

Selection of the End-Expiratory Pressure for Mechanical Respiratory Support (Review)

Roman Y. Ovsiannikov*, Konstantin M. Lebedinskii

I. I. Mechnikov North-Western State Medical University, Ministry of Health of Russia,
47 Piskarevskii prospect, 195067 St. Petersburg, Russia

For citation: Roman Y. Ovsiannikov, Konstantin M. Lebedinskii. Selection of the End-Expiratory Pressure for Mechanical Respiratory Support (Review). *Obshchaya Reanimatologiya = General Reanimatology*. 2022; 18 (6): 50–58. <https://doi.org/10.15360/1813-9779-2022-6-50-58> [In Russ. and Engl.]

*Corresponding author: Roman Y. Ovsiannikov, ovsiannikov.roman@gmail.com

Summary

End-expiratory pressure remains one of the few parameters of mechanical respiratory support whose values have not been strictly regulated using the evidence-based approach. The absence of «gold standard» for end-expiratory pressure optimization together with its obvious significant contribution to the efficiency and safety of respiratory support has driven the search for the optimal method of choosing its values for several decades.

Aim of the review: to identify the optimal methods for determining the values of end-expiratory pressure based on the analysis of its positive and negative effects in the used strategies of mechanical respiratory support.

Material and methods. We analyzed 165 papers from the PubMed, Scopus, and RSCI databases of medical and biological publications. Among them we selected 86 sources that most completely covered the following subjects: respiratory support, end-expiratory pressure, recruitment, ventilation-perfusion relationships, metabolography, and gas analysis.

Results. We outlined the main positive and negative effects of the end-expiratory pressure with regard to both lung biomechanical characteristics and pulmonary perfusion. The evolution of views on the methods of determining optimal values of the end-expiratory pressure was reviewed with the emphasis on a certain «fixation» of the scientific community in recent decades concerning the opening of the alveoli. The promising techniques based on the analysis of the diffusion capacity of the lungs were presented.

Conclusion. Focusing on mechanical lung opening prevents the scientific community from advancing in the optimization of the end-expiratory pressure. Dynamic assessment of pulmonary diffusion efficiency provides a new perspective on the issue, offering additional ways to the development of «gold standard».

Keywords: *end-expiratory pressure; ventilation-perfusion relationships; shunt; alveolar dead space; compliance; gas analysis*

Conflict of interest. The authors declare no conflict of interest.

Read the full-text English version at www.reanimatology.com

Introduction

Mechanical respiratory support (MRS) is one of the most powerful, radical, and widely used methods of life support in the anesthesiologist's arsenal. However, the great potential of mechanical ventilation in the treatment of the most severe patients with acute respiratory failure has always been associated with the understanding of the many risks and possible harm. Several decades of clinical use of MRS have forged a strategy underlying current approaches to selecting mechanical ventilation parameters based on the open-lung concept and pulmonary-protective ventilation concept [1, 2]. Generated by initially controversial and even opposing considerations, today both concepts are equally recognized as obligatory for effective and safe MRS [3].

Currently, the MRS concepts quite specifically determine the recommended values of respiratory cycle parameters and, most importantly, provide guidance for their management regardless of the selected mode of mechanical support [4]. Thus, the

initial tidal volume should be equal to 8 ml per kg of ideal (predicted based on sex and height) body weight with subsequent reduction to 6–7 ml/kg. The respiratory rate should not exceed 35 per minute to achieve the target values of pCO₂ (end expiratory pressure as determined by capnography or arterial blood gas analysis). The ratio of inspiration time to exhalation time should ensure the initiation of the next inspiration at exhalation flow rate zero, controlled by the flow curve [2]. The inspiratory oxygen fraction should be sufficient to achieve a saturation (SaO₂ or SpO₂) of 88–95% followed by titration, if possible, to values <0.7. In these clear algorithms, however, there is a parameter, the positive end-expiratory pressure (PEEP), which values have not yet been so strictly regulated. The recommended PEEP values should be at least 5 cm H₂O but «probably greater than that» [5]. This unique uncertainty demonstrates not only the secondary role of this value in providing efficiency and safety of mechanical lung ventilation. In addition, it reflects high variability of the PEEP optimal value in different patients and

an absence of reasonable and generally accepted approach to its selection. In this paper, the main positive and negative effects of PEEP and the evolution of views on methods to determine its optimal values will be discussed.

The Effects of PEEP

Although the effects of PEEP were described by Alvan L. Barach et al. as early as in 1938 [6], the idea of its unfavorable effect on hemodynamics delayed its use. Only in 1967 David Ashbaugh, Thomas Petty and their colleagues described acute respiratory distress syndrome [7], and the term «residual positive pressure» was coined by John S. Inkster [8] at the IV World Congress of Anesthesiology in London (1968). The immediate purpose of PEEP is to counteract atelectasis, i. e., to compensate for the reduction of end-expiratory lung volume resulting from various disturbances of biomechanics of both lung tissue and chest wall structures [9, 10]. End of exhalation in MRS is the most critical period of the respiratory cycle in terms of possible alveoli collapse. Collapsed alveoli do not participate in gas exchange, the proportion of QS/QT shunt increases and, as a consequence, oxygenation decreases, while cyclic opening of collapsed alveoli leads to their mechanical damage (atelectotrauma) [11, 12]. Such changes can occur not only as a result of severe disease: an increased subphrenic pressure in the supine position is sufficient for their appearance [13, 14], which is commonly seen in obesity, pregnancy and other conditions causing abdominal hypertension [15–17], as well as in the use of hypnotics and myorelaxants [18, 19]. The potential harm and prevalence of such disorders have driven the use of positive end-expiratory pressure in almost all types and regimens of respiratory support.

In turn, excessively high PEEP causes several issues (leaving aside the effect on systemic and extrapulmonary organ hemodynamics manifesting in cardiopulmonary interactions and venous return, the effect of portocaval gradient on hepatic blood flow, and the impact of jugular vein drainage on intracranial pressure, as well as the other extrapulmonary effects). Firstly, PEEP shifts upwards the airway pressure curve, which at the same respiratory volume naturally increases peak pressure and the probability of alveolar barotrauma with the appearance of extra-alveolar gas in lungs [20, 21]. Secondly, it obviously affects pulmonary perfusion, and, as a consequence, blood oxygenation.

Perfusion in the pulmonary system occurs under relatively low pressure: normal pulmonary capillary pressure is 6–12 mm Hg, which is equivalent to 8–16 cm H₂O [22]. John West (1960) described gravitational pressure gradient in capillaries located at different heights of continuous fluid column in pulmonary vascular bed [23]. Vertical size of an

adult lung varies from 20 to 30 cm depending on the size and position of the body [24]. Since the Swan-Ganz catheter enables to measure pressure in pulmonary capillaries usually in the West zone III, and less often in West zone II [22], 20 cm H₂O can be taken as approximate upper limit of hydrostatic addition to measured pulmonary capillary pressure, which prevents capillary collapse under intra-alveolar pressure. In spontaneous breathing, the latter fluctuates ± 1 cm H₂O that does not interfere with blood flow even in the most «gravitationally impaired» zones [25]. However, in case of intra-alveolar pressure increase, e.g. during exertion, coughing, Valsalva test or mechanical ventilation, it may be high enough to compress the capillary system not only in «vulnerable» (upper with respect to the direction of gravity) areas [26].

In fact, the intra-alveolar pressure in mechanical ventilation reaches 30, and in some settings up to 40 cm H₂O. [27]. And if the maximum value of hydrostatic pressure in the pulmonary capillary, taking into account the hydrostatic gradient along the lung height, as shown above, can reach only 36 cm of water column, the recruitment maneuver according to the well-known «40×40» method ensures a rather long (40 s) episode of 40 cm H₂O pressure in all open alveoli with all the ensuing consequences. These consequences include «squeezing» of pulmonary blood flow into those parts of lungs, where it remains mechanically possible, i. e. where alveoli are not opened [28]: in mechanically heterogeneous lungs Pascal's law is valid only for continuous column of fluid in vessels.

From the point of view of pulmonary perfusion, low PEEP is beneficial, while its high values are associated with expanded West zones I and II zones and larger proportion of alveolar dead space (ADS) [29]. The rise of ADS affects external respiration not only by expanding the useless ventilation zone. Blood flow literally «squeezed out» from these zones becomes enhanced in the perfused areas [30]. Such increase in perfusion volume, according to the fundamental ideas of H. Rahn and W.O. Fenn [31] can exceed the possibilities of gas diffusion rate (first of all, of less soluble oxygen!), that will eventually result in venous shunting in lungs as well [32], though the characteristic «non-ventilated but perfused» alveoli are absent in this case. Apparently, this mechanism of local pulmonary circulation overperfusion underlies hypoxemia created by ground glass opacities in novel coronavirus infection COVID-19 [33]. The symmetric effect of such irregularity on blood desaturation in the systemic capillaries, underlying the concept of weak microcirculatory units and explaining the abnormally high venous saturation without participation of arteriovenous anastomoses, was shown in a rather illustrative model [34].

Methods for Determining The Optimal PEEP

As for the methods for determining the optimal PEEP, one cannot avoid a historical overview here as well. Over more than half a century, dozens of different techniques have been proposed, reflecting a certain evolution of approaches [35–37]. The key indicator of the efficiency of ventilation is the diffusion rate of gases. This parameter cannot be assessed directly at the patient's bedside, so the arterial blood gas analysis which allows assessment of perfusion efficiency, took the leading place in selection of optimal parameters of respiratory support. Gas analysis has been used since the introduction of PEEP technique until now, e.g., as FiO_2/PEEP chart of ARDS.net project. However, the invasiveness of this approach and the need for regular blood sampling prompted the search for alternatives. The focus was placed on perfusion and ventilation, which matching directly affects diffusion, with blood gas analysis serving as a reference method.

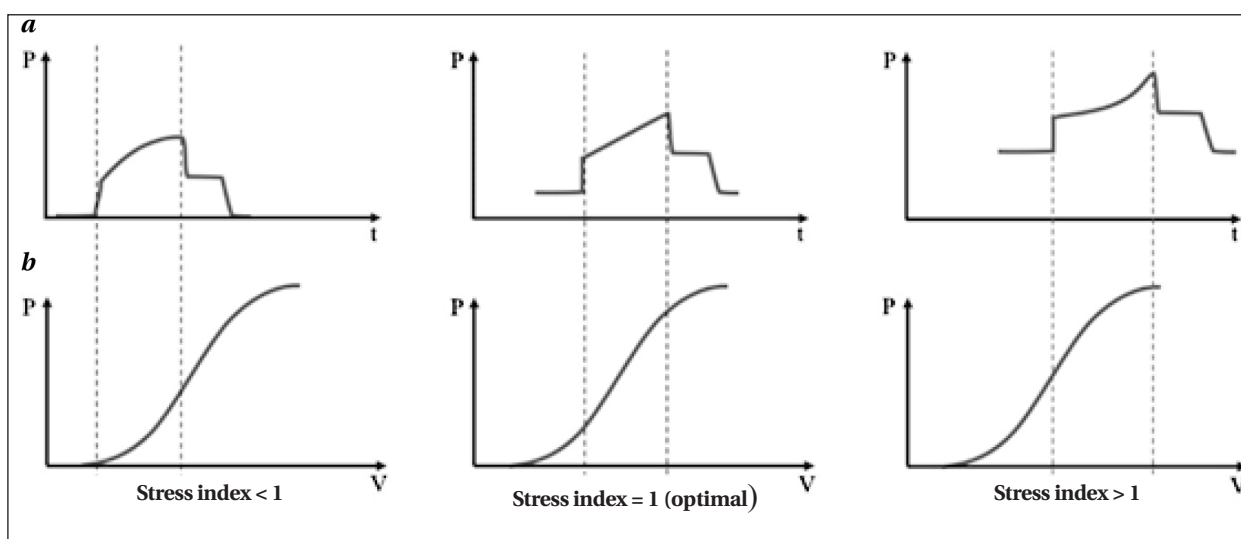
Initially, the negative effect of positive end-expiratory pressure on perfusion was associated mainly with a decrease in cardiac output [38]. Improvement of oxygenation, in turn, was attributed to reduced shunt fraction [39]. The main parameter for determining the optimal PEEP level, in addition to blood oxygen level, was cardiac output. Meanwhile, biomechanical lung parameters, such as static compliance, were only a potential alternative at that time [40].

However, a number of key works underlying the modern concepts shifted the emphasis from perfusion to ventilation, defining the trend for decades to come. Thus, the study of J. Mead, T. Takishima and D. Leith, dealing with biomechanical characteristics of the lungs and the theory of atelectotrauma [41], was the basis of the «open

lungs» concept by B. Lachmann [1], whereas the research of M. B. Amato [2] laid the foundation of a pulmonary-protective ventilation. In these papers, the emphasis was made on lung biomechanics, and the main goal was formulated as «to open alveoli, and while maintaining their patency, reduce damaging effects on lung tissue both from respiratory support device and from the lungs themselves». Recruitment of alveoli in ventilation became the leading purpose of PEEP, and the emphasis of damaging action had shifted towards barotrauma. This approach became the foundation of the modern paradigm of respiratory support, which was reflected in the methods of selecting the optimal values of PEEP.

Compliance, already a true biomechanical parameter, has become the key for most of them. Collapsed as well as overstretched alveoli have low compliance, showing high resistance to further stretching. In fact, several techniques are based on the avoidance of such low compliance. They differ only in the choice of an indicator for PEEP level setting: from direct analysis of static or dynamic compliance [42, 43], searching for inflection points on inspiratory or expiratory pressure-volume curves [44–46], to complex formulas for calculation of the moment of its maximal increase [47, 48]. The idea of finding the point of maximum compliance to set the PEEP level has evolved into the idea of estimating the damaging flow energy analysis. Thus, the so-called «stress index», based on pressure-time curve analysis, has been described [49, 50], and its target values, approximately equal to 1, are reached when most of the inspiration period lies in the zone of maximum compliance (Fig.).

Determination of the optimal pressure zone, in which the flow energy has the least damaging effect, naturally evolved into the concept of minimization of this energy. The driving pressure deter-



Pressure-time (a) and pressure-volume curves (b). The differences in the stress index are shown (author's illustration).

mined as a ratio of tidal volume to respiratory system compliance has become an integral indicator of dynamic stress caused by mechanical ventilation. In everyday practice the driving pressure is calculated as the difference between inspiratory plateau pressure and PEEP. Based on the above, achieving minimum driving pressure is possible both by maximizing compliance, as mentioned above, and by minimizing respiratory volume either directly or through reducing the difference of peak pressure and PEEP, which has become a modern trend in MRS [51].

In addition to the parameters measured inside the respiratory circuit, the assessment of intrapleural pressure is of great interest. As there is no acceptable way of direct assessment of this pressure today, the search went on around indirect approaches, the simplest and most reproducible of which turned out to be esophageal manometry [52–54]. The value of pressure in esophageal lumen, taken equal to intrathoracic (and intrapleural) pressure, enables to calculate transpulmonary pressure, which represents pressure gradient between intra-alveolar and intrapleural pressure. Some authors think that this pressure can reflect the real load on lung tissue, and serve as an indicator of PEEP level adjustment [55–58]. Volumes can also be analyzed: for instance, pulmonary volumes and capacities can be evaluated using the nitrogen washout method. This method is used to analyze end-expiratory lung volume (EELV) during the most dangerous phase of respiratory cycle in terms of atelectasis [59–61].

The physical characteristics of the lung can also be assessed through computed tomography (CT), dynamic bioimpedance measurement, and ultrasonography. CT in theory can allow to detect areas of atelectasis and overstretching and also to predict mechanical density using X-ray density and to estimate the weight of lung tissue to be resisted to open alveoli, thus selecting the optimal level of PEEP [62–65]. However, time-consuming character and potential harm of regular optimization of PEEP through CT scanning do not allow this method to be widely implemented in practice. Bioimpedance has shown to be very promising, although the geometric complexity of the thorax does not allow to precisely specify the conduction of electric current through the tissues [66, 67]. Ultrasound is a much simpler alternative to electromagnetic techniques, which allows assessing alveolar opening with high accuracy, but does not permit to determine the damaging energy of gas flow and alveolar overstretching [68–70].

Chronologically, we can observe certain focusing of researchers first on systemic hemodynamics and then on «recruitment and derecruitment» of alveoli, characteristic for the last two or three decades [71]. Contemporary studies have just started to downplay the significance of total lung recruit-

ment, speaking about physiological prospects of «moderate» opening [72, 73], proving once again that the ultimate goal of MRS is not the maximum number of opened alveoli, but an absolutely different result which is normal (or maximally close to normal!) pulmonary gas exchange, i. e., values of minute oxygen uptake (VO_2) and carbon dioxide elimination (VCO_2) [74, 75]. And this result is not obviously related to the proportion of open alveoli, especially considering the cost of «side effects» that often has to be paid for opening the alveoli and keeping them patent. Thus, PEEP may be unjustifiably high not in terms of alveoli overstretching or reduced venous return, but due to unfavorable redistribution of pulmonary capillary blood flow to the zones of collapsed yet perfused alveoli [76, 28].

Owing to this paradigm shift, the clinicians are able to focus on the clinical and physiological result of diffusion assessed by arterial blood gas analysis and by volumetric gas analysis of respiratory mixture. In this context, the situation partly resembles the evolution of ideas about cardiac preload, when the estimation of ventricular filling pressures in recent decades was supplemented by the possibility to estimate the result of end-diastolic volumes on cardiac chambers [22].

In recent years the technique of volumetric capnography, which helps assess the diffusion processes during the adjustment of PEEP level, has gained popularity [77, 78]. The transient increase in VCO_2 associated with changes in the PEEP level occurs due to an increase in the efficiency of diffusion, which can be globally considered as a positive effect. However, the transient increase in PEEP indicates a decrease in the shunt fraction, while its decrease suggests a reduced alveolar dead space fraction. A transient decrease in VCO_2 level, which can be caused by an increase in shunting or anatomical dead space, is considered negative [79–81].

In view of the recent studies on the interpretation of the VCO_2 to determine the optimal PEEP values [82], the changes of carbon dioxide production are worth noting. The duration of these changes is a major parameter. Variations in VCO_2 associated with altered proportion of alveolar dead space or shunt reflect instantaneous changes in the release of this gas, while they last only a few minutes. Longer variations rather reflect changes in minute alveolar ventilation or metabolic carbon dioxide production rate and are not directly related to PEEP level optimization. Besides an isolated estimation of VCO_2 , it is also possible to estimate lung oxygen uptake (VO_2), which, according to E. V. Ruchina et al. (2013), can be even more sensitive to PEEP level changes compared to VCO_2 [83], probably due to a greater diffusivity of the first gas. Moreover, simultaneous estimation of the exchange of both gases would potentially increase the specificity of this technique.

The prevalence of techniques of respiratory support parameter selection that exclusively focus on alveolar opening reflects the established belief of both researchers and clinicians that optimization of ventilation is similar to optimization of lung gas exchange, which in turn exhaustively confirms the normal blood gas composition. Such an approach has been described in recent papers of leading international specialists and included in contemporary Russian clinical guidelines [71, 84–86]. However, the ultimate goal of both natural and mechanical lung ventilation is the most effective pulmonary gas exchange in the current clinical situation, which depends not on the optimization of ventilation and/or blood flow values, but on their proportional matching implying the maximum achievable value of the diffusion surface of the lungs. Based on the above, dynamic analysis of respiratory gas production and consumption with high time resolution is very promising, in our opinion. As an instrumental alternative to the volumetric capnography, the use of metabolic modules from various manufacturers, whose function is the continuous parallel calculation of the volumes of carbon dioxide produced and oxygen consumed

for the realization of indirect calorimetry, can be proposed. Although their task is not to optimize ventilation, the data obtained with their help have a trend pattern, visually convenient for interpretation, and the simultaneous assessment of the diffusion intensity of the two main gases allows us to hope for greater sensitivity and specificity.

Conclusion

The ambiguity of parameters, methods and criteria for the selection of optimal end-expiratory pressure during mechanical respiratory support emphasizes high individual variability of this parameter in patients and limitation of most known approaches to its selection, focused on the involvement of alveoli in ventilation, but ignoring ventilation-perfusion relationships.

The possibility of dynamic assessment of pulmonary diffusion efficiency makes volumetric oximetry and capnometry a promising approach to the selection of optimal value of end-expiratory pressure, integrally reflecting ventilation-perfusion matching, which requires further study and practical implementation of the method.

References

1. Lachmann B. Open up the lung and keep the lung open. *Intensive Care Med.* 1992; 18 (6): 319–321. DOI: 10.1007/BF01694358. PMID: 1469157.
2. Amato M.B., Barbas C.S., Medeiros D.M., Schettino G. de P., Lorenzi Filho G., Kairalla R.A., Deheinzelin D., Morais C., Fernandes E. de O., Takagaki T.Y. Beneficial effects of the «open lung approach» with low distending pressures in acute respiratory distress syndrome. A prospective randomized study on mechanical ventilation. *Am J Respir Crit Care Med.* 1995; 152 (6 Pt 1): 1835–1846. DOI: 10.1164/ajrcm.152.6.8520744. PMID: 8520744.
3. Кузьков В.В., Суборов Е.В., Фот Е.В., Родионова Л.Н., Соколова М.М., Лебединский К.М., Киров М.Ю. Послеоперационные дыхательные осложнения и ОРДС легче предупредить, чем лечить. *Анестезиология и реаниматология.* 2016; 61 (6): 461–468. DOI: 10.18821/0201-7563-2016-6-461-468. eLIBRARY ID: 28390531. EDN: XXHAGZ. [Kuzkov V.V., Suborov E.V., Fot E.V., Rodionova L.N., Sokolova M.M., Lebedinsky K.M., Kirov M.Yu. Postoperative pulmonary complications and acute respiratory distress syndrome — better prevent than treat. *Anesteziol.Reanimatol/Anesteziologiya i reanimatologiya.* 2016; 61 (6): 461–468. (in Russ.). DOI: 10.18821/0201-7563-2016-6-461-468. eLIBRARY ID: 28390531. EDN: XXHAGZ.].
4. Acute Respiratory Distress Syndrome Network, Brower R.G., Matthay M.A., Morris A., Schoenfeld D., Thompson B.T., Wheeler A. Ventilation with lower tidal volumes as compared with traditional tidal volumes for acute lung injury and the acute respiratory distress syndrome. *N Engl J Med.* 2000; 342 (18): 1301–1308. DOI: 10.1056/NEJM20000 5043421801. PMID: 10793162.
5. Brower R.G., Lanken P.N., MacIntyre N., Matthay M.A., Morris A., Ancukiewicz M., Schoenfeld D., Thompson B.T., National Heart, Lung, and Blood Institute ARDS Clinical Trials Network. Higher versus lower positive end-expiratory pressures in patients with the acute respiratory distress syndrome. *N Engl J Med.* 2004; 351 (4): 327–336. DOI: 10.1056/NEJMoa032193. PMID: 15269312.
6. Barach A.L., Martin J., Eckman M. Positive pressure respiration and its application to the treatment of acute pulmonary edema. *Ann Intern Med.* 1938; 12: 754–795. DOI: 10.7326/0003-4819-12-6-754.
7. Ashbaugh D.G., Bigelow D.B., Petty T.L., Levine B.E. Acute respiratory distress in adults. *Lancet.* 1967; 2 (7511): 319–323. DOI: 10.1016/s0140-6736 (67)90168-7. PMID: 4143721.
8. Craft A. John Scott Inkster. *BMJ.* 2011; 343: d7517. DOI: 10.1136/ bmj.d7517.
9. Rusca M., Proietti S., Schnyder P., Frascarolo P., Hedenstierna G., Spahn D.R., Magnusson L. Prevention of atelectasis formation during induction of general anesthesia. *Anesth Analg.* 2003; 97 (6): 1835–1839. DOI: 10.1213/01.ANE.0000087042.02266.F6. PMID: 14633570.
10. Mélot C. Contribution of multiple inert gas elimination technique to pulmonary medicine. 5. Ventilation-perfusion relationships in acute respiratory failure. *Thorax.* 1994; 49 (12): 1251–1258. DOI: 10.1136/thx.49.12.1251. PMID: 7878564.
11. Warner D.O., Warner M.A., Ritman E.L. Atelectasis and chest wall shape during halothane anesthesia. *Anesthesiology.* 1996; 85 (1): 49–59. DOI: 10.1097/00000542-199607000-00008. PMID: 8694382.
12. Muscedere J.G., Mullen J.B., Gan K., Slutsky A.S. Tidal ventilation at low airway pressures can augment lung injury. *Am J Respir Crit Care Med.* 1994; 149 (5): 1327–1334. DOI: 10.1164/ajrccm.149.5.8173774. PMID: 8173774.

13. Muller N., Volgyesi G., Becker L., Bryan M.H., Bryan A.C. Diaphragmatic muscle tone. *J Appl Physiol Respir Environ Exerc Physiol.* 1979; 47 (2): 279–284. DOI: 10.1152/jappl.1979.47.2.279. PMID: 224022.
14. Petersson J., Ax M., Frey J., Sánchez-Crespo A., Lindahl S.G.E., Mure M. Positive end-expiratory pressure redistributes regional blood flow and ventilation differently in supine and prone humans. *Anesthesiology.* 2010; 113 (6): 1361–1369. DOI: 10.1097/ALN.0b013e3181fcec4f. PMID: 21068656.
15. Mutoh T., Lamm W.J., Embree L.J., Hildebrandt J., Albert R.K. Volume infusion produces abdominal distension, lung compression, and chest wall stiffening in pigs. *J Appl Physiol (1985).* 1992; 72 (2): 575–582. DOI: 10.1152/jappl.1992.72.2.575. PMID: 1559935.
16. Behazin N., Jones S.B., Cohen R.I., Loring S.H. Respiratory restriction and elevated pleural and esophageal pressures in morbid obesity. *J Appl Physiol (1985).* 2010; 108 (1): 212–218. DOI: 10.1152/japplphysiol.91356.2008. PMID: 19910329.
17. Pelosi P., Croci M., Ravagnan I., Tredici S., Pedoto A., Lissone A., Gattinoni L. The effects of body mass on lung volumes, respiratory mechanics, and gas exchange during general anesthesia. *Anesth Analg.* 1998; 87 (3): 654–660. DOI: 10.1097/00000539-199809000-00031. PMID: 9728848.
18. Warner D.O., Warner M.A., Ritman E.L. Human chest wall function while awake and during halothane anesthesia. I. Quiet breathing. *Anesthesiology.* 1995; 82 (1): 6–19. DOI: 10.1097/00000542-199501000-00003. PMID: 7832335.
19. Reber A., Nylund U., Hedenstierna G. Position and shape of the diaphragm: implications for atelectasis formation. *Anaesthesia.* 1998; 53 (11): 1054–1061. DOI: 10.1046/j.1365-2044.1998.00569.x. PMID: 10023273.
20. Dreyfuss D., Saumon G. Ventilator-induced lung injury: lessons from experimental studies. *Am J Respir Crit Care Med.* 1998; 157 (1): 294–323. DOI: 10.1164/ajrccm.157.1.9604014. PMID: 9445314.
21. Голубев А.М., Мороз В.В., Зорина Ю.Г., Никуфоров Ю.В. Морфологическая оценка безопасности «открытия» альвеол. *Общая Реаниматология.* 2008; 4 (3): 102. DOI: 10.15360/1813-9779-2008-3-102. [Golubev A.M., Moroz V.V., Zorina Yu.G., Nikiforov Yu.V. Morphological assessment of the safety of alveolar opening. *General Reanimatology/ Obshchaya reanimatologiya.* 2008; 4 (3): 102. (in Russ.) DOI: 10.15360/1813-9779-2008-3-102.].
22. Кровообращение и анестезия. Оценка и коррекция системной гемодинамики во время операции и анестезии. Изд. 2-е. Под ред. Лебединского К.М. СПб: Человек; 2015: 1076. [Blood circulation and anesthesia. Assessment and correction of systemic hemodynamics during surgery and anesthesia. 2nd Ed. Lebedinsky K.M. (Ed.). St. Petersburg: Man/Chelovek; 2015: 1076].
23. West J.B., Dollery C.T. Distribution of blood flow and ventilation-perfusion ratio in the lung, measured with radioactive carbon dioxide. *J Appl Physiol.* 1960; 15: 405–410. DOI: 10.1152/jappl.1960.15.3.405. PMID: 13844133.
24. D'Angelis C.A., Coalson J.J., Ryan R.M. Structure of the respiratory system: lower respiratory tract. Chapter 36. In: Fuhrman B.P., Zimmerman J.J., (eds.). (Fourth Edition). Mosby. *Pediatric Critical Care.* 2011: 490–498. DOI: 10.1016/B978-0-323-07307-3.10036-9.
25. Beachey W.D. Respiratory care anatomy and physiology: foundations for clinical practice, 2nd ed. St. Louis: MosbyElsevier; 2007: 45–47.
26. Pstras L., Thomaseth K., Waniewski J., Balzani I., Bellavere F. The Valsalva Manoeuvre: physiology and clinical examples. *Acta Physiol (Oxf).* 2016; 217 (2): 103–119. DOI: 10.1111/apha.12639. PMID: 26662857.
27. MacIntyre N.R., Branson R.D., eds. Mechanical Ventilation. 2nd ed. Saunders Elsevier; 2009: 411–412.
28. Лебединский К.М., Артюков Д.А., Борисов М.В., Громова Т.А., Сливин О.А. Раздельная вентиляция легких при их несимметричном поражении: частный случай как демонстрация общей проблемы. *Анестезиология и реаниматология* 2014; 59 (4): 72–74. [Lebedinsky K.M., Artyukov D.A., Borisov M.V., Gromova T.A., Slivin O.A. Independent lung ventilation for asymmetric injury: particular case as a demonstration of a common challenge. *Anesteziol. Reanimatol/ Anesteziologiya i Reanimatologiya.* 2014; 59 (4): 72–74. (in Russ.)].
29. Hakim T.S., Michel R.P., Chang H.K. Effect of lung inflation on pulmonary vascular resistance by arterial and venous occlusion. *J Appl Physiol Respir Environ Exerc Physiol.* 1982; 53 (5): 1110–1115. DOI: 10.1152/jappl.1982.53.5.1110. PMID: 6757207.
30. Wellhöfer H., Zeravik J., Perker M., Blümel G., Zimmermann G., Pfeiffer U.J. PEEP-induced changes of pulmonary capillary wedge pressure, prepulmonary and total intrathoracic blood volume in anesthetized dogs. In: Lewis F.R., Pfeiffer U.J. (eds). Springer, Berlin, Heidelberg. *Practical applications of fiberoptics in critical care monitoring.* 1990: 32–41. DOI: 10.1007/978-3-642-75086-1_4.
31. Rahn H., Fenn W.O. Graphical analysis of the respiratory gas exchange: the O₂-CO₂ diagram. Washington, DC: Am. Physiol. Soc., 1955.
32. Staub N.C., Bishop J.M., Forster R.E. Importance of diffusion and chemical reaction rates in O₂ uptake in the lung. *J Appl Physiol.* 1962; 17: 21–27. DOI: 10.1152/jappl.1962.17.1.21. PMID: 13916422.
33. Dhont S., Derom E., Van Braeckel E., Depuydt P., Lambrecht B.N. The pathophysiology of 'happy' hypoxemia in COVID-19. *Respir Res.* 2020; 21 (1): 198. DOI: 10.1186/s12931-020-01462-5. PMID: 32723327.
34. Ince C., Sinaasappel M. Microcirculatory oxygenation and shunting in sepsis and shock. *Crit Care Med.* 1999; 27 (7): 1369–1377. DOI: 10.1097/00003246-199907000-00031. PMID: 10446833.
35. Sahetya S.K., Goligher E.C., Brower R.G. Fifty years of research in ARDS: setting positive end-expiratory pressure in the acute respiratory distress syndrome. *Am J Respir Crit Care Med.* 2017; 195 (11): 1429–1438. DOI: 10.1164/rccm.201610-2035CI. PMID: 28146639.
36. Lemaire F., Brun-Buisson C. Positive end expiratory pressure. In: Lemaire F., ed. *Mechanical Ventilation.* Springer; 1991: 19–30. DOI: 10.1007/978-3-642-87448-2_2.
37. Gattinoni L., Carlesso E., Cressoni M. Selecting the «right» positive end-expiratory pressure level. *Curr Opin Crit Care.* 2015; 21 (1): 50–57. DOI: 10.1097/MCC.000000000000166. PMID: 25546534.
38. Cournand A., Motley H.L., Werko L. Mechanism underlying cardiac output change during intermittent positive pressure breathing (IPP). *Fed Proc.* 1947; 6 (1 Pt 2): 92. PMID: 20242338.

39. Dantzker D.R., Lynch J.P., Weg J.G. Depression of cardiac output is a mechanism of shunt reduction in the therapy of acute respiratory failure. *Chest*. 1980; 77 (5): 636–642. DOI: 10.1378/chest.77.5.636. PMID: 6988180.
40. Suter P.M., Fairley B., Isenberg M.D. Optimum end-expiratory airway pressure in patients with acute pulmonary failure. *N Engl J Med*. 1975; 292 (6): 284–289. DOI: 10.1056/NEJM197502062920604. PMID: 234174.
41. Mead J., Takishima T., Leith D. Stress distribution in lungs: a model of pulmonary elasticity. *J Appl Physiol*. 1970; 28 (5): 596–608. DOI: 10.1152/jappl.1970.28.5.596. PMID: 5442255.
42. Ferrando C., Mugarra A., Gutierrez A., Carbonell J.A., García M., Soro M., Tusman G., Belda F.J. Setting individualized positive end-expiratory pressure level with a positive end-expiratory pressure decrement trial after a recruitment maneuver improves oxygenation and lung mechanics during one-lung ventilation. *Anesth Analg*. 2014; 118 (3): 657–665. DOI: 10.1213/ANE.0000000000000105. PMID: 24557111.
43. Ярошецкий А.И., Проценко Д.Н., Ларин Е.С., Гельфанд Б.Р. Роль оценки статической петли «давление-объем» в дифференциальной диагностике и оптимизации параметров респираторной поддержки при паренхиматозной дыхательной недостаточности. *Анестезиология и реаниматология*. 2014; (2): 21–26. УДК 616.24-008.64-08: 615.816]-04. [Yaroshetsky A.I., Protsenko D.N., Larin E.S., Gelfand B.R. Significance of static pressure-volume loop in differential diagnostics and optimization of respiratory support in parenchymal respiratory failure. *Anesteziol.Reanimatol/ Anesteziologiya i Reanimatologiya*. 2014; (2): 21–26. (in Russ.). UDC 616.24-008.64-08: 615.816]-04].
44. Gattinoni L., D'Andrea L., Pelosi P., Vitale G., Pesenti A., Fumagalli R. Regional effects and mechanism of positive end-expiratory pressure in early adult respiratory distress syndrome. *JAMA*. 1993; 269 (16): 2122–2127. PMID: 8468768.
45. Ranieri V.M., Giuliani R., Fiore T., Dambrosio M., Milic-Emili J. Volume-pressure curve of the respiratory system predicts effects of PEEP in ARDS: «occlusion» versus «constant flow» technique. *Am J Respir Crit Care Med*. 1994; 149 (1): 19–27. DOI: 10.1164/ajrccm.149.1.8111581. PMID: 8111581.
46. Vieira S.R., Puybasset L., Lu Q., Richecoeur J., Cluzel P., Coriat P., Rouby J.J. A scanographic assessment of pulmonary morphology in acute lung injury. Significance of the lower inflection point detected on the lung pressure-volume curve. *Am J Respir Crit Care Med*. 1999; 159 (5 Pt 1): 1612–1623. DOI: 10.1164/ajrccm.159.5.9805112. PMID: 10228135.
47. Venegas J.G., Harris R.S., Simon B.A. A comprehensive equation for the pulmonary pressure-volume curve. *J Appl Physiol* (1985). 1998; 84 (1): 389–395. DOI: 10.1152/jappl.1998.84.1.389. PMID: 9451661.
48. Harris R.S., Hess D.R., Venegas J.G. An objective analysis of the pressure-volume curve in the acute respiratory distress syndrome. *Am J Respir Crit Care Med*. 2000; 161 (2 Pt 1): 432–439. DOI: 10.1164/ajrccm.161.2.9901061. PMID: 10673182.
49. Ranieri V.M., Zhang H., Mascia L., Aubin M., Lin C.Y., Mullen J.B., Grasso S., Binnie M., Volgyesi G.A., Eng P., Slutsky A.S. Pressure-time curve predicts minimally injurious ventilatory strategy in an isolated rat lung model. *Anesthesiology*. 2000; 93 (5): 1320–1328. DOI: 10.1097/00000542-200011000-00027. PMID: 11046222.
50. Grasso S., Terragni P., Mascia L., Fanelli V., Quintel M., Herrmann P., Hedenstierna G., Slutsky A.S., Ranieri V.M. Airway pressure-time curve profile (stress index) detects tidal recruitment/hyperinflation in experimental acute lung injury. *Crit Care Med*. 2004; 32 (4): 1018–1027. DOI: 10.1097/01.ccm.0000120059.94009.ad. PMID: 15071395.
51. Amato M.B.P., Meade M.O., Slutsky A.S., Brochard L., Costa E.L.V., Schoenfeld D.A., Stewart T.E., Briel M., Talmor D., Mercat A., Richard J.-C.M., Carvalho C.R.R., Brower R.G. Driving pressure and survival in the acute respiratory distress syndrome. *N Engl J Med*. 2015; 372 (8): 747–755. DOI: 10.1056/NEJMsa1410639. PMID: 25693014.
52. Brochard L. Measurement of esophageal pressure at bedside: pros and cons. *Curr Opin Crit Care*. 2014; 20 (1): 39–46. DOI: 10.1097/MCC.0000000000000050. PMID: 24300619.
53. Piraino T., Cook D.J. Optimal PEEP guided by esophageal balloon manometry. *Respir Care*. 2011; 56 (4): 510–513. DOI: 10.4187/respcare.00815. PMID: 21255501.
54. Beitler J.R., Sarge T., Banner-Goodspeed V.M., Gong M.N., Cook D., Novack V., Loring S.H., Talmor D., EPVent-2 Study Group. Effect of titrating positive end-expiratory pressure (PEEP) with an esophageal pressure-guided strategy vs an empirical high PEEP-FiO₂ strategy on death and days free from mechanical ventilation among patients with acute respiratory distress syndrome: a randomized clinical trial. *JAMA*. 2019; 321 (9): 846–857. DOI: 10.1001/jama.2019.0555. PMID: 30776290.
55. Yang Y., Li Y., Liu S.-Q., Liu L., Huang Y.-Z., Guo F.-M., Qiu H.-B. Positive end expiratory pressure titrated by transpulmonary pressure improved oxygenation and respiratory mechanics in acute respiratory distress syndrome patients with intra-abdominal hypertension. *Chin Med J (Engl)*. 2013; 126 (17): 3234–3239. PMID: 24033942.
56. Rodriguez P.O., Bonelli I., Setten M., Attie S., Madorno M., Maskin L.P., Valentini R. Transpulmonary pressure and gas exchange during decremental PEEP titration in pulmonary ARDS patients. *Respir Care*. 2013; 58 (5): 754–763. DOI: 10.4187/respcare.01977. PMID: 23051849.
57. Gulati G., Novero A., Loring S.H., Talmor D. Pleural pressure and optimal positive end-expiratory pressure based on esophageal pressure versus chest wall elastance: incompatible results. *Crit Care Med*. 2013; 41 (8): 1951–1957. DOI: 10.1097/CCM.0b013e31828a3de5. PMID: 23863227.
58. Ярошецкий А.И., Проценко Д.Н., Резепов Н.А., Гельфанд Б.Р. Настройка положительного давления конца выдоха при паренхиматозной ОДН: статическая петля «давление-объем» или транспульмональное давление? *Анестезиол. и реаниматол.* 2014; (4): 53–59. УДК 616.902: 71-06: 615-005.757.6 [Yaroshetsky A.I., Protsenko D.N., Rezepov N.A., Gelfand B.R. Positive end — expiratory pressure adjustment in parenchymal respiratory failure: static pressure-volume loop or transpulmonary pressure? *Anesteziol.Reanimatol/ Anesteziologiya i Reanima-*

- tologiya. 2014; (4): 53–59. (in Russ.). UDC 616.902: 71-06: 615-005.757.6].
59. Olegård C., Söndergaard S., Houlitz E., Lundin S., Stenqvist O. Estimation of functional residual capacity at the bedside using standard monitoring equipment: a modified nitrogen washout/washin technique requiring a small change of the inspired oxygen fraction. *Anesth Analg*. 2005; 101 (1): 206–212, table of contents. DOI: 10.1213/01.ANE.0000165823.90368.55. PMID: 15976233.
 60. Chiumello D., Cressoni M., Chierichetti M., Tallarini F., Botticelli M., Berto V., Mietto C., Gattinoni L. Nitrogen washout/washin, helium dilution and computed tomography in the assessment of end expiratory lung volume. *Crit Care*. 2008; 12 (6): R150. DOI: 10.1186/cc7139. PMID: 19046447.
 61. Dellamonica J., Lerolle N., Sargentini C., Beduneau G., Di Marco F., Mercat A., Richard J.-C.M., Diehl J.-L., Mancebo J., Rouby J.-J., Lu Q., Bernardin G., Brochard L. Accuracy and precision of end-expiratory lung-volume measurements by automated nitrogen washout/washin technique in patients with acute respiratory distress syndrome. *Crit Care*. 2011; 15 (6): R294. DOI: 10.1186/cc10587. PMID: 22166727.
 62. Cressoni M., Chiumello D., Carlesso E., Chiurazzi C., Amini M., Brioni M., Cadringer P., Quintel M., Gattinoni L. Compressive forces and computed tomography-derived positive end-expiratory pressure in acute respiratory distress syndrome. *Anesthesiology*. 2014; 121 (3): 572–581. DOI: 10.1097/ALN.0000000000000373. PMID: 25050573.
 63. Malbouisson L.M., Muller J.C., Constantin J.M., Lu Q., Puybasset L., Rouby J.J., CT Scan ARDS Study Group. Computed tomography assessment of positive end-expiratory pressure-induced alveolar recruitment in patients with acute respiratory distress syndrome. *Am J Respir Crit Care Med*. 2001; 163 (6): 1444–1450. DOI: 10.1164/ajrccm.163.6.2005001. PMID: 11371416.
 64. Reske A.W., Reske A.P., Gast H.A., Seiwerts M., Beda A., Gottschaldt U., Josten C., Schreiter D., Heller N., Wrigge H., Amato M.B. Extrapolation from ten sections can make CT-based quantification of lung aeration more practicable. *Intensive Care Med*. 2010; 36 (11): 1836–1844. DOI: 10.1007/s00134-010-2014-2. PMID: 20689909.
 65. Vieira S.R., Puybasset L., Richecoeur J., Lu Q., Cluzel P., Gusman P.B., Coriat P., Rouby J.J. A lung computed tomographic assessment of positive end-expiratory pressure-induced lung overdistension. *Am J Respir Crit Care Med*. 1998; 158 (5 Pt 1): 1571–1577. DOI: 10.1164/ajrccm.158.5.9802101. PMID: 9817710.
 66. Wolf G.K., Gómez-Laberge C., Rettig J.S., Vargas S.O., Smallwood C.D., Prabhu S.P., Vitali S.H., Zurakowski D., Arnold J.H. Mechanical ventilation guided by electrical impedance tomography in experimental acute lung injury. *Crit Care Med*. 2013; 41 (5): 1296–1304. DOI: 10.1097/CCM.0b013e3182771516. PMID: 23474677.
 67. Mauri T., Eronia N., Turrini C., Battistini M., Grasselli G., Rona R., Volta C.A., Bellani G., Pesenti A. Bedside assessment of the effects of positive end-expiratory pressure on lung inflation and recruitment by the helium dilution technique and electrical impedance tomography. *Intensive Care Med*. 2016; 42 (10): 1576–1587. DOI: 10.1007/s00134-016-4467-4. PMID: 27518321.
 68. Tusman G., Acosta C.M., Costantini M. Ultrasonography for the assessment of lung recruitment maneuvers. *Crit Ultrasound J*. 2016; 8 (1): 8. DOI: 10.1186/s13089-016-0045-9. PMID: 27496127.
 69. Bouhemad B., Brisson H., Le-Guen M., Arbelot C., Lu Q., Rouby J.-J. Bedside ultrasound assessment of positive end-expiratory pressure-induced lung recruitment. *Am J Respir Crit Care Med*. 2011; 183 (3): 341–347. DOI: 10.1164/rccm.201003-0369OC. PMID: 20851923.
 70. Cho R.J., Adams A., Ambur S., Lunos S., Shapiro R., Prekker M.E. Ultrasound assessment of diaphragmatic motion in subjects with ARDS during transpulmonary pressure-guided PEEP titration. *Respir Care*. 2020; 65 (3): 314–319. DOI: 10.4187/respcare.06643. PMID: 31690616.
 71. Gattinoni L., Marini J.J. In search of the Holy Grail: identifying the best PEEP in ventilated patients. *Intensive Care Med*. 2022; 48 (6): 728–731. DOI: 10.1007/s00134-022-06698-x. PMID: 35513707.
 72. Rezoagli E., Bellani G. How I set up positive end-expiratory pressure: evidence- and physiology-based! *Crit Care*. 2019; 23 (1): 412. DOI: 10.1186/s13054-019-2695-z. PMID: 31842915.
 73. Hess D.R. Recruitment maneuvers and PEEP titration. *Respir Care*. 2015; 60 (11): 1688–1704. DOI: 10.4187/respcare.04409. PMID: 26493593.
 74. Власенко А.В., Мороз В.В., Яковлев В.Н., Алексеев В.Г., Булатов Н.Н. Выбор способа оптимизации ПДКВ у больных с острым респираторным дистресс-синдромом. *Общая Реаниматология*. 2012; 8 (1): 13. DOI: 10.15360/1813-9779-2012-1-13. [Vlasenko A.V., Moroz V.V., Yakovlev V.N., Alekseev V.G., Bulatov N.N. Choice of a procedure for optimizing positive end-expiratory pressure in patients with acute respiratory distress syndrome. *General reanimatology/Obshchaya reanimatologiya*. 2012; 8 (1): 13. (In Russ.). DOI: 10.15360/1813-9779-2012-1-13.]
 75. Мороз В.В., Власенко А.В., Яковлев В.Н., Алексеев В.Г. Оптимизация ПДКВ у больных с острым респираторным дистресс-синдромом, вызванным прямыми и непрямыми повреждающими факторами. *Общая Реаниматология*. 2012; 8 (3): 5. DOI: 10.15360/1813-9779-2012-3-5. [Moroz V.V., Vlasenko A.V., Yakovlev V.N., Alekseev V.G. Optimization of positive end-expiratory pressure in patients with acute respiratory distress syndrome caused by direct and indirect damaging factors. *General reanimatology/Obshchaya reanimatologiya*. 2012; 8 (3): 5. (in Russ.). DOI: 10.15360/1813-9779-2012-3-5.]
 76. Karbing D.S., Panigada M., Bottino N., Spinelli E., Protti A., Rees S.E., Gattinoni L. Changes in shunt, ventilation/perfusion mismatch, and lung aeration with PEEP in patients with ARDS: a prospective single-arm interventional study. *Crit Care*. 2020; 24 (1): 111. DOI: 10.1186/s13054-020-2834-6. PMID: 32293506.
 77. Böhm S.H., Kremer P., Tusman G., Reuter D.A., Pullet S. Volumetric capnography for analysis and optimization of ventilation and gas exchange. [in German]. *Anaesthesist*. 2020; 69 (5): 361–1370. DOI: 10.1007/s00101-020-00747-0. PMID: 32240320.
 78. Kremer P., Böhm S.H., Tusman G. Clinical use of volumetric capnography in mechanically ventilated patients. *J Clin Monit Comput*. 2020; 34 (1): 7–116. DOI: 10.1007/s10877-019-00325-9. PMID: 31152285.

79. Yang Y., Huang Y., Tang R., Chen Q., Hui X., Li Y., Yu Q., Zhao H., Qiu H. Optimization of positive end-expiratory pressure by volumetric capnography variables in lavage-induced acute lung injury. *Respiration*. 2014; 87 (1): 75–183. DOI: 10.1159/000354787. PMID: 24296453.
80. Tolnai J., Fodor G.H., Babik B., Dos Santos Rocha A., Bayat S., Peták F., Habre W. Volumetric but not time capnography detects ventilation/perfusion mismatch in injured rabbit lung. *Front Physiol*. 2018; 9: 1805. DOI: 10.3389/fphys.2018.01805. PMID: 30618817.
81. Blankman P., Shono A., Hermans B.J.M., Wesseliuss T., Hasan D., Gommers D. Detection of optimal PEEP for equal distribution of tidal volume by volumetric capnography and electrical impedance tomography during decreasing levels of PEEP in post cardiac-surgery patients. *Br J Anaesth*. 2016; 116 (6): 862–869. DOI: 10.1093/bja/aew116. PMID: 27199318.
82. Ярошецкий А.И., Проценко Д.Н., Бойцов П.В., Ченцов В.Б., Нистратов С.Л., Кудряков О.Н., Соловьев В.В., Банова Ж.И., Шкуратова Н.В., Резепов Н.А., Гельфанд Б.Р. Оптимальное положительное конечно-экспираторное давление при ОРДС у больных с гриппом А (H1N1)pdm09: баланс между максимумом конечно-экспираторного объема и минимумом перераздувания альвеол. *Анестезиология и реаниматология*. 2016; 61 (6): 425–432. УДК: 616.24-008.64-06: 616.921.5]-073. [Yaroshetsky A.I., Protsenko D.N., Boitsov P.V., Chentsov V.B., Nistratov S.L., Kudryakov O.N., Soloviev V.V., Banova Zh.I., Shkuratova N.V., Rezepov N.A., Gelfand B.R. Optimum level of positive end- expiratory pressure in acute respiratory distress syndrome caused by influenza A (H1N1)pdm09: balance between maximal end-expiratory volume and minimal alveolar overdistension. *Anesteziol.Reanimatol/ Anesteziologiya i Reanimatologiya*. 2016; 61 (6): 425–432. (in Russ.). UDC: 616.24-008.64-06: 616.921.5]-073].
83. Ручина Е.В., Шарнин А.В., Лебединский К.М., Мазурок В.А. Оценка функциональной остаточной емкости легких и показателя потребления кислорода во время настройки уровня ПДКВ. *Анестезиология и реаниматология*. 2013; (3): 51–54. УДК 616.24-008.1-073.173. [Ruchina E.V., Sharnin A.V., Lebedinsky K.M., Mazurok V.A. Assessment of functional residual capacity and oxygen consumption during PEEP trial procedure. *Anesteziol.Reanimatol/ Anesteziologiya i Reanimatologiya*. 2013; (3): 51–54. (in Russ.). UDC 616.24-008.1-073.173].
84. Заболотских И.Б., Киров М.Ю., Лебединский К.М., Проценко Д.Н., Авдеев С.Н., Андреев А.А., Арсентьев Л.В., Афончиков В.С., Афуков И.И., Белкин А.А., Боева Е.А., Буланов А.Ю., Васильев Я.И., Власенко А.В., Горбачев В.И., Григорьев Е.В., Григорьев С.В., Грицан А.И., Еременко А.А., Ершов Е.Н., Замятин М.Н., Иванова Г.Е., Кузовлев А.Н., Куликов А.В., Лакхин Р.Е., Лейдерман И.Н., Ленкин А.И., Мазурок В.А., Мусаева Т.С., Николаенко Э.М., Орлов Ю.П., Петриков С.С., Ройтман Е.В., Роненсон А.М., Сметкин А.А., Соколов А.А., Степаненко С.М., Субботин В.В., Ушакова Н.Д., Хороненко В.Э., Царенко С.В., Шифман Е.М., Шукевич Д.Л., Щеголев А.В., Ярошецкий А.И., Ярустовский М.Б. Анестезиолого-реанимационное обеспечение пациентов с новой коронавирусной инфекцией COVID-19. Методические рекомендации Общероссийской общественной организации «Федерация анестезиологов и реаниматологов». *Вестник интенсивной терапии им. А.И. Салтанова*. 2022; 1: 5–140. DOI: 10.21320/1818-474X-2022-1-5-140. [Zabolotskikh I.B., Kirov M.Yu., Lebedinsky K.M., Protsenko D.N., Avdeev S.N., Andreenko A.A., Arsentiev L.V., Afonchikov V.S., Afukov I.I., Belkin A.A., Boeva E.A., Bulanov A.Yu., Vasiliev Ya.I., Vlasenko A.V., Gorbachev V.I., Grigoriev E.V., Grigoriev S.V., Gritsan A.I., Eremenko A.A., Ershov E.N., Zamyatin M.N., Ivanova G.E., Kuzovlev A.N., Kulikov A.V., Lakhin R.E., Leiderman I.N., Lenkin A.I., Mazurok V.A., Musaeva T.S., Nikolaenko E.M., Orlov Y.P., Petrikov S.S., Roitman E.V., Ronenson A.M., Smetkin A.A., Sokolov A.A., Stepanenko S.M., Subbotin V.V., Ushakova N.D., Khoronenko V.E., Tsarenko S.V., Shifman E.M., Shukevich D.L., Shchegolev A.V., Yaroshetsky A.I., Yarustovsky M.B. Anesthesia and intensive care for patients with COVID-19. Russian Federation of anesthesiologists and reanimatologists guideline. *Ann Crit Care /Vestnik intensivnoy terapii im AI Saltanova* 2022; 1: 5–140. (in Russ.). DOI: 10.21320/1818-474X-2022-1-5-140].
85. Ярошецкий А.И., Грицан А.И., Авдеев С.Н., Власенко А.В., Еременко А.А., Заболотских И.Б., Зильбер А.П., Киров М.Ю., Лебединский К.М., Лейдерман И.Н., Мазурок В.А., Николаенко Э.М., Проценко Д.Н., Солодов А.А. Диагностика и интенсивная терапия острого респираторного дистресс-синдрома (Клинические рекомендации Общероссийской общественной организации «Федерация анестезиологов и реаниматологов»). *Анестезиология и реаниматология*. 2020; (2): 5–39. DOI: 10.17116/anaesthesiology20200215. [Yaroshetsky A.I., Gritsan A.I., Avdeev S.N., Vlasenko A.V., Eremenko A.A., Zabolotskikh I.B., Zilber A.P., Kirov M.Yu., Lebedinsky K.M., Leiderman I.N., Mazurok V.A., Nikolaenko E.M., Protsenko D.N., Solodov A.A. Diagnostics and intensive therapy of acute respiratory distress syndrome (Clinical guidelines of the Federation of Anesthesiologists and Reanimatologists of Russia). *Anesteziol.Reanimatol/ Anesteziologiya i Reanimatologiya*. 2020; (2): 5–39. (in Russ.). DOI: 10.17116/anaesthesiology20200215].
86. Ибадов Р.А., Сабиров Д.М., Ибрагимов С.Х., Бурханов Б.Б., Ибадов Р.Р. Механика дыхания и газообмен при остром респираторном дистресс-синдроме, ассоциированном с COVID-19. *Общая реаниматология*. 2022; 18 (5): 24–31. DOI:10.15360/1813-9779-2022-5-24-31 [Ibadov R.A., Sabirov D.M., Ibragimov S.K., Burkhonov B.B., Ibadov R.R. Respiratory mechanics and gas exchange in acute respiratory distress syndrome associated with COVID-19. *General Reanimatology/ Obshchaya Reanimatologiya*. 2022; 18 (5): 24–31. (in Russ.). DOI:10.15360/1813-9779-2022-5-24-31]

Received 02.08.2022
Online First 23.11.2022