Journal of Pediatric Surgery 55 (2020) 1511–1515

Contents lists available at ScienceDirect

Journal of Pediatric Surgery

journal homepage: www.elsevier.com/locate/jpedsurg

Other Neonatal Conditions

# Is thoracoscopic esophageal atresia repair safe in the presence of cardiac anomalies?

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ARTICLE INFO

*Article history:*
Received 27 July 2019
Received in revised form 3 March 2020
Accepted 12 March 2020

*Key words:*
Esophageal atresia
Tracheoesophageal fistula
Congenital heart disease
Thoracoscopic surgery

ABSTRACT

*Background:* Esophageal atresia (EA) is often associated with congenital heart disease (CHD). Repair of EA by the thoracoscopic approach places physiological stress on a newborn with CHD. This paper reviews the outcomes of infants with CHD who had undergone thoracoscopic EA repair, comparing their outcomes to those without CHD.
*Methods:* This was a review of infants who underwent thoracoscopic EA repair from 2009 to 2017 at one institution. Operative time and outcomes were analyzed in relation to CHD status.
*Results:* Twenty five infants underwent thoracoscopic EA repair during the study period. Seventeen (68%) had associated anomalies of whom 9 (36%) had cardiac anomalies. The mean operative time was 217 min. There was no difference in operative time between CHD and non-CHD cases (estimate 20 min longer operative time in the presence of a cardiac anomaly [95% CI −20 to 57]). Two cases were converted to open thoracotomy; both were non-CHD. There was no difference in the time to feeding, time in intensive care unit or time in hospital between CHD and non-CHD cases. Five patients developed an anastomotic leak (two CHD and three non-CHD) of which two were clinical; all were managed conservatively. There was no case of recurrent fistula.
*Conclusions:* This pilot study did not find evidence that thoracoscopic EA repair compromised outcomes in children with congenital heart disease. A prospective multicenter study with long-term follow-up is recommended to confirm whether thoracoscopic repair in CHD is truly equivalent to the open operation.
*Type of study:* Therapeutic.
*Level of evidence:* Level III.



Cameron Haight performed the first successful primary repair of esophageal atresia (EA) in 1941 through a left paravertebral thoracotomy [1,2]. Seven years later, Henry Barrett of Taranaki, New Zealand, performed the first repair in the southern hemisphere [3]. Lobe and Rothenburg pioneered the thoracoscopic approach when they repaired a long-gap EA in a 3-month-old infant in 1999 [4]. The purported advantages of thoracoscopic over open EA repair include better visualization, fewer chest wall deformities and improved cosmesis [5,6]. Two meta-analyses have demonstrated no difference in the leak or stricture rate compared to open repair [7,8]. Despite this, the thoracoscopic approach has not achieved universal acceptance in the pediatric surgical community [9–11]. Some have expressed concerns about the safety of thoracoscopic EA repair. Bishay et al. demonstrated significant hypercapnea and acidosis during thoracoscopic EA repair [12,13]. Pierro recommended further studies to confirm whether thoracoscopic surgery in neonates is safe and to determine its impact on neurological development [14]. In a child with other physiologically important anomalies such as congenital heart disease (CHD), outcomes could be further compromised.

More than 30% of infants with EA have associated CHD [15,16]. Major CHD significantly reduces the chance of survival as shown by Waterston [17] and confirmed by others [18,19]. For this reason, surgeons have avoided the thoracoscopic approach in CHD. In a survey of the International Pediatric Endosurgery Group, CHD was one of the most common reasons to avoid the thoracoscopic approach to EA repair [10]. Others have similarly reported a selective approach [20–22]. Conversely, surgeons have performed thoracoscopic EA repair with success in the presence of major heart defects, including ventricular hypoplasia [23,24]. One multicenter review of thoracoscopic EA repair included 5% of cases that subsequently underwent cardiac surgery: two of these infants later died [21]. Until now, no study has addressed the outcomes of thoracoscopic EA repair with respect to congenital heart anomaly.

Therefore, to address this gap in the literature, we reviewed our experience of EA repair in neonates with and without CHD. The aim of the present study was to determine whether CHD was associated with longer operative times or compromised outcomes in thoracoscopic EA repair.

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https://doi.org/10.1016/j.jpedsurg.2020.03.004
0022-3468/

## 1. Methods

This study was approved by our institutional Research Office. We retrospectively reviewed EA repairs performed at one tertiary referral children's hospital from 2009 to 2017 inclusive (9 years). Thoracoscopic EA repair was first introduced to our unit in 2009.

Patients were identified from the departmental operative database and a surgeon's operative log book. Follow-up was by surgical outpatient clinic.

The inclusion criterion for this study was any infant with EA repaired in the neonatal period. The exclusion criteria were Gross type A (isolated EA), type B (EA with proximal fistula only), or type E (isolated fistula) EA, and EA repair outside of the neonatal period. The primary inclusion criterion for thoracoscopic repair was the presence of a thoracoscopic surgeon. Two surgeons routinely performed thoracoscopic repair while the other three surgeons in the unit routinely performed open repair.

For the thoracoscopic surgeons, the exclusion criteria for a thoracoscopic approach were prematurity <36 weeks' gestation or critically unstable patient. Cardiac anomaly did not preclude a thoracoscopic approach; however, the surgeon and anesthetist could agree on an open approach at their clinical discretion.

Data on demographics, associated anomalies, operative details and outcomes were acquired from electronic clinical records. Operative time was acquired from the operating suite database. Operative time was taken from the start of the bronchoscopy to the end of skin closure and thus included the anesthetic time between bronchoscopy and skin incision. Follow-up was measured from the date of surgery to the date of last follow-up at our institution. CHD was defined as a congenital abnormality of the heart, excluding patent ductus arteriosus (PDA), patent foramen ovale (PFO) and dextrocardia with no internal cardiac structural anomaly. Infants with PDA and/or PFO but no other anomaly were classified as 'no anomaly'. 'Major' cardiac anomaly was defined as any heart defect requiring surgical repair.

## 2. Anesthetic technique

The anesthetic technique of EA repair has been described by others [22,25]. A pediatric cardiac anesthetist assisted all CHD cases. Inhalational induction with sevoflurane was standard. Neuromuscular blockade was used throughout thoracoscopic cases. In open cases, neuromuscular blockage was avoided until after ligation of the fistula. The site of the fistula was determined by bronchoscopy, either flexible or rigid. Endotracheal tube placement was guided by flexible bronchoscopy. When the distance between the fistula and carina was sufficient, the tip of the endotracheal tube was positioned below the fistula and above the carina. Mechanical ventilation with pressure control or pressure control volume guarantee was used. For thoracoscopic cases, minute volume was maintained with a high rate–low volume ventilation strategy. Arterial line access was routine. Two peripheral lines were placed in all cases. Central venous access was used in selected cases. Standard monitoring included heart rate, invasive and noninvasive blood pressure, arterial oxygen saturation, end tidal $CO_2$, and blood gases analysis. Regional cerebral oxygen saturation was monitored at the discretion of the anesthetist but was not used in all cases. When cerebral oxygenation was used, a near infrared spectrometry (NIRS) value of 40% or higher was targeted. Inotrope and vasopressor were administered as indicated. The standard vasopressors were metaraminol and noradrenaline.

## 3. Operative technique

The operative technique of thoracoscopic EA repair has been described by others [20,21]. Briefly, following bronchoscopy, infants are positioned 45° semiprone with the thorax suspended between axillary and hip rolls, taking care to minimize pressure on the chest or abdomen. We use three millimeter ports and insufflation pressures of 3–5 mmHg. We transfix and tie the fistula with a 4.0 braided nonabsorbable suture and perform the anastomosis with 5.0 braided absorbable sutures tied intracorporeally.

### 3.1. Statistical analysis

We grouped cases as 'cardiac' or 'noncardiac'. Univariate analysis of contingency tables was by Fisher's exact test. The distribution of continuous variables was examined in density plots and by the Shapiro–Wilk normality test. Normally distributed variables were compared with Student's t-test and nonparametric data with the Wilcoxon rank-sum test. Missing data were handled by removal from analysis.

Operative time and time to commencement of postoperative feeding were analyzed in general linear mixed models, fitting the presence or absence of cardiac anomalies, gestation, birth weight and age at operation as fixed variables, and surgeon and anesthetist as random variables. Results are reported here as estimated effect sizes, standard errors and 95% confidence intervals (95% CI). Statistical significance was taken as a 95% CI either entirely above or entirely below zero. P values for the mixed models were calculated by ANOVA. For data analysis and plots, we used the packages dplyr [26], ggplot2 [27] and lme4 [28] within the statistical program R [29].

## 4. Results

Of 49 EA repairs during the 9-year study period, 25 were thoracoscopic. Baseline characteristics of thoracoscopic and open cases were similar, as shown in Table 1. Of the thoracoscopic cases, 17 (68%) had associated anomalies including 9 (36%) infants with CHD. Four required surgical repair of CHD. Two of these had complete AV canal defects. There were no single ventricle heart defects. Of the open cases,

**Table 1**
Baseline characteristics in congenital heart disease (CHD) and non-CHD cases, by thoracoscopic or open surgery.

| | Thoracoscopic $n = 25$ | | Open $n = 24$ | | p[a] | p[b] | p[c] |
|---|---|---|---|---|---|---|---|
| | CHD $n = 9$ | No CHD $n = 16$ | CHD $n = 11$ | No CHD $n = 13$ | | | |
| Birth weight, mean grams (SD) | 2523 (490) | 2652 (594) | 2740 (699) | 2566 (984) | 0.56 | 0.56 | 0.85 |
| Gestation, median weeks (IQR) | 37 (1) | 38 (2) | 39 (3) | 37 (6) | 0.62 | 0.19 | 0.84 |
| Age at surgery, median days (IQR) | 1 (1) | 2 (1) | 1 (0) | 1 (2) | 0.003 | 0.26 | 0.13 |
| Any associated anomaly, *n* (%) | 9 (100) | 8 (50%) | 11 (100) | 6 (46%) | 0.02 | – | 1 |
| CHD, *n* (%) | 9 (100) | – | 11 (100) | – | – | – | 0.57 |
| Major CHD (surgically repaired), *n* (%) | 4 (44%) | – | 3 (27%) | – | – | 0.64 | 1 |

CHD, congenital heart disease.
[a] p value of CHD versus no CHD, thoracoscopic cases.
[b] p value of thoracoscopic versus open, CHD cases.
[c] p value of thoracoscopic versus all open, all cases.

**Table 2**
Associated anomalies in congenital heart disease (CHD) and non-CHD cases, by thoracoscopic or open surgery.

| | Thoracoscopic $n = 25$ | | Open $n = 24$ | |
|---|---|---|---|---|
| | CHD $n = 9$ | No CHD $n = 16$ | CHD $n = 11$ | No CHD $n = 13$ |
| Anorectal | 3(33%) | 2 (12%) | 1 (9%) | 0 |
| Duodenal atresia | 0 | 1 (6%) | 1 (9%) | 1 (8%) |
| Genetic syndrome[a] | 1 (11%) | 1 (6%) | 2 (18%) | 1 (8%) |
| Limb | 1 (11%) | 2 (12%) | 0 | 0 |
| Renal | 2 (22%) | 2 (12%) | 0 | 2 (15%) |
| Vertebral | 5 (55%) | 5 (31%) | 4 (36%) | 3 (23%) |
| Vascular[b] | 9 (100%) | 2 (12%) | 11 (100%) | 1 (8%) |
| Complete atrioventricular canal | 2 (22%) | – | 1 (9%) | – |
| Tetralogy of Fallot | 1 (11%) | – | 1 (9%) | – |
| Ventricular septal defect | 5 (56%) | – | 3 (27%) | – |
| Atrial septal defect | 1 (11%) | – | 8 (73%) | – |
| Coarctation of the aorta | 1 (11%) | – | 0 | – |
| TGA | 0 | – | 1 (9%) | – |
| Valvular (other) | 3 (33%) | – | 1 (9%) | – |

CHD, congenital heart disease.
[a] Two cases of trisomy 21, one trisomy 18, and two complex congenital anomaly.
[b] Vascular anomalies not included in the cardiac group were one dextrocardia with no other structural heart defect, one right sided aortic arch, and one bilateral superior vena cava.

two were performed by a 'thoracoscopic' surgeon; one of these patients had CHD, a complete AV canal defect with severe pulmonary hypertension. Associated anomalies are listed in Table 2.

On univariate analysis, thoracoscopic EA took longer to perform than open repair, as shown in Table 3. Neonates who had undergone thoracoscopic repair commenced feeds early in the postoperative period. On multivariate analysis using general linear mixed models, the relationship between the surgical approach and operative time remained significant at $p = 0.0009$, and time to postoperative feeds at $p = 0.03$.

Among thoracoscopic cases, there were no significant differences in time to feeding, intensive care stay, hospital stay, or rate of complications between CHD and non-CHD cases, as shown in Table 3. Data on time to feeding were unavailable for one patient because of death before feeding commenced. Two non-CHD cases were converted to open thoracotomy, one because of physiological instability and the other owing to a long gap. Anastomotic leak occurred in 5 (20%) and anastomotic strictures in 4 (16%). There was no recurrent fistula. Two infants died, one after thoracoscopic repair and one after open repair. Both were diagnosed in the postoperative period with nonsurvivable genetic anomalies diagnosed postoperatively leading to withdrawal of care.

The mean (SD) operative time for thoracoscopic EA repair (with or without CHD) was 217 (51) min. The presence of a cardiac anomaly did not influence operative time. The operative time was influenced by gestational age, being significantly longer in neonates less than 37 weeks' gestation compared to 37 weeks or more (median 260 versus 214 min, respectively).

## 5. Discussion

This study shows that congenital heart defects do not appear to compromise the perioperative outcomes of thoracoscopic EA repair. Our findings are provisional and await confirmation by larger, prospective studies; however, they do support continued use of the thoracoscopic approach by experienced anesthetic and surgical teams in the presence of a cardiac anomaly.

Thoracoscopic surgery presents a physiological challenge in neonates. Insufflated carbon dioxide passes across the pleural membrane to be absorbed into the blood stream. Raised intrathoracic pressure impairs ventilation leading to hypercapnea [30], this in a neonate whose respiration may already be compromised in the newborn period. External pressure on a neonate's chest and abdomen will restrict chest excursion and further compromise ventilation. For this reason, we take care to suspend the chest and abdomen by placing a small roll high in the axilla and a second larger roll under the pelvis when we position neonates for thoracoscopic EA repair.

The pressure of insufflation in the chest may also compromise cardiovascular function by reducing venous return to the heart, cardiac index and mean arterial pressure. Surgeons should maintain the insufflation pressure as low as practical. Evidence that thoracoscopic EA repair is physiologically worse than the open operation has been inconsistent. Researchers at Great Ormond Street Hospital raised concerns about the degree of hypercapnea and acidosis in thoracoscopic procedures in neonates [12–14]; however, others found no significant

**Table 3**
Operative data and outcomes in congenital heart disease (CHD) and non-CHD cases.

| | Thoracoscopic $n = 25$ | | Open $n = 24$ | | p[a] | p[b] | p[c] |
|---|---|---|---|---|---|---|---|
| | CHD $n = 9$ | No CHD $n = 16$ | CHD $n = 11$ | No CHD $n = 13$ | | | |
| Operative time (mean, SD min) | 211 (41) | 220 (57) | 148 (29) | 171 (51) | 0.65 | 0.002 | 0.0001 |
| Two surgeons ($n$, %) | 3 (33%) | 8 (50%) | 0 | 0 | 0.68 | 0.07 | 0.0002 |
| Conversion ($n$, %) | 0 | 2 (12%) | – | – | 0.61 | – | – |
| Days to feed (median, IQR) | 7 (3) | 7 (3) | 9 (5.5) | 11 (39) | 0.81 | 0.44 | 0.03 |
| NICU days (median, IQR) | 12 (3) | 11 (6) | 9 (5.5) | 17 (42) | 0.84 | 0.27 | 0.58 |
| Hospital days (median, IQR) | 16 (13) | 21 (20) | 16 (26) | 17 (54) | 0.46 | 1 | 0.92 |
| Leak ($n$, %) | 2 (22%) | 3 (19%) | 1 (9%) | 4 (31%) | 1 | 0.57 | 1 |
| Stricture ($n$, %) | 0 | 4 (25%) | 0 | 2 (15%) | 0.26 | – | 0.67 |
| Death ($n$, %) | 0 | 1 (6%) | 0 | 1 (8%) | 1 | – | 1 |
| Months of FU (median, IQR) | 38 (34) | 52 (69) | 48 (39) | 47 (30) | 0.84 | 0.94 | 0.76 |

CHD, congenital heart disease. NICU, neonatal intensive care unit. FU, length of follow-up.
[a] p value of CHD versus no CHD, thoracoscopic cases.
[b] p value of thoracoscopic versus open, CHD cases.
[c] p value of thoracoscopic versus all open, all cases.

differences in hypercapnea or acidosis between thoracoscopic and open EA repair [31]. In terms of outcomes, cerebral regional oxygen saturation ($rSO_2$) monitoring may be more meaningful than carbon dioxide levels [32]. At our institution, $rSO_2$ monitoring is used routinely during pediatric cardiac surgery. During thoracoscopic EA repair, low $rSO_2$ levels have been found by some [12] but not others [33,34].

In the presence of cardiac anomalies, the physiological effects of thoracoscopy could be critical. Hypercapnea and acidosis increase pulmonary vascular resistance, which increases strain the right heart and will increase a right to left shunt [35]. In some duct dependent CHDs, maintaining pulmonary vascular resistance can help to avoid pulmonary overcirculation [36,37]; thus, hypercapnea could be helpful. This is one reason to advise early neonatal repair of EA in duct dependent heart disease while pulmonary vascular resistance remains high [37]. Hypercapnea may exert a protective effect on the brain by increasing cerebral perfusion [24]. Given the complexity of thoracoscopic surgery in CHD, we recommend cases are performed with a pediatric cardiac anesthetist and cardiac monitoring.

Short-term outcomes after open EA repair do not appear to be negatively influenced by CHD. Ishimaru et al. found no significant difference in the anastomotic leak or stricture rate between CHD and non-CHD cases in a nationwide registry study from Japan [38]. Until now, no study has assessed the effect of CHD on outcomes after thoracoscopic EA repair.

Long-term outcomes after thoracoscopic procedures in neonates have not been well documented. Costerus et al. demonstrated normal development at two years of age after thoracoscopic EA repair [39]. Harmsen et al. demonstrated reduced motor development scores in children at 5 and 8 years of age, but normal intellectual development after open EA repair [40]. We are not aware of any outcome studies in school-aged children after thoracoscopic repair. In contrast, developmental outcomes after cardiac surgery for CHD are well documented. Survivors of infant cardiac surgery can experience cognitive, behavioral and social impairment; these children require ongoing surveillance, screening and evaluation [41]. The influence of any cardiac anomaly on development should be taken into account in follow-up studies after EA repair.

The present study is limited by its retrospective design and relatively small sample size. The results should be corroborated in future prospective studies. The choice between thoracoscopic and open approach was by surgeon preference. Selection of cardiac cases to thoracoscopic or open could lead to bias. The surgeons who routinely performed thoracoscopic EA repair did perform two open repairs during the study period. Although one case did not have CHD, the other had a severe defect associated with pulmonary hypertension, which certainly influenced the decision to avoid thoracoscopy. A multicenter design could help obtain an adequate sample to detect a difference in postoperative outcome. A further weakness of the present study was the lack of intraoperative blood gas data. As discussed above, others have shown significant acidosis and hypercapnea during EA repair. Regional cerebral oxygenation ($rScO_2$) monitoring was not routinely performed during the study period and, in those cases in which it had been performed, data had not been recorded thus was unavailable for analysis. It will be important to incorporate blood gas and $rScO_2$ data into the design of a future prospective study on thoracoscopic esophageal repair. Our results are further limited by the lack of data on long-term neurological and developmental outcomes.

In conclusion, we did not find evidence that the thoracoscopic approach to esophageal atresia is associated with longer operative time or compromised outcomes in children with congenital heart disease; however, the significant limitations of the paper preclude a recommendation for the safety of the thoracoscopic approach. Since this was a small uncontrolled study, the question of safety remains unanswered. We recommend a prospective multicenter study of thoracoscopic EA repair to analyze intraoperative physiology as well as short-term and long-term outcomes.

## Declarations of interest

None.

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