D.OB:

IP NO:

PH NO:

ADDRESS:

EDUCATIONAL QUALIFICATION OF PARENTS : FATHER-

MOTHER-

SOCIO-ECONOMIC STATUS :

PRESENTING COMPLAINTS:

BIRTH HISTORY:

DIAGNOSIS:

SEPSIS AT THE TIME OF PRESENTATION : YES/NO

ECHO FINDINGS ON SEVERITY OF PAH : MILD/MODERATE/SEVERE

NO OF DAYS OF HOSPITALISATION :

DAYS PRESENCE OF RESIDUAL LESION:

COMPLEXITY OF THE LESION:

SEVERITY OF THE SHUNT(BASED ON SIZE):

ANTHROPOMETRIC MEASUREMENTS

| PARAMETERS | PRE-OP MEASUREMENTS | POST-OP AT 3 MONTHS MEASUREMENTS |
|-----------------------------------|---------------------|----------------------------------|
| WEIGHT (KGS) | | |
| HEIGHT/LENGTH (CMS) | | |
| HEAD CIRCUMFERENCE (CMS) | | |
| MID UPPER ARM CIRCUMFERENCE (CMS) | | |

INFERENCE : TABLE 1

| PARAMETER | Z-SCORE/ PERCENTILE | |
|--------------------------|---------------------|---------------------|
| | PRE-OP | POST-OP AT 3 MONTHS |
| WEIGHT FOR AGE | | |
| HEIGHT/LENGTH FOR AGE | | |
| HEAD CIRCUMFERENCE | | |
| WEIGHT FOR HEIGHT/LENGTH | | |

INFERENCE : TABLE 2

| | WASTING | UNDER-WEIGHT | STUNTING | TYPE OF MALNUTRITION |
|---------|---------|--------------|----------|----------------------|
| | P/A | P/A | P/A | SAM/MAM/CM/ACM |
| PRE-OP | | | | |
| POST-OP | | | | |

P- PRESENT

A- ABSENT

SAM- SEVERE ACUTE MALNUTRITION

MAM- MODERATE ACUTE MALNUTRITION

CM- CHRONIC MALNUTRITION

ACM- ACUTE ON CHRONIC MALNUTRITION

COMMENTS:

ANNEXURE IV- PHOTOGRAPHS

Photo 1 : Stadiometer used for measuring height



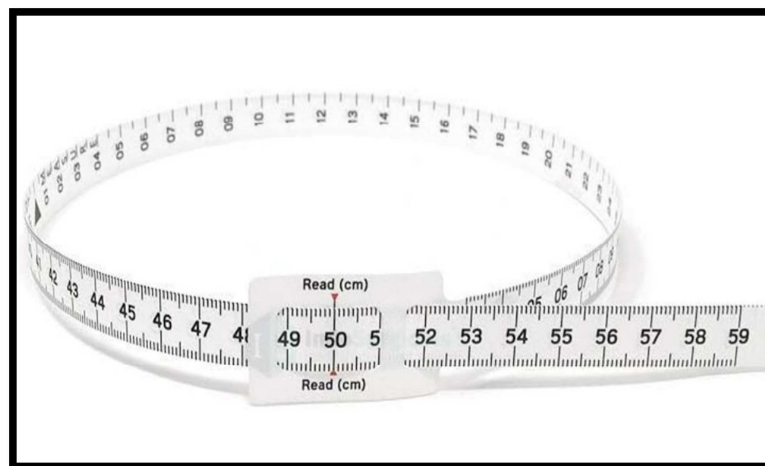
Photo 2 : Infantometer used for measuring length



Photo 3: Multipurpose digital weighing scale ranging from 50g to 120kgs with 5g division was used (meditive brand)



Photo 4 : IS Indosurgicals head circumference measuring tape calibrated upto 60cm and with 1mm graduation was used.



ANNEXURE V- KEY TO MASTER CHART

| Variable | Code | Key |
|------------------------------------|------|-----------|
| Gender | 1 | Male |
| | 2 | Female |
| Type of CHD | 1 | Acyanotic |
| | 2 | cyanotic |
| Gestation | 1 | term |
| | 2 | preterm |
| Sepsis | 1 | Yes |
| | 2 | No |
| Mode of delivery | 1 | NVD |
| | 2 | LSCS |
| Complexity of lesion | 1 | simple |
| | 2 | complex |
| Presence of residual lesion | 1 | Yes |
| | 2 | No |
| wasting | 1 | present |
| | 2 | absent |
| underweight | 1 | present |
| | 2 | absent |
| stunting | 1 | present |
| | 2 | absent |
| microcephaly | 1 | present |
| | 2 | absent |

| | | |
|--------------------------|---|----------|
| severity of shunt | 1 | mild |
| | 2 | moderate |
| | 3 | severe |
| | 4 | n/a |
| severity of PAH | 1 | mild |
| | 2 | moderate |
| | 3 | severe |
| | 4 | No |

**ANNEXURE VI-
MASTER CHART**

