Clinical Aspects and Scoring the Severity of Dyspnea: A Short Review

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Abstract

One of the most frequent complaints in acute patient care is dyspnea. It has several influencing factors—a significant amount of subjective and affective components, therefore it is difficult to develop a universally accepted agreement about dyspnea. Until now we have not had a standard definition and the pathophysiology is also dubious and very difficult. What is sure that neither specialized dyspnea receptor nor dyspnea center in the central nervous system exist. Besides a quite complex neurobiological model, the supply/demand discrepancy at the respiratory muscle level can also contribute to the pathomechanism. Due to the lack of a “gold-standard” instrument which could describe the severity of dyspnea universally this overview presents two simple scoring methods (DSS-4 and the MARKED-risk score) which can be suitable to evaluate the severity of dyspnea and for risk stratification in acute patient care.

Keywords: Dyspnea; Pathomechanism; Scoring; Acute care; COPD; Heart failure

Abbreviations

AUC: Area Under the Curve; BE: Base Excess in Blood Gas Sample; COPD: Chronic Obstructive Pulmonary Disease; CNS: Central Nervous System; Cys-C: Cystatin-C; DO2: Oxygen Delivery; DSS-4: Dyspnea Severity Score-4 dimensions; ED: Emergency Department; FEV1: Forced Expiratory Volume During One Second; hs-CRP: High-Sensitivity C-Reactive Protein; hs-cTnT: High-Sensitivity Cardiac Troponin T; MARKED: Multi Marker Emergency Dyspnea; NT-proBNP: N-terminal pro-B-type Natriuretic Peptide; SpO2: Pulse Oxymetric Saturation; VO2: Oxygen Consumption.

Introduction

The main purpose in writing this small review was to call attention to the difficulties of terminology and pathophysiology, the clinical contradictions and the severity estimation problems of dyspneic patients.

Definition

Dyspnea is the most frequent and one of the most complex respiratory symptoms in acute patient care. We can define it very simply e.g. “inappropriate relationship between respiratory work and total body work” [1] or as “an abnormally uncomfortable awareness of breathing” [2] or in a popular way “the clinicians know what it means”, according to guidelines of The European Society of Cardiology [3]. A more detailed definition of dyspnea from The American Thoracic Society attempts to involve several aspects of the symptoms and reveals the complexity of its nature: “a subjective experience of breathing discomfort that consists of a quality distinct sensation that varies in intensity” and involves “interactions among multiple physiological, social, and environmental factors, and may induce secondary physiological and behavioral responses” [4,5].

Pathophysiology of Dyspnea

Until now we do not know the exact pathophysiology of dyspnea. What seems to be sure is that there are no specialized dyspnea receptors, there is no area of the cerebral cortex that when stimulated causes dyspnea, nor is there a cortical lesion that abolishes the sensation of dyspnea or the perception of other respiratory related stimuli [6]. A neurobiologic model tries to describe the complexity of the pathomechanism in COPD patients [7,8]. Neural inputs from the airways (pulmonary stretch receptors, C fibers), from the lungs (pulmonary stretch receptors, C fibers, J receptors) and from peripheral locomotor and respiratory muscles (muscle spindles, Golgi tendon organs, type 3 and 4 afferents) and the feedback from the central and peripheral chemoreceptors (adequacy of ventilation and gas exchange) with increased central cordial discharge from brainstem and cortical motor centers reach the somatosensory cortex and contribute to the feeling of dyspnea. Increased activation of the limbic system as a result of neuromechanical dissociation contributes to “respiratory distress” [7]. Endogenous opioids can modify breathlessness affecting the respiratory rhythm and nociception [9,10]. This component of the pathomechanism is a useful therapeutic possibility in the clinical practice in most dyspnea cases.

The sense of respiratory effort increases whenever the central motor command to the respiratory muscles has to be increased [6]. However, there is some evidence to suggest that effort and breathlessness are not the same. The sense of effort will be a significant contributing factor to dyspnea when the respiratory muscles are fatigued or weakened in respect of the load [6]. The increased work of the respiratory muscles without adequate energy supply can be a common mechanism in the most forms of dyspnea. The oxygen consumption/supply disturbance (VO2/DO2 relationship) at the tissue level can occur for several reasons and all of them can cause dyspnea. From a clinical approach the most frequent reasons for low oxygen delivery (DO2) are the followings: low cardiac output, disturbed microcirculation, low hemoglobin concentration, low arterial oxygen content, shift of the oxygen dissociation curve, etc. The reasons for increased oxygen demand (VO2) in the respiratory muscles are factors resulting from any kind of increased ventilation, e.g. metabolic acidosis, hypercarbia, fever, sepsis, psychogenic hyperventilation, etc.

A few recent studies support the importance of tissue oxygenation adequacy in the respiratory muscles. In healthy volunteers hyperoxia increased the maximal power output and endurance. It improved arterial, cerebral and muscle tissue oxygenation and reduced the perception of dyspnea [11]. In another study in healthy men the effect of inspiratory muscle training on respiratory muscle electromyography and dyspnea during exercise was studied [12]. The inspiratory muscle training significantly reduced the submaximal dyspnea intensity rating but did not change the electromyography of any inspiratory muscles. However, to prove the role of VO2/DO2 disturbances in the respiratory muscles in the development of dyspnea needs further studies.

Clinical Relevance of Dyspnea

Dyspnea is associated with many disorders from psychological...
problems that are not dangerous to life-threatening conditions. Because it contains a large subjective component its degree does not necessarily correlate well with the severity of the underlying disease, e.g. patients with chronic disease get used to their symptoms and rate their illness much less than the actual severity of that illness. Both in acute or chronic illnesses the psychological condition (negatively affected mood, anxiety, depression) can modify the feeling of dyspnea [13,14]. Because of its complexity it is very difficult to develop a “gold-standard” instrument which could describe the severity of dyspnea in any illness, in any patient and at any time. A universal, validated, accurate, reliable instrument that is reproducible between and among observers and that is sensitive and specific enough to changes would be extremely helpful [15].

We require a standardized dyspnea measurement tool in the emergency department to estimate the severity and the prognosis of dyspnea; during the care of any patient, to evaluate the efficiency of the therapy; and in clinical trials to ensure the exact inclusion criteria and endpoint of the study. This tool would also be useful for the pharmaceutical industry and regulatory agencies when a new drug is being introduced in clinical practice [16,17]. However, until now we have not had a universal tool which would fulfill all these criteria, so we have to use more or less validated instruments for specific situations.

Scoring Instruments

Most of the current scoring systems developed to estimate the severity of dyspnea are based mainly on subjective parameters and concentrate only on cardio-pulmonary disorders. The widely used Borg-scale [18,19] or its modified 10 point version [20] evaluate the patients’ breathlessness from the level of non-existent to the maximum. Similar one-dimensional rating methods exist to measure dyspnea in different conditions (visual analogue scales [21], numerical rating scales [22–24]). The effectiveness of these scales has been proved mainly in patients with COPD or asthma, despite the fact that the correlation of dyspnea is quite weak with most of the lung function parameters, especially with FEV1 [25–26]. The problem with all similar instruments is that they focus only on the global severity of dyspnea influenced by the patients’ actual subjective feelings.

In our previous study [27] we analyzed the relationship between the patients’ subjective dyspnea feelings (10 point numeric scale) and the objectively measured dyspnea severity (13 point score using the deviation of pH, BE and lactate level from the normal value) in a mixed population in two emergency departments. Altogether, we found a dose correlation (p < 0.001) between the subjective dyspnea rating scores and the objective classification scale points (Figure 1), however, the scatter was very large over the whole range of subjective points limiting the usefulness in an individual patient. This observation confirmed the weakness of the one-dimensional rating methods in the measurement of dyspnea severity.

Because dyspnea is a complex symptom with multiple components, it is logical that its exact evaluation supposes the analysis of the different determinants at the same time. All of the recent scoring methods apply this approach. Without the need for completeness we can mention the “dyspnea-12” instrument, which provides a score of physical and affective aspects of dyspnea [28], the “Multidimensional Dyspnea Profile”, which assesses overall breathing discomfort, sensory qualities, and emotional responses [29,30], and the “Provocative Dyspnea Severity Score”, which evaluates the dyspnea response in four stages (A: upright-with oxygen, B: upright-without oxygen, C: supine-without oxygen and D: exercise-without oxygen) [15,31]. Recently, two papers were published for measuring dyspnea in emergency patient care with adequate validation [27,32]. Both studies used a multimarker strategy and involved a mixed patient population, but examined the dyspnea from different points of view.

The purpose of our study [27] was to develop a simple scoring system to evaluate the severity of dyspnea in emergency care immediately after the patient’s arrival at the ED. The basic severity scale involved seven dimensions from 0 to 3 points (exercise tolerance, speech, cooperation, cyanosis, SpO2, breathing, and heart rate/rhythm) and the validation based on the previously mentioned objectively measured parameters. Three hundred and fifty patients were included in the study; 184 patients (53%) had pulmonary and 152 patients (43%) had cardiac problems. Using a forward stepwise model of multiple regression analysis we were able to reduce the number of dimensions from seven to four parameters (DSS-4: exercise tolerance, cooperation, cyanosis, and SpO2) without significantly increasing the prediction error. The prognostic probability to predict the altered pH, BE and lactate values did not decrease significantly compared to the original model where all of the dimensions were included. When the patient had ≥ 7 DSS-4 points the AUC values were 0.78–0.99 with 86–100% sensitivity and 66–99% specificity to predict a severe clinical status (higher the point values better the sensitivity and specificity), indicating the need for urgent intervention.

In the MARKED-risk score study [32] different biomarkers (N-terminal pro–B-type natriuretic peptide (NT-proBNP), highsensitivity cardiac troponin T (hs-cTnT), Cystatin-C (Cys-C), high-sensitivity C-reactive protein (hs-CRP), and Galectin-3) with clinical risk factors were studied as a risk stratification tool for 90-days. 603 patients brought to the ED with dyspnea were included in the final analysis, 57% with decompensated heart failure and 28% with noncardiac diagnosis. Using multivariable analysis the combination of four biomarkers (hs-cTnT, hs-CRP, Cys-C, and NT-proBNP) reached a high predictive accuracy with an AUC for 90-day mortality of 0.83 and the number of biomarkers elevated (i.e. none, 1, 2, 3, or 4) was strongly associated with an increased risk of 90-day mortality (odds ratio: 2.94 per elevated biomarker) with 90-day mortality rates of 0.7%, 4.5%, 10.4%, 25.2%, and 49.1%, respectively (p < 0.001). Incorporating the clinical risk factors in the analysis, the final MARKED-risk score consisted of: age > 75 years, history of heart failure, dyspnea at rest, systolic blood pressure <
110 mmHg, hs-CRP > 25 mg/L hs-cTnT > 0.04 g/L, and Cys-C > 1.125 mg/L. The score showed excellent discrimination (AUC: 0.85) and predicted mortality risk closely resembled the observed mortality risk. The ninety-day mortality rates gradually increased per MARKED-risk score point. The score was categorized into low (≤ 2 points), intermediate- (3 to 5 points), and high-risk categories (≥ 6 points). The 90-day mortality risk of admitted versus nonadmitted patients was similar within each risk score category (low: 1% vs. 2%; intermediate: 11% vs. 15%; high: 39% vs. 46%). In addition, 9% of the patients that were discharged from the ED were in the high-risk category and 39% of these patients died within 90 days, underscoring the clinical prognostic uncertainty and the potential importance of the MARKED-risk score. Table 1 summarizes the important aspects of these two instruments in the clinical practice.

**Conclusions**

The basic problems with dyspnea are that its exact pathomechanism is not known — there is no specific dyspnea receptor and specific center in the CNS, there is no single parameter which would characterize the severity of the illness — the dyspnea feeling is significantly influenced by emotional components, and finally, there is no specific, universally accepted instrument for measuring its severity. In this paper the DSS-4 and the MARKED-risk score are reviewed in more detail, which is suitable for severity estimation and risk stratification in acute patient care. However, there is a need for future research to analyze the effectiveness of these scoring instruments in separate patient populations (cardiac, pulmonary, and others).

**Conflict of Interest**

The author has no financial conflict of interest but he was the principal investigator of the DSS-4 study, therefore the statements in this review could have been influenced by the previous trial.

**References**


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