REVIEW

Dyspnea and leg effort during exercise

Loredana Stendardi\textsuperscript{a, b}, Michela Grazzini\textsuperscript{a, b}, Francesco Gigliotti\textsuperscript{a}, Pamela Lotti\textsuperscript{a, b}, Giorgio Scano\textsuperscript{a, b,*}

\textsuperscript{a}Fondazione Don C. Gnocchi, IRCCS, Pozzolatico, Florence, Italy
\textsuperscript{b}Department of Internal Medicine, Respiratory Disease Section, University of Florence, Italy

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Summary  Dyspnea and leg effort are the major symptoms limiting exercise in healthy subjects and in patients with a variety of respiratory disorders. Quantitative measurement of both symptoms may be obtained by category scales such as VAS and Borg, with the latter being widely used. Furthermore, descriptor clusters of dyspnea help to assess some of the reasons for stopping exercise. The intensity of dyspnea and leg effort are similar in different disease states; this symmetry suggests that the limiting discomfort is a function of the intensity of increased motor drive to peripheral and respiratory muscles. An alternative explanation for the factors which limit exercise is that the subjects stop exercise volitionally when the discomfort associated with continuing exercise exceeds that which they are willing to tolerate. Muscle strength contributes to the intensity of dyspnea and leg effort at a given power output: the greater the muscle force, the lower the symptom. Symptoms also correlate with intensity and duration of a task by a power function in such a way that when minimizing the intensity of a given muscular task by prolonging the duration of activity, the symptom is drastically reduced. Skeletal muscle fatigue may be a factor limiting exercise tolerance both in healthy subjects and in patients with cardiorespiratory disorders. In conclusion, symptom measurement complements physiological measurements, both being essential to a comprehensive understanding of exercise tolerance.

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KEYWORDS
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*Corresponding author. Department of Internal Medicine, Section of Clinical Immunology, Allergology and Respiratory Disease, University of Florence, Viale Morgagni 87, 50134 Firenze, Italy. Tel.: +39 55 4296 414; fax: +39 055412867.
E-mail address: g.scano@dmi.unifi.it (G. Scano).
Introduction

Subjects with low exercise tolerance stop exercising when discomfort is “somewhat severe”, and subjects with high tolerance stop exercising at “maximal” discomfort. The possibility has therefore been considered that symptoms are limiting. Hence, an alternative explanation for the factor limiting exercise is that subjects stop exercise volitionally when the discomfort associated with continuing exercise exceeds what they are willing to tolerate. In this connection, individual symptom tolerance and motivation should be considered. This Review is aimed at defining the main subjective symptoms, that is, dyspnea and leg effort, that allow us to understand exercise tolerance, and at explaining how these symptoms are involved in hindering exercise.

Quantitative measurements of the symptoms

Scoring symptoms may be obtained by using scales such as the visual analog scale (VAS) and Borg scale. The Borg scale is a numerical category with ratio property scale; ratings are related to workload by a power function in keeping with Stevens’s power law i.e., with an exponent similar to that obtained with open magnitude procedures. VAS is a continuous interval scale closed at both ends with maximum and minimum levels. The Borg scale offers the simplicity of semantic scaling using descriptive categories. The validity of the Borg scale is based on the knowledge that the perceptual range is finite, the physical range is finite, and that the ratio properties are known. In turn, the Borg scale incorporates absolute magnitude ranges from zero to maximum, with valid ratio properties. The tagged descriptors to numbers offer another advantage, e.g. the “moderate” tagged to the number 3 conveys a meaning that a distance on VAS such as 10 cm does not. The reliability and responsiveness of the dyspnea score have been documented for over 2 weeks in normal subjects, and over a few weeks or several days in asthmatics, in patients with chronic airflow obstruction, and over a 28-day period in patients with ILD, with no differences in dyspnea rating at 40% and 70% maximal PO. In contrast, Mador et al. found that the coefficients of variability for Borg were at 33% and 66% maximal PO, greater than at maximal PO. Reliability may be different for each mode of exercise, the exercise protocol may be another important variable to consider. Borg and VAS scales are applied with the assumption that they are internally reliable, that is, their scaling properties do not change with position along the scale. Because a maximal dyspnea is never reached at the beginning of nearly all studies, the reliability of a scale is dependent on the accurate recall or imagination of what it feels like to experience the maximum.
A period of greater than usual breathlessness may reduce subsequent estimates in patients with COPD. Prior experience of breathing with an inspiratory load causes a subsequent underestimation of breathlessness once it is removed, compared with an estimate made during an identical test that has not been preceded by loaded breathing in healthy subjects. Thus, a period of increased respiratory sensation may lead to a reduction in perceived dyspnea during exercise upon return to normal conditions.

Dyspnea

The language of dyspnea

Dyspnea is a general term which encompasses an array of different descriptors varying in intensity. The prospective use of descriptors of dyspnea may contribute to our understanding of the mechanisms of exercise dyspnea and assist in identifying a specific diagnosis. Descriptors of dyspnea have been obtained in symptomatic patients with different cardiorespiratory diseases. Standardized descriptors are being grouped in discrete clusters with high discriminating value among diseases. Dyspnea caused by the same stimulus may differ among individuals and be described in different ways, depending on individual experience.

Increased respiratory work/effort is frequently a chosen descriptor cluster of exercise dyspnea both in healthy subjects and in patients with chronic obstructive pulmonary disease (COPD). The intensity of the motor command activating the muscular receptors (muscle spindles, joint receptors, and tendon organs) and the copy of the increased motor command to the sensory cortex are consciously appreciated as effort. During progressive exercise, the pressure, flow, volume, inspiratory time and respiratory frequency may independently modify the perception of effort.

The discrepancy between increased respiratory motor output (or effort) and an inadequate mechanical response of the system, that is, the neuroventilatory dis coupling of the ventilatory pump, is thought to play a major role in the perception of unrewarded inspiration in patients with COPD, or interstitial lung disease (ILD). In COPD, dynamic hyperinflation (i.e., the increase in end-expiratory-lung-volume) during exercise, probably contributes more to the mechanical problems, (i.e., elastic and threshold loads) than the increase in airflow resistance or decrease in dynamic pulmonary compliance. The important consequence of hyperinflation is the severe mechanical constraint on tidal volume expansion. And this accounts for the sensation of inspiratory difficulty.

Deconditioning is a common development in patients with COPD and may contribute to dyspnea with daily activities. Use of a descriptor cluster of dyspnea exercise may reveal that patients stop activities due to increase in breathing more (expected with Deconditioning), rather than to work/effort.

When exercising with external thoracic restriction, healthy subjects frequently identify inspiratory difficulty, rapid and shallow as appropriate descriptors of their respiratory discomfort. Rapid breathing also characterizes the exercise response in patients with ILD. Circumstantial evidence indicates that it is possible to modulate dyspnea by regulating the pattern of breathing. At a constant level of ventilation, either increasing or decreasing respiratory frequency voluntarily from the spontaneously adopted range is associated with an increase in dyspnea.

A copy (corollary) discharge from the brainstem to the sensory cortex is associated with the sensation of air hunger. This chemical corollary discharge is likely weaker than the voluntary corollary discharge from the motor cortex to the sensory cortex. Results by Lansing et al. have shown that increasing chemical stimulation with unchanged ventilation is associated with the sensation of air hunger. Instead, increased ventilation with unchanged chemical stimulation is mostly associated with the sensation of work/effort. The results are consistent with the possibility that breathing responses to change in chemical drive may be regulated in part to minimize sensations of respiratory effort and discomfort. During exercise, the increase in ventilation without increase in PaCO₂ is probably the reason why healthy subjects perceive work/effort, but not air hunger. According to O’Donnell et al., progressive cycling exercise constrains increase in lung volume while increasing ventilatory motor output to the respiratory muscles in patients with COPD; this may increase PaCO₂ and breathing frequency, but lowers tidal volume. In this circumstance, patients select descriptors such as “effort”, unrewarded inspiration, insufficient inspiration, but not air hunger. The likely explanation we suggest lies in the possibility that the stronger voluntary corollary discharge “masks” the weaker chemical corollary discharge from the brainstem.
The affective components

Carrieri-Kolman et al.\textsuperscript{37} have reported that patients with COPD can differentiate between the severity of dyspnea and affective components, i.e. the associated anxiety and distress during exercise, further emphasizing the multidimensional sensation of dyspnea. Jones and Wilson\textsuperscript{15} investigated the possibility that distress caused by breathlessness is different from breathlessness distress was correlated with ventilation, and its score was highly repeatable after several days. At any given level of ventilation, the breathlessness distress score was lower than the breathlessness intensity score, and for any given distress score there was a higher breathlessness intensity score at the end of the exercise. The study suggests that the affective components of dyspnea may be relatively independent of the intensity component, that is, breathlessness has both a level of intensity and an affective component that may be relatively independent of each other.\textsuperscript{15} These authors, however, suggest that intensity and distress could converge and become synonymous at high work levels. The ability to differentiate between dyspnea and distress during exercise has been reported in patients with COPD.\textsuperscript{38}

In healthy subjects and patients with a variety of cardiorespiratory disorders, symptom tolerance and motivation during exercise also pertain to prevailing non-respiratory symptoms, such as leg effort.

Leg effort

The discomfort experienced and associated with exercising peripheral skeletal muscles is another specific sensation commonly cited as limiting exercise during formal exercise testing. The perception of effort needed to drive peripheral skeletal muscles is thought to play an important role in limiting muscular performance in healthy subjects and in patients with cardio-respiratory disorders.\textsuperscript{22,39–47}

Let us now explore the notion that, like dyspnea, leg effort frequently limits exercise in both healthy subjects and in patients. During cycle ergometry both healthy subjects and patients with a variety of respiratory disorders continue to exercise until the intensity of dyspnea or leg fatigue becomes intolerable.

Differences in contractile capacity between respiratory muscles and peripheral skeletal muscles might account for differences in intensities of perception during exercise. Sutton et al.\textsuperscript{48} have shown that both leg effort and dyspnea increase systematically with power output (PO) at sea level and at altitude. At a given PO both symptoms increase with altitude. Moreover, while at sea level leg effort exceeds the perceived intensity of dyspnea during maximum exercise, at altitude (Pb 282 Torr) the ratings for leg effort and dyspnea are the same during maximal PO. Nonetheless, at the same ventilation, dyspnea is similar at all altitudes, whereas leg effort increases substantially with altitude at the same PO. These data suggest a better preserved contractile capacity of the respiratory muscles than the peripheral muscles.\textsuperscript{48}

Across different disease states, the intensity of dyspnea and leg effort is similar.\textsuperscript{1,49} The symmetry between leg effort and exercise dyspnea suggests that the limiting discomfort may be a function of the intensity of the motor drive to the peripheral muscles and respiratory muscles.\textsuperscript{42}

The circulation, ventilation and gas exchange are qualitatively related to the PO of the peripheral muscles during exercise. Exercise limitation is most frequently ascribed to finite boundaries in oxygen transport, and is attributed to circulatory limitation, ventilatory limitation, and neuromuscular limitation, these boundaries being approached but not necessarily reached.\textsuperscript{40} All three interact in contributing to the conscious experience of exercise both in healthy subjects and patients. Nonetheless, based on the experience developed in evaluating thousand of healthy subjects and patients with a variety of cardiorespiratory disorders,\textsuperscript{41} Killian\textsuperscript{40,42} has postulated that exercise is more often limited by symptoms related to discomfort experienced, and associated with the active peripheral skeletal muscles and respiratory muscles.

Perception of symptoms in healthy subjects and in patients with cardiorespiratory disorders

Let us start by considering symptoms limiting exercise in healthy subjects. During a standardized cycle incremental exercise test to maximal capacity, leg effort and dyspnea increase in a linear manner with PO expressed as percentage of maximal PO; in contrast, sex, age or stature do not contribute to rating of symptoms.\textsuperscript{1} Threshold PO below which symptoms are not appreciated lies between 20% and 40% of the maximum PO achieved.\textsuperscript{1,40} Killian et al.\textsuperscript{1} reported that 89% of subjects rated the limiting symptom as sub
maximal. Some with low tolerance (5%) stopped exercise when discomfort was “somewhat severe”, and subjects with high tolerance (20%) stopped exercise at “maximal”.

Studies have compared the response of patients and healthy subjects. Killian et al.\textsuperscript{39} reported that 320 healthy subjects and 93 patients with COPD did not vary significantly from the normal subjects in their reason for terminating the exercise; both groups stopped exercise due to either leg effort, (37% and 43%, respectively) or dyspnea (22% and 20%, respectively) or the association of leg effort and dyspnea (42% and 31%, respectively). The average leg effort was scored 7 u.a. on a Borg scale, and the average dyspnea was scored 6 u.a. in both groups; dyspnea, however, was more frequently limiting in the group with lowest FEV\textsubscript{1} (0–40% pred), while the score of leg effort and dyspnea was not different between patients with mild-to-moderate obstruction (and in one-third of subjects with FEV\textsubscript{1} < 40% pred) and controls. More interestingly, patients stopped exercising at lower work capacity (W\text{cap}) than controls. Ventilatory capacity and circulatory capacity were not significantly different in patients limited by dyspnea or leg effort or a combination of both. Most subjects stopped exercise at submaximal heart rate (HR), submaximal ventilation (VE), and submaximal symptom intensities, making it difficult to isolate the real limiting factor. In turn, exercise capacity may be limited by the same symptoms in patients as in normal subjects, but the limiting symptom intensity may be reached at a lower percentage of W\text{cap}, lower HR/HR\text{cap}%, VE\text{max}/VE\text{cap}%, and VO\textsubscript{2}/VO\textsubscript{2cap} in the former. In line with the data of Killian et al.\textsuperscript{39} Hamilton et al.\textsuperscript{50} reported that 50% of 578 patients with COPD were limited by leg effort and dyspnea, 28% were limited by leg effort, and 12% were limited by dyspnea. The difference between the intensity of leg effort and dyspnea at maximum exercise was greater in subjects in whom leg effort was the prevailing symptom compared with subjects limited by dyspnea or leg effort. Similar results have been obtained by Mahler and Harver in COPD patients.\textsuperscript{46} Other studies reported similar exertion and dyspnea scores during maximal cycle exercise in COPD, and in controls or higher estimates of dyspnea than ratings of leg effort in patients with COPD\textsuperscript{50–53} or ILD.\textsuperscript{10} Comparative data were not reported in patients with asthma in whom the mean rating of dyspnea was 7.4 a.u. on a Borg scale.\textsuperscript{8}

Hamilton et al.\textsuperscript{50} found a significant relationship between maximum predicted work capacity (W\text{cap}\times50\%) and symptoms at W\text{cap}50%. This permitted classification of disability in subjects in whom it was unfeasible to exercise to exhaustion. In the study, 83% (positive predictive value) of patients who rated symptoms greater than moderate (3 a.u. on a Borg scale) at W\text{cap}50% could be classified as disabled; a lower percentage (67%, negative predictive value) of patients who rated symptoms equal/lower than moderate at W\text{cap}50% could be classified as having normal exercise capacity.

What symptoms limit exercise in patients with cardiopulmonary disorders?

Hamilton et al.\textsuperscript{49} and Killian\textsuperscript{41} in a larger series of patients (16,000) with angina, pulmonary impairment, ischemic heart disease, or cardiopulmonary disease, and healthy subjects, revealed a simple picture. When patients were subdivided according to exercise tolerance, the intensity of both dyspnea and leg effort increased as exercise tolerance decreased for all disease types. The proportion of patients limited by dyspnea, leg effort or a combination of both was similar for all disease types.

Patients with chronic heart failure (CHF) complain of exercise limitation. Clark et al.\textsuperscript{34} have shown that CHF patients may stop exercise because of either intolerable exertional dyspnea, or leg fatigue or both at a point where there is apparent cardiopulmonary reserve. Mancini et al.\textsuperscript{45} have suggested that the respiratory function contributes to exercise limitation in these patients: acute unloading on the respiratory muscles decreases dyspnea and increases exercise duration, without changing oxygen uptake and ventilation. Hughes et al.\textsuperscript{56} hypothesized that excessive loading on the respiratory muscle pump might contribute to exertional dyspnea. The observation that slowing of inspiratory muscle relaxation rate occurs in patients with CHF walking until severe breathlessness supports their hypothesis. To stress the respiratory system during exercise Chauhan et al.\textsuperscript{57} used the technique of dead space loading which increases ventilation at a given metabolic rate. Peak minute ventilation increased, the breathing pattern was deeper and slower, while the intensity of dyspnea and leg effort on a Borg scale (4.7 and 5.3 a.u. at peak minute ventilation, respectively) did not change during exercise with added dead space. The study has shown that respiratory function does not contribute to exercise limitation in patients with stable CHF. Despite conflicting reports, in general, integrated cardio-pulmonary and locomotor muscle interaction limit exercise in CHF. Factors involved in leg effort pertain to the sensory domain but also reflect a combination of peripheral muscle weakness and metabolic alteration in the active skeletal muscles (blood perfusion,
oxygen delivery, oxidative metabolism). In this connection, recent studies by Dempsey’s group have focused on the role of ventilatory-locomotor muscle blood flow competition on exercise limitation. In two papers, they have shown that unloading the respiratory muscles with proportional assist ventilation during strenuous exercise in cyclists reduces oxygen uptake and the perception of both breathlessness and leg discomfort, indicating that the work of breathing significantly influences exercise performance. The effect of the normal respiratory muscle load on exercise performance in trained cyclists may be due to the associated reduction in leg blood flow which increases both leg fatigue, and the intensity with which leg effort and respiratory muscle effort are perceived. This also explains the difficulty of discriminating between the two sensations. The link between respiratory work and exercise performance is likely to be due to diaphragm fatigue (see below) which elicits a peripheral vasoconstrictor effect that decreases limb muscle vascular conductance limiting blood flow to locomotor muscles. Interestingly, the effects of respiratory muscle unloading on limb vascular conductance during exercise occurred when the intensity exceeded 80% of maximum. However, in patients with CHF or those with COPD, even sub-maximal levels of exercise may cause fatigue of the respiratory muscle and perhaps cause sympathetically mediated vasoconstriction of limb vasculature. In these cases, respiratory muscle unloading may be especially beneficial to enhancement of exercise performances.

Let us now consider the link between muscle fatigue and symptoms limiting exercise.

Fatigue of the skeletal muscles

Fatigue of the diaphragm and other respiratory muscles is an important mechanism involved in redistribution of blood flow. Development of diaphragm fatigue during exercise is a function of the relationship between the magnitude of the diaphragm’s work and adequacy of its blood supply: the less blood flow available, the less diaphragm work is required to produce fatigue. Muscle force output vs. blood flow imbalance of the diaphragm favoring fatigue may occur during endurance exercise only when the intensity of exercise exceeds 85% \( \text{VO}_2\text{max} \) or arterial hypoxemia is present. Unlike healthy subjects, evidence of overt central or contractile fatigue of the diaphragm, has not been provided in patients with COPD. This is likely to be due to diaphragm adaptation to chronic loading and the degree of adaptation which correlates with the severity of COPD.

Unlike respiratory muscle fatigue, leg fatigue is commonly perceived at peak exercise in patients with COPD. This could be related to the fact that peripheral muscle alterations increasing susceptibility to contractile fatigue are common in these patients. Saey et al. have recently addressed the issues of whether contractile fatigue may contribute to limit exercise tolerance, and whether optimal bronchodilation might affect exercise tolerance, regardless of contractile fatigue of peripheral skeletal muscles in patients with COPD. They found that the dyspnea score is lower at peak exercise in patients with contractile muscle fatigue, compared with patients without contractile fatigue, whereas the perception of leg fatigue is similar between the two groups. The authors’ explanation is that patients who become rapidly intolerant to exercise because of early dyspnea, or limitation in ventilation do not sufficiently activate their peripheral muscles to develop muscle fatigue. Other patients with greater capacity to exercise will be able to challenge their peripheral muscle to the point of fatigue. In patients who fatigued after placebo exercise, ipratropium bromide (IB) did not increase endurance time, whereas in patients who did not fatigue, the endurance time increased after IB. The data indicate that leg fatigue may be a factor limiting exercise tolerance, and may explain why bronchodilation fails to improve exercise tolerance in some COPD patients.

Nonetheless, fatigue is not the sole reason for symptoms limiting exercise. Strength and weakness of the skeletal muscles play a primary role.

Strength and weakness of the skeletal muscles

The subjective effort associated with the generation of a given external PO is inversely related to the strength of the exercising muscles: for weak muscles, a greater use of the available muscle mass is required to maintain a given external PO, resulting in an increased sense of effort. Thus, muscle strength contributes to the intensity of leg effort and dyspnea at a given PO. A doubling of the strength is associated with a decrease in both the sense of effort and dyspnea by 25–30%. Yet, the intensity of leg effort and dyspnea at maximal exercise increases as knee extensor strength and inspiratory muscle strength increases both in
healthy subjects and in patients with cardiopulmonary disease. Kearon et al. quantified the separate contribution of the intensity of exercise and its duration to muscular effort and dyspnea during cycling ergometry in healthy subjects. Perceived leg effort increased by a factor of 4.4 with a doubling of the work rate, and by 1.3 with a doubling of the duration. Perceived dyspnea increased by 5.3-fold with a doubling of the work rate and by 1.4-fold with a doubling of the duration. Thus, in the performance of a given muscular task, minimizing the intensity by prolonging the duration of activity has a dramatic effect on reducing muscle effort and dyspnea. Most subjects will exercise at a level at which they feel compatible: weak subjects select a lower PO to reduce symptom intensity and take longer to complete a given task.

Modality of exercise

Leg exercise

Many of the quoted studies have used cycling as the mode of exercise. Walking, however, may be more familiar and is a whole body exercise compared with cycling. So, the question arises. Do cycling and walking differently affect leg effort and dyspnea? Palange et al. have shown that metabolic and ventilatory responses in COPD patients during walking are different from those during cycling perhaps due to the recruitment of a wider range of muscle groups. This also could explain why the level of dyspnea was greater, and level of leg effort smaller after incremental shuttle walking test compared with an incremental cycling protocol. Marthur et al. compared incremental walking on a treadmill with incremental cycling in severe COPD, and reported that dyspnea was the major symptom limiting both types of exercise. Based on the above reports, Man et al. hypothesized that symptom of leg effort depends on the mode of exercise employed, and that leg effort would be reported less commonly after walking. They found that dyspnea alone was a more commonly cited symptom after incremental walking compared with incremental cycling (81% vs. 34%), and after endurance walking compared with endurance cycling in patients with COPD. These data indicate that (a) symptom limitation is exercise-specific, with walking being much more limited by dyspnea than by leg effort or fatigue, and (b) leg effort is an infrequent symptom after walking in COPD patients. We have recently hypothesized that walking at increased speed or increasing gradient might have different effects on chest wall kinematics and respiratory muscle power components and contribute differently to sensation of respiratory effort. Unlike the starting hypothesis, however, the combination of different patterns of flow and pressure generation made the intensity of respiratory effort similar during walking at ascending gradient with constant speed, and at ascending speed with constant gradient.

Arm exercise

Shoulder girdle, and pectoral girdle muscles are accessory respiratory muscles that help maintain the position of the upper torso and extend arms during unsupported arm use, such as combing, dressing, grooming, and so on. When exercise involves arm elevation, the participation of the accessory muscles in ventilation may be decreased. The rapid and shallow pattern of breathing adopted likely contributes to the sensation of dyspnea. Some patients with severe airflow obstruction experience greater dyspnea and demonstrate dyssynchronous thoraco-abdominal breathing during unsupported arm exercise, but not during leg cycling. Celli et al. also showed that in COPD patients, dyspnea is worse with arm exercise than it is with leg exercise at the same total body oxygen consumption, suggesting that the load borne by the other inspiratory muscles must increase for the same level of ventilation.

In COPD patients with expiratory flow limitation (EFL) the increase in ventilation during leg exercise is accomplished by dynamic hyperinflation which helps minimize EFL but increases dyspnea. Reports on regulation of lung volumes during unsupported arm exercise are scanty: simple arm elevation is associated with an increase in functional residual capacity, or decrease in inspiratory capacity in COPD, whereas arm exercise results in premature termination of expiration and dynamic hyperinflation in flow-limited patients with cystic fibrosis. Preliminary data from our laboratory indicate that both arm effort and dyspnea are associated with an increase in dynamic hyperinflation during upper limb exercise in patients with COPD.

Conclusion

The experience of exercise-related perceptions can have discrete subjective domains. The sense of
effort is generally related to the effort required to
generate the power to breathe. Symptom measure-
ment complements physiological measurements,
both being essential to a comprehensive under-
standing of exercise tolerance. The exercise capa-
city of patients is limited by the same symptoms as
that of normal subjects, but the limiting symptom
intensity is reached at a lower capacity. The
following, however, requires consideration: 1. Cul-
tural, socio-economic, linguistic and educa-
tional backgrounds may influence the use of the
language of dyspnea; 2. Whether improvement of
the physiological derangements modify the lan-
guage of dyspnea has yet to be defined; 3. Would
respiratory muscle training improve exercise capa-
city by preventing or delaying respiratory muscle
fatigue and its effect on blood flow competition
between respiratory and locomotor muscles?

A lot of research is needed to clarify these
points.

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