CLINICAL PROFILE AND SURGICAL OUT COME OF PATIENTS WITH CONGENITAL DIAPHRAGAMATIC HERNIA- A PROSPECTIVE STUDY

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CERTIFICATE

This is to certify that this dissertation entitled ***"CLINICAL PROFILE AND SURGICAL OUT COME OF PATIENTS WITH CONGENITAL DIAPHRAGAMATIC HERNIA"- A PROSPECTIVE STUDY*** submitted **by Dr.R.SRINIVASA KUMAR** to The Tamil Nadu Dr.M.G.R. Medical University, Chennai is in partial fulfilment of the requirement for the award of **MASTER OF CHIRURGIE BRANCH-5 PAEDIATRIC SURGERY** under my guidance and supervision,in the Deportment of Paediatric surgery, Government Rajaji Hospital,Madurai Medical college, Madurai during the period of his postgraduate residency in M.ch paediatric surgery from 2010to2013.

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DECLARATION

I **Dr.R.SRINIVASA KUMAR** solemnly declare that the dissertation titled "***CLINICAL PROFILE AND SURGICAL OUT COME OF PATIENTS WITH CONGENITAL DIAPHRAGAMATIC HERNIA" - A PROSPECTIVE STUDY*** is a bonafide work done by me at Government Rajaji Hospital during 2010 – 2013 under the guidance and supervision of my unit chief **Prof. DR.S.R. REGUNANDAN, M.S., M.Ch.,** Professor & HOD Department of Paediatric Surgery Madurai Medical College & Government Rajaji Hospital, Madurai.

This submitted to the TamilNadu Dr. M.G.R. Medical University, Chennai, in Partial fulfilment of the award of MASTER OF CHIRURGIE, in PAEDIATRIC SURGERY, degree examination to be held in AUGUST 2013

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Date **Dr.R.SRINIVASA KUMAR**

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CONTENT

AIMS AND OBJECTIVE

- To find out the incidence of congenital Diaphragmatic Hernia in Government Rajaji Hospital, Madurai.
- To evaluate the clinical presentation.
- To find out the incidence of Congenital Diaphragmatic Hernia (CDH) diagnosed antenatally.
- Total numbers treated and follow up for 1 year.

INTRODUCTION

The migration of abdominal viscera through a defect in the diaphragm into the chest results in a diaphragmatic hernia. The congenital form exists as three distinct anatomical types: (i) Herniation through the posterolateral foramen of Bochdalek (ii) Herniation through the substernal foramen of Morgagni; and (iii) Herniation through the esophageal hiatus.

The term 'congenital diaphragmatic hernia' (CDH) refers to the herniation of abdominal viscera through the posterolateral foramen of Bochdalek. Congenital diaphragmatic hernia remains one of the most difficult challenges in the paediatric surgery. The surgical aspects are relatively straight forward but the medical management of the associated pulmonary hypoplasia and pulmonary hypertension still eludes us. With each passing decade, new approaches to the medical and surgical management of this entity have been advocated but have not stood the test of time. The multitude of treatment options for CDH reflects our limited understanding of the pathophysiology and explains the relatively fixed mortality rate seen.

REVIEW OF LITERATURE

Historical Perspective:

The first description of congenital diaphragmatic hernia was reported by Lazarus Riverius in the 17th Century in a 24 year old man. Morgagni in 1796 discussed various types of diaphragmatic hernia and described the anterior defect which bears his name. In 1848, Bochdalek described the posterolateral congenital diaphragmatic hernia and recognized the associated pulmonary hypoplasia. Broman suggested that nonclosure of the pleuroperitoneal canal could result in the dorsolateral diaphragmatic defect.

It was only in 1940 when Ladd and Gross reported 16 cases treated by surgery with 9 survivors. It was soon recognized that early surgery did not necessarily improve survival in congenital diaphragmatic hernia and that associated pulmonary hypoplasia and pulmonary hypertension determine outcome. The search for a specific pulmonary vasodilator continues. Preoperative stabilization and delayed surgery were proposed in the early 1980's. In 1976, Bartlett and colleagues reported the first survivors of pulmonary hypertension treated with ECMO. The animal model of CDH in fetal lambs described by deLorimer et al (1967) was an important landmark in understanding the pathophysiology of the defect.

Epidemiology

In an analysis of 5 population based studies on the incidence of CDH, Katz et al reported an incidence of 0.3 per 1000 live births.[1] Sweed and Puri reported 116 infants born with CDH in a discrete geographical population and reported an incidence of 0.315 per 1000 live births.[2] The incidence of CDH depends on the denominator used. The 'hidden mortality' (i.e. those fetuses and neonates who die without recognition of the anomaly) in CDH defines the true incidence of the defect. Puri and Gorman analyzed 47 cases of CDH in a hospital with an autopsy rate of 100 for still births and neonatal deaths.[3] They noted an incidence of 1 per 2107 total births in this study. The male to female ratio was 1:1.8 in this study and this female preponderance has been reported by other authors also.

CDH is generally considered to be a sporadic anomaly, although some familial cases have been reported. Assuming multifactorial inheritance, the expected recurrence risk in a first degree relative is approximately 2%. Nongenetic etiological factors like thalidomide, quinine, nitrofen and a vitamin. A deficient diet has been implicated as causative factors for congenital diaphragmatic hernia.

Embryology

The nitrofen rat model of CDH allowed detailed study of abnormal diaphragmatic development.[4] Kluth et al used scanning electron microscopy on normal rat embryos and rat embryos after nitrofen exposure to detail the embryogenesis of CDH.[5]

The classical theory of diaphragmatic development attributes four sources for the diaphragm (i) the septum transversum forming the central tendon, (ii) mediastinum and dorsal mesogastrium of the esophagus forming the median portion and crura, (iii) pleuroperitoneal membranes forming a small dorsal portion and (iv) thoracic body wall musculature forming the major circumferential muscular part of the diaphragm.

Kluth et al have described the normal embryology of the diaphragm in two phases:

The diaphragm exists as a mass of mesenchyme between the epithelial linings of the pericardial and peritoneal cavities. The dorsal border is established by the pericardioperitoneal canals. The developing liver grows into the septum transversum causing its expansion in a ventrolateral direction. The transverse septum is continuous with the dorsal structures via the pulmonary ridges laterally and the mediastinum

medially. The lateral pulmonary ridges are the forerunners of the pleuroperitoneal membranes.

There is a controversy regarding the amount of contribution of the pleuroperitoneal membranes to form the diaphragm. Broman stated that most of the adult diaphragm was derived from it, whereas Wells believed that the pleuroperitoneal membranes have a minimal contribution in diaphragmatic development. At the end of this first stage of development, the primitive diaphragm consists of the ventral transverse septum and the two dorsal pleuroperitoneal canals (PPC).

Development of the pleural cavity and closure of the pleuroperitoneal canals (4th - 8th gestational week):

The closure of the pleuroperitoneal canal (PPC) is believed to be crucial for normal diaphragmatic development. The theories described for closure of the PPC are -

i. Development of the pleuroperitoneal membrane.

ii. Dorsocranial development of the liver.

iii. Bremer believed that the large suprarenal glands were important in the closure of the PPC.

iv. Wells stated that the PPC was 'crowded out of existence' by the adjacent liver, gonads and suprarenal glands.

v. In 1984, Iritani described a new concept in the development of the diaphragm.6 He described the post hepatic mesenchymal plate (PHMP) as a mass of mesenchyme lying dorsal to the liver and ventral to the pleuroperitoneal canals.

Kluth et al have shown that in the second phase of diaphragmatic development a system of folds appear in the pleural cavity that marks the borderline between the pleural and peritoneal cavity. With the growth of the liver and the PHMP the above mentioned folds form oval openings called the PPC. The liver serves as a matrix for the development and growth of the PHMP. The closure of the PPC is completed by the eighth week and is primarily a result of the dorsolateral growth of the PHMP.

The muscularization of the diaphragm takes place by in situ myoblast differentiation or by migration of myoblasts from the cervical and occipital somites

Lung Development

Lung development is divided into 5 stages:

- **Embryonic stage (3-6 weeks):** The lung originates as a ventral diverticulums form the caudal end of the laryngotracheal groove of

the forgut in the 3rd week. The diverticulum divides to form two lung buds (4th week) which further divide to form the lobar and subsequently the bronchopulmonary segments (6th week).

- **Pseudoglandular stage (6 - 16 weeks):** The conducting airways (16 - 25 generations) are formed by repeated dichotomous branching. The lung appears as multiple small epithelium lined tubules in a mass of mesenchyme. By the 16th week all bronchial airways are formed and no further increase in airway number occurs after this stage. Bronchial cartilage begins to be formed in this stage.

- **Canalicular phase (17-24 weeks):** In this stage the basic structure of the gas exchanging portion of the lung is formed and vascularized. The small blind ending channels become complex; the interstitial tissue decreases and capillary growth starts. Type-I pneumocytes appear at 20 weeks and at 22- 24 weeks, type-II pneumocytes with lamellar bodies can be identified.

- **Terminal saccular phase (24 weeks - term):** There is a marked decrease in interstitial tissue and thinning of the air space walls. The distal airspaces divide into saccules, thus increasing the gas exchanging surface area.

- **Postnatal alveolar phase (Birth - 8 years):** Mature alveoli appear at about the 5th week as around 20 million primitive terminal sacs. This increases to around 300 million alveoli by 8 years of age with the maximum increase being in the first 4 years of life.

Lung Development in CDH

The size of the lungs in the 13 or 14 day old nitrofen-treated embryo is not different from that of a control group. Iritani has proposed primary lung hypoplasia as a cause of congenital diaphragmatic hernia.[6] Kluth et al concluded from their studies that the hypoplastic lung results due to the ingrowth of the liver into the thoracic cavity. The ingrowing liver reduces the available space for the lung to grow properly. Since this defect occurs early in embryogenesis, spontaneous correction of these lung defects is unlikely even after fetal intervention. The balloon studies in fetal sheep actually mimic congenital cystic adenomatoid malformation (CCAM) more than CDH.[7-9].On the other hand, since congenital diaphragmatic hernia is an early embryological event, the lung defects are primarily structural and not as a result of direct compression. The nitrofen model has also provided insights about lung development. The epithelial branching process depends on the interaction of the epithelium with the

surrounding mesenchyme. Collagen type-III and IV, fibronectin and laminin are believed to be important regulators of epithelial development.

Surfactant production by type-II pneumocytes is deficient in CDH.[10] Phosphocholine cyti-dylyl transferase (CTP) is a rate limiting enzyme in the synthesis of phosphatidylcholine which is the primary component of surfactant. Decreased production of fibroblast pneumocyte factor by lung fibroblasts is believed to be responsible for decreased CTP activity in fetal type-II pneumocytes (in congenital diaphragmatic hernia fetuses). This is responsible for the decrease in phosphatidylcholine synthesis in CDH.[10]

On day 18 and 19, control lungs and nitrofen exposed lungs look similar. The lung volume is however lesser in the nitrofen treated group. In the canalicular stage of lung development, the dilated branching tubules are less numerous in the nitrofen exposed fetuses with CDH. The saccular stage shows slit like air spaces with thick septa and more type-II cells per surface area in the nitrofen exposed lungs as compared to normal control lungs. The intra and extracellular multilamellar bodies are markers for maturation of type II pneumocytes. In nitrofen exposed lungs, these lamellar bodies have an abnormal configuration.

Thus, lung development is affected at an early stage of embryogenesis in congenital diaphragmatic hernia and leads to a reduced lung volume and weight. There is a reduction in the numbers of generations of bronchi and an alteration in the number and function of type II pneumocytes. In congenital diaphragmatic hernia, interruption of bronchial airway branching occurs around the 10th week of gestation when the midgut returns to the abdominal cavity. The ipsilateral lung is retarded around the 12th - 14th airway division whereas the contralateral lung is affected at the 16th-18th division.

Pathophysiology of congenital diaphragmatic hernia:

Areechon and Reid were the first to note reduced bronchiolar division in congenital diaphragmatic hernia and this led to the belief that pulmonary hypoplasia is directly responsible for the mortality associated with CDH.[11] The reduced lung volume and functional residual capacity are associated with a commensurate reduction in the alveolar surface area. The dead space is also increased.

Many neonates with congenital diaphragmatic hernia demonstrate adequate oxygenation for a period of time (called the ‘honeymoon period’) before deterioration. Since pulmonary hypoplasia causes a fixed reduction in gas exchange, the 'honeymoon period' cannot be explained,

Persistent pulmonary hypertension and vascular abnormalities in CDH are now believed to be important determinants of outcome, besides the pulmonary hypoplasia.

Pulmonary vascular development in congenital diaphragmatic hernia:

Kitagawa has shown that the number of arterial branches in congenital diaphragmatic hernia is reduced along with an increased wall thickness.[12] Naeye et al studied a series of 12 patients with CDH and noted a significant increase in pulmonary artery muscle mass.[13] There is a decrease in the overall crossectional area of the pulmonary vascular bed.[14] Geggel et al described the morphometric analysis of pulmonary arteries in a series of babies who had died from CDH.[15] Three of these patients had a honeymoon period before eventual death and 4 had distress at birth and could not be resuscitated. In babies who did not have a honey moon period, the muscularization of the arterioles had extended to the level of the alveolar ducts. Thus, the pulmonary vascular abnormalities described in congenital diaphragmatic hernia are:

1. Reduced number of conventional and supernumerary arterial branches.

2. Medial hyperplasia of arteries.

3. Distal muscularizations present till the alveolar duct level. Several studies have demonstrated alveolar growth and pulmonary vascular remodeling after successful repair of the defect. Beals et al reported that in 21 postmortem examination there were changes depending on the postnatal age.[16] The percentage of intra acinar muscularized arteries decreases with age; the lung volume and weight also increase. However, the number of airway generations remains constant.

Fetal and transitional pulmonary circulation:

There is a dramatic increase in the number and size of pulmonary vessels during the second and third trimester of pregnancy. The high pulmonary vascular resistance is maintained by relative vasoconstriction of the pulmonary vascular bed.

Transitional circulation:

At birth there is a sudden decrease in pulmonary vascular resistance which leads to a 10-fold increase in pulmonary blood flow. This increases the pulmonary venous return to the left atrium and helps in closure of the foramen ovale. There is a concomitant increase in the systemic vascular

resistance which reverses the blood flow in the ductus arteriosus. The ductus arteriosus closes (physiologically) in response to increased oxygen tension. The stimuli for this transition and fall in pulmonary vascular resistance are: (i) Lung expansion and onset of ventilation; (ii) Decrease in carbondioxide concentration; and most importantly, (iii) Increase in alveolar oxygen tension.

Persistent pulmonary hypertension (PPHN) in CDH:

The presence of PPHN in congenital diaphragmatic hernia was suspected based on the difference between the pre and postductal arterial blood gas values. Cardiac catheterization has confirmed the presence of persistent fetal circulation in CDH, In CDH, there is a fixed reduction in the cross-sectional area of the pulmonary vascular bed. In addition, this is compounded by a variable functional inadequacy of the pulmonary vasculature which leads to a further decrease in pulmonary blood flow and increases the right to left shunt. The mechanisms which are responsible for PPHN in congenital diaphragmatic hernia are:

Structural factors:

1. Endothelin 1 is a peptide released from pulmonary endothelial cells and causes vasoconstriction via the endothelin A receptor. Kobayashi and Puri reported elevation in endothelin 1 levels coinciding with

clinical episodes of deterioration in CDH infants17.Endothelin may play a role in the proliferation of pulmonary vascular smooth muscle seen in congenital diaphragmatic hernia. Experimental work is presently on to investigate the role of endothelin receptor antagonist in PPHN. Bosentan and BQ-123 are the two known endothelin antagonists and both have shown capacity to reduce proliferation of vascular smooth muscle.

2. TGF- (and insulin like growth factor may also have a role in the pathogenesis PPHN).

3. Vasoactive mediators.
    1. **Nitric Oxide:** The role of nitric oxide in transitional circulation and the etiogenesis of PPHN. Inhaled NO therapy has been used clinically in congenital diaphragmatic hernia usually in combination with surfactant, ECMO, HFV or perfluorocarbons associated gas exchange. Experimental studies have not demonstrated an abnormality or deficiency of the NO system in CDH. The nitrofen rat model of CDH has however shown a significant decrease in nitric oxide synthetase activity.

    2. **Prostaglandins:** Prostacyclin (PGI2) is a potent vasodilator and may play a role in transitional circulation. Leukotrienes C4 and

D4, thromboxane A2 and PAF are potent vasoconstrictors. An imbalance between these mediators may contribute to the pathogenesis of PPHN in CDH. Inhibition of prostaglandin synthesis has been shown to inhibit the pulmonary vasodilation caused by rhythmic distension of the lung. Increased levels of plasma thromboxane A2 have been found during episodes of pulmonary hypertension in the neonate with congenital diaphragmatic hernia.

Parenchymal factors:

Surfactant deficiency - Click et al demonstrated that the fetal lamb model of CDH was surfactant deficient.[10-18] The hypoplastic lungs along with the surfactant deficiency leads to barotrauma (interstitial emphysema) during overaggressive ventilation. This interstitial air trapping further worsens the pulmonary hypertension by compressing the pulmonary vessels. Surfactant therapy decreases pulmonary vascular resistance and improves pulmonary blood flow.

In conclusion, the pathophysiology of CDH involves:

1. Pulmonary hypoplasia causing a fixed reduction in the number of alveoli available for oxygenation.
2. Labile and reactive pulmonary hypertension and persistent fetal

circulation resulting secondary to vascular smooth muscle hyperplasia and vasoactive mediators.

3. Surfactant deficiency and overall diminished compliance leading to barotrauma and ventilation perfusion mismatch.
4. Cardiac dysfunction with lowered cardiac output complicates the pulmonary problems. Relative left ventricular hypoplasia with attenuated muscle mass and cavity size have been noted in the fetus and neonate. This cardiac hypoplasia results from poor venous return to the left heart.

Associated Anomalies

The incidence of associated anomalies in CDH varies between 39% and 50%.[19,20] The incidence of associated anomalies can be as high as 95% -100% in stillborn CDH fetuses. Puri and Gorman noted a high incidence of neural tube defects in still born with CDH.[21] Sweed and Puri analyzed the incidence of associated anomalies in 116 patients with congenital diaphragmatic hernia and its impact on survival.[2] In patients who succumbed during the initial stabilization without surgery (mean age 11.2 hrs), 63% had associated malformations. In the other group of patients who underwent surgery after stabilization, only 8% had associated malformation.

Cardiac anomalies constitute around 63% of the associated anomalies.[19,22] Hypoplastic heart and atrial septal defects were the most common findings on postmortem examination. A low postductal PaO_2, should prompt a search for a hidden cardiac defect. Ventricular septal defects, tetralogy of Fallot, coarctation of aorta and true hypoplastic left heart syndrome may be associated. Cardiac and neural tube defects constituted the majority of defects.

Anatomy of the defect:

The defect is seen on the left side in around 80% cases. Bilateral defects are rare and only 11 cases have been reported in the literature.

The size of the defect varies from the usual 2-3 cm to almost a completely absent hemidiaphragm. A rim of muscle is usually present around the defect which is more prominent anteriorly. This rim of muscle is not evident posteromedially where it is covered by the peritoneum. A hernial sac is reported in about 20% of patients.

Clinical Features:

The onset of symptoms depends on the volume of viscera in the chest and the degree of pulmonary hypoplasia. A history of polyhydramnios may be noted in upto 80% cases. Prematurity is also noted frequently.

Immediate presentation:

The most severely affected babies present with respiratory distress at birth. Cyanosis, tachypnea, grunting and retractions are noted on physical examination; the abdomen is scaphoid; the anteroposterior diameter of the chest is increased; mediastinal shift can be noted by the presence of tracheal deviation and shift of heart sounds; breath sounds are absent on the affected side and bowel sounds may be heard in the chest.

Diagnosis:

The diagnosis is made on the chest radiograph which shows air filled bowel loops in the chest cavity with absence of a visible diaphragmatic margin, mediastinal shift and a relative paucity of abdominal gas. A small portion of the ipsilateral lung may be visible superiorly. The differentiatial diagnosis of CDH on X-rays includes (i) Congenital cystic adenomatoid malformation (CCAM), (ii) Other congenital lung cysts. The passage of a nasogastric tube can help in confirming the diagnosis. In CCAM, the outline of the lung and the diaphragm can usually be made out, mediastinal shift is not marked and normal intestinal gas pattern can be seen. A small amount of dilute barium can be instilled via the nasogastric tube to visualize the stomach and see its position relative to the diaphragm. Contrast studies are rarely required. Ultrasound may be

helpful in identifying and measuring the diaphragmatic defect. A cardiac echocardiogram is performed to exclude associated congenital heart defects, measure the left ventricular mass index and to determine the direction of ductal shunting.

Management:

A neonate without respiratory distress requires only the passage of a nasogastric tube to avoid bowel distension. On the other hand, neonates presenting with distress at birth require intubation and ventilation. The medical management of neonates presenting with respiratory distress at birth is challenging. CDH, unlike in the past, is not considered a surgical emergency and preoperative stabilization is the primary goal of treatment. The surgical aspects of the management are straight forward and relatively free from controversies.

Bag and mask ventilation is contraindicated as it causes bowel distension and increases the mediastinal shift. This mediastinal shift affects the contralateral lung and also compromises cardiac function. The neonate is nursed in the intensive care with the following general measures:

i. Maintain normothermia.

ii. Intravenous fluids.

iii. Nasogastric intubation and drainage.

iv. Inotropic support with dopamine and dobutamine may be needed to maintain the blood pressure and peripheral circulation .

Monitoring and blood gas parameters:

Arterial blood gases are the primary determinants of management protcol and prognosis in neonates with CDH. A right radial artery cannula is inserted after checking adequacy of the ulnar circulation to monitor the preductal blood gases. An umbilical artery catheter is inserted to monitor the postductal blood gas values. Pre and post ductal pulse oximetry probes are used to check the saturation. The hemodynamic status is monitored by recording the blood pressure, the heart rate and capillary filling time. The intravenous fluid volume should be enough to maintain the perfusion but not excess so as to cause opening of the ductus. The ventilatory parameters are adjusted based on the preductal arterial blood gas values. Boix-Ochoa et al were the first to show a correlation between arterial blood gases and outcome.[22] They showed that the initial pH (mean 6.85) at presentation and the $PaCO_2$ (mean 142 mmHg) at presentation was significantly different in non-survivors as compared to survivors (pH-7.17, $PaCO_2$ - 60 mmHg). Mishalany et a! used the arterial

pH on admission in 58 neonates to predict survival – infants with pH>7.2 at presentation had 100% survival, infants with pH>7.0 had 50% survival and infants with pH<7.0 at presentation had only 11% survival.[23] The various arterial blood gas values used as predictors of outcome in CDH neonates are :

1. Arterial $PaCO_2$ and pH: Boix-Ochoa et al showed the importance of the pH and $PaCO_2$ at presentation and its prognostic significance. The significance of a pH value <7.2 and $PaCO_2$>50 mmHg at presentation was highlighted in other studies as well and was an indicator of poor prognosis. It is important to correlate the value of $PaCO_2$ with ventilatory parameters to assess the response to therapy. Bohn et al have described the ventilation index (VI) as[24,47] VI = Ventilatory rate x mean airway pressure. They subsequently correlated the preoperative $PaCO_2$ values with the VI and identified three groups of patients.

(i) $PaCO_2$ < 40, VI < 1000 – 100% survival.

(ii) $PaCO_2$ < 40, VI > 1000 – 38% survival.

(ili) $PaCO_2$ > 40, VI > 1000 - <10% survival.

This grouping of patients based on the $PaCO_2$ and ventilatory parameters has a good predictive value and helps in deciding the timing

of surgical repair. With the introduction of permissive hypercapnia in the management of CDH, the role of $PaCO_2$ in predicting outcome has decreased.

2. Arterial PO_2 (PaO_2) : Boix-Ochoa found in 15 neonates that a PaO_2 of <300 mm Hg on a FiO_2 of 1.0 was associated with a poor prognosis.[22] Ruff et al also noted a high mortality associated with a PaO_2 of <60 mmHg and a pH of <7.2.[25] The value of PaO_2 however depends on the site of sampling (pro vs postductal) and varies significantly with change in ventilatory parameters.

3. Alveolar to arterial oxygen difference ($A\text{-}aDO_2$)

   $A\text{-}a\ DO_2$ = Alveolar PO_2 - Arterial PaO_2

   Alveolar PO_2 = Inspired PO_2 – $PaCO_2$

Normal neonates on room air maintain a $A\text{-}aDO_2$ of <10 mmHg. Large gradients are seen in patients with right to left shunting like CDH. The ratio of the arterial to alveolar PO_2 (a/APO_2) is another good indicator because it is independent of the concentration of inspired air. The normal value of a/APO_2 is 0.8. Harrington et al used $A\text{-}aDO_2$ as a predictor of outcome after hyperventilation and bicarbonate therapy.[26] They found that in survivors the mean postoperative $A\text{-}aDO_2$ was

319 mmHg as compared to 562 mmHg in non-survivors. Bohn et al also noted that the preductal A-aDO_2 in 54 infants with CDH was significantly lower in survivors (mean 223 mmHg) as compared with non-survivors (mean of 474 mmHg).[24]

Timing of Surgery:

The concept of early surgery in congenital diaphragmatic hernia was advocated by Ladd and Gross in the 1940's. It was believed at that point of time that reduction of the hernia would relieve the lung compression and improve oxygenation. It has now been realized that CDH is far from merely a surgical problem. Cartlidge et al demonstrated a significant increase in survival rate from 13% in newborns with CDH operated early as compared to 53% survival in newborns operated after stabilization.[27] The Toronto group reported their pre ECMO experience with equivalent survival with early (52%) versus late (55%) repair. There is increasing physiologic and clinical justification in delaying surgical repair and achieve stabilization. Nakayama et al demonstrated significant deterioration in lung compliance following reduction and closure of the defect.[28] This meant that rather than being beneficial, surgical intervention was detrimental in terms of pulmonary compliance and oxygenation. Sakai et al also showed that pulmonary compliance

decreased 10-77% after surgical repair.[29] It has also been shown that a fall in pulmonary compliance of greater than 50% was associated with a 100% mortality. The factors proposed for this fall in compliance after surgeries are:

(i) Downward displacement and stretching of the diaphragm.

(ii) Distortion of the lower chest wall associated with the incorporation of the internal oblique muscle in the repair.

(iii) Tight abdominal wall closure.

(iv) Ipsilateral displacement of the mediastinum causing over distension of the contralateral lung.

A number of studies have assessed the efficacy of the delayed approach to the repair of newborn parents with congenital diaphragmatic hernia. These studies varied in their conclusions; in some delayed repair was associated with marked increase in survival whereas in others no difference was noted.[30,31]

Nio et al reported the results of a randomized controlled study and could not demonstrate a significant difference in survival rate between immediate (12 patients) and delayed repair (18 patients operated >96 hours) (75% vs 72% survival).[52] The results in the same study with the use of ECMO showed 67% survival in the group operated immediately

and 89% in the group which underwent delayed repair. Jaffray ad Mackinlay showed an improvement in survival from 45% to 59% with a delayed approach. They also stated that a large number of neonates died prior to reaching their surgical centre. This 'natural selection' in congenital diaphragmatic hernia is well known and explains the excellent results reported by Gross in the 1940's. The study by Jaffray et al also showed a 37% survival rate in infants born in central hospitals as compared to 75% survival in peripheral units.This fact raises the pertinent question whether a delayed approach is actually beneficial or helps only in the process of natural selection (and hence improves the survival rate).

The other factor which is probably an important reason for deferred surgery is the resolution of PPHN. Haugen et al have used echocardiography to determine pulmonary artery pressures in 8 neonates with congenital diaphragmatic hernia.[33] The pressures at birth ranged between 45 - 90 mmHg with evidence of bidirectional or right-to-left flow. Pulmonary hypertension resolved after 3 to 20 days (mean of 8 days) as the pulmonary arterial pressures decreased to 25-55 mmHg.

Two studies have evaluated the effect of a 'very delayed' approach (>72 hours) to repair in CDH. Wung et al studied the survival rates in 3 groups of patients: group1 immediate operation

(6 ± 6 hours); group 2-delayed operation (22 ± 23 hours) and group 3-very delayed operation (100 ± 44 hours).[34] All the patients were managed on the principle of permissive hypercapnia. Survival rates in the three groups were 82%, 75% and 94%. The need for ECMO was lesser in group 3 (6%) as compared to group 2 and 1 (25% and 35% respectively). Hirschl et al also reported a survival rate of 56% in 66 patients who underwent delayed repair at 1.2 ± 1.9 days and 68% of these patients required ECMO.[35] With the very delayed approach (repair at mean of 9 ± 5 days), the survival rate significantly increased to 81% and the ECMO utilization rate decreased to 33%.

All the published data suggest that urgent repair does not improve survival or gas exchange. The emphasis of management should be directed towards the treatment of PPHN. Lung compliance studies and the gradual resolution of PPHN detailed earlier definitely justify a delayed or very delayed approach to repair of CDH.

Operative Procedure:

CDH is not a surgical emergency and preoperative stabilization is a prerequisite. Deferred surgery has not in itself increased the survival rates, but has helped in selecting survivors from non survivors. The

principle steps of the operation are:

* Anaesthetic considerations- IV access is required above and below the diaphragm; pulse oximetry in pre and post ductal sites; monitor BP; use muscle relaxants, narcotics and isoflurane for minimizing surgery induced pulmonary hypertension; controlled ventilation using high frequency, low tidal volume, pressure limited ventilation.
* Transabdominal route used because (i) Easier reduction of viscera by the abdominal route, (ii) Accurate visualization of the defect and repair possible, without risking injury to gut and other abdominal organs, (iii) Can correct any other associated intestinal anomaly or perform a Ladd procedure if needed, (iv) Can enlarge the abdomen by manual stretching if required or construct a silo. In case of right sided defects, if only the liver is herniated then a transthoracic approach is advisable. In the presence of bowel an abdominal approach is used for right sided defects also.
* Subcostal incision made 2 cm below rib margin. Draping should allow right chest visualization in case a contralateral pneumothorax develops during surgery.
* Gentle reduction of viscera with liver and spleen last, use of a Red rubber catheter to equalize the intrathoracic pressure facilitates

reduction of viscera.

* Identify the rolled posterior diaphragmatic rim and dissection of the pleural- peritoneal fold to release the posterior rim.
* Closure of the defect by a single layer horizontal mattress non-absorbable suture starting medially. For large defects the options are-synthetic mesh repair, intercostal muscle flaps, internal oblique and transversus abdominis flaps, rectus abdominis flaps, reversed latissmus dorsi flaps.
* Additional procedures like Ladd procedure performed if the patient is stable.
* Abdominal wall closure only after ensuring that respiration is not effected. If closure is under tension then the options are-skin closure only, patch repair or construction of a silo.
* Right sided defects - The reduction of the liver should be done with careful hemodynamic monitoring because kinking of the hepatic vein or vena cava can occur causing a decrease in venous return. Careful dissection of the fibrous attachments of the vena cava to the medial side of the defect will help in reduction. Also the liver is very friable and soft and can be easily traumatized.

Use of Chest Drainage:

The use of prophylactic chest drainage after the repair of CDH is controversial. Pulmonary barotrauma depends on the peak inspiratory pressure and the transpulmonary pressure gradient (TPG). Ipsilateral chest drainage increases the TPG and therefore increases the incidence of barotrauma. The small pocket of air which remains after repair of the defect and no chest drainage acts as a protective cushion by decreasing the TPG. Air leaks cause hypoxia, acidosis and exacerbate the pulmonary hypertension.

Adjunctive Therapies:

Extracorporeal Membrane Oxygenation (ECMO)

The induction of ECMO since the early 1980's led to a tremendous enthusiasm regarding its use in neonates with congenital diaphragmatic hernia. It was believed that ECMO would help in tiding over the period of time required for postnatal vascular remodelling. However, the ELSO registry data has shown that of all babies with acute respiratory failure requiring ECMO, the worst outcome is in babies with CDH.[39] A 58% survival rate, compared with an overall survival of more than 81% has remained essentially unchanged over the past 10 years.

ECMO is an invasive procedure which is expensive and associated with significant risks. The initial selection criteria were based on predictors of high mortality rate (>80%) in newborns with congenital diaphragmatic hernia. Individual centres with ECMO facility have used their own set of criteria for instituting ECMO in neonates with congenital diaphragmatic hernia. The various criteria used in the series reported are:

1. Alveolar to arterial oxygen difference (A-aDO_2) - The A-a DO_2 level of >600 for a duration of 8 hours has been used as a predictor of high mortality and need for ECMO support.[40, 43]
2. Oxygenation index (OI = MAP x FiO_2 x 100 (PaO_2) : The OI criteria have also varied but in general a OI value of >40 for 2-3 hours suggests the use of ECMO.[40,41] Butt et al suggest that ECMO should be offered to congenital diaphragmatic hernia patients with an OI above 30.[43]
3. Other criteria - Preductal PaO_2, ventilation index, pulmonary compliance, percentage of nucleated red blood cells, clinical evidence of deterioration or pulmonary barotrauma.

The idea behind these selection criteria is to avoid offering treatment to neonates with fatal pulmonary hypoplasia. The problem lies in the fact that there is till date no absolute criteria which can predict irreversible

fatal pulmonary hypoplasia. It is also known that neonates may sustain severe lung injury using conventional ventilation. This has led to a trend of offering ECMO support earlier in congenital diaphragmatic hernia to avoid this iatrogenic lung injury. A brief 'honeymoon period' of adequate duration is a good indicator of sufficient pulmonary reserve and such patients may benefit from ECMO.

Timing and duration of ECMO:

ECMO support can be provided before repair of the defect or after the repair. With the advent of delayed repair more infants are being placed on ECMO preoperatively, in the hope of achieving stabilization. The incidence of hemorrhagic complications is high with repair on ECMO and the mortality rate in this group of patients is high. The operative procedure for patients on ECMO has been modified to decrease the incidence of hemorrhagic complications[44] - (i) use of perioperative aminocaproic acid, (ii) liberal use of electrocautery and meticulous hemostasis, (iii) application of fibrin glue, (iv) liberal use of Gore-Tex patch repair instead of extensive dissection, (v) avoid stretching of the abdominal wall and resorting to a silo pouch if needed. Recurrent hernia is common after the repair of congenital diaphragmatic hernia on ECMO and can be seen in upto 50% patients.[45] Ideally, surgery should be

avoided while the patient is on ECMO.

Weaning from ECMO may take days and currently the mean time on ECMO for patients with CDH is 191 hours.[39] The ELSO national registry data also suggests that survival is significantly decreased when more than two weeks of ECMO support is required. In general, an ECMO course of more than 14 days is usually not required. There is also a risk of recurrent hypoxemia and pulmonary hypertension after weaning from ECMO support. It is advisable to leave the neck cannula in place for several days after discontinuing ECMO, with continued heparin infusion to keep the line patent.

Venovenous ECMO is preferred over venoarterial ECMO; the carotid artery does not need to be ligated, pulsatile flow can be maintained and myocardial oxygen delivery is better. Heiss et al found that venovenous bypass was as effective as venoarterial ECMO.[46] The other problem with ECMO in congenital diaphragmatic hernia is the difficulty in achieving proper cannula placement.

Results of ECMO in CDH:

The multitude of retrospective studies using ECMO in neonates with CDH have in general reported an improvement in survival rate.[48,49,50] The

problem in evaluating the results is most series are small, retrospective studies and use different selection criteria. Also, during the same period other management strategies were simultaneously introduced and therefore the true impact of ECMO on the survival rate in high risk CDH babies is not established. Survival rates of 38% to 65% have been reported with the use of ECMO in high risk infants with congenital diaphragmatic hernia.

O'Rourke et al in a study reported a 51% mortality rate in severe congenital diaphragmatic hernia between 1984 and 1987 with the use of ECMO.[51] Before the introduction of ECMO, the mortality rate at their institution was 53%. In two studies from Toronto and Boston covering a period of 14 years survival rates of 52% and 53% were reported. In the Toronto series, the survival rate of around 50% has remained unchanged despite the use of high frequency ventilation. In the Boston series, which has used ECMO as a rescue therapy (instead of HFV), the survival rates have only increased in the most recent period (1991-1994) with the use of pressure limited, permissive hypercapnia ventilation. There is a broad opinion that ECMO does improve the outcome in congenital diaphragmatic hernia. The other important issue with the use of ECMO is the morbidity and long term complications of the procedure. The long

term effects associated with the use of ECMO is detailed later.

The present view is in favour of late surgical repair and the use of ECMO in congenital diaphragmatic hernia is beginning to decrease, either because of disillusionment or because of the success of alternative therapies. At the present time, ECMO in CDH should be reserved for infants with severe PPHN who fail to respond to the alternative modalities available.

High Frequency Ventilation:

High frequency positive pressure ventilation (HFPPV) is defined as the use of positive pressure ventilation with respiratory rates in the range of 100-150 breaths/min. The airway pressure is kept less than 30 cm H_20 to reduce barotrauma whereas the rate is increased to improve gas exchange and decrease $PaCO_{2.}$ High frequency ventilation (HFV) is different from HFPPV, and uses rates in excess of 1 / second with tidal volumes lesser than dead space ventilation. The two modes of HFV in use presently are: (i) High frequency oscillation ventilation (HFOV) - HFOV uses a piston with an oscillating diaphragm which produces a high frequency (5-15 Hz) sine wave which is applied to the airway via a T-piece system. Expiration is active in this system and does not rely on lung recoil, (ii) High frequency jet ventilation (HFJV) - This system uses a

high pressure gas, source with flow interruption which delivers small tidal volumes via a catheter in the airway at frequencies of 1-5 Hz. Expiration is passive in this system.

HFOV has demonstrated the ability to reduce $PaCO_2$ and induce alkalosis in the presence of significant pulmonary hypoplasia. They have found a rapid reduction in the $PaCO_2$ whereas the improvement in PaO_2 was temporary and not sustained beyond 24 hours. The initial improvement in the PaO_2 is probably due to the improvement in pH and a decrease in the right to left ductal shunting. There were 5 survivors in this group of infants where the expected mortality was 100%. The results of other series have also reported a reduction in the $PaCO_2$ levels with HFV but could not demonstrate an increase in survival.[52, 53] With the present focus on permissive hypercapnia, the use of HFV to achieve reduction in $PaCO_2$ levels is obsolete. Bohn et al have shown that in 87 infants in whom HFOV was used (after failure of conventional ventilation), the overall survival was only 11%. The improvement in $PaCO_2$ and PaO_2 is seen consistently but is frequently unsustained.

Surfactant Therapy:

The role of surfactant therapy in neonates with hyaline membrane disease has been established. The surface tension forces at the alveolar

level are reduced with the administration of surfactant, which reduces the tendency for alveolar collapse and improves ventilation at lower peak airway pressures. Studies have documented a relative surfactant deficiency in CDH patients based on a low lecithin/ sphingomyelin ratio seen during fetal life. Click et al have shown in the surgically created lamb model of CDH that there is a reduced level of phosphatidyl choline and increased protein content in lung lavage fluid.[10] Proteins within the alveoli are known to inhibit surfactant activity and this is in addition to the insufficient surfactant production. In a separate study, Click and coworkers in a series of controlled animal studies, administered exogenous bovine surfactant into the endotracheal tube immediately after delivery.[54] They showed a significant improvement in functional residual capacity, lung compliance and PaO_2 within 30 minutes of surfactant administration as compared with control animals. They also demonstrated in a later study that this improvement was sustained till 4 hours post administration and there was an increase in pulmonary blood flow. Suen et al showed that steroid therapy enhanced the endogenous surfactant production in the Nitrofen induced rat model of congenital diaphragmatic hernia.

Inhaled Nitric Oxide (INO) Administration:

Nitric oxide is an endogenous molecule that induces production of CGMP and causes relaxation of vascular smooth muscle. NO has been identified as the endothelial- derived relaxing factor. The role of NO as a highly selective pulmonary vasodilator led to its use in the treatment of PPHN. Animal studies have shown that endogenous NO modulates the pulmonary vascular tone in the fetus during late gestation. The pulmonary vasodilatation produced by oxygen and lung expansion in the immediate newborn period is also believed to be secondary to endogenous NO. Initial studies have demonstrated that administration of INO to full term infants with PPHN resulted in an increase in SaO_2 in almost all patients.[57 58 59]

A prospective, randomized, multicentre study has been performed to evaluate INO therapy in fullterm neonates with respiratory insufficiency (Kinsella et al). This study has compared the efficacy of HFOV with INO in patients who demonstrated a PaO_2 of < 60 mmHg on FiO_2 1.0. Only 10% of the 33 patients with CDH responded to HFOV, 10% to INO and 5% to the combination of both HFOV and INO. The results of INO therapy have been inconsistent and most reported trials are small uncontrolled series. Inhaled NO can decrease the right to left shunting in

infants who have PPHN and improve oxygenation but will not alter the outcome in patients with severe pulmonary hypoplasia. Karamanoukian et al evaluated the efficacy of INO (80 ppm dose) in 8 neonates with CDH.[58] No improvement was noted in the postductal PaO_2. However, oxygenation was improved when INO was administered after decannulation from ECMO. Shah et al demonstrated a temporary increase in postductal PaO_2 in response to 5-10 ppm INO therapy.[59] Administration of surfactant and / or perfluorocarbon may enhance the effect of INO in patients with CDH. Karamanoukian has shown that newborn lambs receiving surfactant and INO had a higher pH and PaO_2 and lower $PaCO_2$ than control lambs who received only INO.[61] The other problem with the use of INO besides the inconsistent response has been the rebound hypertension which is seen after discontinuation. Patients who are NO dependent should have the gas continued during surgical repair.

The other approach has been the attempt to enhance the effect of the endogenous NO. NO produces vasodilatation via CGMP which in turn is inactivated by CGMP phosphodiesterase. Two phosphodiesterase inhibitors, dipyridamole and zaprinast have been used in animal studies. These agents can augment the effect of INO in neonates with CDH and

also reduce the INO dose required. A reduced INO dose can decrease the incidence of rebound hypertension after discontinuation of therapy.

Liquid Ventilation [35]:

The discovery of flurocarbons led to the feasibility of liquid ventilation. Liquid ventilation was propounded because liquid spreads more uniformly in the lung and the liquid filled alveoli have much diminished surface tension forces. Perfluorocarbons are clear, colourless and odourless fluids which have the following properties-(i) Chemically and physiologically inert, (ii) Greater solubility for respiratory gases than blood (50 ml 0_2/dl), (iii) Low surface tension (19 dynes/cm), (iv) Dense fluid (1.9 g/ml), (v) Low vapor pressure hence eliminated by vaporization, (vi) Can be made radio-opaque by adding a terminal bromide molecule.

Two methods of perfluorocarbon assisted ventilation have been described-

i. Total liquid ventilation (TLV)-The lung is filled to total lung capacity with perfluoro carbon. Because of the high viscosity of the fluid, the perfluoro carbon has to be 'expired' in and out of the lung and oxygenated. This requires very complex circuitry equipment and

hence has limited clinical use.

ii. Partial liquid ventilation (PL V) - This technique uses gas ventilation with conventional settings with the lungs filled with a volume of perfluoro carban equal to the functional residual capacity (15 ml / kg).

Major et al evaluated gas exchange in 8 full term newborn lambs with surgically created CDH – four underwent conventional mechanical ventilation and four underwent PLV.[61] The pH, pulmonary compliance and $PaCO_2$ showed improvement in the PLV group. Pranikoff et al used PLV in four infants with CDH on ECMO and noted improved PaO_2 and pulmonary compliance.[62] Of these 4 patients, two survived and one patient developed pulmonary hemorrhage. Currently trials are on to evaluate the safety and efficacy of PLV with perfluorocarbons in newborns with CDH who demonstrate an oxygenation index between 10 and 30. Fauza et al have shown an increase in alveolar number and size in lambs subjected to postnatal lung distension wth perfluorocarbon.[63]

Intravenous Pulmonary Vasodilators:

The 1970's saw a deluge of pulmonary vasodilators being used for the treatment of pulmonary hypertension associated with CDH. However,

none of the agents were selective pulmonary vasodilators, and all produced simultaneous systemic hypotension. This exacerbates the extrapulmonary right to left shunt and worsens the hypoxaemia. Intravenous pulmonary vasodilators may also cause ventilation- perfusion mismatch. Tolazoline is an alpha-blocker and also releases histamine from tissues to induce vasodilatation, Tolazoline also has chohnomimetic properties and inhibits synthesis of the vasoconstrictor thromboxane A2. The side effects of Tolazoline (oliguria, hypotension, gastrointestinal bleed, thrombocytopenia, seizures) and its unpredictable effect led to the discontinuation of its use. Prostacyclin can cause a lowering of the A-aDO_2 and OI values after infusion in high risk CDH patients with PPHN, but does not alter the overall outcome.[14] The mean arterial blood pressure is not effected, but prostacyclin causes an increase in the bleeding time. Prostaglandin D2 (PGD2) causes a variable decrease in the pulmonary artery pressure and improves oxygenation in PPHN. Systemic hypotension is seen with PGD2 infusion. Various other vasodilators like nitroprusside, chlorpromazine, isoprenaline, glucagon and halothane have been used but the results are inconsistent and the associated problems offset the advantages.

Lung Transplantation:

The experience with lung transplantation in newborn infants is limited; Starnes et al have reported their experience in 5 infants with 3 survivors[65] The use of reduced size lung grafts from living donors is being investigated. The left lower lobe or the right middle lobe can be used. In patients with CDH, an ipsilateral lung transplant can be used temporarily till the contralateral lung matures and PPHN resolves. The transplanted lung can then be excised and immunosuppression discontinued. There is a single report of one patient with CDH who has undergone lung transplantation.[66] This patient with right CDH, received a right middle lobe from a newborn donor and is doing well on follow up at 2 years of age. Lung transplantation is a promising option for newborns with CDH and severe pulmonary hypoplasia. The known complications of lung transplantation include delayed somatic growth, risk of infection, lymphoproliferative diseases and obliterative bronchiolitis.

Prognostic Factors:

(A) Antenatal

1. Polyhydramnios - Kitagawa et al suggested that infants born to mothers with polyhydramnios have a 11% chance of survival as compared with the 55% survival without polyhydramnios.

2. Gestational age less than 25 weeks at diagnosis has been shown to be a poor prognostic factor.[67]

3. Presence of an intrathoracic stomach – The presence of stomach in the chest on antenatal ultrasonography is associated with 71% mortality as compared to 7% mortality if the stomach was below the diaphragm.[68]

4. Small lung to thoracic transverse ratio (L/T ratio) - The L/T ratio predicts the degree of hypoplasia and predicts outcome.

5. Low left ventricular mass - The presence of a cardiac anomaly (seen in 30% fetuses) was shown to be universally fatal in a large series from Boston. Fetal cardiac ventricular disproportion in the form of reduced left / right ventricular size before 24 weeks gestation has an associated mortality of nearly 100%.

6. Contralateral lung to head circumference ratio - The ratio of the right lung area (two dimensional area at the level of the atria) to head circumference (LHR) predicts outcome. In a series of 55 cases, a LHR ratio of less than 0.6 was predictive of 100% mortality. LHR > 1.35 predicted survival independent of liver position or gestational age at time of diagnosis.

7. Chromosomal anomalies and other associated anomalies are associated with poor prognosis.[70] Puri et al have shown that 100% of stillborns with CDH have major associated anomalies,[3]

Harrison et al have suggested that cases diagnosed before 25 weeks gestation with a small LHR and liver herniation into the chest are associated with the poorest prognosis.[67]

(B) Postnatal Prognostic Factors

(1) Blood gases

i. pH < 7.2 and $PaCO_2$ > 50 mmHg is associated with poor prognosis (in the era before the concept of permissive hypercapnia was introduced).

ii. Pulmonary function studies - Antunes et al evaluated the oxygen index and functional residual capacity in CDH survivors.[69] All nonsurviving infants had preoperative functional residual capacity of less than 9 ml / kg. On the other hand, only 2 of 15 survivors had preoperative functional residual capacity of less than 9 ml/kg.

iii. Echocardiographic findings - Studies have shown that left ventricular mass of less than 2g/kg is predictive of 100% mortality. Also it has been shown that in patients operated on ECMO if the cardiac angle

(normal value of 45^{o}) did not return to normal after repair, all such neonates did not survive. Dilatation of the pulmonary artery relative to aortic narrowing suggests an impaired left ventricular output. Patients with this abnormal feature died despite ECMO support and surgical intervention.

iv. Side of defect - Right sided defects has a poorer prognosis. Bilateral hernias also have a poor prognosis.

v. Timing of presentation - The mortality rate in infants presenting within 6 hours of birth is high as compared to the almost negligible mortality seen in infants presenting after the first day of life.

Fetal Diagnosis and Therapy:

Antenatal diagnosis of CDH by ultrasonography was described towards the end of the 1970's. This led to the need for identifying reliable prognostic factors antenatally so that proper counselling could be given.

1. Maternal serum alpha-fetoprotein (MS-AFP):

MS – AFP can be detected from the 12^{th} week of gestation and it steadily increases in concentration to peak between week 30-32 low levels of MS – AFP have been identified in CDH, trisomy 21 and trisomy 18.

2. Ultrasonography:

The gold standard for antenatal diagnosis of CDH is now a level 3 ultrasound examination. The features which indicate CDH in a fetus are :

(i) Polyhydramnios (seen in 80% of fetuses with CDH).

(ii) Absent or intrathoracic stomach bubble.

(iii) Mediastinal shift.

(iv) Presence of abdominal viscera in a transverse scan at the level of a four chamber view of the heart.

(v) In extreme cases, fetal hydrops.

(vi) Associated anomalies like cardiac defects, neural tube defects.

False positive diagnosis of CDH by ultrasound is rare.

3. Chromosomal analysis:

The chromosomal anomalies associated with CDH include trisomy 18 and 13, 3p microdeletion and 12p tetrasomy. Amniocentesis is the usual method of obtaining fetal cells for chromosomal analysis.

Fetal Therapy:

Although there have been rapid innovations in the postnatal management of CDH, the 'hidden mortality' is an important factor in the postnatal outcome of antenatallv diagnosed fetuses with CDH. In a prospective study, Harrison et al showed that in 83 fetuses with isolated

CDH diagnosed before 25 weeks of gestation, 58% died despite care in a centre equipped with ECMO.[67] Experimental studies have shown that after creation of CDH early in gestation and subsequent repair, the lung volume improved and the pulmonary arteriolar muscle hyperplasia resolved.[9,71] The selection criteria for fetuses for prenatal therapy is based on identifying antenatal ultrasonographic features associated with poor outcome - diagnosis before 24 weeks gestation, fetal cardiac ventricular disproportion, contralateral lung to head circumference ratio and liver herniation.

The options available for in-utero intervention in CDH are [72-73].

1. Complete in utero repair - This technique is applicable for fetuses that do not have liver herniation. The 'two-step' technique described by Harnson et al is used.[74] A thoractomy and a sub costral incision are used to reduce the viscera by a ‘push – pull’ technique. The diaphragm is reconstructed using a Goretex patch and the abdominal cavity is enlarged by a similar Goretex patch. Color doppler imaging is used to define the position of the portal venous structures and the course of the umbilical vein which would indicate whether the liver is herniated or not. Direct repair of fetal CDH is possible and has achieved favourable result.[75] It has been shown that the overall mortality in 'liver-down' CDH is low in both the fetal surgery group

as well as the postnatal treatment group. These patients have a more benign course, regardless of treatment and therefore the maternal risks associated with fetal surgery do not justify in-utero intervention.

2. PLUG Technique - The early attempts at reduction of CDH in fetuses with liver herniation showed that open surgery was uniformly unsuccessful. The reduction of the liver back into the abdomen results in acute obstruction of the ductus venosus and the reduced umbilical venous return led to fetal death. Experimental work has shown that fetal tracheal obstruction can correct the pulmonary hypoplasia associated with CDH.[76] Tracheal obstruction expands the fetal lung by preventing drainage of fetal lung fluid and this in turn pushes the viscera back into the abdomen. Harrison et al developed a procedure of temporary tracheal occlusion and called it the PLUG (Plug the Lung Until it Grows).[77] The EXIT procedure (Exutero intrapartum tracheoplasty) was developed to maintain the fetoplacental circulation while the fetal airway was secured. The fetal head and shoulders are delivered and the umbilical cord is maintained. The external clip is removed with bronchoscopic monitoring and the trachea is evaluated for any possible damage. The baby is intubated and surfactant is administered. Once the airway is secured, the umbilical cord is divided. Though this technique of

PLUG followed by EXIT is promising, complications encountered during the evolution of this strategy have limited survival. There is also evidence that tracheal obstruction may delay or depress pulmonary maturation and surfactant production.

3. Fetendo plug[78] – Video assisted fetal endoscopy (fetendo) avoids the hysterotomy needed for fetal exposure and decreases the risks of preterm labor. Harrison et al has described a two-trocar videofetoscopic PLUG technique.

Results

(A) Mortality

In 1946, Gross reported a series of 63 infants with CDH with 55 survivors. Since these, despite the advances in pediatric surgery, anaesthesia and intensive care, no subsequent series has achieved such a high survival. The law of natural selection was responsible for this aberrant fact, because the most severely asphyxiated infants died before being referred for treatment.

The mortality in newborn infants with CDH presenting in the first 6 hours of life remains between 40-50. Infants who have symptoms after 24 hours of life have nearly a 100% chance of survival. In an analysis of 30 series documenting outcome in 2024 new borns with CDH, Langham

et al reported that there were 1244 survivors, an overall survival of 0.60 (95% confidence intervals, 0.55 – 0.65%).[79] There is a wide variation in the presentation, associated anomalies and treatment modalities used in these series. The mortality is higher if CDH is diagnosed antenatally and most series report figures between 56-86%.

Long Term Results:

A. Cardiopulmonary consequences

In the acute phase associated cardiac anomalies, left ventricular hypoplasia and cardiac 'stun' associated with ECMO effect outcome. However, the long term cardiac effects in infants with isolated CDH are minimal.

Pulmonary consequences have been investigated in detail, Chartrath et al in 1971 described X-ray and pulmonary function tests in survivors of CDH.[80] Three of 5 infants had normal X-rays and two demonstrated emphysematous changes. The FEV1 and FVC were significantly lower for CDH patients. Wohl et al reported normal FEV1, FVC and other parameters in 19 survivors of CDH aged 6-18 years.[81] However, pulmonary blood flow was reduced to the ipsilateral side in all the cases. It has been shown in ventilation-perfusion radionuclide studies that

ventilation is essentially equal between lungs but the perfusion is more to the contralateral side (ipsilateral is approximately half of the contralateral lung). Despite this most studies report normal exercise tolerance in survivors.[82]

The study by Vacanti et al discussed the pulmonary follow-up in CDH survivors treated with ECMO and reported 65% mortality secondary to complications of chronic ventilator support. The study by Nagaya et al examined two groups of CDH survivors.[83] Patients who did not require ECMO support had matched ventilation and perfusion in both the lungs. The neonates who had compromised gas exchange and required ECMO had significantly decreased ipsilateral ventilation and perfusion. Over a period of 44 months, ipsilateral lung ventilation increased to 88% of the contralateral lung while the perfusion remained nearly fixed. Koumbourlis et al reported that in survivors of CDH there is a catch up growth during the first 2 years with an increase in the FRC.[84] This was associated with improved lung compliance. The associated skeletal abnormalities seen in CDH patients also compromise lung growth.

The incidence of chronic respiratory problems is also high in CDH survivors, especially in those managed with ECMO. Bernbaum noted

bronchopulmonarv dysplasia in 50% of CDH survivors managed with ECMO.[85] Recurrent respiratory infections and repeated hospitalization for chronic respiratory problems are common early in follow-up but the pulmonary status tends to improve with time.

B. Recurrent herniation:

Recurrent CDH is seen in 2-22% cases and diagnosed by CXR or contrast radiography. Van Meurs et al showed a 22% recurrence rate for primary repair and a 40% recurrence rate for patch repair.

Gastrointestinal problems:

Foregut dysmotility is common in CDH survivors and probably results secondary to extrinsic pressure on the developing esophagus by the herniated viscera. The extrinsic pressure may lead to intrauterine dilatation of the esophagus which can impair motility. Also, the length of the intra abdominal esophagus is short and the angle of His is altered. The perihiatal diaphragm is deficient and can lead to a hiatal hernia. The stomach is frequently nonrotated and this accounts for the associated delayed gastric emptying. Stolar et al were the first to note esophageal functional and anatomical abnormalities in CDH survivors.[87]

The incidence of symptomatic GER varies between 17-26% and upto

36% of CDH patients require operative intervention in follow-up.[85-90] Vanamo et al evaluated 60 survivors of CDH and reported that 18% infants developed early symptoms of GER.[89] At late follow-up (mean- followup of 29.6 years), 63% of the patients still had symptoms suggestive of GER and 54% had histological or endoscopic evidence of esophagitis. The etiology of GER in CDH has been explained by-

It is important to look for GER in survivors of CDH who have failure to thrive, recurrent respiratory problems or gastrointestinal symptoms. Medical management of GER and prokinetic agents for improving gastric emptying should be instituted early.

The incidence of adhesive intestinal obstruction in survivors of CDH is around 10%. Inherent malrotation associated with CDH may also predispose to obstruction.

C. Growth and development

Failure to thrive has been noted in many survivors of CDH. Van Meurs et al reported that 50% of ECMO treated CDH survivors were less than the 5th percentile for weight at 1 and 2 years of age.[86] Most reports show that 30-40% of CDH infants are below the 5th percentile for age after 2 years.[90-92]

MATERIAL AND METHODS

- All cases of congenital diaphragmatic hernia admitted in Govt. Rajaji Hospital, Madurai were studied.
- All cases were given adequate preoperative preparation and those surgically treated were followed up for a period of 1 year and their outcome was noted.
- Those cases which were not eligible for surgical treatment were analyzed and their outcome was noted.

OBSERVATION & RESULTS

Preoperative Work up:

All the 60 new borns that were included in the study were thoroughly evaluated. which included

1. Antenatal History
2. Birth History
3. Immediate Postnatal period
4. Through physical examination
5. SpO_2 status
6. Echo Cardiogram
7. X-Ray chest
    a. AP view
    b. Lateral view
8. Ventilator support

After examination and initial evaluation all the new borns were nursed in the neonatal intensive care unit. Children with severe respiratory distress were electively ventilated. T. Sildenafil was used at the dose of 1mg/kg every 8^{th} hourly to reduce the persistent respiratory distress. Children who became stable were extubated and subjected to surgery.

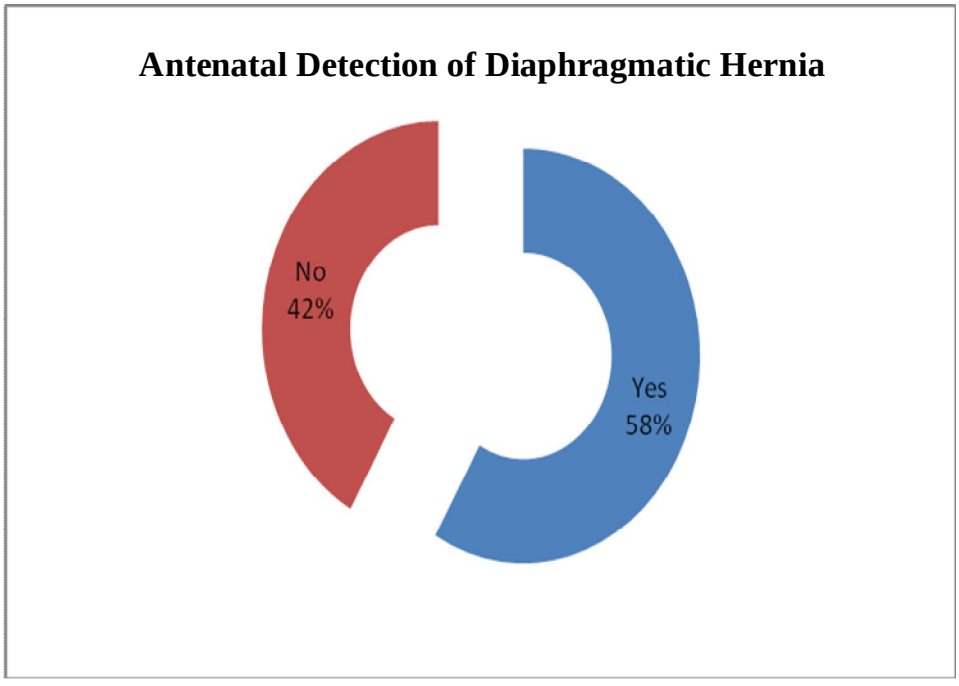
Echo cardiogram and severe distress were used as criteria for ventilatory support.

1. **Antenatal History**

All babies that were born in the hospital & referred from outside, a through antenatal history was obtained for.

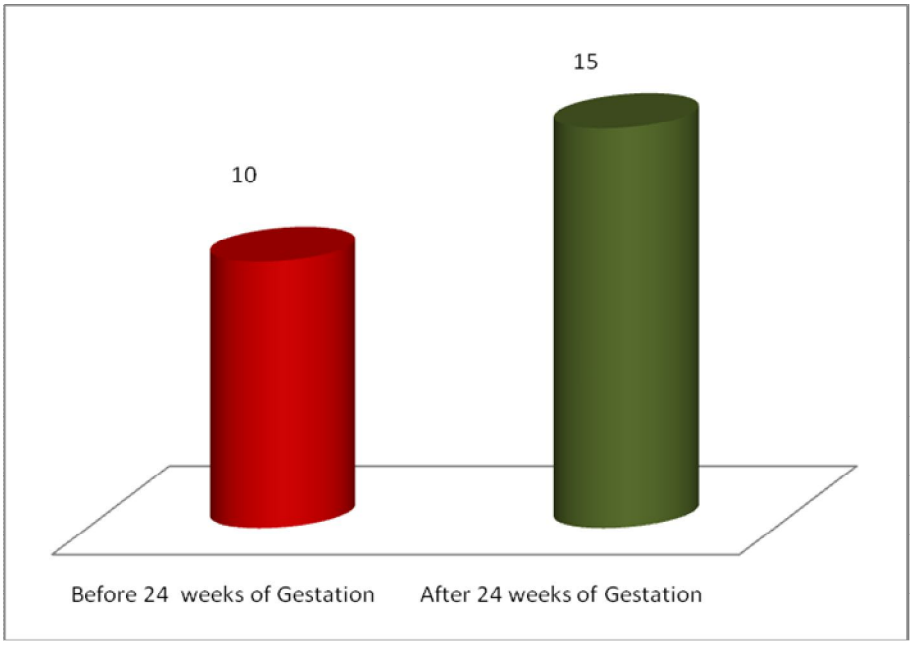
a. Antenatal Detection of Diaphragmatic Hernia

Yes	35
No	25



a. Week of Pregnancy at which Defect was Deducted

Before 24 weeks of Gestation	10
After 24 weeks of Gestation	15



2. Birth History

Normal Vaginal Delivery	35
LSCS	25

Indication for LSCS

Repeat Section	17
Maternal Cause / Fetal Distress	8

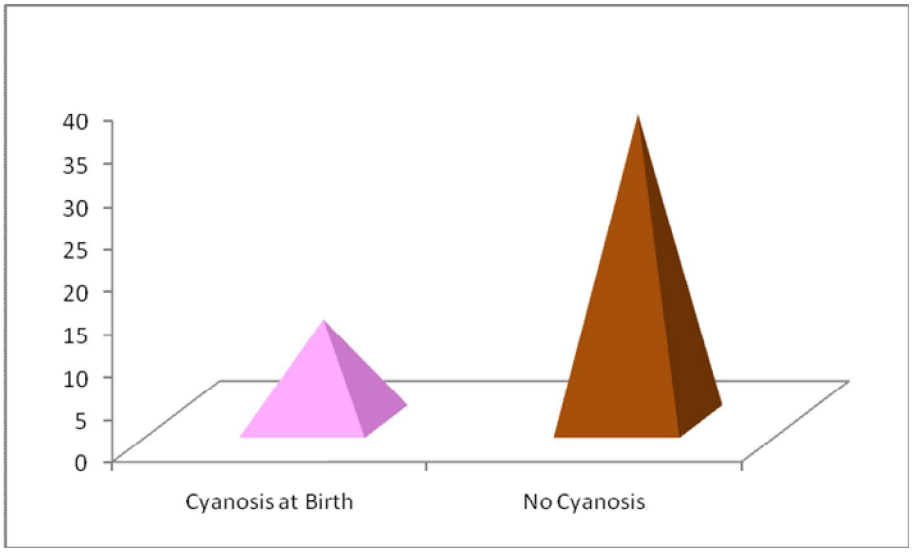
3. Immediate Postnatal Period:

APGAR scores of all babies born in the hospital were noted. Any cyanosis at birth was identified which played an important role in the outcome of the surgery.

a.Cyanosis at Birth

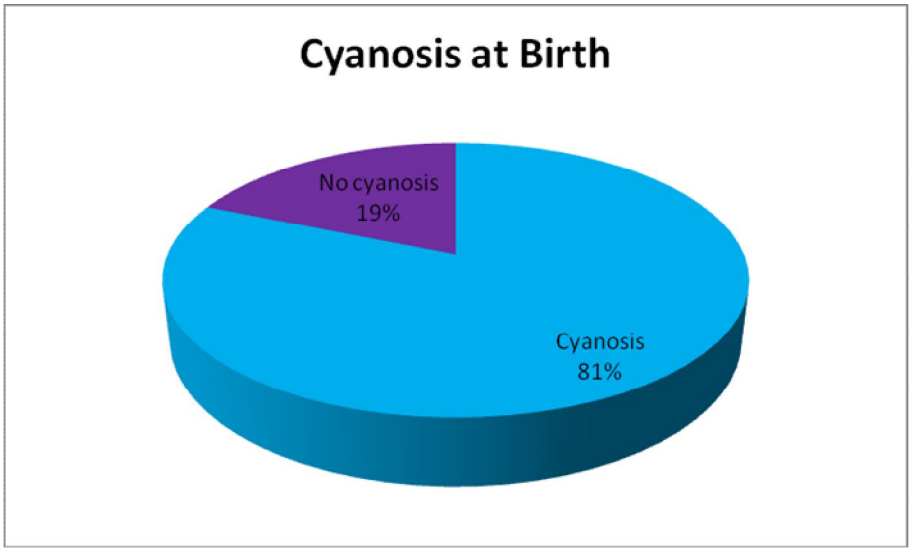
Hospital Birth	
Cyanosis at Birth	12
No Cyanosis	36

n = 44



Delivered Outside	
Cyanosis	13
No cyanosis	3

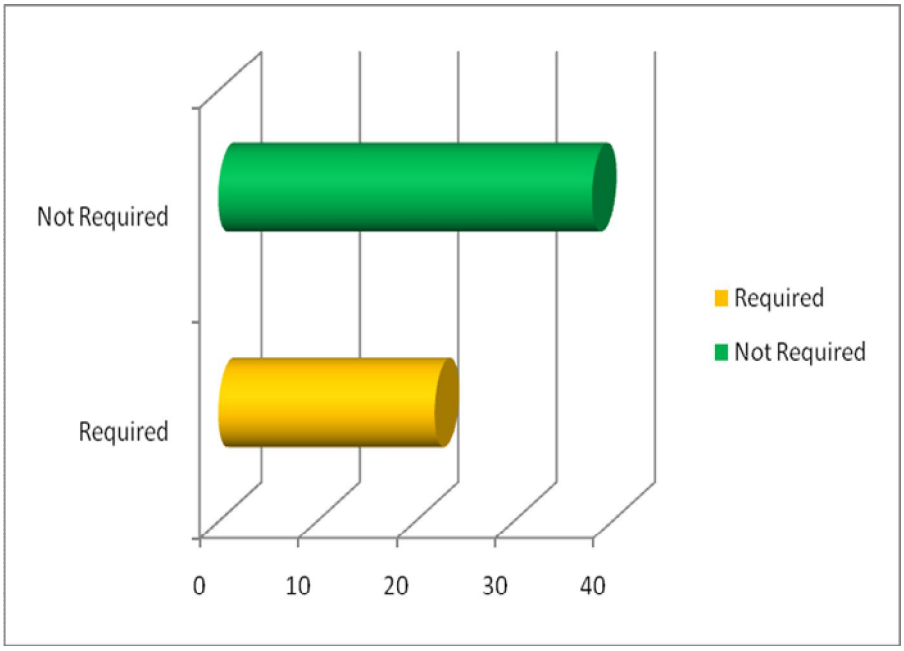
n =16



4.Ventilatory Support:

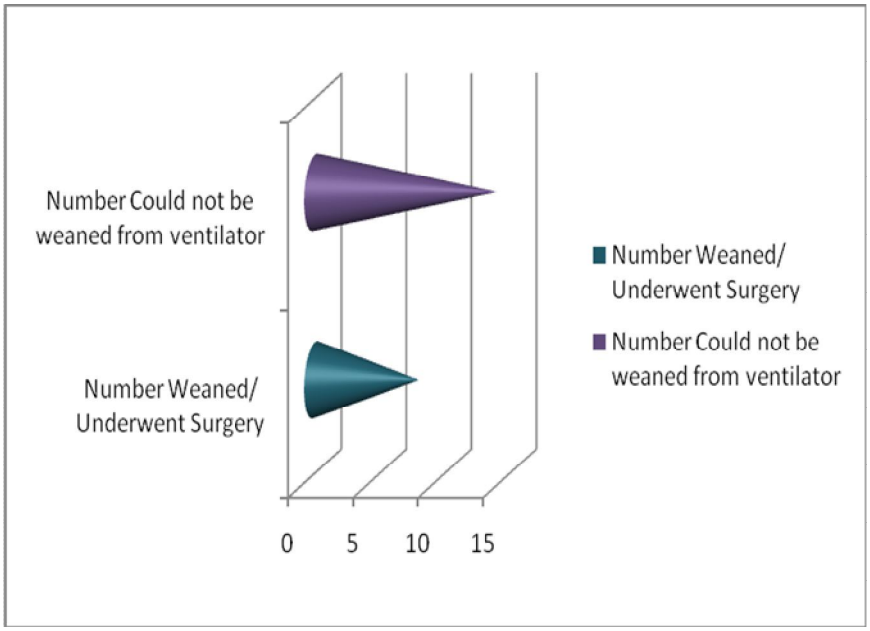
Required	Not Required
22	38

Ventilatory Support:



n = 60

Number Weaned/ Underwent Surgery	Number Could not be weaned from ventilator
8	14



n = 22

5.Physical Examination:

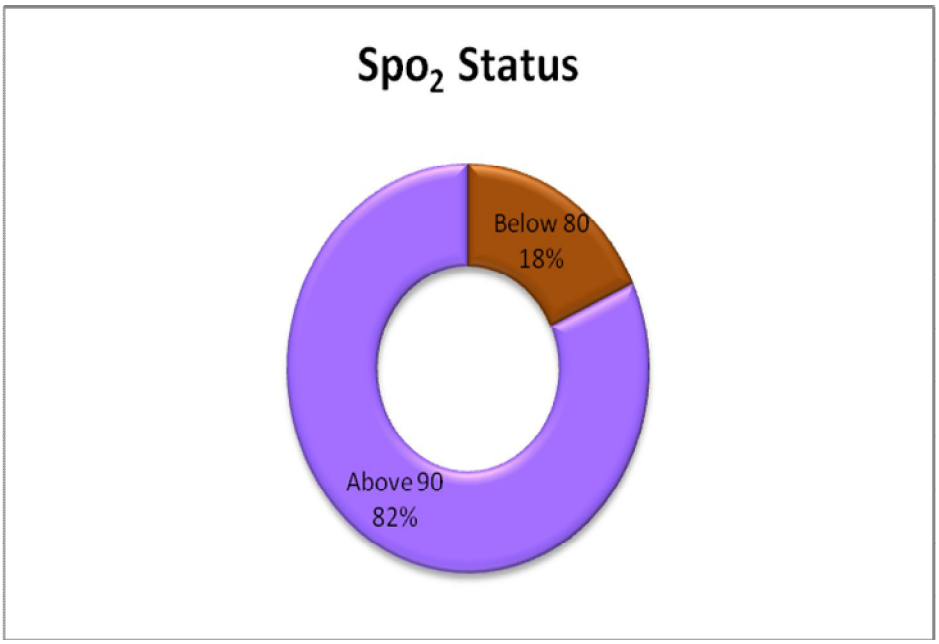
No obvious skeletal defects or any other defects were seen in our group.

6.SpO_2 Status:

All the 44 babies born in Govt. Rajaji Hospital, Madurai were evaluated for Spo_2 using pulseoxymeter.

The 16 out born babies could not be evaluated.

SpO_2 Status		
Below 80%	Above 90%	Total
8	36	44

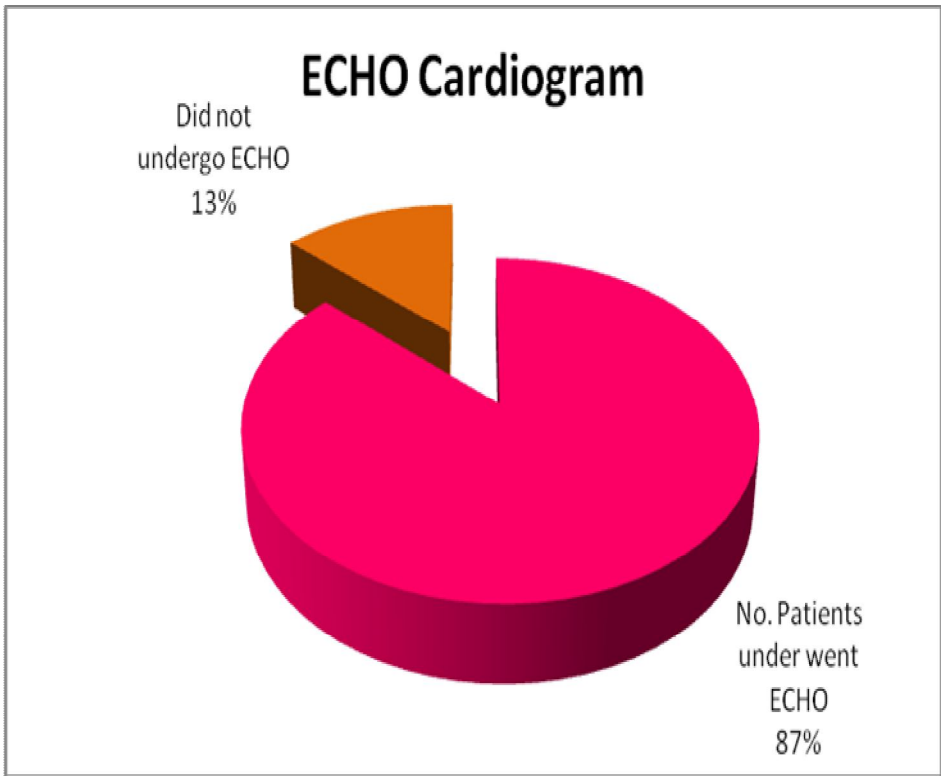


n = 44

7.ECHO Cardiogram:

Since Echo Cardiogram facility was available at a distant location from new born facility eight children were too sick to be shifted to undergo the study.

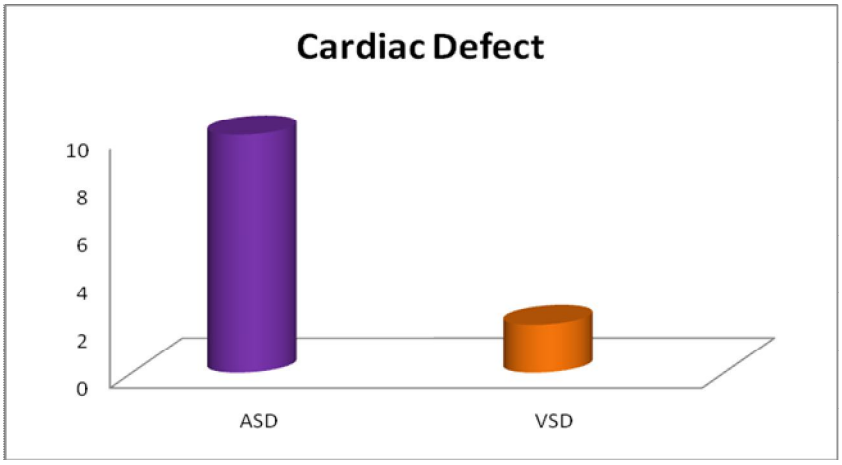
No. Patients under went ECHO	52
Did not undergo ECHO	8



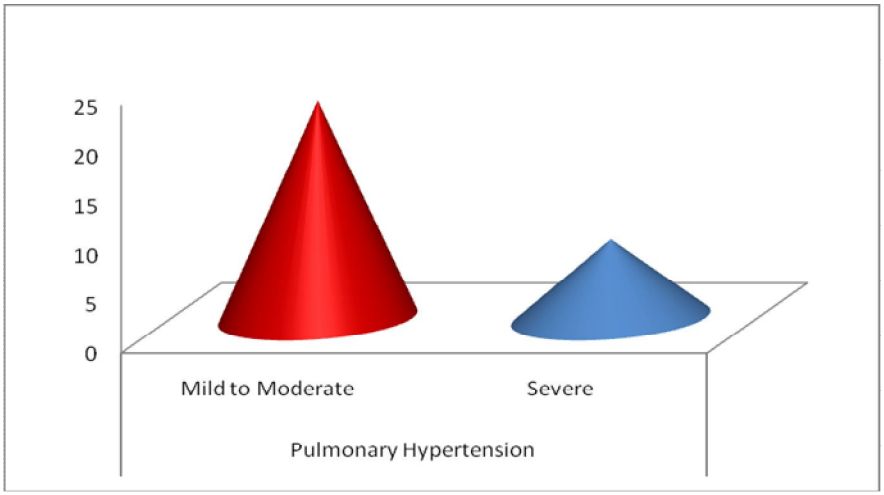
n = 52

Results of ECHO Cardiogram

Cardiac defect			Pulmonary Hypertension	
Present		Absent	Mild to Moderate	Severe
ASD	VSD	40	22	8
10 (small)	2			



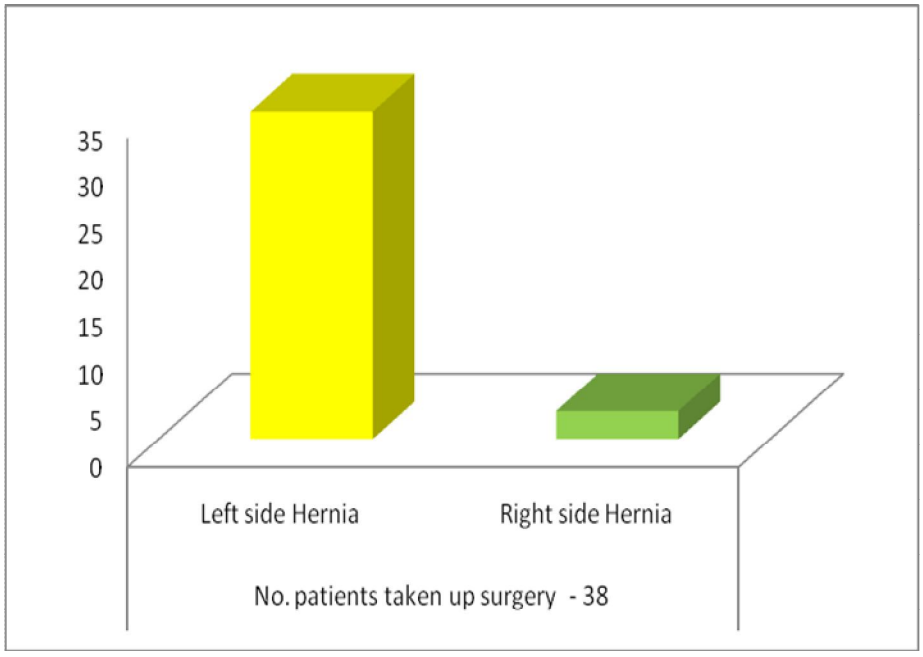
Pulmonary Hypertension



Surgical Procedure:

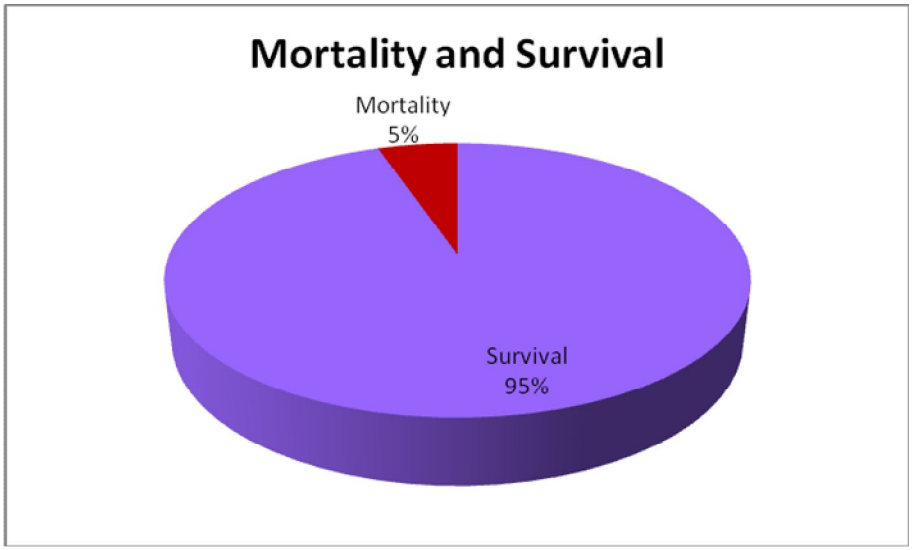
All the patients who were stable after adequate pre operative preparation were taken up for surgery.

No. patients taken up surgery - 38	
Left side Hernia	Right side Hernia
35	3



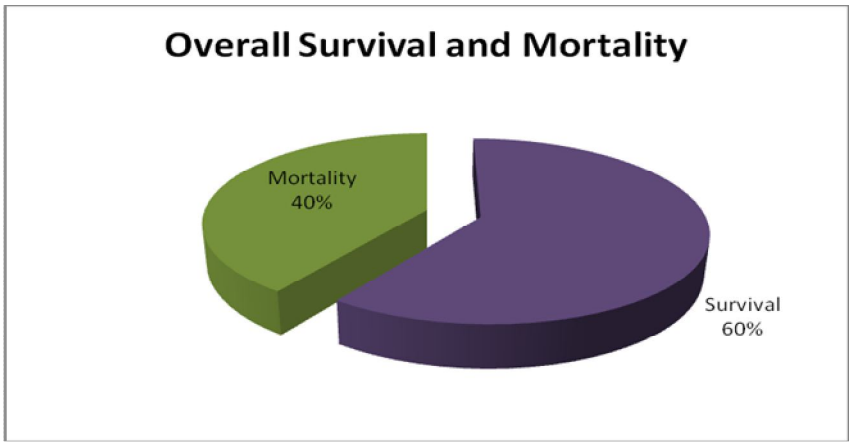
- For all left sided hernias a left side subcostal incision was preferred, in case of right side hernia right postero lateral thorocotomy was done.
- All the left sided hernia that were operated were found to be Bochdalek type.
- No associated malrotation or bowel duplication were seen.
- All the children are being followed up for 1 year post operative period.

Total no.of patients operated - 38	
Survival	Mortality
36	2



Overall Survival and Mortality:

Total No. of patients in the study - 60	
Survival	Mortality
36	24



Total No. of patients - 60			
Deaths - 24			Survived following surgery
Surgery and Death	Ventilated and could not be weaned	Too sick for aggressive resuscitation	
2	16	6	36

Post operative follow up :-

1. 2 patients had post operative adhesive obstruction for which adhesion release was done.
2. 1 patient developed post operative incisional hernia.

DISCUSSION

The results of surgical repair of diaphragmatic hernia depended largely on the preoperative stabilization and type of cases admitted to the hospital.

Late surgical intervention with adequate preoperative surgical preparation has significantly improved the survival rate in patients who under went surgical repair as seen in our study.

Antenatal detection of Congenital Diaphragmatic Hernia helps in delivering such babies at centers that can treat Congenital Diaphragmatic Hernia. Antenatal scan also acts as one of the prognostic indicators in baby that were diagnosed to have congenital diaphragmatic hernia. Antenatal detection before 24 weeks of gestation carries poor prognosis as opposed to those that were diagnosed after 24 weeks and was confirmed by our study.

Use of sildenafil 1mg/kg/8hrs was used in patient who had severe respiratory distress and persistent pulmonary hypertension. Sildenafil therapy was given for 3 – 5 day depending upon their clinical improvement which was also confirmed by Echo Cardiogram before taking up for surgery.

Out of 22 patients who required preoperative ventilation due to severe respiratory compromise, 14 could not been weaned from ventilatory support and eventually succumbed to the disease process. Only 8 patients were weaned from ventilator and taken up surgery.

Those patients who were taken up for surgery were assessed under ASA IV. In our series left side hernias (92%) were more than right sided hernias (8%). Left side sub costal incision was preferred since it was easier to reduce the contents and to look for any associated malrotation and duplication of bowel.

All the left side hernias that we operated were of the Bochdalek type of hernia with spleen and intestine as common contents .Out of 35 babies, 5 had entire bowel, Spleen and stomach inside the left hemi thorax. In all babies diaphragmatic repair was possible with out the use of any prosthetic material. Primary abdominal was closure was possible in all but one baby in which a ventral hernia was created.

In case of right side hernia, right side lateral thorocotomy was preferred. Liver was the primary content which required reduction into the abdomen and primary repair was done. Both in the right and left sided Congenital Diaphragmatic hernia, ICD tube was inserted. Post operative X-ray taken for all patients and checked for adequate repair,

and for the presence of pneumothorax. ICD tube was removed after checking the x-ray on 3rd or 4th postoperative day. Average hospital stay was 8 – 10 days. Only one patient required post operative ventilation and that patient recovered well.

Overall survival rate was 58% which was comparable with other major centers. Of the 38 patients in whom we operated 36 patients survived (95%) and postoperative mortality rate was 5%. Which is considered good when compared to other results.

We consider that most important factor affecting the overall mortality figures in our study has been the admission of an increasing proportion of babies who formerly would have died shortly after birth but are now diagnosed early, resuscitated, intubated and referred, but who finally proved to have insufficient lung tissue for survival.

CONCLUSION

In our institution when newborn were admitted with a prenatal diagnosis of Congenital Diaphragmatic Hernia (CDH)or when CDH babies became symptomatic after birth a careful protocol of respiratory assistance was followed. Immediate postnatal intubation was done; mask ventilation was avoided; whenever ventilators were available small-volume, high frequency and reduced peak pressure mechanical ventilation was started and continued till stabilization of the baby for surgical repair. Pre-ductal and post-ductal percutaneous oxygen saturation measurements helped in the assessment of fitness of the child for surgical intervention. Inotropic drugs were used as necessary and cardiac function and heart anomalies were carefully evaluated. The goal of the preoperative treatments was “stabilization” of the child - $PaO_2 > 40$ mmHg and $PaCO_2 < 60$ mmHg, good myocardial function and adequate renal clearance with reduced or withdrawn inotropic drugs.

Surgical repair of CDH was undertaken only after cardio-respiratory functions were stabilized. A policy of “delayed” surgery coupled with gentle ventilation and was followed.

Congenital Diaphragmatic Hernia (CDH) still continues to show a significant mortality in our institution. This condition continues to remain a significant challenge for obstetricians, pediatric surgeons, neonatologists, and pediatricintensivists.

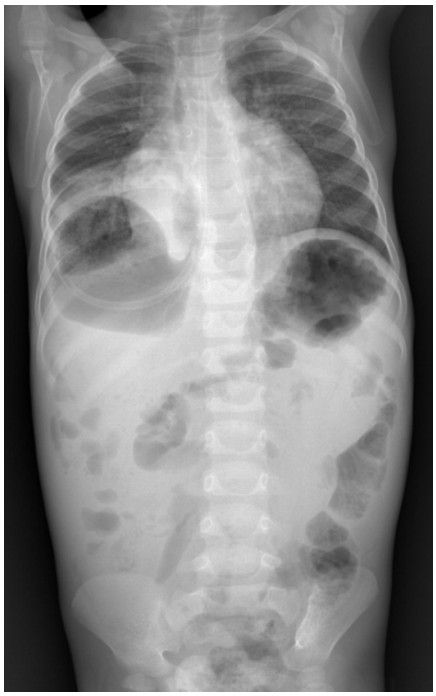


Fig -2.X –ray of Right Side CDH

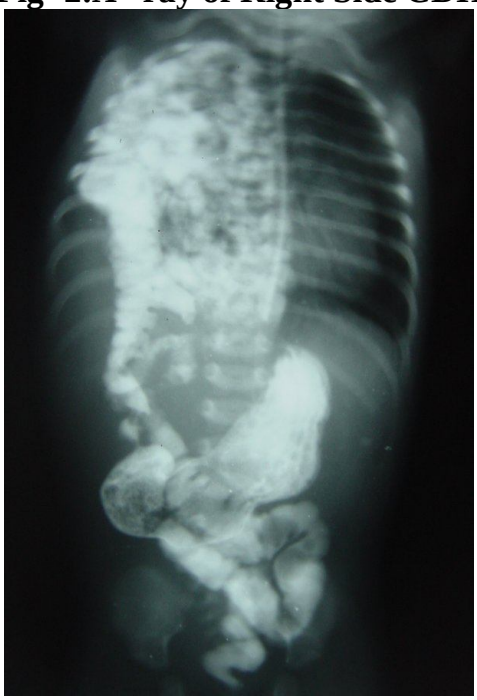


Fig-3. X –ray of Right Side CDH with Contrast

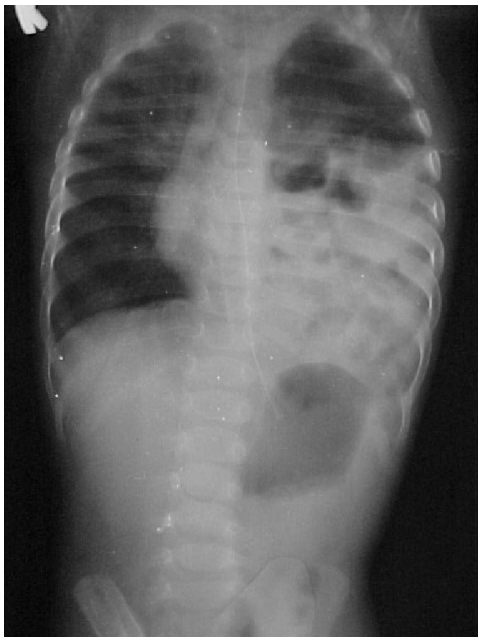


Fig -4. X –ray of Left Side CDH

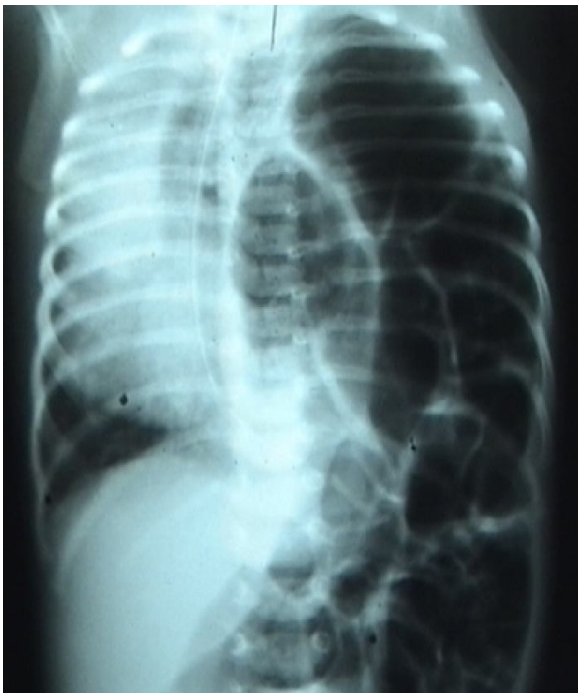


Fig-5. X –ray of Left Side CDH

Fig – 6. Right Side CDH with Liver as Content

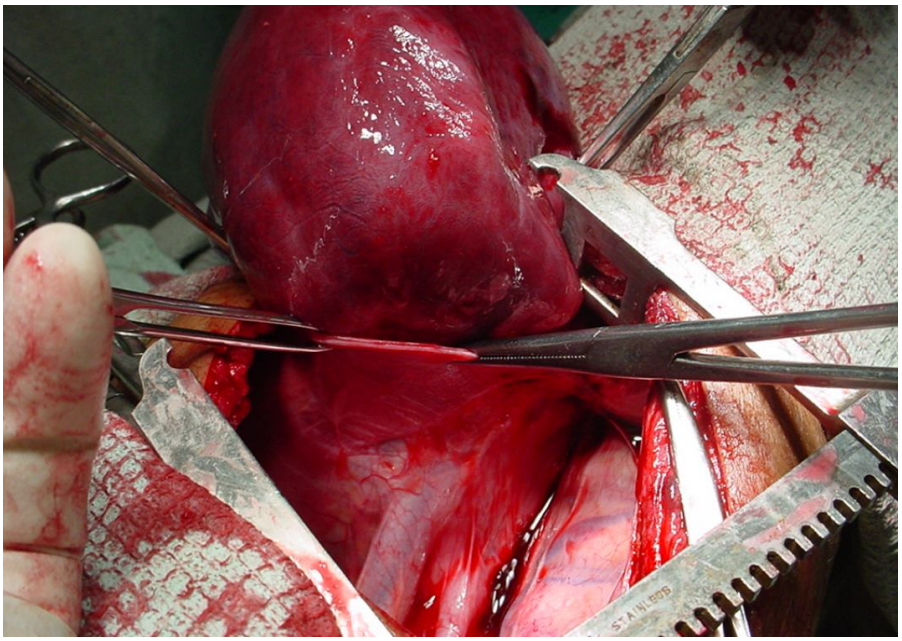


Fig –7. Content Being Reduced

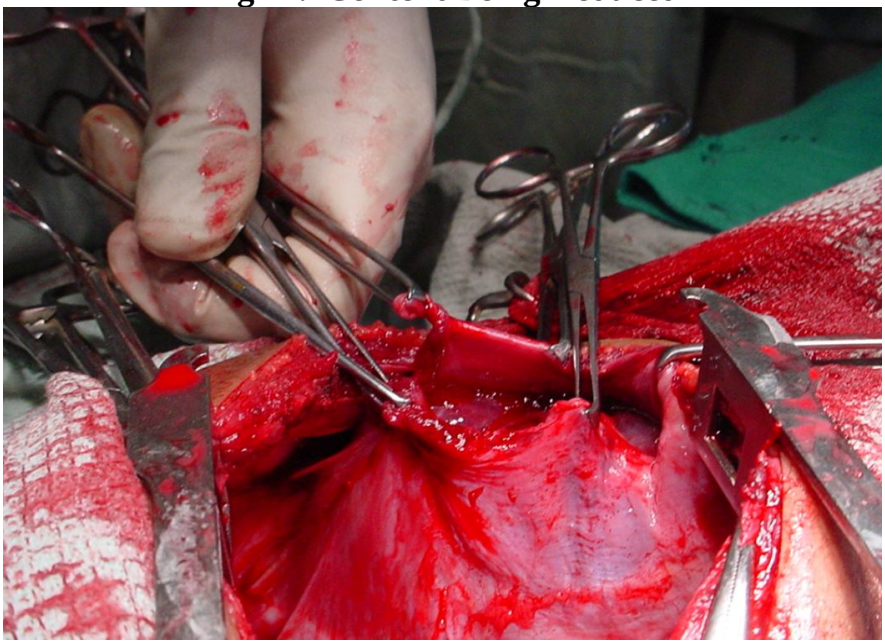


Fig – 8. Content Being Reduced

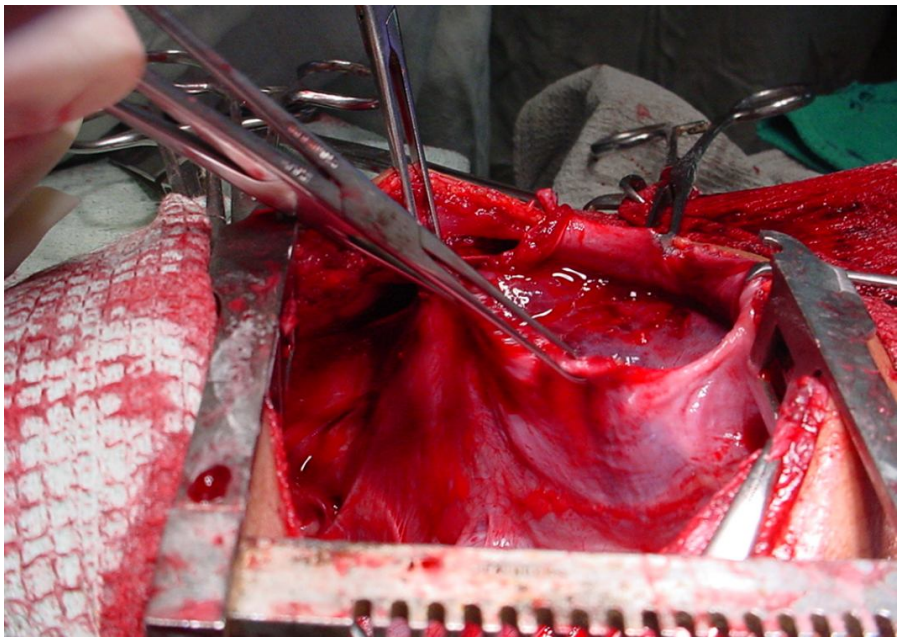


Fig -9. Content Reduced and Repair Being Done

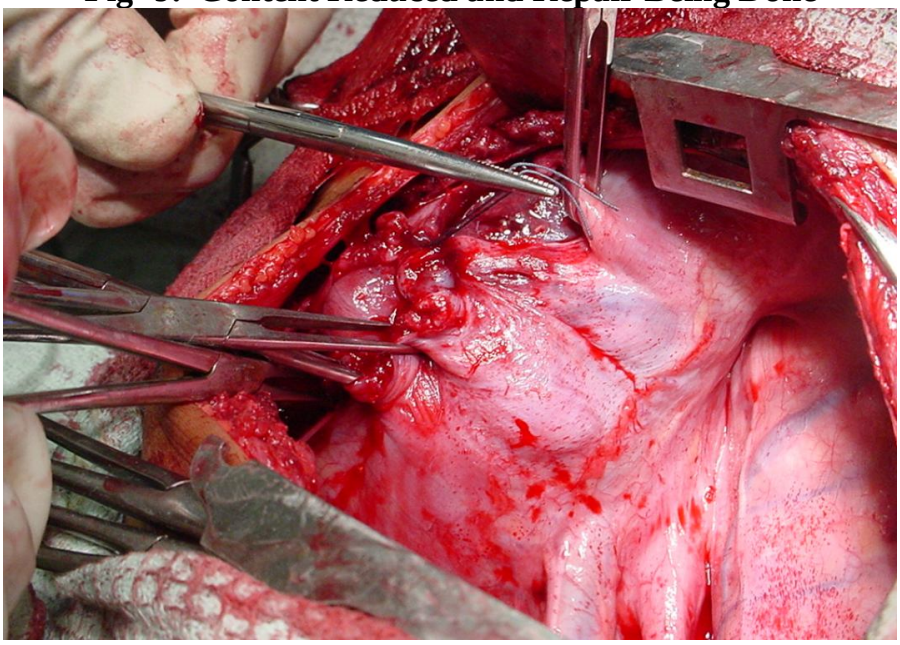


Fig -10. Left Side CDH Defect Being Shown

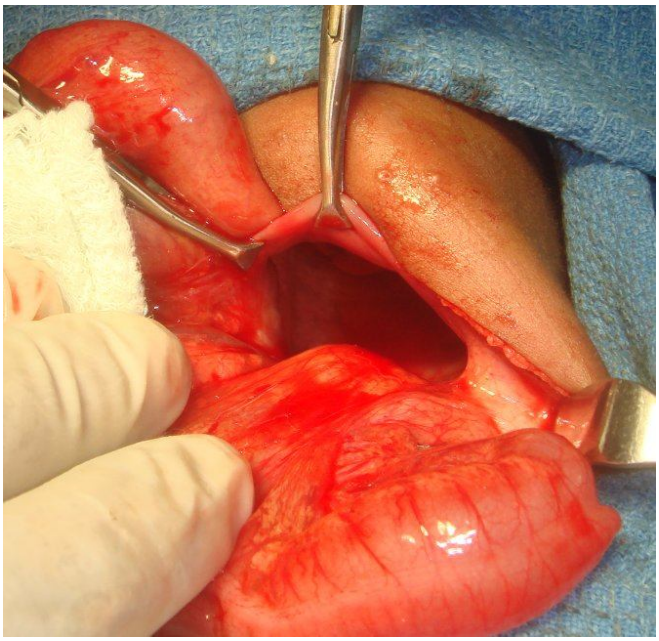


Fig -11. Left Side CDH Defect Showing Hypoplastic Lung

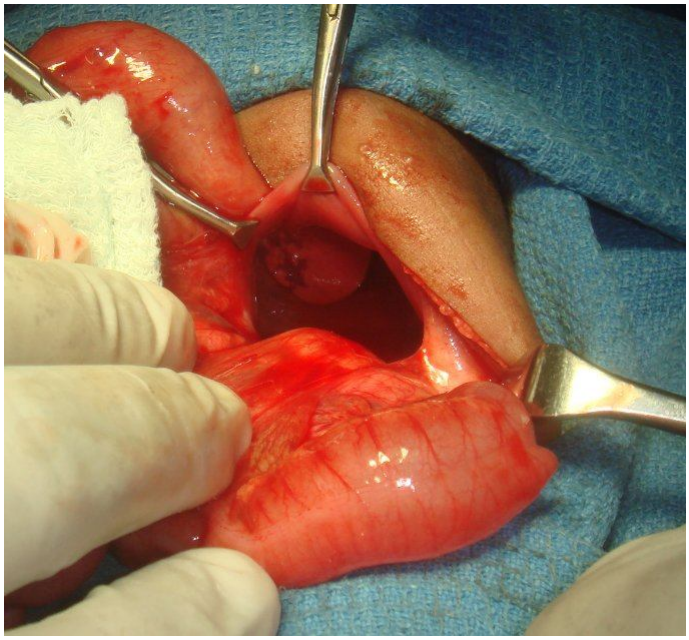


Fig -12. Primary Repair Completed

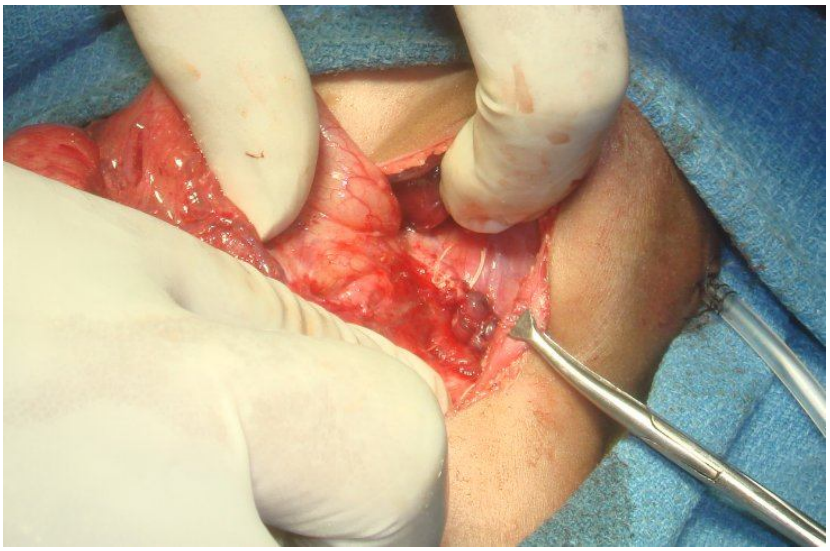


Fig -13. Immediate Post operative Period with ICD Tube

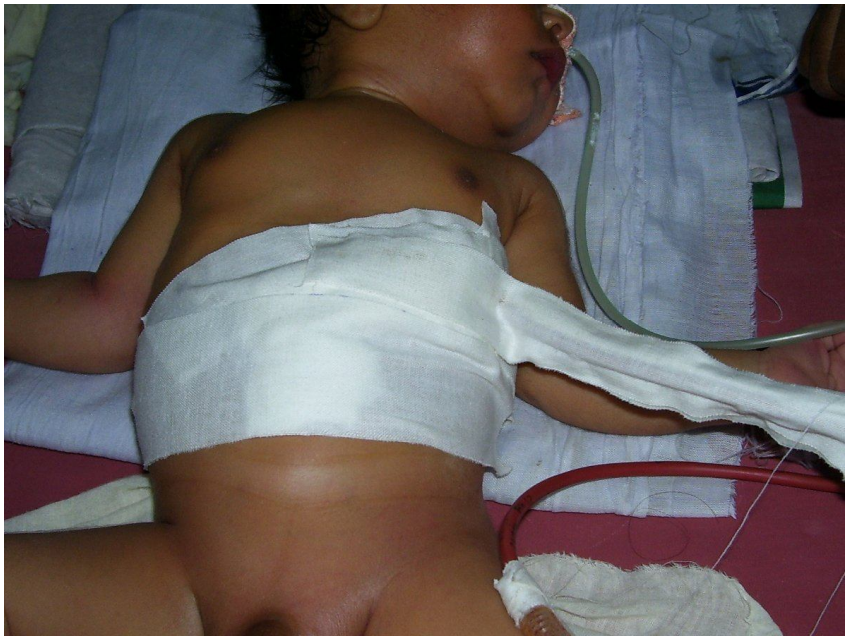
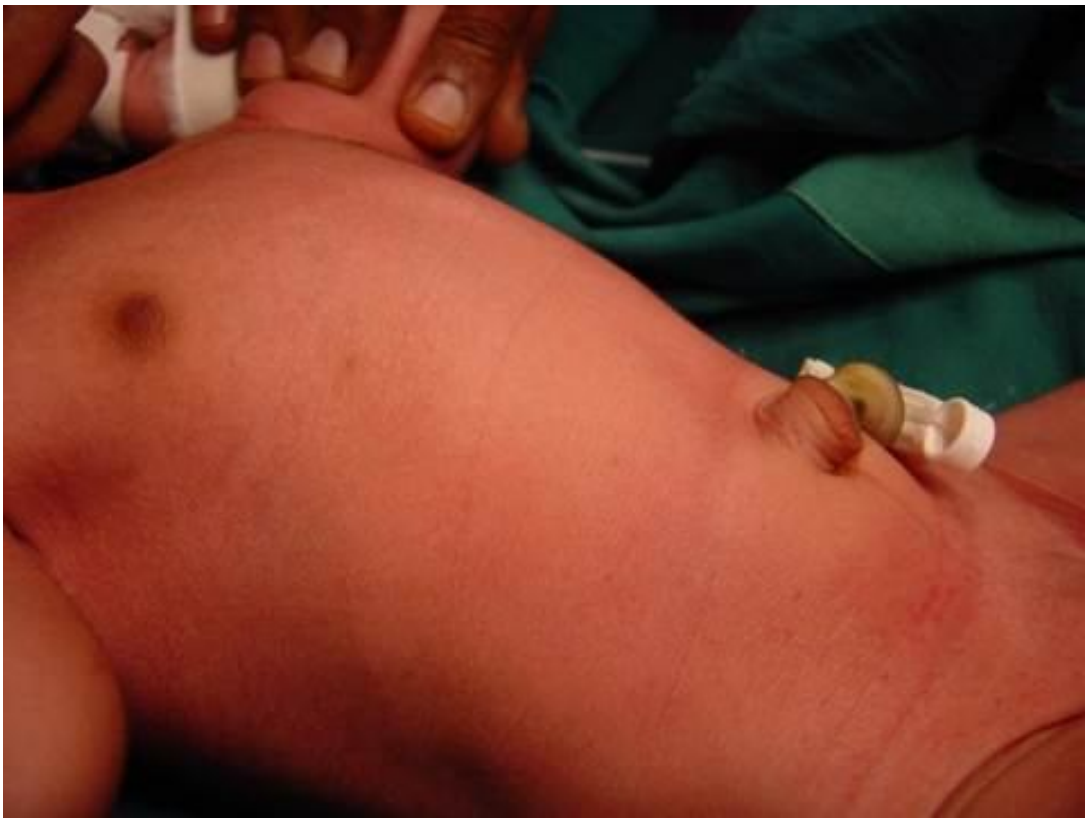


Fig-1. Picture Showing Scaphoid Abdomen and increased AP

Diameter of Chest Wall



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PROFORMA

NAME: **AGE:** **IPNO:**

WEIGHT:

IN BORN BABY:

OUT BORN BABY:

ANTENATAL HISTORY:

BIRTH HISTORY:

CYNOCIS AT BIRTH: **- + -**

RESPIRETORY DESTRESS : **- + -**

SPO2 STAUS: **- RECORDED NOT RECORED**

X-RAY CHEST: **- TAKEN NOT TAKEN**

ECHO CARDIO GRAM: **- DONE NOT DONE**

PRE OPERATIVE VENTILATORY SUPPORT: - YES NO

SIDE OF DEFECT: - LEFT RIGHT

OPERATIVE PRO CEDURE : - DONE NOT DONE

OPERTIVE FINDINGS:

IMMEDIATE POST OP PERIOD: - EVENT FULL UN

EVENT FULL

FOLLOW UP :

ABBRIVATIONS

CDH	-	Congenital diaphragmatic hernia
PPHN/PPHT	-	Persistent Pulmonary Hypertension
ICD	-	Inter costal drain
ECMO	-	Extra corporeal membrane oxygenator
SP O_2	-	Oxygen saturation
NO	-	Nitric Oxide
HFV	-	High Frequency Ventilator
TLV	-	Total Liquid Ventilation
PLV	-	Partial Liquid Ventilation
PaO_2	-	Partial pressure of Arterial Oxygen
$PaCO_2$	-	Partial pressure of Arterial Carbon dioxide

MASTER CHART

S.NO	NAME	AGE	SEX	IP. No	SIDE	DIAGNOSIS	OPERATED	NOT OPERATED	SURVIVAL	MORTALITY
1	B/O ANANTHALAKSHMI	5 DAYS	MALE	26052	LEFT	CDH	√		√	
2	B/O UMAYASELVI	7 DAYS	MALE	26865	LEFT	CDH	√		√	
3	B/O SAMUVEL	8DAYS	MALE	1577	LEFT	CDH	√		√	
4	B/O SELVI	5 DAY	FEMALE	1928	LEFT	CDH	√		√	
5	B/O MALARVIZHI	9 DAY	MALE	4626	LEFT	CDH	√		√	
6	B/O VAIRAMUTHU	4 DAYS	MALE	17525	RIGHT	CDH	√		√	
7	B/O SADHANA	9 DAYS	FEMALE	16152	LEFT	CDH	√		√	
8	B/O CHITRA	6 DAYS	MALE	1963	LEFT	CDH	√		√	
9	B/O JEYALAKSHMI	8 DAYS	MALE	2745	LEFT	CDH	√		√	
10	B.O ANALAKSHMI	7 DAYS	MALE	11718	LEFT	CDH	√			√
11	B/O KALA	9 DAYS	MALE	17445	LEFT	CDH	√			√
12	B/O RASIKA	5 DAY	MALE	18864	LEFT	CDH		√		√
13	B/O RANJANI	6 DAYS	FEMALE	19734	LEFT	CDH	√		√	
14	B/O GIRIJA	4 DAY	FEMALE	44081	LEFT	CDH	√		√	

15	B/O NARMATHA	3 DAYS	MALE	48759	LEFT	CDH	√		√	
16	B/O ABIRAMI	1DAY	FEMALE	53955	LEFT	CDH		√		√
17	B/O PUSPAVALLI	4DAYS	MALE	66690	LEFT	CDH	√		√	
18	B/O TAMILSELVI	1 DAY	FEMALE	92691	LEFT	CDH		√		√
19	B/O UMAMAHESEWARI	1 DAY	FEMALE	25005	LEFT	CDH		√		√
20	B/O VIJYAKUMARI	1 DAY	MALE	80740	LEFT	CDH		√		√
21	B/O VANITHA	5 DAYS	MALE	81117	LEFT	CDH	√		√	
22	B/O HEMALATHA	1 DAY	FEMALE	77282	LEFT	CDH		√		√
23	B/O MUTHUMARI	1 DAY	MALE	67547	LEFT	CDH		√		√
24	B/O MAHALAKSHMI	3 DAYS	MALE	55319	LEFT	CDH	√			√
25	B/O VIGNESWARAN	8 DAYS	MALE	48702	LEFT	CDH	√		√	
26	B/O SUBBULAKSHMI	1 DAY	FEMALE	35760	LEFT	CDH		√		√
27	B/O VEERALAKSHMI	1 DAY	MALE	34403	LEFT	CDH		√		√
28	B/O PRIYA	3DAYS	FEMALE	32659	LEFT	CDH		√		√
29	B/O SAWRIAMMAL	4 DAYS	FEMALE	31292	LEFT	CDH	√		√	
30	B/O SIVAKARTHIKA	6DAY	FEMALE	31132	RIGHT	CDH	√		√	

31	B/O SENBGADEEPA	6 DAYS	FEMALE	26572	LEFT	CDH	√		√	
32	B/O PLEASNT MATHEW	1DAY	MALE	2879	LEFT	CDH		√		√
33	B/O PANDISELVI	10 DAYS	MALE	2452	LEFT	CDH	√		√	
34	B/O AYYAMMALSELVI	2 DAYS	FEMALE	2993	LEFT	CDH		√		√
35	B/O KALEESWARI	3 DAYS	MALE	89967	LEFT	CDH	√		√	
36	B/O KIRUBA	3DAYS	FEMALE	87057	LEFT	CDH	√		√	
37	B/O KALAISELVI	1 DAY	MALE	86344	LEFT	CDH		√		√
38	B/O NAVASAKTHI	6DAYS	FEMALE	79726	LEFT	CDH	√		√	
39	B/O LATHIKA	3DAYS	FEMALE	62233	LEFT	CDH		√		√
40	B/O SELVI	2DAYS	MALE	60331	LEFT	CDH		√		√
41	B/O JAI AKASH	7DAYS	MALE	42504	LEFT	CDH	√		√	
42	B/O BHUVANESWARI	3 DAYS	MALE	85939	LEFT	CDH		√		√
43	B/O ASILYA	1DAY	FEMALE	30894	LEFT	CDH		√		√
44	B/O ANDIAMMAL	5DAYS	FEMALE	21901	LEFT	CDH	√		√	
45	B/O MUMTAJ	3DAYS	FEMALE	8735	LEFT	CDH		√		√
46	B/O MUTHAMMAL	3DAYS	MALE	99008	LEFT	CDH	√		√	

47	B/O ARUNADEVI	7 DAYS	MALE	93230	LEFT	CDH	√		√	
48	B/O MUTHUPILLAI	1 DAY	MALE	84103	LEFT	CDH		√		√
49	B/O REVATHI	6 DAYS	MALE	86260	LEFT	CDH	√		√	
50	B/O MAHESWARI	2DAY	MALE	65674	LEFT	CDH		√		√
51	B/O VARSHA	6DAYS	MALE	59595	LEFT	CDH	√		√	
52	B/O CHELLAMMAL	8DAYS	MALE	58213	LEFT	CDH	√		√	
53	B/O YASHINI	3 DAYS	MALE	56612	LEFT	CDH	√		√	
54	B/O BARAGATH NISHA	1 DAY	MALE	43577	LEFT	CDH		√		√
55	SRINATH	7DAYS	MALE	38784	LEFT	CDH	√		√	
56	B/O KAMATCHI	8 DAYS	MALE	34888	LEFT	CDH	√		√	
57	B/O SELVI	4 DAYS	MALE	36250	LEFT	CDH		√		√
58	B/O RAJESWARI	5DAYS	FEMALE	23258	LEFT	CDH	√		√	
59	B/O PAPATHY	2DAYS	MALE	3261	RIGHT	CDH	√		√	
60	B/O PRIYADHARSHINI	6DAYS	MALE	379	LEFT	CDH	√		√	

Ref. No. 4105/E4/3/2013

Govt. Rajaji Hospital,
Madurai.20. Dated: .03.2013

Institutional Review Board / Independent Ethics Committee.
Dr. N. Mohan, M.S., F.I.C.S., F.A.I.S.,
Dean, Madurai Medical College &
Govt Rajaji Hospital, Madurai 625020.
Convenor

Sub: Establishment-Govt. Rajaji Hospital, Madurai-20-
Ethics committee-Meeting Minutes- approval -regarding.

The Ethics Committee meeting of the Govt. Rajaji Hospital, Madurai was held at 10.00 am to 12.00.pm on 27.03.2013 at the Surgery Seminar Hall, Govt. Rajaji Hospital, Madurai. The following members of the committee have attended the meeting.

1.Dr. V. Nagarajan, M.D., D.M (Neuro) Ph: 0452-2629629 Cell.No 9843052029	Professor of Neurology (Retired) D.No.72, Vakkil New Street, Simmakkal, Madurai -1	Chairman
2. Dr.Mohan Prasad , M.S M.Ch Cell.No.9843050822 (Oncology)	Professor & H.O.D of Surgical Oncology(Retired) D.No.72, West Avani Moola Street, Madurai -1	Member Secretary
3. Dr.L. Santhana Lakshmi,MD Cell.No 9842593412	Associate Professor of Physiology/V.P Madurai Medical College	Member
4. Dr. Parameswari M.D (Pharmacology) Cell.No.9994026056	Director of Pharmacology Madurai Medical College	Member
5. Dr.Moses K.Daniel MD(Gen.Medicine) Cell.No 09842156066	Professor & H.O.D of Medicine Madurai Medical College	Member
6. Dr.D. Soundara Rajan,MS(Gen.Surgery) Cell.No 9842120127	Professor & H.O.D of Surgery Madurai Medical College	Member
7. Dr.Angayarkanni MD(O&G) Cell.No 9443567724	Professor & H.O.D of O&G Madurai Medical College	Member
8. Dr.P.V. Pugalenthi M.S, (Ortho) Cell.No 9443725840	Professor & H.O.D Ortho Madurai Medical College	Member
9. Dr. M. Sundarajan M.S., Mch Cell.No 9994924369 (Neuro Surgery)	Professor (Neuro Surgery) Madurai Medical College	Member
10 Thiru..Pala. .Ramasamy , BA.,B.L., Cell.No 9842165127	Advocate, D.No.72.Palam Station Road, Sellur, Madurai -2	Member
11. Thiru. P.K.M. Chelliah ,B.A Cell.No 9894349599	Businessman, 21 Jawahar Street, Gandhi Nagar, Madurai-20.	Member

The following Project was approved by the committee

Name of P.G.	Course	Name of the Project	Remarks
Dr. R. Srinivasakumar	PG in M.Ch., (Peadiatrics Surgery) Madurai Medical College, Madurai-20 & Govt. Rajaji Hospital Madurai-20	Clinical Profile and Surgical outcome of patients with congenital Diaphragmatic Hernia	Approved

Please note that the investigator should adhere the following: She/He should get a detailed informed consent from the patients/participants and maintain Confidentially.

1. She/He should carry out the work without detrimental to regular activities as well as without extra expenditure to the institution to Government.
2. She/He should inform the institution Ethical Committee in case of any change of study procedure site and investigation or guide.
3. She/He should not deviate for the area of the work for which applied for Ethical clearance.

She/He should inform the IEC immediately, in case of any adverse events pr Serious adverse reactions.

4. She/he should abide to the rules and regulations of the institution.
5. She/He should complete the work within the specific period and apply for if any Extension of time is required She should apply for permission again and do the work.
6. She/He should submit the summary of the work to the Ethical Committee on Completion of the work.
7. She/He should not claim any funds from the institution while doing the word or on completion.
8. She/He should understand that the members of IEC have the right to monitor the work with prior intimation.

DEAN/Convenor
Govt. Rajaji Hospital,
Madurai- 20.

27/3/13

To
The above Applicant
-thro. Head of the Department concerned.

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through the posterolateral foramen of Bochdalek (ii) Herniation through **the** substernal **foramen of Morgagni;** 4

and (iii) Herniation through the esophageal hiatus. The term

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posterolateral foramen of Bochdalek. Congenital diaphragmatic hernia remains

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