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.

RIASSUNTO

Nelle patologie ostruttive respiratorie come l'asma e la BPCO la dispnea è un sintomo molto comune, con caratteristiche differenti a seconda del meccanismo patogenetico sotteso: nella BPCO si verifica inizialmente durante esercizio fisico per poi incrementare progressivamente col progredire dell'ostruzione bronchiale, mentre nell'asma insorge episodicamente ed è causata da un broncospasmo transitorio.

Il linguaggio della dispnea include un'ampia varietà di descrittori clinici di cui è stata valutata la correlazione (di uno o più descrittori) con i meccanismi fisiologici e fisiopatologici sottostanti. Questi studi sono stati effettuati nell'asma piuttosto che nella BPCO ed i descrittori di dispnea sono stati riscontrati utili per identificare i pazienti con asma in pericolo di vita. Tuttavia studi ulteriori sono necessari per esplorare ulteriormente questi descrittori e la loro utilità clinica. In questa rassegna vengono analizzati i meccanismi della dispnea nei vari sottogruppi di patologie respiratorie di tipo ostruttivo, come pure i descrittori di dispnea e la loro utilità nella pratica clinica.

Parole chiave: Descrittori, dispnea, patologie ostruttive respiratorie, test da sforzo.

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

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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 pathohysiological 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 (BorgIN), inspiratory effort was evaluated with esophageal pressure expressed as a fraction of maximal esophageal pressure at isovolume [Pes/PImax]), and breathing pattern, operational lung volumes (end-expiratory/inspiratory lung volumes [EELV/EILV]) were measured and compared at a standard $\dot{V}O_2$ 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/PImax 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 methacoline bronchoprovocation testing, about 15% of the 82 patients with a fall of FEV_1 to values < 50% predicted were reported to be asymptomatic, whereas in another study in patients with history of near fatal asthma perception of dyspnea during external resistive loading was found to be lower compared to normal subjects but comparable to that of patients with non fatal asthma, and low perception of dyspnea was found to be associated with significantly longer hospitalisations, near fatal exacerbations and/or death [6-8].

In asthma, dyspnea is usually described as 'chest tightness', increased 'effort' or 'work' of breathing, or 'difficult breathing', and it has been demonstrated that the symptom quality varies with the degree of bronchoconstriction, e.g. the tightness was found to be associated with mild bronchoconstriction and attributed to vagal stimulation due to increased airways resistance but not to dynamic hyperinflation [9].

However, in a study evaluating the relationship between respiratory symptom intensity and quality and dynamic lung hyperinflation during bronchoconstriction induced by high-dose methacholine challenge (maximum decrease in FEV_1 of 50% from baseline in asthma) in which the qualitative descriptors of dyspnea and functional residual capacity (FRC) were measured at the doses nearest to the provocative concentration of methacholine causing a 20% fall in FEV_1(PC_{20}) 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 PC_{20} 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].

TABLE II: FACTORS AFFECTING SYMPTOMS PERCEPTION IN ASTHMA

- Advanced age
- Suboptimal disease therapeutic control (presence of airways hyperreactivity, peak expiratory flow variability)
- Chronic airflow obstruction
- Asthma medications
- Personality/psychological traits

From [2], mod.

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.

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