ISSN 2221-2582

UDC 616.24-008.444-002.153-073.756.8+616.233–053.2+616.92/.93+66.074.43
DOI: 10.56871/CmN-W.2023.97.17.001

CLINICAL CASE OF CARDIORESPIRATORY MONITORING TO CONTROL SUPPLEMENTAL OXYGEN THERAPY IN A PREMATURE BABY

© Anna Yu. Trapeznikova, Dmitry O. Ivanov

Saint Petersburg State Pediatric Medical University. Lithuania 2, Saint Petersburg, Russian Federation, 194100

Contact information:
Anna Yu. Trapeznikova— MD, PhD; Department of Propedeutics of Childhood Diseases with a course of general childcare.
E-mail: anka.solomaha@yandex.ru ORCID: 0000-0003-4461-4322

For citation: Trapeznikova AYu, Ivanov DO. Clinical case of cardiorespiratory monitoring to control supplemental oxygen therapy in a premature baby. Children's medicine of the North-West (St. Petersburg). 2023;11(2):120–124. DOI: https://doi.org/10.56871/CmN-W.2023.97.17.001

Received: 06.03.2023 **Revised: 11.04.2023** **Accepted: 28.04.2023**

Summary. One of the most common respiratory pathologies in infants born prematurely is bronchopulmonary dysplasia (BPD). Pulmonary hypertension is considered a formidable and difficult to diagnose complication of BPD. Maintaining the proper level of oxygen saturation is an integral part of nursing such patients. Cardiorespiratory monitoring (CRM) is performed to record respiratory pauses and episodes of desaturation during sleep. The article discusses a clinical case of cardiorespiratory monitoring to control additional oxygen therapy in a premature infant suffering from bronchopulmonary dysplasia and BPD-associated pulmonary hypertension.

КЛИНИЧЕСКИЙ СЛУЧАЙ ПРИМЕНЕНИЯ КАРДИОРЕСПИРАТОРНОГО МОНИТОРИРОВАНИЯ ДЛЯ КОНТРОЛЯ ДОПОЛНИТЕЛЬНОЙ КИСЛОРОДОТЕРАПИИ У НЕДОНОШЕННОГО РЕБЕНКА

© Анна Юрьевна Трапезникова, Дмитрий Олегович Иванов

Санкт-Петербургский государственный педиатрический медицинский университет. 194100, г. Санкт-Петербург, ул. Литовская, 2

Контактная информация:
Анна Юрьевна Трапезникова — к.м.н., ассистент; кафедра пропедевтики детских болезней с курсом общего ухода за детьми.
E-mail: anka.solomaha@yandex.ru ORCID: 0000-0003-4461-4322

Для цитирования: Трапезникова А.Ю., Иванов Д.О. Клинический случай применения кардиореспираторного мониторирования для контроля дополнительной кислородотерапии у недоношенного ребенка // Children's medicine of the North-West. 2023. Т. 11. № 2. С. 120–124. DOI: https://doi.org/10.56871/CmN-W.2023.97.17.001

Поступила: 06.03.2023 **Одобрена: 11.04.2023** **Принята к печати: 28.04.2023**

Резюме. Одной из наиболее часто встречающихся патологий респираторного тракта у детей, родившихся недоношенными, является бронхолегочная дисплазия (БЛД). Грозным и трудно диагностируемым осложнением БЛД считается легочная гипертензия. Поддержание должного уровня сатурации кислорода является неотъемлемой частью выхаживания таких пациентов. Для регистрации дыхательных пауз и эпизодов десатурации во время сна проводят кардиореспираторное мониторирование (КРМ). В статье рассматривается клинический случай применения кардиореспираторного мониторирования для контроля дополнительной кислородотерапии у недоношенного ребенка, страдающего бронхолегочной дисплазией и БЛД-ассоциированной легочной гипертензией.

Ключевые слова: недоношенный ребенок; апноэ; кардиореспираторное мониторирование

Key words: *premature infant; apnea; cardiorespiratory monitoring*

INTRODUCTION

Currently, preterm birth is a serious problem that contributes to a significant increase in morbidity and mortality all around the world. Complications that arise from preterm birth are the main causes of neonatal mortality [1].

Bronchopulmonary dysplasia (BPD) is the most common chronic respiratory pathology in pre-

mature infants, and in 45% of cases it occurs in children with small gestational age and extremely low birth weight who have suffered respiratory distress syndrome and perinatal infections [2]. Exposure to invasive measures that damage lung tissue for a long time leads to long-term persistence of changes in lung structures, including alternating areas of hyper-expansion and atelectasis, fibrosis of the alveolar septum of varying severity, fibrosis of the bronchial submucosa, muscle hyperplasia and the development of pulmonary hypertension [3].

However, lung parenchyma damage is not the only cause of the formation of bronchopulmonary dysplasia. At present, there is a growing understanding of the contribution of growth impairment and remodeling of pulmonary vessels to the development of this pathology. Vascular changes can lead to elevated pulmonary vascular resistance and pulmonary pressure, which in turn promotes the development of compensatory right ventricular hypertrophy and the appearance of clinical signs of pulmonary hypertension (PH).

Immaturity of respiratory control, artificial lung ventilation (ALV) using an endotracheal tube, leading to the development of inflammation and, as a result, narrowing of the upper airways, predisposition to obstruction and small airway luminal diameter, characteristic of preterm infants, as well as lung volume reduction and hypoventilation can lead to low ventilation-perfusion and intrapulmonary adaptability, causing a rapid decrease in oxygen saturation, and contribute to the occurrence of episodes of intermittent hypoxemia and frequent fluctuations in oxygenation [4, 5]. Most are corrected by increasing the fraction of inspired oxygen (FiO2), resulting in some degree of hyperoxaemia. This makes it difficult to maintain SpO_2 within the target range (≥92% for preterm infants with BPD and 94–95% or more [6] for premature infants with BPD-associated pulmonary hypertension), and newborns may spend significant periods of time with SpO_2 below or above target values, which requires constant monitoring for the purpose of timely correction. To improve growth, reduce the severity of symptoms of pulmonary hypertension and the risk of sudden death associated with hypoxemia, constant maintenance of an adequate level of oxygen saturation is required in preterm infants with BPD and BPD-associated pulmonary hypertension [4, 7].

Polysomnography (PSG) and cardiorespiratory monitoring (CRM) are used as objective methods for recording respiratory pauses and short-term desaturation episodes. The use of PSG in newborns and children during the first three years of life is difficult. The use of CRM is acceptable for this category of patients. Infants with BPD may have normal oxygen saturation and respiratory rate during outpatient clinic visits while awake, but they are prone to episodes of desaturation during sleep, along with disordered breathing during the night [8, 9].

CLINICAL CASE

Baby E. was born from the first pregnancy, first delivery. The mother is 36 years old (elderly primigravida), with a burdened obstetric and gynecological anamnesis (chronic endocervicitis). Somatic diseases in the mother: hypotension, polyposis of the gastrointestinal tract, varicose veins of the lower extremities, external hemorrhoids in remission, chronic gastroduodenitis in remission. Occupational hazards, smoking denies.

The delivery was first, premature, at 25 1/7 weeks. Features of the course of labor were breech presentation of the fetus, premature rupture of membranes (anhydrous interval was 9 days), anhydramnios, and chorioamnionitis. At 14 weeks of gestation, the mother's blood was found to be Rh-negative without the presence of antibodies.

The birth weight of the baby was 700 g, body length — 27 cm, head circumference — 22 cm, chest circumference — 21 cm. Apgar scores 1 and 5 minutes after birth were 6 and 7 points, respectively. The condition at birth was assessed as severe due to the development of respiratory failure and extreme morpho-functional immaturity. From birth, the girl was on non-invasive ventilation (NIV), later transferred to nasal CPAP (NCPAP) mode, and was oxygenated satisfactorily. On the 23rd day of life (28 2/7 weeks of postconceptional age, PCA), a worsening condition was noted in the form of increased respiratory failure, deterioration of the clinical picture of the gastrointestinal tract (elements of altered blood were noted on the orogastric tube), manifestations of convulsive syndrome, caused by the implementation of a generalized infectious process with damage to lungs and gastrointestinal tract against the background of deep morpho-functional immaturity. She was transferred to mechanical ventilation with moderate parameters, after 10 days (33rd day of life) she was extubated and transferred to NIV, after 16 days (49th day of life) she was transferred to CPAP. From 34 4/7 weeks of PCA (66th day of life), the infant was transferred to supple-

 ISSN 2221-2582

mental oxygen therapy through a loose-fitting face mask. Oxygenation was satisfactory; no signs of increased respiratory failure were diagnosed. On the 89th day of life (37 4/7 weeks of PCA), she was transferred to an oxygen tent (FiO_2 parameters were 0.3). She remained oxygen dependent throughout the entire observation period in the hospital.

At the age of 3 months, due to the appearance of repeated regurgitation, an X-ray contrast examination was performed, gastroesophageal reflux disease and hiatal hernia were detected. Consulted with a surgeon; surgical treatment is not indicated. At 3 months 1 week, the administration of an anti-regurgitation formula was started.

According to echocardiography performed at 38 5/7 weeks of PCA, the mean pulmonary artery pressure is equal to the systemic arterial pressure, there are a moderate right ventricular dilatation without significant myocardial hypertrophy and an atypical patent ductus arteriosus. In agreement with the pediatric cardiologist, therapy with sildenafil was started at a dose of 1.0 mg/kg 4 times a day.

According to the results of echocardiography (43 5/7 weeks of PCA), no signs of congenital heart disease were found, patent ductus arteriosus was not registered, moderate right heart dilation without significant myocardial hypertrophy maintained, the mean pulmonary artery pressure was 40–41 mmHg.

On Neurosonography (43 5/7 weeks of PCA) were diagnosed echo signs of post-hypoxic changes against the background of pronounced morpho-functional immaturity with asymmetric ventriculodilation, mainly due to the occipital horn of the large ventricle of the brain. There are periventricular leukomalacia (cystic form) and grade I intraventricular hemorrhage on the left side in anamnesis.

Electroencephalography data (43 6/7 weeks of PCA) indicated the presence of moderate diffuse changes in the functional state of neurons in the cortex and subcortical structures of the brain; There wasn't epileptiform activity or interhemispheric asymmetry.

Weight gain during observation in the hospital was: for the 1st and 2nd months — 785 g, for the 3rd month — 392 g, for the 4th month — 861 g.

Main diagnosis: Perinatal hypoxic-ischemic cerebral injury, periventricular leukomalacia (cystic form), early recovery period, pyramid deficiency syndrome in the lower extremities. Convulsive syndrome in anamnesis. Delayed psychomotor development.

Associated diagnosis: Bronchopulmonary dysplasia originating in the perinatal period, a "new" form, severe course, period of chronic disease, type I respiratory failure. Respiratory distress syndrome in anamnesis. Pulmonary hypertension. Prematurity 25 1/7 weeks. Extremely low birth weight. Proliferative retinopathy of prematurity, stage III, zone 2. Condition after undergoing laser coagulation of the left retina from 13.06.2017, of both eyes from 27.06.2017. Necrotizing enterocolitis in the fetus and newborn, stage 2a in anamnesis. Cytomegaloviral disease, unspecified. Generalized cytomegalovirus infection, viral carriage in anamnesis. Patent ductus arteriosus, medically closed. Anemia of prematurity, severe, of mixed origin. Acute hemorrhagic gastritis in anamnesis. Gastroesophageal reflux disease without esophagitis.

At 40 1/7 weeks of PCA, cardiorespiratory monitoring was performed during sleep while receiving supplemental oxygen therapy. Oxygen support was turned off for 92 minutes during monitoring to provide a true picture of the ability to maintain oxygen saturation within the target range. The recording duration was 258 minutes. The median oxygen saturation was 91,1% (the entire recording was analyzed), the minimum was 74,0% (recorded only in the absence of supplemental oxygen support). A total of 90 episodes of desaturation were recorded (oxygen desaturation index was 21,1 events/hour), of which 46 episodes (10,8 events/hour) were recorded with a drop in oxygen saturation <5%, 30 episodes were recorded with a drop in SpO_2 by 5–9% (7,0 events/hour), with a drop in SpO_2 by 10–20% — 13 episodes (3,0 events/hour) and with a drop in SpO_2 >20% — 1 episode (0,2 events/hour). All episodes of desaturation were observed in the absence of supplemental oxygen therapy. The number of episodes of desaturation <90% was 64 (15,0 events/hour), <85% — 37 episodes (8,7 events/hour), <80% — 12 episodes (2,8 events/hour). Severe bradycardia (83 beats per minute) was noted in the absence in the supplemental oxygen support. Based on the data obtained, it was decided to continue supplemental oxygen therapy at home.

At 48 3/7 weeks of PCA, repeated cardiorespiratory sleep monitoring was performed at home. Before the study, the infant had been without supplemental oxygen support for 7 days. The recording duration was 296 minutes. The median oxygen saturation during the infant's sleep (93,1 per cent) was lower than recommended standards. Positive dynamics were observed in the form of an

increase in median oxygen saturation compared to previous monitoring. The minimum value of median SpO_2 was 84,0%. Episodes of desaturation with independent recovery were diagnosed (oxygen desaturation index was 19,3 events/hour), with a drop in oxygen saturation <5% — 60 episodes (13,9 events/hour), 5–9% — 23 episodes (5,3 events/hour), 10–20% — 0 episodes. The number of episodes of decreased saturation <90% — 41 (9,5 events/hour), <85% — 1 episode (0,2 events/hour), <80% not registered. At the moments of desaturation, bradycardia was noted (96 beats per minute). Dynamic monitoring was recommended.

CONCLUSION

The formation of BPD in this infant was facilitated by factors such as mother's unfavorable obstetric and somatic anamnesis, premature birth, the need for intensive oxygen therapy due to type I respiratory failure, oxygen dependence up to five months of life. Characteristic clinical and radiological changes indicated the development of BPD. The need for supplemental oxygen therapy at 36 weeks of PCA suggests severe BPD. Hypoxic–ischemic brain injury, grade I intraventricular hemorrhage, and cystic periventricular leukomalacia were diagnosed. Cardiorespiratory sleep monitoring allowed us to establish positive dynamics in the form of an increase in the level of median oxygen saturation in the absence of oxygen support. However, attention is drawn to pronounced desaturation, accompanied by bradycardia, which requires dynamic monitoring.

REFERENCES

1. Blencowe H., Cousens S., Chou D. et al. Born too soon: The global epidemiology of 15 million preterm births. Reprod. Health. 2013; 10: 2. DOI: 10.1186/1742-4755-10-S1-S2.
2. Tracy M.K., Berkelhamer S.K. Bronchopulmonary dysplasia and pulmonary outcomes of prematurity. Pediatr. Ann. 2019; 48(4): 148–53. DOI: 10.3928/19382359-20190325-03.
3. Altit G., Dancea A., Renaud C. et al. Pathophysiology, screening and diagnosis of pulmonary hypertension in infants with bronchopulmonary dysplasia — a review of the literature. Paediatr. Respir. Rev. 2016; 23: 16–26. DOI: 10.1016/j.prrv.2016.11.002.
4. Chandrasekharana P., Lakshminrusimha S. Oxygen therapy in preterm infants with pulmonary hypertension. Semin. Fetal Neonatal Med. 2020; 25(2): 1–7. DOI: 10.1055/s-0036-1592346.
5. Solomakha A.Yu., Ivanov D.O. Osobennosti etiologii i diagnostiki BLD-assotsiirovannoy legochnoy gipertenzii u nedonoshennykh detey [Features of the etiology and diagnosis of BPD-associated pulmonary hypertension in premature infants]. Children's Medicine of the North-West. 2021; 9(2): 22–7. (in Russian).
6. Cummings J.J., Polin R.A. Committee on Fetus and Newborn. Oxygen Targeting in Extremely Low Birth Weight Infants. Pediatrics. 2016; 138(2): e20161576. DOi: 10.1542/peds.2016-1576.
7. Solomakha A.Yu., Petrova N.A., Obraztsova G.I. i dr. Dinamika kardiorespiratornykh i ekhokardiograficheskikh pokazateley u detey pervogo goda zhizni, stradayushchikh bronkholegochnoy displaziey [Dynamics of cardiorespiratory and echocardiographic indicators in children of the first year of life suffering from bronchopulmonary dysplasia]. Neonatologiya: novosti, mneniya, obuchenie. 2019; 7, 1(23): 31–7 (in Russian).
8. Simonneau G., Gatzoulis M.A., Adatia I. et al. Updated clinical classification of pulmonary hypertension. Turk. Kardiyol. Dern. Ars. 2014; 42(Suppl. 1): 45–54.
9. Solomakha A.Yu., Petrova N.A., Ivanov D.O., Sviryaev Yu.V. Narusheniya dykhaniya vo sne u nedonoshennykh detey s bronkholegochnoy displaziey i legochnoy gipertenziey [Sleep breathing disorders in premature infants with bronchopulmonary dysplasia and pulmonary hypertension]. Pediatr. 2017; 8(6): 5–10. (in Russian).

ЛИТЕРАТУРА

1. Blencowe H., Cousens S., Chou D. et al. Born too soon: The global epidemiology of 15 million preterm births. Reprod. Health. 2013; 10: 2. DOI: 10.1186/1742-4755-10-S1-S2.
2. Tracy M.K., Berkelhamer S.K. Bronchopulmonary dysplasia and pulmonary outcomes of prematurity. Pediatr. Ann. 2019; 48(4): 148–53. DOI: 10.3928/19382359-20190325-03.
3. Altit G., Dancea A., Renaud C. et al. Pathophysiology, screening and diagnosis of pulmonary hypertension in infants with bronchopulmonary dysplasia — a review of the literature. Paediatr. Respir. Rev. 2016; 23: 16–26. DOI: 10.1016/j.prrv.2016.11.002.
4. Chandrasekharana P., Lakshminrusimha S. Oxygen therapy in preterm infants with pulmonary hypertension. Semin. Fetal Neonatal Med. 2020; 25(2): 1–7. DOI: 10.1055/s-0036-1592346.
5. Соломаха А.Ю., Иванов Д.О. Особенности этиологии и диагностики БЛД-ассоциированной легочной гипертензии у недоношенных детей. Children's Medicine of the North-West. 2021; 9(2): 22–7.

6. Cummings J.J., Polin R.A. Committee on Fetus and Newborn. Oxygen Targeting in Extremely Low Birth Weight Infants. Pediatrics. 2016; 138(2): e20161576. DOi: 10.1542/peds.2016-1576.
7. Соломаха А.Ю., Петрова Н.А., Образцова Г.И. и др. Динамика кардиореспираторных и эхокардиографических показателей у детей первого года жизни, страдающих бронхолегочной дисплазией. Неонатология: новости, мнения, обучение. 2019; 7, 1(23): 31–7.
8. Simonneau G., Gatzoulis M.A., Adatia I. et al. Updated clinical classification of pulmonary hypertension. Turk. Kardiyol. Dern. Ars. 2014; 42(Suppl. 1): 45–54.
9. Соломаха А.Ю., Петрова Н.А., Иванов Д.О., Свиряев Ю.В. Нарушения дыхания во сне у недоношенных детей с бронхолегочной дисплазией и легочной гипертензией. Педиатр. 2017; 8(6): 5–10.