ФЕДЕРАЛЬНОЕ ГОСУДАРСТВЕННОЕ АВТОНОМНОЕ ОБРАЗОВАТЕЛЬНОЕ УЧРЕЖДЕНИЕ
ВЫСШЕГО ОБРАЗОВАНИЯ «КРЫМСКИЙ ФЕДЕРАЛЬНЫЙ УНИВЕРСИТЕТ ИМЕНИ В.И. ВЕРНАДСКОГО»
МЕДИЦИНСКАЯ АКАДЕМИЯ ИМЕНИ С.И. ГЕОРГИЕВСКОГО

ТАВРИЧЕСКИЙ МЕДИКО–БИОЛОГИЧЕСКИЙ ВЕСТНИК

TAVRICHESKIY MEDIKO-BIOLOGICHESKIY VESTNIK

Том 19 № 3 Volume 19
2016

«Таврический медико-биологический вестник» – рецензируемый научно-практический журнал. Основан в 1998 году Ученым советом Крымского государственного медицинского университета имени С.И.Георгиевского.

Журнал зарегистрирован Федеральной службой по надзору в сфере связи, информационных технологий и массовых коммуникаций Российской Федерации. Свидетельство ПИ № ФС77-61811 от 18 мая 2015 года.

«Таврический медико-биологический вестник» включен в Перечень рецензируемых научных изданий, в которых должны быть опубликованы основные научные результаты диссертаций на соискание ученой степени кандидата наук, на соискание ученой степени доктора наук ВАК РФ 7.12.2015 года по группам специальностей 03.02.00 – общая биология; 14.01.00 – клиническая медицина; 14.02.00 – профилактическая медицина.

Статьи проходят рецензирование в соответствии с требованиями к рецензируемым научным журналам. Статьи, опубликованные в журнале, индексируются в базах РИНЦ (e_Library.ru) и Google Scholar.
Подписной индекс издания в каталоге Агентства «Роспечать» – 80121
Территория распространения: Российская Федерация, страны СНГ. Периодичность выхода – 4 номера в год.

Рекомендован к печати Ученым советом Медицинской академии имени С.И.Георгиевского
(протокол № 12 от 27 октября 2016 г.)

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УЧРЕДИТЕЛЬ
ФГАОУ ВО«КРЫМСКИЙ ФЕДЕРАЛЬНЫЙ УНИВЕРСИТЕТ ИМЕНИ В. И. ВЕРНАДСКОГО»,
МЕДИЦИНСКАЯ АКАДЕМИЯ ИМЕНИ С. И. ГЕОРГИЕВСКОГО»

Журнал основан в 1998 году.
Издается 4 раза в год.

ВОПРОСЫ ПРЕПОДАВАНИЯ
THE TEACHING

ОРГАНИЗАЦИИ ЗДРАВООХРАНЕНИЯ
THEALTH ORGANIZATION

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UDC 616.24-008.4

CURRENT UNDERSTANDING OF ACUTE RESPIRATORY FAILURE

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SUMMARY

Being one of the most common types of organ insufficiency, acute respiratory failure (ARF) is frequently associated with increased death rate and represents a medical emergency. An ability to promptly and adequately manage respiratory failure to prevent further life-threatening deterioration is essential for every healthcare professional. This article is created based on comprehensive and authoritative medical sources to present relevant up-to-date knowledge on the subject of ARF. It will provide an overview of main pathophysiologic considerations, basic principles of diagnosis and management of ARF. The article will be beneficial for medical students and medical professionals who are interested in improvement and refreshment of their knowledge in the area of clinical respiratory medicine.

Key words: acute respiratory failure; lecture; hypoxemia; hypercapnia; ABG analysis; airways; oxygenation; mechanical ventilation.

СОВРЕМЕННЫЕ ПРЕДСТАВЛЕНИЯ ОБ ОСТРОЙ ДЫХАТЕЛЬНОЙ НЕДОСТАТОЧНОСТИ

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РЕЗЮМЕ

Будучи одной из наиболее распространенных форм органной недостаточности, острая дыхательная недостаточность (ОДН) часто характеризуется повышенными показателями летальности и является неотложным состоянием. Навыки своевременного и эффективного вмешательства при дыхательной недостаточности, направленного на предупреждение дальнейшего развития угрожающих жизни осложнений, являются обязательными для врача любой специальности. Созданная на основе фундаментальных и авторитетных источников медицинской литературы, данная статья рассматривает наиболее важные актуальные знания по теме ОДН. В ней представлен обзор основных патофизиологических аспектов, базовых принципов диагностики и лечения ОДН. Материал предназначен для студентов медицинских ВУЗов и врачей, желающих улучшить и обновить свои знания в области клинической респираторной медицины.

Ключевые слова: острая дыхательная недостаточность; лекция; гипоксемия; гиперкапния; анализ газового состава крови; дыхательные пути; оксигенация; механическая вентиляция легких.

INTRODUCTION

Acute respiratory failure (ARF) is a syndrome characterized by rapid onset of inability of the respiratory system to support adequate gas exchange.

Patients with ARF constitute a high percentage of intensive care unit (ICU) admissions [1], and ARF has been associated with mortality rates up to 40% [2]. In a study carried out to investigate the trends for ICU hospitalization due to acute respiratory insufficiency it was determined that most common diseases causing ARF are chronic obstructive pulmonary disease (COPD), pneumonia, chronic heart failure and acute respiratory distress syndrome, with pneumonia taking highest precedence [3]. It was also revealed that in US over a period of 9 years the number of hospitalized patients with the diagnosis of ARF rose from 1,007,549 to 1,917,910, showing a steady increase in the incidence of this syndrome. Nearly 2 million hospitalizations in 2009 resulted in approximately 380,000 deaths and inpatient costs of over $54 billion [3].

Due to the improvement in the techniques and principles of respiratory support and adequate management of ARF over the years, hospital mortality has been reduced from 27.6% in 2001 to 20.6% in 2009 with overall improvement in survival rate [3]. Nevertheless, owing to its multi-etiological nature and high incidence, ARF has remained one of the foremost killers over the past decade [4], making the knowledge of prompt diagnosis and effective management of the syndrome very important.

Basic Definitions and Pathophysiologic Considerations

The main function of the respiratory system is to perform continuous oxygenation of blood and

elimination of the volatile waste product, carbon dioxide (CO_2), into the atmosphere.

ARF is a syndrome characterized by inability of the respiratory system to support adequate gas exchange. The term "acute" denotes rapid onset of respiratory insufficiency (within hours or several days) [5].

There are two main types of ARF:

Hypoxemic ARF (type I) represents oxygenation failure – inability to provide sufficient oxygen (O_2) to the blood (namely hypoxemia) and, in consequence, to the organs (hypoxia). Hypoxemic ARF is diagnosed when arterial blood gas (ABG) analysis shows that a partial pressure of O_2 in arterial blood (PaO_2) is below 60 mm Hg while breathing ambient air [6]. This type of ARF is also known as hypoxemia without hypercapnia.

Hypercapnic ARF (type II) result from inadequate ventilation (alveolar hypoventilation). This type of ARF is diagnosed when a partial pressure of CO_2 in arterial blood ($PaCO_2$) is greater than 45 mm Hg [5, 6].

Normal function of the respiratory system relies upon integrity and sequential action of the main structural components [5]:

Central nervous system (CNS) component (respiratory center, chemoreceptors, CNS fibers) – responsible for continuous support and regulation of respiratory drive.

Chest bellows component (peripheral nervous fibers, respiratory muscles [diaphragm and intercostal muscles], chest wall) – functions as a respiratory pump to produce pressure gradient between atmosphere and alveoli and move air into and out of the lungs.

Airway component (oropharynx, nasopharynx, larynx, trachea, bronchi, bronchioles) – provides the passage for air flow to the respiratory compartment of the lungs.

Alveolar component – gives O_2 to the pulmonary circulation and receives CO_2 from blood by means of passive diffusion through the alveolar-capillary barrier.

ARF with impaired CNS component usually results from drug-induced depression of CNS respiratory drive (e.g. opioid and sedative drug toxicity) and structural CNS affection (e.g. brainstem lesions) [5]. Decreased respiratory rate and tidal volume (volume of one breath; V_T) leads to alveolar hypoventilation, rise in $PaCO_2$ and development of acute respiratory acidosis [7]. In addition, accumulation of CO_2 displaces O_2 from alveolar spaces, leading to hypoxemia [6].

Chest bellows component frequently fail as a result of decreased respiratory muscle strength (e.g. myasthenia gravis, Guillain-Barré syndrome, injury to the cervical spinal cord [C3-5], muscle-relaxing drugs) or mechanical restriction of lung expansion (e.g. thoracic injuries, abdominal compartment syndrome) [5]. Despite adequate central neural drive, patients have limited ventilatory capacity and exhibit rapid shallow breathing (low V_T with increased RR), increased $PaCO_2$, decreased blood pH and PaO_2.

Impairment of the airway component (e.g. COPD exacerbation, asthmatic attack) is characterized by limited expiratory flow, air trapping with increase in dead space, progressive fatigue of the respiratory muscles, and restricted minute ventilation [5]. The consequence is alveolar hypoventilation, CO_2 retention and arterial hypercapnia associated with reduced PaO_2.

ARF with affected alveolar component typically results from diffuse alveolar flooding (e.g. edema fluid, blood, exudates) or atelectasis that decreases alveolar ventilation and prevents O_2 from reaching alveolar-capillary barrier, thus causing significant reduction in PaO_2 level [5]. Common causes include pneumonia, cardiogenic pulmonary edema, acute respiratory distress syndrome, lobar collapse [8]. This hypoxemia is usually resistant to oxygen therapy because of increased right-to-left (intrapulmonary) shunt (when blood from the right heart does not come into sufficient contact with oxygenated air before reaching the left heart) [7, 8]. Patients with a right-to-left shunt may have an abnormally low $PaCO_2$ because of hyperventilation. However, later on, a hypercapnic state develops as a consequence of progressive respiratory muscle fatigue [5].

In summary, impairment of the first three structural components of the respiratory system (CNS, chest bellows and airway) causes acute hypercapnia as a result of alveolar hypoventilation. Therefore, treatment of patients with hypercapnic ARF should be based primarily on improvement of alveolar ventilation, while concurrent hypoxemia usually effectively responds to O_2 therapy. Alternatively, impairment of the alveolar component, which is typically caused by airspace flooding, leads to increased intrapulmonary shunt and causes hypoxemia that is refractory to O_2 therapy despite hyperventilation and reduced $PaCO_2$.

DIAGNOSIS

All patients with severe ARF should be considered as critically ill, and promptly receive simultaneous evaluation and therapy to prevent further potentially life-threatening complications. First of all, quickly check for respiratory effort. If inadequate, immediately proceed with airway opening and assist ventilation (see "Management").

Clinical Presentations. While clinical criteria are unable to accurately predict PaO_2 and $PaCO_2$ levels, and formal diagnosis of ARF should be based on ABG analysis, various clinical signs and symptoms may lead to a suspicion of hypoxemia, hypercapnia or both [8].

Hypoxemia usually presents with early clinical findings of tachypnea, tachycardia, increased blood pressure, and cyanosis [7]. Progressive hypoxia result in CNS manifestations (e.g. agitation, somnolence, seizures, coma), central depression of respiratory drive (occurs when PaO_2 falls below 20 mm Hg)

and permanent anoxic brain damage [9]. Clinical presentations of hypoxemic respiratory failure are frequently amplified in patients with circulatory dysfunction (e.g. shock) and decreased oxygen-carrying capacity of blood (e.g. anemia, carbon monoxide poisoning).

Whereas high CO_2 levels can increase sympathetic nervous system activity with development of tachycardia and rise in blood pressure, acute hypercapnia typically presents with predominant affection of CNS function. Acting as a potent vasodilator for cerebral vessels, retained CO_2 increases intracranial pressure, thus causing headache, dizziness and confusion [9]. Further, CO_2 rapidly diffuses into the cerebrospinal fluid and depresses CNS activity through an acute fall of pH [6]. Severe hypercapnia can result in seizures, hallucinations, lethargy and coma. Hypercapnic patients may present with either hyperpnea or hypopnea, depending on the primary cause and severity of the respiratory dysfunction.

Arterial Blood Gas Analysis. ABG analysis allows the direct measurement of PaO_2, $PaCO_2$, pH, O_2 saturation of hemoglobin and other valuable parameters in samples of arterial blood. Normal range for PaO_2 is 80 to 100 mm Hg (can decrease with age and in supine position) [8]. Normal $PaCO_2$ is 40 mm Hg. $PaCO_2$ is inversely proportional to the alveolar ventilation, directly proportional to the body's CO_2 production, and unaffected by age and position. CO_2 production increases with fever and excessive nutritional carbohydrate consumption, however increased CO_2 production usually does not contribute to hypercapnia without concurrent impairment of CO_2 elimination [8]. Normal pH of human arterial blood is at or close to 7.40. Analysis of the relationship between pH, $PaCO_2$ and bicarbonate concentration helps to differentiate between respiratory and metabolic acidosis.

Pulse Oximetry. The pulse oximeter device noninvasively measures the percentage of hemoglobin in the saturated state (also known as oxygen saturation of blood) using a probe attached to a finger or an earlobe. Decreased pulse oximetry saturation (SpO_2) may predict significant hypoxemia, but acceptable SpO_2 readings (>90%) do not always exclude hypoxemic state. For example, oxygen saturation may be overestimated in cases when hemoglobin is unable to bind to oxygen (e.g. carboxyhemoglobin formation in carbon monoxide poisoning). The inaccuracies of SpO_2 readings may be caused by poor peripheral perfusion, motion artifacts, dark skin pigmentation or nail polish [10].

Imaging. While chest X-ray is still acceptable for preliminary examination in case of clinically suspected respiratory failure, computed tomography (CT) scanning allows a complete investigation of the lung parenchyma, including those lung regions not explorable with traditional anterior-posterior chest X-rays (e.g. anterior pneumothorax, posterior consolidation, atelectasis and effusions) [11].

MANAGEMENT

Whereas treatment of the underlying condition may significantly vary depending on the specific nature of the pathologic process, the general principles of supportive care are similar regardless of the type of respiratory system disorder, and include airways, oxygenation and ventilation.

Airways Opening and Protection. Initial management of any patient with suspected serious abnormalities of vital functions, including ARF, should start from assurance of adequate airway protection from any obstruction (soft tissues, edema, foreign matter, secretions) and aspiration. Signs of airway obstruction may include coughing, audible wheezing, stridor, or absence of any audible breath sounds, if obstruction is complete. Loss of muscular tone in patients with depressed mental status frequently causes the tongue, epiglottis and pharyngeal soft tissues to occlude the upper airways [12]. The basic maneuvers to relieve this obstruction include head tilt with chin lift, and jaw thrust. It is important to remember that the head extension is contraindicated in patients with suspected cervical spine injury.

Appropriately sized oropharyngeal and nasopharyngeal airways may also be highly effective in keeping the tongue and soft tissues from obstructing the posterior pharynx. However, in the semiconscious patient, insertion of an oropharyngeal airway can induce vomiting with possible aspiration due to low esophageal sphincter tone and depressed protective laryngeal airway reflexes. Nasopharyngeal airway is more appropriate, since it rarely induces gagging.

Whereas the aforementioned measures are helpful in providing temporary support to keep airways open, endotracheal intubation by means of direct laryngoscopy constitutes the method of choice for reliable and continuous airway protection from both obstruction and aspiration.

Alternative airway opening methods include placement of laryngeal mask airway, Combitube (also known as an esophageal-tracheal airway), percutaneous cricothyroidotomy and fiber-optic intubation. Suctioning device should also be readily available to eliminate saliva, blood or vomit from the upper airways. Pre-oxygenation (breathing with 100% oxygen for ≥5 min) should be considered in every case in order to improve tolerance to apnea during airway management [12].

Oxygenation. Almost every ARF case requires oxygen supplementation. Supplemental oxygen via nasal cannula or face mask with a goal of maintaining a PaO2 ≥60 mm Hg (which equals a SpO2 ≥90%) is beneficial in spontaneously breathing hypoxic patients, except in cases of right-to-left shunts [9]. While

correction of severe hypoxemia takes precedence over the potential of oxygen toxicity, if possible, it is best to avoid exposure to a fraction of inspired oxygen (FiO_2) greater than 0.6 (60% of O_2 in the inhaled gas mixture) for more than 24 hours. In some patients with chronic CO_2 retention (e.g. COPD) CNS respiratory drive is supported primarily with stimulating effects of hypoxemia rather than hypercapnia. Therefore, in such patients increase in PaO_2 by means of oxygen supplementation can result in depression of central neural drive and decreased ventilation with significant rise in $PaCO_2$ [6].

Ventilation. Ventilatory support is mainly intended to correct hypoxemia, hypercapnia and acidosis, and to decrease the work of breathing.

As part of the basic life support the patient may be ventilated using mouth-to-mouth, mouth-to-nose or mouth-to-face mask breathing. Bag-valve-mask (BVM) device provides an alternative that allows manual delivery of the positive pressure ventilation. The BVM can be connected to the oxygen source. Effective use of this device requires open airways and a tight seal between the mask and the face [12]. BVM may be essential in oxygenating a patient before endotracheal intubation or in cases when invasive airway management is unavailable. One of the potential complications of BVM ventilation is gastric insufflation with subsequent aspiration of gastric contents.

Mechanical ventilators (MV) are the machines capable to control delivery of positive pressure breath to the patient.

The indications for mechanical ventilatory support include [6]:

– apnea and cardiac arrest;

– persistent severe hypoxemia despite oxygen supplementation;

– $PaCO_2$>55 mm Hg with pH <7.25;

– respiratory distress with hemodynamic instability;

– vital capacity <15 mL/kg for neuromuscular diseases.

Positive-pressure MV may provide volume-cycled (VCV) and pressure-cycled ventilation (PCV). VCV delivers a predetermined V_T (volume of one ventilator breath) with preset respiratory frequency at a constant inspiratory flow [13]. The pressure increases until the lungs reach the set V_T. To minimize barotrauma, the V_T should not exceed 6-8 mL/kg of ideal body weight, and the upper pressure limit should be set by the physician.

In PCV the physician presets airway pressure rather than V_T, whereas the V_T becomes a dependable variable and is determined by the lung compliance [6]. The duration of inspiration is determined by preset inspiratory time and respiratory rate. The benefits of PCV as compared with VCV include reduced peak airway pressure with lower risk of barotrauma, and improved distribution of inspired gas [14]. A limitation of the PCV is that alterations of patient's respiratory mechanics (lung stiffness, increased air flow resistance) typically lead to insufficient V_T and decreased minute ventilation [6].

The most frequently used modes of mechanical ventilation are:

Controlled mandatory ventilation (CMV): With this mode of ventilation, spontaneous efforts by the patient do not trigger the ventilator to deliver a mechanical breath, so the patient has no control of ventilator functions [15]. To avoid significant discomfort and potential complications resulting from incoordination between the patient and the ventilator, CMV should be used for apneic, paralyzed and deeply sedated patients [15]. Continuous CMV leads to disuse atrophy of the respiratory muscles [13].

Assist-control ventilation (ACV): Allows patient to initiate a ventilator-delivered breaths by making inspiratory effort and, thus, to determine the respiratory rate and minute ventilation [13]. The inspiratory effort is sensed as a fall in ventilator circuit pressure [14]. If the effort is insufficient to trigger the ventilator, a preset number of breaths (backup rate) with a set V_T will be delivered to the patient to ensure adequate minute ventilation [15, 16]. The sensitivity of ventilator to patient effort is predetermined by a physician.

Intermittent mandatory ventilation (IMV): Mainly employed for weaning from mechanical ventilation, the IMV allows the patient to breathe spontaneously with his own rate and V_T in between the mechanical breaths delivered with predetermined rate and preset V_T [13, 14]. As the patient's spontaneous breathing improves, the ventilator backup rate is gradually reduced. In order to avoid overlapping of mandatory and spontaneous breath, mechanical breaths may be synchronized with patient's spontaneous efforts (SIMV) [16].

Pressure support ventilation (PSV): Assists spontaneous breathing of the patient with preset positive pressure to the ventilator circuit. The pressure support is maintained throughout inspiration. PSV may be used as a primary ventilatory mode in awake patients with adequate ventilatory drive and mild to moderately severe pulmonary pathologies, or during weaning from mechanical ventilation to unload the inspiratory muscles [6, 13]. PSV is better tolerated as compared with other modes, since patient can control the respiratory rate, duration of inspiration and inspiratory flow rate [16].

Airway pressure-release ventilation (APRV): Allows patient to breathe spontaneously at two levels of continuous positive airway pressure (CPAP; continuous mean that pressure is delivered during both inspiration and expiration) [16]. In APRV, periodically, the higher level of CPAP (inspiratory positive airway pressure) is released (decreased) to a lower level (expiratory positive airway pressure) for a moment to allow a larger breath (exhalation) for CO_2 elimination [6]. APRV is designed

to improve oxygenation by keeping the positive airway pressure throughout the respiratory cycle in order to prevent alveolar collapse [16].

Some cases of ARF can be successfully managed with noninvasive positive pressure ventilation (NIPPV) – ventilatory support without the use of an endotracheal or tracheostomy tube [17]. In appropriately selected patients NIPPV improves gas exchange, reduces respiratory work and relieves dyspnea. It can reduce the need of ETI and invasive mechanical ventilation, therefore decreasing the risk of related complications (e.g. injuries from ETI, prolonged weaning, nosocomial pneumonia). The well-established indications for NIPPV include acute exacerbations of COPD and acute cardiogenic pulmonary edema [16]. NIPPV is contraindicated in patients with compromised airways, CNS dysfunction and hemodynamic instability [17].

Финансирование/Funding

Авторы заявляют об отсутствии внешнего финансирования научной работы.

The authors declare that there was no external funding for the research.

Конфликт интересов/Conflict of interest

Авторы заявляют об отсутствии конфликта интересов.

The *authors declare* that there is no *conflict of interest*.

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