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CAUSES AND MANAGEMENT OF DYSPNEA

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Abstract:

Introduction: *Dyspnea is considered a very subjective clinical symptom of breathing difficulty and discomfort that typically consists of qualitatively distinct sensations that differ in strength and can only be recognized by the patient's complaint. Dyspnea is similar to suffocation and is one of the most difficult symptoms experienced by critically ill patients, including those on mechanical ventilation. When a patient cannot report dyspnea, as typifies many critically ill patients, the observed behaviors are characterized as respiratory distress. Expert guidelines can help in the management of dyspnea, however additional empirical evidence to support clinical care is required, and wide variation does exist in clinical practice. The aim of this paper is to address the following questions: (1) How prevalent, intense, and distressing is dyspnea experienced by critically ill patients? (2) How should dyspnea be assessed in the intensive care unit (ICU)? (3) What are current strategies for managing dyspnea during critical illness?*

Aim of work: *In this review, we will discuss dyspnea.*

Methodology: *We did a systematic search for management of dyspnea using PubMed search engine (http://www.ncbi.nlm.nih.gov/) and Google Scholar search engine (https://scholar.google.com). All relevant studies were retrieved and discussed. We only included full articles.*

Conclusions: *Alleviation of respiratory distress is a critical element of care in the ICU. A major goal for ICU care improvement is to not only enhance patient comfort, but to support other favorable outcomes of intensive care that are associated with dyspnea control. Selection of dyspnea assessment techniques to meet the specific communication capabilities of the patient is necessary to gain as much knowledge of what the patient is experiencing as possible. Selection of dyspnea management methods appropriate to the source, or anticipated source, of the symptom, the condition of the patient, and the goals of care requires the concerted efforts of a dedicated team of multidisciplinary health care professionals.*

Key words: *dyspnea, presentation, causes, management, primary care.*

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INTRODUCTION:

Dyspnea is considered a very subjective clinical symptom of breathing difficulty and discomfort that typically consists of qualitatively distinct sensations that differ in strength and can only be recognized by the patient's complaint[1]. Dyspnea is similar to suffocation and is one of the most difficult symptoms experienced by critically ill patients, including those on mechanical ventilation [2]. When a patient cannot report dyspnea, as typifies many critically ill patients, the observed behaviors are characterized as respiratory distress [3].

Expert guidelines can help in the management of dyspnea, however additional empirical evidence to support clinical care is required, and wide variation does exist in clinical practice. The aim of this paper is to address the following questions: (1) How prevalent, intense, and distressing is dyspnea experienced by critically ill patients? (2) How should dyspnea be assessed in the intensive care unit (ICU)? (3) What are current strategies for managing dyspnea during critical illness?

In this review, we will discuss the most recent evidence regarding dyspnea.

METHODOLOGY:

We did a systematic search for management of dyspnea using PubMed search engine (http://www.ncbi.nlm.nih.gov/) and Google Scholar search engine (https://scholar.google.com). All relevant studies were retrieved and discussed. We only included full articles.

The terms used in the search were: dyspnea, presentation, causes, management, primary care.

DYSPNEA MECHANISMS

The pathophysiologic explanation for dyspnea happens when there is a disorder in respiratory function. Normal respiration is a function of the complex assimilation of the respiratory control system including voluntary, autonomic and emotional responses. Voluntary control from the cortex were recognized in functional MRI studies of healthy human subjects during volitional breathing.[4]. The autonomic responses are controlled in the brainstem and are basic and vital to the existence of the organism and will override conscious controls. So, telling a dyspneic patient to slow down their breathing is a very challenging exercise.

Respiratory sensors include central (medulla, pons) and peripheral chemoreceptors (aortic and carotid bodies) and peripheral sensory receptors located in the chest wall, airways, and lungs. Changes in respiratory function will produce blood gas imbalances (hypoxemia and hypercarbia) and changes in thoracic displacement. Peripheral afferents play only a small role in respiratory control.

Activation of the respiratory center triggers an increased respiratory and cardiac response through activation of the parabrachial complex in the pons, sympathetic nervous system, and activation of the adrenal medullary catecholamines (epinephrine and norepinephrine). Stimulation of cardiac and pulmonary responses from central respiratory control and the sympathetic nervous system leads to compensatory responses, involving accelerations in heart and respiratory rates, increased lung volumes through recruitment of thoracic accessory muscles, changes in muscle tone, and increases in mean arterial pressure [5]. These cardiorespiratory responses are supposed to reinstate respiratory homeostasis and preserve life.

The awareness of difficulty breathing, dyspnea, and the related emotional responses of fear and anxiety are produced when there is pathology compromising normal respiratory functioning and is recognized by antecedent conditions, neurophysiologic,
pulmonary, and emotional responses, and patient subjective experiences and behaviors [6].

ANTECEDENT CONDITIONS AND MECHANISMS

Many of the common clinical conditions can cause dyspnea. There are many physiologic conditions have one or more similar mechanisms to produce difficult breathing, including respiratory effort, blood gas imbalances, and afferent mismatch. A sense of respiratory effort occur by conscious awareness of voluntary stimulation of the diaphragm, intercostals, and sternocleidomastoid muscle. Muscle receptors send a feedback about muscle force and tension, and data from these chest wall receptors produce the conscious awareness of respiratory effort. The respiratory muscles also stimulate autonomic central respiratory motor centers (ventromedial pons and medulla) that can add to the sense of effort.

Sense of effort comes from awareness of the motor order produced during a breathing task. One theory suggests that, while voluntary activation of muscles, a corollary discharge is produced from the oligosynaptic corticospinal pathway to the pontomedullary respiratory centers at the same moment that the efferent command is sent to the muscles, this corollary discharge is sensed as effort. The sense of effort is higher when the respiratory muscles are tired or deconditioned, when there is an elevated elastic or resistive load, or when the level of ventilation is higher. Hypercapnia and hypoxemia have been known to produce an involuntary motor response for a while started in the brainstem to elevate ventilation by increased rate and volume of breathing. Higher volume is accomplished by activation of the accessory muscles (intercostals and sternocleidomastoid) via the ventromedial pons. This higher recruitment of accessory muscles lead to the previously discussed higher sense of effort that correlates with dyspnea. Hypercapnia and hypoxemia also make independent contributions to the sensation of dyspnea [7].

Hypercarbia send reports of dyspnea and fear when the partial pressure of carbon dioxide (PCO2) is elevated by five to ten mm Hg from the person's baseline level. Severe hypercarbia (PCO2 >80 mm Hg) leads to a narcotic effect that will inhibit emotions and, at very high levels suppresses, the brainstem respiratory center causing death. Moosavi and colleagues20 (2003) showed that hypoxemia also has an important thresholds. Air hunger ratings is elevated acutely when levels of oxygen decreased to 50 mm Hg or less. Severe, persistent hypoxemia contributes to global brain ischemia and suppression of all brain functions until total brain death happens.

Additionally, a mismatch between afferent information (chemical and mechanical) and outgoing motor commands from the brainstem produces dyspnea. When subjects received an inspiratory flow less than that which they identified as most comfortable, they experienced dyspnea.21 Other investigators showed that healthy subjects are more comfortable when the mode of mechanical ventilation is like that the pattern is determined by the subject than when the pattern is enforced [8].

These findings propose that a deviation from an anticipated pattern of ventilation will lead to an elevation in uncomfortable respiratory sensations. Pathologic clinical conditions that lead to dyspnea do so by more than 1 mechanism. But, sense of effort is shared by most of the pathologic conditions, involving asthma, chronic obstructive pulmonary disease (COPD), congestive heart failure, interstitial lung disease, and neuromuscular problems. Hypoxemia and hypercarbia are also considered very common to all the clinical conditions that lead to dyspnea particularly during severe exacerbations and respiratory failure.

Conditions that produce dyspnea

There are many conditions that produce dyspnea, the most common conditions that lead to dyspnea presentation are:

Pulmonary:
Asthma, Adult respiratory distress syndrome, Chronic obstructive pulmonary disease
Cystic fibrosis, Interstitial lung disease, Lung cancer, primary or metastatic, Pleural effusion, Pneumonia, Pneumothorax Pulmonary arterial hypertension
Pulmonary embolism Radiation pneumonitis

Cardiovascular
Heart failure Congenital heart anomalies Superior vena cava syndrome

Neuromuscular
Amyotrophic lateral sclerosis Muscular dystrophy
Multiple sclerosis Myasthenia gravis Deconditioning

Miscellaneous
Hypervolemia Anemia

PREVALENCE, INTENSITY, AND DISTRESS IN THE INTENSIVE CARE UNIT

There are many studies performed in different ICU settings established that dyspnea is among the most prevalent, intense, and distressing physical symptoms experienced by critically ill patients who can provide a symptom self-report. In a new study of critically ill patients with cancer getting ICU care, thirty four percent of those who could self-report symptoms experienced moderate or severe dyspnea [9].

In one study of more than four hundred interviews with 171 critically ill patients at high risk of dying revealed dyspnea in forty four percent of evaluations. 2 Among those receiving mechanical ventilation, another researcher revealed that almost one-half of patients had dyspnea. The symptom experience of cognitively impaired patients, as typifies the critically ill and/or dying patient, is less well-established [10].

ASSESSMENT

The keystone of efficient symptom control is systematic symptom assessment. Typically, symptoms are reported and rated by patients themselves, using a tool that is simple enough and brief to prevent the burden while providing adequate data for clinical use. The Condensed Form of the Memorial Symptom Assessment Scale26 and the Edmonton Symptom Assessment Scale are techniques of this kind that measure a diverse group of physical symptoms including dyspnea and psychological symptoms. Additionally, a 10-item symptom scale incorporating 10 symptoms was validated in a large group of ICU patients.

In summary, symptom-specific techniques are readily available to get self-reports of dyspnea. For patients who cannot speak because of endotracheal intubation or other reasons, doctors should give chances to report through head shaking (yes/no) or pointing on a visual analog scale.

When the patient is capable to report but not verbalize symptom information, there could be a role for speech language pathologists to assist or augment the patient's ability to communicate and to assist communication by different methods. Simple strategies involve alphabet and numbers boards, although more sophisticated modalities include electronic speech-generating devices or a touch screen requiring minimal physical pressure to activate message buttons [11].

Though some patients cannot provide a dyspnea self-report, doctors should not assume that they cannot experience respiratory distress. Though other symptom information is less accurate than the patient's own report, the symptom intensity and/or distress of critically ill patients who cannot provide self-reports must still be studied. Two main approaches have been used for this purpose: (1) behavioral assessment and (2) proxy assessment.

Behavioral Symptom Assessment

The Respiratory Distress Observation Scale (RDOS) is the only recognized behavioral scale for assessment of respiratory distress when a patient is not able to report dyspnea. RDOS is an ordinal scale with 8 observer-rated parameters: heart rate, respiratory rate, accessory muscle use, paradoxical breathing pattern, restlessness, grunting at end-expiration, nasal flaring, and a fearful facial display.

Each is scored from zero to two points and the points are summed. Scale scores range from zero signifying

no distress to sixteen implying the most severe distress Behavior variables that comprise the RDOS were identified from videotaping mechanically ventilated patients undergoing a failed ventilator weaning trial and experiencing naturally occurring dyspnea. Construct validity was established through correlation with hypoxemia [12] and use of oxygen.

Convergent validity was shown by comparison with dyspnea self-report on a visual analog scale. Discriminant validity was concluded with comparisons of RDOS from COPD patients with dyspnea to patients with acute pain and healthy volunteers.

Proxy Symptom Assessment

The use of symptom reports from surrogates, like family members, or doctors themselves, in many patient populations continues to be controversial. In some specific studies, patients categorize their symptoms higher than proxy reporters, while in other reports the opposite is true.

WHAT ARE CURRENT STRATEGIES FOR MANAGING DYSPNEA DURING CRITICAL ILLNESS?

The first line to manage dyspnea is by optimizing the management of the underlying etiologic condition such as with inotropes and diuretics for heart failure exacerbations, bronchodilators for COPD, thoracentesis, or antibiotics to name a few. Mechanical ventilation, either invasive or noninvasive, is the most valid ways of reducing dyspnea from respiratory failure, although not without the well-understood burdens to the patient. Some patients will not want to undergo mechanical ventilation and the treatment of dyspnea must depend on other interventions, like those directed at releasing the sensation of dyspnea and the associated emotional response; these can be effective singly or in combination.

The best positioning is patient specific. For example, dyspnea in COPD is decreased by upright positioning with arms elevated on pillows or a bedside table.38,39 In unilateral lung disease the patient may find a side-lying position optimal with the "good" lung up or down to increase perfusion and/or ventilation. Using the patient as his or her own control and measuring dyspnea or respiratory distress in different positions allows identification of the optimal position. Patient activity, whether active or passive, increases oxygen consumption and may lead to dyspnea. Nurses can coordinate all patient care and are integral to ensuring staggering of activity to minimize or prevent dyspnea.

Oxygen is helpful to decrease the severity of dyspnea caused by hypoxemia. But no benefit from oxygen in comparison to medical air was found in a multinational study of patients with advanced lung disease who were not hypoxemic.40 Moreover, patients who were near death and at risk for dyspnea continues to be comfortable without oxygen [13].

Opioids, most commonly immediate release oral morphine or intravenous fentanyl, are the gold of drugs of dyspnea that is resistance to disease-modifying drugs, and its effectiveness has been showed in clinical trials [14]. Effectiveness has not been concluded for other opioids such as hydromorphone or hydrocodone, or other routes such as transdermal or intravenous. The doses of opioids for acute dyspnea exacerbations are less well-known than those used to treat acute pain; "low and slow" titration of an immediate release form given intravenously and repeated every fifteen minutes should be provided till the patient demonstrates or displays relief. Around the clock dosing could be best if the patient has dyspnea continuously or at rest, however with as-needed dosing for episodic dyspnea [15].

The effectiveness of benzodiazepines as the main treatment for dyspnea has not been well concluded. Doctors fears about respiratory depression are in contrary to the evidence; no patients in studies of opioid use for dyspnea had respiratory depression. Doctors are advised to consider patient comfort as a priority and, in so doing, call on the use of interdisciplinary communication, assessment, decision making, and drug titration skills to provide relief for all patients.

CONCLUSIONS:

Alleviation of respiratory distress is a critical element of care in the ICU. A major
goal for ICU care improvement is to not only enhance patient comfort, but to support
other favorable outcomes of intensive care that are associated with dyspnea control.
Selection of dyspnea assessment techniques to meet the specific communication capabilities of the patient is necessary to gain as much knowledge of what the patient is experiencing as possible. Selection of dyspnea management methods appropriate to the source, or anticipated source, of the symptom, the condition of the patient, and the goals of care requires the concerted efforts of a dedicated team of multidisciplinary health care professionals.

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