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COMPLICATIONS OF THE LUNG ARTIFICIAL VENTILATION APPARATUS

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ABSTRACT

Currently, the development of the industry, the growth of man-made disasters and armed conflicts have significantly increased the number of victims with severe trauma, which is one of the main causes of disability and death of people of working age. Artificial lung ventilation (ventilator) is an integral part of the intensive care complex for all patients in critical condition, and in most cases it is not possible to avoid prosthetics of the respiratory function for victims with severe trauma.

Keywords: prolonged tracheal intubation; complications; fibrotracheobronchoscopy stenosis.

INTRODUCTION

As a result of experimental studies, it has been proven that inadequately selected ventilation parameters lead to the development of ventilator-induced lung damage, while its following manifestations are distinguished: barotrauma, volumotrauma, atelectatic trauma and biotrauma (Dreyfuss D. et al, 1998, Tremblay L. et al, 1997). Overgrowth of the alveoli and cyclic opening and closing of the collapsed alveoli are the main trigger mechanism for the development of biotrauma, which activates macrophages that produce the main proinflammatory cytokines (TNF-a, IL-1, IL-6, IL-10). Inflammatory mediators, in turn, damage lung tissue, leading to the development of nonspecific inflammation.

Both in the literature of the last century and in modern guidelines for ventilation of intact lungs, the authors recommend using sufficiently large respiratory volumes of 10-12 ml per kilogram of body weight, and there are no clear recommendations for the selection of positive end-of-exhalation pressure (MPC).

As a result of clinical studies, it has been proven that the use of the concept of "protective" ventilation, which includes: limiting peak inspiratory pressure to 35 cm of water, using small respiratory volumes (6 ml/kg), achieving optimal plateau pressure (no more than 30 cm of water), increasing the functional respiratory surface of the lungs by opening alveoli, due to the use of positive end-of-exhalation pressure, leads to a decrease in mortality in patients with OSHI/ARDS. However, there are still unresolved issues: "Does ventilation damage intact lungs and does the percentage of complications associated with ventilation decrease when using the concept of protective ventilation?". Clinical studies and observations in this area are extremely few, and their results are uninformative. At the same time, there are studies

demonstrating that artificial ventilation using large respiratory volumes (10 ml/kg BMI) is an independent predictor of the development of acute lung injury. These questions served as the motivation for this study and determined its purpose and objectives.

THE PURPOSE OF THE STUDY

To study the effect of various modes of artificial ventilation on the development of lung damage and to assess the associated complications in patients with severe trauma.

RESEARCH MATERIALS AND METHODS

There were 78 seriously injured patients under observation, who underwent ventilation for more than 48 hours.The age of the examined patients ranged from 18 to 70 years (33.6 ± 12.1), while the majority of patients (n=72) were of working age (up to 50 years), mainly men (n=61). The victims were classified as severe and extremely severe patients (the average assessment of the severity of the condition upon admission on the APACHE II II scale was 18.3 ± 3.4 points).

The selection of patients was carried out according to the inclusion criteria:

- victims with severe traumatic brain injury, multiple and severe combined trauma of any gender,
- age from 18 to 70 years,
- the need for respiratory support for extrapulmonaryindications,
- The estimated duration of respiratory support is at least 48 hours.

THE RESULT OF THE STUDY

In order to determine the clinical significance of biophysical trauma, that is, mechanical factors of lung damage, we studied the change in the biomechanics of respiration depending on the use of a particular ventilator regimen.

In the I group of victims, the average dose during the entire period of controlled ventilation varied from 703.6 ± 75 to 798.57±74 ml, in the II group, the average DOSE ranged from 439.5±53 to 450±45 ml.

During the entire period of controlled ventilation, the peak pressure (PIP) in group I of patients exceeded its maximum permissible values set out in the concept of safe ventilation in patients with OPL/ARDS - 35 cm H20, but did not exceed 50 cm H20. In the II group of victims, the maximum PIP did not exceed 26 cm NG. 4 of the 39 victims in group I developed pneumothorax in the period from 5 to 10 days (right-sided in two cases, left-sided in one case and bilateral in another). This complication was not associated with invasive manipulations and was regarded by us as a barotrauma.

When assessing the dynamics of changes in plateau pressure, it was revealed that during the entire period of controlled ventilation in the 1st group of victims, the plateau pressure was significantly higher compared to the second group. The period of maximum increase in alveolar pressure in the I group of patients corresponded to the period of maximum increase in PIP, that is, from 3 to 6 days (27,4±3,4; 27,6±3,95; 27,8±4,2 and 27.3±3.1 cm H20). However, this indicator in the "traditional" ventilation group did not exceed the maximum permissible values

of 30 cm H20. In the II group of affected patients, Pplateau did not exceed 18 cm H2O during the entire period of controlled ventilation.

When comparing the severity of OPL on the LIS scale, it was revealed that in the period from day 2 to day 15, patients with the "traditional" IVL regimen developed significantly more significant lung damage. However, the average score in this group of patients corresponded to moderate lung damage, that is, it did not exceed 2.5 points. In the II group of patients, the average assessment of lung damage did not exceed 0.5 points. The risk of developing OPL when using the "traditional" ventilator regimen was 4.37 (2,337 - 8,189. 95% CI (p = 0.0001)), however, the risk of ARDS in patients with initially intact lungs is significantly lower - OR 0.89 (0.984 - 11.466. 95% Ci (p = 0.04)).

In order to determine the role of biotrauma in the development of ventilator-associated lung damage, we quantified the concentration of inflammatory mediators (TNFa, ILip, IL6 and IL4) in bronchoalveolar lavage fluid (BALf) in patients of both groups in the first three days and then a day later for 7-1 1 days from the moment of onset

The results showed that the concentration of proinflammatory mediators (TNFa, ILip and IL6) in the BALJ group with a "traditional" ventilation regimen during the entire period of respiratory support was significantly higher compared to group II (Table 3). The concentration of the anti-inflammatory mediator IL4 in both groups varied slightly and these differences were not significant.

When assessing the frequency and severity of pneumonia on the DOP scale, it was found that both groups of NSAIDs began to develop as early as 3 days from the beginning of respiratory support (7.69% and 2.56% of the total number of victims in groups I and II, respectively (p = 0.078)). On day 5, the frequency

NSaid in group I reached 84%, while in group II it reached only 23% (p=0.0001). In order to assess the severity of pneumonia depending on the use of different ventilation modes, we analyzed these indicators using the cross-tabulation method, and the reliability of the frequency difference was determined using the chi-square criterion (Table 5). The majority of the victims of both groups had moderate pneumonia.

The maximum number of cases of severe pneumonia was noted on day 9 in group I and on day 11 in group II, however, in group I patients severe pneumonia developed significantly more often. Extremely severe NSAIDs in the group with the "traditional" ventilation regime developed in 2 patients, there were no cases of extremely severe pneumonia in group II. The duration of stay of the survivors in the ICU was 21.9 ± 5.6 and 15.75 ± 2.9 days in groups I and II, respectively (p = 0.0002). We have identified risk factors for the duration of stay of victims in the ICU for more than 14 days. The relative risk of an increase in the duration of treatment in the ICU during ventilation in the "traditional" mode was 2.0 (0.18 - 23.6) 95% CI, with impaired consciousness < 6 points on the Glasgow scale on day 6 -14.2 (1.8 - 113.9) 95% CI, if the patient has NSAID on day 7 - 2.7 (0.9 - 6,7). The presence of OPL in victims is not a risk of increasing the length of stay in the ICU. The total mortality rate of the victims on the 28th day was 19.2%. Mortality on day 28 in the affected groups was 20.5% (n=8) and 17.9% (n=7) in groups I and II, respectively, there were no significant differences (p = 0.5).

CONCLUSIONS

Long-term artificial lung ventilation (more than 48 hours) using respiratory volumes of 10-12 ml/kg of ideal body weight and a positive end-of-exhalation pressure of 5 cm H20 in patients with severe trauma and intact lungs leads to a more frequent development of lung damage compared with artificial ventilation in the "protective" mode (35 out of 39 and 12 out of 39 cases in groups I and II, respectively ($p = 0.0001$), OR 4.375 (2.337-8.189) 95% LI).

Lung damage in victims with the "traditional" mode of artificial ventilation • is more severe, as evidenced by a high score on the LIS scale (1.43 and 0.5 points in groups I and II, respectively ($p < 0.0001$)).

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