A DISSERTATION ON

USE OF PULSE OXIMETRY SCREENING IN EARLY DETECTION OF CONGENITAL HEART DISEASE

M.D (BRANCH VII)

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CERTIFICATE

This is to certify that the dissertation entitled **"USE OF PULSE OXIMETRY SCREENING IN EARLY DETECTION OF CONGENITAL HEART DISEASE"** submitted by **Dr. V. EZHIL SRINIVASAN** to the Faculty of Paediatrics, The Tamil Nadu Dr. M.G.R. Medical University, Chennai in partial fulfillment of the requirement for the award of M.D. Degree Branch VII (Paediatrics) is a bonafied research work carried out by him under our direct supervision and guidance.

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DECLARATION

I, **Dr.V. EZHIL SRINIVASAN** solemnly declare that the dissertation titled "**USE OF PULSE OXIMETRY SCREENING IN EARLY DETECTION OF CONGENITAL HEART DISEASE"** has been prepared by me.

This is submitted to the **Tamilnadu Dr.M.G.R.Medical University**, Chennai in partial fulfillment of the rules and regulations for the M.D.Degree Examination in Paediatrics.

Place: Madurai

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CONTENTS

INTRODUCTION

Among all congenital malformations, congenital heart disease is a relatively common problem with an incidence of 5-8 in every 1000 live births[1-9]. Of this 25% will have critical congenital heart disease.[9-12] Congenital heart disease account for about 10% of infant deaths and about half of deaths due to congenital malformations in developed countries[2]. Now a days even complex congenital heart disease can be treated with the appropriate surgical or catheter intervention[13]. Early diagnosis of congenital heart disease is important for a good clinical outcome. Unrecognized or delayed diagnosis of some severe congenital heart diseases can lead to cardiac failure, cardiovascular collapse, and even death[14-18].

However diagnosis of congenital heart disease in the first few days of life is difficult because of an initial lack of specific clinical signs[2]. Clinical examination remains the most frequently used method to diagnose congenital heart disease in new borns[19-22]. In particular the presence of a heart murmur can raise the suspicion of congenital heart disease. But routine neonatal clinical examination fails to detect more than 50% of infants with congenital heart disease [23]. More than 55% of them have no murmur[24] and most of them are discharged before a

diagnosis can be made and readmitted with severe heart failure or cardiovascular collapse[23].

Therefore there is a need for effective screening program for early detection of congenital heart disease. This disease would be ideally suited for a screening program if simple and reliable method were available. Recently pulse oximetry has been suggested as a screening tool for detection of congenital heart disease especially cyanotic and critical congenital heart diseases in asymptomatic new born.[10,11,26] These babies have a decreased oxygen saturation due to right to left shunt which is either intracardiac or at Patent ductus arteriosus (PDA) level. This can be measured by pulse oximetry.

REVIEW OF LITERATURE

CARDIAC DEVELOPMENT

Knowledge of the mechanisms of cardiac development is necessary in understanding congenital heart defects and developing strategies for prevention.

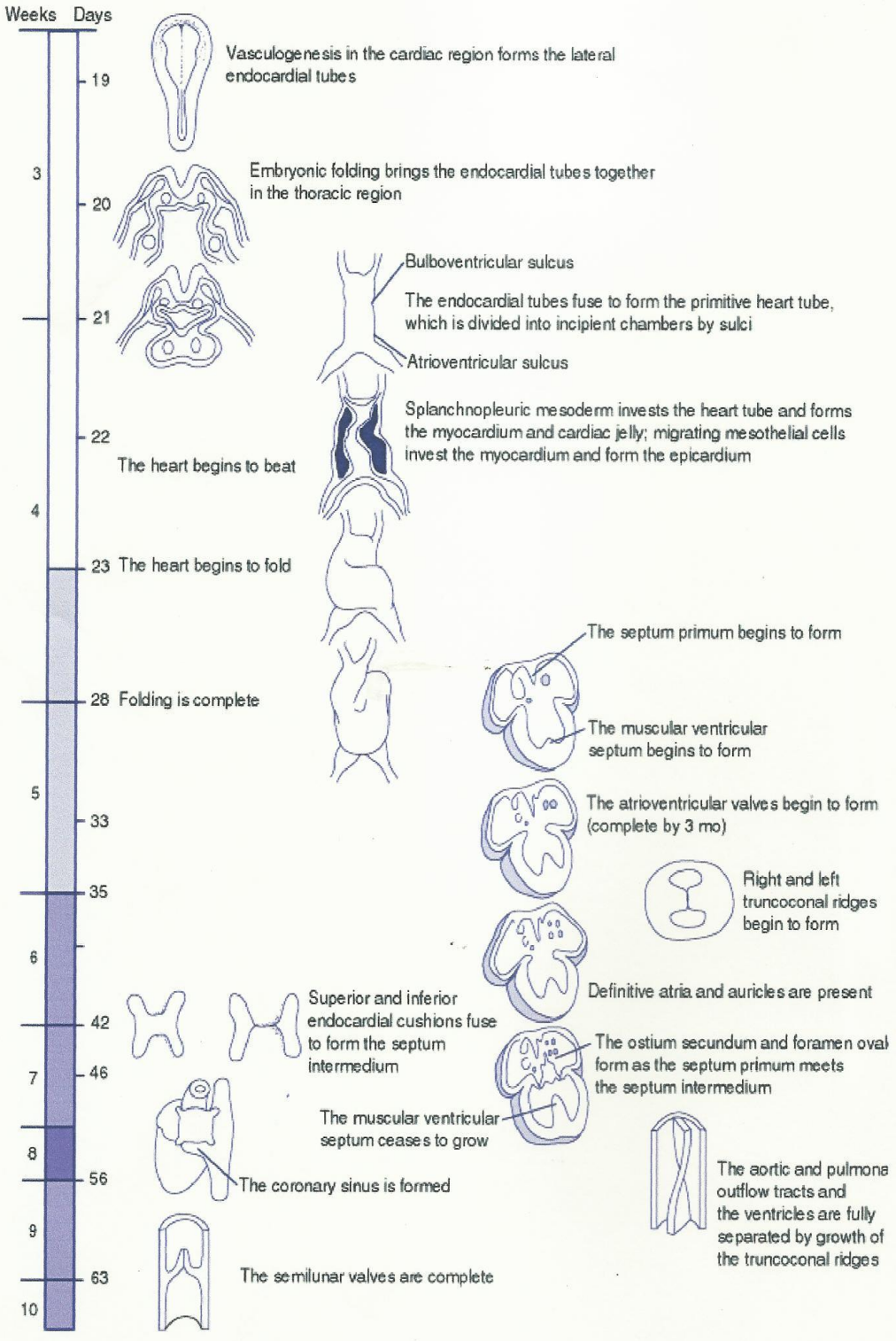
EARLY CARDIAC MORPHOGENESIS

The fetal heart is morphologically developed at 8th week of gestation[5,28]. First identifiable cardiac precursors are angiogenetic cell clusters arranged on both sides of the embryos central axis. These clusters form paired cardiac tube by 18 days of gestation. The paired tubes fuse in the mid line on the ventral surface of the embryo to form the primitive heart tube by 22 days. As early as 20-22 days, before cardiac looping, the embryonic heart begins to contract and exhibits phases of the cardiac cycle that are surprisingly similar to those of in a mature heart[29]. Morphologist have identified segments of the heart tube that were believed to correspond to structures in a mature heart, the sinus venosus and atrium (right and left atria),the primitive ventricle (left ventricle),the bulbus cordis (right ventricle) and the truncus arteriosus (aorta and pulmonary artery).

CARDIAC LOOPING

Between day 23-28 the heart tube is making a loop to the right moving the atrial portion cranially, the ventricular part caudally and the out flow tract remains positioned cranially[30]. When something goes wrong at this stage, the results can be a physiologically corrected transposition of the great arteries or an incorrect relation between the left atrium and aorta causing an AV plane displacement resulting in a double inlet left ventricle or a double outlet right ventricle.

The normal asymmetry caused by the rightward rotation determines the situs of the fetus and normal situs is called situs solitus. If the normal asymmetry does not occur, the result can be situs inversus (mirror image of the normal atrial relationship), situs ambiguous or left or right atrial isomerism (bilateral left sided or bilateral right sided atria).[29]



TIMELINE. FORMATION OF THE HEART.

The chamber differentiation then occurs between days 27-37 and can be divided into four parts beginning with the development of aortic arches. There are originally 6 paired branchial arches developed. They form at different times and regress in a complex way. The first one develops further to become vessels in the face and carotid arteries. Only the 6 th remains complete as the aortic arch. Interruptions here can result in a vascular ring compressing the trachea, like double aortic arch, right aortic arch with left ligamentum arteriosum or pulmonary artery sling (aberrant left pulmonary artery arising from the superior surface of the right pulmonary artery and coursing between the trachea and esophagus).[29]

CARDIAC SEPTATION

Septation of the ventricles begins at about embryonic day 25. The muscular part of inter-ventricular septum grows from the bottom (apex) to the top where the large part is constituted of the perimembranous septum. Naturally defects have results in muscular VSD or perimembranous VSD.

Septation of the atria begins at 30 days of embryonic life. It grows from both sides (septum primum) leaving a patent foramen ovale(PFO) open in the middle. A second septum, septum secondum is

then growing down to the right of the septum primum. The secondum part does not cover the foramen ovale during fetal life, because it is important that the PFO remains open until after birth[28,29].

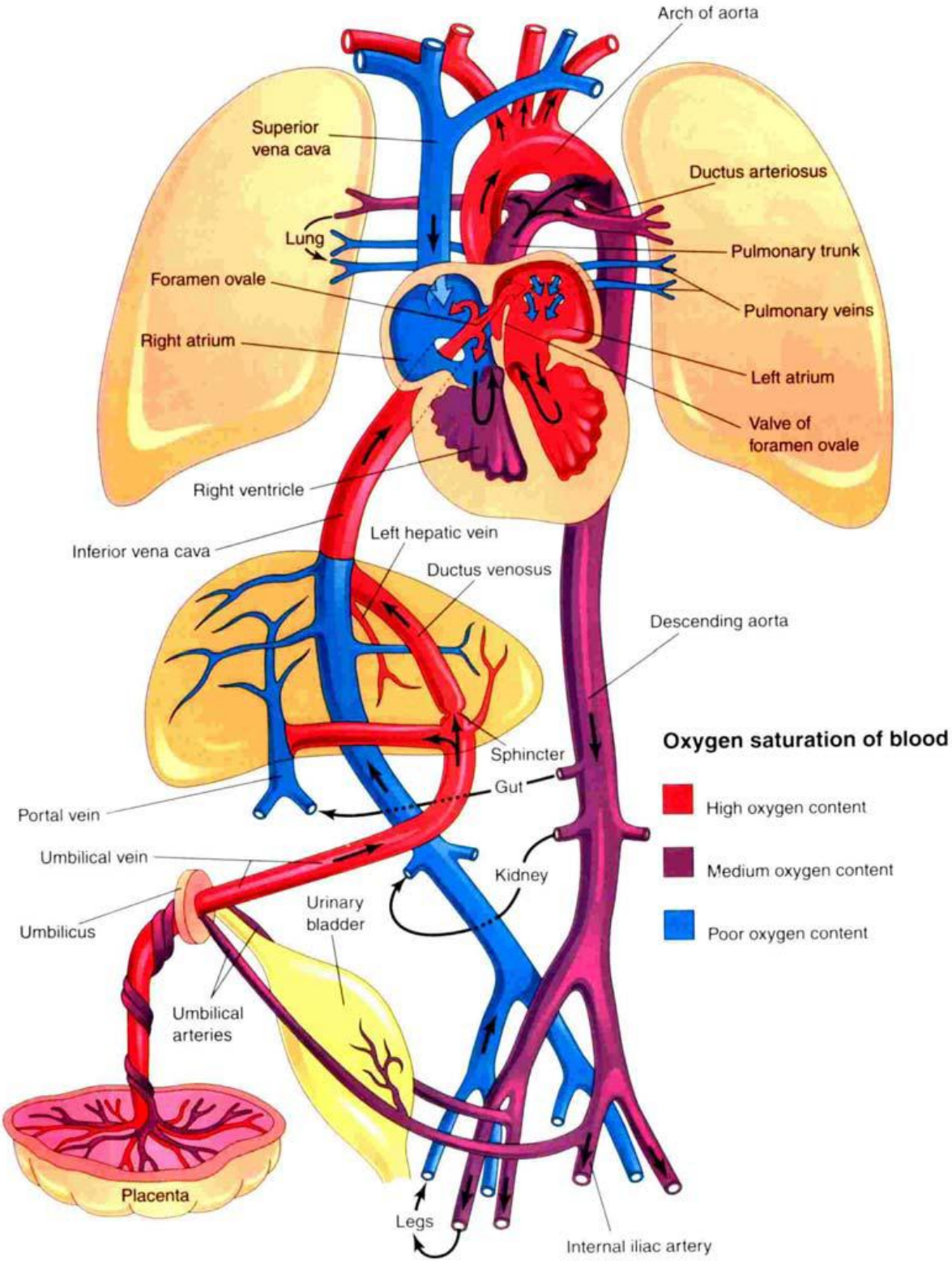
Lastly the septation of the out flow vessel is completed between day 35-42. The out flow septation from one into two, is formed as a spiral and it something goes wrong here, the result can be a common atrial trunk (truncus arteriosus), aorta pulmonary window (APW), transposition of the great arteries (the out flow wall is growing straight instead of spiral formed causing parallel outflows), tetralogy of fallot (the wall is shifted rightwards) or hypoplastic left heart syndrome(HLHS) when the wall is shifted leftwards. The conducting system develops from day 35 to birth and the coronary circulation between day 48-51. Innervation of the heart takes place from day 49 to birth. Formation of the AV valves occurs by excavation and cushions in the ventricles and semilunar valves of excavations and growth in the pulmonary artery and aorta. Incomplete formation of the valves can result in Ebstein anomoly, stenosis or atretic valves.

FETAL CIRCULATION

In the fetal circulation, the right and left ventricles exist in a parallel circuit, as opposed to the series circuit of a newborn or adult. In the fetus, the placenta provides for gas and metabolite exchange. The lungs do not provide gas exchange, and vessels in the pulmonary circulation are venous constricted. Three cardiovascular structures unique to the fetus are important for maintaining this parallel circulation: the ducuts venous, foramen ovale, and ducuts arteriosus.

Oxygenated blood returning from the placenta flows to the fetus through the umbilical vein with a PO2 of about 30-35 mm Hg. Approximately 50% of the umbilical venous blood enter the hepatic circulation, whereas the rest bypasses the liver and joins the inferior vena cava via the ductus venous, where it partially mixed with poorly oxygenated inferior vena cava blood derived from the lower part of the fetal body. This combined lower body plus umbilical venous blood flow (PO2 of = 26-28 mm Hg) enters the right atrium and is preferentially directed across the foramen ovale to the left ventricle and is ejected into the ascending aorta. Fetal superior vena cava blood, which is considerably less oxygenated (PO2 of 12-14 mm Hg), enters the right atrium and preferentially traverses the tricuspid valve, rather than the foramen ovale, and flow primarily to the right ventricle.

FETAL CIRCULATION



From the right ventricle, the blood is ejected into the pulmonary artery. Because the pulmonary arterial circulation is vasoconstricted, only about 10% of right ventricular outflow enters the lungs. The major portion of this blood (which has a PO2 of = 18-22 mm Hg) bypasses the lungs and flows through the ductus arteriosus into the descending aorta to perfuse the lower part of the fetal body, after which it returns to the placenta via the two umbilical arteries. Thus, the upper part of the fetal body (including the coronary and cerebral arteries and those to the upper extremities) is perfused exclusively from the left ventricle with blood that has a slightly higher PO2 than the blood perfusing the lower part of the fetal body, which is derived mostly from the right ventricle. Only a small volume of blood from the ascending aorta (10% of fetal cardiac output) flows across the aortic isthmus to the descending aorta.[109]

THE TRANSITION FROM FETAL TO NEONATAL CIRCULATION

With birth, the function of gas exchange is transferred from the placenta to the lungs, and therefore from the systemic circulation to the pulmonary circulation. The venous and arterial circulations are separated, and not only are the fetal shunts unnecessary, but their persistence may lead to circulatory compromise. The transition from the fetal to the neonatal circulation thus includes elimination of the placental circulation, lung expansion, and increase in lung blood flow so that the entire cardiac output can be accommodated, and closure of the foramen ovale, ductus arteriosus, and ductus venosus.

For most congenital structural heart disease, the fetal shunt pathways allow redistribution of ventricular blood flows so that systemic blood flow is adequate and fetal growth and developments are usually normal. Uncomplicated VSDs do not alter the circulation significantly in either the fetus or immediate newborn period, with the important exception of premature infants. With severe left heart obstruction, the burden of systemic and pulmonary blood flow is transferred to the fetal right ventricle, with reversal of blood flow at the foramen ovale, and systemic blood flow almost entirely transmitted via the ductus arteriosus. This "ductal-dependent" systemic circulation is poorly tolerated in the newborn, because normal closure of the ductus arteriosus progressively

decreases systemic blood flow and progresses to circulatory failure and shock. Severe right heart obstruction is also well tolerated in the fetus, because the combined fetal cardiac output can be transferred to the aorta, with the ductus arteriosus supplying predominantly lung blood flow. After birth, such "ductal-dependent" pulmonary blood flow can lead to critically low levels of pulmonary blood flow and severe cyanosis with closure of the ductus arteriosus.

An understanding of fetal hemodynamics and the acute and chronic changes that occur with transition to the newborn circulation are important for the care of normal newborns and are crucial to the recognition, diagnosis, and management of the newborn with significant congenital heart disease.[109]

CAUSES OF CONGENITAL HEART DEFECTS[31]

The etiology is multifactorial including

- Genetic factors (chromosomal disorders, single – gene disorders and polygenic disorders)
- Environmental risk factors (smoking, paints, varnishing, pesticides, solvents and hair dye).
- Teratogenic drugs

  Alcohol (septal defects)

  Diazepam, Corticosteroids, Hydantoin (pulmonary and aortic stenosis),

Lithium, Trimethadione (transposition of the great arteries,

Tetralogy of fallot, hypoplastic left heart syndrome),

Folate antagonists, thalidomide (conotruncal abnormalities),

Retinoic acid (conotruncal and aortic arch abnormalities)

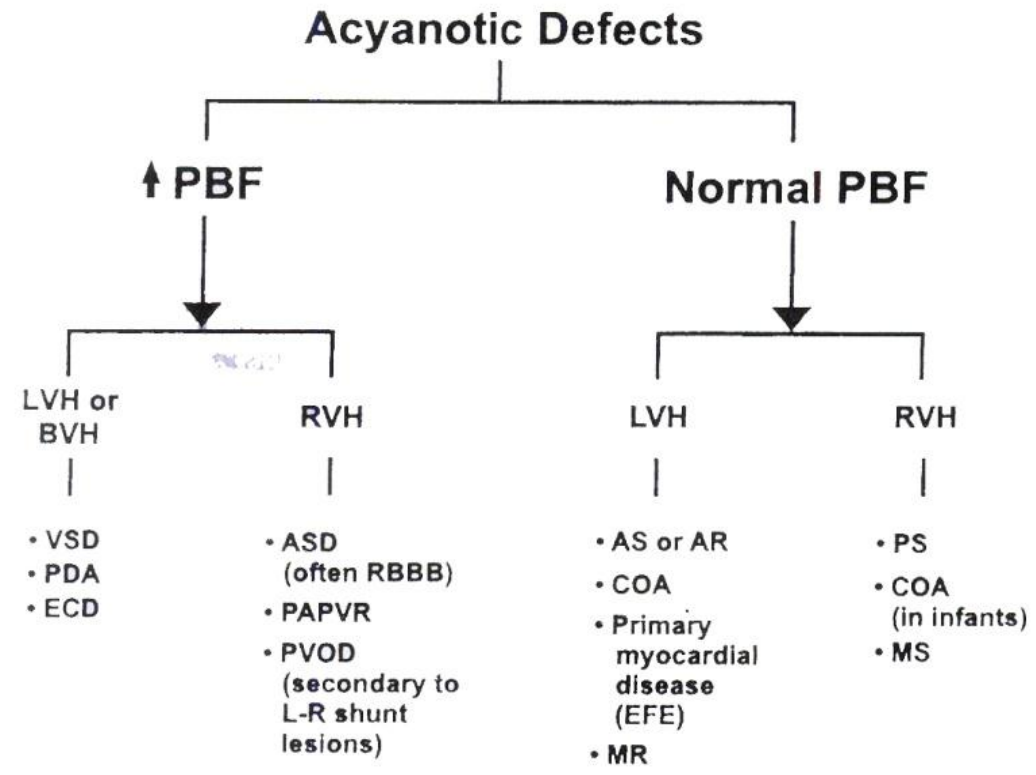
- Maternal disease increasing the risk are Epilepsy (pulmonary stenosis), poorly controlled Diabetes (tetralogy of Fallot, truncus arteriosus, double outlet right ventricle), poorly controlled Phenylketonuria (Tetralogy of Fallot), Systemic Lupus Erythematosus (AV – block grade III).
- Infections such as Rubella during the first trimester (Patent Ductus Arteriosus and peripheral pulmonary artery stenosis) and other viral infections (Coxsackie and HIV).

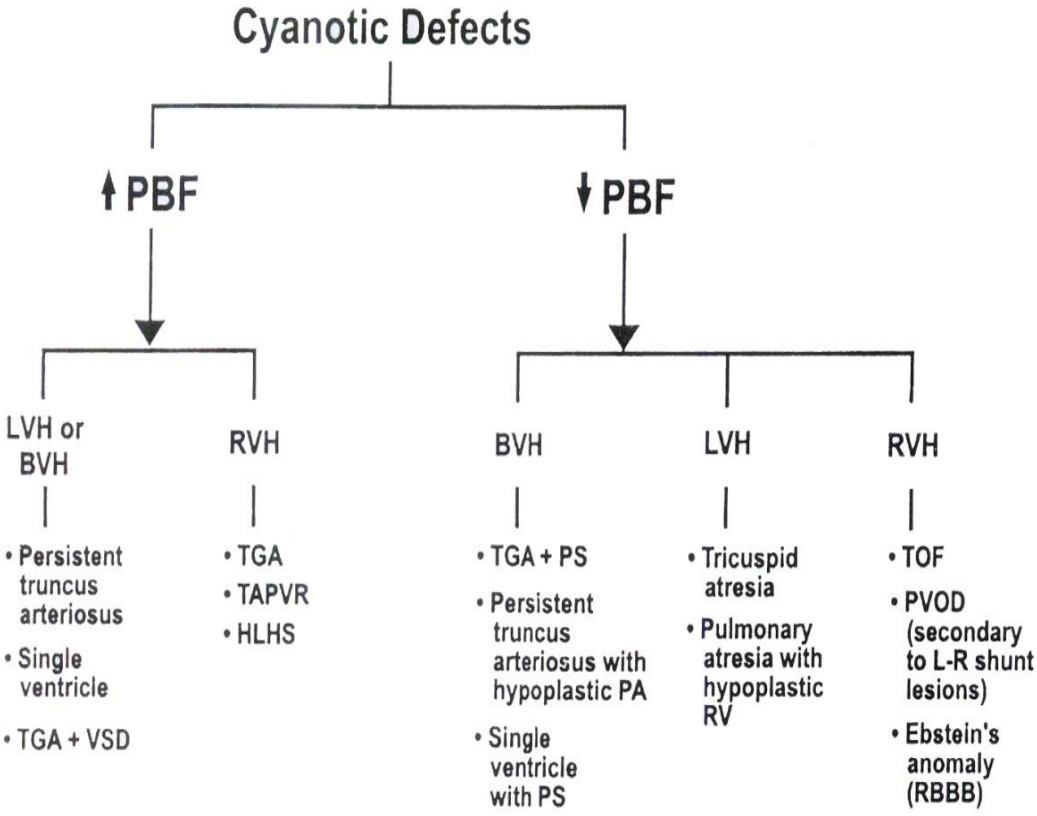
STRUCTURAL CARDIAC DEFECTS

They are classified into

(i) Acyanotic congenital heart defects.

(ii) Cyanotic congenital heart defects.


Acyanotic Defects
↑ PBF
Normal PBF
LVH or BVH
RVH
LVH
RVH
• VSD
• PDA
• ECD
• ASD (often RBBB)
• PAPVR
• PVOD (secondary to L-R shunt lesions)
• AS or AR
• COA
• Primary myocardial disease (EFE)
• MR
• PS
• COA (in infants)
• MS


Cyanotic Defects
↑ PBF
↓ PBF
LVH or BVH
RVH
BVH
LVH
RVH
• Persistent truncus arteriosus
• Single ventricle
• TGA + VSD
• TGA
• TAPVR
• HLHS
• TGA + PS
• Persistent truncus arteriosus with hypoplastic PA
• Single ventricle with PS
• Tricuspid atresia
• Pulmonary atresia with hypoplastic RV
• TOF
• PVOD (secondary to L-R shunt lesions)
• Ebstein's anomaly (RBBB)

CLINICAL PRESENTATIONS OF CONGENITAL HEART DISEASE IN THE NEONATE

The timing of presentation and accompanying symptomatology depend on

- Nature and severity of the anatomic defect.
- The alterations in cardiovascular physiology secondary to the effects of the transitional circulation. (closure of the ductus arteriosus and the fall in pulmonary vascular resistance).

In the first few weeks of life, the many heterogenous forms of heart diseases were presented in a limited number of ways. They are

- Heart murmur
- Cyanosis
- Congestive heart failure
- Arrhythmia

HEART MURMUR

Heart murmurs are not uncommonly heard when examining neonates. Estimates of the prevalence of heart murmurs in neonates vary widely from <1% to 50%> depending on the study. Murmurs heard in newborns in the first days of life are often associated with structural heart disease of some type, and therefore may need further

evaluation, particularly if there are any other associated clinical symptoms.

Pathologic murmurs tend to appear at characteristic ages. Semilunar valve stenosis (systolic ejection murmurs) and atrioventricular valvar insufficiency (systolic regurgitant murmurs) tend to be noted very shortly after birth, on the first day of life. In contrast, murmurs due to left to right shunt lesions (systolic regurgitant ventricular septal defect murmur or continuous PDA murmur) may not be heard until the second to fourth week of life, when the pulmonary vascular resistance has decreased and the left-to-right shunt increase. Therefore, the age of the patient when the murmur is first noted and the character of the murmur provide important clues to the nature of the malformation. [107]

CYANOSIS

Cyanosis (bluish tinge of the skin and mucous membranes) is one of the presenting signs of congenital heart disease in the neonate. Cyanosis usually indicates underlying hypoxemia (diminished level of arterial oxygen saturation). Depending on the underlying skin complexion, clinically apparent cyanosis is usually not visible until there >3 g/dl of desaturated hemoglobin in the arterial system. Therefore, the degree of visible cyanosis depends on both the severity of hypoxemia (which determines the percent of oxygen

saturation) as well as the hemoglobin concentration. For example, consider two infants with similar degrees of hypoxemia each having an arterial oxygen saturation of 85%. The polycythemic newborn (hemoglobin of 22g/dL) will have 3.3 g/dL (15% of 22) desaturated hemoglobin and be more easily appreciated to be cyanotic than the anemic infant (hemoglobin of 10 g/dL) who will only have 1.5 g/dL (15% of 10) deasaturated hemoglobin. True central cyanosis should be a generalized finding (i.e., not acrocyanosis, blueness of the hands and feet only, which is a normal finding in a neonate). [107]

CONGESTIVE HEART FAILURE(CHF)

CHF in the neonate is a Clinical diagnosis made on the basis of the existence of certain signs and symptoms rather than on radiographic or laboratory findings (although these may be supportive evidence for the diagnosis). In early stages, the neonate may be tachypneic and tachycardiac, with an increased respiratory effort, rales, hepatomegaly, and delayed capillary refill. In contrast to adults, edema is rarely seen. Diaphoresis, feeding difficulties, and growth failure may be present. Finally, CHF may present acutely with cardiorespiratory collapse, particularly in “left-sided” lesions. Hydrops fetalis is an extreme form of intrauterine CHF. [107]

Critical Congenital Heart Disease (CCHD)

1. All babies with duct dependent circulation are included in this group. But a baby can have a life threatening heart defect and not being defined as duct dependent. “Paediatric cardiologists commonly defines” CCHD as a condition that either is duct dependent or requires surgery or intervention during the first month of life to survive.[32,33]
2. Mellander and Sunnegardh use the definition of critical as “a heart defect that most likely would have caused circulatory collapse or death if surgery or catheter intervention had not been performed before 2 months of age”.[12,34].
3. **Sendelbach et al.** defined CCHD by listing diagnoses, not severity or time before death or intervention and include tetralogy of Fallot, pulmonary atresia, truncus arteriosus, transposition of the great arteries, total anomalous pulmonary venous return and tricuspid atresia in the cyanotic group and coarctation of the aorta, critical aortic stenosis, interrupted aortic arch and hypoplastic left heart syndrome in the left heart obstructive group[6].

DUCT DEPENDENT LESIONS

In certain conditions the presence of patent ductus is essential for existence of life. In the absence of blood flow in the ductus the child will

die. This is called an obligatory PDA. The conditions that require a patent ductus are as follows[108]

1. Ductus is the only means of the pulmonary blood flow

- Pulmonary atresia with intact ventricular septum
- Tricuspid atresia with intact septum

2. Ductus is the only means of the systemic blood flow

- Aortic atresia
- Preductal coarctation of aorta
- Hypoplastic left heart syndrome

3. Ductus is the means of bidirectional blood flow

- Transposition of great arteries with intact septum

CUSTOMARY PRACTICE

Timely diagnosis of congenital heart disease (CHD) is challenging but critical. Children with such life threatening defects may not initially have symptoms or the symptoms may be vague, and the condition is not detected in the majority of cases [23, 36, 37.]

Strategies that have been suggested to improve early detection of CHD include prenatal ultrasound screening, prolonged hospital stay after delivery, one or more post discharge examinations during the first or the following two weeks of life, and training clinicians to detect “silent” CHD. However, all these strategies are cumbersome and costly and their

value is doubtful. Despite the apparent need, until recently, there were no other means for early detection of CHD [14,39,40].

Since the late 1980s, prenatal ultrasound has been used to screen for congenital anomalies. An anatomic ultrasound is typically performed at 18 to 20 weeks gestation. During this process many, but not all, cases of CHD can be identified by a methodical scan.[41]

Prenatal ultrasound, performed by those with specific training in congenital heart disease, can identify a variety of CHD lesions. However, numerous studies have reported that even when fetal ultrasound is routinely performed during pregnancy, fewer than 50% of cases of CHD are identified[42-47].

There are several factors that might account for the relatively low prenatal CHD detection rate. The quality of anatomic ultrasounds varies considerably. A number of medical professionals, including radiologists, perinatologists, and general obstetricians with varying degrees of training, as well as technicians, perform these ultrasounds. In addition to concerns about the quality, there may be limited access to prenatal ultrasound. The availability of anatomic ultrasound is likely to be particularly limited in certain low socioeconomic status groups. Therefore, although prenatal ultrasound plays an important part in the timely identification of CHD,

population based data demonstrate that this methodology by itself is insufficient to identify a high proportion of cases.[48-52]

Screening for congenital heart disease by primary care providers is currently accomplished by physical examination and on subsequent nursery visits. Supplemental tests, including electrocardiograms, pulse oximetery, and chest radiographs, are often obtained in suspicious cases.[26]

Skilled physical examination, a sensitive and specific screening tool in older children, does not always distinguish between neonates with and without congenital heart disease.[54,55] Hypoxemia is difficult to detect in newborns, and the transitional circulation masks important clinical findings such as absent femoral pulse while the ductus arteriosus remains patent. Perhaps most importantly, physical examination skills are on the decline in current trainees.[56]

Heart murmurs have a prevalence of between 0.6% and 4.2% in newborns and are mistakenly considered a hallmark of heart disease.[24,57] They often do not accompany critical heart defects, particularly those with valve Atresia and transposition. Flow murmurs of the transitional circulation, transient tricuspid regurgitation, and small ventricular septal defects are common and of no clinical importance in newborns. Conversely, murmurs of many important complex heart defects, such as

tricuspid atresia with ventricular septal defect, double – outlet right ventricle, and total anomalous pulmonary venous return, emerge only after the decline in pulmonary resistance and after neonatal discharge and are often heard but not considered pathological. Practicing pediatricians currently have limited experience in discriminating innocent from pathological murmurs. In a contemporary series in which echo-cardiography was performed to evaluate for possible heart disease based on suspicious physical examination, fewer than 15% of subjects were found to have significant congenital heart disease[53].

Clinical experience and epidemiological observations suggest that although physical examination, electrocardiogram, and chest radiograph are useful identifying many cases of serious congenital heart disease postnatally, they do not have sufficient sensitivity and specificity to detect all cases. Echocardiography, although an essential diagnostic tool, has serious limitations as a universal screening tool, particularly its cost.[55] When used positive results (usually related to the transitional circulation) of either false of clinically benign diagnoses (eg, small muscular ventricular septal defects). In addition, there may be an inadequate supply of trained personnel who could perform this screening with a reasonable degree of accuracy. Therefore, there is considerable interest in improving the detection of CHD with novel diagnostic techniques. [105]

PULSE OXIMETRY AND DETECTION OF CHD

A common feature of many forms of congenital heart disease is hypoxemia. Hypoxemia results from the mixing of systemic and venous circulations or parallel circulations as one might see in dextro-transposition of the great arteries. Hypoxemia may result in obvious cyanosis. However, generally, 4 to 5 g of deoxygenated hemoglobin is needed to produce visible central cyanosis, independent of hemoglobin concentration[58]. For the typical newborn with a hemoglobin concentration of 20 g / dL, cyanosis will only be visible when arterial oxygen saturation is <80%; if the infant only has a hemoglobin concentration of 10 g / dL. The saturation must be <60% before cyanosis is apparent. Importantly, those children with mild hypoxemia, with arterial oxygen saturation of 80% to 95%, will not have visible cyanosis. Moreover, the identification of cyanosis is particularly problematic in black and Hispanic neonates because of skin pigmentation[58].

The majority of CHD lesions present with some degree of hypoxemia in the newborn period. The below table demonstrates the frequency of the most common forms of CCHD based on data from the Metropolitan Atlanta Congenital Birth Defects Surveillance Program and the likelihood of having some degree of hypoxemia in the newborn period. To improve timely detection of CHD, a number of investigators

have proposed that pulse oximetry be considered as a complementary modality to the newborn physical examination[11,73].

LESION	PREVALENCE / 10000 LIVE BIRTH	HYPOXEMIA
OUTFLOW TRACT DEFECTS		
Tetralogy of fallot	6.1	MOST
D-transposition of the great arteries	4.0	ALL
Double – outlet right ventricle	1.7	SOME
Truncus arteriosus	1.0	ALL
TAPVC	1.2	ALL
Ebstein anomaly	0.6	SOME
RIGHT OBSTRUCTIVE DEFECTS		
Tricuspid atresia	0.5	ALL
Pulmonary atresia with intact septum	0.8	ALL
Pulmonary stenosis, atresia	6.3	SOME
LEFT OBSTRUCTIVE DEFECTS		
Hypoplastic left heart	3.3	ALL
Coarctation of the aorta	4.7	SOME
Aortic arch atresia or hypoplasia	1.0	SOME
Critical aortic valve stenosis	1.6	UNCOMMON
OTHER MAJOR HEART DEFECTS	12.4	SOME

Pulse oximetry is used routinely in the assessment of young children in neonatal intensive care units and emergency departments and has been proposed as an adjunct to the assessment of the new born in the delivery room[62](Neonatal Resuscitation Protocal 2010 guidelines). As such, some have proposed that pulse oximetry be considered as a vital sign equivalent in importance to pulse, respirations, and blood pressure[63].

PULSE OXIMETRY SCREENING

Every baby is cyanotic until birth. The first breaths and increasing pulmonary circulation induce a rapid rise in pulse oximetry oxygen saturation (Spo2).

PULSE OXIMETRY IN THE NORMAL NEW BORN

The mean preductal (right hand) and postductal (foot) SpO2 of healthy newborns at the age of 2 min has been shown to be 73% (range 44–95%) and 67% (34–93%), respectively. In that study of 50 newborns, these values increased at 10 min to 92% (65–99%) and 89% (62–99%), respectively, and in every newborn both measurements reached 95% within 1hr. The reduction in the difference between preductal and post ductal measurements reflects the diminishing ductal right to left shunt. However, a slower disappearance of the shunt has been seen in 4–5% among larger populations. Thus, use of SpO2 for screening is not practical before the age of 1–2 h. Thereafter, the normal baseline SpO2 is quite stable at about 98%, except for short periods when it is lower, in particular during feeding and apnoeic spells[10, 64-66].

O'Brien and colleagues have defined reference data for oxygen saturation in healthy full term infants during their first 24 hours of life. The median value at 20 to 24hours of life (97.8%) is similar to the results for healthy full-term infants between 2 and 7 days of age (97.6%).Other investigators have reported similar results[66-69].

PULSE OXIMETRY IN CONGENITAL HEART DISEASE

Byrne et al found that cyanotic CHD with obligatory or mixing cyanosis was associated with either a generally low level of SpO2 <88%.[70] However, a pure left to right shunt does not affect systemic oxygenation and is not detectable by pulse oximetry. These phenomena form the basis of pulse oximetry screening for CHD.

AGE AT PULSE OXIMETRY SCREENING

Because newborns with critical congenital heart disease (CCHD) may have clinical deterioration in the first 48 hours of life, one would ideally use oximetry screening soon after delivery. However, arterial oxygen saturation varies considerably in the first 24 hours, with many healthy newborns having arterial saturations of less than 95%. As such, oximetry screening before 24 hours of life can result in a significant number of false-positive results. A study from the United Kingdom reported that the false-positive rate was as high as 5% when oximetry screening was performed in the first 24 hours compared with 1% at the time of hospital discharge[71]. Therefore, to achieve an acceptable specificity, testing 24 hours after birth would appear to be the most reasonable strategy.

CUT-OFF SATURATION

The establishment of a cutoff threshold for an abnormal SpO2 is important. Other factors being constant, a higher threshold will increase sensitivity and at the same time decrease specificity. Setting the SpO2 cutoff value closer to the normal level will decrease the number of false-negative screening results at the cost of increasing the number of false-positive screening results. Conversely, a lower SpO2 threshold will lower sensitivity and raise specificity. Although a number of SpO2 thresholds have been proposed, many investigators believe that an SpO2 of <95% is appropriate. One study suggested that SpO2 <92% be considered a positive sign of hypoxemia however, others have argued that a low threshold is likely to result in a number of infants with critical congenital heart disease (CCHD) being misclassified as normal without markedly improving specificity[72].

PROBE SITE

Newborns who have obligatory or mixing cyanotic CHD have reduced SpO2 both preductally and postductally. Defects with post ductal dependent systemic circulation exhibit higher preductal than post ductal SpO2 due to right to left shunting of unoxygenated blood to lower body[73]. Pulse oximetry screening is thus most effective with postductal probe placement.

SIGNAL QUALITY AND NEWBORN BEHAVIOUR

Technical errors are minimized by accepting only high quality pulse oximetry signals confirmed by a good pulse signal displayed simultaneously.[75,76] Measurements should not be made when the infant is moving, crying, eating or has an apnoeic spell.[66,76] The heart rate displayed by the oximeter should be within the range expected for a calm, regularly breathing newborn (about 90–160/min). The highest stable SpO2 value, maintained longer than the averaging time of the pulse oximetry, is recorded. [37,74,75]

LIMITATIONS OF PULSE OXYMETRY SCREENING

There are technical limitations to oximetry measurement in the newborn. As noted above, the mean SpO2 in the newborn at 24 hours of age is 97% to 98%. However, when continuous pulse oximetry is used, multiple investigators have demonstrated periodic and or sustained desaturation below 95% during sleep, feeding, and crying.[64,66,69,77] Sustained rather than variable hypoxemia is consistent with the diagnosis of cyanotic congenital heart disease. Low oximetry readings in the setting of normal arterial oxygen saturation have been reported by multiple investigators.[78,79] In fact, falsely low oximetry readings in the newborn population are known to be associated with low peripheral perfusion and

motion artifact,[80,81] probe placement site and partial probe detachment[82] , and hyperbilirubinemia or dyshemoglobinemias.

There has also been concern that pulse oximeters may not be as accurate in darkly pigmented adults and children. At low SpO2 levels 70%, commercially available oximeters appear to overestimate arterial saturation by 3% in darkly pigmented subjects[85]. However, when SpO2 is 90%, measurement bias related to skin pigmentation appears negligible (0.2%). Lastly, the quality of oximetry measurements may be lower when performed in a screening setting.[9]

PULSE OXIMETRY

The first commercially available pulse oximeter was developed by Takuo Aoyagi and was marketed by Nihon Koden in 1974.

Pulse oximetry is a non-invasive way of measuring the oxygen saturation in arterial blood SpO2. A pulse oximeter is based on spectral analysis and combines two technologies, spectrophotometry and optical plethysmography.[87]

SPECTROPHOTOMETRY

A spectrophotometer measures light intensity as a function of the colour or more specifically the wavelength of light. There are two classes of spectrophotometers, single beam and double beam. Single beam spectrophotometer measures the absolute light intensity, whereas double

beam spectrophotometer measures the ratio of the light intensity from two different light paths. The pulse oximeters use double beam for measuring the hemoglobin oxygen saturation.

OPTICAL PLETHYSMOGRAPHY

A plethysmograph is a pulse-volume recorder, a way of measure the pulsatile changes from the arterial blood at the sensor site.

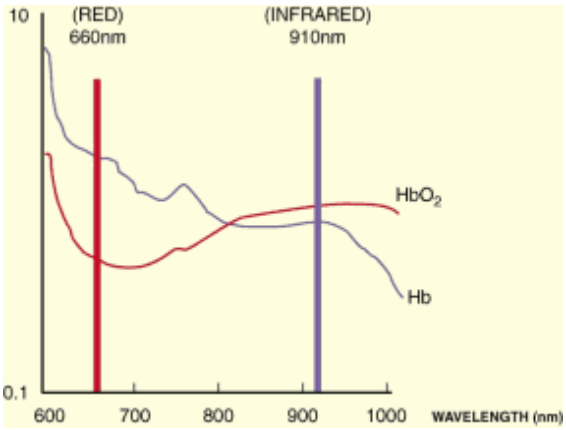
PRINCIPLES OF PULSE OXIMETRY TECHNOLOGY

The principle of pulse oximetry is based on the red and infrared light absorption characteristics of oxygenated and deoxygenated hemoglobin. Oxygenated hemoglobin absorbs more infrared light and allows more red lights to pass through. Deoxygenated (or reduced) hemoglobin absorbs more red light and allows more infrared light to pass through. Red light is in the 600-750 nm wavelength light band. Infrared light is in the 850-1000 nm wavelength light band.

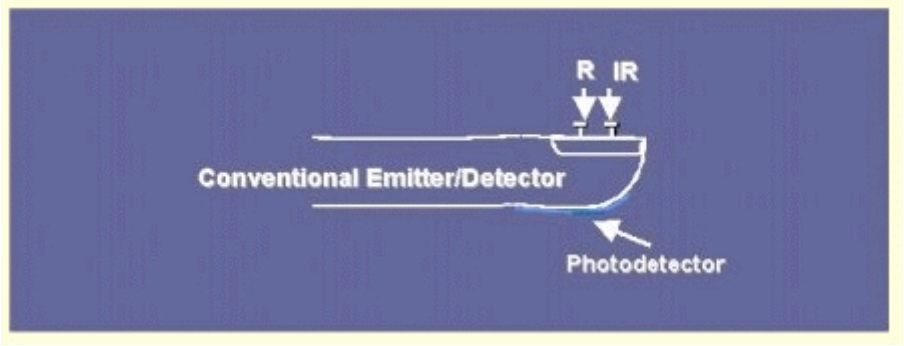
Pulse oximetry uses a light emitter with red and infrared LEDs that shines through a reasonably translucent site with good blood flow. Typical adult and pediatric sites are the finger, toe, pinna (top) or lobe of the ear. Infant sites are the foot or palm of the hand and the big toe or thumb. Opposite the emitter is a photo detector that receives the light that passes through the measuring site.

The principle of Beer Lambert law is used to measure the relative concentrations of reduced hemoglobin (RHb) and oxygenated hemoglobin(O_2 Hb) in pulse oximeters. The ratio of light absorbed at the red light to that of infrared light A660nm (RHb) / A940 nm (O_2 Hb) correlates with oxygen saturation, since the concentration of a given solute in a solvent is measured by the amount of light that is absorbed by the solute at a specific wavelength.[87] After the transmitted red (R) and infrared (IR) signals pass through the measuring site and are received at the photodetector, the R/IR ratio is calculated. The R/IR is compared to "look-up" tables (made up of empirical formulas) that convert the ratio to a SpO2 value. Most manufacturers have their own look-up tables based on calibration curves derived from healthy subjects at various SpO2 levels. [88]

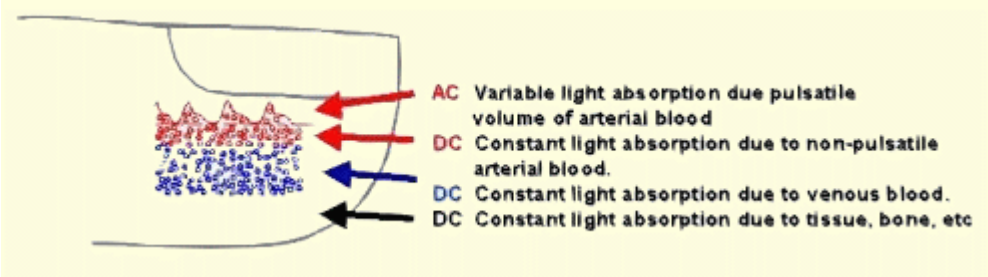
A pulse oximeter can be calibrated in two ways, either displaying functional saturation or fractional saturation. [69,89] Functional saturation is the quantity of Oxyhaemoglobin (HbO_2) expressed as a percent of haemoglobin that can transport oxygen (since MetHB and COHB cannot transport oxygen, they are not included). The functional SpO2 value is obtained by multiplying the fractional saturation by 1.02. Fractional saturation is the HbO2 expressed as a percent of all the hemoglobin measured, including carboxyhaemoglobin and



Difference in wavelength of red and infrared light



Light emitter with red and infrared LED being received by photodetector which passes through the measuring site.



The different tissues affecting light absorption

methaemoglobin.[87,90.] That means that a pulse oximeter calibrated for fractional saturation displays about 2% lower values than an oximeter calibrated for functional saturation.[69,91] The calibrations are performed by the manufacturer and thus cannot be changed by the user.

$$\text{Functional SpO2} = \frac{\text{O2Hb}}{\text{O2Hb + RHb}} \times 100$$

$$\text{Fractional SpO2} = \frac{\text{O2Hb}}{\text{O2Hb + RHb + MetHb + COHb}} \times 100$$

The major change that occurred from the oximeters of the 70s to the oximeters of today was the inclusion of arterial pulsation to differentiate the light absorption in the measuring site due to skin, bone, pigmentation and venous blood, from that of arterial blood. The former absorbs a constant amount of light over the time. The arteriolar bed pulsates and absorbs variable amounts of light during systole (peak) and diastole (trough), as blood volume increases and decreases. The ratio of light absorbed at the peak and trough is translated into an oxygen saturation measurement. Since peaks occur with each heartbeat or pulse, the term pulse oximetry was coined.

LIMITATIONS WITH CONVENTIONAL TECHNOLOGY PULSE OXIMETRY[92-103]

- Motion artifact (when the patient is moving, the venous blood is also moving and the pulse oximeter adds the moving venous blood to the AC component, thus the displayed SpO2 is underestimated).
- Light interference (phototherapy and bright light can affects SpO2 accuracy).
- Skin pigmentation (overestimations of SpO2 in dark pigmented skin, as skin darkens, the oximeter performance deteriorates. This could be a result of empirical calibration data derived from predominantly white volunteers).
- Low peripheral perfusion states
- Dyshaemoglobinemia (carboxyhemoglobin and methemoglobin are unable to carry oxygen.SpO2 readings may appear normal even in the hypoxic patient.)
- Low oxygen saturation
- Nail polish
- Irregular heart rhythm
- Temperature (low peripheral temperature and vasoconstriction contributes inaccuracy).

Still, conventional pulse oximetry accuracy suffered greatly during motion and low perfusion and made it difficult to depend on when making medical decisions. The advent of **"NEXT GENERATION"** pulse oximetry technology has demonstrated significant improvement in the ability to read through motion and low perfusion[92-103].

AIM AND OBJECTIVES

- To detect the usefulness of pulse oximetry screening in early detection of congenital heart disease (CHD).
- To evaluate combined pulse oximetry and clinical examination as a screening method for congenital heart disease (CHD) in asymptomatic newborns.

MATERIALS AND METHODS

STUDY DESIGN

Prospective study

STUDY PLACE

Post natal ward, Government Rajaji Hospital, Madurai.

STUDY PERIOD

September 2010 to August 2011

STUDY POPULATION

New born babies present in postnatal ward of Government Rajaji Hospital, Madurai.

INCLUSION CRITERIA

ASYMPTOMATIC TERM NEW BORN babies present in post natal ward.

EXCLUSION CRITERIA

Sick babies admitted to NICU at birth, Preterm babies, newborn babies with prenatal diagnosis of CHD, babies with external congenital anomaly were excluded from the study.

METHODOLOGY

This study was a hospital based prospective study. In this study we screened asymptomatic term new born babies in the post natal ward for congenital heart disease using pulse oximetry and cardiac clinical examination between September 2010 and August 2011.

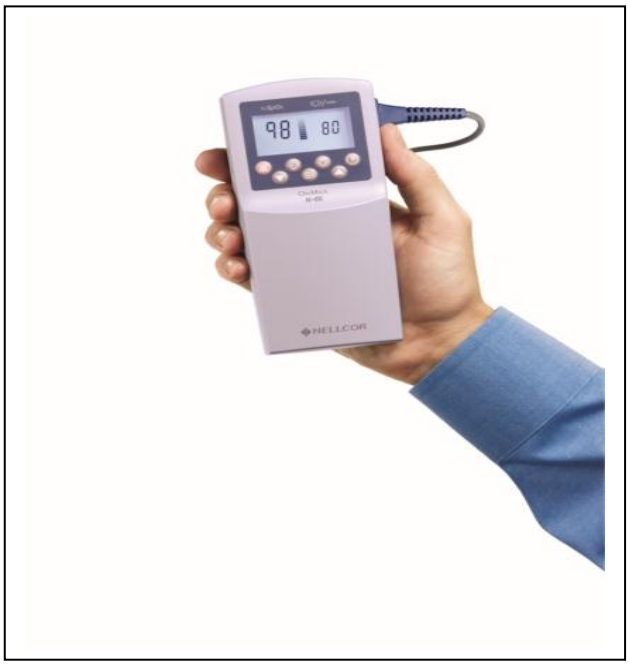
Sick babies admitted to NICU at birth, preterm babies, newborn babies with prenatal diagnosis of CHD, babies with external congenital anomaly were excluded from this study.

Asymptomatic term new born babies were screened by pulse oximetry using **NELLCOR OXIMAX N- 95 HAND HELD NEW GENERATION PULSE OXIMETER**. Pulse oximetry readings were taken in a quiet or sleeping newborn from right foot. The probe was cleansed with alcohol swab before each use.

The readings were recorded after stabilization for one minute, according to the manufacturer instructions. The functional oxygen saturation of ≥95% was accepted as normal. If the new born baby had oxygen saturation below 90%, echocardiography was performed. In the case of a new born with oxygen saturation between 90%-94%, a second measurement was performed six hours later. If the oxygen saturation remained below 95%, echocardiography was performed.

NELLCOR OXIMAX N – 95 HAND HELD

NEW GENERATION PULSE OXIMETER

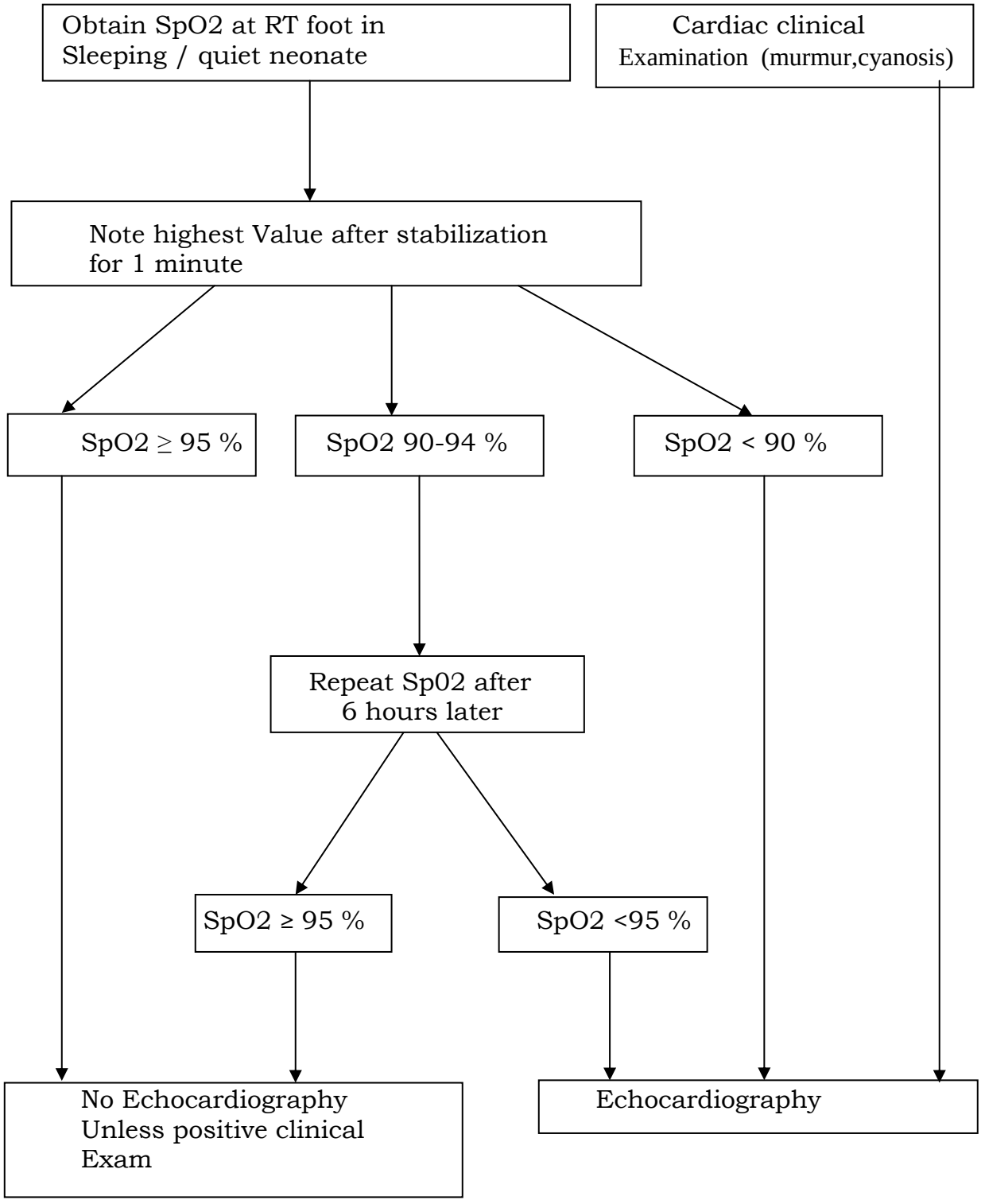


PULSE OXIMETRY SCREENING

Cardiac clinical examination screening was also performed for all babies simultaneously with pulse oximetry screening. We looked for central cyanosis and cardiac murmur. Echocardiography was performed for babies having cardiac murmur or central cyanosis. The cardiac clinical examination and pulse oximetry screening were carried out between 24 to 48 hours of age.

New born babies who underwent echocardiography were categorized as having either a normal heart or a structurally malformed heart.

STUDY PROTOCOL



OBSERVATION AND RESULTS

A total of 2437 asymptomatic term newborn babies entered the study and they were screened by pulse oximetry and clinical examination. Male babies were 1269(52%). Female babies were 1168(48%). Screening was performed at a median age of 36 hours. Birth weight ranged from 2kg to 3.8 kg. Average birth weight was 2.6kg. 24.5% of babies were born to consanguineous parents and 75.5% of babies were born to nonconsanguineous parents. 50% of babies were of first order. 51.4% of babies were delivered by labour naturale.

Table - 1

SEX DISTRIBUTION OF SCREENED BABIES

SEX	NUMBER	PERCENTAGE
MALE	1269	52%
FEMALE	1168	48%
TOTAL	2437	100%

SEX DISTRIBUTION OF SCREENED BABIES

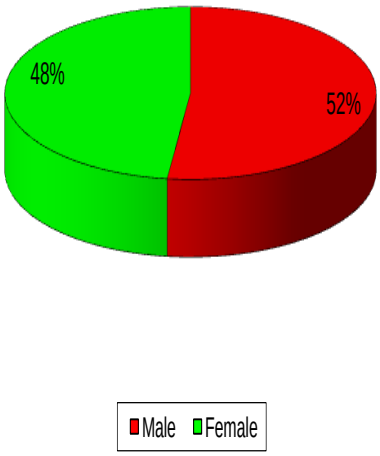


Table - 2

MODE OF DELIVERY OF SCREENED BABIES

MODE OF DELIVERY	NUMBER	PERCENTAGE
NATURAL LABOUR	1254	51.4%
LSCS	970	39.8%
OUTLET FORCEPS	165	6.8%
BREECH	48	2.0%
TOTAL	2437	100%

MODE OF DELIVERY

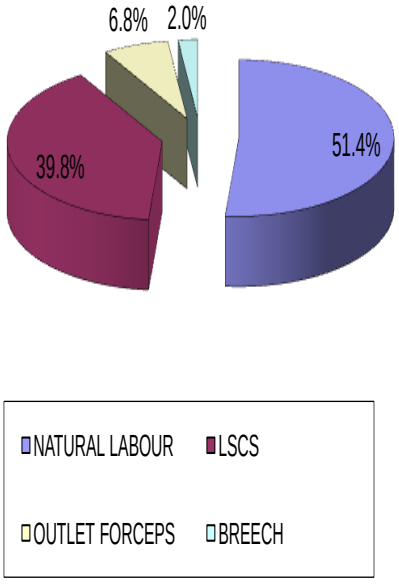


Table - 3

PULSE OXIMETRY SCREENING

Total number of babies screened	2437
Total number of babies with negative screening (Oxygen saturation ≥95%)	2413 (99%)
Total number of babies with positive screening (Oxygen saturation <95%)	24(1%)
Total number of CHD confirmed by Echocardiography in babies with positive pulse oximetry screening	17(70.8%)
Total number of babies with positive pulse oximetry screening but having NORMAL HEART confirmed by Echocardiography	7(29.2%)

2,231(91.5%) new borns had oxygen saturation values greater or equal to 95%, while 206(8.5%) newborns had oxygen saturation less than 95%. Out of 206 babies 3 babies had oxygen saturation less than 90%, 203 babies had oxygen saturation between 90%-94%. Pulse oximetry measurements were repeated after 6 hours for the 203 babies. The results showed that 21 babies had **persistent** low oxygen saturation between 90 to 94% and other 182 babies had oxygen saturation greater or equal to 95%. A total of 24 babies (1%) required echocardiography because of low oxygen saturation values (3 babies-<90%, 21 babies 90-94%).

3 echocardiograms were done based on low pulse oximetry value < 90%. One had saturation of 88% and was diagnosed with TRICUSPID ATRESIA. Another baby with saturation of 85% was diagnosed as d-TGA. The third baby had PULMONARY ATRESIA with a saturation of 83%.

21 Echocardiograms were done based on low pulse oxymetry values between 90-94%. Among the 21 babies, one had a saturation of 91% and was diagnosed with TOF. Another baby with a saturation of 93% was diagnosed as ASD (OP) with AV canal defect. All other 19 babies had saturation of 94% and echocardiography results showed 7 normal hearts, 8 small PDA and 4 small ASD (OS).

Table - 4

OXYGEN SATURATION OF BABIES WITH NEGATIVE PULSE OXIMETRY SCREENING (SpO2 ≥ 95%)

OXYGEN SATURATION (%)	NUMBER OF BABIES	PERCENTAGE (%)
95	293	12.1
96	490	20.3
97	587	24.3
98	731	30.3
99	193	8.0
100	119	5.0
TOTAL	2413	100

OXYGEN SATURATION OF BABIES WITH NEGATIVE PULSE OXIMETRY SCREENING
(SpO2 > 95%)

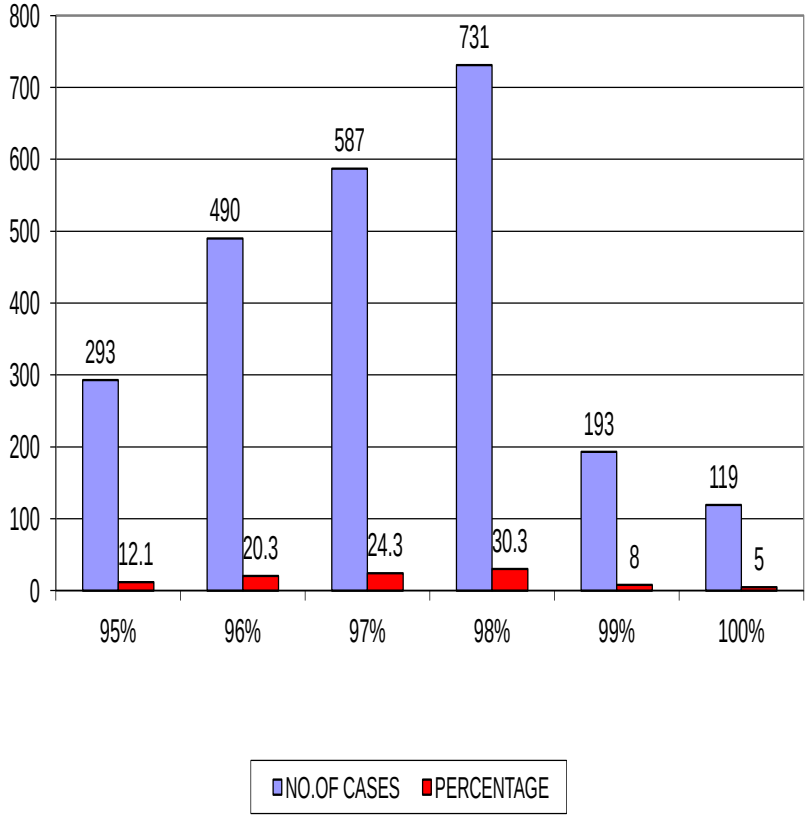


Table - 5

OXYGEN SATURATION OF BABIES WITH POSITIVE PULSE OXIMETRY SCREENING (SpO2 <95%)

Oxygen Saturation	No.of Babies
<90%	3
90%-94%	21
Total	24

Table - 6

ECHOCARDIOGRAPHY RESULT OF BABIES WITH OXYGEN SATURATION <90%

ECHOCARDIOGRAPHY RESULT	NUMBER	Oxygen Saturation
Tricuspid atresia with ASD (os),PDA, sub aortic VSD	1	88 %
d-TGA, ASD, PDA	1	85%
Pulmonary atresia with VSD, PDA	1	83%
TOTAL	3	

OXYGEN SATURATION OF BABIES WITH POSITIVE PULSE OXIMETRY SCREEING (SpO2 < 95%)

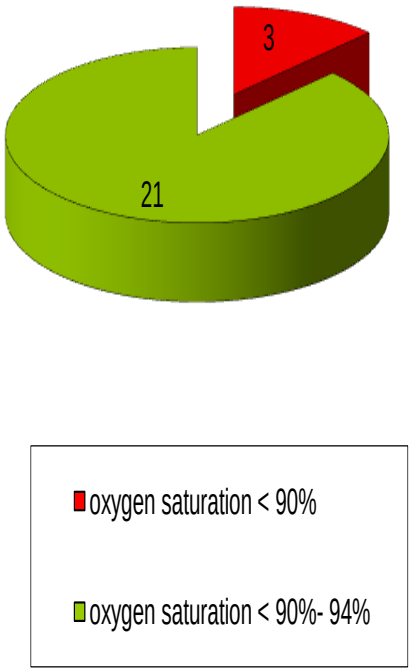


Table - 7

ECHOCARDIOGRAPHY RESULT OF BABIES WITH OXYGEN SATURATION 90%-94%

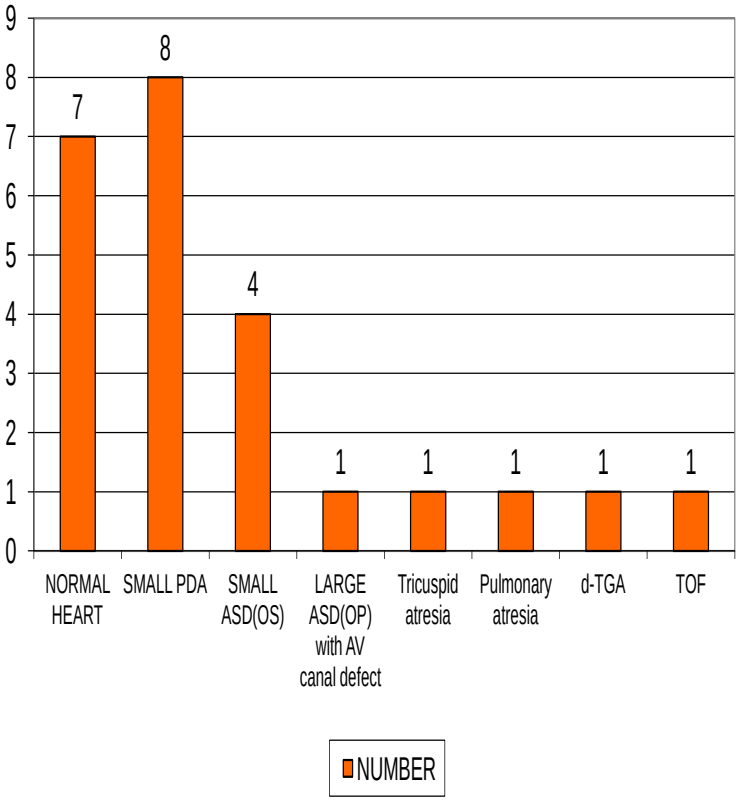
ECHOCARDIOGRAPHY RESULT	NUMBER
NORMAL HEART	7
SMALL PDA	8
SMALL ASD(OS)	4
LARGE ASD(OP) with AV canal defect	1
TOF	1
TOTAL	21

Table - 8

ECHOCARDIOGRAPHY RESULTS OF BABIES WITH POSITIVE PULSE OXIMETRY SCREENING

ECHOCARDIOGRAPHY RESULT	NUMBER	PERCENTAGE
CONGENITAL HEART DISEASE	17	71
NORMAL HEART	7	29
TOTAL	24	100

ECHOCARDIOGRAPHY RESULT OF BABIES WITH POSITIVE PULSE OXIMETRY SCREENING



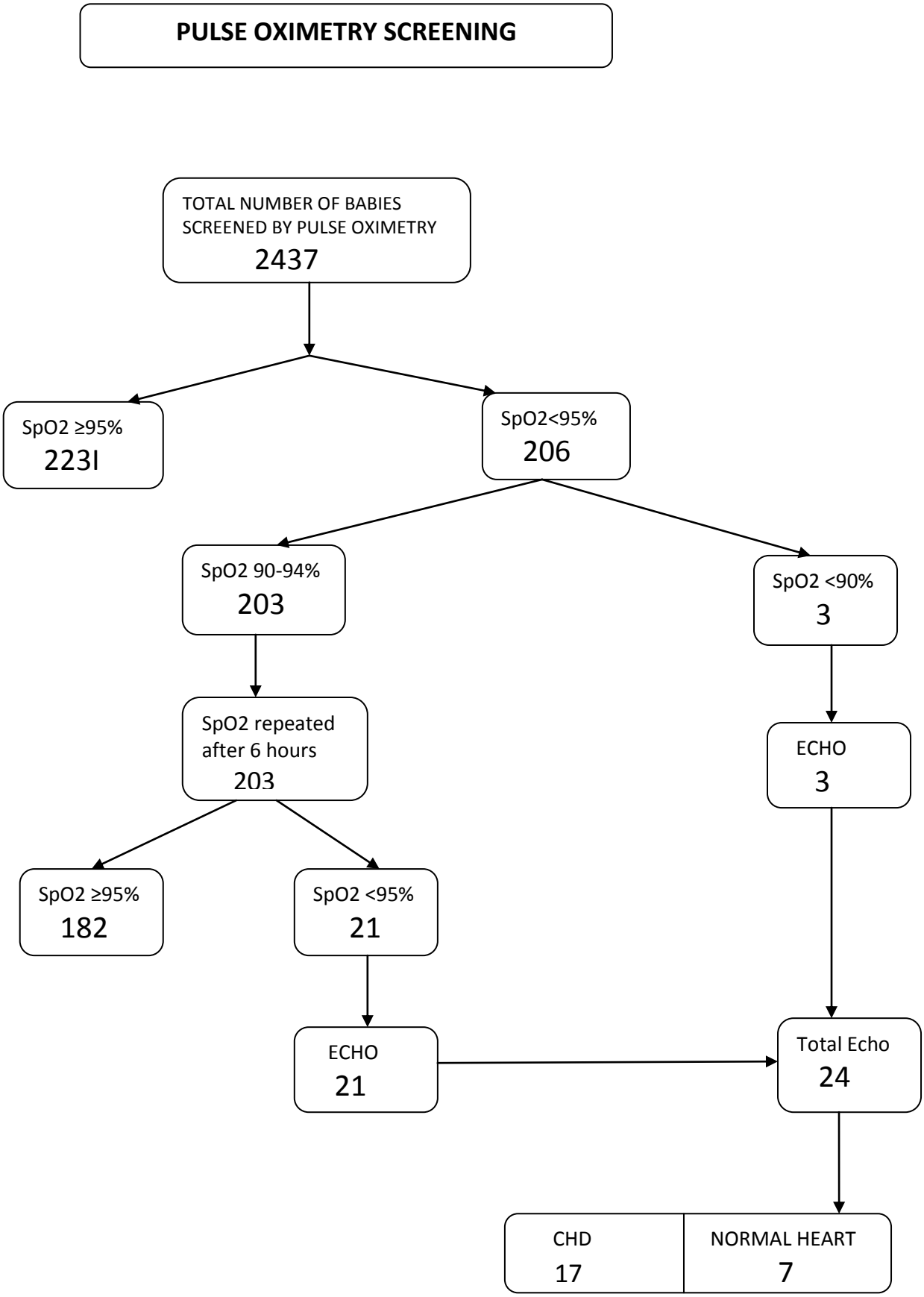

PULSE OXIMETRY SCREENING
TOTAL NUMBER OF BABIES SCREENED BY PULSE OXIMETRY
2437
SpO2 ≥95%
223I
SpO2<95%
206
SpO2 90-94%
203
SpO2 <90%
3
SpO2 repeated after 6 hours
203
ECHO
3
SpO2 ≥95%
182
SpO2 <95%
21
ECHO
21
Total Echo
24
CHD
17
NORMAL HEART
7

Table - 9

CARDIAC CLINICAL EXAMINATION

Total number of babies screened	2437
Total number of babies with negative screening	2401 (98.5%)
Total number of babies with positive screening	36(1.4%)
Total number of CHD confirmed by Echo Cardiography in babies with positive clinical examination screening	25(69%)
Total number of babies with positive clinical examination screening but having NORMAL HEART confirmed by Echocardiography	11(31%)

Cardiac clinical examination screening was performed simultaneously with pulse oximetry screening. Babies were looked for murmur and cyanosis. 36 babies had murmur and were considered screening positive. None of the baby had central cyanosis.

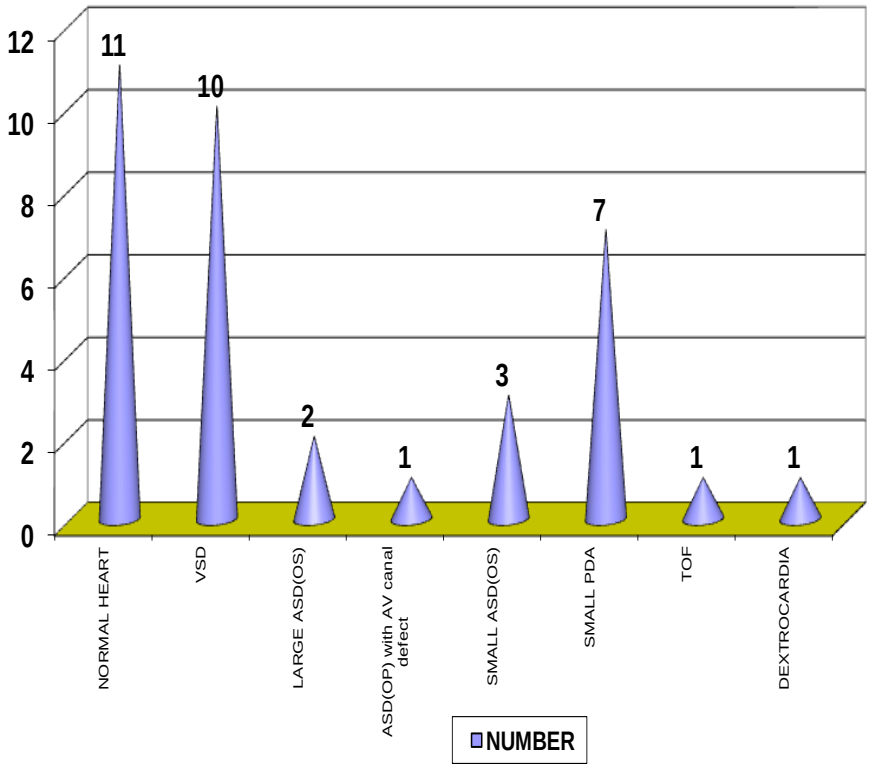
36 echocardiograms were done based on presence of murmur. Among the 36 babies, echocardiography showed 10 cases of VSD, 2 cases of large ASD(OS),1 case of ASD(OP) with AV canal defect, 3 cases of small ASD, 7 cases of small PDA, 1 case of TOF, and 1 case of isolated dextrocardia. 11 of them showed normal heart. Among these TOF,ASD(OP) with AV canal defect,3 cases of small PDA also had low oxygen saturation in addition to murmur.

Table - 10

ECHOCARDIOGRAPHY RESULT OF BABIES WITH POSITIVE CLINICAL EXAMINATION

ECHOCARDIOGRAPHY RESULT	NUMBER
NORMAL HEART	11
VSD	10
LARGE ASD(OS)	2
ASD(OP) with AV canal defect	1
SMALL ASD(OS)	3
SMALL PDA	7
TOF	1
DEXTROCARDIA	1
TOTAL	36

ECHOCARDIOGRAPHY RESULT OF BABIES WITH POSITIVE CLINICAL EXAMINATION SCREENING



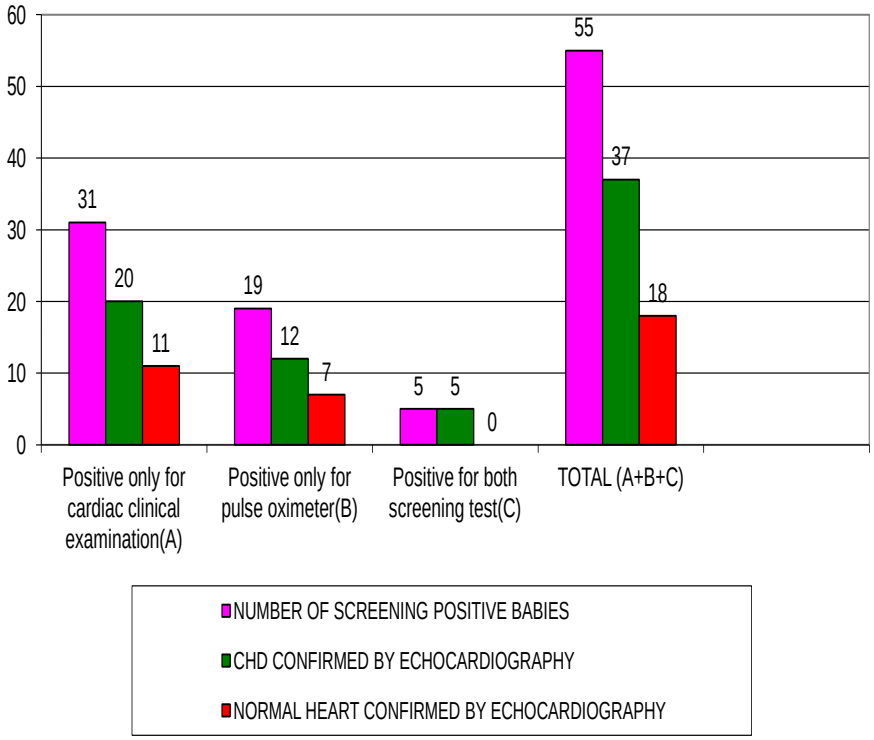
SUMMARY

In our study 2437 babies were screened by pulse oximetry and clinical examination. 31 babies were positive only for clinical examination screening. Out of 31, 20 babies had congenital heart disease.19 babies were positive only for pulse oximetry screening. Out of 19, 12 babies had congenital heart disease including 3 life threatening cyanotic congenital heart disease. 5 babies were positive for both screening tests and all 5 had congenital heart disease. In total, 55 babies were screening positive of which 37 babies had congenital heart disease. Our study showed that combined screening method identified more number of cases with congenital heart diseases than any single method of screening. It was found to be statistically significant. (P value-0.0235)

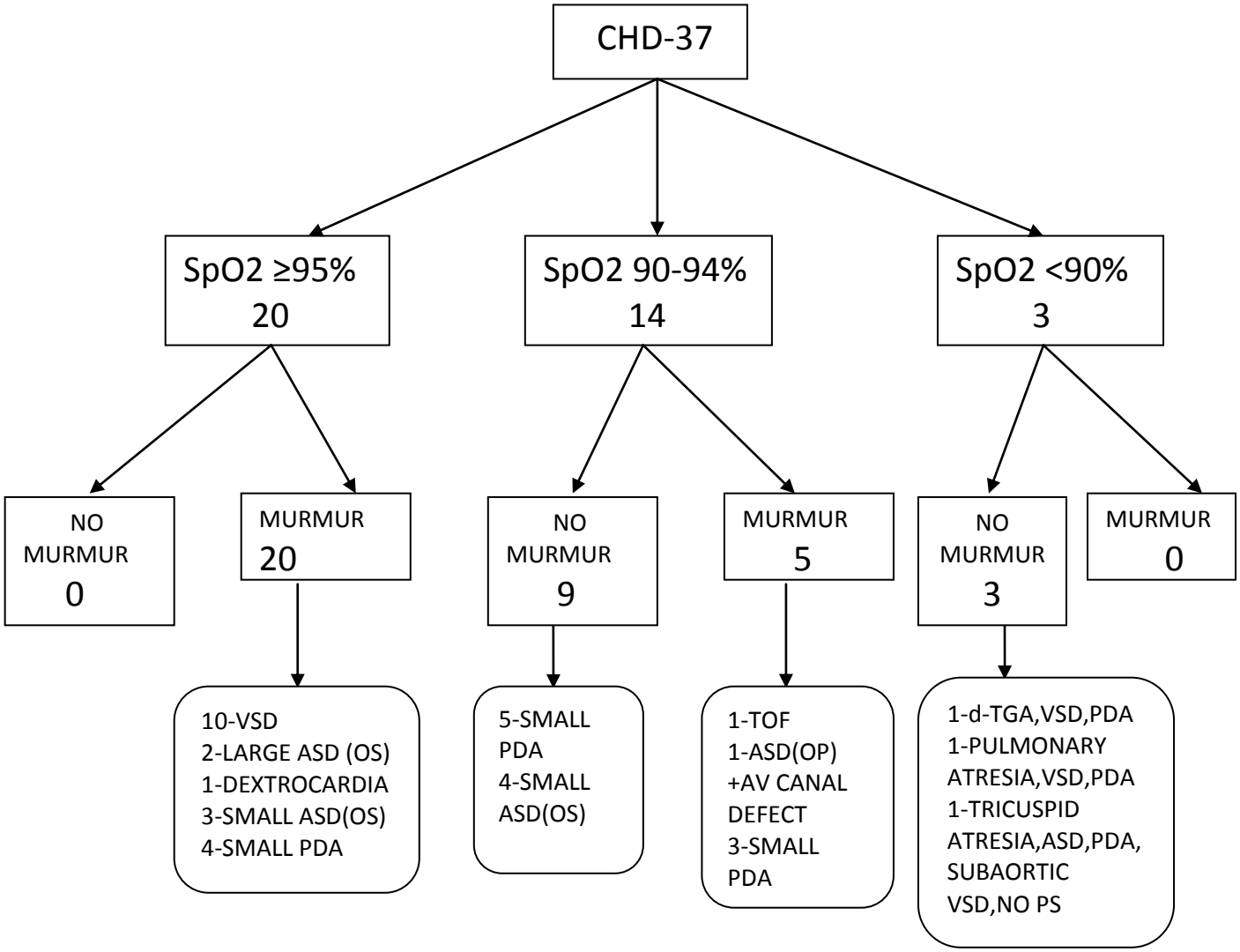
Table - 11

SCREENING METHOD	NUMBER OF BABIES	CHD CONFIRMED BY ECHO	NORMAL HEART CONFIRMED BY ECHO
Positive only for cardiac clinical examination(A)	31	20	11
Positive only for pulse oximeter(B)	19	12	7
Positive for both screening test(C)	5	5	0
TOTAL NO.OF SCREENING POSITIVE BABIES(A+B+C)	55	37	18

SUMMARY OF SCREENING POSITIVE BABIES AND THEIR ECHOCARDIOGRAPHY RESULTS



SCREENING CHARACTERISTICS OF CHD



DISCUSSION

Some babies born with a cardiac defect may appear healthy soon after birth and may be sent home before their cardiac defect is detected. These babies are at risk of developing serious complication within the first few days or weeks of life and may have significant morbidity and mortality. Routine pulse oximetry screening may help in early identification of these babies before they land up in complications.

EFFECTIVENESS OF PULSE OXIMETRY SCREENING

In this study abnormal pulse oximetry was found in 24 babies of which 17 had CHD on echocardiography.5 babies had major CHD [1-Transposition of the Great Arteries(d-TGA),1- Pulmonary Atresia (PA), 1- Tricuspid Atresia (TA), 1- Tetralogy of Fallot (TOF), 1-ASD(OP) with AV canal defect], 12 babies had minor CHD[4-small ASD, 8-small PDA]. Out of 17 CHD babies, 5 babies had murmur [1-TOF,1-ASD(OP) with AV canal defect,3-small PDA] and 12 babies had no murmur.

This study showed that pulse oximetry can detect CHD in asymptomatic new born babies which have been missed by routine clinical examination. Notably pulse oximetry identified cases of life threatening complex cyanotic CHD such as Transposition of great arteries (TGA), Tricuspid Atresia (TA), Pulmonary Atresia (PA), none of which had been detected clinically.

Pure left-to-right shunts such as ventricular septal defect, atrial septal defect, or patent ductus arteriosus should not be detected by pulse oximetry, but some of these defects (5-17%) proved to be screen positive.[10,37,75,104] This happened probably due to bidirectional shunting during early postnatal pulmonary hypertension.

TIME OF PULSE OXIMETRY SCREENING

If pulse oxymetry screening is performed after a few days of life, there will be a reduced incidence of false positives, because of the physiologic decrease in the pulmonary vascular resistance, but a newborn with a ductal-dependant CHD could deteriorate rapidly if the ductus arteriosus had already closed. Measurements performed shortly after birth may lead to an increased number of false positives.[10,104,102]

In this study the babies were screened between 24-48 hours of age in the view of early detection of CHD and to decrease the number of false positive results. The false positive rate of our study is 0.28% which is slightly higher than other studies. This higher false positive rate is due to early screening of healthy babies with delayed transitional circulation.

SATURATION CUT-OFF

Our decision to use a 95% cut-off was based on published normal pulse oximetry values in healthy newborns[66,69] and saturation differences observed in infants with left obstructive heart disease and obligate right to

left shunt across the ductus arteriosus.[60] Pulse oximetry is known to overestimate arterial oxygen saturation at low saturations and underestimate it at high saturations.[84,65,60] The sensitivity and specificity remained quite stable using a cut-off ranging from 92% to 95%, whereas a cut-off below 92% led to a rapid decrease of sensitivity.

COMPARISON OF OUR STUDY WITH OTHER PULSE OXIMETRY STUDIES

Comparison with literature is difficult because the study designs, methods, target CHD, technical details and way of expressing the results are different.

A case-control study, **(Hoke et al; 2002)** included 2,972 newborns admitted to well baby nurseries and 32 newborns with CHD. An oxygen saturation < 92% in the leg or a saturation 7% lower in the leg than in the arm was defined as abnormal. Of the well babies, 57 had an abnormal pulse oximetry reading; of these, four babies were found to have congenital heart defects, including one case of coarctation of the aorta. A fifth baby was found to have pulmonary hypertension. Of the 32 newborns with congenital heart disease, 11/13 (85%) with left-heart obstructive disease had abnormal oxygen saturation as did 15/19 (79%) of babies with other congenital heart defects[73].

Reich et al; 2003, Babies were assessed with a single pulse oximetry reading. Of 2,335 eligible newborns, those with an oxygen saturation ≤ 94% that persisted with a second reading were further assessed by echocardiogram. An echocardiogram was performed in 88 infants, which was abnormal in 43 babies, and 13 required management. In this study, pulse oximetry was nearly 100% specific for detecting cyanotic congenital heart disease; as a result, there was no increase in the number of echocardiograms generated[26].

Koppel et al; 2003 assessed the sensitivity, specificity, predictive value and accuracy of pulse oximetry screening of 11,281 asymptomatic newborns in two hospitals. Diagnostic echocardiogram was performed in infants with oxygen saturation ≤ 95% within the first 24 hours of life. Three babies with CHD were identified (but none had a critical left-heart obstructive lesion). Two patients with negative screens were later readmitted; one with aortic coarctation and one with hypoplastic left pulmonary artery with aorto-pulmonary collaterals. No unnecessary echocardiograms were generated by the study. The sensitivity of the screening test was calculated to be 60%, with a specificity of 99.95%, a positive predictive value of 75% and a negative predictive value of 99.98%.[11] Our study also did not identify critical left heart obstructive

lesion like Hypoplastic left heart syndrome, coarctation of aorta, critical aortic stenosis.

Arlettaz et al; 2006 evaluated the effectiveness of pulse oximetry screening to detect structural CHD on the first day of life in all babies (n=3,663) born during a one-year period who were > 35 weeks gestation and had no respiratory distress. Also evaluated was whether pulse oximetry combined with clinical examination is superior to clinical exam alone to diagnose CHD. Oxygen saturation readings from one foot were considered normal with oxygen saturations > 95%. Twenty-four infants had repeated oxygen saturations < 95% and had an echocardiogram. Of these, 17 had CHD and five had pulmonary hypertension. One child had a myocardial tumour and one infant had a normal heart. No false negative cases were reported [75]. This study showed high detection rate of CHD by pulse oximetry. This may be due to inclusion of babies with CHD diagnosed prenatally.

Bakr et al: 2005 evaluated combined pulse oximetry and clinical examination as a screening method for CHD in all asymptomatic infants (excluding those admitted to the neonatal unit) born during a six-month period. Of 5,211 infants, five babies were identified with an oxygen saturation <94%, which was considered abnormal. One infant had total anomalous pulmonary venous return, one had pulmonary atresia, one had

a large ventricular septal defect and another child had a truncus arteriosus. The fifth child had a normal heart. Three babies were later diagnosed with CHD that was not detected by pulse oximetry screening when being investigated for failure to thrive and respiratory symptoms. The sensitivity of the combined method of screening was 77%, whereas it was 30.8% for oximetry alone and 46% for clinical exam alone, Specificity was approximately 100% for all methods. The positive predictive value was 80% for pulse oximetry alone [37].

Table - 12

DETECTION OF CRITICAL CONGENITAL HEART DISEASE

(CCHD) LESIONS FROM PULSE OXIMETRY SCREENING STUDIES

CHD	Hoke[73]	Richmond[10]	Koppel[11]	Reich[26]	Bakr[37]	Rosati[74]	Arlettaz[75]	Kawalec[53]	Our study
NO OF BABIES SCREENED	2876	5626	11281	2114	5211	5292	3262	27200	**2437**
DORV	0	0	0	0	0	0	3	0	**0**
HLHS	0	0	0	0	0	0	3	2	**0**
PA	0	3	0	0	1	0	1	0	**1**
d-TGA	1	3	0	1	0	0	2	0	**1**
TAPVC	0	0	2	1	1	1	0	1	**0**
TRUNCUS	0	0	1	2	1	0	3	0	**0**
TA	0	0	0	0	0	0	0	1	**1**
AA/AS	0	0	0	0	0	0	1	0	**0**
TOF	1	1	0	2	0	0	0	0	**1**
AVSD	0	0	0	1	0	0	1	0	**0**
CoA	1	2	0	0	0	1	1	3	**0**
PS	1	0	0	0	0	0	1	0	**0**
TOTAL	4	9	3	7	3	2	16	7	**4**

Table – 13

PULSE OXIMETRY SCREENING STUDIES FOR CRITICAL CONGENITAL HEART DISEASE (CCHD)

First author	No of babies screened	Age at screening (h)	Probe location	Cut off normal	False positive	False positive rate(%)	True positive
Hoke[73]	2876	<24	H&F	≥92	53	1.84	4
Richmond[10]	5626	11.7	F	≥95	51	0.91	9
Koppel11	11281	72	F	≥96	1	0.01	3
Reich [26]	2114	>24	H&F	≥95	2	0.09	1
Bakr [37]	5211	31.7	H&F	≥94	1	0.02	3
Rosati [74]	5292	72	F	≥96	1	0.02	2
Arlettaz[75]	3262	8	F	≥95	7	0.21	17
Kawalec[53]	27200	26	F	≥95	13	0.05	7
Meberg[104]	50008	6	F	≥95	324	0.65	43
Sendelbach[6]	10976	4	F	≥96	636	4.5	0
Our study	**2437**	**36**	**F**	**≥95**	**7**	**0.28**	**4**

EFFECTIVENESS OF CLINICAL EXAMINATION SCREENING

In our study 2437 healthy new born babies were screened. Out of the 2437 babies 36 had heart murmur. The prevalence of murmur was 14.8 per 1000 of normal new borns during the period of study. The reported prevalence of heart murmur in neonate varies from 6 per 1000 to 42 per 1000. These variations may be due to the examiner's skills and experience, the timing and frequency of examination, the conditions under which examination takes place and the size of population studied[24].

All those babies with murmur underwent echocardiography. CHD were detected in 25(69.4%) babies and 11 (30.6%) babies had structurally normal heart. Out of the 25 babies with CHD, 10 (40%) babies had VSD which was the common lesion.

Table - 14

COMPARISON OF OUR STUDY WITH OTHER CARDIAC CLINICAL EXAMINATION SCREENING STUDIES

First author	No of babies screened	No of babies with murmur	Prevalence of murmur per 1000 babies	Babies with CHD confirmed by ECHO	Babies with normal heart confirmed by ECHO
BANSAL[27]	2603	62	23.8	46(74.2%)	16(25.8%)
LARDHI[25]	6333	87	13.7	61(70.1%)	26(29.9%)
MEHRDED[35]	2928	91	31.1	47(51.6%)	44(48.4%)
AINSWORTH[24]	7204	46	6.3	25(54.3%)	21(45.7%)
OUR STUDY	**2437**	**36**	**14.8**	**25(69.4%)**	**11(30.6%)**

Table – 15

FIRST AUTHOR	NO OF BABIES WITH CHD	NO BABIES WITH VSD
BANSAL[27]	46	19(41.3%)
LARDHI[25]	61	25(40.9%)
MEHRDED[35]	47	16(34.0%)
AINSWORTH[24]	25	15(60.0%)
OUR STUDY	**25**	**10(40.0%)**

Tanner et al observed innocent murmur in 44(48.4%) and congenital structural heart defects in (51.6%) newborns[38]. Rein et al found that 24% of heart murmurs in neonates were innocent.[61] Another study has shown that the prevalence of heart murmur is 13.7 per 1000 neonates. If a murmur is heard, there is a 42.5% chance of presence of an underlying cardiac malformation.[86] Yet another study stated that 84% of heart murmurs in neonates were caused by heart diseases and only16% were innocent and urged subsequent echocardiography.[21] Many types of congenital heart disease may be associated with an asymptomatic murmur, the most common being ventricular septal defect (VSD)[27,25,35] Our study also showed similar results. We found that 30.6% of heart murmurs were innocent and 69.4% of heart murmurs had underlying CHD ; among them VSD was the most common lesion (40%).

EFFECTIVENESS OF COMBINED [PULSE OXIMETRY+ CLINICAL EXAMINATION] SCREENING

In this study, we evaluated the efficacy of combining pulse oximetry with clinical examination in screening for CHD. In our study, CHD was detected in 37 babies (20 babies by clinical examination screening +12 babies by pulse oximeter screening +5 babies were positive for both screening test). Out of them 25 babies had murmur and 12 babies had no significant positive clinical examination including

murmur, but had abnormal pulse oximetry. Of the 12 babies 3 babies had cyanotic congenital heart disease.

Our study showed that pulse oximetry can detect CHD in asymptomatic newborns which was missed by routine clinical examination especially life threatening cyanotic congenital heart disease. Clinical examination also picked up another group of cases that were missed by pulse oximetry. Most of the acyanotic heart diseases were detected by means of a murmur. So the combined approach had an additive effect and resulted in more efficient screening.

CONCLUSION

- This study indicates that pulse oximetry is a noninvasive, reliable and useful screening tool for an early detection of congenital heart diseases especially cyanotic congenital heart diseases.
- The normal oxygen saturation (negative pulse oximetry screening) does not rule out CHD especially acyanotic congenital heart disease.
- The prevalence of murmurs detected at routine examination of neonates is 1.48%.
- About 69.4% murmurs were due to an underlying cardiovascular malformation. So detection of a cardiac murmur may be a clue to the presence of an underlying heart disease particularly in asymptomatic newborn.
- The absence of a murmur does not exclude serious heart disease.
- This study concludes that the combination of pulse oximetry and clinical examination results in early detection of CHD. Pulse oximetry has an additive effect and results in more efficient screening.
- This study suggests that combined screening (pulse oximetry + cardiac clinical examination) should be used as a screening method for detection of CHD.

LIMITATIONS

- Small sample size
- For all babies, a follow up evaluation was not performed.
- Sensitivity and specificity could not be calculated since echocardiography was not performed on screening negative babies to know the true negative and false negative cases.

RECOMMENDATIIONS

This study recommends pulse oximetry to be added to routine clinical examination as a screening method for early detection of CHD.

BIBLIOGRAPHY

1. Hoffman JIE, Kaplan S. The incidence of congenital heart disease. JACC. 2002;39:18 90-900.

2. Abu-Harb M, Hey E, Wren C. Death in infancy from unrecognized congenital heart disease. Arch Dis Child. 1994;71: 3-7.

3. Koenig P, Ziyad MH, Zimmerman F. Essential Pediatric Cardiology. NewYork: The McGraw-Hill Companies; 2004.

4. Sunnegårdh J. Barnkardiologi-en översikt. Lund: Studentlitteratur; 2000.

5. Srivastava D, Baldwin HS. Molecular determinants of cardiac development. In: Allen HD, Gutgesell HG, Clark EB, Driscoll DJ, eds. Moss and Adams' Heart Disease in infants, children, and adolescents including the fetus and young adult. Vol 1. 6th ed. Philadelphia: Lippincott Williams & Wilkins; 2001:1-20.

6. Sendelbach DM, Jackson GL, Lai SS, Fixler DE, Stehel EK, Engle WD. Pulse oximetry screening at 4 hours of age to detect critical congenital heart defects. Pediatrics. Oct 2008;122(4):e 815-820.

7. Pradat P, Francannet C, Harris JA, Robert E. The epidemiology of cardiovascular defects, part I: a study based on data from three large registries of congenital malformations. Pediatr Cardiol. May-Jun 2003;24(3):195-221.

8. Ferencz C, Neill CA. Cardiovascular malformations: prevalence at livebirth. In: Freedom RM, Benson LN, Smallhorn JF, eds. Neonatal Heart Disease. London: Springer-Verlag; 1992:19-29.

9. Reich JD, Connolly B, Bradley G, Littman S, Koeppel W, Lewycky P, Liske M. Reliability of a Single Pulse Oximetry Reading as a Screening Test for Congenital Heart Disease in Otherwise Asymptomatic Newborn Infants: The Importance of Human Factors. Pediatr Cardiol. Oct 12 2007.

10. Richmond S, Reay G, Abu Harb M. Routine pulse oximetry in the asymptomatic newborn. Arch Dis Child Fetal Neonatal Ed. Sep 2002;87(2):F83-88.

11. Koppel RI, Druschel CM, Carter T, Goldberg BE, Mehta PN, Talwar R, Bierman FZ. Effectiveness of pulse oximetry screening for congenital heart disease in asymptomatic newborns. Pediatrics. Mar 2003;111(3):451-455.

12. Wren C, Reinhardt Z, Khawaja K. Twenty-year trends in diagnosis of lifethreatening neonatal cardiovascular malformations. Arch Dis Child Fetal Neonatal Ed. Jan 2008;93(1):F33-35.

13. Boneva RS, Botto LD, Moore CA, Yang Q, Correa A, Erickson JD. Mortality associated with congenital heart defects in the United States: trends and racial disparities, 1979 –1997. Circulation. 2001;103:2376 – 2381.

14. Keuhl KS, Loffredo CA, Ferencz C. Failure to diagnosed congenital heart disease in infancy. Pediatrics. 1999; 103:743-747.

15. Byard RW, Moore L (1991) Total anomalous pulmonary venous drainage and sudden death in infancy. Forens Sci Intl 51:197–202

16. Grech V (2000) Diagnostic and interventional trends in tetralogy of Fallot and transposition of the great arteries in a population-based study. Pediatr Cardiol 21:368–373

17. Silove ED (1994) Assessment and management of congenital heart in the newborn by the district paediatrician. Arch Dis Child 70:F71–F74

18. Varan B, Tokel K, Yilmaz G (1999) Malnutrition and growth failure in cyanotic and acyanotic congenital heart disease with and without pulmonary hypertension. Arch Dis Child 81:49–52

19. Archer N (1999) Cardiovascular disease. In: Rennie J, Roberton N (eds) Textbook of neonatology, 3rd edn. Churchill Livingstone, Edinburgh, pp 673–713

20. Danford DA (1995) Cost-effectiveness of echocardiography for evaluation of children with murmurs. Echocardiography 12:153–162

21. Du ZD, Roguin N, Barak M (1997) Clinical and echocardiographic evaluation of neonates with heart murmurs. Acta Paediatr 86:752–756

22. Frommelt MA (2004) Differential diagnosis and approach to a heart murmur in term infants. Pediatr Clin N Am 51:1023–1032

23. Wren C, Richmond S, Donaldson L (1999) Presentation of congenital heart disease in infancy: implications for routine examination. Arch Dis Child Fetal Neonatal Ed. 80:F49–F53

24. Ainsworth SB, Wyllie JP, Wren C (1999) Prevalence and clinical significance of cardiac murmurs in neonates. Arch Dis Child Fetal Neonatal Ed 80:F43–F45

25. Amer Abdullah Lardhi. Prevalence and clinical significance of heart murmurs detected in routine neonatal examination. Journal of the Saudi Heart Association Volume 22, Issue 1 , Pages 25-27, January 2010

26. Reich JD, Miller S, Brogdon B, et al. (2003) The use of pulse oximetry to detect congenital heart disease. Pediatrics 111: 451–455

27. Bansal M, Jain H. Cardiac murmur in neonates. Indian Pediatr. 2005;42:397–398

28. Allan L, Hornberger LK, Sharland G. Textbook of Fetal Cardiology. London: Greenwich Medical Media; 2000.

29. McLachlan J. Medical Embryology. Wokingham: Addison-Wesley; 1994.

30. Henderson D, Hutson MR, Kirby ML. Embryos, Genes and Birth Defects. In: Ferretti P, Chopp A, Tickle C, Moore G, eds. 2 ed. Chichester: John Wiely & Sons; 2006:342-363.

31. Rose V, Clark E. Etiology of congenital heart disease. In: Freedom RM, Benson LN, Smallhorn JF, eds. Neonatal Heart Disease. London: Springer- Verlag; 1992:3-17.

32. Liske MR, Greeley CS, Law DJ, Reich JD, Morrow WR, Baldwin HS, Graham TP, Strauss AW, Kavanaugh-McHugh AL, Walsh WF. Report of the Tennessee Task Force on Screening Newborn Infants for Critical Congenital Heart Disease. Pediatrics. Oct 2006;118(4):e1250-1256.

33. Rosati E, Chitano G, Dipaola L, De Felice C, Latini G. Indications and limitations for a neonatal pulse oximetry screening of critical congenital heart disease. J Perinat Med. 2005;33(5):455-457.

34. Mellander M, Sunnegardh J. Failure to diagnose critical heart malformationsin newborns before discharge--an increasing problem? Acta Paediatr. Apr 2006;95(4):407-413.

35. Mehrdad Mirzarahimi. Heart Murmur in Neonates: How Often Is It Caused by Congenital Heart Disease? Iran J Pediatr Mar 2011; Vol 21 (No 1), Pp: 103-106.

36. Abu-Harb M, Wyllie J, Hey E. et al. Presentation of obstructive left heart malformations in infancy. Arch Dis Child Fetal Neonatal Ed 1994. 71 179–183.

37. Bakr A F, Habib H S. Combining pulse oximetry and clinical examination in screening for congenital heart disease. Pediatr Cardiol 2005. 26 832–835

38. Tanner K, Sabrine N, Wren C. Cardiovascular malformations among preterm infants. Pediatrics 2005;116(6):e 833-8.

39. Ward K E, Pryor R W, Matson J R. et al Delayed detection of coarctation in infancy : implications for timing of new born follow up. Paediatrics 1990, 86 972, pg 976.

40. Garne E, Stoll C, Clementi M. et al Evaluation of prenatal diagnosis of heart diseases by ultrasound: experience from 20 European countries. Ultrasound Obstet Gynecol 2001. 17 386–391.

41. Allan LD. A practical approach to fetal heart scanning. Semin Perinatol. 2000;24:324 –330

42. Acharya G, Sitras V, Maltau JM, Dahl LB, Kaaresen PI, Hanssen TA, Lunde P. Major congenital heart disease in Northern Norway:

shortcomings of pre- and postnatal diagnosis. Acta Obstet Gynecol Scand. 2004;83:1124 –1129

43. Hunter S, Heads A, Wyllie J, Robson S. Prenatal diagnosis of congenital heart disease in the northern region of England: benefits of a training programme for obstetric ultrasonographers. Heart. 2000;84:294 –298

44. Klein SK, Cans C, Robert E, Jouk PS. Efficacy of routine fetal ultrasound screening for congenital heart disease in Ise` re County, France. Prenat Diagn. 1999;19:318 –322

45. Randall P, Brealey S, Hahn S, Khan KS, Parsons JM. Accuracy of fetal echocardiography in the routine detection of congenital heart disease among unselected and low risk populations: a systematic review. BJOG. 2005;112:24 –30

46. Tegnander E, Williams W, Johansen OJ, Blaas HG, Eik-Nes SH. Prenatal detection of heart defects in a non-selected population of 30,149 fetuses: detection rates and outcome. Ultrasound Obstet Gynecol. 2006;27:252–265

47. Westin M, Saltvedt S, Bergman G, Kublickas M, Almstro¨m H, Grunewald C, Valentin L. Routine ultrasound examination at 12 or 18 gestational weeks for prenatal detection of major congenital heart malformations? A randomised controlled trial comprising 36,299 fetuses. BJOG. 2006;113:675– 682

48. Queisser-Luft A, Stopfkuchen H, Stolz G, Schlaefer K, Merz E. Prenatal diagnosis of major malformations: quality control of routine ultrasound examinations based on a five-year study of 20,248 newborn fetuses and infants. Prenat Diagn. 1998;18:567–576

49. Timor-Tritsch IE. As technology evolves, so should its application: shortcomings of the “18-week anatomy scan.” J Ultrasound Med. 2006;25:423– 428

50. Bofill JA, Sharp GH. Obstetric sonography: who to scan, when to scan, and by whom. Obstet Gynecol Clin North Am. 1998;25:465– 478

51. Forrester MB, Merz RD. Use of prenatal diagnostic procedures in pregnancies affected with birth defects, Hawaii, 1986 –2002. Birth Defects Res A Clin Mol Teratol. 2006;76:778 –780

52. Gavin NI, Adams EK, Hartmann KE, Benedict MB, Chireau M. Racial and ethnic disparities in the use of pregnancy-related health care among Medicaid pregnant women. Matern Child Health J. 2004;8:113–126

53. Kawalec W, Blaz W, Turska-Kmiec A, Zuk M, Helwich E, Tobota Z. Pulse oximetry as a population screening test in detection of critical congenital heart disease in presymptomatic newborns: Polish multicentre study. Cardiol Young. 2006;16(suppl 2):25. Abstract

54. Danford DA. Sorting through the haystack: decision analysis and the search for heart disease among children with murmur. J Pediatr. 2002;141:465– 467

55. Griebsch I, Knowles RL, Brown J, Bull C, Wren C, Dezateux CA. Comparing the clinical and economic effects of clinical examination, pulse oximetry, and echocardiography in newborn screening for congenital heart defects: a probabilistic cost-effectiveness model and value of information analysis. Int J Technol Assess Health Care. 2007;23:192–204

56. Gidding SS, Anisman P. What pediatric residents should learn (or what pediatricians should know) about congenital heart disease. Pediatr Cardiol. 2003;24:418–423.

57. Patton C, Hey E. How effectively can clinical examination pick up congenital heart disease at birth? Arch Dis Child Fetal Neonatal Ed. 2006;91:F263–F267.

58. Lundsgaard C, Van Slyke DD. Cyanosis. Medicine. 1923;2:1–76

59. Lees MH. Cyanosis of the newborn infant: recognition and clinical evaluation. J Pediatr. 1970;77:484–498

60. Schmitt HJ, Schuetz WH, Proeschel PA, Jaklin C (1993) Accuracy of pulse oximetry in children with cyanotic congenital heart disease. J Cardiothorac Vasc Anesth 7:61–65

61. Rein AJ, Omokhodion SI, Nir A. Significance of a cardiac murmur as the sole clinical sign in the newborn. Clin Pediatr (Phila) 2000;39(9):511-20

62. O'Donnell CP, Kamlin CO, Davis PG, Carlin JB, Morley CJ. Clinical assessment of infant colour at delivery. Arch Dis Child Fetal Neonatal Ed. 2007;92:F465–F467

63. Katzman GH. The newborn's SpO2: a routine vital sign whose time has come? Pediatrics. 1995;95:161–162.

64. Toth B, Becker A, Seelbach-Gobel B. Oxygen saturatin in healthy Newborn infants immediately after birth measured by pulse oximetry. Arch Gynecol Obstet 2002. 266 105–107.

65. Gidding SS (1992) Pulse oximetry in cyanotic congenital heart disease. Am J Cardiol 70:391–392

66. O'Brien L M, Stebbens V A, Poets C F. et al Oxygen saturation during the first 24 hours of life. Arch Dis Child Fetal Neonatal Ed 2000. 83 F35–F38.

67. Poets CF. Assessing oxygenation in healthy infants. J Pediatr. 1999;135:541–543

68. Poets CF, Stebbens VA, Lang JA, O'Brien LM, Boon AW, Southall DP. Arterial oxygen saturation in healthy term neonates. Eur J Pediatr. 1996;155:219 –223

69. Levesque BM, Pollack P, Griffin BE, Nielsen HC. Pulse oximetry: what's normal in the newborn nursery? Pediatr Pulmonol. 2000;30:406-412.

70. Byrne B J, Donohue P K, Bawa R D. et al Oxygen saturation as a screening test for critical congenital heart disease [abstract]. Pediatr Res 1995. 37(Suppl)198A.

71. Thangaratinam S, Daniels J, Ewer AK, Zamora J, Khan KS. Accuracy of pulse oximetry in screening for congenital heart disease in asymptomatic newborns: a systematic review. Arch Dis Child Fetal Neonatal Ed. 2007;92:F176 –F180

72. Niermeyer S, Shaffer EM, Thilo E, Corbin C, Moore LG. Arterial oxygenation and pulmonary arterial pressure in healthy neonates and infants at high altitude. J Pediatr. 1993;123:767–772

73. Hoke T R, Donohue P K, Bawa R D. et al Oxygen saturation as a screening test for critical congenital heart disease: a preliminary study. Pediatr Cardiol 2002. 23 403–409.

74. Rosati E, Chitano G, Dipaola L. et al Indications and limitations for a neonatal pulse oximetry screening of critical congenital heart disease. J Perinat Med 2005. 33 455–457.

75. Arlettaz R, Bauschatz A S, Mönkhoff M. et al The contribution of pulse oximetry to the early detection of congenital heart disease in newborns. Eur J Pediatr 2006. 16 594–98.

76. Poets C F, Southall D P. Noninvasive monitoring of oxygenation in infants and children: Practical considerations and areas of concern. Pediatrics 1994. 93 737–746.

77. Mok JY, McLaughlin FJ, Pintar M, Hak H, Maro-Galvez R, Levison H. Transcutaneous monitoring of oxygenation: what is normal? J Pediatr. 1986;108:365–371

78. Hay WW Jr, Rodden DJ, Collins SM, Melara DL, Hale KA, Fashaw LM. Reliability of conventional and new pulse oximetry in neonatal patients. J Perinatol. 2002;22:360 –366

79. Hay WW Jr. Physiology of oxygenation and its relation to pulse oximetry in neonates. J Perinatol.1987;7:309 –319

80. Severinghaus JW. History and recent developments in pulse oximetry. Scand J Clin Lab Invest Suppl. 1993;214:105–111

81. Barker SJ, Shah NK. Effects of motion on the performance of pulse oximeters in volunteers.Anesthesiology. 1996;85:774 –781

82. Clayton DG, Webb RK, Ralston AC, Duthie D, Runciman WB. Pulse oximeter probes: a comparison between finger, nose, ear and forehead probes under conditions of poor perfusion. Anaesthesia.1991;46:260 –265

83. Poets CF, Stebbens VA. Detection of movement artifact in recorded pulse oximeter saturation. Eur J Pediatr. 1997;156:808–811

84. Gerstmann D, Berg R, Haskell R, Brower C, Wood K, Yoder B, Greenway L, Lassen G, Ogden R,Stoddard R, Minton S. Operational evaluation of pulse oximetry in NICU patients with arterial access. J Perinatol. 2003;23:378 –383

85. Bickler PE, Feiner JR, Severinghaus JW. Effects of skin pigmentation on pulse oximeter accuracy at low saturation. Anesthesiology. 2005;102:715–719

86. Wu MH, Chen HC, Lu CW, et al. Prevalence of congenital heart disease at live birth in Taiwan. J Pediatr 2010;156(5):782-5.

87. Kamat V. Pulse Oximetry. Indian J Anaesth. 2002;46(4):261-268.

88. Barker SJ, Tremper KK. The effect of carbon monoxide inhalation on pulse oximetry and transcutaneous PO2. Anesthesiology. May 1987;66(5):677-679.

89. Salyer JW. Neonatal and pediatric pulse oximetry. Respir Care. Apr2003;48(4):386-396; discussion 397-388

90. Sola A, Chow L, Rodigo M. Pulse oximetry in neonatal care in 2005. A comprehensive state of the art review. An Pediatr 2005;62:266 - 280.

91. Valmari P. Should pulse oximetry be used to screen for congenital heart disease? Arch Dis Child Fetal Neonatal Ed. May 2007;92(3):F219-224.

92. Chan MM, Chan MM, Chan ED. What is the effect of fingernail polish on pulse oximetry? Chest. Jun 2003;123(6):2163-2164.

93. Rubin AS. Nail polish color can effect pulse oximeter saturation. Anesthesiology. 1988;68:825.

94. Shah NK, Barker SJ, Hyatt J. Comparison of alarm conditions between new pulse oximeters during motion at normal and lower oxygen saturations [abstract]. Anesthesiology. 1995;83:A1067.

95. Barker SJ, Shah NK. The effects of motion on the performance of pulse oximeters in volunteers (revised publication). Anesthesiology. Jan 1997;86(1):101-108.

96. Barker SJ, Shah NK, Hyatt J. Pulse oximetry and motion artefact:a study of accuracy and reliability [abstract]. Anesthesiology. 1995;83:A450.

97. Petterson MT, Begnoche VL, Graybeal JM. The effect of motion on pulse oximetry and its clinical significance. Anesth Analg. Dec 2007;105(6 Suppl):S78-84.

98. Trivedi NS, Ghouri AF, Shah NK, Lai E, Barker SJ. Effects of motion, ambient light, and hypoperfusion on pulse oximeter function. J Clin Anesth. May 1997;9(3):179-183.

99. Giuliano KK, Higgins TL. New-generation pulse oximetry in the care of critically ill patients. Am J Crit Care. Jan 2005;14(1):26-37; quiz 38-29

100. Sahni R, Gupta A, Ohira-Kist K, Rosen TS. Motion resistant pulse oximetry in neonates. Arch Dis Child Fetal Neonatal Ed. Nov 2003;88(6):F505-508.

101. Giuliano KK, Liu LM. Knowledge of Pulse Oximetry Among Critical Care Nurses. Dimensions of Critical Care Nurses. 2006;25(1):44-49.

102. Gnanalingham M G, Mehta B M, Siverajan M. et al Pulse oximetry as a screening test in neonates. Arch Dis Child 2001. 84(Suppl I)A35.

103. Feiner JR, Severinghaus JW, Bickler PE. Dark skin decreases the accuracy of pulse oximeters at low oxygen saturation: the effects of oximeter probe type and gender. Anesth Analg. Dec 2007;105(6 Suppl):S18-23, tables of contents.

104. Meberg A, Brun C. Pulse oximetry screening for congenital heart defects in newborn infants. (Abstract) of XIX Nordic Congress of Perinatal Medicine. 19–21 May 2005, Lund, Sweden.

105. Mahle WT, Newburger JW, Matherne GP, Smith FC, Hoke TR, Koppel R, et al. Role of pulse oximetry in examining newborns for congenital heart disease. A scientific statement from the American Heart Association and American Academy of Pediatrics. Circulation.2009;120: 447-8.

106. Myung K.Park Pediatric cardiology for practitioners 5 th edition pages no.75-76

107. John P.Cloherty, Eric C.Eichenwald,Ann R.Stark manual of neonatal care 6th edition pages 387-393

108. de Wahl Granelli A, Wennergren M, et al. Impact of pulse oximetry screening on detection of duct dependent congenital heart disease:a Swedish prospective screening study in 39 821 newborns. BMJ 2009; 338: A3037.

109. Kliegman, Behrman, Jenson, Stanton. Nelson text book of paediatrics, volume-2 pages 1851-1856

PROFORMA

S.NO:

BABY OF:

ADDRESS:

IP.NO:

MOTHER DETAILS:

AGE(Y)	GRAVIDA	PARA	CONSANGUINITY	FAMILY H/O CHD	AN SCAN	INFECTION	DM	DRUGS
				YES	YES	YES	YES	YES
				NO	NO	NO	NO	NO

BABY DETAILS:

MODE OF DELIVERY N/LSCS/FORCEPS/BREECH	GESTATIONAL AGE (TERM/PRETERM)	DATE OF DELIVERY	TIME OF DELIVERY	SEX (M/F)	WT(KG)

SCREENING DETAILS:

DATE OF SCREENING	TIME OF SCREENING	BABY AGE AT SCREENING
1		
2		

CLINICAL EXAMINATION SCREENING:

CONGENITAL ANOMALY	CYANOSIS	TACHYPNEA	TACHYCARDIA	MURMUR
YES/NO	YES/NO	YES/NO	YES/NO	YES/NO

SCREENING POSITVE	SCREENING NEGATIVE

PULSE OXIMETRY SCREENING:

1. OXYGEN SATURATION (%)			2. OXYGEN SATURATION(%) AFTER 6 HOURS IF NECESSARY		
≥95	90-94	<90	≥95	90-94	<90

SCREENING POSITIVE	SCREENING NEGATIVE

ECHOCARDIOGRAPHY RESULTS:

NORMAL	ABNORMAL

ECHOCARDIOGRAPHY DIAGNOSIS:

MASTER CHART

S.NO	NAME OF THE BABY	MOTHER DETAILS						BABY DETAILS				CLINICAL EXAMINATION						PULSE OXIMETER READING			ECHO RESULT
		CONSANGUIINITY	FAMILY H/O CHD	AN SCAN	DM	INFECTION	DRUGS EXPOSURE	MODE OF DELIVERY	GESTATIONAL AGE	SEX	WT (KG)	TACHYCARDIA	TACHYPNEA	CYANOSIS	MURMUR	CONGENITAL ANOMALY	P / N	TIME (HOURS)	RT FOOT (O2 SATURATION %)	P / N	
1	B/O Vijaya	NCM	A	N	A	A	A	N	T	F	2.8	A	A	A	-	A	N	30	88	P	TRI ATRESIA
2	B/O Jones	NCM	A	N	A	A	A	L	T	M	2.8	A	A	A	-	A	N	38	85	P	d-TGA
3	B/O Chellakani	CM	A	N	A	A	A	L	T	F	2.2	A	A	A	-	A	N	32	83	P	PULATRESIA
4	B/O Jothi	CM	A	N	A	A	A	N	T	F	2.0	A	A	A	+	A	P	37	91	P	TOF
5	B/O Isakiselvi	CM	A	N	A	A	A	L	T	F	2.7	A	A	A	+	A	P	38	97	N	VSD
6	B/O Leelavathi	CM	A	N	A	A	A	N	T	M	2.4	A	A	A	+	A	P	34	98	N	VSD
7	B/O Panchu	NCM	A	N	A	A	A	L	T	F	2.5	A	A	A	+	A	P	32	97	N	VSD
8	B/O Jenagai mari	NCM	A	N	A	A	A	L	T	F	3.0	A	A	A	+	A	P	29	95	N	VSD
9	B/O Sudha	NCM	A	N	A	A	A	L	T	M	2.6	A	A	A	+	A	P	38	96	N	VSD
10	B/O Jeyalaksmi	NCM	A	N	A	A	A	N	T	M	3.4	A	A	A	+	A	P	40	98	N	VSD
11	B/O Jansirani	CM	A	N	A	A	A	N	T	M	3.2	A	A	A	+	A	P	38	99	N	VSD
12	B/O Karthiga	NCM	A	N	A	A	A	N	T	F	2.4	A	A	A	+	A	P	35	96	N	VSD
13	B/O Sivasakthi	NCM	A	N	A	A	A	L	T	M	2.8	A	A	A	+	A	P	38	98	N	VSD
14	B/O Mareeswari	CM	A	N	A	A	A	L	T	M	2.3	A	A	A	+	A	P	30	97	N	VSD
15	B/O Chinaponnu	NCM	A	N	A	A	A	L	T	F	3.0	A	A	A	+	A	P	37	97	N	L-ASD
16	B/O Thenmozhi	NCM	A	N	A	A	A	N	T	F	3.8	A	A	A	+	A	P	36	96	N	L-ASD
17	B/OSudha	NCM	A	N	A	A	A	N	T	F	2.7	A	A	A	+	A	P	31	93	P	ASD(OP)
18	B/O Ramya	CM	A	N	A	A	A	N	T	F	2.4	A	A	A	+	A	P	38	98	N	DEXTROCAR
19	B/O Pothumponnu	NCM	A	N	A	A	A	L	T	M	2.3	A	A	A	+	A	P	37	96	N	S-ASD

20	B/O Muthulakshmi	NCM	A	N	A	A	A	N	T	M	2.8	A	A	A	+	A	P	35	96	N	S-ASD
21	B/O Shahirabanu	CM	A	N	A	A	A	L	T	F	2.6	A	A	A	+	A	P	30	95	N	S-ASD
22	B/O Periyatchi	NCM	A	N	A	A	A	L	T	M	3.1	A	A	A	-	A	N	32	94	P	S-ASD
23	B/O Lalitha	NCM	A	N	A	A	A	N	T	F	3.7	A	A	A	-	A	N	30	94	P	S-ASD
24	B/O Kowsalya	NCM	A	N	A	A	A	N	T	F	3.3	A	A	A	-	A	N	32	94	P	S-ASD
25	B/O Natchiammal	CM	A	N	A	A	A	N	T	F	2.9	A	A	A	-	A	N	29	94	P	S-ASD
26	B/O Amsavalli	NCM	A	N	A	A	A	N	T	M	2.7	A	A	A	+	A	P	38	97	N	S-PDA
27	B/O Hemalatha	NCM	A	N	A	A	A	N	T	F	2.5	A	A	A	+	A	P	35	97	N	S-PDA
28	B/O Kathijabanu	NCM	A	N	A	A	A	L	T	M	2.5	A	A	A	+	A	P	39	96	N	S-PDA
29	B/O Chithra	CM	A	N	A	A	A	N	T	F	3.0	A	A	A	+	A	P	35	97	N	S-PDA
30	B/O Jeevitha	CM	A	N	A	A	A	L	T	M	2.8	A	A	A	-	A	N	31	94	P	S-PDA
31	B/O Nishanthini	NCM	A	N	A	A	A	N	T	F	2.2	A	A	A	-	A	N	30	94	P	S-PDA
32	B/O Selvamani	NCM	A	N	A	A	A	L	T	M	2.5	A	A	A	-	A	N	26	94	P	S-PDA
33	B/O Santhi	CM	A	N	A	A	A	L	T	M	2.9	A	A	A	-	A	N	29	94	P	S-PDA
34	B/O Lalitha	NCM	A	N	A	A	A	N	T	F	3.4	A	A	A	-	A	N	26	94	P	S-PDA
35	B/O Pandiammal	NCM	A	N	A	A	A	N	T	M	2.7	A	A	A	+	A	P	28	94	P	S-PDA
36	B/O Murugayee	NCM	A	N	A	A	A	N	T	F	2.4	A	A	A	+	A	P	25	94	P	S-PDA
37	B/O Devika	NCM	A	N	A	A	A	L	T	F	2.8	A	A	A	+	A	P	29	94	P	S-PDA
38	B/O Muneeswari	NCM	A	N	A	A	A	L	T	M	2.9	A	A	A	+	A	P	30	98	N	NS
39	B/O Palanichithra	CM	A	N	A	A	A	N	T	M	2.8	A	A	A	+	A	P	32	95	N	NS
40	B/O Shanthi	NCM	A	N	A	A	A	N	T	M	2.2	A	A	A	+	A	P	31	97	N	NS
41	B/O Sudha	NCM	A	N	A	A	A	L	T	F	3.3	A	A	A	+	A	P	30	96	N	NS
42	B/O Jenifer	NCM	A	N	A	A	A	L	T	M	2.4	A	A	A	+	A	P	31	98	N	NS
43	B/O Vidhya	NCM	A	N	A	A	A	N	T	M	2.7	A	A	A	+	A	P	36	97	N	NS
44	B/O Devi	CM	A	N	A	A	A	N	T	M	3.4	A	A	A	+	A	P	32	96	N	NS
45	B/O Chithra	NCM	A	N	A	A	A	N	T	M	2.9	A	A	A	+	A	P	29	97	N	NS
46	B/O Jeyanthi	CM	A	N	A	A	A	L	T	F	2.7	A	A	A	+	A	P	31	97	N	NS
47	B/O Saranya	NCM	A	N	A	A	A	N	T	F	2.6	A	A	A	+	A	P	33	98	N	NS
48	B/O Selvi	NCM	A	N	A	A	A	N	T	F	2.7	A	A	A	+	A	P	35	98	N	NS
49	B/O Chinaponnu	CM	A	N	A	A	A	L	T	M	2.5	A	A	A	-	A	N	29	94	P	NS
50	B/O Muthumari	NCM	A	N	A	A	A	L	T	F	2.8	A	A	A	-	A	N	30	94	P	NS

51	B/O Kavitha	NCM	A	N	A	A	A	N	T	F	2.6	A	A	A	-	A	N	28	94	P	NS
52	B/O Alagu	NCM	A	N	A	A	A	N	T	M	2.4	A	A	A	-	A	N	31	94	P	NS
53	B/O Muthukili	CM	A	N	A	A	A	N	T	F	2.6	A	A	A	-	A	N	30	94	P	NS
54	B/O Jeyakodi	NCM	A	N	A	A	A	L	T	F	2.9	A	A	A	-	A	N	29	94	P	NS
55	B/O Parimalam	NCM	A	N	A	A	A	N	T	M	3.1	A	A	A	-	A	N	32	94	P	NS

[CM-consanguineous marriage, NCM-non consanguineous marriage, A-absent, N- natural labor, L-LSCS, T-term, M-male, F-female, + present, - negative, NS-normal study, S-small, L-large, P- Positive Screening, N-Negative Screening]

ABBREVIATIONS

CHD	Congenital Heart Disease
CCHD	Critical Congenital Heart Disease
CHF	Congestive Heart Failure
COHb	Carboxy Haemoglobin
Hb	Haemoglobin
RHb	Reduced Haemoglobin or Deoxyhaemoglobin
IR	Infra-Red
LED	Light Emission Diode
MetHb	Methaemoglobin
O2Hb	Oxyhaemoglobin
R	Red
AS	Aortic stenosis
AVSD	Atrio ventricular Septal Defect
ASD	Atrial Septal Defect
CoA	Coarctation of Aorta
DORV	Double outlet right ventricle
HLHS	Hypoplastic Left Heart Syndrome
IAA	Interrupted Aortic Arch
PA	Pulmonary Atresia
PDA	Patent Ductus Arteriosus

PFO	Patent Foramen Ovale
PS	Pulmonary Stenosis
TA	Tricuspid Atresia
TAPVR	Total Anomalous Pulmonary Venous Return
TGA	Transposition of the Great Arteries
TOF	Tetralogy of Fallot
VSD	Ventricular Septal Defect