Respiratory function after birth in infants with congenital diaphragmatic hernia

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ABSTRACT

Objective To characterise the transitional pulmonary physiology of infants with congenital diaphragmatic hernia (CDH) using measures of expiratory tidal volume (TV) and end-tidal carbon dioxide ($ETCO_2$).

Design Prospective single-centre observational study.

Setting Quaternary neonatal intensive care unit.

Patients Infants with an antenatal diagnosis of CDH born at the Children's Hospital of Philadelphia.

Interventions TV and $ETCO_2$ were simultaneously recorded using a respiratory function monitor (RFM) during invasive positive pressure ventilation immediately after birth.

Main outcome measures TV per birth weight and $ETCO_2$ values were summarised for each minute after birth. Subgroups of interest were defined by liver position (thoracic vs abdominal) and extracorporeal membrane oxygenation (ECMO) treatment.

Results RFM data were available for 50 infants from intubation until a median (IQR) of 9 (7–14) min after birth. TV and $ETCO_2$ values increased for the first 10 min after birth, but intersubject values were heterogeneous. TVs were overall lower and $ETCO_2$ values higher in infants with an intrathoracic liver and infants who were ultimately treated with ECMO. On hospital discharge, survival was 88% (n=43) and 34% (n=17) of infants were treated with ECMO.

Conclusion Respiratory function immediately after birth is heterogeneous for infants with CDH. Lung aeration, as evidenced by expired TV and $ETCO_2$, appears to be ongoing throughout the first 10 min after birth during invasive positive pressure ventilation. Close attention to expired TV and $ETCO_2$ levels by 10 min after birth may provide an opportunity to optimise and individualise ventilatory support for this high-risk population.

WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ Infants with congenital diaphragmatic hernia (CDH) experience immediate physiological derangements after birth, but limited data inform our understanding of transitional physiology in this population.

WHAT THIS STUDY ADDS

⇒ Lung aeration, as evidenced by expired tidal volumes (TVs) and end-tidal carbon dioxide ($ETCO_2$) levels, appears to be ongoing throughout the first 10 min after the birth for infants with CDH during invasive positive pressure ventilation. Absolute measures of respiratory function immediately after birth are heterogeneous for these infants.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

⇒ Close attention to expired TV and $ETCO_2$ levels throughout the postnatal transition may provide an opportunity to optimise and individualise ventilatory support for this high-risk population.

INTRODUCTION

Congenital diaphragmatic hernia (CDH), a herniation of the abdominal viscera through a defect in the diaphragm, is a relatively common birth defect occurring in up to 1 in 2500 live births.[1 2] Mortality and morbidity of infants with CDH are significant and are related to the degree of pulmonary hypoplasia, size of diaphragmatic defect and associated anomalies.[2–7]

The wide clinical spectrum and severity of CDH make prenatal counselling and postnatal prognosis challenging. Prenatal measures such as lung to head ratio (LHR), observed to expected lung to head ratio (O/E LHR) and the position of the liver are used to predict CDH severity but are insufficient to individualise postnatal management.[8–10]

Immediately after birth, infants experience a dramatic cardiopulmonary transition, which results in a rapid decrease in pulmonary vascular resistance with increased pulmonary blood flow and arterial oxygen saturation levels. In infants with CDH, this physiological transition is poorly understood but is likely compromised due to pulmonary hypoplasia, pulmonary vascular hypoplasia and bowel gas distension in the chest that may impair lung expansion and pulmonary blood flow.

The delivery room management of infants with CDH is primarily based on expert opinion. Limited data guide delivery room interventions, and ventilatory outcomes from the delivery room are highly variable. Respiratory function monitoring (RFM) is an innovative tool to monitor and troubleshoot respiratory support during resuscitation, but it is not widely used in infants with congenital anomalies.[11–14] RFM uses an in-line sensor to measure and display key respiratory variables, including exhaled tidal volume (TV) and end-tidal carbon dioxide ($ETCO_2$) levels, in real time during ventilation. RFM during initial lung aeration may provide

insights into the postnatal transitional physiology during initial stabilisation after birth and as a result, allow for individualised care. This study sought to characterise the transitional postnatal pulmonary physiology of infants with CDH using measures of expiratory TV and $ETCO_2$.

METHODS

Design

This was a prospective single-centre observational study of all inborn infants with an antenatal diagnosis of CDH born at the Children's Hospital of Philadelphia between August 2020 and December 2021 who received active treatment.

Hospital resuscitation protocol

Per protocol, all infants with CDH are intubated immediately after birth, and intermittent positive pressure ventilation (PPV) with a T-piece ventilator is initiated with peak inspiratory pressures (PIPs) of 20–25 cmH_2O, positive end expiratory pressure of 5 cmH_2O and a ventilation rate of 40–50 breaths/min. Within this range, initial PIPs are selected based on the anticipated severity using prenatal measures such as liver position, LHR and O/E LHR. Subsequent ventilatory settings are adjusted as needed based on clinical discretion throughout resuscitation. For infants with an abdominal liver position, initial FiO_2 is 0.3 and infants are transitioned to a conventional ventilator as soon as the endotracheal tube is secured. For infants with a thoracic liver position or a right-sided CDH, initial FiO_2 is typically 0.5 and infants are transitioned to high-frequency oscillatory ventilation (HFOV) after the endotracheal tube is secured. FiO_2 is titrated to achieve preductal oxygen saturation goals of 85% by 10 min after birth. Following intubation and gastric decompression, umbilical lines are placed and an arterial blood gas is obtained.

Monitoring equipment

RFM measurements were recorded and calculated with a Philips NM3 Respiratory Profile Monitor (Koninklijke Phillips NV, Amsterdam, The Netherlands) with the sensor placed between the endotracheal tube and respiratory device immediately after intubation. Expiratory TV, peak inflation pressure, positive end expiratory pressure and $ETCO_2$ were simultaneously recorded. Recordings were analysed for the first 10 min after birth as that was the duration of time with T-piece ventilation where consistent data were available for all infants prior to transition to a ventilator. The first 10 min after birth also reflects the most acute changes in physiology, consistent with Apgar scoring and target oxygen saturation goals in the delivery room. Exhaled TVs were summarised for each minute after birth. Characteristics of individual inflations (such as leak) were not assessed. As an exploratory analysis, we summarised respiratory function measurements beyond 10 minutes after birth.

All remaining covariates were abstracted from the resuscitation record, which is documented in real time by a dedicated recording nurse, or from the medical record.

Main outcome measures

Statistical analysis included summary statistics of demographic data. Subgroups of interest were defined by liver position on antenatal imaging (thoracic vs abdominal) and extracorporeal membrane oxygenation (ECMO) treatment at any time during the hospitalisation. RFM variables were summarised for each minute after birth and compared between subgroups using the non-parametric Wilcoxon rank-sum test. A non-parametric regression with a locally weighted scatterplot smoothing (LOWESS) line was fitted for TV per birth weight and $ETCO_2$ values at each minute after birth. The default span length of 0.75 was used to create the LOWESS curve. While lower values of span were considered, they resulted in volatile curves that deviated from the overall trend of the data. All analyses were conducted in R V.4.1.2. We considered the results from the Wilcoxon rank-sum test to be statistically significant if the $p<0.05$. Infants were followed for inpatient outcomes such as ECMO treatment and survival to hospital discharge.

Table 1 Characteristics of infants with congenital diaphragmatic hernia (n=50)

Characteristic	n (%), mean±SD, median (IQR)
Sex (male)	25 (50)
CDH side (left)	39 (78)
Liver position (up)	33 (66)
Mode of delivery (vaginal)	26 (52)
Gestational age (weeks)	37.8±1.7
Birth weight (kg)	3.0±0.5
LHR*	0.87 (0.60–1.17)
O/E LHR*‡ (%)	37.0 (28.4–50.6)
1 min Apgar	7 (5–8)
5 min Apgar	8 (7–9)
Time to preductal SpO_2>85% (min)	9 (6–9)
Time to HR>100 beats/min (min)	0 (0–1)
Time to intubation (min)	1 (1–2)
First arterial $PaCO_2$ (mm Hg)	62 (48–76)
Liver up subgroup	60 (48–82)
Liver down subgroup	62 (50–69)
First postductal arterial PaO_2 (mm Hg)	48 (39–67)
Liver up subgroup	44 (38–67)
Liver down subgroup	53 (48–67)

*LHR and O/E LHR were measured by mid-trimester ultrasound using the trace method of the TOTAL trial.[10]
†LHR available for all 50 infants, O/E LHR available for 49 infants; one infant was diagnosed >32 weeks gestational age and the TOTAL trial calculator has not been validated beyond 32 weeks gestational age.[10]
CDH, congenital diaphragmatic hernia; HR, heart rate; LHR, lung to head ratio; O/E, observed to expected LHR; $PaCO_2$, arterial carbon dioxide tension; PaO_2, arterial oxygen tension; SpO_2, oxygen saturation.

RESULTS

Among 55 infants with CDH born between August 2020 and December 2021 who received active treatment, 50 infants were included in this study. Data were not obtained for five infants due to insufficient time for equipment set-up or atypical resuscitation (two infants were born via precipitous vaginal delivery, two infants were born via urgent caesarean section for fetal intolerance of labour and one infant was born immediately following the removal of fetal endoscopic tracheal occlusion balloon). Overall, 66% (n=33) of infants had a thoracic liver position with a median LHR of 0.87 and a median O/E LHR of 37% using the trace method of the TOTAL trial (table 1).[10] The median (IQR) time to intubation was 1 min (1–2) after birth. Survival to discharge was 88% (n=43/49) with one infant not yet discharged, and 34% (n=17) of infants were treated with ECMO.

RFM data were available for 50 infants from the time of intubation until a median (IQR) of 9 (7–14) min after birth. Expiratory TVs per birth weight and $ETCO_2$ values are shown in table 2. In subgroup analysis, TVs were significantly lower from

Table 2 Expiratory tidal volumes by BW and $ETCO_2$ levels by minute after birth

Minute after birth	Tidal volume by BW (mL/kg), median (IQR)	$ETCO_2$ (mm Hg), median (IQR)
1 n=16	1.33 (1.00–2.25)	39.0 (24.8–58.0)
2 n=35	2.00 (1.07–2.58)	48.0 (33.0–60.5)
3 n=42	2.25 (1.33–3.29)	58.5 (46.3–65.5)
4 n=46	2.86 (2.06–4.67)	55.5 (45.3–66.5)
5 n=47	3.00 (2.35–4.72)	61.0 (46.5–68.0)
6 n=40	3.20 (2.53–5.25)	58.0 (46.0–67.3)
7 n=40	3.55 (2.33–5.05)	58.0 (46.0–64.3)
8 n=36	4.00 (2.75–5.22)	56.5 (44.5–63.0)
9 n=33	4.00 (2.59–6.00)	52.0 (43.0–64.0)
10 n=28	4.27 (2.81–5.08)	50.0 (45.0–63.3)

BW, birth weight; $ETCO_2$, end tidal carbon dioxide level.

minutes 2 to 7 after birth in infants who were ultimately treated with ECMO and were significantly lower from minutes 5 to 7 after birth in infants with the liver in the thoracic cavity as shown in online supplemental table 1. $ETCO_2$ values were significantly higher at multiple time points after birth in infants with the liver in the thoracic cavity but were only significantly higher from minutes 5 to 6 in infants who were ultimately treated with ECMO as shown in online supplemental table 2. TVs per birth weight and $ETCO_2$ values for each minute are shown in figures 1 and 2. While both values increased throughout the first 10 min after birth in aggregate, intersubject values at each minute were heterogeneous across the population. TVs were overall lower and $ETCO_2$ values higher in infants with the liver in the thoracic cavity and infants who were ultimately treated with ECMO. TVs per birth weight and $ETCO_2$ values for each minute for infants based on O/E LHR are shown in online supplemental figures 1 and 2. Limited data available beyond the 10-min study period are shown in online supplemental table 3; these should be interpreted cautiously given the decreasing sample size.

DISCUSSION

We characterised the transitional pulmonary physiology of 50 infants with CDH and found that respiratory function immediately after birth is heterogeneous in this population despite a standardised delivery room resuscitation protocol. Initial lung aeration, as evidenced by expired TVs and $ETCO_2$ levels, appears to be ongoing throughout the first 10 min after birth during invasive PPV. TVs were lower and $ETCO_2$ levels higher in infants who were ultimately treated with ECMO and in infants with an intrathoracic liver. TVs in the first minutes after birth depend on lung compliance and resting lung volume, which vary considerably in infants with pulmonary hypoplasia.[14 15] Previous RFM studies demonstrated that TVs during spontaneous breathing could be used to estimate the severity of pulmonary hypoplasia among infants with CDH.[15] Lower TVs and compliance in the first minutes after birth have been associated with mortality, chronic lung disease and need for inhaled nitric oxide.[14–16]

O'Rourke *et al* studied the first and last minutes of invasive PPV during delivery room resuscitation in infants with CDH; differences in pulmonary mechanics were evident between survivors and non-survivors as early as the first minute. They reported that TVs >2.1 mL/kg in the first minute and >3.8 mL/kg in the last minute predicted survival.[16] Mank *et al* studied expired TVs of spontaneous breaths following intubation in infants with CDH for the first 10 min after intubation and reported that TVs <2.2 mL/kg predicted mortality and morbidity.[15] For comparison, median TVs in our cohort were 1.33 mL/kg in the first minute and 4.27 mL/kg by 10 min after birth.

The minimum pressures needed to aerate the newborn lung in the first minutes after birth without causing barotrauma are unknown. Initial PIPs in our hospital protocol are lower than those reported by O'Rourke *et al*, where median PIPs were >30

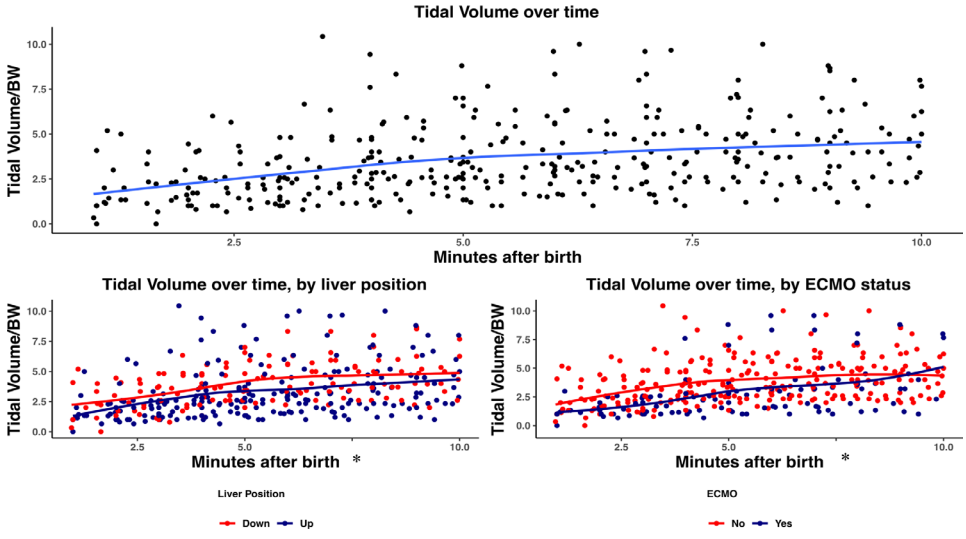

Figure 1 Tidal volume (per birth weight (BW)) over time for the first 10 min after birth. Top panel shows the entire cohort, and lower panels demonstrate subgroups based on antenatal liver position and postnatal extracorporeal membrane oxygenation (ECMO) treatment. A locally weighted scatterplot smoothing (LOWESS) line is shown. *TVs were significantly lower from minutes 5 to 7 ($p<0.05$) in infants with the liver in the thoracic cavity and from minutes 2 to 7 ($p<0.05$) in infants who were ultimately treated with ECMO.

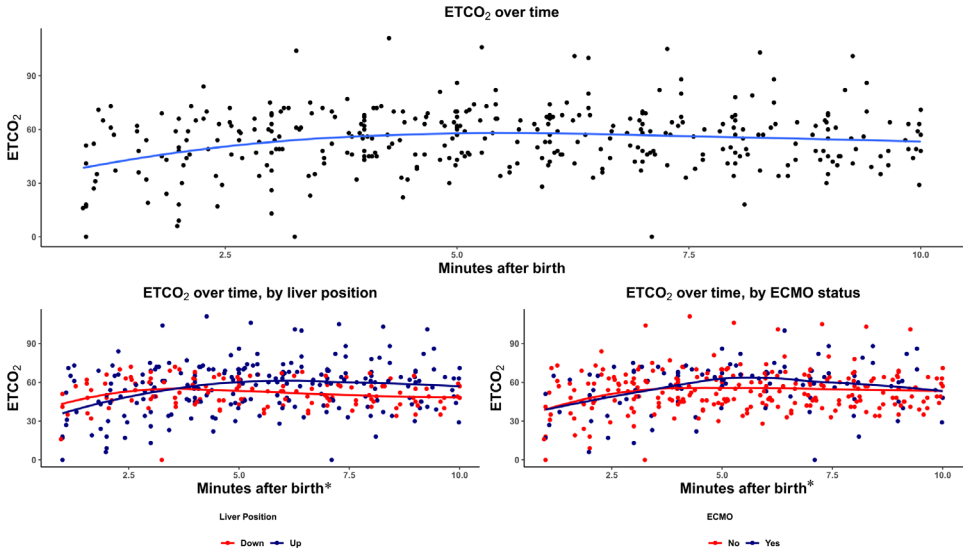

Figure 2 End-tidal carbon dioxide level ($ETCO_2$) over time for the first 10 min after birth. Top panel shows the entire cohort, and lower panels demonstrate subgroups based on antenatal liver position and postnatal extracorporeal membrane oxygenation (ECMO) treatment. A locally weighted scatterplot smoothing (LOWESS) line is shown. *$ETCO_2$ levels were significantly higher ($p<0.05$) at multiple time points in infants with the liver in the thoracic cavity but were only significantly higher from minutes 5 to 6 ($p<0.05$) in infants who were ultimately treated with ECMO.

cmH_2O for both survivors and non-survivors at both the first and last minutes of RFM recording.[16] The lower PIPs used in our hospital resulted in highly variable observed TVs, even within high-risk subgroups such as infants with an intrathoracic liver. These findings highlight the heterogeneous transitional physiology in this population and suggest a role for individualised care immediately after birth.

We also assessed $ETCO_2$ levels, which are not as accurate as serum samples, particularly in infants with CDH who may experience impaired pulmonary blood flow. In a recent study by Gien *et al*, three arterial blood gases obtained within the first hour after birth demonstrated that an insidious onset of acidaemia and a mixed respiratory and metabolic acidosis occur in infants with CDH by 60 min after birth.[17] Our data showed that $ETCO_2$ levels increased over the first 5 min after birth and then stabilised. However, as arterial CO_2 levels were not obtained until after umbilical line placement at around 60 min after birth, we were unable to correlate $ETCO_2$ levels with simultaneous arterial CO_2 levels. While extensive work has been done to characterise the postnatal pulmonary transition in lamb models of CDH, these models may not reflect the heterogeneity of the human condition. Kashyap *et al* initiated sustained inflation followed by intermittent PPV in a lamb model of CDH, clamping the umbilical cord only when a TV of 4 mL/kg had been achieved or at 10 min after birth. Only two out of seven CDH lambs reached the target TV of 4 mL/kg at any point (at 20 and 25 min), whereas all six control non-CDH lambs reached the target at approximately 5 min. Infants in our cohort underwent immediate cord clamping and by 10 min after birth, the median (IQR) TV was 4.27 mL/kg (2.81–5.08).[18] Such discrepancies emphasise the need for caution when applying knowledge from preclinical models to human physiology. Given the physiological differences between preclinical models of surgically created CDH and humans, clinical studies are needed to better characterise transitional physiology among newborn infants with CDH.

Our findings confirm that measures indicating more severe pulmonary hypoplasia are associated with worse outcomes, such as ECMO treatment. However, there are important differences between our work and previous reports: we assessed repeated measures of expired TVs during mandatory invasive PPV during initial resuscitation in the largest cohort of CDH infants studied in the delivery room. Median TVs increased from 1.33 mL/kg at 1 min after birth to 4.27 mL/kg by 10 min after birth in our study suggesting that the timing of measurement is important to contextualise RFM measurements for this population in the delivery room setting.

Future work could use real-time RFM measurements to improve both oxygenation and ventilation outcomes. Closer attention to TVs during the first inflations after delivery is critical to optimise the postnatal transition and choose the best ventilation strategy. This line of investigation is already underway in preterm infants, where the role of RFM is being investigated in the delivery room.[11–13] The utility of RFM monitoring to guide ventilatory support in real time during resuscitation of infants with CDH merits further investigation. While our study was not blinded and clinicians could view RFM data, resuscitations occurred without clinicians using the data to change practice. In addition, RFM measurements could ultimately improve CDH prediction models, which currently rely on basic information from antenatal imaging and demographics.

We acknowledge limitations of our observational study. TV and $ETCO_2$ levels are not the only measures that contribute to lung aeration; we used these readily available non-invasive measures as proxies of lung aeration. RFM data were only collected once the endotracheal tube was in place and during T-piece PPV; they were not available at every minute assessed for each infant. While RFM can continue while using the conventional ventilator, it is not compatible with the use of HFOV; thus, data were not available beyond 10 min after birth for infants who were transitioned to HFOV. The available data demonstrated a median TV of 5.4 mL/kg by 20 min after birth, but these findings should be interpreted cautiously given the limited sample size. More consistent data beyond 10 min after birth may have provided interesting information on ongoing trends. Mindful of the potential non-random nature of missing data (more severe infants were likely placed on HFOV sooner) and the small sample size, we did not attempt to fit longitudinal regression models, but used the LOWESS method to evaluate the

general trend in the data. Given the flexible nature of LOWESS curves that change with the amount of data, patterns around the edges of the data should be interpreted cautiously since data may be sparse at those points.

There are many study strengths including a description of the postnatal physiological transition of the largest sample of infants with CDH using repeated measures of expired TVs during invasive PPV. Our institution is a large delivery and referral centre. While many infants are classified as severe prenatally, the LHR and liver position data support that our cohort encompassed a wide spectrum of severity. Finally, data were obtained for 91% of eligible infants including detailed demographics and outcomes, increasing the generalisability of our findings.

CONCLUSION

Respiratory function immediately after birth is heterogeneous for infants with CDH. Lung aeration, as evidenced by expired TVs and $ETCO_2$ levels, appears to be ongoing throughout the first 10 min after birth during invasive PPV. Close attention to expired TVs and $ETCO_2$ levels by 10 min after birth may provide an opportunity to optimise and individualise ventilatory support for this high-risk population.

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Contributors KTW and EEF conceptualized and designed the study, collected and analysed the data, drafted the initial manuscript, and reviewed and revised the manuscript. LS, KM, EK and KTVH collected data and reviewed and revised the manuscript. LM, HLH, NER, AA, MAP, HBP and JF assisted in designing the study, analysing the data, and reviewed and revised the manuscript. EEF provided oversight and serves as the guarantor for the article. All authors approved the final manuscript as submitted and agree to be accountable for all aspects of the work.

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Competing interests None declared.

Ethics approval This study was deemed exempt by the Institutional Review Board (IRB 20–0 17 675) with a waiver of parental consent.

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REFERENCES

1 Langham MR, Kays DW, Ledbetter DJ, *et al*. Congenital diaphragmatic hernia. *Clin Perinatol* 1996;23:671–88.
2 Shanmugam H, Brunelli L, Botto LD, *et al*. Epidemiology and prognosis of congenital diaphragmatic hernia: a population-based cohort study in Utah. *Birth Defects Res* 2017;109:1451–9.
3 Rygl M, Pycha K, Stranak Z, *et al*. Congenital diaphragmatic hernia: onset of respiratory distress and size of the defect: analysis of the outcome in 104 neonates. *Pediatr Surg Int* 2007;23:27–31.
4 , Lally KP, Lally PA, *et al*, Congenital Diaphragmatic Hernia Study Group. Defect size determines survival in infants with congenital diaphragmatic hernia. *Pediatrics* 2007;120:e651–7.
5 Kardon G, Ackerman KG, McCulley DJ, *et al*. Congenital diaphragmatic hernias: from genes to mechanisms to therapies. *Dis Model Mech* 2017;10:955–70.
6 Wang Y, Honeyford K, Aylin P, *et al*. One-Year outcomes for congenital diaphragmatic hernia. *BJS Open* 2019;3:305–13.
7 Rygl M, Pycha K, Stranak Z, *et al*. Congenital diaphragmatic hernia: onset of respiratory distress and size of the defect: analysis of the outcome in 104 neonates. *Pediatr Surg Int* 2007;23:27–31.
8 Hedrick HL, Danzer E, Merchant AM, *et al*. Liver position and lung-to-head ratio for prediction of extracorporeal membrane oxygenation and survival in isolated left congenital diaphragmatic hernia. *Am J Obstet Gynecol* 2007;197:422.e1–422.e4.
9 Snoek KG, Peters NCJ, van Rosmalen J, *et al*. The validity of the observed-to-expected lung-to-head ratio in congenital diaphragmatic hernia in an era of standardized neonatal treatment; a multicenter study. *Prenat Diagn* 2017;37:658–65.
10 Dekoninck P, Gratacos E, Van Mieghem T, *et al*. Results of fetal endoscopic tracheal occlusion for congenital diaphragmatic hernia and the set up of the randomized controlled TOTAL trial. *Early Hum Dev* 2011;87:619–24.
11 Pahuja A, Hunt K, Murthy V, *et al*. Relationship of resuscitation, respiratory function monitoring data and outcomes in preterm infants. *Eur J Pediatr* 2018;177:1617–24.
12 Hunt KA, Murthy V, Bhat P, *et al*. Tidal volume monitoring during initial resuscitation of extremely prematurely born infants. *J Perinat Med* 2019;47:665–70.
13 van Zanten HA, Kuypers KLAM, van Zwet EW, *et al*. A multi-centre randomised controlled trial of respiratory function monitoring during stabilisation of very preterm infants at birth. *Resuscitation* 2021;167:317–25.
14 te Pas AB, Kamlin COF, Dawson JA, *et al*. Ventilation and spontaneous breathing at birth of infants with congenital diaphragmatic hernia. *J Pediatr* 2009;154:369–73.
15 Mank A, Carrasco Carrasco C, Thio M, *et al*. Tidal volumes at birth as predictor for adverse outcome in congenital diaphragmatic hernia. *Arch Dis Child Fetal Neonatal Ed* 2020;105:F1–5.
16 O'Rourke-Potocki A, Ali K, Murthy V, *et al*. Resuscitation of infants with congenital diaphragmatic hernia. *Arch Dis Child Fetal Neonatal Ed* 2017;102:F320–3.
17 Gien J, Palmer C, Liechty K, *et al*. Early abnormalities in gas exchange in infants with congenital diaphragmatic hernia. *J Pediatr* 2022;243:188–92.
18 Kashyap AJ, Crossley KJ, DeKoninck PLJ, *et al*. Neonatal cardiopulmonary transition in an ovine model of congenital diaphragmatic hernia. *Arch Dis Child Fetal Neonatal Ed* 2019;104:F617–23.