"CLINICAL AND ECHO-CARDIOGRAPHIC STUDY OF NEONATES WITH SUSPECTED CONGENITAL HEART DISEASE"

Submitted By

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DISSERTATION SUBMITTED TO THE

In partial fulfillment of the requirement for the degree of

DOCTOR OF MEDICINE

In

PEDIATRICS

Under the guidance of

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ACKNOWLEDGEMENT

On completion of this scientific document it gives me deep pleasure to acknowledge the guidance provided to me by my distinguished mentors.

With privilege and respect I would like to express my profound gratitude and indebtedness to my Guide and esteemed teacher, **Dr. S.S.KALYANSHETTAR**$_{MD,}$**,** Professor**,** Department of **PEDIATRICS**, B.L.D.E.U's Shri. B. M. Patil Medical College, Bijapur, for his constant inspiration, extensive encouragement and support, which he rendered in pursuit of my post-graduate studies and in preparing this dissertation.

I am extremely grateful to my eminent and esteemed teacher **Dr. S.V.PATIL**$_{\mathbf{MD}}$, Professor & Head Of the Department, Pediatrics for his overall guidance and inspiration without which this would not have seen the light of the day. I am grateful to him for what I have learnt from him and for his kind support.

I sincerely thank my teacher **DR A.S.AKKI**$_{MD,DCH}$ Professor**,** Department of **PEDIATRICS** B.L.D.E.U's Shri. B. M. Patil Medical College, Bijapur for his constant inspiration and help.

I sincerely thank my teacher **DR R.H.GOBBUR**$_{MD,DCH}$ Professor**,** Department of **PEDIATRICS** B.L.D.E.U's Shri. B. M. Patil Medical College, Bijapur for his constant inspiration and help.

I am greatly thankful to my teacher **Dr.M.M.PATIL**$_{\mathbf{MD}}$, Department of Pediatrics for his timely suggestions and willingness to help all the time.

I sincerely thank to medical superintendent for allowing me to do echo-cardiography free of cost.

I sincerely thank my fellow postgraduates and friends for their support and co-operation. My sincere thanks to nursing staff of Department of **PEDIATRICS,** Shri.

B. M. Patil Medical College, Bijapur for their kind co-operation from time to time in carrying out the study.

I am thankful to **Dr. M .S. BIRADAR** Principal, B.L.D.E.U's Shri. B. M. Patil Medical College Hospital and Research Centre, Bijapur, for permitting me to conduct & utilize resources in completion of my work.

I express my thanks to the library staff and all hospital staff for their kind co-operation in my study.

I am deeply indebted to my parents, and sisters whose constant encouragement and inspiration led me to successful completion of my dissertation work.

Finally I pray to God for continued blessing and success.

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LIST OF ABBREVIATION

CHD	-	Congenital heart disease
NICU	-	Neonatal intensive care unit
ECHO	-	Echocardiography
CVS	-	Cardiovascular system
SVC	-	Superior vena cava
IVC	-	Inferior vena cava
RV	-	Right ventricle
RA	-	Right atrium
LV	-	Left ventricle
LA	-	Left atrium
PA	-	Pulmonary artery
PV	-	Pulmonary Vein
PVR	-	Pulmonary vascular resistance
PDA	-	Patent ductus arteriosus
ASD	-	Atrial septal defect
VSD	-	Ventricular septal defect
PGE2	-	Prostaglandin E2
PO_2	-	Partial pressure of oxygen

PPHN	-	Persistent pulmonary hypertension of newborn
CHF	-	Congestive heart failure
TOF	-	Tetralogy of Fallot
PS	-	Pulmonary stenosis
TGA	-	Transposition of great arteries
CoA	-	Co-arctation of aorta
AS	-	Aortic stenosis
MS	-	Mitral stenosis
PS	-	Pulmonary stenosis
TS	-	Tricuspid stenosis
AR	-	Aortic regurgitation
TR	-	Tricuspid regurgitation
MR	-	Mitral regurgitation
BP	-	Blood pressure
S1	-	First heart sound
S2	-	Second heart sound
S3	-	Third heart sound
S4	-	Forth heart sound
P2	-	Pulmonary component of second heart sound

A2	-	Aortic component of second heart sound
LBBB	-	Left bundle branch block
RBBB	-	Right bundle branch block
TAPVR	-	Total anomalous pulmonary venous return
PAPVR	-	Partial anomalous pulmonary venous return
WPW	-	Wolf- Parkinson’s- White
ECD	-	Endocardial cushion defect
HOCM	-	Hypertrophic obstructive cardiomyopathy
IHSS	-	Idiopathic hypertrophic subaortic stenosis
AV	-	Atrioventricular
HLHS	-	Hypoplastic left heart syndrome
ECG	-	Electrocardiography
LVOT	-	Left ventricular outflow tract
RVOT	-	Right ventricular outflow tract

ABSTRACT

ABSTRACT

BACKGROUND: In common usage CHD refers to structural heart defects that are present at birth. History, physical examination, chest x-ray, ECG, Echocardiography help in identifying presence of CHD. Most commonly neonates with CHD presents with murmur, cyanosis and or congestive heart failure. Echocardiography provides reliable & reproducible information on cardiovascular form & function. Detailed cardiac structures can be identified, with differentiation from normal to abnormal anatomy.

OBJECTIVE: Clinical and echocardiographic study of neonates with suspected heart disease, admitted in NICU(Neonatal intensive care unit) & postnatal wards of Shri B. M. Patil medical college, Hospital and research Centre, Bijapur. To determine profile of congenital heart disease in neonates

DESIGN: Prospective study

SETTING: A tertiary care center, University hospital in Bijapur

PARICIPANTS: The study included 74 neonates admitted to NICU & post natal wards.

RESULTS: Total number of neonates admitted were 2091 during study period. In our study total 74 patients were included, out of them 22 (30%) cases were normal & 52 (70%) cases were having CHD. In our study total 52 patients were having congenital heart disease, out of them 27 (51.92%) cases were male & 25 (48.08%) cases were female, 16 (30.77%) cases were ASD, 19(36.53%) cases were having multiple

defects, 9 (17.31%) cases were having PDA, 5 (9.62%) cases were having VSD & 3 (5.77%) cases were having cyanotic CHD.

Most frequent symptom which was observed in cases was feeding difficulty 12 (46.15%) cases, followed by fast breathing 10 (38.46%) cases.

In our study most common clinical examination finding found was respiratory distress, 12 (33.33%) cases followed by 10 (27.78%) cases of bounding pulses

Congenital malformations were seen in 5 (13.89%) cases. The incidence of cardiac murmur was 35.38 per 1000 admissions & incidence of congenital heart disease was 24.86 per 1000 admissions. We found 8 cases of PPHN & were excluded from study.

CONCLUSION : Utility of clinical & echocardiographic examination in assessment of neonates with heart murmurs in diagnosis of congenital heart disease is valuable. Echo-cardiography gives correct anatomical diagnosis in suspected congenital heart diseases.

Early diagnosis helps in predicting the prognosis & for planning future treatment decisions.

Most common congenital heart disease found in our study was ASD.

KEY WORDS: Echo-cardiography, murmur

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INTRODUCTION

Congenital malformations of heart & circulation are not fixed anatomic defect that appear at birth but instead are anomalies in flux that originate in early embryo, evolve during gestation, and survive dramatic circulatory alterations at birth[1].

At beginning of 20th century Dr. William Ostler wrote in his textbook of medicine that congenital heart disease (CHD) was of "limited clinical interest as in large proportion of cases anomaly is not compatible with life & in others nothing can be done to remedy the defect or even relieve the symptoms". In the years since then, the outlook for children with CHD has improved dramatically. This remarkable progress is due to synergistic advances in pediatric & fetal cardiology, cardiac surgery, neonatology, cardiac anesthesia, intensive care & nursing[2].

Congenital heart disease (CHD) is the most common birth defect encountered in clinical setting. The presentation in newborn with CHD can range from absence of symptoms to complete cardiovascular collapse. Most commonly neonates with CHD presents with murmur, cyanosis and or congestive heart failure[3].

History, physical examination, X-ray, ECG, echo-cardiography help in identifying presence of CHD. Echo-cardiography is the application of ultrasound to evaluation of cardiovascular system. Over the past several decades echo-cardiography has become the gold standard for determination of CHD. Echocardiography provides reliable & reproducible information on cardiovascular form & function. Detailed cardiac structures can be identified, with differentiation from normal to abnormal anatomy.

Using high resolution two dimensional echo-cardiography, components of hemodynamics, such as blood flow velocity & spatial direction can be used to derive pressure measurements by use of Doppler echo-cardiography. Information acquired is

non invasive, real time & acquired at patient bedside[4]. The advent of echocardiography with Doppler color flow measurements has made it possible to diagnose lesions that are asymptomatic, minor and even without murmurs[6].

AIMS AND OBJECTIVES

1. Clinical and echo-cardiographic study of neonates with suspected heart disease, admitted in NICU(Neonatal intensive care unit) & postnatal wards of Shri B. M. Patil medical college, Hospital and research Centre, Bijapur
2. To determine profile of congenital heart disease in neonates

REVIEW OF LITERATURE

Congenital heart disease was defined as by Mitchell et al as "A gross structural abnormality of the heart or intra-thoracic great vessel that is actually or potentially of functional importance". Within a prospective study of 56,109 total births, 457 patients have been found to have congenital heart disease. The overall incidence is 8.14/1000 total births[5].

In a Study done by J I E Hoffman, the incidence of congenital heart disease (CHD) in the Western industrialized world has varied from a low value of about 3 to 5 per 1000 live births to about 12 per 1000 live births. Most of the lower incidence figures were obtained before there were sufficiently well trained pediatric cardiologists and before the success of cardiac surgery put a premium on early and correct diagnosis of CHD. The advent of echocardiography with Doppler color flow measurements has made it possible to diagnose lesions that are asymptomatic, minor, and even without murmurs[6].

In a retrospective study done in Kanpur, India Clinical examination, echocardiography and color doppler were used as diagnostic tools. A prevalence of 26.4 per 1000 patients was observed[7].

According to Du ZD, Roguin N, Barack M, clinical evaluation could determine the presence or absence of heart disease in most neonates, lesion specific diagnosis was not quite satisfactory. Echocardiography is necessary for neonates with a clinically diagnosed heart disease or possible heart diseases, and may be unnecessary for those with innocent murmurs diagnosed by pediatricians[8].

FETAL CIRCULATION:

Fetal circulation differs from adult circulation in several ways. Almost all differences are attributable to fundamental differences in site of gas exchange. In adults gas exchange occurs in lung. In the fetus, placenta provides the exchanges of gases & nutrients[9].

The fetal circulation is arranged in parallel, rather than in a series, right ventricle delivering the majority of its output to placenta for oxygenation, & left ventricle delivering its majority of output to heart, brain & upper part of the body. However there is mixing of streams at atrial & great vessel level that diverts blood from immature lungs to the placenta for oxygen exchange. This parallel circulation permits fetal survival despite wide variety of cardiac lesion[10].

There are four shunts in fetal circulation: placenta, ductus venosus, foramen ovale, ductus arteriosus. The placenta receives the largest amount of combined (i.e. right & left) ventricular output (55%) & has lowest vascular resistance in fetus. The superior vena cava (SVC) drains upper part of body, including the brain, whereas inferior vena cava (IVC) drains lower part of the body & placenta. Because the blood is oxygenated in placenta, oxygen saturation in IVC is higher than SVC. The highest partial pressure of oxygen is found in umbilical vein (32mm hg).

Most of SVC blood goes to right ventricle (RV). One third of IVC blood with higher oxygen saturation is directed by christa dividance to left atrium (LA) through foramen ovale, whereas remaining two thirds enters right ventricles & pulmonary artery. The result is that the brain & coronary circulation receive blood with higher oxygen saturation than lower half of body. Less oxygenated blood in pulmonary artery (PA) flows through widely open ductus arteriosus to descending aorta & than to placenta for oxygenation

Because lungs receive only 15% of combined ventricular output, branches of pulmonary artery are small. Also, RV is larger & more dominant than left ventricle (LV). RV handles 55% of combined ventricular output, where LV handles 45% of combined ventricular output. In addition, pressure in RV is identical to that in LV (unlike that in adult) [9].

FETAL CARDIAC OUTPUT

Unlike the adult heart, which increases its stroke volume when the heart rate decreases, the fetal heart is unable to increase stroke volume when the heart rate falls because it has a low compliance. Therefore, fetal cardiac output depends on heart rate, when the heart rate drops as in fetal distress, serious fall in cardiac output results[9].

CHANGES IN CIRCULATIONS AFTER BIRTH:

The primary change in circulation after birth is a shift of blood flow for gas exchange from the placenta to the lungs. The placental circulation disappears, & pulmonary circulation is established

1. Removal of placenta results in following:

a. An increase in systemic vascular resistance

b. Cessation of blood flow in umbilical vein resulting in closure of ductus venosus

2. Lung expansion results in following:

a. A reduction of pulmonary vascular resistance (PVR), an increase in pulmonary blood flow, & fall in pulmonary artery (PA) pressure

b. Functional closure of foramen ovale as a result of increase pressure in left atrium (LA) than right atrium (RA). The LA pressure increases as a result of increased pulmonary blood flow & increased pulmonary venous return to LA. The RA pressure falls because of closure of ductus venosus

c. Closure of ductus arteriosus as a result of increased arterial oxygen saturation[9]

PULMONARY VASCULAR RESISTANCE

The PVR is as high as systemic vascular resistance near or at term. The high PVR is maintained by an increased amount of smooth muscle in wall of pulmonary arterioles & alveolar hypoxia resulting from collapsed lung With expansion of lungs & resulting increase in alveolar oxygen tension, there is initial & rapid fall in PVR. This rapid fall is secondary to direct vasodilating effect of oxygen on pulmonary vasculature. Between 6 & 8 weeks after birth, there is slower fall in PVR & PA pressure. This fall is associated with thinning of medial layer of pulmonary arterioles. A further decline of PVR occurs after 2 years. This may be related to increase in number of alveolar units & their associated vessels [9] .

Many neonatal conditions causing inadequate oxygenation may interfere with normal maturation (i.e., thinning) of pulmonary arterioles, resulting in persistent pulmonary hypertension or delay in fall of PVR. They include:

1. Hypoxia & or high altitude
2. Lung disease (Hyaline membrane disease)
3. Acidemia
4. Increased pulmonary artery pressure secondary to large ventricular septal defect or Patent ductus arteriosus (PDA).
5. Increased pressure in LA or pulmonary vein (PV) [9].

CLOSURE OF DUCTUS ARTERIOUSES

Functional closure of ductus arteriosus occurs within 10-15 hours after birth by constriction of medial smooth muscles in ductus. Anatomical closure is completed in 2-3 weeks of age by permanent changes in endothelium & subintimal layer of

ductus. Acetylcholine & bradykinin also constrict ductus. Oxygen, prostaglandin E_2 (PGE2) levels, & maturity of newborn are important factors in closure of ductus [9].

Oxygen & the ductus. A postnatal increase in oxygen saturation of systemic circulation (from Po_2 of 25mm Hg in utero to 50mm of Hg after lung expansion) is the strongest stimulus for constriction of ductus smooth muscle, which leads to closure of ductus. The responsiveness of ductus smooth muscle to oxygen is related to gestational age of newborn; the ductal tissue of premature infant responds less intensely to oxygen than that of full term infant. This decreased responsiveness of immature ductus to oxygen is due to its decreased sensitivity to oxygen induced contraction; it is not the result of lack of smooth muscle development, as immature ductus constricts well in response to acetylcholine [9].

Prostaglandin E & Ductus.

1. Decrease in PGE2 levels after birth results in constriction of ductus. This decrease results from removal of placental source of PGE2 production at birth & from marked increase in pulmonary blood flow, which allows effective removal of circulating PGE2 by lungs
2. Constricting effects of indomethacin & dilator effects of PGE2 & prostaglandin I_2 are greater in ductal tissue of immature fetus than of near term fetus
3. Maternal ingestion of large amount of aspirin, an inhibitor of prostaglandin synthatase, may harm the fetus because aspirin may constrict the ductus during fetal life & may result in persistent pulmonary hypertension of newborn (PPHN) [9].

Reopening of constricted ductus. Before true anatomic closure occurs, the functionally closed ductus may be dilated by reduced arterial Po_2 or increased PGE2 concentration. The reopening of constricted ductus may occur in asphyxia & various pulmonary diseases (as hypoxia & acidosis relax ductal tissue). Ductal closure is delayed at high altitude. There is much higher incidence of PDA at high altitude than at sea level

Response of pulmonary artery & ductus arteriosus to various stimuli. The PA responds to oxygen & acidosis in opposite manner from ductus arteriosus. Hypoxia & acidosis relax the ductus arteriosus but constricts the pulmonary arterioles. Oxygen constricts the ductus but relaxes the pulmonary arterioles. The PAs are also constricted by sympathetic stimulation & alpha adrenergic stimulation. Vagal stimulation, beta adrenergic stimulation & bradykinin dilate PA[9].

PREMATURE NEWBORNS

Two important problems that premature infants may face are related to the rate at which pulmonary vascular resistance (PVR) falls & responsiveness of ductus arteriosus to oxygen

The ductus arteriosus is more likely to remain open in preterm infants after birth because premature infant's ductal smooth muscles does not have a fully developed constrictor response to oxygen. In addition, premature infants have persistently high circulating levels of PGE2 (possibly caused by increased production or decreased degradation in lungs), and premature ductal tissue exhibits an increased dilatory response to PGE2

In premature infants, pulmonary vascular smooth muscle is not as well developed as in full term infants. Therefore, fall in PVR occurs more rapidly than in

mature infants. This accounts for early onset of a large left to right shunt & congestive heart failure (CHF) [9].

CLASSIFICATION OF CONGENITAL HEART DISEASES [1]:

1. **Acyanotic without a shunt**
    a. Malformations originating in left side of heart
        1. Obstruction to left atrial inflow - pulmonary vein stenosis, mitral stenosis, cortriatrium
        2. Mitral regurgitation
        3. Primary dilated endocardial fibroelastosis
        4. Aortic stenosis
        5. Aortic regurgitation
        6. Coarctation of aorta
    b. Malformations originating in right side of heart
        1. Acyanotic Ebstein's anomaly of tricuspid valve
        2. Pulmonary stenosis
        3. Congenital pulmonary valve regurgitation
        4. Idiopathic dilatation of pulmonary trunk
        5. Primary pulmonary hypertension
2. **Acyanotic with a shunt (left to right)**
    a. Shunt at atrial level
        1. Atrial septal defect
        2. Partial anomalous pulmonary venous connection
        3. Atrial septal defect with mitral stenosis (Lutembacher syndrome)
    b. Shunt at ventricular level
        1. Ventricular septal defect (VSD)

2. VSD with aortic regurgitation
3. VSD with left ventricular to right atrial shunt

c. Shunt between aortic root & right side of heart
    1. Coronary arteriovenous fistula
    2. Ruptured sinus of valsalva aneurism
    3. Anomalous origin of left coronary artery from pulmonary trunk

d. Shunt at aorto-pulmonary level
    1. Aortopulmonary window
    2. Patent ductus arteriosus
    3. Shunts at more than one level
    4. Complete common atrioventricular canal

3 Cyanotic

a. Increased pulmonary arterial blood flow
    1. Complete transposition of great vessels
    2. Taussig Bing anomaly
    3. Truncus arteriosus
    4. Total anomalous pulmonary venous connection
    5. Univentricular heart with low pulmonary vascular resistance & no pulmonary stenosis
    6. Common atrium
    7. Fallot's tetralogy with pulmonary atresia & increased collateral arterial flow
    8. Tricuspid atresia with nonrestrictive VSD.

Normal or decreased pulmonary arterial blood flow

1. Dominant left ventricle
    a. Tricuspid atresia
    b. Pulmonary atresia with intact ventricular septum
    c. Ebstein's anomaly of tricuspid valve
    d. Single morphologic left ventricle with pulmonary stenosis
2. Dominant right ventricle
    a. No pulmonary hypertension
        1. Pulmonary stenosis or atresia with ventricular septal defect
        2. Pulmonary stenosis or atresia with intact ventricular septum & right to left interatrial shunt
        3. Pulmonary stenosis with complete transposition of great arteries
        4. Double outlet right ventricle with pulmonary stenosis
    b. Pulmonary hypertension
        1. Atrial septal defect with reversal shunt
        2. Ventricular septal defect with reversal of shunt
        3. PDA or aortopulmonary window with reversal of shunt
        4. Double outlet right ventricle with high pulmonary vascular resistance
        5. Complete transposition of great vessels with high pulmonary vascular resistance
        6. Total anomalous pulmonary venous connection with high pulmonary vascular resistance
        7. Hypo plastic left heart syndrome

3. Normal or nearly normal ventricles

   Pulmonary arteriovenous fistula vena caval to left atrial communication

DIAGNOSIS OF CONGENITAL HEART DISEASE.

Clinical presentation of congenital heart disease in neonates:

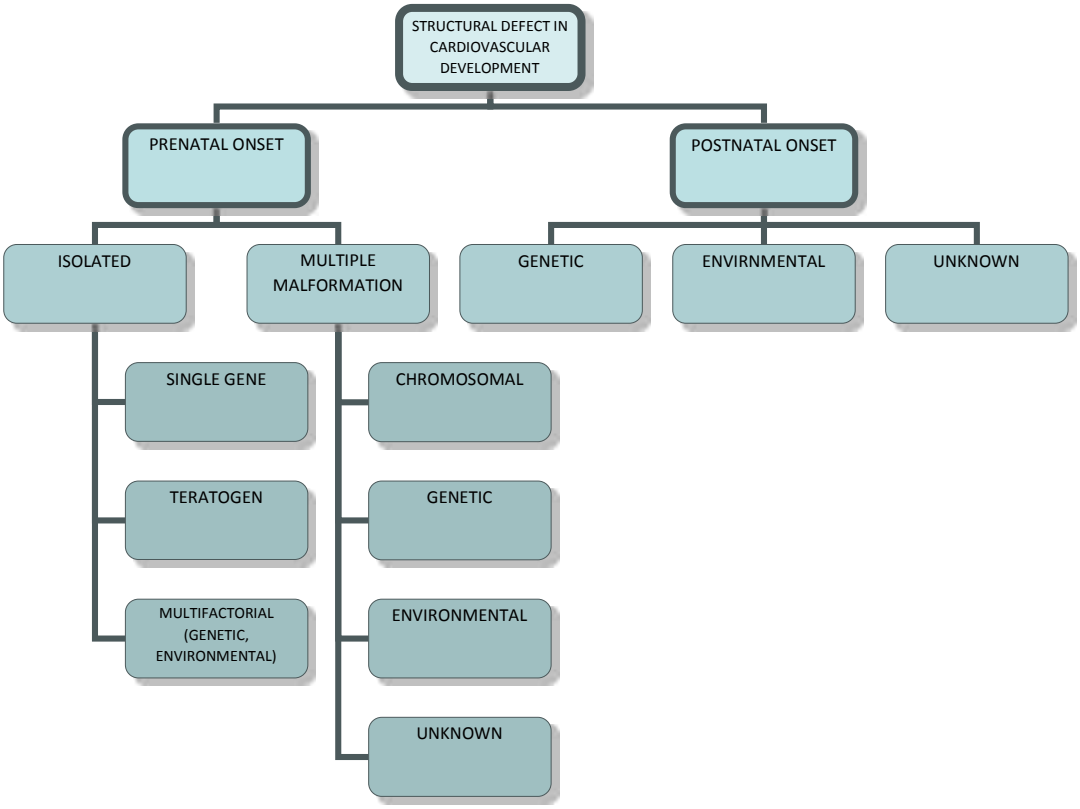
Timing of presentation & accompanying symptomatology depends upon-

1. Nature & severity of anatomic defect
2. In utero effects of structural lesion
3. Alteration in cardiovascular physiology secondary to effects of transitional circulation: **closure of ductus arteriosus & fall in PVR[2].**

In first few weeks of life, many heterogeneous forms of heart disease present in surprisingly limited number of ways (in no particular order nor mutually exclusive):

a. Cyanosis
b. CHF (with most extreme presentation being cardiovascular collapse & shock)
c. An asymptomatic heart murmur
d. Arrhythmia[2].

When evaluating an infant or child with structural defect it must be first determined whether the defect has a prenatal or postnatal onset. The term prenatal onset is used to designate structural abnormalities that are present at birth, whereas postnatal onset is used to designate structural abnormalities that are not present at birth, but rather develop postnatally. Once distinction between prenatal & postnatal onset has been made, a rational differential diagnosis can be developed, since this determination narrows the diagnostic possibilities considerably[11].



A DEVELOPMENTAL APPROACH TO CHILD WITH STRUCTURAL DEFECT[11].

HISTORY:

Gestational & natal history:

Infections, medications, excessive alcohol intake may cause CHD, if they occur early in pregnancy

Infections:

1. Maternal rubella infection during first trimester of pregnancy results in multiple anomalies, including cardiac defects. (patent ductus arterious, peripheral pulmonary stenosis, fibromuscular & intimal proliferation of medium & large arteries, ventricular septal defects, arterial septal defects)[11,12]
2. Infection by cytomegalovirus, herpesvirus & coxsackievirus B are suspected to be teratogenic if they occur in early pregnancy. Infection by these viruses later in pregnancy causes myocarditis
3. Human immunodeficiency virus infection (in illicit drug users) has been associated with infantile cardiomyopathy[12].

Cardiovascular abnormalities caused by teratogens[11]

Recognizable phenotypes (Syndromes)	Cardiac abnormalities
CHEMICAL TERATOGENS	
Fetal alcohol syndrome	VSD, ASD, TOF, coarctation of aorta
Fetal hydantoin syndrome	VSD, TOF, PS, PDA, ASD, coarctation of aorta
Fetal trimethadione syndrome	Combined defects
Fetal valproate syndrome	Nonspecific
Fetal carbamazepine syndrome	VSD, TOF
Retinoic acid embryopathy	Conotruncal malformations
Thalidomide embryopathy	Conotruncal malformations
Fetal warfarin syndrome	PDA, peripheral pulmonary stenosis
Lithium	Ebstein anomaly, tricuspid atresia, ASD
Maternal PKU fetal effects	TOF, VSD, coarctation of aorta
Maternal lupus fetal effects	Complete heart block, cardiomyopathy, L- transposition of great arteries
Maternal diabetes	Transposition of great arteries, VSD, coarctation of aorta, hypertrophic cardiomyopathy

Ventricular septal defect (VSD), atrial septal defect (ASD), tetralogy of fallot (TOF), Patent ductus arteriosus (PDA), pulmonary stenosis (PS)

Perinatal history:

Maternal history of premature rupture of membranes, fever, or use of sedatives or anesthetics raise concern about sepsis & decreased respiratory effort. For infants with cyanosis, gestational age, apgar scores, history of meconium aspiration are useful to determine likelihood of hyaline membrane disease, perinatal asphyxia, persistent pulmonary hypertension of newborn (PPHN), or pneumonia. The response to oxygen helps to distinguish a cardiac from pulmonary basis for cyanosis[13,14].

Birth weight:

Birth weight provides important information about nature of cardiac problem

1. If an infant is small for gestational age, this may indicate intra-uterine infection or use of chemicals or drugs. Congenital rubella syndrome, fetal alcohol syndrome are typical examples
2. Infant with high birth weight, often seen in infant of diabetic mothers, show a higher incidence of cardiac anomalies. Infants with TGA often have a birth weight higher than average; these infants are cyanotic[12].

POSTNATAL HISTORY:

Weight gain, general development may be delayed in infant & children with congestive heart failure (CHF) or severe cyanosis. Weight is affected more than height. If weight is severely affected than pediatrician should suspect a more general dysmorphic condition. Poor feeding of recent onset may be an early sign of CHF in infants, especially poor feeding is result of fatigue & dyspnea[12].

TIME OF ONSET OF CONGESTIVE FAILURE[15]

AGE	LESION
Birth-1 week	Duct dependent systemic circulation (hypoplastic left heart syndrome, critical aortic stenosis, severe coarctation, arch interruption); total anomalous pulmonary venous return (obstructed), congenital mitral & tricuspid valve regurgitation, ebstein anomaly
1-4 weeks	Patent ductus arteriosus (PDA) in preterms, VSD with coarctation, persistent truncus arteriosus, transposition with largr VSD or PDA, severe coarctation,), congenital mitral & tricuspid valve regurgitation, single ventricle physiology with unrestricted pulmonary blood flow

FAMILY HISTORY:

Congenital heart disease affecting a previous child or a parent increases the risk for structural heart disease in infants[16]. Sudden death is associated with many genetic diseases, including cardiomyopathy, prolonged QT interval, Brugada syndrome, Marphan syndrome, arrhythamogenic right ventricular dysplasia. Marphan syndrome is frequently associated with aortic aneurism or with aortic or mitral insufficiency, Holt oram syndrome (ASD & limb anomalies). PS secondary to dysplastic pulmonary valve is common in Noonan's syndrome. Lentigines, electrocardiogram abnormalities, ocular hypertelorism, pulmonary stenosis, abnormal genitalia, retardation of growth & deafness (LEOPARD syndrome) is often associated with PS & cardiomyopathy[12,17] .

INITIAL DETECTION OF MURMUR:

Knowledge of time a murmur was initially detected leads a clinician to consider different categories of cardiac disease. For neonates, murmur detected in first 6 hours of life typically involve valve regurgitation (tricuspid valve from perinatal stress, mitral valve from cardiac dysfunction) or valve stenosis, whereas murmurs detected after 6 hours of age can also represent shunt lesions that presents as pulmonary vascular resistance falls (e.g. ASD, VSD, PDA, peripheral pulmonary stenosis). Some neonates may only be examined by physician after 6 hours of age, & others may have both valve stenosis & septal defects (e.g. tetralogy of fallot) [13].

PHYSICAL EXAMINATION:

Inspection:

Much information can be gained by simple inspection without disturbing a sleeping infant. Inspection should include following: general appearance, any obvious syndrome or chromosomal abnormalities, color (i.e. cyanosis, pallor, jaundice), respiratory rate, dyspnea & retractions, sweat on forehead, chest inspection[18].

Cyanosis associated with arterial desaturation is called central cyanosis. Cyanosis associated with normal arterial saturation is called peripheral cyanosis. Even mild cyanosis in newborn requires thorough investigation. Peripheral cyanosis may be noticed in newborn who are exposed to cold & those with CHF because in both conditions peripheral blood flow is sluggish, losing more oxygen to peripheral tissues. Cyanosis is also noted in polycythemic patients with normal oxygen saturation[18].

Pallor may be seen in infants with vasoconstriction from CHF or circulatory shock or in severely anaemic infants. Newborns with severe CHF & those with congenital hypothyroidism may have prolonged physiologic jaundice. PDA & pulmonary stenosis are common in congenital hypothyroidism[18].

Most reliable **respiratory rate** is taken during sleep. Resting respiratory rate of more than 40 breaths/ minutes is unusual, & more than 60 breaths/ minutes is abnormal at any age. Tachypnea with tachycardia is the earliest sign of left sided heart failure. If the newborn also has dyspnea or retraction, it may be a sign of left sided heart failure or significant lung pathology[18].

Sweat on forehead:

Infants with CHF often have cold sweat on forehead. This is an expression of height-ended sympathetic activity as a compensatory mechanism for decreased cardiac output[18].

Palpation:

Peripheral pulses[18]:

1. The physician should count the pulse rate & note any irregularities in the rate & volume. Increased pulse rate may indicate excitement, fever, CHF, arrhythemia. Bradycardia may mean heart block, digitalis toxicity. Irregular pulse rate suggest arrhythmia, but sinus arrhythmia (acceleration with inspiration) is normal.
2. Right & left arm & an arm & leg should be compared for volume of pulse. Every patient should have palpable pedal pulses, either dorsalis pedis, tibialis posterior or both. Weak leg pulses & stronge arm pulses suggest coarctation of aorta (COA). If the right brachial pulse is stronger than left brachial, the cause may be COA occurring near the origin of left subclavian artery or supravalvular aortic stenosis (AS). A weaker right brachial pulse than the left suggest an aberrant right subclavian artery arising distal to coarctation.
3. Bounding pulses are often found in aortic run-off lesions such as PDA, aortic regurgitation (AR), large systemic arteriovenous fistula, and persistent truncus

arteriosus (rare). Pulses are bounding in premature infants because of lack of subcutaneous tissue & because many have PDA.

4. Weak thread pulses are found in cardiac failure or circulatory shock or in leg of patient with COA. A systemic to pulmonary artery shunt (either classic Blalock-Taussig shunt or modified Gore-Tex shunt) or subclavian flap angioplasty for repair of COA may result in absent or weak pulse in the arm affected by surgery. Arterial injuries resulting from previous cardiac catheterization may cause weak pulse in affected limb.

Apical impulse: Its location & diffuseness should be noted. In neonates apical impulse is in 4^{th} intercostal space just to left of midclavicular line. An apical impulse displaced laterally or downwards suggest cardiac enlargement[18].

Thrills are vibratory sensations that represent palpable manifestations of loud, harsh murmurs. Their presence & timing should be noted. Point of maximal impulse to check for estimation of chamber hypertrophy. With right ventricle (RV) predominance, the impulse is maximal at lower left sternal border or over the xiphoid process; with left ventricle (LV) predominance, the impulse is maximal at apex. Tapping suggest large volume & heaving suggest increased pressure[2, 13, 18] .

BLOOD PRESSURE MEASUREMENT:

While four extremity blood pressure are often obtained, with the rare exceptions of aortitis or aberrant origin of subclavian artery in patient with coarctation, obtaining blood pressure (BP) in right arm & one leg provides sufficient screening. The right arm is preferred because origin of left subclavian artery can be stenosed in some patient with co-arctation. Unless a femoral artery is injured in previous catheterization, pressure should be similar in both legs. The inflatable bladder should have a length sufficient to fully encircle circumference of extremity &

width to cover about 75% distance between joints on either end of portion of extremity around which the cuff is placed[13] .

There are several techniques to measure blood pressure, including palpation & Doppler method, each of which estimates systolic pressure. Auscultation which is technically difficult in infants. Another method, the flush technique, is rarely used & yields values closer to mean pressure[13].

AUSCULTATION:

Auscultation of heart requires more skill, it also provides more valuable information than other methods of heart examination. Bell type chest piece is better suited for detecting low frequency events, whereas diaphragm selectively picks up high frequency events. When bell is firmly pressed against chest wall it acts as diaphragm by filtering out low frequency sounds or murmurs & picking up high frequency events. One should not limit examination to the four traditional auscultatory areas. The entire precordium, as well as the sides & the back of the chest, should be explored with stethoscope. Systemic attention should be given to following aspect[18].

1. Heart rate & regularity
2. Heart sounds: intensity & quality of heart sounds, especially the second heart sound (S2), should be evaluated. Abnormalities of the first heart sound (S1) & third heart sound (S3) & presence of gallop rhythm or the forth sound (S4) should be noted.
3. Systolic & diastolic sounds: An ejection click in early systole provides a clue to aortic or pulmonary valve stenosis. A mid-systolic click provides important

clues to diagnosis of mitral valve prolapse. An opening snap in diastole (present in mitral stenosis) should be noted

4. Heart murmurs: heart murmurs should be evaluated in terms of intensity, timing (systolic or diastolic), location, transmission & quality.

HEART SOUNDS:

First heart sound: The S1 is associated with closure of mitral & tricuspid valves. It is best heard at apex or lower left sternal border. Splitting of S1 may be found in normal children, but it is infrequent. Abnormally wide splitting of S1 may be found in right bundle branch block (RBBB) or Ebstein's anomaly. Splitting of S1 should be differentiated from ejection click or S4[18].

Second heart sound: the S2 in upper left sternal border (i.e., pulmonary valve area) is of critical importance in pediatric cardiology. The S2 must be evaluated in terms of degree of splitting & intensity of pulmonary closure component of the second heart sound (P2) in relation to intensity of aortic closure component of the second heart sound (A2). Although best heard with diaphragm of stethoscope, both components are radially audible with bell[18].

Splitting of S2: in every normal child with exception of occasional newborns, two components of S2 should be audible in upper left sternal border. The first is A2, second is P2[18].

Normal splitting of S2: degree of splitting of S2 varies with respiration, increasing with inspiration decreasing or becoming single with expiration. Absence of splitting (i.e. single S2) or widely spilt S2 usually indicates abnormality[18].

Summary of abnormal S2[18]

Abnormal splitting:

Widely split & fixed S2

Volume overload (e.g. ASD, PAPVR)

Pressure overload (e.g. PS)

Electrical delay (e.g. RBBB)

Early aortic closure (e.g. MR)

Occasional normal child

Narrowly split S2

Pulmonary hypertension

AS

Single S2

Pulmonary hypertension

One semilunar valve (e.g. pulmonary atresia, aortic atresia, persistent truncus arteriosus)

P2 not audible (e.g. TGA, TOF, severe PS

Severe AS

Paradoxically split S2

Severe AS

LBBB, WPW syndrome

Abnormal intensity of P2:

Increased P2 (e.g. pulmonary hypertension)

Decreased P2 (e.g. severe PS, TOF, TS)

AS, aortic stenosis; ASD, atrial septal defect; LBBB, left bundle branch block; MR, mitral regurgitation; PAPVR, partial anomalous pulmonary venous return; PS, pulmonary stenosis; RBBB, right bundle branch block; TGA, transposition of great arteries; TOF, tetralogy of Fallot; TS, tricuspid stenosis; WPW, Wolf-Parkinsons- White

Third heart sound[18]:

The S3 is somewhat low-frequency sound in early diastole & is related to rapid filling of ventricles. It is best heard at apex or lower left sternal border. Loud S3 is abnormal & is audible in conditions with dilated ventricles & decreased ventricular compliance (e.g. large VSD, CHF).

Forth heart sound or atrial sound:

The S4 is relatively is relatively low frequency sound of late diastole & is rare in infants & children. When present it is always pathologic & is seen in conditions with decreased ventricular compliance or CHF. With tachycardia it forms a "Tennesse" gallop[18].

Gallop rhythm:

A gallop rhythm is rapid triple rhythm resulting from combination of a loud S3, with or without an S4, & tachycardia. It generally implies a pathologic condition & is commonly present in CHF[18].

HEART MURMURS:

Detection of heart murmur on routine examination may be a clue to presence of heart disease & offers possibility of early presymptomatic diagnosis. Auscultation is therefore part of routine neonatal examination[19]. Early referral of all babies with murmurs for definitive diagnosis is recommended. Absence of murmur does not exclude serious heart disease. If murmur is heard there is 54% chance of there being an underlying cardiac malformation[20]. Clinical presentation & deterioration may be sudden & some treatable defects may even cause death before diagnosis[21].[22] Each heart murmur must be analyzed in terms of intensity (grade 1 to 6), timing (systolic or diastolic), location, transmission, & quality[18].

Intensity[18]:

Intensity or loudness is graded from 1 to 6, based on recommendations of Samuel A. Levine in 1933

Grade 1: Barely audible

Grade 2: Soft, but easily audible

Grade 3: Moderately loud, but not accompanied by thrill

Grade 4: Louder & associated with thrill

Grade 5: Audible with stethoscope barely touching chest

Grade 6: Audible with stethoscope off the chest

The difference between grade 2 & 3 or grade 5 & 6 may be somewhat subjective. Intensity of murmur may be influenced by status of cardiac output. Thus, any factor that increases the cardiac output (e.g. fever, anemia, exercise, anxiety) intensifies any existing murmur or may even produce a murmur that is not audible at basal conditions[18].

Classification heart murmurs[18]:

Based on timing of heart murmur relation to S1 & S2, the heart murmur is classified as systolic, diastolic, or continuous murmur.

1. **Systolic murmurs**
2. **Diastolic murmurs**
3. **Continuous murmur**

Systolic murmurs[18]:

Most heart murmurs are systolic in timing in that they occur in between S1 & S2. Systolic murmurs were classified by Aubrey Leatham in 1958 into 2 subtypes according to time of onset: 1. Ejection type, 2. Regurgitant type.

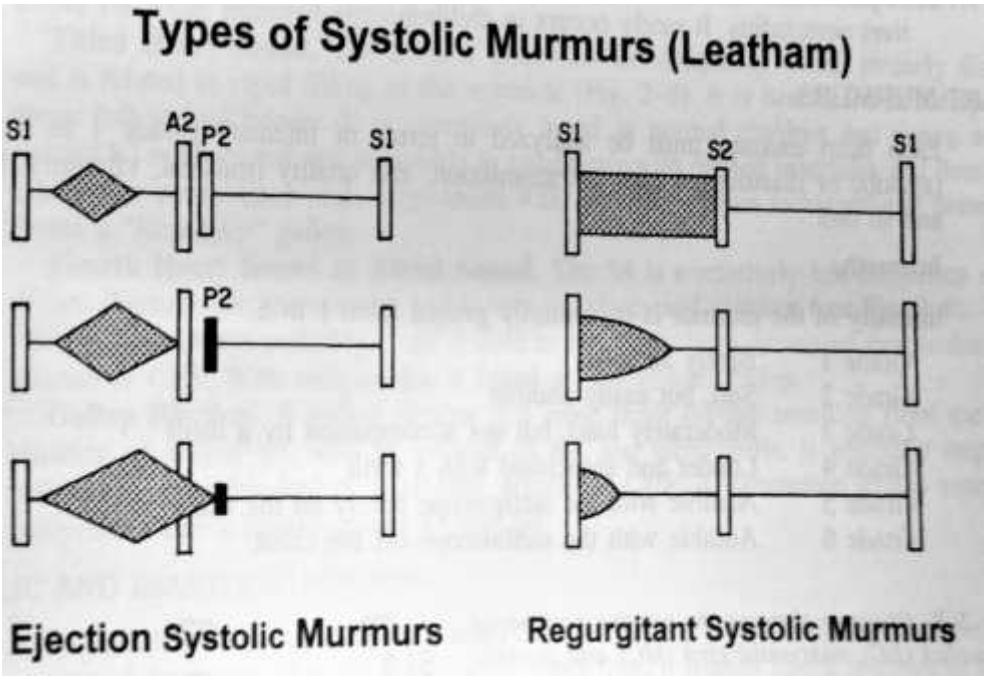
Joseph Perloff classified systolic murmurs according to their time of onset & termination into four subtypes: 1. Midsystolic (or ejection), 2. Holosystolic, 3. Early systolic, 4. Late systolic. The holosystolic murmur & early systolic murmur of Perloff is are same as regurgitant murmur of Leatham.

Midsystolic murmurs begin after S1 & end before S2. Midsystolic murmur coincides with turbulent flow through semilunar valves & occur in following setting: 1. Flow of blood through stenotic or deformed semilunar valves, such as seen during fever, anemia, and thyrotoxicosis. 3. Innocent murmurs. The intensity of murmur increases towards middle & then decreases during systole (crescendo-decrescendo or diamond shaped in contour). The murmur usually ends before S2.

Holosystolic murmurs, begin with S1 & occupy all of systole up to S2. No gap exists between S1 & onset of murmur. The intensity of holosystolic murmurs usually levels off all the way to the S2. Holosystolic murmurs are caused by the flow of blood from a chamber that is at a higher pressure throughout systole than receiving chamber, & they usually occur when semilunar valves are still closed. These murmurs are associated with only the following conditions: VSD, mitral regurgitation (MR), tricuspid regurgitation (TR).

Early systolic murmurs, begin with S1, diminish in decrescendo, & end well before S2, generally at or before midsystole.

The term late systolic murmur applies when a murmur begins in middle to late systole & proceeds up to S2. The late systolic murmur of mitral valve prolapse is prototypical.



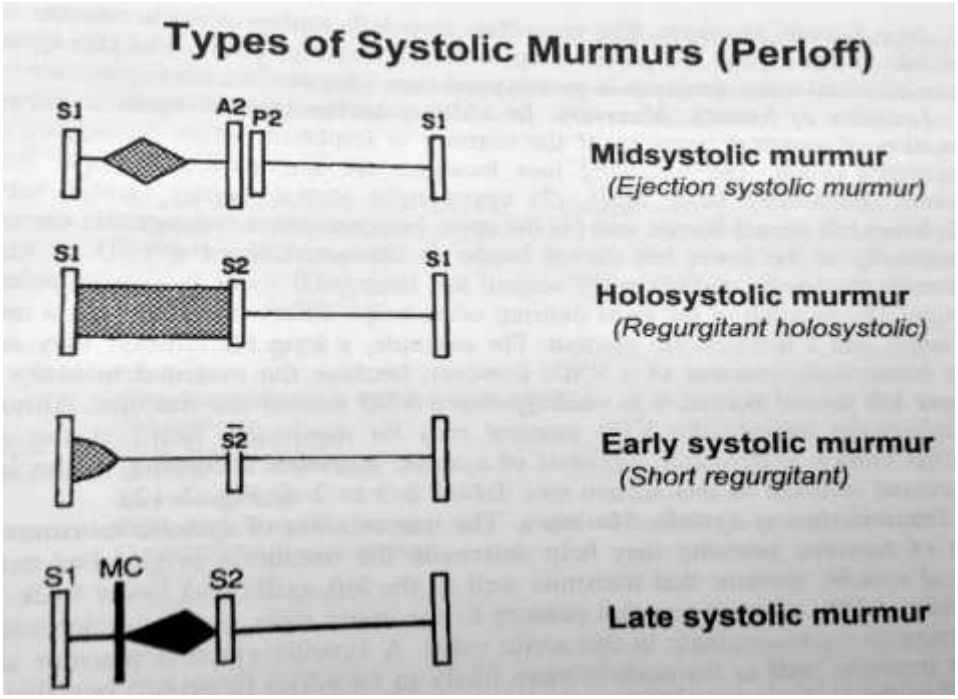


Diagram Showing Systolic Murmur[18]

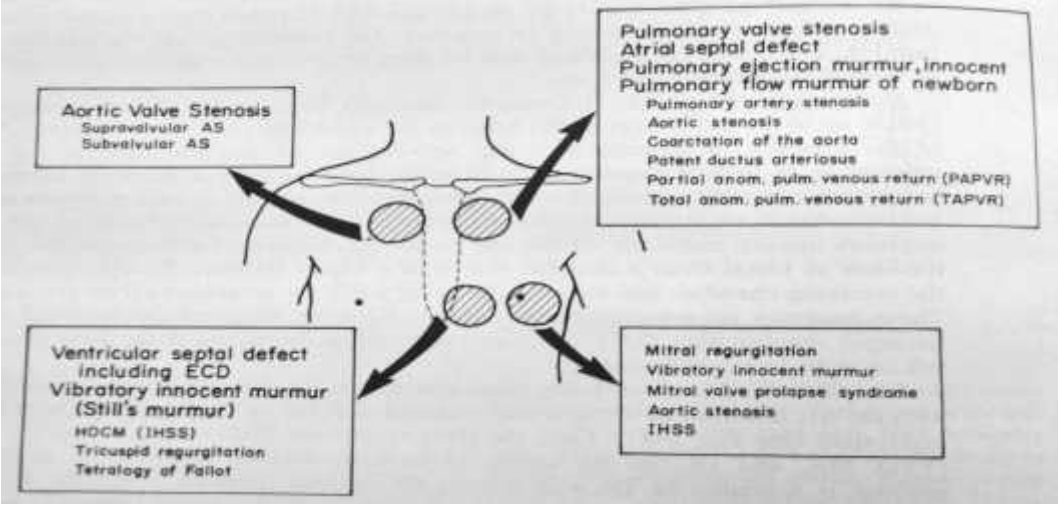


Diagram showing systolic murmur audible at different location. AS, aortic stenosis; ECD, endocardial cushion defects; HOCM, hypertrophic obstructive cardiomyopathy; IHSS,idiopathic hypertrophic subaortic stenosis[18]

Diastolic murmur[18]:

Diastolic murmurs occur between S2 & S1. Early diastolic decrescendo murmurs occur early in diastole, immediately after S2 & are caused by incompetence of aortic or pulmonary valve. Because aorta is high pressure vessel, aortic regurgitation (AR) murmur are high pitched & best heard with diaphragm of stethoscope at third left intercostal space. AR murmur radiates well to apex.

Pulmonary regurgitation (PR) murmur also occur early in diastole. They are usually medium pitched but may be high pitched if pulmonary hypertension is present. They are best heard at third left intercostal space & radiate along left sternal border.

Mid-diastolic murmurs start with a loud S3 & are heard in early or mid-diastole but are not temporally midway through diastole. These are always low pitched & best heard with bell of stethoscope applied lightly to chest. These murmurs are caused by turbulence in mitral or tricuspid flow secondary to anatomic stenosis or

relative stenosis of these valve. Mitral mid-diastolic murmurs are best heard at apex & are often referred to as an apical rumble, although they sound more frequently like hum than rumble. These murmurs are associated with mitral stenosis (MS) or a large left to right shunt VSD or PDA, which produces relative MS secondary to large flow across normal sized mitral valve. Tricuspid mid-diastolic murmurs are best heard along the lower left sternal border. These murmurs are associated with ASD, partial anomalous pulmonary venous return (PAPVR), total anomalous pulmonary venous return (TAPVR), because they all result in relative tricuspid stenosis. Presystolic (or late diastolic) murmurs are also caused by flow through the atrioventricular (AV) valve during ventricular diastole. These low frequency murmurs occur late in diastole or just before onset of systole & are found in anatomic stenosis of mitral or tricuspid valve.

Continuous murmur:

They begin in systole & continue without interruption through the S2 into all or part of diastole. Continuous murmurs are caused by following:

1. Aortopulmonary or arteriovenous connections (e.g., PDA, arteriovenous fistula, after systemic to pulmonary artery shunt surgery, persistent truncus arteriosus)
2. Disturbances of flow pattern in veins (e.g., venous hum)
3. Disturbance of flow pattern in arteries (e.g., coarctation of aorta, pulmonary artery stenosis)

The murmur of PDA has machinery like quality, becoming louder during systole, peaking at S2, & diminishing in diastole. This murmur is maximally heard in left infraclavicular area or along left sternal border. With pulmonary hypertension only systolic component can be heard, but it is cresendic during systole. Less common

continuous murmurs of severe coarctation of aorta may be heard over the intercostal collaterals. The continuous murmur of pulmonary artery (PA) stenosis may be heard over the right & left anterior chest, sides of chest & back[18].

NORMAL OR INNOCENT MURMURS:

Murmurs that occur in absence of either morphologic or physiologic abnormalities of heart or circulation have been called normal, innocent, functional, physiologic, or benign[23].

Seven types of normal systolic murmurs & three types of normal continuous murmurs are known[24].

NORMAL MURMURS:

a. Systolic:

1. The vibratory systolic murmur of Still
2. The pulmonary artery systolic murmur
3. The branch pulmonary artery systolic murmur
4. The supraclavicular systolic murmur
5. The systolic mammary soufflé
6. The aortic sclerotic systolic murmur
7. The cardiorespiratory systolic murmur

b. Continuous:

1. The venous hum
2. The continuous mammary soufflé
3. The cephalic continuous murmur

Normal murmurs are never solely diastolic, with one exception- the transient left basal holodiastolic or mid-diastolic ductus arteriosus murmur, sometimes heard

during first 3 or 4 days of life. A valve like structure at pulmonary arterial end of ductus is responsible for selective diastolic flow[23].

VIBRATORY SYSTOLIC MURMUR:

It was described by George F. Still in 1909. The murmur ranges from grade 1 to 3/6 & is loudest between apex and lower left sternal edge in supine position. The quality is distinctive. Vibratory or buzzing with a uniform, medium, pure frequency that requires stethoscope bell for best assessment. The murmur begins shortly after first heart sound & is typically confined to first half of systole with relatively long gap between end of murmur & second heart sound. [23, 25, 26, 27]

PULMONARY ARTERY SYSTOLIC MURMUR:

The murmur is mid systolic with maximum intensity in second left intercostal space next to sternum. Best heard in supine position with stethoscope diaphragm or moderate pressure of bell during full held exhalation[23, 25, 26, 27]

PULMONARY FLOW MURMUR OF NEWBORNS:

This murmur is commonly present in newborns especially those low birth weight. The murmur usually disappears by 3-6 months of age. If it persists beyond this age, a structural narrowing of pulmonary arterial tree should be suspected. It is best audible at upper left sternal border. Although the murmur is only grade 1 to 2/6 in intensity, it transmits impressively to right & left chest, both axilla, & back

VENOUS HUM:

Originates from turbulence in jugular venous system. This is continuous murmur in which diastolic component is louder than systolic. Murmur is maximally audible at right & or left infraclavicular & supraclavicular areas. Heard only in upright position, disappears in supine position. It can be obliterated by rotating the head or gently occluding the neck veins by fingers[18].

SUPRACLAVICULAR SYSTOLIC MURMUR:

Early systolic ejection murmur, best heard in supraclavicular fossa or over carotids. Produced by turbulence in brachiocephalic or carotid arteries. This bruit can be heard in children of any age[18].

TRANSIENT SYSTOLIC MURMUR OF TRICUSPID REGURGITATION:

It is indistinguishable from that of VSD, in that it is regurgitant & is maximally audible at lower left sternal border. It disappears in a day or two. It is believed that minimal tricuspid valve abnormality produces regurgitation in presence of high pulmonary vascular resistance, but regurgitation disappears as pulmonary vascular resistance falls. Therefore, this murmur is more common in infants who had fetal distress or neonatal asphyxia, because they tend to maintain high pulmonary vascular resistance for longer period [18].

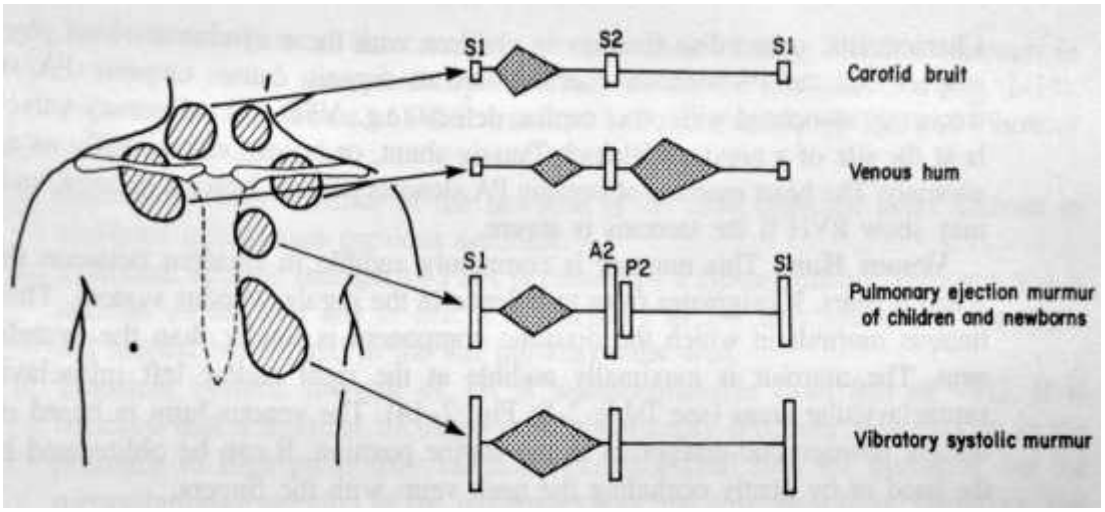


Diagram of innocent heart murmurs[18].

SPECIAL FEATURES IN CARDIAC EVALUATION OF NEWBORN:

Newborn infants have right ventricular (RV) dominance with a thick RV wall & elevated pulmonary vascular resistance secondary to a thick medial layer of pulmonary arterioles. Thick pulmonary artery smooth muscle gradually becomes thinner & by 6-8 weeks of age resembles that of adults. Most perinatal changes in

hemodynamics are related to normal evolution (thinning) of pulmonary vascular smooth muscle, resulting in a gradual fall in pulmonary vascular resistance & loss of RV dominance. Premature infants in general have less RV dominance & lower pulmonary vascular resistance than do full term neonates, adding variability to this generalization [2, 13].

Normal physical findings in neonates[18]:

1. The heart rate is generally is faster in newborns than older children & adults. (the newborn rate is usually 100 beats/minute, with a normal range of 70-180 beats/minute
2. A varying degree of acrocyanosis is the rule than exception.
3. Mild arterial desaturation with arterial partial pressure of oxygen (Po_2) as low as 60 mm of Hg is not unusual in an otherwise normal neonate. This may be caused by an intrapulmonary shunt through an as yet unexpanded portion of lungs or by a right atrium-to-left atrium shunt through a patent foramen ovale.
4. The right ventricle (RV) is relatively hyperactive, with point of maximal impulse at lower left sternal border rather than at apex.
5. S 2 may be single in first days of life.
6. An ejection click (representing pulmonary hypertension) is occasionally heard in first hours of life.
7. Newborn may have an innocent heart murmur. Four common innocent murmurs in newborn are:
    a. Pulmonary flow murmur of newborn is most common heart murmur in newborn.

b. Transient systolic murmur of PDA is caused by a closing ductus arteriosus & is audible on 1st day of life. It is grade 1 to 2/6, only systolic, at uppar left sternal border & in left infraclavicular area.

c. Transient systolic murmur of tricuspid regurgitation (TR) is indistinguishable from that of VSD. This murmur is more common in infant who had fetal distress or neonatal asphyxia because they tend to maintain high pulmonary vascular resistance for longer period

d. Vibratory innocent murmur is counterpart of Still's murmur in older children

Abnormal physical findings in neonates[18]:

1. Cyanosis, particularly when it does not improve with administration of oxygen, suggest a cardiac abnormality.
2. Decreased or absent peripheral pulses in lower extrimities suggest coarctation of aorta (COA). Weak peripheral pulses throughout suggest hypoplastic left heart syndrome (HLHS) or circulatory shock. Bounding peripheral pulses suggest aortic run off lesion, such as PDA or persistent truncus arteriosus.
3. Tachypnea of greater than 60 breaths/minute with or without retractions suggest cardiac abnormality
4. Hepatomegaly may suggest defect. A midline liver suggest asplenia or polysplenia syndrome
5. A heart murmur may be a presenting sign of congenital heart defect, time of appearance of heart murmur depends on nature of defect.
   a. Heart murmurs of stenotic lesions & those due to atrioventricular (AV) valve regurgitation are audible immediately after birth & persist because these murmurs are not affected by pulmonary vascular resistance.

b. Heart murmurs of large VSD may not be audible until 1 to 2 weeks of age, when pulmonary vascular resistance becomes sufficiently low to allow shunt to occur.

c. Murmur of an ASD appears after a year or two, when compliance of right ventricle (RV) improves to allow significant atrial shunt.

6. Even in absence of heart murmur newborn may have serious heart defect that requires immediate attention (severe cyanotic heart defect such as transposition of great vessels or pulmonary atresia with a closing PDA). Infants who are in severe congestive heart failure (CHF) may not have a loud murmur until myocardial function is improved through anticongestive measures
7. Irregular cardiac rhythm & abnormal heart rate suggest cardiac abnormality.

TOOLS IN DIAGNOSIS OF CONGENITAL HEART DISEASES:

A complete history & physical examination enables the physician to compile an appropriate differential diagnosis, order tests in suitable manner, and efficiently care for patient. The physical examination needs to be complete because heart disease can affect multiple organ systems. Most murmurs are detected as part of clinical examination[27]. Adding a chest x-ray & electrocardiography (ECG) to pediatrician's evaluation increases cost with little gain in accuracy[28].

Electrocardiography:

It is still the quickest, safest, least expensive diagnostic tool in cardiology & is unparalleled in its ability to register arrhythmias & conduction defects. With proper interpretation, ECG also offers a useful reflection of cardiac position, chamber enlargement, and myocardial damage[29]. The insensitivity of electrocardiogram severely limits its effectiveness as a screen[30, 31, 32] & poor specificity as shown by Sharma et al [33]. ECG rarely adds to clinical evaluation of an asymptomatic heart murmur. It rarely leads to change in diagnosis

Chest roentgenography:

The following information can be gained from x-ray films: heart size & silhouette; enlargement of specific cardiac chambers, pulmonary blood flow or pulmonary vascular markings & other information regarding lung parenchyma, spine, bony thorax, and abdominal situs. Posteroanterior & lateral views are routinely obtained[34]. The chest X-ray is inexpensive & easily available & so has been the focus of investigation as a screening test for congenital heart disease but classic x-ray findings for congenital heart diseases, however appear late in the clinical course. Chest X-ray examination is often misleading in evaluation of an asymptomatic heart murmur & interpretation is poorly to moderately reproducible[35, 36] .

Abnormal cardiac silhouette[34]:

1. A “boot shaped heart” with decreased pulmonary blood flow is typically seen in infants with cyanotic tetralogy of Fallot (TOF). This is also seen in some infants with tricuspid atresia. Typical of both condition is the presence of the hypoplastic main PA segment.
2. A narrow wasted & “egg shaped” heart with increased pulmonary blood flow in a cyanotic infant strongly suggests transposition of great arteries. The narrow waist results from the absence of large thymus & abnormal relationship of great arteries.
3. The “snowman” with increased pulmonary blood flow is seen in infants with the supracardiac type of total anomalous pulmonary venous return (TAPVR). The left vertical vein, left innominate vein, & dilated SVC make up the snowman’s head.

   In heavily exposed film, the precoarctation & post coarctation dilatation of aorta may be seen as a “figure of 3”. This may be confirmed by barium esophagogram with E shaped indentation.

Pulmonary vascular markings[34]:

One of the major goal of radiologic examination is assessment of pulmonary vasculature

INCREASED PULMONARY BLOOD FLOW:

Increased pulmonary vascularity is present when the right & left PAs appear enlarged & extend into the lateral third of lung field. There is increased vascularity to lung apices where the vessels are normally collapsed. Increased pulmonary blood flow in an acyanotic child represents ASD, VSD, PDA, partial anomalous pulmonary venous return (PAPVR) or any combination of these. In cyanotic infant, increased

pulmonary vascular markings may indicate TGA, TAPVR, hypoplastic left heart syndrome (HLHS), persistent truncus arteriosus, or a single ventricle[34].

DECREASED PULMONARY BLOOD FLOW:

It is suspected when hilum appears small, remaining lung fields appears black, & vessels appears small & thin. Ischemic lung fields are seen in cyanotic heart disease with decreased pulmonary blood flow such as critical stenosis or atresia of pulmonary or tricuspid valve, including TOF[34].

Echocardiography[37]:

Echocardiography (ECHO) is application of ultrasound to the evaluation of the cardiovascular system. It is extremely useful, safe, noninvasive test used for diagnosis & management of heart disease. Echo studies, provide anatomic diagnosis as well as functional information. This is especially true with incorporation of Doppler echo & color flow mapping.

The M- mode echo provides an “ice pick” view of heart. It has limited capability in demonstrating the spatial relationship of structures but remains an important tool in evaluation of certain cardiac conditions & functions, particularly by measurement of dimensions & timing. It is usually performed as part of two dimensional echo studies. The two dimensional echo has an enhanced ability to demonstrate the spatial relationship of structures. This capability allows a more accurate anatomic diagnosis of abnormalities of heart & great vessels. The Doppler & color mapping study has added ability to detect easily valve regurgitation & cardiac shunts during the echo examination. It also provides some quantitative information such as pressure gradients across cardiac valves & estimation of pressure in great arteries & ventricles. Echo examination can be applied in calculation of cardiac output & magnitude of cardiac shunts[37].

M-MODE ECHOCARDIOGRAPHY:

M-mode echo is obtained with ultrasonic transducer placed along the left sternal border & directed towards the part of heart to be examined.

Although the two dimensional echo has largely replaced the M-mode echo in diagnosis of cardiac diseases, M-mode echo retains many important applications including the following[37]:

1. Measurement of dimensions of cardiac chambers & vessels, thickness of ventricular septum & free walls
2. Left ventricular systolic function
3. Study of motion of cardiac valves (e.g. mitral valve prolapse, mitral stenosis, pulmonary hypertension) & interventricular septum
4. Detection of pericardial fluid.

TWO DIMENSIONAL ECHOCARDIOGRAPHY:

Two dimensional echo examinations are performed by directing the plane of transducer beam along a number of cross-sectional planes through the heart & great vessels. A routine two dimensional echo is obtained from four transducer locations: parasternal, apical, subcostal, & suprasternal notch positions. Important cardiac structure can be measured on freeze frame of two dimensional echo studies[37].

DOPPLER ECHOCARDIOGRAPHY:

The Doppler echo combines the study of cardiac structure & blood flow profiles. The Doppler effect is a change in observed frequency of sound that results from motion of source or target. When moving object or column of blood moves towards the ultrasonic transducer, frequency of reflected sound wave increases (i.e., positive Doppler shift). Conversely when blood moves away from transducer, the frequency decreases (i.e., negative Doppler shift). Doppler ultrasound equipment

detects a frequency shift & determines the direction & velocity of red blood cell flow with respect to ultrasound beam[37].

The two commonly used Doppler techniques are continuous wave & pulsed wave. The pulsed wave emits a short burst of ultrasound, & Doppler echo receiver "listens" for returning information. Continuous wave emits a constant ultrasound beam with one crystal, & another crystal continuously receives returning information[37].

The Doppler echo technique is useful in studying the direction of blood flow; in detecting the presence & direction of cardiac shunts; in studying stenosis or regurgitation of cardiac valves, including prosthetic valves; in assessing stenosis of blood vessels; in assessing hemodynamic severity of a lesion including pressures in various compartments of cardiovascular system; in estimating cardiac output or blood flow; & in assessing diastolic function of ventricles. The Doppler echo is usually used with color flow mapping to enhance technique's usefulness[37].

Cardiac catheterization & angiography:

Cardiac catheterization & angiography usually constitute the final definitive diagnostic test for most cardiac patients.

INDICATIONS:

Indications for these invasive studies vary from institution to institution & from cardiologist to cardiologist. The following are considered indications by most cardiologists:

1. Selected newborns with cyanotic congenital heart disease (CHD) who may require palliative surgery or balloon atrial septostomy during the procedure
2. Selected children with CHD when the lesion is severe enough to require surgical intervention

3. Children who appear to have had unsatisfactory results from cardiac surgery
4. Infants & children with lesions amenable to balloon angioplasty or valvuloplasty
5. Children with hypoplastic or stenotic pulmonary arteries, especially those associated with pulmonary atresia & TOF, extensive collateral arteries require angiography to delineate the extent of abnormalities. (Echo is not useful in studying blood vessels within the lung parenchyma)[37].

ECHOCARDIOGRAPHIC FINDINGS OF CONGENITAL HEART DISEASES:

1. **ATRIAL SEPTAL DEFECT (ASD)[38]:**

1. A two dimensional echo study is diagnostic. The study shows the position as well as size of defect, which can be best seen in subcostal four chamber view. In secundum ASD, a dropout can be seen in mid atrial septum. Primum type shows a defect in lower atrial septum; SVC type of sinus venosus defect shows a defect in posterosuperior atrial septum
2. Indirect signs of significant left-to-right atrial shunt include RV enlargement & RA enlargement, as well as dilated PA, which often accompanies an increase in flow velocity across pulmonary valve. These findings indicate functional significance of defect
3. Pulsed Doppler examination reveals a characteristic flow pattern with maximum left to right shunt occurring in diastole. Color flow mapping enhances the evaluation of hemodynamic status of ASD.
4. M-mode echo may show increased RV dimension & paradoxical motion of interventricular septum, which are signs of RV volume overload[38].

2. VENTRICULAR SEPTAL DEFECT[38]:

1. Two dimensional echo & Doppler studies can identify the number, size, & exact location of defect; estimate PA pressure using modified Bernoulli equation; identify other associated defects; estimate the magnitude of shunt
2. Cardiac valves serve as marker of specific types of VSD except for trabecular septum. The membranous VSD is closely related to aortic valve, inlet VSD to tricuspid valve, & infundibular VSD to semilunar valve. Collection of selected parasternal, apical, subcostal views that are useful in locating site of VSD
3. The membranous septum is closely related to aortic valve. In apical & subcostal five chamber views, it is seen in LV outflow tract just under aortic valve. In parasternal short axis view at level of aortic valve, it is seen adjacent to tricuspid valve. These are best views to confirm membranous VSD.
4. Simple inlet VSD is seen is seen beneath AV valve, but small amount of tissue remains under the valve. In AV canal type of VSD, AV valves are at same level. The subpulmonary, suprscristal infundibular VSD lies under aortic valve[38].

3. PATENT DUCTUS ARTERIOSUS (PDA)[38]:

1. Its size can be assessed by two dimensional echo in high parasternal view or in suprasternal notch
2. Doppler studies that are performed with sample volume in PA immediately proximate to ductal opening provide important functional information
3. The dimension of LA & LV provide an indirect assessment of magnitude of left-to-right ductal shunt. The larger the shunt, greater the dilatation of these chamber[38].

4. COMPLETE AV CANAL DEFECT:

1. Apical & subcostal four chamber views are most useful in evaluating the anatomy & functional significance of defect. These views show both an ostium primum ASD & an inlet muscular VSD. The full extent of ASD & VSD can be imaged during systole when common anterior leaflet is closed
2. Combined use of subcostal transducer & parasternal short axis examination may show a cleft in mitral valve, presence of bridging leaflets, number of AV valve orifices. These views may also image abnormal position of anterolateral papillary muscle.
3. The subcostal five chamber view may image a goose neck deformity, which is characteristic of an angiocardiographic finding[38].

5. TRANSPOSITION OF GREAT ARTERIES:

1. In parasternal long axis view, great artery arising from posterior ventricle has sharp posterior angulation towards lung, which suggests that this artery is PA. In contrast to normal intertwining of great arteries, the proximal portion of great arteries run parallel
2. In parasternal short axis view, the “circle & sausage appearance” of great arteries is not visible. Instead, the great arteries appear as “double circles”. The PA is in center of heart & coronary arteries do not arise from this great artery. The aorta is usually anterior & slightly to the right of PA, the coronary arteries arise from aorta.
3. In apical & subcostal five chamber views, the PA arise from LV, & aorta arise from RV.

4. The status of atrial communication both before & after balloon septostomy, is best evaluated in subcostal view. Doppler examination & color flow mapping should aid in functional evaluation of atrial shunt
5. Frequently associated defects such as VSD, left ventricular outflow tract (LVOT) obstruction or pulmonary valve stenosis are found. Subaortic stenosis or COA rarely occurs.
6. The coronary arteries can be imaged in most patients by using parasternal & apical views[39]

6. SINGLE VENTRICLE:

1. The most important diagnostic sign is the presence of a single ventricular chamber into which 2 AV valves open
2. The following anatomic & functional information is important from a surgical point of view:

a. Morphology of single ventricle (e.g. double inlet LV , double inlet RV)
b. Location of rudimentary outflow chamber, which is usually left & anterior
c. Size of bulboventricular foramen & whether there is obstruction at foramen
d. Presence or absence of D-TGA or L-TGA, stenosis of pulmonary or aortic valve size of PAs
e. Anatomy of AV valves. The position of mitral & tricuspid valve in addition to stenosis, regurgitation, hypoplasia, or straddling of these valves should be checked
f. Size of ASD
g. Associated defects such as COA, interrupted aortic arch, PDA[39].

METHODOLOGY

Materials and Methods:

It is a prospective study. Clinical and echocardiographic study of neonates with suspected heart disease, admitted in NICU & postnatal wards of Shri B. M. Patil medical college, Hospital and research Centre, Bijapur

Source of data:

- All neonates with heart murmurs admitted to NICU & post natal wards
- Neonates presenting with signs and symptoms of CCF(congestive cardiac failure) or cardiogenic shock
- Neonates with PPHN were excluded from study.

Duration of study- 1 Year

1. **Methods of collection of Data:**

After taking written informed consent from the parents and fulfilling inclusion and exclusion criteria of neonates were included in the study.

Method of study:

- All neonates admitted to NICU & Post natal wards were evaluated for presence of murmur & any other evidence of CHD
- Neonates were examined 48 hours after delivery and before discharge
- Detailed clinical examination of each neonate was done including assessment of gestational age, anthropometry.
- Detailed CVS(Cardiovascular system) examination was done in each case for presence or absence of murmur & the same will be noted.
- All peripheral pulses were palpated

- Murmur was classified as organic or functional. Signs of CCF if present will also be noted. Other systems will also be examined.
- All neonates with murmurs & those suspected to have CHD were subjected to detailed echocardiographic, color Doppler investigation & any other investigation needed.
- Heart diseases were classified according to clinical evaluation& echocardiography and will be tabulated & interpreted accordingly.
- Statistical analysis of the data was done by appropriate formula.

Sample size;

Determination of sample size (n)

$$n = \frac{(1.96)^2 \times p(1-p)}{d^2}$$

$$n = \frac{(1.96)^2 \times 0.267 \times 0.733}{0.14066^2}$$

$$n = 38$$

p= prevalence (p= 0.267 with reference to article by, Bansal M, Jain H.Cardiac murmur in neonates. Indian Pediatrics.2005; 42(4):397-398)

d=Permissible error

Hence 38 cases were included in study.

RESULTS & OBSERVATIONS

A total of 74 neonates were included in our study. Neonates were admitted in NICU & post natal wards of Shri B. M. Patil medical college hospital & research Centre, Bijapur. Study was conducted over a period of 12 months. Total number of neonates admitted were 2091 during study period. Out of them 74 were having murmur & 52 babies were having congenital heart disease as diagnosed by echo-cardiography. The incidence of cardiac murmur was 35.38 per 1000 admissions & incidence of congenital heart disease was 24.86 per 1000 admissions. 8 cases of PPHN were found and were excluded from the study.

TABLE 1: TOTAL CASES

	CASES	PERCENTAGE (%)
CHD	52	70
NORMAL	22	30
TOTAL	74	100

In our study total 74 patients were included, out of them 22 (30%) cases were normal & 52 (70%) cases were having CHD.

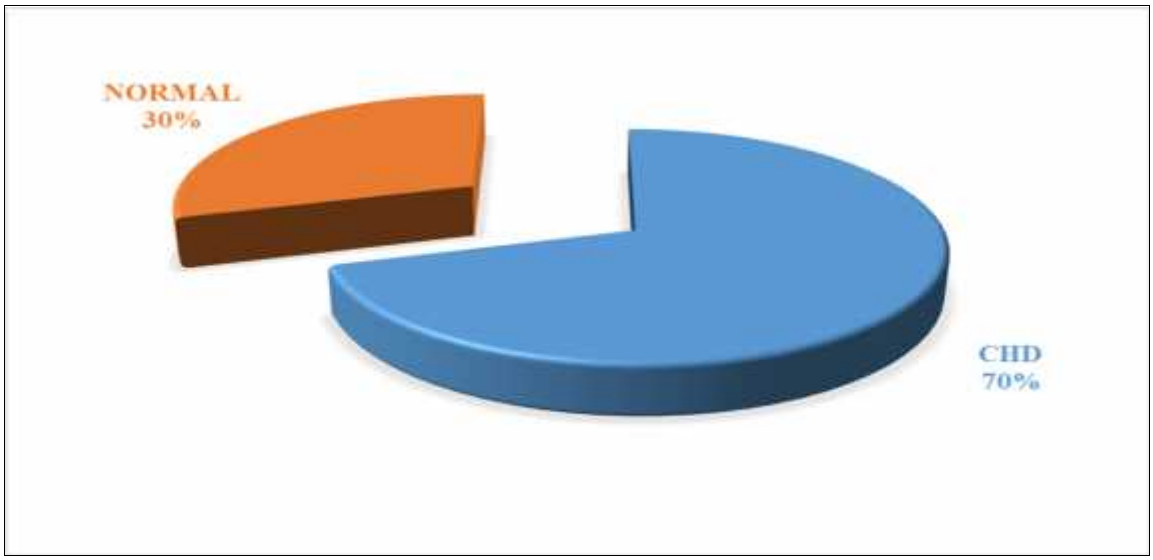


FIGURE 1: CASES

Table 2: SEX DISTRIBUTION

	CASES	PERCENTAGE(%)
MALE	27	51.92
FEMALE	25	48.08
TOTAL	52	100

In our study total 52 patients were having congenital heart disease, out of them 27 (51.92%) cases were male & 25 (48.08%) cases were female.

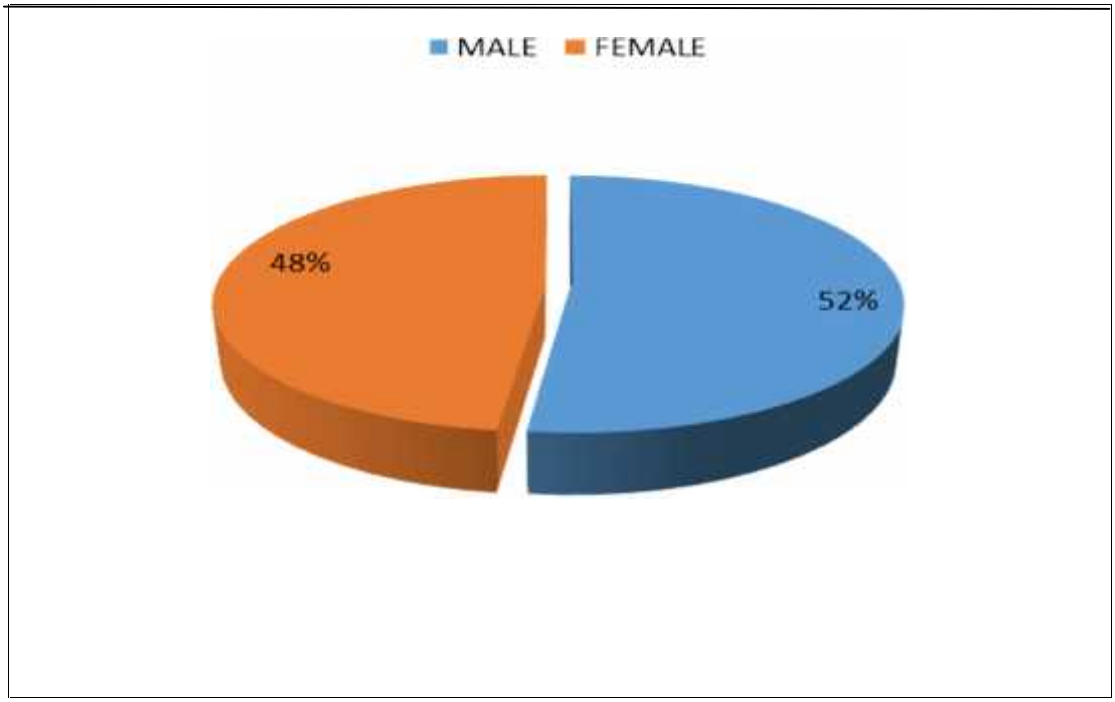


FIGURE 2: SEX DISTRIBUTION

TABLE 3: DISTRIBUTION BY CONSANGUINITY

	CASES	PERCENTAGE (%)
CONSANGUINEOUS	15	28.85
NON CONSANGUINEOUS	37	71.15
TOTAL	52	100

In our study total 52 patients were having congenital heart disease, out of them 15 (28.85%) cases were having a history of consanguineous marriage by parents & 37 (71.15%) cases were having non consanguineous marriage by parents.

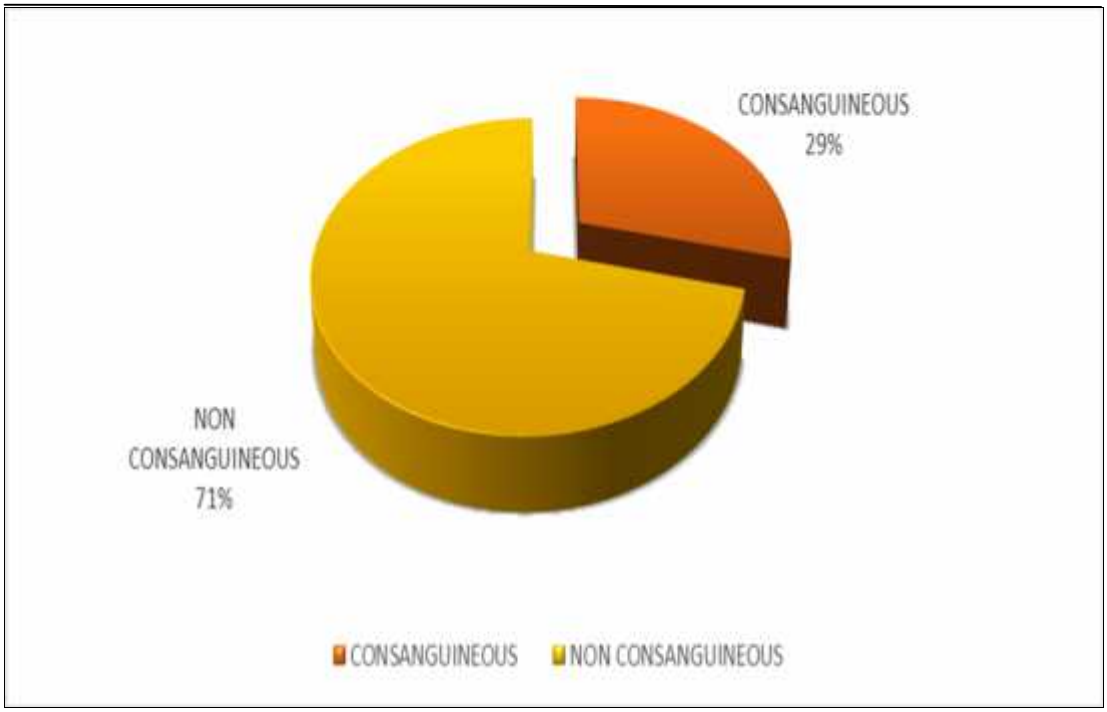


FIGURE 3: DISTRIBUTION BY CONSANGUINITY

TABLE 4: DISTRIBUTION BY GESTATIONAL AGE

GESTATIONAL AGE	CASES	PERCENTAGE (%)
PRETERM	28	53.85
TERM	24	46.15
TOTAL	52	100

In our study total 52 patients were having congenital heart disease, out of them 28 (53.85%) cases were preterm & 24(46.15%) cases were term.

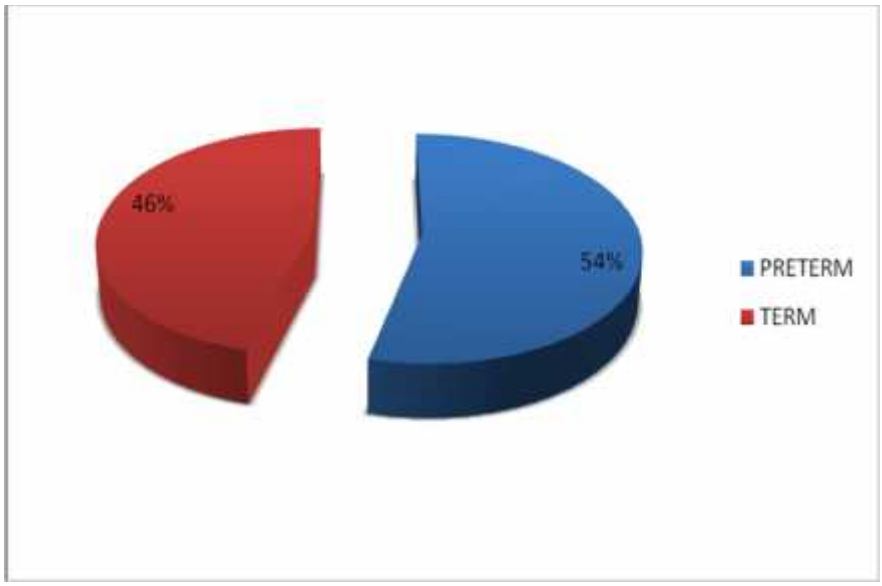


FIGURE 4: DISTRIBUTION BY GESTATIONAL AGE

TABLE 5: DISTRIBUTION BY WEIGHT

WEIGHT	CASES	PERCENTAGE (%)
SGA	24	46.15
AGA	27	51.92
LGA	1	1.92
TOTAL	52	100

In our study total 52 patients were having congenital heart disease, out of them 24 (46.15%) cases were SGA, 27(51.92%) cases were AGA & 1 (1.92%) case was LGA.

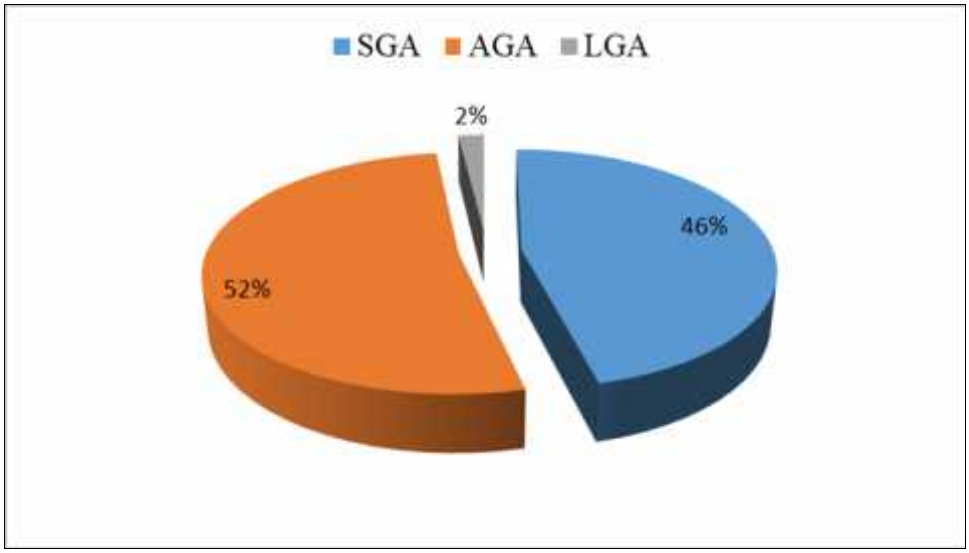


FIGURE 5: DISTRIBUTION BY WEIGHT

TABLE 6: ECHOCARDIOGRAPHIC DIAGNOSIS OF CHD

CHD	CASES	PERCENTAGE (%)
ASD	16	30.77
ASD +PDA	15	28.85
VSD	5	9.62
PDA	9	17.31
ASD+VSD	3	5.77
ASD+VSD+PDA	1	1.92
CYANOTIC	3	5.77
TOTAL	52	100

In our study total 52 patients were having congenital heart disease, out of them 16 (30.77%) cases were ASD, 19(36.53%) cases were having multiple defects, 9 (17.31%) cases were having PDA, 5 (9.62%) cases were having VSD & 3 (5.77%) cases were having cyanotic CHD.

Here, multiple defects includes cases of ASD +PDA, ASD+VSD, ASD+VSD+PDA.

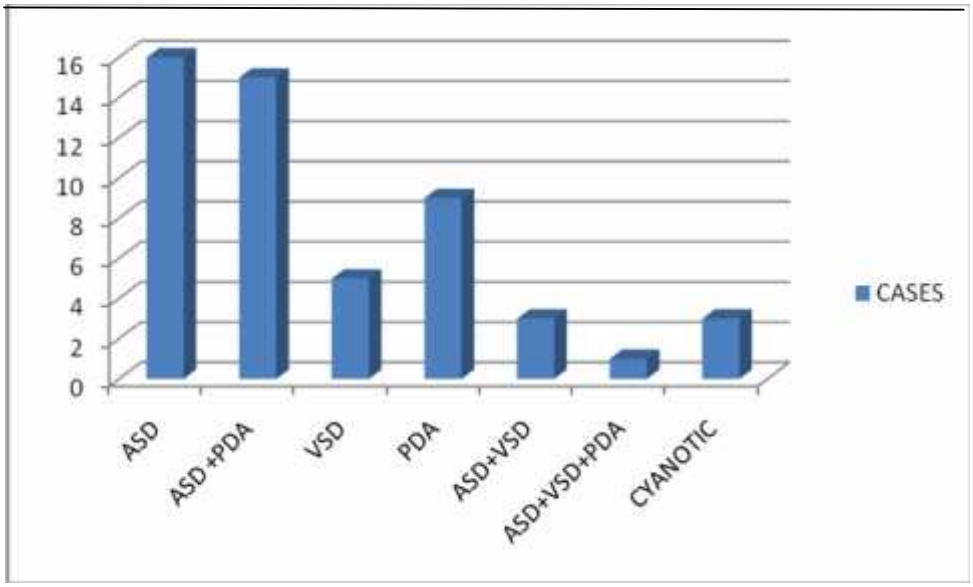


FIGURE 6: ECHOCARDIOGRAPHIC DIAGNOSIS OF CHD

TABLE 7: MATERNAL HISTORY

MATERNAL HISTORY	CASES	PERCENTAGE (%)
INTRA-UTERINE INFECTION	3	20
GESTATIONAL DIABETES MELLITUS	5	33.33
POLYHYDRAMNIOUS	3	20
OLIGOHYDRAMNIOUS	4	26.66
TOTAL	15	100

In our study frequently associated maternal history was gestational diabetes mellitus, 5 (33.33%) cases, followed by other conditions like oligohydramnious 4 (26.66%) cases, 3 (20%) cases of each intra-uterine infection & polyhydramnious.

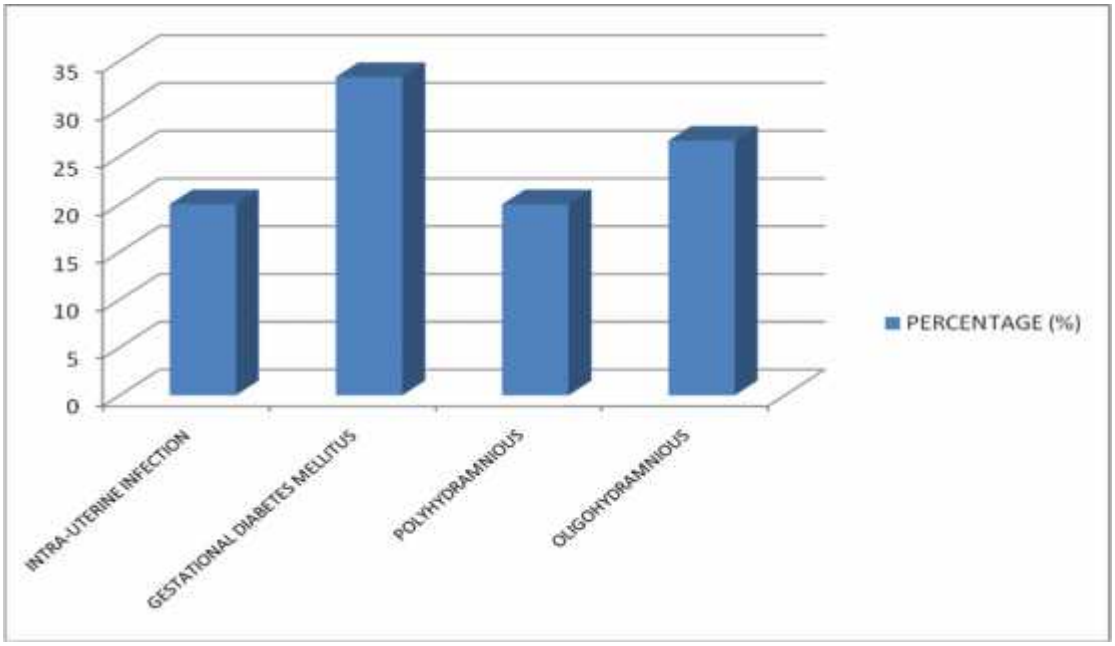


FIGURE 7: MATERNAL HISTORY

TABLE NO 8: SYMPTOMS

SYMPTOMS	CASES	PERCENTAGE (%)
CYANOSIS	3	11.53846
FAST BREATHING	10	38.46154
FEEDING DIFICULTY	12	46.15385
SWEATING	1	3.846154
TOTAL	26	100

Most frequent symptom which was observed in cases was feeding difficulty 12 (46.15%) cases, followed by fast breathing 10 (38.46%) cases.

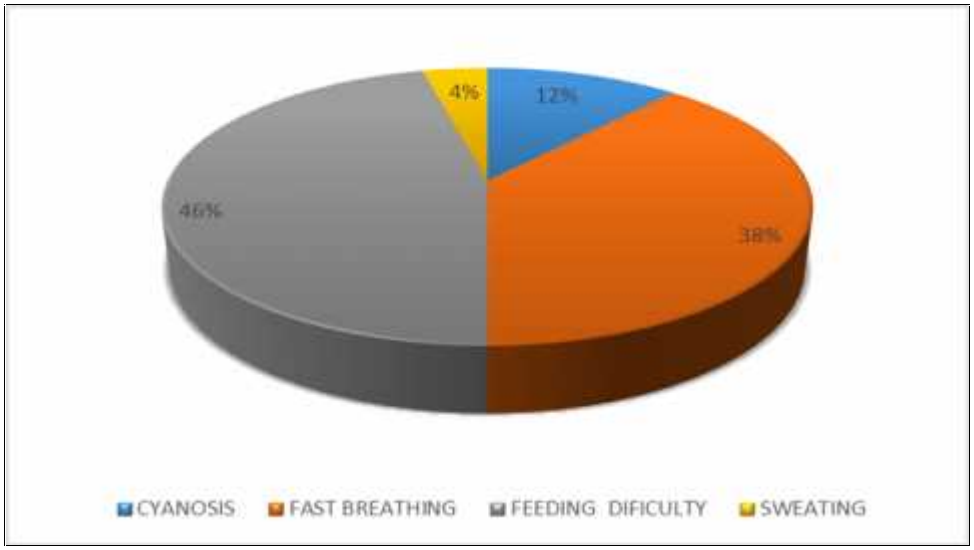


FIGURE 8: SYMPTOMS

TABLE NO 9: CLINICAL EXAMINATION FINDINGS

CLINICAL EXAMINATION FINDINGS	CASES	PERCENTAGE (%)
CONGENITAL MALFORMATION	5	13.89
CENTRAL CYANOSIS	3	8.33
PALLOR	4	11.11
EDEMA	1	2.78
DIAPHORESIS	1	2.78
RESPIRATORY DISTRESS	12	33.33
BOUNDING PULSES	10	27.78

In our study most common clinical examination finding found was respiratory distress, 12 (33.33%) cases followed by 10 (27.78%) cases of bounding pulses

Congenital malformations were seen in 5 (13.89%) cases.

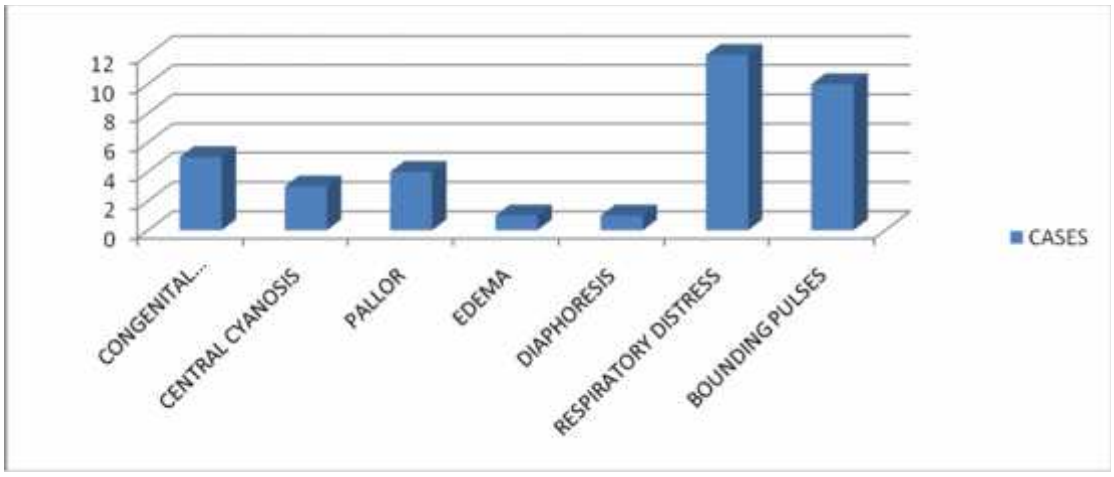


FIGURE 9: CLINICAL EXAMINATION FINDINGS

TABLE NO 10: LOCATION OF MURMUR

LOCATION OF MURMUR	CASES	PERCENTAGE (%)
LOWER LEFT STERNAL BORDER	38	50.67
UPPER LEFT STERNAL BORDER	35	46.67
APEX	1	1.33
TOTAL	74	100

In our study, we found that most common location of murmur was lower left sternal border 38 (50.67%) cases followed by 35 (46.67%) cases with location of murmur at upper left sternal border.

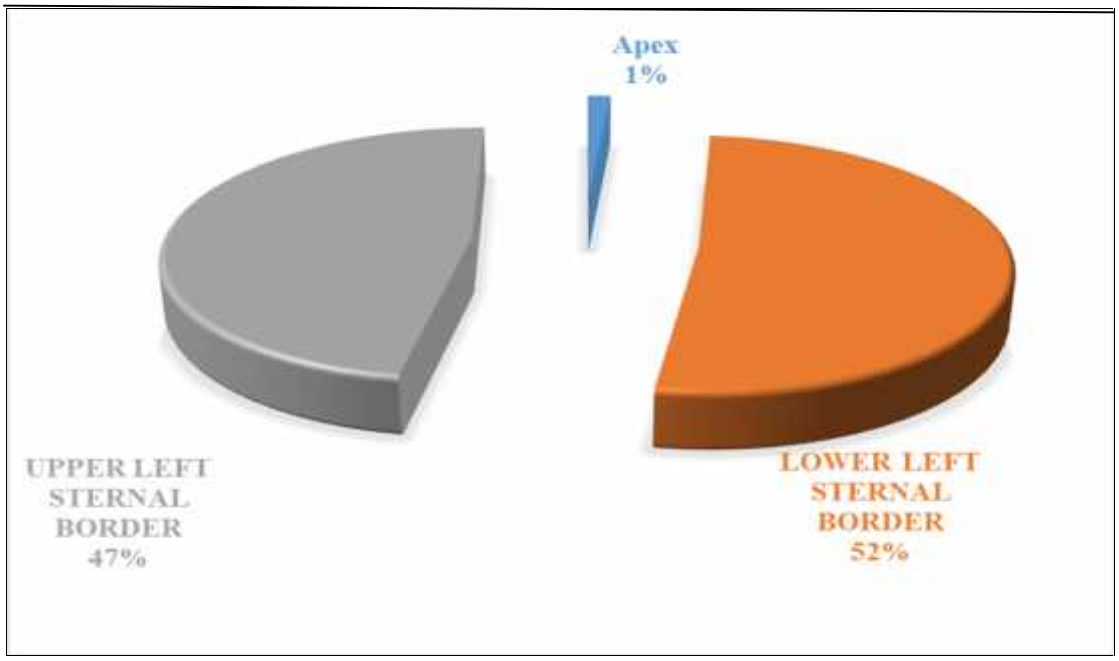


FIGURE 10 : LOCATION OF MURMUR

TABLE NO 11: CENTRAL CYANOSIS - POSITIVE PREDICTIVE VALUE, SENSITIVITY, & SPECIFICITY

	POSITIVE	**NEGATIVE**	**TOTAL**
CENTRAL CYANOSIS PRESENT	3	1	4
CENTRAL CYANOSIS ABSENT	49	21	70
TOTAL	52	22	74

The positive predictive value of cyanosis is 75% in diagnosing congenital heart disease which is statistically significant.

The sensitivity of cyanosis as a clinical examination finding is 5.8% & specificity is 95.5% in diagnosing CHD.

TABLE NO 12: FAST BREATHING - POSITIVE PREDICTIVE VALUE, SENSITIVITY, & SPECIFICITY

	POSITIVE	NEGATIVE	TOTAL
FAST BREATHING PRESENT	10	1	11
FAST BREATHING ABSENT	42	21	63
TOTAL	52	22	74

The positive predictive value of fast breathing is 90.90% in diagnosing congenital heart disease which is statistically significant.

The sensitivity of fast breathing as a clinical examination finding is 19.23% & specificity is 95.45% in diagnosing CHD.

TABLE NO 13: BOUNDING PULSE - POSITIVE PREDICTIVE VALUE, SENSITIVITY, & SPECIFICITY

	POSITIVE	NEGATIVE	TOTAL
BOUNDING PULSE PRESENT	10	4	2
BOUNDING PULSE ABSENT	42	18	72
TOTAL	52	22	74

The positive predictive value of bounding pulse is 71% in diagnosing congenital heart disease which is statistically significant.

The sensitivity of bounding as a clinical feature is 19.20% & specificity is 81.80% in diagnosing CHD.

TABLE NO 14: FEEDING DIFFICULTY - POSITIVE PREDICTIVE VALUE, SENSITIVITY, & SPECIFICITY

	POSITIVE	**NEGATIVE**	**TOTAL**
FEEDING DIFFICULTY PRESENT	12	1	13
FEEDING DIFFICULTY ABSENT	40	21	61
TOTAL	52	22	74

The positive predictive value of feeding difficulty is 92.30% in diagnosing congenital heart disease which is statistically significant.

The sensitivity of feeding difficulty as a symptom is 23.07% & specificity is 95.45% in diagnosing CHD.

TABLE NO 15: RESPIRATORY DISTRESS - POSITIVE PREDICTIVE VALUE, SENSITIVITY, & SPECIFICITY

	POSITIVE	**NEGATIVE**	**TOTAL**
RESPIRATORY DISTRESS PRESENT	12	5	17
RESPIRATORY DISTRESS ABSENT	40	17	57
TOTAL	52	22	74

The positive predictive value of respiratory distress is 70% in diagnosing congenital heart disease which is statistically significant.

The sensitivity of respiratory distress as a clinical feature is 23.10% & specificity is 77.30% in diagnosing CHD.

TABLE NO 16: DIAPHORESIS - POSITIVE PREDICTIVE VALUE, SENSITIVITY, & SPECIFICITY

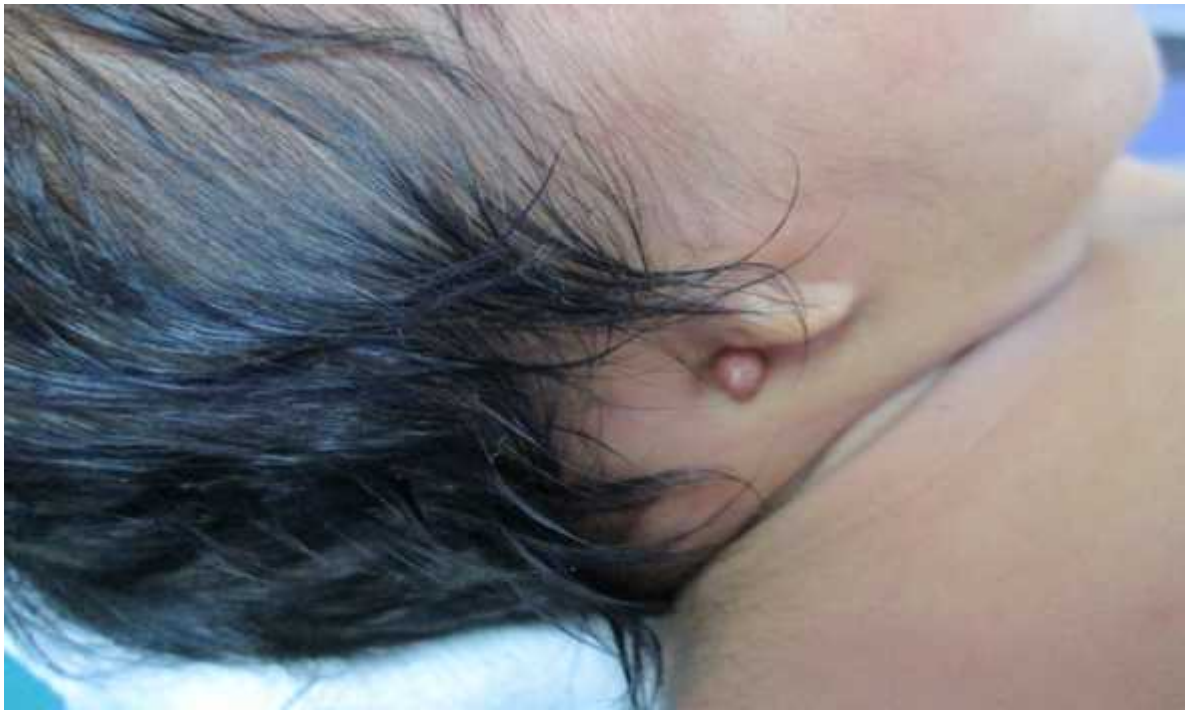
	POSITIVE	**NEGATIVE**	**TOTAL**
DIAPHORESIS PRESENT	1	1	2
DIAPHORESIS ABSENT	51	21	72
TOTAL	52	22	74

The positive predictive value of diaphoresis is 50% in diagnosing congenital heart disease which is statistically significant.

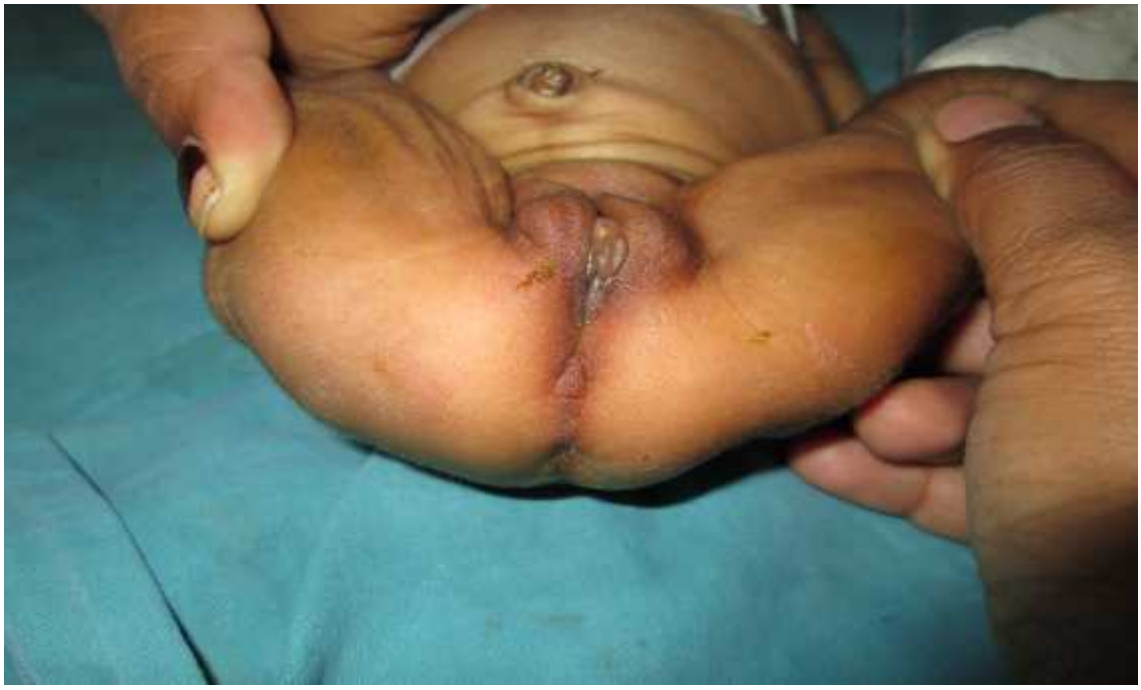
The sensitivity of diaphoresis as a clinical feature is 19.23% & specificity is 95.45% in diagnosing CHD.

PHOTO GALLERY

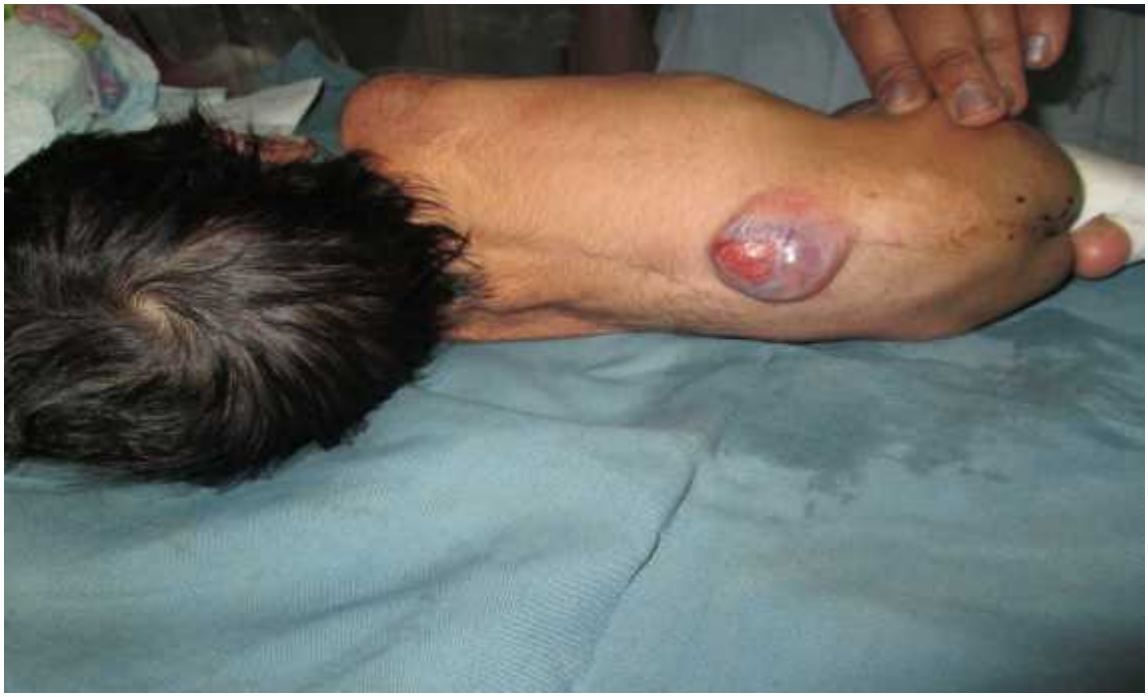
1. ABSENT MIDDLE FINGER



2. MALFORMED EAR

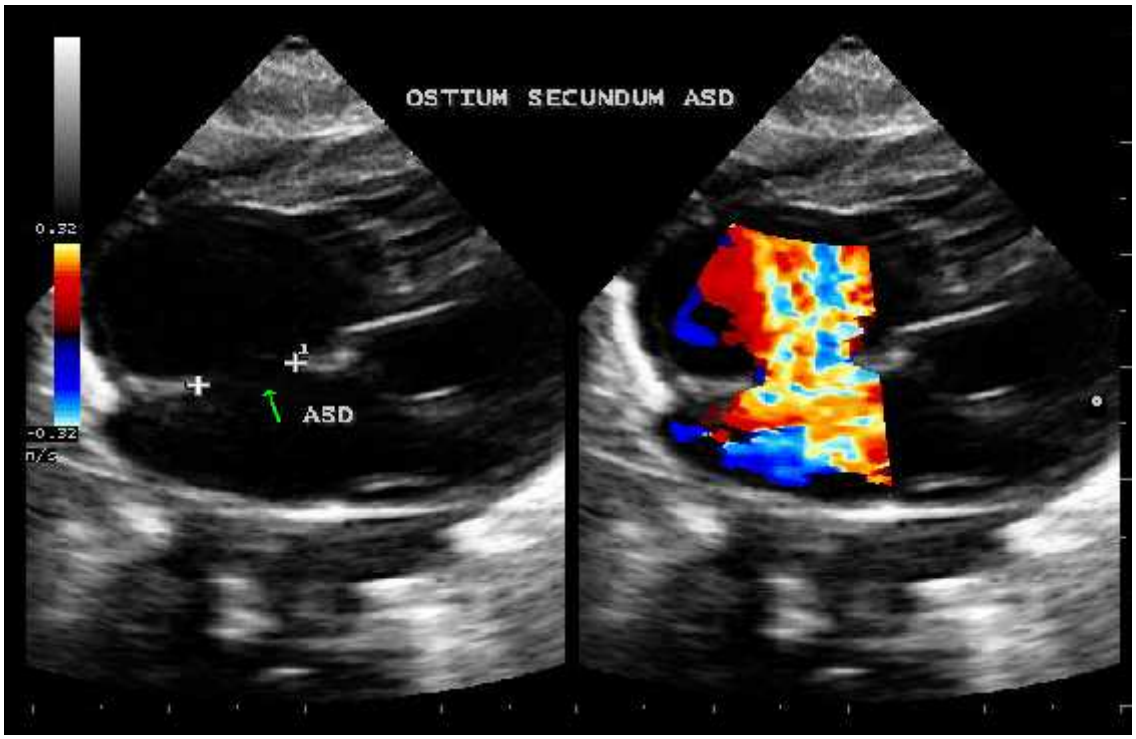


3. IMPERFORATE ANUS

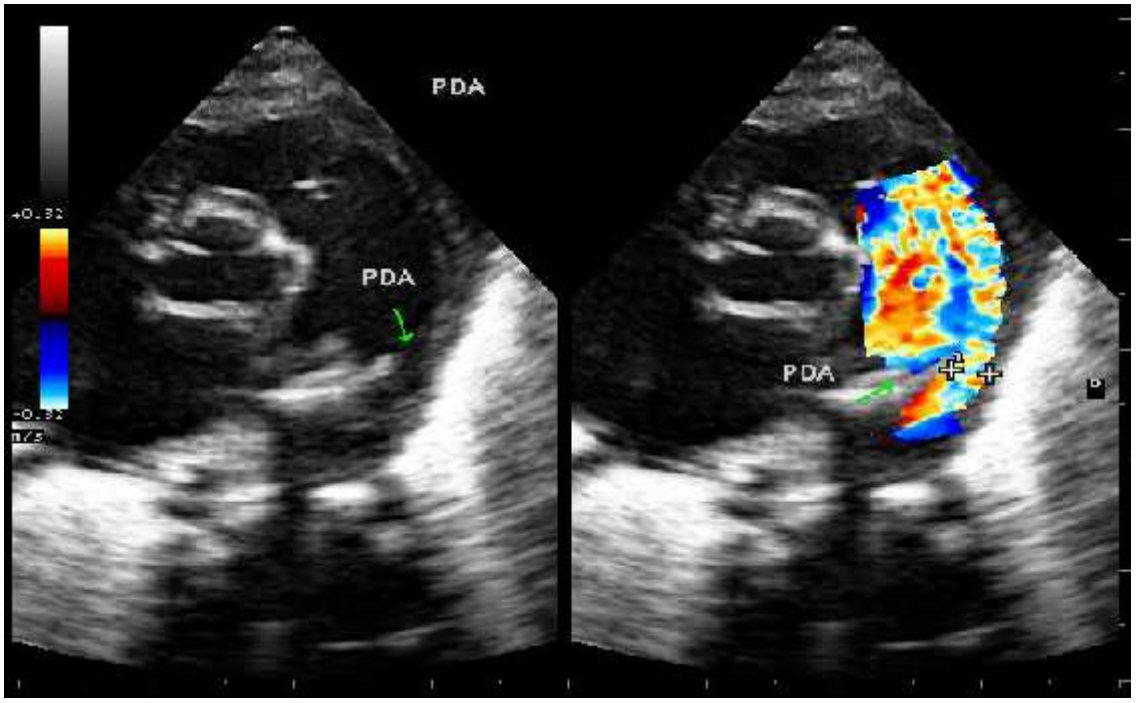


4. MENINGOMYELOCELE

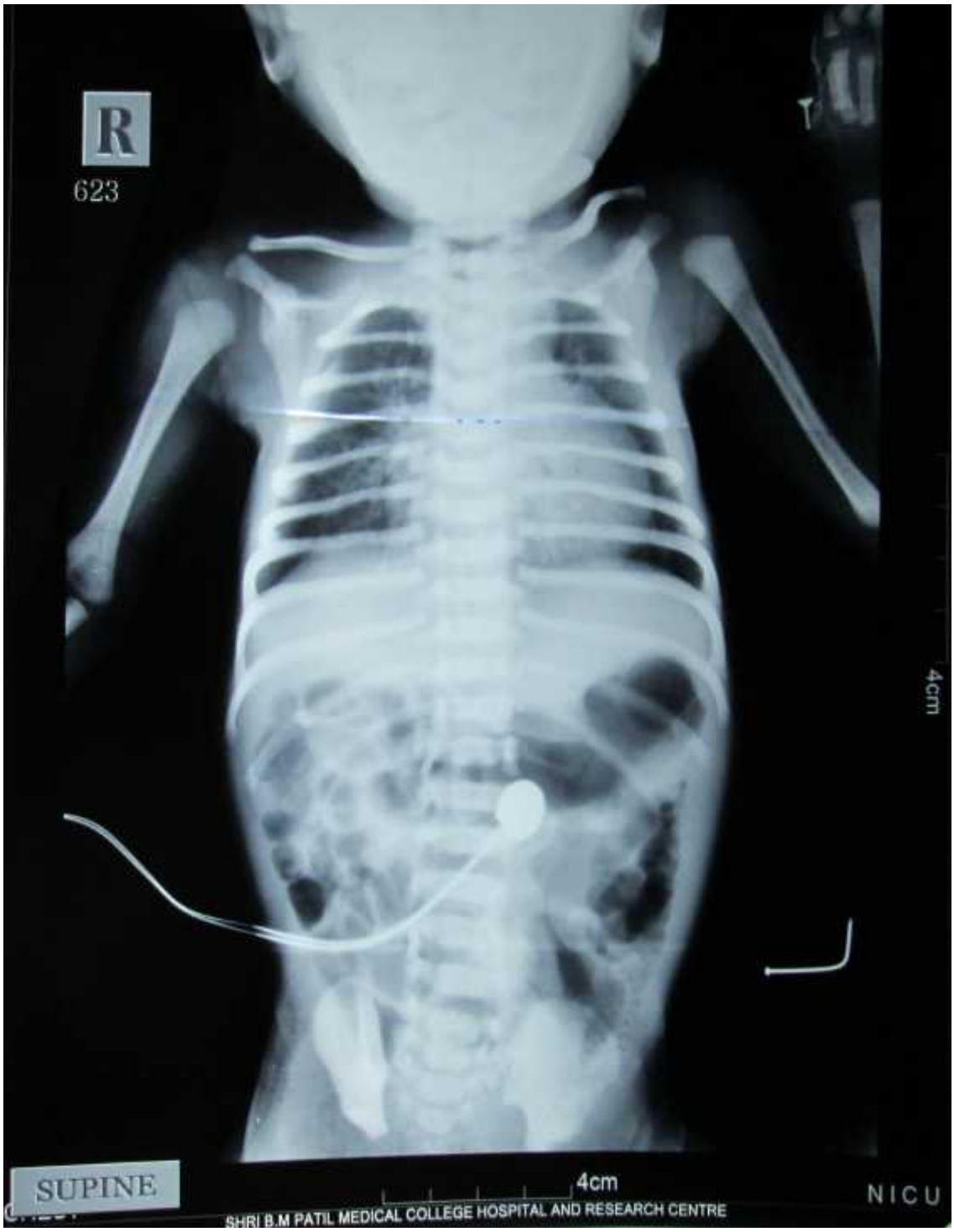
5. CLEFT LIP & CLEFT PALATE



6. OSTIUM SECUNDUM ASD



7. PDA



8. PATIENT WITH D-TGA WITH VSD.

CHEST X-RAY SHOWING CLASSIC "EGG - SHAPED HEART"

DISCUSSION

Congenital heart disease (CHD) encompasses a broad & diverse range of conditions that manifest from prenatal period to adulthood. In common usage CHD refers to structural heart defects that are present at birth. History, physical examination, chest x-ray, ECG, Echocardiography help in identifying presence of CHD. It is possible to identify & determine the severity of specific lesions through echocardiography. The presentation of CHD can range from absence of symptoms to complete cardiovascular collapse. A substantial proportion of patients with CHD have significant problems involving other organ system. Pediatricians need to identify associated conditions, since they might have significant bearing on outcome.

In this study all neonates underwent thorough clinical examination & neonates with heart murmur more than 48 hours were included in study. Both term & preterm neonates were included. All these neonates were subjected to detailed echocardiography & color Doppler examination. A total of 74 neonates were included in study. Study was done over a period of 1 year.

Total number of neonates admitted to neonatal intensive care unit & post natal wards were 2091, out of which 74 had murmur. In our study the incidence of heart murmur among neonates was 35.38 per 1000 neonates admitted to neonatal intensive care unit & post natal wards. The incidence of congenital heart disease was 24.86 per 1000 neonates admitted to neonatal intensive care unit & post natal wards.

Bansal et al in their study have shown incidence of murmur of 23.81 per 1000 live births. The incidence of congenital heart disease was 10.75 per 1000 live births[40].

	Bansal et al[40]	Present study
Incidence of murmur	23.81	35.38
Incidence of CHD	10.75	24.86

Farrer et al studied 8096 neonates & found murmurs in 112 neonates with incidence of 13.8 per 1000 live births[41].

Sean B Ainsworth et al in their study found an incidence of 6 in 1000 babies had murmur undergoing examination by junior pediatricians. About 50% murmurs were due to underlying structural heart disease & this examination lead to recognition of 37% of all heart disease diagnosed in infancy[42].

Kociszewska et al, found 107 (8.3%) out of 1291 newborn had murmur & 93 (86.9%) of these 107 infants had congenital heart disease confirmed by echocardiography[43]. In present study the incidence of CHD & murmur was higher than these studies.

In the present study it was observed that there were 24 (46.15%) SGA neonates, 27 (51.92%) neonates were AGA, 1 (1.92%) neonate was LGA. In a case study by Sadia Malik et al draw a conclusion that infant with CHD were approximately twice as likely to be SGA[44].

In study by H. H. Kramer, et al patient with CHD were often small for gestational age (15%) or had low (<2500g) birth weight (8.6%) [45].

In our study it was observed that there were 27 (51.92%) male, 25 (48.08%) female had CHD. In a study done by Anshula Tendon et al they found 59 (53.63%) female & 51 (46.36%) male neonates were having CHD [46].

	Anshula Tendon et al[46]	Present study
Male	46.36%	51.92%
Female	53.63%	48.08%

In our study there was history of consanguineous marriage in parents of 15 (28.85%) neonates & 37 (71.15%) neonates were having history of non-consanguineous marriage in parents.

In a study done by Anshula Tendon et al, they found that consanguineous marriages were prevalent in 26.36% [46], which is comparable with our study.

	Anshula Tendon et al[46]	Present study
Consanguineous marriage	26.36%	28.85%
Non-consanguineous marriage	73.64%	71.15%

In present study there were 28 (53.85%) preterm neonates & 24 (46.15%) term neonates diagnosed as CHD. In a study by A. Khalil et al also had incidence of congenital heart disease significantly higher in preterm neonates than term neonates. (22.86/1000 vs. 2.36/1000)[47].

In present study, the most frequently associated maternal history was maternal diabetes 33.33% (5 cases), followed by oligohydramnious 26.67% (4 cases), followed by polyhydramnious 20% (3 cases), & intrauterine infections 20% (3 cases).

In a study done by A. Bassili et al about 1% cardiovascular malformations were attributed to maternal diseases disease like diabetes mellitus, phenylketonuria,

SLE, another 1% cardiovascular malformations associated with infectious teratogens like rubella infection. Maternal respiratory tract infection & cytomegalovirus has been proved as risk factors for congenital heart disease[48].

In study done by MD Mahbubul Haque et al, the presentation of CHD cases on admission were[49]:

Signs & symptoms	MD Mahbubul Hoque et al	Present study
Cyanosis	8	3
No cyanosis	14	-
Respiratory distress with or without cyanosis	16	12
Reluctance to feed	12	12
Heart failure	1	1

In present study signs & symptoms were comparable.

In present study most common clinical feature found was respiratory distress 33.33% (12 cases), bounding pulses 27.79% (10 cases), congenital malformation 13.89% (5 cases), pallor 11.11% (4 cases), and cyanosis 8.33% (3 cases).

In our study, total 74 neonates were found to have murmur on clinical examination. Out of them 1 case (1.33%) had murmur at apex, 35 cases (46.67%) had murmur at upper left sternal border, 38 cases (50.67%) had murmur at lower left sternal border.

In present study, out of 74 neonates echocardiography revealed 22 cases (30%) murmurs as functional murmurs & remaining 52 cases (70%) murmurs due to underlying heart disease.

Du ZD et al[8] also concluded in his study that 84% of heart murmur in neonates was due to structural heart disease & 16% were due to functional murmur.

The characteristics of functional murmur were as follows[8]:

1. No cyanosis
2. All murmurs were systolic in nature
3. No murmur was associated with thrill
4. No associated dysmorphism

	Du ZD et al[8]	**Present study**
Murmur due to structural heart disease	84%	74%
Functional murmur	16%	30%

In present study, out of 52 cases of CHD echocardiography showed 16 cases (30.77%) of ASD, 19 cases (36.54%) of multiple defects, 5 cases (9.62%) of VSD, 9 cases (17.31%) of PDA, & 3 cases (5.77%) of cyanotic heart disease. In present study multiple defects includes (ASD + PDA, ASD + VSD, ASD + VSD + PDA).

The cyanotic CHD diagnosed by Echocardiography were d-TGA with VSD, complex CHD with complete AV canal malformation with single AV valve, & hypoplastic left heart syndrome (single ventricle).

Echo diagnosis in MD Mahbubul Hoque et al study[49]:

Structural defect	No (%)
VSD	8 (36.6)
ASD	7 (31.8)
TOF	3 (13.6)
TGA	3 (13.6)
Complex cyanotic heart disease	1 (4.5)

Echo diagnosis of present study:

Structural defect	No (%)
ASD	16(30.77)
ASD +PDA	15(28.85)
VSD	5(9.62)
PDA	9(17.31)
ASD+VSD	3(5.77)
ASD+VSD+PDA	1(1.92)
CYANOTIC	3(5.77)

In a study done by MD Mahbubul Hoque et al [49], most common isolated CHD found was VSD, in present study most common isolated CHD found was ASD.

Bansal et al found VSD as most common lesion accounting to 65.63% of lesion. This high incidence could be because preterm infants were not included in this study [40].

In present study, it was observed that 3 neonates were having central cyanosis. The positive predictive value of cyanosis as clinical feature was found to be 75%. We found that the sensitivity of cyanosis as a clinical examination finding is 5.8% & specificity is 95.5% in diagnosing CHD.

In present study, we observed that 10 neonates were having bounding pulse. The positive predictive value of bounding pulse is 71% in diagnosing congenital heart disease which is statistically significant.

The sensitivity of bounding pulse as a clinical feature is 19.20% & specificity is 81.80% in diagnosing CHD.

In present study, it was observed that 12 neonates were having feeding difficulty. The positive predictive value of feeding difficulty as clinical feature was found to be 92.30%. We found that the sensitivity of feeding difficulty as a symptom is 23.07% & specificity is 95.45% in diagnosing CHD.

In present study, it was observed that 10 neonates were having fast breathing. The positive predictive value of fast breathing as clinical feature was found to be 90.90%. We observed that the sensitivity of fast breathing as a clinical examination finding is 19.23% & specificity is 95.45% in diagnosing CHD.

In present study, we observed that 12 patients were having respiratory distress.

The positive predictive value of respiratory distress is 70% in diagnosing congenital heart disease which is statistically significant. The sensitivity of respiratory distress as a clinical feature is 23.10% & specificity is 77.30% in diagnosing CHD.

SUMMARY

Total of 74 neonates were included in study. We found that 22 (29.73%) murmurs as functional murmurs & 52 (70.27%) murmurs were because of underlying structural heart disease, among total 74 neonates that were included in study. The incidence of murmurs in neonates was 35.38 per 1000 admission. Incidence of congenital heart disease was 24.86 per 1000 admission.

The most common echocardiographic diagnosis of single congenital heart disease found was ASD 16 (30.77%) cases, 19 (36.53%) cases had multiple defects & 3 (5.77%) cases of cyanotic congenital heart disease were found. There were 5 (13.89%) cases with other congenital malformations.

CONCLUSION

Utility of clinical & echocardiographic examination in assessment of neonates with heart murmurs in diagnosis of congenital heart disease is valuable. Echo-cardiography gives correct anatomical diagnosis in suspected congenital heart diseases.

Early diagnosis helps in predicting the prognosis & for planning future treatment decisions.

Most common congenital heart disease found in our study was ASD.

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ANNEXURE-I

B.L.D.E. UNIVERSITY'S
SHRI.B.M.PATIL MEDICAL COLLEGE, BIJAPUR-586 103
INSTITUTIONAL ETHICAL COMMITTEE

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this college met on 18-10-2012 at 3-30pm to scrutinize the Synopsis of Postgraduate Students of this college from Ethical Clearance point of view. After scrutiny the following original/corrected & revised version synopsis of the Thesis has been accorded Ethical Clearance.

Title "clinical and echocardiographic Study of neonates with Suspected heart disease"

—x—x—

Name of P.G. student Dr. Gaurav Ram Upadhye
paediatrics

Name of Guide/Co-investigator Dr. S.S. Kalyanshettar
Assoc prof of paediatrics

DR.TEJASWINI. VALLABHA
CHAIRMAN
INSTITUTIONAL ETHICAL COMMITTEE
BLDEU'S, SHRI.B.M.PATIL
MEDICAL COLLEGE, BIJAPUR.

Following documents were placed before E.C. for Scrutinization
1) Copy of Synopsis/Research project.
2) Copy of informed consent form
3) Any other relevant documents.

ANNEXURE-II

CASE PROFORMA

SCHEME OF CASE TAKING:

Name	**:**	**CASE NO**	**:**
Age	**:**	**IP NO**	**:**
Sex	**:**	**DOA**	**:**
Religion	**:**	**DOD**	**:**
Residence	**:**		

Occupation & Income

Of parents :

MATERNAL MEDICAL History-

1. History suggestive of diabetes mellitus yes/no

2. History suggestive of cardiac diseases yes/no

3. History suggestive of renal diseases yes/no

4. History suggestive of hypertension /PIH/eclampsia yes/no

5. History suggestive of chronic diseases yes/no

6. History suggestive of chronic drug intake yes/no

7. History suggestive of anaemia yes/no

8. History suggestive of intercurrent infections yes/no

9. Past History :

10. Family history: History of CHD in other siblings

11. ANC : ANTENATAL:

NATAL:

POSTNATAL:

12) General Physical Examination

SINGLE/TWIN…….

EGA: by dates…….weeks: by exam…….weeks;

LENGTH……..cm; MAC……..cm

BIRTH WEIGHT……..g; HC…….cm

NEWBORN MATURITY RATING AND CLASSIFICATION

ESTIMATION OF GESTATIONAL AGE BY MATURITY RATING

Symbols : X - 1st Exam O- 2nd Exam

NEUROMUSCULAR MATURITY

	0	1	2	3	4	5
Posture						
Square Window (Wrist)	 90°	 60°	 45°	 30°	 0°	
Arm Recoil	 180°		 100°-180°	 90°-100°	 <90°	
Popliteal Angle	 180°	 160°	 130°	 110°	 90°	 <90°
Scarf Sign						
Heal to Ear						

Gestation by Dates _____ wks

Birth Date _____ Hour _____ am pm

APGAR _____ 1 min _____ 5 min

SCORING SECTION

1st Exam =X 2nd Exam=O

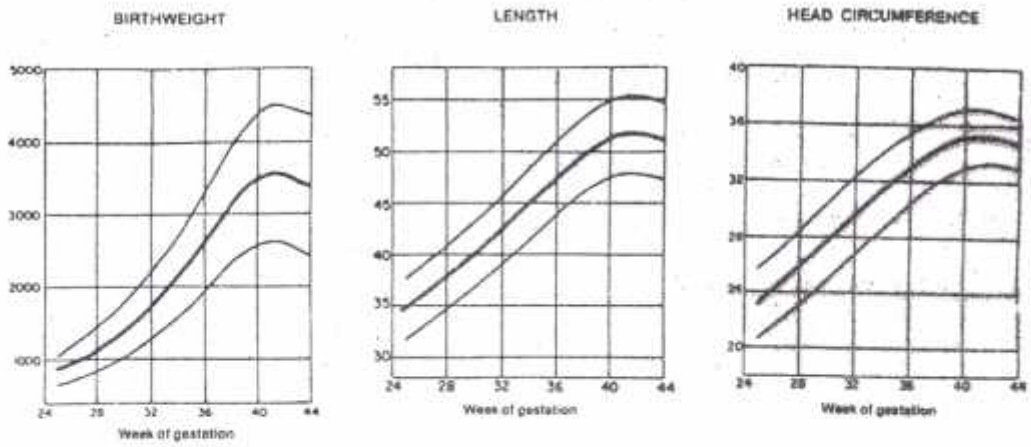
Estimating Gest Age by Maturity Rating	_____ Weeks	____ Weeks
Time of Exam	Date_____ Hour____ am pm	Date_____ Hour____ am pm
Age at Exam	_____Hours	_____Hours
Signature of Examiner	_____ _____M.D.	_____ _____M.D.

PHYSICAL MATURITY

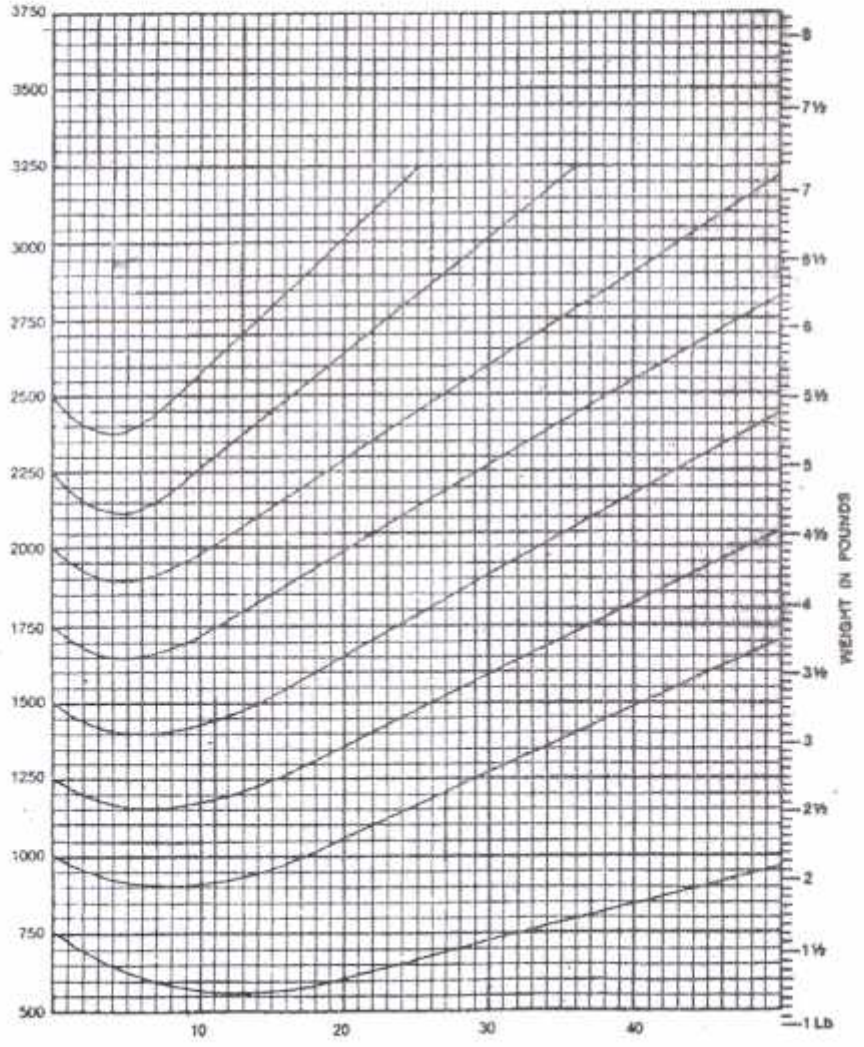
	0	1	2	3	4	5
Skin	gelatinous red. transparent	Smooth pink, visible veins	superficial peeling & /or rash few veins	cracking pale area rare veins	parchment deep cracking no vessels	leathery cracked wrinkled
Lanugo	none	abundant	thinning	bald areas	mostly bald	
plantar Creases	no crease	faint red marks	anterior transverse crease only	creases ant. 2/3	creases cover entire sole	
Breast	barely percept.	flat areola no bud	stippled areola 1-2 mm bud	raised areola 3-4 mm bud	full areola 5-10 mm bud	
Ear	pinna flat, stays folded	sl. curved pinna; soft with slow recoil	well-curv. pinna; soft but ready recoil	formed & firm with instant recoil	thick cartilage ear stiff	
Genitals	scrotum empty no rugae		testes descending, few rugae	testes down, good rugae	testes pendulous deep rugae	
Genitals	prominent clitoris & labia minora		majora & minora equally prominent	majora large, minora small	clitoris & minora completely covered	

MATURITY RATING

Score	Wks
5	26
10	28
15	30
20	32
25	34
30	36
35	38
40	40
45	42
50	44



GROWTH RECORD



13) General examination:

- **Vitals:**
- **Skin**
- **Craniofacial:**
- **Oral cavity**
- **Chest:**
- **Extremities:**
- **Back:**
- **Spine:**
- **Genitalia:**

14) Systemic Examination

- **Cardiovascular system:**
- **Respiratory system:**
- **Abdomen:**
- **Central nervous system:**

Impression

Provisional Diagnosis:

Investigation:

Complete Hemogram:

Special investigations:

1) Chest x-ray
2) ECG
3) Echo-cardiography

Final Diagnosis:

ANNEXURE-III

CONSENT FORM

PURPOSE OF RESEARCH:

I have been informed that the present study will help in assessing clinical and echocardiographic study of neonates with suspected heart disease

PROCEDURE:

I understand that after having obtained a detailed clinical history, thorough clinical examination and relevant investigations, a final work up for the etiological identification and appropriate management is planned.

RISK AND DISCOMFORTS:

I understand that my baby might experience some pain and discomforts during the examination or during the treatment. This is mainly the result of my baby's condition and the procedures of this study are not expected to exaggerate these feelings which are associated with the usual course of treatment.

BENEFITS:

I understand that my baby's participation in the study will have no direct benefit to the baby other than the potential benefit of the treatment.

CONFIDENTIALITY:

I understand that the medical information produced by this study will become a part of hospital records and will be subject to the confidentiality. Information of sensitive personal nature will not be part of the medical record, but will be stored in the investigations research file.

If the data are used for publication in the medical literature or for teaching purpose, no name will be used and other identifiers such as photographs will be used only with special written permission. I may see the photograph before giving the permission.

REQUEST FOR MORE INFORMATION:

I understand that I may ask more questions about the study at anytime; Dr. Gaurav Ram Upadhye at the department of pediatrics is available to answer my questions or concerns. I understand that I will be informed of any significant new findings discovered during the course of the study, which might influence my baby's continued participation. A copy of this consent form will be given to me to keep for careful reading.

REFUSAL FOR WITHDRAWAL OF PARTICIPATION:

I understand that my baby's participation is voluntary and that I may refuse to participate or may withdraw consent and discontinue participation in the study at any time without prejudice. I also understand that Dr. Gaurav Ram Upadhye may terminate my baby's participation in the study after he has explained the reasons for doing so.

INJURY STATEMENT:

I understand that in the unlikely event of injury to my baby resulting directly from participation in this study, if such injury were reported promptly, the appropriate treatment would be available to me. But, no further compensation would be provided by the hospital. I understand that by my agreements to participate in this study and not waiving any of my legal rights.

I have explained to _____the purpose of the research, the procedures required and the possible risks to the best of my ability.

_____ _____

Dr.Gaurav Ram Upadhye
(Investigator)

Signature of Guardian

Date:

STUDY SUBJECT CONSENT STATEMENT:

I confirm that Dr.Gaurav Ram Upadhye has explained to me the purpose of research, the study procedure, that I am willing to allow my baby to undergo the investigation and the possible discomforts as well as benefits. I have been explained all the above in detail in my own language and I understand the same. Therefore I agree to give consent to participate in this research project.

_____ _____

(Guardian) Date

_____ _____

(Witness to signature) Date

ANNEXURE-IV

KEY TO MASTER CHART

B.W	-	Birth Weight
G. A	-	Gestational age
DOE	-	Date of echo-cardiography
RD	-	Respiratory distress
S	-	Small for gestational age
A	-	Appropriate for gestational age
L	-	Large for gestational age
C Ma	-	Congenital malformation
NCM	-	Non consanguineous marriage
CM	-	Consanguineous marriage
T	-	Term
P	-	Preterm
F	-	Female
M	-	Male
D	-	Days
GDM	-	Gestational diabetes mellitus
PO	-	Polyhydramnious
OL	-	Oligohydramnious
IU	-	Intra-uterine infection
N	-	No
Y	-	Yes
UL	-	Uppar left sternal border
LL	-	Lower left sternal border
ASD	-	Atrial septal defect
VSD	-	Ventricular septal defect
PDA	-	Patent ductus arteriosus
FM	-	Functional Murmur
TGA	-	Transposition of great arteries

MASTER CHART

NO	NAME	IP NO	DOE	AGE	SEX	BIRTH WEIGHT (KG)	G.A	S/A/L	CONSANGUINITY	MATERNALRISK FACTOR	CYANOSIS	FAST BREATHING	FEEDING DIFFICULTY	SWEATING	HEART RATE/MINUTE	BLOOD PRESSURE (mm of H	C.Ma.	CENTRAL CYANOSIS	PALLOR	RD	BOUNDING PULSE	DIAPHORESIS	EDEMA	GRADE	LOCATION	DIAGNOSIS
1	B/O SAVITA	19693	20-10-12	9D	F	1.9	P	A	NCM	N	N	N	N	N	124	50/40	N	N	N	N	N	N	N	3	UL	ASD+PDA
2	B/O SUNITA	21658	22-10-12	6D	F	1.7	T	S	NCM	N	N	Y	Y	N	130	56/36	N	N	N	Y	Y	N	N	3	UL	ASD+PDA
3	B/O ROOPALI	23960	22-10-12	3D	M	2.2	P	A	NCM	GDM	N	N	N	N	136	52/42	N	N	N	N	N	N	N	4	UL	ASD+PDA
4	B/O RENUKA	22110	23-10-12	4D	M	2.6	T	A	NCM	N	N	N	N	N	132	60/40	N	N	N	N	N	N	N	2	LL	FM
5	B/O KASTURI	23063	31-10-12	5D	M	2.8	T	A	NCM	N	N	N	N	N	136	64/40	N	N	N	N	N	N	N	2	LL	FM
6	B/O ANITA	24456	1-11-12	8D	M	1.3	P	S	CM	N	N	Y	Y	N	178	54/36	N	N	N	Y	Y	N	N	4	UL	PDA
7	B/O JYOTI	24468	9-11-12	7D	F	1.4	P	S	NCM	PO	N	Y	Y	N	164	52/34	N	N	Y	Y	Y	N	N	4	UL	PDA
8	B/O YAMUNA	24960	13-11-12	3D	F	3	T	A	NCM	N	N	N	N	N	136	64/42	N	N	N	N	N	N	N	3	LL	FM
9	B/O ANITA	25559	13-11-12	5D	F	2	P	A	NCM	N	N	N	N	N	122	52/42	N	N	N	N	N	N	N	3	AP	FM
10	B/O LAXMI	25949	17-11-12	3D	M	1.6	T	S	NCM	N	N	N	N	N	136	50/38	Y	N	N	N	N	N	N	3	LL	ASD+VSD
11	B/O SUMAN	22813	22-11-12	5D	F	2.5	T	A	NCM	N	N	Y	Y	N	170	60/36	N	N	N	Y	Y	N	N	4	UL	ASD+PDA
12	B/O SHILPA	26223	24-11-12	7D	M	1.3	P	S	CM	N	N	N	N	N	130	44/34	N	N	N	N	N	N	N	3	UL	ASD+PDA
13	B/O SHOBHA	25906	26-11-12	15D	M	3	T	A	NCM	GDM	N	N	N	N	140	60/40	N	N	N	N	N	N	N	2	UL	ASD
14	B/O SUMASLATA	26394	26-11-12	7D	M	1.9	P	A	NCM	N	N	N	N	N	140	52/40	N	N	N	N	N	N	N	2	LL	FM
15	B/O SUJATA	26493	26-11-12	11D	M	2.9	T	A	CM	N	N	N	N	N	146	62/44	N	N	N	N	N	N	N	3	LL	FM
16	B/O YASHODA	69	4/1/2013	4D	M	1.4	P	S	CM	OL	N	N	N	N	124	50/34	N	N	N	N	N	N	N	2	UL	ASD
17	B/O VIJAYALAXMI	997	14-1-13	4D	F	1.7	T	S	NCM	N	Y	N	N	N	138	52/34	Y	Y	N	N	N	N	N	3	LL	d-TGA WITH VSD
18	B/O SAROJA	1859	22-1-13	4D	M	2.1	P	A	NCM	N	N	Y	Y	N	160	52/40	N	N	N	Y	Y	N	N	4	UL	ASD+PDA
19	B/O SHILPA	1778	29-1-13	11D	M	1.5	T	S	NCM	N	N	N	N	N	124	44/36	N	N	N	N	N	N	N	3	LL	FM
20	B/O REKHA	1932	2-2-2013	11D	M	2.3	P	A	CM	GDM	N	N	N	N	130	52/40	N	N	N	N	N	N	N	3	UL	ASD
21	B/O LAXMI	3213	14-2-13	5D	F	1.6	T	S	NCM	N	N	N	N	N	128	46/38	N	N	N	N	N	N	N	3	UL	ASD
22	B/O SUNITA	3272	19-2-13	13D	M	3	T	A	NCM	N	N	N	N	N	136	60/38	N	N	N	N	N	N	N	3	LL	FM
23	B/O SALMA	3578	19-2-13	4D	F	2.6	T	A	NCM	N	N	N	N	N	128	64/42	N	N	N	N	N	N	N	3	LL	FM
24	B/O ASMA	3783	21-2-13	3D	M	1.4	P	S	CM	IU	N	N	N	N	136	48/34	N	N	N	N	N	N	N	4	LL	VSD
25	B/O VIJAYALAXMI	5023	4-3-13	8D	F	3.6	T	L	NCM	N	N	N	N	N	130	64/42	N	N	N	N	N	N	N	4	LL	VSD
26	B/O SHANTABAI	5345	4-3-13	10D	F	1.4	P	S	NCM	N	N	N	N	N	122	48/36	N	N	N	N	N	N	N	3	UL	PDA
27	B/O SANGAMMA	5818	5-3-13	5D	M	1.6	T	S	CM	N	N	N	Y	N	142	58/46	N	N	N	N	N	N	N	4	UL	ASD+PDA
28	B/O SANGEETA	6512	12-3-13	4D	F	1.9	P	A	CM	N	N	N	N	N	124	48/36	N	N	N	N	N	N	N	3	UL	PDA
29	B/O GAYATRI	6014	13-3-13	8D	M	2.7	T	A	NCM	N	N	N	N	N	128	62/44	N	N	N	N	N	N	N	3	LL	FM
30	B/O SHAILA	6928	14-3-13	4D	M	1.3	P	S	NCM	N	N	N	N	N	146	52/40	Y	N	N	N	N	N	N	3	UL	ASD+PDA
31	B/O SHANTABAI	7096	16-3-13	3D	F	2.1	P	A	NCM	N	N	N	N	N	140	48/38	N	N	N	N	N	N	N	3	LL	ASD

34	B/O HEMLATA	8818	3-4-13	4D	F	2.6	T	A	NCM	N	N	N	N	N	132	60/40	N	N	N	N	N	N	N	3	UL	ASD+PDA
35	B/O SUREKHA	9120	11-4-13	3D	F	1.3	P	S	CM	N	N	N	N	N	128	50/40	N	N	N	N	N	N	N	3	UL	ASD+PDA
36	B/O SHIVUBAI	9116	22-4-13	3D	M	1.5	T	S	NCM	N	N	N	N	N	140	48/36	N	N	N	N	N	N	N	3	LL	FM
37	B/O UMASHREE	9176	25-4-13	6D	F	2	P	A	NCM	N	N	Y	Y	Y	162	58/38	N	N	N	Y	N	Y	N	4	UL	ASD+VSD+PDA
38	B/O KAVITA	10512	29-4-13	13D	F	2.7	T	A	NCM	N	N	N	N	N	140	62/40	N	N	N	N	N	N	N	3	LL	FM
39	B/O RENUKA	12939	14-5-13	3D	F	2.8	T	A	NCM	N	N	N	N	N	134	64/40	N	N	N	N	N	N	N	3	LL	FM
40	B/O SAMEERA	13328	19-5-13	3D	F	1.6	T	S	CM	N	N	N	N	N	126	56/36	N	N	N	N	N	N	N	2	UL	ASD
41	B/O LAXMI	14728	1-6-13	4D	M	2	P	A	NCM	N	N	N	N	N	130	56/36	N	N	N	N	N	N	N	3	LL	FM
42	B/O SHIRIN	15140	5-6-13	5D	F	2.4	P	A	NCM	GDM	N	N	N	N	140	60/38	N	N	N	N	N	N	N	2	LL	ASD
43	B/O SUJATA	16415	17-6-13	3D	M	1.7	T	S	NCM	N	N	N	N	N	122	56/36	N	N	Y	N	N	N	N	4	LL	VSD
44	B/O MAHANANDA	15131	19-6-13	4D	M	1.9	P	A	CM	N	N	N	N	N	126	58/40	N	N	N	N	N	N	N	3	LL	FM
45	B/O HALLIMA	17763	26-6-13	3D	F	2	P	A	CM	N	N	N	N	N	124	56/38	N	N	N	N	N	N	N	3	UL	ASD+PDA
46	B/O HEENA	17826	1-7-13	7D	M	1.6	T	S	NCM	IU	N	Y	Y	N	166	58/34	N	N	N	Y	Y	N	N	4	UL	PDA
47	B/O KASTURI	18323	5-7-13	3D	F	2.5	T	A	NCM	N	N	N	N	N	130	58/40	N	N	N	N	N	N	N	3	LL	ASD
48	B/O ROOPA	18674	12-7-13	5D	M	1.3	P	S	CM	N	N	N	N	N	136	46/36	Y	N	N	N	N	N	N	3	LL	VSD
49	B/O SIDDAMMA	19334	17-7-13	4D	M	2.6	T	A	NCM	N	N	N	N	N	140	60/40	N	N	N	N	N	N	N	3	LL	FM
50	B/O RENUKA	19665	19-7-13	3D	M	2.6	T	A	NCM	N	N	N	N	N	136	62/40	N	N	N	N	N	N	N	2	LL	FM
51	B/O SAVITRI	19105	22-7-13	22D	M	1.3	P	S	NCM	OL	N	N	N	N	146	48/38	N	N	N	N	N	N	Y	4	LL	ASD+VSD
52	B/O SAVITA	19535	23-7-13	20D	F	2.7	T	A	NCM	N	N	N	N	N	122	60/42	N	N	Y	N	N	N	N	3	UL	ASD
53	B/O SAVITA	19953	31-7-13	9D	F	1.7	T	S	NCM	N	N	N	N	N	126	50/38	N	N	N	N	N	N	N	3	LL	FM
54	B/O MOHINI	21100	3-8-13	3D	F	2.8	T	A	NCM	PO	N	N	N	N	128	64/42	N	N	N	N	N	N	N	3	UL	ASD+PDA
55	B/O MAMTAZ	21911	15-8-13	7D	M	2.6	T	A	NCM	N	N	N	N	N	130	60/40	N	N	N	N	N	N	N	2	LL	FM
56	B/O ASHIWINI	22828	19-8-13	3D	F	1.4	P	S	CM	N	N	N	N	N	136	50/36	N	N	N	Y	Y	N	N	3	UL	ASD+PDA
57	B/O CHANDRIKA	23118	24-8-13	5D	F	2.9	T	A	NCM	OL	N	N	N	N	140	62/44	N	N	N	N	N	N	N	3	UL	ASD+PDA
58	B/O JUBEDA	27520	27-8-13	5D	F	1.4	P	S	CM	N	Y	Y	Y	N	170	46/36	Y	Y	N	Y	N	N	N	4	LL	SINGLE VENTRICLE
59	B/O AISHA	23930	3-9-13	3D	M	1.3	P	S	NCM	N	N	N	N	N	144	46/38	N	N	N	N	N	N	N	3	LL	ASD
60	B/O ANITA	24587	5-9-13	6D	F	2.5	T	A	NCM	N	N	Y	Y	N	124	56/34	N	N	N	Y	Y	N	N	4	UL	PDA
61	B/O KAVERI	26780	7-9-13	4D	M	2.9	T	A	NCM	GDM	N	N	N	N	134	60/40	N	N	N	N	N	N	N	4	LL	ASD+VSD
62	B/O GUNDAKKA	26788	17-9-13	8D	M	2.1	P	A	NCM	N	N	N	N	N	140	56/38	N	N	N	N	N	N	N	3	LL	ASD
63	B/O DURGAWWA	26613	18-9-13	15D	M	2.6	T	A	NCM	N	N	N	N	N	130	62/40	N	N	N	N	N	N	N	3	LL	FM
64	B/O AARTIBAI	27624	26-9-13	16D	M	2	P	A	NCM	N	N	N	Y	N	126	56/38	N	N	N	N	N	N	N	3	UL	PDA
65	B/O TABASUM	28791	30-9-13	9D	M	2.7	T	A	CM	N	N	N	N	N	122	60/34	N	N	N	Y	Y	N	N	4	LL	VSD
66	B/O NIMBAWWA	27821	7-10-13	3D	F	1.5	T	S	NCM	IU	N	N	N	N	146	46/36	N	N	N	N	N	N	N	3	UL	PDA
67	B/O JYOTI	29411	7-10-13	5D	M	2.5	T	A	NCM	N	N	N	N	N	128	60/40	N	N	N	N	N	N	N	3	UL	ASD
68	B/O SIDDAMMA	28317	8-10-13	8D	M	1.4	T	S	NCM	N	N	N	N	N	136	48/38	N	N	N	N	N	N	N	3	LL	FM
69	B/O SANGEETA	30124	8-10-13	5D	M	2	P	A	CM	N	N	N	N	N	130	56/38	N	N	N	N	N	N	N	2	UL	ASD
70	B/O SANGEETA	391	20-11-13	18D	M	1.9	P	A	NCM	PO	N	N	N	N	132	58/38	N	N	N	N	N	N	N	3	UL	ASD
71	B/O SHAMSHADBEE	770	26-11-13	13D	M	2.6	T	A	NCM	N	N	N	N	N	146	64/40	N	N	N	N	N	N	N	2	UL	ASD
72	B/O SHRUTI	3531	14-12-13	3D	F	2.7	T	A	NCM	N	N	N	N	N	140	62/42	N	N	N	N	N	N	N	3	LL	FM
73	B/O GOURAMMA	4906	21-2-14	5D	M	2.7	T	A	NCM	N	N	Y	Y	N	164	58/34	N	N	N	Y	Y	N	N	4	UL	ASD+PDA
74	B/O SAVITRI	9858	10-4-2014	4D	M	1.9	P	A	NCM	N	N	N	N	N	126	52/38	N	N	N	N	N	N	N	3	UL	PDA