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ORIGINAL ARTICLE

Diaphragmatic paralysis among very low birth weight infants following ligation for patent ductus arteriosus

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Abstract Management of diaphragmatic paralysis (DP) among newborn infants remains controversial, especially for very low birth weight (VLBW) infants following ligation for patent ductus arteriosus (PDA). This study aimed to characterize the impact of DP after PDA ligation among VLBW infants. Clinical characteristics of DP cases treated with either diaphragmatic plication or conservative methods were described as well. The medical records of VLBW infants who underwent PDA ligation in Chang Gung Memorial Hospital between January 2000 and December 2011 were retrospectively reviewed, and DP was suspected if postligation chest X-rays showed an elevation of the left diaphragm as confirmed by a chest ultrasonograph. For each DP case, three other infants that received PDA ligation with proximate birth dates and who were closely matched in terms of gestational age (±1 week) and birth weight (±10 %) were selected as the control group. A total of eight preterm infants were diagnosed as having DP and 24 infants were selected as the control group. The affected infants usually presented with respiratory distress and extubation failure. The study demonstrated that, among our patient population, DP was associated with a significantly longer duration of ventilator dependency (56.1±16.0 vs. 29.8± 17.7 days, p=0.001) and a higher incidence of severe bronchopulmonary dysplasia (87.5 vs. 23 %, p=0.002). For selective infants with DP-related ventilatory failure after PDA ligation, surgical plication may facilitate extubation. Diaphragmatic paralysis should be evaluated carefully among VLBW infants receiving PDA ligation because of its adverse impact on ventilator dependency and correlation to a higher incidence of severe bronchopulmonary dysplasia.

Keywords Bronchopulmonary dysplasia · Diaphragmatic paralysis · Patent ductus arteriosus · Preterm infants · Very low birth weight · Plication

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Introduction

Acquired diaphragmatic paralysis (DP), usually a consequence of phrenic nerve injury, is not uncommon during birth [25] or following iatrogenic procedures such as chest tube insertion or cardiopulmonary surgery [19]. Unlike adults or older children, diaphragmatic movement is critical in the breathing of infants, and thus infants usually present with respiratory illnesses if the diaphragm becomes paralyzed or decreases in movement. Immediate plication of the paralyzed diaphragm has been suggested, but many other articles agree to a 2-week observation period for spontaneous recovery in the pediatric population [10, 27].

Patent ductus arteriosus (PDA) is a common problem in preterm infants, especially for very low birth weight (VLBW, <1,500 g) infants. Since PDA ligation is the most common cardiovascular surgery performed in this group, we

therefore focused on the DP issue among VLBW infants following PDA ligation. Common complications of PDA ligation include air leak syndrome, pulmonary edema, hypotension requiring vasopressor support, and recurrent laryngeal nerve palsy [5, 21]. Recurrent laryngeal nerve palsy-associated left-sided vocal cord paralysis is a well-known morbidity following PDA ligation, and the incidence has been reported as up to 40 % in extremely low birth weight infants [4]. On the other hand, diaphragmatic paralysis following PDA ligation has only been mentioned in a few articles [3, 14, 17, 18, 31]. Furthermore, the increasing requirement of oxygen after surgical ligation will participate in the development of bronchopulmonary dysplasia (BPD) and retinopathy of prematurity (ROP) as well [26].

Management of DP among newborn infants is still controversial, particularly for VLBW infants with DP following PDA ligation [10, 12, 24, 27]. Oxygen plays an important role in the pathogenesis of morbidities in preterm infants; therefore, we queried if hopeful waiting should be applied in this vulnerable population. Since there was no comprehensive and recent information on this issue, we conducted this study to compare the outcomes of VLBW infants with and without DP following PDA ligation. We also compared the effect among subgroups of infants treated conservatively and treated with surgical plication. To minimize the confounding effects of gestational age (GA) and birth weight (BW), a case–control method was used.

Materials and methods

This case–control study was conducted in the neonatal intensive care unit (NICU) at Chang Gung Memorial Hospital and used the NICU database. The database recorded all infants' medical information from admission to the NICU until discharge. Medical records of VLBW infants who underwent PDA ligation during the period between January 2000 and December 2011 were retrospectively reviewed.

Infants with hemodynamically significant PDA who failed or were contraindicated to pharmacological treatments were selected to receive PDA ligation. Routine postoperative chest X-rays were arranged for every infant after PDA ligation and then followed concordantly with the clinical situation. All of these postoperative radiographs were inspected. Diaphragmatic paralysis was suspected in infants if the postoperative chest X-ray showed an elevation of the left diaphragm one intercostal space higher than the right side. All suspected cases were confirmed by chest ultrasonographs and, if decreased diaphragmatic movement or paralysis was found, were assigned to the study group. For each DP case, three other compatible infants who received PDA ligation with a near date of birth and closely matched for GA (±1 week) and BW (±10 %) were selected from the cohort to form the control group (n=24), giving a matching ratio of 3:1. The control patients were also selected to match the study patient in terms of resuscitation at delivery, presence of severe IVH (IVH grades III–IV) in the first 3 days of life, and early-onset sepsis.

Antenatal, perinatal, and neonatal data including GA, BW, gender, mode of delivery, Apgar scores, need of resuscitation at delivery, presence of respiratory distress syndrome (RDS), use of artificial surfactant, demand for oxygen, days of oxygen dependency, hospital stay, and survival were recorded. Age at diagnosis and surgical ligation for PDA, age at identification of diaphragmatic paralysis, age at diaphragmatic plication if done, and age at extubation were recorded as well. Major comorbidities of prematurity such as BPD, ROP/severe ROP (stages 3–5), NEC (≧stage II), and IVH/severe IVH (grades III–IV) were documented. The definition for BPD was in accordance with 2001 NICHD criteria [11], in which for infants with GA <32 weeks BPD is diagnosed with the need for supplemental oxygen ≧28 days and the severity is evaluated at 36 weeks postmenstrual age as mild (breathing room air), moderate (need for <30 % oxygen more than 12 h daily), and severe (need for ≧30 % oxygen more than 12 h daily and/or positive pressure). Positive pressure means positive pressure ventilation or nasal continuous positive airway pressure (CPAP), but not high-flow nasal cannula. Infants treated with oxygen less then 28 days and infants with respiratory support only for apnea but not for lung parenchymal disease were not considered to have BPD. Retinopathy of prematurity was classified according to the international classification of retinopathy of prematurity [1]. Intraventricular hemorrhage was graded by Papile's classification system [23].

Statistical analysis was performed using IBM SPSS Statistics version 19.0. Categorical data were analyzed using the standard χ^2-test or Fisher's exact test where appropriate. Continuous data were analyzed using the independent t-test for between-group comparisons. Because of the nature of our independent variables and small sample size, simple linear or logistic regression was not applied. Statistical significance was defined as p <0.05. The statistical power was calculated by using G*Power 3.1 program and assessing at least 0.8 as standard of adequacy.

Results

A total of eight VLBW infants who underwent PDA ligation met the criteria for diaphragmatic paralysis during the past 12 years (Table 1). During this period, a total of 236 VLBW infants received PDA ligation by either of the two cardiovascular surgeons with similar surgical technique. Left mid-axillary mini-thoracotomy was performed through the fourth intercostal space with the muscle-sparing method (i.e., preserving the latissimus dorsi). The pleural cavity was entered and the mediastinal pleura over the proximal descending

Table 1 Characteristics of VLBW infants with diaphragmatic paralysis following PDA ligation

Case	Gender	GA (week)	BW (g)	Mode of delivery	Apgar score (1)	Apgar score (5)	Usage of artificial surfactant	Ligation age (day)	DP (identified age, day)	Plication (age, day)	Ventilator dependency (day)	Oxygen dependency (day)	Hospital stay(day)	Morbidities
1	M	28	760	CS	5	7	Yes	11	Left (29)	No	57	82	101	Severe BPD, ROP stage 3
2	M	27	1,220	CS	4	7	Yes	13	Left (43)	No	67	90	98	Severe BPD, ROP stage 3
3	M	29	1,000	VD	5	6	Yes	22	Left (40)	No	55	63	73	Severe BPD, ROP stage 2
4	M	28	1,480	CS	3	6	Yes	15	Left (21)	No	36	53	78	Mild BPD
5	M	26	1,040	VD	5	7	Yes	9	Left (13)	Yes (67)	71	93	104	Severe BPD, ROP stage 3
6	M	29	1,280	VD	5	6	Yes	9	Left (13)	Yes (31)	34	70	77	Severe BPD
7	F	27	1,340	CS	1	4	Yes	7	Left (34)	Yes (38)	50	132	150	Severe BPD, ROP stage 2
8	M	26	790	CS	5	8	Yes	24	Left (28)	No	79	98	125	Severe BPD, ROP stage 3

GA gestational age, *BW* birth weight, *DP* diaphragmatic paralysis, *CS* Cesarean section, *VD* vaginal delivery, *BPD* bronchopulmonary dysplasia, *ROP* retinopathy of prematurity

thoracic aorta was opened longitudinally. The ductus was isolated, looped, and ligated with pledgeted sutures. A chest tube was routinely placed intraoperatively and usually removed after 24 h if no air leak, hemothorax, or chylothorax ensued. Incidence of postligation DP was estimated to be 3.4 % among VLBW infants in our study. Seven out of the eight infants were male. Gestational age ranged from 26 to 29 weeks, and BW varied from 760 to 1,480 g. All affected infants had a low 1-min Apgar score and required intubation and mechanical ventilation after birth. Development of RDS was universal. Artificial surfactant was administrated at least once for each DP case. Ductal ligation was performed at an average of 13.8 days of chronological age (range, 9 to 24 days). The mean weight at the time of ligation was 1,005 g (860 to 1,420 g). All target patients were identified and confirmed with left unilateral diaphragmatic paralysis within 1 month following surgery, accompanied by varied respiratory symptoms, including respiratory distress and extubation failure.

Twenty-four patients were recruited into the control group. There was no significant difference in terms of GA, BW, mode of delivery, Apgar scores, usage of artificial surfactant, and age at ligation between DP and control group (Table 2). Male babies were predominant in the DP group; however, there was no statistical difference.

Comparing outcomes and major complications, our study demonstrated that the duration for infants being mechanically ventilated was significantly longer in DP cases (56.1± 16.0 vs. 29.8±17.7 days, p=0.001, power=0.96), which could result in a higher incidence of severe BPD (87.5 vs. 23 %, p=0.002, power=0.93) (Table 3). The days of oxygen dependency and hospital stay were longer in DP cases but without statistical difference. By the 2001 NICHD definition, the diagnosis of BPD was very common in both DP and control groups (100 and 92 %, respectively), but dissimilarity emerged if assessed by severity; the longer duration for infants being mechanically ventilated, the higher the incidence to develop severe BPD. The incidence of threatening/severe ROP (≧stage 3) was 50 % in our DP cases; however, there was no statistical significance (50 vs. 17 %,

Table 2 Demographic data of DP cases and control group

	DP cases (n=8)	Control group (n=24)	p-value
Gender, male	7/8 (88 %)	13/24 (54 %)	0.204
Gestational age (week)	27.5±1.2	27.7±1.2	0.673
Birth weight (g)	1,114±260	1,088±218	0.788
Mode of delivery, VD	3/8 (38 %)	10/24 (42 %)	1.000
Apgar score at 1 min	4±2	5±2	0.063
Apgar score at 5 min	6±1	7±1	0.075
Usage of artificial surfactant	8/8 (100 %)	22/24 (92 %)	1.000
Age at PDA ligation (day)	13.8±6.3	11.3±7.0	0.376

Table 3 Outcomes and major complications between DP cases and control group

	DP cases (n=8)	Control group (n=24)	p-value
Demand for oxygen			
Duration of intubation (day)	56.1 ± 16	29.8 ± 17.7	0.001*
Oxygen dependency (day)	86.8 ± 22.4	70.2 ± 26.8	0.128
Hospital stay (day)	100.8 ± 26.4	84.3 ± 22.8	0.1
Major complication			
BPD	8/8 (100 %)	22/24 (92 %)	1
Mild	1 (12.5 %)	6 (27 %)	
Moderate	0	11 (50 %)	
Severe	7 (87.5 %)[a]	5 (23 %)[b]	0.386[c], 0.002[d]*
ROP (≧stage 3)	4/8 (50 %)	4/24 (17 %)	0.152

PDA patent ductus arteriosus, *BPD* bronchopulmonary dysplasia, *ROP* retinopathy of prematurity, *CPAP* continuous positive airway pressure, *NA* not applicable

*p<0.05 (indicates statistical significance)

[a] Among DP cases with severe BPD, six were with CPAP support and one was still intubated at 36 weeks' postmenstrual age

[b] Among the control group with severe BPD, three were with CPAP support and two were still intubated at 36 weeks' postmenstrual age

[c] Compare the incidence of moderate and severe BPD between the two groups

[d] Compare the incidence of severe BPD only between the two groups

p=0.152). No infant in either group had severe IVH. All DP cases in our study survived.

In the current study, three of the eight DP cases had received diaphragmatic plication (cases 5, 6, and 7) and others were treated conservatively (Table 1). Diaphragmatic plication was executed via thoracotomy though a minimally invasive wound at the sixth intercostal space. These three cases were extubated successfully 3, 4, and 12 days (mean, 6.3 days) after diaphragmatic plication, respectively. However, the total duration of ventilator dependency (51.7±18.6 vs. 58.8±15.9 days, p=0.61) and the duration of postligation ventilator dependency (43.3±18.5 vs. 41.8±14.6 days, p=0.91) were not significantly different between groups. Also worthy to note is the fact that the patient in case 5 had not received diaphragmatic plication until weaning from the ventilator failed three times, which led to a longer duration (71 days) of ventilator dependency.

Discussion

The overall incidence of diaphragmatic paralysis in children following cardiac surgery is estimated at 1.2 to 4.6 % retrospectively [6, 7, 10, 13, 15, 16, 22]. The most notorious operative procedure is the creation of the Blalock–Taussig shunt [24, 26]. The first case in literature reporting DP following PDA ligation was in 1977 [31]. Benjacholmas et al. described short-term outcomes of PDA ligation in 42 preterm infants and found that one infant had DP and needed surgical plication [3]. To date, only a few cases were reported in the literature in this field [14, 17, 18]. The reason was speculated to be its rare occurrence; however, it could be due to small sample size as well. With critical review and a larger population in our study, it seemed that DP incidence among VLBW infants was as high as that in other pediatric populations after cardiovascular surgery. Based on our findings, DP should be considered and diaphragmatic movement should be evaluated if VLBW infants develop respiratory distress and extubation failure after PDA ligation.

Our study is the first study using case–control methods to systemically discuss diaphragmatic paralysis following PDA ligation in VLBW infants. We demonstrated that VLBW infants with DP indeed have a longer duration of ventilator dependency and a higher incidence of developing severe BPD. Since infants with severe BPD are not only at high risk for pulmonary morbidity and mortality during the first 2 years of life but also anticipated to have long-term neurologic and motor sequelae [29], we believe that these findings merit more attention and need more aggressive therapeutic strategies.

Previous data showed that one third of paralyzed diaphragms will recover spontaneously by a maximum seven weeks and others will become less symptomatic [6, 16]. Notwithstanding, the situation might differ in infants who are vulnerable to respiratory complications related to DP due to cranial displacement of the flaccid diaphragm in the supine position, weaker intercostal muscles, higher compliance of the thoracic cage, and more susceptibility to diaphragm fatigue [20, 22]. The treatment of DP in infants, particularly for preterm infants, is still a dilemma, and the optimal timing for plication remains controversial. Immediate plication has been advocated earlier [2], but a waiting period of 2 to 6 weeks for infants with DP has also been recommended [10, 15, 27]. Younger patients who have lower weights usually require more respiratory support. Gallagher and Jog reported two extremely low birth weight infants with DP after PDA ligation and advised early plication in such a unique group for the sake of complications such as BPD [9, 12].

Surgical plication for affected diaphragms has been proven as a simple and safe procedure with a low complication rate in treating DP, even in very preterm infants [30]. Evidence also indicates that it does not interfere with the recovery of normal diaphragmatic function [28]. In the current study, three cases were extubated 3, 4, and 12 days post-surgical plication. Combined with the two previously reported cases that were extubated 7 and 6 days postoperatively [9, 12], we could speculate that plication is as effective in treating VLBW infants as in older patients and that for selected infants with DP-related ventilation failure surgical plication may facilitate extubation. Notably, one of the patients (case 5) did not receive diaphragmatic plication until weaning from the

ventilator failed three times, which ultimately led to a longer duration of mechanical ventilation. This might be the reason why we could not statistically show the advantage of plication in either the duration of ventilation dependency or oxygen-associated major complication in the current study. We recommend that plication could be arranged once the diagnosis is confirmed and the patient's condition is suitable for surgery.

There are a few limitations of this study that should be discussed. First, this is a retrospective, single-center observational study of a small patient population during a long period. Although a case–control method was conducted, it is difficult to eliminate all the confounding factors in managing a multifactor disease like PDA or BPD. We recognized that the ventilation policy for extremely preterm infants changed during the study period, including the concept of lower target oxygen saturation, permissive hypercapnia, and early CPAP instead of endotracheal ventilation [8]. Our strategy was to enroll control cases with their birth date nearer to each index case in order to compare cases in a most likely background. Second, the risk factor for DP could not be clarified in the current study. Most preligation characteristics were matched in DP and control group by a case–control method; thus, no significant difference could be identified. Since PDA ligations were performed by either of the two experienced surgeons, who used a similar technique and shared an equal incidence of developing DP, the operative factor was unobvious. However, we have to point out that we could not eliminate the influence of preligation respiratory condition on postligation ventilator dependency due to missing data in our database and its retrospective design. Third, the small sample size limited the statistical result. We failed to present the impact of DP on developing ROP, a complication usually correlating to prolonged oxygen exposure, and the benefit of plication on ventilator dependency. The need for additional data from other centers about DP following PDA ligation and the need for a multicenter prospective study of diaphragmatic plication are important to understand this poorly described complication.

Conclusion

Infants may be complicated after ductal ligation by diaphragmatic paralysis, which may explain ongoing ventilator dependency and eventually worsening of bronchopulmonary dysplasia. Early surgical plication in this select group of infants may be feasible to facilitate extubation but needs further investigations for its impact to be systemically evaluated.

Conflict of interest The authors have indicated they have no personal financial relationships relevant to this article to disclose.

References

1. An International Committee (1984) An international classification of retinopathy of prematurity. Br J Ophthalmol 68:690-697
2. Affatato A, Villagra F, De Leon JP, Gomez R, Checa SL, Vellibre D, Sanchez P, Diez Balda JI, Brito JM (1988) Phrenic nerve paralysis following pediatric cardiac surgery. Role of diaphragmatic plication. J Cardiovasc Surg (Torino) 29:606–609
3. Benjacholmas V, Namchaisiri J, Lertsarpcharoen P, Punnahitananda S, Thaithumyanon P (2009) Short-term outcome of PDA ligation in the preterm infants at King Chulalongkorn Memorial Hospital, Thailand. J Med Assoc Thai 92:909–913
4. Benjamin JR, Smith PB, Cotten CM, Jaggers J, Goldstein RF, Malcolm WF (2010) Long-term morbidities associated with vocal cord paralysis after surgical closure of a patent ductus arteriosus in extremely low birth weight infants. J Perinatol 30:408–413
5. Chiang MC, Lin WS, Lien R, Chou YH (2004) Reexpansion pulmonary edema following patent ductus arteriosus ligation in a preterm infant. J Perinat Med 32:365–367
6. Dagan O, Nimri R, Katz Y, Birk E, Vidne B (2006) Bilateral diaphragm paralysis following cardiac surgery in children: 10-years' experience. Intensive Care Med 32:1222–1226
7. de Leeuw M, Williams JM, Freedom RM, Williams WG, Shemie SD, McCrindle BW (1999) Impact of diaphragmatic paralysis after cardiothoracic surgery in children. J Thorac Cardiovasc Surg 118:510–517
8. Eichenwald EC, Stark AR (2008) Management and outcomes of very low birth weight. N Engl J Med 358:1700–1711
9. Gallagher PG, Seashore JH, Touloukian RJ (2000) Diaphragmatic plication in the extremely low birth weight infant. J Pediatr Surg 35:615–616
10. Hamilton JR, Tocewicz K, Elliott MJ, de Leval M, Stark J (1990) Paralysed diaphragm after cardiac surgery in children: value of plication. Eur J Cardiothorac Surg 4:487–490
11. Jobe AH, Bancalari E (2001) Bronchopulmonary dysplasia. Am J Respir Crit Care Med 163:1723–1729
12. Jog SM, Patole SK (2002) Diaphragmatic paralysis in extremely low birthweight neonates: is waiting for spontaneous recovery justified? J Paediatr Child Health 38:101–103
13. Joho-Arreola AL, Bauersfeld U, Stauffer UG, Baenziger O, Bernet V (2005) Incidence and treatment of diaphragmatic paralysis after cardiac surgery in children. Eur J Cardiothorac Surg 27:53–57
14. Koehne PS, Bein G, Alexi-Meskhishvili V, Weng Y, Buhrer C, Obladen M (2001) Patent ductus arteriosus in very low birthweight infants: complications of pharmacological and surgical treatment. J Perinat Med 29:327–334
15. Kunovsky P, Gibson GA, Pollock JC, Stejskal L, Houston A, Jamieson MP (1993) Management of postoperative paralysis of diaphragm in infants and children. Eur J Cardiothorac Surg 7:342–346
16. Lemmer J, Stiller B, Heise G, Alexi-Meskishvili V, Hubler M, Weng Y, Berger F (2007) Mid-term follow-up in patients with diaphragmatic plication after surgery for congenital heart disease. Intensive Care Med 33:1985–1992
17. Mandhan P, Brown S, Kukkady A, Samarakkody U (2009) Surgical closure of patent ductus arteriosus in preterm low birth weight infants. Congenit Heart Dis 4:34–37
18. Mavroudis C, Cook LN, Fleischaker JW, Nagaraj HS, Shott RJ, Howe WR, Gray LA Jr (1983) Management of patent ductus arteriosus in the premature infant: indomethacin versus ligation. Ann Thorac Surg 36:561–566
19. Mearns AJ (1977) Iatrogenic injury to the phrenic nerve nerve in infants and young children. Br J Surg 64:558–560
20. Mickell JJ, Oh KS, Siewers RD, Galvis AG, Fricker FJ, Mathews RA (1978) Clinical implications of postoperative unilateral phrenic nerve paralysis. J Thorac Cardiovasc Surg 76:297–304

21. Noori S (2010) Patent ductus arteriosus in the preterm infant: to treat or not to treat? J Perinatol 30(Suppl):S31–S37
22. Oktem S, Cakir E, Uyan ZS, Karadag B, Hamutcu RE, Kiyan G, Akalin F, Karakoc F, Dagli E (2010) Diaphragmatic paralysis after pediatric heart surgery: usefulness of non-invasive ventilation. Int J Pediatr Otorhinolaryngol 74:430–431
23. Papile LA, Burstein J, Burstein R, Koffler H (1978) Incidence and evolution of subependymal and intraventricular hemorrhage: a study of infants with birth weights less than 1,500 gm. J Pediatr 92:529–534
24. Shoemaker R, Palmer G, Brown JW, King H (1981) Aggressive treatment of acquired phrenic nerve paralysis in infants and small children. Ann Thorac Surg 32:250–259
25. Stramrood CA, Blok CA, van der Zee DC, Gerards LJ (2009) Neonatal phrenic nerve injury due to traumatic delivery. J Perinat Med 37:293–296
26. Teixeira LS, McNamara PJ (2006) Enhanced intensive care for the neonatal ductus arteriosus. Acta Paediatr 95:394–403
27. Tonz M, von Segesser LK, Mihaljevic T, Arbenz U, Stauffer UG, Turina MI (1996) Clinical implications of phrenic nerve injury after pediatric cardiac surgery. J Pediatr Surg 31:1265–1267
28. van Onna IE, Metz R, Jekel L, Woolley SR, van de Wal HJ (1998) Post cardiac surgery phrenic nerve palsy: value of plication and potential for recovery. Eur J Cardiothorac Surg 14:179–184
29. Walsh MC, Yao Q, Gettner P, Hale E, Collins M, Hensman A, Everette R, Peters N, Miller N, Muran G, Auten K, Newman N, Rowan G, Grisby C, Arnell K, Miller L, Ball B, McDavid G (2004) Impact of a physiologic definition on bronchopulmonary dysplasia rates. Pediatrics 114:1305–1311
30. Williams O, Greenough A, Mustfa N, Haugen S, Rafferty GR (2003) Extubation failure due to phrenic nerve injury. Arch Dis Child Fetal Neonatal Ed 88:F72–F73
31. Yon TF, Amka P, Pildes RS, Tatooles CJ (1977) Diaphragmatic paralysis after surgical ligation of patent ductus arteriosus. Lancet 2:461