Delayed Surgical Repair and ECMO Improves Survival in Congenital Diaphragmatic Hernia

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One hundred ten infants with congenital diaphragmatic hernia (CDH) developed life-threatening respiratory distress in the first 6 hours of life. Associated anomalies were present in 33%. Twenty-eight of 65 infants (43%) treated before 1987 (pre-extracorporeal membrane oxygenation [ECMO] era) survived after immediate CDH repair, and mechanical ventilation with or without pharmacologic support. Only two of 16 (12.5%) infants requiring a prosthetic diaphragmatic patch survived. Since 1987, 31 of 46 (67.4%) infants with birth weight, gestational age, and severity of illness similar to the pre-1987 group survived. All patients were immediately intubated and ventilated. Seven (four with lethal chromosomal anomalies) infants died before treatment, and 30 stabilized (partial pressure of carbon dioxide [P_{CO_2}] < 50; partial pressure of oxygen [P_{O_2}] > 100; pH > 7.3) and underwent delayed CDH repair at 5 to 72 hours. Fifteen did well on conventional support and survived. Fifteen infants deteriorated after operation: 11 were placed on ECMO with eight survivors, and four infants were not considered ECMO candidates. Nine babies failed to stabilize initially and were placed on ECMO before CDH repair (alveolar-arterial gradient > 600 and oxygenation index > 40), and seven survived. The overall survival rate was 80% at 3 months in this ECMO-treated group. Early mortality was due to inability to wean from ECMO (one), intracranial hemorrhage (one), liver injury (one), and pulmonary hypoplasia (one). Nine of 11 babies requiring a prosthetic patch in the post-1987 ECMO group survived (81.8%). There were three late post-ECMO deaths (3 to 18 months) of right heart failure (two) and sepsis (one). Symptomatic gastroesophageal reflux occurred in nine cases, six requiring a fundoplication in the bypass babies. Recurrent diaphragmatic hernia occurred in nine cases (five ECMO). The overall survival rate was significantly improved in the delayed repair/ECMO group (67% *versus* 43%; $p < 0.05$) and was most noticeable in infants requiring a prosthetic diaphragm (81.2% *versus* 12.5%; $p < 0.005$). These data indicate that early stabilization, delayed repair, and ECMO improve survival in high-risk CDH. Early deaths are related to pulmonary hypertension and can be reversed by ECMO.

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NEONATES BORN WITH a congenital posterolateral diaphragmatic hernia (Bochdalek's hernia) represent a significant clinical challenge. Despite early prenatal sonographic diagnosis and planned delivery at a high-risk obstetrical center, and the availability of neonatal intensive care, modern infant ventilators, pharmacologic manipulations, and emergency operation, the reported mortality rate ranges from 33% to 60%.[1–6] Some reports suggest that in babies with congenital diaphragmatic hernia (CDH), persistent pulmonary hypertension of the newborn (PPHN) results in cardiopulmonary instability or death and is exacerbated by early surgical intervention.[1] These observations stimulated the concept of delayed surgical intervention after medical stabilization before attempted repair of the diaphragmatic defect. During the stabilization phase, attempts are made to ameliorate PPHN by hyperventilation, "gentle ventilation," induced alkalosis, and pharmacologic vasodilator therapy (Priscoline, Ciba Pharmaceutical Co., Summit, NJ). In recent years, the use of extracorporeal membrane oxygenation (ECMO) either before or after surgical repair has been employed as a method of improving oxygenation (in the presence of significant right-to-left shunts), reducing pulmonary hypertension, and minimizing barotrauma that often accompanies conventional ventilation.[2–6] The role of ECMO as a method of improving survival in CDH has been controversial.[7,8] This report concerns 111 infants with CDH that presented with respiratory distress in the first 6 hours of life and evaluates the influence of delayed repair and ECMO on survival.

Patient Material

One hundred eleven infants presenting with symptomatic CDH in the first 6 hours of life were treated at the

James Whitcomb Riley Hospital for Children, Indiana University Medical Center from 1975 to 1992. A retrospective analysis comparing sex, gestational age, associated anomalies, severity of symptoms, and clinical management was performed. Statistical analysis was by unpaired t test or chi square analysis. There were 31 girls and 80 boys, ranging in gestational age from 28 to 42 weeks (mean, 38 ± 4 weeks) and weighing between 1500 and 3800 g (3200 ± 520 g). Eighty-five patients had a left-sided hernia, 13 a right-sided defect, two had bilateral defects, and two a central defect. Severity of illness was evaluated by $(A - a)D_{O_2}$ (alveolar-arterial difference), oxygenation index (OI), and ventilatory index (VI) measurements (Table 1). Comparison of the best preductal and postductal partial pressures of oxygen and carbon dioxide (Pa_{O_2} and Pa_{CO_2}) were recorded. Complete data for some patients very early in the series was often not available from archival records in the pre-ECMO years, where only $(A - a)D_{O_2}$ values could be calculated.

All infants were intubated, mechanically ventilated, paralyzed (pancuronium 0.1 mg/kg), or sedated (morphine 0.10 mg/kg or seconal 1 mg/kg) at the time of diagnosis of CDH. Gastrointestinal decompression was maintained with an orogastric tube. Early in the series, most patients were born in outlying hospitals and transported by ambulance to our tertiary neonatal facility. Before 1987, emergency surgical intervention was the standard of care at our institution. Since 1987 (corresponding with the increased rate of CDH detection by prenatal ultrasound), 24% (11 of 46) of the patients were delivered at the Indiana University Hospital high-risk obstetrical unit and transferred to the neonatal intensive care unit (NICU) at the J.W. Riley Hospital for Children shortly after birth. After 1987, delayed surgical repair (surgeon dependent) with or without the adjuvant use of ECMO was employed. Conventional ventilation or high-frequency methods were used to achieve a $Pa_{O_2} > 100$ mmHg, Pa_{CO_2} 30 to 35 mmHg, and a pH of 7.40 to 7.5. Seven neonates died without surgical repair because of the presence of lethal chromosomal abnormalities in four infants and inability to hyperventilate and achieve a Pa_{CO_2} less than 50 mmHg in three. Thirty infants stabilized initially and underwent laparotomy and diaphragmatic repair at 5 to 72 hours of age. Eleven of these cases subsequently deteriorated after operation and were placed on ECMO at 10 to 168 hours of age. Four children died after operation without going on bypass because of associated complicated congenital heart disease and giant omphalocele in one, reversion to persistent pulmonary hypertension of the newborn (PPHN) at 12 days of age in one and physician/family decision in two. Nine infants were placed on ECMO bypass before surgery and had their diaphragmatic repair 72 to 144 hours into the ECMO run (ECMO criteria for CDH, Table 2). The total length of bypass ranged from 144 to 336 hours. Bleeding complications were minimized by maintaining an activated clotting time of 170 to 190 seconds, platelet counts of 120 to 150,000 mm^3 and application of fibrin glue on all suture lines. All of the operative procedures while on bypass were performed in the NICU using pancuronium for paralysis and fentanyl infusions (10 μg/kg/hour) in the ECMO circuit.

TABLE 1. *Calculations Used in Assessing Severity of Illness*

$$A\text{-}aD_{O_2} = FI_{O_2}(760 \text{ partial pressure } H_2O) - Pa_{O_2} - \frac{Pa_{CO_2}}{0.8^2}$$

$$OI = MAP \times \frac{FI_{O_2}}{Pa_{O_2}} \times 100$$

$$VI = RR \times MAP$$

OI, oxygenation index; VI, ventilation index; RR, respiratory rate; MAP, mean airway pressure.

TABLE 2. *Present ECMO Criteria for CDH Infants at Riley Hospital*

Gestational age >32 wk
Weight >1600 g
$BPDPCO_2$ <50

Failure to stabilize on conventional therapy

Diaphragmatic replacement was required in 16 patients with large diaphragmatic defects before 1987 using Gore-tex (Gore) in eight infants, Prolene mesh in four, Marlex in two, and abdominal wall muscle flaps in two. Only two of the 16 patients survived (12.5%). In the delayed therapy/ECMO era (after 1987), 13 neonates required diaphragmatic reconstruction, with Gore-Tex used in all cases. In 11 of these infants, ECMO support was used either before or after CDH repair. Eleven of the 13 infants survived (84.5%), including nine of 11 in the ECMO-treated group (81.8%). There were four early ECMO deaths (20%) due to massive intracranial hemorrhage (one), hepatic injury (one), and pulmonary hypoplasia (two), all occurring in the first 2 weeks of life; only one infant was successfully weaned from bypass. There were three additional late post-ECMO deaths (at 3, 12, and 18 months) due to right-heart failure (two), and unrelated sepsis (one) (Fig. 1). A comparison of $(A - a)D_{O_2}$, VI, and OI for the three treatment groups is outlined in Table 3.

One third of all the patients had associated congenital anomalies (Table 4), with trisomy 13 and 18 and cardiac defects (lethal defects) carrying the highest mortality rate regardless of the method of treatment. The overall survival rate was significantly improved in the delayed repair/ECMO patients (67%) *versus* the pre-ECMO group (43%) undergoing emergency surgery ($p < 0.05$). The improved outlook is most notable in those infants requiring a prosthetic diaphragmatic replacement (Fig. 2). Eighty-four per

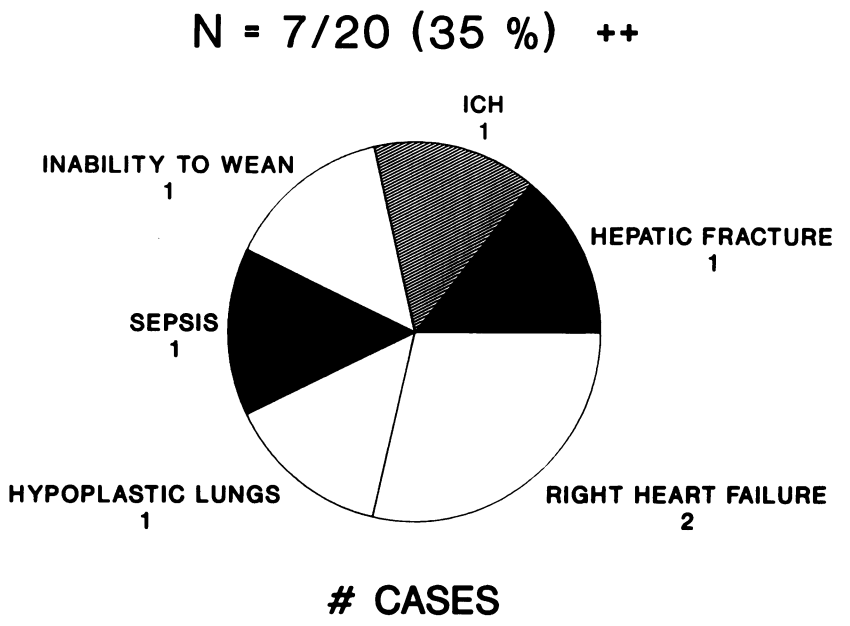

FIG. 1. A 35% long-term mortality (7 of 20) was seen in the patients who required ECMO support.

cent (11 of 13) of the babies survived after delayed repair or ECMO (9 of 11 ECMO) compared with only 12.5% (2/16) of infants treated before 1987.

Symptomatic gastroesophageal reflux was noted in 20 patients. An antireflux procedure was required in 11 infants (9%) overall, including six of the 20 (30%) babies treated with ECMO (Fig. 3). Recurrent diaphragmatic hernia occurred in nine cases, including five in the ECMO-treated group (1 to 12 months after repair) (Fig. 4). Additional surgical procedures were required in 55 cases and are listed in Table 5.

Discussion

In 1940, Ladd and Gross reported survival in 64 of 72 children (71%) with CDH, the youngest being 40 hours old.[9] With the widespread availability of ultrasound, prenatal diagnosis of CDH became commonplace, allowing planned delivery in high-risk obstetrical centers. Rather than improving the outcome for CDH, the overall survival actually decreased to less than 50% as more affected infants presented for repair. This "hidden mortality" initially described by Harrison et al.[10] indicated that many infants with CDH previously died before or during transport to

TABLE 3. *Comparison of Severity PPHN in Patients Treated With and Without ECMO*

	Delayed, Non-ECMO	ECMO
$A\text{-}aD_{O_2}$		
Survivors	283 ± 188	604 ± 51*
Deaths	617 ± 52*	638 ± 12*
VI		
Survivors	337 ± 124	1321 ± 271*
Deaths	1346 ± 275*	1349 ± 408*
OI		
Survivors	20 ± 13	46 ± 15
Deaths	67 ± 55	90 ± 53

* NS.

TABLE 4. *Associated Anomalies*

Musculoskeletal	Club foot	4
	Cleft lip/palate	3
	Arthrogryposis	1
	Bilateral hip dislocation	1
	Thoracic dystrophy	1
	Hemihypertrophy	1
	Hemivertebrae	1
Urologic	Cryptorchidism	7
	Duplicated ureter	2
	Horseshoe kidney	1
	Hypospadias	1
	Ureteropelvic junction obstruction	1
Gastrointestinal	Hiatal hernia	8
	Duodenal atresia	2
	Imperforate anus	2
	Omphalocele	2
	Ectopic pancreas	1
	Annular pancreas	1
Cardiac	ASD	9
	VSD	4
	A-V canal	1
	Cor triatriatum	1
Chromosomal	Trisomy 18	7
	P-chromosome	4
	Trisomy 13	1
	46 xy/45 × mosaic	1
	46 xy translocation	1

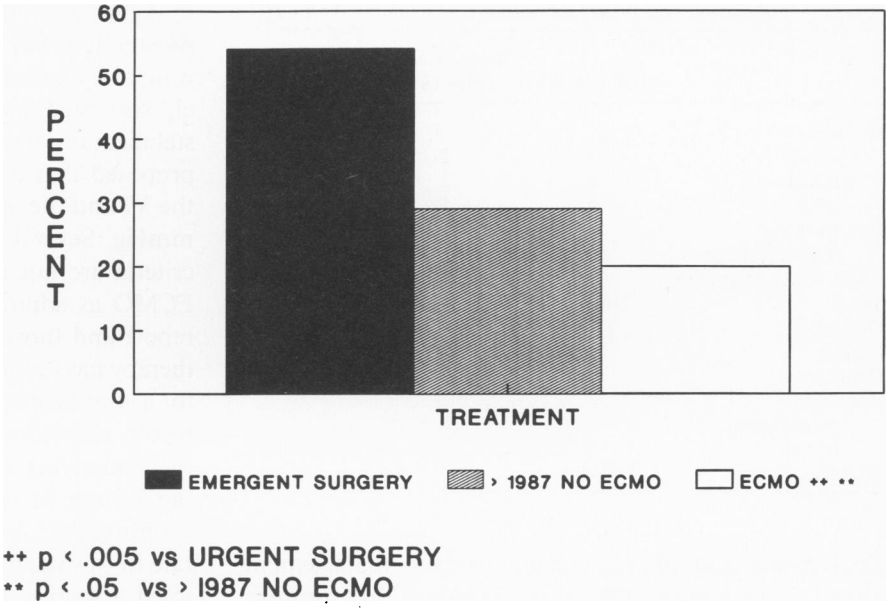

FIG. 2. The operative mortality rate (<3 mo) is reduced in patients undergoing delayed repair with or without ECMO support compared with emergency surgery.

a high-risk center. Adzick et al.[11] followed 94 cases of CDH diagnosed by prenatal ultrasound and recorded only a 20% overall survival and recognized a number of poor prognostic indicators including bilateral diaphragmatic defects, polyhydramnios, and ultrasound diagnosis before 23 weeks gestational age.[11]

Although the previous mainstay of care included emergent operative repair, more than half the babies who presented in the first few hours of life with respiratory distress died. Dibbins and Weiner[12] suggested that the early deaths in instances of CDH were related to persistent pulmonary hypertension of the newborn (PPHN) with progressive hypoxemia, the result of right-to-left shunting.[12] Neonates that present with early respiratory distress tend to fall into two categories: (1) those that fail to improve despite sur-

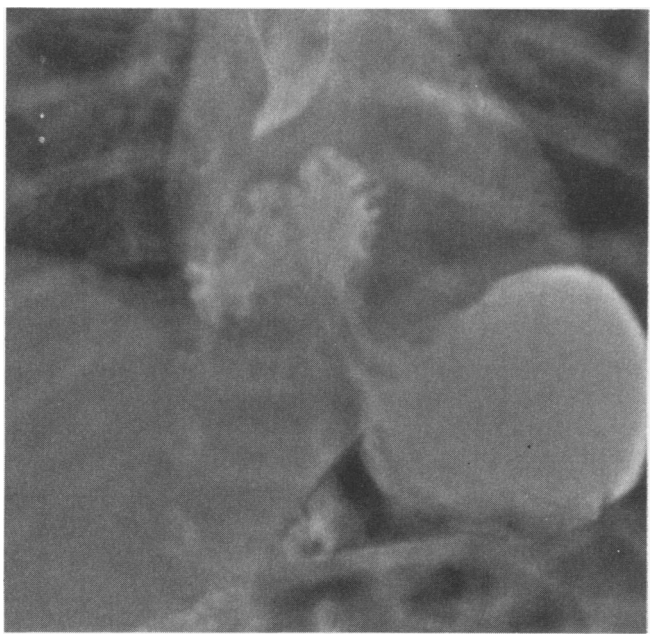

FIG. 3. This 3-month-old ECMO survivor had massive GER associated with a hiatal hernia and required a Nissen fundoplication.

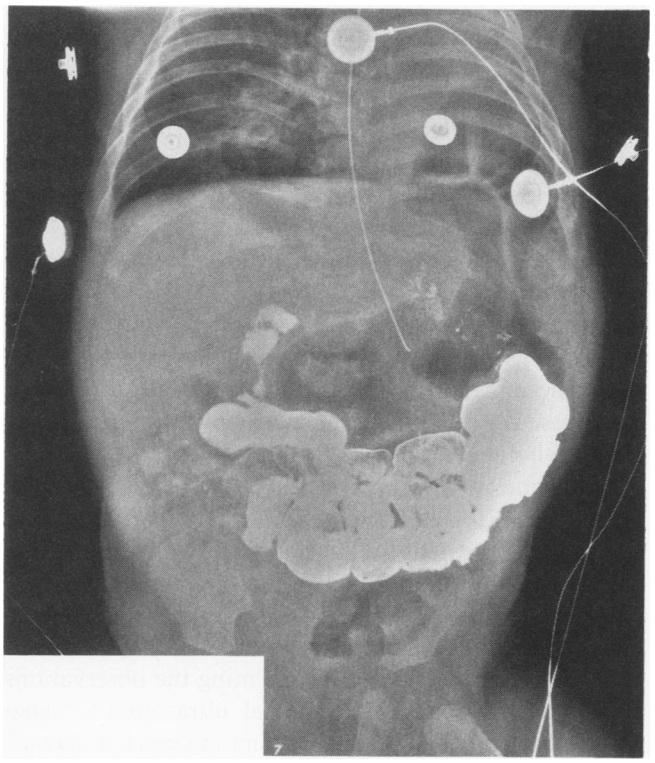

FIG. 4. At 12 months of age, this child with a large Gore-tex patch had feeding difficulties and was noted to have a recurrent posterolateral diaphragmatic hernia.

TABLE 5. *Subsequent Operative Procedures in 111 Infants With CDH*

	Before 1987	Non-ECMO After 1987	ECMO After 1987
Hernias			
Ventral	6	2	1
Inguinal	6	2	2
Antireflux	4	1	6
Postoperative intussusception	1	—	—
Small bowel obstruction	1	1	—
Trach	3	—	3
Orchiopexy	1	—	1
Cardiac	1	—	—
Dehiscence	1	—	—
Recurrent diaphragmatic hernia	2	2	5
Urologic	—	1	2

gical repair and pharmacologic medical management aimed at attenuating PPHN or (2) babies that improve after repair and go through a temporary stable "honeymoon period," followed by a sudden spiraling crescendo of right–left shunting and death. In autopsy examinations of babies that failed to demonstrate improved oxygenation, after operation the lungs are not only hypoplastic but there is a thickened smooth muscle in the medial layer of the pulmonary vessels associated with a generalized decrease in the number of arterial branches. It is this excessive muscularization of the pulmonary vessels that may predispose some CDH babies to "overreact" to small changes in arterial pH or oxygen tensions.[13,14] Pharmacologic modulation using selective pulmonary artery vasodilators such as tolazoline and nitroprusside may not be successful.[15,16] In this series, pharmacologic agents did not alter the overall mortality rate, although Priscoline did allow for transient stabilization while preparing for ECMO cannulation and lengthened the observed honeymoon periods in the pre-ECMO era. Control of systemic pressure is important during vasodilator therapy and both fluid boluses and administration of ionotropic agents (dobutamine and dopamine) may be necessary to prevent cardiovascular collapse during tolazoline infusion (initially 1 mg/kg bolus followed by 1 mg/kg/hour).

Emergent surgical repair of the CDH seemed logical to reduce the herniated viscera, prevent bowel incarceration and ischemia, restore the cardiac shadow to the midline, and improve ventilation of the compressed contralateral lung. Langer et al.,[17] however, documented that the viscera would often move from one side of the diaphragm to another through the defect (confirming the observations of Harrison's group with prenatal ultrasounds),[10] suggesting that the risk of bowel incarceration was low. Sakai[18] documented that the surgical repair could actually worsen ventilation by decreasing compliance (a decrease in compliance of 50% raised the mortality rate to 100%). Changes in compliance were related to distortion of the chest by prosthetic patch closure, compression of the abdominal wall, and overinflation of the contralateral lung. Bohn et al.[1] suggested that delayed surgical repair might allow for stabilization of the pulmonary artery smooth muscle and proposed that death could be predicted on the basis of the VI and the ability to reduce the Pa_{CO_2} to less than 40 mmHg. Several authors have documented that Bohn's criteria are not reliable predictors of death when using ECMO as adjunctive therapy. The data in the present report and those of others suggest that delayed surgical therapy has not increased mortality rate, but in fact allows for a stratification of patients for early ECMO or surgical repair, depending on the clinical course.[2–5,19] Certainly the underlying pulmonary hypoplasia is not altered by the timing of the repair, but PPHN and subsequent shunting does account for the significant early mortality rate in this population. Touloukian and Markowitz[20] devised a scoring system based on the preoperative chest radiograph and the presence of aerated lung, side of the defect, initial location of the stomach, (above or below the diaphragm), and the presence or absence of a pneumothorax.[20] Seventy-five per cent of those infants with the stomach in the chest (indicative of a large defect) died in the pre-ECMO group, and 65% survived since 1987 ($p < 0.05$). Some authors argue that the best preductal or best postductal Pa_{O_2} are most important in predicting survivorship. Others would suggest that all neonates with a CDH (and no life-threatening anomalies) should be considered for ECMO.[8] We have previously offered ECMO therapy only to those infants with at least one preductal Pa_{O_2} greater than 100 mmHg and a preductal Pa_{CO_2} less than 50 mmHg, a normal head ultrasound, and no concomitant life-threatening chromosomal abnormalities. Using these criteria, some children may die without surgical repair of the defect, a decision that must be made by the health care team in conjunction with the family.

Bartlett and associates have shown that certain neonates with PPHN can be salvaged with ECMO and along with others have extended this therapy to the CDH infant.[21,22] The Extracorporeal Life Support Oxygenation registry now includes more than 1000 infants with CDH treated with ECMO, with a survival rate of 61% (predicted mortality rate of >80%).[23] Cardiac ECHO and head ultrasounds are obtained before consideration for ECMO support. If an intracranial bleed already exists, the baby is not a candidate for systemic heparinization, which is presently needed for ECMO support. We also exclude neonates with prolonged hypoxia or a prolonged cardiac arrest before initiating extracorporeal support. Either venoarterial (V-A) or veno-veno (V-V) bypass is instituted, depending on the infant's cardiac ejection fractions (good myocardial function required for V-V bypass). In the premature or low-birthweight infant, a cephalad catheter is

placed at the base of the jugular bulb to prevent intracranial venous hypertension, reduce the risk of bleeding, and allow for measurements of cerebral oxygen extraction. Two babies in this series who were 33 and 34 weeks estimated gestational age were successfully managed on ECMO using this technique without evidence of intracranial bleeding. Most commonly, the operative repair is performed by an abdominal approach (although some authors suggest that this be accomplished through the chest while on ECMO to minimize trauma to the liver and spleen). There is an attempt to increase the right of domain for the reduced bowel by manual stretching of the abdominal wall and devising a diaphragm closure that does not distort the chest wall (using a prosthetic material) when a large defect is present. A temporary Dacron silo occasionally may be required to manage the abdominal incision similar to the care of the newborn with an abdominal wall defect.[24] If ECMO is contemplated, thrombinglue (a combination of thrombin, calcium, and cryoprecipitate) is applied to the suture lines. In both the preoperative and postoperative periods, fluid administration is minimized (to prevent excess fluid sequestration into the pulmonary parenchyma), because these neonates may demonstrate an exaggerated fluid retention response. Controlled yet gentle ventilation is employed to achieve adequate oxygenation without inducing barotrauma. High-frequency jet-oscillating ventilation was used in four babies as a "bridge" to ECMO. One CDH repair was performed with the baby on the jet ventilator (she ultimately required ECMO support at 6 days of life because of intractable recurrent PPHN). If alkalosis is not achieved with hyperventilation, then bicarbonate infusions of 0.1 to 0.3 mEq/kg/hour may be added to minimize the risk of PPHN and to maintain the pH at 7.45 to 7.5. There is a wide spectrum of PPHN in this population of neonates, and therapy must be designed for each individual patient. Infants who have been stable during the period of delayed surgical management may begin to shunt during transport to the operating theater. We suggest that these high-risk newborn patients undergo CDH repair in the NICU rather than be subjected to factors that influence pulmonary artery spasm (hypoxia, hypercarbia, hypothermia, stress-induced release of epinephrine, and acidosis). In the current report, the use of ECMO and delayed CDH repair has resulted in a significant improvement in overall survival (67% *versus* 43%). These observations were most pertinent in babies with large diaphragmatic defects in which an 88% (14/16) mortality rate was noted before the availability of ECMO. Early death was reduced to 20% in babies requiring a prosthetic since employing initial stabilization, delayed surgical intervention, and ECMO. These data differ from those reported by O'Rourke et al.,[7] which failed to demonstrate the efficacy of ECMO on survival in babies with CDH presenting in the first 6 hours of life. Other centers, however, concur with our findings—suggesting that ECMO enhances CDH survival. Unlike Wilson et al.,[25] we found that delayed surgical therapy coupled with ECMO before or after surgery had a favorable impact on survival. In addition, Nakayama documented that pulmonary compliance changes were reduced when delayed surgery and ECMO were used.[26] In our cases, both therapies were used beginning in 1987, which may account for the differences between Wilson's results and those in this series. There was no statistical difference in survival in our post-1987 cases, however, between babies who had ECMO instituted before or after CDH repair. Clearly, 30% of the deaths in the pre-ECMO era had honeymoon periods before their deaths and might well have benefited from ECMO support.

Gastroesophageal reflux (GER) has become a significant postoperative problem after CDH repair, particularly in babies that require a large prosthetic diaphragmatic patch. This may be related to a weak diaphragmatic crus or a hiatal hernia. In the current series, six (42% of the survivors) of the 20 ECMO-treated patients required an antireflux operation to prevent recurring episodes of aspiration. An additional four infants with GER were managed medically, and avoided antireflux surgery. In addition to the occurrence of GER, Stolar et al.[27] suggest that the esophagus in some CDH patients may be ectatic. There was a high correlation of ectatic esophagus and the presence of prenatal polyhydramnios. We have not noted this occurrence in our patients.

Recurrent diaphragmatic hernias present a difficult problem, especially if there is minimal native diaphragmatic tissue present at the time of repair, and the prosthetic material must be secured around a rib. An attempt is made to create a slightly patulous graft that will, it is hoped, develop a flattened appearance as the infant's chest grows in cross-sectional area. This also may prevent the distortion of the chest wall and a subsequent decrease in compliance. There were nine total recurrent diaphragmatic hernias (four were non-ECMO patients). The five recurrent CDH defects in the ECMO group occurred between 1 and 12 months after the initial repair and were confirmed by contrast radiography. One child had two separate recurrent diaphragmatic hernias, and eventually required a latissimus dorsi muscle flap for closure.

One child has profound mental retardation, seizures, and evidence of a cerebral infarct after ECMO. All the other ECMO survivors are within 2 to 4 months of their chronologic ages in developmental testing, most lagging behind in speech development. Three children experienced long-standing cardiac arrhythmias (supraventricular tachycardia), and one has a unilateral vocal cord paralysis.

Future therapy for these infants may include *in utero* CDH repair for the particularly high-risk group of fetuses (polyhydramnios, prenatal diagnosis before 23 weeks ges-

tational age) as carefully outlined by Harrison et al.[28] In addition, pulmonary transplantation of a single parental lobe may be possible in those infants who survive ECMO but have potentially lethal pulmonary hypoplasia.[29] Theoretically, the transplanted lobe of lung could be subsequently removed as the hypoplastic native lung grows and matures, adding alveolar surface area.

Two infants in the ECMO-treated group died as a result of right-sided cardiac failure at 3 and 18 months after birth. Although both patients were on chronic ventilator support at the time of their deaths, each had a delay in diagnosis and had echocardiographic evidence of right ventricular hypertrophy. We suggest that all CDH children have subsequent cardiac ECHO examinations to follow the size of the ventricular wall to detect chronic changes and initiate early ventilatory support (in addition to supplemental oxygen) and medical management aimed at maximizing right heart function. This may prevent the late deaths attributed to pulmonary hypoplasia and chronic lung disease in some series.[8,25]

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Discussion

DR. KATHRYN D. ANDERSON (Washington, DC): This is a careful longitudinal study of the evolution of treatment of congenital diaphragmatic hernia in a single institution.

Before the development of extracorporeal membrane oxygenation (ECMO) in neonates by Bartlett and his colleagues, more than 50% of infants with diaphragmatic hernia died whatever was done for them. Dr. West and her colleagues have shown a rise in survival from 43% in their pre-ECMO era to 67% after 1987, with a remarkable survival of nearly 80% of patients operated on while on ECMO.

The problem with these patients is that they are a very heterogeneous group, and comparing experiences between institutions is like comparing apples and oranges. Having said that, I will now compare our oranges with your apples.

At Children's in Washington, DC, we have treated 102 infants with diaphragmatic hernia since starting our ECMO program 7½ years ago. Of these, 49 did not need ECMO and all of them survived. The other