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Prolonged Ventilation In Infancy

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Abstract

Not enough attention has been paid to the problem of prolonged ventilation in infants. To improve respiratory support for these infants, we have developed a protocol for prolonged ventilation using Volume Targeted Ventilation. To study the safety and effectiveness of this protocol, we conducted a study on a group of 79 infants in the PICU. At the beginning of treatment, we achieved the following median ventilation settings: Vt - 11 mL/kg, PIP - 28 cmH2O, PEEP - 7.9 cmH2O, FiO2 - 52%, and RR (SIMV cycles/min) - 23. These levels of ventilation were well tolerated by infants. Our primary clinical outcomes included mortality rate, complications, reintubation rate, asynchrony index, and time of ventilator setting de-escalation. Pneumothorax occurred in only two infants (2.6%). This indicates the safety of our protocol. During the weaning process, reintubation was observed in 16 cases (20.5%). The mortality rate in the study group was 7.6% (6 cases). Based on these findings, the applied protocol for mechanical ventilation can be considered effective for infants requiring prolonged ventilation.

Keywords: prolonged ventilation; infants; protocol; weaning; patient-ventilator asynchronies; bronchopulmonary dysplasia

1.Introduction

According to the literature, not enough attention is paid to the problems of prolonged ventilation (PV) in infancy. Most of the published scientific papers, clinical practice guidelines, and guidelines on mechanical ventilation (MV) in neonates are almost exclusively targeted at the early stages of pulmonary disease in the neonatal period. Much less researches are devoted to the problems of PV in infants who have undergone any serious illness in the early neonatal period.

As perinatal care has improved, the number of such children has steadily increased. Because of the number of high-risk births, including premature and extremely premature, is increasing every year. The experience of providing assistance to such children at the perinatal centers (PCs) is improving, the survival rate of these children is increasing, therefore, more and more children in need of PV are being transferred to the regional children hospitals for the second stage of care. Full–term infants in need of PV are transferred to second stage of hospitals after 30 days, and premature infants (including those with extremely low birth weight (ELBW) are transferred 100 days after birth.

In this group of children, in addition to respiratory distress syndrome (RDS) and bronchopulmonary displasia (BPD), which are usually the main diagnosis, there are many comorbidity, such as newborn asphyxia, sepsis, congenital pneumonia, congenital malformations of the heart, lungs, gastrointestinal tract (GIT), central nervous system (CNS), perinatal brain damage (PBD), necrotizing enterocolitis (NEC), congenital defects in surfactant proteins, congenital metabolic defects, etc. These infants are at the high risk of multiple and serious long-term complications [1]. This group of patients is decisive in the development of disability and infant mortality, and this population needed huge resources in intensive care units [2]. More over there is no consensus definition on PV in infancy that is a major limitation to investigations in the field [2].

Therefore, the process of weaning from MV in this group of patients looks non-standard. An attempt at early extubation are usually unsuccessful, the ventilation process is delayed. Such patients are delayed on MV for a long time, in this group there is a high incidence of disability and death.

Obviously, withing group of these patients, we need to rethink the main goals of therapy, including emphasis on the earliest possible extubation in favor of the earliest detection of diseases which could be reasons of PV, adequate long-term support for patients, which is extremely important for the development of lung tissue and, ultimately, full-fledged rehabilitation.

In this way, we believe that Volume Targeted Ventilation (VTV) is ideally suited for PV in infants, as it allows for faster weaning from MV and is quite safe, while also helping to avoid many complications.

2. Definition and etiology

By the analogy with definition of the prolonged weaning from MV [3, 4], we decided that a PV in infants should be considered as MV for more than 7 days, regardless of unsuccessful weaning/extubation attempts during these 7 days (even if a child was on a non-invasive respiratory support (nasal continue positive airway pressure (NCPAP), higher frequency nasal cannula (HFNC), etc.) or on the spontaneous respiration (SR) with or without additional oxygen supply) for some period withing of these days.

The etiology of this condition includes predominantly BPD. This diagnosis is often seen in infants with PV, and it typically does not respond to standard treatment. These are usually premature babies or newborns with severe perinatal asphyxia who need therapeutic hypothermia. They have experienced intrauterine growth retardation and are often infected with the TORCH (Toxoplasma, Rubella, Cytomegalovirus, Herpes virus and others) complex or have severe birth injuries.

The next possible causes of PV could be congenital lung abnormalities, such as slings, lung sequestrations, tumors, congenital emphysema, tracheobronchomegaly, or other congenital defects. The most common congenital abnormality of the GIT among infants with PV is diaphragmatic hernia. Other extralung congenital abnormalities include congenital heart defects (CHD) that require considerable surgical intervention. In some cases, this may involve multiple stages and a long period of postoperative recovery, including for PV.

In addition, PV can be associated with congenital defects of the CNS, such as vein of Galen malformations, encephaloceles, myelomeningoceles, holoprosencephalies, and agenesis of the corpus callosum. Other conditions that can occur with PV include Dandy-Walker syndrome, Miller-Dieker syndrome, Zellweger syndrome, and others.

Some congenital metabolic disorders can also be linked to PV. These include mitochondrial defects, problems with ketolysis and ketogenesis, issues with the oxidation of fatty acids, renal tubular acidosis, organic acidaemias, disorders of pyruvate dehydrogenase complexes, pyruvate carboxylases, and gluconeogenesis. As well there are diseases not detected by tandem mass spectrometry (TMS). We observed three patients with congenital metabolic disorders that were not detected by TMS: one case of congenital glycosylation disorder and two cases of non-ketotic hyperglycinemia.

The NEC is another potential risk to PV because it is

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a critical condition that can lead to multiple organ failures. This requires intensive care and the use of MV with very severe parameters. Neonatal sepsis and congenital pneumonia can also cause severe conditions such as septic shock or multipple organ failure (MOF). These conditions can also lead to PV.

Recently, we have encountered another serious condition related to PV: hemofagocytic lymphohistiocytosis (HLH). Familial HLH is an invariantly fatal rare disease with a frequency of 1.2 per million[5]. Secondary HLH can develop due to various conditions such as infections, cancer, or auto-inflammatory or autoimmune diseases. In fact, more than 90% of children with HLH have acquired forms of the disease [6]. Non-antigenic triggers of HLH may include severe trauma, use of interferons and other medications for treatment, and reactive oxygen species [6]. Mechanisms of acquired HLH may include PV [6]. Consequently, BPD and other conditions associated with PV may pose a risk of HLH development, and according to the feedback system, they may also contribute to the development of PV.

Several complications can significantly burden the MV up to the PV. These include first of all, perivenricular and intraventricular hemorrhages (PIVH and IVH), and periventricular leukomalacia (PVL), which are considered independent predictors of disability and infant mortality. Other complications that occur with PV include air-leak syndrome, tracheobronchomalacia, and tracheal stenosis.

3.The development of prolonged ventilation

The first option for the development of PV may include newborns who initially don't have severe respiratory disorders. They might receive surfactant treatment and non-invasive respiratory support (NCPAP, HFNC) or support MV with mild settings with low oxygen demand. Subsequently, the respiratory status gradually worsens, which is manifested by diffuse pulmonary opacities on X-rays, and the early formation of "air traps" in the form of air bubbles, which inevitably leads to intubation and more stringent ventilation parameters. This group may be linked with either an acceleration of lung maturation due to the effects of an intrauterine inflammatory process or to the influence of various comorbid conditions, including drug sedation, on the deterioration of respiratory drive and subsequent ventilator-associated lung injury [7].

The second group of patients experienced mild to moderate RDS with initial improvement leading to successful extubation in some cases. However, the respiratory status of these patients later deteriorated, necessitating reintubation and increased respiratory support due to failure to wean from the ventilator. This ultimately resulted in the development of PV. This group was most common and may be associated with the effects of relative hyperoxia and ventilator-induced lung injury during early lung maturation [7].

The third group of patients typically experience severe RDS that is unresponsive to surfactant treatment, requiring strict ventilator settings. These conditions rarely improve significantly and may progress to BPD and eventually pulmonary fibrosis. These children are also at risk for congenital pneumonia or pulmonary hypoplasia, and their condition can be complicated by pulmonary hypertension [7].

It is important to note that the full clinical picture of BPD may not become apparent until the second month after birth. However, even in the first week of life, there is increasing resistance to airflow, followed by inhomogeneous aeration of the lungs, increased bronchial secretions which can lead to so-called "wandering" atelectasis and a trend towards lung hyperinflation during the second and third weeks of life [7]. Despite using permissive hypercapnia, infants with ELBW, who require PV, may require an increase in tidal volume (Vt) by the third week [7].

Newborns with PV may have lesions in their lung parenchyma and airways. In most cases, there is obstructive damage to the airways, but the clinical presentation can vary widely and the balance between pulmonary and bronchial injury may differ among patients and even within the same individual's lungs. Both larger and smaller airways may be affected, resulting in different clinical manifestations depending on the nature of the lesion [7].

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When the large airways are affected, mucosal and submucosal injuries may occur as a result of prolonged intubation [7]. This can lead to subglottic tracheal stenosis and local obstruction due to airway granuloma or vocal cord dysfunction. One common cause of large airway obstruction is tracheobronchomalacia, which is likely caused by prolonged cyclic stretching of immature airway structures [7]. Tracheobronchomalacia may cause dynamic airway obstruction during expiration, which may be manifested as expiratory stridor and abnormalities in the flow-volume curve on a ventilator monitor. In children, this condition can also lead to inspiratory stridor, due to the collapse of the extrathoracic trachea during SR [7].

Small airway obstruction is typically caused by mucous edema, smooth muscle hypertrophy, excessive secretion, and bronchoconstriction. The severity of these conditions can vary, and in many cases, bronchodilators may not be effective [7].

The lung parenchyma in PV gradually becomes heterogeneous. The heterogeneity of the pulmonary parenchyma, along with the involvement of the respiratory tract in PV, results in significant variations in the time constants of ventilation in different regions of the lungs [7]. This leads to a complex pathophysiological process, making it challenging to optimize ventilation for patients.

Certain areas of the lungs exhibit low airway resistance, known as "fast compartments," and are rapidly filled and emptied of air. Conversely, areas with higher airway resistance, or "slow-filling" or "slow-emptying compartments," demonstrate a slower inflation or deflation of terminal respiratory units, respectively [7].

4. The hallmarks of MV in infants on PV

In cases of RDS, ventilation with a high frequency of respiratory cycles and short inspiratory and expiratory times primarily directs gas flow towards areas of the lungs with fast compartments. This results in an increase in dead space and a decrease in the ventilation-perfusion ratio [7].

This condition can potentially damage relatively healthy lung tissue through overgrowth and limit the amount of gas reaching areas with slow compartments and long expiration times. In multi-compartment lungs, air clearance is almost entirely dependent on emptying slow compartments, which are significantly impaired if the expiration constant is not sufficient [7]. As a result, air trapping occurs in these regions, leading to a significant decrease in respiratory rate (RR), an increase in inspiratory and expiatory time, and a need for optimization of ventilation in infants requiring PV due to perinatal factors [7].

The second important difference between MV in RDS and PV is the need for a significantly higher Vt [7]. This is due to the increased alveolar dead space caused by the inhomogeneous inflation of the lungs and the formation of "air traps". Additionally, there is an increased anatomical dead space due to acquired tracheobronchomegaly. In order to maintain minute ventilation at 250-300 mL/kg/min, with a slow respiratory rate of 20 cycles per minute, the Vt should be between 12.5 and 15 mL/kg [7]. Although these volumes are critical and potentially dangerous, they can lead to lung volume trauma in infants [8].

The actual minute ventilation needed to maintain adequate CO2 removal is individual for each patient. It depends on several factors:

1. The total physiological dead space (the sum of the alveolar and anatomical dead spaces).

2.The number of alveolar lesions, which reduce the gas exchange surface area.

3.The level of permissive hypercapnia, which helps remove CO2 by increasing the diffusion gradient for that gas.

4.The patient's spontaneous inspiratory force, which can increase the total minute ventilation.

The third major difference between PV and RDS in newborns is the need for significantly higher levels of positive end-expiratory pressure (PEEP) [7].

In newborns with RDS, the small airways, which are able to accommodate a limited volume of air, can

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easily collapse during expiration, resulting in limited exhalation. This is likely due to the lack of elasticity in the immature connective tissue in these patients.

Even the larger airways can collapse due to tracheobronchomalacia, causing similar restrictions on gas flow and similar responses to high PEEP levels [7].

Using high levels of PEEP during "air trapping" development may seem counterintuitive, as there are guidelines against using high PEEP in obstructive conditions to prevent air trapping. However, this strategy is highly effective in practice. It keeps the airways open during expiration and ensures maximum gas exchange, especially in slow-flow regions of the lung [7].

Volume Targeted Ventilation (VTV) is the preferred method for MV in newborns requiring PV [7]. One of the bests modes of ventilation withing VTV is Synchronized Intermittent Mandatory Ventilation (SIMV). Since spontaneous infant respiration on PS is aimed at ventilation of “fast” compartments with relatively high RR and low Vt, while low RR of SIMV breaths with high Vt is mainly aimed on ventilation of “slow” compartments [7]. However, it is important to avoid generating too high RR on the PS. This can have the opposite effect and lead to the formation of air "traps." Considering all the above, we have developed a protocol for PV in infants using VTV.

5.The protocol for PV in infants

1. The first step in the protocol is to select the ventilation mode. The preferred mode is SIMV, which can be configured for volume-controlled ventilation with a descending flow pattern or with the Volume Guarantee (VG) option, depending on the brand of ventilator you use.

2.The second step is to set the targeted tidal volume (Vt) for SIMV breathing. The starting volume should be between 6 and 8 millilitres per kilogram for infants and 8 to 10 millilitres for older infants with severe BPD. In cases of severe BPD, the Vt may need to be increased, sometimes reaching up to 12 to 15 millilitres per kilogram [7]. The step for increasing the Vt is 1 millilitre per kilogram.

3. Setting the PIP limit:

- For incipient BPD, the PIP limit is 26 cm H2O [7].

- For severe BPD, the PIP limit is 30 cm H2O [7]. However, there may be individual cases where higher values are required. The choice of this indicator will depend on the “working” PIP. The PIP limit should be 3-5 cm H2O higher than the “working” PIP, and sometimes it may be +20% of the “working” PIP [7]. This means that it is also titrated.

4. Setting up pressure support (PS) for spontaneous breathing. Initially, the pressure support is set to 10-12 cmH2O [7]. Then, it is adjusted to achieve a spontaneous Vt of 4-6 ml/kg [7]. This is done until tachypnea disappears.

5. Setting the PEEP. Initially, set the PEEP at 8-10 cmH2O [7]. Make sure your patient's blood pressure is stable and there is no hypoperfusion before adjusting! The change step should be 0.5-1 cmH2O [7].

To understand if you need to increase PEEP, use the "flow–volume" curves. This tool helps you identify any restrictions in the flow of exhaled air at low tidal volumes. If you notice any limitations, it may be a sign that you need to raise the PEEP.

Another way to determine the optimal level of PEEP is to find the value that provides the best dynamic compliance and the lowest resistance in the respiratory tract. This is known as the "best PEEP".

6. The RR setting. The RR for SIMV breathing is set at 20-25 breaths per minute. The inspiratory time is between 0.5 and 0.7 seconds [7]. The expiratory flow should return to zero before the next inspiration. In some cases, this can even result in a decrease in the RR of up to 15 breaths per minute [7].

7. Weaning from the ventilator.

The decision to initiate the weaning process is usually made after a significant period of stability has been achieved, an adequate and sufficiently prolonged increase in the infant's body weight, and their ability

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to respond and interact with medical staff and parents.

If the oxygen fraction in the inspired air (FIO2) stays below 0.4 for a long time without significant desaturation episodes and there is no pulmonary hypertension, it is a sign to start reducing the level of respiratory support [7].

We gradually decrease the targeted Vt. We make changes one or two times a week, moving forward in small steps. After each change, we observe how the child reacts to the new situation. If there is an increase in FIO2 and the mechanical work of the respiratory muscles (respiratory work) increases, the changes should be reversed to their original levels, and the weaning process should be suspended. If the baby tolerates the changes well, without significant backlashes, the weaning process can be gradually continued.

The PEEP may decrease slowly, alternating with a decrease in Vt and PIP. We conclude that the child is ready for extubation after testing their ability to breathe independently. We reduce the frequency of synchronized mandatory breaths (in SIMV mode) to the lowest possible minimum and leave the child on the PS for a long time. Infants with BPD can be extubated at significantly higher PIP, Vt, and PEEP parameters than premature newborns with RDS [7].

Next, you might need to use the NCPAP mode. The PEEP level is set at the mean airway pressure (MAP) before extubation. Typically, this is around 7-10 cmH2O. However, according to research, even a PEEP of 10-15 cmH2O is quite effective and well tolerated by patients in this condition [7].

If the weaning process is fail, a differential diagnosis should be conducted. When BPD is diagnosed, dexamethasone therapy is prescribed. Dexamethasone is usually prescribed for newborns no earlier than the first week of life. Dexamethasone should be administered in the following cases:

1.Long-term dependence on MV (more than seven days);

2.Failed attempts of extubation;

3.FiO2 above 35-50%;

4.The need for high PIP during ventilation;

5.MAP above 7-10 cmH2O;

6. X-Ray evidence indicating a decrease in lung tissue transparency.

7.Contraindications for prescribing dexamethasone:

1. Current active fungal infection with appropriate markers of a systemic inflammatory response.

2. Any stage of enterocolitis.

3. Persistent hyperglycemia greater than 8.6 mmol/L (for children with a body weight of less than 2,000 g., above 7 mmol/L).

4. Obstructive hypertrophic cardiomyopathy, as determined by echocardiography.

5. Persistent arterial hypertension.

Dexamethasone is prescribed at an initial dose of 0.15 mg per kilogram per day for three days before the patient is extubated. If the patient is successfully extubated, the dose is reduced to 0.1 mg per kilogram for 4-6 days, then to 0.05 mg per kilogram for 7-8 days, and finally to 0.02 mg for 9-10 days before stopping the medication.

If weaning is fail, for example, if the patient is not ready for extubation or needs to be reintubation, the dose of dexamethasone is increased to 0.3 mg per kilogram for three days. During this time, it is important to conduct a differential diagnosis in order to exclude adrenal insufficiency, birth defects of surfactant proteins, HLH, infectious diseases, congenital metabolic disorders, PVL and other conditions that may cause prolonged ventilation.

6.Own observations

To evaluate the effectiveness of the protocol, we examined 79 patients who were transferred to the pediatric intensive care unit (PICU) from the nearest PCs with a diagnosis of BPD. The inclusion criteria were considered to be the following: gestational age 23 – 40 weeks, birth weight 450 – 2000 g., PV after birth (for more than one month).

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The criteria for exclusion are as follows: CHD requiring surgery with cardioplegia and hypothermia, congenital malformations, and PBD with significant neurological deficits that prevent the patient from developing sufficient respiratory drive, even after proper neurosurgical treatment.

Demographic data and clinical characteristics and outcomes were summarized using descriptive statistics presented as number and percentage, median with interquartile range (25th; 75th percentile) as appropriate. The statistical tests used to test normality of data distribution were Shapiro–Wilk and Kolmogorov–Smirnov. For comparing two dependent samples (groups) were used Wilcoxon mathched pairs test, to compare binary variables, we used Fisher's exact test, p-value < 0.05 was considered statistically significant. Statistical analyses were performed using a Statistica 13 Package. Relative risk (rr) and Number Needed to Treat (NTT) analysis calculated with application MedCalc.

6.1 Study population

The characteristics of the studied group of infants are shown in Table 1. As the table indicates, the majority of the children were born prematurely. The median gestational age was 26 weeks, while the minimum gestational age was as low as 23 weeks.

Table 1: The study population

Parameters	Me (LQ-UQ)	minimum	maximum
Gestational age (weeks)	26 (25 – 28)	23	40
Birth weight (g)	940(692 – 1110)	450	1960
Postnatal age (days)	92(63-128)	14	311
Duration of MV (days)	92(59 – 162)	30	366
Pregnancy parity	3(2-4)	1	11
Birth parity	2(1-2)	1	5

The Me birth weight was 960 grams, with a minimum body weight of 450 grams. The median age of infants admitted to the PICU is 92 days. The youngest infant was 14 days old, while the oldest was 311 days old. Thus, the study group included 50 patients (64.1%) with ELBW, 18 patients (23.1%) with very low birth weight (VLBW), 10 patients (12.8%) with low birth weight (LBW), and 1 full-term baby.

Five patients (6.4%) in the ELBW group had an Apgar score of less than three points five minutes after birth. However, none of them experienced a drop in blood pH from the umbilical vein below 7.0 immediately after birth. In 37 patients (78.2%), the Apgar score was between three and seven points within the first five minutes after birth.

In 63 cases (80.7%), antenatal steroids was used to prevent RDS. Surfactant was administered after birth in 73 cases (93.6%).

Chest X-rays of 13 patients (16.6%) revealed lung fibrosis in addition to severe BPD. Fifty-six patients (71%) showed signs of Itsenko–Cushing syndrome because they received periodic treatment with dexamethasone for BPD.The median duration of MV for our infants was 92 days. This ranged from a minimum of 30 days to a maximum of 366 days on the ventilator. High-frequency ventilation was used briefly in three cases (3.8%), but the main method was conventional ventilation (CV). In one case, a tracheostomy was performed on a six-month-old child. In another case, tracheal stenosis developed.

Before starting CV, NCPAP was used in 10 cases (12.8%), and after extubation — in 25 cases (32%). Caffeine citrate was given to 70 patients (89.7%), with 16 of them receiving it from birth to prevent BPD.

In 13 (16.6%) cases, abdominal surgery was performed. In 7 (8.9%) cases, the patent ductus arteriosus was surgically closed. In one case, retinal laser coagulation was done.

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According to the nosological structure (Table 2), the most common diseases were RDS (98.7%) and BPD (100%). Sepsis was detected in 35.9% of cases, while NEC and pneumonia was observed in 12.8% of cases. Among the complications, anemia was the most prevalent (100%), neurological complications (up to 78.2% in total), cardiopathy (20.5%) and retinopathy of prematurity (38.3%).

Table 2: The structure of nosology

Diseases	Abs.	%
Respiratory distress syndrome	77	98,7
Bronchopulmonary dysplasia	79	100
Sepsis	28	35,9
Necrotizing enterocolitis	10	12,8
Asphyxia	8	10,2
Pneumonia	10	12,8
Congenital cytomegalovirus infection	5	6,4
Multiple congenital malformations	3	3,8

6.2 Clinical and Respiratory Management at Admission

At the initial stage of the study, after admission to the PICU, infants were on MV with Pressure Control Ventilation (A/C - PC) mode for a period of several minutes to several hours. This was due to the fact that the Intermittent Mandatory Ventilation – Pressure Control (IMV -PC) mode was used during the transportation of infants.

The situation was further complicated by the fact that many infants were under deep sedation during transportation. This was accompanied by the shutdown of patient's respiratory drive. At the same time MV represented by autocycling and no triggering with passive inflation of lungs, which was followed by tightening of ventilation settings. Consequently, it took patients time to recover from sedation and to have a more accurate assessment of their respiratory condition.

During this period, the "working" Vt, the "working" PIP, and the "best" PEEP are determined. The flow-volume curve is being studied to find the optimal settings for full exhalation. Therefore, this time period may vary depending on the individual patient and the specific clinical situation. After that, it switches to the SIMV-VC mode, which has a downward flow curve, and starts the developed ventilation protocol.

Asynchronies were determined non-invasively, under visual control, using flow and pressure curves. The asynchrony index (AI) was evaluated starting from the first day of therapy in PICU and up to extubation [9].

6.3 Results

Table 3 shows the values of the ventilation settings that were achieved in patients in this group after being admitted to the PICU. We present fairly high values for ventilation settings. However, it should be noted that our patients tolerated them well. The incidence of complications such as pneumothorax in the neonatal period was 8 (10.2%) cases. However, during our treatment, pneumothorax developed only in 2 (2.6%) cases ($p<0.05$, Fischer's exact test). This indicates that the method of MV we used is quite safe.

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Table 3: The dynamic changes in ventilation settings.

Ventilation settings	T1	T2	Wilcoxon test
Targeted Vt (ml/kg)	11(9-12)	7(6-8) (2 w)	<0,05
"Working" PIP cm H2O	28(27-29)	22,4 (21-23) (72 h)	<0,05
"Best" PEEP cm H2O	7,9 (7-8)	6,9 (6-7) (2 w)	<0,05
FiO2 of targeted SpO2	52 (48-57)	34(30 -38) (24 h)	<0,05
RR (SIMV cycles/min)	23 (22-26)	13(10-15) (24 h)	<0,05
Asynchrony index (AI)(%)	35 (24-43)	12 (10-13) (ext.)	<0,05

T1 - the initial stage of treatment, after transportation to PICU and the start of VTV ventilation mode, T2 - a time point for estimating changes in ventilation settings over time: w – weeks, h – hours, ext -before extubation, AI (%) =number of asynchronous cycles / total RR (cycle frequency of the device + number of lost respiratory attempts) *100.

The oxygen fraction (FiO2) needed to achieve the target saturation level of 89-95% decreases during the first day to less than 40% (see Table 3). This decrease is statistically significant ($p<0.05$, Wilcoxon test).

From 24 to 72 hours after starting treatment, there is an automatic reduction in PIP levels by 20% or more (see Table 3). This reduction is also statistically significant ($p<0.05$, Wilcoxon test). However, the length of time it takes for the child to be ready to be weaned from the ventilator varies greatly from one infant to the next.

The frequency of “BPD spells” in the infants decreases, which may require sedation or even the use of muscle relaxants with increasing PEEP by 1-2 cm, depending upon severity. Actually, these occur due to thrachea-/bronchomalacia, as infants have increased airway compliance. Usually, this infants clinically present with significant airway obstruction and severe cyanosis. Bronchodilators are ineffective in this situation. We use generally fentanyl (1-3 μg/kg) for the short term, for prolonged sedation, midazolam for infants >32 weeks gestational age up till 6 months with starting dose 0.06 mg/kg/h (1 μg/kg/min). For infants >6 months old starting dose 0.05 – 0.12 mg/kg, supporting dose 0.o5 - 0.12 mg/kg/h (1-2 μg/kg/min). Fore paralysis, we are using rocuronium bromide 0.3 – 0.6 mg/kg/dose.

The median of asynchrony index (AI) at the beginning of therapy was 35% (Table 3), whereas before extubation, Me AI was already 12% ($p<0.05$, Wilcoxon test). In the structure of patient-ventilator asynchronies, at the beginning of treatment in our PICU, the frequency of auto-triggering was 69.6% (55 patients), double triggering 83.5% (66 babies), ineffective triggering occurred in 72 infants (91%), early switching was noted in 24 children (30.4%), switching delay to exhalation occurred in 77 patients (97.4%). Before extubation, the following were most often detected early switching 55,6% (44 patients) and double triggering 45,5% (36 infants), other types of asynchronies no more then 4% of study population. Thus, AI may be another indicator of infant weaning readiness.

Neonatal reintubation was performed in 65 (83.3%) cases. The average number of re-intubations per patient was 2, and the maximum was 10. At our level of care, reintubation was performed in only 16 (20.5%) patients ($p<0.05$, Fisher's exact test). The average number was 1 and the max was 3.

The mortality rate was 7.6% in the study group (6 cases), which is lower than the a priori expected mortality rate of 25% for patients who have ineffective weaning [3]. So, out of the 79 patients, 72 infants were successfully extubated up till one year of life, which is 91,1 % of them. The duration of weaning infants with PV ranged from 16 to 55 days. The median weaning period was 29 days, with a lower quartile (LQ) of 21 days and an upper quartile (UQ) of 36 days. We calculated the relative risk (rr) and the Number

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Needed to Treat (NNT) for our protocol based on the known mortality rates for ineffective weaning. It was considered unethical to use a comparison group in this study, so we calculated the risk and benefit without one. The results showed the following: rr – 0.3, NNT (Benefit) – 5.6 with 95% confidence interval (CI) -3.4 to 15.4.

7. Discussion

Usually, most infants who are on PV need to be intubated from birth. According to the European Consensus Guidelines on the Management of Respiratory Distress Syndrome, VTV should be used in these cases (A1) [10].

For premature infants with respiratory distress syndrome (RDS) and birth weights ranging from 1250 to 2500 grams, the target tidal volume (Vt) is set between 4 and 4.5 ml/kg [11]. For infants with birth weights between 700 and 1249 grams, Vt is adjusted to 4.5–5 ml/kg [11]. For infants weighing less than 700 grams, the target Vt is 5.5–6 ml/kg [11].

In premature infants with a gestation period of 32 weeks or more and normal lungs, the target Vt should be between 4 and 4.5 ml/kg [11].

For premature infants with RDS and birth weights between 1250 and 2500 grams, the PIP limit is usually set at 26 cm H2O. For infants weighing 1249 grams or less (including all children with ELBW), the limit is set at 24 cm H2O [11].

For premature infants with a gestation period of 32 weeks or more and normal lung development, the maximum PIP level is usually set at 18 cm H2O [11].

If the ventilator has the AutoFiO2 option, the target saturation limits are set at 89–94%.

The PEEP level is selected to ensure that the FiO2 is at an acceptable level, no higher than 30%. The SpO2 should also meet the target levels, and the hemodynamics should remain stable. The PEEP level is usually set between 6 and 8 cm H2O for these infants [11].

The RR depends on the patient's respiratory activity. These ventilation settings can be adjusted depending on the patient's breathing efforts and changes in blood gas composition.

As pulmonary compliance improves with these ventilation settings, the PIP and FiO2 decrease automatically in real time. This ensures maximum safe ventilation for the newborn.

When moderate ventilation settings characteristic of normal lungs are reached, the patient should be immediately extubated. This is done under the condition of normal blood gas parameters, a stable acid-base state, and stable hemodynamics. It is important that the device for NCPAP or noninvasive ventilation is within walking distance [11].

If the respiratory drive is insufficient, caffeine can be used to facilitate weaning from the ventilator. It is also used in a set of measures to prevent breathing problems in premature infants.

Infants who have been on PV (more than 7 days) usually do not effectively wean. As a rule, such children need reintubation and resumption of MV. In addition, they are at risk of developing BPD [12].

We often observe phenomena of increased respiratory drive in children on a ventilator with PV, the causes of which are not exactly clear, perhaps this is a consequence of a false picture of improved respiratory biomechanics. This is due to the fact that not only pulmonary, but also thoracic or even thoracoabdominal compliance affects the pressure in the respiratory tract to a greater extent [7].

During periods of increased respiratory drive and an increase in the RR of the patient, as a result of increased work of the respiratory muscles, there is a drop in airway pressure, which is reduced from the PIP level, and the volume of inspiration may remain acceptable or even slightly increased, as a result, the PIP level decreases to normal values, chest retractions are not observed, so how else is available weakness of the respiratory muscles. At the same time, on the monitor of the ventilator, the doctor can see a picture of "false" well-being, characteristic of the absence of pathology on the part of the lungs; in most cases, the blood gas composition may correspond to the norm or tend to a slight respiratory alkalosis, which is considered acceptable by many authors [11], and this

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general picture is decisive in making a decision about weaning from the ventilator and early extubation of the patient. Such weaning is unintentionally early, and, as a rule, it is followed by reintubation, and sometimes the need for unacceptably long-term use of non-invasive respiratory support methods and deterioration of the general condition of a sick newborn baby. Therefore, all newborns on MV need to be prevented from unintentionally early extubation, which can also be a warning of PV.

This is why we believe that AI assessment is important in newborns on MV. Ineffective triggering is associated with a weakness in the patient's inspiratory efforts, up to the complete absence of respiratory activity (then auto-triggering takes place) or the presence of PEEPi, which increases the efforts required to activate the trigger [13]. In newborns, weak inspiratory efforts would be associated with low gestational age, asphyxia, sepsis, congenital metabolic defects, CNS lesions – IVH, PVLM, Dandy – Walker syndrome, etc. congenital malformations. Auto-triggering can also be generated by cardiogenic oscillation or leakage from the respiratory circuit [13]. In cases of severe BPD, it is highly likely that there is a PEEPi. To prevent auto-triggering and/or ineffective triggering, PEEP needs to be increased to a higher level compared to RDS.

Double triggering occurs when there is high demand for ventilation from the patient and a very short inspiratory time (Tinsp) [13]. In newborns, this condition can be observed with hyperpnea. Usually it can be associated with acidosis, liver failure, febrile states, hypovolemia, central nervous system damage, and congenital metabolic defects associated with hyperammonemia. Sometimes the cause of the hyperpnea remains unrecognized. Double triggering can also be associated with low PaO2/FiO2, A/C mode, short Tins, high PIP, high level of PEEP [13]. Ineffective triggering may be associated with male gender, increased blood bicarbonate and alkalosis, high pSOFA (nSOFA) score, low trigger sensitivity, high Vt, high PIP, and high Psupport [13]. Excessive sedation and muscle relaxation may also be a risk factor for patient-ventilator asynchronies [13].

As far as we believe, the measures to reduce the risk of patient-ventilator asynchrony in newborns may include the following:

*Antenatal steroids prevention of RDS

*Adequate neuroprotection at the antenatal level using magnesium sulfate in accordance with WHO recommendations

*Adequate surfactant administration, the use of caffeine citrate in premature infants from birth for the prevention of BPD

*Invasive respiratory support includes ventilation in synchronized VTV modes, in particular VG, when using higher frequency ventilation (HFV), also with the VG option

*Rational use of sedation and muscle relaxation

*Manipulations with ventilation settings: careful adjustment of Tinsp and RR (to avoid hyperventilation in premature infants), PEEP, implication with the principles of lung protective, the use of permessive hypoxemia and hyperkapnia to prevent Ventilator-Induced Lung Injury (VILI)

* Monitoring of pressure and flow curves in the respiratory tract in order to timely diagnose asynchronies, AI assessment.

During periods when the patient himself generates an increased Vt, it is most likely necessary to increase the target Vt — this can also prevent significant fluctuations in mean airway pressure (MAP), which could cause another serious complication in neonatal PV — IVH [11].

The time constant in children on long-term ventilation increases from 0.14±0.01 s on the 10th-20th day of postnatal life to 0.33±0.02 s at the age of 6 months and 0.48±0.03 at the age of 1 year [7].It is known that it takes three-time constants to exhale 95% of the inspiratory volume, and 5-time constants to completely finish expiration. Taking into account the data given that at the age of 6 months of postnatal life the time constant is 0.3 s, 1.5 s (0.3 s×5=1.5 s) is required to complete exhalation [7]. The inspiration time constant should be shorter than the expiration time constant, but the inspiration time should be at

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least 0.5 s. Accordingly, RR in 6 months of postnatal life in a child on PV with severe BPD should be no more than 30 per minute (60 s/2 s per cycle [0.5 s+1.5 s]=30 per minute). And the inspiraton:expiration ratio (I:E) approximately equal to 1:3. At an older age, with a very severe degree of BPD, even 20 respirations per minute may be necessary (60 s/3 s per cycle [0.5 s+2.5 s]=20 per minute) [7], and the I:E ratio is approximately 1:5. Therefore, the low frequency of respiratory cycles during PV in infants of the first year of life is sufficiently justified.

Many experts in perinatal medicine, including neonatologists, pediatricians, and intensive care specialists, believe that using high levels of ventilator settings such as PEEP, Vt, and PIP can be dangerous for newborns and infants. This is because they may lead to the development of air leakage syndrome and VILI. However, a 25-year study published in 2024 shows that independent risk factors for pneumothorax have been identified: intubation at delivery room, preterm premature rupture of membranes > 7 days, surfactant administration, and low gestational age [14]. Thus, we do not see among the risk factors those settings of ventilation that we are forced to keep high during MV in infants with PV. As for VILI, the preventive strategy is VTV [7, 11, 15]. Infants with a confirmed diagnosis of chronic pulmonary disease, such as severe BPD, can tolerate high levels of these settings (PIP and Vt) well [7]. This is consistent with our observations.

The study has limitations, including the impossibility of randomization due to ethical considerations. Further research will focus on expanding the study population in order to increase the reliability of the results. This will also allow the use of this protocol and, possibly, some modifications for groups of patients who were excluded from this study. This could significantly improve the quality of respiratory support for infants with PV.

8.Conclusions

1. This study highlights that despite the high values of ventilation settings such as Vt, PIP, PEEP, and the low RR of SIMV inspirations, it can be concluded that the MV protocol used for PV in infants of the first year of life with respiratory insufficiency of perinatal origin is well tolerated and safe enough, as evidenced by statistically significant results ($p<0.05$) the low incidence of pneumothorax compared to the neonatal stage and the low mortality rate are 3 times lower than the a priori probability of death in pathology associated with ineffective weaning from the ventilator.

2.Given the short period of time for significant automatic de-escalation of PIP by 20% or more ($p<0.05$), as well as a significant rapid decrease in FiO2 to 40% or less ($p <0.05$), and a significantly lower frequency of reintubation compared to the neonatal period ($P < 0.05$), rr - 0.3, NNT (Benefit) - 5.6 with a 95% CI of 3.4 - 15.4, it can be concluded that the use of the protocol of MV in SIMV mode is an effective strategy for infants of first year of life with perinatal lung injury.

Conflict of Interest

The authors declare no conflict of interest.

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