Workstation 2: Strategies in pulmonary rehabilitation for the severe COPD patient

Breathing exercises to relieve dyspnea in severe COPD

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KEY MESSAGES

- Lung hyperinflation is closely associated with expiratory flow limitation and has major clinical consequences for breathlessness and exercise intolerance in patients with COPD.
- Dynamic hyperinflation (e.g. during exercise or exacerbations) critically limits tidal volume expansion and negatively impacts the demand/capacity balance of the already compromised respiratory muscles.
- A growing disparity between increased central neural drive and the reduced respiratory muscular/mechanical response due to hyperinflation contributes importantly to the perception of respiratory discomfort and breathlessness during exertion.
- Several non-pharmacological treatment options are available that can help to either 1) directly reduce dynamic hyperinflation; 2) indirectly improve dynamic hyperinflation by reducing ventilatory needs for a given level of exertion or 3) assist the overburdened respiratory muscles to better cope with the increased loads and improve symptoms during exertion.
- Several simple breathing strategies (e.g. pursed lips breathing, active expiration, and body positioning techniques) that are sometimes used spontaneously may be taught to patients to improve symptoms at rest and during exercise.
- Subjective benefits in terms of dyspnea improvement during exertion need to be evaluated in individual patients and weighed against the costs, risks, and time investment of the different interventions.

SUMMARY

Introduction

Lung hyperinflation is highly prevalent in patients with chronic obstructive pulmonary disease (COPD) and occurs across the continuum of the disease [1]. It is most pronounced in patients with severe airflow obstruction. Important negative consequences of (dynamic) hyperinflation during activities include:
1) limits on VT expansion resulting in early ventilatory mechanical limitation; [1-5]
2) increased elastic and threshold loading on the inspiratory muscles resulting in an increased work and oxygen cost of breathing (reduced efficiency); [5-10]
3) functional inspiratory muscle weakening due to mechanical disadvantage (i.e. operating at shorter lengths) and increased velocity of shortening (needed to create higher inspiratory flows);[5;7;11-13]
4) negative impact of increased inspiratory muscle work on leg blood flow and muscle fatigue;[14-17]
5) carbon dioxide (CO₂) retention; [18] and
6) adverse effects on cardiac function and central hemodynamics.[19-22]
The growing disparity between increased central neural drive and the reduced respiratory muscular/mechanical response due to hyperinflation contributes importantly to the perception of respiratory discomfort during exertion. The physiological rationale and efficacy of selected breathing exercises aimed at improving symptoms caused by these alterations in breathing pattern will be discussed below. Breathing exercises are mostly aimed at reducing symptoms of dyspnea during exertion by assisting the overburdened respiratory muscles to better cope with the increased loads.

**Breathing exercises**

**Deep and slow breathing**

Training patients to transiently breathe slowly and deeply during exercise has been hypothesized to decrease hyperinflation, work of breathing, and improve symptoms and exercise capacity. Some patients with severe airflow obstruction and lung hyperinflation spontaneously use pursed lip breathing (PLB) to reduce expiratory flow and slow down respiratory rate and experience improvements in dyspnea both at rest and during exercise. Patients who do not adopt PLB spontaneously show variable responses. During PLB patients perform a moderately active expiration through half-opened lips, thereby inducing expiratory mouth pressures of about 5cmH2O. It has been shown that slowing down expiratory flow reduces the tendency of airways to collapse and resulting air trapping. This is probably the reason why the technique seems to be most effective in patients with severe loss of lung elastic recoil pressure and tracheobronchial collapse. Adopting PLB at rest reduces breathing frequency and increases VT, while V'E is usually maintained or slightly reduced. Reducing dead space and improving alveolar V'E in this way probably explains improvements in gas exchange that are typically observed. These effects have also been observed when a slower and deeper breathing pattern was adopted without using PLB. In a recent small study teaching patients with COPD yoga breathing resulted in a slower, deeper pattern of breathing and improvements in oxygen saturation with no increases in symptoms. Effects of long term breathing retraining interventions on changes in spontaneous ventilator pattern are still lacking. Data from Bianchi and colleagues obtained by using optoelectronic plethysmography contribute to our understanding as to why not all patients with COPD obtain symptom relief from PLB (and therefore probably not spontaneously use it). They observed that reductions in breathing frequency and increases in VT during PLB were achieved by either increasing or decreasing EELV. The group that hyperinflated during PLB did not perceive symptomatic benefits whereas patients who decreased their EELV did (Figure 3). Reductions in EELV were positively correlated with improvements in Borg Dyspnea scores and with a greater level of expiratory flow limitation. PLB during exercise has only been studied in few small studies, with mixed results in terms of dyspnea reduction and improvements in exercise capacity. Spahija et al. performed the only study that measured changes in EELV and respiratory muscle effort after PLB during constant work bicycle exercise. They found a strong correlation between changes in dyspnea sensation during exercise and both changes in EELV and inspiratory muscle effort occurring with PLB. Interestingly, while all patients reduced their breathing frequency during PLB, only two out of eight subjects were able to reduce their EELV and to decrease their inspiratory effort. These were also the only patients who perceived improvements in dyspnea during exercise. Patients who increased their EELV during PLB experienced worsening of symptoms. Reductions in EELV with PLB again seemed to be related to levels of static hyperinflation (more hyperinflated patients tended to improve more) but the sample size was too small to demonstrate a statistically significant relation. Further research will be needed to identify and select patients that might benefit from PLB during exercise. It will further be important to standardize the technique and to define the amount of training, instruction and reinforcement needed to apply it successfully.
Collins and colleagues used a computerized ventilation feedback intervention aimed at slowing respiratory rate during exercise, in combination with an exercise training program. They showed surprising reductions in respiratory rate, ventilation and DH at isotime during a constant load cycling task. These improvements were related to improvements in dyspnea during exercise. Feasibility and persistence of these positive effects in the absence of the feedback still need to be determined in order to make this approach applicable for clinical practice.

**Active expiration**

In healthy subjects active expiration is not present during resting breathing and only occurs with increased ventilation (e.g. physical activity). In patients with COPD abdominal muscle activity is often present during resting breathing and this occurs more often in patients with more severe airflow obstruction. Contraction of abdominal muscles increases abdominal pressure during active expiration. It has been hypothesized that, while this increase in abdominal pressure is unlikely to facilitate lung emptying or contribute to lowering of end expiratory lung volume, it should result in lengthening of the diaphragm, thereby optimizing its end-expiratory length tension characteristics.

Diaphragm displacement and its contribution to tidal volume during resting breathing was shown not to be different in COPD patients in comparison to healthy subjects. Recently it was also demonstrated that increased expiratory muscle activity in patients with COPD was accompanied by preserved dynamic diaphragm function in comparison with healthy controls during exercise. In patients with very severe airflow obstruction and hyperinflation the contribution of the diaphragm during exercise was reduced.

It is not clear whether the spontaneously present activity of expiratory muscles should be further stimulated in patients with COPD. Reybrouck et al. observed larger decreases in FRC and improvements in pressure generating capacity of inspiratory muscles in patients performing active expiration with electromyography feedback compared to patients who received instructions without myofeedback. Symptomatic benefits were unfortunately not assessed and no studies on the effects of active expiration training during exercise have been performed so far. Casciari and colleagues studied a range of breathing retraining strategies including PLB and active expiration techniques and found additional improvements in exercise capacity in those patients that performed their rehab program in combination with breathing retraining strategies. The relative contribution of each of the different components of the breathing retraining program to the observed benefits is however unclear. Dodd et al. studied the effects of a belt strapped around the abdomen to improve length tension characteristics of the diaphragm during exercise. While this intervention resulted in improved maximal transdiaphragmatic pressure generation it increased relative load on the diaphragm and the diaphragmatic tension time index during exercise and decreased endurance time on the bicycle in comparison with unstrapped exercise.

In summary, spontaneous activity of the abdominal muscles is, depending on the severity of hyperinflation and expiratory flow limitation, often already present at rest in patients with COPD and probably helps to preserve diaphragmatic function during rest and during exercise. Whether further improvements in diaphragmatic function after stimulated active expiration can result in improvements in dyspnea and exercise capacity needs to be studied.

**Body Positioning Techniques**

Forward leaning is (in analogy with active expiration) often spontaneously used by patients in an attempt to decrease dyspnea, possibly by improving the length tension relationships of the diaphragm. Relief of dyspnea is often experienced by patients in the forward leaning position. The presence of hyperinflation and paradoxical abdominal movements have been shown to be related to relief of dyspnea in this position. Forward leaning is associated with a significant reduction in electromyographic activity of scalene and sternocleidomastoid muscles, an increase in pressure generating capacity of inspiratory muscles and diaphragm efficiency (Figure 4).
significant improvements in thoracoabdominal movements.[49-51] In addition, forward leaning with arm support allows accessory muscles (pectoralis minor and major) to significantly contribute to rib cage elevation.[26] Use of a rollator while ambulating allows forward leaning with arm support, decreases dyspnea, and increases exercise capacity.[52;53]

**Diaphragmatic breathing**

Several studies have described an increase in rib cage contribution to chest wall motion and/or asynchrony between rib cage and abdominal motion in patients with COPD that correlates with airflow obstruction and hyperinflation of the rib cage.[54-56] During diaphragmatic breathing patients are instructed to move the abdominal wall predominantly during inspiration and to reduce upper rib cage motion.[26] This aims to:

1) improve chest wall motion and the distribution of ventilation;
2) decrease the contribution of rib cage muscles to and the energy cost of breathing; and
3) to improve dyspnea and exercise performance.[26]

It has been consistently shown that patients with COPD are able to voluntarily change their breathing pattern to more abdominal movement and less thoracic excursion during diaphragmatic breathing.[57-59] Diaphragmatic breathing however often results in more asynchronous breathing movements and has not been shown to improve breathing pattern and ventilation distribution.[57-60] In most patients mechanical efficiency of breathing decreased whereas work and oxygen cost of breathing increased resulting in worsening of dyspnea.[57;60;61] In summary, there is no evidence from controlled studies to support the use of diaphragmatic breathing in hyperinflated patients with COPD.

**Inspiratory muscle training (IMT)**

Strengthening inspiratory muscles by specific training programs has been applied frequently in patients with COPD to alleviate dyspnea symptoms and improve exercise capacity.[62;63] The rationale for IMT is to compensate for the negative consequences of acute-on-chronic hyperinflation (e.g. exercise, exacerbations) on the demand/capacity imbalance of the already compromised respiratory muscles in COPD.[13;62-64] IMT has been shown to improve inspiratory muscle function (pressure generating capacity and endurance) and to reduce dyspnea and improve exercise capacity when applied as a stand-alone intervention with controlled training loads.[62] The intervention seems to be most effective in patients with compromised inspiratory muscle function (Figure).[62] Effects on dyspnea symptoms and exercise capacity when combining it with an exercise training intervention are however still not conclusive.[62;65;66] In this context it should however be noted that additional effects of other interventions exerting acute physiological effects during exercise testing (e.g heliox, or oxygen supplementation, and assisted ventilation techniques) were difficult to establish when combining them with an exercise training program.[67;68] Larger studies will be needed to show additional effects of interventions that are combined with exercise training since the latter is by itself already a very effective intervention.[68] Significant enhancement in the velocity of inspiratory muscle shortening during resistive breathing tasks, and increases in the size of type II muscle fibres following IMT have been previously observed in patients with COPD.[69;70] These improvements might be of clinical relevance to patients with respiratory muscle weakness secondary, in part, to lung hyperinflation since improved muscle performance characteristics may improve dynamic function during exercise. Similar to non-invasive ventilator support, IMT is not likely to directly affect hyperinflation at rest or the increase in EELV during exercise. Improvements in dyspnea during exercise in response to IMT and NIV are probably mostly related to adjustments in the demand/capacity imbalance in the setting of high inspiratory muscle work and functional weakening induced by DH.[62;63;71;72] The direct effects of IMT on operating lung volumes during exercise have so far only been investigated in a single study.[73] Petrovic and colleagues showed that IMT could reduce the rate of DH but, unfortunately, did not provide data on inspiratory muscle work and neuromechanical dissociation during exercise.[73] Detailed measurements of inspiratory muscle function during exercise in response to inspiratory muscle training have however not been performed so far. The additional effects of IMT on exercise performance seem to be related to the presence of
inspiratory muscle weakness.[62] Positive effects were only observed in studies with cautious control of training intensity (i.e. training loads of more than 30% Pt,max) [74] and careful patient selection. Patients with impaired inspiratory muscle strength, a ventilatory limitation to exercise with dyspnea as the main factor limiting exercise seem to have more potential to benefit.

Conclusions

Strong evidence supports the use of exercise training to reduce symptoms, increase exercise capacity and improve quality of life in patients with COPD. Properly conducted exercise training has been shown to reduce ventilatory needs and dynamic hyperinflation during exercise at a given workrate. Several interventions might be useful adjuncts to exercise in primarily ventilatory limited and hyperinflated patients who experience severe breathlessness during activities. Breathing exercises might be applied on a trial and error basis taking into account perceived subjective benefits in terms of symptom reduction. Even though the evidence base for most breathing exercises (except for inspiratory muscle training) is small application of these techniques might be worthwhile (except for diaphragmatic breathing) since even modest benefits, when achieved with simple, non-invasive, and inexpensive interventions such as PLB, active expiration and body positioning might be of value to patients. With all these interventions careful patient selection, proper and repeated instruction, control of techniques, and repeated assessments of perceived benefits in terms of symptom improvements is necessary. The transfer effects of controlled breathing exercises from resting to exercise conditions are not well established and further research should be performed in this area.

FIGURES

Figure 1. Changes in volumes of the chest wall ($\Delta V_{CW}$) with pursed lip breathing (PLB) in comparison with normal breathing at rest (QB). Closed symbols indicate end-expiratory volume; open symbols indicate end-inspiratory volume. Bars are means ± SEM. Adapted from: Bianchi R, et al. Chest Wall Kinematics and Breathlessness During Pursed-Lip Breathing in Patients With COPD. Chest. 2004; 125:459–465.
**Figure 2.** Mean data for 4 subjects with COPD in supine, standing, erect sitting, and forward leaning position. $\Delta E_{di} = $ inspiratory phasic change in diaphragmatic electromyograph; $\Delta P_{di} = $ inspiratory phasic change in transdiaphragmatic pressure. The ratio $\Delta P_{di}/\Delta E_{di}$ is an index of the efficiency of the diaphragm.


**REFERENCES**


SUGGESTED READING

   Results of this study illustrate how the progressive increase in resting hyperinflation as the disease advances has major implications for dyspnea and exercise limitation in COPD.

   This paper summarizes the mechanisms of increased work of breathing in COPD, as caused by increases in resistive and elastic loading, using pressure-volume plots popularized by E.J.M. Campbell.

   This statement provides an update on the mechanisms, assessment, and management of dyspnea and illustrates how imbalances in demand-to-capacity ratios caused by hyperinflation relate to the intensity and quality of dyspnea in COPD.

   This statement summarizes the rehabilitative treatment options available for improving dynamic hyperinflation, dyspnea, exercise capacity, and quality of life in patients with COPD.

   This paper describes a method of IPS that can be used to improve exercise endurance in patients with COPD. The authors have subsequently also applied this method during a pulmonary rehabilitation program.
   The only study to date that evaluated respiratory effort, dynamic hyperinflation, and dyspnea responses to pursed lips breathing during a constant work rate cycle exercise test.

   This paper describes the evaluation of expiratory muscle activity during exercise in patients with COPD. It was concluded that dynamic diaphragmatic function was not different in health and in COPD throughout exercise.

   This classic study illustrates that diaphragm efficiency is better and related to reduced dyspnea in the forward leaning position in comparison with erect sitting and standing in severely hyperinflated patients with severe expiratory flow limitation.