INTRODUCTION

Skin to skin Care (SSC) is the practice of giving infants skin-to-skin contact with their parents, enabling them to share warmth and natural closeness. It was developed in Bogotá, Colombia, and has been used successfully in South America, Europe, and currently in the United States and all over the world ***(Charpak, 1997).***

Breastfeeding benefits have been demonstrated in multiple studies to be dose-responsive or, in other words, related to the amount of breast milk received. For example, fully breastfed infants have been shown to have lower overall illness rates, whereas minimal breastfeeding has not been found to be protective. Breastfeeding duration also affects child morbidity. A recent comprehensive review of the world literature to determine health benefits of exclusive breastfeeding for six months compared with exclusive breastfeeding for 3 to 4 months noted a decrease in the risk of gastrointestinal infection even in developed settings. One of the proven benefits of breastfeeding is that it increases the skin to skin contact and care of infants through skin to skin care that has a vital role in their growth, development and health outcomes. These benefits have been shown in sick babies as preterm or very small babies but have not been addressed in infants with congenital heart disease ***(Kramer, 2001).***

Infants with congenital heart disease (CHD) encounter some difficulties during feeding due to the energy required during sucking. There are no universally criteria for determining which feeding method is the most appropriate for those infants. Our aim is to identify the method of feeding that consumes the least amount of oxygen allows babies to be more physiologically stable, have lower heart rates (HR) and higher oxygen saturations (SaO2).

Our research hypotheses assume that: 1) SaO2 is higher and HR is lower in infants with CHD who are fed by using breastfeeding than those fed by bottle feeding. 2) SaO2 is higher and HR is lower in infants with CHD who are fed by cup feeding than those fed by bottle feeding. 3) There are differences in SaO2 & HR in infants with CHD who are fed by cup or by breast feeding. 4) Mothers are less satisfied when they use a method of feeding other than breast feeding

Oxygenation has been shown to be improved on SSC, to the extent that SSC is used successfully to treat respiratory distress. The breathing becomes regular and stable, and is coordinated with heart rate. When removed from incubator and placed SSC, oxygen saturation may rise slightly, or the percentage of oxygen provided to maintain good saturation can be lower (***Kirsten, 2001)***.

Heart Rate is increased when placed SSC. Though we can regard this increase as being with the clinically normal range, what is seen is actually a return to the physiologically normal heart rate, the lower rate being due to "protest despair behavior". Infants removed from incubators and placed SSC show a rise in temperature and a dramatic drop in glucocorticoids, as predicted by the "protest-despair response". Mothers are able to control the infant's temperature within a very narrow range, far better than an incubator (***Kirsten, 2001)***.

To accomplish this, mother's core temperature can rise to two degrees Centigrade if baby is cold, and fall one degree if baby is hot. Skin-to-skin contact is better than incubator for rewarming hypothermic infants. Also Nutrition is improved with SSC, both with respect to the mother’s ability to breastfeed, and with respect to the newborn’s utilization of the feed. The volume of mother’s milk is greatly increased, and the frequency of feeds provided likewise. Even without the increased milk, with the vagal

stimulation the infant receives, the gut is better able to use the milk provided, and grows faster (***Kirsten, 2001)***.

There is a controversy as to whether infant with CHD with or without heart failure benefit from breastfeeding also the relationship between congenital heart and skin to skin care has not been examined thoroughly.

Aim of work:

This study is designed to:

1- Compare health, growth and developmental outcome of infants with CHD exposed to almost fully breastfeeding for six months and continued breastfeeding into the second year with infant with CHD exposed to any formula feeding from before six months and early cessation of breastfeeding before one year (after controlling for severity of defect and operative procedures).

2- Compare complications of CHD as chest infections, cyanotic spells, anemia and liver disease of infants with CHD exposed to almost fully breastfeeding for six months and continued breastfeeding into the second year with infant with CHD exposed to any formula feeding from before six months and early cessation of breastfeeding before one year (after controlling for severity of defect and operative procedures).

3- Evaluate the clinical intervention of SSC on the outcome of infant with decompensated congenital heart disease (CHD) in breastfed and artificially fed infants. This will be assessed as follows:

- Does SSC improve the blood gases status?

- Does SSC reduce complications or control already present complications?

- Does hospital stay improve mother satisfaction and lessen her anxiety score?

I- Congenital Heart Disease

Incidence of congenital heart disease (CHD):

A congenital heart defect is a problem which is present at birth. In the general population, CHD are the most common of all congenital birth defects. They are caused by improper development of one or more structures of the heart or the blood vessels of the heart during fetal development and may produce symptoms at birth, during childhood and sometimes not until adulthood ***(Moller and Hoffman, 2005).***

About 0.5-0.8% of all children are born with CHD (about 8-10 out of every 1000 children every year). ***(Fixler et al., 2009).***

More than forty thousand babies all over the world are borne each year with CHD; 4000 will survive their first year (the children heart foundation). ***(Ferenz et al., 2008).***

Table (1): relative frequency of major congenital heart lesions:

Type of lesion	% of all lesions:
Ventricular septal defect	30-35
Atrial septal defect	6-8
Patent ductus arteriosus	6-8
Coarctation of Aorta	5-7
Tetralogy of fallot	5-7
Pulmonary valve stenosis	5-7
Aortic valve stenosis	4-7
D-transposition of great arteries	3-5
Hypoplastic left ventricle	1-3
Hypoplastic right ventricle	1-3
Truncus arteriosus	1-2
Total anomalous pulmonary venous return	1-2
Tricuspid atresia	1-2
Single ventricle	1-2
Double outlet right ventricle	1-2
Others	5-10

Excluding patent ductus arteriousus in preterm neonates, bicuspid aortic valve, physiological pulmonary stenosis and mitral valve prolapse ***(Ferenez et al., 2008).***

The number of adults with problems connected to congenital heart defect is rising, passing the number of children with congenital heart defects in most Western countries. This group is referred to as grown up congenital heart diseases patients. ***(Rudolph et al., 2008).***

Etiology of congenital heart disease:

Environmental and adverse maternal conditions:

Two to 4% of cases of CHD are associated with known environmental or adverse maternal conditions and teratognic influences, including maternal diabetes mellitus, phenylketonuria, or systemic lupus erythematosus, congenital rubella syndrome and maternal ingestion of drugs (lithium, ethanol, warfarin, thalidomide, antimetabolites, anticonvulsant agents)***(Goldmuntz,2001).***

Women who have seizure disorders and need to take anti-seizure medications may have a higher risk of having a child with congenital heart disease, as do women who take lithium to treat depression. Mothers who have phenylketonuria and who do not adhere to the special diet necessary to manage the disease during pregnancy have a higher risk of having a child with congenital heart disease. Also, women with insulin-dependent diabetes mellitus (particularly if not controlled) or systemic lupus erythematosus may have a higher risk of having a child with heart defects.

Counseling is important for women with these chronic illnesses before becoming pregnant. A mother who contracts rubella during her pregnancy

has a very significant chance of having a baby with birth defects, including congenital heart disease ***(Packard, 2007).***

Family history and CHD:

Incidence of congenital heart disease in the normal population is approximately 0.08%. However, the risk increases when either parent has congenital heart disease (CHD), or when another sibling was born with CHD.

One always considers the following statistics:

- The risk of having baby with CHD is 1.5-5%, if there is previous baby with CHD, depending on the type of CHD in the previous one.
- If there are two children with CHD, then the risk increases to 5-10%, to have another child with CHD.
- If the mother has CHD, then the risk of having a baby with CHD ranges from 2.5-18 %(average 6.7%).
- If the father has CHD, the risk is 1.5-3%.
- Congenital heart defects involving obstructions to blood flow in the left side of the heart have a higher rate of reoccurrence than other heart defects.
- If another child is born with CHD, it can be a different type of defect that seen in the first child.
- Some heart defects are considered to have autosomal-dominant inheritance, meaning that a parent with the defect has a 50% chance, with each pregnancy, to have a child with the same heart defect, and males and females are equally affected. Similarly, there is also a 50% chance that an offspring will not be affected.

So the consultation with a genetic counselors or genetic specialist is encouraged for women with congenital heart disease before becoming pregnant. In families with CHD either in the parents or offspring, fetal echocardiography can be performed in the second trimester, at about 18-22 weeks of pregnancy, to determine the presence of major heart defects in the fetus ***(Packard, 2007).***

Chromosomal abnormalities and CHD:

Five to 8% of all babies with CHD have a chromosome abnormality. Chromosomes are the structures in the cells that contain genes; genes code for traits such as eye color and blood type.

Usually there are 46 chromosomes in each cell of the body having too many or too few chromosomes results in health problems and birth defects. Structural defects of the chromosomes, where a piece of the chromosome is missing or present in duplicate, also cause health problems.

There are a number of chromosomal abnormalities associated with congenital heart defects. Some of these include the following:

- Down syndrome.
- Trisomy 18 and Trisomy 13.
- Turner's syndrome.
- Cri du chat syndrome.
- Wolf-Hirahhorn syndrome.
- DiGeorge syndrome ***(deletion 22q11).***

Chromosomal analysis can be performed from a small blood sample to rule out the presence of a chromosomal abnormality in a child with a congenital heart defect ***(Parckard, 2007).***

Single gene defects:

Because a single gene or short segments of DNA cannot be observed microscopically, molecular genetics techniques are used to detect mutations. Information from the Human Genome Project and other advances in the filed of molecular genetics have dramatically improved geneticist's ability to prenatal diagnose genetic disorders ***(Eugene Hoyme, 2004).***

There are an estimated 70.000 genes contained on the 46 chromosomes in each cell of the body. Genes come in pairs, one which is inherited from the mother, the other from the father.

Genes not only compose individual traits, but also compose health problems when gene alteration (mutation) is present.

When a gene is mutated, a number of health problems may occur in a person, due to the single underlying genetic mutation. Several health problems with one genetic cause are referred to as a syndrome. Some of the genetic syndromes associated with a higher incidence of heart defects include:

- Marfan syndrome.
- Smith-Lemli-Opiz syndrome.
- Ellis-van Creveld.
- Holt-Oram syndrome.
- Noonan syndrome.
- Mucopolysaccharidoses.

Other genetic syndromes which are not due to a single gene defect but are associated with CHD include Goldenhar syndrome (hemifacialmicrosomia), William's syndrome, and VACTERL association (tracheal and esophageal malformations associated with vertebral, anorectal, cardiac, renal, radial, and limb abnormalities).

So if a child has been diagnosed with a chromosomal or other genetic abnormality, genetic counseling is helpful to determine the risk of heart defects occurring in future children ***(Packard, 2007).***

Medical research point out the importance of social differences between countries and population, when explaining differences in health. This ideology has created a social programming model**,** which means that the effect of the early social environment on health is mediated by the social environment and the school achievement during growth, and by employment opportunities, living conditions, and lifestyle factors ***(Vågerö, 1995)***.

Heart disease morbidity and mortality

Osmond, (1993) reported that CHD mortality decreased progressively with increasing birth weight. Since then there have been several, (mainly retrospective cohort studies) which have replicated these observation and also demonstrated the association between size at birth and non-fatal CHD.

The association between birth weight and disease outcome is, with few exceptions, consistent with data based upon the older generations born in the early 1920s from different countries ***(Vagero D, 1994)***.

Classification of Congenital Heart Diseases

Congenital heart diseases are classified into:

A- Congenital cyanotic heart diseases.

B- Congenital acyanotic heart diseases.

C- Others.

A- Congenital acyanotic heart diseases: (80%).

They are subdivided into:

1- Congenital acyanotic heart diseases with increased pulmonary blood flow:

- Ventricular septal defect.
- Atrial septal defect (ostium primum and ostium secundum).
- Partial anomalous pulmonary venous drainage.
- Patent ductus arteriosus.
- Aorticopulmonary defect.
- Endocardial cushion defect.

2- Congenital acyanotic heart diseases with normal pulmonary blood flow:

- Pulmonary stenosis.
- Aortic stenosis.
- Aortic coarctation.

B- Congenital cyanotic heart diseases: (20%)

They are subdivided into:

1- Congenital cyanotic heart diseases with decreased pulmonary blood flow:

- Fallot's tetralogy.
- Transposition of great arteries with pulmonary stenosis.
- Double outlet right ventricle with pulmonary stenosis
- Tricuspid Atresia.
- Pulmonary Atresia.
- Ebstein anomaly.

2- Congenital cyanotic heart diseases with increased pulmonary blood flow:

- Transposition of great arteries
- Total anomalous pulmonary venous drainage
- Hypoplastic left heart syndrome.
- Single ventricle.
- Truncus arteriosus.
- Eisenmenger syndrome.

C- Others

1- Abnormalities of the aortic arch:

- Right sided aortic arch.
- Vascular rings.

2- Abnormalities of the coronary arteries:

- Coronary arteiovenous fistula.
- Ruptured sinus of Valsalva aneurym.

- Anomalous origin of the left coronary artery from pulmonary artery.
- Anomalous origin of the right coronary artery from pulmonary artery.
- Ectopic origin with aberrant proximal course of coronary artery.

3- Abnormalities of the heart position:

- Ectopia cordis.
- Diverticulum of the left ventricle.
- Dextrocardia ***(Allen et al., 2008)***.

Diagnostic modalities of Congenital Heart Diseases:

1- History

Some symptoms could suggest presence of congenital cardiac abnormality including: antenatal history suggestive of maternal diabetes mellitus, systemic lupus erythematosus, viral infection during pregnancy, natal history of prematurity, obstructed labour, cyanosis, respiratory distress, onset of presentation at birth, after few months. Cardiac symptoms of infants e.g. feeding difficulties, sweating, cyanosis, recurrent chest infections, and older children e.g. exercise intolerance. Others as, chest pain. Family history of relatives with congenital heart, muscle weakness, early stroke or ischemic heart disease. ***(Amplarz et al., 2006).***

2- Clinical examination:

Cyanosis, signs of heart failure as tachycardia, tachypnea, and enlarged tender liver, pericardial pulses may show big pulse volume, radio-femoral delay, weak pulses, and blood pressure assessment by auscultation,

palpation and Dinamap, cardiac examination by combined inspection, palpation, and auscultation. Presence of Other congenital anthropometric measures may detect failure to thrive ***(Perloff et al., 2005).***

3- Investigations:

Chest X-ray: demonstrates the chamber enlargement of the heart, lung vascularity, chest infection***. (Allen et al., 2008).***

Electrocardiogram: demonstrates chamber enlargement, Arrhythmia, Drug effects (e.g. Digitalis toxicity) ***(Amplatz et al., 2006).***

Echocardiography and Doppler: demonstrates chamber enlargement, cardiac structure, cardiac contractility (FS %), valvular lesions, septal defects, intracardiac pressures, gradients and flow direction, thrombi, vegetations, tumor, assessment of coronaries and pericardial effusion**.** ***(Rudolph et al., 2008).***

Cardiac MRI, MRA, CT& multi-slice CT are used for evaluation of coronary anatomy, evaluation of details of complex lesions ***(Allen et al., 2008)***

Catheterization and Angiography are used for the diagnosis and/or interventional purposes, measurement of pressures, O2 saturation, demonstration of abnormal anatomy, evaluation of pulmonary vascular resistance, diagnostic angiography ***(Rudolph et al., 2008).***

Management strategy of Congenital Heart Diseases:-

Management of CHD may include medical management by drugs, interventional management using cardiac catheterization and surgical management.

Medical management of congenital acyanotic heart diseases:-

Diet: patients are advised to have a well balanced diet (heart healthy), **Vaccination**: routine schedule and Influenza vaccine, prophylaxis and treatment of Infective endocarditis most congenital heart diseases except ASD, treatment of heart failure and arrhythmia if present, Iron therapy: increases tolerance and RBCs deformability and decreases stroke.

Medical management of congenital cyanotic heart diseases:-

Treatment of cyanotic spells, Protaglandin E1 infusion: if duct dependent lesion, Propranolol: in patients with infundibular pulmonary stenosis (Fallot's tetralogy), Exchange transfusion: in cases of increased blood viscosity.

Surgical and interventional management of congenital heart disease:

Interventional management:-

1-Ballon dilatation of the valvular pulmonary and aortic stenosis and coarctation of aorta.

2- Rashkind (balloon atrial septostomy):

Echo guided (bedside), indicated in transposition of great arteries, pulmonary atresia with intact septum, and improves mixing at the atrial level, access: umbilical vein or femoral vein (>3 days). Catheter is passed into right atrium then left atrium (across foramen ovale), inflation of the balloon at the end with rapid withdrawal producing a tear.

2- Stenting of aortic Coarctation and patent ductus arteiosus in duct dependent lesions.

Surgical management:

Surgical management of acyanotic heart diseases:

Palliative surgery:

a- Pulmonary artery banding: palliative procedure to protect lung vascularity

Corrective surgery:

a- Patent ductus arteriosus ligation: Closed heart surgery

b- Closure of ventricular septal defects: Patch closure or direct suture

c- Closure of atrial septal defects: Patch closure or direct suture

d- Repair of coarctation: Resection with end to end anastomosis Subclavian flap aortoplasty

Surgical management of cyanotic heart diseases:

Palliative surgery:

a- Shunt operations (Blalok Tausing shunt): Anastomosis between Subclavian artery and ipsilateral pulmonary artery

b- Glenn shunt: Anastomosis between superior vena cava and right pulmonary artery.

Corrective surgery:

a- Arterial switch: Performed for transposition of the great arteries before the age of two weeks.

Cutting aorta and pulmonary arteries and changing them around.

b- Fontan operation: Anastomosis between superior and inferior vena cavae to the right pulmonary artery.

c- Norwood operation: Anastomosis between pulmonary artery and ascending aorta, right Blalok Tausing shunt and atrial septostomy.

d- Rastelli operation: Performed for transposition of the great arteries with ventricular septal defect and pulmonary stenosis, cutting pulmonary artery and connect it to the right ventricle, intracardiac tunnel between left ventricle and aorta.

e- Atrial switch operation: Performed for transposition of the great arteries within the first year of age, creating a tunnel directing SVC and IVC to left side and another tunnel directing the pulmonary veins to the right side***. (Rudolph et al., 2008).***

Prognosis of Congenital Heart Diseases

Most congenital heart defects are well tolerated by the fetus because of the nature of fetal circulation, even the most severe cardiac defects (e.g. Hypoplastic left heart syndrome) can be usually well compensated by the fetal circulation. In this example, the entire fetal cardiac output well be ejected by the right ventricle via ductus arteriosus into both the descending and the ascending aortae (the latter filling in retrograde fashion). It is only after birth when the fetal pathways (ductus arteriosus and foramen ovale) are closed that the full homodynamic impact of an anatomic abnormality becomes apparent***. (Atkin et al., 2007).***

One notable exception is the case of severe regurgitant lesions, most commonly of the tricuspid valve. In these lesions (e.g. Ebstein anomaly), the parallel fetal circulation can not compensate for the volume overload imposed on the right side of the heart. In utero fetal heart failure, often with pleural and pericardial effusions and generalized ascites (non immune hydrops fetalis) may occur ***(Copel and Kleinman, 2005).***

Although the most significant transitions in circulation occur in the immediate perinatal period, the circulation continues to undergo changes after birth, and the later changes may also have a homodynamic impact on cardiac lesions and their apparent incidence. As pulmonary vascular resistance falls in the first several weeks of life, left to right shunting through intracardiac defects increases and symptoms become more apparent. Thus, in patients with ventricular septal defect, heart failure is often manifested between the first and the third months of age ***(Amplatz et al., 2006).***

The severity of various defects can also change dramatically with growth; some ventricular septal defects may become smaller and even close as the child ages. ***(Moller and Hoffman, 2005).***

Alternatively, stenosis of the aortic or pulmonary valves which may be mild in the newborn period may become worse if the valve orifice growth does not keep place with patient growth. ***(Burn et al., 2004)***

The physicians should be always alert for associated congenital malformations, which can adversely affect the patient's prognosis. Identifying the etiology and pathogenesis has been difficult because of the large number of anatomically district congenital cardiovascular malformations with varying clinical consequences. Here again, the pathologic classification is of value in the analysis of the epidemiologic data set. ***(Jordy et al., 2007).***

More than forty thousand are born each year with a congenital heart disease; 4000 will survive their first year (the children's heart foundation) ***(Ferenez et al., 2008).***

At least 50% of congenital heart diseases are minor and can be surgically corrected but they account for over half of the deaths from congenital abnormalities in childhood. ***(Moller and Hoffman, 2005).***

Twice as many children die each year from congenital heart diseases than all forms of pediatric cancers (the children's heart foundation). ***(Mirchall et al., 2006).***

It represents one of the most common cause of death of children at different ages starting from abortion of the fetus, stillbirth, death of the patient at birth, during infancy, during early childhood, during late childhood and may at adulthood***.(Burn et al., 2004).***

Preventive Measures of Congenital Heart Diseases:

Of course, every pregnant woman needs to know all what she can do to ensure the health of her baby.

Prospective parents can take positive lifestyle steps to increases the chance that their babies will be born with a healthy heart. ***(Hall et al., 2006)***

1- Genetic counseling for the prospective parents:

Fluorescent insitu hybrization allows clinicians rapidly screening of suspected cases once a specific chromosomal or genetic abnormality has been identified and determination of the recurrence risk of this anomaly. Genetic counseling is based on through communication concerning the issues of the child birth defects and helps the family and the child, it should:

- Comprehend the medical facts, including diagnosis, probable clinical course and therapy options.
- appreciate the way heredity contributes to the etiology

- Understand the recurrence risk within the family and alternatives for dealing with that risk.
- Choose a course that is appropriate for the family based on risk, goals, ethical and religious standards.
- Make the optimum adjustment within the family. ***(Burn et al., 2007).***

2-Avoidance of maternal febrile illness during pregnancy:

Any pregnant woman should avoid contact with people who have flu or other fever related illness as any fever related illness during the first trimester of pregnancy may carry a two fold higher risk of development of congenital heart diseases. ***(Allenetal., 2008)***

3-Vaccination:

A blood test should be done early in the pregnancy to see if the pregnant woman is immune to rubella or not. If the mother is not immune, she must avoid any possible exposure to rubella and influenza and should be immunized immediately following delivery. ***(Copel and Kleinman, 2005).***

4- Avoidance of intake of tonics (vitamins, iron, folic acid and calcium) during the first trimester:

Physicians should be made aware that a woman is pregnant before prescribing any medications for her and should also be aware of the drugs increasing the risk of congenital heart diseases and the measures taken by the pregnant to avoid the development of congenital heart diseases ***(Hall et al., 2006).***

5- Intake of tonics (vitamins, iron, folic acid and calcium) staring from the second trimester:

It is recommended for woman to take a daily multivitamin containing 400 micrograms of folic acid which is essential for normal growth and development of the fetus. It has a protective effect against the development of the heart defects. Folic acid is recommended to be taken before and during pregnancy but it should be avoided in the first trimester to avoid teratogenicity ***(Scanlon et al., 2007).***

6- Management of consanguineous mating:

Parental consanguinity especially the first cousins should be avoided as far as possible especially if there is positive family history of congenital heart diseases. Genetic counseling is strongly recommended for consanguineous couples***. (Becker et al., 2008).***

7- Management of maternal intake of caffeine, smoking and alcohol intake:

Maternal smoking and addiction of alcohol and other drugs should be avoided; also maternal intake of caffeine should be lowered especially during pregnancy. ***(Nora et al., 2001).***

8- Management of maternal intake of contraceptive pills:

Maternal intake of female sex hormones are associated with increased congenital heart diseases, so it should be lowered as far as possible and it should be stopped at least one month before pregnancy***.(Campbell et al., 2008).***

9- Management of maternal intake of Tocolytics during pregnancy:

Maternal intake of Tocolytics during pregnancy should be lowered as possible. Some physicians prescribe Tocolytics as a routine drug for any

Pregnant women; this should be avoided to avoid teratogenicity ***(Moller and Hoffman, 2005).***

10- Adequate maternal diet and avoidance of maternal depression during pregnancy:

Pregnant women should have careful antenatal care and close follow up and should be made aware of useful nutritive diet and to ensure safety of herself and her baby. Depression should be avoided during pregnancy. ***(Nora et al., 2001).***

11- Avoidance of exposure to radiation:

Maternal exposure to radiation should be avoided during pregnancy e.g. exposure to X-rays and mobile networks. ***(Copel and Kleinman, 2005).***

12- Maternal and paternal age:

Maternal and paternal ages above 30 years is associated with increased risk of congenital heart diseases, so it is recommended to get married before 30 years as possible***.(Allen et al., 2008).***

13- Maternal education and family problems:

Maternal education below a diploma degree (high school) and presence of family problems during pregnancy are associated with increased risk of congenital heart disease, so they should be avoided as possible**.** ***(Ferencz et al., 2008).***

14- Close follow up of maternal diabetes mellitus:

Close observation and follow up of blood sugar level should be done frequently during pregnancy especially in diabetic mothers as poorly controlled sugar level especially in type I diabetes mellitus is associated with a high rate of congenital heart diseases. ***(Hall et al. 2006).***

15- Control of diet in phenylketonuria:

The risk can be reduced with strict dietary control (low phenylalanine diet) both before conception and during pregnancy ***(Campbell et al., 2008).***

Expectant mothers should receive good antenatal care as many congenital defects can be discovered on routine ultrasound examination and fetal echocardiography which can diagnose congenital heart diseases of the fetus, some of these defects can be managed while the baby is still intrauterine. ***(Allen et al., 2008).***

Fetal echocardiography is recommended for diabetic mothers to facilitate the early diagnosis of congenital cardiovascular malformations. ***(Amplatz et al., 2006).***

The delivery can be anticipated and the appropriate medical personnel (such as pediatric cardiologist, cardiothoracic surgeon and neonatologist) can be present and ready to help as necessary, such preparation can mean the difference between life and death for some babies. ***(Hall et al., 2006).***

Heart failure in infants

Definition of heart failure

Heart failure is defined as a state in which the heart cannot deliver an adequate cardiac output to meet the metabolic needs of the body ***(Bernstein, 2004)***.

Etiology of heart failure

The etiology of heart failure in children is age- dependent as shown in table (2) ***(Bernstien, 2004)***.

Among children who develop heart failure, 80%do so in the first year of life, most commonly from congenital cardiac anomalies: of 20% who develop cardiac failure after 1 year of age, in half it is related to congenital anomalies; and in the other half, it is related to acquired conditions ***(Ross,2001).***

Table (2): Etiology of heart failure ***(Bernstien, 2004).***

Fetal	Severe anemia (hemolysis, fetal-maternal transfusion, parvovirus B19-induced anemia, Hypoplastic anemia) Supraventricular tachycardia ventricular tachycardia complete heart block
Premature Neonate	Fluid overload Patent ductus arteriosus Ventricular septal defect Cor pulmonale (bronchopulmonary dysplasia) Hypertension

Full-term neonate	Asphyxial Cardiomyopathy Arteiovenous malformation (vein of Galen, hepatic) Left-sided obstructive lesions (coarctation of aorta, Hypoplastic left side of the heart) Large mixing cardiac defects (single ventricle, Truncus arteriosus) Viral myocarditis
Infant-Toddler	Left-to-right cardiac shunts (ventricular septal defects) Hemangioma (arteiovenous malformation) Anomalous left coronary artery Metabolic Cardiomyopathy Acute hypertension (hemolytic- uraemic syndrome) Supraventricular tachycardia Kawasaki's disease
Child-Adolescent	Rheumatic fever Acute hypertension (glomerulonephritis) Viral myocarditis Thyrotoxicosis Hemochromatosis-hemosiderosis Cancer therapy (radiation, doxorubicin) Sickle cell anemia Endocarditis Cor pulmonale (cystic fibrosis) Cardiomyopathy (hypertrophic, dilated)

Pathophsiology of heart failure

Heart failure is now thought as a disorder of the circulation, not merely a disease of the heart (***Burch, 2002***).

Since the response of the circulation to stress is governed by neurohormonal mechanisms, in addition to haemodynamic factors,

Several lines of evidence suggest that neurohormonal mechanisms play a central role in the progression of heart failure (***Kay et al., 2001).***

Activation of sympathetic nervous system and rennin-angiotensin-aldosterone system in congestive heart failure exerts a direct deleterious effect on the heart that is independent of the haemodynamic actions of these endogenous mechanisms ***(Collins-Nakai, 2002).***

Pathophsiologic events in different stages of congestive heart failure:

1. Stage of asymptomatic and mildly symptomatic left ventricular systolic dysfunction:

A. Positive inotropic mechanisms:

The key event leading to heart failure is loss of a critical quantity of functioning myocardial cells after an injury to the heart (***Peterson, 2000)***.

To compensate for myocardial cell loss, both haemodynamic and neurohormonal mechanisms are activated to enhance the contractile force of the non-injured myocardium, and so preserve cardiac function (***Elliot, 2000).***

A decrease in the ability to empty the ventricle during systole increase the tension on the non-injured parts of the heart during diastole; the ventricle responds to this increase in diastolic tension (preload) by enhancing its contraction (Frank-Starling principle)(***Burch, 2002***).

A decrease in the ability of the ventricle to eject blood into the aorta activates the sympathetic nervous system; the resulting stimulation of B-adrenergic receptors in the non-injured myocardium increases both the force and frequency of contraction (***Bruns and Canter, 2002)***.

These two compensatory mechanisms involve different, but complementary, intracellular calcium dependent inotropic pathways.

Whereas sympathetic activation increases the delivery of calcium to myofilamentes, ventricular dilatation enhances sensitivity of myofilamentes to calcium (***Schwartz et al., 2001).***

B. Stress reducing mechanisms:

Although haemodynamic and neurohormonal mechanisms provide inotropic support for the injured heart, they can introduce an important risk. Both ventricular dilatation and sympathetic activation (by constriction of peripheral arteries and veins) strikingly increase the internal stress on the heart wall during diastole, which can dramatically distort its architecture and accelerate its energy expenditure (***Buchhorn et al., 2001*)**.

To prevent such adverse structural and functional effects, the circulation closely regulates the magnitude of ventricular dilatation and sympathetic activation (***Nogueria et al., 2000***).

An increase in diastolic wall stress in ventricle leads to induction of specific wall stress in ventricle leads to induction of specific proto-oncogens (c-fos and c-myc) that trigger synthesis of myofibriller proteins. The subsequent increase in wall thickness reduces ventricular strain and dilatation by distributing the excess stress among an increased number of sarcomeres (***Jackson et al., 2000).***

The myofibriller proteins synthesized during haemodynamic stress have the biochemical characteristics of fetal myocardium and are bioenergetically more efficient than their adult isoforms (***Soongswang et al., 2002)***.

Both quantitatively and qualitatively, cardiac hypertrophy reduces the energy expenditure of the overloaded heart ***(Ross, 2001)***.

An increase in diastolic wall stress in the atria suppresses the actions of the sympathetic nervous system. Atrial stretch stimulates atrial baroreceptors that inhibit sympathetic outflow from the vasomotor centre in the central nervous system ***(Brown, 2000).***

Atrial stretch leads to the secretion of atrial natriuretic peptide, which inhibits the release of noradrenalin and the actions of this neurotransmitter on peripheral blood vessels. The peptide also exerts direct vasodilator and natriuretic effects that reduce the haemodynamic load on the heart (***Maunouray et al., 2000)***.

Stress-reducing mechanisms (whether triggered by atrial or ventricular stimuli) have a central role in limiting the adverse consequences of ventricular dilatation and sympathetic activation consequences of ventricular dilatation and sympathetic activation (***Chen and Brurnett, 1999***).

C. Loss of stress reducing mechanisms:

Prolonged ventricular distention leads to necrosis and fibrosis of the ventricular wall, which compromises the hypertrophic response and restricts the heart capacity to normalize wall stress (***Khalil et al., 2000)***.

Prolonged atrial distension leads to structural and functional changes in atrial response endings, which reduce the ability of these baroreceptors to inhibit sympathetic outflow from the vasomotor centre. Abnormalities of baroreceptors function has been described in both experimental and clinical heart failure (***Jackson et al., 2000***).

Prolonged atrial distension leads to depletion of natriuretic peptides, such that their release after an increase in atrial pressure is blunted ***(Mair et al., 1999***).

Natriuretic peptides are synthesized in the ventricles as well as the atria, but the magnitude of this response is inadequate (***Richchards et al., 1999).***

These events represent a critical loss of the ability of the circulation to limit ventricular wall stress and the release of vasoconstrictor hormones. Ventricular dilatation progresses, the sympathetic nervous system become persistently activated, and heart failure begins (***Burch, 2002***).

D. Loss of positive inotropic mechanisms:

When wall stress increases after the loss of load-reducing mechanisms, the heart becomes more dependent on endogenous inotopic processes to maintain cardiac function. The long-term activation of these inotropic mechanisms leads to loss of their effects on myocardial contractility (***Kay et al., 2001).***

The failing heart loses the ability to enhance its inotropic state in response to increases in ventricular volume. When sarcomeres are stretched to their limits because of progressive ventricular dilatation, increases in preload do not enhance systolic ejection, so the Frank-Starling curve becomes both depressed and flattened (***Morrow et al., 2000).***

The failing heart loss its ability to responds to the positive inotropic effects of endogenous and exogenous catecholamines. This attenuation results from changes in the cardiac β-adrenergic pathway, which include down regulation of β-receptors (mainly β-1) and uncoupling of β-receptors from their effectors enzyme, adenylate cyclase ***(Bruns and Canter, 2002).***

Such uncoupling seems to be related to an alteration in the guanine nucleotide binding proteins that stimulate (Gs) or inhibit (Gi) the

interaction of the receptor and the enzyme. Both a decrease in (Gs) and an increase in (Gi) have been reported in patients with heart failure ***(Schwartz et al., 2001).***

Prolonged ventricular dilatation and sympathetic activation not only lead to loss of their beneficial effects on cardiac contractility, but accentuate their adverse actions on ventricular wall stress ***(Robbins, 2000).***

Concomitant with loss of the Frank-Starling mechanisms, the failing ventricle loses its ability to augment its function so as to overcome increases in resistance (after load) (***Jackson et al., 2000).***

Not only is heart failure accompanied by loss of responsiveness to β-adrenergic stimuli in the heart, but also it is characterized by an enhanced responsiveness to α-adrenergic stimuli in peripheral vessels (***Buchhorn et al., 2001).***

The resulting constriction of systemic arteries and veins strikingly increases the pressure and volume in the heart and exacerbates the load on the left ventricle ***(Nugent et al., 2001).***

The same endogenous mechanisms that exert favorable effects in the normal heart (by increasing its inotropic state) produce detrimental effects in the failing heart (by increasing its wall stress) (***Nogueria et al., 2000).***

Consequently, the heart has less capacity to overcome stress rather than to eject blood. Systolic function cannot be sustained, and cardiac output falls ***(Ross, 2001).***

2. Stage of symptomatic moderate to severe left ventricular systolic dysfunction:

When cardiac output falls, systemic perfusion pressure is maintained by two mechanisms: peripheral vasoconstriction and sodium retention. Both are characteristic findings in patients with established heart failure ***(Soongswang et al., 2002).***

The development of peripheral vasoconstriction and sodium retention represents an important shift in circulatory priorities, with the cardiovascular system moving from a state of compensation to a state of decompensation ***(Burch, 2002).***

Whereas before the onset of heart failure, endogenous mechanisms are directed to support of cardiac function, after the onset of heart failure, the circulation's main object is to support systemic perfusion pressure ***(Venugopalan et al., 2000).***

Peripheral vasoconstriction and sodium retention cannot be explained only by the excessive activation of endogenous vasoconstrictor system; their development requires loss of counter-regulatory vasodilator influences, both in blood vessels and in the kidneys ***(Morrow, 2000).***

A. Peripheral vasoconstriction:

Many neurohormonal systems that are activated in patients with heart failure exert potent constrictor effects on peripheral blood vessels ***(Richards et al., 1999).***

The sympathetic nervous system is activated early in the disease process, whereas the rennin-angiotensin-aldosterone system is usually triggered once symptoms develop, and vasopressin is released mainly in the terminal phase of the disease ***(Peterson, 2000).***

In addition to circulatory vasoconstrictor factors, heart failure is accompanied by an increased release of locally active vasoconstrictors produced by vascular endothelium as endothelin. Endothelin

concentrations are increased in heart failure in proportion to the severity of the disease ***(Frey et al., 2000).***

In patients without heart failure, the actions of endogenous vasoconstrictor factors are counterbalanced by endogenous vasodilators ***(Sutsch et al., 2000).***

Atrial natriuretic peptide normally inhibits release of noradrenalin, rennin, and vasopressin as well as their actions on peripheral blood vessels ***(Jensen et al., 1999).***

The release of endothelium-derived relaxing factor normally offsets the actions of endothelin (***Seneri et al., 2000).***

In heart failure the actions of circulating and locally active vasodilators are attenuated ***(Love et al., 2000).***

Not only is the release of atrial natriuretic peptide blunted after long term atrial distension, but also the peptide (once released) loses its ability to suppress the release of renin or dilate peripheral blood vessels

(Masson et al., 2000).

The release of endothelium-derived relaxing factor is strikingly diminished in patients with heart failure. ***(Frey et al., 2000).***

As the result of the loss of vasodilator factors, the actions of vasoconstrictors are left unopposed ***(Sutsch et al., 2000).***

The resulting vasoconstriction is enhanced a process of mutual neurohormonal amplification. Activation of the sympathetic nervous system increases the release of renin, and angiotensin enhances the release of both noradrenalin and vasopressin ***(Richards et al., 1999).***

B. Sodium retention:

In addition to peripheral vasoconstriction, activation of neurohormonal systems leads to salt and water retention in patients with heart failure ***(Elliott, 2000).***

This alteration in fluid balance results from the direct and indirect effects of renin-angiotensin- aldosterone system on glomerular and tubular function ***(Fruhwald et al., 1999).***

Angiotensin augments sodium reabsorption directly and indirectly by stimulating the release of aldosterone. ***(Chen and Burnett, 1999).***

Angiotensin causes water retention by increasing water intake (by stimulating the thirst center) and decreasing water excretion (by releasing pituitary vasopressin) ***(Mair et al., 1999).***

Salt and water retaining effects are potentiated by the stimulation of renal sympathetic nerves and by a fall in renal blood flow ***(Ross, 2001).***

The actions of endogenous salt retaining systems are normally offset by the actions of endogenous salt excreting systems (atrial natriuretic peptide and prostaglandins) ***(Nogueria et al., 2000).***

In patients without heart failure, atrial natriuretic peptide increases sodium and water excretion by direct effect on glomerular and tubular function, as well as by an inhibitory effect on the release and actions of renin and vasopressin ***(Troughton et al., 2000).***

Counterbalancing effects of atrial natriuretic peptide are lost in patients with heart failure who show little change in renin releases or sodium excretion following infusion of the peptide ***(Fruhwald et al., 1999).***

This attenuated natriuretic response seems to be directly attributable to decreased renal blood flow, which alters intrarenal haemodynamic and triggers intrarenal release of vasoconstrictors ***(Masson et al., 2000).***

Renal hypoperfusion leads to the intrarenal release of prostaglandins that exert some natriuretic and diuretic effects. However, these actions are limited by the inhibitory effects that renal hypoperfusion itself has on sodium and water excretion ***(Kay et al., 2001).***

Clinical features of heart failure

In children, the signs and symptoms of heart failure are similar to those in adults ***(Berstien, 2004).***

Patients with heart failure present with a variety of symptoms, most of which are non-specific ***(Burch, 2002).***

The accuracy of diagnosis by presenting clinical features alone is often inadequate ***(Kay et al., 2001).***

Several epidemiological studies have used clinical scoring system to define heart failure, although the use of these systems is not recommended for routine clinical practice ***(Collins-Nakai, 2002).***

In infants, heart failure may be difficult to identify. prominent manifestations include tachypnea, feeding difficulties (the infant with heart failure often takes less volume per feeding, becomes dyspneic while sucking and may perspire profusely), poor weight gain, excessive perspiration, irritability, weak cry, and noisy labored respiration with intercostals and subcostal retraction as well as flaring of the alae nasi ***(Bernstien,2004).***

Table (3): Symptoms and signs in heart failure (Watson et al., 2000).

Symptoms:
Dysnea
Orhopnea
Paroxysmal nocturnal dysnea
Reduced exercise tolerance,lethargy,fatigue
Nocturnal cough
Wheeze
Ankle swelling
Anorexia
Symptoms of the underlying cause
Signs:
Cachexia and muscle wasting
Tachycardia
Pulses alternans
Elevated jugular venous pressure
Displaced apex beat
Right ventricular heave
Crepitations or wheeze
Third heart sound
Edema
Hepatomegally(tender)
Ascites

Investigations in heart failure

Clinical assessment is mandatory before detailed investigations are conducted in patients with suspected heart failure, although specific

clinical features are often absent and the condition can be diagnosed accurately only in conjunction with more objective investigations, particularly echocardiography***(Bowen,2000).***

Although echocardiography is now increasingly available, appropriate pre-referral investigations include chest radiography, 12-lead electrocardiography and renal chemistry ***(Elliot, 2000).***

Chest X-ray examination:

The chest X-ray examination has an important role in the routine investigations of patients with suspected heart failure; and it may also be useful in monitoring the response to treatment ***(Soongswang et al., 2002).***

Cardiac enlargement (cardiothoracic ratio >50%) may be present. the presence of cardiomegaly is dependent on both the severity of haemodynamic disturbance and its duration. cardiomegaly is frequently absent ***(Shaddy, 2001).***

An increased cardiothoracic ratio may be related to left or right ventricular dilatation, left ventricular hypertrophy, and occasionally a pericardial effusion, particularly if the cardiac silhouette has a globular appearance. Echocardiography is required to distinguish reliably between these different causes ***(Nougueria et al., 2000).***

In the presence of high pulmonary venous pressures frank pulmonary edema occurs, with" bats wing" appearance in the lungs, although this is dependent on the rate at which pulmonary edema has developed. In addition, pleural effusions occur, normally bilaterally, but if they are unilateral the right side is more commonly affected ***(Gordon and child, 2000).***

12-Lead electrocardiography:

The 12-lead electrocardiography tracing is abnormal in most patients with heart failure, although it can be normal in up to 10% of cases ***(Nugent et al., 2001).***

Common abnormalities include Q waves, abnormalities in the T wave and ST segment, left ventricular hypertrophy, bundle branch block, and atrial fibrillation ***(Kay et al., 2001).***

Electrocardiography is a useful screening test as a normal electrocardiographic tracing makes it unlikely that the patient has heart failure secondary to left ventricular systolic dysfunction ***(Petrson, 2000).***

The combination of normal chest X-ray finding and abnormal electrocardiographic tracing makes a cardiac cause of dysnea very unlikely (***Dimitriu et al., 2000).***

Echocardiography:

Echocardiography is the single most useful non-invasive test in the assessment of left ventricular function; ideally it should be conducted in all patients with suspected heart failure. Although clinical assessment, when combined with chest X-ray examination and echocardiography, allows a preliminary diagnosis of heart failure, echocardiography provides an objective assessment of cardiac and function (***Collins-Nakai, 2002).***

Management of heart failure

Management of acute heart failure:

Common presenting features include anxiety, tachycardia, and dysnea. Pallor and hypotension are present in more severe cases. The triad of hypotension, oliguria and low cardiac output constitutes a diagnosis of cardiogenic shock (***Balaguru et al., 2000).***

Severe acute heart failure is a medical emergency, and effective management requires an assessment of the underlying cause, improvement of the haemodynamic status, relief of pulmonary congestion and improves most of tissue oxygenation (***Millane et al., 2000).***

Oxygen should be administrated initially; long term use of oxygen may be counterproductive, perhaps because of its effect as a systemic vasoconstrictor. Oxygen is administrated using a rigid plastic hood in neonates and nasal canula in older children ***(Wernovsky and Hoffman, 2001).***

In the acute management of severe cardiac failure, endotracheal intubation and mechanical ventilation may be indicated. Children may be present in extremis with respiratory failure due to fatigue of the overworked ventilatory muscles. After intubations and mechanical ventilation, paralysis and deep sedation can reduce these patients' requirements for cardiac output, allowing time for more definitive management of their heart failure (***Clark, 2000).***

Arterial blood gases provide valuable information about oxygenation and acid-base balance. The base excess is a guide to actual tissue perfusion in patients with acute heart failure. A worsening (more negative) base excess generally indicate lactic acidosis, which is related

to anaerobic metabolism, and is a poor prognostic feature. Correction of hypoperfusion will correct the metabolic acidosis, bicarbonate infusions should be reserved for only the most refractory cases ***(Davies et al., 2000).***

Intravenous loop diuretics, such as furosemide (lasix), induce transient venodilatation, when administrated to patients with pulmonary edema, and this may lead to symptomatic improvement even before the onset of diuresis. Loop diuretics also increase the renal production of vasodilator prostaglandins ***(Prandota, 2001).***

In case of severe refractory heart failure in which the cardiac output remains critically low, the circulation can be supported for a critical period of time with inotropic agents. For example dobutamine and dopamine have positive inotropic actions, acting on the β-1 receptors in cardiac muscle. ***(Gibbs et al., 2000).***

Morphine (0.1mg/kg) and other sedatives may be useful in treating the tachypneic, dyspenic and dusky infant who has severe respiratory distress associated with congestive cardiac failure and pulmonary edema. Sedation may results in apnea in children who have impeding respiratory failure from fatigue and in those with underlying pulmonary disease. Close monitoring and preparations for emergency incubation are warranted if sedatives are used ***(Calligaro and Burman, 2001).***

Fever should be treated aggressively in children with acute heart failure. Fever increases cardiac output approximately 10% to 15% per degree centigrade ***(Clark, 2000*)*.***

Management of chronic heart failure:

General advice:

Social activity:

Social activity should be encouraged, and care should be taken to ensure that patients avoid social isolation.

If possible, patients should continue their regular lifestyle with adaptations to accommodate a reduced physical capacity where appropriate **(Wernovsky and Hoffman, 2001).**

Table (4): Management of chronic heart failure (***Millane et al., 2000).***

General advice
Social activity
Vaccination (influenza, pneumococcal)
General measures
Diet
Anemia management
Treatment options-pharmacological
Diuretics
Dioxins
Angiotensin converting enzyme inhibitors
B-blockers
Spironolactone
Vasodilators (hydralazine/nitrates)
Anticoagulation
Antiarrhytmic agents
Positive inotropic agents
Treatment options-device and surgery
Valve replacement (or repair)
Pacemaker or implantable cardiodefibrrillator
Ventricular assist devices
Heart transplantation

Immunization and antibiotic prophylaxis:

Chronic heart failure predisposes to and can be exacerbated by pulmonary infection and influenza and pneumococcal vaccinations should therefore be considered in all patients with heart failure. Antibiotics prophylaxis, for dental and other surgical procedures, is mandatory in patients with primary valve disease and prosthetic heart valves ***(Balaguru et al., 2000).***

- **General measures:**

Diet and nutrition:

Infants with heart failure may fail to thrive because of increased metabolic requirements and decreased caloric intake. Increasing daily calories is an important aspect of their management. Severely ill infant may lack sufficient strength for effective sucking because of extreme fatigue, rapid respirations, and generalized weakness. In these circumstances, nasogastric feeding may be helpful ***(Bernstein, 2004).***

Restriction of sodium may be useful as an adjunct to treatment with high dose diuretics, particularly if the condition is advanced. In general, patient should be advised that they should avoid foods that are rich in salt and not to add salt to their food (***Gibbs et al., 2000).***

The ideal food for infants is breast milk because of its low solute and low salt content which is ideal for these patients.

Fluid restriction should be considered in patient with severe symptoms, those requiring high dose diuretics, and those with tendency towards excessive fluid intake. High fluid intake negates the positive effect of diuretics and induces hyponatremia ***(Prandota, 2001).***

Management of anemia

Anemia often occurs in children with chronic cardiac failure. It is usually a mild normochromic anemia, not related to iron or nutrient deficiency, and may be similar to the "anemia o chronic disease". It may improve with heart failure treatment. ***(Morrow, 2000).***

In patients with severe uncompensated heart failure, severe anemia imposes a cardiac volume overload proportional to the degree of anemia. A dysfunctional left ventricle may lack the contractile reserve to compensate for anemia by increased cardiac output ***(Hobbs et al., 2000).***

Transfusion is usually tolerated if given slowly. Unfortunately, the small number of leukocytes that contaminate packed erythrocyte transfusion expose the patient to foreign antigens and may make tissue matching for subsequent cardiac transplantation problematic ***(Clark,2000).***

Pharmacological management

Table (5): Aims of pharmacological management in chronic heart failure ***(Gibbs et al., 2000).***

To achieve improvement of symptoms
Diuretics
Digoxin
Angiotensin converting enzyme inhibitors
To achieve improvement in survival
Angiotensin converting enzyme inhibitors
β-Blockers
Oral nitrates plus hydralazine
Spironolactone

Diuretics

Diuretics are effective in providing symptomatic relief and remain the first line treatment, particularly in the presence of edema. Nevertheless, there is no direct evidence that loop and thiazide diuretics confer prognostic benefit in patients with congestive heart failure ***(Fogel, 2002)***.

Digoxin:

Digitals have been used in medical treatment of congestive heart failure for more than 200 years ***(Clarck, 2000).***

In human with congestive heart failure, coronary sinus norepinephrine decreases within 30 minutes of intravenous administration of digoxin ***(Kanstrup et al., 2000).***

Also it was found that digitals reduce the activation of renin-angiotensin-aldosterone system ***(Diller and Smucker, 2000).***

Angiotensin converting enzyme (ACE) inhibitors:

Angiotensin converting enzyme inhibitors(ACEIs), when used in asymptomatic and symptomatic patients with left ventricular systolic dysfunction, improve long term cardiac morbidity, mortality and quality of life(Calligaro and Burman,2001). They inhibit the production of angiotensin II, a potent vasoconstrictor and growth promoter ***(Gibbs et al., 2000).***

Angiotensin receptor antagonists:

Orally active angiotensin II type 1-receptor antagonists, such as losartan, represent new class agents that offer an alternative method of blocking the renin-angiotensin-aldosterone system. The effect of angiotensin II receptor antagonists on haemodynamic, neuroendocrine activity and

Exercise tolerance resembles those of ACE inhibitors ***(Balaguru et al., 2000).***

β-Blockers:

For many years, physicians believed that beta-adrenergic blockers were contraindicated in patients with chronic heart failure because the sympathetic nervous system provided important short term support for failing heart (***Bruns and Canter, 2002).***

From haemodynamic point of view it was believed that beta-blockers have adverse effects in patients with advanced left ventricular heart failure ***(Shaddy et al., 2002).***

Since the realization that long term activation of the sympathetic nervous system may be deleterious to the heart, there has been increasing interest in the use of β-blockers in the treatment of heart failure ***(Gachara et al., 2001).***

Hydralazine-isosorbide dinitra combination (vasodilator agents):

This combination is an alternative therapy when angiotensin converting enzyme inhibitors are contraindicated or cannot be tolerated. (***Vaskamann et al., 2001).***

Other positive inotropic agents:

Except for cardiac glycoside all positive inotropes are reserved for parenteral use in end stage heart failure, as abridge to transplantation or in acute exacerbation of heart failure. ***(Gibbs et al., 2000).***

β-agonists:

Available β-agonists include dobutamine (predominant β-1, less β-2 effects) and dopexamine (predominant β-2, some β-1effects) they cause

acute haemodynamic improvement followed by tolerance after several days ***(Venugopalan and Worthing, 2000).***

cAMP-phosphodiesterase inhibitors

They increase contractility (cAMP) and induce vasodilatation (vascular cAMP); they induce short term haemodynamic improvement and have some value for the treatment of acute exacerbation of heart failure ***(Balaguru et al., 2000).***

Cardiac transplantation:

The outcome in cardiac transplantation is now good, with long term improvement in survival and quality of life of patients with severe heart failure ***(Millane et al., 2000).***

The long-term survival of the transplanted human heart is compromised by accelerated graft atherosclerosis, which results in small vessel coronary artery disease and an associated deterioration in left ventricular performance. This can occur as early as three months and is the major cause of graft loss after the first year ***(Chiu et al., 2001).***

The anti-rejection regimens currently used may results in an acceleration of coronary atherosclerotic vascular disease (***Young, 2000).***

Rejection is now a less serious problem, with the use of cyclosporine and other immunosuppressant agents ***(Venugopalan et al., 2000).***

The supply of donors limits the procedure. 20% of patients listed for transplantation die on the waiting list, with 60% receiving transplants at two years (most within 12 months ***)(Nield et al.,2000).***

Intra-aortic balloon counter pulsation and left ventricular assist devices may be valuable during the wait for transplantation ***(Wand et al., 2001).***

II- Breastfeeding Management

Benefits of breastfeeding

Breast milk is the natural first food for babies, it provides all energy and nutrients that the infant needs for the first months of life, and it continues to provides up to half or more of a child's nutritional needs during the second half of the first year, and up to one-third during the second year of life ***(Kramer , 2001).***

Human milk provides optimal benefits for all infants, including premature and sick newborns.

The American **A**cademy of pediatrics encourages pediatricians to promote, protect, and support breastfeeding in their individual practices as well as in hospitals, medical schools, communities, and the nation (**American *Academy of Pediatrics Policy Statement, 1997*).**

Breastfeeding provides optimum health, nutritional, immunological and developmental benefits to children and protection from postpartum complications and future diseases for mothers **(*Kramer, 2001*).**

Benefits of breastfeeding for children:

1. **Enhancement of immune system and resistance to infections**: The infant's immune system is not fully mature until about two years of age. Human milk contains infections fighting factors transferred to the child including agents that act against viruses, bacteria, and intestinal parasites.

Breastfeeding reduces upper respiratory and lower respiratory tracts infection, urinary tract infections and diarrhea.

Also human milk contains factors enhance immune response towards influenza, tetanus, diphtheria and polio.

Breastfeeding protect babies from sudden infant death syndrome ***(Ryan, 2002).***

2. **Protection against chronic diseases**: Anti-inflammatory factors in human milk reduce the incidence of diabetes mellitus and some bowel diseases as ulcerative colitis and Crohn's disease. Babies on breastfeeding are less likely to develop Hodgkin's disease or leukemia ***(Kramer, 2001).***

3. **Nutritional, physical and mental benefits**: Human milk is ideally balanced and easily digested form of infant nutrition.

Human milk is less stressful on immature infant kidneys and contains lipids and enzymes that promote efficient digestion and enhance nutrient absorption. The composition of human milk changes with the age of the baby to fulfill the changing needs for the growing child. Breastfeeding reduces the risk of childhood obesity. Brain growth is enhanced by human milk as it contains polyunsaturated long-chain fatty acids. School age children who were breastfed score higher on cognitive and IQ tests and tests of visual acuity. Breastfeeding decreases the incidence of dental cavities and the need for orthodontist **(*Ryan, 2002)***.

Benefits of breastfeeding for the mothers:

1. Women who start breastfeeding immediately after birth increase the level of Oxytocin hormone which stimulates uterine contraction lowering the risk of postpartum hemorrhage ***(Kramer, 2001).***

2. Women who breastfeed are more likely to lose their pregnancy weight and less likely to become obese ***(Ryan, 2002).***

3. Breastfeeding reduces the risk of ovarian and premenopausal breast cancer, heart diseases, and osteoporosis. The more months the women breastfeed over their lifetime, the greater the protection ***(Kramer, 2001).***

4. Breastfeeding is a natural contraception as it inhibits ovulation during lactation by the increased level of prolactin, which inhibits the secretion of pituitary gonadotropins ***(Kramer, 2001*)*.**

5. Breastfeeding reduces the risk of postpartum maternal depression and psychosis and yields the emotional satisfaction to both mother and her baby ***(Rayan, 2002***).

6. The economic benefits of breastfeeding are notable as breast milk does not cost money in comparison with human milk substitutes ***(Kramer, 2001).***

7. Formula has along history of recalls for bacterial contamination or mis-manufacture which results in many cases of illness, permanent injury, or death ***(Ryan, 2002)***.

Benefits of the breastfeeding for low birth weight infants:

Mothers of LBW infants produce milk higher in protein and other nutrients. Human milk contains an enzyme that helps LBW babies to digest fat more efficiently. Breastfeeding of LBW infants helps the protection of them against gastrointestinal and infectious disease. Also, human milk enhances brain stem maturation in premature and LBW infants and raises childhood IQ test scores. Breastfeeding the premature and LBW infants reduces the hospital costs and length of hospital stay ***(Julie et al., 2003)***.

Exclusive breastfeeding

Exclusive breastfeeding (EBF) means that only breast milk is given to the baby without any other additives for the first six months of life ***(WHO, 2000)***.

Despite of high BF rates, exclusive breastfeeding (EBF)

Rates remain low in many developing countries despite numerous health benefits associated with this behavior ***(WHO, 2002)***.

In Egypt, exclusive breastfeeding up to six months is around 15% according to the results of demographic health survey carried out by Egyptian Ministry of Health and Population in 2005***(DHS,2005).***

Complementary feeding practices vary substantially across regions, and include the addition of liquids, porridges, and semisolid foods to the infant's diet very early in life. This has been associated with high rate of diarrhea which is a very common cause of infant mortality.

For these reasons WHO has recommended EBF up to six months ***(WHO, 2002)***.

In addition to the benefits of breastfeeding - whether to the mothers or to infants - exclusive breastfeeding reduces the infant mortality due to common childhood illnesses, such as diarrhea or pneumonia, and helps for quicker recovery during illness.

Also EBF for a minimum of four months decreases the risk of Type I diabetes mellitus (insulin-dependent diabetes mellitus) for children with a family history of diabetes. In addition, EBF decreases the incidence of asthma and eczema ***(Ryan, 2002)***.

Promotion of successful breastfeeding

WHO and UNICEF launched the Baby-Friendly Hospital Initiative (BFHI) in 1992 and recently updated it in 2006. The aim was to implement the Global Strategy of Infant d Young Child Feeding released by the World Health Assembly in 2003 which included the BFHI-updated

with evidence to strengthen maternity practices that support breastfeeding. "The foundation for the BFHI is Ten steps for successful breastfeeding in the context of protecting, promoting and supporting breastfeeding ". These Ten steps are as follows:

1. Have a written breastfeeding policy that is routinely communicated to all health care stuff.

2. Train all health care staff in the skills necessary to implement this policy.

3. Inform all pregnant women about the benefits and management of breastfeeding.

4. Help mothers to initiate breastfeeding within the first hour after birth (through skin to skin).

5. Show mothers how to breastfeed and how to maintain lactation even if they are separated from their infants.0

6. Give newborns no foods or drinks rather than breast milk, unless medically initiated.

7. Practice rooming-in which means allowing mothers and infants to stay together 24 hours a day.

8. Encourage breastfeeding on demand, day and night.

9. Give no artificial teats or pacifiers to the breastfed infants.

10. Foster the establishment of breastfeeding support groups and refer mothers to them on discharge from hospital or clinic ***(WHO, 2002).***

When to start breastfeeding?

The baby should be placed skin to skin immediately after delivery and left for one uninterrupted continuous hour or up to the first breastfeed. Thereafter baby is fed on demand or to the feeding cues but should not be

Left for more than 3 hours without breastfeeding in the first few days. The more the mother nurses her baby, the more milk is produced and the likely the baby is to lose weight.

Breastfeeding can be stopped once the baby falls asleep or refuses to suck. As he \ she grows and gets older, breastfeeding will gradually improve. The mother will most likely have to help her baby to open his mouth wide enough to attach to the areola as much as possible.

Many low birth weight (LBW) babies nurse only one breast at each feeding, so mothers will need to pump or massage and express the other breast to maintain milk flow. While these babies are training at the breast, they can receive any extra breast milk-expressed by the mother by a cup or spoon. Lactation depends on the suckling-swallowing-breathing rhythm. If the suckling reflex is poor as there is lack of coordination between suckling and swallowing, the baby should be fed by gastric tube with encouraging skin-to-skin contact to develop his\her feeding reflexes ***(Kavanaugh et al., 1995).***

Basic feeding techniques.

Frequency of feeding:

Infants who are breastfeeding well will feed 8 to 12 times or more in 24 hours, for a minimum of 8 feedings every 24 hours. Limiting the time at the breast is not necessary and may be harmful to the establishment of a good milk supply ***(Mark et al., 2003).***

Unrestricted breastfeeding helps prevent pathological engorgement, increases the duration of breastfeeding, decreases initial infant weight loss, increases rate of weight gain, promotes earlier onset of mature milk production ***(Ellerebee et al., 1999)*** decreases the incidence of jaundice, and stabilizes neonatal serum glucose levels***(Yamauchi,1997)***.

In multiple infants, breastfeeding is possible. Research and case studies have demonstrated that most mothers of multiples are capable of producing most or all of the milk that two to four infants require ***(Bleyl, 2001).***

The benefits of breastfeeding are particularly advantageous for twins, triplets, and other higher order multiples. In addition to offering optimal nutrition and immunological protection to these often preterm or otherwise compromised infants, breastfeeding helps ensure frequent mother-infant interaction with each baby***(Gromada, 1999).***

Attention to early feeding cues facilitates correcting latch-on and effective suckling which reinforces the mother's response to her infant.

Table (6): Cues in baby watching

Stage of readiness to feed	Baby cue
Early	Wiggling, moving arms or legs
Early	Rooting, fingers to mouth
Mid	Fussing, squeaky noises
Mid	Restless, crying intermittently
late	Full cry ,aversive screaming pitch, color turns red

Feeding the baby at night:

Once lactation is established, night feds provide the infant with a substantial proportion of his 24 h intake. It has been demonstrated that prolactin release in response to night-time suckling is greater than during the day (***Gupta et al., 2001***); thus milk production may get its greatest 'boost' when the baby feeds at night, and if the milk is not removed as it is formed the volume of the milk in the breast will rapidly exceed the capacity of alveoli. The consequent engorgement is not only uncomfortable for the mother, but it will begin the process of lactation suppression ***(Royal College of Midwives, 2002).***

Positioning and latch-on:

It is unclear wither the use of specific breastfeeding position in the delivery room is correlated with better outcome; therefore, it would be prudent to allow the mother to breastfeed in any position that seems comfortable for her or her infant. Options include the cradle hold, cross cradle hold, football hold, side-lying position and Australian hold.

To achieve a proper latch-on, the mother should support her breast using the C-hold (*Figure* 1). It is important not to have the fingers too close to the nipple because this can result in distortion of the natural shape of the nipple. The infant is then brought close to the breast, with the mother just touching the nipple gently on the baby's lips. This touch should initiate a reflex causing the infant to open its mouth widely. At this point, the infant should quickly be brought closer to allow a proper latch-on. If the latch-on is done correctly, the entire nipple and most of the areola should be in the baby's mouth. Newborns are often quite alert during the first hour of life. If a good latch-on is achieved, the infant should be sucking well at the breast and may continue to do so for about 10 minutes ***(Sinusas&Gagliardi, 2001).***

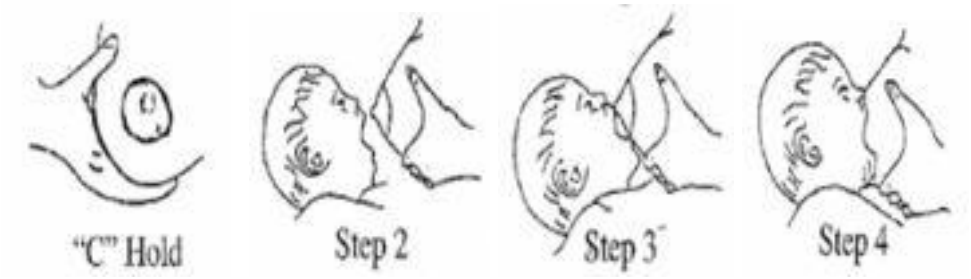


Figure (1) c-hold technique of latch on

Before removing the baby from the breast, the mother breaks the suction by inserting her finger into the baby's mouth and gently pressing the baby's chin down. Initially, the baby tends to feed for several minutes at each breast. The resulting reflex (let-down reflex) in the mother triggers milk production ***(Market al., 2003).***

The production of milk depends on sufficient suckling time, so feeding times should be long enough for milk production to be fully established. During the first few weeks, the infant should be encouraged to nurse on both breasts with each feeding; however, some infants fall asleep while feeding at the first breast. The breast used last should be used first for the next feeding ***(Mark et al., 2003)***.

Mechanism of breastfeeding:

While the baby is breastfed, his jaw and tongue most work together in a coordinated rhythm. Once the baby latches on the breast, the baby's tongue cups the breast in a rhythmic motion, pressing his mother's breast up against his palate. This flattens and elongates the flesh around his mother's nipple and the back of his tongue drops to form a grooved passageway for the milk to flow from the nipple. Then the baby swallows and takes a breath. The baby's lips should be flanged out and rest against the breast to make a seal **(*Mohrbacher&Stock, 2005)***.

Evacuation of the breast:

Fat content of milk increases during the feeding. Therefore time limits or enforced change from the first to the second breast should be avoided **(*Woolridge, 1990)***. Infants whose mother's milk has lower fat content will breastfeed longer to obtain sufficient calories **(*Tyson et al., 1992*)**.

Expression of breast milk:

If the baby is not able to breastfeed right, the mother should begin to pump her breasts as soon as possible. This will keep her hormonal level up and stimulate her milk to come in. She should begin pumping as soon after the delivery as possible if the baby is taken away to the neonatal intensive care unit (NICU). She should be shown how to manually express or pump as early as the baby is taken away to keep the hormonal level up and trigger the body to produce milk. Without regular and complete emptying of both breasts for at least 6-8 times a day, the milk supply may dry up.

There are two methods for milk breast expression:

A- Manual expression:

This is the preferred method. The mother should not be asked to express her milk if she had been recently fed her baby. It is started with massage of the whole breast in order to stimulate her milk flow and milk ejection reflex, then gentle expression of the milk is done inward, downward pressure on the areola. Pressure on the surrounding parts of the areola (on average 6 cm of diameter) between the thumb and the index finger was repeated in a clockwise direction. The backwards thrust of both fingers compresses the breast, and then the pressure of both fingers that pinch the areola advancing the hand has an effect to promote the release of the

milk, which is gathered in a plastic sterile container placed under the breast ***(Bergman and Jurisoo, 1994).***

B-Mechanical expression:

Table (7): Types of Breast Pumps

Type of pump	Description	Advantages	Disadvantages
Manual powered pump	**Hand powered**	**Small. Portable, quite, inexpensive.**	**Labor intensive single pumping only Difficult to achieve adequate suck frequency or suction pressure.**
Battery-powered pump	**Usually a hand pump that comes with a battery option; also, minimum electric pump**	**Small, portable, relatively quite, inexpensive Double pumping using two separate pumps**	**May go through batteries quickly May provide inadequate suction pressure With some models, only manual cycling**
Electric diaphragm pump	**Small electric pump that uses a circular diaphragm to create suction pressure**	**Relatively small and quite Double or single pumping**	**May be difficult to achieve enough suction pressure to empty breast fully With most models, only manual cycling. Requires electricity or car battery (with adapter option)**

Electric piston pump	**Medium-sized electric pump that uses a piston moving back and forth in a chamber to create suction pressure**	**Efficient and compact usually has optional carrying case (size of a briefcase or backpack) Double or single pumping Automatic cycling**	**More expensive Requires electricity o r car battery (with adapter option)**
	Large piston-driven electric pump that creates physiologic suction pressures and rates	**Highly efficient: most accurately recreates baby's suction pressure an cycling rate Double or single pumping Automatic cycling**	**Large and heavy Highly expensive: usually only practical to rent this type of pump Requires electricity**

(Biabioli, 2003).

Procedures of Pumping, Storing and

Transporting Breast Milk for Infants in NICU.

1. Wash the hands.

2. Massage the breast gently.

3. Pump the breast 10-15 minutes, 8-10 times a day. It's recommended that the mother pumps every 21\2-3 hours during the day and at least one time at night. Milk production depends upon supply and demand. The more often the mother pumps, the more milk will be produced.

4. Put the breast milk into a sterile container supplied by the hospital. Containers should be filled to the line marked to allow for expansion with

freezing. Place the cap on the bottle tightly without touching the inside of the cap or container.

5. Place a label including name, date, and time on each container.

6. Refrigerate or freeze milk after pumping. Breast milk may be stored for 48-72 hours (2-3 days) in the refrigerator, 2-3 months in the freezer and 6 months in a deep freezer. While freezing breast milk place it in the back of the freezer, not in the door.

7. If the mother is pumping for her infant in the hospital and she plans to take the milk at home then, put the container of milk on ice during transporting it.

8. If the milk will not be taken to the hospital within 48 hours, it should be put in the freezer and transported as frozen packed in blue ice in an insulated bag.

9. The mother may store milk from both breasts in one container. Each expression should be chilled or refrigerated after the pump session. The mother shouldn't add fresh pumped milk to frozen milk in a container.

10. Wash the parts of the pump that come into contact with the breasts or breast milk after each use in warm soapy water. Rinse thoroughly and allow to air dry. Once a day sterilize the parts of the pump for 20 minutes in boiling water ***(Human Milk Banking Association of North America, 2005).***

Artificial baby milk versus breast milk

Infant formula was designed to be a medical nutritional tool for babies who are unable to breastfeed. Evidence is piling up to show that formula is not a safe alternative for feeding babes as it does not fully meet the nutritional, growth and immunological needs of infants, leaving their immune systems flail. An infant's immune system has three aspects: her

own immature, developing immune system; the small component of immunities that passes through the placenta during natural child birth (and to a lesser degree with premature births and cesarean sections); and the most valuable, living portion that is passed on through mother's milk on an ongoing basis. Remove any of these components and you take away a vital supporting structure ***(Palmer, 2004).***

The relative risks of formula

It is clear that feeding infant an artificial formula instead of breastfeeding, increases their relative risk of death. Table (8) shows figure from a study measuring infant mortality risks during certain age ranges.

	0 to 2 months	2-3 months	4-5 months	6-8 months	9-11 months
WHO	6	4	2.5	2	1.5

Table (8) Suggest relative risks for infant deaths

(WHO, 2000).

A relative risk of 6 here means that a child who was not breastfed through the time period has six times the risk of dyeing during his first year as a child who had received any breast milk through that period.

How does formula play into these deaths?

Sudden Infant Death Syndrome

Sudden Infant Death Syndrome (SIDS) accounts for a full 10% of U.S. infant deaths. Several studies performed in the United States and other industrialized nations reveals increased risks of SIDS among babies who receive formula instead of breast milk. ***(Hauck et al., 2003).***

Heart, circulatory and respiratory failure

Formula-fed infants demonstrated many episodes of inadequate oxygenation, some apnea and high blood pressure, which were not seen among the breastfed infants ***(Wilson, 1998; Chen et al., 2000)*.**

Necrotizing enterocolitis

In the United Kingdom, it was discovered that confirmed cases of necrotizing enterocolitis occurred in three times as many infants who received no breast milk as in those who received both breast milk and formula. For infants who exclusively received both breast milk, necrotizing enterocolitis occurred six to ten times less often than among wholly formula-fed infants ***(Lucas & Cole, 1990)***.

Formula-fed infants may develop amore virulent Gram-negative and less diverse intestinal colonization pattern, which may promote a greater proportion of serious Gram-negative infections among these infants ***(AAP, 2005***).

Diarrhea:

A WHO study revealed a risk of diarrhea for formula-fed babies in developing nations averaging more than sixteen times that of breastfed babies. A summary article for industrialized nations demonstrated an average of triple the risk of diarrhea for formula-fed babies ***(WHO, 2000)***.

Respiratory illnesses:

Numerous studies document higher numbers of respiratory infections among formula-fed infants than among those who are breastfed. It is clear that respiratory infections are at least triple in the United States for formula-fed infants. The death rate is likely to be even higher, since some of these studies note that the severity and extent of respiratory illnesses are considerably higher once they occur ***(Bacharach et al., 2003)***.

Cancer:

A joint study between the United States and Canada on neuroblastoma, a common childhood cancer, revealed a doubled risk for children who did not receive breast milk for more than one year. Formula fed have an increased risk of childhood leukemia ***(Daniels et al., 2002)***..

Low Birth-Weight and Pre-term Birth

Feeding with human milk, compared with formula, is probably advantageous for preterm a low birth-weight infants, and it has been recommended that entral feeding should be introduced as early as possible, to reduce the risk of systemic infection among very premature infants ***(Killbride et al., 2003; AAP, 2005).*** A U.S. study performed at George Washington University Hospital found that the number of infections among formula-fed infants in the neonatal intensive care unit

2.5 times than that among those receiving human milk ***(El-Mohandes et al., 1997)***.

Another study found 6 times the duration of upper respiratory infections among formula-fed infants in the intensive care unit than among formula-fed infants in the intensive care unit than among those receiving human milk ***(Blaymore-Bier et al., 2002)***.

And finally, it is worth noting that the eye damage that can occur in very low birth-weight infants, retinopathy of prematurity, it occurs only half as often in infants who receive some breast milk ***(Hylander et al., 2001)***.

III- Skin-to-skin care.

Introduction

Early and prolonged skin-to-skin contact in the postnatal period is recommended by The UNICEF Baby Friendly Hospital Initiative (BFHI) which should last until the first feed or for as long as the mother wishes. Skin-to-skin care (SSC) is defined by BFI as holding the baby naked in a prone position against the mother's skin between the breasts. Skin-to-skin contact is seen as a potential mechanism for promoting early breastfeeding ***(Carfoot et al., 2005).***

A series of worldwide studies documented the safety of skin to skin care (SSC) and its effectiveness in promoting physiologic stability (temperature, heart and breathing rate) in infants ***(Bohnhorst et al., 2001).***

Although the relationship between SSC and lactation has been studied less symmetrically, the duration of breastfeeding appears to be higher for skin-to-skin infants than for incubator controls ***(Charpak et al., 2001).***

Mothers frequently note feeling of milk ejection and leaking, and many reported that they express the largest milk volumes immediately following SSC. Yet milk ejection is but one of the effects of Oxytocin that occurs during the developing maternal infant relationship. Emerging evidence reveals that Oxytocin coordinates both the causes and effects of positive social interactions ***(Uvnas-MOberg, 1997).***

During social interaction, oxytocin can be released by sensory stimuli perceived as positive, including touch, warmth and odors ***(Uvnas-Moberg, 1998).***

Even with just 30 minutes daily of skin-to-skin holding, mothers experience greater increase in milk volume between 2 to 4 weeks than mothers not doing skin-to-skin holding.

Furthermore, it has been speculated that skin-to-skin holding may trigger the production of maternal milk antibodies to specific pathogens in the infant's environment through mechanisms in the enteromammary pathway ***(Hurst et al., 1997).***

Procedures for skin to skin holding.

First, infants can be safely placed in SSC while very small and mechanically ventilated ***(Tornhage et al., 1999).***

Second, there is no scientific reason to restrict the duration of SSC, unless an infant becomes physiologically unstable while on the mothers chest. Typically, SSC session is ended based upon the mother's availability rather than infant criteria.

Third, the position of the infant in SSC is important in maintaining physiologic stability, and recliners are ideal in achieving this position. The infant should be placed upright between the mother's breasts, with the side of the face against the internal surface of one breast. The recliner is angled back to allow the infant's body to remain at a 45to 60 angle from the floor. A mirror positioned to allow the mother to observe her infant's face is helpful during these sessions ***(Kirsten et al., 2001).***

The infant should not have to turn the head toward the breast; the mother may need assistance in holding her breast so as to present the nipple squarely into the infant's mouth, which is stimulated to open by stroking the center of the lower lip with the nipple. When the lower lip is touched by the nipple the infant will open widely, extending the tongue under the nipple. The breast will be drawn into the mouth, the nipple and

the areola elongates into a teat, and the suckling reflex initiated ***(Righard& Alade, 1992).***

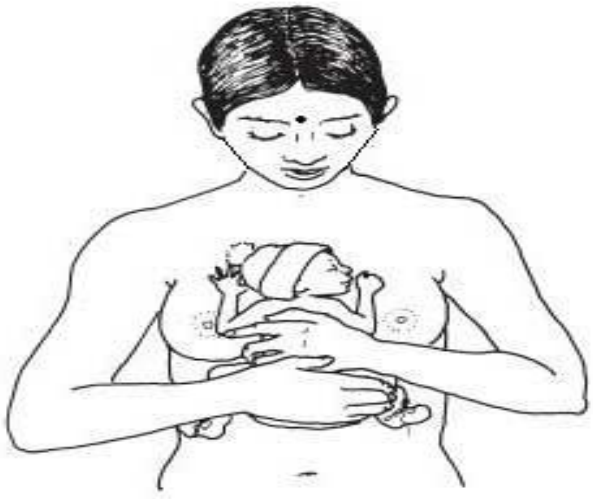


Figure (2).

The baby should be placed between the mother's breasts in an upright position

Kangaroo Mother Care

In 1978 Edgar Rey, a Colombian pediatrician, concerned with the problems arising from a shortage of incubators and the impact of separating women from their newborns in neonatal care units, developed what is called Kangaroo Mother Care (SSC), a healthcare technique for low birth weight infants that is at least as effective as traditional care in a neonatal care unit. In1979, Rey and Martinez started programme in Bogotá, Colombia, in response to shortage of incubators severe hospital infections and in 1983 UNICEF brought attention to this programme ***(Rey and Martinez, 1983).***

In 1996, the first international workshop, hosted by Adreano Cattaneo and his team, noted over thirty different terms used, agreed to use SSC as a definition of the programmer of skin-to-skin contact, breastfeeding and early discharge.

The term "K C" refers only to the intervention "intrahospital maternal-infant skin-to-skin contact". In 19998, the first international conference on SSC arranged by Susan Ludington-Hoe and also in the same year, second international workshop in Bogotá, Colombia, arranged by Nathalie Charpak and her team focused on research and implementation. In 2000, the third international workshop took place in Yogyakarta and Indonesia ***(Charpak et al., 2001).***

Definition of Kangaroo Mother Care

Kangaroo mother care is a universally available and biologically sound method of care for all newborns, but in particular for low birth weight babies, with three components….

1- Skin-to-skin contact

2- Exclusive breastfeeding

3- Support to the mother-infant dyad ***(Charpak et al., 2000).***

A- Skin-to-skin contact

The more skin-to-skin contact, the better the net result. It should ideally start at birth and for 24h but shorter periods are still helpful.

B- Exclusive breastfeeding

Breastfeeding can be either by direct suckling or by feeding expressed breast milk.

C- Support to the mother-infant dyad

It means that whatever is needed for the medical, emotional, psychological and physical well being of mother and baby is provided to them, without separating them. This might mean adding ultramodern equipment if available, or purely intense psychological support in contexts without any resources ***(Tessier et al., 1998).***

In Bogotá, Colombia, where SSC started, "early discharge" is regarded as the third part of the definition. This also a form of support where hospitals are overcrowded, but it also requires a good community support system ***(Cattaneo et al., 1998).***

The way of performing SSC.

In SSC, low birth weight babies who are unable to regulate their body temperature remain with their mothers as incubators, main source of stimulation and feeding. Newborns are attached to mothers and other caregivers' chests in skin- to- skin contact, wearing only a nappy and a baby bonnet, and are kept upright 24 hours a day. Mothers can share the role of provider of the kangaroo position with others, especially the babies' fathers without disrupting breastfeeding routine. The caregiver

should sleep in a semi-sitting position. The SSC begins as soon as the baby no longer requires other support from the neonatal care unit, although intermittent skin-to-skin contact has been used in ventilated infants ***(Ludington-Hoe et al., 1998).***

Exclusive breastfeeding (plus vitamins) is attempted, and growth is closely monitored. Breast milk is fortified if infants are not thriving. Infants will reject permanent contact once they achieve regulation of their body temperature, at a median age of 37 weeks after conception ***(Charpak et al., 1997).***

SSC usually starts in hospital with an adaptation process. During adaptation and after discharge, caregivers attend a day clinic where they are trained, infants are monitored, and the caregiver enmeshes in a social peer network. Care is thereafter provided at home with follow up visits as needed. SSC can be implemented in various facilities at different levels of care. It may be the best option if neonatal care units are unavailable. If a neonatal care is available but overwhelmed by demand, SSC allows rationalization of resources by feeding up incubators for sicker infants, and even in well-resourced neonatal care units, it still enhances bonding between mother and infant and breastfeeding ***(Charpak et al., 2001).***

Evidence backs the effectiveness and safety of SSC in stable preterm infants. In low birth weight infants weighting 2000 gms or less, who are unable to regulate their temperature, SSC is at least as safe and effective as traditional care with incubators ***(Charpak et al., 1997).***

An opened randomized controlled trial in Bogotá, Colombia, assessed the long term clinical effects of SSC by randomized 746 low birth weight infants. Followed up at 12 months of age corrected for gestational age (93% children) found that SSC had improved successful breastfeeding rates and infections were milder in those children. Hospital stay was

reduced in "Kangaroo" newborns weighting 1500 gms or less. A non-significant reduction in mortality and slight improvements in developmental indices were found with SSC. The investigators found no significant differences in physical growth patterns or in the rates of cerebral palsy, failure to thrive, visual problems, deafness, or hip dysplasia ***(Charpak et al., 2001).***

Blind assessment of bonding between mother and infant by using videos in a sub-sample of 488 mother-infant dyads found that bonding improved markedly with SSC, as did neuro- developmental evaluations in infants at higher risk. ***(Mew et al., 2003).***

Benefits of SSC.

Benefits of SSC to the baby:-

(i)- Many studies confirmed that skin-to-skin contact assists the preterm babies in the following:

1- Temperature regulation is at least as good as that obtained inside the traditional incubator. Some pieces of evidence suggest that it is even better.

2- Regular breathing patterns with a decrease of apneic episodes and periodic respiration are more frequent than in non exposed infants.

3- Trancutaneous O2 levels do not decrease.

4- Improved regulation of infant's behavioral state: longer alertness periods, less crying, etc.

5- No additional risk for infection.

6- Higher rates and higher duration of breastfeeding. ***(Anderson, 1991).***

(ii)- In another study the workers found that heart rate increased within normal ranges (144/152/148 b/m), skin temperature increased (36.3 vs. 36.9˚C), no difference in apnea or bradycardia and more quiet sleep (19% vs. 9.5%) ***(Ludington-Hoe et al., 1994).***

(iii)- In a randomized controlled trial, pneumograms have been performed in preterm infants during skin-to-skin contact. Heart rate and O2 saturation monitoring were not affected by skin-to-skin contact ***(Sontheimer et al., 1995).***

(iv)- Ludington-Hoe et al. carried out a randomized controlled trial in which SSC was done 2-3 hours a day. They found that there was no difference between infants of SSC and those of traditional care in abdominal temp. Higher toe temperature was found in SSC infants (35.1vs. 33.6˚C) and breast temperature of SSC infants' mothers was equal to neutral thermal environment ***(Ludington-Hoe et al., 2000).***

(v)- In another trial study evaluating how different delivery-ward routines influence temperature in newborns. The study results showed that delivery-ward routines influence skin temperature in infants in postnatal period. Allowing mother and her baby to the ward routine of skin-to-skin contact after birth may be a "natural way" of reserving stress-related effects on circulation induced during labour. On the other hand, extended skin-to-skin contact with the mother is the most effective way to maintain the infant's temperature and decrease the "stress of being born" ***(Bystrova et al., 2003).***

(vi)- Another study carried out by Chow et al. to test the hypotheses that SSC infants would have higher mean tympanic temperatures, less weight loss, more optimal behavioral states, and lower acuity (length of stay).infants on SSC had higher mean temperature (3703˚C vs. 37.0˚C), more quite sleep(62%vs. 22%), and less crying (2% vs. 6%). mothers should be encouraged to keep their stable preterm infants warm by skin-to-skin contact inside their clothing, thereby encouraging self-regulatory feeding ***(Chwo et al., 2004).***

(B)- Effects of SSC on the survival of small babies:

(i)- In evaluation of the results of introducing the kangaroo mother care, as the exclusive means of caring for low birth weight infants, instead of traditional incubators and standard equipment used, the survival of babies born<1500 grams improved from 70% to 90%. The method was well accepted by the community, and easily grasped by all hospital staff. ***(Bergman and Jurisoo, 1994).***

(ii)- Two cohort studies were carried out in 1994 and found that SSC was not associated with greater mortality than the control intervention (traditional neonatal incubator care unit) and that breastfeeding was more successful and lasted longer ***(Charpak et al., 1994).***

(iii)- SSC was safe regarding mortality and even suggested an almost two-fold reduction in mortality risk in kangaroo infants. Kangaroo

infants' early growth was as good as for same infants, and when they reached one year of corrected age, head circumferences of kangaroo infants were slightly better than those of same infants ***(Charpak et al., 1997).***

(C)- Effects of SSC on growth and development of babies:

(i)- Charpak et al. assessed in their study the effectiveness and safety of SSC on low birth weight infants. They conclude that their results supported the earlier findings of the beneficial effects of SSC on mortality and growth. Use of this technique would humanize the practice of neonatology, promote breastfeeding and shorten the neonatal hospital stay without compromising survival, growth or development ***(Charpak et al., 2001).***

(ii)- Another study examined whether kangaroo care (SSC) intervention in premature infants affects parent-child interactions and infant development. The workers of the study conclude that SSC had a significant positive impact on the infant's perceptual, cognitive and motor development and on the parenting process. They speculate that SSC has both a direct impact on infant development by contributing to neuropsychological organization and indirect effect by improving parental mood, perceptions and interactive behavior ***(Feldman et al., 2002).***

(iii)- The effects of mother-infant skin-to-skin contact(Kangaroo Mother Care) on autonomic functioning , was studied and concluded that

neurodevelopmental profile is more mature for SSC infants, particularly habituation and orientation***(Feldman and Eidelman, 2003).***

Benefits of SSC to the parents:

(i)- Mothers reported self-confidence, feelings of fulfillment, less stress and were confident in breastfeeding ***(Anderson, 1991).***

(ii)- An evaluation of kangaroo mother-child relationship as compared to control families was conducted by Tessier and Colombian team. Main findings were:

Kangaroo mothers had a better feeling of competence for caring for and raising their premature infants, they were also more sensitive to health and developmental needs of their infants at higher risk for developmental impairment and general morbidity. Additionally, a change in the family structure of kangaroo infants was observed, allowing for a greater participation of the father in the care of the infant, and an improvement in the maternal self-esteem. These changes persisted at least during the first year of life. These findings suggest that SSC could play an important role in preventing abandonment and child abuse, which are more prevalent in cases of prematurity and early and prolonged separation of the mother and her newborn infant ***(Tessier et al., 1998).***

(iii)- A comparison of kangaroo mother care (SSC) and conventional cuddling care (CCC) in premature and small for gestational age infants was done in randomized controlled trial.

It was find that, infants in both group's experienced equivalent maintenance of or rise in temperature while out of the incubators, equal weight gain, equal length of hospital stay and equal duration of breastfeeding ***(Kathryn et al., 2000).***

(iv)- an evidence based research showed that continuous skin-to-skin contact between the mother and the infant, exclusive breastfeeding, and early home discharge in the kangaroo position lead to significantly increased in body temperature and weight gain. A meta-analysis showed that kangaroo position increases the uptake and duration of breastfeeding. Investigations of the behavioral effects of SSC show rapid quiescence. The psychosocial effects of SSC included reduced stress, enhancement of mother-infant bonding, and positive effects on the family environment and the infant's cognitive development ***(Charpak et al., 2005).***

Benefits of SSC to the baby in special pathological situations:

* ***Respiratory distress****:*

(i)- A study was done on preterm intubated infants by exposing them to skin-to-skin contact with their parents. It concluded that this technique was safe and promoted parental attachment. ***(Gale et al., 1993).***

(ii)- A case study was done on mechanically ventilated infant. It was offered SSC while it was still ventilated. She thrived thereafter ***(Parker and Anderson, 2002).***

(iii)- The potential advantages of SSC for ventilated infants and their mothers have been discussed in a case study. The data from the study suggested that SSC might assist in, rather than retarded, recovery from respiratory distress. SSC might also foster maternal relaxation and minimize maternal stress ***(Swinth et al., 2003).***

**** Neonatal Jaundice:***

Ludington-Hoe and Swinth determined the safety and efficacy of allowing kangaroo mother care (SSC) one hour per day during the course of phototherapy using a fiberoptic phototherapy panel. This pilot work suggests that SSC using a fiberoptic panel during phototherapy may be safe, but further study was needed ***(Ludington-Hoe and Swinth, 2004).***

Financial Benefits of SSC:

A paper by Cattaneo and his colleagues reported on a multicenter trial conducted in five countries that evaluated in- hospital SSC (skin-to-skin contact and breastfeeding) resulted in SSC can save costs in hospital in developing countries ***(Cattaneo et al., 1998).***

In summary and from the previous show, there is sound evidence regarding effectiveness and safety of SSC in terms of mortality, early infectious morbidity, growth and development, promotion, and maintenance of breastfeeding. There is also good evidence on the beneficial effects of SSC on the establishment of a healthy bonding between mother and infant, which is also related to better cranial growth, good performance in neurodevelopmental tests and in the familial attitude towards the premature infant, and the provision of a cozy, nurturing and stimulating home environment.

SUBJECTS AND METHODS

I- Subjects

This is a non randomized controlled trial for assessing the effect of STS care on the vital signs on infant with heart failure according to the mode of feeding.

Sample

The study was conducted on 55 babies with congenital heart disease; 33 male and 22 female aged from a few weeks up to 2 years .They were grouped as 15 control and19 breastfeeding and 21 artificial fed babies.

Dropouts

From the study 2 patients |were under I.V. line medication and 3 patient's mother refuses to do STS care.

Duration of study

The clinical work was conducted over a period of about two years from 2008 to 2010 and from 10/2010 to present time data compilation, statistical analysis, drafting and revision

Site

The study was done in Cardiac Ward, in Abu El Resh Pediatric University hospital, of Cairo University Hospitals.

Selection criteria:

- All infants under 24 months of age hospitalized for heart failure
- Almost fully breastfeeding in the first six months and received foods after 7 months with continued breastfeeding or received infant milk

formula before six months and were then only on formula or other milks feeding.

- Received medication for heart failure

- Neither exposed to operative procedure nor operative procedure with residual cardiac dysfunction necessitating treatment.

Exclusion criteria

In the intervention groups we excluded:

- Infants with heart failure due to any other cause.

- Other congenital anomalies apart from heart defect

- Neurological disease

- Mother sickness or severely malnourished

- Infant with CHD that was totally corrected and heart failure is secondary to chest infection or other cause than heart.

***II*- Tools**

a- History taking using predesigned questionnaires including a full history, stressing on mother's antenatal history, perinatal history, post natal history for both mothers and babies, feeding history, vaccination history and morbidity and allergic history.

b- STAI test: This is a test which was designed to asses two different parameters; state of anxiety and trait anxiety by self assessment. The study used the parameter state anxiety which assesses the mother's feeling at a particular moment in time of examination. It was done before doing SSC and after doing it. The test is composed of 20 questions which are answered by the mother.

c- Pulse oximeter: the oxygen saturation of the babies was measured by using pulse oximeter. A pulse oximeter is a small medical device that

measures the oxygen saturation in blood. It is consisted of a computerized monitor and a lighted sensor probe that clips onto the patient index fingers or ear lobe or the bridge of the nose with a plastic clip. It measures the oxygen saturation of the blood hemoglobin in the tissue capillaries by transmitting a beam of light through tissue to receiver. As the amount of saturated hemoglobin alters the wave length of the transmitted light, analysis of the received light is transmitted into percentage of oxygen saturation of the blood. Normal range is 95-100% nanometer.

III-Methods

a- Assessment of the baby

All infant were admitted for one day and assessed as follow:-

a- Weight to nearest gm.

b- Supine height to nearest mm and body mass index.

c- Head circumference.

d- Vital signs (H.R, R.R, temp.)

e- PaO2 saturation, H.R, PO recorded just before the feeding, every min. during feeding & last 5min. after feeding (without SSC.).

b- Skin to skin care

SSC is a technique practiced on newborn, usually preterm, infants wherein the infant is held, skin-to-skin, with an adult. SSC for pre-term infants may be restricted to a few hours per day, but if they are medically stable that time may be extended, SSC seeks to provide restored closeness of the newborn with mother or father by placing the infant in direct skin-to-skin contact with one of them. This ensures physiological and psychological warmth and bonding. The kangaroo position provides

ready access to nourishment. The parent's stable body temperature helps to regulate the neonate's temperature more smoothly than an incubator, and allows for readily accessible breastfeeding.

Typically in SSC, the baby wears only a diaper and a head cap and is bended in an upright position to the mother’s bare chest by a strip of cloth in a manner that extends the baby’s head and neck to prevent apnea. The mother wears a shirt or hospital gown with opening to the front. The cloth wraps around and under the baby’s bottom to create flexion.

The tight bundling is enough for the mother’s breathing and chest movement to stimulate the baby’s breathing. The baby in SSC is ideally cared in this position for 24 /7 i.e. 24hrs a day seven days per week. SSC is a term used to indicate care of the baby in the SSC mode but for limited period of time.

c- Feeding modality

1- Breastfeeding

The mothers were asked to breastfed their babies and the proper technique of breastfeeding and correct latch on were clearly explained and demonstrated to them as follow:

To latch baby properly, turn baby toward you, stroke baby's lower lip lightly with your nipple and wait to nurse until baby has a very wide open mouth. Do not let the baby suck on the tip of your nipple. Typically this will feel very "pinchy" if your baby is not taking nearly the entire areola in her mouth.

Exclusive breastfeeding (EBF) means that only breast milk is given to the baby without any other additives for the first six months of life ***(WHO, 2000).***

2- Artificial feeding

Other baby chosen in the study were artificial feeding, all of them were formula feeding, the mothers were asked how the prepare the formula, frequency of feeding, sterilizing the prepared formula and was advised about breastfeeding for their future babies

d- Psychometery

It was done by using STAI test and the results were scored according to the scoring system of the test.

Statistical procedure

Data was statistically analyzed using SPSS (statistical package for social science) program version 13 for windows and for all the analysis a p value < 0.05 was considered statistically significant:

- Data are shown as mean, range or value and 95% confidence interval (95% CI) and frequency and percent.

Chi square test was done for qualitative variable analysis and p-value < 0.05 was considered significant.

Student t- test was done for normally distributed quantitative variables to measure mean and standard deviation and p-value < 0.05 was considered significant.

Mann-Whitney test was done for quantitative variables which are not normally distributed and p-value < 0.05 was considered significant.

ANOVA test was done to compare three variables **;** one qualitative variable and the other two are quantitative variables of normally distributed variables and p-value < 0.05 was considered significant to detect mean and standard deviation where post hoc tests done to detect the relationship between variables within groups .

Kruskal-Wallis test was done to compare three or more variables**;** one qualitative variable and the other variables are quantitative variables of none normally distributed variables and p-value < 0.05 was considered significant and to detect mean and standard deviation where post hoc tests were done to detect the relationship between variables within groups.

LSD test is a post hoc test it was done to variables of significant difference of more than two groups of normally distributed data after ANOVA test to detect the significant difference between either groups.

Repeated measures ANOVA test was performed to differentiate changes in different follow up results of normally distributed studied variables and p-value < 0.05 was considered significant.

All data are tested with kolmogorov- Smirnov Z test and most of them were found normally distributed and so presented with mean ± SD. And using parametric testes on doing association or correlation

Ethical consideration

Consent was taken from the authority of the Hospital of Cairo University to practice the study work.

An oral consent was taken from the mother undergone the study.

A full explanation was said to each mother of the study about the procedures done, how to do it, why to do, benefits, hazards and their outcome.

RESULTS

The study was conducted on 55 babies with congenital heart disease; 33 male and 22 female aged from a few days up to 2 years .They were grouped as:

Group I

19 breastfeeding babies with CHD who were cared with SSC

Group II

21 Artificial feeding babies with CHD who were cared with SSC

Group III

15 babies with CHD not exposed to SSC control group

Both breastfed and artificially fed and19 breastfeeding and 21 artificial fed babies

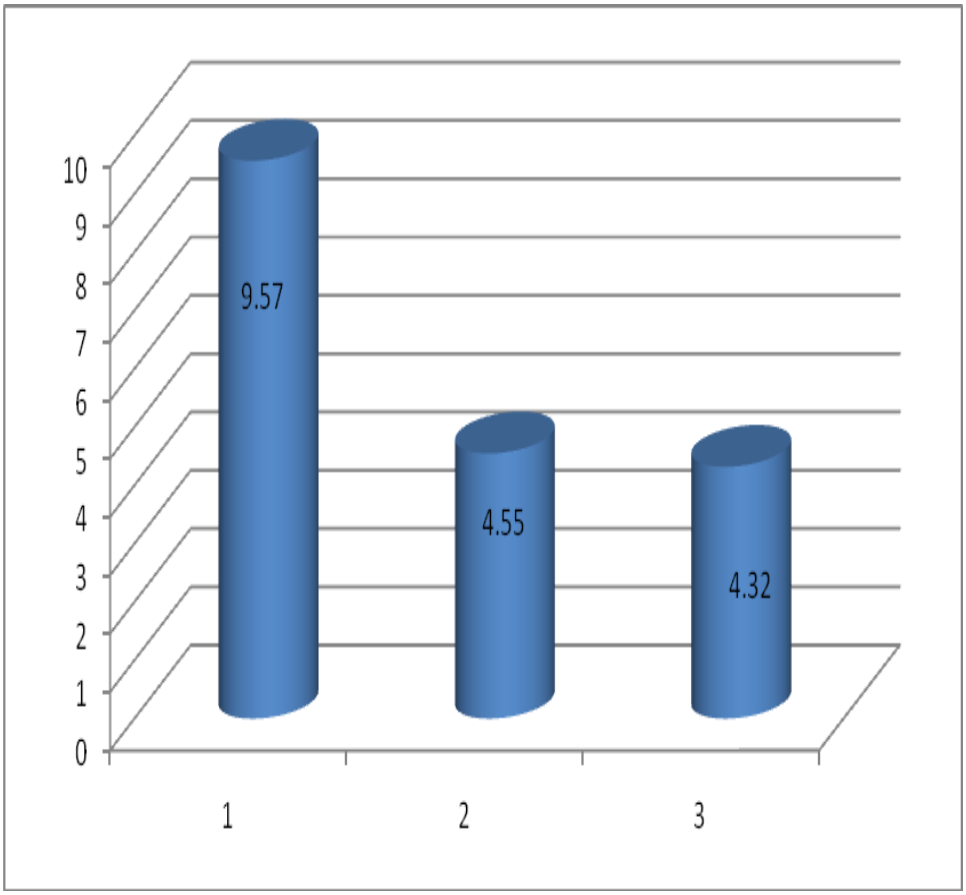
The findings are presented in the following tables:

Table (9): Comparison of age by mean and S.D. in the Studied Groups

Studied variable	**Control** *(N=15)* **Mean ± SD**	**Exclusive breast feeding and STS care** *(N=19)* **Mean ± SD**	**Artificial feeding and STS care** *(N=21)* **Mean ± SD**	**ANOVA Test:**	**P-value**	**LSD post Hoc test**
-Age (months)	9.57 ± 5.04	4.55 ± 2.81	4.32 ± 1.55	13.72	<0.01**	P1= < 0.01** P2=< 0.01** P3= > 0.05

The table show significant statistically difference in mean and SD in age by the studied groups

Figure (3): Comparison of age by mean and S.D. in the Studied Groups



1=control group

2= fully breastfeeding group

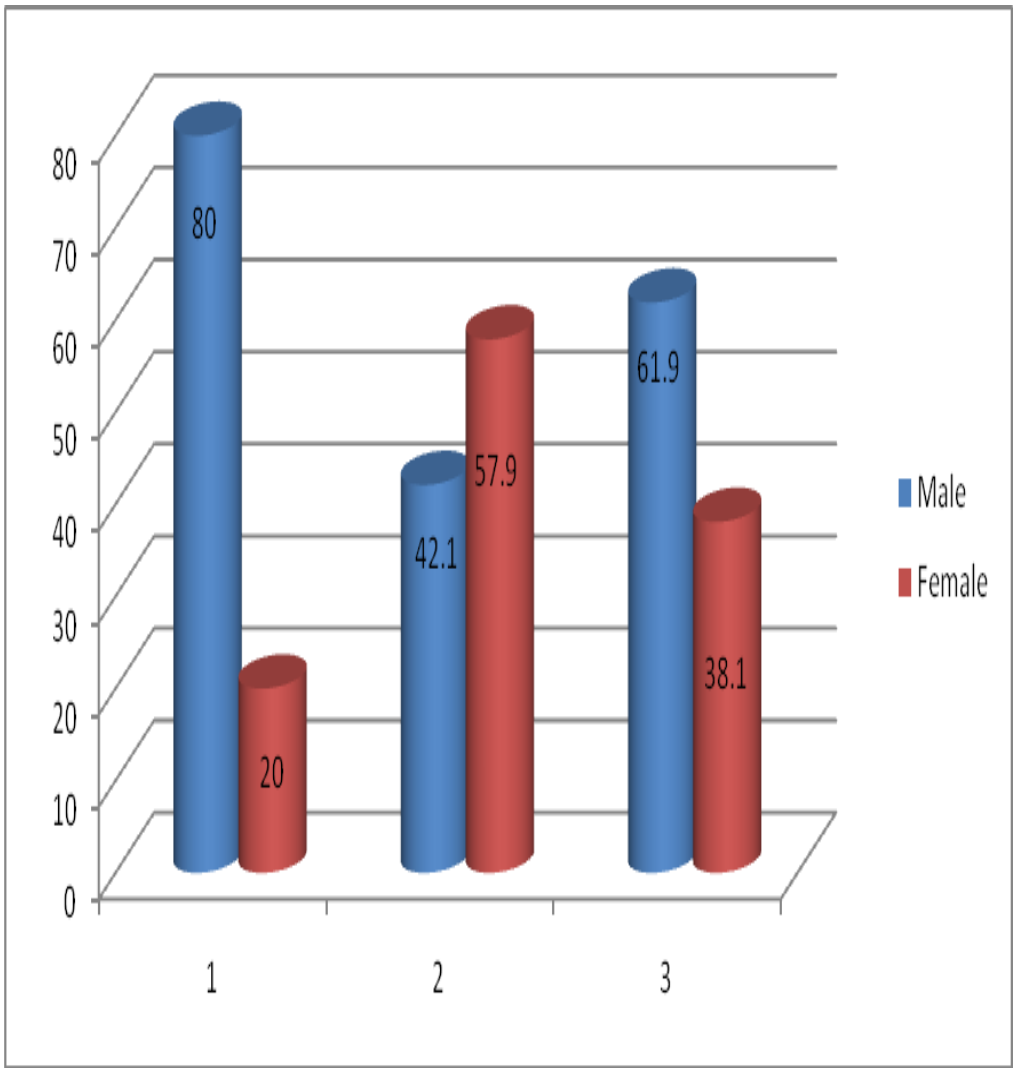
3= artificial feeding group

Table (10): Comparison of gender distribution between groups.

Gender	**Groups**			**X^2 test**	
	Control group ***(N= 15)***	**Fully breast feeding and STS care** ***(N=19)***	**Artificial feeding and STS care** ***(N=21)***		
	No %	***No %***	***No %***		
-Male	12 80.0	8 42.1	13 61.9	5.07	>0.05
-Female	3 20.0	11 57.9	8 38.1		
Total	**15 100.0**	**19 100.0**	**21 100.0**		

The table show significant statistically difference (<0.05) in gender in the groups under study

Figure (4): Comparing gender distribution in the Studied Groups.



1=control group

2= fully breastfeeding group

3= artificial feeding group

Table (11): Comparison of epidemiological data by frequency distribution in groups under study.

Studied variables	Groups			X^2 test	p-Value	p- value between groups compared with X^2 test
	Control group *(N=15)*	Exclusive breast feeding and STS care *(N=19)*	Artificial feeding and STS care *(N=21)*			
	No %	*No %*	*No %*			
Mother age						
-≤ 19 years	**2 13.3**	**0 0.0**	**3 14.3**	**12.07**	**> 0.05**	**P1= > 0.05**
- 20 – 29 yrs	**8 53.3**	**14 73.7**	**17 81.0**			**P2= > 0.05**
- 30 – 39 yrs	**5 33.3**	**3 15.8**	**0 0.0**			**P3= > 0.05**
- ≥ 40 yrs	**0 0.0**	**2 10.5**	**1 4.8**			
Residence						**P1=> 0.05**
-Urban	**4 26.7**	**7 36.8**	**15 71.4**	**8.3**	**<0.05***	**P2=<0.01****
- Rural	**11 73.3**	**12 63.2**	**6 28.6**			**P3= < 0.05***
Consanguinity						
- First degree	**4 26.7**	**7 36.8**	**4 19.0**	**6.31**	**>0.05**	**P1=> 0.05**
-Second degree	**1 6.7**	**3 15.8**	**0 0.0**			**P2=> 0.05**
- Third degree	**10 66.7**	**9 47.4**	**17 81.0**			**P3=> 0.05**
Family history of CD			**21 100.0**	**2.76**	**>0.05**	**P1=> 0.05**
- No	**13 86.7**	**17 89.5**	**0 0.0**			**P2=> 0.05**
- Yes	**2 13.3**	**2 10.5**				**P3=> 0.05**
Total	**15 100.0**	**19 100.0**	**21 100.0**			

P1= between control and exclusive breast feeding with STS care group
P2= between control and artificial feeding with STS care group
P3= between exclusive breast feeding with STS care group and artificial feeding with STS care group

The table show epidemiological data of the groups under study.

There is no significant statistically difference except in residence where there is significant statistically difference (<0.05) in the groups under study.

Table (١٢): Comparing Mean and S.D. of age of diagnosis of infants in the studied groups

Studied variable	**Control** *(N=15)* **Mean ± SD**	**Exclusive breast feeding and STS care** *(N=19)* **Mean ± SD**	**Artificial feeding and STS care** *(N=21)* **Mean ± SD**	**ANOVA Test:**	**P-value**
-Number of brothers	1.6 ± 1.45	1.53 ± 1.07	1.09 ± 0.9	1.84	> 0.05
-Age of diagnosis in weeks	7.67 ± 5.97	10.68 ± 8.2	9.28 ± 5.5	1.68	> 0.05

The table shows no significant statistically difference in mean and SD of age of diagnosis in the groups under study.

Table (١٣): comparing prenatal health and medical status of mothers of infants in studied groups using frequency distribution and chi-square

Studied variables	Groups			X^2 test	p-Value	p-value between groups compared with X^2 test
	Control group (N=15)	Exclusive breast feeding and STS care(N=19)	Artificial feeding and STS care (N=21)			
	No %	No %	No %			
Site of diagnosis						
-Private clinic	11 73.3	11 57.9	12 57.1	1.16	>0.05	P1=>0.05
-Hospital	4 26.7	8 42.1	9 42.9			P2=>0.05
						P3= > 0.05
Other congenital disease				0.81	>0.05	P1=>0.05
-No	12 80.0	16 84.2	19 90.5			P2=>0.05
-Yes	3 20.0	3 15.8	2 9.5			P3=>0.05
Mother infection before delivery				1.09	>0.05	P1=>0.05
-No	10 66.7	15 78.9	17 81.0			P2=>0.05
-Yes	5 33.3	4 21.1	4 19.0			P3=>0.05
Mother drugs before delivery				2.2	>0.05	P1=>0.05
-No	14 93.3	17 89.5	21 100.0			P2=>0.05
-Yes	1 6.7	2 10.5	0 0.0			P3=>0.05
Mother bleeding before delivery				1.33	>0.05	P1=>0.05
-No	14 93.3	18 94.7	21 100.0			P2=>0.05
-Yes	1 6.7	1 5.3	0 0.0			P3=>0.05
Mother wt gain before delivery				1.94	>0.05	P1=>0.05
-No	12 80.0	18 94.7	19 90.5			P2=>0.05
-Yes	3 20.0	1 5.3	2 9.5			P3=>0.05
Mother anemia before delivery				0.53	>0.05	P1=>0.05
-No	7 46.7	9 47.4	12 57.1			P2=>0.05
-Yes	8 53.3	10 52.6	9 42.9			P3=>0.05
Mother others before delivery				0.79	>0.05	P1=>0.05
-No	12 80.0	14 73.7	14 66.7			P2=>0.05
-Yes	3 20.0	5 26.3	7 33.3			P3=>0.05
Total	15 100.0	19 100.0	21 100.0			

P1= between control and exclusive breast feeding with STS care group
P2= between control and artificial feeding with STS care group
P3= between exclusive breast feeding with STS care group and artificial feeding with STS care group

The table show prenatal health and medical status of mothers of infants in studied groups. There is no significant statistically difference in the groups under study.

Table (١٤): Comparing perinatal health status of infants in studied groups by frequency distribution

Studied variables	Groups			X^2 test	p-Value	p- value between groups compared with X^2 test
	Control group *(N= 15)*	Exclusive breast feeding and STS care *(N=19)*	Artificial feeding and STS care *(N=21)*			
	No %	*No %*	*No %*			
Type of labor						**P1=> 0.05**
-Vaginal	**8 53.3**	**10 52.6**	**7 33.3**	**2.01**	**>0.05**	**P2=> 0.05**
-CS	**7 46.7**	**9 47.4**	**14 66.7**			**P3=> 0.05**
Peripartum problems						**P1=> 0.05**
-No	**14 93.3**	**16 84.2**	**19 90.5**	**0.78**	**>0.05**	**P2=> 0.05**
-Yes	**1 6.7**	**3 15.8**	**2 9.5**			**P3=> 0.05**
Postnatal cyanosis						**P1=> 0.05**
-No	**11 73.3**	**15 78.9**	**14 66.7**	**0.76**	**>0.05**	**P2=> 0.05**
-Yes	**4 26.7**	**4 21.1**	**7 33.3**			**P3=> 0.05**
Postnatal respiratory distress				**0.19**	**>0.05**	**P1=> 0.05** **P2=> 0.05**
-No	**11 73.3**	**13 68.4**	**14 66.7**			**P3=> 0.05**
-Yes	**4 26.7**	**6 31.6**	**7 33.3**			
Postnatal jaundice						**P1=> 0.05**
-No	**13 86.7**	**14 73.7**	**15 71.4**	**1.24**	**>**	**P2=> 0.05**
-Yes	**2 13.3**	**5 26.3**	**6 28.6**		**0.05**	**P3=> 0.05**
Postnatal difficulty in breast feeding						**P1=> 0.05**
-No	**13 86.7**	**15 78.9**	**17 81.0**	**0.35**	**>**	**P2=> 0.05**
-Yes	**2 13.3**	**4 21.1**	**4 19.0**		**0.05**	**P3=> 0.05**
Total	**15 100.0**	**19 100.0**	**21 100.0**			

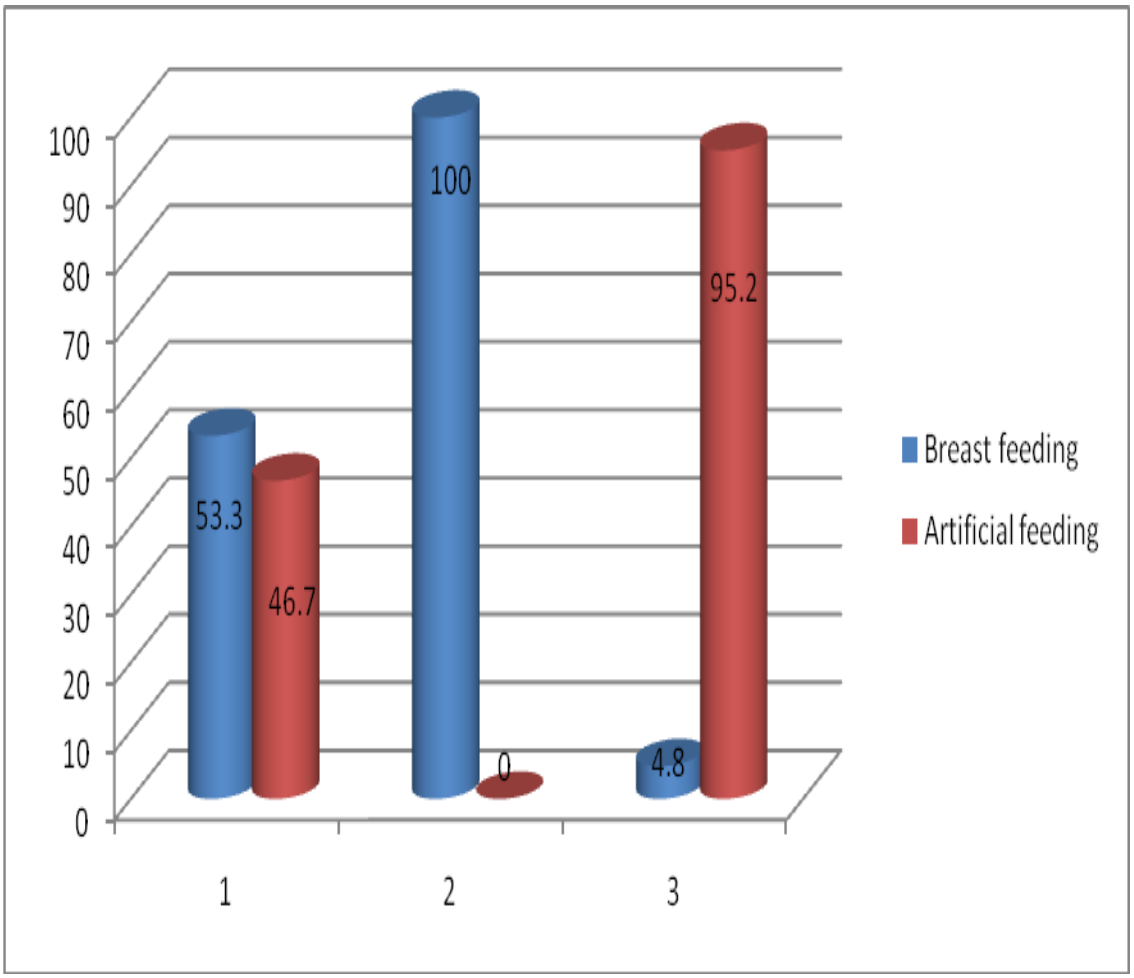
The table shows no significant statistically difference in perinatal health status of infants in studied group

Table (١٥): Comparing mode of feeding and feeding problem in groups under study by frequency distribution

Studied variables	Groups			X^2 test	p-Value	p- value between groups compared with X^2 test
	Control group (N=15) No %	Exclusive breast feeding and STS care (N=19) No %	Artificial feeding and STS care (N=21) No %			
Mode of baby feeding						
-Breast feeding	8 53.3	19 100.0	1 4.8		<0.01**	P1=<0.01**
-Artificial feeding	7 46.7	0 0.0	20 95.2	39.6		P2=<0.01**
						P3=<0.01**
Feeding problem colic						P1=> 0.05
-No	12 80.0	12 63.2	18 85.7	2.96	> 0.05	P2=> 0.05
-Yes	3 20.0	7 36.8	3 14.3			P3=> 0.05
Feeding problem vomiting				3.92	> 0.05	P1=> 0.05 P2=> 0.05
-No	9 60.0	14 73.7	9 42.9			P3=> 0.05
-Yes	6 40.0	5 26.3	12 57.1			
Other Feeding problems				0.78	> 0.05	P1=> 0.05 P2=> 0.05
-No	12 80.0	16 84.2	19 90.5			P3=> 0.05
-Yes	3 20.0	3 15.8	2 9.5			
Appetite						
-Good	10 66.7	11 57.9	8 38.1	8.43	> 0.05	P1=> 0.05
-Moderate	5 33.3	7 36.8	7 33.3			P2=> 0.05
- Poor	0 0.0	1 5.3	6 28.6			P3=> 0.05
Total	15100.0	19 100.0	21 100.0			

The table show mode of feeding and feeding problem in groups under study. There were no significant statistically difference except in the mode of feeding where there was highly significant statistically difference and in vomiting where there was significant statistically difference in the groups under study.

Figure (5): Mode of baby feeding



1=control group

2= fully breastfeeding group

3= artificial feeding group

Table (16): Comparing health problems by frequency distribution in the infants in the study groups.

Studied variables	Groups			X^2 test	p-Value	p- value between groups compared with X^2 test
	Control group *(N= 15)*	Exclusive breast feeding and STS care *(N=19)*	Artificial feeding and STS care *(N=21)*			
	No %	*No %*	*No %*			
Weight loss						
-Recent	**4 26.7**	**8 42.1**	**5 23.8**	**4.36**	**>0.05**	**P1> 0.05**
-Continuous	**10 66.7**	**11 57.9**	**16 76.2**			**P2=> 0.05**
-No	**1 6.7**	**0 0.0**	**0 0.0**			**P3=> 0.05**
Hospital admission						**P1=> 0.05**
-No	**1 6.7**	**3 15.8**	**3 14.3**	**0.7**	**>0.05**	**P2=> 0.05**
-Yes	**14 93.3**	**16 84.2**	**18 85.7**			**P3=> 0.05**
Gastroenteritis						**P1=> 0.05**
-No	**13 86.7**	**17 89.5**	**19 90.5**	**0.13**	**>0.05**	**P2=> 0.05**
-Yes	**2 13.3**	**2 10.5**	**2 9.5**			**P3=> 0.05**
Bronchitis						**P1=> 0.05**
-No	**5 33.3**	**11 57.9**	**7 33.3**	**3.08**	**>0.05**	**P2=> 0.05**
-Yes	**10 66.7**	**8 42.1**	**14 66.7**			**P3=> 0.05**
Pneumonia						**P1=> 0.05**
-No	**5 33.3**	**8 42.1**	**10 47.6**	**0.73**	**>0.05**	**P2=> 0.05**
-Yes	**10 66.7**	**11 57.9**	**11 52.4**			**P3=> 0.05**
Common cold						**P1=> 0.05**
-No	**13 86.7**	**15 78.9**	**20 95.2**	**2.39**	**>0.05**	**P2=> 0.05**
-Yes	**2 13.3**	**4 21.1**	**1 4.8**			**P3=> 0.05**
Pharyngitis						**P1=> 0.05**
-No	**13 86.7**	**16 84.2**	**20 95.2**	**1.37**	**>0.05**	**P2=> 0.05**
-Yes	**2 13.3**	**3 15.8**	**1 4.8**			**P3=> 0.05**
Otitis media						**P1=> 0.05**
-No	**15 100.0**	**18 94.7**	**21 100.0**	**1.93**	**>0.05**	**P2= ----**
-Yes	**0 0.0**	**1 5.3**	**0 0.0**			**P3=> 0.05**
Other causes of hospital admission and morbidity history	**11 73.3**	**16 84.2**	**17 80.9**	**0.64**	**>0.05**	**P1=> 0.05** **P2=> 0.05**
-No	**4 26.7**	**3 15.8**	**4 19.1**			**P3=> 0.05**
-Yes						
Total	**15 100.0**	**19 100.0**	**21 100.0**			

The table show no significant statistically difference in comparing health problems in the groups under study.

Table (17): Comparing vaccination status by frequency distribution in infants under study

Studied variables	Groups			X^2 test	p-Value	
	Control group *(N= 15)*	Exclusive breast feeding and STS care *(N=19)*	Artificial feeding and STS care *(N=21)*			
	No %	No %	*No %*			
Zero age of vaccination intake						
-No	1 6.7	0 0.0	0 0.0	2.72	> 0.05	P1= > 0.05
-Yes	14 93.3	19 00.0	21 100.0			P2=> 0.05
						P3=---
Two months of vaccination intake						P1= > 0.05
-No	3 20.0	3 15.8	2 9.5	0.81	> 0.05	P2= > 0.05
-Yes	12 80.0	16 84.2	19 90.5			P3= > 0.05
Four months of vaccination intake						P1=>0.05
-No	4 26.7	7 36.8	8 38.1	0.57	> 0.05	P2=>0.05
-Yes	11 73.3	12 63.2	13 61.9			P3=>0.05
Six months of vaccination intake						P1=<0.01**
-No	6 40.0	17 89.5	17 81.0	11.5	<0.01**	P2= < 0.05*
-Yes	9 60.0	2 10.5	4 19.0			P3= > 0.05
Nine months of vaccination intake						P1=<0.01**
-No	8 53.3	18 94.7	21 100.0	17.34	<0.01**	P2=<0.01**
-Yes	7 46.7	1 5.3	0 0.0			P3= > 0.05
One year of vaccination intake						P1= > 0.05
-No	13 86.7	18 94.7	21 100.0	3.02	> 0.05	P2= > 0.05
-Yes	2 13.3	1 5.3	0 0.0			P3= > 0.05
1 and ½ year of vaccination intake						P1= > 0.05
-No	14 93.3	19 100.0	21 100.0	2.72	> 0.05	P2= > 0.05
-Yes	1 6.7	0 0.0	0 0.0			P3= ----
Fever side effects of vaccination						P1= > 0.05
-No	11 73.3	13 68.4	18 85.7	1.76	> 0.05	P2= > 0.05
-Yes	4 26.7	6 31.6	3 14.3			P3= > 0.05
Medication history						P1=<0.01**
-No	6 40.0	16 84.2	19 90.5	13.18	<0.01**	P2=<0.01**
-Yes	9 60.0	3 15.8	2 9.5			P3= > 0.05
History of food allergy						P1= > 0.05
-No	13 86.7	17 89.5	20 95.2	0.85	> 0.05	P2= > 0.05
-Yes	2 13.3	2 10.5	1 4.8			P3= > 0.05
Total	15 100.0	19 100.0	21 100.0			

The table shows vaccination status of infants of the group under study. There were no significant statistically difference in all groups except at 6^{th}, 9^{th} and at medication history where there were highly significant statistically difference (<0.01) in the groups under study.
We notice that significant difference due to increased age of control group

		Group I		
		Control	Skin to Skin Care group	Total
Type of CHD	VSD	5	13	18
		33.3%	32.5%	32.7%
	ASD	1	0	1
		6.7%	.0%	1.8%
	PDA	0	4	4
		.0%	10.0%	7.3%
	PS	0	2	2
		.0%	5.0%	3.6%
	TGA	2	1	3
		13.3%	2.5%	5.5%
	DCM	1	2	3
		6.7%	5.0%	5.5%
	A-V canal	1	2	3
		6.7%	5.0%	5.5%
	TAPVR	0	1	1
		.0%	2.5%	1.8%
	ASD, TGA and Coarctition of Aorta	1	0	1
		6.7%	.0%	1.8%
	VSD, PDA, PA and DORV	1	0	1
		6.7%	.0%	1.8%
	VSD and TGA	1	0	1
		6.7%	.0%	1.8%
	VSD and PDA	1	0	1
		6.7%	.0%	1.8%
	VSD, ASD and PDA	1	0	1
		6.7%	.0%	1.8%
	VSD and ASD	0	6	6
		.0%	15.0%	10.9%
	PS and A-V canal	0	2	2
		.0%	5.0%	3.6%
	VSD, ASD and TGA	0	3	3
		.0%	7.5%	5.5%
	DCM and TAPVR	0	1	1
		.0%	2.5%	1.8%
	VSD, ASD, PS and TGA	0	1	1
		.0%	2.5%	1.8%
	VSD, ASD and Truncus Arterusus	0	1	1
		.0%	2.5%	1.8%
	VSD, ASD, PDA, Coarctition of Aorta andAS	0	1	1
		.0%	2.5%	1.8%
Total		15	40	55
		100.0%	100.0%	100.0%

Table (18): description of type of congenital heart defect in the infant under study by frequency distribution in theses versus control

Figure (6): description of type of congenital heart defect in the infant under study by frequency distribution in theses versus control

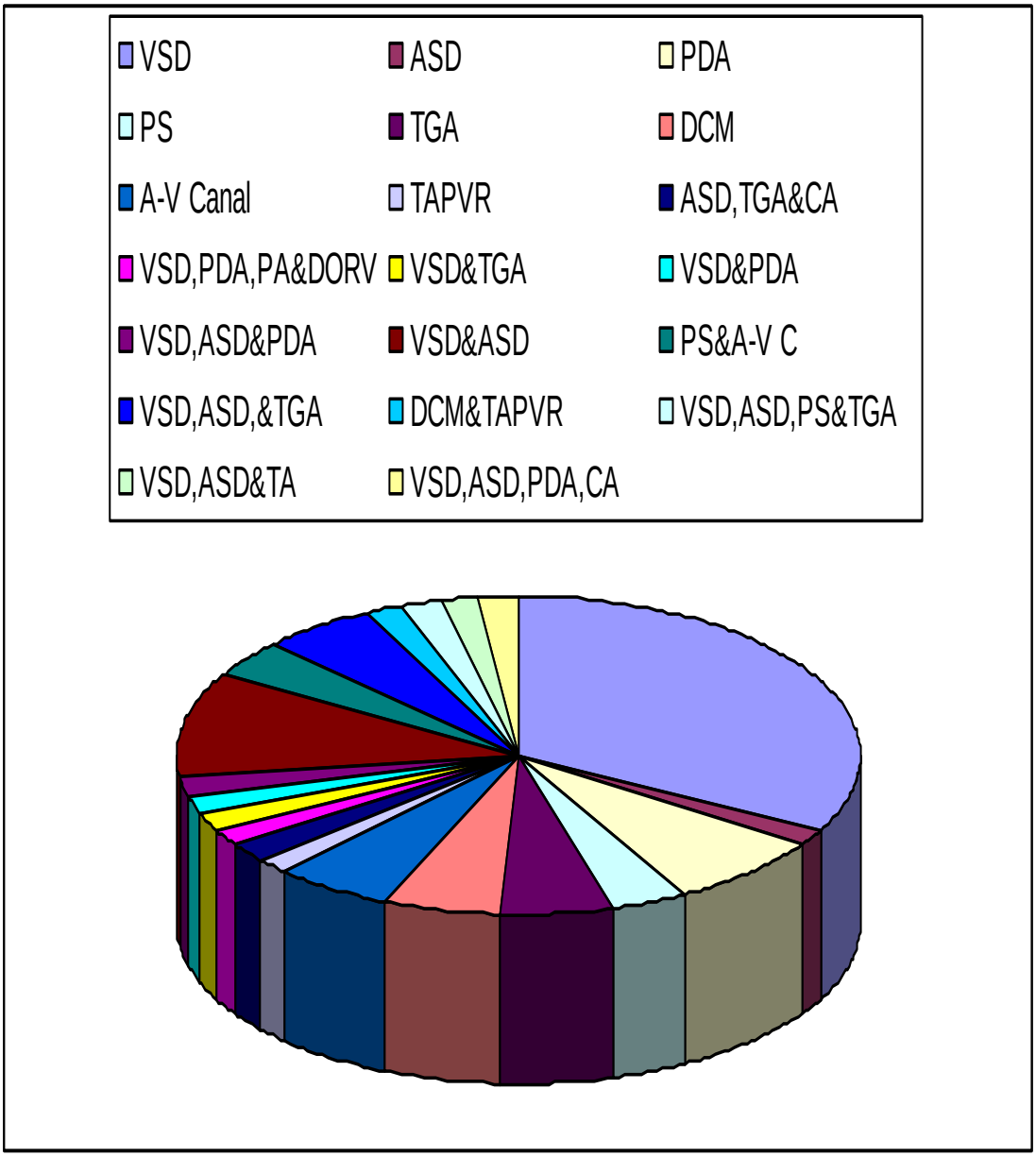


Table (19): comparing anxiety state of mothers of infants according to mode of feeding of the group under study by frequency distribution and chi-square

Studied variables	Groups			X^2 test	p-Value	p- value between groups compared with X^2 test
	Control group *(N= 15)*	Exclusive breast feeding and STS care *(N=19)*	Artificial feeding and STS care *(N=21)*			
	No %	*No %*	*No %*			
Anxiety state of the mother						
-Week anxiety -	3 20.0	0 0.0	1 4.8			P1= < 0.05*
-Less than anxiety	0 0.0	1 5.3	0 0.0		<	
-Moderate anxiety	2 13.3	0 0.0	0 0.0	18.8	0.05*	P2= > 0.05
-More than moderate anxiety	3 20.0	13 68.4	11 52.4			P3= > 0.05
- Severe anxiety	5 33.4	3 15.8	8 38.1			
-Very severe anxiety	2 13.3	2 10.5	1 4.8			
Total	**15 100.0**	**19 100.0**	**21 100.0**			

P1= between control and exclusive breastfeeding with SSC care group
P2= between control and artificial feeding with SSC care group
P3= between exclusive breastfeeding with SSC care group and artificial feeding with SSC care group

The table shows significant statistically difference (<0.05) in anxiety state of mothers of infants according to mode of feeding of the group under study.

Table (20): comparison of mean and SD of anxiety score before and after exposure to SSC among the exclusively breastfed babies

Exclusive breastfeeding and SSC care	**STAI**		Paired t-test:	**P-value**
	Before group **Mean ± SD**	**After group Mean ± SD**		
	46.6 ± 9.93	23.9 ± 3.68	10.6	< 0.01**

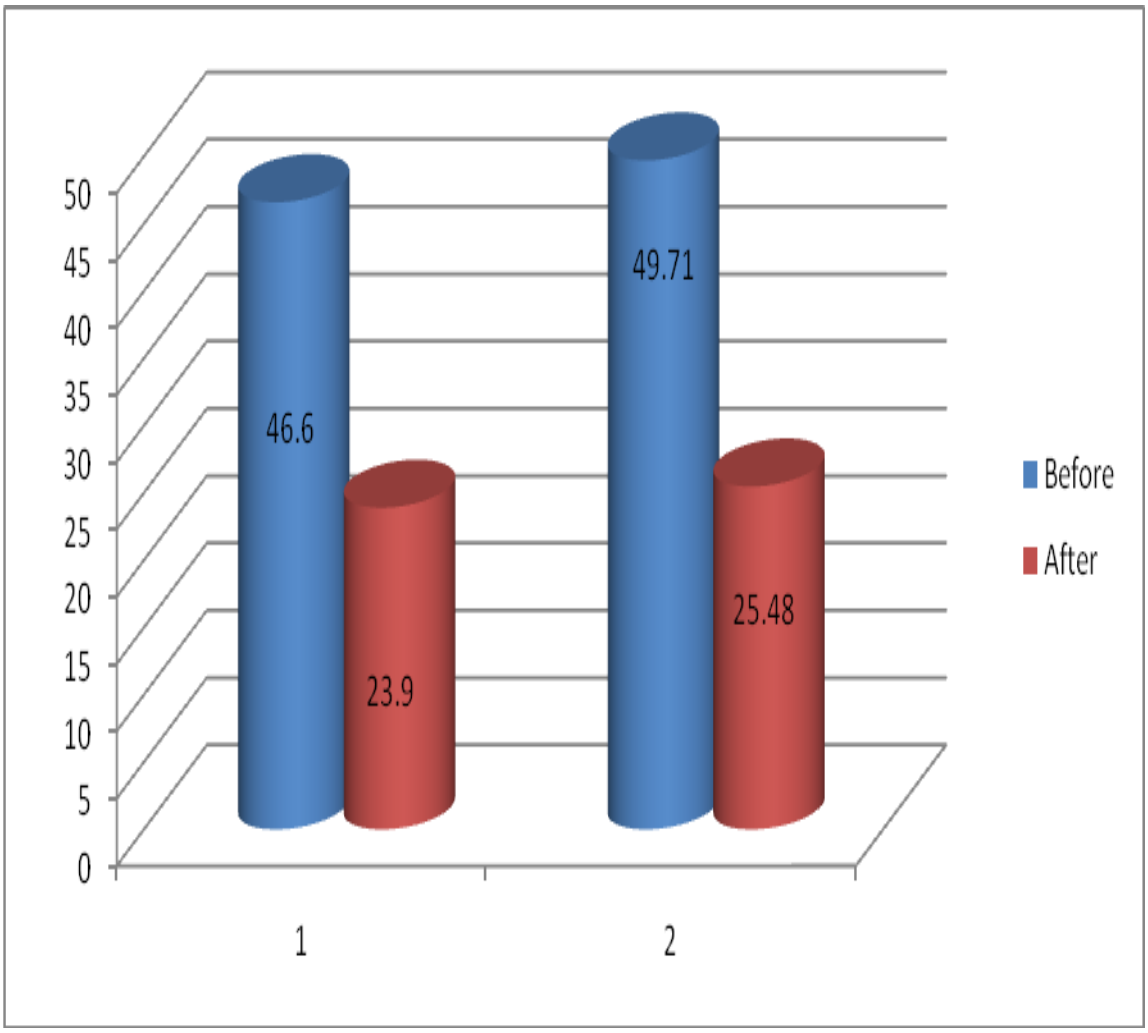
The table shows highly statistically difference in mean and SD of anxiety score before and after exposure to SSC among the exclusively breastfed babies.

Table (21): comparison of mean and SD of anxiety score before and after exposure to SSC among the artificially fed babies

Artificial feeding and SSC care	**STAI**		Paired t-test	**P-value**
	Before group **Mean ± SD**	**After group Mean ± SD**		
	49.71 ± 9.25	25.48 ± 4.52	12.68	< 0.01**

.The table shows highly statistically difference in mean and SD of anxiety score before and after exposure to SSC among the artificially fed babies.

Figure (7): Exclusive breast feeding and artificial breast feeding in STAI



1=fully breastfeeding group
2= artificial feeding group

Table (22): Frequency distribution of hospital admission of exclusive breastfeeding babies in the study group

Studied variables	Hospital admission in exclusive breast feeding group				X^2 test	p-Value
	No *(N=3)*		Yes *(N=16)*			
	No	*%*	*No*	*%*		
Anxiety state of the mother						
-Weak anxiety	0	0.0	0	0.0		
-Less than moderate anxiety	1	33.3	0	0.0	6.27	<0.01**
- Moderate anxiety	0	0.0	11	68.8		
- More than moderate anxiety	2	66.7	0	0.0		
- Severe anxiety	0	0.0	3	18.8		
- Very severe anxiety	0	0.0	2	12.5		
Total	**3**	**100.0**	**16**	**100.0**		

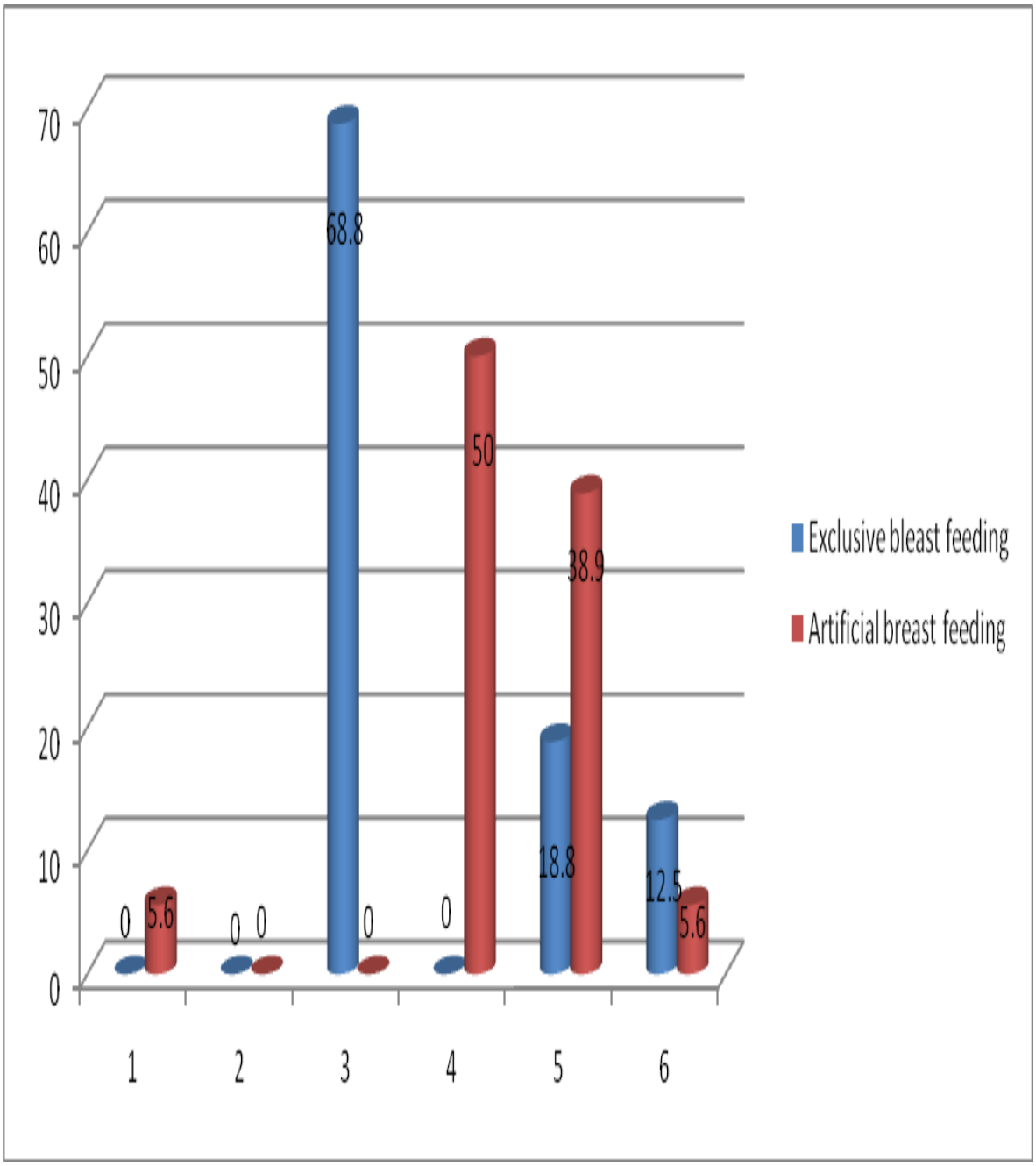
The table shows highly statically difference (<0.01) in hospital admission of exclusive breastfeeding babies in the study group

Table (23): Frequency distribution of hospital admission of artificial feeding babies in the study group

Studied variables	Hospital admission in artificial feeding group				X^2 test	p-Value
	No *(N=3)*		Yes *(N=18)*			
	No	*%*	*No*	*%*		
Anxiety state of the mother						
-Weak anxiety	0	0.0	1	5.6		
-Less than moderate anxiety	0	0.0	0	0.0		
- Moderate anxiety	0	0.0	0	0.0	0.49	> 0.05
- More than moderate anxiety	2	66.7	9	50.0		
- Severe anxiety	1	33.3	7	38.9		
- Very severe anxiety	0	0.0	1	5.6		
Total	**3**	**100.0**	**18**	**100.0**		

The table shows no significant statically difference in hospital admission of artificial feeding babies in the study group

Figure (8): Hospital admission in exclusive breast feeding and artificial feeding



1=Weak anxiety
2=Less than moderate anxiety
3= Moderate anxiety
4=More than moderate anxiety
5= Severe anxiety
6= Very severe anxiety

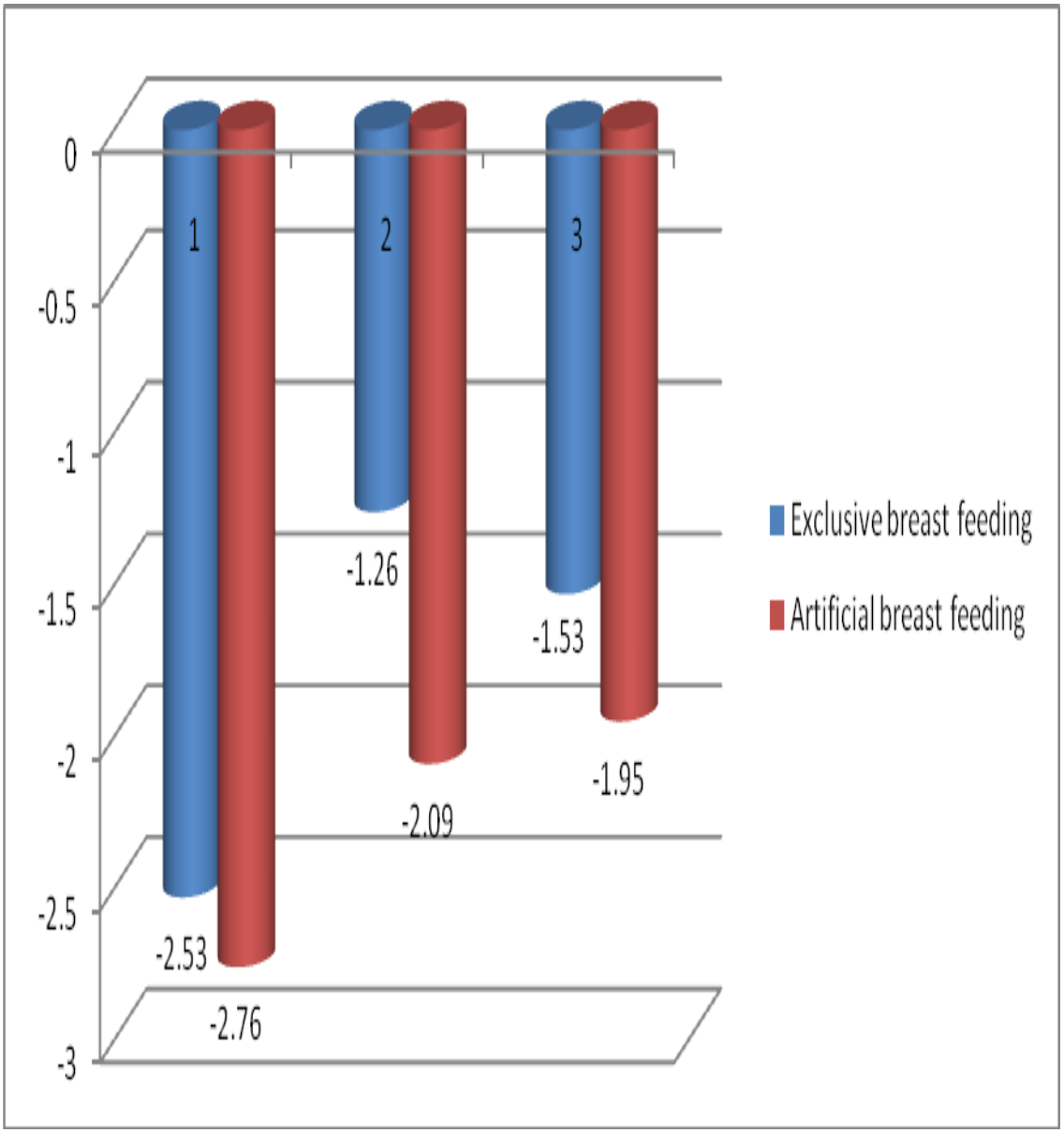
Table (24): comparing mean and SD of anthropometric data (Wt, Length& HC) by Z-score in the study group.

Studied variables	Control group *(N=15)* Mean ± SD	Exclusive breast feeding and STS care group *(N=19)* Mean ± SD	Artificial feeding and STS care group *(N=21)* Mean ± SD	ANOVA test	P-Value	LSD post Hoc p-value
Pregnancy duration in months	8.97 ± 0.13	9 ± 0.01	8.93 ± 0.24	**0.96**	> 0.05	
Frequency of feeding interval in hrs	3.13 ± 1.17	2.53 ± 0.77	2.62 ± 0.47	**2.63**	> 0.05	
Weight Z scores	- 2.07 ± 0.88	- 2.53 ± 0.9	- 2.76 ± 0.44	**3.76**	< 0.05*	P1= > 0.05 P2= < 0.01** P3= > 0.05
Length in Z scores	- 1.27 ± 1.58	-1.26 ± 1.41	- 2.09 ± 1.22	**4.66 ***	> 0.05	
HC in Z scores	- 1.67 ± 1.29	- 1.53 ± 1.02	- 1.95 ± 1.2	**1.92 ***	> 0.05	

*** Kruskal Wallis test**

The table shows anthropometric data (Wt, Length& HC) by Z-score. There were none statistically significant difference except in weight Z-score (<0.05) among all groups in the study groups.

Figure (9): comparing mean and SD of anthropometric data (Wt, Length& HC) by Z-score in the study group.



1= Weight

2= Length

3= Head circumference

Table (25): Comparing mean and SD of vital signs in the study groups

Studied variables	**Control group** *(N=15)* **Mean ± SD**	**Exclusive breast feeding and STS care group** *(N=19)* **Mean ± SD**	**Artificial feeding and STS care group** *(N=21)* **Mean ± SD**	**ANOVA test**	**p-Value**	**LSD post Hoc p-value**
Heart rate	141.8 ± 29.6	136.7 ± 16.99	141.09 ± 12.11	**0.35**	> 0.05	
Respiratory rate	47.6 ± 12.46	51.95 ± 11.32	49.7 ± 7.79	**0.73**	> 0.05	
Oxygen saturation	83.07 ± 14.65	93.5 ± 8.2	90.24 ± 12.07	**3.39**	< 0.05*	P1= < 0.05* P2= > 0.05 P3= > 0.05

- **Kruskal Wallis test**

The table shows none statistically significant difference in mean and SD of vital signs except in oxygen saturation which shows significant difference (<0.05) among all groups in the study groups.

Table (26): Comparing mean and SD of HR before, during 6 min. & 5 min. after feeding the babies in the group under study.

Studied variables	Control group *(N=15)* Mean ± SD	Exclusive breast feeding and STS care group *(N=19)* Mean ± SD	Artificial feeding and STS care group *(N=21)* Mean ± SD	ANOVA test	P-Value
HR before SSC	141.07 ± 30.35	140 ± 15.19	141.09 ± 12.11	**0.02**	> 0.05
HR 1st m during breast feeding	135.1 ± 33.03	141.7 ± 14.9	145.1 ± 13.7	**0.88**	> 0.05
HR 2nd m during breast feeding	137.7 ± 32.7	144.05 ± 15.42	146.45 ± 11.17	**0.72**	> 0.05
HR 3rd m during breast feeding	138.6 ± 33.04	144.6 ± 11.94	147.45 ± 12.2	**0.79**	> 0.05
HR 4th m during breast feeding	136.4 ± 32.49	143.8 ± 12.51	147.7 ± 12.96	**1.26**	> 0.05
HR 5th m during breast feeding	131.44 ± 30.24	143.6 ± 12.37	147.2 ± 12.43	**2.56**	> 0.05
HR 6th m during breast feeding	151.25 ± 10.63	141.7 ± 12.75	152 ± 12.19	**1.46**	> 0.05
HR 5 m after breast feeding	132.9 ± 31.08	140.26 ± 13.3	140.9 ± 10.49	**0.79**	> 0.05
HR saturation after breast feeding	0 ± 0	141.84 ± 12.88	143.48 ± 10.32	**0.19 ***	> 0.05

*** t- test**

The table shows mean and SD of HR before, during 6 min. & 5 min. after feeding the babies in the group under study. There were no significant statistically difference.

Figure (10): Comparing mean and SD of HR during 10min. feeding the babies in the group under study

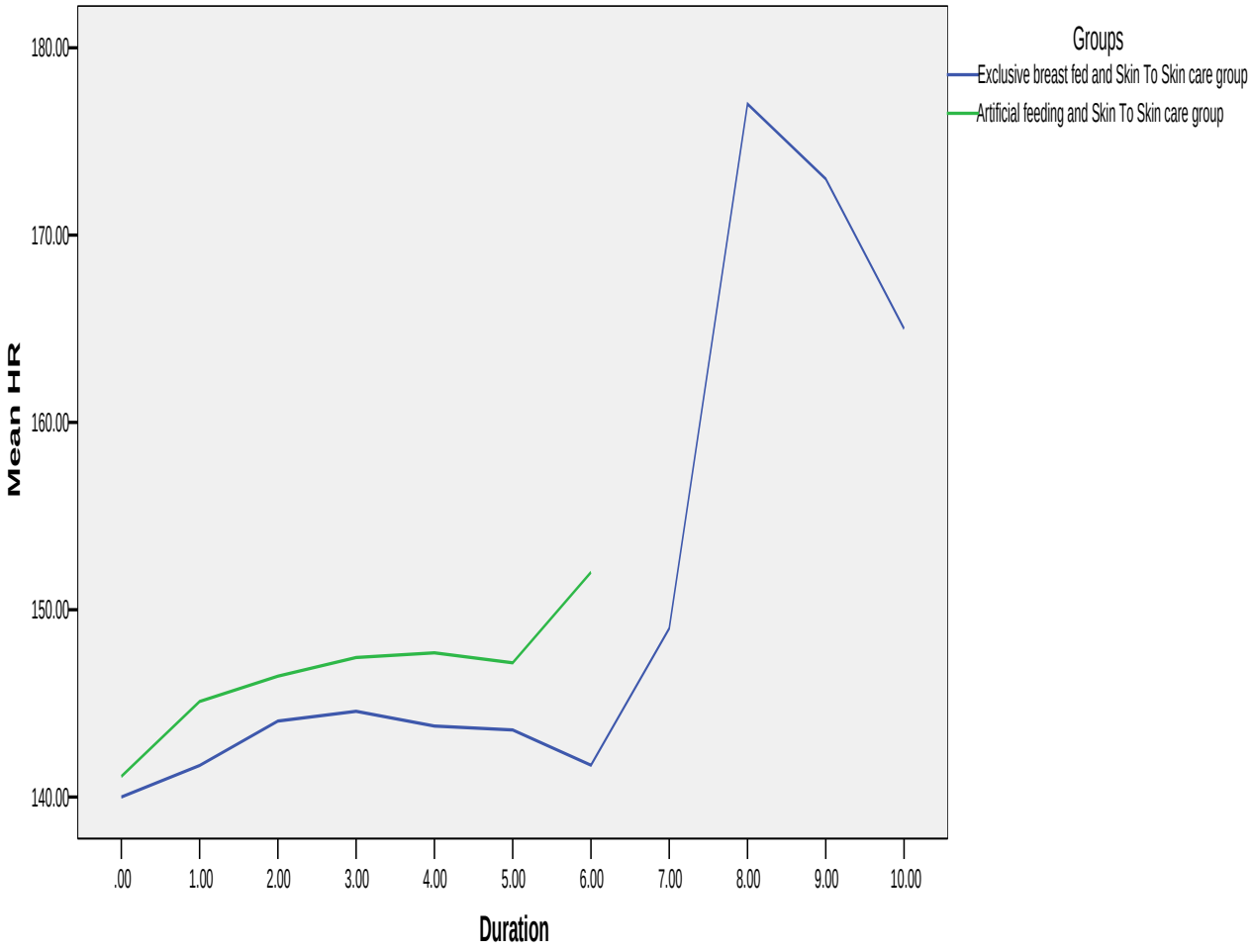


Table (27): comparing mean and SD of SO2before, during 6 min. & 5 min. after feeding the babies in the group under study.

Studied variables	Control group *(N=15)* Mean ± SD	Exclusive breast feeding and STS care group *(N=19)* Mean ± SD	Artificial feeding and STS care group *(N=21)* Mean ± SD	ANOVA test	p-Value
SO2before SSC	84.5 ± 14.07	92.89 ± 8.28	89.9 ± 13.22	**1.99**	> 0.05
SO21st m during breast feeding	86.9 ± 11.36	93.63 ± 8.52	89.7 ± 13.08	**1.31**	> 0.05
SO22nd m during breast feeding	87.7 ± 11.11	94.58 ± 7.79	89.25 ± 12.76	**1.79**	> 0.05
SO23rd m during breast feeding	88.9 ± 12.9	95.2 ± 7.71	89.1 ± 12.37	**1.86**	> 0.05
SO24th m during breast feeding	89.1 ± 12.03	95.53 ± 7.51	89.2 ± 12.1	**2.12**	> 0.05
SO25th m during breast feeding	91.11 ± 11.61	94.9 ± 7.19	89.7 ± 12.5	**1.22**	> 0.05
SO26th m during breast feeding	97.75 ± 2.87	95.2 ± 6.2	85 ± 13.82	**3.09**	> 0.05
SO25 m after breast feeding	89 ± 11.78	94.8 ± 7.97	90.43 ± 12.49	**1.23**	> 0.05
SO2saturation after breast feeding	0 ± 0	94.16 ± 7.17	90.14 ± 12.53	**1.5 ***	> 0.05

*** t- test**

The table shows mean and SD of SO2before, during 6 min. & 5 min. after feeding the babies in the group under study.

There were no significant statistically difference.

Figure (11): Comparing mean and SD of SO2during 10min. feeding the babies in the group under study.

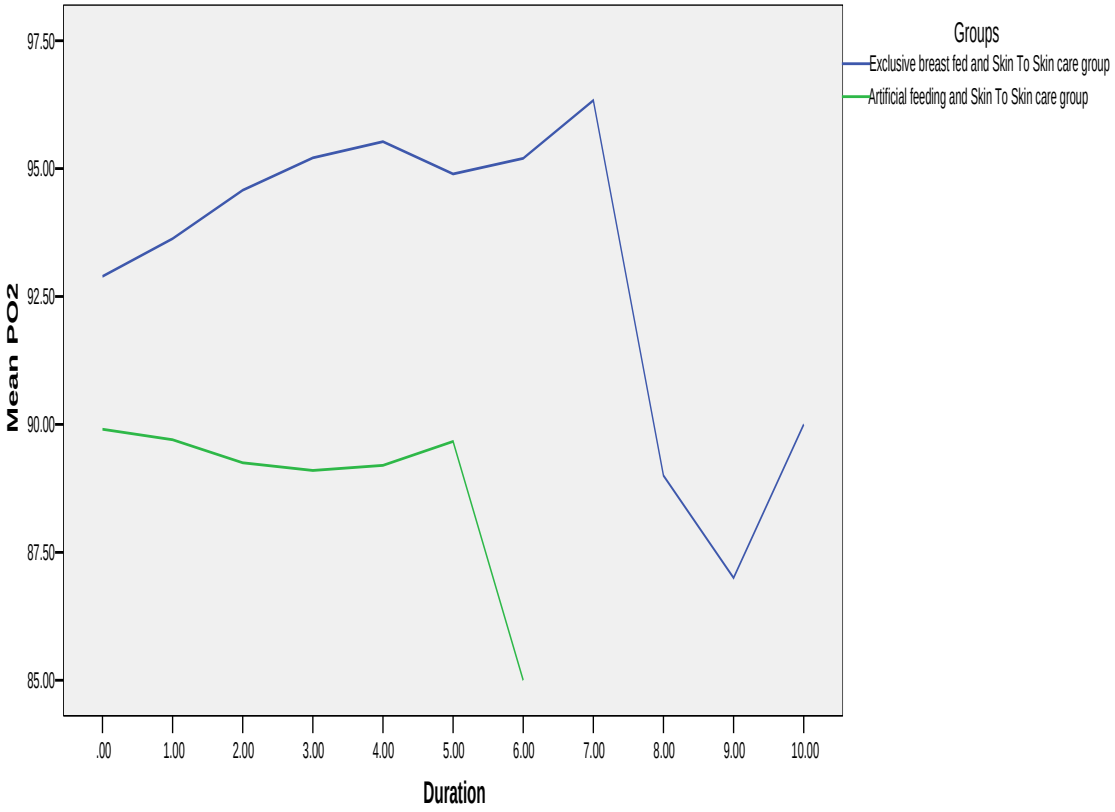


Figure (12): comparing mean and SD of SO2 before, before, 5 min. and after SSC in the group under study.

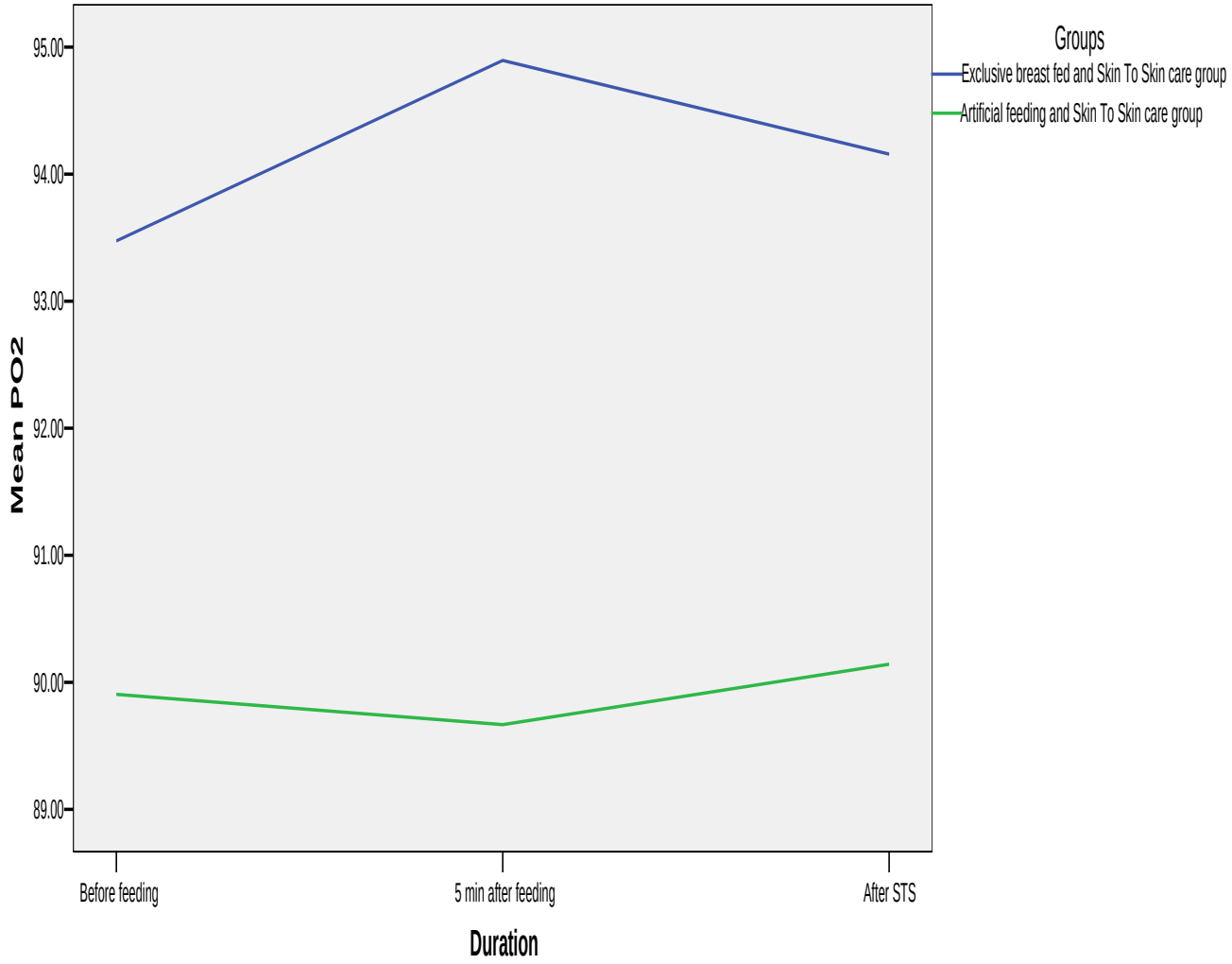
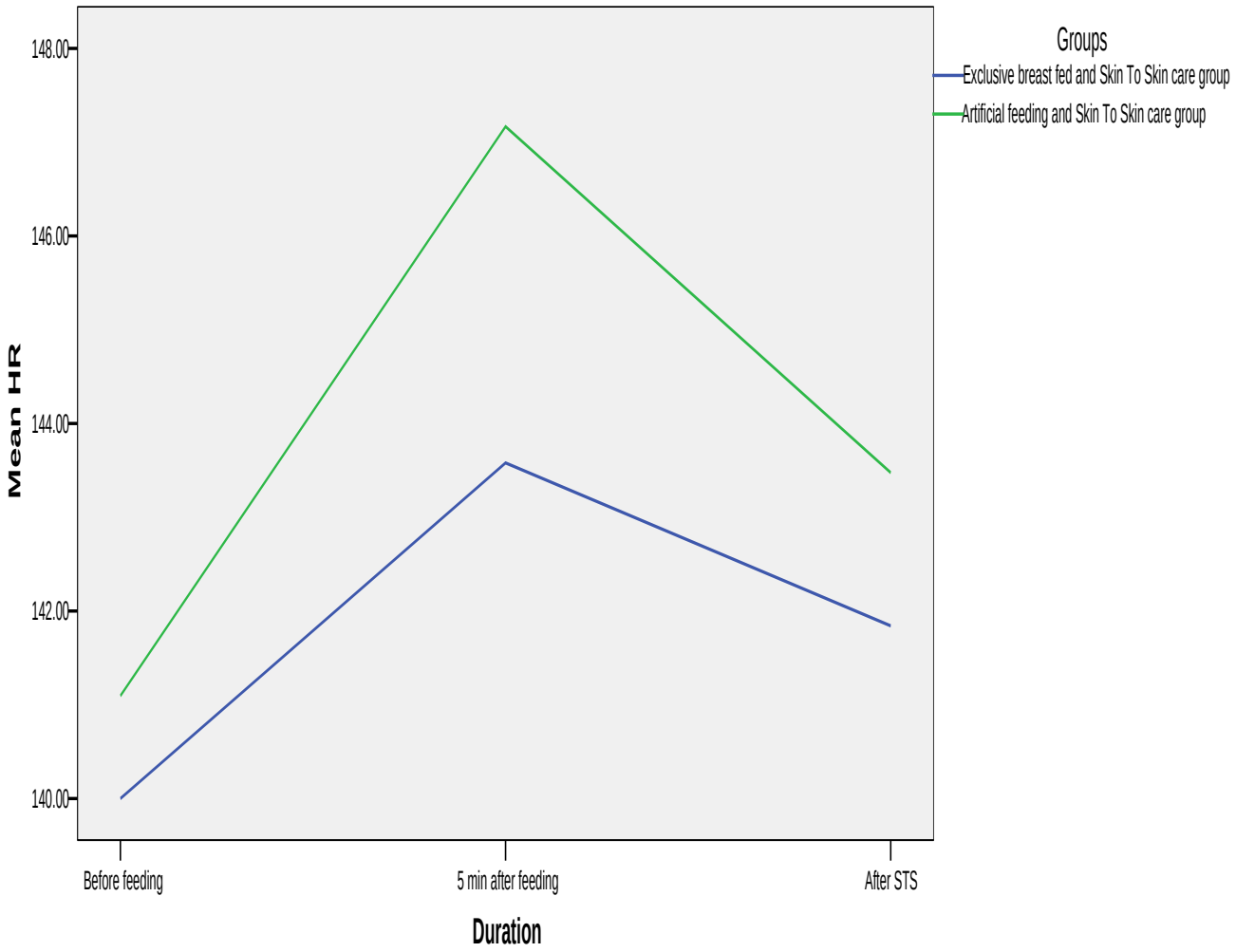


Figure (13): Comparing mean and SD of HR before, before, 5 min. and after SSC in the group under study.



Discussion

A congenital heart defect **(CHD)** is a problem which is present at birth. In the general population, congenital heart diseases are the most common of all congenital birth defects ***(Moller and Hoffman, 2005)***.

More than forty thousand babies all over the world are born each year with congenital heart diseases; 4000 will survive their first year (the children ***(Ferenz et al., 2008)***.

Kangaroo Mother Care **(SSC)** is method that was implemented in 1978 for low birth weight **(LBW)** infant, and underwent rigorous scientific evaluation in 1989.

Current evidence indicates that SSC is at least as good as traditional care with incubators. Studies have shown that SSC is safe, works at a fraction of the cost of an incubator, improves breastfeeding rates, and improves bonding between mother and infant.

Parental sense of fulfillment and confidence are also improved. During the past 15 years SSC has been practiced in 25 developing countries in Asia, Africa, and Latin America. Its use is also supported in industrialized countries such as France, Sweden, the United Kingdom and the United States. Almost two decades of implementation and research have made it clear that SSC is more than an alternative to incubator care and should be encouraged in affluent settings ***(Charpak, 2006)***.

However the benefits offered by SSC care for sick grown babies have not been studied. Babies with congenital heart disease in heart failure (HF) may benefit from SSC care, since SSC care may improve oxygenation as shown by the care of LBW by SSC ***(Charpak, 2006)***.

Also there is a controversy whether sick babies with CHD and HF need to fed on special formula feeds rather than breast milk. Also, to date, no studies were conducted to compare oxygen saturation in relation to mode of feeding in babies with CHD in Egypt.

The aim of this study is to shed more light on the effectiveness of breastfeeding with or without skin to skin care on the outcome of infant with CHD with or without heart failure.

The study examined the baby oxygen saturation and heart rate of the babies with congenital heart disease exposed to breastfeeding and SSC care versus others exposed to artificial feeding and SSC

The babies in the study were grouped in to three groups:

- 19 infants ≤24 months exclusive breastfeeding(for first 6 months) +SSC
- 21infants ≤24 months formula feeds given in first 6 months + SSC
- Control group of 15 infants, age and sex matched, with heart failure not exposed to skin to skin irrespective of feeding history.

The mothers of the babies were exposed to psychometrical assessment by STAI test.

Effect of breastfeeding versus artificial feeding on anthropometric measures:

In the current study, we found that there were no differences between breastfed group and artificially fed group, but when compared to control group there was difference between artificially fed babies with lower Z-score in weight among artificially fed babies. Other than that there was no

difference in the Z-scores of the length and head circumference, between the study groups.

Our results agree with the results obtained by another study conducted by ***Vohr et al., (2006)*** who found no difference in mean weight, length and head circumference in that study in both breast milk and formula fed groups.

Kramer et al., (2007) showed that the exclusive breastfed babies had higher growth rate than artificially fed babies during the first 3 months, with no difference in weight parameters after 3 months age.

No significant difference was observed in relation to height, body mass index, waist or hip circumference, triceps or subscapular skin fold thickness. Our results agree with the findings of ***Dewey.(1998).*** In affluent populations, breast-fed infants generally exhibit a different pattern of growth than formula-fed infants. The average weight gain of the former is lower than that of the latter, even after complementary foods are introduced. In some studies, the length gain is also lower among breast-fed infants, whereas in others there is no significant difference in linear growth between feeding groups. Growth in head circumference does not differ by feeding mode ***(Dewey, 1998)***.

Breastfeeding versus formula feeding in relation to morbidity

Our study showed that repeated admission to the hospital was 84.2% among breastfed group in comparison to 85.7% in artificially fed group. Both were lower comparable to the control group of 93.3% but with no statistical difference.

This could be explained by the fact that most hospital admission is due to recurrent heart failure which is related to the severity of the defect rather than the mode of feeding.

However in our series, formula fed had a higher rate of exposure to bronchitis (66.7%) compared to breastfed (42.1%).

These results agree ***with Marvin (1984)*** who reported a greater frequency of upper respiratory infections in the bottle-fed group,

Our results also agree ***with Popkin (1990) Wright, et al (1998) and Ryan (2002)*** , and other workers who demonstrated that breastfeeding decreases infant morbidity more than the formula feeding

In our study we found that bronchitis was one of the lower respiratory tract infection and was the most common cause of morbidity in comparison with others. However its incidence was higher in formula fed infants compared to breastfed babies. This agrees with other findings of other workers (***Cushing, et al., 1998, Cesar et al., 1999)***.

Otitis media was reported more frequently in breast fed babies than formula fed according to our study. This contradicts with other studies conducted to estimate frequency of the disease according to type of feeding (***Duncan et al., 1993, Dewey et al. 1995, Scariati et al., 1997)***.

The difference in the results from the author point of view may be due to various causes; such as small number of study group, also many other intervening factors such as smoking, socioeconomic level, environmental sanitation, mother education, and level of health service provided, especially the majority of breastfeeding are from rural area.

SSC and heart rate and oxygen saturation

In our study we found statistically significant initial increase followed by a decrease in the heart rate to the normal value. There was an increase

in oxygen saturation in both breastfed and formula fed groups exposed to SSC in comparison to the group that was not exposed to SSC.

However the breastfed group showed an even lower decrement than that of the formula fed group. This suggests synergistic effect of both SSC and breastfeeding.

These results agree with ***Bohnhorst et al., (2001), and Fohe et al., (2000)*** who mentioned that heart rate increased initially during SSC by 5bpm.

Also ***Ludington-Hoe et al., (1991)***, demonstrated an increase in the heart rate with SSC within the normal physiological range. Similarly ***Acolet et al., (1989) and Hosseini et al, (1992)*** supported the previous findings showing that during SSC there was increase in the heart rate within normal physiological range.***)***.

Measurements of heart rate during SSC had variable results in scattered studies. Pretest\ test\ posttest designs in many of them reported no statistically significant difference in heart rates compared to lying to lying in the incubator or when compared to traditional holding ***(Ludington, et al., 1999, Fischer et al., 1998, Messmer, et al., 1997, Bauer et al 1996; Bier et al., 1996; Bosque et al., 1995.***

However, decreases in heart rate have been associated with less stress, calming\ soothing experiences, quiet sleep, or mean heart rates that include bradycardias. In a time series randomized controlled design, ***Gazzolo et al., (2000)*** found that mean heart rate was lower in postoperative cardiac term infants during SSC provided after extubation.This suggests that SSC decreased postoperative stress and discomfort.

Gray et al., (2000) reported that infants had a no rise in the heart rate, as well as less crying and grimacing when heel sticks were done during SSC compared to lying in a bassinet, suggesting that the SSC group experienced less stress and discomfort.

A randomized test\ retest design, ***by Legault and Goulet (1995)*** found no significant differences in heart rate between SSC and traditional holding, but rather was more bradycardia in the traditional holding group.

Bohnhorst et al., (2001) found an increase in frequency of combined bradycardia and hypoxemia during SSC. The authors attributed the increase in bradycardia and hypoxia to an increase in no regular breathing patterns during SSC, suggesting that caregivers should monitor infants during SSC but that SSC is relatively safe for these infants.

Also we found that oxygen saturation is significantly higher with SSC in both groups in comparison to control groups. Also it is higher in breastfed babies than formula fed babies.

Dodd (2005) highlighted that, oxygen is important as a fuel for metabolism and growth especially in the brain.

Oxygen saturation is a noninvasive but gross measure of oxygenation in preterm infants. Poor oxygenation is a serious threat to metabolism and physiologic functioning, leading to poor heart function of normal SaO2 varies from NICU to NICU as identified by ***Chow et al., (2004).***

Most NICUs define normal as 90% to 98% saturation, while many NICUs set SaO2 alarms at 88% or greater as an acceptable level in preterm babies as reported by ***Tin et al., (2001).***

Acolet et al., (1989), reported that there was an increase in SaO2 in SSC in the study which was conducted in 14 very low birth weight infants, which supports the current study Similarly***, Kadam et al,(2005)***

Added that, in the SSC group there was higher oxygen saturation when comparing it with the control group. Also ***Foe et al, (2000)*** supported those results which go in line with their study result. However, the research investigator thinks this is due to satisfaction after breastfeeding and infant sleeping into his\her mother chest.

Several studies on SSC outcomes on SaO2 report no significant differences in SaO2 between SSC and incubator care ***(Ludington-Hoe et al., (1999) ; Fischer et al., (1998) ; Messmer et al., (1997) ;Mooncey et al., (1997) ; Bauer et al., (1996) ; Bosque et al., (1995) ; Ludington-Hoe, et al (1991) ; Acolet et al (1989))***.

Other studies indicated a statistically significant increase in SaO2, but the differences were within 1% to 2% and greater than 90% ***(Fohe, et al., 2000; Gazzolo, et al 2000; Bier et al., 1996; Legault, & Goulet, 1995)***.

Some workers reported a decrease in oxygenation with SSC, but again the differences were clinically insignificant ***(Ludington Hoe et al., (1994). However, Bohnhorst et al., (2001)*** documented more episodes of desaturation less than 80% during SSC in a study with 8 infants.

Although most studies reported that oxygenation appears to be stable or improved. ***Bohnhorst et al. (2001)*** study suggests that preterm infants need monitoring during SSC.

Ludington-Hoe et al. (2001) study supported those of the current study concluding that the mean cardio-respiratory outcomes remained within clinically acceptable ranges during SSC. Regular breathing increased for infants receiving SSC compared to infants receiving standard NICU care.

<u>Effect of SSC on STAI</u>

In our study, we found that mothers who practiced SSC whether breastfeeding or formula feeding had a significant decrease in their

anxiety score when compared to mothers who did not practice. Moreover when STAI was measured before and after SSC, there was considerable reduction in the anxiety state in both breastfed and artificially fed babies.

We found no literature to date, on the effect of the practice of SSC in grown babies with CHD and HF on mother's psychological status

However ***Cattaneo and his colleagues in 1998*** who studied 285 mother-infants pairs who were low birth weight exposed to SSC for 20 hours daily. They reported a significant increase in maternal emotional comfort with SSC. Also ***Dombrowski et al., in 2001*** and ***Burkhammer et al., in 2004*** described mothers with post-partum anxiety associated with eclampsia and post–partum depression and concluded that the introduction of SSC with breastfeeding had synergistic effects on sedating and calming both mother and baby and empowering the mother to care and nurture her baby.

In Egypt, ***Abul Fadl et al. (2006)*** studied the depression score of mother exposed to SSC and found those mothers had low depression score than mothers who did not practice SSC with their LBW babies.

A study was conducted for 73 preterm infants who received SSC in the neonatal intensive care unit that were matched with 73 control infants who received standard incubator care. Workers observed that at 37 weeks' GA, mother-infant interaction, maternal depression, and mother perceptions showed more positive effective responses, touch, and adaptation to infant cues, and infants showed more alertness and less gaze aversion. Mothers reported less depression and perceived infants as less abnormal ***(Feldman et al., 2002)***.

The same workers followed the mother infant dyads at 3 months' corrected age. They reported that infant temperament, maternal and

paternal sensitivity and the home environment (with the Home Observation for Measurement of the Environment [HOME]) showed that mothers and fathers of SSC infants were more sensitive and provided a better home environment. At 6 months' corrected age, cognitive development was measured with the Bayley-II and mother-infant interaction was filmed. The SSC mothers were more sensitive and infants scored higher on the Bayley Mental Developmental Index (SSC: mean = 96.39; controls: mean = 91.81) and the Psychomotor Developmental Index (SSC: mean = 85.47; controls: mean = 80.53). The workers concluded that SSC had a significant positive impact on the infant's perceptual-cognitive and motor development and on the parenting process. They speculate that SSC has both a direct impact on infant development by contributing to neurophysiological organization and an indirect effect by improving parental mood, perceptions, and interactive behavior **(*Feldman et al., 2002)***. ***Ohgi et al., in 2005*** carried out a randomized controlled trial upon 153 low birth weight infants and resulted in higher scores of emotional states of contentment of the mothers at 40 weeks and 12 months post partum.

Our study showed an even better psychological state (less anxiety score) among mothers who practiced SSC with breastfeeding in comparison with mothers who practiced SSC with formula feeding , which indicate that breastfeeding has an additive or augmenting and synergistic effect on the emotional state of mothers .

Effect of hospital admission on the anxiety state

Anxiety scores were much higher in the mothers of exclusively breastfed whose babies were admitted to hospital. However in the non breastfed, hospital admission had no effect on the severity of the anxiety score.

This could be explained by the higher bonding which is characteristic of mother who are breastfeeding their babies.

Effect of feeding session on O2 saturation and heart rate

In our study, we found that heart rate and oxygen saturation were statistically elevated within normal range in both breastfed and formula fed groups. There was a statistically significant difference between breastfed group and formula fed group.

However there was statistically significant difference in oxygen saturation during feeding session and at 10 minutes after feeding in both groups. In the breastfeeding group oxygen saturation did not decrease below 90% while formula fed babies scored lower results.

These results are similar to the results obtained by ***Shivpuri, et al. (1983)***. who found that transcutaneous PO2(TcPo2) in healthy preterm infants during bottle feeding, dropped an average of 13mm Hg for infant born at 34to 36 weeks gestation. TcSo2dropped an average of 10 mm Hg for infant 36 to 38 weeks gestation. A later study found that one factor associated with changes in respiratory pattern in preterm infants was milk flow. During bottle feeding with a traditional nipple, preterm infants in this study exhibited significant decreases in frequency of respirations and tidal volume. ***(Mathew, 1991).***

Singer et al., 1992) showed that even after discharge from the hospital, significant desaturation is associated with bottle feeding of preterm infants with bronchopulmonary dysplasia.

These studies show the effects of bottle feeding on oxygenation in preterm infants. Two studies have examined the effects of breastfeeding on oxygenation. Findings from studies ***(Meier, 1988; Bier et al., 1993)***

Reported that breastfeeding is not associated with desaturation during feeding. When stable preterm infants in Meier's study were fed by bottle, TcSo2dropped during sucking bursts, replicating previous research findings. However, during breast feedings, these same infants did not experience changes in TcPo2. An additional finding of this study was that TcSo2levels continue to drop for at least 10 minutes post bottle feeding. But after breast feeding, TcSo2levels remained at the infant's baselines. Bier et al., found similar decrease in oxygen saturations during bottle feeding, with no decreases during breast feeding. Another study was conducted in babies with CHD found the same results; ***(Marino et al., 1995)*** found that infants with CHD were more likely to have decreases in oxygenation during bottle feeding than during breast feeding.

Also, in our study there were significant difference in heart rate between breastfed group and formula fed group with the mean heart rate of the breast fed babies lower than that of formula fed babies.

These results agree with those obtained by ***Butte et al., (1991)***.and the results of ***Zeskind et al., (1992).***

In conclusion the results of this study suggest that the *context* in which breast-feeding occurs results in a more complex and energy-efficient pattern of behavioral organization than the *context* of bottle-feeding.

Summary

The growing evidence of the benefits of skin to skin care (SSC) shown by different studies has encouraged us to study the possibility of introducing this technique with babies with CHD. We are unaware of any other similar studies that have examined the relationship between SSC and congenital heart disease.

There is also much controversy about the effect of breastfeeding compared to artificial feeding on the health and growth and development of babies with congenital hear disease (CHD).

Our study included 55 babies with congenital heart disease **that were divided into three groups as follows**: 19 breastfeeding and 21 artificial fed babies and 15 babies who were taken as control babies with CHD but not exposed to the intervention irrespective of their mode of feeding.

Group I: 19 breastfeeding babies with CHD who were exposed to SSC

Group II: 21 Infant milk formula feeding babies with CHD who were exposed to SSC

Group III: 15babies with CHD not exposed to SSC control group both breastfed and artificially fed

Selection criteria:

- All infants under 24 months of age hospitalized for heart failure.
- Almost fully breastfeeding in the first six months and received foods after 7 months with continued breastfeeding or received infant milk formula before six months and were then only on formula or other milks feeding.
- Receive medication for heart failure
-

- Neither exposed to operative procedure nor operative procedure with residual cardiac dysfunction necessitating treatment.

Exclusion criteria in the intervention groups:

- Infants with heart failure due to any other cause.
- Other congenital anomalies apart from heart defect
- Neurological disease
- Mother sickness or severe malnutrition.
- Infant with CHD that was totally corrected and heart failure is secondary to chest infection or other cause than heart.

All subjects included in the study were subjected to the following:

1. Thorough family history including maternal illness and history of the previous pregnancies.

2. Thorough maternal antenatal history (with special focus on smoking, infections, drugs and any medical problems occurred during pregnancy).

3. Thorough obstetric history.

4. Postnatal history including respiratory distress, cyanosis or jaundice.

5- Assessment of the baby

All infant were admitted for one day and assessed as follow:-

- ❖ Weight to nearest gm.
- ❖ Supine height to nearest mm and body mass index.
- ❖ Head circumference.
- ❖ Vital signs (H.R, R.R, temp.)

Pao2 saturation, H.R, PO recorded just before the feeding, every min. during feeding & last 5min. after feeding (without SSC.).

- ❖ Assessing feeding protocol including type of feeding and its frequency and amount.
- ❖ Assessing the breastfeeding to ensure correct attachment via observation of the mother position, infant holding, and usage of both breasts alternatively and ensure exhaustion of one breast before shifting to the other.
- ❖ Counseling the mother to resolve any problems faced her during feeding or care by SSC.

6. Infants of group (1) were breastfed and cared for by SSC, while infants of group (11) were artificially fed and cared by SSC. Group (III) infants were not exposed by SSC and considered as control group.

7. The mothers of all neonates underwent assessment for anxiety using the STAI test and were done by self assessment questionnaires.

The aim of our study was:

1- Compare health, growth and developmental outcome of infants with CHD exposed to almost fully breastfeeding for six months and continued breastfeeding into the second year with infant with CHD exposed to any formula feeding from before six months and early cessation of breastfeeding before one year (after controlling for severity of defect and operative procedures).

2- Compare complications of CHD as chest infections, cyanotic spells, anemia and liver disease of infants with CHD exposed to almost fully breastfeeding for six months and continued breastfeeding into the second

year with infant with CHD exposed to any formula feeding from before six months and early cessation of breastfeeding before one year (after controlling for severity of defect and operative procedures).

3- Evaluate the clinical intervention of SSC on the outcome of infant with decompensated congenital heart disease (CHD) in breast fed and artificially fed infants. This will be assessed as follows:

- Does SSC improve the blood gases status?

- Does SSC reduce complications or control already present complications?

- Does hospital stay improve mother satisfaction and lessen her anxiety scores?

Our results showed

In the study we found that bronchitis as one of the lower respiratory tract infection was the most common cause of morbidity in comparison with others and we found that its incidence was higher in formula feeding than breastfeeding.

In our study we found statistically significant increase followed by a decrease in heart rate within normal value, and increase in oxygen saturation in both breastfed and formula fed groups exposed to SSC in comparison to the group that was not exposed to SSC. We found that heart rate decrease was within the normal physiological range in both groups. However the breastfed group showed a lower decrement than that of the formula fed group. This suggests synergistic effect of both SSC and breastfeeding.

In our study, we found that mothers who practiced SSC whether breastfeeding or formula feeding had a significant decrease in their anxiety score when compared to mothers who did not practice. Moreover when STAI was measured before and after SSC, there was considerable reduction in the anxiety state in both breastfed and artificially fed babies

Our study showed an even better psychological state (less anxiety score) among mothers who practiced SSC with breastfeeding in comparison with mothers who practiced SSC with formula feeding, which indicate that breastfeeding has an additive or augmenting and synergistic effect on the emotional state of mothers .

Severe and very severe states of anxiety were much higher among the formula feeding mothers compared to the exclusively breastfed mothers. This indicates that formula feeding increases the stress state of mothers and decreases their coping.

There was statistically significant difference in oxygen saturation during feeding session and at 10 minutes after feeding in both groups. In the breastfeeding group oxygen saturation did not decrease below 90% while formula fed babies scored lower results

Also, in our study there were significant difference in heart rate between breastfed group and formula fed group with the mean heart rate of the breast fed babies lower than that of formula fed babies.

Conclusion

SSC is shown to be an effective, efficient, acceptable, safe, affordable cost effective method for caring for infants with CHD with HF and can be used as adjunctive to ordinary medical treatment and can be very costly in our health care system.

Breastfeeding is primarily necessary to augment and support the benefits of SSC to the infants and mothers; also it can add support to baby health status by improving his respiratory and cardiac function and increase infant- maternal bonding. However much effort is needed to overcome the resistance towards changing practices towards the encouragement of SSC, and the practice of breastfeeding.

This requires the adoption of intensive locally revised and adapted educational programs that could be used to facilitate the introduction of the SSC and breastfeeding programs to our health and medical staff.

Recommendations

1. Designing multicenter studies including larger number of subjects and longer follow up periods to ensure the valuable efficiency of SSC for infants with congenital heart disease.

2. Designing large scale studies to assess needs of implementation of SSC including service introducer's opinion surveys.

3. Designing of national guidelines for promotion of SSC and its implantation for service providers with special care to the regional hospitals which need further support more than the capital ones.

4. Deigning of training programs in SSC for medical service providers and health staff in cardiology units.

5. Creating public awareness about SSC and breastfeeding through mass media.

6. Introducing SSC in the curriculum of undergraduate medical and nursing schools.

7. Change the attitude of health provider toward breastfeeding babies with CHD.

8. Designing of programs which encourage and aware mother of better results of breastfeeding babies with CHD and change the attitude of them toward it.

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