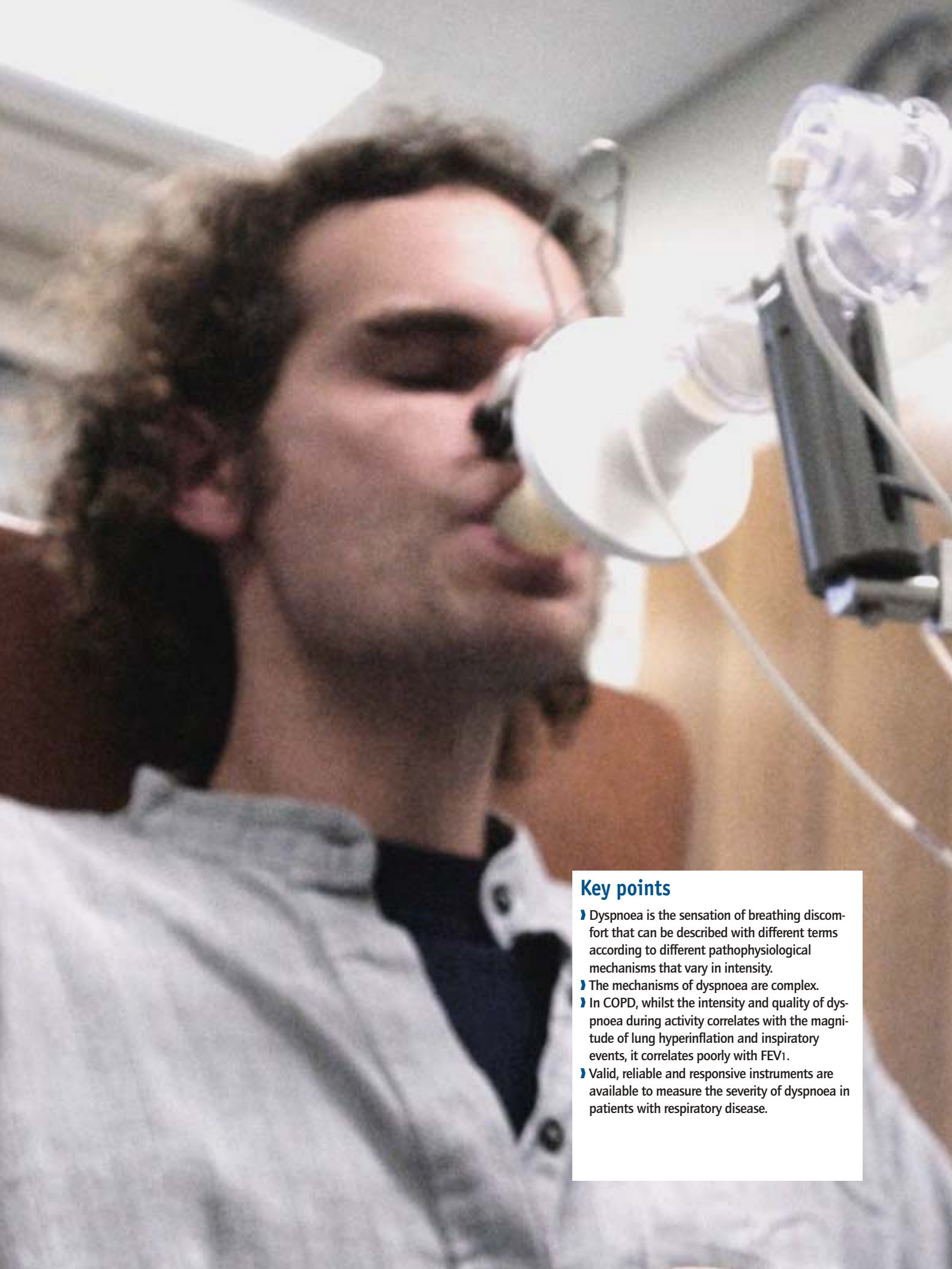

Key points

- Dyspnoea is the sensation of breathing discomfort that can be described with different terms according to different pathophysiological mechanisms that vary in intensity.
- The mechanisms of dyspnoea are complex.
- In COPD, whilst the intensity and quality of dyspnoea during activity correlates with the magnitude of lung hyperinflation and inspiratory events, it correlates poorly with FEV_1.
- Valid, reliable and responsive instruments are available to measure the severity of dyspnoea in patients with respiratory disease.

Photo: Jan Turnbull

Dyspnoea and its measurement

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CME article: educational aims

- To introduce dyspnoea and explain its mechanisms.
- To present dyspnoea descriptors, which may help in the understanding of the language of dyspnoea, and to relate these to specific diseases.
- To describe some of the methods available for the measurement of dyspnoea.

Summary

Dyspnoea, a term used to characterise a subjective experience of breathing discomfort, is perhaps the most important symptom in cardiorespiratory disease. Receptors in the airways, lung parenchyma, respiratory muscles and chemoreceptors provide sensory feedback *via* vagal, phrenic and intercostal nerves to the spinal cord, medulla and higher centres. Knowledge of dyspnoea descriptors can help in understanding the language of dyspnoea and these are presented here. It is important to appreciate that differences in language, race, culture, sex and previous experience can all change the perception of and the manner in which the feeling of being dyspnoeic is expressed to others. Therefore, standard tools to measure dyspnoea are available. In addition, there are tools that can help to relate the severity of symptoms with observed levels of cardiac and pulmonary responses while performing supervised tasks. A range of methods are described here. Inventories that involve aspects of dyspnoea related to quality of life are not yet a routine part of the history and physical examination, although they have proved useful in the clinic. Measurement instruments may involve a cost for use, may be self-administered or require an interviewer, and can vary in the time required for completion and scoring.

"Dyspnoea is perhaps the most important symptom in cardiorespiratory disease, although it is the least well understood. It has been defined previously as: "a term used to characterize a subjective experience of breathing discomfort that consists of qualitatively distinct sensations that vary in intensity. The experience derives from interactions among multiple physiological, psychological, social and environmental factors, and may induce secondary physiological and behavioural responses" [1]. This definition underlines: 1) the importance of language when describing a symptom; 2) the different qualitative sensations covered by the term dyspnoea; 3) the involvement and integration of multiple sources of neuronal information concerning breathing; and 4) the physiological and behavioural consequences. In other words, breathlessness is a complex and scarcely understood sensation that can be

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perceived and described only by the individual who is experiencing it [2]. Despite this scientific relativism, this article tries to describe dyspnoea in a scientific manner, in which it can be defined by numeric characterisation, that is "to be able to measure" [3].

Mechanism of dyspnoea

Receptors in the airways, lung parenchyma, respiratory muscles and chemoreceptors provide sensory feedback *via* vagal, phrenic and intercostal nerves to the spinal cord, medulla and higher centres [4].

Peripheral and central chemoreceptors can sense changes in arterial oxygen tension, carbon dioxide tension and pH. Indeed, in patients with impaired ventilatory function, chronic respiratory failure may be revealed by exercise and, therefore, may induce dyspnoea. This is reflected by changes in blood gases during exercise, specifically when lung diffusion is impaired or alveolar dead space is increased.

Among the pulmonary receptors, slowly adapting stretch receptors, located principally in the large airways, respond to increases in lung volume. Rapidly adapting receptors in the airway epithelium respond to different stimuli, such as particulate irritants, direct stimulation of the airways and pulmonary congestion. Juxtapulmonary receptors are non-myelinated fibres (C-fibres), that are located near pulmonary capillaries and in the bronchial and laryngeal mucosa, and are stimulated by mechanical and chemical stimuli. Almost all of the afferent signals from pulmonary receptors are ultimately carried to the central nervous system (CNS) *via* the vagus nerve. Mechanical receptors for volume, flow, muscle shortening, muscle tension and chest wall displacement provide a peripheral sensory feedback, modulating the intensity of central motor output at the central sensory level. Dyspnoea may reflect the perception of effort; that is the awareness of the efferent motor command from the CNS to the respiratory muscles.

Dyspnoea may occur when a greater than expected respiratory muscle activity is required to produce a given amount of ventilation. This has been described as "length-tension inappropriateness" [5]. With regard to the respiratory system, "length" actually corresponds to the change in lung volume, while "tension" corresponds to the respiratory pressures produced. Intensity of dyspnoea is influenced by a mismatch between the respiratory motor command from the CNS and afferent feedback arising from the receptors (described previously) in the respiratory system (neuroventilatory dissociation (NVD) of the respiratory pump) [6–8]. In chronic obstructive pulmonary disease (COPD) patients during exercise, the relationship between effort (motor output) and the anticipated ventilatory consequence (instantaneous change in tidal volume) is seriously disrupted, *i.e.* NVD, as a result of weakened or less effective inspiratory muscles due to dynamic hyperinflation (DH). Under these conditions, the patient experiences marked inspiratory difficulties. The psychophysical basis of NVD probably resides in the complex central processing of integrated sensory information relative to: 1) the level of central motor command output [9]; and 2) instantaneous feedback from a number of respiratory mechanoreceptors that provide proprioceptive information.

Other mechanisms

Dyspnoea may also result from a lack of increase in the ejection fraction of the left ventricle. Dyspnoea can occur due to lactate production at the skeletal muscle level. This acidosis can occur at low levels of exercise, thus reducing the walking distance achievable by a patient and generating dyspnoea. Lastly, there are some aspects of the central processing at the CNS level that are potential contributors to the perception of dyspnoea [10].

Language of breathlessness

Dyspnoea descriptors can help in the understanding of the language of dyspnoea [11–15]. Nevertheless, differences in language, race, culture, sex and previous experience can all change the perception and the manner that the feeling of being dyspnoeic is expressed to others [16–18]. For example, in a study in COPD patients [18], at any given level of exercise, females were more breathless than males. Vagal activity contributes to the sensation of "chest tightness", a term frequently reported by asthmatic patients, which may arise from the stimulation of sensory receptors within the lungs mediated through vagal pathways [19]. Several clinical conditions are characterised by descriptors as "work/effort" [11]. The intensity of the motor command to ventilatory muscles relayed to the sensory cortex

(corollary discharge), alone or in combination with force generation and respiratory muscle contraction, can be perceived as a sensation of "effort" and considered as difficult breathing [20, 21]. Patients with interstitial lung disease frequently use terms such as "rapid" and "shallow" to describe their respiratory discomfort [11, 12, 15, 22]. Descriptions such as "air hunger", "need to breathe" and "urge to breathe" appear to be related to an increased respiratory drive [13].

Dyspnoea and specific diseases

COPD

Pathophysiological factors known to contribute to dyspnoea in COPD patients include: increased intrinsic mechanical loading of inspiratory muscles, the inspiratory threshold load (the dynamic intrinsic positive end-expiratory pressure (PEEPi) [23]); increased mechanical restriction of the thorax; inspiratory muscle weakness; increased ventilatory demand relative to capacity; gas exchange abnormalities; dynamic airway compression; cardiovascular factors; and any combination of the above [24]. Neither the forced expiratory volume in one second (FEV_1) nor the FEV_1-to-vital capacity ratio are good predictors of dyspnoea in patients with severe chronic airflow obstruction. There is a close correlation between hyperinflation (as demonstrated by reduction of inspiratory capacity) during exercise and the intensity of exercise dyspnoea [8, 9]. In contrast to healthy subjects who report a perception of increased effort/work at the end of exhaustive exercise, patients with COPD select descriptors of inspiratory difficulty and unsatisfied inspiration (*i.e.* "can't get enough air in") [9]. During exercise, COPD patients also report qualitative perceptions of "unsatisfied" or "unrewarded" inspiration, "shallow breathing" and "inspiratory difficulty". These distinct sensations are associated with DH and its negative mechanical effects, *i.e.* PEEPi and the uncoupling of the normal association between respiratory effort and ventilatory output [9, 25].

Asthma

Patients describe spontaneous and induced asthma using similar terms [26, 27]. In induced asthma, the initial sensation of "chest tightness" reflects the breathing discomfort resulting from mild bronchoconstriction; the sensation of "work" or "effort" of breathing is experienced with a more severe FEV_1 decrease and hyperinflation [28]. Perception and descriptors of dyspnoea are also, and more remarkably, important for the definition of asthma severity. It has been shown that patients suffering from near-fatal asthma (NFA) attacks have a blunted perception of dyspnoea [29]. Patients with a low perception of dyspnoea had statistically significantly more hospitalisations, NFA attacks and deaths during a follow-up period [30]. In addition, it has been found that perception of dyspnoea is blunted in NFA patients both at rest and at the end-point of various exercises [31]. In addition, the mechanisms involved in the exercise limitation observed in NFA patients were different from those found in non-NFA subjects: the former stopped exercising mainly because of leg discomfort, whereas the latter stopped predominantly because of dyspnoea [31].

Table 1 Descriptors for dyspnoea in different conditions

Rapid breathing	*Chronic heart failure*
Incomplete exhalation	*Asthma*
Shallow breathing	*Restrictive diseases*
Increased work/effort	*COPD, interstitial lung disease, neuromuscular disease, chest wall diseases*
Suffocation	*Chronic heart failure*
Air hunger	*COPD, chronic heart failure*
Tight chest	*Asthma*
Heavy breathing	*Asthma*

Interstitial/restrictive lung disease

Patients with restrictive lung disease adopt a tightly constrained breathing pattern, probably as a strategy for avoiding dyspnoea [32]. They frequently complain of "work/effort", "unsatisfied inspiration", "inspiratory difficulty", and "rapid" and "shallow" breathing [32–35]. These sensations have their physiological basis partially in an impaired ability to increase lung volume and displace the thorax appropriately in the setting of an increased ventilatory drive.

Congestive heart disease

Patients with chronic heart failure may stop exercising because of intolerable exercise dyspnoea, leg fatigue or both at a point where there is apparent cardiopulmonary reserve [36]. During exercise, these patients describe their dyspnoea using the cluster "suffocating at rest ", "rapid breathing", "air hunger" [9], a "need to sigh" [11], or "work/effort" [14].

Measurement

The two major reasons for measuring dyspnoea are: 1) to discriminate symptom severity between individuals; and 2) to evaluate dyspnoea changes for a given individual [37]. Both psychophysical methods and clinical scales have been used to assess breathlessness [37]. Although dyspnoea is a subjective sensation, the principles of psychophysics (the study of the relationship between a stimulus and the response) can be applied in order to quantify the severity of breathing discomfort. This technique usually involves the measurement of perception of breathing changes in response to externally added loads [38]. This approach has led to great progress in the understanding of respiratory sensations; however, technical aspects and time requirements limit its application in the routine setting.

Visual analogue scale

A visual analogue scale (VAS) or a category-ratio scale may be used to assess dyspnoea during an exercise test [37, 38]. With VAS, the subject is instructed to provide a quantification of his/her dyspnoea, placing a mark on a horizontal or vertical line, usually 100 mm in length, with or without descriptors like "no breathlessness" and "intolerable breathlessness" or significant images (figure 1) positioned as anchors at the two extremes [39].

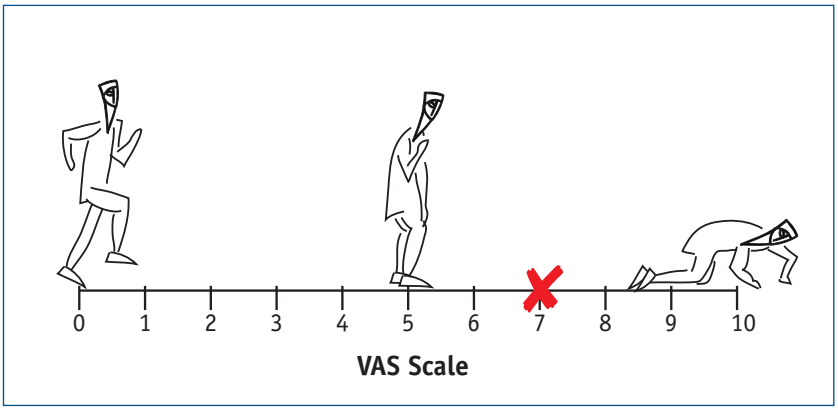

Figure 1
The patient is asked to evaluate his/her dyspnoea on a visual analogue scale (VAS).

Category-ratio scale

The modified 0–10 category-ratio Borg scale is the most widely used scale to rate dyspnoea during exercise testing [40]. This scale consists of a vertical line labelled 0 to 10, with nonlinear spacing of verbal descriptors of severity corresponding to specific numbers. The subject can choose the number or the verbal descriptor to reflect presumed ratio properties of sensation or symptom intensity (table 2). The VAS and the Borg scale provide similar scores during incremental cardiopulmonary exercise testing in healthy subjects and in COPD patients [41], although the Borg scale is more correlated with pulmonary function test values than VAS [42]. The descriptors on the Borg scale permit comparisons among individuals, based on the assumption that the verbal descriptors on the scale describe the same intensity for different subjects. Usually, both healthy individuals and patients with cardiorespiratory disease stop exercising at submaximal (at ratings between 5 and 8 on the Borg scale) intensities of dyspnoea and/or leg discomfort [37], independent of the peak power obtained [43]. Patients can also give ratings at specific times (iso-time) or work load (iso-workload) increments during the exercise test [37]. A numerical value or descriptor on the Borg scale may be used as a dyspnoea "target" (as opposed to a measured length in mm on the VAS) for prescribing and monitoring exercise training [44].

Table 2 Borg scale

Modified 0–10 category-ratio Borg scale	
0	*Nothing at all*
0.5	*Extremely weak*
1	*Very weak*
2	*Weak*
3	*Moderate*
4	*Somewhat strong*
5	*Strong*
7	*Very strong*
10	*Extremely strong*
*	*Maximal*

Clinical dyspnoea scales: MRC scale

Since 1959, the Medical Research Council (MRC) scale [45] has been used extensively as a discriminative instrument, based on the magnitude of task that provokes dyspnoea (figure 3). The MRC scale is simple to administer and correlates with other dyspnoea scales and with scores of health status [46]. It has been recently included in a global index that is able to predict the risk of death from any cause and from respiratory causes among patients with COPD [47]. MRC and other similar scales focus only on one dimension that affects dyspnoea; furthermore, the grades are quite broad, so that it may be difficult to detect small but important changes with particular interventions [37].

Table 3 MRC scale

Medical Research Council (MRC) scale

Five statements about perceived breathlessness:
1. I only get breathless with strenous exercise
2. I get short of breath when hurrying on the level or up a slight hill
3. I walk slower than people of the same age on the level because of breathlessness or have to stop for breath when walking at my own pace on the level
4. I stop for breath after walking 100 yards or after a few minutes on the level
5. I am too breathless to leave the house

BDI-TDI

The Baseline (BDI) and Transition (TDI) Dyspnoea Indexes include two components: functional impairment and magnitude of effort, in addition to magnitude of task that provoked breathing difficulty [37, 48]. The BDI is a discriminative instrument used to measure dyspnoea at a single point in time, whereas the TDI was developed as an evaluative instrument to measure changes in dyspnoea from the baseline state. Ratings or scores for dyspnoea are obtained from an interviewer, who selects a score for each of the three components based on the patient's answers, using the specific criteria for the grades as described for the instruments. Translations in different languages are available [37].

CRDQ

Dyspnoea is one of four components of a quality of life instrument, the Chronic Respiratory Disease Questionnaire (CRDQ), for patients with respiratory disease [49]. The individual patient is asked to select the five most important activities that have caused dyspnoea over the previous 2 weeks by recall and by then reading from a list of 26 activities. The severity of dyspnoea is graded by the patient selecting a score on a scale (range 1–7) for each of the five activities. The overall score can then be divided by the number of activities (usually five) selected by the patient. There is no correlation between the effort of dyspnoea evaluated by the Borg scale and the dyspnoea category of the CRDQ [37].

Other questionnaires

Other multidimensional questionnaires include the UCSD Shortness of Breath Questionnaire [50], the Pulmonary Functional Status and Dyspnea Questionnaire [51] and the Oxygen-Cost Diagram (OCD) [52]. The UCSD Questionnaire asks patients to indicate how frequently they experience shortness of breath on a 7-point scale during 21 activities of daily living. For a total of 24 items, there are three additional questions about limitations due to shortness of breath, fear of harm from overexertion and fear of shortness of breath. The OCD is a VAS with 13 activities along a 100-mm line. The position of these activities along this vertical line corresponds approximately to their oxygen requirements. The patient is asked to indicate the level of activity at which they begin to experience dyspnoea. The OCD score is measured in mm. The shorter the distance, the greater the breathlessness. This measure is simple to use and, for this reason, has been used quite widely [2]. Although the St. George's Respiratory Questionnaire (SGRQ) includes questions about dyspnoea as part of the symptom component for measuring health status, there is no specific score or grade for dyspnoea [53].

Which measurement is best

The BDI/TDI and the UCSD Shortness of Breath Questionnaire have demonstrated the highest levels of reliability and validity among six different measures of dyspnoea (including the American Thoracic Society (ATS) dyspnoea scale, OCD, VAS and the Borg scale) [54]. The BDI showed higher correlations with the 6-minute walking distance test, quality of well-being score, lung function, depression score and anxiety score, as compared with the UCSD questionnaire [54]. In patients with chronic airflow obstruction, dyspnoea measured using VAS and BDI correlated with measures of exercise capacity, but the proportion of shared variance with exercise loaded to the greatest with breathlessness measured using the BDI [17]. Borg scores for dyspnoea, whether at rest or at peak work rate, did not correlate with any of the measures of exercise capacity. In a factor analysis [46], the MRC, the BDI, the OCD, the activity component of the SGRQ and dyspnoea of the CRDQ were all grouped into the same factor, and the frequency distribution histograms of these measurements showed virtually the same distribution. The Borg scale, at the end of maximum exercise, was found to be a different factor. These measurements demonstrated the same pattern of correlation with physiological data [46]. Subjective scales, such as the VAS and Borg scales, were the best subjective scales to reproducibly measure changes of symptoms during steady-state exercise before and after drug intervention [55]. VAS at peak exercise, BDI/TDI and CRDQ

adequately reflected the beneficial effects of pulmonary rehabilitation [56]. In a factor analysis study in asthmatic patients, airway obstruction appeared to be an independent dimension or factor. Dyspnoea independently characterised the condition of asthma. Submaximal exercise tolerance could not be associated with the symptom of dyspnoea [57]. Borg dyspnoea score at peak exercise appears to be the best NFA indicator [34].

It is not possible to predict a patient's intensity of dyspnoea and related disability from physiological data. For example, COPD patients may have relatively mild airflow obstruction but severe dyspnoea. Therefore, there is a need to specifically measure dyspnoea with a specific tool, according to the desired purpose. Indeed, the Global Initiative for Chronic Obstructive Lung Disease [58] and the ATS [59] have both recommended that a patient's perception of breathlessness be included in any new staging system for COPD.

Educational questions

1. Dyspnoea may reflect the awareness of:
   a) the efferent motor output from the CNS
   b) the afferent information from the respiratory muscles to the CNS
   c) both
   d) neither.
2. The neuroventilatory dissociation of the respiratory pump influences dyspnoea mostly in:
   a) healthy subjects
   b) patients with respiratory disorders
   c) neither.
3. Exercise dyspnoea strictly correlates with:
   a) decrease in FEV_1
   b) increase in dynamic hyperinflation
   c) both
   d) neither.
4. Hypoxia is the most important cause of an increase in dyspnoea:
   a) true
   b) false.
5. VAS and Borg scores measure:
   a) change in chronic dyspnoea
   b) exercise dyspnoea
   c) both
   d) neither.

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Suggested further reading

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Suggested answers

1. a) The efferent motor output from the CNS.
2. b) Patients with respiratory disorders.
3. b) Increase in dynamic hyperinflation.
4. b) False.
5. b) Exercise dyspnoea.