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Nemaa Mazyed Abdulazeez
Anesthetist, Ministry of Health - Medical City - Baghdad Teaching Hospital, Baghdad, Iraq

Abdul Rasool Ibadi Abdulhasan Al-Krizi
Anesthetist, Ministry of Health - Baghdad Medical Office - Al-Karkh, Al Karkh General Hospital, Baghdad, Iraq

Afraa Qasim Kadhim
Anesthetist, Chairman of Anesthesia Department In Pediatric Surgery Department Ministry of Health - Medical City - Children Welfare Teaching Hospital, Baghdad, Iraq

Corresponding Author:
Nemaa Mazyed Abdulazeez
Anesthetist, Ministry of Health - Medical City - Baghdad Teaching Hospital, Baghdad, Iraq

General anesthesia with preserved spontaneous breathing through an intubation tube

Nemaa Mazyed Abdulazeez, Abdul Rasool Ibadi Abdulhasan Al-Krizi and Afraa Qasim Kadhim

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Abstract
The aim of the study was to study the possibility of preserving spontaneous breathing in patients during planned surgical interventions under general anesthesia with tracheal intubation.
Material and Methods: 112 patients who underwent planned surgical interventions under general endotracheal anesthesia were randomly divided into two groups. Patients of the first group underwent compulsory mechanical ventilation in the volume control mode, patients of the second group underwent assisted ventilation of the lungs in spontaneous breathing mode with apparatus support.
Conclusion: According to the results of the study, the possibility of safe use of the spontaneous breathing mode with hardware support during some surgical interventions in patients with initially healthy lungs has been shown.

Keywords: Pressure support, assisted ventilation, spontaneous breathing, general anesthesia, lung function

Introduction
Artificial lung ventilation (ALV) at one time significantly expanded the possibilities of surgery in performing operations that were previously considered impossible. Classic tetrad: amnesia, analgesia, neuro-autonomic defense and total myoplegia with mechanical ventilation, for a long time they became those "whales" on which general anesthesia rested. However, perhaps now is the time to remember that there are only two reasons for performing mechanical ventilation during anesthesia in a patient with initially healthy lungs:
1) the requirements of the surgical technique;
2) respiratory depression caused by most anesthetic drugs.
However, total myoplegia, if necessary for surgeons, is only at certain stages of the operation; and perhaps the respiratory depression caused by modern anesthetics is somewhat exaggerated?
This is especially true if we take into account the fact that in addition to its undeniable advantages (especially in resuscitation), mechanical ventilation has a number of disadvantages.

Effect of mechanical ventilation on lung function
The understanding of the duality of the effects of mechanical ventilation became apparent for a long time. Thus, in 1974, Webb and Turney [1] showed a relationship between increased airway pressure and infrastructural damage to the lungs. Their observations were ignored for about 10 years, until other researchers confirmed and expanded their work [2]. Electron microscopy in animals showed changes in the pulmonary epithelium, capillary endothelium, alveolar hemorrhage, the appearance of hyaline membranes [3].
Spontaneous breathing involves active movements of the chest to create a negative pressure gradient that allows gas to move. In conditions of mechanical ventilation, the gas-narcotic (or gas) mixture is insufflated into the lungs under positive pressure.
The primary effect of mechanical ventilation on lung function depends primarily on the initial state of the lungs. The positive effect of mechanical ventilation is manifested, first of all, in case of lung pathology with a serious violation of the ventilation-perfusion ratio. In such cases, mechanical ventilation increases the number of functioning alveoli, contributing to the partial expansion of atelectasis, an increase in the gas exchange area and an

improvement in oxygenation. With relatively healthy lungs, mechanical ventilation prosthetics the function of external respiration, and nothing more. However, in the process of prolonged ventilation, regardless of the initial state of the lung tissue, the negative effect of mechanical ventilation on lung condition.

According to different authors, respiratory complications develop in 3-4% of patients after elective endotracheal anesthesia and in 20% of patients in emergency anesthesiology [3, 4].

Harmful factors of forced mechanical ventilation [5-8]:

1) turbulent air flows in the bronchi cause an uneven distribution of the respiratory mixture over the pulmonary fields;
2) altered regional relationships between alveolar, arteriocapillary and venous pressure increase the amount of extravascular water in the pulmonary interstitium, hindering the outflow of lymph;
3) ventilation in large volumes washes out the surfactant, leading to atelectasis and hypoxemia, despite an increase in the oxygen fraction;
4) high peak inspiratory pressure leads to barotrauma of the most compliant alveoli, ruptures, hemorrhages, release of inflammatory mediators, aggravating oxygenation disorders.

It is possible to reduce these undesirable effects by applying special modes, conditions and ventilation parameters, but it is unrealistic to prevent completely, at least for now.

Effect of mechanical ventilation on the respiratory muscles

Mechanical ventilation is associated with a large number of complications, including pneumonia, cardiovascular disorders, barotrauma, and ventilator-associated lung injury (VILI). Recent animal studies have shown that CMV can be the cause of diaphragm dysfunction [9]. Decreased diaphragm contractility, decreased FEV is called ventilator-associated diaphragm injury.

we (VIDD). The decrease in the contractility of the diaphragm is inversely proportional to the duration of mechanical ventilation [10].

Atrophy, remodeling of diaphragm fibers, oxidative stress, and structural damage may be potential mechanisms of VIDD that make it difficult to wean from the respirator [11]. Accordingly, in the absence of other proven reasons, it is wiser to avoid prolonged mechanical ventilation.

Two days of mechanical ventilation reduces the contractility of the diaphragm by 42% with unchanged DO, intra-abdominal pressure, unchanged function of neuromuscular transmission. The initial phenomena of atrophy are noticeable after 12 hours of mechanical ventilation in CMV mode (continuous mechanical ventilation) and are more pronounced in the diaphragm than in skeletal muscles. PEEP increases the rate of atrophy. The reasons are an increase in the breakdown of proteins with a decrease in their synthesis. Thus, 6 hours of mechanical ventilation leads to a 30% decrease in the level of respiratory muscle protein synthesis, a 65% decrease in myosin synthesis. A 5-fold increase in protein breakdown (by 470%) was observed in animals after 18 hours of CMV [10-12]. It is worth recalling that CMV is the main regimen used in anesthesiology.

Oxidative stress is a combination of protein and lipid peroxidation. Oxidized proteins become more susceptible to proteolytic enzymes, which, as a result of proteolysis, leads to a deterioration of the excitation-contraction cycle and to a decrease in the force of contractions of the diaphragm. Mostly insoluble proteins with a molecular weight of 40 KD (actin) to 200 KD (myosin) undergo oxidation and proteolysis [11].

Structural intracellular changes in the diaphragm of animals are noted after 4-8 hours of forced mechanical ventilation and consist in:

- rupture of myofibrils;
- swelling of mitochondria;
- the appearance of drops of fat in the cytoplasm;
- vacuolization of myofibrils.

The authors found the same changes in the anterior intercostal muscles of animals, while in the gastrocnemius muscles of the hind limbs, no such violations were found [13].

According to the literature data obtained on the rabbit model, most cases of VADD are associated with mechanical ventilation in the CMV mode, while in the Assist Control mode, which allows spontaneous breathing, the dysfunction phenomena are less pronounced (decrease in diaphragm contraction after 3 days by 48% in the CMV group versus 14% in the A / C group) [14-16].

The impairment of gas exchange in the lungs can be the result of parenchymal damage to the lungs, induced by hypoxemia and respiratory muscle fatigue, which is characterized by hypercapnia. Sepsis, heart failure, and malnutrition can induce depletion of the respiratory muscles.

Contractility and coordinated contraction of the diaphragm and intercostal muscles determines the effectiveness of spontaneous breathing.

Spontaneous breathing improves perfusion, metabolism, contractility of the respiratory muscles, and improves hemodynamics [17-19]. An important advantage of diabetes mellitus is a decrease in the mean airway pressure in comparison with MVL. PSV reduces the patient's inhalation effort, reduces oxygen demand, prevents diaphragm fatigue, reduces intrapulmonary shunt and improves extubation rates after prolonged mechanical ventilation. Minimal alveolar ventilation is not guaranteed in PSV mode, therefore careful observation of patients is imperative. APRV, BiPAP modes provide ventilation even when the patient's diabetes mellitus stops [20-28].

According to some authors, even periodic episodes of spontaneous breathing during MVL can reduce the harmful effects of mechanical ventilation on the respiratory muscles [14].

Effect of mechanical ventilation on the development of atelectasis

It has long been known that pulmonary shunt enlarges during anesthesia, but until recent years its causes were not fully understood. Benedixen in his work (1963) proposed the “concept of atelectasis”, which explained the decrease in PO2 during general anesthesia. Later, other authors found a decrease in PO2 during induction into anesthesia. After the advent of CT, Brisler in the 80s demonstrated the rapid development of lung tissue compaction in certain areas under general anesthesia (OA) [29].

Gas exchange in the lungs regularly decreases during general anesthesia with mechanical ventilation. This can lead to a decrease in blood oxygenation. In most cases, these

disorders are caused by the formation of atelectasis, which are not detectable by X-ray, but can be detected by computed tomography (CT). Atelectases increase the intrapulmonary shunt, can activate alveolar macrophages that release interleukin 1 and TNF, which reduces the function of the surfactant and leads to lung damage. As has been shown [30], in adults with healthy lungs, 20–25% of the lung tissue in the basal regions or 15% of the total lung tissue can be atelectasized during mechanical ventilation, which gives about 15% of the shunt. It is now recognized that atelectasis to varying degrees develops in 90% of patients, regardless of the use of muscle relaxants and the type of anesthesia.

It is assumed that the use of nitrous oxide accelerates the development of atelectasis due to the rapid absorption of N2O in poorly ventilated areas. A significant correlation was found between the localization and size of the zones of the intrapulmonary shunt and the localization and size of atelectasis (r = 0.81) [32].

In addition to atelectasis of the lung tissue, early expiratory airway closure (EAPC) plays a role in ventilation and oxygenation disorders during mechanical ventilation, further reducing effective ventilation. In the zones of EZDP develops a violation of ventilation-perfusion relations.

With age, the number of lung regions with a decreased ventilation-perfusion ratio increases [33]. In one of the works, it was proved that the degree of EZDP and the amount and severity of atelectasis are directly proportional to the patient's body weight and age [34].

Collapse of the alveoli and EZDP can contribute to impaired ventilation and the development of infection. The cause of atelectasis and EZDP can be impaired muscle tone, high oxygen fractions during induction and maintenance of anesthesia [34].

According to the literature, an increase in inspiration time and an inversion of the inspiration-expiration ratio can open non-ventilated areas of the lungs [35]. They can also be straightened using the recruitment maneuver [31, 33]. However, these techniques are laborious, have recently begun to be used in resuscitation and are little known to anesthesiologists, in addition, not all anesthesia and respiratory devices (NDA) are suitable for carrying out these measures.

In the literature, we found only four articles published in different years, the authors of which allowed the solution of the arisen contradiction in the use of auxiliary ventilation modes in the process of anesthesia [25, 36, 37].

Returning to the beginning of this article, we are forced to ask again the question, is it really necessary to have total myoplegia and mechanical ventilation during general anesthesia? And is it possible, at least part of the intraoperative period, to be carried out in conditions of preserved spontaneous breathing?

To answer this question, this study was undertaken.

The aim of the study was to study the possibility of preserving spontaneous breathing in patients under the conditions of elective surgery under general anesthesia with tracheal intubation.

Materials and Methods

Intraoperative protection was carried out by the IPA method (inhalation induction and maintenance of anesthesia) based on sevoflurane. A total of 112 patients were examined (mean age 43 ± 12 years; 49 men and 63 women). No initial pathology of the lungs was revealed in patients in both groups. ASA functional status from I to III class.

The study was carried out with the following types of surgical interventions: discectomies for herniated intervertebral discs at the lumbar level, resection of tumors of the spines, thyroidectomies, mastectomies with simultaneous TRAM plasty, gastric resections, laparoscopic hernioplasty (Fig. 1).

The distribution of surgical interventions into groups was carried out in a random order. The duration of the operation in the 1st group was 131 ± 26 minutes, in the 2nd group - 143 ± 37 minutes.

Respiratory support was provided by Frontline Sirius and Datex Ohmeda anesthesia devices. The patients were divided into two groups depending on the ventilation option (mechanical ventilation or IVIV).

In the 1st group of patients (51 people), during general anesthesia, compulsory mechanical ventilation was performed with volume control, in the norm ventilation mode.

In group 2 (61 people), during general anesthesia, auxiliary mechanical ventilation was performed in the Pressure Support mode. After induction anesthesia and tracheal intubation, muscle relaxants were not used, a gradual decrease in the respiratory rate achieved the onset of spontaneous respiratory activity of patients, then the ventilator was switched to Pressure Support mode, with a maximum trigger sensitivity of 2 L / min, Psupp varied from 10 to 16 mm aq. Art., depending on the tidal volume. Some of the patients were able to administer anesthesia with completely spontaneous breathing.

Doses of drugs used to maintain anesthesia are shown in table. one.

The peculiarity of the use of fentanyl in the 2nd group was the limitation of its bolus at a dose of no more than 50 μg per administration, due to the danger of inhibition of spontaneous respiration at a larger single dose, or a constant infusion of fentanyl was used at an average rate of 2.1 ± 0.4 μg / kg • hour, supplemented, if necessary, with a bolus at a dose of not more than 50 μg per administration.

The studied parameters: respiratory rate, tidal volume, tracheal extubation time, mean airway pressure (Pmean), end-expiratory carbon dioxide (EtCO2), capillary oxygen saturation, difference

oxygen content in the respiratory mixture FiO2-FeO2, respirator index RI. For convenience of comparison, the respiratory index FiO2 at the stage of maintaining anesthesia in both groups was set equal to 0.5.

BIS monitoring was carried out according to the generally accepted technique. After the end of the operation (since the imposition of the last suture to the surgical wound) in both groups, the sevoflurane delivery was stopped and the time of awakening and extubation was counted. Tracheal extubation was performed after the restoration of adequate spontaneous breathing, pharyngeal and laryngeal reflexes, the ability of patients to perform elementary commands (clench the hand, open the eyes, raise the head).

Conclusion

According to the results of the study, the possibility of safe use of the spontaneous breathing mode with hardware support in some surgical interventions, at least in some patients with initially healthy lungs and in some types of planned surgical interventions, has been shown.

Of course, this is the first, albeit promising, study in this direction. It leaves more questions than it offers solutions. So, it remains unstudied how universal the proposed method is? Is it possible to repeat the technique for other types of operations and longer duration? Elderly patients and children? With the initial pathology of the lungs? Etc. And finally, we have to answer the main question: how clinically significant is the proposed solution? Is it possible to neutralize the negative ventilation effects?

Only with a positive answer to this question does it make sense to introduce the proposed method, because otherwise, it only creates additional problems for the anesthesiologist (constant monitoring of breathing).

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