Descriptors of dyspnea in obstructive lung diseases
Descrittori di dispnea nelle patologie ostruttive delle vie aeree

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ABSTRACT
In obstructive lung diseases such as asthma and COPD dyspnea is a common respiratory symptom with different characteristics given the different pathogenic mechanisms: in COPD initially it can occur during exertion but then it increases progressively along with the airflow obstruction, whereas in asthma it occurs episodically and is caused by transient bronchoconstriction.

The language of dyspnea includes a large range of clinical descriptors which have been evaluated for their correlation (of one or several descriptors) with underlying physiologic/physiopathologic mechanisms. These studies were done in asthma rather than in COPD, and dyspnea descriptors were found to be useful in identifying patients with life-threatening asthma. However further studies are needed to further explore such descriptors and their clinical utility.

This review discusses dyspnea mechanisms in various obstructive lung disease subsets as well as the descriptors of dyspnea and their utility in clinical practice.

Keywords: Descriptors, dyspnea, exercise testing, obstructive lung diseases.

INTRODUCTION
Obstructive lung diseases are represented mainly by asthma and by chronic obstructive pulmonary disease (COPD) which are both inflammatory diseases of the airways but with different pathogenesis and clinical manifestations: in asthma inflammation is triggered and maintained usually by allergens, renders the bronchial muscle hyperreactive to various stimuli including allergens themselves, and the consequent bronchospasms manifest clinically with episodic dyspnea and wheezing subsequently relieved with rescue bronchodilator medication. In COPD cigarette smoking exposure is the main risk factor and the airways inflammation resulting from such aggression is progressive, reducing the airways lumen, and respiratory symptoms are dominated by
dyspnea, cough and sputum production which increase in severity with disease progression or during disease exacerbations.

Dyspnea is a common clinical denominator in both asthma and COPD and its different characteristics in the two diseases reflect the different pattern of inflammation and of airways obstruction. This review discusses separately the mechanisms of dyspnea in asthma and COPD, its descriptors, and their clinical utility.

**DYSPNEA IN COPD**

In COPD dyspnea is one of the main respiratory symptoms present during stable or exacerbated state. It manifests initially during exertion and then, as the disease progresses, also during rest. It is caused by airflow obstruction which is secondary to airways inflammation, airways remodelling and sputum hypersecretion; reduced lung elastic recoil due to emphysema and the obstruction of small airways result in incomplete air expelling and dynamic hyperinflation (“air trapping”) [1].

**Mechanisms of dyspnea in COPD**

Overall exertional dyspnea in COPD is the result of complex pathophysiologival mechanisms including dynamic hyperinflation, increased ventilatory demand relative to impaired capacity, hypoxemia, hypercapnia, and neuromechanical dissociation (Table I) [1].

During exercise the reduced pulmonary elastic recoil contributes to an increase in the end-expiratory lung volume, and dynamic hyperinflation (DH) occurs. There are several adaptive mechanisms to the DH, including the reconfiguration of the rib cage in order to contain the overinflated lungs, and an increased activity of the respiratory muscles, in particular of the diaphragm, in order to generate increased pressures able to compensate the reduced elastic recoil, but such mechanisms can usually be compensatory during resting or during more reduced airways inflammation (such as in stable disease). During exertion or increased inflammation (exacerbation) the increased DH impairs the ability of the respiratory muscles to increase the intrathoracic pressure in response to the increased drive of breathing. The increased breathing rate is consequent to the inability of the overinflated lung to maintain or increase the inspiratory capacity during exercise [2]. During exercise such abnormalities are augmented by increased ventilatory demands due to ventilation perfusion mismatches, metabolic acidosis, hypoxemia, increased sympathetic tone, etc. [1].

Gas exchange abnormalities occurring in COPD patients during exercise are represented by arterial hypoxemia and/or hypercapnia: the former occurs in the milder COPD stages due to reduced ventilation and shunting, whereas in more advanced COPD alveolar hypoventilation adds to the above mentioned mechanisms. Exercise hypercapnia in COPD is due to reduced respiratory drive, breathing patterns altered to minimize respiratory discomfort, or ventilatory muscle fatigue and has been shown to be associated with greater dynamic hyperinflation [3].

Neuromechanical dissociation (NMD) between the attempted and actual respiratory effort which occurs between efferent motor neurons and afferent fibers has been hypothesised as a physiopathogenic mechanism in chronic obstructive pulmonary diseases such as COPD and asthma [1]. In a study evaluating the perception of exertional breathlessness during symptom-limited incremental cycle exercise testing in normal subjects and in patients with chronic airflow limitation, perceived inspiratory difficulty was assessed with the Borg scale (Borg IN), inspiratory effort was evaluated with esophageal pressure expressed as a fraction of maximal esophageal pressure at isovolume \((\text{Pes/Plmax})\), and breathing pattern, operational lung volumes (end-expiratory/inspiratory lung volumes \((\text{EELV/EILV})\)) were measured and compared at a standard \(\text{VO}_2\) of 50% predicted maximum. Breathlessness descriptors chosen and reported immediately after exercise differed between normal subjects and patients with chronic airflow obstruction. Descriptors of increased ‘work/effort’ and ‘heaviness’ of breathing were chosen by both normal subjects and patients with airflow obstruction whereas the latter category constantly chose descriptors such as ‘increased inspiratory difficulty’ (75%), ‘unsatisfied inspiratory effort’ (75%), and ‘shallow breathing’ (50%). The ratio of Pes/Plmax to VT/predicted VC was identified as the strongest correlate with the Borg scale [4].

The differences between the quality of descriptors for dyspnea among healthy subjects and patients with airflow obstruction suggest that the latter category receives altered peripheral sensory afferent information from mechanoreceptors in the respiratory muscles, chest wall and lungs signaling to them that the mechanical response of the ventilatory system is insufficient or inappropriate for the effort expended [5].

This sense of increased effort is believed to be the result of corollary discharge relayed from the motor cortex to the sensory cortex in the forebrain. Unlike normal subjects, in whom respiratory effort matches the ventilator demand both at rest and during exercise, in patients with COPD there is increasing disparity between effort and ventilatory output (i.e. neuromechanical dissociation) which becomes

**TABLE I: PATHOPHYSIOLOGICAL MECHANISMS OF DYSPNEA IN COPD**

- Dynamic lung hyperinflation
- Increased ventilatory demand relative to impaired capacity
- Hypoxemia
- Hypercapnia
- Neuromechanical dissociation
more prominent as exercise progresses, because dynamic hyperinflation constrains tidal volume expansion. Consequently COPD patients experience intolerable dyspnea very quickly during exercise, and describe it as ‘no room to breathe’ [5].

DYSPNEA IN ASTHMA

In asthma dyspnea is the clinical manifestation of episodic bronchospasm, and its presence represents a sign of suboptimal (therapeutic) control of the disease. Its severity can vary with the severity of the asthma attack or exacerbation and it can be perceived differently. For instance, some patients can perceive promptly the mild respiratory symptoms with increasing frequency, i.e. an asthma exacerbation occurring while on maintenance therapy, and can act promptly by self administering appropriate medication in order to reduce the flared inflammation; other patients, on the contrary, are unable to perceive appropriately such symptoms at their onset and may seek medical care for a severe/life-threatening asthma.

Dyspnea perception in asthma can be influenced by various factors (Table II) and knowledge of these factors as well as of the descriptors of dyspnea may contribute to improve disease control.

Dyspnea perception and its influencing factors in asthma

In asthma it is difficult to differentiate between ‘normal’ and ‘abnormal’ (poor or exaggerated) perception of dyspnea [2]. Poor perception of dyspnea means underestimation of its severity and this has been demonstrated to be a major risk factor for life-threatening asthma (LTA) and disease morbidity/mortality: in a study evaluating symptoms perception in subjects undergoing methacholine bronchoprovocation testing, about 15% of the 82 patients with a fall of FEV1 to the provocative concentration of methacholine (PC20) and at the highest dose of methacholine (maximum response), asthma patients selected descriptors such as ‘inspiratory difficulty’, ‘chest tightness’, ‘unsatisfied inspiration’, or ‘work’ at the dose nearest to PC20 but were more frequently selected at the highest methacholine dose. Furthermore patients who reported chest tightness at maximum response had greater airflow obstruction and dynamic hyperinflation than those who did not report chest tightness [10]. Under therapy, the frequency of reporting descriptors such as ‘chest tightness’ has been found to decrease, whereas the frequency of reporting others such as ‘work’ or ‘breathing effort’ persisted concomitantly with airflow obstruction [11,12].

CONCLUSIONS

The clinical descriptors of dyspnea in asthma and COPD can be relevant for both diagnostic and therapeutic purposes: in the initial clinical evaluation of an unexplained dyspnea they might be able to provide information which is additional to the evaluation of physiologic parameters, e.g. in a patient with COPD asymptomatic at rest, obtaining descriptors for exertional dyspnea factors can help in identifying the limiting factor of the exercise capacity, or in a patient with asthma certain dyspnea descriptors can signal suboptimal therapeutic control of disease or may help in detecting life threatening forms of asthma.

The language of dyspnea includes a large range of descriptors, and although a certain amount of research evaluating the correlation of one or several descriptors with underlying physiologic/pathophysiologic mechanisms has been already done, more studies are needed to further explore such descriptors and their clinical utility. Such studies can be greatly helped by the use of dyspnea questionnaires which offer standardized clinical descriptors able to detect rapidly the cause of breathing abnormalities: despite the fact such questionnaires are commonly used in COPD to evaluate dyspnea and the effects of various thera-
pies on its severity, few studies have focused on dyspnea descriptors and their clinical utility in COPD; this has more frequently been done in asthma and the results obtained and discussed above should encourage similar studies in COPD.

Overall documentation of dyspnea descriptors in obstructive lung diseases has, beyond pure research interest, a clinical applicability in disease diagnosis, in severity ascertainment and in therapeutic follow up, and further studies can therefore improve the knowledge in the field.

CONFLICT OF INTEREST STATEMENT: The author has no conflict of interest to declare in relation to the subject of this manuscript.

References