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Controlled breathing and dyspnea in patients with chronic obstructive pulmonary disease (COPD)

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Abstract—Controlled breathing is included in the rehabilitation program of patients with chronic obstructive pulmonary disease (COPD). This article discusses the efficacy of controlled breathing aimed at improving dyspnea. In patients with COPD, controlled breathing works to relieve dyspnea by (1) reducing dynamic hyperinflation of the rib cage and improving gas exchange, (2) increasing strength and endurance of the respiratory muscles, and (3) optimizing the pattern of thoracoabdominal motion. Evidence of the effectiveness of controlled breathing on dyspnea is given for pursed-lips breathing, forward leaning position, and inspiratory muscle training. All interventions require careful patient selection, proper and repeated instruction, and control of the techniques and assessment of its effects. Despite the proven effectiveness of controlled breathing, several problems still need to be solved. The limited evidence of the successful transfer of controlled breathing from resting conditions to exercise conditions raises several questions: Should patients practice controlled breathing more in their daily activities? Does controlled breathing really complement the functional adaptations that patients with COPD must make? These questions need to be addressed in further research.

Key words: breathing exercises, chronic obstructive pulmonary disease (COPD), controlled breathing, dyspnea, inspiratory muscle training, physiotherapy.

INTRODUCTION

Dyspnea is an important and debilitating symptom in patients with chronic obstructive pulmonary disease (COPD) [1]. Some pathophysiological factors known to contribute to dyspnea include (1) increased intrinsic mechanical loading of the inspiratory muscles, (2) increased mechanical restriction of the chest wall, (3) functional inspiratory muscle weakness, (4) increased ventilatory demand related to capacity, (5) gas exchange abnormalities, (6) dynamic airway compression, and (7) cardiovascular effects [2]. The relief of dyspnea is an important goal of the treatment of COPD, an irreversible airway disease. In addition to some conventional treatments, such as bronchodilator therapy, exercise training, and oxygen therapy, controlled breathing is also applied to alleviate dyspnea.

Controlled breathing is an all-embracing term for a range of exercises, such as active expiration, slow and deep breathing, pursed-lips breathing (PLB), relaxation therapy, specific body positions, inspiratory muscle training, and diaphragmatic breathing. The aims of these exercises vary

Abbreviations: COPD = chronic obstructive pulmonary disease, EMG = electromyography, FRC = functional residual capacity, IMT = inspiratory muscle training, NCH = normocapnic hyperpnea, PCO_2 = partial pressure of carbon dioxide, PImax = maximal inspiratory pressure, PLB = pursed-lips breathing, RV = residual lung volume.

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considerably and include improvement of (regional) ventilation and gas exchange, amelioration of such debilitating effects on the ventilatory pump as dynamic hyperinflation, improvement of respiratory muscle function, decrease in dyspnea, and improvement of exercise tolerance and quality of life. In patients with COPD, controlled breathing is used to relieve dyspnea by (1) reducing dynamic hyperinflation of the rib cage and improving gas exchange, (2) increasing strength and endurance of the respiratory muscles, and (3) optimizing the pattern of thoracoabdominal motion. In addition, psychological effects (such as controlling respiration) might also contribute to the effectiveness of controlled breathing (however, these effects are not discussed in this overview).

CONTROLLED-BREATHING TECHNIQUES TO REDUCE DYNAMIC HYPERINFLATION

Hyperinflation is due to altered static lung mechanics (loss of elastic recoil pressure, static hyperinflation) and/or dynamic factors (air trapping and increased activity of inspiratory muscles during expiration, dynamic hyperinflation). The idea behind decreasing dynamic hyperinflation of the rib cage is that this intervention will presumably result in the inspiratory muscles working over a more advantageous part of their length-tension relationship. Moreover, it is expected to decrease the elastic work of breathing, because the chest wall moves over a more favorable part of its pressure volume curve. In this way, the work load on the inspiratory muscles should diminish, along with the sensation of dyspnea [3]. In addition, breathing at a lower functional residual capacity (FRC) will result in an increase in alveolar gas refreshment, while tidal volume remains constant. Several treatment strategies are aimed at reducing dynamic hyperinflation.

Relaxation Exercises

The rationale for relaxation exercises arises from the observation that hyperinflation in reversible (partial) airway obstruction is, at least in part, caused by an increased activity of the inspiratory muscles during expiration [4]. This increased activity may continue even after recovery from an acute episode of airway obstruction and hence contributes to the dynamic hyperinflation. However, hyperinflation in COPD is mainly due to altered lung mechanics (loss of elastic recoil pressure and air trapping) and is not associated with increased activity of inspiratory muscles during expiration [5]. Relaxation is also meant to reduce the respiratory rate and increase tidal volume, thus improving breathing efficiency. Several studies have investigated the effects of relaxation exercises in COPD patients. Renfoe [6] showed that progressive relaxation in COPD patients resulted in immediate decreases in heart rate, respiratory rate, anxiety, and dyspnea scores compared to a control group; but only respiratory rate dropped significantly over time. No significant changes in lung function parameters were observed. In a time series experiment (A-B-A design), Kolaczkowski et al. [7] investigated, in 21 patients with emphysema (forced expiratory volume in 1 s [FEV_1] 40% of the predicted value), the effects of a combination of relaxation exercises and manual compression of the thorax in different body positions. In the experimental group, the excursion of the thorax and the oxygen saturation increased significantly. Dyspnea was not assessed.

In summary, relaxation exercises have scantly been studied in patients with lung disease. However, from such studies, a positive tendency toward a reduction of symptoms emerges.

Pursed-Lips Breathing

PLB works to improve expiration, both by requiring active and prolonged expiration and by preventing airway collapse. The subject performs a moderately active expiration through the half-opened lips, inducing expiratory mouth pressures of about 5 cm H_2O [8]. Gandevia [9] observed, in patients with severe lung emphysema and tracheobronchial collapse, that the expired volume during a relaxed expiration increased, on average, by 20 percent in comparison to a forced expiration. This suggests that relaxed expiration causes less “air trapping,” which results in a reduction of hyperinflation. Compared to spontaneous breathing, PLB reduces respiratory rate, dyspnea, and arterial partial pressure of carbon dioxide (PCO_2), and improves tidal volume and oxygen saturation in resting conditions [10–14]. However, its application during (treadmill) exercise did not improve blood gases [15].

Some COPD patients use the technique instinctively, while other patients do not. The changes in minute ventilation and gas exchange were not significantly related to the patients who reported subjective improvement of the sensation of dyspnea. The “symptom benefit patients” had a more marked increase of tidal volume and decrease of breathing frequency [15]. Ingram and Schilder [13]

identified, prospectively, eight patients who did experience a decrease of dyspnea at rest during PLB and seven patients who did not. No significant difference between the two groups was found in the severity of airway obstruction. However, in the group of patients who showed a decrease of dyspnea during PLB, a lower elastic recoil pressure of the lungs was observed. This indicates that these were patients with more emphysematous lung disease and thus more easily collapsing airways. In addition, this group revealed a significantly larger decrease in airway resistance during PLB in comparison to the other group. It appears that patients with loss of lung elastic recoil pressure benefit most, because in these patients, the decrease of airway compression and the slowing of expiration improve tidal volume. Indeed, Schmidt et al. [16] observed that the application of positive expiratory mouth pressure, with constant expiratory flow, did not lead to significant changes in the vital capacity. Instead, reducing expiratory flow resulted in a significant increase in vital capacity in patients with emphysema.

Breslin [10] observed that rib cage and accessory muscle recruitment increased during the entire breathing cycle of PLB, while transdiaphragmatic pressure remained unchanged. In addition, duty cycle dropped and resulted in a significant decrease of the tension-time index (the product of the relative contraction force and relative contraction duration), TTdi, of the diaphragmatic contraction. These changes might have contributed to the decrease in dyspnea sensation.

In summary, PLB is found to be effective to improve gas exchange and reduce dyspnea. COPD patients who do not adopt PLB spontaneously show variable responses. Those patients with loss of elastic recoil pressure—i.e., more emphysematous lung defects—seem to benefit more from practicing this technique. Its effectiveness during exertion needs further research.

Active Expiration

Contraction of the abdominal muscles results in an increased abdominal pressure during active expiration. This lengthens the diaphragm and contributes to operating the diaphragm close to its optimal length. Indeed, diaphragm displacement and its contribution to tidal volume during resting breathing was not different in COPD patients than in healthy subjects [17,18]. In addition, active expiration will increase elastic recoil pressure of the diaphragm and the rib cage. The release of this pressure after relaxation of the expiratory muscles will assist the next inspiration. In healthy subjects, active expiration is brought into play only with increased ventilation [19]. However, in patients with severe COPD, contraction of abdominal muscles becomes often invariably linked to resting breathing [20].

Erpicum et al. [21] studied the effects of active expiration with abdominal contraction on lung function parameters in patients with COPD and in healthy subjects. In both groups, FRC decreased while transdiaphragmatic pressure (Pdi) increased. The increase in Pdi was explained by the improved starting position of the diaphragm and the increased elastic recoil pressure. The effects on dyspnea were not studied. Reybrouck et al. [22] compared, in patients with severe COPD, the effects of active expiration with and without electromyography (EMG) feedback of the abdominal muscles. They reported a significantly larger decrease in FRC and increase in maximal inspiratory pressure (PImax) in the group receiving active expiration with EMG feedback.

Casciari and colleagues [23] studied additional effects of active expiration during exercise training in patients with severe COPD. During a bicycle ergometer test, they observed a significantly larger increase in maximum oxygen uptake after a period of additional controlled breathing was added to the training program.

Although active expiration is common in resting breathing and during exercise in COPD patients, and it seems to improve inspiratory muscle function, the significance of abdominal muscle activity remains poorly understood. First, if flow limitation is present, then abdominal muscle contraction will not enhance expiratory flow and might even contribute to rib cage hyperinflation [24]. In addition, relaxation of the abdominal muscle will not contribute to inspiratory flow or reduce the work of breathing performed by the inspiratory muscles. Secondly, abdominal muscle recruitment may still optimize diaphragm length and geometry [24]. However, the mechanism is still unclear, as Ninane and colleagues [20] observed that contraction of the diaphragm started just after the onset of relaxation of the abdominal muscles.

In summary, active expiration is a normal response to increased ventilatory requirements. In COPD patients, spontaneous activity of abdominal muscles is, depending on the severity of airway obstruction, often already present at rest. Active expiration improves diaphragm function, but its effect on dyspnea remains unclear.

Rib Cage Mobilization Techniques

Mobilization of rib cage joints appears a specific aim for physiotherapy, as rib cage mobility seems to be reduced with obstructive lung disease. The potential importance of mobility exercises in these patients is in line with the observed persistent hyperinflation after lung transplantation [25]. Indeed, after double lung transplantation (mainly in cystic fibrosis patients), without any mobilization of the rib cage, a significant reduction of hyperinflation is observed [25]. However, FRC and residual lung volume (RV) are persistently increased—130 and 150 percent predicted, respectively—after lung transplantation in lung disease developed during childhood, as well as in lung disease developed during adulthood [25]. This might be due to remodeling and structural changes of the rib cage. In the presence of restored lung mechanics after lung transplantation, rib cage mobilization might be of benefit in these patients. In patients with COPD, however, the basis for such treatment seems weak, as altered chest wall mechanics are related primarily to irreversible loss of elastic recoil and airway obstruction. Rib cage mobilization will not be effective in COPD patients with altered pulmonary mechanics and is therefore not recommended.

CONTROLLED-BREATHING TECHNIQUES TO IMPROVE INSPIRATORY MUSCLE FUNCTION

Reduced endurance and strength of the inspiratory muscles are frequently observed in chronic lung disease and contribute to dyspnea sensation [26]. It is believed that when respiratory muscle effort (ratio of the actual inspiratory pressure over the maximal inspiratory pressure, PI/PImax) exceeds a critical level, breathing is perceived as unpleasant [27]. Improvement of respiratory muscle function helps to reduce the relative load on the muscles (PI/PImax) and hence to reduce dyspnea and increase maximal sustained ventilatory capacity. This might also imply an improvement of exercise capacity in patients with ventilatory limitation during exercise.

Controlled breathing and body positions are meant to improve the length-tension relationship or geometry of the respiratory muscles (in particular of the diaphragm) and to increase the strength and endurance of the inspiratory muscles. According to the length-tension relationship, the output of the muscle increases when it is operating at a greater length, for the same neural input. At the same time, the efficacy of the contraction in moving the rib cage might improve. Also, the piston-like movement of the diaphragm increases and thus enhances lung volume changes. As mentioned before, and in contrast to what is often believed, diaphragm displacement, and its contribution to tidal volume during resting breathing, was not different in COPD patients [17,18]. During increased levels of ventilation, the contribution of the diaphragm is reduced in more severe COPD [28]. The diaphragm can be lengthened by increasing abdominal pressure during active expiration (see above) or by adopting such body positions as forward leaning. Specific training of the respiratory muscles will enhance their strength and/or endurance capacity.

Body Position

Relief of dyspnea is often experienced by patients in the forward leaning position [29–32], a body position commonly adopted by patients with lung disease. The benefit of this position seems unrelated to the severity of airway obstruction [30], changes in minute ventilation [29], or improved oxygenation [30]. However, the presence of hyperinflation and paradoxical abdominal movement were indeed related to relief of dyspnea in the forward leaning position [30]. Forward leaning is associated with a significant reduction in EMG activity of the scalenes and sternomastoid muscles, an increase in transdiaphragmatic pressure [30,31], and a significant improvement in thoracoabdominal movements [30–32]. From these open studies, it was concluded that the subjective improvement of dyspnea in patients with COPD was the result of the more favorable position of the diaphragm on its length-tension curve. In addition, forward leaning with arm support allows accessory muscles (Pectoralis minor and major) to significantly contribute to rib cage elevation.

In summary, the forward leaning position has been shown to improve diaphragmatic function and, hence, improve chest wall movement and decrease accessory muscle recruitment and dyspnea. In addition, accessory muscles contribute to inspiration by allowing arm or head support in this position.

Abdominal Belt

The abdominal belt is meant to be an aid to support diaphragmatic function. Herxheimer [33] studied the effects on the position and excursion of the diaphragm of both an abdominal and a rib-cage belt, in sitting and supine positions, in patients with COPD and asthma, as well as in

healthy subjects. During breathing at rest, the application of the abdominal belt resulted in a cranial displacement of the diaphragm, in both patients and healthy subjects. No changes in maximal excursions of the diaphragm or changes in lung function parameters (vital capacity) were reported. Later on, the more elastic Gordon-Barach belt was introduced by Barach [34]. Barach and Seaman [35] reported, in patients with emphysema, an increase in the excursion of the diaphragm and a reduction of the activity of accessory muscles during application of the abdominal belt when patients adopted the forward leaning position. Dodd et al. [36] compared the effects of the use of an abdominal belt at rest and during a submaximal endurance bicycle ergometer test in patients with severe COPD. They found a significant increase of maximal transdiaphragmatic pressure. However, during exercise, the relative force of the contraction (Pdi/Pdimax) and the tension-time index (TTdi) of the diaphragm were significantly increased. This significantly shortened endurance time on the bicycle ergometer test.

No additional beneficial effects were observed from the application of an abdominal belt in COPD patients.

Respiratory Muscle Training

Recent studies in patients with COPD have shown natural adaptations of the diaphragm at cellular (increased proportion of type I fibers) and subcellular (shortening of the sarcomeres and increased concentration of mitochondria) levels, contributing to greater resistance to fatigue and better functional muscle behavior [37,38]. Despite these cellular adaptations, both functional inspiratory muscle strength [39] and inspiratory muscle endurance [40] are compromised with COPD. Inspiratory muscle training (IMT) may further enhance these spontaneous adaptations.

Three types of training are currently practiced: inspiratory resistive breathing, threshold loading, and normocapnic hyperpnea (NCH). During NCH, the patient is asked to ventilate maximally for 15 to 20 min [41]. The equipment for this type of training has always been complicated, but recently a simpler partial rebreathing system was developed [42]. In a randomized controlled trial, 8 weeks of home-based NCH improved respiratory muscle endurance, 6 min walking distance, and maximal oxygen uptake, as well as health-related quality of life, but not the baseline dyspnea index in COPD patients [43].

During inspiratory resistive breathing, the patient inspires through a mouthpiece and adapter with an adjustable diameter. This resistance is flow-dependent. Adequate training intensity is only achieved by feedback of the target pressure, since flow and pressure are tightly coupled [44]. More recently, a flow-independent resistance was developed, called threshold loading, which is a valve that opens at a critical pressure [45,46]. Training intensity varies among these studies from as high as maximal sustained inspiratory (Müller) maneuvers [47] and 50 to 80 percent PImax [47–50] to low intensity at 30 percent of PImax [50–53]. All these studies have in common the careful control of the target pressure during training or the application of threshold loading [46].

Most studies observed that breathing against an inspiratory load increased maximal inspiratory pressure [44,47–54] and the endurance capacity of the inspiratory muscles [44,49,50,54]. Indeed, in an animal model, respiratory muscle training revealed significant hypertrophy of predominantly type I and IIa fibers in the diaphragm [55,56]. A recent study in COPD patients showed significant increases in the proportion of type I fibers and size of type II fibers in the external intercostals after IMT [57]. Additionally, dyspnea [52,53,58] and nocturnal desaturation time [49] decreased, while exercise performance tended to improve [59]. These outcomes were confirmed in a recent meta-analysis [59].

IMT, when coupled with exercise training, has been shown to improve exercise capacity more than exercise training alone [47,48,51,53]. The additional effect of IMT on exercise performance seemed to be related to the presence of inspiratory muscle weakness [59]. Again, these effects were only observed in studies with cautious control of training intensity (i.e., training load more than 30% PImax [60]) and careful patient selection. Patients with impaired inspiratory muscle strength and/or a ventilatory limitation to exercise performance seem to have more potential to benefit.

At present there are no data to support resistive or threshold loading as the training method of choice. Resistive breathing (breathing through a small hole) has the disadvantage that the inspiratory pressure is flow-dependent [44]. Threshold loading has the advantage of being independent of inspiratory flow rate [46], but requires a buildup of negative pressure before flow occurs, and hence, is inertive in nature. Belman et al. [61] showed that similar workloads were obtained during resistive loading and threshold loading. Threshold loading enhances velocity of inspiratory muscle shortening [62]. This might be an important additional effect, as this shortens inspiratory

time and increases time for exhalation and relaxation. Since inspiratory muscles are at risk for the development of muscle fatigue in these patients, increased relaxation time may prevent the development of inspiratory muscle fatigue.

Well-controlled IMT has been shown to improve inspiratory muscle function, resulting in additional improvement of exercise capacity and decrease of dyspnea and nocturnal desaturation time in patients with inspiratory muscle weakness. Training intensity should be at least 30 percent of the maximal inspiratory pressure for 30 min per day. Neither threshold loading nor resistive breathing has been shown to be superior to the other.

CONTROLLED-BREATHING TECHNIQUES TO OPTIMIZE THORACOABDOMINAL MOVEMENTS

Alterations of chest wall motion are common in patients with asthma and COPD. Several studies have described an increase in rib cage contribution to chest wall motion and/or asynchrony between rib cage and abdominal motion in these patients [63–65]. The mechanisms underlying these alterations are not fully elucidated, but appear to be related to the degree of airflow obstruction, hyperinflation of the rib cage, changes in diaphragmatic function, and increased contribution of accessory inspiratory muscles to chest wall motion. Indeed, increased firing frequency of single motor units of scalene and parasternal muscles [66], as well as the diaphragm [67], were observed in COPD patients compared to age-matched control subjects. In contrast to what is often suggested, diaphragm displacement and shortening during tidal breathing was not different in COPD patients compared to healthy subjects [17,18]. This indicates that the diaphragm displacement (actively and passively) still contributes to tidal breathing.

Activity of accessory muscles is positively associated with the sensation of dyspnea, whereas diaphragm activity is negatively related to dyspnea sensation [68]. Consequently, diaphragmatic breathing, or slow and deep breathing, is commonly applied in physiotherapy practice, in an attempt to correct abnormal chest wall motion; to decrease the work of breathing, accessory muscle activity, and dyspnea; to increase the efficiency of breathing; and to improve the distribution of ventilation.

Diaphragmatic Breathing

During diaphragmatic breathing, the patient is told to move the abdominal wall predominantly during inspiration and to reduce upper rib cage motion. This aims to (1) improve chest wall motion and the distribution of ventilation; (2) decrease the energy cost of breathing, the contribution of rib cage muscles, and dyspnea; and (3) improve exercise performance.

All studies show that during diaphragmatic breathing, COPD patients are able to voluntary change the breathing pattern to more abdominal movement and less thoracic excursion [69–71]. However, diaphragmatic breathing can be accompanied by increased asynchronous and paradoxical breathing movements, while no permanent changes of the breathing pattern are observed [69–72]. Although abdominal and rib cage movement clearly changed, no changes in ventilation distribution were observed [70]. In several studies, an increased work of breathing, enhanced oxygen cost of breathing, and reduced mechanical efficiency of breathing have been found [71–73]. In addition, dyspnea worsened during diaphragmatic breathing in patients with severe COPD [71,73], whereas pulmonary function [74] and exercise capacity [75] remained unaltered.

In summary, there is no evidence from controlled studies to support the use of diaphragmatic breathing in COPD patients.

Timing of Breathing

Since, for a given minute ventilation, alveolar ventilation improves when a patient breathes at a slower rate and higher tidal volume, this type of breathing is encouraged for patients with impaired alveolar ventilation. Several authors have reported a significant drop in respiratory frequency, and a significant rise in tidal volume and PaO_2 during imposed low-frequency breathing at rest in patients with COPD (see discussion of PLB). Unfortunately, these effects might be counterbalanced by an increase in the work of breathing. Bellemare and Grassino [76] demonstrated that for a given minute ventilation, fatigue of the diaphragm developed earlier during slow and deep breathing. This breathing pattern resulted in a significant increase in the relative force of contraction of the diaphragm (Pdi/Pdimax), forcing it into the critical zone of muscle fatigue. From this point of view, a breathing pattern that reduces Ti/Ttot is beneficial. Shortening inspiratory time allows more time for expiration and thus for relaxation of the inspiratory muscles and

lung emptying. However, to achieve a similar tidal volume, inspiratory flow (VT/Ti) has to increase, and this might additionally load the inspiratory muscles.

In summary, slow and deep breathing improves breathing efficiency and oxygen saturation at rest. However, this type of breathing is associated with a breathing pattern prone to induce respiratory muscle fatigue. Reducing the duty cycle might be an interesting option, but it has not yet been studied.

SUMMARY

Evidence for the effectiveness of controlled breathing on dyspnea is demonstrated for PLB, forward leaning position, and inspiratory muscle training. Diaphragmatic breathing has not been shown to be beneficial. PLB improves dyspnea and oxygen saturation and reduces diaphragm activation. Patients with emphysema especially seem to benefit from PLB. Forward leaning position improves diaphragm function, reduces rib cage muscle activity, and alleviates dyspnea. Finally, inspiratory muscle training improves inspiratory muscle strength and endurance, as well as dyspnea and the patient's quality of life. In addition, exercise performance was enhanced in patients with inspiratory muscle weakness. Use of abdominal muscle contraction during expiration seems to be beneficial to diaphragm function, but the effects on dyspnea were not studied.

In all these interventions, careful patient selection, proper and repeated instruction, and control of these techniques and assessment of their effects is necessary. The transfer effects of controlled breathing from resting conditions to exercise conditions are not well established. Further, we cannot be sure whether controlled breathing really alters or complements the functional adaptations of the respiratory pump.

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